Pathology lab -UGS MODULE

DR OMAR HAMDAN

في هذا الملف ستجدون تجميع لصور الأمراض من محاضرات الدكتور عمر مع تبييضهم

قام على إنجاز هذا العمل: عمران يونس شهد الايوبين سان محمود



Autosomal dominant polycystic kidney disease

Gross description

Markedly enlarged kidneys (up to • 8 kg) composed of <u>sub-capsular</u> cysts up to 4 cm

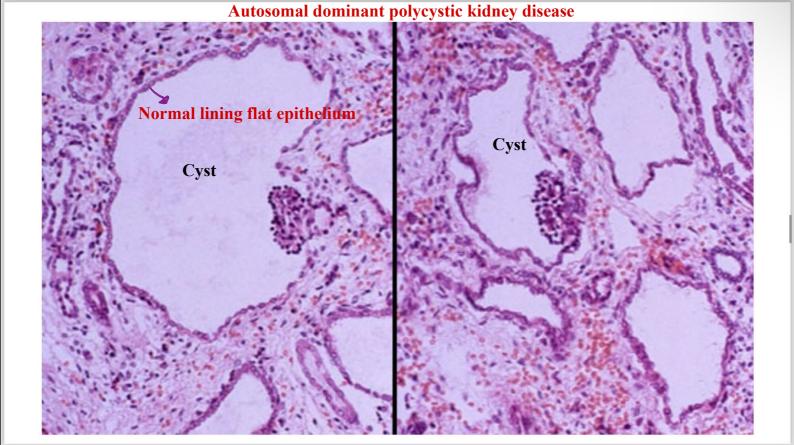
Cysts contain clear to brown fluid •

No invasion of renal capsule by cysts







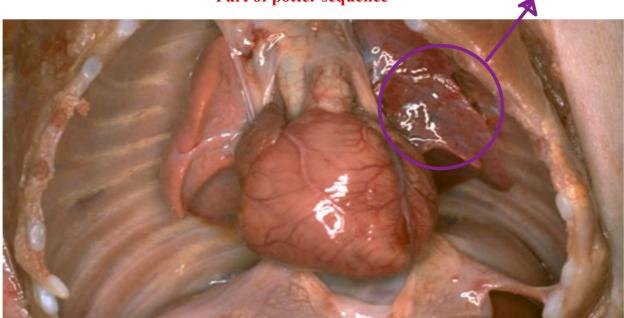


With autosomal recessive polycystic kidney disease

Lung

Pulmonary hypoplasia

Part of potter sequence

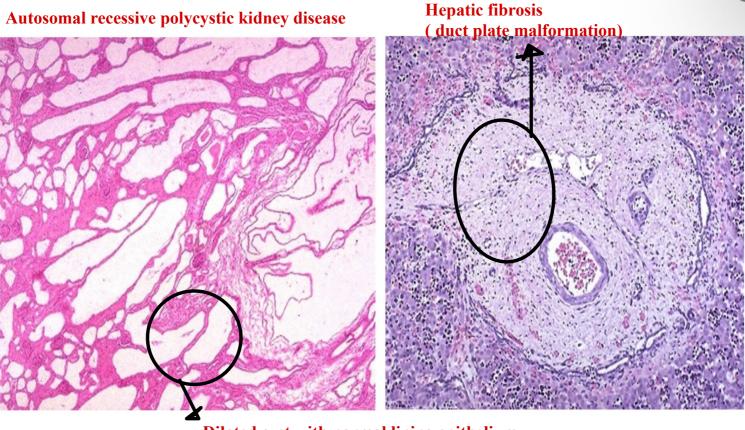


Autosomal recessive polycystic kidney disease

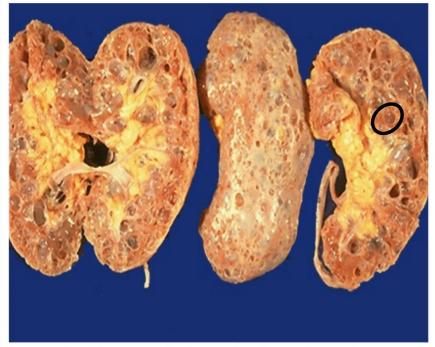
Small cyst



Smooth surface without capsule invasion



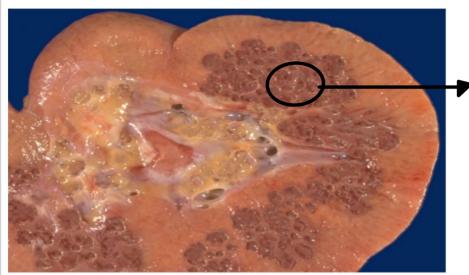
Dilated cyst with normal lining epithelium



These adult kidneys are about normal in size but have a few scattered **small cysts**, none of which is over 2 cm in size. This is cystic change associated with chronic renal dialysis.

Acquired recessive polycystic kidney disease

Cyst



Medullary spongy kidney

• Cyst

Note the **0.1 to 0.5 cm cysts** involving the inner medullary and papillary regions in this kidney. Note that the cortex appears normal. This is medullary sponge kidney (MSK), which is congenital, but most often occurs sporadically without a defined inheritance pattern. It is often bilateral, but incidental and found only on radiologic imaging studies, with an incidence of 0.5 to 1% in adults. MSK may become symptomatic in young adults, with onset of recurrent hematuria and/or urinary tract infection as a consequence of formation of calculi, which develop in 60% of cases. Renal failure is unlikely to occur, but may result from severe pyelonephritis.



Simple renal cysts, as seen here, have thin walls and are fluid filled. They can be multiple, but they are never as numerous as with polycystic change, and they do not predispose to chronic renal failure or to neoplasia. Such simple cysts become more common as persons become older.



This is a multicystic dysplastic kidney. This condition must be distinguished from ARPKD because it occurs only sporadically and not with a defined inheritance pattern, though it is more common than ARPKD. The cysts of multicystic renal dysplasia are larger and more variably sized than those of ARPKD. Often, multicystic renal dysplasia is unilateral. If bilateral, it is often asymmetric. If bilateral, oligohydramnios and its complications can ensue, just as with ARPKD.



