Handbook of Urology

Editors

Urology Department faculty members

Faculty OF Medicine

Sohag University

FOR Medical Students

First edition

IN LOVING MEMORY

OF

PROFESSOR MAHMOUD RYAD MOTAMED

THE FOUNDING FATHER OF THE DEPARTMENT OF UROLOGY AT SOHAG UNIVERSITY

Urology

Department of Urology
Faculty of Medicine
Sohag University

Handbook of Urology
for
Medical Students

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PREFACE

this handbook introduces medical students to the common urologic diseases and disorders. It will also be a valuable guide for the general practitioner. The book includes most of the topics in the field of urology, including urologic symptoms and signs, focused examination, pediatric urology, andrology, uro-oncology, voiding dysfunction and urolithiasis genitourorinary trauma, urinary tract infection, and obstructive uropathy.

While preparing this text, we made sure that the content is clear, concise, and easily accessible. It provides the reader with the principles of urology as well as the most recent advances in diagnostic and therapeutic tools. The book also addresses important aspects of the dynamics of the interaction between the urologist and the patient, and between the urologist and medical team. In the end, for the medical student, the most valuable resources are the patient, co-team members, and the supervisors.

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Acknowledgements

In The name of Urology department at Sohag University, I would like to acknowledge the legacy of **Professor Mahmoud Ryad Motamed**, the founding father of the Department of Urology at Sohag University. This department, which he started from scratch, has grown tremendously and now includes the various urology subspecialties. I would like express my appreciation and gratitude to my colleagues in the Department of Urology for their contributions to this book and for all the effort they put into editing and refining its content.

Finally, our special thanks to **Dr. Islam Abd el-wareth** for his valuable contributions to the editing of the book.

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Sohag University.

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Abbreviations

BOO ---- bladder outlet obstruction

BPH ---- benign prostatic hyperplasia

CBC ---- complete blood count

CIC ---- clean intermittent catheterization

CIS ---- carcinoma in situ

CRF ---- Corticotrophin releasing factor

DD ---- differential diagnosis

DRE ---- digital rectal examination

ED ---- erectile dysfunction

ESR ---- erythrocyte sedimentation rate

FSH ---- follicular stimulation hormone

GFR ---- glomerular filtration rate

HCG ---- human chorionic gonadotropin

HIV ---- human immunodeficiency virus

HLA ---- human leukocyte antigen

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RUG ---- retrograde urography

SCC ---- squamous cell carcinoma

SWL ---- Shock Wave Lithotripsy

TB ---- Tuberculosis

TCC T ---- Transitional cell carcinoma tumor

TUR ---- Transurethral resection

TURBT ---- Transurethral resection of bladder tumour

TURP ---- Transurethral resection of the prostate

TVT ---- tension-free vaginal tape

UDS ---- urodynamic study

UMNL ---- Upper motor neuron lesions

UPJ ---- ureteropelvic junction

URS ---- Ureteroscopy

U/S ---- Ultrasound

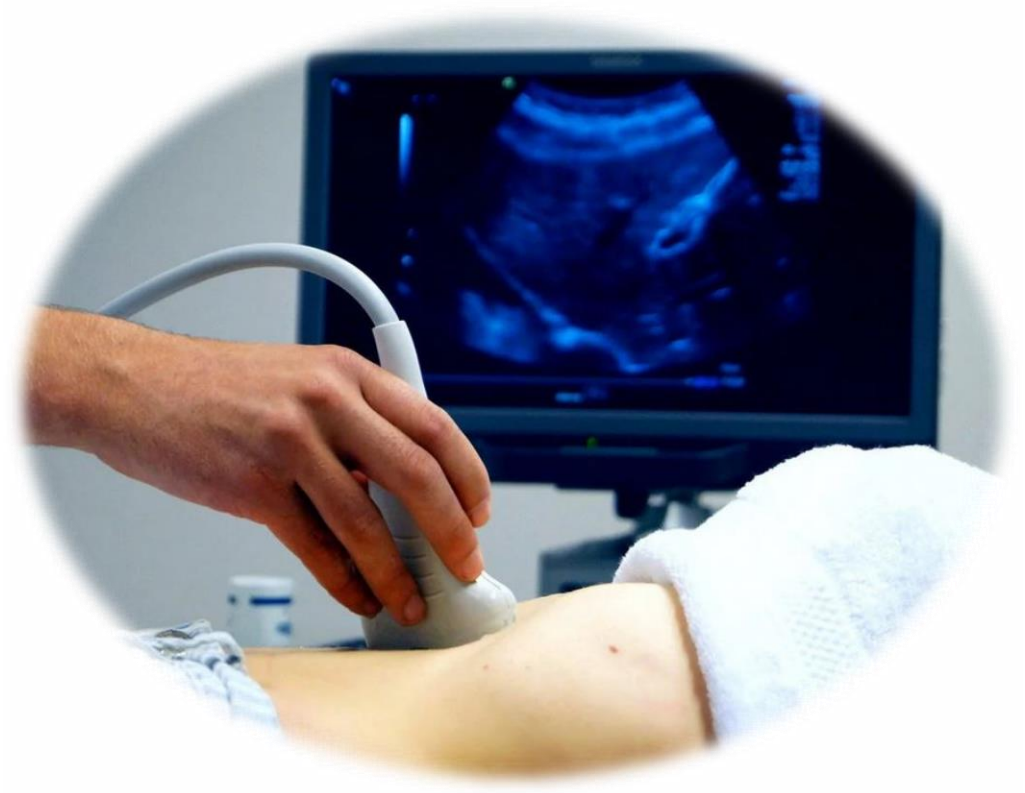
UTI ---- urinary tract infection

VCUG ---- voiding cystourethrogram

VIU ---- visual internal urethrotomy

VUR ---- Vesicoureteral reflux

Urology case evaluation



PROF/DR - ABDEL BASSET BADAWY

Urology case evaluation

History:-

Upper urinary tract symptoms:

• Flank pain.

•Renal swelling. When there is huge enlargement of the kidney/s e.g. Hydronephrosis, polycystic kidney disease and Wilms' tumor.

Hematuria usually total may be intermittent or continuous as in urothelial tumor; urinary and stones renal cell carcinoma.

• Oliguria and anuria. Oliguria is urine output of less than one ml/Kg/hr in infants, 0.5ml/Kg/hr in children and 400 ml/24 hours in adults, while anuria is cessation of urine production resulting in urine output less than 100 ml/24 hours in adults

• Polyuria. It is the excess production of urine to more than 3 liters per 24 hours

. It can be caused by diabetes mellitus, diabetes insipidus, chronic renal failure, post-obstructive diuresis, compulsive water intake and diuretics.

Flank Pain:

Causes of flank pain:

•Renal and ureteric stones:

This is the commonest cause. It may be chronic dull aching pain especially in cases of renal stones. On the other hand, stone migration leads to hyperperistalsis of pelvicalyceal system (renal colic) or ureter (ureteric colic). Colic is felt as severe intermittent pain of sudden onset and sudden offset.

According to site of stone, pain may **radiate to** the suprapubic region, ipsilateral groin, scrotum, testis, labia and urethra down

to the tip of the penis. It may be associated with GIT symptoms (nausea, vomiting and abdominal distension), hematuria, dysuria, urgency and frequency.

•Infection:

As acute pyelonephritis, infected hydronephrosis, pyonephrosis, renal abscess and perinephric abscess. There is associated fever and local tenderness. Pain can be throbbing in nature with pus under tension.

- Ureteropelvic junction obstruction and ureteric stricture: - (PUJO)

Pain is usually chronic dull aching due to distension of renal capsule. It may be precipitated or accentuated by diuresis.

- Renal or ureteric tumors:

Pain is considered a late presentation for upper urinary tract tumors. It is usually preceded by hematuria in cases of urothelial tumors.

•Blood clots

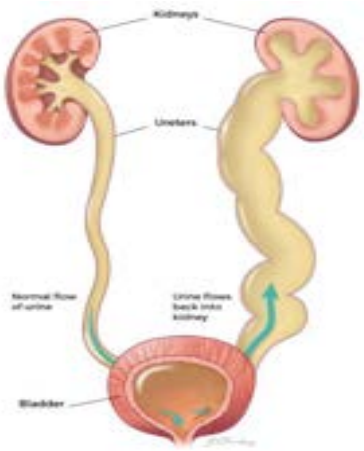
From any source in the upper urinary tract may produce colicky pain during their passage.

• Other causes

E.g. cystic diseases of the kidney and vesicoureteric reflux.

Differential diagnosis of renal pain (Non-urologic causes of flank pain):

They are **not** usually associated with urine changes or LUTS.



Vesicoureteric reflux

•Myo-skeletal causes e.g.:

Muscle spasm, sprain or inflammation, rib fracture and herniated intervertebral disc. Pain is provoked by certain body positions, trunk movements, coughing, sneezing and respiration. It may radiate to the lower limbs.

- Gastro-intestinal causes e.g.:

Irritable bowel syndrome (abdominal distension and change in bowel habits), cholecystitis (fatty dyspepsia and jaundice) and appendicitis (rebound tenderness at the right iliac fossa and psoas spasm).

- Gynecological causes e.g.:

Torsion of an ovarian cyst and ectopic pregnancy (in females especially in the child bearing period and associated with menstrual changes).

- Herpes zoster:

Pain is severe and revealed by appearance of specific eruption along the course of the intercostal nerves.

•Basal pleurisy and pneumonia

(Related to respiration, associated with pulmonary symptoms as dyspnea and cough).

Lower urinary tract symptoms (LUTS):

1. Storage LUTS (irritative):

They are caused by irritative bladder pathology e.g. bladder stone, inflammation, bladder diverticulum and bladder malignancy.

- Increased frequency of micturition: > 8 times / 24 hours.
- Nocturia (nocturnal frequency): > one time / night.
- Urgency: sudden severe compelling desire to micturate that cannot be deferred.
- Urge incontinence: involuntary urine leakage associated with urgency.

2. Voiding LUTS (obstructive)

- Hesitancy: The difficulty to start micturition.
- Intermittency = interrupted stream.
- Weak stream or narrow stream (of narrow caliber).
- Straining during voiding.
- Terminal dribbling.
- Sense of incomplete evacuation of the bladder.
- Acute retention of urine: It is the maximal obstructive form of LUTS.

3. Pain:

- Suprpubic pain:

Due to different bladder pathologies e.g. acute bacterial cystitis and anterior bladder wall malignancy.

- Urethral pain:

Usually is expressed as burning micturition (dysuria). It is due to inflammation (urethritis & cystitis), stones (urethral, bladder & intramural) and bladder ulcer or cancer.

- Low back, perineal or peri-anal pain:

May be caused by different diseases including prostatic and urethral pathologies e.g. stones, inflammations (urethritis, prostatitis & prostatic abscess)

4. Urethral discharge:

It is detected in the clothes and not related to the act of micturition. Its amount, color and associated symptoms should be assessed. It may be; Mucous, muco-purulent and purulent discharge.

5. Swelling:

- Suprapubic swelling: mostly due to chronic urine retention.
- Perineal or penile swelling: due to urine and/or blood extravasation after urethral trauma or penile fracture.

6. Lower urinary tract fistulae and sinuses:

The cause can be congenital, inflammatory, post- traumatic (accidental or surgical) or neoplastic e.g. Vesico-cutaneous (suprapubic), vesicovaginal, vesicorectal or ureterovaginal.

- Urachal = congenital umbilical vesico-cutaneous urinary fistula.
- Urethro-cutaneous (perineal or penile), urethrovaginal or urethrorectal.

ACUTE URINARY RETENTION

It is the inability to pass urine in spite of severe painful desire to void with full urinary bladder.

Etiology:

1. Stone impacted in the urethra or bladder neck.
2. . Urethral trauma with complete rupture
3. Prostatic diseases as benign prostatic hyperplasia (BPH) and prostatic abscess.
4. Neuropathic bladder e.g. after spinal cord injuries
5. . Occasional causes e.g.: - Reflex urinary retention due to severe painful perineal and anal conditions e.g. after surgeries for piles or anal fissure.
6. Obstructed Foley catheter.
7. Acute urethritis and impermeable urethral stricture.
8. Bladder neck or prostatic cancer.
9. Phimosis.
10. Hysterical retention.

Urine changes:

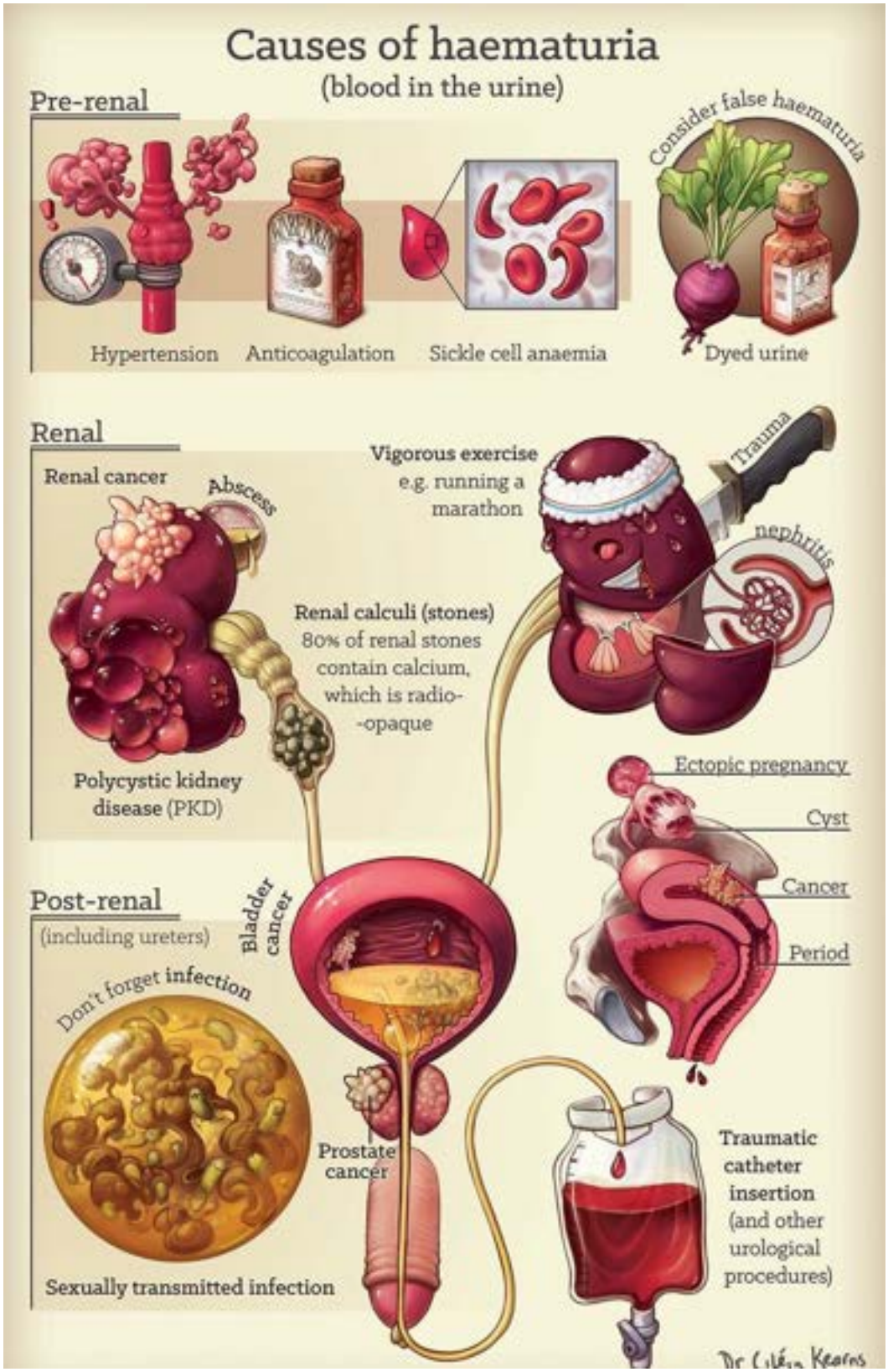
- Hematuria
- Pyuria (macroscopic or gross): in urinary tract infection (UTI) e.g. pyelonephritis and cystitis.
- Crystaluria: e.g. phosphaturia and less commonly oxaluria or uricosuria.
- Necroturia: the passage of whitish or pinkish pieces of necrotic tissues with urine in advanced bladder cancer.
- Pneumaturia: the passage of gases in urine due to fistula (e.g. vesico-intestinnal or vesico-colic), UTI with gas-forming organisms (especially in diabetics) or after recent urologic instrumentation.
- Discolored urine as in: pseudomonas infection (greenish), jaundice (brownish or olive green) and some administered foods or drugs (e.g. reddish with rifampicin).
- Bad unpleasant smell of urine: due to UTI by E.coli or urease producing micro-organisms.

HEMATURIA

It is the presence of more than three RBCs / HPF in urine sediment after centrifugation. It may be microscopic or macroscopic (gross).

Etiology:

1. Glomerular hematuria due to e.g. glomerulonephritis (e.g. post-streptococcal), IgA nephropathy and systemic lupus erythematous.
2. Non-glomerular hematuria can be caused by any urologic pathology e.g. urolithiasis, infections, trauma, BPH, polycystic kidney disease and tumors.
3. Post-urologic open or endoscopic surgery as primary, reactionary or secondary hemorrhage.
4. Coagulation & bleeding disorders and anticoagulant therapy.
5. Cyclic hematuria (menstrual bleeding mixed with urine) in vesicouterine fistula and endometriosis of the bladder or ureter.


Causes of haematuria
(blood in the urine)
Pre-renal
Hypertension
Anticoagulation
Sickle cell anaemia
Consider false haematuria
Dyed urine
Renal
Renal cancer
Abscess
Vigorous exercise
e.g. running a marathon
Trauma
nephritis
Renal calculi (stones)
80% of renal stones contain calcium, which is radio--opaque
Polycystic kidney disease (PKD)
Ectopic pregnancy
Cyst
Cancer
Period
Post-renal
(including ureters)
Bladder cancer
Don't forget infection
Prostate cancer
Traumatic catheter insertion (and other urological procedures)
Sexually transmitted infection

Evaluation:

A- History of:

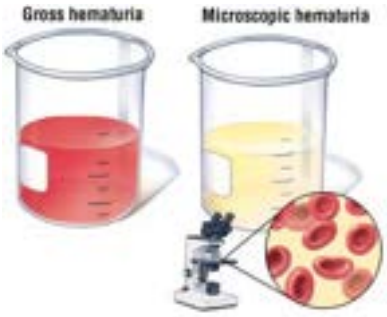
- Any possible cause e.g. trauma, operations and exposure to bilharziasis.

- Relation of hematuria to the act of micturition. It may be:

- Initial (at the start of micturition followed by clear urine) due to a lesion at the posterior urethra or bladder neck.

- Terminal (at the end of micturition preceded by clear urine) due to bladder lesion e.g. bilharziasis.

- Total (the whole urine is red) due to bladder or upper urinary tract lesions.



- Color of hematuric urine: bright red urine means fresh bleeding and brownish urine means old bleeding.

- Presence of clots confirms true hematuria and may cause obstructive LUTS up to clot retention. The shape of clots may point to the origin of bleeding. Thread-like clots are of renal or ureteral origin, while discoid clots are mostly of urinary bladder lesions.

- Associated pain helps in localization of the pathology e.g. renal pain with upper urinary tract lesions and painful micturition with bladder lesions.

- Painless hematuria is present in e.g. BPH, transitional cell carcinoma (TCC) of urinary bladder, glomerulonephritis and bleeding tendencies.

- Bleeding at other sites suggests a systemic cause.

B- Clinical examination:

•General examination: To detect any other bleeding sites and systemic effects of blood loss e.g. pallor, tachycardia and hypotension.

• Abdominal examination for renal swellings and clot retention.

• DRE for e.g. BPH and bladder tumor.

• Inspection of a voided urine sample

C- Differential diagnosis:

• Total red discoloration of urine due to e.g. some drugs (as rifampicin), foods (as beet roots). The onset and offset are clearly related to the causative ingested material. The discolored urine is not turbid, with no clots or urologic manifestation.

•Bleeding per urethra which means blood trickling from the urethra without voiding due to a urethral lesion or trauma distal to the urinary sphincters. Blood is detected at the external urethral meatus and the clothes.

• Contamination by menstrual bleeding, thus urine analysis is better avoided during menses.

D- Investigations:

- • Urine analysis (**essential**):
  - - It confirms the diagnosis by detection of RBCs.
  - - The cause of hematuria may be detected e.g. bilharzial ova, bacteria and crystals.
  - - It differentiates between glomerular hematuria (dysmorphic RBCs, proteinuria and RBCs casts) and non-glomerular hematuria (eumorphic RBCs).
- • Abdominal U/S (**essential**) can detect urologic lesions e.g. renal or bladder stones or tumors.

  • Further investigations are individualized and tailored according to the cause.

  •When the cause of hematuria is not detected, we resort to other specific investigations

including checking the bleeding profile, urine cytology, cystoscopy, uretero-renoscopy and renal biopsy.

E- Treatment:

Entails treatment of the cause besides:

- Simple general measures: e.g. bed rest, good hydration and hemostatic drugs.
- With massive hematuria: hospitalization, vital signs monitoring and blood transfusion.
- For clot retention: Evacuation of clots using Nelaton catheter followed by continuous bladder irrigation using three-way Foley catheter.
- Specific measures to stop massive bleeding e.g. hemostatic dose of radiotherapy in bladder cancer and angio-embolization in renal trauma or tumors.

Male genital symptoms

- **Erectile dysfunction (ED).**

- **Ejaculatory disorders.**

An ejaculation:-Inability to ejaculate semen as in spinal cord injuries, postoperative in surgeries that damage pelvic nerves

Premature ejaculation: - when ejaculation happen sooner than man or his partner would like during sex.

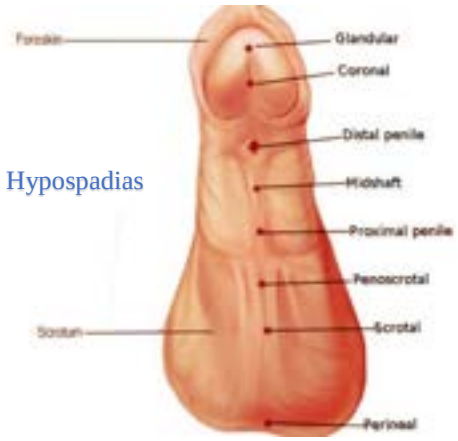
Delayed ejaculation: - man take an extended period of sexual stimulation to release semen

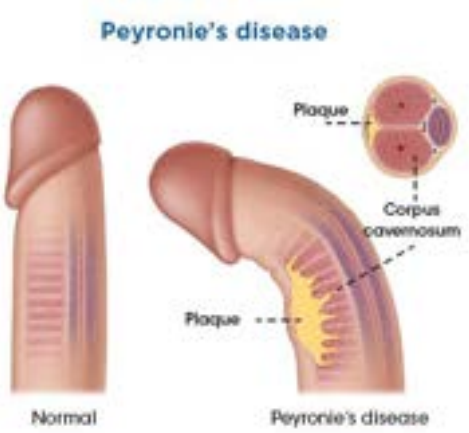
- **Subfertility**. Sexually mature male unable to impregnate a fertile woman
- **Scrotal conditions** either:
    - - Pain: referred (from the upper urinary tract) or due to local cause (as testicular torsion, trauma, mumps or epididymitis)

- - Swelling e.g. epididymo-orchitis, hydrocele, varicocele, spermatocele, hernia or testicular tumor.
- - Empty scrotum e.g. testicular maldescent
- - Scrotal sinus e.g. posterior TB epididymal sinus

• **Position of the external urethral meatus** at an abnormal site e.g. or epispadias.

•**Penile conditions** e.g. penile curvature, micropenis, concealed penis, priapiasm and Peyronie's disease.





Uremic manifestations

Mild cases are asymptomatic (discovered incidentally) or show non-specific symptoms as headache, lack of concentration, anorexia and easy fatigability.

The symptoms of renal failure include:

• Lack of concentration (an early symptom).

• Headache and blurring of vision.

• Easy fatigability, tachypnea and palpitation (due to anemia, acidosis and hypertension)

•Gastrointestinal manifestations (Early: anorexia & dyspepsia. Late: dry mouth, metallic taste, nausea, vomiting, hiccup & abdominal distension)

• Bleeding tendency (late) e.g. epistaxis and hematemesis.

• Itching (late).

UROLOGIC EXAMINATION.

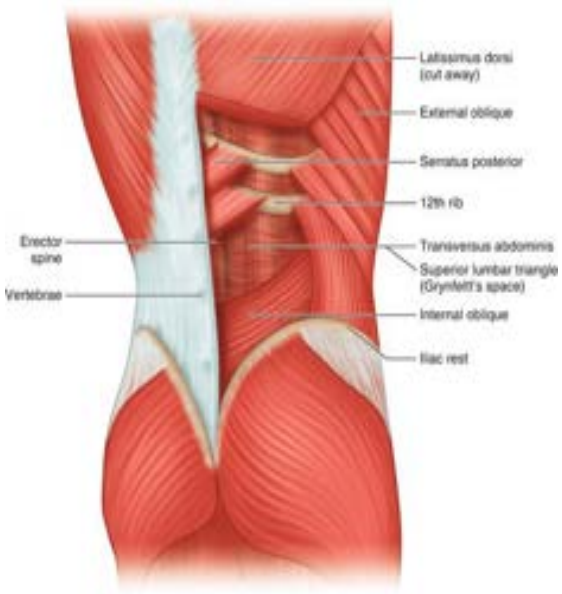
The loin is the anatomical region with the following **boundaries**:

>Superiorly: the lower border of the last rib

>Inferiorly: the iliac crest

>Posteriorly: the later border of sacrospinalis muscle

>Anteriorly: the mid-axillary line



The renal (costo-vertebral) angle lies between the lower border of the last rib and the lateral border of sacrospinalis muscle.

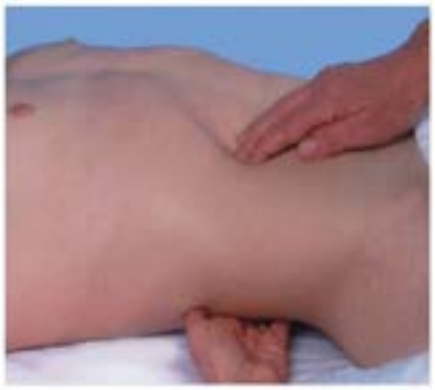
Loin examination is conducted mainly through:

- Inspection with sitting and lateral positions for e.g. scars, incisional hernias, swellings and sinuses.
- Palpation with supine position for e.g. tenderness, rigidity and swellings.
- Percussion for shifting dullness or any palpable swelling.

Bimanual palpation of the loin:

- The kidney is palpated while the patient is breathing deeply.
  - The left hand is placed posteriorly with the index overlies the last rib on the right side while the little finger overlies the last rib on the left side. The tips of the other three fingers (the thumb is not employed) stop at the lateral border of the sacrospinalis muscle i.e. in the renal angle. The left hand only pushes anteriorly for renal ballottement.
  - The right hand is placed anteriorly starting below the level of the umbilicus and moved towards the costal margin. On the right side, it is parallel to the costal margin. On the left side, it is perpendicular to the costal margin.

Signs characterizing a renal swelling:



Kidney palpation

- Site: retroperitoneal, so it is best felt at the renal angle unless the kidney is ectopic.
- Border: always it is rounded and never sharp.
- The swelling disappears below the costal margin so that one can insinuate the hand between the swelling and the costal margin unless the swelling is huge.

- Shape: reniform or oval.

- Movement and mobility: Up and down with respiration (within the paranephric space). This can be restricted or lost in pyonephrosis, infiltrating renal tumors or recurrent cases.

- Renal ballottement is antero-posterior movement within the perinephric space. When the left hand pushes the swelling anteriorly, the swelling is felt by the right hand and then returns back to the left hand. Ballottement is lost when a disease affects the perinephric space and fat (e.g. perinephritis, pyonephrosis, infiltrated by renal tumor) or previous renal surgery (adhesions). A huge renal swelling has no space for ballottement and it is felt by both hands at the same time which is called renal contact.

- All renal swellings are dull on percussion. However, a band of colonic resonance can be detected over the swelling.

- **Differential diagnosis of renal swelling:**

I- Parietal loin swellings:

>Move antero- posteriorly with respiration.
>Persist or become more prominent on abdominal wall muscles contraction.
>May overlie the costal margin.

II- Intra-abdominal swellings:

>Liver: Right sided, sharp border, the hand cannot be insinuated between the swelling and the costal margin and always dull on percussion.

>Spleen: Left sided, sharp notched border, the hand cannot be insinuated between the swelling and the costal margin, always dull on percussion and the direction of movement is toward the right iliac fossa.

>**Colon**: on right or left side, sausage shape, ill-defined borders and resonant on percussion.

>**Retroperitoneal swellings**: irregular shape & surface, firm to hard in consistency and not ballottable (fixed to the posterior abdominal wall).

III- Different types of renal swellings: (Abdominal sonography can easily distinguish each type of these swellings)

- Hydronephrosis
- Infected hydronephrosis

- Pyonephrosis

- Simple renal cysts
- Polycystic kidney diseases - Renal tumors

Male external genitalia examination:

The informed and consenting patient should be examined by both hands in both standing and supine positions.

I- Penis and urethra:

- The prepuce normally conceals the glans completely and the preputial opening is not narrow. It may be abnormal in e.g. phimosis, hypospadias and epispadias.

- The external urethral (urinary) meatus normally appears as a dry vertical slit situated at the tip of the glans with two lips that can be separated apart to see inside. It may be abnormally e.g. stenosed, located ventrally (hypospadias) or located dorsally (epispadias).

- The glans is normally conical in shape and flaccid with the meatus at its tip. Abnormally, it may have e.g. inflammation (balanitis) and fistula.

- The penile shaft is normally flaccid straight (without curvature), with identifiable ventral corpus spongiosum, smooth surface (without induration) and no tenderness. The penile size is age related. The lower normal length of the stretched penis is 1.9 cm in infants and 7 cm in adults.

- Abnormally, the penis may be e.g. small sized (micropenis), buried below the surface of the prepubic skin (with obesity) or bowed (with chordee).

II- Scrotum, testes, epididymis and spermatic cords:

•The scrotum

Normally has two compartments with an apparent median raphe in between, and corrugated pliable skin. Some of the scrotal lesions include e.g. scrotal sinus, bifid scrotum, and different skin diseases.

•The testis

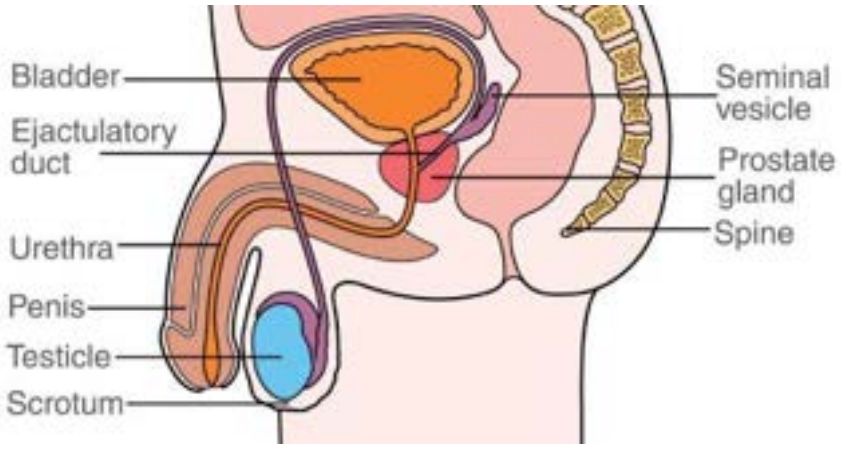
Normally lies at the bottom of the scrotum. It has oval shape, smooth surface, firm consistency and a characteristic testicular sensation. The testicular size is age related with no significant difference between the two testes. The testis is covered by tunica vaginalis; which has two layers (parietal and visceral) with minimal potential space in between. Abnormally, the testis may be e.g. agenetic, ectopic, inflamed and torsed.

•The epididymis

Is a ribbon-like structure that normally lies posterior to the testis. A laterally situated palpable sulcus separates the testis from the epididymis. The epididymis has a head (behind the upper pole of the testis), body and tail. It may be inflamed or the site of tuberculous scrotal sinus.

• The spermatic cord

Has two parts; scrotal and inguinal. The scrotal part is available for palpation. The spermatic cord is normally soft except the cord like vas deferens. Lesions that can be detected include e.g. varicocele, thickened or beaded vas, lipoma and encysted hydrocele of the cord.



Differential diagnosis of inguino-scrotal swellings:

Examination should be directed towards identification of swelling:

• Origin:

The scrotal neck test differentiates between pure inguinal swellings (all above the scrotal neck), pure scrotal swellings (all below the scrotal neck) and inguino-scrotal swellings (above, below and distending the scrotal neck).

•Consistency:

The bipolar fluctuation test detects cystic swellings (hydroceles) present in this mobile part. Gurgling sensation is present in hernias containing intestine (enterocele), while doughy sensation is present in hernias containing omentum (omentocele).

•Relation to the testis:

The testis may be separable from the swelling (e.g. acquired inguinal hernia and encysted hydrocele of the cord), the seat of the swelling (testicular abscess or tumor) or concealed by the swelling with lesions involving the surrounding tunica vaginalis (e.g. vaginal, infantile & congenital hydroceles and congenital inguinal hernia).

• Variation in size:

The swelling may be constant in size (e.g. infantile hydrocele and epididymal swellings), marked instant variation in size (e.g. hernias) or gradual variation in size over the day (e.g. congenital hydrocele or varicocele).

Inguinoscrotal swellings include e.g.:

1-• Hydrocele:

It is abnormal fluid collection between the two layers of the processus vaginalis at the inguinoscrotal region related to the testis and/or the spermatic cord.
Normally, the processus vaginalis is obliterated from the internal inguinal ring to the upper scrotum, leaving a small potential space that surrounds the testis (tunica vaginalis).
Hydroceles if not complicated usually present as chronic cystic painless swellings and they include:

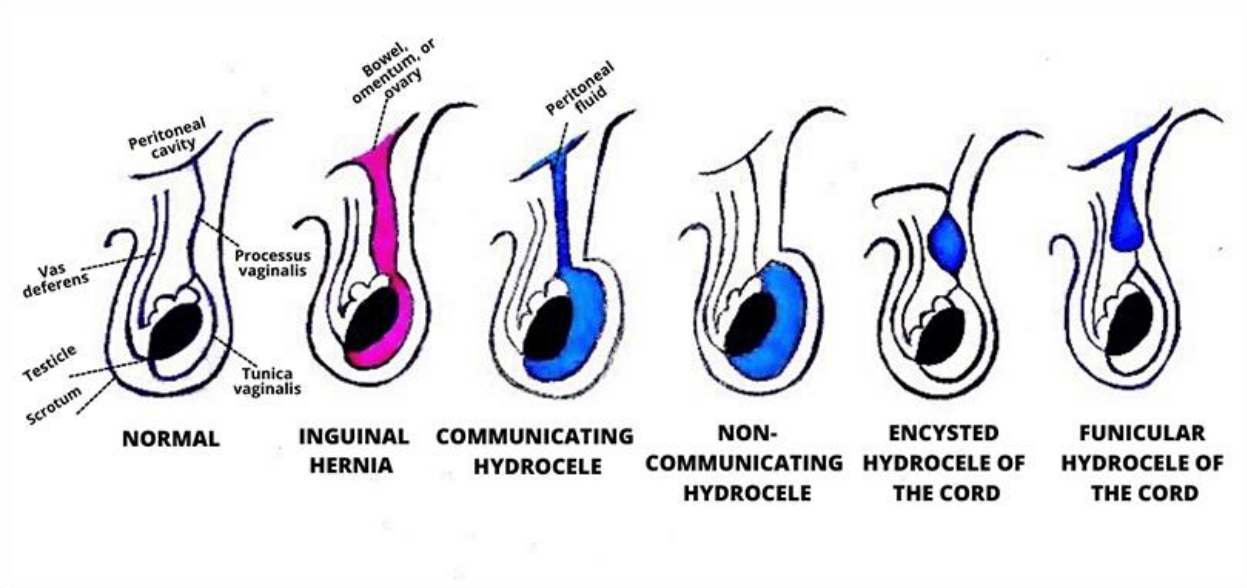
Congenital (communicating) hydrocele: It is inguino-scrotal swelling surrounding the testis and spermatic cord with a minute communication between its sac and the peritoneal cavity. Thus it slowly evacuates and refills. It is the only type of hydrocele that shows variation in size.

Infantile (non-communicating) hydrocele: It simulates the congenital type without communication with the peritoneal cavity; so there is no variation in size.

Encysted hydrocele of the cord: The unobliterated part of the processus vaginalis is isolated from both the peritoneal sac and the testis. It may be scrotal, inguinal or inguinoscrotal. The testis is not involved in the swelling.

Hydrocele of hernial sac: It is primarily an inguinal swelling (outside the cord) due to fluid accumulation in a closed hernial sac without any other content.

Vaginal hydrocele: It is a pure scrotal swelling surrounding the testis. It may be primary (with no detectable cause) or secondary (due to e.g. epididymo-orchitis, testicular tumor and ligation of lymphatics during varix ligation or inguinal hernia repair).



Special clinical tests:

- - Pinching of the parietal layer of tunica vaginalis in minimal vaginal hydrocele.
- - Positive trans-illumination test due to the clear nature of the fluid. In pyocele, hematocele or chylocele trans-illumination is negative.
- - Scrotal ultrasonography is mandatory when the testis cannot be clinically evaluated to exclude testicular malignancy or atrophy.

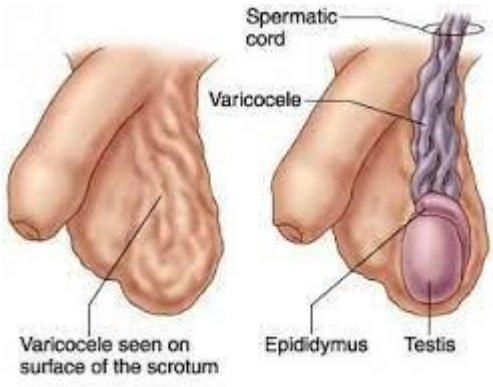
Treatment:

- - Conservative management: in neonates and infants waiting for spontaneous resolution over the first year. Surgery is indicated with persistence of hydrocele.
- - Surgical excision of the sac in all types of hydrocele. In uncomplicated vaginal hydrocele, eversion of the parietal layer of the tunica vaginalis is enough.
- - Treatment of the cause: in cases of secondary vaginal hydrocele.

2-• Inguinal hernia:

It may be congenital (with patent processus vaginalis and the testis is one of the contents) or acquired (with a new peritoneal hernia sac and the testis is outside). If uncomplicated it shows characteristic signs; namely expansile impulse on cough and reducibility. It may be complicated by irreducibility, obstruction or strangulation, so it needs repair as soon as possible.

3-• Varicocele:



It is an inguinoscrotal diffuse compressible cord swelling and its size increases with straining and decreases in supine position. It may also present with subfertility or dragging scrotal pain. It is usually left sided and may be bilateral.

4-•Spermatocele:

It is an epididymal cyst with unclear fluid containing sperms. Thus it is a pure scrotal swelling related to the upper pole of the testis (forming with the testis an 8-shaped figure). It varies in size and sometimes it resembles a third testis.

5-•Testicular tumor:

It is usually a painless firm testicular swelling with loss of the characteristic testicular sensation. It may be associated with rapidly formed vaginal hydrocele.

Digital rectal (anorectal) examination:

Digital rectal examination (DRE) begins by inspection of the perineum and ends by inspection of the examining finger after doing bimanual examination.

The steps include:

I- Positioning:

The informed and consenting patient acquires one of the following positions:

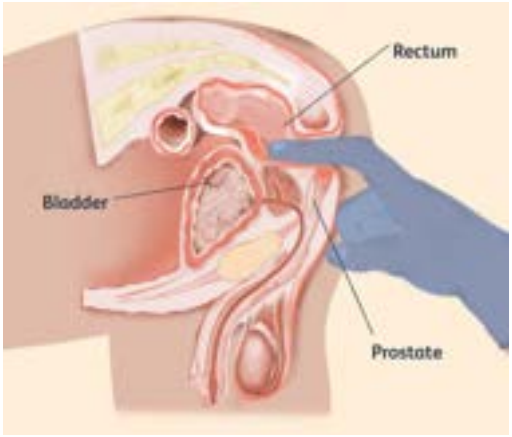
- Supine position (with bilateral flexion of both hip and knee joints) is the most frequently used in urologic practice.
- Knee-elbow position.
- Left lateral position

II- Inspection for:

- Normal corrugation around the anal orifice. Loss of corrugations is present if the external anal sphincter is atonic. This raises the possibility of neuropathic bladder.
- Perianal and perineal abscess, sinus or fistula
- Discharge, prolapsed piles or rectal prolapse.
- Signs of trauma e.g. perineal urinary extravasation and hematoma.
- Scar of previous operation (e.g. urethroplasty) or skin disease (e.g. tinea cruris)

III- Finger introduction:

- The hands should be gloved and the examining index finger well lubricated.
- The bulb of the index finger is applied to the anal verge in a light or superficial palpation manner to test for any tenderness or spasm.
- The pressure is increased (deep palpation) till the anal sphincter is relaxed, then the distal phalanx is flexed to be introduced into the anal canal.



- Proceed to introduce the middle phalanx to reach the prostate region, then the proximal phalanx to reach the bladder base.

DRE is not routine in children, and if indicated the little finger is used.

- Findings:

1. Anal sphincter tone:

Actually there is great anatomical and functional similarity between the anal and urethral sphincters. They share the same source of nerve supply. Each sphincter has two components; one is involuntary and the other is voluntary.
There is a normal degree of gripping or resistance to the examining finger by the anal sphincter tone.

This anal tone may be abnormally:
- Lost or decreased in lower motor neuron lesions, direct trauma to the sphincter and in the elderly.
- Exaggerated in uncooperative patient, upper motor neuron lesions, painful perianal conditions and anal canal stenosis the anal tone can be:

- Spontaneous:
- Voluntary: It is obtained by the intentional contraction of the sphincter after asking the patient to squeeze the examining finger during DRE as if the patient is trying to hold flatus. It is resorted to when the spontaneous gripping is doubtful or seems to be absent or weak.
- Reflex (the bulbocavernosus reflex): It is provoked by squeezing the glans or by gentle controlled traction on an indwelling catheter if the patient is catheterized.

2. The membranous urethra:

A stone or catheter can be felt in the midline below the prostate.

3. Prostate:

A normal prostate is not tender, with smooth flat surface & rubbery consistency and its base is easily reached. The soft base of the bladder is felt above. The prostate has two lateral sulci (between the lateral prostatic edges and the soft rectum) and a median furrow (bilobed).

DRE signs of benign prostate enlargement include:

- Convexity of surface = ↑ of antero-posterior dimension.
- Exaggeration of the lateral sulci = ↑ of antero-posterior and the transverse dimensions. - difficulty or failure to reach the prostate base = ↑ of cephalo-caudal dimension

In acute prostatitis and prostatic abscess, the prostate is extremely tender and the patient cannot tolerate DRE.

(NB) In prostate cancer, signs of malignancy include nodular surface, hard consistency obliteration of one or both lateral sulci, obliteration of the median furrow and frozen pelvis. There is no tenderness unlike bladder cancer.

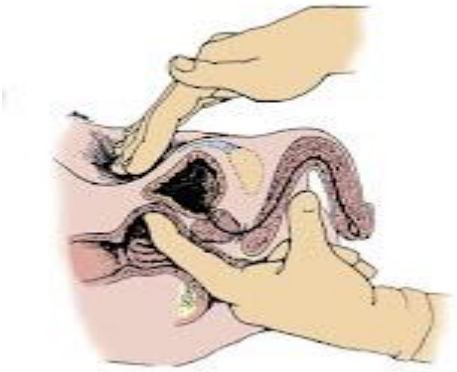
4. Bladder base is:
- normally soft, with smooth surface and not tender.
- Tender in acute bacterial cystitis, acute urine retention and bladder cancer - indurated, firm with irregular surface in posterior wall bladder cancer.

5. The seminal vesicles are normally impalpable. They may palpable with obstruction, bilharzial affection or malignant involvement.

6. The rectum: the examining finger is rotated around to detect any rectal pathology e.g. rectal polyp or cancer.

IV- Bimanual examination:

It is an essential step of DRE to assess the bladder between the examining finger (in the rectum) and the left hand (at the supra-pubic region). It helps to detect:
- Cystic bladder mass (urine) in chronic urine retention or huge bladder diverticulum.
- Solid bladder mass (cancer) which is usually tender. It may be mobile or fixed.



V- Inspection

Of the examining finger for blood, pus or mucus.

INVESTIGATIONS IN UROLOGY

Radiological Investigations in Urology

Ultrasonography (US)

A non-invasive imaging modality of the urinary tract especially kidneys and urinary bladder, however, unreliable in imaging of the ureters due to the over-lying bowel gas.

Indications

•Assessment of haematuria to rule out renal malignancy

•Characterization of renal masses: ultrasound can differentiate simple renal cysts (smooth, well-demarcated wall, reflecting no echoes; benign) from solid renal masses.

•Diagnosis of hydronephrosis.

•Allows ultrasound-guided nephrostomy insertion on emergency basis

•Measurement of post-void residual (PVR) urine volume.

•Placement of a suprapubic catheter.

• Measurement of prostate size

• Ultrasound guided prostate biopsy to rule out malignancy in patients with elevated serum PSA and/or abnormal DRE.

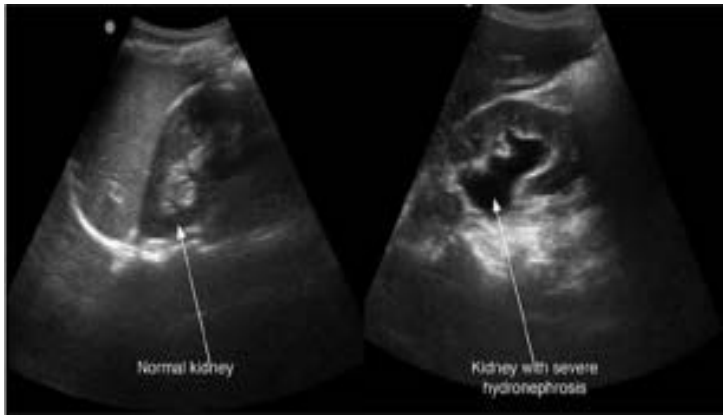
•Investigation of azoo-spermia

•Characterization of testicular tumours, hydrocele, epididymal cysts

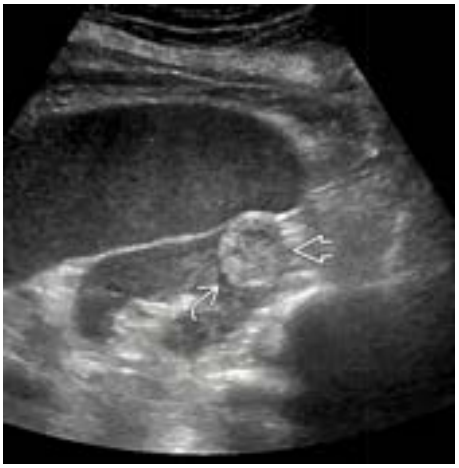
Assessment of testicular blood flow in suspected torsion.

•Assessment of testicular trauma

Investigation of infertility (varicoceles, testicular atrophy).



Normal vs hydronephrotic kidney



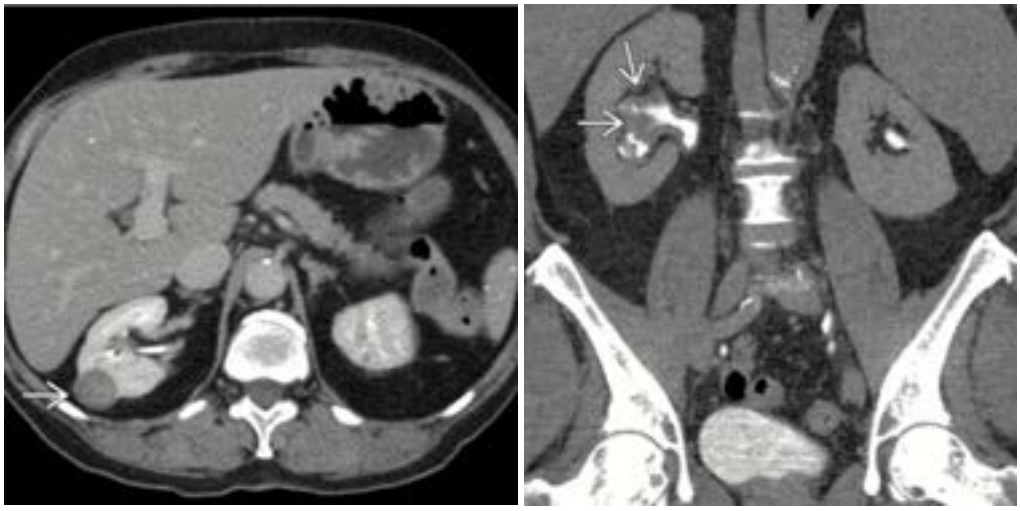
renal ultrasound showing renal tumour

Computed Tomography (CT)

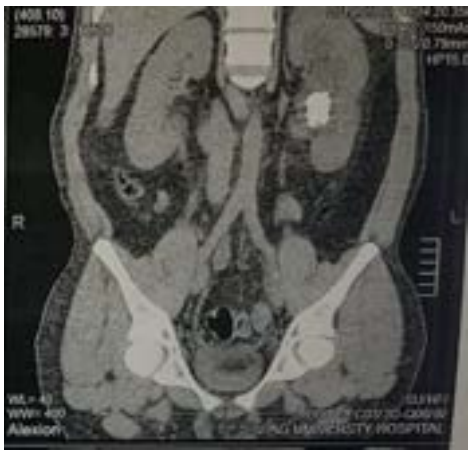
CT detects small differences in X-ray absorption values of different body organs and tissues providing a wide range of densities. Computer calculates the attenuation of each pixel and reconstructs this into an understandable image. CT scans without contrast can detect urinary tract stones. Administration of IV contrast (CT Urogram) is used to investigate haematuria to rule out malignancy in the urinary tract.

Indications for CT urinary tract

1- Visible Haematuria Workup: To determine the site and cause of urinary tract bleeding to rule out urinary tract malignancy with 65% sensitivity and 98% specificity
2- Assessment of renal masses
3- Staging of renal cancer
4- Assessment of stone size and location
5- Grading of renal trauma
6- Determination of the cause of hydronephrosis.



CT Urograms showing an example of RCC (Left image) and right renal pelvic TCC (right image).



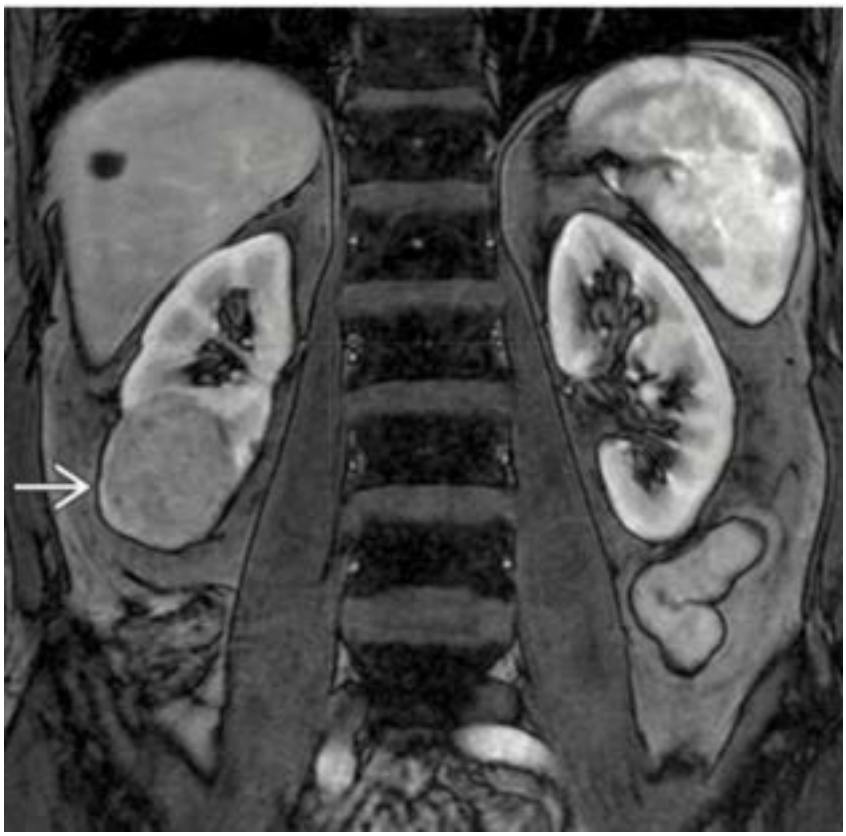
CTU film show stone pelvis LT kidney

Magnetic Resonance Imaging (MRI)

"MRI utilises the magnetic properties of the hydrogen nucleus in water molecules, and therefore in all body tissues. In a magnetic field, protons align along the direction of the field, and the application of pulsed alternating radio waves gives photon energy to hydrogen protons, thereby changing their alignment. When the radio-waves are switched off, the protons realign into their resting spin state and emit photon energy, which is detected by coils and generates an image". Gadolinium contrast can be used to speed up the relaxation time of protons, thereby increasing the contrast between normal and pathological tissues.

Indications

1- Staging of bladder and prostate cancer
2- Diagnosing phaeochromocytomas
3- Investigation of Neurogenic-LUTS in the presence of lumbar or thoracic back pain or associated with loss of perineal sensation or disturbances of bladder sensation.
4- Staging of renal cancer
5- Investigation for ureteric stones in pregnancy.



MRI showing right renal Tumour

Radioisotope imaging

Radioactive isotope e.g. technetium—99m that emits gamma rays, allowing the radiation to penetrate through tissues and reach a gamma camera placed adjacent to the patient.

Mercaptoacetyl-triglycyl (MAG3) renogram

Following IV injection, MAG3 is excreted within 15 seconds from the kidneys (90% by tubular secretion and 10% by glomerular filtration) and reaches the bladder in 3min. A time–activity curve can be recorded for each kidney known as MAG3renogram.Images are collected onto a film at 2s intervals for the first 1min and then at 20s intervals for the remainder of the study (30min in total).

Normal renogram has three phases

Phase I- steeply rising curve lasting 20–30s.

Phase II-a slowly rising curve to a peak.

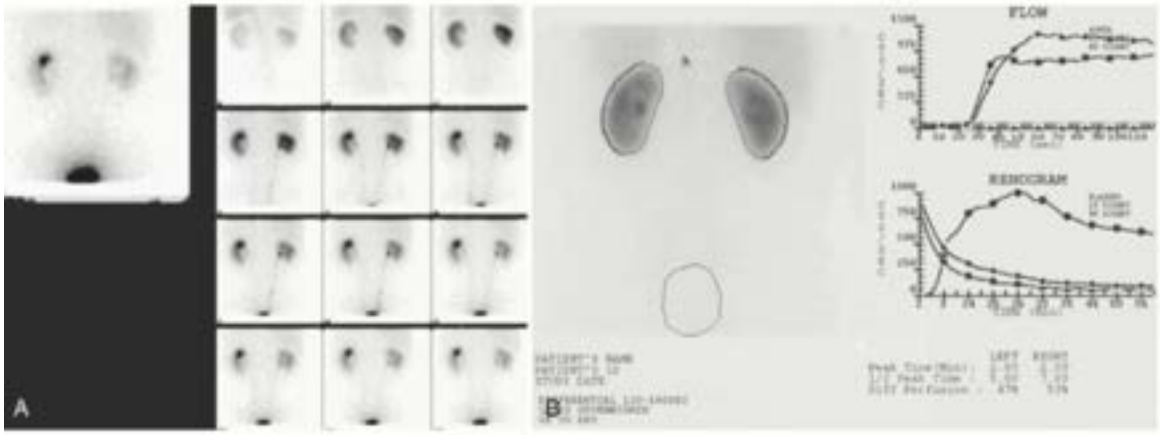
Phase III- a curve that descends after the peak.

Indications

1- Assessment of split renal function
2- Investigation for presence of renal obstruction

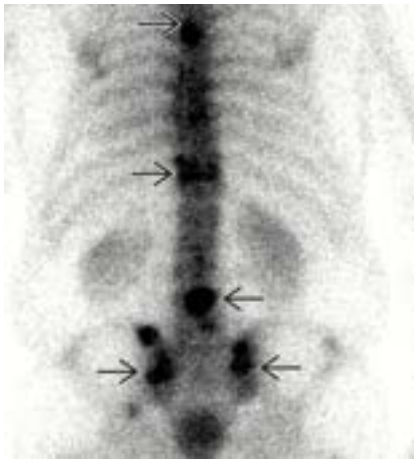
Dimercaptosuccinic acid (DMSA) scanning

DMSA, labelled with technetium—99m is taken up by the proximal tubules with very little being excreted in urine allowing a static imaging of the kidneys. It is useful in assessment of split renal function and detection of scars in the kidney.



Radioisotope bone imaging

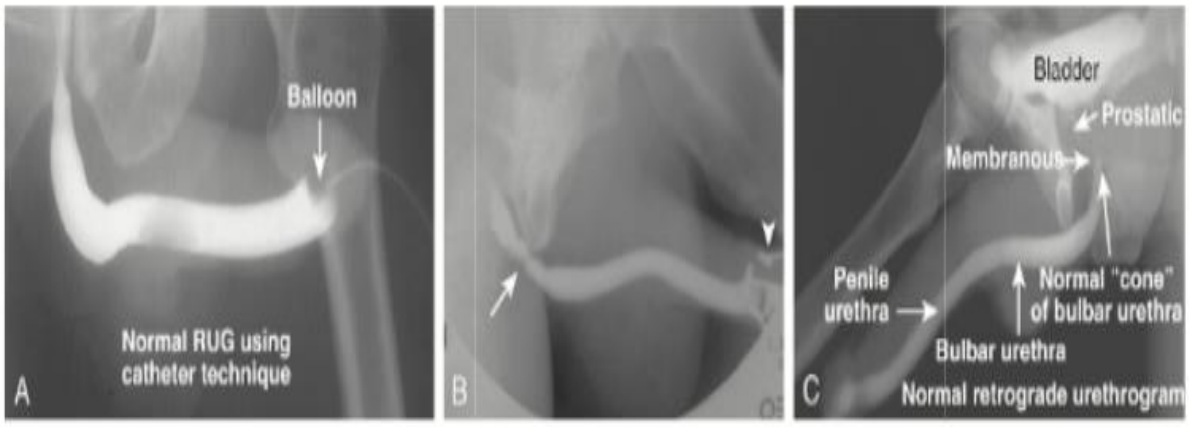
Technetium—99m labelled methylene diphosphonate (MDP) is taken up by areas of bone where there is blood supply and osteoblastic activity. Its uptake can be increased in bone metastases e.g., secondary to prostatic cancer (multifocal with predilection to spine). Differential diagnoses are bony fractures, osteomyelitis, and osteoma.



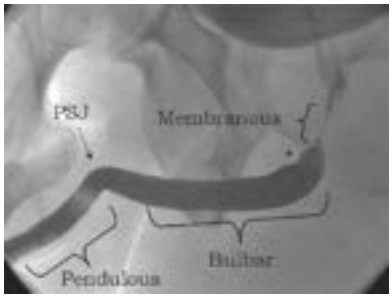
Bone scan with axial bony metastasis

Video cysto-urethrography (VCUG)

For the diagnosis of vesico-ureteric reflux (VUR) during voiding and assessment of bladder neck function in patients with neurogenic bladder, fistulae, suspected bladder rupture, urethral strictures and urethral injuries.



An example of urethrogram showing normal urethra



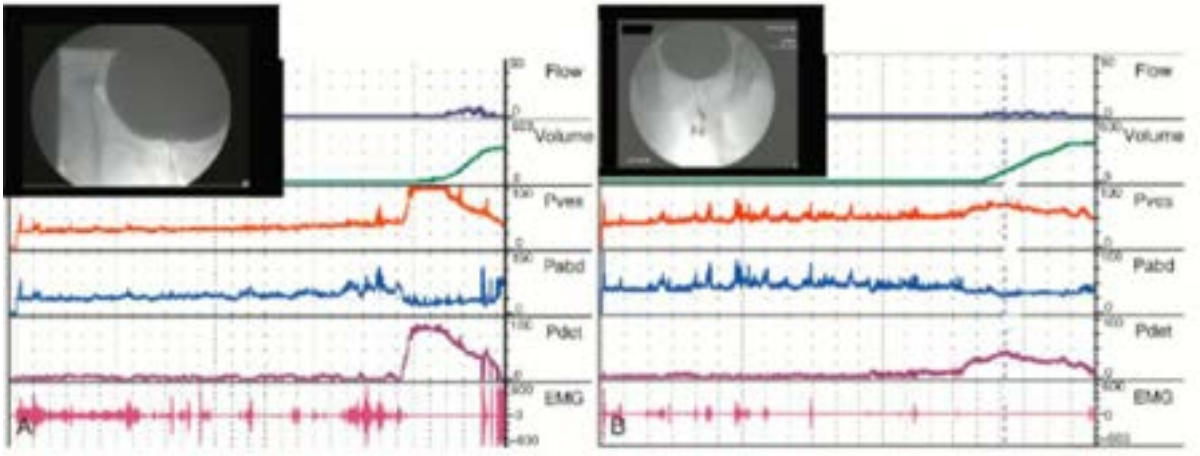
Urodynamic

The recording of bladder pressure during bladder filling and voiding, occasionally combined with fluoroscopy. Urodynamics aim to assess the bladder and urethral sphincter behaviour during bladder filling and during voiding, reproducing patients symptoms in clinical setting.

Urodynamics consist of two tests: Filling cystometry and Pressure Flow Study (PFS). Bladder pressure measured by a urethral catheter and abdominal pressure measured by a pressure line inserted into the rectum or vagina in women are recorded as the bladder fills. The pressure developed by the detrusor muscle can be derived by subtracting the abdominal pressure from the pressure measured within the bladder. All pressures are recorded in cmH_2O, and flow rate is measured in mL/s. A computerized printout of these pressures and flow rate is obtained. During bladder filling, the presence of detrusor overactivity can be detected. During voiding, the key parameters are the point of maximum flow rate and the detrusor pressure at that point, this can be used to define the presence of bladder outlet obstruction by using a predesigned nomogram.

Indications

1- Diagnosis of bladder outlet obstruction
2- Diagnosis of detrusor overactivity
3- Diagnosis of low bladder compliance
4- Diagnosis of urinary incontinence



An example of urodynamics trace combined with video-fluoroscopy

Renal Function Test

Glomerular filtration rate (GFR)

Creatinine is 113D-amino acid derivative, urinary clearance of creatinine is used for estimation of GFR applying the formula Clearance (GFR) = U X V/P

U is urinary creatinine concentration; P is plasma creatinine concentration, and V is urine volume.

Normal GFR is 130 ml/min per 1.73 m^2

Stage	Definition	GFR (ml/min per 1.73 m^2)
1	Kidney damage with normal GFR	>90
2	Mild decrease in GFR	60-89
3	Moderate decrease in GFR	45-59
4	Severe decrease in GFR	15-29
5	Kidney Failure	<15

Serum Prostate Specific Antigen (PSA)

PSA is a 34-kDa glycoprotein enzyme produced by prostatic epithelial cells. Its function is to liquefy the ejaculate, enabling fertilization. The half-life of serum PSA is 2.2 days. The normal range for the serum PSA assay in men is <3.0 ng/Ml. PSA > 3ng/ml in men aged 50-69 years old should trigger investigations to rule out prostatic cancer.

Urine Analysis

Urine Dipstick Test

A quick testing of urinary pH, proteins, erythrocytes, nitrite, leukocytes, and ketones

Component	Normal value	
pH	**5.5 to 6.5**	
Protein	**80-150 mg/24 hrs.**	
Erythrocytes	**<3 RBCs per HPF**	**Persistent microscopic haematuria in adults >40 years old require diagnostic cystoscopy to rule out malignancy**
Leucocytes	**0-1 per HPF**	**-Not all patients with bacteriuria have significant pyuria. -False negatives: concentrated urine, glycosuria, presence of urobilinogen, consumption of large amounts of ascorbic acid. -False positives: contamination.**
Nitrite	**Nil**	**Nitrites in the urine suggest the possibility of bacteriuria (35-85% sensitivity and 90% specificity)**

Midstream urine sample positive for leucocytes and nitrite is suggestive of UTI, if patient is symptomatic (e.g., dysuria, cloudy urine considers commencing him/her on empirical antibiotics and send the urine sample for further C&S.

Microscopic examination of Urine

RBCs morphology

RBCs derived from the glomerulus are dysmorphic. RBCs derived from tubular bleeding or from renal pelvis, ureters, or bladder bleeding have a normal shape.

