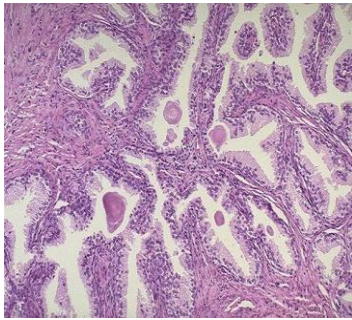


## *OSPE / Pathology*



**Done by:**

**Pathology Group -2013**

**\* نقاط موجزة أتمنى من الجميع معرفتها قبل البدء بدراسة الملخص :**

**أولاً :** تم العمل على هذا الملخص من قبل مجموعة من الطلاب "جزاهم الله عنا كل خير" , و لضيق الوقت لم نتمكن من إرساله و مراجعته مع دكتور جمال , و لكننا نعد أن نقوم بذلك بعد إجازة عيد الأضحى " بإذن الله تعالى " .

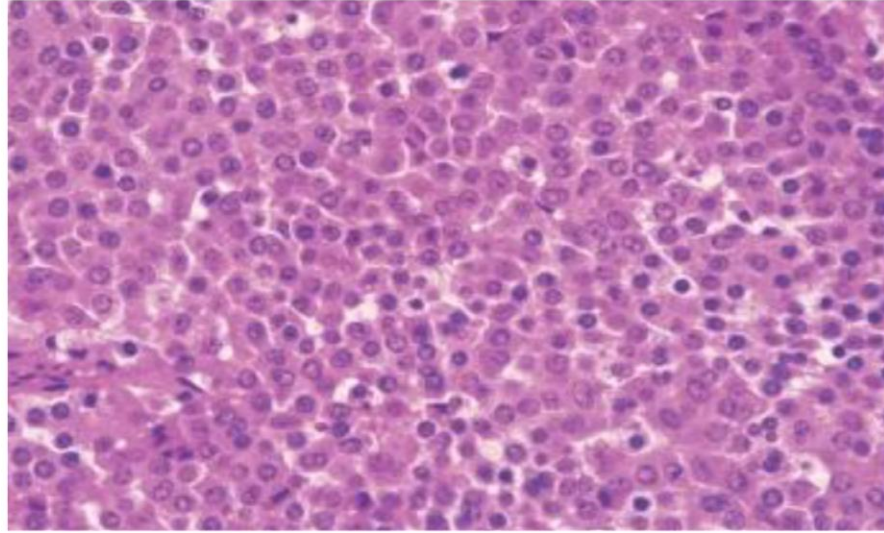
**ثانياً :** هذا الملخص جهد بشري , قابل للخطأ و الصواب , فإن أصبنا فمن الله و إن أخطأنا فمن أنفسنا و الشيطان

**ثالثاً :** مراجع كل صورة تختلف من طالب لآخر , حاولنا بقدر الإمكان أن نضع اسم كل مرجع لكل معلومة قمنا بكتابتها ..

أخيراً نتقدم بالشكر الجزيل لكل من ساهم في إنجاز هذا العمل سائلين المولى لهم التوفيق في الدنيا والآخرة ..

**لكل من قرأ أو استفاد .. لا تنسانا  
من دعواتك ..**

# Pituitary Adenoma



## Description :

### Microscopic :

- 1-Uniform polygonal cells arranged in sheets and cords.
- 2-Supporting connective tissue (reticulin is sparse).

**nuclei:** uniform Mitotic activity is scanty. **Cytoplasm:** acidophilic.

Cellular monomorphism and the absence of a significant reticulin distinguish pituitary adenomas from non-neoplastic anterior pituitary parenchyma.

### Macroscopic "Gross" :

**Micro adenoma(small):** well circumscribed , soft and confined within sella turcica.

**Macro adenoma(large) :** distend the sella turcica , compress the optic chiasm and erode the clinoid processes. 30% of cases are not capsulated. infiltrating the adjacent bone . foci of hemorrhage and necrosis are common in large adenoma.

## Cont' ...

### Types:

- 1-Prolactin cell (**lactotroph**) adenoma
- 2-Growth hormone cell (**somatotroph**) adenoma
- 3-Thyroid-stimulating hormone cell (**thyrotroph**)adenomas
- 4-ACTH cell (**corticotroph**) adenomas
- 5-Gonadotrophcell adenomas
- 6-Mixed (**plurihormonal**) adenomas :growth hormone
- 7-prolactin mixed adenomas most common
- 8-Hormone-negative adenomas

### Resources :

Basic Pathology (**Robbins**)  
7th edition page 722-723

**Underwood** Pathology 4th  
edition page 438-439

### Complications:

**1-Non-functioning macroadenomas** may cause Hypopituitarism because they encroach and destroy the anterior pituitary parenchyma & will compress **the optic chiasm(visual field abnormalities) & will increase intra-cranial pressure.**

**2-Functioning** adenoma will cause Hyperpituitarism thus it will cause endocrinal abnormalities.

**Prolactinomas:** amenorrhea and galactorrhea.

**Growth hormone adenomas :** gigantism (children), acromegaly (adults), impaired glucose tolerance, and diabetes mellitus.

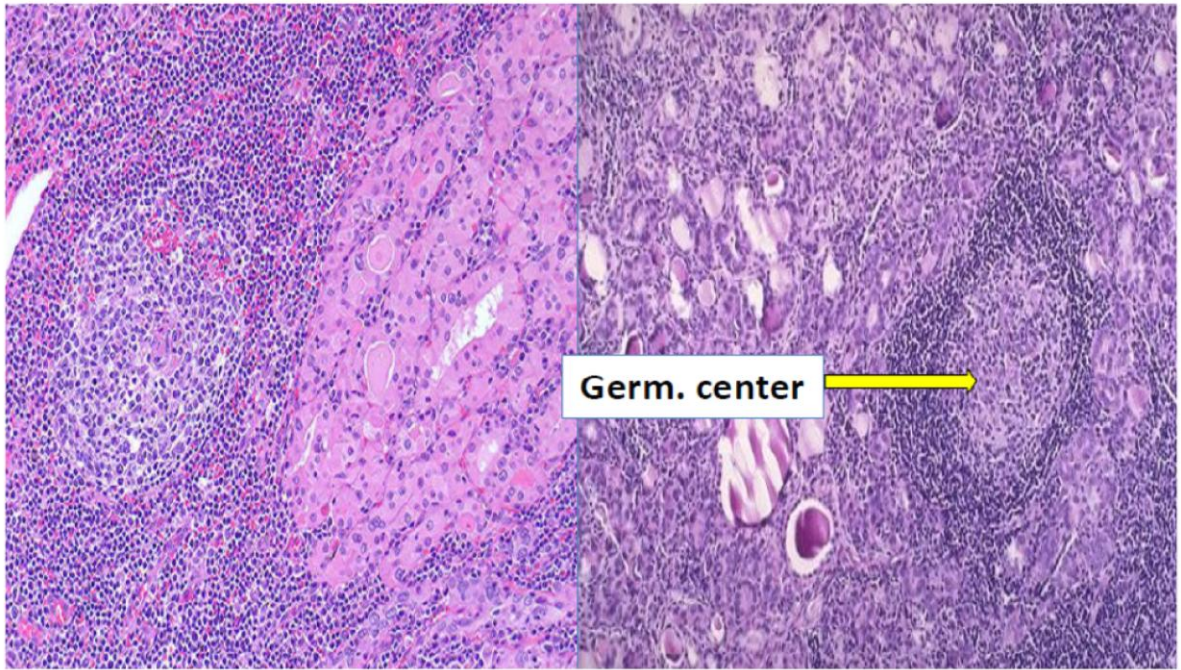
**Corticotroph cell adenomas:** Cushing syndrome.

### Risk Factors:

-family or personal history of (MEN1).



