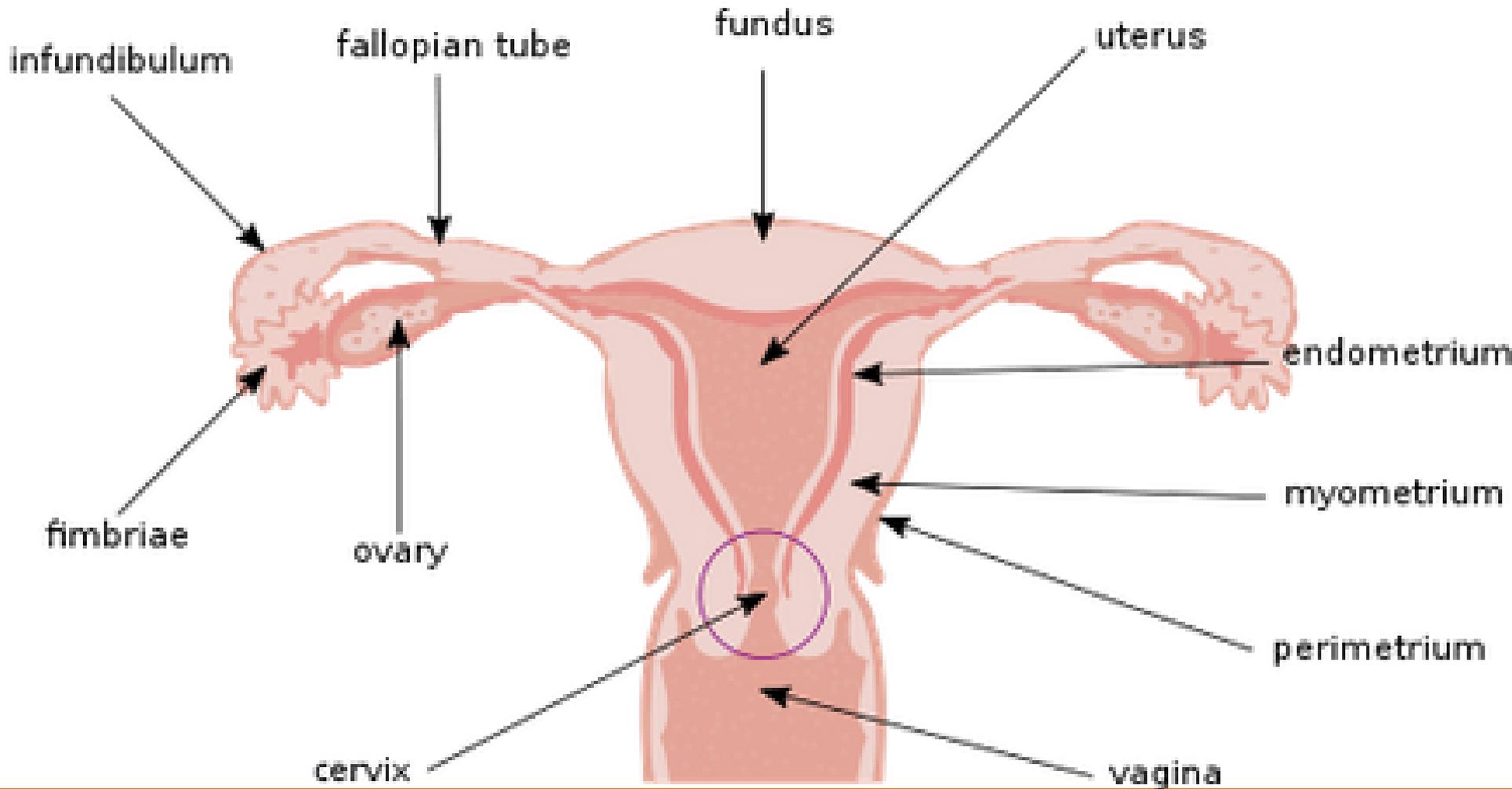


Female Genital System uterine pathology

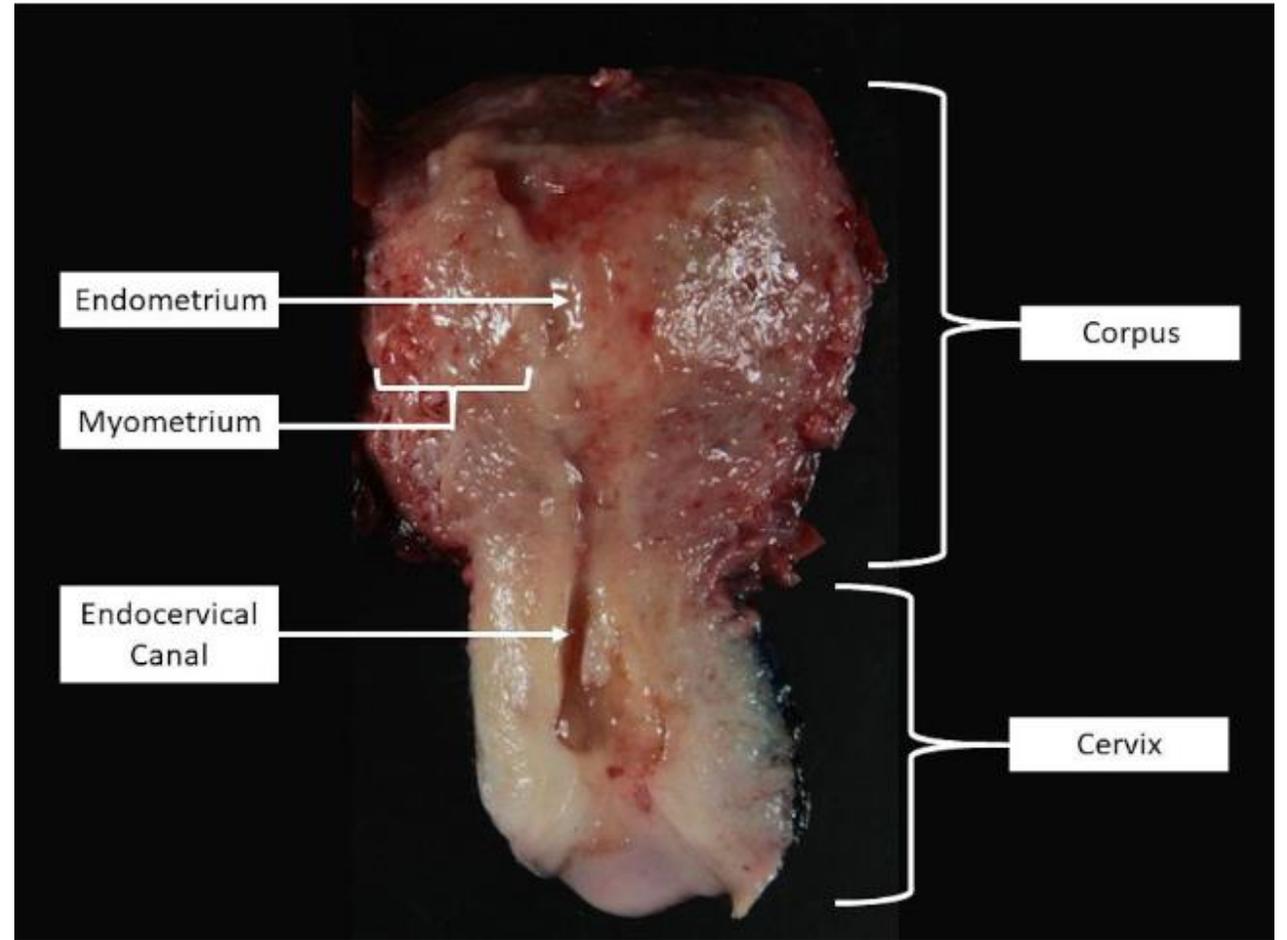
DR.EMAN KREISHAN, M.D.

