

Pathology of male genital system

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THE MALE GENITAL SYSTEM

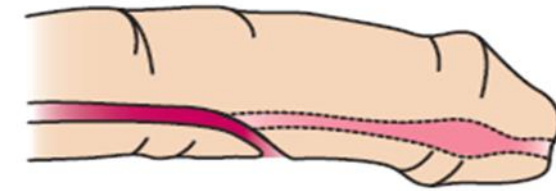
penis

CONGENITAL ANOMALIES

1-Hypospadias and Epispadias:

Malformation of the urethral groove and canal may create an abnormal opening either on the ventral surface of the penis (hypospadias) or on the dorsal surface (epispadias). Either of these anomalies can be associated with failure of normal descent of the testes and with malformations of the urinary tract. Hypospadias, the more common of the two, occurs in approximately 1 in 300 live male births.

Hypospadias



Epispadias



2-PHIMOSIS:

Definition: *This is congenital stenosis of the orifice of the prepuce.*

Complications:

- *Balanitis (inflammation of glans penis).*
- *Urinary tract obstruction (bilateral hydronephrosis).*



Inflammation:

BALANITIS: *This is inflammation of glans penis caused by pyogenic bacteria as staphylococci, streptococci & gonococci. It is predisposed to by phimosis. It is precancerous.*

Tumors

Tumors of the penis are uncommon. The most frequent neoplasms are squamous cell carcinoma and benign genital warts (condyloma acuminatum).

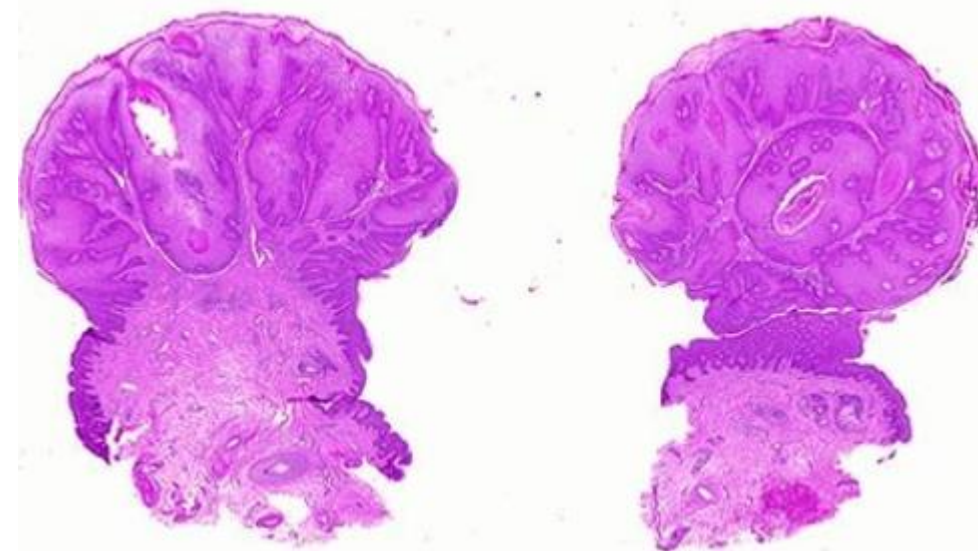
Benign Tumors and Tumor-Like Conditions:

Condyloma Acuminatum:

is a benign sexually transmitted wart caused by human papillomavirus (HPV). It is related to the common wart and may occur on any moist mucocutaneous surface of the external genitals in either sex. “Lowrisk” HPV serotypes (HPV 6 and, less frequently, HPV 11) are the most frequent cause of condylomata acuminata.

Pathological features:

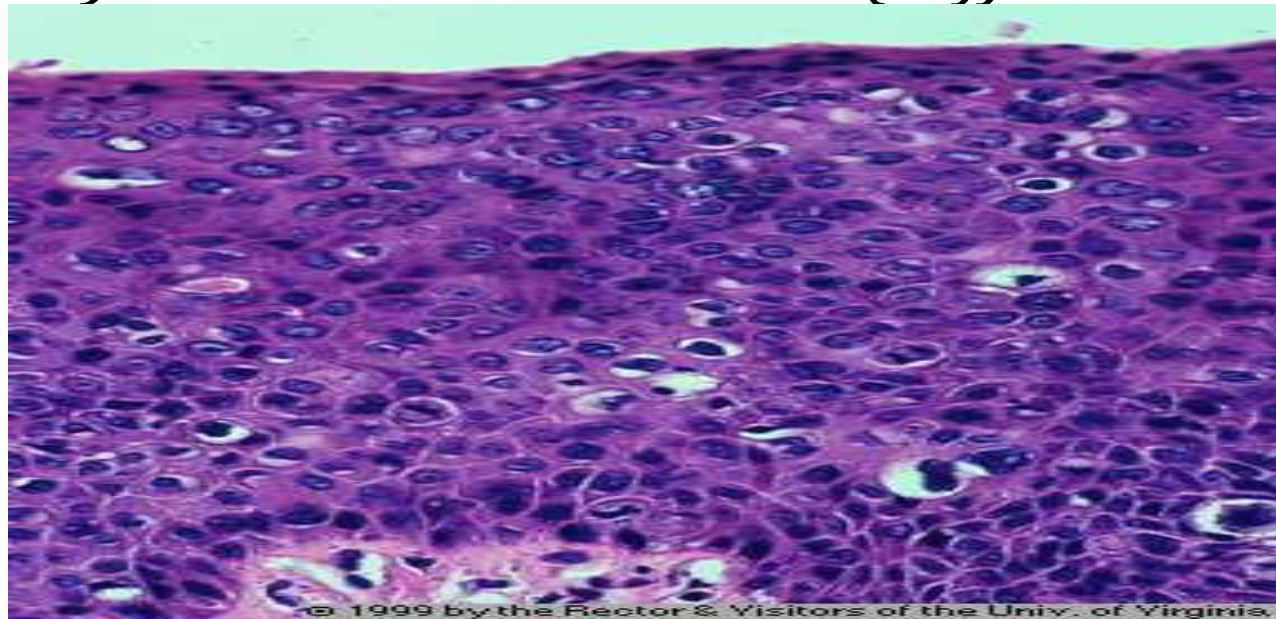
Condylomata acuminata may arise in the external genitalia or perineal areas. Penile lesions usually occur in the coronal sulcus and inner surface of the prepuce. They consist of single or multiple sessile or pedunculated, red papillary excrescences that are several millimeters in diameter . Histologically, a branching, villous, papillary connective tissue stroma is covered by epithelium that may have considerable superficial hyperkeratosis and thickening of the underlying epidermis (acanthosis).



Malignant Tumors

Squamous Carcinoma in situ/Penile Intraepithelial Neoplasia:

As at other sites, such as uterine cervix, in situ squamous lesions span a range of morphologies from CIS to less severe derangements. These lesions are encompassed by the umbrella term penile intraepithelial neoplasia (PeIN). All are squamous lesions confined to the epidermis by an intact basement membrane. PeIN may be HPV-related (undifferentiated PeIN) or non-HPV-related (differentiated).

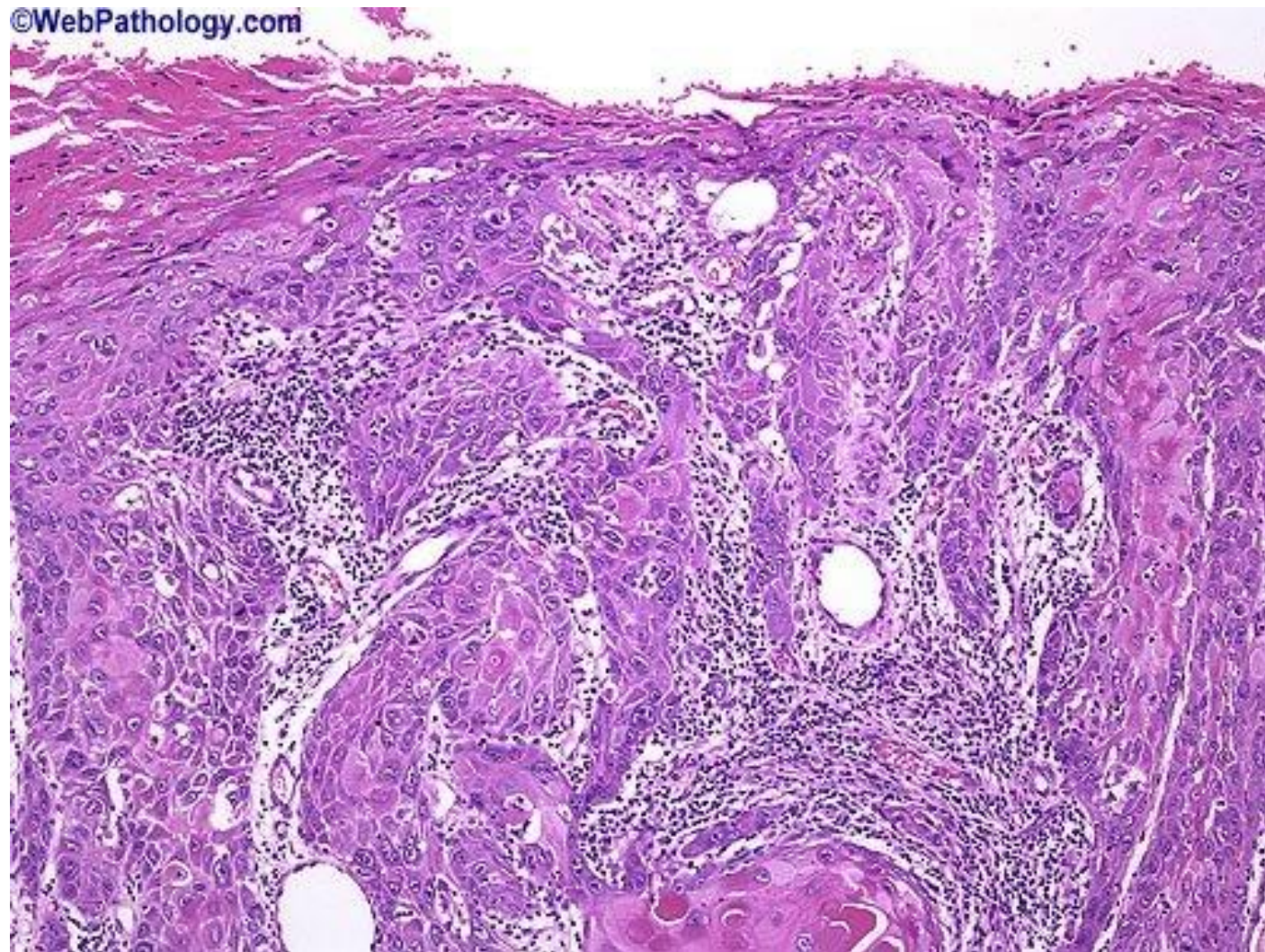


Invasive Squamous Cell Carcinoma

Squamous cell carcinoma of the penis is associated with poor genital hygiene and high-risk HPV infection. Penile carcinoma affects middle-aged and older patients (40 to 70 years of age). Circumcision confers protection, and hence penile cancer is extremely rare among Muslims and Jews