Casts

A protein coagulum formed in the renal tubule and takes the shape of the tubule. If the cast contains only muco-proteins, it is called a hyaline cast. Seen in pyelonephritis or chronic renal disease. RBC casts contain trapped erythrocytes and are diagnostic of glomerular bleeding secondary to glomerulonephritis. WBC casts are seen in acute pyelonephritis.

Crystals

Calcium oxalate, uric acid, and cysteine are precipitated in acidic urine. Crystals precipitated in alkaline urine include calcium phosphate and triple phosphate.

EMBRYOLOGY

DEVELOPMENT

Mammals develop three kidneys in the course of intrauterine life:

1. The *pronephros,*
2. The *mesonephros,* and
3. The *metanephros.*

The first two kidneys regress *in utero,* and the third becomes the permanent kidney.

Embryologically, all three kidneys develop from the intermediate mesoderm

pronephros

Is seen late in the third week, and it completely degenerates by the start of the fifth week.

The second kidney, the mesonephros,

Is also transient, but in mammals it serves as an excretory organ for the embryo while the definitive kidney, the metanephros, begins its development

In males, some of the cranially located mesonephric tubules and mesonephric duct become;

1. **Ureter, pelvis and collecting tubules of the kidney**
2. **Triagone of the bladder**
3. **The efferent ductules of the testes**
4. **The epididymis**
5. **Vas deferens**
6. **Ejaculatory ducts**
7. **Seminal vesicles**

In females,

1. **Ureter, pelvis and collecting tubules of the kidney**
2. **Triagone of the bladder**
3. **Remnants of cranial and caudal mesonephric tubules form small, nonfunctional mesosalpingeal structures termed the epoophoron and paroophron. Gartener's duct is the remnant of mesonephric duct.**

The definitive kidney (the *metanephros*)

Forms in the sacral region as a pair of new structures, called the *ureteric buds*, orignate from the distal portion of the mesonephric duct (Wolffian) and come in contact with the condensing blastema of *metanephric mesenchyme* at about the 28th day.

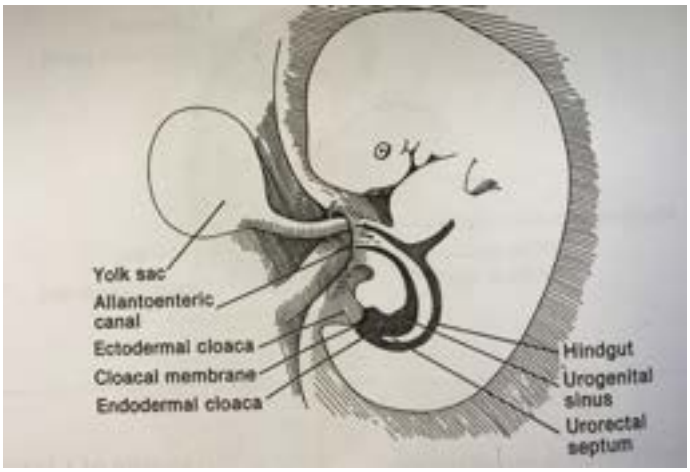
Between the sixth and ninth weeks the kidneys ascend to a lumbar site just below the adrenal glands.

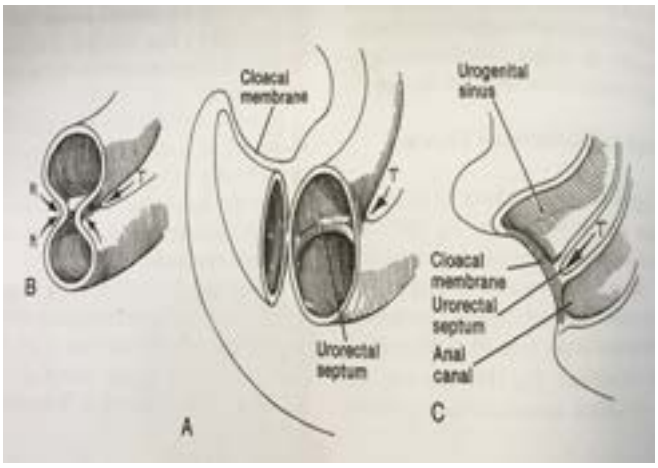
Bladder and ureter development

Formation of the urogenital sinus:

At the third week of gestation the cloacal membrane remains a bilaminar structure composed of endoderm and ectoderm.

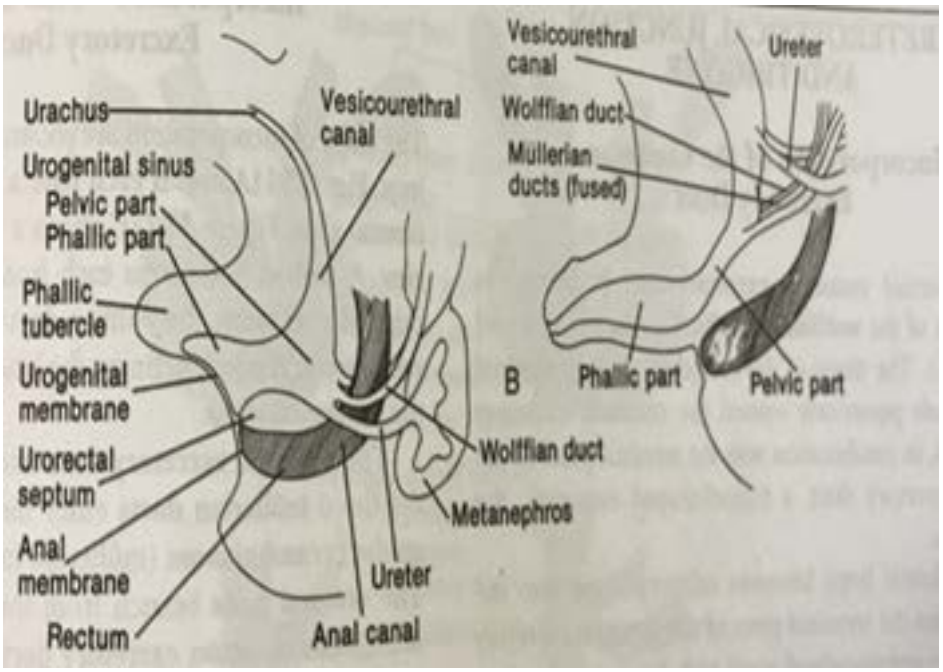
Division of the cloaca into an anterior urogenital sinus and a posterior anorectal canal occurs by the midline fusion of two lateral ridges of the cloacal wall and by a descending **urorectal septum** (a tongue of mesenchyme).





The mesonephric (wolffian) duct fuses with the cloaca by the 24th day and remains with the urogenital sinus during the cloacal separation.

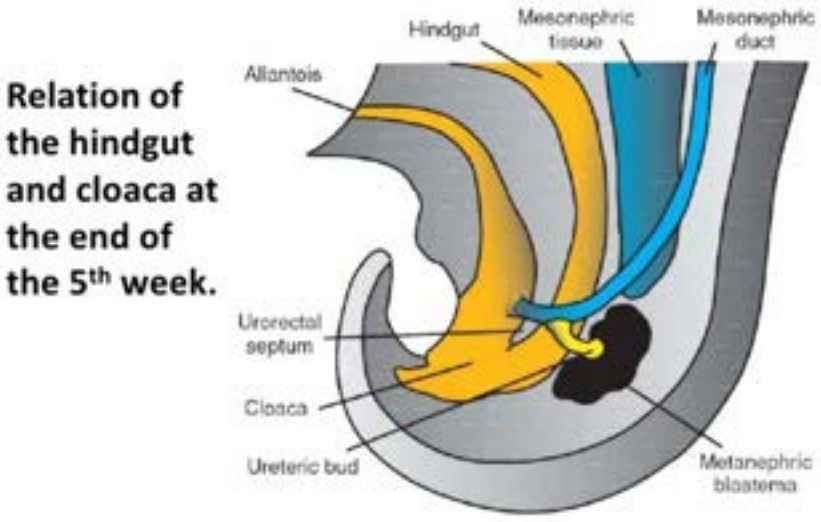
The entrance of the nephric duct into the primitive urogenital sinus serves as a landmark distinguishing the cephalad vesicourethral canal from the caudal urogenital sinus.



The vesicourethral canal

Gives rise to the bladder and pelvic urethra, whereas **the caudal urogenital sinus** forms the phallic urethra for males and distal vaginal vestibule for females.

According to the classic view the right and left common excretory ducts fuse in the midline as a triangular area, forming the primitive trigone, structurally different from bladder and urethra



The ureteric bud penetrates the metanephric mesoderm (blastema).

Development of the ureter

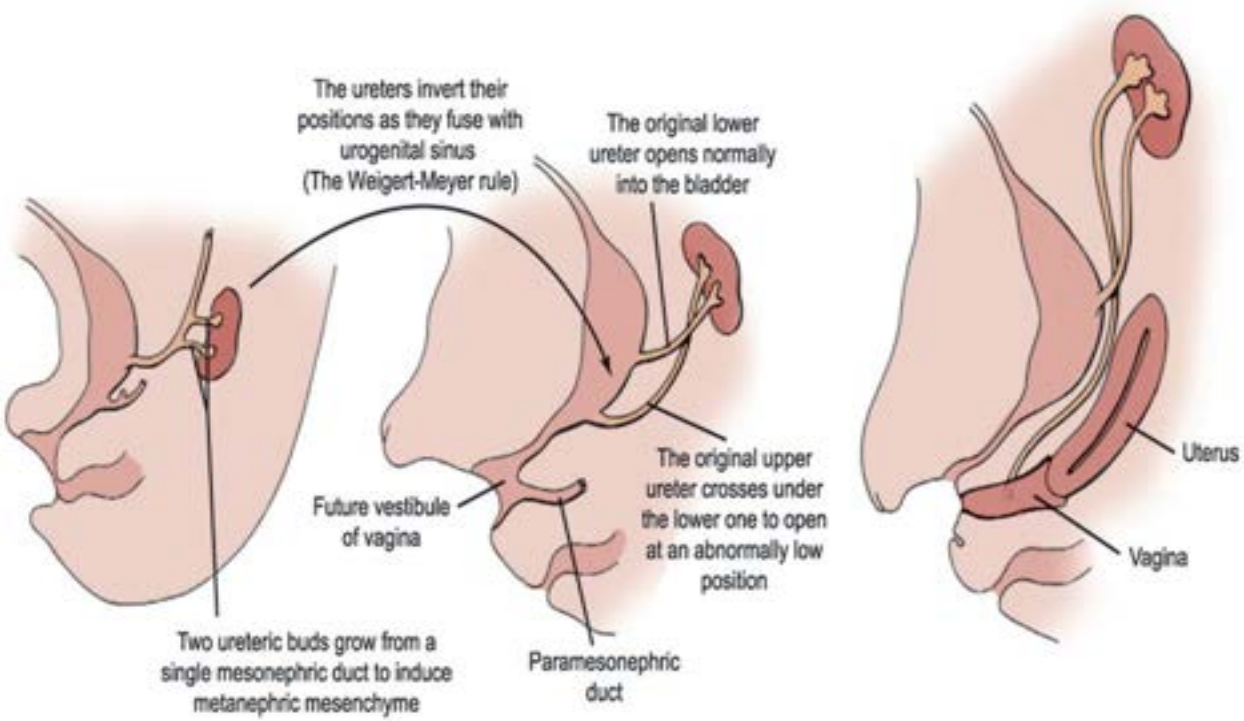
According to **Weigert-Meyer rule**

1- An abnormally lateral lower pole ureteral orifice may result from a ureteric bud arising too low on the nephric duct, therefore resulting in premature incorporation and migration within the developing bladder. In such a ureteral orifice, vesicoureteral reflux is more likely to occur due to an inadequate intramural tunnel.

2- In contrast, an abnormally caudal upper pole ureteral orifice may result from a ureteric bud arising too high on the nephric duct.

It may drain at the bladder neck and verumontanum or remain connected to the nephric (wolffian) duct derivatives such as the vas deferens in **males**

In **females**, the ectopic upper pole ureter may insert into the remnants of the nephric ducts (e.g., Gartner duct cyst) or vaginal vestibule

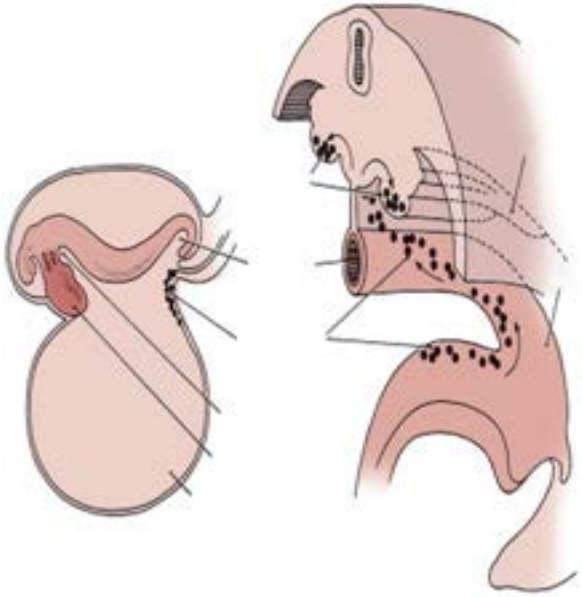


Development of gonads

During the **fifth week**, primordial germ cells migrate from the yolk sac along the dorsal mesentery to populate the mesenchyme of the posterior body wall near the 10th thoracic level.

In both sexes the arrival of primordial germ cells in the area of future gonads serves as the signal for the existing cells of the mesonephros and the adjacent coelomic epithelium to proliferate and form a pair of **genital ridges** just **medial** to the developing mesonephr

During the sixth week the cells of the genital ridge invade the mesenchyme in the region of future gonads to form aggregates of supporting cells called the **primitive sex cords**.



The primitive sex cords will subsequently **invest** the **germ cells** and support their development.

The genital ridge mesenchyme containing the primitive sex cords is divided into the cortical and medullary regions

Development of the male genital organs

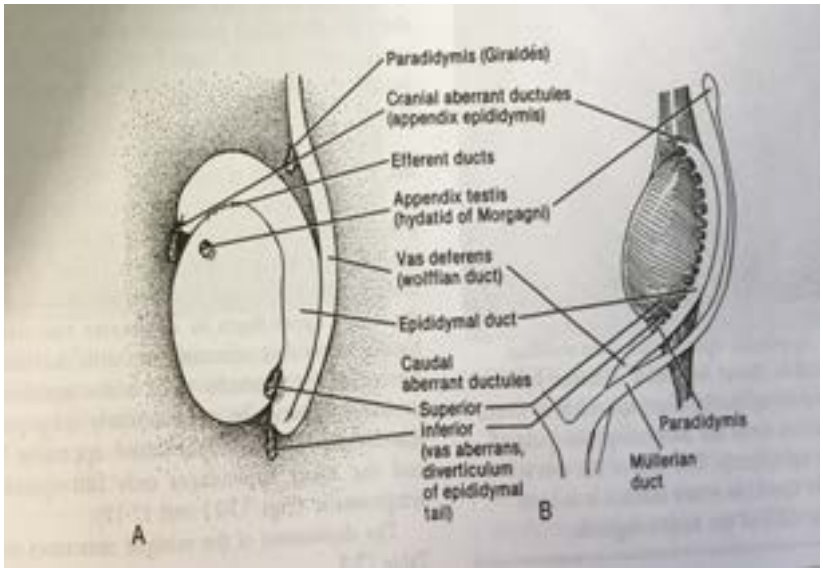
Under the influence of *SRY* (the *S*ex-determining *R*egion of the *Y* chromosome), cells in the medullary region of the primitive sex cords begin to differentiate into *Sertoli cells,* while the cells of the cortical sex cords degenerate.

Sex cord cells differentiate into Sertoli cells only if they contain the SRY protein; otherwise the sex cords differentiate into ovarian follicles.

During the **seventh week**, the differentiating Sertoli cells organize to form the ***testis cords***. At puberty these testis cords associated with germ cells undergo canalization and differentiate into seminiferous tubules.

The testis cords distal to the seminiferous tubules also develop lumen and differentiate into a set of thin-walled ducts called the ***rete testis***.

The tubules of rete testis connect with 5 to 12 residual tubules of nephric ducts, called ***efferent ductules.***



As the developing Sertoli cells begin their differentiation in response to the SRY protein they also begin to secrete a glycoprotein hormone called ***müllerian-inhibiting substance* (MIS).**

MIS causes the paramesonephric (müllerian) ducts to regress rapidly between the 8th and 10th weeks.

Small müllerian duct remnants can be detected in the developed male as a small tissue protrusion at the superior pole of the testis, called the ***appendix testis***, and as a posterior expansion of the prostatic urethra, called the ***prostatic utricle.***

Prostate and Seminal Vesicle Development

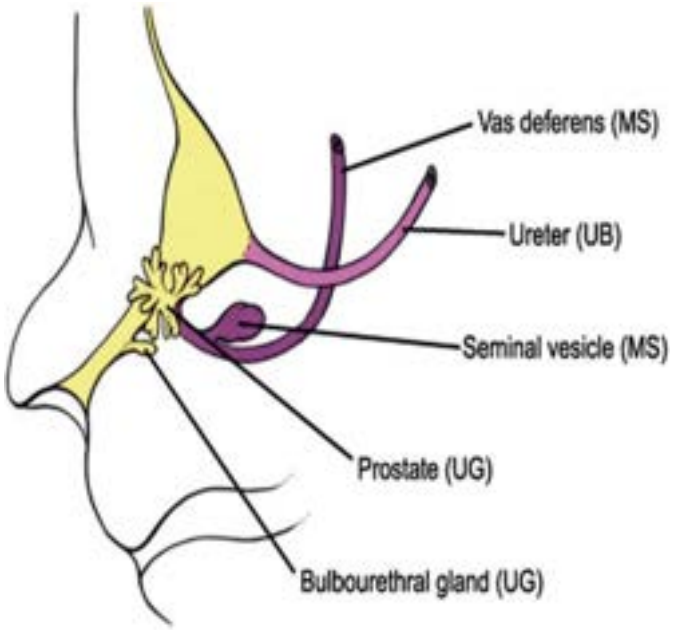
The seminal vesicles

Originate from the distal nephric ducts, whereas the prostate and bulbourethral glands develop from the urogenital sinus.

They therefore have different embryologic origins.

The initial event in prostatic development is an outgrowth of solid epithelial cords from the urogenital sinus epithelium into the surrounding mesenchyme during weeks 10 to 12 of gestation.

Circulating androgens produced by fetal testes play a critical role in the development of the prostate



Development of Female Genital Structures

In female embryos the primitive sex cords do not contain the Y chromosome, do not elaborate SRY protein, and therefore do not differentiate into Sertoli cells.

In the absence of Sertoli cells and SRY protein, therefore, MIS synthesis, Leydig cell differentiation, and androgen production do not occur.

Consequently, male development of the genital ducts and accessory glands is **not stimulated** and female development ensues.

In females the primitive sex cords degenerate and the mesothelium of the genital ridge forms the secondary cortical sex cords.

These **secondary sex cords** invest the primordial germ cells to form the ovarian follicles.

The germ cells differentiate into oogonia and enter the first meiotic division as primary oocytes.

The follicle cells then arrest further germ cell development until puberty, at which point individual oocytes resume gametogenesis in response to a monthly surge of gonadotropins.

The distal tips of the paramesonephric ducts adhere to each other just before they contact the posterior wall of the urogenital sinus

The wall of the urogenital sinus at this point forms a small thickening called the *sinusal tubercle.*

As soon as the fused tips of the paramesonephric ducts connect with the sinusal tubercle the paramesonephric ducts begin to fuse in a caudal to cranial direction, forming a tube with a single lumen.

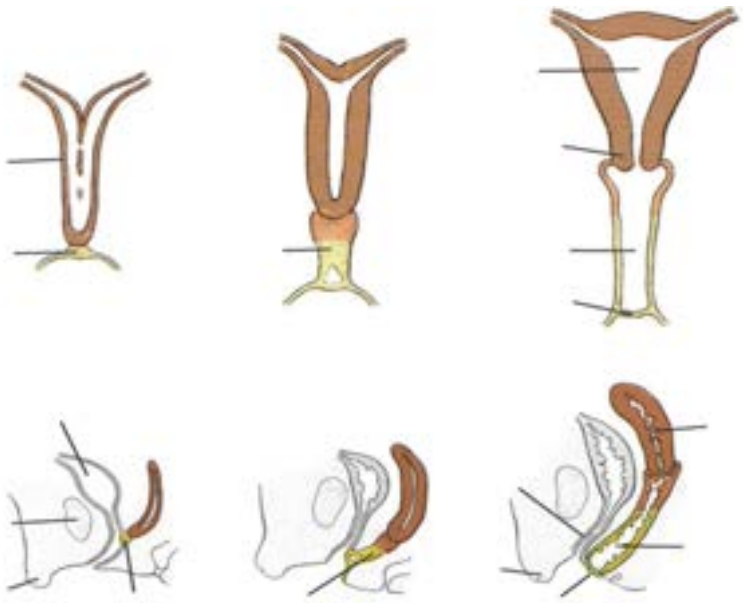
This tube, called the ***uterovaginal canal***, becomes the superior portion of the vagina and the uterus.

The unfused, superior portions of the paramesonephric ducts become the fallopian tubes (oviducts).

The funnel-shaped superior openings of the paramesonephric ducts become the infundibula.

The endodermal tissue of the sinusal tubercle in the posterior urogenital sinus continues to thicken, forming a pair of swellings called the *sinovaginal bulbs.*

These structures give rise to the lower third of the vagina.



Precursor	Male Organ	Female Organ
Indifferent gonad	Testis	Ovary
Primordial germ cells	Spermatozoa	Ova
Sex cords	Seminiferous tubules	Follicular cells
Mesonephric tubules	Efferent ductules, paradidymis, appendix epididymis	Epoöphoron
Wolffian (mesonephric) duct	Ductus deferens, seminal vesicles, epididymis	Gartner's canal
Müllerian (paramesonephric) duct	Appendix testis (hydatid), prostatic utricle	Fallopian tube, vagina (part)
Upper urogenital sinus	Bladder, prostatic urethra	Bladder, urethra
Lower urogenital sinus	Urethra	Vestibule
Genital tubercle	Penis	Clitoris
Genital folds	Penile urethra (floor)	Labia minora
Genital swellings	Scrotum	Labia majora

Development of External Genitalia

Initially, the cloacal membrane represents an elongated midline structure, which extends from the root of the umbilical cord to the future site of perineum distally.

This bilayered cloacal membrane "retracts" into the perineum as a result of cranial and medial migration of mesodermal cells into the anterior body wall

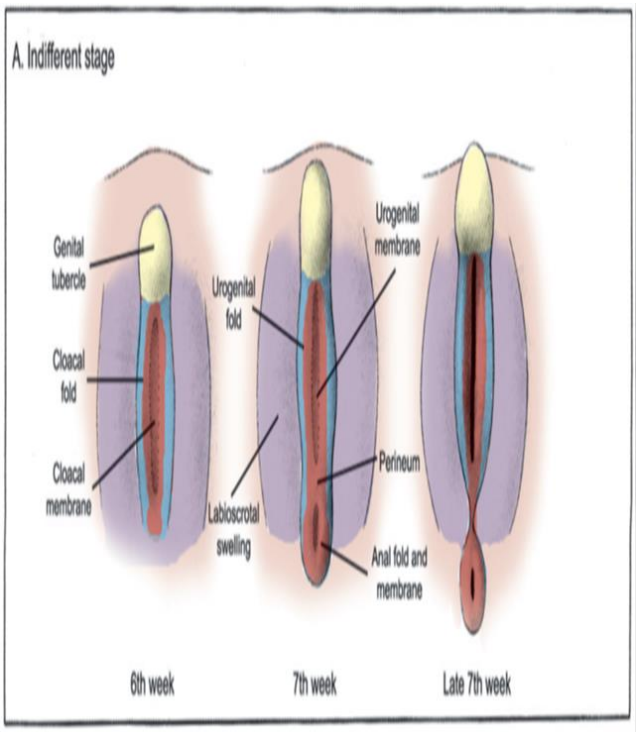
These migrating mesodermal cells give rise to the musculature of the medial portion of the anterior abdominal wall, the mesenchymal portion of the anterior bladder wall, the pubic symphysis.

Failure of migration of these mesodermal cells into the midline results in bladder exstrophy and other associated genital defects

Development of the external genitalia

The external genitalia derive from a pair of **labioscrotal swellings**, a pair of urogenital folds, and an anterior genital tubercle.

Male and female genitalia are morphologically similar until the seventh week

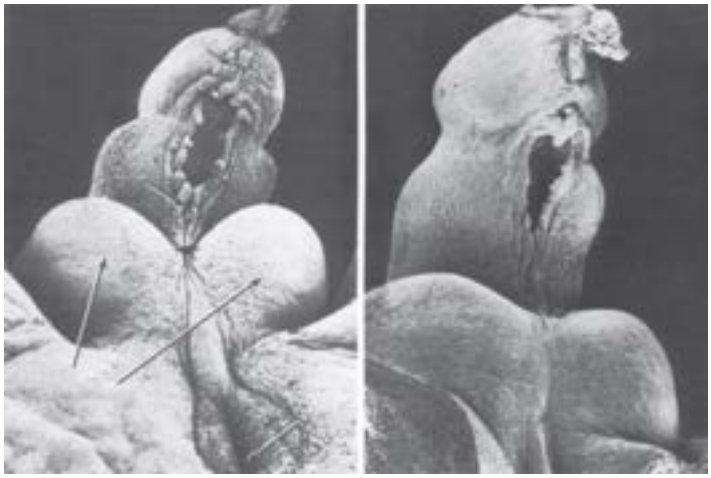


Development of the male external genitalia

In males, the urogenital folds fuse and the genital tubercle elongates to form the penile shaft and glans.

A small region of the distal urethra in the glans is formed by the invagination of surface epithelial tag.

The fused labioscrotal folds give rise to the scrotum.

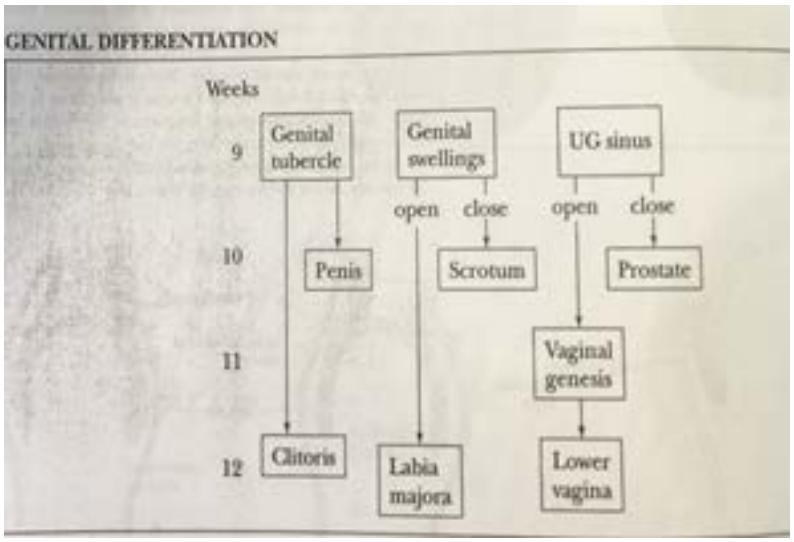


Development of the female external genitalia

In females the genital tubercle bends inferiorly to form the clitoris.

The urogenital folds remain separate to become the labia minora.

The unfused labioscrotal folds form the labia majora.



Gonadal Descent

Before gonadal differentiation, the testis lies near the developing kidney, loosely held in place by two ligamentous structures.

The dorsal ligament is referred to as **the cranial suspensory ligament (CSL),**

The ventral ligament later develops into **the gubernaculum.**

Between 10 and 15 weeks, the testis remains close to the future inguinal region during the enlargement of the abdominal cavity while the ovary moves more cranially.

The testis is anchored near the inguinal region by enlargement of the gubernaculum and regression of the CSL

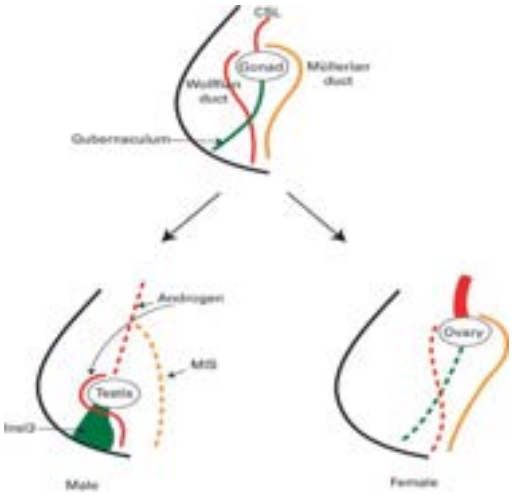
In females, the CSL continues to develop, keeping the ovary close to the kidney while the gubernaculum involutes.

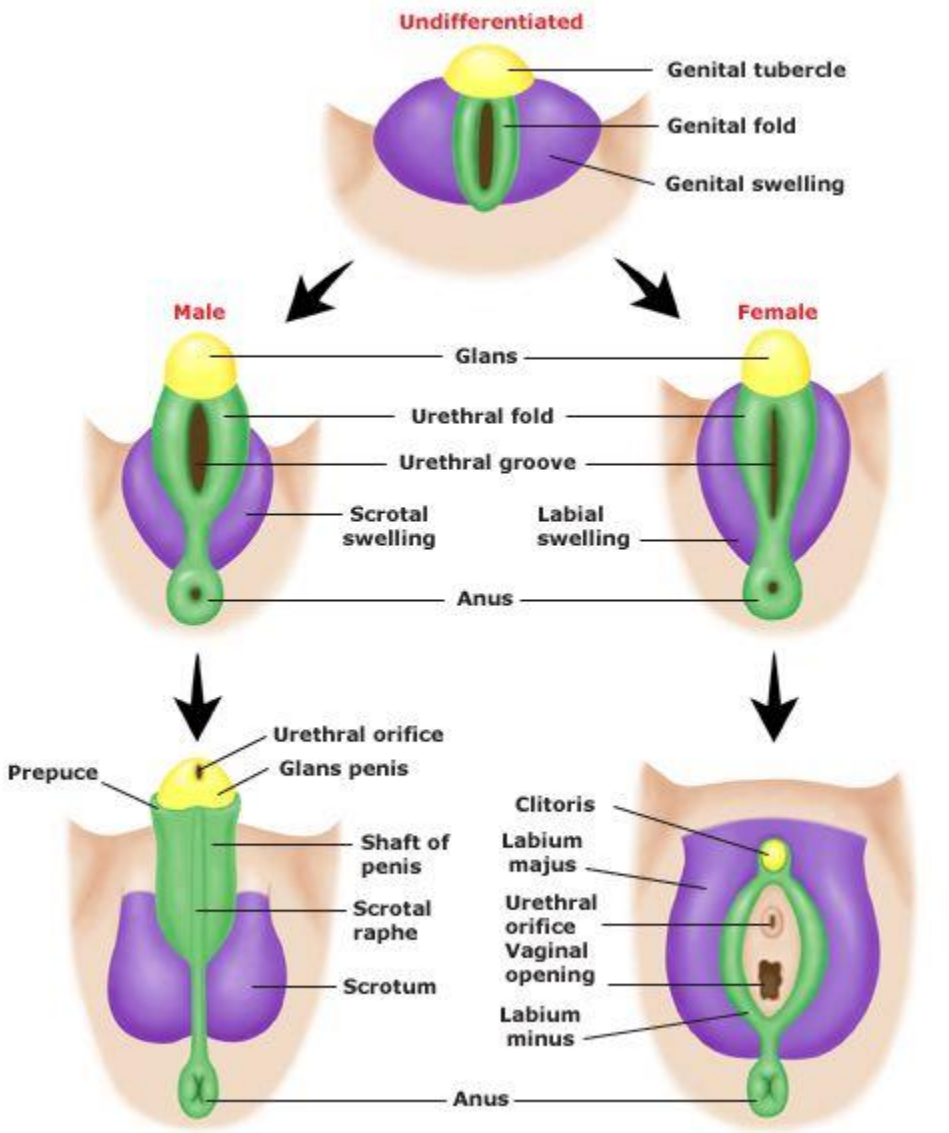
In males, androgen induces resorption of the CSL while the gubernaculum enlarges to become a ligamentous body, "holding" the testis close to the inguinal region.

Role of androgen in testicular descent still remains unclear

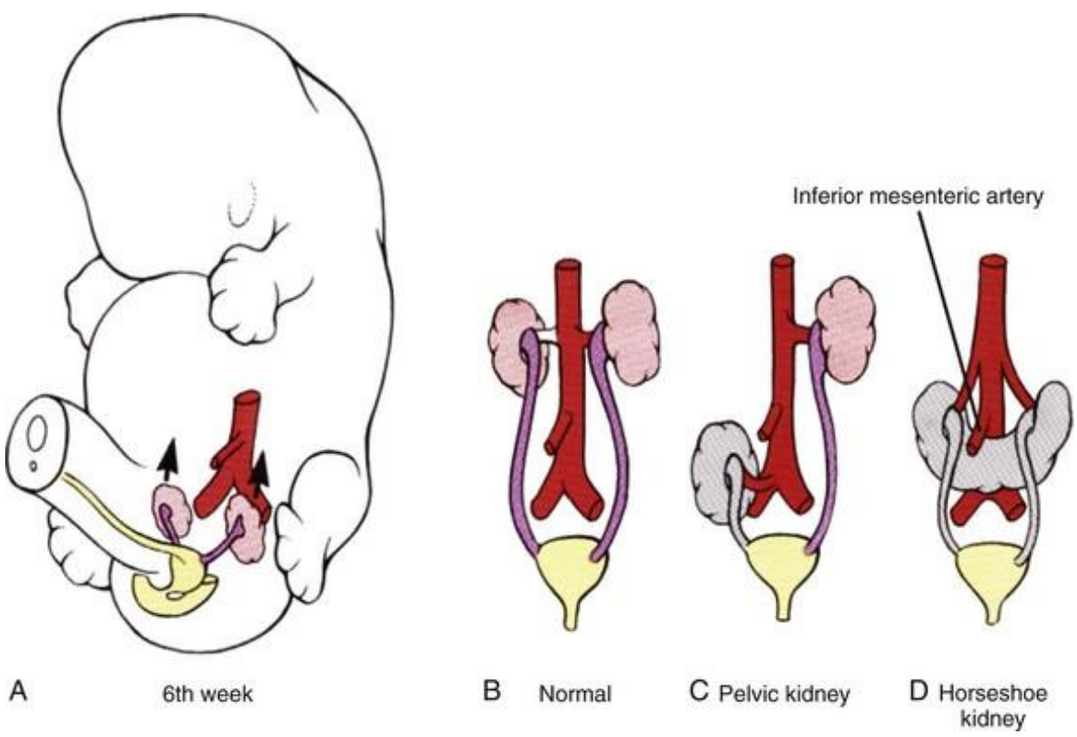
It has a role in the regression of the CSL.

The second migratory step (the inguinoscrotal phase) is thought to be more androgen dependent.





CONGENITAL ANOMALIES OF THE URINARY TRACT



ANOMALIES OF THE KIDNEY:-

1- Anomalies in number :

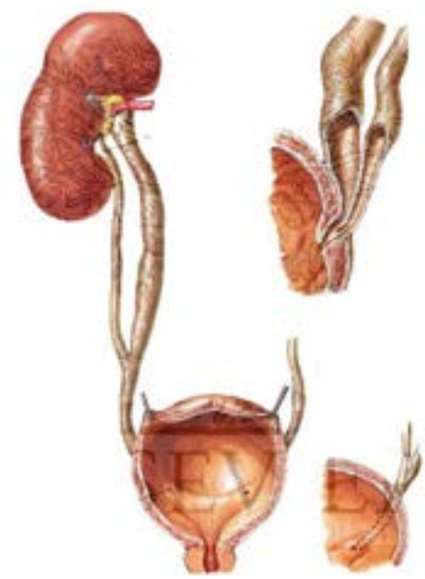
a) Duplex kidney:

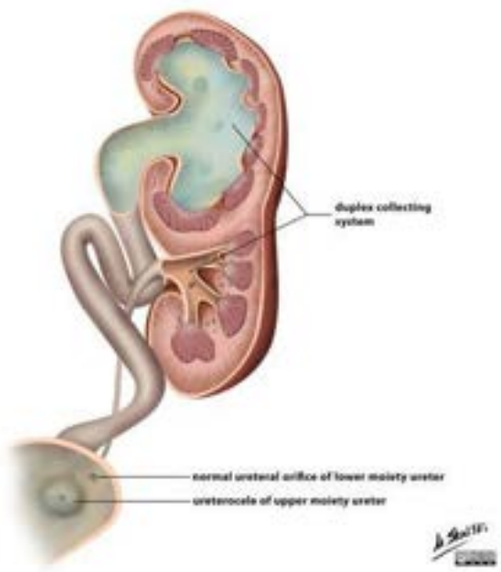
Due to 2 ureteric buds (complete duplication) or early branching of urteric bud (incomplete duplication or bifid system.

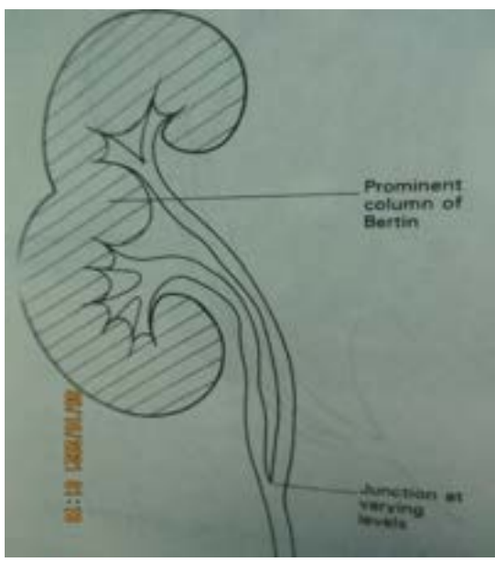
- Complete duplication: upper moiety is smaller (1/3 of renal mass) & its ureter develop ureterocele and open inferior on the trigone . The lower moiety is bigger (2/3 of renal mass) and its ureter is refluxing and open superiorly on the trigone (Weigert –Mayer low). FIG
- Incomplete duplication (see – saw reflux)

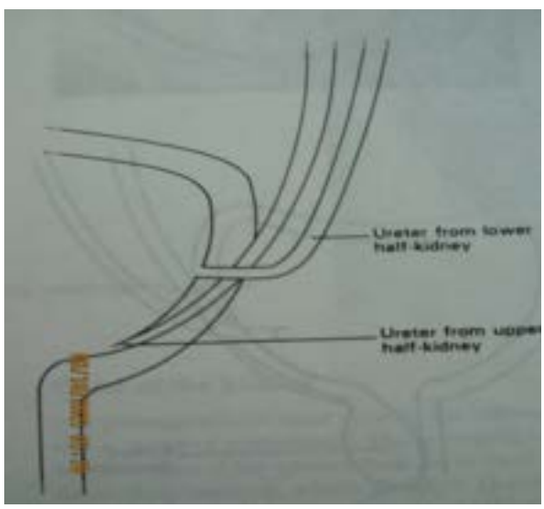
b) Single kidney (renal agenesis):

5% of general population. Ureteric bud maldeveloped or absent. Other kidney shows compensatory hypertrophy - There may be hemitrigone or absent ipsilateral testis









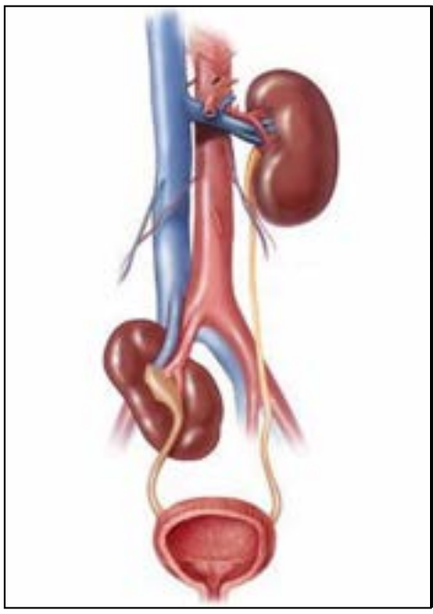
2- Anomalies of position :

A- ECTOPIC KIDNEY:

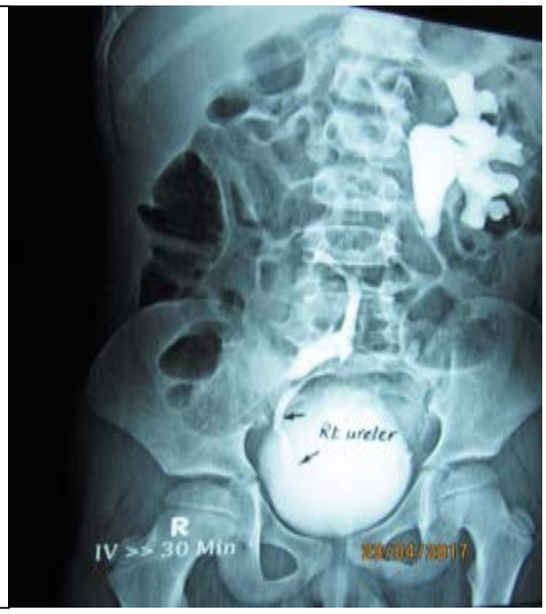
May be (low lumber – iliac or pelvic in position). Most common is the pelvic kidney.

It is malrotated with anterior renal pelvis and multiple blood supply entering its medial aspect.

Significance: it is badly drained – liable to stone formation – low in position suspect with other emergencies (appendicular mass and tubo-ovarian mass) – has multiple vessels and its approach is from lateral and anterior aspect.



Ectopic pelvic kidney



Ectopic Rt kidney & lt pujo

B- Crossed ectopia with or without fusion :

a- Left or right according to the side from whitch it crosses and its ureter open in its original site

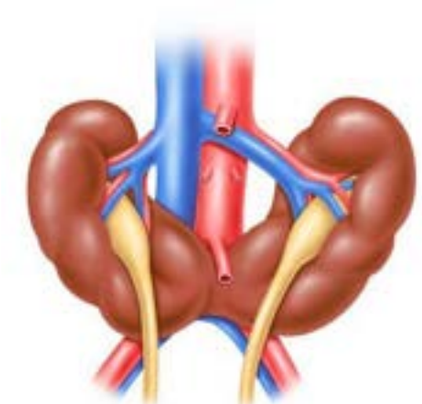
1- L- Shaped if crosses vertically
2- 2- S- shaped if it crosses horizontally.

3 – Anomalies of fusion:

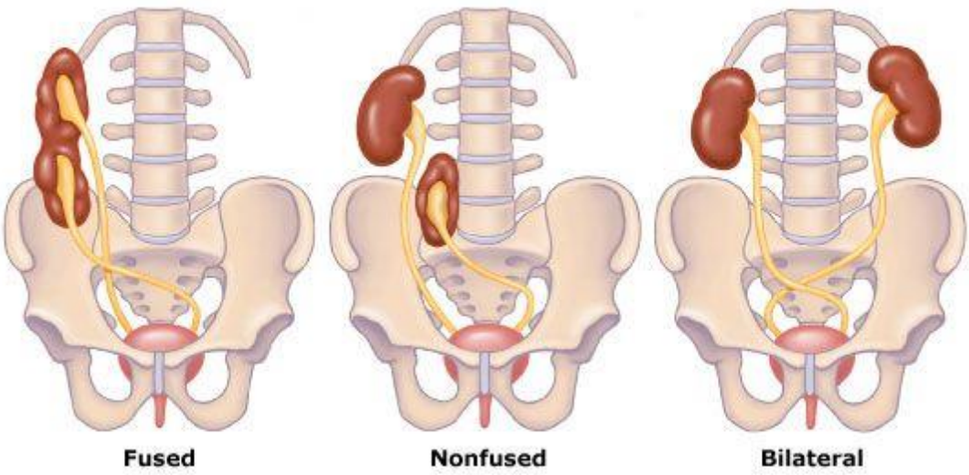
Horse – shoe kidney: is low in position (isthmus opposite 3rd lumbar vert. hampered by inferior mesenteric artery) – malrotated and medially displaced

- Has multiple vessels and high upj insertion. SO it is potentially obstructed and liable to stone formation.

a- Disc –shaped or pancake kidney if bilateral ectopic pelvic kidneys fused along their medial borders



b- Crossed renal ectopia with fusion (L OR S – shaped kidneys).



4 – Vascular anomalies:

Usually there is single renal artery in 75% of population = vascular anomalies in 25%, the most common is aberrant lower polar artery that may cross behind PUj causing puj obstruction. Actually it is an early branching of the renal artery.

5-Cystic anomalies:

a- **Adult polycystic kidney disease**: its bilateral, hereditary dominant. Kidneys are replaced by multiple innumerable cysts (intrarenal obstruction)
Presentaion : i- intrauterine by large fetal abdomen that obstruct labour ii- adult presentation by dragging loin pain - hypertension – recurrent uti – stones or haematuria
Causes of hematuria: stones – ruptured renal varices or papillary cystadenocarcinoma) – finally chronic renal failure

- **Management:** conservative with ttt of complications: uti – stones –hematuria and finally renal failure by hemodialysis or renal transplant from nonrelated donor.

B - Multicystic **kidney**: due to early ureteral atresia. Unilateral – non-functioning – replaced by multiple numerable cysts – parenchyma in between isdysgenetic

TTT – conserve if moderate sized or do LAP nephrectomy if large

C- **Simple renal cyst(s)** : commonly polar – thin smooh wall – clear awater like fluid – may be multiple or complicate (infection – trauma – hemorrhage – malignant degeneration)
TTT: conservative assurance – interfere if hugely enlaged , or obstruct or complicate by aspiration and injection of sclerosant OR by marsupialization

6 – Hypoplastic kidney:

This is a small kidney in all parameters: small size – smaller parenchymal mass – smaller and fewer calyces- small ureter.

Presentation: hypertension – renal pain – recurrent UTI

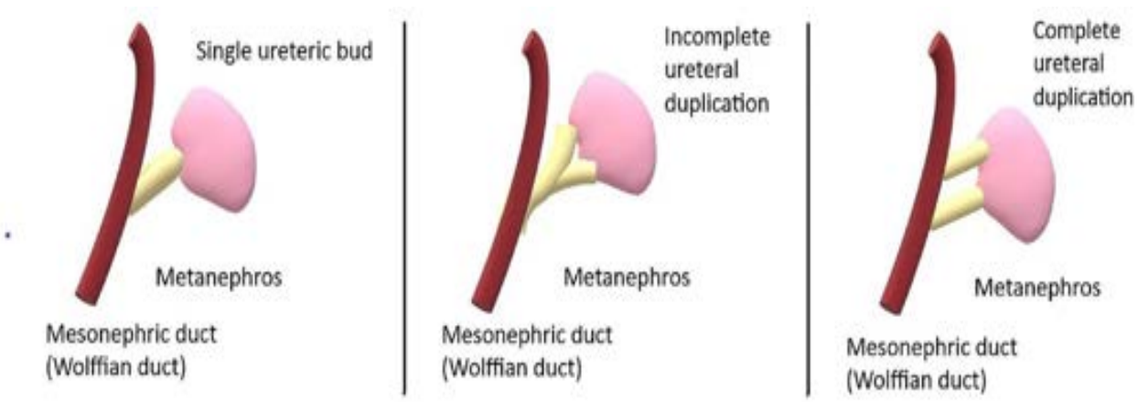
DD: Small kidney (poststrepto coccal chronic GN – Post chronic pyelonephritis – renal artery stenosis – after partial nephrectomy.)

TTT: treatment of complications – if hypertension or non-function → nephrectomy.

URETERIC ANOMALIES

1- URETERAL DUPLICATION :

- Duo to double ureteric bud: upper moiety ureter open inferiorly in the trigone and develop ureterocel e and the lower moiety ureter open superiorly and runs a short course so it's refluxing. The two ureters run inside a common sheath so dilation of one of them may obstruct the other.
- TTT. If obstruction or recurrent infection; → ureteroneocystostomy with common sheath. If refuse → incision of ureterocel then subureteric injection of deflux to correct VUR.



2- URETEROCELE :

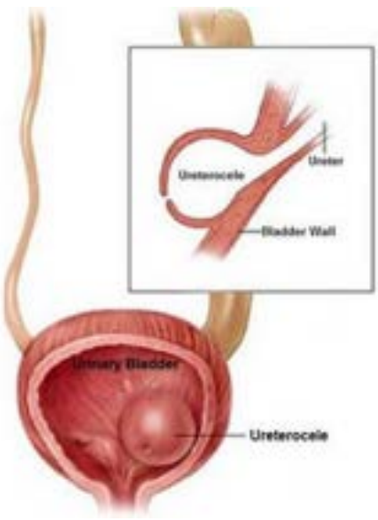
Cystic dilatation of intramural ueter due to orifice stenosis and weak support.

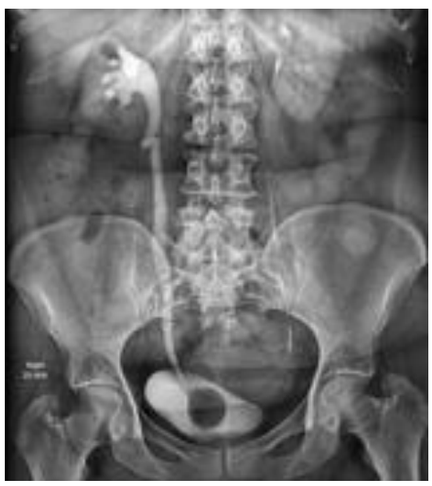
TYPES:

A)-Orthoptopic ureterocele > totally inside the bladder -.Mostly single system ureterocele and May associated with ipsilateral VUR or contralateral VUR. May form stones inside it and may reach a large size.

B) – ectopic ureterocele : may prolapsed through the bladder neck and protrude through the intoitus in female child . Common with double system.

TTT. : Incision as a temporary procedure – excision and antireflux as a definitive procedure.



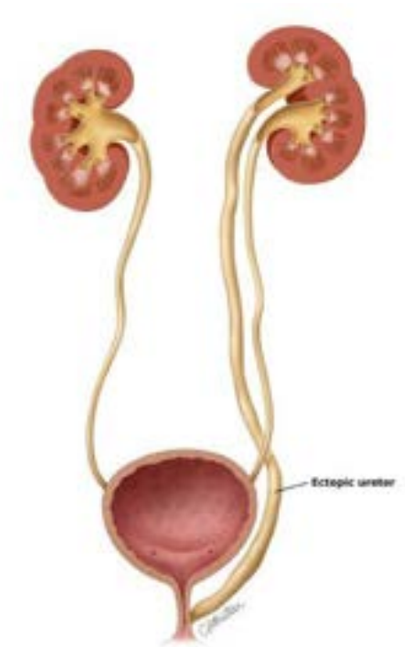


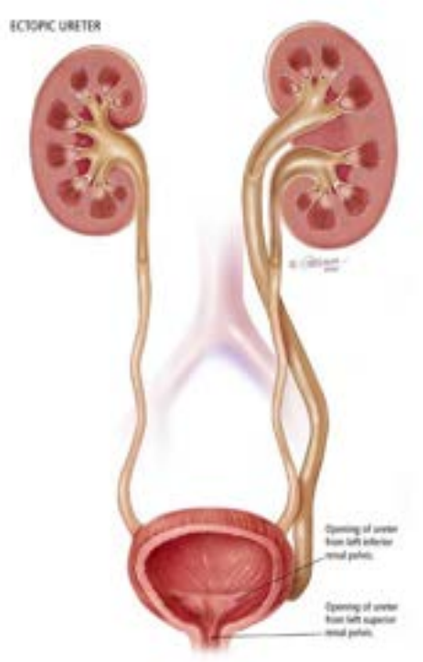
IVU SHOW RT URETROCLE

3- Ectopic ureter :

Ureter opens outside the trigone (bladder neck, seminal vesicle or vestibule in female causing paradoxic incontinence)

Ttt: if good functioning renal unit → ureteroneocystostomy with antireflux or do nephrectomy if no function ipsilateral kidney.





4- VESICOURETERIC REFLUX ;(primary VUR) :

Due to lack of backing of intravesical ureter or shortening of the intramural ureteral tunnel. It depend on the grade of reflux – stability of the bladder – associated Hutch diverticulum – presence of infection and age of the child

Grading system:

Grade I: - refflux doesn,t reach renal pelvis with or without ureteral dilatation)
Grade=II: - reflux reaches pelvicalyceal system without pelvic dilatation)
Grade III; - Mild to moderate ureteral and renal pelvis dialation
Grade IV: - Moderate dilatation of the ureter with or without kinking – blunt fornices
Grade V: - Gross dilatation and kinking of the ureter +/- intraparenchymal reflux.

Consequences: recurrent UTI → focal renal scarring → hypertension and further renal deterioration. This depends on reflux grade, uti , concomitant anomalies

Diagnosis: Lab workup - VCUG or nuclear scan +- IVU +- CYSTOURETHROSCOPY to categorize ureteral orifice shape and to do positioned instillation of contrast material.

Management; THE goal of ttt is to avoid late complications (eg reflux nephropathy). This depends on age and sex of the child and grade of reflux

1 year ----------→ conservative ttt (antibiotic prophylaxis)

1-5 years ---→ grade 1-3 (conservative ttt) ---grade IV TO V -→ surgery

- More than 5 years
  (Boys → rarely surgery
  Girls → surgery due to higher rate of uti).
  1-Further indications of surgery: recurrent febrile infections despite antibiotic
  2- Additional abnormalities

URINARY BLADDER ABNORMALITIES

1-Septated Bladder

2– Hour – glass bladder

3-Congenital bladder diverticulum:

urachal divericulum from the dome of the bladder – Hutch divericulum (paraureteric and may reach large size)

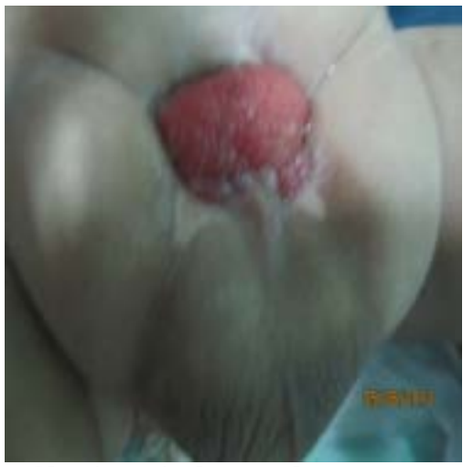
5- Urachal anomalies

(Patent urachus – urachal sinus – urachal diverticulum – urachal cyst)

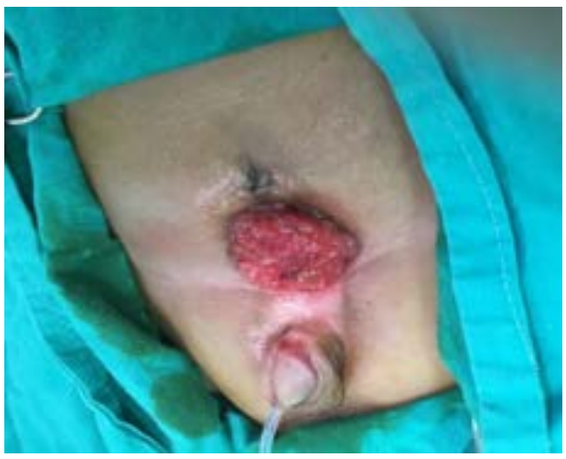
6- Vesical extrophy :

The bladder, bladder neck the whole urethra as well as the infraumblical part of the anterior abdominal wall and symphysis pupis are opened.

There is lateral rotation of both femora and bilateral hip subluxation →waddling gait and shortening of the inguinal canal leading to bilateral hernias



Classic vesical extrophy



Isolated vesical extrophy (very rare)

Treatment is multidisplinary:

1- Closure of the bladder within 48 hours after birth

2- Epispadias repair at age 3 years

3- Bladder neck repair at 5 years

OR DO COMPLETE PRIMARY REPAIR OF EXTROPHY (CPRE)

URETHERAL ANOMALIES

1 – **PHIMOSIS:**

Congenital narrowing of the prepuce opening preventing retraction an the prepuce distends during micturition Leading to repeated inflammation and even urine retention.

TTT: - is urgent circumcision or dorsal slit.

NB

(PARAPHIMOSIS) IS an acquired condition resulting from repeated inflammation of the preputial ring so when forcibly retracted

This leads to glans edema and strangulation of the glans

TTT: - is urgent dorsal slit

2 – **Congenital external meatal stenosis**:
The most important and frequently missed anomaly if untreated may end by cronic retention and renal failure
TTT: - External meatotomy or better to do meatoplsty

3-HYPOSPADIAS
4 – EPISPADIAS
5: POSTERIOR URETHRAL VALVE
6 – Urethral duplication (complete or incomplete)
7 – Urethral cyst

HYPOSPADIAS

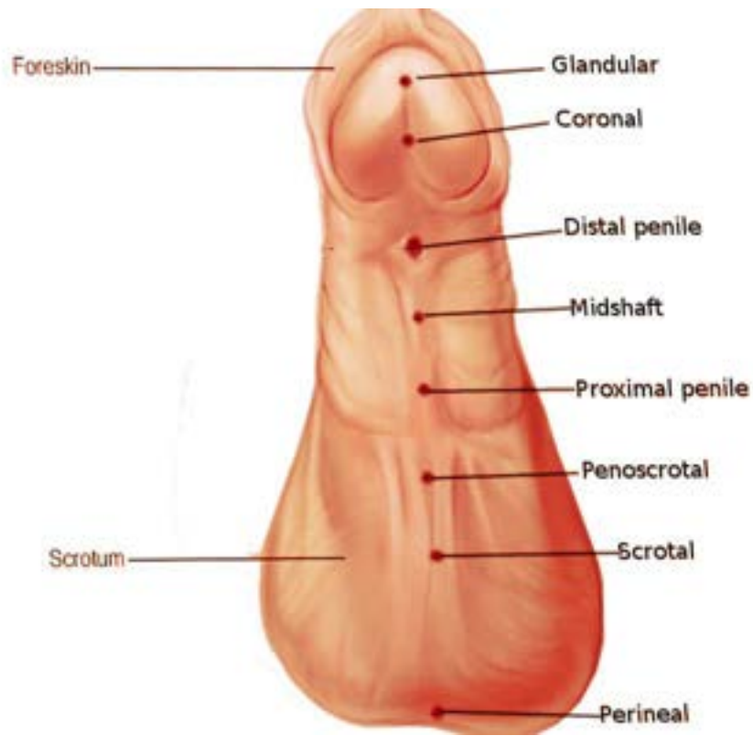
Definition: congenital anomaly 0f the phallic part of the urethra in which the urethra opens on the ventral aspect from the midperineum down to the glans penis.

Types: according to position of external meatus:

1 – Anterior (distal) variant and it includes; glandular, coronal, subcoronal and distal penile → 70 % of hypospadias cases

2 – Middle (midshaft) 10% of cases

3 – Proximal or posterior hypospadias and it includes (proximal penile, penoscrotal, scrotal and prineal or pseudovaginal variant → 20 %



INCIDENCE: one in 150 -300 live births – **common** in males and **rare** in females.

Etiology:

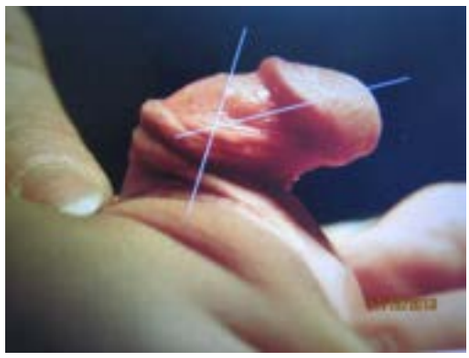
Unknown – common in twins due to placental insufficiency – mothers receiving contraceptive pills - run in families - elderly mothers.

Anatomical considerations: examine and assess the following:

1 - **Meatus:** position shape and size

2 – **urethral plate** ; part of the opened urethra from hypospadiac meatus up to glans tip – covered by glistening mucosa and has a fine capillary pattern (Measure length, width and depth orhe base degree of grooving) .

3- **Chordae** (dysgentic spongiosum extending from the meatus up to the base of the glans) It causes ventral bending of the penile shaft . Asses the degree of chordate before and after excision by doing artificial erection.



4 – **Prepuce:** ample, deficient or circumcised

5: **Glans**: size (small or normal for age of the child) – shape (conical or flat)

6 – **Perimeatal penile skin**: adherent or mobile – healthy or scarred

Timing of repair:

From **6m up to 18 months** according to expertise and facilities – if delayed due to ignorance of the parents: during the 4th year

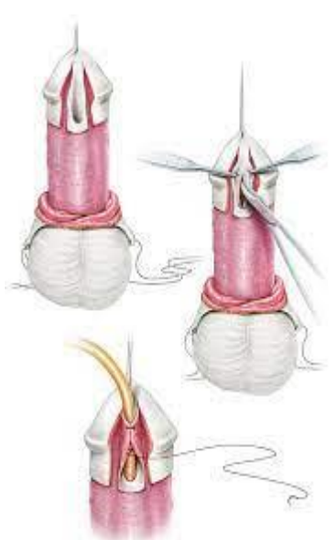
Aim of repair:

1- To create an apical meatus at glans tip for forward direction of urinary stream thypospadias micturates like a camel.
2- To have a straight penil shaft covered by normal non hairy non scarred penile skin
3- To have cosmetically acceptable external genitalia

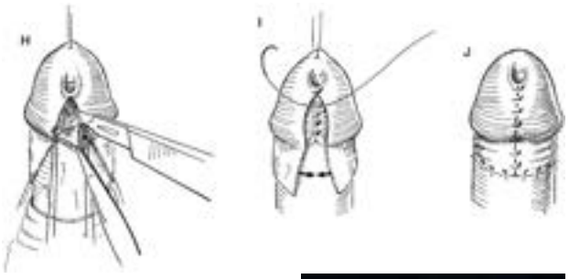
Steps of repair:

1- **Orthoplasty:-**
Or excision of chordae and artificial erection till you get a straight penis
(Types of chordae):
a- superficial or skin chordate excised during penile skin degloving
b – Deep or true chordate: used as additional coverage (y into I spongioplasty)
c- Ventral corporal disproportion manged by multiple fairy cuts d- congenital short urethra

2- **Urethroplasty:**
Depends on location of the meatus - width and depth of urethral plate – availability of local tisssues - peresence and degree of chordate and preference and experience of the surgeon.
FOUR catogories :
a- Meatal advancement and glanzplasty .
b- Urethral plate tubularization without (Duplay) or with dorsal incision (TIP) repair . c- Urethral plate augmentation (transverse preputial island flap repair)
d –urethral plate substitution (tubed flap or graft repair – Duckett tubed repair)



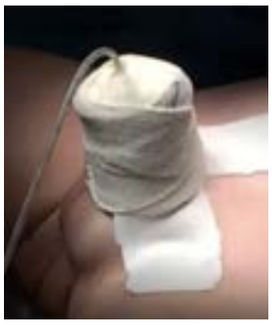
3- **GLANS Plasty**:
Creation of glans wings and closure over the repaied urethra.



4- **Skin cover**
To have a normally looking penis covered by non-hairy skin.

5- **Urine diversion**
By silastic stent.

6- **Dressing**
Cover the repair by porous compressive sponge of gauze



Follow up:
Outpatient office visits once weekly for one – 3 months: remove the stent after one week and notice the urinary stream and any complications.

COMPLICATIONS

1- Recent Postoperative : hematoma – edema – superficial infection – wound dehiscence
2- Delayed and late complications :
a- meatal problems (retraction – stenosis – disfigured meatus)
b- Urethral stricture and divericular formation
c – Fistula (most common complication – may reach 5-40 % according to operator – technique – age of the child).
d – Cosmetic complications (recurrent chordate – ugly scars – abnormally looking genitalia)

REDO SURGERY:
It carries high rate of complications specially fistula (40 % recurrence).

