Male genital system pathology

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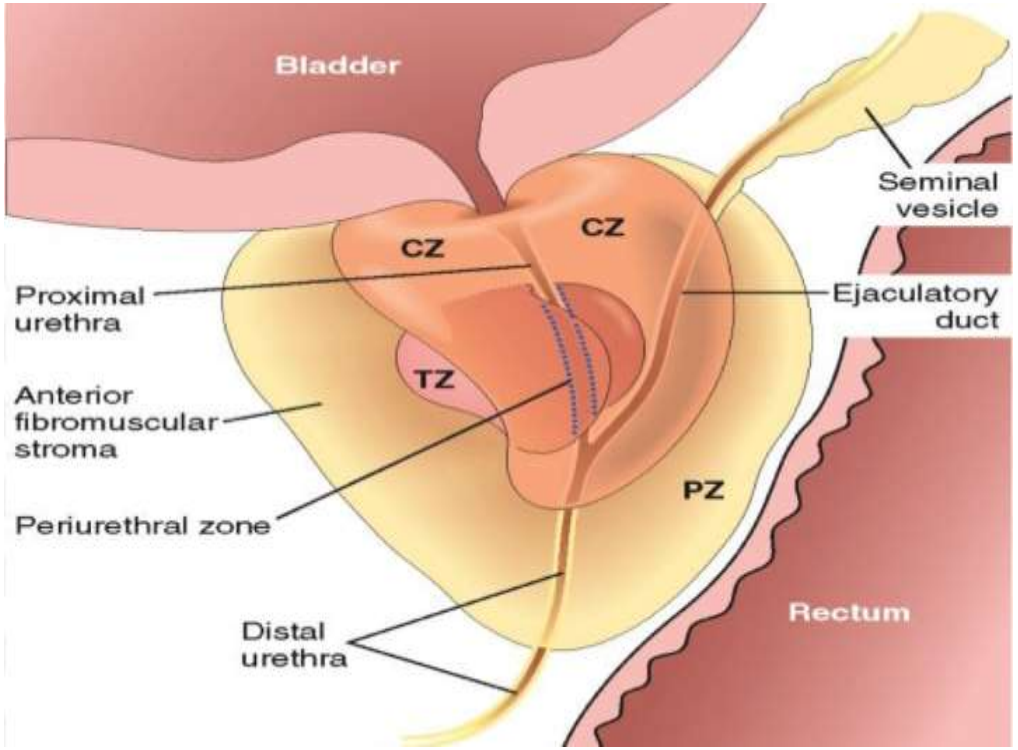
Prostate gland pathology

Anatomically:

- Composed of 5 lobes;

anterior, posterior, middle and two lateral.

- Weighs 20 grams in normal adult.



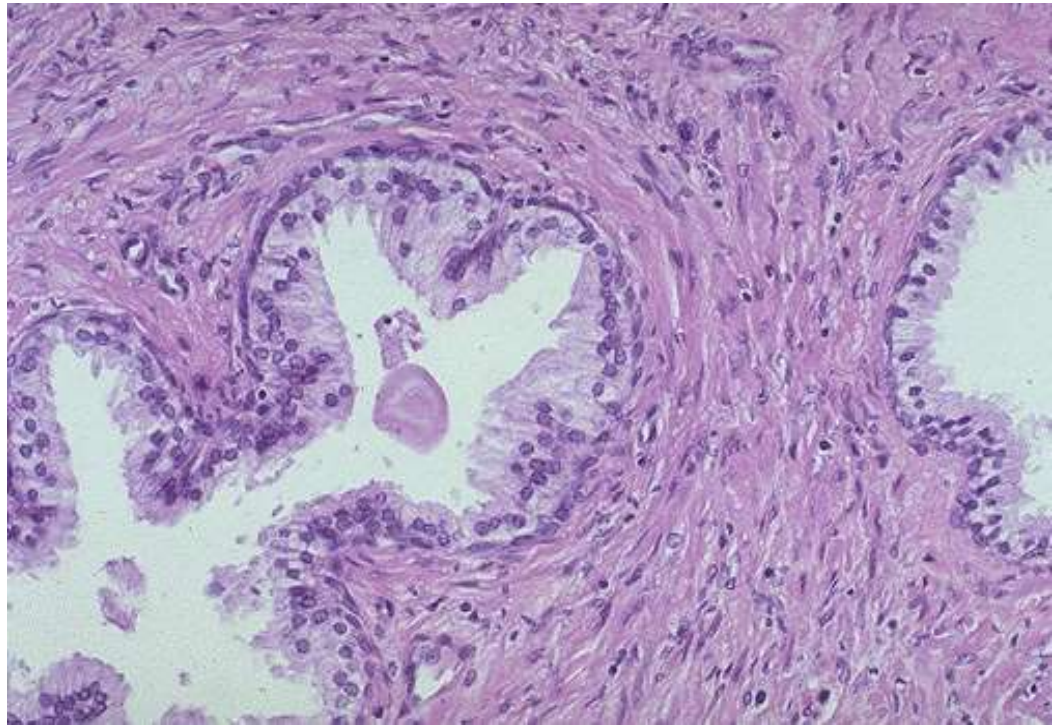
Histologically:

It composed of glands in fibromuscular stroma.

The glands composed of two layers of cells:

1.Basal: (flattened or cuboidal).

2.Apical: (tall or columnar).



Prostatitis

Acute bacterial prostatitis

- Mainly due to E. coli, staphylococci.
- **Routes of infection**: either **ascending** from urethra (commonest) or **hematogenous** route.
- **Predisposing factors:** reflux disease, following surgical manipulation of prostate (e.g., catheterization, cystoscopy or urethral dilation).
- **Clinically**: fever, chills, and dysuria.
- **On rectal examination** the prostate is tender.
- The diagnosis can be established by urine culture and clinical features.
- **Biopsy** of a gland in which acute prostatitis is suspected **is contraindicated**, as this may lead to sepsis.

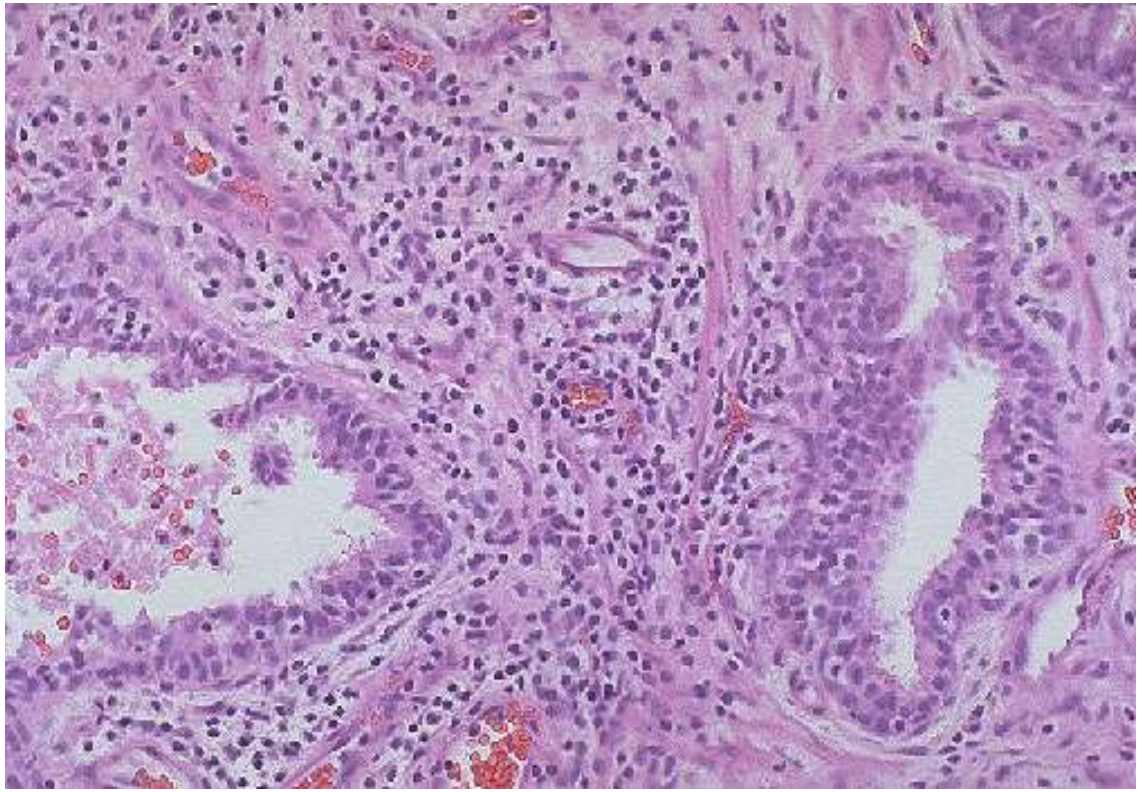
Chronic prostatitis

- **Sources:**

1. Follow episodes of acute prostatitis.

2. Develop insidiously, without previous attacks of acute prostatitis.

- **Types:**
- **Chronic bacterial prostatitis: (**positive bacterial culture**).**
- **Chronic a bacterial prostatitis:** commonest, negative bacterial culture, just increase WBC in prostate secretion.
- **Mic:** lymphoid infiltrate &glandular injury.
- A special variant of chronic prostatitis, **granulmatous prostatitis.**



Chronic prostatitis. Numerous small dark blue lymphocytes are seen in the stroma between the glands

Nodular hyperplasia of the prostate (Benign prostatic hyperplasia .BPH).

- Hyperplasia of glands and stroma results in large nodular enlargement in the peiurethral region of the prostate.
- **Age:** BPH frequency rises with age, reaching 90% by the eight decade of life.
- **Site:** Most of **BPH** lesions arise in the **transitional & central zones (periurethral)**, While most prostatic adenocarcinoma arises in the peripheral zone (mainly posterior lobe).
- **It is not a premalignant lesion.**

Pathogenesis of benign prostatic hyperplasia:

- The precise molecular etiology of this hyperplastic process is **unknown.**
- The pathogenesis is related to the action of **androgen**, so not occur in males castrated before the onset of puberty nor in men with genetic disease that block androgen.
- **Dihydrotestosterone (DHT)** derived from testosterone by the action of 5 alfa reductace stimulate glandular &stromal proliferation so 5 alfa reductace inhibitor is used for the treatment of BPH.

