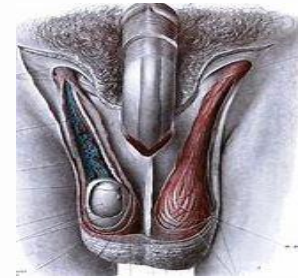
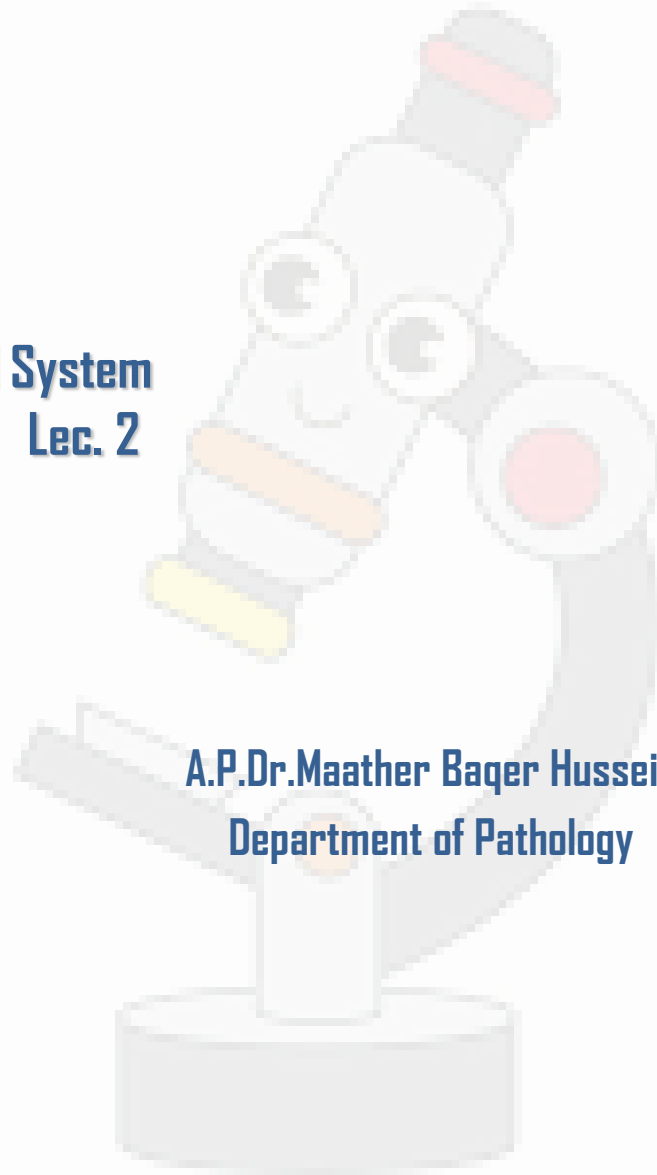


**Male Genital System
Pathology Lec. 2**



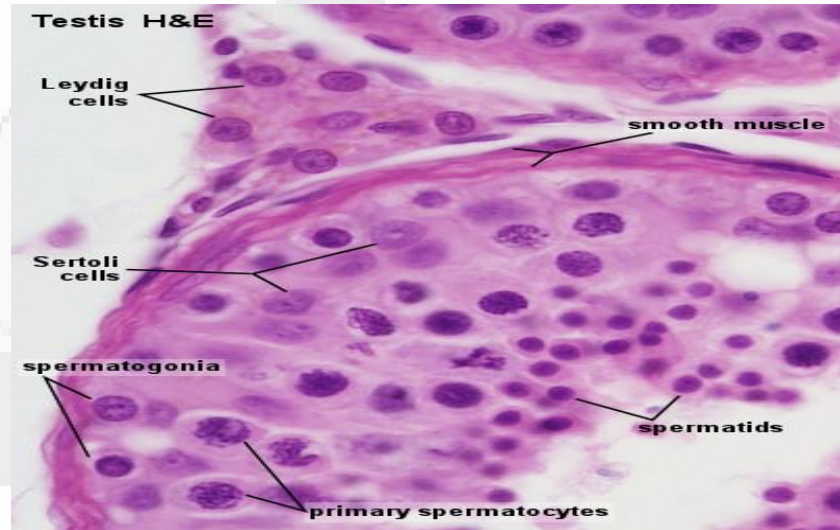
**A.P.Dr.Maather Baqer Hussein
Department of Pathology**



Testis



Here is a normal testis and adjacent structures. Identify the **body of the testis**, **epididymis**, and **spermatic cord**. Note the presence of two vestigial structures, the **appendix testis** and the **appendix epididymis**.

