بسم الله الرحمن الرحيم

**This file contains the following topics:

- 1) Neurogenic bladder
- 2) Urologic trauma
- 3) UTI
- 4) Urolithiasis
- 5) Pediatric urology
- 6) Renal cancer

- 7) Bladder cancer
- 8) BPH
- 9) Prostate cancer
- 10) Benign scrotal swelling
- 11) Erectile dysfunction
- 12) Infertility

**Missed topics:

- 1- Introduction (Signs & Symptoms)
- 2- Urinary Catheter
- 3- Testicular cancer



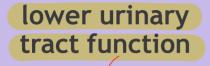


- Neurogenic bladder is a term used to describe lower urinary tract (LUT) dysfunction resulting from a neurologic disease or process. result voiding dysfunction
- Up to 80% of neurological diseases develop Lower Urinary Tract Symptoms (LUTS) - frequency/ergency/urge incontinence/poor stream
- Accurate diagnosis and proper management of LUT dysfunction in the neurogenic population consist of two main goals: (1) to preserve the safety of the bladder with lowpressure storage and adequate emptying and (2) to maintain a reasonable quality of life in relationship to the bladder. Pt with shock & frequency

≠ inadequate emptying result in result unin -> 1 p. in bladder -> back p. on Kidney i we treat manistration of neurological d. on lower uninary tract -> to preserve low storage p. & adequate emptying to preserve upper tract

female " obese & bedridden + " - (wet genitalia 2

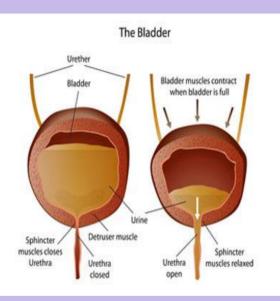
develops users/



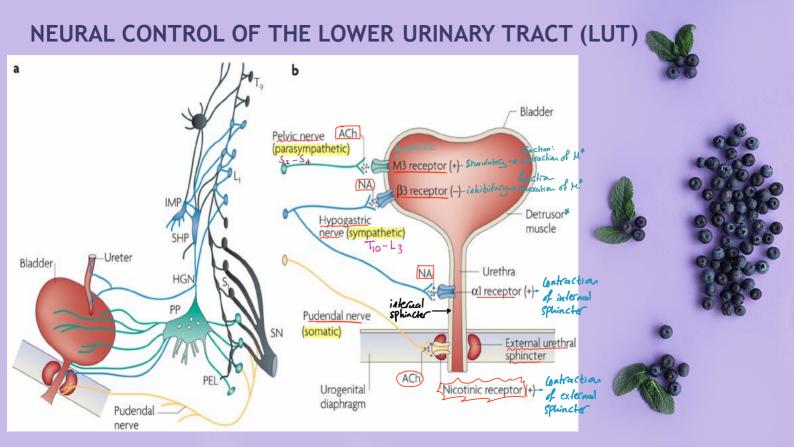
urine storage and micturition <u>depend on</u> the <u>coordination of</u> the bladder, <u>bladder neck</u> (sphincter) , <u>urethra</u> and urethral sphincter <u>bladder</u> M. (<u>detruer</u> M.)

coordination between the muscles of the lower urinary tract is mediated by neural pathway in the ; brain spinal cord

peripheral nerve -PNS





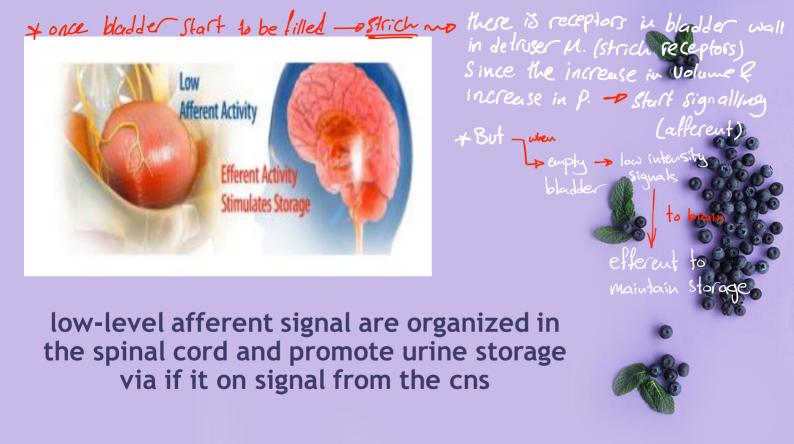




Phase	Storage phase	Voiding phase
Parasympathetic	Off	On
Sympathetic	On	Off
Somatic	On	Off

- Pelvic splanchic nerve (Parasympathetic) signals lead to contraction of the Detrusor (M3 receptors = Stimulatory).
- Hypogastric nerve (Sympathetic) signals lead to Relaxation of the Detrusor (B3 receptors = Inhibitory) and contraction of Internal Urethral sphincter (A1a receptors = Stimulatory).
- Puodendal nerve (Somatic) signals lead to Contraction of the external urethral sphincter (Nicotinic receptors = Stimulatory)

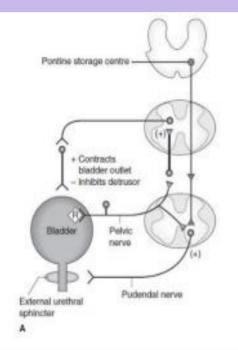




urine storage reflux

 During normal bladder filling, sympathetic (hypogastric nerve) and somatic (pudendal nerve) neural mechanisms mediate the contraction of the internal smooth and external striated urethral sphincters, respectively. As the bladder fills, sympathetic-mediated inhibition of the detrusor allows for the bladder to accommodate increasing volumes at low intravesical pressures

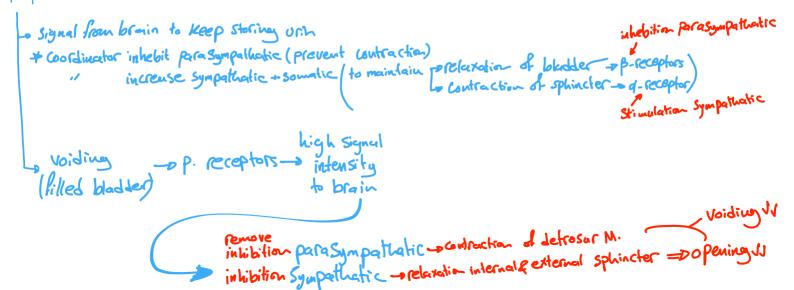
so this reflects is *on* when we have " low intensity signal and ssocially causes"





- # Brain micturation center in frontal loop
- # low signal OR situation not sutable for urination (socially unsutable) ---> brain send signals for pontine (the coordinator)

* pontive (micharation center inside it)



tawy problem in periphral N. or Jesion sacral or infrasacral lesion · flaccid bladder (without sensation or contraction - bugp contractility) · increase residual Urin · symp. - con't emptying bladder · mainly -o voiding Symp.

Spinal Cord or intrapontine Suprasacial esian · loss of storage function · storage symp. - trequency, urgency, urge incontinence, nocturia · Coordination affected; so present with both storage & Voiding Symp. - Poor Stream intermetancy, inability complete emptying increased residual volume disorganization, dissynergia blu 2 splicters in addition to detrosur overactivity

center (brain) subrapontine lesion

· loss of storage function · storage symp. - frequency, urgency urge incontinence, nocturia · solitates some (a

• Sphincter <u>Spare</u> (synergia preseved) so the emptying will be completly <u>so no residual urin</u>

· function : sphincter normal detrosur overactivity

N. & sphincter one of them contracted the other relaxed



high level afferent signal are stimulated by intravesical pressure resulting in increased activity to the brain

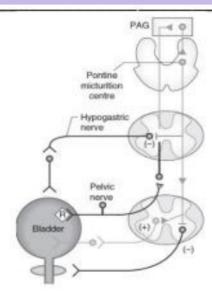
descending efferent pathway then cause voluntary bladder contraction and the flow of urine

normal voiding is. therefore, a function of a positive feedback mechanism



Urine voiding reflex A positive feedback loop

 When an individual decides that it is time to urinate, the pontine micturition center (PMC) is released from tonic inhibitory control of the higher cortical and subcortical centers of the brain, initiating the voiding process. The parasympathetic system then switches "on," stimulating a detrusor contraction and relaxation of the pelvic floor and external and internal urethral sphincters





Nervous system damage and LUT dysfunction



Suprapontine lesion

- · History: predominantly storage symptoms
- · Ultrasound: insignificant PVR urine volume
- · Urodynamics: detrusor overactivity
- Upper motor neuron



Over-

active

Each filling episode of bladder will leave to activation of the pontine center.

Storage symptoms : Frequency , Urgency & Nocturia.

Spinal (infrapontine-suprasacral) lesion

- · History: both storage and voiding symptoms
- · Ultrasound: PVR urine volume usually raised
- Urodynamics: detrusor overactivity, detrusorsphincter dyssynergia

Upper motor neuron

Pontine center isn't receiving signals from below.

Sacral/infrasacral lesion

- · History: predominantly voiding symptoms
- · Ultrasound: PVR urine volume raised
- Urodynamics: hypocontractile or acontractile detrusor

Lower motor neuron Weak bladder. Underactive • • • • • • • Voiding symptoms : Weak stream , Retention , Hesitancy & Dribbling.





History and Physical Examination *Spinel Shock - 1st Smonths - manifistation of LMNL (infrasocrat manifistation)

- A detailed history should factor in urinary tract symptoms, <u>neurologic symptoms</u> and diagnosis (if known), the clinical course of the neurologic disease, <u>bowel symptoms</u>, <u>sexual function</u>, comorbidities, and use of prescription and other medication and therapies. to have sympathetic or parasympathetic SE therapies.
- assessment of patient mobility, hand function, cognitive function and social support are also important respine and inder & para plasa in addition to dissynergial he can't empty his bladder doveractivity (back
- Other factors to consider are risk and history of urinary tract infections, decubitus ulcers, and other urologic factors that may contribute to LUT dysfunction such as prostate enlargement in men and urethral hypermobility in women. Superimposed viologic symp. -> Complications.

=> if the patient is incapacitated -> induelling Catheter inserted

dean intermittent ale a

History and Physical Examination

A general physical examination should include blood pressure measurement, an abdominal examination, an external genitalia examination in males and a vaginal examination if clinically indicated to look for pelvic floor prolapse in women along with a rectal exam to look for fecal loading or alteration in anal tone

. A focused neurological examination is also recommended. This may include assessment of cognitive function, ambulation and mobility, hand function, and lumbar and spinal segment function, including testing sensation and reflexes in the urogenital area.

Investigations



A. Urine Testing - residual urin Stasis, infection
 B. Measurement of Renal Function - high p. system in bladder - black p. to kidney
 C. Upper Tract Evaluation - stasis(stone formation)
 D. Urodynamic Investigations
 F. Lugarante
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MANAGEMENT OF NEUROGENIC BLADDER

مروة الخرشة : Presented by

Goals of Accurate diagnosis and proper management of LUT dysfunction





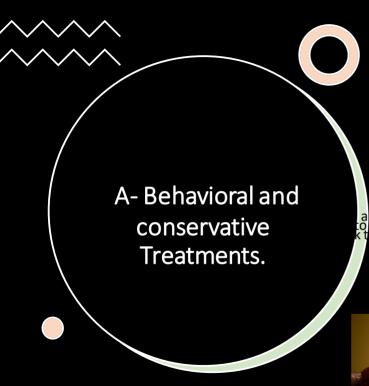
(1) to preserve the safety of the bladder with low pressure storage and adequate emptying.

(2) to maintain a reasonable quality of life in relationship to the bladder.

Management of neurogenic bladder

- 1.Management of Storage Dysfunction:
- A-Behaveioral and conservative Treatments.
 - **B-** Pharmacotherapy.
 - **C-Neuromodulation.**
 - **D- Onabotulinumtoxin A**
 - **E- Surgical.**

- 2-Management of voiding dysfunction:
- A- Medications.
- B- Catheterization.



• Lifestyle interventions

1. Moderation of fluid intake to 1-1.5 litres per day

2. Avoid alcohol, caffeine

3. Drug regimens avoiding diuretics,

4. control of chronic cough and constipation ,

5. cessation of smoking .

S. exclusion or treatment of urinary tract infection ,

7. weight reduction is desirable .



Behavioral therapy

1. Timed voiding

2. Pelvic floor muscle training and exercise (including pelvic floor relaxation).

3. Delayed voiding.

4. Double voiding.

5. Biofeedback

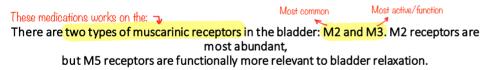


B. Pharmacotherapy TWO LINE OF MEDICATIONS [] Anticholinergic drugs (Antimuscarinic)

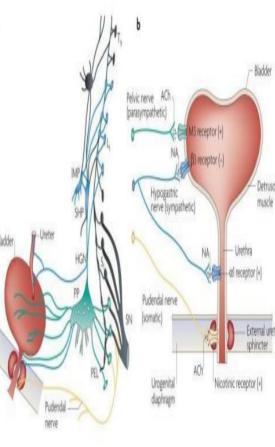
*To inhibit detrosur contraction (inhibit parasympathatic)

Anticholinergic medications are the mainstay of pharmacotherapy for individuals with neurogenic detrusor Overactivity (level of evidence la) and are considered to be first-line therapy, at times in combination with clean intermittent catheterization (CIC).

Anticholinergic medications aim to increase bladder capacity and reduce episodes of urinan incontinence secondary to neurogenic detrusor Overactivity.







Adverse effects of anticholinergic medications most commonly

include dry mouth, blurred vision, constipation, tachycardia, and confusion, some or all of which may already be present in the neurogenic patient.

#Affect contractility of bladder "SE

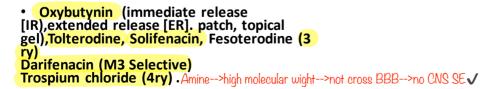
Difficulty emptying the bladder is another potential adverse

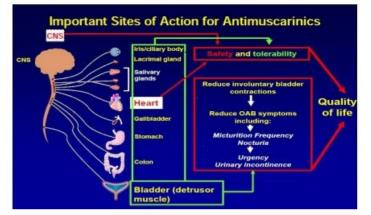
event of these medications, which should be considered in any individual who has an elevated PVR and in individuals with

Individual who has an <u>elevated PVR</u> and in individuals with multiple sclerosis, stroke, or Parkinson's discase.

It is also recommended to monitor PVRS after Starting treatment with an anticholinergic medication and to take into account that these medications can cross the blood-brain barrier, can reduce bladder emptying increasing the risk for uninary tract

infection, and can precipitate or exacerbate constipation.







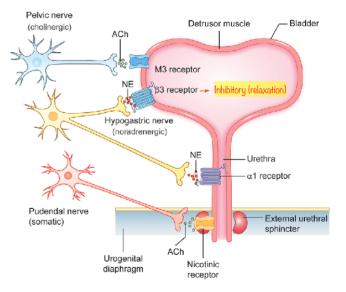
Beta-AR agonists

Over 97% of B-adrenergic receptors in the bladder are of the p3 type, representing the main method of bladder relaxation in humans.

These receptors serve to relax the detrusor muscle, making them an ideal therapeutic target. The selective Stimulate & enhance relaxation of bladder p3-adrenoceptor agonist mirabegron was approved in by the Food and Drug Association (FDA) in 2012 for

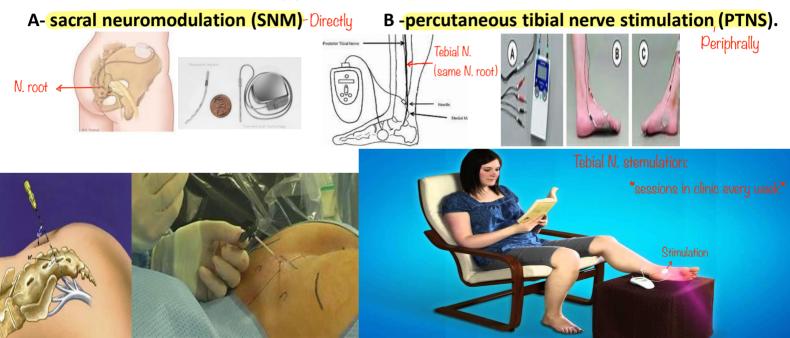
clinical use as an additional medication used to treat overactive bladder symptoms.

The main side effects of this medication are cardiovascular with a mean rise in blood pressure of up to 2.4 mm Hg and small increases in heart rate .> Recommendation to monitor HR & BP



الحل <-- Abnormal afferent signals--> الحل stimulation of N. root--> inhibit exitatory stimuli to bladder (then maintan storage -relaxation of C- Neuromodulation detrosur M.-

 Neuromodulation is a well-established third-line treatment for nonneurogenic overactive bladder, but its use in neurogenic bladder is relatively less established.
 Neuromodulation is currently performed in two varieties:

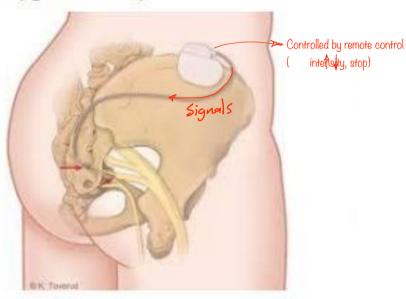


nyperactivity

Sacral neuromodulation -> Battery & lead at normal root position

Stimulation of the S3 / S4 nerve root by an implanted electrical pulse generator

The stimulator is a small electrical pulse generator, approximately the same size as a cardiac pacemaker, and is commonly implanted in the upper outer quadrant of the buttock.



D. Onabotulinumtoxin A

+inhibition of NMJ => relaxation of detrasor M.

• Onabotulinumtoxin A works by blocking the release of acetylcholine from nerve endings (exocytosis), resulting in the blockage of neural transmission and alteration of afferent sensory input. It is important to consider that these injections may increase the PVR among individuals who void, introducing the need for CIC or other more invasive bladder management strategies.

9 through Cystoscope & intradetrosur indection & these toxins later on -=>degradation(efficiency=6-8 months) & they have to repeat this management One vial contains 100 units of Botalinum toxin type A One vial contains 100 units of Botalinum toxin type A* (from Clastratium botalinum)

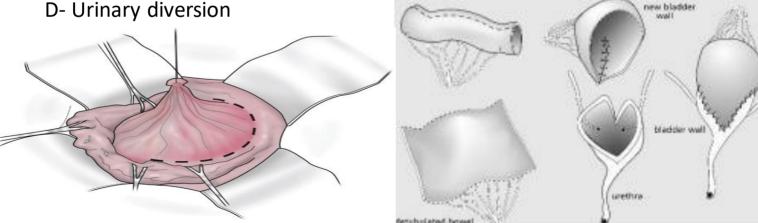
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Allerga

ALLERGAN

• E. Surgical: Spastic bladder & has low volume

- A- Clam augmentation cystoplasty . from ileum
- B- Detrusor myectomy.
- C- Denervation procedures.
- **D-** Urinary diversion



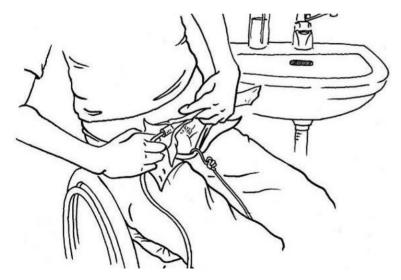
1 Volume L p

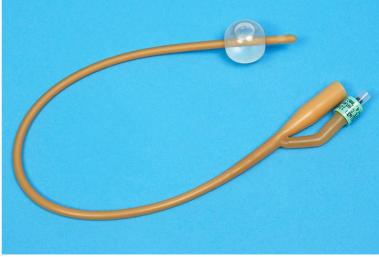
2-management of voiding dysfunction -- High RV

Cholinergomemics -- Medications -- Withdrawn from markets boz their high SE

-Catheterization Indwelling (change every 3 months)--> pt continously draining his bladder

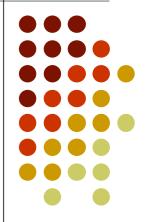
Clean intermittent catheterization (CIC)--> pt can catheterize him/herself at time feeling for voiding







Urological Trauma



introduction



- Urologic trauma is classified according to the location of injury:
- 1- **upper urinary tract** (kidneys and ureters)

2- **lower urinary tract** (bladder and urethra), and **external genitalia** (penis, scrotum, and testes).

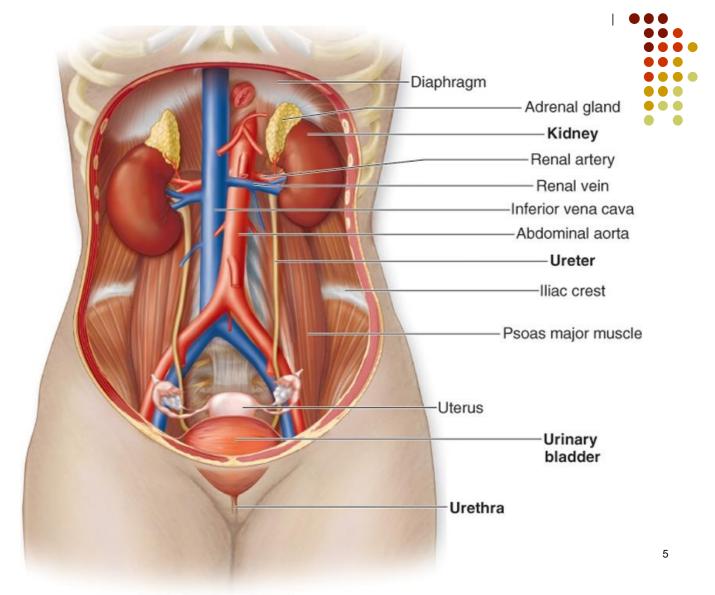


Renal trauma

Very rare -> 1-10% maximum of abdomenal trum -> with renal indury (rare)

Renal Anatomy

- side
- The kidneys are retroperitoneal organs on each side of the vertebral column (T12-L3), at about the level of the twelfth rib.
- The left kidney is lightly higher in the abdomen than the right.
- On an average, each kidney weighs 150 g.
- The kidneys have the following coverings:
- 1. Fibrous capsule
- 2. Perirenal fat
- 3. Renal fascia
- 4. Pararenal fat



Relevant anatomy



- The superior aspect of the kidneys is somewhat protected by the lower ribs.
- the lower poles are inferior to the 12th ribs.
- The parenchyma of the kidney has a segmental arterial supply." Lend arteries, no anastonasis)
 & venouse drainage - venouse drain communication blue all parts of renal paryncheme.
- there is numerous anatomical variations, including pelvic kidneys; horseshoe kidneys; and multiple renal arterial, venous, and ureteral duplications.

Renal trauma



- The kidney is the most commonly injured structure in the urinary tract, accounting for 1% of all traumatic injuries. Nevertheless, operative intervention is pursued in only approximately 10% of renal injuries.
 W. rend intervente concrete.
- The majority of renal injuries are **mild** and can be managed conservatively.

Mode of Injury



- The mechanisms of renal injuries are classified as **blunt** (closed) or penetrating (open).
- Blunt injuries tend to fracture along the planes between the segmental vessels, but penetrating injuries cross the segmental vessels.
- Blunt trauma (90%) can be the result of direct compression or deceleration.
 - Motor vehicle accidents
 - falls
 - direct blows to the flank
 - Deceleration injuries are particularly dangerous because they may lead to renal vascular injuries.



 Penetrating wounds(10%) are ones open to the external environment.

- Gunshots
- Stab wounds
- Renal injuries from penetrating trauma tend to be more severe and less predictable than those from blunt trauma.

Box 10.1 Staging of the renal injury

Using CT, renal injuries are staged according to the American Association for the Surgery of Trauma (AAST) Organ Injury Severity Scale. Higher injury severity scales are associated with poorer outcomes.

- Grade Contusion or subcapsular hematoma with no parenchymal laceration
- Grade II Parenchymal laceration of cortex <<u>1 cm</u> deep, no extravasation of urine (i.e., collecting system intact) (Fig. 10.1)
- Grade III Parenchymal laceration of cortex >1 cm deep, no extravasation of urine (i.e., collecting system intact)
- Grade IV Parenchymal laceration involving cortex, medulla, and collecting system OR segmental renal artery or renal vein injury with contained hemorrhage

Grade V Completely shattered kidney OR avulsion of renal hilum



Figure 10.1 Renal CT with IV contrast in blunt trauma patent shows a ¹¹ superficial (grade 2) laceration amenable to nonoperative management.

Initial emergency assessment



- 1. Initial assessment of the trauma patient should include (ABC) controlling any external bleeding and resuscitation of shock as required, and securing of the airway.
- 2. Take careful history
- 3. Physical examination is usually carried out during stabilization of the patient.
- 4. When renal injury is suspected, **further evaluation** is required for a prompt diagnosis.

+ Usin analysis; h<u>ematuria</u> (most impindicutor of Usinary indusy) full sib fracture, brusing, ecomosis, pulsating hematoma on flank

History and physical examination



- Haemodynamic stability should be decided upon admission
- History should be taken from conscious patients, witnesses, and rescue team personnel regarding the time and setting of the incident.
- Abnormal kidneys are more liable to injury thus, a history of past renal surgery, and known pre-existing renal abnormalities (large cysts, stones) should be recorded.

Pelvic Kidney, horse show kidney, malformations.

 In penetrating wounds Do examination of thorax, abdomen, flanks and back for should be obtained (eg. Bruises, rib fractures...etc)

- The following findings on physical examination may indicate possible renal involvement:
- 1. Haematuria
- 2. Flank pain
- 3. Flank ecchymosis
- 4. Flank abrasions
- 5. Fractured ribs 11th / 12th
- 6. Abdominal distension
- 7. Abdominal mass
- 8. Abdominal tenderness.



Laboratory studies

-Urinanalysis:

 It is the single most important laboratory test in the evaluation of renal injuries.

• Hematuria is an important indicator of injury to the kidney, yet the presence or absence of hematuria should be viewed in the clinical context and not used as the sole decision point in the assessment of a patient with a possible renal laceration.

• **The degree** of hematuria, **does not** precisely correlate with the severity of injury.

Imaging studies



- The goal of radiographic assessment is to provide complete and accurate staging of renal injuries in order to determine the need for and to plan operative management.
- Indications:

 - Gross hematuria
 Microscopic (>5 RBCs per high-powered fi eld [hpf]) or dipstick hematuria in a hypotensive patient (systolic blood pressure of <90 mmHg recorded at any time since the injury) 3 Pediatric with microscopic hematuria even with Stable Vitaks
 - History of rapid deceleration with evidence of multisystem trauma (e..g., fall from a height, high-speed motor vehicle accident).
 - Penetrating chest and abdominal wounds (knives, bullets) with any degree of hematuria or suspicion of renal injury based on wound location



 These criteria do not apply to <u>pediatric</u> trauma patients; in this population, significant renal injury can occur even in the setting of microscopic hematuria without hypotension.

Ultrasonography

- Advantages
- 1. It is noninvasive
- 2. It may be performed in real time in concert with resuscitation
- 3. It may help define the anatomy of the injury.

• Disadvantages

- 1. Optimal study results related to anatomy require an experienced sonographer
- 2. Bladder injuries may be missed.

Computed Tomography



• Advantages:

- 1. It allows unsurpassed functional and anatomic assessment of the kidneys and urinary tract
- 2. It allows for the diagnosis of concurrent injuries.

Disadvantages:

- 1. It requires intravenous contrast in order to maximize information about functionality, hematoma, and, possibly, bleeding.
- 2. The patient must be stable enough to go to the scanner
- 3. Full urinary assessment is dependent on the timing of contrast and scanning in order to view the bladder and ureters.

Intravenous Pyelogram Ivo



Advantages

- 1. It allows functional and anatomic assessment of both kidneys and ureters,
- 2. It may be performed in the emergency department or operating room. (Unstable pt ou table)

محدة تكون بيل - ٢

Disadvantages

- 1. It requires multiple images for maximal information.
- 2. The radiation dose is relatively high.
- 3. Findings do not reveal the full extent of injury.



Angiography - Curative aim (empolisation of a.), not for diagnosis or evaluation only!

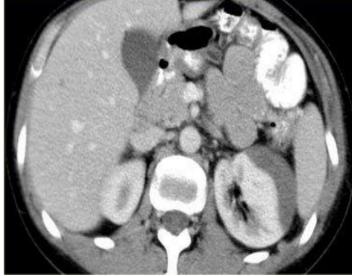
• Advantages:

- It has the capacity to aid in both the diagnosis and treatment of renal injuries
- 2. It may further define injury in patients with moderate IVP abnormalities or with vascular injuries.

• Disadvantages:

- 1. It is invasive reveal a indection of
- 2. It requires contrast high dose
- 3. It requires mobilization of resources to perform the study, which may be time-consuming
- 4. The patient must travel to the radiology department.





Abdominal CT scan demonstrating multiple kidney lesions of varying radiographic densities On the left a typical subcapsular hematoma, which is also a grade I renal injury.

Management of renal trauma



Stable patients, following grade 1-4 blunt renal trauma, should be managed conservatively with bed-rest, prophylactic antibiotics, and continuous monitoring of vital signs until haematuria resolves

Indications for surgical management include:

- 1. Haemodynamic instability
- 2. Exploration for associated injuries
- 3. Expanding pulsatile retroperitoneal haematoma during laparotomy
- 4. grade IV (in haemodynamically unstable patient) and, especially, grade V injuries often require nephrectomy to control bleeding (grade V injuries function poorly if repaired).
- Patients with **penetrating trauma** to the kidney require renal exploration unless radiographic imaging clearly delineates injury that can be managed safely nonoperatively in a hemodynamically stable patient.

Post-operative management and follow up



- Repeated imaging is recommended for all hospitalized patients within 2 to 4 days following renal trauma
- Within 3 months of major renal injury, patients' follow-up should involve:
 - 1. Physical examination
 - 2. Urinalysis
 - 3. Individualized radiological investigation
 - 4. Serial blood pressure measurement
 - 5. Serum determination of renal function
- Long-term follow-up should be decided on a case-by-case basis

Complications

• Early complications:

-occur within the first month after injury -include:

1-bleeding 2- infection

3-perinephric abscess 4-sepsis

5-urinary fistula 6-hypertension

7-urinary extravasation 8-urinoma.

Delayed complications:

-include:

1- bleeding 2- hydronephrosis

3-calculus formation 4-chronic pyelonephritis

5-hypertension 6-arteriovenous fistula

7-pseudoaneurysms.



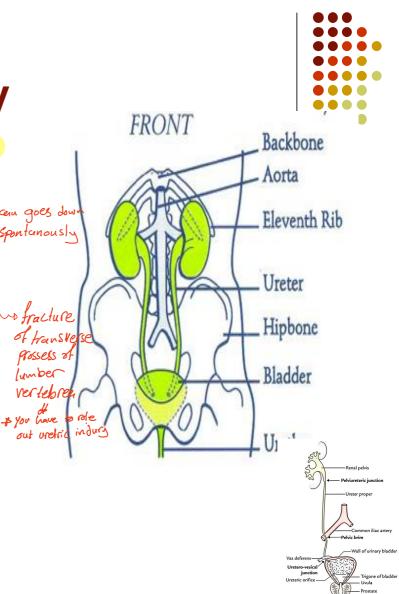
Ureteral Anatomy

- The ureters are retroperitoneal tubes measuring each about 25 cm long and about 3 mm in 3-5mm stones can goes dow diameter.
- They descend with an inclination medially on the posterior abdominal wall opposite the tips of the lumbar ~ fracture along tip of transverse processes. They transverse prosses of lumber continue following the contour of the pelvis.
 - They pass obliquely in the wall + you fine of the bladder of 1 inch before they open at the sides of the trigone.

3 narrow areas of ureter:

vertebra

the ureteropelvic junction (UPJ), the ureterovesical junction (UVJ), and the crossover of the common iliac arteries.



Ureteral Trauma



- Trauma to the ureter is relatively <u>rare</u> and accounts for only 1% of all urinary tract trauma.
- The ureters may be injured as a result of external violence (penetrating or blunt trauma) or by iatrogenic injury during an operation.

MCC - introgenic (undroscope, abdomenal surgery/gyne Surgery

Mode of Injury:

- **Simple**: Perforation (latrogenic)
- **Complex**: Transection (extensive surgeries, RTA)

Diagnosis - easy to miss

- e
- No classic clinical symptoms and signs associated with acute ureteral trauma caused by external injury. (EASY TO MISS)
- Ureteral trauma should be suspected in:
- 1. Penetrating abdominal injury and blunt deceleration trauma, in which the kidney and renal pelvis can be torn away from the ureter.
- 2. Isolated ureteral injuries if missed: subsequent evidence of upper tract obstruction, urinary fistula formation and sepsis.
- 3. Gynecological pelvic surgery: complaints of flank pain, vaginal leakage of urine or become septic.

Laboratory Studies



hematuria determin

- Laboratory evaluation should include urinalysis, urine culture, complete blood count, and creatinine, kert determination from the serum and drainage.
- Interestingly, hematuria is not a reliable finding in ureteral injuries, as only 74% of cases involve gross or microscopic hematuria. A failure to observe hematuria may be seen with a completely transected ureter or partial transection of an adynamic segment.

Imaging Studies

• **CT**: It is the criterion standard for evaluating abdominal injuries due to blunt trauma in stable patients. Contrast-enhanced CT scanning is highly sensitive in detecting urine extravasation and thus can be considered the primary imaging modality to evaluate for ureteral integrity in the stable patient.

• **IVP**: used in patients who must undergo immediate exploratory laparotomy for a penetrating injury to the abdomen



- Retrograde pyelography: it is the most sensitive radiographic study for the diagnosis of ureteral injury. It may be used in the stable patient as an adjunct to other imaging modalities when other clinical information is needed. It also has the added advantage of facilitating the placement of a ureteral stent in the same session, if indicated.
- Antegrade ureterography: it is not routinely used in diagnosing ureteral injuries. It is useful in conjunction with percutaneous nephrostomy tube placement or placement of an antegrade ureteral stent.



Classification of ureteral lesions



Grade	Description of Injury
I	Contusion or hematoma
II	eas < 50% transection
Ш	> 50% transection
IV	Complete transection with < 2 cm
	devascularization
V	Avulsion with > 2 cm devascularization

managemaent

Contusion

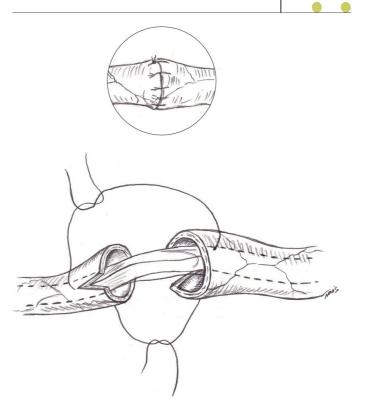
Although a contusion may be considered a minor injury, it can result in a stricture if left untreated. Extensive areas of contusion may even result in ureteral necrosis secondary to microvascular damage. Minor injuries can be treated with internal ureteral stenting, while severe or large areas of contusion should be excised and ureteroureterostomy performed.

Partial transection

Partial ureteral transections can be repaired with primary closure. Instead, the injured segment should be resected, with 2-cm margins, and the edges reapproximated. For non-ballistic injuries, the defect may be closed primarily by closing the longitudinal laceration transversely in order to avoid narrowing of the lumen).

Complete transections; upper, middle and distal ureteral injuries

1. Ureteroureterostomy: involves an end-to-end repair of ureteral defects smaller than 3 cm . This is mostly performed in the upper and mid ureter.