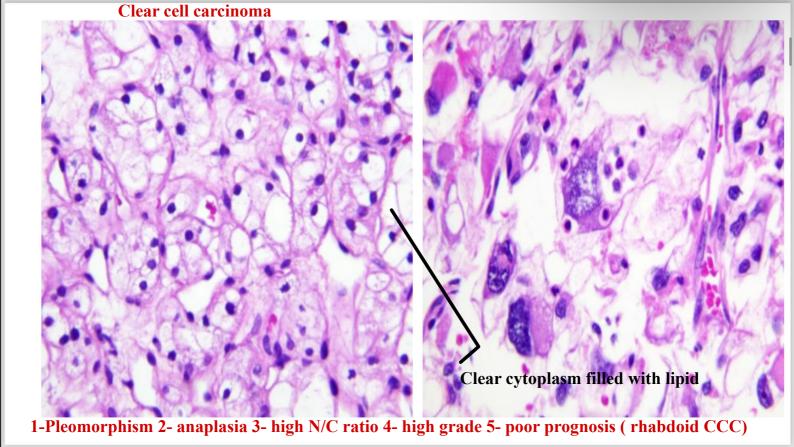
Cystic change resulting from long-term renal dialysis may rarely give rise to renal cell carcinoma. A large irregular tan variegated **mass** is seen here on sectioning of a kidney that has large **cysts** arranged around the mass.

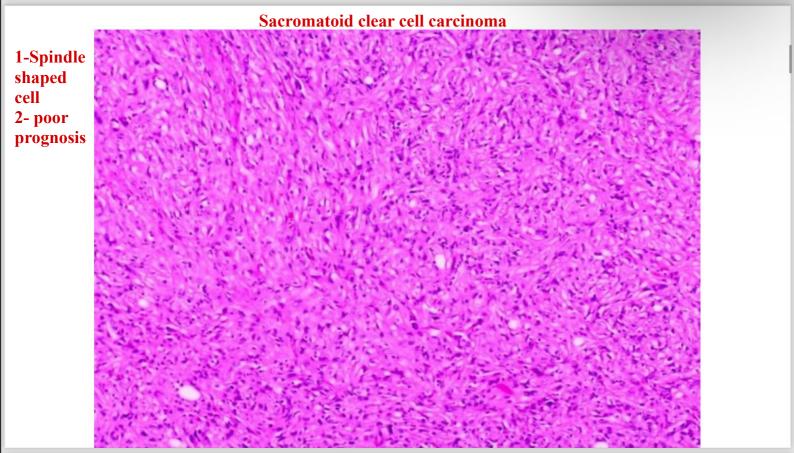
- Renal cell carcinoma

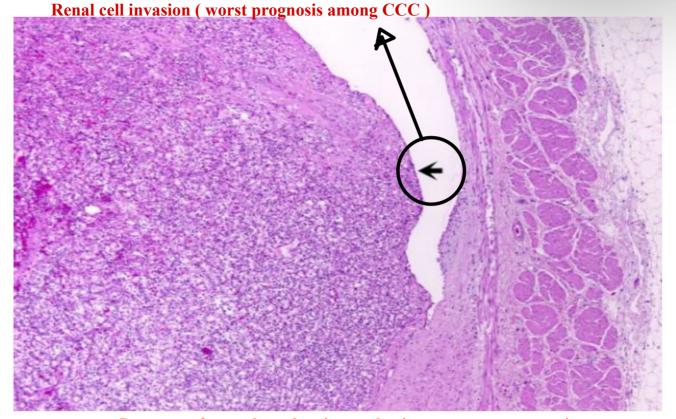


Clear cell carcinoma

- 1- golden yellow 2- lipid filled 3- VHL gene on chromosome 3

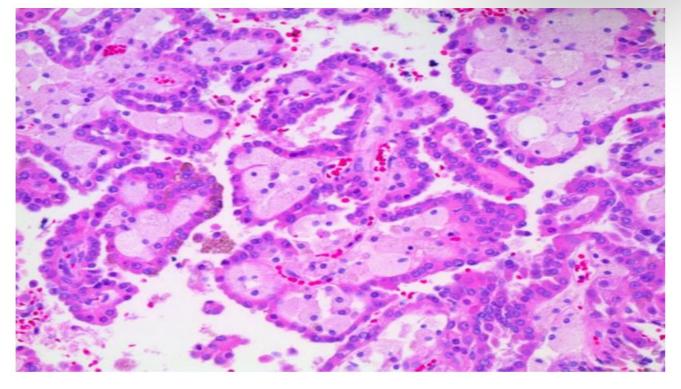




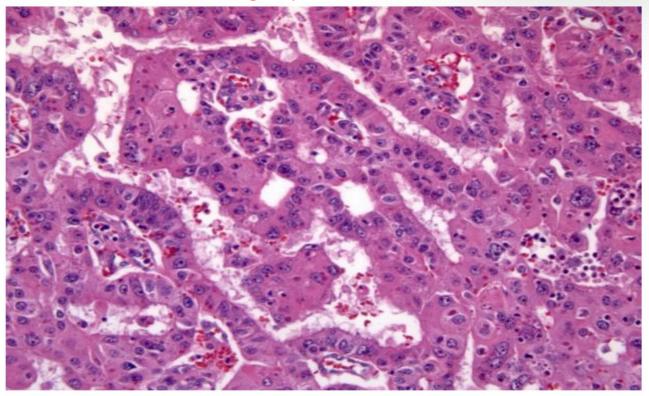


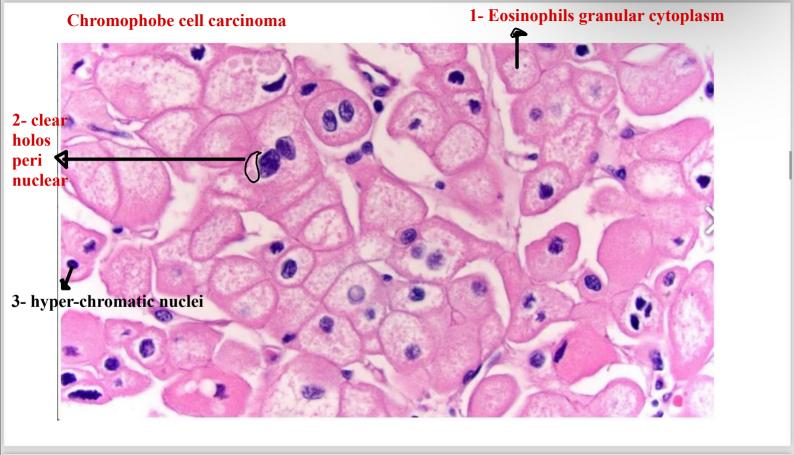
Presence of even thrombus in renal vein mean poor prognosis