Clinical features:

- Features are divided into:

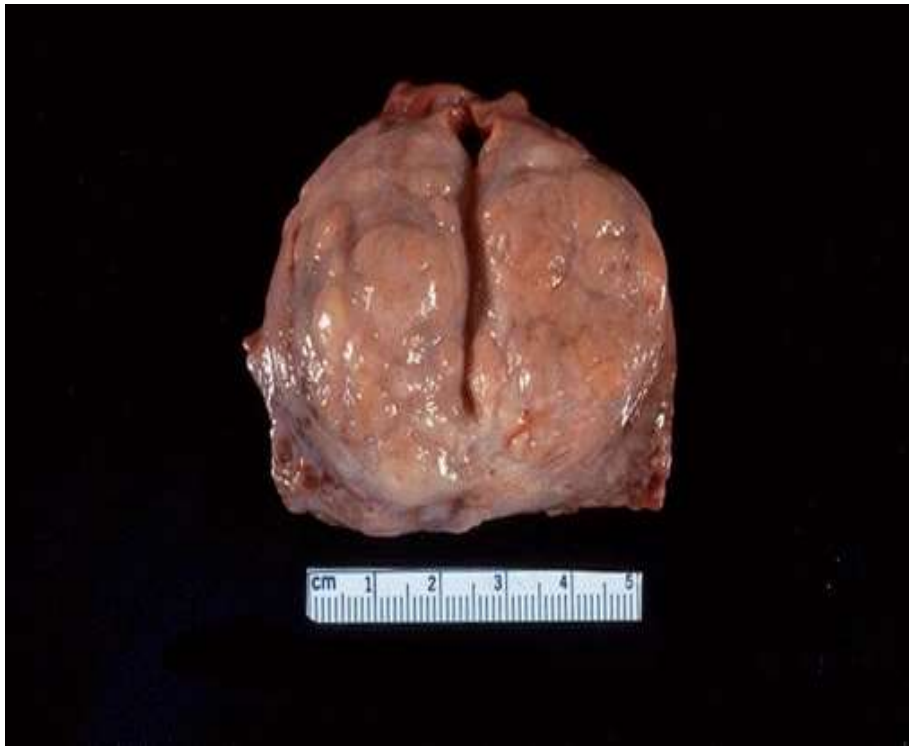
I. **Obstructive symptoms** (hesitancy or difficulty initiating the stream, straining to void, a reduced flow, an intermittent stream or a sensation of incomplete emptying).

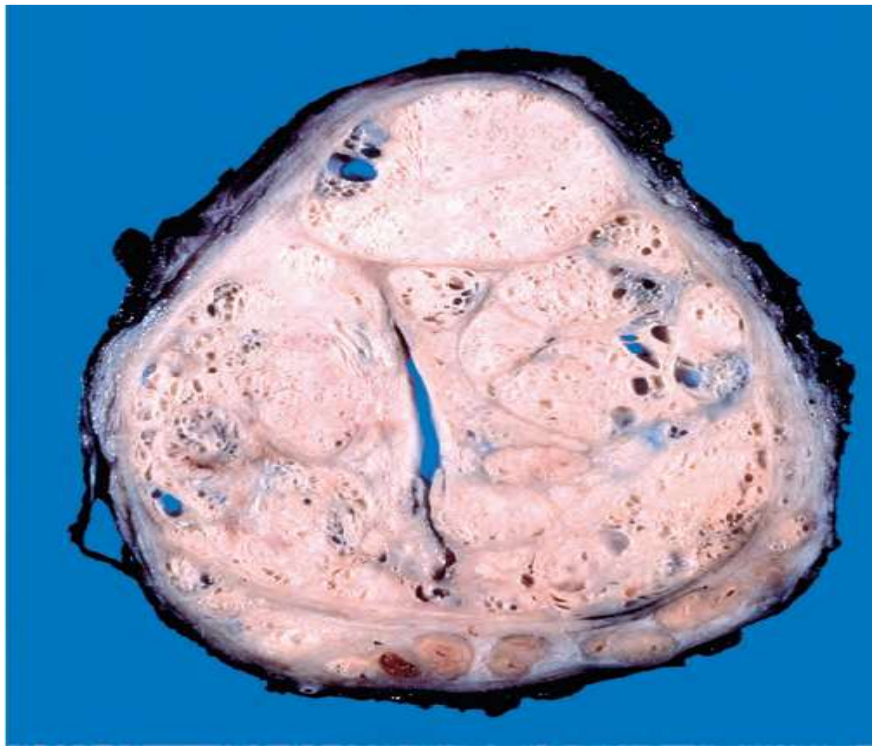
II. **Irritative symptoms** (frequency, urgency, nocturia and urge incontinence).

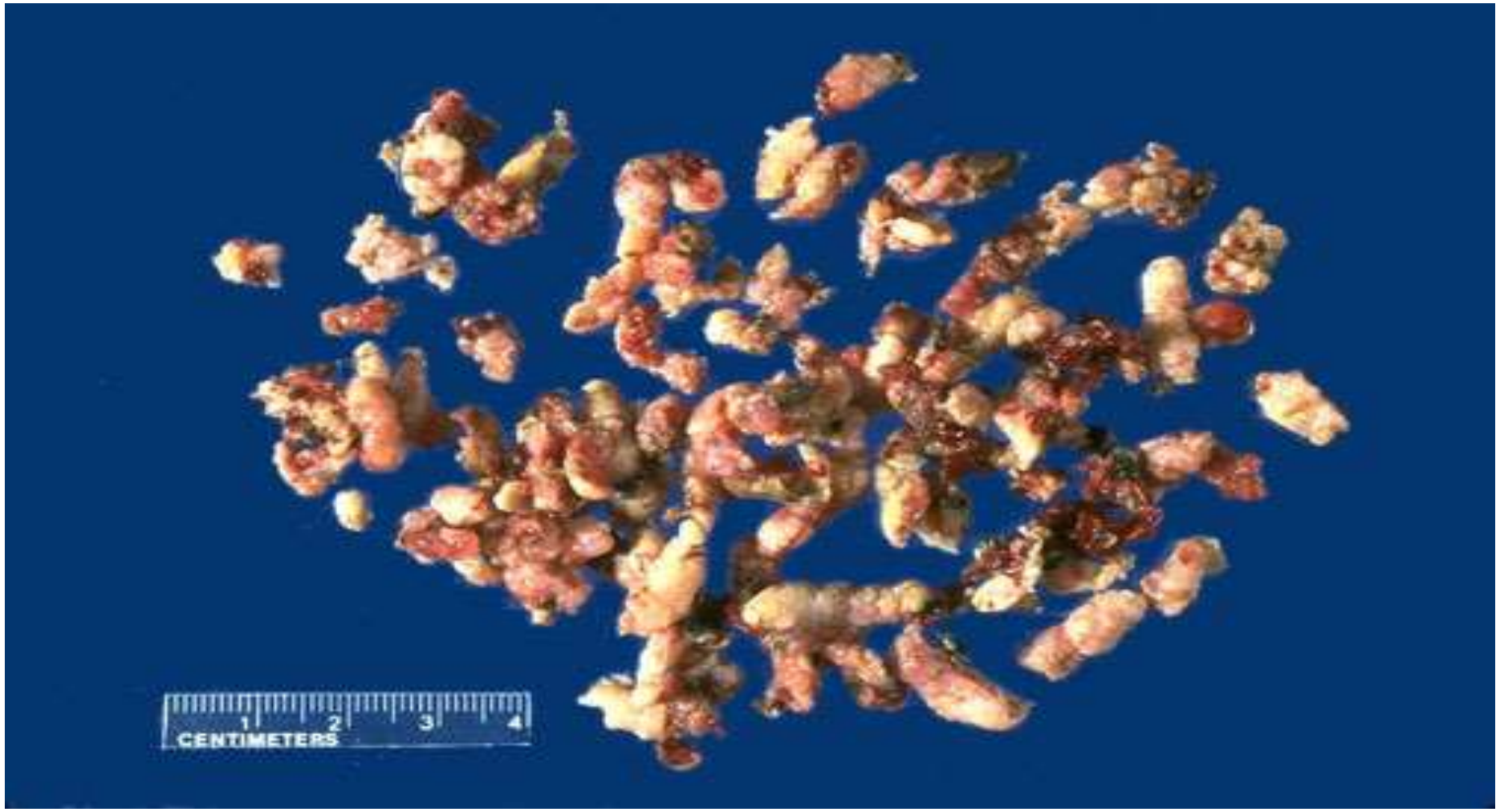
Morphology of Benign Prostatic Hyperplasia.

Gross:

- The prostate is **enlarged** (60 to 100 g) and even greater in severe cases.
- Cut section shows: **multiple well circumscribed nodules** with milky fluid secretion and cystic changes.



