MALE GENITAL SYSTEM

22 Male Reproductive System

I. Overview

II. Testes

III. Epididymis

IV. Ductus (Vas) Deferens

V. Accessory Glands

VI. Penis

22 Male Reproductive System

I. Overview

A. Testis (22.1)

B. Epididymis

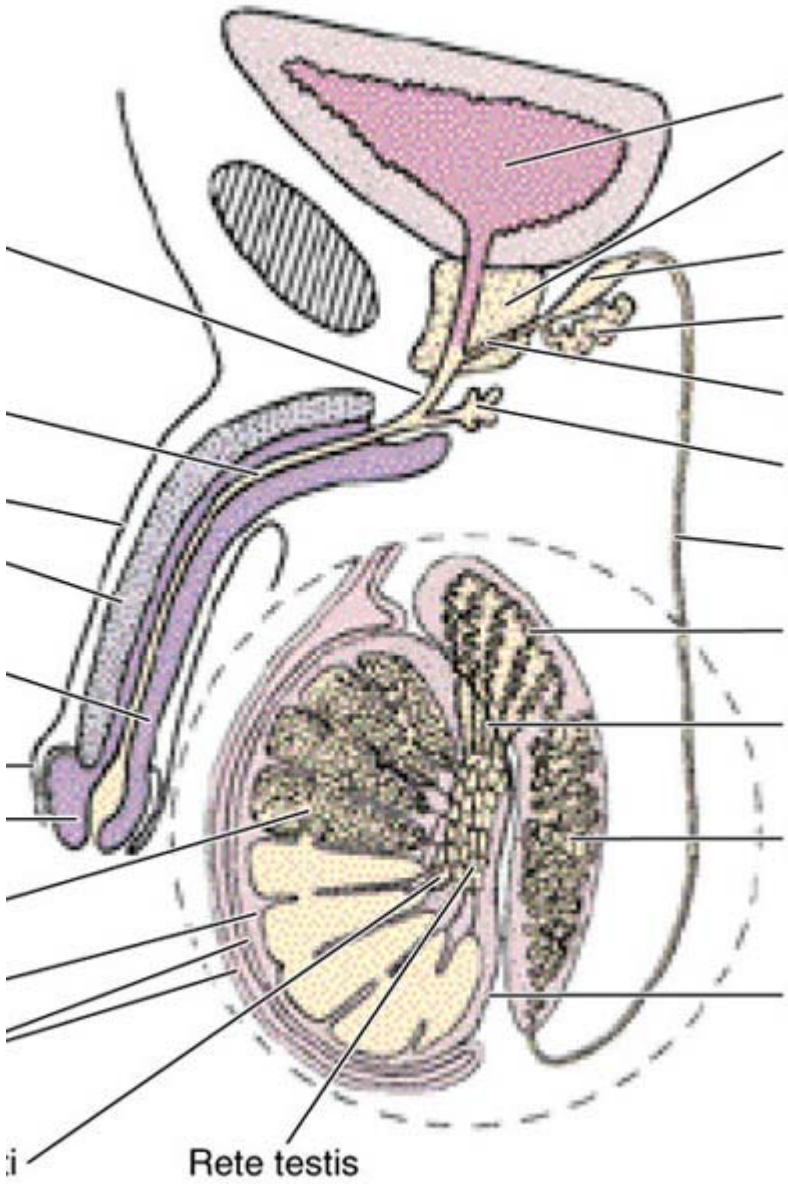
C. Ductus deferens

D. Urethra

E. Penis

F. Seminal Vesicle

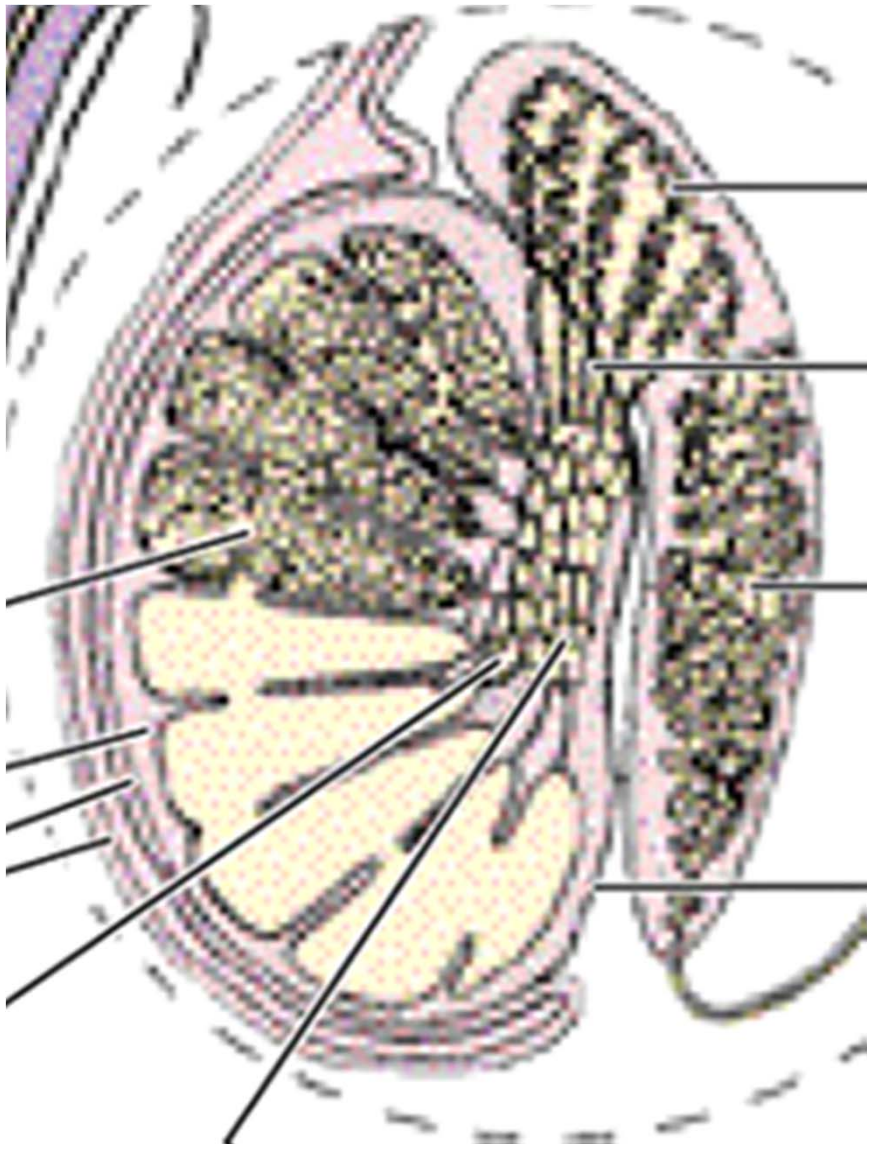
G. Prostate



II. Testis

B. Parenchyma (22.1)

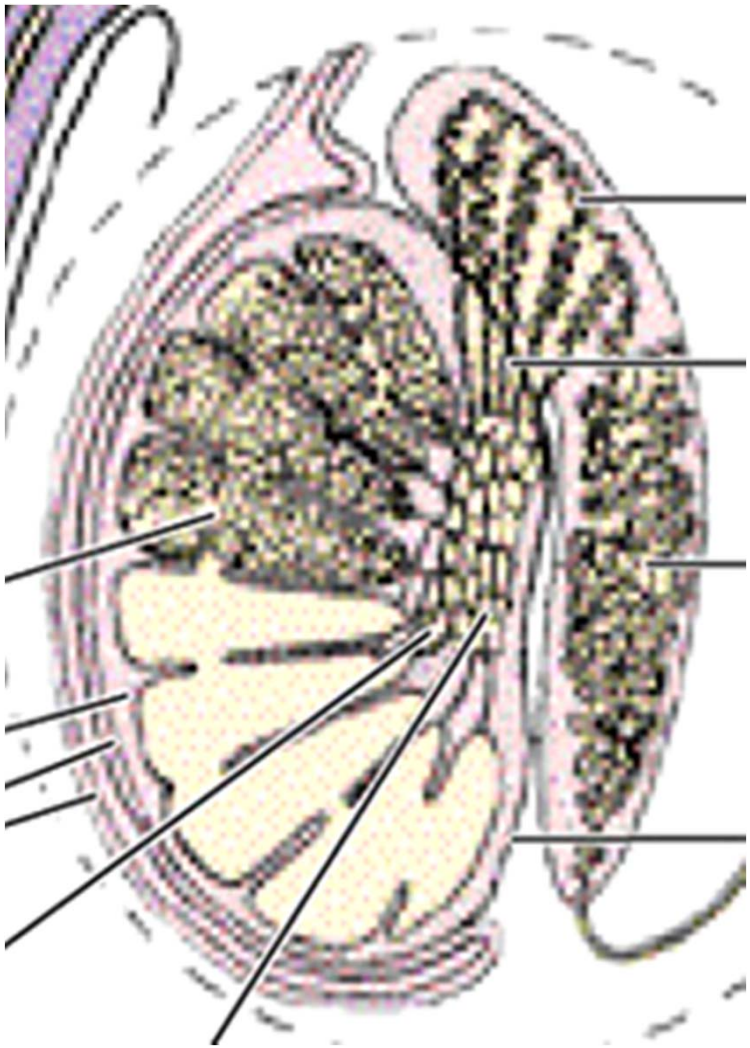
1. Seminiferous tubules
2. Straight tubules
3. Rete testis
4. Ductuli efferentes



II. Testis

A. Stroma (22.1)

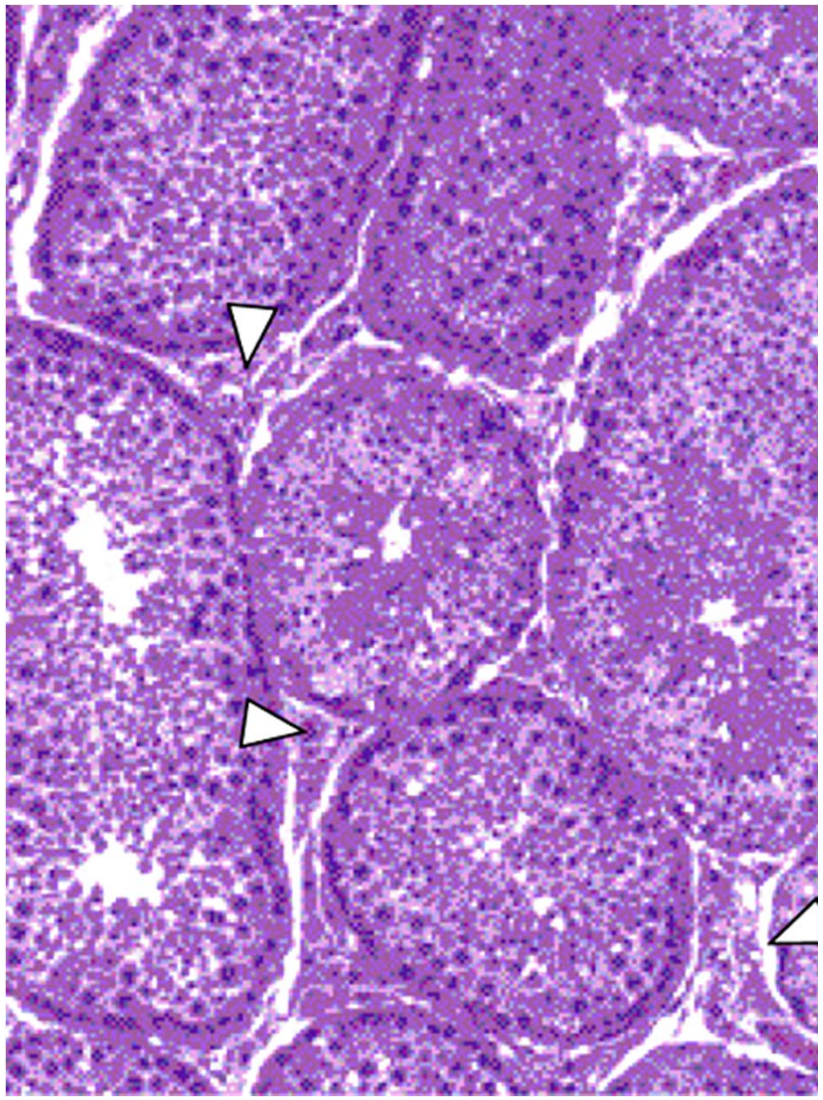
1. tunica albuginea
2. tunica vaginalis
3. mediastinum testis
4. septa
5. testicular lobules



II. Testis

C. Seminiferous tubules (22.2)

