OBS & GYNE

BY : SARA GHAITH

INDEX

Obstetrics:

INDEX

Gynecology:

INDEX

Additions:

INTRODUCTION

Resources:

- **Kaplan book**
- **Dossier**
- **Blueprints book**
- **Lecture Notes & Rounds**

These summaries might not contain all the topics ….. but I tried to cover most of them ☺

I would like to thank my amazing colleagues for their magnificent help in writing the summaries:
Bushra Tbakhi , Haitham Al-lahham , Dania Hudhud, Eman Abdelghani , Media Nowzad and Saba Jaradat .
It wouldn't have been done without their great help ….

BEST OF LUCK !
SARA GHAITH

This newly typed edition was organized by Leena Al Nsour and Zaid El-Adwan with special thanks to the people who participated in the typing process:

Abdallah Massad
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Farah Abu abeeleh
Farah Alsaadi
Hala Abu faris
Karam Darwish
Leen Alkurdi
Leen Makahleh
Lina Allawama
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Manar Alafeshat
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Mariana Nusseir
Zahra Muneer
Myassar Alzoubi
Nada Saleh
Noor Badayneh
Nura Alramahi
Ola Juneidi
Omar Salaymeh
Omar Sarhan
Rahaf Akroush
Rahaf Omaish
Rawan Alsallal
Roqaya Alaani
Sara Asaad
Shahid Alaabid
Shifar Habashneh
Somaya Alkiswani
Sura Alkhalili
Tayma Khawaldeh
Yaman Jarrar
Yasmeen Al Shebli

Gyne & Obs History

Patients profile:
Name, age, medically free?, residence, married since, G#P# (mention previous c/s), LMP (sure? Regular? OCPs? Lactating?), EDD, GA, blood group (if –ve ask about her husband's blood group), history of blood transfusion, booked or not and by who, admission date and department.

Chief complaint:
HPIx:
pain→ SOCRATES
Associated symptoms → passage of liquor, show or blood
HTN and DM since when if present
any medical illness during current pregnancy? If yes; since when, controlled or not, compliance to medications, risk factors and complications.

History of current pregnancy:
Spontaneous, uneventful pregnancy? Planned?
Diagnosed by (missed periods, urine or serum hCG, U/S)
Date of first antenatal visit, total wt. gain and current Hb.

Specific questions in:
1st trimester:
-normal symptoms of pregnancy.
-Spotting of bleeding.
-Any complications.
-What investigations were done.
-What screenings were done.
-Pap smear.
-Medications.
-Hba1c control, BP control.

2nd trimester:
-Improvement of pregnancy symptoms.
-Any new symptoms.
-Quickening.
-Sugar/Hba1c control
-Triple or quadruple test.
- U/S to Rule out congenital anomalies.
-Complications.

3rd trimester:
-U/S.
-APH.
-Infections.
-In high risk patients: investigations fot PET, S&S of IUGR.
-Control of sugar, Hba1c and BP.
-OGTT screening.
-Hb.
-Complications.

Past obstetric history:

G#P#
Detailed fetal outcome for each birth: gender, alive or dead, weight, NVD or C/S (what's the indication), blood transfusion, postpartum and antepartum complications, lactation.

Detailed abortions history: trimester, gender, cause, followed by D&C or not, was medically terminated? What drugs.

Past medical history: HTN/DM/renal disease/anemia/epilepsy/thyroid/cardiac disease.

Past surgical history:

C/S: when, indication and type.
Any other surgeries.

Drug history: Medications and allergies

Family history:
-Consanguinity.
-HTN, DM, multiple pregnancies, inherited diseases or mental retardations.
-Family history of abortions or DVT (thrombophilia).

Social history: married since, smoking, alcohol, substance abuse, insurance, occupation, # of children, exposure to radiations or toxins.

Gyne history:

- Age of menarche.
- Menses: regular, every how many days if regular, duration, presence of clots, # of pads per day, fully or partially soaked pads, volume of blood.
- Intermenstrual bleeding, postcoital bleeding and dysmenorrhea.
- Vaginal discharge
- Contraceptive methods, and for how long.
- Periods of infertility: cause and if tried any conception methods than normal intercourse.
- Last pap smear.

Follow up after delivery

Patient: Name, age, medical status, P#, mention previous C/S or any PSHx, mode of delivery (i.e. patient underwent c/s), indications and type of anesthesia if C/S.

Fetal outcome: gender, alive or dead, weight, NICU or not, and duration.

Today is day ……. Post op/ post delivery, check if the patient looks well and if she is ambulating, lactating, urinating or on foleys, defecating, passing flatus. Normal lochia or not.

Ask about these symptoms: dizziness, headache, fever, SOB, palpitations, chest pain, LL swelling, and any new symptom.

Physical examination:
-Fundal height and palpate the uterus if contracted or not.
-Chest exam.
-Incision site or dressing: comment on site, appearance, discharge, oozing tenderness, masses redness and hotness.
-Episiotomy site: same comments as incision site.
-Check for PPH.

Hormones of Pregnancy

Peptide Hormones	Steroid Hormones
1. hCG	Progesterone
2. hPL	Estrogen
3. CRH	Androgen
4. Prolactin	Glucocorticoids

A. Peptide Hormones:

hCG: Human Chorionic Gonadotropin

- Source: Placental syncytiotrophoblasts
- Structure: Glycoprotein with 2 subunits: alpha – nonspecific & beta – specific
- Levels:
  Appears in maternal blood ***10 days after fertilization***
  Peak at **9-10 wks**
  After normal pregnancy, it returns to normal within **2-4 weeks**
  In the last first trim. B hCG doubles every 72 hrs
- Purpose:
  1. Maintains corpus luteum production of progesterone until placenta can ztake over.
  2. Regulate steroid biosynthesis in placenta + fetal adrenals?
  3. + testosterone production in fetal male testes

  ** DDx for increased hCG:
  1- Twins
  2- Hydatidiform Mole
  3- Choriocarcinomas
  4- Embryonal carcinoma

  ** DDx for decreased hCG:
  1- Ectopic
  2- Abortion (threatened or missed)

hPL: Human placental lactogen

- Source: Placenta
- Levels: increases through pregnancy (parallels placental growth), max at 36 wks.

- Effects: Antagonizes insulin; so contributing to the predisposition of Gestational DM

** DDx for decreased hPL:
1- Threated abortion
2- Intrauterine growth restriction

CRH: Corticotropin releasing hormone
- Source: initially from the fetal hypothalamus & from the placenta towards the end of the pregnancy
- Effect: Plays a role **in initiating labor**

Prolactin:
- Source: Ant. Pituitary – in response to increase estrogen
- Purpose: + Postpartum milk productions whose symptoms are:

➔ Increases by an increase in estrogen
➔ Decreased by an increased progesterone

B. Steroid hormones:

Progesterone:
- Source:
  - Non- pregnant – corpus luteum (CL)
  - Pregnant < 7 weeks – CL
  - 7-9 Weeks – CL and placenta
  - > 9 weeks – placenta
- Purpose: Early – secretory endometrium (ready for implantation)
  Late – stabilization of myometrium by inhibiting premature contractions.

Estrogen:
Forms:

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

Androgens:

- Source: Mainly from fetal adrenals
- Purpose: Precursor f'or Estradiol & Estriol in Placenta

Glucocorticoids:

- Source: Fetal adrenal glands + Placenta
- Purpose/ Effect: Fetal Lung Maturity
  +increases in labor
  Stria/ increase BP/ Glucosuria

Physiological Changes in Pregnancy

Skin:

- Increased **Vascularity** (under the effect of Estrogen and progesterone (E & P)):
  1. Spider angiomata
  2. Palmar erythema
  3. Chadwick sign

- Increased **Pigmentation** (due to increased Melanocyte- Stimulating hormone (MSH), E & P)
  1. Linea Nigra
  2. Chloasma/ melasma
  3. Darkening of the nipple & areola

Pruritic dermatologic disorders unique to pregnancy: [severe pruritis]	
Urticaria	2nd & 3rd trim. Lesions/erythema, urticarial papules of plaques Abdominal / thigh/ buttocks/ arms/ legs *NO effect on the fetus* Steroids and antipleuritic drugs
Cholestasis	3rd trim. *Increase risk of stillbirths* Excoriation is common Generalized/ palms and soles Management: Ursodeoxycholic Acid

Hair Changes:

- Mild hirsutism is common.
- Normal pregnancy increases amount of hair in androgen stage (growth), BUT excessive virilization is abnormal ➔ you should think of androgen-secreting tumors.

Ocular Changes:

Increase thickness of the cornea:

1. Edema induces 3%
2. Affects contact lenses

Decreases intraocular pressure:

1. Glaucoma improves ☺
2. Minimally decreases visual fields

Normal Pregnancy State:

1. Hyper lipedema
2. Glycosuria
3. Anabolic

Carbohydrate Metabolism:
Increase in insulin resistance in the 2nd trim. (due to increase in hPL)

Body Water Metabolism:
- Water retention is common and is a normal part of pregnancy.
- Pitting edema of ankles and legs (especially at the end of the day)
- Factors:
  Increase in venous pressure ➔ due to compression of IVC and pelvic v.
  Decrease interstitial colloid osmotic pressure.

Hematologic changes:
Blood volume:
- Increases by 50%: Increase of the plasma volume is greater than the increase in erythrocytes, which leads to dilutional anemia (normal physiological anemia).

- Significance:
1. Meets the demand of the enlarged uterus.
2. Protection against impaired venous return.
3. Protects against blood loss at the time of delivery.

Iron:
- Iron Absorption increases with pregnancy.
- Requirements increase to 1000 mg/day.
- Most iron is used in hematopoiesis especially in the 2nd half of pregnancy
- Iron from the diet is insufficient to meet the needs of the pregnancy ➔ so patients must take supplemental iron.
- Average Hb➔ 12g/dl
- Hb< 10-10.5 g/dl is considered abnormal (anemia). Some consider <11g/dl abnormal).

RBC:

RBC mass	Increases by 30%
Hct	Decreases by 15%
Hb	Decreases by 15%
MCHC	No change
MCV	No change

Dilutional anemia is decrease in Hb relative to plasma volume, while true anemia is decrease in O_2 carrying capacity.
So, physiological anemia is NOT a TRUE Anemia.

ESR: CRP & ALP ➔ increases

WBC:
- Due to increase in Estrogen and cortisol, WBC increases up to 16,000/mm^3 in the 3rd trim.
- Maximum increase is during the intrapartum stage ➔ might reach 26,000-30,000

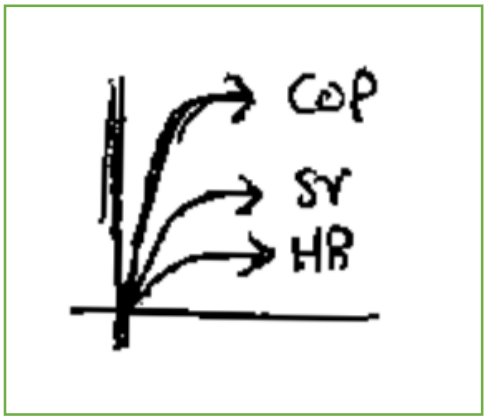
Platelets:
Mild decrease BUT within normal range

Immunology:
Increase in granulocytes and CD8
Decrease in CD4 and monocytes
Cell-mediated immunity ➔ humeral immunity

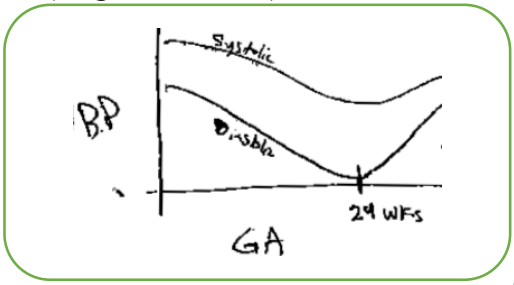
Coagulation factors:
- Increase in all factors (mainly I.VII, VIII,IX, X), except factos (XI and XIII)
- Increase in fibrinogen
- Increase resistance to activated protein C
- Decreased protein S

* Pregnancy is a hypercoagulable state which increases the risk for stroke/DVT/ PE.

Cardiovascular Changes:



Arterial BP: Systolic ↓, Diastolic ↓↓
Venous BP: Central ↔, Femoral ↑
Peripheral Vascular resistance PVR: ↓ by 30%
Plasma Volume: ↑ by 50%
Stroke volume ↑ by 20-40% (Peak – 20 weeks)
HR: ↑ (Peak – 32 weeks)
COP: (Peak – 20 weeks)
* CO = SV x HR
* During pregnancy, uterus receives 15% of COP (large amount!!)
* Systolic and diastolic blood pressure drops, However; after 24 weeks of gestation, it starts to increase but NEVER becomes higher than pre-pregnancy blood pressure state ➔



* *Murmurs:*
- Mid-systolic ejection murmur, (heard on the left sternal border and is due to increase in COP.
- Diastolic murmurs are NEVER Normal and must be investigated if found.
* *Heart:*
- Displaced to the left and upward.
- Apex is moved laterally.
- Mild hypertrophy (Cardiomegaly is seen on CxR)

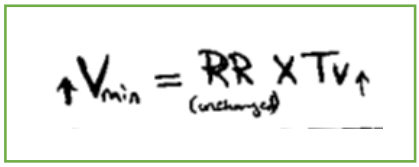
> COP increases intrapartum BUT the MAXIMAL increase is during postpartum. Why?
> Due to autotransfusion of uteroplacental blood rapidly into the peripheral circulation➔ Increasing the risk of heart failure postpartum

Gastrointestinal Changes:
Smooth muscle relaxation due to increase in progesterone causes a decrease in GI motility: this leads to:

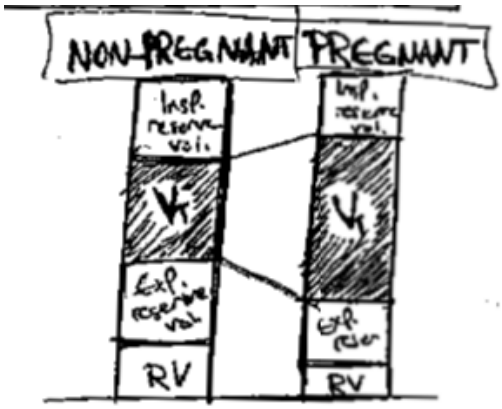
A. Decrease in gastric motility + increase in gastric emptying time + decrease esophageal sphincter tone.
   -The decrease in gastric motility causes an increase in the residual volume inside the stomach ➔ GERD (also caused by an increase intrabdominal pressure).
B. Decrease in colonic motility ➔ increase in water absorption + resulting constipation
C. Decrease gallbladder motility ➔ cholethiasis

Hepatic physiology changes:
- Increase in protein synthesis (estrogen effect).
- Decrease in album concentration due to dilutional effect.
- Increase in clotting factors and cholesterol.
- Normal AST, ALT, GGT and bilirubin.

Respiratory Changes:
- The main change in the lungs ➔ *Tidal volume* (V_T): increases by 40%.
- *Respiratory rate (RR)* ➔ unchanged.
- *Minute Ventilation (V_{min})*: Volume of air moved by the lungs in 1 minute. Therefore, (V_{min}) = RR x V_T. So V_{min} increases.



- *Residual volume*: decreased by 20% due to the pressure of the uterus on the lungs.
- *Total lung capacity* ➔ decreases.
- *FEV_1 and Peak flow* ➔ unchanged
- *ABGs* ➔ Respiratory alkalosis, why?
  Increase in V_{min} ➔ decreases PCO_2 ➔
  Increases pH (7.4 ➔7.45).
  So in order to compensate
  ➔ increase in HCO_3 secretion
  ➔which leads to an increase in urine pH.



All decrease except V_T

Renal Changes:

A. Anatomy:
   Kidney size (increase in blood flow) + Renal pelvis Volume + ureteral volume ➔ All increase.
B. Physiology:
   Renal plasma flow + GFR + Cr Clearance ➔ All Increase
C. Labs:
   BUN + Serum Cr + Serum Uric Acid (UA) ➔ All decrease (due to an increase in GFR)
   + Glucosuria and normally there is <u>no proteinuria</u>.

Endocrine Changes:

A. Pituitary: Increase in size by 100% by term ➔ due to increase in blood flow. This makes it susceptible to ischemic injury, Sheehan syndrome, from Postpartum hypotension and hemorrhage.
   *Prolactin levels increase at term preparing for lactation.
B. Adrenals: No change in size, but increase in the production of cortisol (2-3x). Cortisol along with hPL, estrogen and progesterone predispose the patient to GDM.
C. Thyroid:
   - Thyroid gland increases in size ➔ due to increase in blood flow.
   - Thyroid binding globulin TBG increases
   - Total T_3,T_4 increases due to increase in TBG.
   - TSH, TRH, Free T_3,T_4 are unchanged!

Fetal circulation:
In utero shunts:

- Ductus venosus: Umbilical v. to IVC.
- Foramen Ovale: Right atrium to left atrium.
- Ductus Arteriosus: Pulmonary artery to descending aorta.

Medical Complications in Pregnancy

Cardiovascular Diseases:-

Types: 1) Rheumatic Heart disease: a) The most commonly acquired lesion
b) Most common is Mitral Stenosis

2) Coronary Artery Disease (rare in childbearing age)

3) Congenital Heart Disease: a) Most common is ASD and VSD
b) Most common cyanotic heart disease is Tetralogy of Fallot

*complication increase by 50% in mid pregnancy
** Cyanotic CHD are: a) Truncus Arteriosus b) TGA c) Tricuspid Arteriosus d) Tetralogy of Fallot
Maternal Mortality Risk:

- Low Risk (<1% risk of death): ASD, VSD, PDA (3 D's)

Minimal Mitral Stenosis, Porcine heart valve, corrected TOF

- Intermediate Risk (5-15%): Mitral Stenosis with atrial fibrillation, Artificial heart valve, uncorrected TOF, Marfan with aortic valve diameter.
- High Risk (25-30%): Pulmonary HTN, Eisenminger Syndrome, Marfan with aortic valve dilation > 4cm, Peripartum Cardiomyopathy.

  should be advised to terminate pregnancy

Signs of heart disease:

- Any diastolic or continuous murmur
- Any systolic murmur with thrill
- Any severe arrhythmia
- Unequivocal cardiac enlargement

In Details

Mitral Stenosis:

Pathophysiology: In patients with mitral stenosis, increase in preload (due to increase in blood volume) leads to Left atrium overload. The increase in pressure in the left atrium causes Pulmonary Hypertension.

-What worsens Mitral Stenosis is an increase in heart rate and an increase in blood volume (Normal changes in pregnancy)

-Tachycardia associated with labor and deliveries drastically increase pulmonary hypertension

-25% of patients with mitral stenosis have heart failure for the first time during pregnancy

- increased fetal risk of intrauterine growth restriction

-Peripartum period is the most hazardous

Management:

a) Decrease Tachycardia

b) Decrease excessive IV volume

c) Consider intrapartum SBE Prophylaxis

Mitral Valve Prolapse (MVP):

- Asymptomatic

- Physical Exam shows Midsystolic click

- **SAFE IN PREGNANCY**

- Consider SBE prophylaxis

Aortic Stenosis (AS):

- Similar problems to MS

- Avoid tachycardia and fluid overload

- Give SBE prophylaxis

Marfan's Syndrome:

- Autosomal Dominant, connective tissue disease, if associated with dilated aortic valve> 4cm then there is an increased risk of aortic dissection.

- Treatment is Surgical correction

Eisenminger's Syndrome:

-Right- Left bidirectional shunt+ Pulmonary Hypertension

- Extremely dangerous to the mother

-Only 25% of pregnancies reach term!

-Treatment: avoid Hypotension and terminate pregnancy.

Peripartum cardiomyopathy: (Idiopathic cardiac decompensation)

-Risk factors: AMA, Multiparity, Multiple Gestations, HTN

**Mortality Rate is 75%.

-Management: Terminate the pregnancy and supportive care with ICU care.

-Management Antepartum: Left Lateral Rest, avoid strenuous activity, avoid anemia, Digitalis and diuretics as indication (for HF), Fetal echo (if man has congestive Heart disease).

-Magangement Intrapartum: Aim for vaginal delivery (avoid induction of labour, consider assisted delivery to shorten the 2nd stage), O2, Sedation, Monitor IV volume (strict input and output), SBE prophylaxis (except for ASD)

-Management Postpartum: Watch for PDH, Observe for pulmonary Edema.

NYHA classification

Class 1: No symptoms of decompensation

Class 2: No symptoms at rest, mild limitation

Class 3: No symptoms at rest, marked limitation

Class 4: Symptoms at rest which increases with exertion

Thyroid Disease:

-**Hyperthyroidism** could be caused by:

1- Graves Disease (most common)
2- Toxic Nodule
3- Hydatidiform mole
4- Toxic Diffuse

-Complications: Uterine complications (Abortions, Prematurity, Intrauterine Growth Restriction)

-Treatment of Hyperthyroidism:

a) Medical: Propylthiouracil (PTU) and Methimazole

b) Surgical: Indication is: failure of medical treatment.

Radiotherapy is contraindicated

Hyperthyroidism is noted in Hyperemesis Gravidarum (HG) and GTD

*Graves triad is: 1) High free T4. 2) High TSH. 3) TSH Reactive antibodies

***Normal Thyroid Physiology:**

-Increased Thyroid BF leads to Thyromegaly

-Increased GFR leads to increased iodine excretion and decreased plasma iodine

-Estrogen causes increased production of TBG and increased total T3,T4.

-Fetal thyroid function begins as early as 12 weeks.

***Thyroid Storm:**

-An acute life threatening hyper metabolic state in patients with thyrotoxicosis.

-It presents with Fever, tachycardia and severe dehydration, often associated with HF

-Treatment consists of a) B-blockers (decrease tachycardia) b) Steroids (decrease peripheral conversion) c) Iodine (decrease production of T3 and T4).

Hypothyroidism: (Most common cause is Hashimoto)

-Treatment: Levothyroxine (increased Requirement during pregnancy)

-Complications:

1- Increased Risk of abortions

2- Infertility

3- PET

4-Abortion

Note: Both iodine deficiency and excess can cause hypothyroidism.

-Hypothyroidism triad is: 1) low T4. 2) High TSH. 3) Anovulation and infertility

- Overt Hypothyroidism: High TSH and Low T4.

- Subclinical Hypothyroidism (more Common): High TSH and Normal T4.

Anemia:

- HB < 10-10.5 g/dL during pregnancy
- Abnormal Heme (IDA or Folate Deficiency) or Globin (sickle cell anemia or Thalassemia)

Iron Deficiency Anemia (IDA) (Nutritional Anemia) **most common anemia in Females**

- IDA triad: Hb < 10, MCV <20, RDW >15%
- Risk factors: a) chronic bleeding b) Poor Nutrition c) frequent Pregnancy
- Maternal Requirements of Iron : 1Gram (1000 mg)

Divided into 300 mg placental, 200 mg fetus, 500 mg labor

- Symptoms: usually asymptomatic, if symptomatic presents with general malaise, palpitations, ankle edema.
- NO effect on fetus.
- Treatment FeSO4 (325 mg.)

Folate Deficiency (nutritional Anemia)
Folate triad is Hb < 10, MCV > 100, RDW >15%
Folate stores are enough for 90 days
On peripheral smear we can see Multisegmented Neutrophils, Macrocytic anemia
Risk Factors:

- Chronic Hemolytic Anemia (sickle cell anemia)
- Anticonvulsants (phenytoin/ barbiturates)

Requirements: (prevention), 0.4 mg (if High risk then give up to 4mg)
Treatment: 1mg/day PO

Sickle cell Anemia:

- Inherited Autosomal Recessive
- Risk factor: Africans and Mediteranians ** in trait, Increased risk of UTI**
- Screening: Presence of HBS (doesn't differentiate carrier state from disease)
- Diagnosis: Electrophoresis
- Treatment: avoid hypoxia, give tonics, monitor fetus
- Complications: Antibodies, IUGR, FD, Preterm delivery

Liver Disease:

Intrahepatic cholestasis of pregnancy:

- Increased by estrogen, 2nd half of pregnancy, increased risk with multiple Pregnancies.
- Symptoms: intractable pruritis of palms and soles (worse at night) **without skin finding** (it disappears after delivery)
- Lab: mild increase in bilirubin and increase of bile acids from 10-100%
- Complications: No effect on mother, increased risk of PTL and stillbirths
- Management: Ursodeoxycholic Acid (treatment of choice) (mechanism of action is that it dissolves bile acids, antihistamines, cholestyramine)

Antenatal Fetal testing should be initiated at 34 weeks.

Acute Fatty Liver:

- Inherited disease, Rare and life threatening
- 3rd trimester
- Maternal Mortality is 50-70%!!
- Etiology: disordered metabolism of fatty acids by mitochondria of the fetus (deficiency LCHAD)
- Symptoms (gradual onset of symptoms)
  Non-specific flu like symptoms (nausea, vomiting, anorexia and epigastric pain)
  Jaundice and Fever (70%), HTN, Proteinuria and edema
- Labs: Hypoglycemia (liver failure and decreased glycogen stores) and increased ammonia (No clearance)
- Complications: acute Renal failure/ Hepatic encephalopathy/ coma
- Management: ICU and hydration, delivery
  ** Resolution follows delivery**

• UTI/pyelonephritis/bacteriuria :

Asymptomatic bacteriuria	Acute cystitis	Acute pyelonephritis
No urgency No frequency No burning No fever +ve urine analysis and culture	Urgency Frequency Dysuria No fever +ve urine analysis and culture	Serious ! Urgency Frequency Dysuria +ve urine analysis and culture
Outpatient treatment: Oral antibiotic (Nitrofurantoin)		Admit patient IV hydration IV cephalosporin +/- Gentamicin
complications: If untreated →30% will develop pyelonephritis		Sepsis ARDS Preterm labor

• Thrombophilia :

Group of disorders that promote blood clotting
Most of them are asymptomatic

WORK UP :

- Protein C,S
- Factor 5 leiden
- Homocysteine
- Prothrombin
- Antithrombin 3

Risk factors :

- Immobilization
- Surgery
- Pregnancy
- Family history

Types :

Acquired	Inherited
• factor 5 leiden mutation /m.c • prothrombin mutation /m.c • antithrombin 3 deficiency • protein C,S deficiency	Antiphospholipid syndrome

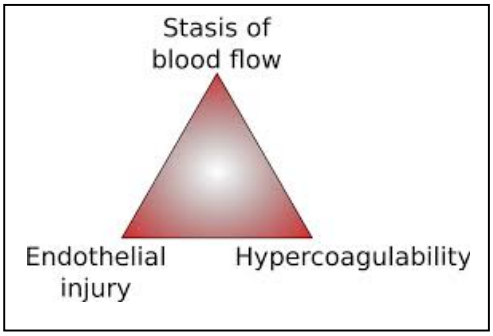
Complications:

- Abortions
- Stillbirths
- Abruption of placenta
- Sever preeclampsia toxemia
- Increase risk of DVT/PE

treatment:

- Subcutaneous heparin
- LMWH
- Low dose aspirin
- Postpartum: warfarin for 6-8 weeks (safe in breast feeding)

- **Thromboembolism:**



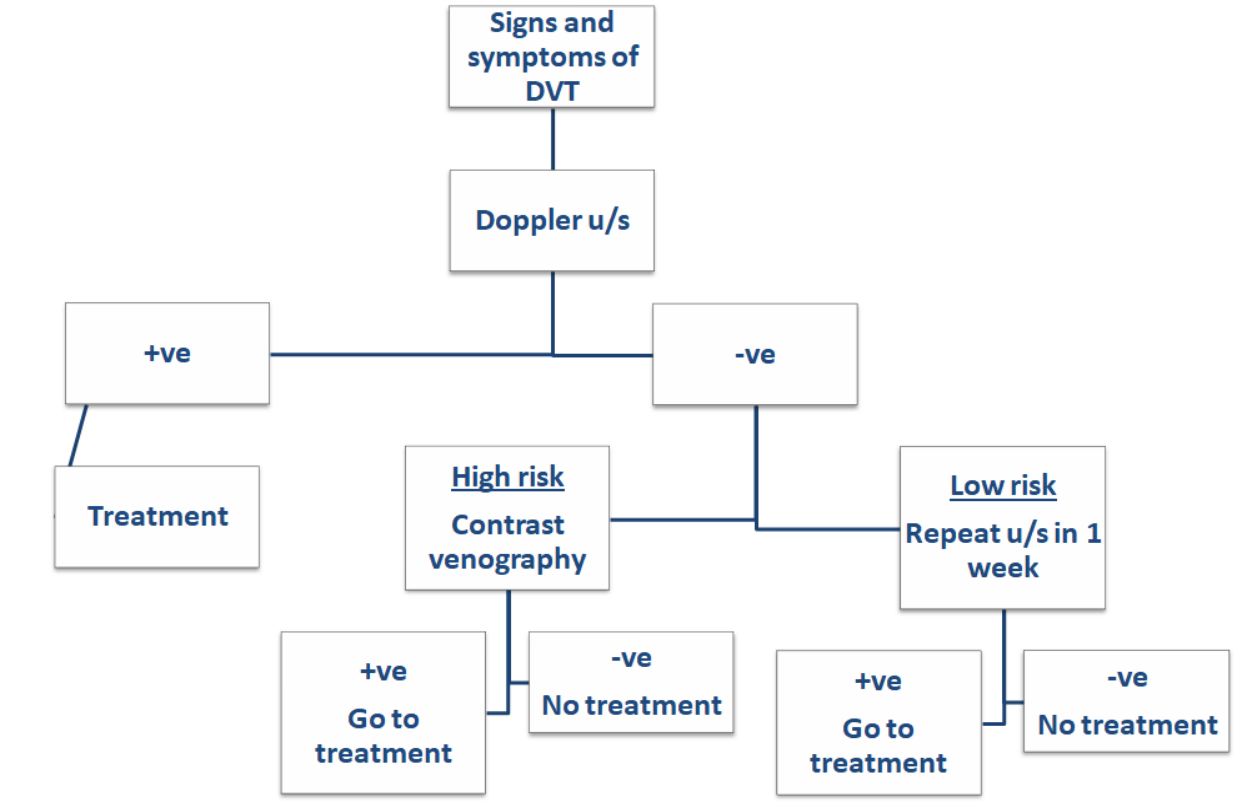
Pathophysiology: Virchow's triad
The highest risk is in postpartum
Endothelial injury; e.g. Traumatic delivery or C/S

- **Superficial thrombophlebitis**

Diagnosis of exclusion
Doesn't predispose VTE but mimic serious conditions
Symptoms: localized pain and sensitivity
Management: conservative

- **DVT**:

May come asymptomatic
Diagnosis: duplex Doppler (best initial test)
Venography: (gold standard)



Treatment: full anticoagulation and IV heparin to increase PTT 1.5-2.5 times of control / warfarin is contraindicated
Monitoring: by anti –x levels

- **Pulmonary embolism:**

Signs and symptoms: Maybe asymptomatic / 80% chest pain and SOB/ tachypnea 90%
Investigations: ECG /CXR/ABG
Diagnosis: angiogram (best initial test)
Pulmonary angiogram (most definitive): most common indication: -ve CT angio in high risk patient
Treatment: full anticoagulation and IV heparin no warfarin

Hypertension

- **Pregestational HTN:** ≥ 140/90 onset before 20 weeks of gestation
- **Gestational HTN:** ≤ 140/90 onset after 20 weeks of gestation and no proteinuria
- **Preeclampsia toxemia (PET)**

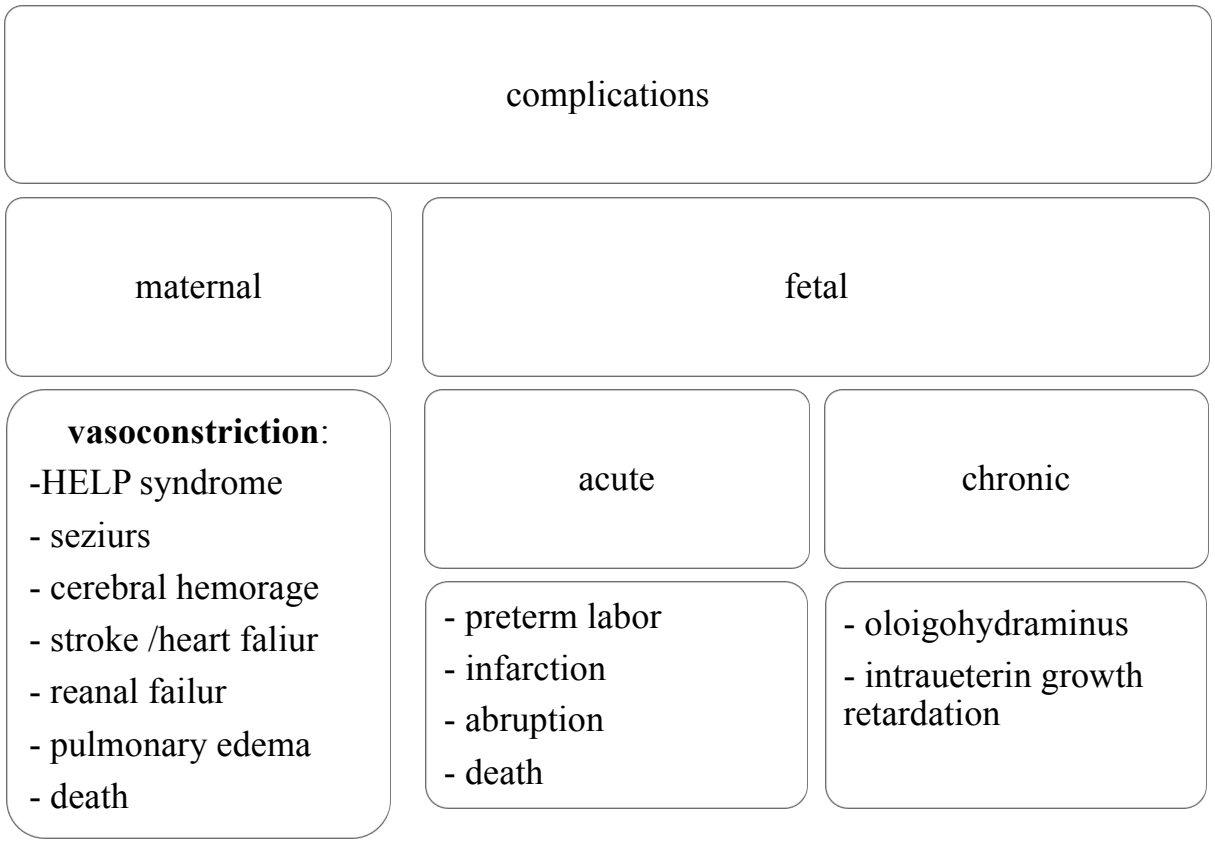
		dipstick	Or 24 h urine collection	
Mild	More than 140/90 + proteinuria	+1/+2	300mg	
Severe	More than 160/110 + proteinuria	+3/+4	5g	Symptoms of PET or increase LFT or DIC or pulmonary edema

Symptoms:

- **CNS**: headache /blurry vision/scotoma/stroke
- Independent edema (periorbital)
- **Chest**: pulmonary edema
- **Abdomen**:
  - nausea, vomiting, RUQ pain = HELP
  - Weight gain…edema
  - Vaginal discharge, painful bleeding = abruption
  - Oligouria / frothy urine = renal failure
  - Fetal movement = intrauterine growth retardation
- Lower limb edema
- Hyper reflexes

Risk factors:
Pathophysiology: diffuse vasoconstriction

- Previous history of PET
- Primigravida …increase risk x8
- Extremes of age
- DM/HTN/renal failure /thrombophilia
- Big uterus polyhydramnios /macrosomia/multiple pregnancy
- Hydrops fetalis
- Hydrated mole



Approach:

- **History**: ask risk factors / symptoms/ complications
- **Physical exam:** vital signs / general/chest exam/abdomen / lower limb
- **Investigation**: hemo concentration: Hb /Hct/BUN/Cr/ uric acid
- Dipstick and 24 h urine collection and KFT
- DIC profile (PT, PTT, D-dimer, fibrinogen, platelets)
- Biophysical profile, non-stress test by U/S
- Doppler
- Presentation/placenta/movement by U/S

management:

- admit the patient
- fetal and maternal monitoring
- if PET is mild:
  - Less than 36 weeks = expectant management
  - More than 36 weeks =deliver (induction)
- If PET is severe = deliver (induction if patient is stable)
  - Mode of delivery is normal vaginal delivery unless there is an indication for c/s

* HELP syndrome occurs more in multigravida than in Primigravida

Medication:

- **Acute**: hydralazine (1st line), labetolol
- **Chronic**: methyl dopa, nifedipine

* Prophylactic: MgSo4 (to prevent seizures) loading dose is 5g bolus then 2g/h for 48hrs (it prevents seizures but doesn't treat HTN)
* ACEI is contraindicated
* Eclampsia =PET+ unexplained seizure /treatment: diazepame

REMEMBER:
Sever preeclampsia is diagnosed if:
blood presser is more than 160/110 OR **symptomatic (epigastric pain /headache/visual changes) and blood presser is more than 140/90** OR **DIC/increase LFT/pulmonary edema and blood presser is more than 140/90**

MgSO4:
There is risk for respiratory failure

you should monitor it's toxicity clinically (more than 10g):

- hyporeflexia (1st sign)
- Respiratory rate /chest
- pulse
- urine output

Anti dote for it is calcium gluconate

Diabetes mellitus
Carbohydrate metabolism intolerance

Types :

- Pre-gestational (incidence is 5-10%)
- Gestational (incidence is 3-5%)

Diabetogenic hormones:

- HPL
- Placental insulinase
- Cortisol
- Progesterone

Gestational DM:
It appears during pregnancy in the 2nd half of 2nd trimester (starts at 24-28weeks)
risk factors:

- Age (especially if younger than 25)
- Obesity (more than 90)
- Family history of DM type 1 or 2 or gestational DM
- PCOS
- Recurrent infections (recurrent vulvovaginitis)
- HTN/PET
- Previous history of GDM /macrosomia /polyhydramnios/obstructed labor
- History of unexpected fetal death /neonatal death /congenital anomalies /intrauterine growth retardation
- Current pregnancy: polyhydramnios /macrosomia

Treatment:
In GDM, we start with diet and life style modifications, if failed , we start metformin/insulin
In Pregestational diabetes, we stop Pregestational oral hypoglycemic and start metformin /insulin (insulin has better control +not teratogenic unlike hypoglycemic

* **Dose of insulin in pregestational DM:**
  In general, the dose is higher than the pregestational dose, why?
  Due to diabetogenic hormonal effects
  Doses:
  Decrease in 1st trimester, we start to increase the dose in 2nd trimester, peak at 3rd trimester
* Note: pregnancy affects diabetes and vice versa

Effects of pregnancy on DM (pregestational DM)

- Difficult to control DM due to: increase body weight /increase volume distribution/hyperemesis gravidarum
- Recurrent hospitalization due to: recurrent hypo and hyperglycemia /UTI
- Increase DM emergencies: hypoglycemia /DKA/diabetic coma
- Increase vascular complications

- Increase neurologic symptoms: peripheral neuropathy /GI discomfort

Effects of DM on pregnancy:
Maternal side:

- Increase risk of abortion
- Increase incidence of preterm labor and PROM (due to polyhydramnios/macrosomia/recurrent UTI)
- Increase risk of traumatic delivery and Increase c/s and 50% macrosomia
- Increase risk of postpartum hemorrhage
- Increase risk of wound infection
- PET and gestational HTN 25% , IF ALREADY DIABETIC risk is 40%

Fetal side:

1. Congenital anomalies: only in pregestational DM (TYPE 1 OR 2) and depends on HbA1C in 1st trimester
    - Types:
    - Cardiovascular: VSD/ASD/TOF
    - CNS: caudal regression syndrome/ spinapifida /brain cyst /dany walker
    - Renal: polycystic kidney /multicystic kidney /renal obstruction
    - Limb and GI anomalies
    - Sinus inversus

> The most common anomaly due to DM is cardiovascular, but the most specific is caudal regression syndrome

2) Preterm Labor and its complications:
   30s-40s mortality
   Immature visceral organs
   Prolonged hyperbilirubinemia and hospitalization
3) Neonatal complications:
   Ketoacidosis and hypoglycemia (especially in DM type I)
   Hypothermia/RDS/kerticterus/poor weight gain
   Electrolyte disturbances (hypocalcemia, hypomagnesemia, polycythemia)
4) Increased chance to be diabetic (30%), HTN and cardiovascular disease
5) Intrauterine death and IUGR

Maternal investigations

- Blood sugar monitoring
- HbA1c and OGTT
- TFT (35% association in DM type I)
- KFT
- 24-hr urine collection, urine dipstick
- +/- hypertensive workup

At each trimester do:
-HbA1c
-TFT (DM1)
-KFT

In type I, II, you should refer to consultations:
*Ophthalmo consultation:
If background retinopathy → benign
Proliferative retinopathy → delivery C/S (to avoid valsava that will increase risk of retinal detachment)
*Cardio consultation: Baseline ECG and echo
*Endocrine consultation
*Nephro consultation (especially if HTN)
These conditions are NOT mandatory to repeat unless the disease progressed.

