GYNECOLOGY

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❖ GYNECOLOGY

❖ Contents:

❖ Contraception

- International prevention of conception.

➢ Methods:

- Natural Methods
- Barrier methods and spermicides
- IUCD
- Hormonal methods
- Surgical sterilization

➢ 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)

➢ 1. Natural methods – the least effective one

- **A- Periodic abstinence**: rhythm or calendar method MOA: It emphasizes **fertility awareness and abstinence shortly** before and after ovulation period. Effectiveness :relatively low **(50-80%).**
- **ovulation assessment method**: 1.ovulation prediction kits(detects LH surge) 2.Basal body temperature 3.cervical mucus evaluation.
- Advantages :Uses neither chemical nor mechanical barriers.

B-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.

C- 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.

- **Advantages** :1.No cost 2.No effects on nursing
- **Disadvantages** :Actual efficacy rate is low.

2. Barrier methods and spermicides :

MOA :**mechanical obstruction/**The only methods that protect against STDs male and female condoms.

A. **Male condoms:**

Effectiveness : **increased by spermicides 85-90%**

Advantages :low cost /**No STDs except for HPV and HSV.**

Disadvantages : 1.decrease sensation 2.hypersentivity from latex 3.may rupture

B. Female condoms:. Must **not be removed for 6-8 hours** after intercourse.

Effectiveness is 80%

Advantages :1.No STDs (except HPV and HSV) 2.Self-induced

C. Diaphragm: Dome-shaped. Placed into the vagina before the intercourse and left placed 6-8 hours after it. **Effectiveness** :**80%** .

Disadvantages :1. inserted by clinician 2.hypersentivity to latex

Complications :1. risk of vaginal tract injuries 2.colonization of staph, leads to toxic shock syndrome.

D. Cervical cap: Silicon cap that fits directly over cervix .Effectiveness :**80%**

Disadvantages : 1.Inserted by clinician 2.Dislodgment

- **Spermicides** :used adjunct with other barriers Forms: Creams, gels, suppositories. Acts as a mechanical barrier.
- MOA:-disrupts cell of spermatozoa.

3. IUD

- The **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 and increase cervical mucus thickening. 0
- Effectiveness : - Paragard 99.1% while **mirena 99.9%.**

Advantages are :

Long term contraception : **copper used for 10 years** /Mirena used for 5

cost-effective / early reversibility / can be immediately inserted after spontaneous abortion in the first trimester.

Disadvantages : Risk of expulsion (1st year) / Inserted by physician / Pain, bleeding and infection / Perforation at time of insertion.

Indications:

- ✓ When OCPs are contraindicated
- ✓ Long Term protection
- ✓ Low risk of STD
- ✓ Menorrhagia/ dysmenorrhea

Contraindication:

• Absolute : Pregnancy/ bleeding/ i**nfection** / Copper / Wilson disease / **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 :** OCPs: (estrogen+ progesterone)

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

✓ Effectiveness: 99.8% administered 1*1 , **21 days**

✓ **Side Effects :**

✓ **Estrogen related:**

- **CVA/ MI/ PE/ DVT**

- **MIGRAINE/ headache and tiredness**
- **Fluid retention/ bloating /Breast changes(tenderness, enlargement)**
- **Loss of Libido, cervical CA.**

✓ **Progesterone related:**

- **breakthrough bleeding/ irregular bleeding**
- **acne/ baldness/ weight gain**
- **irritability and depression /hypertension**
- **cholestasis**

❑ **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/ 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**.**

- ✓ Treated breast CA more than 5 years without recurrence.
- ✓ Obesity (IBM I more than 35)
- ✓ Migraine without aura

- **Transdermal patches:**

✓ 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% o placed in the vagina 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 OCB are contra-indicated and **lactating mothers.**

2-Injections = Depo-Provera:

✓ 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. After D/c of injections, **they experience delayed in ovulations (6-8 months)**

3-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

➢ **Surgical sterilization:**

➢ **Tubal ligation :**

✓ Surgically excluding of fallopian tubes.

✓ low risk of pregnancy but if it happens, it will be ectopic

➢ **Vasectomy** :

✓ Ligation Of vase deferens Done in the office under local incision in the upper outer aspect of each scrotum.

✓ Unlike tubal ligation it is not immediately effective (needs 6-8 weeks) so patient should use another form of contraception and till azoospermia is confirmed by semen analysis (3 times) Safe, simple, cheap more affective

➢ Menstrual cycle

- ✓ Menstrual cycle it's 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 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

➢ Premenstrual syndrome

- 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 3 or more consecutive cycles.
- Absent pre-ovulatory.
- Present only post-ovulation (luteal phase)
- Interfere with normal function.
- Resolve with onset of menses.

- **PMS and PMDD both have similar symptoms, but PMDD has markedly severe symptoms. The symptoms of PMS do not impair daily activities; however, the symptoms of PMDD do affect the activities of daily living.**

- **Symptoms:**

  - ✓ Fluid retention; breast tenderness, extremely edema, weight gain and bloating.
  - ✓ Emotional nervous; nervous tension, mood swings, depression, irritability, anxiety
  - ✓ 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 salt.

• Lifestyle: relaxation technique and regular exercise.

• Medication: **SSRI (treatment of choice; 1st line)** ex: fluoxetine, and OCPs.

❑ Dysmenorrhea

Pain with menses gone after menses with absence of pain, generally, between menstrual periods.

- **Primary: between 17 and 22 years.(2 years of menarche)**

  - ✓ recurrent, ***crampy*** lower abdominal pain along with **nausea vomiting and diarrhea** that occurs during menses in the absence of pelvic pathology.

- Onset of symptoms: begins several hours prior the onset of menses and continue 1-3 days.

- Pathophysiology: has been attributed to **uterine contractions**(they have increased uterine activity, increase resting tone, increased contractility and frequency of contractions) with **ischemia and progesterone** production.
- Risk factors: early menarche, decease parity, diet, exercise, heavy smoking and psycho.

