بسم الله الرحمن الرحيم

BY: LEEN ATTAR

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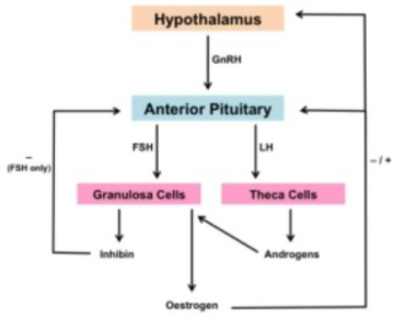
Puberty

Definition

The process of reproductive & sexual development & maturation that changes a child into an adult.



HPO axis

- The development of ant. pituitary starts between 4th & 5th weeks of gestation.
- The childhood period : low levels of gonadotropins.
- 6-8 years of age : first steriods to rise in blood are DHEA & DHEAS, shortly before FSH begins to increase.
- 8-9 years of age : Pulsatile secretion of gonadotropins.

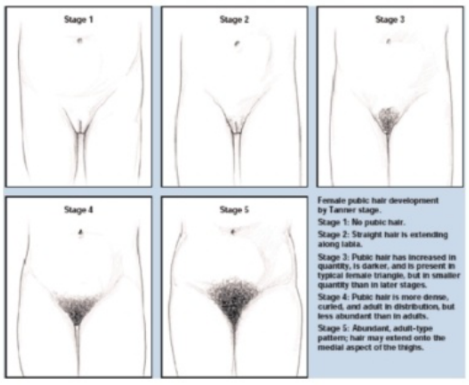
Physical changes of puberty (starting from the age of 8-9yrs)

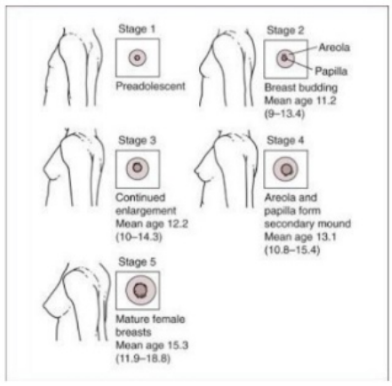
- Thelarche = Breast development
- Adrenarche = Pubic & axillary hair growth
- Growth spurt
- Menarche = Onset of menstruation

Variety of Puberty Onset

- Race → Blacks earlier
- Heredity
- Body weight → More weight earlier menarche
- Exercise

Tanner staging





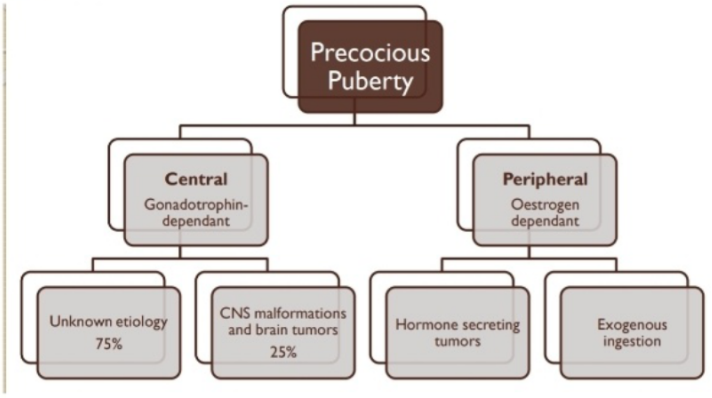
Menarche

- Mean age= 12.8 yr
- Initially cycles may be anovulatory, irregular & unpredictable.

Disorders

Precocious Puberty

- Onset of puberty : <8y in girl, <9y in a boy
- Types :

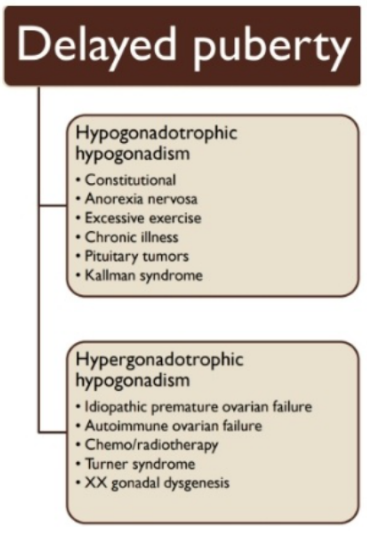


Investigations:
- Hormone profile (LH & FSH High in Central type, Low in Peripheral type)
- Hand & wrist X-ray (Bone age > chronological age ⇒ pathological cause of precocious puberty)
- Brain imaging + pelvic US + Tumor markers

Treatment
- Treat the underlying cause
- GnRH analogue therapy

Delayed Puberty

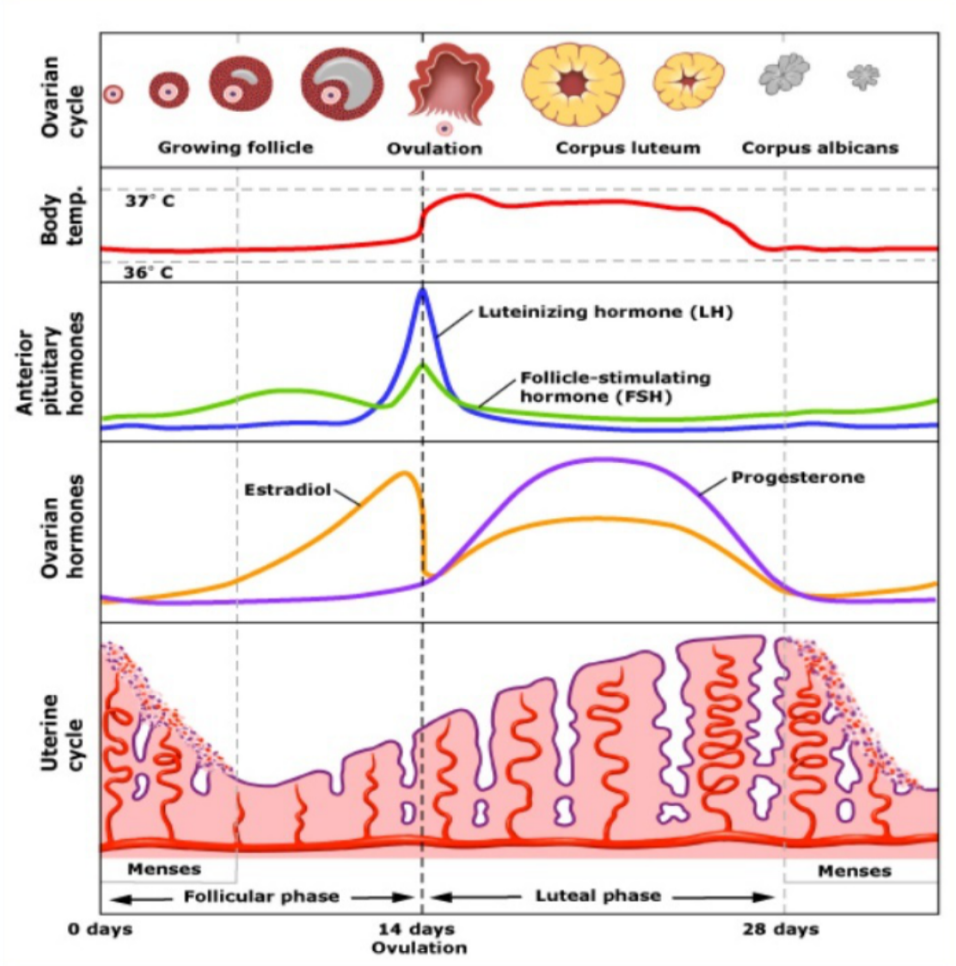
- No signs of secondary sexual characteristics by age 14y.
- Types :



Investigations
- FSH, LH
- Karyotyping
- Pelvic US
- X-ray to determine bone age (Bone age < Chronological age ⇒ pathological)

Treatment
- Treat underlying cause.
- watchful waiting
- Gonadal hormone replacement & Growth hormone therapy

Menstrual cycle



- The cyclical changes that occur in the female reproductive system.
- Normal menstrual cycle is a 28 Days (21-35 days).
- Average Menses= 4 days, more than 7 days is abnormal.
- Average amount is 30-50 ml without clots

》 Follicular phase: FSH causes E2 secretion.
》 Ovulation: LH surge cause oocyte to be released
- Many follicles are stimulated 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

Criteria

- Recurrence in 3 or more consecutive cycles
- Absent PRE- ovulatory
- Present only POST- ovulation (letual phase)
- Interfere with normal function both physical & behavioural
- Resolve with onset of menses

Symptoms

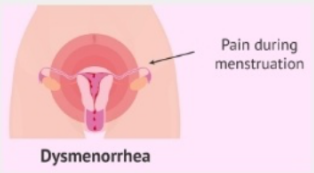
- Pain: dyspareunia / breast tenderness / headache / back & abdominal pain
- Bloating & wt. gain/ fluid retention & edema
- Emotional nervous / mood swings / anxiety & depression

Treatment

- Nutritional: balance diet / ⬇️ Caffeine & sugar & salt
- Medication: SSRI (fluoxetine) / NSAIDs/ OCPs

	Premenstrual syndrome (PMS)	Premenstrual dysphoric disorder (PMDD)
Definition	• The onset of severe discomfort or functional impairment prior to menstruation	• Severe affective symptoms and behavioral changes that cause clinically significant disturbance of daily life
Diagnostic criteria	• Present in the 5 days prior to the beginning of menstruation for at least 3 consecutive cycles • End within 4 days after the beginning of menstruation • Interfere with normal daily life activities	• Present up to 7 days prior to the onset of menstruation for the majority of cycles within one year • ≥ 5 symptoms that are marked and/or persistent (e.g., depressed mood, anxiety, anger, affective lability, sleep disturbances, change in appetite, pain, headache) • Significant interference in daily life (work, home, social activities, interpersonal relationships)

Lifestyle: Regular exercise



1 Primay / spasmodic

- Synonyms – Essential/ Intrinsic/ Functional
- Recurrent, crampy lower abdominal pain, may radiate to the back or inner thighs (Associated symptoms: nausea + vomiting + diarrhea + lower backache / headache) in the absence of pelvic pathology.
  - Sympathetic fibres: Uterus T10, T11, TI2, L1. Cervix S2, S3 and S4.
- **Between 17 and 22 years (Initial onset: within 2 years of menarche)**
- **Begins several hrs prior the onset of menses & continue 1-3 days.**
- Pathophysiology
  - ▪ Uterine contractions with ischemia & progesterone production (increase prostaglandins E2/F2)
  - ▪ Uterine abnormalities
  - ▪ Cervical obstruction + Imperforate hymen
- Risk factors — Early menarche / long & heavy flow / decrease parity/ heavy smoking / psychological / obesity (diet & exercise)
- **Management: NSAIDs (1st choice), OCPs (2nd choice), General measures (diet,Ex, palliative), Surgical.**

2 Secondary

- Synonyms - Extrinsic/ Congestive/ Organic
- Recurrent, dull aching lower abd pain WITHOUT nausea & vomiting & diarrhea, depends on the underlying cause, NOT limited to the menses.
- **Older women 30-40 years**
- Less related to the first day of the flow & onset few days prior to menses and continues throughout the cycle and even after cessation of menses.
- Associated symptoms → dyspareunia / infertility/ uterine bleeding
- On Examination
  - General: look for anemia.
  - Abdomen: look for a mass
  - Per vaginum: enlarged uterus or uterine masses
- Etiology
  - Uterine causes→ PID / IUD / Adenomyosis / Fibriods / Polyps
  - Extrauterine causes→ Endometriosis (MCC) / Adhesions / Ovarian cyst / Cervical stenosis
- **Partial or NO response to NSAIDs**

Hirsutism

Excess terminal, coarse hair growth in androgen-dependent areas of the body, including the chin, upper lip, chest, and back.