❖ Frequency of visits (in diabetics)
1st trimester – monthly
2nd trimester – every 2 weeks
3rd trimester – weekly

According to JUH
Follow up in non-diabetics:
Up to 32 weeks – monthly
32-37 weeks – every 2 weeks
>37 – weekly

- **Fetal Follow Up and Investigations** -screening by U/S
- 1st trimester: viability
  GA by crown-rump length
  nuchal translucency
- 2nd trimester: (level 2 U/S) between 18-22 wks
  for structural anomalies.
- 3rd trimester:
  -AFI and NST (modified biophysical profile BPP)
  Between 32-36 wks
  -Placenta

Nuchal translucency:
It's lymphatic fluid at the back
Normal < 3 mm
If > 3, chromosomal abnormalities, renal disease or diaphragmatic hernia

95% of gross anomalies can be detected by level 2 U/S.

-Doppler U/S
-U/S for estimated fetal weight
between 34-37 wks to
induce labor before occurrence of macrosomia.

In POOR control diabetes:
Oligohydramnios and IUGR
due to vasoconstriction.
While in moderate control:
polyhydramnios and macrosomia.

- **Time of delivery**
  Planned to deliver at 38 weeks
  If good control (without any comoborbidities) → at 39 weeks.
- **Mode of delivery** (depends on obstetric history and fetal factors
  NVD unless there is an indication for C/S

Indications for C/S in DM:

- Uncontrolled DM/ IUGR
- Fetal distress
- Fetal weight > 4.5 kg
- Proliferative retinopathy

Vacuum, forceps are NOT used when macrosomia is present due to increased risk of shoulder dystocia.

Postpartum monitoring for diabetics:
*incision care/ prevent infection
Pregestational DM

- STOP insulin for (24-48) hrs postpartum or any hypoglycemic because placenta is gone!

  Then either:
  Back to pregestational dose
  Or back to ½ gestational dose.

- 24 hr urine collection for creatinine and protein clearance 6 weeks postpartum + ophthalmo appointment 12-14 weeks postpartum.

Gestational DM (GDM)

- STOP insulin
  & Do OGTT 6 weeks postpartum to make sure that she returned to normal state.

*25-35% will develop DM after pregnancy so screen for DM II at PP visit and every year thereafter by fasting blood glucose (FBG)
*50% risk to develop GDM in subsequent pregnancies.

INTRAPARTUM (during delivery), glucose requirement increases so less insulin is needed.

SCREENING → By OGTT

- In low risk patients: between 24-48 weeks. (In Jordan, because of increased number of patients with diabetes we do it as soon as possible)
- In high risk patients: As soon as possible!

Normally,
HbA1c <6.5
Random <200
FBS<100 in pregnancy it should be <92
OGTT Dx (See next page)
Loading dose: 75g -- FBS: >92 -- 1hr postprandial: >180 (>160 in Jordan) -- 2hr postprandial >155

COUNSELLING
For pregestational DM patient (Anteconception clinic)

1) Tight glycemic control (HbA1c control prior 8-12 weeks before conception)
2) Talk about diet, exercise and insulin before and after conception
3) Talk about effects of DM on pregnancy and effects of pregnancy on DM
4) Increased risk of neural tube defects (NTD) – so put patient on 4 mg folate.

History taking for a diabetic mother:

- Before pregnancy: ask about control of blood sugar in the previous 2-3 months
  ask about her diet, exercise, and weight loss.
  Then do
- Early booking
- Baseline investigations
- More frequent visits
- Management: adjust dose (decreases in 1st trimester, increase in 2nd and 3rd trimester, decrease intrapartum)
- Mode of delivery

OGTT

JUH Protocol:

- Do FBS for all patients at booking
  - If FBS>126 mg/dl → Preexisting DM → Treat as DM
  - If FBS 92-126 mg/dl → Do 75g OGTT immediately
  - If FBS <92 mg/dl → Do 75g OGTT at 24-28 weeks

*Criteria for GDM:

75g FBS: >= 92 1st hour: >= 180 2nd hour: >=153

*Patient's instructions before OGTT test:

1) Normal diet 3 days before test
2) Fasting for 12 hours
3) No smoking
4) Remain seated during test

US Protocol:

First step – Screening test → 50 g glucose load, then measure blood glucose 1 hour later, if >= 130-140 mg/dl → positive test, go to step two.

Step two – OGTT → 100 g glucose

FBS>105
1 hr>90
2 hr>165
3 hr>145

If >=2 readings are high, then it's GDM

If one is abnormal, then it's impaired glucose tolerance.

Antenatal Care

Aims:

1) Determine the health state of mother and child.
2) Determine GA
3) Initiate plan for obstetric care (routine vs high-risk)
4) Decrease maternal/perinatal mortality and morbidity

1st trimester: (0-13) weeks
2nd trimester: (14-27) weeks
3rd trimester: (28-Birth)

Embryo: Fertilization – 8 weeks
Fetus: 9 weeks- birth

FIRST VISIT and 1st trimester:

- History and P/E
- Labs:
  - CBC
  - Blood group, Rh, Antibody screen (Indirect Coombs)
  - UA and urine culture
  - Pap smear
  - Blood sugar
  - Gonorrhea and chlamydia cultures and PCR
  - Infection screen: Rubella/ Syphilis/ HBV/ HIV/ TB

Parity: **TPAL**
Term, **P**reterm, **A**bortuses, **L**iving children

Freq. of visits:
<28 wks: every month
28-36 wks: every 2-3 wks
>=37 wks: every wk

Note:
41-42 wks: every 2-3 days for fetal testing

- U/S

Notes:
*Rubella IgGAb: If the antibodies are positive it means that there is no primary infection during pregnancy (Abs give life long immunity)
If negative: - DON'T give vaccine (because it's live attenuated)
Risk of primary infection (especially in 1st trimester)
*HbsAb: If positive: successful immunization
If positive HbsAg indicates previous/current infection (HIGH risk for vertical transmission)
-Positive HbeAg indicates highly infectious state!

Initial prenatal labs STDs:

Chlamydia/ GC	Screening	DNA Probes (PCR)
HBV	Screening	HbsAg
Syphilis	Screening Definitive	VDRL/RPR MHA/FTA
HIV	Screening Definitive	ELISA (detects Abs) Western Blot (detects Ags)

2nd trimester: -Triple marker test: MS-AFP, HCG, Estriol

Quadruple marker test: MS-AFP, HCG, Estriol, Inhibin-A

Maternal Serum Alpha Feto Protein (MS-AFP) → Elective NOT routine prenatal test

AFP: is a major serum glycoprotein of the embryo

Peaks at 12 weeks (in fetus and amniotic fluid)

Rises until 30 weeks (in the maternal serum)

Fetal serum AFP	Peaks at 12 weeks
Amniotic fluid AFP	Peaks at 12 weeks
Maternal AFP	Peaks at 30 weeks

MS-AFP: Performed within 15-20 weeks GA

Normal: 0.75-2.5

- DDx for increased MS-AFP (>2.5)
  - Wrong date (most common)
  - Twin pregnancy
  - NTD (Neural Tube Defect)
  - Ventral Wall Defects (VWD): (Gastrochisis or omphalocele)
  - Renal disease
  - Sacrococcygeal teratoma
- DDx for decreased MS-AFP (<0.75-0.85)
  - Wrong date
  - Trisomy

Sensitivity for trisomy 21 detection increases up to 70% if triple test was done (not only MS-AFP)

Trisomy 21 (Down's syndrome)

↓MS-AFP

Estriol

Hcg

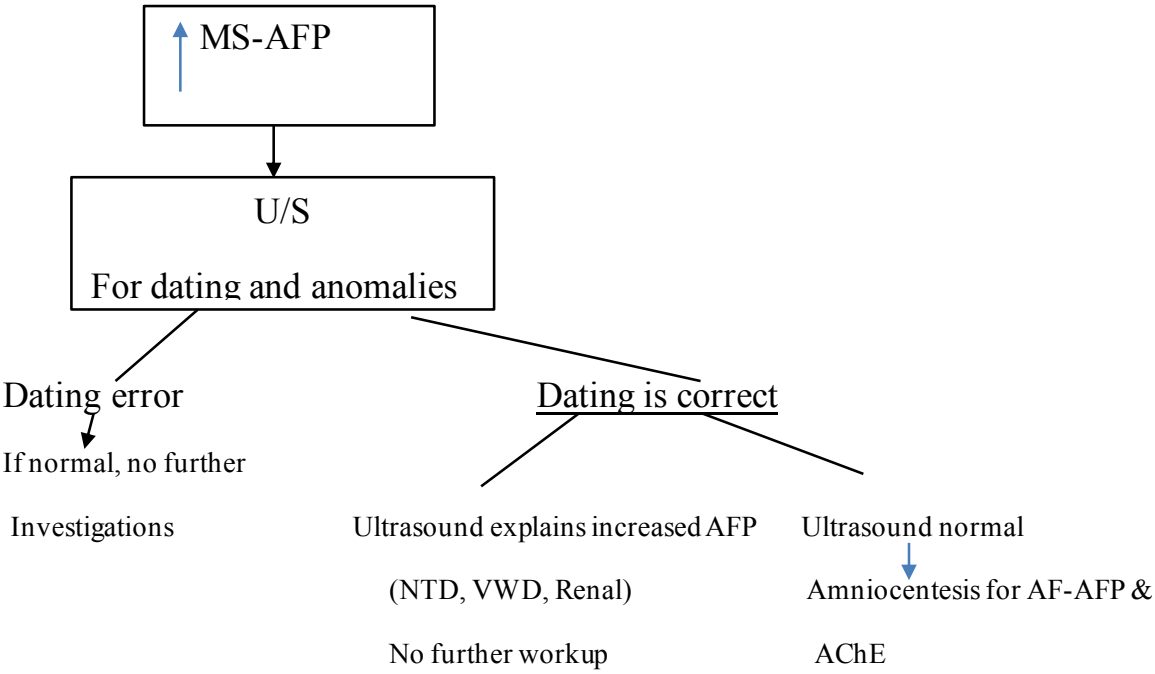
Trisomy 18 (Edward's syndrome)

MS-AFP

↓Estriol

↓Hcg

Next step: Karyotyping if U/S unexplained.



3rd trimester

-CBC
-OGTT
-BP and urine dipstick (risk of PET)
-U/S
-Indirect coombs test (atypical antibody screen AAT): if negative, give anti-D (RhoGAM) at 28 weeks of gestation.

Ultrasound

Types: Transvaginal (TV) and transabdominal (TA)
This table is for the TV US:

Estimated gestational age (weeks)	B-hCG (IU/L)	Visualization
5	>1500	Gestational sac
6	>5200	Fetal pole
7	>17500	Heart activity*

Usually heart activity is detected between 8-12 weeks and as early as 6 weeks.

US benefits during pregnancy:

1st Trimester (0-13 weeks):

- Accurate dating of gestational age by CRL (crown-rump length)
- Fetal viability by FHA (fetal heart activity)
- Visualization of gestational sac
- Site of implantation to rule out ectopic pregnancy
- Number of gestational sacs
- Nuchal translucency (NT): normal NT is <3mm, it is abnormal when >3mm and the most important differential diagnosis are :
    1. Chromosomal abnormalities (down syndrome)
    2. Renal abnormalities
    3. Diaphragmatic hernia

2nd trimester (14-27 weeks):

- 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)

3rd trimester (28-birth):

- Placental location in relation with the internal os
- Biometric measurements in relation to gestational age to rule out Intrauterine growth restriction (IUGR)**
- Fetal weight

- Presentation
- Biophysical profile (BPP)
- Postmature placental signs (calcifications)

**Biometric measurements are used to estimate the fetal weight, and they are:

1. Head circumference (HC)
2. Biparietal diameter (BPD)
3. Abdominal circumference (AC)
4. Femoral length (FL)

Other indications for US:

- Gestational trophoblastic disease (GTD)
- Abdominal pain or bleeding
- Ectopic pregnancy
- Fibroids
- Locate intrauterine contraceptive device (IUCD)
- Chronic villous sampling (CVS) and amniocentesis

US in pregnancy:

- Do at booking
- Nuchal translucency at 11^{th} week
- 18-22 weeks: congenital anomalies

Overview of antepartum testing: NST/AFI/BPP/CST/Doppler

Indications:

- Most common one is decreased fetal movements**
- Diabetes
- Chronic HTN
- Postdate pregnancy
- Intrauterine growth restriction IUGR

**Fetal movements (Kick counting): 10/day but not adequate for primary fetal screening in high risk patients, if fetal movements decrease do Non-stress test NST, if absent fetal movements do US.

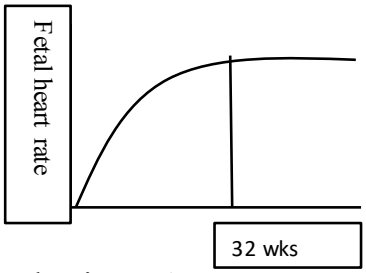
These tests are highly accurate in confirming fetal well-being but poor in predicting fetal jeopardy

NST (Non-stress test): think of accelerations (response of fetal heart to fetal movements):

It assesses the frequency of fetal movements

Prerequisites:

- Healthy moving fetus
- >= 28 weeks gestation (we don't depend on it until (30-32) weeks of gestational age.



If the fetal movements are decreased, NST is the next step

NST has two results:

1- Reactive: 2 accelerations (>=15 beats/min) in 20 minutes lasting >15 seconds…highly predictive of fetal well-being

Non-reactive: not meeting the criteria above, the next step is vibroacoustic stimulation (VAS), if still non-reactive do biophysical profile (BPP) or

contraction stress test (CST). 80% of non-reactive NST are false positive because the fetus is sleeping or it is premature (2 differential diagnosis for false non-reactive NST).

Amniotic fluid index (AFI):

2 ways to assess it:

1- Sum the 4 quadrants, normal (5-25), borderline (5-8), oligohydraminos <5 and polyhydramnios >25
2- Single deepest pocket: normal 2-8

*Functions of Amniotic fluid (AF):

- Proper growth
- Fetal lung maturity
- Barrier against infections
- Protection and cushioning

*Consequences of decreased AF:

- Lung hypoplasia
- Limbs contractures

Biophysical profile (BPP):

Component	0	2
NST	Non-reactive	Reactive
AFI	Abnormal	Normal
Fetal breathing (chest movements)	Absent	Present (>= 1 episode for >=20 secs)
Body movements	Absent	Present (>=2 movements in =< 30 mins)
Tone	Absent	Present (>=1)

All are done by US except NST.

Scores:

- 8,10: reassuring, repeat as indicated
- 4,6:concerning, management depends on gestational age, if >=36 weeks deliver!!!!!, if <36 weeks repeat after 12-24 hours or do CST
- 0,2: Ominous!!prompt delivery

*Modified BPP: only NST and AFI, the predictive value is almost as high as a complete.

Contraction Stress Test (CST)

It assesses the ability of the fetus to tolerate transient decrease in intervillous blood flow that occurs with uterine contractions.

Prerequisites:

≥ 3 contractions in 10 mins

Contraindications:

Whenever uterine contractions are hazardous to the mother or fetus

- Previous classical incision
- Previous myomectomy
- Placenta Previa
- Incompetent cervix
- Preterm Rupture of Membrane (PROM)
- Preterm labor

Criteria for late deceleration

- Gradual increase or decrease in fetal heart rate (unlike variable which is rapid increase or decrease)
- Comes after uterine contraction

Indication:

Biophysical Profile of 4 or 6

• Absent late deceleration (with ≥ 3 uterine contractions in 10 mins)
→ CST negative
→ Reassurance (Repeat if indicated)
• ≥ 50% late deceleration (with ≥ 3 uterine contractions in 10 mins)
→ CST positive
→ Deliver!

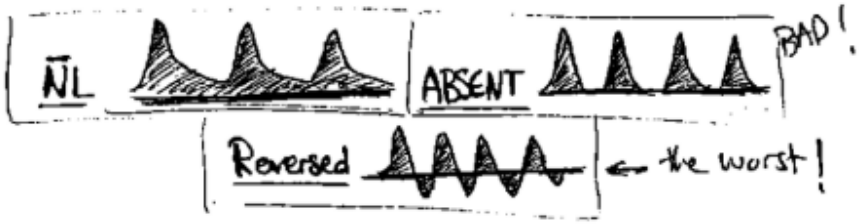
Umbilical Artery Doppler

Based on measurement of diastolic flow

Indications:

Intrauterine growth retardation (IUGR) features

Non-reassuring if absent flow or reversed diastolic



EXAMPLE

Patient came with decrease fetal movement → do Non stress test:

➔ If reactive → repeat as needed

➔ Nonreactive → do Vibroacoustic Stimulation (VAS):

➔ Reactive → repeat as needed

➔ Still nonreactive →do Biophysical Profile (BPP)

➔ 0,2 → Delivery!

➔ 4,6 → Contraction stress test (CST):

➔ Positive → Delivery!

➔ Negative → repeat as needed

➔ 8,10 → repeat as needed

Cardiotocography (CTG)

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

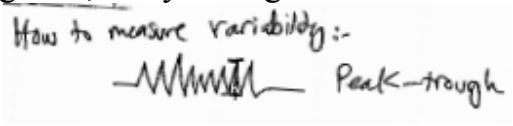
How to read it?

- Name/Date-Time/ Gestational Age
- Baseline HR
  - Normal 120-160 bpm
  - Tachycardia >160
  - Bradycardia <120

- Variability: (reflects sympathetic and parasympathetic)
  - ○ Short-term (beat-beat variability):
    Normal(5-25 bpm), Decreased(<5 bpm), Absent(<3 bpm)
  - ○ Long-term: frequency and amplitude of changes in baseline
    Normal(3-10 cycles/min)

    Causes of decreased variability:
    - Fetal Sleep (usually lasts for 25 mins)
    - Maternal Sedation (with drugs)
    - Fetal Distress (acidosis, PH<7.25)
    - Prematurity (<28 weeks)

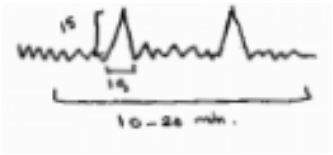
We start doing NST at 28 weeks (to see variability) because at 28 weeks Parasympathetic nervous system is developed. BUT we don't depend on it until 32 weeks.
The most common cause of non-reactive NST is fetal sleep
So we either wait up to 25 mins or we wake him up! (by vibroacoustic stimulation, by tuning fork, or by letting mom eat something sweet)



• Accelerations:
Fetal heart changes in relation to uterine contractions

- If no change → abnormal
- Acceleration → normal response

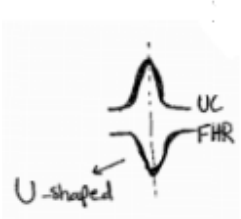
Criteria:
At least 2 accelerations of ≥15 bpm that last ≥ 15 seconds within 10-20 mins.



• Decelerations:

EARLY

- Benign (abnormal if recurrent >15%)
- Due to head compression (which increase vagal response), seen in head enlargement (increase ICP → Increase vagal response)
- Mirror image of uterine contraction (onset with uterine contraction and ends with it)



LATE

- BAD
- Due to uteroplacental insufficiency → hypoxia / acidosis → fetal distress

- Onset of deceleration is to the right of uterine contraction (starts after the uterine contraction ends)
- Severity: (determined by the amplitude of drop in fetal heart rate)
    - Mild < 15 bpm
    - Moderate 15-45 bpm
    - Severe > 45 bpm

Variable (most common)/(seen mostly in 2nd stage of labor)

- Due to cord compression (partial or complete) or prolapse
- Decelerations are NOT related to uterine contractions
- Severity:
    - Mild < 30 seconds
    - Moderate 30-60 seconds
    - Severe > 60seconds

Mixed → difficult to define

- This is CTG of ____ was taken on _day/date/hour_, GA is ___
- FHR baseline is ____
- Good/decreased beat to beat variability
- It's reactive/ no reactive (according to acceleration)
- There is no/ or there are deceleration (specify the type)

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.

Causes of fetal tachycardia		
Maternal	**Medications**	**Fetal**
- Anxiety - Fever - Intrauterine infection - Hyperthyroidism	- Excessive use of oxytocin -Terbutaline/ Epinephrine	- movement/ fetal stimulation - Hypoxia (early) - Prematurity

Causes of fetal bradycardia		
Maternal	**Medications**	**Fetal**
- Supine hypotension ?? - Hypothyroidism	- Epidural Anesthesia -Narcotics/ Sedatives	- Sleeping (not more than 40 mins) - Hypoxia (late) - Cord compression/ prolapse

Causes of fetal distress	
Fetal	Anemia/ Infection/ Twin-twin transfusion syndrome
Umbilical cord	Prolapse/ Hematoma/ Vasa Previa/ One artery/ Short cord/ True knot
Placental	Abruption/ Infarction/ Post mature placenta
Uterine	Tetanic contractions/ hyper stimulation (excessive use of oxytocin)
Maternal	HTN/ Hypotension/ Severe anemia/ Cardiac distress/ pulmonary distress/ seizures

Non-reactive NST	**Non-reassuring NST**
BAD! Needs urgent C/S (1st stage) or assisted vaginal delivery (2nd stage) <u>In the presence of 1 of the following</u>: (1) Severe bradycardia/tachycardia (2) Absent variability (& > 32 wks) (3) Late deceleration (4) Early/variable deceleration >50% or with acidosis or recurrent early >15%	(1) Mild/Moderate bradycardia or tachycardia (2) decrease variability but not lost (3) Early/variable <50%

MANAGEMENT

1. Tilt (change position from supine to left lateral)
2. Give 100% O_2
3. IV Fluids (If late deceleration)
4. STOP Oxytocin (to rule out uterine hyper stimulation) + (In late deceleration)
5. Monitor maternal blood pressure

Then if:

- Improved → monitor
- Persistent abnormal pattern → Fetal scalp PH → ≤ 7.2 → deliver
  → >7.2 → monitor
- Prolonged deceleration → consider immediate delivery

If Stage 1 of labor → urgent C/s If stage 2 of labor → Assisted NVD

Notes:

• NST: correlate FHR to fetal movement without uterine contraction indications: decrease fetal movement.

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

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

negative CST = Reactive

positive CST = Non-reactive

Induction of labor

- **Induction of labor**: is the attempt to begin labor in a non-labor in patient.
- **Augmentation of labor:** intervening to increase the already present contractions.
- **Indications:**

Maternal:

-Chorioamnionitis

-Severe Pre-eclampsia/Eclampsia

-Maternal diseases : Diabetes Mellitus, Renal disease, Chronic pulmonary, disease, Chronic hypertension, Antiphospholipid syndrome.

Fetal:

-Intrauterine growth restriction
-Fetal Demise
-Post term baby
-Infection
-Non-reassuring fetal testing
-Premature Rupture of Membranes
-Oligohydraminos
-Isoimmunization

- **Contraindications:**

Maternal:

-Placenta Previa/ Vasa Previa
-Classical Cesarean section or prior uterine surgery/ Previous myomectomy
-Malpresentation
-Active Herpes Simplex Virus

Fetal:

-Acute distress

-Transverse lie

-Cord Prolapse

- **Methods:**

Medical:

1- Oxytocin:
-Synthetic Polypeptide hormone then increases contractions.
-Acts Promptly when given Intravenously.
-half life is about 5 minutes.

Antepartum Hemorrhage

- **Antepartum Hemorrhage:** Vaginal bleeding occurring after 24 weeks of gestation.
- **Incidence:** 5% of all pregnancies.
- **Causes:**

Systematic	Local
Bleeding disorders, Liver disease Medications (warfarin/ aspirin)	**Non-gyne:** Hemorrhoids, fissures, Hematuria **Gyne:** -Cervical: Lacerations, polyp, intercourse trauma, cancer -Uterine: rupture -Placental: Previa, Abruption -Fetal: Vasa Previa

- **Complications:**
  Decreased fetal movement
  Post Partum Hemorrhage
  Anemia, Renal Failure, Sheehan Syndrome
  Disseminated intravascular coagulation, Amniotic fluid embolism

- **Risk Factors:** (According to cause)

Previa: (Painless, Causeless, Recurrent)
-Previous history of placenta previa
-Cesarean section, Myomectomy, Pelvic surgery
-Multiple gestation, Multiparity, Advanced maternal age
-Uterine Anomaly, smoking

} Prevent normal implantation

-Malpresentation

Abruption: (most common cause of painful antepartum hemorrhage, most common cause of antepartum hemorrhage, most common cause of obstetric disseminated intravascular coagulation)

-Previous history of abruption
-Chronic Hypertension, Pre-eclampsia toxemia
-Smoking, Alcohol, Cocaine
-Vascular degeneration, Diabetes mellitus, Collagen deficiency
-Polyhydraminos, multiple gestation, macrosomia (uterine distension)
-Trauma, road traffic accident
-Short Cord, Premature Rupture of Membranes, Severe decompression, delivery of first baby

} Increased blood pressure Vascular Problem Uterine Distension

MOST IMPORTANT COMPLICATION: DISSEMINATED INTRAVASCULAR COAGULATION

Placenta Accreta: (Painful)
-Multiple Cesarean section
-Previous manual removal of products of conception
-History of pregnancy termination
-increased alpha-fetoprotein

Uterine Rupture:
-Vaginal birth after cesarean section, previous classical incision, more than 2 cesarean section, Myomectomy
-Grandmultiparity, Macrosomia, Malpresentation
-Excessive oxytocin dysfunctional labor
-Road traffic accident

} -Increased intrauterine Pressure -decreased wall strength

Vasa Previa:
-Accessory lobe (succinate)
-Multiple Gestation
-Velamentous Cord

- **Management**

General : Admit, get help, ABC/Vitals, Give O2, insert 2 Large Canulas, Determine blood type, Cross matching, Prepare blood unites, Hemoglobin, Hematocrit, Prothrombin time(PT) , Partial Thromboplastin time (PTT) , International Normalized Ratio (INR), D-dimer, Fibrinogen, Kidney function Test, Liver Function Test.

Specific : Alkaline denaturation (APT), coombs, kliehmer, transabdominal ultrasound, Per vaginal exam and speculum, post partum hemorrhage prophylaxis.
Fetal : Non stress Test (NST), Biophysical profile.
Placenta Previa : (depends on gestational age)

>36 weeks	Cesarean section
<36 weeks & stable	Expectant management, Dexamethasone, Tocolytic
<36 weeks & unstable	Emergent Cesarean section

Abruption of Placenta: (Depends on severity)

Mild <36 weeks	Expectant management
Mild >36 weeks	Deliver
Moderate	Deliver
Severe	Most probably the baby is dead, Stabilize mother, deliver by Cesarean if baby is alive

Vasa Previa: Deliver by Cesarean Section
Uterine Rupture: Repair (hysterectomy if you cannot stop the bleeding)
Accreta, Increta, Percreta: Delivery and hysterectomy

- **Notes**

-Placenta Previa : abnormal Implantation of placenta (Maternal Blood)
Types: Complete
Incomplete
Marginal
Low-lying
Contraindications: Speculum, PV exam, Intercourse
Complications :
Maternal : Shock, multiple C/S, Anemia, Sheehan, Death, increased risk of placenta accreta
Fetal: Malpresentation, Intrauterine growth restriction, Preterm labor, Premature rupture of membranes.
-Abruption: Premature Seperation of Placenta from the uterine wall before the delivery of the baby. (Maternal Blood)

Types:	Concealed
	Visible
Severity :	Mild <1/4
	Moderate <1/4-2/3
	Severe >2/3

Pregnant + Vaginal Bleeding + Pain > abruption until proven otherwise
-Placenta Accreta: Invading the endometrium reaching the myometrium but not invading it.
-Placenta Increta: invading the myometrium
-Placenta Percreta: Reaching the serosa
-Vasa Previa: Present when fetal vessels traverse the internal os. (Fetal blood)

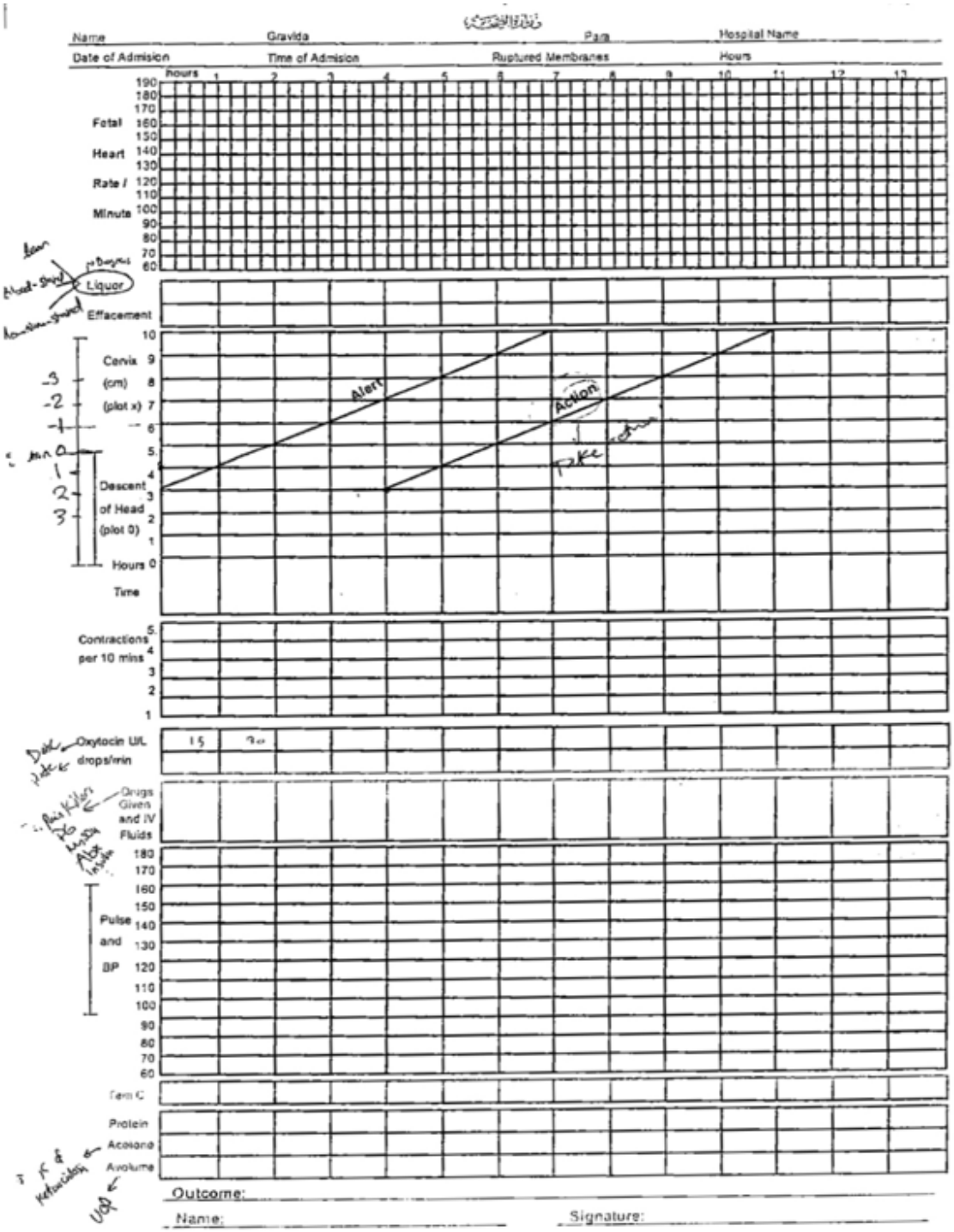
Classical Triad: Rupture of membranes Painless vaginal bleeding, Fetal bradycardia

-Uterine Rupture: Complete: Includes peritoneum
Incomplete: without peritoneum

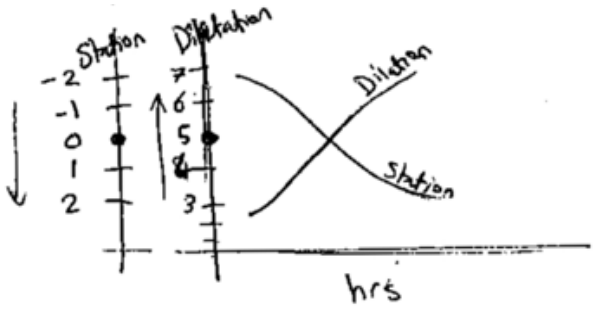
Signs and symptoms:

Maternal	**Fetal**
Abdominal Pain (severe and sudden)	Fetal Distress
Hypovolemic shock (decreased blood pressure, tachycardia, anxiety)	Loss of station
Chest Pain (due to irritation of blood below diaphragm)	You can feel the body parts of the fetus on the abdomen
Absent contraction	

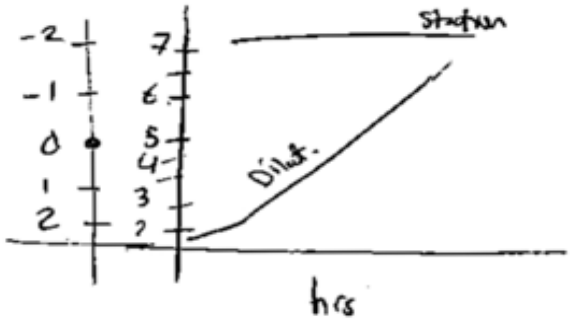
Partograph


Name
Gravida
Para
Hospital Name
Date of Admision
Time of Admision
Ruptured Membranes
Hours
Hours 1 2 3 4 5 6 7 8 9 10 11 12
Fetal Heart Rate / Minute
190 180 170 160 150 140 130 120 110 100 90 80 70 60
Liquor
Effacement
Cervix (cm) (plot x)
10 9 8 7 6 5 4 3 2 1 0
Alert
Action
Descent of Head (plot 0)
-3 -2 -1 0 1 2 3
Hours
Time
Contractions per 10 mins
5 4 3 2 1
Oxytocin U/L drops/min
Drugs Given and IV Fluids
Pulse and BP
180 170 160 150 140 130 120 110 100 90 80 70 60
Temp C
Protein
Acetone
Avolume
Outcome:
Name:
Signature:

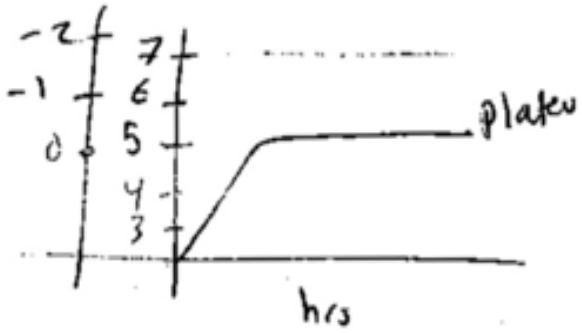
Normal curve (crossing)



Failure to descend



Failure to progress



Cesarean section C/S

Definition:

It is the delivery of the fetus and the placenta through an incision in the abdomen and uterine walls.

Incidence:

It is the most common obstetrics operation in the USA.
Increased due to:

- Raised awareness of the seriousness of fetal distress/death (FD)
- Increased average maternal age.
- Decreased use of forceps.
- Increased number of primary C/S which is an indication for repeated C/S.
- Socioeconomic factors (Doctors profit from doing C/S).

C/S has **no** increased risk on the fetus but **increased** maternal morbidity/mortality.

Types:

Classical C/S -not used anymore unless indicated-
It is a vertical incision in the upper uterine segment (contractile type)

Indications:

1. Prematurity because the lower uterine segment (LUS) develops at 28wks.
2. Fibroid or malignancy obstructing LUS.
3. Transverse lie.
4. Cervical CA (decreases risk of dissemination)
5. Dead mother because it's faster.

Lower Uterine Segment C/S (LUSC/S)
Incision is made in the non contractile part making it the uterine incision of choice, can be vertical or **transverse** which is better due to the lesser risk of vertical extension into the upper segment.

Disadvantages of classical C/S	**Advantages** of LUSC/S	**Disadvantages** of LUSC/S
Increased risk of rupture	Less likely to rupture	LUS has to be formed
More bleeding	Less bleeding	Longitudinal fetal lie
More adhesions	Less adhesions	Risk of bladder/ureter injury
Less healing	Better healing	Risk of extension to cervix, vagina, uterine vessels.
VBAC is unsafe	VBAC is ok after	

Indications for C/S: the most common indication for C/S is repeated C/S but the most common indication for primary C/S is FD.

For urgent C/S:

- Fetal
  - FD (most common)
  - Cord prolapse

- Maternal
  - Severe bleeding
  - Impending maternal death (unstable)
  - Severe PET
  - Uterine rupture

For elective C/S: before onset of labor or before the appearance of complications.

- Fetal
  - FD during labor (in cases of post maturity/HTN/ prolonged labor).
  - Malpresentation (brow / shoulder / face / breech).
  - Gross prematurity (26-32 wks) fetus is prone to intracranial hemorrhage 2ry to hypoxia during vaginal delivery.

- Maternal
  - Previous incision (2 previous LUSC/S or 1 previous classical C/S) or history of myomectomy with entrance to uterine cavity.
  - Placenta previa except type 1 (low lying placenta)
  - Vasa previa
  - Cephalopelvic disproportion (CPD)
    Elective C/S if hx of CPD

Urgent C/S if trial of labor has to be terminated due to FD/abnormal cxns.

- Active HSV infection
- Cervical CA or fibroid in the pelvis
- Previous successful repair of vesicovaginal/ rectovaginal fistula
- Bad obstetrics hx and several stillbirths
- Uterine inertia
- Severe placental abruption and baby is still alive

Contraindications:

1. Absence of an appropriate indication.
2. Dead fetus or fetal abnormalities incompatible with life.
3. Lack of appropriate facilities.

Complications:

- **Immediate** (within first 24 hrs)
  - Death from anesthesia
  - Hemorrhage (>1 liter)
  - Amniotic fluid embolism
  - Paralytic ileus
  - Injury of the bladder, ureter , blood vessels, bowel.

