

Antenatal care Module part 2

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RH ISOIMUNIZATION

Objectives

- At the end of this session, students should be able to
- Diagnose RH in compatibility and Iso-immunization

ABO System & Pregnancy

- Majorities of hemolytic diseases are due to **ABO** incompatibility
- Foetus inherits one gene from each parent
- There is a **20% chance of ABO incompatibility** of mother & foetus
- Only **5%** chance of developing hemolytic disease in type A & B infants of type O mothers, that is only of milder forms

Con't

- Newly discovered property of serum is **Rh factor** (ab directed against **RBC** surface Ag
- **85%** of Individuals with agglutinated RBs classified as **Rh**
(rhesus) +ve
- **15%** of the population whose cells didn't react=Rh –ve remains the leading cause of fetal/neonatal death

Genetic characteristics of Rh antigen

- Five major antigenic loci determining Rh status
-C,D,E,c,e
- presence of D antigen results Rh +ve individual
- Genotypic nomenclature
 - C is the alternative to c
 - E is the alternative to e
 - no phenotypic alternative to D is found

Rh isoimmunization/sensitization

Definition -any woman with antibody titer of $>$ than 1:4 is considered as **Rh** sensitized

Causes-fetus must have Rh +ve RBCs & Mother Rh-ve RBCs

- sufficient No of fetal RBCs must get into Maternal circulation

- Mother must have immunologic capacity to produce antibodies directed against the D-antigen

Incidence of Rh incompatible pregnancy

- 15% in Whites (most often in Caucasians)
- 5-8% in Black Americans
- 1-2% in Asiatic groups
- 34 % in Basques of Spain

In Whites population

- Rh –ve woman has **85%** chance of mating Rh+ve Man
- Approximately **60%** of Rh +ve men are **heterozygous** and **40%** of Rh+ve men are **homozygous**
- The recurrence risk of Rh+ve fetus due to homozygous father will be 100% and 50% with heterozygous father
- Over all chance of an Rh +ve man producing Rh +ve fetus is 70%

Con't

- The initial exposure to the D-antigen results production of **IgM** antibody (doesn't cross placenta)
- Subsequent exposure to D-antigen results production of maternal **IgG** (cross placental barrier)
 - response time of 2nd immunization is much shorter
 - requires only small Rh exposure

The risk of Rh sensitization for all Rh –ve ABO compatible woman

- **8%** after **first Rh +ve pregnancy**
- 16% total risk b/c 8% another risk is added for 2nd pregnancy
- Over all risk of anti D immunization seems < than 50% after infusion of **< than 30ml** of Rh incompatible cells with multiple deliveries
- Risk with infusion of large volumes (200ml) is > than **80%** but not **100%**

Detection of fetal RBCs in maternal circulation

- 7% of pregnancies in 1st trimester
 - 16% of >> in 2nd >>
 - 29% of pregnancies in 3rd >>
- approximately 50% of normal deliveries have measurable transplacental bleeding during or after delivery

Factors ↑fetomaternal hemorrhage

- Cesarean section delivery
- Manual removal of placenta
- Bleeding placenta previa
- Abruptio placenta
- Intrauterine manipulation
- ECV
- Tubal pregnancy
- Amniocentesis
- Multiple gestation

Con't

- Amount of fetomaternal hemorrhage necessary to cause immunization varies probably due to
 - immunogenic capacity of the Rh +ve RBCs
 - and immune responsiveness of the mother
- As small as **0.1ml** of Rh +ve RBCs shown to sensitize **3%** of Rh-Ve volunteers
 - Over all about 16 % of Rh-ve women will become isoimmunized by their 1st Rh-incompatible but ABO compatible pregnancy , if not treated with Rh immunoglobulin

Grand mother theory

- Theoretical possibility of Rh sensitization in the 1st pregnancy



An Rh-female fetus exposed to maternal Rh+cells in utero by maternal-fetal bleed then that fetus is already sensitized by her **1st** pregnancy

Prevention of D(Rh) isoimmunization

1. Prenatal testing

2. Administration of RheoGAM (Rh immunoglobulin prophylaxis)

Prenatal testing at 1st visit of each pregnancy every mother

-ABO blood group, Rh type & antibody screen for Rh-

Rh immunoglobulin prophylaxis

Rh-mother but not D-isoimmunized is candidate for prophylaxis

at 28wks, 35-36wks .

Rh-, unsensitized, admitted for delivery –Ab screen is routinely done
if → -ve & new born is Rh+ =Rheo GAM should be given.

Pregnancy induced hypertension

Objectives

- Diagnose Hypertensive disorder during pregnancy
- Manage and/ or refer Hypertensive disorder during pregnancy

Definition

- **Pregnancy related hypertension is defined as blood pressure $>140/90$ mm Hg in two occasions at least 6 hours apart, or a single BLOOD PRESSURE recording of $160/110$ mm Hg in a woman who was normotensive prior to 20 weeks of gestation**
- ***Proteinuria:***
 - – 0.3 g protein in a 24-hour urine specimen or
 - – 1+ on dipstick
- **Hypertensive disorders complicate 5 to 10 percent of all pregnancies**

Terminology and Classification

1. **Gestational hypertension:** refers to hypertension (usually mild) **without** proteinuria developing after the 20th week of pregnancy in a previously normotensive pregnant woman.
2. **Preeclampsia:** refers to the new onset of hypertension and proteinuria after 20 weeks of gestation in a previously normotensive woman.
3. **Chronic hypertension:** is defined as hypertension that antedates pregnancy; is present before the 20th week of pregnancy; or persists after 12 weeks postpartum

4. Superimposed preeclampsia: is diagnosed when a woman with chronic hypertension develops new onset proteinuria after 20 wks of gestation

- Women with chronic hypertension & preexisting proteinuria (before 20 wks) are considered preeclamptic if there is an exacerbation of blood pressure to the severe range (systolic 160 mmHg or diastolic 110 mm Hg) in the last half of pregnancy, especially if accompanied by symptoms or a sudden increase in proteinuria.

Eclampsia: describes the development of grand mal seizures or coma in a woman with preeclampsia

- Important causes of convulsion or coma like cerebral malaria, meningitis, hypoglycemia, previous seizure disorder, head injury or intracranial space occupying lesions have to be ruled out

Etiology & pathogenesis

- **Most theories concerning the etiology and pathogenesis showed gestational hypertensive disorders are more likely to develop in women who:**
 - **Are exposed to chorionic villi for the first time(immunologic incompatibility)**
 - **Are exposed to a superabundance of chorionic villi, as with twins**
 - **Have preexisting renal or cardiovascular disease**
 - **Are genetically predisposed to hypertension developing during pregnancy**
 - **Abnormal invasion of spiral arteries by trophoblastic tissues**

- **A fetus is not a requisite for preeclampsia**
- **Regardless of precipitating etiology, the cascade of events that leads to the preeclampsia syndrome is characterized by a host of abnormalities that result in vascular endothelial damage and subsequent vasospasm, transudation of plasma, and ischemic and thrombotic sequelae**

Diagnostic tests & Procedures

- **Laboratory evaluation that helps to characterize end organ involvement and determine disease severity includes:**
 - **Quantification of protein excretion**
 - **Serum creatinine concentration**
 - **Serum uric acid concentration**
 - **LFT(ALT, AST) , Biluribin level**
 - **Platelet count**
 - **Lactic acid dehydrogenase concentration (LDH)**

MANAGEMENT OF DIFFERENT STAGES OF PIH

1. Gestational hypertension

- Manage on outpatient basis
- Follow up for increasing BLOOD PRESSURE, urine (for proteinuria) & fetal condition wkly
- If blood pressure worsens, manage as mild pre-eclampsia
- Counsel the woman & her family about danger signals indicating severe preeclampsia or eclampsia.
- If all observations remain stable, allow to proceed with normal labor (but better not to post term)

2. Mild pre- eclampsia

- Gestational age less than 37 weeks
 - Outpatient twice wkly follow up is preferable as long as signs remain unchanged or normalize if it is convenient for the patient).
 - Monitor blood pressure, urine protein, & fetal condition, twice wkly.
 - Counsel about the danger signals (symptoms and signs of severe pre-eclampsia
 - Encourage the woman to eat a normal diet (salt restriction should be discouraged)
 - No medications (do not give anticonvulsants, anti hypertensives,

3. Severe pre-eclampsia

- **Clinical Features - includes any one or more of the following**
- **Diastolic blood pressure 2X 110mmhg after 20wks gestation & proteinuria of >3+ ($\geq 5\text{gm}$ in 24hrs)**
- **Any of these manifestations of multi organ involvement**
 - **Headache: -increasing frequency, unrelieved by regular analgesics**
 - **(frontal/occipital)**
 - **Clouding of vision (blurred vision/photophobia)**
 - **Oliguria (<400 ml urine in 24hrs) (followed by rapid wt gain)**
 - **Upper abdominal pain (epigastric or right upper quadrant pain)**
 - **Pulmonary edema (rapid shallow breathing, cyanosis, rales).**
 - **Fetal growth restriction**
 - **Abruptio placenta**

- **Lab changes include**
 - **Platelets < 1 00,000**
 - **Serum uric acid (↑ ed)**
 - **Serum creatinine (↑ ed)**
 - **Significantly altered liver function tests**
 - **Hyperbilirubinemia**
 - **Elevated liver enzymes (AL T, AST, LDH)**

- **Management of severe Pre-eclampsia**
 - *Prevent convulsion with magnesium sulfate or valium*
 - *Control hypertension*
 - *Delivery as soon as possible*

- **Anticonvulsant therapy (seizure prophylaxis) should be instituted**
 - In all pre-eclampsics during labor & continued for 24 hrs after delivery
 - In all severe pre-eclampsics during admission & continued during period of evaluation & observation.
 - Magnesium sulfate is the drug of choice for preventing & treating convulsions in severe pre-eclampsia & eclampsia.

Cont'd.

- **Anti - hypertensive drugs should be used if the diastolic blood pressure is ≥ 110 mmhg**
- **The goal is to keep the diastolic blood pressure b/n(90-100 mm hg)**
- **Some of the drugs that can be used are**
 - **Hydralazine**
 - **Nifedipine**
 - **Aldomet (metyl dopa)**

Cont'd.

- **Termination of pregnancy in the management of (pregnancy induced hypertension) PIH**
- **Delivery remains the only definitive treatment for PIH**
- **Timely delivery minimizes maternal & neonatal morbidity & mortality**
- **Expectant management is potentially harmful in the presence of severe PIH, fetal maturity or suspected fetal compromise**
- **Optimize maternal status before intervention to delivery (through resuscitation &stabilization)**

- **Delivery**

- **Delivery should occur within 24hrs of the onset of symptoms in severe pre-eclampsia, & within 12 hrs of the onset of convulsions in eclampsia.**
- **If vaginal delivery is not anticipated within this time limit, delivery should be by cesarean section.**

- **Route of delivery**

- Depends on **gestation age , fetal condition & presentation, cervical condition & maternal condition**
- Vaginal delivery is preferable to cesarean section for women with PE (even if with severe disease).
- It is desirable, if possible, to avoid the added stress of surgery & anesthesia, because of multiple physiologic abnormalities

Cont'd.