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

➢ Secondary:

- ✓ recurrent dull aching lower abdominal pain usually without nausea and vomiting and diarrhea
- ✓ Etiology: endometriosis; others: adenomyosis, chronic PID, fibroid, surgeries, trauma and IUCD
- ✓ Clinical features:
  - Develops in older women**,(30-40 years)** and less related to the first day of the flow.
- ✓ May have associated symptoms; such as dyspareunia, infertility or abnormal uterine bleeding

❑ Fibroids (leiomyoma)

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)

- **Risk factors**:

• Black, obese, non-smoker women

• Perimenopausal, nulliparity (or old age at first pregnancy) • Family history

- **50-60% asymptomatic**
- If symptomatic, the **most common symptom is bleeding(Menorrhagia)**
- **Pain**: Acute infarct, dysmenorrhea.
- **Pressure symptoms**: GUT: frequency/retention /hydronephrosis. GI: Bloating /constipation /rectal pressure.
- **Obstetric problems**: Infertility, recurrent abortions, preterm labor, placenta previa, abruption, malpresentation, IUGR, APH, PPH, C/S

❑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, metro menorrhagia)

3. **Sub serosal:** • Just below the serosa / peritoneum /if very large can **cause pressure symptoms/**If connected with a stalk they are called pedunculated

4. **Parasitic:** • Originally, they are pedunculated sub serosal, the stalk becomes necrotic and breaks away from the uterus and receives its blood supply from abdominal organs

5. Cervical

6. Interligamentous • It grows laterally into the **broad ligament**

- **P/E:**

- ✓ General: pallor, vital signs (tachycardia).
- ✓ Abdomen: increase in fundal height, mass.
- ✓ Bimanual: localized, **non-tender, irregular mass** or cobble stone.

➢ **Investigations:**

- The most common diagnostic method: U/S
- **MRI (to differentiate it from adenomyosis)**
- D&C, biopsy, hysteroscopy(definitive).
- ***The most definitive diagnosis : biopsy***

➢ Treatment :

• **No treatment if asymptomatic**(observation and follow up by serial PV/ultrasound)

• **Medical :**

- ✓ Pre surgical shrinkage: **GNRH analogues** decrease the size by **70%**, **given 3-6 months before surgery**, re-growth after stopping (not used for definitive treatment just before surgery)
- ✓ Invasive radiation (embolization): **uterine artery embolization**, preserves uterus, but you don't preserve fertility.

• **Surgical :**

- ✓ **Myomectomy** : preserves fertility ,laparoscopy/laparotomy

(**1/3 of fibroids recur following myomectomy)**

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

Histology : it has a **psudocapsule** of compressed smooth muscle

cells (contain a few blood vessels and lymphatics)

➢ **Degeneration of the fibroid :**

• **Hyaline (most common)**

• **Red /hemorrhagic (most common in pregnancy)**

➢ **Management of fibroid degeneration during pregnancy :**

• Bed rest

• analgesia

• No surgery

❑ Adenomyosis

✓ It is the extension of endometrial **glands and stroma** into the **uterine musculature** more than 2.5 mm beneath the basalis layer.

✓ About 15% of patients with adenomyosis have associated endometriosis.

✓ often it is an incidental finding during a pathologic examination, where it is seen in up to 60% of women in their **40s.**

✓ Risk factors : D&C/ multipara

✓ Mostly **asymptomatic**

✓ If symptomatic : most common symptoms are **secondary dysmenorrhea and menorrhagia**

✓ Approach

- uterus is **globular and diffusely enlarged**
- Tenderness is immediately before and during menses

- Pelvic examination reveals the uterus to be generally symmetrically enlarged.
- U/S and MRI (to differentiate between fibroid and adenomyosis)
- **Biopsy (the only definitive diagnoses is through histology)**

➢ **Treatment :**

❑ Medical :

✓ levonorgestrel IUD (synthetic progesterone) /OCP

✓ NSAIDS and GNRH agonists(for pain and bleeding)

❑ Surgical : **Hysterectomy (definitive)**

Leiomyoma vs	**Adenomyosis**
Firm	**Soft**
localized	**Diffuse**
Non-tender	**Tender**
Pseudo capsule	**True capsule**

➢ Endometriosis

- Endometriosis: is a **benign condition** in which endometrial glands and stroma are present **outside the uterine cavity and walls.**
- Endometriosis respond cyclically to ovarian steroidal hormone production. The implants proliferate under estrogenic stimulation and slough when support from estrogen and progesterone is removed
- At least 1/3rd of women with **chronic pelvic pain** have visible endometriosis.

- Endometriosis typical patient: female in her 30s, nulliparous, and infertile.
- **Risk factors:**
  1. Family history
  2. Race
  3. Autoimmune diseases

➢ **Endometriosis pathogenesis:**

- Genetic predisposition clearly plays a role.
- **3 Hypotheses:**

1- The **retrograde menstruation theory of Sampson**: **most accepted one** .Which proposes that endometrial fragments transported through the fallopian tubes at the time of menstruation implant and grow in various intra abdominal sites.

2- - The Mullerian Metaplasia theory of Meyer.

3- - The lymphatic spread theory of Halban

☒ 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)

4. Rectosigmoid

☒ Endometriosis symptoms:

* **Triad of: 1- Dysmenorrhea 2- Dyspareunia 3- Dyschezia .**

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

Dyspareunia is generally associated with deep thrust penetration during intercourse and occurs mainly when the cul- de- sac and vaginal fornix are involved.

- If the ovarian capsule is involved with endometriosis, ovulatory pain and midcycle vaginal bleeding often occur.

☒ **Endometriosis signs:**

- Tender, fixed adnexal mass is appreciated on bimanual examination
- The uterus is fixed and retroverted in a substantial number of women with endometriosis.
- rectovaginal exam to feel the **uterosacral nodularity.**
- ovarian implants are associated with significant scarring of the ovary. Histologically, two of four characteristics must be found in the endometrioma specimen to confirm the diagnosis: endometrial epithelium, endometrial glands, endometrial stroma, and hemosiderin-laden macrophages.
- CA-125 serum levels are frequently elevated in women with endometriosis.

