Pathology

Male genital system

Learning Outcomes

By the end of the lecture, you will be able to:

1.Identify congenital anomalies of male genital system

2.Identify inflammatory lesions of male genital system

3.Describe gross and microscopic picture of senile prostatic hyperplasia

4.Recognise pathologic features of prostatic and testicular tumors

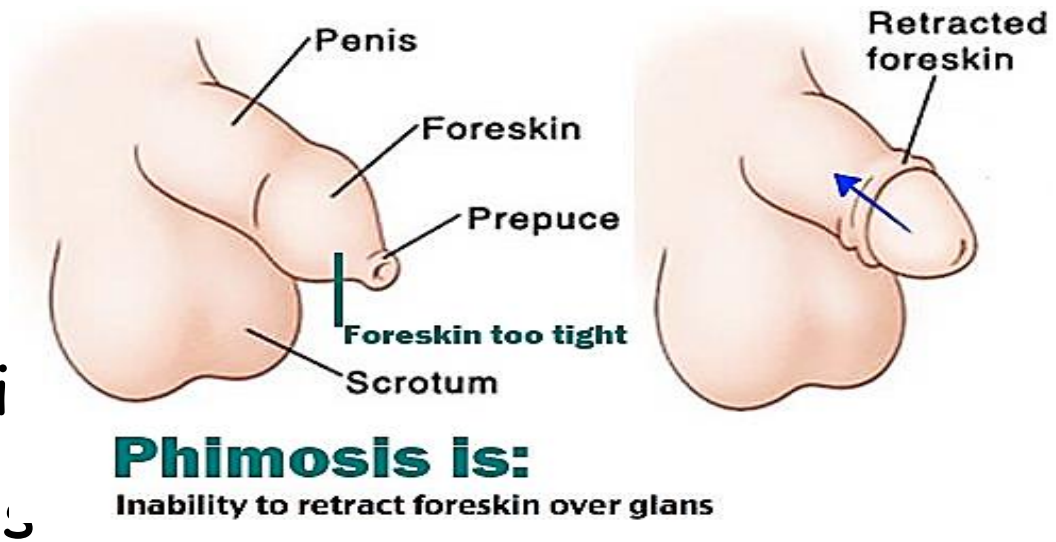
Agenda

- Congenital anomalies
- Inflammatory lesions
- BPH
- Prostatic carcinoma
- Testicular tumours
- Diseases of tunica vaginalis

Congenital anomalies

1. Phimosis:

It is narrowing of the opening of the prepuce.



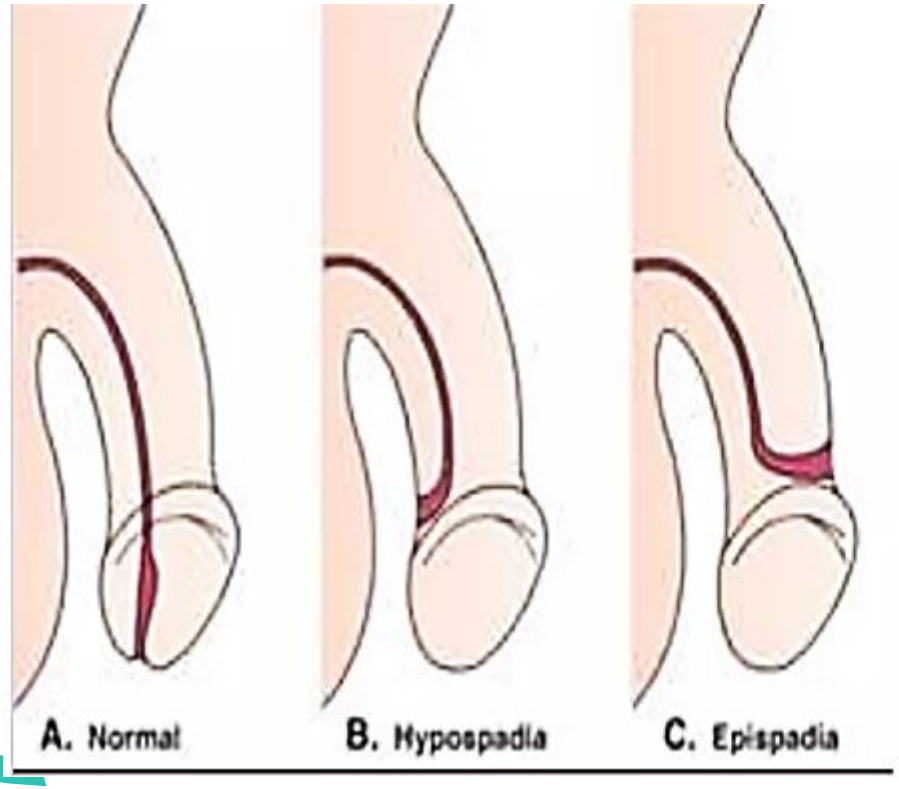
Complications:

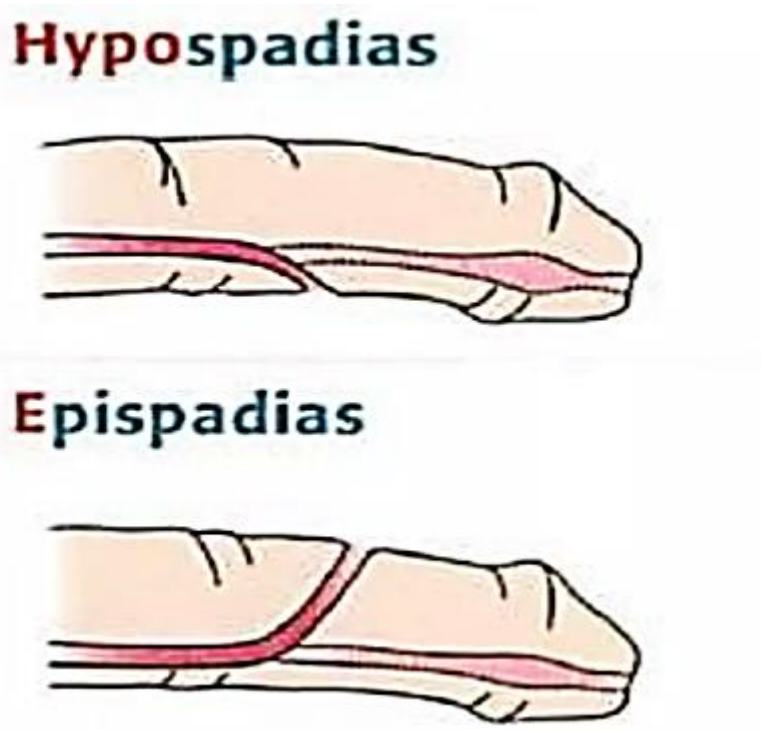
1. Balanitis: Inflammation of glans peni
2. Squamous cell carcinoma of the penis (due to accumulation of smegma that predispose to both infection and carcinoma).
3. Gradual urinary tract obstruction.