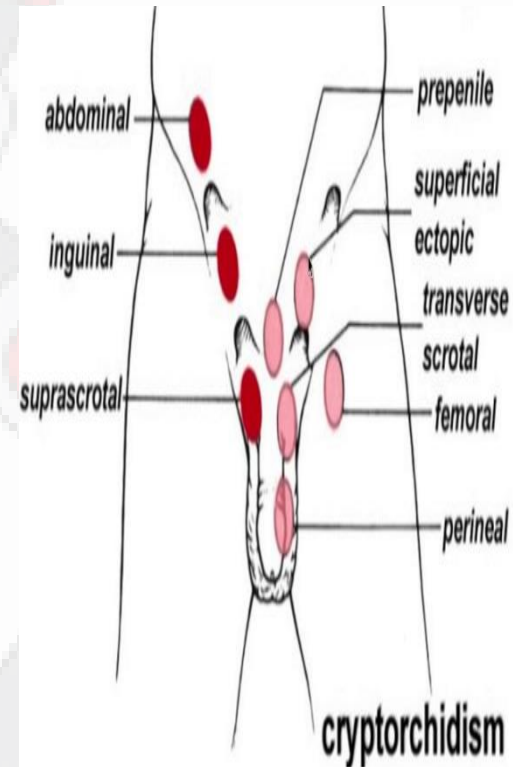


DEVELOPMENTAL DISORDERS

Cryptorchidism:-

- Affects 1% of 1-year-old boys and represents failure of descent; it is usually unilateral and an isolated anomaly, **but it is bilateral in 25% of patients** and can occur with other genitourinary malformations.
- Its incidence is 0.2% in adult male population.

In **70%** of cases, the undescended testis lies in the **inguinal ring**, in **25%** in the **abdomen** and, in the remaining **5%**, it may be present at other sites along its descent from **intra-abdominal location to the scrotal sac**.



Etiology

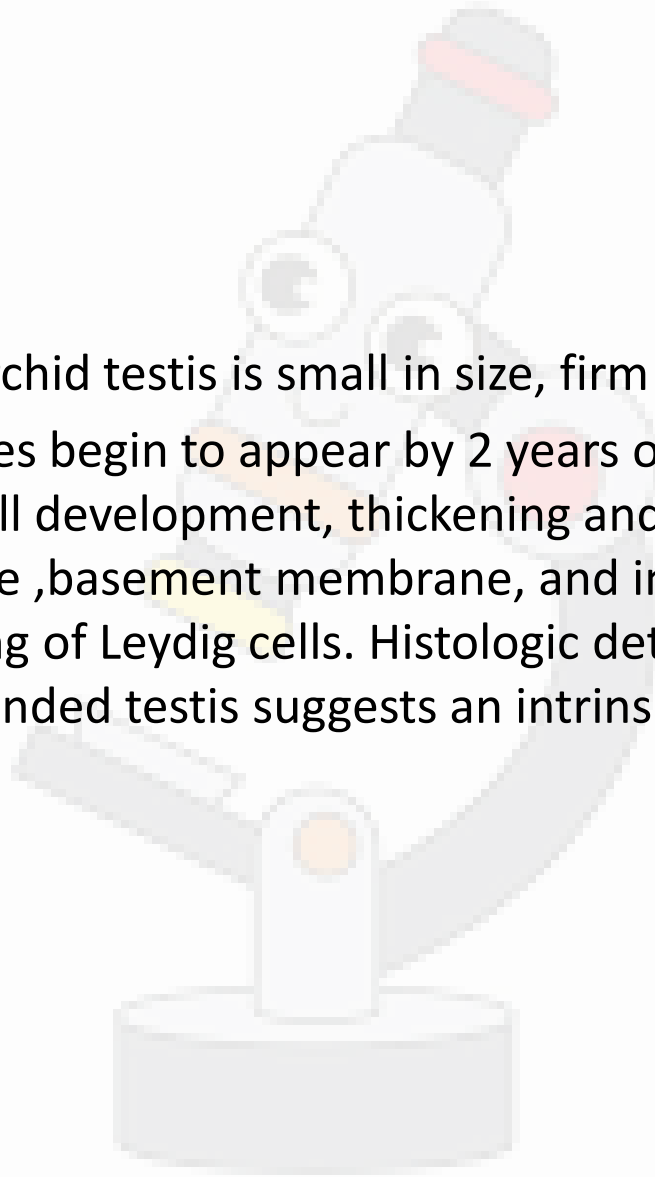
The exact etiology is not known in majority of cases. Following factors are implicated:

- **1. Mechanical factors** e.g. short spermatic cord, narrow inguinal canal, adhesions to the peritoneum.
- **2. Genetic factors** e.g. trisomy 13, maldevelopment of the scrotum or cremaster muscles.
- **3. Hormonal factors** e.g. deficient androgenic secretions.

CLINICAL FEATURES :- As such, cryptorchidism is completely asymptomatic and is discovered only on physical examination. Following significant adverse clinical outcome may result:

- The Male Reproductive System and Prostate
- **1. Sterility-infertility**
- **2. Inguinal hernia**
- **3. Malignancy** Cryptorchidic testis is at **30-50 times increased risk of developing testicular malignancy.**

- **GROSS** The cryptorchid testis is small in size, firm and fibrotic.
- **MICRO** The changes begin to appear by 2 years of age, these include decreased germ cell development, thickening and hyalinization of seminiferous tubule, basement membrane, and interstitial fibrosis along with relative sparing of Leydig cells. Histologic deterioration in the contralateral descended testis suggests an intrinsic defect in testicular development.



Regressive Changes

Atrophy and Decreased Fertility

Atrophy and decreased fertility can be:

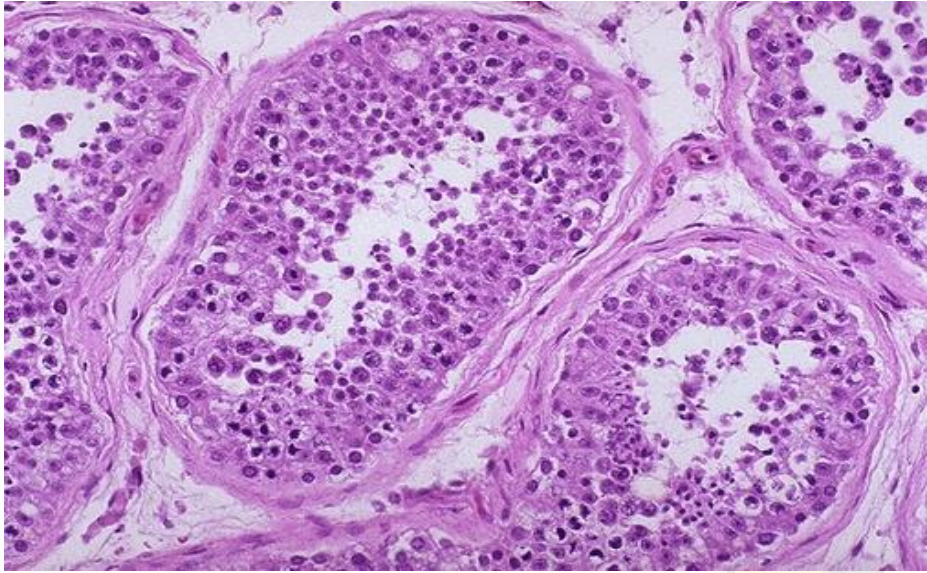
- **Primary**, due to a developmental abnormality (e.g., Klinefelter syndrome).
- **Secondary** to cryptorchidism, vascular disease (e.g., atherosclerosis), inflammatory disorders, hypopituitarism, malnutrition, persistently elevated levels of follicle-stimulating hormone, exogenous androgenic or anti-androgenic hormones, radiation, and chemotherapy.