☒ **The definitive diagnosis is generally made on the basis of the characteristic gross and histologic findings obtained at laparoscopy. (biopsy of any suspicious lesions improves diagnostic accuracy).**

☒ **Management**

Different modalities of treatment can be used depending on the age, severity and previous trials of treatment.

A- **Medical tx: to decrease the pain, not definitive management**

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

- Pseudo pregnancy state: COCP, Progesterone

- Pseudo menopause state : GnRH agonist

- High androgen, low estrogen state: Aromatase inhibitors (Inhibit the conversion of androgens to estrogens at the tissue level

B- Surgical tx

- **Adhesion lysis and excision of the endometrial implants**
- **Definitive Mx is : TAH with BSO(removal of the uterus and ovaries)**

☒ **Endometriosis and infertility:**

- 40-60% of females with endometriosis are infertile
- Due to :

1. Adhesions

2. Dyspareunia

3. High PG levels (Affect tubal motility, and Corpus Luteum Function)

4. increased macrophages (engulf sperms !)

5. High levels of prolactin in 10% of cases

- **Tx of infertility in endometriosis is assisited reproduction :IVF/IUI trial.**

❑Hirsutism

- The presence of terminal (coarse) hair in females in a male like pattern
- where vellus hair is transformed into terminal hair under the influence of androgens.
- **Causes:**

1. PCOS, androgen secreting tumor.
2. Hypothyroidism, Cushing, CAH, increase in prolactin.

3. Exogenous: drugs.

4. Idiopathic.

- ❑ **Investigations:**

- ✓ We order prolactin, TSH and free testosterone then if

- Normal testosterone : 5 alpha reductase overactivity /idiopathic.

- 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).

- ❑ **Management:**

• Lifestyle modification (decrease weight).

• Idiopathic: cosmetic (laser/ waxing/ electrolysis)/ Finasteride (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) and

6 months (if irregular).

❑ Primary Amenorrhea

- 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)

❑ Causes: either anatomical or hormonal

1- Anatomic:

• Imperforate Hymen

• Vaginal Agenesis/Septum

• Müllerian agenesis(Mayer-Rokitansky-Küster-Hauser (MRKH))

2- Hormonal:

• Gonadal Dysgenesis (Turner Syndrome)

• Androgen Insensitivity

• Hypothalamic-Pituitary Insufficiency

❑Approach:

✓ First of all we take history , do P/E then we go for U/S

✓ We look for the uterus and ovaries

- A) If there is uterus , then we order hormonal profile : FSH,LH

▪ If LH,FSH **are low** then the cause is : Hypothalamic-Pituitary Insufficiency

- If LH,FSH **are high** then we **order karyotyping** :

XX: premature ovarian failure

XO: Turner syndrome

- B) if there is NO uterus seen by U/S, we confirm this by MRI

✓ Then we order karyotyping with testosterone level

XX: female with Mullerian agenesis

XY: androgen insensitivity

☒ **Secondary Amenorrhea**

Causes:

✓ PREGNANCY (most common cause) :rule out by (B-HCG)

✓ ANOVULATION (progesterone is missing):PCOS, high prolactin

✓ Estrogen deficiency: Premature ovarian failure ,Sheehan Syndrome

✓ Outflow obstruction : Asherman Syndrome or Cervical Stenosis

☒ **Approach:**

✓ History and physical examination

✓ B.hCG level to rule out pregnancy

✓ Progesterone Challenge Test (PCT): we give her progesterone for 5 days then

- If there is bleeding: the cause is **anovulation**
- If there is **no bleeding** ,we Give estrogen and progestin (Estrogen Progesterone challenge test (EPCT)):
  - if there is bleeding ,the cause is :**low estrogen**
  - if there is no bleeding , the cause is: **outflow obstruction**

❑ Abnormal uterine bleeding (AUB)

- Any deviation from normal bleeding (menses)
- **Menstrual abnormalities (important)**

✓ **Poly**menorrhagia: Uterine bleeding occurring at intervals of **< 21days**.

✓ **Menorrhagia/ hypermenorrhea**: Prolonged (> 7 days) or excessive (> 80 mL) uterine bleeding occurring at regular intervals.

✓ **Oligo**menorrhea: Uterine bleeding occurring at intervals **> 35 days**.

✓ **Metro**rrhagia: Bleeding occurring at frequent**, irregular intervals.**

✓ Menometrorrhagia: Combination of both menorrhagia and metrorrhagia; uterine bleeding that is prolonged or excessive, frequent, and irregular.

✓ **Dysfunctional uterine bleeding**: Caused by ovulatory dysfunction

❑ **Causes of AUB**

- PALM-COEIN classification system:
- PALM are

✓ Polyps
✓ Adenomyosis
✓ Leiomyoma (fibroids)
✓ Malignancy and hyperplasia

- COEIN are

✓ Coagulopathy
✓ Ovarian dysfunction
✓ endometrial causes
✓ Iatrogenic
✓ Not yet specified

❑ Approach :

✓ Detailed Hx
✓ PHYSICAL EXAMINATION:

• General: BMI/pallor/hirsutism/bruising
• Vital signs
• Head and neck : thyroid
• Abdomen: tenderness/masses/uterine size
• Gynecological exam: PV (bimanual)/ speculum (discharge/laceration/polyps/)

✓ INVESTIGATION:

• Labs: CBC / TSH/prolactin/Estrogen/progesterone/B-hcg

• U/S, D&C with biopsy, laparoscopy

❑ TREATMENT: according to the cause

☒ **Acute Excessive Bleeding :**

✓ A woman who presents with heavy bleeding needs to be assessed for hemodynamic stability

✓ Hospitalization and **transfusion** are generally recommended for women who have severe anemia (hemoglobin ≤7 g/dL) and those who are hemodynamically unstable.

✓ Imaging studies can usually be delayed until the heavy bleeding is controlled. increased risk factors for cancer, biopsy is indicated once the bleeding has been stabilized and her hemoglobin level is normal.