Congenital anomalies

2. Hypospadias and epispadias of the penis

- Hypospadias: the urethra opens at ventral surface of the penis.
- Epispadias: the urethra opens at dorsal surface of the penis.





Congenital anomalies

2. Hypospadias and epispadias of the penis

- Complications:

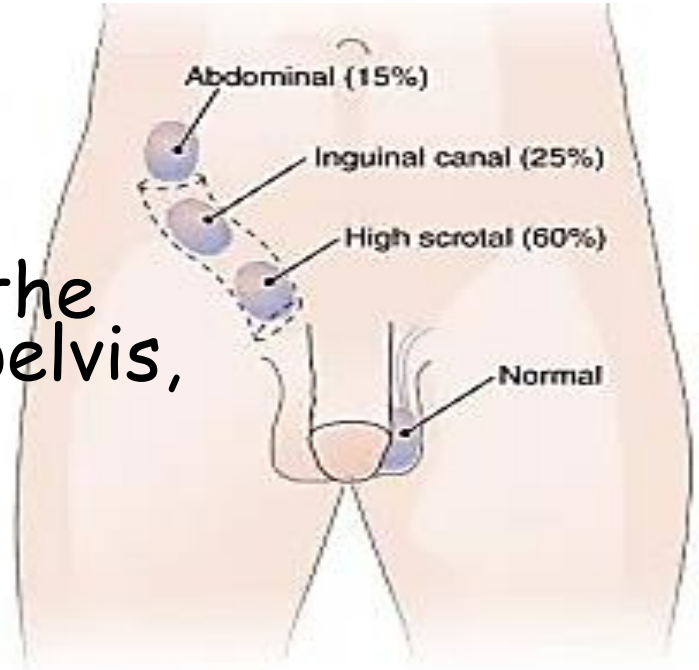
1. Urinary obstruction (as the urethral opening is often constricted).

2. Sterility (when the orifice is near the base of the penis).

Congenital anomalies

3.Cryptorchidism(undescended testis):

Most common congenital abnormality of the genitourinary tract



Def:

Failure of descent of one or both testis into the scrotum. The testis is found in the abdomen, pelvis, or in the inguinal canal.

Causes:

1. Hormonal disturbances: Deficiency of gonadotrophic hormone of pituitary
2. Obstruction of the pathway: Organic obstruction prevents the descent.
3. Other defects: Short spermatic vessels or vas deferens or mal-development of the scrotum or cremaster muscle.

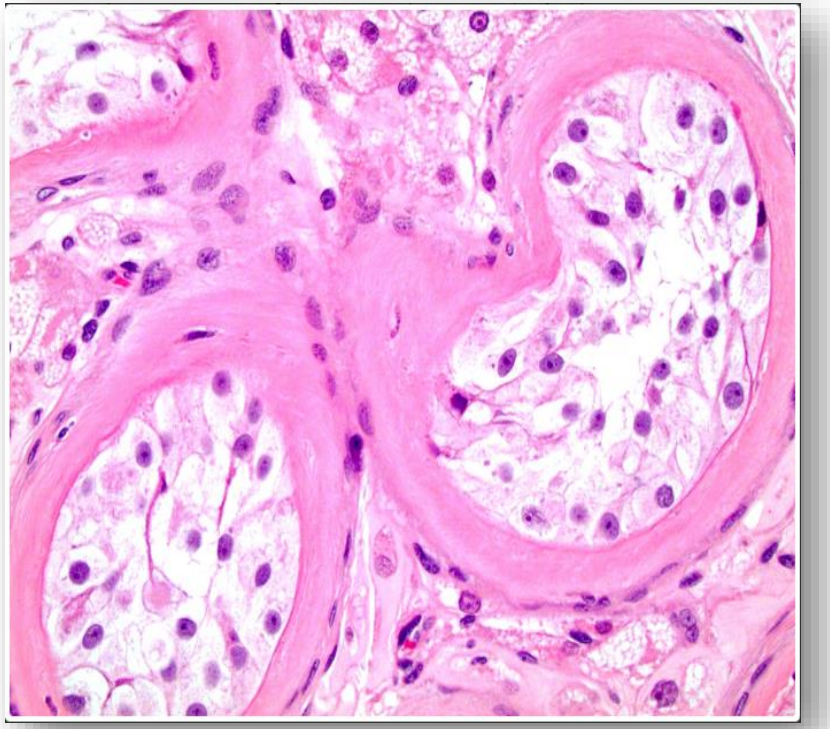
Congenital anomalies

3.Cryptorchidism(undescended testis):

ME:

-Atrophy of the seminiferous tubules with fibrosis.

-Germ cell neoplasia insitu which is likely a precursor of subsequent germ cell tumors



Complications:

Infertility (in bilateral cases).

Malignancy (precancerous for seminoma).

Inflammatory diseases

Prostatitis

Inflammation of the prostate.

Causes:

1. Organisms: E-coli, gonorrhea, staph. aureus, strept. pyogens…etc.
2. Routes of infection:

- Direct spread from cystitis, urethritis, urethral instrumentation.
- Blood borne infection

Pathology:

1. Acute suppurative prostatitis which may form abscess.
2. Chronic nonspecific prostatitis follows acute.

Inflammatory diseases

Seminal vesiculitis:

Inflammation of seminal vesicles.

Causes:

It may be 2ry to prostatitis or blood borne.

Funiculitis:

Inflammation of spermatic cord.

Causes:

1. Direct spread from epididymis.
2. Lymphatic spread from seminal vesicles.
3. Blood spread.

Effect: It leads to diffuse thickening of spermatic cord.

Inflammatory diseases

Orchitis:

Inflammation of testis.

Causes:

1. Bacterial orchitis:

Due to spread of infection from:

a) Epididymis directly

b) Spermatic cord by lymphatic or through vas deferens

c) Blood borne

Pathology: Acute inflammation with suppuration resulting in scarring and sterility.