TESTICULAR MALDESCENT

It includes (retractile testis – undescended testis - ectopic testis and ascending testis)

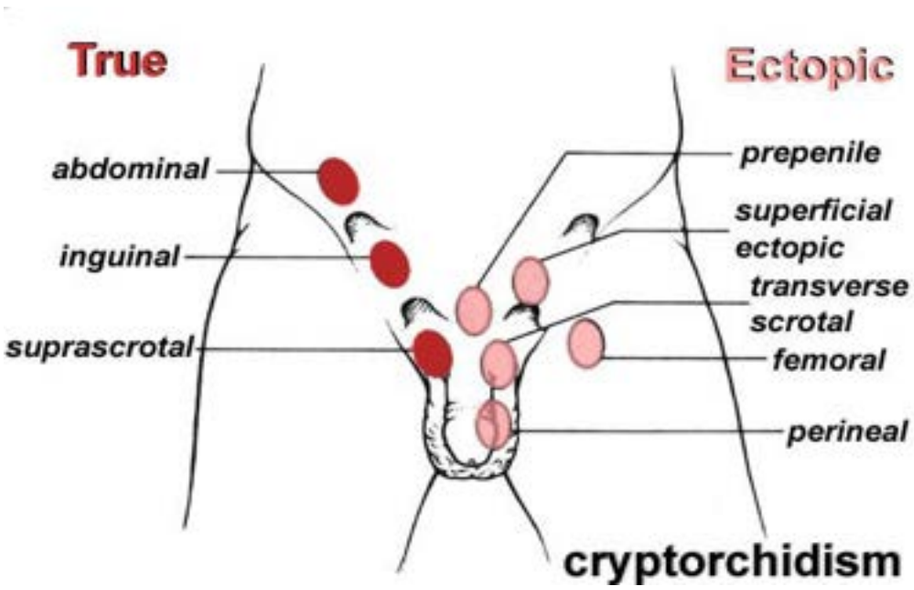
DEFINITIONS:

1 – UNDESCENDED TESTIS:
A Testis that failed to descend to the base of the scrotum and arrested at some point along its normal path of descent (NB; normal path of descent is from the lower pole of ipsilateral kidney, along the side wall of the pelvis through the inguinal canal to the base of the scrotum)

2 – **Ectopic testis**:
Is a testis that had been diverted away from its normal path of descent after it passed through the external inguinal ring to reside in an ectopic site (superficial subinguinal pouch -= most common site =, penopubic or prepubic; femoral triangle, perineal and transeptal to contralateral scrotal compartment).

3 – **RETRACTILE TESTIS**:
A Testis that ascends through external ring to be inguinal or even abdominal due to hyperactive cremasteric but descends to the base of the scrotum and stay there

4 – **Ascending testis**:
is the testis that was felt at the base of the scrotum at some time by the mother but now it is at a higher position and can't be brought down to the base of the scrotum;



INCIDENCE:

IN full term babies 3 -5 % of newly born babies.

In prematures: 30 % of newly born boys

By the end of the first year: only 0.5 % of full-term and 1% of pre-matures will remain with maldescent

Timing of orchiopexy:

12: 18 months Wait for one year for spontaneous descent and don't delay beyond 18 months before perminant histological changes which occur by 18 months of age (decline in number and size of seminefrous tubules and number of (**DARK**) spermatogonia per tubules - thickening of basement membrane of the tubules and decline in number of interstitial cells of Leyidig cells)

Aim of orchiopexy:

1 – Improve fertility potential (90 % in unilateral and decrease to 50 % in bilateral undescent) – Paternity is not affected in unilateral cases.

2 – To facilitate self-examination to early detect malignancy if it happens (seminoma after 40 yars old) – Undescent increases the malignant transformation potential to 80 times more than general population.

3 – To improve the psychic milieu of the child.

MANAGEMENT:

1- NO role for hormonal treatment in descent of a UNILATERAL undesceded testis. It may improve fertility potential (LHRH ANALOUGE).
2- In bilateral cases it may aid in spontaneous descent in 20% of cases and may improve fertility potential
3- Variable dosage regimens (HCG 400 UP TO 5000 IU IM for three doses.
SURGERY IS THE PRINCIPAL TREATMENT OF UNDESCENDED, ECTOPIC AND ASCENDING TESTIS

ACCORDING TO THE SITE OF THE TESTIS AND LATERALITY AND WHETHER THE TESTIS IS PALPABLE OR IMPALPABLE

A- **UNILTERAL PALPABLE TESTIS** (undescended , ectopic and ascending)-→ orchiolysis and scrotal orchiopexy in sub-dartos pouch

B- Bilateral undescended palpable

Testes: sequential orhiopexy separated by 3 months apart.

C- UNLATERAL IMPALPABLE TESTIS :

i- Laparoscopy and manage accordingly (look for the testicular vessels and the vas

ii- If low (below common iliac bifurcation) with long mesentry (the testis can reach contralateral internal ring) do orchiolysis and orchiopexy. Also if it is beebing through the internal ring. If the vessels and vas inter the internal ring do inguinal exploration → if testis size is good ; do orchiopexy if atrophic nubbin do orchiectony

iii- If high or short mesentry (if good testis size → do short reroting or Fawler – Steven staged or one stage if collateral circulation is good == if small sized with good scrotal contralateral testis → orchiectomy) .

D – BILATERAL IMPALPABLE TESTIS

(This is an intersex state – also in unilateral undescent with hypospadias → 25 % of intersex problem);

COMMONLY mistaken with a FEMALE CHILD WITH CONGENITAL ADRENAL HYPERPLASIA – CAH) ----→

I – Biochemical investigstions : a- basal testosterone level if low give HCG stimulation , if T is doubled -- > functioning testes .

2 – Karyotyping (46xy)

3 – Laparoscopy and manage the lower testis first and the better size first

Complications if left untreated:

1 Testicular atrophy especially the inguinal or perineal testis from compression and friction. The higher the testis the more liability to atrophy

2 – Testicular torsion in abdominal w long mesentry and atrophy due to higher abdominal temperature.

3 – Malignant generation especially in abdominal testis

4 – Drastic effects on fertility especially in bilateral cases,

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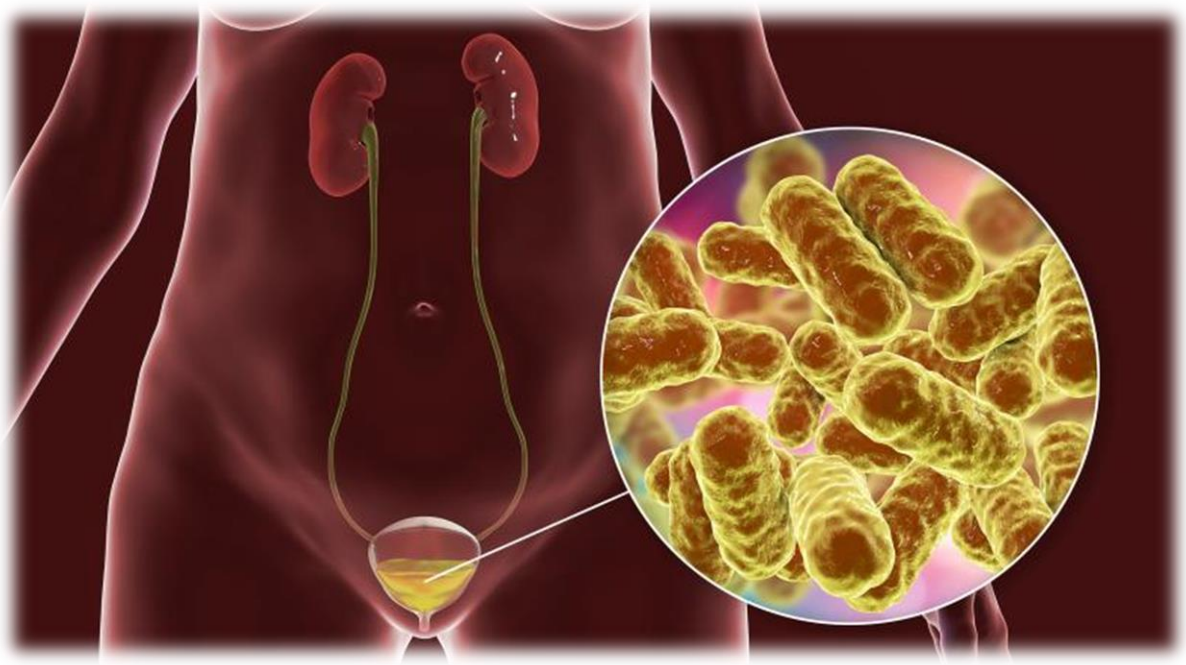
BACTERIAL INFECTIONS OF THE GENITOURINARY TRACT



Bacterial Infections of the Genitourinary Tract

Introduction

Urinary tract infection (UTI) is a term that is applied to a variety of clinical conditions ranging from the asymptomatic presence of bacteria in the urine to severe infection of the kidney with resultant sepsis.

EPIDEMIOLOGY

1- In newborns up to 1 year of age, bacteriuria is present in 2.7% of boys and 0.7% in girls. The incidence of UTI in uncircumcised males is higher than in circumcised males (1.12% compared with 0.11%) during the first 6 months of life.
2- In children between 1 and 5 years of age, the incidence of bacteriuria in girls increases to 4.5%, while it decreases in boys to 0.5%. Most UTIs in Children younger than 5 years are associated with congenital abnormalities of the urinary tract, such as vesicoureteral reflux or obstruction.
3- The incidence of bacteriuria remains relatively constant in children 6–15 years of age. However, the UTIs in these children are more likely to be associated with functional abnormalities of the urinary tract, such as dysfunctional voiding.
4- During adolescence, the incidence of UTI significantly increases (to 20%) in young women, while remaining constant in young men. The major risk factors for women 16–35 years of age are related to sexual intercourse and diaphragm use. Later in life, the incidence of UTI increases significantly for both males and females. For women between 36 and 65 years of age, gynecologic surgery and bladder prolapse appear to be important risk factors. In men of the same age group, prostatic hypertrophy/obstruction, catheterization, and surgery are relevant risk factors.
5- For patients older than 65 years, the incidence of UTI continues to increase in both sexes. Incontinence and chronic use of urinary catheters are important risk factors in these patients.

Table 14–1. Epidemiology of UTI by age, group, and sex.

	Incidence (%)		
Age (y)	Female	Male	Risk factors
<1	0.7	2.7	Foreskin, anatomic GU abnormalities
1–5	4.5	0.5	Anatomic GU abnormalities
6–15	4.5	0.5	Functional GU abnormalities
16–35	20	0.5	Sexual intercourse, diaphragm use
36–65	35	20	Surgery, prostate obstruction, catheterization
>65	40	35	Incontinence, catheterization, prostate obstruction

PATHOGENESIS

- Bacterial Entry

There are four possible modes of bacterial entry into the genitourinary tract.
1-periurethral bacteria ascending into the urinary tract causes most UTI. Most cases of pyelonephritis are caused by the ascent of bacteria from the bladder, through the ureter and into the renal parenchyma. Consequently, the short nature of the female urethra combined with its close proximity to the vaginal vestibule and rectum likely predisposes women to more frequent UTIs than men.
Other modes of bacterial entry are uncommon causes of UTI.
2-Hematogenous spread can occur in immunocompromised patients and in neonates.
3-Lymphatogenous spread through the rectal, colonic, and periuterine lymphatics has been postulated as a cause for UTI
4-Direct extension of bacteria from adjacent organs into the urinary tract can occur in patients with intraperitoneal abscesses or vesicointestinal or vesicovaginal fistulas.

- Host Defenses

Host factors have an essential role in the pathogenesis of UTI.
1-Unobstructed urinary flow with the subsequent washout of ascending bacteria is essential in preventing UTI.
2-The urine itself has specific characteristics (its osmolality, urea concentration, organic acid concentration, and pH) that inhibit bacterial growth and colonization. It also contains factors that inhibit bacterial adherence, such as Tamm–Horsfall glycoprotein
3-The epithelium lining the urinary tract not only provides a physical barrier to infection but also has the capacity to recognize bacteria in order to innate host defenses.
4-the normal flora of the periurethral area or the prostate and the presence of vesicoureteral reflux.
NB: - in children, the presence of vesicoureteral reflux does not increase their susceptibility to UTI but does allow bacteria to be inoculated into the upper tract and the infection to progress.

CAUSATIVE PATHOGENS

Most UTIs are caused by a single bacterial species.
1- At least 80% of the uncomplicated cystitis and pyelonephritis are due to **E. coli**, with most of pathogenic strains belonging to the O serogroups.
2- Other less common uropathogens include Klebsiella, Proteus, and Enterobacter spp. and enterococci.
3- In hospital-acquired UTIs, a wider variety of causative organisms is found, including Pseudomonas and Staphylococcus spp.
UTIs caused by S. aureus often result from hematogenous dissemination. Group B beta-hemolytic streptococci can cause UTIs in pregnant women.

DIAGNOSIS

There is a high false-positive rate, especially from bagged specimens. Suprapubic aspiration avoids potential contamination; however, due to its invasiveness, it is rarely used except in children and selected patients.
Urine obtained from a urinary catheter is less invasive than a suprapubic aspiration and is less likely to be contaminated than that obtained from a voided specimen. If a patient has an indwelling catheter, a urine specimen should be obtained from the collection port on the catheter.

- **Urine analysis**

The urine can be immediately evaluated for leukocyte esterase, Urinary nitrite
Microscopic examination of the urine for WBCs and bacteria is performed after centrifugation. When bacteria counts are >100,000 CFU/mL, bacteria can be detected microscopically.

- **Urine Culture**

The gold standard for identification of UTI is the quantitative culture of urine for specific bacteria. The urine should be collected in a sterile container and cultured immediately after collection

Table 14–3. Probability of UTIs based upon urine culture.

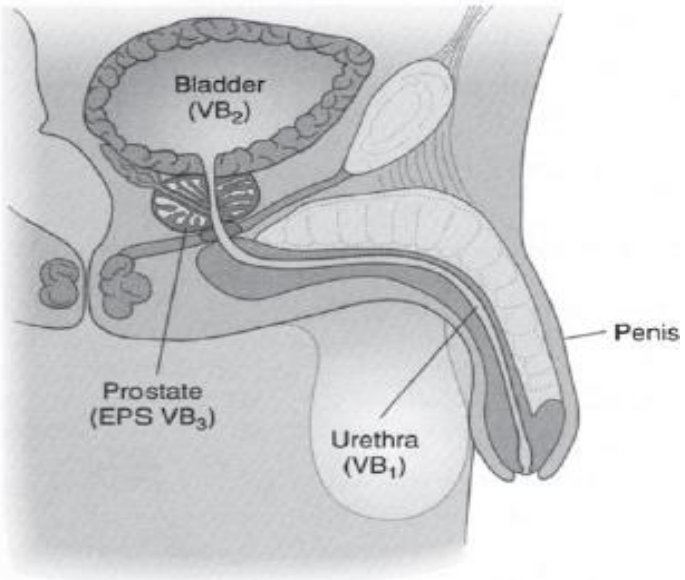
Collection	CFU	Probability of infection (%)
Suprapubic	Gram negative any Gram positive >1000	>99
Catheterization	$>10^5$ 10^{4-5} 10^{3-4} $<10^3$	95 Likely Repeat Unlikely
Clean catch		
Male	$>10^4$	Likely
Female	3 specimens: $>10^5$ 2 specimens: $>10^5$ 1 specimen: $>10^5$ 5×10^4–10^5 1–5 × 10^4 symptomatic 1–5 × 10^4 nonsymptomatic $<10^4$	95 90 80 Repeat Repeat Unlikely Unlikely

UTIs, urinary tract infections; CFU, colony-forming unit.

- **Localization Studies**

Occasionally, it is necessary to localize the site of infection.
1- For upper urinary tract localization, the bladder is irrigated with sterile water and a ureteral catheter is placed into each ureter. A specimen is collected from the renal pelvis.
2- A specimen is collected at the beginning of the void and represents possible infection in the urethra.
3- A midstream specimen is next collected and represents possible infection in the bladder.

4- The prostate is then massaged and the patient is asked to void again. This specimen represents possible infection of the prostate.



▲ **Figure 14–1.** Localization of lower urinary tract infection. A positive culture in the voided bladder urine specimen VB_1 suggests infection of the urethra, while in VB_2, an infection of the bladder, and in EPS or VB_3, an infection of the prostate.

ANTIBIOTICS

Table 14–6. Recommended antimicrobial agents and duration of therapy based upon the type of UTI.

Diagnosis	Pathogen	Choice of antibiotics	Duration of therapy
Cystitis	*E. coli* *Klebsiella* *Proteus*	1st: TMP-SMX 2nd: Fluoroquinolone	1–3 days
Pyelonephritis	*E. coli* *Proteus* *Klebsiella* *Enterobacteria*	1st: Gluoroquinolone 2nd: 2nd generation cephalosporin 3rd: Aminopenicillin/BLI	7–10 days
Complicated UTI	*E. coli* *Enterococci* *Pseudomonas* *Staphylococci*	1st: Fluoroquinolone 2nd: Aminopenicillin/BLI 3rd: 3rd generation cephalosporin Aminoglycosides	3–5 days after afebrile
Prostatitis	*E. coli* *Enterobacteria* *Pseudomonas* *Enterococci*	1st: Fluoroquinolone 2nd: 2nd generation cephalosporin 3rd: 3rd generation cephalosporin	Acute: 2 weeks Chronic: 4–6 weeks
Epididymitis	*E. coli* *Enterobacteria* *Enterococci* *Chlmaydia* *Ureaplasma*	1st: Fluoroquinolone 2nd: 2nd generation cephalosporin 1st: Doxycycline 2nd: Macrolide	2 weeks

BLI, beta-lactamase inhibitor; TMP-SMX, trimethoprim plus sulfamethoxazole.

Table 14–5. Recommended antimicrobial agents for common genitourinary pathogens.

Bacteria	Oral therapy	Parenteral therapy
Gram-positive cocci		
Staphylococcus aureus	Nafcillin, nitrofurantoin, ciprofloxacin	Nafcillin, vancomycin
Staphylococcus epidermidis	Ampicillin, nitrofurantoin, ciprofloxacin	Ampicillin, penicillin G
Staphylococcus saprophyticus	Ampicillin, nitrofurantoin, ciprofloxacin	Ampicillin, penicillin G
Streptococcus, group D *S. faecalis* (enterococci) *S. bovis*	 Ampicillin, nitrofurantoin Penicillin G, ampicillin	 Ampicillin plus gentamicin Ampicillin, vancomycin
Streptococcus, group B	Ampicillin, cephalosporin	Ampicillin, cephalosporin
Gram-negative cocci		
Neisseria gonorrhoeae	Ciprofloxacin plus doxycycline	Ceftriaxone
Gram-negative rods		
Escherichia coli	TMP-SMX, ciprofloxacin, nitrofurantoin	Gentamicin
Enterobacter spp.	TMP-SMX, ciprofloxacin, nitrofurantoin	Gentamicin plus piperacillin
Gardnerella vaginalis	Metronidazole, ampicillin	Metronidazole
Klebsiella spp.	TMP-SMX, ciprofloxacin	Gentamicin plus cephalosporin
Proteus spp.	Ampicillin, TMP-SMX, ciprofloxacin	Ampicillin, gentamicin
Pseudomonas aeruginosa	Carbenicillin, tetracycline, ciprofloxacin	Gentamicin plus piperacillin
Serratia spp.	TMP-SMX, carbenicillin	TMP-SMX, amikacin
Other pathogens		
Chlamydiae	Tetracycline, erythromycin	Tetracycline, erythromycin
Mycoplasmas, ureaplasmas	Tetracycline, erythromycin	Tetracycline, erythromycin
Obligate anaerobes	Metronidazole, clindamycin	Metronidazole, clindamycin

TMP-SMX, trimethoprim plus sulfamethoxazole.

CLINICAL PRESENTATION

- **KIDNEY INFECTION**

1-Acute Pyelonephritis

Acute pyelonephritis is defined as inflammation of the kidney and renal pelvis, and its diagnosis is usually made clinically.

A. Presentation and Findings

Patients with acute pyelonephritis present with chills, fever, and costovertebral angle tenderness.
Lower-tract symptoms such as dysuria, frequency, and urgency.
Sepsis may occur, with 20–30% of all systemic sepsis resulting from a urine infection.
E. coli is the most common causative organism, accounting for 80% of the cases.

B. Radiographic Imaging

Contrast-enhanced computed tomography (CT) scans can accurately demonstrate findings, confirming the diagnosis of pyelonephritis.
Radionuclide study with 99mTc-dimercaptosuccinic acid is equally sensitive in detecting the perfusion defects of pyelonephritis.
Renal ultrasonography is important to rule out concurrent urinary tract obstruction but cannot reliably detect inflammation or infection of the kidney.

C. Management

The management of acute pyelonephritis depends on the severity of the infection.
In patients who have toxicity because of associated septicemia, hospitalization is warranted.
If bacteremia is present, parenteral therapy should be continued for an additional 7–10 days and then the patient should be switched to oral treatment for 10–14 days.
In patients who are not severely ill, outpatient treatment with oral antibiotics is appropriate.
For adults, treatment with fluoroquinolones or TMP–SMX is well tolerated and effective.
Therapy should continue for 10–14 days.

2-Emphysematous Pyelonephritis

Emphysematous pyelonephritis is a necrotizing infection characterized by the presence of gas within the renal parenchyma or perinephric tissue. About 80–90% of patients with emphysematous pyelonephritis have **diabetes**; the rest of the cases are associated with urinary tract obstruction from calculi or papillary necrosis.

A. Presentation and Findings

Fever, flank pain, and vomiting that fails initial management with parenteral antibiotics. Pneumaturia may be present. Bacteria most frequently cultured from the urine include *E*.coli, Klebsiella pneumoniae, and Enterobacter cloacae.

B. Radiographic Imaging

Gas overlying the affected kidney may be seen on a plain abdominal radiograph [kidneys, ureters, bladder (KUB)].
CT scan is much more sensitive in detecting the presence of gas in the renal parenchyma than renal ultrasonography.

C. Management

Prompt control of blood glucose and relief of urinary obstruction are essential, in addition to fluid resuscitation and parenteral antibiotics. The mortality rate is 11–54%.
In combination with medical treatment, percutaneous drainage appears to be helpful in accelerating resolution of the infection and minimizing the morbidity and mortality of the infection.
Nephrectomy may be required if there is no function in the affected kidney. About 3–4 weeks of parenteral antibiotic therapy is usually required.

3-**Chronic Pyelonephritis**

Chronic pyelonephritis results from repeated renal infection, which leads to scarring, atrophy of the kidney, and subsequent renal insufficiency. The diagnosis is made by radiologic or pathologic examination rather than from clinical presentation.

A. Presentation and Findings

No symptoms, but they may have a history of frequent UTIs.
The diagnosis is made incidentally when radiologic investigation is initiated to evaluate for the complications associated with renal insufficiency, such as hypertension, visual impairments, headaches, fatigue, and polyuria.
In these patients, urinalysis may show leukocytes or proteinuria but is likely to be normal. Serum creatinine levels reflect the severity of the renal impairment. Urine cultures are only positive when there is an active infection.

B. Radiographic Imaging

Intravenous pyelogram or CT scan can readily demonstrate a small and atrophic kidney on the affected side.
Ultrasonography similarly can demonstrate these findings.
Dimercaptosuccinic acid (DMSA) is the best imaging modality to look for renal scarring.

C. Management

The management of chronic pyelonephritis is somewhat limited because renal damage incurred by chronic pyelonephritis is not reversible.
Eliminating recurrent UTIs
Identifying and correcting any underlying anatomic or functional urinary problems
Long-term use of continuous prophylactic antibiotic therapy may be required to limit UTIs and renal scarring.
Rarely, removal of the affected kidney may be necessary due to hypertension or having a large stone burden in a non-functioning kidney.

4-Renal Abscesses

Renal abscesses result from a severe infection that leads to liquefaction of renal tissue; this area is subsequently sequestered, forming an abscess. They can rupture out into the perinephric space, forming perinephric abscesses. When the abscesses extend beyond the Gerota's fascia, paranephric abscesses develop.
Abscesses that form in the renal cortex are likely to arise from hematogenous spread, whereas those in the corticomedullary junction are caused from gram-negative bacteria in conjunction with some other underlying urinary tract abnormalities, such as stones or obstruction.

A. Presentation and Findings

The most common presenting symptoms in patients with renal/perinephric abscesses include fever, flank or abdominal pain, chills, and dysuria.
A flank mass may be palpated in some patients.

B. Radiographic Imaging

Renal abscesses can be accurately detected using ultrasonography or CT scans.
CT scans can demonstrate an enlarged kidney with focal areas of hypoattenuation early on during the course of the infection.
Once the inflammatory wall forms around the fluid collection, the abscess appears as a mass with a rim of contrast enhancement, the "ring" sign.
Intravenous pyelogram and kidneys, ureter, and bladder tests are less sensitive tests in detecting renal/perinephric abscesses, with results being normal in about 20% of the cases.

C. Management

The appropriate management of renal abscess first must include
1-Appropriate antibiotic therapy. Empiric therapy with broad-spectrum antibiotics aminoglycoside or third-generation cephalosporin) is usually recommended.
2-If the patient does not respond within 48 hours of treatment, percutaneous drainage under CT scan or ultrasound guidance is indicated.

3-If the abscess still does not resolve, then open surgical drainage or nephrectomy may be necessary.

5-Xanthogranulomatous Pyelonephritis

Xanthogranulomatous pyelonephritis (XGP) is a form of chronic bacterial infection of the kidney. The affected kidney is almost always hydronephrotic and obstructed. In most cases, XGP occurs unilaterally. Severe inflammation and necrosis obliterate the kidney parenchyma. Characteristically, foamy lipid-laden histiocytes (xanthoma cells) are present and may be mistaken for renal clear cell carcinoma

A. Presentation and Findings

Patients with XGP commonly present with flank pain, fever, chills, and persistent bacteriuria. A history of urolithiasis is present in about 35% of the patients.
On physical examination, a flank mass can often be palpated. Urinalysis commonly demonstrates WBCs and protein.
E. coli or Proteus species are commonly cultured from the urine. However, one-third of patients with XGP have no growth in their urine

B. Radiographic Imaging

CT scan is the most reliable method in imaging patients suspected of having XGP. It usually demonstrates a large heterogeneous, reniform mass. The renal parenchyma is often marked with multiple water-density lesions, representing dilated calyces or abscesses.
On contrast enhanced images, these lesions will have a prominent blush peripherally, while the central areas, which are filled with pus and debris, do not enhance. An area of central calcification surrounded by a contracted pelvis may also be seen.
Ultrasonography does not provide comparable anatomic details with those obtained from CT scan.
It is not uncommon for XGP to be misdiagnosed as a renal tumor because of their similar appearances on radiologic imaging.

C. Management

The management of XGP is dependent on accurate diagnosis.
1- Nephrectomy is performed and a diagnosis is made pathologically.
2- In those in whom a diagnosis of XGP is suspected, kidney-sparing surgery such as a partial nephrectomy is indicated.
3- There are reported cases of treating XGP with antibiotic therapy alone or in combination with percutaneous drainage; however, these treatments are not likely to be curative in most patients and may lead to complications such as renal cutaneous fistula.

6-Pyonephrosis

Pyonephrosis refers to bacterial infection of a hydronephrotic, obstructed kidney, which leads to suppurative destruction of the renal parenchyma and potential loss of renal function. Because of the extent of the infection and the presence of urinary obstruction, sepsis may rapidly ensue, requiring rapid diagnosis and management.

A. Presentation and Findings

Patients with pyonephrosis are usually very ill, with high fever, chills, and flank pain. Lower tract symptoms are not usually present.
Bacteriuria and pyuria may not be present when there is complete obstruction of the affected kidney.

B. Radiographic Imaging

Imaging with renal ultrasonography can be performed to rapidly diagnose pyonephrosis. Ultrasonographic findings include persistent echoes in the inferior portion of the collecting system, fluid–debris level with dependent echoes that shift with positional changes.

C. Management

Management of pyonephrosis includes
1- Immediate institution of antibiotic therapy Broad-spectrum antimicrobials are indicated
2- Drainage of the infected collecting system, through the lower urinary tract (such as using a ureteral stent) should be reserved for patients who are not septic. Extensive manipulation may rapidly induce sepsis and toxemia.
In the ill patient, drainage of the collecting system with a percutaneous nephrostomy tube is preferable.
3- Once the infection is treated, additional imaging evaluation is required to identify and treat the cause of the urinary obstruction, such as urolithiasis or ureteropelvic junction obstruction.

- **BLADDER INFECTION**

Acute Cystitis:

Acute cystitis more **commonly affects women than men**.
The primary **mode of infection** is ascending from the periurethral/vaginal and fecal flora.
The diagnosis is made clinically.
In **children**, the distinction between upper and lower UTI is important.

A. Presentation and Findings

Patients with acute cystitis present with **irritative voiding symptoms** such as dysuria, frequency, and urgency. Low back and suprapubic pain, hematuria, and cloudy/foul-smelling urine are
Also common symptoms. Fever and systemic symptoms are rare.

Investigation

Urinalysis demonstrates WBCs in the urine, and hematuria may be present.
Urine culture is required to confirm the diagnosis and identify the causative organism.

Causative organism

E. coli causes most of the acute cystitis. Other gram-negative (Klebsiella and Proteus spp.) and gram-positive (S. saprophyticus and enterococci) bacteria are uncommon pathogens.

Risk factors

Diabetes and lifetime history of UTI are risk factors for acute cystitis. Of interest, the use of oral or vaginal estrogen was not protective in postmenopausal women with recurrent acute cystitis.

B. Radiographic Imaging

In uncomplicated infection of the bladder, radiologic evaluation is often not necessary.

C. Management

Management for acute cystitis consists of a short course of oral antibiotics. TMP–SMX, nitrofurantoin, and fluoroquinolones have excellent activity against most pathogens that cause cystitis.
Single-dose therapy for the treatment of recurrent cystitis/ UTI appears to be less effective; however, fluoroquinolones with long half-lives (fleroxacin, pefloxacin, and rufloxacin) may be suitable for single-dose therapy .Resistance to penicillins and aminopenicillins is high and thus they are not recommended for treatment.

Recurrent Cystitis/UTI:

A. Presentation and Findings

Recurrent cystitis/UTI is **caused either by**
Bacterial persistence or reinfection with another organism.
If bacterial persistence is the cause of recurrent UTI, the removal of the infected source is often curative, whereas preventative therapy is effective in treating reinfection.

B. Radiographic Imaging

When bacterial persistence is the suspected cause, radiologic imaging is indicated.
Ultrasonography, More detailed assessment with intravenous pyelogram, cystoscopy, and CT scans may occasionally be necessary.

When bacterial reinfection is the suspected cause of recurrent cystitis, the patient should be carefully evaluated for evidence of vesicovaginal or vesicoenteric fistula.

C. Management

Management of recurrent cystitis, again, depends on its cause.
Surgical removal of the infected source (such as urinary calculi) is needed to treat bacterial persistence. Similarly, fistulas need to be repaired surgically to prevent bacterial reinfection.
In most cases of bacterial reinfection, medical management with prophylactic antibiotics is indicated.
Low dose continuous prophylactic antibiotic has been shown to reduce the recurrences of UTI by 95%.
Alternatively, intermittent self-start antibiotic therapy can be used in treating recurrent cystitis in some women.

Malakoplakia:

Malakoplakia is an uncommon inflammatory disease of the bladder that can also affect other parts of the urinary tract, including the ureters and kidneys. In the bladder, it manifests as plaques or nodules made of large **histiocytes** (von Hansemann cells) with laminar inclusion bodies.

A. Presentation and Findings

Risk

Malakoplakia more commonly affects women than men and is associated with a history of UTI. Patients with malakoplakia often have chronic illness or are **immunosuppressed**.

Presentation

In patients with malakoplakia of the bladder, irritative voiding symptoms (urgency and frequency) and hematuria are common. When the disease affects the ureter or kidney, the Patient may present with fever, flank pain, or flank mass. When it affects both kidneys, signs and symptoms of azotemia or renal failure may be present.

B. Radiologic Imaging

Ultrasonography or CT may demonstrate a mass in the bladder and evidence of obstruction if the disease extends to the ureter.
It is often difficult to distinguish malakoplakia from malignancy (transitional cell or renal cell carcinoma) with radiologic imaging. The diagnosis is often established after biopsy.

C. Management

Management of malakoplakia primarily consists of antibiotic therapy, Consequently, TMP–SMX and fluoroquinolones are recommended in the treatment of malakoplakia.
Bethanecol and ascorbic acid, which enhance phagolysosomal activity, may have some benefits.

- **PROSTATE INFECTION**

Acute Bacterial Prostatitis:

Acute bacterial prostatitis refers to inflammation of the prostate associated with a UTI.
It is thought that infection results from ascending urethral infection or reflux of infected urine From the bladder into the prostatic ducts.

A. Presentation and Findings

Acute bacterial prostatitis is uncommon in prepubertal boys but frequently affects adult men. It is the most **common urologic diagnosis in men younger than 50 years**.

Presentation

Patients with acute bacterial prostatitis usually present with an abrupt onset of constitutional (fever, chills, malaise, arthralgia, myalgia, lower back/rectal/perineal pain) and urinary symptoms (frequency, urgency, and dysuria). They may also present with urinary retention due to swelling of the prostate. Digital rectal examination reveals tender, enlarged glands that are irregular and warm.

Investigations

Urinalysis usually demonstrates WBCs and occasionally hematuria. Serum blood analysis typically demonstrates leukocytosis. Prostate-specific antigen levels are often elevated. The diagnosis of prostatitis is made with microscopic examination and culture of the prostatic expressate and culture of urine obtained before and after prostate massage. In patients with acute prostatitis, fluid from the prostate massage often contains leukocytes with fatladen macrophages.
Urethral catheterization should be avoided. Culture of urine and prostate expressate usually identifies a single organism, but occasionally, polymicrobial infection may occur.

Causative organism

E. coli is the most common causative organism in patients with acute prostatitis. Other gram-negative bacteria (Proteus, Klebsiella, Enterobacter, Pseudomonas, and Serratiaspp.) and enterococci are less frequent pathogens. Anaerobic and other gram-positive bacteria are rarely a cause of acute prostatitis.

B. Radiologic Imaging

Radiologic imaging is rarely indicated in patients with acute prostatitis. Bladder ultrasonography may be useful in determining the amount of residual urine.
Transrectal ultrasonography is indicated only in patients who do not respond to conventional therapy.

C. Management

Empiric therapy directed against gram negative bacteria and enterococci should be instituted immediately while awaiting the culture results. Trimethoprim and fluoroquinolones have high drug penetration into prostatic tissue and are recommended **for 4–6 weeks**.

Patients who have sepsis, are immunocompromised or in acute urinary retention, or have significant medical comorbidities would benefit from hospitalization and treatment with parenteral antibiotics.
Patients with urinary retention secondary to acute prostatitis should be managed with a suprapubic catheter because transurethral catheterization or instrumentation is contraindicated.

Chronic Bacterial Prostatitis:

In contrast to the acute form, chronic bacterial prostatitis has a more insidious onset, characterized by relapsing, recurrent UTI caused by the persistence of pathogen in the prostatic fluid despite antibiotic therapy.

A. **Presentation and Findings**

Most patients with chronic bacterial prostatitis typically present with dysuria, urgency, frequency, nocturia, and low back/perineal pain.
These patients usually are afebrile and not uncommonly have a history of recurrent or relapsing UTI, urethritis, or epididymitis caused by the same organism.
Others are asymptomatic,
Investigation for bacteriuria,Urinalysis demonstrates a variable degree of WBCs and bacteria in the urine, depending on the extent of the disease. Serum blood analysis normally does not show any evidence of leukocytosis. Prostate-specific antigen levels may be elevated.

Table 14–8. Technique of localization cultures (four-cup test) for the diagnosis of prostatitis.

1. Preparation:
   - Require that the patient has a full bladder
   - Retract foreskin of uncircumcised men
   - Clean glans with soap/water or povidone-iodine
2. Collection:
   - Collect first 10 mL of voided urine (VB1)
   - Discard the next 100 mL
   - Collect the next 10 mL of voided urine (VB2)
   - Massage prostate and collect prostate expressate (EPS)
   - Collect first 10 mL of voided urine after massage (VB3)
   - Immediate culture and microscopic examination of all specimen
3. Interpretation:
   - All specimens <103 CFU/ mL → not bacterial prostatitis
   - VB3 or EPS >10× CFU of VB1 → chronic bacterial prostatitis
   - VB1 > other specimens → urethritis or specimen contamination
   - All specimens $>10^3$ CFU/mL → treat for UTI and repeat test
4. Caution:
   - Sensitivity of the test may not be high (Lipsky, 1999)
   - Time consuming and expensive
5. Alternative:
   - Voided specimen before and after prostate massage (Nickel, 1997)

CFU, colony-forming unit; UTI, urinary tract infection.

B. **Radiologic Imaging**

Radiologic imaging is rarely indicated in patients with chronic prostatitis. Transrectal ultrasonography is only indicated if a prostatic abscess is suspected.

C. **Management**

Some patients respond to antibiotic therapy, corticosteroids, and temporary bladder drainage. **Transurethral resection of the prostate** may be required in patients who do not respond to treatment and have significant outlet obstruction.

Prostate Abscess:

Most cases of prostatic abscess result from complications of acute bacterial prostatitis that were inadequately or inappropriately treated. Prostatic abscesses are often seen in patients With diabetes; those receiving chronic dialysis; or patients who are immunocompromised, undergoing urethral instrumentation, or who have chronic indwelling catheters.

A. Presentation and Findings

Similar symptoms to those with acute bacterial prostatitis.
Typically, these patients were treated for acute bacterial prostatitis previously
On digital rectal examination, the prostate is usually tender and swollen. Fluctuance is only seen in 16% of patients with prostatic abscess.

B. Radiologic Imaging

Imaging with transrectal ultrasonography or pelvic CT scan is crucial for diagnosis and treatment.

C. Management

Antibiotic therapy in conjunction with drainage of the abscess is required.
Trans-rectal ultrasonography or CT scan can be used to direct transrectal drainage of the abscess.
Transurethral resection and drainage may be required if transrectal drainage is inadequate.

- **URETHRITIS**

Types of Urethritis:

Infection/inflammation of the urethra can be categorized into those types caused by Neisseria gonorrhoeae and by other organisms (Chlamydia trachomatis, Ureaplasma urealyticum, Trichomonas vaginalis, and herpes simplex virus).
Most cases are acquired during **sexual intercourse.**

A. **Presentation and Findings**

Patients with urethritis may present with urethral discharge and dysuria.
Obstructive voiding symptoms are primarily present in patients with recurrent infection, in whom urethral strictures subsequently develop.
With gonococcal urethritis are asymptomatic.
The diagnosis is made from examination and culture of the urethra. It is important to obtain the specimen from within the urethra, rather than from just the discharge.

B. **Radiologic Imaging**

Retrograde urethrogram is indicated only in patients with recurrent infection and obstructive voiding symptoms. Most patients with uncomplicated urethritis do not require any radiologic imaging.

C. **Management**

Gonococcal urethritis, ceftriaxone (250 mg intramuscularly) or fluoroquinolones (ciprofloxacin 250 mg) or norfloxacin (800 mg) may be used.
Nongonococcal urethritis, treatment is with tetracycline or erythromycin (500 mg four times daily) or doxycycline (100 mg twice daily) for 7–14 days.
Sexual partners of the affected patients should be treated, and protective sexual practices (such as using condoms) are recommended.

- **EPIDIDYMITIS/ORCHITIS**

Causes of Epididymitis/Orchitis:

Infection and inflammation of the epididymis most often result from an ascending infection from the lower urinary tract.
In men younger than 35 years are due to sexually transmitted organisms (N. gonorrhoeae and C. trachomatis);
Those in children and older men are due to urinary pathogens such as E. coli. Other causes of epididymitis/orchitis in young children are due to a postinfectious inflammatory reaction to pathogens such as mycoplasma pneumoniae, enteroviruses, and adenoviruses, which often

follows a more benign course. In homosexual men who practice anal intercourse, E. coli and other coliform bacteria are common causative organisms.

A. Presentation and Findings

Patients with epididymitis/orchitis present with severe scrotal pain that may radiate to the groin or flank. Scrotal enlargement due to the inflammation of the epididymis/testis or a Reactive hydrocele may develop rapidly.
Other symptoms of urethritis, cystitis, or prostatitis may be present before or concurrent with the onset of scrotal pain.

On physical examination, an enlarged and red scrotum is present, and it is often difficult to distinguish the epididymis from the testis during the acute infection. A thickened spermatic cord can occasionally be palpated.

Urinalysis typically demonstrates WBCs and bacteria in the urine or urethral discharge.
Serum blood analysis often reveals leukocytosis.

B. Radiologic Imaging

Scrotal Doppler ultrasonography or radionuclide scanning can be used to confirm the diagnosis. **Scanning rules out torsion**.

C. Management

Oral antibiotic,bed rest, scrotal elevation, and the use of nonsteroidal anti-inflammatory agents are helpful in reducing the duration of the symptoms
For patients with sepsis or severe infection, hospitalization and parenteral antibiotic therapy may be needed.
Open drainage is indicated in cases in which an abscess develops.

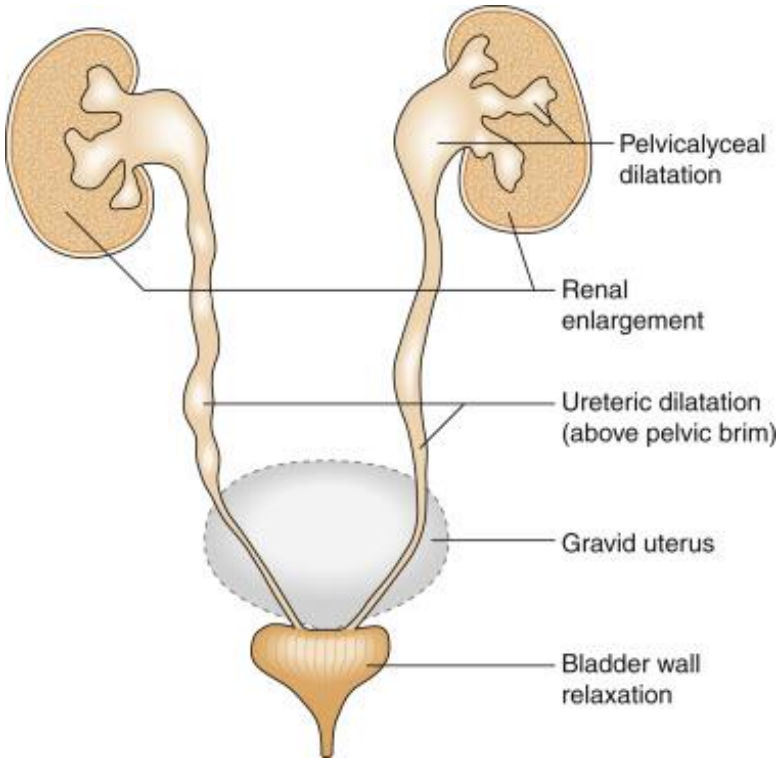
SPECIAL CIRCUMSTANCES

UTI Related to Pregnancy

Anatomical physiological changes

With pregnancy, there are anatomic and physiologic changes to the urinary tract due to compression by the gravid uterus and alterations in the hormonal milieu.

- Renal length increases approximately by 1 cm during normal pregnancy as a result of increased vascular and interstitial volume. The glomerular filtration rate increases by 30–50%, most likely secondary to the increase in cardiac output.
- Typically, there is significant ureteral dilation with resultant urinary stasis during the second and third trimesters of gestation. This hydroureter is attributed to the smooth muscle–relaxing effects of progesterone and the Mechanical compression of the ureters by the uterus at the level of the pelvic brim.
- The bladder is also affected, both physically and physiologically. The enlarged uterus displaces the bladder superiorly and anteriorly. The bladder becomes hyperemic, and its capacity is increased, most likely due to the effects of progesterone.



Incidence

bacteriuria is a clinically relevant finding in pregnant women. It is estimated that the prevalence of bacteriuria is 4–6%, which is not significantly different from that in nonpregnant women of comparable age.
Overall, the incidence of acute bacterial pyelonephritis is 1–4% in pregnant women.

About 60–70% of the episodes of pyelonephritis occur during the second and third trimesters of pregnancy,
For asymptomatic individuals, **significant bacteriuria** is defined as two voided urine cultures with $>10^5$ CFU/mL of a single organism. For symptomatic pregnant women, $>10^3$ CFU/mL is considered to be significant.

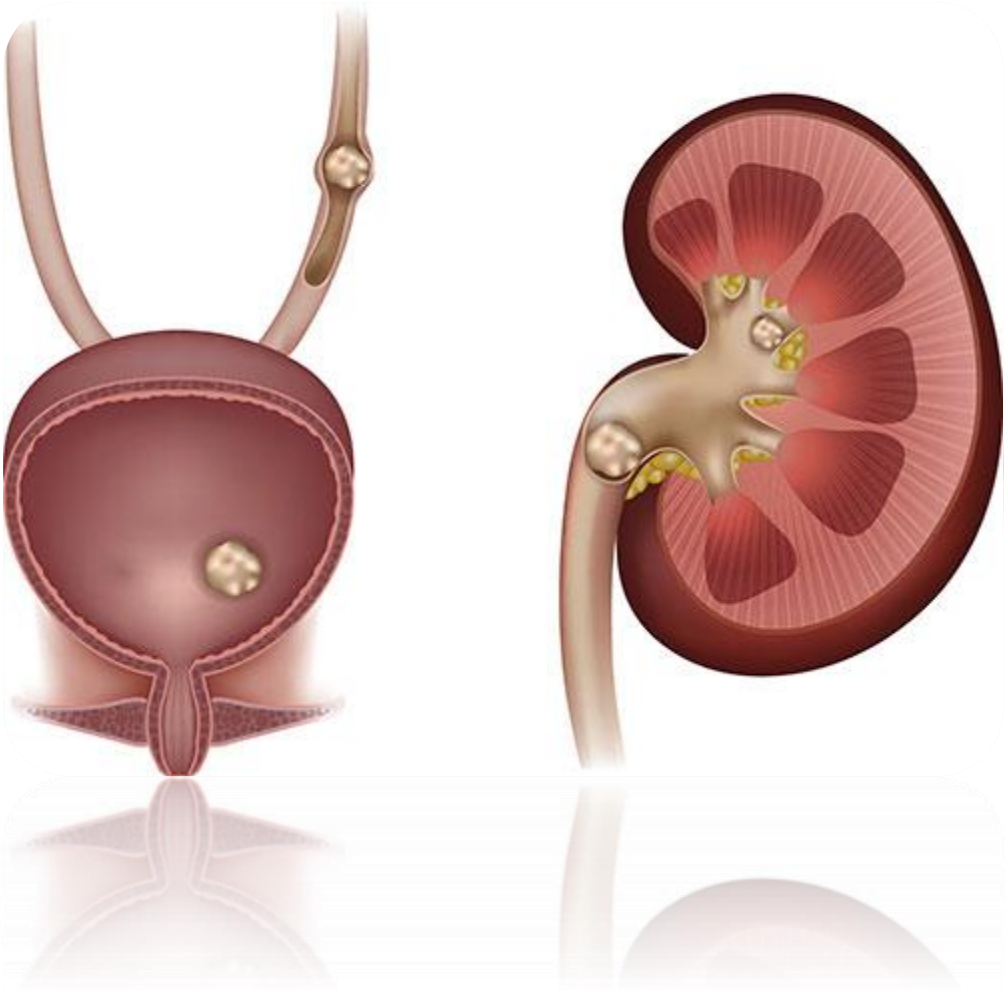
Treatment

Pregnant women who are found to have bacteriuria should be treated with penicillins, oral cephalosporins, or Fosfomycin trometamol. However, amoxicillin is not recommended because of the rate of bacterial resistance.

A 3-day course is suggested, although single-dose therapy may be effective in some patients.

Repeat urine culture to document eradication of bacteriuria is necessary in all patients.
Patients with acute bacterial pyelonephritis should be treated with parenteral cephalosporins, penicillins with beta-lactamase inhibitors, or monobactams.

URINARY STONES



Urinary stones

Incidence:

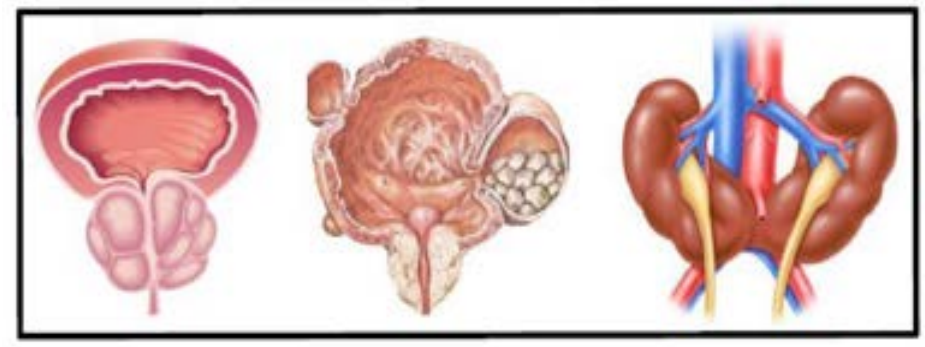
• Peak incidence is between 20 - 40 years old (No age is immune).

• Males > Females.

• It's common in Egypt due to hot dry climate.

• It presents in 10 - 20 % of population.

• ↑risk with metabolic syndrome, DM & CVD.



Etiology:

A. Urine stasis:

As in BPH, bladder diverticulum, neurogenic bladder& strictures (lower end of ureter & urethra) → allow stones to form.

B. Excess normal constitutes of urine:

i) Low urine volume →↑its concentration.

ii) Excess urinary excretion of calcium:

- Idiopathic (inherited autosomal dominant trait). - Hyperparathyroidism.
- Prolonged immobilization.
- ↑intake of milk.

iii) Excess urinary excretion of **uric acid** (end product of purine):

- Gout.
- Chemotherapy.
- Purine rich food e.g. meat, liver & kidney.

iv) Excess urinary excretion of oxalates:

- - Idiopathic (type I & II: autosomal recessive).
- - ↑intake of strawberries, green leafy vegetables & boiled tea. - Loss of terminal ileum e.g. resection in Crohn's disease.

C. Presence of abnormal constituents in urine:

i) Infection:

- - Produces epithelial desquamation upon which calculi deposit.
- - Infection with urea splitting organisms:

  - **Proteus mirabilis** (COMMONEST)

  - Ureaplasma.
  - Urealyticum.
  - Pseudomonas.
  - Klebsiela.
  - Some staph. aureus.
  - Some strains of E-coli (13 %).

- - Splitting of urea into ammonium & carbon dioxide.
- - Causing urine alkalinization → favors formation of phosphate (struvite) stones.

ii) Foreign bodies:
- Act as a nidus for stone formation as non-absorbable sutures, ureteric stents or catheters.

iii) Vitamin A deficiency :(rare)

- Causes hyperkeratosis of urothelium → epithelial debris acts as a nidus for stone formation.

iv) Cystinuria

(Autosomal recessive disease on chromosome 2) → Cystine stone formation.

D. Genetic factors:

Distalortype1RTA, Marfan's syndrome &Wilson's disease. Pathophysiology:

Saturation Super-saturation Nucleation
Crystal growth & aggregation Crystal retention STONE FORMATION

• Normal substances that inhibit stone formation (Nucleation):

- Tamm Horsfol protein. - Nephrocalcin.
- Uropontin.

Types of stones:

1. Calcium oxalate stones: COMMONEST (up to 70 %)


Calcium oxalate stone

- - Monohydrate or Dihydrate.
- - Monohydrate stones are very hard stones (ESWL resistant).
- - Develop in acidic urine (pH < 6).
- - Surface: **Spiky**, Consistency: **Hard**, Radiological: **Densely opaque**,

  Color: **Dark brown or Black**. **N.B:**

  - The activity of stone disease correlates with urinary oxalate excretion rather than the calcium.

  - Oxalate is the strongest chemical promoter of stone production.

2. Calcium phosphate stones: (6 - 20 %)


Calcium phosphate stones

- - Develop in alkaline urine (pH > 7.5).
- - Rule out infection, RTA & hyperparathyroidism.
- - Surface: **Smooth**, Consistency: **Hard**, Radiological: **Opaque**,

  Color: **Yellowish white.**

- - They can attain a large size e.g. **staghorn** calculus → fills renal pelvis and calyces.

N.B: specific causes of calcium stones:
• Hypercalcuria (Absorptive, Renal & Resorptive). • Hyperuricosuria.
• Hyperoxaluria.
• Hypocitraturia:

Citrate forms a soluble salt with calcium → - - Ca stone formation. - Normal citrate level in urine: 300 - 900 mg/24 hours.

3. **Mixed oxalate & phosphate stones: (6 - 20 %)**
4. **Mg ammonium phosphate stones (infection=struvite): (6 - 20 %)**

Triphosphate stones

- Triphosphate stones.
- Surface: Smooth, Consistency: friable, Radiological: Faint opaque,

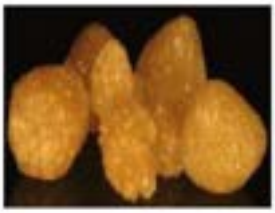
Color: Yellowish white.
- Most common cause of staghorn stone.

5. **Urate stones: (6 - 17 %)**
   - - Surface: Smooth, Consistency: Hard, Radiological: the only lucent type, Color: Golden yellow.
   - - Associated with hyperuricemia (+/- gout).
   - - It favors calcium oxalate & calcium phosphate stone formation.
   - - May form staghorn.
   - - Conditions in which urate stones can be formed:
     - Low PH (4.5-5).
     - Low urine volume.
     - Hyperuricosuria (acid urine with low ammonium excretion, autosomal dominant disorder).


Urate stone

6. **Cystine stones: 1-2%**

- Occurs in cystinuria (recurrent, ESWL resistant).
- Surface: Smooth, Consistency: Hard, Radiological: Faint opaque.


Cystine stones

7. **Miscellaneous, Xanthine, silicate, drug metabolites (1 - 4 %).**

N.B:
- Radiolucent stones don't appear in X-ray but appear in IVP (filling defect), U/S & spiral CT without contrast.
- Cystine & Calcium oxalate monohydrate are resistant to ESWL.


Staghorn stone

Clinical picture:
Symptoms:

A. Renal & ureteric stones:

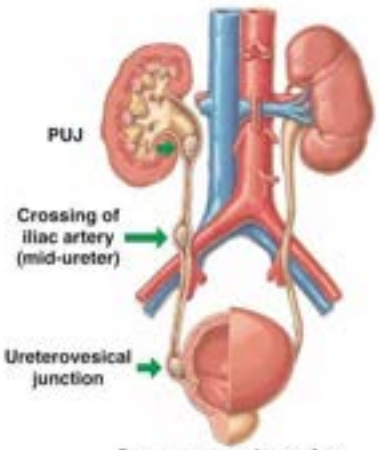
- - Asymptomatic (accidentally discovered) in staghorn stone or tiny stone in a calyx.
- - Symptoms when a stone gets impacted → obstruction.
- - Common sites for stone impaction are:
    1. i) Pelvi-ureteric junction.
    2. ii) Pelvic brim as ureter crosses external iliac artery.

  iii) Uretero-vesical junction.

- - Pain (**the main symptom**)
    1. a) Renal pelvic stone

       • Character: fixed dull aching.
       • Site: renal (costovertebral) angle.
       • Radiation: to the back or hypochondrium anteriorly. • ↑↑by movement.

    2. b) Ureteric stone
       - Character: colicky pain due to:
         1. i) Stretch of the capsule of the kidney caused by: hydronephrosis.
         2. ii) Spasm of ureteric smooth muscle.
       - In upper 1/3 → colicky pain in the loin radiating to groin or testis in males or labia majora in females.
       - In middle 1/3 → iliac pain simulating appendicitis.
       - In lower 1/3 → pain at end of micturition referred to the tip of penis Associated with frequency.



Common sites for stone impaction

- - Nausea &/or vomiting: during pain d.t irritation of coeliac ganglion.
- - Abdominal distention.
- - Hematuria: gross or microscopic d.t mucosal injury during stone migration.

- - Ureteric stones are formed either due to migration of renal stones or stricture of ureter.
- - Ureteric stones can also cause a ureteric stricture.
- - A moving stone is often more painful than a static stone.

B. Urinary bladder stones:

- From mild discomfort to suprapubic pain which is:

- Referred to the tip of penis / labia majora or to the perineum.
- More prominent at day time (during micturition).
- ↓during sleep or in recumbent position as stone moves away from the trigone.

- Frequency of micturition:
• Caused by irritation of bladder mucosa & trigone.
• Timing: at first → only diurnal, but if cystitis develops → diurnal & nocturnal. - Difficult or burning micturition & interrupted urine stream during micturition.

- Acute urine retention D.T bladder neck obstruction by a stone. - Terminal hematuria (most common).

N.B: classification of bladder stones:

Primary (migratory)	Secondary
- Occurs in sterile urine. - Usually oxalate stone. - Can irritate bladder mucosa causing hematuria.	- Occurs in presence of infection. - Usually phosphate stones. - Occurs in bladder only. - **Commonest** bladder stone.

C. Urethral stones:

- Sudden arrest of urine followed by retention.
- Severe urethral pain, difficulty & interruption of micturition stream.

D. Manifestations of infection (pyelonephritis):

- Fever, rigors & loin or flank pain.

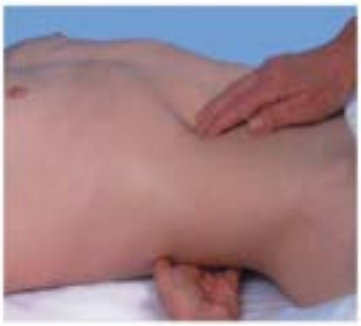
Signs

A. General

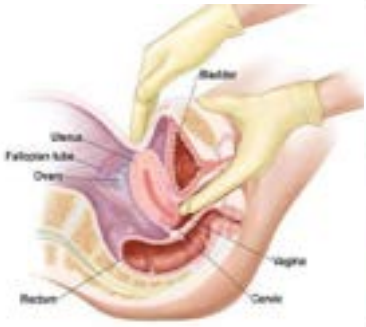
- Features of uremia (renal failure) which is preceded by calcular anuria.

B. Local

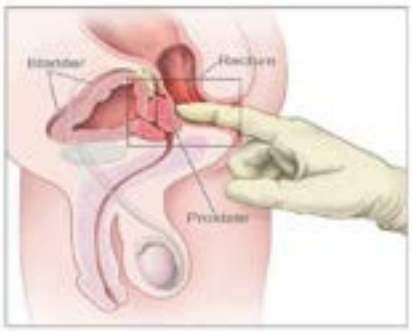
- Tender renal swelling.
- Palpable kidney → hydronephrosis.
- Tenderness in iliac fossa in ureteric stones.
- Suprapubic tenderness in bladder stones.
- Infra-umbilical dullness in retention or fully felt bladder by palpation.
- Stone in the penile (anterior) urethra may be palpated.



Kidney palpation



PV examination



PR examination

Complications:

1. Obstruction → stasis, back pressure & proximal dilatation.

a) Partial or intermittent:

i) Ureteric stones → hydroureter & hydronephrosis.

ii) Pelvi-ureteric junction stone → hydronephrosis.

iii) At neck of calyx → calycial dilatation (hydrocalyx).

b) Complete:

i) Calcular anuria

ii) Acute retention d.t obstruction in the urinary bladder or urethera.

2. Infection as stasis invites infection e.g. pyelonephritis, pyonephrosis, cystitis &

Urethritis followed by ascending infection to the other side.

3. Hematuria d.t repeated ulceration of the mucosa.

4. Deterioration of renal function d.t obstruction or infection RF & uremia.

5. Squamous metaplasia of urinary transitional epithelium sq. cell carcinoma

Investigations:

A. Laboratory:

a) urine analysis:

1. Microscopic hematuria (at least 90%) or
2. Crystals of same type that are forming the stone.
3. In struvite stones:

   i) PH is from 8 to 9.

ii) Pyuria & bacteriuria.
iii) Culture: urea producing organism.

b) Stone analysis to detect the type of stone.

c) Serum PTH, Ca, P & uric acid to detect the cause.

d) Metabolic workup (24hoursurinecollection) for total volume, pH,Ca, citrate, Mg, oxalate, P, Na, uric acid & cystine & creatinine.

- Indicated in stone former, bil. Staghorn stones & in children.

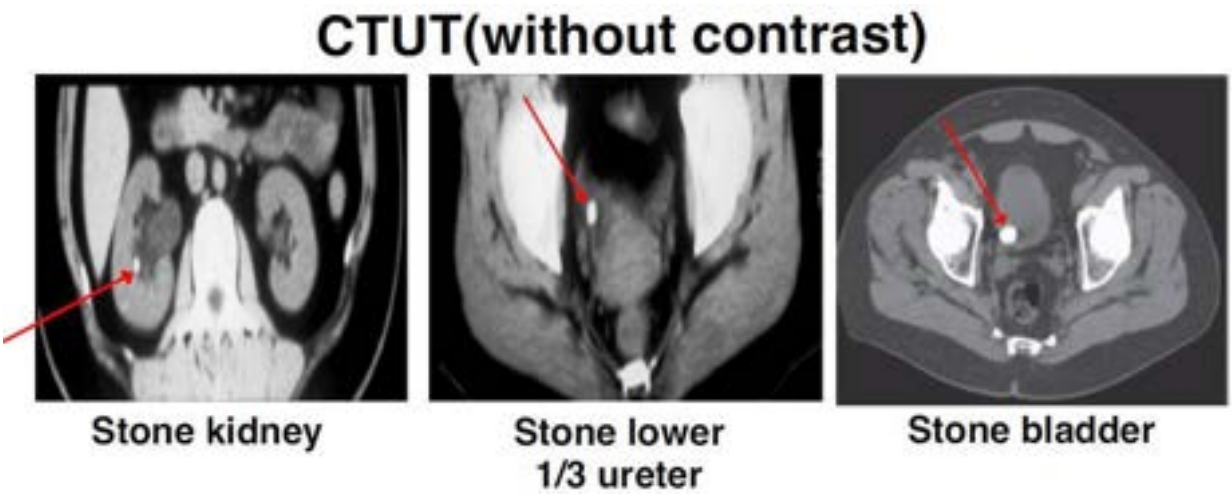
e) KFTs for renal impairment.

B. Radiological:

1. Computed tomography (CT) (the first-choice) GOLD STANDARD

- CT UT without contrast (spiral CT) , visualizes the smallest stones.
- Images can be reconstructed with computer software to produce high quality images of the whole urinary tract.
- It can detect the stone density (Hounsfield unit).

- Visualizes both radio-opaque and radiolucent stones.
- Without contrast safe in renal impairment.
- Contraindicated in pregnancy.
- Contrast is added to exclude other causes for hematuria e.g. bladder cancer.

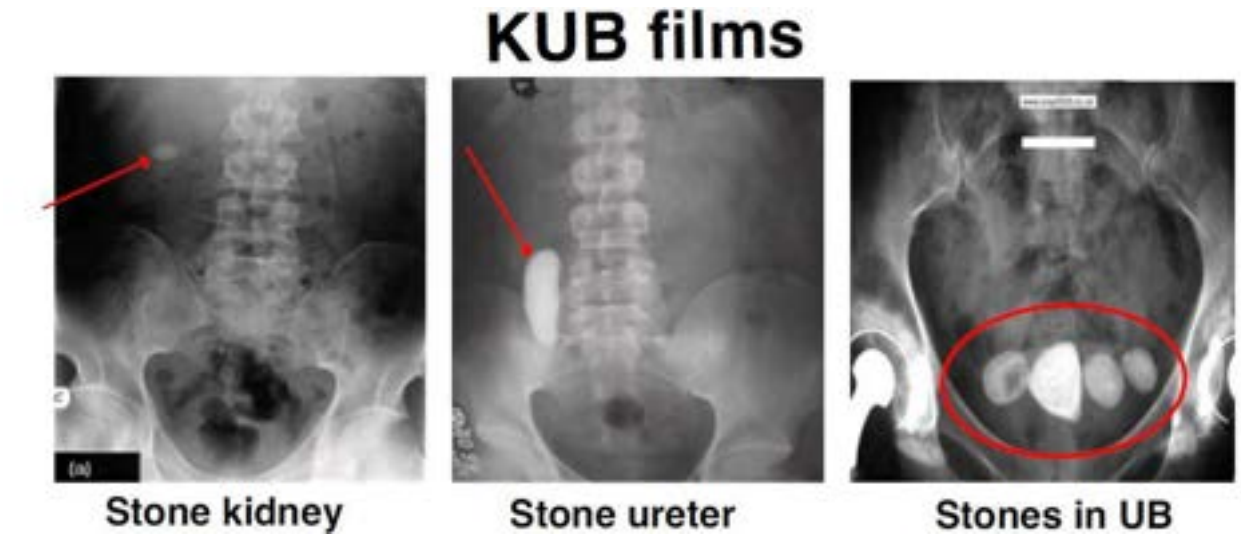


2. Plain UT (KUB) (stones are radio-opaque in 90%)

- In descending order of density calcium oxalate is the densest, followed by Calcium phosphate, struvite & cystine.

- Uric acid stones are radiolucent.

- A doubtful shadow can be diagnosed by Lateral view , renal stone lies on the vertebral bodies while GB stones lie anterior to the vertebral bodies.



