G.U.S.

Pathology

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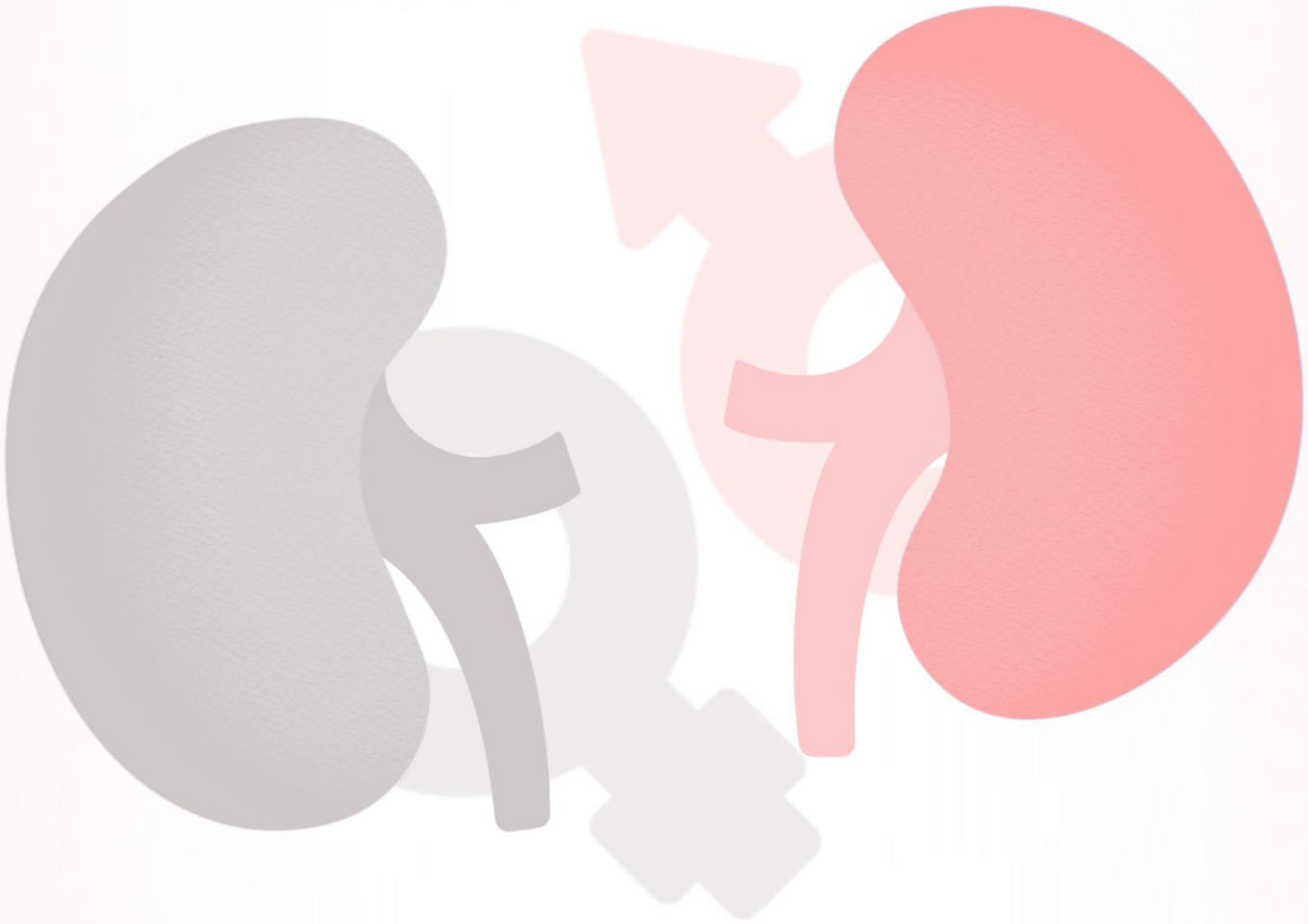

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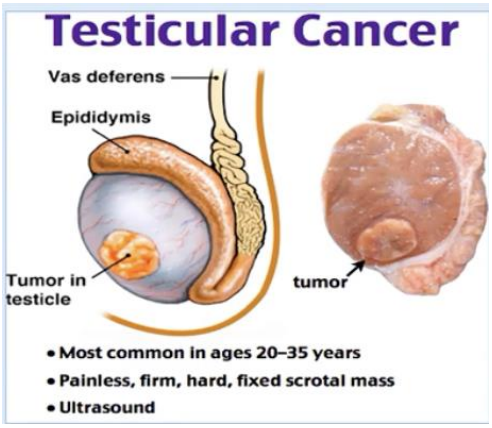
Writer: Reham badayneh

S.corrector: Lina abdelhadi

F.corrector: Ibrahim Elhaj

Doctor: Nisreen Abu Shahin

Testicular Neoplasms



- Peak incidence at 15-34 yr.