The morphologic alterations are identical to those seen in cryptorchidism.

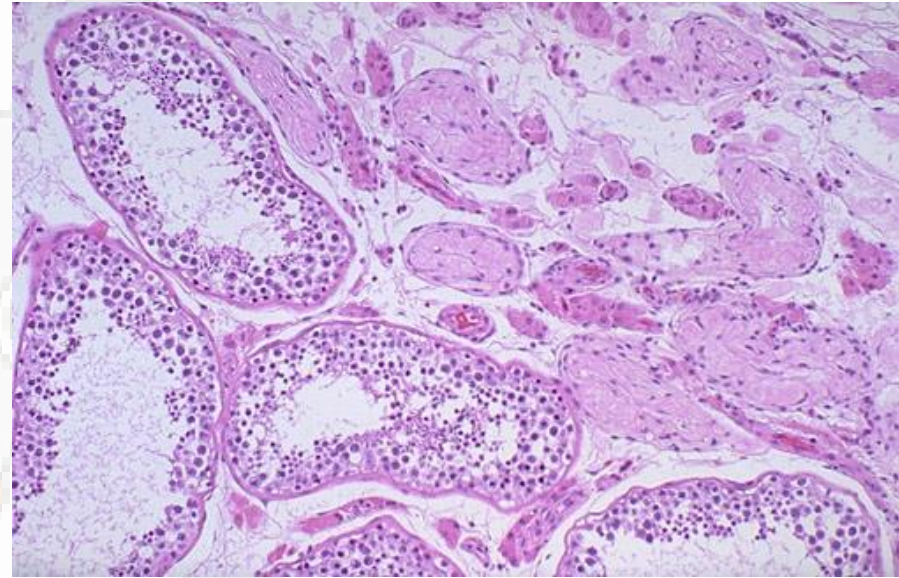
Bilateral atrophy may occur with a variety of conditions including chronic alcoholism, hypopituitarism, atherosclerosis, chemotherapy or radiation, and severe prolonged illness.



On the left is a normal testis. On the right is a testis that has undergone atrophy.



Normal testis. The seminiferous tubules have numerous germ cells. Sertoli cells, with cytoplasm that extends between the germ cells, are inconspicuous. Small dark oblong spermatozoa are seen toward the center of the tubules.

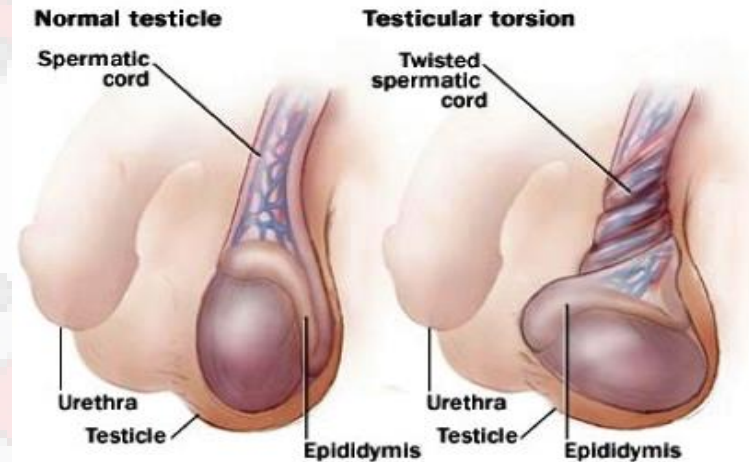


Focal atrophy of tubules seen here to the upper right. The most common reason for this is probably childhood infection with the mumps virus, which produces a patchy orchitis. However, it is unusual for this infection to cause enough atrophy to significantly affect the sperm count.