14-5-2025

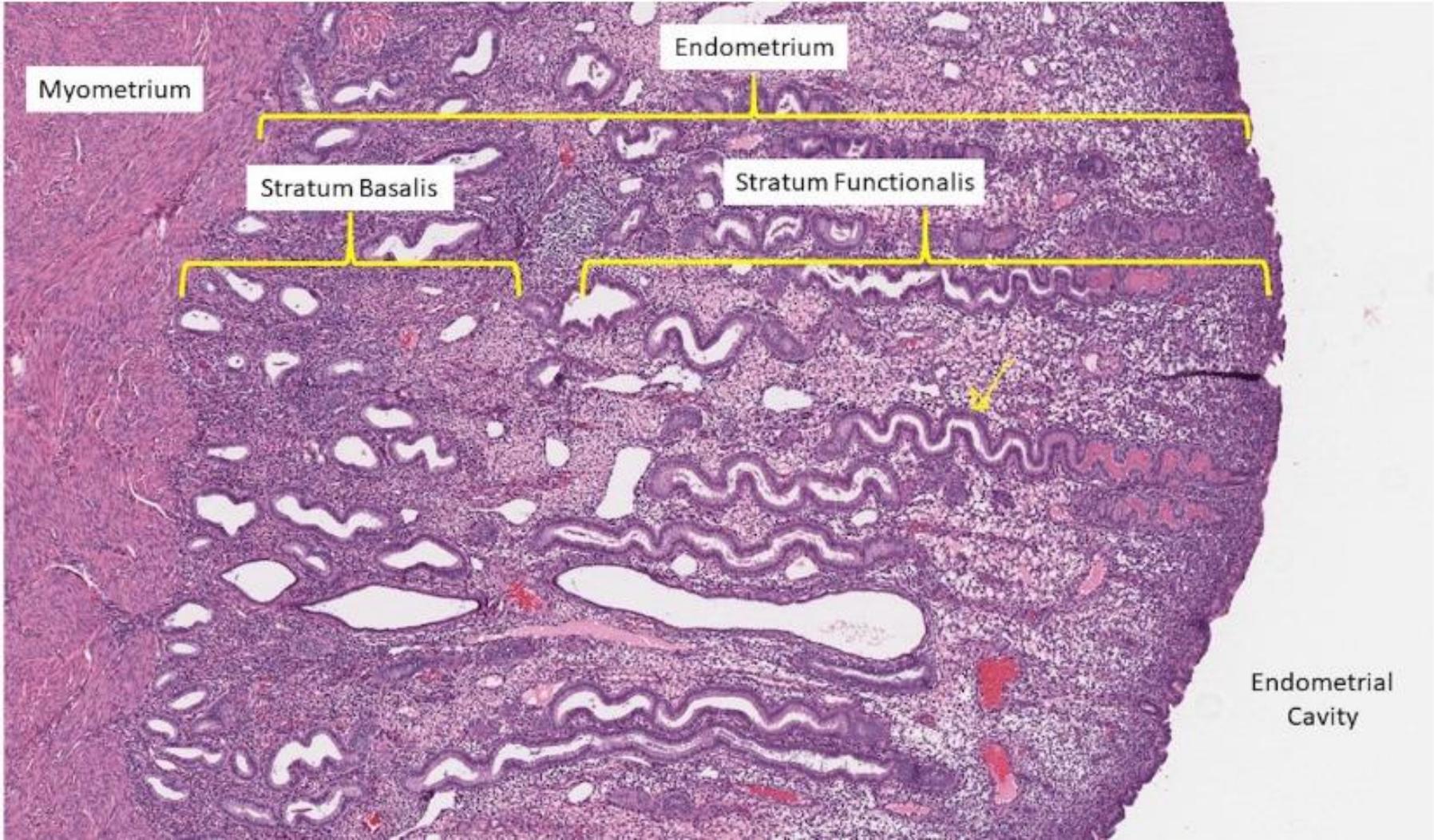
Anatomy



Gross anatomy



Histology



Uterine pathology

➤ Non-neoplastic:

- Endometritis.
- Adenomyosis.
- Endometriosis.
- Hyperplasia.

➤ Neoplastic:

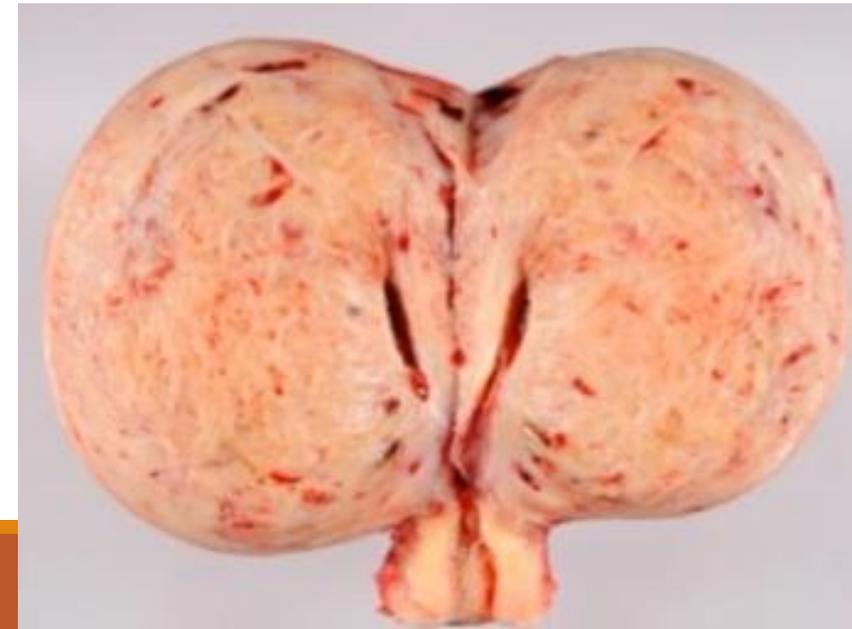
- Hyperplasia.
- Malignant tumors.

1. Endometritis

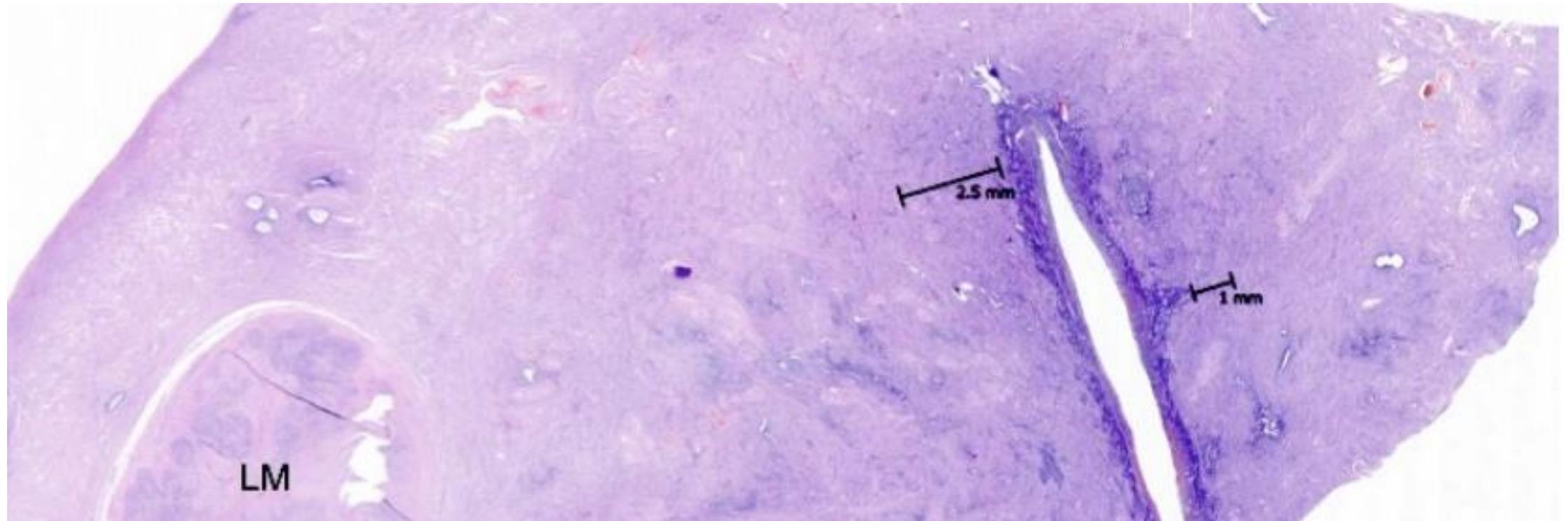
- Inflammation of the endometrium.
- Risk factors:
 - Pelvic inflammatory disease (PID).
 - Retained products of conception.
 - Intrauterine device (IUCD).
- Clinically: fever, abdominal pain, menstrual abnormalities, infertility & ectopic pregnancy due to damage to the fallopian tubes.
- Management: Correct the cause, antibiotics.