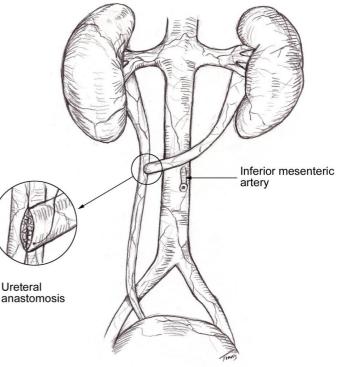
- 2. Ureteropyelostomy: the ureter may be anastomosed directly into the renal pelvis, this is done in case of proximal ureteral injury.(uper)
- **3. Ureterocalicostomy:** If the renal pelvis or ureteropelvic junction is damaged beyond repair the <u>ureteral stump</u> may be sewn end-to-side into an exposed renal calyx. (upper)



Transureteroureterostom (segnificant segmental loss)

4

y: this can be performed to manage an extensive defect that involves the mid or upper ureter if the length for anastomosis to the bladder is insufficient. This involves bringing the injured ureter across the midline to the recipient ureter in an end-to-side anastomosis

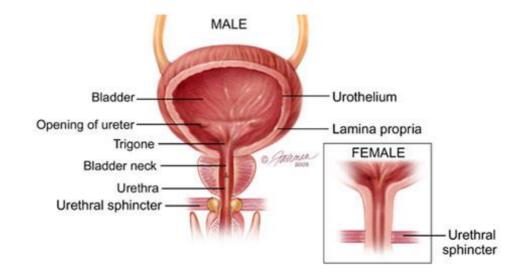


, brettic reimplantation

- **5. Ureteroneocystostomy**: Injuries to the lower ureter are usually associated with disruption of its blood supply from the iliac vessels. Therefore, these injuries are best repaired with ureteroneocystostomy. The principles of repair include debridement and spatulation of the ureter, tunneling in the bladder wall toward the bladder neck in a 3:1 ratio of tunnel length to diameter of ureter. (Inver)
- 6. Vesicopsoas hitch: This procedure involves mobilizing the bladder and pulling it superiorly and laterally by fixing it to the psoas tendon. (Invertional Control of Control o
- **7. Boari bladder flap :** For injuries too long to be bridged with the psoas hitch procedure alone, a Boari flap can be created to provide an additional 12-15 cm of length (*lover*)

Bladder Anatomy

- The adult bladder is located in the anterior pelvis and is enveloped by extraperitoneal fat and connective tissue. It is separated from the pubic symphysis by an anterior prevesical space known as the space of Retzius. The dome of the bladder is covered by peritoneum, and the bladder neck is fixed to neighboring structures by reflections of the pelvic fascia and by true ligaments of the pelvis.



Relevant Anatomy

- The type of extravasation depends upon the location of the laceration and its relationship with the peritoneal reflection.
- If the perforation is above the peritoneal reflection, the extravasation is intraperitoneal.
- If the injury is below the peritoneal reflection, the extravasation is extraperitoneal.

With an anterosuperior perforation, urinary extravasation may be intraperitoneal, extraperitoneal or both. If the tear is posterosuperior, fluid can spread intraperitoneally and/or retroperitoneally.

Bladder Trauma (rafe)



- Lower urinary tract injury may be caused by either blunt, penetrating, or iatrogenic trauma.
- 70-97% of patients with bladder injuries from blunt trauma have associated pelvic fractures.
- An exceedingly light blow may rupture the <u>fully distended</u> bladder, but the <u>empty bladder</u> is seldom injured except by crushing or <u>factor</u> penetrating wounds.

Class	ification
Туре	Description
1	Bladder contusion
2	Intraperitoneal rupture (dome is most weak part of the bladder) in grad fever, irretation of displiagon
3	Interstitial bladder injury
4	Extraperitoneal rupture - on in Pelvis
a.	Simple
b.	Complex
5	Combined injury





- The two most common sign and symptoms are gross haematuria and abdominal tenderness in patients with major bladder injuries.
- Other findings may include the inability to void, bruises over the suprapubic region and abdominal distension.
- Extravasation of urine may result in swelling in the perineum, scrotum and thighs, as well as along the anterior abdominal wall within the potential space between the transversalis fascia and the parietal peritoneum. (if left undiagnosed → abscess may occur)
- Intraperitoneal extravasation may lead to referred shoulder pain and respiratory distress.

Laboratory studies and Imaging studies



- Macro / Microscopic haematuria (95%)
- Cystography: standard diagnostic procedure; most accurate. (contrast)
- <u>CT</u>: <u>method of choice</u> for evaluation of blunt or penetrating abdominal / pelvic injury.
- IVP: high false negative results
- U/S: not routinely used for evaluation of bladder injury





- The first priority in the treatment of bladder injuries is stabilization of the patient and treatment of associated lifethreatening injuries.
- Blunt trauma:
- → Extraperitoneal rupture: catheter drainage. folg's→2-3w
- → Intraperitoneal rupture: surgical exploration.
- Penetrating injuries: All bladder perforations due to a penetrating trauma should undergo emergency exploration and repair
- The **perivesical hematoma** should be **left undisturbed** to avoid introducing bacteria.

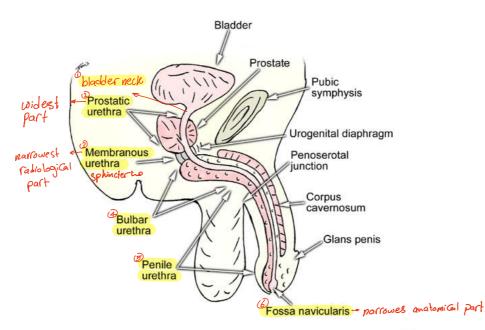
Urethral Anatomy



• Male urethra:

Canal for the discharge of urine and seminal fluid. It is 18-20 cm in length.

The male urethra may be divided into 2 portions. The posterior urethra includes the prostatic urethra and the membranous urethra, The anterior urethra stable by includes 3 segments. The bulbar urethra courses through the proximal corpus spongiosum and ischial cavernosus-bulbospongiosus muscles to reach the penile urethra. The penile urethra then extends through the pendulous portion of the penis to the final segment, the fossa navicularis. The fossa navicularis is invested by the spongy tissue of the glans penis.





• Female urethra:

Canal for discharge of urine that is 4 cm in length. It corresponds to the prostatic and membranous urethra in the male (posterior urethra). The anterior urethra corresponds to the labia minora

Relevant Anatomy



- The membranous urethra is prone to injury from pelvic fracture
- pelvic fracture The bulbar urethra is susceptible to blunt force injuries because of its path along the perineum
 - Straddle-type injuries from falls or kicks to the perineal area can result in bulbar trauma. Conversely, the penile urethra is less likely to be injured from external violence because of its mobility, but iatrogenic injury from catheterization or manipulation can occur, which is also possible in the fossa navicularis.

* MCC of indury of vrethra - introgenic (traumatic foly's Cath. Cystoscopy ...)

A diagnosis of acute urethral trauma should be suspected from the history. A pelvic fracture, or any external penile or perineal trauma, can be suggestive of urethral trauma.

 In conscious patient, a thorough voiding history should be obtained to establish the time of last urination, force of urinary stream, painful urination and presence of haematuria.

Diagnosis any pelvic fracture pt, penile or perineal trauma evaluate pretura

• On digital rectal examination, the so-called high-riding prostate may be identified, or the prostate may appear to be absent (a large pelvic hematoma causes superior displacement of the prostate when the urethra is disrupted)

- The following clinical indicators of acute urethral trauma warrant a complete urethral evaluation:
 - 1. Blood at the meatus / Blood at the vaginal introitus
 - 2. Haematuria: although non-specific, haematuria on a first voided specimen may indicate urethral injury.
 - 3. Pain on urination or inability to void (disruption)
 - 4. Haematoma or swelling

Retrograde urethrography is the radiographic imaging study of choice for evaluation the patient with suspected urethral injury.

This study should precede any attempts to pass a urethral catheter.

+any suspected urthial indury -> dou't try to enterine foly's cath (partial indry >> traumatic indury)

Management -> with or without vrin relention, management is Urin divargine (subrapubic Cath.)

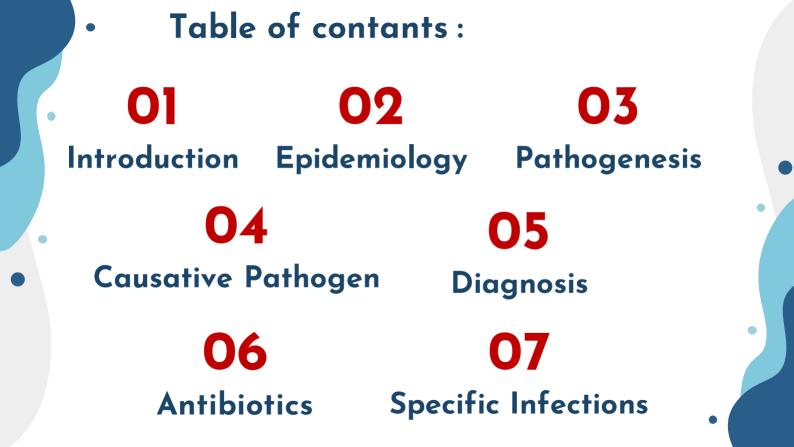
Anterior urethral injury

- Blunt trauma
 - Partial tears: suprapubic cystostomy to divert urine away from site of injury, and this is maintained for 4 weeks to allow healing.
 - Complete tear: end to end anastomosis
- Open injury: surgical exploration and repair
- Posterior urethral injury
 - Partial tear : cystostomy
 - Complete tear: urethroplasty later on

Urinary Tract Infection

Dr. Fadi Sawaqed

Presented by : Monther Al-Qatawneh Amin Al-Qatawneh





Introduction

- Urinary tract infection (UTI) is a term that is applied to a variety of clinical conditions ranging from localized infection of the bladder with lower urinary tract symptoms to pyelonephritis with severe infection of the kidney and the potential for resultant urosepsis.
- Accurate diagnosis and treatment of a UTI is essential to limit its associated morbidity and mortality and avoid prolonged or unnecessary use of antibiotics.
- Unfortunately, because of the increasing rates of bacterial resistance to various antibiotics, medical therapies are becoming less efficacious



. Epidemiology

- In the <u>neonatal period</u> <u>males</u> are twice as likely as females to experience a UTI.
- From ages 1 to 6 months the rate of UTI is equal between genders, but from 6 to 12 months of age the rate of UTI in male and female children is 1 to 4.
- Overall, UTIs are more common in females
- The incidence of UTI in uncircumcised males is 85% higher than circumcised males

• over all UTI more common in females

Epidemiology of UTI by age, group, and sex

Incidence (%)						
Age (y)	Female	Male	Main risk factors			
<1	0.7	2.7	Foreskin, anatomic GU abnormalities			
1–5	4.5	0.5	Anatomic GU abnormalities, functional 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			

GU = genitourinary; UTI = urinary tract infection.

Epidemiology

Risk factors for UTI include:

- Circumcision status.
- History of prior UTI.
- Sexual activity among older populations.
- Anatomical abnormalities like vesicoureteral reflux, ureterocele, ureteropelvic junction obstruction & posterior urethral valves.
- Functional abnormalities : neurogenic bladder, and bladder and bowel dysfunction
- Certain medical conditions like <u>diabetes</u>. <u>obesity</u>. sickle cell trait.



Pathogenesis

Understanding of:

- The mode of bacterial entry,
- Host susceptibility factors,
- Bacterial pathogenic factors

Are essential to tailoring appropriate treatment for the diverse clinical manifestations of UTI.





Bacterial Entry

the site through which micro-organisms enter the susceptible host



Bacterial Entry

MC mode of ends It is generally accepted that periurethral bacteria with a uropathogen from the gut ascending into the urinary tract causes most UTI.

- The colonization of the urethra and migration to the bladder leads to invasion of the bladder mediated by pili and adhesions factors.
- 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



Bacterial Entry

- Other modes of bacterial entry are uncommon causes of UTI.
 - **I-Hematogenous** spread can occur<u>(in immunocompromised patients)</u> <u>and in</u> [neonates]. Staphylococcus aureus, Candida species, and Mycobaeterium tuberculosis are common p<u>athogens</u> that <u>travel</u> th<u>rough the blood</u> to infect the urinary tract.
 - **II-Lymphatogenous** spread through the rectal, colonic, and periuterine lymphatics has been postulated as a cause for UTI;
 - **III-Direct extension** of bacteria from adjacent organs into the urinary tract can occur in patients with intraperitoneal abscesses or vesicointestinal or vesicovaginal fistulas.



02 Host Defense

The protection an organism is afforded against infections Types.

& most imp. & 1st defence mechanism => voiding (washout)



Host Defense

- Unobstructed urinary flow with the subsequent washout of ascending bacteria is essential in preventing UTI.
- In females, normal vaginal and periurethral flora contain microorganisms like lactobaeillus that <u>help prevent uropathogenic colonization</u>
- 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 in<u>hibit bacterial adherence</u>, such as Tamm-Horsfall glvcoprotein (THG)
- **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 activate innate host defenses.
- Specific serum and urinary antibodies are produced by the kidney to enhance bacterial opsonization and phagocytosis and inhibit bacterial adherence



O3 Bacterial Pathogenic Factors

Bacterial Pathogenic Factors

Pathoaenesis

- The ability of E. coli to adhere to epithelial cells is mediated by ligands located on the tips of the bacterial fimbriae (pili), implied adhesion of bacteria with epithelium
- Most uropathogenic <u>E. coli</u> strains produce hemolysin, which initiates tissue invasion and makes iron available for the infecting pathogens
- The presence of K antigen on the invading bacteria protects them from phagocytosis by neutrophils
- Encapsulation and biofilm formation hidden bacteria from host defence mechanism



Causative Pathogen

- Most UTIs are caused by a single bacterial species. At least 80% of the uncomplicated cystitis and pyelonephritis in premenopausal women are due to *E. coli*.
- <u>Other less common</u> uropathogens include <u>Staphylococcus saprophyticus</u>, <u>Klebsiella</u> <u>Proteus</u>, and <u>Enterobacter spp. and enterococci</u>.
- In hospital-acquired UTIs, a wider variety of causative organisms is found, including Pseudomonas and Staphylococcus spp.
- In children, the causative bacterial spectrum is slightly different but there is still a predominance of <u>E. coli</u> among inpatient and outpatient populations. Enterobacter, Enterococcus, and Klebsiella species make up the remainder of common culprits of pediatric UTI.
- Anaerobic bacteria, lactobacilli, corynebacteria, streptococci (not including enterococci), and Staphylococcus epidermidis are found in normal periurethral flora. They do not commonly cause UTIs in healthy individuals and are considered common urinary contaminants. - Urin Culture & Sensitivity

imp. to determine simple excamplicated UTL indection in healthy pt with N. virueity tract exactory fluction of amplicates and approximatives ass with UTI
 Diagnosis
 Standard diagnosis of UTI is completed by urinalysis and urine culture of 100 CFU/mL (Where CFU = colony-forming units) of bacteria.

- Occasionally, localization studies may be required to identify the source of the infection. Most often, the urine is obtained from a voided specimen.
- An uncomplicated UTI consists of an infection in an otherwise healthy patient with normal urinary tract anatomy.
- On the other hand, a complicated UTI can occur when anatomic abnormalities, immunocompromised state, or multi-drug-resistant bacteria allow for increased
- bacterial colonization or decreased therapeutic efficacy.

Urine Analysis looking for acidity, color

- The urine can be immediately evaluated for leukocyte esterase, a compound produced by the breakdown of white blood cells (WBCs) in the urine and is 95% sensitive for UTI in children with symptoms.
- Positive leukocyte esterase indicates the presence of 5—15 WBC per high-power field (hpl).
- Urinary nitrite is produced by reduction of dietary nitrates by many Gram-negative bacteria.
- Esterase and nitrite can be detected by a urine dipstick and are more reliable when the bacterial count is >100,000 colony-forming units (CF Us) per milliliter.
- Combined positive nitrite and leukocyte esterase on urine dipstick analysis is 80— 90% sensitive and 60—98% specific for UTI



- Microscopic examination of the urine for WBCs and bacteria is performed after centrifugation.
- For children, urine concentration should be taken into consideration when diagnosing infants with UTI.
- A pyuria threshold of 3 WBC/hpf in dilute urine and 6 WBC/hpf in concentrated urine is noted for a diagnosis of UTI

Presence of wBC's in Urin

+ Pyuria is one of main finding in Urin analysis - DDx: bacterial infection

Dacterial intection mycotubirculum (TB intection) inflammatian (interstitial cystitis, Chronic Prostatitis) uithout backerial inflammatian (interstitial cystitis, Chronic Prostatitis) uithout backerial inflammatian (interstitial cystitis, Chronic Prostatitis) uithout backerial with carcinoma institu



Urine Culture

diagnosis

- The gold standard for identification of UTI is the quantitative culture of urine for specific bacteria.
- Defining the CPU/ml. that represents clinically significant infection can be difficult. It is dependent on the method of collection, the sex of the patient, and the type of bacteria isolated.
- Traditionally, cultures demonstrating 100,000 CPU/ml. are considered diagnostic of a UTI, but now AAP guidelines suggest pyuria and 50,000 CPU/ml. of a single organism are diagnostic of UTI

Localization Studies

- Occasionally, it is necessary to localize the site of infection.
 Unix sample directly from one kidney of how one under a through coshosopy
 For upper urinary tract localization, the <u>bladder</u> is irrigated with sterile
 - water and a ureteral catheter is placed into each ureter. read reval pelvis
 - A specimen is collected from the renal pelvis. Culture of this specimen will indicate whether infection in the upper urinary tract is present.
 - In men, infection in the lower urinary tract can be differentiated (Figure). the 1st voiding part (1th voided sample) reflect urethral Colonization
 - Urethra A specimen is collected at the beginning of the void and represents distribution of th (VB1) possible infection in the urethra (VB1). Next, a midstream specimen effect the orin that is stored in the bladder (VB2) is collected and represents possible infection in the bladder. The prostate is then massaged and the patient is asked to void again (VB3), this specimen represents possible infection of the prostate.

Bladde (VBa

(EPS VBa)



Antibiotics

- The goal in treatment is to eradicate the infection by selecting the appropriate antibiotics that would target specific bacterial susceptibility.
- The general principles for selecting the appropriate antibiotics include:
 - Consideration of the infecting pathogen (antibiotic susceptibility, single-organism vs polyorganism infection, pathogen vs normal flora, community vs hospital-acquired infection);
 - The patient (allergies, underlying diseases, age, previous antibiotic therapy, other medications currently taken, outpatient vs inpatient status, pregnancy);
 - The site of infection (kidney vs bladder vs prostate)
 Pybniphritis
 Postalitis

		Diagnosis	Choice of antibiotics	^ǿ Duration of therapy
•	•	Cystitis	1st: TMP-SMX 2nd: Fluoroquinolone	1–3 days
placentalized de la cecheristics mediavism of action Side effects Antibio	tics	Pyelonephritis	1st: Gluoroquinolone 2nd: 2nd-generation cephalosporin 3rd: Aminopenicillin/BLI	7–10 days
	Trimethoprim—Sulfamethoxazole Fluoroquinolones - ciprofloxacin, levofloxacin Nitrofurantoin (hos perfued veurpattur SF) Aminoglycosides (nephrotoxicity & ototoxicity) if you give it for long time you have to follow bearing function (acutionet		1st: Fluoroquinolone 2nd: Aminopenicillin/BLI 3rd: 3rd-generation cephalosporin Aminoglycosides	Afebrile: 2 weeks Febrile: continue for additional 3–5 days after last fever (minimum 2 weeks)
UTI for adults.			1st: Fluoroquinolone 2nd: 2nd-generation cephalosporin 3rd: 3rd-generation	Acute: 2 weeks Chronic: 1-6 weeks
			cephalosporin 1st: Fluoroquinolone 2nd: 2nd-generation cephalosporin or 1st: Doxycycline 2nd: Macrolide	14 days
Pencility allergy -5) Cephalosporins -2 nd , 3 nd and 4 th generations; be	Cephalosporins - 2 nd , 3 rd and 4 th generations, bcz 1 st generation mainly concerning glam + 4 ^{ch} Generations			Single dose 7 days
		^a Complicated UTI: infection in the setting of metabolic, immunocom- promised, functional, or anatomic abnormality ^b If suspicion of sexual transmitted disease as source.		



- Acute Pyelonephritis
- Emphysematous Pyelonephritis
- Renal Abscesses
- Xanthogranulomatous Pyelonephritis
- Acute Cystitis
- Acute Bacterial Prostatitis

Acute Pyelonephritis

- Inflammation of the kidney and renal pelvis, and its diagnosis is usually made clinically.
- Present with chills, fever, and costovertebral angle tenderness.
- Accompanying lower tract symptoms such as dysuria, frequency, and urgency.
 - acute Pylowepute Sepsis may occur, with 20—30% of all systemic sepsis resulting from a urine infection.
 - Urinalysis commonly demonstrates the presence of WBCs and red blood cells in the urine.
 - Leukocytosis, increased erythrocyte sedimentation, and elevated levels of C-reactive protein are commonly seen on blood analysis.
 - Bacteria are cultured from the urine, when the culture is obtained before antibiotic treatment
 is instituted. <u>E. coli</u> is the most common causative organism, accounting for 70-80% of the cases.

Acute Pyelonephritis Management:

- The management of acute pyelonephritis depends on the severity of the infection. In patients who have toxicity due to associated septicemia, hospitalization is warranted. Approximately 10-30% of all adult patients with acute pyelonephritis require hospitalization. Empiric therapy with intravenous ampicillin and aminoglycosides. Alternatively. amoxicillin with
- home ttt clavulanic acid or a third-generation cephalosporin can be used.
 - For adults, fluoroquinolones or TMP-SMX is well tolerated and effective.
 - Outpatient treatment with an initial parental antibiotic (ceftriaxone or gentamicin) followed by oral therapy for 7 14 days most of acute pylouephritis
 - If bacteremia is present, parenteral therapy should be administered for 7—10 days and then the patient should be switched to oral treatment for an additional10-14 days.
 - Pregnant patient with concerns for pyelonephritis requires admission with parental antibiotics secondary to the risk of preterm labor. +

mmuno Compromised

Emphysematous Pyelonephritis

- Emphysematous pyelonephritis is a necrotizing infection characterized by the presence of gas within the renal parenchyma or perinephric tissue.
- About 95% of patients with emphysematous pyelonephritis have diabetes;
- women experience this condition 6 times more commonly than men.
- Other contributing factors include renal failure, immunosuppression, obstructed upper tracts, and polycystic kidneys.
- 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 (66%); Klebsiella pneumonia (26%): and Proteus. Pseudomonas, and Streptococcus spp.

Emphysematous Pyelonephritis

- The diagnosis of emphysematous pyelonephritis is made after radiographic examination.
- 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.
- In the management of emphysematous pyelonephritis, prompt control of blood glucose and relief of urinary obstruction are essential, in addition to fluid
- resuscitation and parenteral antibiotics

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.

Specific Infections

- Infector + Hematogenous spread of staphylococci, particularly from infected skin lesions. Patients with diabetes, those undergoing hemodialysis, or intravenous drug abusers have been at high risk for developing renal abscesses.
 - Fever, flank or abdominal pain. chills, and dysuria. Many of the symptoms have lasted for more than 2 weeks

#U/s -> anechoic mass within or displacing the kidney. posterior acoustic enhancement, or lack of vascularity on doppler # CT -> can demonstrate an enlarged kidney with local areas of hypoathemation early during the infection

fill pl doesn't respond within 42 of treatment, percataneous drainage under CT scan or Urs guidance is indicated, particularly for abscesses >3cm

Xanthogranulomatous Pyelonephritis

- Chronic bacterial infection of the kidney. unilaterally. The affected kidney is almost always hydronephrotic and obstructed
- 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
- Flank pain. fever, chills. and persistent bacteriuria. A history of urolithiasis is present in about 35% of patients
- <u>Flank mass</u> can often be palpated. <u>Urinalysis</u> commonly demonstrates leukocytes, bacteria. and proteinuria. <u>Serum blood</u> reveals <u>anemia</u> and may show hepatic dysfunction in approximately

50% of the patients

Xanthogranulomatous Pyelonephritis

- E. coli or Proteus species are commonly cultured from the urine.
- Computed tomography scan is the most reliable method in imaging patients suspected of having XGP. It usually demonstrates a large, heterogeneous, nonenhancing reniform mass.
- 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.
- In some cases, XGP is misdiagnosed as a renal tumor and a nephrectomy is performed and a diagnosis is made pathologically.
- In those in whom a diagnosis of <u>XGP is suspected</u>, kidney-sparing surgery such as a partial nephrectomy is indicated in focal disease. However, when the infection is diffuse, a
- nephrectomy with excision of all involved tissue is warranted.

tota

• Specific Infections • Acute Cystitis • Uncomplecated

- Urinary infection of the lower urinary tract, principally the bladder.
- Acute cystitis more commonly affects women than men.
- The primary mode of infection is <u>ascending</u> from the periurethral/vaginal and fecal flora.
- The diagnosis is made <u>clinical</u>.
- Present with irritative voiding symptoms such as <u>dysuria</u>, frequency, and <u>urgency</u>.
- Low back and suprapubic pain, hematuria, and cloudy/foul-smelling urine are also
- common symptoms. Fever and systemic symptoms are rare.

* Urin analysis - presence of WBCs diagnostic. Urin culture is required

Kttt for short time: THP, with fur untain, fluoroguinalones have excellent activity

* recurrent galits/UN Gused by either persitence or reinfection with another organism

Specific Infections

Acute Bacterial Prostatitis

- Inflammation of the prostate associated with a UTI- It is believed that infection results from ascending urethral infection or reflux of infected urine from the bladder into the prostatic ducts.
- Uncommon in prepubertal boys but frequently affects adult men/
- It is the <u>most common</u> urologic diagnosis in men younger than 50 years and third most common in <u>men older than 50 years</u>
- Present with an abrupt onset of constitutional (fever, chills, malaise, arthralgia, myalgia, lower back/rectal/perineal pain) and urinary symptoms (frequency, urgency, dysuria). They may also present with urinary retention due to swelling of the prostate.
- Digital rectal examination reveals a tender, enlarged gland that is irregular and warm.

Specific Infections

Acute Bacterial Prostatitis

- Urinalysis usually demonstrates WBCs and occasionally hematuria.
- Serum blood analysis typically demonstrates leukocytosis.
- Prostate-specific antigen levels are often elevated.
- 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 <u>4-6 weeks</u>.
- 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.

Urolithiasis

Epidemiology

The lifetime prevalence of Urolithiasis is approximately 12% for men_and 7% for women in the United States, and it is rising.

<u>Peak</u> incidence occurs in people aged <u>35-45</u> years.

male-to-female ratio of 3:1.

Calcium stones represent about 80%.

Pathophysiology

supersaturation of the urine by stone-forming constituents, including calcium, oxalate, and uric acid.

Etiology

- Environmental: A low fluid intake, with a subsequent low volume of urine production, produces high concentrations of stone-forming solutes in the urine.
- Metabolic Hypercalciuria is the most common metabolic abnormality.

- The four main chemical types of kidney stones are:
- . Calcium stones
- · Uric acid stones
- . Struvite stones
- Cystine stones

Calcium stones

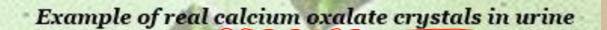
Calcium oxalate, calcium phosphate, and calcium urate are associated with the following disorders:

Hyperparathyroidism

Increased gut absorption of calcium

Renal calcium leak

Hyperoxaluría (fat malabsorption)



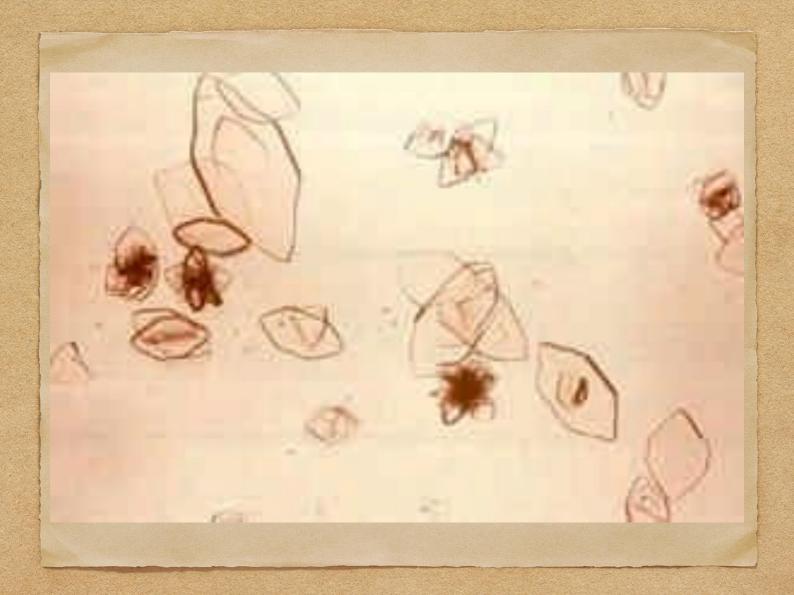
Struvite stones Struvite stones account for 15% of renal calculi. They are associated with chronic urinary tract infection ((ITI) with gramnegative, urease-positive organisms that split urea into ammonia, which then combines with phosphate and magnesium to crystalize into a calculus. (Isual organisms include Proteus, Pseudomonas, and Klebsiella species. Escherichia coli is not capable of splitting urea and, therefore, is not associated with struvite stones.



Uric acid stones

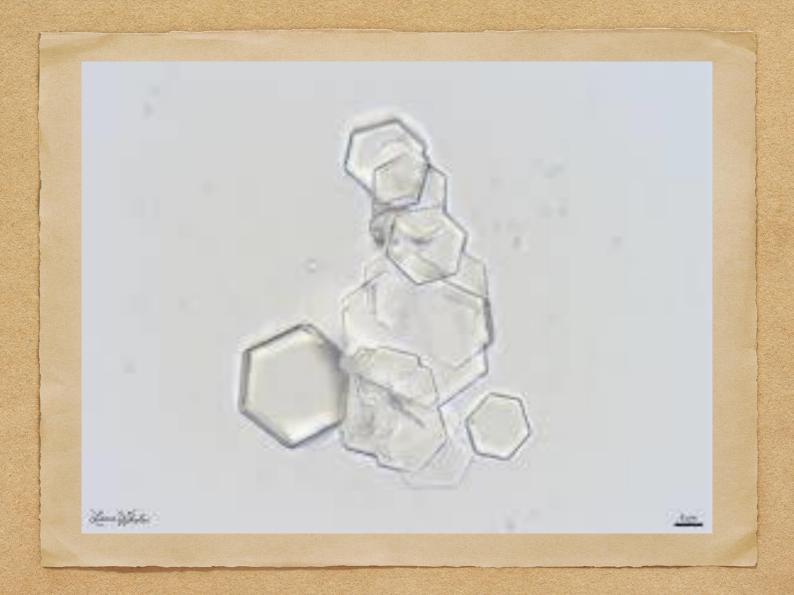
(Iric acid stones account for <u>6%</u> of renal calculi. These are associated with urine pH less than <u>5.5</u>, high purine intake (eg, organ meats, legumes, fish, meat extracts, gravies), or malignancy (ie, rapid cell turnover).

Approximately 25% of patients with uric acid stone have gout. النقرس



(ystine stones

Cystine stones account for 2% of renal calculi. They arise because of an intrinsic metabolic defect resulting in failure of renal tubular reabsorption of cystine, ornithine, lysine, and arginine. (Irine becomes supersaturated with cystine, with resultant crystal deposition.



Risk factors

- Patient habits: lower calcium intake, higher oxalate intake, higher animal protein intake, lower potassium intake, higher sodium intake, or lower fluid intake.
- . History of prior urolithiasis
- . Family history

Clinical manifestations

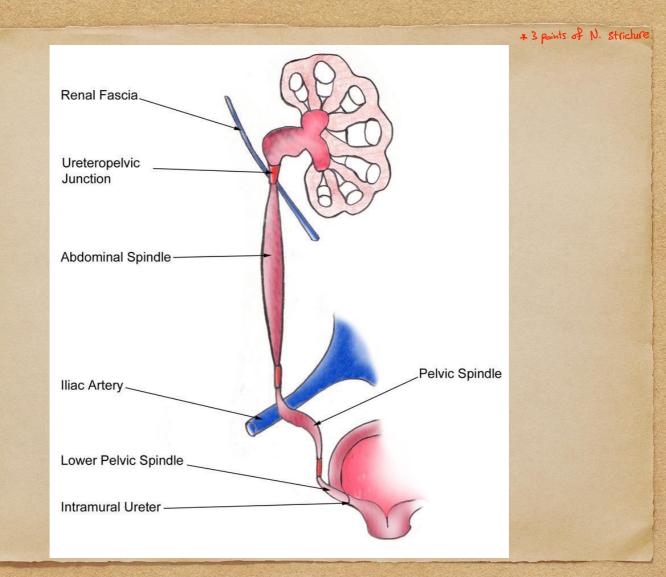
Asymptomatic

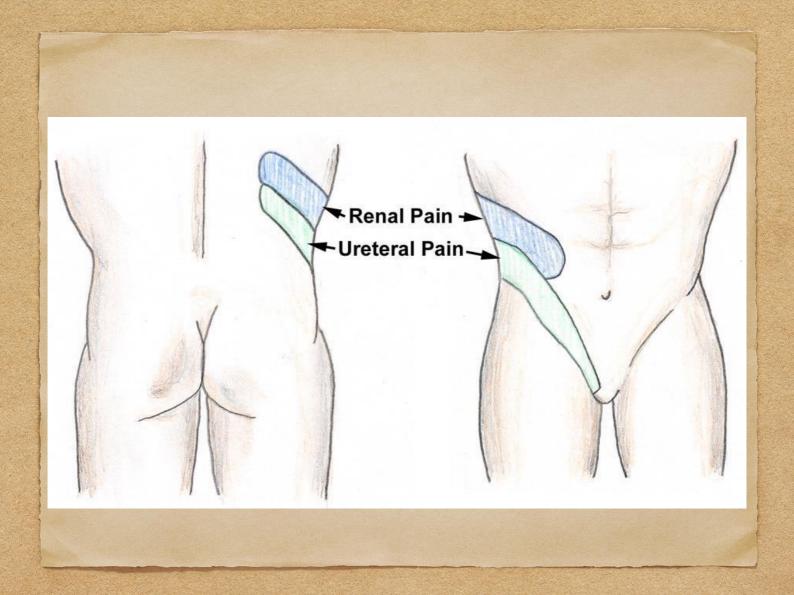
Acute onset of severe colicky flank pain radiating to the groin

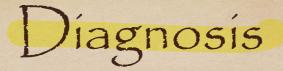
gross or microscopic hematuria

nausea, and vomiting

Physical examination is often unremarkable







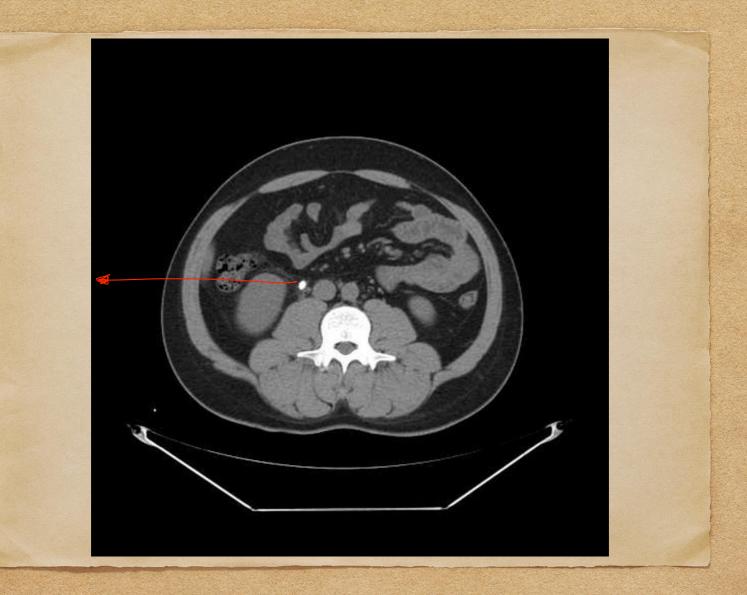
- . STONE score
- . Low probability (0-5)
- . Moderate probability (6-9)
- <u>High</u> probability (10-13)

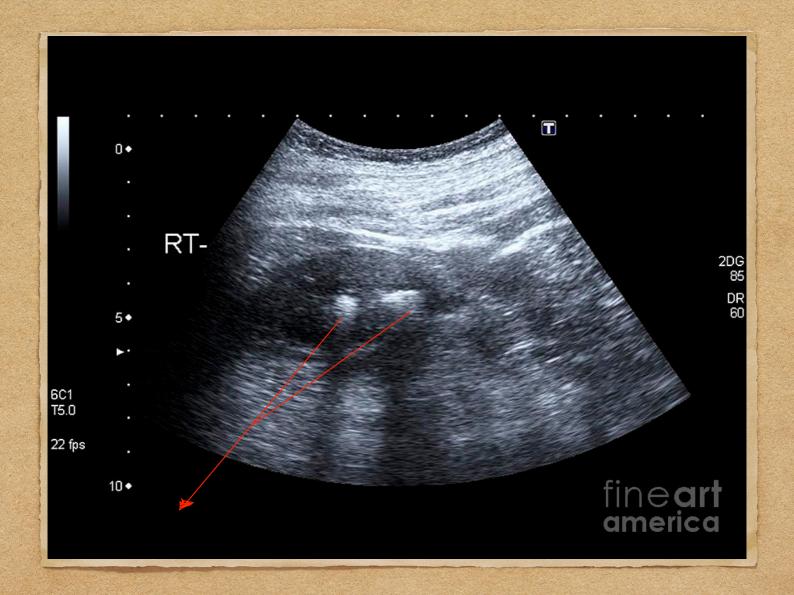
Factor	Points	
Sex:		
Female	0	
Male	2	
Duration of pain:		
>24 hours	0	
6–24 hours	1	
< <u>6 hours</u>	3	
Race:		
Black	0	
Non-black	3	
Nausea and vomiting:		
None	0	
Nausea alone	<u>1</u>	
Vomiting alone	2	
Hematuria:		
Absent	0	
Present	3	

Imaging: Gold standard: non-contrast spiral CT.