- Most common tumors in men (15-34 yr).

- 10% of cancer deaths.

- Include:

I. Germ cell tumors: (95%); all are malignant in postpubertal males.

II. Sex cord-stromal tumors: less common, generally benign.

- Risk Factors:

1. More common in white people (**whites > blacks**).

2. **Cryptorchidism**: Risk of cancer in undescended testis, and even contralateral descended testis.

3. **Intersex syndromes** (Androgen insensitivity syndrome; Gonadal dysgenesis).

4. **Family history**: (4 to 10 X in their fathers and brothers of affected men).

5. **cancer in one testis** (↑risk of cancer in contralateral testis).

6. **isochromosome of short arm of chromosome 12**, i(12p): (in virtually all germ cell tumors, regardless of their histologic type).

7. **intratubular germ cell neoplasia** (in situ lesion): Most testicular tumors in postpubertal males arise from it.

- Testicular germ cell tumors are sub-classified into: -

I. Seminomas.

II. Non-seminomatous germ cell tumors (NSGCT).

Includes several variants: -

1-Embryonal carcinoma

2-Yolk sac tumors

3-Choriocarcinoma

4-Teratoma

Note:

- In any individual case the **histologic appearances** may be:

1. Pure (i.e., composed of a single histologic type, 40% of cases).

2. Mixed (composed of more than one histologic type, 60% of cases).

I. Seminomas

Make up to 50% of all testicular tumors.

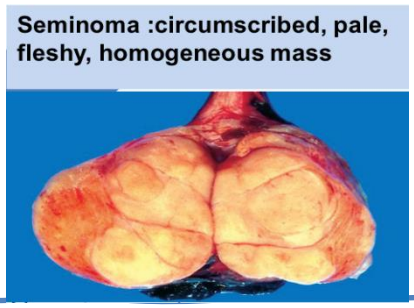
Classic seminoma:

-40-50 years old, rare in prepubertal children, painless enlargement of testis

-Histologically identical to *ovarian dysgerminomas* and to *germinomas* occurring in the CNS and other extragonadal sites.

Morphology (Grossly):

soft, well-demarcated tumors, usually without hemorrhage or necrosis.

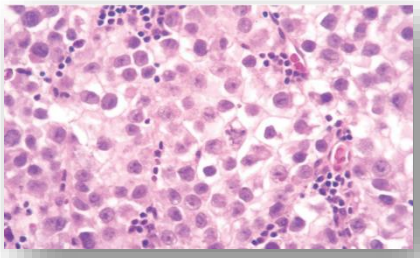


Morphology (Histologically):

-large, uniform cells with distinct cell borders, clear, glycogen-rich cytoplasm, round large nuclei, and 1-2 conspicuous nucleoli.

-The cells arrayed in small lobules with intervening delicate fibrous septa.

-A lymphocytic infiltrate is usually present.



II. Non-seminomatous germ cell tumors

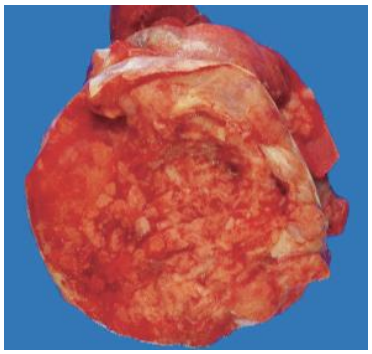
a. *Embryonal carcinomas*

20-30 years old.

More aggressive than seminoma.