Vascular Disorders

- **Testicular Torsion** :-

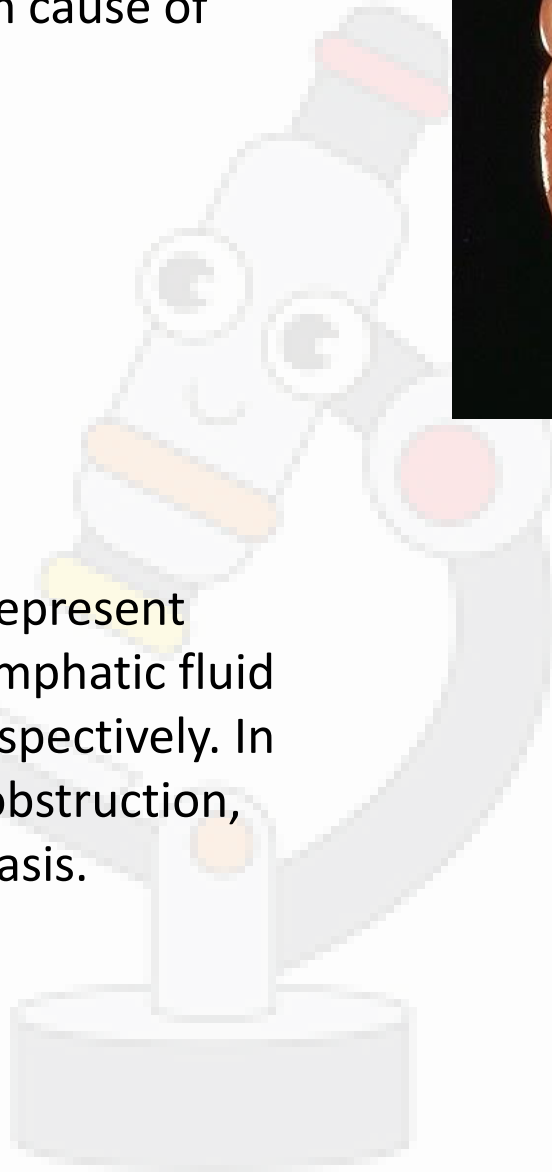
- Is an uncommon condition, but a medical emergency. It occurs when twisting of the spermatic cord cuts off the venous drainage, leading to hemorrhagic infarction.
- Greater mobility from incomplete descent or lack of a scrotal ligament predisposes to this condition.
- Immediate treatment by surgically untwisting and suturing the cord in place to prevent future torsion will prevent infarction.
- Could be occur at any age
- **Micro** There may be coagulative necrosis of the testis and epididymis, or there may be haemorrhagic infarction.



- **Hydrocele** is an accumulation of serous fluid within the tunica vaginalis & represents the most common cause of scrotal enlargement.



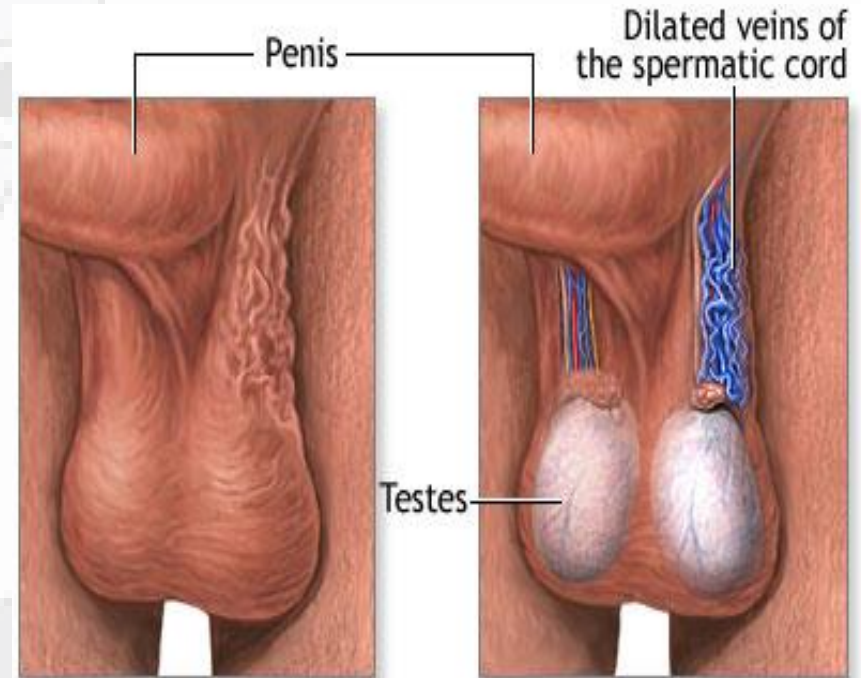
- **Hematocetes & chyloceles**, represent accumulations of blood or lymphatic fluid within the tunica vaginalis respectively. In extreme cases of lymphatic obstruction, caused, for example, by filariasis.