Papillary cell carcinoma (determined by histopathological architecture)



Papillary cell carcinoma





Triphasic histological pattern: 1- blatesma (small round blue cell) Wilms tumor

Wilms tumor **Triphasic histological pattern:** 2- Epithelial tubules & glomeruli

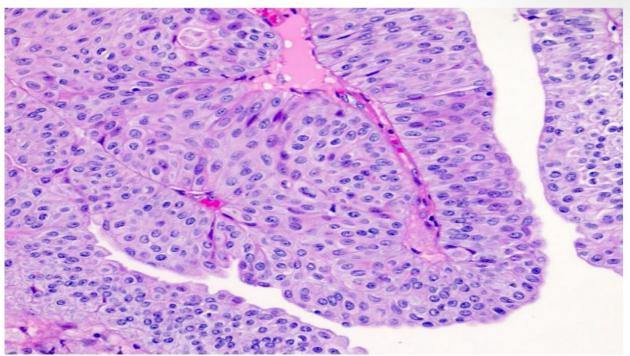
3- Stromal or mesenchymal **Triphasic histological pattern:** Wilms tumor



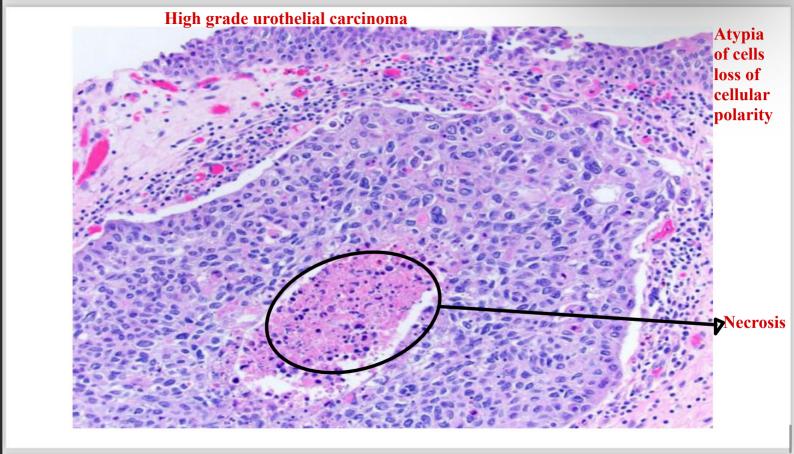


Papillary urothelial carcinoma (most common urinary bladder tumor)

