



THE UNIVERSITY OF  
JORDAN

# Uterine Pathology

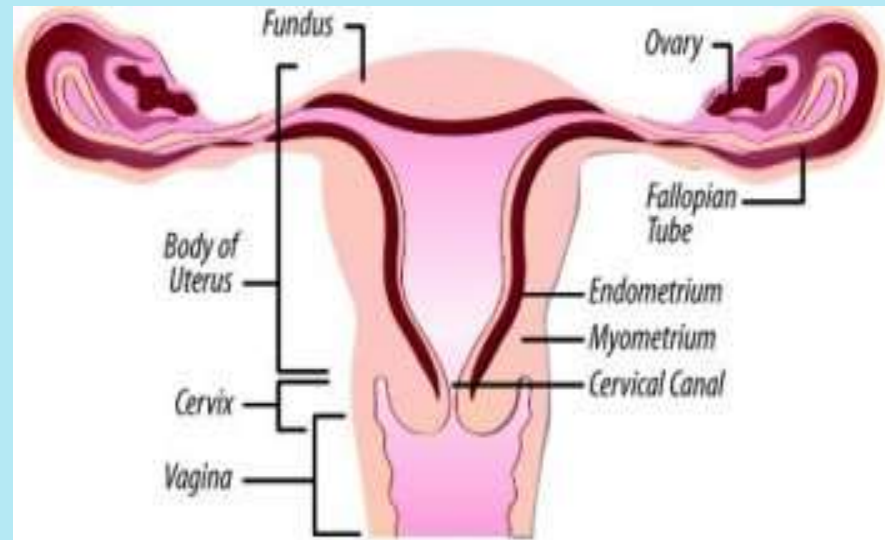
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University of Jordan,  
School of Medicine

# Endometrium

- ▶ Endometritis
- ▶ Adenomyosis
- ▶ Endometriosis
- ▶ Endometrial Polyps
- ▶ Endometrial Hyperplasia
- ▶ **Endometrial Carcinoma**

# Myometrium

- ▶ Leiomyoma
- ▶ **Leiomyosarcoma**



# ENDOMETRITIS

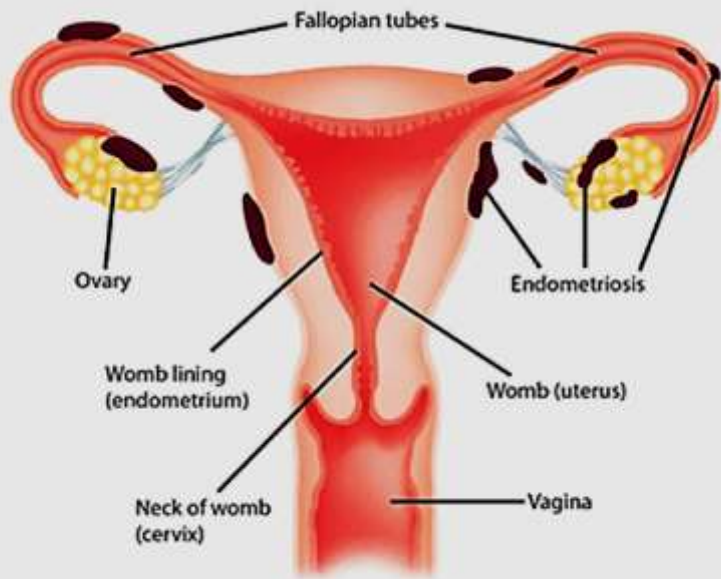
- ▶ Inflammation of the endometrium.
- ▶ Causes:
  - 1- infections - pelvic inflammatory disease (PID)
  - 2- miscarriage or delivery
  - 3- intrauterine device (IUCD).
- ▶ acute or chronic
- ▶ fever, abdominal pain, menstrual abnormalities, infertility and ectopic pregnancy due to damage to the fallopian tubes.
- ▶ Rx: removal of cause, antibiotics, D&C.