2. Adenomyosis

- The presence of endometrial tissue (stroma, glands, or both)in myometrium between muscle bundles.
- Result in thickened uterine wall & enlarged uterus due to reactive muscle hypertrophy.
- Presentation: menorrhagia, dysmenorrhea.
- Usually Coexist with: endometriosis.



Microscopic features



3. Endometriosis

- The presence of estrogen-dependent endometrial tissue outside the uterine cavity.
- Affecting women in the reproductive years.
- Usually it's a multifocal process involving:
 - ❖ pelvic structures: ovaries, uterine ligaments, rectovaginal septum, cul de sac
 - ❖ OR involves distant areas of peritoneal cavity or periumbilical tissues.

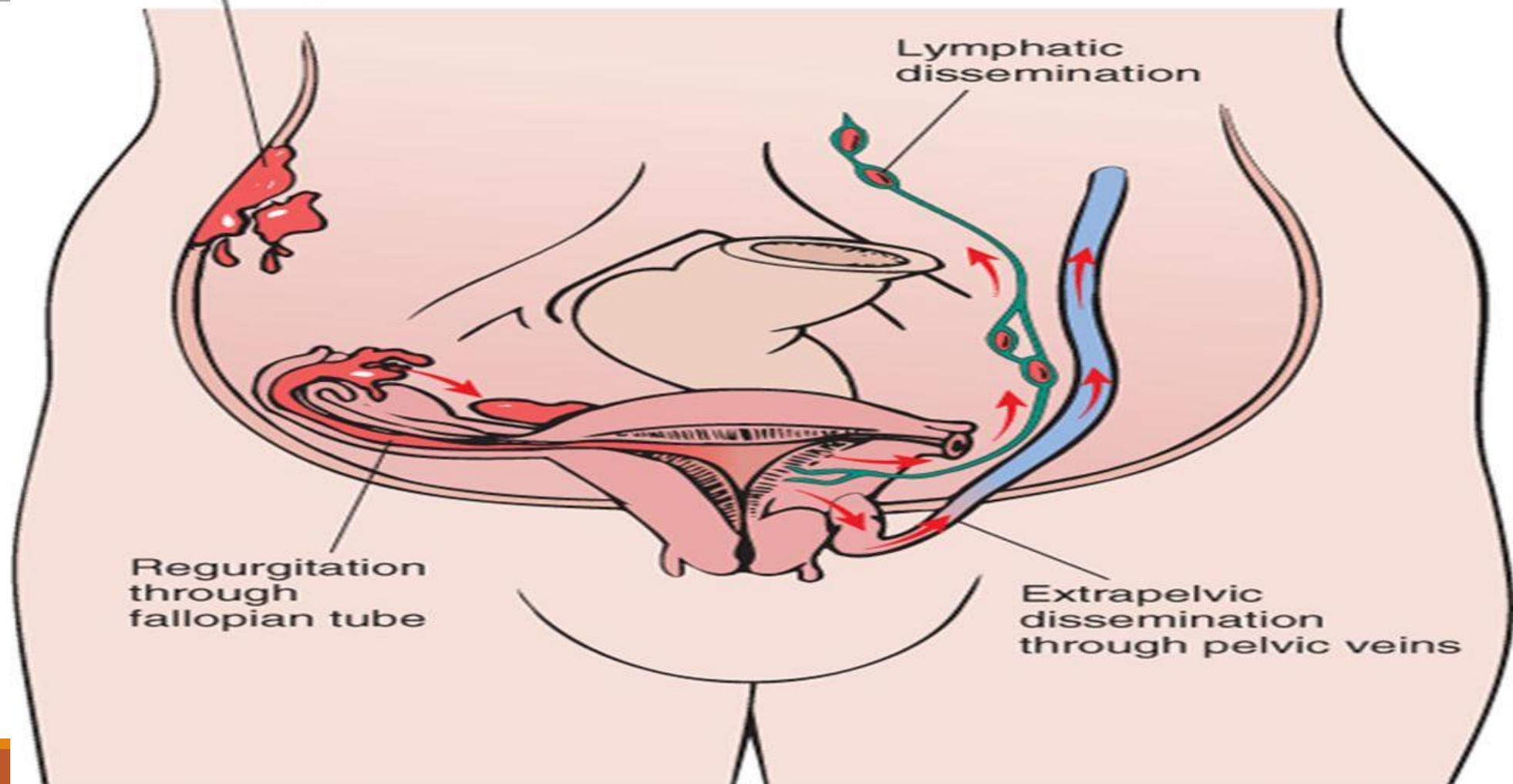
Endometriosis - Pathogenesis

Four hypotheses:

1. Regurgitation theory, **favored**, → menstrual backflow through the tubes → implantation.
2. Benign vascular and lymphatic dissemination.
3. Metaplastic theory, endometrial differentiation of coelomic epithelium
4. The extrauterine stem/progenitor cell theory.

Endometriosis - Pathogenesis

Metaplastic differentiation of coelomic epithelium



Regurgitation through fallopian tube

Lymphatic dissemination

Extrapelvic dissemination through pelvic veins

Clinical presentation

- Clinically presented with pain and infertility.....
- Dysmenorrhea.
- pain on defecation.
- dyspareunia
- dysuria

Endometriotic foci

- consists of functioning endometrium → undergoes cyclic bleeding → organization of blood → widespread fibrosis → adhesions among pelvic structures.



Gross features

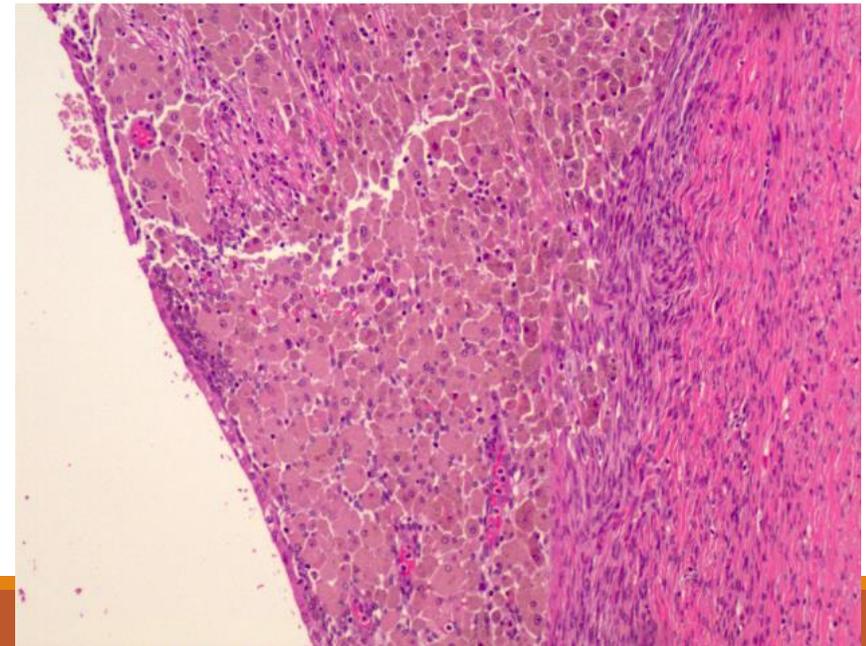
Ovarian endometriosis: chocolate cyst.