☒ **Medical management:**

A. First-line medical therapy usually involves hormonal manipulations once the bleeding is minimal and the patient is stable.
high-dose progestin-only therapies have been recommended as first-line treatment for acute heavy menstrual bleeding
B. Treat the underlying cause.

☒ **Dysfunctional Uterine Bleeding (DUB)**

- Abnormal uterine bleeding **NOT due to organic** gynecological disease.
- mainly after Puberty/ premenopausal

❑ **Menopause**

❑ It's the **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 of menopause: **51 years**
- ✓ It's usually preceded by premenopausal period (climacteric) which is the transition optimal menstrual condition to menopause
- ✓ **Premenopause** :The period immediately prior to the menopause (when the endocrinological, biological, and clinical features of approaching menopause commence
- ✓ **Climacteric period** : The transition from the reproductive phase to the non-reproductive state
- ✓ **Premature menopause**: Is menopause that occurs at age <40 yr may be caused by **surgical removal of both ovaries** (with or without hysterectomy) or **iatrogenic ablation of ovarian function** (. by chemotherapy or radiation).
- ✓ **Premature ovarian failure** usually 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 age (<30)
- ✓ **Pathophysiology of menopause**

Most women ovulate about 400 times between menarche and menopause and during this time, 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 (decreases) :
2- Estradiol (decreases).
3- Estron increases
4- Androgen the production decreases / receptors became more sensitive
5- progesterone declines to low levels
6- FSH and LH :increase FSH greater than 40

❑SYMPTOMS:

➢ Early symptoms are: Short Term Effects (0-5y):

✓ Amenorrhea(2ry): the most common symptom

✓ Vasomotor symptoms:

- Night sweats
- Hot flashes 85% of women experience hot flashes as they pass through the climacteric, but about half of these women are not seriously disturbed by them.

✓ Psychological symptoms: depression, anxiety, insomnia and irritability.

✓ Loss of concentration and poor memory

➢ Intermediate Effects (5-10y)

✓ Vaginal dryness/Dyspareunia

✓ Sensory urgency/Recurrent UTIs/Urogenital prolapsed/Stress incontinence

➢ Long Term (>10y)

✓ **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 a progestin) shouldn‟t be used as the first line treatment.

✓ **Cardiovascular disease**

- The m.c.c of mortality in 50% in postmenopausal women
- Increase LDL , decrease HDL

✓ **Dementia**

➢ **Investigations**

FSH > 40 IU/L, preferably 2 measurements, 2 weeks.

➢ **Management**

- We have non-hormonal vs hormonal therapy
- Non hormonal : Antidepressant , B blocker
- **HRT :**

✓ Estrogen + Progesterone (to protect the endometrium from unopposed estrogen) or ERT: Estrogen alone (they‟re only indicated in females post hysterectomy)

✓ Minimum effective dose for shortest duration

✓ Average 2-3 years

✓ Indicated in case of Vasomotor symptoms or vaginal atrophy

☒ Ectopic pregnancy

- it's a pregnancy that is located **outside the uterine cavity**. (Not outside the uterus cause cervix implant is an ectopic pregnancy.
- Incidence is increased
- it's one of the leading cause of maternal mortality 6%.
- **Sites are**

1-Fallopian tubes (95%)

Ampulla: most common site, the widest part 5-6 mm

Isthmus: wall is thicker

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%

- **Risk factors**

1-previous history of ectopic pregnancy.

2- **PID (STD) and infection**: Due to -intratubal or peritubal adhesions and 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

6- smoking

7-congenital malformation of the uterus

✓ Deferential diagnosis for first trimester bleeding: abortion / ectopic pregnancy/ molar pregnancy.

✓ **Clinical presentation**

- 4–6 weeks after their last menstrual period.Triad 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

✓ Physical exam

- Bimanual exam :palpate adnexal mass –cervical motion tenderness

✓ Investigation

1-Labs :B-HCG/ To confirm pregnancy

✓ Doubling, in Normal pregnancy HCG after 72 hours

✓ If it is increased without doubling then its ectopic pregnancy.

2-ultrasound

- ✓ -you may see adrenal mass
- ✓ -you may see free fluid in the pouch of doglus
- ✓ -fetal heart in the adnexa

  *Ultrasound findings suggestive of ectopic pregnancy: **Absent intrauterine sac, complex adnexal mass, fluid in cudle sac.**

3-laparoscopy (the definitive Diagnostic):Diagnostic and therapeutics

☒ Management

It 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 FHA, size <4cm

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)

- **Surgical:**

Indications are :

- ➢ failure of medical treatment, unstable, B-HCG>1500, positive FHA, size>4cm.

✓ if stable we do laparoscopy, If unstable we do laparotomy
✓ Salpingectomy (removal of tube)
✓ Salpingostomy (incision on the anti-mesenteric portion of the tube) /higher risk for recurrence

☒ Gestational Trophoblastic Disease

- Group of interrelated disease resulting in abnormal proliferation of trophoblastic **(placental) tissue**

- 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)

- **Common findings**

✓ produce Beta hCG
✓ Extremely sensitive to chemotherapy
✓ The **most curable gynecologic malignancy** and fertility preservation

☒ Molar pregnancy /hydatiform mole

-Types :
1)Complete (classic) 90%
2)Incomplete(partial) 10%

☒ 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
7) history of OCP use
8) blood Group A

- ✓ -HCG subunits are alpha and beta, alpha subunit is also found in LH FSH, TSH (hyperthyroidism)
- ✓ Increase hCG leads to hyperemesis gravidarum and pre-eclampsia

☒ Complete mole :

- ✓ -The majority of hydatidiform moles have a 46,XX karyotype. Both of the X chromosomes are paternally derived. This results from fertilization of an **"empty egg"** by a haploid sperm (23 X), which then duplicates to restore the diploid chromosomal complement (46,XX).
- ✓ -Absent Fetal tissue / Fetal RBCs
- ✓ -Hydropic(swollen) grape like vesicles with Severe hyperplasia
- ✓ **Presentation**: Abnormal vaginal bleeding Is common
  Fundal height large for date /High B-hCG level (more than 100,000)
  -hyperemesis gravidarum (nausea/vomiting)
  -hyperthyroidism symptoms (heat intolerance, diarrhea..)
  -pre-eclampsia symptoms(headache, visual disturbances, epigastric pain, HTN)
- ✓ higher risk for invasive mole 15-25%/ 4% risk for choriocarcinoma
- ✓ Follow up: 14 weeks for B-hCG to become normal