1. ~ 250 m / testis
2. stratified (germinal) epithelium
   a. spermatogenic cells
   b. Sertoli cells
3. basal lamina with myoid cells
4. ~ 150-250 μm diam.
5. CT layer



Lesions of testis

- CONGENITAL
- INFLAMMATORY
- NEOPLASTIC

Scrotal disorders

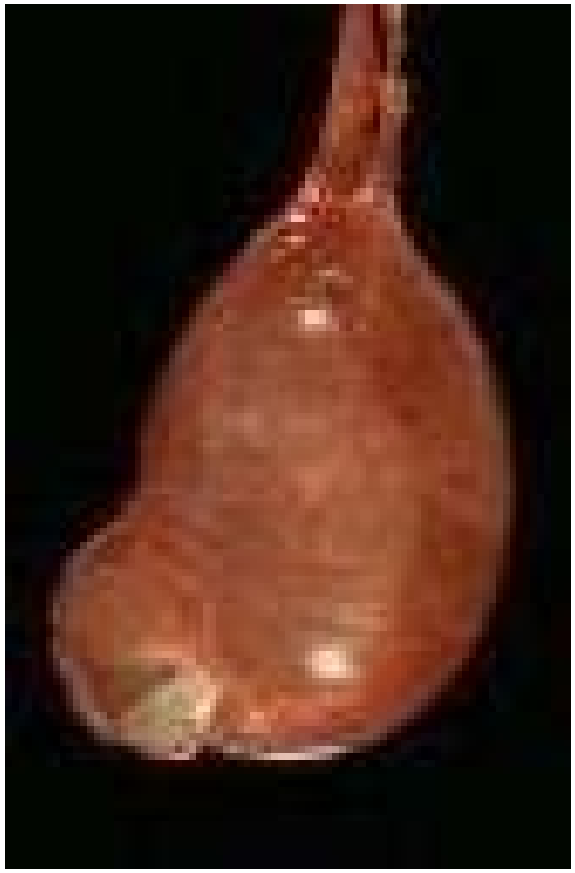
- Hydrocele

  -most common cause of scrotal enlargement

  -soft painless swelling of the scrotum

  -serous fluid in the tunica vaginalis

  -Trauma, inflammation, tumor



Scrotal disorders

- Hematocele
  - blood in the tunica vaginalis
  - results from trauma in most cases
  - surgical removal may be required if there is large accumulation.
- Chylocele
  - accumulation of lymph fluid in tunica vaginalis
  - may be caused by filariasis.