2. Viral: mumps orchitis
3. Traumatic orchitis
4. Autoimmune orchitis

Inflammatory diseases

Chronic specific inflammation of male genital system:

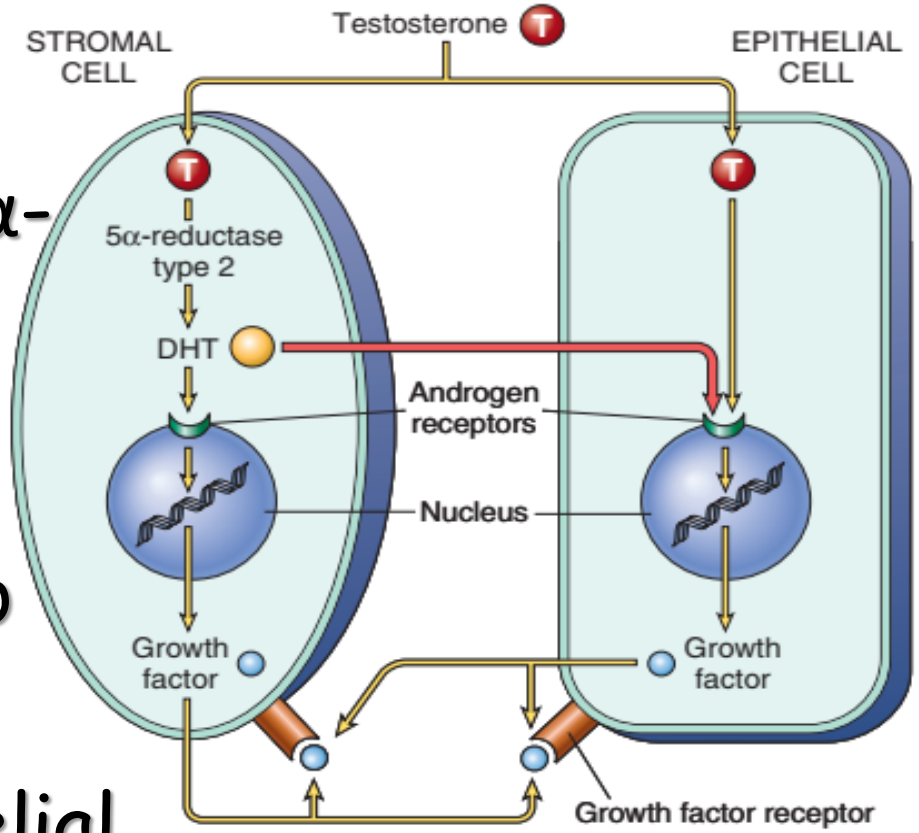
a) Tuberculosis.
b) Syphilis.
c) Filariasis
d) Bilharziasis.

Benign(senile) prostatic hyperplasia

- Extremely common.
- ≈50% of men at 50 Years are affected, ≈95% are affected above 70 Years.
- Gradual onset with slow progression

Pathogenesis:

- Hormonal changes with increase in 5 α-reductase enzyme in prostate that convert testosterone to dihydroxytestosterone (DHT)
- Accumulation of DHT(10 times more potent than androgens in combining to the androgen receptor and dissociate more slowly).
- Increasing the proliferation of epithelial & stromal cells and decreasing epithelial cells apoptosis.



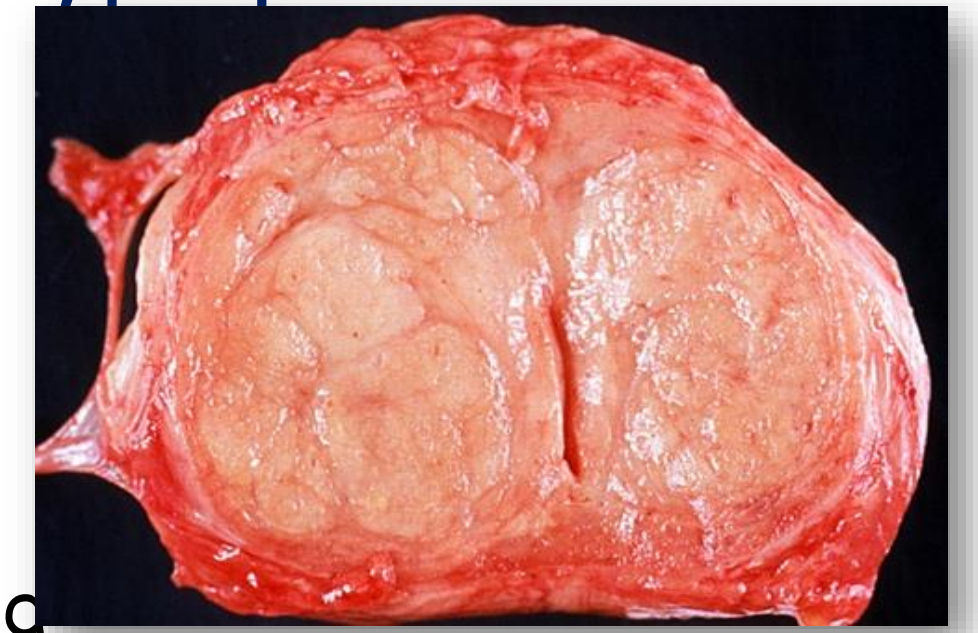
Benign(senile) prostatic hyperplasia

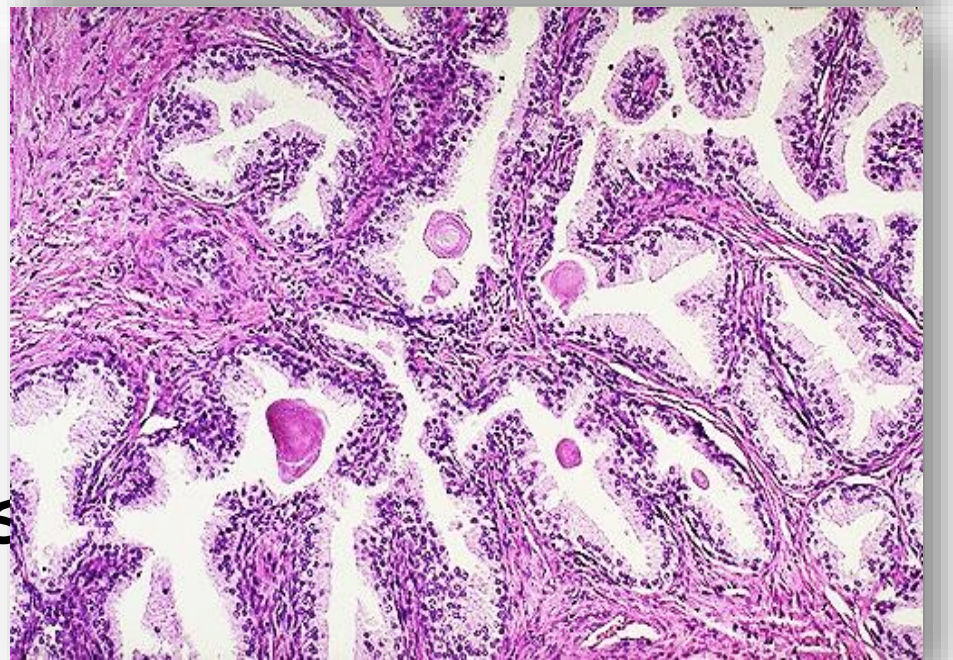
Naked Eye:

- The gland is enlarged nodular, may show cystic change.
- Firm, rubbery in consistency.