# Hashimoto Thyroiditis



## Notes

:\*Destruction of the follicles may lead to **hashitoxicosis** then **hypothyroidism**.

## Notes:

The b-cells in this lesion “within the **germinal center**” produce **anti-TSH** receptors antibodies blocking the action of TSH.

## Description:

### Microscopic :

Lymphoplasmacytic infiltrate ,Lymphoid follicles with germ centers ,Destruction of thyroid follicle , **Askanazy (Hurthle) cells** (metaplastic FC) “hurthle cells are mostly present ,but in this picture they’re not” .

### Grossly :

The gland diffuse and enlarged , the capsule is intact , the cut surface pale , gray tan , firm consistency .

### Hurthle Cells :

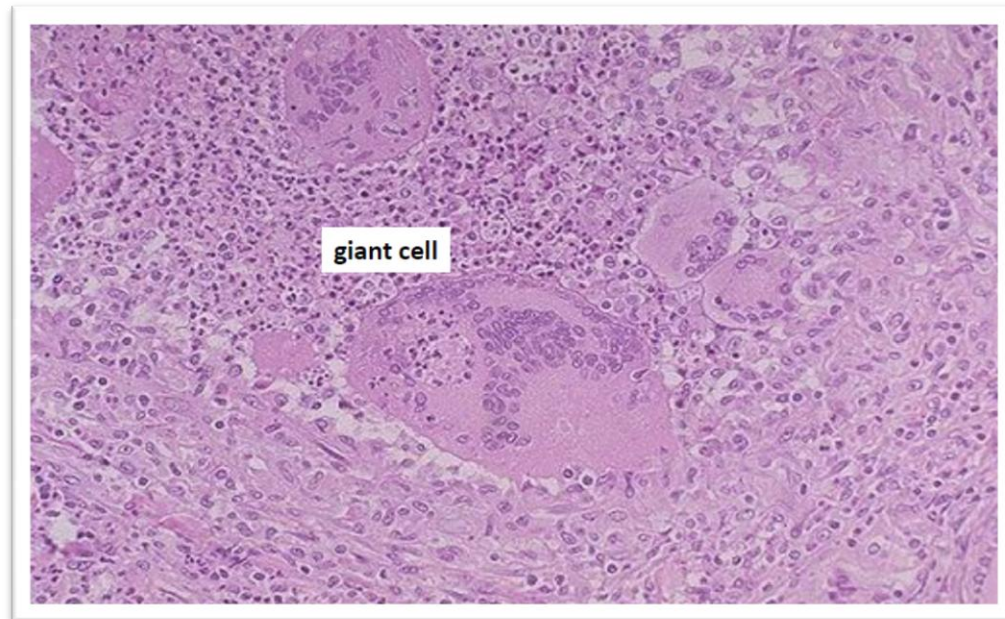
Thyroid follicles are atrophic and are lined in many areas by epithelial cells distinguished by the presence of **abundant eosinophilic, granular cytoplasm** , these cells are **termed ; Hurthle , or oxyphil , cells** . And , it is a **metaplastic response** of the normally low cuboidal follicles .

**Complication:** Goiter, Heart problems, Mental health issues, Myxedema, Birth defects , b-cells lymphoma “non-hodgkin” .

**Frequency :** more in women in women b/w 45-65 years

**Causes:** Genetic auto-immunity caused by Polymorphism to the immune regulation genes “**CTLA4**” and “**PTPN22**”

# Granulomatous Thyroiditis



**Description** : Symmetric or irregular enlargement, Aggregates of PNL, lymphocytes, plasma cells, and histiocytes , Damage of acini, Multinucleated giant cells .

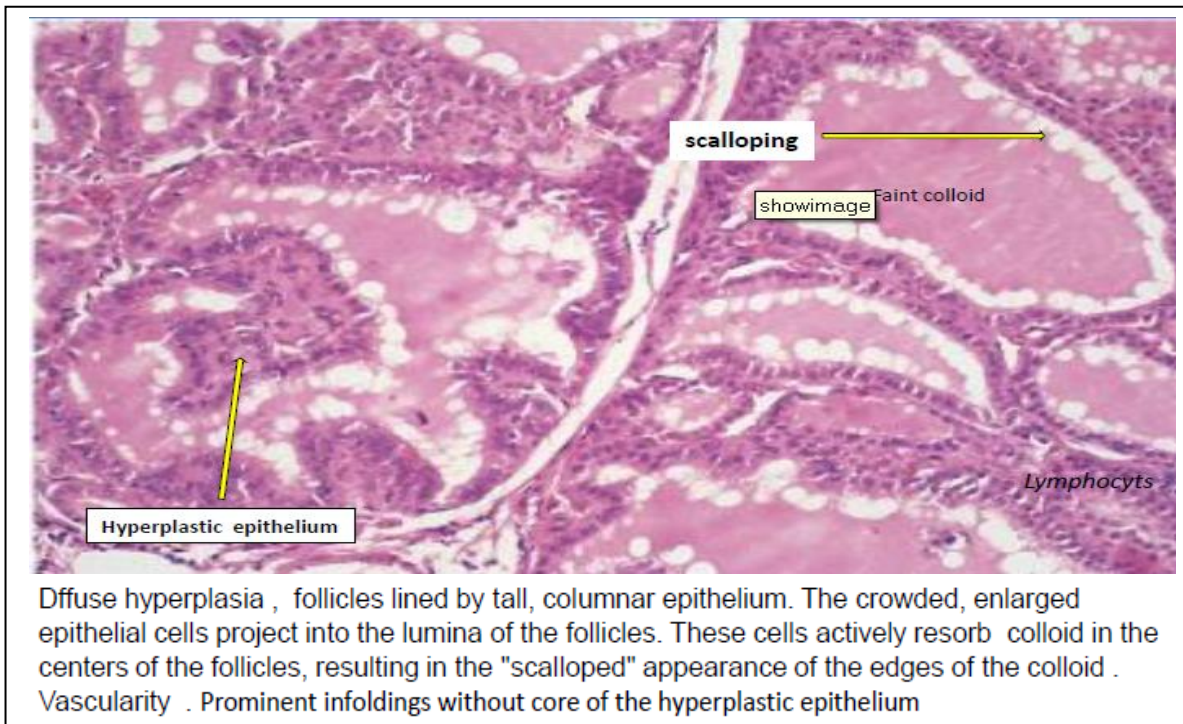
**Grossly** : firm with intact capsule , maybe unilateral or bilateral enlarged .

**Cause** : viral infection

Sources for these two pictures “*Hashimotos thyroiditis* , and *Granulomatous thyroiditis*” : Robbins as well as Dr.Ola's Lecture .



# Graves Disease “thyroid”



**Gross:** Thyroid gland itself is **diffusely enlarged** but **not nodular**.

## Microscopic:

**Hyperplastic thyroid** characterized by **many papillary infoldings** within follicles with presence of **tall columnar appearance** of the hyperplastic follicular epithelial cells. Presence of **clear vacuoles** indicate **increase processing of colloid** for increase output of hormone.

hyperthyroidism.

- **Definition:** Goiter with diffuse thyroid hyperfunction due to TSH receptor dysfunction
- **Pathogenesis:** Due to anti-TSH receptor antibody that stimulates the receptor, resulting in the production of T3& T4.
- **Epidemiology:** Occurs between 20 and 40 years of age; predominance of female to male, with a ratio of 7:1.
- **HLA associations:** *HLA-B8 and HLA-DR3*.

# Multinodular Goiter



Shown here enlarged thyroid follicles lined with inactive, flattened epithelial cells and filled with abundant stored colloid

**Gross:** Multinodular goiter are often **asymmetric**, both **lobes are enlarged**

**Microscopic:** the follicular epithelium may be **hyperplastic** in the early stages of disease or **flattened** and **cuboidal** during periods of involution. **Colloid is abundant** during the latter periods. With time, recurrent episodes of hyperplasia and involution combine to produce a **more irregular enlargement** of the thyroid, termed **Multinodular goiter**.