Testis & Epididymis

Cryptorchidism – undescended testis

- Arrest in the descent of testis at some point
- .8% of live male births
- More common on right side
- Unilateral in majority but 25% cases –B/L
- 70% cases testis lies in the inguinal ring
  25% in the abdomen and 5% in the remaining
- ETIOLOGY – Mechanical, genetic and
  hormonal factors

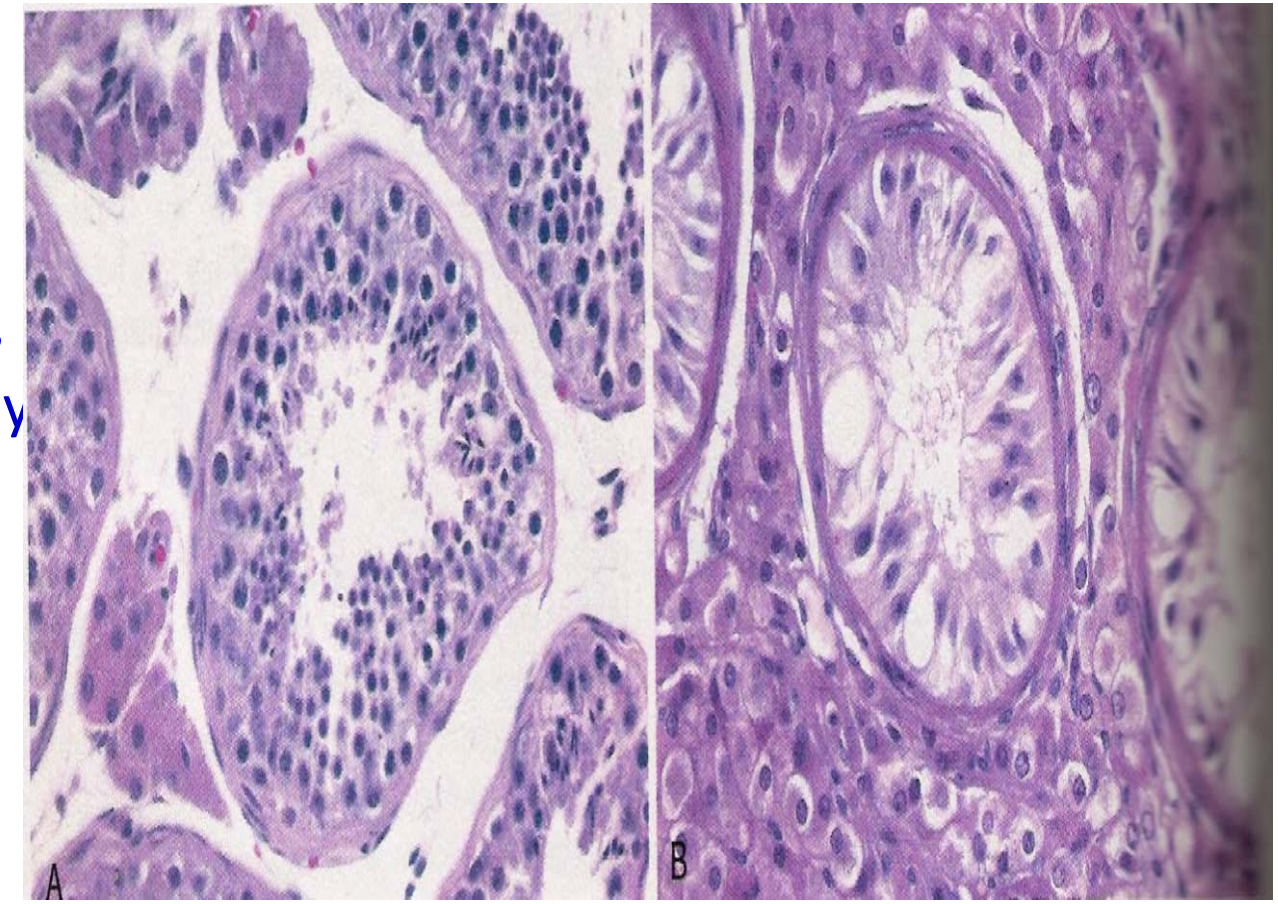
Cryptorchidism

- **Gross** – cryptorchid testis is small in size,firm and fibrotic.

- **M/E**- progressive loss of germ cell elements

  - tubular atrophy and leydig cell hyperplasia

  –marked hyalinisation and thickening of the basement membrane of the spermatic tubules.



Cryptorchidism

- Sterility/infertility – lower scrotal temperature necessary for spermatogenesis.
- Malignancy – 35 times increased risk

  most commonly –seminoma
- Risk in intra-abdominal testis > testis in inguinal canal
- Other causes of testicular atrophy –trauma, radiation, chemotherapy and cirrhosis of liver.

Male infertility

- Failure to conceive after one year of regular coitus without contraception.
- Causes in males –

  Pretesticular – hypopituitarism. Estrogen excess.

  Testicular – agonadism, atrophy, germ cell aplasia, maturation arrest.

  Post testicular- B/L obstruction, infections (gonococcal, chlamydia), immotile cilia syndrome

Assessment of testis

- Semen examination – normal volume – 3-4 ml
  normal count -
  80% morphologically normal and motile.
- USG, CT scan
- Hormones – Gonandotrophins, androgens, estrogen
- Biopsy

Inflammations

- Testis – orchitis
- Epididymis – epididymitis.
- Epididymo-orchitis – Etiology varies with age of the patient.

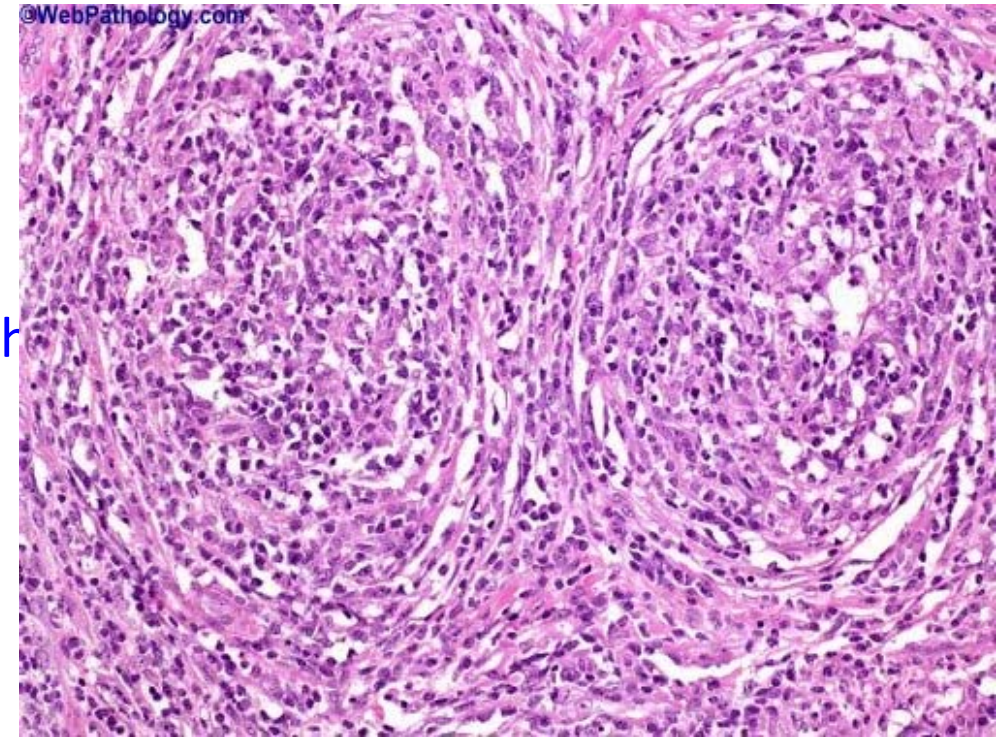
  - children – gram negative bacilli, congenital anomaly
  - <35 y/o – STD – N.gonorrhea, C.trachomatis
  - >35 y/o – UTI – E.coli, Pseudomonas

Epididymo-orchitis

- May be- acute or chronic, granulomatous inflammation.
- Acute – painful, tense, swollen testis
- Chronic – variable degree of atrophy and fibrosis.
- Classic
    - TB and gonorrhea begin in epididymis
    - syphilis begins in testis

Granulomatous orchitis

- Exact etiopathogenesis not known
- Probably – autoimmune, TB(from urethra)
- Occurs as u/l painless testicular enlargement
- Gross – enlarged testis with thickened tunica.
- M/E – non-caseating granulomas in the seminiferous tubules, peritubular fibrosis.



