Practical Cases in
OBSTETRICS AND GYNECOLOGY

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Foreword

Nagnath Kottapalle

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To
My family Dr Avinash, Dr Ashwini
and Aditya

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Foreword

It is my proud privilege to write the Foreword for this book "Practical Cases in Obstetrics and Gynecology," authored and edited by Dr Kanan Yelikar, Prof. and Head of Department of Obstetrics and Gynecology in Govt. MCH, Aurangabad, Maharashtra.

This book is specially meant for MBBS and above students, to help them out for their practical preparation. It is a common observation of all the teachers that, a student has lot of theoretical knowledge, not knowing exactly how to put it in proper words during viva-voce. Here is the place of this book, for every student... A must read book in Obstetrics and Gynecology for practical exams.

I appreciate the efforts and hard work of Dr Kanan Yelikar in bringing all the professors and teachers together, from renowned institutions, to contribute to this book.

I wish all the success to Dr Kanan Yelikar. I admire the attitude of a teacher in her heart of imparting knowledge to everyone, wherever and whenever possible.

Our very best wishes to you.

Prof Dr Nagnath Kottapalle
Vice-Chancellor
Dr Babasaheb Ambedkar
Marathwada University
Aurangabad (MS)

Preface

I am extremely happy to bring about my book on "Practical Cases in Obstetrics and Gynecology" for MBBS and above students. The idea of editing and publishing such a type of book, came in, when my daughter Ashwini was appearing for her III MBBS practical exams. When she demanded that I must teach her complete syllabus of Obstetrics and Gynecology in 2 hours time, which was next to impossible. And the idea of having a book totally, dedicated to practical cases was born.

I am thankful to all my contributors, who have been carefully chosen, for their vast experience in teaching who were kind enough for their timely contribution.

I would like to take this opportunity to thank the Dean, Govt. MCH, Aurangabad and the Director Medical Education, State of Maharashtra for permitting to go ahead with my endeavour.

Thanks to Dr Nagnath Kottapale, Vice Chancellor Dr. Babasaheb Ambedkar Marathwada University for his Foreword. It would not have been possible to publish this book without Jaypee Brothers Medical Publishers specially Mr Tarun Duneja who was kind enough to accept my book.

Last but not the least, my colleagues and postgraduates from the department of Obstetrics and Gynecology who supported me throughout the year (special mention of Dr Sonali Deshpande, Dr Ajeet Deshmukh, Dr Kapil and Dr Swati).

I am indebted to my family members Dr Avinash, Ashwini, Aditya and my mother in law Smt. Laxmibai Yelikar for bearing with my long absentees.

I am thankful to all my patients and my dear students past and present, who are the constant source of inspiration.

Kanan Yelikar

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Part I: Obstetrics

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PART 1: OBSTETRICS

1

Abortions

Abha Singh

1. What is abortion?

WHO definition is induced or spontaneous termination of pregnancy before 20 weeks of gestation.

2. What is the incidence of abortion?

About 10-20% of all pregnancies. 3/4th occur before 16 weeks and out of these 75% before 8 weeks.

3. What are the different common types of abortion met in clinical practice?

Common varieties of abortion are:

- Threatened
- Inevitable
- Incomplete
- Complete
- Missed

In developing countries incidence of septic abortions are also quite high approximately 10% of abortion requiring admission to the hospital.

4. What is the most common cause of first trimester abortion?

Genetic factors in the form of chromosomal abnormalities in the conceptus is the most common cause.

5. What are the common chromosomal abnormalities of the conceptus?

Autosomal trisomies occur in about 50% cases. The most common trisomy is 16.

6. What is blighted ovum?

Blighted ovum is the failure of development of fetal pole even after the gestational sac diameter of 2.5 to 3 cm or more on trans abdominal sonography.

7. At what gestational age, fetal pole should appear in the gestational sac normally (it is a sonographic diagnosis)?

4.5 weeks by TVS (18 mm gestational sac)
5.5 weeks by TAS (25 mm gestational sac)

8. How will you treat a patient diagnosed to have a blighted ovum?

If the duration of gestation is confirmed termination of pregnancy is advised after counselling, otherwise a repeat USG scan should be done after a week.

9. How will you diagnose a case of threatened abortion.

Threatened abortion is the one where process has started but has not progressed to inevitable, so clinically patients will present with bleeding per vaginum which is bright red in color, mild to moderate in amount with some degree of lower abdominal pain and backache. Bleeding precedes the pain.

On examination the general condition is good, external OS is closed and uterus corresponds to the period of amenorrhoea. It is important to subject the patient to USG for confirmation of cardiac activity.

10. What are the features of inevitable abortion? How will you treat it?

Continuous contraction and dilatation of OS is associated with inevitable abortion. Sonographically it shows gestational sac separated from decidua and in the process of expulsion.

According to weeks of gestation measures are used to complete the process of expulsion.

- If < 12 weeks, D and E or S and E
- If > 12 weeks, Syntocinon 10 units in 500 ml of NS, 40-60 drps/min.

If profuse bleeding is there but OS closed hysterectomy may have to be done (very rare).

11. Which is the most important prognostic factor on USG in threatened abortion.

Presence of fetal cardiac activity. It is associated in 98% cases with continuation of pregnancy.

12. What is incomplete and complete abortion?

When a part of conceptus is left inside the uterus it is known as incomplete variety. It is most commonly met with and is a dangerous entity as patient may present with shock due to continuous bleeding per vaginum.

Clinical features of incomplete abortion

- Continuous pain
- Bleeding P/V (at times profuse)
- Patulous external OS.

Complete abortion—when whole of conceptus is expelled enmasse with subsidence of pain and bleeding.

13. What are the sonographic criteria for missed abortion?

- Absence of cardiac activity
- Sac diameter 25 mm or > without a yolk sac/ embryo (TAS).
- Sac diameter 18 mm without yolk sac/embryo TVS

14. What are the features of septic abortion?

- Increase temperature 100.4°F per 24 hrs or/more
- Offensive vaginal discharge
- Lower abdominal pain and tenderness.

15. What are the common organisms associated with septic abortion?
A. Mixed infection is common. Organism associated are *Bacteroides*, *Streptococci*, *Cl. welchii*, *Cl. Tetani*, *E. coli*, *Klebsiella*, *Staphylococcus* and *Pseudomonas*.

16. How is severity of septic abortion classified?
It is classified in grades. Mildest being grade I.
Grade I—Infection limited to uterus.
Grade II—Infection involving tubes, ovaries and adjacent organ.
Grade III—Generalized peritonitis, endotoxic shock, acute renal failure.

17. What are the complications associated with septic abortion?
- Septicemia
- Hemorrhage
- Shock
- Bowel injuries
- Uterine perforation.

18. Indications of active surgery in septic abortion?
- Hemorrhage
- Presence of foreign body
- Intestinal injury
- Unresponsive peritonitis.

19. What are the basic investigations in any of the varieties of abortion?
- Urine analysis
- Complete blood count
- Serum quantitative B HCG
- Rh factor determination
- Pelvic USG.

20. How can cervical trauma be minimized?
Preoperatively
- Consider USG assessment of the gestational age
- Appropriate cervical preparation
- PG analogue should preferably be used.

Intraoperatively
- Grasp the cervix with TWO vulsellum tenacula
- Use graduated dilators
- Dilate against appropriate countertraction.

Management of cervical tear
- If small and not bleeding—no action
- If large amount of bleeding—hemostatic polyglactin sutures
- May require packing and admission for over night observation.

21. What is habitual or recurrent pregnancy loss?
Three or more consecutive abortions before 20 weeks is known as recurrent pregnancy loss.

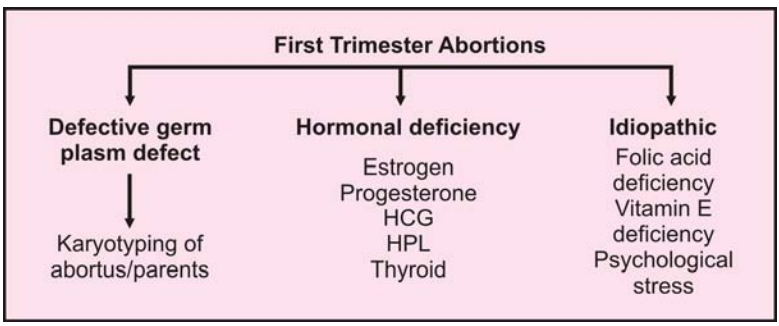
22. What causes are associated with recurrent pregnancy loss? **

Etiology of RPL

1. Genetic abnormalities (50% to 60%)
   - Autosomal trisomy 16, 21 and 22
   - Triploidy
   - Monosomy 45 x
   - Tetraploidy
   - Structural rearrangements.
2. Endocrine abnormalities 10 to 50%
   - Luteal phase defect, progesterone deficiency
   - Serum progesterone levels less than 15 ng/ml, indicates progesterone supplementation.
   - Thyroid deficiency
   - Diabetes
   - Increased androgen
   - PCOS.
3. Chorioamniotic separation 5 to 10%
4. Incompetent cervix 8 to 15%
5. Infections 3 to 5%
   Bacterial vaginosis and TORCH group of infections. Abortions occur around 14 weeks.
6. Abnormal placentation 5 to 15%
7. Immunological abnormalities 3 to 5%
   Anti phospholipid antibody syndrome
   Anticardiolipin antibody
   Lupus anticoagulant
   Alloimmune etiology is less common.
8. Uterine anatomic abnormalities 1 to 3%
   - Uterine synechiae
   - Mullerian abnormalities
   - DES exposure in uterus.
9. Unknown reasons.

23. How will you proceed to investigate a case of RPL?

I. *Interconceptional period*
(thorough medical, surgical and obstetric history)



II. *Trimester*

1. Blood sugar 1. Fasting 2. Post meal
   - VDRL
   - ABO grouping of husband and wife
   - Thyroid function
   - TORCH
2. Urine analysis
3. Dilator test for incompetence
4. Hysterocervicography
5. High cervical swab
6. Karyotyping

24. What are the surgical options for the treatment of cervical incompetence? Name the commonly used operations.

Commonly used surgical procedures are Shirodkar and McDonald. Other surgical procedures—Lash and lash, Page's procedure, Arias operations, Baden and Baden, Wurm procedure, Ritter and Ritter, Shirodkar's and McDonald's operation.

What is the success rate of McDonalds vs Shirodkar operation?

In both varieties 80-90%.

Which one is preferred and why?

MacDonalds, because it is technically easy.

When will you remove the stitch?

At 37 completed weeks of gestation or if patient goes in labor, whichever is earlier.

25. What is the optimal timing for cerclage operations?

A. It should be done preferably in 2nd trimester at 14-16 weeks, 2 weeks prior to the wastage of previous pregnancy.

26. What is the percentage of risk with increasing number of abortion and with increasing abortions.

Nos of abortion	*Subsent risk*
0	15%
1	19%
2	35%
3	47%

27. What are the contraindications of encirclage operation?

- IUD
- Congenitally abnormal fetus
- Bleeding P/V
- Intrauterine infections
- Uterine contraction
- Ruptured membranes
- Cervical dialatatiion > 4 cm effacement > 50%
- GA > 32 weeks.

2 Pregnancy Induced Hypertension

Shrinivas Gadappa

1. What are salient features in history taking in patient of hypertensive disorders in pregnancy?

Following are the important facts must be included in all cases of hypertensive disorders in pregnancy:

Mrs. _____ a _____ years old lady, married for _____ residing at _____ G.P.A. with _____ months amenorrhea presents with a C/o edema feet, face, fingers (specify the site) for _____ days.

Her L.M.P. was on _____ E.D.D. is on _____ her past cycles were every 28 days.

No H/ suggestive of heart disease
No H/ suggestive of liver disease
No H/ suggestive of deep vein thrombosis
No H/ suggestive of renal disease
No H/ suggestive of chronic hypertension

H/o headache, visual disturbances, auditory disturbances, epigastric pain (suggestive of severe pregnancy induced hypertension and impending eclampsia)

H/o pregnancy induced hypertension in mother of the patient (inherited as an autosomal recessive trait)

H/o suggestive of diabetes mellitus (predisposing factor)

H/o excessive enlargement of the uterus, suggestive of multiple pregnancies/ molar pregnancy

H/o pregnancy induced hypertension in past pregnancy.

H/o diminished fetal movements (suggestive of fetal compromise)

Examination

General examination is done as usual, with stress on the distribution and severity of edema, presence of pallor and its severity, and icterus, if any. The blood pressure is checked in right upper limb in 15^0 lateral position and sitting posture. The mean arterial pressure is calculated by using the formula:

$$\text{Mean arterial pressure} = \frac{\textit{Systolic pressure} + 2 \times \textit{diastolic pressure}}{3}$$

Obstetric examination is done as usual. Special attention is given to the number of fetuses present and evidence of intrauterine growth retardation.

Calculation:

Symphisofundal height (SFH), abdominal girth, height and weight of the patient, pondral index.

Investigations:

1. Hemoglobin
2. Blood group, VDRL, PPTCT
3. Urine analysis: Albuminuria, glycosuria, evidence of chronic renal disease.
4. Renal chemistry: blood urea nitrogen, serum creatinine, serum electrolytes serum uric acid (more than 4 mg/dl is suggestive or prognostic value, the fetal prognosis being inversely related to the level of uric acid)
5. Fundoscopy: To be repeated every week (for evidence of chronic hypertension as well as to grade the changes due to pregnancy induced hypertension)
6. Platelet count
7. Serum fibrinogen, fibrinogen-fibrin degradation products (to detect chronic intravascular coagulation)
8. Investigations to detect fetal growth retardation
9. Investigations to assess fetal well being.

2. How are hypertensive disorders in pregnancy classified?

In 2000, the National High Blood Pressure Education Program Working Group revised the classification system for this set of conditions. Four categories are now recognized for hypertension in pregnancy : (1) Chronic hypertension (2) Gestational hypertension (3) Preeclampsia and eclampsia, (4) Chronic hypertension with superimposed preeclampsia.

3. Define gestational hypertension?

Gestational hypertension is defined as per following criteria:

BP > 140/90 mm Hg without proteinuria for first time during pregnancy.

BP returns to normal < 12 weeks postpartum. Final diagnosis made only postpartum. May have other signs or symptoms of preeclampsia, for example, epigastric discomfort or thrombocytopenia.

4. Who are prehypertensives?

Individuals with systolic blood pressure 120 to 139 mm Hg or diastolic 80 to 89 mm Hg are considered as prehypertensives.

5. How is chronic hypertension defined?

BP > 140/90 mm Hg before pregnancy or diagnosed before 20 weeks gestation not attributable to gestational trophoblastic disease.

Or

Hypertension first diagnosed after 20 weeks gestation and persistent after 12 weeks postpartum is defined as chronic hypertension.

6. Are women with chronic hypertension considered high-risk pregnancies?

Yes. Because of poor placental vascular development and ongoing elevations of blood pressure, the pregnancy is at risk for intrauterine growth restriction, abruption and stillbirth. From a maternal perspective, the patient may develop superimposed preeclampsia and all of the associated consensus.

7. How will you define superimposed preeclampsia on chronic hypertension?

New onset proteinuria > 300 mg/24 hours in hypertensive women without proteinuria before 20 weeks of gestation.

A sudden increase in proteinuria or blood pressure or platelet count < 100,000/mm^3 in women with hypertension and proteinuria before 20 weeks' gestation is defined as superimposed preeclampsia on chronic hypertension.

8. What is the definition of preeclampsia?

Preeclampsia is defined by hypertension and proteinuria. For women who do not have chronic hypertension, an elevation of BP above 140 mmHg systolic or 90 mmHg diastolic meets the criteria. Proteinuria is defined as more than 300 mg of protein in a 24-hour urine collection.

9. What are the amendments made in new definition of preeclampsia?

In the past, an increase in blood pressure above baseline (a 30 mmHg increase in systolic or a 15 mmHg increase in diastolic) also met the blood pressure definition of preeclampsia, but this relative definition has been discarded in favor of the absolute definition of 140 mmHg systolic or 90 mm Hg diastolic

Proteinuria is technically defined by the outcome of a 24 hour urine collection. In practice however, the consistent presence of more than trace protein on urine dipstic correlates well with proteinuria of 300 mg in 24 hours. Therefore the diagnosis is commonly made without a formal 24 hours evaluation.

Edema of the face and hands used to be a component of the diagnosis of preclampsia. This criterion has been dropped because of its poor predictive value. Many pregnant women with normal blood pressure have hand and facial edema.

10. How often does preeclampsia occur? Which women are at greater risk?

Preeclampsia occurs in 6-8% of all live births. Risk factors include nulliparity, extremes of reproductive age (< 15 and > 35 years of age), history of preeclampsia in a first-degree female relative, history of preeclampsia in a prior pregnancy, diabetes, chronic vascular or renal disease, chronic hypertension and multiple gestation.

11. Do we know what causes preeclampsia?

No. Preeclampsia has been described since the time of the ancient Greeks. However, the cause remains unknown. We know that hypertension,

proteinuria and the other signs and symptoms of the illness are merely the outward manifestations of a systemic illness characterized by vasoconstriction and hypovolemia. All organs, including the fetoplacental unit, show evidence of poor perfusion.

12. What are some of the theories of the cause of preeclampsia?

Immunologic response: Inappropriate maternal antibody response to the fetal allograft results in vascular damage from the circulating immune complexes. This theory is supported by an increased prevalence of the disease in pregnancies with limited prior antigen exposure (young nulliparas) and in situations with increased fetal antigen (twins, molar pregnancy, hydropic pregnancies and diabetics with large placentas). Actual measurement of immune complexes has been inconsistent.

Circulating toxins: Vasoconstrictive substances reportedly have been extracted from blood, amniotic fluid, and the placenta in women with preeclampsia. Symptoms have been reproduced in some but not all animal studies.

Endogenous vasoconstrictors: Increased sensitivity to vasopressin, epinephrine and norepinephrine has been reported. Loss of normal third-trimester resistance to angiotensin II has also been noted.

Endothelial damage: Primary endothelial damage results in a decrease in prostacyclin production (potent vasodilator) and a relative increase in thromboxane A_2 (relative vasoconstrictor). Low-dose heparin or low dose aspirin may play a role in prevention, but the cause of the endothelial damage and prostaglandin change is unclear.

Primary disseminated intravascular coagulation: Microvascular thrombin formation and deposition have been noted, producing vessel damage especially in the kidney and in the placenta.

Abnormal trophoblastic invasion of uterine vessels

Dietary deficiency

Genetic factor: HLA DRW4—Maternal maladaptation to cardiovascular or inflammatory changes of normal pregnancy.

13. How is preeclampsia classified? What are the implications of the classification?

Preeclampsia is classified as mild and severe. Any of the following parameters classify a woman's preeclampsia as severe:

- Systolic blood pressure > 160 mmHg or diastolic blood pressure > 110 mmHg on two occasions at least 6 hours apart
- Proteinuria > 5 g/24 hr
- Oliguria < 500 cc/24 hr
- Cerebral or visual symptoms
- Epigastric or right upper quadrant pain
- Low platelets
- Elevated serum creatinine

- Elevated liver function tests
- Fetal growth restriction
- Pulmonary edema or cyanosis

The definition of mild preeclampsia is any preeclampsia that is not considered severe. There is no category of moderate preeclampsia.

The classification of severity is directly related to management and the decision about when to deliver the baby.

14. Enumerate the predictive tests for preeclampsia.

Currently there are no screening tests for preeclampsia that are reliable, valid and economical. Few selected tests are enumerated below:

1. Roll over test (Gant and colleagues 1974). Predictive value (true positive) 33%.
2. Uric acid: elevated uric acid levels > 5.9 mg / dl at 24 weeks.
   Positive predictive value 33%
3. Fibronectin—Elevated serum cellular fibronectin at 12 weeks.
   Positive predictive value 29%, Negative predictive value 98%
4. Coagulation activation ratio of PAI-1: PAI-2 is predictive (Chappell 2002)
5. Oxidative stress increases levels of *malondialdehyde*, increased levels of Homocystine.
6. Cytokines—Over 50 cytokines are present, of these, few are elevated in preeclampsia which include—interleukins, TNF-α
7. Placental peptides—Elevated levels of sFlt 1.
8. Fetal DNA—Fetal DNA in maternal serum.
9. Uterine artery Doppler velocimetry—evaluation at 12-18 weeks.
   Sensitivity for prediction is 78%
   Positive predictive value = 28%

Combined with inhibin A and activin A the predicative value is 86% if both are abnormal and 17% if both are unaltered.

15. What fetal and maternal risks are associated with preeclampsia?

Fetal risks include:

- Growth restriction
- Oligohydramnios
- Placental infarction
- Placental abruption
- Consences of prematurity (when maternal disease necessitates delivery remote from term)
- Perinatal death.

Maternal risks include:

- Central nervous system manifestations, including seizures and stroke
- DIC and its complications
- Increased likelihood of cesarean delivery
- Renal failure
- Hepatic failure or rupture
- Death

16. What are the basic management objectives for pregnancy complicated by preeclampsia?

Basic management objectives are:

1. Termination of pregnancy with least possible trauma to the mother and the fetus.
2. Birth of the infant who subsently thrives.
3. Complete restoration of health of the mother.

17. How will you monitor the patients of hypertension after hospitalization?

A systematic evaluation is instituted which includes the following:

1. Detailed examination followed by daily scrutiny for clinical findings such as headache, visual disturbances, epigastric pain and rapid weight gain.
2. BP reading in sitting position every 4 hrs, except between midnight and morning.
3. Measurement of proteinuria daily, at least every 2 days.
4. Daily weight record.
5. Measurement of serum creatinine, hematocrit, platelets and serum liver enzymes, the frency to be determined by the severity of hypertension.
6. Fetal growth and AFI, Doppler sos.

18. Describe the management of preeclampsia?

Delivery of the fetus "cures" preeclampsia. With delivery, signs and symptoms of preeclampsia resolve, although the time required for resolution is variable. The difficulty in therapy is deciding when to deliver the baby. Traditionally, all term pregnancies with preeclampsia - mild or severe - were delivered. For preterm pregnancies, those with severe disease were delivered and those with mild disease were managed conservatively with bedrest, antihypertensives and close fetal and maternal surveillance.

Recently there have been efforts to extend preterm pregnancies complicated by severe preeclampsia rather than proceeding with immediate delivery. Administration of a short course (48 hours) of steroids to the mother confers significant benefit to premature newborns. Therefore, if preeclampsia develops before 32 weeks, even a short period of conservative management prior to delivery may be beneficial. Conservative management of severe preeclampsia, in order to gain greater fetal maturity, should only be attempted in carefully selected patients and should be managed by experienced obstetrician. These women should be hospitalized and must be observed closely for signs of worsening maternal disease or evidence of fetal compromise.

Obviously, at term the decision is easy to make, whether the preeclampsia is mild or severe. The difficulty arises when preeclampsia is remote from term. The degree of severity and the fetal gestational age are taken into consideration, and either the patient is delivered or is placed at bed rest. With bed rest therapy, close maternal and fetal surveillance is performed until the pregnancy reaches term or the degree of severity worsens, dictating the need to deliver.

19. What is the role of expectant management in severe preeclampsia?

Women with severe PIH are usually delivered without delay. This approach advocates conservative or "Expectant" management in selected group of women with the aim of improving infant outcome without compromising safety of mother. Expectant management includes treatment with labetolol, nifidipine and $MgSO_4$, with vigilant monitoring. Indications for delivery include uncontrollable BP, fetal distress, placental abruption, renal function deterioration, HELLP Syndrome, persistent severe symptoms or an attainment of 34 weeks of gestation. Average pregnancy prolongation in different studies were 8 to 15 days with neonatal morbidity and few maternal complications; no maternal death. This expectant management has to be thought twice before implementing, should be strictly in expert judicial high tech institutes with 24 hrs emergency services available.

20. How should delivery be accomplished in patients with preeclampsia?

There is no advantage of cesarean delivery over vaginal delivery for preeclampsia. Therefore, delivery route should be based on obstetric indications. An indication for cesarean section may be the inability to accomplish a vaginal delivery within a fixed, specified time, governed by maternal condition.

21. What is home health care in preeclampsia?

Further hospitalization is not warranted if hypertension abates within few days. Most women with mild hypertension without proteinuria are managed at home. These women should be instructed in detail about reporting symptoms. Home blood pressure and urine protein monitoring and frent evaluation by a visiting nurse is mandatory, commonly practiced in well, intelligent groups.

22. What are the predictors for declining / worsening maternal well being and fetal well-being?

Indicators for declining maternal well-being are:

1. Increased BP
2. Worsening symptoms (i.e. WS)
3. Hemoconcentration (increased creatinine, urea, uric acid)
4. Increasing proteinuria
5. Worsening of DIC profile.

Indicators of declining fetal well-being are:

1. Nonreactive NST
2. Positive contraction stress test
3. Declining BPP
4. Serial USG shows growth parameter slowing or arrest of growth
5. Decreased fetal movement.

23. What is the role of antihypertensives in preeclampsia?

Mild elevations in blood pressure usually are not treated with antihypertensive. With more marked elevations (diastolic > 110 mmHg or a mean arterial pressure > 125 mmHg), medications with rapid onset, such as

hydralazine and labetalol, are used intravenously (Generally such management is undertaken while also proceeding with delivery, due to the severity of the disease).

Diuretics are generally not used as first-line treatment because preeclampsia is characterized by vasoconstriction and intravascular depletion which are worsened by diuretics. As for other antihypertensive agents, work has shown that treatment of patients with mild-to-moderate hypertension (i.e., 90-110 mmHg diastolic pressure) does not decrease perinatal morbidity or mortality. Therefore, antihypertensive therapy is not usually used. In severe hypertension (> 110 mmHg diastolic pressure) alpha methyl dopa 1-2 gm/day, nidifipine 10 mg tid, max dose of 120 mg. Labelotol 100 mg bid is used. Maximum dose is 300 mg in 24 hrs. More than likely, delivery needs to be undertaken in this circumstance, and rapid-acting antihypertensive agents (i.e., intravenous hydralazine or labetalol) are used to control severe hypertension during labor.

In a patient with known chronic hypertension whose elevated blood pressure is believed to be due to underlying disease rather than preeclampsia, an increase in antihypertensive therapy may be appropriate.

24. What is role of labetolol in treatment of preeclampsia?

Women on labetolol have significantly lower MAP. But there is no difference between two groups in the form of prolongation of pregnancy and gestational age at birth. However incidence of growth restriction is twice in labetolol group compared to other antihypertensive agents. It is a good drug to control acute hypertensive crisis.

25. How will you start seizure prophylaxis in severe preeclampsia?

Prevention of seizures done by following regimen:

Pritchard regimen

4 gm $MgSO_4$ as a 20% solution intravenously at a rate not to exceed 1 g/min. Followed promptly with 10 gm of 50% $MgSO_4$ one-half (5 gm) injected deeply in upper outer quadrant of both buttocks through a 3 inch long, 20 gauge needle.

Every 4 hrs thereafter 5 gm of 50% solution of $MgSO_4$ injected deeply in upper outer quadrant of alternate buttock but after careful monitoring. **Zuspan regimen.**

4-6 gm $MgSO_4$ IV bolus slowly followed by 2 gm/hr maintenance for 24 hrs after delivery after ensuring that:

a. Patellar reflex present
b. Respiratory rate > 14/min
c. Urine output > 30 ml / hr or 100 ml in 4 hrs.

26. Should all women with gestational hypertension or preeclampsia be treated with $MgSO_4$ for seizure prophylaxis?

The purpose $MgSO_4$ is to reduce the likelihood of seizures. Seizures occur less frently in women with gestational hypertension and mild preeclampsia compared to women with more severe disease. In addition, like any medication, $MgSO_4$ can have side effects, the most significant of which is

respiratory compromise due to respiratory suppression and pulmonary edema. Because of this risk and given the lower risk of seizures in women with mild disease, many clinicians reserve anticonvulsive prophylaxis for women with severe preeclampsia, HELLP syndrome, or eclampsia.

27. What are the serum magnesium levels and their effects?

Dose	*Effect*
5-8 mg / dl	Therapeutic level
10 mg / dl	Loss of DTR
15 mg / dl	Respiratory paralysis
25 mg / dl	cardiac arrest

Intravenous calcium gluconate is the antidote for $MgSO_4$ toxicity.

28. Is there a way to prevent preeclampsia?

Numerous interventions have been attempted. Dietary manipulation with decreased sodium intake or increased calcium intake and pharmacologic therapy with prophylactic low-dose aspirin have been extensively studied with randomized controlled trials. Unfortunately neither of these interventions have been able to reduce the incidence of preeclampsia.

29. Does preeclampsia recur in subsent pregnancies?

Yes. If preeclampsia occurs in the first pregnancy there is a 25% chance of recurrence in subsent pregnancies. The recurrence rate appears to be affected by gestational age at onset in the first pregnancy, severity, underlying maternal diseases and underlying obstetric diseases. Women who develop preeclampsia early in pregnancy, or who develop severe preeclampsia, those with chronic medical conditions (e.g., chronic hypertension, renal disease) and those with no apparent fetal contribution (such as fetal aneuploidy) are at greater risk to develop preeclampsia in the future.

Multiparous patients who have had preeclampsia have a recurrence rate of up to 50% in subsent pregnancies.

30. Can women with a pregnancy complicated by preeclampsia take birth control pills after delivery?

Yes. Preeclampsia, especially in a primiparous patient, does not contraindicate the use of oral contraceptives after delivery.

31. How will you define eclampsia?

Seizures that cannot be attributed to other causes in women with preeclampsia is defined as eclampsia.

32. What is the management of the patient during convulsion?

Management during convulsion:

a. In the premonitory stage, a mouth gag is placed in between the teeth to prevent tongue bite and should be removed after the clonic phase is over.
b. The air passage is to be cleared off the mucus with a mucus sucker. The patient's head is to be turned to one side and the pillow is taken off. Raising the footend of the bed facilitates postural drainage of the upper respiratory tract.

c. Oxygen is given until cyanosis disappears.
d. Institution of anticonvulsants and other medication.

33. How is delivery accomplished in eclamptic patients?

As with preeclampsia, obstetric indications are used to determine route of delivery. It is not unusual to encounter evidence of fetal compromise in eclamptic patients, but this is not necessarily an indication for cesarean delivery. In general, fetal resuscitation is best accomplished in utero by controlling the maternal state. Stabilizing the patient (i.e. airway, oxygen, circulation and control of seizure activity) improves fetal status and subsently neonatal outcome. Labor can then be initiated and vaginal delivery accomplished, assuming no obstetric indications for a cesarean delivery.

34. Is $MgSO_4$ used for eclamptic patients?

In the past there have been advocates for other agents, particularly phenytoin. However, a randomized controlled trial in women with eclampsia clearly favored $MgSO_4$ over phenytoin for recurrent seizure prophylaxis.

35. What is HELLP syndrome?

HELLP is an acronym for a syndrome of **H**emolysis, **E**levated **L**iver function tests and/or **L**ow **P**latelets. HELLP syndrome is thought to be a subcategory of severe preeclampsia. Patients may or may not have other signs of preeclampsia. HELLP syndrome often has a rapidly decelerating downhill course. Most clinicians deliver infants expeditiously regardless of the gestational age.

36. What causes midepigastric pain in HELLP syndrome?

Liver capsule distention produces midepigastric pain, often with associated nausea and vomiting. Liver capsule distention can lead to hepatic rupture with poor maternal and fetal outcome.

37. What are the Eden's prognostic criteria of eclampsia?

Immediate: Once the convulsion occurs, the prognosis becomes uncertain.
Prognosis depends on many factors and the ominous features are:

1. Long interval between the onset of fit and commencement of treatment (late referral)
2. Antepartum eclampsia specially with long delivery interval
3. Number of fits more than 10
4. Coma in between fits
5. Temperature over 120° F with pulse rate above 120 / minute.
6. Blood pressure over 200mm Hg systolic.
7. Oliguria (< 400 ml/24 hours) with proteinuria > 5 gm / 24 hours
8. Non-response to treatment.
9. Jaundice.

38. What are the criteria for diagnosis of aggressive inpatient management?

Aggressive inpatient management includes:

Diagnostic criteria

1. Mild or severe preeclampsia > 37 weeks' gestation

2. Severe preeclampsia; < 26 weeks' gestation
3. Severe preeclampsia; 26-34 weeks' gestation, when associated with maternal jeopardy:
   i. Severe persistent headache
   ii. Persistent visual changes
   iii. Hepatocellular injury
   iv. Thrombocytopenia or other evidence of DIC
   v. Pulmonary edema
   vi. Abruptio placentae
4. Severe preeclampsia; 26-34 weeks' gestation, when associated with fetal jeopardy:
   a. Repetitive severe variable decelerations
   b. Repetitive late declarations
   c. Repetitive BPP < 4
   d. Oligohydramnios [amniotic fluid index (AFI) < 4 cm]
   e. IUGR (estimated fetal weight < fifth percentile)
5. Chronic hypertension with superimposed preeclampsia at any gestational age.
6. Eclampsia or HELLP syndrome at any gestational age

39. What are the guidelines for aggressive inpatient management?

Guidelines

1. Maintenance of diastolic BP between 90 and 100 mm Hg. Further reduction of BP jeopardizes placental blood flow. Appropriate antihypertensive medications include:
   a. Hydralazine (direct arteriolar vasodilator), which causes baroreceptor sympathetic stimulation [increasing heart rate (HR) and cardiac output (CO)], thus preserving placental blood flow.
   b. Labetalol (nonselective B-blocker), which preserves utero-placental blood flow.
2. Prevention of convulsions with intravenous (IV) magnesium sulfate
   a. Administration of loading dose of 5 g IV over 20 minutes, and maintenance infusion at 2 g/hr. The maintenance IV infusion should be given for 24 hours after delivery.
   b. Watching for clinical evidence of magnesium toxicity
   c. Absence of toxicity is ensured as long as deep tendon reflexes are obtainable.
   d. IV calcium gluconate is the antidote for magnesium toxicity.
3. **Initiation of delivery**: Labor can be induced anticipating vaginal delivery if the patient is stable and there are no contraindications. Otherwise, cesarean delivery is indicated.

40. What are the diagnostic criteria and treatment modality for conservative inpatient management?

Conservative inpatient management is appropriate in the following cases:

a. Mild preeclampsia that is remote from term (> 37 weeks).

Guidelines include:

i. Monitoring BP every 4 hours
ii. Performing a daily urine dipstick for protein
iii. Performing twice-weekly 24 hour urine protein measurements.
iv. Performing weekly liver function tests and electrolyte levels.
v. Initiating delivery if criteria for severe preeclampsia are met.

b. Severe preeclampsia in carefully selected cases.

1. All of the following criteria must be met.

a. Gestational age > 26 weeks but < 34 weeks
b. BP persistently > 160/110 mm Hg
c. Absence of fetal jeopardy
d. Absence of maternal jeopardy

2. Guidelines include:

a. Intensive maternal and fetal monitoring in a tertiary perinatal center.
b. Cautious volume expansion
c. Aggressive antihypertensive therapy (e.g. hydralazine, labetalol)
d. Anticonvulsant therapy (e.g. magnesium sulfate)
e. Corticosteroids to enhance fetal lung maturity.

3. Initiation of delivery if maternal or fetal deterioration occurs

41. What are the diagnostic criteria and treatment modality for conservative outpatient management?

Conservative outpatient management.

1. Patient selection criteria:
   a. Transient hypertension (i.e., BP in the mildly elevated range, no proteinuria)
   b. Uncomplicated chronic hypertension (without superimposed preeclampsia)
2. Guidelines include:
   a. Bed rest in the left lateral position.
   b. Home BP monitoring
   c. Twice-weekly outpatient visits.
   d. Initiation of delivery if maternal or fetal deterioration occurs.

42. What are the causes of maternal deaths in eclampsia?

Cause of maternal death is:

1. Cardiac failure
2. Pulmonary edema
3. Aspiration and/or septic pneumonia
4. Cerebral hemorrhage
5. Anuria
6. Pulmonary embolism
7. Postpartum shock
8. Puerperal sepsis.

43. What are the causes of perinatal mortality in eclampsia?

Fetal: The perinatal mortality is very high to the extent of about 30-50%.

The causes are:

1. **Prematurity –** Spontaneous or induced.
2. **Intrauterine asphyxia** due to placental insufficiency arising out of infraction, retroplacental hemorrhage and spasm of uteroplacental vasculature
3. **Effects of the drugs** used to control convulsions
4. **Trauma** during operative delivery.

44. Does preeclampsia have risk of hypertension in later life?

No. Preeclampsia does not increase the risk of hypertension later in life. An exception may be women who have recurrent preeclampsia, which may suggest unrecognized chronic hypertension or other underlying maternal diseases.

45. MCQ 1 and 2

For each of the following cases, select the most appropriate management option.

Case 1: A 38-year old woman gravida 1, presents for a routine visit at 39 weeks gestation. Blood pressure is noted to be persistently 140/90 mm Hg, and urine protein is +2. She is completely asymptomatic and her physical examination is otherwise unremarkable. Cervix is 2 cm dilated, 90% effaced, with the fetal vertex at 0 station.

Case 2: A 25-year old woman gravida 2 para 0 presents at 33 week's gestation for a routine visit. In the office, her blood pressure is noted to be 150/100 mm Hg, and urine protein + 3. She is otherwise asymptomatic.

Options:

a. Immediate cesarean section
b. Induction of labor
c. Admission to hospital for observation
d. Outpatient observation

Ans: The answers are Case 1-B, Case 2-C.

The first patient has a diagnosis of mild pregnancy-induced hypertension (PIH) at term. Because she does have proteinuria, she may be further classified as preeclampsia, she may be further classified as preeclamptic. The indicated treatment of PIH of any severity at term is delivery. Because her cervix is favorable, induction of labor is the preferred method of delivery. Induction can be started with intravenous oxytocin, and parenteral magnesium sulfate should be used for seizure prophylaxis.

The second patient presents with a more difficult problem because she is preterm. She appears to have PIH on examination, but does not at this point meet criteria for severe disease. With mild disease in a preterm patient, observation and evaluation for severe disease is indicated. Because of the serious complications that can occur, the patient is best managed in the hospital until sufficient evaluation to exclude severe PIH is completed. If further evaluation reveals severe disease, delivery is indicated.

3

APH

Kanan Yelikar, Sonali Deshpande

1. What is APH?

A. Antepartum hemorrhage is defined as any bleeding from and into the female genital tract between fetal viability (28 weeks) and delivery of the fetus.

2. What is perinatal death?

A. Perinatal death is late fetal and early neonatal death.

3. What is the incidence of APH?

A. 2-5% is the incidence of APH.

4. What are the causes of APH?

A. Placenta Previa—31%
Abruptio placenta—22%
Undetermined– 47%

5. Define placenta previa?

A. Placenta that is implanted entirely or in part in the lower uterine segment

6. What are the different types of placenta previa?

1. Major (Type 2 posterior 3 and 4)
2. Minor (Type 1 and 2 anterior)

Type	*Description*
Low lying placenta previa	Placenta is in the lower uterine segment but the lower edge does not reach the internal OS (Stops short by 3 cm.
Marginal placenta previa	Lower edge of the placenta reaches internal OS but doesn't cover it.
Partial placenta previa	Placenta covers the internal OS asymmetrically
Total placenta previa	placenta covers the OS symmetrically

7. What are the etiological factors of placenta previa?

1. Damage to the endometrium or the myometrium predisposes a low implantation and subsent development of placenta previa, e.g. previous uterine surgery or uterine infection.

Increased risk of placenta previa 1.2% after one or more LSCS as compared with non scared uterus (25%)

2. Abnormal fetal presentation and congenital malformations are strongly associated with placenta previa.
3. Increasing maternal age, parity multiple gestation, anaemia, closely associated with placenta previa.

8. Theories to explain placenta previa

1. Drooping down theory—fertilized ovum drops and is implanted in the lower segment may be the cause.
2. Persistence of chorionic activity in the deciduas capsularis.
3. Defective deciduas.
4. Big surface of placenta.

9. What is the clinical presentation of placenta previa?

The two classical presentation of placenta previa are:

a. Painless, sudden onset, causeless and recurrent bleeding. The 1st episode is usually not severe but on some occasion it may be severe.
b. Fetal malpresentation in late pregnancy.

10. How will you diagnose placenta previa?

In current obstetrics the diagnosis of placenta previa is made on the basis of a routine ultrasound scan. In symptomatic patients sudden onset, painless bleeding may be affecting the general condition of the patient.

Obstetrics examinations—uterus soft and nontender. There is typically high floating head or malpresentation and the fetal heart rate is usually normal.

- P/S exam is done to rule out local causes of APH.
- P/V is contraindicated.
- Transvaginal USG is superior to transabdominal and do not report any hemorrhagic complication due to the use of TVS
- MRI—Expensive but excellent method as both placental edge and cervical canal can be readily visible.

11. What are the M/M options for placenta previa? M/M depends on the stage of pregnancy and extent of hemorrhage.

A. Expectant M/M

Care comprises judicious noninterference and intensive monitoring and is called Johnson and Macafee protocol.

The prerequisites are

1. Gestation less than 37 weeks.
2. Maternal and fetal condition stable.
3. Patient not in labor.

Protocol

1. Complete bed rest with bedside toilet facilities. IV diazepam 5 mg to improve compliance.
2. Blood should be grouped, cross-matched and reserved for the patient at all times.

3. Iron, calcium supplementation should be continued. Laxative may be given to avoid strain at stools.
4. The patients PR, BP and FHR are monitored till her condition is stable. Hb estimation at regular interval.
5. USG examination preferred for placental location.

The expectant management should extend for more than 1 week in duration to call it successful.

B. Definitive Management

Indication

1. First bout of bleeding after 37 completed weeks
2. Successful conservative t/t. brings the patient up to 37 weeks
3. Bleeding that makes vitals unstable
4. Foetal distress/fetal death.

Protocol—Resuscitation of patient with

a. Type 1 to Type 2 anterior → artificial rupture of membrane (ARM) + pitocin (enhancement of labor)
b. Type 2—post. and Type 3 and Type 4—CS.

12. What are the different difficulties that are encountered during LSCS?

1. As the lower segment is vascular, the dilated vessels should be ligated and incised in between when the incision is made.
2. In anteriorly situated placenta, the placenta may have to be cut or separated to deliver the baby. All these manipulation require great dexterity and speed.
3. Placental bed oozing requires compression and placental bed hemostatic sutures.
4. PPH—Obstetric hysterectomy may be required.
5. Lateral extension of incision
6. Removal of placenta may be difficult due to placenta-accreta.

13. What is Stallworthy's sign?

A. Slowing of FHR on pressing the head down into the pelvis which soon recovers promptly when the pressure is released is S/O the presence of low lying placenta specially of post type.

14. What are the different complication of placenta previa?

Maternal:

- APH with shock
- Preterm labor
- Malpresentation
- Cord prolapse
- Increased operative interference
- PPH
- Sepsis
- Anemia
- Fetal

- Prematurity
- Asphyxia
- IUFD
- Birth injuries.

15. Why PP is more prone for PPH?

1. Interface retraction of LUS on which placenta is situated
2. Trauma to LUS and cervix due to extreme softness.
3. Occasional morbidly adherent placenta.

16. What do you mean by migration of placenta?

Early in pregnancy the placenta appears to cover the internal cervical os but as the uterus enlarges, the lower uterine segment develops and placenta moves in cephalad direction. This is called migration of placenta. Repeat USG at 30 weeks of gestation will give the complete Idea about the migration.

17. What is double set up examination?

In some symptomatic patient after 36 weeks when transabdominal USG is not capable of accurately diagnosing placenta previa, this double set up examination is used.

Keeping every thing ready for LSCS, P/V examination is performed to determine the placental localization.

But because of TVS, this procedure is obsolete in obstetrics.

18. How to evaluate the severity of bleeding?

a. In mild bleeding (patient had lost less than 15% of her intravascular volume)—HO change in vitals urine output).
b. Moderate bleeding (blood loss between 15-30%) tachycardia, hypotension, c/o. Inadequate circulatory volume (pallor thirst clammy extremities).
c. Severe bleeding (> 30 to 40% of her blood volume) S/O hypovolemic shock oliguria, anuria apathy, agitation.

19. Which is choice of Tocolytics in placenta previa?

T. Nifedipine, 10-20 mg 8 hrly every 4 to 6 weeks.

20. What do you mean by abruptio placenta?

Complete/partial separation of the normally situated placenta prior to the birth of the foetus.

21. How will you classify abruptio placenta?

Grade I	Retrospectively diagnosed Blood loss 150-500 ml Fetus is not at risk.
Grade II	APH is accompanied by the classic features of the abruption placenta Blood loss may be more than 500 ml Fetus at risk.
Grade III	APH accompanied with classic features of the abruption placenta. Fetus death Presence/absence of coagulopathy.

22. What are the clinical features of abruption placenta?

Painful uterine Bleeding associated with features of toxaemia and maternal condition is out of proportion to the visible blood loss is concealed variety.

Height of uterus may be more than period of amenorrhoea uterus tense, tender, rigid FHS usually absent.

On USG—S/o separation of placenta situated in upper segment.

23. What are the different etiological factors of abruption placenta?

1. Maternal hypertension and vascular disease.
2. High parity, poor nutrition especially folic acid deficiency (not proved)
3. Maternal drug abuse—cocaine smoking.
4. Sudden decrease in the height of uterus (in hydramnios)
5. Trauma, kick, ECV.

24. What are the different varieties of abruption placenta?

a. Revealed—Following separation of placenta, the blood insinuates downwards between the membranes and the deciduas. Ultimately the blood becomes visible in the cervical canal.
b. Concealed—The blood is collected in between the membranes and decidua. Blood is prevented from coming out of the cervix by presenting part. Blood percolates in the amnictic sac after rupturing the membranes.
c. Mixed—Part of the blood is collected inside and a part is expelled out.

25. What is couvelaire uterus?

This condition is associated with severe variety of abruptio placentae. The uterus becomes dark port wine color which may be patchy or diffuse due to sequestration of blood in uterine myometrium.

26. What are the investigation that you should perform in case of abruption placenta?

Hb%, urine →, Blood group and Rh typing, coagulation profile, LFT, KFT.

26. What is the role of USG abruption placentae?

1. It may help to locate RP clot in slight vaginal bleeding when patients general condition is stable.
2. May show FHA and presentation in severe abruption.

27. How to estimate the blood loss in abruption placenta?

Blood loss is calculated by formula (weight of RP clot in gm x 3)

28. What are the objectives in the t/t of abruption placenta?

According to Pritchard's:

1. Keep hematocrits of at least 30% and
2. Urinary output of at least 30 ml/hr.

29. What are the complications of abruption placenta?

Maternal	Hemorrhagic shock
	DIC
	ARF
	PPH
	Sepsis

Fetal	IUFD
	Prematurity
	Fetal distress

30. What are the bedside tests for monitoring blood coagulation disorders?

a. Clot observation test (Weiner) 5 ml of venous blood is placed in a 15 ml dry test tube and kept at 37°C.

Usually, clot forms in 12-30 min and clot retract and remain firm at the end of one hour.

b. No clot after 30 minutes → Fibrinogen < 100 mg/ dl

c. Clot formed but disintegrated within 30 minutes clot lysis → Excessive fibrinolytic activity

d. Clot retraction denotes adequate functioning of intact platelets and divalent cations. Normal clot should remain intact for at least 48 hrs. If it dissolves within 24 hrs fibronolysis is more. It means plasma fibrinogen level is less than 100 mg/dl.

31. Why DIC is common in abruption placenta?

Due to abruption, liberation of thromboplastin leading to the consumption of platelets and coagulation factors (consumptive coagulopathy) and intravascular coagulation.

$$\text{Abruption} \xrightarrow[\text{Intravascular coagulation}]{\text{Thromboplastin}} \text{Consumptive coagulopathy}$$

The fibrin may be converted to FDP with plasmin causing defective hemostasis.

Thus depending on the extent of placenta separation, DIC can cause coagulation failure, renal cortical/tubal necrosis and microangiopathic hemolysis.

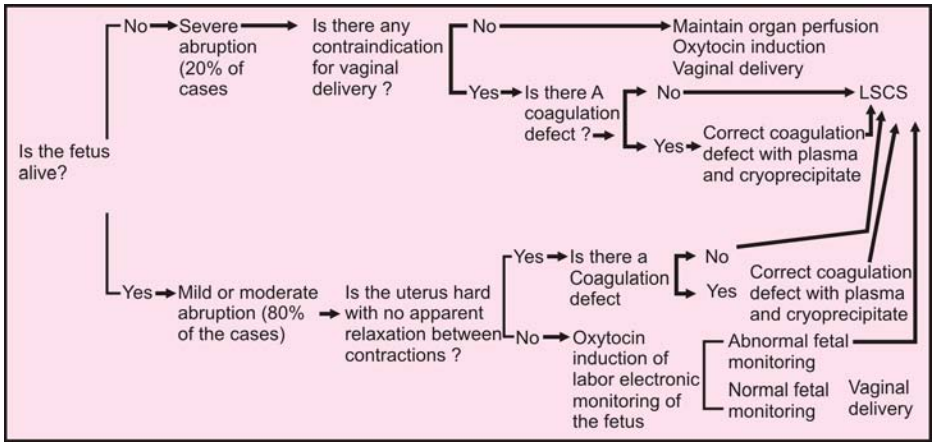
32. For fetal demise, now much placenta should be detached?

For fetal demise, the placental detachment is usually greater than 60%

33. Why there is severe abdominal pain in abruption placenta?

Abdominal pain is due to the RP clot causing intravasation of blood and disruption of myometrial fibres.

34. How will you manage abruption placentae?



4 Anemia in Pregnancy: Modern Aspects of Diagnosis and Therapy

Sunita Mittal, Nupur Gupta

Anemia is the most common medical disorder affecting pregnancy especially in developing countries, and contributes significantly to maternal morbidity and mortality. It is responsible for 40-60% of maternal deaths in non-industrialized countries and causes direct and indirect deaths from cardiac failure, hemorrhage, infection and preeclampsia.

DEFINITION

Anemia is characterized by a hemoglobin concentration of less than 11 gm% (7.45 mmol/l), (in developing countries, 10 gm%) and hematocrit of less than 0.33.

ICMR defines severity of anemia depending on the level of hemoglobin.

Category	*Severity of anemia*	*Hemoglobin level (g/dl)*
1	Mild	10.0-10.9
2	Moderate	7.0-10.0
3	Severe	< 7.0
4	Very severe (decompensated)	< 4.0

PHYSIOLOGICAL ANEMIA OF PREGNANCY

The term "*physiological anemia of pregnancy*" results due to hemodilution secondary to an increase in plasma volume, where hematocrit drops by 3 to 5 percentage points, plasma volume expands by 46 to 55% whereas red cell volume expands by 18 to 25%. Although the maternal red blood cell mass also increases during gestation, its expansion and the expansion of the plasma volume are not synchronous. Thus, hemoglobin concentrations and hematocrit declines throughout the first and second trimesters, reach a nadir early in the third trimester, and then rise again nearer to term.

IMPACT OF ANEMIA ON PREGNANCY

Anemia is a risk factor for both mother and fetus. In India, anemia is the second most common cause of maternal deaths, accounting for 20% of total maternal deaths. Deaths from severe anemia result from heart failure, shock, or infection due to lowered resistance to disease. There is an increased susceptibility to

infection and increase in incidence of asymptomatic bacteriuria, UTI with megaloblastic anemia.

Anemia diagnosed early in pregnancy is associated with increased risks of **low birth weight** (LBW) and **preterm delivery**. Stillbirth was not associated with mild anemia (10.0 to < 11.0 and 9.5 to < 10.5 g dL) or high hemoglobin (=14.6 g dL in either trimester) in either the first or second trimester of pregnancy.

Moderate anemia (9.0 to < 10.0 and 8.5 to < 9.5 g dL) measured before 28 weeks' gestation was significantly associated with an increased risk of stillbirth among non-black women but not in black women.

Low intake of dietary folate < 240 µg/d leads to 2 fold increase in PTL and low serum folate at 28 weeks causes a 10 fold increase in LBW infants. Complications of megaloblastic anemia may manifest as abortion, prematurity, dysmaturity and fetal malformations.

IRON STATUS OF INFANTS AT BIRTH

Iron is transported actively across placenta; fetus achieves normal hemoglobin and serum iron levels despite iron deficiency anemia (IDA) in mother, but fetal iron stores are lower; therefore they are predisposed to anemia 6 times more, later in infancy.

APPROACH TO DIAGNOSIS OF ANEMIA IN PREGNANCY

- History of nonspecific symptoms such as dyspnoea on exertion, weakness, palpitation, ankle swelling, headache, worsening of preexisting angina, tinnitus, taste disturbances, dysphagia, skin and nail changes, lack of appetite, etc.
- Related to underlying disorder are poor dietary history, recurrent pregnancy at short intervals, gastrointestinal bleeding or hematuria, previous history of anemia, repeated blood donations (> 3 per year), Intrauterine devise usage, NSAID abuse, family history of thalassemia, etc.

PHYSICAL EXAMINATION

Signs of anemia include pallor of skin, mucous membranes and palms, koilo/platynychia, cheilosis, glossitis, etc. and clinical decompensation is associated with altered mental state, apathy, pallor, tachycardia, tachypnea, increased JVP, heart murmurs, ankle edema, postural hypotension, etc.

LABORATORY APPROACH FOR DIAGNOSIS OF ANEMIA

Investigations are tailored to detect the type of anemia and probable cause for it. A CBC (complete blood count) TLC, DLC, TRBC, PCV, Hb, RBC indices, total platelet count and peripheral smear are done in all and the rest are individualized based on clinical suspicion and initial reports.

I. Peripheral smear: for blood cell morphology and parasites like malarial parasite. In anemia red blood cells show hypochromia, anisocytosis, poikilocytosis, microcytosis or polychromasia. Differential diagnosis of hypochromic anemia is depicted in Table 4.1. Abnormal cells like premature

red cells (normoblasts, megaloblasts, polychromasia) may be present. White blood cells show hypersegmented neutrophils (megaloblastic anemia) or leucopenia (aplasia, hypoplasia of bone marrow) and thrombocytopenia is present in aplastic or megaloblastic anemia. Possibility of thalassemia is suspected if microcytosis is more severe than expected or there is presence of basophilic stippling and target cells.

Table 4.1: Criteria for physiological anemia

Hemoglobin	10 gm%
Total RBC count	32 million/mm^3
RBC morphology	Normal morphology on peripheral smear
PCV (Hematocrit)	30%

Table 4.2: Impact of anemia on mother and fetus

Mother	*Fetus/placenta*
Reduced blood reserves	Prematurity
Low exercise and mental performance	Preterm labor
Cardiovascular strain	Amnion rupture
Tiredness	IUGR
Reduced immune function	Abnormal trophoblast invasion
Infection	Fetal programming
Negative thermoregulation	Diseases in newborn
Increased risk of blood transfusion	

II. Quantitative measurement

a. Hemoglobin by cyanmethoglobin method (most accurate), Sahli's method (most commonly used), alkali hematin, taliquist and copper sulphate method.

b. **Hematocrit (packed cell volume—PCV):** The value is given as a percentage of red blood cells in a volume of blood. Normal values are as follows: 1st trimester: 35–46%, 2nd trimester: 30–42%, 3rd trimester: 34–44% and Postpartum: 30–44%.

c. Normal RBC count: 4.2–5.4 million RBCs per µL

d. Red blood cell indices

$$\text{MCV} = \frac{\text{PCV (\%)} \times 10}{\text{RBC } (10^{12}/\text{l})}$$

Normal value—80-100 Fl

$$\textbf{MCH} = \frac{\text{Hb (g/dl)} \times 10}{\text{RBC } (10^{12}/\text{l})}$$

Normal 28-32 pg

$$\textbf{MCHC} = \frac{\text{Hb (g/dl)} \times 100}{\text{PCV}}$$

Normal 32-36 gm/dl (%)

e. Reticulocyte count—Normal 0.5-2.0 %

$$\text{Reticulocyte \%} = \frac{\text{No. of reticulocytes}}{\text{1000 RBC observed}} \times 100$$

Reticulocyte count is representative of bone marrow condition.

High:

1. Hemolytic anemia
2. After acute hemorrhage
3. Pernicious anemia or iron deficiency anemia after treatment.

Low:

1. Aplastic anemia
2. Iron Deficiency anemia before treatment
3. Pernicious anemia before treatment
   states (B_{12}, folic acid, or Fe deficiency).

f. ESR: Mild rise is often seen in anemia.

III. Biochemical findings

a. Serum iron: reduced (below 60 µg/dl)

b. Serum ferritin: reduced (below 12 µg/100 ml). It is the most sensitive and specific test of iron deficiency anemia. It is the first abnormal test. It reflects iron stores accurately, is stable and not affected by recent iron ingestion. It acts both as iron storage and an acute phase protein.

Total iron binding capacity (TIBC)

Normal: 300 to 350 µg/dl

c. Percentage saturation of total iron binding protein (TIBC)

$$\textbf{Transferrin saturation (\%)} = \frac{\text{Serum iron} \times 100}{\text{TIBC}}$$

Normal value 20-45%

Iron Deficiency < 16%

It is second to be affected in iron deficiency.

d. Serum and red cell folate

e. Serum vitamin B_{12}

f. RBC protoporphyrin Protoporphyrin accumulates in the red cells when it has insufficient iron to combine with to form haem. It takes 2-3 weeks to become abnormal after the iron stores are depleted. It helps to differentiate between IDA and thalassemia.

Normal: 20-80 µg/dl

IDA: increased (> 100 µg/dl)

i. FIGLU

j. Osmotic fragility test of RBCs

k. Coomb's test

l. Perl's stain for siderocytes and sideroblasts

IV. Stool examination for helminthiasis (hookworm) infestation and occult blood consecutively for 3 days.

V. Urine: Hematuria, casts, protein to rule out chronic renal disease; schistosomes and occult blood for schistosomiasis.

VI. Other tests: Sputum chest X-ray, renal functions and serum proteins.

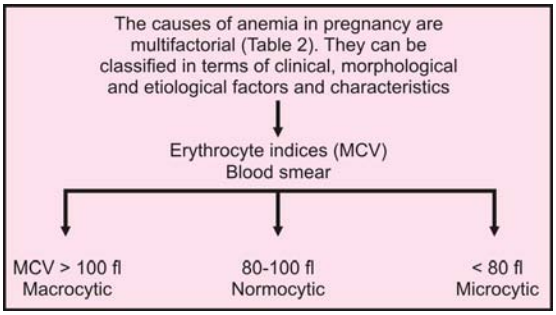
VII. Special tests

Hypochromic red cells (HRBC)

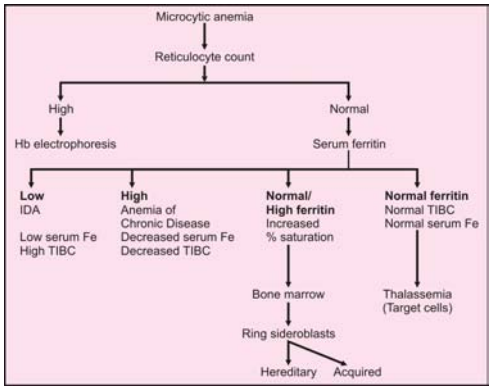
Measurement of hypochromic red cells and percentage of HRBC of total red cells that are released into the circulation in cases of severe anemia are used to determine the severity of iron deficiency, differentiate thalassemia.. It is also used for monitoring of therapy and its effects.

VII. Bone marrow smear examination using potassium ferrocyanate for cellularity, nature of cell reaction and hemosiderin deposits. It is the most accurate method of iron stores. Marrow examination is seldom done. It is only indicated in refractory cases, to diagnose hypoplastic anemia and kala-azar (LD bodies).

CLASSIFICATION AND ETIOLOGY



Microcytic anemia commonly is classified as:
Disorders of iron metabolism —anemia of chronic disease
Disorders of globin synthesis—hemoglobinopathies
Disorders of haem synthesis—sideroblastic anemia and lead poisoning.



The overall **iron requirement in pregnancy** is 1030 mg (800-1650 mg) (Table 4.3). Daily requirement is 4 mg/day (2.5 mg/d in early pregnancy), 5.5 mg/day from weeks 20-32, and 6.6 to 8.4 mg per day from 32 weeks onwards.[17]

Table 4.3: Symptoms and signs of anemia

Symptoms	*Signs*
Lassitude Fatigue, palpitation Breathlessness Dimness of vision Insomnia, angina Parasthesia in fingers and toes	Pallor of skin, mucus membrane, palms, conjunctiva Tachycardia Cardiac dilatation Systolic murmur Oedema Jaundice (hemolytic anemia) Hepatosplenomegaly Sternal tenderness

IRON DEFICIENCY ANEMIA

Iron deficiency anemia (IDA) is the commonest type of nutritional anemia in pregnancy. Iron absorption is affected by diet, and a number of other factors; iron loss depends on physiological and pathological factors. It is depicted in Table 4.4. Iron is absorbed in duodenum and upper jejunum, mainly in ferric form. IDA develops due to increased requirements, defective iron absorption or low dietary intake in pregnancy.

Table 4.4: Differential diagnosis of hypochromic anemia

	Iron deficiency anemia	*α Thalassemia trait*	*Anemia of chronic disease*	*Sideroblastic anemia*
Severity	Variable	Mild	Mild	Variable
MCV	↓	↓	N or ↓	N or ↓
Serum ferritin	↓	N	N or ↑	↑
Serum iron	↓	N	↓	↑
TIBC	↑	N	↓	N
Stainable Iron Red cell	-	+	+	+
protoporphyrin	↑	N	↑	↑ or N
Hb A2	N or ↓	↑	N	↓
Hb F	N	↑	N	N

Sources of Dietary Iron

There are two distinct types of dietary iron: haem and non-haem iron.

Hem iron is a part of hemoglobin and myoglobin and is therefore present in meat, fish and poultry and blood products. It accounts for 10-15% of iron

intake in developed countries and < 1% in developing countries. It has a high bioavailability and 20 -30% absorption rate.

Non-haem iron is mainly found in cereals, tubers, vegetables and pulses. It also forms a part of exogenous iron originating from the soil, dust, and water or cooking vessels. Another source may be the fortified iron present in sugar, salt and flour. Absorption of non-haem iron is highly variable and depends on the balance between the foods that promote or inhibit iron availability.

Dietary Iron Bioavailability and Types of Iron Diets

The broad categories of iron bioavailability may be characterized as follows:

1. **Low bioavailability diet** is a simple monotonous diet based on cereal roots and tubers with negligible quantities of meat, fish and vitamin C. Typically seen in developing countries. Iron absorption is usually < 5%, e.g. diet containing < 30 gm meat of < 25 mg vitamin C per day.
2. **Intermediate bioavailability diet** consists mainly of cereals, roots or tubers but includes some food of animal origin and/or vitamin C. Bio-availability is around 10% e.g. 30-90 gm of meat or 25-75 mg of vitamin C per day.
3. **High bioavailability diet** generous quantities of meat, fish, poultry and vitamin C. Bioavailability is around 15%, e.g. > 90 gm of meat or > 75 mg of vitamin C per day.

THERAPEUTIC OPTIONS

ORAL IRON THERAPY

Iron preparations are available as tablets, capsules, liquid and drops. The percentage of elemental iron varies with the molecular weight of the iron compounds (Table 4.5). Tablets may be *uncoated, enteric-coated or sustained/ slow release preparations.*

Table 4.5: Bone marrow changes in anemia

Iron deficiency anemia—hypercellular bone marrow, erythroid hyperplasia, normoblastic cell reaction, hemosiderin storage nil
B_{12}, folate deficiency anemia—hypercellular bone marrow, giant metamyelocytes, dysplastic megakaryocytes, hemosiderin normal
Hemolytic anemia—hypercellular bone marrow, normoblastic cell reaction, hemosiderin increased
Hypoplastic anemia—hypocellular bone marrow, scanty cells with adipose tissue, hemosiderin normal

One should explain patients about possible side effects and reassure that they will usually settle along the course of therapy. Iron tablet should be taken half to one hour after meals. Tea, coffee and calcium supplements with or within one hour of intake of iron tablet should be avoided. Foods rich in vitamin C may be taken to enhance dietary iron absorption.

Government of India, Ministry of Health and Family Welfare has recommended routine iron supplementation in a dose of 100 mg of elemental

iron with 500 µg folic acid for a period of 100 days in pregnancy (GOI, 2000). Provision of **routine iron supplementation** to pregnant women who do not have iron deficiency anemia (Hb > 10 g/dL) results in a positive effect on women's iron status indicators, The impact of routine iron supplementation on iron status indicators includes reduced prevalence of low pre-delivery hemoglobin (Hb), low serum iron and ferritin, low Hb at 6 weeks after delivery.

Drug Interactions may result in reduced iron absorption or interference with other medications. Drugs decreasing iron absorption are antacids, H_2 receptor blockers, tetracyclines, proton pump inhibitors and calcium supplements. Drugs less well absorbed in presence of iron are methyldopa, L-dopa, quinolones and penicillamine.

Side effects: The oral administration of iron can cause gastrointestinal side effects such as epigastric discomfort, nausea, vomiting, constipation and diarrhoea. The frency of side effects is directly related to the dose of iron. Tolerance improves if taken with meals. Also, the slow release preparations have better tolerance.

Response to iron therapy: There is an increase in reticulocyte count within 5-10 days of start of therapy. Rise in hemoglobin is 0.3 to 1 gm per week (same for oral and parenteral therapy). Response is also indicated by a feeling of well being and an improvement in appetite.

Non Responders to Oral Therapy

When there is no increase in hemoglobin levels after four weeks of oral therapy, then cause of resistant iron deficiency should be evaluated. Possibilities are:

- Inaccurate diagnosis
- Ineffective oral intake—poor compliance, side effects and antacid intake.
- Ineffective absorption—malabsorption states, gastric surgery, etc.
- Continuing blood loss—worm infestations, bleeding piles, NSAID abuse, occult malignancy, etc.
- Coexisting infection
- Other types of anemia—aplastic anemia, myelodysplastic syndrome, anemia of chronic disease, etc.
- Concomitant folate deficiency.

Blood Transfusion Guidelines

- Severe anemia beyond 36 weeks
- Patients not responding to oral or parenteral iron
  Packed cells are preferred for transfusion to prevent cardiac overload.

Carbonyl iron is the purest from of iron containing 98% of elemental iron. It is a small particle preparation of highly purified metallic iron. Carbonyl describes the process of manufacture of iron particles, not their composition. Its absorption depends on its solubilization by gastric acid and the slow rate of solubilization is responsible for its low toxicity (wide margin of safety) and a high bioavailability. Unlike ferrous and ferric salts, it is inert and incapable of reacting with strong chelators of iron.

Iron polymaltose complex (IPC) contains nonionic iron and polymaltose in a stable complex. It is one of the causes of failure of response to iron therapy due to its poor bioavailability and low absorption rate. There are several reports of inadequate or slow rise in hemoglobin; therefore it should not be used for oral iron therapy in human.

PARENTERAL IRON THERAPY

It has no advantage over oral iron if the latter is well tolerated.

Indications for Parenteral Iron

- Inadequate gastrointestinal iron absorption
- Intolerance to requisite dose of oral iron
- Chronic blood loss exceeding the oral replacement
- Combination with recombinant human erythropoietin
- Potential contraindication to blood transfusion.

There are four types of **parenteral iron complexes** on the basis of kinetic and thermodynamic properties.

Type I complexes	Iron dextran Iron dextrin	100,000 Da. T ½ 3-4 days	High complex stability Slow iron release
Type II complexes	Iron-III Hydroxide saccharate complex (iron sucrose complex)	30,000–100,000 Da. T ½ - 6 hours	No biological polymers (No anaphylaxis) Dose: 1 to 4 mg/kg body weight
Type-III complexes	Iron-III gluconate Iron-III citrate Iron-III sorbitol	< 50,000 Dalton	Labile week complexes Liver necrosis
Type-IV complexes	Mixture of at least 2 different classes	Releases iron to different types of proteins	Intravenous dose not recommended

Type III and IV are clinically not safe.

INTRAMUSCULAR

Jectofer (iron-sorbitol citrate complex) is only used by intramuscular route.
Jectofer plus also contains folic acid and Vit B_{12}, other than iron.
Inferon (iron-dextran) can be given by both intramuscular and intravenous route.

Both are given deep into the upper outer quadrant of the buttock; skin is laterally displaced before injection (Z track technique) to prevent staining of the skin. After injection, iron is slowly absorbed via lymphatics, then taken up by hepatic RE cells. It is then transferred to erythrocytes. Precautions should be taken in patients with liver disease and those with known hypersensitivity.

Dose (Holly)

$$= \frac{13.5 - Y}{100} \quad 4000 \times \quad 3.4 \times 500$$

Y is the patient's current hemoglobin and 500 mg as storage iron. 4000 stands for blood volume (66 ml/kg). Each ampoule contains 50 mg/ml of elemental iron and its test dose is 0.5 ml on day 1.

Daily dose should not exceed 5 ml (250 mg of iron).

INTRAVENOUS THERAPY

Iron dextran and recently iron sucrose (*Venofer, Ancifer*) are the intravenous preparations of choice for parenteral therapy. Iron dextran can also be given intravenously in the form of a drip. In this manner, total requirement of iron can be given in a single sitting. It is essential to give a small test dose intravenously before giving the total dose. Adrenaline should be kept ready in a syringe to counter anaphylactoid reactions. The iron dextran complex has been associated with serious side effects (toxic, allergic reactions and even severe anaphylaxis). However, the type II iron sucrose complexes that release iron to the endogenous iron binding proteins with a half life of about 6 hours carry a minimal risk of allergic accidents and overload. Thus, iron sucrose complex therapy is a valid first line option for safe and rapid reversal of iron deficiency anemia.

Indications

- Small muscle mass
- When absorption through muscle is impaired (e.g. edema)
- Intramuscular injection is contraindicated (coagulation disorders)
- Patient is unreliable

Calculation of the total dose of iron:

1. Total dose = 0.3 WD + 50% for replenishment of body stores
   W = Patient's weight in pounds
   D = (100 – Hb %) = observed hemoglobin conc. in percentage
2. The simplest formula is:
   Total dose of elemental iron (mg) = wt (kg) × Hb deficit (Normal Hb[14]-Patient Hb) × 2.21 + 1000
3. Total dose = 250 mg × per gm of Hb below normal

It is customary to add 1000 mg to above to replenish iron stores. Oral iron should be stopped at least 24 hours prior to avoid reaction.

Total dose infusion (TDI): An intravenous infusion is started with 1 litre of either 5% dextrose or 0.9% NaCl (isotonic saline). 1 ml of intravenous iron dextran is injected and observed for 15 to 30 min. If no adverse effects are observed, rest of the dose is added to the bottle and the infusion run for 4 to 6 hours (10 drops per min for first 20 min, then 40 drops per min till completion). Maximum dose should not exceed 2.5 gm (50 ml iron dextran) on 2 consecutive days. All emergency drugs are kept ready. Oral iron therapy is stopped one week before TDI. TDI has several advantages: eliminates repeated and painful

intramuscular injections, treatment is complete in 1 or 2 days and is more cost effective.

Precautions: Saturation or near saturation of iron binding capacity is a contraindication to parenteral administration regardless of hemoglobin level (thalassemia or recent oral iron intake). Toxic reactions include headache, nausea, shivering, urticaria and fever, local phlebitis and anaphylaxis (around 6%). A final advantage of parenteral administration is that a known quantity of iron is placed into the patient, and when a properly calculated dose is used, iron deficiency should not recur.

Side Effects

a. **Local reaction:** skin staining, pain and abscess formation
b. **Systemic:** Fever, arthralgias, myalgia, nausea, vomiting, diarrhea, body ache, skin rash, chest pain, abdominal pain, lymphadenopathy, angioneurotic edema.
c. **Anaphylactic shock** which has even led to death in a few cases.

MEGALOBLASTIC ANEMIA IN PREGNANCY

Definition: The term 'megaloblastic anemia' refers to anemias that are associated with enlargement of proliferating erythroid precursor cells as megaloblasts, megalocytes and macrocytes.

Classification: Folate and Vit B_{12} deficiency anemia.

Folate requirement increases in pregnancy from 100-150 µg/d to 200-250 µg/d. There is no change in gastrointestinal absorption of folate in a healthy pregnancy. Folic acid prevents the first occurrence and recurrence of neural tube defects. Vitamin B_{12} deficiency rarely occurs in pregnancy as it usually causes infertility. Daily requirement of vitamin B_{12} is 3 µg/day.

Predisposing factors that may lead to folic acid deficiency anemia in pregnancy

- Increased requirement
- Multiple pregnancy
- Underlying hemolytic anemia (e.g. sickle cell anemia)
- Inadequate diet
- Urinary tract infection
- Drugs that impair folate metabolism, absorption or utilization (e.g. Trimethoprim, phenytoin, ethanol, nitrofurantoin, barbiturates).
- Seasonal (more likely in winter in some geographical areas secondary to decreased availability of fresh products).

Tests for macrocytic anemias (Folate and Vitamin B_{12} deficiency):

a. **Hemoglobin and peripheral smear:** In both folic acid and vitamin B_{12} deficiency, there is presence of macrocytic red cells (MCV > 100 fl, MCH > 33 pg, MCHC normal), which appear large and oval. Hypersegmented neutrophilic leukocytosis may be the earliest morphological change to appear in blood. Low reticulocyte count, nucleated RBC, Howel Jolly bodies, pancytopenia are also seen.

b. **Serum folate concentration:** As a screening test, it has a drawback that serum folate levels have day to day variation and postprandial increases have been noted. Thus normal levels may be observed in deficient individuals. Thus, it is not a reliable test in pregnancy.
c. **Red cell folate:** More reliable indicator of the folate status as it does not reflect the daily and short term variations of plasma folate. The test however, lacks specificity, because red cell folate level is decreased in over half of the cases with vitamin B_{12} deficiency.
d. **Excretion of form iminoglutamic acid (FIGLU):** No longer recommended as a screening test because already there is increase FIGLU in normal pregnancy.
e. **Serum vitamin B_{12}:** Due to evidence of functional B_{12} deficiency in individuals with low normal serum B_{12} levels, the measurement of serum B_{12} levels is currently questionable. Women who have significant cobalamin deficiency are often infertile. Pregnancy has a little effect on overall cobalamin status due to the availability of maternal stores and small fetal requirement.
f. Liver and kidney function tests to rule our chronic hepatorenal diseases.
g. Stool for occult blood and analysis.
h. Upper and lower GI endoscopy and/ or barium studies of gastrointestinal tract.
i. Pelvic ultrasound if required.
m. Bone marrow—Megakaryocytes may be abnormally large with decreased granulation and bizarre nuclear appearance. Look for metamyelocytes, RBC precursors, pronormoblasts and nuclear cytoplasmic asynchrony.

TREATMENT OF FOLATE DEFICIENCY ANEMIA

One mg folic acid OD orally replenishes body stores in three weeks. In the presence of malabsorption, doses up to 5 mg/day may be required. Vitamin B_{12} deficiency must be excluded in patients on folic acid therapy as such treatment may result in progressive neurological deterioration.