- **Definition:** nodular Enlargement of the thyroid gland . reflect impaired synthesis of thyroid hormone most often caused by dietary iodine deficiency .
- **Pathogenesis:** Impairment of thyroid hormone synthesis leads to a compensatory rise in the serum TSH, which, in turn, causes hypertrophy and hyperplasia of thyroid follicular cells enlargement of the thyroid gland .
- **Epidemiology**
  - 1-Endemic goiter"more common": occurs in areas where food supply contain little iodine .  
**Iodine deficiency → decreased T3&T4 → increased TSH → hyperplasia of acini (parenchymatous) → accumulation of colloid**
  - 2- Sporadic: occurs less commonly than endemic goiter **Ingestion of goiterogenous or enzyme deficiency** that interfere with thyroid hormone synthesis

- **Complications :**

1. **Because the mass is large will lead to :**

- a. airway obstruction
- b. dysphagia
- c. compression of large vessels in the neck

2. **A hyperfunctioning ("toxic") nodule may develop >>** hyperthyroidism ,This condition, known as Plummer syndrome
3. **Ability to mask or to mimic neoplastic diseases of the thyroid .**



# Adrenocortical Adenoma



## Description:

### Macroscopic:

**Solitary, circumscribed, lobulated, capsulated, yellow to yellow-brown mass , compressing remaining adrenal tissue** with yellow to yellow brown cut section owing to the **presence of lipid within neoplastic cells**. The functional status of an Adrenocortical adenoma cannot be predicted from its gross or microscopic appearance. weight: less than 30g.

### Microscopic:

They are usually composed of **lipid rich cells** similar to those encountered in the normal zona fasciculata. the neoplastic cells are vacuolated because of presence of intracytoplasmic lipid . There is **mild nuclear pleomorphism**.

*Cont...*

**Causes “Etiology”:**

1-P53 & P57 mutations.

2- The multiple endocrine neoplasia (MEN1) gene.

**Clinical Features :**

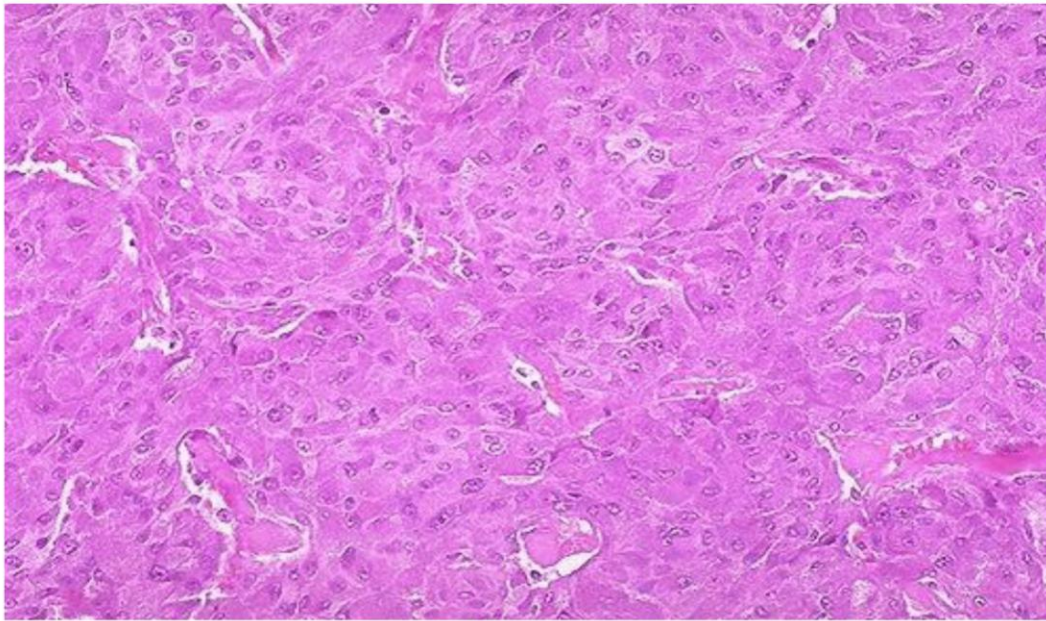
1. Weight gain
2. moon face
3. buffalo hump
4. (striae) on the skin (abdomen, thighs, breasts , arms )
5. Thinning, fragile skin that bruises easily

Basic Pathology (Robbins) 7th edition page 743-745

**Complication :**

1. osteoporosis
2. hypertension
3. Diabetes
4. Frequent or unusual infections
5. Loss of muscle mass

# Pheochromocytoma



## Microscopic picture

Cells show **polymorphism**.

Tumor cells: Polygonal to spindle shaped tumor cells. That are grouped with their supporting cells to form small nests called ( **Zelballen** ).

**Cytoplasm**: is granular, amphophilic with eosinophilic globules in some cases.

**Nuclei**: **bizarre** nuclei and **multinucleated**.

Invasion of gland capsule or even vessels maybe +ve.

Gross: **small circumscribed yellowish to large and hemorrhagic**. Weighting: **100gm up to 1-4kg!**

Benign tumor of chromaffin cells.

\* Cells of pheochromocytoma produce: 1) epinephrine. 2) norepinephrine. 3) peptides. 4) steroids.

\* Site of pheochromocytoma: **85% in chromaffin cells of adrenal medulla & 15% in chromaffin cells of extra-adrenal paraganglia.**

**Histogenesis**: neural crest → sympathogonia → pheochromoblast will give chromaffin cells if its in adrenal medulla  
→ pheochromocytoma, if it gives chromaffin cells outside the adrenal medulla → paraganglioma.

## Cont...

**Pathogenesis:** 90% sporadic, 10% familial ( alone or part of hereditary syndrome like **men2A** and **men2B** ). \*

Most important clinical feature is hypertension with **palpation**, **tachycardia**, **sweating** and **apprehensive feeling**.

This is benign, if it was malignant, it will metastases to :

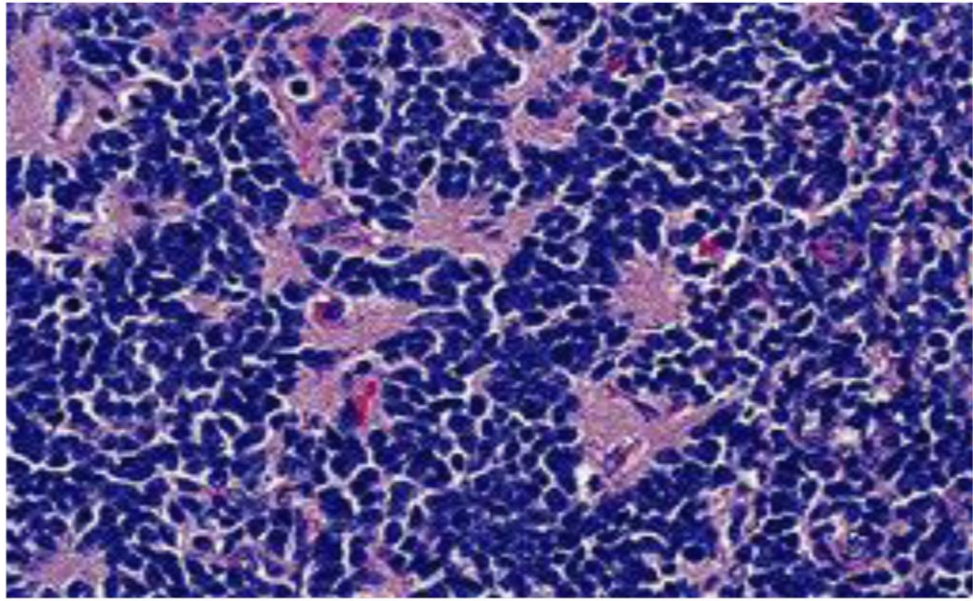
1. Regional LNs
2. Bone
3. Lung
4. Liver

**How to diagnose pheochromocytoma histologically?**

Oxidation and polymerization ( chromaffin reaction to confirm catecholamines in the granules.