☒ Partial mole:

- ✓ In the "incomplete" or partial mole, the karyotype is usually a triploid, often 69,XXY (80%).
- ✓ These lesions, unlike complete moles, often present with a coexistent fetus. The fetus usually has a triploid karyotype and is defective.
- ✓ Few hydropic with Minimal or no hyperplasia

- ✓ **Presentation** : Missed abortion/ Size of uterus equals dates /Slightly elevated B-HCG
- ✓ 2-4% risk for invasive mole /NO risk for choriocarcinoma
- ✓ Follow up : 8 weeks for B-HCG to become normal

☒ **Diagnosis**

- Ultrasonography is noninvasive and reveals a "snowstorm" pattern that is diagnostic for complete mole
- once evacuated, the definitive diagnosis

❑ Treatment:

- ✓ **Suction evacuation** followed by sharp curettage of the uterine cavity, regardless of the duration of pregnancy is the standard of tx.
- ✓ Patient must be monitored with weekly serum assays of β-hCG until three consecutive levels have been normal. Monthly β-hCG levels should then be followed until three consecutive levels have been normal
- ✓ Patients **should use effective contraception during follow-up**
- ✓ About 95-100% of patients with GTN who have a good prognosis are cured of their disease. Patients with poor prognostic features can be expected to be cured in only 50-70% of cases.
- ✓ The majority of the patients who die have brain or liver metastases.

☒Polycystic Ovarian Syndrome (PCOS)

- Polycystic ovary syndrome (PCOS) is recognized as one of the most common endocrine/metabolic disorders of women.
- The age of onset is most often premenarchal .
- A condition of chronic anovulation resulting subfertility, irregular Bleeding, obesity and hirsutism.
- Criteria for diagnosis: presence of at least 2 of the following criteria, **after ruling out other causes.**

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.

☒ Not all females with PCOS have polycystic ovaries on US and not all females with ovarian cysts have PCOS.

☒ While obesity and insulin resistance are strongly associated with the syndrome, they are not essential to the diagnosis; indeed about half of patients are non-obese.

☒ **High-risk groups** — A number of conditions are associated with an increased prevalence of PCOS:

- Women with oligo-ovulatory infertility
- Obesity / insulin resistance
- Type 1 , type 2 DM
- A history of premature adrenarche
- First-degree relatives with PCOS
- Women using antiepileptic drugs

☒ Signs and symptoms

-Menstrual irregularities (secondary amenorrhea)
-Hyperandrogenism symptoms: hirsutism, acne, obesity, alopecia.
- subfertility
-Obesity
-DM and acanthosis nigricans
-Metabolic syndrome:

☒ Initial evaluation by :

1- History and physical exam then US
2- Laboratory investigations:
TSH, Prolactin ,FSH,LH ,ILGF1 ,lipid profile, Testosterone

☒ Treatment:

- Certain lifestyle changes, such as diet and exercise, are considered first-line treatment / Weight loss reduce hyperandrogenism and insulin resistance
- Pharmacologic treatments for anovulation, hirsutism, and menstrual irregularities.
- Medications for such conditions include oral contraceptives, metformin, clomiphene, and spironolactone.
- Hormonal Therapy : OCPs: The mechanism: Regulate the periods, a significant reduction in hair growth
- 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
- Clomiphene citrate : triggers the brain's pituitary gland to secrete an increased amount of follicle stimulating hormone (FSH) and LH (luteinizing hormone).
  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

☒ Infertility

- Inability to achieve pregnancy **after one year** of regular unprotected intercourse
- **Fecundability** (monthly chance of pregnancy) is 20%
  - Within one year, 85% of pregnancy occurs.
  - Investigations for infertility start after one year

- About 14-16% of couples are involuntarily infertile
- Primary infertility when it occurs without any prior pregnancy (most common type, around 70% of cases).
- Secondary infertility when it follows a previous conception
- Female factors are responsible of infertility in 40% of cases , 30% are because of male factors and a combination of these two are found in 10% of cases. In the remaining 20% of couples, no explanation can be found; their infertility is classified as unexplained.
- **Causes of infertility:**

  ☒ **Female etiology:**

✓ Ovarian:

- Polycystic ovarian syndrome (MCC) 85%

- Premature ovarian failure

Hyper-prolactenemia /Thyroid disorder/Obesity

✓ 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.

✓ Uterine:

- Congenital malformation

- Submucosal fibroid/ Polyps

- Asherman syndrome (adhesions)

✓ Cervical:

- Cervical stenosis/Chronic cervical inflammation

- Mullerian duct abnormality

• Treatment : surgical dilatation, intrauterine insemination (IUI)

☒ Male etiology:

✓ Environmental exposure: smoking, alcohol, excessive heat
✓ Sexual dysfunction: erectile, ejaculation.
✓ Structural factors: varicocele, testicular torsion, vasectomy
✓ Abnormal semen: mumps, anti-sperm antibody
✓ Genetic factors: cystic fibrosis, Klinefelter, immobile cilia.\

☒ Unexplained infertility

Infertility is considered unexplained if : normal semen analysis , confirmed ovulation & patent ducts.

☒ Evaluation:

- History and physical exam

- Investigations (start from the least expensive):

• Abnormal semen

• Anovulation

• Tubal disease: more expensive, invasive, hysterosalpingogram

✓ Semen analysis:

- If abnormal, the first next step is to repeat the test again within 4-6 weeks.

- If abnormal, then we go for intrauterine inoculation (IUI) for fertility.

✓ Anovulation:-

- Investigations: TSH, T4, PRL,, midluteal progesterone, PCT
- Correctable causes: low T4, high prolactin
- If uncorrectable: polycystic ovarian syndrome (PCOS): ovulation induction by clomiphene citrate.