- · Ultrasound, especially in pregnant cases.
- · KUB









Presentation

Incidentally

- Symptoms:
- Pain

sudden severe flank pain that is colicky (waves of increasing severity are followed by a reduction in severity, but it seldom goes away completely). It may radiate to the groin as the stone passes into the lower ureter.

- Hematuria (microscopic or occasionally macroscopic)
- Malaise, weakness, and loss of appetite can also occur.

acute management

1-Pain relief

NSAID (diclofenac or ketorolac tromethamine) IM or IV, Orally or per rectum, provides rapid and effective pain control. Its analgesic effect is partly <u>anti-</u> inflammatory, partly by <u>reducing ureteric peristalsis.</u>

Opiate analgesics When NSAIDs are inadequate, such as morphine are added.

 Calcium channel antagonists (nifedipine) may reduce the pain of ureteric colic by reducing the frequency of ureteric contractions upper ureteral stones, whereas
 Alpha-blockers (Tamsulosin) may be used for distal ureteral stones.

Antiemetic drug (metoclopramide)

Antibiotic therapy

Only if the patient has clinical evidence of a UTI

- No need to encourage the patient <u>to drink copious</u> <u>amounts of fluids</u> or to give <u>them large volumes of</u> <u>fluids intravenously</u> in the hope that this will flush out the stone.
 - Excess urine output will tend to cause a greater degree of hydronephrosis in the affected kidney, which will make ureteric peristalsis even less efficient than it already is. Peristalsis, can only occur if the walls of the ureter above the bolus of urine can coapt, i.e., close firmly together. If they cannot, as occurs in a ureter distended with urine, the bolus of urine cannot move distally.

2-watchful waiting

- Chances of spontaneous stone passage depend principally on stone size.
- Many ureteric stones are 4 mm in diameter or smaller and most such stones (90%+) will pass spontaneously, given a few weeks (3weeks) with analgesics for exacerbations of pain.
- Stones that have not passed in 2 months are much less likely to do so, though large stones do sometimes drop out of the ureter at the last moment.

- Therefore, accurate determination of stone size (on plain abdominal X-ray or by CTU) helps predict chances of spontaneous stone passage.
- Calcium channel blocker (nifedipine) and Alphablockers may assist spontaneous stone passage and reduce frequency of ureteric colic.

 Asymptomatic stones followed over a 3-year period are more likely to require intervention or to increase in size or cause pain if they are >4 mm in diameter and if they are located in a middle or lower pole calyx. The approximate risks, over 3 years of followup, relative to stone size.

Table 8.3 Approximate 3-year risk of intervention, pain, or increasein stone size

		Stone size				
	<5 mm	5–10 mm	11–15 mm	>15 mm		
% Requiring intervention	20%	25%	40%	30%		
% Causing pain	40%	40%	40%	<u>60%</u>		
% Increasing in size	50%	55%	60%	7 <u>0%</u>		

Some stones are definitely not suitable for watchful waiting. Untreated struvite (i.e., infection related)
 staghorn calculi will eventually destroy the kidney if untreated and are a significant risk to the patient's life unless patient comorbidity is such that surgery would be a higher risk than watchful waiting.

Indications for stone removal

- Pain that fails to respond to analgesics or recurs and cannot be controlled with additional pain relief
- Impaired renal function (solitary kidney obstructed by a stone, bilateral ureteric stones, or pre-existing renal impairment that gets worse as a consequence of a ureteric stone)
- Prolonged unrelieved obstruction (generally speaking, ~4–6 weeks)
- Social reasons.

• These indications need to **be related to the individual patient**—their stone size, their renal function, presence of a normal contralateral kidney, their tolerance of exacerbations of pain, their job and social situation, and local facilities (the availability of surgeons with appropriate skill and equipment to perform endoscopic stone treatment).

نماني Emergency temporizing and definitive treatment of the stone

- When the pain of a ureteric stone fails to respond to analgesics or renal function is impaired because of the stone, then temporary relief of the obstruction can be obtained by insertion of *a JJ stent* or *percutaneous nephrostomy tube*(Percutaneous nephrostomy tube can restore efficient peristalsis by restoring the ability of the ureteric wall to coapt)
- but the stone is still present
- subsequent definitive treatment is still required

Kidney stone treatment options:

- *Stone fragmentation techniques
- *Flexible ureteroscopy and laser treatment
- *Percutaneous nephrolithotomy (PCNL)
- *open stone surgery
- *medical therapy (dissolution therapy)

Stone fragmentation techniques;

extracorporeal lithotripsy

Intracorporeal techniques of stone fragmentation



The technique of focusing externally generated shock waves at a target (the stone)

Three methods of shock wave generation are commercially available:—

electrohydraulic, electromagnetic, and piezoelectric

Efficacy of extracorporeal lithotripsy

fragmentation with ESWL depends on

stone size and location,

anatomy of renal collecting system,

degree of obesity

stone composition.

It is less effective for

*stones >2 cm diameter

*lower-pole stones in a calyceal diverticulum (poor drainage)

*composed of cystine or calcium oxalate monohydrate (very hard).

complications after ESWL :

<u>Common</u>

-Bleeding on passing urine for short period after procedure

- Pain in the kidney as small fragments of stone pass after fragmentation

- UTI from bacteria released from the stone, needing antibiotic treatment.

Occasional

Stone will not break as too hard, requiring an alternative treatment
 Repeated ESWL treatments may be required

- Recurrence of stones

Intracorporeal techniques of stone fragmentation

Electrohydraulic lithotripsy (EHL)

A high voltage applied across a concentric electrode under water generates as a spark. This vaporizes water, and the subsequent expansion and collapse of the gas bubble generates a shock wave.

Intracorporeal techniques of stone fragmentation

Electrohydraulic lithotripsy (EHL)

EHL is an **<u>effective</u> form of stone fragmentation**. The shock wave is not focused, so the EHL probe must be applied within 1 mm of the stone to optimize stone fragmentation.

Principal uses are for bladder stones (wider safety margin than in the narrower ureter)

Pneumatic (ballistic) lithotripsy

A metal projectile contained within the handpiece is propelled backward and forward at great speed by bursts of compressed air.

It strikes a long, thin, metal probe at one end of the handpiece at 12Hz

(12 strikes/second) transmitting shock waves to the probe, which, when in contact with a rigid structure such as a stone, fragments the stone.

This technique is used for stone fragmentation in the ureter or kidney

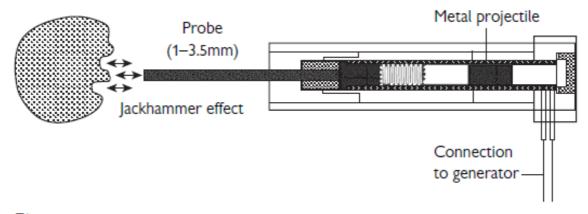


Figure 8.3 The Lithoclast: a pneumatic lithotripsy device. This figure was published in Walsh PC, et al. *Campbell's Urology*, 8th edition, pp. 3395–979. Copyright Elsevier 2002.

advantage

*Pneumatic lithotripsy is very safe

*The device is low cost and requires low maintenance

disadvantage

*Its ballistic effect has a tendency to cause stone migration into the proximal ureter or renal pelvis, where the stone may be inaccessible to

further treatment

*The metal probe cannot bend around corners, so it cannot be used for ureteroscopic treatment of stones within the kidney or with a flexible ureteroscope

Ultrasonic lithotripsy

An electrical current applied across a piezoceramic plate located in the ultrasound transducer generates ultrasound waves of a specific frequency (23,000–25,000 Hz).

The ultrasound energy is transmitted to a hollow metal probe, which in turn is applied to the stone

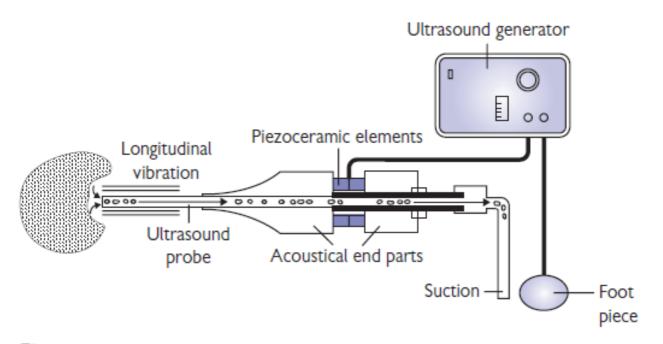


Figure 8.4 The Calcuson: an ultrasonic lithotripsy device. This figure was published in Walsh PC, et al. *Campbell's Urology*, 8th edition, pp. 3395–979. Copyright Elsevier 2002.

The stone resonates at high frequency and this causes it to break into small fragments that are then sucked out through the center of the hollow probe.

Soft tissues do not resonate when the probe is applied to them and thus are not damaged.

This technique can only be used down straight, rigid instruments.

Principal uses include fragmentation of renal calculi during PCNL. - perculaneous nephrolithdomy

Laser lithotripsy

- The holmium: YAG laser is principally a photothermal mechanism of action, causing stone vaporization.
- It has minimal shock-wave generation and therefore less risk of causing stone migration .

The 200-micron fiber is very flexible and can be used to gain access to stones even within the lower pole of the kidney

Principal uses are for ureteric stones and small intrarenal stones

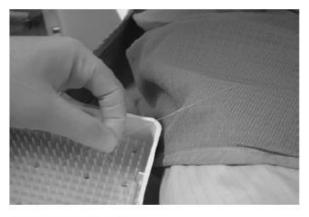


Figure 8.5 A laser fiber.

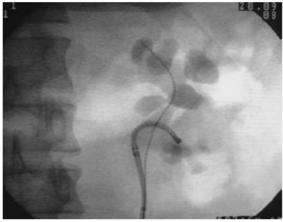


Figure 8.6 Access to the lower pole of the kidney with a flexible ureteroscope.

Flexible ureteroscopy and laser treatment





Extracorporeal shock were lithotripsy

*Lower pole stone

*Cystine stones.

*Obesity such that PCNL and ESWL is difficult

*<u>Musculoskeletal deformities</u> such that stone access by PCNL or ESWL is difficult or impossible (e.g. kyphoscoliosis).

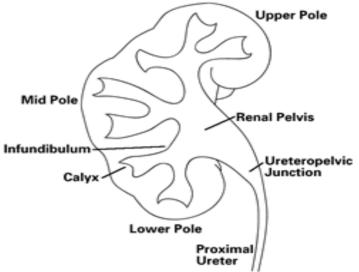
*Stone in a calcyceal diverticulum

*Stenosis of a calcyceal infundibulum or at angle between renal pelvis and infundibulum.

*Bleeding diathesis

*Horseshoe or pelvic kidney

*Patient preference



percutaneous nephrolithotomy

PCNL is the removal of a kidney stone via a <u>track</u> d<u>eveloped between the surface of the skin</u> and the collecting system of the kidney.

Steps of PCNL

1)inflation of the renal collecting system (pelvis and calyces) with fluid or air by ureteric catheter.

2) percutaneous puncture of a renal calyx with a nephrostomy needle



Figure 8.7 A ureteric catheter is inserted into the renal pelvis to dilate it with air or fluid.



Figure 8.8 A nephrostomy needle has been inserted into a calyx.

3.guidewire is inserted into the renal pelvis to act as a guide over which the track is dilated

4. access sheath is passed down the track and into the calyx, and through this a nephroscope can be advanced into the kidney

5. ultrasonic lithotripsy probe is used to fragment the stone and remove the debris.





Figure 8.9 A guide wire is inserted into the renal pelvis and down the ureter; over this guide wire the track is dilated.

Figure 8.10 An access sheath is passed down the track and into the calyx, and through this a nephroscope can be advanced into the kidney.

Indications for PCNL

- stones >3cm in diameter, those that have failed ESWL and/or an attempt at flexible ureteroscopy and laser treatment.
- It is the first-line option for staghorn calculi
- For stones 2-3cm in diameter, options include ESWL ,flexible ureteroscopy and laser treatment, and PCNL.
 PCNL gives the best chance of complete stone clearance with a single procedure, but this is achieved at a higher risk of morbidity.

open stone surgery

- Complex stone burden (projection of stone into multiple calyces, such that multiple PCNL tracks would be required to gain access to all the stone)
- Failure of endoscopic treatment
- Anatomic abnormality that precludes endoscopic surgery (e.g. retrorenal colon)
- <u>Body habitus</u> that precludes endoscopic surgery (e.g. gross obesity, kyphoscoliosis)
- Patient request
- Non-functioning kidney

Specific complications of open stone surgery

*wound infection (the stones operated on are often infection stones)

*f<u>lank hernia</u>

*wound pain

*stone recurrence

medical therapy (dissolution therapy)

- Uric acid stones
- Dissolution therapy is based on
- ✓ <u>hydration</u>,
- ✓ urine alkalinization → sodium bicarbonate
 or potassium citrate
- ✓ Allopurinol,
- ✓ dietary manipulation

Cystine stones

The aim is to do the following:

- **Reduce cystine excretion** (dietary restriction of the cystine precursor amino acid methionine and also of sodium intake to <100 mg/day)

 Increase solubility of cystine by alkalinization of the urine to >pH 7.5, maintenance of a high fluid intake, and use of drugs that convert cystine to more soluble compounds.

D-penicillamine, N-acetyl-D-penicillamine, and mercaptopropionylglycine

bind to cystine—the compounds so formed are more soluble in urine

Pediatric urology

done by : Manar Omoush Eliza Dumour Samar Jaradat

Undescended testes

 The testes descend into the scrotum in the third trimester (passing through the inguinal canal at 24– 28 weeks), Failure of testicular descent results in cryptorchidism (or undescended testes).

• Incidence:

Incidence is 3% at birth (unilateral > bilateral). Approximately 80% will spontaneously descend by 3 months. The incidence at 1 year is 1%.

A we wait until age of 6 months & more before doines surgery

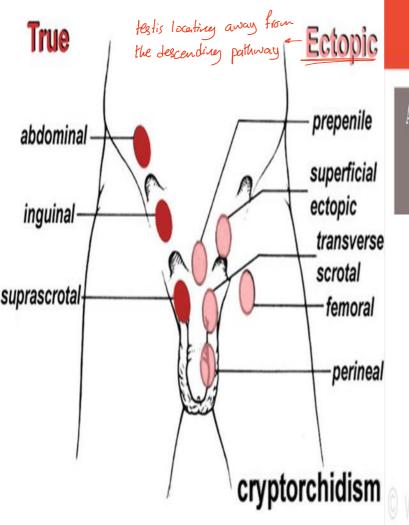
Classification

Retractile: an intermittent active cremasteric reflex causes the testis to retract up and out of the scrotum.

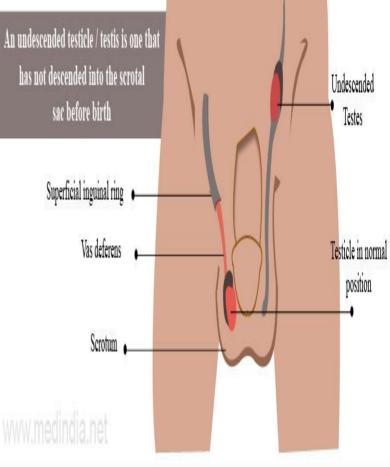
Ectopic (<5%): abnormal testis migration below the external ring of the inguinal canal (to perineum, base of penis, or femoral areas)

 Incomplete descent (~95%): testis may be intraabdominal, inguinal, or prescrotal
 Atrophic/absent

* freat these pt's semilarly



UNDESCENDED TESTICLES



Risk factors

These include preterm infants, low birth weight, small for gestational age, and twins.

• Etiology This includes :

- 1) abnormal testis
- 2) gubernaculum
- endocrine abnormalities (low level of androgens [hCG], (LH), calcitonin gene-related peptide)
- 4) decreased intra abdominal pressure (prune-belly syndrome, gastroschisis).

Pathology

There is degeneration of Sertoli cells, loss of Leydig cells, and atrophy and abnormal spermatogenesis.

- Long-term complications
- Relative risk of cancer is 40-fold higher in the undescended testis. Most are seminomas; carcinoma in situ represents a small percentage (~2%).
- Reduced fertility
- Increased risk of testicular torsion
- Increased risk of direct inguinal hernias

Diagnosis

- Full examination is required to elucidate if the testis is palpable and to identify location.
- Assess for associated congenital defects.
- If neither testis is palpable, consider chromosome analysis (to exclude an androgenized female) and hormone testing (high LH and FSH with a low testosterone indicates anorchia).
- before discharging any newborn male pt we have to examin the genitalia, and part of examination is scrotal examination to assess of there is testicles or no (so diagnoses of undescuending testicle can be immediatly after birth)

Management

- Treatment should be performed within the first year.
- Hormone therapy (hCG, LHRH) stimulates testosterone production.
- **Surgery** consists of inguinal exploration, mobilization of spermatic cord, ligation of processus vaginalis, and securing the testis into a dartos pouch in the scrotal wall **(orchidopexy).**

Done btw 1-2 y

 Intra-abdominal testes may require division of spermatic vessels to provide extra length (Fowler-Stevens procedure, relying on collateral blood flow from vas), two-stage procedures, or microvascular autotransplantation.

gring we should observe pt with undescended testicles: ~~?

No + to preserve fertility & testicular growth + PSychological issues in pt to have suggle testicle

Unilateral

Vesicoureteric reflux (VUR)

Definition

Results from abnormal retrograde flow of urine from the bladder into the upper urinary tract. (up to Kidneys)

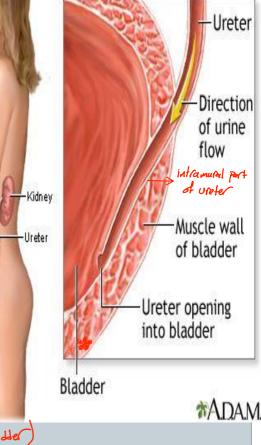
Epidemiology
 incidence >10%;
 younger > older;
 girls > boys (5:1)

Siblings of an affected child have a 40% risk of reflux

Pathogenesis

• The <u>ureter</u> passes obliquely through the bladder wall (1-2 cm), where it is supported by muscular attachments (physiological that prevent urine reflux during bladder filling and voiding. The normal ratio of intramural ureteric length to Bladderureteric diameter is 5:1. - that Prevent • Reflux occurs when the intramural length of ureter

is too short (ratio<5:1). Oreter short insection in bladder



Classification:

no pathological causes

• **Primary reflux** (1%) results from a **congenital** abnormality of the ureterovesical junction.

• Secondary reflux results from urinary tract dysfunction associated with elevated intravesical pressures. neurogenic bladder, obstruction of wrething

Causes include posterior urethral valves (reflux seen in 50%), urethral stenosis, neuropathic bladder, and detrusor sphincter dyssynergia (DSD).

Pt voiding against closed splincter -> Produce high P. in bladder induce reflux

* perflux induce renal domage in 2 forms:

(1) 2° to reflux of infected usin up to Kidney lead to recurrent pyelonephritits no in renal cortex (2) water nammer effect, usin will go high up to kidney with high flow - this will hit the renal collecting sys. f then lead to industry in cortex of kidney

• Complications

VUR associated with UTI can result in reflux nephropathy with hypertension and progressive renal failure. * if we menimize risk of bladder infection or cystitis - we will reduce risk of renal indury - of size in failure the * " " reduce bladder p. we can menimize water hammer indury from reflux into kidney for servative the

and up with reflux nephopathy

Seriously in reflux

Presentation

(recurrent febrile UTI in pediatric age group -> we have to think Patients have symptoms of UTI, fever, dysuria, suprapubic or abdominal pain, failure to thrive, vomiting, and diarrhea.

Renal imperment, hydronephrosis...

Diagnosed by MCUG (VCUG)

Investigation

- Urinalysis and culture to diagnose UTI
- Urinary tract ultrasound scan and VCUG Contrast indected into bladd
- Urodynamic assessment

draguose presence or absence of reflux (unilateral/bilderal) & grade of reflux

• DMSA scan to detect and monitor associated renal cortical scarring

• According to voiding cystourethrogram-->we diagnose the grade of reflux & presence or absence of reflux

Kidney and bladder ultrasound grading

• **Grade I** – reflux into non-dilated ureter

Grade II – reflux into the renal pelvis and calyces without dilatation

- **Grade III** mild/moderate dilatation of the ureter, renal pelvis and calyces with minimal blunting of the fornices
- **Grade IV** dilation of the renal pelvis and calyces with moderate ureteral tortuosity
- **Grade V** gross dilatation of the ureter, pelvis and calyces; ureteral tortuosity; loss of papillary impressions



Bilateral high grade reflux Grade 5

Massive bilateral reflux is seen on cystogram in young child.

Management

#Keep pt on conservative ttt

Correct problems contributing to secondary reflux. Most primary VUR grade I–II cases will resolve spontaneously (~85%), with 50% resolution in grade III. Observation and medical treatment are initially recommended.

Medical treatment

Low-dose antibiotic prophylaxis should be given to keep the urine sterile and lower the risk of renal damage until reflux resolves. **Anticholinergic** drugs are given to treat bladder overactivity • If we fail to keep urin sterile go for ¬

 (ureteroneocystostomy± ureteroplasty) or subureteral injection

Surgical Management

re-employed in of wreter

minimally invassive

- Indication for surgery:
- If it is not possible to keep the urine sterile and reflux persists
- If acute pyelonephritis recurs despite a strict medical regimen and chronic suppressive antimicrobial therapy.
- If increased renal damage is demonstrated by serial excretory urograms or nuclear scan.
- High grade reflux (grade IV or V not an absolute indication)

Hypospadias ventrally located meature

(2) absent of penil ventral skin

with 5 association. Overtrally locative meatures

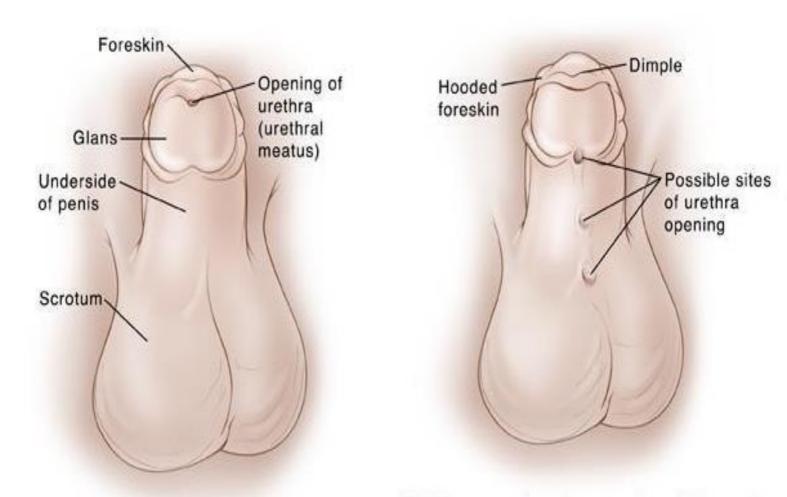
3 penile Chordee

Definition

(5) deviation of median raphy to one side of penis Hypospadias is a congenital deformity in which the opening of the urethra (the meatus) occurs on the underside (ventral) part of the penis, <u>anywhere from</u> the glans to the perineum. It is often associated with a hooded foreskin and chordee (ventral curvature of the penile shaft).

• It is the most common congenital malformation of the urethra. It occurs in 1 in 250 live male births

4 the more the proximal the meaturs the more the severity of hypospadras



Normally, the opening of the urethral meatus is located at the tip of the penis.

With hypospadias, the opening of the urethra is located on the underside of the penis or near the scrotum.

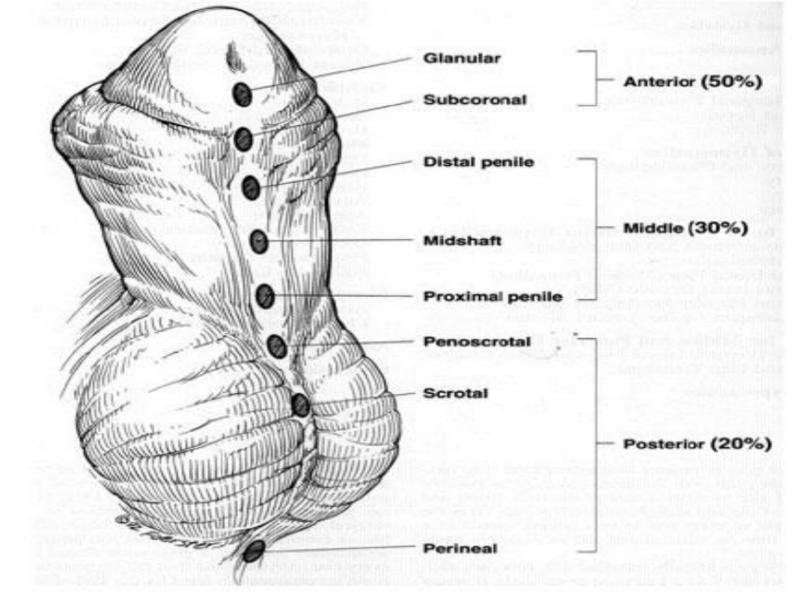


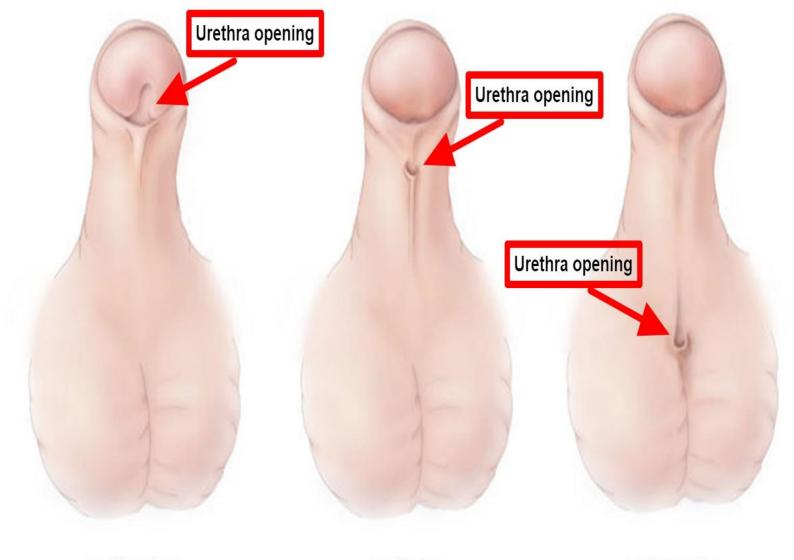
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Classification

- Hypospadias can be classified according to the anatomical location of the urethral meatus
 Anterior (or distal)—glanular, coronal, and
- subcoronal (~50%) mild affect fertility
- Middle—distal penile, midshaft, and proximal penile (~30%) fo reassure parents
- **Posterior** (or proximal)—penoscrotal, scrotal, and perineal (~20%)





Subcoronal

Midshaft

Penoscrotal

Etiology

• Hypospadias results from incomplete closure of urethral folds on the underside of the penis during embryological development.

• This is related to a defect in production or metabolism of fetal androgens, or the number and sensitivity of androgen receptors in the tissues.

Diagnosis

- A full clinical examination will make the diagnosis. However, it is also important to seek out **associated abnormalities** that will need treatment (undescended testes, inguinal hernias, and hydroceles).
- Patients with absent testes and severe hypospadias should undergo chromosomal and endocrine investigation to exclude intersex conditions

#reassure family:

1) shouldn't do circumcission before age of 1y (that done at time of deffinitive hypospadias repair)

2) this congenital pathology will not increase incidence of UTI or not affect fertility or erection in the future

Treatment

Whatever the site of hypospadias!

ttt of choice (doing cosmatic & functional surgery)

Surgery is indicated where deformity is severe, interferes with voiding, OR is predicted to interfere with sexual function. Surgery is now performed between 6 and 12 months of age.

Local application of testosterone for 1 month preoperatively can help increase tissue size.

The aim of surgery is to correct penile curvature (orthoplasty), reconstruct a new urethra, and bring the new meatus to the tip of the glans using urethroplasy, glanuloplasty, and meatoplasty techniques.

Complications

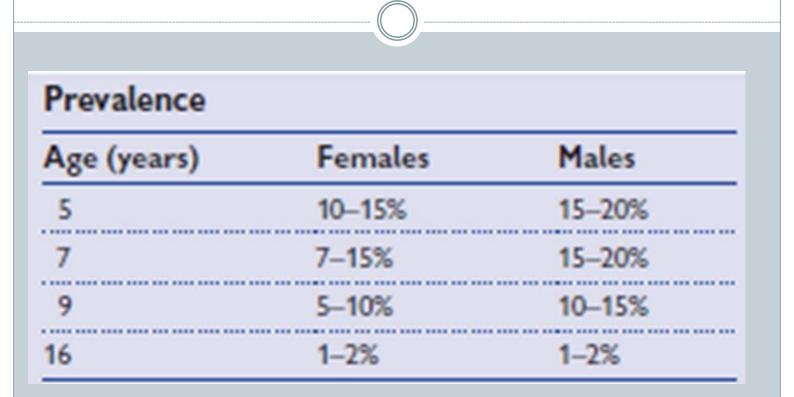
• These include bleeding, infection, urethral strictures, meatal stenosis, urethrocutaneous fistula, urethral diverticulum, and failed procedures requiring reoperation Nocturnal enuresis: passage of urin during sleep involuntary

- **Enuresis** is normal but involuntary voiding that occurs at an inappropriate time or social setting, during the day, night, or both.
- Nocturnal enuresis :

describes any involuntary loss of urine during sleep.







Classification

Primary: never been dry for more than a 6-month period **Secondary:** re-emergence of bed wetting after a period of being dry for at least 6 months

• Etiology

- Familial
- Delay in functional bladder maturation
- Altered antidiuretic hormone (ADH) secretion; abnormal decrease in ADH levels at night causes increased urine production (nocturnal polyuria)
- Altered sleep/arousal mechanism
- Psychological factors
- UTI (1% of cases)



• **History**: <u>frequency</u> of episodes; daytime symptoms; new or recurrent; family history; UTIs; bowel problems; psychosocial history

• Examination: exclude organic causes (neurological disease)

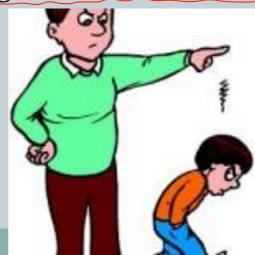
• **Investigation**: urinalysis (infection, specific gravity is reduced in nocturnal polyuria, glucose, protein); voiding diary

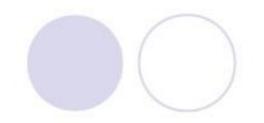
Management

• Behavioral

Provide reassurance; bladder training; motivational techniques to improve the child's self-esteem; conditioning therapy (an alarm is connected to the child's underwear, which is triggered with the first few drops of urine).

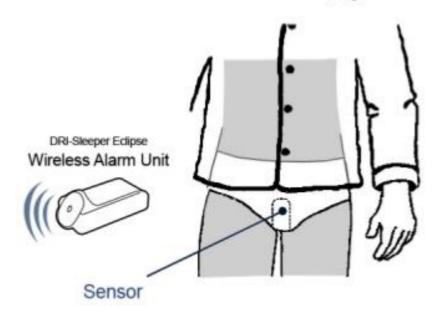








Wet Alarm Therapy





Pharmacological

- Imipramine—a tricyclic antidepressant with anticholinergic, antispasmodic properties.
- DDAVP or desmopressin⁽(synthetic analogue of ADH) given intranasally or <u>orally</u> Or (suplingual tablet) Ih before bedtime

Prognosis

 15% of patients have spontaneous resolution of symptoms per year.

Posterior urethral valves (PUV) pro mail

Congenital valve in prostatic urethra only ir male pt

Definition

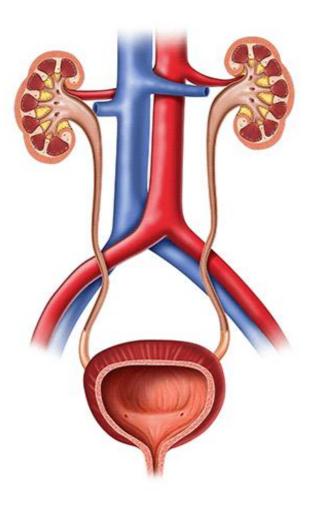
Posterior urethral valves (PUV) are ab<u>normal congenital</u> <u>mucosal fold in the prostatic (posterior) urethra causing</u> lower urinary tract obstruction. Incidence 1in5000 male. <u>lead high P in bladder - back up to kidney; inducing rend failer</u>

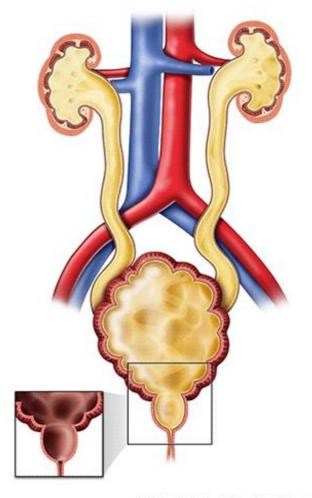
Cause:

Normal male urethra has small, paired lateral folds (plicae colliculi) found between the lateral, distal edge of verumontanum and lateral urethral wall. PUVs probably represent a congenital overgrowth of these folds from abnormal insertion of Wolffian ducts into the posterior urethra during fetal development.

Normal System

Posterior Urethral Valves (PUV)





- The <u>verumontanum</u>, or mountain ridge, is a distinctive landmark in the <u>prostatic</u> urethra, important in the systemic division of posterior valve disorders:
- **1.** Type I Most common type; due to anterior fusing of the *plicae colliculi*, mucosal fins extending from the bottom of the verumontanum distally along the prostatic and membranous urethra
- 2. Type II Least common variant; vertical or longitudinal folds between the verumontanum and proximal prostatic urethra and bladder neck
- 3. Type III Less common variant; a disc of tissue distal to verumontanum, also theorized to be a developmental anomaly of congenital urogenital remnants in the bulbar urethra

Usually we detect <u>maderate</u> <u>Walve</u>, usually identified - Presentation - Jepend on severity of obstruction during antimatal U/S

<u>Prenatal US features</u>

These include bilateral hydroureteronephrosis, dilated bladder with elongated ectatic posterior urethra, thick-walled bladder, oligohydramnios (reduced amniotic fluid), and renal dysplasia.