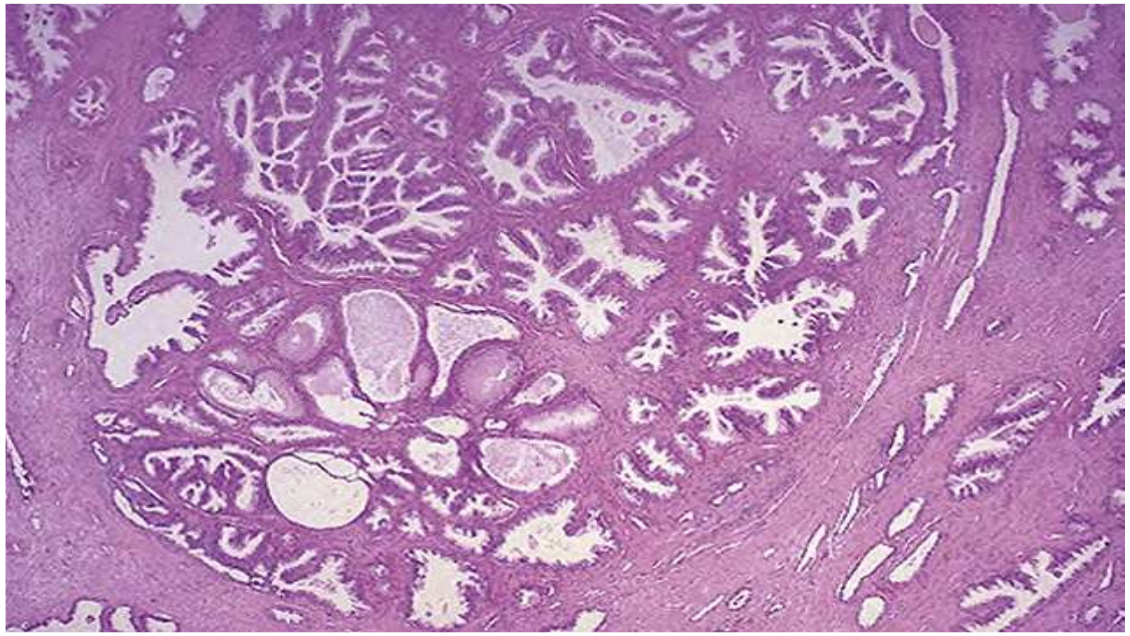


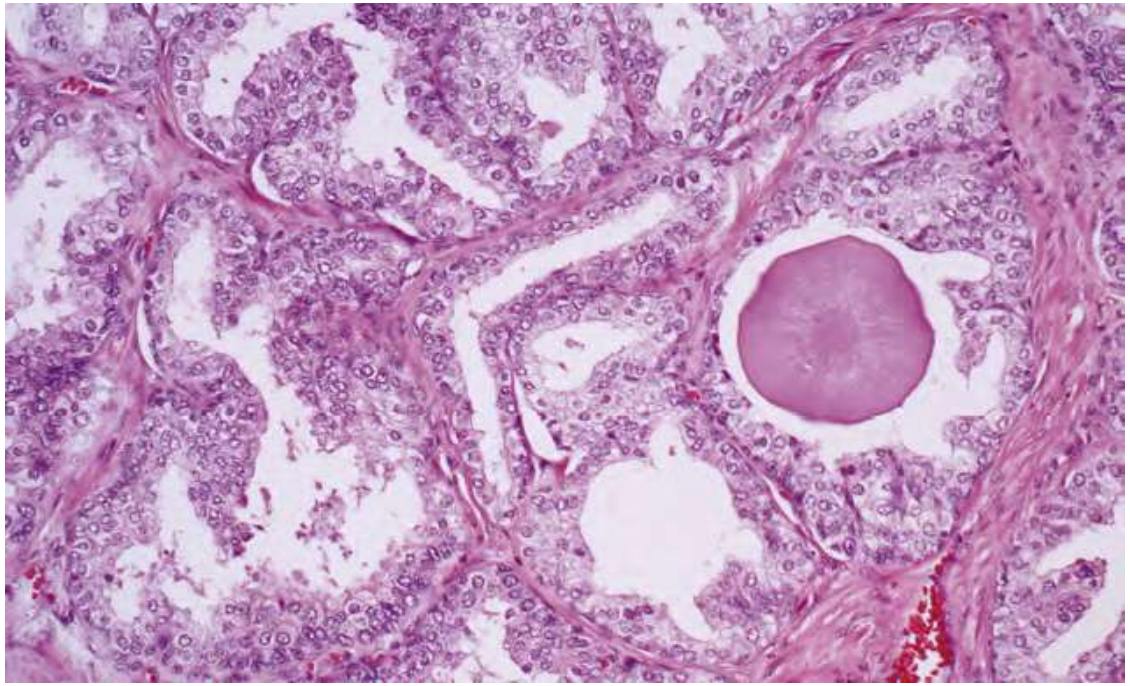


A frequently performed operation **(TURP)** for symptomatic nodular prostatic hyperplasia is a transurethral resection, which yields the small **"chips"** of rubbery prostatic tissue seen here

Mic:

- In BPH both glandular & fibromuscular stromal tissues are proliferated within hyperplastic nodules.
- Hyperplastic nodules contain small to large cystically dilated glands, lined by two layers of cells; an inner columnar and an outer cuboidal or flattened basal cells & contain inspissated, proteinecious material called Corpora amylacea in their lumina.





Carcinoma of prostate

- **Adenocarcinoma of the prostate** is the most **common** carcinoma in male & **second** most common cause of cancer related deaths in men older than 50 years of age, after carcinoma of the lung.
- **Etiology**:

Unknown, but **predisposing factors** are:

- Increasing age.
- Race (Black).
- Family history.
- Hormonal level: not develop in male castrated before puberty &the growth of carcinoma can be inhibited by orchiectomy or by the administration of estrogens.
- Environmental: increased with certain industries (Cadmium).

Clinical features:

1. **During early stages:** mainly silent, diagnosed incidentally on routine digital rectal examination, because most of cancers are aroused **at peripheral area of gland classically in a posterior lobe.**

2. **More extensive disease:** local discomfort &evidence of lower urinary tract obstruction like BPH.

3. **More aggressive carcinoma**: discovered due to metastases (bone metastases, which are commonly to axial bones.

Screening tests:

- Screening tests for diagnosis of prostatic carcinomas are:

1. Increase PSA (prostatic specific antigen) serum level (4 ng/ml is the upper limit of normal).

2. Digital rectal examination (prostatic carcinomas arise in the outer (peripheral) glands & hence may be palpable as irregular hard nodules by rectal digital examination).

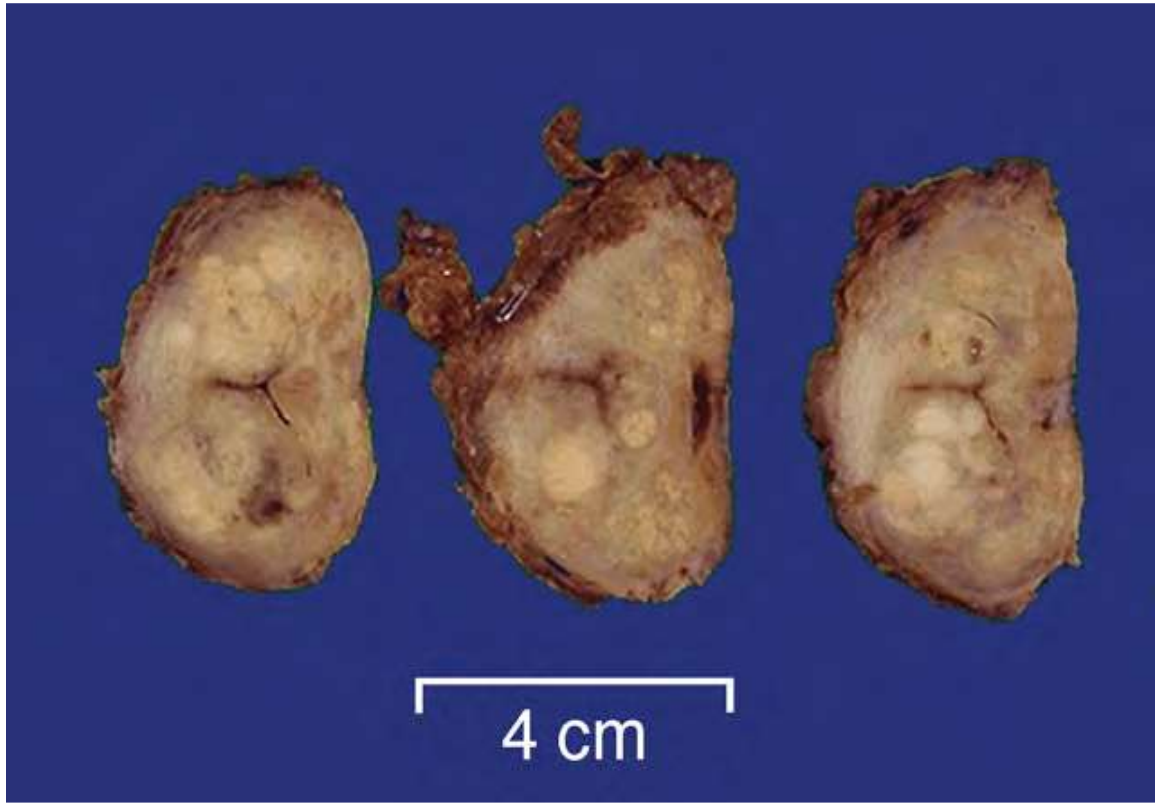
3. Transrectal sonography & needle biopsy.

- **All these tests should be used in combination in diagnosis of prostatic carcinomas.**

Morphology of prostatic carcinoma:

Gross:

- Irregular gray white – yellow hard nodules.



