

Male Repro

Benign Prostatic Dz

Normal Anatomy

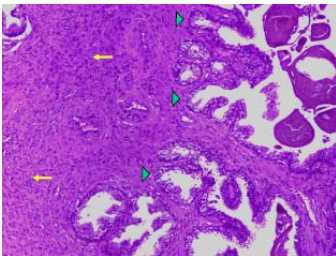
- transition zone → surrounds urethra
- central zone → surrounds ejac ducts
- epithelial cells → secrete PSA & PAP (prostate acid phosphatase)
- basal cells → HMWCK
- if malignant → AMACR/P504S

BPH

- nodules in the transition zone → atrophy of peripheral zone & urethra



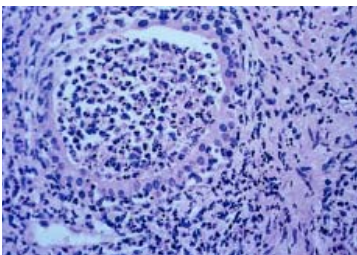
not just change of glands: cellular stroma too



treatment: transurethral resection/block androgen

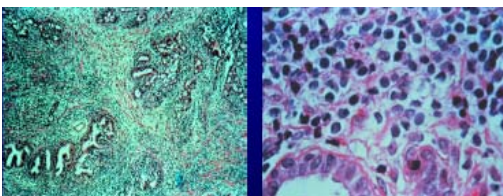
Acute Bacterial Prostatitis

- cause: UTI
- rectal exam: swollen, boggy, tender
- urine: + (80% E.Coli)
- > 10 leukocytes/HPF
- treatment: antibiotics
- Sheets of **neutrophils** w/abscess



Chronic Bacterial Prostatitis

- caused by recurrent UTIs
- first 5 lines same as above, but **lymphocytes**



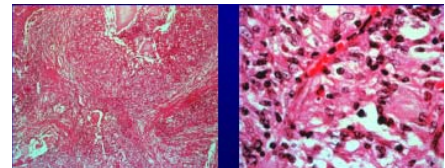
may have atypia

Nonbacterial Prostatitis

- NO UTI
- rectal exam: (usually) normal
- Microscopic: same as chronic bacterial (lymphocytes)
- treatment: NOT antibiotics. Use oxybutynin (relieve SM tension)
- atrophy

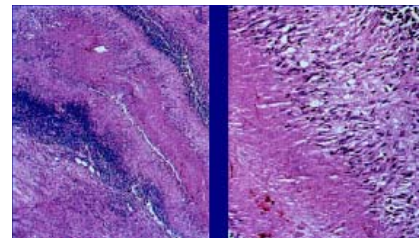
Nonspecific Granulomatous Prostatitis

- obstructive symptoms
- rectal exam: **nodular and hard, looks like carcinoma**
- caused by duct rupture
- microscope: giant cells, lots of cell types, no discrete granulomas



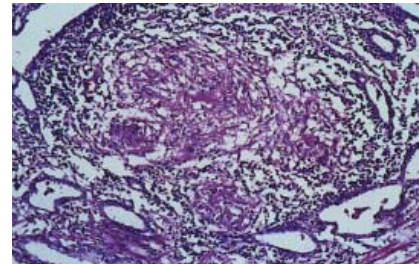
Post-transurethral resection Granulomatous Prostatitis

- may be found from 9 days to 5 years after TURP
- microscope: central zone of fibrinoid necrosis
- rim of palisaded epithelioid histiocytes



Post-BCG Granulomatous Prostatitis

- ↑ PSA → lesions mimic carcinoma**
- microscope: looks like TB granuloma, may be caseating



Male Repro

Prostate Carcinoma

Risk Factors

age

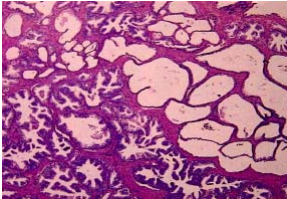
FH

race (lowest in asia, highest in blacks)

