



Obstetric Syllabus

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2 Hormones in pregnancy

3 Physiological changes

4-6 Antenatal care

7-12 Medical complications in pregnancy

13-14 HTN in pregnancy

15 DM in pregnancy

16 Pelvis

17 Labor

18-19 Malpresentation

20-21 Stages of labor

22 Episiotomy

23-24 Puerperium

25-26 Amniotic fluid

27-28 Gestational age

29-31 Rh immunization

32-33 Multiple gestations

34-36 Antepartum Hemorrhage

37 Postpartum Hemorrhage

38-40 Miscarriage

41-42 PROM

43 Cervical incompetence

44 TORCH

Hormones in pregnancy [Fetoplacenta largely controls the endocrinologic events of the pregnancy]

Steroid Hormones

progesterone

Source:

- Non pregnant – corpus luteum (CL)
- Pregnant <7 weeks – CL
- 7-9wks – CL & placenta
- > 9 weeks – placenta

Purpose:

Early – secretory endometrium (ready for implantation)

Late – stabilization of myometrium by inhibiting premature contractions.

Estrogen

Form	State	Source
Estradiol (E2)	Non-pregnant Reproductive age	Follicle (Granulosa cells)
Estriol E3	Pregnant	Placenta (From fetal adrenal DHEAS)
Estrone E1	Menopause	Adipose tissue

Androgen

Source: Mainly fetal adrenals

Purpose: Precursor for Estradiol & Estriol in Placenta

Glucocorticoid

Source: Fetal adrenal glands + Placenta

Purpose: Fetal Lung Maturity

Peptide Hormones

hCG (human chorionic gonadotropin)

⇒ Maintain corpus luteum for first 10 wks

Source: placental syncytiotrophoblast

A glycoprotein with 2 subunits
α (non-specific), β (Specific)

-Begins to rise 8 days after Ovulation.
-Appears in maternal blood 10 days after Fertilization.

» 1st trimester: Double every 72hrs.
» Peaks at 9-10 weeks.
» Then Decline to more constant level.
» After normal pregnancy: Return to normal within 2-4 weeks.

» DDx for ↑ hCG:

- Twins
- Hydatidiform Mole
- Choriocarcinomas
- Embryonal carcinoma

» DDx for ↓ hCG:

- Ectopic
- Abortion (threatened or missed)

detect since 1wks
" 2wks

serum test
urine test

hPL (Human placental lactogen)

Source: Placenta

Antagonizes the cellular action of insulin & decreases maternal glucose utilization → Increases glucose availability to the fetus.

Play a role in the pathogenesis of gestational DM.

Appears at 2nd trimester.

Maternal serum concentrations of hPL parallel placental weight.

CRH (Corticotropin Releasing Hormone)

Source: Fetal hypothalamus & placenta towards the end of the pregnancy.

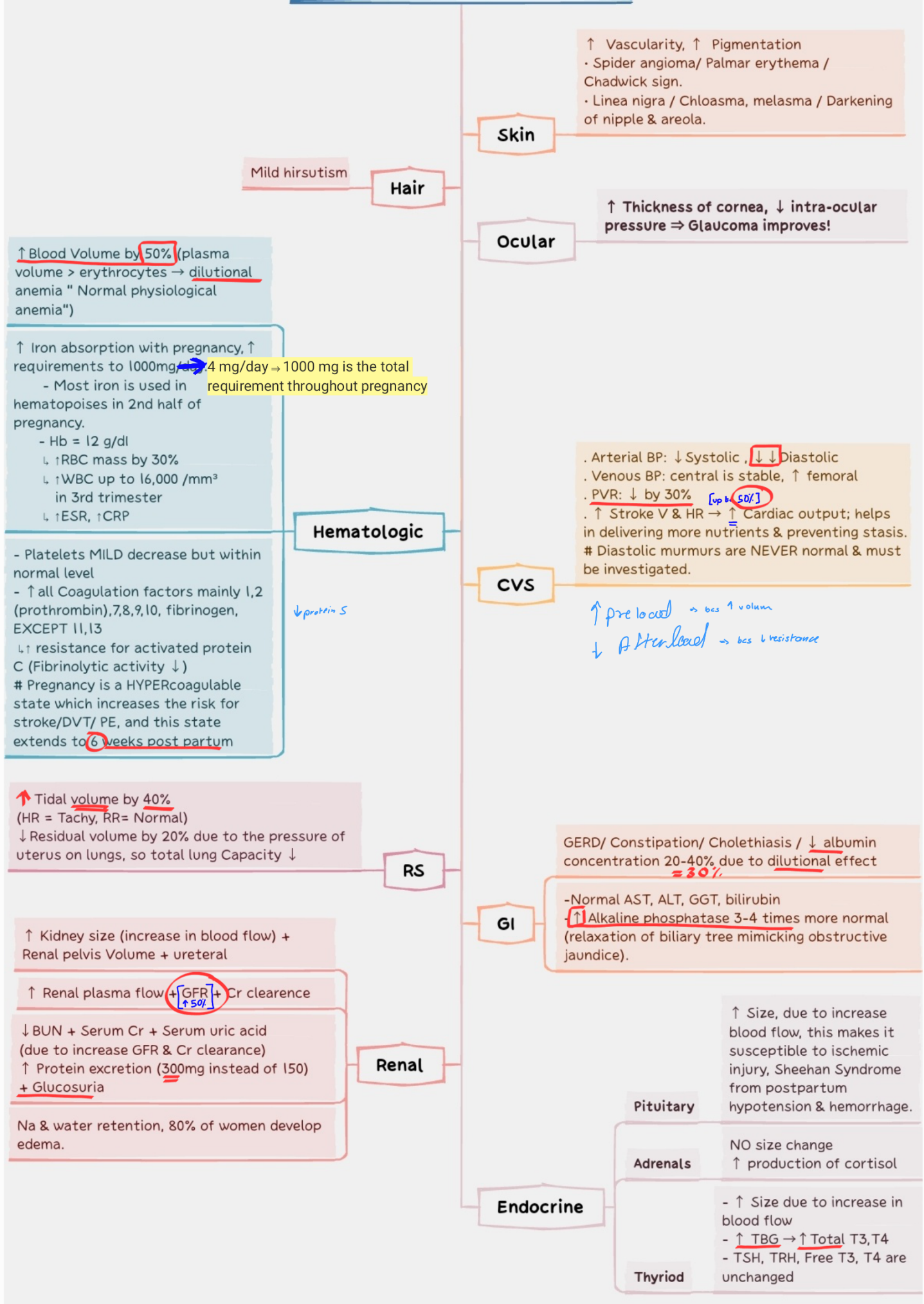
Activation & amplification of LABOR for both preterm & term.

Prolactin

Source: Maternal & fetal ant. pituitary (in late pregnancy)

Stimulation of postpartum milk production

Physiological Changes



Antenatal Care

Healthcare that women receive throughout pregnancy.

Frequency of check-ups

- Until the 28th week of pregnancy: monthly
- From the 28th week until the 36th week: every two weeks
- From the 36th week until birth: every week
- ~ In high-risk pregnancies, frequent visits are usually warranted

1st trimester (first 13 weeks)

Personal and family history, previous pregnancies + physical examination + BP + wt.

Labs

- CBC / Hb
- Blood group, Rh, Antibody screen (Indirect Coombs)
- Urine analysis and urine culture
- Pap smear
- Gonorrhea & chlamydia culture & PCR
- Infection screen: Rubella/ Syphilis/ HBV/ HIV/ TB

US



- Evaluate the presence & location of the pregnancy (intrauterine or ectopic pregnancy).
- Determine the number of fetuses.
- Confirm gestational age (due date).
- Document crown-rump length and cardiac activity. → Between 45-84 mm
- Screen for abnormalities of the cervix, uterus, and placenta.
- Fetal heart monitoring -- at 6 weeks.
- Nuchal translucency (NT) at 11th wks: normal NT is ≤ 3 mm, it is abnormal when > 3 mm



2nd trimester (14 - 27 weeks)

Labs

- 50mg, 1 hr OGTT (at 24-28 weeks) / 100-g, 3 hrs OGTT to confirm the diagnosis in case of positive initial screening
- Repeat Hb, CBC ...
- Triple marker test: MS-AFP, hCG, Estriol OR
- Quadruple marker test: MS-AFP, HCG, Estriol, Inhibin-A.
- ↳ DDx for ↑ MS-AFP (> 2.5)
 - Wrong date (most common)
 - Twin pregnancy
 - Neural Tube Defect
 - Ventral Wall Defects: Gastroschisis or omphalocele
 - Renal disease
 - Sacrococcygeal teratoma
- ↳ DDx for ↓ MS-AFP ($< 0.75-0.85$)
 - Wrong date
 - Trisomy

US

- Rule out congenital anomalies by detailed US between 18-22 weeks (typically at the 20th week)
- Amniotic fluid index (AFI)
- Cervical length and changes
- Cervical incompetence (funneling)

Physical exam

Fundal height and position of the fetus (Leopold maneuver)

3rd trimester (28 - 38/40weeks)

Labs

- CBC, OGTT, BP and urine dipstick (risk of PET)
- Indirect Coombs test (atypical antibody screen AAT): if negative, give anti-D at 28 weeks of gestation.
- Screening for Group B streptococcus between 36 & 37wks of gestation (vaginal and rectal swab) because colonization by this bacteria may cause chorioamnionitis & neonatal infection

US

- Placental location in relation with the internal os
- Presentation
- Biophysical profile (BPP)
- Postmature placental signs (calcifications)
- Fetal weight
- Biometric measurements in relation to gestational age to rule out Intrauterine growth restriction (IUGR):
 1. Head circumference
 2. Biparietal diameter
 3. Abdominal circumference
 4. Femoral length

NOTES

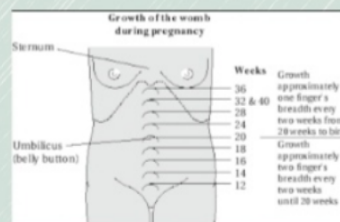
Estimated Delivery Date = +9 months +7 days ⇒ From LMP

US → Crown-rump length

For LMP to be reliable reference for GA calculation, it has to be REGULAR for at least 3 cycles prior to conception.

Gestational Age by:

- Abdominal exam → by fundal height
- From symphysis pubis to umbilicus = 20 weeks
 - ↳ Above it, every cm = 1 week
 - After 36 weeks the fetus descends into the pelvis.
 - # So fundal height is useful to predict GA from 20-36 weeks.



Leopold maneuver



To determine the presentation, position, and engagement of the fetus

1st

Bimanual examination of the fundal height & fetal position (longitudinal/oblique/transverse)

2nd

Bimanual examination of the location of the fetal back (on the mother's left or right side)

3rd

One hand grasps above the symphysis in an attempt to determine if the presenting part of the fetus is engaged.
↳ In cases of cephalic presentation, the fetal head feels hard and ballotable; if the fetus is in a breech position, a soft and less movable rump can be felt.

4th

Bimanual determination of the location of the fetal brow and the degree of flexion of the head. Usually performed during the later stages of pregnancy when the fetus has entered the pelvic inlet.

Antepartum fetal surveillance

Biophysical profile (BPP) = 4Ds

- Ultrasound exam
 - Fetal movement
 - Fetal tone
 - Fetal breathing
 - Amniotic fluid volume
 - NST

→ need 20-30 mins.

Indications

congenital anomaly.

Non-invasive test (CTG) that measures FHR reactivity to fetal movements.

~ Usually performed in the third trimester



Nonstress test (NST)

Indications

- » Maternal medical conditions (e.g., gestational diabetes, preeclampsia)
- » Fetal conditions (e.g., fetal heart defects, fetal growth restriction) that increase the risk of fetal hypoxic injury or death

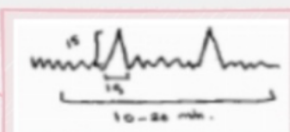
NST: correlate FHR to fetal movement without uterine contraction

Cardiotocography (CTG)

Open: <https://geekymedics.com/how-to-read-a-ctg/>

It assesses fetal wellbeing by measuring the relationship between fetal heart rate and uterine contractions during labor.

- A normal fetal heart rate is between 110-160 bpm.
- Normal variability is between 5-25 bpm.



Accelerations

Fetal heart changes with uterine contractions

- If no change → abnormal
- Acceleration → normal response

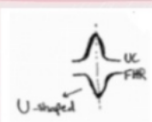
Criteria:

At least 2 accelerations of ≥ 15 bpm last ≥ 15 seconds within 10-20 mins.

» Reactive/Reassuring NST criteria:

- Rise in HR > 15 bpm ABOVE baseline
- Good variability
- No deceleration
- Normal baseline

» Non-reactive/Non-reassuring if not meeting the criteria above.



Early

Due to head compression, seen in head enlargement (increase ICP → Increase vagal response)

Late

- Bad, due to uteroplacental insufficiency → hypoxia / acidosis → fetal distress

Variable

Most common, seen mostly in 2nd stage of labor, NOT related to uterine contractions, due to cord compression (partial or complete) or prolapse

Mixed

Decelerations

CTG: correlate FHR with uterine contraction (during labor).

CST: Induce uterine contraction (by low-dose oxytocin) and monitor FHR, to see if the fetus can handle NVD or not. (not done anymore)

Negative CST = Reactive, Positive CST = Non-reactive

Modified biophysical =

1 - NST

2 - AFI

Medical Complications in Pregnancy

1. Heart Disease

Rheumatic HD

The most common lesion associated with rheumatic HD $\Rightarrow$ Mitral stenosis (MS), followed by mitral regurgitation.

During pregnancy, the mechanical obstruction associated with MS worsens as cardiac output increases which lead to increase in preload (due to $\uparrow$ blood ^{50%} volume), and that increase left atrium overload, leading to pulmonary HTN.