Microscopically

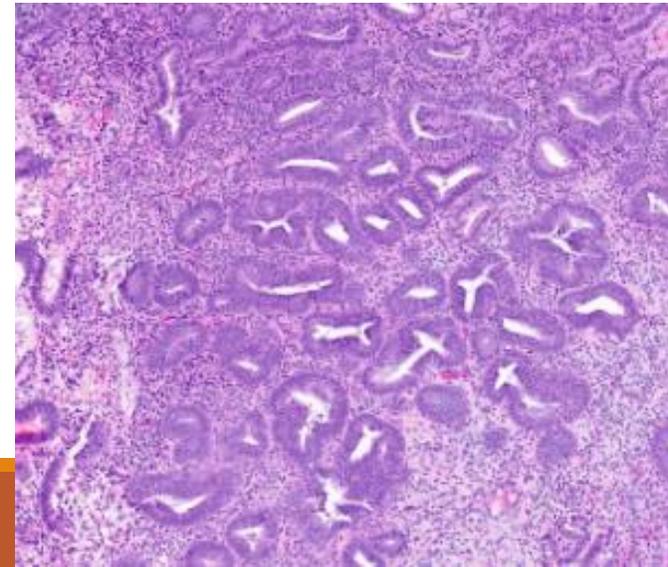
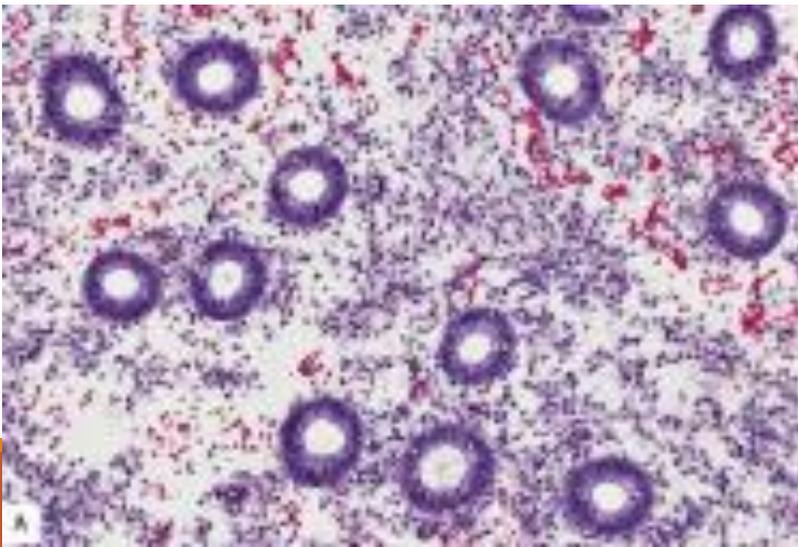
At least 2 of the following 3 features

- ❖ endometrial glands.
- ❖ endometrial stroma.
- ❖ hemosiderin pigment.



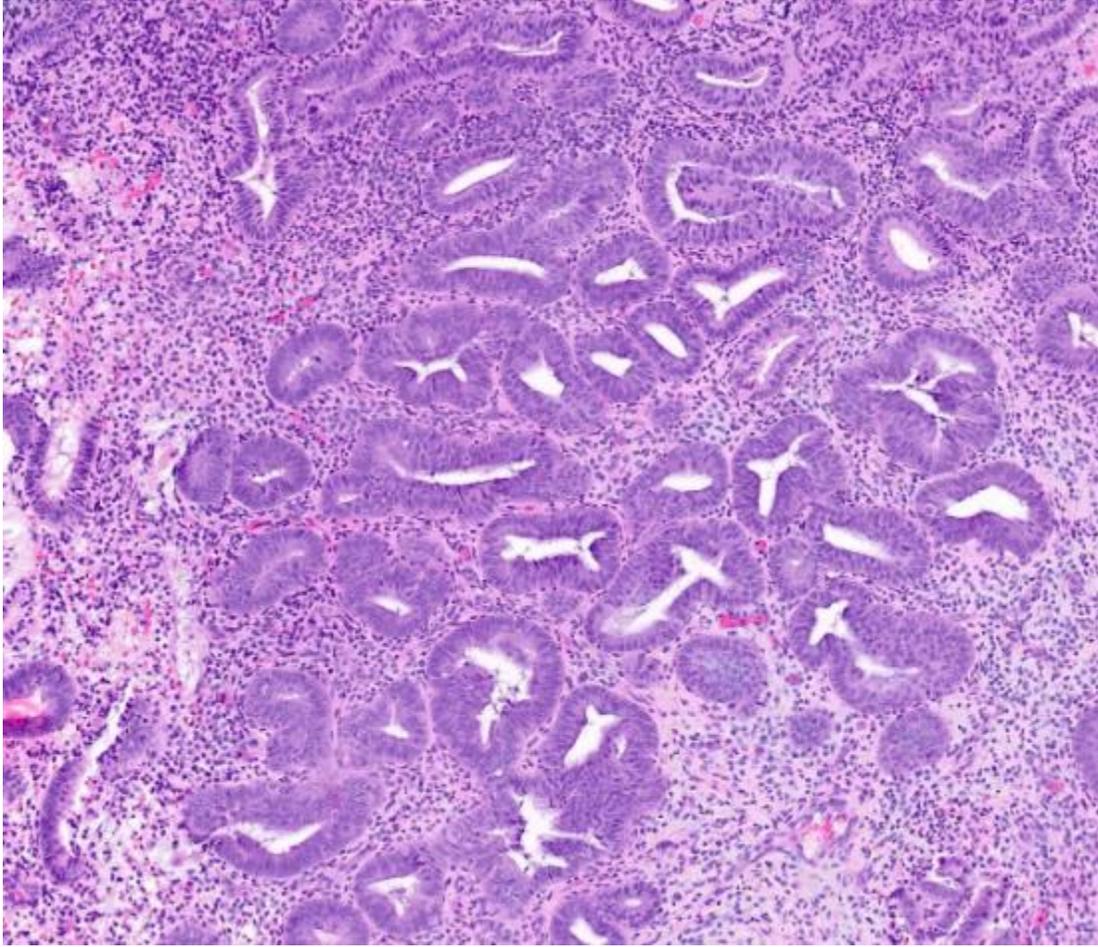
4. Endometrial Hyperplasia

- Proliferation of endometrial glands with a resulting increase in gland to stroma ratio.
- Increased endogenous or exogenous estrogen, unopposed by progesterone .
- Chronic estrogenic stimulation without progesterone affects glands to a greater extent → glandular overgrowth (hyperplasia)