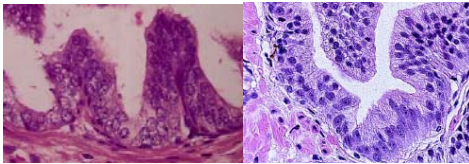
diet (dietary fat plays role -> AMACR plays role in β -oxidation of BCFA's from meat & dairy)

hormones

high grade pin \rightarrow strongly associated w/invasive adenocarcinoma



large glands with basal cells (cancer has no basal cells) & cytological atypia



crowding and stratification of epithelial cells, enlarged nuclei

can have tufted pattern into glands

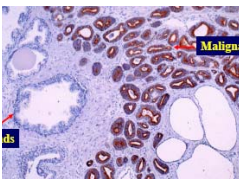
Remember:

- 1) cytological atypia
- 2) nuclear enlargement
- 3) prominent nucleoli

Pathology

majority arise from peripheral zone (75%)

normal glands are LARGE, cancer glands are SMALL



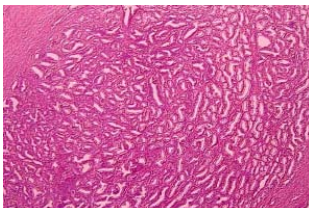
NO basal cells

Tumor Grading: Gleason

Cytological features play no role!

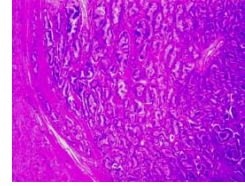
Grade 1:

rare. Just small glands, closely packed.



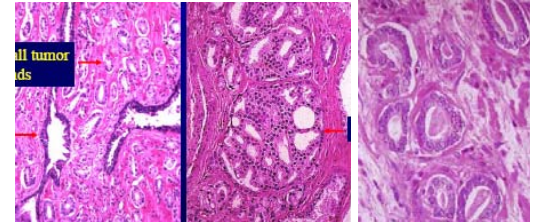
Grade 2:

rare. slightly less uniform glands, sharp edge



Grade 3:

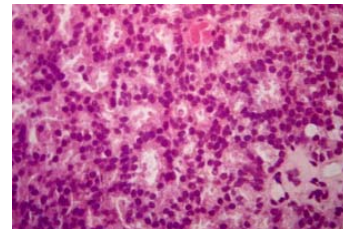
infiltrative, growth between glands, but still some space between glands



cribriform patterns possible

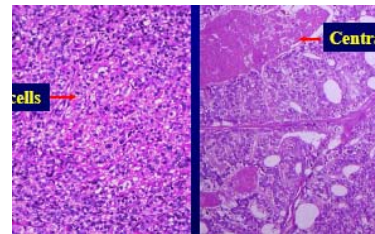
Grade 4:

fused glandular tumor: no stroma between glands



Grade 5:

central necrosis



no glands recognizable

Gleason score: most predominant + second most predominant

Spread & Metastasis

LN (most common):

pelvic \rightarrow retroperitoneal

sometimes supradiaphragmatic/left supraclavicular

Prognosis:

gleason score

tumor stage

PSA

tumor volume

race

Male Repro

Pathogenesis

Chromosome abnormalities: HPC1 gene 1q24-25

P53: chrom 17

BCL-2: inhibits apoptosis

RB: chrom 13

Normal prostatic epithelium → carcinoma: 17p loss common theme

Hormones

AR gene polymorphic. ↓ CAG repeats: more sensitive to androgens

Growth factors:

c-erbB-2 (Her-2/neu)

transmembrane protein w/tyrosine-kinase activity
structural and sequence homology w/EGFR
blocks gap-junction intercellular communication and disrupts cadherin-catenin cell-cell adhesion system.
associated w/aggressive tumors

Oxygen radicals create damage: protect w/ lycopene, vit E & selenium

Inflammation generates ROS → NSAIDS ↓ risk
↑ risk with previous syphilis/gonorrhea

