GU Oncology

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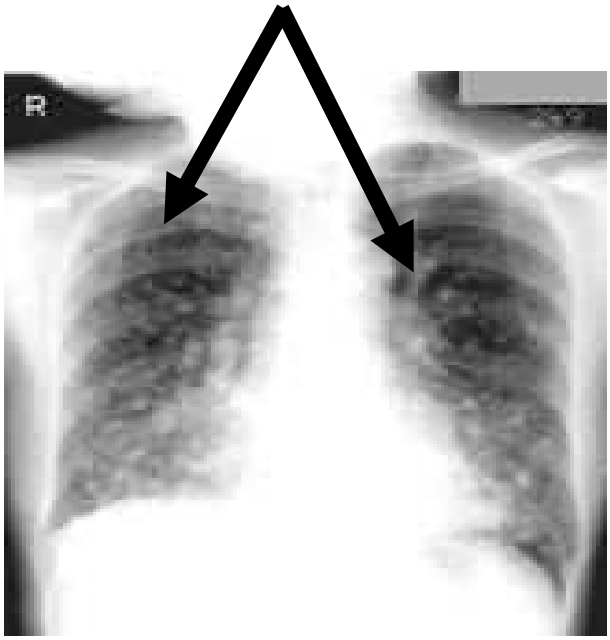
Renal Tumors

- They are usually malignant. Benign tumours of the kidney are rare
- All renal neoplasms (masses) should be regarded as potentially malignant till proven otherwise.
- Renal cell carcinomas arise from the proximal tubule cells.
- Male : female ratio is approximately 2:1
- Increased incidence seen in von Hippel-Lindau syndrome.(a familial disease).
- Renal cancer is Very peculiar it can grow pathologically into the renal sinus, and it can grow into the renal vein → IVC → Heart (without even causing metastasis to anywhere else).
- Blood born spread can result in 'cannon ball' pulmonary metastases



Cannon Ball:

Multiple nodules, within both fields of the lungs

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Clinical features

- 10% present with classic triad of: 1haematuria

  2. Loin pain

  3. Mass

In year 2008 this is not true, this presentation is less common. And the reason is that today if you get a hiccup or slight abdominal pain and go to any doctor. They would order an ultrasound or CT scans for the simplest of reasons. So, most patients are caught earlier as ((**incidental findings**)).If the pnt. presents w/ the triad it's usually too late, and the pnt is already t3 or t4

- Paraneoplastic syndrome:

  Renal cancer is very peculiar about paraneoplastic syndrome.

  - Why does it occur?

  - Bcuz the tubular cells involving cancer bcome hyperfunctioning.

  - How does it present?

  -It can present as: - pyrexia of unknown origin.

  - Hypertension.

  - Anemia.

  - Polycythaemia (due to erythropoietin production).

  - Hypercalcaemia (due to production of a PTH-like hormone).

Pnt. Can present with: Stople syndrome(sorry I'm not sure 'bout the name). It's a liver disfunction with coaulopathy. The pnt starts bleeding from everywhere simply because of a renal tumor.

-How is paraneoplastic syndrome treated?

-By removing the primary tumor.

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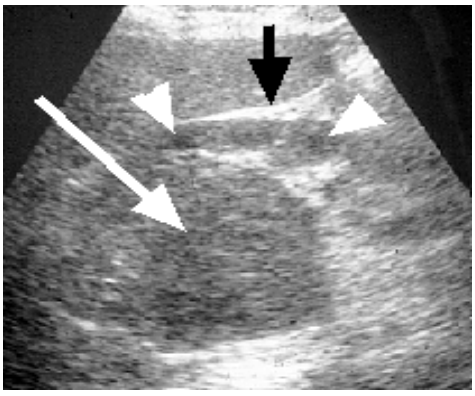
Investigations

Golden Rule in Any Type of Cancer: 1. Diagnose.

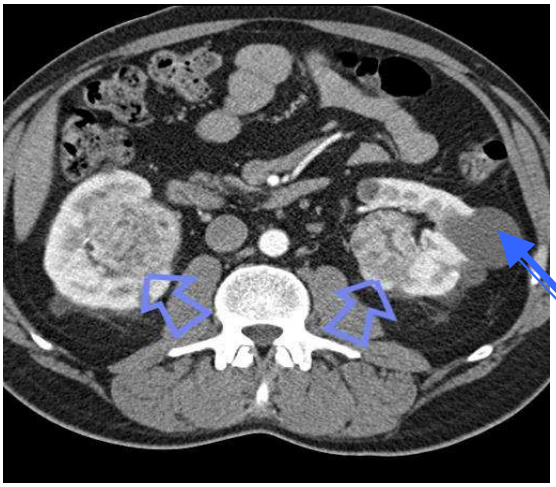
2. Stage.