TREATMENT OF VITAMIN B_{12} DEFICIENCY ANEMIA

Parenteral: Injection of hydroxycobalamin 1000 µg daily intramuscularly for one week, then weekly for one month and then monthly thereafter. Response to therapy is characterized by reticulocytosis which occurs within one week of initiation of therapy.

Oral: 1000 to 2000 gm/day is found to be as effective as parenteral route. Combination products containing vitamin B_{12} and intrinsic factor are also available but are not readily absorbed. Because of the risk of allergic sensitization, their use is not currently recommended.

Intranasal gel: Containing cyanocobalamin has recently been labeled for maintenance therapy of patients in hematological remission after parenteral vatamin B_{12} therapy. Weekly dose provides 500 gm of drug require life long maintenance therapy consisting of 100 µgm of injection vitamin B_{12} every one to three months.

HEMOGLOBINOPATHIES (Inherited abnormalities of hemoglobin production)

Although < 3% of all cases of anemia diagnosed during pregnancy are inherited, 2 to 3 lakh severely affected children are one born each year.

α- and β- Thalassemia Trait

They are common in black populations and in people of Mediterranean descent. Diagnosis is made in pregnancy on the basis of microcytic anemia (MCV < 80 fl) combined with a normal serum ferritin and unresponsiveness to oral iron therapy. A mild hypochronic microcytic anemia is seen in both the homozygous state for α+ thalassemia (-α /- α) and heterozygous state for α-0 thalassemia. Routine antenatal screening for thalassemia is advocated. **Preconception counseling** focuses on evaluation of baseline cardiac function with MUGA, ECHO and ECG; excluding hypothyroidism (seen in 9%); IDDM (seen in 4.9 to 20%); and impaired glucose tolerance (IGT) in 25% of patients.

Genetic counseling of the couple is essential prior to conception. Evaluation of partner and hemoglobin electrophoresis is a must. **Prenatal diagnosis** is offered to the couple to rule out fetal affection by chorionic villous sampling and cordocentensis.

Ferritin is monitored every month; aim is to achieve a level of 1000 to 2000 μg/L. For management of pregnancy, stop desferrioxamine and vitamin C as it increases absorption of iron and precipitates cardiac damage. Folic acid 5 mg/day is administered. Cardiac function should be carefully monitored at 12 weeks, at delivery and in puerperium. Blood transfusion is given to maintain hemoglobin > 10 g/dL. Fetal surveillance is performed to rule out IUGR. There is no hematological indication for induction of labor. High caesarean section rate is due to cephalopelvic disproportion (short maternal stature) and fetal distress (hypoxia due to placental iron deposition).

SICKLE CELL DISEASE IN PREGNANCY

Most common disorder of hemoglobin structure associated with pregnancy. The defect associated with Hemoglobin S occurs when the neutral amino acid valine is substituted for negatively charged glutamic acid at the sixth position from the N-terminus of the α chain. Sickle hemoglobinopathies are classified as major and minor disorders The gene offers protection against malaria. More than one-third of pregnancies in women with sickle cell anemia terminate in abortion, stillbirth or neonatal death. **Sickle cell trait** as compared to sickle cell anemia does not increase maternal morbidity, perinatal morbidity, perinatal morbidity and mortality.

Management

- Confirm diagnosis
- Offer counseling. Genetic implications for the newborn using prenatal diagnosis should be discussed.

- Specific complications
- Anemia—most severe is hemoglobins, hemoglobins α thalassemia
  Folic acid 5 mg/day, blood transfusion to maintain hemoglobin > 10 g %
  Iron supplements should not be given until iron studies demonstrate iron deficiency anemia.
- Pain crisis should be differentiated from abruptio placentae, ectopic pregnancy and pyelonephritis.
- Search for precipitating cause and treat (infection, dehydration). Rehydration, oxygen and narcotic analgesics are used.
- Fetal surveillance is done from 34 weeks to detect IUGR.

Prophylactic transfusion is one of the main controversies in managing pregnant patients with sickle cell disease. There were no significant differences in perinatal outcome in women who were prophylactically transfused compared with those transfused only for medical or obstetric emergencies. Prophylactic transfusion was associated with a reduction in painful crisis. Omission of prophylactic transfusions will not harm pregnant patients with sickle cell disease or their offspring".

Indications for transfusion

- Who has already undergone a pregnancy that ended in death of fetus
- Preeclampsia
- Acute chest syndrome
- Neurological deficit of recent onset
- Severe anemia, hemoglobin < 5 g%, HCT < 15%, reticulocyte count < 3%
- In preparation of surgical intervention.

Complications

- Serious maternal morbidity such as pneumonia, pyelorephritis, cholecystitis, pulmonary embolism and skin infections is noted in approximately 30 to 40% of women with SCD.
- 70% of patients with Hemoglobin S-S and approximately 30% of those with Hemoglobin SC have vasoocclusive crisis.

On **follow up,** closely observe postpartum period for evidence of crisis and thrombosis. Depo-provera, progestin only pills can be used, but combination OCPs and IUCDs are generally not recommended.

ROLE OF ERYTHROPOIETIN IN PREGNANCY

Erythropoietin (EPO) is produced in response to fetal hypoxia and anemia and is essential for fetal erythropoiesis. Its measurement enables the more accurate timing of hypoxic injury. EPO is important in the production and differentiation of erythroid progenitor cells into red blood cells. The human EPO gene is located on chromosome 7 (7 pter-q22).

The molecular weight of EPO is 34,000 Daltons and its half life is 2 to 4 hours. It is rapidly cleared by the liver. It is produced in the interstitial peritubular cells of the kidneys in adults.

Maternal EPO levels rise 2 to 4 times during normal pregnancy. Fetal ertythropoiesis begins at 14 days of gestation in the yolk sac followed by liver and spleen between 6 to 10 weeks and finally in the bone marrow thereafter. EPO is present in the amniotic fluid as early as 7 weeks of gestation and in the cord blood at 16 weeks of gestation. Its level rises with fetal growth as the oxygen demand increases along with a rise in hematocrit. In normal and abnormal pregnancies, there is a good correlation between cord blood and amniotic fluid EPO levels. On the contrary, there is no correlation between maternal and amniotic fluid EPO levels, suggesting little or no placental transfer. Cord blood EPO levels are raised in IUGR, Rh isoimmunization, maternal hypertension, preeclampsia, maternal alcohol and smoking, diabetes, acute fetal asphyxia with abnormal outcome.

Dose = 15-500 units/kg/week subcutaneous every 2-3 days.

Clinical applications of EPO in obstetrics are summarized in Table 4.6. The major therapeutic use of EPO involves its use in the treatment of anemia in patients with end-stage renal disease. It has also been useful in anemic patients without renal disease in the postpartum period. The major side effects are due to increase in red cell mass. Mild flulike symptoms are common but disappear with repeated administration. Rare side effects include hypertensive encephalopathy, iron deficiency, seizures, clotting of dialyzer, hyperkalemia and hyperphosphataemia.

Table 4.6: Development of iron status testing

Blood smear	1889
Erythrocyte indices	1897
Serum iron	1937
Bone marrow	1948
Ferritin	1972
EC Scattergram	1984
Transferrin receptor	1986

APLASTIC ANEMIA IN PREGNANCY

Etiopathogenesis

a. Primary idiopathic aplastic anemia

An autoimmune cause may be responsible for the majority of cases.

b. Secondary aplasia or hypoplasia

Aplastic anemia is classified as severe if there is < 25% normal cellularity in bone marrow, granulocytes < 500/μL, platelets < 20,000/μL and reticulocyte count < 1%.

- Bone marrow transplantation increases survival rate by 50-80%.
- During pregnancy supportive therapy is the main objective.
- Maternal mortality rate is 15%.

- Management options are
  - Maintain hemoglobin levels by blood transfusion.
  - Prevent and treat infections
  - Stimulation of hematopoiesis with androgens (remission rate with steroids only 12%)
  - Splenectomy
  - Bone marrow transplantation after delivery
  - The only indication to terminate pregnancy prematurely is the inability to support the patient with transfusion and the need to proceed with either marrow transplantation or antithymocyte globin therapy.

OBSTETRIC MANAGEMENT OF ANEMIC PATIENTS

Antenatal period

- UTI, asymptomatic bacteriuria, preterm labor

Management of second stage of labor

- Propped up position
- Oxygen inhalation
- Morphine for analgesia
- Rapid digitalization in case of cardiac failure
- Cut short second stage of labor (forceps
- Antibiotic prophylaxis.

Active management of third stage of labor

- Oxytocin given within 1 minute of delivery of baby (10 units iv, acts in 2-3 mins.)
- Controlled cord traction
- Uterine massage
- Avoid IV methergin

Postpartum period

- Continue iron and folic acid for 3 months
- Energetic treatment of infection
- Lactation failure, subinvolution and thromboembolism to be taken care of.

Maternal mortality can occur in last trimester, during labor, immediately after delivery and during the postpartum period due to cardiac failure and pulmonary edema.

Table 4.7: Etiological classification of anemia in pregnancy

Nutritional deficiency
- Iron deficiency
- Folate deficiency
- B_{12} deficiency
- Vitamin A deficiency
- Anemia due to protein malnutrition and scurvy

Hemorrhagic (secondary to blood loss)
- Acute: Early trimester bleeding, APH
- Chronic: Piles

Hemolytic
- Acquired: Associated with preeclampsia (HELLP syndrome), chronic infection
- Congenital: Congenital acholuric jaundice, sickle cell anemia

Bone marrow suppression
- Aplastic anemia
- Secondary to chronic systemic disease

Hemoglobinopathies

Infection and parasitic infestation
- Malaria, hookworm
- HIV

Table 4.8: Hematologic changes in pregnancy

Characteristics	*Normal adult women*	*32-34 weeks gestation*	*Increased/ decreased*
Plasma volume (ml)	2600 ml	3850 ml	1250 ml increase
Red cell mass (ml)	1400 ml	1640 ml (without iron supplement) 1800 ml (with iron supplement)	
Hemoglobin (g/dl)	12-14	11-12	Decreased
Red blood cells $(10^6/mm)^3$	4-5	3-4-5	Decreased
Packed cell volume	0.36-0.44	0.32-0.36	Decreased
MCV (fl)	80-97	70-95	Decreased
MCH (pg)	27-33	26-31	Decreased
MCHC (%)	32-36	30-35	Decreased
Serum iron (µg/dl)	60-175	60-75	Decreased
Total iron binding capacity (µg/100ml)	300-350	350-400	Increased
% saturation	30	15	Decreased
Requirements of iron (mg/day)	1.5-2.0	4.0	Increased

Table 4.9: Morphological classification of anemia in pregnancy

Normocytic normochromic anemia
Microcytic hypochromic anemia
Macrocytic normochromic anemia
Microcytic normochromic anemia (simple microcytic)

Table 4.10: Iron requirement in pregnancy

	Average (mg)	*Range (mg)*
External iron loss	170	150-200
Expansion of RBC mass	450	200-600
Fetal iron	270	200-370
Iron in placenta and cord	90	30-170
Blood loss at delivery	150	90-310
Total requirement	1030	800-1650

Table 4.11: Iron metabolism in pregnancy

Dietary Iron
↓
Intestinal mucosa (Absorption 1 mg/d)
↓
Plasma Iron – Plasma Iron - Plasma Iron – obligatory losses
(3 mg pool. (2 mg/d in females)
↓ ↓ ↓
Erythroid marrow RE pool Interstitial fluid and tissues
(25 mg/d uptake) (6 mg/d)
↓
Circulating RBCs ↓
(2100 mg/d pool)
↓ Ferritin stores
Reticuloendothelial pool (25 mg/d)
↓
RE pool

Table 4.12: Stages in development of iron deficiency

Parameters	*Normal*	*Iron depletion*	*Iron deficient erythropoiesis*	*Iron deficiency anemia*
Hemoglobin (g/l).	150 g/l	130 g/l	100 g/l	50 g/l
MCV	N	↓	↓	⇓
MCHC	N	N	↓	⇓
Marrow Iron stores	Present	Reduced	Absent	Absent
S. Fe/TIBC (µ g/l)	1000/3000	750/3000	500/4500	250/6000
Serum ferritin (µg/l)	100	20	10	< 10
Erythrocytes	Normal	Normal	Normal	Hypochromic microcytosis

Table 4.13: Factors affecting the iron status of a pregnant women

Iron absorption	*Iron loss*
Dietary iron (haem and non-haem) **Enhancers of absorption** • Haem iron • Proteins • Meat • Ascorbic acid • Fermentation • Ferrous iron • Gastric acidity • Alcohol • Low iron stores • Increased erythropoietic activity (high altitude, hemolysis, bleeding) **Inhibition of iron absorption** • Phytates • Calcium • Tannins • Tea and coffee • Herbal drinks • Fortified iron supplements	**Basal losses from desquamation of GIT, GUT skin** **Physiological factors** • Menstruation (1 mg/day) • Delivery • Lactation **Pathological factors** • Hookworm and other helminths • Hemorrhage from GIT • Allergies • Occult blood losses

Table 4.14: Choice of oral preparations with elemental iron composition

Preparation	*Molecular Iron (mg/tab)*	*Elemental Iron (mg/tab)*	*Percentage of iron (%)*
Ferrous Fumarate	200	66	33
Ferrous Sulphate* (anhydrous)	300	74	37
Ferrous Sulphate* (desiccated)	200	60	30
Ferrous Gluconate**	300	36	
Ferrous Succinate	100	35	12

* Least expensive and most commonly used preparation.
** Preparation with least gastrointestinal side effects.

Table 4.15: Advantages of Iron sucrose complex

Highly safe
Highly stable
Low tissue accumulation and toxicity
High availability for ertythropoiesis
Rapid incorporation (bone marrow and red cells)
Prevents iatrogenic iron depletion (e.g. after rhEPO)

Table 4.16: Causes of megaloblastic anemia

Deficiency	*Clinical states*
1. Folate deficiency	A. Inadequate intake B. Increased requirement: pregnancy, malignancy, infancy C. Malabsorption: tropical and nontropical sprue D. Impaired metabolism: methotrexate, dihydrofolate reductase inhibitors
2. B12 deficiency	A. Malabsorption a. Inadequate production of intrinsic factor (IF): Pernicious anemia, gastrectomy, congenital absence of IF b. Disorders of terminal ileum: tropical and nontropical sprue, intestinal resection c. Competition for cobalamin: fish tapeworms B. Inadequate dietary intake

Table 4.17: Classification of sickle hemoglobinopathies

Major disorders
Sickle cell anemia Hemoglobin SS.
Hemoglobin SC disease
Hemoglobin S α thalassemia
Minor disorders
Sickle cell trait Hemoglobin AS (most common)
Hemoglobin C trait
Hemoglobin C disease
Hemoglobin SE
Hemoglobin SD

Table 4.18: Clinical applications of EPO in obstetrics

Fetal	Treatment of anemia of prematurity
	Treatment of severe Rh isoimmunization to correct anemia
	Marker of fetal hypoxia
Maternal	**Anemia during treatment of pregnancy**
	Iron deficiency
	Sickle cell anemia
	Chronic renal failure
	AIDS
	Rheumatoid arthritis
	Malignancy
	Autologous blood transfusion
	Placenta praevia
	Placenta accreta
	Treatment of postpartum anemia

DEFINITION AND CLASSIFICATION

- Anemia: Insufficient Hb to carry out O_2 requirement by tissues.
- WHO definition: Hb concentration < 11 gm%
- CDC definition: Hb concentration < 11 gm% in 1st and 3rd trimesters and < 10.5 gm% in 2nd trimester
- For developing countries: Cut off level suggested is 10 gm%

  WHO technical report Series no. 405, Geneva 1968
  Centre for disease control, MMWR 1989;38:400-4

WHO classification of anemia

Degree	*Hb%*	*Hematocrit (%)*
Moderate	7-10.9	24-37%
Severe	4-6.9	13-23%
Very severe	< 4	< 13%

History

Poor pre pregnancy iron balance

1. Whether she suffered from systemic diseases like tuberculosis, infection like malaria, kala-azar.
2. Menstrual disorders—excessive bleeding during menstruation
3. Whether she comes from low socioeconomic class
4. Dietary habits—vegetarian or non vegetarian, low in calorie, rich in phytates
5. Gastrointestinal infection/infestation—worm infestation
6. Family history of thalassemia, sickle cell disease
7. Drug—anti convulsants.

Obstetrical history:

- Frent child birth
- PPH

Frently asked questions:

1. What is the WHO definition of anemia in pregnancy?

Hb concentration < 11 gm/dl

2. What is the magnitude of the problem?

Globally is around 30%. In developing countries and India incidence is around 40-90%.
Maternal mortality (all round) 600-700/100,000
Anemia directly—50%
Indirectly—50% due to toxemia, eclampsia, hepatitis, malaria, APH, PPH, cardiac disease, obstructed labor.

3. What is the cause of anemia in pregnancy?

- Physiological
- Pathological

  Nutritional iron deficiency/folic acid /vitamin B_{12}/protein deficiency
  Hemorrhagic (acute/chronic).

Hemolytics (familial/acquired)
Bone marrow insufficiency
Hemoglobinopathies.

4. What are the types of anemia?

Hypochromic—Microcytic anemia—iron deficiency anemia (other causes are infection/nephritis/hemoglobinopathies)

Megaloblastic anemia, hypersegment of neutrophils, fully hemoglobinised RBC.

5. What is the daily requirements of iron during pregnancy?

< 20 weeks—0.7 mg
20-30 weeks—5 mg
> 32 weeks—8 mg

6. What is the total iron requirement of pregnancy?

900 mg approximately

7. When is the greatest physiological anemia during pregnancy?

32 weeks.

8. What are the adverse effects of anemia during pregnancy?

Maternal: cardiac failure, toxemia, antenatal infection, thrombophlebitis, progressive anemia, puerperal enterocolitis, refractory diarrhoea, puerperal psychosis

Foetal: prematurity, IUGR, anemic baby, hypothermia, hypertension in adulthood

9. How will you diagnose a case of anemia?

a. Screening
b. Clinical feature—Hb < 7-8 gm/dl
c. Lab investigations—Hb% decreased, hematocrit decreased, MCV decreased

Peripheral smear examination:

- Microcytic and hypochromic cells in iron deficiency
- Macrocytic cells in folate/vitamin B_{12} deficiency
- Normocytic cells in other causes or early nutritional deficiency
- Schisto/aniso—and poikilocytes in hemolytic anemia
- Sickle cells in sickle cell anemia
- White blood cells hypersegmented in megaloblastic anemia
- Target cells in thalassanemia
- Platelets may be decreased
- Parasites such as *Plasmodium malariae* and Leishmania may be seen.

Serum ferritin < 12 mcg/l
Transferritin saturation < 15 %
Elevated protoporphyrin level
Serum iron concentration < 60 mcg /dl

Transferring receptor concentration increased (promising new indicator), iron binding capacity increased, Hb electrophoresis,

Hb concentration, serum ferritin, transreceptor concentration—excellent depiction of iron status.

10. When you will do medical evaluation for thalassemia trait and sickle cell disease?

When with oral iron for 1 to 1.5 month, no improvement and compliance reconfirmed. Then Medical evaluation for thalassemia trait and sickle cell disease should be done.

11. How long iron therapy should be continued in anemic lady after delivery?

Continued till 6 weeks to 3 months postpartum (till the clinical and hematological state comes to normal)

12. What is the cause of failure of oral iron therapy?

Non compliance due to side effects, wrong diagnosis, continued blood loss, coexisting chronic disease, concomitant folic acid deficiency, inefficient absorption of.

13. What is the indication of parenteral iron therapy?

Pregnant women with severe iron deficiency in last month, contraindication to oral iron therapy, GIT intolerance to oral iron, malabsorption syndrome, rapid blood loss.

14. What are the management options?

Pre-pregnancy—treatment of cause before conception, pre pregnancy balanced diet, education and health support, build up iron stores during adolescence.

15. How will you manage a case of severe anemia during labor?

In bed—prop up position, O_2 inhalation, sedation, tranquilizer, shortening of 2nd stage of labor, restrict IV FLUIDS, IV ergometrine should be avoided, prophylactic prostaglandins, exchange transfusion with packed cells, if cardiac failure—fursemide, digitalis, antibiotics, during puerperium—antiemetic therapy, diuretics, antibiotics, physiotherapy, contraceptive advice.

16. In what form does the fetus get the iron?

Maternal transferrin through placenta.

17. What is the earliest marker of response to iron therapy?

Increase in reticulocyte count within 5-10 days.

18. What is the average rise in Hb per week with iron deficiency anemia?

0.8 mg/dl per week

19. What is the daily folate requirement in non pregnant patients and how much in pregnancy?

50 µg. It is increased three fold in pregnancy.

20. What is the WHO recommendation for daily folate intake?
400 µg.

21. How do you diagnose folate deficiency?
Low Hb, peripheral smear, oval macrocyte hypersegmented neutrophils, low reticulocyte count and low plasma folate, low red cell folate.
Bone marrow—large erythroblast, giant abnormal cell, metamyelocytes.

22. What are the effects of folate therapy if one has B_{12} deficiency?
It can worsen the neuropathy.

23. What is the prophylactic dose of folate?
400 µg/day.

24. What is the therapeutic dose of folate?
5 mg/day.

25. What is the daily requirement of Vit B_{12} during pregnancy?
3 µg/day.

26. What are the special clinical features of B_{12} deficiency?
Neurological symptoms, numbness parasthesia, weakness ataxia, poor concentration, dementia, psychosis.

27. What do you mean by hemoglobinopathy?
- A collective term for the inherited disorders of Hb synthesis
- Disorders of globin synthesis, e.g. thalassemia
- Structural Hb variants, e.g. sickle cell anemia, HbC.

28. How you will diagnose thalassemia?
Hb estimation, peripheral smear examination, decreased MCV, decreased MCH and HbA2.

29. What is sickle cell disorders?
- Structural Hb variant
- Exists in homo and heterozygous forms
- Under hypoxic conditions, HbS polymerizes, gels or crystallizes/hemolysis of cells, and thrombosis of vessels in various organs
- In long-standing cases, multiple organ damage.

30. What is sickle cell crisis?
Acute events that occur in individuals with sickle cell disease.
Two major types of crisis—vaso-occlusive and hematologic crisis.
Most crisis during pregnancy are vasocclusive occurring in the latter half of pregnancy or in the peurperium.

31. What is prophylaxis against nutritional anemia (PANA)?
A nutritional anemia during pregnancy. 100 tabs of 100 mg of elemental iron in the second half of pregnancy.

32. How do you calculate the dose of iron?
0.3 × weight in lbs. × (100-Hb %) + 50% to replenish the stores.

33. Parenteral iron therapy?

It has no advantage over oral iron if the latter is well tolerated.

INTRAMUSCULAR

Initial test dose of 1 ml is given IM.
Jectofer (iron-sorbitol citrate complex) only IM.
Jectofer plus also contains folic acid and Vit B_{12}, other than iron.
Imferon (iron-dextran) IM/IV.

INTRAVENOUS THERAPY

Iron dextran and recently iron sucrose (*Venofer, Ancifer*) are the intravenous preparations of choice for parenteral therapy.

34. Side effects of oral/injectable iron.

Oral	Parenteral
Gastrointestinal side effects such as epigastric discomfort, nausea, vomiting, constipation and diarrhoea. The frency of side effects is directly related to the dose of iron.	a) **Local reaction:** skin staining, pain and abscess formation b) **Systemic:** Fever, arthralgias, myalgia, nausea, vomiting, diarrhoea, body ache, skin rash, chest pain, abdominal pain, lymphadenopathy, angioneurotic edema **Anaphylactic shock** which has even led to death in a few cases.

35. Comparison of difference iron preparations?

There are four types of **parenteral iron complexes** on the basis of kinetic and thermodynamic properties.

Type I complexes	Iron dextran Iron dextrin	100,000 Da. T ½ 3-4 days	High complex stability Slow iron release
Type II complexes	Iron-III Hydroxide saccharate complex (iron sucrose complex)	30,000–100,000 Da. T ½ - 6 hours	No biological polymers (No anaphylaxis) Dose: 1-4 mg/kg body weight
Type-III complexes	Iron-III gluconate Iron-III citrate Iron-III sorbitol	< 50,000 Dalton	Labile week complexes Liver necrosis
Type-IV complexes	Mixture of at least 2 different classes	Releases iron to different types of proteins	Intravenous dose not recommended

Type III and IV are clinically not safe.

36. How will you give parenteral therapy?

Intramuscular therapy: Initial test does of 1 ml, IM given. The inj. are given 2 ml inj. with 2 inch needle deep into upper quadrant of the buttock using a "Z" techni (pulling the skin and subcutaneous tissues to one side before inserting

the needle) and additional precaution is to inject small quantity of air or saline down the needle before withdrawing it.

Intravenous therapy: Initial test does of 1 ml IV.

Total Dose Infusion (TDI): An intra venous infusion is started with 1 litre of either 5% dextrose or 0.9% NaCl (isotonic saline). 1 ml of intravenous iron dextran is injected and observed for 15 to 30 min. If no adverse effects are observed, rest of the dose is added to the bottle and the infusion run for 4 to 6 hours (10 drops per min for first 20 min, then 40 drops per min till completion). Maximum dose should not exceed 2.5 gm (50 ml iron dextran) on 2 consecutive days.

37. Adjuvant therapy of iron deficiency anemia?

1. **Diet:** Balanced diet rich in proteins, iron and vitamins and which is easily assailable is prescribed.
2. **To improve the appetite and facilitate digestion:** Preparation containing acid pepsin.
3. **Vitamin C—**Food rich in vitamin C
4. Treatment of worm infestation.

38. What is dimorphic anemia?

- This is the commonest type of anemia in the tropics.
- Is related to dietary inadequacy or intestinal malabsorption.
- Deficiency of both iron and folic acid or vitamin B_{12}.
- The red cells become macrocytic or normocytic and hypochromic or normochromic.
- Bone marrow picture is predominantly megaloblastic as the folic acid is required for the development of the number of red cell precursors.
- The treatment consists of prescribing both the iron and folic acid in therapeutic doses.

39. General investigations in anemia?

Other investigations for anemia:

- Urine examination
- Stool examination
- Bone marrow
- Radiograph of chest
- BUN/serum creatinine.

40. Place of blood transfusion?

- Patient with severe anemia seen in later months of pregnancy (beyond 36 weeks) in order to improve the anemic state and oxygen carrying capacity of blood before the patient goes into labor. The primary concern is not only to correct anemia but also to make the patient fit to withstand the strain of labor and blood loss following delivery.
- Refractory anemia. Anemia not responding to either oral or parenteral therapy in spite of correct diagnosis.
- To correct anemia due to blood loss and to combat PPH.

41. Special investigations.

1. Sickling test i) early, and ii) late
2. Hb electrophoresis
3. Direct Coombs test
4. Indirect Coombs test
5. Enzyme studies of metabolic pathways of G6PD.

5

Heart Disease in Pregnancy

Kanan Yelikar

1. How do you evaluate a case of heart disease?

i. Symptomatology.
ii. Clinical examination.
iii. Investigations to decide the etiology, structures involved, severity of heart disease, heart size and cardiac rhythm.

2. What are the basic investigations required.

1. CBC, TLC DLC, Urine for microscopic hematuria (SABE)
2. ASO titers
3. X-ray chest—to look for cardiomegaly: Straightening of the left heart border Kerley B lines.
4. ECG
5. Echocardiology

3. What is the prognosis of Heart disease during pregnancy?

A. Immediate prognosis depends on functional capacity of heart and antenatal care provided.

4. What are the factors which precipitate failure?

1. Anaemia
2. Upper Respiratory infection
3. Pre-eclampsia.
4. Tachycardia of any origin, arrhythmias.
5. Recurrence of active rheumatic fever
6. Hyperthyroidism.
7. Excessive weight gain.
8. Bacterial endocarditis (characterized by pallor, pyrexia, petechieal hemorrhage, palpable spleen, positive blood culture, microscopic hematuria).

5. How will you manage a case of Heart disease during pregnancy?

Antenatal care:

Frent visits, cardiologists opinion.
Advice given—adequate rest, SRD

- Avoid exertion, cold , infection, avoid excessive wt gain, anemia.
- Advice hospital admission routinely.
- Grade I two weeks prior to EDD
- Grade II at 28 wks.
- Grade III/IV: As soon as the diagnosis is made.

6. Management in the ward?

- Bed rest
- Sedation
- Diuretic
- Prophylactic antibiotics
- Digoxin if pt is in failure.

7. How will you manage labor?

Patient is preferably delivered on a comfortable bed rather than the table.

- Propt up position
- O_2 kept ready
- Drugs-sedatives, diuretics, antibiotics, digoxin, restriction of iv fluids.
- Use of prophylactic forceps to cut down the second stage of labor.

8. How is the course of labour in heart disease.?

The labour is usually easy, due to congestion of the pelvis.

9. What is rule of five?

There are 5 occasions when patient can land up in failure

- 1.5 weeks of gestation
- 2.5 months of gestation
- 3.5 weeks before EDD
- 4.5 hours after onset of labor
- 5.5 minutes after delivery

10. How do you manage the puerperium?

Puerperium is again a critical period, since patient is likely to go in failure.

- Hospitalized for 2 weeks.
- Prophylactic antibiotics
- Puerperal fever of any origin is treated vigorously.
- Breast feeding is contraindicated only in grade III-IV disease, as the exertion of BF may precipitate failure.

11. What is the ideal contraception?

Barrier method is ideal. Permanent sterilization should preferably be done, in the interval period. If the patient is not fit, husbands vasectomy is advised.

12. When should the valvotomy be done?

Preferably in the interval period. In cases of unresponsive failure and patients' strong will to continue pregnancy, valvotomy can be considered around 14-18 weeks.

13. What are the two types of heart failures, you come across?

a. The right sided failure/congestive cardiac failure (CCF) evident by increase JVP, palpable liver and edema feet. Seen wherever considerable myocardial damage is there. Responds to diuretics, SRD, rest and digitalis.

b. The left sided failure or pulmonary edema seen in cases of tight mitral stenosis.

Tight mitral stenosis
↓
Obstruction to blood flow from left atrium to left ventricle
↓
Left atrial pressure ↑
↓
Transmitted to pulmonary veins and capillaries
↓
Pulmonary edema

S/s

1. Paroxysmal nocturnal dyspnea
2. Cough
3. Frothy sputum
4. Hemoptysis
5. Crepts, bronchospasm,

Treated by diuretics and B blockers.

14. In which cases pulmonary edema is common?

Pulmonary edema is seen in

- Young patients
- Tight Mitral stenosis
- Small hearts.
- Regular rhythm
- Efficient right ventricle
- Healthy myocardium

15. What are the indications of MTP in heart disease?

1. Primary pulmonary hypertension
2. Eisenmenger's syndrome
3. Pulmonary veno-occlusive disease
4. Severe lung disease, with pulmonary hypertension.
5. Co-arctation of aorta.
6. Marfan's syndrome with coarctation of aorta.

16. How much is the maternal mortality? and what are the causes

The maternal mortality is about 1%

The causes are:

1. Pulmonary edema
2. CCF
3. Pulmonary embolism
4. Active rheumatic carditis
5. Rupture of cerebral aneurysm.

18. What is the effect of pregnancy on heart disease?
If the cardiac reserve is po or,cardiac failure occurs.

19. What is the effect of heart disease on pregnancy?
- IUGR
- Preterm labour.

20. What is NYHA classification?
Class 1: Asymptomatic
Class 2: Symptomatic with heavy exercise
Class 3: Symptomatic with light exercise
Class 4: Symptomatic at rest

21. How will you classify heart disease?
1. RHD
2. CHD
3. IHD
4. Cardiomyopathy

22. What is critical mitral stenosis?
Valve area less than 1 sq cm.

23. What are indications of LSCS in case of heart disease?
Apart from all obstetric indications, uncorrected coarctation of aorta is the only indication for LSCS in heart disease.

24. What is prophylactic regimen for SABE/
Ampicillin 1 gm IM/IV And Gentamicin 1.5 mg/kg body weight

Give initial dose 30 min prior to procedure and repeat the dose every 8 hours for two doses.

6 Diabetes Mellitus in Pregnancy

Kanan Yelikar

1. What is gestational diabetes mellitus? GDM.

Glucose intolerance first diagnosed during the current pregnancy It is diagnosed by routine or selective screening of pregnant women.

2. When do you suspect a pre existing DM?

When fasting plasma glucose greater than 126 mg/dl, GDM diagnosed in first trimester or recurrent GDM in a subsent pregnancy or random blood glucose > 200 mg/dl.

3. How do you screen, for GDM?

Screening is done by oral 50 gm glucose without reference to food and estimation of glucose after 1 hr. If it is more than or equal to 140 mg/dl is c/as/positive glucose challenge test (GCT).

4. When is GCT done?

In low risk patients routinely at 24-28 wks (Period of maximum diabetogenesis). If GCT is negative, all low risk factors, then do not repeat. If single high risk factor is present and GCT is normal, repeat GCT after 4 weeks.

5. What are the high risk factors, for screening.

- Age > 25 years
- Obesity (BMI > 29 kg/sq.m)
- Glycosuria on two or more occasions
- PCOD
- Diabetes in first degree relative
- H/O prior GDM
- Previous HO congenital anomaly/macrosomia or previous unexplained stillbirth. Recurrent candidiasis/UTI/chronic hypertension/recurrent pre-eclampsia/RPL.

6. When and how will you do oral glucose tolerance test?

When GCT is positive, 100 gm – 3 hrs oral glucose tolerance test is done. GDM is diagnosed if FBG is > 95 mg/dl. 3 hrs post meal is 140 mg/dl.

7. What is white's classification of diabetes during pregnancy?

Class A is the least complicated (GDM)
Class B age > 20 yrs, duration < 10 years.
Class C, D, F, R, H corresponds to Benign retinopathy, nephropathy, proliferate retinopathy and coronary artery disease.

8. What are the effects of diabetes on pregnancy?

If poor glycemic control in first trimester congenital malformations seen in 22- 25%. Pathologic levels of glucose are potent, teratogens. Anomalies involve heart, central nervous system, and genitourinary system, caudal regression syndrome and sacral agenesis.

Fetal macrosomia due to fetal hyperinsulinemia, deposition of fat in subcutaneous tissue and visceral organs leading to hypertrophy.

- Unexplained stillbirth
- Abortions in uncontrolled diabetes.
- Increased infection like UTI/vulvovaginitis
- Increased incidence of preeclampsia.
- Hydramnios
- During labor shoulder dystocia due to macrosomia.

9. What are the effects of pregnancy on diabetes?

Anti insulin hormones (progesterone, HPL, cortisol etc.) result in increased insulin requirement. Pregnancy is diabetogenic.

- Pregnant diabetic women are more prone for ketoacidosis, due to rapid lipolysis.
- Accelerated ketogenesis, rapid mobilization of fat, increased ketogenesis, lower fasting glucose levels. Fasting hypoglycemia. Therefore snacks are important to prevent hypoglycemia and ketosis.

11. How will you manage a case of diabetes?

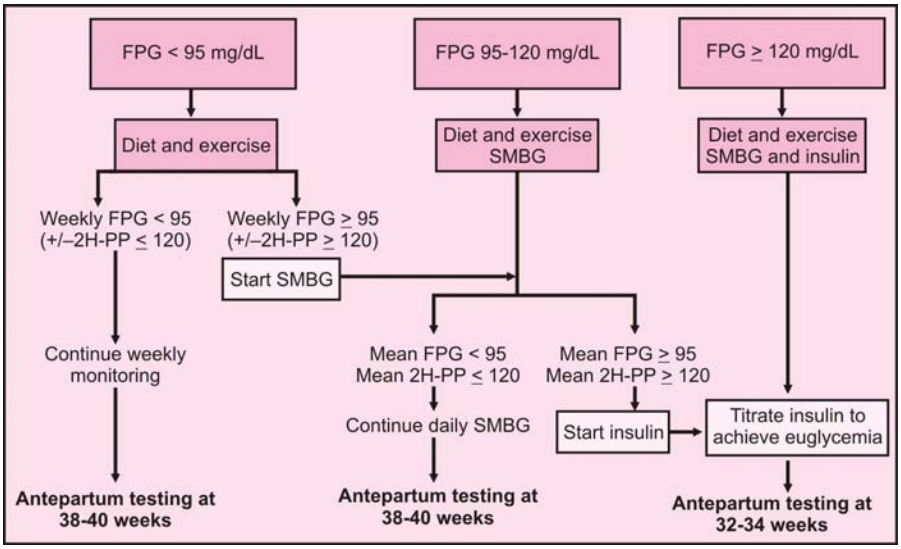
It is divided into 1) Medical management. 2) Obstetric Management.

12. What is the medical management?

1. Diet and nutrition (1800 to 2500 k cal/day, divided in 3 meals and 3 snacks)
2. Achieving glycemia SMBG (self-monitoring blood glucose postprandial 2 hr < 120 mg, 1 hr < 140 mg.)
3. Patients should be immediately switched on to inj. Insulin as soon as diagnosed.

Time of day	*Capillary glucose target (md/dL)*
Fasting	60-95
Before lunch, dinner, or bedtime snack	60-95
Oostprandial	
2 hr postprandial	< 120
1 hr postprandial	< 140
2 AM to 6 AM	60-90

13. Insulin regime



General guidelines for starting doses of insulin therapy

Trimester	*Total units of insulin*	*Twice daily*	*Splitl dose*
1st	0.6-0.7 units/kg	AM: two thirds of total	Short or rapid acting: two thirds of AM Intermediate acting: one third of AM
2nd	0.7-0.8 units/kg		
3rd	0.8-0.9 units/kg	PM: one-third of total	Short or rapid acting: one half of PM Intermediate acting: one half of PM

14. What is the obstetric management?

Antenatal screening for birth defects. Alpha fetoproteins at 16-20 weeks and detailed USG scanning (for open neural tube defect and other structural anomalies) glycosulated HB.

With good glycemic control pregnancy can safely be carried up to 37 weeks, with fetal monitoring by DFMC/NST/BPP/DOPPLER. Consider induction of labor, at 37 completed weeks of gestation.

15. What is the role of steroids in preterm diabetic patient

Steroids are better avoided since steroids result in sudden hyperglycemia, which is difficult to control.

16. Indications of LSCS.

1. Elderly primigravida
2. Multigravida with a bad obstetric history
3. Diabetes difficult to control
4. Obstetric complications like toxemia, fetal compromise
5. Presence of large baby.

17. Intrapartum maternal glycemic control.

On the morning of induction of labor or LSCS patient is kept NBM.

- Continuous glucose infusion (5% dextrose/lactated ringers solution 125 ml/hr.)
- IV insulin drip 0.5 to 1 unit/hour.
- Glucose levels should be maintained between 100-119 mg/dl.
  If blood glucose < 100 mg/dl—insulin is discontinued.
- If > 150 mg/dl glucose should be withheld.
  If > 180 mg /dl, then IV insulin bolus 2-4 units may be given.

18. Management after delivery.

Immediately after delivery, patients do not require any insulin, as glucose levels drop down.

19. What is the ideal contraception?

Barrier method.

20. Future

- Follow a diabetic diet
- Maintain ideal body weight
- Daily exercise.

21. Neonatal complications

1. Hypoglycemia
2. RDS
3. Hyperbilirubinemia
4. Polycythemia
5. Hypocalcemia.

22. Perinatal mortality

Perinatal mortality is 2-3 times more. Stillbirths are seen in last 4 weeks and are due to maternal ketoacidosis or foetal hypoglycemia.

7

Antenatal Care

Pradeep Sambarey

Health during pregnancy is directly related to health before pregnancy.

Preconceptional care and counseling is an integral part of prenatal care.

Aim is to promote, protect and maintain the health of the mother during pregnancy.

Antenatal care is a regular and periodic supervision and care of a woman during pregnancy to ensure the delivery of a term, healthy baby of idly healthy mother without any complication during and after pregnancy. Minimum nine antenatal visits are important to achieve this goal. However WHO recommends minimum 3 antenatal visits as follows:

1st at 16 weeks to 20 weeks
2nd at 28-32 weeks
3rd at term

NEED FOR ANTENATAL CARE

1. To screen high risk cases likely to develop complications to mother and baby in pregnancy, labor and puerperium.
2. Prevention, early diagnosis, investigations and treatment of these complications.
3. Immunization for tetanus and anemia prophylaxis.
4. Education and counseling to prepare the woman physically and mentally for pregnancy, labor, breast-feeding, baby-care, and contraception.
5. Advice regarding hygiene, nutrition, sanitation
6. Discussion with the couple for time and place of delivery.

1. How do you screen those cases?

Proper history taking, general and systemic examination, obstetrical examination and if required speculum and vaginal examination and specific investigations.

2. When should be the first antenatal visit?

As early as after missing first period and definitely no later than 2nd missed period.

3. What specific points you will cover in history?

Age of marriage, previous deliveries, LMP, previous losses, surgeries done, birth- weights, condition of babies, any symptoms like dyspnea, breathlessness, bleeding and any treatment received.

The aim of initial visit is to assess health status of mother and fetus which includes genetic, obstetrical and psychological risk assessment.

4. Contents of obstetrical examination.

Proper inspection, overdistension, scar on abdomen, contour, lie, presentation and position, uterine irritability and tenderness, multiple fetal parts, gross fetal anomaly, scar tenderness and fetal heart auscultation.

5. Determination of gestational age.

By LMP [Naegele' rule], any event near last period, quickening, palpation and sonography.

Naegele's rule—LMP + 9 calendar months + 7 days

LMP + lunar 10 months if previous 3 cycle are regular.

6. Define para.

It is the number of [delivered] pregnancies beyond period of viability [28 weeks].

Multipara is one having delivered 2 or more viable babies while she is Grand multi if she delivers 5 or more babies beyond viability.

7. What is gestational age and fertilization age?

Gestational age is calculated from last menstrual period while fertilization age is calculated from the date of ovulation/fertilization (2 weeks less than gestational age).

8. What is the goal for ANC care?

The final goal is to decrease maternal morbidity and mortality and deliver a live healthy baby and to reduce perinatal morbidity and mortality.

9. What are the common causes of maternal mortality in India?

Hemorrhage, anemia, Heart disease, PIH [preeclampsia and eclampsia], jaundice, obstructed labor, rupture uterus, sepsis are some common causes. A proper antenatal screening and care, identifying risk cases and timely proper management in labor can reduce maternal deaths.

10. What investigations are to be done in antenatal clinic?

Hb, urine examination, blood grouping and Rh typing, VDRL and sonography are to be done in first visit. Subsequently Blood sugar, thyroid function, TORCH test.

Doppler studies are done as and when required.

11. Need for Hb estimation to be done frequently.

As anemia is the commonest medical disorder in pregnancy and has a serious impact on pregnancy outcome, it is important to diagnose and treat anemia in pregnancy. As the most common cause of anemia is nutritional deficiency and increased demand, it can be managed with proper diet and iron therapy.

12. What is the average weight gain in pregnancy?

The overall weight gain is 11 kg with one kg in first and five kg each in second and third trimester. Less weight gain may be associated with IUGR while excessive weight gain may be a feature of preeclampsia.

13. What are common causes of oedema feet in pregnancy?

Oedema feet is diagnosed by pressing medial malleolus or lower third of tibia with thumb for 5 seconds. The common causes are physiological, preeclampsia, anemia with hypoproteinemia, cardiac failure, nephrotic syndrome, etc.

14. How will you do vaginal examination initially and what information will you get?

After emptying bladder vulva is inspected for discharge, infection, prolapse, etc.

Then speculum examination is done for vaginitis, cervix condition, external os, prolapse, etc. Bimanual examination is done for consistency and direction of cervix, uterine size, position and consistency and any adnexal pathology.

15. What information is obtained from ultrasonography in pregnancy?

First trimester—Diagnosis of pregnancy, twins, abortions, hematomas, molar pregnancy, small fibroids, ectopic pregnancy.

Second trimester—Growth of fetus, molar pregnancy, congenital anomalies, twins, intrauterine death, length of cx.

Third trimester—Growth of fetus and IUGR, liquor, abruption, placental site, major and minor anomalies, scar integrity, fetal studies.

16. What are indications for color Doppler studies?

Cases of IUGR, PIH, chronic hypertension, APH, elderly primipara, oligo-hydramnios, diabetes, poor weight gain, previous losses, decreased fetal movements are common cases for Doppler studies. Uterine artery, umbilical vessels and middle cerebral artery are considered for these studies.

17. What is Triple test?

Triple test includes maternal serum alpha feto-proteins, serum estriol and beta HCG estimation. It is done at 16-18 weeks to diagnose Down's syndrome. In addition inhibin A is added in Quadrupal test.

18. What are the indications for genetic studies?

1st trimester: 1) nuchal thickness, and 2) CVS.

Cases like elderly women, consanguineous marriage, previous anomalous child, gross hydromnios, diabetic women, gross IUGR, etc. are subjected for genetic studies.

Low serum alpha feto proteins, low pregnancy associated plasma protein-A and elevated HCG are suggestive of Down's syndrome. Amniocentesis is done between 14 to 16 weeks while cordocentesis is done at 18 weeks for karyotyping, fetal anomalies, thrombocytopenia, cord blood studies and fetal therapy.

19. When is screening for gestational diabetes done?
In low risk cases it is done at 24-28 weeks while high risk cases (family history, previous hydromnios, macrosomias, stillbirths, neonatal deaths, moniliasis, obesity) are subjected to screening as soon as pregnancy is diagnosed.

20. What is the risk in smoking and alcoholic women?
In smoking women there is increased fetal carboxyhemoglobin, reduced uteroplacental blood flow and fetal hypoxia. The effects are abortions, low birth weight, prematurity, IUGR, intrauterine deaths and abruption.

Alcohol is like a potent teratogen and cause fetal alcohol syndrome characterized by growth restriction, facial abnormalities and CNS dysfunction.

21. Is HIV testing mandatory in pregnancy?
Though is not mandatory, it is ideal to do HIV testing of a woman during pregnancy as there are about 40% chances of vertical transmission of HIV to the fetus. But is important to do pretest counseling and an informed consent is obtained for HIV testing.

22. What in PNDT act?
Prenatal diagnostic tests including ultrasonography are done only to detect genetic/chromosomal/congenital anomaly and sex determination is not to be done.

23. What dietary advice is given to the pregnant woman?
Her diet should adequate proteins, carbohydrates and important nutrients. Fresh green vegetables, fruits, milk, eggs, meat, fish, cheese, pulses should be part of her diet.

24. What are the social factors in improving antenatal care?
Proper health education and awareness, removing false ideas and beliefs, access to ANC clinics, dietary improvements, audiovisual aids, communication with local leaders and health workers, holding camps and meetings, counseling etc. will help in improvement of ANC care and services.

25. Should air travel be done in pregnancy?
Air travel [5000 feet and more] should be avoided in 1st and 3rd trimester due to hypoxic effects on fetus, premature delivery and possible deleterious effects on development.

26. In what cases you anticipate poor fetal growth?
Cases like PIH, chronic hypertension, nephritis, anemia, poor nutritional status, twins, smoking, alcoholic women, APH, elderly women, etc. should be anticipated for IUGR.

27. What is the importance of folic acid in pregnancy?
Folic acid deficiency is associated with abortions, abruption, PIH, neural tube defects and poor growth of fetus [Requirement-400 micrograms per day].

28. What symptoms are considered important in pregnancy?
Symptoms like dyspnea, palpitation, pain in abdomen, vaginal bleeding, syncope, vomiting , headache, visual symptoms, watery discharge, chest pain, breathlessness, less or absent fetal movements are important in pregnancy.

29. What are various causes for pain in abdomen in pregnancy?

Women complaining of pain in abdomen should be examined for abortion, vesicular mole, ectopic pregnancy, retroverted gravid uterus, preterm labor, abruption

Rupture uterus, torsion of gravid uterus or ovarian cyst, associated surgical and medical conditions, etc. depending on gestational age.

30. Importance of blood grouping and Rh typing.

Every pregnant woman should have her blood group and Rh factor known. Rh –ve woman is at risk of isoimmunization of the fetus, severe anemic woman will require blood transfusion, cases of APH and PPH, V mole, ectopic pregnancy, hemoglobinopathies will also require blood.

31. Indications for hospitalization in pregnancy.

All high risk pregnancies require hospitalization as and when required. Cases of PIH, Heart disease, APH, uncontrolled diabetes, severe anemia, severe IUGR, etc. will require admission earlier.

32. How will you minimize perinatal morbidity and mortality?

Good antenatal care, screening for high risk cases, prevention of prematurity, improving general condition of mothers, analyzing previous losses, investigating such cases, timely hospitalization, proper conduction of labor, making neonatal care facilities available, contraceptive advice, immunization, etc. will help in improving perinatal outcome.

NONSTRESS TEST

Relationship between fetal movement and fetal heart rate is documented and studied.

Basal heart rate of 120-160, two acceleration of 15 beats for 15 seconds, good beat to beat variability, no spontaneous decelerations are considered as reactive NST.

Reactive NST means the baby is well at least for 1 week. Nonreactive NST is an indication of intrauterine fetal hypoxia.

CONTRACTION STRESS TEST

Relationship between uterine contractions [induced] and fetal heart rate documented.

It is a test for uteroplacental reserve or sufficiency. Decelerations followed by uterine

Contractions is a +ve CST, while no decelerations is a –ve CST.

BIOPHYSICAL PROFILE

2 points for +ve and 0 points for –ve finding.

1. Fetal breathing movements—1 breathing for 30 second in 30 minutes.
2. Fetal gross body/limb movements—3 body/limb movements in 30 minutes.
3. Fetal tone—1 movement of extension followed by flexion in 30 minutes.
4. Reactive NST.

5. Amniotic fluid pocket of 2 cm or more.
   Score of 8-10 satisfactory, score of 6 means possible hypoxia.
   Score of 4 means probable hypoxia, 0-2 means hypoxia or compromised fetus.

33. Contraindications in pregnancy.

All live virus vaccines (rubella, measles, mumps, varicella, yellow fever). Tetracyclines, chloramphenicol, cytotoxic drugs, DES, lithium, valproate, alcohol, ACE inhibitors, mephiprostone, radioactive iodine.

34. How do you examine ANC?

- Height
- Weight
- Blood pressure
- Hemoglobin
- PPTCT
- Urine—routine and microscopy
- Blood group Rh typing.

35. How do you classify high risk factor?

1. Social factor
   a. Age
   b. Height
   c. Weight
   d. Literacy
   e. Parity
   f. Socioeconomic
   g. Nutrition.
2. Past Pregnancy performance
3. Pre existing medical/surgical illness.
4. Present pregnancy factors.

Q. What is BOH?

Bad obstetric history is defined as when the present pregnancy outcome is affected by the disaster in the past pregnancy.

Q. Gravidogram

1. Plot the symphysis—fundus height.
2. Use the symphysis—fundus height graph to assess whether the fetus is growing adequately.
3. Plot the patient's weight and assess whether the weight gain is normal.

8

Normal Labor

Kanan Yelikar, Ashwini Yelikar

1. How will you define labor?

Labor is the process by which viable fetus is expelled out of the uterus.

2. What is normal labor?

Normal labor is defined as:

- Spontaneous in onset.
- With vertex presentation
- Unaided (without help of forceps/ventouse)
- Completed within 12-18 hrs in prime and 8-20 hrs in multi.
- With a healthy mother and a healthy baby.

3. How do you calculate expected date of delivery EDD.

Naegles formula EDD = 9 calendar months +7 days or 10 lunar months or 280 days or 40 weeks from LMP (last menstrual period).

4. What percentage of patients deliver on EDD?

Only 4% deliver on the given EDD.
80% deliver 2 weeks earlier or 1 week later.

5. What are the theories of initiation of labor?

The proposed theories are:

1. Fetoplacental contribution: accelerated production of oestrogen and prostaglandins.
2. Alteration in concentration of estrogen: progesterone ratio.
3. Triggered synthesis of prostaglandins
4. Increased activity of receptions in uterus

6. How will you differentiate between true and false labor pains?

True labor pains are characterized by (TLP)

- TLP start as low backache and radiated towards medial side of the thigh.
- The intensity, frency and duration of contraction increases as time passes on.
- TLP not relieved by analgesics or plain water enema.
- TLP is associated with show (blood stained mucous discharge) simultaneous dilatation of cervix and hardening of uterus.
- TLP associated with formation of bag of waters.

7. What are the stages of labor?

Labor is divided conventionally in 3 stages. Fourth stage of observation is added to it.

First stage of labor starts with onset of true labor pains up to full dilation of cervix (i.e. 10 cm of dilatation) Average duration is 12 to 18 hrs in primi and 6 to 10 hours in multi. Second stage of labor is defined as starts from full dilation of cervix up to expulsion of fetus (1-2 hrs) Third stage: Begins with delivery of baby up to expulsion of placenta (15-30 mins.)

Fourth stage: stage of observation (1 hour after the expulsion of placenta)

8. What is retraction?

The muscle fibres are permanently shortened after each contraction. This shortening is called as retraction.

9. What are events in first stage of labor?

- Uterine contractions
- Cervical dilation and effacement.
- Descent of the presenting part.

10. What is cervical effacement?

Taking up of the cervix in the formation of lower uterine segment. It is described as percentage of the length of the cervix, which is included in the LUS. For example, 25%, 50%, 75% and 100% effaced is fully effaced.

11. Events in the second stage?

Second stage deals with the expulsion of fetus. Normal duration of second stage is up to 2 hrs in primi and 1 hour in multi.

12. What are the causes of prolonged second stage of labor.

The causes could be faults in the power, passage or passenger.
For example, power—Incoordinate uterine action
Passage —Contracted pelvis
Unforeseen cephalopelvic disproportion
Passage—Malposition like persistent occiptio-posterior position.

13. What are the events of third stage of labor.

Separation of placenta and expulsion of placenta along with the membranes.

14. What are the ways of separation of placenta?

1. Schultze method: Central separation of placenta.
2. Mathew Duncani: Marginal separation of placenta.

15. How is the 3rd stage bleeding controlled?

1. Contraction and retraction of uterine muscle fibers leads to constriction of the blood vessels, due the "figure of eight" arrangement of fibers also called as living ligatures.
2. Apposition of the walls of the uterus.
3. Natural clotting mechanism control the bleeding.

16. How will you mange a normal labor?

Confirm that the patient is in active labor, i.e. cervical dilatation more than 3 cm. Preliminary P/V examination to confirm,

- dilatation and effacement of cervix.
- status of membranes/liquor
- Position/Station of vertex.
- Assessment of adequacy of pelvis.
- Watch for progress of labor by doing P/V examination every 4 hour and plotting the partogram.

17. How will you reduce pain during labor?

Pain relief by inj. Tramadol 50 mg im at 4 cm of dilatation or inj. diazepam and fortwin (2 mg + 6 mg IV) helps to alleviate pain.

18. How do you monitor the fetus?

Normal fetal heart rate ranges from 120-140 per minute.

19. What is fetal distress.

A FHR < 120 or more than 160 is called as Fetal distress. Meconium stained liquor is confirmatory of fetal distress.

20. What is caput succedaneum?

It is a soft swelling on the fetal head, which is due to serous collection beneath the scalp, due to venous congestion, due to compression of fetal head by the cervical rim (girdle of contact)

21. What is the site of formation of caput?

It is usually formed on the posterior surface of either of the parietal bone, depending on the position of the vertex. It is not limited by the sutures and hence irregular in shape.

22. What is cephalhematoma?

Cephalhematoma is a hemorrhagic collection between the pericranium and the flat bone of the skull. It is unilateral, limited by the suture lines of the skull.

23. What is the treatment of cephalhematoma?

No active treatment is necessary. Blood is absorbed within 6 to 8 weeks of time. Resorption of the hematoma leads to jaundice in the neonate.

9

Partograms

Shyam Desai

INTRODUCTION

The time honored mean cervical dilatation—Time Curve of Friedman (1955) constitutes the basis for partography. The descent of the presenting part normally begins well before the cervix reaches full dilatation and progresses until the presenting part reaches the perineum. The pattern is very variable and is influenced by parity as well. The sigmoid curve for cervical dilatation and the slope of fetal descent, at best, should be considered as idealized visual aids to comprehend the temporal relationships of cervical dilatation and the descent of the presenting part. The concept of the nomogram introduced by John Studd (1973) provides a basis for comparison of progress of labor in a eutocic and dystocic patient. The addition of 'Alert Line' and 'Action Lines" to the partogram help to draw attention to an emerging dystocia and prompt the clinician in charge of the case to take remedial actions in time or to transfer the patient to a better facility where appropriate action can be undertaken in good time to ensure satisfactory obstetric outcome. Partography forms the basis of the concept of "Programmed Labor" (Daftary et al 1985) which provides close supervision of an actively managed labor, satisfactory labor analgesia and improved obstetric outcome.

HISTORY

- Friedman E (1955)—Mean cervical dilatation time curve
- Studd J (1973)—Nomogram
- Philpott RH and Castle WM—Cervicographs and alert and action lines.
- Daftary SN, et al. (1977)—Cervicographs of Indian parturient primigravida
- Daftary SN, et al. (2001) Concept of programed labor.

Illustrative Partogram

This represents a dynamic documentation of the events in labor as they unfold in time. And provide a record of the parameters denoting maternal and fetal well-being. A constant reference of the patient's partogram to a nomogram provides markers for detecting dystocia in good time. The alert and action lines provide guideposts for judging the efficacy of interventions adopted to

rectify dystocia and lastly—alert the clinician to adopt timely obstetric interventions to achieve optimum obstetric outcome.

Contents

Besides recording cervical dilatation and the station of the presenting part with passage of time in labor. The partogram also documents the following additional information.

- Temperature—every 3-4 hours
- Pulse rate—every half hour
- Blood pressure every half hour
- Fetal heart rate (FHR)—every 15 minutes
- Uterine contractions every 15 minutes
- Cervical dilatation—every 2-4 hours, depending on the stage of labor and parity.
- Station of the fetal head—every 2-4 hours.
- Liquor and moulding
- Medication and IV fluids
- Urine output—test albumin, sugar and acetone.

Interpretations

Diagnostic Criteria

Labor pattern	*Nullipara*	*Multipara treatment*	*Preferred treatment*	*Exceptional*
Prolongation disorder				
1. Prolonged latent phase	> 20 hr	> 14 hr	Rest and sedation	Oxytocin Induction/cesarean section (CS)
Protraction disorders				
1. Protracted active phase Rate of cervical dilatation	<1.2 cm/hr	< 1.5 cm/hr	Expectant and	CS for CPD
2. Protracted descent	< 1.0 cm/hr	< 2.0 cm/hr	Supportive treatment	CS for CPD
Arrest disorders				
1. Prolonged deceleration	> 3 hrs	> 1 hr	No CPD – oxytocin	Rest if exhausted
2. Secondary arrest of dilatation	> 2 hrs	> 2 hr		
3. Arrest of descent	> 1 hr	> 1 hr	Suspected CPD—CS	Cesarean section
4. Failure of descent	Failure of descent		With CPD–CS	

Results

- Reduced incidence of prolonged labor
- Early detection of dystocia
- Early implementation of supportive measures
- Early detection of fetal/maternal distress
- Timely transfer of difficult labor cases
- Anticipation of obstetric transfer
- Avoidance of difficult obstetric interventions
- Improved maternal morbidity and mortality
- Improved perinatal outcome

- Optimizing outcome of labor
- Promoting the concept of safe motherhood.

Programmed Labor

This concept combines

- Partographic management of labor
- Active management of labor
- Recognizing dystocia early
- Providing timely supportive treatment
- Labor analgesia
- Timely and safe obstetric intervention
- Avoiding traumatic deliveries
- Active management of the third stage of labor to reduce blood loss and prevent PPH
- Promoting the concept of safe motherhood.

Questions

1. Name the persons who contributed to Partography

a. E. Friedman
b. John Studd
c. Phillpot and Castle
d. Daftary et al.

2. What is partogram?

a. It refers to the mean cervical dilatation—time curve
b. It consists of: (i) Latent phase (ii) active phase, and (iii) phase of deceleration.

3. What abnormal labor patterns can you diagnose with the help of partogram?

a. Prolongation disorder
b. Protraction disorders
c. Arrest disorders.

4. What is prolonged labor?

a. Duration > 20 hrs in nullipara or > 14 hours in multipara
b. It should be treated with immediate cesarean section
c. Rest and sedation are helpful to correct the disorder
d. Induction of labor is indicated, if pregnancy termination is desirable, if the cervix is unfavorable—consider cesarean section.

5. What is protracted active phase of cervical dilatation? How will you treat it?

a. Rate of cervical dilatation < 1.2 cm /hr in a primigravida
b. Rate of cervical dilatation < 1.5 cm/hr in a multipara
c. Consider administration of oxytocin in absence of CPD.

6. What is secondary arrest of labor?

a. Non progress in cervical dilatation for > 2 hours in a nullipara

b. Non progress of cervical dilatation for > 2 hours in a multipara
c. Consider presence of hitherto unsuspected CPD.

7. When will you call a case as arrest of descent?
a. Failure of descent of presenting part > 1 hour.
b. Under observation the caput increases
c. In cases of occiptoposterior presentations—time required may be more.
d. Deliver the baby by cesarean section if the station is low.

8. What is programed labor?
a. Monitor progress of labor on a partogram
b. Monitor fetal well-being on electronic fetal heart rate monitor
c. Provide labor analgesia
d. Active management of labor.

9. What are the benefits of programed labor?
a. Provides pain relief and comfort during labor
b. Does not require active supervision
c. Reduces duration of labor
d. Improves obstetric outcome.

10. What are the drugs used in programed labor?
a. Oxytocin to optimize pains
b. Morphine to relive pain
c. Drotavarine to facilitate cervical dilatation
d. Active management of third stage of labor to reduce its duration and blood loss.

11. What is alert line?
Alert line is a nomogram, i.e. the course of labor followed in normal labor? Patients partogram should always be on the left of alert line. When patients partogram crosses the alter line on the right side, it denotes prolonged labor.

12. What is action line?
WHO has given a second line, parallel to alert line, 4 hours apart from alert line.

13 What is to be done, when patients partogram crosses the alert line?
Refer the patient to a higher centre. Reevaluate the patient. The commonest cause is unforeseen CPD. Revise the decision rule out CPD and labor can be enhanced by ARM or oxytocic agents.

14. What will you do if the line crosses action line.
Take action, LSCS.

15. What are the advantages of partogram?
Can be used by paramedicals.
- Early referral after detection of labor abnormalities.
- Early interventions in abnormal labor patterns.
- Avoid maternal morbidity and improve perinatal outcome.

10

High Risk Pregnancy

Manorama Purwar, Ashish Zararia

1. Define High risk pregnancy?

Pregnancy is defined as high risk when the probability of an adverse outcome for the mother or neonate is increased over and above the baseline risk of that outcome among the general pregnant population by presence of one or more risk factors.

2. What do you mean by a risk in high risk pregnancy and how do you calculate it?

Risk reflects the incidence of an adverse health outcome and it is calculated as odds ratio.

3. Why there is a need to focus on high risk pregnancy?

At primary and secondary health care center recognition of high risk pregnancy and timely referral to higher health care centre is essential. High risk pregnancy needs vigilance and more care during antepartum, intrapartum and postpartum period to improve pregnancy outcome. For example recognition of a case of preeclampsia can prevent its complications.

4. What is the role of preconception counseling in high risk pregnancy?

By counseling we can explain the magnitude of risk and special investigation may be carried out to improve obstetric outcome wherever possible.

5. How preconception care is helpful in diabetes and neural tube defect?

Normalization of blood glucose values in diabetic women and supplementation of folic acid may prevent neural tube defect.

6. What is the risk in pregnancy for following ethnic groups?

A. Indian subcontinent, middle east
B. Mediterranean, American Black
C. Far east
D. Africa

A. Thalassemia, sickle cell disease
B. G6PD deficiency
C. Hepatitis B (chronic carrier)
D. HIV infection leading to MTCT (mother to child transmission).

7. What are the maternal and fetal risks of smoking?

Maternal

1. Preterm delivery
2. Placental complications like placental insufficiency and placental abruption
3. Intrauterine growth retardation
4. Premature rupture of membranes
5. Ectopic pregnancy.

Fetal

1. Low birth weight babies (LBW)
2. Neurobehavioral abnormality
3. Sudden infant death syndrome
4. Cleft lip/cleft palate.

8. How do you prevent it?

1. Counseling and social support
2. Stopping smoking by midpregnancy will improve outcome
3. Reducing smoking is better for pregnancy if not possible to stop completely
4. Specific targeted intervention program for high risk group.

9. What is the daily requirement of folic acid during pregnancy?

400 µg /day (0.4 mg /day) periconceptionally (at least 4 weeks before and 8 weeks after conception).

10. How do you classify underweight and overweight woman during pregnancy?

Two ways

1. Body mass index (BMI) calculated as (kg/m^2)
   Thin < 20
   Normal 20 to 24
   Overweight 25 to 30
   Obese > 30.
2. Prepregnancy body weight
   Underweight < 45 kg
   Overweight > 85 kg.

11. What are the obstetric problems of overweight woman?

1. Pre eclampsia
2. Gestational diabetes
3. Shoulder dystocia
4. Postpartum hemorrhage
5. Postoperative morbidity.

12. What is the total weight gain by a healthy nulliparous pregnant woman during normal pregnancy?

12.5 kg
Upto 20 weeks: 4 kg
After 20 weeks: 8.5 kg.

13. What do you mean by teenage pregnancy?
Pregnancy in a girl aged between 13 to 19 years.

14. What do you recommend to pregnant woman resting for long distance air travel?
Long distance air travel is recommended up to:
1. 36 weeks gestation in singleton pregnancy
2. 32 weeks gestation in twin pregnancy
3. Need to prevent deep vein thrombosis is to be emphasized.

15. Define Grand multipara and its complications.
Woman who had 4 or more viable births is a grand multipara
Complications are:
1. Antepartum hemorrhage
2. Anemia
3. Labor abnormalities
4. Postpartum hemorrhage

16. Enumerate obstetric problems of elderly primi?
1. Hypertension
2. Gestational diabetes
3. Prolonged labor
4. Congenital abnormalities of newborn

17. What is Rh sensitization?
Rh negative mother giving birth to an Rh positive child is Rh sensitization

18. In which trimester chances of congenital syphilis are high?
Second trimester of pregnancy

19. How TORCH group of infection affects a fetus?
- Fetus may have
- Hydrocephalus
- Chorioretinitis
- Convulsion
- Jaundice

20. Name few single gene disorders? To whom you will offer prenatal diagnosis for single gene disorder:
Single gene disorders are:
1. Achondroplasia
2. Sickle cell disease
3. Thalassemia
4. Cystic fibrosis
5. Friedrich's ataxia
6. Hemophilia.

Women will be offered prenatal diagnosis when there is
1. H/O previously affected child
2. Family history
3. For recessive diseases carrier screening programs like sickle cell disease.

21. What are the technis available for prenatal diagnosis?

1. Biochemical assay (triple test)
2. Ultrasound
3. DNA diagnoses.

22. How do you Screen a high risk woman through history?

- Age < 20 or > 29 years
- H/O two or more abortions (spontaneous or induced)
- Stillbirth
- Previous Congenital anomaly malformation
- Preterm birth
- Birth weight of previous baby > 3.5 kg
- Grand multipara
- Previous LSCS or uterine surgery
- Difficult forceps delivery
- Preeclampsia/eclampsia
- Antepartum hemorrhage
- Anemia
- 3rd stage complication
- Previous medical disease.

23. What investigations need to be done for evaluation of high risk pregnancy preconceptionally?

- Hb, CBC
- ABO, Rh typing
- Blood sugar: fasting and post meal
- Screening for hemoglobinopathy
- VDRL
- TORCH infection
- HIV
- Hepatitis B, C
- Urine routine
- Cervical culture
- Cervical cytology.
- Thyroid profile
- Hysteroscopy and laparoscopy for müllerian duct anomaly
- Ultra sonography of pelvis
- Screening for sexually transmitted disease.

24. How nutritional factor affects pregnancy?

1. Folic acid deficiency causes neural tube defect
2. Iron deficiency produces anemia and its complications
3. Diet containing low proteins lead to hypoproteinemia and its complications; diet containing low proteins and low calories lead to intra uterine growth restriction
4. Obesity has its own complications.

25. What are the social factors which makes pregnancy high risk?

1. Substance abuse
   Drug abuse
   Alcohol
   Smoking
2. Domestic violence
3. Occupational hazard
4. Improper housing
5. Financial constraints.

26. How do you assess fetal well being in high risk pregnancy?

- Establish gestational age
- Monitor fetal growth
- Monitor fetal well being by daily fetal movement count, nonstress test, and
- Biophysical profile, CST, umbilical and uterine Doppler.

27. How do you establish gestational age?

- Menstrual history
- USG
- Uterine size

28. How do you evaluate a high risk pregnancy?

These can be evaluated under 4 headings:

I. Social factors
   a. Age < 16 years, > 35 years
   b. Parity primipara and grandmultipara
   c. Height > 139 cm
   d. Weight < 45 kg and > 85 kg

II. Past pregnancy factors
   a. H/o infertility
   b. H/o two or more abortions
   c. H/o third stage complication
   d. H/o child birth > 9 lbs and < 5 lbs
   e. H/o difficult labour
   f. H/o LSCS
   g. H/o PIH

III. Medical and surgical factors
   a. Gestational diabetes
   b. Chronic renal disease
   c. Cardiac disease
   d. Other significant medical disorder.

IV. Present pregnancy factors
   a. Rh negative mother
   b. Anemia
   c. Malpresentation
   d. Hydramnios
   e. IUGR
   f. Hypertension
   g. Postmaturity
   h. PROM
   i. Bleeding < 20 weeks and > 20 weeks.

11

PROM

Sadhana M Tayade

1. What is prelabor rupture of membranes (PROM)?

Prelabor rupture of membranes is rupture of membranes beyond 37 weeks of pregnancy and one hour before onset of labor.

2. What is PPROM/SPROM/prolonged ROM?

PPROM—Preterm prelabor rupture of membranes before 37 weeks of gestation.

SPROM—Spontaneous prelabor rupture of membranes is ROM after or with onset of labor.

Prolonged ROM—Any ROM that persists for more than 24 hours and prior onset of labor.

3. What is the incidence?

Incidence of PROM is 8% Incidence of PPROM is 2%

4. What are the high risk factors?

High risk factors are:

- H/o PROM in previous pregnancy
- Low socioeconomic status
- Tobacco chewer
- Uterine over distention
  - Polyhydramnios
  - Multiple gestation pregnancy
- Cervical or vaginal infection
- Bacterial vaginosis
- Group B Streptococci
- Mycoplasma
- Ureaplasma
- *Neisseria gonococci*
- Chlamydia intercourse.

5. What will be the associated symptoms?

Patient will mainly have complaint of gushing of fluid from vagina.
She may have complaint of fever and pain in abdomen.

6. What are the latrogenic causes of PROM?

PROM can be seen in following cases:

- Amniocentesis
- Cervical encirclage.

7. What will be the differential diagnosis according to the symptoms?

a. Urinary incontinence
b. Vaginal discharge.

8. How will you confirm the diagnosis?

A. Methods to confirm rupture of membranes
   1. Vaginal pooling
   2. Vaginal fluid ferning
   3. Vaginal fluid pH (nitrazine).

B. Other bedside evaluation
   1. Visualize cervix with speculum to estimate dilation
   2. DNA probe for Chlamydia and gonorrhea
   3. Group B Streptococcus culture from vagina and rectum
   4. Fetal monitoring for well-being.

C. Laboratory investigations blood (CBC, CRP):
   Urine (routine and microscopy)
   Vaginal swabs.

D. Advanced diagnostics to consider:
   1. Consider ultrasound
      a. May help confirm diagnosis (oligohydramnios)
      b. Determines fetal position and placental location
      c. Estimates fetal weight.
   2. Amniocentesis
      a. Evaluate fetal lung maturity
      b. Method to confirm ROM in uncertain cases
         i. Uses indigo carmine dye 1 ml in 9 ml sterile NS
         ii. Instilled into uterus via amniocentesis
         iii. Vaginal tampon turns blue within 30 min in ROM.

9. What is the pathophysiology of PROM?

At term, programed cell death and activation of catabolic enzymes, such as collagenases and mechanical forces result in ruptured membranes. PROM occurs probably due to the same mechanisms. It is also appears to be linked to underlying pathological processes, most likely due to inflammation and infection of the membranes.

10. What is the nitrazine paper test?

It is a diagnostic test that is around 90% sensitive. The normal vaginal pH is between 4.5 and 6.0 where amniotic fluid is more alkaline with a pH of 7.1-7.3. Nitrazine paper turns blue if patient has PROM, as a pH of vaginal fluid will be more than 6.0.

11. What are the causes of false-positive nitrazine paper test?
- Bacterial vaginosis
- Blood/semen in vagina
- Alkaline antiseptics.

12. What is the significance of ferning pattern?
Ferning (Arborization) under light microscope indicate PROM.

A separate swab should be used to obtain fluid from posterior fornix or vaginal sidewalls. Contamination with vaginal blood may cause false-negative test and cervical mucus can result in false positive. Ferning is due to drying of salts, contained in amniotic fluid.

13. What are the types of swabs that should be taken?
- Posterior fornix
- Vaginal sidewalls
- Cervical swab for chlamydia, gonorrhea.
- perianal/anal swab for Group B Streptococcus.

14. What are the maternal complications?
- Chorioamnionitis
- Placental abruption
- Operative intervention.

15. What are the fetal complications?
- Preterm birth
- Respiratory distress syndrome
- Fetal restriction deformities
- Pulmonary hypoplasia
- Increased neonatal morbidity and mortality.

16. When will you decide expectant line of management?

Expectant management of PROM include a course of antibiotics with steroids. Expectant management and waiting for spontaneous Labor may be considered in selected patients for 1st 12-24 hours, depending upon maternal and fetal condition.

17. What are the factors that determine outcome of PROM?
Outcome of PROM mainly depend upon maternal infection, duration of PROM and gestational age of pregnancy. Plan of management of PPROM remote from term should include the neonatal and maternal medical team. These patient's should be delivered at places with the availability of NICU.