- **Post partum Care**
 - **Anticonvulsive therapy should be maintained for 24hrs to 48 hrs after delivery or the last convulsion, whichever occurs last**
 - **Continue anti-hypertensive therapy as long as the Diastolic BLOOD PRESSURE is ≥ 110 mmhg**

Premature Rupture of Membranes

Objectives

- At the end of the session student will be able to :
- Define premature rupture of membranes
- Diagnose Premature rupture of membranes
- Manage and/ or refer Premature rupture of membranes

Definition

- is rupture of membranes (ROM) before the onset of labor after 28 weeks of gestation
- Pre term PROM: - is rupture of membranes before 37 completed weeks of gestation
- Term PROM is rupture of membranes after 37 completed weeks of gestation
- Prolonged PROM is rupture of membranes for > 12 hrs

Possible of causes

- **The exact cause of rupture is not known, although many conditions are associated with PROM**
- **Maternal infection (eg, urinary tract infection, lower genital tract infection, sexually transmitted diseases)**
 - **Intrauterine infection**
 - **Cervical incompetency**
 - **Hydramnios**
 - **Decreased tensile strength of membranes**

Effects of PROM

- **PROM is an important cause of preterm labor, prolapse of the cord, placental abruption, and intrauterine infection.**
- **Chorioamnionitis is an important sequela of PROM and may precede endomyometritis or puerperal sepsis**

Clinical Findings

- **1. Symptoms**

Symptoms are the key to diagnosis;

- ❖ the patient usually reports a sudden gush of fluid or continued leakage.
- ❖ Additional symptoms that may be useful include the color and consistency of the fluid and the presence of vernix or meconium,
- ❖ reduced size of the uterus, and increased prominence of the fetus to palpation.

Cont'd.

2. Sterile Speculum Examination

- This examination is the key to differentiating PROM from vaginitis, and urinary incontinence.
- The examiner should look for the 3 hallmark confirmatory findings associated with PROM:
 1. Pooling—the collection of amniotic fluid in the posterior fornix.
 2. Nitrazine test—a sterile cotton-tipped swab should be used to collect fluid from the posterior fornix and apply it to Nitrazine paper. In the presence of amniotic fluid, the Nitrazine paper turns blue, demonstrating an alkaline pH (7.0–7.25).
 3. Ferning—Fluid from the posterior fornix is placed on a slide and allowed to air-dry. Amniotic fluid will form a fernlike pattern of crystallization

Cont'd.

- **If no free fluid is found, a dry pad should be placed under the patient's perineum and observed for leakage**
- **Ultrasound can be done**

Natural history of PROM

- The duration of PROM (latent period) is inversely related to the gestational age at the time of rupture of membranes
- <26wks GA: - 30-40% gain at least 1 week and 20% gain at least > 4weeks
- At term: - 80% go into labor within 24hrs of rupture of membrane

Management of PROM

- **General Management**
- Confirm the diagnosis
- Assess -maternal & fetal well being & check for signs of labor
- Determine gestation age from the last normal menstrual period, milestones of pregnancy or ultrasound
- Determine cervical status- by sterile speculum examination (avoid digital examination)

Cont'd.

- **Check for signs of intra-amniotic infection (chorioamnionitis) including**
 - • **Maternal fever & tachycardia**
 - • **Fetal tachycardia (FHB > 160 beats per minutes)**
 - • **Tender uterus**
 - • **offensive cervical discharge**
 - • **Leukocytosis (increase WBC count)**

Management

- **Depends on**
 - **Maternal condition-presence or absence of chorioamnionitis**
 - **Fetal condition**
 - **Gestational age**

Cont'd.

- If there are signs of uterine infection at any time during the pregnancy, Manage as chorioamnionitis:
- Start treatment with broad-spectrum, high dose. IV antibiotics
- Induce labor & expedite delivery, without any delay despite the GA; consider cesarean section only if abnormal labor occur.

Continue antibiotics post partum, at least for 24hrs after the mother becomes non febrile

Cont'd.

- **Management of Term PROM (>37wks GA)**
 - Wait for spontaneous onset of labor for 8hrs
 - If not start induction
 - Institute prophylactic anti-biotic when the duration of ROM > 12hrs

Cont'd.

- **Management of Pre term PROM**
 - At this gestational age , expectant management is preferred (in absence of chorioamnionitis), because of the significant risks associated with pre- maturity; & attempts should be made to prolong the latent period.

Cont'd.

- **Expectant management, when chosen at any gestational age, consists of the following Principles.**
 - **Avoid digital cervical (pelvic) examination**
 - **Advise bed-rest, to potentially enhance amniotic fluid re- accumulation & possible delay onset of labor.**
 - **Complete pelvic rest- to avoid infection**
 - **Use of steroids, as in pre term labor, to accelerate fetal lung maturity are indicated**

Cont'd.

- **Provide prophylactic antibiotics**
- **Advantages possibly include**
 - **Increased latency period**
 - **Decreased incidence of maternal & neonatal morbidity & mortality**

Cont'd.

- **implement- surveillance for infection when duration of PROM exceeds 12 hrs, which may include monitoring the following:**
 - Maternal pulse & temperature- every 4-6hrs
 - FHR every 4-6hrs
 - Uterine tenderness or irritability (or pain)
 - WBC count & differential- changes, daily

Cont'd.

- Indications for delivery (i.e. termination of expectant Management) include:
 1. Onset of labor
 2. Gestation age ≥ 37 wks
 3. Evidence for fetal distress
 4. Evidence for intra uterine infection.

ANTEPARTUM HAEMMORRH AGE

Objectives

- At the end of this session student will be able to :
- Define APH
- Identify cause of APH
- Diagnose APH
- Manage and/ or refer APH

ANTEPARTUM HAEMMORHAGE

- **APH- is bleeding from genital tract after the 28th weeks of gestation up to delivery of the fetus.**
- **The incidence is 2-3% of all pregnancies**
- **Causes of APH**
- **Placental causes**
 - **Abruptio placenta**
 - **Placenta praevia**
 - **Vasa praevia**

Cont'd...

- **Non placental causes**
 - Local causes (pathology of the cervix, vagina & vulva)
 - Heavy show
 - Uterine rupture
 - Bleeding disorder
- **Unclassified causes(50%)**

- **General measures**
- Take thorough hx and do pertinent p/E
- **DON'T DO PV AND PR EXAMINATION**
- Secure iv line and resuscitate if needed
- Determine blood group , Rh and hct
- Prepare blood products
- Try to know the source of the bleeding

- **Diagnosis**
- Ultrasound to localize the placenta and retro placental clot
- Speculum examination after bleeding stopped and after ruling out major degree of placenta praevia

To diagnose local causes

To collect specimen for cervical smear

PLACENTA PREVIA

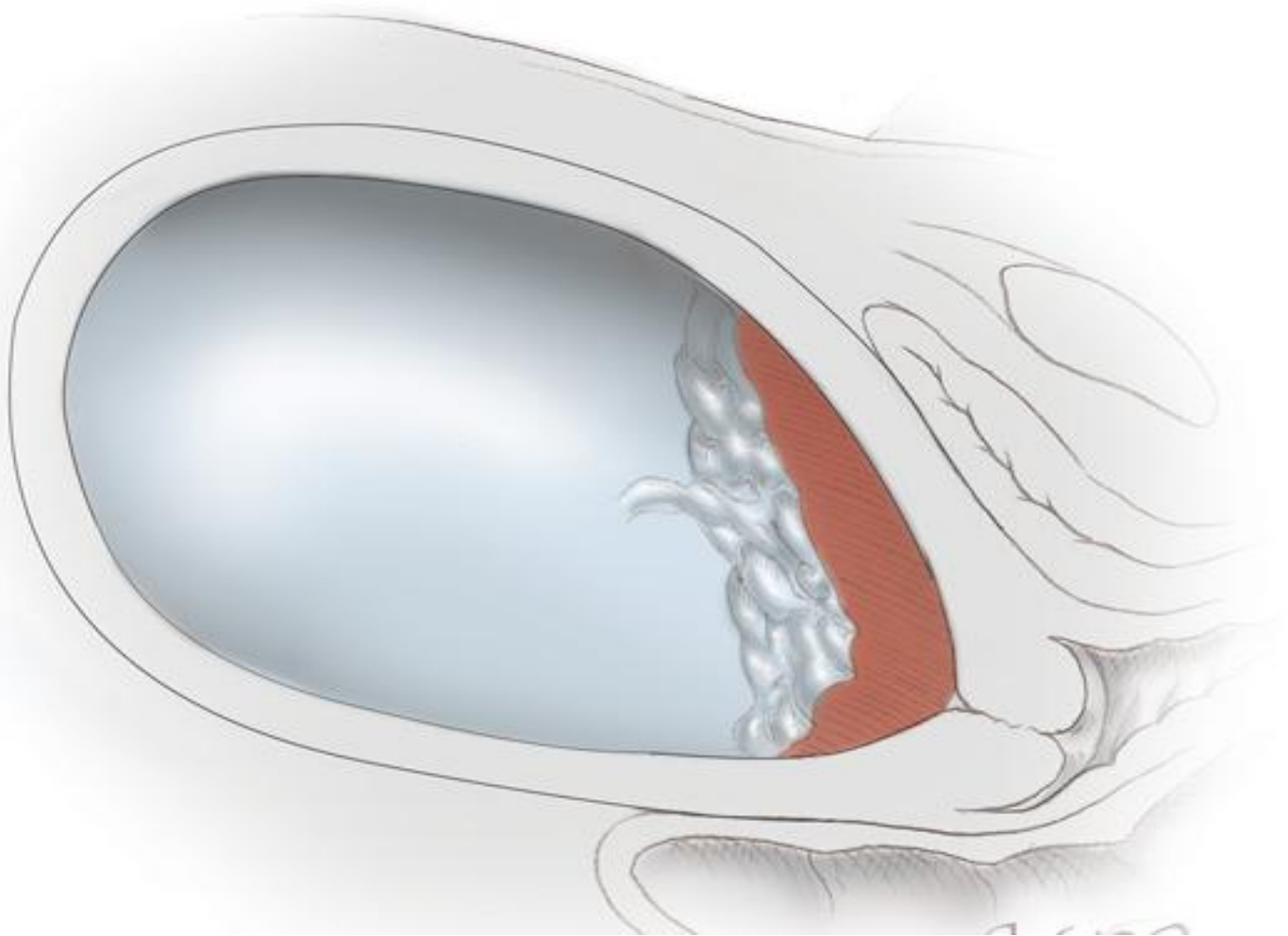
Definition: Placental implantation in the lower uterine segment within the zone of effacement & dilatation of cervix or before presenting part.

Predisposing factors.

- Scarred uterus
- Advanced age
- Multiparity
- Multiple pregnancy

- **Classification:** minor degree (type I) & major degree (Type II, III, IV)
- **Type I (Low lying)** - Placenta is implanted in lower segment but doesn't reach to internal os
- **Type II (marginal)** - (anterior & posterior) – Placenta reaches the internal os
- **Type III (partial)** – Placenta covers the internal os partially
- **Type IV (totalis)** – The placenta covers the whole internal os

Cont'd.



Cont'd...

- **Management**

General

- Admission to a unit with operative and blood transfusion facility
- Resuscitation as indicated
- **Avoid vaginal /rectal examination**
- Monitor fetal & maternal condition
- Hct, Blood group & Rh determination
- Prepare at least 2 units of cross –matched blood
- Anticipate PPH (Due to uterine atony or abnormally adherent placenta)

Cont'd...

- Decide on the final mode of management conservative versus aggressive
- **Delivery is indicated if**
 - 37 wks completed
 - Severe hemorrhage
 - IUFD
 - IUGR
 - Fetal distress
 - Major congenital anomalies

- **Expectant Management:** if bleeding is minimal and if it is a preterm pregnancy:
 - Bed rest in hospital
 - Maternal condition – vaginal bleeding & vital sign
 - Fetal condition – FHB & Kick chart daily, Biophysical profile once or twice per week, fetal growth
 - Betamethasone 12 mg in 2 doses for 28-34 weeks gestation
 - Termination after 37 completed weeks of gestation

- Mode of delivery: –

- Vaginal – type I or Type II a

- Caesarean section- If

- Major degree of placenta previa (Type II posterior, III & IV),

- Fetal distress,

- Severe uncontrolled hemorrhage.