- Diagnosis can often be confirmed by renal ultrasound
- CT scanning allows assessment of renal vein and caval spread (It's a MUST in staging renal carcinoma).
- If you're suspecting a clot in the renal vein, IVC (extends above the diaphragm) or even in the heart, you should do an echocardiogram, doppler of the vena cava.



Ultrasound to the kidney:

The white arrow is pointing to a mass (this mass is typically picked up on US in case of renal cell carcinoma)



CT scan of a pnt w/ VHL disease

They usually end up with bilateral renal cell carcinoma.

This pnt here the arrows are pointing to tumors growing into the kidneys

Simple renal cysts have 0% chance of malignant transformation. So we have to differentiate. (Remember radiology ;))

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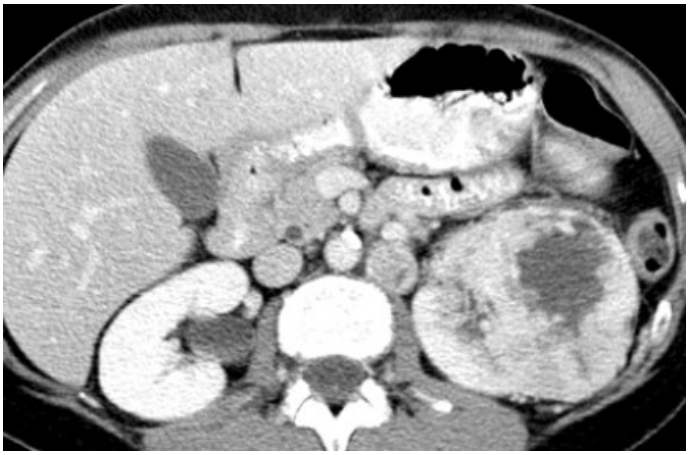

Other example of a renal mass

There is necrosis in the centre of the kidney cuz the kidney lost its blood supply

This is typical cancer appearance

Management

- Unless extensive metastatic disease it invariably involves surgery.
- Surgical option usually involves a radical nephrectomy .
- Kidney approached through either a transabdominal or loin incision.
- Renal vein ligated early to reduce tumour propagation.
- Kidney and adjacent tissue (adrenal, perinephric fat) excised.

Golden rule: V.V.imp!!!

Renal Cancer DOES NOT respond to chemotherapy. It DOES NOT respond to radiation therapy it ONLY responds to SURGERY and it only respondes to surgery if it's a localized disease. If it's a metastatic disease it's TOO LATE ☹)

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Important Notes in Kidney CA Management:

- Renal CA has a narrow window of opportunity, if u diagnose the pnt early your gonna do a nephrectomy within the next month or 2, cuz that's his only chance of cure (and it's a good chance of cure 96 % 5 year survival rate)once the disease is no longer localized it drops to 30%
- Until two yrs we had nothing else to offer the pnt, there was interleukins and interferons which gave 10% improvement.
- Nowadays we have TKI: tyrosine kinase inhibitors. They just came out in 2006. They are designed to be targeted therapies. They inhibit the growth of the tumor by inhibiting the vascular endothelial growth factor (VEGF).
- Very soon your gonna hear of someone that won the nobel prize for discovering the super medication for kidney CA they normally use them for kindney CA liver CA and other cancers as well. They're not chemotherapeutic agents, they have little side effects. However in case of kidney CA they've gave us significant results of 1 year survival rate this is something unheard of in kidney Ca as apposed to the usual 5 or 6 mnth survival rate in metastatic kidney CA. these are hopefull medications which we don't know much about right now and how to combine them with surgery.
- But these don't change the fact that **the only chance for cure in a pnt w/ renal CA is surgery**
- Unless extensive metastatic disease treatment invariably involves surgery.
- Surgical option usually involves a radical nephrectomy **(gold standard)**(taking out the entire kidney , adrenal glands, surrounding Gerota's fascia and the lymph nodes in the hila of the kidney).