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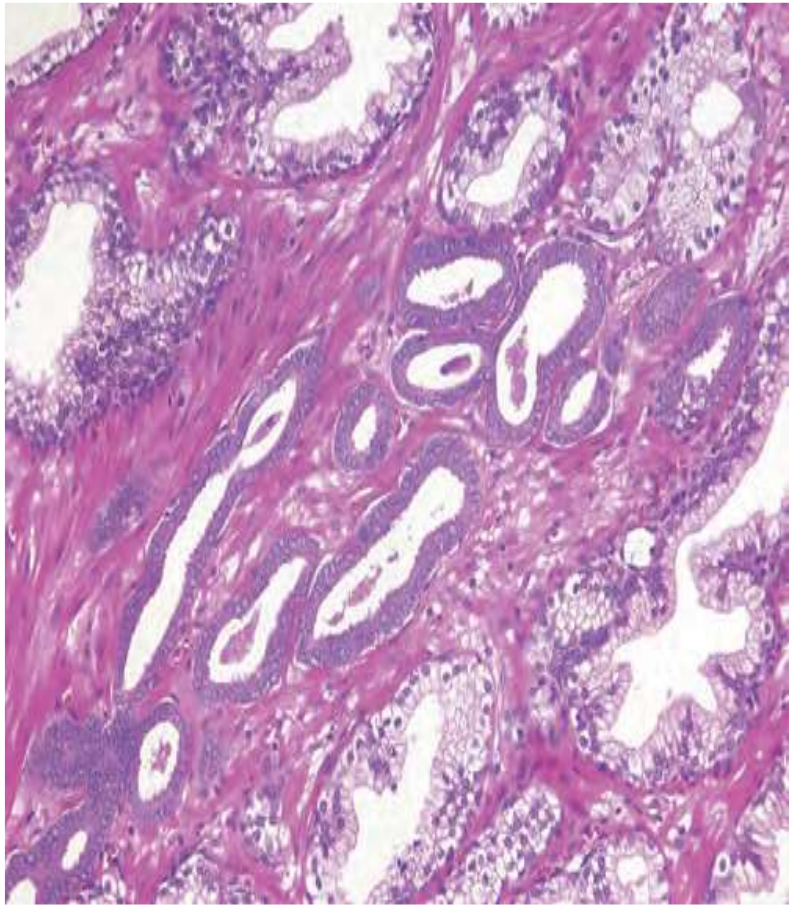
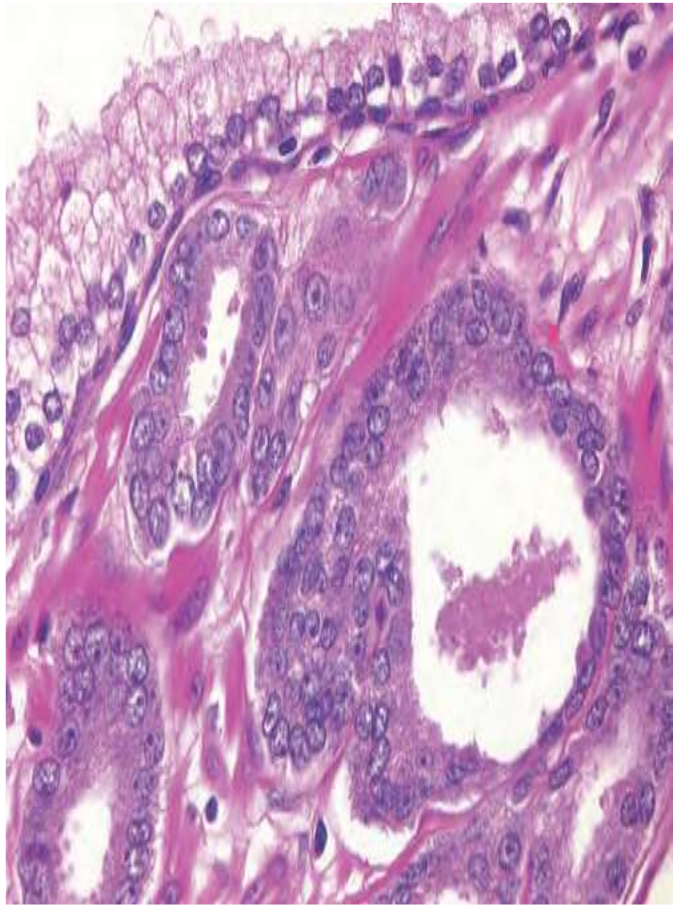
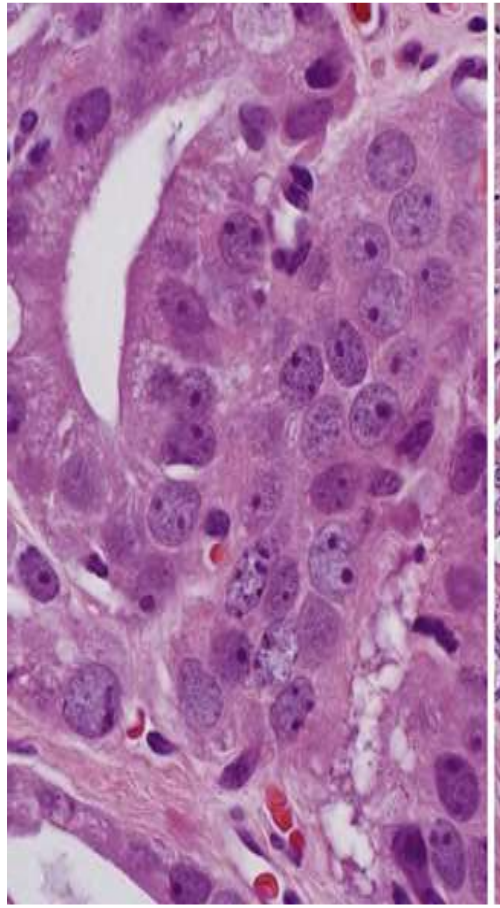


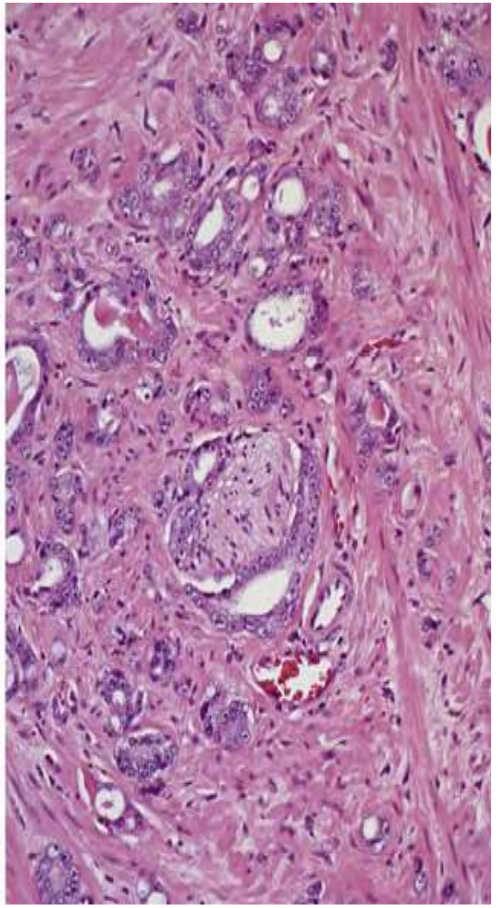
small crowded glands with little stroma



single layer of cuboidal cells absence of the basal cell layer



large nuclei and prominent nucleoli



perineural invasion

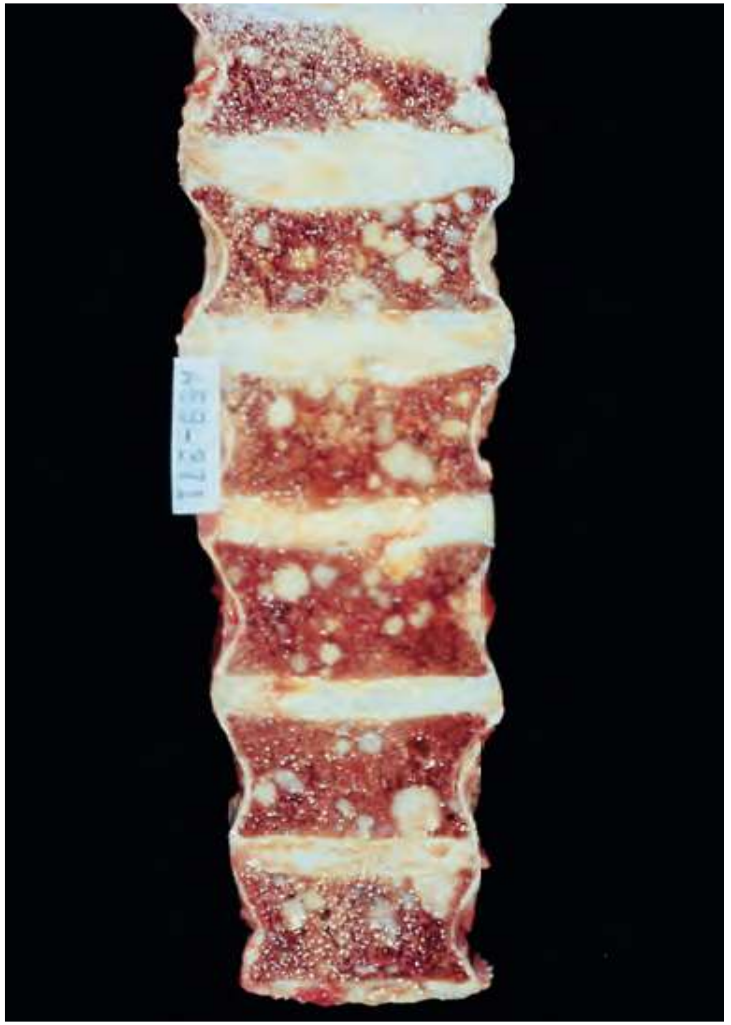
Metastasis:

1. Direct (local organs).
2. Blood stream (bone, lung/pleura, liver)

➢ Bone metastases (commonly to axial bones), which are either:

- **Osteoblastic** (bone producing) lesion, COMMONEST one & indicate advanced cancer or **osteolytic** (destructive) lesion.

3.Lymph nodes (obturator).



Grading of prostatic carcinoma:

Gleason grading system is consisting of five grades depends on glandular differentiation and growth pattern of the tumor.

- Grade I: well differentiated.
- Grade V: scattered poorly differentiated, cords and sheets.