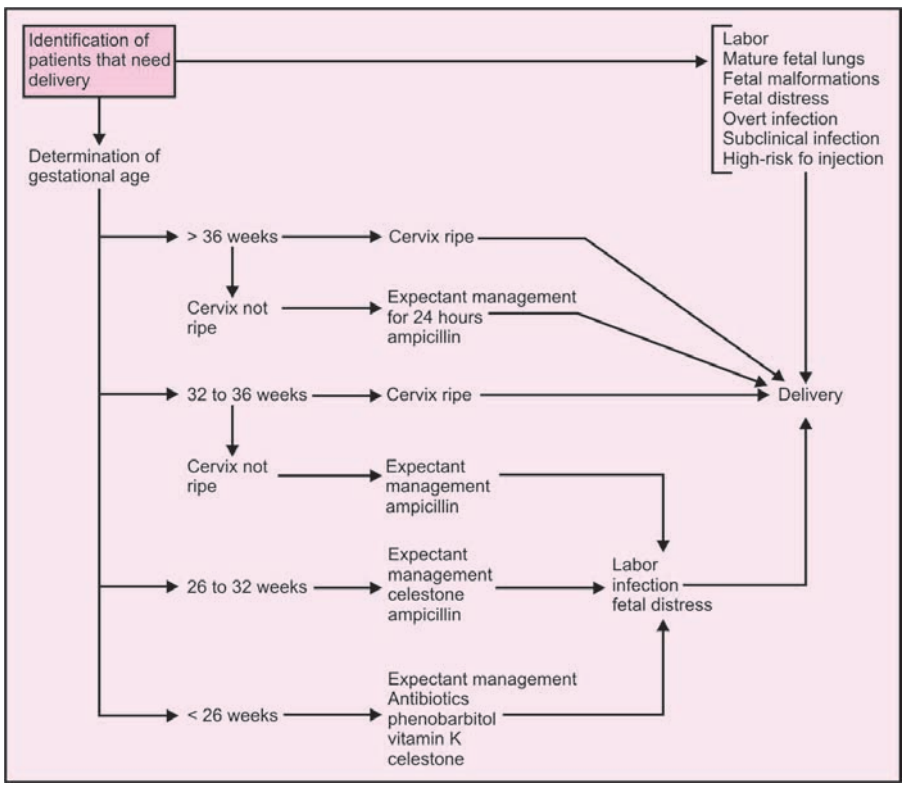
18. What is the medical line PPROM of management?
Medical line of management include antibiotics and steroids:
Corticosteroids decrease perinatal morbidity and mortality after preterm PROM. The most widely used and recommended regimens include intramuscular betamethasone (Celestone) 12 mg every 24 hours for two days, or intramuscular dexamethasone (Decadron) 4 mg every 6 hours for 24 hours.

Antibiotics: Giving antibiotics to patients with preterm PROM can reduce neonatal infections and can be given according to following regimen.

1. Erythromycin 250 mg
2. Co-amoxiclave 375 mg
3. Erythromycin and co-amoxiclave.

Tocolytic therapy: Tocolytic therapy may prolong the latent period for a short time but do not appear to improve neonatal outcomes. A short course of Tocolytic drugs can be administer to allow initiation of antibiotic, corticosteroid administration, and maternal transport.

19. How will you manage a case of PROM according to the gestation?



20. How you will manage term prelabor rupture of membranes (PROM)?

A. Induction of labor
   1. Fetus 36 weeks gestation
   2. Weight > 2500 grams
   3. Fetal lung maturity adequate.

1. Methods for induction of labor
2. Consider oxytocin induction of labor.
   1. Spontaneous labor onset within 48 hours in 90%
   2. Oxytocin decreases PROM infection rates
   3. Oxytocin does not increase cesarean rates in PROM.

3. Consider cervical ripening if unfavorable cervix.
   a. Decreases risk of chorioamnionitis in PROM
   b. Does not increase cesarean rate in PROM
4. Indications' for GBS prophylaxis
   a. Prolonged ruptured membranes anticipated > 18 hours
   b. Fever > 38°C.

21. When will you deliver the patient immediately?

1. Labor
2. Mature fetal lungs
3. Fetal malformation
4. Fetal distress
5. Overt infection
6. Subclinical—infection (as seen in repeated PIV/DM/sickle cell/Rh D, immunosuppressants).

12

Postdatism

Hemant Deshpande

1. What is the definition of post-term?

Prolonged pregnancy is used to refer to those pregnancies advancing beyond the expected date of delivery (EDD). Postdatism term, better to be avoided.

Post-term is used to designate pregnancies that advance beyond 42 weeks or 294 days from the first day of last menstrual period.

2. What is the Incidence of post-term?

Incidence of post-term pregnancy is 7.5% when the diagnosis is based on menstrual dating. The incidence is 2.6% when diagnosis is based on early ultrasound examination and 1.1% when ultrasound and the menstrual history coincided.

Incidence: Average is 7-8% although it varies form 3-14%

Incidence is low where:

1. Where first trimester USG is routinely done and gestational age is confirmed
2. Where induction is routinely practiced when patient crosses her EDD.

3. What is the etiology of post-term pregnancy?

1 Idiopathic—in most cases the cause is unknown
2 Anencephaly without hyrdamnios
3 Placental sulfatase deficiency
4 Adrenal hypoplasia in fetus
5 Hereditary—in family and also in patient herself post date there is 3 times increased chance of recurrence
6 Extrauterine pregnancy
7 Diabetes
8 Hypothyroid gravida
9 Age (elderly), parity (primi), sex (male fetus) and sedentary habits.

4. What is postmaturity dysmaturity syndrome?

Only 20-40% cases of post-term pregnancy show features of post maturity as follows:

- Loss of subcutaneous fat and muscle mass

- Long thin limbs
- Dry skin (dehydration) with wrinkling and desquamation of epidermis (peeling off)
- Absence of vernix caseosa and lanugo hair
- Long nails and hair
- Meconium staining of skin, nails and amnion
- Attentive apprehensive look
- Sole-creases.

25% of the remaining pregnancy show macrosomia (wt > 4.5 kg) and 40-50% quite normal.

5. Clifford's classification of postmature infant.

Stage I—Clear amniotic fluid
Stage II—Skin is discolored green.
Stage III—Skin yellowish-green

6. Determination of gestational age?

Clinical: Known LMP
P/V examination in 1st trimester
USG between 7 to 11 weeks.
Date of fruitful coitus (Specially during ART)

- Quickening 18-20 weeks in primigravida, 16-18 weeks in multigravida
- USG between 16 weeks to 24 weeks.

7. What do you mean by excellent dates?

Known LMP with regular cycle, not using OC pills, during the last 3 months and the last period was normal in duration and amount of flow.

- USG between 7 to 11 weeks
- USG between 16 to 24 weeks.

8. What are the amniotic fluid changes associated with prolonged gestation?

Amniotic fluid volume decreases.

38 weeks 1000 ml
40 weeks 800 ml
42 weeks 480 ml
43 weeks 250 ml
44 weeks 160 ml

Amniotic fluid < 400 ml after 40 weeks of gestation is associated with fetal complications.

9. What are the placental changes associated with prolonged gestation?

Placenta shows a decrease in diameter and length of chorionic villi, fibrinoid necrosis, and accelerated atherosis of chorionic and decidual vessels. Appearance of hemorrhagic infarcts, foci for calcium deposition and formation of white infarcts seen. Infarcts are present in 60 and 80% of post-term placentas, and they are more common at the placental borders.

10. What are the maternal risks in post-term pregnancy?

As such perse, due to postterm there is no risk to the mother except for secondary to induction and operative deliveries (forceps, LSCS).

11. What fetal problems are associated with prolongation of pregnancy?

1. Intrapartum fetal distress—25% (umbilical cord compression and chronic placental insufficiency)
2. Meconium aspiration syndrome
3. Fetal trauma due to macrosomia (shoulder dystocia)
4. Post maturity syndrome.

Perinatal problems:

A. Mortality doubles in 43rd weeks and 5 times in 45th week. There is increase in intrapartum deaths.
B. Perinatal morbidity increases by 35% in post-term pregnancy.
   1. Intrapartum fetal distress (birth asphyxia) due to:
      a. Placental insufficiency
      b. Cord compression
      c. Oligohydramnios
      d. Meconium aspiration
      e. Prolonged labor.

      Perinatal morbidity in post-term pregnancy
   2. Hypoglycemia in newborn
   3. Hypovolemia
   4. Polycythemia
   5. Adrenocortical hypo function
   6. Intracranial hemorrhage.

12. What are the cord problems seen in post-term pregnancy?

- Cord compression due to oligohydramnios
- Wharton's jelly in cord decreases vulnerable to compression
- Percentage of fetal Hb decreases so oxygen saturation in umbilical vein decreases (from normal 60 to < 30%)
- Hypoxia or vagal stimulation by cord compression leads to evacuation of Meconium from large bowl to the amniotic fluid
- Thick particulate type.

13. What are the causes of oligohydramnios in post-term pregnancy?

- Oligohydramnios is due to less urine formation which may be

Placental insufficiency → Hypoxia
Redistribution of blood
To vital organs like
Brain/heart ← Decreased ← Decreased
Renal blood flow
Urinary output

14. How will you asses fetal well being in post-term pregnancy?

A. *Clinical*:
   1. Fundal height
   2. Abdominal girth

3. Weight of patient
4. Clinical assessment of liquor
5. Daily fetal movement count (DFMC) chart.

B. *Biophysical*:

i. Non stress test NST: Acceleration of fetal heart rate with fetal movement in normal.

ii. Biophysical profile: Introduced by manning in 1980. It is done on USG

- Modified BPP (Clark 1989): here NST (vibroaccoustic stimulation test – VAST) + amniotic fluid index (AFI) are done
- Doppler wave form studies: Absence or reversal of blood flow during diastole in umbilical vessels indicate fetal jeopardy.

15. How often you will do the fetal well-being test?

- DFMC chart
- NST twice (preferably thrice) a week
- AFI by USG twice or thrice a week
- Biophysical profile twice weekly.

16. How will you manage postterm?

Wait and watch if, there are no maternal or fetal risk factors, till 41 weeks of gestation. Think about induction of labour after 41 weeks of gestation.

17. What is the labour management of post-term pregnancy?

Induction of labour is done if expected weight of fetus is less than 4000 grams.

i. Cervix is ripe (score > 5)—induction is done by oxytocin drip and ARM done as soon as feasible.

ii. Cervix is unripe (score < 5)—cervical ripening prostaglandin (PGE_2 gel) and then oxytocin drip and ARM.

18. What is the intrapartum management of post-term pregnancy?

Left lateral position.

- Oxygen
- ARM as soon as possible (meconium detection, scalp pH testing)
- Continuous electronic fetal CTG
- If Shoulder dystocia occurs maneuvers may be required, i.e. exaggerated lithotomy position with suprapubic pressure, McRoberts maneuver, delivery of posterior shoulder, wood's cork screw maneuver, Rubin's maneuver, etc.
- Cesarean section is carried out for 1) fetal macrosomia 2) fetal distress.

19. What do you mean by shoulder dystocia?

In vertex presentation when there is difficulty in delivery of the shoulders, due to macrosomia should dystocia is common in:

D Diabetes mellitus in pregnancy
O Obesity
P Postdatism
E Excessive weight gain of mother
A Anencephaly.

13

Previous Cesarean Section

Kanan Yelikar

History: Special points pertaining to LSCS:
- Indication of LSCS (documented proof in the form of previous hospital records)
- Gestational age at the time of cesarean section
- Elective/emergency.

Indication of previous LSCS:
- Contracted pelvis
- Cephalopelvic disproportion
- Malpresentations (breech/transverse lie /POP)
- Maternal diseases PIH/diabetes
- IUGR/PPROM
- Macrosomia.

Emergency:
- Failure to progress
- Placenta praevia (weak scar)
- Prolonged labor/obstructed labor (chances of sepsis are more).

Intraoperative history:
- Technical skill of surgeon
- Duration of Surgery
- Type of anesthesia
- H/o. blood transfusion
- Any complication during CS
- PPH
- Perinatal outcome.

Weight of the baby/cry/NICU admission/breastfeeding.

Postoperative:
- Febrile illness/morbidity
- Hospital stay/removal stitches
- H/o wound infection.

Interval between present and past pregnancy. Previous vaginal delivery making the scar weak:

- Menstrual history
- Obstetric history
- General examination/systemic examination
- P/V examination.

Diagnosis: Second gravida with previous LSCS at term (high risk pregnancy).

1. What was the indication of previous LSCS in your case?

Q. How will you define LSCS?

This is defined as abdominal route of delivery by giving an incision over the abdomen and uterus.

2. What are the types of cesarean section?

Cesarean sections are of two types, depending on the type of uterine incision:

a. *Classical cesarean section:* Incision is vertical and on the upper segment.
b. *Lower segment:* Incision is transcurvilinear on the lower uterine segment.

Q. Define LUS and UUS.

LUS is defined as that portion ,which develops from isthmus (portion between anatomical internal os and histological internal os). It is thin during labor. Passive and the peritoneum is loosely attached. All incisions are taken on this segment of the uterus.

3. Advantages of lower segment.

- Apposition is good
- Lower segment is inert during puerperium
- Scar is good. Rupture may occur only in labor in 1 to 2%.

4. Disadvantages of Upper segment:

- Healing of scar is poor
- Rupture occurs 5 to 10 times more (can occur during pregnancy also).

5. How do you classify CS depending on the time of CS?

- Elective CS done after 38 weeks of gestation
- Patient not in labor
- It is possible only in a booked patient, who attends the ANC regularly
- Emergency CS when patient is already in labor.

6. Advantages/disadvantages of elective CS

- Patient is well prepared, surgically, e.g. NBM psychologically—she is prepared.
- Relatives are available
- Senior surgeons/blood bank/anesthetists are available
- Patient is fit for anesthesia.

Disadvantages: LUS may not be well formed.

- If dates are not sure, possibility of prematurely cannot be ruled out (with USG this is becoming less)

7. What are the management options for previous LSCS?

Patient can be kept for VBAC/repeat LSCS.

Vaginal birth after cesarean section (VBAC) once a cesarean always a cesarean (Craigins 1916) is often misinterpreted and hence many practitioners revert away from VBAC (Last 2 decades VBAC is gaining more popularity since different studies, have proved that 60 to 80% of previous LSCS for nonrecurrent indication can deliver vaginally). A patient can be selected for VBAC after thorough counseling of the patient and her husband.

- Facility to carry out immediate LSCS must be present (within 10-15 minutes), i.e. fully equipped hospital set up
- Pelvis should be adequate. Rule out CPD
- Cervix should be favorable/single fetus/longitudinal lie/vertex presentation
- Placenta should be fundal
- Strength of the scar as assessed by the history and local examination.

Contraindications for VBAC/indications for repeat LSCS (ERCD) elective repeat cesarean delivery:

1. Previous 2 LSCS
2. Previous classical CS/hysterotomy/myomectomy
3. Previous CS with Breech/placental previa/macrosomia/associated medical factors
4. H/o previous perinatal death/long standing infertility/elderly patient.

8. What is the place of VBAC in modern obstetrics?

Vaginal birth after cesarean section (VBAC) once a cesarean always a cesarean (Craigins 1916) is often misinterpreted and hence many practitioners revert away from VBAC (Last 2 decades VBAC is gaining more popularity since different studies, have proved that 60 to 80% of previous LSCS for nonrecurrent indication can deliver vaginally.

9. What is trial of labor and trial of scar?

All VBAC are trial of scar. Trial of labor is a terminology coined to define mode of delivery in a non-scarred uterus, where possibility of labor ending in a CS is kept in mind, e.g. primi with minor degree of CPD.

10. How will you conduct VBAC?

- All criterias for VBAC to be fulfilled
- Careful monitoring of labor
- Use of partogram to detect any abnormal labor pattern
- Slightest doubt about scar dehiscence—prepare the patient for CS.

11. What are the symptoms/signs for scar dehiscence?

Symptoms—suprapubic pain:

- Vaginal bleeding
- Bladder tenesmus.

Signs
- Maternal tachycardia/hypotension
- Variable fetal heart rate pattern
- Tenderness over the uterine scare (palpated in a transverse direction)
- Failure to progress.

12. What is scar dehiscence/rupture of uterus?

Scar dehiscence means whenever the scar tissue either partially or completely gives way. It is also called as rupture of the scar during labor. It could be complete when amniotic cavity and the peritoneal cavity communicate with each other, i.e. all the layers of uterine wall tear off.

Incomplete scar rupture means the scar thickness partially tears off (both cavities do not communicate). Rupture of uterus is usually kept reserved for rupture during spontaneous labor in case of unscarred uterus.

13. How do you asses scar integrity?

Factors in history/imaging techniques.

a. History:
   - Technical skill of surgeon
   - Previous indications for CS—Placenta previa makes a scar healing week. Obstructed labor associated with sepsis and hence weak scar
   - Lateral extensions in incisions
   - Short interval between previous and present pregnancy
   - Previous VBAC makes a scar weak
   - Present pregnancy factors like twins/hydramnios/placenta previa (anterior).

b. Imaging:
   - Historiography 6 months with after CS wedge depression
   - USG.

14. Indications to terminate VBAC
- Fetal distress
- Scar dehiscence
- Unforeseen CPD.

15. Technique of LSCS:

Skin incisions – Vertical — Midline
Paramedian

- Transverse—Pfannensteil.
- Misguav Ladach—no sharp dissection/only Stretching of tissue with figures)
- Uterine incisions — Upper segment
  — Lower segment — Transcurvilinear
  Inverted T
  J shaped U shaped

16. Indications of classical CS/upper segment CS.
- Lower uterine segment is not accessible either due to adhesions or extreme vascularity as in major degree placenta previa.

- When urgent delivery of the baby is must as in case of deteriorating maternal condition.

17. Difficult situations in delivering the baby.

- Floating head: Which can be delivered with the help of forceps application
- Head is deep in pelvis as in obstructed labor. It can be delivered either by pushing the head from below or patwardhans method (where the shoulders are delivered first, followed by trunk—breech and then head)
- In major degree placenta praevia—either go by the side of placenta or go through the placenta.
- Dry labor where liquor is all drained out
- Transverse lie/hand prolapse.

18. Should you remove placenta manually during CS in all cases?

Temptation to remove placenta manually should be avoided. Since, it leads to more PPH and sometimes incomplete removal.

19. Role of exteriorization of uterus?

I.e. brining out the uterus throw the abdominal incision for better visualization and proper suturing, is advantages under special situations. For example, uncontrolled PPH, where extension of incision is suspected, bleeder cannot be located. However few authors advocated routine exteriorization of uterus in all cesarean sections.

20. Exploration of scar after VBAC.

Routine palpation/exploration of the scar is not required after every VBAC. It is indicated after:

- Instrumental delivery
- PPH
- Unexplained shock.

21. Decision-induction—incision-delivery interval.

Recent advances/update changing trends. Due to advent of safety issues in LSCS, the spectrum of indications are widening. The main reasons are safe anesthesia, antimicrobials, blood transfusion, improved fetal monitoring equipments, neonatal ICU facilities incidence is swinging between 10 to 40%. Newer indications for CS.

Previous CS with Breech, macrosomia, severe PIH with unripe cervix, multiple gestation, failure to progress, fetal distress. Last 2 decades, the professionals are trying to reduce the incidence of primary cesarean section. Think 10 times before a patient. For primary cesarean section. Do not think for 10 secs to prepare a patient of previous LSCS.

14

Rh Negative Mother

Kanan Yelikar, Sonali Deshpande

1. What is the incidence of Rh -ve women in India?

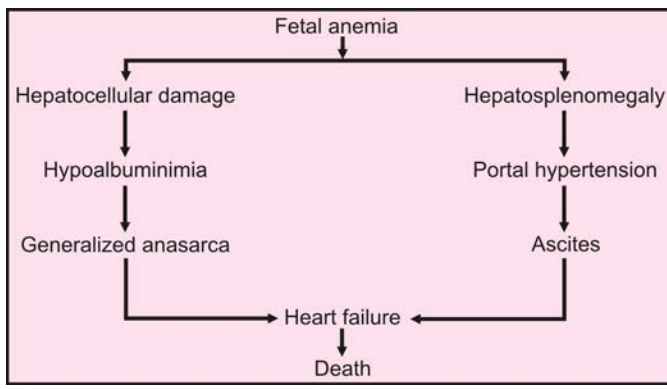
The incidence of Rh -ve women ranges from 5-10%.

2. What do you mean by isoimmunization?

Isoimmunization is defined as a production of immune antibodies in an individual in response to an antigen derived from another individual of the same species provided the first one lacks the antigen.

3. How is the pathogenesis of Rh disease in the fetus?

- Transplacental hemorrhage occurs in 75% of pregnancies which is followed by primary immune response
- Primary immune response is slow (several weeks), weak and IgM antibodies which do not cross the placenta
- A secondary exposure to Rh +ve cells leads to a secondary immune response
- Secondary immune response is rapid (1 day), strong and predominantly IgG antibodies which cross the placenta
- After secondary immune response, IgG antibodies which are formed cross the placental barrier and coats the fetal RBCs causing extravascular hemolysis. To overcome this situation, extramedullary erythropoiesis occurs in liver and spleen and nucleated RBC precursors (erythroblasts) are released in peripheral circulation.



4. Why erythroblastosis fetalis term is used?

As a result of fetal anaemia, extramedullary hemopoiesis occurs in the liver and spleen and nucleated RBC precursors (erythroblasts) are released into the peripheral circulation hence the name.

5. What are the manifestations of hemolytic disease of newborn?

1. Congenital anaemia of the newborn
2. Icterus gravis neonatorum
3. Hydrops fetalis.

6. What are the characteristic features of hydrops fetalis?

Hydrops fetalis is characterized by ascites, pericardial and pleural effusion, skin edema, cardiac failure leading to fetal loss.

7. What is the incidence and extent of Rh isoimmunization?

Risk of immunization increases with the amount of fetomaternal hemorrhage.

	RBCs	Risk of Rh isoimmunization
Ist trimester	0.03 ml	3%
3rd trimester	0.25 ml	45%

8. How will you quantify FMH?

FMH will be quantified either by technic of immunofluorescence flow cytometry (IFC) compared with traditional **Betke** Kleihauer testing.

IFC detects approximately twice the no of fetal RBCS as compared to Kleihauer.

9. What is Kleihauer count?

Approximate volume of fetal blood entering into the maternal circulation is estimated by kleihauer count using acid elution technic to note the number of fetal RBCS per 50 LPF.

If there are 80 fetal erythrocytes in 50 LPF in maternal peripheral blood films, it represents a FMH to the extent of 4 ml of fetal blood.

10. What are the indications for anti-D prophylaxis?

- Antepartum at 28 weeks
- After spontaneous/induced abortion/ectopic pregnancy
- Postpartum if fetus is Rh +ve
- After CVS, amnio and cordocentesis
- After bleeding episodes during pregnancy.

11. What is the rational for giving antepartum prophylaxis?

A. 15% of Rh sensitizations are caused by small FMH that occurs between 20-40 weeks. Before 20 weeks the chances of FMH are very less.

12. What is the recommended dose of anti-D?

Recommended dose	Recommended dose
1st trimester abortion	50 m gm
1st trimester ectopic	50 m gm
2nd trimester abortion	300 m gm
2nd trimester ectopic	300 m gm

Prophylaxis at/ about 28 weeks	300 m gm
3rd trimester amnio centers	300 m gm
FMH (as estimated by different test)	Depending on quantum of hemorrhage.

13. What is Rh immunoglobulin?

It is a biological product derived from human placenta. It is efficacious, safe, stable, free from other blood group antigens or antibodies and free from HIV, Hepatitis B, C and other viral diseases like mumps and measles.

14. Choice of prophylaxis monoclonal/polyclonal?

a. Polyclonal
   - Proven technology
   - Covers all 30 epitopes of Rh antigen
   - Approved by US FDA.
   - Dependent on plasma donor.

b. Monoclonal
   - Unproven technology
   - Covers only 1 epitope of Rh antigen
   - Not approved by US FDA
   - Independent plasma donor.

15. How to minimize FMH?

1. Precaution during LSCS:
   - Prevent blood spillage in the peritoneal cavity
   - Manual removal of placenta should be avoided.
2. Avoid administration of ergometrine for active m/m of III stage of labor
3. Amniocentesis should be done USG guided so as to avoid injury to the placenta
4. Early clamping of the cord
5. Forcible attempt to perform ECV under anaesthesia should be avoided
6. MRP should be done gently
7. To refrain from abdominal palpation as far as possible in abruptio placentae
8. Avoid Rh +ve blood to Rh-ve female
9. Prevent active immunization by giving anti D. Don't allow pregnancy to overrun the dates.

16. Up to how many days, anti-D can be administered?

Up to 15 days of delivery. Ideal is within 72 hrs.

16. What is the critical value of serum bilirubin to develop kernicterus?

The neonate with severe hyperbilirubinemia is at risk of developing kernicterus if bilirubin levels are in excess of 25 mgm /dl at term or even 12 mg/dl in preterm neonates.

17. What are the indications of intrauterine blood transfusion?

Indicators of fetal anemia:

- Edema
- Hydrops

- Fetal hematocrit < 30% in nonhydropic fetus.
  Increased MCA peak systolic velocity Doppler.

18. How is Intrauterine transfusion given?

Intraperitoneal Transplacental
Intravascular Transperitoneal
Intracardiac

Amount of blood to be transfused is calculated by Bowman's formula:
(Weeks of gestation – 20) × 10 ml.
Packed O Rh -ve blood is used.

19. What are the complications of intrauterine therapy?

- Cord constriction
- Umbilical cord hematoma
- Fetal bradycardia
- Fetal exsanguinations
- Preterm labor
- Increase in maternal antibody level.

20. What are the investigations to be sent in cord blood?

- 5 ml of blood should be taken
- Plain bulb—blood group Rh typing, direct Coombs test , serum bilirubin
- Oxalated bulb—Hb%, blood smear for the presence of immature RBCS.

21. How will you manage the affected newborn?

- Phototherapy—degrades the bilirubin by photooxidation and excreted out. Fibre-optic delivery system
- Phenobarbitone—increase the activity of glucoronyl transferase. Dose 2 mgl kg thrice a day
- Intravenous immunoglobulin
- Hemoxygenase inhibitor
- Double volume exchange transfusion
- Erythropoietin.

22. What are the indication of exchange transfusion in a newborn?

- Cord blood Hb < 15 %
- Previous definite H/o an affected baby due to homolytic disease
- Birth Weight < 2.5 kg
- Rapidly developing jaundice with unconjugated bilirubin rises more than 5 mg%.

23. What are the different antibodies are formed as a result of Rh isoimmunization?

Two types of antibodies are formed:

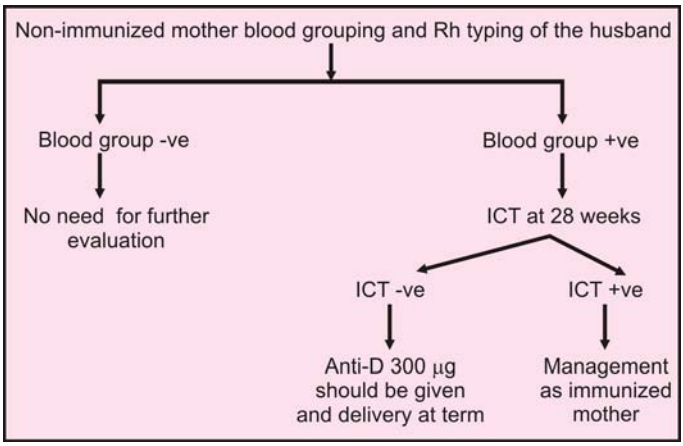
a. *Saline antibodies (IgM):* This is in the maternal circulation and these are larger molecules they can not pass through placenta and not harmful to foetus.
b. *Albumin antibodies (IgG):* This type of antibody appears in the maternal circulation as a result of secondary immune response. These are small molecules can pass through the placenta and cause damage to the fetus.

24. When does the icterus becomes evident? Why?

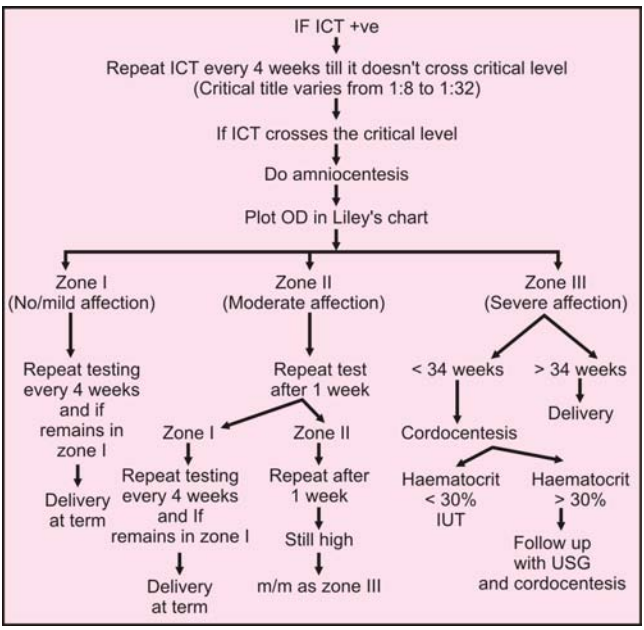
In Rh sensitized baby within 24 hrs hemolysis starts in utero.

25. Management of Rh -ve mother

A. Non immunized mother blood grouping and Rh typing of the husband.



B. Immunized Mother



- USG should be done frently depending on the severity of the cause to assess foetal health.
- Doppler velocimetry—increase MCA peak systolic velocity.
- NST is done from 28 weeks onwards. Sinusoidal and deceleration patterns correlate with severe foetal anemia.

15

Preterm Labor

Jyoti Bindal

1. What is preterm labor?

Frent uterine contraction with or without pain with progressive cervical dilatation and effacement from 20 weeks (developed countries) and 28 weeks (developing countries like India) to 37 weeks of gestation, (onset of labor with intact membrane before 37 weeks) (Labor and delivery before 20 weeks of gestation is called as miscarriage.)

2. What causes preterm labor?

I. General maternal problems/behavioral problem.
   1. Smoking, alcoholism, cocaine abuse
   2. Malnutrition
      i. Stress
      ii. Coitus.

II. Pregnancy complications
   1. PIH, thyrotoxicosis and SLE syndrome
   2. Hydramnios
   3. Multiple pregnancy
   4. APH
   5. Previous history of abortion and preterm birth
   6. Chronic diseases (anemia, liver disease, renal disease, cardiac).

III. Uterine causes
   1. Bicornuate/unicornuate/septate uterus
   2. Submucous fibroid
   3. Cervical incompetence
   4. Uterine hypersensitivity.

IV. Infection
   1. PROM
   2. Chorioamnionitis
   3. Asymptomatic bacteriuria
   4. Bacterial vaginosis
   5. Intrauterine infections (Chlamydia, group B Streptococci).

V. Fetal causes: Chromosomal and structural anomalies.

VI. Iatrogenic
   1. Induction of labor

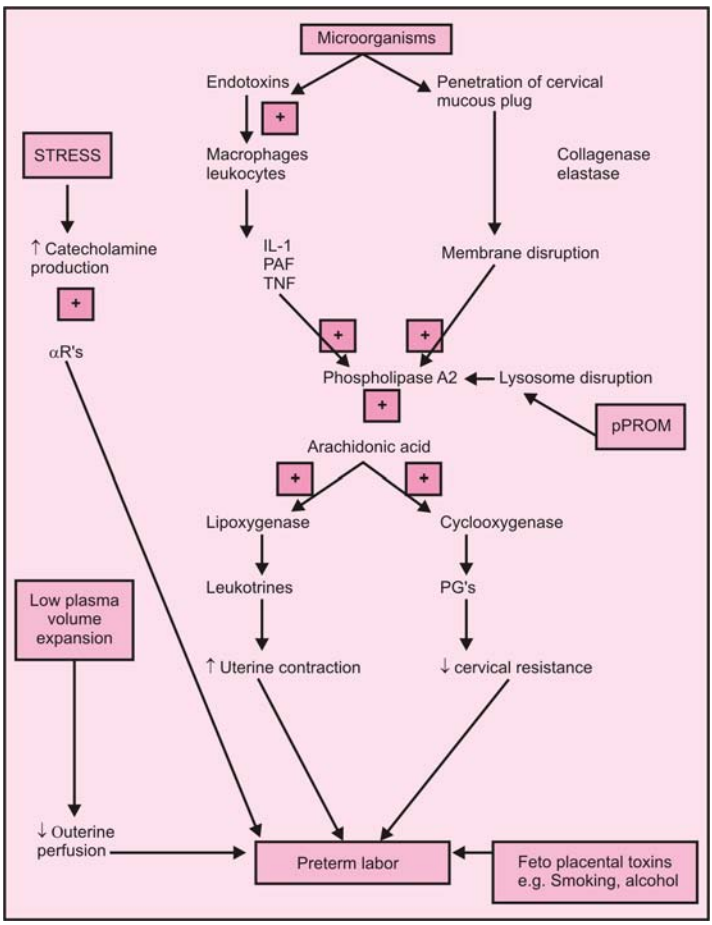
Etiology
1/3rd **are Idiopathic**
1/3rd are associated with PROM
1/3rd associated with complications
- Uterine anomaly
- Infection
- Iatrogenic

2. External version
3. Amniocentesis
4. Cordocentesis.

3. Risk factors for preterm labor?

- Poor socioeconomic status
- Maternal smoking
- Low pre-pregnant weight
- Maternal age < 18 years or < 40 years
- Prolonged standing
- Longer hours of work
- Coitus after 30 weeks
- Lack of weight gain or weight loss during pregnancy
- Low birth weight of previous infant.

4. Pathophysiology of preterm labor?



5. Warning signs of preterm labor?

- Menstrual like cramps
- Low, dull backache
- Pressure (feels like baby is pushing down)
- Increase or change in vaginal discharge
- Fluid leak from vagina
- Uterine contraction with or without pain, 10 minutes apart or closer.

6. Management of patient at risk of preterm labor?

- Regular antenatal visits
- Education about preterm labor
- Aggressive treatment of cervical and vaginal discharge
- Serial pelvic examination
- Serial USG examination
- Coital abstinence
- Limitation of physical activity
- Change in working condition
- For patients with history of second trimester loss or spontaneous preterm birth, close monitoring during ANC.

7. Predictors of Preterm labor?

1. Risk markers: As described earlier.
2. HUAM (home uterine activity monitoring): Involves telemetric recording of uterine contraction and its transmission to a monitoring center and daily feed back from the health care practitioner to offer patients support and advice. Not recommended now a days.
3. Biochemical markers
   a. Salivary oestriol:
      > 18 ng/ml before 34 weeks indicate preterm labor
      Poor sensitivity and specificity
   b. Serum collagenase
      - Increases in preterm labor
      - Detected by ELISA.
   c. Relaxin hormone: Increases in preterm labor.
   d. Fetal fibronectin
      - Most important predictor of preterm labor
      - Its presence in cervicovaginal secretion > 50 ng/ml during 20-37 weeks indicated preterm labor
4. USG

Can reveal	Fetal malformation	Risk factors for
	Hydramnios, multiple pregnancy	preterm labor
	Fetal age	
	BPP, breathing pattern	
	Cervical length and extent of dilatation	

   - Funneling of cervix with internal os dilatation > 20 mm and shortening of cervix (cervical length < 25 mm) between 24-28 weeks may increase the relative risk of preterm labor

- Normal fetal breathing stops 24- 48 hrs before the onset of labor, this finding is specific in presence of intact membrane and in absence of APH.
- USG: 65-70% predictive for preterm labor.

5. Screening for lower genital tract infection
   Including bacterial vaginosis

8. What is bacterial vaginosis?

Normal lactobacillus predominant vaginal flora, is replaced by anaerobic bacteria, i.e. gardnerella vaginalis and mycoplasma hominis.

9. How do you diagnose bacterial vaginosis?

- Vaginal pH > 4.5
- Amine (fishy) odor with KOH
- Presence of clue cells (vaginal epithelial cells glued with dead bacilli)
- Few WBC with mixed flora

10. Investigations/laboratory studies in preterm labor?

a. Complete hemogram—including CBC, differential count
b. Urine analysis and culture
c. Serum C reactive protein (> 0.8 = chances of preterm labor)
d. Electrolytes and serum glucose: In cases requiring B_2 agonist tocolytics
d. USG examination
e. Amniocentesis
   - For fetal lung maturity
   - Bacteriological study

f. Cervicovaginal fluid—microscopic with gram staining
   - Culture and sensitivity
   - Fetal fibronectin levels.

11. Role of amniocentesis to detect infection?

Infection indicated by

- Increase leucocyte count
- Decrease glucose
- Increase IL-6 (Most sensitive test for detection of amniotic fluid containing bacteria)
- Positive gram staining
- Positive culture.

Q.12. Diagnosis of preterm labor?

- More subjective than objective
- Require serial examination (preferably by same examiner)

Criteria (proposed by AICOG in 1997)

- 4 Contraction in 2 min or 8 in 60 min. plus progressive change in the cervix.
- Cervical dilatation > 1 cm.
- Cervical effacement > 80%.

Q.13. What is the management of preterm labor?

It varies according to the condition of the patient:

Group A: Patient who are at risk
Management as described earlier

Group B: Patient with warning signs of threatened preterm labor

- Limitation of physical activity
- Referral to tertiary care centre
- Antibiotics—Ampicillin/ampicillin + Sulbactam (commonly used)
- Tocolytics—Beta adrenergics, calcium channel blockers, magnesium salts, etc.

Group C: Management of patient with established preterm labor

a. Patients who need to be delivered immediately
   - Maternal diseases: PIH, hypertension, chronic renal disease, cardiac disease, SLE, acute or chronic infection.
   - Advanced labor—5 cm dilatation or > 80% effacement with membrane bulging.
   - PPROM
   - Congenital and chromosomal anomalies in fetus
   - IUGR
   - Chorioamnionitis
   - If adequate fetal lung maturity is present.

b. Patients who do not require immediate delivery
   Drugs given
   - *Tocolytics*: After ruling out their contraindications
     IV route effective than oral
   - *Antibiotics*: It is only helpful when preterm is associated with chorioamniotis or PPROM or any condition predisposing to maternal infection, e.g. immunosuppressive drugs.
   - *Glucocorticoids*
     - Rule out contraindications (e.g. diabetes mellitus, as steroids aggravate the hyperglycemia)
     - Used where maturity is within 34 weeks with immature fetal lung and there is definite interval of > 24 hrs between delivery and drug administration with maximum limit within 7 days of last dose.
       Agents used are: - Betamethasone—12 mg—24 hrs—apart
       - Dexamethasone—4 mg—6 hrly—24 hrs
   - *Phenobarbitone*:
     - Dose 10 mgm/kg maximum upto 500-700 mgm IV in 30 minutes
     - It may prevent intraventricular bleeding in newborn
     - Role is controversial.
   - Vit K_1:
     - Dose 10 mgm IM followed by 20 mgm orally or injection to be repeated every week before 32 weeks
     - It may prevent intraventricular bleeding in newborn
     - Role is controversial.

14. What steps should be taken during delivery of preterm infant?

Ist Stage of labor

- Epidural anesthesia: —alleviates labor pain
  —improves oxygenation
- Avoid strong sedatives—suppress the fetal respiratory center

IInd stage of labor

- Premature bearing down should be discouraged otherwise fetus may suffer ICH.
- Intrapartum fetal monitoring
  Avoid fetal scalp monitoring—may cause scalp hemorrhage
- Liberal episiotomy
  - To shorten the 2nd stage
  - To reduce the force compressing the soft fetal skull
- Outlet forceps superior to ventouse extraction—less likely to cause ICH.
- Elective caesarean delivery does not confer much benefit to the baby and should be done only after weighing the benefit to the neonate against the maternal morbidity of operative delivery.
- The possibility of a PPH should be borne in mind if the woman was administered tocolytic drugs before delivery and preventive measures should be taken.

15. What steps should be taken for the neonate immediately after birth?

- Cut and clamp umbilical cord immediately
- To prevent overloading of blood circulation
- For immediate resuscitation by pediatrician
- Established breathing fast and avoid prolonged anoxia, but remember that prolonged oxygen therapy is also harmful and can lead to broncho-pulmonary dysplasia and retrolental fibroplasia
- Avoid infection by proper antibiotics
- Vitamin K and phenobarbitone—not recommended now-a-days
- Intratracheal surfactant if available
- Maintain euthermia
- IV nutrition preferred to oral feed for first few days. Breast-feeding is started once the suckling reflex is established.

16. What are the consences of preterm labor?

Prematurity is the major consence, resulting in risk of neonatal morbidity and mortality. This mostly occurs due to:

- Birth asphysia
- Respiratory distress syndrome
- Jaundice
- Hemorrhage
- Hypocalcemia

- Hypoglycemia
- Sepsis
- Feeding difficulties
- Necrotizing entrocolitis
- PDA
- IVH
- Sensorineural deafness
- Developmental delay
- Iatrogenic complications, e.g. retrolental fibroplasia
- Reduced growth potential
- Recurrent respiratory infection
- Learning difficulties
- Social enforced separation of mother and baby
- SIDS (sudden Infant death syndrome)
- Blindness.

17. What are tocolytic drugs? Why they are used in preterm labor?

Drugs that inhibit uterine contraction are called as tocolytic drugs.

Tocolytic are useful in preterm labor for

1. Adding few days to fetal maturity. To provide time for completing corticosteroid therapy and utero transfer.
2. Pulmonary maturity will reduce the risk of RDS in the newborn.

18. What are the contraindications of tocolytic therapy?

The conditions are the following:

1. Severe hemorrhage
2. Abruptio placentae
3. Pregnancy induced hypertension
4. Intra uterine fetal death
5. Chorioamnionitis
6. Pulmonary hypertension
7. Known intolerance to tocolytics
8. Fetal maturity (after 34 weeks of pregnancy)
9. Lethal fetal anomaly
10. Severe intrauterine growth retardation
11. Uncontrolled insulin dependent diabetes
12. Asthmatic patient (tachyphylaxis)
13. Patient on MAO inhibitor.

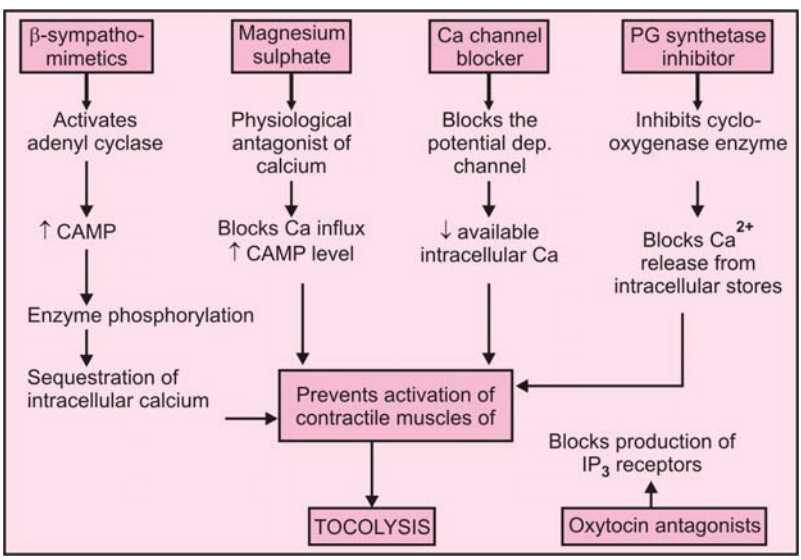
19. What are the different tocolytic agents and how are they used?

	Drugs	*Class of drugs*	*Dose IV*	*Dose oral*
1	Isoxsuprine (Duvadilan)	b mimetic	30-60 mg in 500 ml dextrose (.1 mg/min-1 mg/min)	5-10 mg
2	Ritodrine	b mimetic	150 mg in 500 ml (4 mg/hr-12mg/hr)	10 mg 2 hrly (1st dose 30 min before stopping I/V drip) to maximum 120 mg in 24 hrs
3	Terbutaline	b mimetic	5 mg/min Maintenance dose 2-5 mg/min max. 30 mg	5 mg 6-8 hrly subcutaneous 25 3-4 hrly
4	Salbutamol	b mimetic	20-50 mg/min maintenance dose 2-5 mg/min	—
5	Indomethacin	Anti-prostaglandin	50 mg initially reduced to 25 mg 4 hrly × 24 hrs	Same as I/V dose Loading 50-100 mg followed by 24
6	Sulindac	Prostaglandin synthetase inhibitor	—	200 mg 12 hrly for 48 hrs
7	Magnesium salt		$MgSO_4$—4 g in 10% dextrose over 20 min followed by 2% maintenance infusion at 2 g/hr	Oral salts— Magnesium gluconate 1 gm BD Magnesium oxide— 200 mg TDS Magnesium chloride— 535 mg QID
8	Nifedipine	Calcium channel blockers		30 mg followed by 20 mg TDS Sublingual—10 mg every 15-20 min in the 1st hr thereafter 60-160 mg/day.
9	Atosiban or Antocin or Tractocile	Oxytocin antagonist	7.5 mg bolus then 7.5 mg in doses of 300 mg/min for 3 hrs (18 mg/hr) then 100 mg/min upto 48 hrs (6 mg/hr) Total dose should not exceed 330 mg over 48 hrs	—
10	Nitroglycerine	NO donors	Used as transdermal patch—10 mg patch approximately to lateral aspect of thigh and is repeated after 2 hrs if contraction do not stop. Not more than 2 patches are used in 24 hrs. Can be kept for 48 hrs)	
11.	L-Arginine	NO donor	Under trial	
12.	Na nitroprusside	NO donor		

19. What are the adverse effects of commonly used tocolytic agents?

	Drugs	*Maternal effect*	*Fetal effect*
1.	b mimetic	Tachycardia, hypotention, hyperglycemia, hyperinsulinemia, hypokalaemia, hypocalcemia, hyperlipidemia, tremor, cardiacarrthymia, myocardial infarction pulmonary oedema and fluid retention headache, chest pain, liver damage Contraindication in cardiac disease diabetes, hyperthyroidism, pregnancy induced hypertension, intrauterine growth retardation, fetal death, antepartum hemorrhage	Fetal hypoglycemia hypocalcemia hypotension, ileus.
2.	Indomethacin	Hepatitis, renal failure, gastrointestinal bleeding	Premature closure of ductus arteriosus and pulmonary hypertension, intracranial hemorrhage, necrotizing enterocolitis, renal dysfunction
3.	Nifedipine	Hypotension, tachycardia, headache, dizziness, nausea	Acidosis and fetal asphyxia, jaundice
4.	Magnesium sulphate	Hypotension, MS paralysis, drowsiness, cardiac arrest, pulmonary oedema, respiratory depression, tetany	Fetal hyperglycemia, neonatal drowsiness, lethargy, respiratory depression
5.	Atosiban	Nausea, vomiting, headache	

20. What is the mode of action of various tocolytics?



21. What is the role of corticosteroids in preterm labor?

Antenatal corticosteroid therapy reduces the risk of respiratory distress syndrome (by 50%), necrotizing enterocolitis and intraventricular hemorrhage in the preterm newborn.

- Effective between 30-34 weeks of gestation
  (< 30 weeks - type II pneumocytes scanty—drug not effective)
  (> 34 weeks - cell produce adequate surfactant—no use of drug)
- Betamethasone 12 mg 24 hrly × 2 doses
  Dexamethasone 4 mg 6 hrly × 4 doses
- These drugs have negligible immunosuppressive effect and are safe even in presence of infection, pregnancy induced hypertension and tuberculosis also in diabetes with caution.
- The drug effect appears 24 hrs after the infusion and persists for 7 days.
- Multiple weekly doses of corticosteroids is not recommended now-a-days.
- Betamethasone is better than dexamethasone.
- Dexamethasone cause intraventricular and periventricular leukomalacia leading to cerebral palsy.

22. Ineffective treatment for preterm labor?

1. Bed Rest (expectant management)
   - Strict bed rest for > 3 days increases risk of developing blood clots in the legs or lungs (1 in 1000 to 16 in 1000)
   - There is no evidence that long term bed rest lower the risk of preterm labor.
2. Cervical encirclage
   Cervical encirclage is the surgical closure of an incompetent cervix.
   - A recent study has shown that cerclage used to close a shortened cervix (identified using TVS) is ineffective for preventing preterm labor and delivery.
   - It also carries a risk of infection and pregnancy loss. It is also associated with increased medical intervention and doubling of the risk of puerperal pyrexia.
   - So it should not be done in cases other than proved cervical incompetence.
3. Home uterine activity monitoring
   - Research have shown that HUAM is expensive and has no proven effect on delaying early birth.

Q.23. Some important statistical data's?

Impact of preterm labor

Incidence in developed countries	-	6-7 %
Incidence in India	-	10%

Responsible for 75% of all neonatal mortality
Responsible for 50% of long term neurological impairment in children
Relative risk of preterm labor

Low socioeconomic status	-	2 times
Black race	-	2 times

Recent cocaine use	-	4 times
Prolonged periods of standing during pregnancy	-	1.3 times
Weight gain < 0.27 kg/week during pregnancy	-	2.54 times
Patient with asymptomatic bacteriuria	-	2 times
Group B streptococci in vaginal culture	-	18.7%

Increase incidence of preterm labor is found among women delivering male infants (odds ratio 1:2)

Low magnesium and high relaxin level are also associated with increased incidence of preterm labor.

Risk in subsent pregnancy

One preterm birth = 14.3%
Two preterm birth = 28%

- Bacterial vaginosis has been suggested as the causative agent in upto 50% cases of idiopathic preterm labor.

Relation between fetal weight and survival

1000-1500 gm > 90% survival
< 1000 gm—survival decreases 10-15% for each decrement of 100 gm
< 500 gm—survival is rare.

According to gestational age survival rate

72% at 28 weeks
While it is 50-60% at 26 weeks
and increases 0.9-3% per day from 25-30 weeks
50% neonatal death occurs in 24 hrs and another 15% in 48 hrs of birth.

24. Recent predictor of preterm labor?

Fetal fibronectin

- Glycoprotein present in high concentration in maternal blood and in amniotic fluid.
- Play role in intercellular adhesion during implantation and in maintenance of placental adhesion to decidua.
- Detectable in cervix vaginal secretion until 16-20 weeks of gestation thereafter disappears until near term unless premature rupture of membrane occurs.
- > 50 ng/ml during 20-37 weeks indicates preterm labor.
- Positive test at 24 weeks—60 fold increase in risk of preterm labor within 4 weeks of sampling
- 20 times risk of clinical chorioamnionitis
- Negative predictive value—90-95%.

16 HIV Infection during Pregnancy

Deepti Dongaonkar

It is essential for every MBBS graduate to know about diagnosis and principles of managing HIV infection. HIV infection during pregnancy needs special attention.

History:

When was first HIV case reported in the world?
When was the first case reported in India and which city?
When was the word HIV coined?
When and where the organism for HIV was discovered?
What is window period?
What is the natural history of HIV? (Stages of the disease)
Universal precautions to be taken by care providers
Post exposure prophylaxis and drug given for the same
HIV controlling organization at center and state level

Types of HIV viruses

HIV type 1—serology A,B,C,D…….L,M,N
HIV type II
(HIV I type E is common in India)

Prevalence and incidence (NACO 2004-2005)

Cumulative—Total 5.3 million in India (Dec 2005) affected
Male to female ratio: 60:40
New infection per year—16000 per year (20,000 in 2005)
General population less than 1%
Pregnant women in South India (including Maharashtra) more than 1%
Commercial sex worker—40-60%
In STD clinics—20-30%

Modes of transmission:

Horizontal transmission:

Sexual—homosexual, heterosexual, bisexual
Intravenous drug abuse
Infected blood transfusion

Vertical transmission:
Mother to child during pregnancy and childbirth

Minor route of transmission:
Postmortem examination
Ear prick or tattooing needles
Wet kissing with oral ulcer in one partner
Intra muscular/subcutaneous injection
Washing and cleaning surgical instrument/linen
Washing/wiping blood splash with cuts on finger
Surgery procedure/open wound dressing
Blood collection/tooth bite.

Indication for HIV testing:
Screening during every pregnancy
Recurrent pelvic infection, vaginal ulcers or discharge
Chronic ill health, 10% weight loss in 6 months
Screening each tuberculosis patient
Person who received multiple blood transfusion
Suspected opportunistic infection
Second marriage following death of a partner in young age
Person with drug abuse/chronic alcoholism
Malignancy before 40 years of age (for prognosis)
Screening during pregnancy:
Under PPTCT (Prevention of parent to child transmission) program. All pregnant women, irrespective of gestational age are offered, voluntary testing. During ANC, a patient goes through:

- Pretest counseling, written consent, distribution of reports, post-test counseling
- Nevirapine prophylaxis.

Types of Tests for HIV diagnosis
Adult:
Antibodies detection tests —ELISA test, rapid test, immunocomb test
Antigen detection tests—western blot, immunoblot
Viral detection—bDNA PCR, RT PCR (RNA PCR), NASBA.

Neonates:
Up to 2 months—p24 antigen (western blot)
Up to 18 months—RT PCR, DNA PCR
After 18 months—ELISA based like adults.

HIV risk associated per exposure with high risk behavior (UNAIDS/NACO)

Blood transfusion	–	95%
Vertical transmission	–	25-40%
IV drug abuse	–	0.67%
Homosexual	–	0.04%
Heterosexual	–	0.1-0.3%
Needle injury to HCW	–	0.4%

(heterosexual is common mode of transmission in India due to frency of exposure).

Prevalent mode of HIV spread (differ in northern and Southern states) NACO

Sexual – 80-90% (in southern states),
Among women 80-90% are housewives (from promiscuous husband)
Blood transfusion – 2-5 %
IVDU - 15 - 20 % (in southern states)
- 70 - 80 % (n Manipur and Nagaland).

Determinant of Mother to child transmission of HIV

Prenatal factors	*Intrapartum factors*	*Postpartum factors*
Nutrition	ART	Breast vs formula feed
CD4	Duration of rupture of	Breast feed duration
CD8	membranes	Mixed feed
Viral Load	Vagina Douche*	Mastitis/nipple infection
S T I	Vaginal vs caesarian	ART to baby
ART	Duration of labor	Maternal ART with BF

*Savlon or 1% betadine is good for vaginal douches

Association of HIV and Tuberculosis

25-30 % of Tuberculosis patients may be HIV positive
60% of HIV patient may suffer from TB in lifetime
Extra-pulmonary TB is common among HIV infected person
TB is common cause of death among Indians with HIV (adult and children)
Presence of TB during pregnancy increases risk of transmission
Risk reactivation of old healed TB lesion is known
Occurrence of TB hastens the progression of the disease.

Antiretroviral drugs available

Nucleoside analog (NRTI)	*Non-nucleoside analog (NNRTI)*	*Protease inhibitor (PI)*	*Fusion inhibitors (FI) (under trial)*
Zidovudine	Nevirapine	Indinavir	
Lamivudine	Delavirdine	Ritonavir	
Didanosine	Efavirenz	Sauinavir	
Stavudine	Nelfinavir	Amprenavir	
Abacavir			
Zalcitabine			
Adefovir			
Tenofovir			

ART in red are commonly used in pregnancy

Indications for starting ART

1. Symptomatic HIV disease
2. CD4 cell count fewer than 350 copies/cmm

3. Viral load more than
   10,000 copies/ml by branched DNA PCR
   20,000 copies/ml by RNA PCR
4. Acute retroviral syndrome within 6 months of documented seroconversion
5. Prevention of mother to child transmission
6. Post exposure prophylaxis.

Contraindication for ART

Maternal factors	*Fetal factors*
Hb less than 8.0 gms%	Fetal demise
Thrombocytopenia (< 100,000/ cmm)	Fetal anomaly
Total WBC < 1000/cmm	Fetal hydrops or ascitis
Radiotherapy or chemotherapy	Fetal severe anemia
Anti HIV vaccine	Oligo or polyhydromnios
S creatinine more than 1.5 mg % (130umol /L)	
Creatinine clearance > 70 ml/min	

Preferred ART combinations

For PPTCT—single NRTI or 2 NRTIs or 2 NRTI + 1 NNRTI
PEP—2 NRTI or 2 NRT + 1 NNRTI or 2 NRTI + 1 PI
Other patient—2 NRTI + 1 NNRTI or 2NRTI + 1 PI
(Consult expert before stating therapy to avoid drug interaction and toxicity).

Monitoring patient on ART—Every three to four monthly

Specific	*Nonspecific*	*Extra if required*
CD4 count	Hb, CBC, total	Urine or stool culture
CD8 count	lymphocyte count	ART drug sensitivity
Viral load	LFT, RFT	X-ray chest for chronic cough
Specific fungi,	X-ray chest if known	USG for liver, kidney, pelvis
virus culture	Tuberculosis	Bone marrow aspiration, MRI

Side effects and toxicity known with long term use of ART

Hematological	*Metabolic*	*Systemic*	*Bones*
Anemia	Lipodystrophy	Hepatotoxicity	Avascular necrosis
Pancytopenia	Insulin resistance	Nephrotoxicity	of femur neck
Thrombocytopenia	Lactic academia	Polyneuropathy	Osteopenia
	Hypertriglyceridemia	Hypersensitivity	osteoporosis
		GIT disturbances	

Indication for discontinuation of ART

Toxicity related	*Disease progression related*
Hb fall below 8.0 gm%, pancytopenia	No fall in viral load with 3-4 month ART
Lactic academia	Development of malignancy
Hepatic dysfunction with HBV/HCV infec	Try change of ART combination
Drug intolerance/drug sensitivity	or do drug resistant test

Always change drug with known toxicity or stop all drugs at a time to avoid development of drug resistance. Consult expert.

FREQUENTLY ASKED QUESTIONS

1. Should HIV testing be mandatory or voluntary during pregnancy?
Voluntary. Mandatory testing may cause program failure in long-term. Voluntary testing with pre test counseling gives an opportunity for health education and for HIV awareness drive. It also encourage for behavioral change.

2. What is HIV transmission rate from Mother to child in India?
Without drug (ART) it 25-40% (36% author study in 1998)
With drug (ART) intervention 2-12% (differ with combination and duration)

3. When does MTCT occur?
It can occur any time during pregnancy.

It is more (15-20%) in late months of pregnancy and maximum (70%) during labor. Breast-feeding can risk 7-15% children fed for more than 5 months.

4. What care should be taken during pregnancy in HIV infected mother?
Route ANC visit patter followed. Avoid any maternal infection, which may reduce immunity. Promote health with good hygiene and nutrition. Screen for TB, genital infections. Prevent anemia, hypoproteinemia. Encourage hospital delivery and early arrival to hospital in labor. Encourage ART.

5. What care should be taken during labor in HIV infected women?
should arrive early in labor. Vaginal douche should be given to minimize neonatal infection. Avoid prolong labor and rupture of membranes (ROM). ROM (ARM) may be done in indicated cases. Avoid instrumental delivery and CS. Cesarean section should be done for obstetrics indications only. Continue ART. Avoid milking of card. Encourage early clamping.

Health care provider should observe universal precaution for self-protection.

6. What ART should be given?
Zidovudine, Lamivudine and Nevirapine are standard drugs used during pregnancy and labor. Selection of drugs (ART) depends on viral load, CD4 and CD8 count.

7. What are different ART regimen given in prenatal period for pregnancy with HIV?
A. ACTG 076 (NY 1992)—ZDV 100 mg five times a day, 14-38 weeks.
B. Thai (1995)—ZDV 300 mg twice a day from 36 to 40 weeks
C. PETRA regimen (s Africa, Tanzania 1998)
D. PACT 185 regimen (USA 1998).
(In India we follow Thai regimen/Uganda regimen/combination regimen depending on affordability and availability. Same ART needs to be given during labor).

8. How effective are ART in reducing MTCT?
It depends on ART chosen, duration of prophylaxis taken. It also depends on immunological parameter and viral load before starting ART. Nutrition plays important role too.

8. How effective is single dose Nevirapine given during labor?
Single dose Nevirapine 200 mg (Uganda 1998) is observed to provide 50% protection to new born at birth (HIV NET study 1998, Uganda). It works provided given 3 hour prior to birth. If given within one hour of birth, protection is not guaranteed.

9. What is the risk associated with caesarian section?
Caesarian section does not guaranty protection from HIV. Besides women may have delayed healing, would infection due to surgical intervention. Risk to HCW increases and MTCT reduction is variable.

10. How do we know newborn is infected with HIV?
Demonstrate virus in placenta or cord blood. Do P24 antigen detection or RTPCR within one month or ELISA at 18 month.

11. Why ELISA at 18 months?
Maternal immunoglobulins do circulate in baby's blood for 12-18 months. Hence to avoid false positive HIV test, do ELISA after 18 months.

12. Should ART be recommended even if women can not afford ART?
Yes! ART helps in reduction of plasma viral load. Duration of therapy will determine the proportion of reduction of viral load and thereby protection to newborn. At few center ART is free of cost through Govt of India (NACO).

13. Is ART safe during pregnancy?
Not always. Zidovudine, Lamivudine are safe during pregnancy. Nevirapine may cause sensitivity reaction like Steven-Johnson syndrome. Each has side effects if used for long period and monitoring with blood parameters is essential. (see table 2). Protease inhibitors are to be avoided during pregnancy. (for drug, doses and duration consult expert).

14. What ART newborn is given at birth?
New born is given Syrup Nevirapine 2 mg/kg with 72 hours of birth. If mother has arrived late during labor, (no ART given) new born is given two doses, first within 10 minutes followed by booster dose at 72 hours of birth.

15. What is the effect of pregnancy and childbirth on progression of the HIV disease?
Pregnancy does not accelerate the progression of HIV disease. Immunological parameter reverses to nonpregnant state within 3 months.

16. What is the effect of HIV on pregnancy outcome?
HIV does not affect pregnancy outcome. Poor outcome are due to secondary infections, poor nutrition and hygiene.

17. How safe is breast-feeding?
Breast-feeding is not absolutely safe. Risk is known to be 7-15 %. It is minimum in first 5 months. Considering risk associated with bottle-feeding, breastfeeding is permitted in Indian setting for five months. Encourage early weaning.

18. What is the role of induced abortions in HIV infected pregnancy?
Abortion are encouraged before five months. To avoid future stigma to children, discrimination in school, orphan hood, sibling separation, MTP is better choice.

19. What Contraception is safe in HIV infected women?
None is safe except condom. IUCD is relatively contraindicated, unless women observe good genital hygiene. Oral pill not recommended as has drug interaction with ART and failure are known due to reduced efficacy. Permanent sterilization is best. Despite sterilization couple should use condom to avoid exchange of microbial flora.

20. Should discordant couple be encouraged for pregnancy?
If wife is HIV seronegative, MTCT is unlikely if she is not in window period. But possible future widowhood and orphan hood be considered before making any decision. If husband is seronegative, risk of MTCT remains depending on how advance is the disease in mother's body.

21. Should infertility investigation be encouraged in HIV positive couple or discordant couple?
No! Extensive counseling is required. If couple insists, artificial reproductive technis may be recommended.

22. What are the universal precautions observed during labor?
1. Use of following things is advocated sterile gloves, sterile gown, sterile footwear, sterile headcover, mask and goggles.
2. Avoid needle pricks
3. Avoid splashing of blood.

17 Grand Multipara and Obstructed Labor

Pushpa S Junghare

HISTORY

1. Obstetric history should be asked in details including
   - Duration of marriage
   - Gravidity: No of times she was pregnant
   - Parity: No of times she delivered.
   - Details regarding her previous pregnancies and deliveries.
2. Socioeconomical history
3. Educational history
4. Awareness regarding contraception
5. History pertaining to presence of anemia, diabetes hypertension.

GRAND MULTIPARA (FAQ)

1. Define grand multipara.

A woman who has delivered 4 or more viable babies, and is pregnant for the fifth time.

2. Is it very commonly encountered condition?

As family size is decreasing a case with grand multiparty is less commonly encountered.

3. What are the risks of grand multiparity?

Anemia, PIH, pregnancy aggravated hypertension are more common. The hemorrhage of all varieties, i.e. before, during and after delivery is doubled, uterine ruptured tripled. The incidence of caesarian section rate is also increased.

4. Any other associated risk factors?

Patient is older in age, her cardiovascular system is less resilient, obesity might have set in. Majority of these patients may be poor, overworked and tired, unregistered or not having antenatal care. Prolonged lactation can deplete them of calcium.

5. What obstetrical complications are encountered in a grand multipara?

1. Twin pregnancy
2. Placenta praevia

3. Abruptio placenta
4. Malpresentations
5. Preterm labor.

6. What are complications during labor?

1. Malpresentation —due to pendulous abdomen together with the high angle of pelvic inclination—breech presentation, transverse and obli lie are more common.
2. Delayed engagement of head can give rise to PROM and cord complications.
3. Cephalopelvic disproportion may occur. The babies tend to get larger with successive pregnancies. Moreover the pelvic inclination is increased and forward subluxation of sacrum upon the sacroiliac joints reduces the true conjugate.
4. Incidence of caesarean section is there by increased.
5. Precipitate labor might occur.
6. Obstructed labor.
7. Rupture of uterus.

7. What are IIIrd stage or postpartum problems?

With every pregnancy and delivery the uterus undergoes increase in fibrous tissue and decrease in elastic tissue. This affects the contractility. III stage bleeding and postpartum bleeding is more common. Operative interference may add to traumatic PPH.

8. How to manage a grand multipara?

Patient is asked to attend regular antenatal OPD as a high risk patient. Her anemia should be prevented or corrected with appropriate iron therapy. Patient reporting late in pregnancy may need blood transfusion for the same. One should be vigilant regarding hypertension or presence of diabetes mellitus.

9. What about Obstetric management?

After ruling out malpresentation and cephalopelvic disproportion, patient can be given trial of vaginal delivery. It should be a hospital delivery.

Labor should be more meticulously monitored so as to detect cord complications, malpresentation like brow presentation or obstructed labor.

Timely intervention will be helpful. Patient should be electively taken for LSCS if there is transverse lie at term or cephalopelvic disproportion.

10. How to manage III stage?

As this patient is high risk for III stage bleeding and PPH, prophylactic measures against this complication should be taken. I V line should have been set up during labor, blood should be kept ready and uterotonic drugs can be used to prevent postpartum hemorrhage.

11. Give details.

IV methargin 0.4 mg can be given to mother at birth of anterior shoulder or 10 units of pitocin can be given IM or combined methargin and oxytocin can be given IM injection—Carboprost 125 mg IM can also be given at birth of anterior shoulder or any time later.

12. Any newer alternatives?

If it is not possible to give injectables and drugs which require cold storage, tablet Misoprostol 200 to 1000 mg can be given orally, sublingually or kept in rectum for prevention of PPH.

13. What if patient does not respond and develops PPH?

After trying all conservative methods to treat atonic PPH and after ruling out traumatic PPH, patient can be treated more vigorously. Laparotomy should be performed to expedite hysterectomy rapidly. Blood transfusion should be given as required. Delay in treatment might land the patient in irreversible shock, Sheehan's syndrome, DIC, etc.

14. What about obstructed labor and rupture uterus?

These are preventable complications and good antenatal and intrapartum supervision should take care of this. Still unbooked patient may come with these problems. After resuscitation, irrespective of foetal outcome, patient should be operated. After doing caesarean section for obstructed labor, bilateral tubectomy should be ideally carried out. After rupture of uterus, patient can be salvaged by laparotomy and repair of uterus followed by tubectomy. If the trauma is irreparable, one should go ahead with hysterectomy.

15. What advise you will give after delivery?

If patient deliveries vaginally with no complications, she should be advised tubectomy operation before discharge.

16 Are complications of grand multiparity preventable?

One should discourage grand multiparity. Every patient coming for antenatal care and delivery should be counseled regarding small family size and family planning methods.

A grand multi coming in first-half of pregnancy should be given the option for MTP followed by some contraception.

17. How to tackle malpresentation?

Transverse, Obli or unstable lie is more common in grand multipara simply because of pendulous abdomen, baby size may be average and pelvis might be adequate. In such cases external cephalic version during last few weeks of pregnancy or early labor may be attempted. If this fails patient should posted for LSCS.

18. How to minimize morbidity and prevent mortality in mother and child?

One should be necessarily very alert when a grand multipara patient is admitted with any compliant anticipate the complications and she should be treated as a dire emergency. A transverse lie may end up as neglected shoulder presentation, cord prolapse, obstructed labor or even rupture uterus.

A senior obstruction should be informed early when ever such patient is admitted, so that the patient is properly managed.

18 Occipito Posterior Position

Kanan Yelikar, Ashwini Yelikar

1. How will you define presentation?

It is defined as that part of the fetus, which occupies the lower pole of the uterus, e.g. cephalic and podalic.

2. Define lie, position, attitude, presenting part

Lie: It is the relation of the long axis of the fetus to the long axis of uterus or maternal spine, e.g. longitudinal lie, oblique lie and transverse lie.

Position: this is the relation of the denominator on the presenting part, with the four quadrants of the pelvis, e.g. LOA left occipito anterior means, the presentation is vertex, denominator is occiput, position is left anterior and lie is of course longitudinal.

Attitude: It is the relations of different parts of the fetus with each other, e.g. the universal attitude is that of flexion.

Presenting part: It is that part of the fetus which is at the cervix or which is first felt on doing a P/V examination.

3. What are the incidences of different presentation

Cephalic 96%

- Vertex 95.5%
- Face 0.4%
- Brow 0.1%

Podalic 4%

- Breach 3.5%
- Transverse 0.5%

4. Define occipito-posterior position?

In vertex presentation, when the occiput lies in one of the posterior uadrants or lies over sacroiliac joints or on the sacrum directly.

5. What are varieties of OP?

1. Oblique
2. Direct OP or occipito-sacral
3. Occipito-transverse.

6. Which position is more common?

Right occipito-posterior (ROP) is more common, because right obli diameter of the pelvis is slightly more than the left oblique.

7. What are the etiological factors for OP?

1. Anthropoid shape of the pelvis
2. Deflexed head
3. Poor uterine action
4. Cephalopelvic disproportion.

8. How to confirm the diagnosis?

- Suprapubic flatness
- Limbs felt easily anteriorly
- Delayed engagement of head.

P/V sausage shaped bag of membranes anterior Fontanel felt easily.

- Early rupture of membranes
- Sagittal suture in right oblique.

9. How will you manage OP?

Watchful expectancy 80 to 90% of OP rotate anteriorly and deliver vaginally.

10. What are the unfavorable mechanisms?

POP persistent occipito posterior position if the head fails to rotate, in spite of good uterine contractions, till the end of 2nd stage of labor.

11. How will you mange POP?

1. Cesarean section in modern obstetrics.
2. Manual rotation.
3. Forceps rotation and extraction (Keillands forceps)
4. Ventouse
5. Craniotomy if baby is dead.

19

Breech Presentation

Sonali Deshpande

1. What is the incidence of breech presentation?

The incidence of breech presentation is 20% at 28 weeks and drops to 5% at 34 weeks and 3% at term.

2. What are the etiological factors for breech presentation?

The etiological factors are:

1. Prematurity
2. Maternal factors—multiparity, polyhydramnios, contracted pelvis, uterine anomalies, fibroid uterus
3. Placental—cornual-fundal attachment of placenta, placenta previa
4. Fetal—multiple pregnancy, hydrocephalus, anencephaly, IUFD.

3. What are the different varieties of breech presentation?

There are 4 varieties:

a. Complete (20%)—flexion at thighs and knees
b. Frank (60-80%)—flexion at thighs and extension at knees
c. Footling (10%)—single or double, with extension at thighs and knees
d. Knee—single or double, with extension at thighs and flexion at knees.

4. What do you mean by recurrent or habitual breech?

When the breech presentation occurs in 3 or more consecutive pregnancies it is called as habitual or recurrent breech. The causes are congenital malformation of uterus and repeated cornufundal attachment of placenta.

5. How will you diagnose breech presentation clinically?

P/A Hard globular ballottable fetal head is found to occupy fundus.

Lateral grips indicate back on one side and small fetal parts on other.

On Pawlik's grip, ballottable breech if not engaged and after engagement the fourth maneuver shows the firm breech to be beneath the symphysis. Fetal heart sounds are above the level of umbilicus.

On P/V examination, both ischial tuberosities, the sacrum, the anus, and sometimes the external genitalia. Sometimes the finger may be meconium stained.

6. What do you mean by complicated breech?

When the breech presentation is associated with obstetric conditions which adversely influence the prognosis such as prematurity, placenta previa, contracted pelvis.

7. What is the importance of meconium in breech presentation?

Presence of meconium before the fetus is in pelvis is abnormal and should be considered as a possible sign of fetal distress.

8. What is the role of USG in breech presentation at term?

USG should be done to know the type of breech , placental localization , EFW, fetal anomalies, amniotic fluid index and to know the flexion of head.

9. What is difference between spontaneous, assisted breech and total breech extraction?

a. In spontaneous breech delivery—The entire fetus is expelled by natural forces of mother, with no assistance other than support of baby as it is being born.
b. Assisted breech—The fetus is delivered by the natural forces as far as the umbilicus. The remainder of the baby is extracted by the attendant. (assistance is required above the level of umbilicus)
c. Total breech extraction—The entire body of the fetus is extracted by the attendant (assistance is required below the level of umbilicus).

10. How will you select the mode of delivery in breech presentation?

The choice of abdominal/vaginal route depends upon the history of previous breech delivery, type of breech, flexion of fetal head, fetal size, estimated fetal weight, quality of uterine contractions, size of maternal pelvis and other obstetric complications.

11. What is ECV? How is it performed?

ECV is external cephalic version that is the procedure of converting breech of a longitudinal lie or transverse lie into cephalic presentation.

One or two operators are necessary for the procedure. The procedure should be performed in a setting where fetus can be monitored and LSCS can be performed immediately. NST should be done before and after the procedure. USG to confirm breech presentation and to assess the AFI.

Administration of tocolytics may be beneficial.

The patient should be tilted laterally to prevent supine hypotension.

First the fetal breech is elevated out of the maternal pelvis. The version is attempted by turning the fetus in forward roll (move the fetus in the direction of the fetal nose). If attempts at inducing a forward roll motion are unsuccessful, the opposite direction may be attempted. The amount of force exerted is gauged by the patients pain tolerance.

After versions, fetus should be monitored for 30 min.

Rh immunoglobulin should be administered to Rh negative mothers.

12. What are the contraindications for ECV?

Absolute contraindications: III trimester bleeding, ruptured membranes, severe fetal anomalies, uterine anomalies, multiple pregnancy, preeclampsia.

Relative contraindications: uterine scar, maternal obesity, oligohydramnios, IUGR.

13. What are the complications of ECV?

Fetal heart rate deceleration, fetomaternal hemorrhage, PROM, abruptio placenta, fetal brachial plexus injury and rarely amniotic fluid embolization.

14. What are the advantages of ECV?

The success rate is 70-80%. The benefits of ECV include the increased probability that the fetus will be in the cephalic presentation when labor sets in and to achieve uncomplicated vaginal delivery. It also decreases the LSCS rates.

15. Why to perform ECV at 36 completed weeks?

a. If spontaneous version was to occur, it would have happened spontaneously
b. Risk of spontaneous reversion is decreased after a successful version at term
c. Should complication arise during attempted version, it is possible to accomplish an emergency delivery at term.

16. What are the factors associated with successful version?

Multiparity, less gestational age, frank breech are associated with successful version.

Lower success rates with nullipara, advanced cervical dilatation, anterior placenta, low station of breech, EFW < 2.5 kg and > 3.5 kg.

17. What are the principles of Breech delivery?

a. Progress of labor should be left undisturbed until the presenting part starts descending the perineum and then give liberal episiotomy.
b. Avoid undue haste. Gentle suprapubic pressure to maintain the flexion of fetal head by the assistant.
c. Keep the back anteriorly. Never pull from below, let it get pushed from above.

18. What is Zatuchni and Andros scoring system?

For making the decision of abdominal/vaginal route in breech presentation, this system is used.

Table 19.1: Zatuchni-Andros Breech scoring

	Add 0 points	*Add 1 point*	*Add 2 points*
Parity	0	1	2
Gestational age, week	39+	38	<37
EFW, lb	8	7-8	<7
Previous breech	0	1	2
Dilation	2	3	4
Station	-3	-2	-1

If the score is 0-4, cesarean delivery is recommended.