MORPHOLOGY

Squamous cell carcinoma of the penis usually originates in glans or inner surface of the prepuce near the coronal sulcus. Macroscopically the tumors may be irregular, fungating cauliflower-like masses; flat, indurated lesions; or large verruciform/papillary tumors. The World Health Organization (WHO) 2016 classification of penile squamous cell carcinoma recognizes HPV-related and non-HPV-related categories



TESTIS AND EPIDIDYMISS

CONGENITAL ANOMALIES

2-CRYPTORCHIDISM:

Definition: This is failure of descent of one or both testis into the scrotum.

Aetiology

- Short vas.
- Hormonal disturbances.
- Anomalies of testis, scrotum or inguinal canal.

Sites: The undescended testis may exist in abdomen, pelvis or inguinal canal

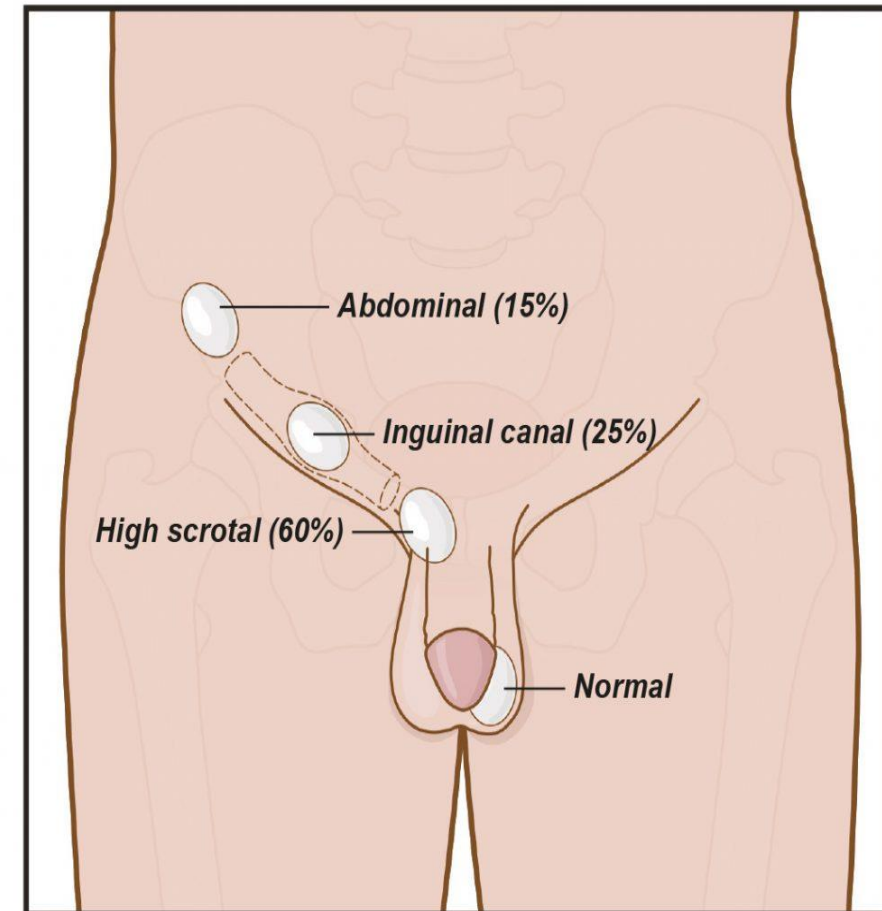
Complications: If untreated

- Testicular atrophy (infertility).
- Cryptorchidism is precancerous.

3-OTHER ANOMALIES as

a)polyorchidism: supernumerary testis.

b)splenic-gonadal fusion



ORCHITIS & EPIDIDYMITIS

1-Acute Epididymo Orchitis: Bacterial infection through direct spread (from cystitis, urethritis.. etc), lymphatic or blood spread. It is predisposed to by low immunity (as diabetics). It may lead to abscess formation. Healing by fibrosis may epididymal obstruction (infertility may occur in bilateral cases)

2-Chronic nonspecific epididymitis and orchitis: May complicate the acute form.

3-Mums Orchitis (Viral): It is a complication of mumps

4-Radiation Orchitis

5-Idiopathic Granulomatous Orchitis: Granulomatous inflammation of unknown aetiology.

REMARK: Other types of epididymo-orchitis include:

1-Specific Infections Bilharzial , Tuberculous, Syphilitic and Fungal infections

2. Spermatic Granuloma of epididymis and vas. This is granulomatous inflammation occurring in the interstitial tissue around escaping sperms. The etiology is unknown, but it is likely post-traumatic.

3-Malakoplakia of testis and epididymis.

TUMORS OF TESTIS

CLASSIFICATION OF TESTICULAR TUMORS ACCORDING TO ORIGIN

1-Germ Cell Tumors: Malignant

- a) Seminoma & spermatocytic tumor***
- b) Teratomas, embryonal carcinoma, teratocarcinoma, choriocarcinoma***
- c) Yolk sac tumor***

2-Sex Cord-Stromal Tumors: Benign or malignant

- a) Leydig cell (interstitial cell) tumor***
- b) Sertoli cell tumor (androblastoma)***
- c) Granulosa cell tumor***
- d) Mixed types, Sertoli-Leydig cell tumor***

3-Mixed Germ Cell- Sex Cord Stromal Cell Tumors (Gonadoblastoma)

4-Tumors of Rete Testis: a) Adenoma, cystadenoma, adenofibroma b) Carcinoma

5-Lymphoma.

6-Paratesticular Tumors (tumors of epididymis, tunica vaginalis and soft tissue) as :

a Adenomatoid tumor

b) Neurofibroma, leiomyoma, lipoma

c) Mesothelioma

c) Epididymal cystadenoma & carcinoma d) Rhabdomyosarcoma

e) Vascular tumors f) Others

7-Secondary Tumors (metastases).

Table 21.7 Pathologic Classification of Common Testicular Tumors

Germ Cell Tumors Derived From Germ Cell Neoplasia in Situ
Noninvasive germ cell neoplasia Germ cell neoplasia in situ
Tumors of a single histologic type (pure forms) Seminoma
Nonseminomatous germ cell tumors Embryonal carcinoma Yolk sac tumor, postpubertal type Choriocarcinoma Teratoma, postpubertal type Teratoma with somatic-type malignancy
Nonseminomatous germ cell tumors of more than one histologic type Mixed germ cell tumor
Germ Cell Tumors Unrelated to Germ Cell Neoplasia in Situ
Spermatocytic tumor
Teratoma, prepubertal type
Yolk sac tumor, prepubertal type
Mixed teratoma and yolk sac tumor, prepubertal type
Sex Cord–Stromal Tumors
Pure tumors
Leydig cell tumor
Sertoli cell tumor
Other
Tumor Containing Both Germ Cell and Sex Cord–Stromal Elements
Gonadoblastoma

I-BENIGN TUMORS

1-LEYDIG CELL TUMOR (interstitial cell tumor):

A rare benign tumor.

Gross: It appears as a rounded yellowish mass

Microscopy: It consists of proliferated Leydig cells.

The tumor cells secrete androgens & oestrogens. In children it may → precocious puberty

2-SERTOLI CELL TUMOR: *A rare benign tumor secreting oestrogens.*

3-OTHERS as:

a) Mature teratoma

b) Benign paratesticular tumors as epididymal adenoma, neurofibroma, leiomyoma, lipoma, angioma & others.

Spermatic Cord and Paratesticular Tumors:

Lipomas are common lesions involving the proximal spermatic cord, identified at the time of inguinal hernia repair. Although diagnosed as “lipomas,” many of these lesions probably represent retroperitoneal adipose tissue that has been pulled into the inguinal canal along with the hernia sac, rather than a true neoplasm.

Adenomatoid tumor is the most common benign paratesticular tumor . Although these lesions are mesothelial in nature, Adenomatoid tumors are usually small nodules, typically occurring near the upper pole of the epididymis. The importance of this lesion is that it is one of the few benign tumors that occur near the testis. If the pathologist can identify the nature of this lesion in intraoperative frozen sections, local excision of the adenomatoid tumor can spare the patient from an orchiectomy.

The most common malignant paratesticular tumors are rhabdomyosarcomas in children and liposarcomas in adults.

I-MALIGNANT TUMORS

SEMINOMA (40%)

Definition & Origin: It is a malignant tumor arising from germ cells (seminiferous) of the testis. it is the commonest testicular tumor (40%) & has a better prognosis, since it is radiosensitive.

Gross: The testis is moderately enlarged. Cut section shows a noncapsulated solid homogenous pale yellow growth, sometimes with small necrotic foci.

Microscopy: Many types:

1-Classic Seminoma: It consists of uniform large cells with abundant clear cytoplasm and large central nuclei containing large nucleoli. The cells exist in groups separated by fibrous bands invariably infiltrated by lymphocytes and plasma cells.

2-Spermatocytic tumor: It consists of cells showing marked size variations (a mixture of small cells, large cells & bizarre giant cells). Mitoses may be numerous and lymphocytic infiltration is absent.