# Neuroblastoma



Malignant tumor of neuroblast cell of neural crest.

Most common tumor of childhood when CNS tumors are excluded.

Appear in first year of life and can **regress spontaneously** or change into **benign ganglioneuroma**.

Site: **adrenal medulla or anywhere in the sympathetic nervous system.**

Histogenesis: **neural crest → sympathogonia → neuroblast → neuroblastoma**

Pathogenesis: **sporadic mainly, rarely familial.**

## **Gross:**

small nodules to HUGE masses that fill the abdomen of the infant.

## **Microscopic picture:**

rosettes, formed by small, regular, dark tumor cells arranged around a central, pale fibrillar core

**( Homer – wright rosettes ).**

Round to oval tumor cells

**Nuclei:** large, hyperchromatic, mitotic and surrounded by scanty (little) cytoplasm.

**Neuroblastoma** likes to invade the **renal vein to extend into IVC.**

# Ovarian Serous Tumors

## Serous tumors :

- It's a surface epithelial tumor "**originated from the coelomic mesothelium that covers the surface of the ovary**"
- **Frequency : 65-70%**
- **Age affected : 20+ years**
- **They're of three types :**
  1. **Benign "60%" = [ bilateral in 10% of cases ]**
  2. **Borderline "25%" = [bilateral in 30% of cases]**
  3. **Malignant "15%"**

## Benign are usually :

- **Cystic "Cystadenoma"**
- **Or , can have accompanying stromal component "cystadenofibroma"**

## Malignant are :

- **Cystic "cystadenocarcinoma"**
- **Or , Solid "Carcinoma"**

There's also an intermediated form "**borderline or currently referred to as *tumor of low malignant potential***". They're low grade cancers with limited invasive potential , thus they have a better prognosis than the fully malignant ovarian carcinoma .

## Risk Factors :

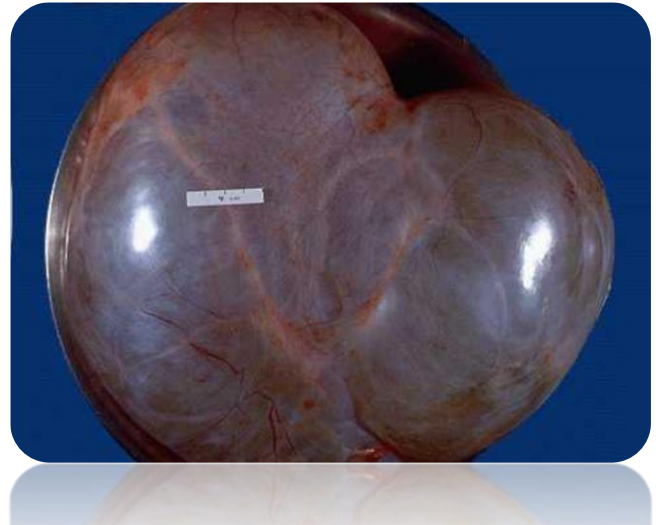
- Null parity
- Family History
- Gonadal Dygenesis

## Causes :

- In most of familial cases , **Mutations in BRCA1 "30% average lifetime" , and BRCA2 .**
- Expression of the protein "**HER2/NEU**"
- **Mutation in p53 .**

Cont' ...

## *Simple Cystadenoma “serous tumor”*



### **Identification :**

#### *Simple Serous cystadenoma*

### **Description (Macroscopic) :**

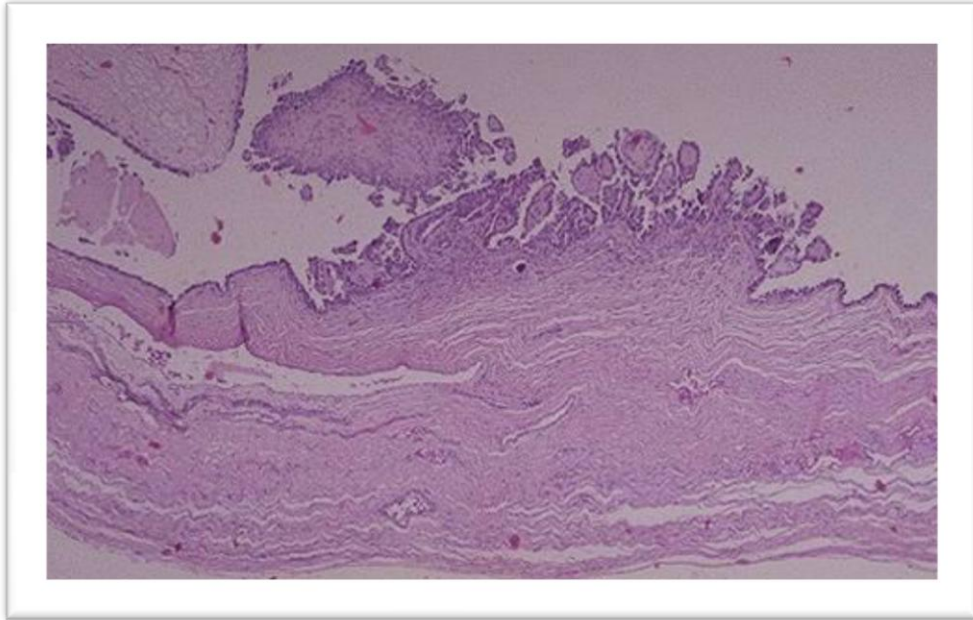
*Thin walled, multilocular cyst (may also be unilocular) usually contains watery fluid with smooth glistening outer surface.*

### **Description (Microscopic) :**

*Single layer of tall columnar epithelium lining the cyst “cysts”, the cells are partially ciliated and partially dome-shaped secretory cells .*

## *Papillary serous cystadenoma (border line)*

### *Serous Tumors*



#### **Identification :**

*Papillary serous cystadenoma (border line)*

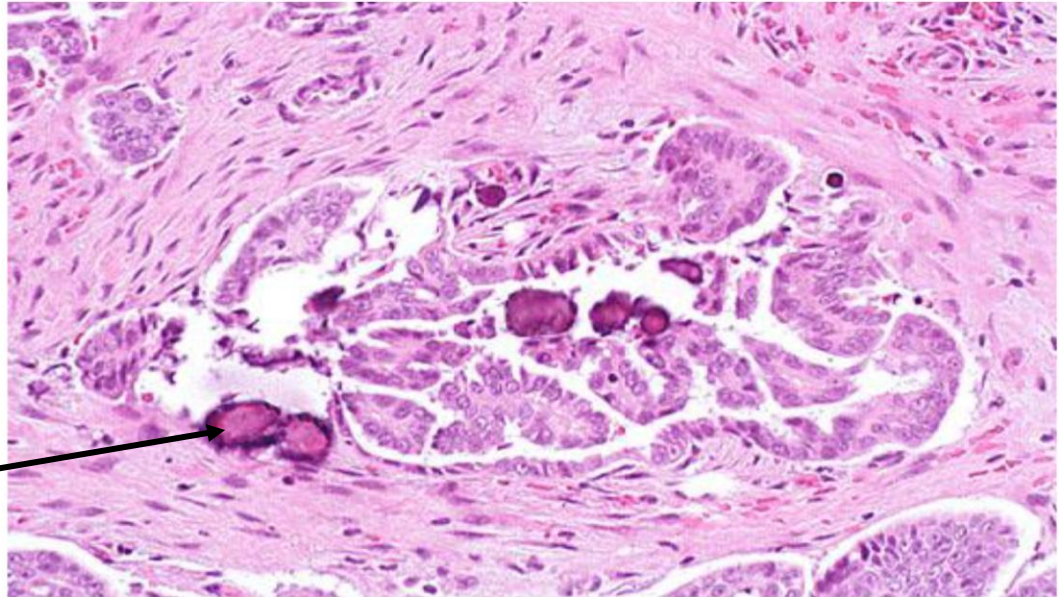
#### **Description :**

Cystic cavities lined by **columnar and cuboidal cells** with **papillary projections of epithelium extending into the lumen of the tumor** , these papillary projections are **complex and multilayered** with **nuclear atypia and mitotic activity** . There is no invasion of the stroma or capsule.