Diagnosis

Screening: for asymptomatic

Diagnostic testing: for symptomatic pts

Methods

DRE: simple but poor for screening, misses 40% of cancers

TRUS: more accurate than DRE, makes biopsy safer

PSA

serine protease product of human kallikrein gene

most in serum bound in complex: α-1-antichymotrypsin

in semen, most is free

cutoff levels: 4 ng/mL, but some variations by age and race

Efficacy:

80% sensitive, 59% specific (↑ false positives)

20-40% of pts with organ-confined carcinoma have normal PSA

% free PSA

lower the free/total PSA ratio → ↑ likelihood of cancer

Used for follow-up: elevated levels indicated recurrent disease

fast rate of change: indicates cancer instead of BPH

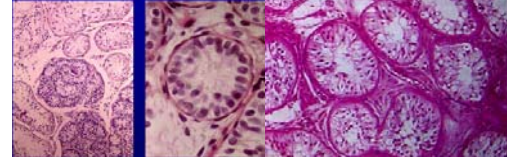
Benign Testicular Dz

Cryptorchidism

failure to descend

risk of developing germ cell tumors if untreated is ↑ 15x

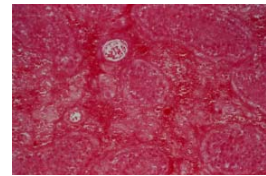
pathology: if not removed, ↓ # germ cells, hyaline thickening of BM, fibrosis



Torsion of testis

severe pain

microscopic: hemorrhagic & necrosis



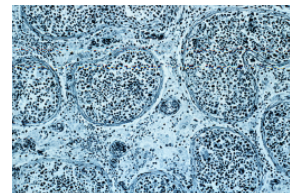
if recurrent, can cause small fibrotic testis

Nonspecific orchidoepididymitis (Acute & Chronic)

Pathogenesis: UTI

< 35: Chlamydia trachomatis; over 35: E. Coli

Acute → neutrophils, abscess



Chronic → lymphocytes

complication: sterility from extensive fibrosis

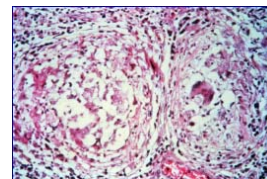
Idiopathic granulomatous orchitis

unknown cause

unilateral

granulomas, no evidence of infection

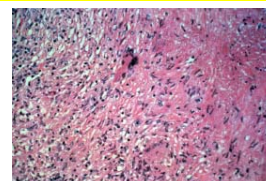
multinucleated giant cells



Tuberculosis orchidoepididymitis

diagnosis: urine culture

granulomas w/ caseating necrosis

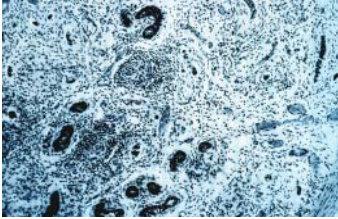


AFB for mycobacteria: positive

Male Repro

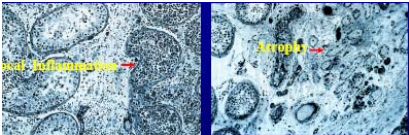
Syphilis orchidoepididymitis

bilateral, painless testicular enlargement
endothelial cell proliferation with endarteritis
lots of nonspecific inflammation
syphilitic gumma: necrosis surrounded by fibrous capsule



Mumps orchidoepididymitis

testicular involvement in kid's mumps is rare
mostly unilateral
focal infection & atrophy



subsequent sterility is rare

early: inflammation, interstitial edema
late: tubular (focal) atrophy
contrast w/undescended testis → diffuse atrophy

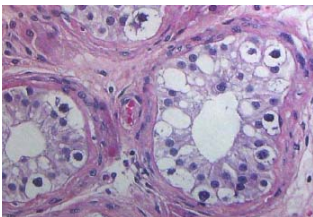
Testicular Tumors

Risk factors for germ cell tumors

cryptorchidism
↑ risk in contralateral testicle too
prior history of GCT
race: more common in whites than blacks
FH
genetics (chromosome 12?)
testicular dysgenesis:
testicular feminization
klinefelter