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NEOPLASMS OF TESTIS

- *Testicular neoplasms are the most important cause of firm, painless enlargement of the testis.*
- occur in roughly 5 per 100,000 males, with a peak incidence between the ages of 20 and 34 years.

- Tumors of the testis represent a heterogeneous group of neoplasms composed of germ cell tumors and sex cord/stromal tumors.
- In adults, 95% of testicular tumors arise from germ cells, and all are malignant.
- Neoplasms derived from Sertoli or Leydig cells (sex cord/stromal tumors) are uncommon and, in origin, usually pursue a benign clinical course.

ETIOLOGY-

Cryptorchidism is associated with a 3- to 5-fold increase in the risk of cancer in the undescended testis, as well as an increased risk of cancer in the contralateral descended testis.

- Intersex syndromes, including androgen insensitivity syndrome and gonadal dysgenesis, are also associated with an increased frequency of testicular cancer.
- Cytogenetic studies, the most common of which is an isochromosome of the short arm of chromosome 12.
- The risk of neoplasia is increased in siblings of males with testicular cancers,
- The development of cancer in one testis is associated with a markedly increased risk of neoplasia in the contralateral testis.

Testicular tumors - Classification

- Germ cell tumors(95%)
  - seminoma
  - embryonal carcinoma
  - yolk sac tumor
  - teratomas
  - choriocarcinomas
- Stromal tumors
  - leydig cell tumor
  - sertoli cell tumor

Testicular tumors - Classification

- Combined germ cell – stromal tumors
  - Gonadoblastoma
- Other tumors – Malignant lymphomas

- Minimally differentiated – Embryonal carcinoma
- Seminiferous differentiation – Seminoma
- Somatic differentiation – Teratomas
- Trophoblastic differentiation – Choriocarcinoma
- Yolk sac differentiation – Yolk sac tumor

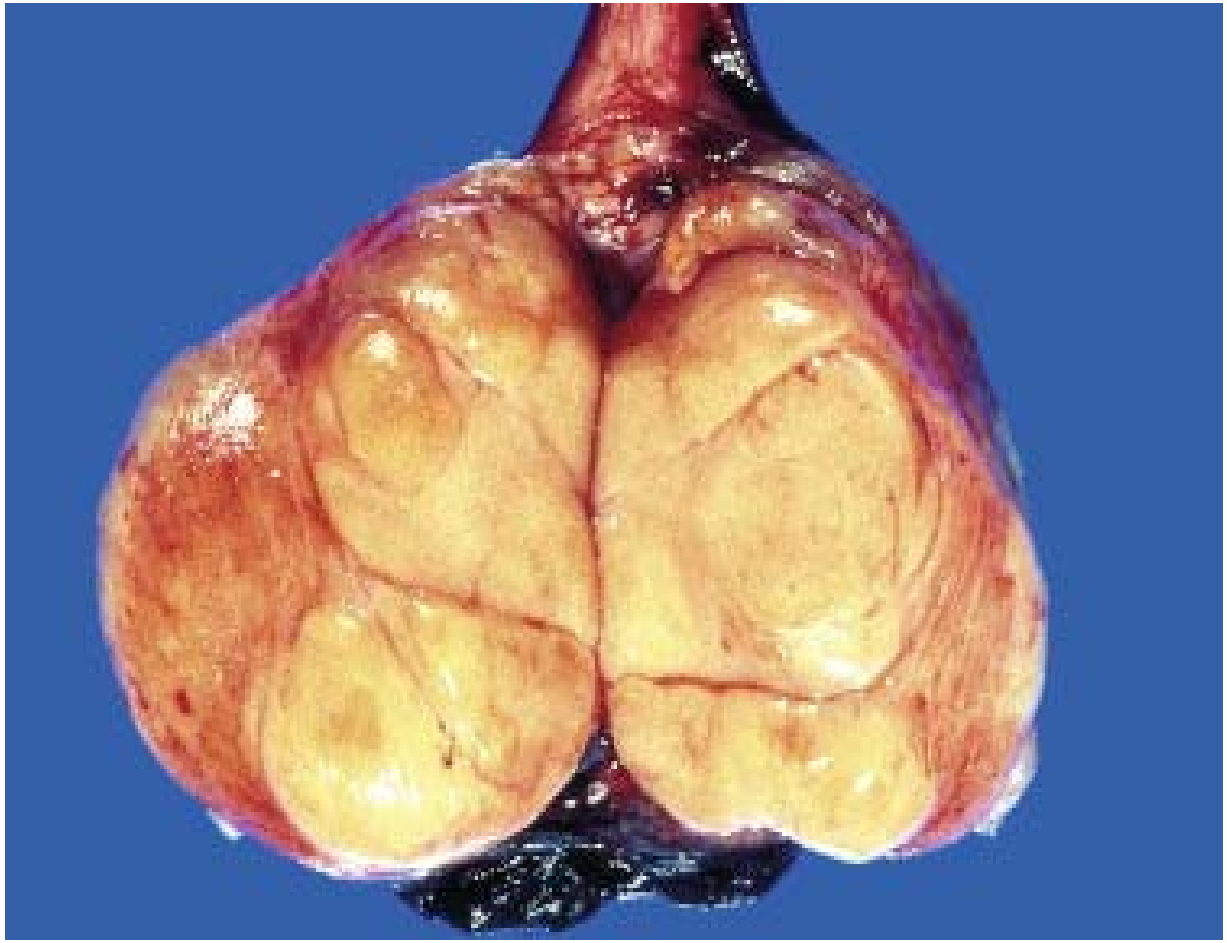
- The World Health Organization classification is the most widely used .
- Germ cell tumors of the testis are divided into two broad categories, based on whether they contain a single histologic pattern (60% of cases) or multiple histologic patterns (40% of cases)
- This classification is based on the view that germ cell tumors of the testis arise from primitive cells that may either differentiate along gonadal lines to produce *seminomas* or transform into a totipotential cell population, giving rise to *nonseminomatous germ cell tumors*
- Such totipotential cells may remain largely undifferentiated to form *embryonal carcinomas,* may differentiate along extra-embryonic lines to form *yolk sac tumors* and *choriocarcinomas,* or may differentiate along somatic cell lines to produce *teratomas*

- Testicular tumors arise from in situ lesions characterized as *intratubular germ cell neoplasia.*
- This lesion is present in conditions associated with a high risk of developing germ cell tumors (e.g., cryptorchidism, dysgenetic testes).
- Furthermore, foci of such in situ lesions are seen in testicular tissue adjacent to a testicular germ cell tumor.