- Asymptomatic patients may develop symptoms of cardiac decompensation or pulmonary edema as pregnancy progresses.

- A.fib is more common in patients with SEVER MS, all women who develop A. fib during pregnancy experience congestive HF. *A.fib + Mitral stenosis = Congestive HF.*

$\Rightarrow$ 25% of pts with MS develop HF for the 1st time during pregnancy.

$\Rightarrow$ MS increase the risk of IUGR

Management of MS: $\downarrow$ tachycardia, $\downarrow$ excessive volume load, considers intrapartum subacute bacterial prophylaxis.

Congenital HD

- Patients with persistent ASD or VSD and tetralogy of fallot with complete surgical correction generally tolerate pregnancy well.

- Patients with primary pulmonary hypertension or cyanotic heart disease with residual pulmonary hypertension are in danger of experiencing de-compensation during pregnancy.

Significant Pulmonary HTN with Eisenmenger syndrome ^{cyanotic} is a CONTRAINDICATION to pregnancy $\rightarrow$ due to high maternal mortality.

Antepartum care in heart disease

- Avoidance of excessive wt. gain & edema $\rightarrow$ BY rest in left lateral decubitus position at least for 1hr daily/ Avoid excessive salt intake/ Diuretics if HF.
- Avoidance of strenuous activity / Avoidance of anemia.
- Avoidance of infection $\rightarrow$ Routine screening for STDs & UTIs, immunizations.
- Fetal echo-cardiography {Women with congenital HD have a high risk of having children with HD}.

Management of Delivery and the Immediate Postpartum Period

◇ Delivered vaginally UNLESS obstetric indications for C/S are present

- ↳ Labor in the lateral decubitus position

- ↳ Frequent assessment of vital signs, urine output, pulse oximetry, adequate pain relief

- ↳ Pushing should be avoided during the 2nd stage of labor (assisted delivery by outlet forceps, vacuum extractor)

◇ The immediate postpartum period

- ↳ Monitor fluid balance (avoiding overload)

- ↳ Prevention of uterine atony (avoiding depletion from blood loss) with oxytocin and uterine massage.

- ↳ Prevent endocarditis: prophylactic antibiotic is given to high-risk patients (prosthetic valves, unrepaired or incompletely repaired congenital heart disease, or a previous history of bacterial endocarditis)

2. Gastrointestinal Disorders

Hyperemesis gravidarum

Persistent nausea and vomiting associated with ketosis and loss of more than 5% of pre-pregnancy body weight.

Risk factors

1. First pregnancies
2. Multiple pregnancies
3. Trophoblastic disease

» Clinical presentation

History of intractable vomiting, beginning in the 1st trimester, significant abdominal pain, absent tenderness.

» Signs

Weight loss, dry and coated tongue, and decreased skin turgor

» Work up

- Urine tests for ketonuria
- Blood tests for electrolytes and acetone (electrolyte disturbances

↓ usually seen: hypokalemia, hyponatremia, and hypochloremic alkalosis)

↑ • BUN, creatinine, lipase, liver function and amylase (usually elevated)

↑ • TSH; hyper-thyroid

» Treatment: Symptomatic.

↓ If outpatient management fails, patients must be admitted for IV administration of fluids, electrolytes, glucose, vitamins, and medical therapy.

Gastroesophageal Reflux Disease (more common 70%)

Clinical presentation

- ~ Discomfort aggravated by meals and the recumbent position
- ~ Water brash, which is a salty taste and produces nausea.

Treatment

- ~ Symptomatic: Upper GI endoscopy is not necessary, unless there is significant GI bleeding
- ~ Give instruction to patient: refrain from eating large and late meals/ avoid the recumbent position especially after meals/ use an extra pillow to elevate the head when sleeping.
- ⇒ Antacids If failed: H2 receptor antagonist (cimetidine) or a proton pump inhibitor (omeprazole)

Intrahepatic Cholestasis

Clinical presentation

- Itching (most intense on the palms and soles and it is the MAIN SYMPTOM), without abdominal pain or a rash

Features

- Cholestasis and pruritus in the 2nd half of pregnancy without other major liver dysfunction
 - Tendency for recurrence with each pregnancy
 - An association with OCPs and multiple gestations
 - No maternal hepatic sequelae
 - Increased rate of meconium-stained amniotic fluid and fetal demise
- ↓ Treatment ⇒ Ursodeoxycholic acid

3 Endocrine Disease

Thyroid diseases common in pregnancy, 2% of pregnancies.

- Normally, during pregnancy $\uparrow$ Estrogen $\rightarrow \uparrow$ TBG
 - ↳ Serum levels of free T3 and T4 usually remain in a normal range.
- hCG & TSH have similarities, in 1st trimester, a Hormone Spillover syndrome can occur in which hCG stimulates TSH receptors & gives a biochemical picture of hyperthyroidism, and it resolves spontaneously as pregnancy advances $\rightarrow$ common in multiple pregnancies, trophoblastic disease & hyperemesis gravidarum.
- Iodine deficiency; neurological cretinism is the leading preventable cause of mental handicap. The developing cochlea, cerebral neocortex & basal ganglia are most sensitive to iodine deficiency, especially in 2nd trimester, resulting in deaf-mutism, intellectual deficiency & spastic motor disorder.

Hyperthyroidism

$\downarrow$ TSH, $\uparrow$ Free T4 & T3

Complications

Fetal $\Rightarrow$ IUGR, Stillbirth, Prematurity.
Maternal $\Rightarrow$ PET, Congestive HF, Thyroid storm

Management

Medical treatment is the first line

- Significant maternal tachycardia: B-blocker
- Antithyroid agents
 - ↳ PTU should be used in 1st trimester & carbimazole in the 2nd & 3rd trimesters.
 - ↳ No teratogenesis has been linked with anti-thyroid drugs.
 - ↳ Both drugs cause agranulocytosis.
 - ↳ Advise them to lactate; doses should be split throughout the day, with feeding to occur before a dose where possible, and monitoring of neonatal thyroid function.
 - ◇ Biochemically euthyroidism remains the goal.
 - ◇ TSH levels should be measured monthly when control is good & more frequently when the diagnosis is new or there is a relapse.

Surgical management during 2nd trimester is recommended ONLY when medical treatment fails, compression from a large goitre, or suspicion of malignancy.

Radioactive iodine is contraindicated in pregnancy. Lactation should be stopped 4 weeks if it's given in the puerperium.

TSH receptor stimulating antibodies cross the placenta & the risk of fetal graves' disease after 20 weeks is directly proportional to their titre $\rightarrow$ The risk is very LOW.

Signs of fetal thyrotoxicosis: Tachycardia, excessive movements, fetal growth restriction, oligohydramnios & goitre.

Consequences of thyrotoxicosis $\rightarrow$ premature delivery, craniosynostosis, intellectual impairment, hydrops fetalis, polyhydramnios related to oesophageal pressure, obstructed labor from neck extension related to goitre.

Management:

>34 weeks of GA $\rightarrow$ delivery, otherwise high dose of antithyroid drugs should be given, the pregnant woman can take thyroxine if she becomes clinically hypothyroid and this will not cross the placenta "Add-back therapy"

Requires prompt recognition and aggressive reversal of thyrotoxicosis with antithyroid drugs (PTU & methimazole), and supportive management of signs & symptoms.

Thyroid storm

- Give an initial loading dose of 300mg to 600mg PTU ^{GI} enterally, then 150mg to 300mg every 6hrs. Can be given by nasogastric tube or can be compounded as a rectal suppository.
- Iodides are given; they rapidly inhibit the release of thyroid hormones, administered several hrs after PTU to avoid the buildup of hormones stored.
- B-Blockers /IV fluids /antipyretics /correction electrolyte imbalances / glucocorticoids to inhibit the conversion of T4 to T3 & prevent adrenal insufficient /frequent monitoring of vital signs & fetus.

Avoid delivery during thyroid storm

Hypothyroidism

- » Untreated hypothyroidism ↑ TSH, ↓ T4 → Require urgent initiation at thyroxine.
- » Previously diagnosed hypothyroidism → Rapid achievement of euthyroidism
- » Subclinical hypothyroidism ↑ TSH ONLY → Thyroxine
- » Hypothyroxinemia ↓ T4, normal TSH → Asymptomatic

Complications: if left untreated

1. Spontaneous abortion
2. Preeclampsia
3. Abruption & prematurity
4. Low-birth-weight or stillborn infants
5. Lower cognitive function in offspring.

Treatment

Once diagnosed, therapy such as levothyroxine should be started, and serum TSH levels should be measured 3 times during pregnancy if hypothyroidism is well controlled.

- ✦ TSH is the one you should depend on it.
- ✦ Biochemically euthyroidism remains the goal.

Factors could influence thyroxine dosage during pregrang

- Reduce absorption in the 1st trimester related to nausea & vomiting
- Malabsorption resulting from the binding of thyroxine to iron & calcium supplements
- Altered compliance
- Normal variation in the dosage
- Suboptimal control prior to conception

Only 1st trimester control influences fetal wellbeing

4. Epilepsy

◇ An increase in seizure frequency in 15-37% of pregnant women, possibly related to non-adherence to medication or sleep deprivation.

◇ 1-2% experience a tonic-clonic seizure in labor or within the first 12hrs after delivery.

Effects of pregnancy on Antiepileptic drugs ⇒ Reduction in total plasma concentration & the unbound fraction of AEDs, reduction in lamotrigine levels results in aggravation of seizures.

Implications for fetal and child development

1. Seizure

» Tonic-clonic seizures are associated with:

- * Fetal hypoxia
- * Fetal intracranial haemorrhage
- * Fetal loss (spontaneous abortion, stillbirth, and perinatal loss)

2. Fetal loss

3. IUGR associated with polytherapy (number of medications + severity of the disease)

4. Major malformations (Congenital heart defects, neural tube defects, urogenital defects like glandular hypospadias, and orofacial clefts)
- Risk is highest in 1st trimester

- Phenytoin has lower risks than other AEDs.

5. Minor malformations

- Fetal anticonvulsant syndrome: A group of malformations +- developmental delay that can affect some babies if they are exposed to AEDs.

6. Long-term developmental effects

- Poorer developmental outcomes with valproate.

Risks to the baby and the prognosis of the pregnancy are much **WORSE** if the pt. is **NOT** taking the medication.

Supplements

Folic acid — Folate supplementation before 3 months of conception = 5 mg/day

Vit. K — 1 mg of parenteral vit. K at birth to all children born to mothers taking enzyme-inducing AEDs

If the mom is having absence seizures → higher risk of having a baby complaining of seizures than if she has generalized or partial seizures.

Breastfeeding should be encouraged

» No need to change the medication for the mom.

Phenytoin, carbamazepine & valproate ⇒ found in low concentrations in breast milk

Phenobarbital & primidone ⇒ found in higher level

↳ Immature drug metabolism & reduced serum protein binding in infants may lead to drug accumulation.

lamotrigine & topiramate ⇒ significantly excreted in breastmilk, but no adverse effects have been reported

Management

Localisation related epilepsy $\Rightarrow$ Carbamazepine

» Change the valproate only if the pt. presented to the clinic pre-conception

» If she is already pregnant, for 8 wks, and her seizures are controlled by valproate
 $\rightarrow$ Do not change it.

Follow up by

- Detailed anatomical US to detect structural anomalies between 18-20 weeks.
- Alpha-fetoprotein at 15-22 weeks

Delivery

- Vaginal delivery by a consultant obstetrics unit with an epilepsy team if there's no indication for C/S.
- Recurrent or single prolonged tonic-clonic seizures $\rightarrow$ Subbuccal midazolam, IV lorazepam, or rectal diazepam.
- Clobazam 10 mg if seizures increase in perinatal period.
- Benzodiazepines can be associated with sedation in the newborn.

- Advice NOT to stop medication even if they are fasting.
- Differentiate between epileptic attack & eclampsia $\rightarrow$ totally different management.

Contraception

Types

Enzyme-inducing AEDs
[carbamazepine, phenytoin, phenobarbital, primidone, topiramate]

Non-enzyme inducing AEDs
[Valproate, levetiracetam, gabapentin, vigabatrin, tiagabine, pregabalin]

Copper IUD, mirena IU or medroxy-progesterone acetate injections

- All other methods may be affected (high failure rate).

If she wants OCPs $\Rightarrow$ Needs to double the dose by taking 2 packs of OCPs at the same time

All methods of contraception maybe offered

Emergency contraception — Copper IUD

lamotrigine + OCP $\rightarrow$ Potential increase in seizures due to a fall in the levels of lamotrigine.

HTN disorders of pregnancy

Pre-eclampsia

- Multisystem disorder.
- PREEclampsia: HTN at least 140/90 recorded on 2 separate occasions & at least 4 hrs apart, proteinuria 300mg, and end-organ dysfunction.
- AFTER the 20th week of gestation.
- Resolving by 6th postpartum week.

		dipstick	Or 24 h urine collection	
Mild	More than 140/90 + proteinuria	+1/+2	300mg	
Severe	More than 160/110 + proteinuria	+3/+4	5g	Symptoms of PET or increase LFT or DIC or pulmonary edema

Superimposed preeclampsia

Preeclampsia with chronic HTN.

HELLP Syndrome

Coagulation activation & liver failure
hemolytic anemia (schistocyte, ↑ bilirubin, ↓ haptoglobin)

A life-threatening form of preeclampsia characterized by Hemolysis, Elevated Liver enzymes, and Low Platelets.

More likely to be multiparous, >25 years old and less than 36 weeks of gestation.