Causes

- Androgenic
  - Adrenal hyper-androgenism
    - Congenial Adrenal Hyperplasia (CAH)
    - Cushing syndrome
    - Androgen secreting tumors
  - Ovaries hyper-androgenism
    - PCOS
    - HyperAndrogenic Insulin Resistance & Acanthosis Nigricans (HAIR- AN) syndrome
    - Androgen secreting tumors, like sertoli- leydig cell tumor
- Medication related side effects
  - Corticosteroids
  - Exogenous androgens (ex. Danzol)
  - Immune modulating agents (ex. cyclosporin)
- Idiopathic
  - Cushing syndrome
  - Thyriod dysfunction
  - Hyper-prolactinemia
  - Acromegaly

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

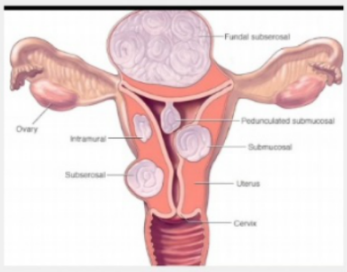
Investigations

Order for:
- Testosterone
- Prolactine
- TSH

- Normal Testosterone — Think about 1) 5α-Reductase OVERactivity 2) Idiopathic
- ↑ Testosterone — Do DHEA
  - ↑⇒ Adreal causes [CAH, Adrenal cancer] so do CT abdomen
  - Normal ⇒ Ovary causes [PCOS, ovarian cancer], so do LH, FSH, US, CT-pelvis

Management depending on the cause

- Idiopathic ⇒ Cosmetic (laser/ waxing), Spironolactone
- PCOS ⇒ Metformin/ OCP/ GnRH agonist
- Adrenal ⇒ Prednisolone / spironolactone
- Tumor or cushing ⇒ Surgical
- 5 alpha reductase ⇒ Fenasteride (5-alpha reductase inhibitor)



Fibroids (leiomyoma)

*** LOCAL PROLIFERATION OF SMOOTH MUSCLE CELLS OF THE UTERUS**

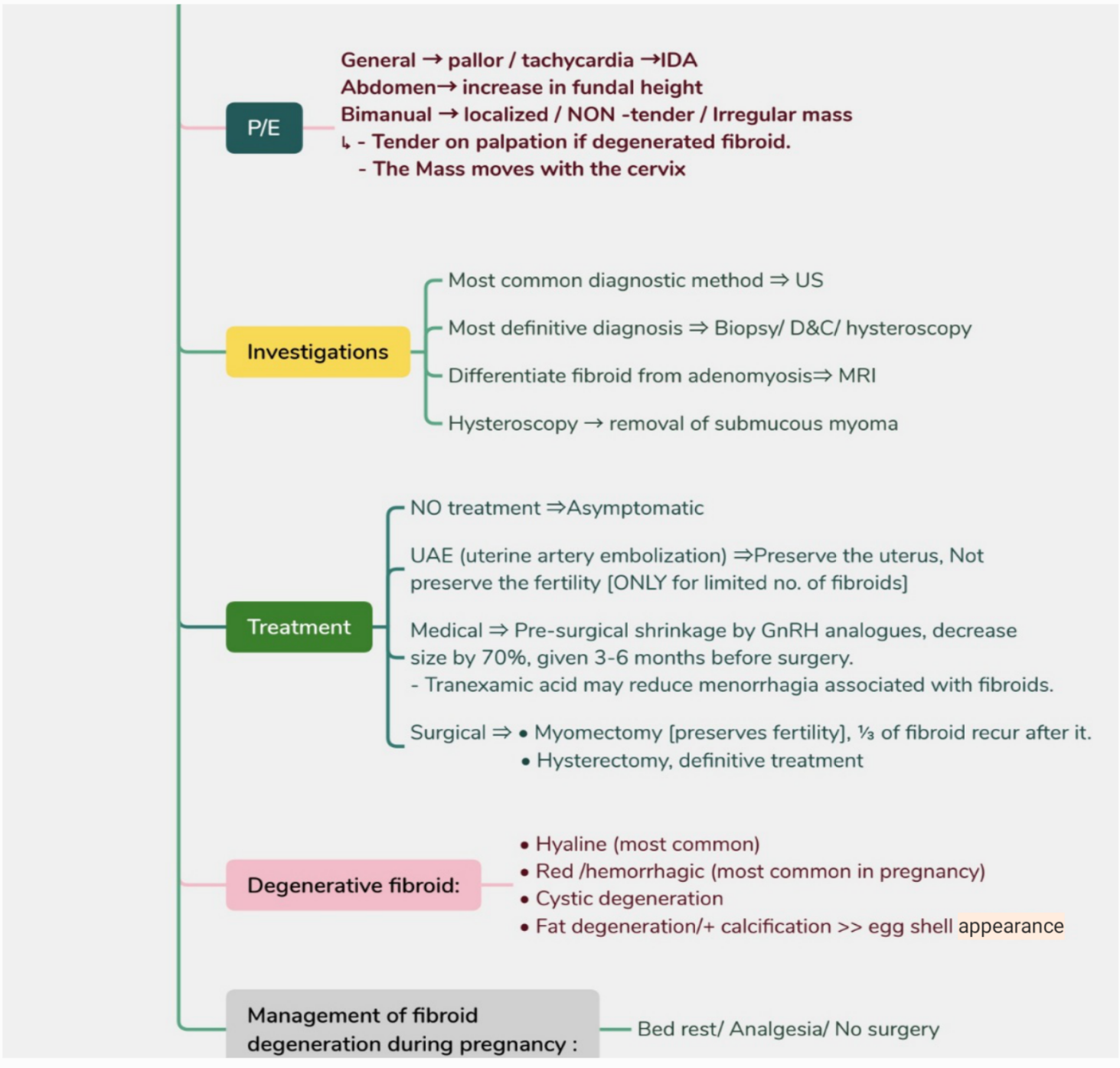
- Benign/ idiopathic
  - Malignant transformation rare = 0.1%
- Asymptomatic (Mostly) ~ 50%- 60%
- Multiple
- Increase with pregnancy and exogenous estrogen (Hormone responsive)
- Obstetric problems: Infertility, recurrent abortions, preterm labor, placenta previa, abruption, malpresentation
- Histology: psudocapsule of compressed smooth muscle cells, contain few blood vessels & lymphatics

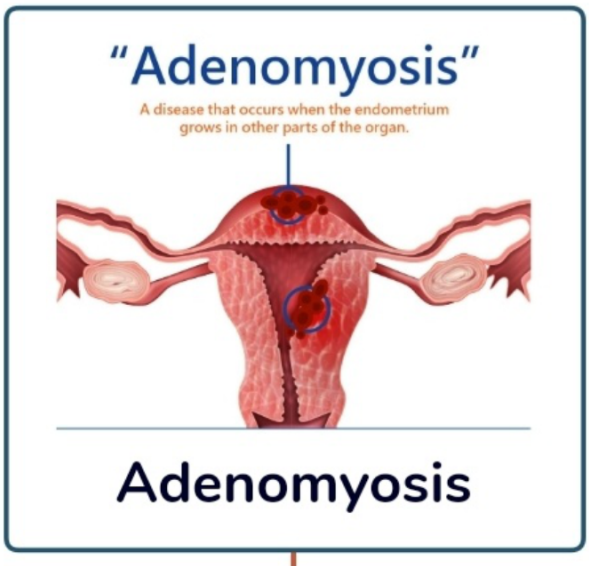
Classifications

- **Submucosal → Beneath endometrium / most common symptom : Bleeding (Menorrhagia / Metro menorrhagia). IF connected with a stalk ⇒ pedunculated submucous. (Fundal Pedunculated submucosa → Abortion & Infertility)**
- Intramural → Within muscular wall / Asymptomatic.
- Subserosal → Cause pressure symptoms
  IF connected with a stalk ⇒ pedunculated subserosal.
- Parasitic → Pedunculated, stalk becomes necrotic & breaks away from uterus, the recives it's blood from abdominal organs.
- Cervical/ Interligamentous (grows laterally into the broad ligament).
  - If it located in the lower uterine, may increase the likelihood of:
    1. Fetal malpresentation
    2. Caesarean section.
    3. Postpartum hemorrhage.

Risk factors:

- Nulliparity (or increase age of 1st pregnancy)/ Perimenopausal
- Early menarche (<10 YO)
- Reproductive age (25-45)
- African- American
- Obesity
- Family history


P/E
General → pallor / tachycardia →IDA
Abdomen→ increase in fundal height
Bimanual → localized / NON -tender / Irregular mass
↳ - Tender on palpation if degenerated fibroid.
- The Mass moves with the cervix
Investigations
Most common diagnostic method ⇒ US
Most definitive diagnosis ⇒ Biopsy/ D&C/ hysteroscopy
Differentiate fibroid from adenomyosis⇒ MRI
Hysteroscopy → removal of submucous myoma
Treatment
NO treatment ⇒Asymptomatic
UAE (uterine artery embolization) ⇒Preserve the uterus, Not preserve the fertility [ONLY for limited no. of fibroids]
Medical ⇒ Pre-surgical shrinkage by GnRH analogues, decrease size by 70%, given 3-6 months before surgery.
- Tranexamic acid may reduce menorrhagia associated with fibroids.
Surgical ⇒ • Myomectomy [preserves fertility], ⅓ of fibroid recur after it.
• Hysterectomy, definitive treatment
Degenerative fibroid:
• Hyaline (most common)
• Red /hemorrhagic (most common in pregnancy)
• Cystic degeneration
• Fat degeneration/+ calcification >> egg shell appearance
Management of fibroid degeneration during pregnancy :
Bed rest/ Analgesia/ No surgery



Adenomyosis

Benign disease, extension of endometrial glands and stroma into myometrium (INside the uterus), more than 2.5 mm beneath baselis layer.
Peak incidence ⇒ 40s

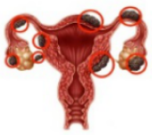
Risk factors: Endometriosis/ Uterine fibroids/ MultiParity/ D&C or past surgeries/ Excess of estrogen (The association between endometrial hyperplasia & adenomyosis)

Symptoms:
- Mostly Asymptomatic
- Menorrhagia, may cause anemia
- Dysmenorrhea, Dyspareunia
- Intermenstrual spotting
- Chronic pelvic pain
- Pressure symptoms: bowel & urinary

Approach
- P/E⇒ Uterus is globular & diffusely ,(SYMMETRICAL) enlarged/ Tender before and during menses.
- U/S & MRI ⇒ To differentiate between fibroid & adenomyosis.
- Biopsy ⇒ Definitive diagnosis

Treatment
- ♧ Medical: - OCPs/ Levonorgestrel IUD
  - NSAIDs & GnRH agonist
- ♧ Surgical: Hysterectomy (definitive)

Leiomyoma ⇒ Firm | Localized (Asymmetrical)| Non-tender | Pseudocapsule
Adenomyosis ⇒ Soft | Diffuse (symmetrical) | Tender



Endometriosis

Benign condition which inflammatory tissue similar to normal endometrial glands & stroma present OUT-side the uterus, respond cyclically to ovarian steroidal hormone.

Epidemiology
- 30s, nulliparous, infertile.
- ⅓ of women with chronic pelvic pain have visible endometriosis.

Etiology

3 Hypothesis:
° Retrograde menstruation (most acceptable one)
° Mullerian Metaplasia theory of Meyer
° The lymphatic spread theory of Halban
+ Genetic predisposition plays a role.

Risk factors

Family history/ Race- white / Autoimmune diseases/ High social class (delay marriage)

Sites
- Ovarian (Most common), known as endometrio-mas OR chocolate cyst
  - >4cm: surgical remover
  - <4cm: OCPs / symptomatic
- Broad ligament/ Peritoneal surfaces of the cul-de-sac including the uterosacral ligaments (Nodularity) and posterior cervix/ Rectosigmoid — Bluish/ brown spots

Symptoms

⇒ Dysmenorrhea / Dyspareunia / Dyschezia
⇒ Chronic pelvic pain: upon mense, pertonial stretch
⇒ Infertility (due to inflammation and adhesions)

Diagnosis

》 Laparoscopy (gold standered) + Histology (Negative histo doesn't exclude diagnosis)
• Histology: 2 out of 4 must be found in the endometrioma specimen ⇒ Endometrial epi / Endometrial glands / Endometrial stroma / Hemosiderin- laden macrophages.
• U/S: Ground glass appearance of ovary >> chocolate cyst.
• Laparoscopy: Kissing ovaries (endometriosis until proven otherwise)
• Bimanual exam: Tender, fixed adnexal mass.
• Rectovaginal exam: To feel uterosacral nodularity.
• Ovarian implants associated with scarring of the ovary.