# ADENOMYOSIS

- ▶ endometrial stroma, glands, or both embedded in **myometrium**.
- ▶ Thick uterine wall, enlarged uterus.
- ▶ Derived from stratum basalis → no cyclical bleeding.
- ▶ menorrhagia, dysmenorrhea (due to enlarged uterus, uterine contractions are exaggerated )

# ENDOMETRIOSIS

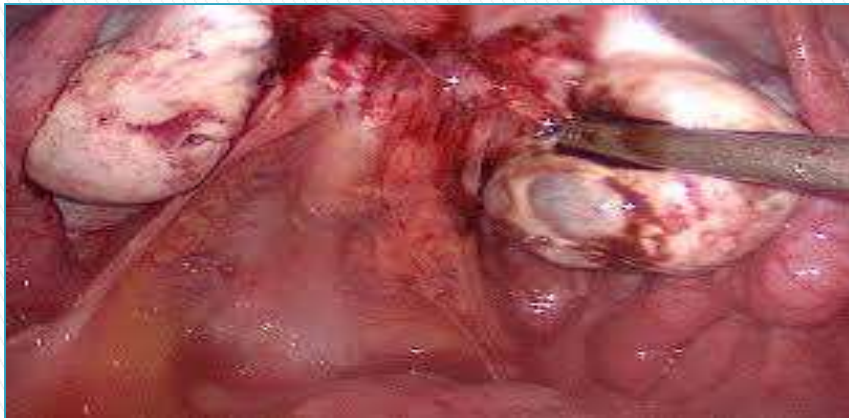
- ▶ endometrial glands and stroma **outside the uterus (not cancer !)**.
- ▶ 10% in reproductive yrs; ↑ infertility.
- ▶ dysmenorrhea, and pelvic pain, pelvic mass filled with blood (**chocolate cyst**).
- ▶ Multifocal in pelvis (ovaries, pouch of Douglas, uterine ligaments, tubes, and rectovaginal septum).
- ▶ Sometimes distant sites (e.g. umbilicus, lymph nodes, lungs, ...)



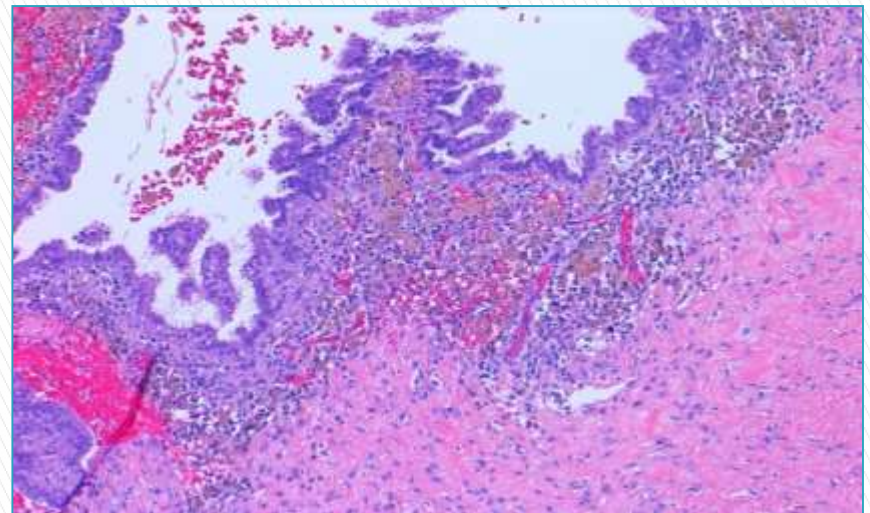
Common locations of endometriotic lesions