ABRUPTIO PLACENTAE

Definition: premature Separation of the whole or part of the placenta from the normal implantation site.

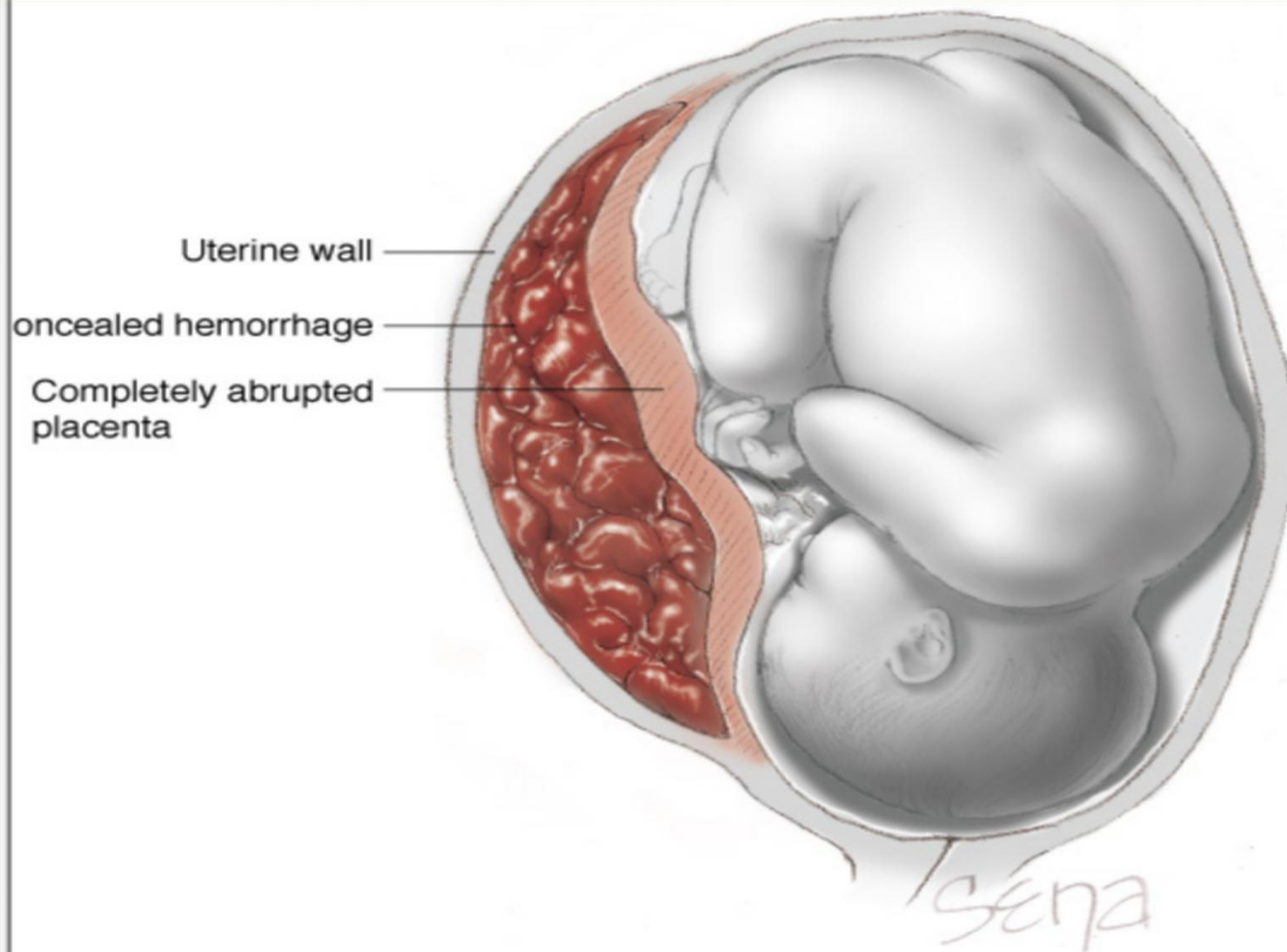
- **Predisposing factors:** -
- **Hypertension;** due to spasm and degenerative changes in the decidual arterioles.
 - Advanced age
 - Multiparity;
 - Multiple pregnancy;
 - Myoma
 - polyhydramnios;
 - Low socio economic
 - Trauma
 - Smoking; Cocaine use

- **Types**

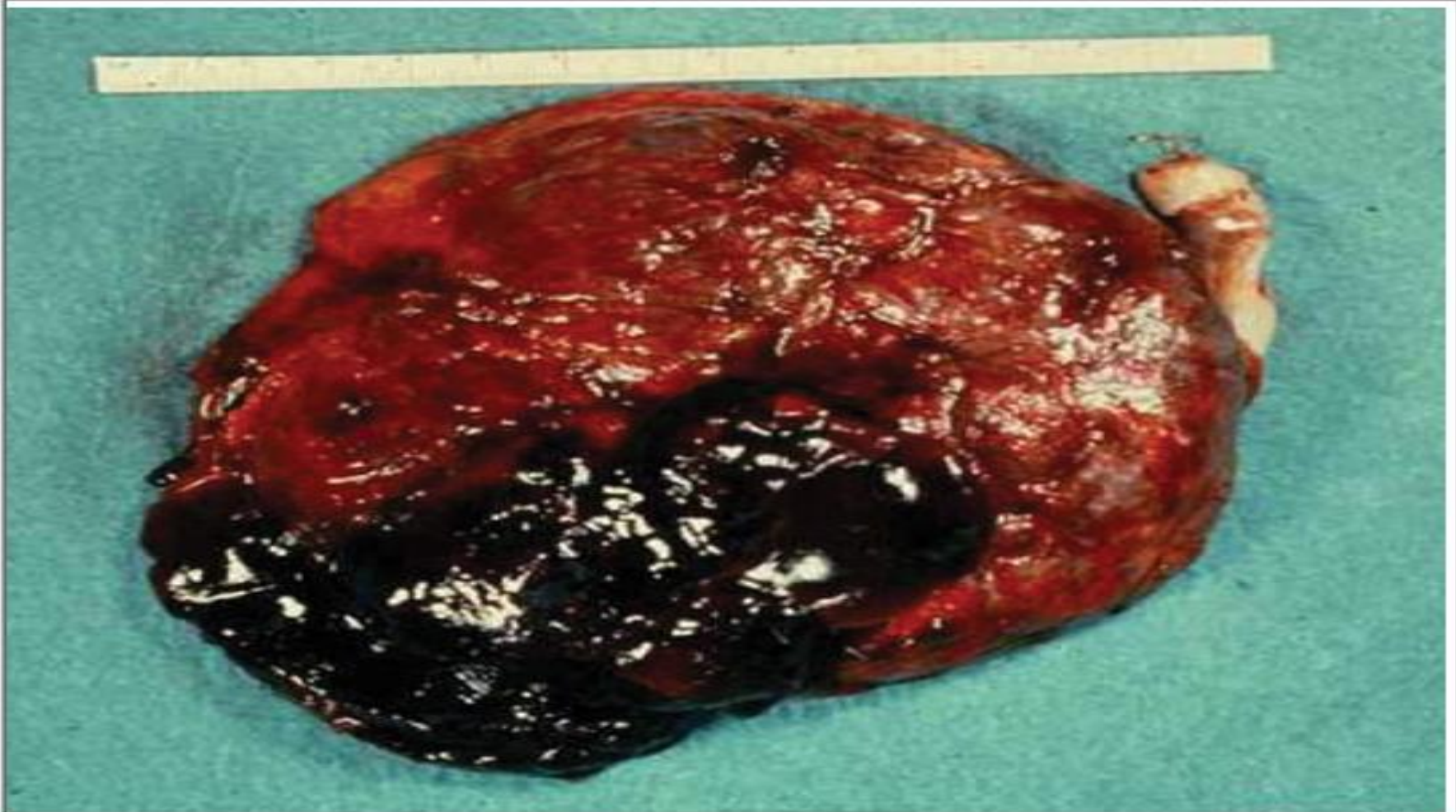
- ✓ **concealed –accounts for 20% of cases**
- ✓ **revealed _accounts for 80% of cases**

- **Patients may have abdominal pain, uterine contractions, uterine tenderness, and a non reassuring fetal heart rate tracing**
- **The diagnosis of placental abruption is primarily clinical, but u/s, laboratory, or pathologic findings can be used to support the clinical diagnosis.**

- **Management**
- depend upon the **severity of the abruption**, the **gestational age**, and **maternal** and **fetal status**
- **Live fetus at or near term** — The fetus should be delivered expeditiously by the quickest, safest method if it is alive, the pregnancy is at least 34 weeks of gestation
- **Vaginal delivery is reasonable if the maternal status is stable and the fetal heart tracing is reassuring.**



Source: Cunningham FG, Leveno KJ, Bloom SL, Hauth JC, Rouse DJ, Spong CY: *Williams Obstetrics, 23rd Edition*. <http://www.accessmedicine.com>



Source: Cunningham FG, Leveno KJ, Bloom SL, Hauth JC, Rouse DJ, Spong CY:
Williams Obstetrics, 23rd Edition: <http://www.accessmedicine.com>
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Partial placental abruption with adhered clot.