- **Seminomas,** sometimes referred to as "classic" seminomas, account for about 50% of testicular germ cell neoplasms.
- Seminomas are large, soft, well-demarcated, usually homogeneous, gray-white tumors that bulge from the cut surface of the affected testis.
- The neoplasms are typically confined to the testis by an intact tunica albuginea. Large tumors may contain foci of coagulation necrosis, usually without hemorrhage. The presence of hemorrhage should prompt careful scrutiny for an associated nonseminomatous germ cell component to the tumor

- Microscopically, seminomas are composed of **large, uniform cells with distinct cell borders, clear, glycogen-rich cytoplasm, and round nuclei with conspicuous nucleoli**
- The cells are often arrayed in small lobules with intervening fibrous septa. A lymphocytic infiltrate is usually present.
- A granulomatous inflammatory reaction may also be present.
- In as many as 25% of cases, cells staining positively for human chorionic gonadotropin (hCG) can be seen.

GROSS



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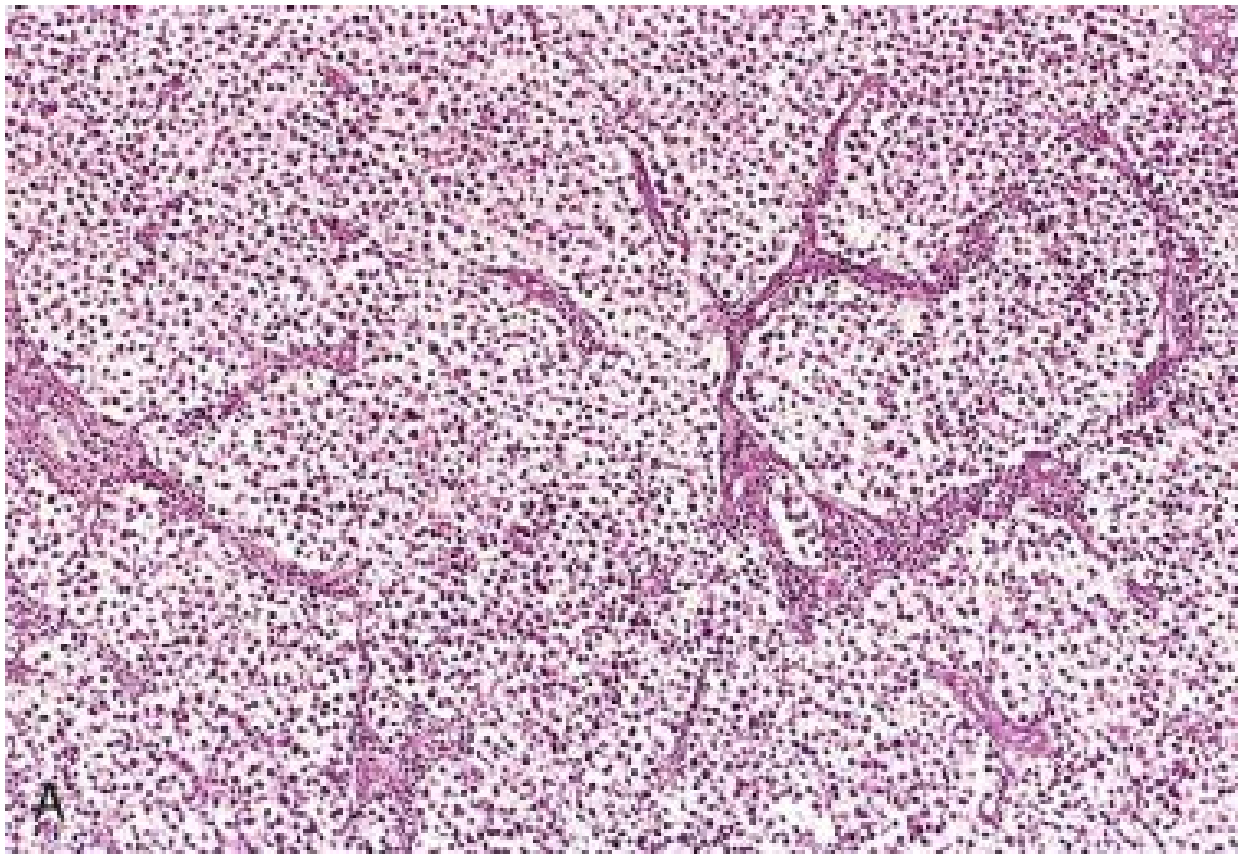

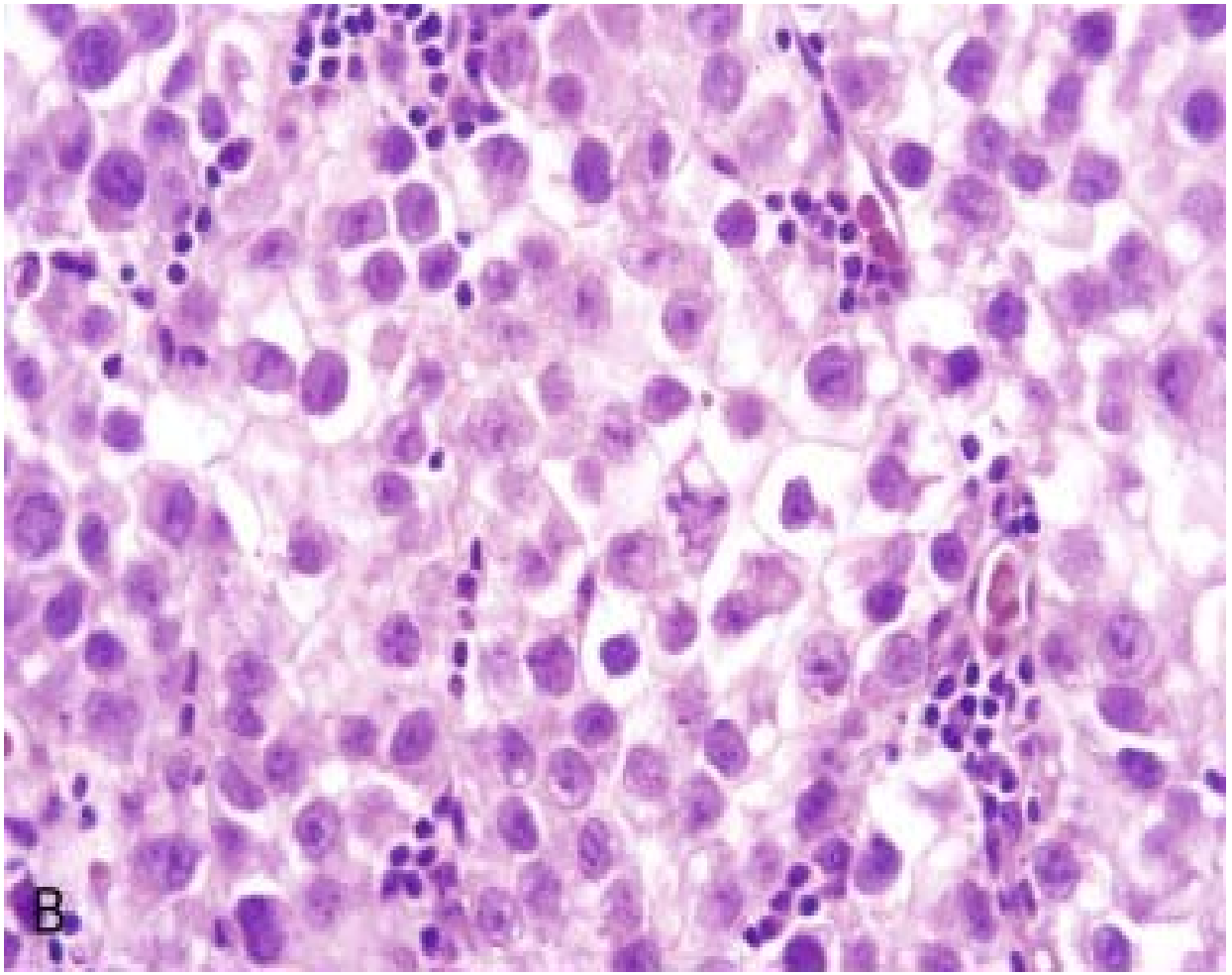
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SPERMATOCYTIC SEMINOMA

- Morphologic variant of seminoma is the so-called **spermatocytic seminoma.**
- These tumors occur in older patients, contain a mixture of medium-sized cells, large uninucleate or multinucleate tumor cells, and small cells with round nuclei that are reminiscent of secondary spermatocytes.
- There is no association with intratubular germ cell neoplasia,
- metastases are exceedingly rare, in contrast to the behavior of classic seminoma.