“Chocolate“ cyst in an ovary



Intraoperative view of endometriosis

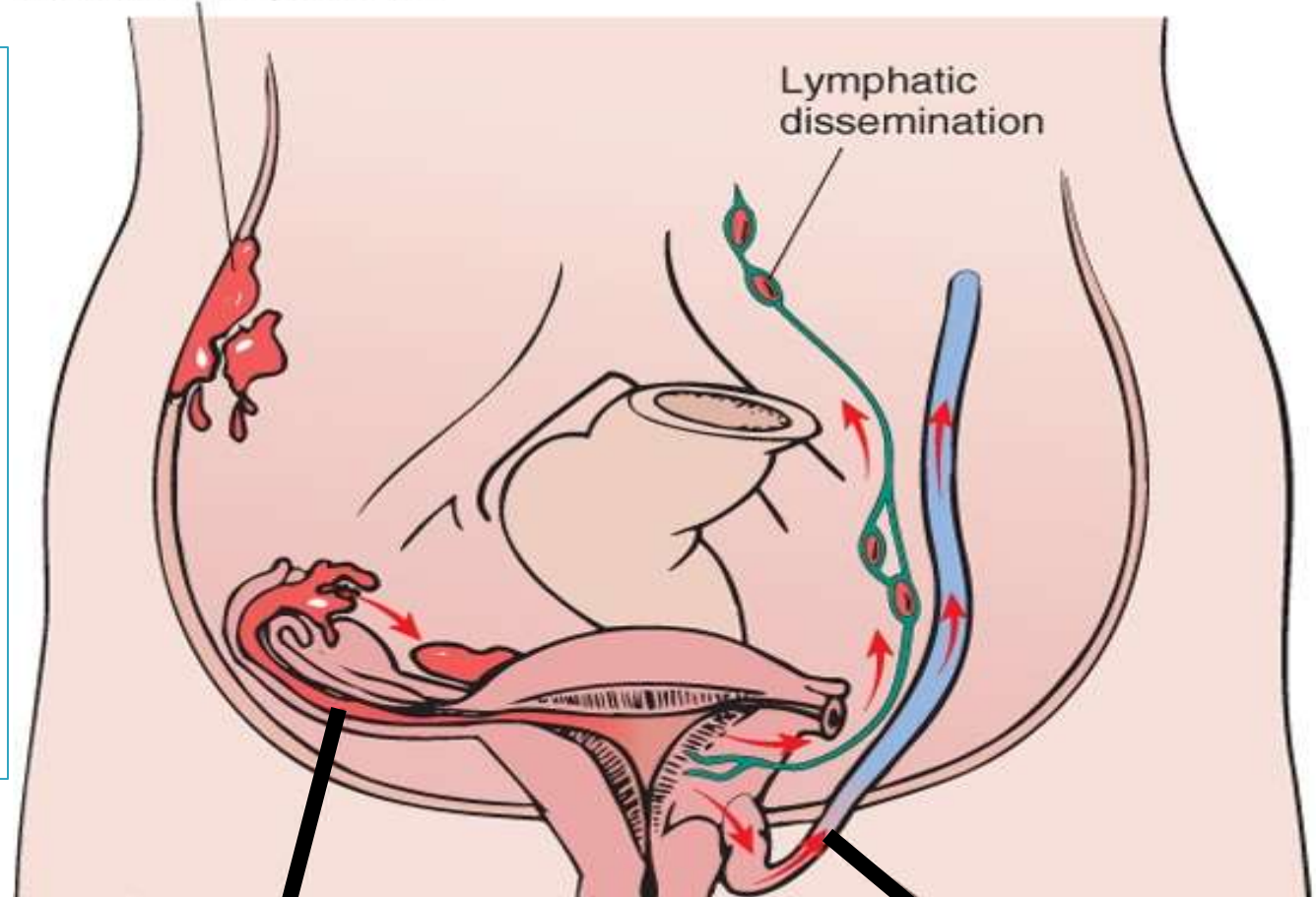


Microscopic view of endometriosis

# ENDOMETRIOSIS- Pathogenesis

- ▶ 4 theories:
- ***Regurgitation theory.*** (most accepted). Menstrual backflow through tubes and implantation..
- ***Metaplastic theory.*** Endometrial differentiation of coelomic epithelium.
- ***Vascular or lymphatic dissemination theory.*** explain extrapelvic or intranodal implants.
- ***Extrauterine stem/progenitor cell theory,*** proposes that circulating stem/progenitor cells from bone marrow differentiate into endometrial tissue

Metaplastic differentiation  
of coelomic epithelium



Conceivably,  
all pathways  
are valid in  
individual  
instances.