3. Ultrasonography

Advantages:

i) Simple

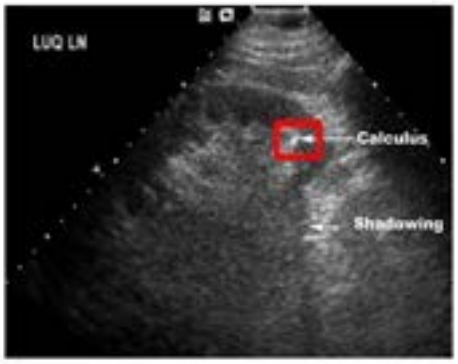
ii) Visualize the kidney & the degree of back pressure changes.

iii) Diagnose bladder stones & radiolucent calculi.

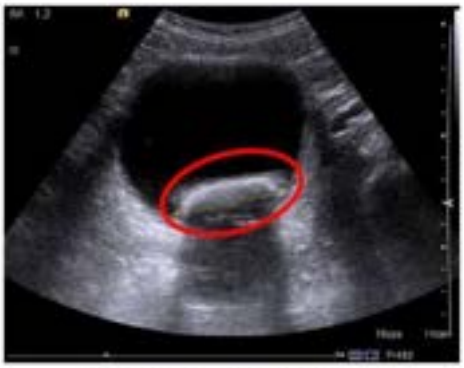
iv) SAFE in pregnancy.

Disadvantage: not reliable to visualize ureteric stones.

Ultrasound



Stone kidney



Stone bladder

4. Intravenous urography (IVU / IVP):

- RARELY used.

- Radiolucent stones appear as a filling defect.

- Contraindicated in renal impairment.

- Appearance of renal calyces at various grades of hydronephrosis:

• Normally >> cupped.

• 1st grade >> clubbed.

• 2nd grade >> ballooned.

• 3rd grade >> crescents (sac of urine).

5. MRI:

Used in case of:

i) Pregnancy.

ii) Dye sensitivity of IVU.

iii) Renal impairment.

Factors affecting management of stones:

1) Stones related factors	2) Anatomical factors	3) Patient factors
- Site. - Size. - Number. - Composition.	- Obstruction. - Hydronephrosis. - Horse-shoe kidney. - PUJ obstruction. - Ectopic pelvic kidney. - Bladder stone d.t BPH.	- Obesity. - Infection. - Coagulopathy. - Pregnancy. - Single kidney. - HTN. - Elderly or Pediatrics

4) Facilities availability.

Prevention:

- - High water intake esp. in hot weather.
- - Avoid excess intake of salt.
- - Eat mixed balanced diet.
- - Do NOT reduce the intake of calcium.
- - Avoid excess intake of animal protein.
- - Avoid nuts, spinach, grapefruit, almonds, black tea & dark chocolate as snack (rich in oxalate).
- - Many small meals are better than few & large ones.

Treatment:

A. Treatment of renal colic:

- - Most cases are treated as outpatients.
- - Hospitalization for patients with severe colic with persistent vomiting

(It represents 1% of all emergency room visits) →
1. Parenteral analgesics:

i) NSAID as indomethacin or diclofenac.
ii) Narcotic analgesics as opiates (in resistant cases).
2. Anti-emetics, e.g. metoclopramide.

3. IV fluids are given (according to normal patient's requirements) if vomiting is persistent.
- Forcing excess fluids during attack → flush out the stone, but if not → ↑ pressure in an obstructed system → ↑ pain. 4. Antibiotics (if UTI is suspected).

B. Medical treatment for both renal & ureteric stones:

- - Purposes:
  • Treatment of acute episode by pain relief & stone expulsion.

  • Prevention of stone recurrence or new stone formation.

- - Pharmacological agents:

1) NSAID

2) Thiazides	3) Orthophosphates	4) Citrates
- Act by ++ Ca resorption in DCT & ++ Na excretion →↓urinary Ca by 150 mg/ day in normo-calcuric pts. & 400 mg/day in hypercalcuric pts. - **DOC** for renal hypercalcuria. - Long term therapy → • volume depletion. • ↓ECV. • proximal tubular Na & Ca resorption. - Dose: 50 mg twice daily.	• - Act by binding dietary Ca in intestine → ↓Ca absorption →↓urinary Ca by 50 % in pts. with absorptive hypercalcuria & 25 % in pts. without this disorder. • - Disadvantages: • intolerable • ↑inhibitor activity of urine &↑renal excretion of phosphate & citrate.	• - New promising method for prevention of recurrent Ca oxalate stones. • - Agents: • sodium potassium citrate. • potassium citrate. • - Dose: 30-60 mEq/ day in 3 divided doses or single evening dose.

- **N:B:** in urate stones → chemo dissolution using potassium citrate effervescent + high fluid intake + low purine diet in certain occasions i.e. residual stone after PCNL or lower pole non obstructing renal stone.

5) α blockers	6) Magnesium	7) Allopurinol
- Act as **medical expulsive** therapy via relaxation of smooth muscle while maintaining tonic propulsive contractions.	• - Act by ↑urinary Mg → produce Mg: CA in urine → protection against stone formation. • - Mg ↓renal tubular citrate resorption by citrate chelation → ↑citrate excretion. • - S/E: GIT intolerance.	• - Act by inhibition of xanthine oxidase & ↓urate production. • - S/E: drug reactions e.g. severe hypersensitivity with thiazides. • - Dose: 100 mg 3 times a day.

C. Surgical treatment of renal stones:

- Lower pole:

1. a) ≤ 0.4 cm & asymptomatic → conservative (90 % can pass spontaneously).
2. b) 0.5-2 cm → ESWL vs. RIRS vs. mini PCNL.
3. c) >2cm→PCNL.

- Non lower pole :(in renal pelvis)

1. a) 1-2 cm → ESWL vs. RIRS vs. mini PCNL.
2. b) > 2 cm → PCNL or pyelolithotomy (open/laparoscopic/robot assisted)
3. c) Huge (Staghorn stones):

• Staged PCNL.
• PCNL + ESWL + PCNL (sandwich technique). • PCNL + RIRS (new technique).
• pyelonephrolithotomy

D. Surgical treatment of ureteric stones:

- Upper:

1. a) <1cm→ESWL
2. b) > 1 cm → RIRS by flexible ureteroscopy & LASER disintegration of the stone → JJ stent insertion → ESWL (< 2 cm) or PCNL (> 2 cm).
3. c) Huge/impacted stones→ uretrolithotomy (open/laparoscopic/robot assisted).

- Mid or lower:

1. a) < 5 mm + **no infection, no hydronephrosis, normal other kidney** → analgesics, α blocker (e.g. tamsulosin) & CCB.
2. b) > 5 mm → semi rigid uretroscopy & retrieval of stone.
3. c) HUGE stones → uretrolithotomy.

- N.B:

- RIRS: retrograde intrarenal surgery.
- LASER disintegration is the only modality in RIRS.
- Ureteric stone passage rate:
    1. i) Upper 1/3: 49 %
    2. ii) Middle 1/3: 58 %

    iii) Lower1/3:68%

- 75 % of ureteric stones (< 5 mm) pass spontaneously irrespective of location.
- Up to 95 % of ureteric stones (≤ 4mm) pass within 40 days.
- If ureteric stone can't be removed as one part → lithotripsy is done be Laser, pneumatic (ballistic) or US techniques.

E. Bladder stones:

- - **1-2 cm** → transurethral cystolithotripsy, if failed → percutaneous cystolithotripsy.
- - **> 2 cm** → Suprapubic cystolithotomy.
- - **In pediatric age group** → percutaneous cystolitholapaxy.
- - 1ry cause should be treated in 2ry type e.g. TURP + cystolithotomy.

F. Urethral stones:

- Urethral indwelling Foley's catheter to push the stone into the bladder, if failed → suprapubic catheter insertion.

- Then deal with the stone as if it's a bladder stone.

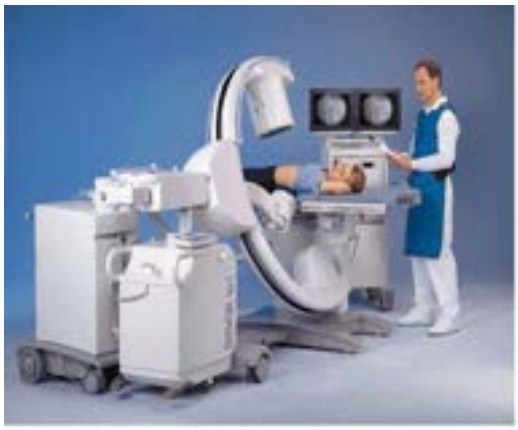
Prognosis:

- Recurrence rate after initial stone: 25-75 % in 10-20 years of follow up.

Various modalities for stone disintegration & removal

1. Extracorporeal shock wave lithotripsy (ESWL):

- Most common way to treat stones.



ESWL

Principle (done under IV sedation)

• High-energy shock waves that are focused from outside the body on the stone (after its visualization using x-ray or ultrasonic imaging)

• The stone is fragmented → pass out through the normal pathway.

• High fluid intake is maintained to facilitate passage of gravels. −

Advantages: least invasive & suitable for risky patients.

Disadvantages:

1. **i)** 20 % of stones won't be broken into small pieces.
2. **ii)** 20 % of stones will fail to pass out of the patient after the ttt.

iii) Painful passage of stone pieces.

Contraindications:

a) Urological:

i) Presence of distal obstruction (fragments will not pass).

ii) Stone (>2cm) except after debulking of stone by PCNL.

iii) Solitary kidney except after stenting of ureter to avoid acute ureteric obstruction & anuria.

iv) Renal insufficiency (kidney has no power to push fragments).

v) Acute episode of renal infection.

b) Non-urological:

i) Pregnancy.

ii) Arterial aneurysm near stone (renal or AAA).

iii) Uncorrected coagulopathy or bleeding diathesis

Complications:

a) Related to stone passage:

i) Transient attacks of hematuria.

ii) Renal colic.

iii) Steinstrasse.

b) Related to shock wave:

i) Skin bruising.

ii) Sub-capsular hematoma.

iii) Pancreatitis.

iv) Urosepsis.

v) Hearing loss for the physician

Efficacy of ESWL:

1. **a) Stone kidney:**<2cm→upto90%
2. **b)** Upper ureteric stones:
    1. **i)** Non impacted → up to 90 %
    2. **ii)** Impacted → up to 60 %
3. **c)** Mid ureteric stones: up to 80 %

2. Percutaneous nephrolithotomy (PCNL):

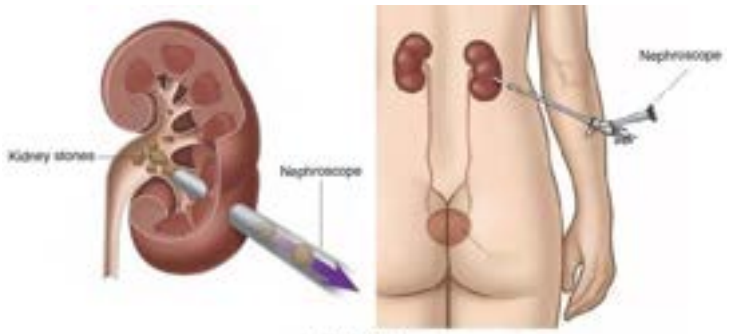
- Indications:

i) Large stone burden (> 2 cm).

ii) Large lower pole lower stone.

iii) Ca. oxalate monohydrate stone or Cystine stone.

iv) Distal obstruction.



PCNL

v) Abnormalities of renal & upper tract anatomy.

vi) Abnormalities of patient's anatomy e.g obesity & deformity.

vii) ESWL failure.

- Contraindications:

i) Pregnancy (X-ray exposure).

ii) Bleeding diathesis.

iii) Renal anomalies, but in horseshoe kidney is done through upper calyx.

- Technique:

1. Contrast material is injected up a ureteric catheter to visualize the pelvicalyceal system under X-ray or US.

2. A needle is passed to the pelvis through which a guide-wire is inserted.

3. The tract is dilated over the guide wire to a size sufficient to accommodate the nephroscope.

4. Stone manipulation:

- Small stones < 1cm □ directly extracted through the sheath.

- Large stones are fragmented first through nephroscope by either ultrasonic, pneumatic or laser lithotripsy.

5. Removal of residual fragments by using irrigating fluid.

6. Finally, a nephrostomy tube is left in situ for 48 hours to allow drainage of residual tiny fragments, urine & blood.

Advantages:

i) Very small endoscopic incision (1 cm).

ii) Minimal operative & post-operative side effects.

iii) Short hospital stays (about 3 days).

- Complications:

i) Kidney bleeding.

ii) Extravasation of irrigating fluid into peritoneum or retroperitoneum.

iii) Perforation.

iv) Injury to adjacent organs e.g. pleura & colon.

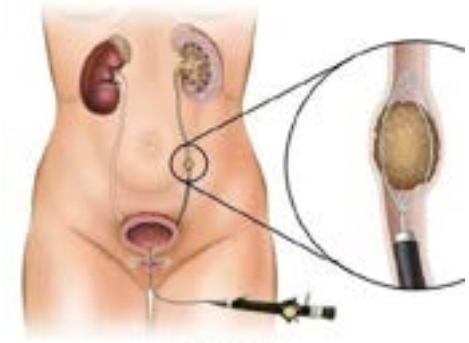
v) Infection.

3. Uretroscopy:

- - Is the **gold standard** for treatment of middle and lower ureteric stones.
- - Flexible ureteroscopes are available for diagnostic & therapeutic uses because of the deflection capabilities.
- - Rigid ureteroscopes are **easier** for stone manipulation.
- - The stones are removed using the stone basket or graspers (size ≤ 0.6 cm)
- - Endoscopic lithotripsy devices are used for stone fragmentation (> 0.6 cm), (ultrasonic, laser or pneumatic).

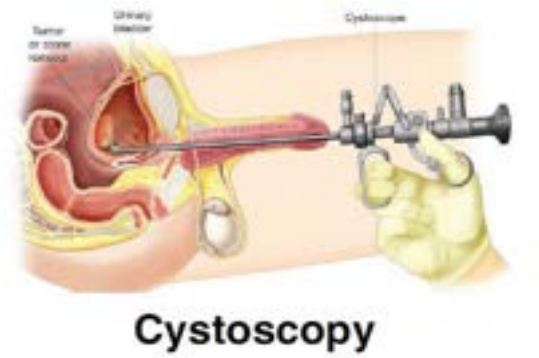
- Complications:

1. i) Failure.
2. ii) Mucosal abrasions.
3. iii) False passages.
4. iv) Ureteral perforation.
5. v) Ureteral avulsion.
6. vi) Ureteral stricture.



URS

4. Cystoscopic lithotripsy:



- Indication: stones < 2 cm.

- Steps:

i) The stone is crushed cystoscopically by ultrasonic waves or by pneumatic shock waves.

ii) Fragments are then lavage cystoscopically to outside the bladder by Ellik evacuator.

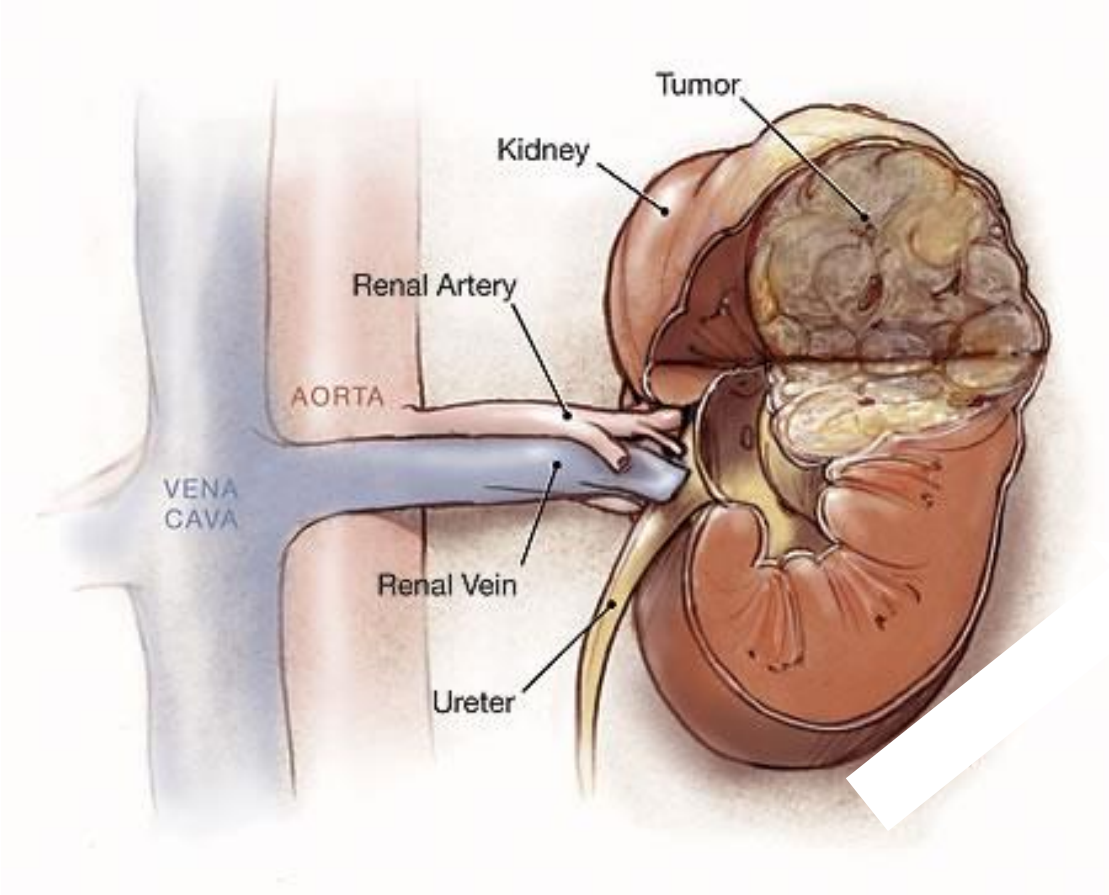
5. Open Pyelolithotomy

6. Open Nephrolithotomy

7. Open Uretrolithotomy

8. Open Cystolithotomy:

RENAL TUMORS



RENAL TUMORS

- **Primary tumors**
- **Tumors of renal parnchyma**
  - Benign: adenoma, oncocytoma, angiomyolipoma
  - Malignant: RCC , Wilm's tumor
- **Tumors of PCS:**
  - Benign: papilloma
  - Malignant: TCC, sq.CC , adenocarcinoma
- **Secondary tumors**
- **Metastatic tumors**
- **leukemia , lymphoma**

Renal Cell Carcinoma

Incidence: It constitutes 3% of adult cancers and 85% of primary malignant renal tumors with an age incidence between 4^{th}-6^{th} decade and a male to female ratio of 2:1. The condition is bilateral in 2% of cases with no side predilection

Etiology:

Risk factors for RCC can be related to

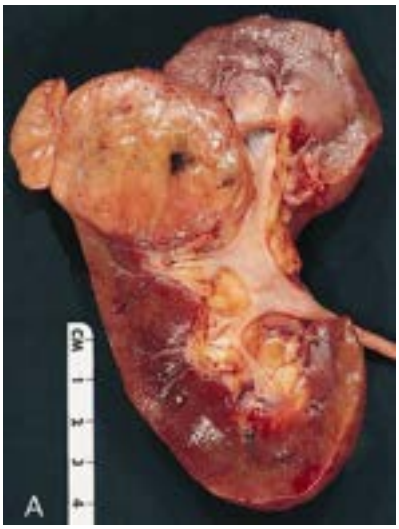
- I- **Life style** related risk factors : Incidence of RC increases with
  - Cigarette smoking: two fold increase in risk
  - Obesity
  - Exposure to some chemicals: cadmium, asbestose
- II- **Hereditary** risk factors : Incidence of RC increases with
- Von Hippel-Lindau disease that occurs due to mutation in VHL gene. It is charcterized by tumors in multiple organs as RCC, cerebellar hemangioblastoma, retinal angiomata, pheochromocytoma
- Hereditary RCC can also occurs in association with some chomosomal changes

- III- **Other** risk factors: Incidence of RC also increases in patients with acquired cystic disease of the kidney and those with +ve family history of renal tumors

Pathology

Gross picture:

- RCC can be bilateral in 2% of cases with no site or side predilection. It may be mutifocal within the same side in 6% of cases. Most tumors are round to ovoid in shape, yellowish to greyish in coloure with areas of hge, necrosis and calcification. The tumor may be circumscribed by a pseudocapsule of compressed parenchyma and fibrous tissue rather than a true histologic capsule.

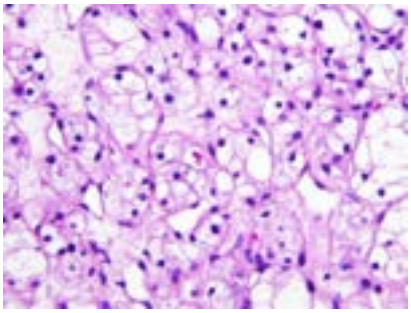


Microscopic picture:

Cell of origin is proximal convoluted tubules

Histopathological types: 5 subtypes

1) Clear cell carcinoma 75%-85%
2) Paillary 14%
3) Chomophobic 4%
4) Collecting duct< 1%
5) Unclassified



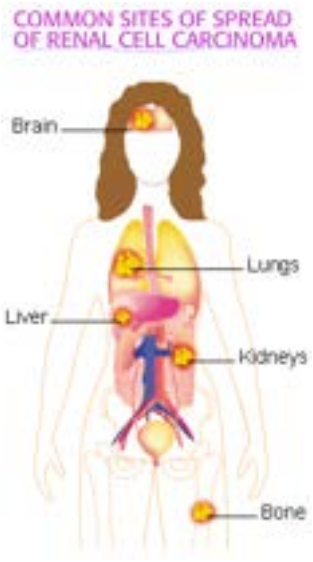
- There is no difference in prognosis between those subtypes

Spread

- Local : to Gerota 's fascia, perinephric fat and to posterior abdominal wall muscles
- Lymphatic spread: to regional L.N. (perirenal L. N. , para-aortic L.N.)
- Vascular spread : to renal vein and IVC
- Bl.born metastases : to lung (cannon ball metastases) , to brain , liver and bones

Clinical Picture

- **Asymptomatic**: in small sized tumor < 4m cm , detected accidentally during imaging investigation for another pathology
- **Classic triad**: 15% of cases
  - Gross hematuria: 40% of cases
  - Abdominal or flank pain : 40% of cases
  - Palpable abdominal mass: 20% of cases
- **Metastases**:
  - Lung : dyspnea, irritative cough
  - Bone : bony boring pain, pathological fracture
  - Liver:;Rt hypochondrial pain , jaundice ,
  - Brain: headache , projectile vomiting
  - 2ndry varicoceles



- **Para-neoplastic syndrome**: in 3-10% of cases

  It is **non-metastatic systemic symptoms**. Its presence at time of diagnosis doesn't mean poor prognosis but its persistence after nephrectomy means residual or recurrent tumor

  Manifestations f paraneoplastic syndrome can include erythrocytosis , hypercalcemia , hypertension , hepatic dysfunction and hormonal changes such as Cushing syndrome, hypo-glycaemia and hyperprolcatinemia

Investigations

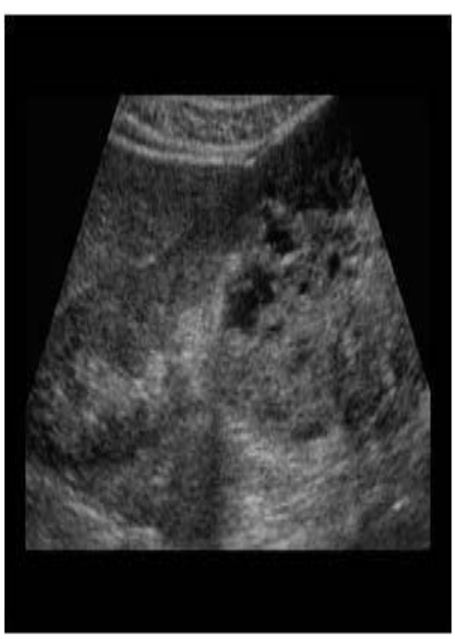
- **Laboratory investigation** :

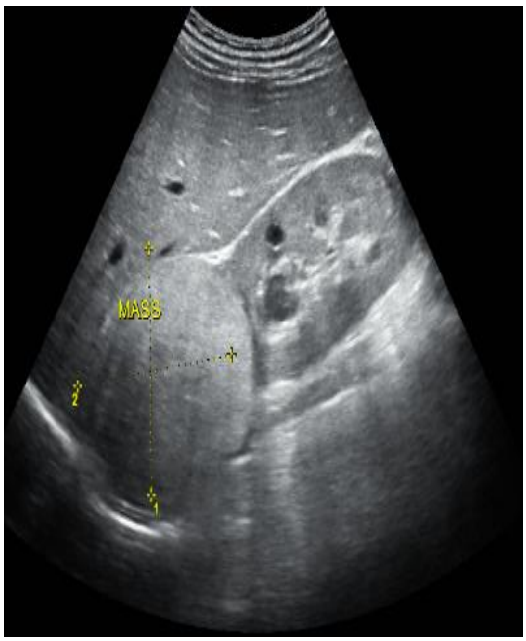
  urine analysis , blood picture , renal function test , liver function test

- **Imaging investigations** :
  - Confirm diagnosis
  - Search for metastases

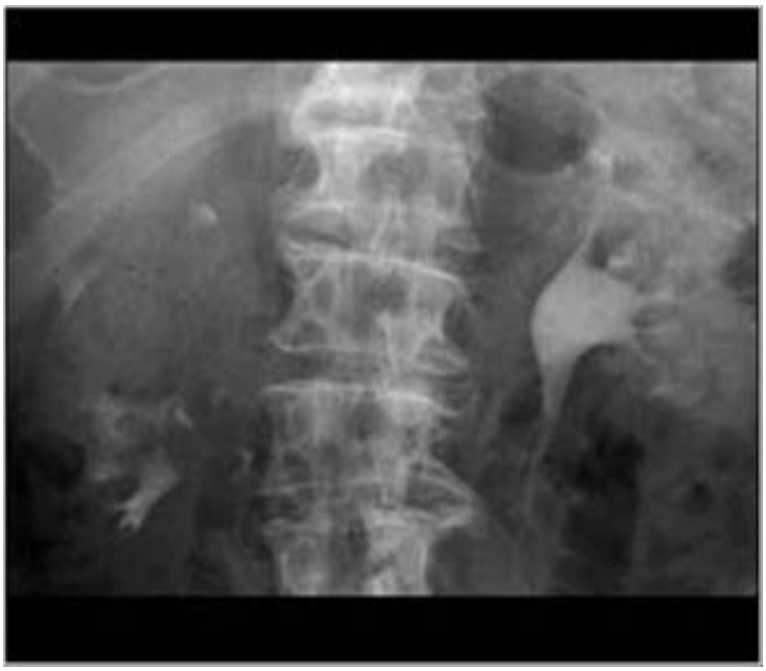
1. Ultrasound :

   helps to visualize the tumor as an echogenic mass in the renal parenchyma and also helps in the differentiation between solid and cystic lesions it may help in tumor staging (abdominal metastases)

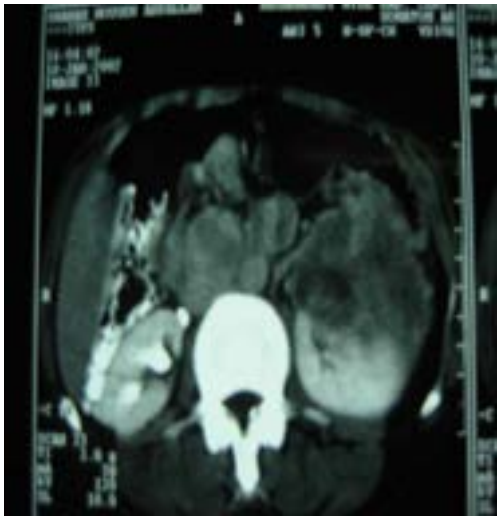


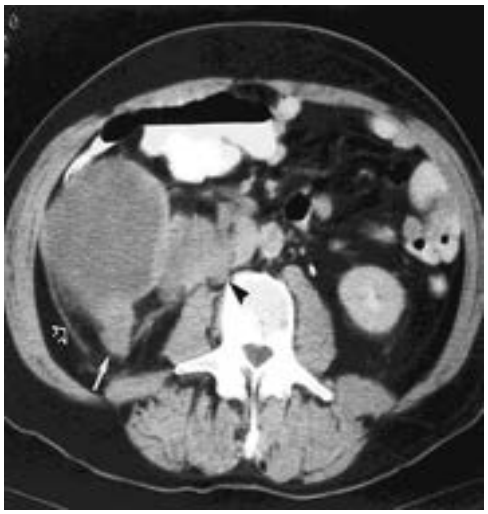


2. IVU : may show rat tail appearance of the calyces or amputated calyx or even non- secreting PCS



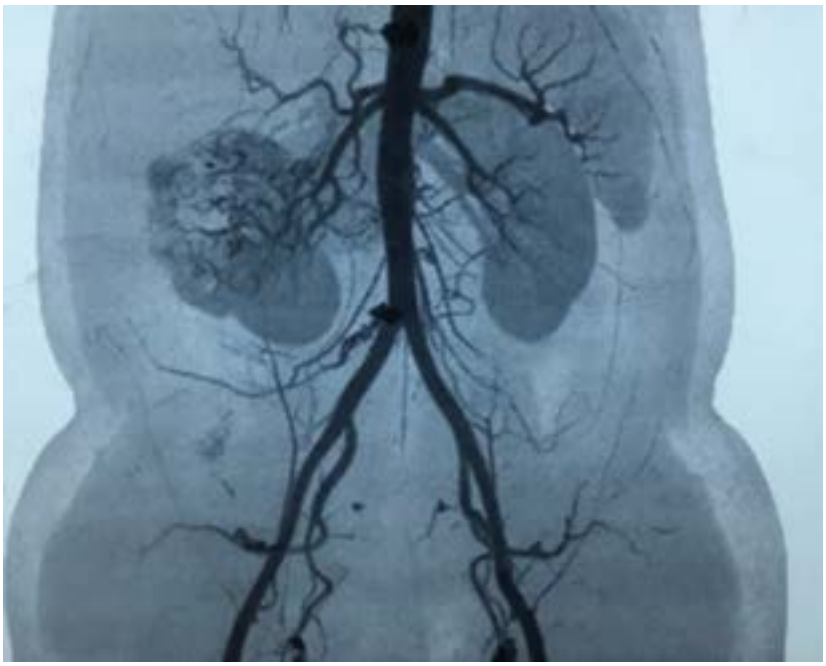
3. Ct scan or MRI (Abdominal and Chest + brain)): it is more accurate than IVU and ultrasonograpgy in the diagnosis and staging of renal tumor .





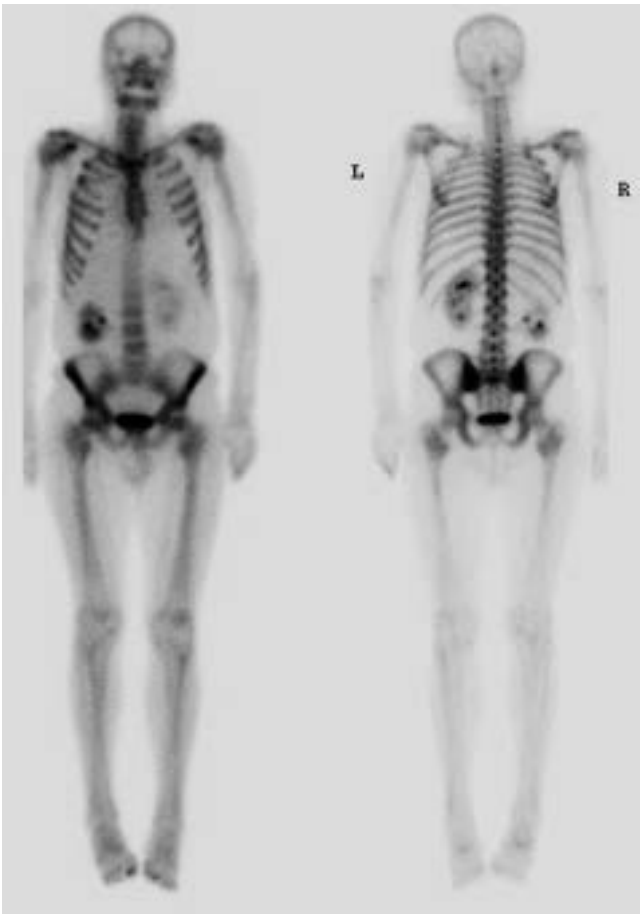
4. Angiography:

   - Detect hyper-neo-vascularity
   - Detect A.V fistula
   - DD between tumors and renal cysts
   - Important before partial nephrectomy



Angiography

5- Bone scan: in case of suspected bony metastases.



Staging: TNM staging

T – primary tumor	Tx	Primary tumor cannot be assessed
	T0	No evidence of priomary tumor
	T1	Tumor ≤ 7cm in its gretest dimensions , limited to the kidney T1a Tumor ≤ 4cm T1b Tumor > 4 cm
	T2	Tumor > 7cm in its greatest dimensions
	T3	Tumor extends to the major veins or directly invades adrenal gland or perinephric tissue but not beyond Gerota's fascia T3a Tumor directly invades adrenal gland or perinephric tissue but not beyond Gerota's fascia T3b Tumor extends to the renal vein or vena cava below the diaphragm T3c Tumor extends to the vena cava above the diaphragm
	T4	Direct tumotr invasion beyond Gerota's fascia
N- Regional lymph nodes	Nx	Regional lymph nodes cannot be assessed
	N0	No evidence of regional lymph nodes metastases
	N1	Metastasis in a single regional lymph node
	N2	Metastasis in more than one regional lymph node
M- metastases	M0	No distant metastases
	M1	distant metastases

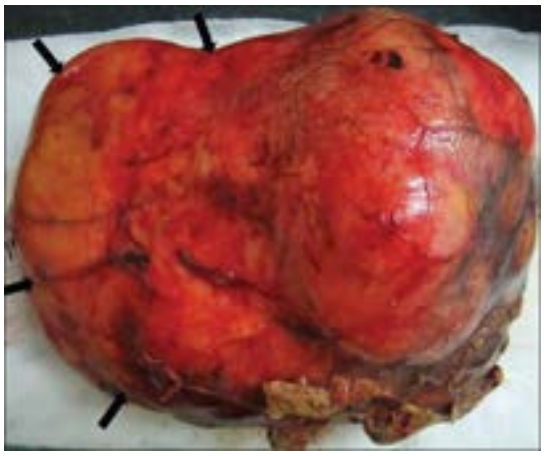
DD of renal mass

- Advanced hydronephrosis: rounded border , tense cystic in consistency, lobular surface
- infected hydronephrosis : high grade fever, tender kid
- pyonephrosis : moderately enlarged , mild tenderness, limited mobility
- renal cyst
- PCK : bilateral renal mass , bossy surface
- renal tumor: hard consistency , irregular surface

Treatment

I- Radical Nephrectomy

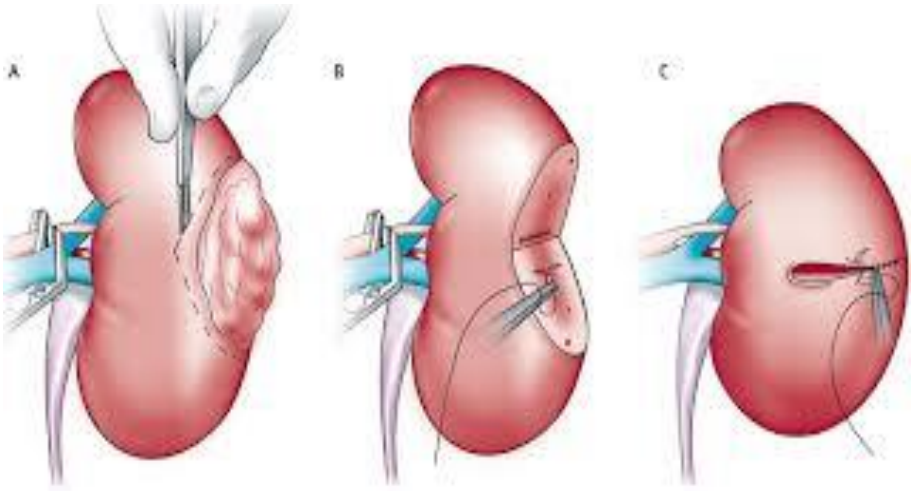
- It is the treatment of choice for patients for organ confined, non-metastatic RCC.
- Kidney is removed outside Gerota's fascia with removal of the ipsilateral adrenal gland and proximal ½ of ureter



II- Partial nephrectomy:

It is indicated in the following cases

1) Bilateral tumors
2) Tumor in an only functioning kidney
3) Small sized tumor< 4cm



III- Treatment of metastatic renal cell

1- Nephrectomy :
To alleviate local symptoms and to get better response to adjuvant therapy

2- Adjuvant therapy

RCC is radioresistent and chemoresistent.

Adjuvant therapy can include

- Radiotherapy can be used to manage metastases(brain metastases)
- Immune therapy: response in 10% of cases
  - Alpha Interferone
  - Interleukin2
- Molecular-targeted therapy:
  - Sunitinib → response in 20% of cases

Prognosis

- 5 yr. survival is stage dependent
- Stage 1→ 80-85%
- Stage 2→ 70%
- Stage 3 → 50%
- Stage 4→ 8%

Other malignant tumors

- Metastatic tumors:
  - The most common malignancies in the kidney,
  - 12% of patients dying of malignancy have renal involvement
- Renal Lymphoma and Leukemia
  - Kidney is involve in 34% of patients dying of progressive lymphoma or leukemia.
  - Usually detected at autopsy

WIlms tumor (Nephroblastoma)

The tumor was named after Max Wilms who characterized the tumor in 1899

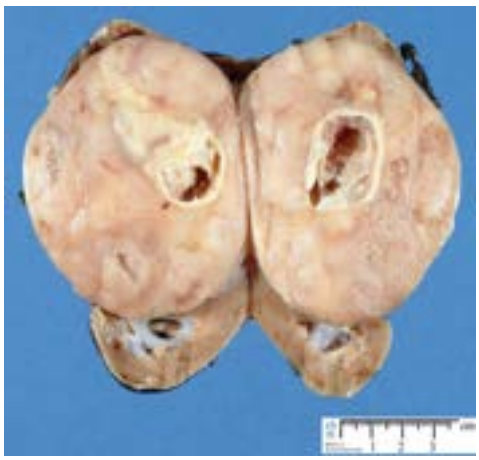
Incidence

- It is the most common primary malignant renal tumor of childhood constituting 80% of childhood GU tumors and 5% of all childhood cancers
- It has an age incidence of 2-5 ys (median age, 3.5 years) with no sex predilection
- Incidence increases in patients with (Associated cong.anomalies)
  - Beckwith-Wiedemann syndrome: characterized by visceromegally o kidney, pancreas and gonads. Neoplasm develops in 10% of cases
  - WAGR syndroms: Wilms tumor , Aniridia , Genito-urinary anomalies, mental Retardation
  - Isolated hemihypertrophy
  - Cong.aniridia
  - Genito.U T anomalies: hypospadias , renal fusion

Pathology

- **Gross P:**

Large lobulated grey usually soft tumor with areas of necrosis and hemorrhage . The tumor usually compresses the adjacent normal renal parenchyma, forming a pseudocapsule . There is no side or site predilection . The tumor is usually unicentric, but may be multicenteric in 7% of cases. It is usually unilateral but may bilateral in 5% of cases



M.P.

- Wilm's tumor arises from abnormal proliferation of the primitive metanephric blastema without differentiation into tubules and glomeruli
- Histopathologically , it is classified into:

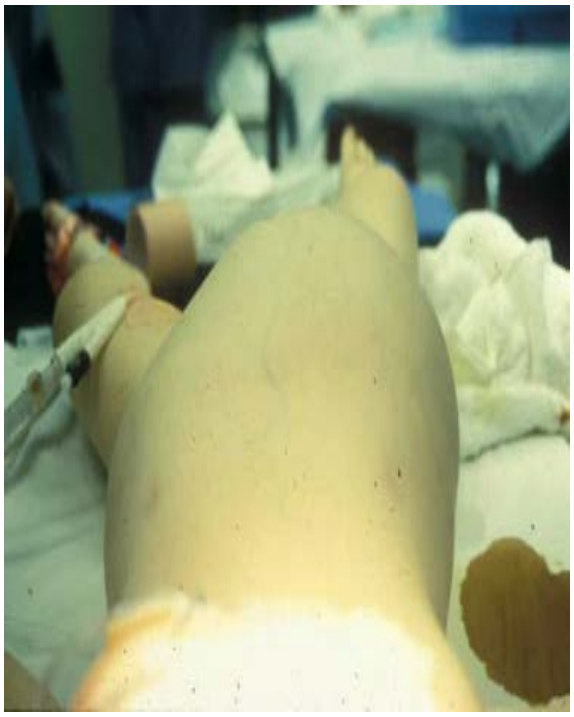
1) Tumors of favorable histology
2) Tumors of un-favorable histology

Tumors of favorable histology	Found in around 90% of cases and has a better prognosis	characterized by the presence of 3 well differentiated components of tissue (Triphasic nephroblastoma) 1. Metanephric blastema: (undifferentiated blastema) 2. Epithelieum: abortive tubules and glomeruli 3. Stroma component: striated Ms, cartilage
Tumors of un-favorable histology:	found in approximately 10% of patients BUT reported in almost half of the tumor deaths	Three subtypes 1. nuclear atypia (anaplasia, focal or diffuse), 2. sarcomatous tumors "rhabdoid“ 3. Clear cell types.

Clinical presentation

- Local symptoms
- Mass: (75% of cases)
  - Palpable, smooth abdominal mass.
  - Rarely crosses the midline
  - The tumor is quite large in relation to the size of the child.(lemon on 4 sticks)
- Pain: (in 30% of cases)
  - Usually diffuse dull ache
  - Acute abdomen may occur in case of rupture of the tumor with hemorrhage into the free peritoneal cavity.

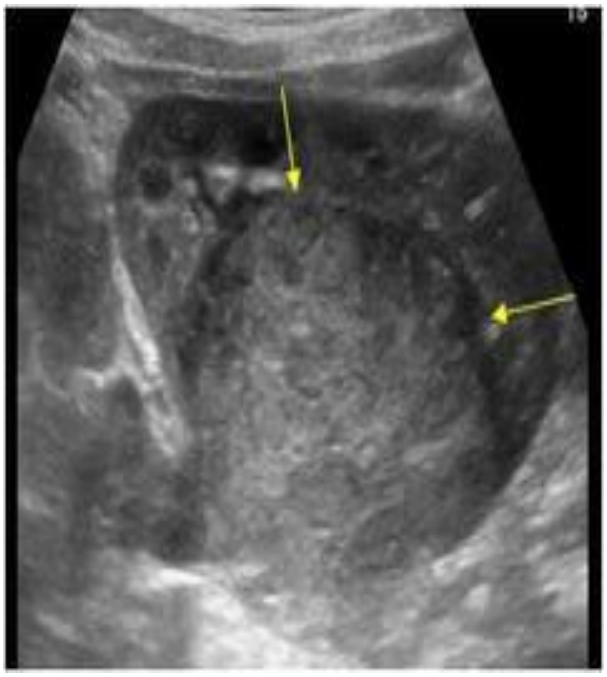
- Gross hematuria .(not common)



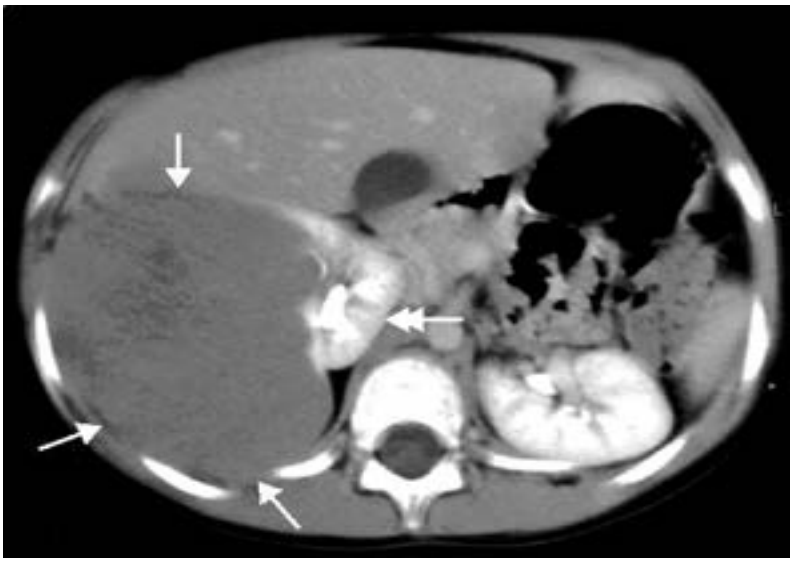
- General symptoms:
  - Fever
  - Hypertension: present in 25% of cases and has been attributed to increased plasma renin levels.
- Non-urologic symptoms
  - due to tumor expansion with compression of adjacent organs
  - Due to metastases
- Associated cong. Anomalies: e.g
  - aniridia, hemihypertrophy, and genitourinary anomalies

Imaging investigations

- ☐ Ultrasound : showing solid mass arising from the kidney



- ☐ CT : to confirm the diagnosis and also for staging



- ☐ Chest CT: to exclude chest metastases
- ☐ IVU

DD of renal mass in children

Cystic

- Hydronephrosis:
  - PUJO, ureterocele, PUV
- Multicystic kidney

Solid

- Neonates: < 1 year
  1. cong. Mesoblastic nephroma
  2. Hepatoma
  3. Leiomyosarcoma
- Child > 1yr
  1. Wilms
  2. neuroblastoma

Features that distinguish between Wilms tumor and Neuroblastoma:

	Wilms Tumor	Neuroblastoma
Abdominal mass	Yes	Yes
Primary origin	Intrarenal	Extrarenal Arising from adrenal gland or paravertebral sympathetic ganglia
Physical examination	Displacing mass, mainly confined to the flank	Non-mobile mass. more likely to cross the midline
Pattern of tumor spread	Direct expansion with displacement of adjacent structures	Encasement of vessels and aortic elevation
Other	Intrinsically displaces urinary collecting systems	Externally displaces kidney Neural extension Often calcified

Staging:-

I	Tumor limited to the kidney and completely excised. • The renal capsule is intact and was not ruptured before removal. • There is no residual tumor
II	Tumor extends through the perirenal capsule but is completely excised • There may be local spillage of the tumor confined to the flak • tumor may have been biopsied • Extarenal vessels may contain tumor thrombus or be infiltrated by the tumor
III	Residual non-hematogenous tumor confined to the abdomen , • L.N. involovement • diffuse peritoneal spillage • peritoneal implants • tumor extending beyond surgical margins • tumor not completely removed
IV	Hematogenous metastases to lung , liver, bone, brain , or other organ
V	Bilateral renal involvement at iagnosis

Treatment

I- **Radical nephrectomy** : removal of the entire kidney within Gerota's fascia

II- **adjuvant therapy**: Following radical nephrectomy, the child is managed with

- Chemotherapy: used in all cases with a combination of actinomycin , vincristine and doxorubicine.
  Chemotherapy can be complicated with : B.M suppression, hepatic dysfunction, cardiomyopathy

- Radiotherapy : used in all cases except stage I favorable histology
  Radiotherapy can be complicated with scoliosis , hypogonadism with temporary azoospermia or ovarian failure and hepatocellular carcinoma

Bilateral Wilm's tumor

- ☐ In about 5% of cases
- ☐ Treatment:
  1. initial biopsy → *followed by*
  2. Preoperative chemotherapy → *followed by*
  3. CT or MRI to assess the reduction in tumor volume → *followed by*
  4. second look surgery to do partial nephrectomy

Prognosis:-

- ☐ 5 year survival
    1. FH: around 85% ~ 95% depending upon stage
    2. UFH : around 50%~60% depending upon stage

Tumors of PCS

Incidence:

- These are tumors affecting the pelvi calyceal system and ureter
- Upper UT urothelial tumors constitute 5-10% of urothelial tumors while bladder tumors constitute 90% of cases
    - Upper UT urothelial tumors are more common in the pelvi calyceal system than the ureter
    - In 10-20% of cases the tumor is multifocal
    - In 20 % of cases, there is concurrent bladder tumor

Classification

- **Benign**: Fibro-epithelial polyp
- **Malignant**
    - TCC : 90% of cases
    - Squamous CC and adenocarcinoma in 10 % of cases that may develop in response to chronic irritation , infection or stone disease
    - Malignant tumors may be multifocal

Diagnosis

Clinical presentation

- ☐ Hematuria : that may be microscopic or macroscopic with the passage of thready blood clots
- ☐ Loin pain : often insidious dull aching pain

☐ Obstructed ipsilateral kidney with hematonephrosis

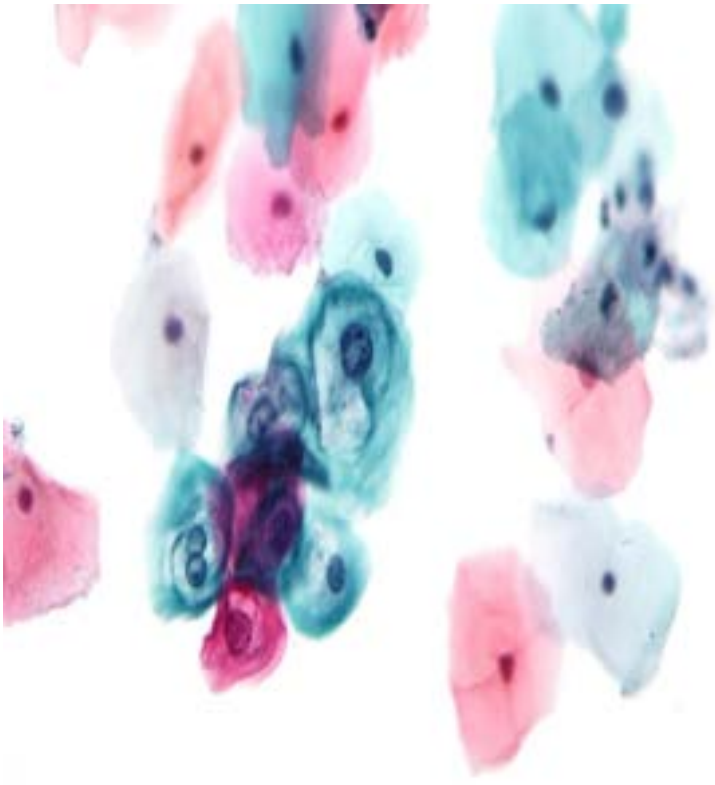
☐ General symptoms: malaise , weight moss (should arouse the suspicion of metastases)

Investigations:

Lab investigations:

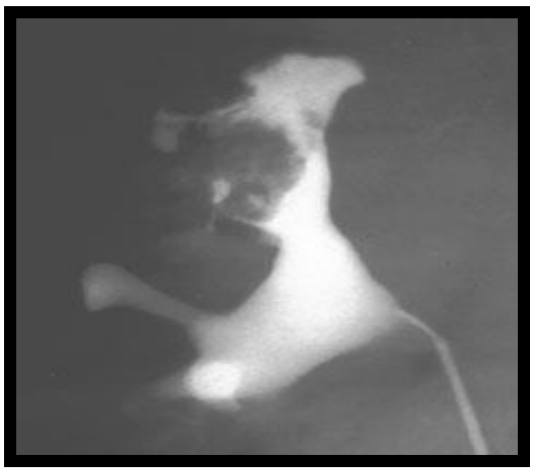
- Urine analysis:
- RBCs
- Urine cytology :
  - Urine sample is centrifuged and the specimen is then stained with papanicolaou stain. This technique can detect malignant cell in urine sample with NO localization of the site of malignancy

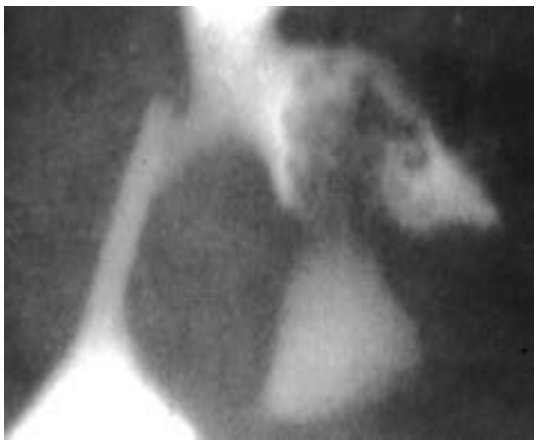
- CBC , renal F T , Liver F T



Imaging investigations:

- ☐ Retrograde uretrography (retrograde injection of dye into the PCS through a ureteric catheter) → filling defect in the PCS
- ☐ IVU or retrgrade ureterography → filling defect in PCS or ureter

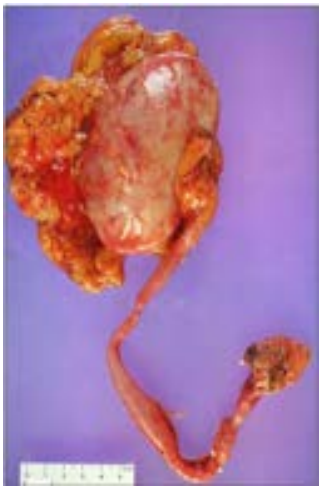




Differential diagnosis

- ☐ Polyp , raduiolucent stone , Fungus ball
- ☐ Ureteroscopic biopsy
- ☐ Pelvi-abdominal CT or MRI for tumor diagnosis and staging
- ☐ Diagnostic cystoscopy and ureteroscopy: to identify tumor criteria and to take biopsy from suspicious lesions
    - ☐ Flexible uretreoscopy is better

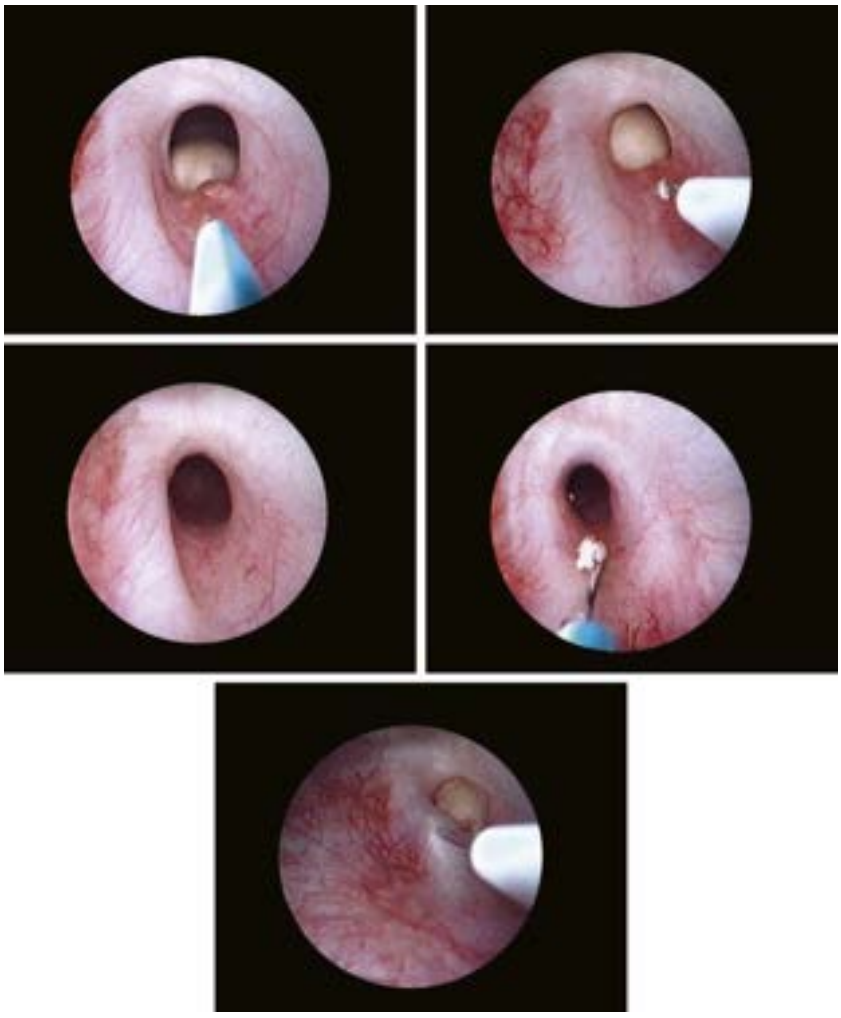
Management



I- Radical nephroureteroectomy:

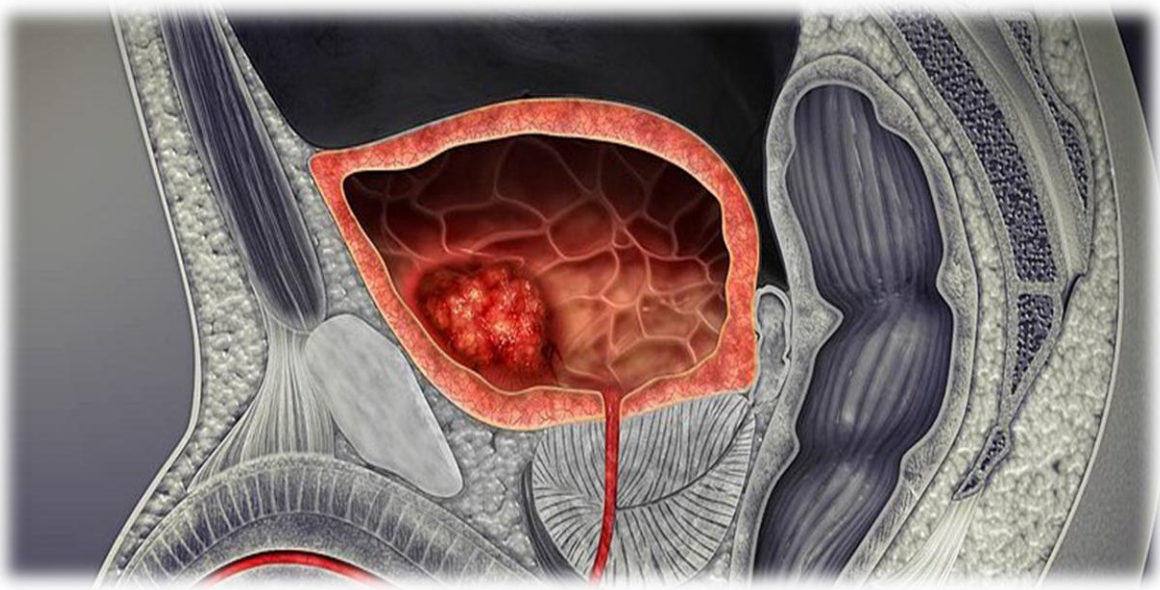
- Indications
    - in patients with non-metastatic tumor that is high grade or muscle invasive or multifocal
- Technique:
    - en-block radical removal of the kidney , entire ureter and a cuff of bladder followed by post-operative bladder instillation of chemotherapy to lower the intra-vesical recurrence rate

- **II- Kidney sparing surgery :**
- Indications:
    - in patients with non-metastatic tumor that is low grade ,unifocal and superficial lesion
    - In patients with tumor in Only Functioning kidney bilateral tumors
- Technique
    - Endoscopic tumor fulguration
- Segmental ureterctomy (resection of the affected segment)



Endoscopic tumor fulguration

BLADDER CANCER



BLADDER CANCER

Incidence

Bladder cancer is more than 2.5 times more common in men than in women

In men, it is the **fourth most common cancer** after prostate, lung, and colorectal cancers, accounting for 6% of all cancer cases

Age

Bladder cancer can occur at any age—even in children for transitional cell carcinoma being sixth and seventh decades of life.

Etiology and Risk Factors

Tumor Suppressor Genes

- Deletions or inactivation of these (so-called cancer suppressor genes) could encourage unregulated growth or failure to direct damaged DNA cells to programmed cell death

1- P53:- The p53 gene is the most frequently altered gene in human cancers

2- Retinoblastoma Gene, Its Product, and Regulators p15, p16, p21, p27, and p19

- Amplification and Overexpression

A third type of carcinogenic genetic mechanism is amplification or overexpression of normal genes that encode for growth factors or their receptors.

Predisposing factors for cancer bladder

1- Occupational Exposure Risk Factors

- 1-Aniline dyesused to color fabrics, are urothelial carcinogens.
- 2-naphthylamine, 3-aminobiphenyl, 4-nitrobiphenyl, 5-diaminobiphenyl (benzidine), and 2-amino-1-naphthol.

Most bladder carcinogens are aromatic amines.

Occupations reported to be associated with increased risk of bladder cancer include

- painter,
- truck driver,
- drill press operator,
- leather worker,

2-Cigarette Smoking **(35%) (smooker: non smooker) (4:1)**

3-Coffee and Tea Drinking

4-Analgesic Abuse

5- Artificial Sweeteners

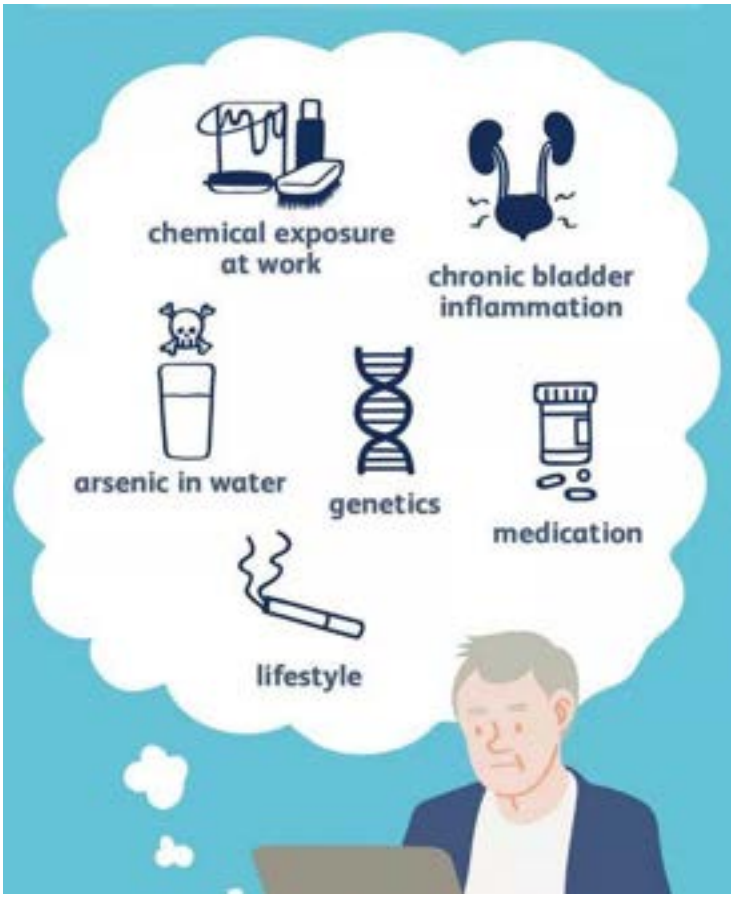
6- Chronic Cystitis and Other Infections

1. **The presence of indwelling catheters**

2. **Calculi** is associated with an increased risk for squamous cell carcinoma of the bladder.

3. ***Schistosoma haematobium*** cystitis appears to be causally related to the development of bladder cancer

4. **The role of exposure to the human,papillomavirus (HPV)**



7- Pelvic Irradiation **+-chemotherapy (2-4 folds)**

8-Cyclophosphamide **(9 fold)**

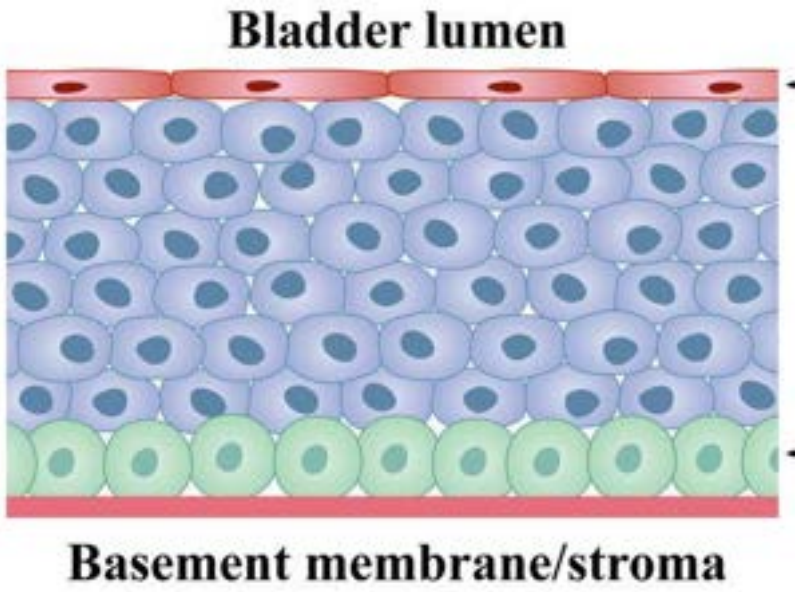
9-Tryptophan Metabolites

Pathology

Normal Bladder Urothelium

The urothelium of the normal bladder is three to seven layers thick.