- **Early** (day 1 – 3 weeks)
  - Secondary hemorrhage
  - Endometritis, atelectasis, UTI, wound infection, thrombophlebitis
  - DVT/PE
  - Subrectal hematoma
  - Wound dehiscence

- **Late** (more than 3 weeks)
  - Uterine rupture (LUSC/S 0.5% but classical C/S 0.7%)
  - Adhesions causing intestinal obstruction or infertility
  - Hernias & fistulas

Indications for cesarean hysterectomy

1. Uterine atony (most common)
2. Placenta accreta
3. Uterine rupture
4. Extension of a low transverse incision
5. Fibroid preventing closure

VBAC (Vaginal Birth After C/S)

Usually done 18 months after a previous C/S.

Prerequisites:

- NO maternal/fetal contraindications to labor
- Previous lower uterine segment C/S (with documentation of uterine scar)
- Informed consent regarding the risks and benefits
- Facilities to perform emergency C/S

Contraindications:

- Previous classical C/S
- Maternal/fetal contraindications to labor
- Previous lower vertical scar (unless absence of extension)

Imp: Hx taking with counseling (OSCE)

A patient who had a previous C/S came to you and wants to know your delivery options for this pregnancy.

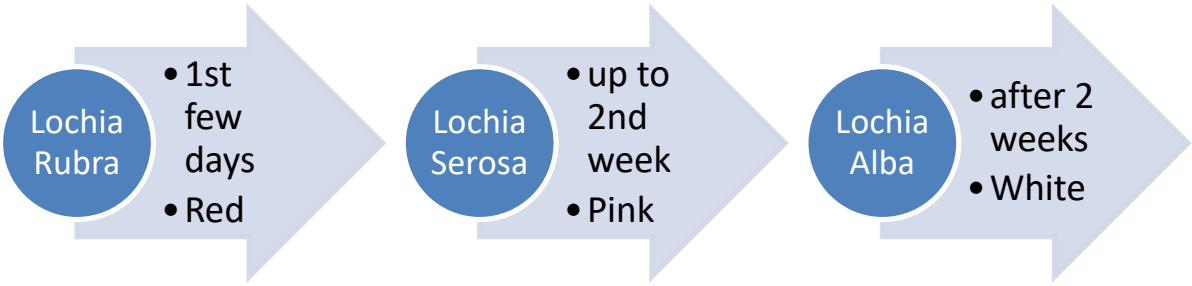
1. Patient profile
2. Ask about the previous C/S:
    - Type of C/S: classical or lower uterine segment
    - Since when? What gestational age?
    - Indications
    - Presentation? Fetal status?
    - How long was the labor? Dilation?
    - Fetal outcome?
    - Medical problems during that pregnancy
3. Ask obstetrical Hx:
    - Any other deliveries?
    - Any previous vaginal deliveries?
    - Complications?
4. Tell the patient about the risk of vaginal birth after C/S (small risk of uterine rupture)

Puerperium

It's the period of time in which organ systems return to their pre-pregnant state (6 weeks postpartum – EXCEPT the urinary system, which needs 12 weeks to return to normal).

Normal puerperium (physiological changes)

1. Reproductive System
   - Uterus
     - Involution occurs 10-12 days postpartum – the uterus returns to the pelvis due to estrogen withdrawal
     - Uterine contractions are present (to keep venous placental sinuses closed)
   - Lochia
     - Superficial layers of the endometrial decidua that are shed through the vagina during the 1st 3 postpartum weeks:



   - Cervix
     - Internal os closes in the 2nd week

2. 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)

3. Urinary System
   - Radiological studies should be delayed for 2-3 months
   - All postpartum women who cannot void should be promptly catheterized

4. GIT
   - Postpartum constipation (management: oral hydration and stool softeners)
   - Postpartum hemorrhoids (management: oral hydration, stool softeners, and sitz bath)

5. CVS
   - Risk of VTE is *higher* postpartum

Management Postpartum

- Vital signs
- Monitor vaginal bleeding and episiotomy site
- Uterine fundal height and uterine contraction
- Give analgesia

Follow up:

- Urinating/defecating/passing flatus/lactating/ambulating/lochia
- Ask about any other complaint
- Psychological support
- Postpartum contraception (remember: ovulation returns before menses, usually 3 months postpartum):
    - Lactation: anovulation for 3 months postpartum
    - Diaphragm: fit it in 6 weeks postpartum
    - IUD: place it 6 weeks postpartum (can be done immediately, but with high rate of expulsion)
    - Estrogen+Progestin OCPs: if not lactating, start 3 weeks postpartum (increases risk of VTE)
    - Progestin only (POPs or Depo-Provera): start immediately (no risk of thrombosis)

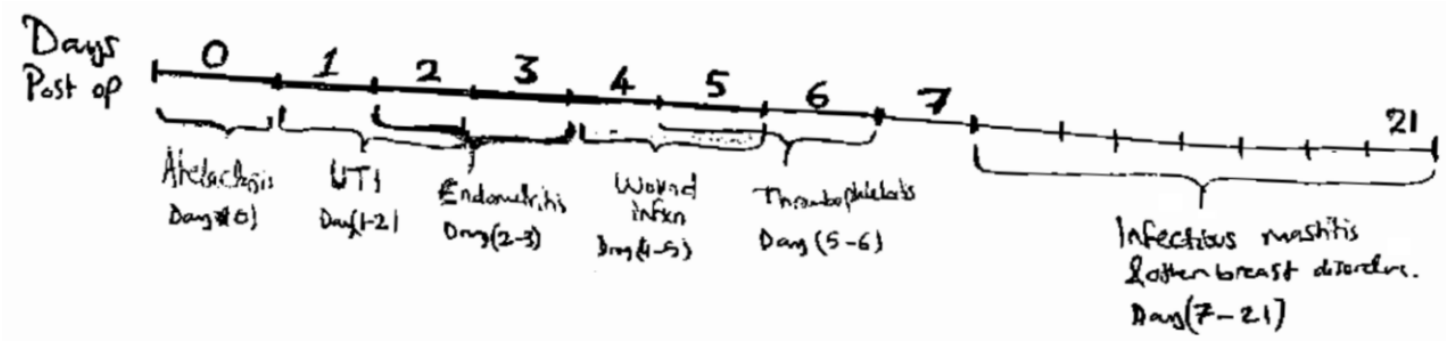
- Postpartum immunization:
    - RhoGam: if mom is Rh-ve and baby is RH+ve give 300mg within 72 hours postpartum
    - Rubella: if mom is IgG –ve, give active immunization of live-attenuated rubella virus and AVOID pregnancy for 1 month.

Postpartum Complications (Abnormal Puerperium)

1. Breast disorders
- Engorgement
  - Over-distention of the breast due to milk collection
  - 3rd day postpartum
  - Physical exam: hard and tender with nodules of enlarged breast tissue; enlarged breast covered by dilated veins.
  - Treatment: breast care (cleaning); frequent emptying by suction (manual/pump)
- Cracked nipple
  - Due to vigorous sucking by the baby and bad breast care
  - Symptoms: pain, bleeding, tenderness during sucking
  - Complications: breast abscess
  - Prevention: breast care; use breast pad; baby should *not* sleep with the nipple in their mouth
  - Treatment: AVOID feeding from the affected side (to allow healing); proper care of the breast (cleaning and suction)
- Puerperal mastitis
  - Source of infection: the baby's mouth flora has *Staphylococcus aureus*
  - Types:
    1. Cellulitis: infection in the interlobular tissue
    2. Adenitis: infection of lactiferous ducts
  - Signs and symptoms: fever, tachycardia, pain and tenderness with localized areas of hotness and erythema (affected lobe is tender and tense)
  - Complications: breast abscess
  - Treatment: continued breast feeding should be encouraged; analgesia, antipyretics, and broad spectrum antibiotics; breast care.
- Breast abscess
  - Segment of breast is tender with edema and hyperemia
  - Signs and symptoms: fever, axillary lymph node enlargement
  - Treatment: STOP breast feeding; analgesia, antipyretics, and antibiotics; incision and drainage.

2. Postpartum hemorrhage: mentioned in postpartum hemorrhage summary
3. Postpartum fever

- CAUSES: the 6 W's
  - Wind → atelectasis
  - Water → UTI
  - Womb → endometritis
  - Wound → wound infection
  - Walk → superficial thrombophlebitis
  - Weaning → breast disorders (cracked nipple/abscess/mastitis)
- The 6 W's occurrence:



Causes in details:

1) Atelectasis (day #0)
   - Risk factors:
     - general anesthesia for cesarean section
     - smoking
   - Clinical examination:
     - mild fever
     - patient is unable to take deep breaths
     - rales on auscultation
   - Management:
     - pulmonary exercises (deep breaths, incentive spirometry)
     - ambulation
   - Chest X-ray is not indicated

2) Urinary tract infection (day #1-2)
   - Risk factors:

    - Multiple intrapartum catheterization and multiple intrapartum vaginal examination due to prolonged labor
- Clinical examination:
    - High fever
    - Costophrenic angle tenderness
    - Positive urine analysis & urine culture
- Management:
    - Single dose IV antibiotics

3) Endometritis (day #2-3) – polymicrobial infection
    - Risk factors:
        - Emergency cesarean section after prolonged rupture of membranes and prolonged labor
    - Clinical examination:
        - Moderate to high fever
        - Uterine tenderness
        - Absent peritoneal signs, peristalsis should be present
    - Management:
        - Multiple broad-spectrum agents (IV antibiotics) - Gentamycin+Clindamycin
4) Wound infection (day #4-5)
    - Risk factors:
        - Emergent cesarean section after prolonged rupture of membranes and prolonged labor
    - Clinical examination:
        - Persistent spikes of fever despite antibiotics
        - Pain and cellulitis
        - Wound abscess or draining
    - Management:
        - IV antibiotics
        - Open wound and pack it
        - Closure is usually by secondary intention

5) Septic thrombophlebitis (day #5-6)

- Risk factors:
    - Emergent cesarean section after prolonged rupture of membranes and prolonged labor
- Clinical examination:
    - Persistent fever swings (spiking fever) despite antibiotics
    - Normal pelvis and physical examination
- Management:
    - IV heparin for 7-10 days keeping PTT at 1.5-2.5x of baseline

6) Infectious mastitis (day #7-21) – due to staph.
    - Risk factors:
        - Nipple cracking
    - Clinical examination:
        - fever of variable degrees
        - localized, unilateral breast tenderness, erythema & edema
    - Management:
        - Oral Cloxacillin
        - Breastfeeding can be continued
        - **Do ultrasound to rule out abscess if not responsive to antibiotics

DVT & PE

[Mentioned in medical complications of pregnancy]

Postpartum psychological reactions

- **Impaired maternal-fetal bonding**
    - Seen in the first few days post delivery
    - It's lack of interest or emotions for the newborn
    - Risk factors:
        - Increased risk when contact of the baby is limited e.g. NICU, poor social support
    - Management: psychosocial evaluation and support

- **Postpartum blues** (day #2) – first few days
  - Very common (50-70%)
  - Clinical presentation:
    - Mood swings & tearfulness
    - <u>But</u> normal physical activity and care of self and baby is seen
  - Management: conservative (social support)

- **Postpartum depression** (day #21) – onset up to 1 month
  - Common (5-15%)
  - Clinical presentation:
    - Feeling of despair & hopelessness
    - Patient does not get out of bed & does not care for self or baby
  - Management: psychotherapy and antidepressants

- **Postpartum psychosis** (day #21) – first few weeks
  - Rare! (<1%)
  - Clinical presentation:
    - Loss of reality & hallucinations occur
    - Bizarre behavior
  - Management: Admit the patient, antipsychotics & psychotherapy

Postpartum Hemorrhage (PPH)

Definition

Blood loss >500 cc in vaginal delivery and >1000 cc in cesarean section

Blood loss is usually underestimated

Classification

- Primary (early): within 24 hours of delivery
  - The most common cause is uterine atony
  - 2nd most common cause is infection
- Secondary (late): after 24 hours of delivery
  - Most common cause is infection

Incidence

- 5% of all deliveries
- It is the most common cause of death in developing countries

Causes

Causes of PPH: 5 T's
Tone
Trauma
Tissue
Thrombosis
Turned over

1. Tone: Uterine atony
   - The most common cause (50%)
   - Risk factors:
     - Overworked uterus:
       a. Rapid labor and increased oxytocin
       b. Prolonged labor
     - Overdistended uterus:
       a. Multiple pregnancy
       b. Polyhydramnios
       c. Macrosomia
     - Infected uterus: chorioamnionitis
     - Relaxed uterus (drugs):
       a. $MgSO_4$
       b. B-adrenergic agonists
       c. Halothane

- Clinical presentation: doughy uterus, above umbilicus
- Management:
  - Uterine massage (1st step!)
  - Uterotonics: Oxytocin, Methergine, PG $F_2\alpha$ (Hemabate)
  - B-lynch suture can be done if failed massage and drugs

2. Trauma: Genital laceration
   - Risk factors:
     - Uncontrolled vaginal delivery
     - Traumatic vaginal delivery
     - Operative vaginal delivery
   - Clinical presentation: lacerations and contracted uterus (rule out atony)
   - Management: surgical repair (suturing)

3. Tissue: Retained products of conception (POC) & endometritis
   - Risk factors: (for retained POC)
     - Accessory placental lobe (common)
     - Placenta accrete (rare)
   - Physical examination: missing cotyledons and contracted uterus
   - Management: manual removal of placenta or curettage under ultrasound guidance

4. Thrombosis: Obstetric DIC (rare!)
   - Risk factors:
     - Abruptio placenta (common)
     - Severe PET
     - Amniotic fluid embolus
     - Fetal demise
   - Physical examination:
     - Generalized oozing
     - Petechiae
     - Contracted uterus
   - Management: Remove POC, ICU, blood products

5. Turned over**:** Uterine inversion (rare)
Risk factors: myometrial weakness (most common), previous history 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 its normal place (by elevating the vaginal fornices) then give IV oxytocin.

6. Unexplained hemorrhage:
What to do? Ligation of the uterine vessels /internal iliac or hysterectomy**.**
Complications: chronic anemia, renal failure, Sheehan syndrome, maternal mortality.

Sheehan syndrome
Cause: postpartum hemorrhage
Leading to anterior pituitary insufficiency
1st symptom: lactation failure (prolactin)
Hormones involved: prolactin >FSH/LH> TSH/ACTH (least to be affected)

Summary:
Uterus not palpable: think of uterine inversion
Uterus like dough: think of atony
Tears vagina & cervix: think of lacerations
Placenta incomplete: think of retained products of conception
Diffuse oozing: think of DIC
Persistent bleeding: Unexplained!

Management of PPH
Corner stone is prevention. How? - Active management of second stage
- Proper management of third stage
By:
- Early cord clamping (20-30) sec of cutting

- Separation signs: lengthening of umbilical cord, gush of blood, globular shape uterus.
- Oxytocin (10 IV) after shoulder delivery & massage
- control cord traction with suprapubic pressure
- examine placenta / speculum / observe vital signs 1hr post op.

Get Help:

Stabilize /assess/ 2 large cannulas/ o2 mask /iv fluids/ cross match /blood type / prepare 2 units ➔ uterine massage / oxytocin / folly's catheter == if failed➔ Methergine/hemabate == if failed➔ manual exploration / dilation & curettage➔ vaginal ballooning / arteries ligation (uterine artery /hypogastric artery) ➔

1) If stable➔ radio consult for uterine artery embolization
2) If unstable ➔ laparotomy / hysterectomy

Investigations: CBC/ Blood group / cross match/ coagulation profile / KFT / LFT

OSCE

History: patient profile, chief complaint

- Bleeding: onset / volume (# of pads) /color/ frequency /clots / duration / tissues Dizziness / LOC /palpations / SOB --- Sx of anemia.

- **Delivery history:** mode / duration of 2nd & 3rd stage / instrumental / manual removal of placenta / anesthesia / complications / episiotomy / prolonged oxytocin.
- **Gyne history:** obstetric History gravida/para, previous PPH, previous APH.
- **Past medical history:** fever, tenderness, discharge, history of vaginal bleeding, HTN, PET, GDM, coagulopathy.
- **Past surgical history:** uterine surgeries, previous episiotomies.
- **Past social History:** smoking, alcohol.

Maternal injuries

1) Episiotomy
Incision in the perineum (skin, vagina, perineal muscles) to increase space available for delivery during second stage of labor.

Indications:
Suspected maternal and/or fetal compromise
Delivery needs to be expedited
Suspected fetal compromise
Shoulder dystocia to aid with performing internal manoeuvres
Anticipation of significant perineal and or rectal trauma

Types:

A) Midline: from fourchette down the perineal midline raphe towards the anal verge.
Layers cut: vaginal epithelium, perineal skin, transverse perineal muscles , medial fibers of Bulbospongiosus muscle.
Disadvantages: risk of anal sphincter & anal muscles injury (incontinence).
Advantages: less bleeding, better healing , less pain

B) Mediolateral
Incision at a 45^{o} angle inferiorly from midline of the fourchette
Layers cut: vaginal epithelium, perineal skin, transverse perineal muscles , medial fibers of Bulbospongiosus muscle.
Disadvantages: more bleeding and pain, less healing
Advantages: no risk of anal sphincter injury.
C) Lateral: useless, not done anymore.

Episiotomy repair:
- Good view and lightning
- prepare analgesia
- Inspection for extension

- secure bleeding
- Repair in layers: close epithelium starting 1 stitch above the apex, obliterate dead space beneath vaginal suture line.

Care:

Ice pack to decrease edema
Sitz path
Stool softener

Complications:

Extension & perineal tears
Hematoma formation
Infections
Break down
Fistula (with 4th degree tears)
Skin tags and granulation tissues
Dyspareunia and vaginismus

2) Perineal and vulvar lacerations

Precipitating factors:

1st delivery
Instrumental delivery
Unintended delivery
Precipitate delivery
Macrosomia

Classification:

1st degree: epithelium & sup epithelium tears of perineum and vagina.
2nd degree: epithelium & superficial muscles but not sphincter.
3rd degree: epithelium & superficial muscles and anal sphincter.
4th degree: reaches the rectal mucosa.

*** Cervical lacerations**

Types:

- Multiple small tears in epithelium.
- Deep lateral tears.
- Complete tears extend to lower segments.

- Annular detachment of the cervix: partial or complete:

Causes: pressure necrosis from fetal head, inappropriate suturing of an incompetent cervix, vacuum before full dilatation, labor with cervical cerclage in situ.

Management: abortion, healing by second intention.

Causes:

- Spontaneous.
- Instrumental delivery before complete dilatation.
- Precipitate delivery.

Complications:

- Cervical incompetence
- Cervical dystocia (difficult labor and delivery due to mechanical obstruction at the cervix).
- Cervical ectropion: when the soft cells (glandular cells) that line the inside of the *cervical* canal spread to the outer surface of your *cervix*.

3) Uterine rupture

Dehiscence: uterine scar separation without penetrating serosa or peritoneal cavity

Rupture: separation with penetrating peritoneum

Incidence

- Previous scar: with induction of labor➔ 0.7%
  Without induction of labor ➔0.5%
- Previous classical scar: with labor ➔ 4.7%
  Without labor ➔ 2.2%

Risk factors:
- VBAC (50-70%) ➔ most common cause
- Previous 2 scars
- Excessive oxytocin
- Dysfunctional labor
- Grandmultiparity
- RTA

Diagnosis:
Maternal anxiety
Instability and shock
Vaginal bleeding
Pain not associated with contraction

P/E :
Fetal distress or demise
Cessation of labor
Tender uterus
Signs of peritoneal irritation
Easley palpable fetal parts through abdominal wall
U/S diagnosis
Management: surgical

4) Uterine inversion

5) Paragenital hematoma
Infralevator: more painful but less dangerous
Supralevator: less painful but more dangerous

Miscarriage (abortion)

- **Definition "according to WHO"**

Expulsion or extraction of products of conception before the age of viability (<24weeks)

- **Types:**

1. **Threatened abortion**
   - Slight painless vaginal bleeding
   - No abdominal pain (because there are no uterine contraction)
   - Closed cervix
   - +) Fetal heart activity
   - 90% progress satisfactory

Complication

- High risk of preterm labor
- Antepartum hemorrhage
- Intrauterine growth restriction
- May become inevitable abortion or recurrent abortion

Management

- Admit the patient, bed rest
- Monitor fetal heart daily
- Monitor bleeding
- Monitor vital signs, presence of pain or any complain
- Give progesterone if needed
- Give anti-d
- Ask for antenatal care

2- **Inevitable abortion**
   - Heavy painful bleeding
   - Uterine contraction → severe abdominal pain
   - Opened cervix but no passage of products of conception
   - Fetal heart may or may not be present

Management

- Terminate pregnancy regardless fetal heart
- Resuscitation, Hb , Hct , iv fluid , iv cannula , 2 units of blood

Terminate of pregnancy depend on gestation age

- <12 weeks → by pervaginal evacuation (by suction)
  Don't use curette, why ? High risk of asherman syndrome and perforation, give oxytocin before suction or ergometrine
- >12 weeks → medical termination (Misoprostol (PGE1) – cytotec)

Why? Because after 12 weeks bony part start to develop → this high risk if done with suction

3- Complete abortion

- Heavy bleeding
- Sever lower abdominal pain
- Closed cervix
- Passage of all product of conception

Management

- Conservative and give oxytocin

4- Incomplete abortion

- Heavy bleeding
- Severe lower abdominal pain
- Open cervix
- Passage of some products of conception

Management

- <12 weeks → pervaginal evacuation
- >12 weeks →medical termination

To differentiate by ultrasound

- Empty uterine cavity → complete
- Retained products of conception → incomplete

5- Missed abortion

- No abdominal pain
- No bleeding
- Decrease signs and symptoms of pregnancy (decrease nausea and vomiting breast tenderness and engorgement)
- Closed cervix
- No fetal heart
- Small for gestational age

Management

- Terminate of pregnancy
- <12 weeks → by dilatation and evacuation (pervaginal)
- >12weeks by medical termination
- But keep in mind the risk of Disseminated intravascular coagulation (DIC) so we should do DIC profile (PT , PTT , D-dimer ,fibrinogen , PHs) usually happens if 1 month of missed abortion , if before 1 month → mild degree
- Due to high risk of infection Give antibiotics (prophylactic)12 hrs. prior the evacuation , Covers gram negative and gram positive → drug of choice : clindamycin and metronidazole

Diagnosis of missed abortion on ultrasound

- If two ultrasound at least 7 days apart showed embryo of >7 weeks gestational age (gest-sac > 20mm , crown to rump length > 10 mm)

Summary

- Speculum (cervical os) :
    1. Closed→ do ultrasound
        - viable → threatened abortion
        - Non-viable → missed abortion

2. Open → do ultrasound
    - gest sac intact → inevitable abortion
    - Product of consumption some left → incomplete abortion
    - Product of consumption all gone → complete abortion

Causes of first trimester abortion

- Aneuploidy (chromosomal abortion) – most common – turner syndrome and trisomy 16
- Anticardiolipin antibody or antiphospholipid syndrome (rare) → cause of recurrent abortion
  Ex : patient with SLE produce antibodies of their own vascular system and fetoplacental tissue
  Treatment: Subcutaneous heparin
- Infections : rubella / varicella
- Past medical history : DM , Thyroid
- Drugs : methotrexate , some antiepileptic drugs

Causes of second trimester abortion

- Cervical laceration (most common)
- Thrombophilia
- Uterine abnormalities
- Uterine fibroids (decrease perfect implantation)
- Infection (with or without rupture of membrane)

First Trimester Bleeding

Differential diagnosis

- Abortion
- Ectopic pregnancy
- Molar pregnancy
- Laceration
- Infection
- Non gyne causes

History

- **Patient profile, last menstrual period, gestational age**
- **History of present illness**
    - Bleeding details – amount (spotting) , numbers of pads , clots , tissues , vesicles color , onset , duration , progression , frequency , first time, Circumstance (postcoital) , trauma ?
- **associated symptoms**
    - Fever / weight loss , decrease or increase nausea and vomiting
    - Abdominal pain (SOCRATES) shoulder tip pain , unilateral
    - Labor – like pain ?
- **Gyne history**
    - Menarche
    - Menses (before) regularity , duration , frequency
    - Intermenstrual bleeding , postcoital bleeding and dyspareunia
    - Discharge (amount , color , smell)
    - Last pap smear
- **On anticoagulant ? bleeding from other orifeces ?**
- **Trauma history , procedure during this pregnancy**
- **Past medical history**
    - Dm
    - hypertension
    - Thyroid
    - SLE
    - Infection

- Fibroid

- **Past sexual history**
  - D& c
  - Trauma
  - in vitro fertilization
  - intra uterine contraceptive device
  - Pelvic or abdominal surgery
- **Drug history**
  - Anticoagulant
  - Oral contraceptive pills
  - acetylsalicylic acid (aspirin)

- **Social history**
  - Smoking and alcohol

Cervical incompetence

Definition :

It's weakness in the circular layer of internal OS (site of reflection of vaginal wall on the cervix).

Causes / Risk factors :

1. Idiopathic (Mostly)
2. Previous Hx of : Cervical incompetence / abortion / pre-term labor
   or family Hx of : Cervical incompetence / 2nd trimester abortion / pre-term labor
3. Hx of cervical injury → 1- past surgical Hx → D&C / cone biopsy / colposcopy /…
   2- Traumatic → large baby / instrumental vaginal delivery
4. Weak cervix → 1- Uterine anomalies / DES (Diethylstilbestrol) exposure
   2- collagen disease

Presentation :

1) Painless 2nd trimester abortion (with sudden rupture of membrane , gush of fluid , passage of all Products of conception)
2) Incidental routine exam
3) Bleeding / discharge / bulge / rupture of membrane
4) Recurrent 2nd trimester abortion

Deferential Dx :

• preterm labor • premature rupture of membrane • chorioamnionitis • uterine contractility

Dx :

Hx (presentation)

P/E : painless effacement on cervix exam

Investigations :
In pregnant → U/S (cervical length of funneling)

in non-pregnant → Hystosalpingogram (funneling) .. hegar dilators ((7) من الكبير للصغير)

Management :

- ❖ Previous Hx of cervical incompetence & wants to be pregnant :
  - elective cerclage (12-14 weeks) .. not before , to rule out chromosome abnormalities
  - removed (36-38 weeks)
  - expectant till delivery
- ❖ If pregnant :
  1) pre-viable :
  * expectant management * & emergent cerclage (or elective termination)
  2) viable :
  * Expectant (bed rest – Betamethasone – tocolysis (if there are contractions) * cerclage

Types of cerclage :

- ❖ Transvaginal :

1- Macdonald stitch : at cervical – vaginal junction
2- Shirodkar stitch : at the internal OS (submucosal)

- ❖ Transabdominal :

3- 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 (IUFD)

OSCE :

counseling for female who wants to be pregnant with +ve Hx of 2nd trimester abortion

- define cervix incompetence
- prophylactic cerclage , timing of insertion & removal
- types
- give folic acid
- follow up when pregnant

Premature rupture of membrane (PROM)

Definitions :

Premature ROM : ROM before the onset of labor regardless of GA
preterm ROM : ROM at < 37 weeks GA
prolonged ROM : ROM > 24 h before delivery

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 hemorrhage
6- Infection / UTI
7- Smoking / nutritional def. (zinc – vit.C)
8- Trauma / intercourse / iatrogenic (artificial rupture of membrane ARM)

Deferential Dx :

Urine → UIC/UTI
Semen → seminal collection
Blood → mucus plug
Discharge → infection

False positive Nitrazine test :

- Blood in the vagina
- Semen (sperms)
- Discharge (Trichomonas)
- UTI (proteus)
- Antiseptic use for cleaning

Complications :

1- Oligohydramnios
2- Preterm labor
3- Chorioamnionitis

4- Abruption of placenta
5- Mal-presentation
6- Cord prolapse

Dx :

Hx : (typically , gush of watery fluid reaching thighs)

P/E :
- vital signs
- fundal height → small for gestational age
-Aseptic vaginal speculum → pooling sign

U/S : Amoniotic fluid index (AFI) decreased .. But we should know what was before

Special Tests :
- nitrazine test (yellow → blue) ↑ PH
- fem test (crystals on microscope)
- Tampon test / Dye amniocentesis (invasive)
- Amnisure Test (old test) not done (dipstick that detects (placental & macroglobulin-7) protein)

Management :

- Admit & monitor (rule out infection – stable fetus ? – vital signs & P/E – labs – U/S)
- IV fluid / antibiotics (erythromycin)
- Tocolytics & dexamethasone (contraindicated in corioamnionitis & fetal distress)
- If infection is present or fetal distress → prompt delivery
- Never do PV (per vaginal exam)

Investigation & follow up :

- vital signs : tachycardia – fever – RR – BP – (rule out infection)
- P/E : abdominal palpation for tenderness and contractions
  aseptic speculum for discharge / prolapse / cervix
- Fetal heart to rule out fetal distress
- U/S to rule out Oligo & check movements & presentation

- BPP (biophysical profile)
- Investigation (ESR – CRP –CBC – WBC –urine/blood culture – vaginal swap & culture).

Aseptic speculum is used for :
- Dx (pooling test) , the patient have to cough
- rule out cord prolapse
- assess cervix (if dilated)
- High vaginal swap

In PROM , search for any **evidence of chorioamnionitis** :
- fever / uterine tenderness
- maternal / fetal tachycardia
- ↑ WBC
- foul smelling discharge
- uterine tenderness

*cut off point for management is 36 weeks (NICU حسب امكانية ال) in jordan
(34 weeks in developed countries)
it depends on :
- Amount of Amniotic fluid loss
- stability of fetus
- presence / absence of infection
if there is infection → delivery
No infection → if < 36 wks , expectant (iv fluid , antibiotics) .. if >36 wks , delivery

Risk of prematurity death is more than the risk of infection death.

Presentation :
Gush of fluid +/- Associated symptoms :
1. Abdominal pain / contractions
2. Passage of blood
3. Discharge , fever , chills & rigors
4. ↓ fetal movement
5. Urinary / intercourse symptoms

Preterm Labor (PTL)

Definition :
It's the occurrence of REGULAR contractions associated with cervical changes (dilations & effacement) after the age of viability & before 37 wks.
Threatened PTL → Regular contractions BUT no evidence of cervical changes

Incidence :
- 7-12 % of all deliveries
- >85 % of prenatal Mortality & Morbidity !
- 2/3 associated with PROM (premature rupture of membrane)

Deferential Dx :
- Braxton hicks
- UTI

Risk factors : The most important risk factor is Multiple Gestations

- **Maternal :**
  1. Wt <50 Kg (□ 3), Bad nutrition , low socioeconomic status
  2. Extremes of Age / Race (□ 2 in black)
  3. Fever , infection (UTI) , discharge , foreign body (IUCD)
  4. Pregnancy complications : previous Hx of PTL , PROM , Abortion , Abruption , PET , DM
- **Fetal :**
  1. Big Uterus : Multiple gestations (most common) / polyhydramnios / macrosomia
  2. Fetal anomalies
  3. Fetal death
- **Uterine factors :**
  1. Uterine anomalies
  2. Uterine fibroid
  3. Cervical incompetence
- **Social Hx :**
  smoking / Alcohol / Trauma / excessive intercourse (if female sensitive to prostaglandin)

- **Surgical Hx :**
  uterine / pelvic
- **Family Hx :**
  Hx of PTL , relative husband

Investigations :
- V/S (vital signs)
- Abdominal exam (fundal height , tenderness , contractions)
- Aseptic vaginal speculum (cervical dilation , pooling , discharge (culture))
- PV (if no PROM) → Bishop score
- U/S : BPP (biophysical profile) , presentation , cervical length
- fibronectin

Management :
according to Gestational age :
- if <36 wks → expectant management (iv fluid , tocolysis , betamethasone)
Except if contraindicated :
• chorioamnionitis / severe abruption • fetal distress • maternal distress
- if >36 wks → delivery

Screening & prediction : (difficult!)

1. **Fetal fibronectin test** (expensive!)
   most common cause of ↑ Fetal fibronectin is → PTL !
   also infection & stress of hemorrhage
   * if Fetal fibronectin test is +ve → ↑ risk of PTL
   between (18-36) wks → if +ve , give 1 course of corticosteroid to enhance lung maturity
   * done for high risk group (patients with short cervix , +ve fibronectin test)
2. **Cervical U/S assessment** (high sensitivity)
   normal cervical length is 2.5 cm
   if <2.5 cm → risk
   if < 1.5 cm → 26% deliver preterm <34 wks
   * routine measurement at (22-24) wks

Risk scoring
not important , based on risk factors

Prevention:

- In case of patient with cervical incompetence -> cerclage
- In case of patient with short cervix -> progesterone (might delay delivery)
- In case of patient with vaginal infection -> Treat the infection, but not recommended to give antibiotics in all patients with high risk of preterm labor.

Progesterone:

- Uses of progesterone:
  - Positive history of preterm labor
  - Short cervix
  - After acute tocolysis
- Not effective in multiple pregnancies
- Harms of progesterone:
  - High risk intrauterine fetal death.
  - High risk of abortions in <20 weeks pregnancies.
  - High risk of gestational diabetes.

Steroids: given to enhance lung activity.

- Beneficial in <34 weeks pregnancies (between 24-36 weeks)
- Effect lasts for 1 week
- Repeated courses of steroids will cause harmful effect on the brain's development, so only one dose given (2 doses max)

Tocolysis

It's the attempt to prevent contraction and progression of labor.

Aims of tocolysis:

- To allow lung maturity
- Reduces the risk of complications associated with preterm delivery (prematurity)

Contraindications:

- Absolute contraindications:
  1. Chorioamnionitis
  2. Fetal distress
  3. Fetal death
  4. Congenital anomalies incompatible with life.
  5. Sever pre-eclamptic toxemia.
- **Relative contraindications:**
  1. Intrauterine growth retardation
  2. Mild pre-eclamptic toxemia.
  3. Mild vaginal bleeding.
  4. Cervical dilation >4cm (success rate to stop labor after 4cm is poor)

Tocolytic agents:

- **Beta-adrenergic agonist (Terbutaline, Ritodrine):** not used any more (many maternal side effects)
  - Dangerous if combined with steroids -> Death
  - Causes maternal hyperglycemia -> fetal hyperglycemia+hyperinsulinemia -> neonatal hypoglycemia (after delivery)

- Side effects:
  1. Pulmonary edema (most serious)
  2. Tachycardia/headache/anxiety
  3. Glucose intolerance
  4. myocardial infarction/ arrhythmias
  5. neonatal hypoglycemia
  6. paralytic ileus/ anemia
  7. maternal death
- Must check for:
  1. CXR
  2. CBC
  3. K^+
  4. Glucose

- **Magnesium Sulfate*:* $MgSO_2$**
  - Not very effective
  - Narrow therapeutic index (5-8 mg/dl)
  - Preferable in cardiac diseases, hyperthyroidism, DM in which beta-adrenergic agonists are contraindicated
  - Used in pre-eclamptic toxemia (prophylaxis from seizures)
  - It causes vasodilation-> flushes/dizziness/decrease in temp.
  - Excreted by kidneys (contraindicated in renal failure)
  - Works at neuromuscular junction (contraindicated in myasthenia gravis)
  - Most common side effect is respiratory depression and it is the most dangerous.
  - Monitoring is critical:
    1. At 10mg/dl -> Absent reflexes
    2. At 12mg/dl-> Oliguria (monitor urine output)
    3. At 17mg/dl-> Respiratory distress syndrome and respiratory depression.
    4. At 20mg/dl -> Cardiac arrest.

- **Prostaglandin synthase inhibitor (Indomethacine):**
  - Side effects:
    1. Premature closure of ductus arteriosus (most important): so it is safe before 32 weeks, but not after because it becomes sensitive to Indomethacine.
    2. Oligohydramnios (can be used as a treatment for polyhydramnios)
    3. GI complications.

- **Oxytocin Antagonist (Atosiban)**
  - Safest with minimal side effects
  - Very expensive (but cost effective in relation to side effects.

- **Calcium Channel Blockers (Nifidipine):**
  - Side effects: headache/ flushing/dizziness.

Postdate Pregnancy

History:

General: Age, confirm gestational age, medical disease, ANC

Risk factors:

- Advanced maternal age/ DM / Thyroid problems (maternal)
- Congenital anomalies (fetal)
- Previous history of post-date pregnancy, family history, history of induction.

Complications:

- Low amniotic fluid: Oligohyramnios
- Placenta: Maturation/Thrombus/Calcifications.
- Fetus: Postmaturity and macrosomia / shoulder dislocation / increased chance of caesarian section, traumatic vaginal delivery, increased intrauterine fetal death.

Physical exam:

- Abdomen: fundal height
- PV: cervical dialation

Postdate pregnancy: >40 weeks

Post-term pregnancy: >42 weeks

Investigations:

- **U/S**
- **Fetal monitoring (non-stress test/Biophysical profile)**

Management:

Bishop Score>6

41^{+5}

→ Indications of labor

- Otherwise, conservative.

Bishop Score

A PV exam screening system to determine if labor is likely to commence or induction of labor will be required.

(DESCO)

SCORE	0	1	2	3
Dilatation	closed	1-2	3-4	>=5
Effacement	<30%	40-50%	60-70%	>=80%
Station	-3	-2	-1	>=1
Consistency	firm	medium	soft	-
Os (position of internal os)	posterior	Mid-position	anterior	-

A score of:

- <6:
  - unfavorable cervix
  - Induction is less likely to be successful
- >=6:
  - Cervix is favorable
  - More likely to have successful vaginal delivery and induction
- >8:
  - Higher probability for successful induction and vaginal delivery.

