Carcinoma of prostate gland


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**Incidence**

most prevalent cancer in males
second leading cause of male cancer deaths
❏ lifetime risk of a 50 years man for Carcinoma of prostate is 50%, and risk of death is 3%

**Risk factors**

not known (but requires testes as disease is not present in eunuchs)
❏ urban blacks have increased incidence
❏ family history
- 1st degree relative = 2x risk
- 1st and 2nd degree relatives = 9x risk

❏ high dietary fat increases risk by 2x

**Pathology**

❏ adenocarcinoma
- > 95%
- often multifocal

❏ transitional cell carcinoma (4.5%)
- associated with TCC of bladder
- not hormone-responsive

❏ endometrial (rare)
- carcinoma of the utricle

**Anatomy**

❏ 60-70% of nodules arise in the peripheral zone

❏ 10-20% arise in the transition zone

❏ 5-10% arise in the central zone

**Methods of spread**

❏ local invasion
❏ lymphatic spread to regional nodes
- obturator > iliac > presacral /para -aortic

❏ hematogenous dissemination occurs early
❏ bony metastasis to axial skeleton is very common (osteoblastic)
❏ soft tissue metastasis is less common with liver, lung and adrenal metastases occurring most frequently

❑ obstructive and irritative symptoms uncommon without spread
❑ suspect with prostatism, incontinence +/- back pain
❑ hard irregular nodule or diffuse dense induration involving one or both lobes is noted on DRE
❑ differential diagnosis of a prostatic nodule
- prostate cancer (30%)
- benign prostatic hyperplasia
- prostatitis
- prostatic infarct
- prostatic calculus
- tuberculous prostatitis

**Clinical features**

Type I: occult type
Type II: LUTS
Type III: acute or chronic retention
Type IV: symptoms due to metastasis

**DRE**

- PSA can be falsely elevated
- DRE does not palpate entire prostate gland
- Abnormal: nodules, hard spots, soft spots, enlarged

DRE

BPH
- Size may be quite big
- Consistency: firm & elastic
- Surface: smooth
- The midline sulcus between the two lateral lobes is well felt
- The seminal vesicles feel normal
- The gap between the enlarged prostate & the lateral pelvic wall is clear on both sides
- The rectal mucous membrane moves freely over the enlarged prostate

Carcinoma of prostate
- Size is usually not very big
- Consistency: hard

- Surface: irregular & nodular
- The sulcus is usually obliterated

- The seminal vesicles maybe invaded by tumor & feel hard & irregular
- This gap is obliterated by invasion of the cancer

- The rectal mucous membrane is adherent & can't be moved over the prostate

**Diagnosis**

❑ digital rectal exam (DRE)
❑ PSA (prostate specific antigen) elevated in the majority of patients with CaP
❑ transrectal ultrasound (TRUS) —> size and local staging
❑ TRUS-guided needle biopsy
❑ incidental finding on TURP
❑ bone scan may be omitted in untreated CaP with PSA < 10 ng/ml
❑ lymphangiogram and CT scanning to assess metastases

**Prostate specific antigen (PSA)**

- produced by prostatic epithelium
    - serine protease which liquefies semen coagulum which forms after ejaculation
- normally tiny amounts in serum
- elevated levels can occur in localised or metastatic prostate CA
- but levels can increase in other conditions of the prostate and in ~ 20% CA cases PSA may be normal, so no value as screening test

Transrectal sonogram of the prostate.

Looking up from the feet of a patient toward his head

**Staging (TNM 1997)**

❑ T1: clinically undetectable tumour, normal DRE and TRUS
❑ T2: confined to prostate
❑ T3: tumour extends through prostate capsule
❑ T4: tumour invades adjacent structures (besides seminal vesicles)
❑ N: spread to regional lymph nodes
❑ M: distant metastasis
❑ tumour grade (Gleason score out of 10) is also important
    • 1-4 = well differentiated
    • 5-6 = moderately differentiated

- 8-10 = poorly differentiated

**Watchful Waiting**

- aka Active Surveillance
- PSA every 6 months
- Slow growing cancer
- Delay for other diseases to improve
- Comorbidities prevent other treatment
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**Prostatectomy**

- Perineal, Retropubic, Suprapubic – depends on patient anatomy and surgical history
    - Nerve-sparing
    - Robotic

**Hormone Therapy**

- LHRH analogs
    - Lupron, Zoladex
- Androgen blockades
    - Casodex, Eulexin, Nilandrone
- Estrogen therapy (DES)
- NOT orchidectomy

**Treatment for Recurrence/Metastasis**

- Hormones
- Orchidectomy
- Radiation to metastasis
- Radioisotopes
    - strontium-89 (Metastron)
    - samarium-153 (Quadramet)
- Chemotherapy
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**Treatment**

❑ T1 (small well-differentiated CaP are associated with slow growth rate)
- if young consider radical prostatectomy, brachytherapy or radiation
- follow in older population (cancer death rate up to 10%)

❑ T2
- radical prostatectomy or radiation (70-85% survival at 10 years) or brachytherapy

❑ T3, T4
- staging lymphadenectomy and radiation or hormonal treatment

**N>0 or M>0**

- requires hormonal therapy/palliative radiotherapy to metastasis
- bilateral orchiectomy - removes 90% of testosterone
- LHRH agonists (e.g. leuprolide (Lupron), goserelin (Zoladex))
  - initially stimulates LH, increasing testosterone and causing ”flare”
  - later causing low testosterone
  - side effects include “hot flashes”
- estrogens (e.g. DES)
  - inhibits LH, and cytotoxic effect on tumour cells
  - increase risk of cardiovascular side effects

**N>0 or M>0**

- antiandrogens
  - steroidal (e.g. cyproterone acetate) and non-steroidal (e.g. flutamide) both compete with dihydrotestosterone (DHT) for cytosolic receptors
  - testosterone levels do not decrease (and may increase), so potency may be preserved
  - inhibitors of steroidogenesis (e.g. ketoconazole, spironolactone)
    - block multiple enzymes in the steroid pathway, including adrenal androgens
  - greater androgen blockade can be achieved by combining an antiandrogen with LHRH agonist or orchiectomy
  - local irradiation of painful secondaries or half-body irradiation

**Prognosis**

❑ **Stage T1-T2: excellent, compatible with normal life expectancy**
❑ **Stage T3-T4: 40-70 % survival at 10 years**
❑ **Stage N+ and/or M+: 40% survival at 5 years**
❑ **prognostic factors: tumour stage, tumour grade, PSA value**