Early features are associated with poor prognosis.

<u>Newborn and infants</u>

These children have respiratory distress (secondary to pulmonary hypoplasia), palpable abdominal mass (hydronephrotic kidney or distended bladder), ascites, UTI, electrolyte abnormalities, and failure to thrive. <u>Older children</u>

Milder cases may present later with recurrent UTI, poor urinary stream, incomplete bladder emptying, poor growth, and incontinence. There is a risk of renal failure, vesicoureteric reflux, and voiding dysfunction (overactive or underactive bladder), also described as valve bladder syndrome.

Investigation

Investigation

- Ultrasound scan of kidneys and bladder.
 MCUG should be diagnosed during biding plase
 VCUG shows distended and elongated posterior
- VCUG shows distended and elongated posterior urethra, partialy filled anterior urethra, bladder neck hypertrophy; GOLD STANDARD FOR DIAGNOSING PUV.
- Isotope renal scan (MAG-3, DMSA) assesses renal function.
- Video urodynamics allows diagnosis of associated voiding dysfunction, urethra, bladder neck



Prostatic Vretura

if bad value: high grade reflux, trabeculated bladder & bilateral reflux

Figure 15.9 VCUG in infant with posterior urethral valves shows dilated elongated prostatic urethra and thickened bladder neck.

Treatment: MCUG - value! - Gtheter insortian - then value in bladder to drain vulgaration bkodder + minimize A by resecto scope in bladder

COMMENCE PROPHYLACTIC ANTIBIOTICS IMMEDIATELY, CHECK SERUM .ELECTROLYTES DRAIN THE BLADDER WITH A PEDIATRIC FEEDING TUBE. IF THERE IS IMPROVEMENT, CYSTOSCOPY AND TRANSURETHRAL ABLATION OF VALVE (CUTS AT 5 AND 7 O'CLOCK WITH ELECTROCAUTERY) IS RECOMMENDED (COMPLICATIONS INCLUDE URETHRAL STRICTURES).

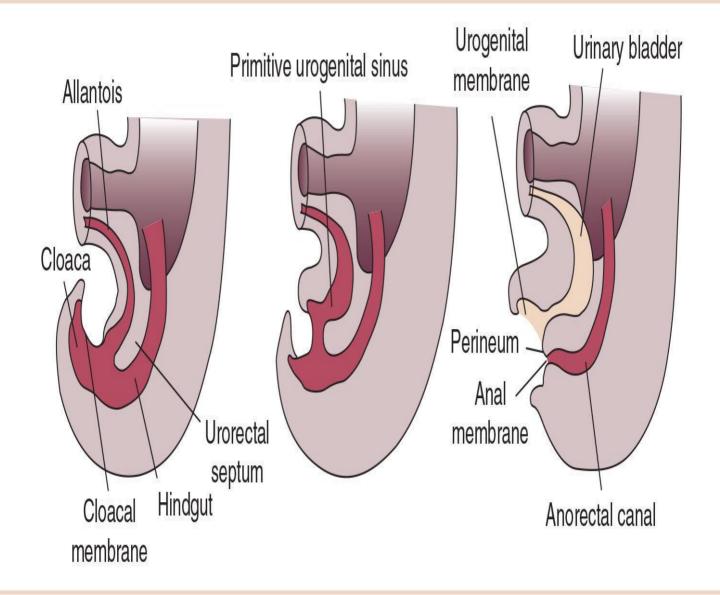
IF UPPER TRACTS REMAIN DILATED WITH RAISED CREATININE AFTER BLADDER DRAINAGE, A TEMPORARY CUTANEOUS VESICOSTOMY IS INDICATED (COMMUNICATING STOMA BETWEEN THE BLADDER DOME AND SUPRAPUBIC ABDOMINAL WALL, ALLOWING FREE DRAINAGE OF URINE). AN ALTERNATIVE IS URETEROSTOMY DRAINAGE. VALVE ABLATION IS PERFORMED AT A LATER STAGE.



Bladder extrophy results from defective development of the anterior bladder and lower abdominal walls, leaving the posterior bladder wall lying exposed on the abdomen.

♦M:F >2:1

Risk increased in family , younger maternal age and increased parity.



Embryology

• An embryological malformation results in the abnormal overdevelopment of the cloacal membrane, which prevents in-growth of lower abdominal tissues. The cloacal membrane normally perforates to form the urogenital and anal openings, but in extrophy there is premature rupture, resulting in a triangular defect below the umbilicus. The timing of this rupture determines the type of defect (bladder extrophy, cloacal extrophy, OR epispadias).

Associated anomaly:

- Urinary tract defect:VUR.
- Bone defect: Widening of the pubic symphysis
- Genital defect: Epispadias.
- * Musculofascial defect: Inguinal , femoral hernia.



Investigation

- Typical features seen on prenatal ultrasound scan include :
- lower abdominal wall mass
- absent bladder filling
- low-set umbilicus
- small genitalia
- abnormal iliac crest widening.



 At birth, cover the bladder with plastic film and irrigate regularly with sterile saline.

Trauma to the bladder mucosa can eventually result in squamous metaplasia, cystitis cystica, or adenocarcinoma and squamous cell carcinoma after chronic exposure.

Dr said:

1) cover bladder mucosa with guze or wet sponge to avoid dryness of bladder mucosa

2) reffared pt to tertiary center to deal with pt (immediatly)

- Selected cases are suitable for one-stage repair, but most require a <u>three-stage procedure</u>:
- *Newborn*: <u>pelvic osteotomy</u> (cutting bone to correct deformity) with external fixation with closure of bladder, abdominal wall, and posterior urethra
- 6–12 months: epispadias repair
- **4–5 years:** Bladder neck reconstruction (Young–Dees– Leadbetter procedure) and anti-reflux surgery (ureteric reimplantation) are performed when there is adequate bladder capacity and children can participate in voiding protocols.
- Where bladder capacity is too small, bladder augmentation or urinary diversion is required.

Prognosis

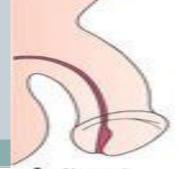
Even with successful surgery, patients may have long-term problems with

- > Incontinence
- > Urinary reflux
- > Repeated UTI
- Bladder adenocarcinoma
- Colonic adenocarcinoma
- > Uterine prolapse

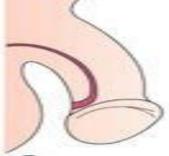
• Sexual function and libido are normal in extrophy patients.

Epispadias Very rafe

- The urethra open in the dorsal surface of the penis any where from the glans, penile shaft or most commonly the penopubic region.
 - Etiology represents failure of closure of the cloacal membrane, resulting in the bladder and urethra opening directly through the abdominal wall
 - M:F ... 5:1
 - High morbidity -+ incontinence, infertility, reflux.



A. Normal



Hypospadia



C. Epispadia

Management

• at 6–12 months >> This involves *urethroplasty* with functional and cosmetic reconstruction of the external genitalia (penile lengthening and correction of chordee).

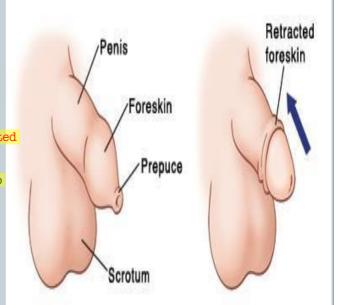
- The modified Cantwell–Ransley technique is commonly used in males. It describes mobilizing the urethra to the ventral aspect of the penis, with advancement of the urethral meatus onto the glans with a reverse MAGPI (meatal advancement-glanuloplasty).
- From age 4–5 years >> when children can be toilet trained, bladder neck reconstruction can be performed (Young–Dees–Leadbetter procedure). This achieves continence, and any bladder residuals may then be emptied by urethral catheterization.



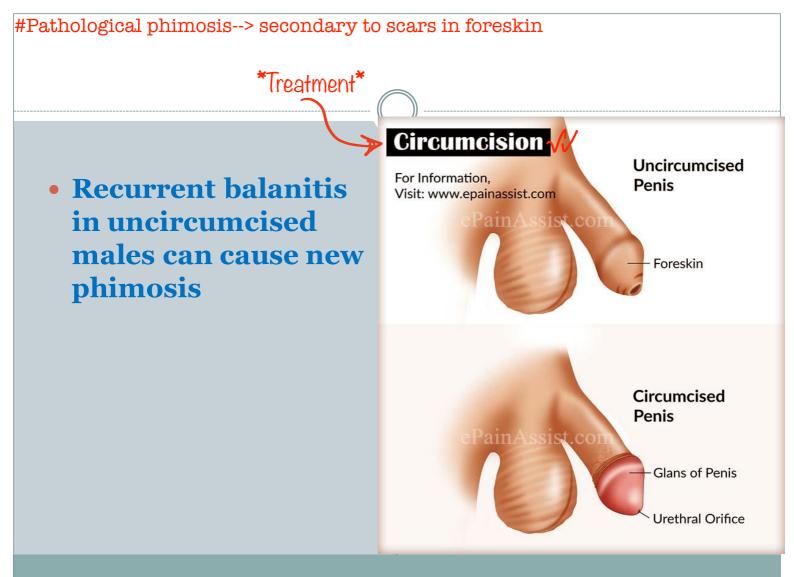
Fight foreskin, causing recurrent infection -UTI- (collection of bacteria under forskin)+ ballooning of foreskin during

Is when the foreskin cannot be retracted behind the glans.

- A physiological phimosis is present at birth due to adhesions between the foreskin and glans.
- As the penis develops, ^{circumcision} epithelial debris (smegma) accumulates under the foreskin, causing gradual separation.
- 90% of foreskins are retractile at age <u>3 years</u>, (<1% of phimosis at age <u>17</u>)



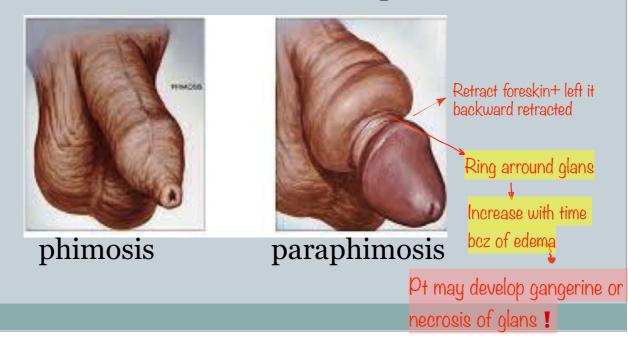
The unretracted foreskin and prepuce cover the penis. Retraction of the foreskin uncovers the head of the penis.



Treatment

- Older children with phimosis, suffering recurrent infection (balanitis), can be treated with a 6-week course of topical 0.1% dexamethasone cream, which acts to soften the phimosis and allow foreskin retraction (avoid circumcision where possible).
- Adults may require a dorsal slit or circumcision for recurrent balanitis, voiding obstruction, or difficulties with sexual intercourse.

• Paraphimosis is when the <u>uncircumcised foreskin</u> is retracted under the glans penis and the foreskin becomes edematous, and cannot be pulled back over the glans into its normal anatomical position.

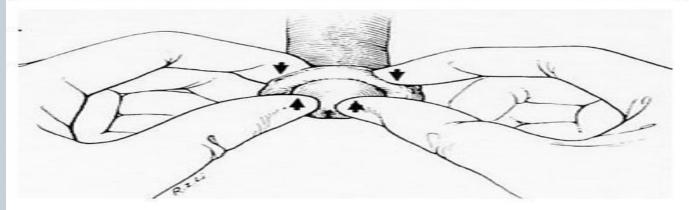


- It occurs most commonly in teenagers or young men and also in elderly men (who have had the foreskin retracted during catheterization, but where it has not been returned to its normal position).
- Paraphimosis is usually painful. The foreskin is edematous and a small area of ulceration of the foreskin may have developed.

Treatment:

- Manual reduction is preferred using ice packs, elastic compression, and topical anesthetic such as 2% lidocaine gel.
- Operative dorsal slit may be required in refractory cases.
- Elective circumcision for definitive treatment (paraphimosis tends to recur).

Figure 5. Paraphimosis Reduction



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ARE DEFINED AS CONGENITAL CONDITIONS IN WHICH THE DEVELOPMENT OF CHROMOSOMAL, Gonadal, or Anatomical Sex is Atypical. They are estimated to affect 1 in 4500 births.

DSD are subdivided into:

*SEMINIFEROUS TUBULE DYSGENESIS(KLINFELTER SYNDROME XXY,46XXMALE).

***TURNER SYNDROME 45XO.**

*TRUE HERMAPHRODITES 46XX,XY WITH BOTH OVARIAN AND TESTICULAR TISSUE.

*MIXED GONADAL DYSGENESIS (STREAK GONADS AND Ambiguous genitalia).

*PURE GONADS DYSGENESIS (FEMALE WITH STREAK GONADS).

Diagnosis:

*DETAILED HISTORY AND MATERNAL HISTORY ESPECIALLY DRUGS USED DURING THE PREGNANCY AS STEROID AND CONTRACEPTIVE PILLS

*GENERAL EX MAY SHOW ASSOCIATED SYNDROME ,EVIDENCE OF DEHYDRATION, POSITION OF URETHRAL MEATUS ,CARFUL PALPATION MAY SHOW THE PRESENCE OF TESTES AND THE PRESENCE OF FEMALE PSUDOHERMAPHRODITES.

CHROMOSOMAL ANALYSIS CONFIRM KARYOTYPE.

*SERUM ELECTROLYTE ,TESTOSTERONE ,DHT FOR SALT WASTING IN CAH.

*17 HYDROXY PROGESTERONE DONE AFTER 3 DAYS CAN DIAGNOSE 21 HYDROXYLASE DEFICIENCY.

*HCG STIMULATION TEST CAN DIAGNOSE ANDROGEN RESISTANCE AND 5A REDUCTASE DEFICIENCY.

Treatment

A MULTIDISCIPLINARY APPROACH IS REQUIRED WITH FULL PARENTAL INPUT. GENDER ASSIGNMENT OF AMBIGUOUS GENITALIA IS GUIDED BY THE FUNCTIONAL POTENTIAL OF GONADAL TISSUE, REPRODUCTIVE TRACTS, AND GENITALIA, WITH THE AIM OF OPTIMIZING PSYCHOSOCIAL WELL-BEING AND PRODUCING A STABLE GENDER IDENTITY.

PATIENTS HAVE A HIGHER RISK OF GONADAL MALIGNANCY, WHICH REQUIRES SURVEILLANCE AND/OR REMOVAL OF GONADAL TISSUES AND HORMONE REPLACEMENT.

*Renal cysts and Benign renal tumors

*Renal cyst :

*The most common (70%) are simple cysts, present in >50% of those aged >50y.

*Rarely symptomatic, usually incidental finding on abdominal imaging

Etiology:

Developmental - Multicystic dysplastic kidney (MCDK)

Genetic Autosomal recessive polycystic kidney disease (ARPKD),

massive kidneys with early renal failure in children associated with hepatic disease and blood vessels of brain and heart

autosomal dominant polycystic kidney disease (ADPKD), progressive bilateral disease leading to hypertension and renal fallure associated with hepatic cysts and cerebral aneurysms Cysts associated with systemic disease

Von Hippel-Lindau syndrome (VHLS), renal cysts, cerebellar and retinal hemangioblastomas, pancreatic and epididymal cysts
 30-40% incidence of renal cell carcinoma

Luberous sclerosis (TS)

Autosomal dominant syndrom charactrized by mental retardation, epliepsy, adenoma sebacaum, and other hamartomas

Acquired Simple cysts, acquired cystic renal disease, medullary sponge kidney (MSK): dilatations of the collecting ducts usually benign course, but predispose to calcium phosphate stones

Malignancy Cystic renal cell carcinoma (RCC)

*Solitary (simple) cyst of the kidney

The cyst:

- Contains Clear Fluid & may contain altered blood .
- Lined by **Flat** epithelium.
- Surrounded by **Fibrous** tissue



Usually **ASYMPTOMATIC**.

-Dull aching **PAIN** in the loin due to stretch of renal capsule.

-A SWELLING may be felt in the loin.

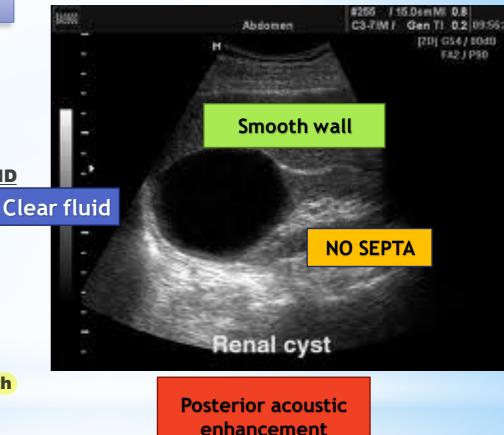
-Clinical picture of complications (e.g. Hematuria , pyuria...etc.)



Hemorrhage -Rupture, pressure on ureter (→ Hydronephrosis) -Infection & Calcification.



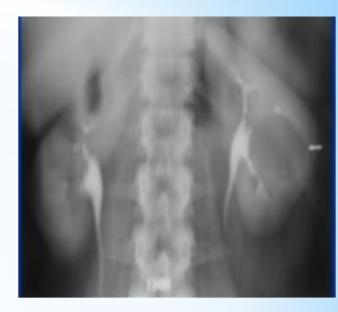
- A. <u>FOR DX:</u> 1. U/S in very helpful
- Differentiates between <u>SOLID</u> and <u>CYSTIC</u> lesions.
- Features of cyst are:
- (a)Anechoic (echolucentabscent of echoes)
- (b) Posterior acoustic enhancement
- (c) Sharply marginated smooth walls



*2. IVU - for reveal tumor if suspected

*• smooth amputated calyx. beak , claw sign ; resulted from stretching

of calyces by cyst



3. Renal angiography: (not done now) \rightarrow differentiate between <u>CYST</u> and <u>TUMOR</u>.

- Cyst \rightarrow avascular
- Tumor \rightarrow Hypervascular



*Cystic dysplastic kidneys. Micturating cystourethrogram on an infant with dysplastic kidneys on ultrasound showing vesicoureteric reflux into tortuous ureters and abnormal calyces with parenchymal filling and blush.

Bosniak Renal Cyst Classification System

according CT scan w/o contrast

- **I Simple cyst** with a hairline-thin wall.
 - No septa, calcifications, or solid components.
 - Water attenuation, no enhancement.
- II Septa: few hairline-thin in which not measurable enhancement may be appreciated.
 - **Calcification:** fine or a short segment of slightly thickened may be present in the wall or septa.
 - High-attenuation: uniform in lesions (< 3cm) that are sharply marginated and do not enhance.
- **IIF Septa**: multiple hairline-thin in which not measurable enhancement of septum or wall is appreciated.
 - Minimal thickening of wall or septa, which may contain calcification, that may be thick and nodular, but no measurable contrast enhancement.
 - No enhancing soft-tissue components.
 - attenuating renal lesions > 3 cm

Measurable enhancement

Cystic mass with thickened irregular or smooth walls or septa in which measurable enhancement is present

Burgery ; partial or radical replacectomy) T

we should treat them

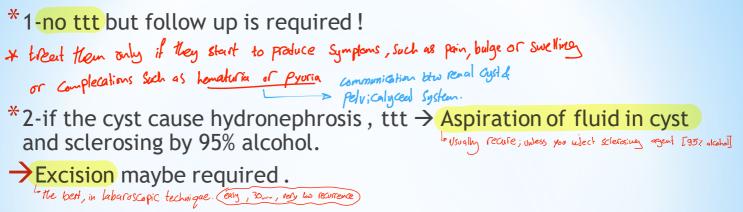
III

hus high % of being RCC

- Enhancing soft-tissue components

Clearly malignant cystic masses that can have all of the criteria of category III but also contain distinct enhancing soft-tissue components independent of the wall or septa

*Treatment :



3-Atypical cyst (hemorrhagic, thick wall or cloudy fluid):

- → PNA of content for analysis . Blood , high fat content or +ve cytology gives high suspicion of malignancy .
- ightarrow Excise the extrarenal portion of cyst .
- \rightarrow Partial nephrectomy maybe considered .



	Table 1. WHO Classification of Benign Renal Masses	
	Classification	Types
	Epithelial Tumors	Onocytoma Papillary adenoma
BENIGN	Mesenchymal Tumors	Angiomyolipoma Leiomyoma Hemangioma Reninoma Schwannoma Lymphangioma
Angiomyolipoma	Mixed Epithelial and Mesenchymal Tumors	Mixed epithelial and stromal tumor Cystic nephroma
Renal adenoma	Metanephric Tumors	Metanephric adenoma Metanephric adenofibroma Metanephric stromal tumor
Oncocytoma		



a BENIGN RENAL EPITHELIAL NEOPLASM that arise from the intercalated cells of collecting ducts that comprises approximately 5-9% of renal tubular epithelial tumors

accounting for 3-7% of renal tumors, more common in males.

They occur with RCC in 7-32% of the cases.

Presentation :83% present as an incidental finding or with flank pain or hematuria

*gross appearance:

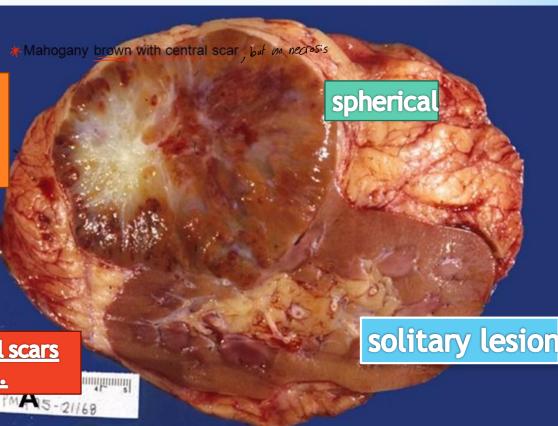
mainly unilateral

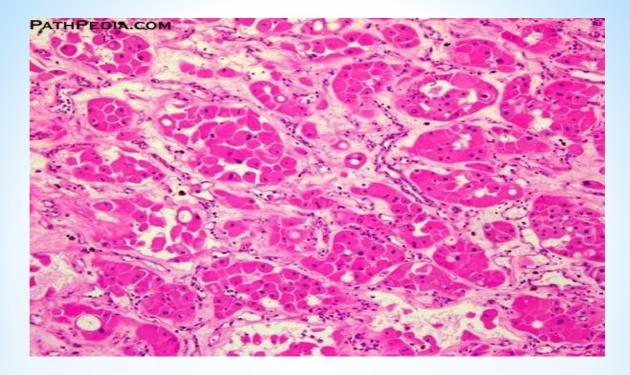
can be multiple and bilateral

Mean size 4-6cm

Well defined fibrous capsule with 10-20% extending into the perinephric fat

brown with a <u>central scars</u> <u>but no necrosis.</u>





Histological: organized eosinophilic cells originating from intercalated cells of collecting duct, mitoses are rare and they are considered benign, not known to metastasize. *Diagnosis :

*oncocytoma cannot often be distinguished radiologically from RCC, it may coexist with RCC.

*rarely they exhibit a **SPOKE WHEEL** *characteristic & oucocytoma* **PATTERN** on **CT scanning**,





PARTIAL/RADICAL NEPHRECTOMY

> 4 cm

HUGE

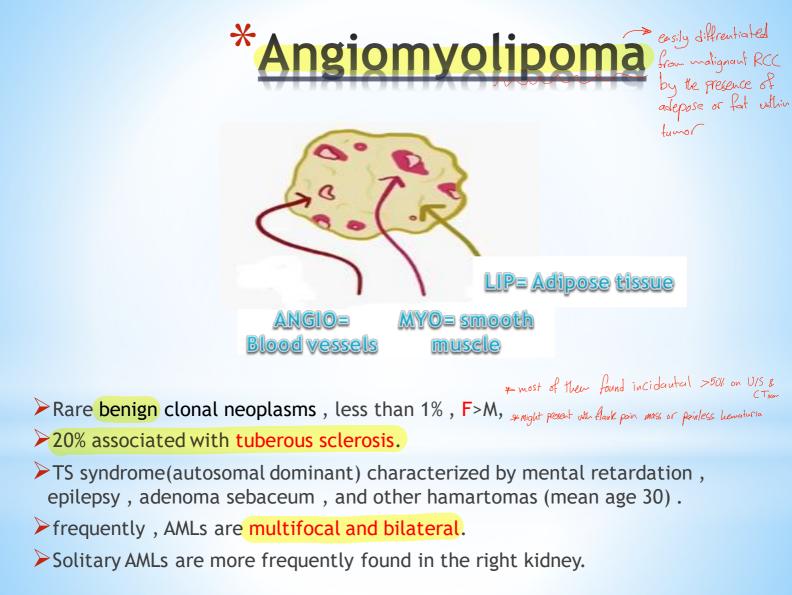
Minimally invasive techniques :
1- Radiofrequency ablation (RFA). (less than 3-4cm)

SMAL

4 -> left intented

AGEMENTS

2- High intensity focused ultrasound (HIFU). Non invasive therapeutic techniques that uses non ionizing ultrasonic waves





incidental finding >50% on US and CT, they may present with

- flank pain,
- palpable mass, or painless haematuria.
- Massive and life-threatening retroperitoneal bleeding occurs in up to 10% of cases

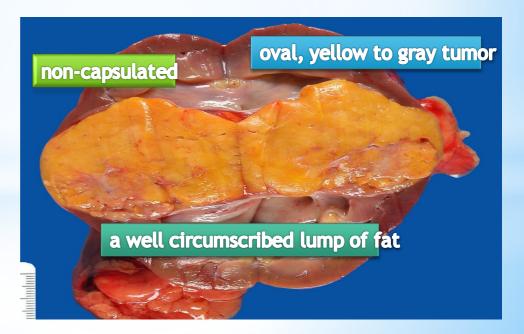
(Wunderlich's syndrome). is spontaneous, nontraumatic renal hemorrhage confined to the subcapsular and perirenal space. It may be the first manifestation of a renal angiomyolipoma (AML), or rupture of renal artery or intraparechymal aneurysm

Usually seen in

Pregnant women appear to be at an increased risk for hemorrhage .

AML is composed of <u>PERIVASCULAR</u> epithelioid cells (PEC)

Gross appearance:



*Management :

1- asymptomatic AMLs can be followed with serial US if <4 cm .

2- symptomatic AMLs (bleeding) or >4 cm should be treated surgically or by embolization. if symptometric with bleeding or >4 cm

*emergency nephrectomy or selective renal artery embolization may be life saving . Very rare (for wander lich's syndrom)

*in patient with TS , in whom multiple bilateral lesions are present , conservation treatment should be attempted .

Investigation : Pasy to diffrentiate from RCC

*Ultrasound reflects from fat, hence a characteristic bright echo pattern. This does not cast an 'acoustic shadow' beyond, helping to distinguish an AML from a calculus.

CT shows fatty tumour as low density in 86% of AMLs

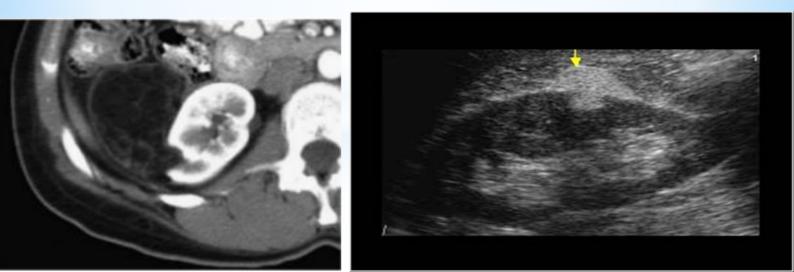
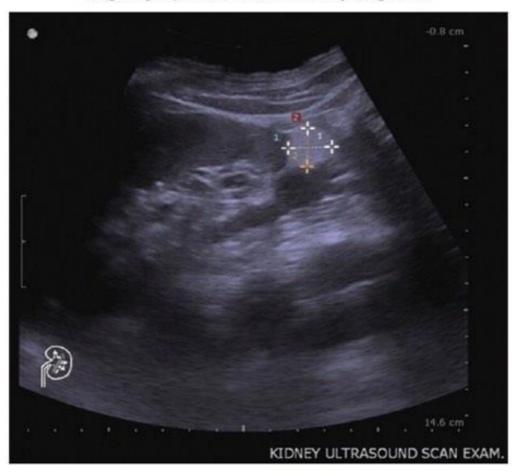
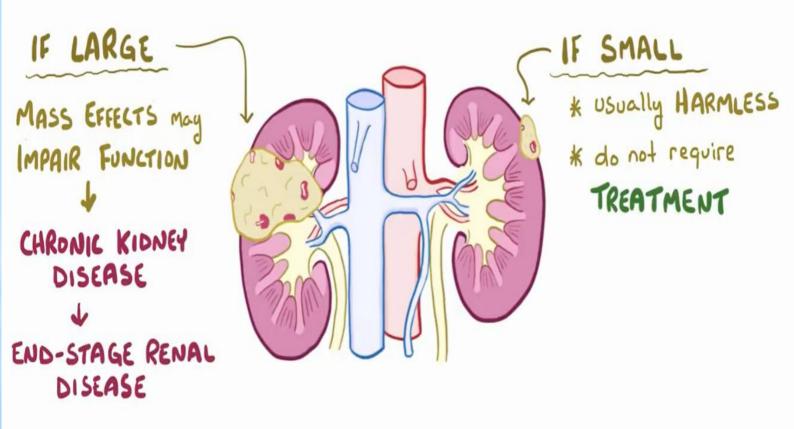


Figure 1: Kidney ultrasound.Ultrasound reveals a homogeneous, well-defined, hyperechoic lesion in the left lower kidney, which demonstrates the presence of macroscopic fat. Renal angiomyolipoma is the most likely diagnosis



ANGIOMYOLIPOMA



ANGIOMYOLIPOMA

ANGIOMYOLIPOMAS have a LARGE TUMORS can develop high density of BLOOD VESSELS IRREGULAR BLOOD VESSELS difficult to RESECT that can DILATE alternatively DESTROY the forming an ANEURYSM BLOOD SUPPLY with EMBOLIZATION that can RUPTURE TUMOR can become Can cause FLANK PAIN NECROTIC & SHRINK HYPOVOLEMIC SHOCK POSTEMBOLIZATION SYNDROME (esp. if near major BLOOD VESSEL) L fever, flank pain & malaise



Renal adenomas are the most common form of benign renal tumor. Their cause is unknown. incidence increase with age.

It is traditionally classified into three distinct types : 1-renal papillary adenoma (the most common renal epithelial

neoplasm)

2-renal tubular adenoma

3-alveolar renal adenoma

- ▶7 23 % at autopsy
- ► M:F = 3:1

Cytogenetics : which may exhibit trisomy of chromosome 7 & 17

- small cortical lesion < 1 cm</p>
- Rarely symptomatic

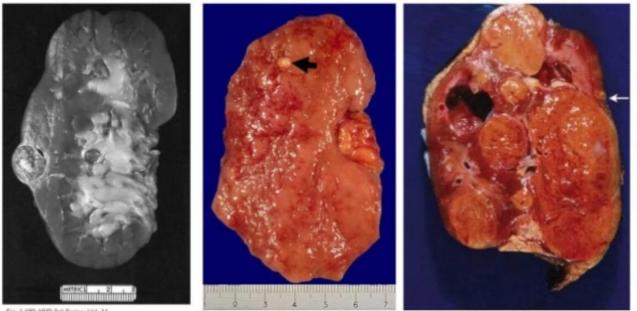


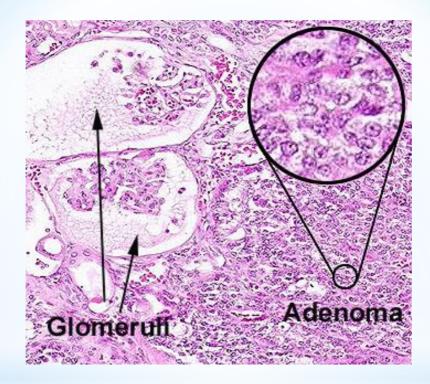
Fig. 1-199 AFIP 3rd Series, Vol. 11 RENAL CORTICAL ADENOMA



gross appearance :

solid ,**non-capsulated** kidney tumor, and are typically small.

Majority are solitary but may be multifocal



Histological : organized cells with no atypia or little atypia

Adenoma cells look much like low-grade RCC cells under a microscope. In fact, while they are considered benign, there is presently no known cellular classification to differentiate them from RCCs. Many researchers and physicians regard them as early stage precancers, to be treated accordingly.

Controversy as to whether this represent benign or premalignant neoplasm

Incidental finding on CT

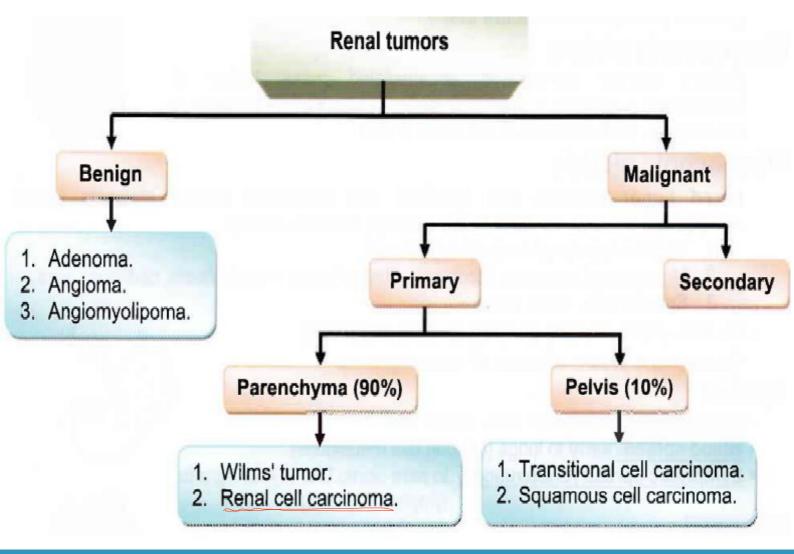
Management : Partial / radical nephrectomy if mass > 3cm due to increase risk of meatstasis ~ bcz of any reval was appearing on Tsom or U/s other than angioungolipour, usually not difficultated whether it is beingn or maliguent, keeper almost always after dive partial or radical apprectorm.

Other rare tumors:

- *Leiomyoma
- *Hemangioma
- *Lipoma
- *Juxtaglomerular cell tumors
- *Fibroma
- *Rhabdomyoma

Malignant renal tumors we will talk about one of them.

Done by: Batool Al Hbaishan



Renal cell neoplasms

- Malignant renal tumors
- Renal cell carcinoma
- Nephroblastoma (wilm's tumor
- Neuroblastoma

Renal cell carcinoma (<u>hypernephroma</u>/Grawitz's tumor), clear cell, rend cell ca

misnomer, because we

thought it originated from

- The commonest of renal tumours
- 85% of renal malignancies
- constituting 2–3% of all cancer deaths
- The most lethal of all urological tumours, approximately 40% of patients dying of the condition.
- more in males (M:F = 2:1)
- peak incidence of sporadic RCC is between 50–70y of age.

Renal cell carcinoma Risk factor

Smoking cigarettes doubles the risk of developing kidney cancer.

✓ **Gender. Men** are 2 to 3 times more likely to develop kidney cancer than women.

Race. Black people have higher rates of kidney cancer.

✓ Age. Kidney cancer is typically found in adults and is usually diagnosed between the ages of 50 and 70.

 Nutrition and weight Research has often shown a link between kidney cancer and <u>obesity</u>, vitamins A, C, E, and fruit and vegetable consumption are protective.

✓ High blood pressure HTN

Overuse of certain medications esp. Painkillers containing phenacetin Diuretics and analgesic pain pills, such as aspirin, acetaminophen, and ibuprofen, have also been linked to kidney cancer.

Occupational with asbestos and cadmium exposure

Chronic kidney disease

Long-term dialysis are at very high risk of devoloping RCC the highest risk of developing this CA

✓ Family history of kidney cancer People who have first-degree relatives with kidney cancer have an increased risk of developing

Anatomical risk factors include polycystic and horseshoe kidneys.