Intratubular Germ Cell Neoplasia (ITGCN)

malignant germ cells within seminiferous tubules



precursor lesion of invasive GCT → progresses in most cases

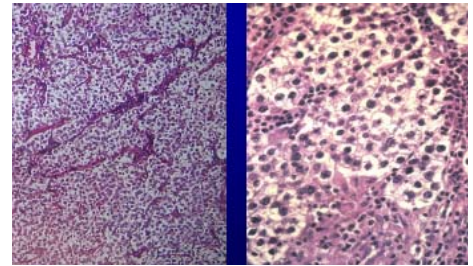
can stain + for PLAP & p53

NOT associated w/spermatocytic seminoma

Classic Seminoma

rare over age 50: age 35-45 typical

Tumor cells (clear cytoplasm) w/Lymphocytes!



lobules/nests with fibrous septa

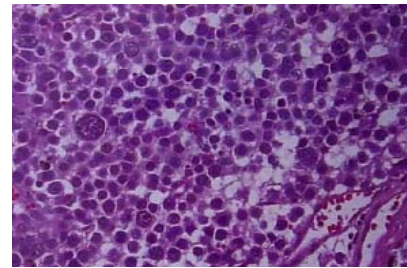
tumor marker: PLAP (placental alkaline phosphatase) in 40%

Spermatocytic Seminomas

old men, over age 65

slow growing, rarely metastasizes, excellent prognosis

3 types of cells



most are medium

a few giant cells

small cells that look like lymphocytes

NO lymphocytic infiltration

NOT associated with cryptorchidism or other GCT's (unlike above)

Embryonal Carcinoma

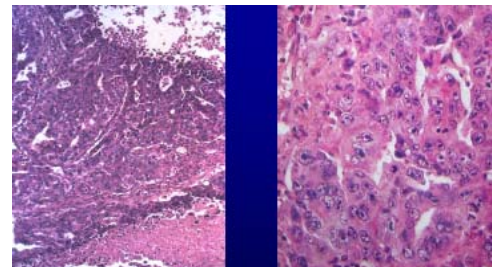
most aggressive GCT: metastasize early

responds well to chemo

ages: 20-30

Ugly looking cells

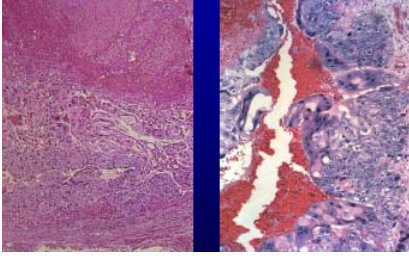
Necrosis



Male Repro

Choriocarcinoma

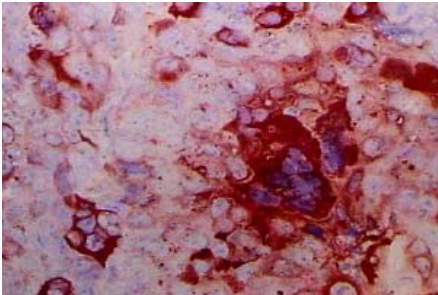
hemorrhage



highly malignant, aggressive behavior:
metastasize early. ↑ lung/brain mets

age: 20-30

usually symptomatic → produce HCG →
gynecomastia



2 types of cells: cyto & syncytiotrophoblasts

Yolk Sac (Endodermal Sinus) Tumor

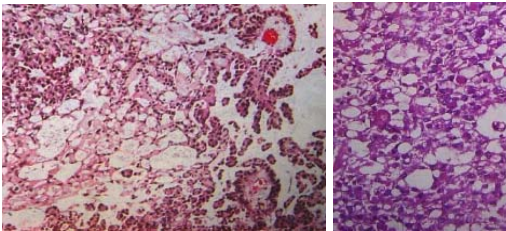
Most common testicular tumor in infants &
children

excellent prognosis in pure form

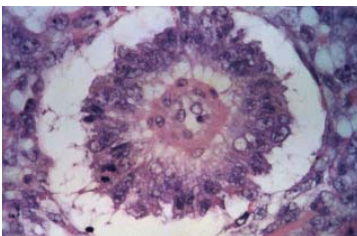
In adults, usually occurs as part of mixed