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There are two ways to approach nephrectomy

1. the classic approach:

- Going through a flank incision (shark bite incision)

or thoracoabdominal incision. where we crack the chest open then take it down to the abdomen. (because the kidney is a very deep structure in the abdomen, to get there it's quite difficult).

- Very painful.

- Hospitalization for a week-10 days.

2. Laparoscopic nephrectomy:

-discribed ten yrs ago by Clayman in California, and has adapted very quicly.

-a small appendectomy scar.

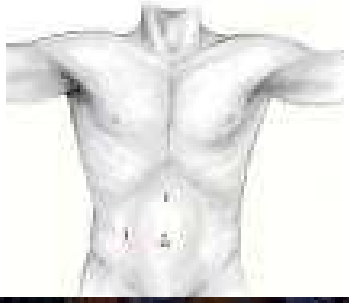
- Hospitalized for 2 days.

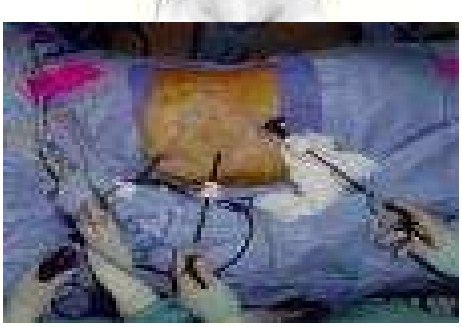
- Pain is much less.

- Back to work in 2 or 3 weeks.

- much less morbid than a radical nephrectomy (an open one).

- the only cases we don't use this procedure in are the very big tumors or those who require partial nephrectomy.





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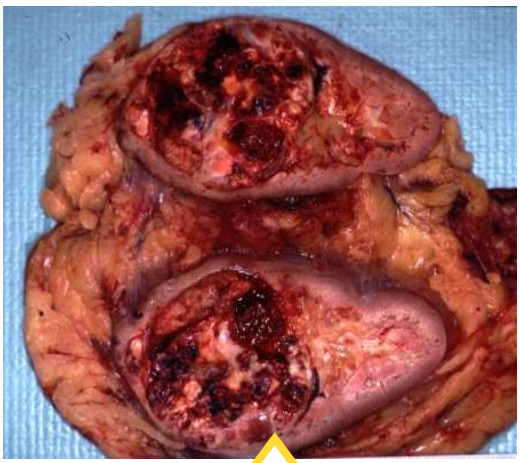
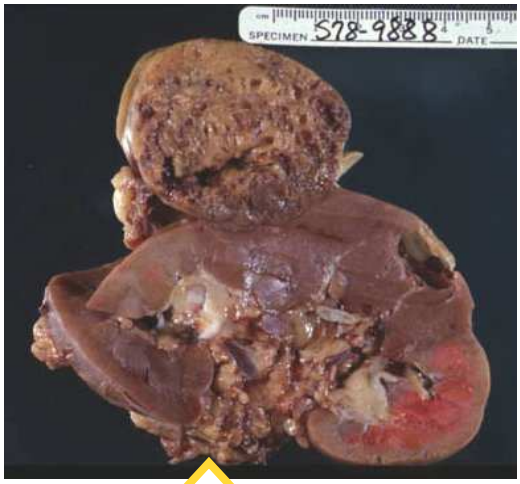


Tumor in the middle of the kidney

*This is an example of a kidney sliced in half after surgery. And you can see the tumor here involving the upper pole of the kidney, areas of hemorrhage, areas of necrosis.

* In case of a solitary kidney (only one functioning kidney): nephrectomy is the only option.



Exophillic tumor growing on the kidney

* In case of a solitary kidney: partial nephrectomy can be performed (not just tumor excision cuz we'r afraid of metastasis).

Partial nephrectomy

It started out w/ pnts that had 1 kidney, then we started doing it on pnts who have 2 kidneys but who have exophillic tumors in upper or lower poles (it's an easy surgery without violating the parenchyma of the hilum of the kidney, and it's been noticed that pnts with tumors that are 4 cm or less, and are at a good location, can have an equal survival rate if you do a partial or radical nephrectomy ...this is very useful in pnts who have diabetes, long standing HTN or who are prone to develop renal failure sometime in their life, so we spare their kidneys. It's called (nephron sparing surgery).