- Extravillous trophoblast fails to penetrate myometrium

- spiral arteries don't develop normally
↓
placental hypoperfusion

- placental biopsy ⇒ Fibrinoid necrosis

Pathophysiology

Disorder of placenta

Placental bed biopsies have demonstrated that trophoblast invasion is patchy in pre-eclampsia and the spiral arteries retain their muscular walls.
↳ This is thought to prevent the development of a high flow, low impedance uteroplacental circulation.

Defective trophoblast invasion results in relative under-perfusion of the placenta and this releases factors into the maternal circulation that targets the vascular endothelium. → vasospasm & coagulation

Risk factors

- 1) 1st pregnancy
- 2) Extremes of age
- 3) Methamphetamine use
- 4) BMI > 35
- 5) Hx of preeclampsia (family or personal) / Chronic HTN
- 6) Obesity/ DM/ Thrombophilia / chronic kidney disease
- 7) Multiple gestation/ polyhydramnios/ macrosomia
- 8) Gestational trophoblastic disease
- 9) Antiphospholipid antibodies. / Lupus

Clinical manifestations

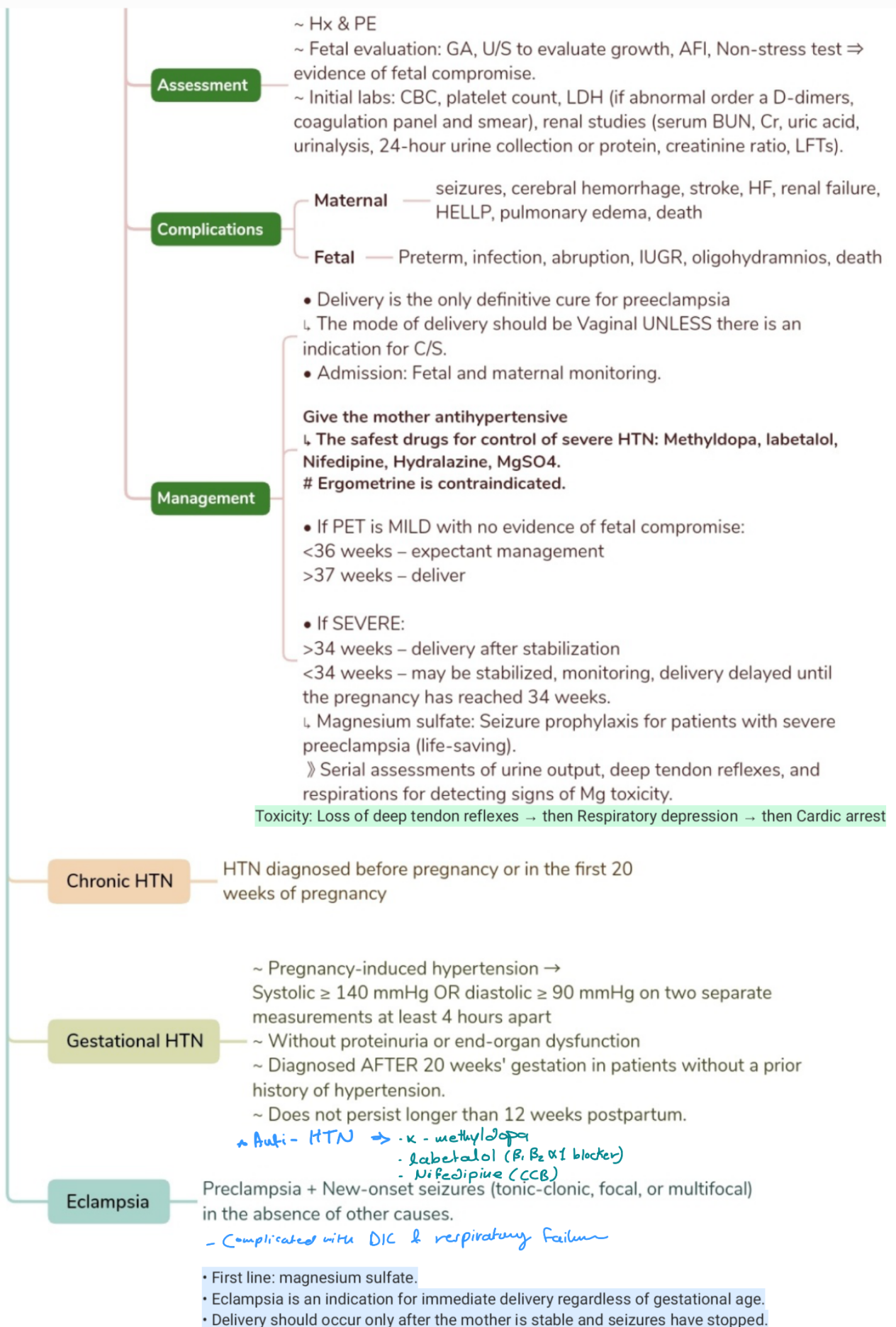
CNS — Headache / blurry vision/ scotoma/ stroke → Neurological examination may reveal hyperreflexia & clonus in sever cases.

CVS / RS — Edema / Pulmonary edema

Renal — Glomerulo-endotheliosis, impaired GFR & selective loss of intermediate wt. proteins (Albumin/transferrin) → proteinuria & edema

Hematological — Thrombocytopenia in PET → BAD prognostic

Liver — Nausea, vomiting + RUQ pain = HELLP (hemolysis & low platelet count)



DM in pregnancy

Definitions

- Pre-gestational DM** — Diabetes present before pregnancy and may be either type 1 or 2.
- Gestational DM** — Glucose intolerance with onset after 20 weeks of pregnancy.

Diabetogenic hormones

- HPL (secreted from the placenta in the 2nd half of pregnancy and explains the development of DM at the same time period)
- Placental insulinase
- Cortisol
- Progesterone
- Prolactin

Risk factors

1. Age (<25 years old)
2. Obesity
3. Family history of GDM or DM
4. PCOS
5. Recurrent infections
6. HTN/PET
7. Previous history of GDM/ Macrosomia/ Polyhydramnios
8. Current pregnancy: Polyhydramnios/ Macrosomia.
9. History of unexplained fetal death/ Neonatal death/ Congenital anomalies/ IUGR

Complications

~ Glucose crosses the placenta by facilitated diffusion, causing fetal hyperglycemia and eventually fetal hyperinsulinemia.

~ Pre-gestational DM is associated with a higher rate of maternal & fetal complications.

Maternal complications

1. Difficulty in controlling diabetes due to: increase body weight /increase volume distribution/hyperemesis gravidarum
2. Recurrent hospitalization due to: recurrent hypo and hyperglycemia /UTI
3. Increased risk of abortion
4. Increased risk of preterm labor and PROM
5. Increased risk of traumatic delivery
6. Increased risk of developing DM (type 2), metabolic syndrome, obesity and CVS disease.
7. Increased risk of vascular or end organ involvement or deterioration.

Fetal complication

1. Congenital anomalies: Only in pre-gestational DM.
 - ↳ Most common: cardiac anomalies (VSD/ASD/TOF)
 - ↳ Most specific and pathognomonic for DM: Caudal regression.
2. Macrosomia and traumatic delivery
3. Neonatal hypoglycemia, hypocalcemia, hypomagnesemia.
4. IUGR
6. Intrauterine fetal death
7. Increased risk to develop DM, HTN, CVS disease

Protocol for Dx

- Perform a FBS test for all patients at booking
- FBS >126: preexisting DM
- FBS 92-126: 75g OGTT immediately
- FBS <92: 75g OGTT at 24-28 weeks.

↳ Usually diagnosed at the beginning of 3rd trimester

Screening

Screen All pregnancies at 24-28 weeks

- Initial Screening** — OGTT 50g, 1hr ⇒ Blood glucose should be <135 mg/dl
- Confirmation test** — 100g, 3hr OGTT >140 mg/dl

Management

1. Control glucose levels 3 months prior to conception.
2. Diet and exercise.
3. Pharmacological therapy: Insulin.
4. Antepartum care: Ophthalmology, cardiac, endocrine and nephrology consults.
5. Timing of delivery:
 - » Well-controlled GDM without complications → Wait for spontaneous delivery.
 - » Earlier intervention is indicated → If complications arise or GDM is no longer controlled.
 - » If the fetus weighs more than 4500 g → C/S.
 - » If there isn't an indication for C-section, normal vaginal delivery

Pelvis

Pelvic Anatomy

The pelvic inlet

The transverse diameter of this plane= 13.5cm, it's the wider than the AP diameter= 11cm

the fetal head enters the pelvis in the transverse position.

The plane of greatest diameter

the fetal head rotates to the anterior position.

The plane of least diameter (Midplane)

The narrowest point= 10.5cm, the most instances of arrest of descent occur at this level.

The pelvic outlet

The site of low pelvic arrest.

Pelvic Shape

Gynecoid (Most common)

The classic female type, found in 50% of women.

It is round at the inlet, with the widest transverse diameter only slightly greater than the anteroposterior diameter.

The fetal head generally rotates into the occipito-anterior position in this shape.

Android

The typical male type of pelvis, found in less than 30% of women.

It has a triangular inlet.

The fetal head is forced to be in occipito-posterior position to conform to this narrow anterior pelvis.

Anthropoid

Found in 20% of women.

The AP diameter is much larger than the transverse diameter.

Fetal head can engage only in the AP diameter.

Platy pelloid

Described as flattened gynecoid, found in 3% of women.

It has a short AP and wide transverse diameter, giving it an Oval-shaped inlet.

The fetal head has to engage in the transverse diameter.

Fetal Head

The cranial bones at birth are: Thin, weakly ossified, easily compressible, interconnected only by membranes

↳ Allow them to overlap under pressure & to change shape to conform to the maternal pelvis, a process known as Molding.

The membrane-occupied spaces between the cranial bones are known as sutures.

The membrane-filled spaces located at the point where the sutures intersect are known as fontanelles, the most important of which are the Anterior and Posterior fontanelles

Labor

Definition

Progressive cervical effacement and dilation, resulting from regular uterine contractions that occur at least every 5 minutes and last 30 to 60 seconds each, amplitude of contractions 50-60mmHg.

- Normal occur at 40 wks
- preterm < 37 wks.

Presentation

The part of the fetus in the lower pole of the uterus overlying the pelvic brim

Types

Cephalic or vertex

Occiput/ Widest diameter is biparietal (9.5 cm)

↳ Vertex: Area between the fontanelles & bounded laterally by the parietal eminences

Face

Mentum

Breech

Sacrum/ Inter-trochanteric (10 cm)

Transverse

Shoulder/ Bi-acromial diameter (11cm)

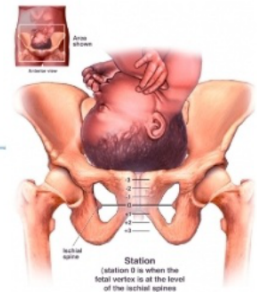
Shoulder: Olecranon process

Engagement

When the widest diameter of the presenting part of the fetus enters the pelvic inlet.

Station

Relation between the presenting part and the ischial spines of the mother.



Position

Relation of the presenting part & the mother's pelvis. In vertex presentation, the occiput is the reference point.

Occipitoanterior (OA)

- Most common position; normal.
- Occiput of fetus towards symphysis pubis
- # Any other position is considered malposition.
- Left OA (LOA) is most common.
- ↳ Left means the occiput of the fetus is against the left side of the anterior pelvis.

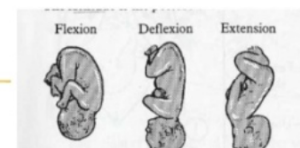
Occipitoposterior (OP)

- Most OP rotate spontaneously to OA.
- May cause prolonged second stage of labor
- Arrested labor may occur when the head does not rotate and/or descend.
- Delivery may be complicated by perineal tears or extension of an episiotomy.

Attitude

Posture of the fetus

- Normal attitude: full flexion
- Other attitudes: deflexion, extension



Lie

Describes the orientation of the long axis of the fetus to the long axis of the uterus.

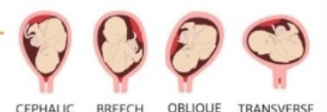
↳ Lie can be transverse, longitudinal or oblique.

- ~ Longitudinal lie, both axes are aligned.
- This is considered a normal lie.
- Both breech & cephalic have normal lies.

~ Transverse lie, the long axis of the fetus lies perpendicular to the long axis of the uterus.

~ Oblique lie, the fetal long axis is at an angle to the bony inlet.
↳ Usually is transitory and occurs during fetal conversion between other lies.

Both the transverse & oblique lies predispose to shoulder presentation.



Malpresentation

Breech (most common)

Fetal buttocks or lower extremities present into the maternal pelvis.

Etiology

Fetal

- Prematurity: Before 28 weeks, 25% of fetuses are presenting as a breech.
 - ↳ Prematurity is a major factor predisposing to breech presentation.
- Fetal structural anomalies (e.g., hydrocephaly) may restrict the ability of the fetus to present as a vertex.
- Multiple gestation.

Maternal

- Uterine anomalies (e.g., bicornuate uterus).
- Oligo/polyhydramnios.
- Contracted maternal pelvis.
- Uterine surgery.
- Pelvic tumors that obstruct the birth canal.

Placental

Placenta previa.

Classification

- » Frank (extended): 65%
- » Complete (flexed): 25%
- » Incomplete/footling: 10%

Variations of the breech presentation



Diagnosis of breech presentation

Leopold examination

The firm fetal head is palpated in the fundal region, the softer & smaller breech occupies the lower uterine segment above the symphysis pubis.

Vaginal examination

In a frank breech, the fetal buttocks, anus, sacrum, and ischial tuberosities can be palpated.

Breech presentation

By US

Management

Most practices is: Deliver all breeches by cesarean

Mother expel the fetus by her own effort with uterine contractions, never pull from below (Hands-off).