Renal cell carcinoma etiology



**Clear cell renal cell carcinoma:

This is associated with deletion of chromosome 3p and/or mutations of the VHL gene.

Von Hippel–Lindau (VHL) syndrome

Half of individuals with this <u>autosomal dominant</u> syndrome, characterized by pheochromocytoma, renal and pancreatic cysts, and cerebellar hemangioblastoma,

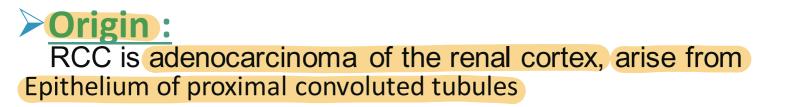
develop RCC, often bilateral and multifocal.

Patients typically present in third, fourth, or fifth decades.

•Chromophobe RCC is a result of loss of chromosome 17

•<u>A papillary variant of RCC</u> also has an autosomal dominant familial component, characterized by trisomy 7 and 17

•Tuberous sclerosis complex (TSC) is a genetic condition associated with changes in the skin, brain, kidney, and heart. People with TSC also have an increased risk of developing angiomyolipomas of the kidney and kidney cancer



<u>Site</u>:

Usually unilateral mainly from upper pole.

** Bilateral tumor (1-2%) :

• Von-Hippel Lindau disease

hereditary papillary renal carcinoma (gendic)

Macroscopic picture :

- Mass (mainly from upper pole of kidney) infiltrating edge
- area of <u>hemorrhage</u>, necrosis and scarring
- golden yellow color due to high lipid content
- They are usually circumscribed by a <u>pseudocapsule of</u> <u>compressed tissue</u>
- 7-20% are multifocal,10–20% contain calcification, and 10– 25% contain cysts or are predominantly cystic.
- •usually invades the <u>pelvis early, capsule late</u>

Microscopic picture :

Adenocarcinoma:

a. Cells:

- clear cell type (due to lipid, cholesterol and glycogen content)
- or granular cell type (due to increased mitochondria in the cytoplasm of the cell).

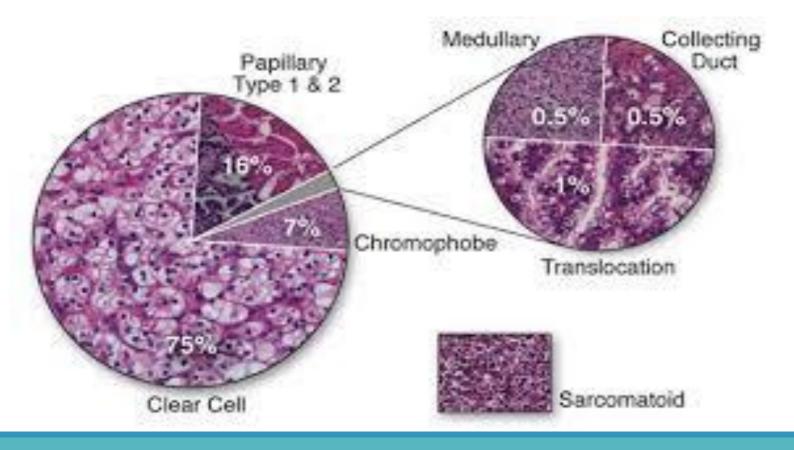
b. Vascular CT. •



- By <u>Direct</u> extension to adrenal gland (7.5% in tumors >5 cm), through the renal capsule, into the renal vein , inferior vena cava (IVC), right atrium
- By Lymphatics to hilar and para-aortic lymph nodes

Hematogenous to lung (75%), bone (20%), liver (18%), and brain (8%). So most common organ metastasis to the Kickney is the lung from kidney to lung un add N

Histological classification of RCC



Histological classification of RCC :

<u>Conventional (70–80%):</u> arise from the proximal tubule; highly vascular; <u>cells</u> <u>clear (glycogen, cholesterol</u>) or granular (eosinophilic cytoplasm, mitochondria)

• Papillary (10–15%): papillary, tubular, and solid variants; 40% multifocal

• <u>Chromophobe (5%)</u>: arises from the cortical portion of the collecting duct

• <u>Collecting duct (Bellini)</u>: rare; young patients; poor prognosis

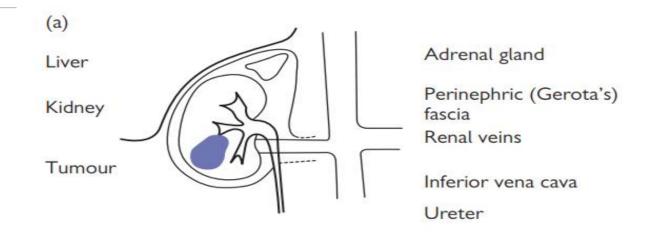
• <u>Medullary cell</u>: <u>rare</u>; arises from calyceal epithelium; young, Black, sickle- cell sufferers; poor prognosis

Grading is by the Fuhrman system

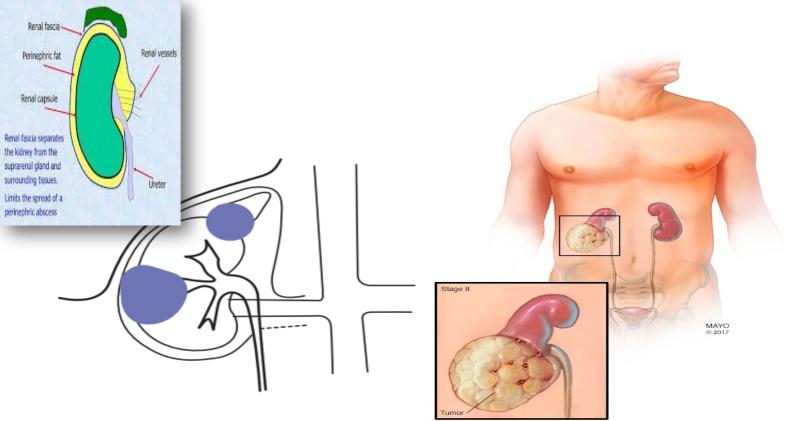
** based on nuclear size , outline, and nucleoli

Grade 1 = well-differentiated Grade 2 = moderately differentiated Grade 3 and 4 = poorly differentiated

is the most important prognostic indicator for RCC

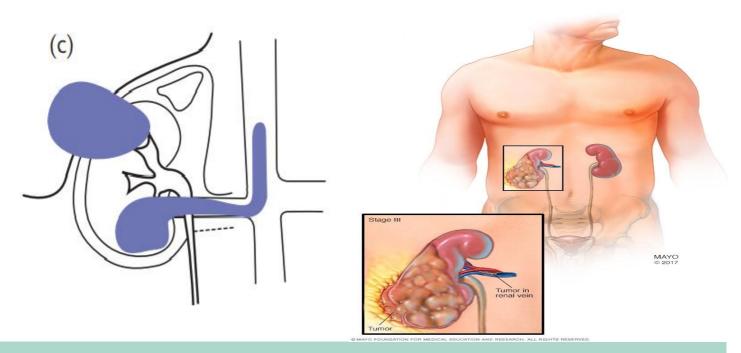


Primary tumor is limited to kidney (T1/T2). T1a is < 4 cm And T1b >4 cm But not >7 cm T2 is >7 cm



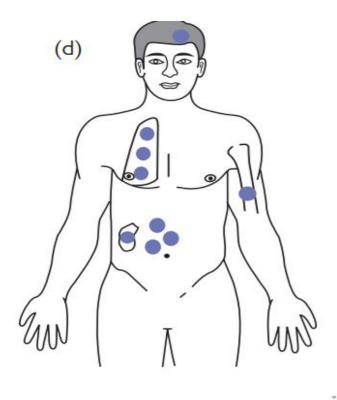
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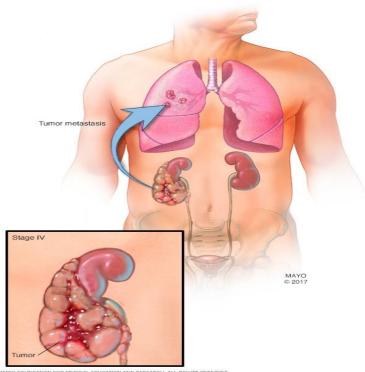
(b) Primary tumor invades perinephric tissue but not beyond perinephric fascia or invades adrenal gland (T3a)



(c) Primary tumor extends into renal veins or IVC <u>below</u> the diaphragm (T3b);

above the diaphragm into right atrium or invades wall of vena cava (T3c); or outside perinephric fascia (e.g., into liver, bowel, or posterior abdominal wall) (T4)





(d) N and M staging: involves multiple para-aortic or paracaval nodes; pulmonary, bone, or brain metastases (T1–4N2M1)

(a)		Table 6.13 TNM staging of RCC	
Liver	Adrenal gland	Tx	Primary tumor cannot be assessed
Kidney Tumour	Perinephric (Gerota's) fascia	ТО	No evidence of primary tumor
	Renal veins	T1	Tumor <7 cm, limited to the kidney
	Inferior vena cava limitted to		Tumor is <mark>4 cm or less</mark> , limited to kidney
	Ureter kidney.	Т <mark>1</mark> Ь	Tumor <mark>>4 cm but <7 cm</mark> , limited to kidney
(b)		T2	Tumor >7 cm, limited to the kidney
		T3	Tumor extends outside the kidney, but not beyond Gerota's (perinephric) fascia
		T3a	Tumor invades all denal gland or perinephric fat
		ТЗЬ	Tumor grossly extends into renal vein or subdiaphragmatic IVC
		T3c	Tumor grossly extends into supradiaphragmatic IVC or heart; invades wall of vena cava
	Adrenal gland	T <u>4</u>	Tumor invades beyond Gerota's fascia
(-) @)(@)		Nx	Regional (para-aortic) lymph nodes cannot be assessed
		N0	No regional lymph node metastasis
		N1	Metastasis in a single regional node
		N2	Metastasis in 2 or more regional nodes
		Mx	Distant metastasis cannot be assessed
		MO	No distant metastasis
ZWI X	I WS	M1	Distant metastasis present

prognosis -->Depends on T classification & staging

Box 6.5 Prognosis-5-year survival							
 Organ-confined 	T1a	90-100% Best survival rate					
-	T1b	80–90%					
	T2	70–80%					
 Capsular transgression/adrenal 	T3a	60–70%					
 Renal vein or IVC thrombus 	T3b/c	50–80% (25% with IVC wall invasion)					
• Visceral/lymph node involvement	T4 or N+	5–30%					
 Distant metastasis 	M+	5–100%					
Nomograms have been developed to predict risk of recurrence							

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Clinical presentation

50% detected incidentally (mc) U/s or CT

symptomatic presentation

✤ 50% of patients present with <u>hematuria</u>

♦ 40% with flank pain

30% of patients notice a mass

10% patients exhibit the classic triad of hematuria, flank pain, and mass

Clinical presentation

Symptomatic presentation

25% have symptoms or signs of <u>metastatic disease</u> (bone pain, night sweats, fever, fatigue, weight loss, hemoptysis)

pyrexia of unknown origin (9%),

acute varicocele 5%

Iower limb edema

10-40% paraneoplastic syndromes*, with any disease stage

^yparaneoplastic syndromes

Table 6.14 Paraneoplastic syndrem	omes due to release of certain hormous
Syndrome associated with RCC	Cause
Anemia	Hematuria, chronic disease
Polycythemia	Ectopic secretion of erythropoietin
Hypertension (25%)	Ectopic secretion of renin, renal artery compression, or AV fistula
Hypoglycemia	Ectopic secretion of insulin
Cushing	Ectopic secretion of ACTH
Hypercalcemia (10–20%)	Ectopic secretion of parathyroid hormone-like substance
Gynecomastia, amenorrhea, reduced libido, baldness	Ectopic secretion of gonadotrophins
Stauffer syndrome: <u>hepatic</u> dysfunction (increased liver function testing), fever, anorexia	Unknown; resolves in 60–70% of patients post-nephrectomy Treated by excising tumor by partial or radical nephrectomy

Clinical examination

may reveal <u>abdominal mass, cervical</u> <u>lymphadenopathy</u>, <u>non-reducing varicocele</u>, <u>or</u> <u>lower limb oedema</u> (both suggestive of venous involvement).

Investigation :

CBC **Full blood count** may reveal polycythemia or anemia.

KFT *** kidney function test** Serum creatinine and electrolytes, calcium,

LFT *****liver function tests

Urine cytology and culture: should be normal.

 $Urine analysis \rightarrow$ hematuria

Investigation:

Radiological evaluation :

• Abdominal – pelvis US: cyst or solid , biopsy for diagnosis and staging. > best modulity to diagnosis Pt with RCC CT scan : before and after contrast , most important investigation Urinary tract with IV contrast any solid-enhancing renal mass is considered a renal carcinoma until proven otherwise!! FNA, or CT quided FNA (biopsy) => CI in pt suspected remail mass, except: *Lymphadenopathy >2 cm is highly indicative of metastases

- inferior vena cava or renal vein (MRV)
- Iocally advanced disease
- renal insufficiency

OMRI: with gadolinium contrast for imaging the:

• Plain urinary tract (PUT) obliterated psoas shadow.

○<u>IVU</u> pelvi-calyceal system is → Dilated, Enlarged Amputated Distorted (DEAD))

• **Renal angiography** differentiates benign from malignant (now replaced by <u>CT</u>), also, Angiography may be helpful in planning partial nephrectomy or surgery for horseshoe kidneys

OCT-guided fine needle aspiration (FNA) : limited value !

□ false-negative biopsy results

□risks of hemorrhage (5%)

Utumor spillage (seeding)

olf doubt persists, **isotope renography** is used.

***When RCC is diagnosed radiologically, staging chest CT will follow and bone scan, if clinically indicated. Any suggestion of renal vein or IVC involvement on CT may be further investigated with Doppler USS or MRI.

Treatments modalities :

According to size & depend if localized or metastasis

* Localized disease

/radical nephrectomy : <u>*F*>4</u>cm

•remove the kidney with reveal capsule eportue for but & with gerotus fascia, with excise upper helf of under, ± LN ± Adrenal gland • Open approach:

**gold-standard curative treatment of localized RCC !

- 1. Transperitoneal, thoracoabdominal, flank incision
- 2 excise the kidney with the perinephris (Gerota fascia)
- 3. **ipsilateral adrenal gland**: tumors >5 cm , upper pole tumors , evidence of adrenal invasion
- With **regional nodes**: hilar , para-aortic , paracaval 4.

Laparoscopic approach: transperitoneal, retroperitoneal: masses of up to 10–12 cm

partial nephrectomy :

•<u>multifocal, bilateral tumors</u>, particularly if the patient has <u>VHL</u> syndrome or <u>single functioning kidney</u>

•**small** (<4 cm) tumors, even with a normal contralateral kidney

•local recurrence!

Tumor ablation therapy : thermal ablation by heating (radiofrequency ablation, or RFA) and cryotherapy



Metastatic RCC :

lung, liver, bone subcutaneous sites, and central nervous system

Palliative

Nephrectomy for palliation of symptoms (pain, hematuria)

≻if inoperable, arterial embolization can be helpful

>Hormone therapy and cytoxic chemotherapy : have little role

<u>Radiotherapy</u>: is useful for palliation of metastatic lesions in <u>bone and brain</u>, and in combination with surgery for <u>spinal cord compression</u>

<u>نمان</u> : Immunotherapy الم

•IL-2 (Aldesleukin)

Interferon A-2b

boz RCC very vascular & Plaluce lots of vessles

Molecular targeted therapies : Sunitinib, Sorafenib, Temsirolimus, Everolimus, Bevacizumab

Palliative care : megestrol acetate , steroids

**<u>Resection of</u> a solitary metastasis is an appropriate option for a small number of patients, usually a few months after nephrectomy, to ensure the lesion has remained solitary

Nephroblastoma (wilm's tumour)



Pathologically contains renal tissue with various degree of differentiation

Produces a mixed histological picture of epithelial structures

Resembling tubules and variety of mesenchymal tissues

Affect 1:10000 live birth , it is the 4th malignancy in children, represent 10% of childhood malignant tumour

Almost equal in **both sex**

It is may be familial in 1-2% of cases

 \searrow

60% presented before the age of three years

90% presented as abdominal mass

10% of tumours are bilateral

40% have **metastatic spread** at presentation , but don't prevent cure

Diagnosis : can be confirmed by **US** and **CT** scan

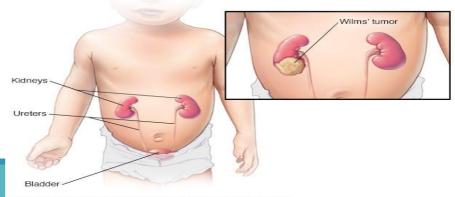
Treatment : with nephrectomy and postoperative chemotherapy and radiotherapy



Prognosis :

Stage 1 (localized to kidney) has 3years survival of >90% and cure rate 90%

Stage 4 (Hematogenous spread) has 3years survival less than 30%



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	AAILM 2	RUU		
Incidence	- 10% of childhood malig. - 3 – 4 yrs old - ♂ = ♀	- 75% of renal tumors - 50 – 70 yrs old - More in ∂		
Pathology	 Bilat 5 – 10 % Embryonic tissues Early capsule & Late pelvis Mixed tumor 	- Mainly untilateral - PCT - Early pelvis & Late capsule - Adenocarcinoma		
C/P	 Main present: abd. Mass Associated \$: familial types pain, hematuria, HTN. 	- Main present: hematuria - Associated \$: paramalignant - pain, mass, 2ry varicocele		
Investigations	- IVP: displaced pelvi-calyceal system (rarely invaded) U/S + CT + Ascending pyelogram + PUT + Staging + Preoperative→			
Treatment	 Operable: Radical resection Inoperable: chemotherapy then resection + radiotherapy for malignant LN → Post-op.: chemotherapy or radiotherapy 	Radical nephrectomy. In advanced tumors: symptomatic TTT		
Prognosis	80 % 5 year survival	- 60 % 5 year survival - 70% 3 year survival		

Secondary renal tumors

<u>lung</u> (20%)

breast (12%)

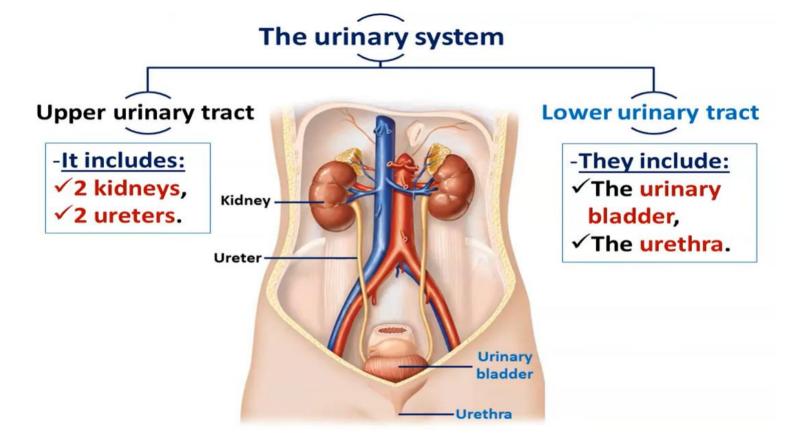
stomach (11%)

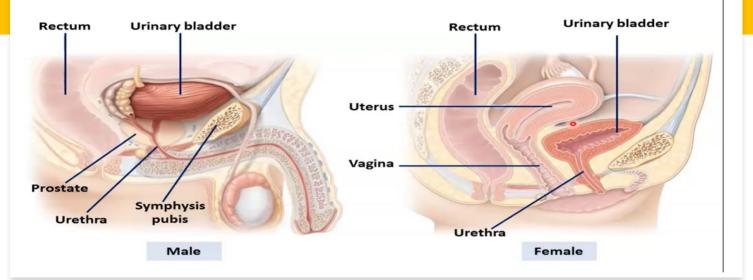
Albuminuria and hematuria are common

pain and renal insufficiency are rare

Bladder Cancer

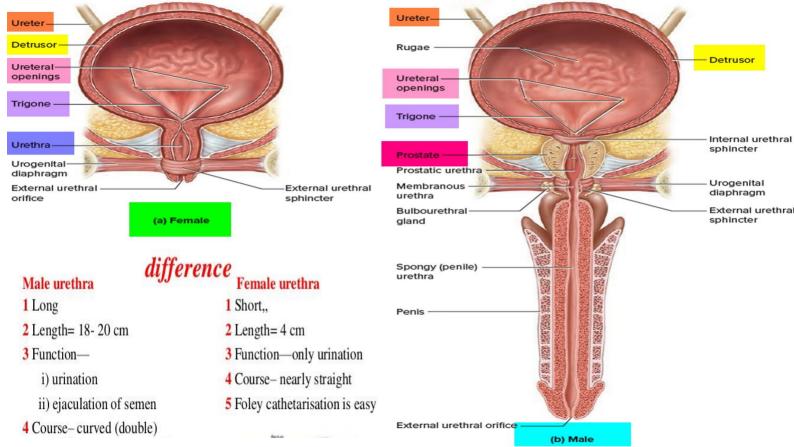
Presented by : Sondos Qatawneh Wafa Mohammad Neveen Bderat

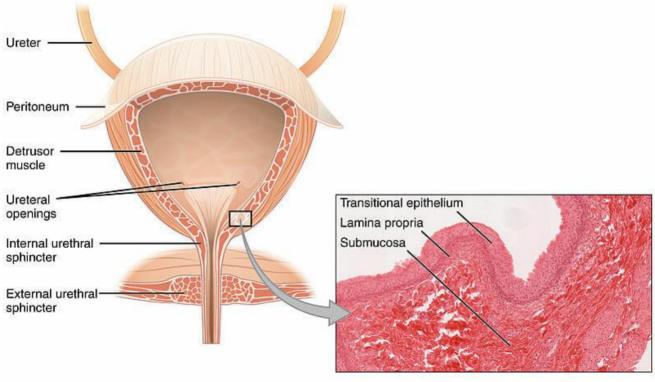




Urinary Bladder

- The urinary bladder is a hollow, collapsible muscular sac that <u>stores</u> and <u>expels</u> urine
- Collect urine (500-700 ml)
- <u>situated in</u> the pelvic cavity posterior to the pubic symphysis.
- In males, it is directly anterior to the rectum
- in females, it is anterior to the vagina and inferior to the uterus





What is Bladder Cancer?

- Bladder cancer is the second most common urological malignancy, accounting for 5000 deaths in the UK in 2001. This represents 3% of all cancer deaths.
- Incidence is ~13,000 per year, indicating that the majority of patients have curable or controllable disease.



Risk factors

> Smaking & they have greater unin residual in bladder post uningtion

- •<u>Men</u> are 2.5 times more likely to develop the disease than women, may be associated with greater urine residuals in the bladder.
- <u>Age</u> increases risk , most commonly diagnosed in the <u>8th</u> decade and rare <50 years.
- <u>Racially</u>, <u>Black people have a lower incidence</u> than White people, but inexplicably they appear to carry a poorer prognosis.
- Environmental carcinogens found in urine, are the major cause of bladder cancer.

- <u>Chronic inflammation of bladder mucosa</u>: <u>bladder stones</u>, <u>long</u>-<u>term catheters</u> - praphysic, imapplized or Geneticed at (isk of developing squamous cell type of bladder CA
- <u>Smoking</u> is the major cause of bladder cancer in the developed world > 10, to back N after quil smoking
- Occupational exposure to carcinogens, in particular aromatic hydrocarbons like aniline
- <u>Drugs</u>: phenacetin and cyclophosphamide.
- Pelvic radiotherapy. Especially for females with cervical cancer

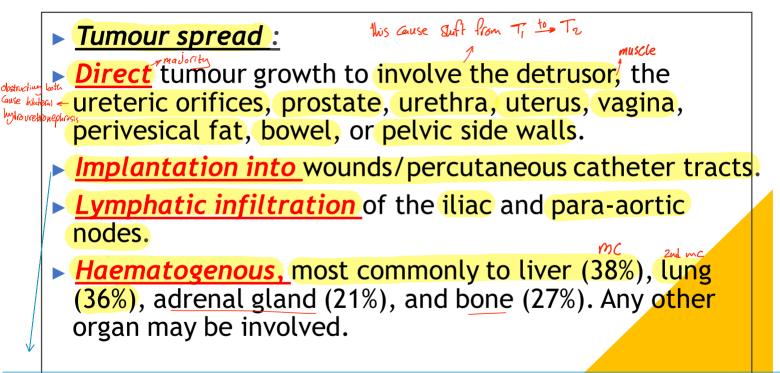
Bladder cancer: pathology and staging

- Benign tumors of the bladder, including inverted papilloma and nephrogenic adenoma, are uncommon.
- The vast majority of primary bladder cancers are malignant and epithelial in origin.

transitional

Types of Bladder Cancer

- >90% are transitional cell carcinoma (TCC)
- 7% are squamous cell carcinoma (SCC)
- 75% are SCC in areas where schistosomiasis is endemic (irag & egypt)
- 2% are adenocarcinoma
- Rarities include phaeochromocytoma, melanoma, lymphoma, and sarcoma arising within the bladder muscle
- Secondary bladder cancers are mostly metastatic adenocarcinoma from gut, prostate, kidney, or ovary



that is why if a pateint arrived to ER with urin retention and you will not able to inserte foly's catheter due to strecture or BPH and he's known to have bladder CA it is CI to insert Subrapubic catheter (Cystostomy); bcz of frisk of percutanous catheter tract seeding, therfore you should admite the petient and do Cystoscope and insert a foly's catheter under vision

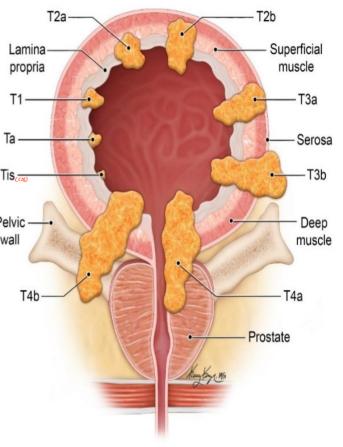
Histological grading

is divided into: well, moderately, and poorly differentiated (abbreviated to G1, G2, and G3 respectively).

Staging

is by the TNM (1997) classification. All rely upon *physical examination and imaging*.

I	Table 6	5.8 2002 TNM staging of bladder carcinoma	
	Tx	Primary tumor cannot be assessed	
	ТО	No evidence of primary tumor	
ť	Та	Noninvasive papillary carcinoma - Superlascial bladder ch (only on mulosc	a)
atheld lar	Tis	Carcinoma in situ	
Sure for	T1	Tumor invades subepithelial connective tissue	4
adder cA	M. invading	Tumor invades muscularis propria (detrusor): T2a inner half; T2b outer half	т
invasive bl	Т3	Tumor <mark>invades beyond muscularis propria into perivesical fat:</mark> T3a = microscopic; T3b = macroscopic (extravesical mass)	Т
× '	T4a	Tumor invades any of prostate, uterus, vagina, bowel	Pe
	T4b	Tumor invades pelvic or abdominal wall	W
	Nx	Regional (iliac and para-aortic) lymph nodes cannot be assessed	
	N0	No regional lymph node metastasis	
	N1	Metastasis in a single lymph node <2 cm in greatest dimension	
	N2	Metastasis in a single lymph node 2–5 cm or multiple nodes <5 cm	
	N3	Metastasis in a single lymph node or multiple nodes >5 cm in greatest dimension	
	Mx	Distant metastasis cannot be assessed	
	MO	No distant metastasis	
	M1	Distant metastasis present	



<u>Urothelial carcinoma</u> (transitional cell carcinoma)

Urothelial carcinoma (transitional cell carcinoma) UC may be single or multifocal.

Because 5% of patients will have a synchronous upper tract UC and metachronous recurrences may develop after several years, the urothelial fi eld-change theory of polyclonality is favored over the theory of tumor monoclonality with implantation (seeding).

Upper urinary tract cancer can present with bladder cancer in 20-30%
 because of urine flow which leads to malignant cells implantation, while
 *bladder cancer can present with upper urinary tract cancer in 2-3%.

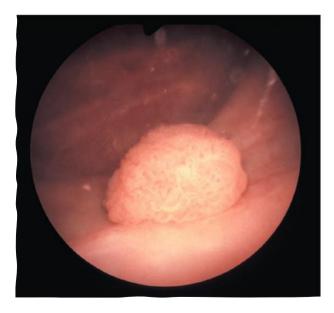
- Primary TCC is considered clinically as superficial or muscleinvasive:
- 70% of tumours are papillary, usually G1 or G2, exhibiting at least 7 transitional cell layers covering a fibro-vascular core (normal transitional epithelium has ~5 cell layers).
- Papillary TCC is usually superficial, confined to the bladder mucosa (Ta) or submucosa (T1). 10% of patients subsequently develop muscleinvasive or metastatic disease.
- However, a subset of superficial TCC, G3T1 tumours, are more aggressive, with 40% subsequently upstaging.
- 10% of TCC have mixed papillary and solid morphology and 10% are solid. These are usually G3, half of which are muscle-invasive at presentation.

CS superfascial bladder CA

- 10% of TCC is flat carcinoma in situ (CIS). This is poorly differentiated carcinoma, but confined to the epithelium and associated with an intact basement membrane. 50% of CIS lesions occur in isolation; the remainder occur in association with muscleinvasive TCC.
- CIS usually appears as a flat, red, velvety patch on the bladder mucosa; 40% of such lesions are CIS, the remainder being focal cystitis of varying aetiology.

* Cystoscopy: Chik for bladder (A if pt presented with painless hematuria

Cystoscopy show a superficial papillary tumor



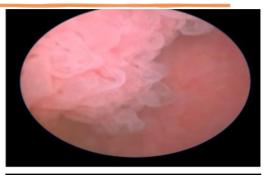


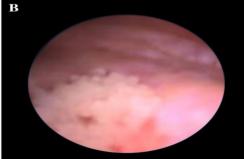


- The cells are poorly cohesive, up to 100% of patients with CIS exhibiting positive urine cytology, in contrast to much lower yields (17-72%) with G1/2 papillary UC.
- From 40% to 83% of untreated CIS lesions will progress to muscleinvasive UC, making CIS the most aggressive form of superfi cial UC.

of bladder CA

- Papillary urothelial neoplasm of low malignant potential (PUNLMP) The World Health Organization (WHO) defines PUNLMP as a papillary urothelial tumor that resembles an exophytic urothelial papilloma but shows increased cellular proliferation exceeding the thickness of normal urothelium.
- They are typically small (1–2 cm) and have little, if any, cytological atypia.
- Treatment and follow-up are the same as for low-grade noninvasive urothelial carcinoma.







Poorer prognosis them transitional cell CA

Squamous cell carcinoma

SCC is usually solid or ulcerative and muscle-invasive at presentation. SCC accounts for only 1% of UK bladder cancers. SCC in the bladder is associated with chronic inflammation and urothelial squamous metaplasia, rather than CIS. In Egypt, 80% of SCC is induced by the ova of Schistosoma haematobium. 5% of paraplegics with long-term catheters develop SCC. Smoking is also a risk factor for SCC. *Long term Catheters bladder stores*

The prognosis is better for bilharzial SCC than for non-bilharzial disease, probably because it tends to be lower grade and metastases are less common in these patients.



- Adenocarcinoma is rare, usually solid/ulcerative and carries a poor prognosis. One third originate in the urachus, the remnant of the allantois, located deep to the bladder mucosa in the dome of the bladder.
- in the dome of the bladder. Adenocarcinoma is a long-term (10 years) complication of bladder exstrophy and bowel implantation into the urinarytract, particularly bladder substitutions and ileal conduits
 - There is association with cystitis glandularis, rather than CIS.

Bladder cancer: presentation

* without burning micluritian * v suprapubic pain * v loin pain onveltimidstream (from bladder)

Symptoms

- The most common presenting symptom (85% of cases) is painless haematuria. This may be initial or terminal if the lesion is at the bladder neck or in the prostatic urethra. 35% of patients >50 years and 10% <50 years with macroscopic haematuria have bladder cancer. History of smoking or occupational exposure is relevant.
- Asymptomatic microscopic haematuria, found on routine urine stick-testing. Up to 16% of females and 4% of males have stick-test haematuria: less than 5% of those <50 years, while 7-13% of those >50 years will have a malignancy.
- Pain is unusual, even if the patient has obstructed upper tracts, since the obstruction and renal deterioration arise gradually, Pain may be caused by locally advanced (T4 disease).

dysuria

- Filling-type lower urinary tract symptoms, such as urgency or suprapubic pain. There is almost always microscopic or macroscopic haematuria. This so-called malignant cystitis is typical in patients with CIS.
- Recurrent urinary tract infections and pneumaturia due to malignant colovesical fistula, though less common than benign causes (diverticular and Crohn's disease).
- More advanced cases may present with lower-limb swelling due to lymphatic/venous obstruction, bone pain, weight loss, anorexia, confusion, and anuria (renal failure due to bilateral ureteric obstruction).
- Urachal adenocarcinomas may present with a blood or mucus umbilical discharge or a deep subumbilical mass (rare).



- General examination may reveal pallor, indicating anaemia due to chronic renal impairment or blood loss.
- Abdominal examination may reveal a suprapubic mass in the case of locally advanced disease.
- Digital rectal examination may reveal a mass above or involving the prostate.

Bladder cancer: diagnosis and staging

- After a urinary tract infection has been excluded or treated, all patients with microscopic or macroscopic haematuria require investigation of their upper tracts, bladder, and urethra. Usually, renal ultrasound and flexible cystoscopy, performed under local anaesthesia, are first-line investigations.
- If these fail to find a cause, an IVU or CT scan and urine cytology are justified second-line investigations.
- Patients with predominantly filling-type LUTS, suprapubic pain, or recurrent UTI/pneumaturia should also have urine cytology and cystoscopy.

* Always the diagnostic modulity of choice in bladder CA is rigid or flexible cystoscope.

- CT scan before and after IV contrast is becoming the first-line radiological investigation of haematuria. It is faster and more sensitive than ultrasound or IVU in the detection of renal (parenchymal and urothelial) and ureteric tumours. However, it carries a higher radiation dose and is more expensive.
- CT scan also detects some bladder tumours, but may overcall bladder wall hypertrophy as tumour and will miss flat CIS and urethral pathology.
- Thus it cannot replace cystoscopy. If there is hydronephrosis in association with a bladder tumour, it is likely that the tumour is causing the obstruction to the distal ureter. This tends to be caused by muscle-invasive disease rather than superficial TCC.

- False -ve cytology is frequent (40-70%) in patients with papillary TCC, but more sensitive (90-100%) in patients with high-grade TCC and CIS. False +ve cytology can arise due to infection, inflammation, instrumentation, and chemotherapy.
- If all investigations are normal, consideration should be given to nephrological disorders that may cause haematuria, such as glomerulonephritis.
- Fluorescent in situ hybridization (FISH) (sensitivity 77%, specifi city 98%) and other tests such as NMP-22 (sensitivity 56%, specifi city 85%) may be helpful in the evaluation.

<u>Transurethral resection of bladder tumour (TURBT</u>)^{*} Jiagnostic & therapeutic

Staging investigations

- are usually reserved for patients with biopsy-proven muscle-invasive bladder cancer unless clinically indicated, since superficial TCC and CIS disease are rarely associated with metastases.
- Pelvic CT or MRI may demonstrate extra-vesical tumour extension or iliac lymphadenopathy, reported if >8mm in maximal diameter.
- Chest X-ray
- <u>Cnest X-ray</u>
 f we suspect have metastasis
 <u>Isotope bone scan</u> is obtained in cases being considered for radical treatment.
- Staging lymphadenectomy(open or laparoscopic) may be indicated in the presence of CT-detected pelvic lymphadenopathy if radical treatment is under consideration.

TCC MANEGMENT

Bladder cancer (non-muscle invasive TCC): surgery and recurrence Ta, Tis, T, TURBT - transvrithral = resection for bladder timer - followed by intravasical chemotherapy (mitomycin)

As a primary treatment, a visually complete tumor resection is adequate for 70% of newly presenting patients with Ta/T1 superficial disease. The remaining 30% of patients experience early recurrence, 15% with upstaging. Because of this, it is **standard care that all new patients receive adjuvant treatment with a single dose of post-operative intravesical chemotherapy (usually mitomycin).** *Complications* of TURBT are uncommon, including bleeding, sepsis, bladder perforation, incomplete resection, and urethral stricture.