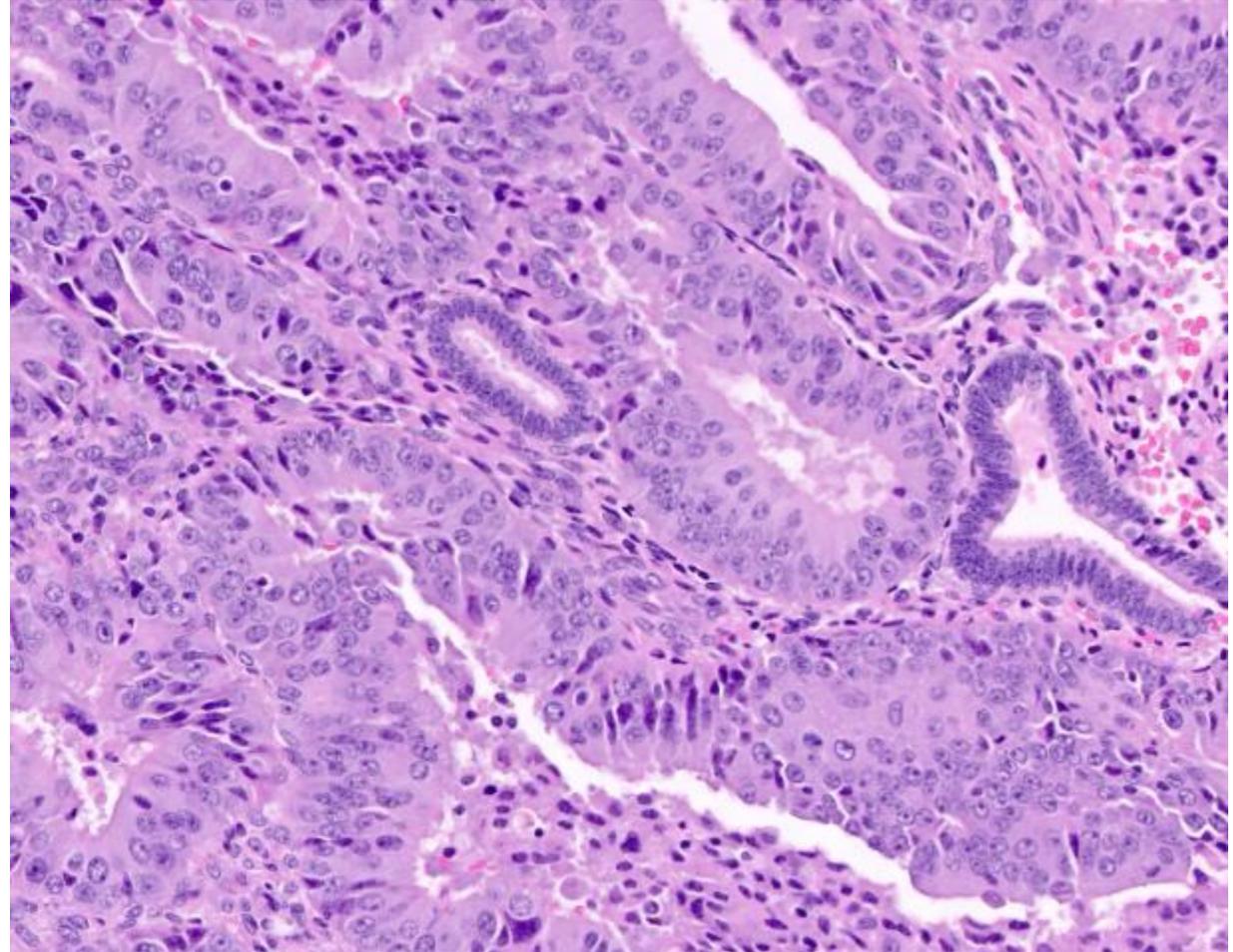


Two categories based on the presence of cytologic atypia:

1. Hyperplasia without atypia; low risk for progression to endometrial Ca.
2. Hyperplasia with atypia(**endometrial intraepithelial neoplasia (EIN)**) higher risk for progression to endometrial Ca. → 20%.



Hyperplasia without atypia



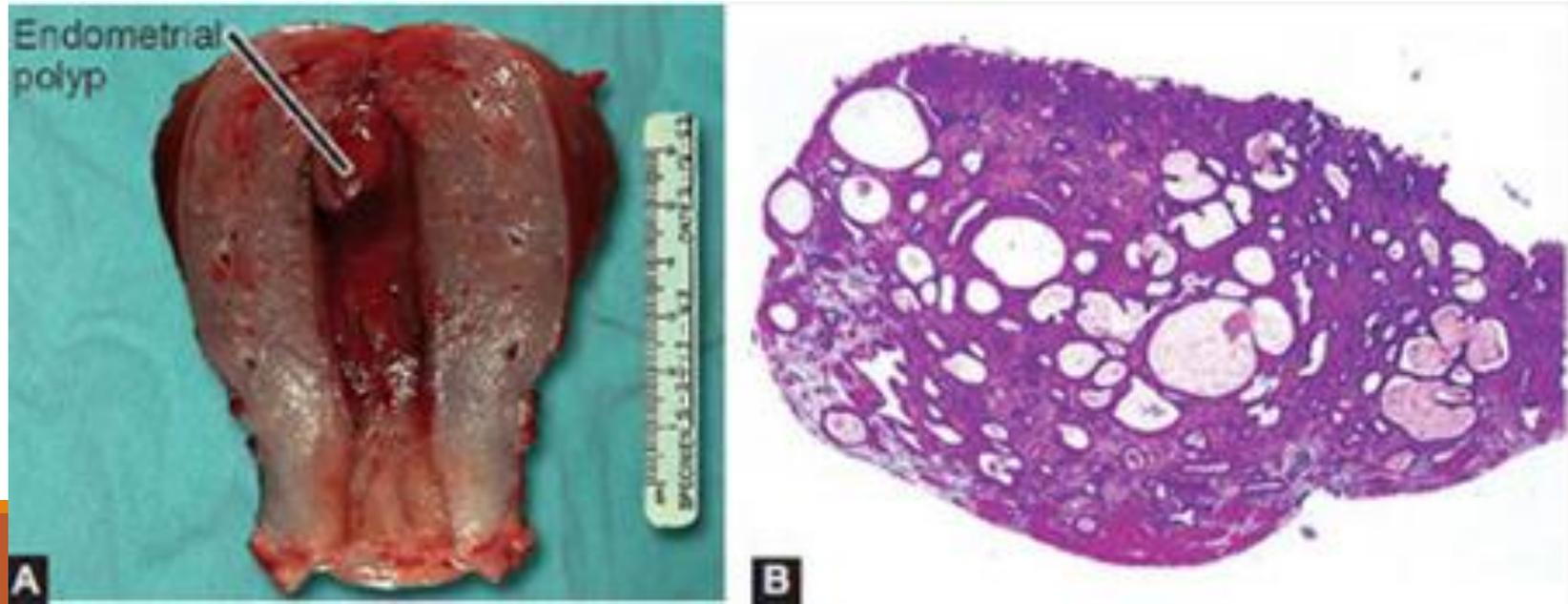
Hyperplasia with atypia

Tumors

- Endometrial tumors.
- Myometrial tumors.

1. Endometrial Polyps

- Exophytic masses of variable size that project into the endometrial cavity.
- Endometrial dilated (cystically) glands, with small muscular arteries and fibrotic stroma.
- Present with abnormal uterine bleeding.



2. Endometrial Carcinoma

- The most frequent cancer occurring in the female genital tract.
- Affecting female between 50s & 60s.

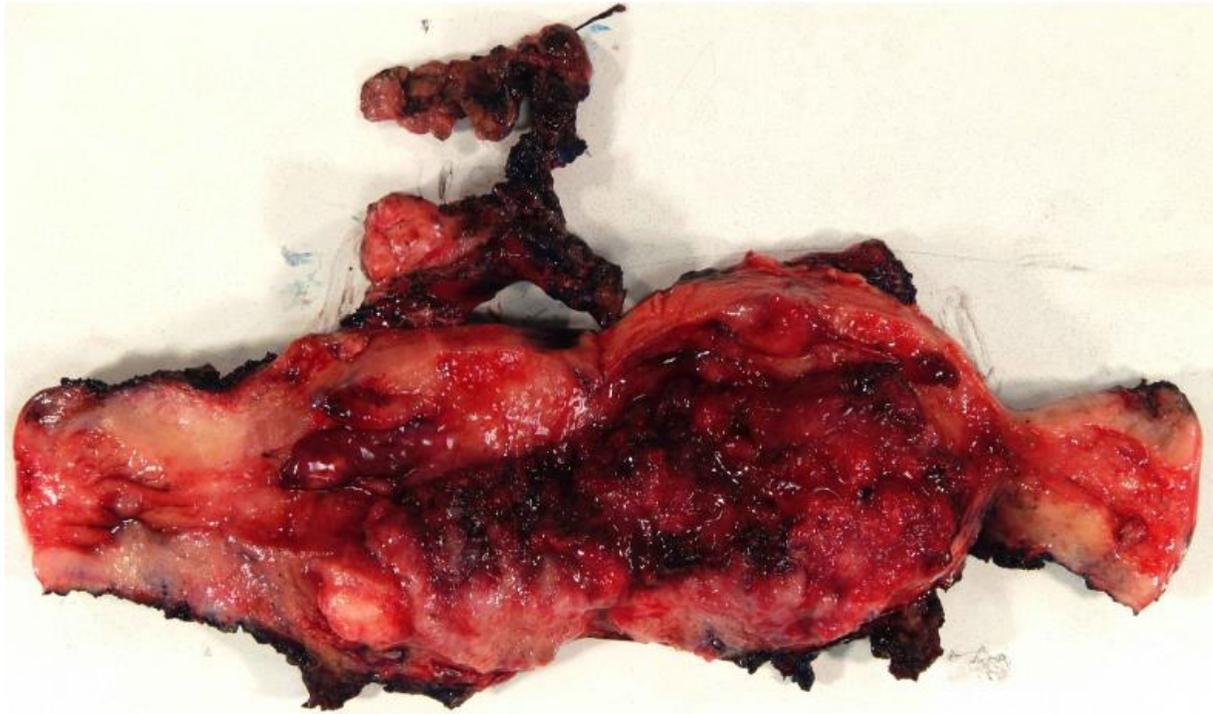
- Presentation: irregular or postmenopausal bleeding. With progression, the uterus enlarges.