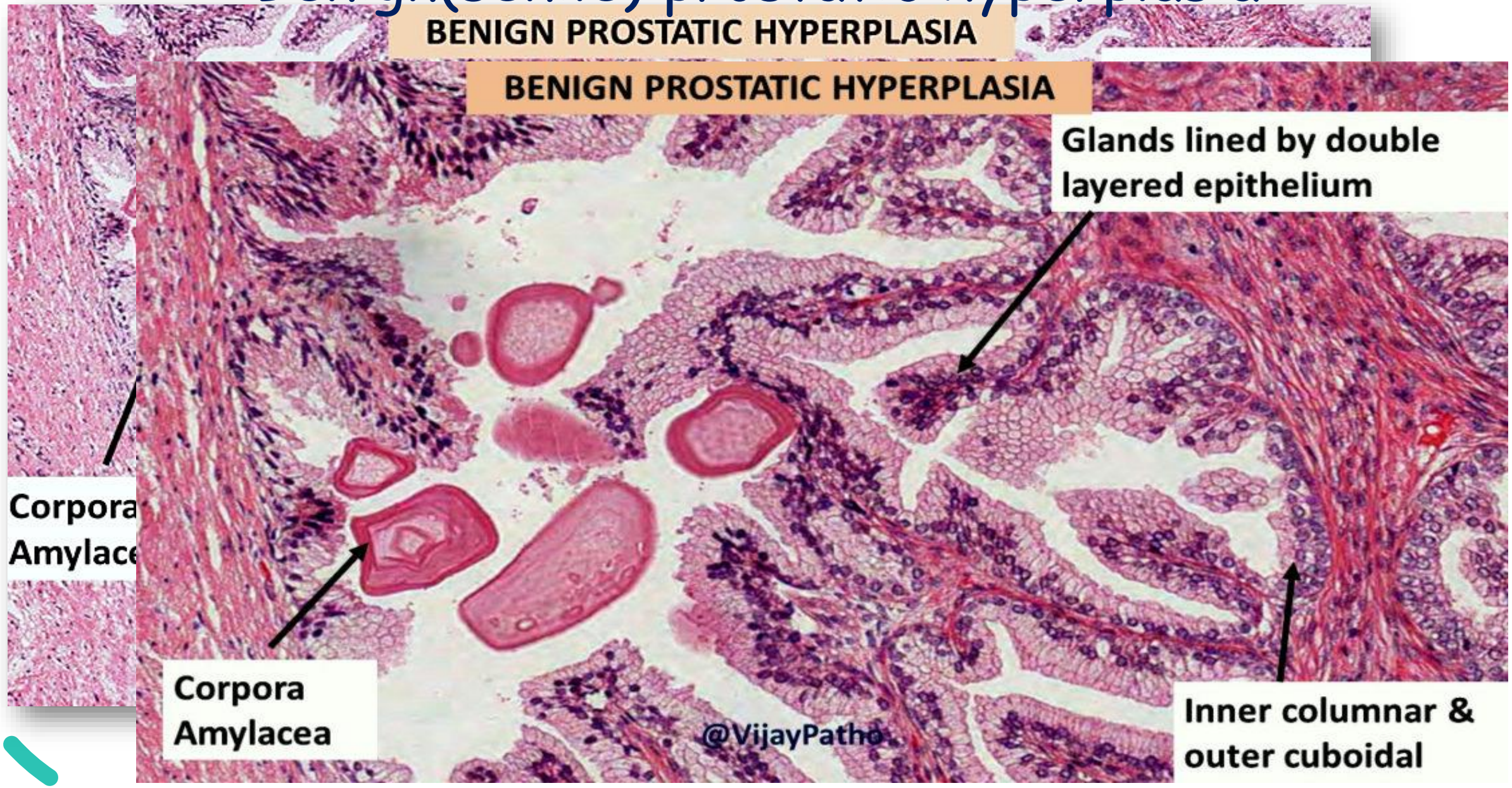
Microscopic Examination:

- Hyperplasia of the acini which are larger than normal, variable in size and shape and lined by tall epithelium with frequent papillary projections.
- The acini may contain numerous corpora amylacea.
- Hyperplasia of the fibromuscular stroma with lymphocytes &plasma cells





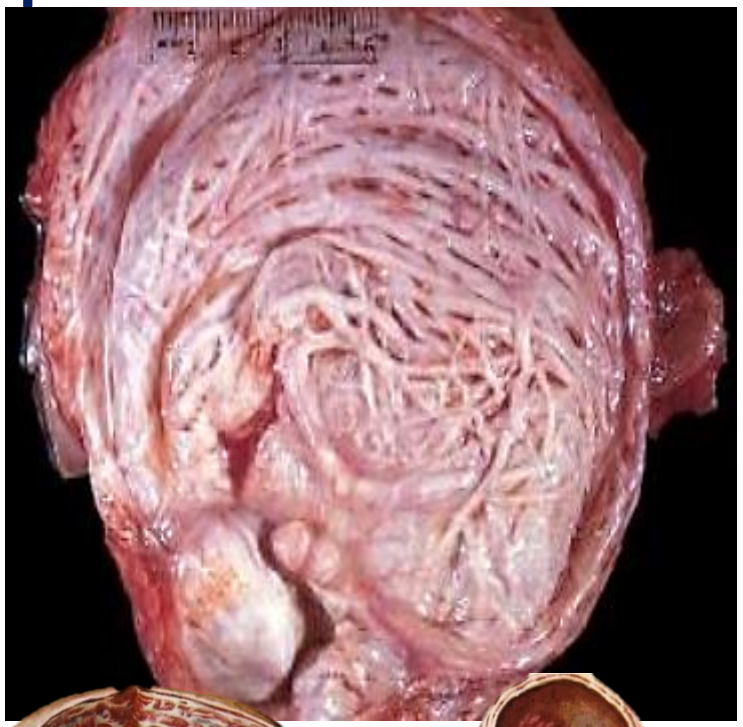
Benign(senile) prostatic hyperplasia



Benign(senile) prostatic hyperplasia

Clinical picture:

- Obstructive symptoms (Prostatism) due to compression on prostatic urethra and internal urethral meatus (interferes with the sphincter mechanism).