✓ Tubal disease:

- HSG (hysterosalpingogram), if abnormal: consider laparoscopy.
- Tuboplasty: reconstruct damaged oviducts
- Salpingectomy and IVF: if severely damaged

✓ Unexplained infertility:

- First try ovulation induction and IUI anyway, and then IVF.

☒ **Indications for IVF (in vitro fertilization):**

✓ Oligospermia
✓ Irreparable tubes
✓ Unexplained fertility

☒ **Pelvic inflammatory disease (PID)**

- It's an inflammation of the **female upper genital tract** (uterus, tubes, ovaries, ligaments) caused mostly by ascending infection from the vagina and cervix.
- it is **rarely a single organism** that's responsible for PID, organisms include:

✓ **Chlamydia trachomatis (mc)**
✓ **Neisseria gonorrhea.**
✓ **E.coli / Streptococcus**

❑ **Risk factors:**

✓ Age <35 (especially in teens)

- ✓ Multiple sexual partners
- ✓ Unprotected intercourse
- ✓ IUCD (while other contraceptive methods decrease the risk)
- ✓ Nulliparity
- ✓ History of STD

- **Acute vs chronic PID**

❑ **Acute PID**

- Bilateral abdominal **tenderness**, cervical motion tenderness and mucopurulent **discharge.**
- increased number of **WBCs /ESR and positive culture**

❑ **Diagnosis:** It's **mainly clinical** based on a triad of symptoms and signs, including: pelvic pain, cervical motion and adnexal tenderness.

- Other symptoms include: lower abdominal pain, excessive vaginal discharge, chills, direct or rebound abdominal tenderness
- Management: outpatient regimen of Ceftriaxone IM in a single dose PLUS Doxycycline orally twice a day for 14 days unless there is indication for admission .
- The clinical 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. Tuboovarian abscess demonstrated on U/S or suspected clinically
    5. Pregnancy.
- **Sexual partners** of women with PID should be evaluated and treated for urethral infection
- **TUBO-OVARIAN ABSCESS**
- It's an **end-stage** process of acute PID.
- Symptoms: severe bilateral pain /**septic patients**, high fever, elevated heart rate, decreased blood pressure, peritoneal signs, adnexal masses.
- On CT: bilateral complex pelvic masses.

- **Management**: Admit, IV clindamycin + Gentamycin. 75% of women respond to ABs alone. Failure of medical therapy suggests the need for drainage of the abscess.

❑ Chronic PID

- Presentation: chronic bilateral pain, infertility, dyspareunia, ectopic pregnancy, abnormal bleeding
- P/E: cervical motion tenderness and bilateral adnexal tenderness,
- **No discharge, No fever or tachycardia.**
- Investigations: normal WBCs and ESR, -ve culture, on US: hydrosalpinx.
- **Diagnosis is by laparoscopy** by visualization of pelvic adhesions
- Managements: 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.

☒ Vaginal Discharge

❖ Types are:

- Bacterial vaginitis 50%
- Candida vaginitis 30%
- Trichomonas vaginitis 20%

☒ Bacterial Vaginitis (Gardenella)

- Not a true infection, but **alteration in concentration of normal vaginal bacteria (Gardenella>lactobacillus)**
- Sexually assocciated disease and not an STD (no effect with treatment of sex partner)
- Symptoms: "Fishy" odor due to anaerobe, 50 % are asymptomatic.
  - ✓ Speculum exam: PH > 5, NO inflammation, Discharge is homogenous
  - ✓ **Positive "Whiff test"**: Vaginal secretions with KOH: fishy smell.
  - ✓ Wet mount "clue cells": are squamous epithelial **cells** covered with bacteria

- Treatment is only in symptomatic patients: Metronidazole OR Clindamycin. For 7 days.

☒ Candida Albicans Vaginitis:

- **It is STD**
- Symptoms: **severe itching and burning/ white discharge ,cheese like**
- Speculum exam: PH < 4.5 (normal) with Inflammation and White discharge (cheesy) /Wet mount : pseudomyxoma
- Treatment: Oral fluconazole and Azole cream
- Risk factors:- Pregnancy /antibiotics /DM/ Immune suppressants

☒ Trichomonas vaginitis

- Flagellated pear-shaped protozoan
- Can reside asymptomatically in male seminal fluid/**its STD**
- Symptoms: itching and burning
- Speculum exam: **PH > 5 with Green frothy discharge**. **Strawberry cervix** /Wet mount exam: Trichomonas
- Treatment: Metronidazole for both partner

❑ Pelvic Organ Prolapse

- **protrusion** of the pelvic organs into the vaginal canal or beyond the vaginal opening (like a hernia)
- **Causes /Risk factors are :**
- Obesity and chronic cough
- Constipation/ heavy lifting
- Weaning of pelvis:(decrease in connective tissue)
- Age, menopause, HRT
- Multiple vaginal deliveries /**Forceps (most important one)**/macrosomia
- previous history or family hx of pelvic organ prolapse (POP)

✓ Vagina is supported at **three levels**, a defect in any of these levels produces a different type of prolapse according to the herniated organ.

❑ Classification:

✓ **Level 1**: **Apical** support of vagina which is cardinal ligament and uterosacral ligament / any defect in this level : uterine prolapse

✓ The type of apical prolapse that occurs post-hysterectomy is called **vault prolapse** .it occurs due to loss of the integrity of the anterior and posterior vaginal walls.

✓ **Level 2: Anterior :** support vagina by Pubocervical Facia/any defect in this level : Cystocele (Urinary Bladder prolapsed) :MC type

Posterior: either rectocele or enterocele

✓ **Level 3: Perineal membrane** and body/any defect : Deficient Perineum

☒ **Presenting Symptoms**

-feeling of vaginal fullness, heaviness, that often progress over the course of the day and are most noticeable after prolonged standing or straining.