## Ovarian Serous Tumors

### Papillary Serous



#### Identification :

*Papillary Serous cystadenocarcinoma*

#### Description :

**More complex growth** with **infiltration** or frank effacement **of the underlying stroma** by solid tumor , the individual tumor cells display the usual features of all **malignant neoplasia** and with **more extreme** degrees of **atypia** , the cells may become **undifferentiated** , **psammoma bodies** are found “ characterize serous tumors , although they’re not specific for neoplasia when they’re found” .

**Psammoma Bodies : Laminated Calcifications .**

#### Resources :

- *Robbins Basis of Pathology “7<sup>th</sup> edition”*
- *Robbins Basics of Pathology “8<sup>th</sup> edition”*
- *Essentials of Rubin’s Pathology “5<sup>th</sup> edition”*
- *Core Pathology “ 3<sup>rd</sup> edition”*
- *Weater’s Histopathology “5<sup>th</sup> edition”*

## *Benign cystic Teratoma (Dermoid cyst) Ovary*



### **Description:**

- Unilocular cysts containing hair and cheesy sebaceous material
- Thin wall lined by an opaque, gray- white, wrinkled, apparent epidermis
- Hair shafts frequently protrude
- Tooth structures and areas of calcification

### **FACTS:**

- ✓ Germ cell origin.
- ✓ Bilateral 10-15%
- ✓ 1% turn malignant esp squamous cc.
- ✓ 10-15% undergo torsion producing acute surgical emergency .
- ✓ Mature &cystic =benign
- ✓ Immature tissue &solid =malignant

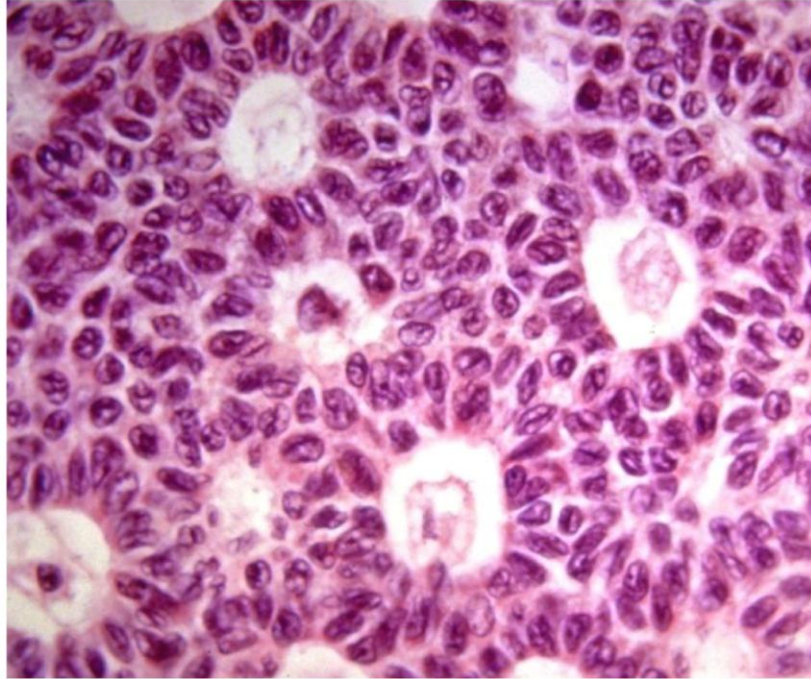
**Histogenesis:** The tumors are derived from unfertilized germ cells.

### **complication :**

1. it will cause mass effect to the surrounding tissue
2. infertility
3. it may undergo malignant transformation

• source: ROBBINS

## Granulosa Cell Tumor ( Ovary )



### Morphologic features:

#### Grossly:

May be tiny or large , gray to yellow with cystic spaces .

#### Microscopic:

- Composed of mixture of cuboidal granulosa cells in cords , sheet , and strand and spindled or plump lipid-laden thecal-cells.

-Granulosa cella may recapitulate ovarian follicle as " **Cell-Exner bodies**"

- **Sex cord** origin tumor of the ovary
- Affect **any age** ,but mostly **menopausal**
- **Unilateral** and **5-25%** can be malignant .
- May secrete **large amounts** of estrogen → **breast cancer** , or **endometrial cancer**

Robbins ;  
729 , 732

## Leiomyoma



### Grossly :

- well circumscribed , firm , gray-white masses with whorled cut surface.
- It could be intramural (within the *myometrium*) or submucosal (lie directly under the *endometrium* ) or subserosal (lie directly under *serosa* ).

### Microscopic :

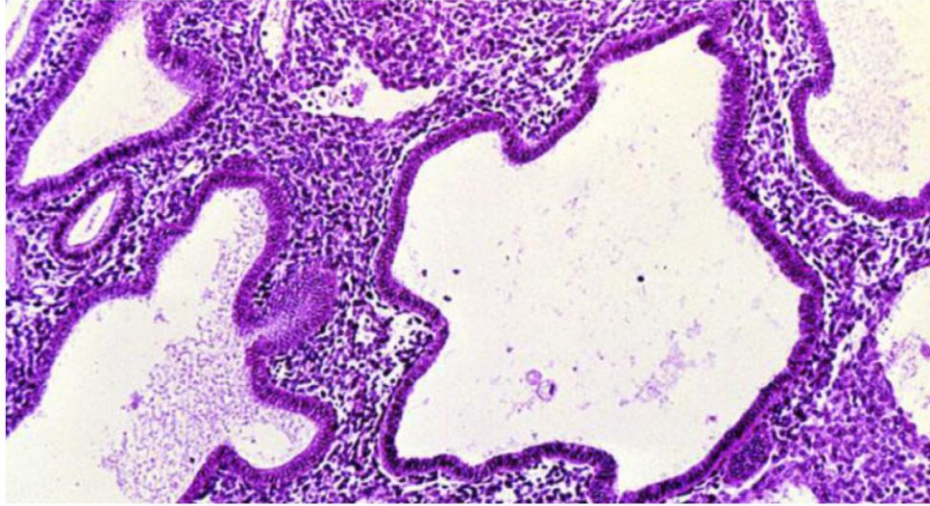
- Whorling bundles of smooth muscle cell.
- Foci of fibrosis , ischemic necrosis , calcification , cystic degeneration , hemorrhage may be present .