EMBRYONAL CARCINOMA

- **Embryonal carcinomas** are ill-defined, invasive masses containing foci of hemorrhage and necrosis.
- The constituent cells are **large and primitive looking, with basophilic cytoplasm, indistinct cell borders, and large nuclei with prominent nucleoli.**
- The neoplastic cells may be arrayed in undifferentiated, solid sheets or may contain glandular structures and irregular papillae In most cases, other patterns of germ cell neoplasia (e.g., yolk sac carcinoma, teratoma, choriocarcinoma) are admixed with the embryonal areas.
- Pure embryonal carcinomas comprise 2% to 3% of all testicular germ cell tumors.
- As with other germ cell tumors of the testes, foci of intratubular germ cell neoplasia are frequently present in the adjacent

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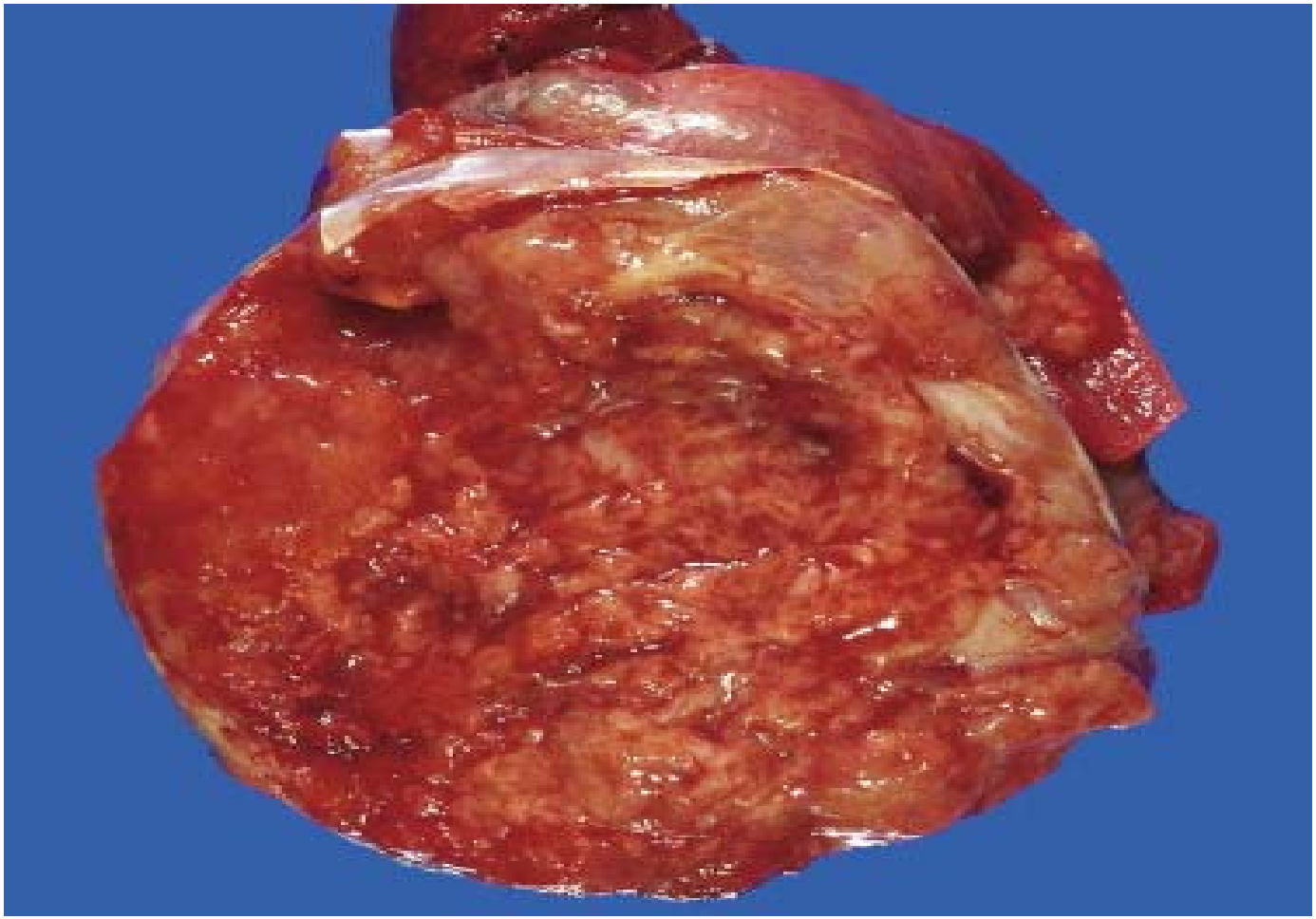

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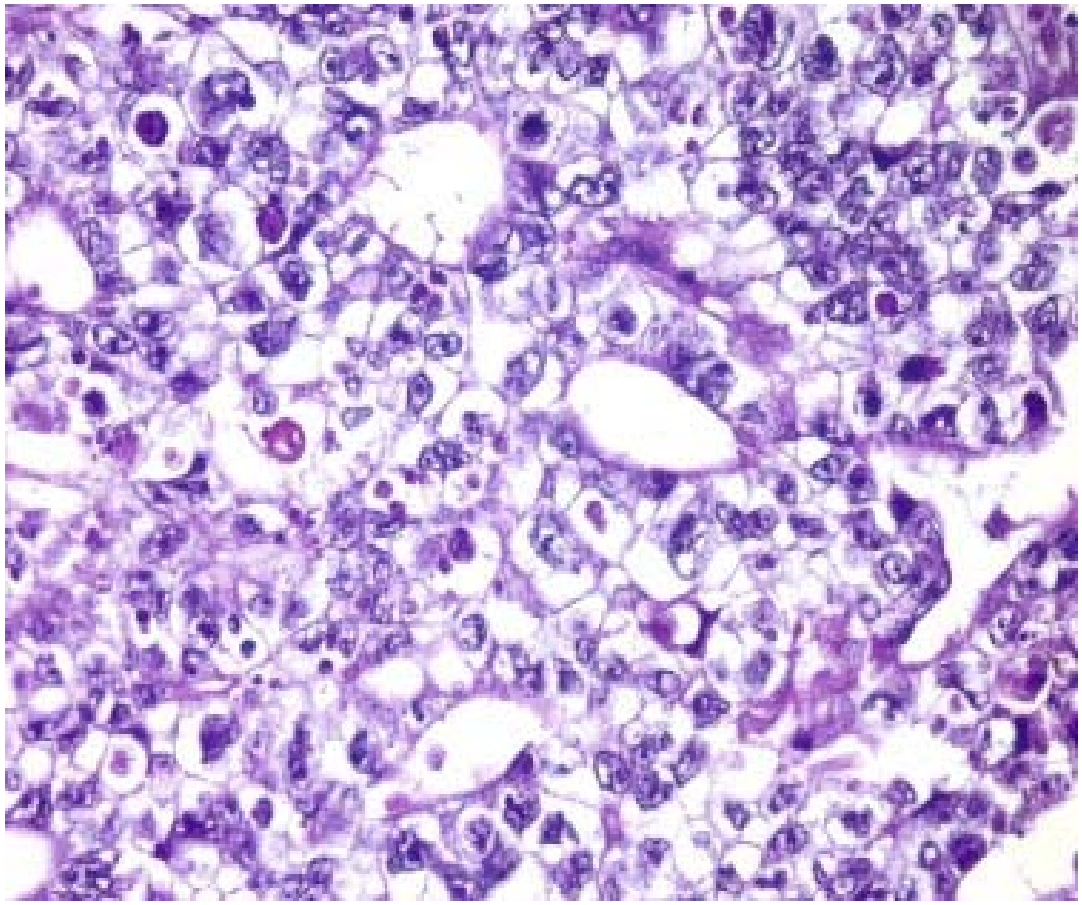