3-Other types such as Classic seminoma with trophoblastic giant cells & anaplastic seminoma (seminoma with high mitotic activity).

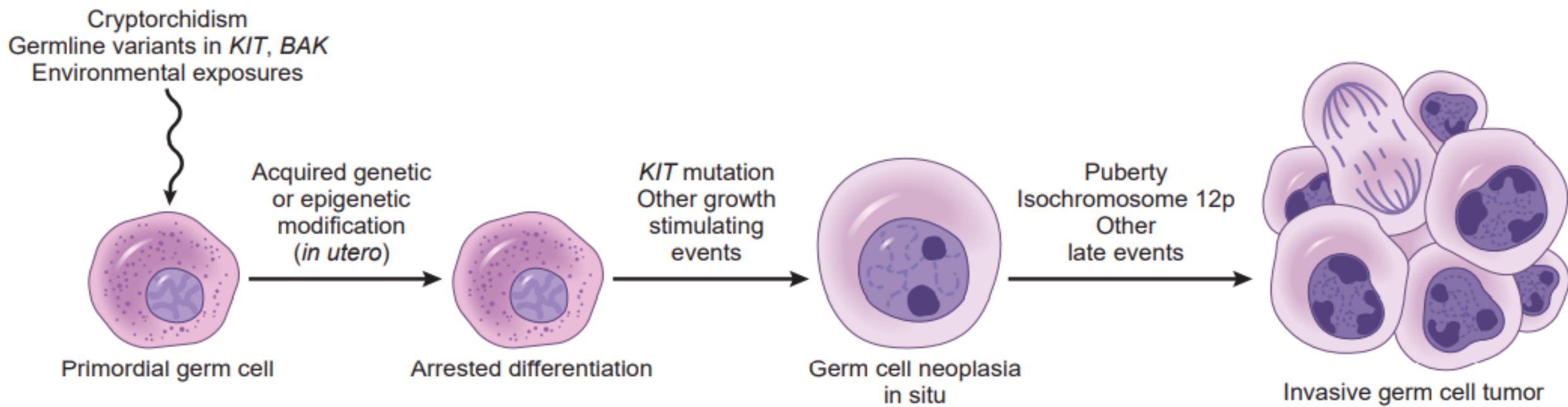
Age: Spermatocytic tumor occurs in old age groups (around 65 years). Other types of seminoma occur around the age of 30-40 years.

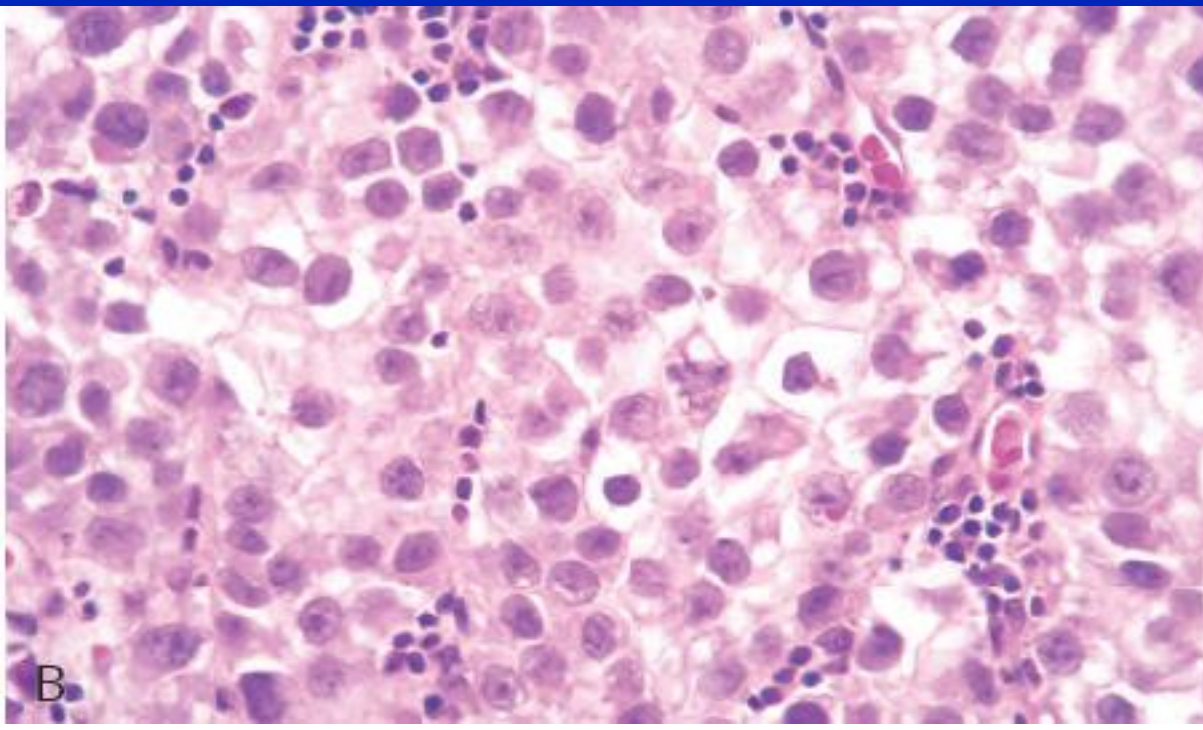
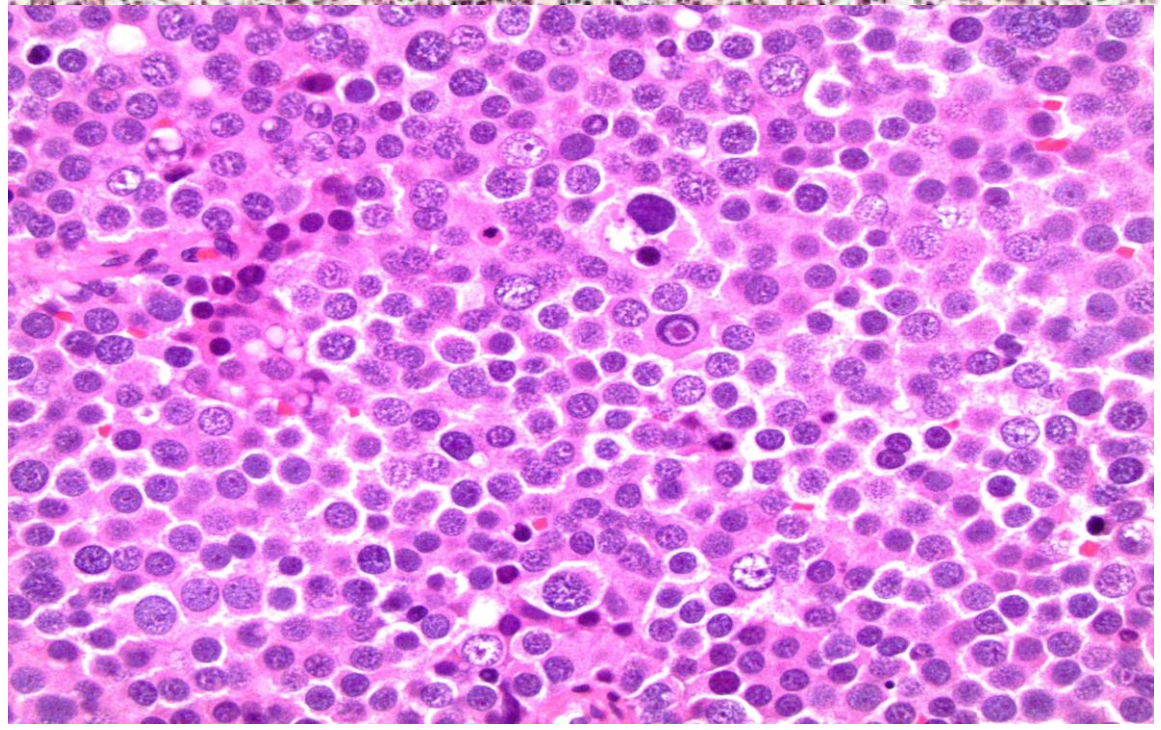
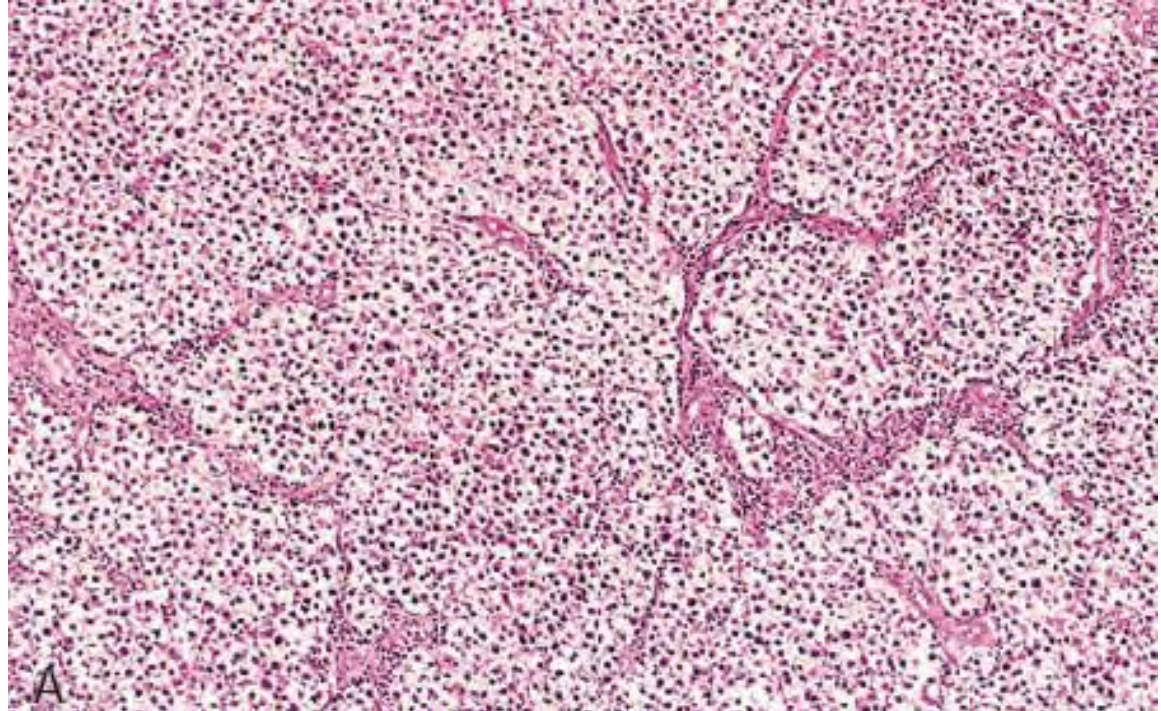
Spread of Seminoma (& other malignant testicular tumors).