Criteria for vaginal delivery

- Gestational age should be at least 36 weeks.
- Must be frank or complete breech.
- Fetal weight should be between 2.5 & 3.8kg.
- Fetal head must be flexed.
- Must be no indication for cesarean delivery.
- Anesthetist should attend the delivery & obstetrician must be experienced

Complications

- ~ Umbilical cord prolapse
- ~ Entrapment of aftercoming head
- ~ Asphyxia
- ~ Increased risk of CTG abnormalities.
- ~ Birth trauma; forceful traction, can involve the brachial plexus (Erbs palsy).

Face (2nd MC malpresentation)

Fetal head is hyper-extended such that the fetal face (between chin and orbits) is the presenting part.

Presenting diameter is the submento-bregmatic (9.5 cm)

- Reference point for position is the fetal chin (mentum).
- Mentum is facing the symphysis pubis (mento-anterior), vaginal delivery should be expected >> Forceps, but not vacuum.
- Mentum posterior and mentum transverse must be delivered by C/S delivery.

Complications — Prolonged labor is common.

» When spontaneous vaginal delivery or forceps delivery occurs, perinatal morbidity & mortality are similar to vertex presentations.

Brow

Presenting part is between the facial orbits and anterior fontanelle resulting from the extension of the fetal head such that it is midway between flexion (vertex presentation) and hyperextension (face presentation).

Presenting diameter is the supra-occipito-mental diameter, which is the longest anterior-posterior fetal diameter (13.5 cm)

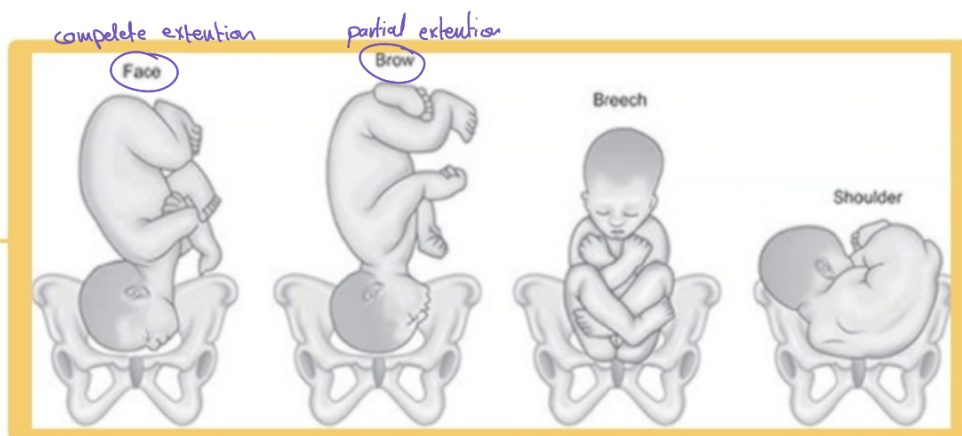
» Brow presentation is unstable.

- 50-75% will convert to either a face presentation or vertex and will subsequently deliver vaginally.
- Persistent brow presentation, the large presenting diameter makes vaginal delivery impossible and C/S is required.

Management

Compound

Shoulder



Sub-mento-bregmatic
= 9.5 cm
From below the chin to ant. fontanelle
⇒ palpation of chin is essential in diagnosis

Supra-occipito-mental (Mentovertebral)
= 13.5 cm
⇒ palpate orbit & ant. fontanelle

Sacrum
Intertrochantric
= 10 cm

* Op = Occipito-frontal = 11 cm → From root of the nose to post. fontanelle → Deflexion (Military)

* Vertex = Sub-occipitobregmatic = 9.5 cm → complete flexion ⇒ palpation of post. fontanelle =

Stages of Labor

1st stage
6-18hrs in nulliparous women
2-10hrs in multiparous women
» AVG is 10 hours



Latent phase

Cervical effacement and early dilation (up to 4cm)

The length of the latent phase is highly variable, it is longer in primi-parous women

More rapid cervical dilation occurs (from the end of latent phase until 10cm of dilation)

1cm/hr - primiparous/ nulliparous

1.2cm/hr - Multiparous

Three P's affect the active phase

- **Passage:** pelvis size & shape
- **Passenger:** fetus size & position
- **Power:** uterine contraction >200 MVU are adequate.

If the rate of dilation is LESS than this or NO cervical changes in station for 2hrs,

Evaluate the patient for:

- ~ Uterine dysfunction
- ~ Fetal malposition
- ~ Cephalo-pelvic disproportion

If there's a problem in the passenger or pelvis, then the following could be evident:

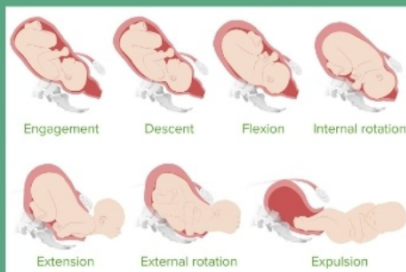
- Fetal caput
- Extensive molding
- Palpable overlapping sutures

If No cervical changes occur or no station changes occur within 2hrs in the setting of adequate uterine contractions (Active phase arrest) ⇒ C-section

⇒ partogram (when the pt. admitted to labor ward up to fully dilation)

2nd stage

- Begins after the cervix is completely dilated (10cm) to the delivery of the newborn.
- 30 mins to 3hrs in primigravid & 5 to 30 mins in multigravida.



Cardinal movements

1. Descent

Completed by the force of uterine contractions & bearing down.

2. Flexion

During descent, resistance from the cervix, pelvic walls and floor causes the fetal cervical spine to be further flexed.

3. Internal rotation.

Largest diameter of fetal head is encircled by the vulvar ring. Vertex has reached station +5.

↓ An episiotomy may aid in reducing perineal resistance.

4. Extension "Crowning"

5. External rotation

Returns the head to its original position at the time of engagement → Allow the head to align itself with the fetal back and shoulders.

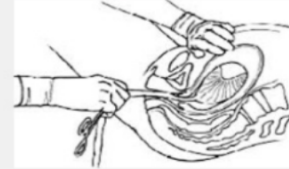
6. Expulsion

Delivery of the anterior shoulder (under symphysis pubis), followed by the posterior shoulder (over the perineal body), and finally the body of the newborn.

- 3rd stage**
- Starts immediately after the baby's delivery.
 - Delivery of the placenta occurs within 2-10 minutes.

- Signs of placental separation include:
1. A fresh show of blood from the vagina.
 2. Umbilical cord lengthening outside the vagina.
 3. Rising of the fundus of the uterus.
 4. The uterus becomes firm and globular.

After these signs have appeared $\Rightarrow$ gentle cord traction (Pull) & counter pressure between the fundus and symphysis (Push) are applied



The placenta should be examined to ensure its complete removal (no missing cotyledons) and to detect placental abnormalities.

- 4th stage**
- 4-6 hours after the delivery of the placenta and stabilization of the mother

Continuous close monitoring of the patients' vital signs and uterine blood loss $\rightarrow$ to prevent postpartum hemorrhage.

$\downarrow$ Most cases of serious postpartum hemorrhage occur during this stage

* Baby wt + placenta $\rightarrow$ AF

$\downarrow$ $\downarrow$ $\downarrow$

2-3 Kg $\frac{1}{6}$ fetus $> 5\text{cm} = 500\text{ml}$

[8-10 cm
after 38 wks]

Episiotomy

Incision in the perineum (skin, vagina, perineal muscles) to increase space

during second stage of labor

Indications

- Suspected maternal and/or fetal compromise
- Delivery needs to be expedited
- Anticipation of significant perineal and or rectal trauma

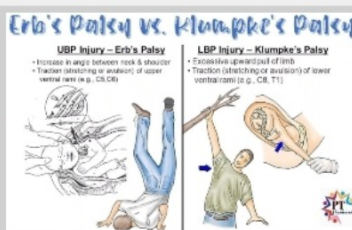
Shoulder Dystocia

Shoulder dystocia occurs when anterior, or less commonly the posterior fetal shoulder impacts on the maternal symphysis, or sacral promontory

Risk factors

1. Fetal macrosomia
2. Maternal DM / Obesity
3. Multiparity
4. Post-term gestational age
5. Short stature of the mother
6. Previous Hx of macrosomic birth or shoulder dystocia.
7. Labor induction / Prolonged labor
8. Epidural analgesia
9. Operative vaginal delivery

Complications



Erb palsy (major neonatal complication): Excessive traction on the brachial plexus (C5,C6) by the delivery attendant

Klumpke's paralysis: variety of partial palsy of the lower roots of the brachial plexus (C8, T1)

Clavicular fracture, Humeral fracture

Hypoxia, brain injury, and death

Management

Recognized at delivery by retraction of the fetal head, which is called the "turtle sign"

Maneuvers

Initial maneuver → Supra-pubic pressure
» Downward or lateral pressure with the hand over the maternal suprapubic to guide the anterior shoulder under or away from the symphysis pubis.



Others: McRoberts maneuver, Gaskin" or "all-fours", Wood maneuver

If none of maneuvers is successful, one or both clavicles must be fractured, by pressure on the clavicle directed away from the pleural cavity to prevent traumatic puncture of the lungs.

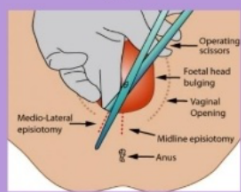
When ALL previous methods failed → Zavanelli maneuver

» Fetal head is manually returned to its pre-situation position, then slowly replaced into the vagina and then into the uterus by steady upward pressure against the head. Delivery is then accomplished by C/S.

Uterine relaxant → required.



Types



Midline

From fourchette down the perineal midline raphe towards the anal verge

Advantages — less bleeding, better healing, less pain

Disadvantages — risk of anal sphincter & anal muscles injury (incontinence).

Mediolateral

Incision at a 45 angle inferiorly from midline of the fourchette

Advantages — NO risk of anal sphincter injury

Disadvantages — more bleeding and pain, less healing

Puerperium

Period of time in which organ systems return to their pre-pregnant state, begins with the delivery of the baby and placenta.
* 6-8 weeks postpartum.

Delivery Room

Skin-to-skin contact between mother & infant benefits:

Infants are more likely to be breastfeeding at 1 and 4 months after delivery.
↳ longer overall breastfeeding duration

It maintains glucose levels better than others.

Thermal stability was significantly improved in late preterm infants, but the improvement was not clinically significant in full term infants.

Reproductive System

Uterus

- Involution occurs 10-12 days postpartum
- Uterine contractions are present (to keep venous placental sinuses closed)

Lochia (Secretions)

Superficial layers of endometrial decidua that are shed through the vagina during the 1st 3wks postpartum:

- 1st few days: Red Lochia (Rubra)
- Up to 2nd week: Pink Lochia (Serosa)
- After 2 weeks: White Lochia (Alba)

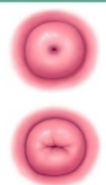
Vagina

- NEVER return to prepartum size
- Hymen is replaced by multiple tags of tissue called the carunculae hymenales (myrtiformes)

Cervix

- The external os never resumes its pregravid shape; it becomes large, transverse, stellate slit after childbirth.
- Histologically, the cervix does not return to baseline for up to 3-4 months after delivery.

Parous and nulliparous cervix



Small: Normal nulliparous cervix. The external os is a small, smooth, circular opening. Enlarged: Normal parous cervix. The external os is a large, transverse, stellate slit.

Normal puerperium (physiological changes)

Breast

- » Colostrum production starts in the latter part of pregnancy until 3 days postpartum
- » Milk production starts on the 3rd-4th day postpartum (under the effect of prolactin)