PRESENTATION AND FREQUENCY

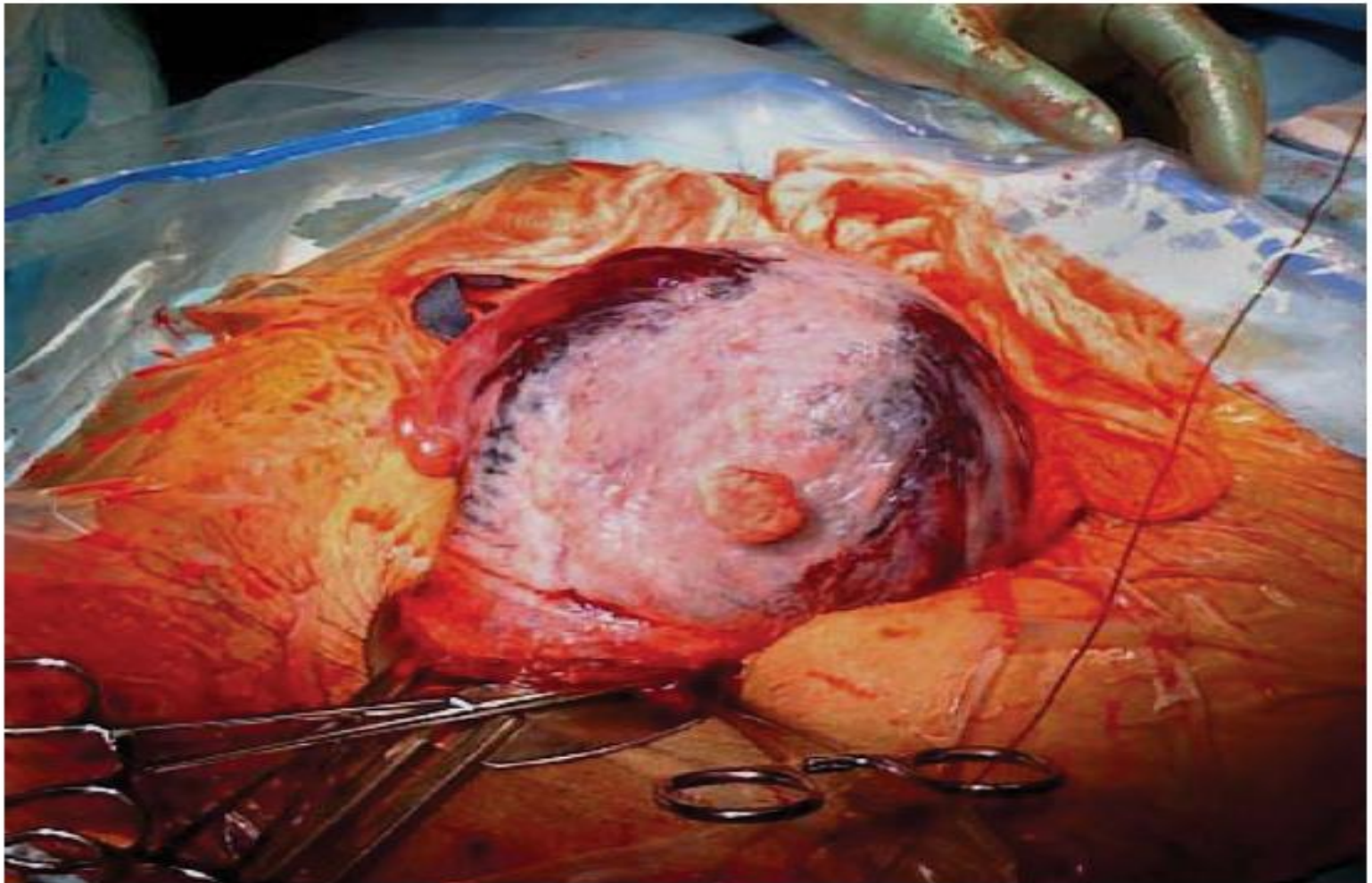
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- **Live fetus remote from term** — Delaying delivery of pregnancies under 34 weeks of gestation is reasonable when tests of fetal well-being are reassuring and there is no evidence of maternal coagulopathy, hypotension, or ongoing major blood loss
- Glucocorticoids to promote fetal lung maturation
- Fetal assessment with biophysical profile is performed at least weekly

Cont'd...

- **Fetal demise at any GA — When fetal death has occurred, the mode of delivery should be that which minimizes the risk of maternal morbidity or mortality**
- **Vaginal delivery is preferable unless urgent delivery is needed to enable stabilization of the mother**

- **Complications after severe abruption**
- ✓ **Couvlaire uterus** – pregnant uterus in which the placenta has detached prematurely with extravasation of blood into the uterine musculature
 - ✓ oxytocin infusion
 - ✓ Hysterectomy reserved for cases of uterine atony & hemorrhage unresponsive to uterotonic agents.
- ✓ **ARF**
- ✓ **DIC**



Vasa praevia

- refers to fetal vessels that traverse the membranes located in the lower uterine segment in advance of the fetal presenting part.
- **Diagnosis:** Fetal distress with no maternal vital sign derangement and most commonly occurs after rupture of membrane

Cont'd...

- **Apt** (alkaline Phosphatase test) test for suspected fetal hemorrhage.
- **MANAGEMENT:** Emergency c/s for viable live fetus
- Determine hemoglobin of the neonate after birth

Cont'd...

- **Local causes-** Treat primary cause
- **APH of unknown cause-**
- **Induction of labor after 37 completed weeks of gestation**
- **Cesarean section indicated if severe bleeding or other obstetric indications.**

PRETERM LABOR

Objectives

- At the end of this session student will be able to :
- Define preterm labor
- Identify cause of preterm labor
- Diagnose preterm labor
- Manage and/ or refer preterm labor

Subtypes of PTB are variably defined

A. By gestational age :

1. Moderate preterm: 32 to <34 weeks(32-34)
2. Late preterm: 34 ^{0/7ths} to 36 ^{6/7ths} weeks (34-37)
3. Very preterm: 28 to <32 weeks(28-32)
4. Extremely preterm:<28 weeks(20-28), (Ethiopia : <28 weeks→ abortion)

B. By birth weight :

1. Low birth weight (LBW): <2500&≥ 1500 grams
2. Very low birth weight (VLBW): <1500& ≥ 1000 grams
3. Extremely low birth weight (ELBW): <1000 grams

Preterm delivery (PTD)---

- Is the major obstetrical & neonatal problem of the developed world
- *is* the leading cause of perinatal mortality including neonatal mortality & morbidity
- The second (after pneumonia) most common cause of under 5 mortality
- **It causes**
 - a. *75% of all fetal & neonatal deaths that are not due to congenital anomalies*
 - b. long term handicap (10% of survivors)
 - c. *50% of all major neurologic handicaps*

Risk factors of PTL

- A. Demographic characteristics
- B. Behavioral factors, &
- C. Aspects of obstetric history
 - Obstetric complications in current or previous pregnancy
 - PIH
 - Placental abnormalities (APH) or insufficiencies
 - PPROM
 - Polyhydramnios or multiple pregnancy
 - Threatened abortion
 - Previous PTB → ↑ 3x (15-40%)

Risk factors of PTL----

➤ Life style factors

- Cigarette smoking ,alcoholism, illicit drug use,
- Inadequate weight gain/overweight
- Young/advanced maternal age
- Low socioeconomic status,
- psychological factors,...

Risk factors of PTL----

- Genetic factors
- Genital tract anomalies(uterus&cervix)
- Maternal medical cxns
 - Pulm-/systemic HTN
 - renal /cardiac diseases
 - Infections
 - Severe anemia
- Short (<3month)Interval b/n pregnancy & PTB

Obstetrical syndromes share the following features:

- 1. Multiple etiologies***
- 2. Chronicity***
- 3. Fetal involvement***
- 4. Clinical manifestations that are adaptive***
- 5. Variable susceptibility due to gene-environment interactions***

Diagnosis of PTL----

- a. Cervical dilatation >1 cm(≥ 2 cm)**
- b. Cervical effacement of ≥ 80 %.**
- Symptoms (pelvic pressure, menstrual-like cramps, watery vaginal discharge, & lower back pain) \rightarrow nonspecific & ignored.**

Management of PTL

- PTL is among the most controversial issues in perinatal medicine
 1. Bed Rest: no evidence supporting or refuting the benefit of either bed rest or hospitalization in women threatening preterm delivery.
 2. Hydration & Sedation
 - Rehydration with 500 mL of crystalloid over 30 minutes & sedation with 8 to 12 mg of IM morphine sulfate → similar outcomes of bed rest only
 3. Tocolysis

Recommended Management of PTL

- 1. Confirmation of preterm labor*
- 2. Expectant management for GA < 34 weeks with no maternal or fetal indications for delivery*
- 3. Corticosteroids if GA < 34 weeks for enhancement of fetal lung maturation*
- 4. maternal magnesium sulfate infusion for 12 to 24 hours has fetal neuroprotection*
- 5. Tocolytic drugs if GA < 34 weeks if no advanced labor*
- 6. If GA \geq 34 weeks , monitor labor progression & fetal well-being*
- 7. For active labor, an antimicrobial is given for prevention of neonatal group B streptococcal infection*

Recommended Screening Strategies to Prevent PTL

1. No current data support use of home uterine-activity monitoring or B. vaginosis screening
2. Screening for risk of preterm labor except historical risk factors is not beneficial in the general obstetrical population
3. Sonography to determine cervical length &/or fetal fibronectin level measurement

Prognosis

- Higher perinatal morbidity & mortality
- With ICU, the survival rate of baby weighing b/n 1000-1500g is > 90%
- With surfactant survival rate of infants born at 26wks is~ 80%
- The fetal survival rate is within 1% of the survival rate at 37 weeks if GA > 34 weeks or EFW > 2500 g

INTRA UTERINE GROWTH RESTRICTION (IUGR)

Objectives

- At the end of this session student will be able to :
- Define IUGR
- Identify cause of IUGR
- Diagnose IUGR
- Manage and/ or refer IUGR

Normal Fetal Growth

- ❑ Sequential tissue organ growth, differentiation & maturation determined
 - ✓ maternal provision of substrate,
 - ✓ placental transfer of these substrates
 - ✓ **fetal growth potential governed by the genome.**
- ❑ cell growth divided into three consecutive phases.
 - ✓ hyperplasia during first 16 weeks - rapid in cell number.
 - ✓ cellular hyperplasia and hypertrophy up to 32 weeks
 - ✓ cellular hypertrophy after 32 weeks --fat &glycogen deposition
- ❑ corresponding fetal growth rates during three phases
 - ✓ 5 g/day at 15 weeks,
 - ✓ 15 to 20 g/day at 24 weeks, and
 - ✓ 30 to 35 g/day at 34 weeks

Factors in fetal growth

- ❑ Many factors implicated in the process of fetal growth
- ❑ precise cellular and molecular mechanisms are not well understood.
- ❑ Early fetal life major determinant of growth is the fetal genome
- ❑ later in pregnancy environmental, nutritional, and hormonal influences become increasingly important

Con't

- ❑ Fetal growth is also dependent on an adequate supply of nutrients
- ❑ Both excessive and diminished maternal glucose availability to the fetus affect fetal growth.
- ❑ Excessive glycemia produces macrosomia, whereas diminished glucose levels have been associated with fetal growth restriction.

Definition

- ❑ A number of definitions of IUGR have been proposed based on percentile, standard deviation (SD), or growth rate.
- ❑ EFW < 10th percentile as determined u/s = SGA
 - ❑ most commonly used clinical definition
- ❑ constitutionally small, growth restricted & small and growth restricted are included
- ❑ If determinants of birth weight (maternal ethnicity, parity, maternal weight, and height) are considered, up to 50%-70% of fetuses will be constitutionally small & not at high risk

What are the causes of IUGR?

- Maternal medical conditions
- Chromosomal anomalies & aneuploidy
- Genetic & Structural anomalies
- Exposure to drugs & toxins
- 1ry placental disease
- Extremes of maternal age
- Low socioeconomic status
- Infections
- Multiple gestation

Which maternal medical conditions result in IUGR?

- Hypertension
- DM with vascular involvement
- Anemia
- Sickle cell disease
- Renal disease
- Malnutrition
- Inflammatory bowel disease
- Intestinal parasites
- Cyanotic pulmonary disease

How does the placenta play a role in the development of IUGR?

- Chronic partial abruption
- Placental infarcts
- Chorioangioma
- Single umbilical artery
- Twin to twin transfusion Syndrome

What infections result in IUGR?

5-10% of IUGR

Congenital infections:

- CMV
- Rubella
- Toxoplasmosis
- Malaria
- Syphilis
- Listeriosis

Which drugs can result in IUGR?

- Alcohol
- Cigarette smoking 3-4X
- Methotrexate
- Anticonvulsants
- Warfarin
- Antihypertensives / β -blockers

What are the genetic disorders that can result in IUGR?

15% of IUGR

- *Down's syndrome T21*
- *Trisomy 13,18*
- *Turner syndrome*
- *Neural tube defects*
- *Abdominal wall defects*
- *Duodenal atresia*
- *Renal agenesis/ Potter's S*

Features suspicious of trisomy

- *Symmetric IUGR*
- *AFV/ Doppler ➡N*
- *Structural abnormalities*
- *Maternal age*
- *Nuchal translucency*
- *Biochemical screening results*

What are the types of IUGR?