Complications :

Gradual urinary tract obstruction:

- Urethra: compressed as a slit.
- Urinary bladder: trabeculations, diverticulations, cystitis, stones.
- bilateral hydroureter, pyoureter.
- bilateral hydronephrosis, pyonephrosis.
- Chronic renal failure.

No relation to malignancy

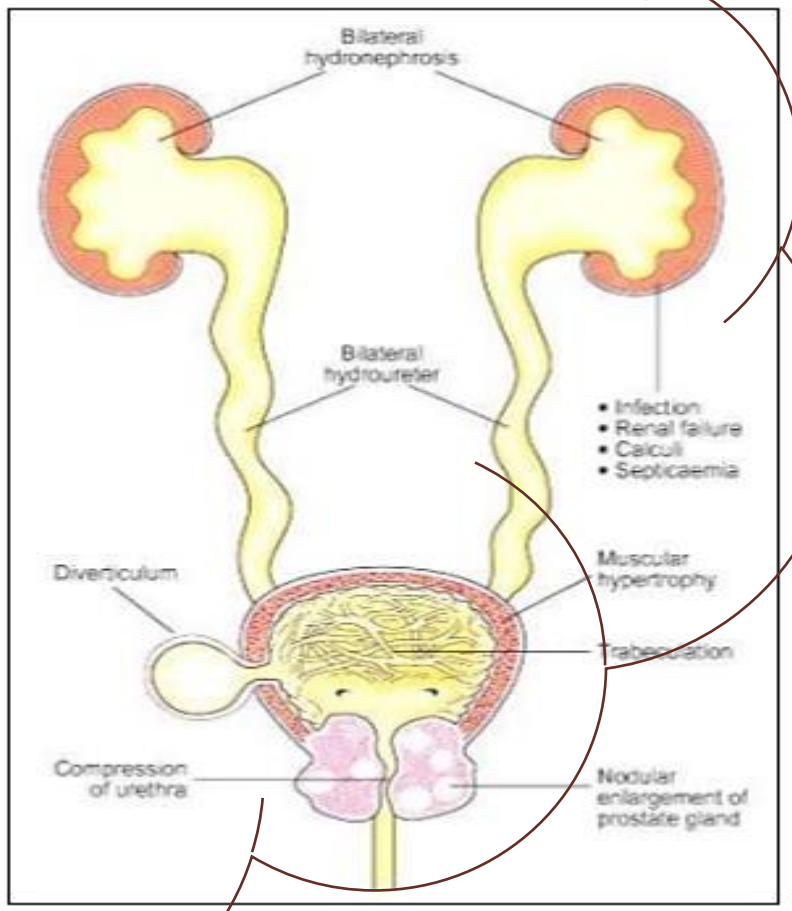
Benign(senile) prostatic hyperplasia

Prostatism:
Hesitancy
Straining to void,
Interrupted flow,
Incomplete emptying,
Terminal dribbling

BPH-Complications:

1. Obstructive Uropathy
2. Bladder hypertrophy
3. Trabeculation
4. Diverticula formation
5. Hydroureter – bilateral
6. Hydronephrosis
7. Lithiasis / stone.
8. Secondary infection.

- Not a risk factor for Carcinoma prostate.



Prostatic Carcinoma

The most common malignant tumor in males >50 years

Predisposing factors :

- Racial factors- More common in African
- Endocrinologic factors- Androgens (so, Orchiectomy reduce the tumor size in Prostatic carcinoma patient).
- Environmental factors-high fat diet, exposur polycyclic aromatic hydrocarbons
- Genetic basis- familial cases (Chromosome No 1 and 10)

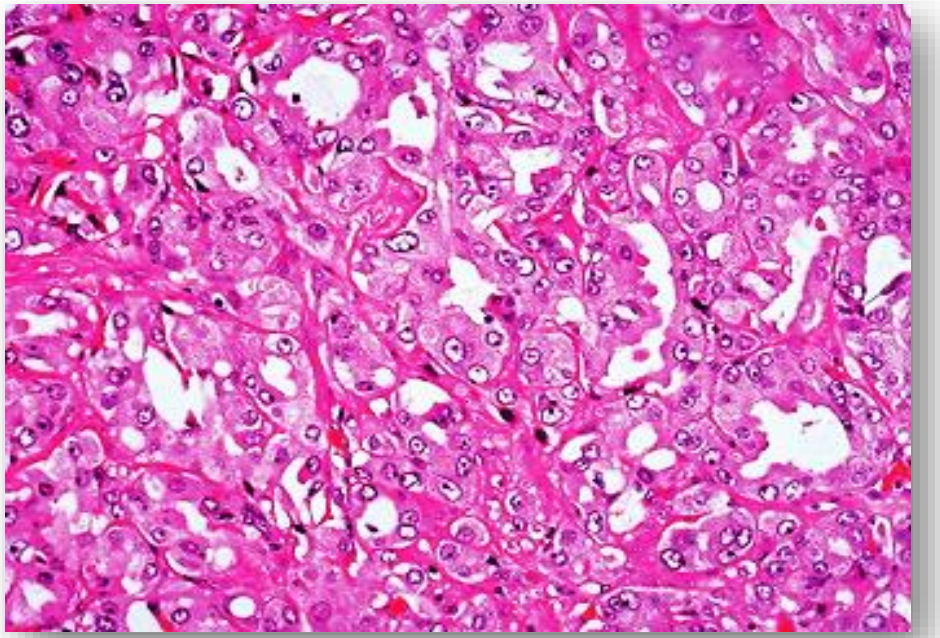
NE:

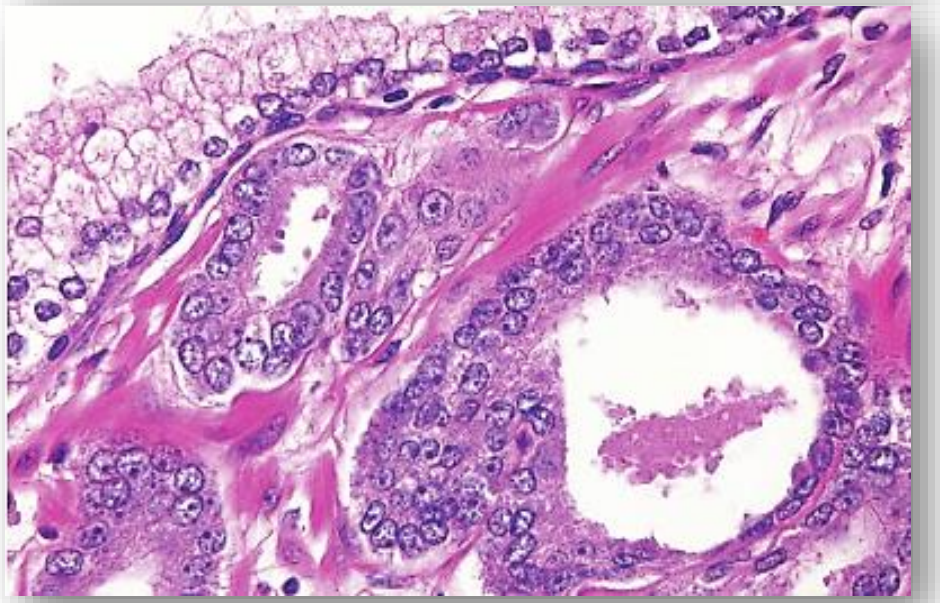
Affect the peripheral zone of the posterior lobe
Uncapsulated mass palpable in rectal exam
Multifocal, gritty and firm

Prostatic Carcinoma

ME:

- **Adenocarcinoma;** malignant glands are lined cells having **prominent nucleoli.**
- Tumor cell produce **mucin** and **acid phasphatase**
- **Gleason Score:** is the system to grade the tumor based on the degree of differentiation among the cells.

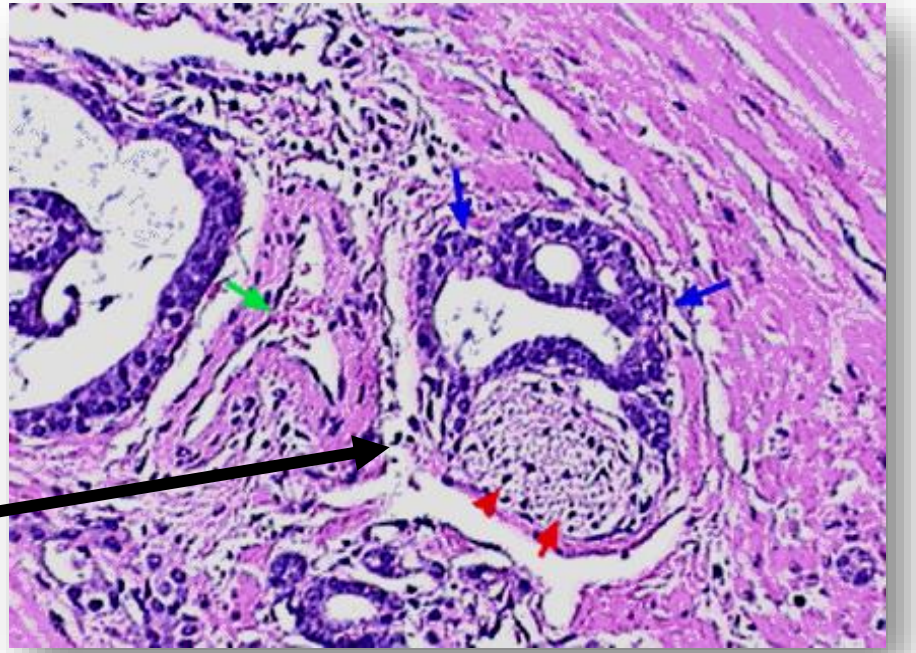




Prostatic Carcinoma

Spread:

- Local spread: to prostatic urethra , seminal vesicles, floor of urinary bladder and rectum
- Lymphatic spread: To para-aortic, iliac lymph nodes
- Perineural invasion
- Hematogenous metastases: bone(most often found in the vertebrae & sacrum), lung, liver
- Bony metastases are often osteoblastic & are associated with elevated serum **alkaline phosphatase**



Prostatic Carcinoma

Tumor markers:

- PSA :used as screening test and for assessing the response to treatment.
- CEA
- Prostatic acid phosphatase.
- Alkaline phosphatase in bone metastasis

Testicular Tumors

Germ Cell Tumor(90%):

- Seminoma.
- Non-seminoma:

- Teratoma.
- Yolk sac: Produce a fetoprotein
- Embryonal carcinoma.
- Choriocarcinoma: Produce HCG.

- Combined (e.g seminoma+ teratoma).

Lymphoma (7%):

Non-germ cells tumors(3%):

- Leydig cell tumor:

- Produce androgen (precocious puberty).

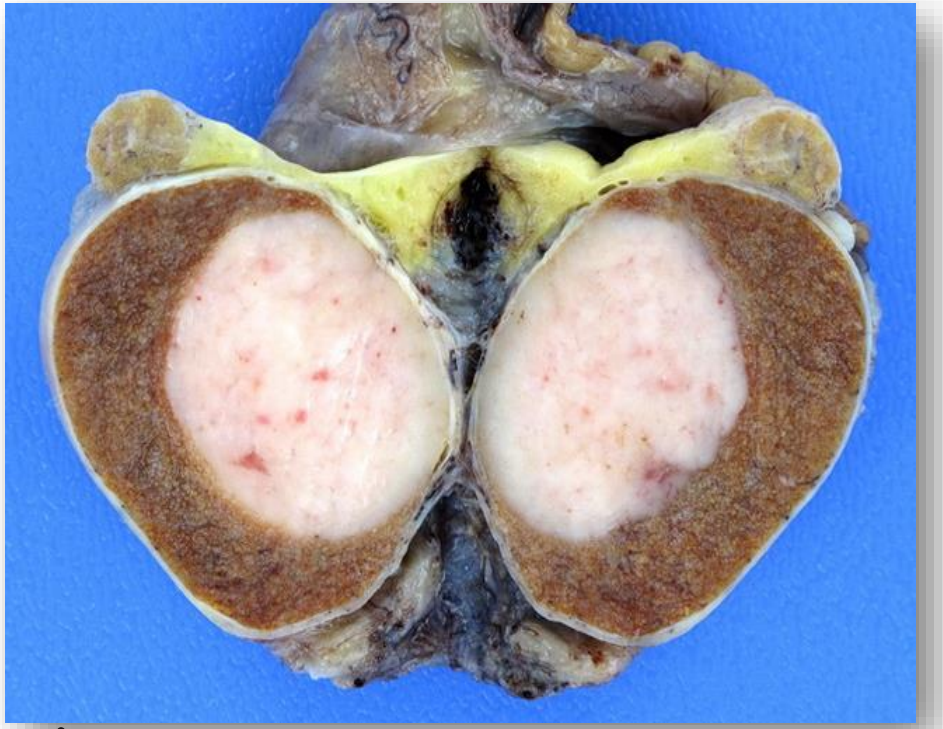
- Sertoli cell tumor:

- Produce estrogen (feminizing characters as gynecomastia).