CVS

Risk of VTE is higher postpartum

Skin and hair

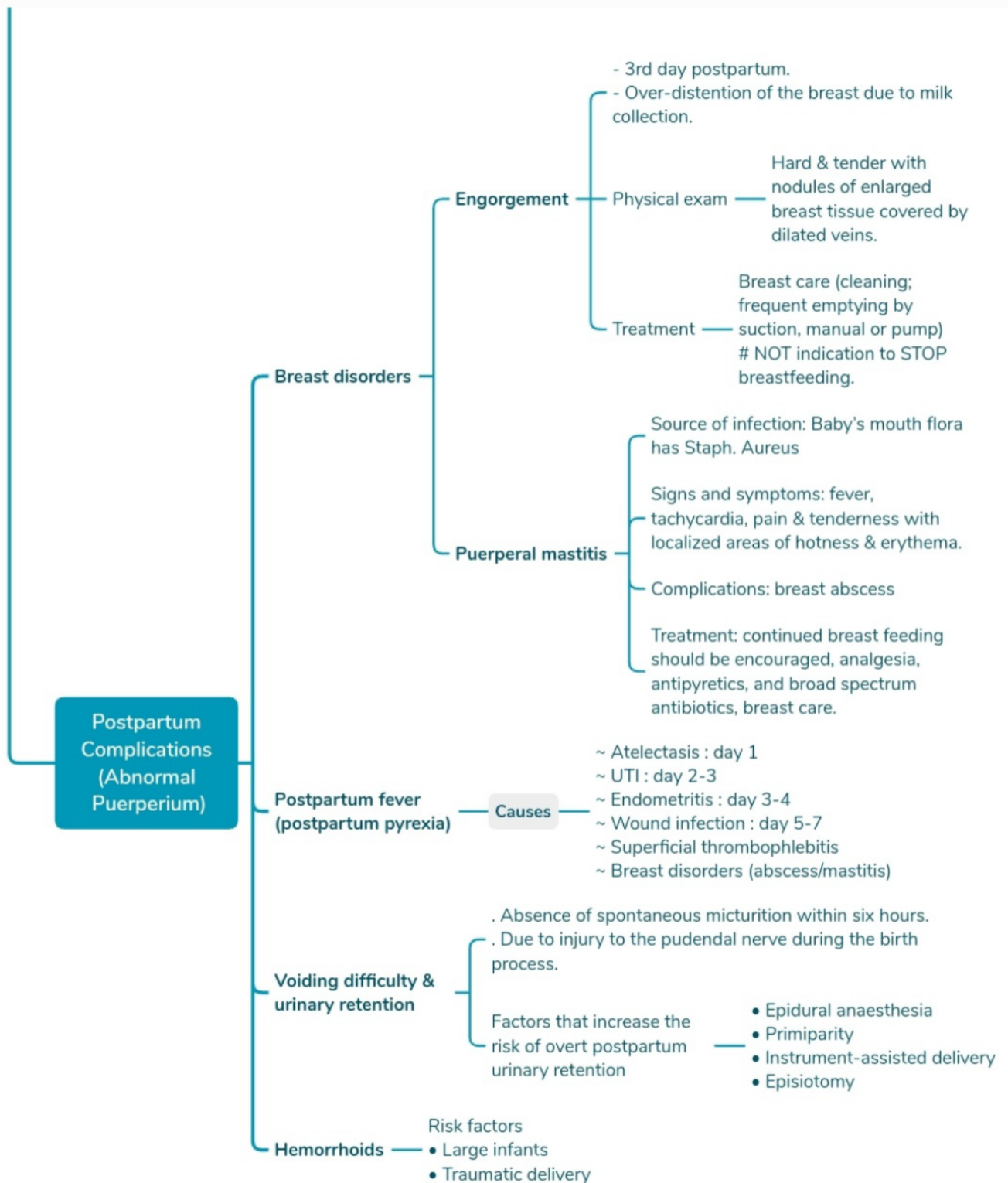
- Striae → Fade from red to silvery but are permanent.
- The abdominal skin → May remain lax if there was extensive rupture of elastic fibres.
- Chloasma → Resolves in the postpartum period.
- Telogen effluvium is the hair loss commonly noted 1-5 months after delivery, usually self-limited with the restoration of normal hair patterns by 6 to 15 months after delivery.

Weight loss

Delivery of the fetus, placenta and amniotic fluid ⇒ around 6 kgs.

- Further loss of 2-7 kgs is due to:

- Involution of the uterus
- Loss of lochial fluid
- Loss of intra- and extra-cellular fluid



Aminotic Fluid

Fluid surrounding the growing fetus in utero.

» It's produced mainly from maternal serum before 14 weeks gestation, while fetal kidneys start excreting it beyond 14 weeks.

Amniotic fluid allows

Growth, movement and development.

» Without AF, the uterus would contract and compress the fetus.

- In cases of leakage in 1st trimester, the fetus may develop structural abnormalities: facial distortion, limb reduction, abdominal wall defects secondary to uterine compression.

Important for fetal pulmonary development

» The absence of adequate AF during mid-pregnancy is associated with pulmonary hypoplasia at birth, which is often incompatible with life.

Protective role for the fetus.

» It contains anti-bacterial activity to inhibit the growth of potentially pathogenic bacteria.

Aiding dilation of the cervix, during labor and delivery.

Aminotic Fluid Index (AFI)

The volume of AF is assessed antenatally, measurements of AF present in all 4 quadrants.

• The amniotic fluid index is measured by dividing the uterus into four quadrants

• The linea nigra is used to divide the uterus into right and left halves.

• The umbilicus serves as the dividing point for the upper and lower halves.

• The transducer is kept parallel to patient's longitudinal axis and perpendicular to the floor.



AFI values range normally from 5-23, depending on the gestational age at measurements.

Single deepest pocket of AF measurement between 2 and 8.

Oligohydramnios [AFI < 5]

Causes

Most common cause is ROM

Mother (Dehydration)

Fetus [Renal anomalies, Potter syndrome (bilateral renal agenesis), Chromosomal Trisomy]

Placenta or other factors [Placental insufficiency, Post-term pregnancy, Medications like ACEIs & Indomethacin].

PE

Size of the uterus is lower than the GA, fetal movements may be reduced.

Complications (usually if occurred <24 weeks)

- Malpresentation
- Fetal asphyxia and hypoplastic lung
- Intra-uterine Growth Restriction (IUGR)
- Fetal anomalies (CHD, Potter face)
- Contractures (arthrogryposis)

+ cord compression cause fetal insufficiency in any uterus contraction

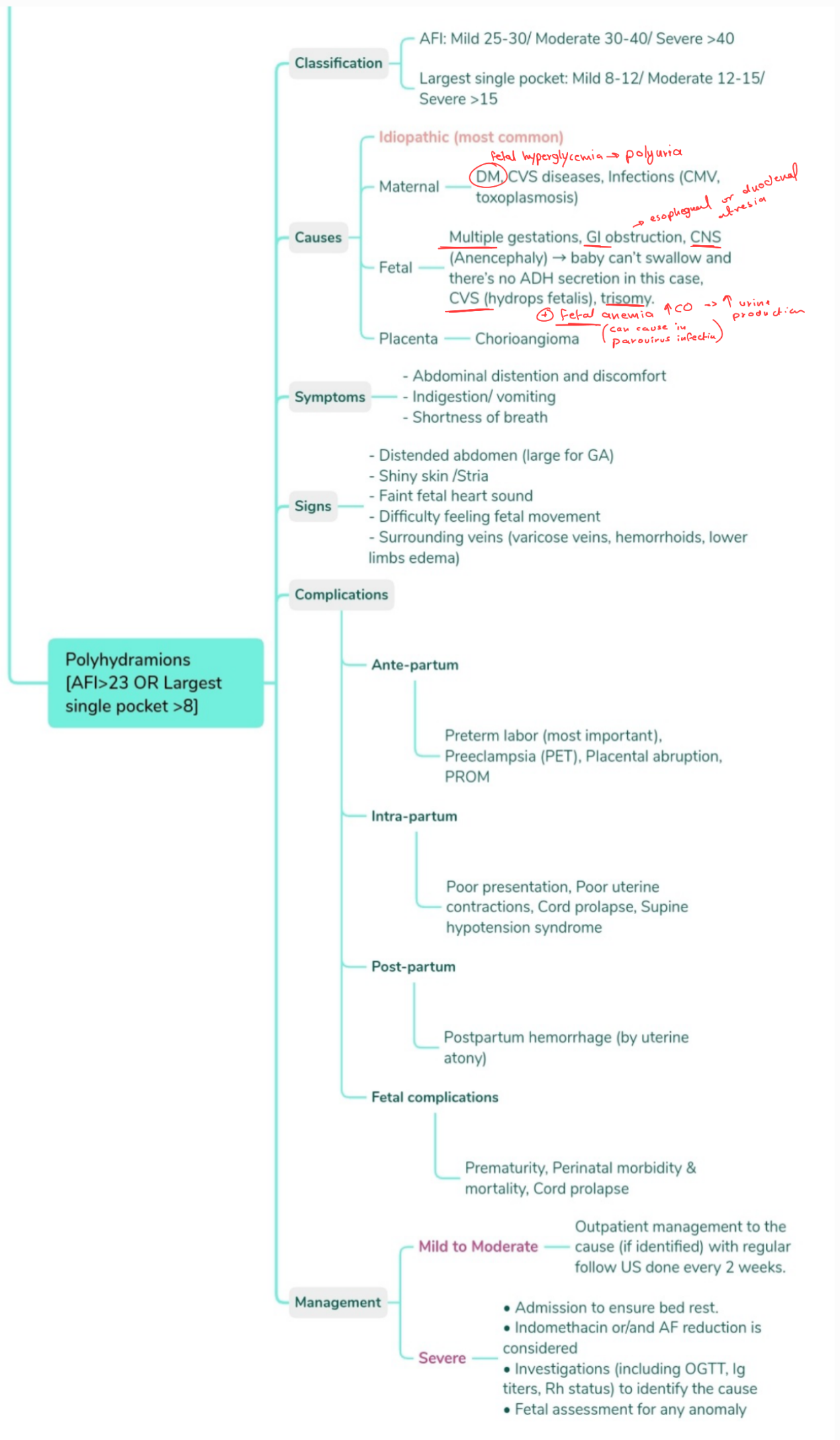
Management (Depends on GA & degree of severity)

<24wk & Severe — Termination

>24wks & Mild — Follow up for signs of chorioamnionitis, AFI, fetal movement

>24wks & Severe (shows cord compressions) — Hydration, Dexamethasone, Amnioinfusion → AF is replaced with normal saline through transcervical intrauterine pressure catheter.

limb deformity
flat face
pulmonary hypoplasia



Gestational Age

Small for GA

Estimated Fetal Weight (EFW) <10th, majority are healthy

Familial in 85% of case

Classified into

Normal (no structural abnormalities, normal liquor and normal umbilical artery doppler) >> Constitutionally small fetus achieves its normal growth potential & has a good prognosis.

Abnormal (IUGR)

Classified into

Symmetrical IUGR

- Insult on fetal growth occurs early.
- Decreased POTENTIAL Both head & body are equally reduced.
- Genetic, TORCH, Chromosomal.

Asymmetrical IUGR: M.C, (due to maternal or placental causes)

- Insult occurs late in pregnancy.
- Decreased SUBSTRATES the body (abdominal circumference) is small while the head (head circumference) is preserved.
- Placental insufficiency, Malnutrition, PIH.

Causes

Fetal

- Trisomies, Turner
- Infections (TORCH, syphilis, malaria, varicella)
- Polyhydramnios, Oligohydramnios
- Gastroschisis, Cardiac defects
- Uterine over-crowding (due to fibroids or multiple gestations) → The baby has decreased potential to grow, increase feeding will NOT help.

Maternal

- ~ ↑ Maternal age
- ~ HTN, Preeclampsia, DM
- ~ CVD, RS, Renal, SLE
- ~ Sickle cell anemia, Malnutrition (low BMI <19)
- ~ Infections, Smoking, Alcohol
- ~ Drugs: Chemo-radiotherapy, teratogenicity drugs (antiepileptic, Beta-blockers)

Placental

- Placental mosaicism
- Placental dysfunction ⇒ Abnormal vascular development at the placenta, continuous, started immediately after conception.
- ↳ Non-branching Angiogenesis (reduce gas exchange cause chronic hypoxia)
- Placenta previa/ Placental abruption/ Thrombosis/ Calcifications/ TTTs (twin-twin transfusion syndrome)

Complications

- Respiratory: meconium aspiration, asphyxia (RDS - Transient tachypnea of newborn)
- Electrolytes/ blood: ^{hypoxic environment} Polycythemia, increased Bilirubin, decreased Calcium, decreased Glucose
- Life manifestations: mental retardation, fetal distress, Intrauterine fetal death

premature or low birth wt complications:

- Hypothermia
- Hypoglycemia
- Hyperbilirubinemia
- Respiratory distress (Distress of surfactant)
- Intracranial hemorrhage
- Persistent fetal circulation (↑ pulmonary vascular resistance) = abnormal pulmonary vessels (small thick walls) → excessive vasoconstriction
- ↓ immune function
- Necrotizing enterocolitis
- Blood shunt right → left [Hypoxemia]

* SIDS

sudden death < 1yr

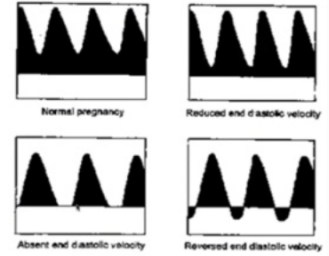
RF

- 1- Stomach sleep
- 2- Maternal smoking
- 3- very young maternal age < 20
- 4- Bed sharing
- 5- prematurity / low birth wt.

Investigations

SERIAL U/S (the most effective way of detecting growth restriction) for uterine artery → Reflection at the downstream (forward) resistance of the maternal side of the placenta

- Low resistance ⇒ good placentation
- High resistance ⇒ poor placentation, absent or reverse of end-diastolic flow



hCG, α -fetoprotein, inhibin A & PAPP-A (low)

Clinical assessment

- Maternal weight gain
- Symphysis Fundal Height, sensitivity is poor
- Serial US Biometry, the most sensitive ⇒ biparietal diameter, head & abdominal circumference, femoral length.
- Liquor volume; decrease renal perfusion & decrease renal output, degree of reduction is reflected by the degree of hypoxaemia.
- NST (Non-Stress Test for cardio activity)
- BPP (Bi-Physiological Profile)
- Umbilical artery doppler
- MCA & ductus venosus doppler

Management

- Lifestyle changes (nutrition, rest, stopping smoking)
- Baby Aspirin (for prevention)
- Fetal monitoring (weekly), NST, Biometry, BPP, Biometric measurement, Liquor
- » Reversed Flow of uterine artery ⇒ indication for delivery by C/S
- » Reduce growth velocity, reduce liquor, abnormal umbilical artery waveform after 34 weeks ⇒ delivery is indicated / before 34 weeks ⇒ steroids should be given.
- » Before 34 weeks and has the signs of decompensation on MCA & ductus venosus doppler ⇒ Delivery and do not wait for CTG

Large for GA

EFW >90-95th percentile for a given GA

Risk factors

- Gestational DM, overt DM
- Prolonged gestation (postdate pregnancy)
- Increased BMI (obesity), increased weight gain during pregnancy
- Multiparity
- Male fetus

Complications

- Maternal**
 - Operative vaginal delivery
 - Perineal lacerations, pelvic floor injury leading to urinary incontinence and pelvic organ prolapse
 - Emergency C/S
 - Postpartum hemorrhage (due to atony)
- Fetal**
 - Shoulder dystocia
 - Asphyxia
 - Birth injury
- Neonatal**
 - NICU admission
 - Erbs palsy
 - Hypoglycemia

Management

- Elective C/S if EFW >4.5kg in diabetic mother or >5kg in non-diabetics
- Early induction

Rh Isoimmunization

Rh(D) antigen is expressed by 30 days of gestation (fetal age = 30 days, 44 days from LMP = 6 wks GA).
» 90% of cases of Rh alloimmunization are due to antibodies to the D antigen, and this is the only form of alloimmunization that can be prevented with Rh immune globulin prophylaxis.