1-Symmetric –20%

- Proportionate decrease in many organ weights including the brain
- Deprivation occurs early
- The fetus is more likely to have an endogenous defect that preclude N development
- U/S biometry ➡ All measurements BPD, FL, AC ➡ ↓

Types of IUGR

2-Asymmetric IUGR—80%

- Relative sparing of the brain
- Deprivation occurs in the later half of pregnancy
- The infant is more likely to be N but small in size due to intrauterine deprivation
- U/S biometry ➡ BPD, FL ➡ N, AC ➡ ↓

Why IUGR often associated with olighydramnios?

- ↓ blood flow to the lungs → ↓ pulmonary contribution to amniotic fluid volume
- ↓ blood flow to the kidneys → ↓ GFR
→ ↓ urine output
- It is present in 80-90% of IUGR fetuses

How to evaluate a case of IUGR?

1-History:

- Current preg
 - ➡ LMP, preg test, quickening
 - ➡ APH, abruptio placentae, & fetal movements
- Previous obstetric Hx particularly looking for IUGR, & adverse outcome
- Medical Hx:
- Hx of recent viral illness
- Drug Hx

EXAMINATION

- Sensitivity ➡ 46-86% in detecting IUGR
- A difference of more than 3cm requires fetal assessment

U/S

- Fetal biometry ➡ for dating then serial measurements
- Anomaly scan
- AF index
- Repeat tests every 1-2 wks

Invasive fetal testing

- Amniocentesis or placental biopsy/ fetal blood sampling ➡ for karyotyping if aneuploidy is suspected
➡ for viral studies if infections suspected
- Carries the risks of ➡ infection, PROM, Preterm labor

Retrospective tests

- Maternal blood tests for ➡ CMV, Rubella, Toxo
➡ Metabolic disorders
- Placenta should be sent for HP
- Postmortem examination

The constitutionally small fetus

- A fetus growing parallel to the lower centiles through out preg
- Anatomically N
- AFV ➡ N
- Slim petite women

Treatment

- Stop smoking / alcohol
- Weekly visits ➡ attention to : FM, uterine growth, maternal wt ,AFV
- U/S every 2-4 wks
- Contraction stress test
- Delivery ➡ 37 wks or earlier if there is fetal compromise
- Glucocorticoids if planing delivery before 34 wks
- Close monitoring in labor/ continuous monitoring
- CS may be necessary

Intra Uterine Fetal Death(IUFD)

Objectives

- At the end of this session student will be able to :
- Define IUFD
- Identify cause of IUFD
- Diagnose IUFD
- Manage and/ or refer IUFD

**Definition: dead fetuses or newborns weighing
> 1000gm**

Or > 28 wks gestation

4.5/ 1000 total births

Diagnosis

Absence of uterine growth

Loss of fetal movement

Absence of fetal heart

Disappearance of the signs & symptoms of pregnancy

X-ray ➡ Spalding sign

Robert's sign

U/S ➡ 100% accurate Dx

Causes OF IUFD

Fetal causes 25-40%

- Chromosomal anomalies
- Birth defects
- Infections

Placental 25-35%

- Abruptio
- Cord accidents
- Placental insufficiency
- Intrapartum asphyxia
- Twin to twin transfusion S

Maternal 5-10%

- DM
- HPT
- Trauma
- Abnormal labor
- Sepsis
- Acidosis/ Hypoxia
- Uterine rupture
- Postterm pregnancy
- Cyanotic heart disease
- Severe anemia

Unexplained 25-35%

Approaches to fetal death to know the cause

- **Maternal medical hx**
- **Current & previous obstetric hx**

2-Evaluation of still born infants

Infant description

- Malformation
- Skin staining
- Degree of maceration
- Color-pale ,plethoric

Umbilical cord

- Prolapse
- Entanglement-neck, arms, ,legs
- Hematoma or stricture
- Number of vessels
- Length

Amniotic fluid

- Color-meconium,
- Volume

Placenta

- Weight
- Adherent clots
- Structural abnormality
- Velamentous insertion
- Edema/ hydropic change

IUFD complications

- Hypofibrinogenemia ➡ 4-5 wks after IUFD
- Coagulation studies must be started 2 wks after IUFD
- Delivery by 4 wks or if fibrinogen ↓ < 200mg/ml

Psychological aspect & counseling

- A traumatic event
- Post-partum depression
- Anxiety
- Psychotherapy
- Recurrence 0-8% depending on the cause of IUFD
- Methods of ablactation

Hyperemesis gravidarum

Objectives

- At the end of this session student will be able to :
- Define Hyperemesis gravidum
- Identify cause of Hyperemesis gravidum
- Diagnose Hyperemesis gravidum
- Manage and/ or refer Hyperemesis gravidum

Definition

- Extreme form of nausea & vomiting in the 1st TM of pregnancy
- The adverse effects of severe vomiting are:
 - DHN,
 - Metabolic acidosis(from starvation),or
 - Alkalosis(from loss of HCl),
 - Electrolyte imbalance(hypokalemia) &
 - Wt loss

Incidence

- Decreasing due to:
 - Better application of FP w/c reduces unplanned Px.
 - Early visit to ANC
 - Potent antihistamic, antiemetic drugs

Etiology

- Obscure but 2 facts are 2 known facts:
 - It is mostly limited to 1st TM (peak in 9th & 10th wk)
 - It is more common in 1st Px, with a tendency to recur again in subsequent Pxs
 - Family Hx-genetic
 - More common in molar Px & multiple Px
 - More common in unplanned Px

Theories

1.Hormonal:

- Excess hCG or higher biologic activity of hCG
- This is proved by the frequency of vomiting at the peak level of hCG & also the increased association with molar Px or multiple Px when z hCG titer is very raised.
- High level of estrogen
- Excessive progesterone
- Other hormones

Con't...

2.Psychogenic

3.dietetic def:- low CHO,def of vitB6,vit B1 & proteins

4.Allergy or immunologic basis

5.Decreased gastric motility;-causes nausea

Metabolic, biochemical,& circulatory changes

- **Metabolic**-glycogen depletion due to poor intake
- **Biochemical**-loss of H₂O & salt in vomitus cause fall in plasma Na,K,Cl
- Urinary Cl fall significantly
- Hepatic dysfunction results in acidosis & ketosis with rise in blood urea & uric acid;hypoglycemia;hyponatremia
- **Circulatory**:hemoconcentration leads to rise in Hgb % tage,RBC count & Hct value
- Reduction in ECF occurs

Risk factors

- Younger age
- Low pregnancy body mass
- Female fetus
- Hx of motion sickness & migraine
- Smoking & obesity are associated with decreased risk of hyperemesis

Clinical course

- The onset is insidious
- ***early:***
 - Vomiting occurs throughout the day
 - Normal day to day activities are curtailed
 - There is no evidence of DHN or starvation

Con't...

- **Late(moderate to severe):**

➤ Sxs:

- Vomiting increases with retching
- Urine quantity is diminished even to the level of oliguria
- Epigastric pain, constipation may occur
- Complication may appear if not treated

Con't...

- Signs

- Features of DHN & ketoacidosis:

- Dry coated tongue
 - Sunken eyes
 - Aceton smell in breath
 - Tachycardia
 - Hypotension
 - Rise in Temp may be noted
 - Jaundice is a late feature

Ddx

medical	surgical	gynecological
Intestinal infestation UTI Hepatitis DKA uremia	Appendicitis Peptic ulcer Intestinal obstruction cholecystitis	Twisted ovarian tumor Red degeneration of fibroid

Investigations

- ***U/A:***
 - small, dark color, high specific gravity, acetone, diminished or even absent Cl
- ***Biochemical & circular change:***
- ***Ophthalmologic exam***
 - Retinal hemorrhage & detachment is unfavorable signs
- ***ECG***
 - Abnormal serum potassium level
- ***Transient Lab. abnormalities occur:***
 - Suppressed TSH or elevated free thyroxine
 - Elevated liver enzymes, bilirubin, amylase, lipase &
 - Altered electrolytes (loss of Na, K, & Cl)

Diagnosis

- The Px is to be confirmed first
- ***U/S:***
 - to confirm Px
 - To exclude other obstetrics(hydatid mole, multiple Px, gynecologic, surgical or medical causes of vomiting)

Rx of HEG

Mx principles

- ✓ To control vomiting
- ✓ To correct fluid & electrolytes
- ✓ To correct metabolic disturbance (acidosis & alkalosis)
- ✓ To prevent serious complications

☐ *If It is primarily symptomatic*

- Frequent, small, dry meals that favor protein over carbohydrates & liquids over solids

Con't...

- **Fluid**

- Withheld oral fluids for at least 24hr
- Total amount of fluid/24 hr approximates 3L of w/c ½ is 5% dextrose & ½ is R/L solution
- Extra amount of 5% dextrose equal to the amount of vomitus & urine in 24 hr, is to be added

- **Drugs**

- antiemetic drugs:promethazine 25mg or prochlorperazin 5mg ,metoclopramide
- Hydrocortisone 100mg Iv in the drip in case of hypotension
- Nutritional support: vitamin B1,B6,C & B12

HEG Chart should be used daily to know the progress

—includes:

- V/S twice daily
- Intake-out put
- Urine for acetone, protein, bile,
- Blood biochemistry
- ECG

—Termination of pregnancy

- *Only in case of intractable HEG*

Malaria

Objectives

- At the end of this session the student will be able to :
- Identify effect of Malaria on Pregnancy
- Diagnose Malaria
- Manage and/ or refer Malaria

Effect of pregnancy on malaria

- ➔ **Break down of acquired malarial immunity**
 - immunosuppression of pregnancy
 - increased protein demand
 - parasite sequestration in placenta
- **Break down most marked in 1st pregnancy**
- **Febrile attacks more in 3rd trimester.**
- **The effect persists for 60 days postpartum.**

Contd.

- **Effect of immunity break down:-**
 - **Increase in frequency & severity of malarial attacks.**
 - **4-12x ↑ed parasitemia**
 - **10x higher average intensity of parasitemia.**

Effect of malaria on pregnant

1. Anemia:-

- ➔ established b/n 20th-28th wk
- ➔ especially partially immune (ST) primigravid
- ➔ PAR= 3 - 15% (all parity, severe anemia)

*Malawi =14.2%(p-I=34.2%), Kenya=12.7%(p-I=32%)

*Eth:-overall(<11g/dl) = 43%(ST) vs 14.7%(UT).

- “ (<8 “) = 7.2 “ “ 1.5 “ .

-no sig. different by gravidity.

Contd.

- 2. 2-10x higher mortality than non-pregnant, 13.2 Vs 6.47%.**
- 3. Increased risk of associated infections; particularly pneumonia and UTI.**

Effect of malaria on labor

- **3rd trimester acute attack can precipitate labor.**
- **Fetal distress is common but frequently not diagnosed .**
- **Increased instrumental delivery.**
- **PPH is badly tolerated.**
- **Cardiac failure may develop immediately postpartum.**

Effect of malaria on puerperium

- **A cause of puerperal pyrexia.**
- **In endemic areas it masks other causes of puerperal pyrexia and delays initiation of appropriate treatment.**
- **Does not interfere with lactation unless gravely ill.**

Effect of malaria on outcome of pregnancy

A. Effect of pyrexia → may activate uterus

→ abortion (1st trimester)

→ premature labor, PAR = 8-36%

B. Effect of placental parasitization:-

→ lower mean birth wt, more in primies.

→ ↑ LBW rate [PAR= 8-14%]

***Eth=15.5%(ST) vs 6.6%(UT)**

=20, 16.3 & 10.5% in G-1,2 & >2

Contd.

→ placental insufficiency → IUGR (PAR =13-70%) & ↑perinatal loss.

3. Effect of transplacental infection:-

→ congenital malaria= If ≤ 7 days or > 7 \hat{S} exposure.

► very rare in ST, more common in UT

→ rarely cause IUFD & ENND.