1-Direct: to testicular adnexa and scrotal wall.

2-Lymphatic: Para-aortic lymph nodes.

3-Blood : Lung, liver, brain, and bone.





Prognosis: Seminomas are radiosensitive tumors.

1-Spermatocytic tumor never metastasizes and has an excellent prognosis.

2-Classic seminomas stage 1 (limited to testis) or stage II (spread to infra diaphragmatic lymph nodes) have also excellent prognosis and over 95% of these patients are cured. With stage III (metastases), the prognosis is unfavorable.

TERATOMA GROUP (32%)

1-Mature (differentiated) teratoma:

A benign tumor that may undergo malignant transformation.

It consists of a mixture of mature tissues derived from ectoderm, endoderm & mesoderm.

It grossly appears as a rounded tumor, usually with multiple cysts.

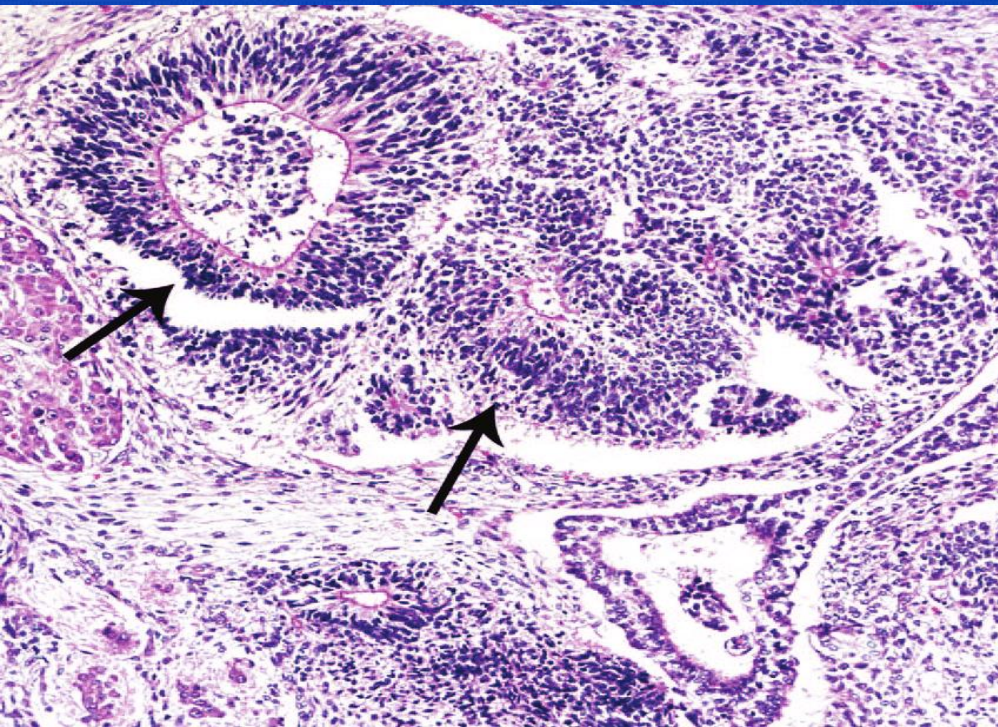
2-Immature teratoma.

This is a malignant tumor which grossly shows necrosis & hemorrhage.

Microscopically it consists of a mixture of tissues derived from ectoderm, endoderm & mesoderm; some of which appear immature (malignant), whether epithelial, mesenchymal or both.



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3-Other tumors which are sometimes classified among the teratoma group:

Embryonal carcinoma: Sometimes considered undifferentiated teratoma. It consists of very primitive cells with high anaplasia & frequent mitosis.

Teratocarcinoma: A mixture of embryonal carcinoma & classic teratoma.

Choriocarcinoma: It is a highly malignant tumor which consists of malignant trophoblastic epithelium (placental type tissue). is sometimes considered as a monodermal teratoma. It secretes human chorionic gonadotropin.

Carcinoid tumor: similar to that of GIT. It is sometimes considered a monodermal teratoma.

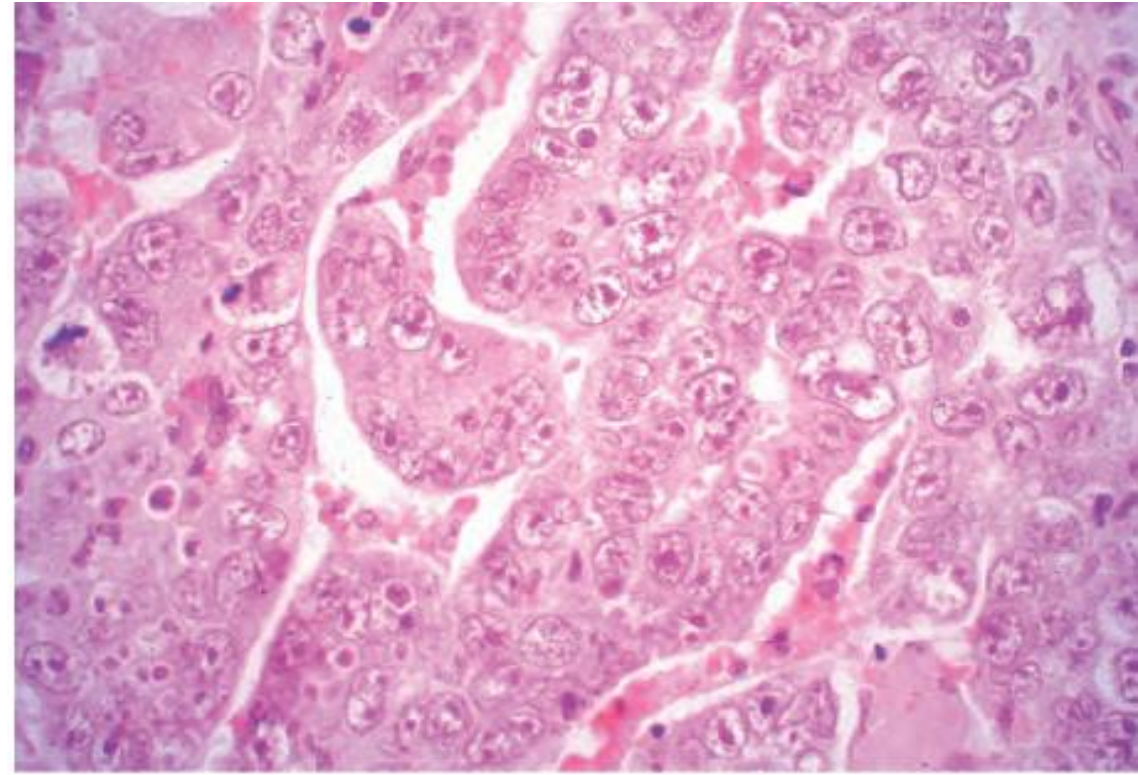
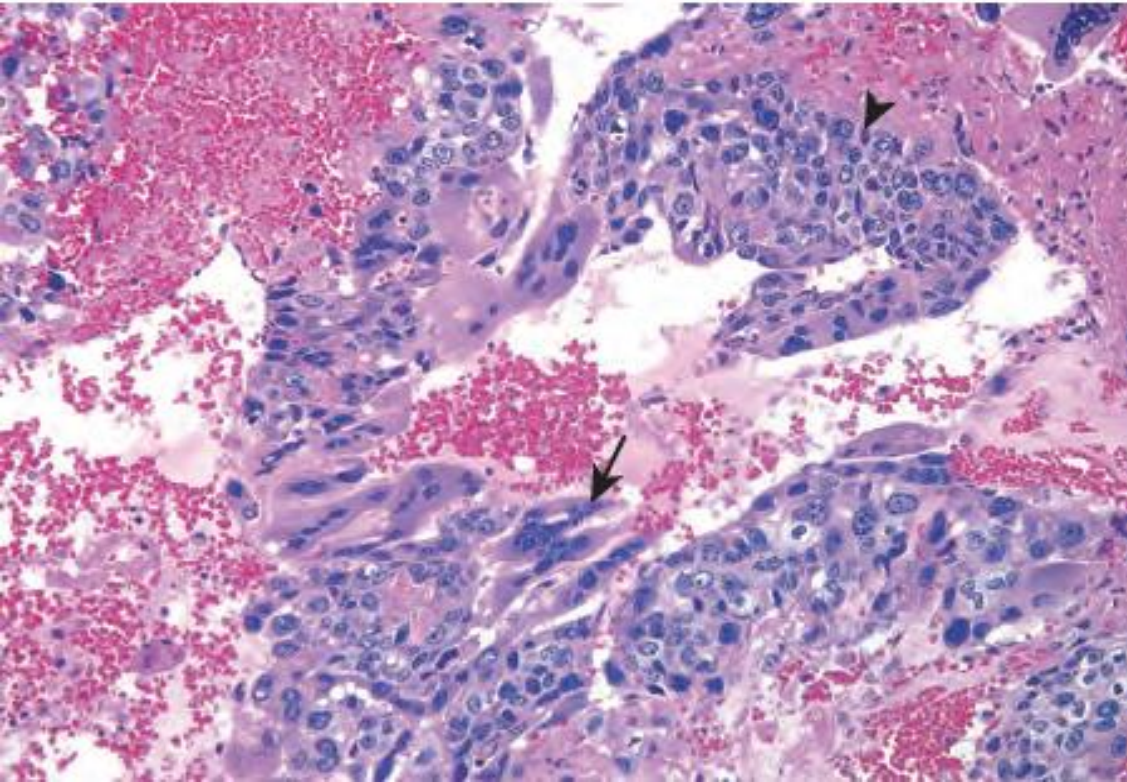


Figure 21.22 Embryonal carcinoma composed of cells with large, hyperchromatic nuclei arranged in sheets and poorly formed glands.