Germ cell Tumors

Seminoma

Most common germ cells with gonadal differentiation.
Age: 40-50y. Unknown before puberty.
Radiosensitive.



Predisposing factors:

- Genetic factors
- Undescended testis
- Atrophic testis
- Venereal diseases (infections).

NE:

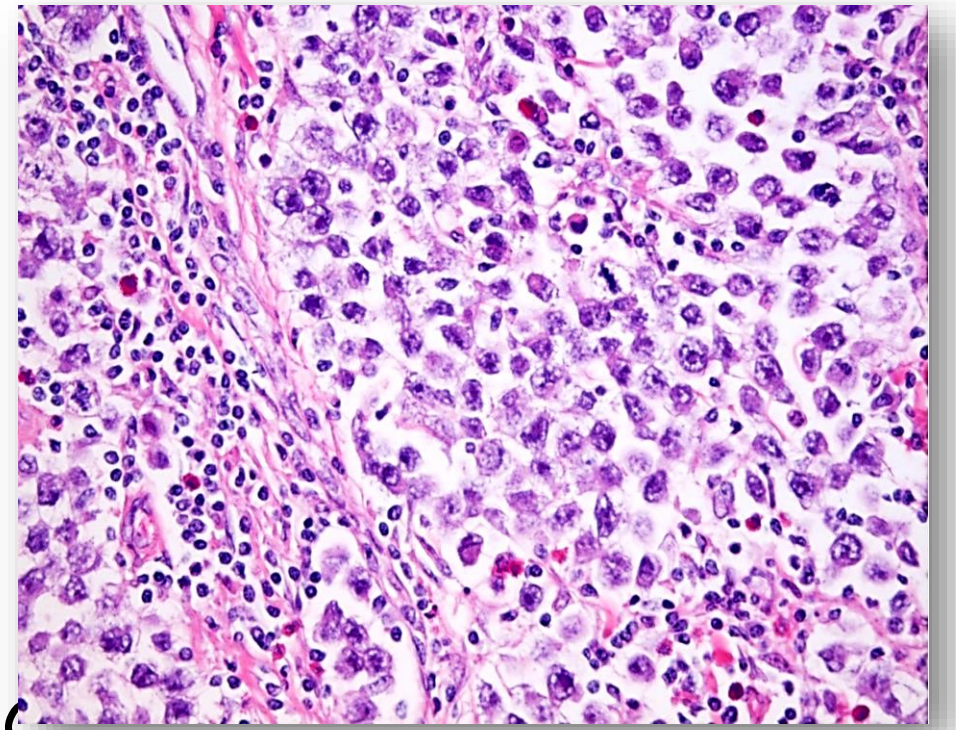
Well defined, fleshy, homogenous cut section (potato like).

Germ cell Tumors

Seminoma

ME:

- Nests and sheets of Spermatogenic cells with clear cytoplasm (due to glycogen content) with, large nucleus and prominent nucleolus.
- Tumour cells are separated by fibrous septa rich in lymphocytes and plasma cells



Spread:

- Local: tunica vaginalis, epididymis, spermatic cord and scrotal skin.
- Lymphatic: common iliac and pancreatic L.N. → mediastinal and supraclavicular L.N
- Blood: Lung, then brain and bones.

Germ cell Tumors

Non-seminoma

Less common germ cells with embryonic differentiation.
Age: 20y, may occur before puberty.
Radioresistant.
Teratoma formed of tissues derived from ectoderm, mesoderm and endoderm
Yolk sac : secretes Alpha fetoprotein.
Embryonal carcinoma.
Choriocarcinoma: secretes human chorionic gonadotrophins (HCG).
Combined (e.g seminoma+ teratoma).

Non-germ cell Tumors

- Most of these are benign, representing 3% of testicular tumors and arise from non germinal cells (Leydig and sertoli cells). Called sex cord stromal tumors
- They include:

1. Leydig (interstitial) cell tumor: It produces androgens which leads to:

Before puberty it leads to sexual precocity.

After puberty, no body changes are detected.

2. Sertoli cell tumor: It produces estrogen leading to feminizing characters.

Tunica Vaginalis

Hydrocele

Collection of serous fluid within tunica vaginalis.

Types and causes:

a) Primary: unknown cause

b) Secondary due to:

1. Diseases of testis, epididymis and spermatic cord trauma, torsion of testis, inflammations and tumors.
2. Generalized oedema.

Effects:

Pressure atrophy of testis

2ry infection (pyocele).

Haematocele

Collection of blood within tunica vaginalis.

Types and causes:

1. Primary: unknown cause
2. Secondary due to:
   - Trauma
   - Malignant tumors of testis.
   - Haemorrhagic blood diseases

Complications (effects):

- Pressure atrophy of testis.
- 2ry infection (pyocele).
- Organization & fibrosis.

Chylocele

Accumulation of lymphatic fluid within tunica vaginalis.

Cause:

It is due to lymphatic obstruction e.g. in filariasis

Varicocele

It is varicosity of the pampiniform plexus of veins in the spermatic cord.