→ umbilical cord parasitemia in Ethiopian study = 1.6%(ST) & 1.1%(UT)

Effect of Malaria on Pregnancy in Stable Transmission Areas

Plasmodium falciparum
malaria

Asymptomatic Infection (**often overlooked**)

Placental
Altered Placental
Sequestration
Integrity

Reduced Nutrient and Oxygen
Transport

Anemia
esp.
primi

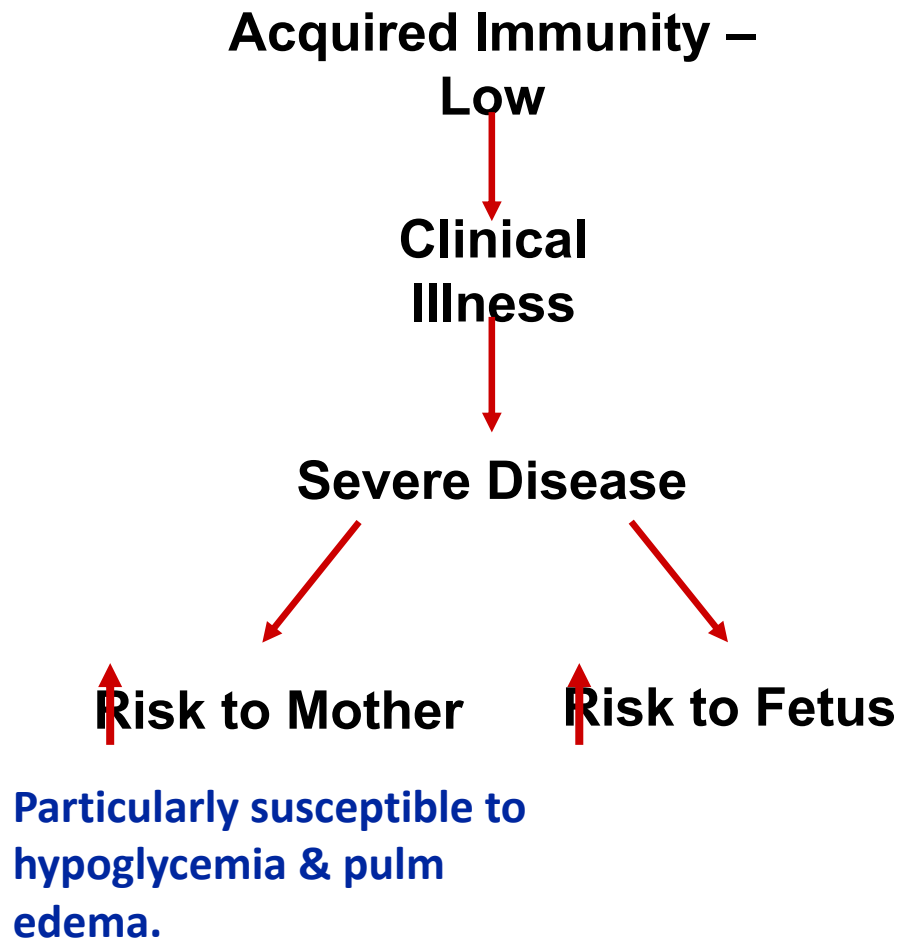
Low Birth Weight
(IUGR)



**Risk of Newborn
Mortality**

**NB: -SEVERITY SYMPTOMS ARE LIKELY FROM ECLAMPSIA,
MENINGITIS....**

Effect of Malaria on **Pregnancy** in Unstable Transmission Areas



Effects on the Pregnant Woman

Effects	Primigravidae in Stable malaria areas	All parities in Unstable malaria areas
• High fever	+	+++
• Placental infection	+++ ++	+ ++
• Puerperal sepsis		
• Complicated malaria	+++	+++
– Severe anemia	-	++
– Cerebral malaria	-	++
– Hypoglycemia	-	++
– Pulmonary edema	+	++
– Acute renal failure		

(+++ =Very Common, ++ =Common, + =Infrequent, -- =Rare)

Effects on the Fetus and Newborn

Effects	Primigravida e in Stable malaria areas	All parities in Unstable malaria areas
• Low birth weight	+++	+
– IUGR	+	++
– Prematurity		
• Abortion	-	++
• Stillbirth	-	++
• Congenital malaria	-	+
	?	+
• Fetal anemia	+	++
• Infant mortality		

(+++=Very Common, ++=Common, +=Infrequent, -- =Rare)

SEVERE FALCIPARUM MALARIA

Diagnostic manifestations

- confusion, or drowsy with prostration.
- Cerebral malaria.
- Severe normocytic anemia.
- Hypoglycemia.
- Metabolic acidosis with resp. distress.
- Fluid & e' disturbance.
- Acute renal failure.
- ARDS.
- Circulatory collapse, Shock, septicemia ("algid malaria").
- Abnormal bleeding.
- Jaundice.
- Haemoglobinuria.
- High fever (>39*c).
- hyperparasitemia (density >5%, & schizontaemia).

SPECIAL CLINICAL FEATURES & Mx OF SEVERE MALARIA IN PREGNANCY

A. Severe malaria:-

➔ manifestations like others

➔ management:-

- Make a rapid clinical assessment.**
- Admit to ICU if possible.**
- If no BF ready, take smear & start treatment empirically.**
- Determine RBS, if not give glucose.**

Contd.

- **Give antimalarial IV, or IM / supp.**
- **Good nursing care.**
- **Look for other possible cause and other complicating or ass. infections.**
- **Avoid drugs as aspirin, steroids...**
- **Monitor therapeutic response, hct, RFT,ele.**
- **In ST areas with severe anemia → full malarial treatment despite –ve BF & no other features.**

Contd.

B. Hypoglycemia:-

➔-commonly asymptomatic

-fetal bradycardia & other signs of distr

**-if on quinine ➔abn. behavior, sweating
& sudden loss of consciousness.**

Rx:-50ml, 50% dextrose/50ml iv-fluid/5min.

-follow with continuous 5 or 10%

dextrose b/c is recurrent in pregnancy

Contd.

C. Pulmonary edema:-

→ -may develop suddenly or immediately after delivery.

-a grave complication, mortality >80%.

→ Mx

D. Anemia:- transfuse if Hct<20%

E. Others = ARF, DIC, hyperparasitemia, fluid & elec. Disturbance, circulatory collapse,...

Poor prognostic indicators in severe malaria

- **Clinical indicators**

- Deep coma
- Convulsions
- Absent corneal reflex
- Decerebrate /
decorticate /
opisthotonus
- Organ dysfunctions

- **Respiratory distress**
- **Circulatory collapse**
- **Papilloedema and/or
retinal oedema.**

Contd.

Laboratory indicators

- **Hyperparasitemia**
- **Peripheral schizontemia**
- **PMN leukocytosis ($>12000/\mu\text{l}$)**
- **Mature pigmented parasites ($>20\%$)**
- **Hct $<15\%$**
- **RBS $<40\text{mg/dl}$**
- **BUN $>60\text{mg/dl}$**
- **Creatinine $>3.0\text{mg/dl}$**
- **Very high plasma TNF**
- **Aminotransferases elevated $>3\text{x}$**
- **Venous & CSF lactic acid $>5\text{mmol/l}$**
- **Low CSF glucose**

ECLAMPSIA Vs CEREBRAL MALARIA

- **Diff. to distinguish b/c of:-**
 - share features= fever, coma, seizure, albuminuria.
 - +ve BF can co-exist.
 - cerebral malaria can occur with –ve BF.
 - eclampsia can occur with lower BP.
- **Advised to give antimalarials to what are probably eclamptics.**

Anti-malarial drugs

- **Choice depends on local sensitivity.**
- **Can be used for prophylaxis, therapy, or prevention of transmission.**
- **Based on phase of action can be:-**
 - 1. Tissue schizonticides- on liver...**
e.g.-proguanil, pyrimethamine, primaquine
 - 2. Gametocidal:- primaquine, chloroquine...**

Contd.

**3. Hypnozoitocidal- in vivax & ovale
e.g.- premaquine**

**4. sporontocidal- inhibit oocyst devt. in
mosquito → inhibit transmission.**

**5. Blood schizontocidal → chloroquine,
quinine, artemisinin, quinidine, ...**

Contd.

- **QUININE:-**
 - an arylaminoalcohol
 - drug of choice in many countries.
 - ➔intercalates in to DNA➔...
 - safe in pregnancy in conventional doses.
 - Overdose➔blindness, coma, convulsion, abortion, ...
 - commonest s/e = hypoglycemia

Contd.

- **CHLOROQUINE:-**
 - inhibit DNA & RNA polymerase.
 - safe in pregnancy in sensitive malaria.
 - more rapidly effective than quinine.
 - resistant strains of vivax reported.
- **FANCIDAR(SP):- (500mg + 25mg)**
 - pregnancy risk factor C.
 - avoid in 1st & late 3rd trimesters.

Contd.

- **PROGUANIL:-**
 - schizonticidal & sporontocidal effects.
 - safe in pregnancy if supplemented with folate
- **A RTHEMETER = inadequate safety data.**
- **ARTEMISININ = no adequate data.**
- **TTC, DOXYCYCLINE, PREMAQUINE & HALOFANTRINE = are contraindicated in pregnancy & lactation.**

PREVENTION OF MALARIA IN PREGNANCY

- Is → a major public health challenge,
→ a priority for RBM,
→ an integral part of 'making pregnancy safer initiative'.
- WHO's a three pronged approach:-
 - A. Effective case Mx of malaria illness.
→ prompt Rx of malaria & consequences.

Contd.

B. Use of insecticide- treated nets [ITN]:-

- through repellent & knocking down effect of the insecticide.**
- Deltamethrin & permethrin = currently in use in our country.**
- in Eth → net ownership = 23.5% at ANC**
 - majority are untreated**

Impact of ITNs on Maternal and Newborn Health

Among Gravidae 1-4, ITNs were associated with:

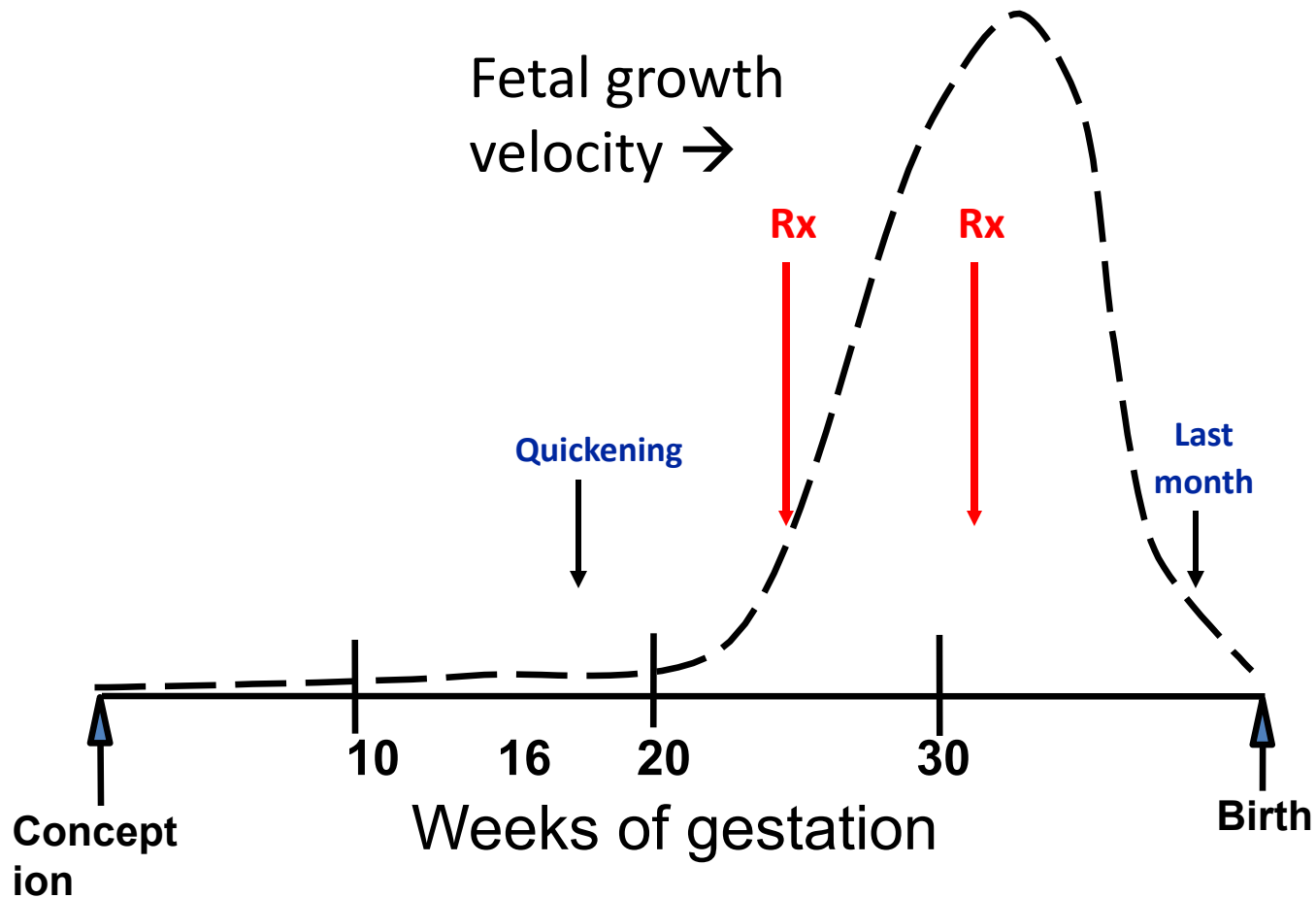
- During pregnancy
 - 38% reduction in peripheral parasitemia
 - 21% reduction in all causes of anemia (Hb < 11 g/dl)
 - 47% reduction in severe malarial anemia
- At delivery- 23% reduction in placental malaria
 - 28% reduction in LBW
 - 25% reduction in any adverse birth outcome
- No trend towards decreasing efficacy with increasing transmission rate

Contd.

C. Intermittent preventive treatment [IPT]:-

- **Based on the assumption that every pregnant women in endemic areas is malaria infected.**
- **Cost effective.**
- **Disadv. → drug adverse effects
→ devt. of
resistance to antimalarials.**
- **Recommendation = to give at least 2 doses of a safe & effective anti-malarial to all pregnant → sulfadoxine-pyrimethamine(SP)**

Rationale for the Timing of the SP Doses



Contd. [IPT]

- **MOH protocol = weekly chloroquine and daily proguanil → throughout pregnancy
→ 2 wks before, during stay
& for 4 wks after leaving. *Use
reported by 0.7% at ANC.**
- **Effects of IPT:- → all
parity**
 - *less antenatal parasitaemia RR=0.53,**

Contd.

*** Less maternal fever episodes,
RR 0.42, 95% CI 0.27, 0.66.**

→ 1st or 2nd pregnancy;

***severe anemia (RR
0.62, 95% CI 0.50-0.78) * fewer LBW
(RR 0.55, 95% CI 0.43-0.70) * higher
mean birth weight *
fewer perinatal deaths, (RR 0.73, CI
0.53- 0.99).**

Malaria & HIV / AIDS

- **Maternal HIV contributes to malaria infection in pregnancy, in prevalent areas (PAR= 10-27%).**
- **Malaria in pregnancy may increase the risk of MTC HIV transmission.**
- **For malaria prophylaxis 3 SP doses at 1st TM, 28 & 34 Wk is required.**
- **Reasonable to give 3 doses to all pregnant in a population if HIV seropositivity rate is >10%.**

Syphilis

Syphilis in pregnancy

- **The course of syphilis is unaltered by pregnancy**
- **The risk of fetal infection depends on the degree of maternal spirochetemia (2° stage > 1° or latent stages) & gestational age of the fetus.**
- **Treponemes may cross the placenta at all stages of pregnancy.**
- **Fetal involvement is rare < 18 wks because of fetal immunoincompetence. But > 18 wks, the fetus is able to mount an immunologic response, & tissue damage may result.**

Syphilis during Pregnancy---

- **The earlier the fetus is exposed, the more severe the fetal infection & the greater the risk of premature delivery or stillbirth.**
- **Antepartum infection in late pregnancy does not necessarily result in congenital infection & only 40–50% of such infants will have definite congenital infection.**
- **Placental infection can occur with resultant endarteritis, stromal hyperplasia, & immature villi.**

Syphilis during Pregnancy---

- **Grossly, the placenta looks hydropic (pale yellow, waxy, & enlarged). Polyhydramnios is frequently associated with symptomatic congenital infection**
- ***About 20% of children born to mothers with untreated syphilis will be normal.***
- ***Without therapy, fetal infections acquired early in pregnancy can result in miscarriage ,IUGR, IUFD,***
- ***hydrops fetalis, neonatal death, preterm birth, low birth weight, congenital anomalies,***
- ***active congenital syphilis in the neonate & long-term sequelae, such as deafness & neurologic impairment.***

PERINATAL TRANSMISSION

- **Universal early antepartum screening & treatment with appropriate antibiotics.**
- **Repeat the screening test during the third trimester (at 28 to 32 weeks) & again at delivery in women who are at high risk for syphilis.**
- ***Vertical transmission can occur at any stage of the disease.***
- ***Primary or secondary syphilis (50%), early latent (40%), Late latent (10 %), & Tertiary disease (10%).***
- ***Ultrasound of a fetus if maternal syphilis is diagnosed after 20 weeks of gestation to look for signs of fetal syphilis (eg, hydrops fetalis, hepatosplenomegaly, Amniotic fluid infection, Placental involvement)***

Effect of Syphilis on Pregnancy

- The more is the duration between infection and conception, the less is the foetal affection.
- Abortion: of a dead foetus after the 4th month of pregnancy when the spirochetes can cross the placenta as the cytotrophoblast starts to disappear.
- 1. Repeated late abortions then premature or mature macerated still born then live born with congenital syphilis or developing it later on.
- 2. Effect of Pregnancy on Syphilis
- Primary lesion which is the sign of early syphilis may be masked if infection occurs during pregnancy.

Laboratory Findings

- **Dark -field examination of specimens from cutaneous lesions usually identifies *T. pallidum***
- ***An immunofluorescent technique is now available for dried smears.***
- ***Silver staining for *T. pallidum* of biopsy specimens, placental sections, or autopsy material → confirm the diagnosis.***
- ***Motile spirochetes can be identified in amniotic fluid in women with syphilis & IUFD.***
- ***PCR is extremely specific for detection of *T. pallidum* in amniotic fluid & neonatal serum & spinal fluid.***
- ***Multiplex PCR (for *T. pallidum*, *H ducreyi*, & herpes simplex →not yet available)***

Nontreponemal Tests

- ***VDRL slide test, Rapid reagin test, & Automated reagin test. Measure reaginic antibody detected by highly purified cardiolipin-lecithin antigen.***
- ***Used principally for syphilis screening they are relatively specific.***
- ***VDRL will be positive 3–6 weeks after infection, or 2–3 weeks after the appearance of the primary lesion; almost invariably positive in the secondary stage.***
- ***The VDRL titer is usually high in secondary syphilis & lower or even nil in late forms of syphilis.***
- ***Satisfactory therapeutic progress → 4X ↓ titer in treated early syphilis or a falling or stable titer in latent or late syphilis.***

Treponemal Antibody Tests

- Detect antibody against *Treponema* spirochetes
 1. ***Fluorescent treponemal antibody-absorption (FTA-ABS) test***
 2. ***Microhemagglutination assay for *Treponema pallidum* (MHA-TP).***
- Both tests are more sensitive & specific than nontreponemal tests
- Remain positive despite therapy & are not given in titers or used to follow serologic response to treatment.

Sensitivity of Serologic Tests in Untreated Syphilis

Mean Percentage Positive (Range) at Indicated Stage of Disease^b

Test ^a	Primary	Secondary	Latent	Tertiary
VDRL, RPR	78 (74-87)	100	95 (88-100)	71 (37-94)
FTA-ABS	84 (70-100)	100	100	96
TP-PA ^c	89	100	100	NA

Treatment of syphilis

A. Early Syphilis & Contacts

B. Primary, secondary, & early latent syphilis (<1 year's duration):

- 1. Benzathine penicillin G 2.4 million units IM once.***
- 2. Tetracycline hydrochloride 500 mg orally 4 times daily or 100 mg Doxycycline twice daily for 14 days, for nonpregnant penicillin-allergic patients.***
 - Erythromycin estolate should not be administered to pregnant women because of potential drug-related hepatotoxicity.***
 - Ceftriaxone 1 g daily IM or IV for 8–10 days may be effective, but data on this regimen are limited.***

Treatment----

- ***Ceftriaxone 1 g daily IM or IV for 8–10 days may be effective, but data on this regimen are limited***

C. Late Syphilis: Includes latent syphilis of indeterminate duration or > 1 year's duration, except neurosyphilis.

- 1. Benzathine penicillin G 2.4 million units IM weekly for 3 successive weeks (7.2 million units total).***
- 2. Tetracycline hydrochloride 500 mg PO QID or Doxycycline 100 mg PO BID for 14 days, for penicillin-allergic patients.***

D. Syphilis in Pregnancy: as above except that tetracycline or erythromycin is not recommended.

Treatment----

- If serologic tests are equivocal (eg, possible biologic false-positive result), treat early & give the second dose of IM 2.4 million units of penicillin

E. Congenital Syphilis is prevented with adequate maternal treatment before 16–18 weeks' gestation

- Treatment after 16–18 weeks may arrest fetal syphilitic infection, but some stigmata may remain.
 1. For asymptomatic infants without neurosyphilis:
Benzathine penicillin G 50,000 U/kg IM stat
 2. For symptomatic infants or those with neurosyphilis
Aqueous crystalline penicillin G 50,000 U/kg IV every 8–12 hours, or procaine penicillin G 50,000 U/kg IM/day for 10–14 days.

Recommended Treatment of Syphilis

Primary, secondary, early latent (<1 year) syphilis

Recommended regimen:

Benzathine penicillin G, 2.4 million units IM once

Alternative oral regimens (penicillin-allergic, nonpregnant women):

Doxycycline 100 mg orally twice daily for 2 weeks

or

Tetracycline 500 mg orally four times daily for 2 weeks

Late latent, tertiary, and cardiovascular syphilis

Recommended regimen:

Benzathine penicillin G, 2.4 million units IM weekly times 3 doses

Alternative oral regimen (penicillin-allergic, nonpregnant women):

Doxycycline 100 mg orally twice daily for 4 weeks

Viral Hepatitis and pregnancy

Introduction

- Viral hepatitis is one of the most common and potentially serious that can occur in pregnancy women.
- Six form of viral have now identified ;HAV,HBV,HCV, HDV,HEV,HGV
- Two of each HAV and HBV can be prevented effectively through vaccination.

Intr...

Hepatitis A

- One third case of acute hepatitis in us
- Incidence in pregnancy is 1 in 1000

Transmission

- Person to person transmission through fecal oral contamination
- Poor hygiene and poor sanitation
- Serious complication uncommon ;case fatality ratio among reported case <1% but 2% in older than 50 years

Intr...