》 CA-125 serum levels may be elevated - can be used to detect recurrance.

Management

■ Medical >> The goal is to induce Amenorrhea
- Pseudo pregnancy state: OCPs, Progesterone ⇒For long period
- Psuedo menopause state: GnRH Agonist ⇒ For temporary tx 3-6 months
- High androgen, Low estrogen state: Aromatase inhibitors.

■ Surgical >> Adhesion lysis & excision of endometrial implants
■ Definitive ⇒TAH with BSO / espicaly if >40 yrs

Infertility

- 40%-60% are infertile, 15% of infertile females have endometriosis.

Due to: 1. Adhesions
2. Dyspareunia (painful intercourse so no sex ⇒ infertility)
3. High PG levels [Affect tubal motility and corpus letum function]
4. Increase macrophages that engulf sperms
5. High levels of prolactin in 10% of cases

♡ Tx of infertility in endometriosis: IVF/IUI trial.

Amenorrhoea

Primary (Menstruation NEVER starts)

- **Absence of menses at age 14 WITHOUT secondary sexual characteristics.**
- **Absence of menses at age 16 WITH secondary sexual characteristics.**

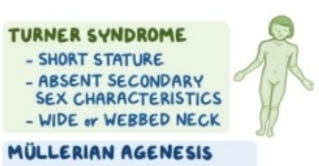
Causes

1. Anatomic:
a) Imperforate hymen
b) Vaginal agenesis/ septum
c) Mullerian agenesis [MRKH]
↳ Atretic uterus, cervix, upper ⅔ of vagina, BUT ovaries develop normally so normal level of FSH & LH

2. Hormonal :
a) Gonadal dysgenesis (Turner)
b) Androgen insensitivity
c) Hypothalamic pituitary insufficiency

3. Endocrine disorders:
a) Kallmann syndrome
(low GnRH, FSH, LH)

The most common cause of primary amenorrhea is chromosomal abnormality⇒ Turner Syndrome (45%) which is Gonadal dysgenesis.
• Mayer-Rokitansky-Küster-Hauser (MRKH) syndrome (second most common cause)



Diagnostics

- History
- Physical
- Pelvic US

Uterus present
$ Exclude >>
• Imperforate hymen • Vaginal atresia
•Transverse vaginal septum.

Test FSH and LH levels.
↓FSH, LH⇒ Hypothalamic-Pituitary Insufficiency (axis)
↑FSH, LH ⇒ Order Karyotyping (e.g.
XX: premature ovarian failure
XO: Turner syndrome
XY: Swyer syndrome

Uterus absent by U/S

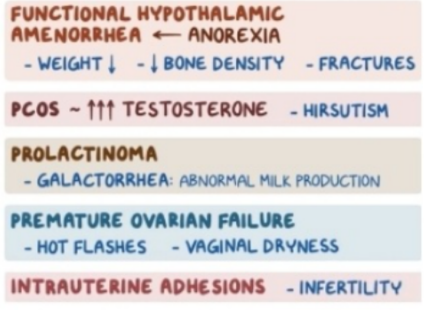
Confirm by MRI
Then order Karyotyping with testosterone level:
XX: ♀ with Mullerian agenesis
XY: Androgen insensitivity

Secondary (Menstruation SUDDENLY stops)

- **Absence of menses for 3 consecutive cycles (if regular) & 6 months (if irregular)**

Causes

✓ Pregnancy, rule out by B-HCG (most common cause)
✓ Functional Hypothalamic Amenorrhea: Dysfunction in the pulsatile secretion of GnRH
[Etiology >> strenuous exercise/ low calorie intake-Anorexia / low bone mineral density/ Sever stress ⇒ Female athlete].
✓ Anovulation: PCOS, Hyper-prolactinemia (also hypo-thyroidism cause hyper-prolactinemia)
✓ Estrogen deficiency: Premature ovarian failure, Sheehan syndrome.
✓ Outflow obstruction: Asherman Syndrome, Cervical stenosis.



Approach

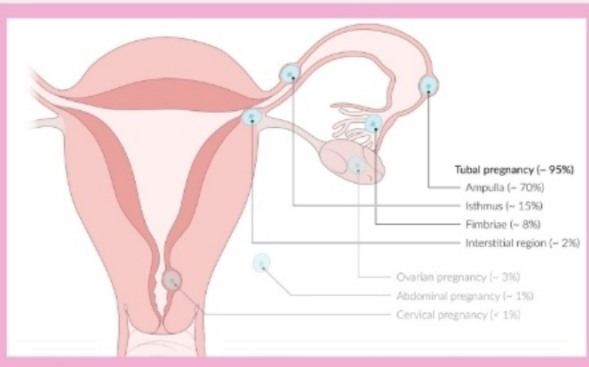
- History
- Physical
- B- hCG (to rule out pregnancy)

Obtain FSH, TSH, Prolactin

Progesterone challenge test
[Give her progesterone for 5 days then stop it!]
⇒ If there is Bleeding ⇒Cause is Anovulation (e.g., PCOS, idiopathic anovulation, premature ovarian failure)

⇒No bleeding ⇒ Give Estrogen & progesterone challenge test
↳ There is Bleeding, cause is low estrogen (hypogonadotropic hypogonadism)
↳ No bleeding, cause is outflow obstruction (e.g., Asherman syndrome)

Ectopic pregnancy



Pregnancy that is located outside the uterine cavity, incidence is increased, it's one of the leading cause of maternal mortality =6%.

*The day of implantation after sperm fertilize ovum is on the 6th day

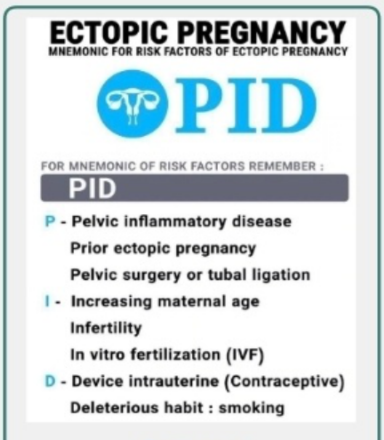
Sites

1. Fallopian tubes (95%)
   - Ampulla (Most common site, width 5-6mm)
   - Isthmus (wall is thicker)

   [Both need short weeks of amenorrhea to appear, Ampulla 6-7 weeks, isthmus <6 weeks]
   - Fimbriae
2. Uterine cornea, needs 10 weeks, most dangerous (risk of rupture).
3. Ovary (3%)
4. Abdomen (1%)
5. Cervix (Very rare)

Risk factors

1- Previous history of ectopic pregnancy (recurrence rate = 30%)
2- PID (STD) and infection:
Due to intratubal or peritubal adhesions & infection may destroy the cilia → suppress migration
3- Previous tubal surgeries ex: tubal ligation
4- Use of ART ex: IVF
5- Use of contraceptive methods : POP, IUCD
6- Smoking
7- Congenital malformation of the uterus
8- Endometriosis



Summary

Clinical presentation

- 4–6 weeks after their last menstrual period.

Triad of
》 Amenorrhea
》 Abdominal pain: acute pain, pelvic or lower abdominal pain radiating to the shoulder-ipsilateral (suspected tubal rupture).
》 Vaginal bleeding: spotting if ruptured, then it's intraperitoneal bleeding.

Approach

✓ Hx & physical >> Bimanual exam: palpate adnexal mass《Cervical motion tenderness》

✓ Investigations

1. Labs: B-HCG⇒ To confirm pregnancy
   ↳ Normal pregnancy HCG doubling after 72 hrs.
   ↳If it is increased without doubling then its ectopic.

2. Transvaginal ultrasound (TVUS)
- Indication: best initial imaging test

⇒Supportive findings
• Empty uterine cavity in combination with a thickened endometrial lining
• Possible free fluid within the pouch of Douglas
• Additional findings in tubal pregnancy
↳Tubal ring sign (blob sign): an echogenic ring that surrounds an unruptured ectopic pregnancy

3. Laparoscopy→Unstable patients suspected of having an ectopic pregnancy

** The discriminatory zone of β-HCG:
▪ 1500-2000 mIU/mL with transvaginal US, & up to 2300 mIU/mL with multiple gestates [2]
▪ 6000-6500 mIU/mL with abdominal US
⇒ Absence of an IU pregnancy on a scan, β-HCG level above the discriminatory zone represents an Ectopic or Recent Abortion.

Management

Depends on stability, site of ectopic pregnancy, state (rupture or not), desire of future fertility

Medical

Drug of choice is MTX (methotrexate)
♤ Indication :
• Stable(not Ruptured) /B-HCG<1500
• On U/S: no IU sac , no fetal heart activity (FHA), size <4cm

♤ Contraindication for MTX:
- Unstable patient or ruptured EP because MTX takes time to work
- Leukopenia/ thrombocyte <100k
- Active renal/ hepatic disease
- Active PUD
- Breast feeding
- +ve FHA (viable pregnancy)

Surgical

Indications:
▪ Failure of medical treatment, unstable, B-HCG>1500, positive FHA, size>4cm.
If stable⇒ Laparoscopy
If Unstable⇒ Laparotomy

1) Salpingectomy (removal of fallopian tube)
2) Salpingostomy (creation of an opening into the fallopian tube →Tube conserving operation

Gestational Trophoblastic Disease

Abnormal proliferation of trophoblastic (placental) tissue.
- ⬆️ B- hCG
- Extreme sensitive to chemotherapy
- Curable gyne malignancy & fertility preservation

Types

1. Molar- Hydatidiform (80%, Benign)
   1) Complete (classic) 90%
   2) Incomplete(partial) 10%
2. Persistent- invasive mole (10-15%,Malignant)
3. Choriocarcinoma (2-5%, malignant)
4. Placental site trophoblastic tumor (very rare, malignant)

* Malignant tendency to metastasize to other organs, especially the lungs.

Molar - Hydatiform
Risk factors:
1) Previous history
2) Extremes of age (<20 or >35)
3) Nulliparity 70%
4) Diet (low beta carotene, low Folic acid and animal fat)
5) Smoking
6) Infertility / History of OCP use
7) blood Group A
8) Women from Asia having a higher incidence.

Types

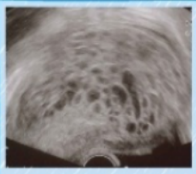
	Complete mole	Partial mole
	• 46,XX karyotype • Absent Fetal tissue - Fetal RBCs • Both of the X chromosomes are paternally derived. • Results from either : 》 75-80% of cases, fertilization of an "empty egg" by a haploid sperm (23 X), then duplicates (46,XX). 》 20-25%, Dispermic Fertilisation of an empty ovum.	Often 69,XXY (90%). ~ In almost all cases ⇒ Dispermic + ovum ~ 10% represent tetra-ploid or mosaic conceptions
	Hydropic(swollen) grape like vesicles with Severe hyperplasia	Present with a coexistent fetus. The fetus usually has a triploid karyotype and is defective
	Presentation ✓ Abnormal vaginal bleeding ✓ Uterus size greater than normal for GA ✓ HIGH B-hCG level (> than 100K) ✓ Endocrine symptoms (hCG subunits: α and β) α-subunit structurally resembles TSH): ↳ - Hyperemesis gravidarum - Hyperthyroidism symptoms - Preeclampsia (before the 20th week of gestation, headache, visual disturbances, epigastric pain, HTN) - Ovarian theca lutein cysts: bilateral, large, adnexal masses, tender, cause abdominal distension.	Presentation ✓ Missed abortion/ Size of uterus=GA ✓ Slightly elevated B-HCG ✓ Vaginal bleeding ✓ Pelvic tenderness
		2-4% risk for Invasive mole NO risk for choriocarcinoma
	Higher Risk for invasive mole 15-25% Risk for choriocarcinoma 4%	Follow up: 8 weeks for B-HCG to become normal
	Follow up: 14 weeks for B-hCG to become normal	

Diagnosis

■ US
▫ "Honeycomb, bunch of grapes", or "snowstorm" pattern ⇒ diagnostic for Complete mole
~ No amniotic fluid/ No fetal parts/ Lack of fetal heart tones

▫ PARTial hydatidiform mole: Fetal PARTs may be visualized/
Fetal heart tones may be detectable/
Amniotic fluid is present/
Increased placental thickness

■ Uterine evacuation & histo examination (for definite diagnosis and treatment)



Complete mole

Treatment

1. Standered ⇒ Suction evacuation followed by sharp curettage of the uterine cavity, regardless of the duration of pregnancy.