-Other symptoms are related to the herniated part:

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

☒ **Diagnosis:** By vaginal examination

☒ **Quantifying and grading**

- Extent of prolapse is evaluated and measured relative to the hymen
- The plane at the level of the hymen is zero
- Above the hymen : negative number
- Below the hymen : positive number

☒ Grading(assigned 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 the level of the hymen (> -1 cm)
- Grade 2: 1 cm above or below the level of the 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-2 cm)
- Grade 4:Complete eversion of vagina & uterus is outside/ Grade 4 is called **procidentia** & it's the most advanced stage

☒ **Management**

✓ Do not treat asymptomatic prolapsed

✓ **Conservative management**: Kegel exercises (voluntary contraction of pubococcygeus muscles)/Estrogen Replacement Therapy/Vaginal pessaries

✓ **Surgical management** :

- Anterior colporrhaphy for cystocele
- Posterior colpoperineorrhaphy for rectocele
- Vaginal hysterectomy for uterine prolapse
- Sacro colpopexy for vaginal vault prolapse

☒ **Urinary Incontinence**

➢ Involuntary loss of urine that is a social or hygienic problem.

➢ Risk factors same as POP

➢ 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 (increase in intra-abdominal pressure)
- **Two mechanisms of stress urinary incontinence:**

  1-**urethral hyper-mobility (85-90%): most common**/ loss of urine is usually in **small amounts**, and patients **doesn't** usually experience night symptoms

2-**Interensic Sphincter Deficiency (ISD) (10-15%)**/ older in age/ have a more severe symptoms that may occur even at rest with larger amounts

☒ **Diagnosis :**

- **Cough Stress Test:** patient 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 management: Kegel exercises Weight reduction/(-**1st line for young females with mild to moderate incontinence)
- **Surgical: Vaginal approach**(TVT- Tension-freeVaginal Tape): synthetic mesh

☒ **Overactive bladder / urge urinary incontinence (UUI)**:

- Most common type of incontinence in males and second most common type in females
- UUI is the involuntary leakage of urine immediately preceded by urgency.
- **In 90% of patients it's idiopathic. In 10% there's underlying neurological cause.**
- **DIAGNOSIS**: In Urodynamic testing: you will find unstable bladder (uninhibited detrusor contractions) , decreased bladder capacity & strong urinary flow
- **TREATMENT**
- Behavior Modification/reduce fluid intake and avoid liquids during the evening hours + reducing caffeine intake /Kegel exercises, are effective for attaining better bladder control/Weight loss is also important
- Drugs (most commonly used): **Anticholinergics** (gold standard)

☒ **Mixed urinary incontinence(SUI+UUI)**

☒ 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**
- Most common cause in females is **large prolapse after surgery**
- **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

➢Ovarian cysts

- Most of cysts are asymptomatic and discovered incidentally and resolve over time without treatment.
- The aim of their management is **to rule out malignancy** and **to avoid cyst complications** (rupture, torsion, hemorrhage).
- It could be cystic or solid / Most of them are cystic
- **Types are : functional and Neoplastic**
- **Benign ovarian mass** : mobile /soft / smooth surface / unilateral
  Usually < 8cm / Septations Not present /Doppler flow not present

☒ Functional (physiological) ovarian cysts:

1-**Follicular cyst**: develops when an ovarian follicle fails to rupture, can persist over several menstrual cycles and reach 10 cm but usually **resolves after 2-4 months/ common in reproductive age /** most follicular cysts are **painless and harmless**

2-Corpus luteal cyst: develops if the corpus luteum grows to over 3 cm and fails to regress normally after 14 days, thin-walled, fluid filled without septations or calcifications, **smaller than follicular cyst** but more firm and is **more likely to cause pain or signs of peritoneal irritation**. This can develop into:

1-**Hemorrhagic cyst**: form when invasion of ovarian vessels into the corpus luteal cyst.

2-**Theca-luteal cysts**: may develop in association with the **high levels of hCG** present in patients with a hydatidiform mole / choriocarcinoma and patients undergoing ovulation induction with Clomiphene **usually bilateral**.

☒ Benign neoplastic ovarian cyst :

❑ A-Epithelial ovarian neoplasms (most common):

-Derived from the **mesothelial cells lining the surface of the ovary**.

-Most likely in women aged > 40 years.

1-**Serous Cystadenoma**: **most common**, resembles the lining of the **follapian tubes**, bilateral in 10% of cases, 70% are benign. They are small, unilocular, characteristically 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, with thick and mucinous fluid, characteristically form " **Pseudomyxoma peritonei**".

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

❑ B-Sex Cord–Stromal Ovarian Neoplasms:

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

1-**Granulosa cell tumors**: malignant tumors (locally malignant but have good prognosis), grow very slowly, solid tumor, recurrence is common, **secrete estrogen and inhibin and predispose to endometrial cancer.**

2-**Theca cell tumor**: benign, solid, unilateral, mostly in postmenopausal women, 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 tumors, found around the age of 30, very rare, small, unilateral, **may produce androgens and signs of virilization**.

❑ **C-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 primarily of ectodermal tissue (such as sweat and sebaceous glands, hair follicles, and teeth), with some mesodermal (bone, cartilage, muscle). 60% asymptomatic

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

☒Diagnosis:

-By manual examination and By pelvic ultrasonography (TV U/S).

☒ **Management of ovarian cysts:**

- Reproductive age:

- Conservative; wait and reexamine the patient after her next menses + follow up by U/S and CA125 checking after 3 months if: Asymptomatic/ < 5 cm
- Surgical exploration (Laparoscopy + cystectomy vs oophorectomy (fertility preserved) if: The lesion complex (has septations or solid component), Simple cyst > 7 cm/Symptomatic or painful.
- **Postmenopausal:**

1-No further action: if simple cyst < 1 cm.

2-Conservative + follow up (repeat U/S and CA125 every 4 months for 1 year) if: Simple cyst 1-5 cm without features of malignancy (low RMI) and normal CA125/Asymptomatic.

3-Surgical exploration (Laparoscopy + bilateral salpingo-oopherectomy +/- hysterectomy if: The lesion is solid, complex, fixed/> 5 cm/Symptomatic or painful.