Polyhydramnios

REMEMBER origin of amniotic fluid is
-In the first 14weeks of GA: maternal plasma serum

->14weeks: fetal kidneys (mainly) and fetal lungs
-Maximum amount of amniotic fluid is on 36wks

Causes of polyhydramnios:

- **Idiopathic** (most common cause)
- **Maternal**: DM, cardiovascular diseases, infections (CMV, toxoplasmosis)
- **Fetal**: multiple gestations, GI obstruction, CNS (anencephaly) –because baby can't swallow and there's no ADH secretion in this case- , CVS (hydrops fetalis), Trisomy
- **Placenta**: chorioangioma

Complications: -remember P's**-**

- **Antepartum:** Preeclampsia (PET), placental abruption, preterm labor (most important), PROM
- **Intrapartum:** poor presentation, poor uterine contractions, prolapse of the cord, supine hypotension syndrome
- **Postpartum:** Postpartum haemorrhage (caused by uterine atony)
- **Fetal complications:** prematurity, perinatal morbidity and mortality, cord prolapse

Diagnosis :

-Clinically:
History (symptoms result from compression of uterus on adjacent structures) as follows:
-abdominal distention and discomfort (skin)

-Indigestion/ vomiting (esophagus)

-Shortness of breath (diaphragm)

-Surrounding veins (varicose veins, haemorrhoids, lower limbs edema)

On physical examination:

-Distended abdomen (large for gestational age)
-Shiny skin
-Stria
-Faint fetal heart sound and difficulty feeling fetal movement

Ultrasound:
-Amnoitic fluid index >25
-Largest single pocket >8

Classification:
Amniotic Fluid Index >25
Mild 25-30, Moderate 30-40, Severe >40

Largest single pocket >8
Mild 8-12, Moderate 12-15, Severe >15

Management:
If **severe polyhyramnios** then :
-Admit the patient + bed rest
-It's HIGH RISK PREGNANCY , do the following (OGTT to R/O DM , fetal anatomy scan, IgG and IgM titers for CMV, Rubella and toxoplasmosis (but it's not routine), Rh investigations (to R/O hydrops fetalis)
-Medical treatment: Endomethecin
-Surgical treatment: Amniotic fluid reduction (can reduce up to 5L on single setting)
-Delivery >34w (to reduce risk of preterm labor)
-Tocolytics and betamethasone if <34w

If **mild-moderate**:
-Outpatient management and according to the cause
-Follow up every 2 weeks for Amnoitic Fluid Index and fetal growth

Oligohyraminios

Causes:
-Rupture of membranes/ leakage (the most common cause)
-Post-term baby
-Renal anomalies (reduced GFR)
-Uteroplacental insufficiency (preeclampsia)
-Potter syndrome (bilateral renal agenesis)
-Dehydration of the mother
-Drugs: Endomethecin, ACEI
-Chromosomal abnormalities: Trisomy
NOTE :trisomy can cause both oligohyraminios and polyhyramnios

Complications: (usually if occurred <24w)

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

Diagnosis:
-Physical Examination
Small for GA, decreased fetal movement

-Ultrasound
AFI <5 , deepest single pocket <8

Management:
-According to GA, severity and cause
-If <24wks and severe then termination of pregnancy
-If >24wks: Mild: follow up for signs of chorioamnionitis, AFI, fetal movement
Severe: hydration, dexamethasone, amnioinfusion
(transcervical intrauterine catheter)

DDx for Small for gestational age (SGA):

-Wrong date
-Rapture of membranes/leakage
-Oligohydramnios
-Transverse lie
-Intrauterine growth restriction (IUGR), fetal anomaly
-Intrauterine fetal death (IUFD)

DDx for large for gestational age (LGA):
-Wrong date
-full bladder
-Fibroid or any mass (mole,ovarian mass, Ca)
-multiple gestations
-Macrosomia
-Polyhyramnios
-Placentomegaly

Small for GA and Intrauterine Growth Restriction

-SGA is when Estimated Fetal Weight (EFW) <10^{th} percentile for a given GA (<2.5kg)

- Majority are healthy
- Familial in 85% of cases

-IUGR is another term used for abnormal SGA

SGA is classified into:
Normal (no structural abnormalities, normal liquor and normal umbilical artery Doppler)
Abnormal (structural abnormalities), **types :**

-Symmetrical (due to fetal causes) –decreased POTENTIAL
Both head and body are equally affected

-Asymmetrical (due to maternal/placental causes) –decreased SUBSTRATES
the body (abdominal circumference) is small while head (head circumference) is preserved due to shunting of blood to brain through foramen ovale
here the insult occurs LATE in pregnancy

Causes of IUGR:

Fetal (remember symmetrical)
Anomalies, trisomies, infections (TORCH, syphilis, malaria, varicella), polyhyramnios, oligohydramnios, uterine overcrowding (due to fibroids or multiple gestations)
-here the baby has decreased potential to grow, increase feeding will not help

Maternal (usually late)
Medical history**:** HTN, preeclampsia, DM, CVD, Respiratory, Renal, SLE, Sickle cell anemia
Social history: poor, malnutrition (low BMI), infections, smoking, alcohol
Drug history: Chemotherapy, radiotherapy, teratogenicity drugs (antiepileptic, Beta-blockers)

Family history and Prevois hx of IUGR

Placental (usually late)
placenta previa, placental abruption, thrombosis, calcifications, TTTs (twin-twin transfusion syndrome), circumvallate placenta

Complications:
Respiratory: meconium aspiration, asphyxia
Electrolytes/ blood: Polycythemia, increased Bilirubin, decreased Calcium, decreased Glucose
Life manifestations : mental retardation, fetal distress, Intrauterine fetal death

Approach:

History (analyse predisposing factors)
Physical Examination: Vitals, fundal height, Leopoid manoeuvres, sonicode (for heart sounds)
Investigations: SERIAL U/S (the most effective way in detecting growth restriction)
(Biometry, NST, BPP, umbilical Doppler, MCA Doppler)

Management:
-In general for prophylaxis: Aspirin, nutrition, stop smoking and antimalarial drugs
-Lifestyle changes (nutrition, rest, stop smoking)
-**Baby Aspirin** (for prevention)
-**Fetal monitoring** (weekly) NST, Biometry, BPP

*if continues growth then conservative
*if static growth then delivery (C/S or normal vaginal delivery)

NOTE: fetal weight on U/S is determined by (U/S biometry):
BPD (biparietal diameter)
HC (head circumference)
AC(abdominal circumference)
FL(femoral length)

Large for Gestational Age (LGA)

When EFW< 90-95th percentile for a given GA
-accuracy in estimating body weight is poor
-Errors in predicting fetal weight is +/- 400gm

What's the difference between LGA and macrosomia?
LGA is as above defined
while macrosomia is (LGA + organomegaly+ fat deposition on upper back and belly) but these two terms are mostly used interchangeably.

Risk factos:

-Gestational DM, overt DM
-prolonged gestation (postdate pregnancy)
-increaed BMI (obesity) , increased weight gain during pregnancy
-multiparity
-male fetus

Complications
Maternal:
-operative vaginal delivery
-perineal lecerations, pelvic floor injury leading to urinary incontinence and pelvic organ prolapse
-emergency C/S
-Postpartum haemorrhage (due to atony)

Fetal:
-shoulder dystocia
-asphyxia
-birth injury

Neonatal:
-NICU admission
-Erbs palsy
-hypoglycemia

Prevention
There's no accurate way for prediction/prevention

Management
-Elective C/S if EFW >4.5kg in diabetic mother or >5kg in non-diabetics
-Early induction (increased failed induction)

Multiple Pregnancy

Number of twin pregnancies increased due to increased use of assisted of assisted reproduction techniques

DDx for fundal height large for date:
1. Wrong date
2. Full bladder
3. Mass (fibroid, CA, molar pregnancy, ovarian mass)
4. Multiple gestation
5. Polyhydramnios
6. Macrosomia
7. Placentomegaly

Types:

1. **Monozygotic (identical):**
   - Dichorionic diamniotic ➔ DCDA (1 placenta, 2 amniotic sacs)
   - Monochorionic diamniotic ➔ MCDA (1 placenta, 2 amniotic sacs)
   - Monochorionic monoamniotic ➔ MCMA (1 placenta, 1 amniotic sac)
   - Conjoined twins

2. **Dizygotic ➔ <u>more common than monozygotic</u>**
   - <u>ALWAYS</u> dichorionic diamniotic (DCDA)

DDx for elevated B-hCG:
1. Wrong date
2. Multiple pregnancy
3. Molar pregnancy
4. CA (Choriocarcinoma)

Risk Factors:

1. Race ➔ more common in blacks
2. Age of parity ➔ older age of parity
3. Family history
4. Ovulation induction
5. Conception after cessation of OCPs

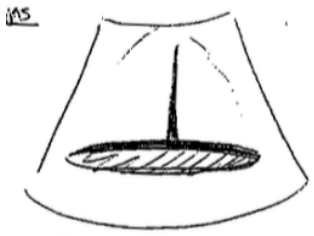
➔ ***<u>Note:</u>*** dizygotic twins have identifiable risk factors while monozygotic twins have NO identifiable risk factors.

<u>Comparison between Dizygotic & Monozygotic:</u>

	Dizygotic	Monozygotic
Incidence	More common in multigravida	More in primagravida
Age	Older age group	20-35 years
Placenta	2 separate	Variable
Sex	May/may not be the same	Some sex
genders	different	Identical

Diagnosis:

- By ultrasound ➔ demonstrates more than one intrauterine fetus
- Special signs:



- T- sign seen in monochorionic twins
  i.e. one placenta

- λ - sign seen in dichorionic twins
  i.e. two placentas

➔ If not T or λ sign, DDx:

- MCMZ
- MC conjoined

Lab findings:

1. Increased B-hCG
2. Increased MS-AFP

Postconception dates to identify twin cleavage:	
DCDA	0-3 days/ Morula
MCDA	4-8 days/ Blastocyst
MCMA	9-12 days/ Embryonic disk
Conjoined	>12 days / Embryo

Complications:

In preterm birth ➔ Give prophylactic steroids
DON'T give tocolytics and cerclage

- **Maternal:**
  1. Hyperemesis Gravidarum
  2. Nutritional Anemia (iron and folate deficiency)
  3. PET / Gestational DM
  4. Preterm labor/ cervical incompetence and increased risk of abortion
  5. Increased C/S
     - **MCMA** is a relative indx for C/S because of risk of cord prolapse
     - **95%** of twins are delivered by C/S
  6. Postpartum hemorrhage/ Antepartum hemorrhage

DC ➔ we usually deliver at 37 weeks

- **Fetal:**
  1. Spontaneous abortion
  2. Prematurity and PROM / SGA
  3. Polyhydramnios
  4. Malpresentation
  5. Congenital anomalies
  6. Twin-Twin Transfusion Syndrome (TTTS)
  7. Cotwin death

Most common complications are:
1. PTL (30%)
2. PROM
3. PPH
4. Operative delivery

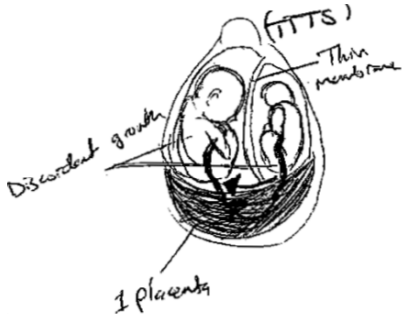
Management:

- **Antepartum:**
  1. Give iron and folate supplements (to prevent anemia) and bedrest after 28 weeks
  2. Monitor BP for PET
  3. Educate mother regarding signs and symptoms of PTL and cervical length measurements
  4. Serial U/S looking for TTTS
- **Intrapartum:**
  1. NVD if both are cephalic or 1st is cephalic
  2. C/S if 1st is non-cephalic or transverse lie
- **Postpartum:**
  1. Watch for PPH (increased risk of atony) ➔ active management of 3rd stage

Some complications in detail:

- **Fetal death:**
  1. **1st Trimester ➔**
     - "Vanishing twin" 75% asymptomatic
     - If occurred <14 weeks there is no risk to the surviving twin
  2. **2nd Trimester ➔**
     - No problem if DC
     - Increased risk of DIC
     - <u>If MC management is:</u> 1. Conservative 2. Selective fetocide 3. Termination of pregnancy 4. Delivery

- **Twin-Twin Transfusion Syndrome:**
  - Only in IDENTICAL twins
  - Mechanism: imbalance between blood flow



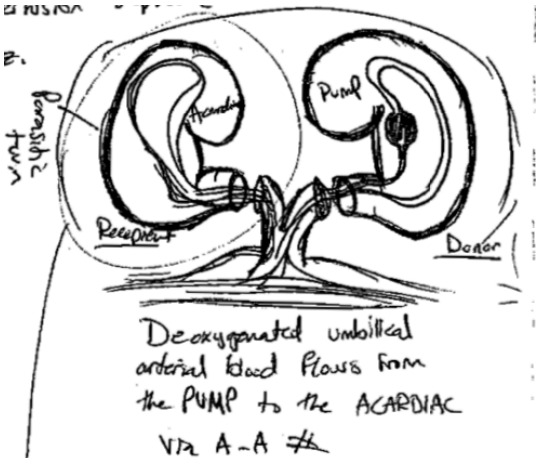
  - ♦ Features:
    1. SINGLE placenta
    2. Thin membrane
    3. Same sex
    4. Discordant growth

Donor	Recipient
Oligohydramnios	Polyhydramnios
Empty bladder	Full bladder
Anemia	Polycythemia (plethora)
IUGR and RF Blood is shunted to vital organs but NOT Kidneys	Hypervolemia - Cardiac: hypertrophy - Hydrops
Velamentous cord	

  - ♦ Stages of TTTS:
    1. Poly and oligo
    2. + empty bladder
    3. + abnormal flow in the umbilical cord
    4. Fetal hydrops
    5. One twin has died
  - ♦ Management:
    1. Selective cord coagulation
    2. Serial amnioreduction (to reduce risk of PTL)
    3. Fetoscopy and laser ablation

- **Conjoined Twins:**
  - BAD prognosis, most of them die
  - Mostly premature delivery by C/S
  - ♦ Diagnosis by U/S:
    a. Monoamniotic twin
    b. Twins face each other
    c. Heads at the SAME level and hyperextended
    d. No change in position with movement

- **TRAP ➔ Twins Reversed Arterial Perfusion sequence**
  - Can occur in both mono and dizygotic
  - ♦ Complications:
    a. PTL (75%)
    b. Polyhydramnios (50%)
    c. Death (of donor)
    d. Poor outcome (in acardiac)
  - ♦ Management:
    a. Conservative ➔ serial BBP and echo
    b. Invasive ➔ amniocentesis, hysterotomy, cord occlusion
  - ♦ Types of Acardiac Twins:
    a. Acephalic ➔ no head or chest
    b. Acormus ➔ ONLY head
    c. Anceps ➔ no heart
    d. Amorphus ➔ not human shape



Average of GA:
1. Twins ➔ 35 weeks
2. Triplets ➔ 32 weeks
3. Quadruplets ➔ 28-29 weeks

Rh- Isoimmunization

- Aka: Rh-disease/Rh-incompatibility/RhD hemolytic disease of the newborn (RhDHDN)
- Rh factor is an RBC surface antigen
- It occurs when maternal antibodies are directed against RBC surface antigen
- RBC surface antigens: D,c,C,e,E
  ➔ the most antigenic is D

Pathophysiology

1. Blood of the fetus may leak into maternal circulation (or from blood transfusion) and after significant exposure, sensitization occurs leading to antibody formation against foreign RhAg (primary response/IgM)
2. Once produced, maternal Rh IgG (secondary response) may cross freely from placenta into the fetal circulation ➔ Ag-Ab complexes ➔ Autoimmune-induced hemolytic anemia

Mechanism/ Cause:

1. Fetomaternal hemorrhage (MCC) ➔ when mom is -ve and the fetus is +ve
2. Blood transfusion

Screening test:

➔Indirect coombs test

Neonatal outcome:

➔ varies from mild jaundice to erythroblastosis fetalis

Risk Factors for sensitization ➔ anything that increases risk of fetomaternal bleeding

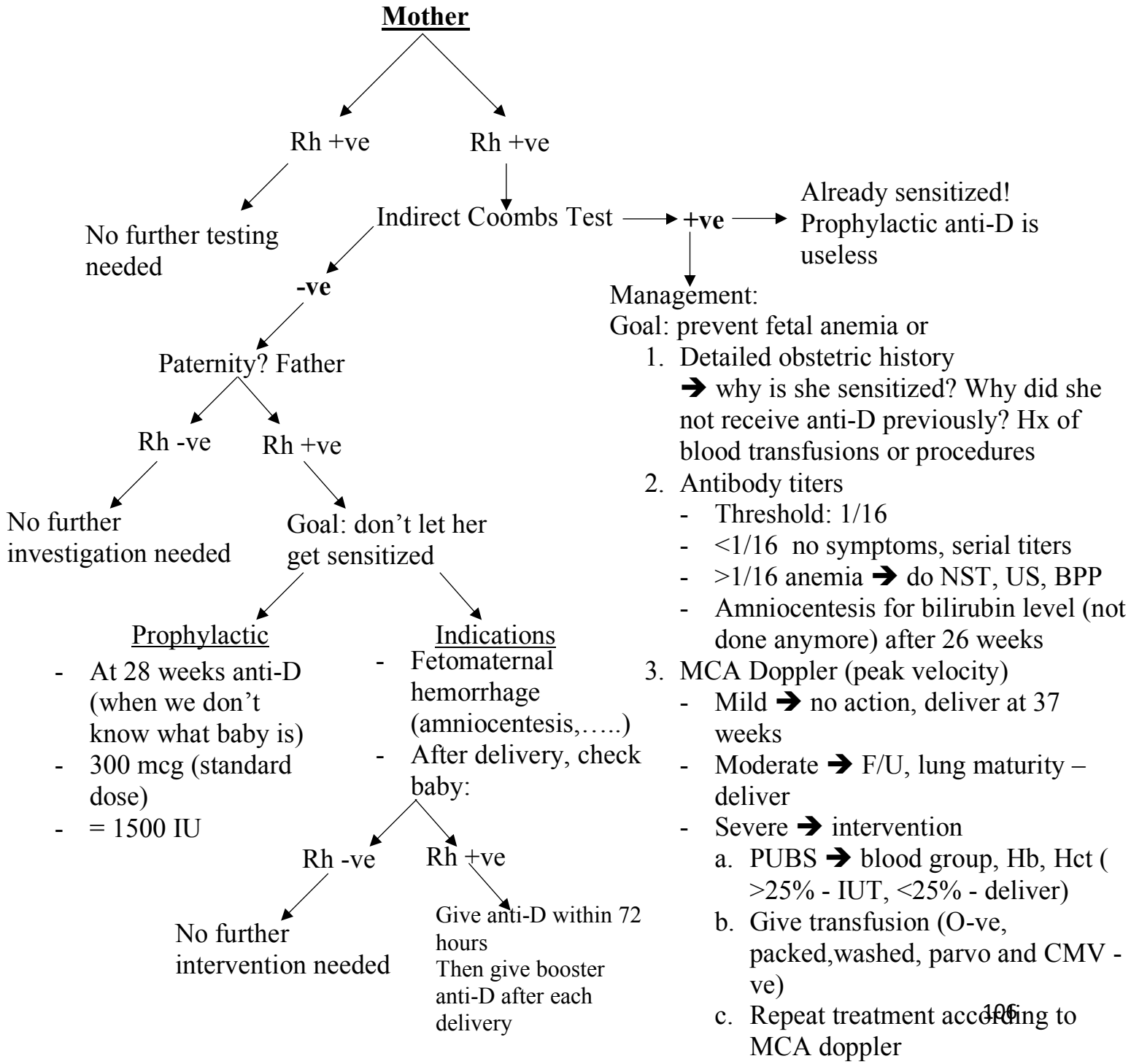
1. Abortions / D&C
2. Molar / ectopic pregnancy
3. Manual removal of the placenta
4. APH (abruption, previa)/ RTA, trauma, falling
5. Procedures: amniocentesis, chorionic villous sampling
6. Blood transfusions

ABO Incompatibility is PROTECTIVE against Rh-incompatibility!
Risk of Rh-disease after ABO Incompatibility is ONLY 1-2%

Rh-immune response:

1. **Primary**
   - **a.** Slow in development
   - **b.** 8-9 weeks (up to 6 months) BEFORE it can be detected after exposure
   - **c.** Usually WEAK, mainly IgM (large MW so it cannot cross the placenta or cause hemolysis)
2. **Secondary**
   - **a.** 2nd exposure to Rh + RBC
   - **b.** Rapid increase in Rh-antiD (IgG) (Small MW so it can cross the placenta and cause hemolysis)

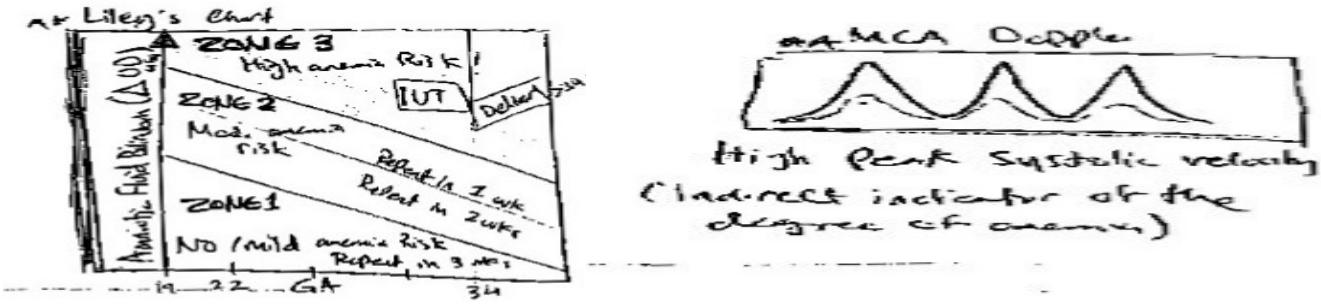
Rh incompatibility Management



ndirect combs' test (AAT Atypical Antibody Titers):Serum being tested for the presence of anti-D mixed cells ,if anti-D present it will adhere to Rh+ cell membrane.

Kleihevr-Betke Test : If there is an index for anti-D (mixing ,fetomaternal bleeding),this test quantifies how much fetal RBCs ;to know anti-D dose .

15 fetal RBCS >>>200 IV (standard dose),so you know you need a higher dose.



Notes:

-Bilirubin titers can reflect the degree of anemia if it is hemolytic anemia but we depend on MCA “middle cerebral artery “ doppler (less invasive).

-This affect the 2nd baby if the mother is sensitized ,unless she is sensitized by something other than normal labor (ex. blood transfusion)OR if there is a large amount of fetomaternal hemorrhage.

-Titers of 1/8 < 1/32 ,1/32 means you had to dilute it 32 times to clear the antibodies . Titers threshold is hospital based.

OSCE station: A 32 year old female patient pregnant ,come to you clinic telling you that her blood group is A- and her husband is AB+, she was told to be sensitized.

Take a proper history and what will be your management ?

-GP: LMP ,GA ,Blood group for her and her husband , hx of blood treatment ,ask also about her previous fetuses if she is multipara.

How much was the titer of combs' test فحص الأجسام المضادة ?

Did your husband do DNA analysis before?(Homo/heterozygous)

Hx of current pregnancy : antepartum hemorrhage ,abruptio placentae ,placenta previa ,RTA/trauma (falling ,procedures),Amniocentesis ,PUBS ,blood treatment.

Past obs hx: Antibiotics use, molar ,ectopic,C-S,D&C, manual removal of Hx of IVFD ,APH, hydrops, stillbirths,delivery of jaundiced baby,NICU, neonatal blood tx, hx of taking anti-D.

Hx of blood treatment.

Management as mentioned before…

-Causes of fetal anemia :

Immunogenic : Rh – immunization

Non-immunogenic:

1.Parvovirus B19.

2.Alpha thalassemia.

3.TTTS(Twin to Twin Transfusion Syndrome).

4.Feto-maternal hemorrhage.

5.Fetal and placental tumors.

-NST findings of fetal anemia :

1.Tachycardia.

2.decrease variability.

3.Sinosoidal pattern.

Contraception

-Definition: International prevention of conception.

-Methods:

- Natural Methods (periodic abstinence, Coitus interruptus, Lactational amenorrhea).
- Barrier methods and spermicides(Male condoms, Female condoms, diaphragm, cervical cap, and spermicides)
- IUCD
- Hormonal methods
- Surgical sterilization (Female sterilization by tubal ligation and male sterilization by vasectomy)

Notes:

- No contraception method /sterilization is 100% effective.
- Efficacy rate :theoretical :the efficacy when used as instructed, and actual: the efficacy when used in real life assuming the variation in consistency of usage.
- Patient's choice of methods depends on efficacy, cost, safety, and religious view.

- Significance of contraception :

1.decrease unintended pregnancies and abortions.

2.provides health and social benefits for mother and children.

3.decrease risk of post partum depression (decrease unwanted pregnancies).

4 Therapeutic benefits : (heavy menses, Acne, hirsutism, endometriosis, decrease risk of endometrial and ovarian cancer).

Methods in details :

1. **Natural methods** – the least effective one
   - Periodic abstinence: rhythm or calendar method

MOA: It emphasizes fertility awareness and abstinence shortly before and after ovulation period…..ovulation assessment method: 1.ovulation prediction kits(detects LH surge) 2.Basal body temperature 3.menestrual cycle tracking 4.cervical mucus evaluation.

Effectiveness :relatively low (50-80%).

Advantages :Uses neither chemical nor mechanical barriers.

Disadvantages :1.needs understanding of physiology 2.regular periods 3.not effective.

- Coitus interruptus :

MOA: withdrawal of penis from the vagina before ejaculation so the majority of semen is deposited outside the genital tract.

Effectiveness :failure rate is high 27%.

Disadvantages :1.High failure rate 1.needs self control.

- Lactational amenorrhea :

MOA: prolactin – induced inhibition of GnRH from hypothalamus resulting in suppression of ovulation.

Criteria :1.Amenorrhea 2.The infant must exclusively breast-feed 3.only for 6 months

The infant must breast feed at least 4 hours per day and 6 hours per night.

Can be combined with POP(progesterone only pills).

Advantages :1.No cost 2.No effects on nursing

Disadvantages :Actual efficacy rate is low.

Notes: - Return of ovulation occurs BEFORE return of menses.

-50% of lactating mothers will begin to ovulate 6-12 months after delivery.

2. **Barrier methods and spermicides** :

MOA :mechanical obstruction

A.Male condoms:

- Types: - Latex (m.c, inexpensive) –polyurethane (newest, expensive)
- Effectiveness : theoretical 98% and increased by spermicides

  Actual 85-90%
- Advantages :1.low cost 2.No STDs except for HPV and HSV.
- Disadvantages : 1.decrease sensation 2.hypersentivity from latex 3.may rupture
- Notes: - It's important to leave a space at the tip to collect the ejaculate and decrease leak.

  -The only methods that protect against STDs male and female condoms.

B. Female condoms:

- polyurethane.
- Must not be removed for 6-8 hours after intercourse.
- Effectiveness :failure rate higher than male condoms by 25%, effectiveness id 80%
- Advantages :1.No STDs (except HPV and HSV) 2.Self-induced
- Disadvantages :1.increase cost 2.bulky

C. Diaphragm:

- Dome-shaped.
- Placed into the vagina before the intercourse and left placed 6-8 hours after it.
- Effectiveness :80%
- Advantages : 1.Lasts for 2 years.
- Disadvantages :1.must be inserted by clinician 2.hypersentivity to latex
- Complications :1.increase risk of vaginal tract injuries2.colonization of staph, may leads to toxic shock syndrome.

D.Cervical cap:

- Silicon cap that fits directly over cervix
- Held in place by suction.
- Effectiveness :80%
- Disadvantages :1.Inserted by clinician
  2.Dislodgment(common cause of failure)
  3.Effectiveness depends on female parity (failure in Para).

B>Spermicides :used adjunct with other barriers

- Forms: Creams, gels, suppositories.
- Types : 1.Nonoxynol – 9 2. Octoxynol – 9
- MOA:-disrupts cell of spermatozoa - Acts as a mechanical barrier.
- Placed in the vagina 30 minutes before intercourse.

3. **IUD** :

- The most widely used method of reversible contraception in the world.
- Types: 1.Paragard (Copper) 2.Mirena (progesterone – only)
- MOA:-Cause sterile inflammatory reaction – prevent implantation –decrease tubal motility and increase cervical mucus thickening.
- Effectiveness : - Paragard 99.1% while mirena 99.9%.

Advantages:

- Long term contraception (Paragard used for 10 years (up to 14 years) Mirena used for 5 years (up to 7 years)
- Effective
- cost-effective
- early reversibility
- can be immediately inserted after spontaneous abortion in the first trimester.
- Less risk of ectopic pregnancy

Disadvantages / side effects:

- Risk of expulsion (1st year)
- Inserted by physician

- Pain, bleeding and infection
- Perforation at time of insertion

Indications:

- When OCPs or hormonal contraception are contraindicated
- Long Term protection
- Low risk of STD
- Menorrhagia/ dysmenorrhea

Contraindication:

- **Absolute** :
  - Pregnancy, bleeding, infection
  - Copper sensitivity (paragrad) / Wilson disease
  - Endometrial Carcinoma
  - Molar pregnancy

- **Relative**:
  - Previous history of ectopic pregnancy
  - Previous history of STD in 3 months
  - Anomalies/ fibroid
  - Nullipara.

4. Hormonal contraception:

- **Combined (estrogen+ progesterone):**
  - OCPs
  - Transdermal patches
  - Vaginal ring

- **Progesterone only methods**
  - Minipills (Pops)
  - Depoprorera
  - Implanon

Combined :

1. OCPs: (estrogen+ progesterone)
   Most contain low dose Ethinyl Estradiol (20- 35 mg) (lowest dose is 15 mg) PLUS Progestin.
   - Estradiol (natural estrogen) is not orally effective.
   - Ethinyl esradiol (synthetic estrogen) is orally effective.

- **MOA**:
  - Interfere with the release of FSH and LH (causes pseudo pregnancy state) that suppress the ovulation.

    (Estrogen decreases FSH > so decreases follicular growth)

    (Progesterone decreases LH> so decreases ovulation)
  - Thickening of cervical mucus (which causes also mechanical contraception)
- **Doses**:
  - Monophasic: Fixed dose of estrogen and progesterone. In each regimen 21/7
    Complications: withdrawal bleeding begin within 3-4 days after completing 21 days.
  - Multiphasic: varies in the dose in each pill to mimic the menstrual cycle
    High effective, less S/E, but expensive.
- **Effectiveness**:
  99.8% administered 1*1, 21 days, 7 placebo
- **Side Effects** :
  1. **Estrogen related:**
     - CVA/ MI/ PE/ DVT
     - N/V/ MIGRAINE/ headache and tiredness
     - Fluid retention/ bloating
     - Breast changes(tenderness, enlargement)
     - Loss of Lipido, cervical changes and CA.
- **Progesterone related:**
  - breakthrough bleeding/ irregular bleeding
  - acne/ baldness/ weight gain/ irritability and depression
  - hypertension/ cholestasis/ breast tenderness.

<u>Non contraceptive uses of oral contraceptive:</u>

1. Helps in Menorrhagia, dysmenorrhea endometriosis, dysfunctional uterine bleeding and treatment of ovarian cysts.
2. Decrees risk of PID and infection (MOA: thickness of cervical mucus)
3. Decrees risk of ectopic pregnancy.
4. Decrees risk of ovarian/ endomaterial and colon CA.
5. Relieves the anovulatory symptoms: acne and hirsutism.
6. Decrees osteoporosis and benign breast disease.

- **Oral contraceptive and malignancies:**

increases the risk of cervical and breast carcinoma and decreases the risk of ovarian, endomaterial and colon cancer.

-Contra-indications:

- **Absolute**:
  - smoker more than 15 cig/day and age more than 35.
  - VTE, PE, CAD, CVA(Venus risk more than arterial risk)
  - uncontrolled hypertension, hypertension with vascular disease.
  - Known or suspected pregnancy, lactating, breast CA.
  - Migraine with aura.
  - abnormal LFT.
  - Breast/ endometrial CA.
  - SLE.
  - Undiagnosed vaginal bleeding.

- **Relative contraindications:**
  - Smoker less than 15 cigarettes per day and age more than 35.
  - Hypertension/ hyperlipidemia/ DM with vascular diseases.
  - Lactating less than 6 months.
  - Viral hepatitis.
  - Treated breast CA more than 5 years without recurrence.
  - Obesity (IBM I more than 35)
  - Migraine without aura.

Note: The risk of venous thrombosis is unaffected by age/ smoking/ duration of use, but affected by BMI and hypertension.

The risk of arterial thrombosis is more affected by smoking and age.

Drug interaction:

- Decrease efficacy of oral contraceptive : phenytoin, barbiturates, Tegretol, Rifampcin.
- Increase efficacy oral contraception methyldopa, Diazepam, TCA, Theophillin.

Missed pills: > 24h

	First 7 days	Second 7 days	Third 7days
One pill	As soon as possible she remembers (next pill at the usual time)		
2 pills	Take one pill (only one) as soon as remembered & continue the pills as usual + additional contraceptive method for 7 days (eg., condoms) + if intercourse occurred in the last 2 days of the 1st 2 weeks (i.e day 13 /14); take an emergency pill		Start new pack + methods of emergency pills in the second 7 days
3 pills	Start new pack + methods of emergency pills in the second 7 days		

Transdermal patches: ortho extra

- continuous release of Ethinyl estradiol and progesterone.
- Effectiveness more than 99% but decrease in Over weight female.
- One batch/ week for 3 weeks then 1 week withdrawal bleeding
- Many causes skin irritation.

Vaginal ring:

- Release daily doses of Ethinyl Estrogen+ Progesterone.
- effectiveness is 98%
- placed in the vaginae for 3 weeks Then removed for one week for withdrawal bleeding.
- Disadvantage: inserted by clinician, discomfort, headache, vaginal discharge recurrent vaginitis

Progesterone only methods:

1. **Minipills** (POPs):
   - progesterone without any estrogen.
   - Lower dose of progestin than in combined.
   - Higher failure rate, effectiveness is 92%.
   - Administration 1 * 1 for 28 days.
   - MOA: cervical mucus thickening, ovulation suppression, endometrial atrophy.

- Indications: when combined OC b's are contra-indicated and lactating mothers.
- Contra-indication: pregnancy suspected or known and undiagnosed bleeding.
- side effects: breakthrough / irregular bleeding, increase follicular cyst, Acne, hirsutism, weight gain, irritability and depression, breast tenderness.

Injections = Depoprovera:

- administration: IM every 3 months and it is the most effective 99.7%
- disadvantages: many cause Amenorrhea then they may cause infertility as long use suppresses the ovulation, decrese bone density (reversible), same a/e of progestin.
- After D/c of injections, they may experience delayed in ovulations (6-8 months)

Implants = Implanon

- Administration: SQ effective 24 hour after placement.
- Provides 3 years of contraceptive coverage.
- Advantage: implantable has a quick return to fertility after removal
- Disadvantage: inserted and removed by physician, side effects of Progestin.

<u>Emergency contraception:</u>

- it prevents pregnancy after unprotected intercourse or in case of contraceptive failure but it doesn't prevent implantation.
- **Types**:
  1. emergency contraceptive pills: high dose of estrogen and progesterone.
     Effectiveness: 89% taken with 72 hours after unprotected intercourse.
     Advantage: short window period and not used for long term contraception.
     2. Emergency IUD insertion: the most effective type.
     nearly 100% effective.
     Inserted within 3 to 5 days of unprotected intercourse.

Disadvantage: inserted by clinician, infection, perforation and bleeding.

Advantage: long term contraception 10 years and more effective.

Surgical sterilization:

Types	**tubal ligation**	**Vasectomy**
	Surgically excluding of fallopian tubes.	Ligation Of vase deferens
	Laparoscopic or hysteroscopy	Done in the office under local incision in the upper outer aspect of each scrotum
	low risk of pregnancy but if it happens, it will be ectopic.	Unlike tubal ligation it is not immediately effective (needs 6-8 wks) so patient should use another form of contraception and till azospermia is confirmed by semen analysis (3 times)
		Safe, simple, cheap more affective

Abdominal Pain in Pregnancy

Obstetric Causes:	**Gynecological Causes:**	**Non-Obstetric/Non-Gynecological Causes:**
• Abortions (Most Common Cause) • Ectopic Pregnancy (With or without rupture) • Normal Pregnancy • Abruptio Placenta • Fulminating Pre-eclampsia • HELLP • Urinary Retention • Round Ligament • Chorioamnionitis (Intra-amniotic Infection)	• Ovarian Cyst Rupture • Adnexal Torsion • Red Degeneration of Fibroid	• Pyelonephritis • GERD • Peptic Ulcer • Acute Pancreatitis • Acute Fatty Liver • Acute Cholecystitis or Cholelithiasis • Bowel Obstruction • Acute Appendicitis • Trauma

- Ask SOCRATES.

  Labor-like pain?

Management of Labor and Puerperium in Anemic Women

- Labor and puerperium are periods of the greatest risk.
- More than 50% of deaths occur in the first 12 hours postpartum.

Management

- O_2, 2 units of blood with cross match.
- Antiseptic procedure during delivery to prevent infection.
- Shorten the second stage of labor by forceps or vacuum to relief the strain on the heart.
- Decrease blood loss to the minimum and replace losses.
- Replace blood loss by packed RBCs and DO NOT overload patient.
- IV methergine if bleeding starts.

During Labor:

- Give prophylactic antibiotics.
- Continue Fe supplements for 6 weeks postpartum.
- Advice early booking in the next pregnancy.

GYNECOLOGY

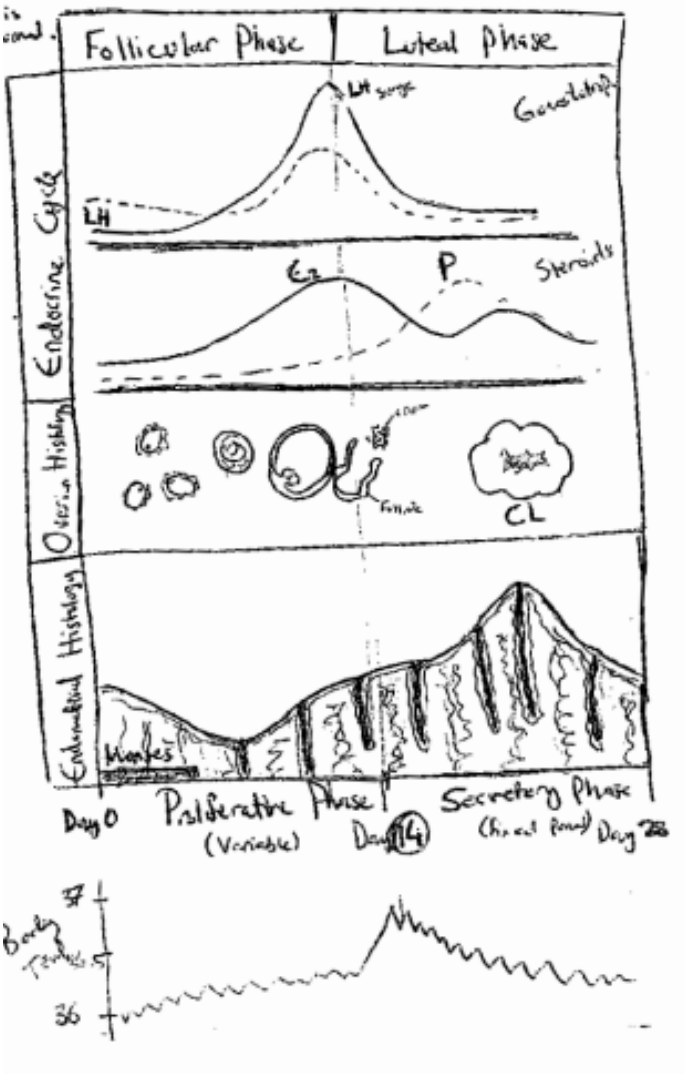
Menstrual cycle

Menstrual cycle it's the cyclical changes that occur in the female reproductive system.

Normal menstrual cycle is a 28 + 7/ - 7 Days (21-35 days).
Average Menses= 4 days, more than 7 days is abnormal.
Average amount is 30-50 ml (without clots), more than 80ml is abnormal.