2. Patient must be monitored with Weekly serum assays of β-hCG until 3 consecutive levels have been normal, then Monthly β-hCG levels until 3 consecutive levels have been normal
(Patients should use effective contraception during follow-up).

3. Chemotherapy (usually methotrexate) if unresolved.

· 95-100% of patients with a GOOD prognosis.
· 50-70% of cases with a POOR prognostic features.
⇒ The majority of the patients who die have brain or liver metastases.

* Oxytocin should be avoided at present since it increases the sensitivity of the uterus to PGs.

Abnormal Uterine Bleeding

Definition

Any symptomatic variation from normal menstruation, in women between menarche & menopause, including intermenstrual bleeding.

Menstrual abnormalities

- ✓ Poly-menorrhagia ⇒ Abnormal frequent menses at intervals of <21 days.
- ✓ Oligo-menorrhea ⇒ Uterine bleeding occuring at intervals >35 days.
- ✓ Menorrhagia (hyper-menorrhagia)⇒ Excessive (>80mL) &/or prolonged menses (>7 days) occurring at regular intervals [Amount].
- ✓ Metro-rrhagia ⇒ Bleeding occuring at irregular intervals [Frequency].
- ✓ Meno-metro-rrhagia ⇒ Heavy & irregular uterine bleeding.
- Dysfunctional uterine bleeding ⇒ Caused by ovulatory dysfunction [Hormonal imbalance], mainly after puberty/ premenopausal.
  - Not due to organic gynecologic disease or pregnancy.

Diagnosis

- Hx & physical: general & gyne [PV bimanual, Speculum]
- PALM- COEIN

Structural causes
Polyps
Adenomyosis
Leiomyoma[a]
Malignancy and hyperplasia
Nonstructural causes
Coagulopathy
Ovulatory dysfunction
Endometrial
Iatrogenic
Not yet classified

- -P : intermenstrual bleeding
- -A : dysmenorrhea, heavy menstrual bleeding, enlarged, globular, tender uterus.
- -L : heavy menstrual bleeding
- -M : heavy menstrual bleeding or irregular bleeding .
- -C : MC von Willebrand disease/ Drugs⇒ Aspirin - Anticoagulant
- -O : irregularity & variable volume
- -E : Endometritis :heavy menstrual bleeding & intermenstrual bleeding.
- -I : Gonadal steriods / Cesarean scar defect
- -N : Arteriovenous malformation

- Investigations
  - Urine test ⇒ Pregnancy test / to rule it out
  - Blood test ⇒CBC,TSH, Prolactin, Estrogen & Progesterone, Iron, Ferritin, Coagulation test, LFT, Creatinine & BUN
  - Saline infusion sonohysterography ,US, D&C with biopsy, laparoscopy
  - Cervical smear/ Transvaginal sonography (TVS)/ Diagnostic hysteroscopy

ACUTE Excessive Bleeding in non-pregnant women

a. Assessed for hemodynamic stability

b. Hospitalization and transfusion for who have SEVER anemia (Hb ≤7 g/dL) & those who are hemodynamically UNstable.

c. Imaging studies can be delayed until the bleeding is controlled
For women who have risk factors for cancer, biopsy is indicated once the bleeding has been stabilized & Hb level is normal.

Tx ⇒
• First line medical therapy once the bleeding is minimal & pt. is stable >> High-dose progestin-ONLY therapies.
• Treat the underlying cause/ D&C/ Balloon/ Selective Embolization of uterine blood vessels.

Chronic Heavy Menstrual Bleeding

The following steps should be considered:
(1) Normalize prostaglandins→ Can reduce blood loss up to 20-30%, NSAIDs may be used.

(2) Anti-fibrinolytic therapy→Can reduce blood loss up to 40%, used to stabilize clots in uterine arterioles, NOT be combined with estrogen- containing medications.

(3) Coordinate endometrial sloughing→ By progesterone, OCPs, patches or vaginal rings.

(4) Endometrial suppression→ Levonorgestrel intrauterine system (LNG- IUS), endometrial ablation.

Permanent cessation of menstruation caused by failure of ovarian estrogen production in the presence of high FSH, LH.
- Diagnosed after 12 months of amenorrhea.

Mean age= 51 Years.

Notes

- **Perimenopause** — Time period from the first instance of climacteric symptoms caused by fluctuating hormonal levels, to 1yr after menopause.
  - Length of perimenopause is 4 years, vary greatly in different women.
- **Premenopause** — The period immediately prior to the menopause
- **Climacteric period** — The transition from the reproductive phase to the non-reproductive state, this phase incorporates the perimenopause.
- **Postmenopause** — Starting from the final menstrual period, regardless of whether the menopause was induced or spontaneous
- **Premature menopause** — Menopause at age < 40
  ↳ May be caused by surgical removal of both ovaries with or without hysterectomy or iatrogenic ablation of ovarian function by chemotherapy or radiation ⇒ Induced menopause
- **Premature ovarian failure** — Age < 30, caused by abnormal karyotypes involving the X chromosome, the carrier state of the fragile X syndrome, galactosemia or autoimmune disorders that may cause failure of a number of other endocrine organs.

Pathophysiology

Most women ovulate about 400 times between menarche & menopause, nearly all other oocytes are lost through Atresia.

When the oocytes either have all ovulated or become atretic, the ovary becomes minimally responsive to pituitary gonadotropins, the ovarian production of estrogen and progesterone ends, and ovarian androgen production is reduced.

Hormones affected

1- Inhibin (decrease): which is the hormone produced from the ovaries causes inhibition of FSH
2- Estradiol (decrease)
3- Estron ⬆️
4- Androgen production decreases, but receptors became more sensitive to it because of less opposition by the estrogen.
5- Progesterone declines to low levels
6- FSH and LH: increase FSH > 40 ⬆️

Symptoms
《 Menopausal HAVOCS: Hot flashes/Heat intolerance, Atrophy of Vagina, Osteoporosis, Coronary artery disease, Sleep impairment 》

✓ Early symptoms [Short-Term Effects (0-5y)]:
▪ Amenorrhea(2ry): the most common symptom
▪ Vasomotor symptoms:
  · Night sweats
  · Hot flashes, 85% of women experience as they pass through the climacteric, but about half of these women are not seriously disturbed by them.
▪ Psychological: depression, anxiety, insomnia & irritability.
▪ Loss of concentration and poor memory.
▪ Joint aches and pains.
▪ Dry and itchy skin due to collagen loss.
▪ Hair changes (coarse hair).
▪ Decreased libido.

✓ Intermediate Effects (5-10y)
- Vaginal dryness
- Dyspareunia
- Sensory urgency
- Recurrent UTIs
- Urogenital prolapsed
- Stress incontinence

✓ Long Term (>10y)
1. Osteoporosis: with estrogen deprivation, osteoclastic activity far exceeds the osteoblasts, ability to lay down bone.
→ Spinal column and femoral neck are most commonly fractured.
Treatment:
· Lifestyle modification (Increase calcium, vitamin D consumption, stop alcohol and smoking, and doing weight-bearing exercises)
· Bisphosphonates (alendronate): first line
· Estrogen (with or without progestin) shouldn't be used as the first-line treatment.

2. Cardiovascular disease → The m.c.c of mortality in 50% in postmenopausal women/ Increase LDL , decrease HDL

3. Dementia

Investigations
· FSH > 30 IU/L, preferably 2 measurements, 2 weeks to 3 months apart.
· Cardiovascular disease risk assessment
· Skeletal assessment
· Breast screening and mammography
· Cervical smear

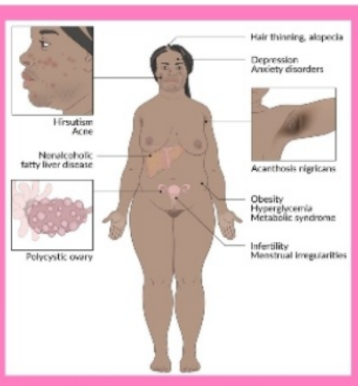
Managment

Non-Hormonal — Antidepressant, B-Blocker, a-Agonist (vasomotor symptoms).

Hormonal
- HRT: Estrogen + Progesterone (to protect the endometrium from unopposed estrogen)
- OR ERT: Estrogen alone (these are ONLY indicated in females post hysterectomy as estrogen alone increases risk of Endometrial cancer).
+- Testosterone (for sexual drive)

▪ Minimum effective dose for shortest duration→ Average 2-3 years.
▪ Indicated in case of Vasomotor symptoms (hot flashes) OR Urogenital symptoms (vaginal atrophy)
▪ Risks: Breast CA / VTE / Endometrial CA
▪ Absolute contraindications: Pregnancy / Breast CA / Endometrial CA / Uncontrolled HTN / Otosclerosis / known VTE or thrombophilia

PCOS



A condition of chronic anovulation resulting subfertility, irregular bleeding, obesity, hirsutism. The age of onset is most often premenarchal.

Criteria for diagnosis

At least 2 of the Rotterdam criteria, after ruling out other causes

1. Oligo / Anovulation
2. Hyperandrogenism [Acne/ hirsutism/ obesity]
3. US ⇒ Polycystic ovaries→ at least 10-12 small follicles in one ovary (mostly bilateral), <10 mm in diameter & /or increased ovarian volume >10ml - string of pearls

* US should NOT be used to diagnose PCOS within 8 years of menarche.

* Obesity and insulin resistance are strongly associated with the syndrome, they are not essential to the diagnosis.