When RhD-negative patients are exposed to the RhD antigen, they may become sensitized (start to recognize D antigen as immunogenic and want to destroy it)
↳ 2 exposures to the RhD antigen are required to produce significant sensitization, unless the first exposure is massive.

» 1st exposure leads to primary sensitization, production of IgM antibodies (which can NOT cross the placenta) for a short period of time.

» 2nd exposure: Rapid production of immunoglobulins (IgG antibodies) that are capable of crossing the placenta.

↳ If the fetus has the RhD antigen, these Abs will coat the fetal RBCs, causing them to be destroyed, or hemolyzed, in the spleen
⇒ Anemia & Hyperbilirubinemia

Causes of sensitization

Placental leak of fetal RBCs into the maternal circulation (feto-maternal hemorrhage) during pregnancy.

- Incidence and size of transplacental (fetomaternal) hemorrhages increase with advancing GA.

↳ Largest hemorrhages occurring at delivery.

At the time of delivery, antibodies appear either during postpartum period or following exposure to the antigen in the next pregnancy.

Prophylaxis AFTER antepartum events

- Miscarriage or induced abortion
- Invasive prenatal diagnostic or therapeutic procedures.
- Blunt abdominal trauma.
- External cephalic version (induce Cephalic presentation by the doctor).
- Ectopic pregnancy.
- Fetal death in 2nd or 3rd trimester ⇒ fetal demise.
- Antepartum hemorrhage in 2nd or 3rd trimester.
- Hydatidiform mole only if partial, as a complete mole does not contain fetal red cells.

Window for giving Anti-D — 72 hrs

Consequences of the destruction of the fetal RBCs

Hemolysis is mild

Fetus can compensate by increasing the rate of erythropoiesis.

Hemolysis is severe

Profound fetal anemia, resulting in extramedullary hematopoiesis, portal hypertension, hypoalbuminemia, hyperbilirubinemia, and heart failure (hydrops fetalis), intrauterine fetal death.

↳ High bilirubin levels can damage the CNS and lead to neonatal encephalopathy and kernicterus (deposition in basal ganglia).

The disease gets worse in subsequent pregnancies in Rh-D antigen alloimmunization

Approach

All D-negative pregnant women should undergo an antibody screen at the first prenatal visit of each pregnancy.

↳ In RhD-negative patients whose anti-D antibody titers are positive (RhD sensitized), & the father was RhD negative, we won't worry about the baby as it's going to be RhD negative as well.

The diagnosis of Rh(D) alloimmunization is based upon detection of anti-Rh(D) antibody in maternal serum.

» In patients with a positive titer less than 1:16, repeat titers should be obtained every 2 to 4 weeks.

If the titer rises to 1:16 or greater, a detailed ultrasound to detect hydrops and Doppler studies of the MCA are indicated.

Prevent alloimmunization

Treat the mother with anti-D Ig (RhoGAM) during & after each pregnancy to prevent anti-D IgG formation.

↳ So we give her the Anti-D antibody before her body forms it.

When a mother is given a dose of anti-D IgG, the antibodies bind to the fetal RBCs that have the D antigen on them and clear them from the maternal circulation.

The goal is to prevent the mother's immune system from recognizing the presence of the D antigen forming antibodies against it.

The routine prophylactic administration of Rh immune globulin at 28 weeks of gestation

» A single 300 microgram (1500iu) dose of anti-D immunoglobulin at 28 wks

OR

» 100 micrograms (500 iu) of anti-D at 28 and 34wks of gestation

** Indirect Coombs Test; antibody screening for pregnant should be repeated before any dosing (we use it also to confirm the diagnosis of Rh immunization)

The incidence of 20 to 30mL of fetal blood entering the maternal circulation at delivery is common at delivery needs → Effective prophylaxis from a single 300 microgram (1500iu) dose of anti-D immunoglobulin.

No need for prophylaxis if

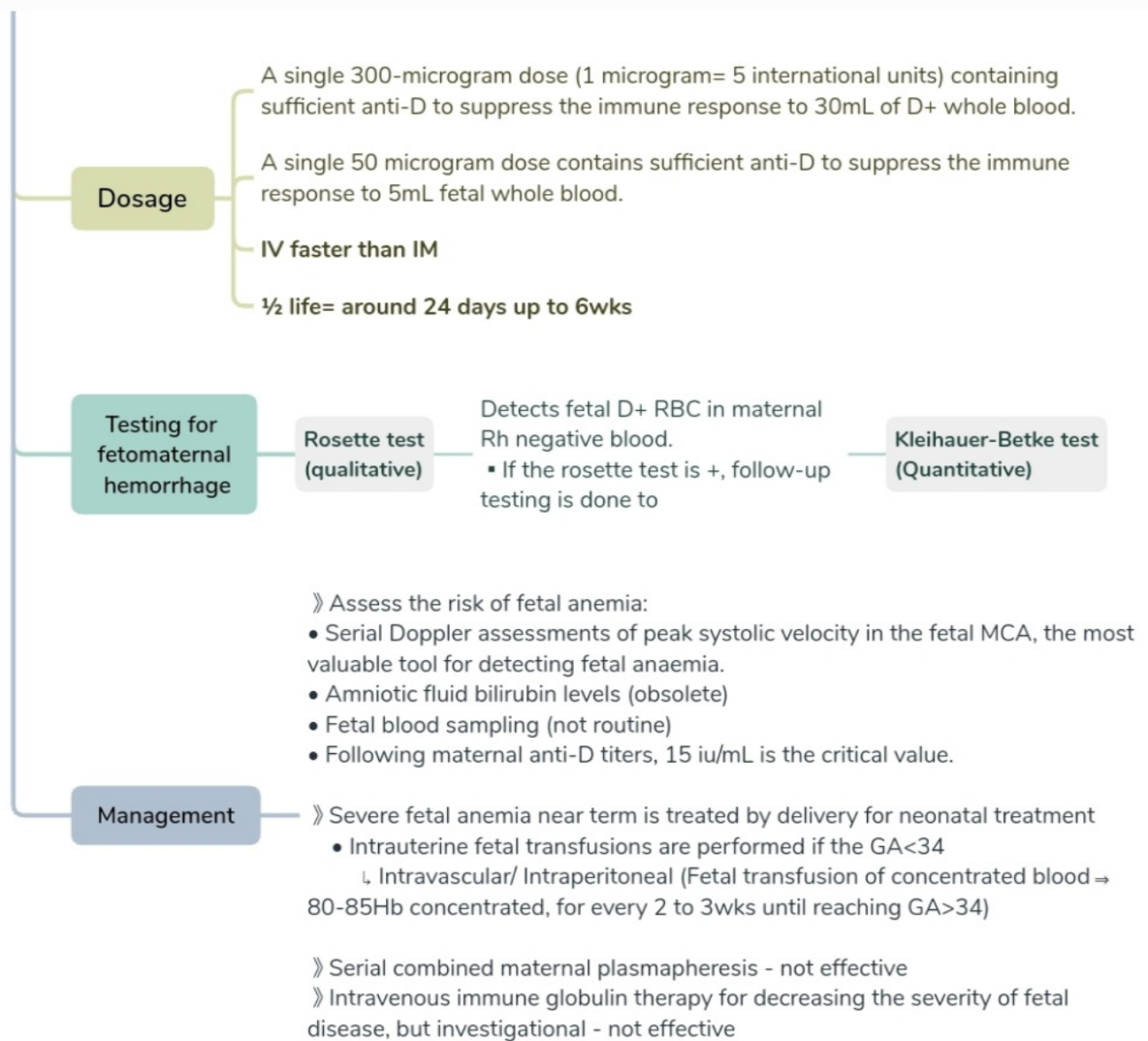
~ The biologic father of the baby is known with certainty to be D-negative

~ Cell-free DNA (cfDNA) results on maternal plasma suggest that the fetus is D-negative.

~ Fetal blood sampling confirmed the fetus to be Rh -ve.

~ Amniocentesis/ CVS confirmed the fetus to be Rh -ve.

~ Complete mole



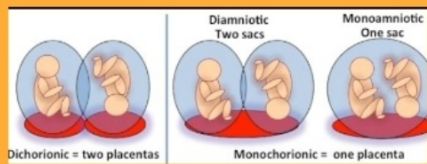
Multiple pregnancies

Definition

Development of more than 1 fetus inside the uterus.

No. of twin pregnancies increased due to increased use of assisted reproduction techniques.

Types



Monozygotic (Identical= SAME sex & genetic): 1/3

30% Dichorionic diamniotic DCDA (2 placenta, 2 amniotic sacs) ⇒ Oocyte deviation in (0-3) DAYS

69% Monochorionic diamniotic MCDA (1 placenta, 2 amniotic sacs) ⇒ (4-8) DAYS

Monochorionic monoamniotic MCMA (1 placenta, 1 amniotic sac) ⇒ (9-12) DAYS

Conjoined twins (AFTER DAY 12)

Dizygotic (ALWAYS dichorionic diamniotic-DCDA) → May or may not be the same sex, different genetics: 2/3

Risk factors/ Causes

- 1- Race (more common in blacks)
- 2- Increases with age and parity
- 3- Family Hx
- 4- Ovulation induction and conception after cessation of contraception

Symptoms

- Exaggerated pregnancy symptoms
- Increased fetal activity
- Uterus size more than date of pregnancy.

Lab findings

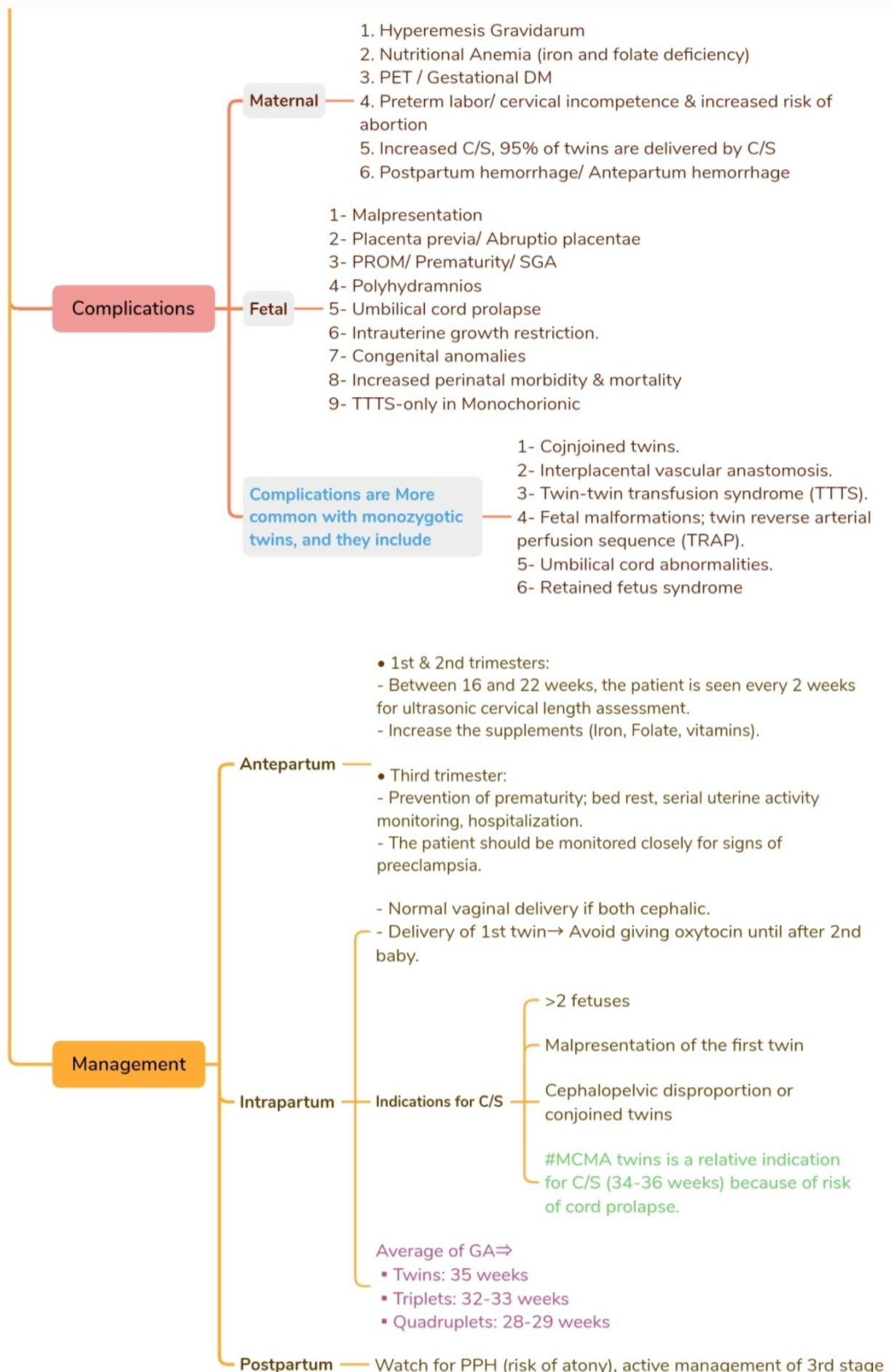
1. Increased B-hCG
2. Increased Maternal Serum α-Feto protien

U/S findings

~ Can be made as early as 6 weeks' gestation.
~ For determination of zygosity

- T- sign seen in Monochorionic
- Lambda (λ) sign seen in Dichorionic

- Thick amnion-chorionic septum is suggestive of Dizygotic twins.
- Thin dividing membrane is suggestive of monochorionic twins.