Causes:

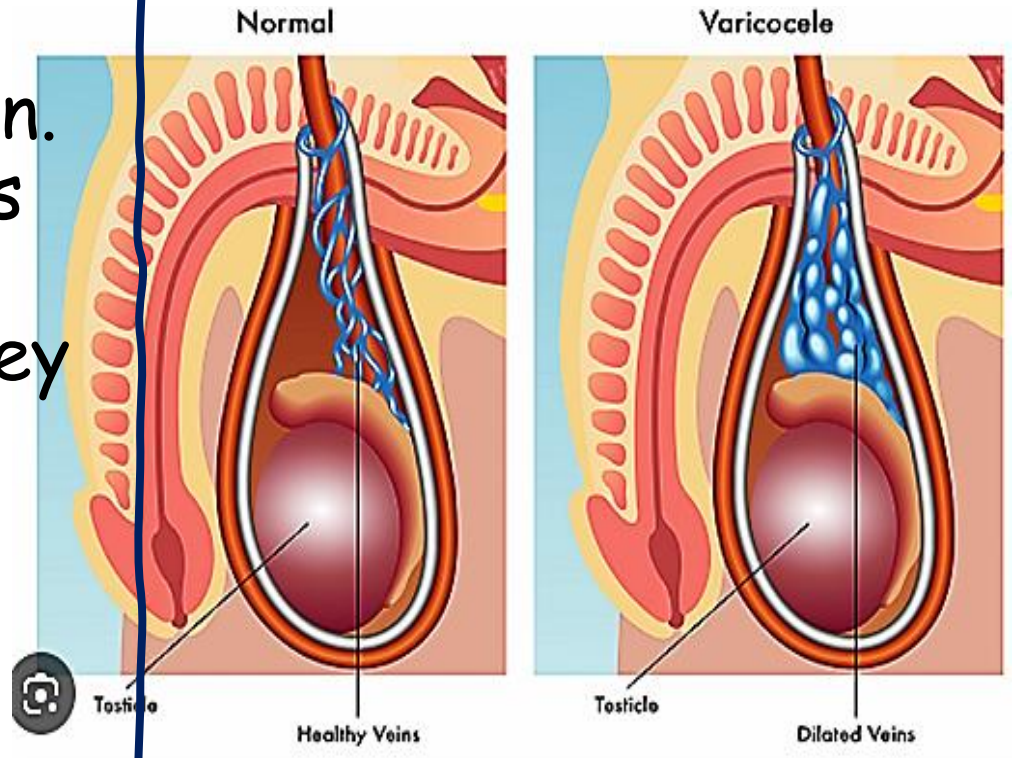
1. Primary: Occur in young unmarried men.
2. Secondary: Usually in middle age. It is due to:

- Pressure on the spermatic vein as kidney tumor
- Right side heart failure
- Venous thrombosis

Pathology:

a) Bag of warm like masses is felt in the scrotum

b) Defective spermatogenesis



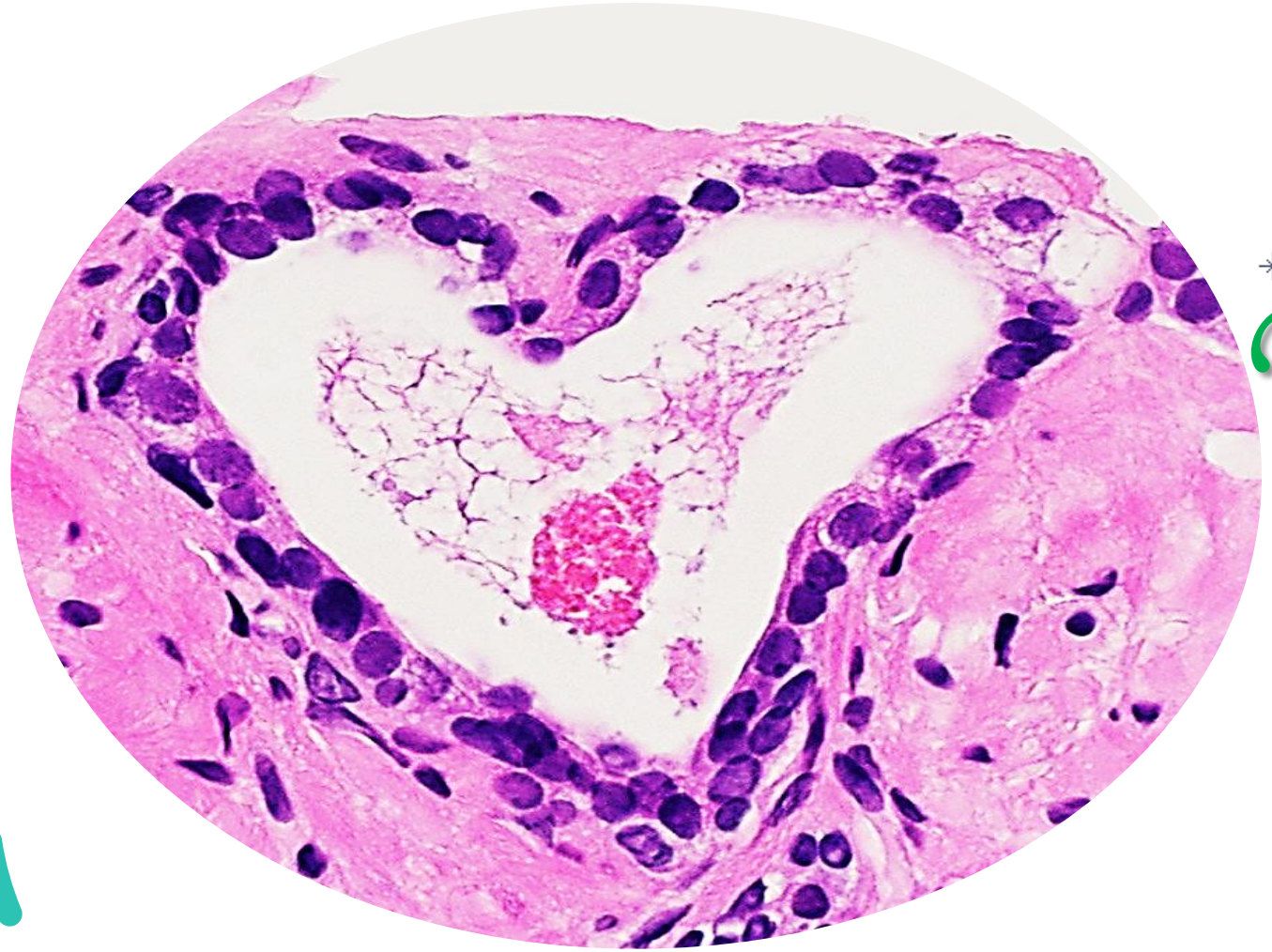
Discussion & Feedback

References & recommended readings

1. Robbins & Cotran Pathologic Basis of Disease, (Robbins Pathology), 2018 ISBN: 978-0-323-35317-5, Edition: 10th
2. Webpath:

   https://webpath.med.utah.edu/webpath.html

   https://www.pathologyatlas.ro/index.php



Thank you