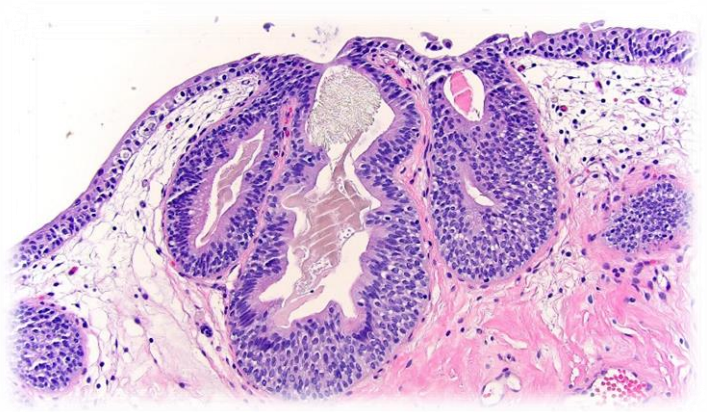
- There is a basal cell layer
- On which rests one or more layers of intermediate cells.
- The most superficial layer is composed of large, flat, umbrella cells.
- The urothelium rests on the lamina propria basement membrane in the lamina propria is a tunica muscularis mucosa containing scattered smooth muscle fibers.



Epithelial Hyperplasia and Metaplasia

- **The term *epithelial hyperplasia*** is used to describe an increase in the number of cell layers without nuclear or architectural abnormalities.
- **Urothelial *metaplasia*** refers to the bladder lining, often in focal areas, demonstrating a nontransitional epithelial appearance, usually with epidermoid (squamous metaplasia) or glandular (adenomatous metaplasia) development.Squamous metaplasia in the absence of **cellular atypia** or **marked keratinization** is a benign condition.
- ***Von Brunn's nests*** are islands of benign-appearing urothelium situated in the lamina propria.
- ***Cystitis cystica*** is von Brunn's nests in which urothelium in the center of the nest has undergone eosinophilic liquefaction.

- ***Cystitis glandularis*** is similar to cystitis cystica except that the transitional cells have undergone glandular metaplasia. Cystitis glandularis may be a precursor of adenocarcinoma.



Urothelial Dysplasia

Is similar to epithelial hyperplasia, except that there are also

- Nuclear abnormalities.
- And partial derangement of the umbrella cell layer.

3-Inverted Papilloma

Benign proliferative lesion associated with chronic inflammation or bladder outlet obstruction.Papillary fronds project into the fibrovascular stroma of the bladder covered by a thin layer of normal urothelium.

4-Vesical Leukoplakia

- *Leukoplakia* is characterized by **squamous metaplasia with marked keratinization**, downward growth of rete pegs (acanthosis), cellular atypia, and dysplasia.
- Generally considered a premalignant lesion that may progress to squamous cell carcinoma in up to 20% of patients.

Urothelial Carcinoma

1-Carcinoma in Situ

- **Gross picture** Carcinoma in situ may appear as a velvety patch of erythematous mucosa, although quite often it is endoscopically invisible.
- **Histologically**, it consists of poorly differentiated transitional cell carcinoma confined to the urothelium.

2-Transitional Cell Carcinoma

Microscopically urothelial (transitional cell) cancers differ from normal urothelium by having an increased number of epithelial cell layers with papillary foldings of the mucosa, loss of cell

polarity, abnormal cell maturation from basal to superficial layers, increased nuclear-cytoplasmic ratio, prominent nucleoli, clumping of chromatin, and increased number of mitoses.

Gross picture Urothelial carcinomas manifest in a variety of patterns of tumor growth, including

1. Papillary (70%)
2. sessile (ulcerative)
3. infiltrating
4. Nodular (10%)
5. Mixed (20%)
6. And flat intraepithelial growth (carcinoma in situ).

3-Squamous Cell Carcinoma

It is 75% in Egypt.

These cancers occur in patients who are, on the average, 10 to 20 years younger than patients with transitional cell carcinoma.

Bilharzial cancers are

- **exophytic, nodular, fungating lesions**
- **usually well-differentiated**
- **Have a relatively low incidence of lymph node and distant metastases. Whether the low incidence of distant metastases is due to capillary and lymphatic fibrosis resulting from chronic schistosomal infection or to the relatively low histologic grade of these tumors is not clear.**

Nonbilharzial squamous cell cancers are usually caused by chronic irritation from

- **urinary calculi,**
- **long-term indwelling catheters** As many as 80% of paraplegics with chronic infections and/or indwelling catheters have squamous changes in the bladder, and about 5% develop squamous cell carcinoma
- **chronic urinary infections**
- **Bladder diverticula.**

Histology

- Squamous cell carcinoma consists, characteristically, of keratinized islands that contain eccentric aggregates of cells called squamous pearls. In a small series of squamous carcinoma patients.

4-Adenocarcinoma

Adenocarcinomas account for less than 2% of primary bladder cancers. They are classified into three groups:

1. Primary vesical,(vesical exstrophy)
2. urachal.
3. Metastatic.

Adenocarcinomas also occur in

- intestinal urinary conduits,
- augmentations,
- pouches, and
- ureterosigmoidostomies.

Patterns of Spread

I-Direct Extension

II-Metastatic Spread

- ***Lymphatic Spread***
- ***Vascular Spread***

liver, 38%;

lung, 36%;

- ***Implantation***

Bladder cancer also spreads by implantation in

- abdominal wounds,
- resected prostatic fossa
- Traumatized urethra.
- Similarly, inadvertent bladder perforation during endoscopic resection

Diagnosis

Signs and Symptoms

- **The most common presenting symptom of bladder cancer is painless hematuria, associated with clots profuse and total**
- The symptom complex of bladder irritability and urinary frequency, urgency, and dysuria can occur
- LOCALLY ADVANCED DISEASE

  Other signs and symptoms of bladder cancer include flank pain from ureteral obstruction, lower extremity edema, and pelvic mass.
- METASTASIS

  Very rarely, patients present with symptoms of advanced disease, such as weight loss and abdominal or bone pain.

Investigations:

Laboratory investigations

Nonspecific: - CBC – urine analysis – kidney and liver function

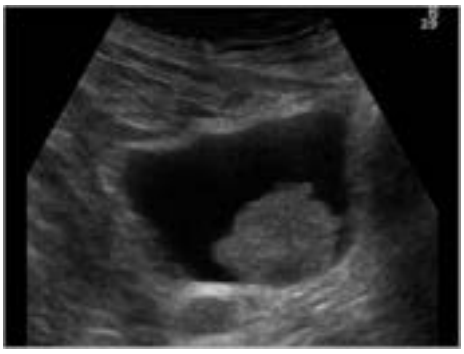
***Specific: - Conventional Microscopic Cytology* – tumor markers**

Radiological investigations

- **Abdominal ultrasound**
- ***Excretory Urography***
- ***Ct abdomen and pelvis with contrast***
- ***PET CT scan***

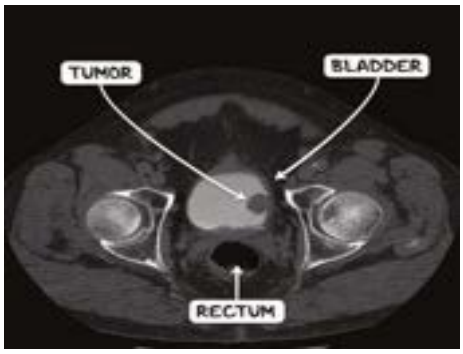
INSTRUMENTAL

***Cystoscopy* and biopsy for Histo-pathological examination**



U/S

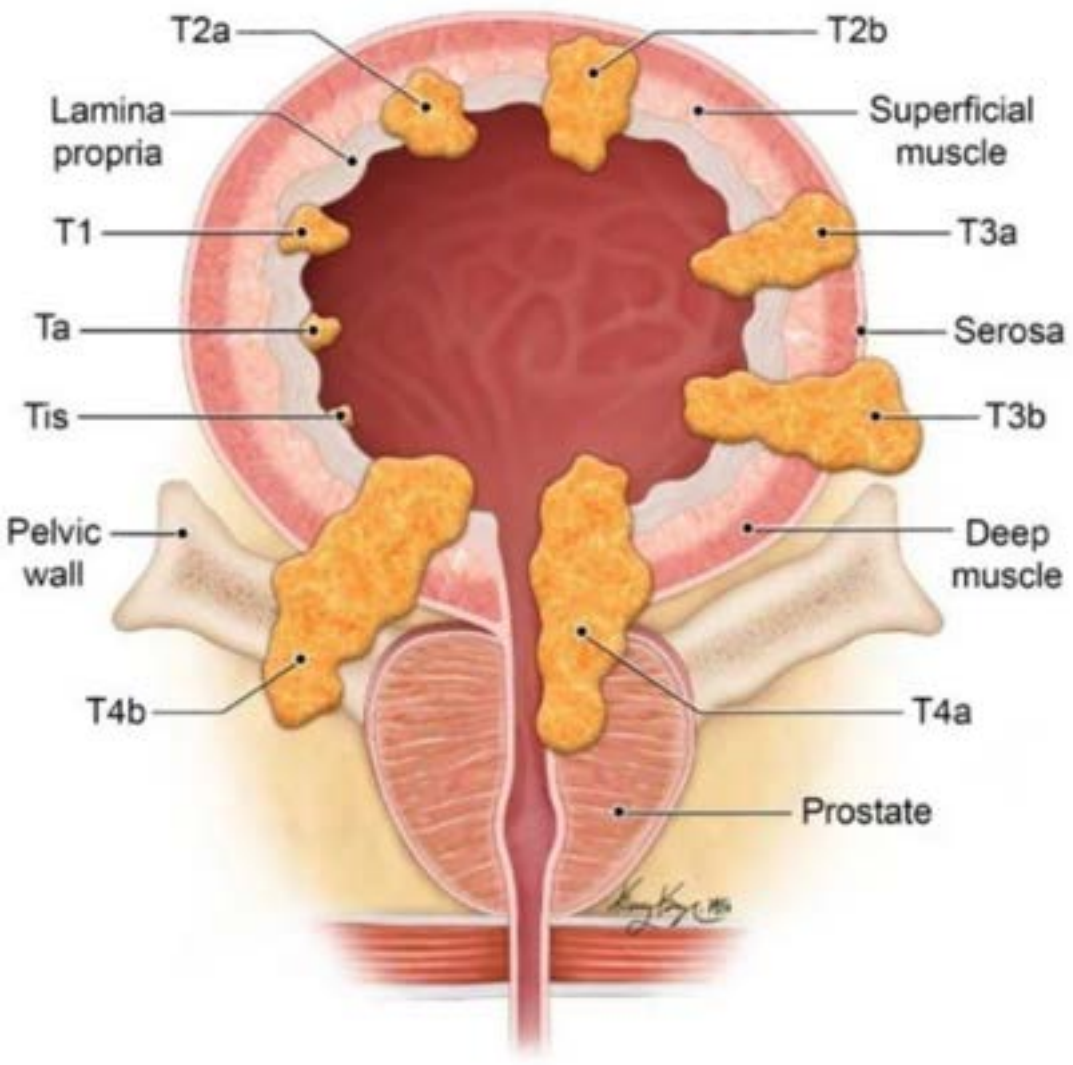
CT



Staging

Staging Systems (T,N,M.)

T-Primary tumour	
TX	Primary tumour cannot be assessed
T0	No evidence of primary tumour
Ta	Non-invasive papillary carcinoma
Tis	Carcinoma in situ
T1	Tumour invades subepithelial connective tissue layer
T2	Tumour invades muscle
	T2a Tumour invades superficial muscle
	T2b Tumour invades deep muscle
T3	T3 tumours -invade perivesical tissue
	T3a -microscopically
	T3b-macroscopically (extravesical mass)
T4	Tumour invades any of the following: prostatic stroma, seminal vesicles, uterus, vagina, pelvic wall, abdominal wall
	T4a Tumour invades prostatic stroma, seminal vesicles, uterus or vagina
	T4b Tumour invades pelvic wall or abdominal wall
N-Regional lymph nodes	
NX	Regional lymph nodes cannot be assessed
N0	No regional lymph node metastasis
N1	Metastasis in a single lymph node in the true pelvis
N2	Metastasis in multiple regional lymph nodes in true pelvis
N3	Metastasis in common iliac lymph node(s)
M-Distant metastasis	
M0	No distant metastasis
	M1a Non-regional lymph nodes
	M1b Other distant metastasis



Treatment

Protocol according to eau guidelines

1- Superficial bladder cancer (NMIBC) refers to Ta, T1, and Tis lesions of any grade.

2- Muscle-invasive and metastatic bladder cancer (categories pT2a and pT2b, pT3a and pT3b or pT4a and pT4).

Superficial bladder cancer

I- Endoscopic Management of Superficial Bladder Cancer

1- TURT

2- Laser Therapy

II- Alternative Therapies

External-Beam Radiation Therapy

III- Intravesical Chemotherapy

1- Bacille Calmette-Guérin (BCG)

2- Chemotherapy instillation (MITOMYCIN C)

IV- Other Forms of Immunotherapy

Patient risk group stratification for management (NMIBC)

Table 2: Risk group stratification	
Low-risk tumours	Primary, solitary, Ta, G1 (low grade), < 3 cm, no CIS
Intermediate-risk tumours	All tumours not defined in the two adjacent categories (between the category of low and high risk)
High-risk tumours	Any of the following: • T1 tumour • G3 (high grade) tumour • CIS • Multiple and recurrent and large (> 3 cm) Ta G1G2 tumours (all conditions must be presented in this point)

Muscle-invasive and metastatic bladder cancer

Radical cystoprostatectomy with suitable urinary diversion

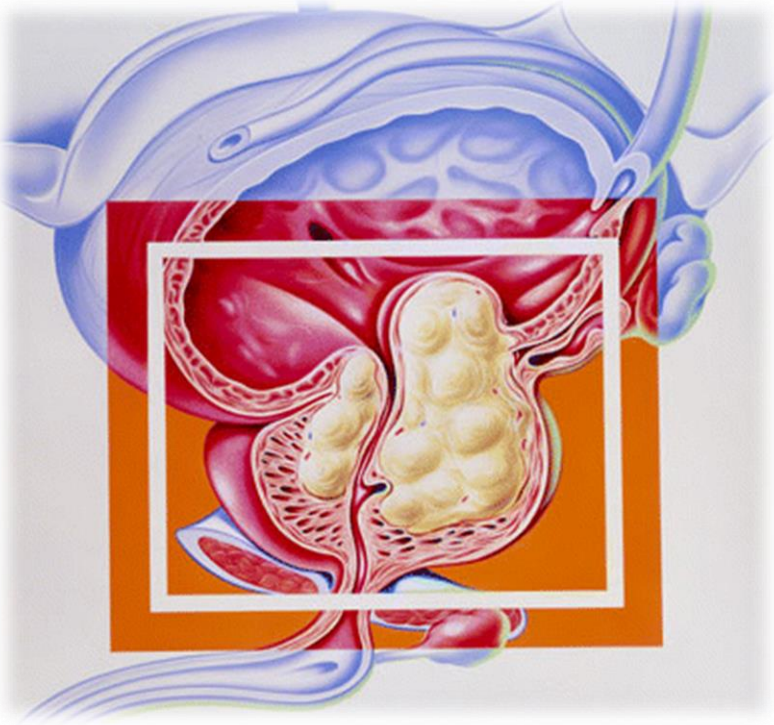
In the male patient and anterior exenteration in the female patient, coupled with en bloc pelvic lymphadenectomy, remain the standard surgical approaches to muscle-invasive bladder carcinoma in the absence of metastatic disease.

Metastatic Bladder Cancer

Systemic Chemotherapy

External beam radiotherapy

BENIGN PROSTATIC HYPERPLASIA



Benign prostatic hyperplasia (BPH)

Benign prostatic hyperplasia is a common disease of the ageing male population significantly impact quality of life by causing lower urinary tract symptoms.

Up to 15-25% of men aged 50-65 years have lower urinary tract symptoms.

Definitions:-

Microscopic: (BPH) refers to histological proliferation.

Macroscopic: senile prostatic enlargement (SPE) refers to organ enlargement due to cellular proliferation.

Clinical: refers to the lower urinary tract symptoms thought to be due to BP obstruction.

However, a significant portion of male lower urinary tract symptoms (LUTS) is due to age-related detrusor dysfunction and other conditions such as polyuria, sleep disorders, and a variety of systemic medical conditions unrelated to the prostate-bladder unit.). It is common for men to have BPE without having LUTS and vice versa.

Etiology:-

Histopathologically, BPH is characterized by an increased number of epithelial and stromal cells in the periurethral area of the prostate and thus correctly referred to as hyperplasia and not hypertrophy.

The observed increase in cell number may be due to epithelial and stromal proliferation or to impaired programmed cell death leading to cellular accumulation.

Androgens, estrogens, stromal-epithelial interactions, growth factors, and neurotransmitters may play a role, either singly or in combination.

Risk factors:-

Aging. Prostate gland enlargement rarely causes signs and symptoms in men younger than age 40. About one-third of men experience moderate to severe symptoms by age 60, and about half do so by age 80.

Family history. such as a father or a brother, with prostate problems means you're more likely to have problems.

Diabetes and heart disease. Studies show that diabetes, as well as heart disease and use of beta blockers, might increase the risk of BPH.

Lifestyle. Obesity increases the risk of BPH, while exercise can lower your risk.

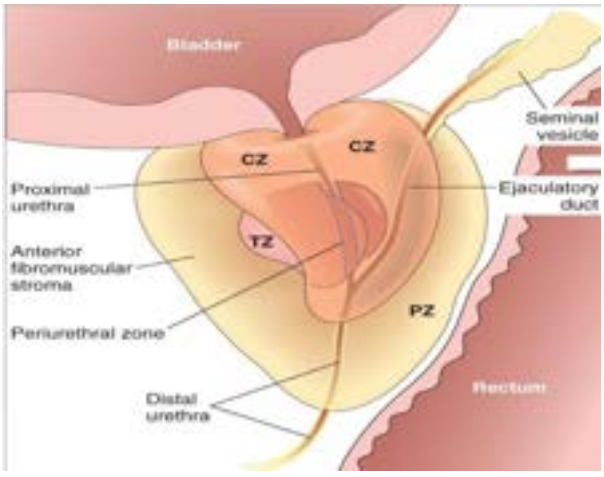
Pathology:-

Anatomic Features

McNeal (1978) demonstrated that **BPH first develops in the peri- urethral *transition zone* of the prostate**

The transition zone consists of two separate glands immediately external to the preprostatic sphincter. The main ducts of the transition zone arise on the lateral aspects of the urethral wall at the point of urethral angulation near the verumontanum. Proximal to the origin of the transition zone ducts are the glands of the *periurethral zone* that are confined within the preprostatic sphincter and course parallel to the axis of the urethra.

All BPH nodules develop either in the transition zone or in the periurethral region. Although early transition zone nodules appear to occur either within or immediately adjacent to the preprostatic sphincter, as the disease progresses and the number of small nodules increases, they can be found in almost any portion of the transition or periurethral zone. However, the transition zone also enlarges with age, unrelated to the development of nodules.



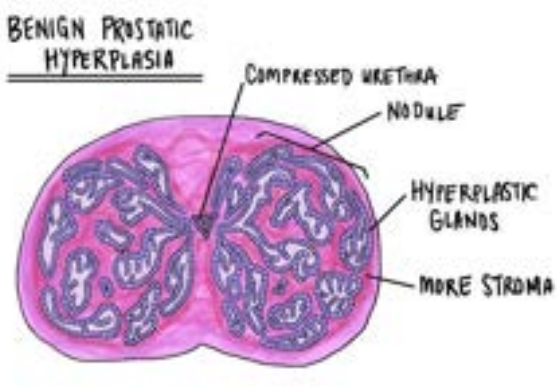
One of the unique features of the human prostate is the presence of the prostatic capsule, which plays an important role in the development of LUTS. Presumably the capsule transmits the "pressure" of tissue expansion to the urethra and leads to an increase in urethral resistance.

Thus the clinical symptoms of BPH in man may be due not only to age-related increases in prostatic size but also to the unique anatomic structure of the human gland.

Clinical evidence of the importance of the capsule can be found in series that clearly document that incision of the prostatic capsule (transurethral incision of the prostate) results in a significant improvement in outflow obstruction, despite the fact that the volume of the prostate remains the same. The size of the prostate does not correlate with the degree of obstruction**.**

Histologic Features

BPH is a hyperplastic and not a hypertrophic process; that is, there is a net increase in the number of cells and not in the size of the cells.



McNeal's studies demonstrate that the majority of **early periurethral nodules are purely stromal in character** (McNeal, 1990). In contrast, **the earliest transition zone nodules represent proliferation of glandular tissue** that may be associated with an actual reduction in the relative amount of stroma. The minimal stroma seen initially consists primarily of mature smooth muscle, not unlike that of the uninvolved transition zone tissue.

These **glandular nodules are apparently derived from newly formed small duct branches** that bud off from existing ducts, leading to a totally new ductal system within the nodule.

This type of new gland formation is quite rare outside embryonic development. This proliferative process leads to a tight packing of glands within a given area as well as an increase in the height of the lining epithelium. There appears to be hypertrophy of individual epithelial cells as well. Again, the observed increase in transition zone volume (TZV) with age appears to be related not only to an increased number of nodules but also to an increase in the overall size of the zone.

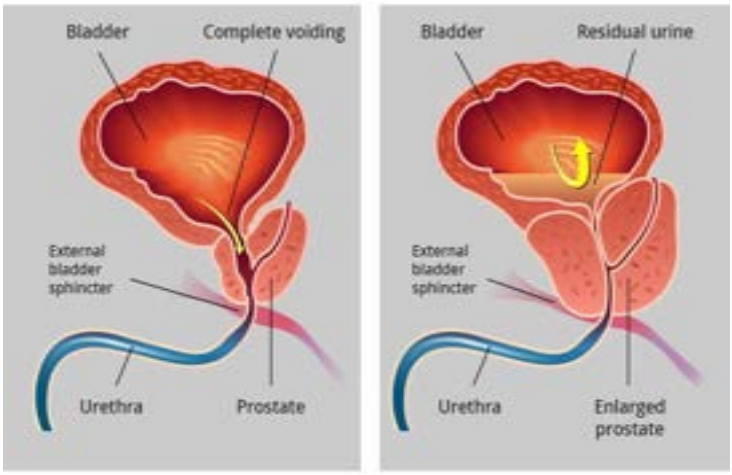
Benign prostatic hyperplasia

The Bladder's Response to Obstruction

Obstruction-induced changes in the bladder are of two basic types.

First, the changes that lead to detrusor instability or decreased compliance are clinically associated with symptoms of frequency and urgency.

Second, the changes associated with decreased detrusor contractility are associated with further deterioration in the force of the urinary stream, hesitancy, intermittency, increased residual urine, and (in a minority of cases) detrusor failure.



The major endoscopic detrusor change, trabeculation, is due to an increase in detrusor collagen

Complications

Acute urinary retention.

Sudden painful inability to void despite full bladder. Is the most common urologic emergency in men .BPH is the most common reason men experience this uncomfortable urinary problem

Chronic urinary retention.

It is relatively painless. Basically, CUR is the persistent inability to completely empty the bladder despite maintaining an ability to urinate. This leads to an elevated post void residual urine volume.

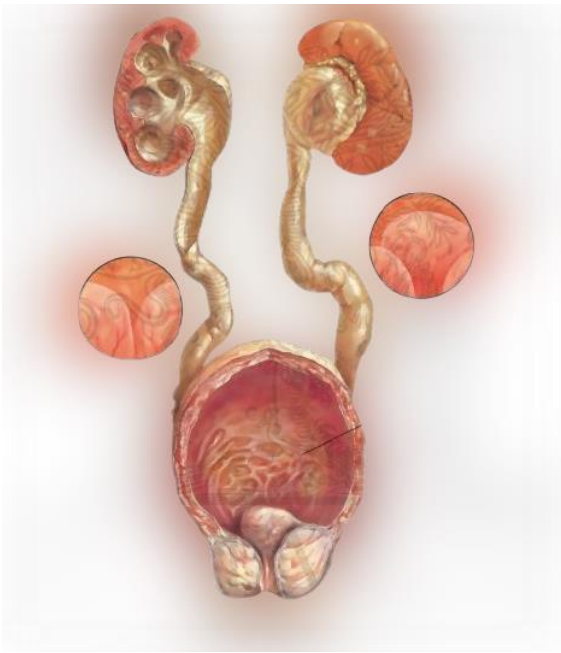
UTI.

Inability to fully empty the bladder can increase the risk of infection in the urinary tract.

Benign prostatic hyperplasia

Bladder stones.

These are generally caused by an inability to completely empty the urinary bladder. Bladder stones can cause infection, bladder irritation, hematuria and obstruction of urine flow.



Bladder damage.

A bladder that hasn't emptied completely can be stretched and weaken over time leading to bladder diverticula. As a result, the detrusor muscle wall of the bladder no longer contracts properly, making it harder to fully empty urinary bladder.

Kidney damage.

Pressure in the bladder from urinary retention can cause urine reflux and Hydro-uretero-nephrosis and renal damage

Many of the complications of progressive BPH are rare, and much of the knowledge comes from studies of men presenting with such complications for treatment (i.e., cases) rather than observing cohorts of men for the development of complications.

Diagnosis:-

Clinical presentation

History

Asymptomatic

International prostate symptom score

7 symptoms with severity grades 0 to 5

Feeling of incomplete emptying

Frequent urination

Intermittency

Urgency, with or without incontinence

Weak or slow stream

Straining

Nocturia

Bother score is an eighth question that is included to assess patient opinion of symptom severity and effect on quality of life

If you were to spend the rest of your life with your urinary condition just the way it is now, how would you feel about that?"

Responses: Delighted, Pleased, Mostly satisfied, Mixed, Mostly dissatisfied, Unhappy, Terrible

Score of 7 or lower: mild quality of life bother

Score of 8 to 19: moderate quality of life bother

Score of 20 or higher: severe quality of life bother

Common additional symptoms included in other, non-validated scores include the following: [2]

Urinary retention

Urinary retention causes back and flank pain

Dribbling

Physical examination

Abdomen

- With urinary retention, a tender, enlarged bladder is palpable
- Urinary retention causes ureteral dilation and hydronephrosis

Back and flank pain will most likely be bilateral, but it may be more prominent on one side or the other, depending on individual anatomy

Digital rectal examination

- Palpable prostate enlargement is usually present but is not necessary for a diagnosis of benign prostatic hypertrophy
- Firmness or nodularity may indicate prostate cancer

The diagnosis of BPH is generally made based on:

Symptoms or the IPSS standardized questionnaire, self-administered by the patient.

Digital rectal examination.

PSA blood test, urinalysis, and, in some cases, a transrectal ultrasound or cystoscopy.

Differential diagnosis

Urinary tract infection (UTI),

prostatitis,

urinary stones in the lower urinary tract,

urethral stricture disease,

neurogenic or overactive bladder,

prostate or bladder cancer,

congestive heart failure.

Some common medications, such as over-the-counter cold medicines containing α-adrenergic agents, can exacerbate LUTS and can even put a man into acute urinary retention if he has underlying BPH.

Laboratory assessment

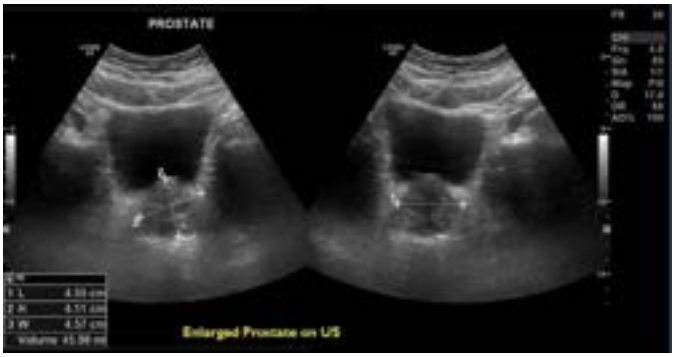
Urine analysis to rule out hematuria.

Like an abnormal digital rectal examination, a urinalysis that shows persistent red blood cells, Urine culture.

Serum creatinine and blood urea nitrogen

PSA testing the traditional upper limit of normal for PSA testing has been 4.0 ng/mL, recent guidelines are

In-office bladder ultrasound scanners are very useful to quickly assess residual urine.



Treatment

Treatment of BPH is always individualized to the patient and involves evaluation of symptoms and bother along with objective findings from examination and laboratory results.

Active Surveillance and Watchful Waiting

For many men, especially those with lower symptom scores and little bother, annual monitoring with digital rectal examination, PSA, urinalysis, and symptom assessment are all that is required.

Complementary and Alternative Medicine

supplements containing a variety of chemicals, including zinc, saw palmetto [7], vitamin E [1].

. Aside from a few European clinical trials of saw palmetto, the use of supplements to help BPH is speculative. Neither supplement had any effect on prostate cancer or BPH to our knowledge.

Medical Therapy

α-Blocker Medications

These agents are directed at the dynamic component of BPH and LUTS by relaxing the smooth muscle tissue in the bladder neck and prostate.

5α-Reductase Inhibitors

The 5α-reductase inhibitors. Finasteride (Proscar) was the first agent in this class (5 mg/day) and is a type 2 inhibitor. Dutasteride (Avodart; 0.5 mg/day) is a type 1 and 2 inhibitor that was approved in 2002. Both drugs prevent the conversion of testosterone to the more active metabolite dihydrotestosterone in the prostate. This inhibition results in involution of BPH tissue and prostatic shrinkage.

Minimally Invasive Procedures

Balloon dilation, transurethral microwave therapy (TUMT), and transurethral needle ablation (TUNA)

The prostatic urethral lift procedure (UroLift)

utilizes small, permanently implanted anchors placed cystoscopically that physically retract and compress the obstructing lobes of the prostate and can provide almost immediate improvement in flow rates and symptom scores.

Water vapor thermal therapy (Rezum) utilizes radiofrequency-generated water vapor (103°C) injected cystoscopically into the prostate tissue, resulting in gradual resorption of the treated tissue and significant improvements in flow rates and symptom scores over the course of weeks following the procedure.

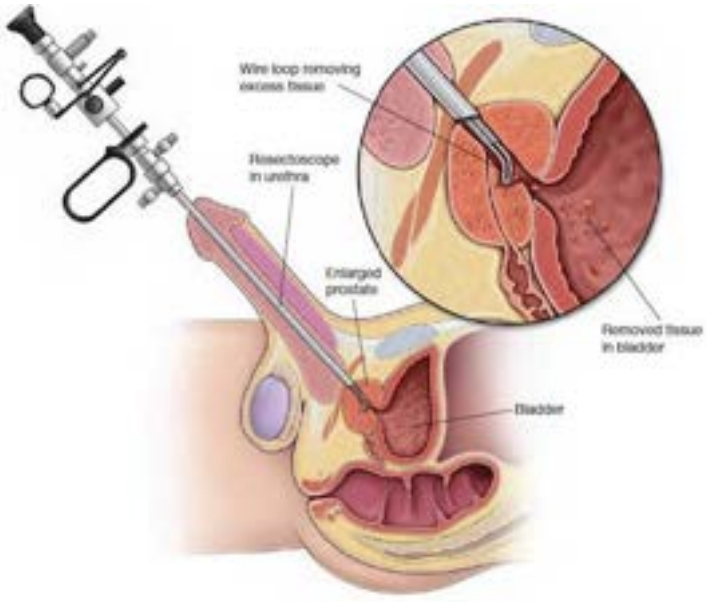
Prostatic artery embolization (PAE) has shown promise in several studies

. High-intensity focused ultrasound was granted FDA approval in 2015 for ablation of prostate tissue but has not gained wide use for BPH management

Surgical Therapy

TURP remains the gold standard treatment for men who have failed to improve on medical therapy, who dislike or are intolerant of medication side effects, or who have developed urinary retention, renal failure secondary to BPH, or large bladder calculi. TURP is associated with relatively low rates of incontinence and erectile dysfunction; however, retrograde ejaculation is an expected and permanent outcome for most men undergoing this surgery. Evolutions of the traditional

TURP procedure now utilize saline irrigation, thereby reducing the risk of hypernatremia during the procedure.

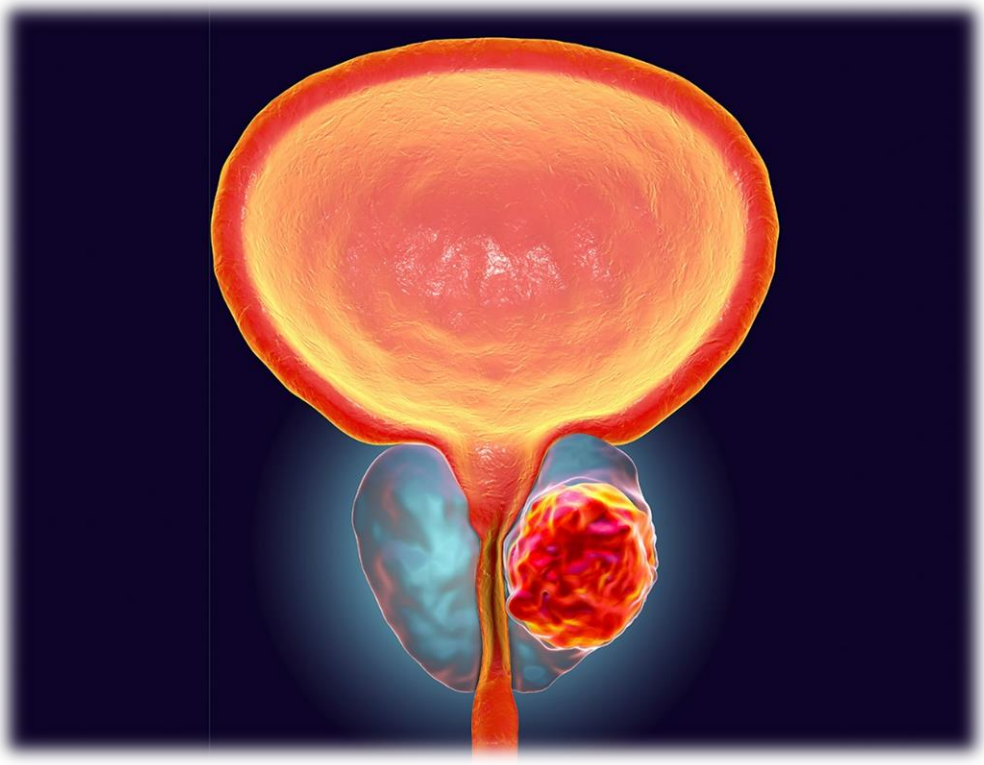


Several laser technologies (Greenlight, Holmium, or Thulium) utilized for the enucleation or vaporization of prostate tissue have resulted in reduced hospital stays, transfusion rates, and duration of catheterization compared with TURP and are recommended treatment options for medically complex cases calling for anticoagulant therapy.

Aquablation utilizes a transurethral water jet to ablate prostate tissue under ultrasound guidance.

Finally, for men with severe BPH and very large glands (>150 g), simple prostatectomy performed robotically or via a small suprapubic incision remains a valuable and highly effective surgical treatment. As with radical prostatectomy used for the treatment of prostate cancer, open or robotic prostatectomy for BPH should be performed by urologic surgeons who are highly experienced in this are

PROSTATE CANCER



Prostate Cancer

(A) Epidemiology and aetiology:

- **Growth of prostate cancer** (PC), like benign prostatic epithelium, is largely under the promotional influence of ***testosterone*** and its potent metabolite ***dihydrotestosterone***.

- **Androgen ablation** by orchidectomy results in programmed epithelial cell death (apoptosis) and involution of the prostate.

- PC is not seen in eunuchs or people with congenital deficiency of 5AR.

- ***Oestrogens***, including phyto-oestrogen isoflavones (genistein, daidzein) found in foodstuffs used in Asian and oriental cuisine, have a similar negative growth effect on PC. This may explain why these races rarely develop (or die of) prostate cancer.

- Other possible ***dietary inhibitors*** of PC growth include vitamin D, the antioxidants lycopene (present in cooked or processed tomatoes) and polyphenols (pomegranate, blueberry, green tea, red wine), isothiocyanates in cruciferous vegetables (sprouts, broccoli), and omega-3 unsaturated fatty acids present, for example, in mackerel and other oily fish. Conversely, arachidonic and linolenic acids and omega-6 polyunsaturated fatty acids (present in high-fat red meat) promote PC cell growth *in vivo* and increases the risk of advanced PC in prospective cohort studies.

- **Obesity** does not confirmed as risk of PC diagnosis but appears to be associated with more aggressive disease.

Other risk factors:

• **<u>Age</u>**:

is an important risk factor for development of histological PC, the disease being **<u>rare below 40y</u>** and becoming increasingly common with rising age, according to post-mortem studies

• **Geographic variation**:

The disease is commoner in Western nations, particularly Scandinavian countries (where low sunlight and vitamin D synthesis may be implicated) and North America. The disease is rare in Asia and the Far East, but US migrants from Asia and Japan have a 20- fold i risk. This suggests an environmental aetiology, such as a Western diet, may be important.

• **Ethnicity**:

Black men are at greatest risk, then Caucasians; Asians and oriental races develop PC uncommonly, unless they migrate to the West. The world's highest incidence is amongst African Americans and Jamaicans.

• **Family history**:

5% of PCs are believed to be inherited. Hereditary PC tends to occur in younger (<60y) men who have a family history. The risk of a man developing PC is doubled if there is one affected first- degree relative and is 4-fold if there are two.

• **Exercise**: appears to confer protection against PC.

Incidence:

- The diagnosis of PC is increasing every year, probably as a result of increasing use of serum PSA testing for both symptomatic and asymptomatic men and the use of more extensive prostatic biopsy protocols.

- PC is **the most commonly diagnosed ♂ cancer** (excluding skin) in the UK and USA.

- The lifetime risk of a man being diagnosed with PC is estimated to be 1 in 8. Most are diagnosed with clinically localized disease, aged 65–79y.

Prevalence:

While the incidence of PC continues to rise (now 78% of all men), the true prevalence of the disease is highlighted by post-mortem studies carried out on men who died of unrelated causes. These have demonstrated histological evidence of PC in 10% of men in their third decade, 34% in the fifth decade, and rising to 67% in the ninth decade.

Mortality:

It is estimated that 3% of men die of PC.

Survival:

Survival rates for PC have been improving for the past 30y. The relative 10y survival rate for men diagnosed was 84% in 2011, compared with only 21% for men diagnosed in 1971–75. The 5y survival for men diagnosed with metastatic PC has improved from 25% to 35%.

Dietary and lifestyle interventions:

- High fat consumption: results in increased production of insulin and IGFs. Diets rich in saturated fat, such as arachidonic, linolenic and omega-6 fatty acids, promote PC cell growth *in vivo* and increases the risk of advanced PC.

- Obese men generally have lower PSA, but higher risk for high-grade or extracapsular disease at presentation, recurrence post-treatment, metastasis, and death.

- Soy products: contain phyto-oestrogens, including the isoflavone genistein. Genistein is a natural inhibitor of tyrosine kinase receptors and inhibits PC cell lines.

- Lycopene: present in cooked tomatoes and tomato products, is considered to reduce the risk of PC progression and inhibits cell lines.

- Selenium supplementation: (0.2mg/day = 2 Brazil nuts) was shown to reduce the risk of developing PC in a melanoma prevention trial. Selenium is a trace element required as an antioxidant.

- Vitamin E supplementation was shown to reduce the incidence of PC in Finnish smokers. It is an antioxidant.

- Vitamins A (retinoids) and D: both inhibit the growth of PC cell lines, and vitamin D receptor polymorphisms appear to predispose certain individuals to PC.

- Pomegranate juice: appears to reduce PSA doubling time during relapse following RP for high-risk disease.

- Green tea: contains polyphenol catechin and antioxidant compounds.

- Coffee: consumption has been associated with PC prevention and improved outcomes, in particular four cups/day of non-filtered coffee that contains diterpenes cafestol and kahweol plus antioxidants that have demonstrated anti-neoplastic activity.

- Exercise: confers a preventative/protective effect against PC development.

(B) Pathology—adenocarcinoma

- By far, the **commonest (>95%) primary PC is *adenocarcinoma***, a glandular epithelial malignancy of the acinar or ductal epithelium.

- The basal cell layer is absent, and the basement membrane is breached by the malignant cells which invade into the prostatic fibromuscular stroma.

- Macroscopically, they tend to be hard and white, though a soft mucin-producing variety exists.

- The prostatic urethra, ducts, or stroma may be invaded by *TCC* of the bladder.

- Prostatic *sarcomas*, the commonest of which is the rhabdomyosarcoma, are rare and mostly seen in childhood.

- *Secondary deposits* (metastases) from other primary sites are rare.

Adenocarcinoma of the prostate

- **Most (75%) adenocarcinomas** are located in the **peripheral zone**, and most (85%) are multifocal.

- Twenty per cent arise more anteriorly in the transition zones, and 5% in the embryologically distinct central zone.

- The tumour spreads locally through the flimsy prostatic capsule (this is absent at the apex and base of the gland) into the surrounding tissue, at which time it is termed '*locally advanced*'.

- The disease may involve the urethral sphincter, corpora of the penis, seminal vesicles, or trigone of the bladder including the distal ureters, but rarely invades through Denonvilliers fascia to involve the rectum.

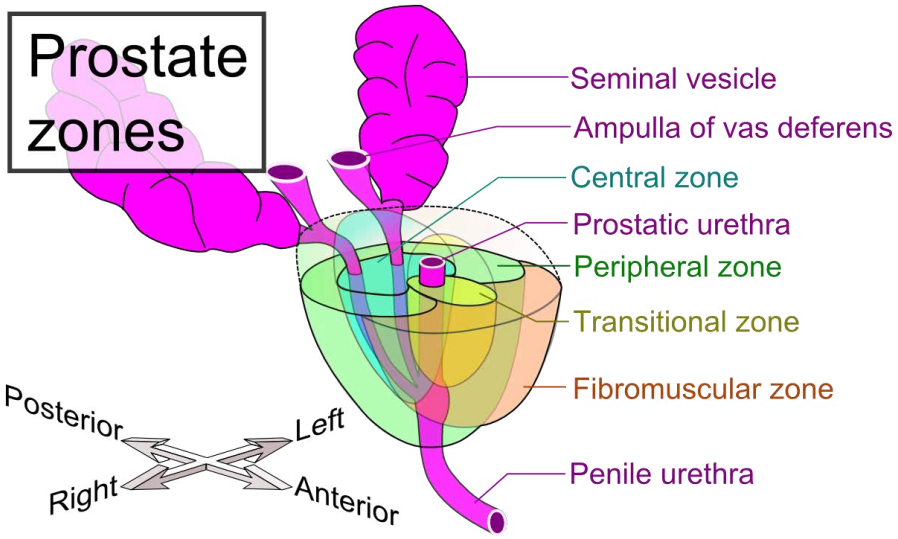
- Local spread is often along the course of autonomic nerves, so-called perineural invasion.

- The **most frequent** sites of ***metastasis* are bone** and lymph nodes of the obturator fossae, internal, external, and common iliac arteries, and presacral regions.

- Soft tissue metastases in the lung, liver, testis, and brain are less common.

- **Bone metastases are characteristically sclerotic, rarely lytic.**

- The axial skeleton (spine, ribs, and pelvis) are most commonly affected, followed by the proximal long bones, clavicles, and skull.



(C) Prostate cancer grading:

- Adenocarcinoma of the prostate is graded by the Gleason system.

- Using low-power microscopy, adenocarcinoma is graded 1–5, according to its gland-forming differentiation (Fig. 1).

- Since most PCs are multifocal and heterogeneous, allowance is made by adding the two dominant grades to give a score of between 2 and 10. If only one pattern is observed, that grade is doubled to give the score. This system is used to grade needle biopsies, TURP, and RP specimens.

- Gleason scores of 2–6 are considered well differentiated; 7 is moderately differentiated, and 8–10 are poorly differentiated.

- Pattern 1&2: Discrete Well-formed Glands
- Pattern 3: discrete glandular units varying in size and shape, with infiltration amongst non-neoplastic acini; most regard as clinically insignificant, without metastatic potential; controversially, some have questioned whether it should even be called cancer!
- Pattern 4: glands with fused, cribriform, glomeruloid, or non-luminal sub-patterns.
- Pattern 5: no glandular differentiation; solid sheets, cords, comedocarcinoma (with central necrosis).

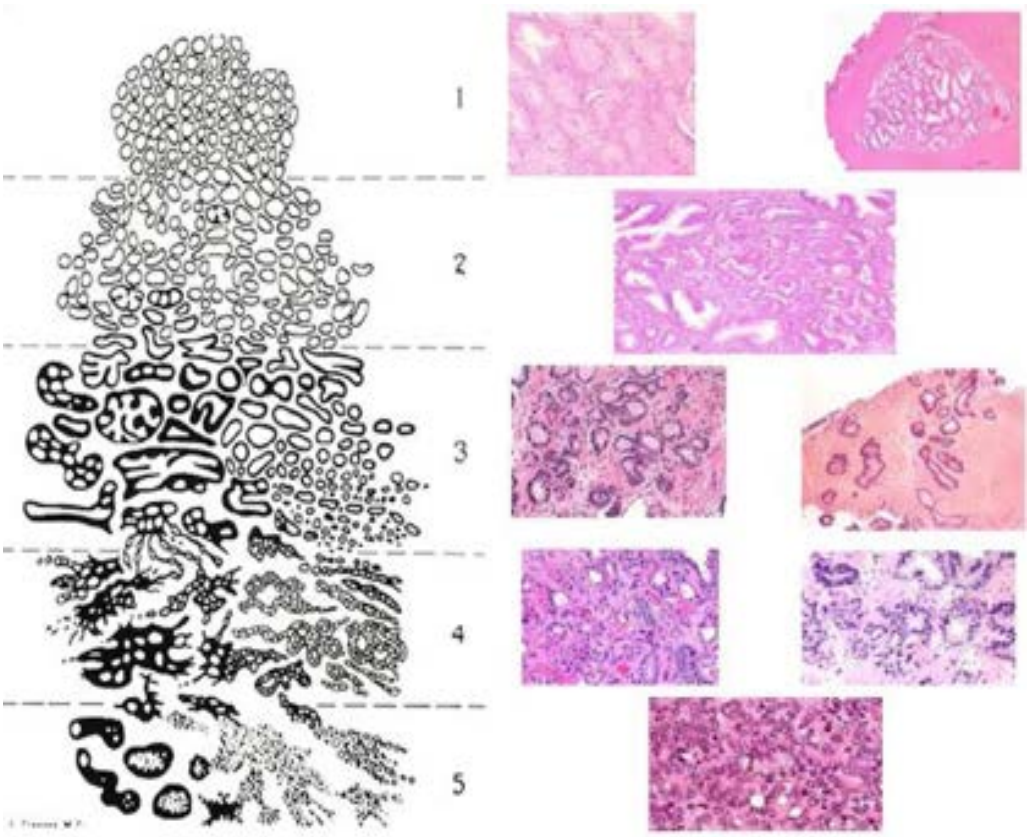


Fig. 1 Gleason grades

In 2016, the Who accepted a simplifying arrangement of Gleason scores into 5 ‘grade groups’?

Grade group	Gleason score	Description
Grade 1	3 + 3 = 6	Only individual discrete, well-formed glands
Grade 2	3 + 4 = 7	Predominantly well-formed glands with a lesser component of poorly formed, fused, or cribriform glands
Grade 3	4 + 3 = 7	Predominantly poorly formed, fused, or cribriform glands with a lesser component of well-formed glands
Grade 4	4 + 4, 3 + 5, and 5 + 3 = 8	Only poorly formed, fused, or cribriform glands, or predominantly well-formed glands with a lesser component lacking glands, or predominantly lacking glands with a lesser component of well-formed glands
Grade 5	4 + 5, 5 + 4 = 9, and 5 + 5 = 10	Lacking gland formation or with necrosis with or without poorly formed, fused, or cribriform glands

(D) Staging and imaging:

- PC staging is by TNM classification (Table 2).

- *T stage* is assessed by DRE (Figure 2), imaging (TRUS, MRI), or examination of RP specimens.

- Imaging resolution limits the reliability in detection of multifocal and microscopic extraprostatic disease. Only 60% of cancers are visible on TRUS, and only 40% of pT3 tumours will be detected.

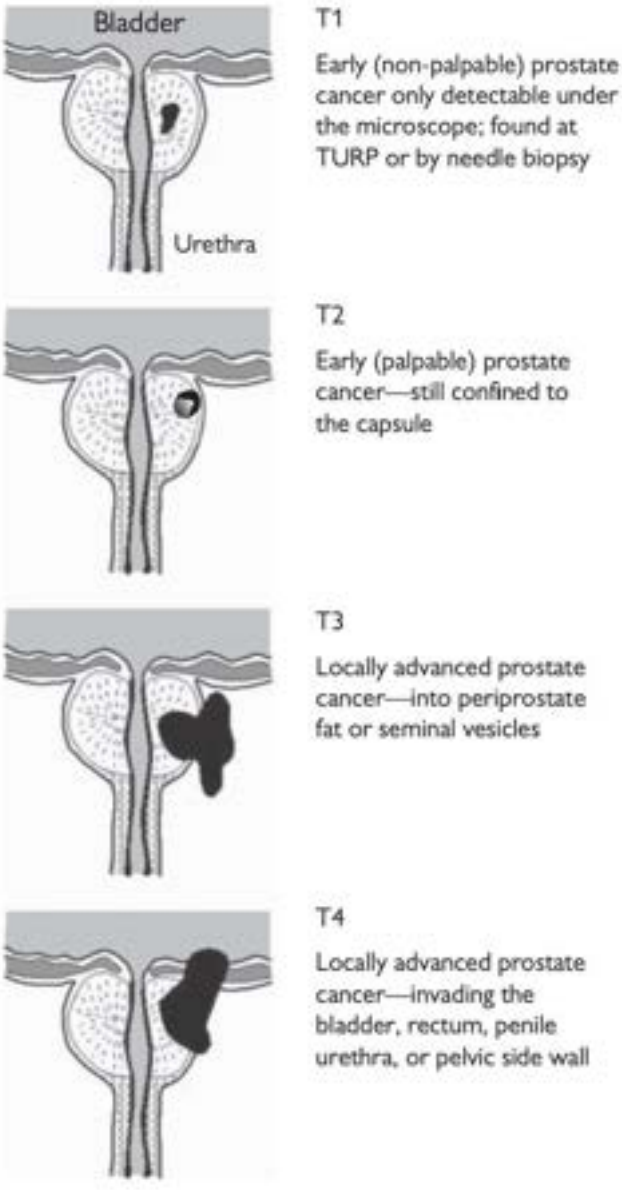


Fig. 2 The T stages of PC

Stage	Definition
Primary tumor	
TX	Primary tumor cannot be assessed
T0	No evidence of primary tumor
T1	Clinically, the tumor is neither palpable nor visible with imaging
T1a	Tumor is an incidental histologic finding in 5% or less of tissue resected
T1b	Tumor is an incidental histologic finding in more than 5% of tissue resected
T1c	Tumor identified with needle biopsy (eg, because of an elevated PSA level)
T2	Tumor confined within the prostate
T2a	Tumor involves one-half of one lobe or less
T2b	Tumor involves more than one-half of one lobe but not both lobes
T2c	Tumor involves both lobes
T3	Tumor extends through the prostate capsule
T3a	Extracapsular extension (unilateral or bilateral)
T3b	Tumor invades seminal vesicle(s)
T4	Tumor is fixed or invades adjacent structures other than seminal vesicles: bladder neck, external sphincter, rectum, levator muscles, and/or pelvic wall
Regional lymph nodes	
NX	Regional lymph nodes were not assessed
N0	No regional lymph node metastasis
N1	Metastasis in regional lymph node(s)
Distant metastasis	
MX	Distant metastasis cannot be assessed (not evaluated with any modality)
M0	No distant metastasis
M1	Distant metastasis
M1a	Nonregional lymph node(s)
M1b	Bone(s)
M1c	Other site(s) with or without bone disease

Table 2 TNM classification of PC

(E) Clinical presentation

Since the introduction of serum PSA testing in the late 1980s, the majority of new patients have non-metastatic disease at presentation. Shown below are possible presentations, grouped by disease stage.

Localized prostate cancer (T1–2)

- Asymptomatic; detected in association with elevated or rising serum PSA or incidental abnormal DRe.
- Incidental finding on MRI or PeT imaging performed for other reasons.
- LUTS (in most cases due to coexisting benign hyperplasia causing Boo).
- haematospermia;
- Hematuria (in most cases due to coexisting benign hyperplasia).
- Perineal or voiding discomfort (probably due to coexisting prostatitis).

Locally advanced cancer, non-metastatic (T3–4N0M0)

- Asymptomatic; detected in association with elevated or rising serum PSA, incidental abnormal DRe or imaging.
- LUTS.
- Haematospermia.
- Hematuria.
- Perineal or voiding discomfort.
- Symptoms of renal failure/anuria due to ureteric obstruction.
- Malignant priapism (rare).
- Rectal obstruction (rare).

Metastatic disease (N1 or M1a, b, or c)

- Asymptomatic ('occult disease'); detected in association with elevated/ rising serum PSA, incidental abnormal DRe or imaging.
- lymphadenopathy (e.g. cervical, inguinal) with proven biopsy diagnosis.
- Swelling of lower limb(s) due to lymphatic obstruction.
- Bone pain, pathological fracture.
- Anorexia, weight loss.
- Neurological symptoms/signs in lower limbs (spinal cord compression).
- Anaemia; bleeding tendency (coagulopathy).
- Dyspnea, jaundice.

Prostatic-specific antigen

- PSA is a 34-kDa **glycoprotein enzyme** produced by the columnar acinar and ductal prostatic epithelial cells.

- It is a member of the **human kallikrein family**, and its function is to liquefy the ejaculate, enabling fertilization.

- PSA is present in **both benign and malignant cells**, although the expression of PSA tends to be reduced in malignant cells and may be absent in poorly differentiated tumours.

- Large amounts are **secreted into the semen**, and small quantities are found in the urine and blood.

- The function of serum PSA is unclear, although it is known to liberate insulin-like growth factor (IGF) type 1 from one of its binding proteins.

- Seventy-five per cent of circulating PSA is bound to plasma proteins (complexed PSA) and metabolized in the liver, while 25% is free and excreted in the urine.

- Complexed PSA is stable, bound to α-1 antichymotrypsin and α-2 macroglobulin. Free PSA is unstable, recently found to consist of two isoforms—pro-PSA is a peripheral zone precursor, apparently elevated in the presence of prostate cancer, and benign PSA (BPSA) is the transition zone precursor and associated with BPH.

- The normal range for the serum PSA assay in men is <4.0ng/mL, though this varies with age. (Table 3)

Age range	Normal PSA range (ng/mL)
All ages	<4.0
40–49	<2.5
50–59	<3.5
60–69	<4.5
>70	<6.5

Table 3 shows a published age-specific normal range (95th centile).

- In the absence of prostate cancer, serum PSA concentrations also vary physiologically, according to race and prostate volume.

- PSA is prostate-specific, but not PC-specific. Other causes of elevated serum PSA are shown in Table 4, the commonest of which is BPH.

- In the presence of infection or instrumentation, PSA should be requested at least 28 days after the event, to avoid a false-positive result, which may cause unnecessary anxiety.

- PSA should not be requested within 2 days of ejaculation or vigorous cycling.

- No significant change to PSA values after a normal DRe.

Cause of elevated PSA	Minor elevation <1.0ng/mL	Intermediate elevation 1.0–20ng/mL	Major elevation 20–100ng/mL
Benign hyperplasia	√	√	
UTI		√	√
Acute prostatitis		√	√
Chronic prostatitis	√	√	
Retention/catheterization		√	
Biopsy, TURP		√	√
Ejaculation, vigorous cycling	√	√	

Table 4. Conditions excluding PC which cause elevated PSA

Indications for checking serum PSA

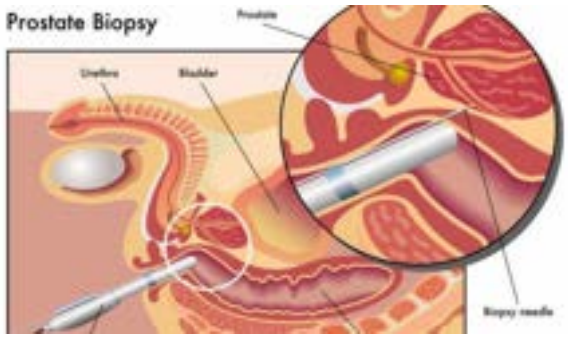
- Patient request, following counselling.
- LUTS.
- Abnormal DRE.
- Progressive bone pain, especially back pain.
- Unexplained anemia, anorexia, or weight loss.
- Spontaneous thromboembolism or unilateral leg swelling.
- Monitoring of prostate cancer patients.

(F) Diagnosis of PC:

1- DRE

2- Multiparametric MRI (**mpMRI**)

3-Trans-rectal ultrasonography (**TRUS**) and TRUS guided biopsy →



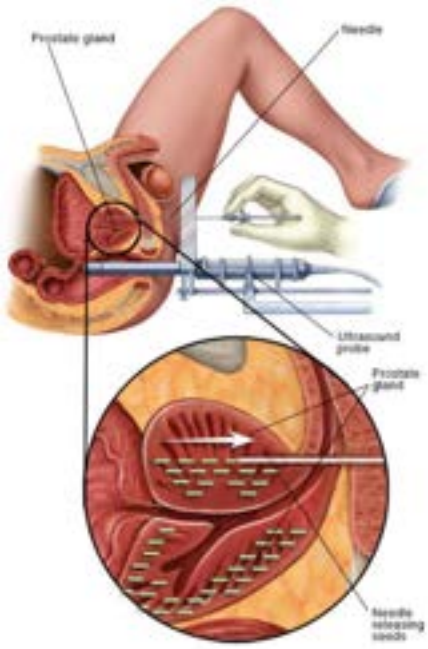
(G) Lines of treatment:

1- Watchful waiting.

2- Active surveillance.

3- Radical prostatectomy.

4- Radical external beam radiotherapy. →



5- Brachytherapy.

6- Hormone therapy (Androgen Deprivation therapy):

a- Surgical castration: bilateral orchidectomy.

b- Medical castration:

LHRh agonists.

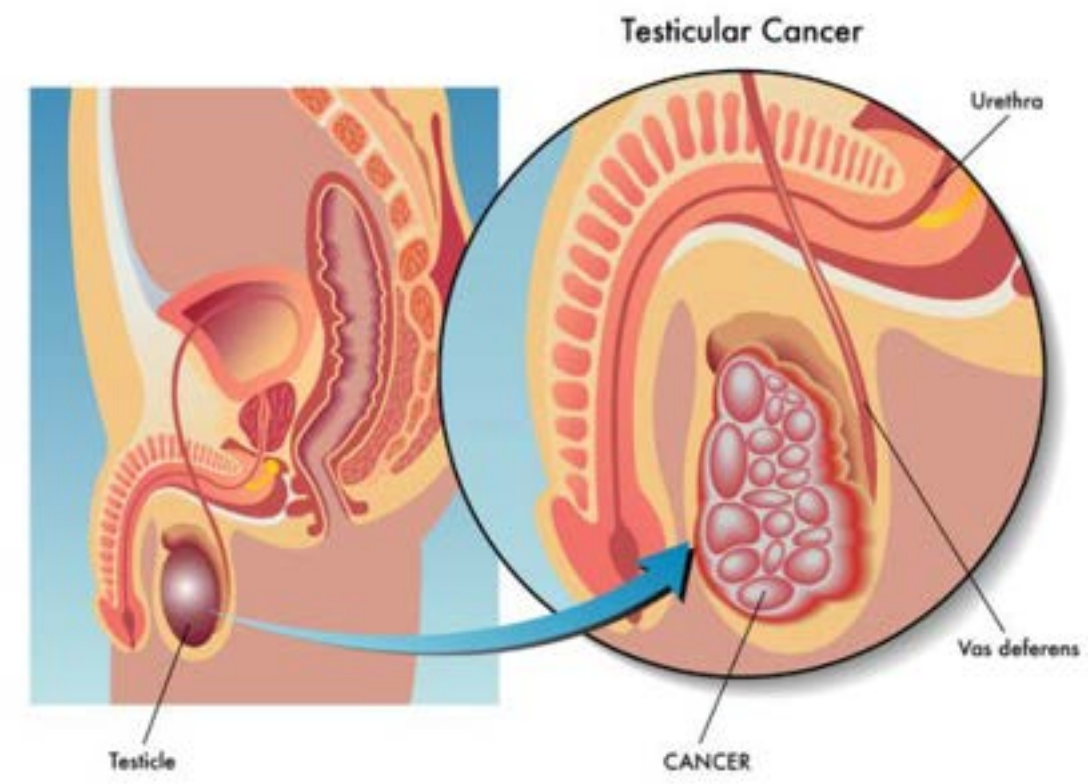
LHRh antagonists.

Oestrogens.

c- Maximal androgen blockade (MAB): medical or surgical castration plus antiandrogen;

7- Chemotherapy.

TESTICULAR TUMORS



TESTICULAR TUMORS

INCIDENCE

- It is the most common malignancy in men in 15 to 35 year age group and the second most common cause of death in men between 35 to 40 years.
- Approximately 2% to 3% of testicular tumors are bilateral, occurring either simultaneously or successively
- Seminoma is the most common histologic type.

Predisposing factors

1. Undescended testis.
    1. Approximately 7–10% of testicular tumors develop in patients who have a history of cryptorchidism esp. if intra-abdominal
    2. Intra-abodominal testis has 3- 14 folds higher risk of malignant transformation
    3. Seminoma is the most common form of tumor in these patients.
    4. Placement of the cryptorchid testis into the scrotum (orchiopexy) does not alter the malignant potential of the cryptorchid testis; however, it does facilitate periodic examination and early tumor detection.
2. Exogenous estrogen administration to the mother during pregnancy → testicular dysgenesis →associated with 3-5 folds higher risk of developing testicular tumors
3. trauma and infection-related testicular atrophy

Classification

- Primary tumors:

1- Germ cell tumors 90-95%

- ☐ Seminoma
- ☐ Non-Seminoma

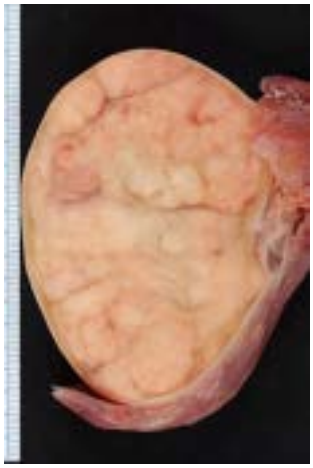
2- Non germ cell tumors 5-10%:

Gonadoblastoma, Sertoli cell tumor, Leydig cell tumors

- Secondary tumors: lymphoma, leukemia

TESTICULAR TUMORS

Germ cell tumors



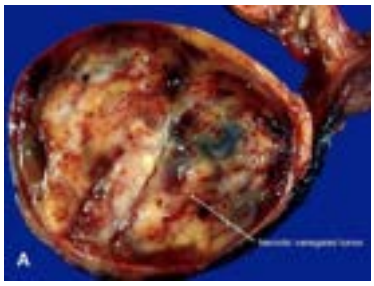
I-Seminoma 35%: occurs in adults

3 sub-types: classic- anaplastic - spermatocystic

II-Non- Seminoma: 4 sub-types

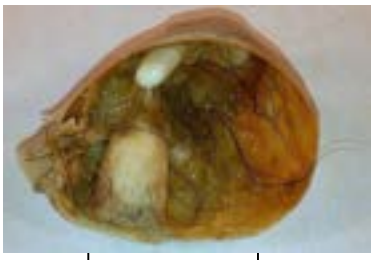
A) Embryonal cell carcinoma 20-25%: occurs in children and adults

The Infantile form is called (yolk sac tumor) and it is the most common testicular tumor of infants and children.



B) Teratoma 5%: It is characterized by the presence of more than one germ cell layer in various stages of maturation and differentiation.

C) Choriocarcinoma 1%: It is an aggressive tumor characterized by early hematogenous widespread metastatic disease with paradoxically, small intratesticular lesions.



Teratoma

D) Mixed cell type: 40%

- ☐ Combination of different histological variants

III- CIS: Carcinoma in Situ (CIS):

- It is expected in the contralateral testis in 5% of patients with unilateral tumor esp. in case of contralateral testicular atrophy (warrants contralateral testicular biopsy)
- Treatment: external beam radiation therapy.