19. What are the fetal dangers in breech delivery?

1. Intracranial hemorrhage
2. Asphyxia:
   Cord compression, cord prolapse, prolonged labor, and aspiration of amniotic fluid and vaginal contents caused by active breathing before the head has been born.
3. Injuries: Hematomas-brain, testicles, sternomastoid and thigh.
   Fractures: Femur, humerus, clavicle and skull.
   Viscera: Liver, adrenals, kidneys and lungs.
   Nerves: Brachial plexus and spinal cord.

20. Why the perinatal mortality is more in breech delivery?

Prematurity, congenital anomalies, birth asphyxia, fetal injury and cord prolapse.

21. What is the incidence of cord prolapse with different varieties of breech?

For footling—15%
Complete breech—5%
Frank breech—0.5%.

22. What is hyperextension of fetal head?

When the angle of extension of fetal neck is more than 90 degrees, it is called hyperextension or the star gazing breech. It is diagnosed on ultrasound by viewing the upper region of spine and fetal head in sagittal plain.

23. What is your opinion about Induction/Augmentation of labor in breech delivery?

Selective oxytocin augmentation should be considered when the clinician is confident that the cause of failed progress is inefficient uterine contractions and not the fetopelvic disproportion.

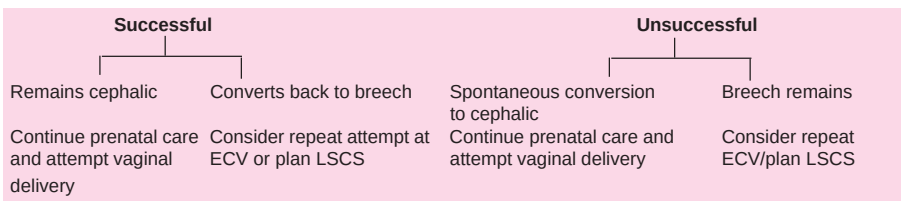
Induction of labor should not be entertained if the cervix is unfavorable.

24. What is the overall incidence of LSCS for breech presentation?

The overall incidence is 15-50% out of which 80% are elective.

25. How will you manage breech presentation at and beyond 36 weeks?

Breech presentation without contraindication for vaginal birth → Perform ECV



26. Why the risk of vaginal delivery is more in preterm breech?

In addition to the complications of prematurity, the preterm breech is at risk of entrapment of fetal head in an incompletely dilated cervix, cord prolapse, hypoxia and intraventricular hemorrhage.

27. Is LSCS justified for preterm breech?

LSCS will probably reduce the trauma but unlikely to eliminate the complications of prematurity. Cochrane database study does not justify a policy of elective LSCS for preterm breech.

28. What are the indications for elective LSCS in breech?

Elective LSCS is generally planned for patients with complicated breech presentations. The high risk factors may include previous LSCS, contracted pelvis, BOH, h/o infertility, placenta previa, IUGR, footling presentation, hyperextension and fetopelvic disproportion.

29. What is the timing and what are the advantages of episiotomy in breech delivery?

After the climbing of breech at perineum, liberal episiotomy is given. The advantages of episiotomy are

1. It helps in vaginal manipulations
2. Facilitates easy delivery
3. For unmoulded aftercoming head, it avoids compression and decompression.

30. RCOG guidelines for the management of breech presentation.

Reducing the incidence of breech presentation by ECV.

There is no evidence to support routine recommendation of the knee-chest position.

All women with an uncomplicated breech pregnancy at term (37-42 weeks) should be offered ECV.

Tocolysis is effective, both when used routinely and when used selectively.

ECV should be done near to facilities for emergency delivery. A cardiotocograph is necessary. Ultrasound is helpful.

There is insufficient evidence to support the use of regional anaesthesia to facilitate ECV.

The best method of delivering a term frank or complete breech singleton is by planned caesarean section.

A trial of labor should be precluded in the presence of medical or obstetric complications that are likely to be associated with mechanical difficulties at delivery.

Intrapartum management

There is no evidence that epidural analgesia is essential and, in selected cases, induction or augmentation may be justified. Fetal blood sampling from the buttocks provides an accurate assessment of the acid-base status, when the fetal heart rate trace is suspect.

Management of the preterm breech and twin breech:

ECV before term has not been shown to offer any benefits.

There is insufficient evidence to support routine caesarean section for the delivery of preterm breech.

There is insufficient evidence to support caesarean section for the delivery of the first or second twin.

It is essential that all details of care are clearly documented, including the identity of all those involved in the procedures.

31. Do you know any postural management of breech presentation?

1. Knee-chest position
2. Supine position with pelvis elevated with a wedge shaped cushion.

32. What are the indications of breech extraction?

Acute fetal distress in fully dilated cervix and to deliver second twin.

33. What are the causes of arrest of aftercoming head?

1. Arrest at brim, deflexed head, contracted pelvis, hydrocephalus
2. Arrest in the cavity deflexed head and contracted pelvis
3. Arrest at outlet deflexed head and rigid perineum.

34. What are the causes of arrest of breech at different levels?

1. Arrest at brim—Fetopelvic disproportion, extended breech, uterine inertia
2. Arrest at outlet—Fetopelvic disproportion, rigid perineum, uterine inertia, hydrocephalus.

34. What will you do if the delivery of head occurs through an incompletely dilated cervix?

The cervix is to be pushed up while traction of the fetal trunk is made by Jaw flexion and shoulder traction method. If necessary, Duhrssen's incision at 2 and 10 O'clock position of the cervix.

35. Which forceps are applied for the delivery of aftercoming head?

The Piper's forceps—These are specialized forceps with pelvic noncephalic application which maintains head in flexed position.

36. How will you differentiate between breech and face presentation?

Breech	Face
1. Anal opening and two ischial tuberosities in one line	1. Mouth and 2 malar prominences make a triangle
2. Suckling absent	2. Suckling present
3. Fingers stained with meconium	3. Not stained so.
4. External genitalia diagnostic	4. Eyes and nose diagnostic.

37. How will you differentiate between foot and hand?

Foot	Hand
1. Toes are of equal length	1. Fingers are of unequal length
2. Toes are shorter	2. Fingers are longer
3. Great toe has lesser mobility	3. Thumb can be abducted easily
4. Firm heel can be felt	4. No equivalent hard thing

38. What is Pinard's maneuver?

The Pinard's maneuver may be needed with a frank breech to facilitate delivery of the leg, only after the fetal umbilicus has reached. Flexion of the fetal knee

by giving pressure in the popliteal fossa associated with abduction of the thigh. This is done under general anesthesia. Procedure is obsolete in modern obstetrics.

39. What is Lovset's maneuver?

This is used where arrest of labor is due to extended arms. Lovset's maneuver is used widely for bringing down an arm in breech presentation. In this, the baby is grasped using both the hands by femuropelvic grip keeping the thumbs parallel to the vertebral column. It includes rotation of the trunk, lowering the trunk, and delivery of the arm.

40. What is Burns-Marshall's technique?

This technique is used for delivering the aftercoming head. The baby is allowed to hang by its own weight. The assistant is asked to give suprapubic pressure. When the nape of neck is visible, baby is rotated towards maternal abdomen by holding the ankles forming a wide arc.

41. What is Mauriceau-Smellie-Veit maneuver?

This is jaw flexion and shoulder traction method used to deliver aftercoming head. The assistant is asked to give suprapubic pressure. The baby is placed on the left supinated forearm. The middle and the index finger of the left hand are placed on the malar bones on either sides to maintain the flexion of the head. The index finger of the right hand is kept on left shoulder, middle finger is on suboccipital region and the ring and the little finger on the right shoulder. Traction is given in downward and backward direction till the nape of neck is visible and then fetus is rotated towards maternal abdomen.

42. What is Prague maneuver?

This maneuver is used in occipitoposterior position of aftercoming head. Back remains posterior. Two fingers of one hand grasping the shoulder of the back-draws fetus from below while the other hand draws the feet up over the abdomen of the mother.

20 Transverse Lie/ Shoulder Presentation

Kanan Yelikar

1. How will you define transverse lie?
Long axis of the fetus is perpendicular to the long axis of uterus. It also includes certain cases of obli lie.

2. What are the different positions?
The acromion process is the denominator. Hence, it could be dorso-superior/ dorso inferior/dorsoanterior or dorsoposterior. The head is usually at the lower level, as compared to podalic part. Depending on which side the head lies, it is further subdivided into right of left. Hence the total positions in transverse lie are eight.

3. Which position is common?
The left dorsoanterior is commonest position.

4. What are the etiological factors?
- Multiparity
- Contracted pelvis
- Hydramnios/twins/placenta praevia
- Arcuate or subseptate uterus.

5. How will you diagnose transverse lie?
- The fundal height is less than period of ameno
- The first pelvic grip is empty
- Fundal grip: fetal pole is not palpable
- FHS are heard in the midline.

6. How to confirm the diagnosis.
The diagnosis is confirmed by P/V, X-ray and USG.

7. How will you manage a transverse lie.
Elective LSCS after 37 weeks. Completed weeks of gestation.

8. What are the favorable events that can occur?
- *Spontaneous rectification:* Converted to vertex.
- *Spontaneous version:* Converted to breech.

- Spontaneous evolution: (Partus corpore conduplicate). The shoulders enter the pelvis, followed by the trunk, breech and then the vertex.
- Spontaneous expulsion: Baby delivered in doubled up position. Possible only in very small baby or dead fetus.

9. What are the unfavorable events?

In a case of transverse lie if the patient is allowed to labor without supervision → Rupture of membranes—liquor drains away → uterus contracts → liquor drains away → uterus contracts closely on the child → hand prolapse sometimes cord prolapse → uterine contractions increase in frequency and strength → retraction of upper segment with thinning of the lower segment → eventually rupture of uterus.

10. When will you do ECV?

ECV is done after 32 weeks till the onset of labor.

11. Describe the procedure.

ECV is an OPD procedure. Anesthesia is not required. FHR is noted carefully before and after the procedure. The baby is rotated in the direction of the nose, and converted to vertex presentation.

12. What are the contraindications?

Absolute contraindications: III trimester bleeding, ruptured membranes, severe fetal anomalies, uterine anomalies, multiple pregnancy, preeclampsia.

Relative contraindications: Uterine scar, maternal obesity, oligo-hydramnios, IUGR.

13. What is IPV?

Internal Podalic version, is done under general anesthesia, so that uterus is well relaxed. The footling is caught and the baby delivered by breach extraction.

Indications of IPV

- 2nd of twin baby with transverse lie.
- Small fetus, adequate pelvis, intact membranes or recently ruptured membranes and facilities to perform LSCS are available.

Problems of IPV

- If uterus does not relax, labor is dry, uterus retracted over the baby, then the procedure becomes difficult. Perinatal morbidity is definitely increased.

14. Role of IPV in modern obstetrics?

- Usually indicated in 2nd of the twin baby with transverse lie.

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Obstructed Labor

Kanan Yelikar, Ashwini Yelikar

1. Define obstructed labor.

When the labor comes to a standstill, in spite of good uterine contractions due to fault in the passage or passenger.

2. Common causes.

Fault in passage:

- Contracted pelvis
- Soft tissue obstruction due to tumors.

Fault in passenger:

- Malpresentation
- Malposition
- Congenital anomalies.

3. Clinical features

H/o of prolonged labor
- Maternal exhaustion
- Dehydration
- Metabolic acidosis
- Genital sepsis.

P/A Bandl's ring
- Colonic distention
- Advanced and edematous bladder
- Hematuria.

4. What is Bandls ring

It is a pathological retraction ring at the junction of active upper segment and distended lower segment. Bandls ring is the effect of obstructed labor. Constriction ring is the cause of obstructed labor.

5. Complications of obstructed labor.

- Dehydration
- Metabolic acidosis
- Sepsis

- PPH
- Rupture uterus
- Death
- Urinary complications.

Remote complications
- Genitourinary fistula
- RVF
- Sheehan syndrome.

6. Effect of obstructed labor on fetus.
- Asphyxia
- Acidosis
- Intracranial hemorrhage
- Infection.

7. Difference between constriction ring and retraction ring.

Constriction Ring	Bandl's Rings
It is manifestation of localized, incoordinate the narrow part of the fetus, i.e. neck. It uterine contractions, usually occurs around present during 1st stage of labor as pronged labor. 2nd stage of labor: pronged second stage no obvious cause. 3rd stage: Retained placenta or diagnosed during MRP. Treated by deep plane of anesthesia.	It rises towards umbilicus as labor advanced. Tenderness over the abdomen present. Round ligaments are taut and tender.

8. Treatment of obstructed labor.
Patients are treated by LSCS.

9. Difficulties during LSCS
- Overstretched LUS
- Sings of threatened rupture
- Bladder is edematous and overstretched.

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Third Stage Complications

Kalpana Mahadik

– PPH
– Retained placenta
– Inversion of uterus
– Shock.

PPH

- Definition: Apparent blood loss more than 500 ml after delivery of foetus.

Quick clinical review

Think of predisposing causes like:

- Over distended uterus
- Hypotonic uterus (twins, hydramnious inertia in first stage)
- Instrumental delivery
- Grand multipara
- Placenta previa
- History of PPH in previous pregnancy.

All these etiological causes are to be thought before occurrence of the complication. A rule has to be followed that delivery of such patient should be personally attended by the treating gynecologist.

*Some **dos** and dont's to be followed are:*

- Keep a oxytocin drip going to such patients even after the foetus and placenta is out. Convert the physiological oxytocin drip to high concentration oxytocin drip.
- Keep the uterotonic drugs like methyl ergometrin, oxytocin, prostodin, mesoprostol ready in labor room when such patient is in labor.
- Keep the bladder empty
- Watchful cat's eye, i.e. after delivery of placenta assessing the woman every 15 minutes for expression of clots.

Dont's:

- Not omitting the IV drip after delivery of placenta.
- Not leaving the woman alone after placental delivery.

- No hurry to shift the patient to bed. Patient must be observed for minimum period of two hours and shifted to bed from labor room after passing urine.

FAQS

1. What are types of PPH?

- Primary (occurring within 24 hours)
- Secondary (after 24 hours but up to puerperium).

2. Causes of PPH

- Atonic uterus 80%
  Overdistended uterus, grand multipara, malnutrition, anemia, APH, prolonged labor, anesthetics, (ether, halothane, cyclopropane) labors induced by oxytocin, mismanaged 3rd stage (retained clots), constriction ring, precipitate labor)
- Traumatic 20%
  Episiotomy, vaginal and cervical tears, paraurethral tears, calporrhexis.

3. Combined
4. Coagulation disorder
   Diminished procoagulants (Washout phenomenon) or increased fibrinolytic activity like in DIC, abruptio placentae, jaundice in pregnancy, thrombocytopenic purpura, HELLP syndrome, Intrauterine death of fetus.

3. What is prognosis in PPH.

Depends on—In how big centre patient is

- If blood bank is available
- How smart is the team around
- How much you anticipate the PPH.

4. What is important? Prevention or Treatment?

Both are equally important.

5. What are preventable causes and how do you act in such situation.

a. Anaemia treatment
b. High risk patients to be recognized like twins, hydramnios, grand multipara, APH, H/o previous 3rd stage complication
c. Anticipation of blood grouping and RH typing with probability of blood transfusion arrangement
d. Slow delivery of Trunk.
e. Preference to local anesthesia than general anesthesia.
f. Active management of third stage of labor.
g. No fiddling or kneading of uterus
h. Complete examination of placenta
i. Continue oxytocin even after delivery of placenta.
j. PNC observation for 2 hours.

6. Management of 3rd stage bleeding in brief.

Principles are:

a. Replacement of blood loss by fluid and blood

b. Keep uterus empty
c. Never forget traumatic PPH

Management started simultaneously on all fronts

- IV drip with 5-10 units oxytocin in half liter fluid
- IV methy ergometrine (4 mg) or IM Carboprost 250 µg or rectal suppository of misoprostol 800 µg.
- Fundal massage and recognizing sign of lost fundus, i.e. inversion
- Catheterize patient
- Controlled cord traction to deliver placenta.
- Decision for manual removal of placenta is to be reserved for half an hour with watchful expectancy.
- Anticipation, diagnosis and enthusiastic treatment of traumatic bleeding.

7. Management of Atonic PPH

a. General management as discussed above
b. High concentration (10-20 units) oxytocin drip or IM carboprost or Mesoprostal suppository.
c. Organize your team, nurses, resident registrar, laboratory, blood bank people and operation theatre staff. Immediately as soon as you see slightest indication of bleeding.
d. Massage uterus, expel clots
e. Bimanual compression
f. Rarely uterine packing
g. Exploration of uterus under general anesthesia
h. Bilateral internal iliac ligation
i. Hysterectomy
j. Uterine artery embolization
k. B. Lynch-Stitch
l. Shivkars pack.

8. What are causes of secondary PPH and how do you manage?

- It occurs between 8th to 14th day of delivery.

Causes:

- Retained bits of placenta
- Infection of vaginal/cervical lacerations/episiotomy wound.
- Subinvolution—low grade infection.
- LSCS—10th to 14th day, infection of uterine wound
- Patient who is on estrogen therapy for suppression of lactation, may exhibit withdrawal bleeding.
- Chorionepithelioma
- Carcinoma cervix
- Placental polyp
- Infected fibroid
- Fibroid polyp
- Subacute inversion of uterus

Retained placenta

Definition: Placenta is said to be retained when it is not expelled out 30 minutes after birth of baby.

Three Clinical Types

- Placenta completely separated but retained.
- Simple adherent placenta in uterine atony
- Placenta incarcerated in uterine cavity (constriction ring)
- Morbidly adherent placenta.

FAQS

1. What is risk involved in placental retention

- Hemorrhage
- Shock due to blood loss, unrelated to blood loss when period is more than 1 hour, frent physical forceful attempt to express placenta.
- Sepsis
- Risk of recurrence in next pregnancy.

2. Management.

Manual removal of placenta (MRP) to be done under general anesthesia, if separated only removal, if adherent it has to be separated from uterine wall and removed followed by a high concentration oxytocin drip.

3. What are associated complications with retained placenta.

- Retained placenta with shock but no hemorrhage. Treated by treatment of shock first, then MRP.
- Retained placenta with hemorrhage same as in third stage hemorrhage.
- Retained placenta with sepsis. Mostly separated and septic. Treatment is removal and broad spectrum antibiotic.
- Retained placenta with episiotomy, repair and MRP.

4. What is placenta accreta, its signs and management?

This is rare condition with placenta directly anchored to myometrium usually with low lying placenta. In this decidual layer is absent. Diagnosis by failure to find line of cleavage between placenta and uterine wall. Treatment is hysterectomy. In young woman placenta is left for being autolyzed, so that conservation of reproductive function is done.

Inversion of Uterus

When uterus is turned inside out. It is acute, following normal delivery

FAQS

1. What are causes?

- Uterine atony
- Placenta accereta
- Short cord
- Pulling of cord

- Crede's expression
- Faulty techni in MRP i.e. rapid withdrawal of hand, first pressure on atonic uterus.

2. What are complications of inversion?

- Shock-Neurogenic
- Associated hemorrhage
- Infection
- Associated pulmonary embolism.

3. How it is diagnosed?

Cupping of fundus or absent fundus, presence of pear shaped mass, reddish purple in color.

4. What is prognosis?

Death due to shock, hemorrhage or embolism.

5. Management.

Treatment of shock, antibiotics and reposition under general anesthesia as early as possible.

Postpartum Shock

Causes are:

- Amniotic fluid embolism
- Intractable postpartum hemorrhage
- Pulmonary embolism
- Unexplained sudden postpartum collapse.

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Multiple Pregnancy

Kanan Yelikar, Varsha Deshmukh

1. What is multiple pregnancy?

When more than one fetus is present in-utero it is known as multiple pregnancy.

2. What are the types of multiple pregnancy?

The types of multiple pregnancy are binovular or dizygous (75%) and Uniovular OR Monozygous (25%)

3. What are binovular twins?

When two fetuses develop from the fertilization of two ova liberated during the same menstrual cycle they are known as binovular twins. These are fraternal twins. They may be of dissimilar sex and looks.

4. Hat are uniovular twins?

These are identical or true twins. They represent complete cleavage of blastodermic vesicle. There is one egg fertilized by single sperm. Therefore the offspring arises from the same germ plasm. The babies are of the same sex and look alike. The blood circulation in these twins intercommunicate in the common placenta.

On rare occasion, the following possibilities may occur:

- If the division takes place within 72 hours after fertilization (prior to morula stage) the resulting embryos will have two separate placenta, chorions and amnions (diamniotic-dichorionic or D/D).
- If the division take place between the 4*th and 8th day after the formation of inner cell mass when chorion has already developed—diamniotic monochorionic twins develop (D/M).*
- If the division occurs after 8th day of fertilization, when the amniotic cavity has already formed, a monoamniotic—monochorionic twins develops (M/M).
- On extremely rare occasion, division occurs after 2 weeks of the development of embryonic disc resulting in the formation of conjoined twins called—Siamese twins. Four types of fusion may occur: i) Thoracopagus (commonest), ii) Pyopagus (posterior fusion), iii) Craniopagus (cephalic), iv) Ischiopagus (caudal).

5. What is Hellin's law?

- Hellin's law expresses the incidence of multiple pregnancy
- Twins—1:80
- Triplets—1:(80)2
- Quadriplets—1:(80)3

6. What is the characteristic feature of development of twins?

The characteristic feature of the twins are:

- Somewhat lighter and smaller than a singleton
- Sometimes the two are unequal in size
- Sometimes one grows normally and the other perishes
- Malformations are common
- Monsters, e.g. thoracopagus are rarely seen.

7. What is the arrangement of placenta and membranes in twins?

In binovular twins the placentae are either separate or fused. Although fused, there is NO vascular connection. In uniovular twins the placenta is single, sharing of fetal circulation. There is single chorion and two amnions.

8. Clinically when do you suspect multiple pregnancy?

The following conditions should alert you of multiple pregnancy

1. Positive family history of twins
2. Hyperemesis
3. Unexplained excessive weight gain
4. Early onset PIH
5. Unexplained severe anaemia
6. Abnormally elevated serum alpha proteins in second trimester
7. H/o excessive fetal movements
8. Height of uterus more than the period of gestation
9. Polyhydramnios

9. How do you confirm the diagnosis of twins?

Ultrasonography shows two sacs and two fetal poles by 5 weeks of gestation

10. What is the role of USG in twins?

USG gives an idea about-chorionicity, gestational age, placental localization, AFI, congenital anomaly, presentations cervical status and conjoined twins.

11. What is the antenatal care in twins?

Frent visits are recommended in twins, i.e. every two weekly after 26-30 weeks. Patient is advised to increase the calorie intake by 300 kcal/d, take extra bed rest, and to report immediately if backache starts. The physician should be vigilant about early diagnosis of preterm labor, PIH and IUGR. Proper advice is given to the patient.

12. What advise will you give to the patient?

1. Balanced and rational account of the risks
2. In spite of the care adverse outcome is possible
3. Idea about the increase burden of physical, emotional and financial factors

13. What are the advantages of bedrest in twins?

Bedrest offers many advantages, e.g. it increases the uterine blood flow, limits the physical exertion, decreases the incidence of preterm labor, reduces the chances of PIH and is believed to improve the perinatal outcome in terms of weight.

14. Enumerate the maternal risks associated with multiple pregnancy?

I. Trimester-abortions, congenital malformations and hyperemesis
II. Trimester anaemia, PIH, polyhydramnios, preterm labor
III. Trimester APH (pl previa and abruption pl), malpresentations, PROM, Labor—malpresentations, PROM, cord prolapse, increased operative interference, PPH.

Puerperium-failing lactation, subinvolution.

15. Why PPH occurs more commonly in twins?

The causes of PPH are uterine overdistention leading to atonicity, more incidence of placenta previa, larger placenta taking more time to separate leading to more blood loss and more sinuses are opened up which again leads to blood loss.

16. What are the P/A findings s/o twins?

Overdistension of the abdomen, palpation of multiple fetal parts, (two heads or three fetal poles) are the findings s/o twins.

Auscultation of two fetal hearts simultaneously by two different personnel, with a difference of 10 beats per min

17. What information is obtained from 1st trimester USG?

1. Two sacs with fetal poles
2. Determination of chronicity
3. Determination of gestational age.

18. What information is obtained from 2nd trimester USG?

II tri USG gives an idea about-chronicity, gestational age, placental localization, AFI, congenital anomaly, cervical status and conjoined twins

19. What weight gain is recommended in twin pregnancy?

25-35 lb or 12. 5-15 kg

20. What are the advantages associated with adequate weight gain?

Adequate weight gain in twin pregnancy is associated with decreased incidence of VLBW, IUGR, preterm labor and better perinatal outcomes.

21. What dose of iron is recommended in twin pregnancy?

Ferrous sulphate is given in the dose of 60-120 mgm till Hb becomes normal, then it is given as 60 mgm/d.

22. What dose of calcium is recommended in twin pregnancy?

1800 mgm/d of calcium is recommended in twin pregnancy.

23. When is the cervical evaluation done in twin pregnancy?

The cervical evaluation done in twin pregnancy if there is a probability of impending preterm labor or at 16-20 weeks of gestation. The features of

impending preterm labor are evidence short cervix, history of bleeding p/v, dilatation of the cervix or descent of the presenting part.

24. What are the features feature of twin pregnancy in labor?

The characteristic feature of twin pregnancy in labor are short latent phase, common in multigravidae, birth weight less than 2. 5 kg and associated with malpresentations.

25. What are the precautions to be taken in labor?

1. Anticipate and recognize the increase potential for complication of labor and delivery in twins
2. Establish IV access
3. Strict FHR monitoring
4. Trained obstetrician available
5. Immediate availability of anaesthetist
6. Availability of trained neonatologist.

26. How fetal presentation affect delivery in twins?

On admission the fetal presentation is established. If there is any doubt USG help can be taken. Generally vertex-vertex are delivered vaginally. Sets in which twin A is non vx is delivered by LSCS. Management of vx-nonvx set is controversial.

27. What are the precautions to be taken after the delivery of first baby in twins?

1. Do not give ergometrine
2. Set up oxytocin drip so that the contractions start and presenting part is fixed
3. Then do ARM so that the chances of cord prolapse are minimized.

28. Of the two babies which one is at a higher risk?

The II baby has a higher chance of fetal distress secondary to cord prolapse and abruption placentae.

29. What are the management options if the II baby is by breech?

The management options if the second baby is by breech are either do ECV or deliver by breech.

30. What are the management options if the II baby is by transverse lie?

The management options if the second baby is by transverse lie are to do ECV or LSCS. If the obstetrician is experienced IPV is also an option.

31. What are the indications of LSCS in twins?

1. Malpresentations
2. Placenta previa
3. Cord prolapse
4. Conjoint twins
5. Monochorionic twin.

32. What is the management of III stage of labor?

PPH is a major complication in twins in third stage of labor.

1. Give ergometrine after the delivery of the second baby
2. Start oxytocin drip 20 units.

3. Keep bladder empty
4. Replace the excessive blood loss by transfusion kept ready.

33. What are the fetal complications in multiple pregnancy?

1. Preterm labor—50% deliver before 37 weeks
2. IUGR—10-25%
3. Increased perinatal morbidity—5 times of singleton pregnancy
4. Abortions—2 times of singleton pregnancy
5. Malformations—2 times of singleton pregnancy.

34. What is fetus papyraceous?

Of the two fetuses one perishes in-utero. The fluid in the dead amnion is absorbed. The body of the dead fetus is compressed in between the live fetus and the uterine wall. This results in a curiously flattened mummified fetus known as fetus papyraceous.

35. What is Vanishing twin syndrome?

Every one in eight pregnancy begins as twins. But of the two fetuses one is absorbed. This is known as vanishing twin syndrome.

36. What is the definitive evidence of uniovular twins?

The definitive evidence of uniovular twins is the acceptance of tissue graft.

36. What is superfecundation?

Two ova from the same cycle may be fertilized by the sperms from different acts of coitus within a short period, it is known as superfecundation

37. What is superfetation?

It is the fertilization of two ova from different cycles, separated by weeks or even months. There is no evidence of this phenomenon in human beings.

38. Why incidence of twins is increasing?

The incidence of twins is increasing due to ART procedures.

Procedures	incidence of twins
Gonadotrophins induction	20-40%
IVF	22%
Clomephene induction	7-13%

39. How does USG help in diagnosis of zygosity?

The separating membrane (> 2 mm thickness) in between the two sacs is diagnostic of dizygous twins. If this membrane is not seen then it is a monozygous pregnancy.

40. What is twin to twin transfusion syndrome?

When there is intercommunication of blood vessels in the placentae, it results in twin to twin transfusion syndrome. When one placenta is fed by an artery from the first twin and drained by a vein that leads to the II twin, it leads to the shifting of blood from the donor twin to the recipient twin, this is known as twin to twin transfusion syndrome.

41. What is the characteristic feature of twin to twin transfusion syndrome?
Due to the intercommunication of blood vessels in the placentae, the donor baby is hypoperfused, undergrown, hypotensive and has oligohydramnios. The oligohydramnios maybe severe to cause "stuck" baby. The recipient twin is polycytmic, edematous and has cardiac hypertrophy and polyhydramnios. Thus both the babies are at risk. The perinatal mortality is as high as 70-80% if it occurs before 28 weeks.

42. What are locked twins?
It is a rare phenomenon. When the I baby is by breech and the II baby is by vertex, the babies may get locked with each other. This occurs more commonly with moamniotic twins.

43. What is the role of selective reduction in multiple pregnancy?
With ART, high order gestations occur frently. Both the fetal and maternal outcomes are compromised in the high order gestations. This outcomes are believed to improve if the pregnancy is reduced to a lower gestation number in late I trimester. In selective reduction in multiple pregnancy Inj KCL is injected in the fetus who is to be reduced.

44. Do twins differ from singleton pregnancy in pulmonary maturity?
Overall, pulmonary maturity is attended at a earlier gestation (3-4 weeks) in twins than singleton pregnancy.

45. What difficulty is seen in the treatment of preterm labor in twins?
In twins the plasma volume is increased up to 50-60%, whereas it increases by 45% in singleton pregnancy. Therefore the cardiac load increases. This poses a special risk for pulmonary edema after giving tocolytics. Hence tocolytics should be used cautiously.

46 What is the incidence of twins in India?
The incidence of monozygotic twins is almost the same through out the world. The incidence of dizygotic twins however changes from place to place. In India it is about 6-8/1000 live births.

Bombay 6. 8/1000
Banglore 7. 3/1000
Kolkata 8. 1/1000

24

Drugs in Obstetrics

Seema Chopra

HISTORY

- Prenatal counselling—drug use during preconceptional period
- History of smoking, alcohol or recreational drug intake in pregnancy
- Drugs used for medical disorders before conception or during pregnancy.

EXAMINATION

- Level-II Ultrasonsography at 18-20 weeks
- Maternal serum markers like alpha feto protein for neural tube defects
- Fetal echocardiography.

QUESTIONS

1. What is a teratogen?

Any agent that acts during embryonic or fetal development to produce a permanent alteration of form or function is known as a teratogen. Currently recognized teratogens include chemicals, viruses, environmental agents, physical factors and drugs.

2. What are the criteria to classify any given agent as a teratogen?

a. The causation because of exposure to teratogen must be proved, with at least three cases with same exposure have been identified earlier.
b. The agent must cross the placenta in sufficient quantity.
c. Exposure to teratogen must be during a critical developmental period.
d. Epidemiological findings must be consistent.

3. Mechanism of teratogenicity.

Teratogens act by disturbing specific pathogenetic processes, leading to cell death, altered tissue growth or abnormal cellular differentiation. These processes may be induced in different cells or tissues resulting in multiple defects. Different drugs can produce similar pathophysiological processes, e.g. fetal hydantoin syndrome because of exposure to phenytoin may cause growth deficiency, mental retardation, craniofacial abnormalities, hypoplasia of the distal phalanges and widely spaced nipples. Similar abnormalities are

also known to be caused by prenatal exposure to carbamazepine and is also similar to fetal alcohol syndrome.

4. What is "critical developmental period" in prenatal period?

The exposure to teratogen can occur during three periods.

a. Preimplantation period—2 weeks from fertilization to implantation and is called all or none period.
b. Embryonic period—2nd to 8th week following conception during which organogenesis occurs. Exposure to teratogens in this period results in embryopathy and is the most crucial period for development of structural malformations.
c. Fetal period for development continues till birth. The exposure during this period results in fetopathy.

5. What are the metabolic pathways affected by exposure to teratogens?

Two major pathways affected are:

- Disruption of folic acid metabolism
- Production of toxic oxidative intermediates.

Folic acid: It is essential for i) methionine production which is required for methylation reaction and thus production of proteins, lipids and myelin ii) for normal meiosis and mitosis.

Oxidative intermediates: Oxides and epoxides produced by drug metabolism are not detoxified by weak fetal epoxide hydroxylase and these free oxide radicals have carcinogenic, mutagenic and other toxic effects in a dose related manner.

6. What is the effect of paternal exposure to teratogens?

Paternal exposure to drugs or environmental agents can lead to increased risk of adverse fetal outcome because of:

- Induction of gene mutation or chromosomal abnormality in sperm
- Drug in seminal fluid can affect the fetus directly
- Altered gene expression in paternal germ cells.

7. What is the risk of birth defect because of exposure to teratogens?

Risk of birth defect in all women is 3%. It is increased by 1-2% or at the most doubled or tripled because of exposure to a confirmed teratogen. Risk versus benefit should be explained as some diseases may pose greater risk to both mother and fetus than use of drug.

8. What is "fetal alcohol syndrome"?

Maternal alcohol consumption is one of the most common recognizable cause of mental retardation in the United States. The affected child shows behavior disturbances, brain, cardiac and spine defects, craniofacial anomalies, learning disorders, epilepsy and cerebral palsy.

9. What are teratogenic effects of antiepileptic drugs?

Phenytoin causes—Fetal hydantoin syndrome.

It includes—Craniofacial anomalies, fingernail hypoplasia, developmental delay, cardiac defects, cleft palate and cleft lip.

Carbamazepine—Fetal hydantoin syndrome, spine bifida.

Valproate—Neural tube defects.

Phenobarbitone—Cardiac anomalies, cleft lip and palate, urinary tract malformations.

10. What is "Warfarin embryopathy"?

Exposed to the coumarin in intrauterine life, the fetus is at an increased risk of warfarin embryopathy characterized by nasal and midfacial hypoplasia, stippled vertebral and femoral epiphysis. Warfarin derivatives exert this effect by decreasing osteocalcins which control embryonic calcification. This syndrome is a phenocopy of chondrodysplasia punctala. It occurs in 8% of women taking warfarin in first trimester.

11. What is the teratogenic effect of Warfarin if taken during second and third trimester?

The defects because of fetal exposure to warfarin in second and third trimester result from hemorrhage in different organs. These include agenesis of corpus callosum, Dandy Walker malformation, microphthalmia, optic atrophy and blindness, mental retardation.

12. What are the fetal risks because of exposure to angiotensin converting Enzyme (ACE) inhibitors?

ACE inhibitors disrupt the rennin-angiotensin system which is essential for normal renal development. In addition, they may provoke prolonged fetal hypotension and hypoperfusion leading to anuria. This results in oligohydramnios, prevents normal lung development and limb contractures, growth restriction, relative limb shortening and skull maldevelopment. Thus "ACE inhibitor fetopathy "results due to fetal effects of ACE inhibitors.

13. What is isoretinoin embryopathy?

Use of Vitamin A isomers for dermatological disorders such as acne during first trimester is associated with a high rate of fetal loss. The risk of malformation in survivors is increased 26 fold. The risk of malformations are bilateral, but asymmetrical and include microtia or anotia (stenosis or agenesis of external ear canal), cardiac anomalies such as outflow tract defects, hydrocephalus and thymic hypoplasia, aplasia or malposition.

14. What are fetal effects of androgen exposure?

Congenital adrenal hyperplasia (CAH) is the Autosomal recessive disorder that results from early fetal exposure to androgens. This results from enzyme deficiencies which prevent fetal adrenal gland from hydroxylation of cortisol precursors, thus androgenic intermediate precursors accumulate. Female fetus shows masculinization of external genitalia and male fetus has abnormal genital growth. Exogenous androgen exposure leads to similar effects in fetus but these changes do not progress after birth compared to CAH.

15. What are fetal effects of exposure to testosterone, anabolic steroids and danazol?

In female fetus, varying degrees of virilization occurs. During first trimester exposure, labioscrotal fusion occurs while later exposure leads to phallic enlargement. Normal female maturation occurs at puberty. Surgical correction may be required for genital malformation. Effect of Danazol exposure is dose related.

16. What are the risks of fetal exposure to estrogens and progestins?

Estrogens—Fetal DES (Diethyl stillisterol)exposure can lead to vaginal clear cell adenocarcinoma, ectopic vaginal glandular epithelium, increased risk of cervical and vaginal intraepithelial neoplasia. Structural anomalies include hypoplastic or T-shaped uterine cavity, cervical collars, septa, hoods and withered fallopian tubes. Exposed male fetuses are at increased risk for cryptorchidism, microphallus, hypospadias, testicular hypoplasia.

Progestins—Depot preparations of Medroxy progesterone acetate can lead to virilization of female fetus and feminization of male fetus.

Oral contraceptives—Have not been shown to cause congenital malformations.

17. Are antimicrobials safe in pregnancy?

Fetal exposure to tetracyclines may cause yellow-brown discoloration of teeth. These can be deposited in long bones of fetus.

Aminoglycosides—Premature fetuses can develop ototoxicity and nephrotoxicity but no congenital defects have been reported.

Sulfonamides—Preterm neonate can manifest hyperbilirubinemia. No association with congenital malformations is shown.

18. What are fetal risks of exposure to antifungal agents?

Griseofulvin—Increased risk of central nervous system and skeletal anomalies, conjoined twins.

Fluconazole—Skull abnormalities, cleft palate, humoral radial fusion and arm abnormalities.

Itraconazole—Limb defects.

19. What are the effects of Cocaine and Cigarette smoking on pregnancy?

Cigarette smoking can lead to gastroschisis, intestinal atresia, cleft lip and palate, abnormalities of chest and arm of same side, hydrocephaly and microcephaly.

Cocaine—causes skull defects, cutis aplasia, porencephaly subependymal and periventricular cysts and visceral infarcts.

20. What are the teratogenic effects of Thalidomide?

Twenty percent of fetuses when exposed to thalidomide between 4th to 7th week of pregnancy manifest structural limb deformities in the form of limb

reduction—upper or lower limb phocomelias. Also reported are gall bladder aplasia and duodenal atresia.

21. Can analgesics be given in pregnancy?

Acetaminophen—Rate of spontaneous abortion may be increased. Few case reports show association with gastroschisis

Aspirin—Safe in low dose.

NSAID's—Ibuprofen, naproxen, ketoprofen and indomethacin have reversible fetal effects when used for short term in third trimester.

Indomethacin—Premature closure of fetal ductus arteriosus leading to pulmonary hypertension. It can also cause oligohydramnios, intraventricular hemorrhage, necrotizing enterocolitis in neonate.

22. What is the effect of anaesthetic agents on pregnancy?

Local/regional anesthesia. Anesthetic agents used are usually free of teratogenic effects. Systemic absorption may cause fetal bradycardia because of depolarizing effects.

General anesthesia: Drugs used cross placenta even if in small amount, but are not teratogens. Occupational exposure to inhalation agents can lead to increased risk of abortions and decreased fertility.

23. Are antihypertensive drugs safe in pregnancy?

β-Blockers—First trimester exposure safety data is lacking, safe in second and third trimester. These drugs may cause growth restriction, neonatal hypoglycemia, B-blockade and hypotension.

Calcium channel blockers—Nifedipine has been associated with pregnancy loss rarely and is usually safe in pregnancy. Verapamil may cause decrease in uterine blood flow.

24. Can diuretics be used in pregnancy?

Thiazide diuretics when used near time of delivery, can cause neonatal thrombocytopenia, bleeding and electrolyte disturbances.

Acetazolamide, spironolactone are not known to cause adverse effects on fetus.

Fursemide can cause increased fetal urine production and patent ductus arteriosus and 8th nerve dysfunctions.

Ethacrynic acid can be ototoxic to the fetus.

25. Antimicrobial use during pregnancy—Is it safe?

Safest are penicillin group of antibacterial agents including piperacillin, tazobact, clavulinic acid. Cephalosporins do not cause adverse effects on embryo or fetus. Erythromycin, azithromycin, clindamycin, vancomycin are not associated with congenital defects.

Chloramphenicol crosses placenta but does not cause increase in congenital anomalies. The serum levels in fetus after maternal administration are insufficient to cause "grey baby syndrome" known to occur with large doses

in preterm neonate. Nitrofurantoin is safe in pregnancy and rarely causes hemolytic anaemia in G6-PD deficient mother. Ciprofloxacin is associated with epiphyseal stippling in fetus.

26. What is the risk factor assigned to different drugs?

Risk factors (A, B, C, D, X) are based upon the level of risk the drug poses to fetus.

Category A—No risk to fetus demonstrated in controlled human studies.

Category B—Animal studies may or may not show fetal effects not confirmed in controlled studies in women.

Category C—Animal studies show fetal adverse effects not seen in human studies or neither animal nor human studies are available.

Category D—Evidence of human adverse effect is there but the benefits outweigh risks.

Category X—Drugs contraindicated in pregnancy. Risks outweigh benefits.

25

Induction of Labor

Kanan Yelikar

1. What do you mean by induction of labor?

IOL is defined as an intervention intended to artificially initiate uterine contraction resulting in the progressive dilatation and effacement of cervix with descent of the presenting part.

2. What are the different indications for induction of labor?

1. Postterm pregnancy
2. IUGR
3. Pre-eclampsia
4. IUD
5. APH
6. PROM/chorioamnionitis.

3. What do you mean by stabilizing induction?

Stabilizing induction describes induction where the presenting part is not within the pelvis. If the delivery can't be delayed, an infusion of oxytocin should be commenced prior to amniotomy to ensure that contractions have commenced and head is over the brim.

4. What is Bishop's score?

	0	*1*	*2*	*3*
Dilatation (CM)	0	1-2	3-4	5-6
Effacement (%)	0-30	40-60	60-70	80+
Station	-3	-2	- 1/0	+ 1/+2
Consistency	Firm	Medium	Soft	
Position	Posterior	Mid-position	Anterior	

Total = 13
Favorable = 6-13
Unfavorable = 0- 5

5. What is modified Bishop's score?

	0	*1*	*2*	*3*
Dilatation (CM)	<	1-2	2-4	> 4
Effacement (%)	> 4	2 - 4	1 - 2	< 1
Station	-3	-2	- 1/0	+ 1/ + 2
Consistency	Firm	Medium	Soft	
Position	Posterior	Mid-position	Anterior	

Total = 12
Favorable = 6, Unfavorable < 6

6. What are the contraindication of induction of labor?

- Contracted pelvis
- CPD
- Malpresentation. Like transverse lie, placenta praevia
- Grand multipara
- Heart disease
- Pre 2 LSCS or previous classical LSCS
- Pregnancy following VVF repair
- Presence of pelvic tumour/carcinoma cervix.

7. What are the different methods of induction?

1. Medical
2. Surgical
3. Mechanical

8. Do you know, nonmedical methods induction of labor?

Yes, castor-oil, bath, enemas breast stimulation, acupuncture, sexual intercourse are suggested to be an effective method of inducing labor. But their benefits are not proved.

9. What is sweeping of membranes?

A. Sweeping or stripping of the membranes involves the digital separation of the membranes from the lower uterine segment which causes an increase in the level of PGF2-alpha

10. What are the different mechanical method of induction of labor?

A. Mechanical methods in the form of hygroscopic tents such as natural laminaria or synthetic sponges impregnated with $MgSO_4$ (lamicela), inflation of balloon of a urinary catheter within the cervical canal.

11. What is ARM?

Artificial rupture of membranes/amniotomy is frequently used as a method of induction. This is one of the surgical methods. Rupture of membranes leads to endogenous release of prostaglandins.

12. What do you mean by failure to respond?

It is defined as failure to enter the active phase of labor after 9 hours of regular uterine contraction. Failure of induction is defined as failure to deliver

vaginally at the end of 24 hours. If patient end in LSCS for indication other than fetal distress.

13. What happens to the cervix during ripening?

During ripening, a gradual dissociation and collagenolytic activity of the collagenase and protease enzyme on collagen bundles and a change in the type of proteoglycans in the ground substance, brings about the ripening effect.

14. Which drugs are used for cervical ripening?

Prostaglandins, oxytocin, estradiol, relaxin, mifepristone.

15. Which prostaglandin is used for cervical ripening?

PGE2 gel is used for cervical ripening. It is instilled in the cervical canal in a dose of 0.5 mg and can be repeated after 6 hrs. A maximum 3 doses can be applied.

16. What are the complications of IOL?

Maternal—cervical tear, rupture uterus, operative delivery. Accidental hemorrhage, infection fetal—fetal distress, sepsis and neonatal jaundice.

17. What are the contraindications to prostaglandins?

Bronchial asthma, heart disease, are the absolute contraindication to PGE2 and PGF2-alpha.

18. What is the antidote for hyperstimulation after PGE2 gel?

Terbutaline is used as an antidote.

19. What is the dose of oral PGE2 tablets for induction of labor?

- 0.5 tabl. Every hour for such 4 tab.
- After 4 hrs, 2 tablets every hour for 6 tablets.
- Maximum 10 tablets.

Side effects—nausea, vomiting, diarrhea.

20. What is the initial dose of oxytocin? What are the drug increments?

Oxytocin 2.50 in 500 cc of normal saline cardiotocography for 20 min to check the fetal condition and frency of contraction.

* Escalation every 30 min. infusion pump until 4-5 contraction/10 min.

Giving set 1 cc = 20 drops 1 cc = 15 drops

2.5 units oxytocin in 500 ml of N saline

Drops/min	Mu/min	Mu/min
10	2.5	3.3
20	5	6.6
30	7.5	9.9
40	10	13.2
50	12.5	16.5

21. Types of induction of labor

Elective and Indicated. Elective induction is done for the convenience of the patient and the doctor. Indicated induction is done for maternal or fetal indications, e.g. PROM, postdated, preeclampsia, congenital malformation of the fetus.

22. What are the pre requisites of IOL?

When possibility of vaginal delivery is fully anticipated.

1. Confirmation of gestational age
2. Fetal presentation, position
3. Estimated fetal size
4. Cervical favor ability
5. Pelvic adequacy.

23. Induction of labor in special situation?

IUFD, Pre LSCS, IUGR, twins, preterm, PROM.

Pre. LSCS induction should be considered very carefully. However, the incidence of scar dehiscence is not different than in a spontaneous labor.

Multiple pregnancies no definitive merits of an active policy of induction of labor.

PROM induction of labor is advantageous to reduce the infective sele in mother and baby.

IUGR carefully done since incidence of intrapartum fetal hypoxia is common

24. What are the different oxytocic agents?

What is the basic difference in the efficacy of PG and Pitocin?

Prostaglandins are effective in preterm induction/in unfavorable cervix/IUFD/preterm PROM.

25. What are the complications of induction of labor?

A infections, accidental hemorrhage/dry labor/rupture uterus, amniotic fluid embolism/fluid retention with pitocin/uterine hypertonus

Fetal complications prematurity/fetal distress/neonatal hyperbilirubinemia (more common with oxytocin).

25. Different complications on uterine contractions.

- A hypertonus single contraction lasting for > 2 minutes
- Tachysystole > 5 uterine contractions in 10 minutes time
- Hyperstimulation hypertonus or tachysystole is associated with abnormal fetal heart pattern.

26 Operative Delivery: Forceps and Ventouse

Shaila Sapre, Vrunda Joshi

FORCEPS

1. Who invented forceps? When?

A member of the Chamberlen family from Woodham Mortimer, Sussex U.K. invented forceps in the later part of 16th Century or the beginning of 17th Century.

The invention was preserved as a family secret through four generations, not becoming generally known until the early part of the 18th century.

However Palfyn in 1723, separately invented forceps.

2. How Did the present day forceps evolve?

Chamberlen forceps—Iron blades with handle.

Smellie of England—Added the English lock or double slot lock, and Shank, to increase the length of the instrument.

Leveret of Paris—Added the pelvic curve in 1747 and French lock with butterfly screw.

3. Indications for forceps application

1. Maternal
   - Maternal exhaustion/prolonged IInd stage of labor
   - Cut down the IInd stage in cardiac or pulmonary disease
   - H/O Spontaneous pneumothorax
   - H/O Heart disease
   - H/O severe anaemia
   - H/O detached retina.
2. Fetal
   a. Fetal distress in II stage of labor
   b. After coming head of breech (pipers forceps)
   c. LBW baby (controversial).

4. What is elective or prophylactic forceps delivery? Who coined this term?

The term was advocated by De Lee.

When interference with forceps is elective with the knowledge that it is not absolutely necessary but it prevents the maternal and fetal complications. Considered to be both fetal and maternal in indication.

5. What are the prerequisites for forceps delivery?

- The cervix must be fully dilated and taken up.
- Membranes must be ruptured.
- The head must be engaged. Station should be at +2
- Rotation should be complete.
- There should not be any disproportion between the head size and that of the pelvic inlet, midpelvis or the outlet. CPD should be ruled out.
- Uterus must be acting.
- Bladder and bowel must be emptied.
- Appropriate anaesthesia should be given. Local/pudendal block.
- Consent and proper documentation

6. What are the different parts of the forceps?

1. Handles
2. Locks—English, French, Sliding
3. Shank
4. Blades.

7. How will you decide the side of the blade?

The right or left blade is decided depending on the side of the maternal pelvis to which they are applied.

8. Why the forceps have different curves?

The forceps have 2 curves:

Cephalic curve—fits the shape of the baby's head and reduces the danger of compression.

Pelvic curve—follows the direction of the birth canal and makes application and extraction easier and also decreases the damage to the maternal tissues.

9. What are the different functions of forceps?

1. Traction—In primigravida the pull required is 18 kg.
   In multigravida about 13 kg.
2. Protection
3. Compression
4. Rotation (only in mid forceps, e.g. keillands)
5. Stimulation of uterine contractions
6. One blade may be used as vectis in delivery of the head in LSCS

10. What do you mean by trial forceps?

It is a tentative attempt of forceps delivery in a case of suspected mid pelvic contraction with a preamble declaration of abandoning it in favor of LSCS, if moderate traction fails to overcome the resistance. This should preferably be performed in operation theater.

11. What do you mean by failed forceps?

When a deliberate attempt in vaginal delivery with forceps has failed to expedite the process. It is called failed forceps. The causes are CPD, malposition or very early application of forceps before the head is low enough.

12. What are the complications of forceps application?

1. Maternal:
   Soft tissue trauma
   Episiotomy extension, IIIrd degree tear.
   Vaginal and cervical lacerations,
   Cervical tear with traumatic PPH
   Urethral/bladder injury
   Uterine Injury (Lus.)
   Rarely rupture of the uterus.
2. Fetal:
   - Transient facial forceps marks
   - Bruising, lacerations
   - Cephalhematomas
   - Facial nerve injury—due to pressure on the nerve as it comes out of the stylomastoid foramen. Less common are depressed skull fractures, Intracranial hemorrhage, tentorial lacerations.

13. Define outlet forceps.

a. The scalp is visible at the introitus without separating the labia.
b. The fetal skull has reached the pelvic floor.
c. The sagittal suture is in the anterorposterior diameter.
d. Fetal head is at or on the perineum.

14. Define low forceps.

Application of forceps when the leading point of the skull is at station + 2 or more.
Rotation 45 degree or less.

15. How do you name forceps?

A. Long curved forceps: High and mid cavity
B. Short curved forceps: Outlet forceps
   Perineal forceps
   Low cavity forceps

16. Names and variations of short curved forceps

Wrigley's
Simpson's

17. What are the advantages of forceps over vacuum?

1. Can be used in preterm baby.
2. Can be used in after coming head.
3. Quickly expedite the delivery in case of foetal distress.
4. Simplicity of the instrument, handy and low cost.

VENTOUSE

1. What are the indications of vacuum extractor?

1. Maternal— Maternal exhaustion
Maternal Heart disease

2. Fetal— Fetal distress in second stage of labour
Failure of descent or rotation

2. Different materials used for vacuum cups/and sizes

Materials:
1. Metal cups
2. Soft cops, silicon cup (silicone rubber or disposable plastic).

Sizes: 1. 30,40,50 and 60 mm.

3. What are the different contraindications of vacuum?

Absolute—CPD, face presentation, aftercoming head

Relative—partially dilated cervix, dead fetus

Congenital abnormalities like hydrocephalus, premature baby.

4. What are the advantages of vacuum over forceps?

1. Same amount of traction can be applied with only ½ the rise in intracranial pressure resulting from the use of forceps.
2. Can be used before full dilatation of cervix.
3. The foetal head can go through the rotation
4. Require less skill
5. Can be applied at high level of station
6 Maternal complications are less

5. How a chignon is formed in a case of vacuum?

After application of cup pointing towards the posterior frontanellae, slowly negative pressure is pumped up until it reaches 0.7 to 0.8 kg/cm2. This takes 8-10 min. As the development of the vacuum progresses, an artificial caput succedaneum called a "Chignon" is formed.

6. What are the conditions to be fulfilled before ventouse application?

A criterias are same as that for forceps.

7. When to abandon the traction in the ventouse application?

If no advancement during 4 successive uterine contractions, it is abandoned on no account, traction should exceed 30 min.

8. What are the complication of ventouse delivery?

Maternal—Soft tissue injuries to cervix, vagina, traumatic PPH

Fetal—sloughing of scalp, cephalhematoma, subaponeurotic hemorrhage intracranial hemorrhage.

9. What are the types of forces exerted by ventouse?

1. Negative outward suction from the vacuum itself.
2. Downward force from the traction
3. Circular force if rotation takes place.
4. Shearing force occurs when the direction of traction is not perpendicular to the surface of scalp.

27

Contraception

Haresh U Doshi

1. Which are the different methods for contraception ?

Methods of contraception: (A) Temporary, (B) Permanent

A. Temporary contraception
   1. Natural and behavioral family planning methods:
      a. Total abstinence
      b. Coitus interruptus
      c. Fertility awareness based methods
      d. Lactational amenorrhoea method.
   2. Barrier contraceptives:
      a. Mechanical:
         — Male-condom
         — Female-condom, diaphragm, cervical cap, dumas cap, etc.
      b. Chemical: Spermicidal substances in the form of foam tablets, creams, jellies, etc.
      c. Combined: Mechanical plus chemical.
   3. Intrauterine contraceptive devices (IUCD).
   4. Steroidal contraceptives: Oral contraceptives (OCs), injectables and others
   5. Emergency contraception—Postcoital contraception
   6. Uterotubal junction devices—sialistic or ceramic plugs
   7. Miscellaneous: Male pill—Gossypol
      Immunological (under research).

B. Permanent contraception
   1. *Male:* Vasectomy operation
   2. *Female:* Different sterilization operations (see Chapter 8).

2. Which are the natural and behavioral methods for contraception ?

The are as follows:
   a. Total abstinence
   b. Coitus interruptus
   c. Calendar or rhythm method : Record the number of days for previous 6 menstrual cycles. Subtract 18 from the length of her shortest cycle.

This is first day of her fertile period. Then subtract 11 days from the length of her longest cycle. This is the last day of her fertile period.

d. Cervical mucous (Billing's method): Here the fertile period starts from the first day of any cervical secretions or feeling of vaginal wetness until the 4th day after the peak day of slippery secretions.
e. Symptothermal method : At least 2 indicators are used to identify fertile period, i.e. BBT + Cervical mucous or BBT + Calendar rhythm.
f. High tech hormonal monitoring : Here small electronic devices detect urinary metabolites of LH and estrogen, estrone-3-glucuronide (E3 G). Threshold level of estrogen determines the beginning of the fertile period and 4 days past a threshold level of LH marks the end of fertile period. "Persona" and "Clearpan" are such personal hands held devices with test strips.

In last 4 methods which detect fertile period the couple avoids sex, uses a barrier method, or uses withdrawal during the fertile time.

g. Lactational amenorrhoea method : This is most useful when mother is doing exclusive breast feeding, she is not menstruating (first 6 months).

3. What is the history of condom? What precautions are taken while using condom ?

The origin of word "Condom" is believed to have come from the Latin word "Condom" meaning a receptacle. Its invention is also attributed to physician Dr. Condom who recommended it to king Charles II to prevent illegal offspring.

Condom should be applied on erect penis and prelubricated one is preferred. If there is no teat, some space pressed empty of air, is left at the end. During sexual act, after ejaculation has occurred one should be sure that condom does not get dislodged from the penis, i.e. penis should be withdrawn while still erect and condom should be held firmly at the root of the penis.

Chemical contraceptive (spermicidal jelly) used along with it gives extra protection and lubrication. Do not use lubricants made with oil. Most of them damage condoms. Store condoms in a cool, dark place, if possible. Heat, light and humidity damage condoms. Handle condoms carefully. Fingernails and rings can tear them. Do not unroll condoms before use. This may weaken them. Also, an unrolled condom is difficult to put on.

Check the condom for tear before throwing it away and if it has torn, use one of the emergency contraceptive method. Do not reuse the condom.

4. What are the advantages and disadvantages of condom ?

Advantages:

1. Easy to use.
2. Relatively cheap.
3. Freely available without medical supervision and prescription.
4. Failure rate is low as compared to physiological and chemical methods.
5. Protects against common vaginal infections including human immuno-deficiency virus (AIDS) infection.

6. Protects against carcinoma cervix. Because carcinoma cervix is promoted by sexually transmitted agent. Suggested agents are: (i) Human papilloma virus, (ii) herpes virus, and (iii) sperm.
7. Safe. No hormonal side effects.
8. Can be used at any age.
9. Often helps prevent premature ejaculation.
10. Can be used where pills and IUCD are contraindicated, i.e. follow up of vesicular mole, diabetes, valvular heart disease.

Disadvantages:

1. Failure rate is high as compared to IUCD and pills.
2. Because it prevents full genital contact, it may decrease sensation making sex less enjoyable for either partner.
3. Couple must take some time to put the condom on the erect penis before sex. In male partner rarely it causes psychological disturbances and even impotence.
4. Disposal is a social problem, it may embarras some to buy condoms.
5. Hypersensitivity reaction to either of the partners.

5. What is the failure rate of condom ? Why it is high ?

Failure rate of condom is 3-18/100 women years observation (3-8/HWY if used with chemical contraceptive). This is also known as pearl index .

Causes of failure are:

1. Incorrect use.
2. Inconsistent use. Not used with each sexual act, e.g. patient under effect of alcohol.
3. Used without chemical contraceptive.
4. Defect in condom.
5. Tearing or bursting of condom during sexual act (4%).

6. Which are the other uses of condom ?

Other uses of condom:

1. In transvaginal sonography it is applied over the vaginal probe.
2. For preparation of mould in vaginoplasty.
3. Immunological cervical factor in infertility. Husband should use it for 3 to 6 months so that antibody level against the sperms in cervical mucous decreases. Then chances of pregnancy increase.
4. Threatened abortion—After few weeks of abstinence if couple resumes sexual relations. Male partner should use condom, because semen contains prostaglandins which may cause abortion.

7. Describe female condom.

Female condom is a newly developed female barrier contraceptive. It has combined features of diaphragm and condom. It consists of two flexible polyurethane rings located at the either end of a 15 cm soft loose fitting polyurethane sheath. The inner ring is placed high in vagina, while outer ring covers the labia and base of penis. It is prelubricated with silicone based lubricant. It is available with different names, e.g. reality, femidom, femshield.

Advantages

1. Controlled by woman.
2. It prevents STDs more effectively than condom as it covers some perineal area also.
3. No allergic reactions.
4. More convenient than male condom as it can be inserted precoitus.
5. Less chances of breakage.

Disadvantages

1. Expensive at present.
2. Some women have difficulties in insertion.

It is meant for single time use, however WHO recommends reuse foe maximum 5 times with proper care for disinfection, washing and drying.

Its failure rate reported in various studies range from 6.8 to 15.1/HWY in first year of use.

8. Why female condom is better than male condom?

Female condom is better than male condom because:

1. Controlled by woman.
2. It covers some perineal area also.
3. More convenient to use as it can be inserted precoitus.
4. Less chances of breakage
5. Reusable for few times

9. What are the contraindications, advantages and disadvantages of vaginal diaphragm?

Contraindications

1. Cystocele or rectocele.
2. Uterine prolapse.
3. Local infection, ulceration.
4. Allergy to rubber.
5. Severe retroversion.
6. VVF or RVF.

Advantages

1. Relatively cheap.
2. Effective as compared to physiological and chemical methods.
3. Used by female so cooperation of male partner is not required.
4. It does not interfere with natural coitus or orgasm of either of the partners and it is relatively harmless.
5. It also gives some protection against PID.

Disadvantages

1. In sensitive patients it may cause embarrassment and some women may consider diaphragm unaesthetic. Use of spermicide is messy.
2. Failure rate is high as compared to IUCD and pills.
3. High degree of motivation of the patient is required for its use.
4. Allergic reactions to rubber may occur.
5. Vaginitis, UTI and rarely toxic shock syndrome are reported.

10. Tell everything about "Today"

Today was available a vaginal sponge made of polyurethane containing one gram of the spermicidal nonoxynol-9. The sponge was shaped like mushroom cap with the concave side covering the cervix. It is 2" in diameter, 1.25" thick and loop is attached to the bottom. It has 3 actions:

1. It releases spermicide during coitus.
2. It absorbs the ejaculate.
3. It blocks the entrance to the cervical canal.

It was effective for 24 hours regardless the frequency of coitus, but then it is discarded. It releases 125-150 mg nonoxynol in 24 hours Its failure rate 9 to 27 per 100 users.

Today is now available in small pessary form 'Today premium' containing 5% Nonoxynol - 9. It dissolves within 10 minutes and is effective for one hour only. Its efficiency rate is more than 99%.

Nonoxynol has bactericidal and virucidal activity so it protects against AIDS. Usually, there are no major side effects. It may produce allergic type reactions and vaginal or penile irritation. Patient may have vaginal discomfort like soreness, itching, stinging. Rarely toxic shock syndrome can occur.

11. Which are the different IUCDs you know ?

Grafenberg ring, a silver coiled wire ring was the first popular IUCD widely used in the past. It was introduced by grafenberg of Germany in 1929. Since then many different types of devices are invented. They are divided into two groups.

a. First generation or inert or unmedicated devices, e.g. Lippes loop, Saf-T-coil
b. Second generation or bioactive or medicated devices containing metals like copper (Cu-T, Cu-7,) zink, silver or containing hormones, e.g. Progestasert, Mirena or containing drugs like tranexamic acid or epsilon aminocaproic acid.

Various IUCDs were used in past, e.g. Grafenberg ring, Chinese ring, Ota ring, Birnberg bow, Margulies coil, soonawala loop, Saf-t-coil, Dalkon shield, Hall-stone ring, Antigon-F, Dana super.

12. What is the difference between Cu-T 200B and Cu-T 380A ?

Copper T (Cu-T 200B) is T shaped device made up of polyethylene which is a biologically inert plastic. It is impregnated with barium sulphate to make it radiopaque. It has 2 monofilament polypropylene threads attached to its lower end. Copper wire is wound around the vertical limb. Surface area of copper is 200 sq. mm, weight is 120 mg and diameter of wire is 0.25 mm. It daily releases 50 μg of copper in first year. Its applicator, made up of synthetic plastic consists of cannula with guard and a plunger rod. Guard is blue and mobile. Cu-T along with its applicator is supplied in a plastic pack presterilized by gamma radiation. Cu-T 200 B has a small ball at its lower end to prevent cervical perforation.

Specifications
Length—36 mm, width—32 mm, weight—120 mg.
Surface area of copper—200 sq mm, diameter—0.25 mm.

T Cu-380A is a T shaped device with 2 solid copper sleeves on transverse arms and copper wire on vertical stem. Total surface area of copper is 380 sq mm (314 sq mm of copper wire and 33 sq mm of each copper sleeve). It has 2 monofilament white threads. Its effective life is estimated to be 6-10 years in T Cu-380 Ag copper wire on the vertical stem has a silver core. The silver core prevents fragmentation of copper and lengthens the effective life of the device.

13. Describe the time and technique of IUCD insertion.

Time of insertion:

1. During menstrual period from 2nd day onwards or within 10 days of menstruation (During menses it is easy to insert and bleeding related to insertion is masked).
2. Immediately after first trimester MTP or spontaneous abortion.
3. Postpartum—After normal or preterm delivery it can be introduced immediately or at the end of 6 weeks. In fact if insertion is done within first 48 hours there are less chances of perforation.
4. At the time of cesarean section.
5. Postcoital—within 5 days of unprotected intercourse in a fertile period.

Technique of IUCD insertion— 'Withdrawal technique':

- Patient is explained about the type, principles, side effects and failure rate, etc. of the device and informed consent is taken.
- Detailed history is elicited and complete pelvic examination is done to rule out contraindications.
- Full aseptic and antiseptic precautions are observed.

Guard is adjusted at the total uterocervical length of that patient after measuring the uterocervical length by sound. Loaded applicator is introduced so that when guard is at external os, tip of the applicator with Cu-T is at fundus. Now plunger is fixed by one hand and cannula is withdrawn over it, so Cu-T is released high up at fundus without being pushed. Then plunger is withdrawn, cannula is withdrawn and threads are cut for 2 to 3 cm from external os so that patient can easily feel it by self examination.

14. What are the contraindications to IUCD insertion?

Contraindications

Absolute

1. Abnormal menstruation—irregular heavy or prolonged.
2. Suspected pregnancy.
3. Recent or past pelvic infection.
4. Suspected malignancy of genital tract.
5. Uterine anomalies, e.g. bicornuate uterus.
6. Uterine pathologies, e.g. fibroid.
7. Bleeding disorders.
8. History of ectopic pregnancy.

Relative

1. Nulliparous patient: Difficult to insert, expulsion rate is high and if infection occurs it can lead to infertility.
2. Previous cesarean section or other scar on uterus.
3. Primary dysmenorrhea.
4. Local infection, i.e. cervicitis, vaginitis.
5. Moderate or severe anemia.
6. Patients on corticosteroid or anticoagulant therapy.
7. Recently terminated vesicular mole.
8. Valvular heart disease, diabetes.
9. Wilson's disease.

15. What is the mechanism of action of Cut 380 A ?

Mechanism of action:

It works primarily by preventing fertilization contrary to the popular belief that it prevents implantation.

1. Presence of device with its nylon thread occupies some space in the uterus and causes mechanical obstruction to ascent of sperms.
2. Transcervical threads cause changes in the cervical mucous and make it hostile to sperms.
3. IgM levels in serum is increased so antifertility action is in part to their ability to produce antibodies.
4. Actions of copper:
   - Directly damages sperms and fertilized ovum.
   - Causes biochemical changes in cervical mucous and renders it hostile. Sperm motility and capacitation are affected.
   - Changes in the endometrium. (a) Enzymatic inhibition, i.e. Carbonic anhydrase (copper replaces zink), Alkaline phosphatase, glycogen synthetase, etc. (b) Intense leukocytic infiltration. (c) Changes in DNA constituent of endometrial cells. (d) Decrease in glycogen content of cells. (e) Increase in fibrinolytic activity of endometrium and (f) Endometrial vasoconstriction and ischemic damage.
5. Increase in tubal motility so fertilized ovum, before it is mature enough for implantation, reaches the uterus which is also unprepared.
6. Increase in uterine contractions which result in expulsion of newly implanted ovum.
7. Device causes low-grade tissue reaction (i.e. inflammatory changes) in the endometrium making it unreceptable for implantation. It also attracts macrophages to its site which engulf the sperms as well as fertilized ovum by phagocytosis.

16. What are the side effects and complications of IUCD ?

Side-effects and complications

1. Menstrual problems—Spotting, heavy periods and prolonged periods frequently occur in first 2-3 months.
2. Cramps like pain in lower abdomen. Removal due to excessive bleeding and pain occurs in 5-15/100 users.

3. Dysmenorrhoea—Spasmodic type, with infection there can be congestive type also.
4. Leukorrhea.
5. Pelvic infection—Risk is more in first month of insertion. Ascending infection can occur at any time as nylon threads constantly project in vagina. Flaring up of already existing infection can also occur.
6. Displacement —Device may penetrate the uterine wall or perforation of uterus and migration into pelvis or upper abdomen. Perforation commonly occurs at the time of insertion. Common sites of perforation are fundal and isthmic region. Incidence of perforations is 1 to 3 per 1000 insertions.
7. Spontaneous expulsion—During first menstrual period or within the first year of insertion. The rate is 5-15/1000 insertions.
8. Failure, i.e. pregnancy (discussed later).
9. Ectopic pregnancy—5% of pregnancies with IUCDs are ectopic, but as the failure rate is very low, overall there is protection as compared to no contraception used. Risk is more with progestasert.
10. Fracture or breaking of IUCD and embedding in the endometrium resulting in decreased efficacy and difficulty in removal. Removal due to excessive bleeding and pain occurs in 5-15/100 users.
11. Rarely fainting and collapse at the time of insertion, if patient is not properly motivated.

17. What advises are given to the patient after IUCD insertion ?

Advice to the patient

- Patient is taught to feel the thread by self-examination and should regularly check the thread and particularly after the first menstrual period.
- She is informed about possible initial reactions : increased bleeding, pain for first 2-3 month, etc. They are treated symptomatically. They gradually disappear with time.
- She should come for follow up after first menstrual period and then yearly.
- She should consult the doctor immediately if she misses a period, it she develops any complications, or if she does not feel the thread.
- The device should be replaced, when time limit is over.
- She should not use intravaginal sanitary measures (menstrual tampons) for few menstrual periods.

18. How will you compare other methods with IUCDs ?

Advantages of IUCD over other measures:

1. Simplicity in insertion.
2. No loss of time.
3. Return of fertility is immediate on removal of device.
4. Cheap (supplied free by Govt.)
5. Does not interfere with sexual act.
6. No systemic side effects.

7. Less failure rate (except pills).
8. Long-term—lasts for 10 years.
9. No effect on breast milk, can be inserted immediately after child birth.

Disadvantages

1 Side effects: As mentioned before.
2. Does not prevent HIV/STDs.
3. Patient cannot use on her own or stop on her own. Some medical help required for insertion and removal.
4. May come out without women's knowledge.
5. Male partner can feel the strings rarely.

19. What are the other uses of Cu-T ?

Other uses of Cu-T are:

1. After breaking of adhesions in a case of uterine synechia (Asherman syndrome) to prevent refusion of walls. Cu-T after removal of copper wire is used.
2. After Straussman operation—to prevent fusion of anterior and posterior walls.

20. How will you manage the patient who does not feel the thread ?

Management of a patient who does not feel the threads:

Possibilities

1. Mistake of the patient.
2. Indrawing of threads inside the uterine cavity or cervix, or it is torn and expelled out.
3. Perforation of uterus and migration in the peritoneal cavity.
4. Spontaneous expulsion.
   - Do per speculum examination, if the thread is seen everything is all right and make the patient to palpate it.
   - If thread is not seen, USG (TVS) is done, if the device is inside the uterus it is easily seen on USG. But if it has migrated in the peritoneal cavity it is not seen on USG. Take simple X-ray abdomen, if device is not seen anywhere in the X-ray, it is expelled out.
   - But if it is seen in the X-ray it is displaced device.
   - IUCD whether it is intrauterine or extrauterine can also be checked by one of the following: (1) A-P and lateral X-ray (2) A-P X-ray and X-ray with sounding after moving the uterus and (3) HSG.

Treatment: Any displaced IUCD requires removal either by laparoscopy or laparotomy. Medical devices cause adhesions and ring shaped cause intestinal obstruction, loop is relatively inert but it also should be removed as patient may be disturbed psychologically.

If it is intrauterine it can be removed by different instruments like simple curette, long artery forceps, uterine dressing forceps, shirodkar hook or with the help of hysteroscope. It may be badly embedded.

21. How will you manage patient having pregnancy with Cu-T *in situ* ?

Management of patient having pregnancy with Cu-T in situ:

Advice should be termination with removal of Cu-T because of following risks: Increased risk of abortion, septic abortion, premature rupture of membranes, premature labor, IUGR, accidental hemorrhage, puerperal sepsis and ectopic pregnancy. But it does not produce any congenital malformation.

With all risks explained, if patient is keen to continue her pregnancy at least Cu-T should be removed-If uterus is less than 12 weeks, if threads are seen and if device can be removed easily without disturbing the pregnancy. Otherwise leave it. CuT may be retrieved at the time of delivery.

22. Tell something about other IUCDs you know ?

Other IUCDs

1. **Multiload-Cu 250 (or Multiload-Cu 375):** It is made of mixture of high density polypropylene and ethylene vinyl acetate copolymer impregnated with barium sulfate. Its shape is different from Cu-T, i.e. side arms of T are bent with small outer projections (Serrated fins) which help to hold the device in place without stretching the uterine cavity. Copper wire with 250 sq. mm (or 375 sq mm) surface area is wrapped around vertical arm. Both are 36 mm long and 21 mm wide, but Multiload Cu-375 has copperwire of 0.4 mm diameter Effective life recommended for Cu-250 is 3 years while that of Cu-375 is 5 years. Its inserter has no plugner and it is introduced by withdrawal technique. It has 2 nylon threads at its lower end, Perforation and expulsion is less with multiload as compared to Cu-T.
2. ***Cu-7 (Gravigard):*** It is "7" shaped device made of polypropylene impregnated with barium sulfate. Copper wire is wrapped around vertical arm. Surface area of copper wire is 200 sq. mm. It is 36 mm long and 26 mm wide. It has only one (instead of usual two) polypropylene thread blue in color. Its proved life span is 3 years. It is inserted by withdrawal technique.
3. **T Cu-220:** It is T shaped device with 7 solid copper sleeves, 2 on transverse arm and 5 on vertical stem. Total exposed surface area of copper is 220 sq.mm. It is developed by Population Council and its effective life is estimated to be 15-20 years. It has 2 threads and it is introduced by withdrawal technique.
4. **Nova-TCu-200 and 200 Ag:** It is T shaped device made of polyethylene with barium sulfate impregnated. The fine copper wire is wrapped around the vertical stem. The surface area of copper is 200 sq.mm. In 200 Ag variety a silver core has been added to the copper wire to reduce its fragmentation. It has 2 white threads and it is inserted by withdrawal technique. As compared to other copper devices it is slightly short (32 mm long instead of 36 mm).
5. **Zicoid:** It is T shaped devised with flexible and resilient side arms with small projection on the inside of the side arms. This prevents irritation of the cervical canal during insertion. It's surface area is 350 sq. mm. and copper wire is placed in the upper part of the stem. Diameter of wire is 0.5

mm. Plunger with special stop at proximal end ensures high fundal placement of the device. Failure rate is 0.2/HWY. It is effective for 5 years.

6. **Flexigard:** It is a frameless device which has a nylon 2 suture with which it is anchored to the uterine musculature. There are 6 copper sleeves on the nylon thread. Total surface area of copper is 390 mm^2. Device is flexible because the sleeves are not connected to each other. It is effective for 5 years.

