NON-NEOPLASTIC PATHOLOGIES OF THE UTERUS

1 ENDOMETRITIS

Inflammation of the endometrium.

CAUSES

- Infections leading to pelvic inflammatory disease (PID)
- Miscarriage or delivery
- Intrauterine devices

ACUTE vs. CHRONIC

ACUTE	CHRONIC
• Fever	• Persistent or recurrent symptoms
• Abdominal pain	• Risk of infertility
• Menstrual abnormalities	• Risk of ectopic pregnancies

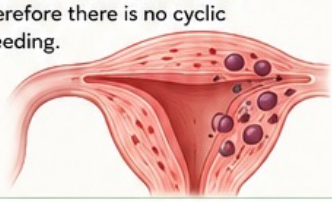
TREATMENT

- Removal of the cause (e.g., infection, IUD removal)
- Antibiotics
- D&C (dilation and curettage)

2 ENDOMYOSIS

Endometrial stroma and glands impregnated in the myometrium.

Derived from stratum basalis, therefore there is no cyclic bleeding.



CLINICAL MANIFESTATIONS

- Thickened uterine wall
- Globular, enlarged uterus
- Menstrual abnormalities:
  - Menorrhagia (heavy bleeding)
  - Dysmenorrhea (severe pain) due to enlarged uterus and exaggerated contractions

KEY POINT

No cyclic bleeding because it is derived from the stratum basalis.

3 ENDOMETRIOSIS

Presence of endometrial glands and stroma outside the uterus. Not cancerous.

- Occurs in ~10% of women in reproductive years
- Increases the risk of infertility

CLINICAL MANIFESTATIONS

- Dysmenorrhea
- Chronic pelvic pain
- Pelvic mass filled with blood ("chocolate cyst")

SITES

PELVIC SITES

- Ovaries
- Douglas pouch
- Uterine ligaments
- Fallopian tubes
- Rectovaginal septum

DISTANT SITES

- Umbilicus
- Lymph nodes
- Lungs

PATHOGENESIS (FUNCTIONAL)

Cyclic bleeding of ectopic endometrium

↓

Inflammation

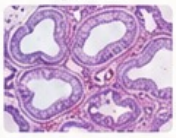
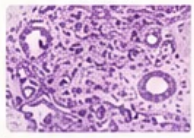
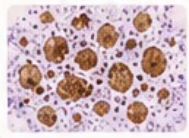
↓

Fibrosis and sealing of the tubules from radiated ends

↓

Destruction of ovaries

DIAGNOSIS (Any 2 of the following)

Endometrial glands	Endometrial stroma	Hemosiderin pigment
		

THEORIES OF PATHOGENESIS

- **Regurgitation theory:** Backflow of menstrual blood into the tubes. (Most accepted theory)
- **Metaplastic theory:** Coelomic epithelium differentiates into endometrial tissue.
- **Vascular or lymphatic dissemination theory:** Spread via blood or lymphatic channels; explains extrapelvic or intranodal implants.
- **Extrauterine stem progenitor cell theory:** Circulating stem progenitor cells from bone marrow differentiate into endometrial tissue.

4 ENDOMETRIAL HYPERPLASIA

Characterized by prolonged or excess estrogen (relative to progesterone).

Excess estrogen → exaggerated proliferation → risk of endometrial cancer.

RISK FACTORS

- Obesity
- Diabetes
- Hypertension
- Infertility
- Prolonged estrogen replacement therapy
- Estrogen-secreting ovarian tumors

SEVERITY DEPENDS ON

ARCHITECTURE (CROWDING)

- Mild
- Moderate
- Severe

PRESENCE OF ATYPIA

- Without atypia → lower risk
- With atypia → higher risk of progression to cancer

SEQUENCE

Excess estrogen (relative to progesterone)

↓

Endometrial hyperplasia

↓

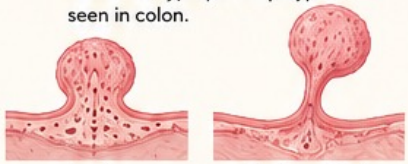
Atypia (in some cases)

↓

Endometrial carcinoma

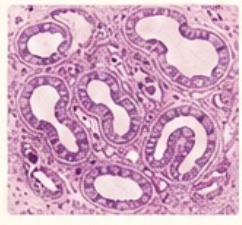
5 BENIGN ENDOMETRIAL POLYP

Similar to hyperplastic polyp seen in colon.