Antepartum hemorrhage

Definition

Vaginal bleeding occurs after 24 weeks of gestation & ^{til the birth of the baby} prior to the birth of the baby.
- Incidence: 5% of all pregnancies.

Blood loss

- Spotting
- Minor hemorrhage: blood loss less than 50 ml
- Major hemorrhage: blood loss of 50–1000 ml, with NO signs of clinical shock
- Massive hemorrhage: blood loss greater than 1000ml and/or signs of clinical shock.

Causes

Insertion of the placenta, partially or fully, in the lower segment of the uterus. $\Rightarrow DS < 2\text{ cm}$

Grades



Placental edge is in the lower uterine segment but doesn't reach the internal os (low implantation)



Placental edge reaches the internal os but does not cover it. (marginal)



Placenta covers the internal os when it is close and is asymmetrically situated (partial).



Placenta covers the internal os and is centrally situated (complete)

Placenta previa

Risk factors

- Previous placenta previa. / prior C/S
- Deficient endometrium because of uterine scar, endometritis, manual removal of placenta, curettage, sub mucous fibroid.
- Multiparity / Multiple pregnancies.
- Advanced maternal age >40yrs.
- Smoking.
- Uterine anomaly.
- Assisted conception

Clinical presentation

Bleeding, usually mild, presents at around 30 wks of gestation (70% painless). $\Rightarrow$ or Asymptomatic.

Diagnosis

- Bleeding with a soft uterus.
- High presenting part & fetal malpresentation (breech/transverse/oblique).
- Normal fetal heart rate (unless there is severe bleeding or associated abruption).

U/S: transabdominal scan is 95% accurate, while the transvaginal scan is 100% mainly for the placental edge in posterior previa

Vaginal examination is contraindicated.

Complications

- Preterm delivery.
- Preterm premature rupture of membranes.
- IUGR (repeated bleeding).
- Malpresentation; breech, oblique, transverse.
- Fetal abnormalities.
- Maternal death due to complications of the CS or uncontrolled haemorrhage from the placental bed.

↳ DIC is due to massive bleeding or associated abruption.

- Morbidly adherent placenta:

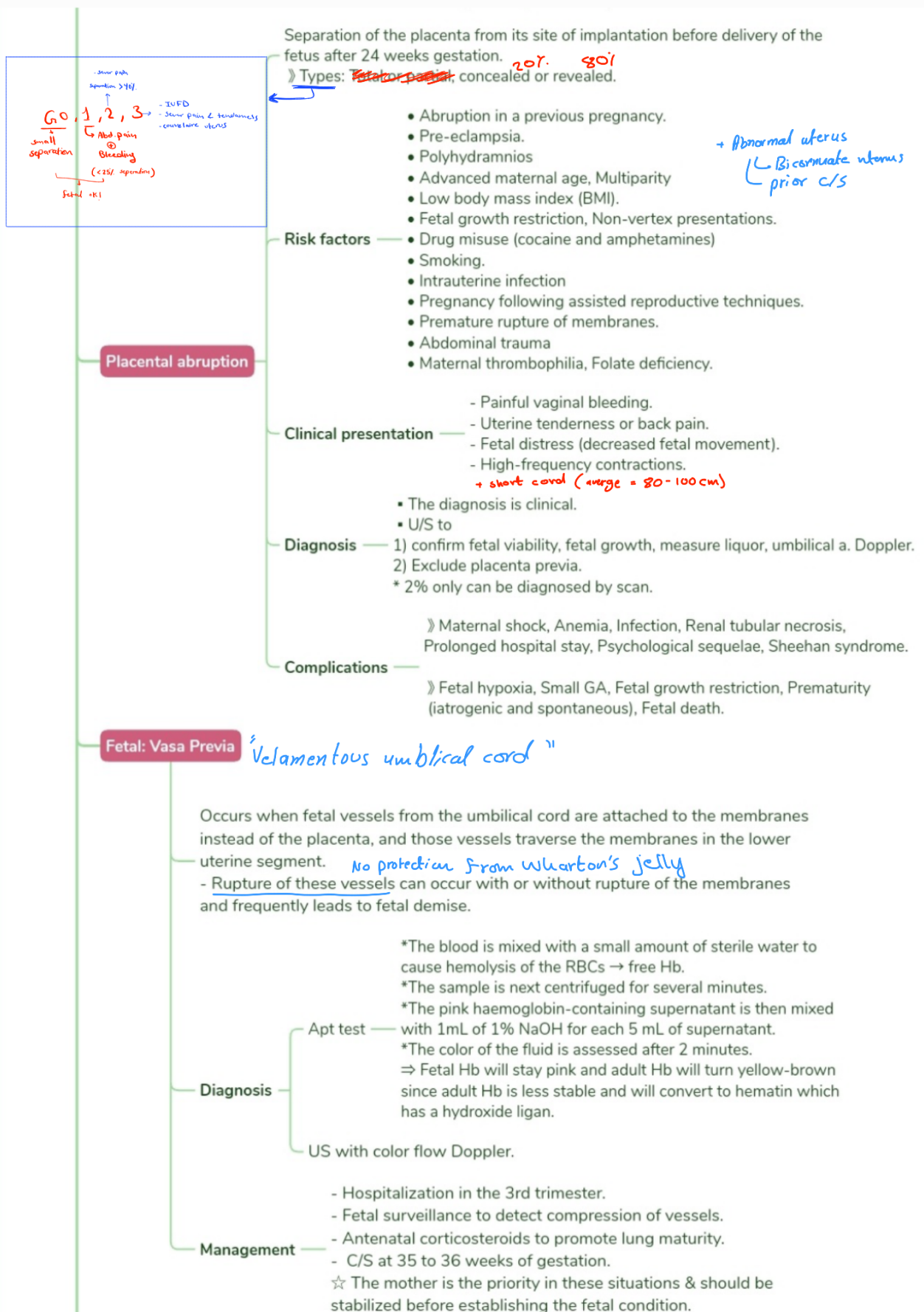
- * Placenta accreta (reaching the myometrium but not invading it)
- * Increta (invading the myometrium)
- * Percreta (Reaching the serosa) $\rightarrow$ bladder rectum

presenting with
no bleeding

RF - C/S
- uterine surgery
- D&C

Placenta "chorionic villous"
attached to myometrium
instead of decidua

∴ Rupture maternal vessels in decidua basalis → Blood separate decidua from uterus



Uterine: Rupture

Complete separation of uterine musculature through all of its layers with all or part of the fetus being outside the uterine cavity (during 1st stage or 2nd stage of labor).
» It can be spontaneous or traumatic or due to previous uterine scar

Previous uterine incisions, the rate is higher with
- Classical & T-shape uterine incisions in comparison to low vertical & transverse incisions
- Repeated CS.

↳ Patients with a prior uterine scar should be advised to come to the hospital for evaluation of ⇒ new onset contractions, abdominal pain, or vaginal bleeding.

Risk factors

Labor complications

1. Cephalopodvic disproportion.
2. Abnormal presentation.
3. Unusual fetal enlargement (hydrocephalus).

Delivery complications

1. Difficult forceps.
2. Breech extraction.
3. Internal podalic version.

Trauma / High parity

Clinical presentation

- Sudden onset of acute severe abdominal pain with some vaginal bleeding.
- Cessation of contractions & easily palpable fetal parts.
- Profound maternal tachycardia and hypotension
- Absence/ deterioration of fetal heart rate & loss of station of the fetal head from the birth canal.

Prognosis

- Fetal death 50-75%, common with extrusion.
- Maternal mortality is high if not diagnosed & managed promptly.
- Maternal morbidity: haemorrhage & infection.

Management

- Stabilization of maternal hemodynamics.
- Prompt C/S with either repair of the uterine defect or hysterectomy (mainly).
- Antibiotics.

Non-obstetric

- Cervical bleeding: cervicitis, cervical neoplasm, cervical polyp.
- Vagina bleeding: Intercourse trauma, neoplasm.

Bleeding that may be confused with vaginal bleeding

- GI bleed: hemorrhoids, IBS.
- Urinary tract bleed: Stone, cystitis, UTI.

Bleeding Disorders

- Congenital (Von-Willebrand's disease)
- Acquired (DIC)

Management

- Women presenting with spotting, no longer bleeding, placenta praevia has been excluded — Go home
- All women with APH heavier than spotting and women with ongoing bleeding — Remain in the hospital at least until the bleeding has stopped.
- Clinicians should offer a single course of antenatal corticosteroids to women between 24- 34 weeks at risk of preterm birth. → depend on amount of bleeding
- Tocolysis should NOT be used to delay delivery in a woman presenting with a major APH, haemodynamically unstable, or fetal compromise.
- Ergometrine-oxytocin (Syntometrine) to manage 3rd stage of labour in women with APH resulting from placental abruption or placenta praevia in the absence of hypertension.

Labour and delivery

- If fetal death is diagnosed → vaginal birth, not with placenta praevia.
- In women presenting with APH before 37 wks, no maternal or fetal compromise and bleeding has settled → No evidence to support elective premature delivery.
- If the woman presents after 37, a minor or major APH → vaginal delivery, but not in placenta praevia or fetal is compromised (C/S)

Postpartum Hemorrhage (PPH)

Blood loss >500cc in vaginal delivery & >1000 cc in C-section

Primary (early): within 24 hours of delivery

The most common cause is uterine atony

2nd most common cause is Traumatic delivery

Secondary (late): after 24 hours of delivery

Most common cause is infection

① Acute endometritis
- Bacterial (polymicrobial)
- After C/S
- Clindamycin + gentamicin
② RPOC → cause endometritis

The most common cause of death in developing countries

Causes of Primary PPH

Tone [Uterine Atony]

Most common cause (50%)

Risk factors

- Overworked uterus: Rapid labor, Increased oxytocin, Prolonged labor
- Overdistended uterus: Multiple pregnancy, Polyhydramnios, Macrosomia
- Infected uterus: Chorioamnionitis
- Relaxed uterus (drugs): MgSO₄, B-adrenergic agonists, Halothane

Clinical presentation

Doughy uterus, above umbilicus

Management

- Uterine massage (1st step)
- Uterotonics: Oxytocin, Methergine, PG F_{2α} (Hemabate), Cytotec (Misoprostol)
- B-lynch suture → if massage & drugs failed

Trauma [Genital laceration]

Risk factors

Traumatic or Operative vaginal delivery

Clinical presentation

Lacerations & contracted uterus (Rule-out Atony)

Management

Surgical repair (suturing)

Tissue [Retained products of conception]

Risk factors

Accessory placental lobe/ Placenta accrete

Physical examination

Missing cotyledons & contracted uterus

Management

Manual removal of placenta OR curettage by US guidance

Thrombosis [Obstetric DIC]

Risk factors

- Abruptio placenta (common)
- Severe PET
- Amniotic fluid embolus
- Fetal demise

Physical examination

Generalized Oozing & Petechiae

Management

Remove POC, ICU, blood products

Turned over [Uterine inversion]

Risk factors

- Myometrial weakness (MC)
- Previous Hx of inversion
- Fundal placenta, placenta accrete
- Too much traction of the cord & fundal pressure.

Physical examination

Beefy bleeding mass (fundus coming through the vagina), uterus not palpable

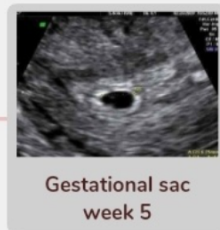
Management

Replace the uterus into the normal place (by elevating the vaginal fornices) then give IV oxytocin.

Unexplained hemorrhage

Need Ligation of the uterine vessels, internal iliac OR hysterectomy.

Problems in early pregnancy



Pregnancy of unknown location (PUL)

No evidence of pregnancy can be seen either inside or outside the uterus

Ectopic pregnancy

Implantation of a pregnancy outside the uterine cavity

Miscarriage

Spontaneous loss of a pregnancy at or before 24 weeks

This could occur during 1st or 2nd trimester ⇒ Most common in 1st trimester

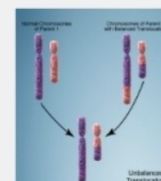
1) Increasing maternal & paternal age → At age of 20 risk 20%, 45 years 93% risk.

2) Previous miscarriages 10% after 3 losses.

3) Poorly controlled DM / Thyroid disease / PCOS / Smoking / Thrombophilia / Antiphospholipid syndrome

• If Antiphospholipid syndrome - low dose Aspirin & LMWH

4) Chromosomal: trisomy, monosomy, triploidy
↳ Balanced translocation of one partner that causes unbalanced translocation & miscarriage



Causes

5) Bacterial vaginosis in 1st trimester: varicella, rubella (TORCH)

6) Alcohol, drugs (MTX, antiepileptic), smoking, cocaine

7) • Uterine abnormalities [Didelphys uterus, Bicornuate uterus]
• Fibroid → we have to check the adequacy of the uterine cavity
• Intra-uterine adhesions



• Uterine abnormalities → open surgery might lead to a post. op infertility.

~ In any uterine abnormalities, or fibroid ⇒ We have to look for urinary (Kidneys) congenital anomalies.

8) Cervical incompetence
↳ Needs to cervical cerclage at the end of 1st trimester



Cervical length BEFORE 24wks = 25mm or less

check for:

- Factor V

- protein C

- protein S

- B2 Micr

- Lupus A.C

- Anti Phospholipid Syndrome

- Anti throm

Indication for thrombophilia

① Recurrent abortion

② Previous pregnancy at IUGR

③ current pregnancy the heart at fetus

appears then

becomes absent

uterine thickness
B-hCG plateau
on evacuation - NO
chorionic villus
treated by MTX

Classifications

Threatened miscarriage

- Vaginal bleeding in the presence of viable pregnancy, before 24 weeks.
- No abdominal pain.
- 50-75% will continue the pregnancy.