Hepatitis B

- Acute HBV in pregnancy is 1 to 2 per 1000
- Chronic HBV in pregnancy is 5 to 15 per 1000

Transmitted parenteral and sexual contact

- Although HBsAg detected in variety of body fluids only serum, semen, saliva are proven infectious

Risk

- Multiple sexual partners
- Perinatal exposure
- Inject drugs percutaneous
- Mortality associated with acute hepatitis is approximately 1%

Intr...

Hepatitis C

transmission

- transfusion of blood product
- Intravenous drug users
- Sexually lower than HBV

Risk

- Maternal HCV viral load
- Risk increased with invasive fetal monitoring and ROM >6 hours

Vaccination for hepatitis

Hepatitis A

Indicated for adult in risk

- Medical indication
- Behavioral risk
- Occupational risk
- Vaccine available in two form as single antigen or combination vaccine
- Given either 6-12 month apart or 6-18 month apart
- Combination vaccine given in three doses at 0,1 and 6 months
- 94-100% immunogenic after first dose

Cont...

Hepatitis B

- All individual with risk factors particularly HCW should vaccine against HBV infection
- Two single antigen vaccine for hepatitis B and one combination vaccine(twinrix) for at risk of hepatitis A and B
- Combination vaccine given in three dose at 0,1 and 6months
- An accelerated schedule 0,7 and 21-30 days followed by booster dose at 12 month is an option when rapid immune response is needed
- Pregnancy is not contraindicated for the vaccine.

Cont...

Table 1. Recommended Dosages and Schedules of Single-Antigen Hepatitis B Vaccines

Vaccine	Age Group	Dose	Volume	No. of Doses	Schedule*
Engerix-B† (GlaxoSmithKline)	0–19 y	10 mcg	0.5 mL	3	Infants: birth, age 1–4, 6–18 mo Alternative for older children: 0, 1–2, 4 mo
	20 y and older	20 mcg	1.0 mL	3	0, 1, 6 mo
Recombivax HB† (Merck & Co.)	0–19 y	5 mcg	0.5 mL	3	Infants: birth, age 1–4, 6–18 mo Alternative for older children: 0, 1–2, 4 mo
	11–15 y	10 mcg	1.0 mL	2	0, 4–6 mo
	20 y and older	10 mcg	1.0 mL	3	0, 1, 6 mo

Clinical manifestation of hepatitis

Subjective symptoms

- Nausea, anorexia, fatigue, malaise, right upper quadrant pain or epigastric pain

Typical physical finding

- Jaundice
- Upper Abdominal tenderness
- Hepatomegaly
- Patient urine darken, stool gray or acholic

Acute hepatitis management in pregnancy

- Hospitalized if have encephalopathy, coagulopathy, or severe debilitation
- Nutritional need should be addressed
- Fluid and electrolyte balanced
- Fresh frozen plasma or cryoprecipitate
- Activity should be limited
- Protect from upper abdomen trauma

Cont...

General test

- Aspartate transaminase (AST)
- Alanine transaminase (ALT)
- Bilirubin
- Coagulation abnormality and hyper amenomia
- Liver biopsy rarely indicated in pregnancy

Specific tests

- Hepatitis A,B,C,D,E

Cont...

Treatment for chronic hepatitis in pregnancy

- Chronic HBV and HCV infection should be referred for evaluation and managed in chronic liver disease
- Use methods to prevent or reduce transmission to other
- All HBsAg laboratory result should be reported

Cont...

- Vertical transmission can be reduced
- Routinely screen all pregnant women for HBsAg
- Approximately 10-20% of sero positive women transmit virus to neonate in the absence of immune prophylaxis
- Frequency of transmission depends on GA that infection occurs; in first trimester (10%) and in 3rd trimester (80-90%)
- CDC recommend that universal immunization for all infants
- Preterm birth weighting <2000 gram and born from women with HBsAg negative first dose vaccine should delay until one month

Intra partal care

- 85% to 95% cases of transmission is due to exposure to maternal contaminated blood or genital tract secretion
- Remaining hematogenous trans placental dissemination and close postnatal contact between infant and infected mother

Risk factor for intrauterine transmission

- Maternal HBV seropositivity
- History of threatened preterm labor
- Higher HBsAg and HBV titer

Cont...

- Route of delivery has not been shown to influence the risk of HCV transmission cesarean section for only obstetric indication
- The risk of transmission through amniocentesis is low in HBV or HCV in chronically
- Breast feeding is not contraindicated for hepatitis positive mother

Immunotherapy

Hepatitis A

- Given for non chronic and self limited course of symptomatic HAV infection
- No specific antiviral agent use as treatment
- Not contraindicated during pregnancy
- Patient close contact should be given post exposure prophylaxis if they have not immunized

Cont...

Hepatitis B

- No specific therapy is available for treatment of acute hepatitis B
- Chronic HBV infection referred for evaluation and treated in management of chronic liver disease
- Lamivudine and HBIG limit intrauterine HBV transmission
- Lamivudine 150mg orally daily starting at 34 wks. in high risk patient viral load($>10^7$)

Cont...

Hepatitis C

- Lack any available prenatal or postnatal pharmacological immunologic measurement to decrease vertical transmission
- Interferon and ribavirin use in adults but contraindicated in pregnancy

Accidental or occupational exposure

- All HCW who may be exposed to blood or blood product should be vaccinated against hepatitis B
- Risk of infection per injury with HBV infected blood is 20% to 30%
- Mucosal contamination from body fluid splash exposure
- Risk of acquiring HCV lower than HBV(30%)and higher than risk of HIV(0.3%)
- So routinely standard precaution should be practice

Urinary Tract Infection in Pregnancy

- ASYMPTOMATIC BACTERIURIA
- Definition
- It is the presence of 100.000 organisms/ml of the same species in two cultured fresh, mid-stream specimens of urine.
- Incidence
- 2-5% of pregnant women. If not treated 30% of them will develop symptomatic infections.
- Complications:
 - 1- Symptomatic infections as cystitis and pyelonephritis.
 - 2- Anaemia.
 - 3- Hypertension.
 - 4- Intrauterine growth retardation.
 - 5- Pre-term delivery.
- Treatment
 - - Ampicillin or cephalosporin 500 mg/ 6 hours for 10 days or
 - - Nitrofurantoin 100 mg/6 hours.

Con't

- Pyelonephritis
- Definition
- It is inflammation of the renal pelvis and parenchyma.
- Incidence
- 30% in cases with asymptomatic bacteriuria and 1% in cases without.
- Predisposing Factors During Pregnancy
- (A) Urine stasis during pregnancy due to:
 - Compression of the ureter by the gravid uterus against the pelvic brim particularly on the right side. So infection is more common on the right side.
 - Relaxation of the ureter by progesterone effect.2.
- (B) Increased urinary excretion of glucose and amino acids favours the growth of bacteria.
- Causative Organisms
 - - Escherichia coli (E.coli)(90%).
 - - Klebsiella, streptococcus, staphylococcus, proteus, pseudomonas and others.

Diagnosis

- Symptoms: started usually after 16 weeks in the form of;
 - - malaise,
 - - anorexia,
 - - nausea and vomiting,
 - - rigors,
 - - dysuria,
 - - urgency and frequency of micturition,
 - - renal pain commonly on the right side.
- Signs:
 - - Fever reaching 40°C ,
 - - rapid pulse,
 - - tenderness in one or both renal angles (costovertebral angle).

Investigations:

- 1.Urine analysis: pus cells, organisms and proteins. Casts and RBCs may be present.
- N.B. Presence of organisms without pus cells suggests contamination, while pus cells without organisms creates suspicion of tuberculosis.
- . Culture and sensitivity : for urine.
- 3. Blood picture : leucocytosis.
- Differential diagnosis:
- Causes of acute abdomen as appendicitis, abruptio placentae and complications of pelvic tumours.
- Causes of vomiting.

Complications:

- 1. Chronicity : with recurrent infections. In these cases, plain X-ray and intravenous pyelography (IVP) should be done after delivery to exclude urinary stones. Chronic pyelonephritis may result in hypertension and renal failure later on .
- 2. Abortion, intrauterine foetal death, IUGR or premature labour may result.
- Treatment
 - 1. Bed rest: light diet and plenty of fluids. Intravenous fluid may be needed if there is vomiting
 - 2. Analgesics and antipyretics.
 - 3. Alkalies: as potassium citrate to inhibit the growth of E.coli.
 - 4. Antibiotics and chemotherapy: The following therapy is started until the result of culture and sensitivity is obtained.
 - -Ampicillin 500 mg/ 6 hours, or
 - - Nitrofurantoin 100 mg/ 6hours, or
 - - Cephalosporins 500 mg/ 6 hours.
 - - Treatment is continued for 7-10 days.

Complicated pregnancy/bad obstetrics history

Definition

- It is the pregnancy in which the mother foetus and / or newborn are at risk of morbidity or mortality during pregnancy, labour and/ or postpartum.
- Incidence
- About 20% of all pregnancies.
- Causes
- (A) Maternal factors:
- Age : below 16 years or above 35 years particularly if the patient is primigravida.
- Grand multiparity: 5 or more previous deliveries.
- Habits: as heavy smoking, alcoholism or drug addiction.

con't

- Bad obstetric history:
- Repeated abortion.
- Repeated preterm labour.
- Prolonged or difficult labour particularly if was ended by stillbirth or neonatal death.
- Operative delivery as caesarean section or forceps.
- History or current medical disorders:(Hypertension, Diabetes, Cardiac, Renal, Pulmonary, Hepatic, Anaemia.)
- Coagulation defects.
- Haemoglobinopathies
- Serious infections as AIDS.
- History of surgery or trauma:
 - ● Myomectomy
 - ● Metroplasty.
 - ● Pelvic trauma.

Con't

- . (B) Foetal factors: Malpresentations and malpositions.● Multiple pregnancy.● Antepartum haemorrhage.● Congenital anomalies.● Premature rupture of membranes.● Rh-isoimmunization.● Intrauterine growth retardation.● Macrosomia.● Poly - or oligohydramnios.● Post-term pregnancy.●

Elderly primigravida

- Definition:
- Primigravida whose age is above 35 years.
- Dangers:
- This woman is more liable to: Hypertension with pregnancy.
- 1. Abruptio placentae.
- 2. Higher incidence of fibroid with pregnancy.
- 3. Post-term pregnancy.
- 4. Uterine inertia and prolonged labour.
- 5. Rigid perineum so instrumental delivery are more needed.
- 6. More caesarean section delivery as the foetus is precious

The grand multipara

- Definition:
- Woman who had 5 or more previous deliveries.
- Dangers:
- This woman is more liable to:
- 1- Anaemia.
- 2- Hypertension with pregnancy.
- 3- Diabetes.
- 4- Placenta praevia.
- 5- Pendulous abdomen.

Cont...

- 6- Malpresentation and malposition.
- 7- Uterine inertia and prolonged labour.
- 8- Instrumental delivery and caesarean section are more needed.
- 9- Obstructed labour which may lead to rupture uterus due to :
 - a) higher incidence of malpresentations and malpositions,
 - b) pendulous abdomen,
 - c) weak uterine muscles,
 - d) some osteomalacic changes in the pelvis,
 - e) larger sized baby,
 - f) false sense of security due to previous normal deliveries.
- 10- Postpartum haemorrhage.

Management

- Frequent antenatal visits.
- Management of the cause.
- Monitoring of foetal well-being .
- Delivery in well - equipped hospital under senior staff supervision

Have a nice time and stay at
Home!!!