Regurgitation  
through fallopian  
tube

Extrapelvic  
dissemination  
through pelvic veins



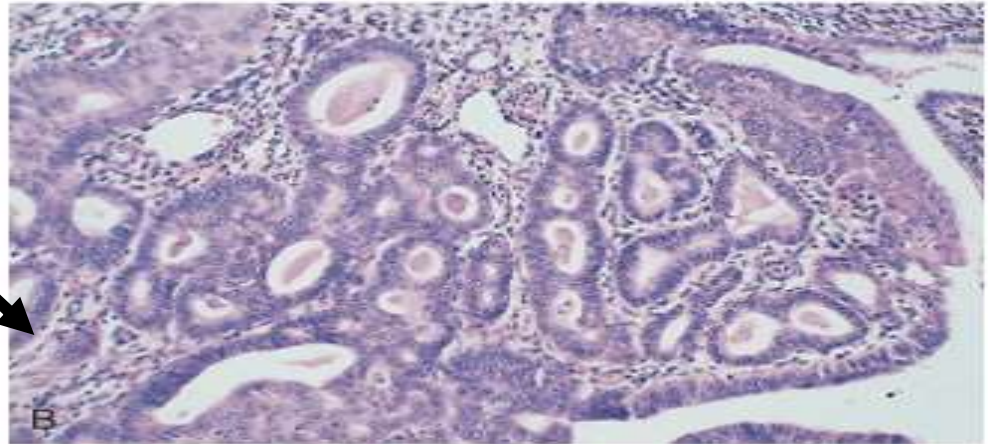
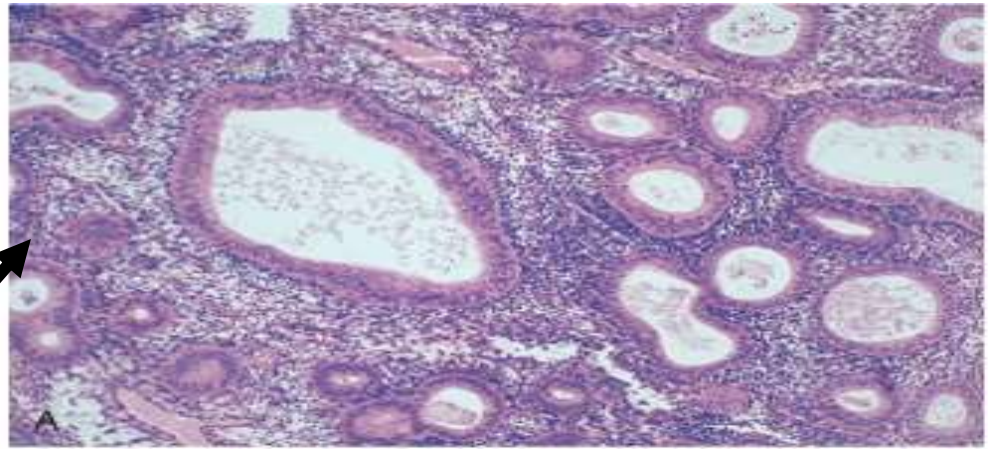
# ENDOMETRIOSIS

- ▶ contains **functionalis endometrium**, so undergoes **cyclic bleeding**.
- ▶ Consequences: fibrosis, sealing of tubal fimbriated ends, and distortion of the ovaries.
- ▶ Diagnosis; 2 of 3 features: **endometrial glands, endometrial stroma, or hemosiderin pigment**.