Staging:

A: no palpable mass (incidental finding)

B: tumor confined to prostate

C: extends beyond the prostate (no metastasis)

D: metastatic disease (bone, lung, liver)

Testes pathology

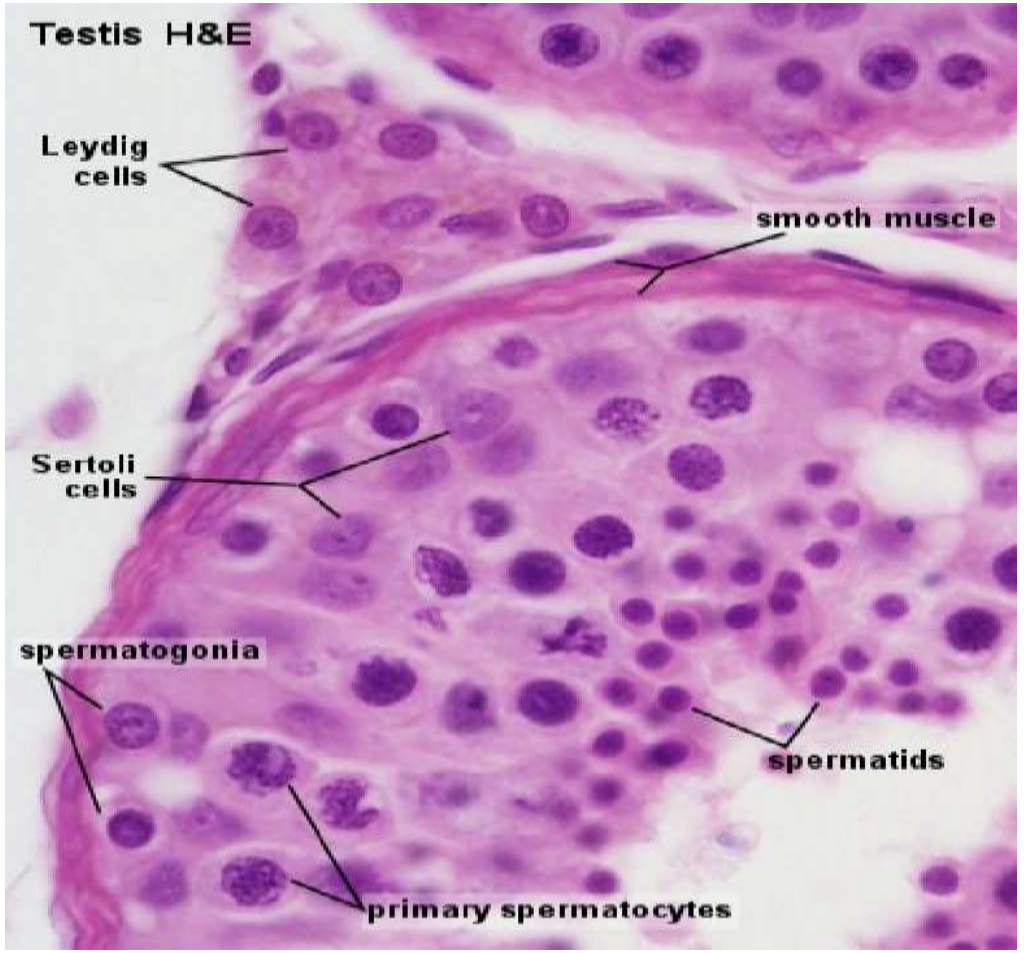
Normal testicular tissue:

➢250 lobules.

➢4 tubules in each lobule.

➢Each tubule is composed of:

- Basement membrane, sertoli cells and germ cells.
- The interstitial cells are (Leydig cells).



Congenital Anomalies
Cryptorchidism:

- **Failure of testicular descent into the scrotum.**
- It is found in approximately 1% of 1-year-old boys.
- Usually unilateral, bilateral in 25% of patients.
- The cause is unknown but may be due to **(Predisposing factors):**

1. Hormonal abnormalities. Mainly androgen abnormalities (androgen is important for descending of testes from the pelvis to scrotum).
2. Intrinsic testicular abnormalities.
3. Mechanical problems (obstruction of the inguinal canal).

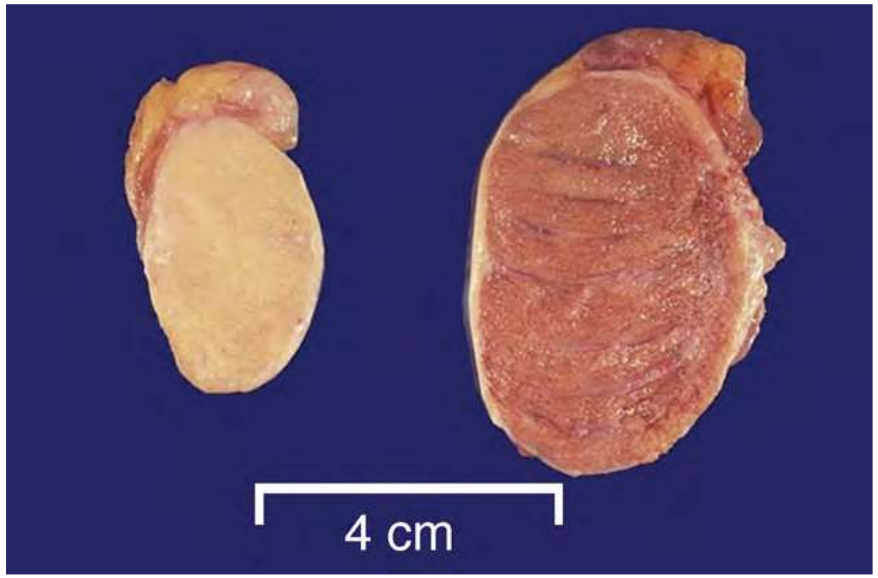
Complications of cryptorchidism:

1. Testicular atrophy.
2. Infertility.
3. Inguinal hernia.
4. **Development of malignancy**; there is increases risk of testicular malignancy by four folds & also there is increases risk of malignancy in contralateral testis.

Morphology

Gross:

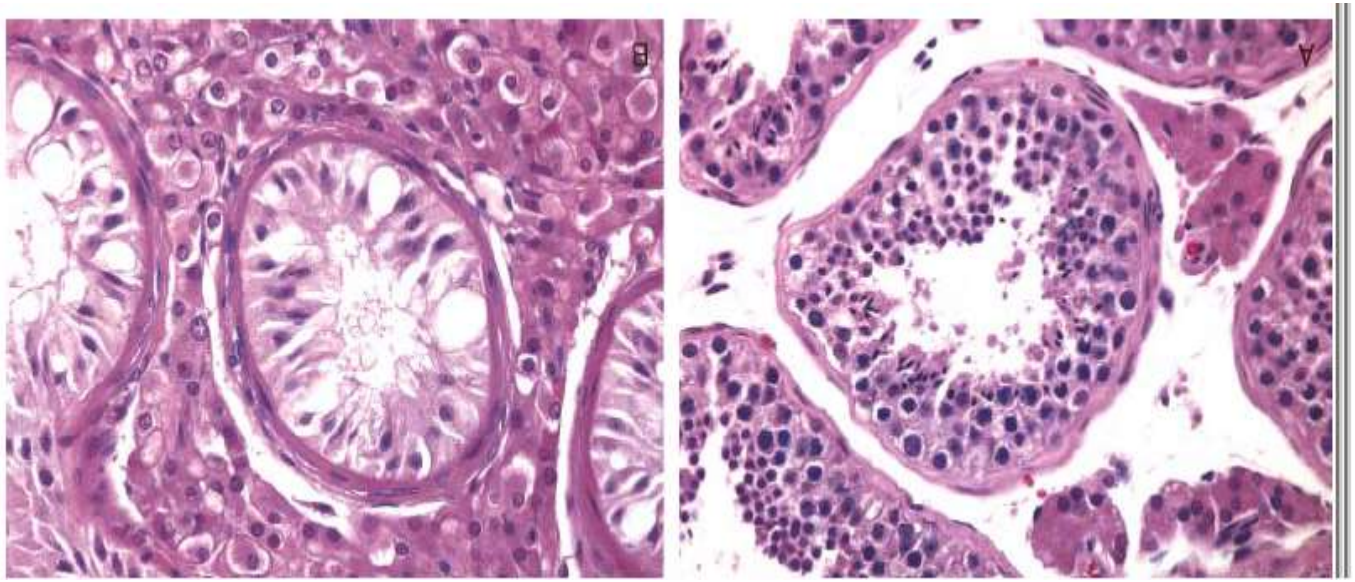
- Small, firm, and pale white.



Mic:

1. There is marked hyalinization & thickening of basement membrane of seminiferous tubules.

2. There are prominent leydig cells, hyperplastic sertoli cells & atrophy of germ cells.



Regressive Changes (Testicular Atrophy):

Causes:

1. Atherosclerosis.
2. Orchitis (inflammation of testes) mainly mumps.
3. Cryptorchidism.
4. Hypopituitarism.
5. Radiation.
6. Liver cirrhosis.
7. Prolonged administration of antiandrogens.
8. primary failure of genetic origin, such as in Klinefelter syndrome.

Testicular Neoplasms

- Less than 1% of all male malignancies.
- Peak age of incidence 15 & 34 years.
- More in whites than in blacks.
- 95% of these tumors arise from germ cells (aggressive but curable tumors) & 5% of testicular tumors sex cord- stromal tumors (usually benign tumors but associated with hormonal syndromes).

Causes of testicular neoplasia:

1. Cryptorchidism.
2. Testicular dysgenesis. Many syndromes are associated testicular dysgenesis & also associated with increased frequency of testicular malignancies.
3. Chromosomal abnormalities mainly chromosome NO. 12
4. Unknown causes

Classification of testicular neoplasia:

1. **Intratubular germ cell neoplasia.** It is mean that the malignant changes are limited to the lining of seminiferous tubules & it is now widely believed that most testicular tumors arise from intratubular germ cells tumors.