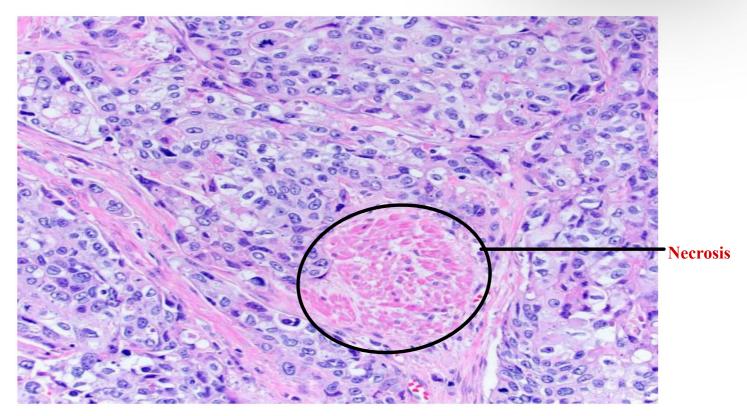
Low grade urothelial carcinoma



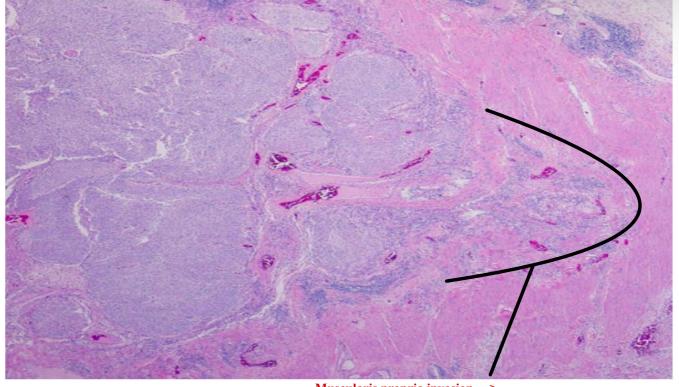
Cells are polarized & arranged



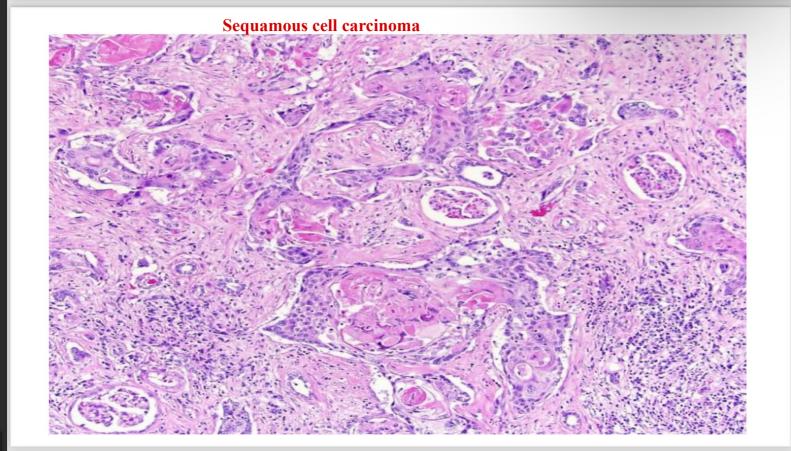
High grade urothelial carcinoma

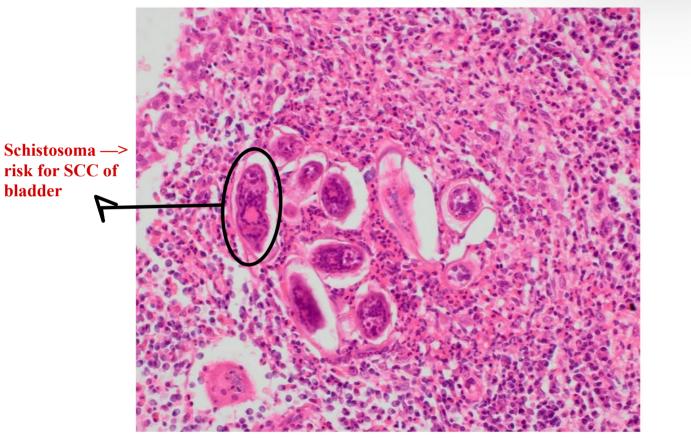


High grade urothelial carcinoma with muscle invasion

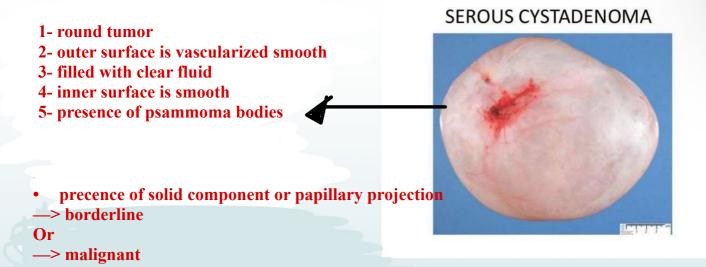


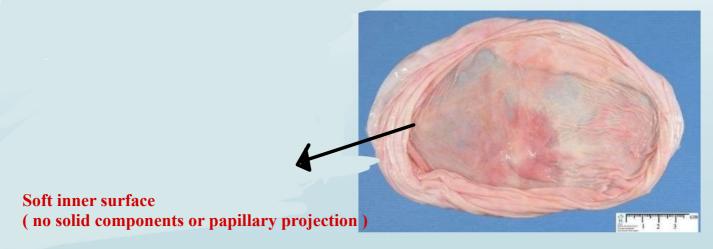
Muscularis propria invasion —> Treatment cystectomy or chemotherapy



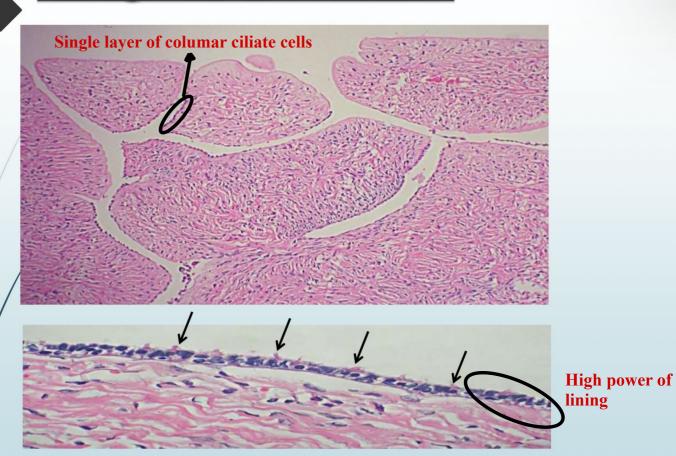


bladder





Benign serous tumors:



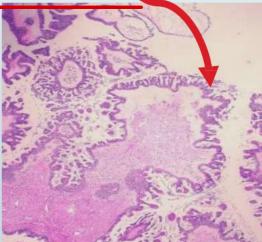
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Border-line serous tumors

- more complex architecture / in histology so called papillary serous tumors mild cytologic atypia
- but **no** stromal So its still not carcinoma invasion
- might be associated with peritoneal implants
- Prognosis intermediate between benign and malignant types (survival with peritoneal metastases 75%)

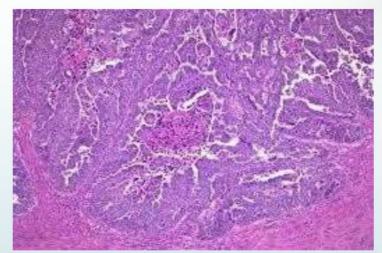
Projection of papillary solid component





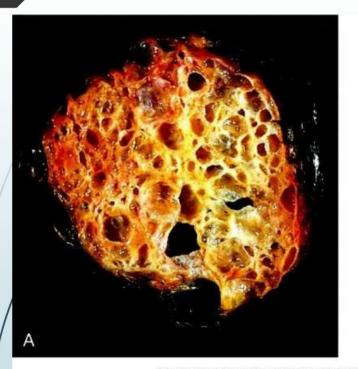
Malignant serous carcinoma

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

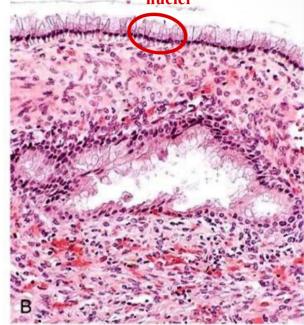


⁻ Has necrosis and mitosis.

Mucinous ovarian tumors



Apical mucin with basal nuclei

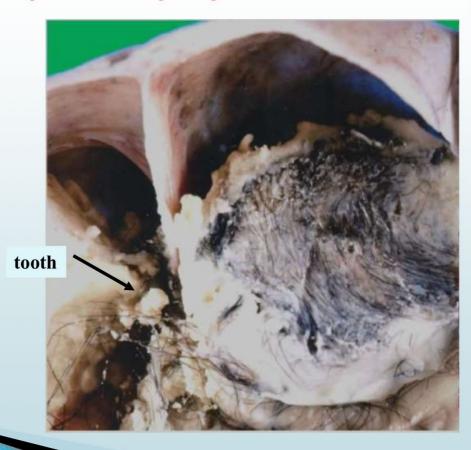


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Histology: similar to GIT mucin producing cells (large bluish (due to mucin) cytoplasm

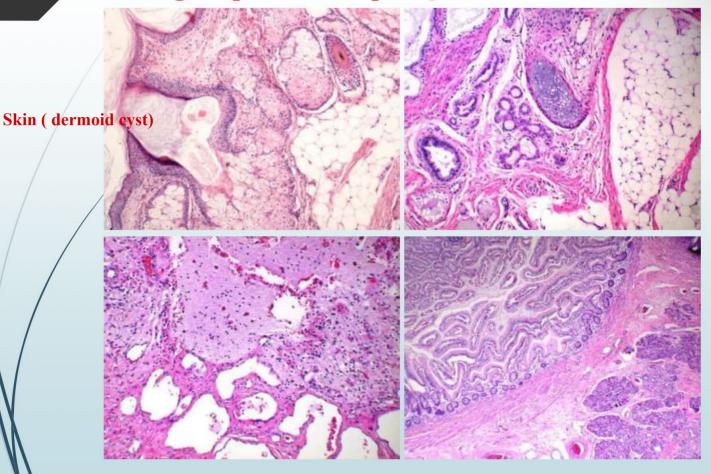
Psammoma bodies are not found in this tumor