YOLK SAC YUMOR

- **Yolk sac tumors,** also termed **endodermal sinus tumors,** are the most common primary testicular neoplasm in children younger than 3 years of age
- In adults, yolk sac tumors are most often seen admixed with embryonal cacinoma. Grossly, these tumors are often large and may be well demarcated.
  - Histologic examination :low cuboidal to columnar epithelial cells forming microcysts, sheets, glands, and papillae, often associated with eosinophilic hyaline globules .A distinctive feature is the presence of structures resembling primitive glomeruli, the so-called **Schiller-Duvall** bodies.
  - α-fetoprotein (AFP) can be demonstrated within the cytoplasm of the neoplastic cells by immunohistochemical techniques.

CHORIOCARCINOMA

- **Choriocarcinomas** represent differentiation of pluripotential neoplastic germ cells along **trophoblastic** lines.
- Grossly, the primary tumors are often small, nonpalpable lesions, even with extensive systemic metastases.
- Microscopically, choriocarcinomas are composed of sheets of small cuboidal cells irregularly intermingled with or capped by large, eosinophilic syncytial cells containing multiple dark, pleomorphic nuclei; these represent **cytotrophoblastic** and **syncytiotrophoblastic** differentiation, respectively
- The hormone hCG can be identified with appropriate immunohistochemical staining, particularly within the cytoplasm of the syncytiotrophoblastic elements.

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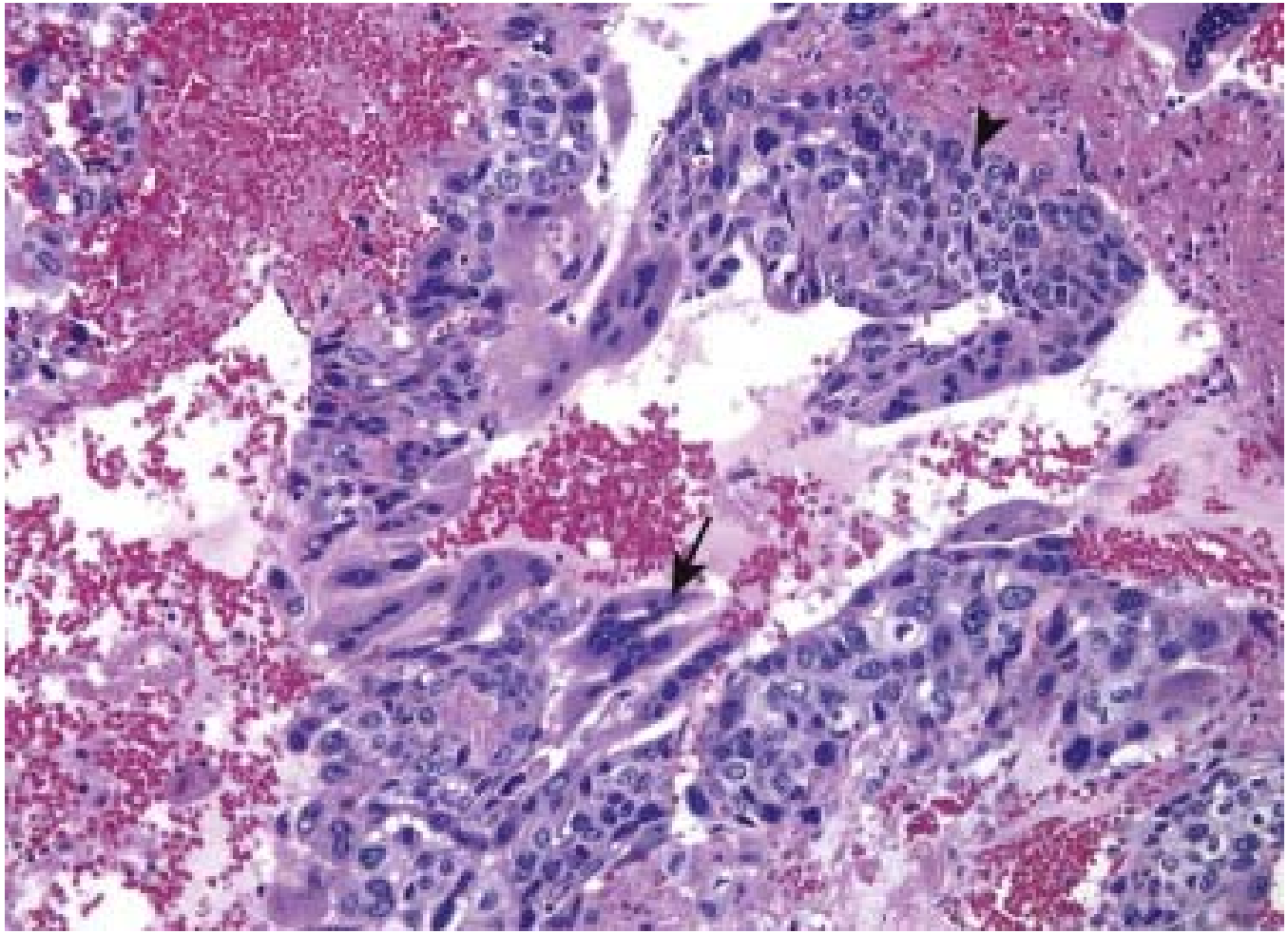

TERATOMAS

- **Teratomas** represent differentiation of neoplastic germ cells along **somatic** cell lines. These tumors form firm masses that on cut surface often contain cysts and recognizable areas of cartilage.
- Histologically, three major variants of pure teratoma are recognized. **Mature teratomas** contain fully differentiated tissues from one or more germ cell layers (e.g., neural tissue, cartilage, adipose tissue, bone, epithelium) in a haphazard array
- **Immature teratomas,** in contrast, contain immature somatic elements reminiscent of those in developing fetal tissue
- **Teratomas with somatic-type malignancies** are characterized by the development of frank malignancy in preexisting teratomatous elements, usually in the form of a squamous cell carcinoma or adenocarcinoma. Pure teratomas in prepubertal males are usually benign.