Spread

- **L.N. metastases:**
- Germ cell tumors of the testis typically spread in a stepwise lymphatic fashion to retroperitoneal lymph nodes from level L1 to L4.
- Invasion of the epididymis or spermatic cord may allow spread to the distal external iliac and obturator lymph nodes.
- Scrotal violation or invasion of the tunica albuginea may result in inguinal L.N.metastases

Clinical presentation:-

Symptoms

- A painless gradual enlargement of the testis with sensation of testicular heaviness.
- ROLE of 10

1. 10% of cases present with Acute testicular pain (may be the result of intratesticular hemorrhage or infarction)
2. 10% of cases present with Metastases.
    - ☐ Back pain (retroperitoneal metastases involving nerve roots) is the most common symptom.
    - ☐ cough or dyspnea (pulmonary metastases);
    - ☐ anorexia, nausea, or vomiting (retroduodenal metastases);
    - ☐ bone pain (skeletal metastases);
    - ☐ Lower extremity swelling (venacaval obstruction).
3. 10% of cases are asymptomatic at presentation with incidental detection following trauma, or self-examination.

Signs

1. A testicular mass: There may diffuse enlargement of the testis or hard area within the substance of the tunica albuginea. The mass is typically firm or hard in consistence, non-tender and separable from the epididymis. It may be surrounded by secondary hydrocele
2. Palpation of the abdomen may reveal bulky retroperitoneal disease; assessment of supraclavicular, scalene, and inguinal nodes should be performed.
3. Gynecomastia: esp. found in non-germ cell tumors

Testicular tumors markers: 3 markers

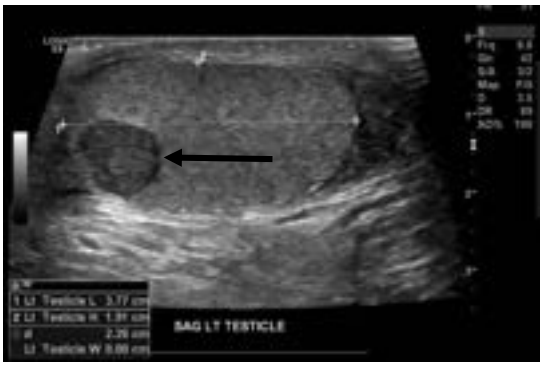
1. **AFP, Alpha-fetoprotein**: elevated to varying degrees in many NSGCTs ,but never found in pure seminomas or pure choriocarcinoma
2. **hCG: Human chorionic gonadotropin** : It is elevated in NSGCTs, and in 10% of seminomas.

3. **LDH. Lactic acid dehydrogenase (LDH):**Its elevation correlates with tumor burden

Assessment of the tumor markers helps in diagnosis, staging, management & follow-up.

Imaging investigations

- Scrotal ultrasonography. : It is important to confirm that the mass is truly intra-testicular especially in the presence of a hydrocele.
- Chest radiographs (posterior-anterior and lateral views)
- computed tomography (CT scan) of the abdomen and pelvis



Staging

T:-tumor

- Tis carcinoma in situ
- T 1 limited to testis
- T2 extend beyond tunica albuginea
- T3 invade cord
- T4 invade the scrotum

N: Regional lymph node

- **N0 no lymph nod**
- **N1 node mass less than 2cm**
- **N2 2-5cm**
- **N3 mass > 5cm**

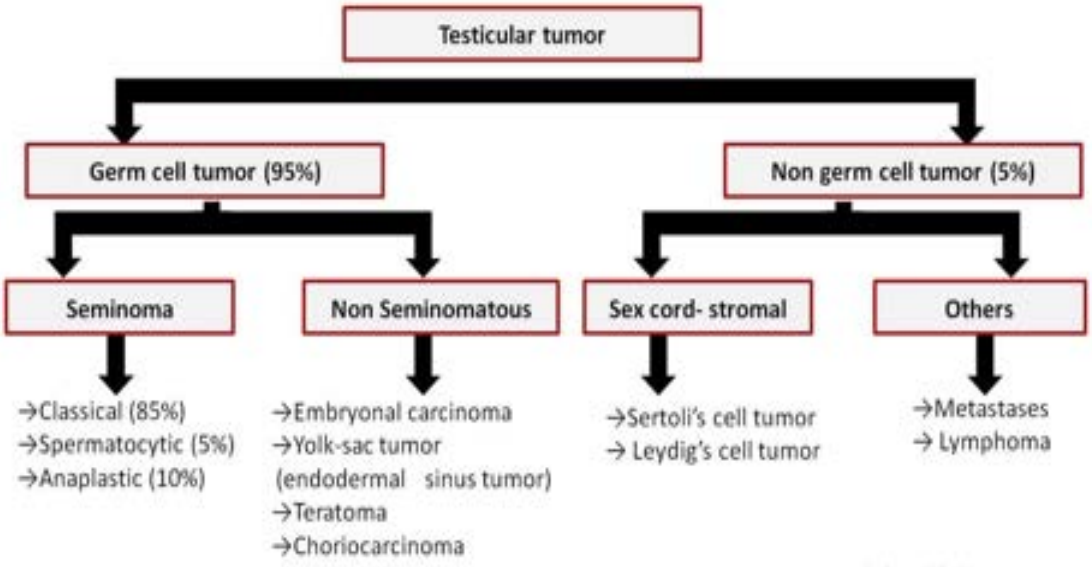
M: metastasis

- **M0 No Distant metastasis**
- **M1 Distant metastasis is present**

TESTICULAR TUMORS

S: Serum tumor markers

- S0 LDH , AFP and hCG within normal limits
- S1 LDH <1.5 and hCG < 5000 mIU/ ml and AFP < 1000ng/ml
- S2 LDH 1.5 -10 OR hCG 5000 -50 000mIU/ ml and AFP 1000 -10 000 ng/ml
- S3 LDH > 10 and hCG >50 000 mIU/ ml and AFP > 10 000ng/ml



Treatment

There is usually a delay time of 3-6 months from initial recognition of the lesion till definitive diagnosis and therapy

I- Radical **orchiectomy** : It should be performed through an inguinal approach with early cross-clamping of the spermatic cord vasculature before delivery of the testis into the field. Scrotal approaches and open testicular biopsies ARE NOT ALLOWD

II- **Adjuvant therapy** : according to tumor histopathology and stage

1- Low-Stage Seminoma: managed with retroperitoneal irradiation (usually 2500–3000 cGy). Chemotherapy should be used as salvage therapy for patients who relapse following irradiation. (95% cure rate)

2- High-Stage Seminoma: managed with primary chemotherapy (cisplatin, etoposide, and bleomycin (PEB), 3 cycles of PEB.

3- Low-Stage Non-seminomatous Germ Cell Tumors are managed with either surveillance (follow-up) or by Retro-peritopneal L.N. Dissection (RPNLD)

TESTICULAR TUMORS

A) Surveillance with follow up using lab. & imaging investigations. It is indicated if the tumor is confined within the tunica albuginea with no evidence of metastasis.

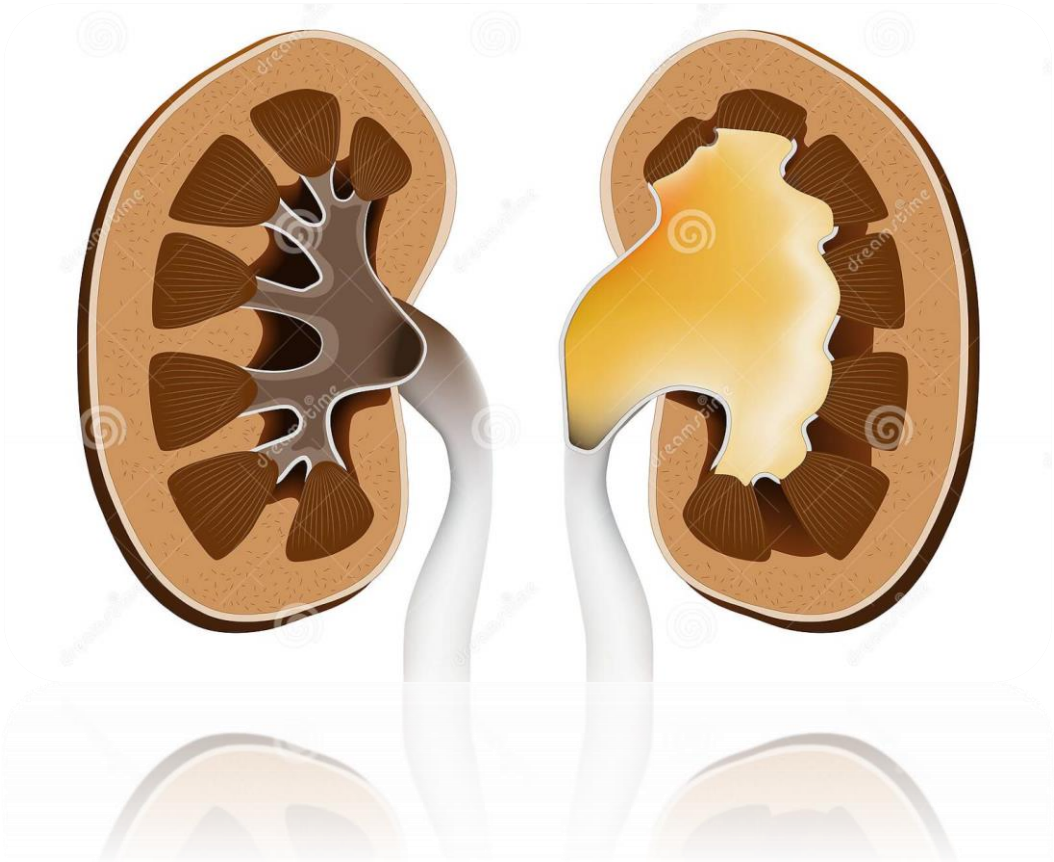
B) RPLND: is indicated in patients not fulfilling the criteria of surveillance

a. To remove all nodal tissue between the ureters from the renal vessels to the bifurcation of the common iliac vessels

b. If the removed nodes are positive for malignancy→ chemotherapy

4- High-Stage Non-seminomatous Germ Cell Tumors managed with Chemotherapy (PEB) followed by RPLND for the residual mass

HYDRONEPHROSIS



Hydronephrosis

Definition

Hydronephrosis means dilation of the pelvicalyceal system with progressive renal atrophy due to chronic partial obstruction to the outflow of urine.

Types of obstruction

Sudden	Insidious
Partial	Complete
Unilateral	Bilateral

Obstruction level

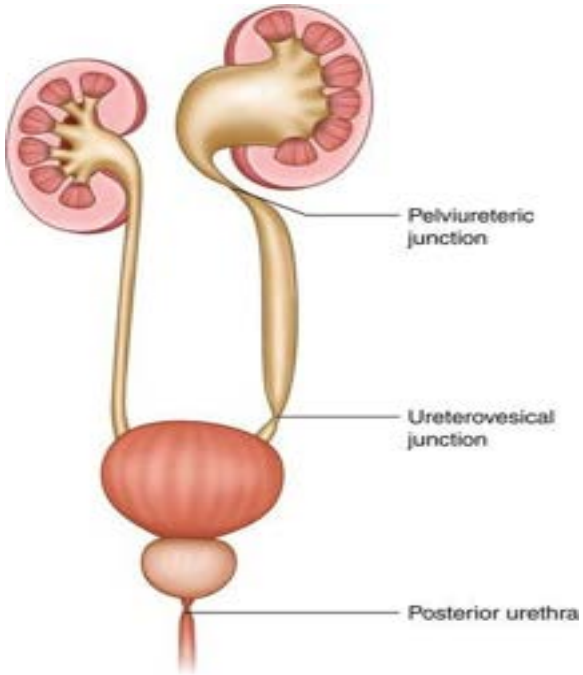
Kidney

Pelvi-ureteric junction

Ureter

Bladder

Urethra



Diagnosis

History

Pain is usually present along the flank with radiation toward the ipsilateral groin or lower abdominal quadrant. If onset is sudden and severe, ureteral stone may be the cause.

If pain is induced by diuresis (e.g., following consumption of alcohol), consider uretero-pelvic junction obstruction ("Dietl crisis").

When obstruction is subacute to chronic, symptoms may absent, and may wax and wane in severity (colic)

Extrinsic compression (e.g., malignancy-associated compression of ureters or retroperitoneal fibrosis) usually has a more insidious onset compared with intrinsic obstruction (e.g., ureteral stone or blood clot).

Hydronephrosis

Nausea and vomiting are typically associated with acute obstruction, usually from an intrinsic cause.

Oliguria/anuria may occur with complete, bilateral obstruction, or with an obstructed solitary kidney.

Lower Urinary tract symptoms (LUTS) dysuria, urinary urgency and frequency, pelvic pressure, and discomfort. are often absent, unless there is an associated condition, including urinary tract infection, distal ureteral stone, or urinary retention.

Hematuria may indicate a stone, urinary tract infection, or malignancy.

The site of obstruction can relate to the presentation:-

With upper tract obstruction frequently presenting with flank pain,

Lower tract obstruction is often associated with obstructive voiding symptoms.

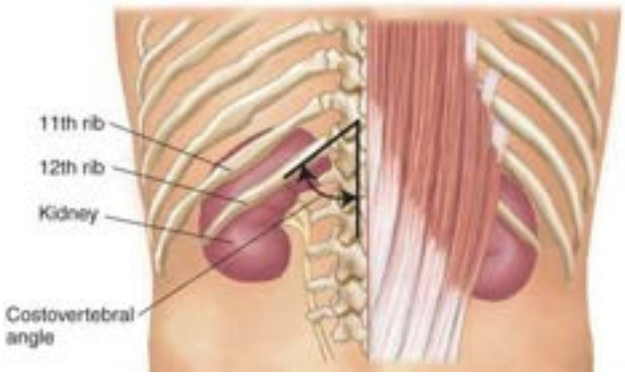
Physical Examination

1- A complete physical examination is mandatory, but may not be helpful in the assessment of hydronephrosis. Special attention should be paid to the following:

Palpable abdominal mass:

Costovertebral angle tenderness is typically not a helpful finding.

An enlarged, often percussible bladder is the most common lower abdominal "mass."

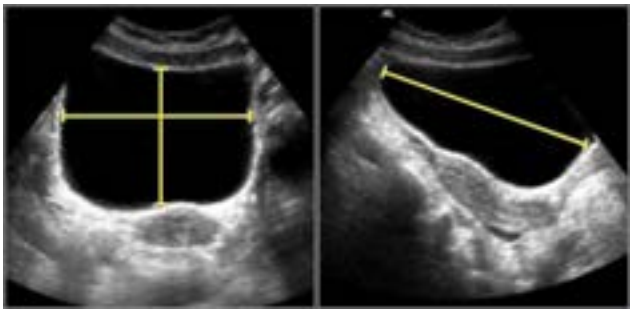


2- Complete genitourinary and pelvic exams:

1. Pelvic exam will assess for pelvic masses or pelvic organ prolapse, which is rarely associated with ureteral obstruction.

2. Digital rectal exam will assess for prostatic abnormality or rectal mass.

3. Residual urine volume due to incomplete emptying of the bladder. Residual urine volume can be evaluated indirectly by bedside ultrasonography or directly by bladder catheterization.



Etiology

Hydronephrosis can be caused by extrinsic or intrinsic factors relative to the urinary tract (Table 1). Causes may be grouped as **congenital or acquired**.

Causes of Hydronephrosis

TABLE 1

Obstructive Intrinsic to the Urinary Tract

Ureter:

Uretero-pelvic junction obstruction,

Uretero-vesical junction obstruction,

stricture, tumor, ureterocele, stone, blood clot, sloughed papilla, infection, hyperplastic polyp

Bladder:

Malignancy, stone, bladder neck obstruction, urine retention

Prostate: Benign prostatic enlargement, prostatitis, calculus, abscess, malignancy

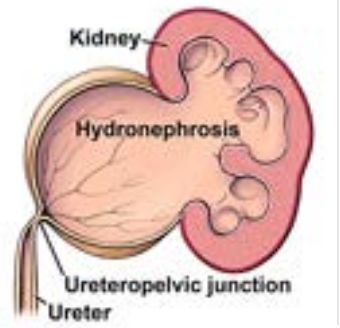
Urethra:

Stricture, stone, diverticulum, malignancy, posterior urethral valves, phimosis

Obstructive Extrinsic to the Urinary Tract

Reproductive system:

- **Uterus:** Pregnancy, prolapse, fibroids, malignancy
- **Ovary:** Malignancy, cyst, abscess



Hydronephrosis

Vascular system:

- **Aneurysm**: Abdominal aorta, iliac vessel
- **Aberrant vessels**: Ureteropelvic junction
- **Venous**: Retrocaval ureter, ovarian vein syndrome

Gastrointestinal system:

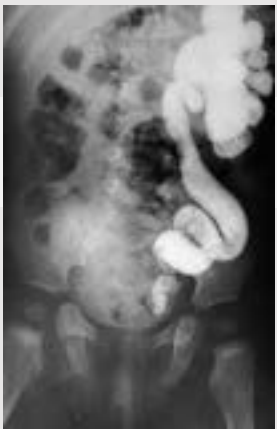
- Inflammatory bowel disease, GI malignancy, abscesses, cysts

Diseases of the retroperitoneum:

- Retroperitoneal fibrosis
- Retroperitoneal malignancy (primary or metastatic deposits)
- Hematoma
- Lymphocele
- Iatrogenic injury

Non-obstructive

Vesico-ureteral reflux
Extra-renal-pelvis
Mega ureter/megacalycosis
Pyelonephritis



Differential Diagnosis

1. Extrarenal pelvis: No true obstruction exists
2. Urinary stones

3. Neoplastic disorders: Kidney, ureter, bladder, urethra, gynecologic, colorectal, metastatic
4. Prostatic hyperplasia
5. Urethral stricture
6. Neurologic disease that produces voiding dysfunction
7. Urinary reflux
8. Urinary tract infection
9. Medication effects
10. Trauma
11. Congenital abnormality of urinary tract
12. Urinary retention
13. Retroperitoneal fibrosis
14. Urinary trauma
15. Iatrogenic injuries

Laboratory Tests

Evaluation of kidney function:

Blood urea nitrogen and creatinine. If azotemia present, bilateral obstruction or unilateral obstruction of a solitary kidney is present.

Electrolyte abnormalities:

Hypo- or hypernatremia, hyperkalemia, and low bicarbonate concentration.

Urinalysis and sediment examination:

White blood cells, red blood cells, or bacteria in the appropriate setting (e.g., infection, stones). Urine microscopy facilitates crystal identification, which may suggest urinary stone disease. The sediment may be normal in obstructive renal disease.

Urine culture:

Urinalysis or presentation suggests urinary tract infection.

Hydronephrosis

Urine cytology:

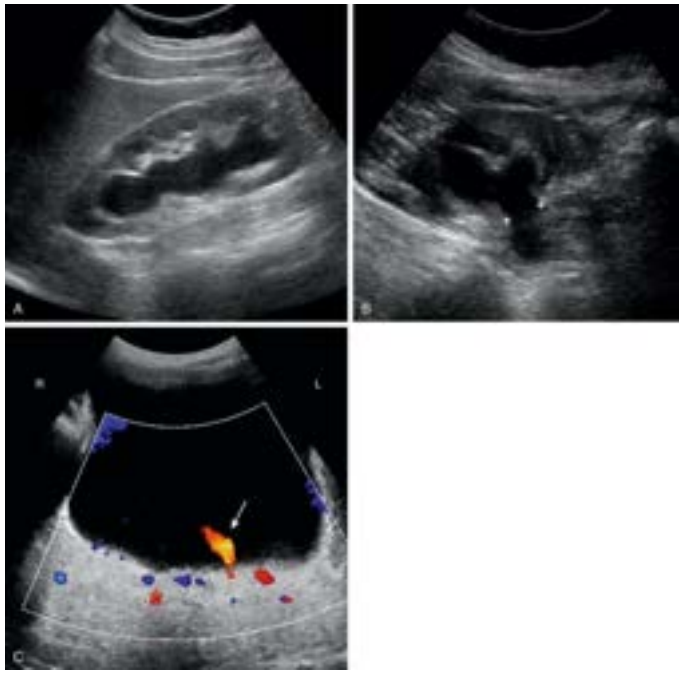
Suspicion for urothelial cancer.

Imaging Studies

Ultrasound

Is an excellent initial screening test, especially for children and pregnant women, with evaluation of the kidneys, portions of the ureters, bladder wall, bladder volume, and contour of the collecting system and ureters

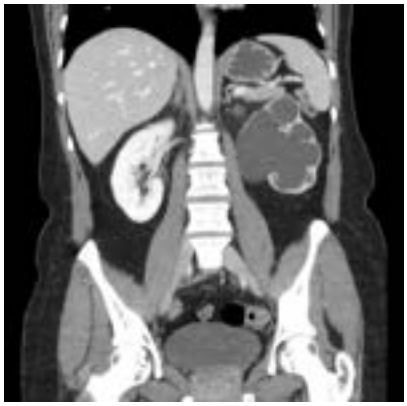
Ultrasound is >90% sensitive and specific for hydronephrosis.



Abdominal, plain film, or kidney, ureter, and bladder (KUB)

Hydronephrosis

Radiographs have limited diagnostic value unless conducted with ultrasound; may demonstrate radiopaque kidney or ureteral stones.

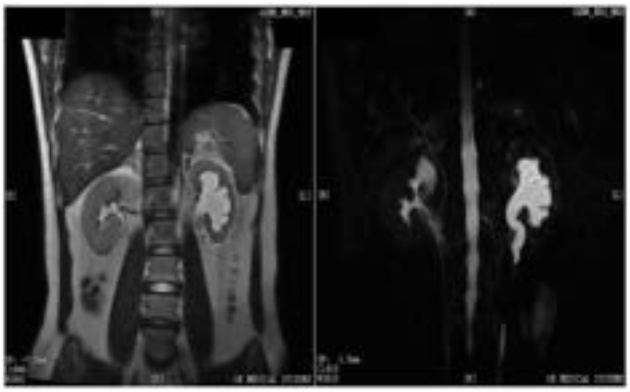


Abdominal CT scan

Without intravenous contrast medium localizes sites of obstruction

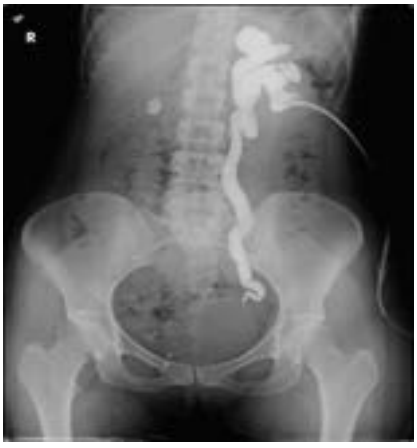
A normal ureteral width by unenhanced CT is 2 to 3 mm wide in adults. If kidney function is normal, CT urography (without and then with contrast, and with delayed images of the ureters), provides anatomic information and is the modality of choice for assessment of upper tract tumors or incidental hydronephrosis.

MRI



Is an alternative to CT that provides detail but cannot directly detect a stone. Severely impaired renal function may preclude gadolinium administration. MRI may be used when other tests are inconclusive or contraindicated (e.g., pregnancy, CKD, radiocontrast media allergy).

Ante-grade or retrograde ureterogram/pyelogram



Is an invasive procedure used when CT or MRI scans with radiocontrast media are contraindicated (e.g., contrast allergy, renal impairment).

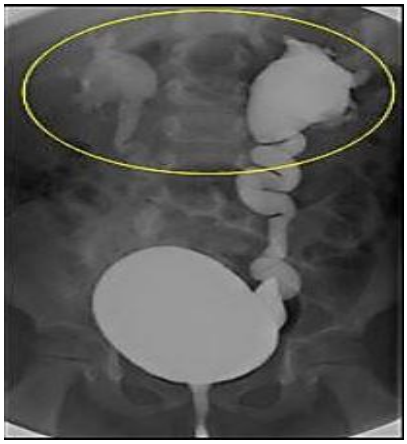
Voiding cystourethrography (VCUG)

Hydronephrosis

Establishes vesico-ureteral reflux and bladder neck or urethral obstruction.

Radioisotope renography (diuretic renography, Tc-99m MAG3 renogram)

Is a functional procedure that provides differential renal function and determines presence of functional obstruction.



Lines of treatment of hydronephrosis:

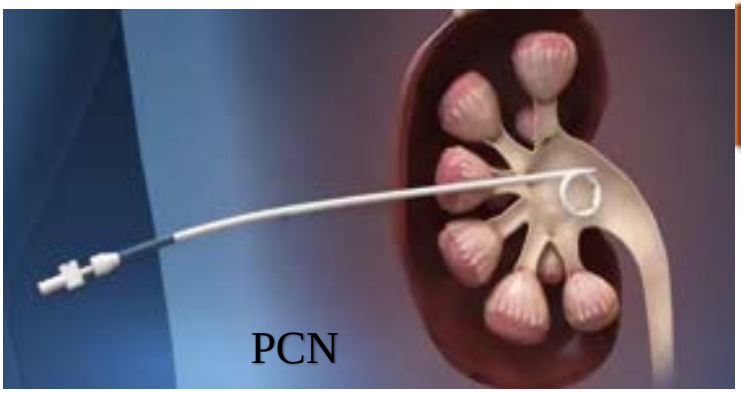
1. Urgent drainage of urine:

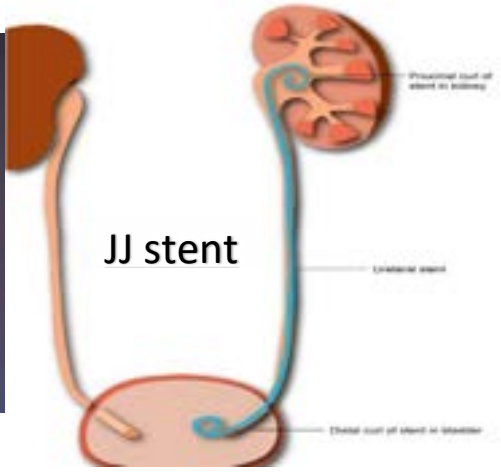
Using a temporary maneuver to overcome critical situations of urosepsis or uremia.

- Indications: - Infected hydronephrosis

  - Decompensated renal function with bilateral hydronephrosis or hydronephrotic solitary kidney.

- Methods: - PCN or JJ stent in cases of upper tract obstruction. - Indwelling urethral catheter in cases of VUR.





2. Treatment of the cause:

When the hydronephrotic kidney is still functioning and the patient is not decompensated.

3. Nephrectomy:

For unilateral hydronephrosis with irreversibly lost function in the presence of a normal contralateral kidney.

4. Renal replacement therapy:

- Indication: when the renal function is irreversibly lost and cannot be improved in a decompensated patient.

- Methods: Dialysis or renal transplantation.

Treatment of ureteral stricture

- **Endoscopic treatment**

For passable stricture by dilatation or endoureterotomy followed by ureteral stenting.

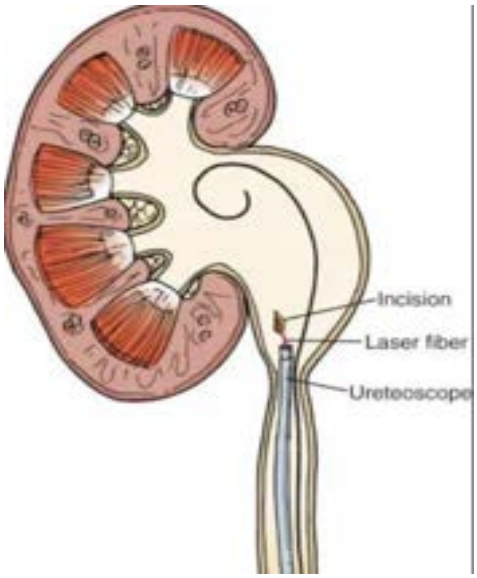
- **Open surgery**

For non-passable stricture and after failure of endoscopy:

Lower third ureter: ureteroneocystostomy (ureteral reimplantation) or Boari's bladder flap.

Middle and upper third ureter: Resection reanastomosis for short stricture and ileal loop Replacement of the ureter for long stricture.

Ureterpelvic junction obstruction: pyeloplasty.



Endoureterotom

Treatment of VUR:

- Conservative measures as frequent bladder voiding and prophylactic low dose antibiotics in Cases without recurrent UTI.

- Cystoscopic injection of a biocompatible bulking agent to produce ureteral orifice coaptation in cases with primary VUR.

- Treatment of the cause of 2ry VUR e.g. bladder outlet obstruction.

- Ureteroneocystostomy with antireflux measure in complicated cases.

Lower Urinary Tract Dysfunctions

Urinary incontinence

Definition: Involuntary leakage of urine.

Classification:

- **Stress urinary incontinence (SUI):**

Involuntary urinary leakage on effort, exertion, sneezing, or coughing. It is due to hypermobility of the bladder base or pelvic floor and/or intrinsic urethral sphincter deficiency. When confirmed on urodynamic testing, it is termed urodynamic stress incontinence.

- **Urgency urinary incontinence (UUI):**

Involuntary urine leakage accompanied or immediately preceded by urgency (a sudden, strong desire to void).

It is due to an overactive detrusor muscle. The urodynamic diagnosis is termed ‘detrusor overactivity incontinence’. It isa component of the OAB syndrome

- **Mixed urinary incontinence (MUI):**

Involuntary leakage associated with urgency and also with exertion, effort, sneezing, or coughing.

It contains symptoms of both SUI and UUI.

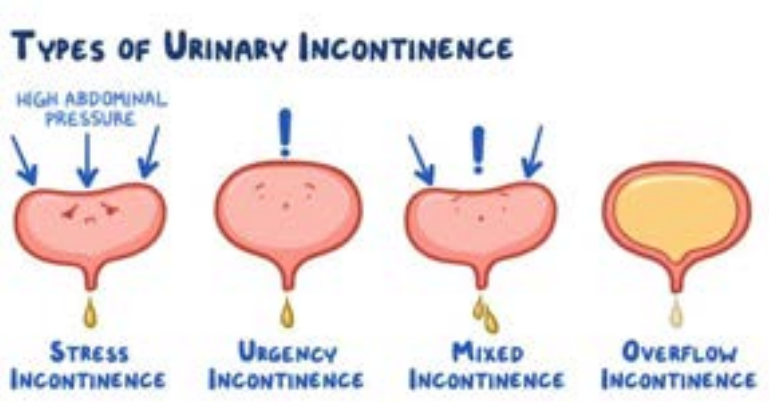
- **Overflow incontinence:**

Leakage of urine when the bladder is abnormally distended with large residual volumes.

Nocturnal enuresis: Loss of urine occurring during sleep.

Nocturnal enuresis can be further classified into primary types (never been dry for longer than a 6-month period) or secondary (there-emergence of bedwetting after a period of being dry for at least6–12 months

- **Continuous incontinence:** Continuous involuntary loss of urine. This is experienced with a vesicovaginal fistula.



Risk Factors of UI

Caucasian > Afro-Caribbean
Genetic predisposition.
Neurological disorders
Anatomical disorders e.g., vesico-vaginal fistula
Childbirth trauma and pregnancy
Pelvic, perineal, and prostate surgeries leading to pelvic muscle and nerve injury, pelvic radiotherapy.
Smoking
Obesity
Ageing
Oestrogen deficiency

Pathogenesis of UI

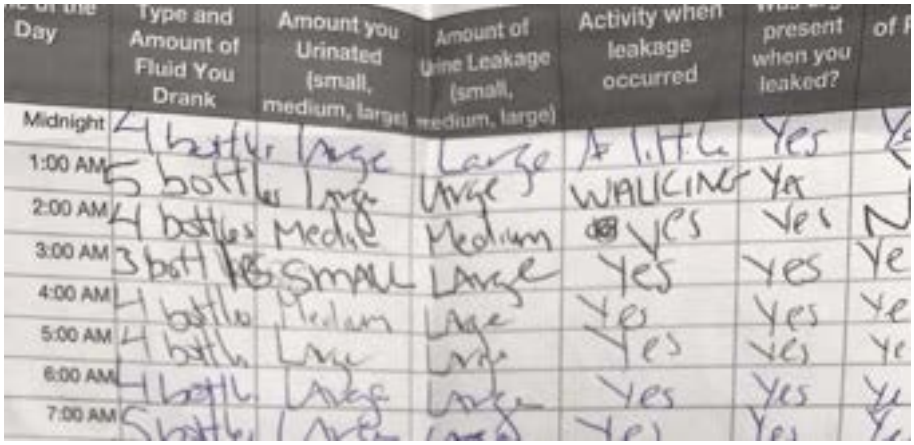
Urgency Urinary Incontinence: Secondary to detrusor overactivity (DO) or involuntary bladder muscle contractions during the storage phase of the micturition cycle. DO It could be idiopathic or neurogenic in origin and can be associated with symptoms of increased urinary frequency, urgency, nocturia or urgency incontinence. These symptoms are known collectively as overactive bladder syndrome (OAB). The pathogenesis could be due to detrusor muscle denervation or disruption of neural control in detrusor muscle cells.

Stress Urinary Incontinence occurs due to either urethral hypermobility or Intrinsic sphincter deficiency.

Clinical Presentation of UI

1-Patient presents with **history** of storage or voiding symptoms,
2-You should ask about the **triggers** for incontinence e.g., coughing or sneezing or if it was preceded by sensation of urgency.
3-You should look for possible **precipitating** factors such as abdominal or pelvic surgery or irradiation, or neurological deficit disorders, or childbirth trauma or pelvic organ prolapse (POP).
4-Important part of the assessment is to use a validated **questionnaire** the objectify the severity patients symptoms and impact on their quality of life such as International Consultation on Incontinence Questionnaire on UI (ICIQ-UI), ICIQ-FLUTS, and ICIQ-MLUTS.
5-Ideally patients should complete a 3-day **voiding diary** to establish the daily amount of fluid intake and urine output, functional bladder capacity, episodes of frequency, urgency, and incontinence.
6-**Abdominal examination** may reveal a palpable bladder, neurological assessment of gait, anal reflex, and perineal sensation.
7-**DRE** should be performed to assess anal tone, prostate cancer, and pelvic malignancy of constipation.

8-**Physical examination in women** should include pelvic examination in the supine and standing and lateral position with a speculum to rule out fistulae, vaginal atrophy, urethral hypermobility, POP. A positive cough test for urinary leakage is pathognomonic for SUI.



Voiding diary

Investigations

- In selected cases, **ultrasound scan** and renal blood function tests e.g., in patients with suspected high pressure chronic retention with overflow incontinence.
- **Pad test**: measures the amount of urinary leakage in 1 hr or in 24 hrs
- **Flowmetry**: Determines the urinary flow rate, time to void, and postvoid residual urine
- **Urodynamics:** Filling cytometry helps establish the diagnosis of DO, low detrusor compliance, reduced bladder maximum capacity and SUI. Video-urodynamics uses contrast and fluoroscopy can visualize movement of the bladder neck and vesico-ureteric reflux (VUR) in neurogenic patients.

Treatment of UI

A. Treatment of SUI

- ***Conservative treatment:***

1- Pelvic floor muscle training (PFMT) with or without biofeedback: for at least 3 months, a patient performing eight contractions 3-times a day. IT improve symptoms in 30% of women with mild SUI.

2- Lifestyle modification: advice patients to stop smoking, avoid constipation, and lose weight if obese.

3- Electrical stimulation e.g. stimulation of the pudendal nerve.

- ***Surgical Treatment of SUI***

1- Urethral bulking agents

Helpful in cases with mild to moderate SUI, or if patients doesn't want or is not fit for more invasive surgical intervention. Under anaesthesia, a transurethral injection of bulking agents such as macroplastique or Bulkamid is done under cystoscopy

guidance to achieve urethral muscosal apposition and closure of the lumen with up to 50% success rate.

2- Colposuspension

An open or laparoscopic surgical procedure of suspension of the bladder neck through a Pfannenstiel incision exposing the paravaginal fascia (either side of the bladder neck) and approximating it to the iliopectineal ligament of the superior pubic rami, using non-absorbable sutures. It carries a durable success rate up to 70% after 10 years of followup. Complications include de novo vaginal prolapse, de novo urgency, voiding difficulty and UTIs.

An illustration of Colposuspension procedure

3- Mid-urethral slings

Synthetic mid-urethral slings are used in women with SUI due to urethral hypermobility or ISD. These are soft polypropylene mesh. They can be performed as a day case. However, the patients must be counselled on risk of mesh related complications including chronic pelvic pain and mesh erosion. Procedure can be done either retro pubic or transo-bturator with success rate up to 85% on long term follow-up.

An illustration of Different Types of midurethral slings and positioning of the tape sub-urethral.

4- Artificial urinary sphincters (AUS)

The AUS is a closed pressurized system of inflatable cuff placed around the bladder neck, connected to a pressure-regulating balloon that is placed extra peritoneally in the abdomen with an activating pump placed in the scrotum in men or in labia majora in women although more commonly used in men with post-prostatectomy incontinence. It is indicated in patients with severe SUI with success rates >90%. But it is associate with high rates of revision due to urethral atrophy and loss of effect or mechanical failure of its components.

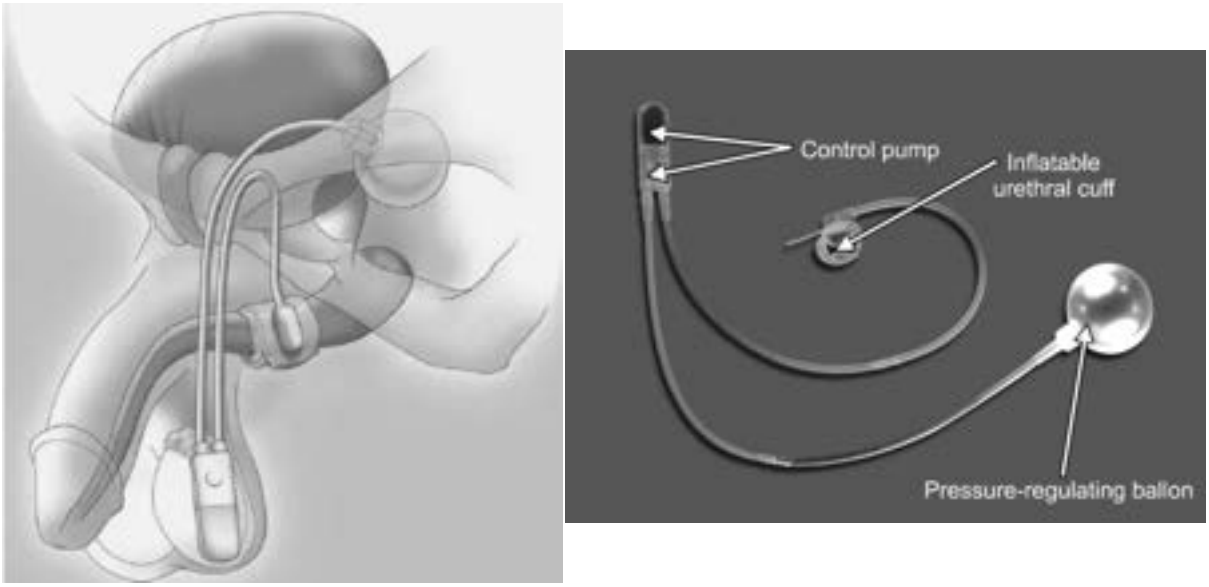


Illustration of AUS implanted in men

B. Treatment of Overactive bladder syndrome (OAB) and Urgency urinary incontinence (UUI)

Antimuscarinics

Acetylcholine acts on muscarinic receptors such as M3 and M2 subtypes on the bladder detrusor muscle to cause involuntary contractions and provoke the symptoms of bladder overactivity. These receptors are the targets of antimuscarinic drugs which inhibit contractions and increase bladder capacity. Side effects include dry mouth and constipation and cognitive disorders and should be avoided in patients with narrow angle glaucoma.

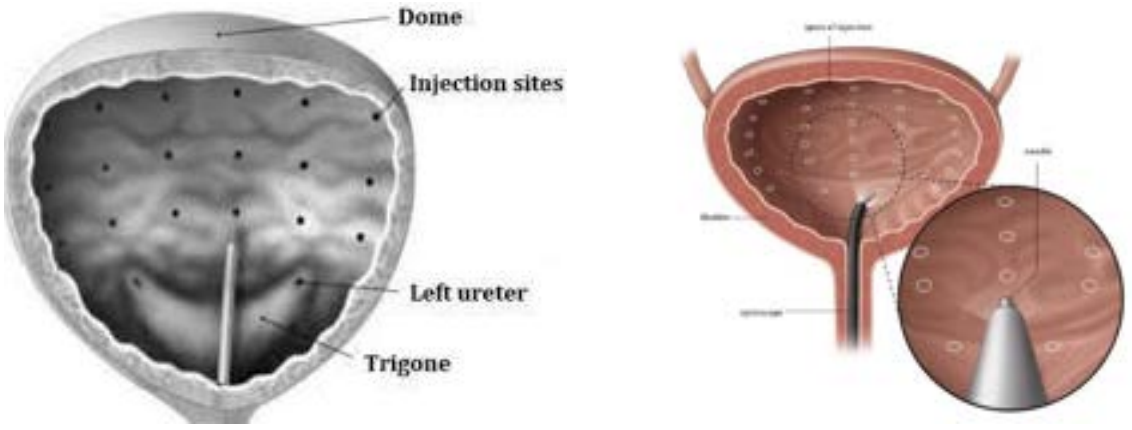
Beta-3 Agonists

They causes smooth muscle relaxation by reducing muscle sensitivity to calcium and can improve bladder storage without affecting contraction of the bladder. They are contraindicated in patients with uncontrolled hypertension.

Botulinum toxin-A

Acts by inhibiting the release of acetylcholine and from presynaptic cholinergic nerve terminals, resulting in regionally decreased muscle contractility and muscle atrophy at the site of injection. It is injected into bladder wall under cystoscopy guidance with success rates up

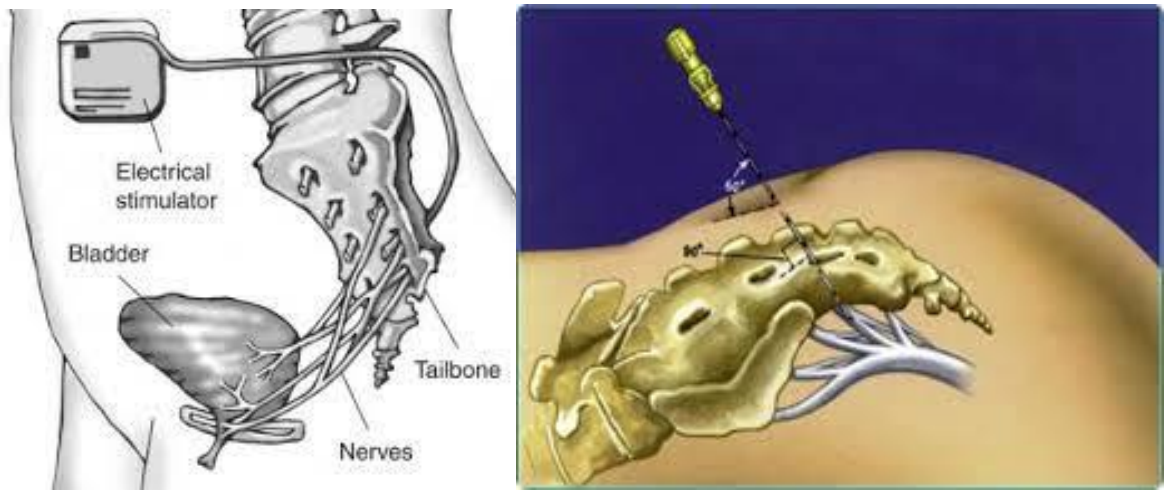
to 50% in control of OAB symptoms but it loses its effect in 6 to 9m months and therefore repeat injection is required. Patients might develop transient urinary retention in the first few days/weeks after injection.



An illustration of intravesical Botulinum toxin-A injection

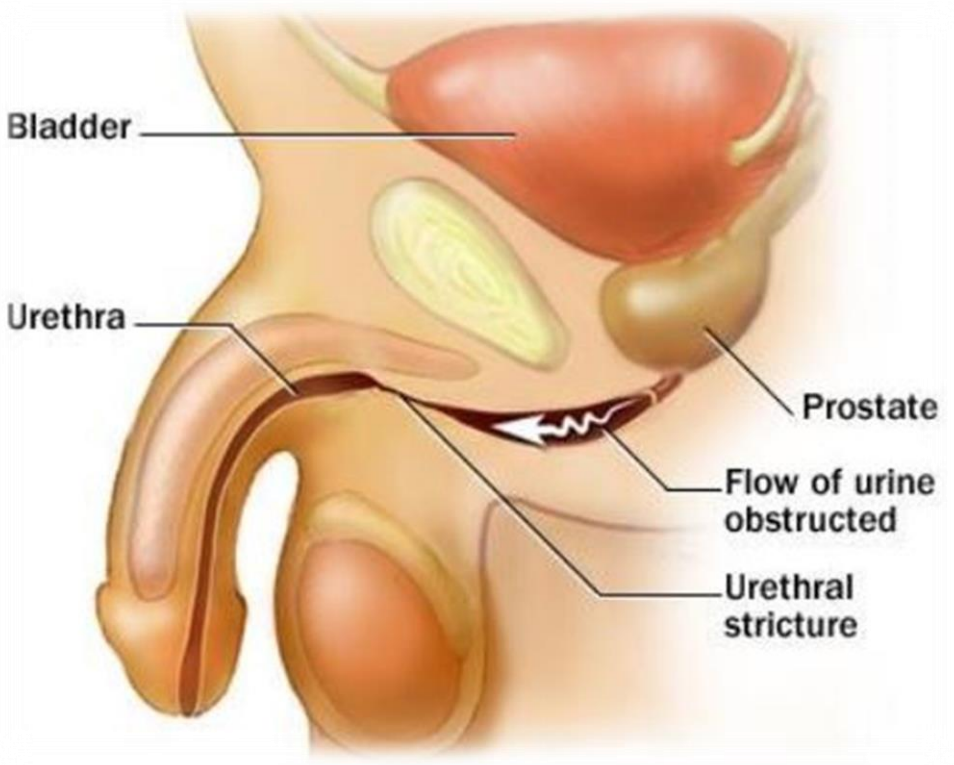
Sacral nerve stimulation (SNS)

It involves electrical stimulation of the sacral nerves (mainly S3) supplying the urinary bladder to suppress reflexes responsible for DO. An initial percutaneous nerve evaluation is performed, followed by surgical implantation of a permanent electrode leads into the sacral foramen with success rate up to 65%



An illustration of SNS

URETHRAL STRICTURE

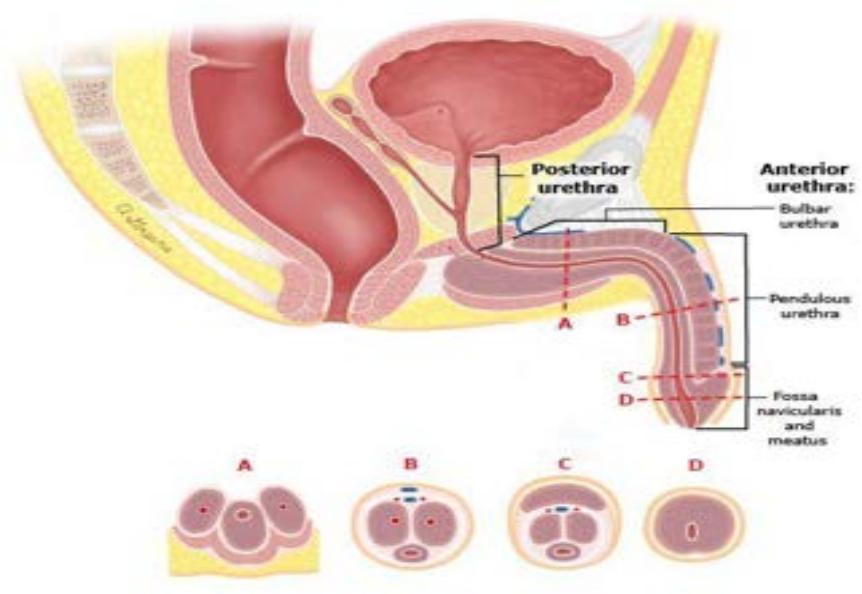


Urethral Stricture

Definition

Urethral stricture is abnormal narrowing of any segment of the urethra surrounded by corpus spongiosum, and specifically implies varying degrees of spongiofibrosis.

The term “spongiofibrosis” refers to scarring of the corpus spongiosum of varying degrees.



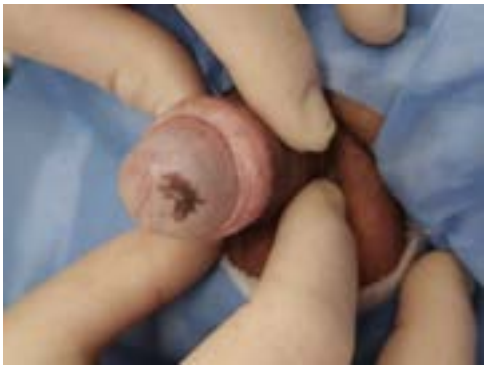
Etiology of Urethral Stricture Disease

1- Iatrogenic, Urethral Instrumentation as catherization or cystoscopy or TURP

2- Trauma urethral injury can result from external trauma that is either blunt or penetrating injuries

3- Inflammatory

Lichen sclerosis (LS) is a progressive sclerosing process, which can involve the penile shaft skin, glans, meatus, or anterior urethra. It is currently the most common inflammatory cause of glandular urethral strictures and acquired meatal strictures



4- Post-infectious

Recurrent gonococcal urethritis once accounted for the majority of anterior urethral strictures.

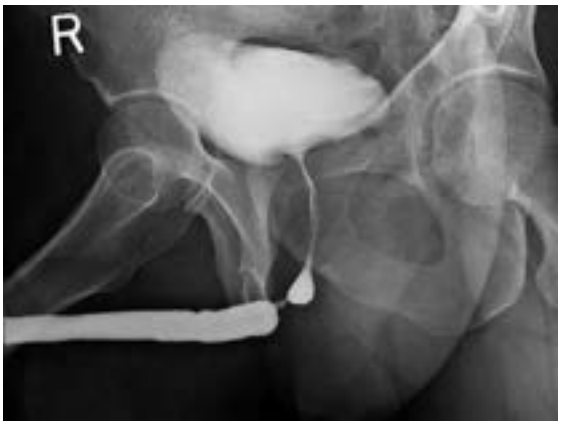
5- Congenital

Congenital urethral strictures are the least common subcategory. It is a diagnosis that can be reasonably made only in the absence of inflammation, trauma, infection, and urethral manipulation.

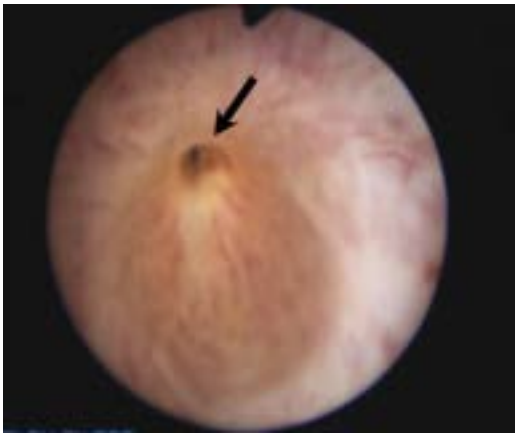
6- Idiopathic

Diagnosis:

- **History**: urethral discharge, trauma or instrumentation. -Obstructive LUTS.
- **Examination** sometimes detects induration at the site of stricture.
- **Abdominal U/S** may detect significant post-voiding residue.
- **Uroflowmetry**: This is a non-invasive test to assess the degree of obstruction and improvement after treatment. It illustrates abnormal voiding pattern and reveals low flow rate.
- **Retrograde urethrography** is the main diagnostic method to delineate the stricture.



- **Urethroscopy**: is used for both diagnosis and endoscopic treatment.



Differential diagnosis

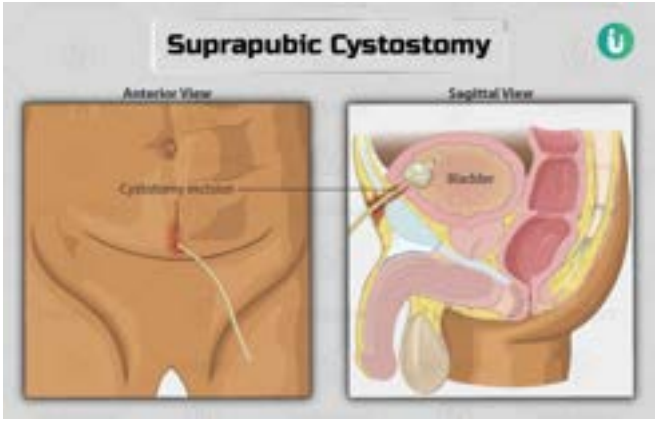
-Other causes of bladder outlet obstruction as BPH.

-Neuropathic bladder disorders which may require neurologic and urodynamic assessment.

Treatment

Depends upon many variables

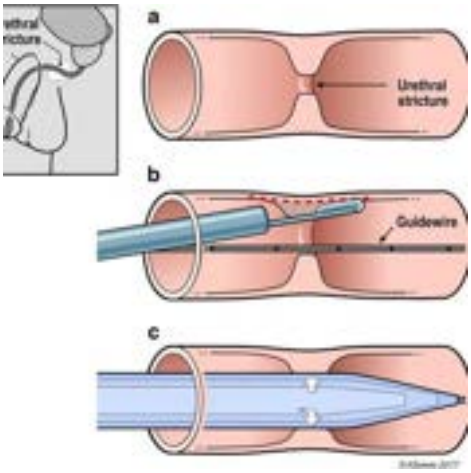
1. **Urgent percutaneous suprapubic cystostomy** insertion for temporary urinary diversion



In cases complicated with

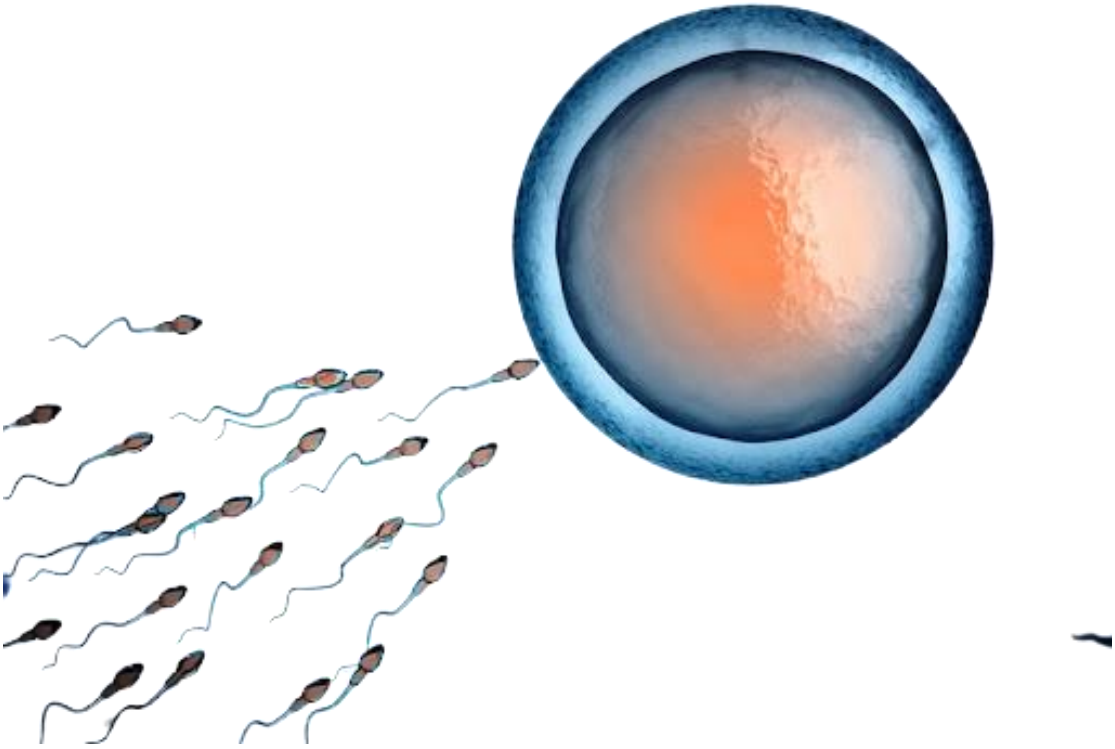
Urine retention, renal impairment due to upper tract dilatation, acute epididymo-orchitis, periurethral suppuration or urethral fistula.

2. **Visual internal urethrotomy (VIU)** for passable short uncomplicated anterior urethral stricture by endoscopic incision of the stricture.



3. **Urethroplasty** for impassable or complicated strictures and failed VIU. It implies excision of the strictured segment and bridging the defect by direct re-anastomosis, local skin flap or buccal mucosal graft.

URO-ANDROLOGY



Erectile dysfunction (ED)

Definition

Erectile Dysfunction is defined as the persistent inability to attain and maintain an erection sufficient to permit satisfactory sexual performance.

Physiology of penile erection:

Upon **visual or auditory stimulation, para-sympathetic discharge** causes the release of Nitric Oxide (NO), which decrease intracellular Ca. Subsequently smooth muscle and vascular wall relaxation which increase the blood flow to the penis and filling of sinusoidal spaces with blood. This compresses the sub-tunical and emissary veins giving rigid erection.

After ejaculation, sympathetic discharge increases and subsequently smooth muscle and arterial constriction causing decrease the blood flow to the penis and the blood is expelled from the sinusoidal spaces.

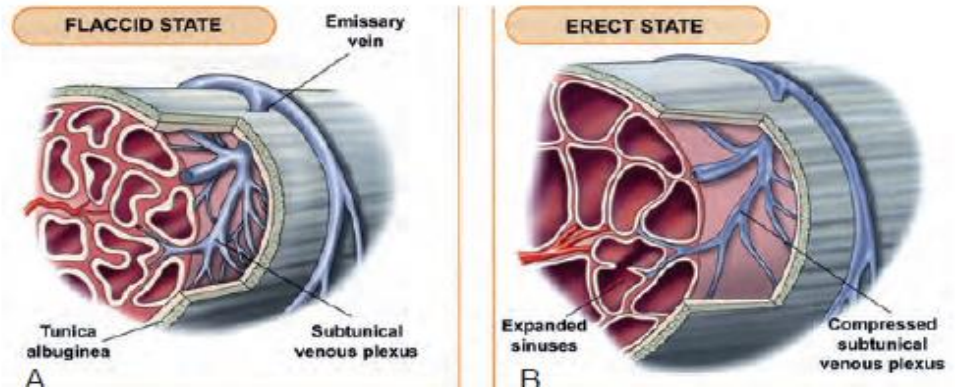


Figure (1) Physiology of penile erection

Risk factors of ED:

Metabolic syndrome (Obesity, hypertension, hyperlipidemia, Diabetes Mellitus, physical inactivity), smoking, antidepressants and antihypertensive drugs.

Causes:

Psychogenic

Organic:

- Vasculogenic: arterial insufficiency or venous leakage due to cardiovascular diseases and diabetes.
- Neurogenic: Cerebrovascular stroke and spinal cord disorders.
- Hormonal: Hypogonadism, hyperprolactinemia, thyroid disorders.
- Post- surgical: Radical cystectomy and prostatectomy.

- Structural: Penile curvatures, penile fractures and penile fibrosis post priapism.
- Drugs: Anti androgens, antidepressants and some antihypertensive.

Evaluation:

History:

Onset and course: Sudden onset and intermittent or situational = psychogenic, while gradual onset and progressive course is associated with organic causes.

Morning erection is absent in most organic causes.

Smoking, history of LUTS, history suggestive of cardiac diseases.

Examination:

Local genital examination, assessment of secondary sexual characters, vascular and neurological examination:

Investigations:

Basic Assessment:

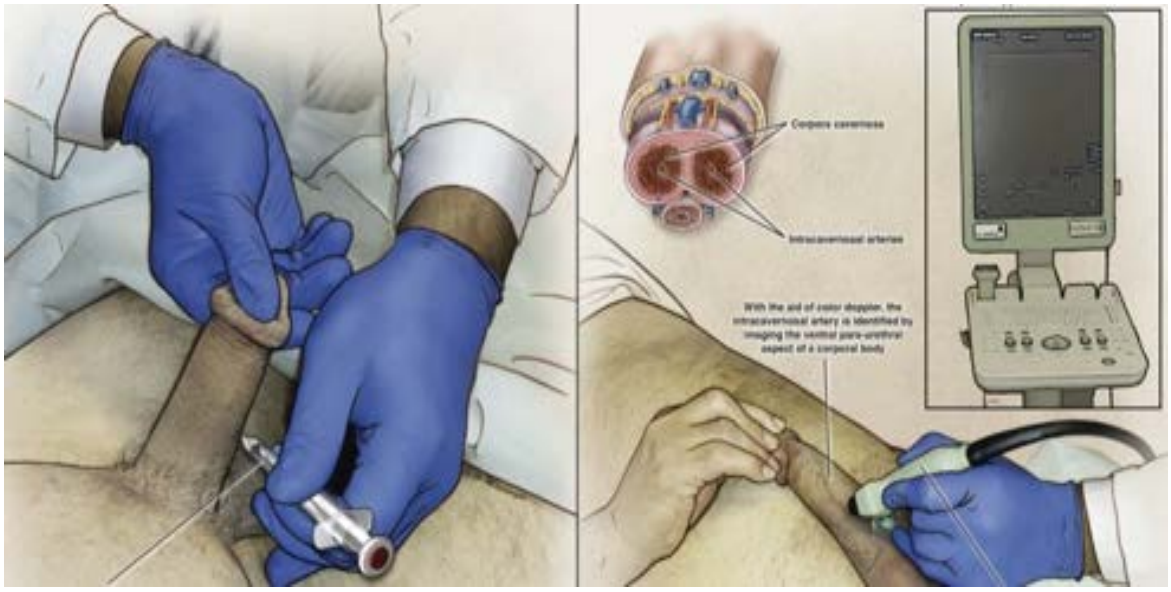
Morning fasting blood glucose, lipo-gram and total testosterone.

Specialized Assessment:

Nocturnal penile tumescence study: differentiate organic from psychogenic ED

Penile Doppler study after injection of vasoactive drugs: Assess the penile arterial flow and the degree of penile venous leak if present.

Hormonal work-up: for cases of hypogonadism and low total testosterone.



Penile Doppler study

Treatment:

Treatment of reversible causes by life style modifications (Weight reduction, stop smoking and exercise), stop or replace the accused drugs

Most **organic causes of ED are not reversible**. This means patients will need lifelong treatment or surgery.

If the organic ED is not reversible, it can be managed by one of the followings:

First line:

A- Oral drugs (Phosphodiesterase inhibitors):

Enhance the NO action by inhibiting the degradation of cyclic GMP. They are not curative. Used on demand.

Parameter	Sildenafil, 100 mg	Tadalafil, 20 mg	Vardenafil, 20 mg	Avanafil, 200mg
C_{max}	560 µg/L	378 µg/L	18.7 µg/L	5.2 µg/L
T_{max} (median)	0.8-1 hours	2 hours	0.9 hours	0.5-0.75 hours
T1/2	2.6-3.7 hours	17.5 hours	3.9 hours	6-17 hours
AUC	1,685 µg.h/L	8,066 µg.h/L	56.8 µg.h/L	11.6 µg.h/L
Protein binding	96%	94%	94%	99%
Bioavailability	41%	NA	15%	8-10%

Contra-indications:

Recent myocardial infarction or stroke

Life threatening arrhythmia

Unstable angina during intercourse

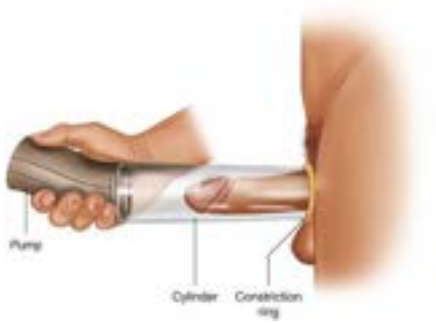
Congestive heart failure

Hypotension <90/50

Hypertension > 170/100

Nitrates use

Antihypertensive or alpha blockers: May cause orthostatic hypotension



B- Vacuum constriction device:

Trap the blood inside the corpora by the vacuum and the constriction placed at the base of the penis. It is used on demand and associated with some side effects as pain, numbness, bruising and inability to ejaculate.

Second line:

If the first line fails to give rigid erection, we can use vasoactive substance injection on demand by

Intra-urethral pellets.

Or

Intra-cavernous injection: efficacy more than 60%

Side effects are penile pain, priapism and penile fibrosis.

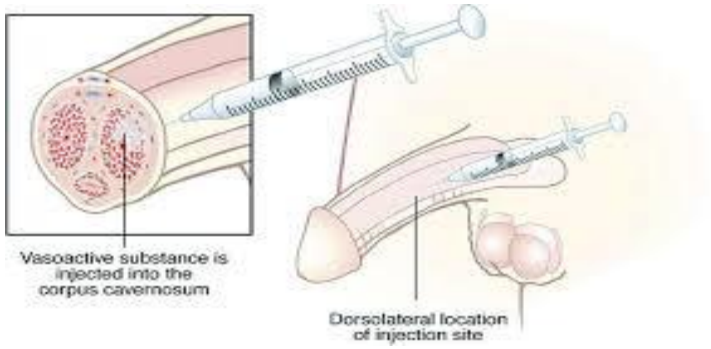


Figure (2) Intra-cavernous self-injection

Implantation of penile prosthesis if all previous treatment failed. Satisfaction rate is more than 90%.