Benign (Mature) Cystic Teratomas



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Benign (Mature) Cystic Teratomas

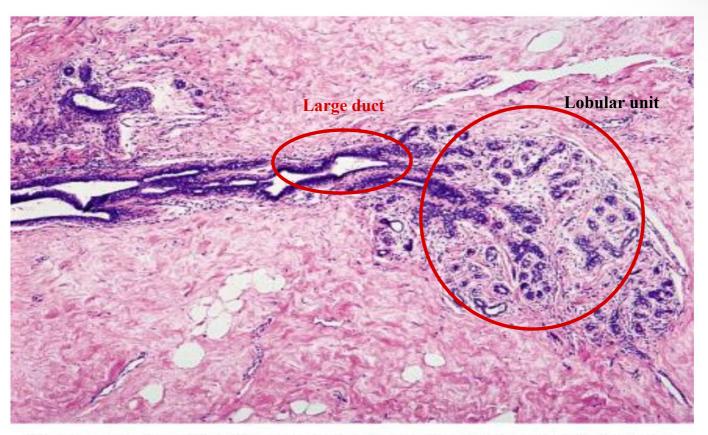


Ectopic pregnancy- Management



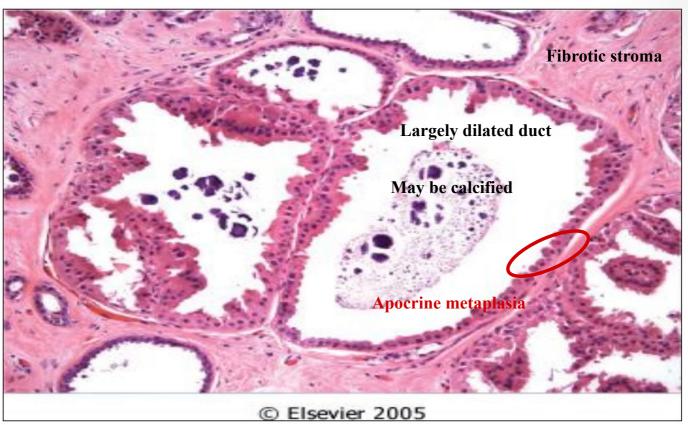
Salpintomy (removal of fallopian tube) id the clinical management of this condition

Normal breast



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Fibrocystic change of breast

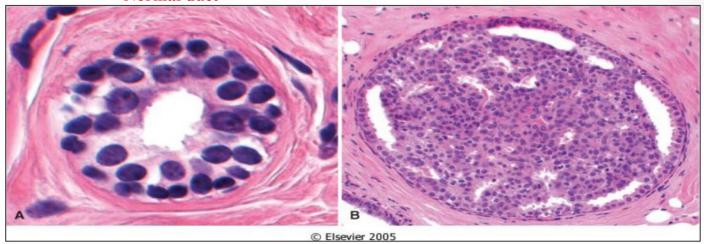


Apocrine metaplasia mean normal myoepithelial cell layer —> eosinophilic cells

Figure 23-7 Apocrine cysts. Cells with round nuclei and abundant granular eosinophilic cytoplasm, resembling the cells of normal apocrine sweat glands, line the walls of a cluster of small cysts. Secretory debris, frequently with calcifications, is often present. Groups of cysts are common findings associated with clustered mammographic calcifications.

Normal duct

Epithelial hyperplasia



Epithelial hyperplasia: The proliferating epithelium, often including both luminal and myoepithelial cells, fills and distends the ducts and lobules.

Figure 23-8 A, Normal. A normal duct or acinus has a single basally located myoepithelial cell layer (cells with dark, compact nuclei and scant cytoplasm) and a single luminal cell layer (cells with larger open nuclei, small nucleoli, and more abundant cytoplasm). B, Epithelial hyperplasia. The lumen is filled with a heterogeneous population of cells of different morphologies, often including both luminal and myoepithelial cell types, irregular slitlike fenestrations are prominent at the periphary.

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1- ducts 2- fibrosis

Sclerosing Adenosis.

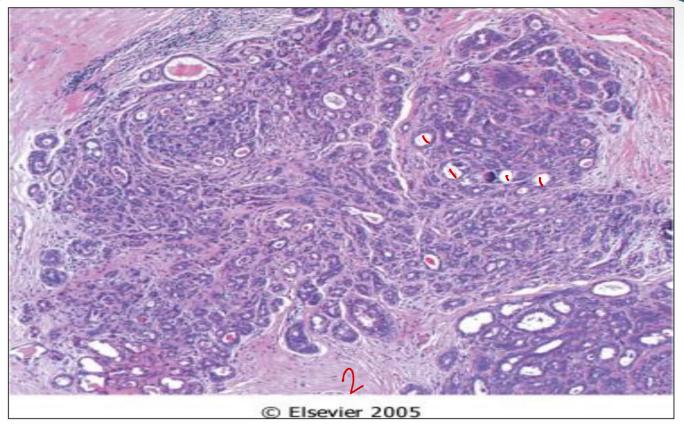


Figure 23-9 Sclerosing adenosis. The involved erminal duct lobular unit is enlarged, and the acini are compressed and distorted by the surrounding dense stroma. Calcifications are often present within the lumens. Although this lesion is frequently mistaken for an invasive carcinoma, unlike carcinomas, the acini are arranged in a swirling pattern, and the outer border is usually well circumscribed.