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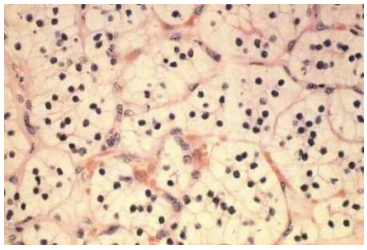
There are 5 types of Renal Cell Carcinoma (RCC is a very broad term):

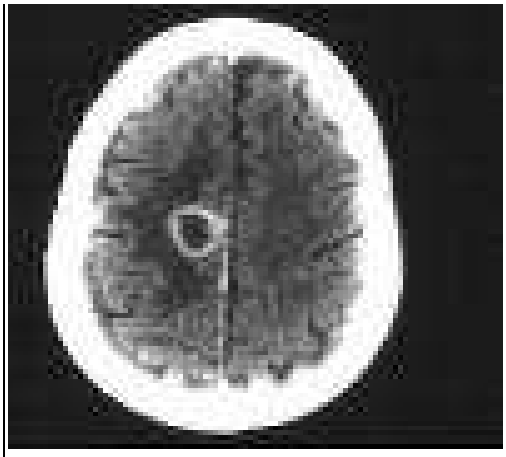
* The most common type is (Clear Cell Carcinoma).
(It's Called CCC cuz the cells in cytology appear clear and vaculated)



Kidney Ca unlike other types of tumors CAN NOT be diagnosed by biopsy (30 – 40% chance error)

Rx in metastatic tumors

- Lymph node dissection of no proven therapeutic benefit, it's purly diagnostic.
- Solitary (e.g. lung metastases) can occasionally be resected **(if there is focal metastases to CNS, Lung or liver it's worth resecting with the kidney tumor cuz there is a 60% chance of cure).**
- Radiotherapy and chemotherapy have **No** role



- This is a pnt with metastasis to the entire body. And reached up to the brain.
- This pnt had surgery in addition to a nephrectomy and 5 years later he's alive and well.

Bladder Tumors Pathology

Of all bladder carcinomas:

- 90% are transitional cell carcinomas (urethelial tumors), arises from the lining of the bladder, the ureter and the kidney itself.
- 5% are squamous carcinoma
- 2% are adenocarcinomas
- TCCs should be regarded a 'field change' disease with a spectrum of aggression
- 80% of TCCs are superficial and well differentiated
- Only 20% progress to muscle invasion and once it

  Does it's bad news. It'll spread everywhere.
- Associated with good prognosis
- 20% of TCCs are high-grade and muscle invasive
- of the 20% with muscle invasion: 50% have muscle invasion at time of presentation. The other 50% will have it later on.
- Associated with poor prognosis

'Field Change' disease

Means that if someone has developed a renal tumor in the bladder it is very likely that the rest of the urethelia is also diseased it just hasn't presented a tumor yet

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Etiological factors

If a pnt presents to you with a history of hematurea the first Q you gotta ask is how many packs for how many years??

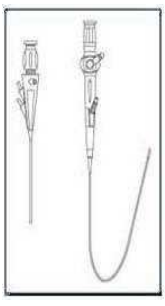
- Smoking, smoking, smoking!! is the most imp.!
- Occupational exposure.
- 20% of transitional cell carcinomas are believed to result from occupational factors
- Chemical implicated - aniline dyes, chlorinated hydrocarbons
- Analgesic abuse e.g. phenacitin
- Pelvic irradiation - for carcinoma of the cervix
- *Schistosoma haematobium* associated with increased risk of squamous carcinoma (most common cause). Schistosoma will cause renal cell carcinoma, but more commonly urothilal carcinoma.

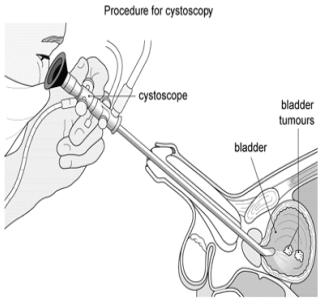
BT Presentation

- 80% present with painless hematuria
- Also present with treatment-resistant infection or bladder irritability and sterile pyuria

Investigation of painless haematuria

- **cystoscopy** (Gold Standard): Flexible or Radio
- Urinalysis
- Ultrasound - bladder and kidneys
- KUB - to exclude urinary tract calcification