Types:

Semi rigid: simple surgery, less expensive, no need for revisions. But less concealment.

Inflatable: Better concealment, resemble the normal erection. Disadvantages: more complex surgery, expensive, require multiple revisions.

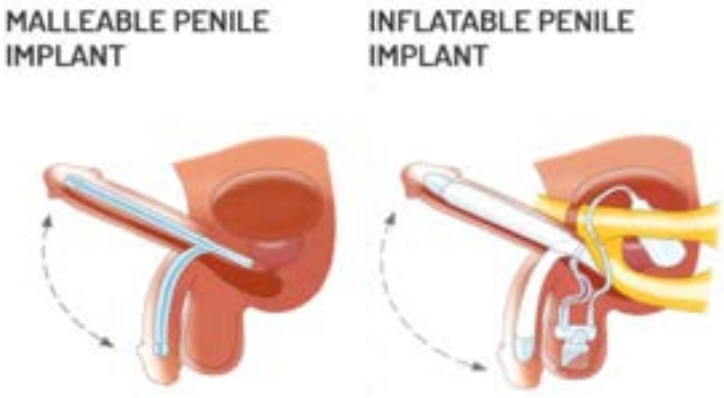


Figure (2): Semi rigid penile prosthesis

Ejaculatory disorders:

Premature ejaculation:

Is **defined** as ejaculation which is always or nearly always occurs before or within one minute of vaginal penetration.

It might be caused by psychological or organic causes (Hyper-excitability of ejaculatory reflex.

Treatment:

Behavioral: start and stop and squeeze techniques.

Pharmacological: Local anesthetics, selective serotonin reuptake inhibitors drugs or combination of both.

Retrograde ejaculation:

Is **defined** as inadequate bladder neck contraction results in propulsion of the sperm back to the bladder during the ejaculation:

Causes:

Surgery to the bladder neck or prostate.

Drugs as alpha blockers.

Neurological causes as in spinal cord injury or peripheral neuropathy.

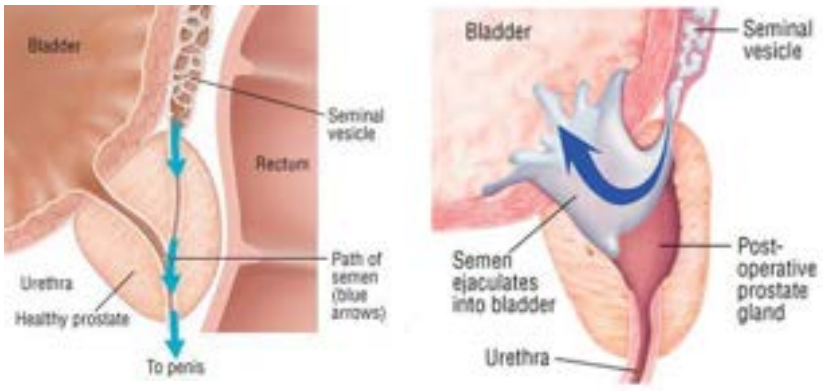
Treatment:

Stop the causative drug.

Alpha agonist drugs daily as pseudo-ephedrine.

Injection of bulking agents into the bladder neck.

Sperm retrieval from the bladder to be used for Assisted Reproductive Techniques (ART) in cases of infertility.



Haemo-spermia:

The presence of blood in the semen. Usually intermittent, benign, self-limiting and no cause is identified.

Causes:

< 40 years, usually inflammatory as prostatitis or urethritis.

> 40 years, Benign enlarged prostate, prostate or bladder malignancy.

Other cause, bleeding tendencies.

Treatment:

Treatment of the cause.

Reassurance in idiopathic cases.

Male subfertility

Failure to conceive after one year of regular unprotected intercourse.

Physiology:

Testis has two functions:

Exocrine function: responsible for the spermatogenesis. This is regulated by pituitary FSH.

Endocrine function: Testosterone production which is responsible for the secondary sexual characters, libido and regulation of spermatogenesis. Testosterone secretion is stimulated by Pituitary LH production.

Hypothalamus secretes Gonadotrophin releasing hormone (GnRh) which stimulate pituitary FSH and LH.

Causes of male subfertility:

I-Pre-testicular causes:

Hypothalamic or pituitary disorders (Decrease FSH and LH or hyperprolactinemia)

Thyroid disorders: Hypo or hyperthyroidism

Adrenal disorders: Hypo or hypoadrenalism.

II- Testicular causes:

Congenital: agenesis, Klinefelter syndrome, Cryptorchidism.

Traumatic: Rupture testes, exposure to heat or radiation, chemotherapy.

Inflammatory: Mumps orchitis, testicular abscess

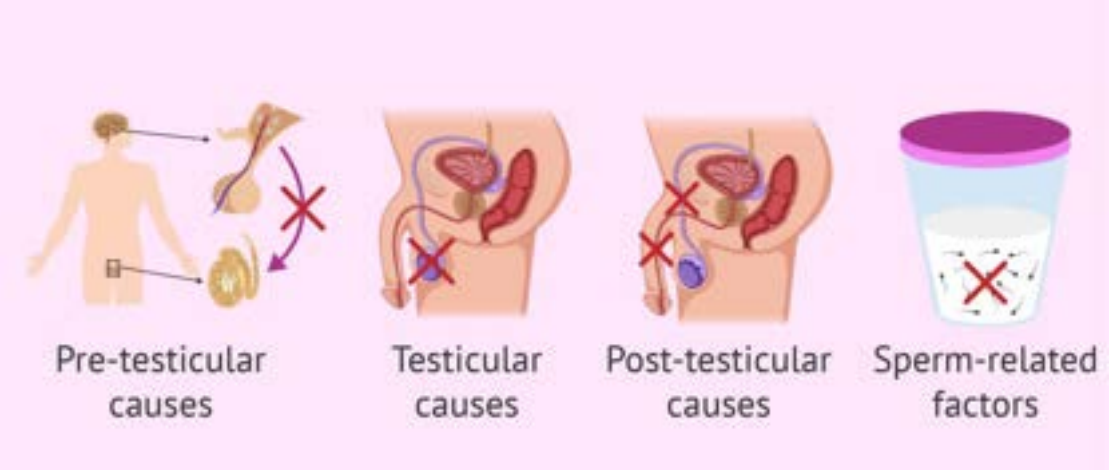
Vascular: Varicoceles (the most common cause), testicular torsion.

III- Post-testicular:

Genital duct obstruction: Ejaculatory duct, epididymal or vasal obstruction (Congenital or post-operative accidental ligation as in hernia operations).

Coital problems: ejaculatory disorders or erectile dysfunction, severe hypospadias or epispadias.

Immunological: Anti-sperm antibodies.



Evaluation:

History:

Primary or secondary, duration of subfertility, wife's age and menstrual irregularities.

Frequency of intercourse or the use of lubricants.

Ejaculatory or erectile dysfunction.

Examination:

General examination: Secondary sexual characters, gynecomastia, obesity, system failure.

Local examination:

Penis: hypospadias, epispadias or chordae.

Testicular size and consistency.

Epididymis and vas: dilated, absent or beaded

Cord: Varicocele.

DRE: Seminal vesicle dilatation or calcification.

Laboratory Investigations:

I-Semen analysis:

Volume > 1.5 ml, Hypospermia is low volume below 1.5 ml

Concentration > 15 million/ml. Oligozoospermia is decreased sperm concentration.

Motility: progressive motility >32%. Asthenozoospermia is decreased sperm motility.

Normal forms > 4%. Teratozoospermia is decreased sperm morphology.

Azoospermia is the absence of sperms in the semen analysis even after centrifugation (causes are the same as causes of male subfertility except the immunological and the coital causes).

II-Hormonal evaluation:

Serum testosterone, FSH, LH and prolactin.

A) Low FSH, LH and testosterone indicate pretesticular causes.

B) Low Testosterone but high FSH indicates testicular causes.

C) Normal hormones indicates obstruction.

Indicated in severe oligospermia, azoospermia

III-Genetic studies:

As Karyotype, Y-chromosome microdeletion. Indicated in severe oligozoospermia and Azoospermia.

Imaging:

Scrotal US and doppler: for evaluation of the testes and assess the degree of varicoceles if present.

TRUS or MRI pelvis: for the diagnosis of the level of genital duct obstruction.

Treatment:

I-Life style modifications:

Stop smoking, practice exercise, reduce weight and eating healthy diet and sometimes vitamin supplementation.

II- Medical treatment:

In patients with hyperprolactinemia or hormonal abnormalities.

III- Surgical Treatment

As varicoceles ligation, Endoscopic or micro-surgical correction of genital duct obstruction.

IV- Assisted reproductive techniques (ART):

In cases of non-obstructive azoospermia not responding to the medical treatment, Micro dissection testicular sperm extraction (MTESE) to be used for ART.

Severe Oligospermia or asthenozoospermia refractory to treatment. Intrauterine insemination (IUI) and Intracytoplasmic sperm injection (ICSI).

Varicoceles

Abnormal dilatation, elongation and tortuosity of the pampiniform plexus of veins.

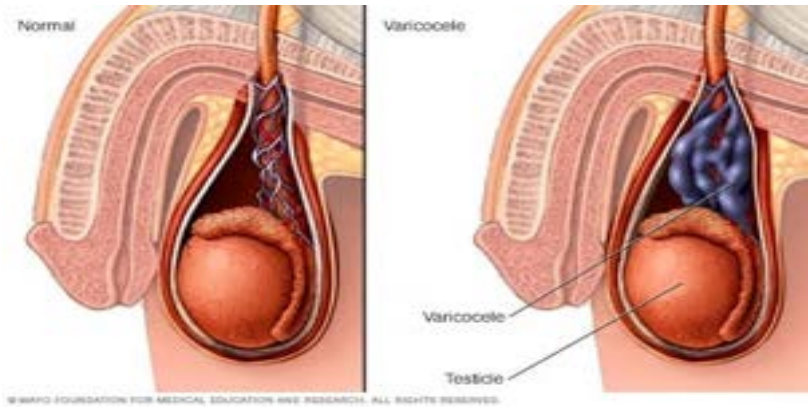


Figure (4): Normal pampiniform plexus and varicocele:

Presentations:

- Usually asymptomatic
- Painless scrotal soft lump.
- Dragging testicular pain with prolonged standing.
- Male subfertility

Examination: In standing and supine position. Varicocele can be graded into

1 Palpable on straining.

2 Palpable without straining.

3 Visible and of course palpable without straining.

Investigations:

Semen analysis: for the diagnosis of any associated abnormalities.

Colored duplex US: Dilated refluxing veins with a diameter ≥ 2.5 mm.

Treatment:

Only surgical treatment in cases of

- Male subfertility and associated semen analysis abnormalities.
- Pain refractory to the treatment.
- Affection of the testicular size.

Surgical options:

Microscopic varicocele ligation via sub inguinal or inguinal surgical approach is considered the gold standard.

Laparoscopic varicoceles ligations may be considered in bilateral cases.

Radiographic occlusion. May be considered in recurrent varicoceles.

Andrological emergencies

Priapism

Definition:

Persistent erection without sexual desire and not relieved by ejaculation that lasts more than 4 hours.

Types:

Ischemic: Veno-occlusive. Penis is rigid, tender and painful. The commonest.

Non-ischemic: arterial or high flow. Penis not fully rigid nor painful.

Causes:

Idiopathic in > 35%.

Drug induced after intra-cavernous injection or over dose of antipsychotics.

Blood diseases: Sickle cell diseases, Thalassemia or leukemia.

Spinal cord injury or multiple sclerosis.

Scorpion bite.

Treatment:

Immediate drainage and intra-cavernous injection of vasoconstrictor drugs.

Surgical shunt surgeries if the drainage failed.

Penile prosthesis implants in cases of neglected priapism associated with penile fibrosis and ED.

Penile Fracture:

Rupture of the tunica albuginea of the corpora cavernosa.

Caused by blunt trauma to the erected penis.

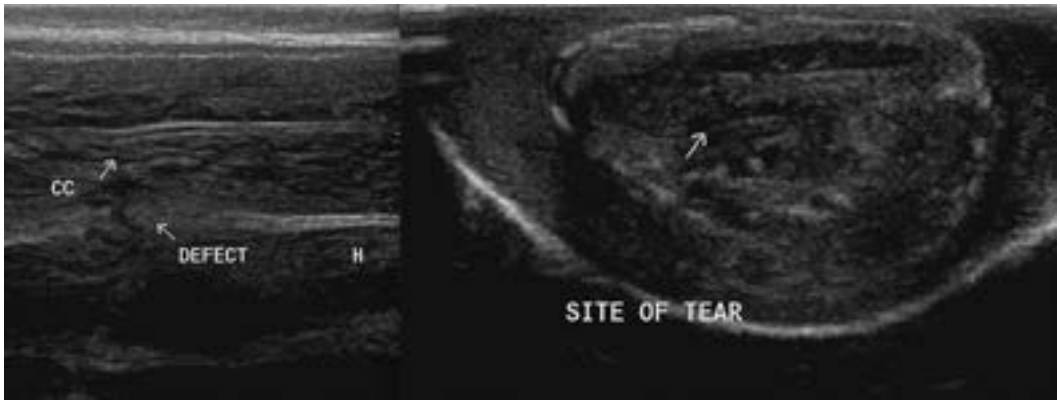
Presentations:

Patient reports hearing of cracking sound during the intercourse followed by detumescence of the erection.

Intense penile pain, swelling and sometimes urethral bleeding.

Investigations:

Penile U/S or MRI can reveal the site of the tear.



Penile U/S reveal the site of the tear.

Complications:

Penile curvature and ED.

Treatment:

Immediate surgical repair.

Priapism

Definition

A persistent penile erection in the absence of sexual stimulation > 4 hours.

Epidemiology

About 5.34 cases per 100,000 men per year

Risk Factors

Sickle cell anaemia: up to 75% of patients with Sickle cell anaemia might experience at least one episode of priapism in their life. Other risk factors include Leukaemia, lymphoma, drugs e.g., 5 alpha reductase inhibitors, trazodone, 5and cocaine.

Aetiology

Obstruction of cavernosal blood outflow secondary to failure of cavernosal smooth muscles to relax, or to blockage of the venous drainage e.g., sickle cell anaemia or pelvic malignancy.

Classification

Ischemic priapism:

More common form of priapism, associated with hypoxia and acidosis which result in local tissues damage.

Non-ischemic priapism:

It occurs when the arterial in-flow exceeds the venous out-flow; with lower risk of ischemia and less tissue damage than in the ischemic type.

Clinical presentation

A “rock hard” painful erection, clinical examination shows that the corpora cavernosum is erect while the glans and corpus spongiosum are soft. Patients with non-ischemic priapism may have a partial, painless erection.

Investigation

Penile blood gas: in ischemic priapism it shows signs of abnormal tissue perfusion ($pO_2<30$, $pCO_2>60$, $pH<7.25$).

Penile blood gases in patients with non-ischaemic priapism show high oxygen levels and no acidosis ($pO_2>90$, $pCO_2<40$, $pH \sim 7.40$).

Complete blood count with haemoglobin electrophoresis considered in patients at risk for sickle cell disease.

Treatment

Immediate treatment is important to avoid the risk of permanent tissue damage and erectile dysfunction as up to **50% of patients** with resolution of the priapism **within 24 hours will retain erectile function**

Compared with almost **none of patients after 36 hours.**

Treatment of Ischemic priapism:

Aims to remove the blood from the corpora in order to allow arterial inflow. It includes aspiration and irrigation of the corpora with normal saline. Intracorporal injection of 1 cc of 1 mg/1mL phenylephrine, diluted in 19 cc of 0.9% normal saline every three to five minutes until detumescence is achieved with close monitoring of vital signs, if unsuccessful, surgical intervention should be considered.

Surgical intervention

Distal shunts: aim to create a communication between the distal corpora cavernosa and the corpus spongiosum of the glans.

Proximal shunts: aim to drain the proximal corporal cavernosa into the corpus spongiosum or into veins such as saphenous vein.

Refractory cases may require dilatation of the corpora cavernosa and insertion of a penile prosthesis.

Treatment of Non-ischemic priapism:

Typically, it resolves spontaneously in 60% of cases). In patients with persistent non-ischemic priapism, selective embolization should be considered.

Phimosis:

Inability to retract the prepuce due to narrowing or stenosis of the outlet.

May be congenital or acquired.

Presented by difficult voiding, acute urine retention.

Treatment id circumcision.

Paraphimosis:

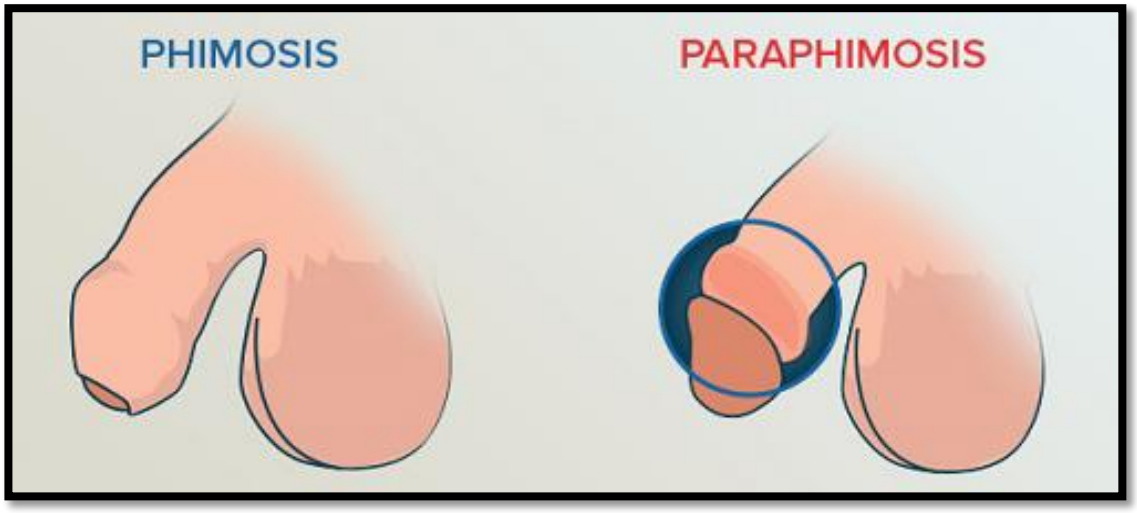
Partial retraction of the skin behind the glans penis forming a constriction ring. This leads to edema, venous congestion and ischemia of the glans with the possibility of subsequent gangrene.

Treatment:

Trial of manual reduction of the prepuce to its normal position.

Dorsal slit at 12 o'clock position in cases of failure of reduction.

Circumcision.



Circumcision complications:

Bleeding, penile skin loss, under or over circumcision, uretho-cutaneous fistula and partial or complete penile amputation.

Causes of penile amputation:

Over use of diathermy.

Accidental injury by the circumcision clamp.

Extensive infection.

Injecting adrenaline with the local anesthesia.

Penile amputation one of the most **catastrophic complications** that needs multiple surgeries (Immediate urine diversion, staged phalloplasty and finally implanting a penile prosthesis).

Never ever use adrenaline for penile skin infiltration.

Never ever cut the skin with a diathermy.

Acute scrotum:

Differential diagnosis:

- Testicular torsion.
- Torsion of appendix of the testis.
- Strangulated inguinal hernia.
- Acute epididymitis or acute epididymo-orchitis.
- Mumps orchitis.
- Testicular trauma.

Testicular torsion

Twisting of the spermatic cord leading to strangulation of the blood supply of the testis and epididymis.

Aetiology:

Lack of testicular fixation inside the tunica vaginalis, which make it freely mobile.

Symptoms:

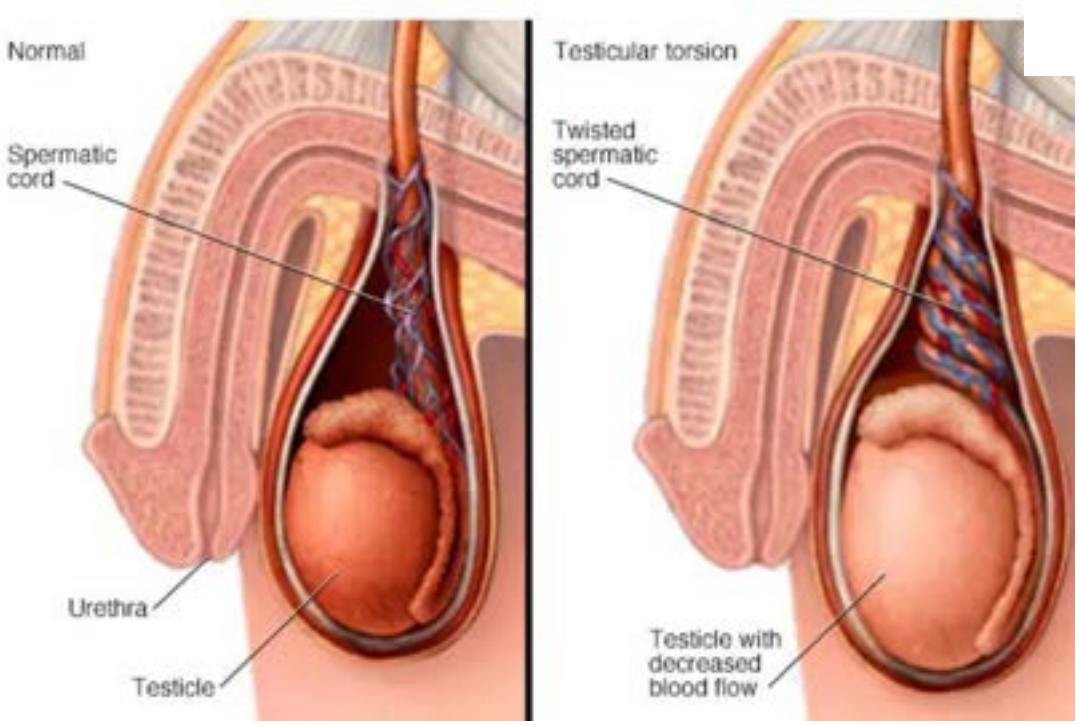
Sudden onset of severe testicular pain, followed by scrotal swelling and GIT manifestation.

Signs:

Enlarged tender testis.

High riding due to twisting and shortening of the cord.

Absent cremasteric reflex



Differential diagnosis:

Acute epididymo-orchities: gradual onset, general constitutional manifestations and fever, associated urinary symptoms.

Management:

Immediate colored duplex US: differentiate the inflammation from ischemia.

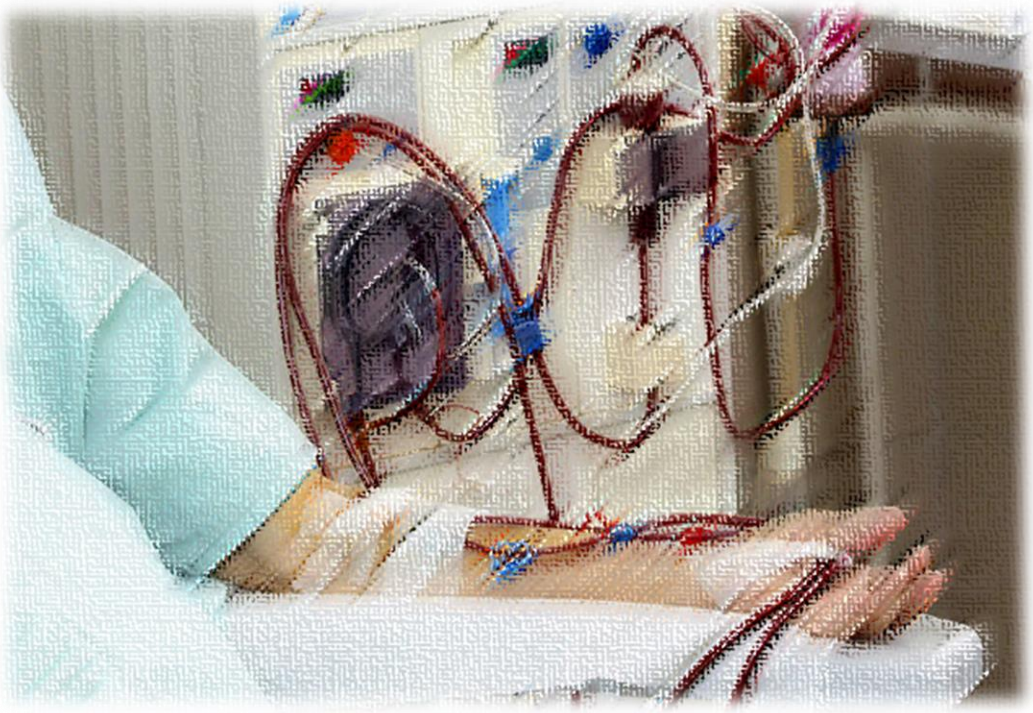
If still in doubt, take the testis out:

Which means immediate surgical exploration

If the testis is viable; detorsion and bilateral testicular fixation.

Gangrenous testis, orchidectomy and fixation of the contralateral testis.

RENAL FAILURE



Renal Failure

Acute kidney injury

Definition:-

Acute kidney injury (AKI, previously known as 'acute renal failure') is a condition in which a patient has a rapid decline in kidney function over hours to days, leading to an accumulation of waste-products of metabolism and disturbances in fluid, acid–base balance, and electrolyte balance.

Incidence:-

2-5% of patients admitted to hospital.

20% of patients in ICU or after cardiovascular surgery

Stages of AKI

Stage	Serum creatinine	Urine output
1	1.5–1.9 × baseline OR ≥0.3mg/dL (≥26.5µmol/L) increase	<0.5ml/kg/h for 6–12h
2	2.0–2.9 × baseline	<0.5ml/kg/h for ≥12h
3	3.0 × baseline OR Increase in serum creatinine to ≥4.0mg/dL (≥353.6µmol/L) OR Initiation of renal replacement therapy OR Decrease in eGFR to <35ml/min per 1.73m² (in those <18 years old)	<0.3ml/kg/h for ≥24h OR Anuria for ≥12h

Oliguria is decreased urine volume to less than 400 ml in a day.

Anuria is complete absence of urine production by the kidneys for 12 hours or more (<30ml/hr).

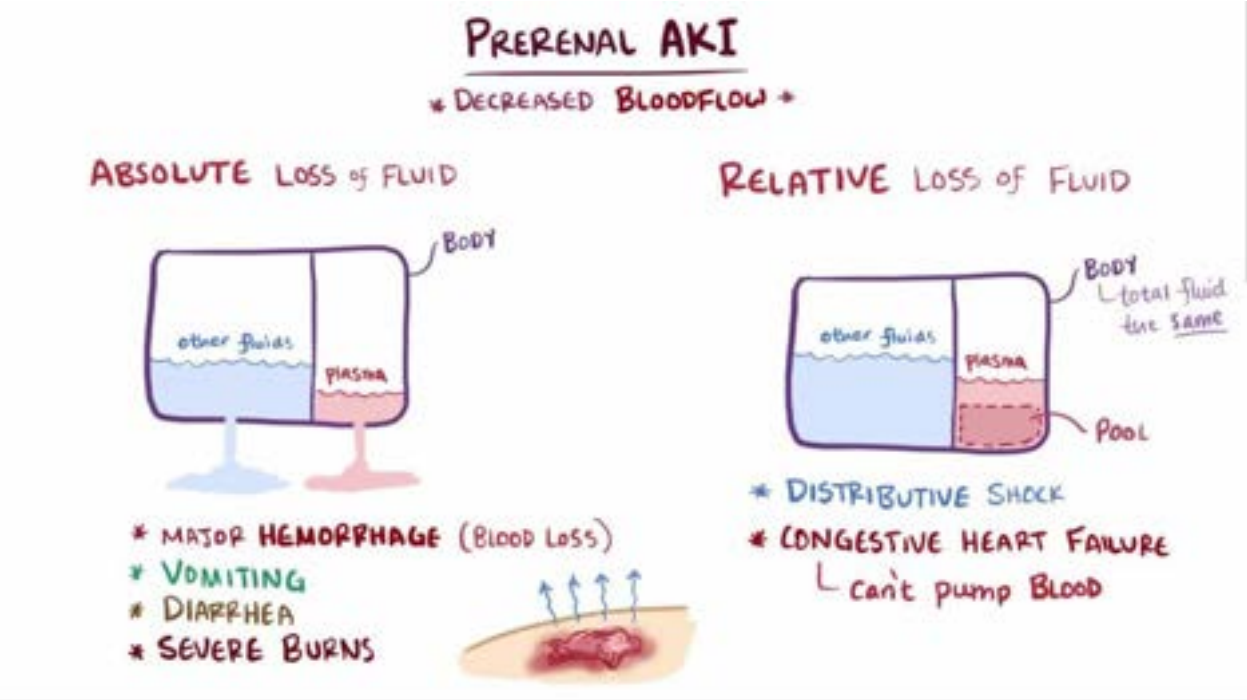
Classifications of AKI:-

AKI is classified based on etiology into pre-renal, renal (intrinsic), and post-renal.

Pre-renal AKI

Causes:-

1. *Hypovolemia: dehydration, prolonged vomiting, diarrhea, burns, excessive sweating.*
2. *Hemorrhagic shock: trauma, surgery and postpartum.*
3. *Sepsis: urinary, biliary.*
4. *Cardiogenic shock: myocardial infarction and pulmonary embolism.*



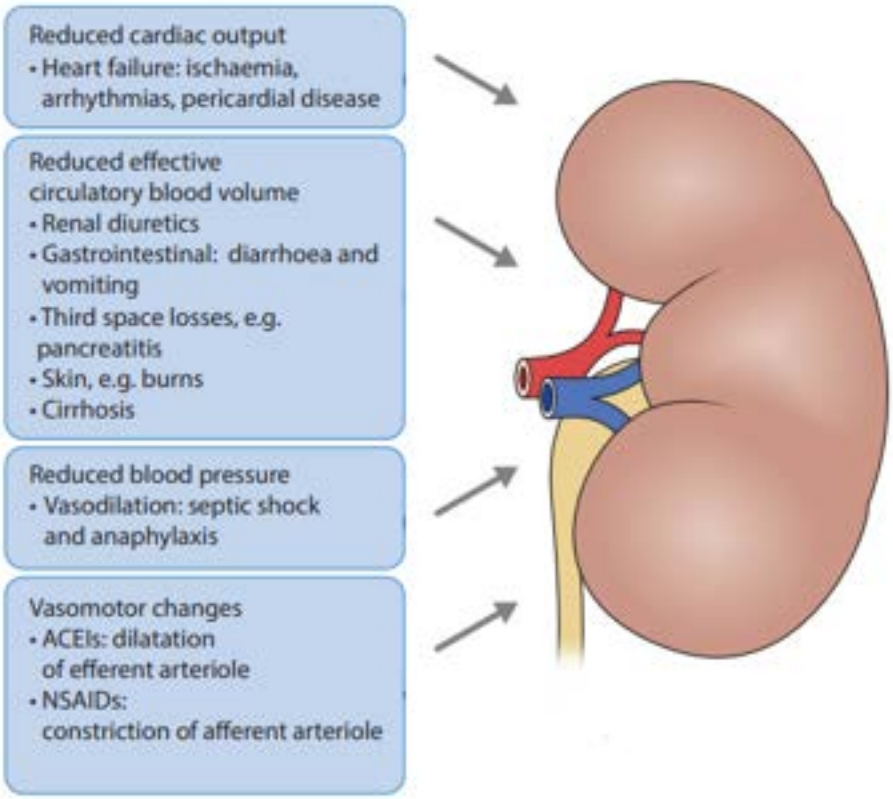
Pathophysiology: *Hypotension → Shock → ↓Renal perfusion→↓ GFR → Oliguria or anuria.*

This lead to: -

Stimulation of renin angiotensin system → V.C and increased sodium reabsorption.

Release of ADH → increase water reabsorption.

There is no injury to the renal parenchyma, and restoration of renal blood flow restores glomerular filtration, reversing AKI.



Intrinsic AKI

Renal parenchyma can be damaged by tubulointerstitial diseases, glomerular diseases, or vasculitis. Prerenal causes, if untreated or severe, can lead to acute tubular necrosis (ATN) which by far is the most common cause of AKI amongst patients in hospitals

The commonest causes include sepsis, nephrotoxins and ischaemia.

Pathophysiology: Acute tubular necrosis (ATN) → oliguria → recovery within 2-3 weeks of proper management. Prolonged prerenal ARF resulting in prolonged renal ischemia may lead to ischemic ATN.

Causes

1- Nephrotoxic drugs given to patients with impaired renal function: Aminoglycosides. Prolonged use of NSAIDs. Angiotensin – converting enzyme inhibitors (ACEIs) Cisplatinum Cyclosporine overdosage in kidney transplant patients.

2- Poisons.

3- Contrast media in patients with diabetes mellitus and multiple myeloma.

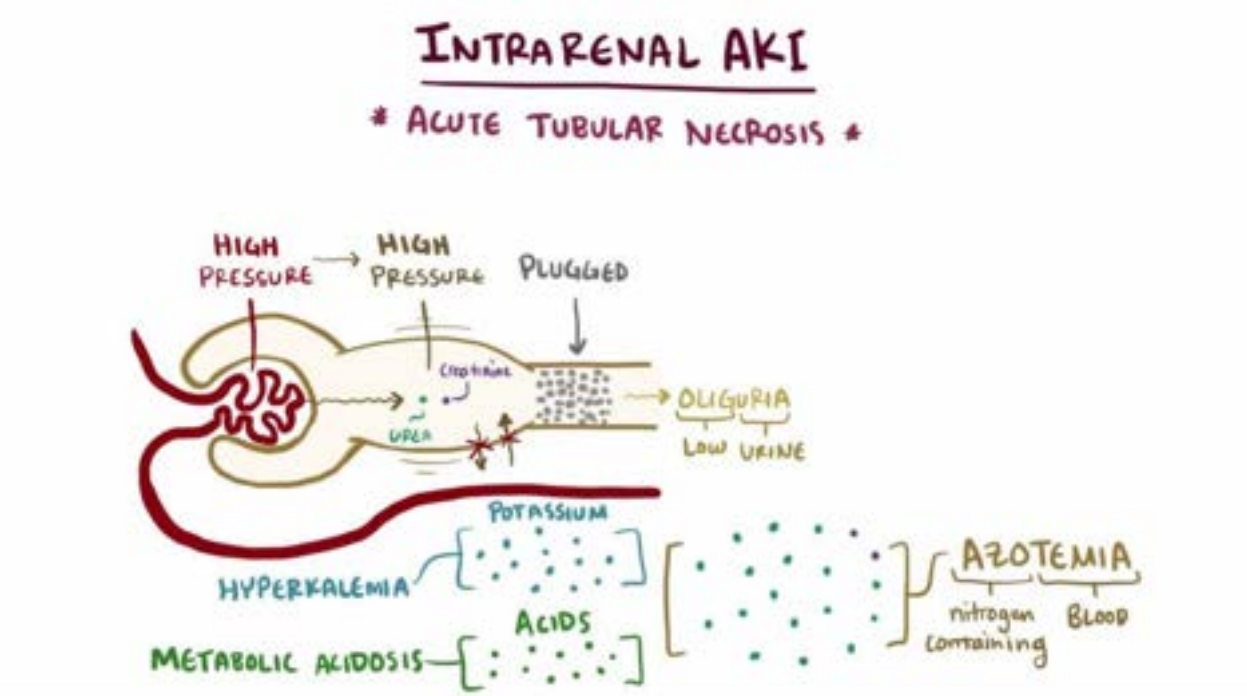
4- Anesthesia.

5- Eclampsia.

6- Incompatible blood transfusion.

7- Disseminated intravascular coagulopathy (DIC). 8- Myoglobinuria: Crush syndrome.

9- Kidney transplantation: prolonged ischemia, acute rejection, cyclosporine toxicity.



Acute Tubular Necrosis (ATN)

The most common cause of ARF in hospitalized patients.

Causes: -

Renal hypoperfusion & ischemia

Nephrotoxic Insults (Endogenous e.g pigment nephropathy or tumour lysis syndrom or exogenous e.g NSAID, Aminoglycosides or contrast media.

Renal Failure

Phases:

- The oliguric phase: last for 1-3 weeks.

- The diuretic phase: progressive increase of urine volume, death, may occur due to fluid and electrolyte abnormalities.

- The recovery phase: renal function returns but the abnormalities of urine concentration and dilution may persist for weeks or months

- Pathophysiology: Renal ischemia → Depletion of ATP → Impaired function of Na/K ATPase enzyme in the plasma membrane → Increase intracellular Na → Cell swelling.
- Treatment:

✓ Early consultation with a nephrologist.
✓ Fluid balance: carefully monitor intake/output and body weight.
✓ Moderate protein intake: (1-1.8 g/kg/day) to maintain positive nitrogen balance.
✓ Restriction of dietary phosphorus, potassium and sodium chloride
✓ Prevent and treat hyperkalemia.

Keep s. bicarbonate >15mEq/L by IV sodium bicarbonate

✓ Drugs: - Review all medications.

- Adjust dosage for renal failure.

- Stop Mg containing medications.

- Diuretics: decrease renal injury and wash out obstructive debris and casts.

- Dopamine (renal dose): cause V.D and increase renal blood flow (RBF).

- Ca channel blockers: increase GFR and renal plasma flow.

- ANP: cause V.D and still under trials.

✓ **Dialysis**: Indications:

- Lab: s.creatinine >10 mg/dl.
  BUN >200 mg/dl.
  K >7 mEq/L
  HCO3 <10 mEq/L

- Clinical: Coma or Convulsions.
  Pulmonary edema.
  Pericarditis.
  Sever bleeding.

Acute Interstitial Nephritis

Causes:

- ✓ Drugs e.g NSAID, aminoglycosides or allopurinol.
- ✓ Sarcoidosis.
- ✓ Infection e.g Streptococcal, viral or legionella.

Presentation:

- ✓ Fever.
- ✓ Sterile pyuria, WBC casts and eosinophiluria.
- ✓ Eosinophilia (>75%).
- ✓ Proteinuria (<0.5 to 1 g/day).
- ✓ Rash (25%).

- Renal Biopsy:

- ✓ Confirm diagnosis.
- ✓ Interstitial edema.
- ✓ Marked Interstitial infiltrate of T lymphocytes and monocytes.

Treatment:

- ✓ Stop causing drug.

Corticosteroids and/or cytotoxic therapy to hasten recovery and reduce interstitial fibrosis

Acute Glomerulonephritis

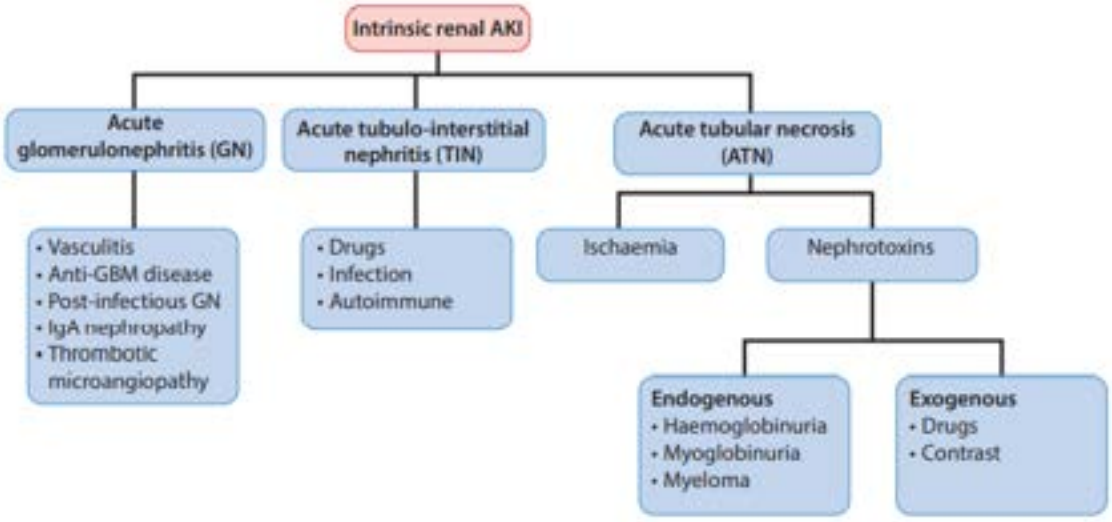
Presence of proteinuria, hematuria and RBCs cast is pathognomonic.

Evaluation:

- ✓ Renal Biopsy.
- ✓ Serologic evaluation for systemic vasculitis, collagen vascular disease, and infectious process.

Treatment:

- ✓ Corticosteroid or Cyclophosphamide.
- ✓ Plasma exchange.



	Pre-renal AKI	Intrinsic renal AKI
BP	Hypotension common	Normal or elevated
Volume status (jugular venous pressure, postural BP)	Evidence of hypovolaemia	Normal
Urine dipstick	Unremarkable	Blood and protein (more in glomerulonephritis than ATN and acute interstitial nephritis)
Urine microscopy	Unremarkable	Red cell casts pathognomonic of acute glomerulonephritis
Response to effective fluid challenge	Improvement in urine output	No effect

Post-renal AKI

It is caused by obstruction of the urinary tract and may be categorized as either supra-vesical or infra-vesical, depending upon the level of obstruction in relation to the bladder.

The classic example is a stone impacted in the ureter of a solitary kidney (spasm of ureteral muscle, edema of the mucosa). There is renal pain and anuria. The urinary bladder is empty. There is circulatory volume and osmotic overload. Serum creatinine is rising and electrolyte imbalance will occur.

Causes

1- Stones are the most common cause. The other kidney may be surgically absent or nonfunctioning.

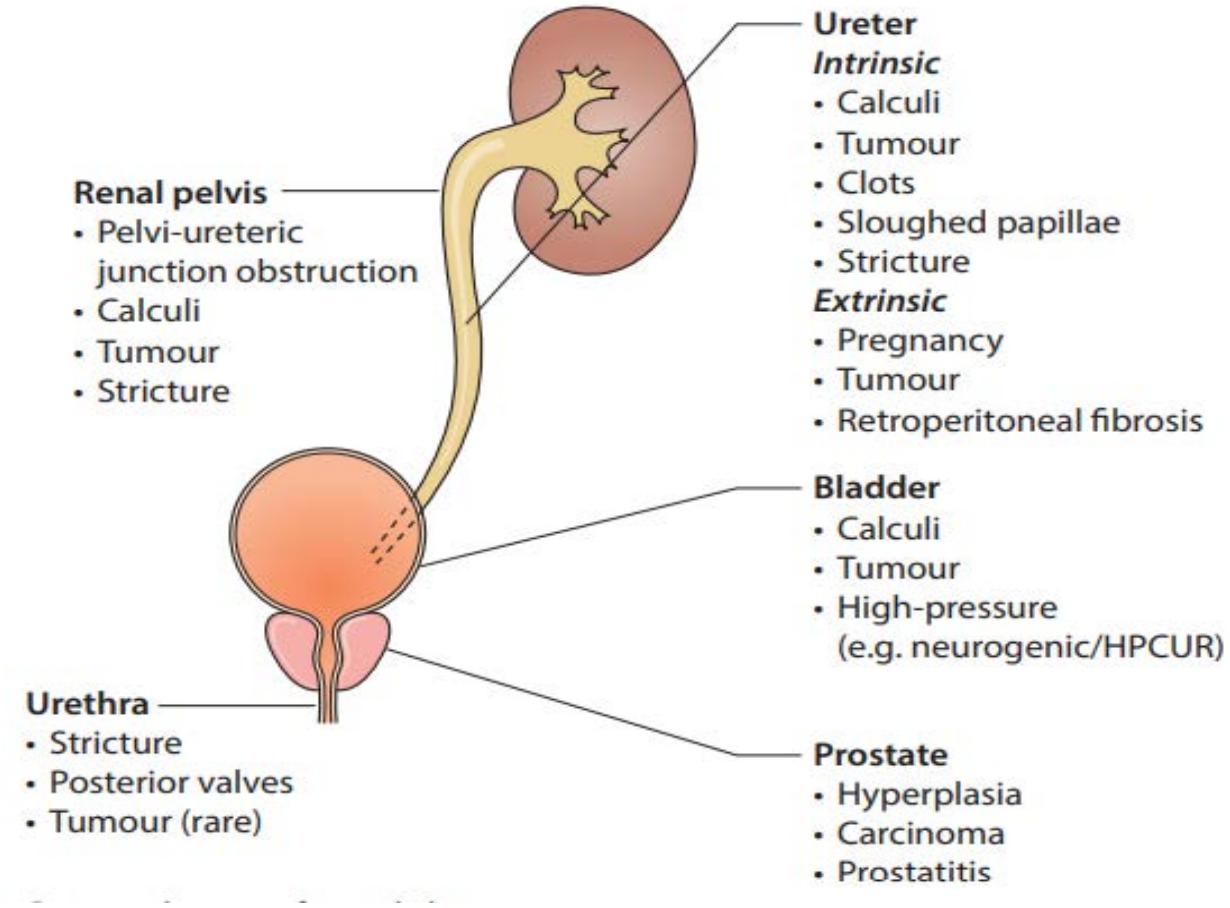
2- Malignant tumors causing bilateral ureteral obstruction: prostate, bladder, cervix, rectosigmoid

3- Surgery: bilateral ligation of the ureters during hysterectomy.

4- Retroperitoneal fibrosis.

5- Bilharzial ureteric strictures.

6- Crystaluria: uric acid crystals in patients receiving chemotherapy for lymphoma and leukemia.



Clinical picture

- Non-specifically unwell.
- Reduced urine output.
- Disordered volume status: Fluid overloaded or Dehydration.
- Hyperkalaemia: cardiac arrest or arrhythmias.
- Uraemia:

✓ Skin: dry skin, pruritis, uraemic frosting (last sign)
✓ GI: nausea, anorexia, weight loss.
✓ Nervous system: involuntary movement (restless legs), peripheral and autonomic neuropathy, psychological disturbances, encephalopathy.
✓ Haematological: anaemia, bleeding.
✓ Sexual dysfunction and reduced fertility.

Investigations

- Urine analysis: Evidence of infection, low specific gravity, tubular cells in intrinsic renal disease, or high specific gravity in prerenal causes.

- Blood tests:

- FBC: • Anaemia: haemolysis or active bleed • WCC: raised in sepsis

• ESR: raised in SLE, myeloma.

- Urea and Creatinine: • Increased plasma urea: creatinine ratio more likely pre-renal cause

• Increased plasma creatinine: urea ratio makes obstruction and intrinsic disease more likely

• Hyperkalaemia: associated with life-threatening arrhythmias

- LFT: • Abnormal in hepatorenal syndrome
- Calcium: • Hypercalcaemia is seen with malignancy and myeloma

- Creatine kinase: • Rhabdomyolysis: consider in patients with a history of burns, crush injuries or lying for a prolonged time Immunology screen:
- ANA: positive in autoimmune disease
- ANCA: small vessel vasculitis:
- Anti-GBM: Goodpasture's disease.
- Imaging: • Ultrasound renal tract (urgent) • Exclude obstruction • Ensure presence of 2 kidneys and assess size: small scarred kidneys suggest CKD.
- Renal biopsy: • Mainly to identify intrinsic renal disease.

- In kidney transplant: - Acute rejection: lymphoblastic cells.

  - Cyclosporine toxicity: damaged tubular and endothelial cells.

<u>Treatment</u>

- In pre-renal AKI it is important to achieve and maintain haemodynamic stability.
- Fluid resuscitation is key and dependent on accurate clinical assessment of fluid balance. Fluid intake should be equal to daily urine volume + sweat + perspiration. Excessive intake may lead to congestive heart failure and pulmonary edema.
- Crystalloids should be given, except in the case of bleeding, where blood and blood products are indicated.

The management of AKI can be divided into general supportive measures and renal replacement therapy.

In particular, obstruction should always be excluded, especially in oligouric and anuric patients with no other obvious cause (e.g. no overt haemodynamic disturbance)

Assess fluid balance:

- Pulse: tachycardia
- BP: hypertension or hypotension
- Jugular venous pressure
- Evidence of pulmonary oedema
- Urine output

Management:

- Give 500ml crystalloid fluid bolus (reduce to 250ml in those with cardiac failure or >75 years old)
- Reassess fluid balance
- If required give further 500ml of crystalloid (250ml in those with cardiac failure or >75 years old)
- Continue maintenance fluids until euvolaemic
- If the patient remains oligoanuric despite adequate filling, defined as having volume unresponsive AKI, they require input from a nephrologist

Post-renal AKI treatment

- Cystoscopy and ureteral catheterization to bypass the obstruction and drain the kidney or urethral catheter.

- Percutaneous nephrostomy (PCN) if ureteric catheter fails.
- Treatment of the cause e.g. ureteroscopic stone removal.

Postobstructive diuresis: relief of obstruction is followed by diuresis due to volume and osmotic overload. Adequate fluid balance should be observed.

- Treatment of hyperkalemia.
- Dialysis.
- Treatment of hyperkalaemia.
- Stabilise the myocardium:
- **1)** 10ml of 10% calcium gluconate IV over 2 min (remember this does not affect the potassium level).
- **2)** Measures to reduce serum potassium (move potassium from extracellular to intracellular compartments):
- Low potassium diet.
- Insulin–dextrose: • 10 units of Actrapid® in 50ml of 50% dextrose over 20 min. Potassium will decrease within 15 minutes. Glucose levels should be monitored carefully to avoid hypoglycaemia
- Beta 2 agonists: • 10–20mg of nebulised salbutamol. Be careful as can cause tachycardia and precipitate arrhythmia.
- Isotonic (1.26%) sodium bicarbonate: • Use under expert guidance only

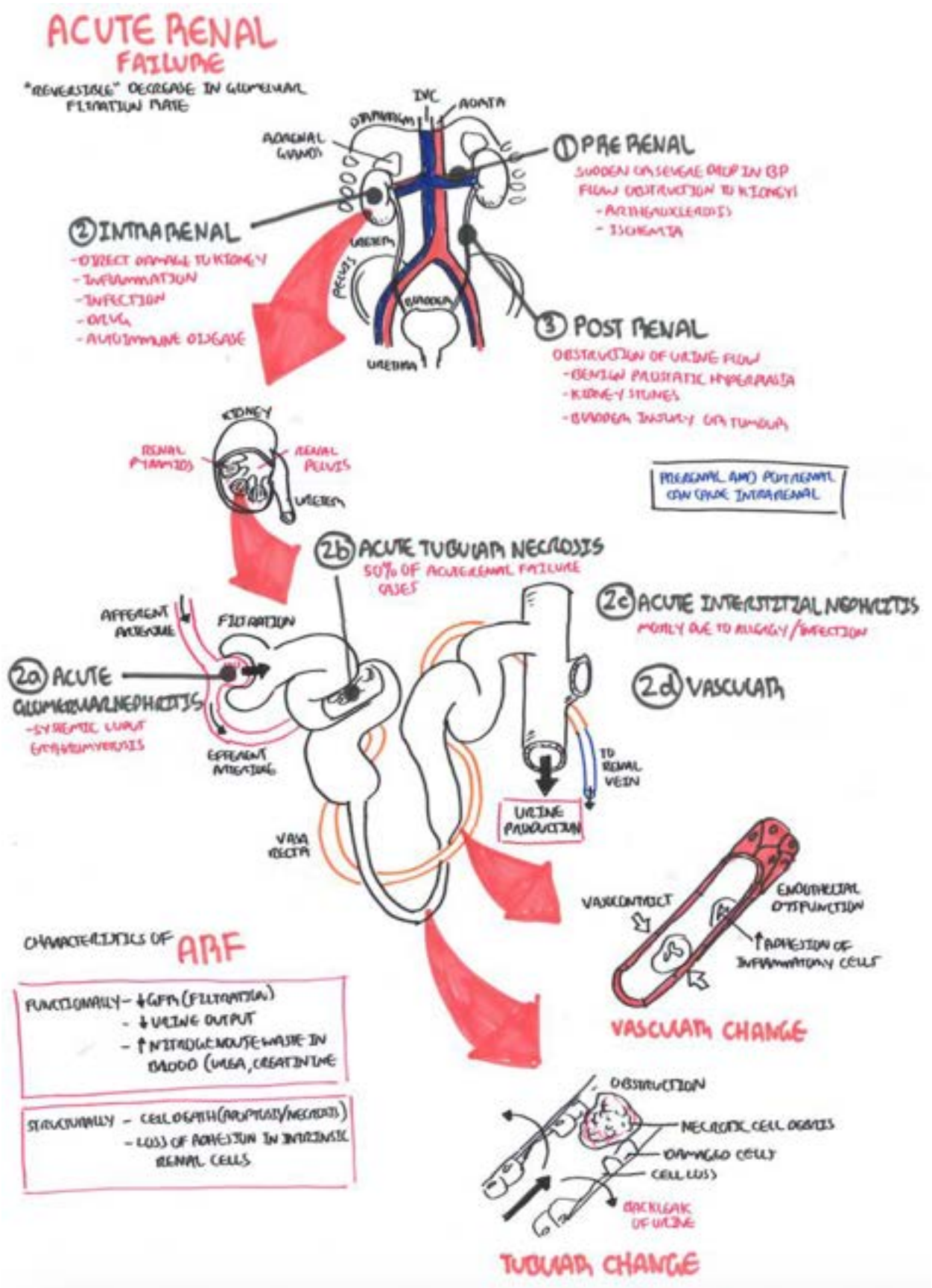
- **3)** Remove potassium from the body:
- Cation exchange resins: • Calcium resonium or sodium polystyrene sulphonate. Removes potassium from the blood in the gut in exchange for sodium.
- Dialysis: • If the above measures fail.

General supportive management

- Investigate and treat the underlying cause.
- Achieve normal haemodynamic status – fluids, vasopressor or inotropic support.
- Adjust dose and frequency of medications appropriately for level of renal function.
- Avoid nephrotoxins – ACEI, NSAID, aminoglycosides.
- Avoid hyperglycaemia.
- Nutritional support (restriction of dietary protein).

Indications for referral to a nephrologist.

- Patients meeting the criteria for renal replacement therapy.
- Diagnosis requires specialist input, e.g. vasculitis, GN or TIN.
- Cause of AKI unclear.
- Inadequate response to initial treatment.
- Renal transplant patient.
- Stage 3 AKI.
- Pre-existing advanced CKD (CKD 4 and 5).


ACUTE RENAL FAILURE
"REVERSIBLE" DECREASE IN GLOMERULAR FILTRATION RATE
IVC
AORTA
DIAPHRAGM
ADRENAL GLAND
① PRE RENAL
SUDDEN OR SEVERE DROP IN BP
FLOW OBSTRUCTION TO KIDNEY
- ANTIHYPERTENSIVES
- ISCHEMIA
② INTRA RENAL
- DIRECT DAMAGE TO KIDNEY
- INFLAMMATION
- INFECTION
- DRUGS
- AUTOIMMUNE DISEASE
URETER
PELVIS
BLADDER
URETHRA
③ POST RENAL
OBSTRUCTION OF URINE FLOW
- BENIGN PROSTATIC HYPERPLASIA
- KIDNEY STONES
- BLADDER INJURY OR TUMOUR
KIDNEY
RENAL PYRAMID
RENAL PELVIS
URETER
PRERENAL AND POSTRENAL CAN CAUSE INTRARENAL
2b ACUTE TUBULAR NECROSIS
50% OF ACUTE RENAL FAILURE CASES
2c ACUTE INTERSTITIAL NEPHRITIS
MOSTLY DUE TO ALLERGY/INFECTION
2d VASCULAR
AFFERENT ARTERIOLE
FILTRATION
2a ACUTE GLOMERULONEPHRITIS
- SYSTEMIC LUPUS ERYTHEMATOSUS
EFFERENT ARTERIOLE
TO RENAL VEIN
URINE PRODUCTION
VASA RECTA
VASOCONSTRICT
ENDOTHELIAL DYSFUNCTION
↑ADHESION OF INFLAMMATORY CELLS
VASCULAR CHANGE
CHARACTERISTICS OF ARF
FUNCTIONALLY - ↓GFR (FILTRATION)
- ↓URINE OUTPUT
- ↑NITROGENOUS WASTE IN BLOOD (UREA, CREATININE
STRUCTURALLY - CELL DEATH (APOPTOSIS/NECROSIS)
- LOSS OF ADHESION IN INTRINSIC RENAL CELLS
OBSTRUCTION
NECROTIC CELL DEBRIS
DAMAGED CELL
CELL LOSS
BACKLEAK OF URINE
TUBULAR CHANGE

Chronic Kidney Disease

Definition:-

- Kidney damage present for at least 3 months, with either structural or functional abnormalities of the kidney with or without decreased glomerular filtration rate (GFR).
- In the early stages of CKD, evidence of kidney damage (e.g. proteinuria, cysts, biopsy changes) may be seen in the presence of a normal GFR (> 60 mL/minute/1.73 m2), while the later stages are characterized by greater functional impairment, with the GFR falling to below 60 mL/minute/1.73 m2.
- Stages of chronic kidney diseases

GFR (mL/minute/1.73 m²)	Disease stage	Comments
> 90	1	Normal, if there is no kidney damage by any criterion
60–89	2	May be normal for age; represents decreased GFR only, if there is no kidney damage
30–59	3	Always abnormal
15–29	4	Always abnormal
< 15 or dialysis	5	End-stage renal failure

Causes:-

- The three most important causes of CKD are

  Diabetes,

  Glomerulonephritis

  Hypertension.

- A variety of disorders are associated with CKD either a primary renal process (eg, glomerulonephritis, pyelonephritis, congenital hypoplasia) or a secondary one (owing to a systemic process such as diabetes mellitus or lupus erythematosus) may be responsible.

Superimposed physiologic alterations secondary to dehydration, infection, obstructive uropathy, or hypertension may put a borderline patient into uncompensated chronic uremia

Clinical features

- CKD usually presents with non-specific symptoms caused by kidney failure and the underlying disease, or is discovered by chance following a routine blood or urine test.
- Specific symptoms usually develop only in severe kidney failure.
- The most common symptoms in late kidney failure include anorexia, nausea, vomiting, fatigue, weakness, pruritus, edema, lethargy, dyspnea, insomnia, muscle cramps, pulmonary edema, nocturia, polyuria and headache.
- Sexual dysfunction is rarely reported voluntarily, but is common.
- Hiccups, pericarditis, coma and seizures are seldom seen except in developing countries when kidney disease presents very late.

Signs of CKD include skin pigmentation or excoriation, anemia, hypertension, postural hypotension, edema, left ventricular hypertrophy, peripheral vascular disease, lung crackles, pleural effusions, peripheral neuropathy and urine abnormalities (presence of blood or protein)

Diagnosis

- Recognition of abnormal kidney function is the key to the diagnosis of CKD. However, blood urea nitrogen (BUN or serum urea) is an extremely poor marker of kidney function, because it varies significantly with hydration status and diet, it is not produced constantly and it is reabsorbed by the kidney.
- Historically, serum creatinine has been used, but this also has significant limitations, particularly the fact that the level can remain within the normal range despite the loss of over 50% of kidney function.
- A ‘gold-standard’ measurement is an isotopic glomerular filtration rate (GFR), but this is expensive and not available in community settings.
- It is now clear that calculated GFR using a formula based on serum creatinine is preferred, and can be a valid, reliable, repeatable and reasonably accurate measure of true GFR in patients with renal Renal Disorders impairment.
- Measurement of serum cystatin C is also a reliable marker of GFR, but this test is not yet widely available.
- The factors that will determine the likelihood of progression to ESKD are: • the level of albuminuria • the initial degree of renal impairment • blood pressure.
- - Treatments aimed at reducing albuminuria and vigorously controlling blood pressure have been shown to slow or even halt the decline in kidney function and to reduce the vascular complications, which are the leading cause of death in CKD.

Clinical approach in chronic kidney disease

	GFR (mL/minute/1.73 m²)	Management
Stage 1		
Kidney damage with normal GFR	≥ 90	Diagnose and treat underlying cause Treat comorbid condition Slow progression by controlling blood pressure and reducing proteinuria Reduce cardiovascular risk
Stage 2		
Kidney damage with mild decrease in GFR	60–89	As for stage 1 and estimate progression
Stage 3		
Moderate decrease in GFR	30–59	Prevent and treat complications
Stage 4		
Severe reduction in GFR	15–29	Prepare for renal replacement therapy (dialysis or transplantation)
Stage 5		
Kidney failure	< 15 (or dialysis)	Renal replacement therapy

Management of chronic kidney disease

Blood pressure

- Aim for < 130/80 mmHg in patients with albuminuria and < 140/90 mmHg in patients without albuminuria
- Use ACE inhibitors in all patients if tolerated and if renal artery stenosis is not present to slow disease progression (independent of their blood-pressure-lowering effect)

Albuminuria

- Reduce albuminuria as much as possible; ACE inhibitors and ARBs are particularly effective, but diltiazem, verapamil and thiazide diuretics can also be used

Conventional cardiovascular risk factors

- Stop smoking
- Eat a low-salt diet
- Take regular exercise
- Treat hypercholesterolemia, if present, with a statin
- (No evidence of benefit of folic acid in hyperhomocysteinemia)

Acidosis*

- Treat with sodium bicarbonate, but can cause fluid overload and worsen hypertension

Hyperphosphatemia†

- Treat with dietary restriction and phosphate binders

Malnutrition

- Must be avoided, although protein restriction can slow progression

Complications of chronic kidney disease

Complication	Effect
Anemia	Left ventricular hypertrophy, fatigue, impaired cognitive functioning
Hypertension	Left ventricular hypertrophy, heart failure, stroke, cardiovascular disease
Calcium phosphate imbalance	Cardiovascular and cerebrovascular disease, vascular calcification, arthropathy, soft tissue calcification
Metabolic bone disease	Bone pain, fractures
Dialysis amyloid	Bone pain, arthropathy, carpal tunnel syndrome
Fluid overload	Pulmonary edema, hypertension
Malnutrition	Increased morbidity and mortality, infections, poor wound healing

Renal Replacement Therapy

Kidney transplantation

Is **the treatment of choice for ESRD** in suitable patients because it improves both the survival and the quality of life (QoL) compared to dialysis.

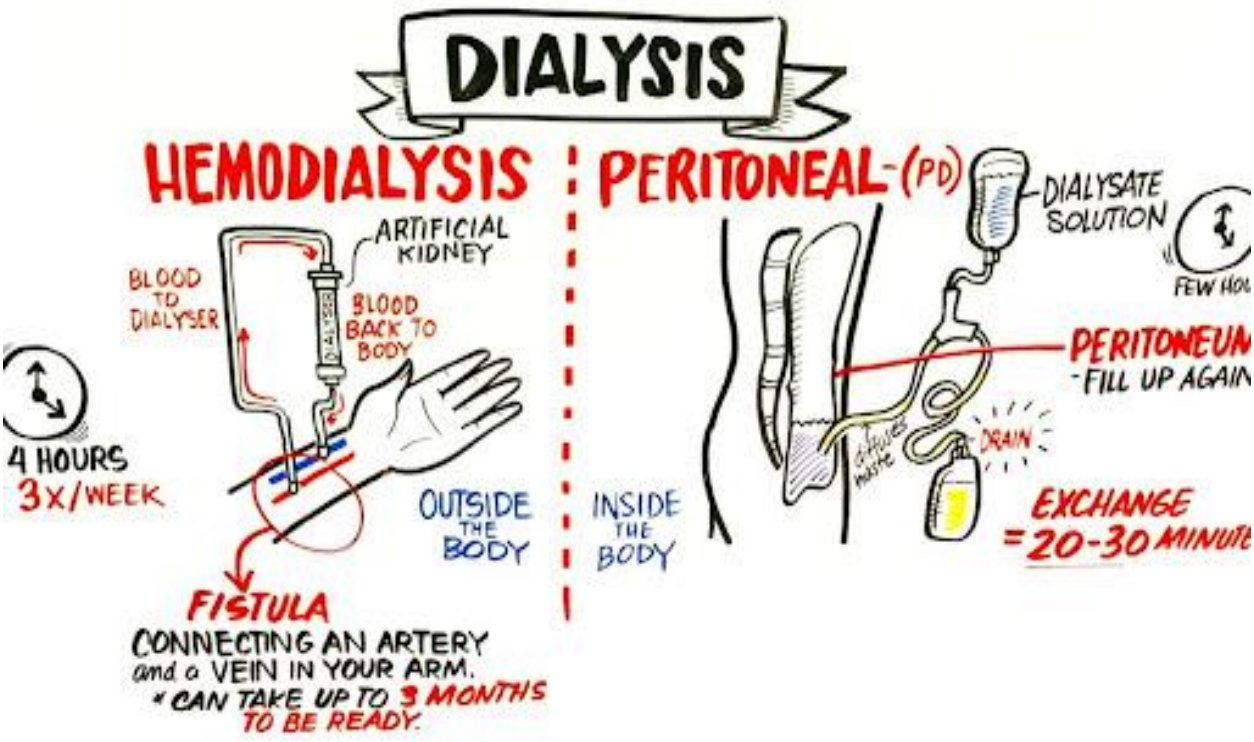
Dialysis

Peritoneal Dialysis

PD is a home-based mode of RRT, which gives a degree of flexibility and control to patients with ESRD in terms of lifestyle.