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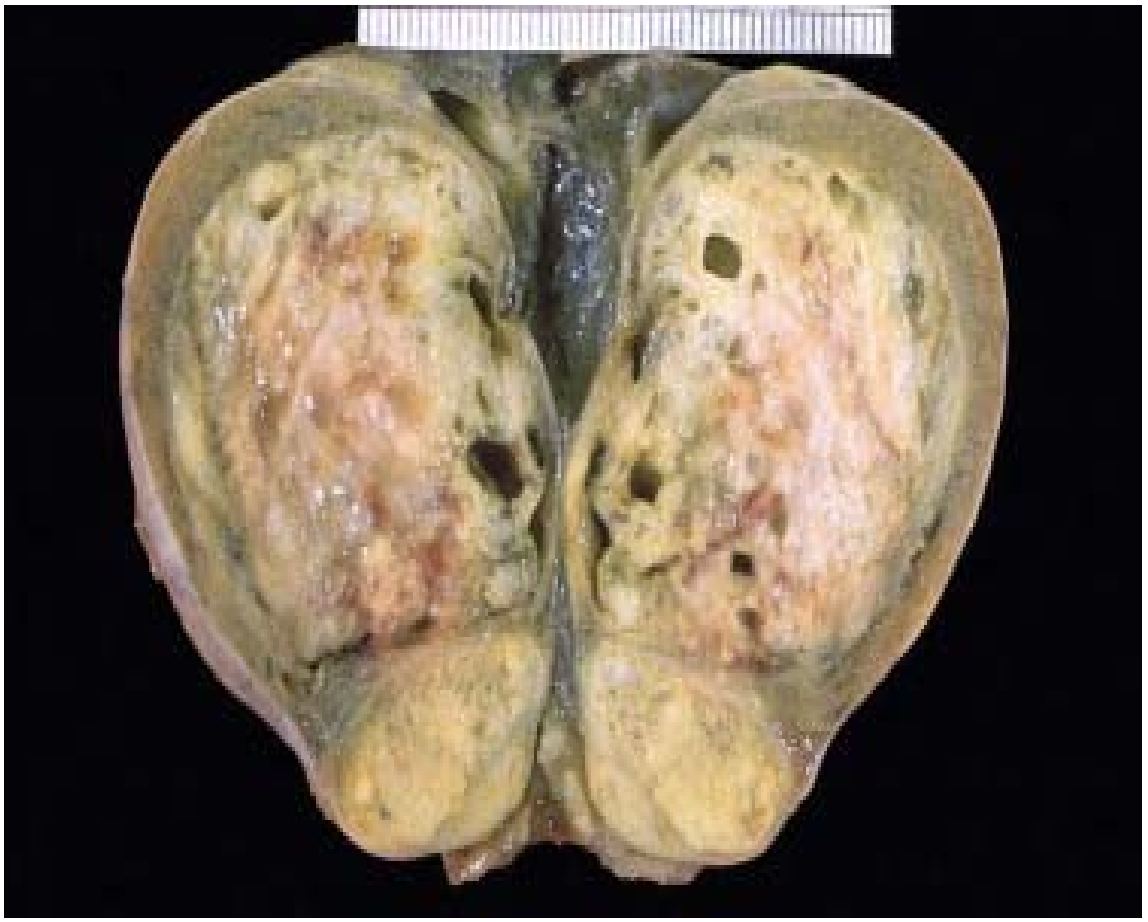

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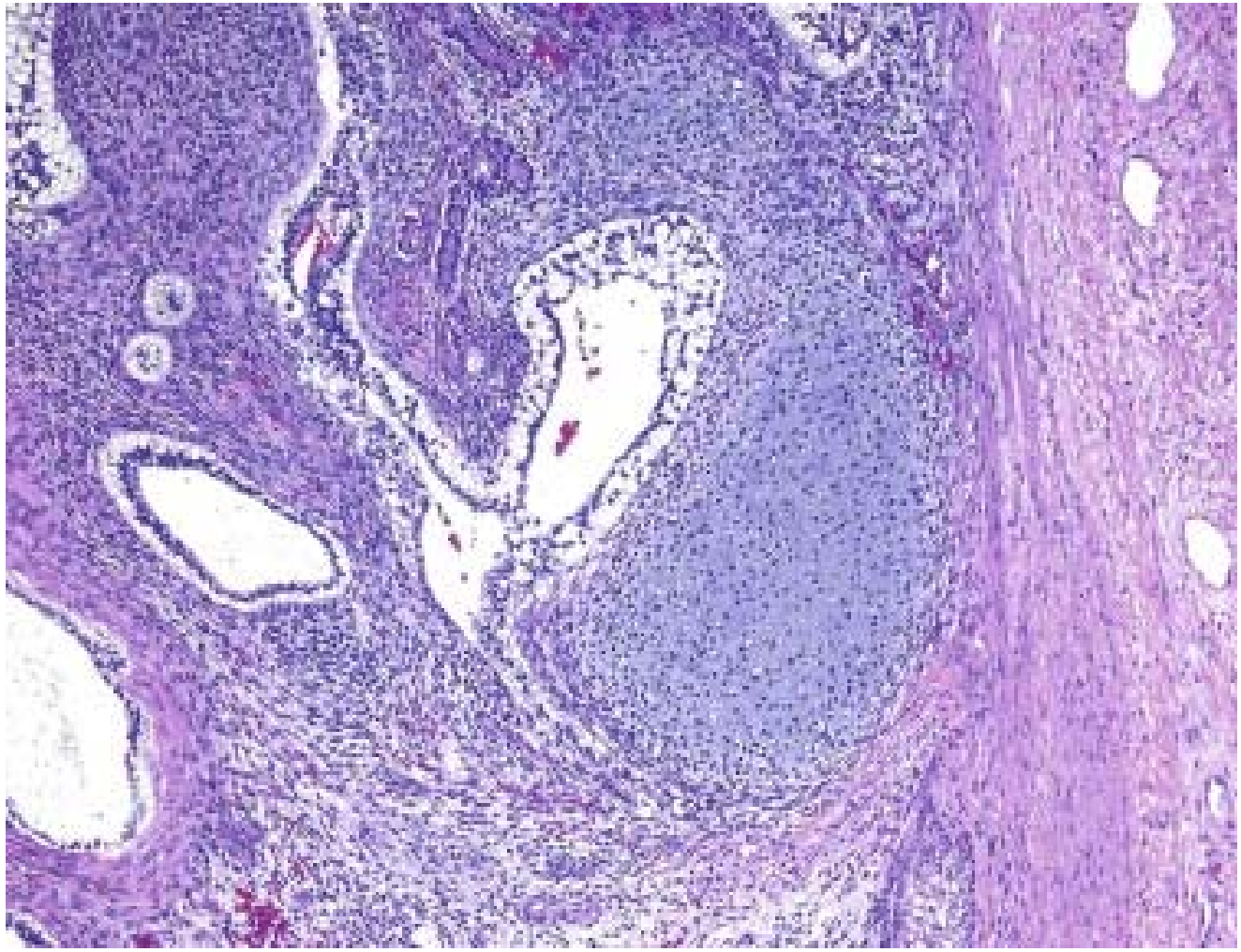

Clinical features:

- *Painless enlargement of the testis*
- However, some tumors, especially nonseminomatous germ cell neoplasms, may have widespread metastases at diagnosis, in the absence of a palpable testicular lesion.
- *Seminomas often remain confined to the testis* for prolonged intervals and may reach considerable size before diagnosis. Metastases are most commonly encountered in the iliac and para-aortic lymph nodes
- In contrast, *nonseminomatous germ cell neoplasms tend to metastasize earlier,*

- Assay of *tumor markers* secreted by tumor cells is important in the clinical evaluation and staging of germ cell neoplasms). hCG, produced by neoplastic syncytiotrophoblastic cells, is always elevated in patients with choriocarcinoma.

  Seminoma, may also contain syncytiotrophoblastic cells without cytotrophoblastic elements and hence may elaborate hCG
- AFP is a glycoprotein normally synthesized by the fetal yolk sac and several other fetal tissues. Nonseminomatous germ cell tumors containing elements of yolk sac (endodermal sinus) often produce AFP

  The presence of AFP is a reliable indicator of the presence of a nonseminomatous component to the germ cell neoplasm
- In addition to their role in the primary diagnosis and staging of testicular germ cell tumors, serial determinations of hCG and AFP are useful for monitoring patients for persistent or recurrent tumor after therapy.

Prognosis

- Histological type and stage dictate therapy and prognosis
- Seminoma- very radio sensitive and has excellent prognosis
- Mixed germ cell tumors- chemotherapy leads to good prognosis.