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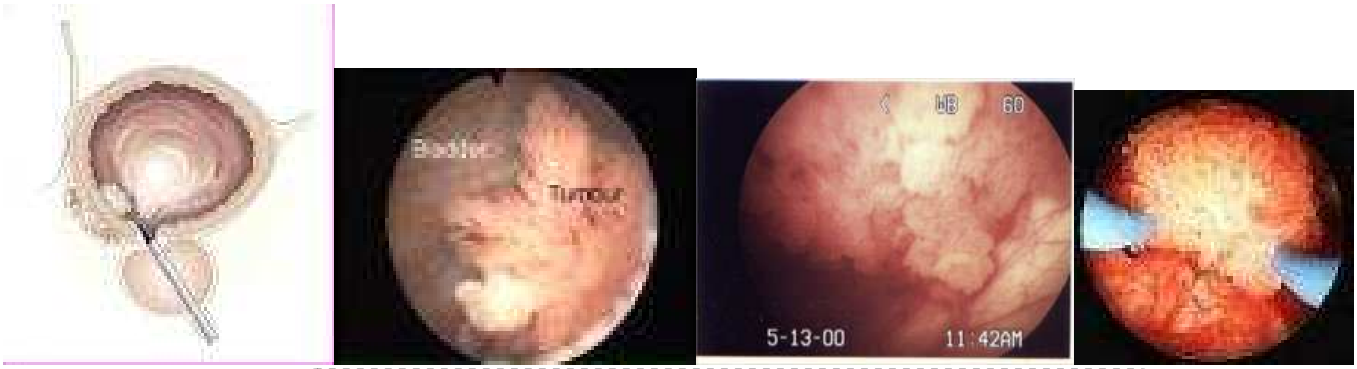
- Urine Cytology (v. imp. In bladder tumors).
- Consider IVU if no pathology identified



The tumor usually looks like a cauliflower (it's papillary). And the more papillary it is the more likely it's gonna be a superficial disease. The more solid it looks, the worse the likely hood of a muscle invasive disease.

After you diagnose a bladder tumor you either biopsy it. Or the more proper thing to do is to a trans urethral transection of the bladder tumor (TURBT). We use a resectoscope, which is a special cystoscope which has a loop at the end of it, which goes behind the tumor, and has an electrical voltage which helps us scrape the tumor off the bladder.

Kidney cancer is the only urogenital cancer that does not produce markers that help us in diagnosing!

What does this dog have to do with bladder tumors :S ??

Bladder tumors secrete various substances in urine, and dogs can smell it!!!

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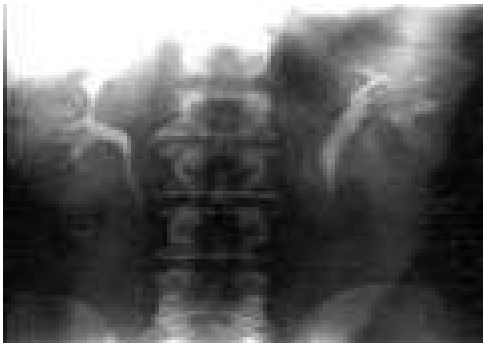


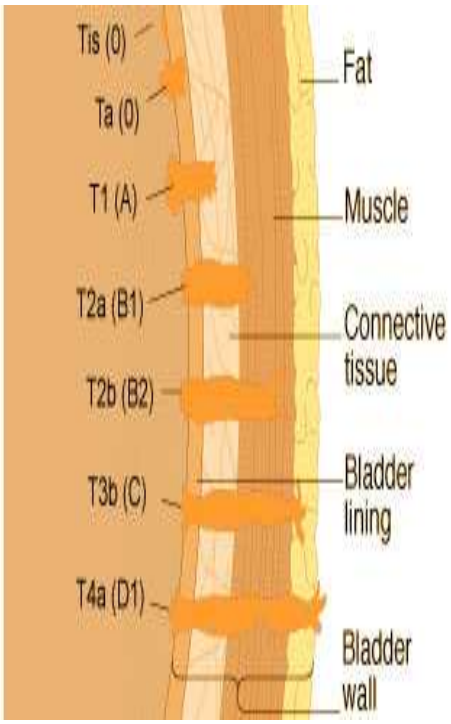
The left kidney has a filling defect in the collecting system. And this is an example if you can get urethelial not only in the bladder, but also in the renal pelvis and ureter

Pathological staging

Requires bladder muscle to be included in specimen.