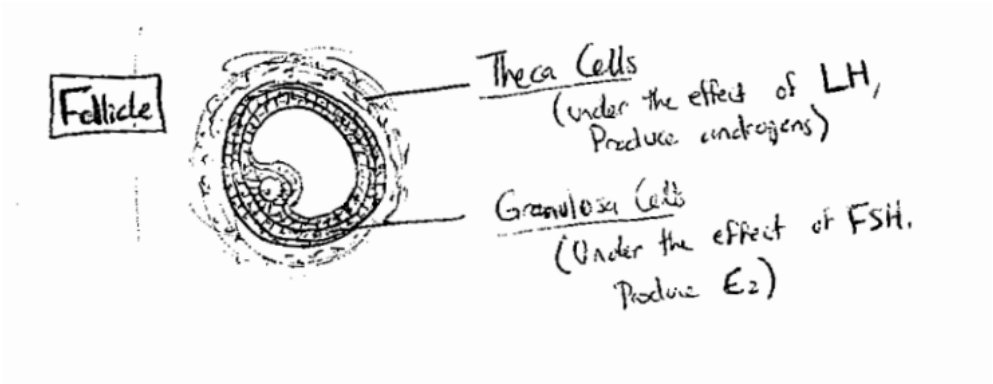
Menstriuation: withdrawal of progesterone causes endometrial
Follicular phase: FSH causes E2 secretion.
Ovulation: LH surge cause oocyte to be released

Many follicles are stimulate by FSH but the follicle that secretes more estrogen than androgen will be released (the dominant follicle).
The dominant follicle releases the most estradiol so that it is the feedback causes LH surge.



The layer of endometrium:

- Basalis Zone: (always present) it form the functional layer.
- Functional zone: sloughed of in each month.



Follicular phase: day 1-14, begin on the first day of menses old hormone levels are decrease and without any negative feedback, GnRH from hypothalamus causes FSH released from pituitary. FSH stimulates leads to maturation of the granulosa cell that secretes E2 → stimulates endometrial proliferation, inhibits LH, LSH (due to negative feedback).

Ovulation: day 14, a critical level of E2 to triggers an LH surge, the LH surge causes the oocyte to be released from follicles. The ruptured follicle will become corpus lutenum which secretes progesterone.

Luteal phase: day 14 to 28, progesterone (secreted from CL) causes the endometrium to mature in preparation for possible implantation, it vascularized and increased grandular secretions.
Progesterone negatively affects LH and FSH.
If fertilization doesn't occur CL will involute; progesterone and estradiol level decrease with endometrial sloughing (menses)
If the hypothalamus pituitary axis is released from inhibition and the cycle begins again.

Summery:

	Ovarian phase	Dominant hormone	Uterine phase
Before ovulation	Follicular	Estrogen	Proliferative
After ovulation	Luteal	progestrone	Secretory

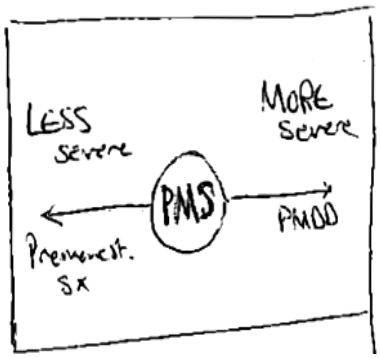
Premenstrual syndrome

Definition: premenstrual syndrome include a wide range of physical and emotional difficulties (if it is more severe, it's called premenstrual dysphoric disorder; PDD).

Diagnosis: The basis for diagnosis is to have symptoms throughout 3 menstrual cycles.

Criteria: (all must be present):

- Recurrence in more than or three consecutive cycles.
- Absent preovulatory.
- Present only postoperatively.
- Interfere with normal function.
- Resolve with onset of menses.



Timing is more important than specific symptoms.

Rule out other differentials; depression and anxiety may present all throughout the cycle.

Symptoms:

- Fluid retention; breast tenderness, extremely edema, weight gain and bloating.
- Emotional nervous; nervous tension, mood swings, depression, irritability, anxiety and crying.
- Autonomic; heart pounding, confusion, dizziness, Insomnia and fatigue.
- Musculoskeletal; muscle ache, joint ache, headache and cramps.

Treatment:

- Nutritional: balance diet, decrease caffeine, decrease sugar and degrees salt.
- Lifestyle: relaxation technique and regular exercise.
- Medication: SSRI (treatment of choice; 1st line) ex: fluxetine, Prozac and OCPs. Others: progesterone suppositories, spironelactone for edema and pyridoxine (B6).

Dysmenorrhea (Pain with menses gone after menses):

1. **Primary:** recurrent, crampy lower abdominal pain along with nausea vomiting and diarrhea that occurs during menses in the absence of pelvic pathology.
   Onset: within two years of menses at
   Etiology: idiopathic (may be myometrial sensitivity to P6).

2. **Secondary:** recurrent dull aching lower abdominal pain usually without nausea and vomiting and diarrhea occur during menses.
   Onset: after the age of 20.
   Etiology: endometriosis; others: adenomyosis, chronic PID, fibroid, surgeries, trauma and IUCD.

Primary dysmenorrheal: the most common gynecological complaint among adolescent female.
Onset of symptoms: begins several hours prior the onset of menses and continue 1-3 days. Does not occur until ovulatory cycle are established.

Pathophysiology: increase production of PGF2 (drop of progesterone)→ dysrhythmic UC, hypercontactility and increased uterine muscle tone → uterine ischemia.
Increase PG leads to nausea, vomiting and diarrhea (stimulates S, M)

Risk factors: early menarche, decease parity, diet, exercise, heavy smooking and psycho.

Management: NSAIDs (1st choice), OCPs (2ed choice).

Secondary dysmenorrheal: it is due to pathology.
Treatment according to the cause.

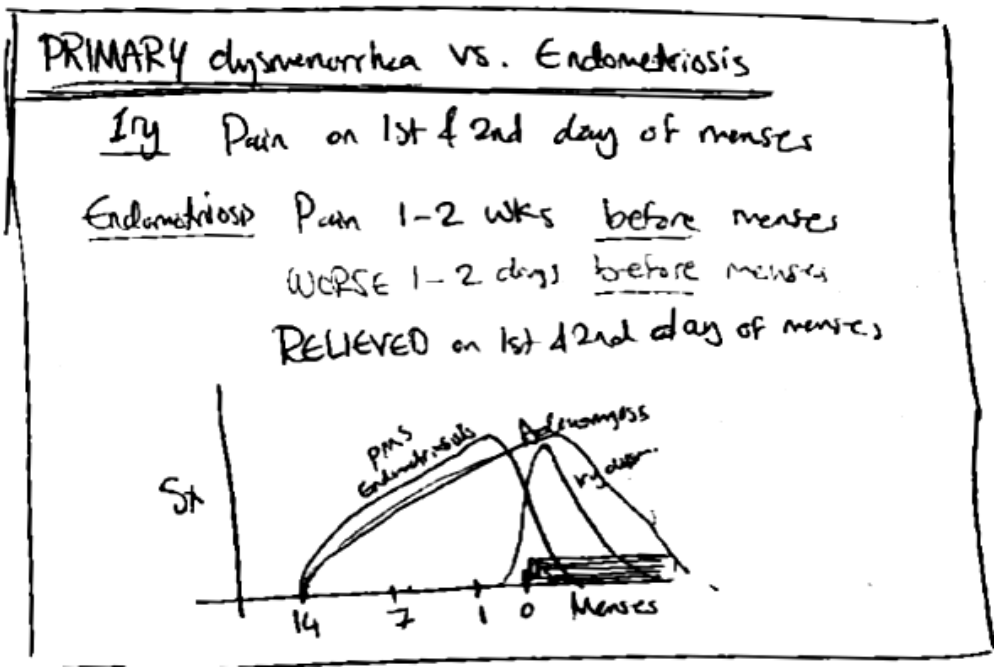
History:

- Age, Parity

- Pain (onset? first and second day of menses (increase/ decrease)? Cyclic?), SOCRATES.
  Severity; interfere with daily activities? need to take OCPs/NSAIDs? (responsive?)
  Associated symptoms: nausea, vomiting, diarrhea, dysparonia, dyschezia and abdominal pain (if not mentioned)

- Gyne history: menarche, menses, dyspareunia, bleeding, discharge, infertility and contraception.

- PMHx
- PSHx
- FHx
- DHX
- Social Hx



Adenomyosis: Pain 1 week before menses continues until cessation of menses.

PMS: pain 1-2 weeks before menses relieve on 1st and 2ed day of menses.

Endometriosis

Definition:

Ectopic endometrial glands and stroma, growing outside the uterus often causing pain and/or infertility.

*Not a premalignant condition.

*It's and major cause of secondary dysmenorrhea.

*Adenomyosis is also an ectopic endometrial glands and stroma but found within the myometrium, it was called endometriosis interna.

Incidence:

*It's common a disease, peak incidence is at the ages between 20-30 years, affects 10-15% of reproductive aged women, and common in nulliparous.

*causes 20% of chronic pelvic pain.

Pathophysiology:

The most accepted theory is retrograde menses.

Sites:

1. Ovaries are the most common site, also known as endometriomas or chocolate cysts
2. Cul de sac (the 2nd most common site)
3. Uterosacral ligament (nodularity on U/S)
4. Rectosigmoid

Risk factors:

1. Family history
2. Race
3. Autoimmune diseases

Symptoms:

- Gyne: **dysmenorrhea**, **dyspareunia**, abnormal bleeding, infertility.
- GI: **dyschezia** (pain on defecation), cyclic rectal pain, cyclic diarrhea and constipation.
- GUT: cyclic hematuria and dysuria.
- RS: cyclic hemoptysis.

* Classical symptoms are dysmenorrhea, dyspareunia and dyscehzia.

*1/3 are asymptomatic.

*symptoms usually improve with pregnancy due to cessation of menses.

*severity of symptoms does not necessarily correlate with amount of ectopic endometrial tissue, but depends on the site and depth of penetration.

Pelvic exam:

Tenderness, fixed retroverted uterus (due to cul de sac adhesions), enlarged adnexa (if ovaries are involved.)

*you should do rectovaginal exam to feel the uterosacral nodularity.

Investigations:

- Normal WBC and ESR, Increased CA 125.
- You may see endometrioma on U/S.
- Laparoscopy: “powder burn appearance”.
- Biopsy: endometrial glands and stroma and hemosiderin laden macrophages (both are cardinal features).

*the colors of endometrium implants varies from red (new) → brown (older) → white (oldest).

*the diagnosis is mainly clinical but definite diagnosis is by laparoscopy and confirmed by histopathology.

Treatment:

Medical: our goal is to induce amenorrhea, causing regression of endometrial implants, so we give drugs that decrease estrogen.

- GnRH agonists (leuprolide): decreases FSH, can't be given for more than 6months (pseudomenopause state).
- Danazol (androgen): decreases FSH and LH (pseudomenopause state).
- Depoprovera or combined OCPS (they put the patients in pseudopregnancy state).

*Endogenous GnRH is secreted in pulsatile fashion and thus increases FSH secretions, meanwhile GnRH agonists down regulate pituitary receptors decreasing FSH secretion.

Surgical:

- Conservative by adhesion lysis and ablation of adhesions and implants to preserve fertility.
- Radical by TAH – BSO procedure, but should provide estrogen replacement therapy.

Note: adenomyosis occurs in older, multiparous women, not responsive to hormones and causes noncyclical pain whereas endometriosis occurs in young nulliparous women, responsive to hormones and causes cyclical pain.

Puberty and Precocious puberty

Puberty:

Is the transition from childhood to the final stage of maturation that allows for reproduction, begins with disinhibition of the pulsatile GnRH secretion (unknown mechanism).

2ry sexual characteristics:

In order:

1. Thelarche: breast development at the age of 10 and the responsible hormone is estrogen (E2).
2. Pubarche (Adrenarche): appearance of pubic hair, at the age of 11, adrenal hormones are the responsible hormones.
3. Menarche (1st mark): after Pubarche growth spurt occurs, at the age of 12 and the responsible hormone is estrogen.

Tanner stages refer to the sequence of events of breast and pubic hair development

Stage 1	Prepubertal child
Stage 2-4	Developmental stages
Stage 5	adult

Precocious Puberty:

More common in females, defined as the appearance of 2ry sexual characteristics before the age of 8 years old.

*our main concern is the stature.

Classification:

1. Incomplete precocious puberty results from transient increase in hormones or unusual end organ sensitivity.
   *Thelarche in incomplete is usually isolated and transient, might be the 1st sign of true precocious puberty.
2. Complete precocious puberty (m.c.c is idiopathic) further classified into:
   - GnRH dependent, characterized by elevated FSH and estrogen (from a premature GnRH release), includes the following:

    - Idiopathic (80%): constitutional, age between 6-7 years, MRI is normal, treated by GnRH agonists and follow up.
    - CNS pathology (rare): CNS lesion (tumor, sarcoid, infection), age is below 6 years, MRI is abnormal and the treatment is variable (surgical or medical).
- GnRH independent, characterized by decreased FSH and increased estrogen (from autonomous ovarian production), includes the following:
    - McCune Albright syndrome (5%): AKA polyostotic fibrous dysplasia, characterized by Café au lait spots and bone deformities, treated by aromatase inhibitor.
    - Granulosa cell tumor, findings include pelvic mass and its treated by surgical removal.

Type	Incomplete	Complete
Changes	In Thelarche or Adrenarache or Menarche (very rare)	Thelarche and Adrenarche and Menarche
Management	Conservative	Variable

Work up:

- Bone age (lt. wrist x-ray).
- LH.
- GnRH stimulation test.
- Brain MRI, Pelvic MRI

Hirsutism

Defined as the presence of terminal (coarse) hair in females in a male like pattern with prevalence of 5-15% of females, where vellus hair is transformed into terminal hair under the influence of androgens.

*Virilism is hirsustism plus masculinizing signs in females (seen in tumors).

Causes:

1. Androgenic**:** PCOS, androgen secreting tumor.
2. Non androgenic: hypothyroidism, cushing, CAH, increase in prolactin.
3. Exogenous: drugs.
4. Idiopathic.

Approach:

History of presenting illness:

Ask about site and progression then according to differential diagnosis.

- Androgenic: acne, voice changes and balding.
- Hypothyroid: fatigue, cold intolerance, weight gain.
- Cushing: striae, HTN, DM, acne.
- Tumor: weight loss, anorexia, pain and heaviness.

Gyne history:

Ask about Menarche, regularity, history of oligorrhea or amenorrhea, puberty problems, infertility, OCP.

PMH:

PCOS, thyroid, hormone problems, HTN, DM, breast CA, Tumors.

Drug history:

Steroids, anabolics, androgens, danazole, phenytoin, cyclosporines, testosterone.

Physical exam

- General: BMI, 2ry sexual characteristics, distribution of hair.
- Vital signs.
- We look for cushing characteristics and we do abdominal and genital exam.

Investigations:

We order prolactin, TSH and free testosterone, when both prolactin and TSH are normal we proceed the following way:

1) Normal testosterone → 5 alpha reductase overactivity or idiopathic.

2) Elevated testosterone we look for DHEA:

- Normal DHEA → ovarian cause (PCOS or ovarian cancer) further investigations include LH/FSH, U/S and CT Pelvis.
- Elevated DHEA → CAH (increased 17-hydroxy progesterone) or adrenal CA (CT abdomen).

*DHEA is made by adrenals.

*Testosterone is a reflection of ovarian hyperplasia.

Management:

- Lifestyle modification (decrease weight).
- Idiopathic: cosmetic (laser/ waxing/ electrolysis).
- 5 alpha reductase: Fenasteride (5-alpha reductase inhibitor).
- Ovarian (PCOS): metformin, OCP and GnRH agonist.
- Adrenal: Prednisolone and spironolactone.
- Tumor: surgical correction.

Amenorrhea

Two types

1)Primary: (no period at all)

-Absence of menses at age 14 without secondary sexual characteristics

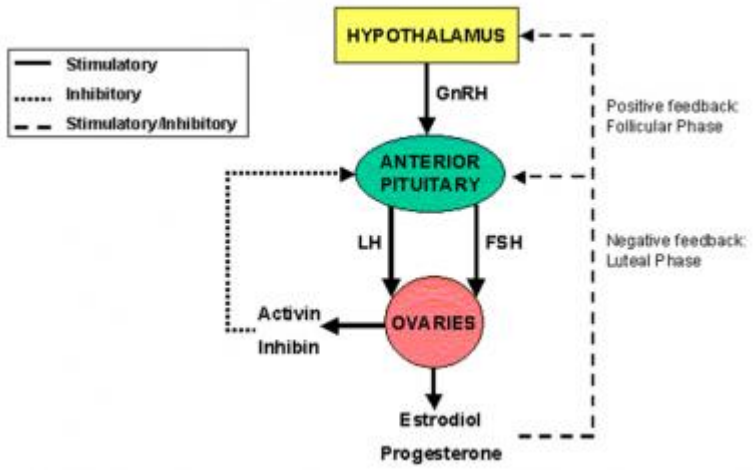
-Absence of menses at age 16 with secondary sexual characteristics

2)Secondary: (in a female who is menstruating)

-Absence of menses for 3 consecutive cycles (if regular) or 6 months (if irregular).

Classification of amenorrhea:

- Disorders of outflow (Axis I)
- Disorders of ovary (Axis II)
- Disorders of Anterior pituitary (Axis III)
- Disorders of CNS (Axis IV)



Primary Amenorrhea

Causes:

1- Anatomic:
   - Imperforate Hymen
   - Vaginal Agenesis/Septum
   - Müllerian agenesis

2- Hormonal:
   - Gonadal Dysgenesis (Turner Syndrome)
   - Androgen Insensitivity
   - Hypothalamic-Pituitary Insufficiency

The most common cause of primary amenorrhea is chromosomal abnormality (45%)

- Gonadal dysgenesis (Most common cause)
- Mayer-Rokitansky-Küster-Hauser (MRKH) syndrome (second most common cause)

20% of primary amenorrhea is physiologic delay.

Note:
Breasts are endogenous assay of estrogen, so the presence of breasts indicates adequate-estrogen production and vice versa.

Approach:

Müllerian Agenesis vs Androgen Insensitivity

Breast PRESENT Uterus ABSENT	Müllerian Agenesis (46, XX) (ovaries present)	Androgen Insensitivity (46, XY) (testes present, NO ovaries)
Uterus ABSENT Why?	Idiopathic	Müllerian Inhibitory factor (MIF)
Estrogen from?	Ovaries	Testes (androgens are converted to estrogen)
Pubic hair?	Present	Absent
Testosterone level?	Normal female level	Male level
Treatment	• No hormones • Create vagina • In vitro fertilization(IVF)-surrogate	• Estrogen • Create vagina • Remove testes

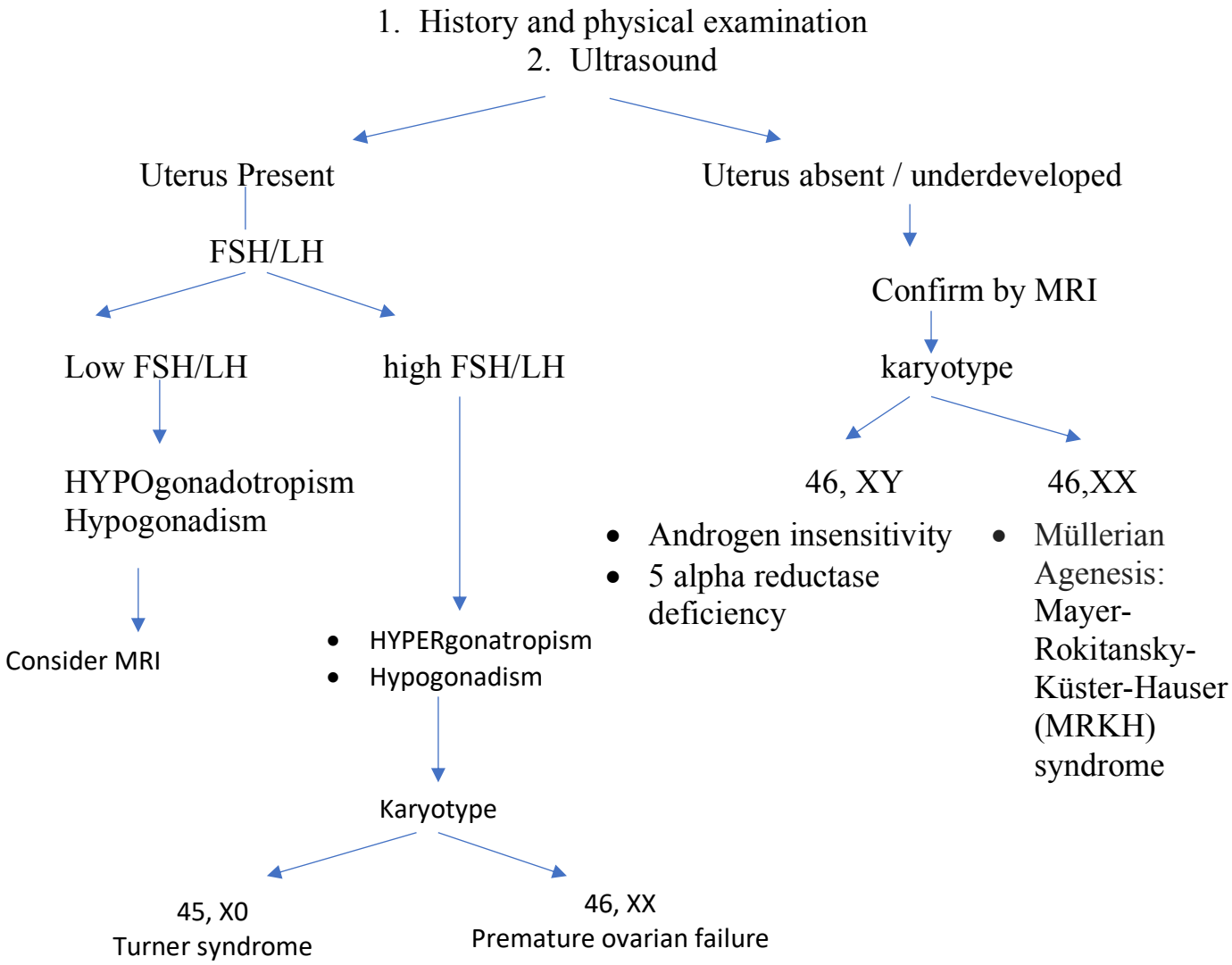
Breast ABSENT Uterus PRESENT	Gonadal Dysgenesis (Turner Syndrome) (45, X0)	Hypothalamus-Pituitary Axis Failure (46, XX)
FSH	Sky high	Low
Why no estrogen?	NO ovarian follicles (streak ovaries)	Follicles NOT stimulated
Ovaries?	"Streak"	Normal
Treatment for Pregnancy	• Estrogen and Progestin • Egg donor	• Estrogen and Progestin • Induce ovulation (human menopausal gonadotropin (hMG))
Diagnostic Test	———————	CNS imaging

If Breast PRESENT and uterus PRSENT, think of imperforate hymen/ vaginal septum/ Anorexia Nervosa/ excessive exercise.

Approach

BY history and Physical examination:

BREAST (secondary sexual characteristic)	UTERUS (+)	 (-)
(+)	-Rare- • imperforate hymen • vaginal septum • Anorexia Nervosa/ • excessive exercise	-Common- Differential diagnosis • Mullerian Agenesis Vs • Androgen insensitivity
(-)	-Common- Differential diagnosis • Gonadal Agenesis Vs • Hypothalamic-pituitary axis failure	-Rare- not clinically relevant



Secondary Amenorrhea

Definition:
if Regular menses: 3 months
if Irregular menses: 6 months

Causes:

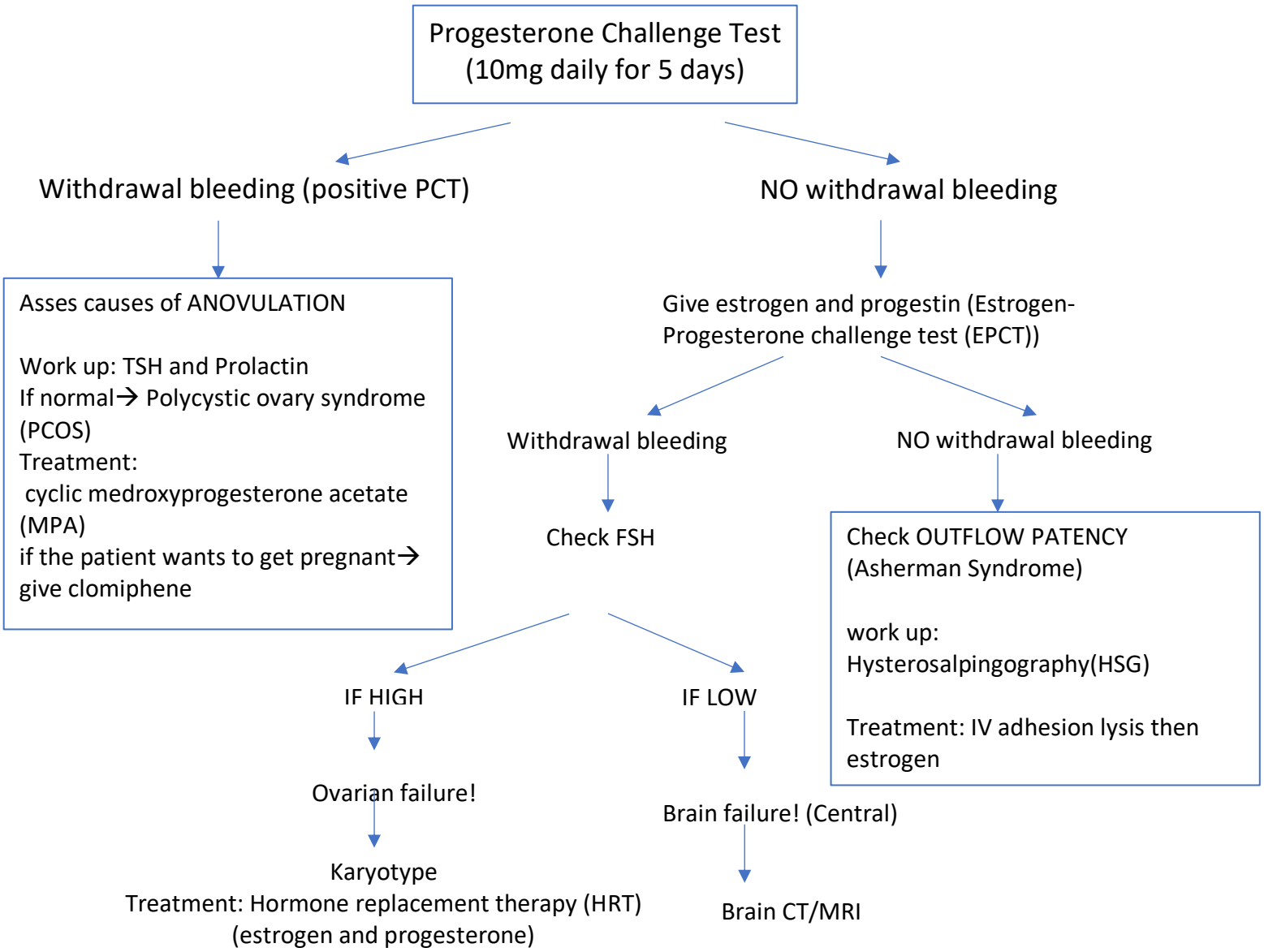
- PREGNANCY (most common cause) → rule out by Beta human chorionic gonadotropin (B-HCG) hormone
- ANOVULATION (progesterone is missing! No corpus luteum)
- Estrogen deficiency
- Outflow obstruction → Asherman Syndrome or Cervical Stenosis

Pathophysiology:

- HYPOgonadotropic hypogonadism→ Hypothalamic pituitary dysfunction
- HYPERgonadotropic hypogonadism→ Ovarian follicular failure
- EUgonadotropic eugonadism→ Pregnancy/ Anovulation/ Uterus/ Outflow tract

Approach:

- History and physical examination
- Progesterone Challenge Test (PCT)



NOTE:

- Positive Progesterone Challenge Test (PCT) is ALWAYS due to ANOVULATION
- Positive Estrogen- Progesterone Challenge test is ALWAYS due to LOW estrogen

Differential diagnosis for both primary and secondary amenorrhea:

CNS (hypothalamus):

- Excessive Exercise
- Anorexia Nervosa
- Stress
- Systemic illness
- Kallmann syndrome

Pituitary:

- Adenoma (eg. Prolactinoma)
- Necrosis (Sheehan syndrome)
- Hyperprolactinemia (phenothiazines)

Reproductive tract:

- Asherman syndrome (IV adhesions)
- Mullerian Agenesis
- Transverse vaginal septum
- Imperforate hymen
- Testicular feminization syndrome (androgen insensitivity)

Ovary:

- Anovulation
- Premature ovarian failure (eg. Autoimmune diseases, chemotherapy, radiotherapy)
- Gonadal dysgenesis
- Resistant ovary syndrome.

OSCE:
History:
Patient profile: Age, marital status
Chief Complaint: Duration of amenorrhea, last menstrual period, lactating?

- Menstrual history:

- Age of menarche
- Menses: regular/irregular, cycle duration, bleeding days, heavy/light
- Any pain/problems? + how long did it last for?
- Any menopausal symptoms?

- Obstetric history

- Have you conceived in the past? Term? Normal deliveries?
- Did you have any problems after delivery? Any heavy bleeding? (Sheehan syndrome)
- Have you ever had a pregnancy terminated? Have you ever had a dilation and curettage (D&C)? (Asherman syndrome)

  - Past medical history:
- Thyroid problems? (hypo/hyperthyroidism)
- Weight? Facial hair/ acne/ deepening of voice? (PCOS)
- Have you noticed discharge from your nipples? Problems with vision/ headache? (pituitary function, prolactinoma)
- Are you eating/ drinking well? Have you lost weight recently? Do you exercise a lot? (Anorexia / excessive exercise)
- Have you noticed that your stomach is increasing in size / feeling bloated/ swelling in your tummy? (ovarian tumor)
- Do you feel tired/ unwell? How are you generally? (systemic illness).

  - Social history:
- Stress
- Sexual abuse

  - Drug history:
- Any drug? (drug side effects)
- Pills? (post pill amenorrhea)

  - Family history:
- When did your mother & sister reach menopause? (premature or failure)

Physical Examination:
General: BMI, secondary sexual characteristics, hirsutism, signs of thyroid/ adrenal disease, associated to the suspected cause

Investigations:
BHCG (to rule out pregnancy)
Thyroid function test, Prolactin
FSH, Testosterone, +/- (PCT/EPCT)
Ultrasound, hysterosalpingography (HSG)

Determination of Sex and Intersex

Intersex: Abnormal condition of being intermediate between female and male. (when there is 1 or 2 differences from the normal characteristics of the sex).

Normally:
Chromosomes:
XY→ influence the growth of testes
XX→ influence the growth of ovaries

Concept:
So chromosomes (XY, XX) will determine the gonads.
AND gonads will determine the features.

If testes are present and function→ it will produce testosterone (virilization) and Mullerian inhibitory factor (MIF) (prevent uterus formation).
So if gonads become nonfunctional (regardless the chromosomes) there will be intersex.

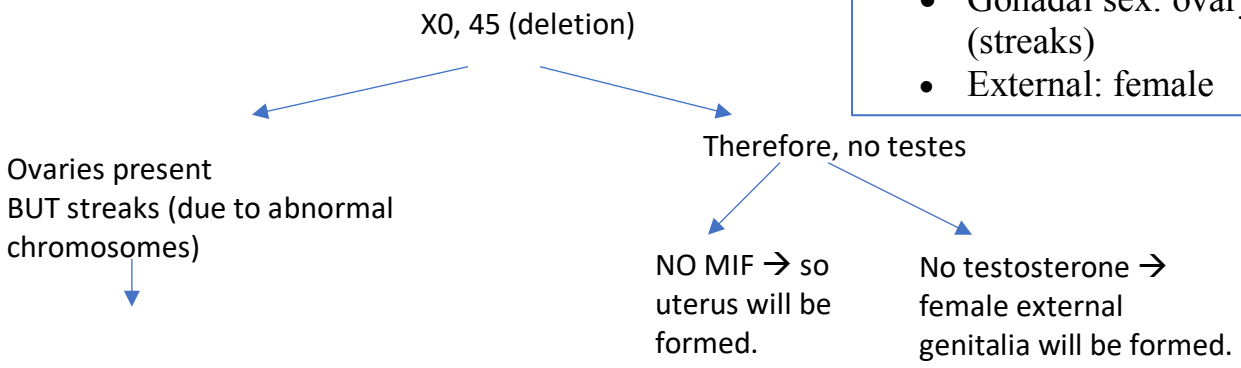
Types of intersex:

1. At the chromosomal level (addition, eg Triple X and klinefelter) and (deletion, eg turner)
2. At the gonadal level (eg. True hermaphroditism)
3. End organ resistance (eg. Androgen insensitivity, 5 alpha reducatse deficiency, and congenital adrenal hyperplasia(CAH))

At the CHROMOSOMAL level:

Turner:
- Chromosome: X0
- Gonadal sex: ovary (streaks)
- External: female

TURNER SYNDROME (female gonadal dysgenesis):



- So primary amenorrhea will develop.
- No secondary sexual characteristics.

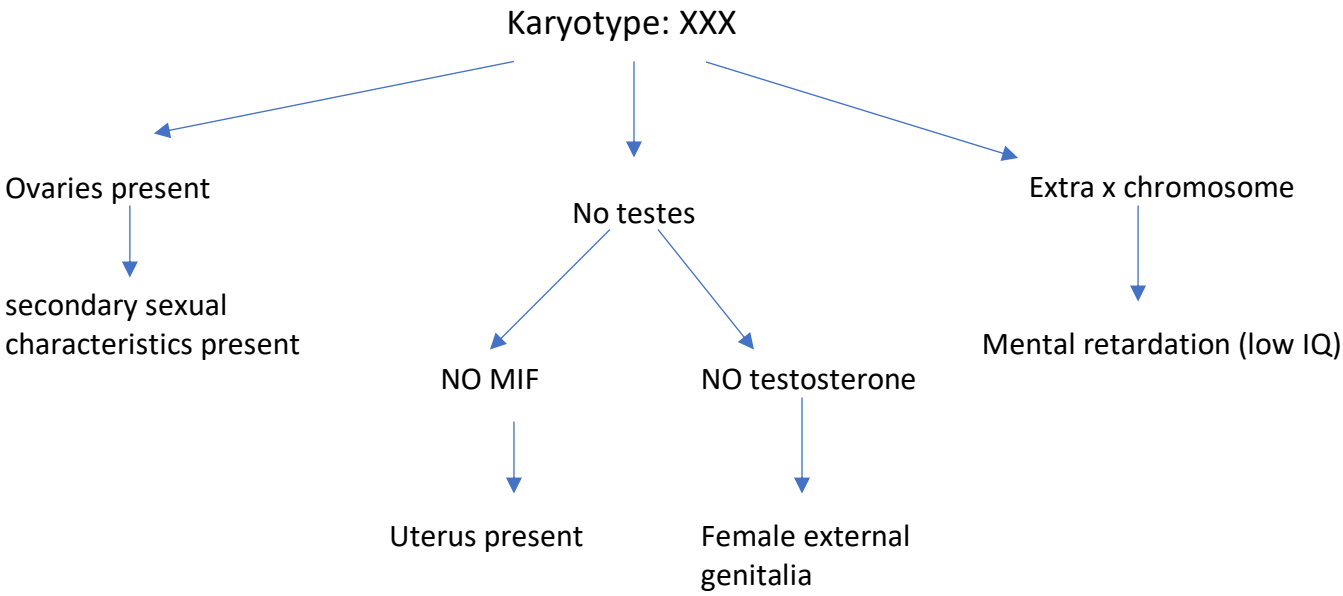
Features:

- Widely spaced nipples
- Webbing of the neck
- Short stature
- Cubitus valgus
- Congenital cardiac defects
- Infertility

Atypical turner (mosaicism):
(X0/ XX)

- Might be normal in appearance
- Might be fertile
- More common than pure turner

Tripple X- Female: (RARE)



Remember!
Whenever there is an extra X- chromosome, there will be mental retardation.

Features:

- Patients may have amenorrhea
- Mental retardation is common due to extra X chromosome.
- Buccal smear shows: 2 barr bodies

Triple X female:

- Chromosome: XXX
- Gonadal sex: ovary
- External: female

Klinefelter's syndrome

- Karyotype XXY, 47
- There's an extra X chromosome: mental retardation and low IQ.
- Testes are present, but they are atrophied, MIF present (Mullerian Inhibiting Factor), no uterus.
- Testosterone present.
- External genetalia: male.

Features:

- Infertility: atrophy of seminefrous tubules leads to azoospermia.
- Gynecomastia.
- Mental retardation: extra X chromosome.
- Labs: FSH high.

At the gonadal level:

True Hermaphroditism

- Karyotype is variable: XX, 46.

  XY, 46 / XXY, 47 (mosaics)
  XY, 46 (rare)

- Essential diagnostic criteria: presence of both testicular tissue and ovarian tissue.
- External genital sex and internal genital sex vary widely.

End organ resistance:

Androgen insensitivity, 5a-hydroxylase deficiency, CAD

Androgen insensitivity

- Aka testicular feminization.
- Karyotype: XY, 46
- Testes are present, MIF present, no uterus.
- Testosterone present but has no effect because there are no receptors.
- Gonadal sex: testes.
- External genetalia: female.

- Estrogen is very high (androgens will be transformed to estrogens): no pubic hair, female secondary characteristics, breasts are well-developed.
- Presentation: most cases present at puberty (due to primary amenorrhea).

Etiology:

The problem is deficiency of androgens in the target organs due to absence of the gene for the androgen receptor.

Features:

- Female of a normal height.
- Well-developed breasts (due to peripheral conversion of androgens)
- Vagina short and blind.
- Scanty or absent axillary hair.
- Labs show normal male level of testosterone.
- Buccal smear is negative.

Management:

-Gonadectomy after puberty, HRT

-Gonadectomy is performed due to risk of CA gonadoblastoma.

5a-Reductase deficiency

- Familial disorder, AR.

- 5a-reductase is a hormone responsible for conversion of testosterone to the more potent hormone DHT (DiHydroTestosterone). When deficient, poor masculinization of external genetalia will occur.

Testosterone ----(5a-reductase)→ Dihydrotestosterone DHT

-Karyotype: XY, 46.

-Testes present, MIF present, no uterus.

-Low levels testosterone (not potent as DHT).

-External genetalia: female.

-Gonadal sex: testes.

Features:

-A male infant who has poor masculinization of external genetalia (it offers as an ambiguous genetalia with a small penis that is capable to ejaculate).

-At puberty: testosterone production increases leading to virilization.

Congenital Adrenal Hyperplasia CAD

-Intersexuality.
-Ch: XX
-Gonadal sex: varies.
-External genetalia: varies.
-Deficiency in the enzymes essential for the conversion of progesterone to cortisol, leading to high levels of progesterone and low levels of cortisol.
-Most common: 21-hydroxylase enzyme deficiency.
-Treatment: cortisone therapy.

Features:
-Clitoral hypertrophy.
-Labiosacral fusion.
All are effects of increased androgens.
-Important! Internal organs, gonadal and chromosomal sex are NEVER affected.

Late-onset CAD:
-AR trait
-Most common form due to 21-hydroxylase deficiency that increases 17-hydroxyprogesterone levels in the blood.

Non-progressive female intersex:
-Due to female fetus exposure to abnormal androgen stimulus in utero.
-At birth: external genetalia are similar to cases of adrenal hyperplasia but not progressive.

Investigations:
-Buccal smear -Chromosome studies
-Hormonal studies -Ultrasound
-Imaging: MRI/CT -Diagnostic laparoscopy
-Exploratory laparoscopy

Polycystic Ovarian Syndrome PCOS

Aka. Stein-Leventhal Syndrome

-Definition: a condition of chronic anovulation resulting subfertility, irregular bleeding, obesity and hirsutism.

-It's a common condition affecting 5% of females in reproductive age.