2. **Seminoma** (classic, tubular)

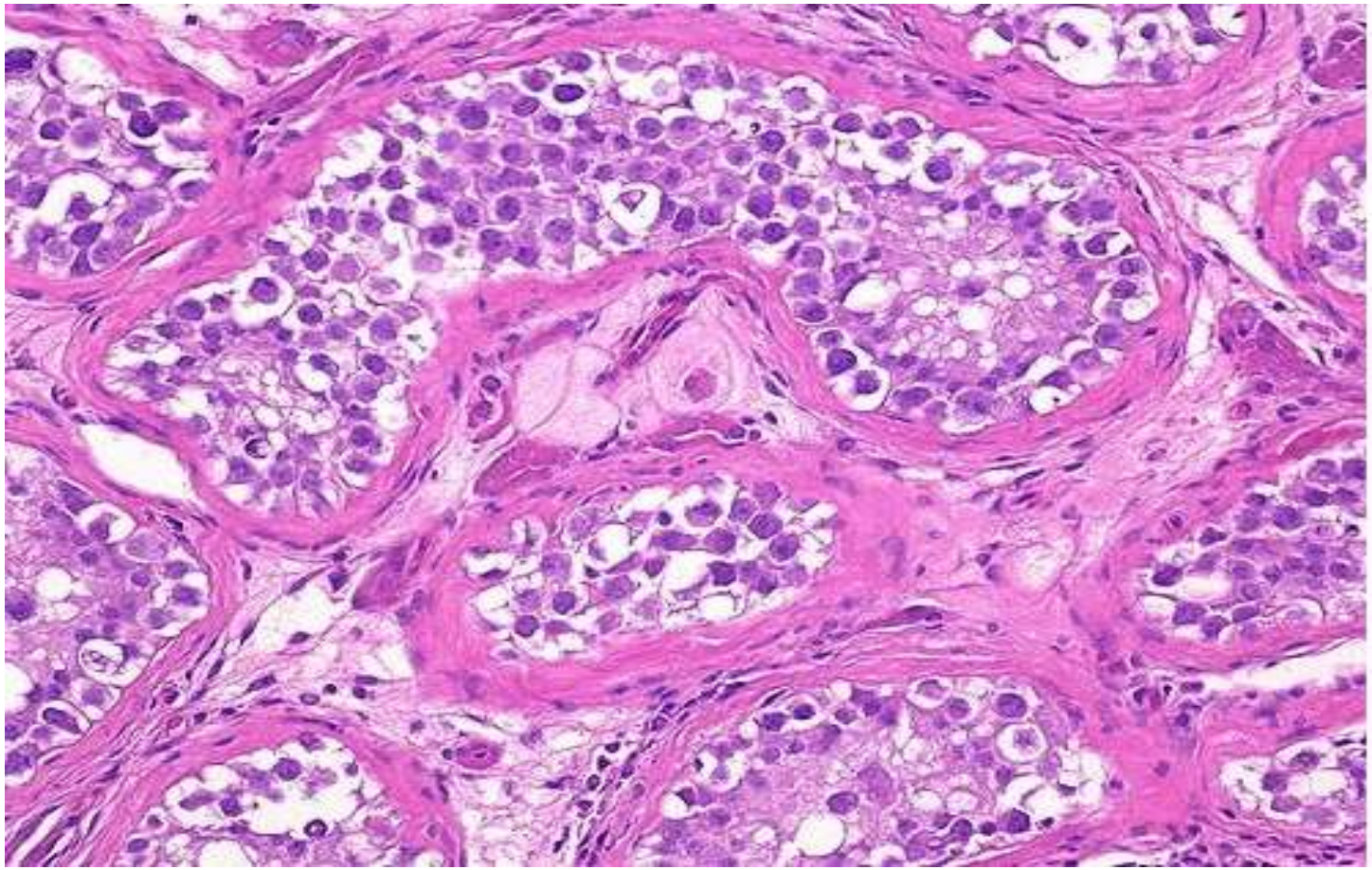
3. **Spermatocytic seminoma**.

4. **Non seminomatous germ cell tumors.**

- Embryonal carcinoma
- Yolk sac tumor
- Teratomas (mature, immature, with malignant transformation)
- Choriocarcinoma
- Mixed

5. Sex Cord–Stromal Tumors

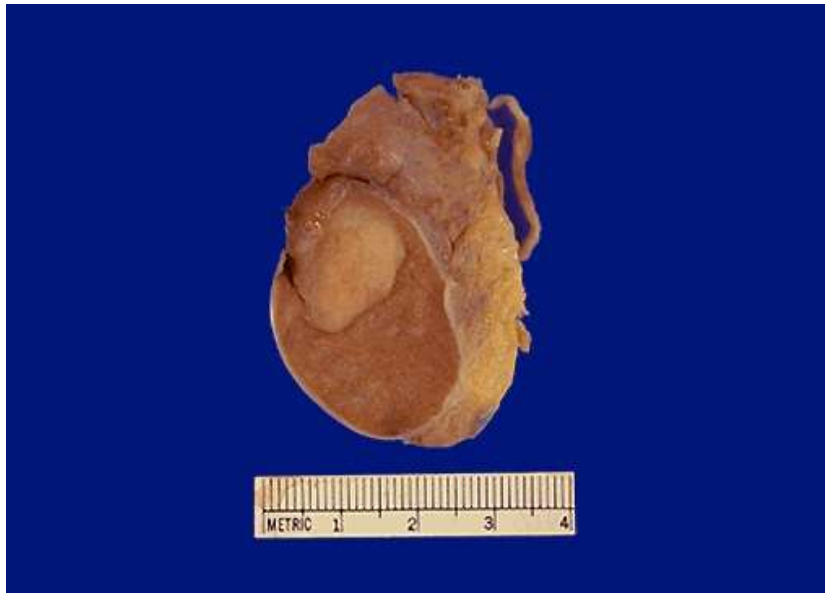
- Leydig cell tumor and Sertoli cell tumor



Seminiferous tubules with intratubular germ cell neoplasia showing large cells with clear cytoplasm,, and prominent nucleoli. There is no spermatogenesis.

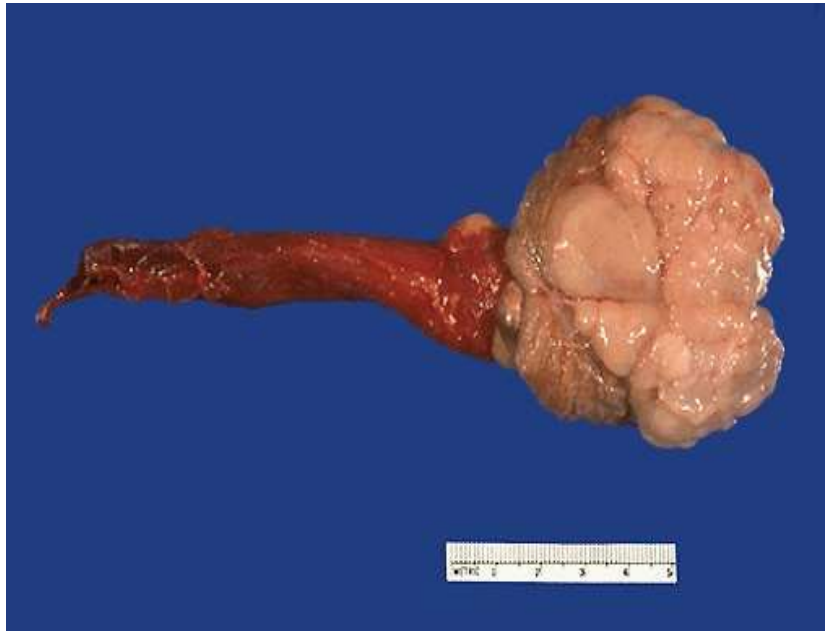
Seminoma

- 50% of testicular germ cell neoplasms.
- Most commonly between the 3rd- 5th decades of life.
- Usually sensitive to radiation and chemotherapy, with a good prognosis.



Gross.

- large, soft, well demarcated, usually homogenous.
- Gray-white, lobulated cut surface.
- Usually there is no hemorrhage.



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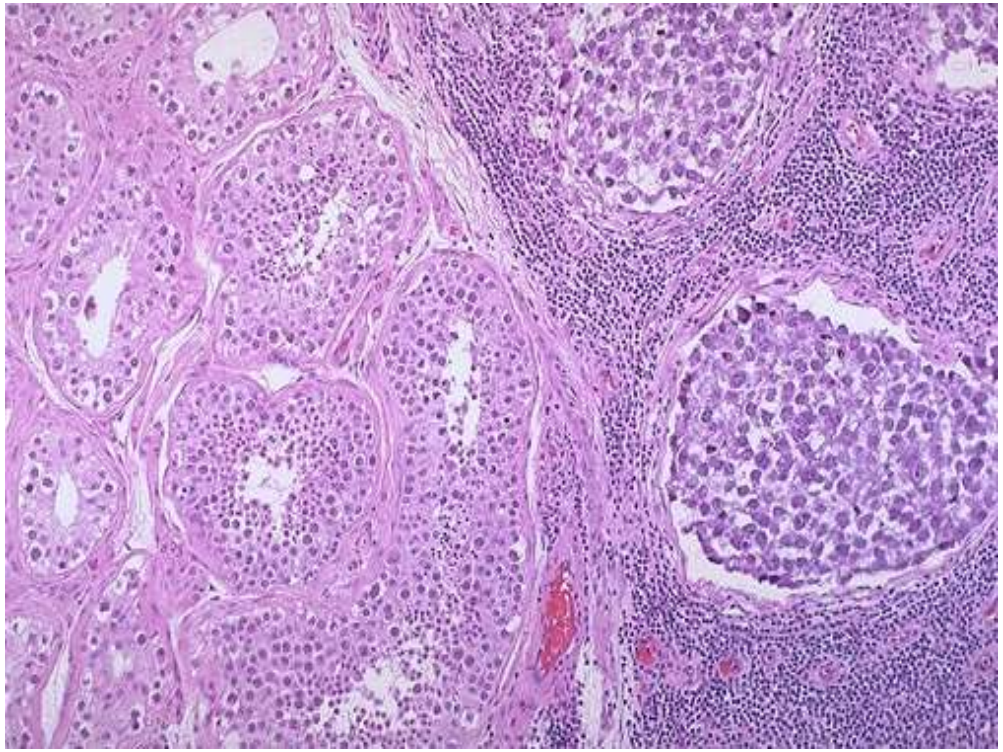
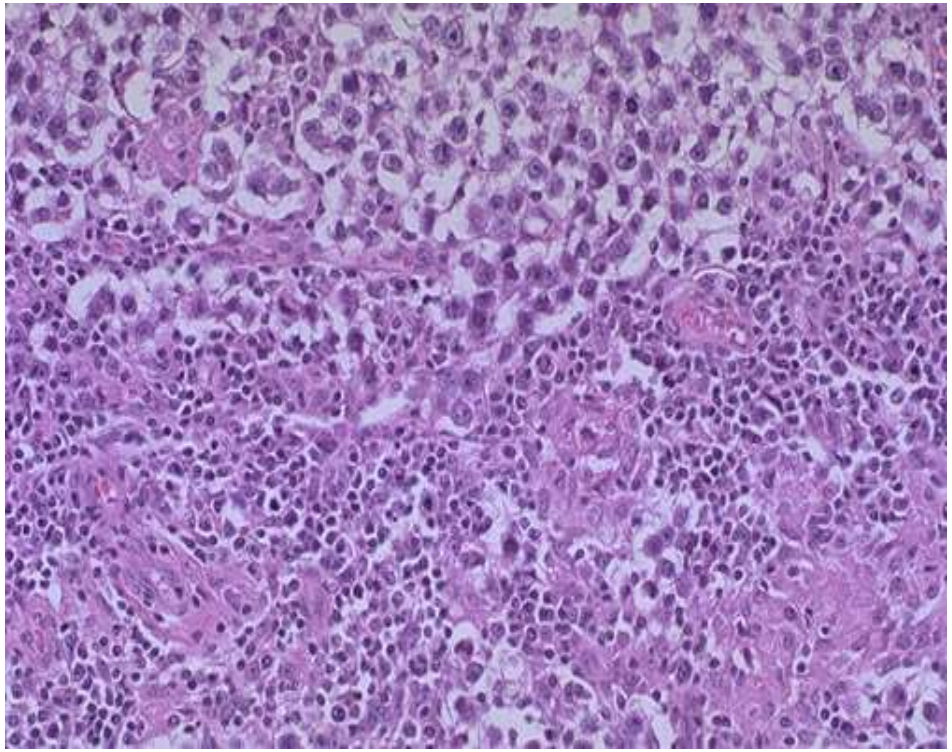


