Pathology of the ovary

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Ovarian Neoplastic Diseases

- 5th most common cancer in women.
- 5th leading cause of cancer death in women.
- **3 Origins of primary ovarian tumors:**
 - 1 surface (coelomic) epithelium (most frequent)
 - 2 germ cells (affects children and young adults)
 - 3 sex cord/stromal cells.
- Each of these cell types gives rise to a variety of tumors

Ovarian Neoplasms

				Nonovarian primary tumor
RIGIN	SURFACE EPITHELIAL CELLS (Surface epithelial-stromal cell tumors)	GERM CELL	SEX CORD-STROMA	METASTASIS TO OVARIES
Verall frequency Proportion of	65%-70% (most frequer	nt) 15%-20%	5%-10%	5%
nalignant ovarian umors	90%	3%-5%	2%-3%	5%
ge group affected	20+ years	0-25+ years (affect and y	ts children All ages	Variable
ypes	 Endometrioid tumor Clear cell tumor 	Teratoma adults Dysgerminoma Endodermal sinus tumor Choriocarcinoma		

Pathogenesis-familial cases

- Risk factors: nulliparity and family history.
 ?? use of OCPs may <u>reduce</u> risk. / oral contraceptive
- Pills
 Only 5%-10% are familial
 Molecular pathogenesis: Mutations in
 BRCA 1 and 2 genes

Pathogenesis- sporadic cases

- BRCA mutations: 10% of sporadic cases other important molecular pathways:
 - **p53** (50%)
 - HER2/NEU over-expression (35%)
 - / K-RAS protein over-expression (30%) (mucinous)

SURFACE EPITHELIAL TUMORS-types:

- **1- Serous**
- **2- Mucinous**
- **3- Endometrioid**
- **4- Clear cell**
- **5- Brenner**

All types include <u>benign</u>, <u>borderline</u>, <u>and malignant</u> tumors

1- Serous Tumors

- **The most frequent ovarian tumors.**
- Include: 60% benign, 15% borderline, and 25% malignant.
- **The most common malignant ovarian tumors (60%)**
- Genetics:
- **BRAF** and **K-RAS** mutations→ borderline & low grade cancers
- **p53** and **BRCA1** mutations → High-grade serous carcinomas

Morphology

- l large cystic, (30 cm).
- May be bilateral.
- filled with a clear serous fluid
- single layer of columnar epithelium. Some cells are ciliated.
- B Psammoma

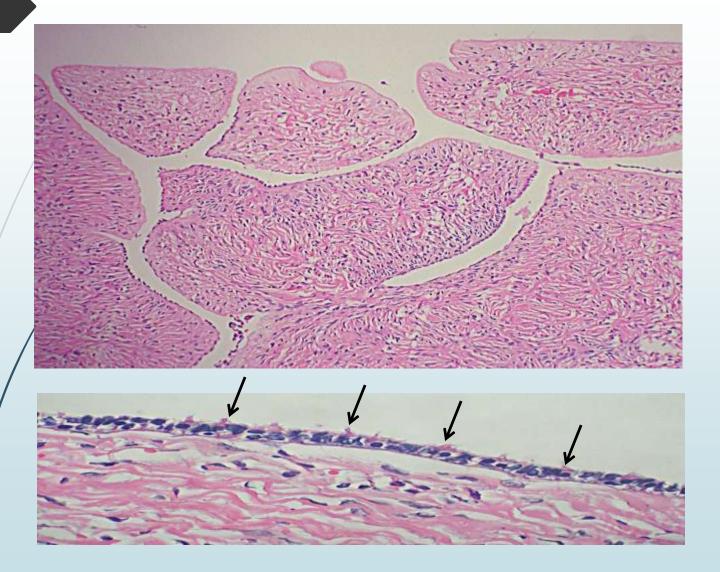
bodies (laminated calcified concretions) are common in tips of papillae of **all** serous tumors