* Congenital adrenal hyperplasia / Androgen secreting tumours / Cushing / Thyroid dysfunction / hyper-prolactinemia ⇒ MUST BE EXCLUDED

Phenotypes

- A: Androgen excess + ovulatory dysfunction + PCO morphology.
- B: Androgen excess + ovulatory dysfunction
- C: Androgen excess + PCO morphology
- D: Ovulatory dysfunction + PCO morphology

Risk factors

1) Women with oligo-ovulatory
2) Obesity/ insulin resistance / Type 1,2 DM
3) History of premature adrenarche
4) 1st degree relatives with PCOS
5) Women using Anti- epileptic drugs

Presentation

✓ Menstral irregularities
✓ Hyperandrogenism symptoms
✓ Subfertility
✓ Obesity / DM / acanthosis nigricans
✓ Psychological: Anxiety, depression, psychosexual dysfunction, eating disorders
✓ Metabolic syndrome / ↑ risk of sleep apnea

Diagnosis

Hx & physical then US

Labs: TSH/ prolactin/ FSH/ LH/ Insulin- like growth factor 1 (ILGF1) / Metabolic screening (lipid profile- 2hr75 g OGTT if BMI>28) / testosterone - SHBG

Treatment

1. Diet & exercise (1st line treatment)→ wt. loss that reduce hyperandrogenism & insulin resistance.
2. Pharmacologic treatment for anovulation, hirsutisms, menstrual irregularity .
3. Surgical : Laparoscopic ovarian drilling - for fertility

⇒ Combined OCPs, Metformin, clomiphene, spironolactone

- COCPs → Regulate periods, reduction in hair growth & minimizes endometrial hyperplasia.
- Laser & electrolysis → Localised hirsutism
- Clomiphene citrate → improve fertility by induce ovulation [Triggers the brain's pituitary gland to secrete an increased amount of FSH & LH, this action stimulates the growth of the ovarian follicle and thus initiates ovulation]

○ Side effects of clomiphene:
- vaginal bleeding
- breast tenderness
- headache
- nausea /vomiting
- diarrhea/ flushing
- blurred vision or other visual disturbances

Ovulation induction in PCOS
-1st line: Letrozole(Anti-estrogen) superior to clomiphene citrate
-2nd line: gonadotrophins (FSH, LH)
-3rd line: IVF

Polycystic ovary syndrome (PCOS)

Etiology
Unknown
Associated with insulin resistance and obesity

Diagnostics: Rotterdam criteria
- Oligoovulation or anovulation
- Hyperandrogenism
  - Clinical features (acne, alopecia, hirsutism) or
  - Laboratory features (↑ testosterone)
- Enlarged and/or polycystic ovaries on ultrasound examination

Treatment
- Weight loss
- No wish to conceive:
  - Combined oral contraceptives (1st line)
  - Metformin (2nd line)
- Wish to conceive:
  - Ovulation induction (1st line: letrozole)

Complications
- Cardiovascular disease
- Type 2 diabetes mellitus
- Malignancy (e.g., endometrial cancer)
- Pregnancy loss

- CVD risk modification: lipid profile monitoring every 2 yrs / BP monitoring every 1yr / Assess for preDM with OGTT / Wt. monitoring / Assess cigarette smoking.

Subfertility

Inability to achieve pregnancy after 12 months of Regular Unprotected Intercourse in women <35 years old, and 6 months in women >35 years.
- Fecundability (monthly chance of pregnancy) is 20%
• Within one year, 85% of pregnancy occurs.
• Investigations for infertility start after one year.

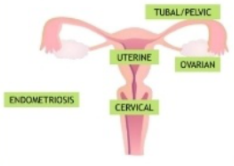
• Primary infertility → Occurs without any prior pregnancy, most common, 70% of cases.
• Secondary infertility →Follows a previous conception.

Important terms

Sub-fertility: A decrease, but not an absence, of fertility potential
Sterility: Complete inability to achieve fertility

Causes

♀ 40% of cases



✓ Ovarian
1. PCOS (WHO-2), most common, 85%.
2. Premature ovarian failure (WHO-3)
3. Hyper-prolactinemia / Thyriod disorder/ Obesity

WHO Classification of anovulation:	
	WHO Class I: Hypogonadotropic Hypogonadal Anovulation
	WHO Class II: Normogonadotropic Normoestrogenic Anovulation
	WHO Class III: Hypergonadotropic Hypoestrogenic Anovulation
	Hyperprolactinemic Anovulation

✓ Tubal
1. PID
2. Surgical procedure or ligation
3. Pelvic adhesions
4. Pseudo-obstruction: mucous plug, tubal spasm (dx: hystrosalpingiogram)
5. Endometriosis: Most common in tubal infertility, sometimes ovarian.
↳ Treated by surgical removal of adhesions, reanastomosis tuboplasty, IVF

✓ Uterine
1. Congenital malformation (mullerian anomalies)
2. Submucosal fibriod / polyps
3. Asherman syndrome (adhesions)

✓ Cervical
1. Cervical stenosis, chronic cervical inflammation
2. Mullerian duct abnormality
↳ Treated by surgical dilatation, IUI

✓ Controversies
1) Immune factors (killer T cell)
2) Thrombophilia
3) Luteal phase defect (No progesterone)
4) Ovarian cyst

♂ 30% of cases

- Environmental → Smoking, alcohol, excessive heat, tight underwear
- Sexual dysfunctional → Erectile, ejaculation
- Structural factors → varicocelle, testicular torsion, vasectomy
- Abnormal semen → mumps, Anti-Sperm Ab
- Genetics → cystic fibrosis, Klinefelter, immobile cilia

10% combination ♂+♀

20% unexplained infertility

Diagnosis

Hx+ Physical

Investigations start from least expensive

1) Semen Analysis
If abnormal→ Repeat test again within 4-6 weeks, if abnormal → IUI / Intra-cytoplasmic sperm injection (ICSI)

2) Anovulation
- Labs: TSH, T4, Prolactin, Midluteal Progesterone, Procalcitonin, Anti-Mullerian hormone [secreted by granulesa cells - to measure the ovarian resevere].
  ↳ Correct the cause if low T4 or high prolactin.
  ↳ Uncorrectable ⇒ PCOS ⇒ induction the ovulation by Clomiphene citrate

- US: Antrial follicle count - to measure ovarian reserve.

3) Tubal disease (more expensive, invasive)
- Hysterosalpingogram HSG if abnormal
↳ Laparoscopy
- Tuboplasty: Reconstruct damaged oviducts
- Salpingectomy & IVF if severely damaged

4) Unexplained infertility:
IUI then IVF

Indications for IVF (in vitro fertilization):
- **Oligospermia**
- **Irreparable tubes**
- **Unexplained fertility**

PID

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

Organisms {Sexually transmitted microorganisms, it is rarely a single organism that's responsible for PID}

- ~ Chlamydia trachomatis (mc)
- ~ Neisseria gonorrhea.
- ~ E.coli / Streptococcus

Risk factors

1. Age < 35 (especially in teens)
2. Multiple sexual partners
3. Unprotected intercourse
4. IUCD (while other contraceptive methods decrease the risk)
5. Nulliparity
6. History of STD

Acute PID

- Bilateral abdominal tenderness / Cervical motion tenderness / Mucopurulent discharge
- ⬆️ no. of WBCs / ESR & +ve culture/ normal urine analysis

Diagnosis

- Mainly Clinical, triad of symptoms → pelvic pain/ cervical motion tenderness / adnexal tenderness
- Other symptoms include: lower abdominal pain, excessive vaginal discharge, chills, direct or rebound abdominal tenderness

Management

Ceftriaxone IM single dose + Doxycycline orally 2×1 for 14 days unless there is indication for admission

↳ Criteria for hospitalization >>

1. Surgical emergencies (e.g., appendicitis) not ruled out
2. Failed oral treatment
3. Severe illness (toxicity: nausea, vomiting, high fever)
4. Tubo-ovarian abscess demonstrated on U/S or suspected clinically
5. Pregnancy

* Sexual partners of women with PID should be evaluated and treated for urethral infection caused by Chlamydia or gonorrhea

↳ Treatment with Doxycycline if sexual contact with partner in the last 6 days.

TUBO-OVARIAN abscess

⇒End-stage process of acute PID.

- Symptoms: severe bilateral pain /SEPTIC patients, high fever, elevated HR, decreased BP, peritoneal signs, adnexal masses.
- On CT: bilateral complex pelvic masses.
- Management: Admit, IV clindamycin + Gentamycin.

75% of women respond to Abx alone.

Failure of medical therapy suggests the need for drainage of the abscess.

Chronic PID

- Chronic Bilateral pain, infertility, dyspareunia, ectopic pregnancy, abnormal bleeding
- Cervical motion tenderness and bilateral adnexal tenderness, No discharge, No fever or tachycardia.
- Investigations: normal WBCs and ESR, -ve culture, on US: hydrosalpinx.

Diagnosis

Laparoscopy by visualization of adhesions

Management

Analgesia and adhesion lysis might be helpful in fertility

- Fitz-Hugh Curtis syndrome: RUQ pain with Chronic PID and peri-hepatitis with adhesions seen at the liver capsule.

Complications of PID

- **Infertility**
- **Abortions (recurrent)**
- **Abnormal bleeding.**
- **Ectopic pregnancy**
- **Dysparunea**

Pelvic organ prolapse

Protrusion of the pelvic organs into the vaginal canal or beyond the vaginal opening.
* Diagnosis by vaginal examination
* 50% of women develop prolapse

Causes /Risk factors

- Multiple vaginal deliveries (most important) / macrosomia/ Forceps
- Obesity and chronic cough
- Constipation/ heavy lifting
- Weaning of pelvis: decrease in connective tissue
- Age, menopause, HRT
- Previous history or family hx of pelvic organ prolapse (POP)
- Hysterectomy

Classification

Level 1
Any defect in apical support of vagina (Cardinal ligament & uterosacral ligament) ⇒ Uterine prolapse.

* Apical prolapse that occurs post-hysterectomy is called vault prolapse, due to loss of the integrity of the ant. & post. vaginal walls.

Level 2
- Any defect of Ant. support {Pubocervical Facia}⇒ Cystocele MC type
- Any defect of Post. support⇒ either Rectocele or Enterocele

Level 3
Any defect Perineal membrane and body ⇒ Deficient Perineum

Presenting Symptoms

Feeling of vaginal fullness/ heaviness, progress over the day & most noticeable AFTER prolonged standing.

Other symptoms

- Cystocele: Stress urinary incontinence (SUI) / urgency/frequency
- Rectocele: Need to manually splint for complete bowel elimination/ soiling & fecal incontinence
- Deficient perineum: Widening of vagina/Unsatisfactory sexual life

Quantifying & Grading

- Extent of prolapse is evaluated relative to the hymen.
- Hymen plane is 0/ Above hymen -ve no./ Below +ve no.
- Grading according to the position of the [most severe portion of prolapse] after the full extent of the protrusion:
  - 》Grade 1 : MORE than 1 cm ABOVE hymen (> -1 cm)
  - 》Grade 2: 1cm above or below hymen (-1cm _ +1cm)
  - 》Grade 3: MORE than 1 cm below the level of the hymen but no further than 2 cm less than the total vaginal length1 (+1cm to TVL- 2cm)
  - 》Grade 4: Complete eversion of vagina & uterus is outside, called procidentia & it's the most advanced stage.

Management

Asymptomatic = NO treatment

Conservative management
1) Kegel exercises (voluntary contraction of pubococcygeus muscles), C/section as effective as kegel exercises in preventing POP (only the first 3 C/S)
2) Estrogen Replacement Therapy
3) Vaginal pessaries

Surgical management
- Anterior colporrhaphy for cystocele
- Posterior colpoperineorrhaphy for rectocele
- Vaginal hysterectomy for uterine prolapse
- Sacrocolpopexy for vaginal vault prolapse

Urinary Incontinence

Involuntary loss of urine that is a social or hygienic problem.
* Risk factors same as Pelvic Organ Prolapse.

Types

Stress urinary incontinence (SUI)
- Most common type of incontinence, strong association with cystocele.
- Involuntary leakage of urine in response to physical exertion, sneezing or coughing (⬆️ intra-abd. pressure)

- Diagnosis
• Cough Stress Test→ pt. is examined with a full bladder in the lithotomy position, asks the patient to cough and observe the urethral meatus for any urine leak

- TREATMENT
• Conservative: Kegel exercises/ Weight loss (mild to moderate)
• Surgical: TVT (Tension free Vaginal Tape): synthetic mesh

■ 2 Mechanisms of stress incontinence:
1. Urethral Hyper-Mobility (85-90%)
Most common/ loss of urine in small amounts/ No night symptoms.
[Defect in Pubo-cervical fascia]

2. Intrinsic Sphincter Deficiency (10-15%): Older in age [Estrogen deficiency]/ more severe symptoms that may occur at rest/ larger amounts of urine.

OverActive bladder / Urge urinary incontinence (UUI)
• Most common type in ♂️ and 2nd most common type in ♀️
• Involuntary leakage of urine immediately preceded by urgency.
• 90% of patients it's idiopathic/ 10% underlying neurological cause.

• DIAGNOSIS
Urodynamic testing→ unstable bladder (uninhibited detrusor contractions), decreased bladder capacity & strong urinary flow

• TREATMENT
1. Behavior (1st line)⇒ Reduce fluid intake & avoid liquids during evening hours + reducing caffeine intake /Kegel exercises/ Weight loss.
2. Drugs (most commonly used): Anti-muscarinics (gold standard), then β3 agonist (relaxation receptors in bladder)
Ant- muscarinics side effects: Dry mouth / constipation/ blurred vision/ somnolence.