↑↑ AFP

looks like lipoma (but small cysts are NOT fatty
cells → AFP)

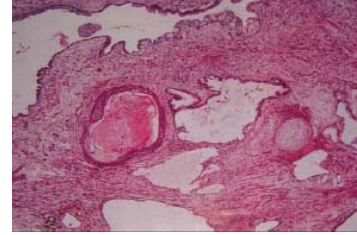


schiller-duval bodies



Mature Teratoma

all 3 germ layers



age: primarily infancy & childhood

in young children: no metastases (benign)

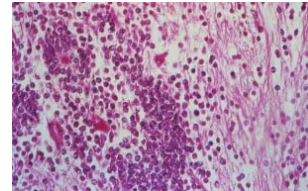
in adult: very aggressive, can met no matter how
differentiated primary tumor is

Immature Teratoma

common component of MGCT

rare in pure form

all 3 germ cell layers but poorly differentiated

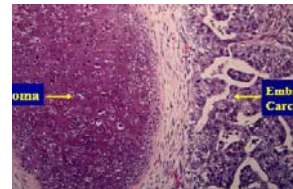


Teratoma with Malignant Transformation

Clear evidence of malignancy in one or more
germ cell layers

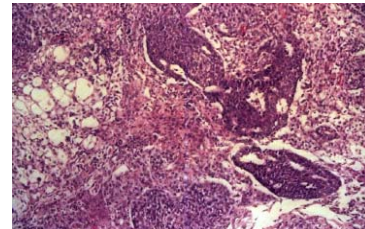
Teratocarcinoma

mature teratoma + embryonal carcinoma



Mixed Malignant GCT

two or more histologic patterns



most common non-seminoma

age: 3rd & 4th decades

Tumor Spread

local: scrotal skin, spermatic cord, epididymic

Lymphatic: periaortic LN, iliac LN

Hematogenous spread: lungs, liver, bone, brain

Markers

PLAP → not very specific

AFP → Yolk Sac

HCG → choriocarcinoma

Penile Squamous Lesions

Definitions

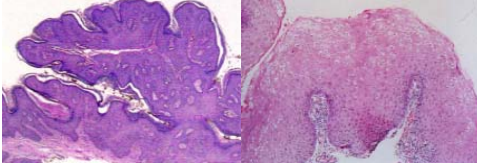
Dysplasia: epithelial cell atypia → premalignant lesion

Carcinoma in Situ → preinvasive lesion

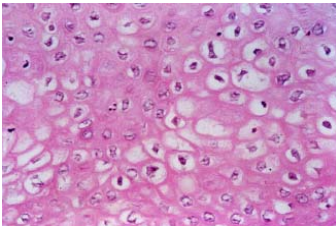
Condylomata Acuminatum

caused by HPV

papillomatous pedunculated or sessile growths with roughened keratotic surface

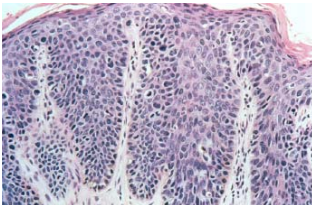


Koilocytes (virus cytopathic effect)



Erythroplasia of Queyrat (EQ)

squamous carcinoma in situ



(same for all 3 SC in situ penile lesions)

usually in glans penis

Erythematous plaque

10% progress to invasive

age: 5th & 6th decade

Bowen's Disease (BD)

squamous carcinoma in situ

usually in sheath

Grayish-white plaque lesion

5-10% progress to invasive

age: 4th & 5th decade

Bowenoid Papulosis (BP)

squamous carcinoma in situ

multiple papular, 2-3 mm lesion

does not progress to invasive, can regress

age: 3rd & 4th decades

Invasive SCC

Risk factors:

1) phimosis (orifice of prepuce too small → circumcision ↓ risk)

2) HPV infection

3) smoking

Microscopic: keratin pearl