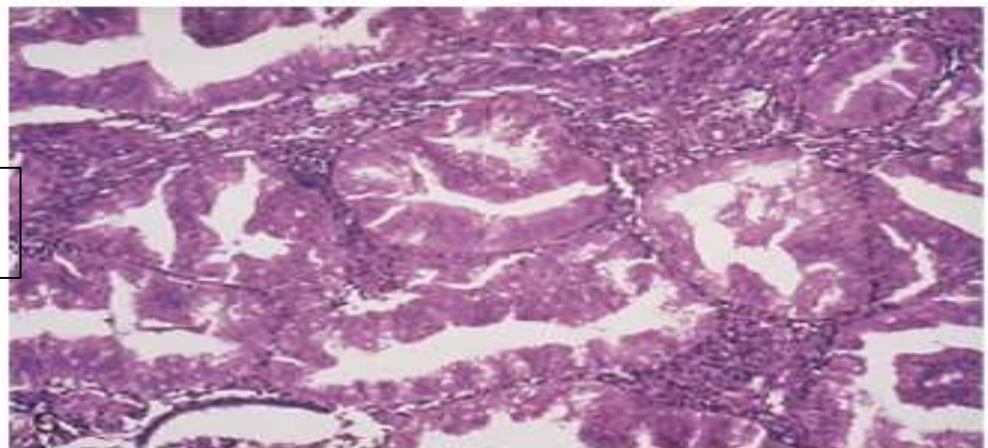
# Endometrial Hyperplasia

- ▶ prolonged or marked excess of **estrogen** relative to progestin → exaggerated proliferation → may progress to cancer
- ▶ risk factors: **Obesity; Diabetes; Hypertension; Infertility; Prolonged estrogen replacement therapy; Estrogen-secreting ovarian tumors.**
- ▶ severity is based on architectural crowding and cytologic atypia, ranging from:
  - 1- Typical hyperplasia (without atypia)
  - 2- Atypical hyperplasia (20% risk of cancer).

**Hyperplasia  
without  
atypia**



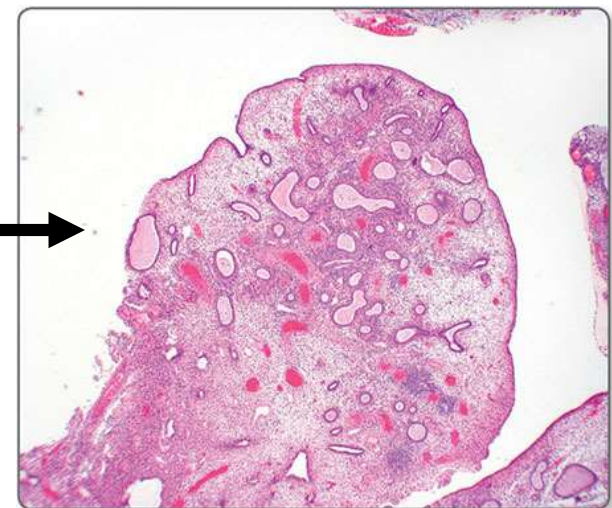
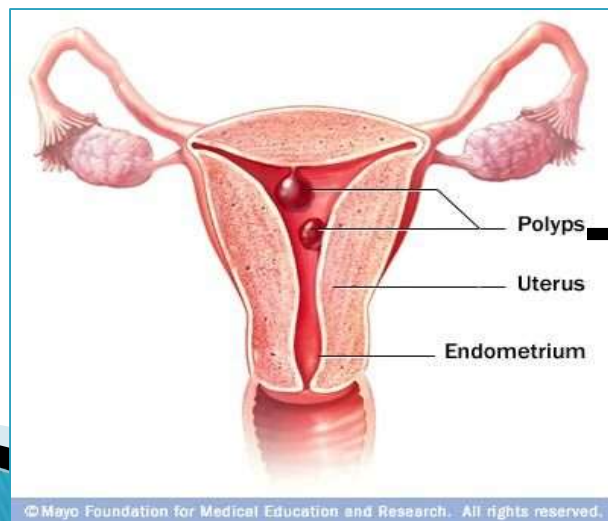
**Atypical Hyperplasia**



# TUMORS OF THE ENDOMETRIUM

## ❖ Benign Endometrial Polyps

- ▶ sessile or pedunculated
- ▶ endometrial dilated glands, with small muscular arteries and fibrotic stroma.
- ▶ no risk of endometrial cancer.



# Endometrial Carcinoma

- ▶ **the most common cancer in female genital tract.**
- ▶ 50s and 60s.
- ▶ Two clinical settings, These scenarios are correlated with differences in histology:
  - 1) perimenopausal women with estrogen excess (Type I cancers: prototype is called **endometrioid** )
  - 2) older women with endometrial atrophy (Type II cancers: prototype is **serous carcinoma**).