- Staged according to depth of tumour Invasion:
- Tis In-situ disease
- Ta Epithelium only
- T1 Lamina propria invasion
- T2 Superficial muscle invasion
- T3a Deep muscle invasion
- T3b Perivesical fat invasion
- T4 Prostate or contiguous muscle

Basically what you have to know is that the bladder is composed of layers (memorize them from the pic)



If the disease is only in the urethilium or only the lamina propria: it's called superficial disease, low grade disease which will recur its rarely gonna progress.**Rx:**usually TURBT with surveillance cystoscopy, we may add immuno therapy.**If the disease is down to the mucsle and through mucsle or to the fat:**this is a muscle invasive disease,and this is bad prognosis disease.**Rx:**radical cystectomy.

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Grade of tumour

- G1 Well differentiated
- G2 Moderately well differentiated
- G3 Poorly differentiated

Carcinoma in-situ

- Carcinoma-*in-situ* is an aggressive disease
- Often associated with **positive cytology**
- 50% patients progress to muscle invasion
- Consider immunotherapy
- If fails patient may need radical cystectomy

Radical cystectomy: remove the entire bladder of the pnt. w/ the prostate and seminal vesicles for the male, the anterior vaginal wall and a hesterectomy for females.

So what happens to the ureters in radical cytectomy?? Where does the urine go??
1) ileal conduit 2) neo-bladder (the way they're done is written down).

Treatment of bladder carcinomas

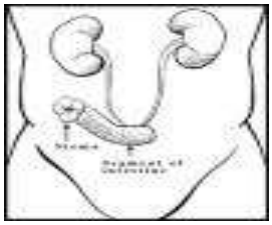
Superficial TCC

- Requires transurethral resection and regular cystoscopic follow-up
- Consider prophylactic chemotherapy if risk factor for recurrence or invasion (e.g. high grade)
- Consider immunotherapy
- BCG = attenuated strain of *Mycobacterium bovis*
- Reduces risk of recurrence and progression

- 50-70% response rate recorded
- Occasionally associated with development of systemic mycobacterial infection.

Rx: Invasive TCC

- Radical cystectomy has an operative mortality of about 5%
- Urinary diversion achieved by:
  - Ileal conduit : (remove the bladder+ attach ureter with ileum then bypass ileum to the outside)(classical, easier, less complications).
  - Neo-bladder: (remove the bladder+ attach ileum with ueter then attach ileum with distal urethra (so the ileum acts as an artificial bladder)–this has many complications such as: there are no muscles in the ileum so there won't be full emptying,so the pnt.'ll have to catheterize himself a lot of the time to empty the urine.) (usually done to young pnts who don't want to carry around a bag of urine. ya3ni mostly 4 cosmetic purposes).



- Local recurrence rates after surgery are approximately 15% and after radiotherapy alone 50%
- Pre-operative radiotherapy is no better than surgery alone
- Adjuvant chemotherapy may have a role

Bladder tumor: and you can see that it's arising from the bladder and growing inside and outside.

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Prostate cancer

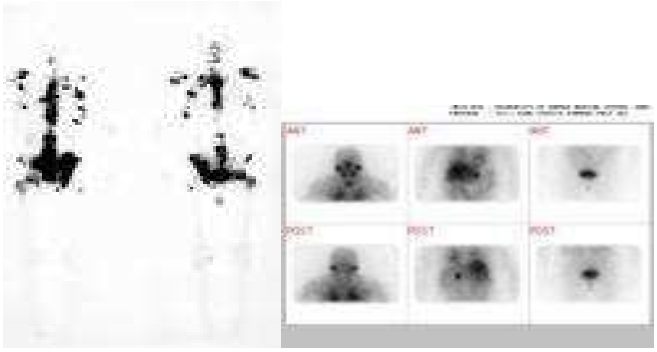
- Commonest malignancy of male urogenital tract
- Rare before the age of 50 years
- Found at post-mortem in 50% of men older than 80 years
- 5-10% of operation for benign disease reveal unsuspected prostate cancer

Pathology

- The tumours are adenocarcinomas
- Arise in the peripheral zone of the gland (BPH arises from the central zone, that's y symptoms appear erlier, while prostate CA symp. appear late).
- Spread through capsule into perineural spaces, bladder neck, pelvic wall and rectum
- Lymphatic spread is common
- Haematogenous spread occurs to axial skeleton
- Tumours are graded by Gleeson classification

Prostate CA LOVES BONES!!!

It can cause plastic and lytic leasions in the bone but mostly PLASTIC!



Supra scan. Dye concentrated in metastatic sites (spine+pelvis). Typical prostate CA. Remember it LUVs bones.