23. What do you know about progesterone containing IUCDs ?

Progesterone containing devices :

a. **"Progestasert"** T shaped device, made up of ethylene vinyl acetate copolymer. Barium sulphate impregnated. It has two monofilament threads black in color. It has its special inserter without a plunger and introduced by modified withdrawal technique. Contains 38 mg progesterone in vertical arm, dispensed in silicon oil and daily releases 65 µg progesterone. Progesterone has added contraceptive effect. It causes decidual changes in the endometrium and glandular atrophy which interfere with normal reproductive process. It is useful where use of another device has caused excessive bleeding or cramping which may be relieved by progestasert, but spotting is increased. Incidence of ectopic pregnancy is increased with it. Unlike Cu-T it is to be replaced every year.

b. **LNG 20: (Mirena, LNG—Intrauterine system)** T shaped device with flexible arms. The shape is that of Nova T but it has capsule on the stem. The core of the capsule contains a mixture of silicone rubber and 60 mg levonorgestrel, which is released at 20 µg/day. It is 32 mm long and 32 mm wide. Its inserter is withdrawal type and it is effective for 5 (possibly 8) years. Apart from contraception it helps in treatment of DUB and Fibroids.

24. Which are the different types of steroidal contraceptives ?

The different types of steroidal contraceptives types:

1. Oral—Commonly known as PILLS
2. Injectable—Intramuscular
3. Newer sustained release systems.

Oral Pills are of different types

1. Combined
2. Phasic
3. Minipill
4. Newer pills.

(Sequential pill withdrawn from the market because of high incidence of endometrial carcinoma and thromboembolic complications).

25. What do you know about combined oral pills?

Combined oral pill

– They are combination of estrogen and progesterone-21 such tablets.
– Supplied in a pack of 28 tablets, where last 7 tablets are placebo containing iron and vitamins, to complete the menstrual cycle.

- Newer pack is started immediately after first pack is over, irrespective of onset or stoppage of menstruation.
- In a Family Planning Program, it is supplied free of charge by Government: Mala- D, Mala-N.
- Commonly used market preparations are Ovral, Ovral-L, Duoluton L, Novelon, etc.

Preparation	Estrogen	Progestogens
Mala D	30 µg	Norgestrel 0.3 mg
Mala N	30 µg	Norethisterone 1.0 mg
Ovral	50 µg	Levonorgestrel 0.25 mg
Ovral L	30 mg	Levonorgestrel 0.15 mg
Duoluton-L	50 µg	Levonorgestrel 0.25 mg
Novelon	30 µg	Desogestrel 0.15 mg
Femilon	20 µg	Desogestrel 0.15 mg

26. What do you know about phasic pills and mini pill ?

Phasic Pills.

Biphasic or Triphasic.

1. Biphasic-All 21 tablets contain E + P but dose of progesterone is doubled after first 10 days, dose of estrogen remaining constant (Ethinyl oestradiol 35 µg. Noroethindrone 50 µg and 100 µg). It has slightly high failure rate and not popular in India.
2. Triphasic-All 21 tablets contain E + P but dose vary in 3 phases, e.g.

a. Triquilar:

Ethinylestradiol	Levonorgestrel	Days
30 µg.	50 µg	1-6
40 µg.	75 µg	7-11
30 µg.	125 µg	12-21

Triquilar is started from the 5th day of cycle while second pack is started after 7 days of pack free interval.

b. "Cyclessa" contains ethinyl estradiol 25 µg and triphasic desogestrel and "Tricycen" contains EE 25 µg and triphasic norgestimate. They are not yet available in India.

Mini pill

- Contains only progesterone, i.e. Norgestrel 75 µg or Norethisterone 350 µg or Levonorgestrel 30 µg.
- Estrogen free pill now available in India is Cerazette containing 75 µg desogestrel.
- To be taken daily one throughout the cycle.
- It inhibits ovulation and also acts locally by making endometrium unreceptable for implantation and renders cervical mucous hostile to sperms.
- Failure rate is comparable to combined OC pills.
- It avoids undesirable side effects of estrogen found with combined pills and sickle cell patients or lactating mothers can take it.

– Menstrual irregularities and amenorrhoea are common with its use.
– Side effects contributed to progesterone content can occur, i.e. alopecia, loss of libido, weight gain, nervous irritability. Androgenic side effects are less with cerazette.

27. Which are newer pills now available ?

Newer pills:

- **Femilon**: It contains only 20 µg ethinyl estradiol (as compared 30 µg in Novelon) and 150 µg Desogestrel. Due to lowest dose of estrogen, estrogen related side effects like nausea, breast tenderness and headache are very low.

  Desogestrel a newer progestogen which has minimal androgenic side effects: reduces the risk of cardiovascular disease and cures acne and hirsutism.
- **Postcoital pill**: Discussed in details under emergency contraception.
- Pill with antiandrogenic effects : **"Diane 35"** contains ethinyl estradiol + cyproterone acetate and **"Yasmin"** contains EE + Drospirenone (aldosterone derivative). Due to antiandrogenic effects they are useful in patients with once and hirsutism. Yasmin due to its antimineralocorticoid activity also decreases BP and weight in the users
- Once a month pill: Each pill contains 3 mg of Quinesterol (long-acting oestrogen) and 12 mg megesterol acetate (Progesterone). It is not available in India.
- Pills with extended cycle length – Seasonale is monophasic pills (EE + LN) for 84 days with one week off.
- Nonestrogen combined pill: Containing RU-486 (mifepristone) in first half of the cycle and progesterone (MPA) in second half of the cycle is under study.

28. What is the mechanism of action of Pills?

Mechanism of action: Pills containing oestrogen and progesterone work in following ways.

1. Their main action is inhibition of ovulation. They act at hypothalamic level inhibiting release of gonadotropin releasing hormone (GnRh). Secretion of FSH and LH from pituitary are thus suppressed, so there is no follicular growth. As there is no LH surge, ovulation is suppressed. It is also suggested that they directly inhibit pituitary as there is no response to exogenous GnRh administration in pill users.
2. They alter the maturation of endometrium making it unsuitable for implantation. Under the effect of progesterone there is stromal edema with glandular exhaustion and atrophy.
3. Progesterone makes cervical mucous thick viscid, scanty and impermeable to sperms.
5. Effect on tubal motility by progesterone is also suggested.

29. What are the contraindications to Pills ?

Contraindications

Absolute

1. Thromboembolic disease.
2. Acute or chronic disease of liver.
3. Cancer of breast or genitals.
4. Pregnancy known or suspected.
5. Focal migraine.

Relative:

1. Hypertension
2. Valvular heart disease
3. Epilepsy
4. Smokers over 35 years
5. Nursing mothers
6. Sickle-cell anemia, thalassemia
7. Asthma
8. Allergic disorders
9. Herpes gestationalies
10. Porphyria
11. Pruritus of pregnancy
12. Varicose veins
13. Obesity
14. Elective surgery
15. Diabetes
16. Crohn's disease.

30. Which are other uses of pills ?

Other uses of pills:

1. Regularization of cycles.
2. DUB.
3. Endometriosis.
4. Postponement of menstrual period.
5. Amenorrhea to test for withdrawal bleeding.
6. Primary dysmenorrhea.
7. Suppression of functional ovarian cysts.

31. What are the side effects and complications of pills ?

Side effects and complications:

1. **Nausea and vomiting** It is very common side effect. It appears to be related to the estrogen content of the pill. It is managed by: (i) reassurance that it subsides as therapy continues (ii) taking the pill after food or with cup of milk (iii) taking the pill at bedtime so that the individual is sleeping during peak concentrations of drug in the blood (iv) changing over to another compound and (v) reducing the estrogen content of the pill.
2. **Break through bleeding** It occurs in almost half of all patients at some time. It may be due to failure to take the pill at the same time each day. It

is more common with low dose pill. However with newer pills cycle control is good. It diminishes after first 3-4 pill cycles.

Management: Double up of pills in an attempt to stop the bleeding and discontinuing the pills to restart them on the fifth day of bleeding are now considered ill-advisable. Following measures should be taken

1. Switching to another low dose compound
2. Increase in the estrogen dose of the pill temporarily or taking short course of additional estrogen temporarily.
3. A search for pathological cause of bleeding must be made.

3. **Absence of withdrawal bleeding (Pill amenorrhoea):** As high as up to 22% of women experience it at some time.

   Management: After ruling out the pregnancy either of the following options is given to the patient:

   1. Reassuring the patient of the benign nature of the amenorrhea and the likelihood of regular menstruation when the pills are discontinued and second pack is started from the seventh day of last pill
   2. If withdrawal bleeding is desired switching to a pill with higher estrogen activity or adding tablet of estrogen, i.e. EE 0.02 mg last 7 days for few cycles is advised. Amenorrhea and oligomenorrhea are less common with triphasic pills.

4. **Hypertension:** It is 3 times more common in pill users. Both systolic and diastolic BP rises. It is minimal with triphasic pills.
5 **Cardiovascular diseases:**
   a. Venous thromboembolism—Deep vein thrombosis is 5-6 times more common in users, while superficial vein thrombosis is 2-3 times more common in users. It is due to estrogen and it is dose dependent.
   b. Myocardial infarction—It is 2-3 times more common in pill users. It is due to both estrogen as well as progesterone. The incidence is now decreasing with greater care in selecting the patients and due to low dose pills.
   c. Cerebrovascular stroke—Cerebral thrombosis is more common than cerebral hemorrhage. It is not dose dependant so it is not decreased with low dose pills.
6. **Weight gain, fluid retention:** 5-50% of women gain weight. Both estrogen and progesterone are responsible and tendency is more in first 6 months of use. Weight gain is negligible with low dose pills.
7. **Breasts:** Pain, tenderness, engorgement are common side effects
8. **Carbohydrate metabolism:** Glucose tolerance is impaired in pill user, latent diabetes may become symptomatic. This is less with low dose pill.
9. **Liver and gall bladder:** Altered liver functions noted in pill users. Cholecystic jaundice can also occur. Gall stones and infection of gall bladder are found in young women on pills.
10. **Post-pill amenorrhoea:** It is found in 3-5/1000 users. It is not related to duration of use and it is common in women having oligomenorrhea before starting the pill.

11. **Neoplasia:**
    a. Cervix—There is higher incidence of dysplasia and ca *in situ* cervix reported in pill users. Little increase in the risk of cervical cancer is reported in women who use OCs for > 5 years.
    b. Breast—Risk of breast cancer is not convincingly proved. It is very slightly increased only in young women, nulliparous patient, after prolonged use and with positive family history.
    c. Liver—For hepatic adenoma (benign) there is unequivocal evidence. The risk is rare (3-4/lakhs) but condition may be fatal due to severe, intraperitoneal hemorrhage. With long term use (8 years or more) there is a risk of hepatocellular carcinoma.
    d. Malignant melanoma-controversial.
12. **Teratogenicity:** If pregnancy occurs during the course of pill or immediately after stopping the pill, there is a very small but definite risk to the fetus.
13. **Miscellaneous:** These include, melasma, headache, giddiness, leukorrhea, acne, change in libido, mental depression, alopecia, leg cramps and some ophthalmic complications like retinal vascular disease, corneal edema or diseases of eyelids and conjunctiva.

32. Which drugs causes drug interaction with OC pills?

They are Rifampicin, Penicillin, Tetracycline, Ampicillin, Barbiturates, Phenytoin, Sulfonamides.

33. How will you manage forgotten pill ?

It patient forgets to take one tablet white (hormonal) she should take it as soon as she remembers and 2nd pill is taken as usual. If she misses for consecutive two days. Then for 7 days she should avoid sex or use condoms. If 7 or more white tablets are left in the pack, she should take all the rest of the pills as usual. But, if < 7 tablets are left in that pack, take rest of the white pills as usual, Don't take brown tablets and start a new pack after the last white tablet of previous pack. She may miss a period. This is okay.

Missing of 1 or more brown pills is of no importance, take the rest of the pills as usual, one each day.

34. What are the beneficial side effects of Pills ?

Beneficial side effects:

1. Menstrual comforts: Regularization of cycle, blood loss is controlled (cure of anemia), relief from primary dysmenorrhea and premenstrual tension.
2. Prevents pelvic inflammatory diseases: Cervical mucous is thickened, menstrual blood loss is reduced, uterine contractions are inhibited (so spread of infection to tubes prevented). Prevents pregnancy and abortion which provide opportunity for infection.
3. Prevents ectopic: As it is highly effective contraception method and also as it prevents PID (except mini pill).
4. Lowers risk of endometrial cancer: Because of progesterone component.

Upto 50% reduction in risk of endometrial cancer and protection persists upto 20 years after discontinuation.

5. Lowers risk of ovarian cancer: Because of suppression of pituitary secretion of Gonadotropins and ovulation. There is 40-80% overall decrease in risk of ovarian cancer among users. Protection lasts for 15 to 20 years after discontinuation.
6. Protection against benign breast diseases. 30-50% decrease in the incidence in current and recent long term users.
7. Protection against functional ovarian cysts, i.e. Follicular cysts, granulosa lutein and theca lutein cysts due to suppression of ovulation (except multiphasic preparation) 50 to 80% reduction.
8. Some studies have shown reduced risk of uterine fibroids, colorectal cancer, rheumatoid arthritis and benign thyroid conditions. However this is not unequivocally proved.
9. Improvement in bone mineral density also occurs.

35. What do you know about non steroidal oral contraceptive pill ?

Centchroman (centron, Saheli) is nonsteroidal oral contraceptive pill. It exhibits weak estrogenic and potent antiestrogenic (5 times) activity. It is started in a dose of 30 mg (1 tablet) on the first day of menstruation, given twice a week for the first 3 months and then once weekly. It is safe and reliable and with correct use its failure rate (1.6 HWY) is comparable to OC pills.

It has no effect on HPO axis, so it does not inhibit ovulation. The contraceptive mechanism is local either by defective implantation of blastocyst due to abnormal decidualization or blastocyst abnormality itself. Added effects are antestrogenic effects on altered tubal motility and poor cervical mucous sperm penetration. Return of fertility is reported within 6 months of discontinuation.

As it is nonsteroidal all the major side effects of steroidal contraceptives are avoided. Menstrual disturbances like oligomenorrhea and amenorrhea are found in > 1/3rd users. Because of these side effects it could not gain much acceptance. It has been successfully used in treatment of DUB. Clinical trials are on for its use in treatment of breast cancer and for HRT.

36. Which are the injectable steroidal contraceptives available ?

Injectable contraception are as follows:

A. Long acting progestins:
   1. Depot-medroxy progesterone acetate **(DMPA)**—150 mg every 3 months (Depo-provera).
   2. Norethisterone enanthate **(NET-EN)**—200 mg every 2 months (Noristerate).

B. Combined injectables (oest + prog.):
   1. DMPA 25 mg—estradiol cypionate 5 mg (Lunelle, Cyclofen)— monthly.
   2. NET-EN 50 mg + estradiol valerate 5 mg (Mesigyna)—monthly.
   3. Dihydroxy progesterone acetophenide 150 mg + estradiol enanthate 10 mg (Deladroxate) – Monthly.

37. What is DMPA ? What are the advantages and disadvantages of DMPA?

DMPA

- It is highly effective, long acting, safe, reversible injectable contraceptive .
- It is given as 150 mg I/M every 3 months with a grace period of 2 weeks late or early.
- It should be given deep I/M in gluteus or deltoid muscle with all aseptic precautions. Injection into the fat (decreases absorption) or rubbing the injection site (increases absorption) changes the efficacy.
- Time: It is given within 7 days of menstrual cycle. It can also be given immediately after abortion and MTP. In Postpartum patient also it can be given immediately within 7 days, however in breast feeding patient one can wait for 6 weeks postpartum.
- Failure rate is very low and it is < 1/100 women years.
- Mechanism of action is same as that of OC pills and it also hinders the rate of ovum transport.
- Return of fertility after DMPA is about 9 months after the last injection (i.e. 5.5 months after the effect of last injection is over). There is no longterm impairment of fertility.

Advantages

- Very effective.
- Long-term—3 monthly injections.
- Does not interfere with sex.
- Reduces the incidence of PID and conidial vulvovaginitis.
- Flexibility in return visit due to 2 weeks grace period.
- Can be used at any age.
- No estrogen related side effects.
- Makes sickle cells crisis less frequent and less painful.
- Decreases seizure frequency in epileptics.
- Can be used in nursing mothers. It increases milk production.
- Helps in preventing endometrial cancer and uterine fibroids.

Disadvantages

- Delayed return of fertility—7 to 9 mths after last injection.
- Menstrual irregularity and ammenorrhoea occurs in 10 to 15% of women.
- Weight gain of 1-2 kg each year may occur.
- Decrease in HDL and increase in LDH cholesterol is reported so women with sever vascular disease should not use it.
- May cause headache, breast tenderness, moodiness, nausea, hair loss, less sex drive.

38. What is Norplant ?

Norplant

- The norplant contraceptive is a long-acting, low-dose, progestin only contraceptive system for women. The drug is delivered by means of 6 silastic capsules implanted subdermally in the woman's arm employing a minor surgical technique. It is highly effective and highly reversible.
- Previously available in 6 capsules (36 mg each) Norplant-2 rod system comprises 2 silastic rods containing a total of 140 mg of levonorgestrel (70 mg/rod). The rods are implanted subdermally in the women's arm employing a minor surgical technique.
- Time: Within 7 days after the onset of menstruation or immediately after abortion or first trimester MTP. The implants become effective within 24 hours of placement.
- Mechanism of action is mainly local on cervical mucous and endometrium but it also inhibits ovulation in about 50% of the cycles.
- Failure rate: Less than 0.5 per hundred women years.
- Contraindications: Anticoagulant therapy, undiagnosed abnormal uterine bleeding, known or suspected pregnancy, hemorrhagic diathesis, liver diseases.
- Side effects: Bleeding disorders, amenorrhoea, nausea, loss of appetite, dizziness, headache, changes in libido, depression, acne, infection at implant site.
- Removal: Implants work upto 5 years, after that they should be removed employing a minor surgical technique.

39. What is Implanon ?

Implanon It is newer implant which consists of an ethinyl vinyl acetate copolymer device (single rod—4 cm long , 2 mm in diameter) containing 68 mg of progestogen 3-ketogestral also known as etonogestral. It releases 30-40 µg of drug/day. It is effective for 3 years. Other single rod devices are Nestorone for 2 years and Uriplant (nomegestrol acetate) for 1 year and ST-1435 .

40. What do you mean by biodegradable devices ?

Biodegradable devices: They deliver progesterone from a carrier that gradually dissolves and disappears. Compared with norplant they are easier to insert and need not be removed. Two are under trial—Capronor (40 mm rod) containing levonorgestrel (effective for 1.5 years) and Norethindrone pellets (1 year).

41. What do you mean by emergency contraception ?

Emergency contraception means a particular type of contraception that is used as an emergency procedure to prevent pregnancy following an unprotected but possibly fertile intercourse.

Indications for emergency contraception include unprotected intercourse, failure of barrier method, unsuccessful withdrawl (coitus interruptus), missed oral contraceptive pills and sexual assault.

42. Which are the different methods of emergency contraception ?

Methods

1. Yuzpe method: It was introduced by a Canadian physician Albert Yuzpe in early 1970s. Combined oral contraceptive pills containing ethinyl estradiol 50 ug and norgestrel 500 µg (or levonorgestrel 250 µg) are taken: 2 pills as early as possible and 2 after 12 hours of first dose. The treatment is most effective if first dose is taken within 12-24 hours of intercourse but it can be taken upto 72 hours.
   OC pills containing lower dose of estrogen (30 µg) should be taken as 2 doses of 4 pills 12 hours apart.
   Failure rate ranges from 0.2-2%.
2. Progesterone only pill: Levonorgestrel 0.75 mg 2 doses 12 hours apart. First dose should be started as early as possible but within 72 hours. It is available as Ecee 2, Pill 72, preventol Norlevo etc.
   It is useful when oestrogen is contraindicated.
3. IUCD : Insertion of copper IUCD within 5 days intercourse (later than hormonal method). It can be kept inserted if couple wants to continue with it as method of temporary contraception.
4. Antiprogesterone : Mefipristone (RU-486) 600 mg oral single dose within 72 hours after unprotected intercourse. Doses as low as 50 and even 10 mg are found effective.

   Failure rate reported for different hormonal methods are < 2% while for IUCD

43. Tell something about silastic vaginal ring and contraceptive patch ?

Silastic vaginal rings: These have advantage over previous methods that women can insert and remove themselves and they are immediately reversible. Progesterone is slowly released from the ring and can be absorbed through the vaginal epithelium. Most rings are between 50 and 60 mm in outer diameter and 7.5-9.5 mm thick. They are pliable and similar to the inert pessaries used for prolapse. The ring containing levonorgestrel 5 mg releases 20 µg per day and can be left in place for 3 months. The ring containing estro + prog (Nuva ring) have also been found useful. Like OCs they are worn for 21 days and then again inserted after 7 days ring free period. Failure rate is less (1.8/HWY) but break through bleeding is a problem.

Contraceptive patch: Combined estrogen + progesterone patch is under trial. It delivers in the blood stream 20 µg of ethinyl estradiol and 150 µg norelgestromin. New patch is worn every week. After wearing for consecutive 3 weeks, 4th weeks is patch free. It may be applied abdomen, buttocks, thigh or upper outer arm. Failure rate is less than 1/ HWY.

28

Ultrasonography in Obstetrics

Kanan Yelikar

1. What are the indications of USG in obstetrics?

There are 'n' number of indications. USG has become a vital, diagnostic tool in obstetrics. It has a very important role in almost all the conditions in obstetrics. Indications in First, second, third trimester.

First Trimester

To confirm the diagnosis of early pregnancy to decide whether the pregnancy is intrauterine or extrauterine, single or multiple and to decide whether it is viable or not and also to decide the gestational age.

2. What do you mean by viable pregnancy on USG?

Viability in ultrasonic language means, presence of cardiac activity and presence of yolk sac.

3. What are the normal findings first trimester USG.

A single gestational sac, with foetal pole, and cardiac activity. No evidence of any subchorionic or subamnionic hematoma.

4. What are the abnormal ultrasonic findings in first trimester?

Blighted ovum: It is empty gestational sac, i.e. gestational sac without embryo. It is also called as an embryonic pregnancy.

5. Which anomaly can be diagnosed in first trimester?

1. Chromosomal anomaly like Dowtis syndrome by measuring muchal translucency.
2. Missed abortion: Presence of gestation sac and fetal pole, but absence of foetal heart activity.
3. Incidental finding of molar pregnancy.

6. Which route of sonography is preferred in first trimester?

Transvaginal sonography is the preferred route, because it gives better resolution hence early diagnosis is possible.

7. How do you decide gestational age?

Measurement of gestational sac and crown rump length (CRL) give a fair idea about the gestational live.

8. How do you diagnose ectopic pregnancy?

Empty uterus (Midline echo), with positive pregnancy test and presence of adnexal mass.

9. Second trimester.

2nd trimester USG is mainly done to rule out fetal congenital structural anomalies. This is called as "Anomaly Scan"

10. What are common anomalies?

1. Open neural tube defects like: anencephaly, spina bifida, meningocele, meningomyelocele, encephalocele, etc.
2. Neural axis anomaly like: Hydrophalus (ventriculomegaly)

11. Anterior abdominal wall defect: gastroschisis, omphalocele.

1. Congenital heart disease, e.g. septal defects.
2. Renal anomalies like multicystic kidneys.
3. GIT esophageal atresia, duodenal atresia.
4. Facial anomalies like deft lip/palate
5. Musculoskeletal anomalies

14. Third trimester (comment on fetus, placenta and amniotic fluid).

To confirm the growth pattern fetal biometry, i.e. measurement of fetal parameters like BPD, FL, AC (biparietal diameter, femur length abdominal circumference).

- Localization of placenta, i.e. in cases of APH to decide whether it is a placenta praevia or it is a normally situated placenta.
- Amniotic fluid, i.e. oligohydramnios or polyhydramnios.

15. What are indications of Doppler in obstetric?

Doppler veolcimetry is used to study the blood flow in different fetal vessels.

16. Which are blood vessels significantly studied?

1. Uterine artery
2. Umbilical A
3. Middle cerebral artery
4. Ductus venosus.

The Doppler studies help us to know whether blood supply to fetus is compromised due to utero-placental insufficiency.

17. Common indications of Doppler.

1. IUGR
2. PIH
3. Diabetes M.
4. Oligohydramnios.
5. Post term pregnancy

18. Other indications of USG in 3rd trimester.

To study the BPP (Biophysical profile) of the fetus, to know the fetal well being, in cases of high risk pregnancy.

19. What are the parameters of BPP?

1. Fetal Breathing movement.
2. Fetal movements.
3. Fetal tone
4. Fetal reactivity.
5. Qualitative amniotic fluid volume.
6. Placental grading.

Score between 7 to 12 indicates a good fetal outcome.

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IUGR

Pankaj Desai, Nilesh Dalal

CASE STUDY

Mrs. A, 28 years, middle socioeconomic status, H/o pregnancy 34 weeks, $G_2P_1A_0$, first fullterm vaginal delivery, was admitted with complaints of swelling over limbs, transient headache and C/o breathlessness.

On examination, pulse—84/min, 140/90 BP, pallor present, pedal edema ++, facial puffiness+, CVS and respiratory system examinations normal, maternal weight gain 10 kg in last month.

On obstetric examination, P/A 28 wks, liquor adequate for the gestational age, cephalic presentation, fetal heart sounds 142/min regular, abdominal wall edema + EBW 1.2 to 1.4 kg.

Investigations:

Hb—8 gm%
T and D—10,500/cu mm, VDRL—nonreactive
HIV—nonreactive, RBS—70 mg%
Urine- R/M—protein traces, no pus cells/no RBC.
Fundus—WNL
Serum creatinine—0.7 mg%
BUN—18 mg%
Serum proteins (total)—5.8 gm%

FAQS ON THIS CASE:

1. What is your provisional diagnosis in this case?

34 weeks pregnancy with IUGR with mild degree of anemia with pregnancy induced hypertension.

2. What are the different types of growth restriction.

Asymmetrical, symmetrical, unclassified and idiopathic.

3. How do you differentiate small for date an IUGR?

By history, clinical examination and biometry.

4. How far your gestational dating is correct on history taking?

Patient is educated, aware of her cycles. Her previous cycles have been regular 3/30 days and so, her dates-can be said to be "excellent."

5. What are the substrates for normal fetal growth?
Most important being oxygen, glucose and amino acids.

6. Which of the two types of IUGR, you think is prognostically bad?
Symmetrical variety, as it reflects some congenital defect or intrauterine infection or chromosomal problem?

7. What maternal condition in your case, can be correlated as cause for IUGR?
Anemia and mild PIH as the cause of IUGR.

8. What are other common causes of IUGR?
Preeclampsia chronic hypertension, diabetes, cardiac disease, placental causes like abnormal placentation, chronic villitis, placenta praevia, and placenta infarcts. Fetal causes like chromosomal abnormalities, infections, and multifetal pregnancies.

9. What other investigations you will like to do, for confirming the diagnosis?
Sonography to do gestational dating (fetal/biometry).

10. What parameters on sonography are taken under consideration for gestational dating?
Biparietal diameter, femur length, abdominal circumference, head circumference, HC/AC, estimated fetal weight.

11. How will you manage this case?
As patient is diagnosed as a case IUGR (History and examination) we will admit her for rest and further investigations. Improve her nutrition and control her blood pressure. After optimum maturity of fetus, deliver her.

12. How will you assess her while admitted in hospital?
By clinical examination, gravidogram, maternal weight gain, AFI, fetal heart sound record by serial USG, CTG.

13. What is the importance of weight gain?
Decreased maternal weight gain during pregnancy is a relatively insensitive sign of inadequate fetal growth. A gain of 12 kg is ideal during pregnancy and if weight gain is suboptimal, fetal growth can be hampered.

14. What other treatment to you, suggest for management of IUGR?
Rest in left lateral position, oral and IV hydration, amino acid supplementation, aspirin. However none of these have been proved to be conclusively helpful.

15. What is the incidence of IUGR in developing country like India?
40-45%

16. What is ponderal index?
PI = Fetal weight/ L3. It is an index for measurement of fetal malnutrition. Normal value is 8.325 + 2.5 (2 SD). If PI < 7 it is indicating IUGR.

17. Is any other radiological investigation helpful in diagnosis of IUGR?
Color Doppler study, to know the resistance and pulsatility index.

18. What is trisomy 21?
Also known as Down's syndrome, and a cause of IUGR, it is a chromosomal abnormality (8 to 12% of growth retarded infants are due to chromosomal abnormality).

20. What does the pneumonic "TORCH" stand for?
Toxoplasma rubella cytomegalovirus and herpes simplex, intrauterine infection by this organism can cause IUGR.

21. In which conditions, you will like to deliver the patient before term?
Anhydramnios (no pockets of fluid that are clear of cord loops at 30 weeks), gestation or beyond. Repetitive fetal heart rate decelerations. Lack of growth over 3 weeks period and mature lung studies. Abnormal UAD (umbilical Doppler velocity).

22. On what parameters, your decision to delivery such a patient depends:
1. Gestational age
2. Underlying etiology
3. Probability of intact extrauterine survival
4. Level of expertise
5. Available technology
6. NICU—Neonatologist availability.

22. When would you deliver her?
As she admitted, we will like to wait for pulmonary maturity to be achieved, which can be indirectly judged by fetal biometry, lecithin sphingomyelin ratio. If liquor volume is adequate management can be continued till optimum fetal growth is achieved.

23. As she has a previous normal delivery no adverse factor can we try for a vaginal delivery?
It depends on whether the fetus is acidotic or otherwise. If on basis of color Doppler the fetus is acidotic then vaginal trial should not be given. If not, with continuous intrapartum fetal monitoring and preparation of immediate intervention, a vaginal trial can be given.

24. What are the neonatal complications of IUGR baby?
The IUGR infant shows signs of soft tissue wasting. The skin is loose, thin and there is little subcutaneous fat. They are susceptible to hypoglycemia, hyperbilirubinemia, necrotizing enter colitis, hyperactive viscosity syndrome.

25. What other neonatal problems are specific to an IUGR?
Perinatal asphyxia, acidosis, persistent fetal circulation, MAS, hypoxic ischemic encephalopathy, hypothermia.

26. What are the clinical indices, that the fetus is developing fetal distress?
Decreased fetal movements, bradycardia or tachycardia and if in labour then meconium stained liquor.

27. Can liquor be increased in cases of oligohydramnios?
Yes, by amnoinfusion procedure.

28. How does one differentiate IUGR/preterm baby?
IUGR—neonate has an old man's look, loss of subcutaneous fat, thinned skin, gestational maturity is ahead of the preterm neonate. Neurological reflexes are well developed.

29. What do you mean by placental insufficiency?
Placenta is the main supportive organ for growth of fetus. Fetoplacental circulation can be hampered due to decreased blood flow, placental aging, reduction in villi and stem capillaries and decrease in stroma and parenchyma, poor trophoblastic invasion.

31. What is the role of dexamethasone and when it should be administered?
Dexamethasone therapy is given to accelerate pulmonary maturation when gestational age is less than 34 weeks.

Dose
- Betamethasone 12 mg—24 hours apart
- Dexamethasone 4 mg—6 hourly to 24 hours.

32. What advise, you would give regarding feeding, of an IUGR neonate?
Early feeding within 1-2 hours is to be started, with 5 to 10 ml of 10% glucose. The feeding is to be repeated at 2 hourly intervals. If baby can tolerate it, expressed breast milk or humanized milk may be given 2 hourly in small amount for 48 hours.

33. What is biophysical profile? (Who devised it)?
It is scoring system to assess fetal well being it takes fetal movements, heart activity, fetal tone, amount of amniotic fluid and fetal breathing movements. A total score of movements (10) is given. In account, lower score suggests fetal distress. Amniotic fluid index is the most sensitive parameter of all.

Manning et al devised the score, which was later on modified by Vintzeleous et al.

34. Which antihypertensive drugs are used in pregnancy? How will you manage this case?
Tab. Methyldopa (250 mg qid) and then taper to (250 mg tds) over a period, to buy time for gaining pulmonary maturity. Nifedepine can be used for acute hypertension. It has quick action, but can interfere in induction of labor and can cause postpartum hemorrhage due to its relaxant effect on uterus.

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Maternal Mortality

Milind Shah

1. How would you define Maternal Mortality?

Maternal mortality is the death of a woman while pregnant or within 42 days of termination of pregnancy, irrespective of the duration or site of the pregnancy, from any cause related to or aggravated by the pregnancy or its management, but not from accidental causes.

2. What are the indicators of maternal mortality?

Indicators of Maternal Mortality

The most common indicators of levels of maternal mortality are:

- Maternal Mortality Ratios: the number of maternal deaths per 100,000 live births;
- Maternal Mortality Rates: the number of maternal deaths per 100,000 women aged 15-49 per year; and
- Lifetime Risks: the probability of maternal death faced by an average woman over her entire reproductive life-span.

Why is Measuring Maternal Mortality Difficult?

1. Maternal deaths are frently under reported and misidentified:
   This is especially true in many developing countries, where people often die outside the formal health care system and subsently where the family must assume the responsibility of registering the death with the responsible local authorities. In this type of environment, such a death is often left unrecorded or information related to the cause of death and the temporal relationship to pregnancy—is not recorded. Studies conducted in developed and developing countries indicate that underreporting of maternal deaths is significant. Some studies have shown that the actual number of maternal deaths for the period under study was double or triple what was initially reported.
2. Maternal deaths are often misclassified:
   In many situations the medical "cause of death" of the woman might not be known and/or noted properly by health professionals or other officials at the time of registry. As mentioned above, omission of the information related to whether the woman was pregnant or had recently delivered

might also be omitted, thus further obscuring the possible causes of death. In some countries, the cause of death can also be intentionally misclassified, especially when it is related to complications of clandestine abortions.

The most recent estimates developed by WHO, UNICEF and UNFPA on the maternal mortality rates worldwide relate that:

- 515,000 women die each year-one every minute-from complications of pregnancy and childbirth;
- Of these deaths, over half (273,000) occurred in Africa, about 42% (217,000) occurred in Asia, about 4% (22,000) in Latin America and the Caribbean, and less than 1% (2,800) in the more developed regions of the world.
- Twelve countries account for 65 percent of all maternal deaths worldwide. These include: India (110,000), Ethiopia (46,000), Nigeria (45,000), Indonesia (22,000), Bangladesh (20,000) Democratic Republic of Congo (20,000), China (13,000), Kenya (13,000), Sudan (13,000), United Republic of Tanzania (13,000), Pakistan (10,000) and Uganda (10,000).
- The life time risk of maternal death varies widely from one region of the world to another. For example, in Africa it varies from 1 in 11 in Eastern Africa, to 1 in 65 in Southern Africa, from 1 in 55 in South-central Asia to 1 in 840 in Eastern Africa, and in Latin America from 1 in 85 in the Caribbean to 1 in 240 in Central America. Within specific regions of the world, it varies from country to country, and further from region to region within each country.

3. Which method would be better to measure MM according to you?

Methods of Measuring Maternal Mortality

The most accurate method of monitoring maternal mortality is through vital registration. Vital registration registers all births and deaths and for death statistics it provides medical certification of the causes. To be efficient, the method must ensures the complete or near-complete reporting of all births and deaths within a specific region or country.

Although considered the most efficient method to track maternal mortality trends, the vital registration approach relies on the proper registration and classification of all deaths, including maternal deaths.

Unfortunately, the vital registration approach is not possible in many developing countries where vital registration systems are lacking or incomplete and causes of death are more often than not incorrectly attributed or are unreported. In response to this reality, alternative methods of determining the level of maternal mortality have been used to estimated maternal mortality. The most well known include:

1. **RAMOS (Reproductive age mortality surveys):** This approach consists of in-depth reviews of deaths among all women of reproductive age. Although RAMOS can provide useful data for program planning, monitoring and evaluation (e.g. not only maternal mortality ratio, but also causes of deaths, high-risk groups and avoidable factors), they are considered complex, time-consuming and costly to conduct.

2. **Household survey using direct estimations:** The household survey method consists of visiting a large number of households for the purpose of seeking data related to maternal deaths. Overall, this method is also considered expensive for most countries, due to the large sample of households that need to be surveyed to ensure reliable and representative results.
3. **Sisterhood Method:** This third method of estimating maternal mortality is based on the collection of information provided by siblings (usually sisters). Although it requires much smaller sample sizes, it is considered a more cost-effective method, especially when conducted in conjunction with existing household surveys. Its major disadvantage lies in the fact that the data collected is usually ten years old and thus, provides little insight of the changes that may have occurred over the recent past.
4. **WHO/UNICEF/UNFPA Estimates:** In 1996, WHO and UNICEF, with the participation of UNFPA developed estimates of maternal mortality for the year 1990. The approach developed consisted of a "a dual strategy" method where: 1) for countries where maternal mortality estimates already existed, the figures where adjusted to account for under reporting and misclassification, and 2) for countries where no reliable estimates were available, a model which permitted the calculation of estimates based on fertility rates and the proportion of births assisted by skilled personnel was used. The exercise (with some adjustments) was repeated in 2001 for the purpose of establishing maternal mortality estimates for 1995. Although this approach provides some sense of the magnitude of the problem, the figures generated are not intended to serve as precise estimates.

4. Though woman survives what are other complications often not accounted properly in obstetrics and what is their impact on maternal health?

In addition to these maternal deaths, as many as 50 million women—more than one quarter of all adult women now living in the developing world—experience maternal health problems annually. These include: uterine rupture, prolapse, hemorrhage, vaginal tearing, urinary incontinence, pelvic inflammatory disease, and obstetric fistula, a muscle tear that allows urine and feces to seep into the vagina.

- It is estimated that as many as 300 million women—more than one quarter of all adult women now living in the developing world—currently suffer from short or long term illnesses and injuries related to pregnancy and childbirth.
- Adolescent girls (15-19 years of age) are at a greater risk during pregnancy and childbirth. In fact, they are twice as likely to die from childbirth as women in their twenties, and if under 15 years of age, five times more likely to die.
- Unwanted pregnancies, which often lead to unsafe abortions, continue to be a major preoccupation for women worldwide. It is estimated that 50 million unwanted pregnancies are terminated each year, and some 20 million of these are deemed unsafe and thus, a threat to women's health

and lives. Furthermore, as with maternal deaths, approximately 95% of unsafe abortions occur in developing countries, causing the deaths of more than 200 women daily.

5. What are causes of maternal death and how far they can be predicted or prevented?

Main Causes of Maternal Death and Injury

During the last 10 years, much has been learned about the main causes of maternal death and injury and a women's chances of experiencing some complications during labor and delivery.

Research in the field of maternal death and injury has confirmed that:

- The direct causes of maternal deaths are similar throughout the world. They include: hemorrhage (25%), sepsis (14%), hypertensive disorders (13%), complications due to unsafe abortions (13%) and obstructed labor (7%).
- An additional 20% of maternal deaths in developing countries are attributed to preexisting medical conditions such as anemia, malaria and HIV/AIDS that are aggravated during pregnancy.
- Obstetrical complications can neither be predicted nor prevented, except those related to unsafe induced abortion. It is estimated that for every 100 women who become pregnant, 15 will develop life-threatening complications mostly around the time of birth.

6. Which are other social and medical factors that influence complications during pregnancy?—AR

Birthing mothers' capacity to survive complications is also influenced by a number of others factors such as:

- Women's poor health and nutrition from childhood as well as during pregnancy
- Inadequate, inaccessible or unaffordable health care services
- Poor hygiene and care during childbirth
- Socioeconomic and cultural factors, such as poverty, women's unequal access to resources - including health care, food and preventive services, their heavy physical work load and their lack of decision - making power in families, communities and societies.

In summary, it is now recognized that maternal death and injury "is rooted in women's powerlessness and unequal access to employment, finances, education, basic health care and other resources. These factors set the stage for poor maternal health even before pregnancy occurs, and make it worse once pregnancy and childbearing have begun..."

It is for this reasons that maternal death and injury are considered a matter of social injustice.

7. We all discuss about 3 Delay Model. Which delay do you feel is more commonly seen in our country or other countries where MM is very high?

The 3 Delay Model

When looking at the issue of access to essential obstetrical care (or medical care at the time of complications), the 3 delay model is often used. This concept

may be useful in helping to identify which delays, or barriers, prevented the birthing mother from accessing appropriate health care when complications arose. They include:

1. The delay to seek care
2. The delay to reach proper medical services
3. The delay in accessing quality care when she reaches the medical facility.

Delay 1: Deciding to seek care

When complications arise, the decision to seek care is the first step which must be taken by the birthing mother, her family and/or her attendant(s) to ensure access to the appropriate medical care needed. This decision may be influenced by many factors such as:

- The ability of the birthing mother and her family or attendants to recognize obstetrical complications
- Who decides when to seek care: the birthing mother herself, her family (husband, mother-in-law) or the assistants
- Knowledge as to where to go to seek appropriate medical assistance; and
- Cultural factors such as the way society views delivery and childbirth (e.g. women are expected to labor in silence).

Delay 2: Reaching the proper medical services

Once the decision has been made to seek medical care, the issue of transportation and/or communication often comes into play. A woman who lives in a rural area, far from health facilities, can face difficulties accessing transportation to get to a health center, especially if she or her family have no means of transportation and/or little financial resources. Furthermore, once at the community clinic, the birthing mother may need to be transferred to a higher level medical facility for services such as blood transfusion or cesarean section. The delays in accessing transportation to ensure timely access to health services is thus extremely important to consider when trying to this barrier is: "is there a village or sub-county plan for emergency transportation in case of obstetrical emergencies?"

Delay 3: Accessing quality care

Once the birthing mother arrives at the health care, it is thus just as important that she accesses the required emergency care services. Access to care delays are usually dependent on a number of factors, such as the number and skill level of staff, availability of drugs, supplies and blood and the general condition of the facility. They may also include:

- delay in the arrival (time wise) of the nurse, midwife or physician attending the patient;
- delay in accessing, in a timely fashion, the needed medical procedure (e.g. cesarean section, blood transfusion).

A good way to gain insight on the quality of this care is to survey the patients on their perception of the care they received.

It is usually recognized that the quicker each delay is dealt with, the greater the chances that a birthing mother and her newborn will survive and be able to live free of any long term injuries.

8. What is impact on families of maternal death or injury?

Women are not the only victims of maternal death and injury. A women's death affects her infant and often her other surviving children, her family and her community at large. Maternal injury can also impact on her families' "economic status."

It is internationally recognized that the impact of maternal death on families and communities are the following:

- In developing countries, a mother's death during childbirth means almost certain death for a newly born infant and severe consensus for her surviving children. Studies have shown that there is an increased probability of older children, especially daughters, dying and of their absenteeism from school. Without education, the cycle of poverty continues.
- Women are more likely than men to spend their income on family welfare. Consently, when a mother dies during childbirth, the family may lose an important, or its only, financial contributor.
- Within the family, the mother usually assumes the main responsibility for maintaining the home and caring for society's dependents—children and the elderly. The loss of a mother thus affects the well-being of the family as a whole.
- When a mother dies, the community loses a vital member whose unpaid labor is often central to community life.

9. Which Essential services for safe motherhood are must to reduce MM and morbidity?

Essential Services for Safe Motherhood

In light of the above, much has also been learned as to Safe Motherhood programming. It is internationally recognized that the essential services for Safe Motherhood include:

- Community education on safe motherhood
- Antenatal care and counseling, including the promotion of maternal nutrition
- Skilled attendance during childbirth
- Care for obstetric complications, including life-threatening emergencies
- Postpartum care
- Services to prevent and manage complications due to unsafe abortion
- Family planning counseling, information, and services
- Reproductive health education and services for adolescents.

10. That's how this concept of reproductive health has come, what is it?

Reproductive Health

A reproductive health approach means that:

- people have the ability to reproduce and regulate their fertility
- women are able to go through pregnancy and child birth safely
- the outcome of pregnancy is successful in terms of maternal and infant survival and well being
- couples are able to have sexual relations free of the fear of pregnancy and of contracting disease (Fathallah, 1988).

The reproductive health approach extends beyond the narrow confines of family planning to encompass all aspects of human sexuality and reproductive health needs during the various stages of the life cycle.

11. We are failing in implementation due to several reasons. So what should be rational for recommending a package of services?

The rationale for suggesting a package approach is to enable program planners to: (1) assess the feasibility and management implications for implementing various combinations of health services at different levels of the health service system in diverse settings; and (2) examine the cost, financing and sustainability implications of implementing these health services.

As there is enormous diversity in India among the regions and states as well as between rural and urban areas, no single package of services can be recommended for nationwide implementation. While in underserved areas such as in the northern states of India there is a continuing need to strengthen the health infrastructure and improve service access, in states with better developed programs such as Tamil Nadu and Kerala, efforts should be now made to expand the range and quality of services provided.

The following services are included in a comprehensive reproductive health services package:

- Prevention and management of unwanted pregnancy
- Services to promote safe motherhood
- Services to promote child survival
- Nutritional services for vulnerable groups
- Prevention and treatment of reproductive tract infections and sexually transmitted infections
- Prevention and treatment of gynecological problems
- Screening and treatment of breast cancer
- Reproductive health services for adolescents
- Health, sexuality and gender information, education and counseling
- Establishment of effective referral systems.

The last two services, health, sexuality and gender information, education and counseling and the establishment of effective referral systems, are not separate services but are critical for the effective implementation of all the other reproductive health services within the service system.

The following package of essential reproductive health services is recommended for nationwide implementation:

- Prevention and management of unwanted pregnancy
- Services to promote safe motherhood
- Services to promote child survival
- Nutritional services for vulnerable groups
- Prevention and treatment of reproductive tract infections and sexually transmitted infections
- Reproductive health services for adolescents
- Health, sexuality and gender information, education and counseling
- Establishment of effective referral systems.

All the services included in this package are presently recommended as a part of the government's Health and Family Welfare Program.

12. Various levels of health service systems have their own problems while executing these services. Which are these problems?

While all the services are theoretically included in the national program and are specified in the various policy and program documents, there have been serious problems with their implementation at various levels of the health delivery system.

The discussion focuses on interventions within each service component that can be implemented at various levels of the health service system. Health interventions that can be implemented at the community, subcenter, primary health center (PHC) and community health center (CHC)/district/subdistrict hospital levels are delineated in tabular form.

The PHC is the weakest link in the chain. While one PHC catered to a population of about 100,000 in the past, according to the present norms there is one PHC for a population of 20,000-30,000 population. In fact, the new PHC is often an upgraded subcenter.

According to the government's prescribed norms, in addition, to a voluntary worker who is paid an honorarium, there is a provision for one male and one female multipurpose worker at each subcenter. The female health worker is an auxiliary nurse midwife (ANM). A subcenter carters to 5000 population in the plains and to 3000 population in hilly and tribal areas. The staffing norms for the new PHC that caters to 30,000 population in the plains and 20,000 population in tribal and hilly areas includes one medical offer, a pharmacist, a nurse midwife, a health educator, one male and one female health assistant and one male and one female health worker. The old PHCs have 1 to 3 physicians and more paramedical staff. The prescribed norms for staff at the CHC include: five qualified or specially trained doctors—a surgeon, an obstetrician gynecologist, a physician, a pediatrician, and a public health physician. In addition, there should be seven nurse midwives, a dresser, a pharmacist/compounder, a laboratory technician, and a radiographer at this facility. A CHC covers approximately 100,000 population (Government of India, 1995). The CHC is expected to have facilities for managing obstetric emergencies. Currently, there are very few CHCs with prescribed staff and facilities.

In many areas staff are not in place at PHCs and CHCs according to prescribed norms. There are particular gaps in the case of women physicians and male multipurpose workers. It is difficult to recruit women physicians for rural facilities. The government proposes to contract private physicians for PHCs and CHCs.

However, community health workers such as traditional birth attendants (TBAs), who are not a part of the formal health system, play an important role in providing reproductive health services at the community level.

Urban health in India has been neglected even though the urban poor are growing in numbers and are increasingly exposed to serious health risks.

13. Prevention and management of unwanted pregnancies itself would lead to marked reduction in MM. What are hurdles we face while advocating contraception and what could be solution for it?

For ensuring contraceptive safety, the program must focus on all clinical procedures especially aseptic technis and on screening clients for contraindications and preexisting health problems.

Developing effective out reach programs should be a high priority if counseling and follow-up services are to be provided, especially in rural areas.

14. One of the definitely preventable cause of MM is providing services for safe abortions? How to go about it? And are really safe abortions really safe in India?

The Medical Termination of Pregnancy (MTP) Act was passed by the Indian Parliament in 1971 but what was thought to be a landmark in social legislation, has failed to translate into reality for the majority of Indian women, particularly in rural areas. Today, there are more illegal abortions in India than there were prior to the MTP Act with about 15,000-20,000 abortion-related deaths occurring annually, mainly among married, multiparous women.

Services for first trimester abortion should be made available at PHCs and facilities for second trimester abortions at CHCs. Unsafe abortion is an important cause for maternal mortality and results in high levels of maternal morbidity in India.

About 11-12 percent of maternal deaths in rural India are due to septic abortion (Government of India, 1990). Septic abortions account for up to 25 percent of all maternal deaths in hospital studies in India.

Although unsafe induced abortion is the greatest single cause of mortality for women, it is also the most preventable. Women need not die or suffer medical consensus from abortions because abortions do not kill women; it is, rather, unsafely performed abortions which kill.

Expanding family planning services is an important strategy for decreasing pregnancy related mortality and morbidity.

Unsafe abortion is an important cause of maternal mortality and results in high levels of maternal morbidity in India. While public sector programs should be strengthened to provide safe abortion services, there is an urgent need to improve the quality of services provided by the private sector as private practitioners are by far the most important providers of abortion services in the country.

While public sector programs should be strengthened to provide safe abortion services, there is an urgent need to examine the quality of services provided by the private sector and to organize training programs for private practitioners, as they are by far the most important providers of abortion services in the country.

15. Coming to Maternity health services, it is debate now days as far usefulness of antenatal services. What is your comment? How do you get about ideal ANC services?

Services for maternal care should be designed to ensure timely detection, management and referral of complications during pregnancy, delivery and the postpartum period.

Antenatal services should be organized to detect and manage complications related to pregnancy such as anemia, infection, preeclampsia, malpresentation and obstructed labor. Women should be educated about the danger signs of pregnancy and provided information on where to seek help. Antenatal visits should provide an opportunity to offer advice and counseling on hygiene, breast-feeding, nutrition, family planning and immunization as well as to treat preexisting conditions such as diabetes and infections such as malaria and tuberculosis that are commonly prevalent, may be aggravated by pregnancy, and may complicate pregnancy (World Bank, 1994).

There should be at least 3-4 antenatal examinations by a health care provider. The first visit should take place as soon as pregnancy is detected, preferably before 10 weeks to confirm the pregnancy; provide nutritional advice and supplements; and provide the first dose to tetanus toxoid immunization. A second visit is recommended at 20-24 weeks to detect and treat abnormalities; to identify and refer cases with complications; and to provide the second tetanus toxoid injection. A visit at 28-32 weeks would enable the service provider to detect malpresentation and to diagnose and treat maternal illnesses. An antenatal visit should be made at 36-38 weeks to confirm the position of the fetus; to make an assessment of cephalopelvic disproportion; and to manage maternal illnesses.

The subcenter must be equipped for examining hemoglobin, blood pressure, urine albumin, and sugar, and for checking body weight and height.

16. Safe delivery services would be definite solution in avoiding many deaths. But again so many difficulties we face for it. How to overcome it?

All deliveries must be managed by trained birth attendants. Because complications can develop without warning, it is critical to put in place effective systems to ensure timely referral and management of emergency complications.

However, the vast majority of births in India take place at home. The National Family Health Survey shows that in 1992-93 only 25.6 percent of all births and 16.1 percent of all rural births were conducted in institutions and as many as 65 percent were delivered by traditional birth attendants (International Institute of Population Studies, 1994).

The need for conducting clean home deliveries using safe delivery kits and the importance of recognizing danger signals for emergency obstetric care should be highlighted.

17. Postpartum period is the period during which maximum maternal deaths take place. Why is it so and what is solution for it?

To date, postpartum programs in India have focused primarily on providing family planning services. Postpartum programs should include services for

the early detection and management of infection and hemorrhage; support for breast-feeding for at least six months; nutrition counseling; and family planning services.

Antibiotic treatment is sufficient to cure infection in more than 80 percent of cases if taken within four days of the onset of fever.

18. Now in spite of this if maternal death takes place, How it should be faced or rather what lessons we should take?

Maternal Death Case Reviews

A maternal death case review is defined as "a qualitative, in-depth investigation of the causes of and circumstances surrounding maternal deaths occurring at health facilities. Maternal death case review focuses on with identifying the factors at the facility and in the community that contributed to the death, and which ones were avoidable".

Usually carried out by facility staff, maternal case reviews provide valuable information on the circumstances—in the facility and in the community—surrounding a death. They are considered affordable and because of their participative approach (e.g. they involve health professionals and at levels and community people) further, they can offer good opportunities for sensitizing and educating people to the issue of maternal mortality.

Verbal Autopsy of Maternal Deaths

Verbal autopsies consists of "a method of finding out the medical causes of death and ascertaining the personal, family or community factors that may have contributed to the deaths in women who died outside of a medical facility." They consist of inquiries collected from lay reporters and relatives to establish the cause of death. The data is usually collected outside the health facilities.

Verbal autopsies are a useful tool for identifying maternal deaths and collecting valuable information about deaths that have occurred outside the health facilities. Further, they provide a great opportunity to obtain family and community members' opinions on issues related to access to and the quality of health services.

Confidential Enquiry into Maternal Deaths

A confidential enquiry into a maternal death consists of "an investigation undertaken by multi-professional teams that identifies weaknesses and deficiencies in the maternal health care system. It investigates individual maternal deaths, with the cooperation of all those involved in the care of the patient, yet removes all identifying details from individual case reports to ensure that information is used only for making recommendations for positive practice changes, and not for punitive action."

Confidential enquiries are not interested in determining who is at fault, but more specifically in determining the deficiencies in the health care systems that may have contributed to the death for the purpose of instituting change as to ensure that future similar deaths are prevented.

Usually, more resource intensive (e.g. time, structure, and support system needed) than the other investigative tools, confidential enquiry methods can be instituted by public health authorities or by government and are usually undertaken and supported at national level by the Ministry of Health.

19. What is AMDD?

Averting maternal deaths and disability program. This program has been funded by Bill and Mellinda Gates foundation. This program aims at reducing maternal mortality by improving EMOC (emergency obstetric care) in India.

PART 2: GYNECOLOGY

31 Gestational Trophoblastic Neoplasia

Neelam Aggarwal

HISTORY

- Amenorrhoea
- Hyperemesis gravidarum
- Vaginal bleeding
- Absent quickening
- Passage of grape like vesicles
- Symptoms suggestive of hyperthyroidism
- Preeclampsia in early pregnancy
- No evidence of fetal movement.

EXAMINATION

- Excessive uterine enlargement , EB absent, doughy feel, external ballotment absent
- Pre-eclampsia
- Tachycardia, tremor, warm skin
- Ovarian cyst +.

ULTRASOUND

- Snow storm appearance
- Thecalutein cysts.

Gestational trophoblastic disease includes a spectrum of interrelated tumors

- Complete/partial hydatidiform mole
- Invasive mole
- Placental site trophoblastic tumor
- Choriocarcinoma.

QUESTIONS

1. What is gestational trophoblastic neoplasia (GTN)?

Gestational trophoblastic neoplasia, originates from trophoblast, is among the rare human tumor that can be cured even in the presence of widespread dissemination. GTN includes a spectrum of interrelated tumors including complete and partial hydatidiform mole, invasive mole, choriocarcinoma and placental site trophoblastic tumor.

2. What are the features of complete and partial hydatidiform mole?

	Complete	*Partial*
Fetus or embryo	Absent	Present
Swelling of chorionic villi	Diffuse	Focal
Trophoblastic hyperplasia	Diffuse	Focal
Karyotype	46, XX (90%); 46 XY	Triploid (90%)

3. What are the classic clinical features of molar pregnancy?

- Vaginal bleeding with passage of grape like vesicles.
- Hyperemesis gravidarum
- Excessive uterine enlargement
- Preeclampsia; features of hyperthyroidism in some
- Presence of ovarian cysts, trophoblastic embolization.

4. How to diagnose molar pregnancy?

- Presence of vaginal bleeding, excessive uterine enlargement
- Pregnancy test positive in dilution
- Snow storm appearance on ultrasound.

CBC
Blood grouping and Rh typing
Serum β-hCG (> 1 lac m iu/ml)
Thyroid function test
USG snow storm appearance of V mole and ovaries for theca lutein cysts. Liver is to be looked for metastasis.
X-ray chest
Coagulation profile
USG in partial mole:

i. Focal cystic spaces in the placental tissues.
ii. Increase in transverse diameter of gestational sac.

5. What is the treatment of molar pregnancy?

- Evaluation for associated medical complications
- Stabilization of patient's condition
- Evacuation of molar pregnancy.

6. Precautions at the time of surgical evacuation of molar pregnancy.

Identify and manage associated medical complications and stabilize patient's condition.

Suction evacuation is the method of choice. Suction canula 12 mm is usually sufficient as there are no fetal parts.

Oxytocic infusion once evacuation is complete (during evacuation oxytocin is contraindicated by some due to fear of trophoblastic embolization).

Advice and help from experienced colleagues to be sought in case of large molar pregnancy.

7. Describe post molar pregnancy follow up of the patient.

- Serum β-hCG every week till 3 samples are negative.
- Serum β-hCG every month for 1 year.
- Serum β-hCG every 3 months for 2 years.
- If value reaches negative within 8 weeks, follow up of the patient can be reduced to 6 months.
- Persistent gestational trophoblastic tumor, develops in about 20% of patients of complete mole and 1% patients of partial mole.
- hCG regression curve helps to monitor the patient. hCG becomes normal at the end of 9 weeks.

8. Future pregnancy after molar pregnancy evacuation.

Women should be advised not to conceive until hCG levels have been normal for six months.

Women who undergo chemotherapy are advised not to conceive for one year after completion of the treatment.

Risk of further molar pregnancy is low (1/55) and > 98% women who become pregnant will not have a further molar or be at increased risk of obstetric complications.

After conclusion of any further pregnancy, at any gestation, hCG estimation is rested to exclude disease recurrence.

9. What are the indications of chemotherapy?

- Evidence of metastases in brain, liver or gastrointestinal tract or radiological opacities > 2 cm on chest X-ray.
- Histological evidence of choriocarcinoma, baseline hCG > 1 lac/ml.
- Rising hCG after evacuation
- Raised hCG 6 months after evacuation.

10 Which are the factors associated with an increased risk of requiring chemotherapy following molar evacuation? (Prophylactic chemotherapy)

- Uterine size much larger than gestational age
- Pre-evacuation serum hCG level > 1,00,000 IU/L
- Bilateral cystic ovarian enlargement.

11. What contraception is advisable following molar pregnancy evacuation?

Patients are encouraged to use effective contraception during the entire period of hCG follow up. If the patient does not desire surgical sterilization, the choice is either barrier contraception or progesterone only oral contraceptives. Intrauterine device is not advisable because of the potential risk of perforation. Barrier or progesterone only pills.

12. How do you diagnose persistent GTN after a non molar pregnancy?

Persistent GTN can occur after non molar pregnancies. Vaginal bleeding is a common presenting symptom but symptoms from metastatic disease, such as dyspnoea or abnormal neurology, can occur. Persistent GTN should be considered in any woman developing acute respiratory or neurological symptoms after any pregnancy. Women with such symptoms or persistent abnormal vaginal bleeding should undergo a pregnancy test.

13. What is the criteria of diagnosis of Post molar GTD?

On an average 20% patients undergoing evacuation of complete mole will develop post molar GTD. Criteria for diagnosis are:

- Rise in β-hCG titre (doubling) in one week.
- Plateauing of titre - < 10% fall in 3 consecutive weeks
- Persistent β-hCG after 10-12 weeks of evacuation.
- High levels of β-hCG > 20,000 IU after 4 weeks of evacuation
- Histological evidence of invasive mole, choriocarcinoma or placental site trophoblastic tumor.
- Evidence of metastases

15. How are the patients classified as high and low-risk?

The total score for a patient is obtained by adding the individual scores for each prognostic factor and classified as follows.

Low risk 6-8

High risk = 9

	Score			
	0	*1*	*2*	*4*
Age (yr)	*< 39*	*> 39*	-	-
antecedent pregnancy	*Hydatidiform mole*	*Abortion*	*Term*	-
Interval between end of antecedent pregnancy and start of chemotherapy (months)	< 4	4 - 6	7 - 12	> 12
β hCG (IU/liter)	$< 10^3$	$< 10^3$-10^4	10^4-10^5	$> 10^5$
ABO groups	—	O or A	B or AB	—
Largest tumor, including uterine (cm)	< 3	3-5	> 5	—
Site of metastases	—	Spleen, kidney	GIT, liver	Brain
Number of metastases	—	1-3	4-8	> 8
Prior chemotherapy	—	—	1 drug	> 2 drugs

16. What are the chemotherapy regimens for low and medium risk?

- Chemotherapy regimens for low and medium risk patients includes
- Methotrexate (MTX)—50 mg by IM injection repeated every 48 hrs × 4
- Calcium folinate—15 mg orally 30 hr after each injection of MTX (Folinic acid)
- Courses repeated every 2 weeks, i.e. days 1, 15, 29, etc.

Q.17. What is the chemotherapy regimen for high risk patients?

EMA—Co regimen

EMA

Day 1	Etoposide
	Actinomycin D
	Methotrexate
Day 2	Etoposide
	Actinomycin D
	Folinic acid rescue starting 24 hrs after Methotrexate infusion

CO

Day 8	Vincristine
	Cyclophosphamide

18. Management of drug resistant disease?

Low risk disease—Decision to alter treatment are made on progressive hCG trend over2-3 values. Low risk and medium risk patients falling Methotrexate whose serum hCG is > 100 IU/L; disease can be cured by substituting actinomycin D hCG > 100 IU/L EMA/CO.

High risk disease—Most patients who have failed EMA/CO can still be salvaged by further chemotherapy and/or surgery.

19. Outline follow up after chemotherapy, hCG

	Urine	*Blood*
Weekly for the first 6 weeks	√	√
Then every 2 weeks until 6 months	√	√
Then monthly × 12	√	x
Then 2 monthly × 6	√	x
Then 3 monthly × 4	√	x
Then 4 monthly × 3	√	x
Then 6 monthly for life	√	x

20. What is Placental Site Trophoblastic tumor (PSTT)

PSTT is a rare form of GTD that develops where the placenta attaches to the uterus. Most PSTTs do not spread to other sites in the body. They are not chemosensitive and must be completely removed by surgery.

- Relative to their mass these tumors produce small amount of hCG and Human placental lactogen (HPL). They tend to remain confirmed to the uterus and metastasizing late in their course.
- USG, hCG estimation, color Doppler, CT mainstay for investigation

- HCG regression curve helps to monitor the patients. And to decide the chemotherapy regime
- Chemotherapy (single drug therapy/multiple drug therapy) forms the mainstay of treatment
- Future pregnancies are unaffected
- Molar pregnancies should be monitored and managed in speialized clinics.

Choriocarcinoma:

- Metastatic GTT occurs in about 4% of patients after molar evacuation, but it is seen more commonly when GTT develops after non-molar pregnancy.
- Choriocarcinoma has high tendency to early vascular invasion within widespread dissemination.
- Symptoms of metastasis may result from spontaneous bleeding at metastatic foci.
  The common sites for metastasis are
  pulmonary—80%, vaginal 30%, Hepatic –10%, Cerebral—10%.

Invasive mole:

- Locally invasive GTT, occurs in almost 15% patients after molar evacuation and infrently after other gestations. Metastatic tumor occurs in 4% of cases.

Clinical Features

1. Vaginal bleeding most common symptom (84%)
2. Excessive uterine size relative to gestational age of patients. (50%)
3. Pre-eclampsia (27%)
4. Hyperemesis (25%)
5. Trophoblastic embolization (2%)
6. Theca lutein cysts 6 cm in diameter.

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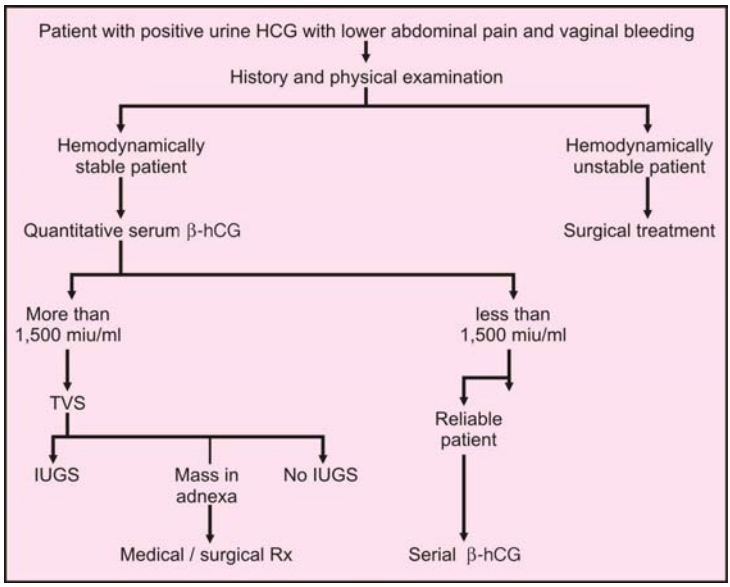
Ectopic Pregnancy

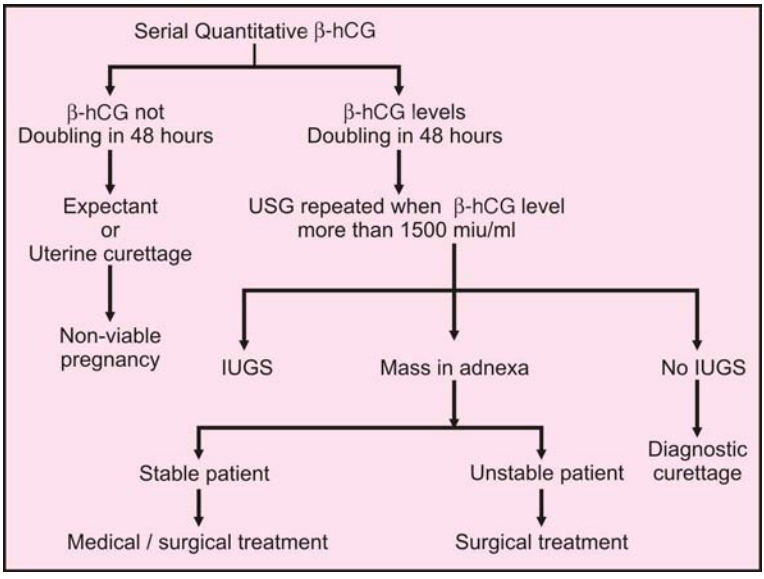
Kalpana Mahadik, Kamini Rao

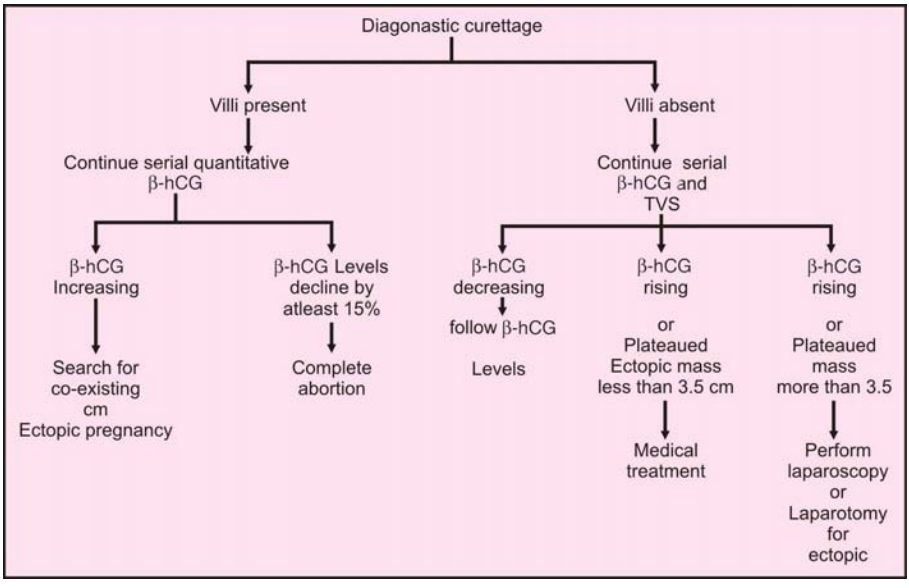
DIAGNOSIS AND TREATMENT

Abdominal pain—95%
GIT symptoms—80%
Amenorrhea with uterine bleeding—60-80%
Dizziness—58%

Per vaginal examination
Enlarged uterus
Pelvic pain with movement cervix
Palpable adnexal mass
Hypotension/tachycardia
Abdominal tenderness with guarding and rebound tenderness.







FREQUENTLY ASKED QUESTIONS

Definition

Fertilized ovum is implanted and develops outside the normal uterine cavity.

Incidence of ectopic prengnancy in natural conception and IVF conception

Natural conception- incidence—1%

IVF conception—2-8%

ICSI-1—2%

Reasons for increased incidence of ectopic in ART
Increased incidence of tubal damage in infertile patients
Larger volume of media used during transfer
Fundal placement of embryos during transfer

Etioloty of ectopic pregnancy

Which are responsible for the fertilized ovum to remain in the tube are:

- Chronic inflammatory disease of the tube
- Excessive elongation of the tube. Diverticulae, accessory ostium.
- Distortion of tube by fibroid
- Intrapelvic adhesions, tubal surgeries IUCD, minipills.

Reasons for increased rates of ectopic

Increasing STD/chronic PID
Increased contraceptive use/tubal surgery
Increasing abortion with post abortal infection
Increasing ART
Better diagnostic techniques

Is there any relation between incidence of ectopic pregnancy and age
Highest rate of ectopic pregnancy occurs between 35-44 years
4 fold increased risk in this age group compared to 15-24 years
Proposed explanation is aging may result in progressive loss of myometrial activity along the fallopian tube.

Probable mechanism by which smoking can cause ectopic pregnancy
1.6-3.5 fold increased risk of ectopic in smokers compared to non smokers.
Probable mechanisms are delayed ovulation, altered tubal, uterine motility, and altered immunity.

Role of estrogen and progesterone in ectopic incidence
Estrogen and progesterone can slow the normal movement of the fertilized egg through the tubal epithelium and result in implantation in the tube.
Women who became pregnant despite using progesterone only o.c.p. have 5 fold increased risk of ectopic pregnancy.

Relation between IUCD and ectopic

- Incidence of ectopic with non medicated IUCD—5%
- Incidence of ectopic with progesterone bearing IUCD—15%
- Incidence of ectopic after tubal sterilization—60%
- After tubal coagulation 51% of pregnancies are ectopic
- After non lap. tubal ligation 12% of pregnancies are ectopic.

General rules often used for HCG to diagnose ectopic.
hCG level should rise at least 66% in 48 hrs and at least double in 72 hours.

1. What are the fates of tubal pregnancy?

a. Tubal mole
b. Tubal abortion
c. Tubal rupture.

2. What are the clinical varieties of ectopic?

a. Acute
b. Subacute/chronic.

3. What are the clinical features of acute ectopic?

- Short period of amenorrhea
- Pain in abdomen
- Vaginal bleeding
- Fainting attack.

This is usually associated with signs of hemorrhagic shock (tachycardia, pallor and hypertension).

4. What are the clinical features of chronic ectopic?

- Apart from the short period of amenorrhea, pain in abdomen, vaginal bleeding dysuria, tenesmus
- Ill looking patient, varying degree of paller with persistent tachycardia
- Features of shock are absent.

5. Diagnosis of ectopic pregnancy

- Clinical features
- Serum B HCG —1500 MIU with midline echo on TVS confirms—diagnosis
- USG—midline echo, with adnexal mass and hemoperitoneum
- Paracentesis.

6. How will you treat ruptured ectopic?

Laparotomy—quick in and quick out, salpingectomy is the procedure of choice.

7. Will you give Anti D prophylaxis in Rh negative mother.

Yes, 50 ug IM should be given.

8. What is culdocentesis?

Aspiration of collected fluid, from posterior fornix, i.e. fluid from POD.

9. Interpretation of culdocentesis

Negative—clear fluid (peritoneal fluid)
Dry tap—Indeterminate (wrong plane)
Positive—bloody tap—(confirms ectopic or hemopertoneum).

Indication for MTX use

- Unruptured tubal pregnancy
- GA < 6 weeks
- Tubal mass not more than 3.5 cm
- Fetus not alive.

Methotrexate regimen

- Multiple dose regimen
- MTX 1 mg /kg IM on day 0, 2, 4, 6, followed by leucovorin 0.1 mg/kg on day 1, 3, 5, 7
- Increased adverse effects with multiple dose regimen—not followed.
- Single dose regimen- MTX 50 mg/m^2 IM
- Both methods are similar in efficacy.

Follow up after MTX injection
- β-hCG on day 4 and day 7 after MTX injection
- A decline in β-hCG at least 15% from day 4-7 post injection indicates a successful medical response
- β-hCG/weekly until they become undetectable.

Success rate of MTX regimen

88-94%.

What is the future pregnancy outcome
- 85% will have normal intra uterine pregnancy
- Recurrent ectopic rate—10-20%
- Recurrence after 1st ectopic—15%, and after 2nd ectopic is 30%.

Future fertility rate with laparoscopy/ laparotomy
- Laparotomy-salpingectomy intrauterine pregnancy rate—25- 70%
- Laparoscopic salpingectomy—50- 60%(similar rates)
- Rate of persistant ectopic in both groups—5-20%
- Higher rate of recurrent ectopic in laparotomy group(7- 28%) compared to laparoscopic group(6-16%). this is due to adhesions after laparotomy group.

Recurrence rate of ectopic with milking of the tube

33%.

Criteria to diagnose primary abdominal pregnancy
- Normal tubes, ovaries.
- No evidence of uteroplacental fistula
- Pregnancy exclusively in the peritoneal surface.

Sonographic criteria to diagnose abdominal pregnancy
- Visualization of sac separate from the uterus
- Failure to visualize uterine wall between the fetus and urinary bladder
- Close approximation of fetal [parts to the abdominal wall
- Eccentric position/abnormal attitude.

Spielberg criteria (ovarian pregnancy)
- Intact tube
- Fetal sac must occupy the position of the ovary
- Ovary must be connected to the uterus by the ovarian ligament
- Definitive ovarian tissue in the sac wall.

Cervical pregnancy (Rutherford criteria)

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Vaginitis

Varsha Deshmukh

HISTORY PERTAINING TO THE CONDITION

A 40-year-old lady presents with white discharge PV associated with itching locally.