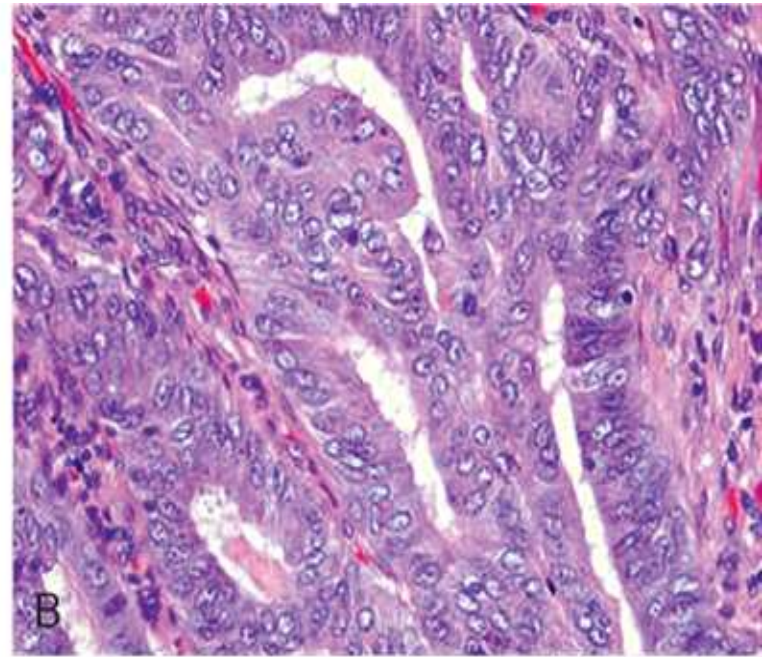
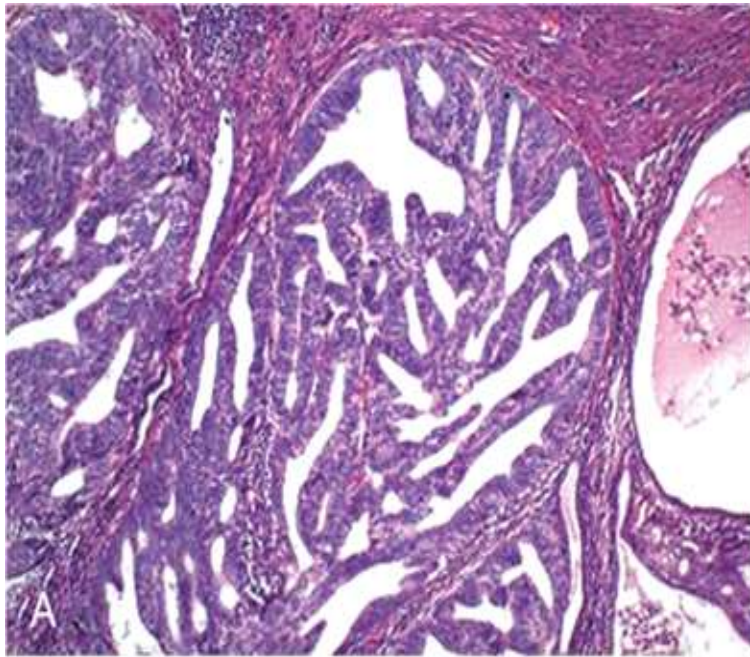
# Endometrioid Carcinoma

- ▶ risk factors: **Obesity; Diabetes; Hypertension; Infertility; Prolonged estrogen replacement therapy; Estrogen-secreting ovarian tumors.**
- ▶ ***precancer lesion is “atypical endometrial hyperplasia”***
- ▶ Mutations in **DNA mismatch repair genes** and ***PTEN***
- ▶ ***Prognosis: depends on stage.***
- ▶ (5-year survival in stage I= 90%; drops to 40% in stages III and IV.)