Classification

WHO classification for patients suffering from anovulation, 3 types:

-WHO 1 (15%): HYPOgonadotropic, HYPOestrogenic.

-WHO 2 (80%): NORMOgonadotropic, NORMOestrogenic.

-WHO3 (5%): HYPERgonadotropic, HYPOestrogenic.

The PCOS patients form a large subgroup of WHO2.

Pathophysiology

Not entirely clear what precipitates it, but once it begins, a vicious cycle occurs:

- **Chronic anovulation:**

Steady state FSH and steady state E2 (estradiol) → no LH surge:

- no corpus luteum→ no progesterone→ unopposed E2→ irregular periods
- chronic anovulation→ no eggs →infertility.

Treatment: Clomiphene citrate (for infertility), metformin, progestins/ OCPs.

- **Insulin resistance and hyoerandrogenism:**

LH:FSH ration is high 3:1 (normal is 1.5:1), this is due to high unopposed estrogen. →LH (and insulin) + theca cells → high androgens

→decreased liver production of SHBG (Sex Hormone Binding Globulin):

→ increased free estrogen→ increased LH:FSH ratio

→increased free testosterone→ hirsutism

Treatment: OCPs

- **Ovarian enlargement:**

Many follicles in various stages of growth, due to steady state FSH and high androgens.

Diagnosis:
Criteria for diagnosis: presence of at least 2 of the following criteria, after ruling out CAD/ androgen-secreting tumors/ Cushing.

1-Oligo/Anovulation
2-Evidence of hyperandrogenism (clinical/lab)
3-Polycystic ovaries on US (>10-12 small follicles in the ovaries)

US criteria for diagnosis: presence of at least 10-12 follicles in one ovary measuring <10mm in diameter &/or increased ovarian volume >10ml.
Characteristic appearance on US: string of pearls.
Although US is a major diagnostic tool, it's not the only one, because not all females with PCOS have polycystic ovaries on US and not all females with ovarian cysts have PCOS.

HAIR-AN syndrome
-A subtype of PCOS that consists of HA-IR-AN:

HA for HyperAndrogen,
IR for Insulin Resistance,
AN for Acanthosis Nigricans.

Signs and symptoms
-Menstrual irregularities
-Hyperandrogenism symptoms: hirsutism, acne, obesity, alopecia.
-Anovulatory infertility/ subfertility
-Obesity
-DM and acanthosis nigricans
-Metabolic syndrome:
abdominal obesity (waist circumference >35cm), dyslipidemia (TG>150, HDL<50), high BP, proinflammatory state (high CRP), prothrombotic state (high PAI-1, high fibrinogen).

Diffirential diagnosis
- Ovarian hyperthecosis
- Idiopathic/ familial hirsutism
- Cushing/ exogenous anabolic steroid
- Stromal hyperthecosis (Valporic acid)
- Drugs (ex. Danazol, Androgenic progestins)
- Ovarian/ adrenal tumors (rapid onset of signs of virilization)
- CAD (late-onset)
- Obesity

Management of polycystic ovarian syndrome

***Aim of treatment**:

- Decrease insulin
- Treatment of hirsutism or acne
- Restoration of regular menstruation
- Prevention of Endometrial hyperplasia or cancer
- Restoration of fertility

1- Weight loss: is the most effective method in restoring normal ovulation and menstruation.
2- Hypoglycemic agents: (metformin), it decreases insulin resistance, helps in losing weight and enhances ovulation.
3- Progestins or oral contraceptive pills: eg: cyproterone acetate. Regulate the periods, induce ovulation and some androgenic effect, minimizes endometrial hyperplasia or cancer.
   Diane: cyproterone acetate and estradiol.
4- Anti-androgens: indication for hirsutism and acne.
   Eg: cyproterone acetate (eg: androcur), spironolactone, flutamide.
5- Clomiphene citrate: improves fertility (induces ovulation).

- For patients who are not responding to clomiphene, diet, or lifestyle modification, there are other options: assisted reproductive technology (ART), IVF etc.

Infertility

- Definition: inability to achieve pregnancy after one year of unprotected sex
- Fecundability (monthly chance of pregnancy) is 20%, so within one year, 85% of pregnancy occur.
- Prevalence is 15%
- Instigations for infertility starts after one year.
- Age influence: fertility declines significantly after the age of 35, and more rapidly after 40.

***Causes:**
- Unexplained 15%
- Females: most commonly due to: anovulation or tubal cause. 50%
- Males: semen problems 35-40%

- **Female etiology:**

Ovarian:

- Polycystic ovarian syndrome (normogonad normogonadin).
- Advanced maternal age.
- Premature ovarian failure.
  *if the second and the third point, high FSH (hypogonad hypogonadin) there is no treatment.

- Hypothalamic amenorrhea – hypogon. Hypogonadin. Treatment is pulsatile GnRH therapy

- Hyperprolactinemia

Tubal:

- Pelvic inflammatory disease
- Surgical procedure or ligation
- Endometriosis
- Pelvic adhesions
  - Treatment is surgical removal of adhesions, reanastomosis tuboplasty, reversal of tubal ligation and invitro fertilization (IVF), there is no role of medical treatment.

Cervical:

- Cervical stenosis
- Chronic cervical inflammation
- Mullerian duct abnormality
- Diethylstilbestrol (DES) exposure
  - Treatment : surgical dilatation, intrauterine insimenation (IUI)

Uterine:

- Congenital malformation
- Submucosal fibroid
- Polyps
- Asherman syndrome (adhesions)
  - Treatment: Myomectomy, operative hysteroscopy.

Metabolic disorders:

- Thyroid
- Liver
- Obesity
- Androgen excess

- **Male etiology:**

- Environmental exposure: smoking, alcohol, excessive heat, radiation…etc.
  Treatment: prevention
- Sexual dysfunction: erectile, ejaculation.
  Treatment: medical
- Structural factors: varicocele, testicular torsion, vasectomy
  Treatment: surgical
- Abnormal semen: mumps, anti-sperm antibody
  Treatment: washed sperms for intrauterine inoculation (IUI), intracytoplasmic sperm injection (ICSI)
- Genetic factors: cystic fibrosis, kleinfelter, immobile cilia.

- **Main causes of infertility:**

- Male factor (semen)
- Anovulation
- Tubal

- Unexplained

Evaluation:

- History and physical exam
- Investigations (start from the least expensive):
  - Abnormal semen
  - Anovulation
- These two points are the least expensive, initial non-expensive test and treatment.
  - Tubal disease: more expensive, follow up , invasive, laparoscopy , hysterosalpingogram (GSH)
  - Unexplained or IVF (in vitro fertilization): most expensive testing and treatment.

Semen analysis:

- Normal values :
  - Volume > 2 ml (2-8 ml)
  - Density >20million/ml
  - Ph (7.2-7.8)
  - Motility>50%
  - Morphology>50%
- If abnormal, the first next step is to repeat the test again within 4-6 weeks.
- If minimally abnormal, then intrauterine inoculation (IUI).
- If severely abnormal, then intracytoplasmic sperm injection (ICSI).

Anovulation:

- History: irregular menses
- Investigations: TSH, T4, PRL, DHCA, midluteal progesterone, PCT, dexa challenge test, clomiphene citrate challenge test, MRI, and u/s.
- Correctable causes: low T4, high prolactin
- If uncorrectable, i.e: polycystic ovarian syndrome (PCOS): ovulation induction by clomiphene citrate or HMG. Side effects: ovarian hyperstimulation.
- Objective data: basal body temperature: flat (no LH surge), low progesterone, biopsy: proliferative endometriosis.

Tubal disease:

- HSG (hysterosalpingogram), if normal: no further testing, abnormal: consider laparoscopy.
- Laparoscopy: indicated if potentially correctible tubal disease is suggested by HSG.
  Tuboplasty: reconstruct damaged oviducts (if possible)
  Salpingectomy and IVF: if severely damaged
- Chlamydia antibody: infection induced tubal adhesions.

Unexplained infertility:

- No pregnancy with normal semen analysis, confirmed ovulation, and patent oviducts.
- Outcome: if no further intervention: 60% spontaneous pregnancy in three years, 2% monthly pregnancy rate
- First try ovulation induction and IUI anyway, and then IVF.

Indications for IVF (in vitro fertilization):

- Oligozoospermia
- Irrepairable tubes
- Unexplained fertility
  - HMG is used prior to procedures, to stimulate the ovaries.

Infertility history:

- Private couple counselling
- Age
- Previous pregnancy of each partner
- Length of time without pregnancy and last date of intercourse
  - **Male history:**
- History of infection (mumps) or excessive heat
- History of testicular cancer
- Drugs or radiation exposure
- Smoking, alcohol, diet
  - **Female history:**
- Polycystic ovarian syndrome (anovulation)
- Pelvic inflammatory disease (tubal disease)

- Endometriosis
- Fibroid
- Pelvic disease, intra-uterine device insertion
- Family history

Induction of ovulation:

- Clomiphene citrate: mechanism of action: competitive receptor for estrogen in hypothalamus
- Letrozole: mechanism of action: aromatase inhibitor (decreases conversion of androgen to estrogen).
- HMG- human menopausal gonadotropin: mechanism of action: elevates FSH, LH concentration directly. Used in severe cases.

- Fertilization: IUI, IVF, ICSI
- Reimplantation: if IVF or ICSI used
- Side effects: ovarian hyperstimulation syndrome (more with HMG than clomiphene).

O**varian hyperstimulation syndrome (OHS):**

- Potentially life-threatening condition
- Occurs after induction of ovulation
- Features: multiple cysts formation, ovarian enlargement, increased papillary permeability which leads to third spacing.
- This will lead to: hypovolemia, electrolyte imbalance, hypoalbumenemia, pleural effusion and acute respiratory distress syndrome, renal failure, hemoconcentration and thromboembolism.
- High risk patients: previous history of ovarian hyperstimulation syndrome (OHS), PCOS (polycystic ovarian syndrome), young petite patients.
- Resolves usually in :
  - One week if the patient doesn't get pregnant
  - One to two weeks if the patient became pregnant
- Management :
  - Correct volume and albumin
  - Anticoagulation
  - Fluid aspiration

Fibroids (leiomyoma)

Notes:

- It is a local proliferation of smooth muscle cells of the uterus.
- Benign
- Idiopathic
- Mostly asymptomatic and needs no treatment
- Almost always multiple
- Hormone responsive (increases with pregnancy and exogenous estrogen)

Types:

1. **Intramural:**
    - Within the wall of the uterus
    - Asymptomatic
    - Cannot be felt on examination unless enlarged
2. **Submucosal:**
    - Just below the endometrium
    - Most common symptom is bleeding (menorrhagia, metromenorrhagia)
    - Distorts the internal contour
3. **Subserosal:**
    - Just below the serosa / peritoneum
    - Usually asymptomatic but if very large can cause pressure symptoms
    - Distorts the external contour
    - If connected with a stalk they are called pedunculated
4. **Parasitic:**
    - Originally, they are pedunculated subserosal, the stalk becomes necrotic and breaks away from the uterus and receives its blood supply from abdominal organs (omentum/mysentry).
5. **Cervical**
6. **Interligamentous**
    - It grows laterally into the broad ligament

Risk factors:

OPRAH WINEFRY

- Black, obese, non-smoker women
- Perimenopausal, nulliparty (or old age at first pregnancy)

Presentation depends on:

- Site
- Size
- Number

- Family history

Approach:

1. **History:**
   - 50-60% asymptomatic
   - If symptomatic, the most common symptom is *bleeding*.
   - So ask about:
     (a) Gyne: Bleeding: (menorrhagia/ postcoital/ metrorrhagia/ dyspareunia).
     (b) Abdominal pain:
     (Socrates) acute infarct, dysmenorrhea.
     (c) Pressure symptoms:
     GUT: frequency/retention /hydronephrosis.
     GI: Bloating /constipation /rectal pressure.
     (d) Obstetric problems:
     Infertility, recurrent abortions, preterm labor, placenta previa, abruption, malpresentation, IUGR, APH, PPH, C/S.
     (e) Complications:
     Degeneration, torsion.
2. **P/E:**
   - General: pallor, vital signs (tachy).
   - Abdomen: increase in fundal height, mass.
   - PV/Bimanual: localized, non-tender, irregular mass or uterus with cobble stone.
3. **Investigations:**
   - CBC, Blood group, coagulation profile (remember she is anemic)
   - Saline infusion sonography, U/S, HSG (doesn't differentiate between fibroid and bicornuate uterus)
   - MRI (to differentiate it from adenomyosis)
   - D&C, biopsy, hysteroscopy(definitive).
   - The most common diagnostic method: U/S
   - The most definitive diagnosis : biopsy

DDx:

- Mass
- Abdominal bleeding

4. **Treatment :**
   - No treatment if asymptomatic(observation and follow up by serial PV)
   - Medical :
     (a) Pre surgical shrinkage: GNRH analogues decrease the size by 70%, given 3-6 months before surgery, regrowth after stopping (not used for definitive treatment just before surgery).

(b) Invasive radiation (embolization): uterine artery embolization, preserves uterus, but you don't preserve fertility.

- Surgical :

(a) Myomectomy : preserves fertility ,laparoscopy/laparotomy (1/3 of fibroids recur following myomectomy)

(b) Hysterectomy : definitive treatment , done when fertility is completed , TAH (total abdominal hysterectomy) / TVH (total vaginal hysterectomy)

Notes:

- Histology : it has a psuadocapsule of compressed smooth muscle cells (contain a few blood vessels and lymphatics)
- Natural history :

(a) Slow growth: most common, asymptomatic, if very large it causes pressure symptoms.

(b) Rapid growth : under the effect of estrogen (e.g. pregnancy), rule out leiomyosarcoma (very rare)

(c) Degenerative:

- Hyaline (most common)
- Red /hemorrhagic (most common in pregnancy)
- Cystic
- Calcific
- Sarcomatous

(d) Shrinkage

Indications for surgical treatment:

If symptomatic:

- Decrease in Hematocrit
- Pressure symptoms
- Symptoms limit lifestyle
- Emergency (torsion)
- Growth After menopause
- Rapid increase in size
- Size > 12 weeks obscuring exam of adnexa

Management during pregnancy :

- Bed rest
- Narcotics
- No surgery

Adenomyosis

Notes:

- Ectopic endometrial glands or tissues are located inside the myometrium.
- Most common presentation is diffused invasion of the myometrium.
- Risk factors : D&C, multipara

Approach:

1. **History:**
    - Mostly asymptomatic
    - If symptomatic : most common symptoms are dysmenorrhea and menorrhagia
2. **P/E:**
    - Bimanual examination : uterus is globular and diffusely enlarged
    - Tenderness is immediately before and during menses
3. **Investigations:**
    - U/S and MRI (to differentiate between fibroid and adenomyosis)
    - Biopsy (the only definitive diagnoses is through histology)
4. **Treatment :**
    - Medical : levonogestril IUD (synthetic progesterone) , OCPs, NSAIDS and GNRH agonists(for pain and bleeding)
    - Surgical : Hysterectomy (definitive)

Leiomyoma	Adenomyosis
Asymptomatic	Symptomatic
firm	Soft
localized	diffuse
Non-tender	tender
Pseudocapsule	True capsule

Pelvic Inflammatory Disease (PID)

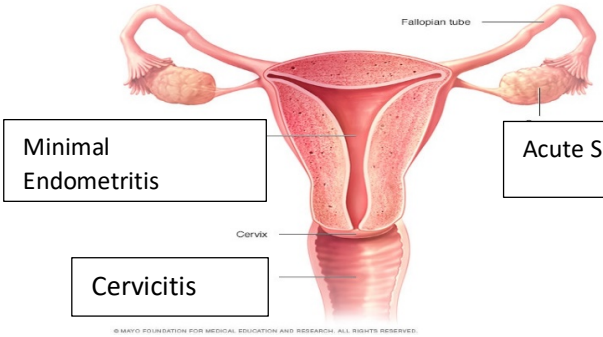
Inflammation of the female upper genital tract (uterus, tube, ovaries, ligament) caused mostly by ascending infection from the vagina and cervix

Organisms

- Neisseria Gonorrhea (second most common)
- Chlamydia Trachomatis (most common)
- E. coli
- Streptococcus
- Gardovella vaginallis /Bacteroids

Rarely is a single organism responsible for PID, but always think of chlamydia and gonorrhea first

Chlamydia is more common the N. Gonorrhea and has less acute symptoms



Minimal Endometritis

Acute Salpingtephotitis

Cervicitis

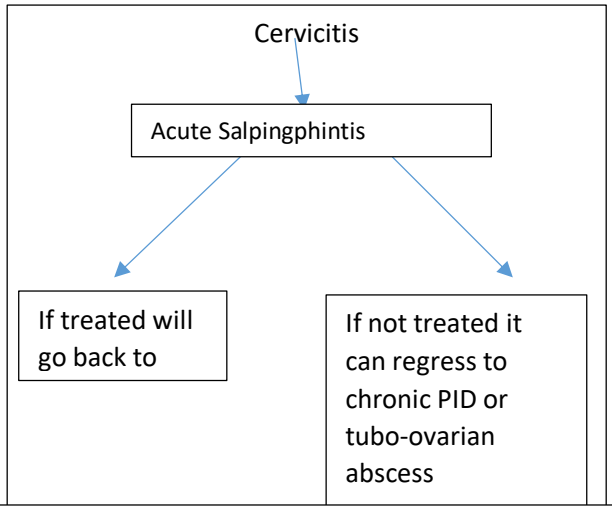
Pelvic Infection –Mechanisms

- Endometritis- lymphatics
- Pelvic TB – Hematogenous
- Acute PID – Ascending

PID is not a vaginal Infection

Risk Factors

- Age < 35 (increase risk x10 in teenagers)
- Multiple sexual partners
- Unprotected intercourse
- IUCD
- Nulliparous
- Concomitant history of STD



- Diagnosis is mainly clinical not bacteriological
  And positive tests are not necessary for diagnosis

Requirement for clinical diagnosis of PID

- Abdominal tenderness
- Adnexal tenderness
- Cervical motion tenderness

Contraception and PID

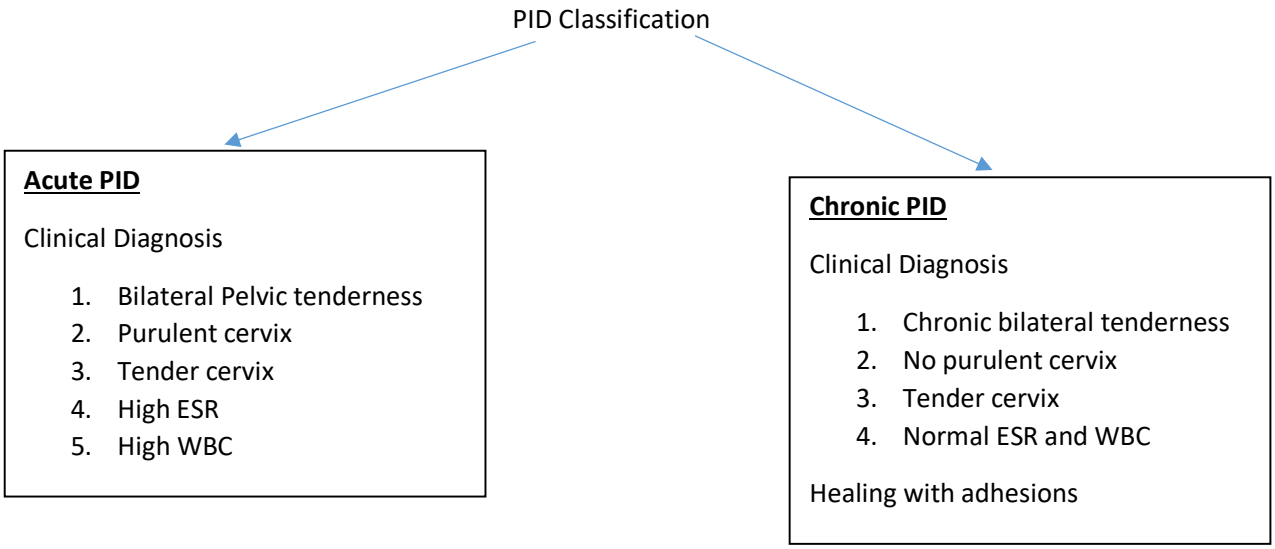
Increase risk

- IUCD

Decrease risk

- Barrier methods
- Combined OCP
- Progesterone

Abdominal pain is usually after menses due to breakage of cervical mucus


PID Classification
Acute PID
Clinical Diagnosis
1. Bilateral Pelvic tenderness
2. Purulent cervix
3. Tender cervix
4. High ESR
5. High WBC
Chronic PID
Clinical Diagnosis
1. Chronic bilateral tenderness
2. No purulent cervix
3. Tender cervix
4. Normal ESR and WBC
Healing with adhesions

Cervicitis	No Symptoms (maybe vaginal discharge) Physical Examination- mucopurulent cervical discharge, no pelvic tenderness, no fever Investigations- positive gonorrhea and or chlamydia Management -Azithromycin +cefixine oral once a day outpatient	
Acute PID	Symptoms- Bilateral pelvic pain Physical Examination- Bilateral abdominal tenderness, mucopurulent cervix discharge cervical motion tenderness Investigations- High WBc and ESR Positive culture Normal U/S Management – Outpatient -Ofloxacin (orally) + metronidazole for 2 weeks Inpatient -Cephalosporin IV+ Doxycycline IV / Clindamycin IV + Gentamycin	Differential Diagnosis ➢ Adnexal torsion ➢ Ectopic pregnancy ➢ Appendicitis ➢ Endometriosis ➢ Diverticulitis ➢ IBD
Tubo-ovarian Abscess	Symptoms – Severe bilateral pain, nausea and vomiting, lower abdominal, pelvic, back and rectal pain Physical Examination- Septic: high temperature High pulse, low BP, peritoneal signs, guarding Rigidity. Bilateral adnexal masses Investigations- positive cervical and blood Culture, high ESR and WBC CT= Bilateral complex pelvic masses Management- Usually responsive to antibiotics IV clindamycin + Gentamycin If no response in 72hours ⇨ explore or percutaneous drainage	Differential Diagnosis ➢ Septic abortions ➢ Diverticular Abscess ➢ Appendical Abscess ➢ Adnexal Torsion

Chronic PID	Symptoms- chronic bilateral pain associated with infertility, dyspareunia, ectopic pregnancy abnormal bleeding no nausea and vomiting Physical Examination- cervical motion tenderness and bilateral adnexal tenderness no discharge, no fever or tachycardia Investigations- WBC and ESR normal Negative cultures U/S hydrosalpinges Diagnosis- by laparoscopy (visualization of pelvi adhesions) Management- mild analgesia Adhesion lysis(helpful in infertility) TAH-BSO(ERT)- if severe unremitting pain	Laparoscopy is gold standard for diagnosis Findings ➢ Adhesions ➢ Pus collected in cul de sac ➢ Fitz-hugh-curtis syndrome :RUQ pain +chronic PID + perihepatitis +violin string adhesions(seen at the liver capsule)

Indications of hospitalization

- Outpatient treatment failure/ incompliance
- Pregnancy
- GI symptoms
- Abscess
- Systematic manifestations
- Peritonitis

Perinatal Infections

- Group B Beta-hemolytic Strep. (GBS)
  - Bacteria found in normal GIT
  - 30% of female have asymptomatic colonization with GBS
  - Do not treat positive GBS culture

Risk Factors

- Prematurity
- Positive maternal GBS urine culture
- Previous baby with positive culture

Transmission rate to neonate ➔ 50% colonization

Attack rate in neonate➔ 0.2% sepsis

Mortality rate in early onset sepsis ➔ 50%

Clinical Findings

- Newborn sepsis
- Acute within hours-days
- Bilateral diffuse pneumonia

Management

- by prophylaxis: Penicillin G

3 prophylaxis groups

- without screening if positive urine GBS previous neonatal sepsis
- based on culture positive third trimester vaginal culture
- Based on risk factors preterm/range of motion>18hrs /intrapartum

Toxoplasmosis

- Caused by parasite toxoplasma gonidil
- Up to 40% of pregnant women have toxo IgG positive those are protected from infection

Clinical presentation

When lethal? ➔ First trimester

When most occur? ➔ Third trimester

Prevention? ➔ Avoid contact with cat feces

Treatment ➔ Pyrnethamine and sulfadiazine

Mode of transmission ➔ infected cat feces/ pausturaized goat milk

Time of vertical transmission ➔primary parasitemia

Residual effect ➔ lifelong immunity

Congenital Toxoplasmosis

- Especially in first trimester

Triad

1. Chorioretinitis
2. Intracranial calcifications
3. Symmetrical IUGR

Effects on

1. Fetus

- Symmetrical IUGR
- Non-immune hydrops
- Microcephalus
- Intracranial calcification

2. Maternal

- Chorioretinitis
- Seizures
- Hepatosplenomegaly
- Thrombocytopenia

Varicella

- DNA virus
- Causes: - Chickenpox ➔ 1ry
  - Herpes zoster ➔2ry
- Clinical presentation ➔ VESICLES (Pruritic)
  More than 90% of females are immune by adulthood
  Maternal complication ➔ Pneumonia (BAD)! ☹
- Mode of transmission ➔ Respiratory droplets
- Residual effect of a 1ry infection is lifelong LATENCY in dorsal root ganglia (not immunity)
- The highest foetal infection occurs during the PERIPARTUM stage (5th day antepartum to the 2nd day postpartum)
- This infection can trigger labour
- Prevention:
  If pregnant ➔ VZIG (Varicella-Zoster Immune Globulin)
  Give it within 4 days of exposure
  If NOT pregnant ➔ VARIVAX (live attenuated vaccine)
- Treatment of (varicella pneumonia / encephalitis / immune-compromised) ➔ Acyclovir (safe in pregnancy)! 😊
- If exposure <20 weeks: **Congenital Varicella Syndrome**, TRIAD:
  1- "Zig-zag" skin lesions (scarring)
  2- Microphthalmia
  3- Extremity hypoplasia

Refer to the hand-written dossier for a helping tiny figure

Other symptoms:
Chorioretinitis, Cataract, Motor-sensory defects

Rubella (German measles)

- RNA virus
- Highly contagious
- 85% of pregnant women are IgG +ve
- Mode of transmission ➔ Respiratory droplets
- Time of vertical transmission ➔ Primary Viremia

- Residual effect is lifelong immunity
- Highest foetal infection occurs during the FIRST trimester
- Prevention:
  If NOT pregnant: Live attenuated vaccine + avoid pregnancy for 1 month from the vaccine administration time
  If pregnant: Nothing to do! ☹️
- Treatment ➔ No specific Treatment!
- **Congenital Rubella**, TRIAD:
  1- Congenital deafness (the most common)
  2- Congenital Cataract
  3- Heart disease

  Other symptoms:
  Mental retardation, Hepatosplenomegaly, Thrombocytopenia, Microcephalus, IUGR, "Blueberry muffin" rash

Refer to the handwritten dossier for a helping minifigure

CMV (Cytomegalovirus)

- The most common CONFENITAL viral infection
- DNA virus
- Sexually transmitted
- Most common cause of congenital deafness (sensorineural)
- 50% of pregnant females are IgG +ve
- Histology: Inclusion bodies
- Clinical presentation ➔ Flu-like symptoms
- Mode of transmission ➔ Body secretions (fluids)
- Residual effect ➔ Lifelong LATENCY (not immunity)
- Transplacental infection rate:
  If 1ry ➔ 50% (higher viral load)
  If 2ry ➔ 1% (like HSV)
- Prevention ➔ Universal precautions with fluids (gloves, …)
- Treatment ➔ Ganciclovir

- **Congenital CMV**, TRIAD:
    1. Neonatal Petechiae (SPECIFIC)
    2. Periventricular calcifications
    3. Symmetrical IUGR

    Neonate with petechiae ➔ Think of CMV

*Remember that calcifications of Toxoplasmosis are intracranial while those of CMV are periventricular!

Refer to the handwritten dossier for a minifigure about the difference between calcifications of Toxoplasmosis and CMV

HSV (Herpes Simplex Virus)

- DNA virus
- Sexually transmitted: (Genital herpes) HSV-2 > HSV-1
  *Remember that:
  Most common cause of PAINFUL genital ulcers ➔ HSV
  Most common cause of PAINLESS genital ulcers ➔ Syphilis
- Clinical presentation ➔ Vesicles/blisters (painful!)
- Definitive diagnosis ➔ Viral culture
- Mode of transmission ➔ Mucocutaneous contact
- Route of vertical transmission (when there are genital lesions) ➔ at delivery
- Residual effect ➔ Lifelong LATENCY (not immunity)
- Attack rate:
  If 1ry ➔ 50%
  If 2ry ➔ 1% (like CMV)
- Mortality rate in neonates ➔ 50%
- Prophylaxis ➔ Valaciclovir (if low asymptomatic transmission)
- Treatment:
  - *Acyclovir → (FDA C)

  Others → (both FDA B)

  - Valaciclovir
  - Famciclovir

HIV

- RNA virus
- Mode of transmission ➔ Body fluids
- Methods of transmission: IV drugs / Sex / Perinatal / Breast feeding
- Residual effect ➔ Lifelong LATENCY (not immunity)
- Results in AIDS and death due to opportunistic infections (TB, Toxoplasmosis, CMV, Coccidioidomycosis … etc.)
- Vertical transmission ➔ Vaginal delivery / Transplacental / Breast feeding
- Transmission rate:
  With Azidothymidine ➔ <10%
  Without Azidothymidine ➔ 30%
- Prophylaxis is given for all +ve mothers at 14 weeks regardless of their titre
- Treatment ➔ HAART therapy (multidrug therapy), NOT ONLY Azidothymidine. Why? To lower the resistance to Azidothymidine.
  Given for all HIV +ve females (↓ CD4, ↑ viral load)
- Delivery route ➔ C/S
  (especially if the viral load is > 1000 and CD4 count is low)
- Neonatal HIV test → +ve passive maternal IgG (if the mother is +ve)

HBV (Hepatitis B Virus)

- DNA virus
- Sexually transmitted
- Mode of transmission ➔ Body fluids / Blood
- Methods of transmission: IV drugs / Sex/ Perinatal
- HbeAg is usually an indicator for infectivity
- Symptoms: Usually none!
- Risk of transplacental infection ➔ LOW (most in 3rd trimester)
- Vertical Transmission ➔ Vaginal delivery (mainly) / Transplacental / Breast feeding
- Vertical Transmission rate:
  If +ve HbsAg ➔ 10%
  If +ve HbsAg & HbeAg ➔ 80%

- Chronicity:
  Adults ➔ 10%
  Neonates ➔ 80%
  (i.e. 80% of infected neonates develop chronic hepatitis)
- Maternal infection: Mostly asymptomatic carrier state
- Mode of delivery ➔ Vaginal
- Perinatal Management:
  - Active and passive immunization
  - Do NOT do scalp procedures

  Management of acute hepatitis ➔ None, maybe interferons

Syphilis

- Spirochetes
- Sexually transmitted
- Caused by Treponema Pallidum (a mobile anaerobic spirochete)
- Residual effect ➔ Neither latency nor immunity
  (i.e. it can be treated with appropriate treatment and a reinfection can occur)
- Mode of transmission ➔ Mucocutaneous contact
- Vertical transmission ➔ Transplacental (mainly)
- Perinatal mortality rate ➔ >50%
- **Congenital Syphilis**:

EARLY	LATE (after 2 years) <weird manifestations>
Hydrops (non-immune)	Hutchinson teeth
Anaemia & thrombocytopenia	Mulberry molars
Macerated skin	Saddle nose
	Sabre shins

  *Therefor; if you see a neonate with hydrops and macerated skin. Think of congenital syphilis.
- Treatment of choice ➔ Penicillin!
  If pregnant ➔ Benzathine penicillin
  If allergic to penicillin ➔ Acute desensitization

- **1ry syphilis**:
  - Localised chancre (painless raised edges)
  - VDRL ➔ -ve
  - Darkfield ➔ +ve
  - FTA-ABS ➔ +ve
- **2ry syphilis**:
  - Systemic
  - Condyloma latum
  - All labs are +ve (VDRL / Darkfield / FTA-ABS)
  - Then 2/3s become **Latent syphilis** and 1/3 becomes **3ry syphilis**
- **Latent syphilis**:
  - Symptoms are absent
  - Physical examination ➔ No signs
  - +ve non-specific tests
  - +ve Treponema pallidum tests
- **3ry syphilis**:
  - Symptoms are present, but variable
  - Gumma in CVS, CNS and bones
    (A gumma is a mass of dead and swollen fiber-like tissue)
  - +ve blood tests
  - +ve CSF (if CNS is involved)

Summary of Perinatal Infections

<table>
<tr><th>INFECTION</th><th>TREATMENT</th><th>LIFELONG</th><th>DELIVERY</th></tr>
<tr><td>GBβS</td><td>Penicillin G</td><td>Colonisation</td><td rowspan="3">Vaginal Delivery</td></tr>
<tr><td>Toxoplasmosis</td><td>Pyrimethamine</td><td rowspan="2">Immunity</td></tr>
<tr><td>Rubella</td><td>NONE ☹</td></tr>
<tr><td>CMV</td><td>Ganciclovir</td><td rowspan="4">Latency</td></tr>
<tr><td>Varicella</td><td rowspan="2">Acyclovir</td><td rowspan="3">C-Section</td></tr>
<tr><td>HSV</td></tr>
<tr><td>HIV</td><td>Azidothymidine</td></tr>
</table>

*__REMEMBER:__

- Toxoplasmosis ➔ Intracranial calcifications / Chorioretinitis
- Varicella ➔ Zig-zag lesions / Small eyes / hypoplastic limbs
- Rubella ➔ Deafness / Congenital heart disease/ Cataracts
- CMV ➔ Petechiae / Hepatosplenomegaly
- Syphilis ➔ Hydrops / Macerated skin
- HSV ➔NONE!
- HIV ➔ NONE!
- HBV ➔ NONE!

*Most common cause of PAINFUL genital ulcers → HSV

*Most common cause of PAINLESS genital ulcers → Syphilis

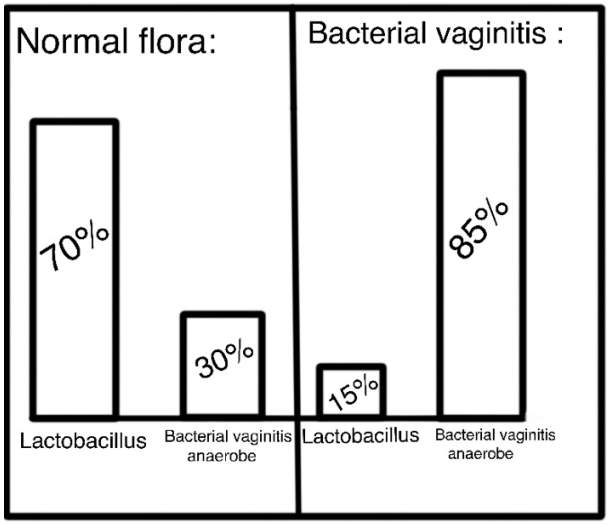
Vaginal Discharge

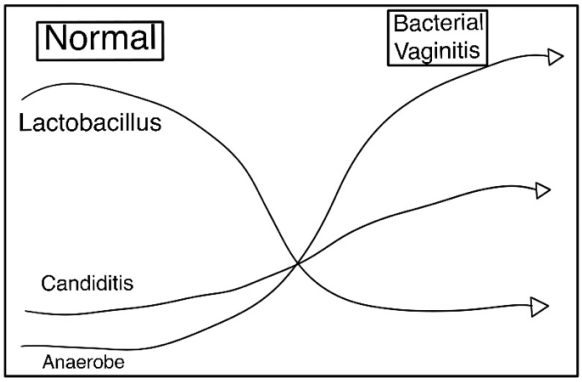
-The most common gynecological complaint.

Differential diagnosis:
- Bacterial vaginitis 50%
- Candida vaginitis 30%
- Trichomonas vaginitis 20%

- **Diagnostic tests**:

  Visual inspection:
  - Inflammatory response
  - Vaginal Discharge
    - (Thin/thick-gray/white/green-forthy





Vaginal PH:

- Normal PH: < 4.5
- Use Natrazine paper

Microscopic exam:

- Wet preparation
- Saline and KOH

- **Bacterial Vaginitis (Gardenella)**
  - Not a true infection, but alteration in concentration of normal vaginal bacteria
  - Seen commonly in postmenopausal females due to decreased estrogen.
  - Most common vaginal discharge in the U.S.
  - Sexually assocciated disease and not an STD (no effect with treatment of sex partner)
  - Associated with premature rupture of membrane and postpartum.
  - **Symptoms**: "Fishy" odor due to anaerobe), 50 % are asymptomatic.
  - **Speculum exam**:
    - PH > 5, no inflammation,
    - Discharge, homogenous
    - Positive "Whiff test"
    - Wet mount "clue cells"

"Whiff test:
Vaginal secretions are mixed with KOH → Amonia/fishy smell.
Whiff = Sniff !

  - Treatment is only in symptomatic patients
    - Metronidazole (safe during pregnancy) or Clindamycin. For 7 days. Oral, suppository and gel forms.

Trichomonas vaginitis

- Flagellated pear-shaped protozoan
- Can reside asymptomatically in male seminal fluid.
- **Symptoms**: itching and burning

Females are more symptomatic than males but both of them should be treated

❖ **Speculum exam:**
  - PH > 5
  - Green frothy discharge.
  - Strawberry cervix

❖ **Wet mount exam:**
  - Trichomonads, WBC in saline preparation.

❖ **Treatment:**
  - Metronidazole

Candida Albicans Vaginitis

- Yeast!
- **Symptoms**: severe itching and burning.

Risk factors:
- Pregnancy
- Treatment with antibiotics
- Diabetes mellitus
- Immune suppressants
- Clothing pattern (more temperature.. more moisture)

❖ **Speculum exam:**
  - PH < 4.5 (normal !)
  - Inflammation
  - White curdy discharge (cheesy)

❖ **Wet mount**
  - **:** pseudohyohae, WBC

❖ **Treatment:**
  - Oral fluconazole
  - Azole cream

Bacterial vaginitis	Trachomonas	Candiditis
Fishy odor	Itching and burning	Itching and burning
Increase in PH: grayish	Increase in PH: green frothy	Decrease in PH: white curdy
No inflammation	Inflammation	Inflammation
Saline: clue cells	Saline: Trich	KOH: Hyphae
Treatment: metronidazole or clindamycin	Treatment: metronidazole	Treatment: azole cream or flucomazole

Physiologic Discharge

It's the result of thin watery cervical mucus discharge seen with estrogen dominance.

- **Risk factors**: chronic anovulation (example: polycystic ovary syndrome)
- **Symptoms**: increase in watery vaginal discharge (the most common symptom) with no itching or burning.

❖ **Speculum exam:**
  - Thin and watery
  - Normal appearing epithelium
  - No inflammation
  - PH < 4.5 (normal)

❖ **Wet mount:**
  - No WBC/"clue cells/ trichomonads/ pseudohyphae

- **Treatment:**
  - Steroid contraception with progestin.. this will convert the thin watery estrogen dominant cervical discharge into a thick sticky progestin dominant mucus.

