429 Pathology Team

Made by:
Sarah Mahasin
Dona Barakah

Notes by:
Nourhan Al-Shamma'
Nujud Al-Hejin

Edited by:
Dona Barakah
Muhammad Al-Harbi

# *** Male Reproductive System Pathology ***

## Testicular & Prostate

**Highlighted ; BASIC**
**Highlighted; EXTREMELY IMPORTANT NOTE**
**Highlighted; MCQ**
**Highlghted ; understand only**

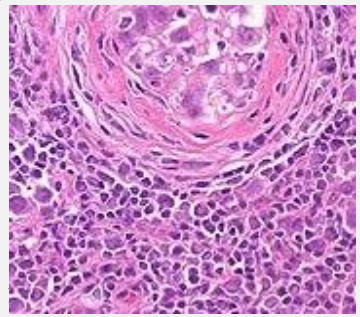
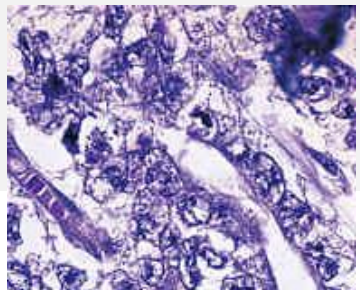
# Testicular Pathology:

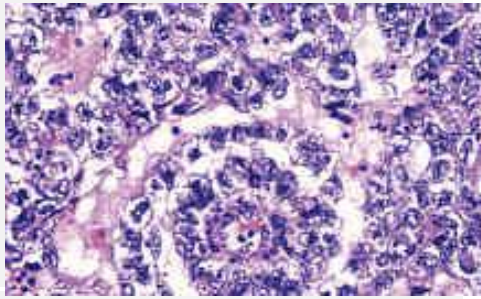
## A. Testicular Inflammation: ((Orchitis; inflammation of Testis))

| | Epididymitis and Orchitis (Non specific) | Granulomatous Orchitis (Autoimmune) | Specific Inflammations: (Gonorrhea OR Tuberculosis) |
|---|---|---|---|
| **Epidemiology** | **BEGIN AS:** Primary urinary tract infection (cystitis, urethritis, genitoprostatitis).<br>Then reach the epididymis/testis **Through:**<br>-Vas deference<br>-The lymphatics of the spermatic cord.<br><br>**CAUSES:**<br>a) **Children:**<br>Uncommon<br>**Associated with** a congenital genitourinary abnormality<br>**infection with** Gram –ve rods.<br>b) **Sexually active men younger than 35 years:**<br>Chlamydia trachomatis and Neisseria<br>c) **Older than 35 Years:**<br>E.Coli and Pseudomonos. | - In **middle –aged men**<br>- Present as Unilateral Testicular mass.<br>- Usually moderately tender (**painful**) but sometimes may present as **painless** testicular mass; mimicking a testicular tumor.<br>- Suspected to be<br>1) Autoimmune<br>2) A response to acid-fast products of disintegrated sperm<br>3) Post-infection<br>4) Trauma or<br>5) Sarcoidosis (inflammation to lymph nodes) | **GONORRHEA:**<br>Course of a neglected gonococcal infection: Extension of infection from the posterior urethra to the prostate to the seminal vesicles and then to the epididymis .<br><br>**TUBERCULOSIS:**<br>-Begin with **Epididymus** → spread to **Testis.**<br>-Usually Primary **associated with**<br>Tuberculous Prostatitis<br>Seminal Vesiculitis |
| **Morphology:** Microscopic Findings | - **Bacterial Invasion**<br>- **Non Specific Acute Inflammation** Characterized by: congestion, edema and infiltration by neutrophils, macrophages and lymphocytes.<br><br>**Starts at** → interstitial connective tissue<br>**Later involves** → tubules<br>**Progress to** → frank abscess → fibrous scarring<br>*Leydig cells are not usually destroyed* | **Granulomas**<br>((resticted within the spermatic tubules)). | **GONORRHEA:**<br>- Can lead to frank abscess<br>- May spread to testis<br>- Can produce a Supurative Orchitis.<br><br>**TUBERCULOSIS:**<br>Caseating Granulomatous Inflammation. |