Alternatives to TURBT

Transurethral cyst diathermy or laser are accepted, quicker and Coagulation less morbid procedure for ablating small superficial recurrences when obtaining tissue for histology is not considered necessary + Usually not done from first time, bcz you need firstly histology to diagnose bladder CA or Pt who have bleeding diathesis of pt are well known to have bludder

Follow-up after TURBT

within 2 3min

Second resection: an early repeat TUR (within 2–6 weeks) should be undertaken:

(a) if the first resection was incomplete (very larg tumor, or complication happen (bleeding))

(b) when the pathologist reports that the resected specimen contains no muscularis propria

(c) if a high-grade, but apparently non-invasive, T1 tumor has been reported since perhaps 10% (3–25%) of high grade T1 tumors are under staged T2 tumors. This strategy improves recurrence-free survival and prognosis while complications include bladder perforation.

Patients with G3T1 TCC, and CIS are at significantly higher risk of recurrence and 40% subsequently upstage. Some patients experience persistent symptomatic multifocal G1/2, Ta/1 recurrent TCC, demanding frequent follow-up procedures. In these circumstances, *adjuvant treatment* is Indicated.

There is no accepted protocol for *upper tract surveillance* in patients with a history of bladder TCC, although EAU guidelines1 recommend yearly imaging (CTU) for patients with high-risk disease.

ADJUVANT THERAPY

after resection A bladder tunor, you need to give Intravesical chemotherapy (e.g. mitomycin C (MMC)

decreases rate of recurrence]

For many patients at low risk of recurrence, the risk reduction seen with single-dose postoperative chemotherapy is equivalent to that seen using weekly instillations for 6 weeks, commencing up to 2 weeks post-TURBT. Such longer courses are still recommended seen using weekly instillations for 6 weeks, commencing up to 2 recurrences, excluding those with high-grade Ta/T1 TCC or CIS

intravasial mitomucin C

low glad bladder CA

ADJUVANT THERAPY

Ite alternated vaccine Intravesical BCG [decreases rate of progression]

BCG produces complete responses in 60–70% of patients. It is as effective as chemotherapy for adjuvant treatment of low- and intermediate risk TCC, therefore, is not often used (except as second-line) because of the additional toxicity.

I usually used for pts with G, T, (high grade superfascial bladder A) & for CIS \$ not immidecte with in 6n/ Just in 6w coarse, every 1w

of 12 recurrence, 11 progression of d. (trisk of progression to M. invassive bladder (A.) benitik over mitomycin

\$ If pt failed after intravasical BCO <u>Chemotherapus</u> (high grade Ti or CIS), if recurrence happen 2 , bcc they have 40-83! we proceed directly into radical cystectory ; bcc they have 40-83! Upgrade fisk to develop into M. invassive

Contraindications to intravesical BCG include:

- Immunosuppressed patients.
- Pregnant or lactating women.
- Patients with haematological malignancy.
- Following traumatic catheterization

muscle invasive TCC T₂, T₃, T₄ Very Jugarous (Tisk of mortality)

radical custortony a with illial incontenent type of unin diversion

temporary JJ

inserted inte

Radical cystectomy with:

in males ; excision of bladder + prostate + seminal Auid + vas diference + destal uretrectomy + urithra, peluic LN [external illiac in timales ; in addetion to uterus, anterior vagina & bilateral ovaries (peluic exenteration)

most commentered conduit urinary diversion (stormer) - incontement type of diversion

• Ureterosigmoidostomy urinary diversion

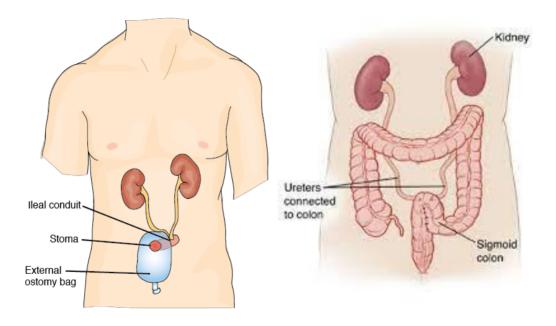
• Continent urinary diversion - Catheterizable Store or

at blodder preservines - rare While usually use partial cystecters in adeno carciname



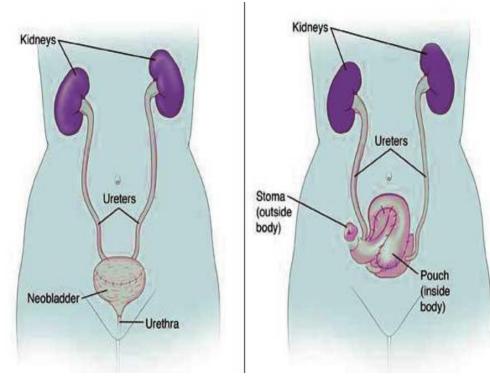
Radical cystectomy:

 complete removal of the urinary bladder, pelvic lymph nodes; external/internal iliac LNs and obturator LNs, and adjacent organs; (prostate, seminal vesicle and proximal part of urethra in males / ovaries, fallopian tubes, uterus, cervix and anterior vaginal wall in females)



Ileal conduit urinary diversion

Ureterosigmoidostomy urinary diversion



Continent urinary diversion

for cystectomy alone
90%+
55–63%
31–40%
10–25%
30%
70%
50%
25%

P

Palliative treatment

RT is effective for *metastatic bone pain* or to palliate symptomatic (bleeding) local tumor.

radictlesapy

Intractable haematuria may be controlled by intravesical formalin or a Alum, hyperbaric oxygen, bilateral internal iliac artery embolization or ligation, or palliative cystectomy/diversion bilateral Ureteric obstruction may be relieved by percutaneous nephrostomy autigrade Involvement of the palliative care team can be very helpful to the patient and family.

Benign prostatic hyperplasia

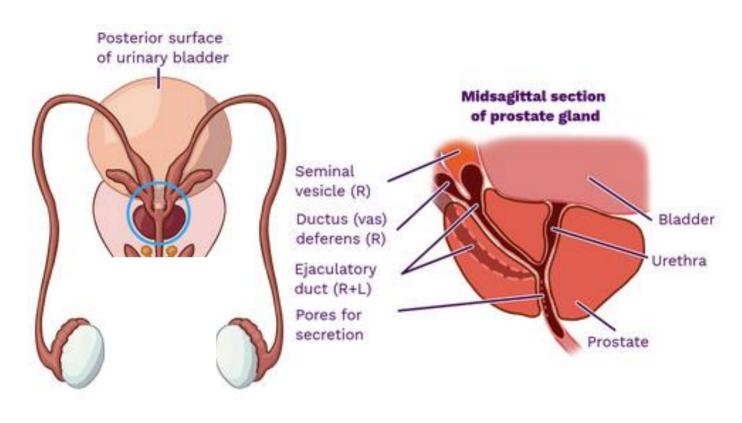
Presneted by : Quataiba Altarawneh Auwid Alshra'ah Omar kafaween

BPH after 40y - Slowly inlargmen of prostate

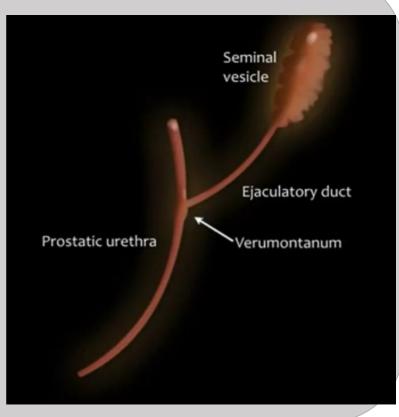
Surgical anatomy

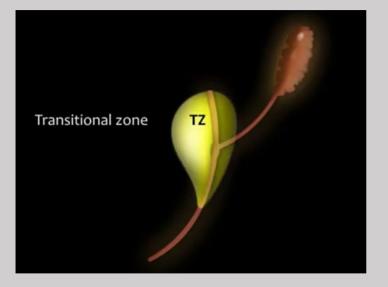
- The prostate is made up of several zones which fit into each other like an egg in an egg cup
- Benign enlargement mainly arises in the <u>transition</u> zone , <u>cancer</u> in the <u>peripheral</u> zone .

A prostate 2 component < strand component ~ precise piblicial ~ ~ prijerplagia



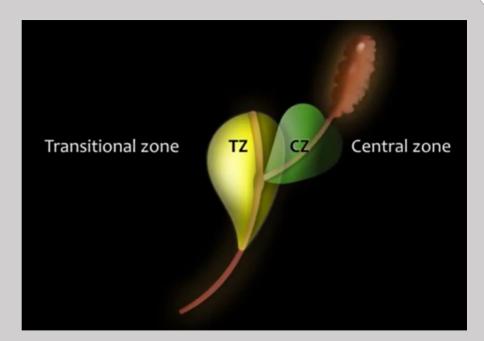
- The ejaculatory duct end in the prostatic urethra at the <u>verumontanum</u>
- Prostatic zones are :
- 1. Transitional
- 2. <u>Central</u>
- 3. <u>Peripheral</u>
- 4. Anterior fibromuscular

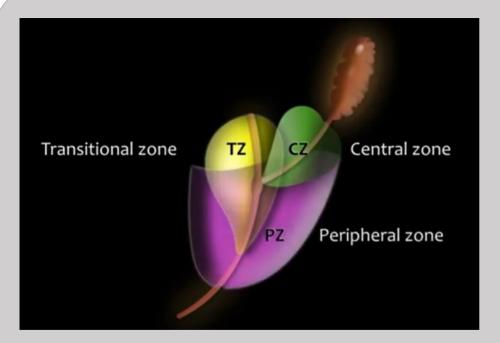




The **Transitional** • **zone** surrounds the prostatic urethra, 5% of the volume of the young adult prostate "This zone enlarges in aging

men resulting in **Benign Prostatic Hyperplasia**" The <u>Central zone</u>
 lies behind the
 transition zone and
 <u>surrounds the</u>
 <u>ejaculatory</u> duct ,
 25% of the volume
 of the young adult
 prostate

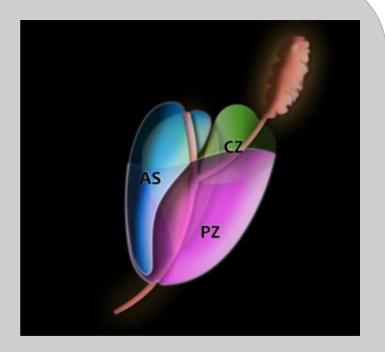


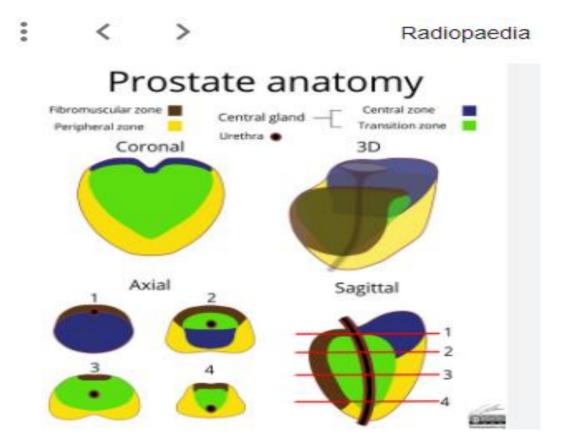


The **Peripheral zone** is the largest area and situated on the posterior and lateral side ,70% of the volume of the young adult prostate

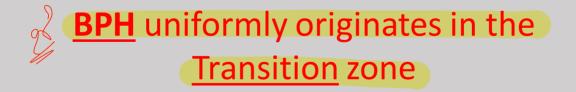
٠

 The <u>Anterior fibromuscular</u> stroma is a thickened area of tissue , it surrounds the <u>base and midportion</u> of the prostate on the anterior side



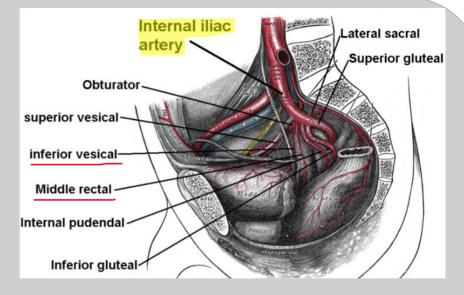


60-70% carcinoma of the prostate (CaP) originate in the <u>peripheral</u> zone , 10-20% in the <u>transition</u> zone and 5-10% in the <u>central</u> zone



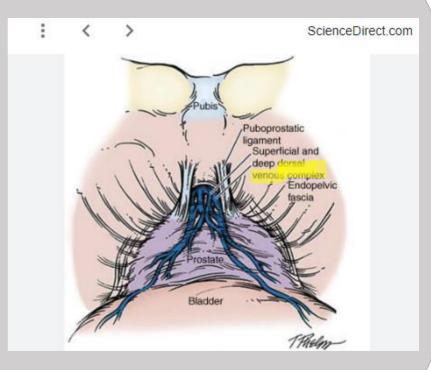
• Blood supply :

Derived from branches of the <u>internal iliac artery</u> (<u>inferior vesicle & middle</u> <u>rectal arteries</u>)



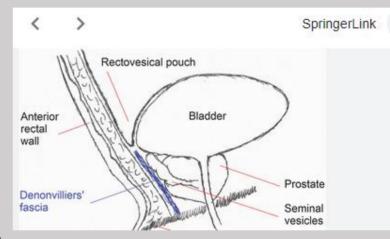
• Innervation : From <u>pelvic plexus</u> • Venous drainage :

Via the <u>dorsal venous complex</u>, which receive the deep dorsal vein of the penis before draining in the internal iliac vein



Anatomical relations

- Anterior to the prostate is the symphysis pubis
- Posteriorly, the prostate is separated from the rectum by the fascia of Denonvilliers.
- Behind and above the prostate lies the bladder, seminal vesicle, vasa deferntia and ureters



 It comprises the most proximal aspect of the urethra

Incidence & Epidemiology

- BPH is the most common benign tumor in the men , and its incidence is age related .
- The prevalence of histologic BPH in autopsy studies rise from approximately 20% in men aged 41-50 to 50% in men aged 51-60, and over 90% in men older than 80
- Some studies have suggested a genetic predisposition, and some have noted racial differences as risk factor for development of BPH
- Heritable form of BPH is most likely autosomal dominant trait , and first-degree male relatives carry an increased relative risk of approximately 4 folds



- The etiology of BPH is not completely understood , but it seems to be multifactorial and endocrine controlled .
- The prostate is composed of both stromal and epithelial elements , and each can rise to hyperplastic nodules and symptoms of BPH
- A positive correlation between levels of free testosterone and estrogen and the volume of BPH was found

* enlargment - compress viething nor outlet resestant = obstructive symptoms (voiding symp.)

DIAGNOSIS OF BPH: CLINICAL FEATURES

 Compression of the prostatic urethra and the way in which the bladder responds to obstruction are the main factors involved in symptom generation.

Poor stream

Hesitancy

Mainly.

- Straining to urinate intermelancy
- Incomplete voiding

Voiding symptoms:

• Double voiding (urinating a second time within 2 hours of the previous void)

· Urinary retention

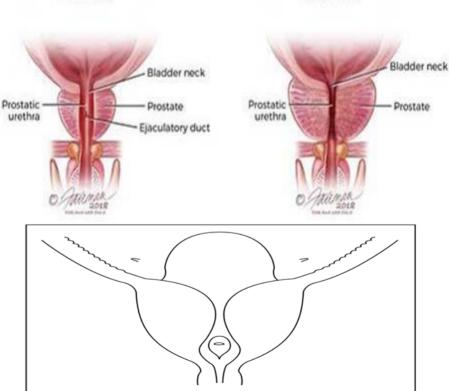
• Terminal dribbling

Irritative symptoms: Storage Symp.

- Frequency
- Urgency
- Nocturia

NORMAL

 The Irritative voiding complaints of BPH results from the <u>middle lobe</u> <u>protrusion into the</u> <u>bladder</u> causing obstruction to the bladder outlet.



ENLARGED

 Lower urinary tract symptoms and their detrimental impact on the patient known as 'bother' are quantified and documented using the International Prostate Symptom Score (IPSS).

It focuses on 7 items that asks patient to quantify the severity of their obstructive or irritative complaints on a scale of 0-5. Thus the score can range from 0-35.

The IPSS valid & reliable in <u>identifying the need to treat patient</u> and in <u>monitoring their</u> <u>response to therapy</u>.

A symptom score of:

- 0-7 is considered mild
- 8-19 is considered moderate
- 20-35 is considered severe

International Prostate Symptom Score (I-PSS)

Patient Name:	Date of birth:	Date completed

In the past month:	Not at all	Less than 1 in 5 times	Less than half the time	About half the time	More than half the time	Almost always	Your score
1. Incomplete emptying How often have you had the sensation of not emptying your bladder?	0	1	2	3	4	5	
2. Frequency How often have you had to urinate less than every two hours?	0	1	2	3	4	5	
3. Intermittency How often have you found you stopped and started again several times when you urinated?	o	1	2	3	4	5	
4. Urgency How often have you found it difficult to postpone urination?	0	1	2	3	4	5	
5. Weak stream How often have you had a weak urinary stream?	0	1	2	3	4	5	
6. Straining How often have you had to strain to start urination?	0	1	2	3	4	5	
	None	1 Time	2 Times	3 Times	4 Times	5 Times	
7. Nocturia How many times did you typically get up at night to urinate?	0	1	2	3	4	5	
Total I-PSS score							

Quality of life due to urinary symptoms	Delighted	Pleased	Mostly satisfied	Mixed	Mostly dissatisfied	Unhappy	Terrible
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?	o	1	2	3	4	5	6

P.E.

 On DRE of BPH result in a smooth, firm, non-tender, enlargement of the prostate. PR: enlarged Prostate, nodular, firm

- Complications of BPH include:
- 1. Retention
- 2. Overflow incontinence
- 3. Hydronephrosis
- 4. Renal insufficiency
- 5. Infection
- 6. Gross hematuria
- 7. Bladder stone

obstructive symp. + large prostate + PSA within normal + PR -> no hard nodules ~ Managment of BPH

DIAGNOSIS OF BPH: INVESTIGATIONS

- LABORATORY investigations:
- 1. A **urinalysis** to exclude infection or hematuria.
 - 2. Serum creatinine measurement to assess renal function are required. Renal insufficiency may be observed in 10% of patients.
 - 3. Serum PSA is considered optional but most physicians will include it in the initial evaluation.

4-CBC

- PSA is present in small quantities in the serum of men with healthy prostates, but is often elevated in the presence of prostate cancer or other prostate disorders.
- PSA is not a unique indicator of prostate cancer, but may also detect prostatitis or benign prostatic hyperplasia.

RADIOLOGICAL investigations:

- Upper tract imaging (intravenous pyelogram or renal ultrasound) is recommended only in the presence of concomitant urinary tract disease or complications from BPH (e.g.: hematuria, UTI, renal insufficiency, hx. of stone).
- 2. **CYSTOSCOPY** : It is not recommended to determine the need for treatment but may assist in choosing the surgical approach in patients opting for invasive therapy. ass. the efficacy of surgery we also ass. of bladder Sewer hypertrophy of M. (bc2 prolonged obstruction)
- 3. ADDITIONAL TESTS : Cystometrogram (pressure measurement inside the bladder) & urodynamic profile are reserved for patients with suspected neurologic disease or those who have failed prostatic surgery.

DIFFERENTIAL DIAGNOSIS

enlargement of prostate due to BPH, Prostate CA

- Other obstructive conditions of the lower urinary tract must be entertained when evaluating men with presumptive BPH, such as:
- Urethral stone
 Urethral stricture, Bladder neck contracture, (A history of previous urethral instrumentation, urethritis or trauma should be elucidated.)
- Bladder stone, (commonly associated with hematuria and pain.)
- CaP, (may be detected by abnormalities on the DRE (hard or lumpy) or an elevated PSA.)
- **Neurogenic bladder disorder**, (history of neurologic disease, stroke, diabetes mellitus, or back injury may be present as well. In addition examination may show diminished perineal or lower extremity sensation or alterations in rectal sphincter tone or constipation.)

· Petrosal Sphincter dyssynergia

¥ old male 50-60s \$ by investigation - no infection \$ U/S PR - Larg Prostate \$ PSA - within Normal Dx as BPH

Treatment

Conservative watch ful waiting

- Encourage fluid intake
- Program the bladder

Medical



- Smooth muscle relaxation
- Reduce epithetical hyperplasia

3 Conventional surgical

- TURP / TUIP
- Simple open prostatectomy.

Minimal invasive

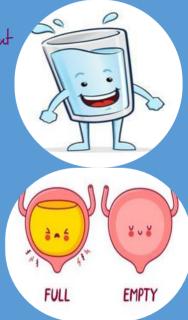
- Get a modern PowerPoint Presentation
- Easy to change colors, photos and Text.

Conservative (watch and wait)

➢ If no complications (no bladder diverticulum, bladder trabeculae or bladder stone) or (enal impairment)
 ➢ mild symptoms □ 0-7 score

Your advices:

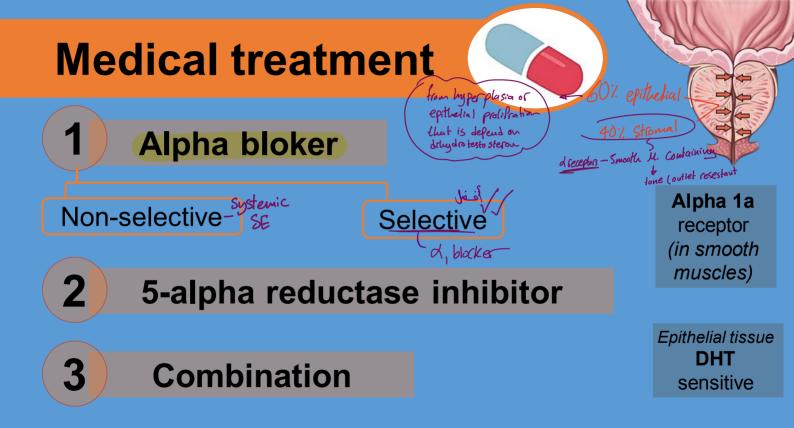
- 1. Adequate fluid intake
- 2. Timed bladder voiding (program the bladder) every 3.
- 3. Avoiding of the smoking or excessive caffeine



When the <u>treatment</u> is indicated ??

- > Bothersome symptoms + affect QOL (quality of lik)
- Complications
 - Acute urinary retention
 - Recurrent UTI
 - Recurrent hematuria

The size is not indication for treatment





Alpha blocker

Non-selective

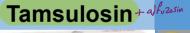
- Good response
- Systematic side effects*
 - 1) Orthostatic hypotension
 - 2) Dizziness
 - 3) Retrograde ejaculation

Phenoxybenzamine

Prazosin (short acting)

Selective

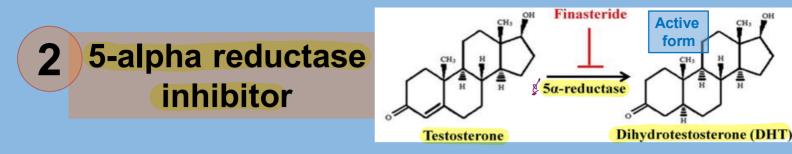
Block alpha 1a (specific)
Less Systematic S.E.





✓ Smooth muscle relaxation □
 ✓ Improvement within 24 h

decrease the outlet resistance



- Epithelial components depend on DHT
- Which will cause hyperplasia of the epithelial tissue
- Finasteride

 epithelial shrinkage



Shrinkage of the epithelial tissue block epithelial politration
Improvement within 2 month and the maximum efficacy within 9 month
Endeduction in Size & Plostate 20/

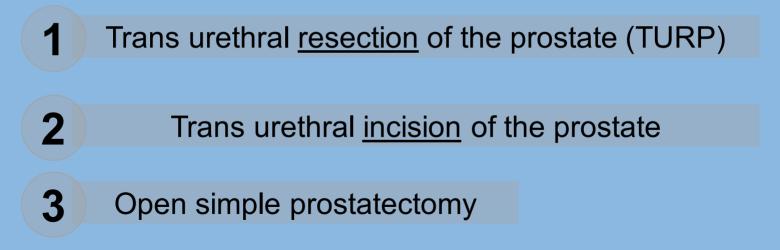


Alpha blocker + 5-alpha reductase inhibitor

4 Anticholinergic

- Just if there is <u>irritating symptoms</u> (urgency , frequency and urge incontinence)
- > But may precipitate **urinary retention**

Conventional surgical

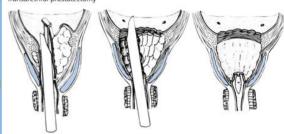


What is the indications for <u>surgery</u>??

- > Failing of the medical treatment persistant of bothering sym. dispite medical the
- Recurrent UTI
- Recurrent hematuria
- Recurrent urinary retention
- Bladder complications (diverticulae / stone)
- Kidney complications (elevated Cr / hydronephrosis)

Trans urethral <u>resection</u> of the prostate (TURP)

- Under GA or spinal anesthesia Insertion of the endoscope contain resection lobe
- Resection of the obstructing tissue (remove)
- > Irrigating fluid hypotonic (glycne & water)



- Improvement of the symptoms score and the flow rate (better than minimal invasive procedure)
- \checkmark but more complication (relatively higher mortality rate)
- The gold standard procedure for bladder outflow obstruction

Complications of the TURP:

- 1. Retrograde ejaculation (majority)
- 2. Impotence
- 3. **Incontinence** (especially men with storage symptoms)
- 24. TUR syndrome Rafe
 - 5. Urethral stricture
 - 6. Bleeding

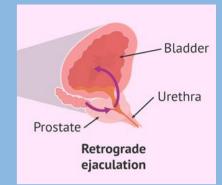
* under irrigative p. ~ virrigated fluid an enter to venous sinuses prosterie the to arculation Causing (*)

TUR syndrome:

Hypervolemic hyponatremic state due to absorption of the hypotonic irrigating fluid

Symptoms are : N+V, confusion, HTN, bradycardia and visual disturbances
 Treatment https://www.sevensule.com (in severe cases)

The surgery should not exceed 90 minute to avoid this syndrome





> endoscope contain **needle** (not resecting lobe)

Deep two Incisions at 7 and 5 o'clock

Lead to relaxation of the prostate

Open simple prostatectomy

- Open surgery (retropupic / transvesical)
 Simple (not radical)
 remove all the prostate tissue except the capsule (compressed peripheral zone)
 enculation of the prostate
- > This procedure does not lower the risk of prostate cancer

Indications:

- 1. Large prostate > 100g ~~ bcz, it needs longer fine => more risk of TUR syndrom
- 2. Bladder stone / diverticulum/inguival hernion
- 3. If lithotomy position is not possible (hip surgery / orthopedic surgery)
- 4. Long urethra / urethral stricture open Joint
- 5. Severe bleeding during the TURP open Jopen inculation Vesion) .

Minimally invasive procedure

- new 1. Laser therapy > blood less operation / very expensive
 - 2. Transurethral electrovaporization of the prostate
 - 3. Transurethral needle ablation of the prostate

 - 4. high-intensity focused ultrasound
 5. Intra-urethral stents (unco-operative patient)
 - 6. Transurethral balloon dilation of the prostate 7. hyperthermica



Prostate Adenocarcinoma Most common form of cancer in men (most prevelent for >65 ver) bc2 of T2 is 2nd most deadly (lung) arround ureture. early preservation (obstructive TZ, (5-10%) may arise from CZ bc2 of this ~> 545 BPH (postutectory) doesn't protect pt from prostate CP Symp. as BPH) Classically posterior lobe by investigation HPR-e land loop Mets to prostate are very rare HPA-e land loop Mets to prostate are very rare Local invasion Lymphatic Most common site for metastasis in Prostatic CA is bone (sclerotic lesion; purely osteoblastic)

Risk factors

Age (>65) <u>most imp</u>. African Americans

Family Hx

1st degree relative = 2X risk
1st and 2nd degree relatives = 9X risk
High dietary fat
Familial prostate CA gene ou chr 1
dust increasing the risk

Clinical features - typical prosentation to BPH

Early prostate cancer usually asymptomatic

If symptomatic:

Obstructive symptoms: hesitancy, decreased force and caliber of the stream, sensation of incomplete bladder emptying ,straining to urinate, postvoid dribbling. Irritative symptoms: frequency, urgency, nocturia

* presentation different in case of local invasion (metastasis): Back pain, incontinence

Bone pain (metastasis)

Leg pain and edema (nodal metastasis ; lymphatic and venous obstruction)

Prostate Adenocarcinoma Me best screening for prostate A: Investigations and Diagnosis: <u>PR</u> + <u>PSA</u>

Digital rectal exam (DRE) ; findings:

Nodularity with heterogenous texture

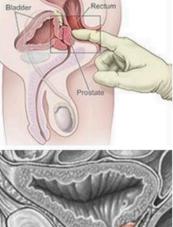
Stony Hard irregular surface

Absence of median sulcus - sit involved both loops

Asymmetry

Tethered rectal mucosa

50% of abnormal DREs are associated with prostate cancer, the remainder being benign hyperplasia, prostatic calculi, chronic prostatitis, or post-radiotherapy change



Prostate Specific Antigen (PSA)

- Enzyme released by prostate during ejaculation
- PSA is prostate-specific, but not prostate cancerspecific.
- Leaks into circulation < 4 ng/ml
 - Other causes of elevated serum PSA are :
 - BPH
 - UTI
 - Prostatitis
 - Prostatic massage
 - Acute urinary retention
 - Biopsy, TURP, surgery
 - Ejaculation
 - -DRE/foly's calleter: zation

PSA moto devate sensitivity a spicificity to test 5 - 2171. unfavorable

In prostate cancer the complexed PSA increases and the free decreases

<10% free PSA (of total) suggestive for cancer

>20% free PSA
suggests benign cause

Gauses of Unfavorable Catio
I prostate CA
I oral autilisis
I prostatitis
I prostatitis
I constantiation
I with Unfavorable Patio:
I oral autilisis

Satio - 2171. untavorable total •PSA density = Serum PSA/prostate volume >0.15 ng/ml/g associated with increase risk of cancer (total PSA) Tolil Usa • PSA velocity The amount of increase in the PSA level in one year, normally < 0.15 mg/year Change of >0.75 ng/ml/year associated with increased risk of cancer - rapid elevation in total PSA - suggestive of CA · PSA doubling time: for already diagnosed postate CA Pl's; to see if it is aggressive or not? / high or low risk?

PSA elevations may help in staging, and following up.

Age adjusted PSA reference ranges

PSA normal range(ng/ml)	age
0-2.5	40-49
0-3.5	50-59
0-4.5	60-69
0-6.5	70-79

Transrectal ultrasound (TRUS)

TRUS is useful in:

prostatic biopsies under TRUS guidance

- Staging information (detect extracapsular extension)
- Measurement of prostate volume.

TRUS-guided needle biopsy

 The decision to proceed to prostate biopsy should be based primarily on PSA and DRE results, but should take into account multiple factors (free and total PSA, pnt age, PSA velocity, PSA density, family Hx, ethnicity and comorbidities)

+ befor TRUS biopsy we prepare pt by antibiotic (indeclin gadamicine, levofloxacin begennes from 3days) + Clear fluid diet at night & fasting at the day of biopsy

Complications of prostatic biopsy

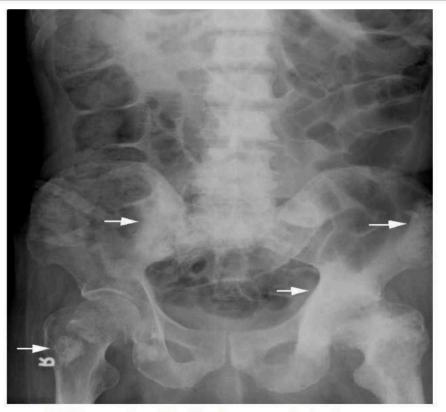
•Vaso-vagal, fainting immediately after the procedure.

MC

Septicaemia.

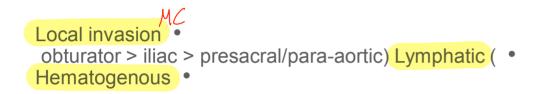
· A cute prostatitis

- Rectal bleeding.
- Mild haemospermia or haematuria, for up to three weeks.



Diffuse osteoblastic bone metastasis

Methods of tumor spread



Prostatic CA is the commonest site of origin for skeletal mets \rightarrow osteoblastic (sclerosis) other (A -> osteolytic



through CTscan & IV contrast / MRI

The **TNM system**, evaluates the size of the tumor, the extent of involved lymph nodes, and any metastasis.

Gleason's system - for grading-

** Used bcz prostate cA is heterogenouse porty differentiated well differentiated

Gleason's system -- J-5 grades

It is a system that relies upon the low-power appearance of glandular architecture under the microscope.

Primary grade - assigned to the **dominant pattern of the tumor** (has to be greater than 50% of the total pattern seen).

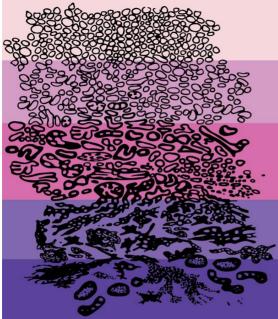
<u>Secondary grade</u> - assigned to the **next-most frequent pattern** (has to be less than 50%, but at least 5%, of the pattern of the total cancer observed).

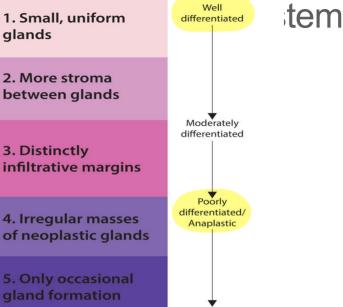
 f_{am} , Gleason sum \rightarrow is the addition of the primary and secondary glandular patterns present 2-10 on microscopic examination

Higher Gleason score are more aggressive and have a worse prognosis.

e.g: a Gleason 3+3 = 6 adenocarcinoma carries a worse prognosis than a 3+2 = 5 cancer of equivalent stage.

Gleason's Pattern





- tumour grade (Gleason score out of 10)
- 2-4 represent well differentiated
- 5-7 represent moderately differentiated
- 8-10 represent poorly differentiated

"T"- Primary tumor

TX: cannot evaluate the primary tumor
T0: no evidence of tumor
T1: clinically undetectable tumor, normal DRE and TRUS
T1a: tumor was incidentally found in less than 5% of prostate tissue resected (for other reasons)
T1b: tumor was incidentally found in greater than 5% of prostate tissue resected
T1c: tumor was found in a needle biopsy performed due to an elevated serum PSA
T2: palpable, confined to prostate
T2a: the tumor is in half or less than half of one lobe of the prostate gland's
T2b: the tumor is in more than half of one lobe, but not both
T2c: the tumor is in both lobes

way through, it is still **T3: the tumor extends through the prostatic capsule** (if it is only part-T2) T3a: the tumor has **spread through the capsule on one or both sides** • T3b: the tumor has **invaded one or both seminal vesicles** •

T4: the tumor has invaded adjacent structures





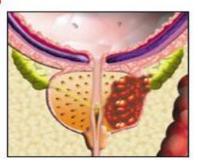


- Rectum

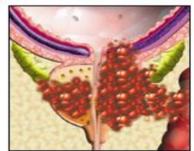
External urinary sphincter muscle



тз



Т4



"N" N0: no regional lymph node metastasis• N1: spread to regional lymph nodes•

"M"
M0: there is no distant metastasis •
M1: there is distant metastasis •
M1a: non-regional L.N •
M1b: the cancer has spread to bone •
M1c: the cancer has spread to other sites (regardless of •
bone involvement)

Prostate cancer mortality risk



 \mathcal{X} This is what determines the management of the patient

Prognostic factors : Tumor stage, grade , PSA value and PSA doubling time



Prognosis

- T1-T2: comparable to normal life expectancy
- T3-T4: 40-70% 10-yr survival
- N+ and/or M+: 4 % 5 yr survival

Treatment

 T1\T2 (localised,LOW-RISK) if young consider radical prostatectomy (<70 y.o.) brachytherapy or radiation.