Normal testis appears at the left, and seminoma is present at the right.. Note the lymphoid stroma between the nests of seminoma.



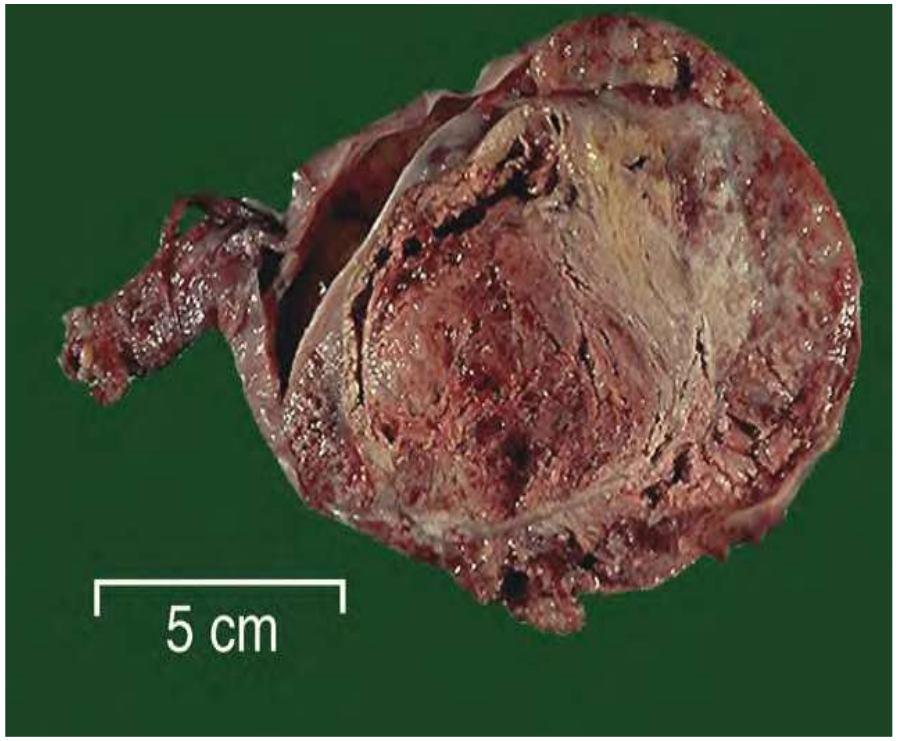
Seminoma of the testis. Microscopic examination reveals large cells with distinct cell borders, pale nuclei, prominent nucleoli, and a sparse lymphocytic infiltrate.

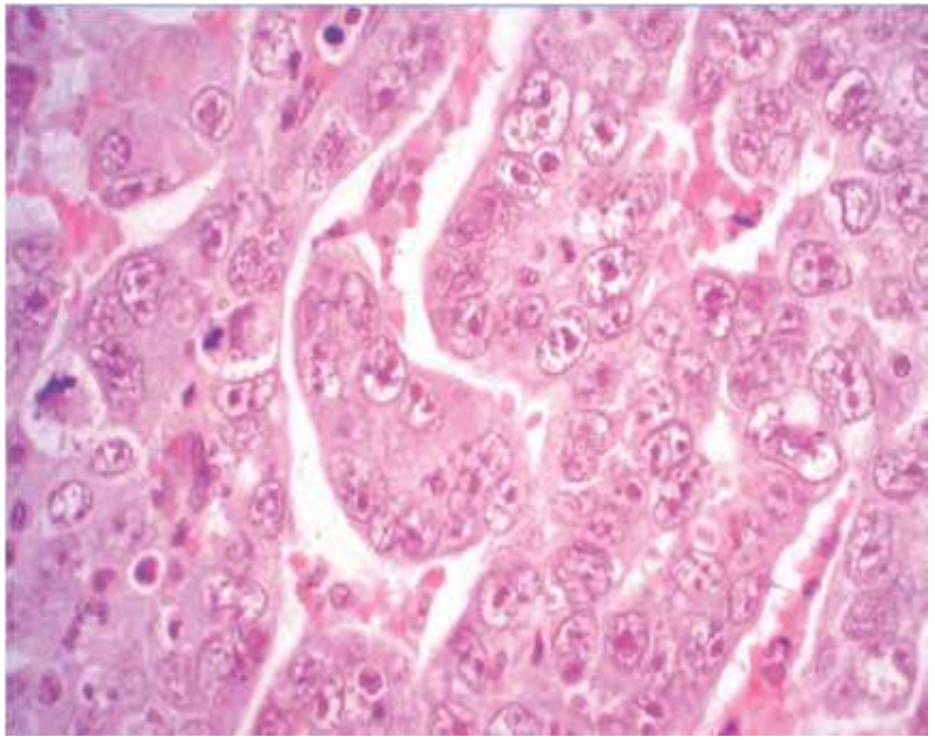
Spermatocytic Seminoma:

- Older patients; usually more than 65 years old.
- This slow-growing tumor does not metastasize, and when treated by surgical resection it has an excellent prognosis.
- Contain a mixture of small, medium sized cells and multinucleated tumor cells.

Embryonal carcinoma

- The peak incidence: 20- 30 years-old age group.
- Ill-defined invasive masses containing foci of hemorrhage & necrosis.
- Sheets of large cells with indistinct cell borders, large nuclei & prominent nucleoli.





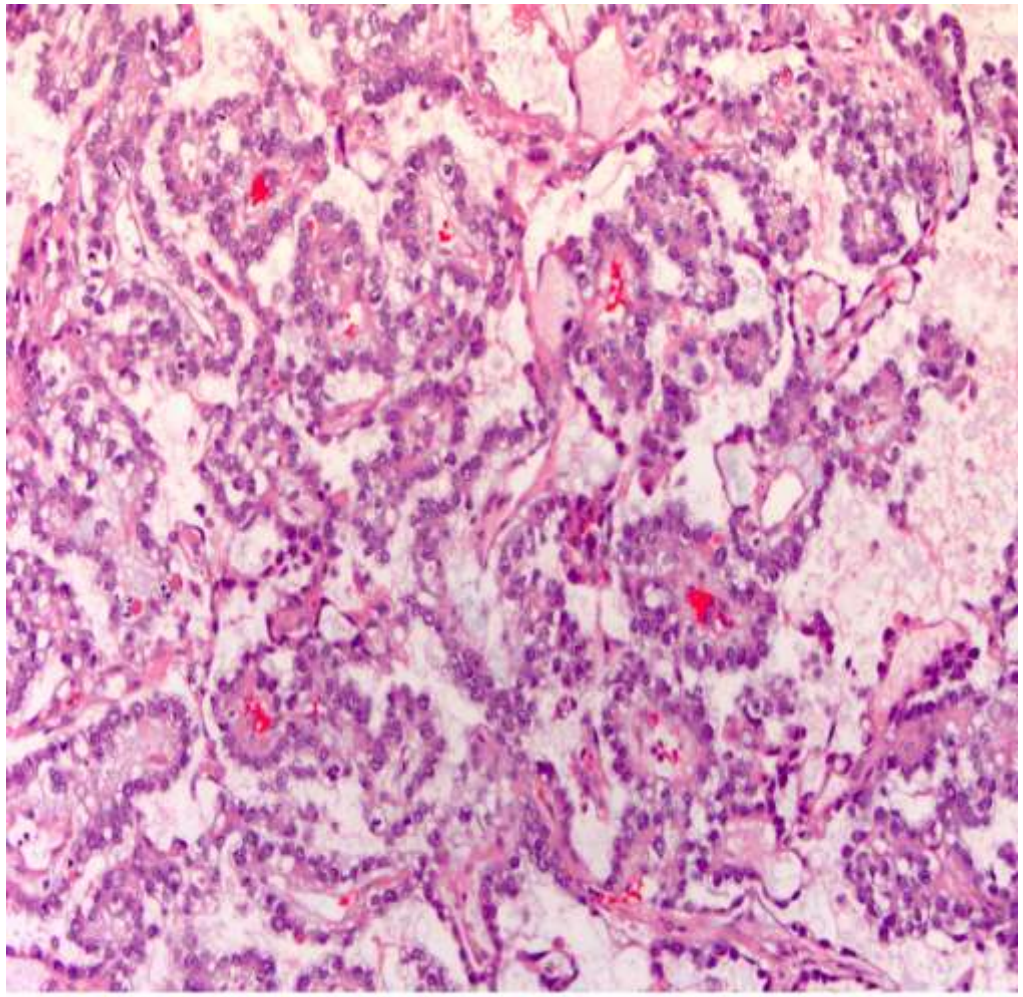
Yolk sac tumors.