ASK about

1. Age — Reproductive age group
   Senile vaginitis
   Physiological—premenstrual, midcycle.
2. Onset — Sudden (due to infection)
   Chronic (cervicitis, erosion).
3. Amount — *Scanty and thick (Candida* vaginitis*)*
   *Copious, thin and frothy (*Trichomonas vaginitis*).*
4. Color — *Curdy white (*Candida vaginitis*)*
   Reddish (senile vaginitis)
   *Greenish (*Trichomonas vaginitis*).*
5. Odor — *Foul smelling (*Trichomonas, gardenella vaginitis, *Carcinoma cervix).*
6. Prutitus—Candida vaginitis.
7. Associated symptoms—Dysuria, lower abdominal pain, pruritus.
8. Histology of diabetes mellitus, prolonged antibiotics, chronic anemia, OC pills, steroids, HIV, STD.
9. O/E-G/E—Nutritional status, anemia.
   L/E—General hygiene, marks of excoriation on thighs and vulva.
   P/S—Cervix healthy.
   Color, quantity, odor of the discharge.
   P/V—tenderness in the fornices.

QUESTIONS AND ANSWERS

1. What is leucorrhea?
Any excessive vaginal discharge that is physiological, i.e. premenstrual or midcycle is known as leukorrhea.

2. What is the source of vaginal discharge?
Vaginal discharge is not from the vagina as the vagina contains no glands. The discharge is from the cervix.

3. What are the types of vaginitis?
Candidal, Trichomonas, gardenella and senile are the different types of vaginitis.

4. What is the commonest type of vaginitis?
Candidal vaginitis is the most common type of vaginitis.

5. What is pruritus vulvae?
Itching of the local genital parts is pruritus vulvae.

6. What systemic disorders are associated with pruritus?
Hepatitis, diabetes and chronic renal failure are associated with pruritus.

7. What skin lesions can cause pruritus?
Scabies, psoriasis, and tinea infection can cause pruritus.

8. What are the predisposing factors for candidal vaginitis?
Diabetes mellitus, pregnancy, OC pills, steroids, HIV, STD are the predisposing factors for candidial vaginitis.

9. How is pregnancy responsible for increased incidence of vaginitis?
The increased oestrogen level during pregnancy makes the vaginal pH favorable for the vaginitis.

10. How does the discharge of candidal vaginitis look like?
The discharge of candidal vaginitis is typically thick ,white and curdy.

11. How does the discharge of trichomonas vaginitis look like?
The discharge of trichomonas vaginitis is copious , thin, greenish, foul smelling and frothy.

12. What lesions will you rule out on the cervix in case of excessive vaginal discharge?
Chronic cervicitis, cervical polyp, CIS, cervical erosion are some of the lesions on the cervix to be ruled out.

13. Why P/V examination is done in a case of vaginitis?
To rule out associated PID P/V examination is done.

14. What investigations will you do on the P/V discharge?
Microscopy and culture are done on the discharge.

15. Would you like to take a pap smear of the patients with P/V discharge? Why?
YES because excessive vaginal discharge can be associated with CIS.

16. In a wet mount preparation what is the characteristic feature of a) Trichomonal infection b) Gardenella infection
In trichomonal infection pear shaped motile organisms can be seen. In Gardenella infection clue cells can be seen.

17. What is added to the slide to observe Candida albicans?
10% KOH solution is added to the slide. It dissolves the debris and makes the fungus seen.

18. What is Whiff test?
When KOH solution is added to the slide there is fishy amine odor in the *Trichomonas* infection. This is a diagnostic test for *Trichomonas*.

19. What does gram staining of the vaginal discharge show?
Gram-positive spores indicate candida infection and Gram-negative diplococci indicate gonorrhea infection.

20. What culture media is specific for
a. Candida
b. Trichomonas
c. Gonococci

Candida-Nickerson's medium
Trichomonas—Stuart's medium
Gonococci—Thayer-Martin medium.

21. What is the role of blood sugar estimation in case of recurrent vaginitis?
Recurrent vaginitis is seen in patients of diabetes mellitus. The glycogen content of the vaginal cells increases thereby predisposing for vaginal infection. Hence diabetes should be ruled out.

22. What is the treatment of trichomonas vaginitis?
Both partners should receive treatment. Condom should be essentially used during intercourse. Regimes recommended are
a. Tab metronidazole 200 mg TDS for 7 days OR
b. Tab metronidazole 400 mg BD for 5 days OR
c. Single dose 2 gm tab metronidazole. Repeat same dose after one week.

23. What is the treatment of Candidiasis?
Clotrimazole is the drug of choice. It can be used in the form of cream (1%) 5 gm vaginally for 7-10 days OR vaginal pessary 100 mg for 6 days OR tab fluconazole 150 mg single dose orally.

24. What is the treatment of Gardenella vaginitis?
Tab metronidazole 400 mg BD for 7 days.

25. What is the treatment of Chlamydia infection?
Cap Doxycycline 100 mg BD for 10-14 days.

26. What is the treatment of Gonorrhoea?
Tab norfloxicillin 800 mg orally for 5 days OR tab ciprofloxacillin 500 mg orally for 5 days.

27. What is the treatment of senile vaginitis?
Local estrogen cream is used in the treatment of senile vaginitis.

28. Is the incidence of Candida vaginitis more common in obstetric practice than Gynaec practice?
In obsterics practice, during pregnancy.

29. What are the factors causing vaginal Candidas in India?
Improper hygiene, untidy undergarments, unclean nailbeds, use of tepid water for washing the urogenital parts as well as defecation.

30. What are the other factors causing Candida vaginitis?
Hot and humid atmosphere, ignorance about the local hygiene and noncompliance of the male partner in the treatment of vaginitis leads to repeated attacks of vaginitis.

31. What are the IDSA guidelines for the classification of vaginitis?
Complicated and uncomplicated are the two types of vaginitis according to the IDSA guidelines.

32. What are the clinical features of vaginitis?
Vulval pruritus, soreness, burning, dyspareunia, dysuria, erythema, noneffective and irritating discharge paravaginally are the symptoms of vaginitis.

33. What is recurrent vaginitis?
When there are 4 episodes of vaginitis in one year or 3 episodes with prolonged antibiotic therapy within one year it is known as recurrent vaginitis.

34. What is the difference between recurrent vaginitis and persistent vaginitis?
There is no symptom free interval in persistent vaginitis.

35. How OC pills are responsible for increased incidence of vaginitis?
The more the concentration of oestrogen in the pill, more are the chances of vaginitis. Estrogen encourages the increased level of glycogen in the vaginal cells which favor the growth of organisms in the vagina.

36. What are the other treatment options for Candida vaginitis?
a. Miconazole cream (2%) or 100 mg tab vaginally
b. Nystatin tab 1 lac units vaginally
c. Teraconazole cream (0.4%) OR pessary 80 mg vaginally.

37. What is the disadvantage of topical therapy over oral therapy?
Topical therapy is messy, lasts for 7 days and is applied only at night whereas oral therapy is single dose, convenient and with equally good results.

38. What is the treatment of recurrent vaginitis?
The principle of treatment of recurrent vaginitis is induction therapy with oral or topical azole followed by maintenance therapy for 6 months.

39. What is the management protocol of recurrent vaginitis?
a. Assessment of the predisposing factors-hematological assessment, control of diabetes, discontinuation of steroid therapy or OC pills.
b. Assurance to the patient
c. Maintenance therapy Azole orally for 6 months.

40. What is the rationale of long-term therapy?
The long term therapy reduces local hypersensitivity, induces a good local immunity and reduces the likelihood of recurrent vaginitis.

41. After how many days of intercourse with an infected partner the lady gets Trichomonas vaginitis?
4-28 days is the time required to contract the infection.

42. How does Trichomonas infection present in males?
In males this infection is mostly asymptomatic. However, it can present as urethritis, pristitis or epidymitis.

43. What is bacterial vaginosis?
Bacterial vaginosis is the most prevalent cause of vaginal discharge. It represents the disturbed vaginal microbial ecosystem rather than true infection. There is no vaginal inflammation.

44. What organism is responsible for bacterial vaginosis?
It has a polymicrobial etiology. No single organism is responsible for it. It is due to the disturbance in the vaginal ecosystem. It is diagnosed after ruling out candida and trichomonas infection.

45. Is bacterial vaginosis sexually transmitted?
No, however it is more prevalent in sexually active women than sexually inexperienced women. Gardenella vaginalis is also isolated from the male partners of women having bacterial vaginosis. Not all studies agree that bacterial vaginosis is sexually transmitted. Further research is required to support that it is sexually transmitted.

46. What are the organisms associated with bacterial vaginosis?
Gardenella vaginalis, mycoplasma homines, bacteroids etc. are the organisms responsible for bacterial vaginosis.

47. What are the clinical features of bacterial vaginosis?
Copious, malodorous thin white discharge paravaginally at the labia and fourchette is diagnostic of bacterial vaginosis.

48. What are the complications associated with bacterial vaginosis?
Preterm labor, chorioamnitis, postpartum endometritis, PROM, sexual dysfunction and psychological upset are some of the complications associated with bacterial vaginosis.

49. What is clue cell?

Clue cells are the vaginal squamous cells showing stippling of cytoplasm due to adherent coccobacilli.

50. Why Trichomonas is sexually transmitted?

As Trichomonas harbors in the male urethra it is sexually transmitted

51. What is "strawberry cervix"?"

It is seen in the Trichomonal vaginalis infection. "strawberry cervix" is a colposcopic finding where petechial lesions in the cervix appears as strawberry like.

52. What is "nugent criteria" in bacterial vaginosis?

It is the assessment of relative concentration of bacterial morphophytes (anaerobes than lactobacillus) characteristic of altered flora is called " nugent criteria" in a gram stained smear.

34 Sexually Transmitted Infection

Jayantdeep Nath

1. What do you mean by STI?

Sexually transmitted infection means those diseases which are predominantly transmitted through sexual contact from an infected partner.

2. Can STI transmit through other means?

STI can transmit through

1. Placenta–HIV syphilis
2. Infected nodule and blood transfusion—hepatitis B, HIV, syphilis
3. Inoculation to infants mucosa—gonococcal, chlamydia, herpes simplex candidiasis.

3. What are STIs?

STIs and causative agents are given in the following:

DISEASES	AGENTS (BACTERIA)
Gonorrhea	Neisseria gonorrhea
Non-gonococcal urethritis	Chlamydia trachomatis (D-k serotype
Syphilis	Treponema palladium
Lympho-granuloma venereum	Chlamydia trachomatis (L type.
Chancroid	Hemophilus ducreyi
Granuloma inguinale	Donovania granulomatis
Non-specific vaginitis	Hemophilus vaginalis
Mycoplasma infection	Mycoplasma hominis
VIRAL	
AIDS	Human immunodeficiency virus (HIV -1 and 2)
Genital herpes	Herpes simplex virus (mainly HSV-2).
Condyloma accuminata	Human papilloma virus (HPV)
Molluscum contagiosum	Pox virus
Viral hepatitis	Hepatitis B virus
CIN (cervical intraepithelial neoplasia)	HPV-16, 18 OR 31

PROTOZOAL	
Trichomonas vaginitis	Trichomonas vaginalis
FUNGAL	
Monilial vaginitis	Candida albicans
ECTOPARASITES	
Scabies	Sarcoptes scabei
Pediculosis pubis	Crab louse (Phthirius pubis).

4. What are the effects of STI in women?

STI may produce adverse effect in several ways in a woman

1. PID, tubal block, infertility, ectopic pregnancy, abortion, chronic pelvic pain.
2. HPV virus may cause oncogenesis of cervical, vulval, vaginal and anal epithelium, herpes simplex is involved in oncogenesis
3. Hepatitis B and C may cause jaundice.

5. Can STI harm pregnancy outcome?

- Abortion, PROM, preterm labor, chorioamnitis, IUGR, IUD.
- Congenital anomalies particularly by syphilis, herpes simplex, etc.
- Post abortal, puerperal infection.
- Difficulties in deliveries. Elective CS is recommended in herpes simplex infection.
- Obstruction or excessive bleeding during delivery in large genital warts.
- Vertical transmission from mother to fetus like HIV, syphilis.

7. Whether the incidence of STI is rising?

The incidence of STI is rising due to several reasons:

- Increasing promiscuity and permissiveness
- Change of sex partner
- Better understanding of mode of transmission
- Antibiotic resistance
- Increasing DNA viral infection
- Lack of sex education
- Increased use of pill and IUCD.

8. What do you mean by classic venereal disease?

Classic venereal disease are those which are recognized as such since long like syphilis, gonorrhea, chancroid, lymphogranuloma venereum, granuloma inguinale.

9. What is normal vaginal flora?

The normal vaginal flora is predominantly aerobic with an average of six species of bacteria, commonest one is hydrogen peroxide (H_2O_2) producing lactobacilli.

10. What is the normal pH of vagina?

The normal pH of vagina is below 4.

12. What is second Generation STI?

Most of the recently recognized STIs are now known as second Generation STIs. Example: AIDS is most recently recognized second generation STI.

13. What is "Internal Dysuria" and "External Dysuria?"

Pain during micturition is known as dysuria.

Internal dysuria: Symptomatic urethritis caused by *C. trichomatous, N. gonorrhea* and occasionally HSV are characterized by " Internal dysuria." Here, dysuria is without urinary urgency and frequency and pain occur during micturition due to some lesion internal to the labia or introitus.

External dysuria: Pain occurs during micturition due to painful contact of urine with the inflamed or ulcerated labia or introitus. This "external dysuria" is associated with vulvar herpes or vulvovaginal candidiasis.

14. What is the indication of Re-treatment therapy in case of syphilis?

Treated woman whose titer rise by four fold (two dilutines) or who do not show a fourfold (two dilutions) decrease in titre over 3 months period—should be retreated.

15. What is the most important diagnostic tests for asymptomatic Trichomonas vaginalis infection?

Polymerase chain reaction (PCR) test is Confirmative test for asymptomatic T. vaginals infection.

16. What are the concentration of treponemes in tissue generally indicates that clinical lesion may appear?

When the concentration of Treponema palladium generally reach at least 10^7/ gm of tissue before the appearance of clinical lesions.

18. What is the most sensitive test for syphilis?

Venereal disease Research Laboratory test (VDRL) is most sensitive test

19. VDRL test becomes positive after how many days of appearance of lesions?

VDRL test becomes positive after 7 days of appearance of lesions.

20. What are the causes of false positive VDRL test?

False positive VDRL test is divided into: (1) Acute false positive (< 6 months) and (2) Chronic false positive (> 6 months)

1. Causes of acute false positive (< 6 months) reaction:
   - Recent viral illness or immunization
   - Genital herpes
   - HIV infection
   - Malaria
   - Parenteral drug use.
2. Causes of chronic false positive reaction (> 6 months):
   - Aging
   - Autoimmune disease

- Systemic lupus erythematosus (SLE)
- Rheumatoid arthritis
- Parenteral drug use.

21. When VDRL test will be done in pregnant women?

VDRL test should be done ideally in every women in first prenatal visit. But in practice, when patient is coming for establishing diagnosis of pregnancy, at that time that is in first trimester it should be done. Women at high risk of exposure should have a repeat test in the third trimester and at delivery.

23. How color of vaginal discharge is best determined?

Color of discharge is best determined by examination against the white background of a swab.

24. What are the causes of painful genital ulcer? What is chancre and chancroid

Causes of painful genital ulcer:

1. Ulcerations due to Herpes simplex infection
2. "Chancroid" or soft chancre caused *by H. ducreyi*.
3. Traumatic ulcer, etc.

Chancre: It is painless, relatively avascular circumscribed, indurated and superficially ulcerated lesions. It is also known as "hard chancre". The chancre is covered by a thick, glairy exudates very rich in spirochetes.

The regional lymph nodes are swollen, discrefes rubbing and non-tender.

Chancroid: It is tender non-indurated irregular ulcerated lesion. The regional lympho nodes are enlarged and painful. It is caused by *H. devcreye*.

26. What is the name of the organism causing syphilis?

The syphilis is caused by *Treponema palladium*—a spirochaete.

27. What are the different types of syphilis?

The syphilis may be (a) primary, (b) secondary, and (c) tertiary.

28. How will you diagnose syphilis?

The diagnosis of syphilis depends on several factors.

History	H/o exposure
	Typical complain
Examination	Different relevant
	Finding

Investigation

1. Dark ground illumination (DGI).
2. VDRL— Flocculation test
   Positive after 6 weeks of initial infection
   Screening test.
3. Complement fixation Wassermann test (PTI test).

4. Specific test.
   - *Treponema palladium* hemagglutination (TPHA)
   - *Treponema palladium* immobilization test (TPI test)
   - Fluorescent treponemal antibody absorption test (FTS-abs)
   - Cenfirmitory test
   - ELISA test for IgG or IgM
   - Immunoblotting and PCR test
   - Confirmatory and sensitive.

29. How does syphilis affect the pregnancy and fetus?

The syphilis may cause late abortion, preterm labor, congenital abnormalities, IUFD. The fetal affect includes fetal antitreponemal. IgM production, sonographic, ascites, hematologic dysfunction, amniotic fluid infection, fetal VDRL reactivity, placentomegaly hepatic dysfunction, anaemia, thrombocytopenia and hydrops.

31. When the lesion of congenital syphilis generally appear?

The onset of lesions of congenital syphilis generally after the fourth month of gestation. Because fetal immunological competence begins to develop at that time. This timing suggest the pathogenesis of congenital syphilis depend on the immune response of the host rather then on a direct toxic effect of T. palladium

Another explanation is that adequate treatment of the mother before 16th with of pregnancy should prevent fetal damage.

32. What are the effect of syphilis in newborn?

The syphilitic baby may appear normal. It may develop weeks, months, years later (manual of obstetrics, Hall and Brews second Indian edition). It includes mucocutaneous lesion, anemia, edema, maculopapular rash, pomphigum on soles and palms, purulent nasal discharge (hemorrhagic sometime; hoarse cry, failure to thrive, pyrexia, widespread osteochondritis).

33. What is the radiological evidence of syphilitic baby?

The radiological examination reveal subperiosteal thickening and patchy rarefaction near the ends of the shaft.

34. How do you treat syphilis?

The syphilis is treated by:

EARLY SYPHILIS

- Inj. Benzathaine penicillin 2.4 million IM after skin test (Venereal disease—Ambrose King Fresh Edition)
- Inj. Procaine penicillin—6,00,000 IM daily × 8 days.

The penicillin is effective as low as 0.03 unit/ml in blood, but the level should be continuous.

In penicillin sensitive cases:

- Tetracycline tab 500 mg 4 times a day × 15 days
- Dexycycline tab 100 mg 2 times a day × 15 days
- Erythromycin tab 500 mg 4 times a day × 15 days.

LATE SYPHILIS

- Inj. Benzathaine penicillin
  3 million IM weekly × 3 weeks
- Inj. Procaine penicillin
  Lacs IM daily × 10 days
  Tetracycline tab 500 mg 4 hours a day × 30 days.
  Dexycycline tab 100 mg 2 times a day × 30 days
  Erythromycin tab: 500 mg 4 times a day × 30 days.

36. How will you treat congenital syphilis?

Procaine penicillin—5,00,000 unit/kg body weight

- 2,00,000 unit IM daily × 10 day.

Inj Benzathaine penicillin

- 50,000 units/kg body weight
- Painful

37. What is Jarish-Herxheimer reaction?

This is a focal and systemic reaction which sometimes follows first dose of anti syphilitic treatment. It has been attributed to the release of destructed treponemas disintegrated product.

38. How will you follow-up the cases of syphilis?

The follow up in the treatment of syphilis is essential.

Early syphilis—Monthly for 6 months, 3 monthly for 18 months
Total 2 years.

After two years

- CSF—If negative—cured.

Test—Reagin test like complement fixation test (Wassermann test)

- Quantitative serological test

Late syphilis

- At start—CSF
- Wasermann reaction,
- After 1 year—FTA-abs
- Neurosyphilis
  Datiner—Thomas concept
  Cell count in CSF normal
  Cured
  6 months, 2 years.

39. How will you treat syphilis in pregnancy?

The idea of the treatment to: (a) To treat maternal infection, and (b) to prevent congenital infection of fetus.

The penicillin is main stay of the treatment and regime are as same as non-pregnant cases. However, some give second dose of benzathaine penicillin after 1 week.

The success to prevent congenital syphilis depends on if started later than

26 weeks pregnancy, if fetal infection is already high, if born prematurely, if born immediately after start of antepartum treatment.

40. What is the alternative to penicillin treatment for syphilis in pregnancy?
There is no satisfactory alternative for penicillin for treating syphilis in pregnancy. If sensitive, desensitization should be done. Alternative drugs may not prevent congenital syphilis.

41. Which is the oldest known venereal disease? Which is the causative organism?
Gonorrhea is the oldest known venereal disease described by Hippocrates as stannary in 400 BC. It is caused by *Neisseria gonorrhea* which is a gram negative diplococci found intracellularly in group of 4 to 8.

42. What is transmission rate between male and female?
The transmission rate of gonorrhea is 75% from male to female and 25% from female to male.

43. What are the symptoms and signs of gonorrhea?
The symptoms and signs of gonorrhea include

i. Local—50% asymptomatic
   Lower genital tract like urethritis, bartholinitis, vulvitis, skenitis, cystitis, proctitis, vaginitis, cervicitis.
   Pharyngitis may occur.
ii. Pelvic infection like salpingitis with tubo-ovarian abscess. Menstrual irregularity, menorrhagia, dyspareunia, infertility.
iii. Metastatic- arthritis, endocarditis, septicemia, perihepatitis.

44. What is Fitz-Hugh Curtis syndrome?
In Gonococcal and Chlamydia salpingitis, perihepatitis is reported in 5% cases. It is called Fitz-Hugh Curtis syndrome.

45. How will you diagnose chronic gonorrhea?
The chronic gonorrhea is difficult to diagnose separately from non past history and positive gonococcal complete fixation test are presumptive.

46. How gonorrhea can present in a women?
Asymptomatic mucosal infection to disseminated gonococcal infection. All the mucosal membranes of body can be involved by *Gonococcus*.

47. How sample is collected to diagnose gonorrhea in a female?
Always to take endocervical swab with the help of large cotton tipped swab containing calcium alginate or synthetic fibres as (raw) cotton or wood may be toxic to gonococcus.

48. What type of bacteria *Gonococcus* is?
It is a gram-negative *Diplococcus* seen within polymorphonuclear cells of patient.

Presence of *Gonococcus* without polymorphonuclear cells is not necessarily indicate gonococcal infection.

49. What is the most sophisticated method of diagnosing gonococcal infection?

Detection of gonococcal ribosomal RNA in the test specimen with the help of Standard DNA probe.

Specificity 99%

Sensitivity close to culture on selective media (Thayer-Martin medium, Mcleodi chocolate agar in 5% CO_2.

50. What is the primary site of infection of gonorrhea in women?

Uterine cervix, endocervix.

51. What is the primary site of infection of gonorrhea in a hysterectomized patient?

Urethra.

52. What is the most important complication of gonorrhea?

PID.

53. What is the most common presentation of disseminated gonococcal infection?

Acute asymmetric polyarthritis and dermatitis.

54. What are few very distinctive features of chlamydia infection?

i. Obligate intracellular bacteria.
ii. Not stable extracellularly.
iii. If untreated chlamydia Infection can persist for years without much discomfort.
iv. One out of 10 infected develops symptomatic infection.
v. Highest rate of infection is found in sexually active adolescents.

55. How Chlamydia infection is diagnosed?

Isolation in a tissue culture medium.

80-90% sensitive in expert labs.

Non-culture method:

i. Direct fluorescent antibody detection — sensitivity. 80%
specificity. 97-98%
ii. Enzyme immunoassay.
iii. DNA probe (ii, iii less efficient than i).
iv. PCR very sensitive test on urine specimen.

56. What is the method of mass screening for chlamydia?

Testing first catch urine specimen of high risk population.

57. What is the impact of HIV infection on clinical manifestations of syphilis?

Increases early morbidity of CNS involvement.

That is early neurosyphilis heard only in association with HIV. Meningitis, optic neuritis, deafness, etc.

Necrotizing encephalitis is also seen. When there is concurrent HIV + syphilis. Suppression of manifestation of secondary syphilis.

58. What is the association between genital cancers and sexually transmitted infections?

The STDs associated with cancer are HPV, HIV, HSV, hepatitis B virus infection.

59. How will you investigate a case of gonorrhea?

The following investigation are done to confirm gonorrhea.

1. Smear—acute phase-reliable, chronic phase—10%
2. Culture- Thayer-Martin's media
   - 97%- combined urethral and cervical culture
3. Complement fixation test positive after ten weeks.
4. Fluorescent test- specific.

60. How will you treat gonorrhea?

The gonorrhea is treated with following antimicrobials.

Penicillin is the drug of choice
Procaine penicillin 4.8 mega unit.
Probenecid 1 g po + ampicillin 3.5 gm or amox 3 gm—Single dose
Uncomplicated:

- Ceftriaxone 125 mg im single dose.
- Cefixime 400 mg PO, single
- Ciprofloxacin 500 mg PO single dose
- Ofloxacin—400 mg single dose

Complicated
Disseminated fonococcal infection
Ceftrixone—1 g im or iv q24 hr
Ceftoaxime (Ig IV q8h)
Ceftizoxime 1 g IVq8h)
Patients allergic to β-lactam drugs
Ciprofloxacin (500 mg IV q12 h)
or
Ofloxacin (400 mg IV q12h)
or
Spectinomycin (2 g IM q12h)

Continuation therapy
Cefixime (400 mg PO bid)
or
Ciprofloxacin (500 mg PO bid)
or
Ofloxacin (400 mg PO bid)

61. How do you know the patient is cured of gonorrhea?

The permanent cure is assumed only after 3 negative smear at weekly interval.

62. What are the effects of gonorrhea in pregnancy outcome?
Gonorrhea can cause, PROM, neonatal gonococcal ophthalmia causing blindness, postpartum endometritis.

63. What is bacterial vaginosis?
It is not actual vaginal infection but a rather a clinical syndrome resulting from replacement of normal H_2O_2 producing lactobacilli with anaerobic bacteria.

64. What are the organism involved in bacterial vaginosis?
The organism involved in bacterial vaginosis are gardenella vaginalis, prevotella, molrilencucess, mycoplasma hominis.

70. What is chlamydial infection?
The infection is caused by chlamydia trachomatis, a gram-negative intracellular obligatory bacteria of D-K serotype.

71. What is the incubation period of chlamydia trachomatis?
The incubation period of chlamydial trachomatous is 7-14 days.

72. What is the clinical features of chlamydia infection?
The symptoms of chlamydia infection include dysuria, dyspareunia, post coital bleeding, intermenstrual bleeding. In 75% cases, it is asymptomatic.

The finding include mucopurulent cervical discharge, cervical oedema, ectopy and friability.

73. What are the effects of chlamydial infection.?
It causes ascending infection causing endometritis salpingitis, PID. It leads to infertility, ectopic pregnancy. The Fitz-Hugh-Curtis syndrome also occurs.

75. How will you treat chlamydial infection?
The following antimicrobials are used for the treatment of Chlamydia.

- Tetracycline—500 mg 4 times a day × 7-14 days
- Erythromycin—500 mg 4 times a day × 7-14 days
- Dexycycline—100 mg 2 times a day × 7-14 day
- Azithromycin—1 gm orally single dose
- The treatment of sexual partner is necessary.

76. What is herpes genitalis?
The herpes genitalis is caused by herpes simplex type 1 and 2. The incubation period is 3-6 days.

77. What are the presentation of herpes genitalis?
Vesicles appear on an erythematous base. The herpes genitalis starts with red painful inflamed area over clitoris, labia, vestibule, vagina, cervix and perineum. It progress to shallow ulcer and heal up by crusting. There may be fever, malaise, dysuria or retention of urine with enlarged tender inguinal lymph node.

78. What are different type of HSV infection?

The HSV infection include

- Primary—no prior infection
- Non-primary first episode of HSV-2
  Previous HSV-1 then HSV-2
- Recurrent
  Previous HSV—1 or 2 infection, repeated infection.

79. How will you confirm your diagnosis?

The diagnosis if confirmed by

Tissue culture—95% sensitive

- Tzanck smear—alcohol fixation and pap smear—70% sensitive
- PCR—highly sensitive
- Detection of virus—ELISA or immunofluorescent.

80. What are the risks of HSV infection?

The risk infection:

1. Cervical dysplasia and cervical cancer
2. Adverse effect on pregnancy—abortion, preterm labor
3. Antenatal fetal infection—5%
4. Transmission to neonates during vaginal delivery (85%) intranatal—50% (Primary) 5% (Recurrent)
5. Effect on babies
   - 10% postnatal infection
   - Localized—skin, eye, mouth—45%
   - CNS—encephalitis—3%
   - Disseminated disease—25%

81. What is the prognosis of babies?

The prognosis is very alarming in babies.

With antiviral therapy

- CNS involvement and disseminated—20-50%
- Disseminated (30%—mortality)
- Localized—good.

Without antiviral therapy

In disseminated infection:

- 90% mortality
- 90% mental retarded
- Localized—17% mortality
- 40% —morbidity.

82. How will you treat HSV disease?

Then herpes simplex virus is treated as follows.

- Antenatal period
- 36 weeks.

Acyclovir therapy

- CS is recommended in active lesion
- 85% reduced risk
- No lesion, no CS
- Ruptured membrane—antiviral therapy reasonable
- Postnatal period—good hygiene, wash hands
- No separation from mother
- Separation from other neonate
- Breast feeding allowed.

83. What drugs will you use in treating HSV infection?

Acyclovir-inhibit intracellular synthesis of DNA:

- 200 mg 5 times a day × 7 day
- 5% tropical cream of acyclovir
- Saline bath of local lesion—10% betadine douches 8 paints
- Intravenous acyclovir—5 mg/kg 8 hrly may be used
- Valacyclovir tab—500 mg BD × 7 days
- Famciclovir tab—125 mg VD × 7 days.

84. Is there any vaccine against HSV infection?

- The vaccine an HSV-2 glycoprotein
- D subunit is effective against seronegative
- HSV I and II, which is 75% effective.

85. What is genital wart?

The genital wart or condyloma accominata are papillary genital lesion caused by human papilloma virus.

86. What are the type of HPV?

Generally in 90% case HPV I and II are involved. In 10% case HPV 16, 18 and 31 are involved which are oncogenic.

87. What are the risk of HPV infection?

HPV infection can effect several ways.

- HPV 16, 18, 31 are oncogenic and can initiate carcinoma of cervix, vagina vulva, anus
- HPV can cause outlet obstruction during delivery and severe hemorrhage during delivery
- Laryngeal papilloma in infant and children—which is low (3.71)
- Poor episiotomy healing.

88. What is the presentation of wart?

The warts are usually multiple with small stalk and cauliflower like growth and can cover whole vagina. It may spread up to cervix also. It is rarely malignant. There are itching and vaginal discharge and vaginal pain.

89. Is cesarean recommended for vaginal wart?

As the vertical transmission is around 3.7%, the cesarean is not recommended. However it may be done if there is dystocia or obstruction or there is a possibility of heavy bleeding during delivery.

90. How will you treat genital wart?

The genital wart is treated by several way:

- 36-81% and 50-80% of bichbroacetic A (BCA) and trichraoacetic Acid (TCA)—Locally 3 times a weak
- 27-65% and 32-79% of podophyllin lotion—10-25%—weekly
- 33-60% and 45-88%; and 0.5% of podofilex solution or gel—5 flucouracil and imigumod 5% cream
- 22-94% of diathermy
- 21-39% and 63-88% of cryotherapy
- 29-95% and 43-93% of laser therapy
  — Surgical removal
- 0-67% and 44-61% interferon therapy
  — 1 gm cream 5 time a day × 8 weeks
  — 1 gm intralesion injection OD × 7 days

The sexual partner should be treated. The biopsy and cervical pap remove should be done.

91. How will you treat it in pregnancy?

Sometimes warts disappear after pregnancy spontaneously. Hence all wart in pregnancy may need not treatment. In pregnancy, selective therapy can be given.

- BCA or TCA
- Cryotherapy
- Laser
- Diathermy

92. Is trichomoniasis is a STD?

Yes, The trichomoniasis accounts for 20-30% of vulvovaginitis and is a STD. 70% male contract from female after single exposure. The rate of male to female is even higher.

93. What is the causative organism?

It is caused by trichomatous vaginalis, a motile anaerobic protozen. Bacterial vaginosis is associated with 60% case of Trichomonas vaginalis.

94. What is the presentation of Trichomoniasis?

The trichomoniasis may be asymptomatic. It may present with vaginal discharge like.

- Profuse, prevent, malodorous vaginal discharge vulval
- Vulval pruritus
- Strawberry cervix—patch erythema of vagina.
- PH > 5

- H/E—motile *Trichomonas* and increase leucocyte
- Clue cell may be found associated bacterial vaginosis
- Whiff test may be positive
- Postoperative cuff cellulitis after hyperectomy.

95. What is the treatment of *Trichomonas vaginalis*?

The following drugs are used in Trichomonas vaginalis.

- Metronidazol—200 mg 3 times a day × 7 days (both partner) Abstinence or use of condom
- 85% success
- Metronidazole—2 gm start
- Tinidazole—300 mg twice a day × 7 day
- 2 gm once only
- Secnidazole—100 mg once a day × 2 days
- Ornidazole
  - 1 gm orally once a day
  - 500 mg BD
  - 500 vaginally
- Ornidazole—25 mg BD × 1 day

96. How will you treat *Trichomonas vaginalis* in pregnancy?

The treatment of *Trichomonas vaginalis* in first trimester is best avoided

Vinegar douche to lower pH, tridfucun suppositories, betadine gel may be used.

- In second half of pregnancy, metronidazole treatment may be given
- Teratogenic and mutogenic effect in fetuses are not detected.

97. What are the effect of *Trichomonas vaginalis* in pregnancy?

Trichomonas vaginalis infection may cause PROM, preterm labor LBW babies

98. What is candidiasis? Is it STD?

The unlvovaginal candidiasis is caused by candida albicance in 85 to 90% cases. It is a increased fungus with branches. C glabrata and C topicalis are other fungi responsible.

99. What is combination substance?

It is the resistance of growth of opportunistic fungi by lactobacillus.

100. Why is there pruritus in candidiasis?

The pruritus is due to fungal infection. There may be hypersensitive phenomenon also.

101. What are the presentation of candidiasis?

The vulvovaginal candidas present usually as follows vaginal.

- Discharge from watery to homogeneously thick, curdy white
- Vaginal sourness, dysparunae, vulvar burning, irritation, external dysuria (splash, dysuria)
- Erythema and oedema of lubia and vulval skin.
- Pustulopapular peripheral lesion.

- Vagina is edematous with adherent whitish discharge
- Cervix appears normal.
- pH of vagina is normal
- Hanging drop preparation—80% cases.
- Wiff test negative
- H/E fungus present
- Fungal culture.

102. How will you treat candidiasis?

The general treatment of candidiasis include.

- Nystatin—100,000 units vaginally × 10-14 day
- 5,00,000 unit orally
- Imidazole derivative
- Miconazole—100 mg clotrimazole vaginal pessaries 1% vaginal cream
  - 7 days
  - 85%
- Terconazole vaginal cream or pessary.
- Ticonazole
- Fluconazole—150 mg start and after 1 week. In serious cases it may be treated after 3 days.
- Ketoconazole—pill or as put.

Recurrent or chronic infection

- Fluoconazole—150 mg for 3 days
- Ketoconazole—400 mg/day until symptom disappear.

Maintenance dose

Fluconazole—150 mg weekly for 6 months.

103. What is soft sore?

The soft sore is caused by Hemophilus decacryi, a gram -ve Streptobacillus.

104. What is the presentation of chancroid?

The presentation chancroid include multiple vesisopushil over vulva vagina and cervix. It from ulcer with inflammatory zone, anterior inguinal lymphadenitis while abscess (Buboes).

105. How will you diagnose chancroid?

The chancroid is diagnosed by

- Culture—Ducereyi bacillus
- Shoal of fish.

106. What is the treatment of chancroid?

The treatment of chancroid include:

- Ceftriaxone—150 mg im single dose
- Cotrimazole—2 tab twice day × 2 week
- Erythromycin—500 mg 6 hrly × 7 day
- Azithral—1 gm orally single dose. The partner should be treated.

107. What is lymphogranuloma venereum?
LGV is caused by L. serotype of Chlamydia trachmatis

108. What is it lesion?
The lesion include painless papule, particle or ulcer in the vulva. The inguinal glands or elderly. The glands became necrose and from abscess (bulo).

109. What are the complication of LV?
The complication of LV include:
- Vulval elephantiasis, perineal scaring and dyspareunia, rectal stricture, sinus and fistula formation.

110. How will you diagnose LGV?
The diagnosis of LGV include:
- Culture of chlamydia L1, 2, 3
- Monoclonal antibody immunofluorescent method
- LGV antigen by ELISA test
- LGV complement fixation test of rising titer
- Frei's test.

111. How will you treat LGV?
The treatment include:
- Tetracycline—500 mg 6 hrly × 2-4 weeks
- Erythromycin—500 mg 6 hrly × 2-4 weeks
- Azithromycin—Multiple dose × 3 weeks
- Sexual partner treatment
- Abscesses—Aspirated not excised
- Normal dilatation of stricture weekly.

112. What is granuloma inguinale?
It is a chronic progressive granulonation disease usually vagina or cervix. It is caused by gram-negative bacillus Donovania granulomatis.

113. What is the presentation of granuloma inguinale?
Papule the granuloma inguinale include pustules in vulva which erode the adjacent tissue. The ulcer look hypertrophic due to indicated granulation tissue. The lymph nodes do not suppurate, enlarged inguinal lymph node (Bubo) which may ulcerate

114. How will you confirm your diagnosis?
The diagnosis is confirmed by demonstration of Donovan bodies.
- Biopsy—plasma cell with Rod shaped cytoplasmic inclusion bodies (Miculicz cell)

115. How will you treat g. inguinale?
It is treated by tetracycline or 500 mg/qid orally × 3 months or chloramphenicol.

It may need local excision or vulvectomy.

116. How the hepatitis B affect pregnancy?

The hepatitis B course is not affected by pregnancy in developed countries. Fulminant hepatitis may occasionally occur. Risk of preterm delivery is increased.

117. How the fetus and neonates are affected by hepatitis B?

The hepatitis B transmission through placenta is rare. Ingestion of infected material during delivery and neonatal period may transmit disease. The breast milk also transmit. The infant may be asymptomatic or may develop fulminant disease and sele.

Table 34.1: Recommended hepatitis B virus immunoprophylaxis of neonates

HBsAg status of mother	*HBVV and HBIG*	*Age*
Positive mothers	HBIG—5 ml IM	Birth-12 hr
	HBVV 1st dose	Birth-12 hr
	HBVV 2nd	1 month
	HBVV 3rd dose	6 month
Unknown	HBIG—5 ml IM	Birth-12 hr
	HBVV 1st dose	Birth-12 hr
	HBVV 2nd HBVV	1month
	3rd dose	6 months
Negative	HBVV 1st dose	Before discharge
	HBVV 2nd	1month
	HBVV 3rd dose	6 months

Above regimen is around 85% effective in preventing development of chronic HBV infection in neonates, it is ineffective in hematogeneous tranplacental infections (15%). Pregnant female suspected of having been exposed to HBV should be treated with HBIG and inoculated with HBV vaccine. This therapy is safe and effective in pregnancy.

118. Is universal screening of pregnant woman for HBsAg necessary?

The universal screening of pregnant women for HbSAg is necessary. The vertical transmission is 10% with negative HbEAg. The neonate without prophylaxis and vaccination may get up to 40%.

119. What is the prophylaxis of neonate of HBsAg positive mother?

The prophylaxis include hepatitis B immunoglobulin (HBIG)—at birth (within 12 hr) at thigh:

- Hepatitis B vaccine within first week (10 microgram IM) in deltoid region
- 1 month, 6 months after birth.

120. What is the effect on fetus and baby with acute infection in pregnancy?

The fetus effect depends on

1st trimester—rare
2nd trimester—6%
3rd trimester—67%
Perinatal period—100%

121. Can the mother with hepatitis B handle and breast fed her baby?
The mother with hepatitis B can handle or breast fed baby after beginning of hepatitis B vaccine administration.

122. What is the vertical transmission from mother to fetus of hepatitis C?
The hepatitis C from mother to fetus is from 0 to 18%

Perinatal transmission is more with titre more than 1 million cepies per ml.

123. How will you manage of a case STD?
The proper history, adequate knowledge and confidential entry are vital. The clinical features should be properly evaluated. The diagnostic test are confirmatory adequate treatment should be started immediately. The sexual partner should be treated. The effect on pregnancy should be evaluated in detail is specially emphasized.

124. How can you prevent STI?
As a health problem, STI needs to be prevented. In the light of AIDS epidemic, the prevention and control of STI are highly essential.

The sex education, advocating barrier contraceptive and frent screening of high risk cases can reduce STI. It is vital for the health of the community that we all take on active interest in control of STD.

35 Dysfunctional Uterine Bleeding

Pralhad Kushtagi

HISTORY

Menstrual

a. Cyclicity—cycle length, regularity (preferably exact dates over 3-4 cycles)
   i. If irregular, presence of pre- or postmenstrual spotting
   ii. If acyclic, postcoital bleeding
b. Flow—amount (change of diapers/pads in a day; if heavy, need for change at night, passage of clots)

This information will help to know the pattern of bleeding (Table 35.1).

Table 35.1: Patterns of abnormal uterine bleeding and their causes

Cycle length	*Bleeding*	*Pattern*	*Cause(s)*
Normal	Prolonged/ excessive	Menorrhagia	Fibroids, adenomyosis, tuberculosis, DUB
Normal	Reduced	Hypomenorrhea	Tuberculosis, DUB
Shortened	Normal	Polymenorrhea	Pelvic inflammatory disease, endometriosis, DUB
Shortened	Excessive	Polymenorrhagia	
Prolonged	Normal/reduced	Oligomenorrhea	Polycystic ovarian syndrome, DUB, tuberculosis, long acting progestins
Acyclical	Irregular, may be excessive	Metrorrhagia	Ulcer/growth on cervix, ca-cervix/ uterus/ovary, irregular ovarian hormone intake
Normal	Spotting	Premenstrual spotting; menorrhagia	(Irregular ripening) corpus luteum deficiency
Normal	Spotting	Postmenstrual spotting; menorrhagia	(Irregular shedding) prolonged corpus luteum activity

Presence of postcoital or acyclical or contact bleeding would indicate lesion on or in the cervix and/or vagina like cancer of cervix or vagina, erosion or ulcer on cervix, atrophic vaginitis.

c. Last menstrual period and exposure to pregnancy is important. With a sudden change from a regular menstrual history to abnormal bleeding pattern, pregnancy related causes are a possibility.

Anovulatory bleeding is typically non-cyclical with an unpredictable pattern ranging from prolonged bouts of spotting to outright hemorrhage. Conversely, predictable cyclic menses, especially with premenstrual molimina (suggestive of progesterone secretion) provide the possibility that a woman is ovulating.

Associated Problems

a. Lower abdominal pain (dysmenorrhea)
   i. in relation to menstruation—before, during, after, midcycle
   ii. intensity—vague, increasing with each cycle.

   Anovular DUB is usually painless. Congestive dysmenorrhea is associated with endometriosis and pelvic inflammatory disease. Spasmodic type is suffered by adolescents where no cause is found and also the women with submucous fibroid and those using intrauterine device. Patients with endometriosis may have progressive dysmenorrhea.
b. Dyspareunia—is a common accompaniment with endometriosis and pelvic inflammatory disease.
c. Symptoms/features/indicators suggestive of:
   i. Hypothyroidism
   ii. Bleeding diathesis
   iii. Tuberculosis.

Other Attributes

a. Age—some causes are more common in some age groups. In reproductive age group pregnancy related and causes secondary to pelvic infection are more common. DUB will be the consideration in extremes of ages. However, one needs to exclude blood dyscrasias and endocrine mediated etiologies in adolescents. In perimenopausal and women of later age emphasis should be in excluding genital malignancies (Table 35.2).

Table 35.2: Causes of abnormal uterine bleeding in order of their occurrence in different age groups

<20	20-40	> 40
Coagulation defects	Pregnancy states	DUB
Endocranial defects	DUB	Iatrogenic
DUB	Infections	Neoplasm
Infections	Endometriosis, fibroid	Pregnancy states
Endometriosis	Iatrogenic	Infections
Iatrogenic	Coagulation defects	Chronic liver/renal dis.
Pregnancy states	Endocranial defects	Endocranial defects
Neoplasm	Neoplasm	Coagulation defects

b. Parity—adenomyosis and cancer cervix is more common in women with high parity, while fibroid, endometriosis and endometrial hyperplasia/cancer are associated with low parity.

c. Contraceptive use—IUCD, ovarian steroidal hormones
d. Other drug use—extraneous ovarian steroidal hormones, anticoagulants, salicylates.

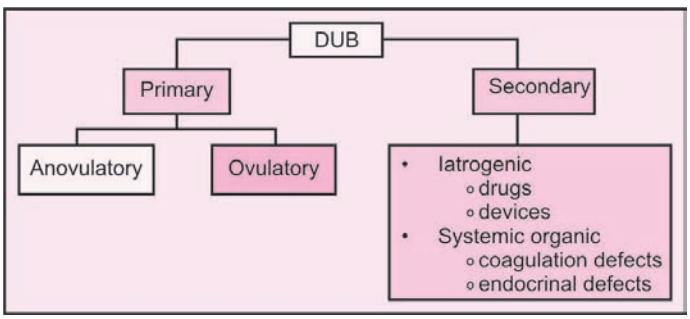
FREQUENTLY ASKED QUESTIONS (FAQS)

1. What is dysfunctional uterine bleeding?

Dysfunctional uterine bleeding (DUB) is best defined as 'abnormal uterine bleeding due to disorder in the endocranial mechanisms of menstruation or physiological mechanisms responsible for arrest of menstruation'. It is an abstract definition and the diagnosis of the entity is by process of exclusion of the causes of abnormal uterine bleeding. The working definition considered by many is 'abnormal uterine bleeding not due to organic disease or the iatrogenic cause.'

2. How is it classified? What is the basis for classification?

Having taken 'absence of clinically detectable pelvic cause for abnormal uterine bleeding' as the working definition, some authors have classified DUB as primary and secondary. The assumption is that causes like hypothyroidism, thrombocytopenia, coagulation factor defects and those due to contraceptive usage or intake of ovarian steroidal hormones, salicylates or anticoagulants which are without pelvic abnormality should warrant inclusion under secondary DUB. Primary DUB is infact the DUB and could be due to aberration in hypothalamo-pituitary-ovarian axis resulting in abnormality in ovulation and is classified as either anovulatory or ovulatory. Since abnormality in hypothalamo-pituitary-ovarian axis would logically mean anovulation, many use the terms DUB and anovulatory DUB interchangeably.



Classification of Dysfunctional Uterine Bleeding

3. Ovulation indicates functioning hypothalamo-pituitary-ovarian axis. Then despite ovulation why do some women have abnormal uterine bleeding?

The mechanism of DUB in ovulatory and anovulatory states is not fully understood. It is likely that local endometrial mechanisms are important in the pathophysiology of DUB. These local mechanisms probably involve uterine vasculature possibly influenced by prostaglandins and other vasoactive substances, altered mechanism of hemostasis, and changes in the process of

tissue breakdown and remodeling. The influence of ovarian steroids upon these processes is likely to be indirect. Apart from the local menostatic mechanism, it is the function and longevity of corpus luteum that results in abnormal uterine bleeding (AUB) patterns.

4. What could be the mechanism for anovulatory DUB?

Menstrual cycles become irregular because of acyclic estrogen production and absence of progesterone withdrawal which leads to endometrial proliferation and eventual erratic breakdown. Estrogen may also stimulate excessive endothelial production of nitric oxide in endometrium.

5. Specify local endometrial mechanisms implicated in DUB

a. Vasculature—Morphometric analysis of uterine spiral arteriole density has shown no correlation with mean blood loss, but myometrial venous density can be increased in DUB. However, it can be functional rather than anatomical changes in the endometrial vasculature that are mainly responsible for excessive menstrual bleeding.
b. Vasoactive substances—prostaglandins, endothelins and nitric oxide are the substances that could have a role in hemostasis.
   i. Prostaglandins—Ovulatory DUB is associated with a shift in the ratio of endometrial vasocostricting PGE2 alpha to vasodilatory PGI and an increase in endometrial concentration of prostaglandins. In persistently proliferative endometrium seen in anovulatory DUB, the availability of arachidonic acid is reduced and prostaglandin synthesis is impaired. Endometrial tissues could be more responsive to the action of vasodilatory prostaglandins in DUB.
   ii. Endothelins—Receptors for endothelins are predominantly located at the endomyometrial junction and are present in increased concentration just before the menstruation. Sential estradiol and progesterone reduce receptor concentrations.
   iii. Nitric oxide (endothelium derived relaxing factor)—Factors which modulate the synthesis and action of nitric oxide could lead to an increase in menstrual bleeding and might be an important mechanism in anovulatory bleeding.
c. Abnormalities of tissue breakdown and remodeling—may contribute to changes in the quantity and quality of menstrual loss. Endometrial breakdown and repair are largely controlled by local factors like lysosomes, matrix metalloproteinases, intercellular adhesion molecules, macrophages and other migratory leukocytes.
   i. Lysosomes—Tissue hypoxia following spiral artery coiling and endometrial regression and vascular stasis stimulates lysosomal activation. Lysosomal enzyme activity in the endometrium is increased in women with ovulatory DUB and that secondary to use of intrauterine devices.
   ii. Matrix metalloproteinases—are a highly regulated enzymes that can degrade most components of extracellular matrix and are expressed cyclically consistent with ovarian steroid hormones. Menstruation is

associated with change in balance between expression of these substances and their tissue inhibitors leading to tissue degradation.

iii. Macrophages and other migratory leukocytes—are increased premenstrually. Macrophages contain lysosomes and can release platelet activating factor and PGE2 that augment menstrual bleeding. Mast cells degranulate premenstrually to secrete heparin, histamine and other vasoactive substances. In DUB, endometrial secretion of heparin-like substances is increased.

iv. Intercellular adhesion molecules and platelet-endothelial adhesion molecules are thought to control binding of leukocytes to endothelial cells and breakdown these bonds may contribute to DUB.

d. Endometrial repair and regeneration seems to be governed by local factors like cytokines through neutrophil chemotaxis, epithelial regeneration through vascular endothelial growth factor and endometrial hemostasis through balanced fibrinolysis. Over activation of the fibrinolytic system may unbalance the hemostatic system, causing early breakdown of thrombi in the endometrial vessels and excessive blood loss.

6. Diagnosis of DUB is based on the assumption that there is no detectable pelvic pathology. If there is a detectable fibroid, can the patient still have DUB?

Fibroid, like anovulatory DUB is a result of hyperestrenic condition. Cervical, broad ligament and subserous fibroids do not produce menstrual abnormality through structural changes in uterus. AUB in such conditions could be considered as DUB. Studies on myomata related bleeding suggest that it is not the increased surface area of the endometrium or local compression of vessels that causes bleeding. Nor does it seem that myomas have any effect on the hypothalamic pituitary ovarian axis. Instead the likely cause of the problem appears to be growth factor dysregulation causing uterine vascular dysfunction. It may then be argued that such dysfunction and dysregulation occurs because of fibroids and hence the AUB can not be considered as DUB.

7. If a patient of uterine prolapse without decubitus ulceration has AUB, could it be considered as DUB?

Apart from the obvious decubitus ulceration and vascular erosion, pelvic congestion in the early stages of prolapse following altered uterine and ovarian circulation (venous congestion) due to anatomic change may influence ovarian steroidogenesis and cause AUB. In the menopausal older age group, estatrophy could result in DUB.

8. What are the necessary investigations for the workup of a case thought to be of DUB?

Investigations are ordered with the intention to know the effect, the type and to exclude other causes of abnormal uterine bleeding (AUB).

a. Hemogram—at least hemoglobin estimate with hematocrit, in all cases. Anemia could be effect as well as the cause. Local tissue anoxia may interfere with tissue regeneration and vascular hemostatic activity.

b. Pelvic ultrasonography—will be of value for all cases in any age group to exclude pelvic causes of AUB and to note the endometrial pattern. Sonohysterogram will be an additional aid for ruling out intrauterine pathology.
c. Coagulogram—restricted to bleeding/clotting time studies with platelet counts especially in adolescents will reveal the cause for abnormal uterine bleeding patterns.
d. Thyroid function tests—the estimation of thyroid stimulating hormone for adolescents and perimenopausal groups will help to exclude subtle hypothyroid state which is not an uncommon cause for AUB.
e. Uterine curettage—should be carried out in all cases of perimenopausal and menopausal age groups to over rule underlying neoplastic lesions. It will provide insight into type of endometrium and ovulatory status for specific hormonal manipulation when empirical treatment has failed in the mid reproductive age groups. It will not be required for adolescents since anovulatory DUB is more of a norm in them. Microbiologic analysis of menstrual blood of first day may help exclude genital tuberculosis in the latter.
f. Other endocrine studies—like assay of follicular stimulating and luteinizing hormones are to be carried out if the patient is obese or features of hirsutism, infrent cycles are present in adolescents and early reproductive age. Prolactin estimation is ordered when the patient fails to respond to medical management or has galactorrhea.

9. If uterine curettage is advised, what is the basis?

Uterine curettage is advised in perimenopausal and menopausal women with recurrent episodes of AUB to rule out uterine adenocarcinoma. In younger women not responding as anticipated to the medical management, the curettage is timed to precede next menstrual period to obtain information about ovulatory status (type of DUB) and the pattern of endometrium. In patients with continuous excessive bleeding, thorough curettage may have a therapeutic benefit. When carried out, curettage may curette out endometrial mucosal polyps, help diagnose submucous fibroid during the procedure and genital tuberculosis on histopathology.

10. Does the knowledge of type of endometrium in DUB influence the management?

The histological type of the endometrium will reflect the hormonal influence and mostly helps in tailoring the management for the case. Anovulatory endometrial patterns can be undone by mimicking the ovulatory effect through supplementation of progestins in the latter half of menstrual cycle or in cases where fertility is also a concern by inducing ovulation. The ovulatory patterns may be set right by inhibiting ovulation or through altering the local menostatic mechanism (e.g. PGSIs, antifibrinolytics, vessel wall stabilizers, hematinics, etc.). It should be remembered that atrophic endometrium though is of anovulatory type would require estrogen supplementation; where as,

ovulatory endometrial patterns due to deficient corpus luteum activity would require progestins (Table 35.3).

Table 35.3: Endometrial type, hormonal status and treatment

	Type of endometrium	*Estrogen*	*Progesterone*	*Directed treatment*
1.	**Anovulatory**			progestins
a.	Proliferative	normal	absent	(from D15-25) or ovulation induction (when infertility is a concern)
b.	Cystoglandular hyperplasia	?	absent	as above
c.	Adenomatous hyperplasia—simple	??	absent	option 1 progestins (from D15-25) and follow up option 2 hysterectomy -if menopausal or perimenopausal - with positive history *family *hyperestrinic states *obese/hypertensive, diabetic)
d.	Adenomatous hyperplasia—complex	???	absent	
e.	Atrophic/ threshold	?	absent	estrogen
2.	**Ovulatory**			
a.	Secretory	normal	present	local menostatics
b.	Irregular ripening (from D15-25.	normal	?	progestins
c.	Irregular shedding	normal	persistent CL	estrogen-progestin combination pills

CL = corpus luteum

11. What is 'threshold bleeding?'

It is the irregular bleeding because of thin endometrium due to poorly developed proliferative phase. In such cases amount of estrogen is less—enough to reach threshold for bleeding, but not enough to produce a full proliferative phase. It is seen in young girls and in perimenopausal phase.

12. What is the mechanism of action of progestins for use in anovulatory DUB?

Apart from it being logical (the patient has AUB because she has not ovulated; and mimicking or inducing ovulation should set it right), the use of progestins

i. help convert potent estradiol to less estrogenic estriol through stimulation of 17 beta hydroxyl steroid dehydrogenase and sulfotransferases.

ii. hinder estrogen receptor replenishment by inhibiting estrogen's induction of its own receptor
iii. suppress estrogen mediated transcription of oncogenes, which property is harvested for reversing premalignant/malignant changes.

13. How do estrogens act to reduce bleeding?

Estrogens are used in the management of DUB because of their effect

i. on capillary/small vessel bleeding by increase in fibrinogen, factors V and IX, and platelet aggregation
ii. on tissue reactions to bradykinin, mucopolysaccharides and capillary permeability
iii. of endometrial proliferation.

14. What is/are the indication/s for hysterectomy in DUB.

The following could be considered as cases for hysterectomy

a. Women not responding to medical management, showing significant effect of bleeding and are beyond 35-40 years of age in whom fertility is not a concern
b. Perimenopausal women who are at risk of endometrial carcinoma
   i. family/ personal history of adenocarcinoma
   ii. known cases of chronic anovulation—polycystic ovarian syndrome, endometriosis
   iii. obese with hypertension and/or diabetes, even if with simple endometrial hyperplasia
   iv. complex and/ or atypical adenomatous hyperplasia
c. Postmenopausal women with
   i. thick endometrium or hazy endomyometrial junction
   ii. repeated episodes of vaginal bleeding.

15. What is the risk of progression of anovulatory endometrial patterns to endometrial carcinoma?

As the extent of unopposed estrogenic action increases, the risk of endometrial carcinoma increases (Table 35.4).

Table 36.4: Type of anovulatory endometrium and risk of endometrial carcinoma

Endometrial pattern	*Risk of endometrial carcinoma (%)*
Simple hyperplasia	1
Complex hyperplasia	3
Simple hyperplasia with atypia	8
Complex hyperplasia with atypia	25

16. When will you consider that a patient has failed to respond to medical management?

A patient of DUB is started and continued for 3 to 6 cycles on the initially planned medical management, hormonal or otherwise. They are then observed

for about 3 cycles without any drugs. If the irregularity persists while on drugs or recurs after several attempts at the medical management, it can then be considered as treatment failure.

17. Can hysterectomy be avoided in patients not responding to medical management?

Levonorgestril intrauterine systems (LNG-IUS), endometrial ablation methods and in some situations uterine artery embolization may help avoid resorting to hysterectomy.

18. Intrauterine devices are thought to cause AUB and LNG-IUS is found to be useful in women who failed to respond to progestins. What is the basis for the claim?

The daily dose of levonorgestrel (20 micrograms) causes desexualization of endometrial stroma, atrophy of endometrial glands, a surface papillary pattern, and a stromal inflammatory infiltrate. The drug being released directly at the endometrium makes these effects effective.

19. What is endometrial ablation? Who are the candidates for the procedure? What are the different methods used?

Endometrial ablation is destruction of endometrium. It could be considered for women with no intrauterine pathology not responding to medical therapy and having length of uterine cavity not bigger than 12 cm. Patients with anovulatory DUB are not good candidates for endometrial ablation. There are varieties of global endometrial ablation devices using different energy sources like hot water, cryocautery, microwave, laser, and radio frency electricity.

20. How will you manage a patient reporting with continuous excessive bleeding?

In extreme circumstances, parenteral therapy with intravenous conjugated estrogens (25 mg) will usually arrest an acute bleeding episode. GnRH agonists have also been used because of their effect of initial stimulation of endogenous estrogen. Intravenous doses of tranexamic acid and/or etamsylate may also help in reducing the bleeding. Parenteral therapy should be followed by 2 to 3 weeks of oral progestins. In such patients where hysterectomy is planned, to tide over the bleeding episode tablets of danazol or injection of testosterone are used successfully while awaiting fitness/preparedness for surgery.

21. Is it possible to correlate pattern of DUB to endocrine and histological changes?

DUB can have its origin in an endocrine imbalance or can occur in normal menstrual cycles (ovulatory DUB).

Table 35.5: Correlation between menstrual patterns with ovarian and endometrial changes in menstruation

Ovulation	*Phase changes*	*Endometrial histology*	*Menstrual pattern*
Normal	Short FP	Normal	Polymenorrhagia, menorrhagia
Normal	Long FP	Normal	Oligomenorrhea, menorrhagia
Abnormal CL	Short LP	Normal with luteal lag or deficient secretory (irregular ripening)	Premenstrual spotting, menorrhagia
Persistent CL	Long LP	Well developed secretory	Prolonged cycles
Anovulation (insufficient follicles)	Short cycle	Deficient proliferative	Polymenorrhagia, metrorrhagia
Anovulation (polycystic ovaries)	Prolonged cycle	Proliferative or hyperplastic	Oligomenorrhea, metropathia hemorrhagic

CL = corpus luteum, FP = follicular phase, LP = luteal phase

MULTIPLE CHOICE QUESTIONS—ONE BEST ANSWER (MCQs)

1. Which of the following is *not* the characteristic of anovulatory DUB?

A. Noncyclic bleeding
B. Painless bleeding
C. Premenstrual molimina
D. Spotting alternating with heavy flow
E. Unpredictable bleeding

2. The nonsurgical treatment of choice for DUB when effectiveness, side effects, and acceptability are considered:

A. Danazol
B. Levonorgestrel intrauterine system
C. Nonsteroidal anti-inflammatory agents
D. Oral contraceptive pills
E. Tranexemic acid

3. Most likely cause of abnormal uterine bleeding in a nulligravida, obese, hirsute woman with normal pelvic examination findings is:

A. Anovulation
B. Medications
C. Mucosal polyp

D. Persistent corpus luteum
E. Theca cell tumor.

4. The most appropriate next step in the management of irregular, scanty vaginal bleeding in a lactating woman who is taking depot medroxy progesterone, is:

A. Combination oral contraceptives
B. Endometrial biopsy
C. Intravenous estrogens
D. Observation for 3 months
E. Progesterone supplementation.

5. Which of the following is *not* the side effect of Danazol?

A. Deepening of voice
B. Hirsutism
C. Leucopenia
D. Tinnitus
E. Visual disturbance.

6. Which is the treatment option for a woman who returns 6 months after the endometrial ablation with amenorrhea, severe cyclical abdominopelvic pain described by her as cramps?

A. Combined oral contraceptives
B. Hysterectomy
C. Levonorgestril intrauterine system
D. Mefenamic acid
E. Repeat endometrial resection.

7. Which of the following statements about northeasterone are correct?

A. Is found to be no more effective than placebo in the short term treatment of menorrhagia
B. Is not recommended for medical treatment of menorrhagia
C. It is a 19-carbon item containing synthetic progestin derived from testosterone
D. It is a 21-carbon atom containing naturally occurring progesterone
E. It is the most prescribed (50%) medical treatment for menorrhagia.

8. Complication that is *not* commonly associated with endometrial ablation is:

A. Fluid overload
B. Hematometra
C. Infection
D. Thermal damage
E. Uterine perforation.

9. The rationale for the therapeutic use of conjugated estrogens for the immediate treatment of DUB is that estrogen:

A. Causes decasualization
B. Leads endometrial proliferation

C. Reduces platelet adhesiveness
D. Stabilizes endogenous clotting factors
E. Withdrawal results in uniform endometrial slough.

10. An obese woman aged 45 years who has fibroids reports with irregular heavy vaginal bleeding in the last 6 months. What is the most appropriate next step in the management of this patient?

A. Combination oral contraceptives
B. Endometrial biopsy
C. Hysterectomy
D. Myomectomy
E. Reassurance and observation for 3 months.

Answers

1.C; 2.B; 3.A; 4.D; 5.D; 6.B; 7.C; 8.C; 9.B; 10.B

36 Amenorrhea

Bharati Shrihari Dhore Patil

1. What is amenorrhea?

Complete absence of menstruation for a period of at least 6 months in a woman in reproductive age.

2. What is the most common physiological cause of amenorrhea?

Pregnancy.

3. What is the difference between primary and secondary amenorrhea?

Primary amenorrhea: (incidence is less than 0.1%)

a. The absence of menses in a female who has never menstruated by the age of 16 years regardless of the normal growth and development of secondary sexual characteristics, or
b. The absence of menses in a female who has never menstruated by the age of 14 years in the absence of the normal growth or development of secondary sexual characteristics.

Secondary amenorrhea (incidence is about 0.7%)

1. The absence of menses for longer than 6-12 months, or
2. The absence of menses for a total of three previous cycle intervals.

4. What are the most common causes of primary amenorrhea?

- Gonadal failure, such as Turner's syndrome.
- Gonadal agenesis accounts for one-third of all patients with primary amenorrhea.
- Müllerian anomalies, such as uterovaginal agenesis (Rokitansky-Küster-Hauser syndrome), are the second most common causes, accounting for 20% of primary amenorrhea.
- Such anomalies occur in 1 per 4,000 female births.
- One-third of patients with gonadal failure have associated cardiovascular or renal abnormalities.

5. A 14-year-old female patient comes with complaints of monthly abdominal pain but no menses. What is possible diagnosis?

Imperforate hymen or transverse vaginal septum.

6. What is Kallmann's syndrome?

Primary amenorrhea that is secondary to inadequate GnRH release and insomnia. These patients have infantile sexual development.

7. Primary amenorrhea and normal breast development but absence of uterus occurs in which two syndromes?

a. Patients with androgen insensitivity or testicular feminization are XY genotype but phenotypically are females because an androgen intracellular receptor is not functioning (a maternal X-linked recessive gene). Androgen induction of the wolffian duct system does not occur despite normal male levels of testosterone. Müllerian inhibiting factor (MIF) is still present, so the müllerian system does not develop. These patients typically have large breasts with immature nipples but no axillary or pubic hair.

   Some incomplete testicular feminization does occur with some pubic hair, axillary hair, and phallic development in patients with Lubs's, Reifenstein's, Rosewater's, and giblet Dreyfus's syndromes. These patients should be allowed to reach normal sexual maturity. After age 20, the gonads should be removed because 20% of these patients will develop a gonadoblastoma or dysgerminoma.

b. Patients with müllerian agenesis have sexual hair and mature nipples; 40% have associated renal anomalies, so an intravenous pyelogram or ultrasound should be performed.

8. Which rare enzyme defects can cause amenorrhea?

17-alpha-hydroxylase deficiency and 17-20-desmolase deficiency.

17-alpha-hydroxylase deficiency prevents the formation of sex hormones. In a 46XX patient, this leads to the absence of breast development even though the uterus is present a 46XY patient, there is no breast development; the uterus is absent because of the presence of MIF. These patients also have hypernatremia, hypovolemia, and hypertension because of increased mineralocorticoid production. There is excessive sodium retention and potassium excretion. Cortisol production is decreased. Therefore, these patients require cortisol as well as sex hormone replacement to attain breast development and prevent osteoporosis.

17-20-desmolase deficiency also prevents the formation of estrogen and testosterone, but not aldosterone, cortisol, or progesterone. These patients do not have breast development.

9. A 15-year-old female patient gives H/o amenorrhea. She is in the 25th percentile for height and weight. She is not an athlete and has an appropriate diet. On physical examination, she has no evidence of breast growth or pubic hair; vagina and uterus are present. What work-up will help determine the cause of amenorrhea?

The patient has no evidence of estrogenization. Serum gonadotropin levels will help to determine if the patient has gonadal failure or unstimulated gonads.

An elevated serum gonadotropin level indicates gonadal failure. A karyotype will usually show an X-chromosome abnormality, causing gonadal dysgenesis. If the patient has an XY chromosome, the gonads must be removed after bone growth. Hypogonadism, indicates unstimulated gonads. These patients must be evaluated for intracranial tumors by checking thyroid function and levels of growth hormone, prolactin, and cortisol. Pituitary stimulation tests may also be appropriate. Skull X-rays to detect calcifications and to evaluate the sella turcica and a CT/MRI of the pituitary region is also recommended.

10. What is athletic amenorrhea ? Should it be treated?

Amenorrhea in athletic women with no other etiology. The cause is not well understood, although many of these patients have eating disorders as well as high stress levels. Many of these women are hypoestrogenic with significant loss of bone density that can lead to osteoporosis and stress fractures. These patients should be encouraged to improve their diet, decrease their stress levels, decrease the amount of strenuous exercise, if possible, and replace their estrogen and progesterone, if other chances do not increase estrogen levels.

11. What percentage of amenorrhea is due to hyperprolactinemia ?

10-20%.

12. How do stress and exercise causes amenorrhea?

Stress and exercise cause the level of catecholamine estrogens and beta endorphins to increase; this influences the release of GnRH by acting on the neurotransmitters. Without appropriate release of gonadotropin- releasing hormone (GnRH), follicle-stimulating hormone (FSH) and luteinizing hormone (LH) are not release appropriately and anovulation occurs, which may lead to amenorrhea.

13. How does weight loss cause amenorrhea?

At less than 15% of ideal body weight, normal release of GnRH does not occur. At less than 25% of ideal body weight, not only GnRH release abnormal, but pituitary release of LH and FSH is abnormal and the pituitary gland cannot be induced to secrete LH and FSH normally, even if GnRH is supplied in the normal pulsatile function.

14. Which drugs cause amenorrhea?

- Any drug that stimulates prolactin secretion
- Antipsychotic, such as phenothiazine derivatives, haloperidol and droperidol (Inapsine)
- Tricyclic antidepressants
- Antihypertensive, such as reserpine and methyldopa
- Antianxiety agents, such as benzodiazepines
- Other drugs, such as metoclopromide (Reglan), opiates, barbiturates, and estrogens.

15. Which psychiatric disorder in adolescent is a major cause amenorrhea?

Anorexia nervosa.

The incidence of this disorder is 1/1,000 white adolescent females. Besides amenorrhea, these patients have severe weight loss, often have bradycardia, hypotension, and have low T3 levels from impaired peripheral conversion of T2 to T3. The mortality rate is 5-15%.

16. What are the pituitary causes of amenorrhea?

Damaged cells—lack of LH and FSH secretion caused by anoxia- thrombosis, or hemorrhage, as in Sheehan's syndrome (related to hypotension in pregnancy) or Simon's syndrome (unrelated to pregnancy).

Neoplasms—Most common of which secrete prolactin, but are not always associated with galactorrhea.

Amenorrhea is also associated with acromegaly and Cushing's syndrome.

17. Which initial blood tests should be performed in the patient with amenorrhea?

Thyroid function tests are to rule out rare cases of asymptomatic hypothyroidism in patient with hypothyroidism, regular menses will resume with thyroid replacement.

Prolactin levels should be measured to rule out hyperprolactinemia, which may occur even without galactorrhea. Patients with galactorrhea should also undergo skull X-ray with a coned-down view of the sella turcica or a CT scan or MRI to rule out a small pituitary adenoma.

18. What is post-pill amenorrhea?

Suppression of the hypothalamic-pituitary axis can persist for several months after discontinuing oral contraceptive agents. The incidence of post-pill amenorrhea lasting greater than 6 months is equal to the incidence of secondary amenorrhea. It is no longer believed that oral contraceptives cause prolonged amenorrhea or infertility, and thus other etiologies must be considered.

19. What is premature ovarian failure?

Ovarian failure and, thus, amenorrhea before age 40.

These patients have symptoms of hypoestrogenism, increased levels of FSH and generalized sclerosis or only primordial follicles with no progression past the antrum stage on ovarian biopsy.

Many patients have autoimmune diseases, such as Hashimoto's syndrome, Addison's disease, and hypoparathyroidism.

If the patient is less than 25-year-old, a karyotype should be performed to determine if the patient is a 46 XX/46 XY mosaic pattern. If so, the gonads must be removed to prevent malignancy after age 20.

Antithyroid antibodies, antinuclear antibodies, and 24 hours cortisol levels should also be checked.

Ovarian failure may also result from irradiation or chemotherapy.

20. What is a progesterone challenge test?

10 mg of oral medroxyprogesterone acetate for 5 days or intramuscular injection of 100-200 mg of progesterone in oil.

The test is positive if any bleeding (even spotting) occurs within 2 weeks of the test. A negative pregnancy test should be obtained first.

21. What does a positive progesterone challenge test indicate?

The patient has a serum estradiol level greater than 40 pg/ml, and thus the anterior pituitary is producing LH and FSH and the endometrium and outflow tract are functioning. This indicates that, if thyroid function tests and prolactin levels are normal, the patient is anovulatory.

22. Which tests should be performed on a patient with amenorrhea after a progesterone challenge test reveals no bleeding?

A course of conjugated estrogen, 2.5 mg for 21 days prior to progesterone challenge, should be used to define a uterine defect as the cause of amenorrhea, such as complete destruction of the endometrial lining by Asherman's syndrome or fibrosis following severe endometritis. Next, the patient's FSH level should be determined. If FSH is elevated, the patient has premature ovarian failure. If FSH is normal, a CT scan of the hypothalamus-pituitary area should be done to rule out a central nervous system tumor, such as craniopharyngioma, or granulomatous diseases (tuberculosis, sarcoid). The patient's reserve of adrenocorticotropic hormone (ACTH) should be checked. If all of these tests are normal, the diagnosis of exclusion is hypothalamic-pituitary failure. All of these patients are hypoestrogenic and require hormone replacement therapy.

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Endometriosis

Sudha Sharma, Vishal R Tandon

1. What is Endometriosis?

The presence of functioning endometrium (glands and stroma) in sites other than uterine mucosa is called endometriosis. Ectopic endometrial tissue may be found in the myometrium than it is called adenomyosis. More commonly these tissues are found at sites other than uterus and are generally referred to as endometriosis.

2. What are endometrial cells?

The endometrium is the mucous surface that lines the inside of the uterus. It contains several layers of cells (i.e., endometrial cells) that vary in appearance and number throughout the menstrual cycle, as the levels of estrogen and progesterone fluctuate. During the luteal phase (the two-week period just before a woman bleeds), for example, the endometrium is thick, its cells are enlarged, the glands bulge, and the arteries are swollen. At menstruation, the endometrium sheds. Following menstruation, new cells grow and the endometrium regenerates. The cells that make up the endometrium normally grow only inside the uterus are called endometrial cells.

3. What is the epidemiological correlation?

- Endometriosis is a disease of the childbearing period. Women are usually 30 to 40 years old at the time of diagnosis. A familial tendency has been identified.
- Its incidence appears to be on increase, partly due to improvements in diagnostic technique and partly due to changing social patterns like late marriage and limitation of family size. It is more commonly amongst the affluent class.
- Evidence of the disease is present in 20 percent of women undergoing laparoscopic investigation for infertility. Approximately 24 percent of women who complain of pelvic pain are subsently found to have endometriosis.
- The overall prevalence, including symptomatic and asymptomatic women, is estimated to be 5 to 10 percent. Because surgical confirmation is necessary for the diagnosis, the true prevalence of the disease is unknown.

4. What is its pathogenesis?

Pathogenesis is not well understood and is probably multifactorial in origin.

Retrograde menstruation (Sampson's theory)

The most widely embraced theory involves retrograde menstruation. This means retrograde flow of menstrual blood through tubes during menstruation which enters the pelvis. While this theory can explain pelvic endometriosis, it fails to explain the endometriosis at distant sites.

Direct implantation

According to this theory the endometrial or decidual tissue starts to grow in susceptible individual when implanted in the new sites. Such sites are abdominal scar following hysterotomy, cesarian section, tubectomy and myomectomy. Endometriosis at the episiotomy scar, vaginal or cervical sites can also be explained by this theory.