- Detrusor overactivity 》Diagnosed by urodynamic observation, causes sensation of urgency

OverFlow urinary incontinence
The involuntary loss of urine that occurs when the bladder overfills.
• Nocturia is common in these patients
• Result from hypotonic bladder, or outflow obstruction.
• The Major causes are DM and neurological diseases.
• Most common cause in males is BPH
• Cause in females is large prolapse after surgery (most common), big cystocele, sever urethral stenosis.
• Management : Treat underlying cause if possible /intermittent self-catheterization or continuous bladder drain (suprapubic)

Bypass incontinence (Fistula)
• Uncommon cause of urinary incontinence
• ALWAYS need surgical correction

Mixed urinary incontinence (SUI+UUI)

Discharge

Bacterial vaginosis (Gardenella)

- **Most common vaginal infection in women (22–50% of all cases)**
- Not a true infection, but lower concentrations of Lactobacillus acidophilus lead to the overgrowth of Gardnerella vaginalis and other anaerobes, without vaginal epithelial inflammation due to an absent immune response.
- Risk factors
  - 1) Sexual intercourse (primary risk factor, but it is not considered an STD - NO effect with treatment of sex partner)
  - 2) Intrauterine devices
  - 3) Vaginal douching
  - 4) Pregnancy
- Clinical features
  - Commonly asymptomatic
  - Increased vaginal discharge, usually grey or milky with a fishy odour
  - Pruritus and pain are UNcommon.
- Diagnostics: diagnosis is confirmed if three of the following Amsel criteria
  - Clue cells
    - Vaginal epithelial cells with a stippled appearance and fuzzy borders due to bacteria adhering to the cell surface.
    - Identified on a vaginal wet mount preparation.
  - **Vaginal pH > 4.5**
  - Positive amine test (whiff test): The addition of KOH to a sample emits a characteristic amine odor.
  - Thin, homogeneous gray-white or yellow discharge that adheres to the vaginal walls
- Treatment is only in symptomatic patients — Metronidazole OR Clindamycin for 7 days.
- Complications — Preterm delivery, spontaneous abortion, postpartum endometritis, reinfection (consider retesting after 3 months).

Candida Albicans Vaginitis

- **It is STD**
- Risk factors
  - Pregnancy {High levels of glycogen, estrogen, and progesterone favor colonization with Candida by providing carbon}
  - Immunodeficiency, both systemic (e.g., diabetes mellitus, HIV, immunosuppression) and local (e.g., topical corticosteroids)
  - Antimicrobial treatment (e.g., after systemic antibiotic treatment)
- Clinical features
  - White, crumbly, and sticky vaginal discharge (cheese-like), odorless
  - Erythematous vulva and vagina
  - Strong pruritus, dysuria, dyspareunia
- Diagnostics
  - Vaginal pH within normal range (4–4.5)
  - Pseudohyphae on a vaginal wet mount with KOH
- Treatment — Oral fluconazole and Azole cream

Trichomonas vaginitis

- Its STD, Flagellated pear-shaped protozoan
  - Can reside Asymptomatically in male seminal fluid
- Clinical features
  - Foul-smelling, frothy, yellow-green, purulent discharge
  - **Strawberry cervix (erythematous mucosa with petechiae)**
  - Pruritus, burning sensation, dyspareunia, dysuria
- Diagnostics
  - pH of vaginal discharge > 4.5
  - Wet mount exam: Trichomonas
- Treatment — Metronidazole for both partner
- Complications — Preterm delivery, intrauterine growth restriction

Ovarian cysts

Mostly asymptomatic, discovered incidentally, resolve without treatment.
* Aim of their management⇒ Rule out malignancy and to avoid cyst complications (rupture, torsion, hemorrhage).
* It could be cystic or solid / Most of them are cystic.

》Benign ovarian mass: Mobile/ Soft/ Smooth surface/ Unilateral < 8cm / NO septations/ NO Doppler flow.

Types

Functional (physiological)

Follicular cyst

- Develops when an ovarian follicle fails to rupture, persist several cycles & reach 10cm, resolves after 2-4 months.
- Common in reproductive age.
- Most follicular cysts are painless.

Corpus luteal cyst

- Develops if the corpus luteum grows to 3 cm and fails to regress normally after 14 days, thin-walled, fluid filled without septations or calcifications.
- Smaller than follicular but more firm.
- May cause pain or signs of peritoneal irritation.

↳ This can develop into:
1-Hemorrhagic cyst: Invasion of ovarian vessels into the corpus luteal cyst.
2-Theca-luteal cysts: May develop with the high levels of hCG present in pts with a hydatidiform mole / choriocarcinoma / pts undergoing ovulation induction with Clomiphene usually bilateral.

Benign neoplastic

Epithelial ovarian neoplasms (mc)

- Derived from the mesothelial cells lining the surface of the ovary.
- Aged > 40 yrs

1- Serous Cystadenoma: MOST common, resembles the lining of the follapian tubes, bilateral in 10% of cases, 70% are benign. They are small, unilocular, form "Psammoma bodies "; calcific concentric concretions.

2- Mucinous Cystadenoma: resembles the endocervical epithelium, bilateral in < 10% of cases, 85% are benign. They are large, multilocular, thick and mucinous fluid, form " Pseudomyxoma peritonei".

3- Endometrial Cystadenoma: difficult to differentiate from ovarian endometriosis, associated with pelvic pain and deep dyspareunia due to adhesions.

Sex Cord–Stromal Ovarian Neoplasms

- Occur at any age, more commonly postmenopausal, may secrete hormones and cause bleeding.

1- Granulosa cell tumors: Locally malignant but have good prognosis, grow very slowly, solid tumor, recurrence is common, secrete Estrogen & Inhibin and predispose to endometrial cancer.

2- Theca cell tumor: benign, solid, unilateral, mostly in postmenopause, secretes Estrogen and causes bleeding.

3- Fibroma: rare, in elderly, hard, mobile, causes ascites and pleural effusion (Meigs syndrome)

4- Sertoli-Leydig cell tumor: low grade malignant, found around the age of 30, very rare, small, unilateral, may produce Androgens and signs of virilization.

Germ-cell tumors (teratoma)

- Most common ovarian neoplasm in the reproductive age.
- 2-3% is malignant.
- Derived from all three germ layers (ectoderm, mesoderm, endoderm).

1- Benign cystic teratoma (dermoid cyst): composed of ectodermal tissue (sweat and sebaceous glands, hair follicles, and teeth), with some mesodermal (bone, cartilage, muscle). 60% asymptomatic.
* monodermal teratomas: struma ovarii (composed entirely of mature thyroid tissue)

2- Mature solid teratoma: rare and must be differentiated from immature teratoma which is malignant.

Diagnosis

Bimanual exam & Pelvic US (TV U/S).

Management

Reproductive age

• Asymptomatic OR <5 cm ⇒
Conservative, re-examine the pt after her next menses + follow up by U/S and CA125 checking after 3 months.

• If complex lesion (septations/ solid component), OR Simple cyst >7 cm, OR Symptomatic⇒
Surgical exploration by Laparoscopy + cystectomy vs oophorectomy (fertility preserved)

Postmenopausal

1- Simple cyst < 1cm⇒ No further action

2- Simple cyst 1-5cm without features of malignancy and normal CA125 OR Asymptomatic⇒ Conservative + follow up (repeat U/S and CA125 every 4 months for 1yr)

3- Solid, complex, fixed, > 5 cm, Symptomatic or painful⇒ Surgical exploration by laparoscopy + bilateral salpingo-oopherectomy +/- hysterectomy

Ovarian CA

- The WORST gynecological CA⇒ Early detection is difficult (asymptomatic) and diagnosis at advanced stage (stage III).
- The leading cause of death of the gynecological cancers.
- Mean age is 50-60 years.

Risk factors

- **Early menarche/ Late menopause / Late age of 1st pregnancy.**
- **BRCA-1 gene/ Family history/ Past history of ovarian cancer**
- **Nulliparity/ Infertility.**
- **Estrogen replacement therapy**

Protective factors

Decreased lifetime ovulation (OCPs, PCOS, tubal ligation) & parity

Signs and Symptoms

■ Symptoms

Nonspecific, 1st symptoms are GI (vague abdominal pain or bloating), pressure symptoms (urinary frequency or urgency or constipation), dyspareunia, menstrual irregularity, swelling due to ascites at late stages.

■ Signs

Vaginal or rectal exam will reveal a solid, irregular, fixed pelvic mass, upper abdominal mass, combined ascites

According to type of cell origin

1- Epithelial origin (80% of ovarian cancer/ Postmenopausal).

》Types

1 ▪ Serous (mc 75%)→ resembles glandular epithelium of the fallopian tube

2 ▪ Endometrial → resembles proliferative endometrium

3 ▪ Mucinous→ resembles endocervical glands

4 ▪ Clear cell (Brenner - undifferentiated)→ resembles secretory or gestational endometrium

High grade serous CA of the ovary arise from Fimbrial end of fallopian tube rather than from the ovary.

2- Germ cell (15%, young women).

Types⇒

▪ Dysgerminoma: the ovarian counterpart of testicular seminoma, the most common malignant germ cell tumor, most frequently encountered ovarian malignancy during pregnancy, excellent prognosis, radiosensitive.

▪ Endodermal sinus tumor (yolk-sac carcinoma): highly malignant, in children and young females, fatal within 2 yrs of diagnosis

▪ Immature teratoma

▪ Embryonal CA

▪ Choriocarcinomarifac

3- Stromal (5%, all ages).

Types⇒ Granulosa cell (E2), Sertoli-Leydig cell tumor (androgen)

4- secondary (metastatic):

krunkenberg tumor: uniform enlargement of the ovaries (bilaterally) due to diffuse infiltration of the ovary stroma by metastatic signet-ring cell carcinoma

Diagnosis

** TV U/S with or without doppler [Most important diagnostic tool].

• Diagnosis requires exploratory laparOtomy (NO role for laparoscopy or abdominal tapping or biopsy for diagnosis or treatment because it may lead to RUPTURE or dissemination of the tumor).

• Work up: CBC, KFT, LFT, CXR

• Tumor markers: (CA-125) (HE4) (AFP: yolk-sac tumor) (LDH: Dysgerminoma) (Inhibin: granulosa cell tumor) (hCG: non gestational ovarian cancer) (CEA-CA19-9: suspected colorectal or pancreatic cancer).

▪ Bone scan for metastasis

Prognostic factors

Pathologic features
* Stage
*Histolgic type
With the exception at clear cell CA (already the worst prognosis)

Biologic factors
Diploid tumors have better prognosis than aneuploid tumors

Clinical factors
* Pt. age
* Volume of ascites

Treatment

• Initial approach to all pts⇒ Surgical exploration of the abd. and pelvis.
• Early stage: TAH, BSO
• In patients with grade 1 or 2 tumors confined to one or both ovaries after surgical staging, NO further treatment is necessary.
• Patients with poorly differentiated (grade 3) are subsequently treated with systemic chemotherapy.
• Advanced stage: cytoreductive surgery → debulking then chemotherapy

Follow-up

Every 3 months for 1 year
Every 6 months yearly

Hx, physical exam, CA125

IF recurrence is suspected clinically or by CA125 — **Imaging**

Endometrial CA

- The MOST COMMON gynecological cancer worldwide.
- Any factor that ↑ exposure to unopposed estrogen (excessive hyperstimulation of the endometrium without the stabilizing effect of progesterone) ↑ risk for type-1 (estrogen-dependent) endometrial cancer to develop.

- Recurrent Disease :
. 75% of cases develop within 2 years of treatment.
. Most commonly at the vaginal vault, 75% of those will be treated by RTX.