- Two histological subtypes:
 - 1. Endometrioid carcinomas.
 - 2. Serous carcinoma.

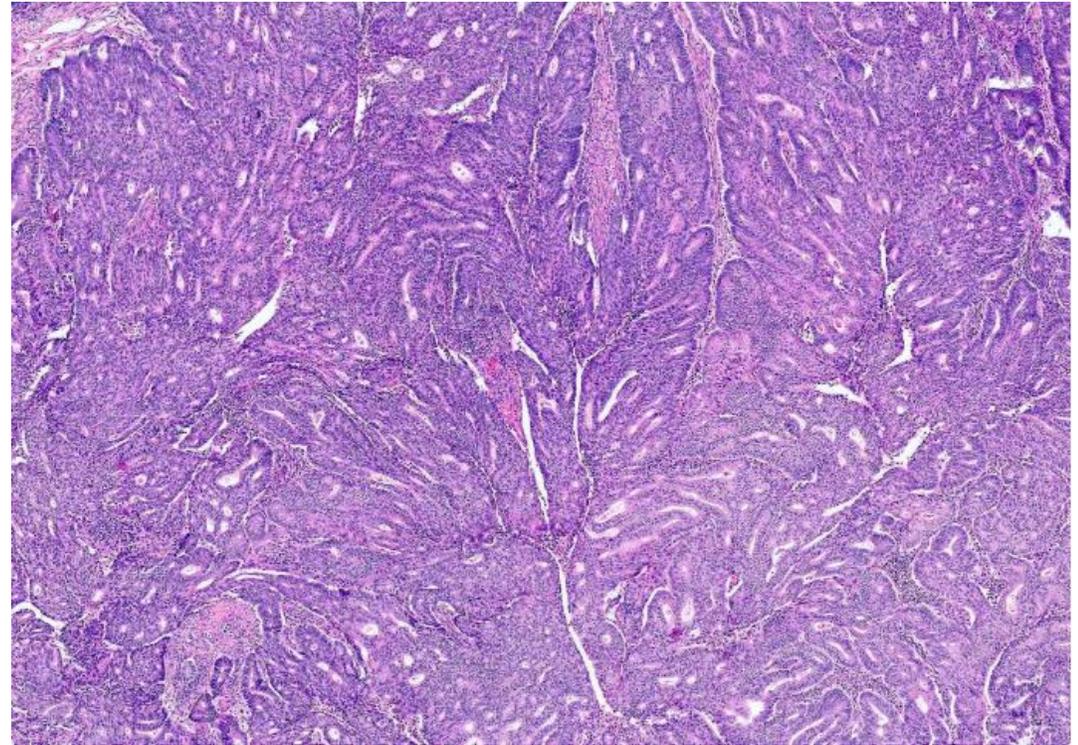
1. Endometrioid carcinomas.

- 80% of cases of endometrial carcinomas.
- Designated Endometrioid because of their histologic similarity to normal endometrial glands.
- Genetic: Mutations in mismatch repair genes & PTEN tumor suppressor gene.
- Risk factors:
 - (1) obesity.
 - (2) diabetes.
 - (3) hypertension.
 - (4) infertility.
 - (5) exposure to unopposed estrogen.
- Prognosis: slow to metastasize, but if untreated, eventually disseminates to regional nodes & distant sites.

Gross and microscopic features



#Friable, hemorrhagic mass occupying the endometrial cavity

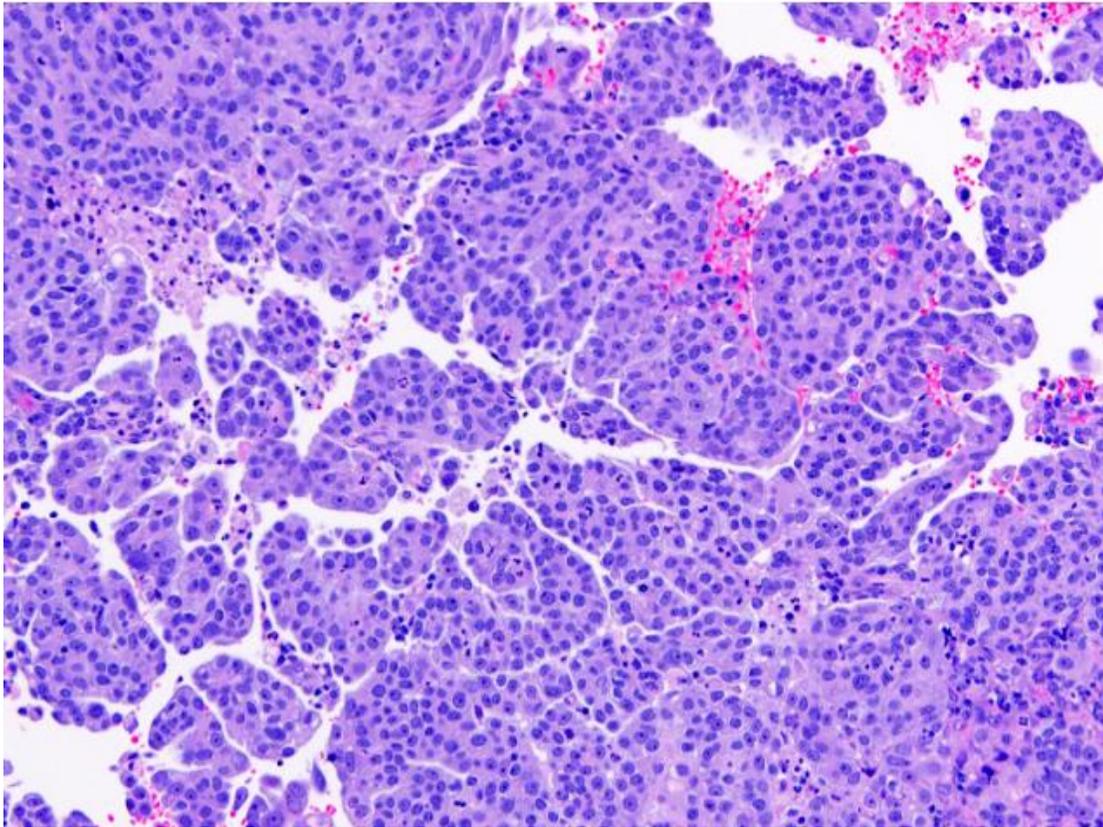


#back to back glands lacking intervening stroma.
#nuclear atypia.