YOLK SAC TUMOR ***(Endodermal sinus Tumor)***

*It mostly occurs in infants.
It consists of very
primitive cells exhibiting
microcystic, glomeruloid,
hepatoid, clear cell and
papillary structures
(resembling the normal
yolk sac).*

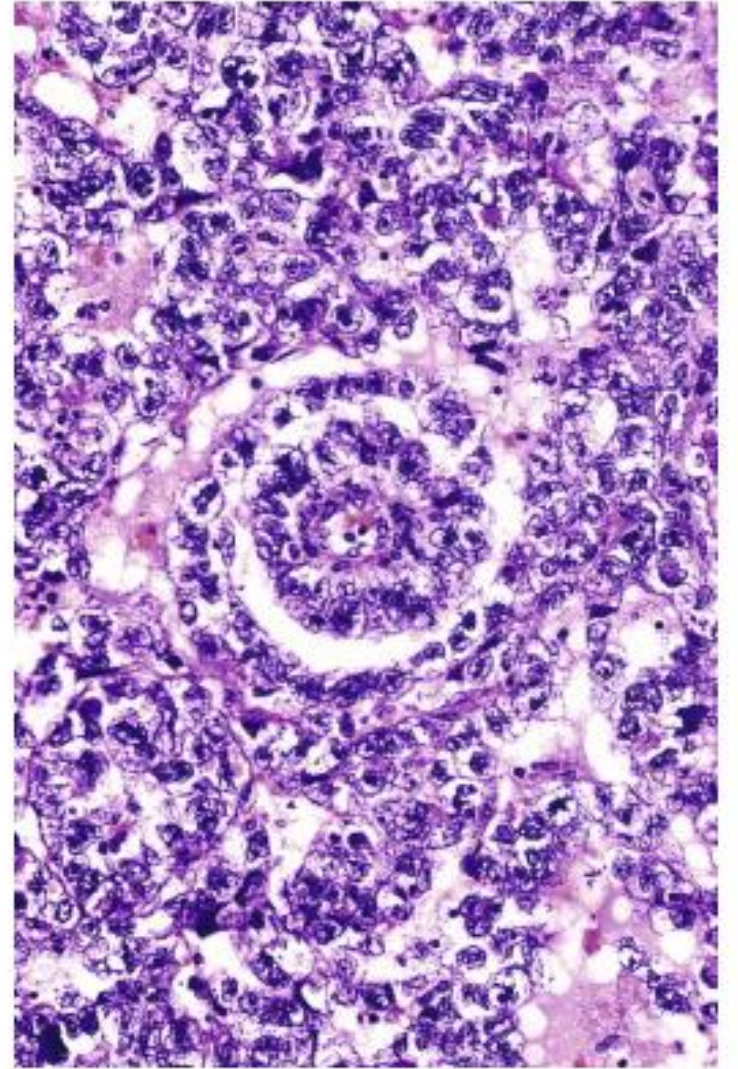


Fig. 18.81 Schiller–Duval body in yolk sac tumor of testis

HYDROCELE

Definition: Accumulation of serous fluid in the tunica vaginalis sac.

Aetiology:

1-Primary (idiopathic) type: This is the most common type:

Undetermined aetiology.

2-Congenital: Patent processus vaginalis.

3-Acquired:

a) Inflammations of epididymis & testis & filariasis of spermatic cord.

b) Tumors of epididymis & testis

c) Trauma and torsion of testis.

Effects: Tunica shows fibrous thickening. Some cases may be complicated by:

1-Testicular atrophy in prolonged cases.

2-Secondary infection.

VARICOCELE

Definition: Varicosity (elongation, dilatation & tortuosity) of the pampiniform venous plexus.

Types:

1-Primary (idiopathic, non-obstructive) type: Common in young males, particularly in the left side.

It may be to marked congestion caused by unrelieved sexual excitement

2-Secondary type due to a)chronic general venous congestion b) obstruction of spermatic veins (e.g left varicocele in case of left renal cell carcinoma)

Effects: Hypospermatogenesis may occur leading to infertility.

HEMATOCELE

Definition: Blood accumulating in the tunica vaginalis sac

Aetiology:

1-Local causes as trauma or tumors.

2-General causes of bleeding as leukemia

Effects:

1-Fibrosis, calcification and pressure atrophy of the testis.

2-Secondary infection (pyocele).

CHYLOCELE

Chylous effusion within the tunica vaginalis sac is caused by lymphatic obstruction (e. filariasis)

INFERTILITY

The causes of male infertility include:

1-PRETESTICULAR CAUSES: Hormonal abnormalities

2-TESTICULAR CAUSES (most common aetiology of infertility). The common patterns are:

Maturation Arrest: Spermatogenesis is arrested at the phase of spermatocytes (or rarely spermatids). No sperms are produced (azoospermia).

Germ cell aplasia (Sertoli Cell Only Syndrome): Azoospermia is due to aplasia of germ cells.

Klinefelter Syndrome: Azoospermia is due to chromosomal abnormality (XXY).

Hypospermatogenesis: This is reduction of all germ cell phases. Sperm formation occurs but in reduced numbers. Varicocele is sometimes associated with hypospermatogenesis. Bilateral orchitis.

3. POST-TESTICULAR CAUSES (OBSTRUCTIVE AZOOSPERMIA)

Spermatogenesis is present, but the sperm pathway is obstructed due to congenital, Idiopathic or post inflammatory strictures of the vas deferens.

PROSTATE

Inflammation:

1-Acute Prostatitis (Prostatic Abscess): *Caused by pyogenic bacteria as staphylococci, gonococci ...etc. Those bacteria reach the prostate directly from urethritis or cystitis, or by blood from distant infections. The prostate is enlarged , Congested & infiltrated by neutrophils. Severe cases lead to abscess formation. The condition may change into chronic nonspecific cystitis.*

2-Chronic Nonspecific Prostatitis: *characterized by chronic inflammatory cell infiltrates (mostly lymphocytes). May follow acute prostatitis and may also accompany prostatic hyperplasia.*

3-Granulomatous prostatitis: *may be caused by a specific infectious agent or may refer to a pattern of tissue reaction to noninfectious stimuli. In the United States, the most common cause is instillation of BCG for treatment of bladder cancer. In this setting, the finding of granulomas in the prostate is of no clinical significance and requires no treatment. Fungal granulomatous prostatitis is typically seen in immunocompromised hosts.*

NODULAR HYPERPLASIA OF PROSTATE

(BENIGN PROSTATIC HYPERTROPHY, SENILE PROSTATIC HYPERTROPHY)

At the age of 30 years, the prostate normally weighs 20 gm.

Benign prostatic hypertrophy (BPH) affects 8% of men in their fourth decade, but the incidence rises to 50% in men in the fifth decade and 75% of men in the eighth decade.

PATHOGENESIS:

No predisposing or protecting factors (other than castration) have been identified. However recent studies relate BPH to an error in the metabolism of dihydrotestosterone

GROSS:

1-The weight of prostate in cases of BPH ranges between 35 g (mild cases) to 800 g or more. The average is around 100 g.

2-BPH affects the central and transitional zones of the prostate around the urethra. This part becomes enlarged and exhibits a nodular cut section. The urethra is compressed. The peripheral prostatic tissue is also compressed. The hyperplastic nodules may have a solid or spongy appearance.

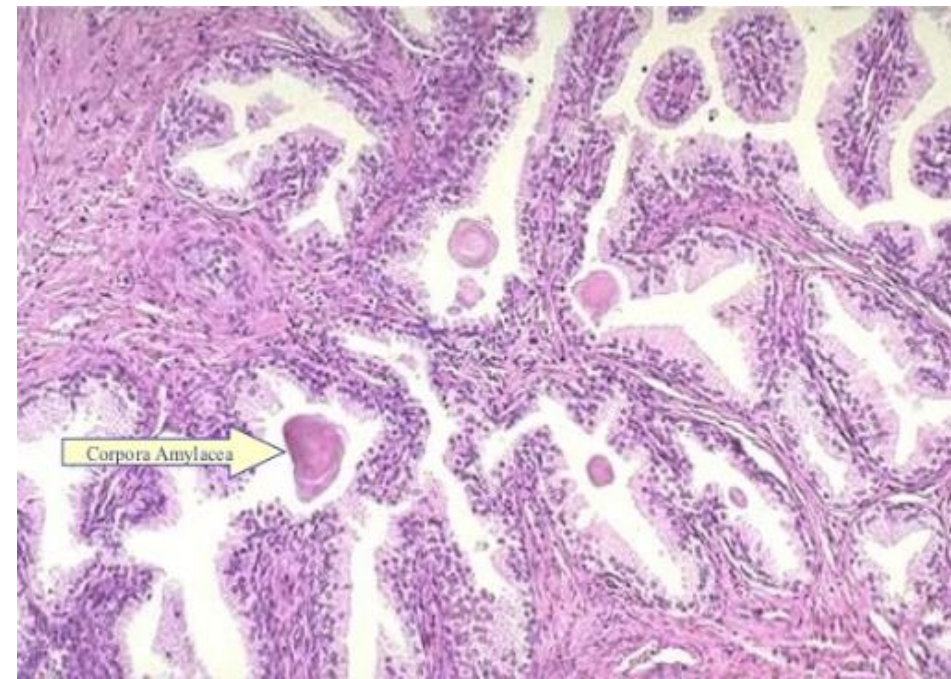
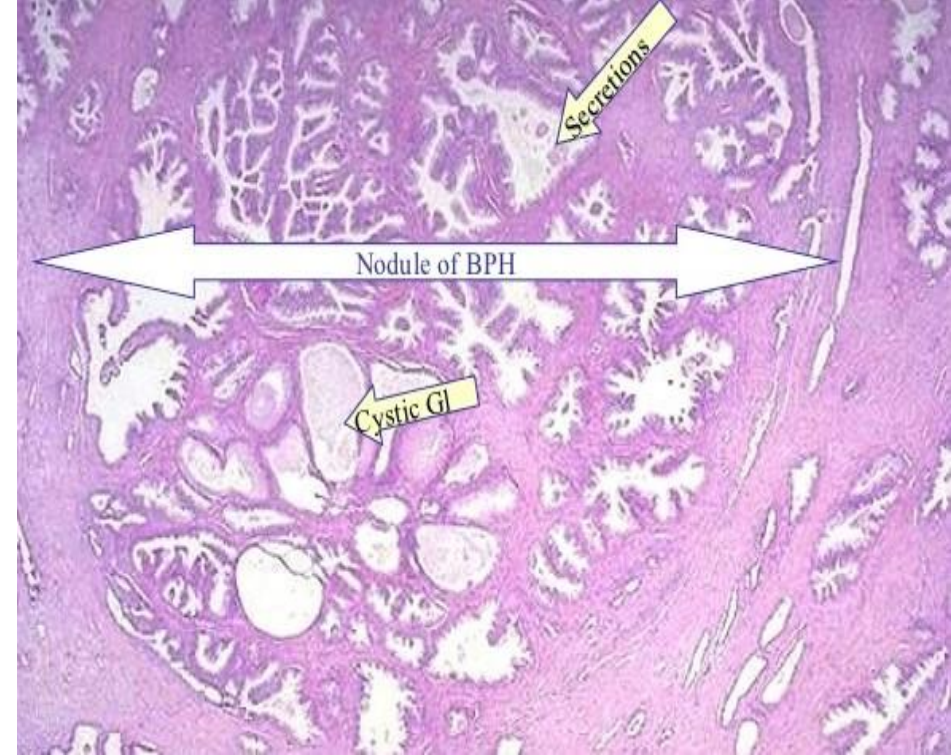