US ⇒ Fetal heart present / Internal OS is CLOSED

Treatment ⇒ Reassurance & Observation
 ↳ No need for admission.
 # Use of Progesterone supplementation.

Inevitable miscarriage

↓
 with ruptured membrane

- Threatened + Increase uterine contractility & vaginal bleeding & lower abd. pain in the presence of OPEN cervical os and pregnancy tissue still present.

Treatment ⇒ Evacuation of the product of conception → Bec at risk of chorioamnionitis + fetal death

long atrophy
 fetal death
 fetal deformity
 cord compression

Incomplete miscarriage

- Vaginal bleeding & abdominal pain where product is expelled incompletely.

US ⇒ Showed retained product of conception >15mm in diameter (with or w/o gestational sac)

Treatment ⇒ Evacuation OR Medical treatment (Misoprostol).
 ↳ complications: Renal & hepatic failure/ DIC / Death.

Complete miscarriage

- Products of conception are expelled completely.
- No significant abd. pain.
- Cessation of bleeding and a closed cervix following a miscarriage

US ⇒ Empty uterus or retained products of conception <15 mm in diameter, falling HCG, where an intrauterine pregnancy was previously confirmed

Treatment ⇒ Reassurance

Missed miscarriage or early fetal demise

- Blighted ovum OR dead fetus.
- Minimal or no symptoms
- Cervix is closed.
- HCG is dropping
- Mean gestational sac diameter >25mm with NO obvious yolk sac or fetal pole
- CRL >7 mm with no fetal heart activity

US ⇒ Blighted ovum, No fetal heart activity, hydropic changes

Treatment ⇒ Conservative, Misoprostol, Surgical.

Septic miscarriage

- Infection of the uterus with ANY TYPE of miscarriage (most common with incomplete or missed)

- ↳ E. coli / Strep Fecalis / Klebsiella.
- Fever, Purulent offensive vaginal discharge, Tachycardia
- PE: Tender uterus → Bilateral [ectopic cause pelvic pain] unilateral
- Complications: Renal failure, DIC

Treatment ⇒ Abx for 12hr then Evacuation

Spontaneous Miscarriage

- >12 wks → Anti-D should be given to all non-sensitized RhD-negative.
- Any instrumentation regardless to GA give anti-D.

Recurrent miscarriage

3 or more recurrent miscarriages, either in consecutive or not.

- Hx**
 - Detailed hx of the last menstrual period (regular/ length/ contraception).
 - Symptoms: vaginal bleeding, pain, diarrhea, urinary symptoms, passage of tissues or vesicles.
 - Past obstetric & gyne hx./ Past medical hx./ Medications
- PE**
 - » General → vital signs, level of consciousness.
 - » Abdominal palpation → masses, distention, pain
 - » Vaginal examination
 - » Tissue → histopathology
- Diagnostic tools** — **US**
 - To exclude causes
 - Mean sac diameter = 8 mm w/o yolk sac, 16 mm w/o embryo.
 - CRL 5mm w/o fetal heart activity.
 - #Discrepancy between US & GA we have to re-scan in 7 days

Entopic = Methotrexate
outside the uterus
in the uterus

6 for viable pregnancy

Medical Treatment — **Misoprostol**

- Mifepristone → Anti-progesterone
- prevent implantation.

- PG analogue, equally effective orally & vaginally.
 - ↳ Vaginally carries less GI side effects.
- Binds to the myometrial cells causing strong myometrial contractions leading to the expulsion of tissues.
- Ripening & dilatation of the cervix.

Management

- Evacuation of the returned product of conception (ERPC)

- When:
1. Persistent excessive bleeding
 2. Hemodynamic compromise
 3. Infected retained products
 - 4) Suspicion of gestational trophoblastic disease
 - 5) pt. prefers it.

Surgical

- Lithotomy position
- The cervix was grasped by vulsella & dilated by Hegar (8-10mm)
- Suction curette (reduce blood loss)
- Oxytocin (for hemostasis)
- ~ Products should be sent for histopathology to exclude ectopic & GTD
- ~ Psychological support

Complications

Uterine perforation

Needs hysteroscopy & laparoscopy to assess the injury + Admitted for observation + IV abx 24-48hrs

Blood transfusion

Repeated evacuation

Infection

Cervical trauma (rare)

PROM

Definitions

- Premature ROM: Rupture of membranes before the onset of labor. *and after the age of viability (24w)*
- Preterm ^PROM: Rupture of membranes before onset of uterine contractions AND before 37 weeks' gestation
- Prolonged ROM: Rupture of membranes that occurs >18 hours before the onset of uterine contractions in term or preterm pregnancies.

Risk factors

- 1- Previous Hx of PROM (recurrence 20%)
 - 2- BIG uterus (multiple gestations – polyhydramnios – macrosomia)
 - 3- Abnormal membrane physiology
 - 4- Cervical incompetence
 - 5- Antepartum haemorrhage
 - 6- Infection / UTI
 - 7- Smoking / nutritional def. (zinc – vit.C)
 - 8- Trauma/ intercourse / iatrogenic
- due to ↑ PGs cause erosion in amniotic sac.*

Complications

- 1- Oligohydramnios
- 2- Chorioamnionitis; possibly leading to Preterm labor and birth, Fetal distress, Stillbirth, Sepsis
- 3- Abruption of the placenta
- 4- Mal-presentation
- 5- Umbilical cord prolapse or injury
- 6- Pulmonary hypertension, pulmonary hypoplasia, ARDS (newborn)
- 7- Postpartum infection
- 8- Endometritis

Diagnosis

Clinical presentation

Sudden “gush” of pale yellow or clear fluid from the vagina reaching thighs.

Sterile speculum examination

Positive pool: amniotic fluid exiting the cervix and pooling in the vaginal fornix

» Litmus test or Nitrazine test: test strips turn Blue

↳ Serve as pH indicators: Amniotic fluid is alkaline and will turn test paper blue. Vaginal secretions are normally acidic and turn test paper red.

+ve ferning
» Positive ~~fer~~ test: fern pattern on glass slide. *→ Na⁺ Cl⁻ in liquor*

» Positive IGF1 appears in the cervix.

» Positive placental α-microglobulin-1 (PAMG-1) in cervicovaginal fluid

» Tampon test / Dye amniocentesis (invasive)

US

Amniotic fluid index (AFI) decreased.

use as a follow up

DDx *pass of urine (acidic)*
excessive normal discharge (acidic)
- liquor (Alkaline)

Management

-Abx.

- Monitor for signs of intraamniotic infection (body temperature, uterine tenderness, WBC count)
- Perform fetal heart rate monitoring to assess for nonreassuring fetal status
- Consider intrapartum risk factors and GBS screening and prophylaxis, depending on whether previous antenatal GBS screening has been performed.

■ Never do PV

-steroids: effective to delivery baby after 24hr of second dose.

contraindicated to give tocolytics in PROM! bcs may mask subclinical potentials

Unstable pt.

- Prompt delivery in:
 - ↳ Patients with signs of intra-amniotic infection, abruptio placentae, cord prolapse
 - ↳ Signs of fetal distress (nonreassuring fetal heart rate)
- Collect cervical cultures & give empiric antibiotic therapy: Ampicillin and gentamicin.

GA: ≥ 37 (term)

- ↳ Delivery by induction of labor.
- ↳ Expectant management (bed rest, pelvic rest) for up to 12–24hrs.

GA: 34 (late-preterm)

- ↳ Expectant management & induction of labor are both reasonable options.
- ↳ Induction of fetal lung maturity: single-course of antenatal corticosteroids if there is no evidence of chorioamnionitis & delivery is anticipated in >24 hrs and <7 days

GA: 24 - 33

- ↳ Expectant management
- ↳ Prophylactic antibiotics: Ampicillin PLUS Erythromycin
- ↳ Single-course of antenatal corticosteroids (betamethasone or dexamethasone)
 - ↳ Tocolysis can be used to delay delivery for up to 48hrs so that antenatal corticosteroids can be administered
 - ↳ Magnesium sulfate (fetal neuroprotection) if delivery is anticipated

GA $< 23-24$

- ↳ Fetal outcome is generally poor
- ↳ Expectant management
- ↳ Same approach as for pregnant women at 24–33 wks

Stable pt.

Tocolysis

Aims of tocolysis

- Allow lung maturity
- Reduces the risk of complications associated with preterm delivery (prematurity)
- Those needing to transfer to a hospital with NICU

Contraindicated in

- Chorioamnionitis
- Advanced labor (cervical dilation > 4 cm)
- Non-reassuring fetal signs
- Abruptio placentae, or risk of cord prolapse.

Tocolytic agents

β_2 agonist $\rightarrow \uparrow cAMP$
Beta-adrenergic agonist (Terbutaline, Ritodrine): not used any more (many maternal side effects)

Magnesium Sulfate: $MgSO_4$

Prostaglandin synthase inhibitor (Indomethacin)

Side effects: Premature closure of ductus arteriosus

Oxytocin Antagonist (Atosiban)

Safest with minimal side effects
 $\rightarrow$ cause water retention

Calcium Channel Blockers (Nifedipine)

Cervical incompetence

Definition

- It's a weakness in the circular layer of internal OS.
- Etiology: Most cases are idiopathic.

Presentation

- Painless 2nd-trimester abortion with sudden rupture of membrane, a gush of fluid & passage of all products of conception.
- Bleeding/ Discharge
- Or Incidental routine exam.

Risk factors

- Previous mid-trimester pregnancy loss and/or preterm birth
- Previous obstetric or gynaecological trauma (e.g., termination of pregnancy, rapid delivery, multiple gestations, or cervical conization)
- Short cervical length: transvaginal cervical length < 25 mm on ultrasound before 24 weeks gestation
- Cervical connective tissue weakness (e.g., Ehler-Danlos syndrome)
- Diethylstilbestrol exposure

Diagnostic

Presentation + P/E: painless effacement on cervix exam

Investigations:

- In pregnant: U/S (cervical length of funnelling)
- In non-pregnant: Hysterosalpingogram (funnelling)/ hegar dilators

Management

Previous Hx of cervical incompetence & wants to be pregnant

- Elective cerclage (12-14 weeks) after ruling out chromosome abnormalities
- Removed (36-38 weeks)
- Expectant till delivery

If pregnant

Pre-viable

Expectant management & emergent cerclage (or elective termination)

Viable

* Expectant * cerclage

Types of cerclage

- Transvaginal
 - 1- Macdonald stitch: at cervical-vaginal junction
 - 2- Shirodkar stitch: at the internal OS (submucosal)
- Transabdominal cerclage (TAC):
 - Permanent
 - Indication: failure of Macdonald & Shirodkar stitch
 - mode of delivery: C/S

Complications of cerclage

- 1- Infection – discharge
- 2- Rupture of membrane
- 3- Preterm-labor
- 4- Lacerations / trauma

Indications for emergent removal of the stitch

- 1- Labor (most common indication)
- 2- Rupture of membrane
- 3- Infection
- 4- intrauterine fetal death

Torch infection

Maternal infection $\Rightarrow$ fetal abnormalities $\begin{cases} \text{Miscarriage} \\ \text{stillbirth} \\ \text{Abnormalities at birth.} \end{cases}$

Toxoplasmosis: protozoa
 (mom) $\begin{cases} 1^{\circ} \text{ infection} \rightarrow \text{lymphadenopathy, fever, chills, sweats} \\ \text{latent} - \text{NOT infect fetus} \end{cases}$

- Diagnosis $\Rightarrow$ IgM, IgG

- Triad in fetus

$\begin{cases} \text{Hydrocephalus} \\ \text{Intracranial calcification} \\ \text{Chorioretinitis (post. uveitis)} \end{cases}$

Rubella

- RNA

- RS

(mom) - Maculopapular + lymphadenopathy + arthritis

(baby) - sensorineural deafness / cataract / PDA
 Blueberry muffin baby

CMV

- DNA herpes

- sexually

(mom) - Mono-nucleosis like $\begin{cases} \text{Rhinitis} \\ \text{pharyngitis} \\ \text{headache} \\ \text{myalgia} \\ \text{Arthralgia} \end{cases}$

(baby) - sensorineural hearing loss

Microcephaly

Small GA

+ **Intracranial calcification**

Periventricular



Others (syphilis, varicella, parovirus B19)

Syphilis (spirochete) - sexually transmitted

$1^{\circ} \Rightarrow$ Chancre $\Rightarrow$ painless ulcer

$\Rightarrow$ Early findings for fetus

- Maculopapular rash (palms, soles)

- runny nose

- Abnormal long bones (legs)

$\Rightarrow$ Late findings for fetus

- saddle nose / deafness / Hearing loss

- Hutchinsonian teeth (notched teeth)

- Mulberry molars (maldevelopment of molars)

- Saber shins (bowed legs)

Herpes zoster (varicella)

- DNA

- primary $\Rightarrow$ chickenpox

- Reactivation: shingles

cause fetal infection

- Newborn symptoms

scars in dermatomal pattern

microcephaly

Hydrocephalus

bluish

limb atrophy

+ Mental retard.

parovirus B19

- ssDNA

- RS

- 5th disease, slapped cheek syndrome

Mom - Mild anemia in normal pt.
 Chronic anemia in sickle cell pt. $\rightarrow$ Crisis

child - short $\frac{1}{2}$ life RBC / $\uparrow$ volume of RBC / $\downarrow$ immunity

$\rightarrow$ Miscarriage, fetal death, Hydrops fetalis