- **VARICOCELE**

Is the dilatation, elongation and tortuosity of the veins of the pampiniform plexus in the spermatic cord. It is of 2 types: primary (idiopathic) and secondary

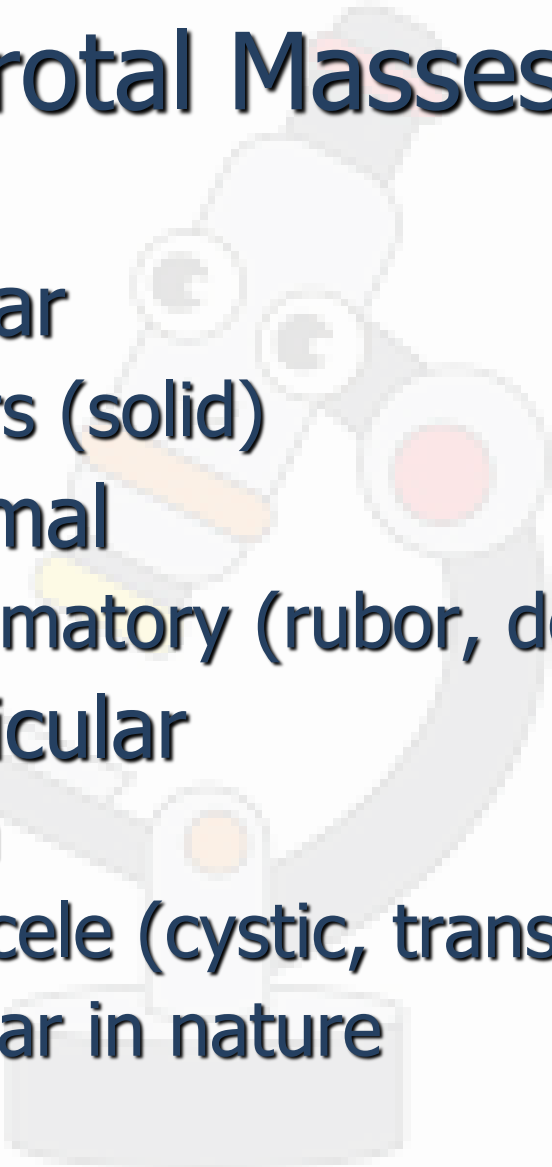
- **Primary or idiopathic form** is more frequent and is more common in young unmarried men. It is nearly always on the left side as the loaded rectum presses the left vein.
- **Secondary form** occurs due to pressure on the spermatic vein by enlarged liver, spleen or kidney.



A varicocele can be felt and sometimes be seen as a tortuous mass on the surface of the scrotum

A varicocele is made up of veins that contain inadequate valves

Scrotal Masses

- Testicular
 - Tumors (solid)
 - Epididymal
 - Inflammatory (rubor, dolor, calor...)
 - Peritesticular
 - Hernia
 - Hydrocele (cystic, trans illuminates)
 - Vascular in nature
- 

TESTICULAR NEOPLASMS

- Testicular neoplasms are the most important cause of **firm, painless enlargement of the testis**.
- The peak age incidence is between the ages of 20 and 34 years.
- Are generally divided into two major categories:
- Germ cell tumors (i.e., 95% of cases) are generally malignant; these are further divided into seminomas and non-seminomas.
- Sex cord stromal tumors are generally benign.