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Complex Sclerosing Lesion (Radial Scar)

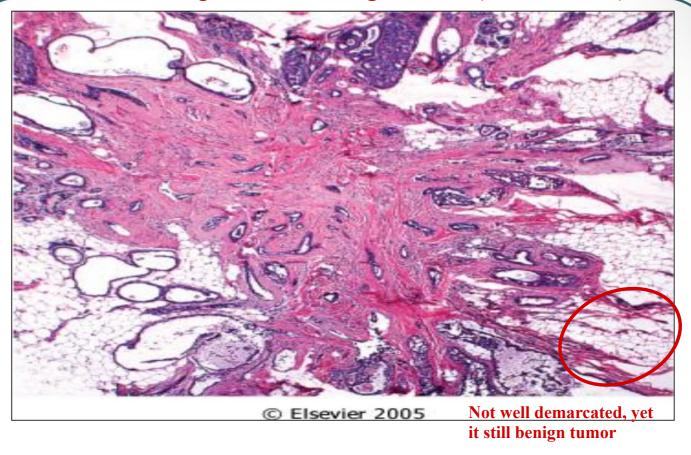
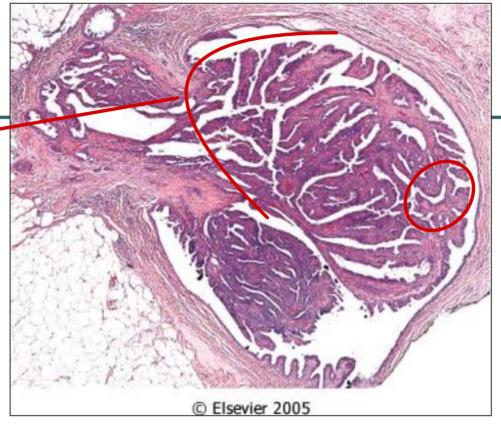


Figure 23-10 Complex sclerosing lesion (radial scar). There is a central nidus consisting of small tubules entrapped in a densely fibrotic stroma surrounded by radiating arms of epithelium with varying degrees of cyst formation and hyperplasia. These lesions typically present as an irregular mammographic density and closely mimic an invasive carcinoma.

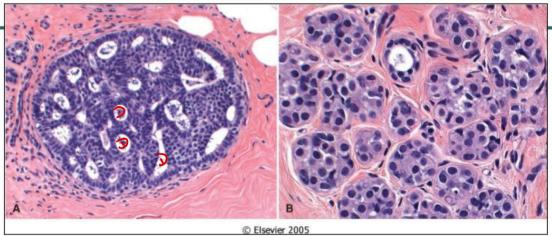
Cyst with benign lining



Papillary structure in the cyst

Once the myoepithlial layer in breast tumor—> malignancy

Figure 23-11 Intraductal papilloma. A central fibrovascular core extends from the wall of a duct. The papillae arborize within the lumen and are lined by myoepithelial and luminal cells.

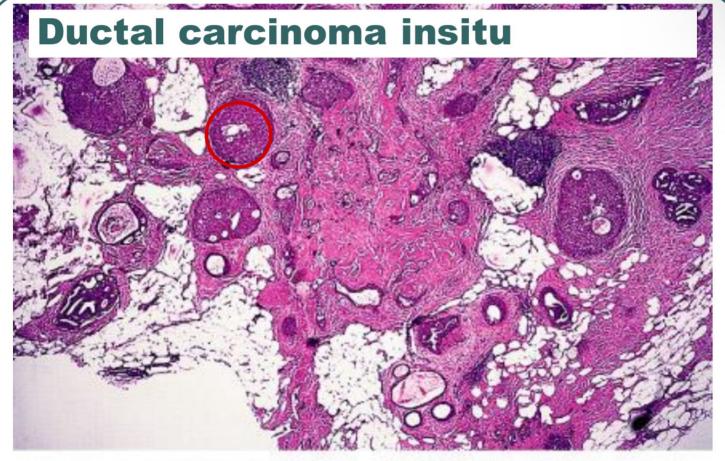


Space are widely scattered everywhere in the lesion—> ductal hyperplasia with atypia

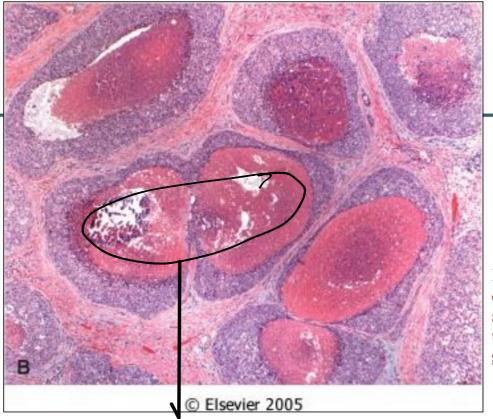
Lobular hyperplasia with atypia

Figure 23-12 A, Atypical ductal hyperplasia. A duct is filled with a mixed population of cells consisting of oriented columnar cells at the periphery and more rounded cells within the central portion. Although some of the spaces are round and regular, the peripheral spaces are irregular and slittlike. These features are highly atypical but fall short of a diagnosis of DCIS. B, Atypical lobular hyperplasia. A population of monomorphic small, rounded, loosely cohesive cells partially fill a lobule. Some intracellular lumina can be seen. Although the cells are morphologically identical to the cells of LCIS, the extent of involvement is not sufficient for this diagnosis.