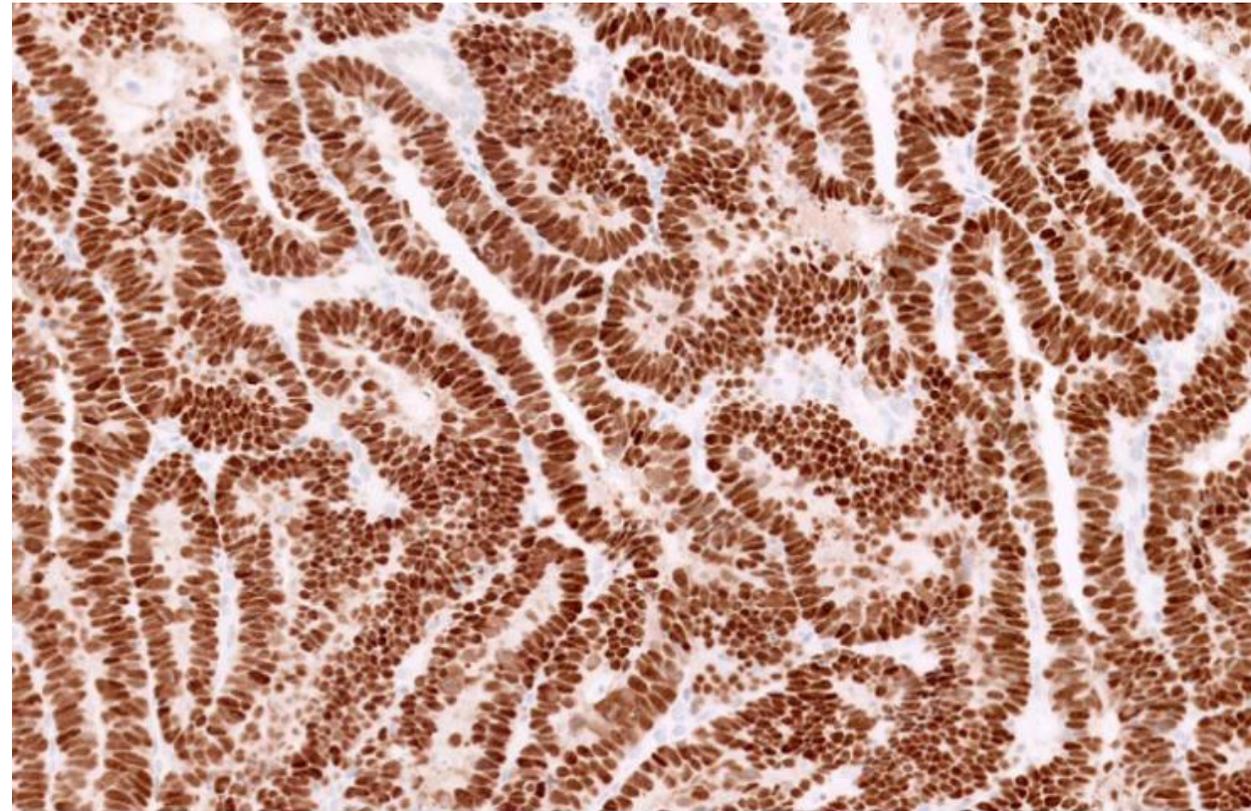
2. Serous carcinoma.

- Less common but far more aggressive.
- Not associated with unopposed estrogen or hyperplasia.
- Genetic: mutations in the TP53 tumor suppressor gene.
- Prognosis: strongly dependent on staging but because of its aggressive behavior → often high-stage disease with a poor prognosis.

Microscopic features



#Sheets and small papillae of endometrial serous carcinoma

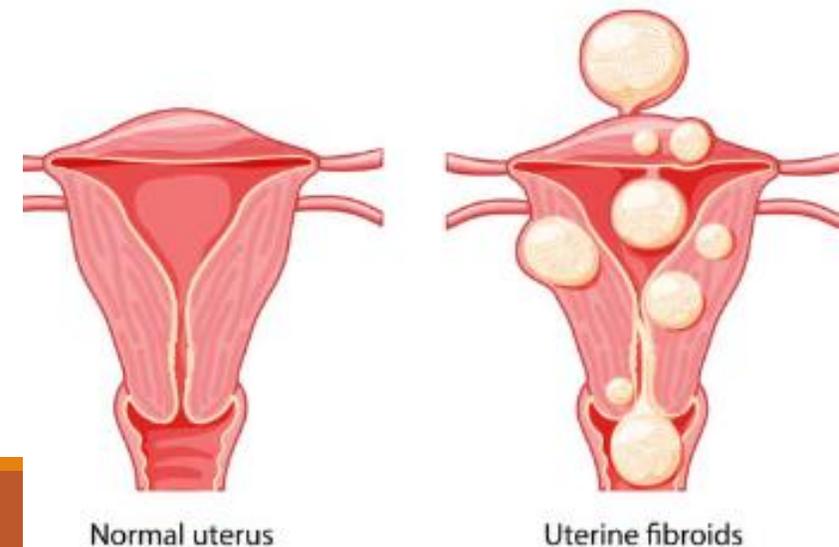


#Strong and diffuse overexpression of p53

Tumors of the Myometrium

1. Leiomyomas (fibroids)

- Benign tumors from the smooth muscle cells.
- The most common benign tumor in females, 30-50% of women of reproductive age.
- Estrogens stimulate the growth; shrink postmenopausally.
- Often asymptomatic, most frequent sign is menorrhagia.
- Rarely, if ever, transform into sarcomas, multiple lesions does not increase the risk of malignancy.



Gross morphology

sharply circumscribed, firm gray white masses with a characteristic whorled cut surface, often occur as multiple tumors.

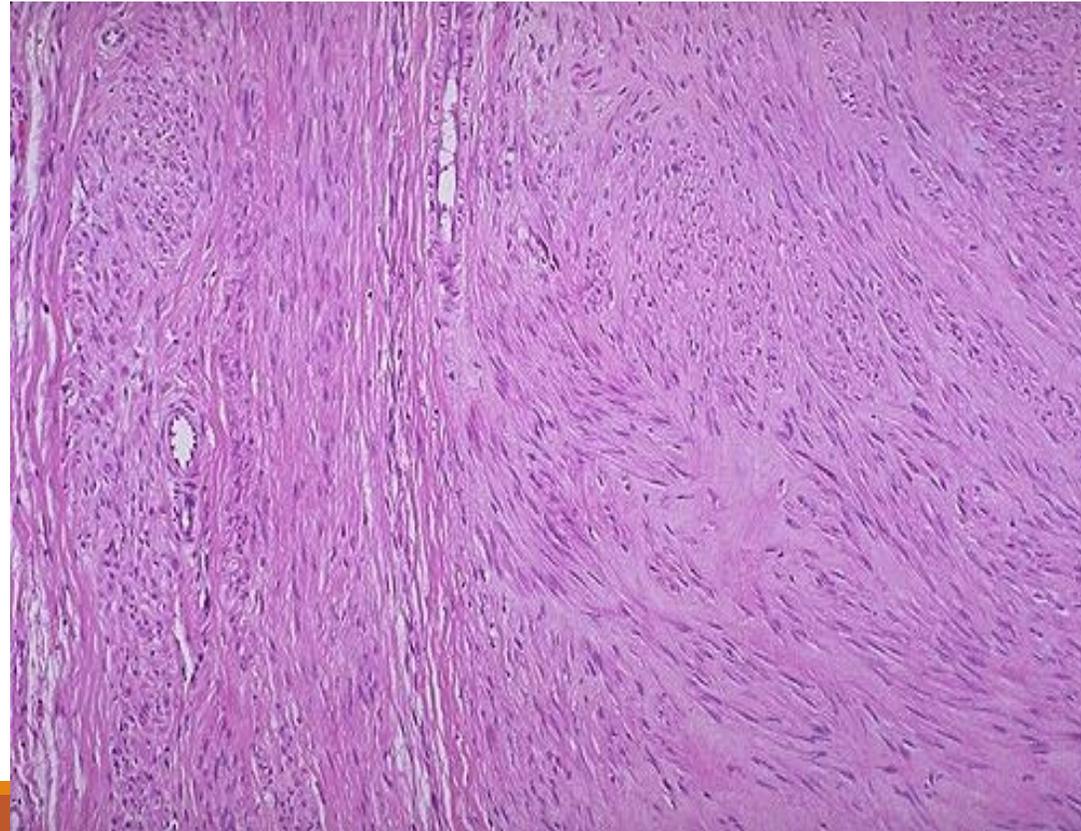
Possible location:

- Intramural.
- Submucosal.
- Subserosal.