- Most common primary testicular neoplasm **in children** younger than 3 years of age.
- In adults, often seen admixed with embryonal carcinoma.
- Histological examination showing**:**

1. low cuboidal to columnar epithelial cells forming sheets, glands, papillae & microcysts.

2. presence of **Schiller-Duvall bodies** (primitive glomeruli like structure).

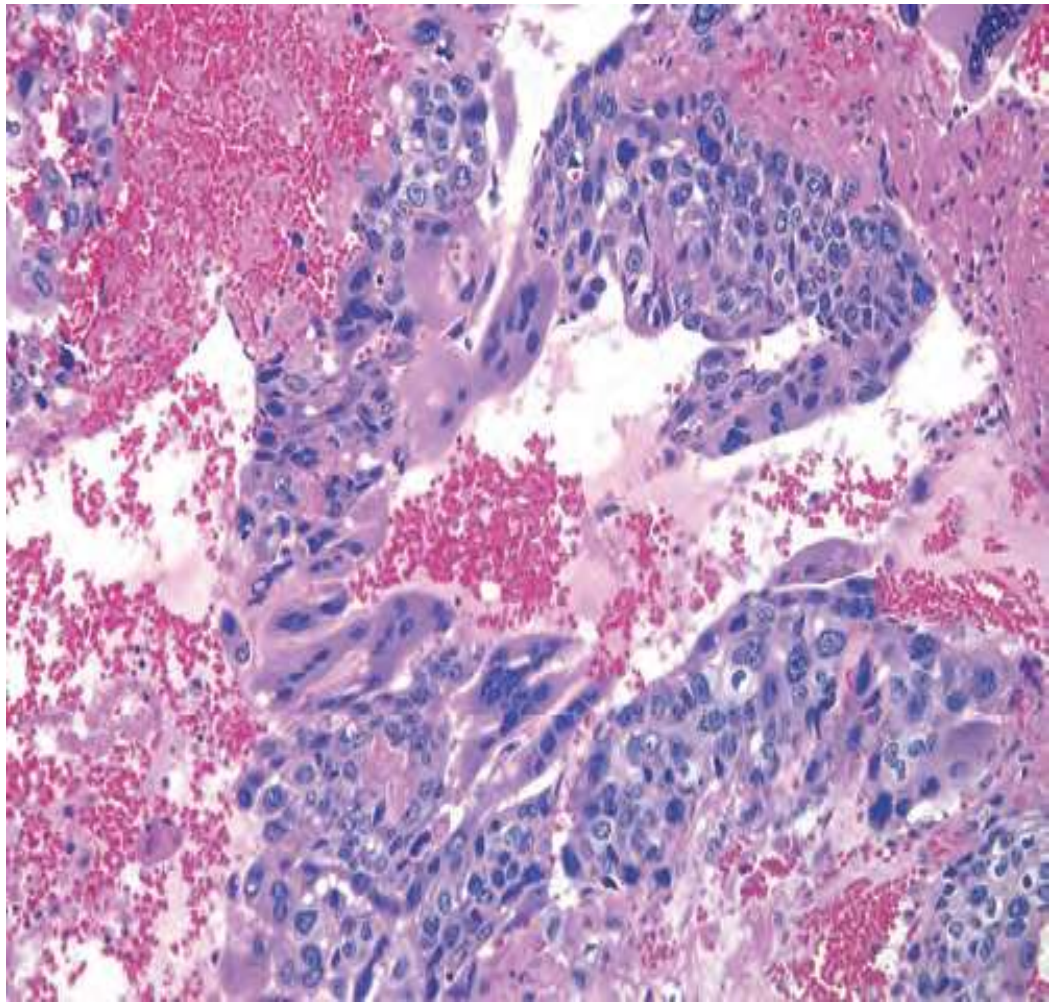
- The tumor cells produce Alfa fetoprotein (AFP), which can be detected in the serum as a tumor marker.



Papillary pattern

Choriocarcinoma

- These tumors are usually mixed with other germ cell tumors.
- Arise in placenta, ovary, mediastinum or abdomen.
- Choriocarcinoma usually small, but almost presented with metastases (mainly to the lungs, liver, CNS).
- **Microscopically:** sheets of small cuboidal cells (cytotrphoblastic cell) mixed with large, multinucleated syncytial cells (syncytiotrophoblstic cells).



Teratoma:

- Most second germ cell tumor in children under 3 years, following the yolk sac tumors.
- They are usually pure, & almost never show metastases, & are associated with many Down's syndrome, klinefelter's syndrome, spina bifida…..etc.
- **Microscopically,** three major variants of pure teratoma are recognized:

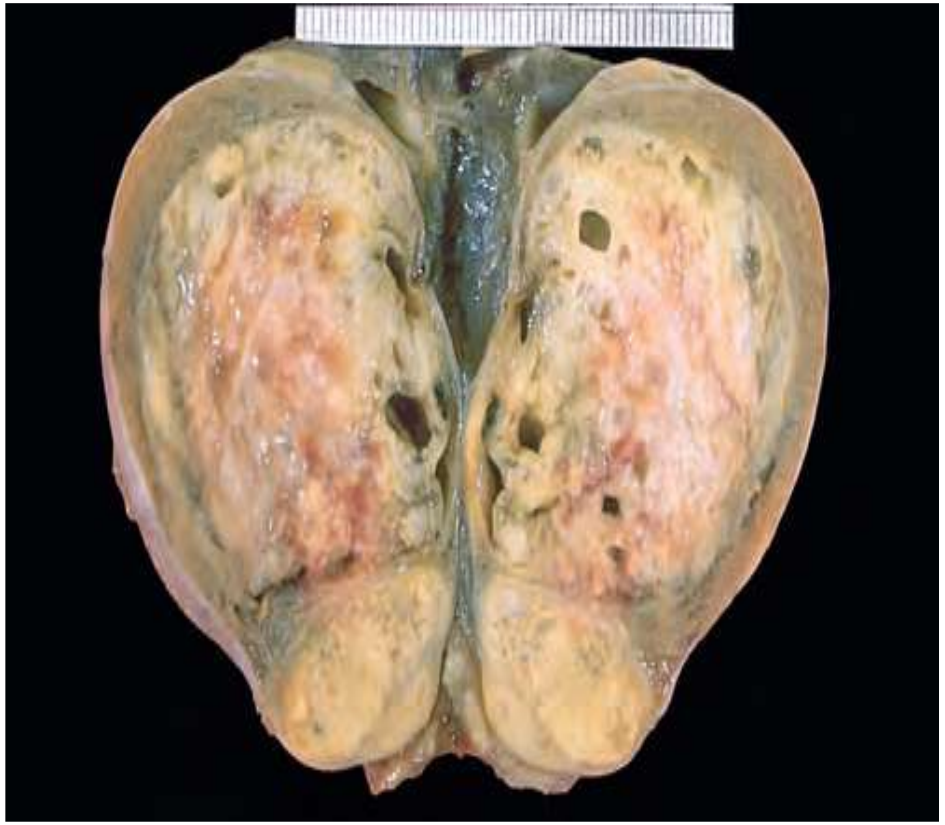
I. Mature Teratoma: Contain **fully differentiated tissues** from one or more germ cell layers (e.g neural tissue, cartilage, adipose tissue, bone, and epithelium).

II. Immature Teratoma: Usually are seen in adults, & they are **contain immature tissue** resemble to fetal tissue (primitive neural tissue, immture cartilage tissue….etc).

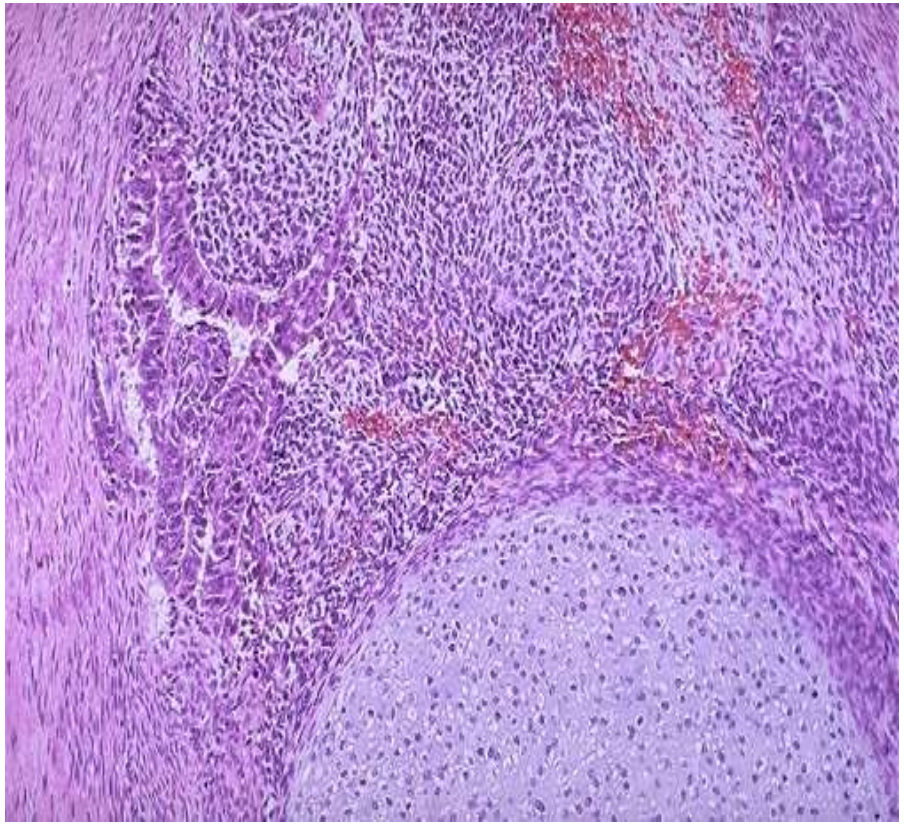
III. Teratoma with malignant transformation: Characterized by:

- Presence of malignant growth in teratoma.
- **Mainly squamous cell carcinoma, adenocarcinoma.**

Morphology of teratoma



The variegated cut surface with cysts reflects the presence of multiple tissue types.



Teratoma consisting of a disorganized collection of glands, cartilage, smooth muscle, and immature stroma.

Thank you