Older patients treated with watchful waiting or hormone treatment (if PSA>20) → Rarely treat localised cancer in the over 80s

• T3\T4

Staging lymphadenectomy and radiation or hormonal

• N>0 OR M>0

Hormonal / palliative radiotherapy for mets Chemotherapy in advanced disease

- Of circulating androgen, 95%, mainly testosterone, is produced by the
- Leydig cells of **the testes** under the infl uence of luteinizing hormone (LH).
- The anterior pituitary synthesizes LH, stimulated by LH-releasing hormone
- (LHRH) produced by the hypothalamus. The remaining 5% of circulating
- androgen is synthesized by adrenal cortex from cholesterol

• All prostate epithelial cells are dependent on androgens to grow or undergo programmed cell death

- Androgen deprivation results in a reduction in PSA and clinical improvement in the majority of patients.
- However, most will still die within 5 years because of the development of androgen-independent growth

Hormonal Therapy (androgen deprivation):

this theese drugs given simultanously with

androgen receptor blocker

Mechanisms of androgen deprivation

- Surgical castration: bilateral orchiectomy loss 95% of androgen (remaining 5% from adrenal)
- • Medical castration: LHRH agonists, LHRH antagonists, estrogens
- • Antiandrogens (steroidal or nonsteroidal): androgen receptor blockade at most imp. : GuRH agonist (Continouse-injection Pattern) every 1 or 3 months target cell
- inhibitors of steroidogenesis
- Maximal androgen blockade (MAB): medical or surgical castration plus anti-HE AT the begining: Stimulation & enlarge Suddenly even can cause pathological feature; bcz of androgen

• Side effects of bilateral orchiectomy and LHRH agonists/antagonists

- • Loss libido
- • Hot flushes
- • Weight gain and obesity
- Gynecomastia
- Anemia
- mood changes
- • Metabolic syndrome (increased blood glucose and lipid profile)
- Osteoporosis and pathological fracture occur in patients on long-term treatment

Prognostic factors

Predictors of poor hormone therapy response include the following:

- More than 5 metastatic lesions
- • Elevated alkaline phosphatase
- Anemia at presentation
- • Poor performance status
- • Low serum testosterone
- • Failure of bone pain to improve within 3 months of treatment
- • Failure of PSA to normalize within 6 months of treatment

Complications

• These include ED, urinary retention, stress incontinence, and recto-urethral fistula (rare).

General principles of management of localized prostate cancer

When considering treatment options for the man with localized prostate cancer, the following factors should be considered in the discussion:

- Patient's life expectancy and overall health status
- Tumor characteristics, including Gleason score, tumor stage, PSA levels, PSA velocity and PSA doubling times
- Risk stratification



Treatment protocol of prostate cancer

- If life expectancy <10 years, Watchful waiting(symptomatic ttt)</p>
- If life expectancy > 10 years,>>asses the risk

Treatment

Low risk

Active surveillance: PSA every 6 months and biopsy - 1 year

intermediate risk

Without metastasis: Radical prostatectomy With metastasis: Short course ADT(androgen deprivation therapy) then Radiotherapy

High risk:

Localized: Radical prostatectomy + EBT (extrnal beam radiotherapy)

Locally advanced: Neoadjuvant hormonal + EBT

Metastasis: Hormonal therapy only (GNRH agonist injection every 1-3 months or surgical castration (bilateral orchiectomy))

watchful waiting and active surveillance

Watchful waiting is based on the premise that some patients will not benefit from definitive treatment of the primary prostate cancer .

Active surveillance is based on the concept that some, but not all patients may derive benefit from treatment of their primary prostate cancer.

Advantages of active surveillance include avoidance of possible side effects and costs of definitive therapy that may be unnecessary, and maintaining quality of life.

Disadvantages include possibly missing an opportunity for cure, the risk of progression and/or metastasis, increased anxiety, increased physician visits and tests, and causing subsequent treatment to be more aggressive.

watchful waiting and active surveillance

Surveillance protocol: (low risk)

Patients must have clinically localized disease and be candidates for definitive treatment and choose observation.

- DRE and PSA as often as every 6 months but at least every 12 months
- Repeat prostate needle biopsy within 6 months of diagnosis if initial

biopsy was <10 cores

• Needle biopsy may be performed within 18 months if >10 cores obtained initially, then done periodically

radical prostatectomy

Radical (total) prostatectomy (RP) is excision of the entire prostate, including the prostatic urethra, with the seminal vesicles. It may be performed by open retropubic, perineal, laparoscopic, or robotically assisted laparoscopic approaches.

RP is indicated for the treatment of men in **good health** with localized prostate cancer whose **life expectancy exceeds 10 years**, with curative intent.

radical prostatectomy

Complication of the surgery :

- intraoperative obturator nerve , ureteral or rectal injury [early]
- 2. it results in high incidence of impotence but a low incidence of severe stress incontinence <2% [late]
- 3. bladder neck stenosis (bladder neck contracture) [late]
- 4. bleeding or infection may happen with any surgery



Indications

clinically localized prostate cancer life expectancy >5 years.

Contraindications

- Severe lower urinary tract symptoms (risk of radiation cystitis)
- Inflammatory bowel disease (risk of radiation proctitis)
- Previous pelvic irradiation

brachytherapy (BT)

This is ultrasound-guided trans perineal implantation of radioactive seeds.

- Indications for BT as monotherapy BT is best for low-risk disease: localized T1–2a, Gleason <6, PSA <10 ng/ml prostate cancer, with a life expectancy >5 years.
- Indications for BT with EBRT
 - In the non-protocol setting, patients with intermediate-risk prostate cancer are sometimes treated in combination: T2b–T2c, Gleason 7, PSA 10–20 ng/ml.

brachytherapy (BT)

- Contraindications to BT
 - previous TURP (risk of incontinence)
 - large-volume prostate (>60 mg), which causes difficulty with seed placement
 - moderate to severe lower urinary tract symptoms (risk of retention).
 - High-risk prostate cancer does not do well with BT monotherapy and should not be performed

Cryotherapy and HIFU

- These two **minimally invasive treatments** for localized prostate cancer
- they are viable alternatives to radical surgery or radiotherapy and that they are options for salvage treatment of organ-confined recurrent disease following radical radiotherapy



Cryotherapy

- Cryotherapy, or cryoablation, for prostate cancer is the controlled freezing of the prostate gland. The freezing destroys cancer cells. Cryotherapy is done under anesthesia. This treatment is for men who are not good candidates for surgery or radiotherapy because of other health issues. For this procedure, the prostate is imaged and measured. Special needles called "cryoprobes" are placed in the prostate under the skin. The needles are guided by ultrasound, to direct the freezing process.
- Complications include ED, urinary retention, stress incontinence, and recto-urethral fistula (rare).



High-intensity focused ultrasound (HIFU)

• HIFU has the potential of **selective destruction** of tissues at depth without damaging intervening structures. Tissue is heated to the point of coagulative necrosis by high-energy ultrasound transmitted to the prostate using a transrectal device.



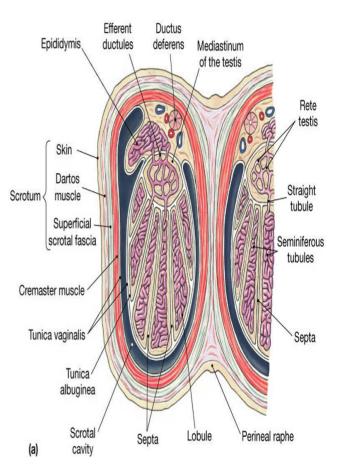
Benign scrotal swellings

Yazeed Aldarabah Rama Shmaileh Sara Aljaafreh

The wall of scrotum has the following layers

1-skin

- 2-superficial fascia(dartos muscle+colles fascia)
- 3-external spermatic fascia derived from the external oblique
- 4-cremasteric muscle derived from the internal oblique
- 5- internal spermatic fascia derived from the fascia transversalis
- 6-tunica vaginalis(remnant of Peritoneum)



- The tunica vaginalis:

- is the lower expanded part of the processus vaginalis
- it becomes closed before birth thus it is closed sac,
- It is invaginated from behind by the testis (covers the anterior, medial, and lateral surfaces of testis).

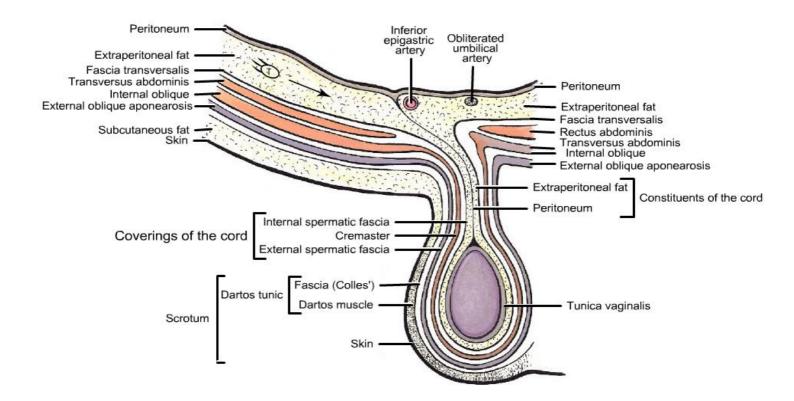
- Lymph Drainage:

 The penis and scrotum, including the tunica vaginalis, drains into the superficial inguinal L.N.

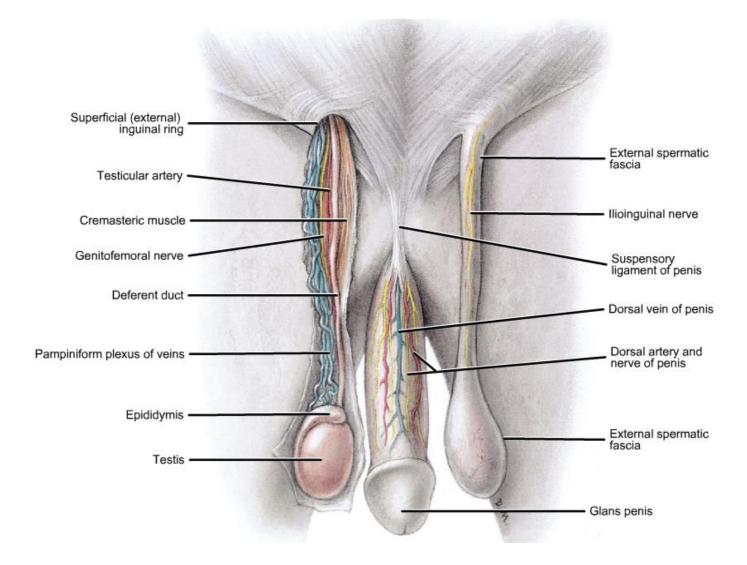
– The cremaster muscle:

- Can be made to contract by stroking the skin on the medial aspect of the thigh i.e. cremasteric reflex.
- The function of the cremaster muscle is to <u>raise the testis</u> and the scrotum upward <u>for warmth and for protection</u>

3 covering seaths of spermatic cord. <u>against injury.</u> * external orimesteric faccia — from external oblique M. * crimosteric M. - internal oblique M. * internal crimosteric fascia - transversus abdomenus M.



& testes are at a temprature about 1-2°C lower them body temprature



Scrotal swellings

Painless

- Hydrocele
- Spermatocele
- Varicocele
- Tumor (non hemorrhagic)
- Hernia

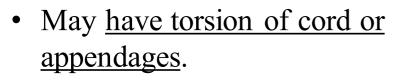
<u>Painful</u>

- Epididymo-orchitis
- Torsion
- Tumor (hemorrhagic)
- Hematocele
- Strangulated indirect Hernia

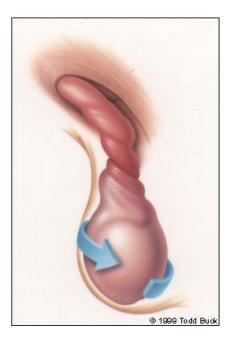
PAINFUL SCROTAL SWELLING

1-Testicular torsion

- It is an Emergency.
- Due to twisting of the spermatic cord with interference to the arterial blood supply+venous occlusion, obstructing the blood supply and venous return and if blood supply isn't restored within 4-6 hours we will lose the testicle.



• Incidence is highest between 10-16 y.o. but may happen at any age.



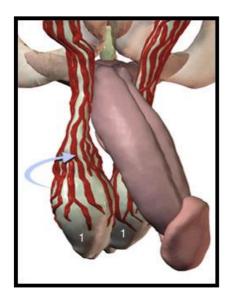
Clinical Feature

- Testicular pain &swelling (Sudden) radiating to the groin, loin, or epigastrium
- sometimes a history of minor trauma to the testis
- previous similar episode
- Most cases spontaneous torsion(50% in sleep).

Other causes(bell clapper deformity, cryptorchidism)



• Anterior surface of each testis run towards the midline.

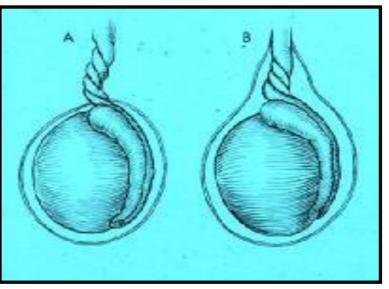




Types:

• Extravaginal: exclusive to perinatal (torsion, the testis, spermatic cord and tunica vaginalis. It is usually <u>ASYMPTOMATIC(</u> we discover it early before appearance of symptoms)...and therefore could be managed by observation.

 Intravaginal: 90%
 of adolescent age group.



A) extravaginal; (B) intravaginal

- On Examination:
- Swollen, painful, testis drawn up to the groinhorezontal lie .
- Absent of cremastic reflex on the affected site
- Elevation of scrotum doesn't provide relife of pain (-ve prehn sign)



• If you are in doubt in case of acute painful scrotum so the scrotum must be explored.

- If untreated infarction of testis will result(within 5-6h necrosis).
- <u>Untwisting should be</u> <u>carried on within 6 hrs. of</u> <u>symptoms</u>.



management

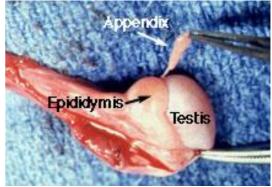
- Rx: EMERGANCY
- \checkmark Explore the testis.
- \checkmark Untwist the testis.
- ✓ If viable we fix it by anchoring it to scrotal septum and if the other testis is abnormal fix it(orchiopexy).
- ✓ If infracted we remove it(orchiectomy).



After detorsion

2-Torsion of testicular appendage:

- Most common structure to twist is the appendix of the testis (pedunculated hydatid of morgagni)
- Usually a more gradual onset, pain moderately severe
- Clinicaly similler to testicular torsion but vertical lie and cremastric reflex preserved)
- Blue dot sign.
- Age:12 24 years age .



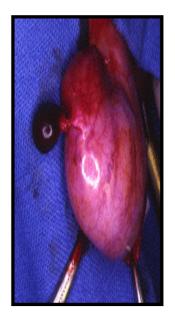


Management

• If the diagnosis is in question or refractory pain, we do a surgical exploration.

Rx;

- conservatively –analgesia (most will subside over 5-7 day)
- immediate operation with ligation and amputation of the twisted appendage.
- when the appendix torsion is late in presentation, it could resemble testicular torsion



3-Testicular trauma

- Usually in sports injuries or violance.
- may result in bleeding into the layers of tunica vaginalis resulting in haematocele.
- S&S: severe pain, scrotal swelling, bruising, tender, enlarged testi,no translumination.





Management

- Investigation:
 - scrotal ultrasound (beware of an underlying malignancy).
- Treatment: CONSERVATIVE
 - Bed rest, ice pack, surgical repair
 - Scrotal elevation
 - Surgical exploration may needed if:
 - 1- expanding scrotal hematoma
 - 2- To evcuate the haematocele and to repair the split in tunica albugenea.
 - 3- very sever pain

4-Epididymitis

• **DEFINITION:**

- Inflammation, Pain, Swelling of epididymis
- Acute: Symptoms usually lasting < 6 weeks
- Chronic: Symptoms usually lasting > 6 weeks
 - May be acute sub-acute chronic
- EPIDEMIOLOGY:
 - Most common cause of acute scrotal pain
 - Age: 16-30 y/o & 51-70 y/o
 - Incidence parallels incidence of Chlamydia & GC

Epididymitis

• ETIOLOGY:

Retrograde infection from the urinary tract.

-Sexually active - Chlamydia, Gonorrhea, E.coli

-Older men and children- E.coli

-Non-infectious - post surgery, drugs

- SIGNS/SYMPTOMS:
 - -Scrotal pain- slow onset
 - -+- Dysuria, frequency, Discharge, Fever
 - -Tenderness and swelling epididymis

clinical features :

- pain, edematous, redness of the scrotum, often associated with pyrexia.
- +/- symptoms of UTI
- Enlarged tender testis and epididymis.
- In children differentiation from torsion is often impossible and scrotum should be explored.

Prehn sign is +ve (prehn sign is when the pain is relieved by elevating the testicles)

Cremastric reflex +ve

+ UTI ass. with high fever chils & right - 3 imp. DDx postatilis (acut)

- A UTI associated with high fever, chills & rigors you need to look for 3 organs:
- Pyelonephritis (kidneys)
- Prostatitis
- Epididymitis

Management

- Investigation:
 - CBC, Urinalysis, Urine Culture, Urethral Swab.
- Treatment:
 - Acute: Bed rest, Analgesia,
 - < 35 y/o
 - Ceftriaxone
 - Doxycyxline
 - > 50 y/o
 - Treat responsible organism
 - ciprofluxacin /Quinilones
 - Chronic: TB-antituberculous drugs.
 - Orchidectomy if fails.

J.

Testicular torsion	Epididymorchitis	
Mechanical twist of the testicle	Inflammatory process	
Acute onset severe pain	Gradual onset mild pain that increase with time	
Afebrile	Associated with high fever, chills and rigors	
w/o urinary symptoms	Associated with dysuria and frequency	
Negative Prehn sign	Positive Prehn sign	
Negative Cremasteric reflex	Positive Cremasteric reflex	
Non-tender prostate	Tender prostate	
Doppler: Decrease testicular flow	Doppler : increased testicular flow	
Testicular position: elevated and more horizontal	Testicular position : normal (vertical)	
Associated with nausea and vomiting due to severe pain	Not associated with nausea and vomiting	

	Testicular torsion	Epididymorchitis
Age	(10-30) years	(16-30) & (51-70) years
Pain	Sudden onset not affected by position	Gradual onset worse when standing
Onset	After exercise, sleep or minor trauma	Rarely after sleep
Time to presentation	< 6 hours	> 24 hours
Past episodes	Frequently >2weeks past	Only if previous infection
Severity	Peaks in hours	Peaks in days
Vomiting	Common from pain	Unusual
Fever	Up to 20%	Up to 95%
Swelling	After 12 hours	Common
Dysuria	Rare	Common
Urinanalysis	30% have wbc/bacteria, voiding complication rare	50% may be normal , voiding complication common
Physical exam	Cremasteric reflexPrehn sign	+ Cremasteric reflex + Prehn sign
Color Doppler	Decreased testicular flow	Increased testicular flow
Management	Antibiotics, if it fails, we do orchidectomy	Surgery (Orchidopexy or Orchidectomy)

5- Orchitis

- **DEFINITION**:
 - Inflammation or infection of the testicles
 - may be related to epididymitis with Extension to testes
- Etiology:
 - bacterial (E. coli, K. pneumoniae, P. aeruginosa, Staph. or Strep)
- viral (MUMPS VIRUS, EBV, coxsackievirus, arbovirus, enterovirus) especially isolated orchitis

ORCHITIS

- SIGNS & SYMPTOMS:
 - similar to epididymitis
 - Pain
 - hematuria, ejaculation of blood
 - entire testes swollen- exquisitely tender
 - Systemic- fever chills, malaise

Investigation:

- Urinalysis: bacteria, WBC's, crystals -commonly in epididymitis
- Obtain urine culture;(why ? If pt have +ve culture with epidedmytise R/O congenital anomaly by US or MCUG (in pediatrics)
- CBC may be helpful
- Radiographic studies
 - -Ultrasonography, Nuclear Scan(tc33)
 - -Doppler US.

Diagnostic test Color Doppler ultrasound

- Noninvasive assessment of anatomy and determining the presence or absence of blood flow.
 - sensitivity: 88.9%
 specificity of 98.8%
 - operator dependent.
 - but its sensitivity for diagnosing torsion is only 80%

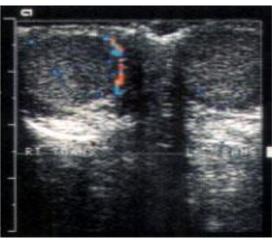


FIGURE 1. Color Doppler ultrasonogram showing acute torsion affecting the left testis in a 14-year-old boy who had acute pain for four hours. Note decreased blood flow in the left testis compared with the right testis

Color Doppler ultrasound

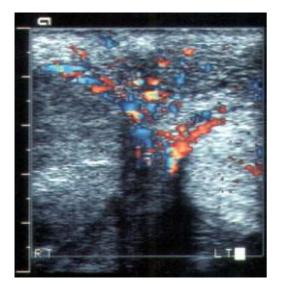


FIGURE 2. Color Doppler ultrasonogram showing late torsion affecting the right testis in a 16year-old boy who had pain for 24 hours. Note increased blood flow around the right testis but absence of flow within the substance of the testis.

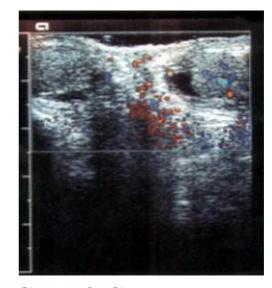


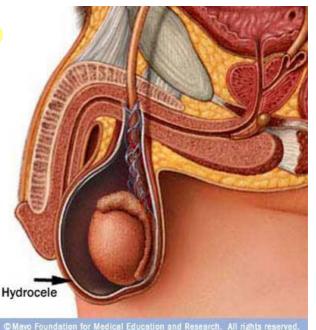
FIGURE 3. Color Doppler ultrasonogram showing inflammation (epididymitis) in a 16-year-old boy who had pain in the left testis for 24 hours. Note increased blood flow in and around the left testis.

PAINLESS SCROTAL SWELLING

1-Hydrocele no pain Swelling only



- Is collection of abnormal quantity of serous fluid in the tunica vaginalis (between tunica albuginia) & tunica vaginalis)
- If it contains pus or blood it is called pyocele or haematocele respectively.
- Hydrocele is more common than the two other varieties.



etiology

1-primary

The cause is unknown.

• It classified as follows;



in pediatrics 1-communicating; -> patent prossessus Vaginalis it connect with the peritoneal cavity, patent open procesus vaginalis -> connection bu abd. covity & processus vaginalis vas ---In pediatric, change size testis on off lydrocele communicating during day (increase hydrocele scrotum during crying and straining) 2- noncommunicating; it obliterated DIOCESSSUS

dose not connect with Communication with peribonal was testis peritoneal cavity scrotum hydrocele

In adult.

1- primag hydrocele - o idio pathic
2- secondary; where the fluid accumulate secondary to pathology inside the testis like epididymo-orchitis, testicular tumor 5-10% and Trauma.

Clinical presentation;



✤ primary hyrocele are most common newborns

Secondary are more common between 20 to 40 years.

Symptoms;

1-painless swelling

2-frequent and painful micturation may occur if hydrocele is secondary to epididymo-orchitis

Hydrocele doesn't affect fertility

Clinical picture

*****Examination;

- Position; the swelling usually unilateral but can be bilateral if communicating we can't feel the cord above the lump.
- Colour and temperature; normal
- Tenderness; primary are not tender but secondary may be tender
- Composition; fluctuant and have fluid thrill if large enough
- Reducibility; can not reduced
- ✤ -ve cought impulse
- * transillumenate -> in any pt with scrotal swelling

Transillumenation





Primary; in children we go through the inguinal canal

Communicating;

most neonatal hydrocele resolve in first 2 year of life if persists repair as herniotomy(inguinal incision).

NEVER do surgery before 2 years of age

EXCEPT in

1- very large amount –

2- if can't differentiate between it and hernia

3- increase intrabdominal pressure

NEVER do needle aspiration EVEN in the non-communicating type(cause it will reaccumulate)

Noncommunicating;

✤usually resolves spontaneously

& excision of of tunica vaginalis - to drain hydrocele inside it

* although in Pediatrics: go through the inguinal Gual bcz we expecting patent possesus vaginalis

In adult; surgical excision; opening the tunica vaginalis longitudinally (scrotal incision), emptying the hydrocele, everting the sac after excising the redundant sac and suturing the sac behind the cord thus obliterating the potential space

Secondary treatment of the underlying condition
Case ;

40 y old man came with painless , transeluminate hydrocele .

What's your next step?

A; scrotal US to R/O testicular tumor

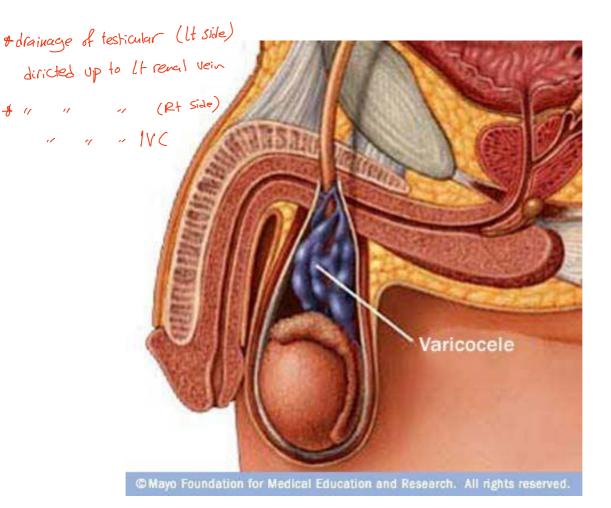
2- Indirect inguinal hernia:

- most common (young , Rt. Side)
- -10% bilateral.
- Hernia in babies are a result of persistent processus vaginalis.
- If strangulated >> painful and may cause testicular atrophy
- Surgery is usually recommended .

Hernia / Hydrocele

		hydrocele	Reducible inguinal hernia	Incarcerated hernia
	💧 🔪 Age	Mostly < 1y	Any age	Any age
	pain	-	-	+++
	Diurnal	None or	Protrudes on	Always
	chan ges	fluctua te	straining	protruded
	si te	scrotum	Groin+scrotum	Groin+scrotum
	💧 🔪 Ex	Can getabove	Can't get above	Can't get above
	Transill - umination	++	- (except in infants)	- /+
	re duci bl e	No	Yes	No
	Rx	Repair if persist >1-2 y	Elective repair	Immediate reduction , urgent repair

3-Varicocele 🟒



Definition

- Is dilatation and tortuosity of the pampiniform plexus, which is the network of veins that drain the testicle.
- Due to defective valve or compression of the vein by a nearby structure.
- Very common; about 15% of men will have some degree of varicocele.
- More common on left side in <u>90%</u>(why?? The longer course of the left spermatic vein and its insertion at a 90° angle into the left renal vein, predisposes to slower drainage and increased hydrostatic pressure.) of cases.
- Bilatral in up to 50% of cases.
- not painful but if long standing may cause a dull aching pain, fertility & subfertility issues, testicular atrophy and a mass (bag of worms)

Primary varicocele :

+ve when standing only.

Secondary varicocele : when varicocele is +ve at BOTH standing and supine positions.

Secondary varicocele could be a sign of a retroperitoneal mass like Renal Cell Carcinoma, Wilms tumor and phaeochromocytoma

- Do retroperitoneal US to role out renal ca in 2 cases ;
 - 1- varicocele on the rt side
 - 2-secondary.
 - 3- palpable abdominal mass
 - 4- acute onset```

& examination - & standing & supine positions in Vareacele of veins Clinical feature

- 1. Appear on standing and disappear on lying down.
- 2. Heavy or dragging sensation in scrotum.
- 3. The veins often described as **'bag of worms'** but feeling like a 'plate of lukewarm spaghetti'.
- 4. The affected testes may be small.
- 5. 90% of Bilateral varicocele may cause infertility.

& long standing - dull aching pain & testicular atrophy

Grading

Grade 0 - Subclinical varicocele, Dx by US or venography

Seve

ncreative

Grade 1 – palpable with Valsalva maneuver on standing

Cliwically defection

Grade 2 - Easily detected without Valsalva maneuver on standing

Grade 3 - Detected visually at a distance





- Diagnosis:
 - Clinical and US.
- Treatment:
 - \checkmark No treatment required in asymptomatic.
 - ✓ Indication of treatment: 25%, of adults have vareacele not all of them needs surgerey

1- impaired sperm quality or quantity (fertility & subfertility issues) Should be related directly to vare cocele 2- pain or dull ache affect quality of life

3- affected testis fail in grow in <u>adolescent causing testicular</u> or pediatric age group to avoid _____

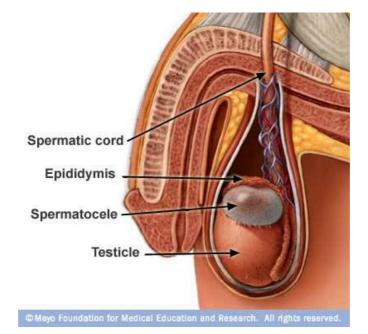
4- discripancy in size more than 20%

4- cosmtic indication

 intervention required either by embolization and obliteration under radiological control or if surgery best tt is varecacele excision - lightim of indicated varecocelectomy is via inguinal approach.

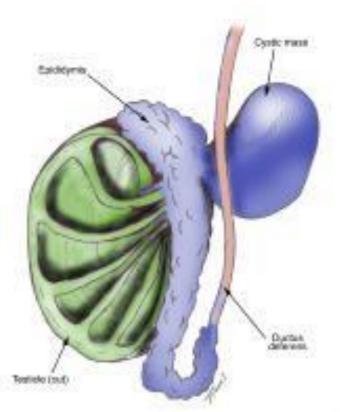
> flow of blood in veins d Prevent varecocele

4- Epididymal cyst



Epididymal cyst (spermatocele)

- Cysts arise from diverticula of the vasa efferentia, they are fluid /sperm filled cysts connected with epididymis.
- THEORIES:
- 1- distal obstruction
- 2- aneurysmal dilation of epididymis
- 2-agglutinated germ cell
- May be small ,large ,multiple, uni or bilateral.
- Usually occur over 40 years.
- S&S: Scrotal swelling, slowly enlarges, painless.
- Lie above and slightly behind the testes.
- You can get above it.
- transluminated



Epididymal cyst

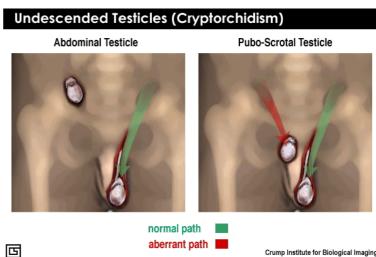
- Usually smooth and lobulated, fluctuant, transilluminates if contains clear fliud.
- US TO ROLE OUT MALIGNANCY
- Rx : none unless **large or painfull** ; surgical excision, and that will compromise the fertility of the testis. In consent form we have to inform the patient about the sideeffect which is infertility.
- AVOID NEEDLE ASPIRATION AS IT CAN LEAD TO INFECTION AND SPILLING OF IRRITATING SPERM WITHIN SCROTUM





CRYPTORCHIDISM

- **DEFINITION:** •
 - Undescended or "Hidden testis"
- EPIDEMIOLOGY:
 - Incidence-
 - 0.7-1% at age 1.
- FTIOLOGY:
 - Uncertain
- COMPLICATIONS:
 - Can lead to infertility and has a higher incidence of malignancy.
- Tx-Orchiopexy



Crump Institute for Biological Imaging

Anatomy & Physiology of erection and ejaculation

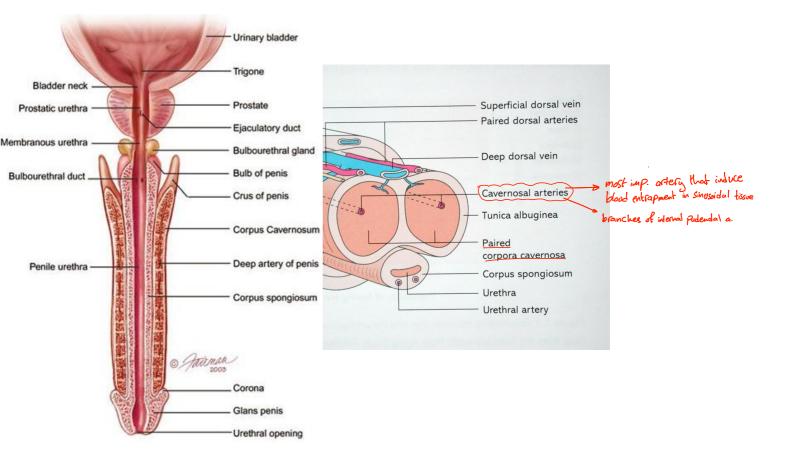
A definition of erectile distinction: inability to achieve

or maintane erection sufficient for sexual relation

```
ninimal role
 ain Ureth(a)
The penile shaft is composed of 3 erectile
columns, the 2 corpora cavernosa and the
                             most imp. part
corpus spongiosum,
                                       that had a contain blood at time of crection
The single corpus spongiosum lies in the ventral
groove between the 2 corpora cavernosa. The
urethra passes through the corpus spongiosum
```

The erectile tissue within the corpora contains arteries, nerves, muscle fibers, and venous sinuses lined with flat endothelial cells, and it fills the space of the corpora cavernosa.

Anatomy of the penis



Arterial supply

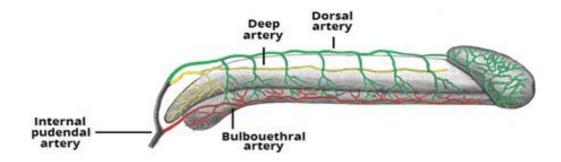
The blood supply to deep structures of the penis is derived from a continuation of <u>the internal</u> <u>pudendal artery</u>, after it gives off the perineal branch. Three branches of the internal pudendal artery flow to the penis, as follows: The artery of the bulb (bulbourethral artery) passes through the deep penile (Buck) fascia to enter and supply the bulb of the penis and penile (spongy) urethra

The dorsal artery travels along the dorsum of the penis between the dorsal nerve and deep dorsal vein and gives off circumflex branches that accompany the circumflex veins; the terminal branches are in the glans penis

The deep penile (cavernosal) artery is usually a single artery that arises on each side and enters the corpus cavernosum at the crus and runs the length of the penile shaft, giving off the helicine arteries, which are an integral component of the erectile process Blood supply to the skin of the penis is from the left and right superficial external pudendal arteries, which arise from the femoral artery

Arterial supply

 main artery is from the internal iliac : internal pudendal artery



 Accessory arteries : Obturator artery vesical arteries femoral arteries

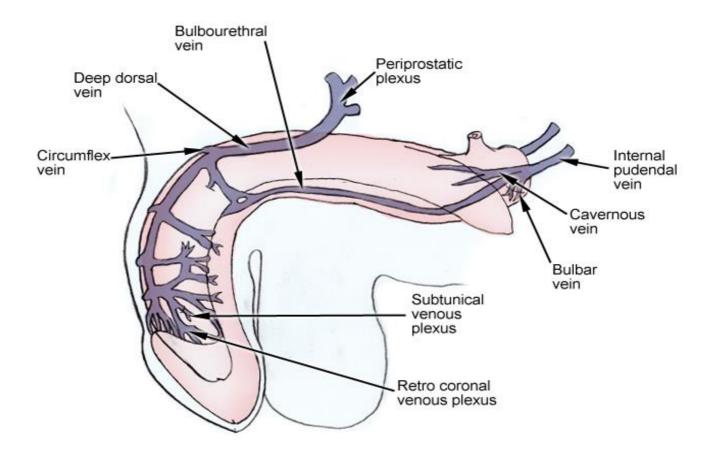
venous drainages

- The penis is drained by 3 venous systems, the superficial, intermediate, and deep
- Superficial veins are contained in the dartos fascia on the dorsolateral surface of the penis and coalesce at the base to form a single superficial dorsal vein, which usually drains into the great saphenous veins via the superficial external pudendal veins.