Coelomic metaplasia (Meyer and Ivanoff theory)

Coelomic metaplasia refers to cells that transform into endometrial cells, perhaps as a result of chronic inflammation or irritation from retrograde menstrual blood. This theory of "coelomic metaplasia" is based on the observation that coelomic epithelium is the common ancestor of endometrial and peritoneal cells, thus allowing transformation of one type of cell into another.

Metastatic theory (Lymphatic theory (Halban) and vascular theory)

It very difficult to explain the occurrence of endometriosis at less accessible sites like the umbilicus, Pelvic lymph nodes, ureter, rectovaginal septum, bowel wall and remote sites like lung ,pleura, endocardium and extremities. Hence, this might be possible through embolization of menstrual fragments trough vascular or lymphatic channels leading to launching of endometriosis at distal sites.

Congenital factors

Endometriosis may be a congenital condition (existed from birth). During fetal development, uterine tissue may remain in the pelvis and grow as a result of hormonal influence. Müllerian remnants can differentiate into endometrial tissue.

Hormonal theory

Ovarian steroid hormones probably play an important role in the induction and maintenance of the disease, as symptoms improve in cases of low hormone levels. Medical treatment that suspends cyclic ovarian function (oral contraceptives, medroxyprogesterone acetate, danazol, gonadotropin-releasing hormone agonist) or surgical removal of the ovaries will result in symptom improvement. Pregnancy causes atrophy of endometriosis mainly through high progesterone levels. Endometriosis is rarely seen before puberty and it regresses after menopause.

5. What recent hypothesis are emerging explaining pathogenesis of endometriosis?

Role of oxidative stress in endometriosis

The presence of elevated concentrations of free radicals and lowered antioxidant potential leads to oxidative stress. The development of oxidative stress in the local peritoneal environment may be one of the links in the chain of events leading to endometriosis. Antioxidant supplementation, immuno-modulators, and selective progesterone receptor modulators with antioxidant effects have been investigated as possible treatments for endometriosis, but compelling evidence on the benefits of the various modalities is lacking.

Genetic and immunological theory

Genetic basis of endometriosis probably accounts for less than 10% of patients. There is increased incidence in 1st degree relatives. Multifactorial inheritance is thought. Endometriosis is more frently diagnosed in patients with infertility than in a normal population scion arises, is there immunological link between endometriosis, recurrent miscarriage and implantation failure. This can possibly be explained by alterations in humoral and cell-mediated immunity in women with endometriosis. Humoral immunological changes include increased formation of antibodies against endometrial antigens, antilaminin-1 autoantibodies and other autoimmune antibodies (e.g. antiphospholipid). Cell-mediated immunological changes include alterations in peritoneal and follicular fluid immune cells and cytokines. The possible negative effect of these immunological changes on folliculogenesis, ovulation, oocyte quality, early embryonic development and implantation in women with endometriosis suggests that infertility in endometriosis patients may be related to alterations within the follicle or oocyte, resulting in embryos with decreased ability to implant.

6. How endometriosis is classified

Stages of Endometriosis

Stage I—Minimal	Few or superficial implants are evident in the early stages of endometriosis
Stage II - Mild	More implants, deeper involvement
Stage III - Moderate	More implants; ovaries affected, adhesions present
Stage IV - Severe	Similar to Stage III, but multiple and more dense adhesions present.

7. What are common sites of endometriosis?

It is seen widely spread throughout the lower pelvis usually below the level of umbilicus. Most often it is seen in the ovaries, posterior cul-de-sac including uterosacral ligaments, in the adnexal region, fallopian tubes, pelvis, peritoneum over bladder, sigmoid colon, back of the uterus, intestinal coils, Pelvic lymph nodes, rectovaginal septum and appendix.

8. Which are less common sites?

Umbilicus, ureter, bowel wall and remote sites like lung, pleura, liver, endocardium and extremities. It is also reported to be seen over scar following hysterotomies, classical caesarean, myomectomy, amputated stumps of cervix, scar of valva and episiotomy.

9. What is most common pathology of endometriosis?

Early implants look like small, flat dark patches or flecks of blue or black paint ("powder-burns") sprinkled on the pelvic surfaces. The small patches may remain unchanged, become scar tissue or spontaneously disappear over a period of months. Endometriosis may invade the ovary, producing blood filled cysts called endometriomas. With time, serum gets absorbed and the blood darkens to a deep, reddish brown or tarry color, giving rise to the description "chocolate cyst." These may be smaller than a pea or larger than a grapefruit. In some cases, bands of fibrous tissue called adhesions may bind the uterus, tubes, ovaries, and nearby intestines together.

10. What are Endometriomas/Chocolate Cysts?

This disease process is also known as Endometriosis of the ovaries. Tiny implants of cells that line the uterine cavity become transplanted and form small cysts on the outside of the ovary. These cysts enlarge and produce endometriosis of the ovary. They respond to hormone stimulation during the menstrual cycle and produce many small cysts, which may then occupy and even replace the normal ovarian tissue. These endometriomas are filled with a thick chocolate-type material, which is the reason they are known as "chocolate cysts". When this type of ovarian cyst ruptures, the material spills over into the pelvis and onto the surface of the uterus, bladder and bowel and the corresponding spaces between. Adhesions can develop because of this rupture and may lead to pelvic pain.

11. Can endometriosis be cancerous?

The endometrial tissue may invade neighboring tissue, but it is not a cancer. In rare instances advanced endometriosis may be associated with ascites, pleural effusions, and large pelvic masses. Interestingly, in only 8% of the cases in one recent study recorded cancerous changes of the ovary diagnosed suggesting possible malignant transformation however it still remains to be confirmed.

SYMPTOMS

Pain

Endometriosis should be considered in any woman of reproductive age who has pelvic pain as it is the most common symptom that generally occurs just before and during menstruation and then decreases after menstruation. About 25 to 67% of women with endometriosis suffer congestive dysmenorrhea (painful menstruation). About 25% experience painful intercourse. Pelvic examinations can also be painful. Depending on the location of the implants, rectal pain and painful defecation may also occur. The diagnosis of

endometriosis should be considered especially if a patient develops dysmenorrhea after years of pain-free menstrual cycles. Abdominal pain or low back ache also can be chief complaints.

Infertility

Infertility is common. In fact, infertility is the most common symptom that prompts a visit to the doctor's office for diagnosis. Endometriosis is diagnosed in up to 60% of infertile women.

12. What can be the possible reasons for infertility?

Extensive scarring interferes with tubal motility or entirely blocking the tubes. Ovarian dysfunction can also be one of the reasons. Increased or decreased secretion of various cytokines that regulate the immunologic processes has been shown to occur among women with endometriosis. These changes may affect sperm, egg function, or embryo development. Abnormal sperm motility has been shown in association with endometriosis. It has been also suggested that endometriotic implants may secrete substances that interfere with normal sperm motility and thereby impair the process of fertilization.

Menstrual symptoms

Menorrhagia is common with adenomyosis and irregular bleeding may occur with cervical and vaginal bleeding. Polymenorrhea is noted with ovarian involvement.

Other symptoms

Painful or difficult defecation, diarrhea, melena and bloody urine if sigmoid or rectum and bladder are involved. If the lungs are involved, endometriosis may cause pleuritic pain) or hemoptysis. If endometrial cells implant in the brain, the patient may experience seizures.

Medical history

About 90% of women with pelvic pain have endometriosis. Pelvic pain that is typical of endometriosis includes menstrual cramps, low back pain that worse during menstruation, and pain in the pelvis that occurs during or after sexual intercourse. Depending on where the implants are located, a woman may feel pain in her rectum during defecation.

Physical examination

The expected positive findings are pelvic tenderness, nodules in the pouch of Douglas, nodular feel of the uterosacral ligaments, fixed retroverted uterus and unilateral or bilateral adnexal mass of varying sizes. Rectal or rectovaginal examination is often helpful to confirm the diagnosis.

Diagnosis

Pelvic ultrasonography, computed tomography and magnetic resonance imaging are occasionally used to identify individual lesions, but these modalities are not helpful in assessing the extent of endometriosis.

Laparoscopy

Most women with endometriosis have normal pelvic findings, and laparoscopy is necessary for definitive diagnosis. It permits to see inside the pelvic region to observe classic lesion of endometriosis.

13. Are there any biochemical markers for diagnosis of endometriosis?

Many patients with endometriosis have an elevated CA-125 blood level (CA-125 is an antigen).

Differential diagnosis of endometriosis by symptom

- Generalized pelvic pain
- Pelvic inflammatory disease
- Endometritis
- Pelvic adhesions
- Neoplasms, benign or malignant
- Ovarian torsion.

Dysmenorrhea

- Primary → Spasmodic
- Secondary → Congestive.

Dyspareunia

- Musculoskeletal causes (pelvic relaxation, levator spasm)
- Gastrointestinal tract causes urinary tract (urethral syndrome, interstitial cystitis) infection
- Pelvic vascular congestion diminished lubrication or vaginal expansion because of insufficient arousal.

Infertility

- Male factor
- Tubal disease (infection)
- Anovulation
- Cervical factors
- Luteal phase deficiency.

14. What are the treatment modalities?

Following modalities are of use:

- Expectant—minimal endometriosis with no other pelvic findings like in unmarried or young married females.
- Hormones and others
- Surgery
  Conservative
  Radical
- Combination of surgery and hormones.

15. What is medical treatment of endometriosis?

Drug

- Combined estrogens and progestogens
- Progestogens (such as dydrogesterone, medroxyprogesterone acetate, norethisterone)
- Synthetic androgens (such as danazol, gestrinone)
- Gonadotrophin releasing hormone analogues (such as buserelin, gasserian, leuprorelin acetate, nafarelin, triptorelin)
- Nonsteroidal antiinflammatory drugs (such as diclofenac, ibuprofen, mefenamic acid)
- Gonadotrophin releasing hormone analogues and any combined hormone replacement therapy (continuous or sential) or tibolone
  The combined oral contraceptive pill for endometriosis.

16. What are the main advantages of OC pills?

The main advantages of the pill are that it is inexpensive and is usually reasonably well tolerated by women. It can also be taken safely for many years if necessary, unlike most of the other hormonal drug treatments for endometriosis.

How it works?

Like all the other hormonal treatments, the pill does not cure endometriosis. Rather, it alleviates the pain of endometriosis by suppressing menstruation and inhibiting the growth of the endometrial implants.

Progestins as a treatment for endometriosis

The progestins are effective treatments for the symptoms of endometriosis. However, like all the hormonal drugs used for endometriosis, they have side effects, which some women find intolerable. They are safer and cheaper than the GnRH-agonists and danazol, which some gynecologists believe makes them appropriate for women who need prolonged or repeated treatments.

How it works?

It is not known precisely how progestins relieve the symptoms of endometriosis, but they probably work by suppressing the growth of endometrial implants in some way, causing them to gradually waste away. They may also reduce endometriosis-induced inflammation in the pelvic cavity. The progestins control pain symptoms in approximately 3 out of 4 women. However, they may not relieve symptoms completely.

Does its use after surgery has any role?

There is some evidence to justify using hormonal drug treatments following surgery to suppress the growth and development of any remaining or new endometrial implants.

Use in recurrent endometriosis

Repeat courses of progestins may be used for women with recurrent endometriosis.

Danazol as a treatment for endometriosis

Danazol is a synthetic androgen. Danazol is an effective treatment for endometriosis, and has the same effectiveness as the other hormonal treatments. However, it has many androgenic (male-like) side effects, including weight gain, increased body hair and acne. Its unpleasant side effects and its tendency to adversely affect blood lipid (cholesterol) levels mean it is not usually the first choice of treatment for endometriosis.

How it helps?

It suppresses its growth and development temporarily, so the disease may recur following treatment. Danazol has a multitude of effects on the body. Some of these effects combine to produce high levels of androgen and low levels of oestrogen in the body. This hormonal environment stops menstruation and suppresses the growth of endometrial implants, causing them to degenerate. The symptoms of endometriosis usually begin to diminish by the end of the second month. Most women will resume ovulating and menstruating within 4 to 6 weeks of stopping treatment. It relieves pain in approximately 90% of women.

GnRH-agonists as a treatment for endometriosis

They are modified versions of a naturally occurring hormone known as gonadotropin releasing hormone, which helps to control the menstrual cycle.

How they work?

When used continuously for periods of longer than 2 weeks, they stop the production of oestrogen by a series of mechanisms. This deprives the endometrial implants of oestrogen, causing them to become inactive and degenerate. They appear to be at least as effective as progestins in relieving pain.

NSAIDs

These drugs can be effective in alleviating pain and inflammation but they do not reduce the size of the implants or treat the source.

How they help?

It is thought that much of the pain of endometriosis, especially menstrual pain, is due to inflammation that may be caused in part by high levels of "bad prostaglandins." Women with endometriosis have been shown to produce an excess of a prostaglandin called PGE2, which causes inflammation, pain, and uterine contractions. Theoretically, NSAIDs would seem to be a good choice for relieving menstrual pain because most of them work by blocking the production of all prostaglandins.

17. What are the recent advances in the medical treatment?

- Gonadotrophin releasing hormone analogues and any combined hormone replacement therapy (continuous or sential) or tibolone
- Aromatase inhibitors as a treatment for endometriosis: At the moment, the treatment of endometriosis with aromatase inhibitors is still

experimental, because the research is still in its early days. Research has shown that aromatase is also found in high levels in the ectopic endometrial tissue of women with endometriosis, which contributes to the growth of their endometriosis. Aromatase inhibitor suppresses the growth of their endometriosis, and reduces the associated inflammation. This, in turn, significantly reduces their pelvic pain.

- GnRH agonist treatment combined with tibolone
- Tumour necrosis factor-{alpha} blockers
- Leflunomide—an immunomodulator—induces regression of endometriosis.

Surgery

Surgery is used to treat moderate to severe cases of endometriosis.

Laparoscopy

A laparoscopy is usually the only surgical option for women who want to preserve fertility. A laparoscopy can be used to freeze the implants, burn them with a laser, or surgically remove them, depending on the type of lesion. In a laparascopy done for diagnostic purposes, the lesions are usually removed at the same time.

18. What is an endometrioma?

An endometrioma is a mass of tissue (noncancerous cyst or tumor) that contains shreds of endometrial tissue.

19. How are endometriomas treated?

Several surgical treatments are available for endometriomas:

- Simple puncture—Draining the fluid from the cyst.
- Ablation—Drain the cyst and remove its base with laser or electrosurgery.
- Cutting away of the cyst wall—This is the procedure of choice to decrease recurrence of disease.
- Draining, drug therapy and surgery—Endometriomas can also be drained, treated with medication, and later removed by surgery.

Laparotomy

In severe cases, however, and in women who choose not to preserve fertility, a laparotomy may be necessary.

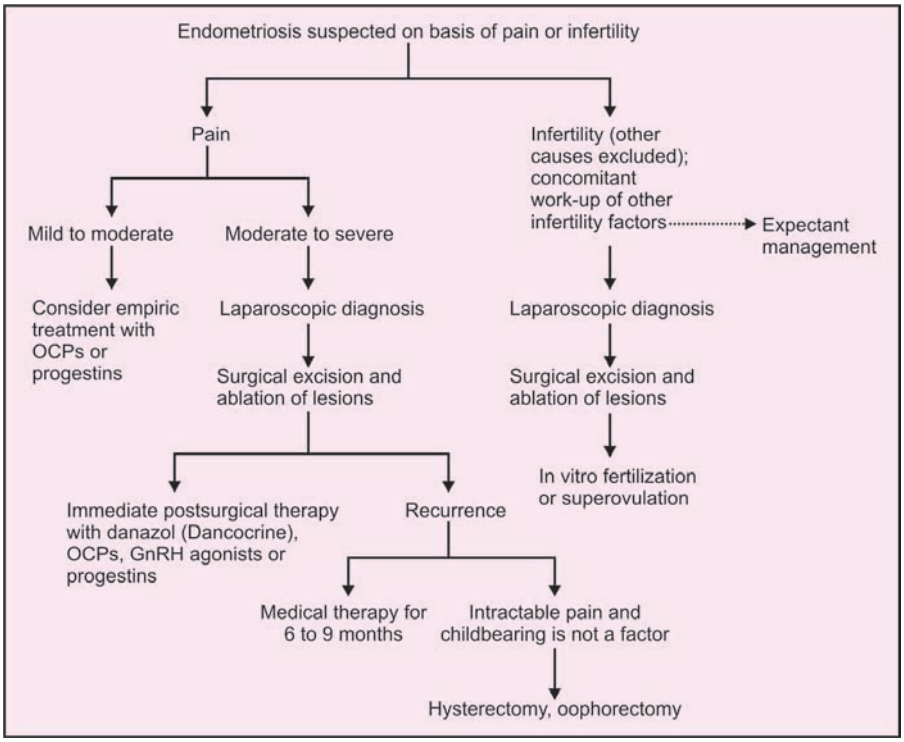
20. How is advanced endometriosis treated?

Laparoscopy or by laparotomy (traditional abdominal surgery, which requires a larger incision) is the management of advanced endometriosis.

21. Is laparoscopy more effective than laparotomy?

Laparoscopy and laparotomy are equally effective in relieving pain and improving fertility. Endometriosis recurs in about 20 percent of cases over 5 years in both procedures. Patients who undergo laparoscopy, however, experience a more rapid and less painful recovery.

22. What treatment algorithm should be followed?



Primary Prevention of Endometriosis:

- Going for pregnancy
- Oral contraceptives use
- Tubal patency test should be avoided just before the commencement of menstruation
- Operations on the genital tract should be scheduled in postmenstrual period
- Classical cesarean section and hysterotomy should be avoided to prevent scar endometriosis.

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Menopause

Girija Wagh, Sanjay Gupte

1. What is menopause? What is climacteric?

The menopause is that point in time when permanent cessation of menstruation occurs following the loss of ovarian activity (men: month, pause: cessation).

The years prior to menopause marked by irregularity of cycle which encompass the change from normal ovulatory cycles to cessation of menses is called premenopausal transition.

Climacteric (ladder): more general term which indicates the period of time when a woman passes from the reproductive stage of life through perimenopausal transition and the menopause to the post-menopausal years.

2. What is menopausal syndrome?

Menopausal transition usually begins in women's forties. During this time, women report of varied physical and psychological symptoms which together comprise the menopause.

3. What are the symptoms of menopause?

1. *Immediate:*
   a. Menstrual irregularity
   b. Vasomotor: Flushes and night sweats (75%)
   c. Psychological: Irritability (92%), lethargy (88%), depression (78%), forgetfulness (62%), crying spells (42%), and insomnia (51%)
   d. Urinary dysuria (20%)
   e. Local.

2. *Intermediate*:
   - Genital
   - Skin and hair
   - Musculoskeletal
   - Joint and muscle spasm (48%)
   - Sexual disturbances (decreased libido (20%))
   - Ocular
   - Weight gain (61%).

3. *Late (hidden):*
   - Osteoporosis
   - CVS—palpitations (44%).

4. What are the changes in the gonadotropins at menopause?

Shortly after menopause, there are no ovarian follicles. There is 10-20 fold increase in FSH and approximately 3 fold increase in LH. These hormones reach maximum levels 1-3 years after menopause. Then they gradually decline. High levels of FSH confirm ovarian failure.

5. Why are the FSH levels higher than LH levels?

LH is cleared from blood faster. Half-life for LH is 20 minutes and for FSH is 3-4 hrs. There is no specific negative feedback peptide for LH like inhibin.

6. How is the fertility potential reduced at menopause? How can one assess the ovarian reserve?

10- 15 years before menopause, there is significant decline in fertility. There is subtle increase in FSH during this period. The increase is due to the quality and quantity of aging follicles. Once the oocyte pool reduces to approximately a 1000 follicles, menopause starts. Decreased fertility may occur without any obvious changes in the menstrual cycle. Despite of decreased fertility, pregnancy is possible.

The functional ovarian reserve can be directly measured by a basal day-3 serum FSH level.

7. How to differentiate between menopausal irregular uterine bleeding and abnormal uterine bleeding?

No unusual presentation or definition of irregular periods is approved. On an average 4-8 years prior to menopause, irregularity in cycle is observed in majority of women.

Most common 3 parameters are seen:

1. Oligomenorrhea (70%)
2. Menorrhagia, metrorrhagia (18%)
3. Sudden amenorrhea.

8. What type of abnormal uterine bleeding requires investigation?

1. Heavier than usual bleeding (blood loss > 80 ml, clots +, anemia)
2. Prolonged bleeding (> 7 days)
3. Frequently menstrual period (cycles < 21 days)
4. Metrorrhagia
5. Postcoital bleeding.

9. What are hot flashes, hot flushes, night sweats? What is their etiopathogenesis and cause?

The symptoms of vasomotor instability are called hot flashes. Some distinguish the Hot Flash from Hot Flush. However, the terms are interchangeable.

A hot flash is a sudden transient sensation that ranges from warmth to ontense heat. A sudden wave of hot sensation spreads over the body, especially the upper body and face (causes flushing). The skin temperature rises as the blood flow to the skin increases, especially in the fingers and toes, where skin

temperature can rise 1-7 degrees. This can follow by an experience of a chill because of reduction of the core temperature.

Hot flashes that occur with drenching diaphoresis (perspiration) during sleep are termed as night sweats. They can occur frently, i.e. hourly or infrently (monthly or weekly). Some women experience them during the pre-menopausal transient phase especially in association with menses.

The precise cause is not known. Thermoregulatory centre situated in the hypothalamus is under the control of catacholamines and catachol-estrogens. There are estrogen receptors present in these centres. The estrogen present in blood combines with catacholamines to produce catachol-estrogen. When there is lack of estrogen, there is an imbalance between catachol and catachol-estrogen, which results in hot flashes.

It is doubtful whether it is estrogen dependent but it has shown a dose dependent response to estrogen supplementation.

10. What is the presentation and cause of urinary and fecal symptoms in menopause?

Urinary complaints like urinary incontinence, recurrent UTI, the urethral syndrome are common in postmenopausal women. Some women can also have fecal incontinence as a result of menopausal changes. Persistent involuntary leaking of urine, i.e. urinary incontinence of all forms affected 10 to 30% women between the age group of 50-64 years. The prevalence of urge incontinence increases with number of postmenopausal years while stress incontinence is more common during perimenopause.

The female urethra and trigone of bladder share the same embryonic origins as the vagina. Therefore they contain estrogen receptors which are affected by the reduced level of estrogen. So urinary incontinence occurs as a result of reduced bladder and urethral tissue tone.

Various other types of urinary presentation like frency, urgency, nocturia are also seen to be associated after middle-age.

Urethral syndrome is characterized by recurrent episodes of urinary frency, dysuria and urgency. Urethral mucus membrane atrophy leads to urge incontinence which is characterized as sudden, strong desire to void, can be many a times misdiagnosed and treated as UTI.

15-26% of women with urinary incontinence also have fecal incontinence. This is due to lowered estrogen level giving rise to neuromuscular dysfunction of the pelvic floor. The anorectum is found to be abundant with estrogen receptors.

11. Why are menopausal women susceptible to urinary tract infection?

Lack of estrogen results in shortening and shrinking of the distal portion of the urethra. This reduces the defence against offending pathogens and increases the risk of UTI.

Reduced endogenous estrogen levels leads to reduction in the epithelial cell glycogen production. This increases the alkalinity of the vaginal, urethral, and bladder environment.

12. Enumerate the factors leading to urinary incontinence during menopause.

1. Reduced endogenous estrogen levels
2. Urethritis and cystitis
3. Neuromuscular dysfunction of the pelvic floor
4. Previous childbirth injuries or damages
5. Substance abuse like smoking, alcohol and caffeine
6. Obesity
7. Medications like diuretics and tranquilizers
8. Neurological disorders like Multiple Sclerosis and Parkinson's disease.

13. Describe the androgen levels at menopause.

The two sources of androgen secretion are: ovaries and the adrenal glands.

The ovary secretes androstenedione and testosterone, and after menopause, predominantly androstenedione.

The androstenedione levels in the blood reduce to half of that seen before menopause. The source of this is mainly from the adrenal gland. The levels of DHEA and its sulphate, DHEA-S, which originate in the adrenal gland, decline markedly with aging, and about 70- 74% less respectively after about 10 years after menopause.

Testosterone production decreases by approximately 25% after menopause, but in most women, the ovary secretes more testosterone after menopause. With the disappearance of follicles and estrogen, the elevated gonadotropins stimulate the ovarian stroma to secrete more testosterone. The total level of testosterone after menopause, however, is reduced because there is no peripheral conversion of androstenedione. Compared with young women, the overall androgen exposure of perimenopausal women is less.

14. Describe the estrogens at menopause.

Postmenopausal women have serum estradiol levels below 15 pg/ml, and mean estrone levels of 30 pg/ml. Although estradiol levels are negligible it has been observed that estrone levels are higher in older men and women. Older women convert androstenedione to estrone. The circulating estradiol levels after menopause is derived from the peripheral conversion of estrone, which intern is derived from the peripheral conversion of androstenedione. This is because the adipose tissue aromatase mRNA expression increases and is the highest in the hip fat, and the lowest in the abdomen. The average postmenopausal production rate of estrogen is approx. 45 mg/24 hr. The percent conversion of androstenedione to estrogen correlates with body weight. This probably is due to the ability of fat to aromatize androgens and reduce levels of SHBG. These low levels of estrogen are insufficient to sustain secondary sex tissues.

	Premenopausal	*Postmenopausal*
Estradiol	40-400 pg/ml	10-20 pg/ml
Estrone	30-200 pg/ml	30-70 pg/ml
Testosterone	20-80 ng/dl	15-70 ng/dl
Androstenedione	60-su ng/dl	30-150 ng/dl

15. Why is there hirsutism at menopause?

The androgen to estrogen ratio changes drastically after menopause, because of the more marked decline in estrogen. With increasing age, a decrease can be measured in circulating levels of DHEA and DHEA-S, whereas androstenedione, testosterone and estrogen remain constant. Mild hirsutism is a reflection of this marked shift in the sex hormone ratio.

16. What is the impact of menopause on the CVS?

Menopause worsens serum lipid and plasma fibrinogen profiles. Estrogen deficiency adversely affects circulating lipid levels, leading to increase in low-density lipoproteins, cholesterol, total serum cholesterol and triglyceride levels. Menopausal estrogen deprivation leads to reduction in HDLP. This positively correlates with adiposity at the trunk and central fat deposition.

Atherosclerosis develops rapidly after menopause. Triglycerides increase in these women and if at levels of 400 mg/dl or higher are a strong risk factor for CHD.

As women grow older their rates of myocardial infarction and stroke approach and then exceed that of men.

In younger women throughout adulthood the HDL cholesterol levels are about 10 mg/dl higher and total and LDL cholesterol levels lower, thus being cardioprotective.

17. How should one evaluate a menopausal woman?

Medical evaluation includes a complete history and physical examination with a focus on the target areas affected by menopause, namely, the CVS, the skeleton, the genitourinary system and the neuroendocrine system.

The stionnaire should include information with respect to hot flashes, sleep disturbances, mood and memory changes and urinary symptoms.

The medical, gynecological, and sexual history along with exercise habits will help assess the health status of these women.

A dietary history should include a specific enquiry into fat and calcium intake.

Physical examination should include blood pressure measurement, examination of thyroid, breast and the CVS. The abdominal and pelvic examinations will also be part of this routine.

Following lab investigations should be performed:

1. Total cholesterol levels and HDL cholesterol levels—if they are deranged, 12 hr fasting lipid profile including triglycerides should be evaluated.

Marked hypertriglyceridemia is an independent risk factor for coronary artery disease in women.

2. FSH and estradiol levels are usually not needed to diagnose menopause. FSH level of > 20 IU/L and estradiol levels of 20-40 pg/ml confirm primary gonadal failure.
3. A PAP smear and a screening mammogram, or at least a thorough breast examination should be performed on menopausal women.

18. What is the effect of exogenous hormones on CVS?

Several observational and epidemiological studies had strongly suggested that ERT could reduce cardiovascular deaths significantly. These studies inspired confidence that estrogen therapy would reverse the negative impact of menopause on the lipid profiles. The angiographic studies had suggested that estrogen could reverse atherosclerosis and improve tone and resilience of the vascular system.

Eg:

1. Leisure World Study, a prospective longitudinal study, documented reduced risk of death due to myocardial infarction.
2. The Lipid Research Clinics Follow-up Study (LRCFS) demonstrated 63% reduction in the relative risk of cardiovascular disease.
3. A population based case-control study in Minnesota concluded 45% reduction in risk of MI in estrogen users.
4. The Nurses Health Study (NHS) found decreased risk of CVD in current users, suggesting that estrogen should be used indefinitely for cardio-protection.
5. The Postmenopausal Estrogen Progesterone Intervention trial (PEPI) suggested beneficial effects of estrogen on lipid profile, except was associated with elevated triglycerides. But, it concluded that other beneficial vascular effects like endothelium dependent vasodilatation, relaxation of smooth muscles, inhibition of lipoprotein oxidation, inhibition of foam formation, decreased fibrinogen levels and direct inotropic effect on the heart were beneficial, and it recommended conventional HRT.

But all these positive effects of HRT were counter-proved by many studies, where it was in fact now accepted that estrogen is not actually cardioprotective. In fact it is associated with risk of CVD's.

1. Finnish study of pooled data from 22 small short term randomized studies found 39% increase in cardiovascular events in estrogen users. This was the first break in the pattern of support for estrogen.
2. The large and respected Frankingham Heart study reported 50% increased risk of CVD's in estrogen users.
3. The Heart Estrogen/Progestin Replacement study (HERS): A carefully designed large scale prospective multicentered placebo-controlled trial of combined therapy for secondary prevention of CHD in postmenopausal women had 2783 postmenopausal non-hysterectomized women with established CHD or at least 50% occlusion of a major coronary artery as subjects. Their mean age is 66.7 years and were randomized to placebo or the most commonly used HRT, i.e. 0.625 mg of oral Conjugated Equine

Estrogen + 2.5 mg of Medroxy Progesterone Acetate daily. The primary endpoints of this study were non-fatal MI or CHD deaths.

The trial began in 1993 and ended after 4.1 years with 179 primary CHD events in the hormone group.

The study concluded that CEE and MPA were not to be used for secondary prevention of CHD, and received endorsement to this effect by the FDA. The subsent HERS II trial, which completed the stipulated time frame also failed to show any cardiovascular benefit.

4. The Women's Health Initiative (WHI) trial, the largest study of menopausal women in which 16608 women were randomized to receive HRT (0.625 mg CEE + 2.5 mg MPA C.C. HRT) or placebo. The trial was scheduled for 10 years, but altered prematurely in July 2002 (5.2 years) when it was discovered that the risk of invasive breast cancer exceeded in HRT group. It also showed increased rates of coronary events, stroke and pulmonary embolism with two-fold increase in risk of venous thromboembolism with HRT group.
5. Estrogen replacement and atherosclerosis (ERA) trial and women's estrogen for stroke trial (WEST) also did not find cardio protective benefits for estrogen.

19. What is the role of estrogen with respect to memory and cognition?

Estrogens play an important role in memory and learning which involves acquisition, encoding and consolidation of new information.

Recently, using sophisticated functional neuro-imaging technique like MRI, SPECT (positron emission tomography), etc. Estrogens have been shown to modulate cerebral blood flow, neurotransmitter function and glucose utilization in postmenopausal women. Increase in cerebral blood flow was particularly noticed in the hippocampus, para-hippocampus, gyrus and temporal regions which are a part of the memory circuit. But, the very recent component study of WHI, named WHIM, has indicated absence of estrogen-induced neuroprotective role with respect to memory and cognition. The HRT increased the risk of probable dementia in postmenopausal women aged 65 years and older.

20. What is postmenopausal osteoporosis? How serious is the problem?

Osteoporosis is a systemic skeletal ds characterized by low bone mass, and a deterioration of micro-architecture of bone fragility as a result of which there is in fracture rate with little or no trauma. Osteoporosis commonly affects the site of cancellous bone earlier and more severely than cortical bone, resulting in increased incidence of osteoporotic fractures in areas with predominance of cancellous bone, namely, vertebral bodies, proximal femur and distal forearm. It is the most prevalent bone problem in the elderly.

21. What is BMD? How do you assess it correctly?

Bone mineral density is bone mass. It increases throughout the growth period and is completely achieved in young women by late adolescence. Therefore, normal diet and normal hormonal environment during adolescence is important to achieve higher peak bone mass. After 30 years, the bone loss

occurs at the rate of 0.7% per year. after menopause, due to estrogen deficiency, the bone loss is accelerated up to 2% per year for 10 years (postmenopausal osteoporosis type 1). The bone loss continues at a much slower rate of 1% per year as the aging related loss (senile osteoporosis type 2).

The BMD can be correctly assessed by DEXA- scanning (Dual energy X-ray Absorptiometry). It is fast and simple, non-invasive method with minimal radiation exposure and can measure BMD at all sites. It has a precision of 1 to 2% with the pencil-beam machine and 0.5-1% with fan-beam machine. At least 2 major osteoporotic sites are scanned. The report gives us total BMD in gms/cm sq as well as T-score and Z-score along with a graph with the fracture threshold.

T-score is defined as the patient's BMD as compared to the peak bone mass in normal young adults. While, Z-score is the patient's BMD compared to the peak bone mass in age-to-sex matched adults.

When T score is less, it indicated bone loss over number of years, probably due to postmenopausal or senile changes. If Z scores are less than-2, the bone loss is due to some pathological cause and needs to be investigated.

22. Describe the effects of estrogen on bone.

Estrogens in women are essential for formation and maintenance of sufficient bone mass. Both the osteoclasts and the osteoblasts have estrogen receptors. Estrogens act directly on the osteoclasts by inhibiting lysosomal enzyme production and prevent bone resorption. Estrogenic action on osteoblasts directly is however not known but it acts indirectly through osteoblasts on osteoclasts. The estrogen promotes production of interleukins from the osteoblasts which inhibit the osteoclast activity.

23. Describe the effects of calcium on bone.

90% of the calcium in the body is in bones. A normal woman stores about 1000 gm of calcium. The 1% of calcium is in circulation and in soft tissue and is necessary for muscle contraction and transmission of nerve impulse. A decrease in calcium intake and/or absorption lowers the serum level of ionized calcium. This stimulates PTH secretion to mobilize calcium from bone by direct stimulation of osteoclastic activity. A deficiency in estrogen is associated with greater responsiveness of bone to PTH. Thus for any given level of PTH there is more calcium removed from bone, raising the serum calcium.

24. What are the risk factors for osteoporosis?

1. Unmodifiable risk factors:
    a. Female sex
    b. Premature menopause < 40
    c. Late menarche
    d. Short stature
    e. Slender built
    f. Ethnic origin (Asian, Caucasian)
    g. Nulliparity
    h. Family history of osteoporosis
    i. Fair skin.

2. Potentially modifiable risk factors:
   a. Smoking
   b. Physical inactivity (burkha)
   c. Low dietary calcium
   d. Low vitamin D
   e. Alcohol, caffeine and nicotine abuse
   f. Estrogen deficiency (as in menopause)
   g. Hyperthyroidism
   h. Hyperparathyroidism
   i. Hyperadrenalism
   j. Anticoagulant therapy
   k. Glucocorticoid use.

25. Describe the clinical features of established osteoporosis.

Most of the osteoporotic postmenopausal women are asymptomatic till fractures occur.

1. Back pain—is a major clinical symptom of vertebral compression fractures. The pain with a fracture is acute, then subsides over 2-3 months and lingers as chronic low back pain due to increased lumbar lordosis. There is loss of height and postural deformities called the "kyphotic Dowager's Hump". 50% women more than 65 years of age have spinal compression fractures (2/3rds are unrecognized). Each complete compression fracture causes loss of approximately 1 cm in height. The commonest sites for vertebral fractures are T12, L1,L2L,3 vertebrae.
2. Colle's fracture—a ten fold increase is seen from age 35 to 60.
3. Hip fracture—0.3 -20% incidence is observed with increasing age from 45 to 85 years. 8% of all hip fractures are associated with osteoporosis.
4. Tooth loss—oral annular bone loss is strongly associated with osteoporosis.

26. How do you diagnose osteoporosis? What are the WHO criteria?

Osteoporosis can be easily diagnosed on plain X-ray spine by prominent horizontal trabeculae and wedging or compression of dorso- lumbar vertebrae due to gross demineralization. But by this time, 40% bone loss has already occurred and patient is in advanced stage of osteoporosis. Early diagnosis should be done in the stage of osteopenia where X-ray is subjective. DEXA scanning is a gold standard to diagnose osteoporosis.

The WHO defines osteoporosis in terms of BMD t-score:

Bone density	*T- score*
Normal	> -1.0
Osteopenic	-1.0 to -2.5
Osteoporotic	< -2.5
Severely Osteoporotic	< -2.5 + 1/>1 fragility fractures

27. What are the NOF guidelines for prevention and treatment of osteoporosis?

National Osteoporosis Foundation 2000 Guidelines suggest initial screening for following group of postmenopausal women:

1. All women over 65 years of age.
2. Women more than 50 years with one or more clinical risk factors
3. Any fracture after age 40
4. Osteoporotic fracture in a first- degree relative
5. Current smoking
6. Weight less than 127 lbs
7. Postmenopausal women presenting with fractures.

Prevention

1. *Primary Prevention*: Should start at menarche and adolescent years with a combination of adequate nutrition, physical exercise and healthy life-style to achieve optimal peak bone mass.
2. *Secondary Prevention*: To diagnose osteoporosis and to prevent osteoporotic fractures and further deterioration.
3. *Tertiary Prevention*: To prevent further osteoporotic fractures where they have already occurred in the past.

Treatment

1. HRT is recommended after menopause but not generally so. But HRT is necessary when osteoporosis or its risk factors already exist.
2. Weight-bearing exercises
3. Smoking and alcohol discouraged.
4. Nonhormonal treatment includes:
   - Biphosphonates
   - Calcitonin
   - Fluoride
   - Calcium (1500 mg elemental Ca/ day) and Vitamin D (20 mg/day) supplementation.

28. What is conventional HRT?

Conventional Hormone Replacement Therapy is replacement of the predominant circulating hormones in the premenopausal woman namely, estrogens, progestogens and to some extent androgens after the decline of ovarian function.

Conventional hormone therapy includes:

Estrogen replacement therapy (ERT)
Estrogen Progestogen therapy (EPRT)
Sential EPRT (SEPRT)
Continuous Combined HRT (CCHRT)
Continuous Combined EPRT (CCEPRT)
Continuous Sential HRT (CSHRT)
If Androgens are given, it is known as AT.

29. What are Tibolone and SERM?

Tibolone is a widely acting gonadomimetic hormone with a combination of all estrogenic, progestogenic and androgenic actions. It is a uni compound with tissue-specific mode of action and is described as STEAR, i.e. Selective

Tissue Estrogen Activity Regulator. It has estrogenic effects on the bone and vaginal tissue, functions as progesterone in endometrial tissue, and has androgenic effects on the brain and liver. It is administered orally as once daily dose of 2.5 mg. It may not be effective against severe vasomotor symptoms in this dose. Its long-term effects are still under research. It is not a conventional HRT as it does not contain estrogen of progesterone.

Side effects include breast tenderness, acne, hirsutism, visual disturbances, headache and weight gain.

SERM's: Selective Estrogen Receptor Modulator is a compound that produces estrogen agonism in desired target tissues such as bone, liver, vascular system, etc. together with estrogen antagonism and/or minimal agonism in reproductive tissues such as breast and uterus. They are a group of anti-estrogens, eR ligands which activate transcriptional activational functions in estrogen receptors TAF 1 (agonistic) and TAF 2 (antagonistic).

There are 3 generations of SERM's:

1st: Tamoxifene

2nd: Raloxifene

3rd: Iodoxifene.

30. What is the MORE study?

The Multiple Outcomes of Raloxifene Evaluation study, which involved 7705 postmenopausal women over 4 years. It showed it had beneficial effects on osteoporosis after 36 months, it reduced the incidence of breast cancer but had no effect on endometrial cancer, 10% alteration in lipid profile.

31. What are biphosphonates?

Biphosphonates are enzyme resistant analogues of pyrophosphates which inhibit mineralization in bone. They reduce the resorption of bone in a dose dependent manner mainly by inhibiting recruitment and promoting apoptosis of osteoclasts. They also indirectly stimulate osteoblast activity. There are 3 generations of biphosphonates:

1st: Etidronate

2nd: Tilidronate

3rd: Risedronate.

Absorption is impaired by food and therefore have to be taken on empty stomach.

Dose: 5 mg/day for prevention and 10 mg/day for treatment.

48% reduction in new vertebral fractures was observed over a period of three years of Alendronate therapy.

32. What are phytoestrogens?

Phytoestrogens are plant compounds that are structurally or functionally similar to steroidal estrogens produced by the body. These compounds are weaker than natural estrogens and are found in herbs, seasoning, vegetables, fruits and drinks. Oats, fennel, liquorice, barley, yeast, etc. contain phyto-estrogens.

Flavonoids are the dominant phyto-estrogen in the human diet with 4000 different flavonoids having been identified.

They are divided into 7 subfamilies:

- Flavons
- Isoflavons
- Flavanones
- Flavonols
- Coumestans
- Lignans
- Chalcones.

Phytoestrogens are selective estrogen enzyme modulators functioning as pro- estrogens when estrogen deficiency is present and as anti-estrogens when excess estrogen is present. Thus they selectively balance the estrogen metabolism in the body. They are antioxidants, bone protective, cancer preventive, anti-atherogenic, and delay aging. It is a good alternative to women in whom conventional HRT is contraindicated.

39 Hormones and Allied Medications in Gynecology

Shirish Daftary

INTRODUCTION

- Hormones play an important role in gynecological practice.
- Availability of natural and synthetic derivatives have widened the cope for clinical application.
- Bioassays have enabled the possibility of assessing deficiency states and hormonal abnormalities to provide rational therapy in appropriate doses.
- Several common hormones in clinical use have been reviewed.

INDICATIONS

The broad indications of use of hormones in gynecological practice include:

- Management of Infertility
- Menstrual disorders
- Contraception
- Menopause and
- Substitution therapy in deficiency states
- Cancer.

COMMON HORMONES USED IN CLINICAL PRACTICE

- Estrogens
- Progestogens
- Androgens
- Antiestrogens
- Antiprogesterone
- Antiandrogens
- Pituitary gonadotropins
- Gonadotropin releasing hormones (GnRH) and analogues.

DETAILS OF HORMONES AND THEIR CLINICAL APPLICATIONS

Estrogens

Structure: Naturally occurring C-18 steroid hormones.

Source: Ovaries, adrenal glands and placenta during pregnancy.

Natural estrogen types: Estriol, estradiol and estrone.

Actions: These are as follows:
- Growth and maturation of female genital tract
- Initiation and maintenance of secondary sexual characteristics
- Provide negative feedback to the pituitary and hypothalamus
- Proliferative phase of the endometrium
- Cervical mucus favorably to sperm ascent
- Preparing the endometrium for progesterone action prior to nidation.

Therapeutic indications:
- Ethynyl Estradiol (EE) as a common component of oral contraceptive pills (OCP)
- In the treatment of dysfunctional uterine bleeding
- Conjugated estrogens in the management of menopause
- Local Application as Dienesterol cream for senile vaginitis
- HRT in perimenopausal and menopausal women—to control symptoms, cardioprotective effect and prevent osteoporosis
- Estrogen patches—HRT for controlling menopausal symptoms
- Intersex cases to promote female sexual development
- Prostate cancer.

Contraindications:
- Suspected genital tract malignancy
- Breast cancer
- H/o—Thromboembolism
- Liver and gallbladder disease
- Cardiac, hypertensive or diabetic women
- Suspected pregnancy and during lactation
- Sickle cell anemia
- Drugs that interact with it include rifampicin, barbiturates, phenytoin, and anticoagulants.

Side-effects and complications:
- Nausea and vomiting
- Water retention and weight gain
- Mastalgia
- Thromboembolism
- Cerebrovascular accidents
- Endometrial cancer/breast cancer
- Gallbladder disease
- Hepatoma.

Progestogens

Structure: Natural steroid hormone with C-21 ring. It is not orally absorbed

Source: Theca cells of the corpus luteum of the ovary.

Types: Pure progesterone and progestogens (synthetic compounds with progestational effects in common clinical use have been listed below:

- Pure progesterone
- Micronized progesterone
- Esters of progesterone—17 α OH progesterone caproate (proluton) and Medroxy progesterone acetate (MPA)
- Retroprogesterone (dydrogestrone, duphaston)
- Alkyl derivatives of testosterone—Ethisterone.
- Alkyl derivatives of nortestosterone—Norethisterone, lynestrenol, norgestral, desogestrel, norgestimate, gestodene.

Routes of administration:

- Oral—singly or along with progestogen
- Intramuscular—Proluton Depot, Injectable DMPA—as long acting contraceptive.
- Implants—Norplant (contraceptive).
- Hormone IUD—Mirena coil.
- Micronized progesterone—oral use, intravaginal use.
- Vaginal ring
- Skin patches.

Therapeutic Uses:

- Luteal phase defect
- Threatened abortion
- Recurrent abortions
- High doses are used to control advanced endometrial cancer.
- Contraception
- Postcoital pill
- Endometriosis
- Prior to TCRE to thin down the endometrium prior to ablation.
- Amenorrhoea—progesterone challenge test
- Hormone replacement therapy
- Patches—in HRT
- Postponement of menses—social/cultural reasons.

Contraindications:

- Undiagnosed vaginal bleeding
- Breast cancer/tumor
- Thromboembolism

Side-Effects:

- Nausea and vomiting
- Headache, mastalgia, water retention, bloating, weight gain, cramps, hirsutism, depression
- Increase in low density lipoprotein and cardiovascular accidents.

Androgens

(a) Testosterone:

Structure: Naturally occurring steroid hormone.

Source: Ovarian stroma and adrenal glands.

Usual level: 1 ng/ml .

Therapeutic uses: Limited because of fear of hirsutism. It is used in the treatment of Infertility for male oligospermia and decreased libido.

Side effects: Hirsutism, virilization.

(b) Danazol: Isoxazole derivative of 17-α ethinyl testosterone.

Actions: Acts directly on the endometrium causing atrophy.

Uses: Treat the following conditions:
- Endometriosis
- Cyclic mastalgia
- To treat diminished libido
- To shrink fibroids
- To thin down endometrium prior to ablation (TCRE).
- Fibrocystic disease of the breast
- Gynecomastia.

Side effects:
- Weight gain
- Hirsutism, acne, muscle cramps
- Breast atrophy
- Voice change
- Increased low density lipoproteins can cause cardiovascular complications.
- Liver damage
- Teratogenic to the female fetus if administered during pregnancy.

(c) Gestrinone

Structure: Trienic 19-norsteroid derivative.

Action: Androgenic, antiestrogenic, antiprogesterone and antipituitary actions similar to danazol. It is used for similar indications. It is milder and better tolerated.

Antiestrogens

Drugs in this category in clinical use are clomiphene citrate and tamoxiphen.

(a) Clomiphene Citrate (CC)

History: Introduced by Greenblatt in 1956.

Structure: CC is a nonsteroidal compound related to diethylstilbestrol (DES). It is a mixture of 2 isomers (cis and trans). The cis fraction is responsible for ovulation induction.

Action: Blocks the negative feedback of estrogen to the hypothalamus and thus stimulates gonadotropin secretions FSH and LH thereby inducing ovulation. This action is optimal in presence of some estrogen in the body.

Indications:
- Anovulatory infertility
- PCOD
- Assisted reproduction
- Male infertility.

Contraindications:
- Ovarian cyst
- Liver disease
- Scotoma.

Side Effects:
- Ovarian enlargement
- Hot flushes
- Nausea and vomiting
- Visual disturbances
- Headache and dizziness
- Hair loss
- Antiestrogen effects on cervical mucus
- Ovarian hyperstimulation
- Multiple pregnancy
- Higher abortion rate
- Neural tube defects in offspring
- Premature ovarian failure.

(b) Tamoxifen

Structure: Nonsteroidal antiestrogenic medication.

Action: Binding and reducing available estrogen receptors.

Uses: Treatment of advanced breast cancer.

Dosage: 10-20 mg twice daily for not more than 5 years in the treatment of breast cancer.

Side effects: Gastrointestinal upsets, dizziness, pruritus, thrombocytopenia, leucopenia, thromboembolism, hot flashes, and vaginal bleeding.

Risks: Endometrial hyperplasia and endometrial cancer. Hence monitoring endometrial thickness on (USG) and endometrial sampling periodically is an important safeguard.

(c) Letrazole

Structure: Nonsteroidal aromatase inhibitor which suppresses estrogen effects.

Dose: 2.5 mg daily for 5 days in every cycle (just like clomiphene citrate).

Indication: Ovulation induction in cases of anovulatory infertility.

Advantage: It scores over clomiphene because it does not have adverse effects on cervical mucus or endometrium.

Antiprogesterones

(a) Mifepristone (RU 486)

Structure: 19-norsteroid derivative of synthetic progestogen norethindrone.

Action: It binds to the receptors in the cell nucleus and blocks progesterone action on the target organ.

Absorption: About 85% of the drug is absorbed after oral intake. Peak levels are reached in 1-2 hours, the half life of the drug is 24 hours. It is excreted in bile and feces.

Indications: (a) Therapeutic abortion in conjunction with misoprostol for medical termination of pregnancy within 45 days of amenorrhoea. (b) Postcoital pill for prevention of pregnancy. (c) Cervical ripening prior to induction of midtrimester MTP (d) Induction of labor in case of intrauterine fetal death. (e) Missed abortion.

Side effects: Headache, gastrointestinal upsets, rash, faintness, adrenal failure if given massive doses, teratogenic if MTP fails.

Antiandrogens

(a) Cyproterone Acetate (Diane 35, Krimson 35, Ginette 35)

Structure: Chemically related to progesterone.

Action: Potent anti-androgen, it competes with dihydrotestosterone for intracellular androgen receptor sites and inhibits its binding. It has a weak corticosteroid effect. In small doses it does not affect the pituitary gland, but large doses cause amenorrhoea, loss of libido. In males it causes loss of libido and suppression of spermatogenesis.

Indications: (a) Prescribed along with ethinyl estradiol (EE) cyclically for 3 weeks in every cycle for 6-12 months—in the treatment of PCOS—hirsutism. Beneficial effects are seen only after 3 months when hirsutism and acne show reasonable clinical improvement. (b) Diane 35 regularizes menstruation (c) Contraceptive effect. (d) Treat acne. (e) Prior to induction of ovulation in PCOS to improve treatment results.

(b) Spironolactone

Structure: Aldosterone antagonist having antiandrogenic and diuretic effects.

Actions: Blocks androgen action are receptor level in hair follicles. It reduces 17α-hydroxylase activity, lowers plasma levels of testosterone and androstenedione.

Uses: Treatment of hirsutism.

Doses: 150 mg daily along with cyclic EE for 6-12 provides adequate relief in most cases. This is followed with maintenance dose of 50 mg/day.

(c) Other antiandrogens: Flutamide and finasteride are other drugs used in the treatment of hirsutism.

Pituitary gonadotropins

The anterior pituitary secretes hormones—FSH, LH and prolactin (PRL).

Source: FSH from menopausal urine
HCG resembles LH extracted from urine of pregnant women.

Uses:
- Induction of ovulation in anovulatory infertility
- Induction of multiple ovulation prior to ART.
- Hypogonadotropic hypogonadism
- Cryptorchism
- Amenorrhoea secondary to pituitary failure
- hCG is used in women with luteal phase defect.

Side effects: Hyperstimulation syndrome and multiple pregnancy.

GnRH and analogues

History: First isolated by Schally, et al and Matsuo, et al 1971.

Structure: Decapeptide.

Biological action: Released by hypothalamus in a pulsatile manner. The pulse rate and intensity determines pituitary release of glycoprotein hormone FSH or LH. Continuous administration however suppresses the pituitary gonadotropins.

Clinical uses:
- Pulsatile GnRH analogues ≅ 5-10 μg IV every 90-120 minutes or 15-20 μg subcutaneous or intranasal 200 μg every 2 hourly is useful in the treatment of hypothalamic amenorrhoea. It can induce cyclic menstruation
- Pulsatile GnRH is used in treatment of hypothalamic infertility
- Down regulation protocol before commencing FSH/LH regime for ovulation induction in infertile women on ART treatment
- PCOS
- Endometriosis
- To shrink the size of fibroids prior to surgery.
- To thin down the endometrium prior to ablation (TCRE).
- Cryptorchism in males.
- Continuous administration (zoladex, leupride) is useful in the treatment of precocious puberty to buy time until normal age of puberty is reached.
- Contraception—expensive and therefore not practical
- Dysfunctional bleeding when other measures have failed.

Side effects:
- Hyperstimulation syndrome
- Multiple pregnancy incidence same as in general population.
- Higher abortion rate
- Prolonged use causes menopausal symptoms and favors osteoporosis.
- Decrease in breast size
- Decreased libido
- Cholesterol and low density lipoproteins.
- Nausea, insomnia, dizziness and myalgia.

Bromocryptine

Structure: Synthetic ergot alkaloid—derivative of lysergic acid and ergoline.

Action: Dopamine agonist, it suppresses prolactin while promoting the secretion of gonadotropins. It induces menstruation, ovulation and pregnancy.

Available formulations: Oral: Parlodel, serocrip, proctinal and cabergolin.

Vaginal tablet: Pergolide.

Parenteral: Parlodel-LAR (glycolipid microspheres).

Therapeutic applications
- Hyperprolactinemia causing galactorrhoea/oligomenorrhea/anovulation and infertility
- Suppression of lactation
- Treatment of prolactinomas.

Special Instructions:
- Bromocryptine may be continued during pregnancy—it does not cause teratogenic effects
- Watch out for growth of prolactinomas (MRI scan and Assessment of visual fields).

Dose:
- Initiate treatment with a low dose of 1.25 mg at bedtime, gradually build up the dose to 2.5 mg twice daily or more as required to normalize prolactin levels
- For those who cannot tolerate oral medication—advise vaginal medication instead
- Parenteral Parlodel—LAR 50 mg every month is prescribed for prolactinomas.

Side effects:
- Nausea and vomiting
- Dizziness and hypotension
- Postural hypotension.

Thyroid deficiency

After diabetes—thyroid deficiency is the second common endocrinopathy affecting women. It affects 0.2% of pregnant women and about 0.5% of women in the general population.

Clinical manifestations: Hypothyroidism is more common.

- Nonspecific symptoms as weakness, lethargy, constipation, feeling excessively cold, infertility, menstrual disturbances, pregnancy loss, mental retardation in offspring. Oral medication with thyroid extract corrects this disorder satisfactorily.
- Hyperthyroidism manifests as increased appetite, skin warmth, palpitation, heat intolerance, failure to gain weight, increased sweating, exopthalmos, enlarged thyroid gland, menstrual disturbances, infertility.

Diagnosis: This is based on evaluating thyroid profile—T_3, T_4 and TSH assays. Thyroid antibodies.

Treatment: As advised by endocrinologist.

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Genital Prolapse

CN Purandare, Madhuri A Patel
Manjusha Yetalkar, Nikhil Purandare

Mrs. XYZ, 26-year-old, a housewife, residing at Parel G_1P_1 came with

- C/o something coming out per vaginum since delivery
- H/o prolonged labor
- H/o lifting heavy weight
- H/o dyspareunia
- No h/o chronic cough, constipation, straining for micturition
- No h/o dysuria, frency of micturition, urgency, incomplete evacuation
- No h/o stress urinary incontinence
- No h/o leucorrhoea, metrorrhagia, postcoital spotting
- No h/o distension of abdomen or mass in abdomen
- No h/o low backache.

M.H.: 3-4 days ——————— Regular, moderate, painless
30 days cycle
LMP: 7 days back

O.H.: Married since 4 year
1 FTND—male child 2-year-old A and H, home delivery
H/o prolonged lab
No H/o AB/SB

Past history: No H/o TB/DM/BA/Surgical illness

Personal history: Sleep (N) Appetite (N)
No bowel/bladder complaints
No addiction

Family history: Not contributory

On Examination:
Weight—52 kg
GC—Fair
Average built and average nourished.

Afebrile
Pulse—84/minute, regularly
BP—110/80 mmHg
CVS—S_1S_2 (N)
No murmur
RS— AEBE
Clear
PA— Soft
No T/G/R
PS— Cervix
Vagina

II° cervical descent
Min cystocele
Min rectocele
No enterocele
SUI not demonstrable (with full bladder).
UCL—4″

Bimanual vaginal examination: Uterus retroverted/NS
Smooth, firm, mobile
Fornices free
Supra vaginal elongation of cervix +.

Diagnosis: $G1_1\ P1_1$ with II° cervical descent with minimal cystocele and rectocele.

QUESTIONS

1. What is prolapse?
A prolapse is the protrusion of an organ or structure beyond its normal anatomical position (Procidentia Latin Procidese = to fall).

2. Why genital prolapse occurs?
Genital prolapse occurs due to relaxation and weakening of pelvic supports which results in displacement of either uterus or vagina or both from its anatomical position.

3. What is II degree genital prolapse?
According to Shaw's classification II degree genital prolapse means cervix descends up to the introitus.

According to Jeffcoate's classification II degree genital prolapse means cervix descends outside the introitus when the women is straining or standing.

4. What is II degree cystocele?
It is prolapse of the upper 2/3 of anterior vaginal wall and underlying bladder up to the introitus.

5. What is II degree rectocele?

It is prolapse of lower 2/3rd of the posterior vaginal wall underlying rectum up to the introituss.

6. What is enterocele?

Enterocoele is herniation of pouch of Douglas which contains loops of small bowel or omentum.

Types

a. Pulsion: in this type the vaginal vault is pushed outwards by conditions which increases the intra-abdominal pressure, e.g. chronic respiratory problems, lifting heavy weights, etc.
b. Traction: There is preexisting weakness in the anterior compartment, uterine does not pulls down the vault.
c. Congenital: Results from the failure of fusion or reopening of the previous fusion of the peritoneum so that the bowel filled cul-de-sac can extend by dissection all the way to the perineal body.
d. Iatrogenic: This follows a vault suspension procedure like Burch/Marshall Marchetti-Krantz which produce a change in the vaginal axis and subject the vault to abnormal procedures.

Type according to site

a. Anterior—anterior to vagina
b. Posterior—located between vagina and rectum
c. Lateral (pudendal enterocele) located lateral to vagina.

7. Why there is chronic backache in a case of genital prolapse?

Chronic backache in a case of genital prolapse occurs due to stretching of pelvic ligaments, i.e. uterosacral and Meckenrodts ligament.

8. Why there is leucorrhoea in a case of genital prolapse?

Leucorrhoea in a case of genital prolapse occurs due to vaginitis or due to decubitus ulcer or due to increased cervical glandular activity associated with congestion.

9. Why there is dyspareunia in a case of genital prolapse?

Dyspareunia in a case of genital prolapse occurs due to:

- Prolapse of ovaries in POD
- Associated with PID, Endometriosis, etc.

10. Why there is bearing down sensation in a case of genital prolapse?

Bearing down sensation in a case of genital prolapse occurs due to stretching of mesentery of the enterocele.

11. Why in severe degree of cystocoele incomplete evacuation of urine occurs and more the patient strains she has more difficulty to pass urine?

When the intra-abdominal pressure is raised during straining, the urine is pushed down into the cystocoele below the level of the external meatus. She can only empty the bladder by reducing the cystocoele into the vagina with her fingers.

12. What are the supports of the uterus?

Supports of the uterus

a. Muscular levator ani
   Coccygeus
b. Urogenital diaphragm—Sphincter urethrae
c. Ligamentous - Transverse cervical
   Pubocervical
   Utero sacral (uterocervical)
d. Round ligament of uterus (almost atavistic)
   Except the round ligament, the other are robust and contain smooth muscles along with fibrous tissue to offer good support
e. Perineal body
f. Anteverted position of uterus.

13. What are etiological factors in this case?

In this case childbirth has resulted into genital prolapse.

During child birth there may be:

- Excessive stretching of pelvic floor muscle and ligaments
- Bearing down before full dilatation
- Prolonged 1st and 2nd stages of labor
- Application of ventouse before full dilatation
- Pudendal nerve injury
- Delivery of big baby
- Resuming heavy work soon after delivery and without pelvic floor exercises.

14. What are the aggravating factors for genital prolapse?

a. Increased intra abdominal pressure
   - Chronic cough
   - Chronic constipation
   - Ascites
   - Tumor
   - Lifting heavy weights
   - Straining at stools.
b. Increased weight of the uterus
   - Subinvolution
   - Myohyperplasias
   - Small tumor.
c. Traction on uterus
   - Vaginal traction
   - Large cervical polyp
   - Pulling strongly on the cervix during operation.

15. What are the D/D of genital prolapse?

a. Congenital elongation of the cervix
b. Cervical polyp
c. Vulvar tumor.

d. Urethral caruncle
e. Chronic inversion of uterus
f. Vaginal/periurethral cysts
g. Diverticulae/prolapse of the urethra.

16. What is the mode of treatment in this patient?

This patient is young and interested in further childbearing function, hence conservative surgical management in form of sling operation is advised.

17. How will you investigate this patient?

- CBC
- RFTs
- LFTs
- Blood group
- Clotting time
- Bleeding time
- Urine analysis.
- X-ray
- ECG
- USG
- Vaginal cytology

18. What are the aims of surgery?

- To relieve symptoms
- To restore anatomy
- To restore sexual function
- To restore reproductive function.

19. What are the different types of sling operation?

a. Shirodkar's abdominal sling
   It is closed loop, posterior sling and static type of sling.
b. Purandare's cervicopexy
   It is closed loop but anterior and dynamic type of sling
c. Khanna's sling: it is open and neutral type of sling
d. Virkuds composite sling: It is combination of static and dynamic support open type and anterior plus posterior type of sling.

20. What is an ideal prosthetic maternal?

a. Biologically nonirritating to tissues
b. Inorganic in nature
c. Easily sterilized
d. Chemically inert in tissue fluids
e. Unaltered by sterilization
f. Non electrolytic, nonhydroscopic
g. Inexpensive and readily available
h. Pliable, nonrigid so as to adjust to tissue movement
i. Strong enough to withstand local stress and intra abdominal pressures.
j. Not subjected to fragmentation
k. Nonopa to X-ray
l. Should be usable in potentially infected area.

21. What are the selection criteria for sling operation?

a. Young women with II/III degree cervical descent wants to preserve the reproductive function

b. Absent or minimal cysto/rectocele
c. Uterocervical length of less than five inches
d. If moderate to large cysto/rectocoele is present it should be repaired from below at the same sitting before performing the sling
e. Not suitable for hypertrophic, lacerated and infected cervix.

22. Describe Shirodkar's sling operation for genital prolapse and its advantages and disadvantages?

- This is conservative type of surgery for genital prolapse where the mersilene tape is fixed to the posterior aspect of isthmus and sacral promontory
- It is an alternative to Purandare's cervicopexy, specially if cervicopexy in past has failed.

Advantages

- It maintains the uterus in its correct anatomical position
- It provides a strong static bony support
- No tendency to enterocele formation
- No difficulty in subsent LSCS.

Disadvantages

- Technically difficult to perform specially on left side where the sling has to pass blindly under the sigmoid mesentery
- Risk of injuring the colon, ureter, iliac vessels and gernitafemoral nerve on left side
- Injury to presacral veins and hematoma formation
- Being a closed loop sling, if it becomes tight, there is a risk of bowel obstruction.

23. What is Purandare's cervicopexy?

In this type of sling anterior rectus sheath or mersilene tape when anterior rectus sheath is defective is anchored to the anterior aspect of isthmus and anterior abdominal wall. It is indicated in II/III degree prolapse with minimal or no cystocele and/or rectocele and no supravaginal elongation of cervix.

Advantages

Technically easy to perform every time the patient strains the recti contract and uterus is pulled up instead of being pushed down as in the natural effect of straining.

Disadvantages

- Injury to the inferior epigastric vessels
- Not correct anatomically, being anterior sling uterus becomes retroverted. To prevent this on right side round ligament plication is done
- Deepens POD, so tendency to develop enterocele.
- As the tape is anchored anteriorly, it may get damaged at subsent LSCS surgery.

24. What is Khanna's sling operation?

In this, mersilene tape is anchored to posterior aspect of isthmus and anterior superior iliac spine. It is fairly easy to perform. There is no risk of bowel obstruction. Being static it doesn't cause anterversion or retroversion of uterus.

Disadvantages

- Tape is easily felt by the patient as it is being superficial
- If skin wound gets infected, periostitis will occur which is a very painful condition and the tape may get detached
- The tape will be uncomfortable for Indian traditional dress wearers.

25. What is Virkud's composite sling operation?

In this surgery, the mersilene tape is anchored from the posterior aspect of isthmus to sacral promontory on right side and anterior abdominal walls on left side and left uterosacral ligament is placated.

Advantages

- Easy to perform
- Provides double support
- Uterus remains anteverted
- No tendency to enterocele formation
- No risk of injury to sigmoid colon, iliac vessels, ureter or genitofemoral nerve
- Being open type of sling no risk of bowel obstruction.

Disadvantages:

- Tendency towards dextrorotation which can be compensated by left uterosacral ligament plication.

26. What are advantages and disadvantages of endoscopic approach to sling operation?

Advantages:

- All the benefits of minimally invasive surgery
- Magnification and better exposure of tissue.

Disadvantages:

- Expert and trained hands are required.

27. What are the other conservative surgical procedures can be done in this age group?

Other conservative surgical procedures can be done in this age group are:

a. Fothergill's operation
b. Shirodkar's modification of Fothergill's operation
c. Nadkarni's operation.

28. What are the principles of Fothergill's operation and disadvantages of it?

Principles of Fothergill's operation are:

- Dilatation and curettage

- Shortening of cardinal ligament
- Partial amputation of cervix
- Sturmdorf suture
- Anterior colporrhaphy
- Posterior colpoperineorrhaphy.

Disadvantages
- Cervical stenosis
- Infertility
- Incompetent os
- Cervical dystocia
- Recurrence after vaginal delivery.

29. What is Shirodkar's modification of Fothergill's operation

Shirodkar's modification of Fothergill's operation is same operation as Fothergill's operation except partial amputation of cervix and sturmdorf suture are not taken as there is no supravaginal elongation of cervix.

30. It patient of genital prolapse of advanced age who is not interested in childbearing and menstrual functions what type of surgery will be advised?

Patient of genital prolapse with advanced age who is not interested in childbearing and menstrual functions vaginal hysterectomy with anterior colporrhaphy and posterior colpoperineorrhaphy is advised.

31. What are the different operations done for Enterocoele repair?

The different operations done for Enterocoele repair are:

Abdominal route

Moschowitz operation is done in a patient of enterocoele associated with condition that requires exploratory laparotomy or along with Burch procedure, Purandare's cervicopexy, ventral suspension to prevent development of enterocoele in future.

Vaginal route

During vaginal hysterectomy high vaginal peritonization is done or during vaginal hysterectomy before peritonization, the peritoneum of the posterior cul-de-sac is sharply mobilized of the anterior surface of the rectum and lower sigmoid and excised. One to three internal McCall sutures are taken depending upon the size of the enterocoele. In internal McCall suture the monofilament zero suture is placed deeply into left pararectal fascia, after which the suture is continued across the front of the sigmoid colon and placed deeply into right pararectal fascia. Similarly one or two external McCall sutures are taken, where suture is taken from posterior vaginal wall to the peritoneum to the same left pararectal fascia across the front of the sigmoid to the right pararectal fascia and brought through vagina. These sutures are tied after completion of anterior colporrhaphy.

32. What are the different operations done to correct posthysterctomy vault prolapse?

Operation to correct posthysterctomy vault prolapse can be done by:

A. Vaginal route.
B. Abdominal route if vagina is narrow or if laparotomy is required for other reasons. Different operations are:
   a. Complete colpocleisis
   b. Sacrospinal vaginal fixation
   c. Williams Richardson's operation
   d. Ventral suspension of vaginal wall
   e. Abdominal sacropexy: The posterior cul-de-sac is obliterated by Moschowitz Culdoplasty where Mersilene tape, Teflon tape or Marlex mesh is used to suspend vault.

33. What are the indication and methods of conservative non surgical management?

Indications

a. Unfit for surgery
b. Decline for surgery
c. Waiting for surgery
d. Prolapse during 1st trimester of pregnancy
e. Puerperal prolapse
f. Planning for pregnancy in near future.

Methods of conservative non-surgical management

Pelvic floor exercises

Pelvic floor exercise also known as Kegel's exercises.

Principles: A sustained program of voluntary isometric contractions of pelvic floor muscles.

Technic: Patient is taught these exercises by asking her to contract her pelvic floor muscles around the examining vaginal finger as she would do so in order to interrupt her flowing stream of urine. Patient is asked to do these exercises at home at least three times a day gradually increasing in duration. Patient should be counseled that is a very important nonsurgical modality of treatment and it is patient dependent. Patient is also advised to avoid strenuous work or factor to prevent recurrence. Patient has to be explained that it is a very time consuming therapy and result will be seen after at least 6 months of continuous and regular exercises.

Vaginal support devices

There are: 1) Pessaries, 2) Cube, and 3) Gehrung.

Principles: It stretches the levator muscle which cause reflex contraction of it over the pessary which prevents it from falling down.

It is a ring shaped device made of polyethylene which is used as pelvic support device. It is most commonly used and simplest to use. It causes less inflammation as it is made up of polyethylene.

It is available in different diameters, according to outer circumferences.

Indications:

a. Prolapse uterus during pregnancy up to first 4 months
b. Immediate postpartum lactation
c. Patient unfit for surgery or not willing for surgery
d. Patient waiting for surgery, e.g. healing decubitus ulcer.

Contraindications:

a. Complete procedentia
b. Very patulous vaginal orifice
c. Stress incontinence
d. Local infection/ulceration.

- Correct size of pessary is chosen by trial and error methods, so that the largest sized pessary is comfortably retained during walking, standing, exercising, etc. it should not be expelled even partially/completely by Valsalva maneuver.
- It should be sterilized before used
- Counseling—regular removal and cleaning should be done. Initially remove overnight, clean with soap and water, lubricate and reinsert in the morning. Later weekly overnight removal to be done.
- Regular follow up is asked
- Explain hazards of leaving the pessary inside for prolonged period like impaction, ulceration, infection, malignancy, SUI, vesicovaginal fistula.

34. What are the complications of genital prolapse?

A. Local complications:
   a. Keratinization
   b. Decubitus ulceration
   c. Hypertrophy of cervix because of congestion, oedema, hyperplasia of cervical glands and supravaginal elongation of cervix.
   d. Incarcerated prolapse
   e. Malignancy.

B. Remote complications:
   a. Infection: UTI, cystitis, pyelitis, pyelonephritis
   b. Calculus formation
   c. Obstructive complications: Because of hypertrophy of bladder wall and trabeculation, constriction of ureters, hydrocereter, hydronephrosis, renal failure.

35. What are the preventive measures for genital prolapse?

- Antenatal physiotherapy
- Prevention of anemia
- Avoid bearing down before full dilatation of cervix

- Avoid prolonged first and second stages of labor
- Generous episiotomy in primigravidae and when required
- Low forceps if delay in II stage of labor
- Perineal tears immediately and accurately sutured after delivery
- Postnatal exercises and physiotherapy
- Early postnatal ambulation
- Adequate rest for 6 month after delivery
- Adequate interval between pregnancies
- Family planning
- Other strategies:
  - — Cessation of smoking
  - — Maintaining ideal body weight
  - — Avoid constipation
  - — Prevent diabetic complications
  - — No role of elective LSCS for protection of pelvic floor.

36. Genital prolapse in pregnancy

Symptoms

- Severe backache
- Excessive vaginal discharge
- Spotting per vagina
- Constipation
- Urinary retention and increased SUI.

Complications

- Abortion—Retention of urine
- UTI—Decubitus ulcers
- PROM—Cervical dystocia
- Cervical tears—PPH
- Subinvolution of uterus
- Puerperal infection.

Management

- Bed rest
- Pessary
- Introital tightening.

37. What is the cause of decubitus ulcer in case of prolapse?

Ulcer is formed due to dependent part of the prolapsed mass outside the introitus. There is initial surface keratinization → cracks → Infection → sloughing → ulceration.

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Uterine Fibroids

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1. What is incidence of fibroid?

Incidence

It occurs in 20-40% of women in the reproductive age group. These are more common in nulliparous or those with only one child and desirous of more. The prevalence is highest between 35 to 45 years age group.

2. What is etiology of fibroid?

Etiology:

Exact etiology not known. It appears to be that the growth of fibroid depends on ovarian steroids, since fibroids are not seen prior to menarche and usually there is regression in size after menopause and no new growth.

3. What are risk factors for fibroid?

Risk factors

Increased risk

- Age
- Nulliparous
- Obesity
- Family history
- Early menarche
- Diabetes
- Hypertension.

4. How will you classify fibroid?

Classification

Fibroids can be classified on the basis of either location or the uterine layer affected.

Corporal fibroids (91.2%)

1. **Submucus fibroids** are—located beneath the endometrium. These are sessile or pedunculated. Submucus fibroids often associated with a disturbed bleeding pattern. The pedunculated fibroids can protrude into

or through cervical canal, and this makes them more susceptible for infection or intermenstrual bleeding.

2. **Intramural or interstitial—**these are the most common sites occurring within the uterine wall.
3. **Subserous—**located just beneath the serosal surface. They grow out towards the peritoneal cavity, and can be sessile or pedunculated. These fibroids are either partially or completely covered by peritoneum. When completely covered by peritoneum, it usually attains a pedicle –called **pedunculated subserous fibroid**. On rare occasions the pedicle may get torn and gets its nourishment from the omental or mesenteric attachment. It is called a **parasitic or wondering fibroid.**

Sometime intramural fibroid may be pushed out between the layers of broad ligament and is called as**—broad ligament fibroid.**

Cervical (2.6%)

These are rare and further sub classified as:

- *Interstitial*—These often displace the cervix or expand so much that the external os is difficult to identify.
- *Subperitoneal*
- *Polypoidal*—According to the position they are classified as:
  - — Anterior
  - — Posterior
  - — Lateral
  - — Central.

5. What are clinical features of fibroid?

CLINICAL FEATURES

Symptoms

Menorrhagia

- Increased total surface area of the endometrium
- Mechanical distortion of the endometrial cavity
- Interference with uterine contractility
- Dilatation and congestion of the endometrial venous plexuses
- Endometrial hyperplasia due to high estrogen
- Pelvic congestion
- Ulceration of endometrium over the submucus fibroid.

Dysmenorrhea or dysmenorrhea

- Congestive as well as spasmodic dysmenorrhea

Subfertility in these cases is due to:

- Distortion and elongation of uterine cavity-difficult sperm ascent
- Impaired uterine contractility—difficult sperm ascent
- Congestion of endometrium—defective implantation
- Corneal block due to fibroid
- Elongation and stretching of tube over large fibroid
- Anovulation associated with hyperestrogenism.

Pressure effects are:
- Pelvic pain or backache
- Bladder—frency or retention
- Ureter—hydroureter, hydronephrosis
- Bowel—constipation, tenesmus.