Ducts fill of cell



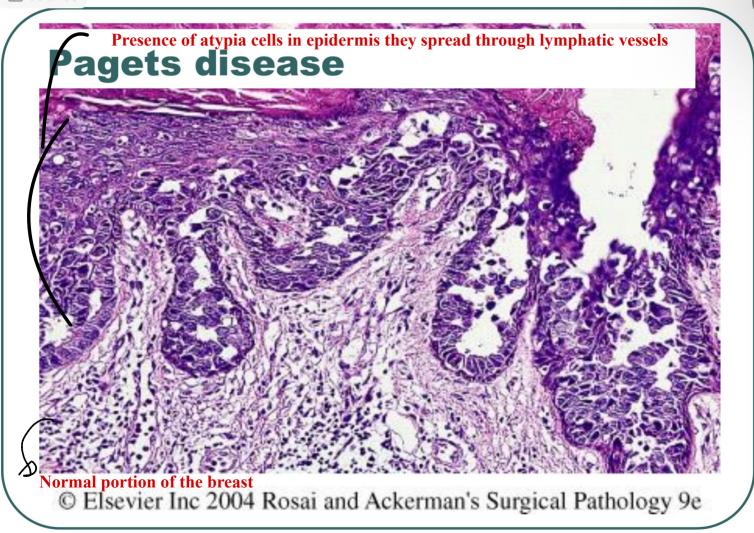


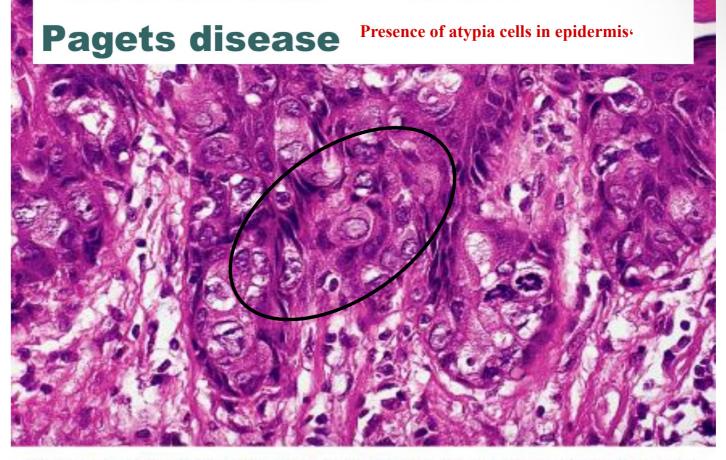


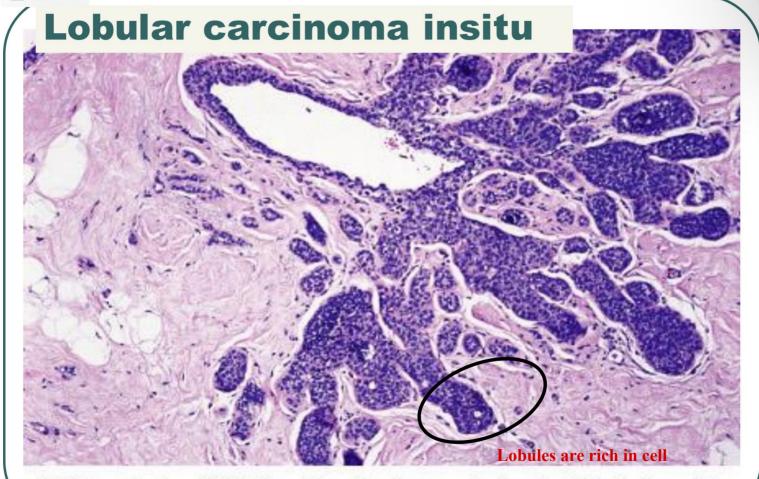
Presence of comedo necrosis always make the tumor of high grade

Called comedo necrosis (its darker in color than the surrounding)

Figure 23-16 A, This mammogram reveals multiple clusters of small, irregular calcifications in a segmental distribution. Suspicious calcifications must be biopsied, as 20% to 30% will prove to be due to DCIS. B, Comedo DCIS fills several adjacent ducts (or completely replaced lobules) and is characterized by large central zones of necrosis with calcified debris. This type of DCIS is most frequently detected as radiologic calcifications. Less commonly, the surrounding desmoplastic response results in an ill-defined palpable mass or a mammographic density.







© Elsevier Inc 2004 Rosai and Ackerman's Surgical Pathology 9e Lobules in lobular carcinoma are usually smaller than ducts of ductal carcinoma

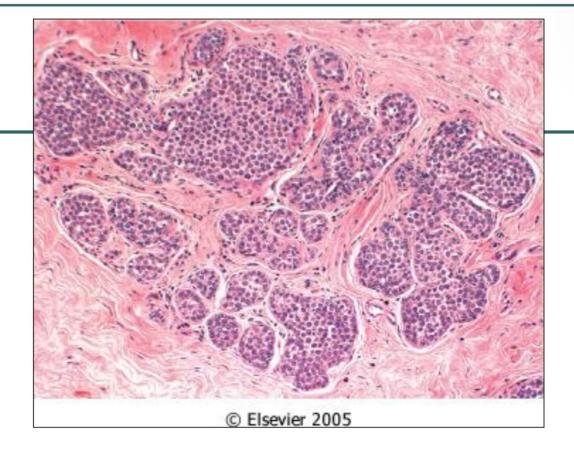
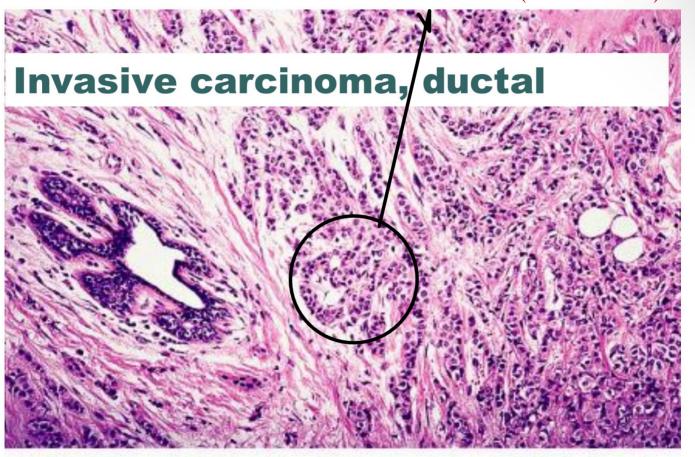
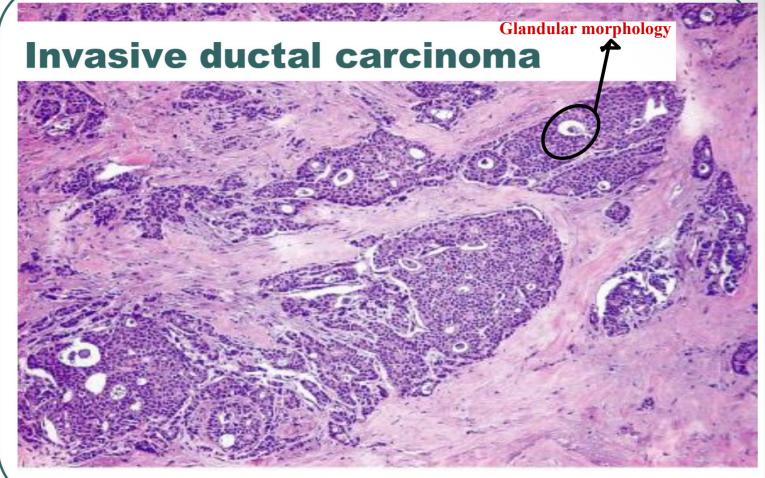


Figure 23-20 Lobular carcinoma in situ. A monomorphic population of small, rounded, loosely cohesive cells fills and expands the acini of a lobule. The underlying lobular architecture can still be recognized.