### Leiomyoma :

- **Benign** tumor in uterus arising from the smooth muscle cells in the myometrium, it also called "*fibroid*" .
- **Most common benign** tumors in **female**.
- Estrogen and oral contraceptive stimulate its **growth** "**Hormone- dependent**"



## Endometrial Hyperplasia



### Identification:

**Simple cystic endometrial hyperplasia**

### Morphology:

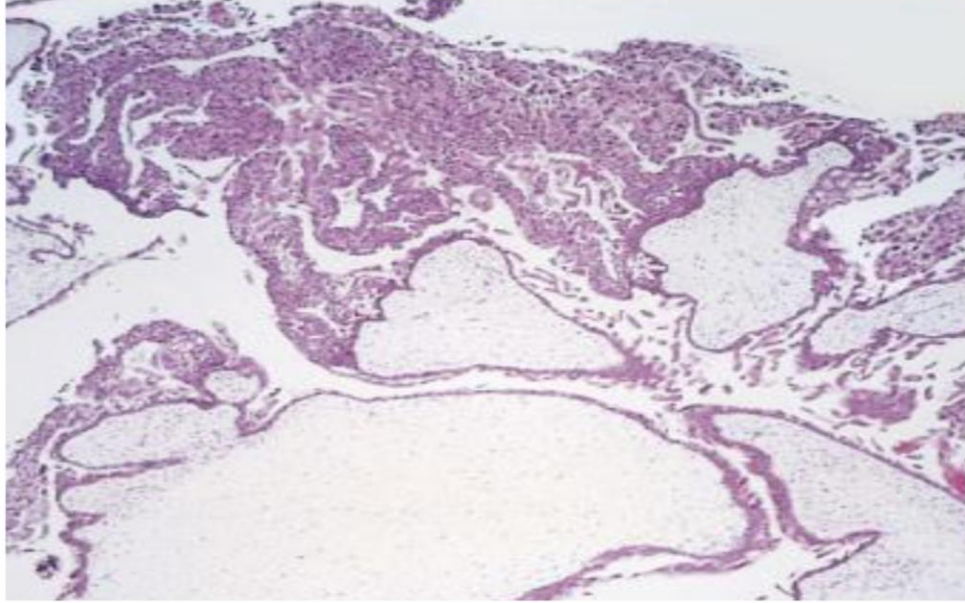
**Hyperplastic** cystically **dilated** endometrial glands lined **by thick layer of hyperplastic** epithelium showing **no evidence of secretory activity** surrounded by **dense cellular hyperplastic stroma** .

**Causes** : Excessive Estrogen.

**Complications** : Endometrial carcinoma

**Grading** : 1-Simple Hyperplasia without atypia **"this picture"** 2- Complex hyperplasia 3- Complex hyperplasia with atypia.

## Hydatidiform mole



There are two types of hydatidiform mole in placenta:

1. **complete** (Does NOT contain fetal parts)
2. **Partial** (contain fetal parts)

**Microscopic appearance** . COMPLETE mole shows>>>

1-Hydropic swelling of villi. "**below**" 2- Virtual absence of vascularization of villi. 3- proliferation of the chorionic epithelium "**Above**"

•In central microvilli there's : 1) Loose 2)Myxomatous 3)Edematous stroma .

•Chorionic epithelium shows some degree of proliferation of both *CYTOTROPHBLAST* & *SYNCYTIOTROPHBLAST* .

**Macro**. In early development of moles uterus will be in normal size ,but in fully developed moles , uterine cavity is (1) filled with a Delicate, Friable mass of thin-walled, (2)Translucent cystic structures, (3)Grapelike structure.

**Causes**: abnormal fertilization

**Complications**: In 2 to 3% of cases, hydatidiform moles may develop into choriocarcinoma

**Resource** : **Robbins** page 736 .

## *Fibrocystic Change of the Breast*



### Description:

#### Grossly :

Irregular palpable lumps, with multiple cysts in cut section.

Some of the cysts has bluish cavity “blue-dome-cyst”.

The unopened cysts contain turbid semi-translucent yellowish fluid.

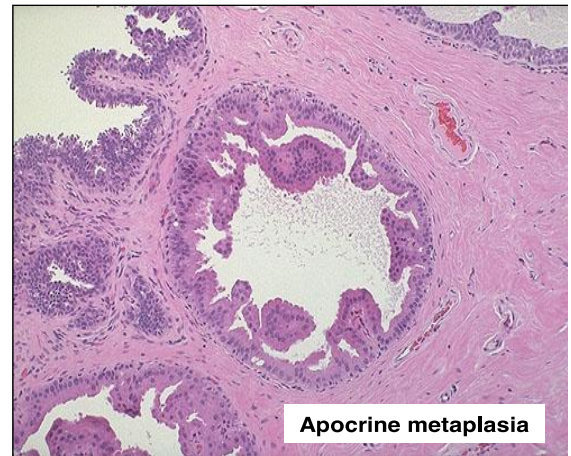
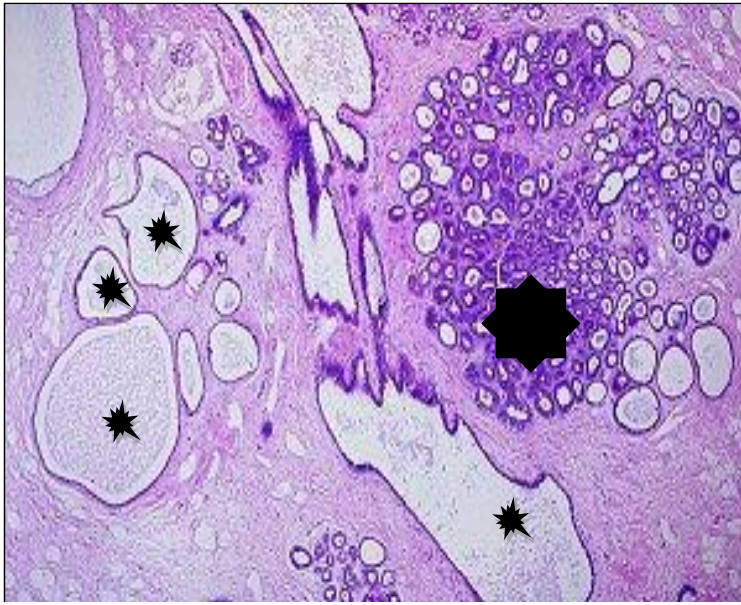
There are fibrosis “whitish areas” .

### Causes:

Unkown cause, but the female hormones monthly variations plays a partial role  
In this changes.



## Fibrocystic Change of the Breast



### Description :



- Benign proliferative condition
- Commonest lump, 10-50% women
- Often multifocal and bilateral
- It happens when normal hormonal response of breasts is disrupted or partially altered

### Types:

#### a- NON- PROLIFERATIVE

cysts, fibrosis, adenosis, apocrine metaplasia

### Mic. Picture : (left & right pic)

- A. Fibrosis
- B. adenosis 
- C. Cystic change 
- D. Apocrine metaplasia
- E. Minimal epithelial hyperplasia (2-4 cells)

#### b- PROLIFERATIVE

##### a. Without Atypia

- i. epithelial hyperplasia, sclerosing adenosis, papillomatosis

##### b. ATYPICAL

- i. Atypical ductal hyperplasia (ADH)
- ii. Atypical lobular hyperplasia (ALH)

### Mic. Picture;

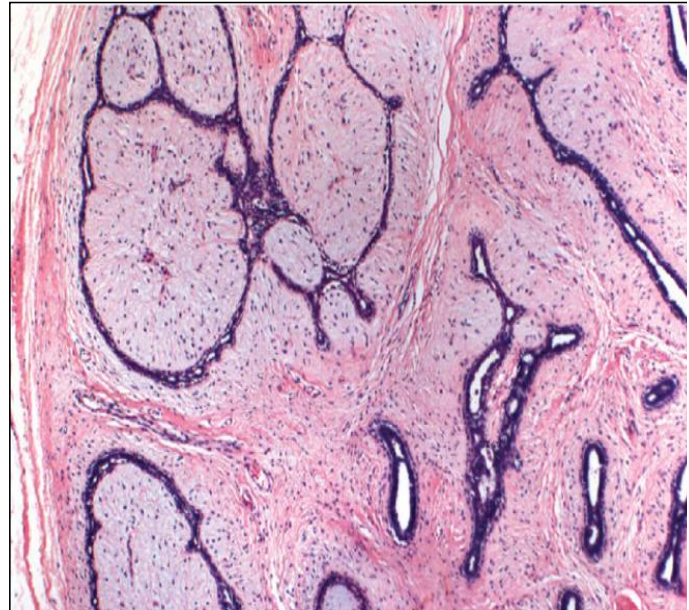
- A. This lesion is also a proliferative type of fibrocystic changes.
- B. Some areas shows epitheliosis "hyperplasia" in which the glands are packed together and has dark appearance.
- C. There are areas of fibrosis and many cystic structures.