SEROUS CYSTADENOMA





Benign serous tumors:



Border-line serous tumors

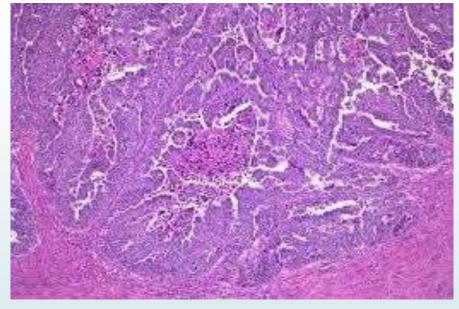
- more complex architecture / in histology so called papillary serous tumors
 mild cytologic atypia
- but no stromal invasion
- might be associated with peritoneal implants
- Prognosis intermediate between benign and malignant types (survival with peritoneal metastases 75%)





Malignant serous carcinoma

Anaplasia of cells and invasion of the stroma. prognosis poor, depends on stage at the time of diagnosis.

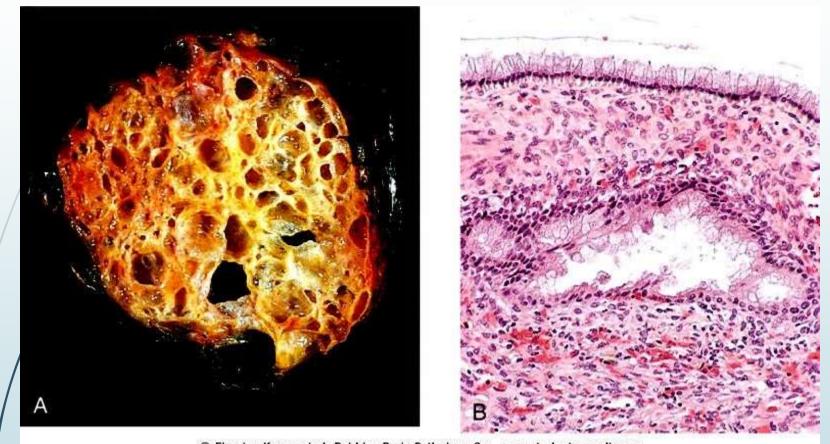


- Has necrosis and mitosis.

2- Mucinous ovarian tumors

- **mucin-secreting** cells.
- Depending on the architectural complexity:
- 80% benign; 10% borderline; 10% malignant(cystadenocarcinoma)
- **Usually large** and **multilocular**.
- psammoma bodies **not** found
- stage is major determinant of prognosis

Mucinous ovarian tumors



© Elsevier. Kumar et al: Robbins Basic Pathology 8e - www.studentconsult.com Histology: similar to GIT mucin producing cells (large bluish (due to mucin) cytoplasm

Germ cell tumors

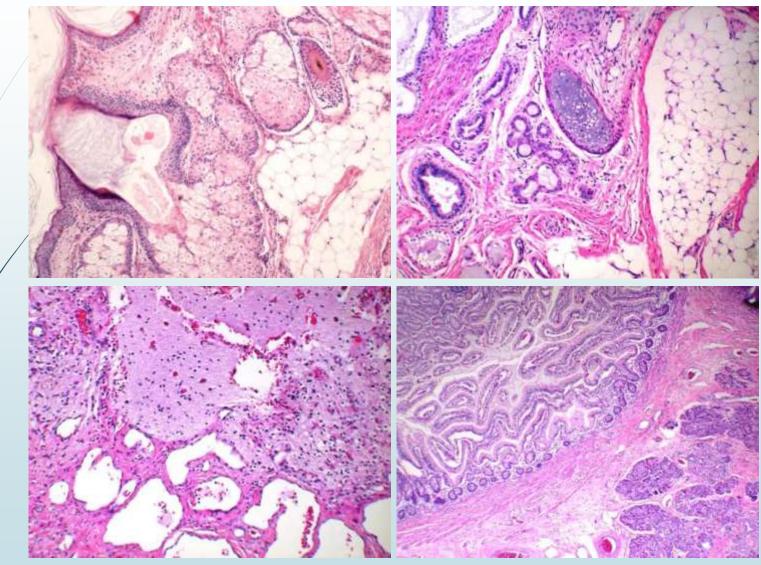
Benign (Mature) Cystic Teratomas:

- totipotential germ cells into mature tissues of all three germ cell layers
- Most discovered incidentally
- 90% unilateral
- Grossly: cyst filled with sebaceous secretion and hair; bone and cartilage; epithelium, or teeth./Because germ cells can differentiate to many tissues.
- ∎ 1% → malignant transformation
- I torsion (10% to 15% of cases)

Benign (Mature) Cystic Teratomas



Benign (Mature) Cystic Teratomas



Clinical Correlations for All Ovarian Tumors * clinical presentation of all is similar:

pain, gastrointestinal complaints, urinary frequency; rarely torsion producing severe abdominal pain mimicking an "acute abdomen."
Ascites (in Fibromas and malignant serous tumors).
Functioning ovarian tumors often come to attention because of hormonal production (Estrogens or androgens).

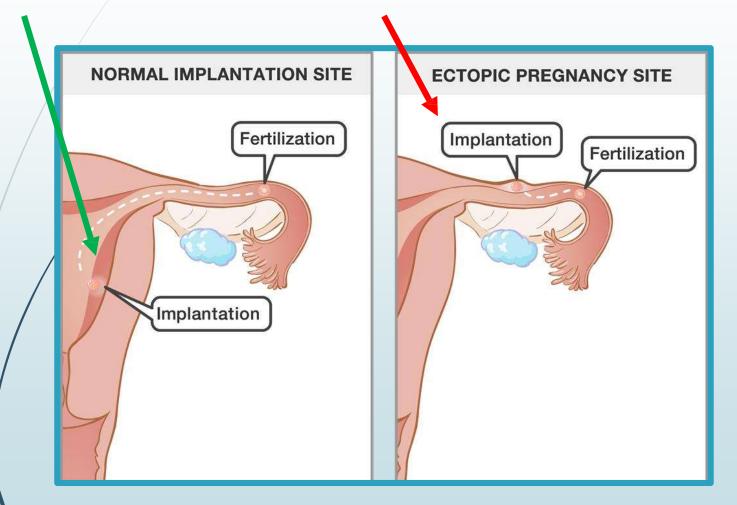
> torsion (twisting of massing pressing on vascular supply causing necrosis) Ascites (accumulation of fluid in peritoneal cavity)

Pathology of the Fallopian tubes

ECTOPIC PREGNANCY

- implantation of the fertilized ovum outside uterus
- Incidence: 1%
- 90% of cases \rightarrow in fallopian tubes
- other sites: ovaries, abdominal cavity
- Predisposing factors: tubal obstruction (50%) PID; inflammatory disease tumors; endometriosis; **IUCD**../Intra-uterine Contraceptive Device
- In 50% : no anatomic cause can be demonstrated.

Normal versus ectopic pregnancy



ECTOPIC PREGNANCY

- Early: development of the embryo and placental tissue
- Later: the placenta burrows through tubal wall causing intratubal hematoma (hematosalpinx) and intraperitoneal hemorrhage.

Rupture of an ectopic pregnancy: intense abdominal pain (acute abdomen), often followed by shock. Prompt surgical intervention is necessary.

Ectopic pregnancy- Management



Tubal malignancies

- considered rare.
- most common histo. type is serous carcinoma.
- Increased in women with <u>BRCA mutations</u> (In studies of prophylactic ophorectomies:10% →occult foci of malignancy in fimbria).
- Because of access to peritoneal cavity, fallopian tube carcinomas frequently spread to omentum and peritoneal cavity at time of presentation.

Thank you

Questions