• Germ cell tumors (malignant)

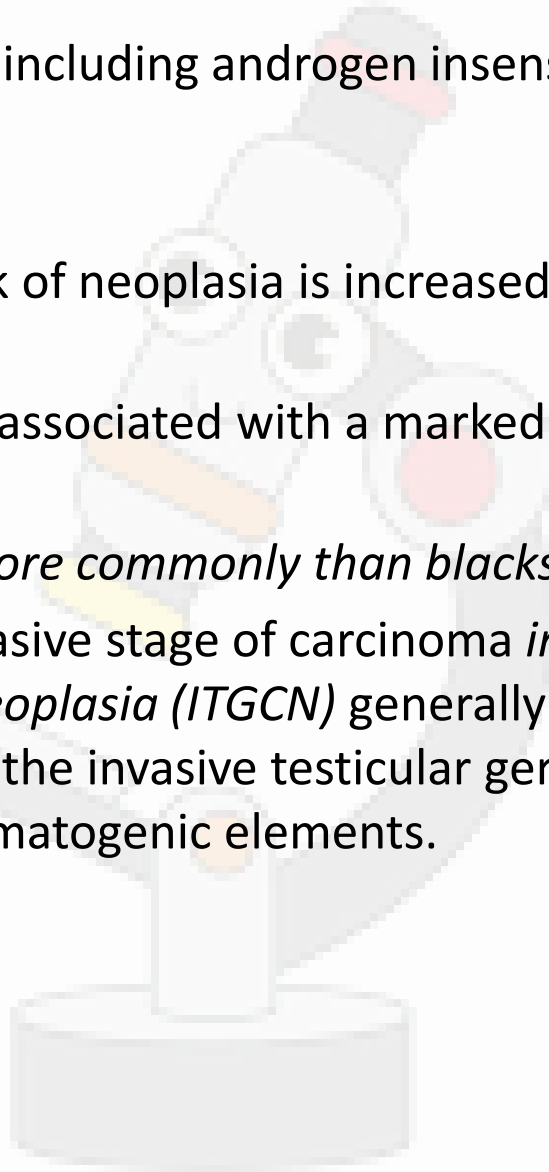
- 1- Seminoma
2. Spermatocytic seminoma
3. Embryonal carcinoma
4. Yolk sac tumour (Syn. endodermal sinus tumour, orchioblastoma, infantile type embryonal carcinoma)
5. Polyembryoma
6. Choriocarcinoma
7. Teratomas (i) Mature (ii) Immature (iii) With malignant transformation
8. Mixed germ cell tumours

Sex cord stromal tumors (benign or malignant)

- Leydig cell orgine , may be hormonally active
 - Sertoli cell tumor (androblastoma)
 - Granulosa cell tumor
 - Mixed Leydig cell & Sertoli cell
-
- **Mixed germ & sex cord cells origin**
 - **Tumors of collecting ducts and rete testis (adenoma, cystadenoma, adenofibroma ,**
 - **Lymphoma**
 - **Paratesticular tumors**
 - **Secondary tumors**