**Causes :** Hormonal imbalance, Increased sensitivity to estrogen

**Complications :** Atypical hyperplasia associated with risk for carcinoma



## *Fibroadenoma*



- Most common benign tumor
- It's called breast mouse (easily movable)
- composed of both proliferating glandular and stromal elements
- 20s-30s
- Giant forms can occur in younger patients (15 cm)

### Description:

#### Microscopic picture

- This lesion shows well circumscribed mass with fibrous capsule.
- There is a marked proliferation of the interlobular stroma "fibrous tissue".
- The proliferative stroma has pushed the duct in which made the lumen disappear in some areas "Intracanalicular pattern"
- The boarder is sharply demarcated from the surrounding tissue.
- Around the ducts there are loose stroma "loosely cellular stroma".

#### Gross picture:

- This gross appearance shows a rubbery, white, well-circumscribed mass.
- Its clearly demarcated from the surrounding yellow adipose tissue.

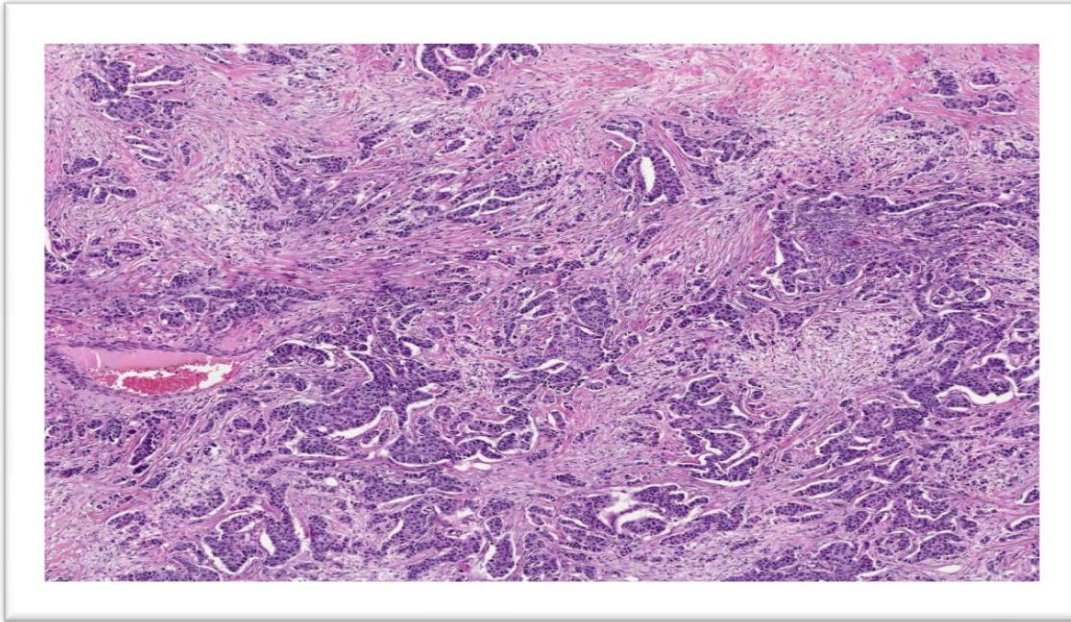
### Aetiology;

fibroadenomas seem to be influenced by estrogen, because they appear most often in premenopausal or pregnant women

### complications;

malignant transformation is rare, but it slightly increases with complex fibroadenoma

## *Invasive ductal carcinoma of no special type (NOS) (Scirrhus )*



**Microscopic appearance:** Sheets, cords and nests of malignant ductal epithelial cells infiltrating dense fibrous stroma infiltrated with lymphocytes, the malignant cells showing features of malignancy

**Gross appearance:** the lesion is retracted, infiltrating the surrounding breast substance, and would be stony hard on palpation.

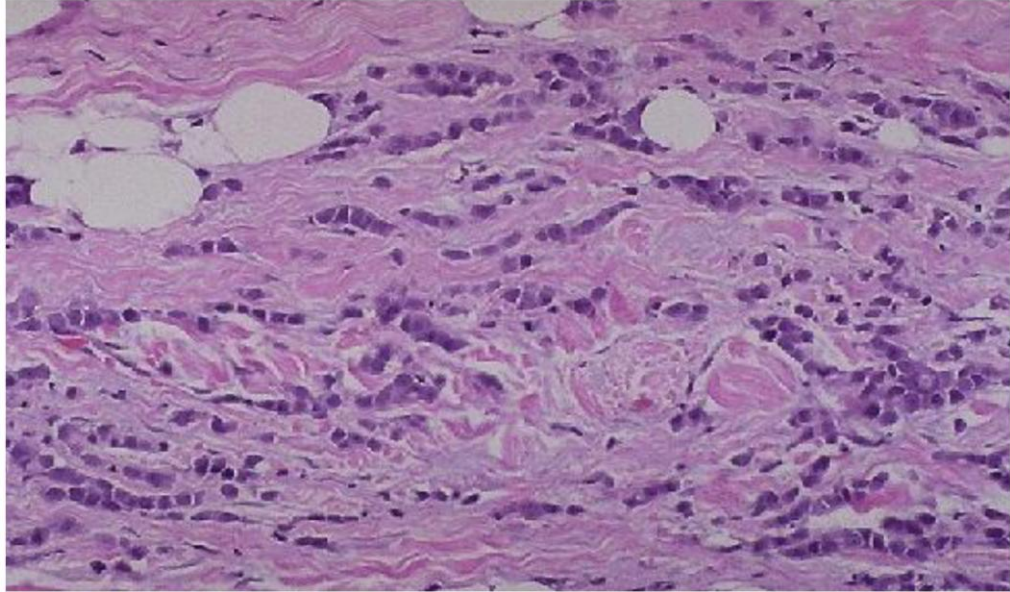
- ✓ **Peau d'orange** (lymphatic obstruction+ thickening/dimpling of the skin)
- ✓ **Paget's disease** of the nipple (ulceration/inflammation due to intraductal spread to the nipple)
- ✓ **Retraction** of the nipple

### **Risk factors:**

1. Age, Obesity, high fat diet
2. Maternal relative with breast cancer
3. Longer reproductive span
4. Nulliparity, Oral contraceptives

**Complications:** distant metastasis (lung, brain and bone)

## *Invasive Lobular Carcinoma*



### Microscopic appearance:

- Small uniform round or oval nuclei diffusely infiltrating the stroma
- Single file pattern (**Indian-file**)

### Risk factors:

1. Age, Obesity, high fat diet
2. Maternal relative with breast cancer
3. Longer reproductive span
4. Nulliparity , Oral contraceptives

**Complications:** **Metastasize** more frequently to CSF, serosal surfaces and pelvic organs.



## *Seminoma of the Testis*



- 1- Basic Robbins.
- 2- Pathology Illustrated.
- 3- Dr. Jamal lecture.

**Type:** Primitive germ cells Differentiate along gonadal line.

**Classification:** 1- Classic seminoma    2- Spermatocytic seminoma    3- Anaplastic S

### **Morphology:**

**Gross:** Well circumscribed, large, soft, gray-white, fleshy, homogeneous tumors that bulge from the cut surface of the affected testis.