☒ Ovarian cancer

- The **worst gynecological cancer** because early detection is difficult (asymptomatic) and diagnosis is almost always at an 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 first pregnancy.
✓ BRCA-1 gene /family history/past history of ovarian cancer
✓ Nulliparity/Infertility.

☒ Protective factors:

- Decreased life time ovulation (OCPs, chronic anovulation (PCOS), tubal ligation) and parity

☒ Signs and Symptoms:

- **Symptoms:** **nonspecific symptoms,** first symptoms to appear are GI symptoms (vague abdominal pain or bloating at early stages), pressure

symptoms (urinary frequency or urgency or constipation), dyspareunia, menstrual irregularity, swelling due to ascites at late stages.

- **Signs**: a vaginal or rectal examination will usually reveal a solid, irregular, fixed pelvic mass; combined ascites

☒ **According to type of cell origin:**

1-Epithelial origin: 80% of ovarian cancer /postmenopausal.

➢ Types are :**Serous (most common)/** Mucinous/ Endometrial

2-Germ cell: 15%, young women.

➢ (Dysgerminoma, Endodermal sinus tumor, Immature teratoma, Embryonal carcinoma, Choriocarcinoma).

3-Stromal: 5%, all ages. (Granulosa cell tumor, Sertoli-Leydig cell tumor).

- **Diagnosis:**
- TV U/S with or without doppler is the 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
✓ -**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
- **Staging is surgical not clinical/ The first stage is the last stage,any recurrence of the disease staged the same stage with.**

• Treatment:

- The initial approach to all patients with ovarian cancer is **surgical exploration** of the abdomen and pelvis.
- **Early stage**: total abdominal hysterectomy (TAH), bilateral salpingo-oophorectomy (BSO)
- In patients with **grade 1 or grade 2 tumors confined to one or both ovaries** after surgical staging, no further treatment is necessary.
- Patients with **poorly differentiated (grade 3)** tumors are subsequently treated with systemic chemotherapy.
- **Advanced stage disease**: cytoreductive surgery ("debulking followed by chemotherapy

☒ Endometrial Cancer

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

• RISK Factors

- Obesity: results in an increased estrone.
- Hypertension/Diabetes mellitus.
- Endometrial hyperplasia.
- Polycystic ovarian syndrome.
- Late menopause/Estrogen replacement therapy (long term).
- Tamoxifen for breast cancer (2-3 fold increased risk)
- Nulliparity.
- Lynch syndrome: hereditary nonpolyposis colon cancer (HNPCC)

☒ Protective factors

-**SMOKING** is protective against endometrial cancer because nicotine blocks estrogen receptors/ **OCP**/ **parity**

☒ Types of endometrial cancer:

1-Type-I (estrogen-dependent): **endometroid type (adenocarcinoma)/**mean age (63-year)/ 5-year survival (85%)/ **good prognosis/ more common.**

2-Type-II (non-estrogen dependent): can be papillary serous (worst type) or clear cell type /mean age (67-year)/ 5-year survival (58%)/ **poor prognosis, less common.**

☒ Symptoms:

- **Postmenopausal bleeding** (the most common symptom

☒ Diagnosis:

- **Any woman with postmenopausal bleeding** should undergo **TV U/S.**
- Depending on the **endometrial thickness on the TV U/S:**
- If it is < 5 mm: should be evaluated after 5 months
- **If it is >5mm**: **D&C and biopsy:**
  - ✓ If the endometrial biopsy reveals endometrial cancer, definitive treatment can be arranged

☒ Pattern of spread:

1-Direct extension. (Most common)

2-Lymphatic dissemination.

3-Hematogenous dissemination. (Worst)

- **Primary staging is final, if we diagnose a lady with stage I and we do resection, and after a while, she comes back with liver metastasis and histopathology shows that it's the same type of resected tumor, it's still stage I**

• Treatment :

Stage IA: TAH, BSO without adjuvant radiotherapy.

Stage IB / or clear cell or serous (type II) with negative lymph nodes: TAH, BSO with adjuvant radiotherapy.

Stage II: modified radical TAH , BSO, may require pelvic and paraaortic lymphadenectomy, adjuvant radiotherapy

☒ **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.**

☒ **Prognostic Factors :**

1-**Histologic type**: 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.

☒Cervical Cancer

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

☒ TYPES

- **Squamous Cell Carcinoma** :Most of it originate from **the transitional zone** ,the **most common type (90%)** and the one 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

☒ 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 or sexual partner with
- ✓ Multiple sexual partners
- ✓ A male with penile condyloma acuminate
- ✓ Lower socioeconomic status
- ✓ **Smoking**
- ✓ Immunodeficiency

- **Note that it's NOT linked to the FREQUENCY of sexual activity, Age at menopause or family history**

☒ PREVENTION

- ✓ **PRIMARY PREVENTION: VACCINATION**
- ✓ **SECONDARY PREVENTION: SCREENING: By physical examination, including a Papanicolaou smear, beginning by age 21:**
  - **21 -30 years: Annually**
  - **>30 years: every 3 years**

☒ Symptoms

Abnormal vaginal bleeding is the first symptom; **usually post-coital bleeding** that's why in patients who are not sexually active, bleeding from cervical cancer usually does not occur until the disease is quite advanced with advanced disease/ Persistent malodorous vaginal discharge, pelvic pain

☒ Physical Examination

- The cervix may be ulcerative. It usually bleeds on palpation (it's highly vascular and friable)
- A rectovaginal examination is essential to determine the extent of disease: diameter of the primary cancer, spread to the parametria & extension into the uterosacral ligaments.

☒ Patterns of Spread

- Direct invasion
- lymphatic spread pelvic lymph nodes and then paraaortic lymph nodes
- hematogenous spread lungs, liver, and bone.

☒ Staging

- **It's the only cancer that's staged clinically (based on physical examination 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: Chemoradiotherapy**
- **Stage 3A: Chemoradiotherapy**
- **Stage 3B: Chemoradiotherapy**
- **Stage 4A: Chemoradiotherapy**