MICROSCOPY:

1-Hyperplastic glands: The glands are enlarged with numerous cystic forms, acini are variable characteristically with a double epithelial lining showing infoldings. The lumens frequently contain inspissated glycoprotein material (corpora amylacea).

2-Hyperplastic fibromuscular stroma with lymphocytic infiltrates.

3.Prostatic infarcts are common in large prostates



EFFECTS AND COMPLICATIONS:

1-Compression of prostatic urethra leads to:

- ✓ Difficult micturition.*
- ✓ Urine retention may occur.*
- ✓ Urinary incontinence may develop.*

2. Urinary tract obstruction

Bladder hypertrophy, dilatation and diverticula, Bilateral hydroureter and hydronephrosis → renal failure.

3. Urinary stasis due to obstruction infection (cystitis, pyelonephritis...etc) → stone formation

CARCINOMA OF THE PROSTATE

cancer prostate is extremely common. The incidence rises steadily with age. Most cases occur after the age of 50 years. Most cases are hormone dependant (androgens), therefore carcinoma does not occur in persons castrated before puberty.

Pathogenesis:

Environmental Factors. It is hypothesized that exposure to carcinogens, estrogens, and oxidants damage prostatic epithelium, setting the stage for acquisition of genetic and epigenetic changes that lead to cancer development.

Inherited Genetic Factors. Studies of twins and families support the existence of important genetic predisposing factors. Men with first-degree relatives affected by the disease have a twofold increased risk, and numerous germline variants have been identified that are associated with risk.

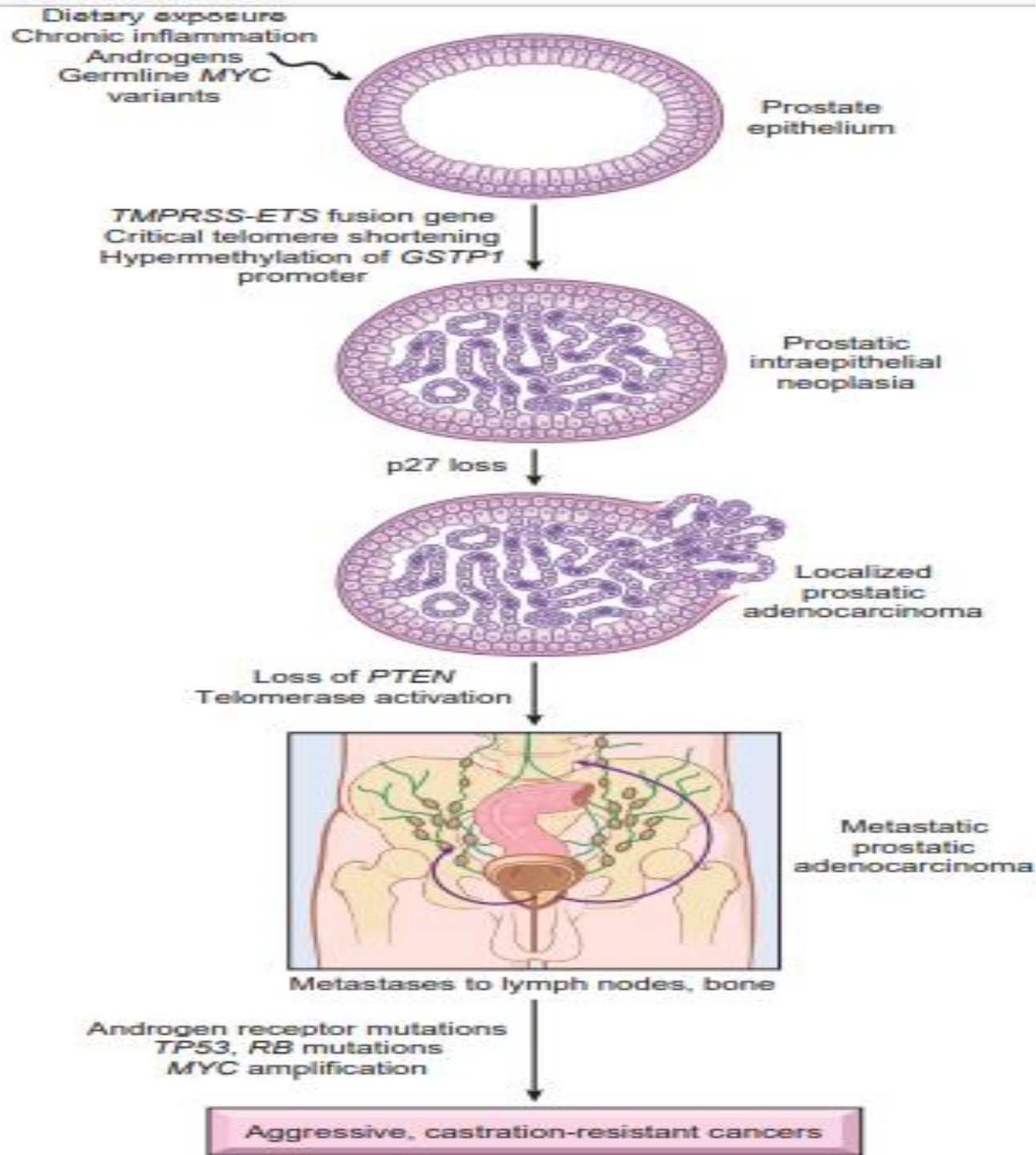
Androgens. Like their normal counterparts, the growth and survival of prostate cancer cells depend on androgens, which bind to the AR and induce the expression of pro-growth and pro-survival genes. The importance of androgens in maintaining the growth and survival of prostate cancer cells can be seen in the therapeutic effect of castration or treatment with antiandrogens, which usually induce disease regression. Unfortunately, most tumors eventually become resistant to androgen blockade.

Acquired Genetic and Epigenetic Alterations. As with other cancers, prostate cancer is caused by genetic and epigenetic alterations that altered the expression of tumor suppressor genes and oncogenes, ultimately leading to acquisition of the hallmarks of cancer . The most common genetic alteration in the prostate is a chromosomal rearrangement that juxtaposes the coding sequence of an ETS family transcription factor gene (most commonly ERG or ETV1) next to the androgen-regulated TMPRSS2 promoter.

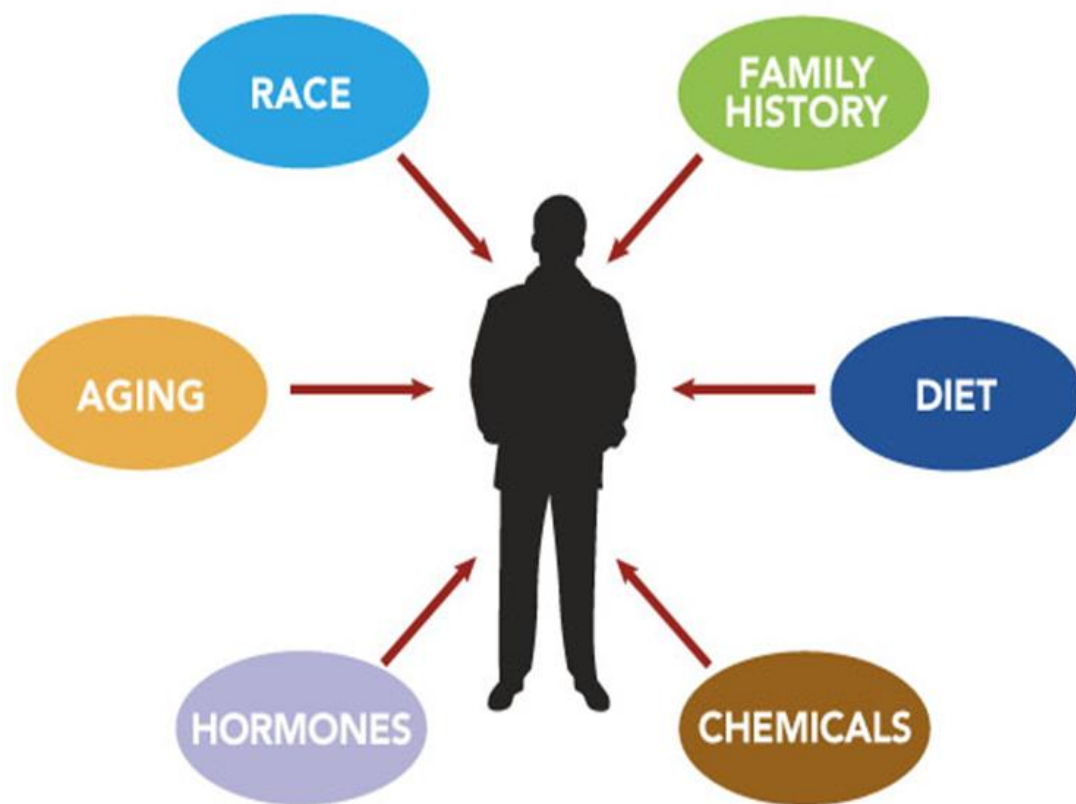
PREMALIGNANT LESIONS

1-Prostatic intraepithelial neoplasia (PIN) was the accepted term for premalignant prostatic lesions. PIN is characterized by hyperplastic glands showing cellular atypia in the form of cellular stratification, nuclear enlargement and nucleolar prominence.