**Microscopic:** in the next page.

**Cause:** remains unknown.

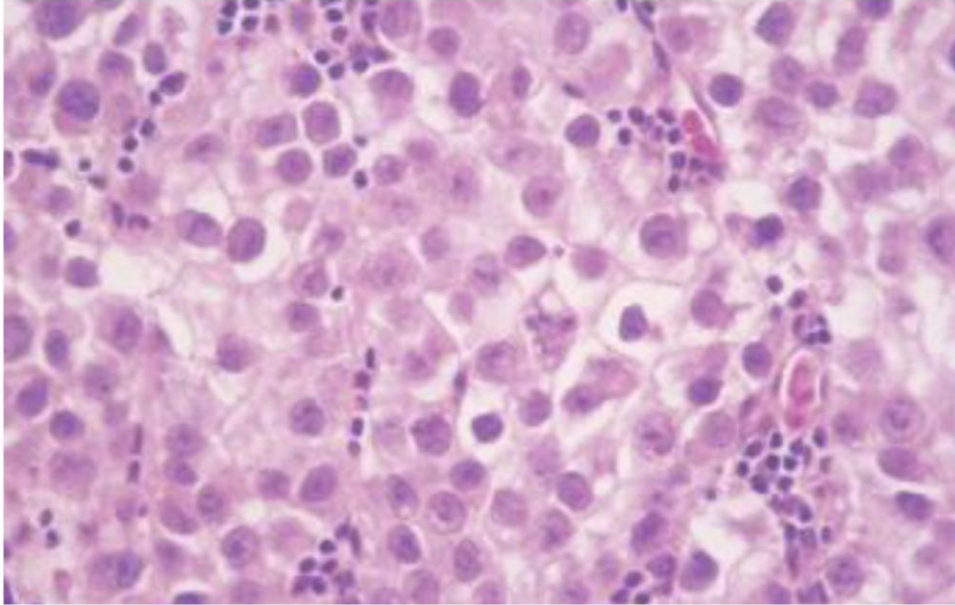
### **Predisposing factors:**

- 1- **Cryptorchidism** (4 folds increase risk of cancer).
- 2- **Abnormalities of sex chromosomes** (Klinefelter syndrome 47 xxy).
- 3- **Cytogenetic abnormalities.**
- 4- Development of cancer in one testis increase risk of neoplasia in contralateral testis.

**Spread:** via lymphatic along the spermatic cord.



## *Seminoma of the Testis*



**Type:** Primitive germ cells Differentiate along gonadal line.

### **Classification:**

1- Classic seminoma    2- Spermatocytic seminoma    3- 3- Anaplastic Seminoma.

### **Morphology:**

**Gross:** In the past page.

### **Microscopic:**

**Classic seminoma:** Large, uniform cells with distinct cell borders, clear, glycogen-rich cytoplasm, and round pale nuclei, prominent nucleoli, and separated by thin fibrous septa with a sparse lymphocytic infiltrate.

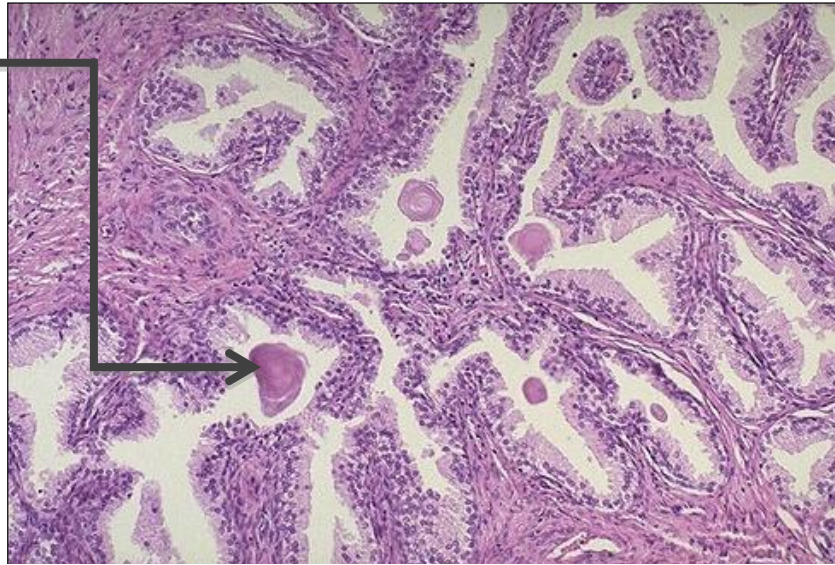
**Cause:** remains unknown.

**Predisposing factors:** As in the past page.

**Spread:** via lymphatic along the spermatic cord.

## *Nodular hyperplasia of prostate*

Corpora amylacia



- **Description:**

- A. this lesion shows glandular & stromal hyperplasia “Mixed hyperplasia”
- B. it shows a glandular enlargement with nodularity in which the glands are closely packed, and the lining epithelium is projecting into the lumen “**papillary projections**”.
- C. The surrounding fibromuscular stroma is hyperplastic.
- D. The normal corpora amylacia is seen here.

- **Causes:**

- A. Excessive testosterone.

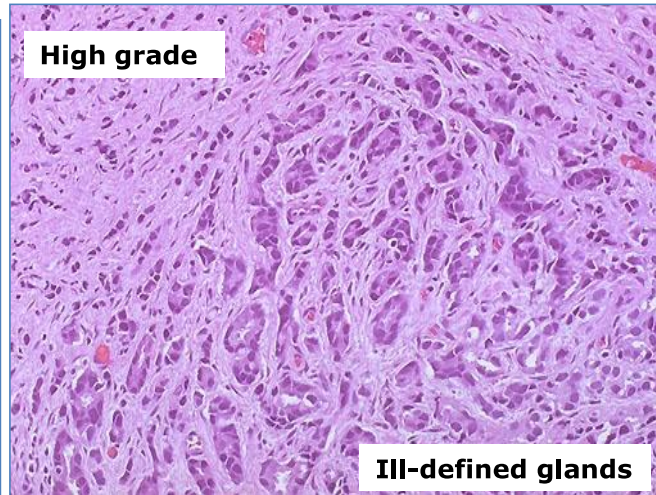
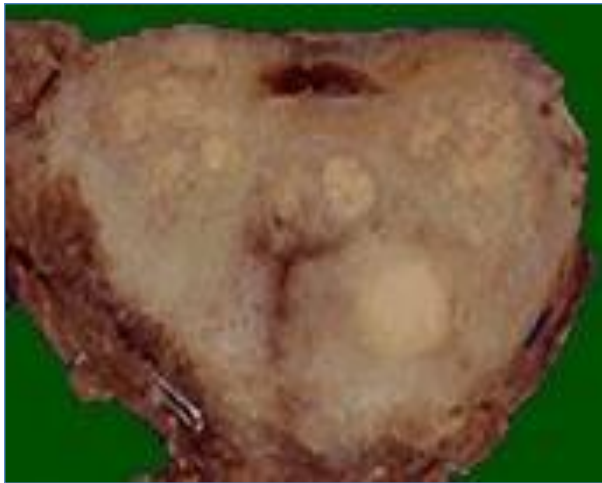
- **Complications:**

- A. Inability to urinate.
- B. Urinary tract infection.
- C. Kidney damage

Robbins & cotran atlas of pathology : page 312

Robbins & cotran basis of pathology : page 994-996

## Adenocarcinoma of the prostate



### • Description:

#### ▪ Gross:

- A. This lesion yellow irregular nodules appearing in the peripheral & peri-urithral zones.\*

\* Note: the yellow color is a hallmark of carcinoma.

#### ▪ Microscopic:

- A. This lesion shows small irregular glands, lined by cuboidal cells, also lost its myoepithelial cells.  
B. The cells here has a pleomorphic nuclei with very large nucleoli.  
C. They are infiltrating all around the stroma.  
D. The glands are crowded and lacks the brachinnng papillary infoldings.

### • Age group:

- A. 60-75 years "VERY rare in males below the age of 45"

### • Complications:

- A. Metastasis