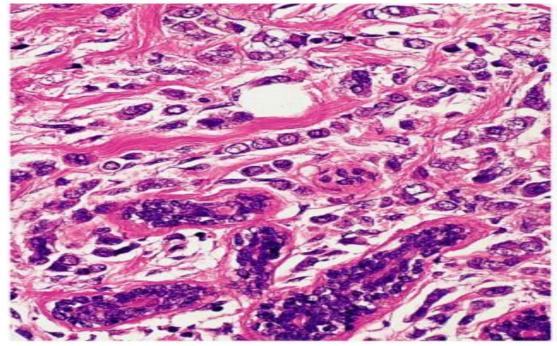
Invasive carcinoma METRIC 1

Glandular tissue (adenocarcinoma)





Invasive lobular carcinoma

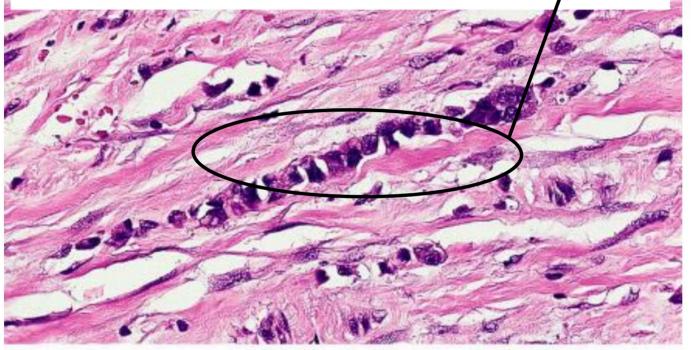


To differentiate between late stage of lobular & ductal carcinoma we use E- cadhern stain

• E-cadhern negative (lobular)

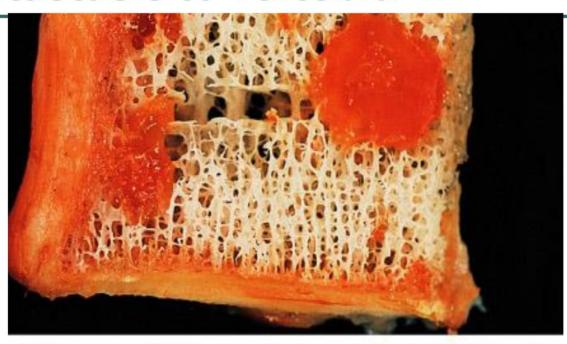
Arranged cells are cancer cell

Invasive lobular carcinoma /



Common

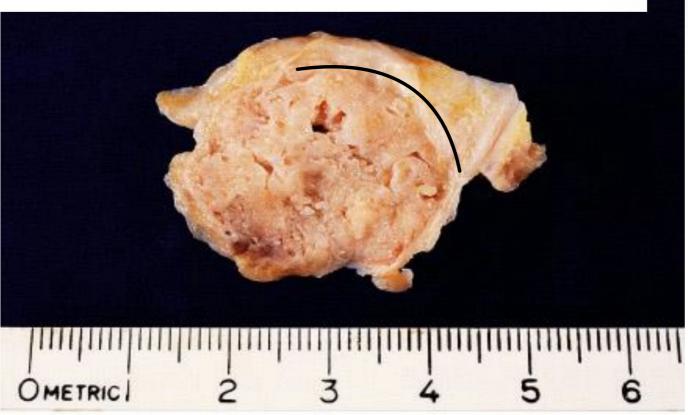
Metastasis to vertebra

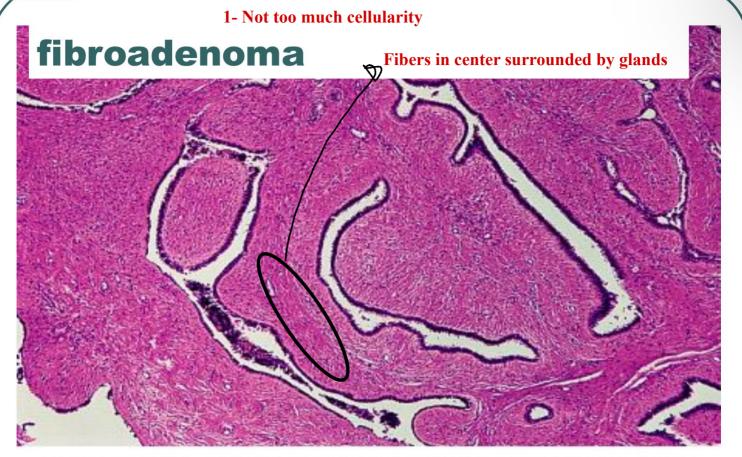


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fibroadenoma

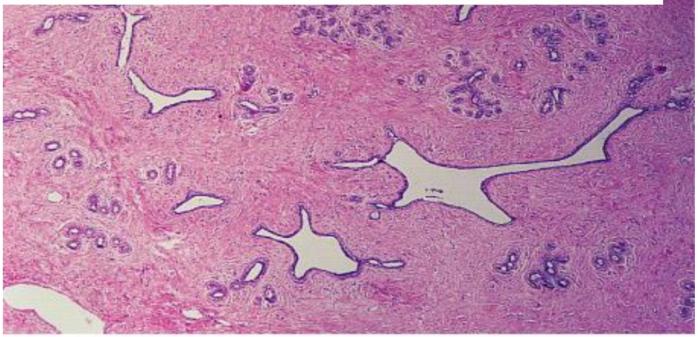
Well demarcated





fibroadenoma

Fibers surround by gland



"وذلِّل كل صعبٍ في خُطانا وبلّغنا تباشيرَ الوصولِ"

ها قد هان ما ظنناه مستحیلًا!

مشاعر مختلفة ما بين أول لاب في كلية الطب وبين آخرهم ؛ اقترب النهايات

السعيدة التي تُقرُّ بها أعيننا وتلتهف لها قلوبنا

نختتم معكم آخر لاب في كلية الطب؛ متمنين لكم التوفيق والرضا

تحية إجلال للمحاربين الذين وصلوا الى هنا، نسأل الله لكم التوفيق في إختباركم غدا وفتح

عليكم فتوح العارفين وألهمكم الصواب فالجواب

مع تحيات#الفريق_الأكاديمي

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