2-Atypical adenomatous hyperplasia (AAH)



RISK FACTORS



AGE	PROBABILITY OF DEVELOPING PROSTATE CANCER
Birth to 49	1 in 441
50 - 59	1 in 57
60 - 69	1 in 21
70 - 79	1 in 12
Birth to Death	1 in 9

57% of prostate cancer cases are diagnosed in men 65 years of age and older.
97% of cases occur in men 50 and older.

Symptoms:

During the early stages of prostate cancer, there are no symptoms. That's why screenings and yearly check-ups are critically important in catching cancer early, before it spreads outside the prostate. In other words, you may not have any symptoms at all and cancer may be detected as a result of a general health check-up where a PSA test and physical exam of the prostate are given.

Many prostate cancer symptoms are very similar to benign prostatic hyperplasia (BHP), prostatitis, erectile dysfunction, or overactive bladder. These include:

- *Strong urge to urinate immediately.*
- *Frequent nighttime urination.*
- *Pain and/or burning when urinating.*
- *Difficulty starting the urinary stream.*
- *A weak urinary stream once it starts.*
- *Dribbling after you're finished.*
- *Pain in the genital and pelvic area.*
- *Pain when ejaculating.*
- *Blood in the urine or semen.*
- *Frequent urinary tract infections.*

Other more serious prostate cancer symptoms may include:

Unexpected weight loss

Pain in the lower back or pelvic area

Anemia

Fatigue

How is Prostate Cancer Diagnosed?

How Do I Get Tested?

A general practitioner or an urologist can perform a full prostate cancer exam. This would usually include a PSA blood test and digital rectal exam, also called a DRE.

A Prostate Specific Antigen (PSA) test measures the level of PSA in the blood. PSA is a substance made by the prostate. The levels of PSA in the blood can be higher in men who have prostate cancer. The PSA level may also be elevated in other conditions.

A Digital Rectal Exam is a test that is done when a doctor or nurse inserts a gloved, lubricated finger into the rectum to estimate the size of the prostate and feel for lumps or other abnormalities.

If the Test Results Are Abnormal?

If the results of early detection tests like the PSA test or the digital rectal exam suggest that you might have prostate cancer, your doctor will conduct further testing. The PSA may be repeated, or you may be sent to a specialist for more tests such as a transrectal ultrasound and a prostate biopsy.

In a prostate biopsy, a tissue sample is taken from your prostate. Cancer can only be diagnosed with a tissue sample

If the Diagnosis Prostate Cancer?

The first thing you should consider doing is to find out about the specifics of your cancer. You should know your stage (location and extent of the tumor) and grade (also known as Gleason score, a measure of the tumor's aggressiveness).

What is a prostate biopsy?

A prostate biopsy removes samples of tissue from the prostate in order to diagnose prostate cancer. This is used after an elevated PSA or abnormal DRE. The tissue is examined by a pathologist to determine if cancer is present. Cancer can only be diagnosed by a tissue sample.

During the biopsy, the doctor will take several 'core' samples from different parts of the prostate using a fine needle. More and more frequently, ultrasound is used to guide the needle and limit damage to any tissue.

What are the side effects of a prostate biopsy?

Infection. The most common risk associated with a prostate biopsy is infection. Rarely, men who have a prostate biopsy develop an infection of the urinary tract or prostate that requires treatment with antibiotics.

Bleeding at the biopsy site. Rectal bleeding is common after a prostate biopsy. If taking blood thinners, talk to the doctor about how to manage them before and after the treatment.

Blood in your semen. It's common to notice red or rust coloring in semen after a prostate biopsy. This indicates blood, and it's not a cause for concern. Blood in semen may persist for a few weeks after the biopsy.

Difficulty urinating. In some men, prostate biopsy can cause difficulty passing urine after the procedure. Rarely, a temporary urinary catheter must be inserted.

Because of these side effects, it can be a good idea to try to eliminate other possible causes of an elevated PSA or abnormal DRE before undergoing a biopsy.

What kind of prostate biopsy are there?

There are three types of prostate biopsies:

Transrectal – The most common biopsy procedure, the doctor – with the guidance of an ultrasound device – inserts needles through the wall of the rectum and into the prostate to take six to twelve samples from different zones of the prostate.

Transurethral – A lighted tiny lens is inserted into the urethra to allow the doctor to see the prostate and then uses a microscopic cutting loop to take samples of tissue.

Transperineal – The doctor makes an incision in the perineum and inserts a needle to take tissue cores of the prostate.

PATHOLOGICAL FEATURES:

Serum markers of prostatic cancer.

1-Prostatic specific antigen (PSA) is very useful marker for the diagnosis and follow up of prostatic adenocarcinoma (Normal serum value: 0-4). It is elevated in cases of prostatic cancer.

2-Acid phosphatase is also secreted by tumor cells and is another useful marker.

Gross: *Most carcinomas of the prostate arise from the peripheral zone, rarely carcinoma arises from the transitional periurethral zone. Cancer prostate vary in size and present as a hard poorly defined yellowish nodule or multifocal nodules (80% multifocal).*

Microscopy: The commonest pattern is prostatic adenocarcinoma. It may be well differentiated, moderately differentiated or poorly differentiated. This grading was modified by Gleason (1977) & this is till now the most accepted method of microscopic grading of prostatic adenocarcinoma.

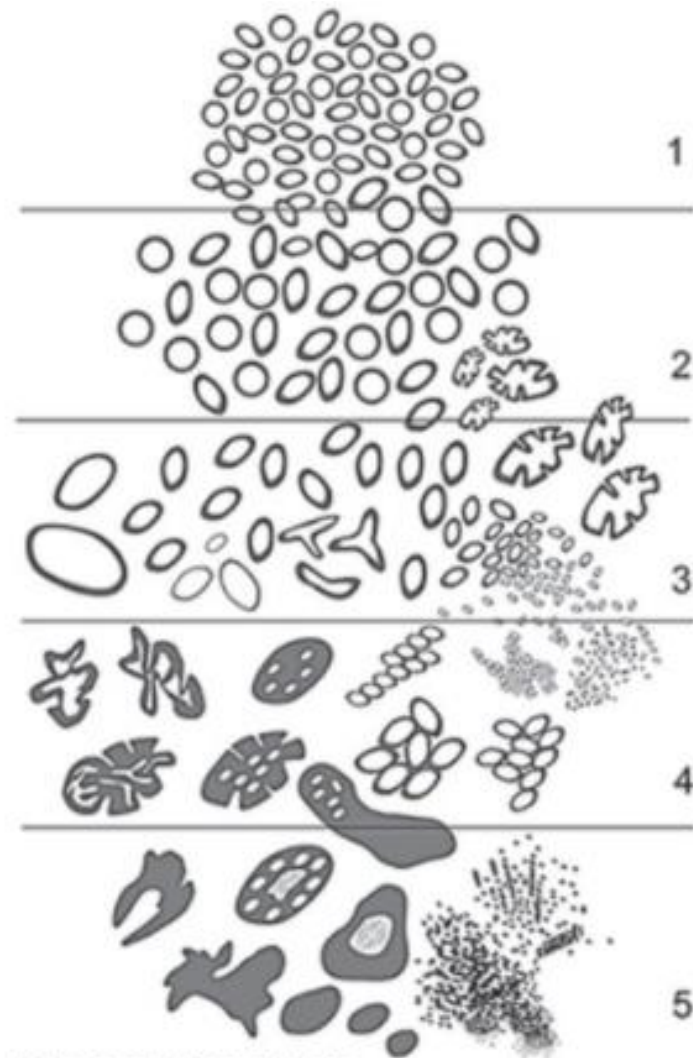
Gleason described five grades of prostatic adenocarcinoma:

1. Gleason grade 1: Closely packed uniform acini lined by a single cell layer.
- 2- Gleason grade 2: Small separate less uniform acini lined by a single cell layer
3. Gleason grade 3: Cribriform or papillary pattern.
4. Gleason grade 4 :Infiltrative fused glands with solitary cells or clear cells
5. Gleason grade 5: Solid sheets, strands or focal tumoral necrosis (comedo pattern).