Risk factors

- Obesity: Increased estrone/ HTN/ DM.
- Endometrial hyperplasia[gland to stroma ratio >50%].
- Late menopause. • PCOS • Nulliparity
- Estrogen replacement therapy(long term)
- Tamoxifen for breast cancer (2-3 fold increased risk)
- Lynch || syndrome: hereditary nonpolyposis colon cancer (HNPCC)

Protective factors

1) SMOKING, because nicotine blocks estrogen receptors
2) OCP 3) Parity

Screening

*Pap smear * TV endometrial thickness >9mm

Types

Type-I (estrogen-dependent)

Endometroid type (adenocarcinoma)/ mean age 63-year/ 5-year survival (85%)/ good prognosis/ MORE common.
↳ Usually begin as hyperplasia & progress to CA.

Type-II (non-estrogen dependent)

Can be papillary serous (worst) OR clear cell type/ age (67-year)/ 5-year survival (58%)/ poor prognosis, less common.
↳ May develop after radiation for cervical CA.

Symptoms

Postmenopausal bleeding (the most common symptom)

Diagnosis

》Depending on the endometrial thickness on the TV U/S:
- < 5 mm: Evaluated after 5 months
- >5mm: D&C and biopsy: If the endometrial biopsy reveals endometrial cancer, definitive treatment can be arranged

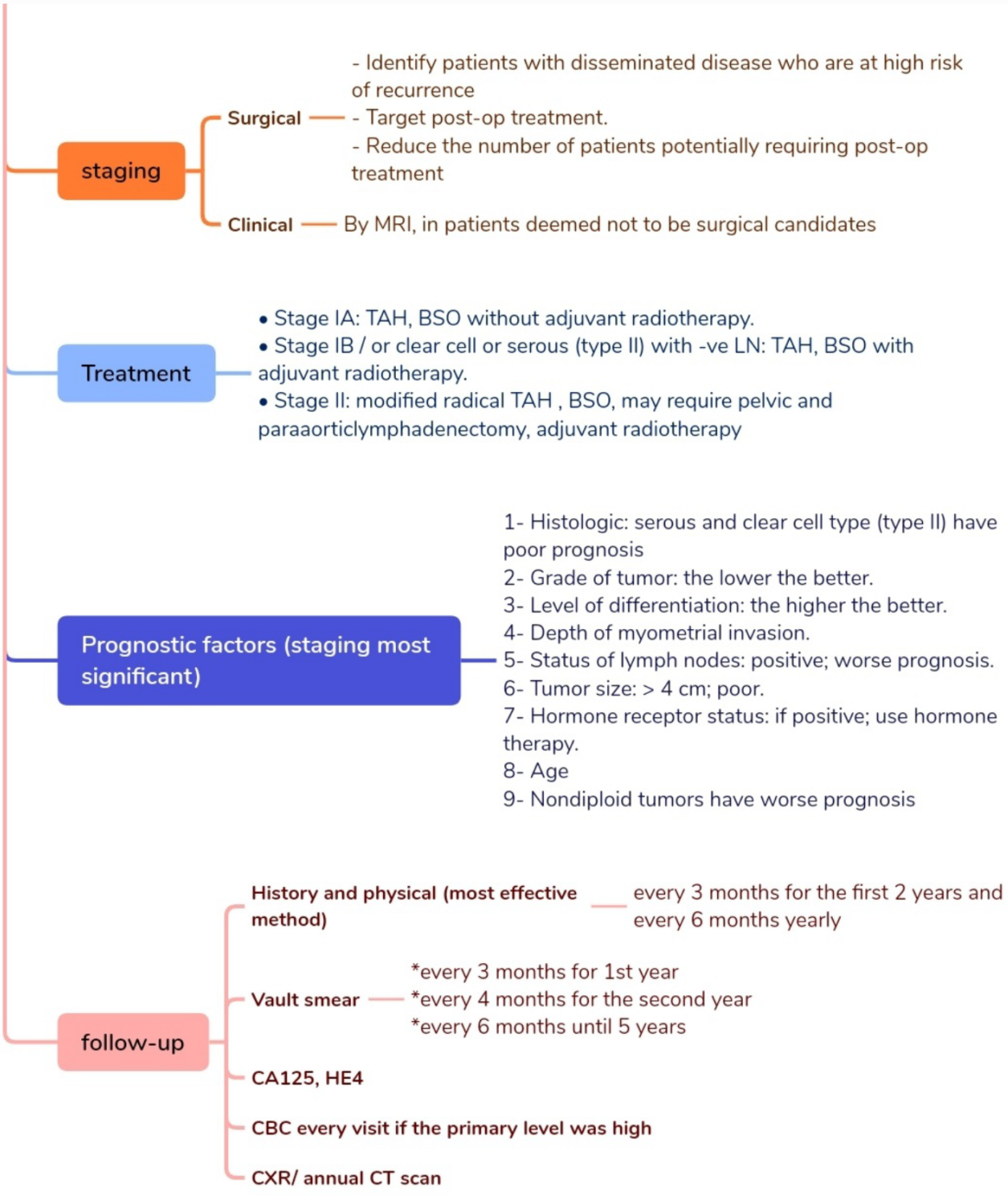
Investigations

Routine — CBC/ KFT/ LFT/ ECG/ Urinalysis/ CXR

Non-routine (Mostly in type 2) — Pelvic US/ MRI (Best)/ CT pelvis & abd./ PET/ CA125

Pattern of spread

1-Direct extension. (Most common)
2-Transtubal
3-Lymphatic dissemination.
4-Hematogenous dissemination. (Worst)
- Primary staging is final, if we diagnose a pt. with stage I and we do resection if she comes back with liver metastasis & histopathology shows it's the same type of resected tumor, it's still stage I.


staging
Surgical
- Identify patients with disseminated disease who are at high risk of recurrence
- Target post-op treatment.
- Reduce the number of patients potentially requiring post-op treatment
Clinical
By MRI, in patients deemed not to be surgical candidates
Treatment
• Stage IA: TAH, BSO without adjuvant radiotherapy.
• Stage IB / or clear cell or serous (type II) with -ve LN: TAH, BSO with adjuvant radiotherapy.
• Stage II: modified radical TAH , BSO, may require pelvic and paraaorticlymphadenectomy, adjuvant radiotherapy
Prognostic factors (staging most significant)
1- Histologic: serous and clear cell type (type II) have poor prognosis
2- Grade of tumor: the lower the better.
3- Level of differentiation: the higher the better.
4- Depth of myometrial invasion.
5- Status of lymph nodes: positive; worse prognosis.
6- Tumor size: > 4 cm; poor.
7- Hormone receptor status: if positive; use hormone therapy.
8- Age
9- Nondiploid tumors have worse prognosis
follow-up
History and physical (most effective method)
every 3 months for the first 2 years and every 6 months yearly
Vault smear
*every 3 months for 1st year
*every 4 months for the second year
*every 6 months until 5 years
CA125, HE4
CBC every visit if the primary level was high
CXR/ annual CT scan

Cervical CA

The mean age is about 52 years, with two peaks : 35-39 y/60-64 y
• Squamous epi→ covers the outer rim of the cervix, columnar→ The inner region. The junction between them is called the original squamocolumnar junction.
• 99% of cervical cancer patients are HPV positive
↳ types 16 and 18 are responsible for 70% of cervical cancers.
↳ Persistent infection with the low-risk types 6 and 11 have been associated only with cervical condylomas and low-grade cervical intraepithelial neoplasia (CIN)

Screening — pap smear (no test is done in symptomatic patients)

Types
• Squamous Cell Carcinoma: Originate from the transitional zone, most common (90%), associated with HPV infection
• Adenocarcinomas: associated with both HPV infection & DES exposure
• Small cell cancer : the worst type , rarely occur
• Large cell cancer : has better prognosis than the Small cell type

Development of Cervical CA
1. Infection of the metaplastic epi of the transformation zone by Oncogenic HPV
2. Viral persistance rather than clearance
3. Clonal progression into precancerous state (Carcinoma in situ)
4. Invasion through basement membrane ⇒ Carcinoma

Risk factors
- Young age at first coitus (<17)/ Young age at first pregnancy
- High parity
- Use of OCP's
- Multiple sexual partners increase the risk of STD's
- Promiscuous sexual partner→partner with multiple sexual partners
- A male with penile condyloma acuminate
- Lower socioeconomic status (developing countries)/ Smoking/ Immunodeficiency

• Note that it's NOT linked to the FREQUENCY of sexual activity NOR age at menopause or family history

Prevention
》PRIMARY PREVENTION: VACCINATION [HPV immunization]
》SECONDARY PREVENTION: SCREENING: By Speculum & vaginal exam, including a Pap smear, by age 21:
• 21 -30 yrs: Annually • >30 years: every 3 yrs

Symptoms
- Abnormal vaginal bleeding is the 1st symptom; usually post-coital bleeding
↳ that's why in pts who are not sexually active bleeding usually does not occur until the disease is quite advanced.
- Persistent malodorous vaginal discharge.
- Pelvic pain
- Dysurea

PE

- Cervix may be ulcerative. It bleeds on palpation (highly vascular and friable)
- Speculum & Bimanual exam
- Rectovaginal exam: Determine extent of disease: Diameter of the primary cancer, spread to parametria & extension into uterosacral ligaments.

Imaging studies

- US ⇒ Assess tumor size & local extent of disease
- Intravenous pyelogram
- CXR/ CT abd & pelvis/ MRI/ PET

definitive diagnosis

Biopsy

Patterns of Spread

- Direct invasion
- Lymphatic spread pelvic lymph nodes and then para-aorta LN
- Hematogenous spread lungs, liver, and bone.

Management

Colposcopy/ D&C/ Cystoscopy/ Urine cytology

Staging

The only cancer stages clinically (based on PE and non-invasive testing).

- Stage 1A: Total simple hysterectomy
- Stage 1B: Radical hysterectomy +pelvic & para-aortic lymphadenectomy
- Stage 2A: Radical hysterectomy +pelvic & para-aortic lymphadenectomy
- Stage 2B/ 3A-3B/ 4A: Chemoradiotherapy

Complications of surgery

- Most common of radical hysterectomy→ Urinary disfunction
- Most serious → Retrovaginal fistula

Contraception
'prevention of conception'

Advantages

1. Decrease un-intended pregnancies and abortions.

2. Provides health & social benefits for mother and children.

3. Decrease risk of postpartum depression, decrease unwanted pregnancies.

4. Therapeutic benefits for Heavy menses, Acne, hirsutism, endometriosis, decrease risk of endometrial and ovarian cancer.

Emergency contraception

Drug or device used after intercourse to prevent pregnancy⇒
1) Pill containing a progesterone receptor modulator, taken within 5 days of intercourse
2) Progesterone, taken within 3 days of intercourse
3) Cupper IUD, within 5 days

Notes

Efficacy of contraceptive method = pregnancy rate per 100 women per year.

Methods

1) Natural - Least effective one

1) Periodic abstinence
Rhythm or calendar method: Fertility awareness & abstinence shortly before and after ovulation period.
↳ Effectiveness : 50-80%.
~ Ovulation assessment method:
1. Ovulation prediction kits (detects LH surge)
2. Basal body temperature
3. Cervical mucus evaluation.
》 Advantages: NO Use chemical/ mechanical barriers.

2) Coitus interruptus
Withdrawal of penis from the vagina before ejaculation, so the majority of semen is deposited OUTSIDE the genital tract.
↳ Effectiveness: 27%.
》 Disadvantages: High failure rate/ Needs self control.

3) Lactational amenorrhea
Prolactin – induced inhibition of GnRH from hypothalamus ⇒ suppression of ovulation.
· Criteria:
The infant must exclusively breastfeed, only for 6 months, breastfeed at least 4hrs/day and 6hrs/night.
》 Advantages: No cost / No effects on nursing
》 Disadvantages: Actual efficacy rate is low

2) Barrier methods and spermicides
~ Only methods protect against STDs
~ Spermicides⇒Creams, gels, suppositories, acts as a mechanical barrier.
· MOA: Disrupts cell of spermatozoa.