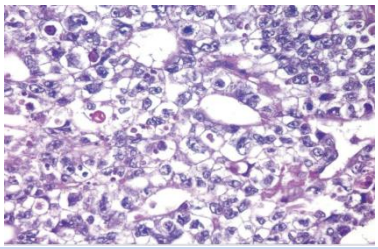
Morphology (Grossly):

- ill-defined masses containing foci of hemorrhage and necrosis



Morphology (Histologically):

-large and primitive-looking tumor cells; basophilic cytoplasm, indistinct cell borders, large nuclei, prominent nucleoli, pleomorphic, and increased mitotic activity



Sheets of undifferentiated cells & primitive gland -like structures. The nuclei are large and hyperchromatic

b. *Yolk sac tumors*

-Most **common** primary testicular neoplasm in children <3 yr., good prognosis in kids.

-In adults: rare and worse prognosis

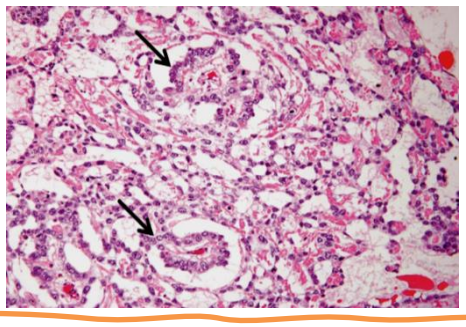
- **AFP can also be detected in the serum.**

Morphology (Grossly):

-large and may be well demarcated.

Morphology (Histologically):

- A distinctive feature is the presence of structures resembling primitive glomeruli, called Schiller-Duvall bodies **(black arrows) VIP.**



c. *Choriocarcinoma*

-20-30 years old, highly malignant, rare <1% of all germ cell tumors, can also arise in the female genital tract , ↑ serum level of HCG.

-It can metastasize early specially to the CNS and the lungs, and the prognosis of this type of cancer is bad.

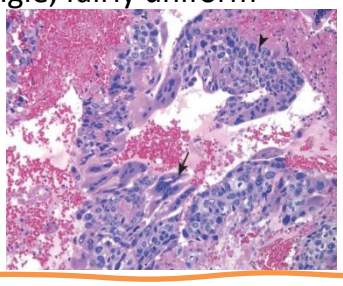
Morphology (Grossly):

necrosis and hemorrhage are extremely common.

Morphology (Histologically):

- 1-**Syncytiotrophoblasts**: large multinucleated cells; containing HCG(black arrow).

2- **Cytotrophoblasts**: single, fairly uniform nucleus(arrow head).



d. Teratomas

-Neoplastic germ cells differentiate along somatic cell lines.

-Reminiscent of the normal derivatives of more than one germ layer (Ectoderm , Mesoderm , Endoderm).

- All ages, common in infants and children; 2nd most common after (yolk sac tumors) .

-In adults: pure is rare (3%). However, the frequency of mixed teratomas with other germ cell tumors ≈ 45%

-In prepubertal males, mature teratomas usually follow a benign course.

-In postpubertal males, all teratomas are malignant, being capable of metastasis regardless of whether they are composed of mature or immature elements.

Morphology (Grossly):

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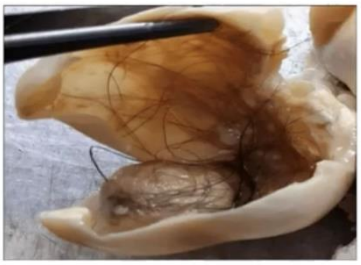
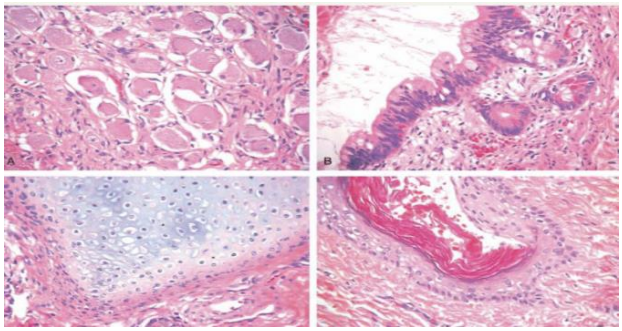


Morphology (Histologically):

- Under the microscope we can see different types of mature tissues.



Clinical Features of testicular germ cell neoplasms:

1-Present with painless testicular mass.

1-Some tumors, especially NSGCT, may have metastasized widely by time of diagnosis.

3-Biopsy of a testicular neoplasm is contraindicated, because it is associated with a risk of tumor spillage.

4-The standard management of a solid testicular mass is radical orchiectomy, based on the presumption of malignancy.

Seminomas and nonseminomatous tumors differ in their behavior and clinical course:

I. Seminomas:

Remain confined to the testis for long periods.