History for vaginal discharge

- **Discharge:** color, smell, amount, nature (thin watery, thick mucous, pus, blood, cheesy)
- **Timing:** duration/ first time ?
- **Associated symptoms:**
  - Fever, pelvic pain, itching, redness, rash ulcer or pain, dysmenorrhea, dyspareunia, postcoital bleeding, intermenstrual bleeding, weight loss, has a husband?, urinary frequency urgency and dysuria.
- **Ask deferential diagnosis:**
  - Cervical cancer and PAP smear.
  - Local → allergies → new perfume?
  - Infection and immunosuppression (Aids, steroids, SLE…)
  - Previous infection.
  - Intrauterine contraceptive device?
  - Drugs? Oral contraceptives, antibiotics, steroids ?
    - **Differential diagnosis:**
      - Infections (PID, bacterial, candida)
      - Local allergy.
      - Urinary.
      - Drugs
      - Immune suppressants.
      - Cancer
- **Physical examination:**
  - Inspect the area for redness, ulcers, discharge.
  - Swab for culture
  - Speculum
  - PAP smear
  - Pelvic ultrasound.
- **Investigations**:
  - WBC, CRP, ESR
  - Wet mount
  - Whiff test
  - PH

Sexually transmitted disease (STD)

STD with ulcers:
- HSV
- Syphilis
- Chancroid
- Lymphogranuloma venereum (LGV)
- Granuloma inguinale (donovanosis)

STD without ulcers:
- Trichomonas vaginalis
- Chlamydia
- Gonorrhea
- Condyloma acuminatum
- HPV
- Hepatitis B
- HIV

- **STD with ulcers:**
  - HSV (in perinatal infants)
  - Syphilis (in perinatal infants)
  - Chancroid:
    - Gram negative bacteria
    - Uncommon in US
    - HIV cofactor
    - **Symptoms:** painful ulcer
    - **Exam:** ragged edge
    - **Diagnosis:** positive culture
    - **Management:** azithromycin
  - Lymphogranuloma venereum (LGV)
    - Steriotype of chlamydia trachomatis
    - Background: uncommon in US
    - **Symptoms:** painless ulcer
    - **Physical exam:** groove sign
    - **Diagnosis:** positive culture (from aspirated lymph node pus)
    - **Management:** doxycycline or erythromycin

-Most common painful /STD is HSV -Most common painless STD is syphilis	Painful STD: - chancroid - HSV

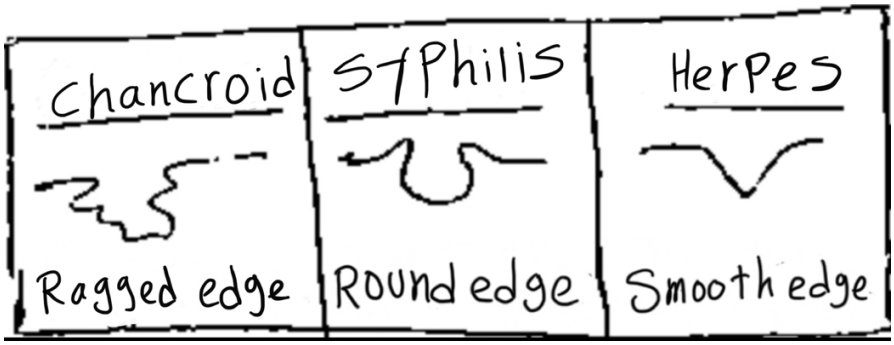
Organisms:
-Bacterial:
- Chlamydia, gonorrhea, syphilis, chancroid, LGV

-Viral:
- HPV, HIV, HBV, HSV

-Protozoa:
- Trichomonas

❖ First most common is HPV
❖ Second most common is trichomonas
❖ Third most common is chlamydia

"Groove sign"
Double genitocrural fold where you lymph node on either sides of inguinal ligament.



- **Granulosa inguinale:**
  - Background: uncommon in US but common in south Africa
  - **Symptoms:** painless ulcer
  - **Physical exam:** beefy red ulcer (due to granolation tissue)
  - **Diagnosis:** microbe: Donovan bodres
  - **Management:** doxycycline(first line of treatment, 100mg x 1) or TMX

❖ **Summary:**
- Chancroid is painful, has ragged edge, inflammation
- LGV has groove sign
- Granulosa inguinale is beefy .. donovan bodies
- Syphilis has a rolled hard edge
- Herpes is painful and has smooth edge, inflammation

- **STD without ulcer:**
  - Trichomonas Vaginitis (mentioned in vaginal discharge)
  - **Chlamydia:**
    - Most common bacterial STD and the third most common STD
    - Common in teens
      - Caused by chlamydia trachomatis (obligatory intracellular)
      - Five ties more common than gonorrhea
      - **Complications:**
      - Adhesions.. pelvic inflammatory disease.. infertility
      - Vertical transmission .. conjunctivitis
      - Reiter's syndrome
      - **Symptoms:** NONE! Even with salpingo oophoritits.
    - **Physical exam:** mucopurulant cervical discharge, positive urethral and cervical motion tenderness

3 serotypes L1-L3
Forms:
Primary lesion, purple painless on genetalia
Secondary: inguinal lymphatics with fever, malaise, and decreased appetite .

- **Diagnosis:** PCE amplification or DNA probe. Culture doesn't detect it. Cervical and urethral screening.
- **Management:** azithromycin or doxycycline and threat sexual partner.

Trachoma: conjunctivitis resulting in eyelash hypercurvature of extended blindness from corneal accesses

Gonorrhea

It's an infection of the pharynx, urethra ,cervix and anal canal
Obligatory G-ve diplococci

Complications

Adhesion > PID >>infertility
Systemic infix can occur

Sites

-Lower reproductive tract: bartholin abscess / cyst
-upper reproductive tract: PID
-disseminated : septic arthritis

Symptoms

(According to site)

-lower reproductive tract: volvovaginal discharge, itching and burning with dysuria and rectal pain.
-upper reproductive tract: bilateral pelvic pain.
-Disseminated of: dermatitis, polyarthralgia, tenosynovitis

*50-90% chance of vertical transmission after exposure to GC

Physical exam

-Inspection: vulvoginitis is seen
-Speculum: mucupurulent cervical discharge bartholin abscess is seen (if obstructed due to acute infection should be incised or drained.
+ve cervical motion tenderness

*Petechial skin lesion/septic arthritis
*endocarditis/meningitis. (Rare)

Investigations
Like chlamydia: urethra, cervical of rectal culture
PCR (gold standard).

Management

Dual therapy
Cefixene +azithromycin (oral dose *1)
don't forget to Treat the sexual partner.

*physician usually treat both chlamydia and GC even if diagnosing only one.

HBV
in perinatal infant
HIV
In perinatal infant

Ectopic pregnancy

Definition: it's a pregnancy that is located outside the uterine cavity. (Not outside the uterus cause cervix implant is an ectopic pregnancy.

Incidence: not that common, 2% of reported pregnancy.
*it's the leading cause of maternal mortality 6%.

Site
1-Fallopian tubes (95%)
Ampulla: most common site, the widest part 5-6 mm
Isthmus: wall is thicker
Fimbria
*both isthmus and ampulla ectopic pregnancy need short weeks of amenorrhea to appear. (Ampulla needs 6-7 week, isthmus <6 week)
2-uterine cornea: need 10 weeks to appear, the most dangerous due to risk rupture
3-cervical: 0.2%
4-ovarian: 0.2%
5-abdomin:02%
There will be placenta on the bowel of this type may continue and reach term.
*4&5 type happen secondary to tubal abortion
*the most common type of ectopic pregnancy is ampulla and the second most common is abdominal

Risk factors
1-previous history of ectopic pregnancy. The biggest RF
2- PID (STD) and infection (TB): most common cause in developed countries. Due to -intratubal or peritubal adhesions -infection may destroy the cilia this will suppress migration
3-previous tubal surgeries ex: tubal ligation
4-use of ART ex: IVF
5-use of contraceptive methods:POP,IUCD (m.c in developing countries
6- smoking
7-exposure to DES
8-congenital malformation of the uterus

*Deferential diagnosis for first trimester bleeding: abortion / ectopic pregnancy / molar pregnancy.

Clinical presentation
Tried of
-Amenorrhea
-Abdominal pain: usually acute pain, pelvic or lower abdominal pain radiating to the shoulder-ipsilateral (suspected rupture).
-Vaginal bleeding: spotting, if ruptured then it's intraperitoneal bleeding.

Approach

History

Ask about LMP/ gyne history/ drug of pregnancy
Ask about abdominal pain (socrates)
Ask about vaginal bleeding
-RF
-DDx
***DDx for ectopic pregnancy:**
A-normal IUP
B-spontaneous abortion
C-molar pregnancy , both b+c have bleeding
D-Ovarian portion/ruptured ovarian cyst
E-PID/ acute appendicitis/ tube-ovary abscess
F-degenerating fibroid , (D,E&F has abdominal pain)

Physical exam

U/s , hemodynamic stability
Abdominal exam
Bimanual exam /pv -palpate adrexial mass -cervical tenderness

Investgaton
1-Labs
*B-HCG
To confirm pregnancy if positive >1500 iu/ml by vaginal ultrasound if >3000/4000 by abdominal ultra sound.
Doubling, in Normal pregnancy HCG must increase 60% after 48 hours and doubles after 72 hours.
If not then it/s ectopic pregnancy.

*progesterone level
>20 mg/ml : good pregnancy
<5 mg/ml: bad pregnancy, either ectopic pregnancy or abx we can't differentiate

5-20: unclear and not helpful

2-ultrasound
-if you see IUP: then this is abx
In this case there are bleeding/abdominal pain.
-you may see adrenal mass
-you may see free fluid in the pouch of doglus
-fetal heart in the adnexia

*Ultrasound findings suggestive of ectopic pregnancy: Absent intrauterine sac, ectopic sac/A+A, complex adrexial mass, fluid in cudle sac.

3-Invasive procedure:
-Culdecentesis (old procedure)
A needle is inserted through the posterior firing of vagina to the pouch of dodges, then we aspirate.
*if aspiration was dark, non clotted we suspect ectopic pregnancy
-uterine curettage (not done anymore)
Indication: used when pt has history of passing tissues (decidua)
So by Dilation and curettage no chronic villi: ectopic pregnancy

4-surgical: laparoscopy (the definitive Diagnostic)
Diagnostic and therapeutics

Management
It depends on
Stability, site of ectopic pregnancy, state (rupture?),desire of future fertility, experience.
A-expectant (observation)
If stable, no significant bleeding, no significant pain, no rupture, falling of HCG, ectopic in tube, size <4cm

B-medical:
Drug of choice is MTX
Indication : stable B-HCG<1500
On U/S: no IUP, no FHA, size <4cm
On D&C: no villi
*MTX is an antimetabolite and folate antagonist.
Treatment with MTX you give one shot 50mg/m2 IM or 1mg/kg
Then follow B-HCG after 3-7 days it should decrease 15%, if not or plate repeat dose after 2 weeks if increase surgery. Don't use NSAIDs with MTX, cause this may potentiate nephrotoxicity
*contraindication for MTX

-unstable patient or ruptured EP (cause MTX takes time to work)
-leukopenia/ thrombocyte <100k
-active renal/ hepatic disease
-active PUD
-breast feeding
-positive FHA (possible viable pregnancy)

C-surgical:
Indication: failure of medical treatment, unstable, B-HCG>1000, positive FHA, size>4cm.
if stable we do laparoscopy, If unstable we do laparotomy
Salpingectomy (removal of tube), no need for F/U
Salpingostomy (incision on the anti-mesenteric portion of the tube)
-used for unruptured distal tube ectopic pregnancy
-spares the tube
-higher risk for recurrence
*you should follow B-HCG down to zero

Gestational Trophoblastic Disease

-Types:
1.Molar pregnancy (80%,benign)
2.Persistent /invasive mole (10-15%, malignant)
3.Choriocarcinoma (2-5%,malignant)
4.Placental site trophoblastic tumor (very rate / malignant)

-Congenital trophoblastic disease : disease group of interrelated disease resulting in abnormal proliferation of trophoblastic (placental) tissue
-Common findings (in all Congenital trophoblastic disease) :
1.Abnormal fetal tissue
2. produce Beta hCG
3.Extremely sensitive to chemotherapy
It is the most curable gynecologic malignancy and fertility preservation

Molar pregnancy

-As known as hydatiform mole (الحمل العنقودي)
-benign
-**Types** :
1)Complete (classic) 90%
2)Incomplete(partial) 10%

-Risk factors:
***Major** :
1) previous history of Congenital trophoblastic disease
2) Extremes of age (<20 or >35)
***Minor:**
3) Nulliparity 70%
4)Diet (low beta carotene, low Folic acid and animal fat) /low socioeconomic factors)
5) Smoking
6)infertility
7) history of OCP use
8) blood Group A

	Complete mole	Partial mole
Genetics -most common Karyotype -Chromosome origin	 -46 , XX -All paternally derived	 -Extra paternal set
Pathology -Coexistent fetus -Fetal RBCs -Chorionic villi -trophoblasts	 -Absent -Absent -Hydropic(swollen) grape like vesicles -Severe hyperplasia	 -Present -present -Few hydropic -Minimal or no hyperplasia
Clinical assessment Assessment of embryo Signs and symptoms -Classic symptoms -Uterine size -theca luteint cysts -hCG level	 -None -Abnormal vaginal bleeding (due to separation of tumor from deciduas) -common -50% large for date -Present in 25%(due to increase beta hCG,LH,FSH) -High(more than 100,000	 -present -Missed abortion -Rare -Size equals dates -Rare -Slightly elevated
Malignant potential -Non metastatic -Metastatic(brain, liver,kidney, lung)	*higher risk for mits -15-25% -4%	 -2-4% -0% none
Follow up Weeks to normal hCG	 14 weeks	 8 weeks

Notes:
-In complete molar pregnancy, there is proliferation of the syncytiotrophoblasts which produces hCG
-HCG subunits are alpha and beta, alpha subunit is also found in LH (large theca luteint cyst > 6 cm), FSH, TSH (hyperthyroidism)
- Increase hCG leads to hyperemesis gravidarum and pre-eclampsia

-**Approach**:
***History** (related to increase HCG) :

- main complain :early vaginal bleeding (causing anemia; shortness of breath, pallor..)
-passage of molar vesicles
-hyperemesis gravidarum (nausea/vomiting)
-hyperthyroidism symptoms (heat intolerance, diarrhea..)
-pre-eclampsia symptoms, onset <20 Weeks, (headache, visual disturbances, epigastric pain, HTN)
-Ask risk factors

***Differential diagnosis :**
- Any thing increases hCG will cause bleeding
- normal intrauterine pregnancy
-Multiple gestations
- Ectopic pregnancy
-Antibiotics
- fibroid
*** Physical examination:**
- General: pallor, signs of hyperthyroidism
-Vital signs : increases Pulse rate and respiratory rate for hyperthyroidism ,increase blood pressure (pre-eclamptic toxemia)
-Abdomen : fundal light large for date
-pelvic exam :expulsion of grape like molar clusters, blood in cervical os, you may palate large bilateral theca luteint cysts
***Diagnosis** :
Ultrasound:
- No fetus (complete), growth restricted fetus (incomplete), no amniotic fluid (complete), low amniotic fluid (incomplete), ("snow storm" appearance in hydropic villi due to swelling of chorionic villi, large bilateral theca luteint cyst(>6 cm, multilobular, complete)
-Lab: beta-hCG : if it was very high >100,000(complete), if it was normal or slightly high (incomplete)
-You should do serial beta HCG follow up, why? Assessment of treatment effectiveness, diagnosis and risk stratification
-Always check for pre-eclamptic toxemia, hyperthyroidism, anemia
***Pathology** :
once evacuated, the definitive diagnosis
***Treatment**:
It will not continue as viable pregnancy so:
Immediate removal of uterine contents and suction curettage (evacuation)
* Prior to evaluation :
-baseline hCG
-chest X ray

-CBC, coagulation profile
-KFT, LFT, TFT
-blood Group, XM, Rh
- correct pre-eclamptic toxemia, hyperthyroidism, anemia
-We do hysterectomy (for placental site trophoblastic tumor) if patient completed her family, no risk of local invasion, doesn't prevent metastasis
***Follow up**
-serial beta hCG
-reliable contraception (6minths-1year) …important
-metastatic follow up :
1) chest X ray, CT for brain, kidney, liver, lung
2) labs (CBC, clotting studied, KFT, LFT, TFT, blood Group, Rh, antibodies)

Invasive mole:

-Malignant gestational trophoblastic disease, invades myometrium or blood vessels
- can spread to extrauterine sites
- 20% of complete moles will develop invasive moles
***Treatment** :
-complete mets work up
-chemotherapy (not dilation and curettage due to risk of uterine proliferation)

Urinary incontinence:

1)**Autonomic** :

-Sympathetic : hypogastric nerve (T10-L2) :continence

-Parasympathetic(cholinergic) : pelvic nerves (S2, S3, S4) : micturition (voiding)

2) **Somatic**: pudendal nerve (S2,S3,S4) :voluntary prevention of micturition by striated muscle of extensor sphincter and pelvic floor

***Cystometric volume measurements :**

-Normal bladder residual <50 ml

-Sensation of fullness (200-250 ml)

-Urge to void (400-500ml)

***Classification** :

-Irritative incontinence

-Stress incontinence

-Urge incontinence

-Overflow incontinence

-Bypass (continuous) incontinence

-other :functional incontinence

Irritative incontinence:

-Etiology : involuntary detrusor contraction (UTI, stone, tumor, foreign body)

-History : Urgency, frequency, dysuria (does occur at night)

-Physical examination : Suprapubic tenderness, normal pelvic of neuro exam

-Investigation :urine analysis, urine culture, CBC

- Management : antibiotics (in infection), Cystoscopy(in microscopic hematuria)

Stress incontinence

- the only one does not occur at night

-Etiology: loss of bladder support (anatomic problem) :intraurethral pressure <bladder pressure

- Risk factors :

1. increase age (menopause)

2.multiple vaginal deliveries

3.obesity

4.constipation,chronic cough

5.chronic heavy lifting

-History :small amount urine loss, on exertion (coughing, sneezing, laughing),does not occur at night

-Investigation : absent detrusor muscle(normal cystometry)

-Management :

Medical: Kegals, hormone replacement therapy

Surgical (mainly) : Urethropexy (tension-free vaginal tape procedure)

Urge Incontinence:

(The only one that has detrusor contractions) .

Hypertonic bladder:

-Etiology: Idiopathic detrusor overactivity, maybe due to upper motor neuron lesion.

-History: large amount of urine loss without warning.
Involuntary.
day and night with urgency.

-Physical examination: empty bladder.

-Investigation: detrusor contractions present.

-Management: medical treatment: Anticholinergics eg: Oxybutynin and Tolterodine (Detrol). NASAIDs, tricyclics.

Surgery ISN'T effective.

Overflow incontinence: HYPOtonic bladder, AKA: Neurogenic bladder.

-Etiology: Neurogenic causes:
LMND, DM, spinal cord and injuries, MS
Obstructive causes.
Drugs: Anticholinergics
α-adrenergic agonists
epidural and spinal anesthesia.

-History: loss of small amounts of urine
Intermittency day and night

-Physical examination: FULL bladder
abnormal neural exam

-Investigation: Increase in residual volume
absent detrusor contractions

-Management: Intermittent self catheterization (especially if chronic disease)
RX: cholinergic eg Bethanechol chloride

**Mixed incontinence:
Urge and stress incontinence
day and night incontinence with exertion

Bypass incontinence: Fistula

-Etiology: pelvic surgery and radio therapy

-History: continuous urine loss DAY AND NIGHT

-Physical examination: NORMAL neuro exam

-Investigation: intravenous pyelogram(IVP) Dye leakage.
IV blue dye---> leaks onto a vaginal tampon.

-Management: Surgical only (not medical).

Functional incontinence:

-It's attributed to factors OUTSIDE the lower urinary tract.
urinary tract is completely normal
Induces physical or mental impairments which prevents the patient from being able to respond normally to cues to void.
Treat the cause if applicable.

OSCE:
-History taking:
age, parity?
ask urinary symptoms: frequency/urgency/ dysuria/ hematuria/ and since when?
AMOUNT of urine loss
Timing (day and night or only daytime), nycturia
Does it increase with excretion?
Continuous or intermittent ?

-Drug history, past medical, past surgical, and exposure to radiation, social history(smoking) and family history.

-Interferes with daily activities (severity)

-ask also about prolapse symptoms: sensation of a lump…..

Pelvic Organ Prolapse (POP)

-Anatomy:
The pelvic flour is made up of the diaphragm and perineal muscle.

-Pelvic diaphragm:
consists of levator ani & coccygeus muscles.
levator ani consists of :(the pubococcygeus, the iliococcygeus, and the puborectalis).

-Perineal membrane (urogenital diaphragm)
-Triangular sheet of dense fibromuscular tissue that spans the anterior half of pelvic outlet.
-vagina and urethra pass through it.

-Uterine support:
Main structures for support: The cardinal ligament, uterosacral ligament and endopelvic fascia.

-Causes and Risk factors: concept: Anything that increases in abdominal pressure and decreases in pelvic support.

-Causes of increased abdominal pressure:
Obesity and COPD(chronic cough)
Constipation/ heavy lifting/ tumors.

-Weaning/relaxation of pelvis:(decrease in connective tissue)
age, menopause, HRT, multiple vaginal deliveries & traumatic vaginal delivery.

-previous history of pelvic organ prolapse (POP)

-Classification: -uterine prolapse
- cystocele (anterior vaginal prolapse): herniation of the bladder
-rectocele (posterior vaginal prolapse): herniation of the rectum.
-enterocele(herniation of the bowel into the pouch of Douglas).

-Grading: of Uterine prolapse (according to dr ayman)
grade 1: Above the hymen >1cm from hymen
grade 2: at the level of hymen plus minus 1cm
grade 3: below the hymen >1cm
grade 4: uterus outside the vagina (Procidentia)

Notes:
*Cystocele: think of it in :
post menopausal woman
anterior vaginal wall protrusion
urinary incontinence

*Rectocele: think of it in:
postmenopausal woman
post vaginal wall protrusion
digitally assisted removal of stool.

-Diagnosis:
Observation at time of pelvic exam, if the patient has increased abdominal pressure then you look for prolapse of vaginal wall/cervix/uterine/rectum.

-Management:
medical:
Kegel exercises (voluntary contractions of pubococcygeus muscle)
ERT
vaginal pessaries

-Surgical:
Anterior and posterior Colporrhaphy (for cystocele/ rectocele)
vaginal hysterectomy

**After repair if the patient wants to get pregnant we usually plan for elective C/s section.

History:OSCE

-patient profile: Age and parity (vaginal delivery)

*Ask symptoms of ---->
- General : embarrassing, lack of pleasure, backache, dyspareunia.

-Urinary: incontinence , retention, dysuria, frequency, nycturia, hesitancy and hematuria.

-GI: Bowel obstruction, constipation, difficult defecation, incomplete defecation, painful defecation, fecal incontinence, soiling and hernia.

-Past medical, surgical, drug, family and social histories.

-ASK FOR RISK FACTORS.

*DDX:
-Prolapse
-urethral diverticula / skene glands.
-intestinal tumor

Cervical Cancer Screening

- Pap smear is used for screening purposes only and not for diagnosis.
- Screening starts at the age of 21 regardless of the age of marriage, not before… why? Because there is increased risk for false positive i.e. normal dysplasia.

Screening program:

- 21- 29 years every 1-2 years
- >30 years every 3 years but if high risk patient screen annually.
- >65-70 years stop the screening if there was a history of 3 negative consecutive pap smears in the last 10 years or no risk factors; otherwise you continue screening annually.
  - Some doctors think we should continue screening for ever even after the age of 70 years

If the patient underwent hysterectomy for a benign condition:

- No pap smears for total abdominal hysterectomy
- Follow the same protocol if supracervical hysterectomy was performed

18 years is a critical age because younger than 18 years the transformation zone is highly undifferentiated → risky for abnormal growth

Older than 18 years differentiation

Prerequisites for pap smear:

There should:

- no blood (bleeding)
- no discharge (infection)
- no semen (no sexual intercourse in the last 48 hours)
- no hormone (hormonal therapy)
- no recent PV

100% of cases with cervical CA have the HPV virus.

Risk factors for cervical CA

- Teenagers
- Multiple sexual partners
- Smoking
- HPV infection (16,18)

How to apply a pap smear important OSCE station:

- Intoduce yourself, good light, exposure, permission, privacy and chaperone
- Lithotomy position, gyne bed
- Explain what you are going to do (not painful and not comfortable)
- Start the examination by inspecting the vulva for laceration, lesions, blood or discharge
- Apply the speculum under aseptic techniques, the speculum should be warm and apply a lubricant may be water but not a gel as it may interfere with the sample interpretation, apply it obliquely then rotate and then fix the sample.
- Inspect the lateral wall of the vagina for polyps discharge or laceration and inspect the cervix for its shape the os and lacerations.
- Apply the brush rotate 180 degrees inside the os
- Apply spatula and then rotate 360 degrees endo and ectocervix
- Then put it on the slide and fix it with 95% alcohol KOH
- Don’t forget to inspect the ant and post vaginal walls

Cytology report discusses several points which are the adequacy of the sample, abnormal cells, inflammatory cells and endometrial cells.

- Adequacy of the sample means that it contains cells from the endo an ecto cervix as well as the t-zone
- Presence of abnormal cells, negative, ASC, LSIL, HSIL
- Inflammatory cells
- Presence of endometrial cells, if normal endometrial cells are present in post menopausal women the next step would be hysteroscopy, D&C.
- **Remember** that normal pap smear does not rule out cancer, 10% false positive rate.

Bethesda III system

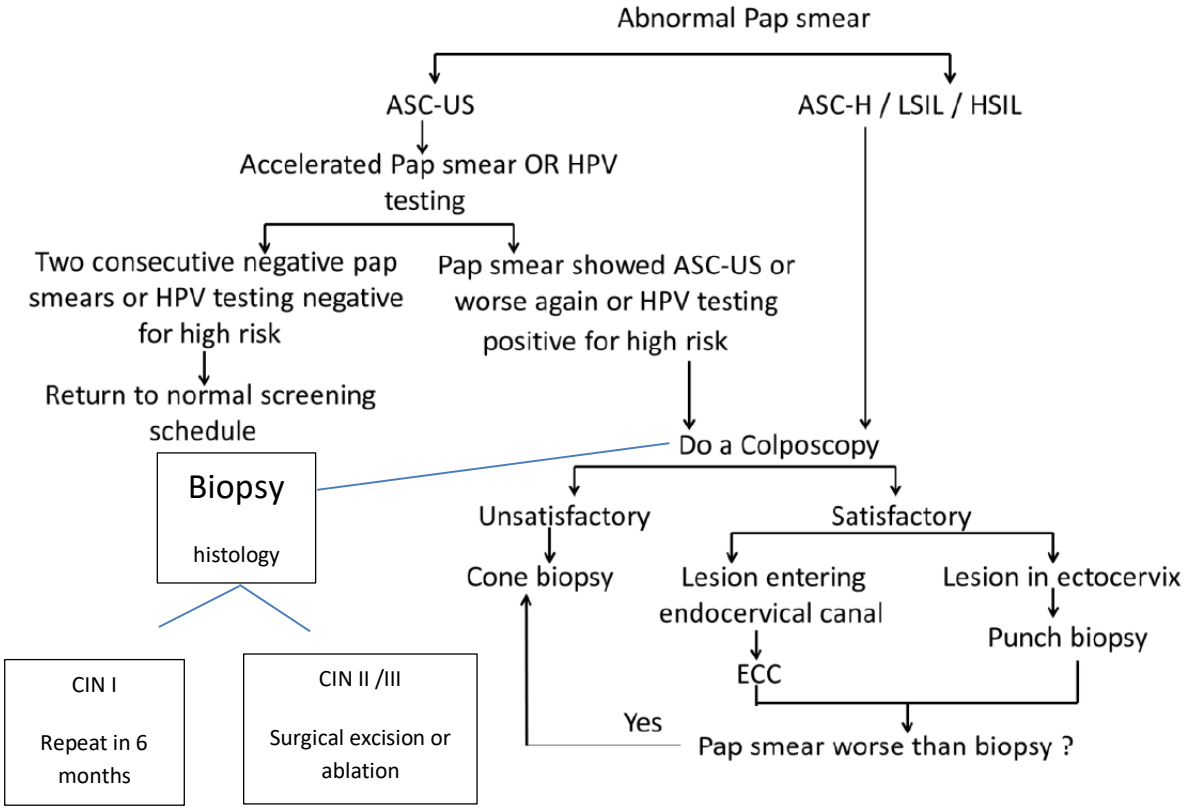
Cytology (pap smear)	Histology (biopsy)
ASC atypical squamous cells ASC-US – undifferentiated significance → ASC-h –cannot exclude HSIL →	Next HPV testing HR—colposcopy LR—pap in 1 year Colposcopy with biopsy
LSIL low grade squamous intraepithelial lesion	CIN I mild dysplastic lesion
HSIL high grade squamous intraepithelial lesion	CIN II moderate dysplastic lesion or CIN III severe dysplastic lesion

Next step in ASC-H, LSIL, HSIL is colposcopy with biopsy

Biopsy results

- CIN I pap smear in 1 year
- CIN II CIN III surgical excision

- CIN is not invasive cancer but precancerous lesion.



- Ablative modalities:
    - Used mainly for CIN II/III but can be used in CIN I
    - Cryothrapy, laser vaporization, electrofulgration.
- Excisional modalities:
    - Used for CIN II/III but can also be used for CIN I.
    - LEEP loop electrosurgical excision procedure, and cold knife excision.
- Hysterectomy:
    - Not routine and is usually not done unless there is recurrent CIN II/III that is proved by biopsy.
- Indications for cone biopsy:
    - Mosaicism
    - Punctation
    - White epithelium
    - Abnormal vessels.

Cervical cancer

- It is the second most common gyne cancer worldwide after endometrial cancer
- The incidence decreased significantly after the introduction pap smear
- 100% of patients with cervical cancer have HPV infection.
- Age of diagnosis **45** years.

➢ Risk factors:
    - Age of first intercourse.
    - Multiple partners.
    - Promiscuous male partner.
    - Hx of STD.
    - Smoking.
    - Others, OCP and increased parity.

➢ History:
    - Most common is an abnormal pap smear.
    - Clinically the first finding is post coital bleeding as the cancer is vascular and friable.
    - Intermenstrual bleeding
    - Post menopausal bleeding

- Sometimes vaginal discharge, dysuria, moderate discomfort.
- Late symptoms:
  - Obstructive signs, uropathy
  - Peritoneal edema
- Metastatic signs:
  - Weight loss and decreased appetite

- Physical examination:
  - No gyne exam is completed without speculum and/or PV.
  - As the disease progresses the cervix may becaome abnormal in appearance with gross erosions, ulcers or masses (they may extend to the vagina).
  - Bimanual exam may reveal pelvic masses.
  - Rectovaginal may reveal peritoneal invasion.
- HPV
  - Sources:
    - Sexually transmitted
    - Contaminated speculum
    - Pools
    - Clothes contaminated with a fluid containing HPV
  - It has more than 75 serotypes
  - 2 types for squamous cell carcinoma:
    - Low risk 6,11 never invasive CA
    - High risk 16, 18 risk for invasive CA
- Intermenstrual bleeding between regular cycles indicates anatomical abnormality such as CA/lesion.

Colposcopy:

- We use it to **determine** the area for biopsy.
- We see a magnified view of the cervix given that there is no bleeding, no discharge and no recent sexual activity in the last 48 hours.
- Procedure: speculum is put after which 8% acetic acid solution is put for 30 seconds then report your findings.
- **Important**: colposcopic findings do not rule out malignancy in high risk patients.

- **Abnormal findings**:
    - Atypical T-zone or abnormal blood vessels.
    - Keratosis.
    - Aceto-white epithelium
        - The acetic does not usually penetrate the normal cells, instead it penetrates the abnormal ones including; inflammatory, premalignant and malignant cells, these become dry→destructed →white color.
    - Punctation.
    - Mosaicism.
    - Suspected frank malignancy.
    - Unsatisfactory colposcopic findings.

Investigations:

- Pap smear (for screening only).
- Colposcopy, direct biopsy.
- Fractional D&C.
- Routine blood tests.
- Ca125 levels, bear in mind that this is not specific for cervical cancer.
- Others:
    - CXR, rule out metastasis, and check if candidate for surgery.
    - Urine cytology/ Cystoscopy/ proctosigmoidoscopy "to check for metastasis".
    - CT/ MRI/ Pet scan.

Fractional D&C:

- Curetting the endocervical canal with a long sharp curette after which we place the sample in a jar, then we dilate the internal os and we curette the endometrium placing the sample in a different jar. **Because**, it is important to differentiate between the two specimens as they are differ in treatment, in cervical CA we <u>spare</u> the ovaries while in endometrial we <u>don't</u> spare them.

Types of cervical CA:

- SCC “squamous cell carcinoma”, it is the most common and associated with HPV.
- Small cell carcinoma, this is the worst type.
- Adeno, associated with
- Large cell type.

Staging:

- It is the only cancer that is staged clinically;
  - on rectovaginal exam, if positive para-metrium involvement it's stage II B
  - on bimanual exam, if pelvic mass → It is stage III B
  - Involvement of bladder (cystoscopy) →It is stage IV A
  - if positive peritoneal cytology → It is stage IV B
- Beyond stage IIA “that is stage IIB and above” the patient is not a candidate for surgery but for chemo and radio treatment.

➢ Stage I:
  - ✓ Ia diagnosis only by microscopic exam
    - Ia1 depth ≤ 3mm or extension ≤ 7mm
    - Ia2 depth 3-5 mm or extension ≤ 7mm
  - ✓ Ib clinically visible – b benshaf
    - I**b**1 greatest dimension: ≤ 4cm
    - Ib2 greatest dimension: > 4cm

➢ Stage II:
  - ✓ clinically visible lesion that invades beyond the uterus, but **not** to the pelvic wall and **not** to the lower third of the vagina.
  - ✓ IIa upper 2/3 of the vagina.
    - IIa1 <4cm
    - IIa2 >4cm
  - ✓ Para-metrium involvement.

➢ Stage III:
  - ✓ extends to the pelvic wall **and/or** involves lower third of the vagina **and/or** causes hydronephrosis or nonfunctioning kidney (unless they are known to be for another cause)
  - ✓ IIIa Lower 1/3 of the vagina but not reaching the pelvic side walls

- ✓ IIIb extension to the pelvic side walls or causing hydronephrosis.

➢ Stage IV:

- ✓ extended beyond the true pelvis or has involved (biopsy proven) the mucosa of the bladder or rectum or distant metastasis.
- ✓ IVa adjacent organs
- ✓ IVb distant organs

- **Treatment**

Depends on the stage.

- For early invasive CA → surgery is the treatment of choice.
- In more advanced cases (IIb and beyond) → radiotherapy and chemotherapy is the standard [cisplatin-based chemotherapy increases sensitivity of cancerous cells to radiotherapy, so its role is as an adjuvant]
- Treatment of premalignant (*CIN) → simple with 100% cure rate.

Stage Ia → surgery (total simple hysterectomy) for Ia 1

Stage Ib and IIa → radical hysterectomy + pelvic and para-aortic lymphadenectomy

Stage IIb and beyond → chemoradiation (no place for surgery)

Note: post-op radiotherapy decreases risk of local recurrence in patients with high risk factors.

- **Follow-up**

1. History, physical examination, pap every 3 months (for the first 2 years) then annually CT and CXR.
2. Vaginal vault is an important area to check in the follow-up. It's one of the first areas to be involved in metastasis and early recurrence.

High risk patients for recurrence:

- +ve lymph nodes
- +ve margins
- Residual parametrial disease

***CIN: cervical intraepithelial neoplasia**

- **Complications from surgery**
  1. Urinary dysfunction (retention) is the most frequent complication of radical hysterectomy as a result of partial denervation of the detrusor muscle.
  2. Shortening of the vagina.
  3. Uterovaginal fistula.
  4. Hemorrhage.
  5. Bowel obstruction.
- **Prognosis**
  - The earlier the intervention, the better.
  - If CA is removed surgically (with 1cm safety margin) then it shouldn't come back.
    But if it recurs that means that a cancer cell has already spread by the time the CA was removed, but wasn't detected before.
  - 85% of recurrence happens within the first 2 years.
  - The 5-year survival:
    Stage I 90%
    Stage II 73%
    Stage III 50%
    Stage IV 25%
  - The most common cause of death in cervical CA is renal failure.

Notes:

1. When we stage a patient with cervical CA then treat her, if it recurs we do NOT change the stage whatever happens.
2. Smoking is a risk factor.
3. Post-menopausal bleeding is never normal.
4. Abnormal pap smear → action must be taken.
5. Never rule out CA if normal pap smear.
6. Pap smear is NOT a diagnostic tool.
7. Colposcopy is NOT diagnostic.
8. The only diagnostic tool is biopsy.
9. It's important post-op to (to avoid urinary retention):
   - Catheterise for 5 days
   - Gradually train bladder after removal of cath.

Endometrial Cancer

- It's the most common gyne CA worldwide.
- Mean age is 63 years
- It's a disease of post-menopause
- More common in developed countries (least common in India)

GYNE CA worldwide		Jordan
# 1	Endometrial	Endometrial
# 2	Cervical	Ovarian
# 3	Ovarian	Cervical
# 4	Vulvar	Vulvar

- **Pathophysiology**
  Unopposed oestrogen (excessive hyperstimulation of the endometrium without the stabilising effect of progesterone)
- **Subtypes**
  Type 1 (80%): endometrial type
  oestrogen-related
  5-year survival 85%

  Type 2 (20%): non-oestrogen related
  5-year survival 58%

One of the golden rules of endometrial CA is its association with ovarian micrometastasis, and if you leave them 30% will develop ovarian CA within 5 years.

- **Risk factors**
  1. Obesity (BMI >30)
  2. Hypertension
  3. DM
  4. Nulliparity
  5. Late menopause
  6. PCOS
  7. Hormone replacement therapy (long term) and Tamoxifen

Smoking is protective against endometrial CA (unlike cervical CA) because nicotine blocks oestrogen receptors, but it decreases the age of menopause.

Never use pap smear for screening or diagnosis of endometrial CA.

- **Symptoms**
  1. Post-menopausal bleeding (PMB)
  2. Or any abnormal vaginal bleeding, regardless of the cause.
- **Screening**
  There is no ideal method for screening, although it's the only CA in the body that you can be sure of its diagnosis 100%

Golden rule: the best CA in any organ is one of the same type of tissue of that organ.

Example: best CA in ovaries is the serous and the worst is endometrial, while the best CA in endometrium is endometrial and the worst is serous.

- **Diagnosis**

  In any patient with PMB → first step is <u>TV U/S</u>

  If endometrial thickness is:

  1. <5mm → benign case (must be evaluated after 5 months)
  2. 5-8mm (cut-off value) → take other risk factors into consideration

     ↓ Next step

     Hysteroscopy + dilation and curettage (check ostea and endometrium) to detect abnormality and take antibiotics.

  3. >8mm → hysteroscopy + dilation and curettage

- **Index for biopsy**
  - Endometrial thickness >= 9mm
  - High Ca 125
  - Female with history of anovulatory cycles (history of pcos)

During hysteroscopy, visualising the ostea is very important because 50% of CA begins there, and you must biopsy from that area as well.

<u>Notes</u>:

1. In any PMB female, consider it malignancy
2. until proven otherwise. Although the most common
3. cause of PMB is atrophic vaginitis.
4. Fractional dilation and curettage is very important
5. to be done (to differentiate whether this lady has
6. primary cervical vs endometrial CA)
7. Ca 125 is high in 50% of cases but it's not specific.