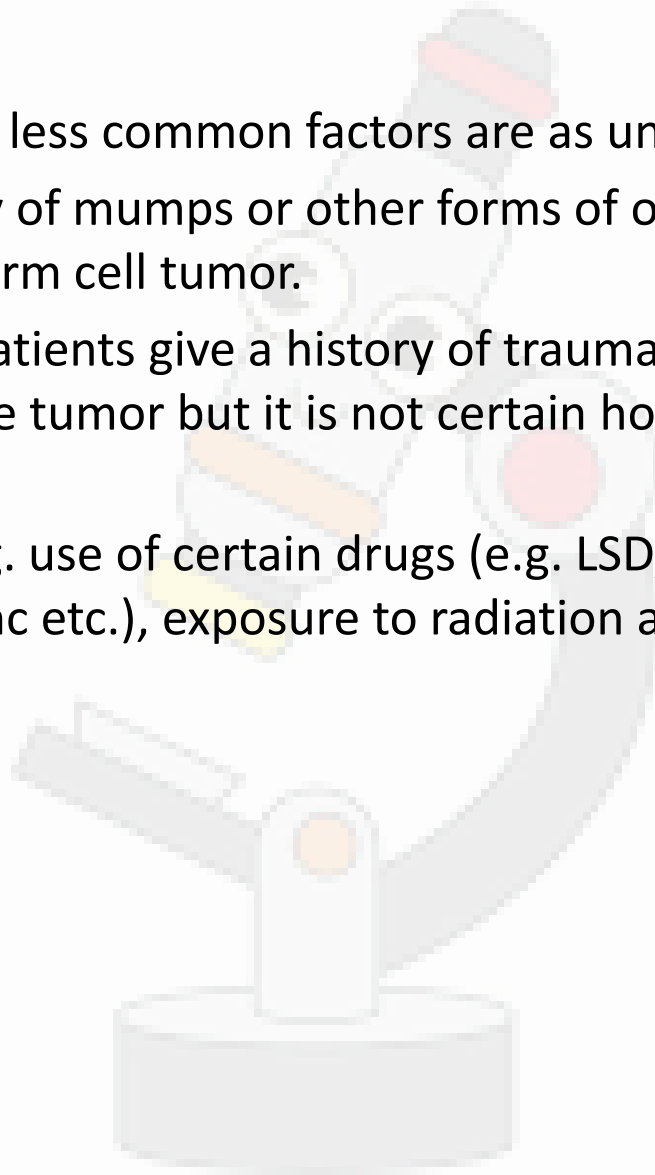
Risk factors:-

- **1. Cryptorchidism.**
- **2. Intersex syndromes** :- including androgen insensitivity syndrome and gonadal dysgenesis.
- **3. Genetic & ethnic as:-**
 - *a. Family history*: the risk of neoplasia is increased in siblings of males with testicular cancers.
 - *b. Cancer in one testis* is associated with a markedly increased risk in the contralateral testis.
 - *c. Whites are affected more commonly than blacks.*
- **4- CIS/ITGCN** :- A preinvasive stage of carcinoma *in situ* (CIS) termed *intratubular germ cell neoplasia (ITGCN)* generally precedes the development of most of the invasive testicular germ cell tumours in adults. CIS originates from spermatogenic elements.



5. Other factors A few less common factors are as under:

- **i) Orchitis** A history of mumps or other forms of orchitis may be given by the patient with germ cell tumor.
- **ii) Trauma** Many patients give a history of trauma prior to the development of the tumor but it is not certain how trauma initiates the neoplastic process.
- **iii) Carcinogens** e.g. use of certain drugs (e.g. LSD, hormonal therapy for sterility, copper, zinc etc.), exposure to radiation and endocrine abnormalities.



Clinical features and diagnosis

- All present as **painless, masses** in the testis.
- The primary may be **occult**, especially pure choriocarcinomas.
- Many cause gynecomastia (after puberty) or precocious puberty (children) according to hormones production ([Sex cord stromal tumors](#)).

The usual presenting clinical symptoms of testicular tumours are gradual gonadal enlargement and a dragging sensation in the testis.

- Tumor Markers
- Some tumors produce agents measurable in the blood.
 - Beta-HCG
 - Placental marker
 - We measure this in pregnancy tests
 - Alpha-feto protein
 - Marker associated with embryonic gut

SPREAD Testicular tumours may spread by both lymphatic and haematogenous routes:

- 1. *Lymphatic spread* occurs to retroperitoneal para-aortic lymph nodes, mediastinal lymph nodes and supraclavicular lymph nodes.
- 2. *Haematogenous spread* primarily occurs to the lungs, liver, brain and bones.

Testicular cancer are staged as follows:

Stage I: tumour confined to the testis.

Stage II: distant spread confined to retroperitoneal lymph nodes below the diaphragm.

Stage III: distant metastases beyond the retroperitoneal lymph nodes



**THANK YOU
FOR YOUR
ATTENTION**