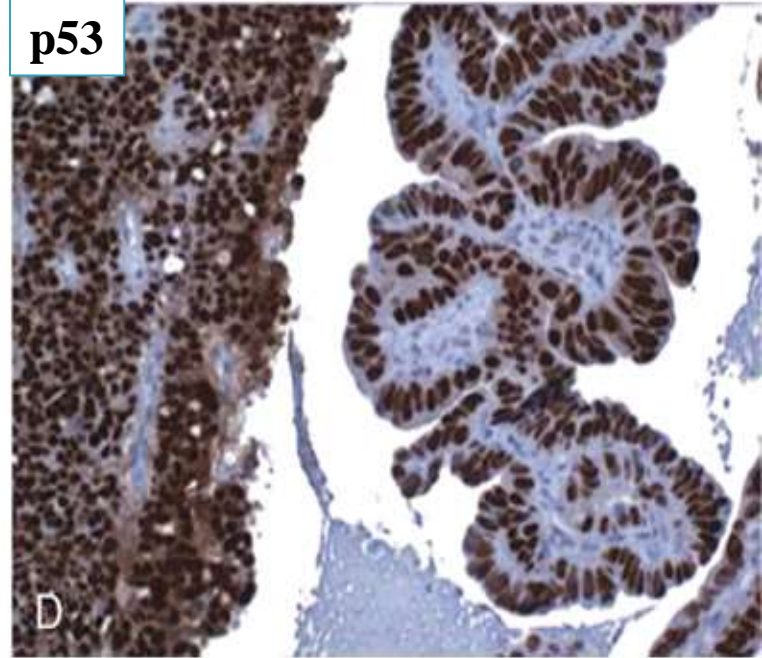
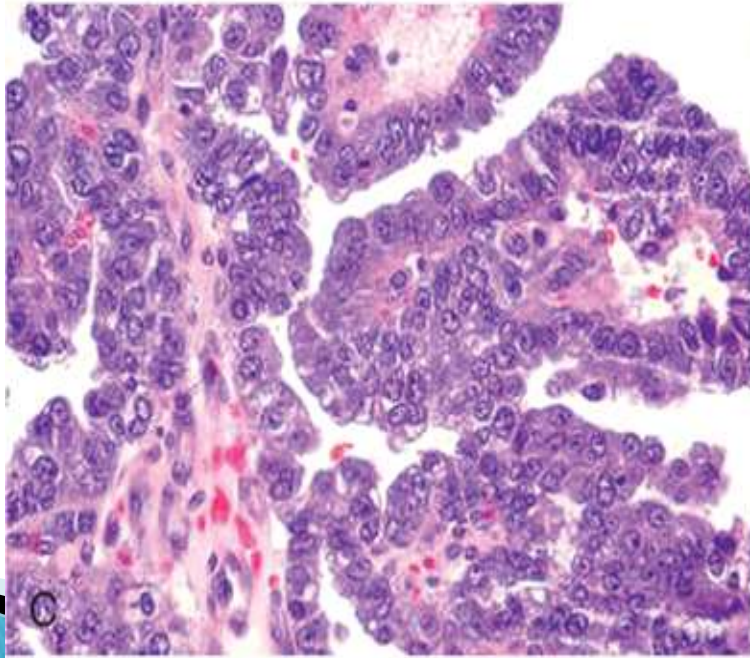
# Serous Carcinoma

- ▶ No relation with endometrial hyperplasia
- ▶ Not hormone-dependent
- ▶ Mutations in *p53* tumor suppressor gene.
- ▶ Prognosis: depends on operative staging with peritoneal cytology. Generally worse than endometrioid ca.