B. **Testicular Tumors:** 95% of malignant ones originate from Germ-Cell. Non-germal ones are generally benign.

1) Germ Cell Tumors:

| | Epidemiology | Gross Morphology | Microscopic Morphology |
|---|---|---|---|
| **1. Seminoma (dysgerminoma in ovaries)** | - **Most common** type of germ cell tumors 50%<br>- Peak incidence in 30s, almost NEVER occur in infants<br>if there was a patient whom is 2 yr old, **it WONT be seminoma** but more probably **yolk sac tumor** or **Teratoma.** (read further next)<br>- Histologically identical to **Dysgerminoma** (name of same tumor occurring in Ovaries) | **The Classic or Typical Seminoma:**<br>- Bulky masses<br>- Homogenous, gray-white, lobulated cut surface<br>- No necrosis or hemorrhage<br>- **In 50%,** the entire testis is involved<br>- **Occasionally** extends to the epididymis, spermatic cord, or scrotal sac | **The Classic or Typical Seminoma:**<br>Sheets of uniform **cells** in lobules separated by delicate fibrous septa.<br>  <br>((Those cells are large, membrane, large nucleus with prominent nucleoli))<br>**Positive for PLAP** in histology & serum |
| **2.Spermatocytic Seminoma** | - Less common variant of Seminoma, but with excellent prognosis<br>- Usually in persons over 65 years of age | - Slow growing tumor<br>- Rarely metastasis | |
| **3. Embryonal CARCINOMA** | - Usually in 20-30 year age group<br>- **MORE AGGRESSIVE** than Seminomas | **Smaller than Seminoma, and are:**<br>- Ill defined, **Invasive Masses**<br>- Containing foci of necrosis & hemorrhage | - Cells grow in **alveolar** or **tubular pattern** as papillary convolutions<br>- Could be present with other neoplasms in 45% of it.<br>  |

| | | | |
|---|---|---|---|
| **4. Yolk Sac Tumor (Endodermal Sinus Tumor)** | - The most common tumor in infant and children up to 3 years of age.<br>- Has a very good prognosis<br>A patient whom is **AFP(+) + 2 yr old**<br>**→ yolk sac tumor** | Non encapsulated, homogenous, mucinous appearance | **Structures resemble endodermal sinuses:**<br>- Schiller-Duval bodies<br>- Hyaline pink globules<br>- AFP positive<br>  |
| **5.Chorioarcinoma** | - Highly malignant tumor<br>- HCG (Human Chorionic Gonadal Test) positive | Small lesions | **Cytotrophoblastic** & **Syncytiotroblastic** cells |
| **6. Teratoma** | - **Any age**, infancy to adult life<br>- **Mature forms** → common in infants and children.<br>→Secondary to yolk sac tumor<br>→ behave as benign<br>- **In post pubertal male**<br>→ All are malignant<br>→All capable of metastasis<br>→Elements are either mature or not | - Various cellular or organoid components<br>- Usually large (5 -10 cm)<br>- Heterogenous appearance<br>- Hemorrhage and necrosis indicate embryonal component<br>- Other Germ cell tumors could arise from teratoma | **Composed of heterogeneous (different) collection of cells or organoid structures like:**<br>- Neural Tissue<br>- Cartilage<br>- Squamous Epithelium<br>- Glandular Components |

## 2) Sex-cord Tumors:

- Leydig-cell Tumor
- Steroli-cell Tumor

**<u>Testicular Tumors Predisposing Factors:</u>**
**- Genetic Factors**
**- Cryptorchidism** (un-descendent testis) as 10% of them
**- Testicular Dysgenesis** (abnormal organ development)
**-** More common in White people than in Black people.

**General Notes on Testicular tumors :**

- Present as an enlargement of the testis.
- Biopsy of a testicular tumor is associated with a risk of tumor spillage therefore it is not recommended.
- The standard management of solid tumors is radical orchiectomy
- Lymphatic spread is common
- Retroperitoneal and para-aortic nodes are first to be involved
- Hematogenous spread to lung, liver, brain, and bones.