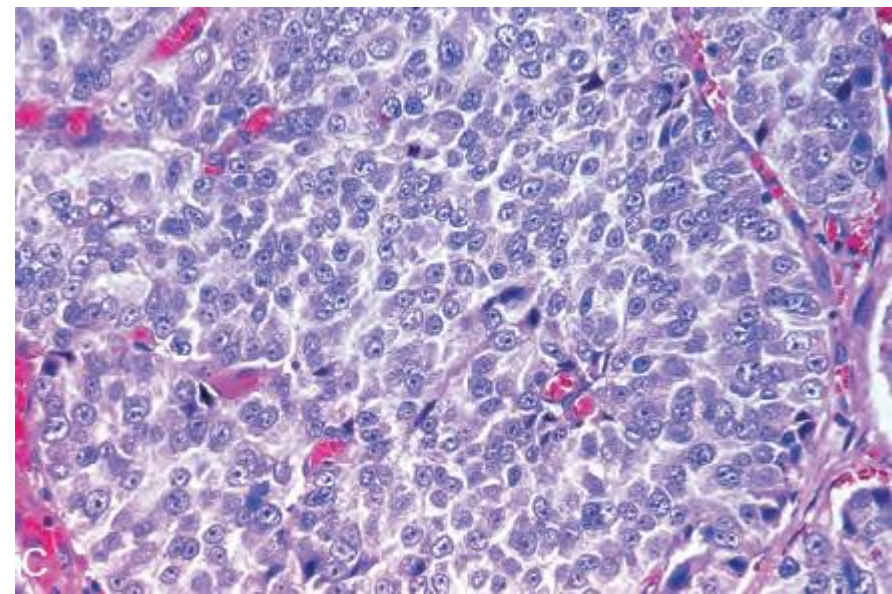
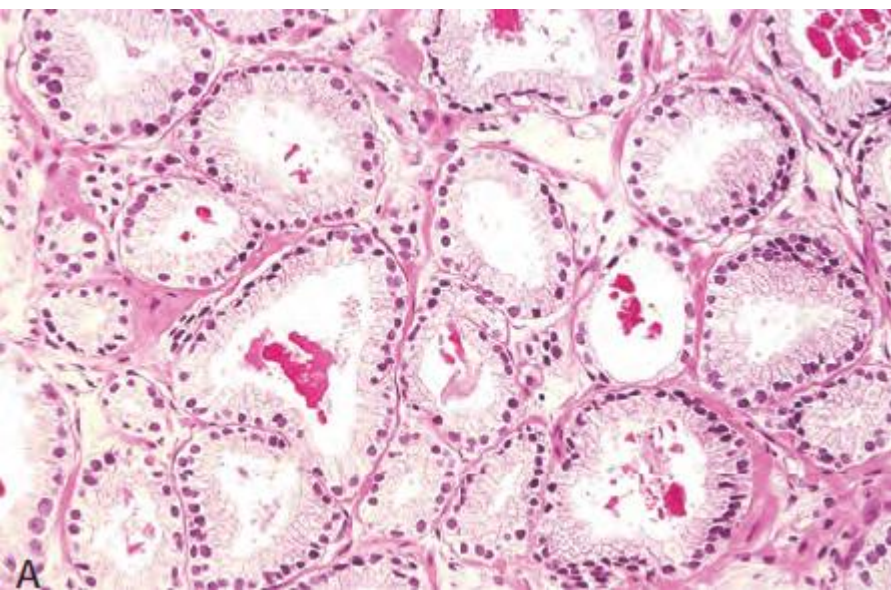
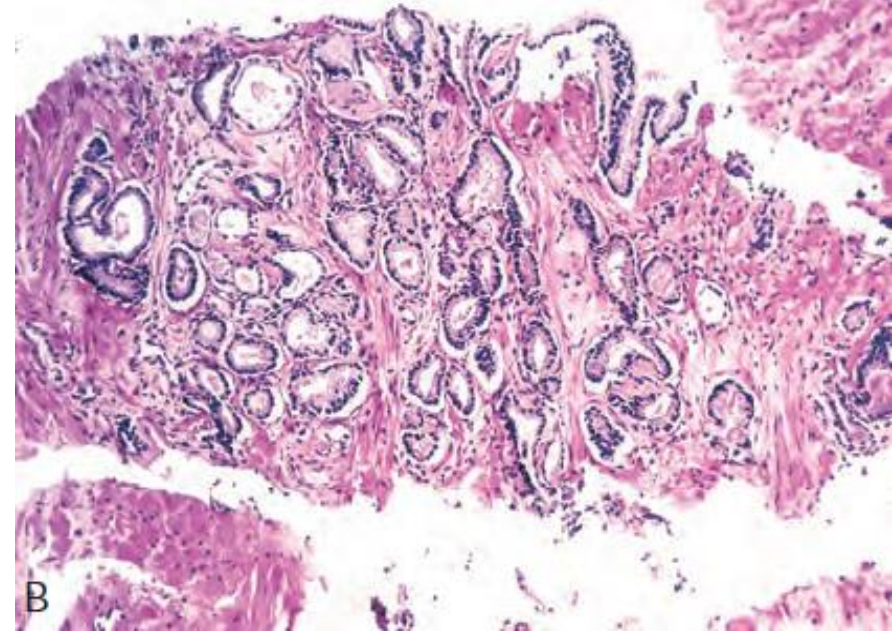
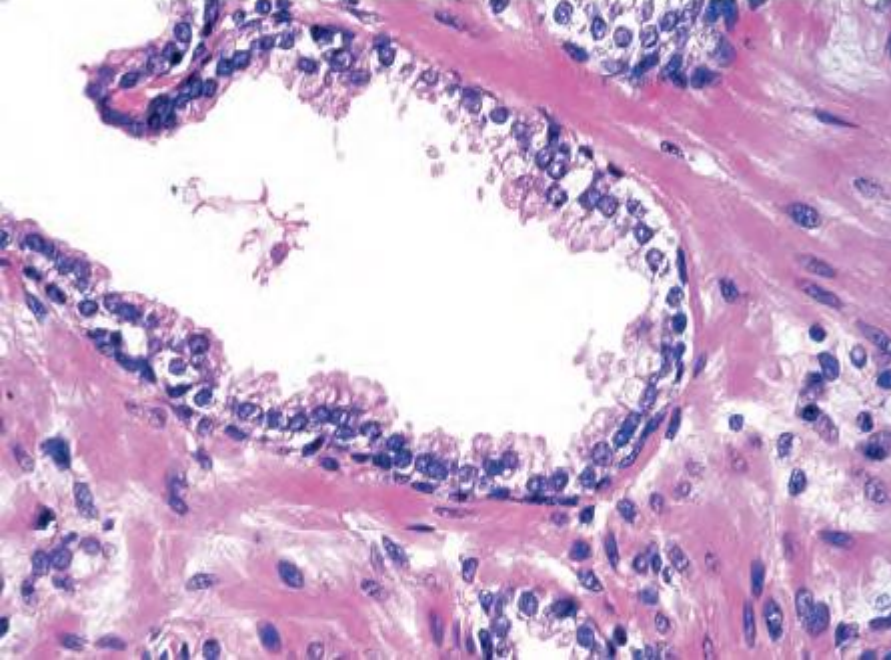
A final Gleason score ; is made by adding the numerical values of the two most predominant grades (e.g. Gleason grades 1+3 = score 4)



D.F. Gleason, MD



Weinzerl | Visual Media
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Gleason scoring system

is interpreted as follows:

1-Low-grade(well differentiated) carcinoma correspond to Gleason score 2-4

2-Medium grade (moderately differentiated) correspond to Gleason score 5-7

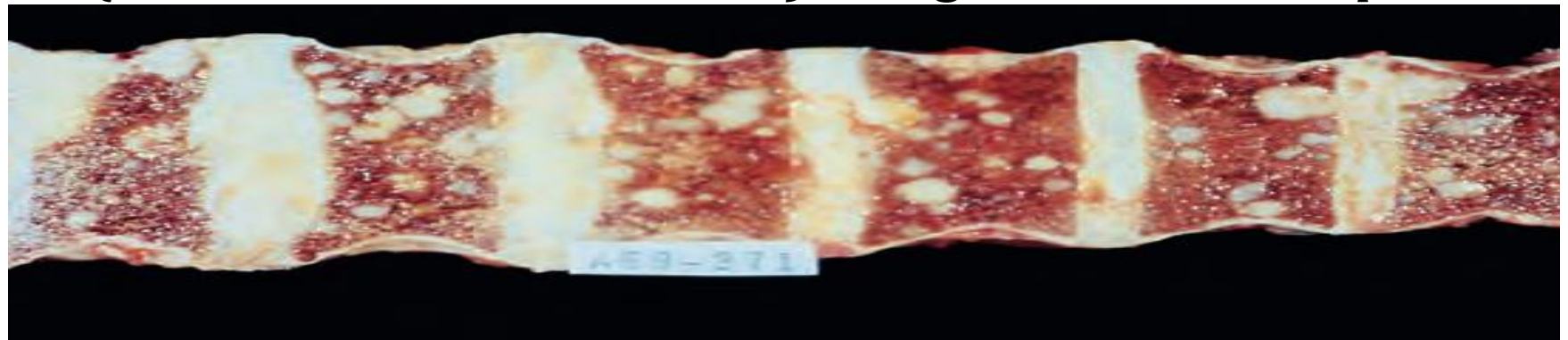
3-High grade (poorly differentiated) carcinoma correspond to Gleason score 8-10

SPREAD:

Direct spread: leads to invasion of prostatic capsule, prostatic urethra, bladder

Lymphatic spread: to pelvic & retroperitoneal lymph nodes, then to supra-

Blood spread to bone (osteoblastic metastases), lungs, skin, brain, penis, liver & adrenal glands



STAGING OF PROSTATIC CARCINOMA:

pT1 Clinically unapparent, tiny tumor.

pT2 Organ confined

pT3 Extraprostatic extension

pT3a Extraprostatic extension (unilateral or bilateral) or microscopic invasion of bladder neck

pT3b Tumor invades seminal vesicles

pT4 Tumor is fixed or invades adjacent structures other than seminal vesicles, such as external sphincter, rectum, bladder, levator muscles, or pelvic wall

The high survival rates for prostate cancer continue over time. The overall 10-year survival rate is 98%, and the 15-year survival rate is 96%. However, for “distant” prostate cancer, or cancer that has spread to bones, organs, or distant lymph nodes, the 5-year survival rate drops from nearly 100% to 30%. “Distant” prostate cancer is more commonly known as advanced prostate cancer.

WHAT CAN I DO?

KNOW YOUR RISK

RISK FACTORS

RACE

FAMILY HISTORY

AGING

DIET

GENE CHANGES

CHEMICALS



GET TESTED

TALK TO YOUR DOCTOR



MAKE A PLAN
TO GET TESTED

MAINTAIN A HEALTHY LIFESTYLE



BE ACTIVE, EAT
NUTRITIOUS MEALS,
MAINTAIN A HEALTHY
WEIGHT



SPREAD THE WORD



TELL A FRIEND