**Endometrioid carcinoma**

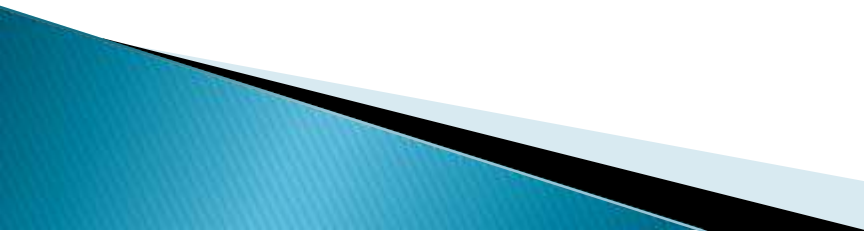


**Serous carcinoma**



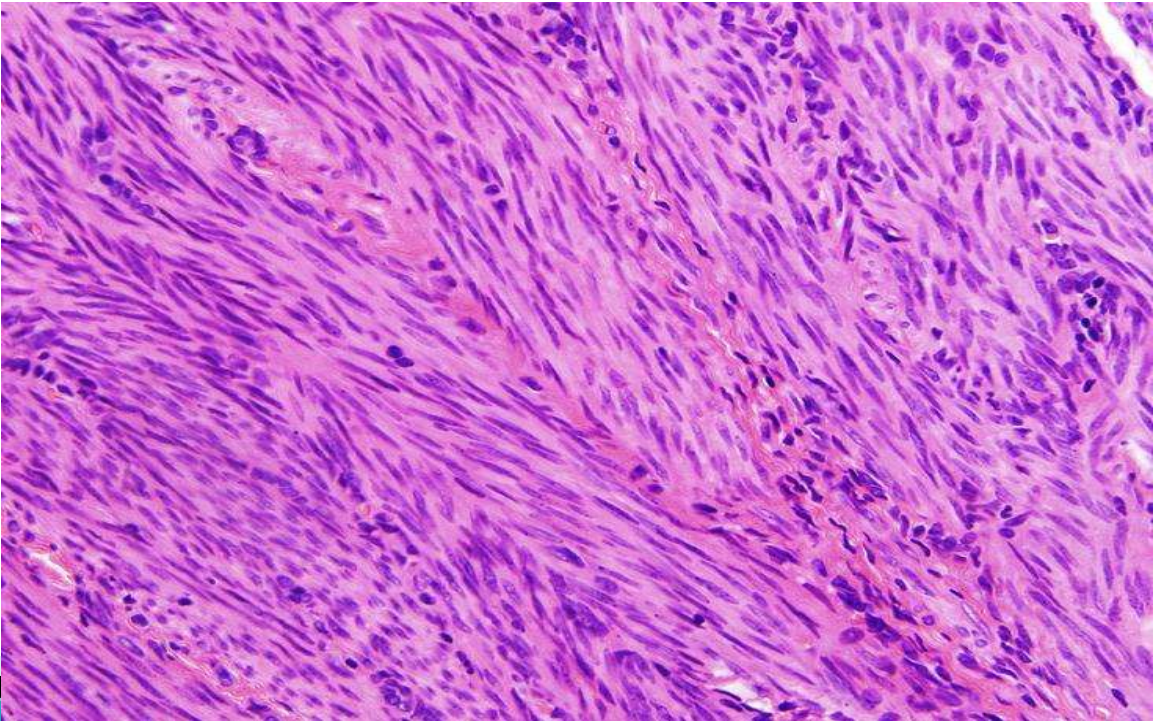
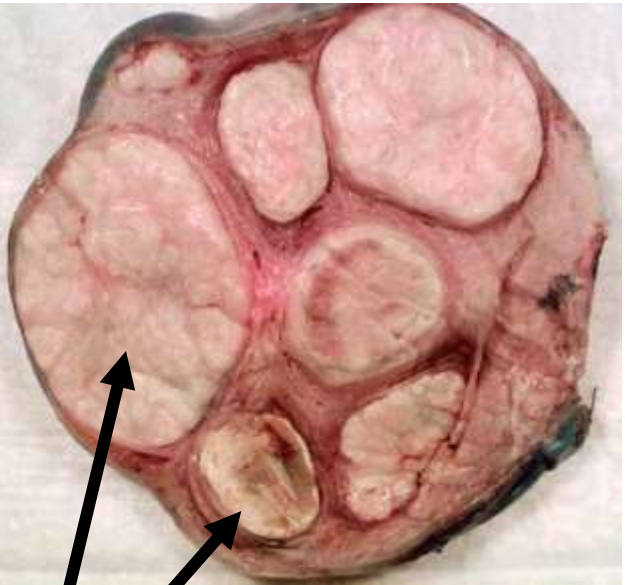


# Tumors of the myometrium

- ▶ **Lieomyoma = fibroids**
  - ▶ Benign tumor of smooth muscle cells
  - ▶ most common benign tumor in females (30% - 50% in reproductive life).
  - ▶ **Estrogen-dependent**; shrink after menopause.
  - ▶ circumscribed, firm masses with whorled cut surface.
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# Leiomyomas

- ▶ Location: (intramural), (submucosal), or (subserosal).
- ▶ may develop hemorrhage, cystic change or calcification.
- ▶ Clinically: asymptomatic or symptomatic; (menorrhagia; a dragging sensation, anemia, etc...)
- ▶ leiomyomas almost **never** transform into sarcomas



# Leiomyosarcoma

- ▶ Malignant counterpart of leiomyoma.
  - ▶ not from preexisting leiomyomas.
  - ▶ hemorrhagic, necrotic, **infiltrative borders**.
  - ▶ diagnosis: **coagulative necrosis, cytologic atypia, and mitotic activity**.
  - ▶ Recurrence common, and metastasis
  - ▶ 5-year survival rate 40%.
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