Histological features

- Bundles of smooth muscle cells mimicking the appearance of normal myometrium

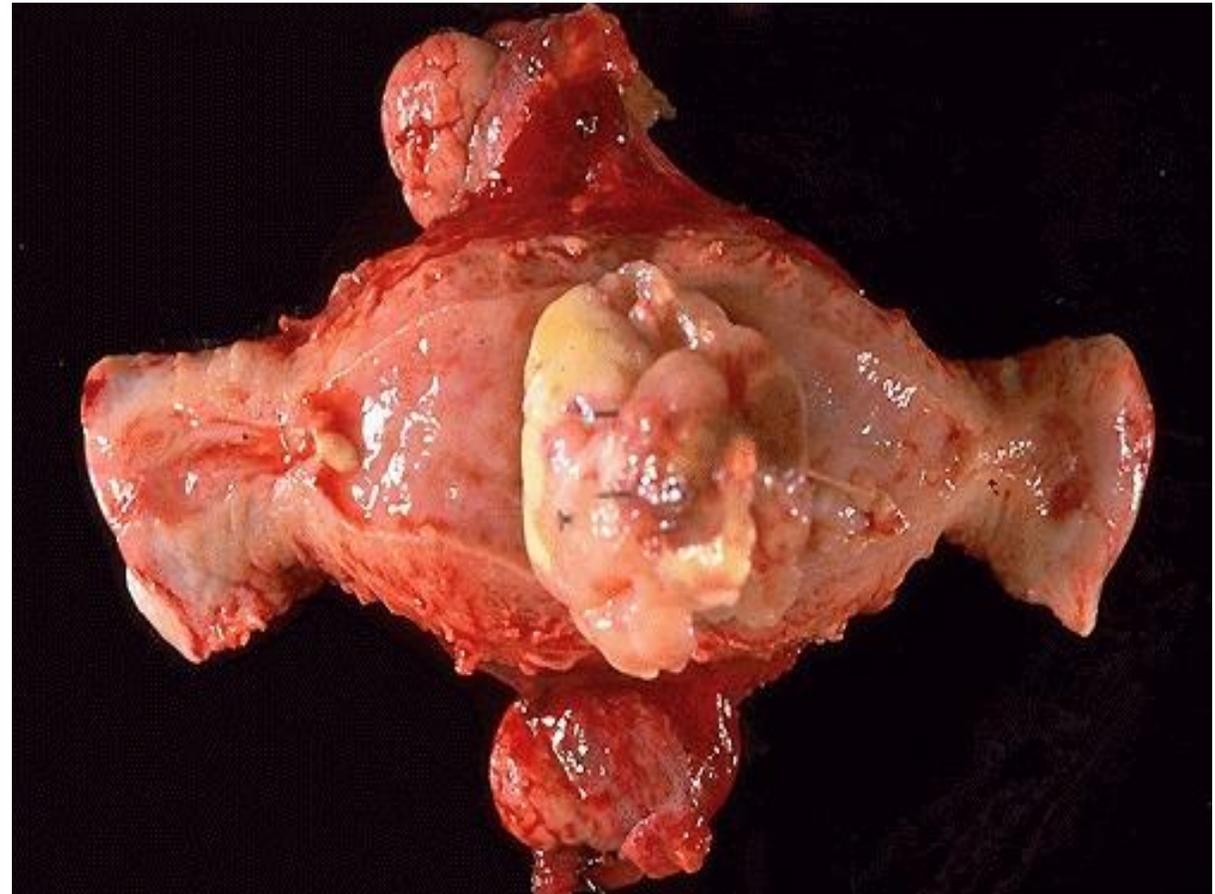


2. Leiomyosarcoma

- Malignant counterpart of Leiomyoma.
- Always arise de novo (not from previous Leiomyoma)
- Solitary and mostly in postmenopausal women.
- Recurrent is common & many metastasize, typically lungs.

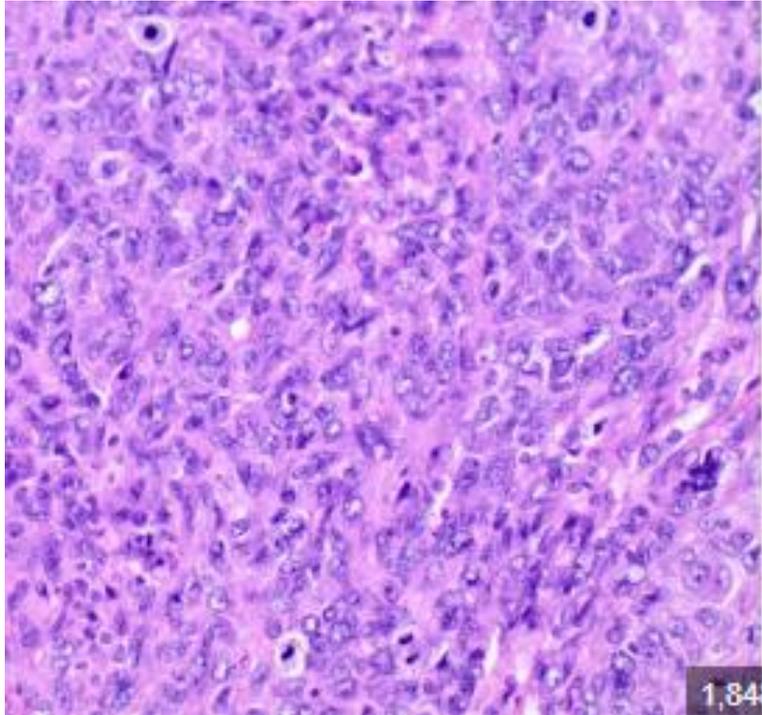
Gross features

soft, hemorrhagic, necrotic masses.
Irregular borders.

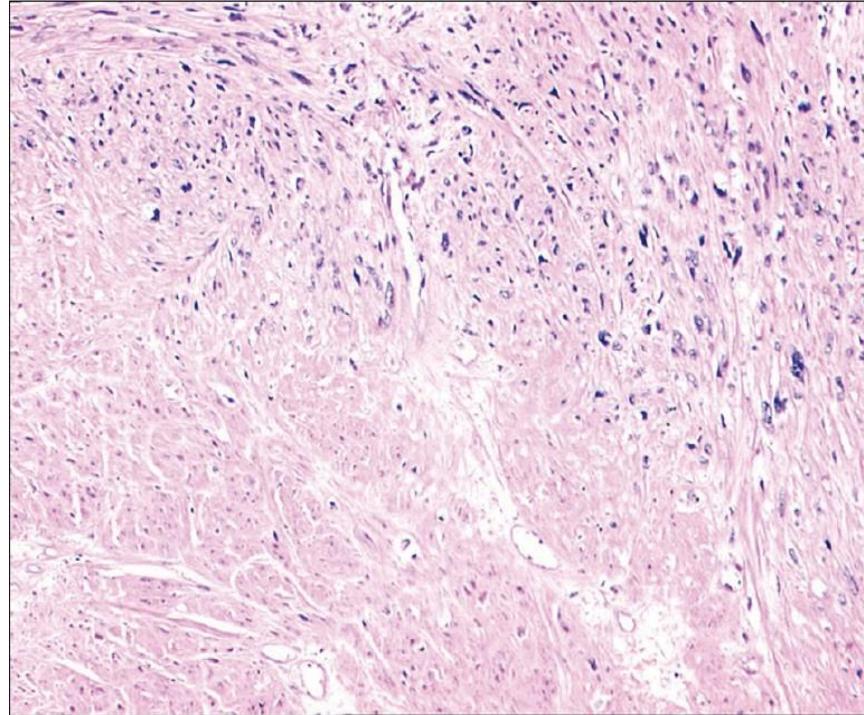


Microscopic features

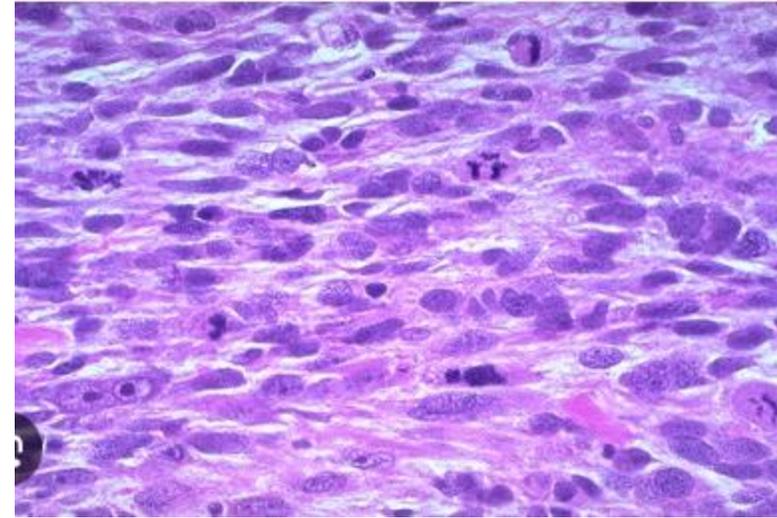
Diagnostic features of leiomyosarcoma:



cytologic atypia



tumor necrosis



mitotic activity