Clinical features

- 60% present with symptoms of bladder outflow obstruction
- 10% are incidental findings at TURP

- Remainder present with bone pain, cord compression or leuco-erythroblastic anaemia
- Renal failure can occur due to bilateral ureteric obstruction

Diagnosis

- With locally advanced tumours diagnosis can be confirmed by rectal examination
- Features include hard nodule or loss of central sulcus
- Transrectal ultrasound is useful in cases of diagnostic doubt
- Transrectal or transperineal biopsy should be performed

Pelvic CT or MRI is useful in the **staging** of the disease

- Bone scanning will detect the presence of metastases
- Unlikely to be abnormal if asymptomatic and PSA < 10 ng/ml

Serum prostate specific antigen (PSA)

- Kallikrein-like protein produced by prostatic epithelial cells (produced by all prostate cells).
- 4 ng/ml is the upper limit of normal
- >10 ng/ml is highly suggestive of prostatic carcinoma
- Can be significantly raised in BPH or prostatitis, but usually more elevated in prostate CA. (it's not a specific test the chance of having prostate CA w/high PSA is 25%,but it's a marker for us to go ahead and biopsy, to predict pnt's outcome "higher PSA, worse prognosis" and to moniter the pnt).
- Useful marker for monitoring response to treatment

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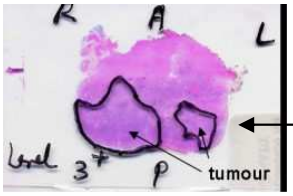
Treatment

Prostate CA responds to almost everything!

- More men die with than from prostate cancer
- Treatment depends on stage of disease, patient's age and general fitness
- Treatment options are for:
- **Local disease**
  - **Observation**
  - **Radical radiotherapy**
  - **Radical prostatectomy:** (remove prostate+seminal V. +pelvic lymph nodes). 3 ways:
    1.open(very bldy 2-3L in surg)
    2.Laparoscopically
    3.Davinci Robot
    (most recent and done in KKUH, the Dr. controls the robotic arms).

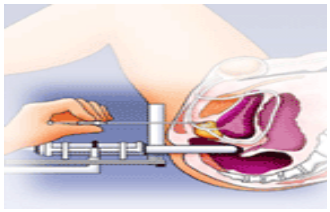
In 1964 a couple of scientists from the Dominican Republic won the Nobel prize for discovering that prostate CA was one of those rare CAs that was testosterone dependent after observing that hermaphrodites NEVER developed prostate CA. they treated the pnt's from CA by performing orchidectomy and therefore removing the testosterone. But many men found orchidectomy offensive. So a canadian doctor developed an LHGH analog which blocks by negative inhibition the pituitary axis which produces LH and FSH and therefore inhibits testosterone synthesis.

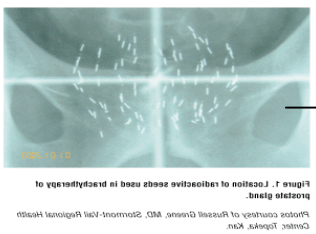
- **Locally advanced disease**
  - Radical radiotherapy
  - Hormonal therapy



Pathology slide of a prostate. Demonstrating that the prostate CA cells usually arise from the periphery of the gl. NOT the centre.

- **Metastatic disease**
  - Hormonal therapy





Brachytherapy: form of radiation therapy invasively insert a device for US and look at the prostate. Then insert these small radioactive iodine seeds. We use a special US or CT to view where the seeds are.(so it's performing radiotherapy close to the tumor, so you don't have to fully expose the pnt. to radiation).

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Hormonal therapy

- 80-90% of prostate cancers are androgen dependent for their growth
- Hormonal therapy involves androgen depletion
- Produces good palliation until tumors 'escape' from hormonal control
- Androgen depletion can be achieved by:
  - Bilateral orchidectomy
  - LHRH agonists - goseraline
  - Anti-androgens - cyproterone acetate, flutamide, Biclutamide
  - Complete androgen blockade

Testicular Tumours

- Commonest malignancy in young men
- Highest incidence in caucasians in northern Europe and USA
- Peak incidence for teratomas is 25 years and seminomas is 35 years
- In those with disease localised to testis more than 95% 5 year survival possible
- Risk factors include cryptorchidism, testicular maldescent and Klinefelter's syndrome

Very peculiar cuz germ cells are what make babies, so they grow very fast. Prostate doubling time is ‘bout two yrs, Testes CA doubling time is 18 days!!! CA’s that grow fast are VERY DEADLY, BUT usually respond VERY well to chemotherapy. (So treatment has to be done QUICKLY!).