The closer is the **male** to puberty → the higher is th chance of malignancy
The younger is the **female** → the lesser is the chance of malignancy

## Table 18-2. Summary of Testicular Tumors

| Tumor | Peak Age (yr) | Morphology | Tumor Markers |
|---|---|---|---|
| Seminoma | 40-50 | Sheets of uniform polygonal cells with cleared cytoplasm; lymphocytes in the stroma | 10% have elevated hCG |
| Embryonal carcinoma | 20-30 | Poorly differentiated, pleomorphic cells in cords, sheets, or papillary formation; most contain some yolk sac and choriocarcinoma cells | 90% have elevated hCG or AFP or both |
| Yolk sac tumor | 3 | Poorly differentiated endothelium-like, cuboidal, or columnar cells | 90% have elevated AFP |
| Choriocarcinoma (pure) | 20-30 | Cytotrophoblast and syncytiotrophoblast without villus formation | 100% have elevated hCG |
| Teratoma | All ages | Tissues from all three germ-cell layers with varying degrees of differentiation | 50% have elevated hCG or AFP or both |
| Mixed tumor | 15-30 | Variable, depending on mixture; commonly teratoma and embryonal carcinoma | 90% have elevated hCG and AFP |

# Prostatic Pathology:

| | Benign Prostatic Hyperplasia (BPH) | Malignant (Adenocarcinoma)<br>(Prostate is a gland !!) |
|---|---|---|
| Pathogenesis | **OCCURANCE:** **Common** in men **over 50** (prevented in males who made a "pre-pubertal castration" – removal of testicles before puberty)<br>(20% in over 40, 40% in over 60 and 90% in over 70)<br>**CAUSE:** Related to **Androgens** action and to **DHT**<br>(**D**i**H**ydro**T**estosterone; the ultimate mediator for prostatic growth) | **Most common** cancer in **men** and the 2nd leading cause of cancer death.<br>**OCCURANCE:** men **over 50** (especially blacks in USA). **Affected by:**<br>Age, race, family history, hormone level and environmental influences.<br>**CAUSE:** related to **Androgens**. |
| Morphology | - Almost usually in the **Inner Aspect of the Prostate.**<br>- Heavy prostate.<br>- **Nodularity** is a hallmark (nodules vary in color & consistency), **due to:** Glandular proliferation/dilatation also fibrous and muscular proliferation → **Aggregation of small-to-large cystically dilated glands.** (important in OSPE)<br>- Needle biopsy through rectal doesn't reach the peri-urethral zone<br>- Adult presents with **urgency** as it causes narrowing of the urethra (important is OSPE) | - Almost usually in the **Peripheral Aspect of the Prostate** (Palpated in rectal exam).<br>- Gritty and firm prostate<br>- **WELL-DEFINED gland pattern glands surrounded by malignant cells)**<br>- Histologic diagnosis is challenging. But we can use rectal biopsy here<br>60 yr old, **back pain** (rectal side) + metastatic lesion in urethra → prostatic carcinoma<br>- Spread by:<br>1)**Local extension** → most commonly involves seminal vesicles and base of Urinary Bladder<br>2)**Hematogenous (through bloodstream) extension** → occurs mainly to bones<br>3)**Lymph extension**<br>4) **Perineural invasion** (growth of tumor along the nerve) is common and typical. |

**Grading and staging of ADENOCARCINOMA:** by **Gleason grading system** based on **degree of differentiation:**
Higher grade → Higher malignancy → Worse prognosis

**Clinical Course of ADENOCARCINOMA:**

- Microscopic ones are Asymptomatic (discovered incidently)
- If it is clinically localized, it doesn't have urinary symptoms unlike benign ones (it arises peripherally faraway from urethra).
- **PSA** (prostate specific antigen) is used for **diagnosis and management** ONLY. HIGHER in **malignancy** than in **hyperplasia** (useful in FOLLOW-UP ONLY)

**Treatment of ADENOCARCINOMA:**

- Clinically localized cancer → treated by **Radical Surgery**
- Too locally advanced cancer → treated by **Radiotherapy**
- To induce ***Remission*** → use **Hormonal Therapy** (Anti-androgen Therapy)