✓ Male condoms
↳ Effectiveness: increased by spermicides 85-90%
》 Adv: low cost / No STDs except for HPV and HSV.
》 Disadvantages: Decrease sensation/ May rupture/ Hypersentivity from latex

✓ Female condoms
Must not be removed for 6-8 hrs after intercourse.
↳ Effectiveness is 80%
》 Adv: No STDs except HPV & HSV/ Self-induced

✓ Diaphragm
Dome-shaped, placed into vagina before intercourse
& left placed 6_8 hrs after it.
↳ Effectiveness = 80%
》 Disadvantages: Inserted by clinician/ Hypersentivity to latex
》 Complications: Risk of vaginal tract injuries/ Colonization of staph, leads to toxic shock syndrome

✓ Cervical cap: Silicon cap fits directly over cervix.
↳ Effectiveness= 80%
》 Disadvantages: Inserted by clinician/ Dislodgment

3) IUD

- Most widely used method of Reversible contraception.
- Types: 1. Paragard (Copper) 2. Mirena (progesterone–only)
- MOA: Cause sterile inflammatory reaction / prevent implantation
/decrease tubal motility / increase cervical mucus thickening.
  ↳ Effectiveness: Paragard 99.1% / Mirena 99.9%.

》 Advantages
- Long term contraception: copper used for 10yrs /Mirena for 5
- Cost-effective
- Early reversibility
- Can be immediately inserted after spontaneous abortion in the 1st trimester.
》 Disadvantages : Risk of expulsion (1st year) / Inserted by physician / Pain, bleeding, infection / Perforation at time of insertion.

Indications
· When OCPs are contraindicated
· Long Term protection
· Low risk of STD
· Menorrhagia/ dysmenorrhea

Contraindication
· Absolute: Pregnancy/ Bleeding/ Infection / Copper / Wilson disease / Molar pregnancy
· Relative: Previous history of pregnancy / Previous history of STD in 3 months / Anomalies/ Fibroid / Nullipara

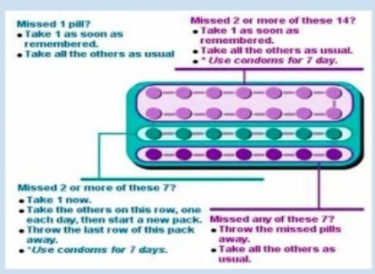
4) Surgical Sterilization

■ Tubal ligation
- Surgically excluding of fallopian tubes.
- Immediately effective
- Will not affect the menstrual pattern
- Low risk of pregnancy but if it happens, it will be Ectopic.
- Procedure methods: laparoscopy/ laparotomy/ through hysteroscopy

■ Vasectomy
- Ligation of vase deferens, done in the office, under local incision in the upper outer aspect of each scrotum.
- Not immediately effective (needs 6-8 weeks)
 ↳ so patient should use another form of contraception till azoospermia is confirmed by semen analysis (3 times)
- Safe, simple, cheap more affective.

5) Hormonal

Combined (Estrogen+ Progesterone)



○ MOA:
1) Interfere with the release of FSH and LH (causes pseudo pregnancy state) that suppress the ovulation.
2) Thickening of cervical mucus
↳ Effectiveness: 99.8% administered 1*1 , 21 days then 7 days break

○ Side Effects:
✓ Estrogen related
· CVA/ MI/ PE/ DVT
· MIGRAINE/ headache/ tiredness
· Fluid retention/ bloating / Breast changes (tenderness, enlargement)
· Loss of Libido, cervical CA.

✓ Progesterone related
· Breakthrough bleeding/ irregular bleeding
· Acne/ baldness/ weight gain
· Irritability & Depression / HTN
· Cholestasis

1) OCPs
~ ⬆️ risk of cervical and breast carcinoma
⬇️ risk of ovarian, endometrial and colon cancer.
~ Uses (other then contraception)⇒ Dysmenorrhea, Benign simple ovarian cyst, Dysfunctional uterine bleeding, Endometriosis.

Contra-indications
■ Absolute
- Smoker >15 cig/day & age >35.
- VTE, PE, CAD, CVA (Venus risk more than arterial risk)
- Uncontrolled HTN, HTN with vascular disease.
- Known or suspected pregnancy, lactating, breast CA.
- Migraine with aura.
- Abnormal LFT/ endometrial CA/ SLE / Undiagnosed vaginal bleeding.
■ Relative contraindications
- Smoker <15 cig/day & age >35.
- HTN/ hyperlipidemia/ DM with vascular diseases.
- Lactating less than 6 months.

2) Transdermal patches
~ Continuous release of Ethinyl estradiol + progesterone.
~ Effectiveness >99% BUT decrease in Over weight 🚺.
~ 1 batch per week for 3 weeks then 1 week withdrawal bleeding
~ Many causes skin irritation.

3) Vaginal ring:
~ Release daily doses of Ethinyl Estrogen+ Progesterone.
~ Effectiveness is 98%
~ Placed in the vagina for 3 weeks then removed for 1week for withdrawal bleeding.
》 Disadvantage: inserted by clinician, discomfort, headache, vaginal discharge/ recurrent vaginitis.

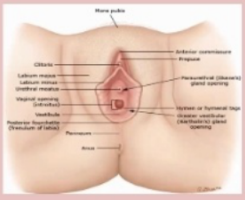
· Progesterone ONLY methods

✓ Minipills (POPs)
- Lower dose of progestin than in combined.
- Effectiveness = 92%, higher failure rate.
- Administration 1 * 1 for 28 days.
- MOA: cervical mucus thickening, ovulation suppression, endometrial atrophy
- Indications: When combined OCP are contra-indicated & lactating mothers.

✓ Injections = Depo-Provera:
- Administration: IM every 3 months
- Most effective 99.7%
》 Disadvantages: Amenorrhea, may cause infertility as long use suppresses the ovulation, after D/C of injections they experience delayed in ovulations (6-8 months)

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

Malignant & Benign of Vulva



Vulva

Located between genito-crural folds laterally, the mons pubis anteriorly, and the anus posteriorly.

It contains labia majora, labia minora, clitoris, vestibule, urinary meatus, vaginal orifice, hymen, bartholin glands, skene ducts.

Early examination & Early biopsy & do NOT start empirical therapy ⇒ Most important

Benign lesions

Non-neoplastic Epithelial disorders

1. Lichen simplex chronicus

Local thickening of the epithelium (hyperplasia), due to prolong itchy.

》Symptoms: pain & itchy in the absence of underlying dermatosis.

》 Signs: White plaques or darker red areas on keratinized skin with a leathery raised surface.

》 Treatment: Intermediate potency topical corticosteroids.

2. Lichen sclerosus

Most commonly found in the anogenital area of Midlife women.

》Symptoms: Intense pruritis, dyspareunia, burning pain, painful bleeding fissures.
↳ Involvement of the anal sphincter ⇒ constipation.
- It starts as isolated pearly white papules & plaques that coalesce & form scars.

》 Signs: White thin & inelastic skin with tissue paper appearance, shrinkage labia Minora, buried clitoris, contraction at the vestibule, scarring around the anus.

》 Diagnosis: Skin biopsy → Shows loss at rete ridges, Atrophic epithelium, Inflammatory cells.

》 Treatment: High potency corticosteriods.

3. Lichen planus

Inflammatory autoimmune process that involves the vagina, vulva &/or mouth.

》Symptoms: Burning, irritation, dyspareunia.

》 Signs: Erythema, erosions on the vulva surrounded by white striae.

》 Treatment: Topical & systemic steroids.

4. Vulvovaginal atrophy

- **Benign tumors (Solid or cystic masses)**
  - **Epidermal inclusion cysts**
    - Non-tender, Mobile, Slow growing lesions
    - Form in obstructed hair follicles
    - Maybe left untreated OR deflated by expressing it's contents (sebum & epithelium)
  - **Bartholin & skene's duct cysts**
    - Bartholin cysts ⇒ present below the hymenal ring at 4 or 8 o'clock.
    - Skene's cysts ⇒ present in the para-urethral near the distal urethra, may cause dyspareunia or urinary tract obstruction.

    》 Treatment if the cyst causes discomfort (walking disturbed or can't sit down) or if there's a change in the site or character at the cyst.

    ↳ treatment , of Bartholin in particular, → Marsupialization or Excision (if you suspect Malignancy OR Recurrent pt. OR Elderly)
  - **Genital warts**
    - Caused by HPV, usually HPV 6 & 11
    - Present at posterior fourchette & lateral vulval walls
    - Treatment → Excision of warts & Antiviral therapy
  - **Hidradenitis suppurativa or acne inversa (inflammation at apocrine glands)**
  - **Urethral caruncle → usually seens in elderly**
  - **Vascular lesions (varicosities, angiomas, hematomas)**
- **Dermatologic disorders** — Psoriasis / Behcet's syndrome / Crohn's disease / Acanthosis nigrincans / Eczema
- **Hamartomas & cysts**
- **Infectious disorders**

- **Malignant**
  - **Vulval intra-epithelial neoplasia**
    - **Squamous**

      Include:
      》Bowmen's disease (Squamous cell carcinoma in situ = VIN grade 3)
      - Mean age= 45
      - 50% Asymptomatic.
      - Elevated lesions, red, brown ⇒ Need biopsy
      - Local superficial excision with margins of 5mm OR skinning vulvEctomy in extensive lesions.
    - **Non-squamous**

      Include:
      1》Paget's disease (Adenocarcinoma in situ = VIN grade 3)
      - Occur in white postmenopausal, may occur in the nipple area of the breast.
      - Itching & tenderness
      - Well demarcated & eczematous, may progress to the mons pubis, buttocks, and thighs.

      2》Non-invasive tumours of melanocytes

Vulval neoplasm
- Uncommon 5%
- 30% of cases of lymphatic metastasis is present (inguinal LN)

Squamous (most common)

- Symptoms: valval Lump or Ulcer, long-standing pruritis (lichen sclerosus)
- Signs: Raised, ulcerated, pigmented lesion.
- Site (More common): labia majora & labia minora

Stage 1 = Acting safely
Stage 4 = Very advanced

Management
》Stage 1a = Radical local excision
》Stage 1b & Stage 2 = Radical local excision + ipsilateral inguinal & femoral lymphadenectomy / Bilateral groin dissection if the lesion is in the midline.
》Stage 3 = Pre-operative radiation or chemo + Radical vulvectomy + Bilateral groin dissection

#pt. with NO lymph node involvement = Good prognosis

Melanoma (2nd most common)

~ May arise denovo or from a pre-existing nevus
~ Commonly involves labia minora or clitoris
~ Occurs in postmenopausal white women
~ Small lesions, tend to metastasized EARLY

Diagnosis — Excisonal biopsy

Prognosis — depend on the depth of penetration into the dermis

Management
- Superficial lesions = Radical local excision
- Deeper lesions 1mm or more = Radical local excision + ipsilateral inguinal femoral lymphadenectomy

Adenocarcinoma

Sarcoma

Diseases of the vagina

Conditions affecting the vulva (lichen sclerosis, eczema) do NOT affect the vagina

Genital infections [Microbiological swabs will confirm the diagnosis]

- Bacterial vaginosis
- Candida albicans
- Trichomonas vaginalis

Erosive lichen planus

- Autoimmune inflammatory skin condition
- Causes vaginal pain & inflammation, if untreated → vaginal stenosis
- **Treatment** — vaginal trainers & intravaginal steriods

VAIN (Vaginal Intraepithelial Neoplasia)

- An extension of cervical intra-epithelial neoplasia
- Asymptomatic
- Less recurrence
- **Treatment** — Cauterization / Excision/ Radiotherapy / Expectant (if the pt. is very old, or the size is very small)

Vaginal CA [progression is usually local]

- Absence of symptoms in early stages
- Advance stage= Bleeding & Discharge & Pain (very late symptom due to infiltration in pelvic nerves), rectovaginal & vesicovaginal fistulae
- Diagnosis — **vaginal biopsy**
- Radio & chemo 1st line treatment