Metastases to iliac and paraaortic lymph nodes .

Hematogenous metastases occur late.

II. Nonseminomatous germ cell neoplasms:

-metastasize earlier, by lymphatic & hematogenous routes (liver and lung mainly).

Assay of tumor markers secreted by germ cell tumors:

-helpful in diagnosis and follow up:

1-HCG is always elevated in choriocarcinoma.

2-AFP (Alpha FetoProtein) is increased in yolk sac tumor.

3-lactate dehydrogenase (LDH) level correlate with tumor burden (tumor size or load), regardless of type.

TREATMENT:

Seminoma:

-extremely radiosensitive

-tends to remain localized for long periods

-best prognosis.

- >95% of patients with early-stage disease can be cured.

Nonseminomatous germ cell tumors:

-Aggressive tumors; chemotherapy.

-choriocarcinoma, which is associated with a poorer prognosis.

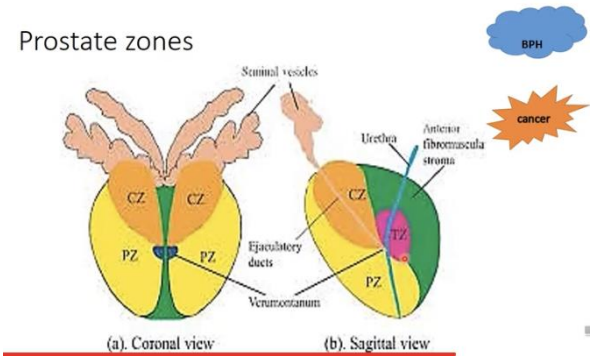
Prostate Gland pathology

Prostate zones:-

1-Central Zone (CZ)

2-Peripheral Zone (PZ)

3- Transitional Zone (TZ)



Benign prostatic hyperplasia (BPH) arises in the transitional zone and part of the central zone of the prostate, whereas Carcinoma of the prostate arises in the peripheral zone. This is very important regarding the symptoms of these conditions; BPH is very close to the urethra so it will have urinary symptoms while carcinoma of the prostate arises in the PZ of the prostate; the development of urinary symptoms will be a late manifestation.

1- Benign Prostatic Hyperplasia (Nodular Hyperplasia):

Extremely common in men >40; frequency rises with age.

Androgen-dependent proliferation of both stromal and epithelial elements ,does not occur in in males with genetic diseases that block androgen activity.

Pathogenesis: Dihydrotestosterone (DHT) is synthesized in the prostate from circulating testosterone by 5α-reductase, type 2.

DHT —> support growth and survival of prostatic epithelium and stromal cells by binding to androgen receptors.

DHT is 10 times more potent.

Morphology:

BPH always occurs in inner transition zone of prostate.

Grossly:

-Prostatic enlargement by many well circumscribed nodules bulging from the cut surface.

-Compressed urethra.

Microscopically:

-Composed of proliferating glands and fibromuscular stroma.

-The hyperplastic glands are lined by 2 cell layers: tall, columnar epithelial cells and a peripheral layer of flattened basal cells.

Clinical features:

Because BPH preferentially involves the inner portions of the prostate, the most common manifestations are :

1-lower urinary tract obstruction .

2-Difficulty in starting stream of urine (hesitancy).

3-Intermittent interruption of urinary stream .

4-Urinary urgency, frequency, and nocturia (bladder irritation).

↑ risk of urinary tract infections

2-Carcinoma of the prostates

The most common form of cancer in men > 40.

↓prostate cancer mortality, due to increased early detection through screening.

PATHOGENESIS:

1. Androgens:

Prostate cancer does not develop in males castrated before puberty.

Cancers regress in response to surgical or chemical castration

2. Heredity:

↑risk among first-degree relatives of patients with prostate cancer.

3. Environment:

Geographical variations, diet: westernized dietary habits.

4. Acquired somatic mutations

The most common gene rearrangements in prostate cancer => fusion genes consisting of the androgen regulated promoter of the TMPRSS2 gene and the coding sequence of ETS family transcription factor. => TMPRSS2-ETS fusion genes

Clinical Features

- 70% - 80% arise in peripheral glands —>palpable as irregular hard nodules on digital rectal examination.

- elevated serum prostate-specific antigen (PSA) level screening tests.

- Bone metastases (axial skeleton) —> osteoblastic (bone-producing) lesions on bone scans.

Good luck