Effects of fibroids on pregnancy can lead to:
- Repeated abortions
- Antepartum hemorrhage
- Malpresentations
- Postpartum hemorrhage
- Inversion of uterus
- Puerperal sepsis
- Defective implantation of placenta.

Effects of pregnancy on fibroid
- Increased size in 20 to 30% cases
- Torsion
- Infection
- Red degeneration
- Expulsion
- Necrosis.

7. What are clinical signs of fibroid?

SIGNS

G/E—anemia due to heavy and prolonged bleeding

P/A—palpable lump if more than 12 weeks size. Firm, nodular, well differentiated mass arising from pelvis and mobile from side to side

P/S—cervix pulled up in large fibroids

P/V—transmitted movements present

-Uterus not felt separately from the mass

-No groove felt between mass and uterus

-Uterus is enlarged and nodular

8. What are different diagnostic modalities for fibroid?

Diagnosis:

Apart from clinical examination various radiological modalities can be used for diagnosis of fibroids.

a. *Ultrasonography (USG):* USG is the modality of choice for diagnosis and evaluation of uterine fibroids. On USG fibroid appear as hypoechoic lesions. Transvaginal sonography (TVS) is the most accurate modality for diagnosis of fibroid. Transabdominal USG more useful in case of large fibroids; entire abdominal cavity should be examined carefully to diagnose pressure effects.
b. *Sonohysterography.*
c. *Magnetic Resonance Imaging (MRI)*: MRI has an important role in defining the anatomy of the uterus and ovaries in patients in whom USG findings are confusing or in case of large fibroids. This is expensive though.

d. *CT scan:* It has limited role in diagnosis of fibroids. On CT scan fibroids are indistinguishable from healthy myometrium unless they are calcified or necrotic.
e. *X-ray:* It has a limited role in diagnosis of fibroids. Unless heavily calcified, fibroids are not detected on radiographs.
f. *Hysteroscopy:* Submucus fibroids or fibroid polyp can be diagnosed on hysteroscopy. It can help us to know the exact origin, size and vascularity of the fibroids. It is also an apt technic for myomectomy in case of cervical fibroid or submucus fibroid.

9. What are differential diagnosis of fibroid?

Differential Diagnosis:
a. Pregnancy
b. Full bladder
c. Adenomyosis
d. Ovarian tumor
e. Endometriosis
f. Tubo-ovarian mass.

10. What is cervical fibroid?

Cervical fibroids:
- Anterior cervical fibroids: These burrow beneath the bladder base, elevating the bladder above it and causing hesitancy and urinary retention. Occasionally while incising the uterovesical fold, an accidental cystotomy may occur unless surgeon is aware of the elevated upper most limits of the bladder.
- Posterior cervical fibroids: These fibroids may grow in pouch of Douglas and may compress the rectum and cause constipation and tenesmus.
- Central cervical fibroids: They protrude in to the cervical canal. This may result in ureteric obstruction, hydronephrosis and hydroureter.
- Lateral cervical fibroids: These fibroids buries between the leaves of the broad ligaments. Uterine artery always runs on its lateral aspect. Identification of this structure is essential step during hysterectomy.

11. What is broad ligament fibroid?

Broad Ligament Fibroids
- These fibroids grow into the layers of broad ligament. The important differentiating feature is that the uterine vessels are always medial to true broad ligament fibroids.

Each of above mentioned fibroids require modifications in the steps during surgical management.

12. Is there any role of expectant management in fibroid?

Yes

Expectant management is recommended in:
- Asymptomatic myoma
- Nearer to menopause
- Less than 12 weeks size
- Follow up and periodic examination should be possible.

13. What are principles of medical management in fibroid?

The objectives of medical management

- To improve menorrhagia
- To minimize size and vascularity
- To correct anemia before surgery
- As an alternative to surgery in perimenopausal women or women with high risk factors for surgery
- Where postponement of surgery is planned temporarily.

Drugs used

a. *Progesterones*—They are not very effective. They mainly correct menorrhagia and hormonal disturbances associated with fibroids. Norethisterone acetate or Medroxyprogesterone acetate 5-10 mg is administered cyclically from day 5th of the cycle for 20 days.
b. *Antiprogesterones*—Mifepristone (RU486) has been found to be more effective in treatment of menorrhagia. It reduces size of the fibroid. A daily dose of 25-30 mg is recommended for three months.
c. *Androgens*—Danazol 200-400 mg can be given daily for three months. This minimizes blood loss significantly or it can produce amenorrhoea. It also reduces size of the fibroid.
d. *GnRH agonists*—For example, goserelin, buserelin, leuporelin acetate are commonly used. These cause pituitary down regulation and suppression of the ovarian function.
e. *GnRH antagonists*—For example, cetrorelix or ganirelix causes immediate suppression of the pituitary gland and ovaries.
f. *Progestogens*—They are not very effective for reducing the size of the fibroids. They mainly correct menorrhagia and hormonal disturbances associated with fibroids. Norethisterones acetate or Medroxyprogesterone 5-10 mg is administered cyclically from day 5th of the cycle for 20 days.
g. *Antiprogesterones*—Mifepristone (RU486) has been found to be more effective in treatment of menorrhagia. It reduces size of fibroid. A daily dose of 25-30 mg is recommended for three months.
h. *Androgens*—Danazol 200-400 mg daily for three months minimizes blood loss significantly or it can produce amenorrhoea. It can reduce size of fibroid.

14. What are different surgical options available for the management of fibroid?

Surgical management: Different modalities of surgical management are as follows-

a. **Myomectomy**—It is a conservative surgical modality. It includes enucleation of myoma from the uterus leaving behind potentially functioning uterus, capable of further reproduction. It is a more risky operation when fibroid is too big or there is a chance of recurrence.
   Indications: The patient is in reproductive age group:
   - Desirous of having baby
   - In cases of recurrent pregnancy wastage.

Contraindications: Other factors for infertility should be ruled out. If the husband is proved infertile and does not want to make use of donor sperms
- Infected fibroid
- Perimenopausal age group with large fibroids.

b. **Hysterectomy**–It is the operation of choice in symptomatic fibroids in patients over the age of 40 years and in those not desirous of future fertility. If there is an adnexal pathology it is also dealt with. If the ovaries are healthy and there is no family history of ovarian cancer, they can be left behind in women over 40 years of age. Removal of healthy ovaries can lead to sudden menopausal syndrome and long-term problems of osteoporosis. Though theses women can be offered Hormone replacement therapy but compliance is poor.
Place of vaginal hysterectomy—Fibroids with size of 10-12 weeks of pregnancy associated with or without a uterine prolapse are better dealt by vaginal route. This requires an experienced surgeon.

15. What is role of laparoscopy in the management of fibroid?

Endoscopic surgery: This requires specialized expensive equipment and training in endoscopic surgery. Advantages are quick recovery and earlier resumption of normal activities.

a. *Hysteroscopic myomectomy*: Indications are submucus myoma causing infertility, pregnancy wastage, abnormal uterine bleeding with pain. Advantages are short duration, rapid recovery, and less postoperative morbidity.
b. *Laparoscopic myomectomy*: It suitable for subserous and intramural fibroids.

16. What are newer modalities of management of fibroid?

Newer therapies for fibroids

a. *Uterine artery embolization*: It is a minimally invasive procedure, which can be used as an alternative to major surgical procedures for fibroids and failure of medical treatment.
b. *Mirena (levonorgesterol IUD):* It is third generation IUD, containing 52 mg of progesterone and licensed for five years use. It releases 20 μg of levonorgestrel daily, which acts directly on the endometrium. Periods become much lighter. If side effects do occur they are transient and include
   - Spotting per vagina
   - Headache
   - Breast tenderness
   - Water retention.

   However this can be inserted only if the cavity is regular and size of uterus is less than 12 weeks.

17. What are complications of fibroid?

Complication:

a. Red degeneration
   — Most frently during pregnancy
   — Abdominal pain, fever

— Aseptic
— Develops purple red colour and fishy odor.

b. Sarcomatous change (0.5%)
c. Torsion
d. Inversion of uterus
e. Capsular hemorrhage
f. Infection
g. Atrophy
h. Necrosis
i. Vacuolar changes
j. Hemorrhage.

18. Why polycythemia is common with fibroid?

a. Altered erythropoietic functions of kidney through ureteric pressure
b. Erythropoietic functions by tumor

19. What are different degenerations occurs in fibroid

- Hyaline degeneration
- Cystic degeneration
- Fatty degeneration
- Calcareous degeneration
- Red degeneration.

20. What is intravenous leomyomatosis?

This is tumour with extension into vascular space outside leiomyoma it is benign in nature and can extended into IVC. Pelvic vein, etc.

21. Why fibroid is called estrogen dependent?

- Increase incidence in childbearing age
- Decrease after menopause
- Increase in size during pregnancy
- High estrogen receptors
- Menorrhagia, hyperplastic endometrium, cystic ovary are associated with metropathia, endometriosis and carcinoma endometrium.

FREQUENTLY ASKED QUESTIONS

- What are the risk factors for fibroids?
- Classify fibroids?
- How patients with fibroids present?
- What are the chief complaints?
- Causes of menorrhagia in fibroids?
- Which types of fibroids are more symptomatic?
- Causes of menorrhagia in fibroids?
- Why fibroids are more common during reproductive age group?
- Factors responsible for infertility in fibroids?
- What are effects of fibroids on pregnancy?
- What are effects of pregnancy on fibroids?
- How will you differentiate fibroadenoma from adenomyosis?

- How will you investigate?
- What are different modalities of management of fibroid?
- Which is the definitive management of fibroid?
- What is myomectomy?
- What are indications and contraindications for myomectomy?
- Advantages and disadvantages of conservative management of fibroids?
- Principals of medical management of fibroids?
- What are newer modalities of management of fibroids?
- What is Bonneys' hood operation or myomectomy?
- What is mirena?
- Non-contraceptive use of mirena.
- What are the principles of myomectomy?
- What are wondering fibroid?
- What is red degeneration of fibroid?

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The Infertile Couple

Mandakini Parihar

1. When will you label a couple infertile?

When the couple has been trying for conception and have been unable to conceive after 1 year of regular unprotected sexual intercourse, we will call these couples as infertile and investigate them for the same.

2. What are the basic investigations advised for the couple?

a. Clinical evaluation for both partners
b. Semen profile
c. Ovulatory status by history and natural cycle follicular monitoring
d. Tubal patency and tuboperitoneal evaluation.

3. What are the common causes of infertility?

The causes can be divided into:

a. Male factor
b. Female factor
   - Ovulatory dysfunction
   - Tubal factor infertility
   - Uterine factor
   - Cervical factor
   - Peritoneal factor
c. Unexplained infertility.

4. What is the percentage of male infertility?

Approximately 40% have causal or associated male factor as the cause of infertility.

5. What are the different investigations when a couple comes for evaluation of infertility?

Investigations done for a couple with infertility:

For males

a. Semen analysis
   Most of the time, this is the only investigation asked for. However, if the initial semen analysis is abnormal, then additional investigations are needed and these are as follows.

b. Endocrine investigation for FSH, LH, Prolactin and testosterone.
c. Evaluation of sperm function.
   For example: Sperm migration test.
   Sperm survival test.
   Hypo-osmotic swelling test.
d. Immunological investigation, e.g. antisperm antibody by ELISA..
e. Microbiological investigation, e.g. semen culture.
f. Testicular biopsy.
g. Varicocele assessment using Doppler blood flow.
h. Genetic investigation by Karyotyping to look for translocations and micro-deletions.
   Tests of ovulation.

For females

a. BBT
b. Hormonal assay. These are baseline hormone evaluations done on the second or third day of the cycle to check for normal HPO axis. These include, FSH, LH, estradiol, TSH and prolactin levels
c. Cervical mucus study
d. Baseline ultrasonography along with natural cycle follicular monitoring for evidence of ovulation
e. Hysterosalpingography (tubal patency)
   If these are abnormal then additional tests may be needed and these include
f. Laparoscopy with hysteroscopy
g. Immunological test (cervical factor), e.g. antisperm antibody, antiphospholipid antibody
h. Endometrial biopsy or curettage.

6. What are the different types of infertility?

These can be divided as:

- Primary and secondary infertility
  or
- Male factor, female factor and unexplained infertility.

7. What are the different ways for detecting ovulation?

a. Basal body temperature
b. Cervical mucus study
c. Endometrial biopsy
d. Urinary LH monitoring
e. Ultrasound follicular studies is the most accurate way and is today the gold standard for ovulation detection
f. Serum progesterone level day 8/9 after ovulation (approximately day 21/22 of a 30 day cycle) > 15 ng/ml.

8. What are the criteria taken into account for normal semen count?

There are wide variations in normal semen counts and hence WHO has defined common minimum criteria for normal semen.

WHO parameters as per 1997 for Normal Semen Count are:

- Volume > 2 ml

- Concentration > 20 million/ml
- Motility > 50% forward progressive motility (combination of rapid and slow forward progressive motility)
- Morphology > 20% Normal.
- Vitality > 75% live
- WBC < 1 million/ml
- Immunobead < 20% adherent
- MAR < 10% adherent.

9. What are the common causes of infertility in women?

- Ovulatory disorder—40%
- Tubal diseases—20-25%
- Endometriosis—10-15 %
- Uterine factor—5%
- Cervical factor—5%
- Unexplained—10-15%.

10. What is meant by follicular studies?

Follicular study is monitoring of growing follicles in the ovary by sonography. Earlier it was done abdominally, but with the advent of vaginal probes, follicular studies are mostly done by the transvaginal sonography as it gives better resolution and information about the pelvic organs than abdominal sonography.

Base line sonography is done to know the ovarian volume and antral follicle count on Day 2/ Day 3 of the menstrual period. There after serial sonography done to evaluate the follicular growth and to see endometrial growth (thickness and Pattern) from day 7/8 onwards. The growing follicle is identified and monitored till the day it ruptures.

11. What are the different types of anovulatory infertility?

Broadly anovulation can be divided into four categories based on the levels of pituitary gonadotrophin hormones:

I—Hypogonadotrophic anovulation

Here the levels of gonadotrophin hormones are very low and hence there is no ovarian response, e.g. Kallman's syndrome, Anorexia Nervosa, exercise induced amenorrhea. Isolated gonadotropin deficiency, pituitary deficiency, etc. Here the gonadotrophin levels are low and hence there is anovulation.

II—Normogonatrotrphic anovulation

Here the levels of gonadotrophin hormones are normal range but ratio is altered and hence there is no or inappropriate ovarian response, e.g. PCOS. Here the levels of gonadotrophin hormones are normal but there is chronic Anovulation due to metabolic disturbance.

III—Hypergonadotrophic anovulation

Here the gonadotrophin hormones are markedly elevated due to resistant ovaries and this signifies absent or nonfunctional ovaries, e.g. premature ovarian failure, absent ovaries, etc.

IV—Hyperprolactinemic anovulation

Raised prolactin levels cause anovulation by affecting the pituitary secretion of the gonadotrophins and this leads to anovulation, e.g. idiopathic hyerprolactinemia, micro and macroadenomas of the pituitary, lactational anovulation, etc.

12. What is meant by PCOS? Who discovered it and when?

Polycystic ovarian syndrome (PCOS) was first described by Irvin F. Stein and Michael L Leventhal in 1939 and hence the earlier name was Stein-Leventhal syndrome.

It is the most common endocrine abnormality affecting female reproductive performance. It leads to infertility, menstrual disturbances and is associated with hyperandrogenism, obesity, insulin resistance. It is a metabolic dysfunction and needs active management to avoid long term complications of hyperestrogenism.

13. What are the clinical feature of PCOS?

- Obesity
- Hirsutism
- Oligomenorrhea/amenorrhea
- Infertility.

14. What is the pathophysiology behind PCOS and in the recommendation for it?

Pathophysiology of PCOS
Intact HPO Axis (But dysfunctional)
↓
Altered LH: FSH Ratio (LH increase)
↓
High Estrogen levels: Low progesterone
↓
↓ SHBG
↓
↓ Free testosterone
(androgenic dominance in microenvironment of the follicle)
↓
Chronic Anovulation

It is also been established that PCOS is associated with profound insulin resistance as well as with defects in insulin secretions. Insulin affects ovarian function in multiple ways, causing anovulation and hyperandrogenism.

15. What are the base line hormonal evaluations done and what do they signify?

Basal level of FSH and LH is done between 2nd and 4th day of menstrual cycle. Along with FSH and LH it is customary to check the levels of Estradiol, TSH and prolactin.

Based on the levels of FSH and LH we can determine the type of anovulation that the patient suffers from and institute the appropriate treatment.

16. Why does hyperprolactinemia cause anovulation?

Hyperprolactinemia has a negative feedback effect on hypothalamic pulsate secretion of GnRH, resulting in gonadotrophin deficiency and secondary ovarian failure. Patient may have amenorrhea and galactorrhea sometimes.

17. What are the different effects of tuberculosis on the genital tract?

Fallopian tubes are the most common site of initial involvement.

Infection can lead to adhesion formation, tubal blockage or tubo-ovarian masses.

Most common presenting symptom is infertility (75%) other may present with abdominal or pelvic pain, abnormal vaginal bleeding or peritonitis.

The tubes may be beaded, rigid lead pipe appearance or develop a hydrosalpinx.

If the uterine endometrium is affected, it results in destruction of the basal layer and subsent intrauterine adhesions—Asherman's syndrome.

18. What are the clinical manifestations of Asherman's syndrome?

Most common clinical manifestation of Asherman's syndrome is secondary amenorrhoea. Patients may also present with infertility and hypo amenorrhea or amenorrhea. Hysteroscopy is the gold standard in diagnosis but it can also be aided USG and HSG.

19. What are the treatment options advised to manage Asherman's syndrome?

Hysteroscopic resection is the primary treatment for Asherman's syndrome. Efficacious treatment also requires prevention of repeat adhesions and IUCD or Foley's catheter have been used in the past to prevent re adhesions along with exogenous estrogen to promote endometrial growth.

20. What are the common infective organisms affecting the fallopian tubes?

Common infective organism affecting fallopian tubes are:

a. Chlamydia trachomatis
b. Neisseria gonorrhea
c. Mycobacterium tuberculosis
d. Mycoplasma hominis
e. Endogenous aerobic and anaerobic bacteria.

21. What is meant by Fitz-Hugh-Curtis syndrome?

Women with PID complaining of acute right sided upper abdominal pain. It is due to fibrosis band extending from under surface of diaphragm to the surface of the liver. The most common organisms causing these in PID are Gonococcal and Chlamydial infection.

22. What is the relevance of hydrosalpinx in infertility management?

Hydrosalpinx represents the end result of a previous acute salpingitis and is often bilateral. The fimbrial end is closed and tubal fluid collects and causes

dilatation of the tube. In patients who are undergoing IVF prior removal of Hydrosalpinx warranted, as fluid contained in it can be embryo toxic and significantly decreases the pregnancy rate.

23. What is endometriosis and what are the different ways in which it causes infertility?

Occurrence of ectopic endometrial tissue outside the cavity of uterus is called endometriosis. It has a uni pathology of a benign proliferative growth process having the propensity to invade the normal surrounding tissue. About 15 % of infertile women suffer from endometriosis.

It interferes with tubal motility and function due to biochemical as well as physical alterations in the tubo-ovarian anatomy. It may inhibit ovulation and cause Luteinized unruptured follicular syndrome, hyperprolactinemia and short luteal phase cycle. Due to the altered tubo-ovarian anatomy there is dyspareunia and hence the frency of sexual intercourse is reduced.

24. What is meant by planned relations?

Planned relation means sexual intercourse done at and around the time of ovulation, signs of ovulation can be monitored either by sonography, urinary LH kit or basal body temperature charting.

25. What is meant by intra uterine insemination? And how is it done?

Intrauterine insemination (IUI) is a process by which semen which is processed in culture media is introduced into the uterine cavity under aseptic conditions. Processing is done so that all the debris, dead sperms and seminal secretions are separated out from motile sperms and a good harvest of actively motile sperms can be introduced in the uterine cavity using a small disposable plastic cannula. The dead sperms and debris need to be removed so as to prevent painful uterine contractions. At the time of ovulation or in the peri-ovulatory period, about 0.5-0.7 ml of motile sperms suspended in culture medium is inserted in the uterus, so as to place it in close proximity with the egg in the fallopian tube.

26. What is IVF? Describe process in brief?

In vitro fertilization (IVF) means fertilization of oocyte by sperm gamete in the laboratory.

The main stay of the program consists of stimulation of the ovary using gonadotrophins and the giving the trigger for oocyte maturation. 34-35 hours later, aspiration of mature oocytes from both ovaries under ultrasound guidance is done. These oocytes are kept in specific culture for a few hours to complete oocyte maturation. Specially processed sperm are kept with these oocytes in culture media under specific temperature and CO_2 environment so that oocytes are fertilized by sperm.

Evidence of fertilization is seen 18 hours and formation of an embryo occurs in the next 24 hours. These embryos when formed are then transferred into uterine cavity after 2-3 days of oocyte retrieval (cell division).

With extended culture media available, we can now culture the embryos up to the blastocyst stage and transfer on day 5/6 after occyte retrieval.

27. Name the world's first IVF baby and the scientists who were responsible for her birth?

First child born, as a result of IVF was Louise Brown in 1978, in Bournehall clinic in London by the efforts of two great scientists, Robert Edwards and Patrick Steptoe.

28. What is general success late with IVF procedure?

As the process of implantation is still a mystery, the overall pregnancy rate with IVF is 35-38%. There will be a 20-25% pregnancy loss due to abortions and hence take home baby rate is usually 22-25%.

29. What is meant by ICSI? And how does it differ from IVF?

ICSI stands for Intra-Cytoplasmic Sperm Injection and is a micromanipulation technique in which a single spermatozoon is injected directly into cytoplasm of mature oocyte using a micropipette. This technic increases the likelihood of fertilization when there are abnormalities in the number, quality or function of sperm.

While in IVF motile sperm are put with mature oocyte in culture media in incubator for 16-18 hrs and fertilization takes place naturally.

30. What is Varicocele and how does it affect fertility?

Varicocele is elongated, dilated and tortuous spermatic vein within the scrotum. The increased vascularity causes the temperature in the scrotum to be raised and hence affects normal spermatogenesis and maturation. It is found in 20-25% of infertile men with oligo/aestheno/teratospermia and can be the cause of low sperm counts and or motility.

31. What are the different types of azoospermia? What are the differentiating features?

Azoospermia is the absence of sperm in the semen and this can be obstructive and Non-obstructive in nature. The differentiating feature between the two types is absence of fructose in obstructive azoospermia.

Though there is absence of sperm in semen in both cases but in patients with obstructive azoospermic sperms can be retrieved by various microsurgical technique from testes/epididymis for IVF and ICSI.

Obstructive azoospermia as the name suggests is absence of sperm due to a physical block and there is presence of mature spermatozoa in the testes and /or epididymis.

Non-obstructive azoospermia is total absence of sperm in the semen and in the testes too. This is usually due to absence of one of the germ cells or testicular destruction beyond repair, e.g. gibrosis and atrophy, post mumps orchitis, Cryptorchidism, etc. As there is no sperm the only option for pregnancy is using donor sperm or adoption.

32. What constitutes the semen?'

Normal semen contains seminal plasma and cellular elements: Spermatozoa, sperm precursors and other cells from gentile tract and few leukocytes. The Seminal plasma consists of seminal proteins, prostatic secretions, fructose and secretions from bulbourethral glands.

33. Write the full form of

PESA: Percutaneous Epididymal Sperm Aspiration.

MESA: Microsurgical Sperm Aspiration.

TESA: Percutaneous Testicular Sperm Aspiration.

TESE: Testicular Sperm Extraction.

And when are the procedures done?

When there is absence of sperm in semen but the cause is obstructive azoospermia, sperms can be retrieved by various microsurgical technique from testes/epididymis for IVF and ICSI and achieve pregnancy.

34. Define unexplained infertility and options for treatment?

Unexplained infertility means failure to establish a pregnancy despite an evaluation uncovering no obvious reason for infertility, or after correction of factor identified as probably responsible for infertility.

It is recommended to try using an ovulation induction agent like Clomiphene citrate (CC) for 3-4 cycles. If there is no pregnancy then, it is reasonable to CC + HMG or only HMG cycles with IUI for 3-4 cycles. If this too fails then IVF with ICSI is the recommended treatment.

35. What are the treatment options in a patient with endometriosis and infertility?

Endometriosis is divided into minimal mild, moderate and severe disease and the treatment will depend on the degree of the disease. However, the most important point to remember is that start treatment for infertility as a primary treatment and there is no place suppression of endometriosis to improve outcome.

For minimal and mild endometriosis, controlled ovarian stimulation with IUI is the recommended treatment. They should be treated as unexplained infertility.

Surgery is the mainstay in patients with moderate to severe endometriosis with infertility. Depending on the degree of spread GnRH agonists may be used for a short term. Goals of surgery are to remove all implants, resect adhesions, relieve pain, reduce the risk of recurrence and adhesion formation and restore the normal anatomy and physiology to whatever extent possible. If endometriosis is of severe type when surgery cannot restore the normal tubo-ovarian relationship or if tubes are blocked then Assisted Reproductive techniques are the only option.

36. What is meant by donor insemination?

When the sperm is obtained from the sperm bank and inseminated in the female partner for achieving pregnancy, the process is called as Donor insemination. The donor is always unknown donor and the process is carried out with consent form both the partners and is legally valid. The sperm used is cryopreserved semen for insemination for the patients with sterility due to diseases or vasectomy, orchidectomy, chemicals or radiation exposure of because of genetic consideration, e.g. hemophilia, Huntington's diseases.

37. Where does one get sperm for Donor insemination and what precautions should be followed prior to donor insemination?

Sperm for donor insemination should be used from cryopreserved semen from certified sperm banks. Each sample of semen is tested for common viral markers, viability of the sample after thawing the sample and is stored in liquid nitrogen for at least 3 months. The donor is tested for viral markers again and when proven negative the sample is released for clinical use. This ensures storage of properly checked sample for various infective organisms including HIV and HbsAg and HbC. Adequate counseling prior to starting the donor cycle treatment is vital.

Couple should be assured of complete confidentiality and the method by which the donors are selected, matched and the possibility of success should be informed. It is necessary to match the blood group of the donor with that of the husband and basic physical characteristics. Confidentiality is the key issue and must be strictly adhered to.

38. What is mean by ART? And what does it include?

ART stands for Assisted Reproductive Technologies. These mean any procedure involving manipulation of gametes *in vitro* and include *In Vitro* Fertilization (IVF), Intracytoplasmic Sperm Injection (ICSI), Gamete Intrafallopian Transfer (GIFT), Zygote Intrafallopian Transfer (ZIFT).

39. What is the most common drug used for ovulation induction and how it is used?

Clomiphene citrate is the most common drug used for anovulatory infertility. It is nonsteroidal compound with weak estrogenic effect.

When it binds to estrogen receptors, central perception of circulating estrogen level is prevented, dampening or abolishing the usual negative feedback response. So GnRH pulsatility and gonadotropin secretions are enhanced which further results in stimulation of follicular growth and development.

Treatment is usually begun following spontaneous menses or progestin withdraws bleed. Clomiphene is prescribed for 5 days beginning with cycle days 2-5. Starting dose is typically 50 mg while a dose of 100 mg is more appropriate for obese patient.

If no response is noted in that cycle, dose should be increased by 50 mg in next cycle until ovulation is noted till maximal dose of 150 mg is reached. It should be reasonable to limit patient to six consecutive cycle, with no more than a dozen lifetime cycle.

40. What is the role of gonadotropin therapy in infertility? Name the different types of gonadotropins available?

For the patients who do not responds to clomiphene therapy or is not a candidate for clomiphene will need administration of exogenous gonadotropins.

Various gonadotrophin preparations available are:
1. Urinary human menopausal Gonadotropins (hMG) (LH: FSH 1: 1).
2. Urinary FSH(u FSH)
3. Highly purified urinary FSH (Uhp-FSH)
4. Recombinant FSH (r FSH)
5. Recombinant LH.

41. What is bromocriptine and what are common side effects?

Bromocryptine is an ergot derivative that stimulates dopaminergic receptors in the brain and anterior pituitary. It is used for treatment of hyper-prolactinemia.

It is usually started at dose of 1.25 mg each evening for first 7-14 days to decrease possible side effects. Dose can be Increased by 1.25 mg every 2-3 weeks until normoprolactinemia is achieved.

Common side effects are: GI disturbances like nausea and vomiting, orthostatic hypotension, nasal congestion and headache.

42. What is Cabergolin and where is it used?

It is a dopamine agonist. It is used in cases of hyper-prolactinemia. It has advantages over bromocriptine as it has longer half-life and a higher binding capacity for D2 receptors. This allows a biweekly dose and hence a significant decrease in the side-effects seen with bromocriptine.

43. What is the role of GnRH analogues in assisted reproduction? Name the types of GnRH analogues?

GnRH analogues are used in assisted reproduction for down regulation of the pituitary and hence achieving an appropriate cycle control by preventing premature LH surge. This prevention of LH surge allows further maturation of follicles which has been shown to increase clinical success rate. The can be classified into agonists and antagonists.

The agonists are further divided into:

A. Nonapeptides.
   - Buserelin
   - Leuprolide
   - Histrelin
   - Goserelin.

B. Decapeptides.
   - Naferelin
   - Triptorelin.

44. What is the drug used to trigger ovulation? When? What dose?

Human chronic gonadotrophin is used as an LH surrogate to trigger ovulation. Injection HCG is usually administered, in doses of 5000- 10,000 IU, when there is at least one follicle measuring 18 mm in diameter. Ideally, the estradiol levels should be at least 200 to 300 pg/ml per mature follicle and endometrial thickness of at least 8 mm.

45. What is meaning of surrogacy?

Gestational surrogacy: Women who have an embryo transferred to their uterus. This embryo genetically belongs to the intended parents, but is implanted in the surrogates womb. At the end of the gestational period the surrogate will deliver and then the child is given to the genetic/biological parents. As per the Indian law, the biological parents must officially and legally adopt the child.

46. What means can we achieve pregnancy in postmenopausal women?

In post menopausal patient pregnancy can be achieved either by means of ovum donation or embryo donation. This is because her own ovaries cannot produce any eggs and hence ova for a younger woman are fertilized with the husband's sperm and then transferred in the menopausal woman's uterus. In embryo donation, the ready embryo from unknown donors is implanted in the uterus.

Three main principles for donor oocyte program are:

a. Preparation of recipient's endometrium
b. Window for embryo transfer
c. Early pregnancy management with exogenous hormones.

47. What are complications of use of gonadotrophins in ART?

By and large the gonadotrophins are quite safe but there can be some immediate side-effects with the use of gonadotrophins.

These are:

- Retention cyst
- Mild injection site reaction
- Headache
- Ovarian hyper stimulation syndrome (mild to moderate)
- Abdominal pain and gastrointestinal symptoms, e.g. nausea, vomiting, diarrhea, abdominal cramps and bloating
- Multiple pregnancy.

UNCOMMON

- Severe OHSS
- Ovarian torsion, a complication of OHSS.

48. What is postcoital test?

It is to evaluate the semen, cervical mucus and the immunological cause of infertility.

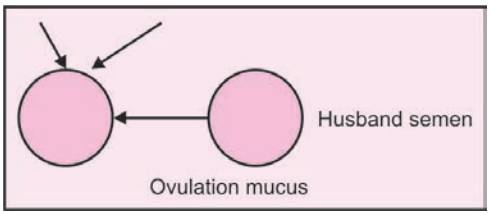
49. Postcoital test?

The couple is asked to have coitus around perivoulatory period. The posterior pool from the vagina is aspirated within 2 hrs of coitus. Normal 10-50 motile sperms/HFP is considered as normal. < 10 sperms or absent sperms or dean sperms are cases for extensive evaluation of the male partner.

50. What is sperm penetration test?

Capability of a live sperm to penetrate, zona free hamster egg, *in vitro* is called as sperm penetration.

51. What is Miller-Kurzrok test?



Normal cervical mucus permits invasion of motile sperm.

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Ovarian Tumors

Swati S Shiradkar

TYPES

1. Nonneoplastic
   - Retention cyst
   - Functional cyst
   - Chocolate cyst
   - TO mass
2. Neoplastic (Benign/malignant)
   - Epithelial tumors
     - Stromal cell tumors
     - Germ cell tumors

POINTS TO BE NOTED IN HISTORY

a. Benign tumors are comparatively asymptomatic unless complication of torsion or rupture occurs. Malignant tumors produce vague symptoms. Functioning ovarian tumors produce menstrual disorders.
b. Type of ovarian tumor is different in different age groups, e.g. dermoid in young girls.
c. Nulliparity, induction of ovulation increases the risk while multiparity, use of OC pills, tubal ligation, breast feeding is protective.
d. Family history of ovarian, lung, colonic malignancy increases the risk.

GENERAL EXAMINATION

a. Evidence of weight loss points towards malignancy.
b. Evidence of hirsutism, obesity points towards functioning ovarian tumors.
c. Tachycardia associated with pain and tenderness points towards torsion.

PER ABDOMEN EXAMINATION

- Characters of benign tumors:
  - Usually free fluid is absent
  - Regular, cystic, mobile nontender tumors
  - Size variable.
- Characters of twisted ovarian tumors:
  - Regular, cystic, tender mass, mobility restricted.
  - Guarding rigidity present, signs of acute abdomen.
- Characters of malignant ovarian tumors:
  - Free fluid usually present/ascitis +

- Irregular, variable consistency and mobility nontender (unless bleeding or rupture of capsule) fixed mass
- Rapidly growing tumor.

PER SPECULUM EXAMINATION

- Position of cervix may be shifted to same side of mass as uterus is deviated to opposite side.

PER VAGINUM EXAMINATION

- Confirmation of findings on abdominal examination
- Cervical movements will not be transmitted to mass as mass is separate from uterus (unless there are dense adhesions with uterus)
  D/D of pedunculated fibroid
- A knotch will be felt between mass and uterus
  D/D of pedunculated fibroid, pelvic TB, TO mass
- Tumor felt in anterior fornix with tenderness, more chances of torsion
- Fixed tumor in posterior fornix, more chances of malignancy.

PER RECTAL EXAMINATION

- Nodularity felt in posterior fornix goes in favor of malignancy.

CONFIRMATION OF DIAGNOSIS

a. USG + color Doppler—Morphological index score
b. CT scan—To know about involvement of surrounding structures, nodes
c. Markers—Mainly carcinoma 125 for epithelial ovarian tumors
   — < 30 m /ml is normal
   — > 96 m/ml in postmenopausal, high risk of malignancy
d. Risk of malignancy index.

QUESTIONS

1. What are the differential diagnosis of an ovarian mass?

- Full bladder
- Pregnant uterus
- Myoma
- Ascites.

2. What is uni feature of ovarian malignancy?

Because of multicenteric origin, there can be epithelial tumor, stromal cell tumors and germ cell tumors. It makes diagnosis critical.

3. What are the features indicating malignancy on USG?

a. Size/Volume (> 8 cm in premenopausal)
b. Papillary projections from inner wall
c. Bilaterality
d. Solid tumors
e. Multiloculations.

f. Presence of ascites
g. Involvement of other organs
Each point carries 1 score.

4. What is role of colour Doppler in diagnosis of ovarian malignancy?

Neoangiogenesis characterized by:

- A-V shunts
- Microaneurysms
- Coiling
- Complex irregular branching
- Central vessels

Most useful in postmenopausal women.

5. What is risk of malignancy index?

- It is index to predict risk of malignancy in given patient.
- Factors considered are:
  a. Menopausal status (M)
     – Premenopausal—score 1
     – Postmenopausal—score 3
  b. Ultrasound morphology scoring (U)
  c. Actual serum CA 125 level.
     – RMI = M × U × S. CA 125 level
     – If > 300 chances of tumor being malignant are high.

6. How to decide line of management of in premenopausal ovarian tumor?

- Cyst < 6 cm, no symptoms, no evidence of malignancy
  Treatment of choice is observation with hormonal suppression of HPO axis
- Cyst > 8 cm, increasing size, symptoms suggesting complications, suspicion of malignancy, exploration is must
- Extent of surgery depends on age, parity and findings at laparotomy
- Treatment may range from:
  – Removal of cyst only
  – OR removal of cyst along with that sided ovary and tube
  – OR Total abdominal hysterectomy with bilateral salphingoopherectomy.
  – OR Total staging laparotomy with radical surgery.

7. What is line of management in postmenopausal ovarian tumor?

- Exploration is must
- Extent of surgery depends on RMI and findings at laparotomy
- In case of suspected benign, total abdominal hysterectomy with bilateral salphingo-oophorectomy
- In case of suspected malignant, staging laparotomy with maximum possible debulking.

8. What are the steps of staging laparotomy?

- Adequate vertical incision.

- Collection of ascetic fluid or peritoneal wash for cytology.
- Examination of liver capsule, undersurface of diaphragm, parietal peritoneum, mesentery, omentum, intestinal surface, palpation of lymph nodes, biopsy from omentum and other suspected sites.
- Examination of tumor itself noting bilaterality, morphology, surface, evidence of local infiltration.
- Decision of extent of surgery, maximum possible debulking, aiming at 5 cm residual disease.

9. What are the advantages of cytoreduction?

- Reduction in ascitis
- Removal of omental cake will reduce nausea.
- Removal of intestinal metastasis will improve intestinal function, nutrition and tolerance to CT and RT.
- Minimal cells in GO phase so better response to CT
- According to fractional cell kill hypothesis less no. of cells at end.
- Requires less duration/dose of CT so better patient compliance.

10. What is plan of T/t in stage Ia, b

- TAH with BSO with omentectomy, follow-up, no need of adjuvant therapy.
- If desirous of childbearing, incase of Ia, conservative surgery with careful follow up and radical surgery as soon as childbearing is complete.

11. What is plan of T/t in stage Ic onwards?

- TAH with BSO with omentectomy.
- Adjuvant therapy, either chemotherapy or in selected cases radiotherapy.
- Second look laparotomy/scopy to confirm response.
- Follow up with SCA-125.

12. What should be plan of T/t if radical surgery is not possible on exploration?

- Maximum feasible cytoreduction at first exploration.
- Three cycles of combination chemotherapy
- Interval cytoreduction
- Remaining three cycles of chemotherapy
- Second look surgery.

13. What is second look surgery?

It is a laparotomy/scopy aiming to:

- Confirm response of therapy after completion of cycles, where there is no residual disease on CT scan, CA-125 levels normal.
- To complete cytoreduction if evidence of residual disease.
- To take decision to change combinations of drugs.

14. What are the prognostic factors in ovarian malignancy?

- Pathological—Histological grade and differentiation.
- Biological—Nuclear characters (ploidy)
- Clinical—Stage, extent of residual disease, presence of ascitis, age

Overall prognosis is 25% 5 year survival.

15. What is secondary laparotomy?

After giving chemotherapy, patients with persistent or recurrent pelvic and abdominal tumors, are candidates for secondary cytoreduction (usually within 9 to 12 months).

16. What are the treatment options available for treatment of malignant ovarian tumor?

- Surgery
- Chemotherapy
- Radiotherapy
- Immunotherapy
- Combined approach.

17. How do you stage Ca ovary?

FIGO staging:

- Stage-I → Growth limited to ovaries
- Stage-II → Growth involving one or both ovaries with pelvic extension.
- Stage-III → Tumor involving one or both ovaries with peritoneal implant outside the pelvis
- State-IV → Distant metastasis.

18. What is the significance of staging ovarian tumor at laparotomy?

It is done to confirm the diagnosis, nature of disease and to assess extent of disease.

19. Why prognosis is bad?

- Early diagnosis is difficult as condition causes vague symptoms and lack of appropriate markers makes mass screening difficult
- Suboptimal surgery either because malignancy is not suspected or surgeon is not experienced to handle such case
- Requirement of radical surgery and side effects of adjuvant therapy increase morbidity.

20. What is advantage of combination of chemotherapeutic drugs?

- Less dose of each drug, decreasing individual toxicity
- Less chances of development of resistance
- Combination of drugs acting at different cell phases increases response in the form of cell kill.

21. Complication of benign ovarian cyst.

- Torsion/axial rotation
- Rupture
- Pseudomyxoma peritoni
- Infection
- Extraperitoneal development.

22. Classification of ovarian tumor?

I. Common epithelial tumors
   a. Serous tumors
   b. Mucinous tumors
   c. Endometroid tumors
   d. Clear cell
   e. Brenner tumors
   f. Mixed epithelial tumors
   g. Undifferentiated carcinoma
   h. Unclassified epithelial tumors.

II. Sex-cord tumors:
   a. Granulosa—stromal cell tumors, theca cell tumors.
   b. Androblastomas: Sertoli—Leyding cell tumors.
   c. Gynandroblastomas.
   d. Unclassified.

III. Lipid (lipoid) cell tumors.
   a. Dysgerminoma
   b. Edodermal sinus tumor
   c. Embryonal carcinoma
   d. Polyembryoma
   e. Choriocarcinoma
   f. Teratoma
   g. Mixed forms.

IV. Gonadoblastoma:
   a. Pure
   b. Mixed with dysgerminoma or other germ cell tumors.

V. Soft tissue tumors not specific to ovary.
VI. Unclassified tumors.
VII. Tumor—like conditions.

23. What is Meig's syndrome?

Consist of:
a. Fibroma of ovary
b. Hydrothorax
c. Ascites.

24. Differential diagnosis of ovarian cyst?

a. Full bladder
b. Pregnant uterus
c. Myoma
d. Ascitis
e. Other tumors.

25. What is parovarian cyst?

Extra peritoneal cyst lying in the broad ligament adjacent to the ovary, below the fallopian tube.

26. What are non-neoplastic causes of ovarian cyst?

- Follicular cyst
- Corpus luteum cyst
- Theca lutein and granulosa lutein cyst
- Polycystic ovarian syndrome
- Endometrial cyst.

27. What is Krukenberg tumor?

Type of secondary ovarian carcinoma they are:

- Bilateral
- Have smooth surfaces which may be slightly bossed.
- Are freely movable in the pelvis.
- Tumors are secondary growth in ovary and most often arise from primary carcinoma of stomach (70%), large bowel (15%) and breast (6%). They arise by retrograde lymphatic spread.

28. What are contents of ovarian pedical?

- **Laterally**
  Infundibulopelic ligament containing structures therein, i.e. ovarian vessels, nerves and lymphatics.
- **Medially**
  Ovarian ligament
  Medial end of the Fallopian tube
  Mesosalpinx containing utero-ovarian anastomotic vessels.
- **Middle**
  Broad ligament.

29. What is pseudomyxoma peritonea?

It is caused due to rupture of mucinous tumors. There is spillage of mucinous material in the peritoneal cavity which leads to adhesion formation. It is recurrent.

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Cancer of Cervix

SR Wakode

1. What is the most common cancer in female next to breast?

Cancer cervix, 80% of genital cancers in India.

2. What are etiological factors for cervical cancer?

Factors predisposing cervical cancer are:

- Early coital activity and child bearing
- More frency of intercourse
- Multiplicity of sexual partners
- Low socioeconomic status
- Viral infections—herpes virus type 2
- Human papilloma virus (16, 18, 31, 33)
- Prostitutes with HIV and STD.

3. How invasive cervical cancer develops in cervical epithelium?

Invasive cervical cancer preceded by dysplastic changes in the cervical epithelium.

4. What is cervical intraepithelial neoplasia?

Histopathological description where part or whole of the thickness of the cervical epithelium is replaced by cells showing varying degree of dysplasia.

5. What is meant by dysplasia?

Dysplasia means a change causing an alteration and disorderly arrangement of the cells of the stultified squamous epithelium.

6. What are the characteristics and dysplastic cells?

The cells are:

- Variable in size and shape
- Large, irregular and hyperchromatic nuclei
- Mitotic figures found in cells.

7. What are types of cervical dysplasia?

- Mild dysplasia (CIN-I)—Undifferentiated cells are confined to the lower third of the epithelial layer.
- Moderate dysplasia (CIN-II)—The lower 50-75% of the epithelial layer is undifferentiated severe dysplasia and The entire thickness of the epithelium
- Carcinoma *in situ* (CIN-III)—This is replaced by anaplastic cells.

8. What types of vaginal infection shows changes of mild cervical dysplasia?

Trichomoniasis.

9. How many years period requires for progression of dysplasia to invasive cervical cancer?

5-10 years period required.

10. How many percent of mild and severe dysplasia progress to cervical cancer?

0.5% of mild and 9.6% of severe dysplasia can progress to cervical cancer.

11. What are the methods of screening for cervical cancer?

Methods for cervical cancer screening are:

1. Pap smear (exfoliative cytology)
2. Speculoscopy
3. Micro-colpohysteroscopy
4. Colposcopy
5. Cervicography.

12. Who should be screened for cervical cancer?

Woman with:

a. Exposed to early sexual life
b. Sex with multiple partner
c. Multipara
d. HIV and HPV infection
e. Unhealthy cervix
f. Inter menstrual bleeding or discharge
g. Age group 25 to 45 years.

13. What are the precautions to be taken before collection of Pap smear?

Precautions to be taken are:

I. Smear collection before bimanual examination
II. No antiseptic application
III. No vaginal douching.

14. Collection of Pap smear done with:

a. Ayre's spatula
b. Cotton swab stick.

15. What is the fixative used for Pap smear?

95% absolute alcohol and ether and fixative sprays are available.

16. What are the reports/Grade of Pap smear?
Grade I—Normal cells
Grade II—Inflammatory cells
Grade III—Suspicious abnormality in cells
Grade IV—Distinctly abnormal, possibly malignant
Grade V—Malignant cells.

17. What is newer classification of Pap smear?
a. Normal cytology
b. Inflammatory smear
c. CIN-I (Mild dysplasia)
d. CIN-II, III and carcinoma *in situ*
e. Malignant cells seen.

18. How much is the incidence of dysplasia in India?
16/1000 patients.

19. What are reasons for false negative pap smear reports?
- Improper smear taking
- No access to squamocolumnar junction (menopausal woman)
- Laboratory error.

20. What you will do if report of pap smear is normal?
Repeat smear yearly for 3 years; then 3 yearly.

21. What are the indications for colposcopy?
Indications for colposcopy are:
a. Abnormal cytology
b. Diagnosis of suspicious invasive lesion
c. Diagnostic directed biopsy.
d. Conservative therapy under colposcopic guidance
e. Follow up cases treated conservatively.

22. What is cervicography?
New technic of screening for cervical cancer involving taking the photograph of the entire cervical os after applying 5% acetic acid to the cervix and sent for evaluation to colposcopist.

23. What are types of biopsy can diagnose cervical malignancy?
Following types of biopsy can diagnose cervical malignancy:
a. Punch biopsy
b. Colposcopic directed biopsy
c. Endocervical curettage
d. Cone biopsy
e. Large loop excision of the transformation zone.

24. How will you treat CIN-I?
CIN-I inflammatory in nature so treatment of infection is done and repeat smear after 3-6 months.

25. What are the treatment modalities for CIN II and III?
CIN II and III treatment modalities are:
I. Local destructive methods
II. Excision of local tissue
III. Surgery.

26. What are the local destructive methods for treating CIN II and III?
Local destructive methods used are:
a. Cryosurgery
b. Fulguration/coagulation
c. Laser ablation.

27. What are the methods of local tissue excision for CIN II, III?
Methods for local tissue excision are:
a. Conization by cold knife
b. Laser conization
c. Large loop excision of the transformation zone (LLETZ).

28. How will you treat CIN II and III in woman over 40 years of age?
CIN II and III in 40 years of woman is treated by—hysterectomy.

29. What are the indications for hysterectomy in severe dysplasia (CIN II, III)?
Indications are:
a. Women over 40 years of age
b. Parity completed
c. Associated problem like DUB, fibroids
d. Not able to do regular follow-up.

30. What type of cervix biopsy you take from cauliflower like lesion?
Punch biopsy.

31. What are the histopathological types of cervical cancer?
i. Squamous cell carcinoma
ii. Adenocarcinoma.

32. What predisposes younger women for endocervical cancer?
Prolonged used of oral contraceptives pills for > 8 years.

33. What are the types of lesion in cervical cancer?
I. Ulcerative growth
II. Cauliflower like lesion
III. Flat raised indication
IV. Endocervical cancer shows barred shaped bulky cervix.

34. What are the modes of spread in cervical cancer?
Cancer cervix spreads by:
a. Direct spread locally
b. Lymphatic spread
c. Blood spread
d. By implantation.

35. What are the commonly involved structures with direct spread of cancer cervix?

Direct spread of cervical cancer occurs to:

a. The parametria on both side
b. Upper part of cervix
c. Endometrium, myometrium and body of uterus.
d. Anterior vaginal wall
e. Bladder
f. Rarely fallopian tubes and ovaries.

36. What group of lymph nodes involved in cancer cervix?

a. Lymph nodes in parametrium
b. Obturator
c. External iliac
d. Hypogastric glands
e. Rarely aortic and lumbar glands.

37. What are the cardinal symptoms of cancer cervix?

There are four main symptoms of cancer cervix:

a. Hemorrhage
b. Discharge
c. Cachexia
d. Pain.

38. What type of bleeding pattern exist in carcinoma cervix?

- Postmenopausal bleeding
- Metrorrhagia (intermenstrual irregular bleeding)
- Post coital bleeding
- Blood stained vaginal discharge.

39. What are the causes of pain in cancer cervix?

Causes of pain in cancer cervix are:

a. Parametritis
b. Lymph nodes involvement at bifurcation of common iliac any causing involvement of obturator nerve
c. Involvement of bladder
d. Sacral plexus involvement by growth
e. Pyometra.

40. What are the four main (Cardinal signs of cancer cervix)?

Four main (cardinal) signs are:

a. Growth bleeds on touch
b. Growth is friable
c. Fixed cervix
d. Induration of cervix.

41. Differential diagnosis of vaginal bleeding following digital examination of cervix?

- Carcinoma cervix

- Vascular erosions ectropion
- Mucous polyp
- Myomatous polyp
- Retained products of conception.

42. Enumerate the list of investigations in a patient of Ca Cx?

Investigations to be done in Ca cervix are:

a. Pelvic examination under anaesthesia
b. Chest X-ray
c. IVP
d. Cystoscopy
e. Proctosigmoidoscopy
f. Colposcopy
g. Endocervical curettage
h. Bone survey
i. CT scan
j. Bone scan
k. Retroperitoneal peri aortic nodes sampling
l. Scalene fat pad biopsy.

43. What is pre invasive carcinoma (stage)?

Carcinoma in situ cases of stage o should not be included in any therapeutic statistics.

44. What are the treatment modalities for cancer cervix?

Treatment modalities are:

a. Surgery
b. Radiotherapy
c. Combined therapy
d. Chemotherapy.

45. What are the indications for Wertheim's hysterectomy?

Removal of total uterus along with upper 3rd of vaginal cuff and bilateral salpingo-oophorectomy with pelvic lymphadenectomy.

Cervix can be conserved in young women.

46. How much is mortality with Wertheim's hysterectomy?

1% mortality.

47. What are the complications of Wertheim's hysterectomy?

Hemorrhage, bladder injury, ureter injury, causing ureteric fistula, sepsis, thrombophlebitis, embolism, paralytic ileus, chest infection, wound sepsis, burst abdomen, scar hernia, mortality.

48. Radiotherapy is indicated in which stages of Cervix cancer?

Stage IIB to stage IV radiotherapy is given.

49. What are the technique of radiotherapy?

Brachytherapy and teletherapy.

50. What is brachytherapy?
It consists of intracavitary radiation using high activity "After loading" remote controlled system.

51. What are the sources of radiation?
Cobalt, Cesium or Selectron is the source of radiation.

52. Indications for chemotherapy in cancer cervix.
- Recurrence of lesion
- Adenocarcinoma of endocervix.

53. What are the drugs used for chemotherapy?
Cisplatin, ifosfamide, cyclophosphamide, vincristine, methotrexate, thiotepa, doxorubicin, epirubicin.

45 Endometrial Carcinoma

Kurdukar DV

Though the carcinoma of endometrium is not so commonly seen as the carcinoma of cervix, as a basic physician one must remember that this is one of the most important causes of **postmenopausal bleeding**.

It won't be an exaggeration that all cases of postmenopausal bleeding be considered as carcinoma of endometrium unless proved otherwise.

The author is aware of the fact that all the aspects related to this condition may not be covered in this set of stionnaire along with very short answers.

And therefore only expectations of and from a basic doctor during examination and while working in resource limited settings are considered and presented herewith as 'Frently asked stions'. Prepared only for an examinee for whom a thorough preparation of this vast subject is impossible overnight.

1. What are the causes of post menopausal bleeding (PMB)?

The causes of post menopausal bleeding are:

a. Organic lesions of the female genital tract, e.g. mainly malignant lesions of vulva, vagina, cervix, uterus, fallopian tubes and ovary.

b. Benign lesions which cause of post menopausal bleeding are atrophic:
   - Endometritis, and senile vaginitis.
     Other uterine lesions which cause of post menopausal bleeding are: Polyps—2-12% , hyperplasia—15-25% cases.
   - Some iatrogenic conditions are tamoxifen given for breast cancer and estrogen replacement therapy for menopausal symptoms also known as 'the youth pill.'

2. Which are the 'high risk factors for the carcinoma of endometrium'?

a. Genetic predisposition, e.g. history of hereditary non-polyposis colon cancer (HNPCC)

b. Late menopause > 52 years risk 2.4 times higher

c. Obesity weight > 10-25 kg risk 3 times and > 25 kg that expected risk 10 times.

d. Diabetes mellitus

e. Hypertension C, D and E together are called as Corpus cancer syndrome
f. Low parity
g. Unopposed estrogen activity:
   - *Endogenous,* e.g. feminizing tumors of ovary, i.e. granulosa or theca cell tumors of ovary
   - *Exogenous,* e.g. estrogen replacement therapy for menopausal symptoms
h. Tamoxifen therapy for breast cancer.

3. What is the histopathological nature of carcinoma of endometrium?

The histopathological nature of carcinoma of endometrium is adenocarcinoma occasionally adenosquamous and very rarely pure-squamous variety.

4. What are endometrial carcinoma precursors?

Different types of endometrial hyperplasia are known and their relative risk of developing into endometrial carcinoma is as follows:

- Simple hyperplasia without atypia—1%
- Complex hyperplasia without atypia—3%
- Simple hyperplasia with atypia—8%
- Complex hyperplasia with atypia—29% very important.

5. Can you prevent progression of hyperplastic endometrium developing into endometrial carcinoma?

Progestin therapy is more effective in preventing progression of hyperplastic endometrium *without* atypia developing into endometrial carcinoma than hyperplastic endometrium with atypia.

6. Are there any 'screening tests' for the diagnosis of endometrial carcinoma in early stages? Like carcinoma of cervix?

'Screening tests' for the diagnosis of endometrial carcinoma are neither warranted nor they are cost effective. However in high risk cases:

a. Pap smear is not very much useful to diagnose endometrial carcinoma in early stages as is useful to diagnose carcinoma of cervix.
b. Transvaginal ultrasonography (TVS) endometrial thickness.
   4 mm or polypoidal mass in the uterine cavity needs further evaluation and progesterone Challenge test have been recommended in post-menopausal cases but they do not have the cost benefit as the 'screening tests'.
   However patients on estrogen replacement therapy for menopausal symptoms and tamoxifen therapy for breast cancer form a high risk group for the carcinoma of endometrium' and these cases must be vigilantly kept under follow-up.

7. What are the main symptoms of endometrial carcinoma?

Pervaginal bleeding is the main symptom.

a. In premenopausal age group, the patient may complain of menometrorrhagia mainly.
b. All cases of postmenopausal bleeding be considered as carcinoma of endometrium unless proved otherwise.

c. Pain because of Colicky uterine contractions (Simpson's pain) in suprapubic region occurring at fixed time of the day lasting for 1-2 hrs.
d. With pyometra patient can have pain and fever.

8. What are the main signs of endometrial carcinoma?

Try to know the risk factors.

- Patient may have undergone D and C for menstrual abnormality. Review the report
- In general examination mention built (obesity) and record her blood pressure. Palpate for lymphadenopathy.
- Do not forget to examine breasts (also in cases of ovarian carcinoma).
- Per-vaginal and rectal examination to confirm the size of the uterus, to know mobility of the uterus, parametrial induration and nodularity of the cul-de-sac.
- Positive Clark's test (negative Clark's test doe not rule out endometrial carcinoma).

9. How will you confirm the diagnosis of endometrial carcinoma?

- All cases of peri- and postmenopausal bleeding must be subjected to endometrial sampling to confirm/rule out carcinoma of endometrium.
- Aspiration biopsy has the diagnostic accuracy of about 90-98%
- Endometrial lavage/brush, vabra aspirator, Tis-U trap, pipelle suction curette
- However, if patient tolerance or cervical stenosis does not permit aspiration biopsy or recurrence of pervaginal bleeding after previous negative/ inadequate biopsy D and C along with (if available) hysteroscopy should be done.
- TVS acts as an adjunct to endometrial sampling.

10. What is G1,G2 or G3 in endometrial carcinoma?

It refers to the histopathological feature of carcinoma of endometrium as follows:

- G1 < 5% Non-squamous non-morular frowth pattern
- G2 6-50% Non-squamous non-morular frowth pattern
- G3 > 50% Non-squamous non-morular frowth pattern.

11. What is squamous differentiation of carcinoma of endometrium'?

- About 15-25% endometrial carcinoma have areas of squamous
- *Differentiation.* Benign squamous differentiation was called as adeno-acanthoma and malignant adenosquamous carcinoma.
- Today both these terms are replaced by the term carcinoma of endometrium with squamous differentiation irrespective of the nature of the squamous component.

12. What is FIGO staging of endometrial carcinoma.?

FIGO staging of endometrial carcinoma is **surgical** and is as follows.

Stage	Subgroup of the Stage	Finding
Stage 'O'	-	Carcinoma *in situ* histologic finding s/o carcinoma
Stage I Cancer confined to the corpus	**IA** **IB** **IC**	Tumor limited to endometrium < or = half myometrial invasion > Half myometrial invasion
Stage II Tumor involves the cervix but no extension beyond the uterus	**IIA** **IIB**	Only endocervical gland involvement Cervical stromal invasion
Stage III Local and/or regional spread	**IIIA** **IIIB** **IIIC**	Tumor involves serosa, adnexa and Positive peritoneal cytology Presence of vaginal metastases Pelvic and para-aortic lymph node Metastases
Stage IV Distant metastases Tumor shows distant metastases	**IVA** **IVB**	Tumor involves bladder and/or Bowel mucosa

13. Describe briefly fractional curettage

Total six specimens endocervical (2) and endometrial (4) are obtained in Fractional curettage as follows. Specimens must be meticulously labelled

a. Endocervical specimen no. 1 from endocervical canal before uterine sounding and cervical dilatation
b. Endocervical specimen no. 2 from isthmus 1 cm beyond the Internal os after cervical dilatation
c. 4 Endometrial specimens are obtained after cervical dilatation as follows.
   Right and left cornual specimen one each (Total 2)
   Anterior and posterior wall of the uterus one each (Total 2).

14. What is the pretreatment evaluation of a case of endometrial carcinoma?

- Review of detailed history and thorough physical examination (Ref to relevant above.)
- High risk factors, associated systemic problem
- CBC chest X-ray, ECG for cardiorespiratory status
- Liver and renal function tests
- Stools for occult blood
- Hysteroscopy (risk of spillage)
- IVP, cystoscopy, rectosigmoidoscopy, Ba enema
- CT abdomen and pelvis to be done as per the merit of the case
- USG and MRI to know the extent of myometrial invasion
- CA-125 to know the extent of extrauterine disease.

15. What are the prognostic variables?

Variable	Good prognosis	Bad prognosis
Age	Younger	Old
Smoking	Heavy	Non-smoker
Histology	Endometroid Type	Non endometroid
Grade	G1/G2	G3
Myometrial invasion	Superficial	Deeper or extrauterine Disease
LVSI*	Absent	Present
Extension to isthmus and cervix	Absent	Present
Size of the tumor	< 2 cm	> 2 cm
Receptors	Positive for estrogens As well as progesterones	Negative for Progesterones
DNA ploidy	Diploidy	Non-ploidy
Genetic	Presence of K-ras oncogene	

*LVSI—Lymph space vascular invasion

Good prognosis: White, obese, nulliparous with well differentiated, superficially invasive, EP receptor positive with high cure rate after TAH with BSO.

Bad prognosis: Black, thin, parous poorly differentiated, deeply invasive, EP receptor negative with metastases.

16. What are different treatment options?

Surgery is the main stay.

a. Total abdominal hysterectomy with bilateral salpingo-oophorectomy along with peritoneal cytology and omentectomy and lymph node sampling.
b. Today laparoscopically assisted vaginal hysterectomy is one more option
c. Vaginal hysterectomy has a limited scope as lymph node sampling and adnexectomy is not possible (very high surgical risks with well differentiated stage I cases).

17. What is adjuvant therapy for endometrial carcinoma?

- Radiotherapy with external beam irradiation or intracavitary
- Intraperitoneal phosphorus.

18. What are the different drugs used in the treatment of endometrial carcinoma?

Hormones progestins for peritoneal cytology positive cases.
Chemotherapeutic agents as doxorubicin, cisplatin, carboplatin and palcitaxel.

19. Can endometrial carcinoma be prevented?

Screening for high risk factors including endometrial sampling before:

a. Estrogen replacement therapy for menopausal symptoms
b. Patients on tamoxifen therapy for breast cancer form a high risk group for the carcinoma of endometrium and these cases must be vigilantly kept under follow up.
c. Premalignant lesions followed and treated
d. Estrogen replacement therapy for menopausal symptoms should be combined with progestogens to counteract unopposed estrogen activity.

Multiple Choice Questions

1. Only one of the following triads is true for endometrial carcinoma

a. Obesity + Hypertension + Diabetes mellitus
b. Obesity + Nulliparity + Diabetes mellitus
c. Obesity + Nulliparity + Hypertension
d. Diabetes mellitus + Nulliparity + Hypertension

2. Only one of the following is a cause and treatment for endometrial carcinoma.

a. Estrogen replacement therapy for menopausal symptoms
b. Tamoxifen
c. Progestogens
d. Gn RH analogues.

3. Only one of the following predisposes to endometrial carcinoma.

a. Combined oral contraceptive pill
b. Sential Pills
c. Minipills
d. None of these.

4. Endometrial carcinoma is commonly associated with.

a. Feminizing tumors of ovary
b. Masculinizing tumors of ovary
c. Endometroid tumors of ovary
d. Germ cell tumors of ovary

5. Only one of the following ovarian tumors predisposes to endometrial carcinoma.

a. Sertoli cell tumor
b. Leydig cell tumor
c. Sertoli-Leydig cell tumor
d. Granulosa cell tumor

6. All of the following are commonly associated with endometrial carcinoma, *except:*

a. Obesity
b. Nulliparity
c. Estrogen replacement therapy for menopausal symptoms
d. HSV II infection.

7. Pain occurring at regular time of the day in a case of endometrial carcinoma is known as:

a. Sim's pain
b. Sims' pain
c. Simpson's pain
d. Sampson's pain.

8. Bleeding after uterine sounding in a case of endometrial carcinoma is known as:

a. Clarke's test
b. Clement's test
c. Cullen's test
d. Cunningham's test.

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Endoscopy

Alka Kriplani, Sumana G

1. What are the indications for laparoscopy?

Indications for diagnostic laparoscopy are:

- Evaluation of a patient with infertility; to look at pelvic pathology, tubal status, patency, adhesions, endometriotic spots, tubercles.
- Acute or chronic pelvic pain
- Ectopic pregnancy
- Endometriosis
- Ovarian cysts
- Acute adnexal torsion
- Pelvic inflammatory disease; when diagnosis is not clear
- To diagnose and treat müllerian duct anomalies.

2. What are the contraindications for laparoscopy?

a. Severe cardiorespiratory disease
b. Large diaphragmatic hernia
c. Generalized peritonitis
d. Ileus/intestinal obstruction
e. Inexperienced surgeon
f. Hemodynamic compromise/instability
g. Large abdominal masses/advanced pregnancy.

3. What are the precautions to be taken at abdominal entry of Veress needle?

a. The abdomen is lifted up; this elevates the umbilicus and puts the peritoneum at stretch
b. The intra-abdominal pressure is kept at 20-25 mm Hg
c. The veress needle is inserted at right angles to the skin directed into the axis of the true pelvis
d. In obese patients, the needle may have to be inserted more vertically.

4. What are the tests done to check peritoneal entry?

a. Hanging drop test—this observes a hanging drop of fluid in the veress needle that flows downward freely if the needle is in the peritoneal cavity

Lifting of the abdominal wall decreases intra-abdominal pressure and enhances the free flow

b. A syringe barrel is attached to the veress needle and 1-2 ml saline poured into it; if the needle is intraperitoneal, the fluid will flow freely into the abdomen
c. Aspiration can be done with a syringe: to make sure that the four B's should be absent—blood, bile, bladder, bowel
d. Gas is insufflated at a low flow rate of 1 lt/min, with the pressure below 10 mm Hg. With patient's respiratory efforts, there should be slight elevation in the pressure if the needle is intraperitoneal
e. If gas is insufflated and the pressure is persistently >10 mm Hg, the needle is probably preperitoneal or has entered the viscus or the omentum
f. At flow rate of 1 Lt/min, in 45-60 sec there should be loss of liver dullness.

5. In difficult cases what are the alternate points of entry?

a. Supraumbilical: Large pelvic masses or previous scar
b. Left/right lower quadrant; lateral border of the rectus at left or right McBurney's point
c. Palmer's point: Left upper quadrant at the lateral border of the rectus 2 finger breadths below the left costal margin.

6. Which is the most dangerous part of the laparoscopic surgery?

a. Primary trocar entry.

7. What is open laparoscopy?

- This technic is used to minimize risk of large vessel and bowel injury during entry
- Useful in patients with previous abdominal surgeries
- Procedure:
  - — A vertical skin incision is given over the umbilicus
  - — Two strong sutures are placed on both the edges of the facial entry
  - — The sutures are lifted to elevate the abdominal wall for blunt or sharp entry into peritoneum
  - — A smooth tipped Hasson's trocar and sleeve are inserted with an acron shaped metal obturator.

8. What are the various laparoscopic methods of sterilization?

- Sialistic ring
- Filschie clip
- Unipolar coagulation
- Bipolar coagulation
- Hulka clip.

9. Why does laparoscopy result in fewer adhesions compared to laparotomy?

a. Absence of retraction and packing that can damage peritoneum
b. Gentle handling of the tissue
c. Lack of drying in room air
d. Absence of foreign bodies.

10. Which type of coagulation has lesser peripheral damage?
Bipolar coagulation.

11. Why is CO_2 the preferred gas for creating pneumoperitoneum?
a. Rapidly absorbed by blood
b. Less likely to lead to embolism
c. Does not support combustion.

12. What is the maximum pressure setting during the laparoscopic procedure?
a. The pressure should not exceed 15 mm of Hg.

13. What is gasless laparoscopy and what are its disadvantages?
a. It is a method that has been introduced in an attempt to provide space in which to operate without the need for continuous gas replacement
b. The anterior abdominal wall is elevated mechanically with a special device
c. Disadvantages:
   i. Pneumoperitoneum causes a dome like elevation of the abdominal wall; the devices used here lift the abdominal wall only in the midline and not in the flanks.
   ii. Access to the upper abdomen and POD is restricted
   iii. There is no increased intra-abdominal pressure to push the bowel away
   iv. The hemostatic effect of pneumoperitoneum does not exist.

14. What are the various laparoscopic procedures possible in endometriosis?
a. Division and resection of the adhesions
b. Excision and ablation of the peritoneal implants.
c. Restoration of the tubo-ovarian relationship
d. Endometriomas—small lesions: electrodessicated, laser ablated or excised. Large cysts: removed by cystectomy
e. Restoration of cul-de-sac by adhesiolysis
f. Ablation of uterosacral ligaments or presacral neurectomy.

15. What are the complications of laparoscopy?
a. Due to anaesthesia:
   i. Metabolic disturbances
   ii. Absorption of CO_2—arrythmias
   iii. Inadequate oxygenation due to overdistention of the abdomen/ steep Trendelenburg.
b. Hemorrhage:
   i. Superficial
   ii. Major vessels: inferior epigastric arteries, aorta, iliac vessels.
c. Perforation of viscus: small bowel, large bowel, stomach.
d. Perforation of the uterus.
e. Extravasation of the gas into extraperitoneal space:
   i. Subcutaneous emphysema
   ii. Mediastinal emphysema.

f. Gas embolism.
g. Intra-abdominal burns/explosion.
   i. Release of methane gas from bowel perforation:
   ii. Electrocautery injuries.
h. Injury to the bladder, ureter.

16. What are the various methods of controlling hemorrhage from an injured inferior epigastric artery?

a. Incision can be widened and the artery ligated under vision
b. A figure of 8 suture placed on either sides of the trocar.
c. From another trocar entry, bipolar coagulation of the vessel or laparoscopic suturing can be done
d. A Foley's catheter can be inserted and the bulb inflated. This can be then pulled and traction applied.

17. Which laparoscopic procedures have highest incidence of ureteric injury?

a. Infundibulopelvic ligament
   i. Oophorectomy
   ii. Adhesiolysis
   iii. Presacral neurectomy
   iv. Endometrial ablation
b. LUNA—uterosacral plication
c. Hysterectomy—closure of vaginal cuff.

18. What are the various methods of avoiding vascular injury at secondary trocar placement?

a. Insertion under vision, by transillumination
b. Operator can alter the trocar's trajectory
c. Accessory trocar to be placed medial or 2-3 cm lateral to umbilical ligaments to avoid injuring these vessels.

19. What are the indications for laparoscopic hysterectomy?

To transform an abdominal hysterectomy into a vaginal one.

Avoid abdominal hysterectomy

Traditional contraindications for vaginal hysterectomy (and hence indications for laparoscopic assistance) include:

a. Poor access to ovaries
b. Nulliparity without descent
c. Previous pelvic surgery
d. History of pelvic inflammatory disease
e. Moderate to severe endometriosis
f. Adnexal masses
g. Known pelvic adhesions
h. No mobility of the uterus
i. Stage I endometrial cancer
j. Uterine size > 280 gm
k. Needing adnexectomy.

20. Definition of laparoscopic hysterectomy?

a. Division of all major uterine pedicles and supports by laparoscopy
b. Reich's definition: "Ligation of uterine arteries laparoscopically."

21. Media used in hysteroscopic surgery.

Hyskon, normal saline, CO_2, glycine 1.5%, sorbitol 3%.

22. What are the indications for diagnostic hysteroscopy?

a. Infertility: to rule out any polyps, adhesions, fibroids, cavity
b. Postmenopausal bleeding
c. Fibroid uterus; diagnose submucous fibroids
d. Abnormal uterine bleeding not responding to medical management
e. Bad obstetric history: uterine anomalies, submucous fibroids, intrauterine adhesions
f. Misplaced intrauterine device.

23. What are the complications of hysteroscopy?

- Fluid overload due to absorption of the media
- Hyponatremia (absorption of hypoosmolar media)
- Intra and post operative bleeding
- Uterine perforation and possible bowel injury.
- Poor visibility
- Gas embolism (CO_2)
- Infection.

24. What are the methods of controlling bleeding complicating hysteroscopy?

a. Aspirate the blood and increase the pressure of the distending medium
b. The bleeding vessel can be coagulated with electrocautery
c. Intrauterine balloon can be inflated and left in situ for 6-12 hours.

25. What is an optical trocar?

Trocars that contain a window at the tip and are designed to handle a laparoscope to look through the window to enable surgeons to view the tissue layers during penetration and to see the abdominal cavity after penetration.

26. What are the indications and contraindications for laparoscopic myomectomy?

a. *Indications*
   i. Pedunculated myomas
   ii. Intramural myomas < 10 cm.
b. *Contraindications*
   i. Diffuse, multiple myomas
   ii. Large uterus > 18 weeks
   iii. Myomas > 10 cm
   iv. Inexperienced surgeon unfamiliar with endosuturing
   v. Submucous > 50 % extension into cavity.

27. What is the classification of uterine adhesions and its management?

a. *Severe*
   i. > ¾ cavity involved
   ii. Agglutination of walls or thick band
   iii. Ostial area and upper cavity occluded.
b. *Moderate*
   i. ¼ to ¾ cavity involved
   ii. No agglutination/adhesions present
   iii. Ostial area and upper cavity partially occluded.
c. *Minimal*
   i. Less than ¼ cavity involved
   ii. None flimsy adhesions
   iii. Ostial area and upper fundus normal.

Treatment

- *Minimal:* Lysed dissection through telescopic sleeve
- *Moderate and severe:* Scissor dissection with operating hysteroscope
- Electrocautery with resectoscope.

Causes

Vigorous curettage, post abortal or postpartum sepsis, Asherman's syndrome secondary to PPH, endometrial tuberculosis, IUCD use, post submucous myomectomy or metroplasty.

28. What is a microhysteroscope and what are its uses?

- This contains a magnifying lens built into the telescope.
- Magnification up to 150 × can be achieved
- Used for studying vascular and glandular structure of the cervix, endometrial lining and tubal mucosa.

29. What are the various methods of laparoscopic management of distal tubal disease?

a. *Fimbriolysis:* Adhesiolysis around the fimbria, tube patent following procedure
b. *Fimbrioplasty:* Adhesiolysis followed by dilatation of fimbrial phimosis by dilator or atraumatic forceps
c. *Salpingostomy:* Terminal hydrosalpinx with a star shaped occluded infundibulum; radial incisions made at ostia and edges everted.

30. What is the technique of ovarian drilling?

The ovarian surface inspected and intestines are kept at a distance. A bipolar or unipolar needle or a Versapoint probe is used. The needle is inserted into the ovarian capsule 0.8 cm deep. Small needle diameter is used to decrease complication rates. No consensus about the number of holes to be made. 10-15 holes can be made in each ovary.

31. What is the European Society of Hysteroscopy classification of fibroids? What are the contraindications for hysteroscopic myomectomy?

Type 0: pedunculated, no intramural extension
Type 1: sessile submucous, < 50 % intramural extension
Type 2: sessile submucous, > 50 % intramural extension

Hysteroscopic myomectomy is done in submucous fibroids. Greater success rates in type 0 and 1 fibroids.

Contraindications include

a. Suspected malignancy
b. Rapidly enlarging mass or irregular vaginal bleeding
c. Cardiac, pulmonary or renal disorder due to fluid overload
d. > 12 weeks size or grossly enlarged uterus.

32. What are the methods of hysteroscopic ligation?

a. Ovabloc system
b. Premoulded intratubal device
c. Tubal screws
d. Essure system.

33. What is the classification of laparoscopic hysterectomy?

JOHNS AND DIAMOND classification:

0 Diagnostic laparoscopy performed before vaginal hysterectomy; no laparoscopic surgery required
1 laparoscopic adhesiolysis or excision of endometriosis before VH
2 Either one or both adnexa freed including infundibulopelvic ligament laparoscopically
3 Bladder dissected from the uterus laparoscopically
4 Uterine arteries transected laparoscopically
5 Anterior and posterior colpotomy done and freeing of the whole uterus laparoscopically.

SUGGESTED READING

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