Haemodialysis

Blood from the patient flows through an extracorporeal circulation over a thin semi-permeable membrane separating it from the dialysate fluid



KIDNEY TRANSPLANTATION

Kidney transplantation

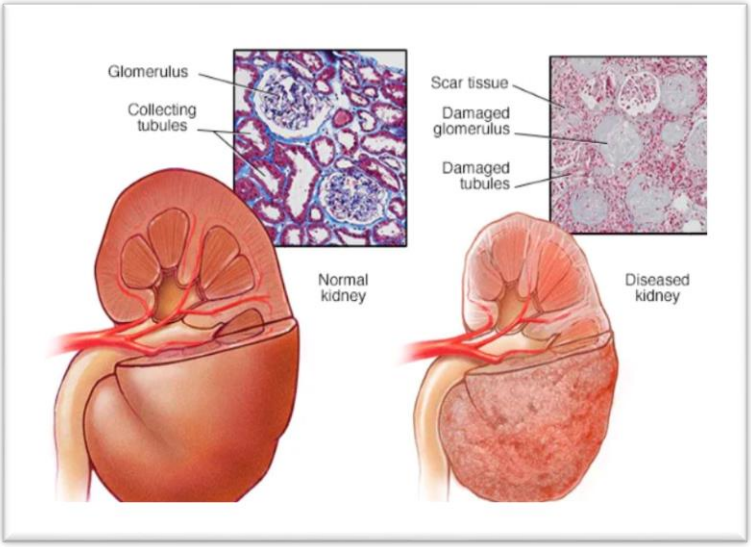
Introduction:-

Renal transplantation has become the **procedure of choice** for the management of patients with end stage renal disease because it effectively replaces both the exocrine and endocrine (erythropoietin production, vitamin D activation, etc.) function of the kidney. Also, it reduces the morbidity and mortality of end-stage renal disease. As well as a better quality of life than dialysis).

Indications:-

The indication for kidney transplantation is (ESRD), which is defined as a glomerular filtration rate below 15 ml/min/1.73 m^2.

Common causes of end-stage kidney disease include:-



Diabetes

Chronic, uncontrolled high blood pressure

Chronic glomerulonephritis, focal-segmental glomerulosclerosis

Polycystic kidney disease

Post-renal causes (reflux nephropathy, obstruction)

Types of renal transplantation

Deceased-donor kidney transplant

Living donor kidney transplant

Preemptive kidney transplant

Contraindication:-

Absolute contraindications:

Advanced physiological age (above 70 years).

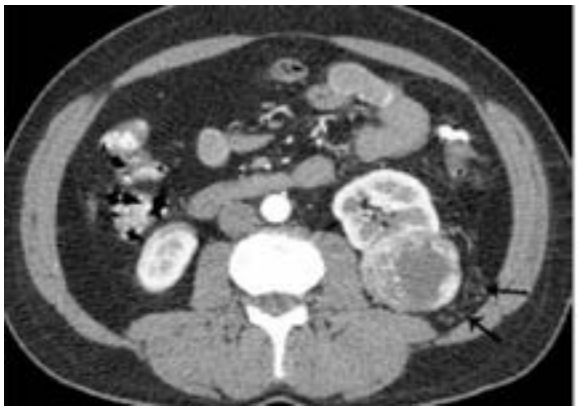
Incurable infection e.g. HIV and cytomegalovirus.

Severe untreatable cardiac disease.

Active malignancy.

Dementia or poorly controlled mental illness

Alcohol or drug abuse



Relative contraindications (if treated, transplantation can be considered)

Urinary tract abnormalities as UTI and BOO.
Active peptic ulcer.

Curable infections and septic foci.

Systemic and metabolic diseases that can potentially damage the graft after successful transplantation e.g. oxalosis, cystinosis and immunological diseases

The donor

Types:

1- Living related

2- Living un-related

3- Cadaver donor

Prerequisites:

Age between 21-60 years with written consent for donation.

Perfect urinary tract and renal function of both renal units.

Free of infectious diseases and malignancy.

Absence of any generalized disease that could adversely affect renal vessel integrity or perfusion e.g. DM or hypertension.

Compatibility evaluation:

Once the patient and donor are accepted,

ABO & Rh blood grouping, HLA tissue typing

Indication of Pre-transplant nephrectomy of the recipient kidney

Uncontrolled renal hypertension.

Nephrotic syndrome.

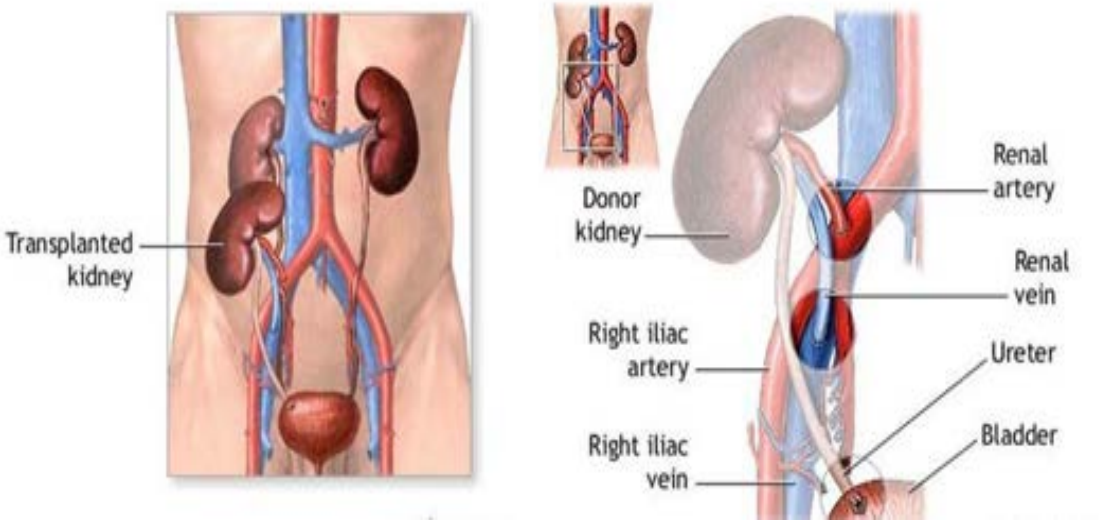
Unresolved urinary tract infection.

Extremely large kidney/s e.g. polycystic kidney disease.

Surgery

The transplanted kidney is placed at the iliac fossa. The renal vessels are anastomosed to the iliac vessels and the ureter is implanted into the bladder.

The patient should be maintained on immunosuppressive therapy for life.



Complications:-

Surgical complications:-

Vascular renal artery thrombosis and renal vein thrombosis

ureteral obstruction

Lymphocele: This complication occurs due to the disruption of associated lymphatic during the exposure of the iliac vessels.

urinary leakage. Urinoma

Immune-suppression induced complications e.g. infections and malignancy

Graft rejection:

1- Hyper-acute (intra-operative)

Happens minutes after transplant, and it is related to the preformed antibody or ABO incompatibility; this is rarely seen now due to the very sensitive cross-match tests performed before the transplant.

2- Acute rejection (within two months)

This can happen any time after transplant, usually within days to weeks after transplant. It classifies into the following:

A) Antibody-mediated rejection-:

Which usually demonstrates evidence of circulating donor-specific alloantibodies and immunological evidence of antibody-mediated injuries to the kidney. Like inflammation of glomeruli (glomerulitis) or peritubular capillaries (peritubular capillaritis).

B) Acute T-cell mediated rejection:

Which is characterized by lymphocytic infiltration of the tubules, interstitium, and sometimes the arterial intima

3- **Chronic rejection**.

It usually develops more than three months post-transplant. It can either be chronic antibody-mediated rejection or chronic T cell-mediated rejection

.

UROLOGICAL EMERGENCIES

Urological Emergencies

Obstructive Uropathy

- Acute urinary retention (AUR)
- Visible Haematuria with Clot Retention

Fournier's Gangrene

Urogenital trauma

- Renal trauma
- Ureteric trauma
- Testicular Trauma
- Bladder Rupture
- Urethral Injury

Andrological emergencies

- Priapism
- Paraphimosis
- Testicular torsion

Obstructive Uropathy

Lower Urinary Tract Obstruction

1- Acute urinary retention (AUR)

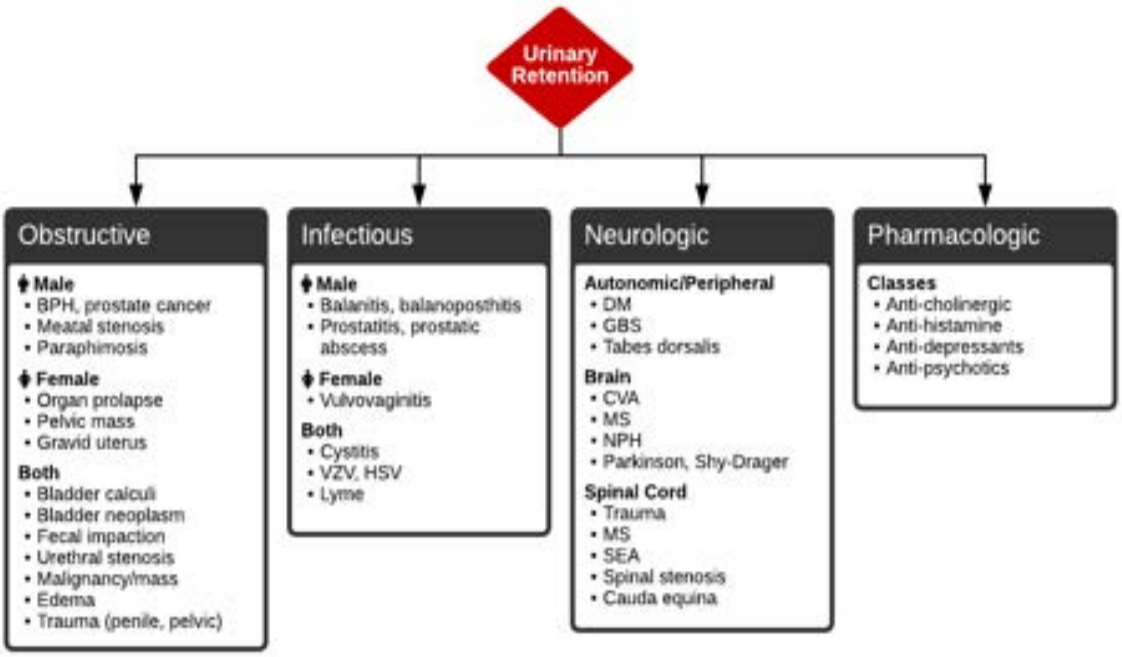
Definition
Involuntary inability to pass urine from the bladder

Epidemiology
AUR is the most common form of urological emergencies. Up to 10% of men in their 70s and 30% of men in their 80s may attend emergency department with at least 1 episode of AUR.

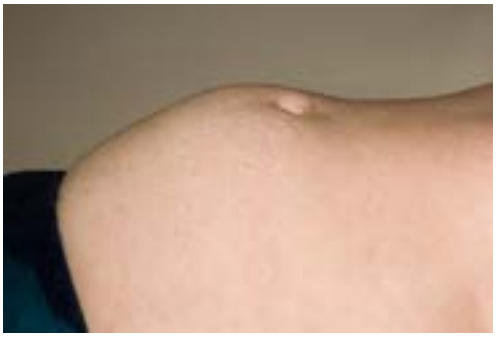
Aetiology

- Bladder Outlet Obstruction
  Obstructed urine flow may occur at the level of bladder neck, urethra, external urethral meatus, or foreskin. Causes of urine outflow obstruction can be stones, tumours, blood clots, benign prostatic enlargement (BPE), or phimosis.

- Bladder Under-contractility
  - Neurologic conditions
  - Drugs: antimuscarinics (decrease bladder detrusor muscle contractility) or sympathomimetic (insufficient relaxation of muscle fibres in the bladder neck).
  - Surgery: AUR is very common in postoperative patients due to regional anaesthesia, narcotic pain medications, constipation, and limited mobility.

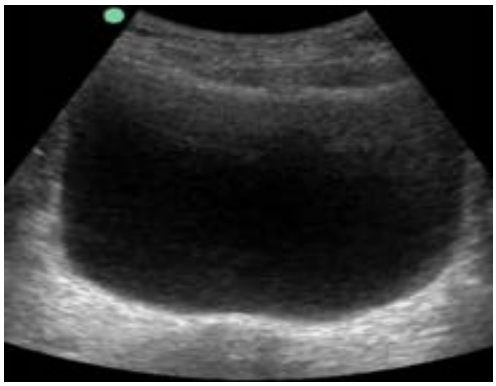


Clinical Presentation



Patients with AUR present with significant lower abdominal or suprapubic discomfort, local examination might reveal a palpable bladder. Local examination might reveal palpable stone in penile urethra, external meatal stenosis, phimosis, and digital rectal examination might reveal BPE.

Investigations



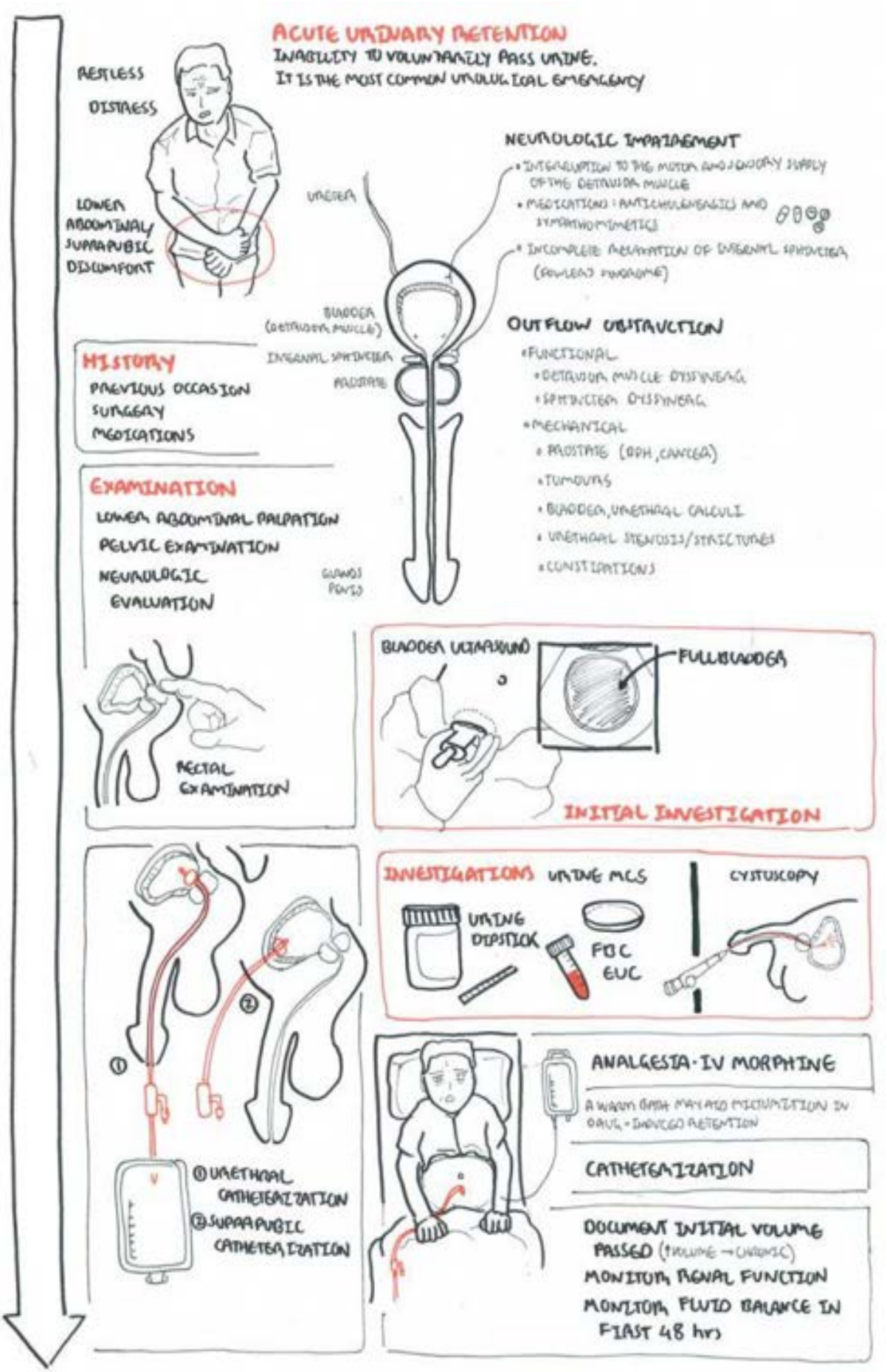
Bladder ultrasound scan will reveal a large volume of urine in the bladder (normal bladder capacity is 400-600 ml).

Treatment

Decompression of the bladder by insertion of a urethral catheter or suprapubic catheter placement if urethral catheter insertion is unsuccessful.

Monitor for post obstructive diuresis post-obstructive diuresis (urine output more than 200 ml/hour for two consecutive hours, or more than 3000 ml/24 hours) to avoid severe electrolyte disturbance.

If the cause is BPE, commence the patient on alpha blockers and arrange for a trial without catheter in 2 to 7 days, if unsuccessful, refer to urologist to council the patient about bladder outlet de-obstruction surgery e.g., transurethral resection of prostate (TURP).


ACUTE URINARY RETENTION
INABILITY TO VOLUNTARILY PASS URINE.
IT IS THE MOST COMMON UROLOGICAL EMERGENCY
RESTLESS
DISTRESS
LOWER ABDOMINAL/ SUPRAPUBIC DISCOMFORT
URETER
BLADDER (DETRUSOR MUSCLE)
INTERNAL SPHINCTER
PROSTATE
NEUROLOGIC IMPAIRMENT
• INTERRUPTION TO THE MOTOR AND/OR SENSORY SUPPLY OF THE DETRUSOR MUSCLE
• MEDICATIONS: ANTICHOLINERGICS AND SYMPATHOMIMETICS
• INCOMPLETE RELAXATION OF INTERNAL SPHINCTER (FOWLER) SYNDROME
OUTFLOW OBSTRUCTION
•FUNCTIONAL
•DETRUSOR MUSCLE DYSFUNCTION
•SPHINCTER DYSFUNCTION
•MECHANICAL
•PROSTATE (BPH, CANCER)
•TUMOURS
•BLADDER, URETHRAL CALCULI
•URETHRAL STENOSIS/STRICTURES
•CONSTIPATION
HISTORY
PREVIOUS OCCASION
SURGERY
MEDICATIONS
EXAMINATION
LOWER ABDOMINAL PALPATION
PELVIC EXAMINATION
NEUROLOGIC EVALUATION
GLANDS PENIS
RECTAL EXAMINATION
BLADDER ULTRASOUND
FULL BLADDER
INITIAL INVESTIGATION
INVESTIGATIONS
URINE MCS
CYSTOSCOPY
URINE DIPSTICK
FBC
EUC
① URETHRAL CATHETERIZATION
② SUPRAPUBIC CATHETERIZATION
ANALGESIA - IV MORPHINE
A WARM BATH MAY AID MICTURITION IN MILD - MODERATE RETENTION
CATHETERIZATION
DOCUMENT INITIAL VOLUME PASSED (↑VOLUME → DIURESIS)
MONITOR RENAL FUNCTION
MONITOR FLUID BALANCE IN FIRST 48 hrs

2- Visible Haematuria with Clot Retention.

Definition

The presence of visible blood in the urine.

Aetiology

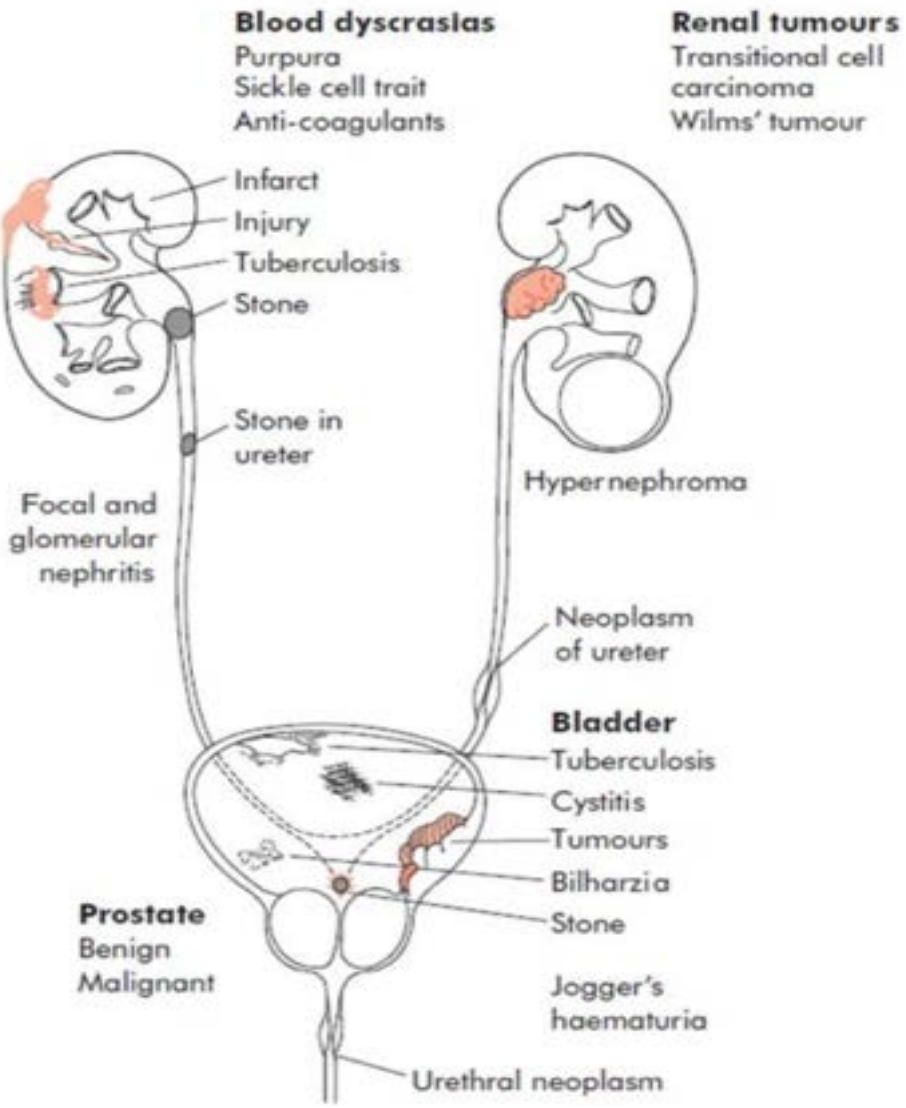
Can be either of renal or urological origin.

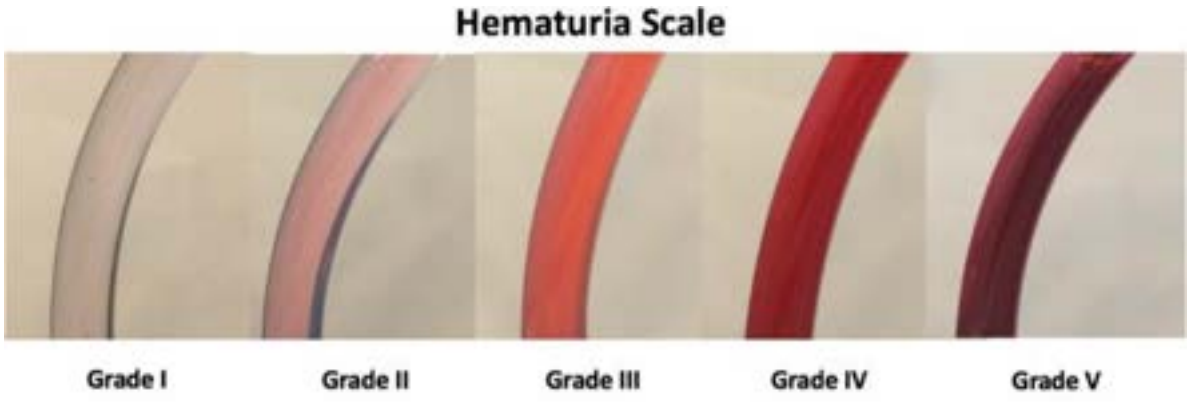
Urological causes include urinary tract malignancy, BPE, trauma, urinary tract stones, or secondary to surgical trauma (iatrogenic).

Differential diagnosis

Other causes of red urine: myoglobinuria, haemoglobinuria, and drugs (e.g., rifampin) or food (e.g. beets).

Severe visible haematuria might be associated with clot formation which might block the bladder neck or the urethra causing what is known as clot retention.





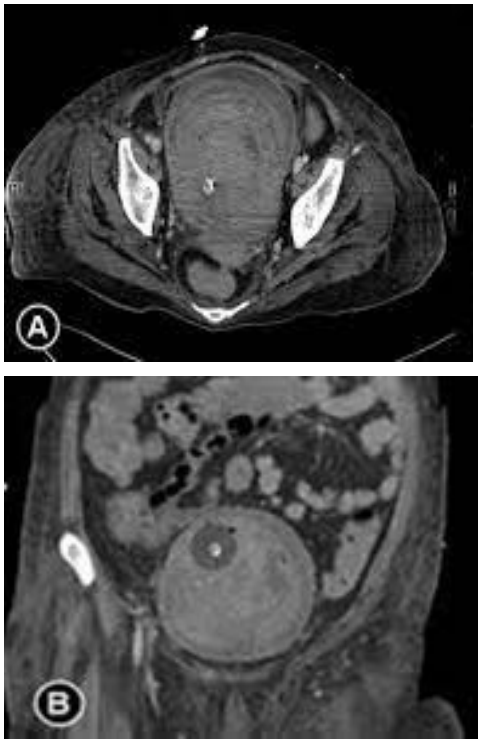
Treatment

Ideally patients with visible haematuria who are still **able to empty their bladder** properly require no catheter insertion, but underlying coagulopathy should be ruled out and treated, and anticoagulants/antiplatelets drugs should be withheld until the haematuria is clear.

Urgent outpatient investigations in the form of diagnostic cystoscopy and CT urogram should be arranged to rule out urinary tract malignancy.

Patients who develop clot retention will require insertion of 3-way 22 Fr urethral catheter, bladder washout with a catheter-tip 60 ml syringe and irrigation with 0.9% normal saline.

If these measures are insufficient, cystoscopy, clot evacuation and stopping of any active bleeding under anaesthesia is indicated especially if the patient's blood haemoglobin level drops and becomes haemodynamically unstable.



CT scan showing organised blood clots inside the urinary bladder

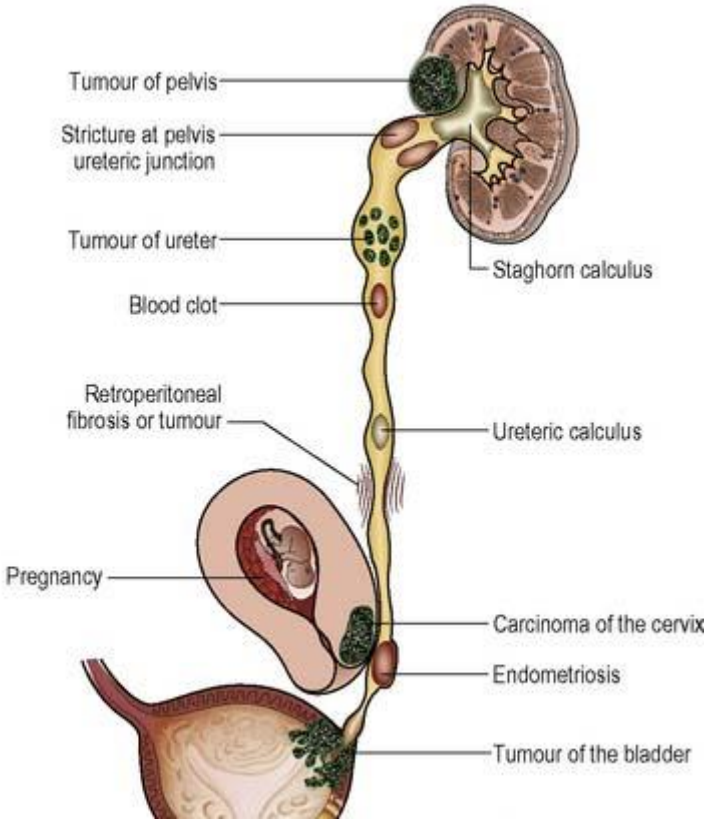
Three-ways catheter used in evacuation of clot retention

Ureteric Obstruction

Causes

Intrinsic: stones (most common cause), ureteropelvic junction obstruction (UPJ-O), ureteric tumours, blood clots.

Extrinsic: compressive blood vessels, extra-mural tumours, or retroperitoneal fibrosis.



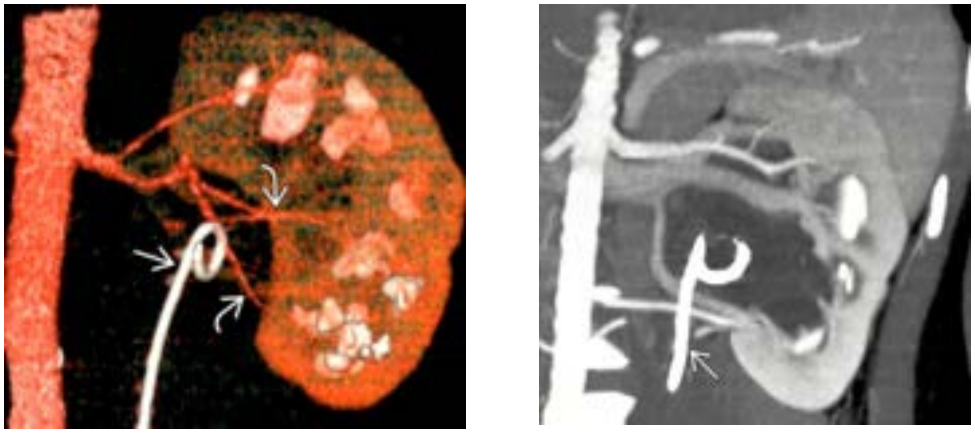
Clinical presentation

History of acute flank pain with or without fever, visible haematuria. Sometimes with oliguria or anuria in patients with bilateral ureteric obstruction or obstructed solitary kidney.

Investigations

Urgent inpatient **non-contrast low dose CT urinary tract** is Mandatory to rule out urinary tract stones and hydronephrosis.

Blood tests may reveal raised inflammatory markers including white cell count (WCC) and C-reactive protein (CRP), deranged overall renal function in the form of **raised serum creatinine** and decline in estimated-glomerular filtration rate (e-GFR). Urine culture and sensitivities (C&S), and blood C&S in patients with fever or suspected uro-sepsis.



Ureteropelvic junction obstruction secondary to aberrant vessel with stent in place

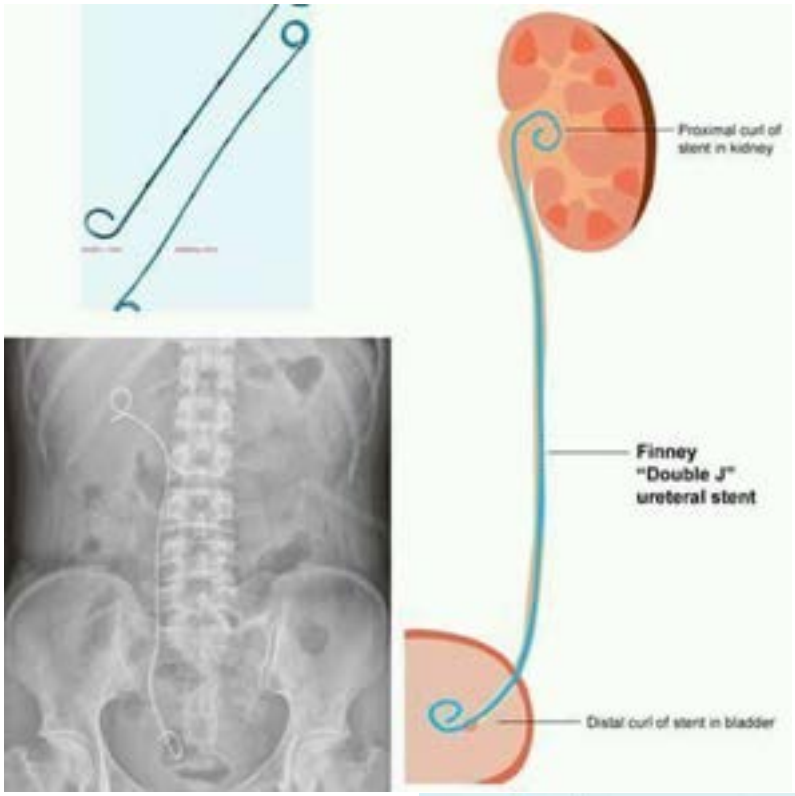
Treatment

Urgent intervention is mandatory in patients **with**

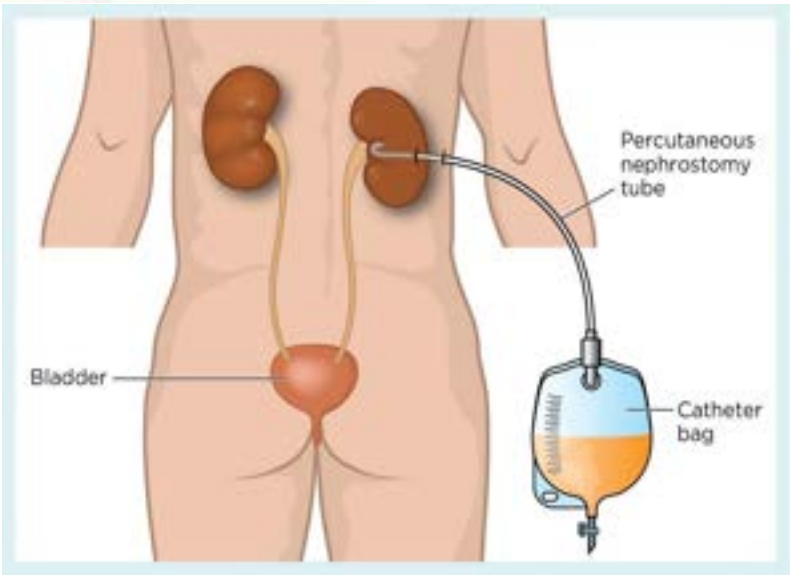
- Bilateral ureteric obstruction,
- Solitary functioning kidney,
- Unilateral ureteric obstruction with severe deterioration in overall renal function, signs of uro-sepsis, or patients with uncontrollable pain.

Intervention will be in the form of

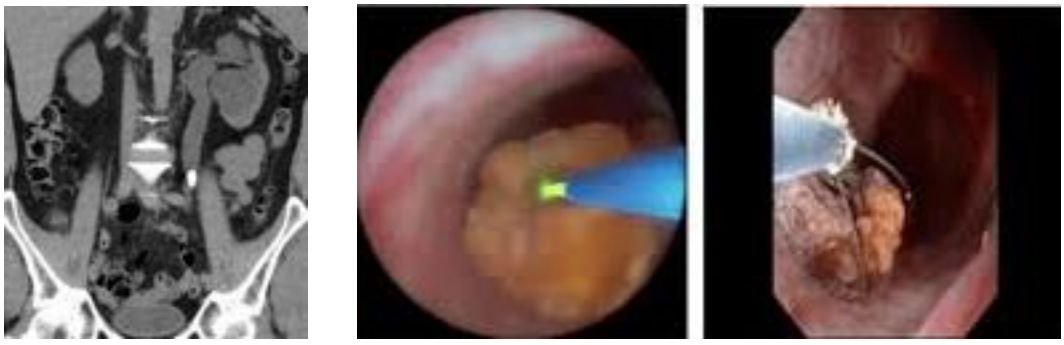
- Urgent cystoscopy and ureteric stent insertion to bypass the cause of obstruction.
- In case of sepsis, percutaneous nephrostomy tube insertion direct tot the renal unit is more appropriate.
- Culture of the urine proximal to the obstruction should be performed once drainage has been achieved, and antibiotic regimen tailored to the culture and sensitivity results.
- In pregnant women with obstructed bilateral renal units, solitary renal unit, uro-sepsis, percutaneous nephrostomy tube insertion should be considered.
- In septic patients, IV antibiotics should be commenced empirically until results of urine and blood C&S are available.
- Once patient improves, further surgical intervention should be planned electively based on underlying aetiology e.g., ureteroscopic stone removal in case of obstructive ureteric stones.



DJ stent



Nephrostomy tube



Laser treatment and stone removal of ureteric stones

Fournier's Gangrene

Definition

A life-threatening necrotizing fasciitis of the soft tissue of the scrotum and perineum.

Epidemiology: Prevalence of 1.6 per 100,000 men per year, usually aged 50 and older with mortality rate in up to 7% of cases.

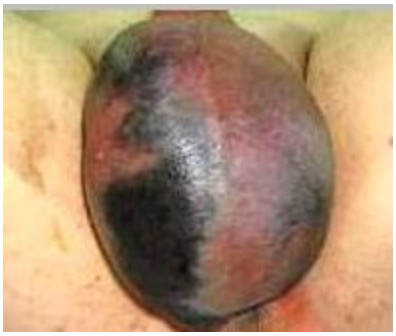
Risk factors: Diabetes mellitus (DM), obesity, immunosuppression, malignancy, smoking, and chronic kidney disease.

Aetiology: Polymicrobial necrotising fasciitis with mixed aerobic and anaerobic bacteria such as *E. coli, enterococci, Klebsiella, or Clostridium.*

Pathogenesis: Necrotizing soft tissue infection spreads along fascial planes causing vascular thrombosis.

Clinical presentation

You must maintain a high index of suspicion for Fournier's gangrene in order to make a prompt diagnosis in the early stages. **Pain** out of proportion to the accompanying physical findings, **localised tenderness** on the scrotum and/or perineum, **skin changes** in the form of bullae, blisters, crepitus, and discoloration. Patients tend to deteriorate dramatically with **signs of sepsis** and septic shock.



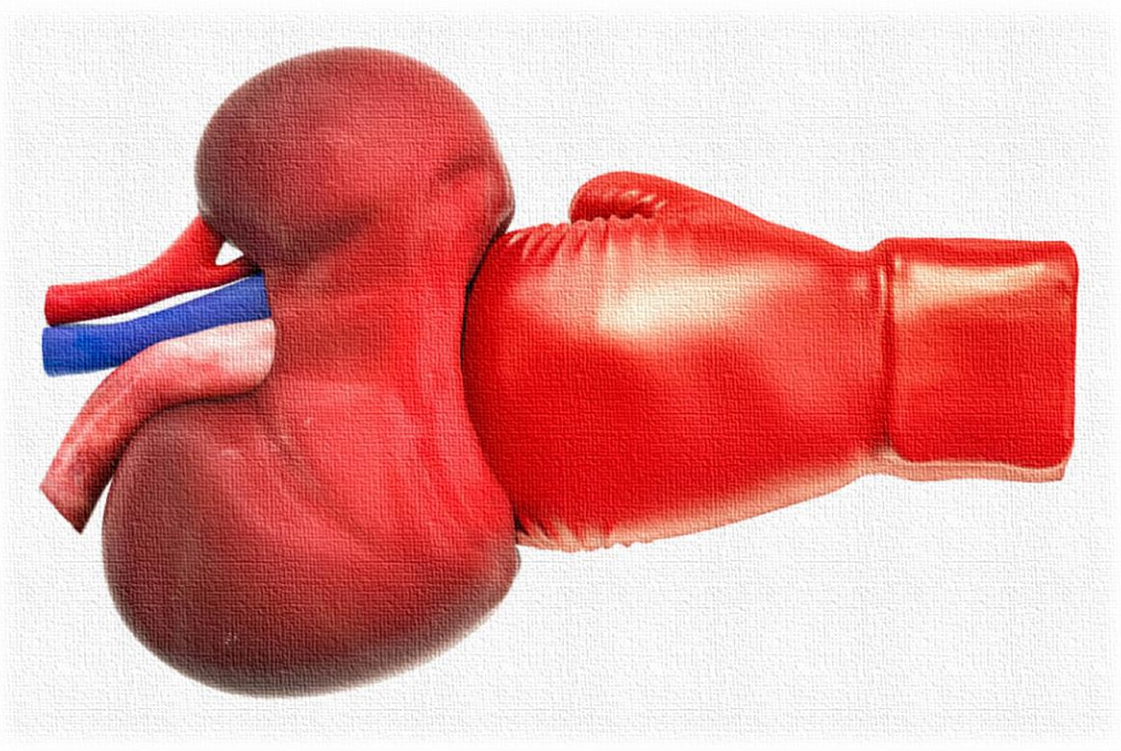
Fournier's gangrene

Treatment

Never try antibiotic only treatment as this is absolutely insufficient and is associated with 100% mortalities.

You must consider immediate aggressive surgical debridement under anaesthesia with hyperbaric oxygen, and IV broad-spectrum IV antibiotics. Patients typically require a redo procedure with additional debridement if necessary within 48 hours and ideally general- and plastic surgery teams need to be involved in the treatment plan as after surviving the initial intervention, patients might require further reconstructive surgery.

UROLOGICAL TRAUMA



1- Rupture of the Urinary Bladder

Epidemiology

Up to 10% of patients with abdominal trauma will have a bladder injury.

Causes

- Iatrogenic: e.g. bladder perforation during a transurethral resection of a bladder tumour (TURBT) and gynaecological procedures
- Direct trauma with intraperitoneal leakage of urine usually occurs at the dome.
- Bladder rupture with extraperitoneal urinary leakage e.g. pelvic fractures.

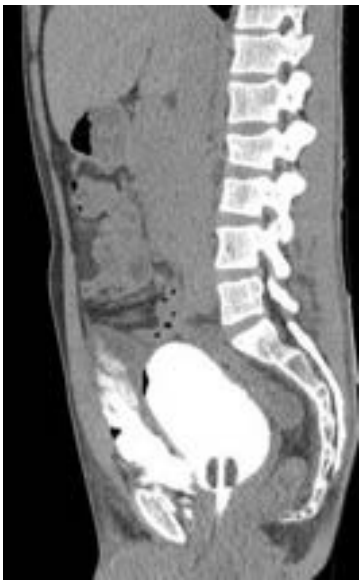
Clinical Picture

History of abdominal trauma or recent surgical procedure, patients may present with visible haematuria, acute urinary retention, inability to void, suprapubic or abdominal pain and tenderness.

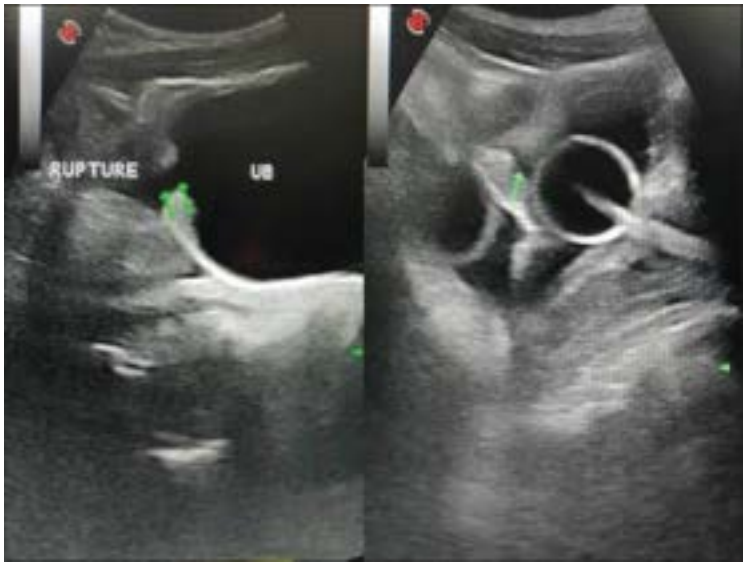
Investigations

Cystogram may confirm and localise the site of bladder rupture and urinary leakage (intra- or extraperitoneal).

Ideally it a catheter is inserted into the bladder unless urethral disruption is suspected, and bladder is filled with up to 300 ml of contrast. Series of images are taken including the moment of voluntary voiding.



CT urogram sagittal view showing extraperitoneal rupture of the bladder



US showing rupture of the bladder

Treatment

Isolated extraperitoneal bladder ruptures can be managed conservatively with urethral catheter drainage **unless** associated with penetrating fragmented pelvic bony fractures, bladder neck or ureteric injury.

Bladder ruptures with intraperitoneal leakage of urine require open surgical repair with thorough assessment of bladder neck and the ureteric orifices, debridement of ischemic tissues and closure of the bladder in watertight layers using absorbable suture and insertion of urethral catheter for 2-3 weeks. Ideally, a cystogram should be done and deemed unremarkable prior to catheter removal.

Urethral Trauma

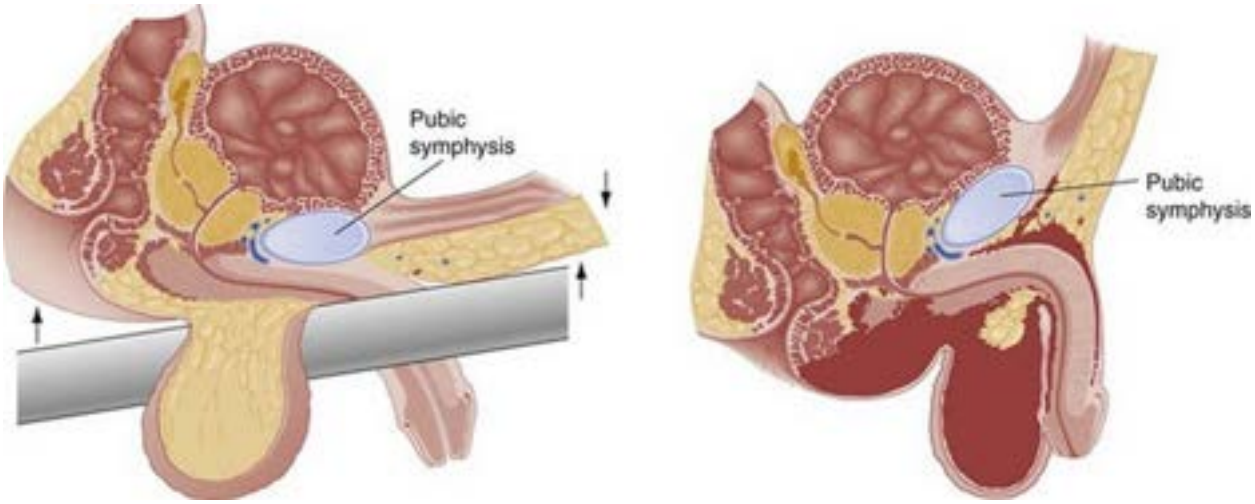
Epidemiology

Urethral injuries are more common in men than in women.

Etiology

Up to 10% of pelvic fracture will have associated posterior urethral injury.

Anterior (penile or bulbar) urethral injuries may occur secondary to or blunt trauma.



Straddle injury

Clinical presentation

Blood at the external urethral meatus in up to 35% of patients, however, its absence does not exclude urethral injury.

AUR, and bruising.

On DRE, **a "high riding" prostate** is suggestive of posterior urethral injury.

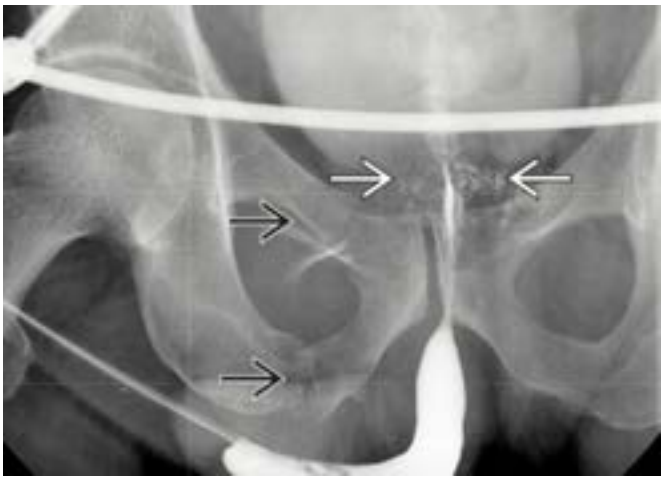
Urethral injury in women might be associated with blood in the vagina or labial bruising.

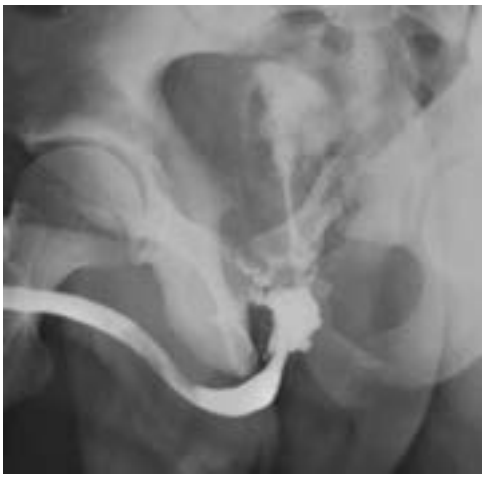
If Buck's fascia is ruptured, urinary leakage might result in a typical pattern of swelling and bruising called **"Butterfly wing"**

Investigations

Retrograde urethra-cystogram

Contrast is gently injected into the urethra; partial urethral injury can be evident by contrast leakage. If contrast doesn't reach the bladder, a complete urethral disruption is suspected.





Posterior urethral trauma

Treatment

Anterior urethral injuries: primary surgical repair with absorbable tension-free sutures and catheter drainage, debridement of potentially ischemic tissues.

Posterior urethral injuries should not undergo primary repair due to increased risk of erectile and urinary complications. Historically, if blood is seen at external meatus urologist tend not to attempt urethral catheter insertion as this carries the risk of converting partial urethral injury to complete urethral rupture.

Ureteric Trauma

Causes

External trauma (penetrating, blunt)

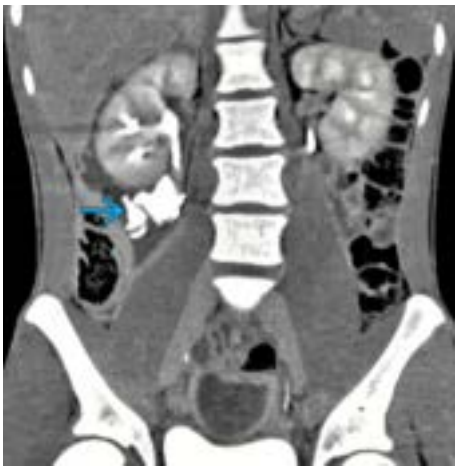
Iatrogenic injury (urological, general surgical or **gynecological** procedures)

Clinical presentation

If overlooked intra-operatively, ureteric injury may present late on the following days postoperatively with ileus, prolonged postoperative pyrexia, and persistent urinary leakage from drains, loin pain (suspecting ureteric ligation)

Investigation

CT urogram with delayed phases imaging. Or on table retrograde uretero-pyelogram



CT urogram showing proximal ureteric trauma with extravasation of the contrast

Treatment

Primary repair with absorbable sutures and stenting for 3-6 weeks should always be considered unless the field is infected, then nephrostomy tube insertion with delayed repair can be considered.

Distal ureteric injuries should be repaired with a primary re-implantation.

Mid-ureteric injuries can be repaired with a uretero-ureterostomy if possible or alternatively Boari flap, and psoas hitch can be considered.

Occasionally, Auto-transplantation of the kidney to the pelvis or bowel interposition can be considered in delayed repair.

Renal Trauma

Epidemiology:

More common in men than women. Up to 10% of cases of abdominal trauma are associated with renal trauma.

Etiology:

Blunt traumas: e.g. Motor car and m accidents and falls.

Penetrating trauma: e.g., gunshot or stab wound.

Clinical presentation:

History of trauma (blunt or penetrating) involving the kidneys, bruising in flank, occasionally visible hematuria, and can be associated with rib fracture.

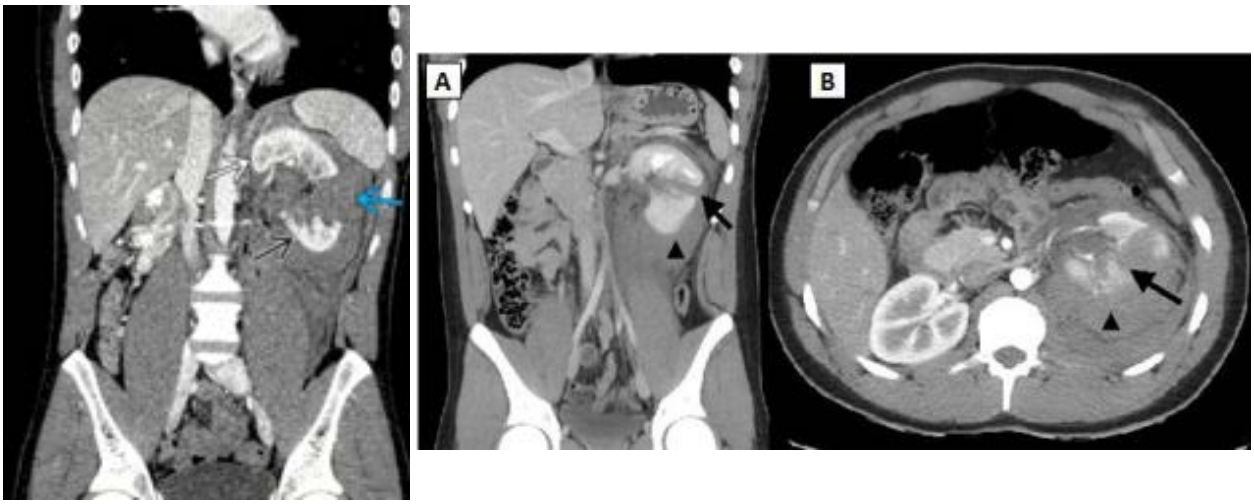
Investigations

Indications for imaging in patients with renal trauma*:*

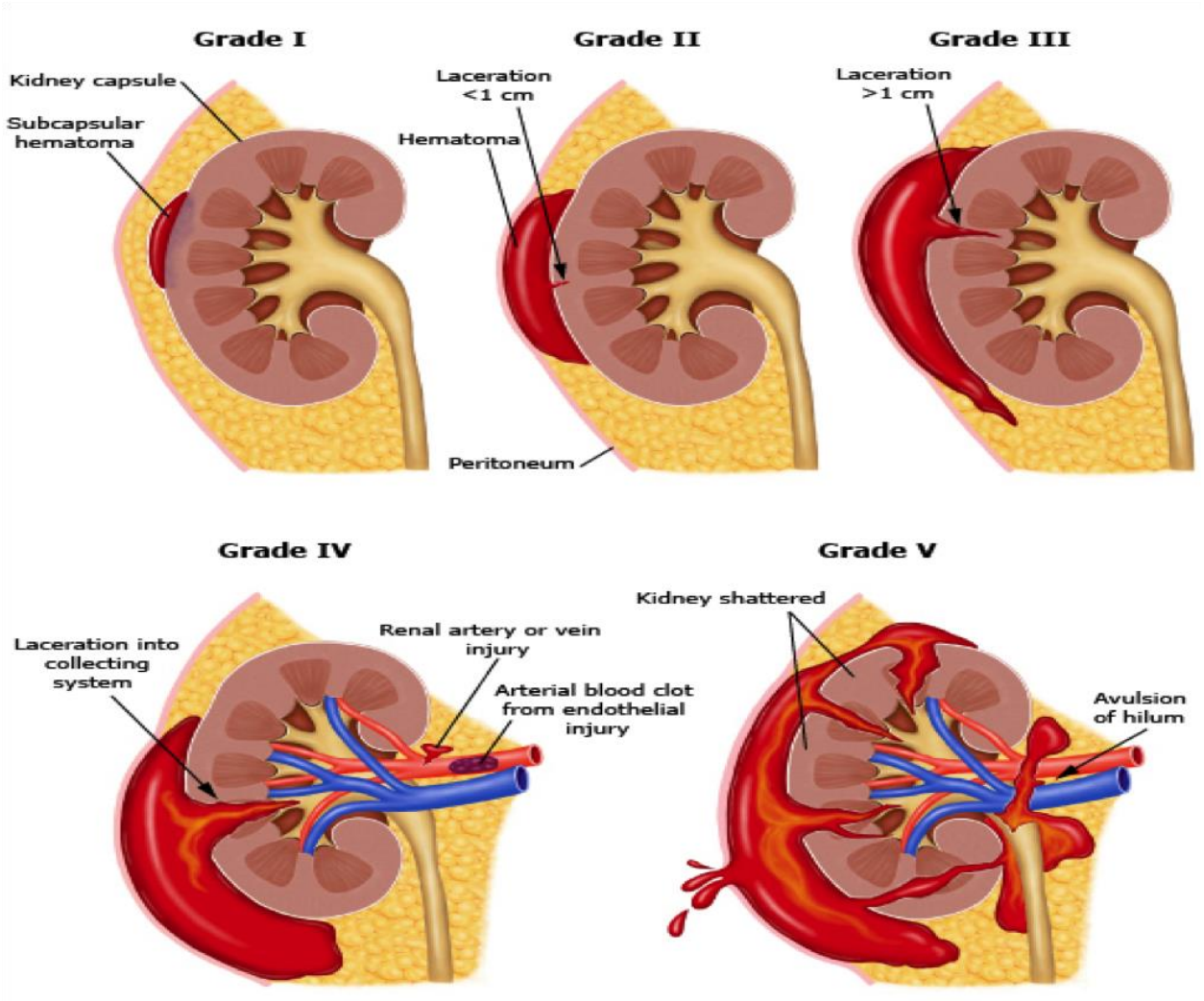
- *Visible haematuria*
- *Penetrating trauma to chest/abdomen*
- *Microscopic haematuria with systolic blood pressure below 90 mmHg*
- *Paediatric renal trauma with microscopic or visible haematuria*
- *On-table IVU in patients taken direct to theatre to control bleeding without CT urogram especially of exploration revealed retroperitoneal haematoma or renal injury requiring nephrectomy to confirm presence of normal contralateral renal unit.*

Renal injuries can be classified radiologically as follows:

- Grade I Contusion or Sub capsular hematoma
- Grade II <1cm parenchymal laceration without extravasation
- Grade III >1cm laceration without extravasation
- Grade IV Laceration into collecting system (extravasation)
- Grade V Shattered kidney, avulsion of renal pedicle



Ct urogram showing severe left renal trauma



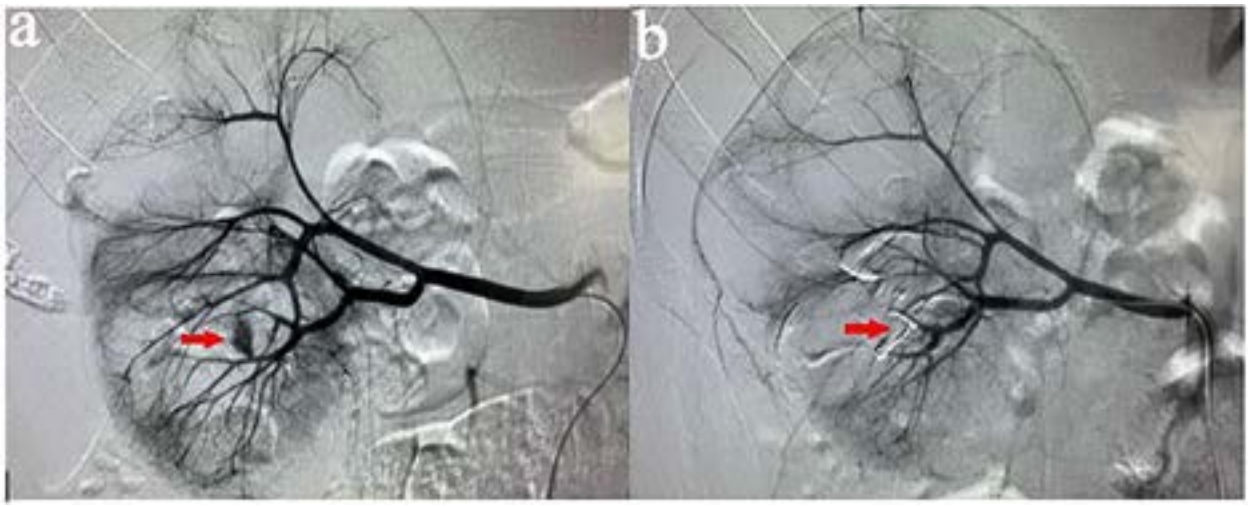
Grading system

Treatment

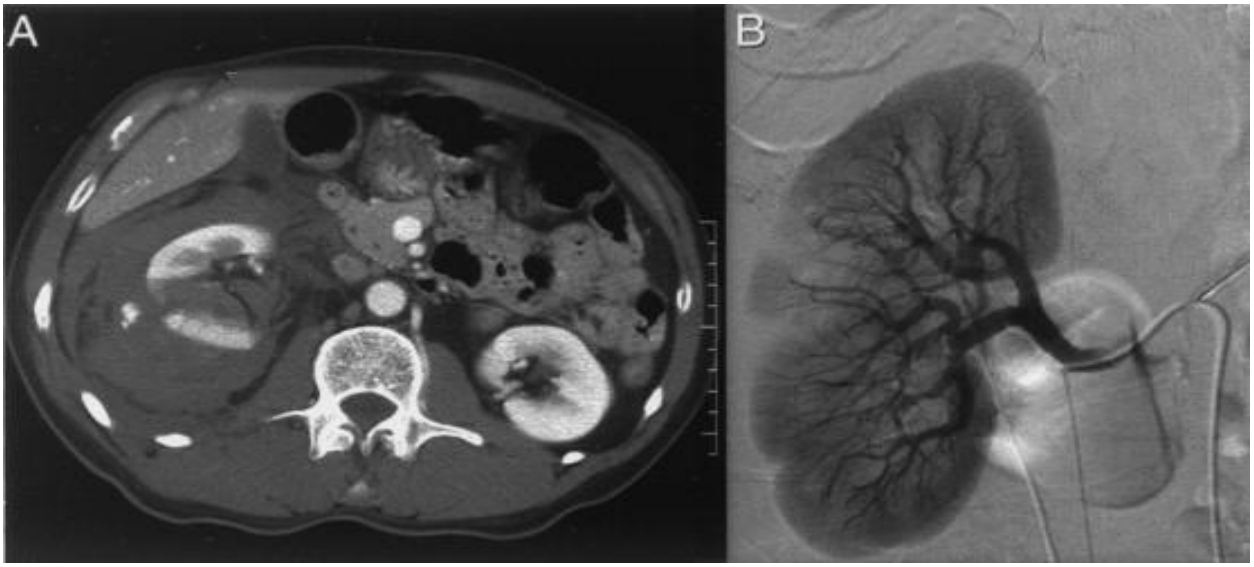
Up to 95% of blunt renal traumas can be managed conservatively. Haemodynamically stable patients can be treated conservatively in the form of bedrest, serial blood hb measurements, and close monitoring of vital signs.

Hemodynamically unstable patients require immediate intervention in the form of:

1- Angioembolization especially if bleeding is from a segmental renal artery.
2- Surgical exploration



Angioembolization (a-pre / b-post)



Testicular Trauma

Causes

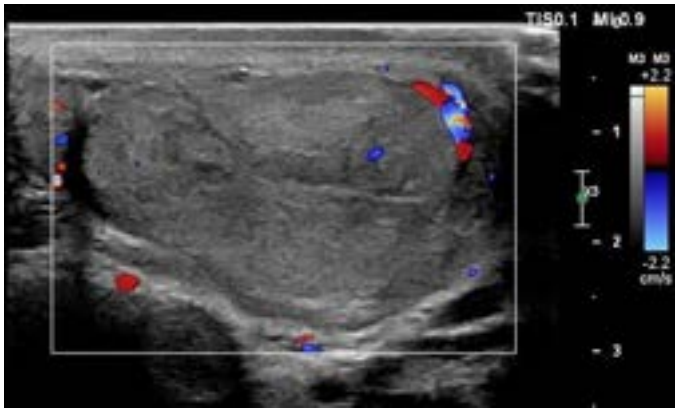
Penetration or blunt trauma.

Clinical presentation

Scrotal tenderness and haematoma that can extend into the spermatic cord and inguinal region.

Investigations

Scrotal ultrasound might reveal large haematocele (blood collection around the testis) or disruption of the tunica albuginea.



Doppler ultrasound scan showing testicular rupture

Treatment

Timely diagnosis and surgical repair of significant testicular injuries (disrupted tunica albuginea, large haematocele) is imperative to avoid remote organ function loss in terms of spermatogenesis and hormone production.

REFERENCES

The Egyptian Urological Association

European Association of Urology

American Urological Association