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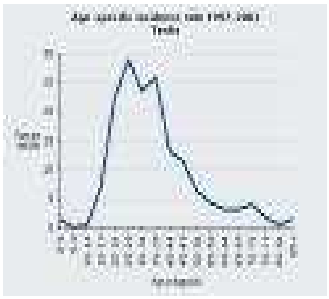
Classification

- Seminomas (~50%)
- Non-Seminoma (~50%)
  - Teratomas
  - Yolk sac tumors
  - Embryonal
  - Mixed Germ cell tumor

Investigation

- Diagnosis can often be confirmed by testicular ultrasound
- Pathological diagnosis made by performing an inguinal orchidectomy
- Disease can be staged by thoraco-abdominal CT scanning
- Tumour markers are useful in staging and assessing response to treatment
- Alpha-fetoprotein (alphaFP)
  - Produced by yolk sac elements
  - Not produced by seminomas
- Beta-human chorionic gonadotrophin (betaHCG)
  - Produced by trophoblastic elements
  - Elevated levels seen in both teratomas and seminoma
- LDH



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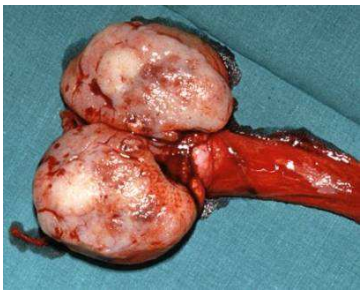
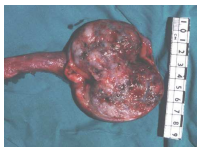
Stage Definition

- I Disease confined to testis
- IM Rising post-orchidectomy tumour marker
- II Abdominal lymphadenopathy
  - A < 2 cm B 2-5 cm C > 5 cm
- III Supra-diaphragmatic disease

Seminomas	None-Seminoma
■ Seminomas are radiosensitive ■ Stage I and II disease treated by inguinal orchidectomy plus ■ Radiotherapy to ipsilateral abdominal and pelvic nodes ('Dog leg') or ■ Surveillance ■ Stage IIC and above treated with chemotherapy	■ None-Seminoma are not radiosensitive ■ Stage I disease treated by orchidectomy and surveillance Vs retroperitoneal lymph node dissection (RPLND)((MOST IMP.!)) "here we mean retroperitoneal LN, para-aortic lymph nodes" Vs Chemo ■ Chemotherapy (BEP = Bleomycin, Etopiside, Cisplatin) given to: ■ Stage I patients who relapse ■ Metastatic disease at presentation
	

Lance Armstrong

He is a hero, he is a living legend, he is Lance Armstrong. One of the world's best cyclists. Lance Armstrong seemed invincible and his future was bright until the bad news hit him that he had cancer.

Then a combination of physical conditioning, a strong support system and competitive spirit took over. He declared himself not a cancer victim but a cancer survivor. He took an active role in educating himself about his disease and the treatment, he underwent aggressive treatment and beat the disease.

Before his recovery, he created the Lance Armstrong Foundation (Live Strong). This marked the beginning of Lance's life as an advocate for people living with cancer and a world representative for the cancer community.

Lance Armstrong's victories in the 1999-2005 Tours de France are awe-inspiring, but the battle against cancer has just begun for all cancer survivors. He planed to lead the fight, and he won the combat of wills by beating his cancer and becoming a spokesperson for all cancer survivors. This was his choice to live strong


Photo Phil O'Connor

Lance's Disease Facts:

Lance was diagnosed with an aggressive form of testicular cancer, containing 60% choriocarcinoma, 40% embryonal and less than 1% teratoma. Lance's treatment lasted from October to December 1996. He underwent two surgeries, one to remove his cancerous testicle and another to remove two cancerous lesions on his brain. Lance received one round of BEP (Bleomycin, Etoposide and Platinol) chemotherapy, followed by three rounds of VIP chemotherapy (Ifosfamide, Etoposide and Platinol. Lance's cancer in the lungs and brain was a result of spreading. from the original testicular cancer. As a result, his treatment protocols were to combat that specific strain of cancer.

LIVESTRONG