Sessile Pedunculated

CHARACTERISTICS



- Endometrial dilated glands
- Small muscular arteries
- Fibrotic stroma

CLINICAL SIGNIFICANCE

No increased risk of endometrial cancer.

TUMORS OF THE UTERUS

1 TUMORS OF THE ENDOMETRIUM

ENDOMETRIAL CARCINOMAS

TWO CLINICAL SETTINGS

- Most common cancer of the female genital tract
- Develops in patients in their 50s–60s

1. ESTROGEN-RELATED (perimenopausal women)

Estrogen excess ↓

Prototype: **Endometrioid carcinoma (Type 1)**

2. NOT ESTROGEN-RELATED (older women with endometrial atrophy)

No relation to estrogen ↓

Prototype: **Serous carcinoma (Type 2)**

ENDOMETRIOID CARCINOMA (TYPE 1)

Resembles	Similar to normal endometrium
Risk factors	• Obesity • Hypertension • Diabetes • Exogenous estrogens (e.g., HRT, tamoxifen) • Estrogen-secreting ovarian tumors • Infertility
Precancerous lesion	Endometrial hyperplasia with atypia
Molecular alteration	Defects in DNA mismatch repair (MMR) genes; PTEN mutation
Prognosis	Stage 1 = 90% 5-year survival; drops to 40% in stages 3 and 4.

SEROUS CARCINOMA (TYPE 2)

- No relation to endometrial hyperplasia or hormones
- Molecular alteration: TP53 tumor suppressor gene mutation
- Managed by operative staging with peritoneal cytology
- Generally worse prognosis than endometrioid carcinoma (Type 1)

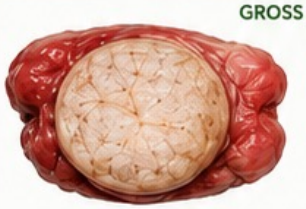
2 TUMORS OF THE MYOMETRIUM

A. LEIOMYOMA (FIBROID)

التليف (Talayuf)

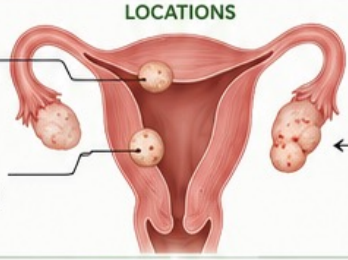
- Most common benign tumor in females
- Estrogen-dependent
  Shrinks at menopause

GROSS FEATURES



- Benign
- Well-circumscribed
- White, firm mass
- Whorled (swirled) cut surface

LOCATIONS



OTHER FEATURES

- May develop hemorrhagic cystic change or calcification

CLINICAL FEATURES

- May be asymptomatic
- Menorrhagia (heavy bleeding)
- Dragging pelvic sensation
- Anemia (due to blood loss)

IMPORTANT POINT

Almost never transforms into sarcomas — leiomyosarcoma **does not arise from a pre-existing leiomyoma.**

B. LEIOMYOSARCOMA

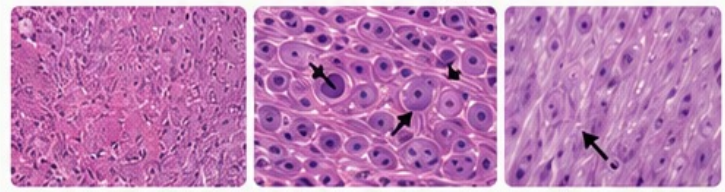
(Malignant counterpart)

Does NOT arise from a pre-existing leiomyoma.

GROSS FEATURES

- Hemorrhage
- Necrosis
- Infiltrative (irregular) borders (not well-circumscribed)

MICROSCOPIC FEATURES (Typical)



Coagulative necrosis | Cytological atypia | Mitotic activity

DIAGNOSIS DEPENDS ON THE PRESENCE OF:

Coagulative necrosis + Cytological atypia + Mitotic activity

CLINICAL BEHAVIOR

- Aggressive tumor
- Tends to spread early
- Poor prognosis

PROGNOSIS

Generally poor

5-year survival: ~20–40%