Causes of PMB:

1. Atrophy 30%
2. Oestrogen therapy 30%
3. Endometrial CA 15%
4. Polyps 10%
5. Hyperplasia 5%
6. Miscellaneous

- **Spread**
  - Direct to cervix, tubes, serosa, and ovaries
  - Transtubal
  - Lymphatic
  - Haematogenous

Hysteroscopy and biopsy:

We visualise the entire cavity of osleum then → we take biopsies from abnormal areas. But if we can't visualise abnormality, we take 5 biopsies (ant, post, 2 lat, and from the fundus)

Pre of investigations:

- Routine CBC, electrolytes
- LFT, glucose
- CT of pelvis/chest/abdomen
- <u>Histopathology</u> (v. imp)
- Others: MRI, PET, CA 125

Staging (Surgical Staging)

Stage I: confined to the uterus

Ia: only uterus, only endometrium
Ib: only uterus, less than half of myometrium invaded
Ic: only uterus, more than half of myometrium invaded

Stage II: uterus and cervix

Stage III: adjacent to the uterus

IIIa: invading serosa/adnexa
IIIb: vaginal or parametrial invasion
IIIc1: pelvic lymph nodes
IIIc2: para-aortic lymph node

**positive inguinal lymph nodes is stage IIIc

Stage IV: metastasis far away from the uterus

IVa: invasion of bladder mucosa and/or bowel
IVb: distant metastasis (e.g. abdominal metastasis)

*Primary staging is final; if we diagnose a lady with stage I and we do resection, after a while she comes back with liver metastasis and histopathology shows that it's the same type of resected tumor it is still stage I + liver metastasis

Treatment

First line of therapy is surgery. All stages include total abdominal hysterectomy except in the case of distant metastasis, and removal of ovaries is mandatory, due to risk of micrometastasis and 30% will develop ovarian cancer within 5 years

Stage I: total abdominal hysterectomy + bilateral salpingo-oopherectomy ± adjuvant radiotherapy

Stage II: modified radical total abdominal hysterectomy + bilateral salpingo-oopherectomy + pelvic laparoscopy + surgical staging

Stage III: radical total abdominal hysterectomy + bilateral salpingo-oopherectomy + pelvic laparoscopy + surgical staging

Stage IV: radiotherapy ± palliative hysterectomy or pelvic exenteration

- Anterior exenteration: removal of vagina + bladder + implanting the uterus in the bowel
- Posterior exenteration: removal of bladder + rectum + diversion of bowel to the outside (colostomy)
- Total exenteration: this is a 12-hour procedure, we remove everything in the pelvis

*Radiotherapy is second line
*Chemotherapy is second line therapy only in papillary serous uterine cancer (PSUC)

Surgical Staging:

1. Omental biopsy
2. Regional lymph node biopsy
3. Biopsy from the peritoneal cavity
4. Cytology (by abdominal washing)

Candidates for surgical staging are:

- Patients with grade 3 lesion
- Grade 2 lesions more than 2 cm
- Type 2 endometrial cancer (non-estrogen related)
- More than 50% myometrial invasion
- Cervical extension

Abdominal Washing:

If positive cytology with risk factors: take it into consideration

If positive cytology without risk factors: you should be careful in the management (because it might be due to the surgeon himself by washing the uterus before, transference of cells to fallopian tubes to the abdomen, false positive cytology)

Follow Up

- CA125, CBC every visit
- CT scan (abdomen and pelvis) every visit
- Chest X-ray
- Vault smear (as the vault is the most common site for early recurrence

- Every 3 months for the first year
- Every 4 months for the second year
- Every 6 month for until the 5th year

Prognosis

Prognostic Factors: the most important prognostic factor is the stage

- Age (the younger the better)
- Histologic type (endometrial type has a good prognosis, and papillary has a bad prognosis)
- Level of differentiation (the higher the better)
- Nuclear grade (the lower the better)
- Presence of lymphovascular invasion (bad prognosis)
- Tumor size (>4 cm has a bad prognosis)
- Hormone receptor status (if positive then it has a better prognosis because e can use hormonal therapy)

Prognosis overall is 75%
Stage I 85%
Stage II 65%
Stage III 45%
Stage IV 15%

Papillary Serous Uterine Cancer (PSUC): it is the worst type of endometrial cancer, because it has an unpredictable behavior, everything can be negative, and patient can still have distant metastasis

Chemotherapy is only second line of therapy in papillary serous uterine cancer, it works as an adjuvant (cisplatin increases sensitivity of malignant cells to radiotherapy)

Tamoxifen is estrogen receptor modulator, used for prevention or treatment of breast cancer but it increases the risk of endometrial cancer (its mechanism of action: E2 antagonist effect on breast, and E2 agonist effect on endometrium)

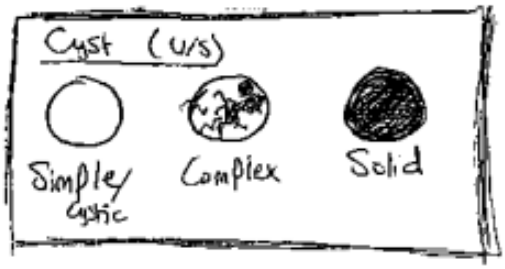
Benign Ovarian Cysts

Benign ovarian cysts are common, they are mostly asymptomatic and are discovered incidentally and many resolve spontaneously.

90% of ovarian cysts are benign (but varies with age) i.e. in post-menopausal women 5% are malignant

Aim of management is to rule out malignancy, and to avoid cyst complications (such as rupture, torsion, and hemorrhage

Most of them are cystic



The most common cause f adnexial mass in reproductive age is pregnancy

Benign Ovarian Tumors

- Physiological cysts (functional cysts)
  - Follicular cysts
  - Luteal cysts
- Benign germ cell tumors (teratoma)
  - Cystic teratoma
  - Mature solid teratoma
- Benign epithelial tumors
  - Serous cystadenoma
  - Mucinous cystadenoma
  - Brenner tumor
  - Clear cell tumor
  - Endometrial cystadenoma
- benign sex cord stromal tumors
  - Granulosa cell tumor
  - Theca cell tumor
  - Fibroma
  - Se
  - Sertoli-Leydig cell tumor

Differential diagnosis for a pelvic mass

1. Full bladder
2. Gravid uterus
3. Fibroid
4. Ovarian cyst
5. Ectopic pregnancy
6. Appendicitis
7. Pelvic inflammatory disease
8. Malignancy

How to differentiate between benign and malignant adnexial masses

1. By physical examination

	Benign	Malignant
Mobility	Mobile	Fixed
Consistency	Soft	Firm
Tumor surface	Smooth	Irregular
Bilateral vs unilateral	Unilateral	Bilateral

2. By ultrasound

	Benign	Malignant
Size	Usually <8cm	Usually >8cm
Consistency	Cystic	Solid/complex
Solid components	Not present	Nodular/papillary
Septations	Not present/singular	Multilocular, thick (>2mm)
Doppler flow	Not present	Present in solid component
Velocity of doppler	-	High velocity
Bilateral vs unilateral	Unilateral	bilateral
Associated features	calcification	Ascites, peritoneal mass, lymphadenopathy

Physiological cysts (functional)

Etiology: from normal physiological events but exaggerated due to inappropriate maturation

Need no treatment unless complication occur, or cyst persists (oral contraceptive pills may be used)

They are simple cysts and are usually <5cm

1. Follicular cyst
    - More common than luteal cysts
    - Result from unruptured follicle
    - Can persist for several menstrual cycles and reach 10cm
    - Usually resolves after 2-4 months
2. Luteal cysts

- Thin-walled, fluid- filled,
- Without septations or calcifications

Benign germ cell tumors (teratoma)

- The most common cyst in reproductive age
- Only 2-3% malignant
- Arise from 3 germ layers, so they can have elements of 3 layers

Types: benign cystic teratoma, and mature solid teratoma

1. Benign cystic teratoma
    - The cyst has epithelial all (skin appendages), teeth, hair, and nervous tissue (ectoderm)
    - Thyroid, bronchus, intestine (endoderm)
    - Bone, cartilage, muscle (mesoderm)
    - 60% are asymptomatic
    - Complex cyst
    - More common than mature teratoma
2. Mature solid teratoma
    - Rare
    - Must be differentiated from immature teratoma which are malignant

Benign epithelial tumors

- Arise from ovarian surface epithelium
- Most likely in females over 40 years

Types: serous cystadenoma, mucinous adenoma, endometrial cystadenoma, Brenner tumor, and clear cell tumor

1. Serous cystadenoma
    - Small, unilocular, fluid is thin and serous
    - Concentric calcific bodies (psammoma bodies)
2. Mucinous cystadenoma
    - Large, multilocular, fluid is thick and mucinous
3. Endometrial cystadenoma
    - Difficult to differentiate from ovarian endometriosis

    - Associated with pelvic pain and dyspareunia due to adhesions
4. Benner tumor
    - Small
    - May secrete estrogen (causes bleeding)
5. Clear cell tumor (mesonephroid)
    - Arise from serosal cells
    - Very benign

Benign sex cord stroma tumors

-Occurs at any age.

-May secrete hormones and present with abnormal bleeding.

- **Types:**

1-Granulosa cell tumors:

-malignant tumors but included here because they are:

Locally malignant but have good prognosis.

-Grows very slowly.

-Recurrence is common.

-Solid tumor.

-Secretes estrogen and inhibin, they predispose to endometrial Cancer.

2-Theca cell tumor:

-Benign, solid and unilateral.

-Mostly in postmenopausal females.

-Secretes estrogen→bleeding.

3-Fibroma:

-Rare tumor that occurs in elderly.

-Hard, mobile and lobulated with glistening surface.

-Causes ascites of pleural effusion (Meigs syndrome).

4-Sertoli-leidig cell tumor:

-Low grade malignant tumors.

-Found around the age of 30.

-Very rare.

-Small and unilateral.

-May produce androgens and signs of virilization.

-Some secrete estrogen.

- **Approach:**

1-History:

-Mostly asymptomatic.

-Ovarian cyst symptoms: pain, menstrual disturbances and symptoms of torsion.

-Rule out symptoms of malignancy: abdominal destination, abdominal pain, bloating, loss of appetite and Post-menopausal bleeding.

-Rule out symptoms of pressure: urinary frequency and constipation.

2-Physical exam: (bimanual)

-Abdominal mass, tenderness, bilaterality and consistency.

3-Beta HCG:

-to rule out pregnancy.

4-Imaging:

-U/S (the most valuable tool for initial evaluation).

-MRI.

5-Tumor markers:

-Not diagnostic but can help to differentiate an ovarian mass.

-CA125: increases in 80% of patients with ovarian CA, Endometriosis, pelvic infections, Fibroids, diverticulitis, IBD and hepatic dysfunction.

-Ovarian Germ-cell tumor:

AFP: yolk sac tumor.
LDH: dysgerminoma.
Inhibin: granulosa cell tumor.

hCG: non gestational ovarian CA.

-If suspected METS:

CEA: suspected colorectal primary.
CA19-9: suspected colorectal or pancreatitis.

Risk of Malignancy Index (RMI):

- Menopausal status:

-Pre-menopausal→1 mark
-Post-menopausal→2 marks

- U/S score:

-Unilateral→1 mark
-Bilateral→2 marks
-multilocular→3 marks

- Serum level of CA125

Multiply points together:
If >40 possible CA
If >200 it's CA

- **Management (Reproductive age):**

1-No further Actions:

-If simple cyst less than 5cm.

2-Conservative (indications):

-Simple cyst ≤7cm without features of malignancy and normal CA125(Risk of CA <1%).

-Asymptomatic.

-Risk of surgery>benefit of cyst removal.

-Or patient preference.

+ Follow up: (simple cyst 5-7)

Repeat U/S and CA125 in 3 months then:

If resolved→ no further action.
If remained unchanged→ repeat U/S annually.
If increase in size/complexity→ surgery (laparoscopic).

3-Surgical: (laparoscopic unless Contraindicated).

-Complex (solid/multiloculated/ bilateral) with RMI less than 200.

-Simple cyst >7cm.

-Symptomatic.

-Suspicion of malignancy.

-[cystectomy vs oophorectomy (fertility consideration)].

- **Management (Postmenopausal female):**

-Risk of CA is high (35%).

1-No further actions:

-If simple cyst <1cm.

2-Conservative:

-Simple cyst <5cm without features of CA and normal CA125(do Follow up).

-Asymptomatic.

-Risk of surgery > benefit.

-Patients preference.

+Follow up

Repeat U/S and CA125 every 4 months for 1 year.

If resolved→ no further actions.
If unchanged after 1 year→ no further action.
If increased in size/complexity→ surgery.

3-Surgical: (bilateral salpingo-oophorectomy +/- hysterectomy)

-Complex cyst with RMI<200.

-Simple cyst >5cm.

-Simple cyst with RMI >200.

**Aspiration of ovarian cyst:

- Should not be used anymore because:
  1-Non-neoplastic cells will recur.
  2-Malignant cysts will be upstaged.
- Image-guided aspiration is considered if:
  1-surgery is contraindicated.
  2-symptomatic.

- **Abdominal mass (generally):**

*Indications of surgery:

-Cystic mass > 5cm without regression after 6-8 weeks.

-Any solid mass.

-Any mass >10 cm.

-Any palpable abnormal mass (pre-/postmenopausal).

-Any mass with papillary vegetations on cyst wall.

-Torsion/ rupture suspected.

Ovarian CA

-The worst gynecological CA (the mcc of death in all gynecological CA).

-Serious but underrecognized.

-Early detection is difficult (asymptomatic), diagnosis is almost always at and advanced stage→ poor survival rate (75% of cases diagnosed in stage 3 when there are abdominal Mets).

*Ovarian CA refers to both ovarian and tubal CA because they share:
1-Genetic origin.
2-Epithelial lining.
3-Risk factors and etiological factors.
4-Hormonal affection.
5-Presentation.
6-Exogenous stimulation.

*Any adnexal mass in post-menopausal female is malignancy until proven otherwise.

*Fertility drugs and OCPs don't increase the risk of Ovarian CA.

- **Epidemiology**:

-5% of all female CA.

-5th mc ca (after breast, lung, colon and uterus).

-2nd most common gynecological CA in jordan, but

-3rd most common gynecological CA worldwide.

-4th most common cause of death from malignancy in females.

-Most common cause of death from gynecological CA.

-Mean age is 60 years.

- **Risk factors:**

-Increased age.

-Late age of 1st pregnancy (imp).

-BRCA1 gene and positive Family history.

-Increased life time of ovulation (early menarche and late menopause).

-Past hx of CA (lung CA, Colon and Endometrial).

-Talc, sanitary pads with talc.

-Estrogen replacement therapy.

- **Protective Factors:**

-Decreased life time of ovulation (decreased risk of monthly trauma).

(OCPs/ Chronic anovulation such as PCOS/ tubal ligation)

-Breast feeding, short reproductive life-time.

- **Classifications: (different ways!)**

1-Primary and secondary tumors (from stomach, breast and bowl).

Krukenberg: Mets to the ovary from Stomach CA.

2-Type of cell origin:

-Epithelial (80%): in post-menopausal.

serous (most common)/ mucinous/ endometrial/clear cell ca (worst Prognosis).

-Germ cell (15%): in young female.

dysgerminoma/ endodermal sinus/ immature teratoma/ choriocarcinoma.

-Stromal (5%): all ages.

granuloma cell tumor/ Sertoli-leydig cell tumor.

Metastatic: bilateral.

- **The most important classification**:

1-Low grade serous (Type 1 tumors):

-Arise from ovarian epithelium of ovarian inclusions.

-Relatively slow.

-Multistep pathway to reach frank malignancy.

-Good prognosis.

2-High grade serous (Type 2 tumors)

-Most common high grade serous.

-Develops rapidly.

-Advanced age at presentation.

-Poorly differentiated.

-Bad prognosis.

- **Symptoms:**

-First symptoms to appear are usually GI symptoms!

1-Increased abdominal size.

2-Bloating.

3-Difficulty of eating or feeling of fullness.

4-Vague non-specific pelvic or upper abdominal pain.

5-Menstrual symptoms.

6-Urinary frequency or urgency.

Physical Exam

Most important signs include the presence of an upper abdominal/pelvic mass and ascites.

Screening

Screening is **only** done in high risk patients. Screening tests include assessing the level of CA-125 tumor marker and performing a TV ultrasound.

Diagnosis

1. Transvaginal ultrasound (the most important diagnostic tool). Check ovarian blood supply, appearance, septations and borders.
2. Diagnosis requires an exploratory laparotomy. There is no role for laparoscopy in diagnosis or treatment as it may lead to rupture and dissemination of the tumor. In the case of the presence of a ruptured ovarian mass, it is important to know whether the cause is spontaneous or iatrogenic as it affects staging. (Tumor stage is upgraded if rupture was iatrogenic.)

Work up

- CBC, KFT and LFT (assess fitness for surgery and the presence of any metastases)
- CXR
- Tumor markers (for follow-up, NOT for initial diagnosis)
- Bone scan (bone mets)
- **Risk of malignancy index (RMI)**

Menopausal state	Not menopausal	1 point
	Post-menopausal	2 points
Ultrasound score	Unilateral	1 point
	Bilateral	2 points
	Multilocular	3 points
Serum level of CA-125		
MULTIPLY THE NUMBERS BY EACH OTHER Example: A post-menopausal female presents with a unilateral mass with serum CA 125 = 17 U/mL. Calculation: **2 x 1 x 17 = 34**. Score of >200 is *malignancy*. If score is >= 40 then *possible malignancy.*		

Spread

- Most important route of spread is **transcoelomic spread**, i.e. the exfoliation of cancer cells into the peritoneum.
- Lymphatic spread.
- Hematogenous spread.

Staging

Staging is surgical, NOT clinical.

Cytology

- If ascites is already present, take a fluid sample for cytology.
- If there is no ascites, inject 500 mL of normal saline into the peritoneal cavity "washing", shake the abdomen and then take a fluid sample.

Stage I	Limited to the ovaries, one or both.
Stage Ia	One ovary; intact capsule; -ve cytology.
Stage Ib	Two ovaries; intact capsules; -ve cytology.
Stage Ic	One or both ovaries; ruptured capsules; +ve cytology.
Stage II	Extension to the tubes, uterus or pelvic organs.
Stage III	Abdominal area outside pelvis. 75% of cases present at this stage.
Stage IIIc	Metastasis to retroperitoneum or inguinal lymph nodes.
Stage IV	Distant metastases (outside the peritoneal cavity). **+ve cytology** always.

Treatment

Treatment is always surgical (for both, early and late stage disease).

- Consider the extent of the disease:
  ➔ Patient's presenting symptoms
  ➔ Patient's wishes regarding parity (fertility preservation can only be done in **stage I**).
  ➔ Patient's fitness in relation with treatment modality.

1. **Surgical treatment**

Early stage disease	Total abdominal hysterectomy + bilateral salpingo-oophorectomy + LAP + para-aortic lymphadenectomy + infracolic lymph nodes
	+/- chemotherapy
	Take a sample for cytology and scrubs from hemidiaphragm.
Advanced stage disease	+ debulking surgery and cytorduction

Radiotherapy is **not** part of the routine management of ovarian cancer.

2. **Cytoreduction**
   - Optimal vs sub-optimal
   - It depends on whether all metastatic nodules are resected or not. Not all metastatic lesions can be resected as there might be pinpoint nodules on the mesentery or the bowel.
   - If the patient is not fit for surgery, she is given neoadjuvant chemotherapy before surgical intervention is made.
   - **Radical debulking** (optimal cytoreduction)
     - Infracolic gutter
     - Paracolic gutter
     - Diaphragm
     - Liver and any other sites of metastatic nodules

Prognosis

- Ovarian cancer carries poor prognosis as 75% of the cases are detected relatively late (stage III).
- The most common cause of death is bowel obstruction.

5-year-survival rates	
Stage I	85%
Stage II	70-80%
Stage III	40%
Stage IV	20%

- **Prognostic factors:**
  - Pathologic factors → stage and grade (histology). **Clear cell carcinoma has the worst prognosis.**
  - Biologic factors → Patients with diploid tumor have a better prognosis.
  - Clinical judgement → extent of residual disease, volume of ascitic fluid, patient's age and performance state.

Low-risk patients	High-risk patients
• Low grade tumor • Not clear cell type tumor • Diploid tumor • Intact capsule • No surface excrescences • No ascites • -ve washing (cytology) • Unruptured (neither spontaneously nor intraoperatively) • No dense adherence	• High grade tumor • Clear cell type tumor • No intact capsule • Surface excrescences • Ascites • +ve washing (cytology) • Ruptured • Dense adherence

Vulvar Neoplasia

Source: Kaplan 223

Benign vulvar diseases include vulvar dystrophy, vulvar dysplasia and carcinoma in situ (CIS).

Vulvar dystrophy

Benign, chronic vulvar lesions without malignant potential.

1. Squamous hyperplasia → thickened keratin and epithelial proliferation. Management involves the use of fluorinated corticosteroid cream.

2. Lichen sclerosus → epithelial thinning. Findings on physical exam include bluish-white papules that can coalesce to form white plaques. Management involves the use of Clobetasol cream (a high potency steroid).

Benign vulvar lesions

1. **Molluscum Contagiosum**
   - Common, benign, viral skin infection.
   - Common in children, sexually-active adults and in immunosuppressed patients.
   - Etiology: Molluscipox virus which forms spontaneously regressing and umbilicated tumors of the skin, rather than pox-like vesicular lesions.
   - Transmission occurs through direct skin contact.
   - Management involves observation, curettage or cryotherapy.

2. **Condyloma Accuminatum** (in STD summary)
   - Benign, cauliflower vulvar lesions.
   - Etiology: HPV (6 and 11)
   - Management involves ONLY treating clinical lesions.

3. **Bartholin Cysts**
   - Bartholin glands are paravaginal glands that are NOT visible usually.
   - Duct obstruction may be secondary to infection (GC). Persistent duct obstruction following successful host defense (no more infection) will

lead to cystic dilation of the duct. In this case, cyst aspiration yields **sterile fluid** as it is not infected anymore.

- Management is conservative unless the cyst results in pressure symptoms due to size, then it is incised and drained.

Premalignant vulvar lesions

Benign lesions with malignant potential.

Most common presenting symptom is *itching*. However, most lesions are asymptomatic.

1. **Squamous dysplasia**
    - Appearance: white, red or pigmented.
    - Lesions are often multifocal.
    - Histology:
        - Cellular atypia restricted to epithelium without breaking the basement membrane.
        - Involving partial thickness.
    - Management involves surgical excision of the lesions.

2. **Carcinoma in situ**
    - Appearance: same as squamous dysplasia.
    - Histology:
        - Cellular atypia restricted to epithelium without breaking the basement membrane.
        - Involving full thickness.
    - Management involves laser vaporization.

Malignant vulvar lesions

- The least common gynaecological cancer. (4% of malignancies of the female genital tract.)
- Seen in very odd age groups >65 years
- Symptoms: Painless or long term pruritus

1. Squamous cell carcinoma:
   - Most common (90%)
   - Patients are mostly diagnosed at stage 1

2. Melanoma:
   - Second most common cause (5%)
   - The most important prognostic factor in this type is the DEPTH of invasion
   -Appearance: Dark/Black lesions

3. Paget Disease:
   - Uncommon
   - Appearance: Red lesions
   - 20% risk of BM invasion
   - Associated with: GI, GU and breast cancer

Diagnosis:
- Biopsy: for any vulvar lesions to rule out caner
-Always consider pre-invasive or invasive vulvar cancer if it is a pruritic lesion

Staging: Surgical staging

stage 0: Carcinoma in situ (Basement membrane intact)

stage 1: confined to vulva, < 2cm, No lymph nodes palpable
- 1a: < 1mm in depth
- 1b: > 1mm in depth

stage 2: confine to vulva, > 2cm , No lymph nodes

stage 3: any size with spread to lower urethra or vagina or anus

stage 4: widespread metastasis
- 4a: upper urethra, bladder, rectum, bilateral lymph nodes, pelvis
- 4b: distant metastasis

Management:

Radical Vulvectomy (not common)	Removal of the entire Vulva (soft fatty tissue/ labia minora and majora/ perineal skin/ clitoris)	Sexual Dysfunction
Modified radical vulvectomy	Wide local excision (if unilateral and doesn't cross midline)	Less sexual Dysfunction
Lymphadenectomy	Inguinal node dissection	Lower edema

For bilateral Lesions (Squamous cell carcinoma); radical vulvectomy is done, but we do frozen section of lymph nodes to determine the surgery.

Post Coital Bleeding

- Introduce yourself
- Patients Profile: Age, married (since?) …
- Chief complaint with duration
- History of presenting illness

-Bleeding details:

1. Onset and duration
2. Amount
3. Color
4. Only after sexual intercourse?
5. First time?

-Associated symptoms:

1. Dyspareunia
2. Discharge (amount, color, smell)
3. Urinary symptoms (dysuria)
4. Weight loss?

-Menstrual history:

1. Last menstrual period
2. Menses: menarche, regularity, frequency and duration
3. Contraception
4. Last pap smear
5. Menopause symptoms? HRT?

-Obst. history:

- Past pregnancy: Delivery, weight, complications
- Any ectopic pregnancies or stillbirth

-Past medical history:

- DM, HTN
- STD, PID, IUCD

-Past social history:

-smoking and alcohol

- Past drug history and allergies

- Family history:

- Cancer or similar problem

Then you tell the patient that she needs:

1. Vaginal examination and swab
2. Urine sample
3. Pregnancy test (possible ectopic pregnancy)

DDx:

1. Infection
2. Cancer
3. Atrophic vaginitis
4. HRT
5. Abuse
6. Others

Postmenopausal Bleeding: (PMB)

Bleeding after the 1yr of amenorrhea

Causes:

1. Atrophic Vaginitis: 30%
2. Exogenous estrogen: 30%
3. Endometrial Cancer: 15%
4. Polyps: 10%
5. Hyperplasia: 5%
6. Miscellaneous < 10%
    - Lacerations
    - Non-gyne causes
    - Bleeding tendency (disorder)

History: OSCE

- Patients Profile:
    - Age
    - Marital status/ LMP/ GP (menopause since when?)
    - Medical illnesses (HTN/ DM/ CHD/ Hepatic/ Renal/ Thyroid/ Bleeding disorders)

- History of presenting illness:

    -Bleeding characteristics:
        1. Onset and duration
        2. Continuous or intermittent
        3. Amount (spotting?) / no. of pads/ clots/ tissue
        4. Color (red or brown)
        5. Only after sexual intercourse?
        6. First time?
        7. Post-coital?
        8. History of trauma

- Associated with:
        1. Abdominal pain
        2. Fever
        3. Weight loss
        4. Dyspareunia
        5. Bleeding from other orifices

- Gyne history
    1. Last menstrual period
    2. Menses: menarche, regularity, frequency and duration
    3. Contraception
    4. Last pap smear
    5. Menopause symptoms? HRT?

-Past social history:
    -smoking and alcohol

- Past drug history and allergies
    HRT, Tamoxifen, Anti-coagulants, OCP's

- Family history:
    - Cancer (breast or gyne)
    - bleeding

Physical examination:

- General
- V/s
- Abdominal exam: masses and ascites
- Gyne exam:
    1. Inspection: for lacerations, lesions, discharge or foreign body
    2. Speculum:
        -inspect for lesions and erosions
        -Look for signs of atrophy (pale, dry epithelium, loss of rugae)
    3.bimanual exam: Assess size, contour, tenderness, adrenxial masses (after speculum due to pap smear)

Investigations:
(according to suspected causes)

1. Lab tests: CBC, TSH, PRL, FSH
    + tumor markers: (LDH, HcG, AFP, CEA, inhibin, Ca-125) if ovarian mass is present

2. ultrasound: both abdominal and transvaginal for masses and endometrial thickening
    - if > 5mm do Hysteroscopy with biopsy

Treatment:

-according to the cause
- If Atrophic vaginitis (once CA is ruled out)
    1. Vaginal estrogen (local): cream, pills, rings
    2. HRT

-If cancer: according to type
-If cervical polyps: surgical removal by hysteroscopic resection of D&C
-If endometrial hyperplasia:
    1. Progestin
    2. Hysterectomy

Dysfunctional Uterine Bleeding (DUB)

Definition:
Abnormal uterine bleeding NOT due to organic gynecological disease.

- **Incidence:**
- Any age
- Puberty
- Menopause

- **causes:**
  - primary
  - secondary (abnormal stimulation by pituitary)

Types:

1- Anovulatory 2- ovulatory

Anovulatory (extremes of age/ affect proliferative phase)

a- Pubertal DUB: primary fail in the pituitary gland which fails to secrete gonadotropins in cyclical sequence.

b- Premenopausal DUB: primary fault in the ovaries which fails to respond to gonadotropins.

Ovulatory (childbearing age DUB/ affects secretory phase)

A- WITHOUT corpus luteum abnormality

1- Oligomenorrhea -infrequent cycles-
Many then have PCOS

2- Polymenorrhea -frequent cycles-
Many progress to oligo or amenorrhea

B- With corpus luteum abnormality

1- Corpus luteum insufficiency: history of premenstrual spotting

2- Corpus luteum prolonged action: history of postmenstrual spotting

Other causes of DUB: -Thyroid – hematological disorders/anemia and IDA

Diagnosis:

- Rule out other causes
- You CANNOT diagnose DUB without taking biopsy (mandatory to rule out malignancy)

Treatment:

- Acute management:
- stabilize the patient (hydration/O2/cannula)
- IV 25mg estrogen/D&C
- Chronic management: (maintenance)
- Medical: 1- Anovulatory: menstrual regulation via OCP 21 days OR minipills (progesterone only pills)
  Progestin IUD (Merina)
  2-Ovulatory: NSAIDs
- Surgical: -if not responsive to medical treatment-
  D&C
  Endometrial ablation
  Hysterectomy (definitive)

Abnormal bleeding

Causes:

1-pregnancy: always rule out in reproductive age
- Diagnosis: causes of first trimester bleeding
- B-hcg and Ultrasound

2- Anatomical:
- Vaginal/cervical lacerations
- Uterine lesions:
(Atrophy/hyperplasia/PID/IUCD/fibroids/adhesions/trauma/surgery/polyps/ Adenomyosis)

3- Hormonal:
- Anovulation (PCOS/thyroid/increased prolactin)
- Dysfunctional uterine bleeding

4- Drugs: Anticoagulant/HRT/Tamoxifen/minipills (progestin only pills)

5- Non gynecological causes:
- GI: hemorrhoids/fissures/rectal bleeding
- UG: hematuria

- Bleeding between **regular** cycles: think of anatomical problems
- **Irregular** menses with unpredictable bleeding: think Anovulation/dysfunctional uterine bleeding

HISTORY

Patient profile, chief complaint with duration

HOPI:

- <u>bleeding details:</u>
  Amount/duration/onset/frequency
  Color/clots/tissue/number of pads
  Circumstances (postcoital?)
- <u>Associated symptoms:</u>
  - Anemia symptoms: pallor/SOB/LOC (when severe)
  - Bleeding from other orifices: (epistaxis/bruising/gums)
  - Symptoms of infection: fever/abdominal pain/discharge
  - Tumor Sx: wt loss/heaviness/malaise/anorexia
  - Hypothyroid Sx: weight gain/cold intolerance/loss of appetite
  - Anovulation: hirsutism/acne/baldness
  - GI/UT symptoms
- <u>Gyne Hx:</u>
  Previous period details (menarche/frequency/duration/amount)
  Infertility

Pap smear
OCPs/lactation/iucd

- Medical Hx:
  Dm/thyroid/caogulopathies/tumors/previous history of same complain
- Past surgical Hx: pelvic surgeries/ history of trauma
- Drug Hx:
  Anticoagulant/mini pills (progestin only pills)/HRT/Tamoxifen
- Family Hx: history of coagulopatheis/Tumors
- Social Hx: stress/smoking/diet (eating disorder)

PHYSICAL EXAMINATION:

- General: BMI/pallor/hirsutism/bruising/secondary sexual characteristics
- Vital signs
- Head and neck : thyroid
- Abdomen: tenderness/masses/uterine size
- Gynecological exam: pubic hair/ PV (bimanual)/ speculum (discharge/laceration/polyps/cervical motion tenderness -for ectopic pregnancy-)

INVESTIGATION:

- Blood type/Rh
- Labs: CBC/DIC profile/ TSH/prolactin/Estrogen/progesterone/B-hcg/kft/lft
- U/S, Pap smear, high vaginal swab, hysterosalpingiography, hysteroscopy, D&C with biopsy, laparoscopy

TREATMENT: according to the cause

Menopause & Hormone replacement therapy

Definition:
It's the permanent cessation of menstruation caused by failure of ovarian estrogen production in the presence of high FSH,LH (diagnosed after 6-12 months of amenorrhea)
Mean age of menopause: 51 years

It's usually preceded by perimenopausal period (climacteric) which is the transition optimal menstrual condition to menopause

Etiology: lack of estrogen due to lack of follicles.

RISK FACTORS:

- Genetics (family history)
- Smoking (reduced age of menopause 3 years)
- Chemotherapy and radiotherapy
- Drugs: steroids
- Others: fair,thin women/ sedentary life style/ decreased calcium intake
  -Not affected by the number of pregnancies or the use of OCP-

Premature menopause:

- Age < 40
- Usually idiopathic
- May be caused by radiotherapy chemo or oopherectomy

SYMPTOMS:

- Amenorrhea(2ry): the most common symptom
  Menses typically become anovulatory and decreases during a period of 3-5 years (perimenopausal) -> then amenorrhea
- Hot ashes (early):
  Sudden unpredictable episodes of skin flushing and sweating, lasts 30 secs to 5 mins (less in obese)
- Atrophy of lower urinary tract: (intermediate)
  Loss of urethral tone leading to -> urgency/frequency/dysuria/interstitial cystitis
- Vaginal changes: (intermediate)
  Shortening of the vagina/ atrophied vaginitis/ dryness leading to dysperunia and libido

Premature ovarian failure:

- Rare
- Age< 30
- Maybe associated with autoimmune disease or Y-chromosome mosaicism

- Osteoperosis: (late)
  Decreased bone density leading to fractures (trabecular)
  ***Sites:** 1- vertebral bodies (most common) : fractures, kyphosis, decreased height

***Diagnosis:** DEXA scan (to assess bone density)
24 hrs-urine hydroxyproline (to assess calcium loss)
***risk factor:** positive family history in a thin white female (most common)
Steroids/decreased calcium/sedentary life style/alcohol/smoking
***Treatment:**
1- life style modification (increased calcium and vitamin D/weight bearing exercise/stop alcohol and smoking)
2- Medical treatment:
1st line therapy: Bisphosphonate (inhibits osteoclasts)/ Raloxifine (SERM)
(increases bone density)
Estrogen therapy (should NOT be used as first line)

Indication for DEXA scan in patients:
- All female >= 65
- Younger post menopause with >= 1 Risk factor (other than female/white)
- Postmenopausal women with fractures

- Cardiovascular diseases: the most common cause of mortality (50%) in menopausal women
  - Increases LDL and risk of MI/CAD - decreased HDL
- Psychological and emotional changes (early)
  Fatigue/dizziness/irritability/anxiety/depression/mood changes

P/E:

Decreased breast size and change in texture
Vaginal,urethral and cervical atrophy

Diagnosis:

Hx/PEx/investigation (increased FSH level >40 normal)
Further investigations to rule out the causes of amenorrhea (Thyroid function test/prolactin/B-hcg)

Routine screening tests:
Lipid profile
Pap smear
Monogram
Occult stool blood
DEXA scan

Hormone Replacement Therapy (HRT)

• Estrogen Replacement Therapy (ERT)	• Estrogen+ Progesterone
Estrogen alone Only used post hysterectomy Risks: increase the incidence of Endometrial Cancer.	Progesterone (MPA) is added to protect the endometrium from constant stimulation (inhibit endometrial hyperplasia/cancer) and reduce the risk of Endometrial Cancer. Not indicated for pt. Without uterus

Indications for HRT

- Presence of hot flashes
- Prevention of atrophic vaginitis

Recommendations

- Short term therapy (<4-5 years) is acceptable for menopausal symptoms relief only
- Prescribe the lowest effective dose
- Doesn't seem to protect CVS disease (in fact it could make it worse)
- Should not be primarily used for osteoporosis, there are other drugs as effective as HRT

Risks

- Increase risk of Breast Cancer
- Increase incidence of Endometrial Cancer (ERT only)
- Thromboembolism/ MI/ Stroke
- Cholelithiasis/ Cholecystitis

Contraindications for HRT/ ERT

- Unexplained vaginal bleeding
- Breast Cancer
- Metastatic Endometrium/ Ovarian Cancer
- Liver disease
- History of DVT/ PE/ MI/ stroke
- Migraine/ HTN

Regimens

- **Continuous Regimen**

Estrogen + Progesterone everyday

Side effect: unpredictable breakthrough bleeding but eventually will lead to amenorrhea

- **Cyclic Regimen**

Estrogen + progesterone for 1-2 weeks/ month or Estrogen (day 1-25) + Progesterone (day 12-25)

Predictable bleeding will occur (withdrawal period) but will not lead to amenorrhea

Adnexal Mass

It is a mass of the ovary, fallopian tube or surrounding connective tissue.

Most common: ovarian.

Differential diagnosis (OSCE station)

- **Ovarian Mass (most common)**

Simple/ hemorrhagic physiologic cyst

Theca cell cyst

Endometrioma

Benign/ malignanr neoplasm

Metastasis

- **Fallopian Tube Mass**

Ectopic pregnancy

Hydrosalpinx

Tubo-ovarian abscess

Fallopian tube cancer

- **Others**

Fibroid

Diverticular abscess

IBD

Appendiceal abscess or tumor

Pelvic kidney

- **Adnexal masses related to pregnancy**

Ectopic pregnancy

Corpus luteum cyst

Theca luteal cyst

- **Differential diagnosis for acute pelvic abdominal pain**

Adnexal torsion

Ruptured/ haemorrhagic ovarian cyst

Acute PID/ tubo-ovarain abscess

- Prepubertal ovarian cyst are never functional because they don't have ovulation.

Amniocentesis

- **Definition**

removal of amniotic fluid by transabdominal aspiration for diagnostic and therapeutic causes.

It is the most common invasive prenatal procedure.

- **Indications**

Diagnostic

1. To detect genetic diseases and chromosol anomalies

Genetic diseases (cystic fibrosis, sickle cell disease, fragile x syndrome , muscular dystrophy)

2. Biochemical testing (αFP level)
3. Neonatal lung maturity

Phosphatidyl glycerol

L/S ratio >2

4. Bilirubin
5. Infection

(WBC, gram stain) for Chorioamnionitis

Therapeutic

1. Blood transfusion for the fetus
2. Drug administration
3. Amnioreduction (in case of severe polyhydramnios)

Usually it is done in females who have a significant risk of genetic diseases:

Advanced maternal age ≥35 years old.

Family history of certain birth defects

Abnormal U/S

Previous child with birth defect

Usually done at (15-18 weeks) of gestation

- **Complications**
- Increase risk of abortion (1%) in early amniocentesis
- Post procedure leakage
- Infection
- Preterm labor
- Injury to the baby or the mother
- Club foot <1%

Cordocentesis : Percutaneous Umbilical Blood Sampling (PUBS)

- **Indications**
- Fetal Hct in hemolytic anemia (now replaced by doppler U/S less invasive than PUBS)
- Rapid fetal karyotype evaluation