Dorsal penile veins ????

These are the veins are going to be compressed to prohibit blood from draining

Venous drainage



Lymphatic drainage

Lymphatic drainage from the glans penis drains into large trunks in the area of the frenulum. These lymphatic vessels then circle to the dorsum of the corona and unite, coursing proximally beneath the deep penile (Buck) fascia, terminating mostly in the deep inguinal nodes of the femoral triangle. Some lymphatic drainage is to the presymphyseal lymph nodes and to the lateral lymph nodes of the external iliac lymphatics.

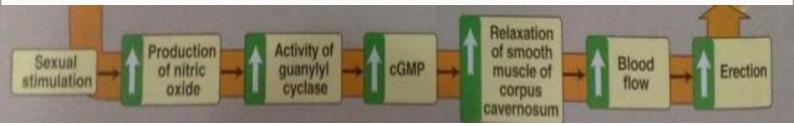
Mechanism of erection sinusoid tissue; blood induce elangertion &

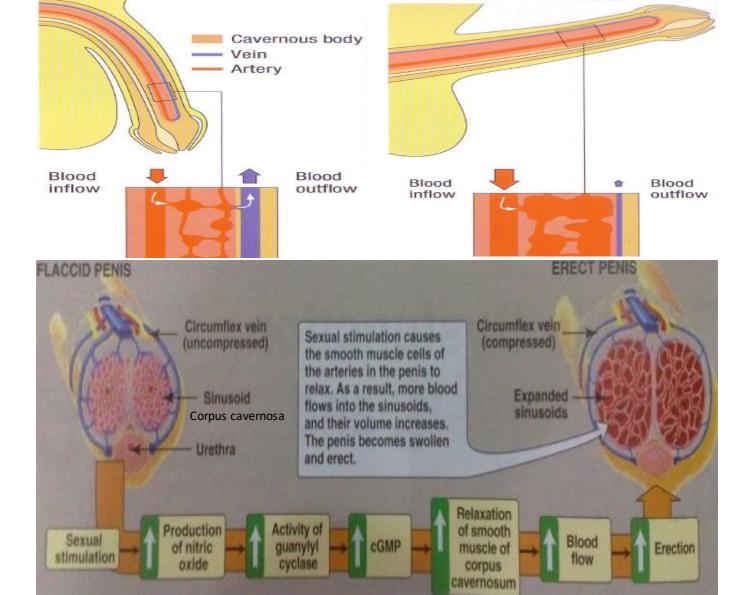
• Neuroendocrine signals from the brain, created by audiovisual or tactile stimuli, activate the autonomic nuclei of the spinal erection centre. Signals are relayed via the cavernosal nerve to the erectile tissue of the copora cavernosa, activating the venoocclusive mechanism

(Mechanism of Penile Erection)

The stimulation result in the relaxation of smooth muscles of corpus cavernosum which leads to increase in the blood flow.

<u>This process is mediated by Nitric oxide(NO) which activates Guanylyl cyclase which forms Cyclic guanosine monophosohatase(cGMP) from guanosine triphosphate(GTP),cGMP produces smooth muscle relaxation and decreases intracellular Ca concentration.
 the sinusoidal the duration of action of cGMP is controlled by Phosphodiesterase (PDE),specially PDE-5 which is the isozyme responsible for the termination of cGMP in the corpus cavernosum.
</u>





★ after arterial dialation & relaxation of Sinussidal tissue ⇒ obstruction of venous outflow from pinis; lead to one way blood flow into pinis so this will induce exection

the name of vein usually closed during erection called emissary vein extreenly imp., any Problem leading to disense Called venous leak or ereclile dyssunction

Innervation

1-stimulation (somatic)

- stimulation of genitals
- sensed by pudendal nerve
- conducted to S2-S4 via pudendal nerve

2-erection is caused by (parasympathetic)

parasympathetic arise in the (lateral horns grey matter of S2-S4)

-and then the pelvic splanchnic go to pre-aortic and prostatic plexuses go to **penis**

- NO causes vasodilation of deep penile arteries
- blood fill erectile tissue
- compression of veins

Innervation

3-emission (sympathetic)

is the beginning part of ejaculation and it is initiated by

sympathetic (lateral horns of T11-L2)

- lumber and sacral splanchnic to pre-aortic plexuses to spermatic tubes and then synapse and

 NE causes : peristaltic contractions of the ductus deferens , seminal vesicle , prostatic gland contracts and then ???>?
 you get contraction of the internal urethral sphincter - semen don't mix with urine

 semen don't go retrogradely into the urinary bladder

4-ejaculation (sympathetic + somatic)

- constriction of internal urethral sphincter (sympathetic)
- impulse arising from ventral horn S2-S4
- pudendal nerve conducts the motor impulse causing
- rhythmic contractions of bulbospongiosus and ischiocavernosus muscles (somatic

Innervation

 Central: medial preoptic area (MPOA) and paraventricular nucleus (PVN) in the hypothalamus are important centres for sexual function and penile erection.

Phases of erectile process

phase	term	description		
<u>0</u>	Flaccid phase N- inflow N- out flow	Cavernosal <u>smooth musc</u> le <u>contracted</u> ; <u>sinusoids empty</u> ; <u>minima</u> l a <u>rterial flo</u> w		
1	Latent (filling) phase inflow > outflow	Increased pudendal artery flow; penile elongation excitement - p c GMP release - pinduce Smooth M. relevation - p lead to Sinussoidal Celasation & Vasodial atotion		
2	Tu <u>mescent phas</u> e	Rising intracavernosal pressure; erection forming		
3	Full erection phase	Increased cavernosal pressure causes penis to become fully erect		
4	Rigid erection phase for few seconds	Further increases in pressure + ischiocavernosal muscle <u>contraction</u> - hardening the penis for few seconds and then disappear		
5	Detumescence phase	Following ejaculation, sympathetic discharge resumes; there is smooth muscle contraction and vasoconstriction; reduced arterial flow; blood is expelled from sinusoidal spaces		
+reduce the pressure => emissary vein will open to drain blood back to circulation				

101 OT VOIDAS MOSULE (VENOUS ICER)

Erectile dysfunction evaluation & treatment

Fadi Abu-Tair

* 2 major causes of crectile Lystunction arteriogenic erectile dys-arterial inflow to pinus (problem in) - athero sclerosis arterics of body reduced by Or failer of Venous Closure (venous leak) Definition

Erectile dysfunction (ED) (also called impotence) describes the 'consistent or recurrent inability to attain and/or maintain a penile erection sufficient for sexual intercourse

> most of Gradiac, diapetic, hypertensive or comorpid pts usually they suffer from erectile dystanct Epidemiology

In men aged 40–70y

- mild ED is found in 17%
- moderate ED in 25%
- complete ED in 10%
- Incidence increases with age with complete ED affecting
- 15% of men in their 70's
- 30-40% in their 80's



ED is generally divided into psychogenic and organic causes It is often multifactorial.

	sever stressful condition		
	Psychogenic may affect	Organic	
Proportion	10%	90%	
Onset	Sudden	Cradual ~> lumen of arteries	
Frequency	Sporadic	All circumstances	
Variation	With partner and circumstance	No	
Age	Younger	Older	
Organic Risk Factors (HTN, DM, Dyslipidemia)	No organic risk factors (wally)	Risk factors present	
Nocturnal/AM erection	Present	Absent	

organic causes : Gradual onset (unless associated with an obvious cause such as surgery, where onset is acute) Loss of spontaneous erections Intact libido and ejaculatory function + Pt with erechile dysbuction — we have to ask spirihically about recturnal or early morning erection — Usually preserved in Psychogenic while lost in organic

Causes of IMPOTENCE			
Inflammatory	Prostatitis		
latrogenic	pelvic surgery/pelvic radiation		
Mechanical	Peyronie's disease		
Psychological	Depression; anxiety; relationship difficulties; lack of attraction; stress		
Occlusive	Arteriogenic: hypertension; smoking; hyperlipidaemia; diabetes mellitus; peripheral vascular		
vascular	disease		
factors	Venogenic: impairment of veno-occlusive mechanism (due to anatomical or degenerative		
Trauma	changes) Pelvic fracture; spinal cord injury; penile trauma		
Extra factors	Other: increasing age; chronic renal failure; cirrhosis		
Neurogenic	CNS : multiple sclerosis (MS); Parkinson's disease; multi-system atrophy; tumour		
Neurogenic	cho. multiple scierosis (Mis), Parkinson suisease, multi-system atrophy, tumour		
	Spinal cord: spina bifida; MS; syringomyelia; tumour		
	PNS : pelvic surgery or radiotherapy; peripheral neuropathy (diabetes, alcohol-related)		
Chemical	Antihypertensives (beta-blockers, thiazides, ACE inhibitors)		
	Anti-arrhythmics (amiodarone)		
	Antidepressants (tricyclics, MAOIs, SSRIs)		
	Anxiolytics (benzodiazepine)		
	Anti-androgens (finasteride, cyproterone acetate)		
	LHRH analogues		
	Anticonvulsants (phenytoin, carbamazepine)		
	Anti-Parkinson drugs (levodopa)		
	Statins (atorvastatin – lipitor [®])		
	Alcohol		
Endocrine	Hypogonadism; hyperprolactinaemia; hypo and hyperthyroidism; diabetes mellitus		

the we prefer to see 2 couples togother.

History -o detailed

Age

Sexual: onset of ED (sudden or gradual); duration of problem; presence of erections (nocturnal, early morning, spontaneous); ability to maintain erections (early collapse, not fully rigid); loss of libido; relationship issues (frequency of intercourse and sexual desire).

- **Sexual function symptom questionnaires:** International Index of Erectile Function (IIEF)
- **Medical and surgical:** enquire about risk factors, including diabetes *mellitus* (ED affects 50% overall and 30% of treated diabetics); cardiovascular disease; hypertension; peripheral vascular disease; endocrine or neurological disorders; pelvic and penile surgery, radiotherapy, or trauma (which damage innervation and blood supply to the pelvis and penis).
- **Psychosocial**: assess for social stresses, anxiety, depression, coping problems, patient expectations, and relationship details.
- **Drugs:** enquire about current medications and ED treatments already tried and their outcome.

Social: smoking, alcohol consumption.



Examination

- Full physical examination (cardiovascular, abdomen, neurological)
- DRE to assess the prostate; assess secondary sexual characteristics
- external genitalia assessment to document foreskin phimosis, penile deformities and lesions
 these lead in (Peyronie's plaques); confirm presence, size, and location of testicles.
 - The bulbocavernosus reflex can be performed to test integrity of spinal segments S2–4 (squeezing the glans causes anal sphincter and bulbocavernosal muscle contraction).

Investigation Anemia? HbAic

- 1. Blood tests: fasting glucose; serum (free) testosterone (taken 8.00–11.00 a.m.); fasting lipid profile are basic work-up tests. SHBG; U&E; LH/FSH; prolactin; PSA; thyroid function test should be selected according to patient's history and risk factor profile.
- 2. Nocturnal penile tumescence and rigidity testing: the Rigiscan device contains two rings that are placed around the base and distal penile shaft to measure tumescence and number, duration, and rigidity of nocturnal erections. Useful for diagnosing psychogenic ED and for illustrating this diagnosis to patients.
- 3. Penile colour Doppler USS: measures arterial peak systolic and end diastolic velocities, preand post-intracavernosal injection of PGE1. to differentiate blue arteriogenic & venogenic exectile dysfunction
- 4. Cavernosography: imaging and measurement of penile blood flow after intracavernosal injection of contrast and induction of artificial erection, used to identify venous leaks.
- 5. Penile arteriography: reserved for trauma-related ED in younger men. Pudendal arteriography is performed before and after drug-induced erection to identify those requiring arterial bypass surgery (although this is less commonly indicated now with the advent of modern penile prostheses).
- **6. MRI:** useful for assessing penile fibrosis and severe cases of Peyronie's disease.

Nocturnal penile tumescence



treatment

Started by behavioral modification

 Correct any reversible causes (i.e. alter lifestyle, stop smoking, change medication, etc.)

Psychosexual therapy

- Aims to understand and address underlying psychological issues and provides information and treatment in the form of sex education, psychosexual counselling
- □ instruction on improving partner communication skills
- cognitive therapy and behavioural therapy (programmed relearning of couple's sexual relationship).
- Pharmacotherapy may be a useful adjuvant.

medical th

\$ drugs ause relaxation of the smooth M. So induce erection:

1-Phosphodiesterase type-5 (PDE5) inhibitors:

first-line therapies

sildenafil (ViagraR), tadalafil (CialisR), vardenafil (LevitraR)

- enhance cavernosal smooth muscle relaxation
- by blocking the breakdown of cGMP by phosphodiesterase. ٠
- Sexual stimulus is still required to initiate events. ٠
- Success is reported in up to 80%. .
- Early use of PDE5 inhibitors following radical prostatectomy can help optimize the return of spontaneous erections (penile rehabilitation).

¿Contraindications:

- patients taking nitrates
- recent myocardial infarction
- > recent stroke bez those drugs Gause sudden drop in BP -> double effect Sudden drop in BP -> may lead to
- hypotension
- unstable angina, non-arteritic anterior ischaemic \succ
- optic nerve neuropathy (NAION).
- Cautions: intermediate and high risk cardiovascular disease requires cardiac review prior to treatment

acute cardiac allest

use with A-blockers, groups with predisposition to priapism.

2-Dopamine receptor agonist: نهان

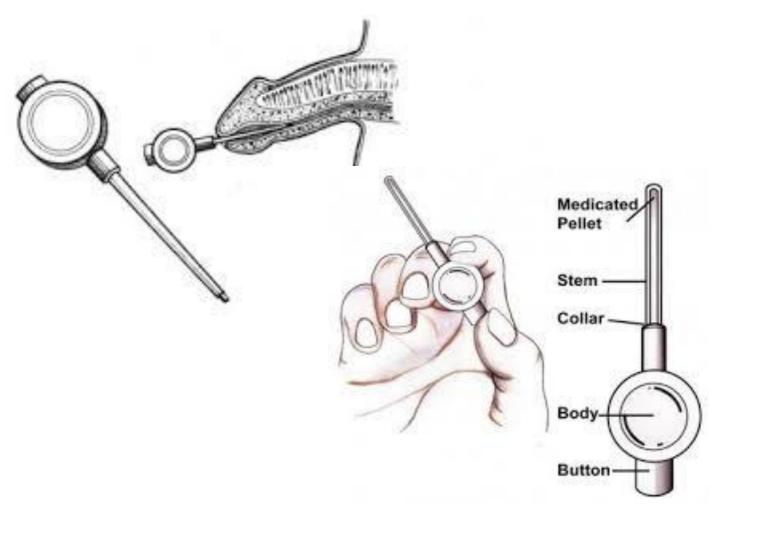
- > apomorphine (UprimaR)
- taken sublingually
- acts centrally on dopaminergic receptors in the paraventricular nucleus of the hypothalamus
- enhance and coordinate the effect of sexual stimuli.
- Side effects: nausea, headache, dizziness
- > Not commonly used

Uncommon

3-Intraurethral therapy:

*injection of gel into viethia; absorption of this gel from viethica (PGE) - Cause relaxation of arterial smooth M.

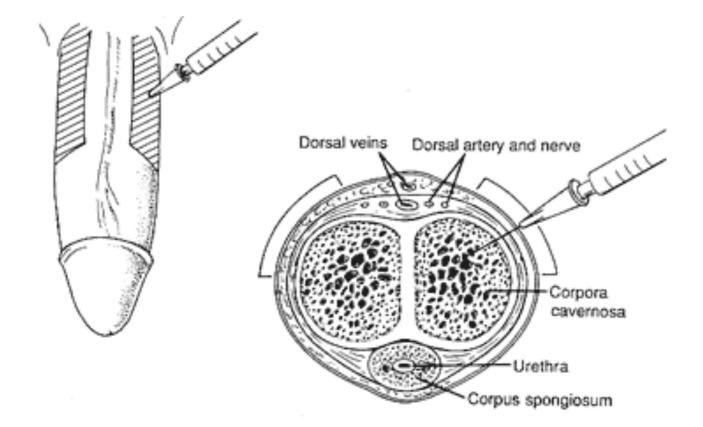
- <u>second-line</u> therapy when oral therapies have been ineffective.
- A synthetic prostaglandin E1 (PGE1) pellet (alprostadil)
- placed into the urethra via a specialized applicator (Medicated Urethral System for Erection (MUSE)TM device).
- Once inserted, the penis is gently rolled to encourage the pellet to dissolve into the urethral mucosa fromwhere it enters the corpora.
- PGE1 acts to increase cAMP within the corporal smooth muscle, resulting in muscle relaxation.
- Side effects: penile and urethral pain, priapism, local reactions.



invassive 4-Intracavernosal injection therapy

Asyringe indected into the penis directed to cavernosal sinusvidal tissue a Cause spontanous erection Alprostadil (CaverjetTM).

- Papaverine (PDE inhibitor).
- Usually given in combination with either phentolamine (Aadrenoceptor antagonist) or PGE1
- Who have failed oral or single-agent injectable therapies.
- Training of technique and first dose is given by a health professional.
- Needle is inserted at right angles into the corpus cavernosum on the lateral aspects of mid-penile shaft.
- Discontinuation rates from penile injection techniques are high.
- Contraindications: sickle cell disease or high-risk candidate for priapism.
- Adverse effects: pain, priapism, haematoma



Vacuum erection device

- > Used when pharmacotherapies have failed. (SE on Pt bcz of medical the)
- It contains three components: a vacuum chamber, pump, and constriction band
- The penis is placed in the chamber and the vacuum created by the pump increases blood flow to the corpora cavernosa to induce an erection.
- The constriction band is placed onto the base of the penis to retain blood in the corpora and maintain rigidity.
- Relative contraindication: anticoagulation therapy.
- Side effects: penile coldness, bruising.

Microvascular arterial bypass and venous ligation surgery

Used in: specialist centres where there is a clear-cut diagnosis of a vascular disorder.

Acts to increase arterial inflow and decrease venous outflow. Rarely used now as it is uncommon for success rates to exceed 50%. inflatable un inflatable into corpora Gavernosa (in center) 2types a last step in treatives pt with erectile dysfunction Penile prosthesis surgers

مجدديل اختصادك للر ttt المت medical ttt جنبو ها فرطانة Pt وyoung -

- Semi-rigid, malleable, and inflatable penile prostheses
 other therapies have failed or are unsuitable
- Also indicated for Peyronie's disease, trauma, and penile fibrosis (i.e. secondary to priapism).
- The device is surgically implanted into the corpora to provide penile rigidity and generally has high satisfaction rates, up to 90%
- Side effects: infection, erosion, mechanical failure, penile shortening, glans may not fully engorge.

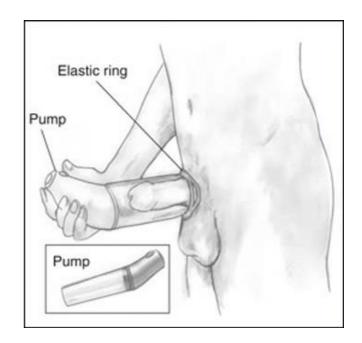
Testosterone replacement therapy

- Indicated for hypogonadism
- available in oral, buccal, intramuscular, pellet, transdermal patch, and gel forms.
- Most guidelines recommend PSA, Hb, and LFT checks before and after starting treatment
- It can improve the results of PDE5 inhibitors in hypogonadal men.

Penile prosthesis

Vacuum erection device





Peyronie's disease, fibrasis appeare in one side of the corputa cavernosa in Junica albuquenia of penis

An acquired benign penile condition characterized by deformity of the penile shaft secondary to the formation of a fibrous inelastic scar on the tunica albuginea.

Epidemiology

Prevalence is 3–9%, predominantly affecting men aged 40–60y.

Pathophysiology

- excessive connective tissue (fibrosis) and increased cellularity with random orientation of collagen fibres. Dorsal penile plaques are most common (66%).
- The corpus cavernosus underlying the lesion cannot lengthen fully on erection, resulting in penile curvature. - to one side

The disorder has two phases:

- Active phase (1–6 months)
- Quiescent (stable) phase (9–12 months

Presented as

- penile pain, a palpable lump (plaque), I may need excision of fibrosis (surgery)
- penile curvature, ED (in 40%),

\$ indications of surgery: - Curvature > 90° - sever pain during crection - erectile dysfunction



Male Infertility

Mohammad al-madhon

Raed ALI

Object....

- Male reproductive physiology
- Etiology of infertility
- Evaluation of male infertility
- Investigation
- Treatment options for male infertility



Male reproductive physiology

How Does the Male Reproductive System Function?

The all male reproductive system is dependent on hormones, which are chemicals that regulate the activity of many different types of cells or organs. The primary hormones involved in the male reproductive system are <u>follicle-stimulating hormone</u>, <u>luteinizing</u> hormone, and testosterone



The hypothalamus secretes luteinizing hormonereleasing hormone

(LHRH), also known as gonadotrophin-releasing hormone (GnRH). This

causes pulsatile release of anterior pituitary gonadotrophins, called follicle-

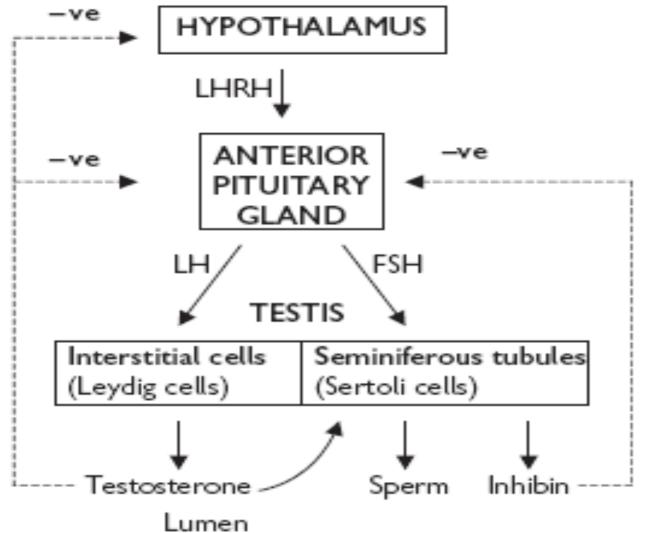
stimulating hormone (FSH) and luteinizing hormone (LH), which act

on the testis.

FSH stimulates the seminiferous tubules to secrete inhibin and produce

sperm; LH acts on Leydig cells to produce testosterone





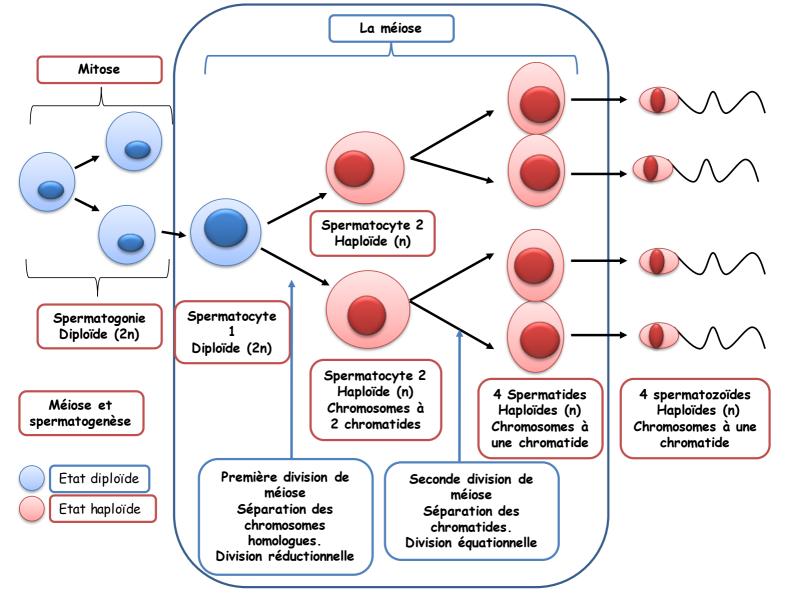


Spermatogenesis

Seminiferous tubules are lined with Sertoli cells, which surround developing germ cells (spermatogonia) providing nutrients and stimulating factors, as well as secreting androgen-binding factor and inhibin Primordial germ(spermatogonia) cells divide to form primary spermatocytes. These undergo a first meiotic division to create secondary spermatocytes (46 chromosomes), followed by a second meiotic division to form spermatids (23 chromosomes). Finally, these differentiate into spermatozoa.

Spermatogenesis takes 74 days. The nonmotile spermatozoa leave the seminiferous tubules and pass to the epididymis, where they undergo maturation (gain motility and the ability to fertilized). Ductal transit time takes another 2 weeks, so the total time from beginning of spermatogenesis to ejaculation is 3 months.

Motile sperm are stored in the globus minor of the epididymis until ejaculation. Spermatozoa that are not released are reabsorbed by phagocytosis.





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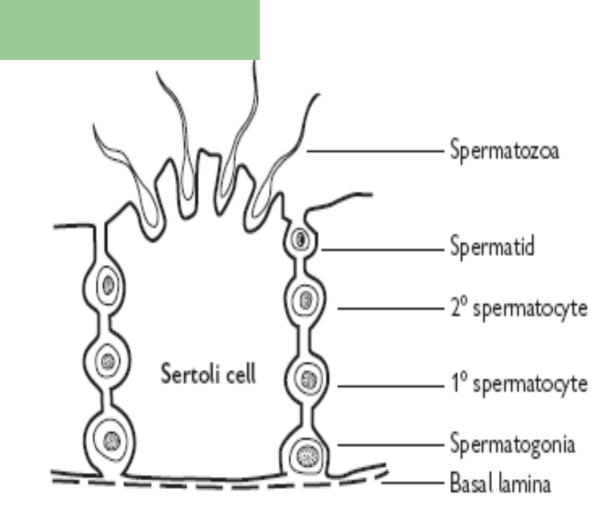
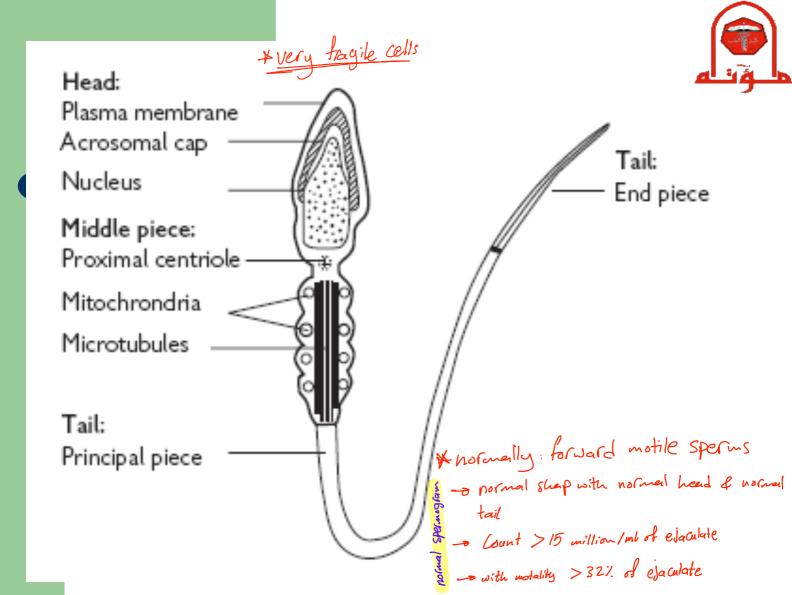


Figure 11.2 Spermatogenesis in the seminiferous tubules of the testis.



Mature sperm

Mature sperm have a head, middle piece, and tail. The head is composed of a nucleus covered by an acrosome cap, containing vesicles filled with lytic enzymes that break down the outer layer of the female ovum. The middle piece contains mitochondria and contractile filaments, which extend into the tail to aid motility. yes y normal spermatogenesis in testicles + Sperm maturation " - + Storage " epididymis + with intact lube - from epididymis to vietura





Etiology and evaluation of male infertility

Definition of infertility

Infertility is failure of conception after at least 12 months of unprotected intercourse

. The chance of a normal couple conceiving is estimated at :

20–25% per month, 75% by 6 months, and 90% at 1 year. Up to 50% of infertility is due to male factors.

Up to 25% of couples may be affected at some point in their reproductive years.



Pathophysiology

Failure of fertilization of the normal ovum is due to defective sperm development, function, or inadequate numbers . There may be abnormalities : of morphology (teratospermia) or motility (asthenospermia), low sperm numbers (oligospermia), combined disorders (oligoasthenospermia), absent sperm (azoospermia).

Abnormal epididymis function may result in defective spermatozoa maturation or transport, or induce cell death.



Etiology

- Idiopathic (25%)
- Varicocele (present in 40%)
- Cryptorchidism (undescended testes)
- Functional sperm disorders: immunological infertility (sperm antibodies); head or tail defects; Kartagener's syndrome
- Erectile or ejaculatory problems
- Testicular injury: orchitis

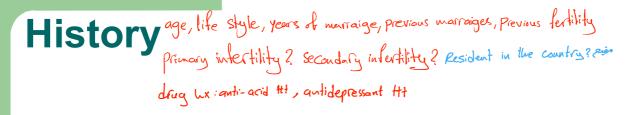


Cont.

Hormone excess: excess prolactin (pituitary tumor)

- Genetic disorders: Kleinfelter's syndrome (47XXY)
- Male genital tract obstruction: congenital absence of vas deferens
- Systemic disease: renal failure; liver cirrhosis; cystic fibrosis
- **Drugs**: chemotherapy; alcohol; marijuana; sulfasalazine; smoking
- Environmental factors: pesticides; heavy metals; hot baths





- sexual and reproductive:: duration of problem; frequency and timing of intercourse; previous successful conceptions; previous birth control; erectile or ejaculatory dysfunction.
- Developmental: age at puberty; history of cryptorchidism; gynecomastia

• Medical and surgical: <u>orchitis</u>; varicocele testicular torsion, <u>trauma</u>, or tumor; sexually transmitted diseases;

for wife:

-z had misscarage before Previously marraide - regular cycle? -> Using drug? - pelvic / abd. Surgery Mabion tubes



Cont.

• Drugs and environmental: previous chemotherapy; exposure to substances that impair spermatogenesis or erectile function; alcohol, *swolking*, *occupation*

• Family: hypogonadism; cryptorchidism

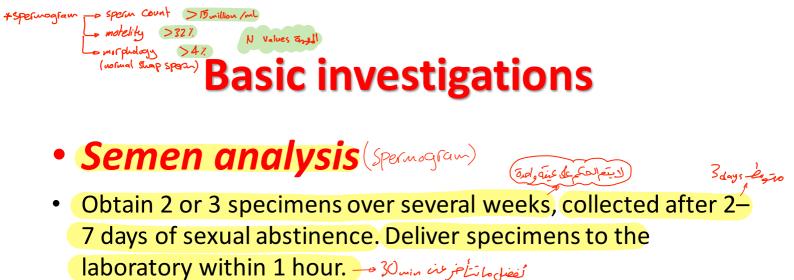




- Perform a full assessment of all systems with attention to general appearance (evidence of secondary sexual development; signs of hypogonadism; gynecomastia).
- scrotum both testicle located in?
 Normal <u>skin</u>, <u>hair</u>, <u>penile size</u>, <u>penile skin</u>
 Urogenital examination should include assessment of the penis
 - (Peyronie's plaque, phimosis, hypospadias); measurement of testicular consistency palpation of epididymis (tenderness, swelling) and spermatic cord (vas deferens present or absent, varicocele); and digital rectal examination of the prostate

Lab investigation of male infertility and Treatment options

Saeed Abdel-kamel



- The mixed agglutination reaction (MAR test) is used to detect antisperm antibodies.
- The presence of leukocytes or round cells in the ejaculate (>1 x 106/mL of semen) suggests infection, and cultures should be requested

Semen analysis parameter	Lower reference limit (95% CI)	
Serum volume	1.5mL (1.4–1.7)	
рН	≥7.2	
Total sperm count	39 x 10º per ejaculate (33–46)	
Sperm concentration	15 x 10 ⁶ per mL (12–16)	
Motility	40% progressive + non-progressive (38-40)	
	32% progressive motility (31–34)	
	Forward progression >grade 2	
Sperm morphology	4% normal forms (3–4)	
Vitality	58% live spermatozoa (55–63)	
Time to liquefy (agglulination of Viscosity)	5-25min 15-30min (Normally)	
White blood cells (WBC)-infection <1 x 10° WBC per mL		
MAR-test (for antisperm antibody)	<50% motile spermatozoa with bound particles	
Zinc	≥2.4µmol per ejaculate	
Semen fructose low: Seminal fluid / semin vesicle obstruction		

Adapted from World Health Organization (WHO) 2010 lower reference limits (5th centile and their 95% CI) for semen characteristics

Table 12.2 Grading of sperm motility

Grade	Type of sperm motility	
0	No motility	
1	Sluggish; no progressive movement	
2	Slow, meandering forward progression	
3	Moving in a straight line with moderate speed	
4	Moving in a straight line at high speed	



- Elevated serum FSH levels (2 x normal) suggest irreversible testicular failure.
- In cases of isolated low testosterone level, it is recommended that morning and free testosterone levels be tested.
- Elevated prolactin is associated with sexual dysfunction and low serum testosterone levels, and usually indicates the presence of a pituitary adenoma.



Table 11.3 Clinical diagnosis on hormone assay

FSH*	LH*	Testosterone	Diagnosis
1	Normal	Normal	Seminiferous tubule damage (defective spermatogenesis)
Normal	Normal	Normal	Normal; or bilateral genital tract obstruction
1	1	Normal/↓	Testicular failure
Ļ	Ļ	Ļ	Hypogonadotrophism

* Follicle stimulation hormone. ** Luteinizing hormone.

Special investigations

- **Chromosomal analysis:** karyotyping to identify Klinefelter's syndrome in patients presenting with azoospermia (5–10% of azoospermic patients have Klinefelter's syndrome), small soft testes, gynaecomastia, d FSH/LH and d testosterone.
- Y chromosome microdeletion assay: to assess AZF—regions a, b, and c.
- 1. AZFa: microdeletion predicts no spermatogenesis.
- 2. AZFc: commonest molecular cause of male infertility (13% of nonobstructive azoospermics and 6% of oligozoospermics). Around 70% will have sperm on testis biopsy.
- **Post-orgasmic urine analysis:** the presence of >10–15 sperm per high powered field confirms the diagnosis of retrograde ejaculation.
- **Fructose:** Although the fructose test is not part of a routine semen analysis, it is useful in cases of azoospermia (absence of sperm in semen). In azoospermia secondary to the absence of vesicles or if there is an obstruction, no fructose is present. In testicular azoospermia, fructose is present.

Testicular biopsy

- Performed for azoospermic patients to help differentiate between obstructive and non-obstructive causes.
- Simultaneous sperm retrieval can be carried out (testicular sperm extraction, TESE) for use in intracytoplasmic sperm injection (ICSI) treatment, either at the time or at a later date (following freezing and storage).
- The degree of spermatogenesis can be histologically scored (The Johnsen score). Only mature spermatozoa (score 8 or above) can be used for fertility treatment.

Testicular biopsy

Table 12.4The Johnsen Score. Histological analysis of testicularbiopsy1

10	Complete spermatogenesis, many spermatozoa	
9	Many spermatozoa, disorganized germinal epithelium	
8	Few spermatozoa (<5–10)	
7	No spermatozoa but many spermatids	
6	No spermatozoa and few spermatids (<5–10)	
5	No spermatozoa or spermatids, but many spermatocytes	
4	Few spermatocytes (<5), no spermatozoa or spermatids	
3	Spermatogonia are the only germ cells	
2	Sertoli cells only	
1	No cells in tubules	

Radiologic Evaluation

- Vasography
- Transrectal Ultrasonography to assi Prostate gland, Seminal vesicle

- Venography
- Scrotal Ultrasonography Fif you find testiculus mass / testiculus atrophy/ Varicocele/ pathology in testicles

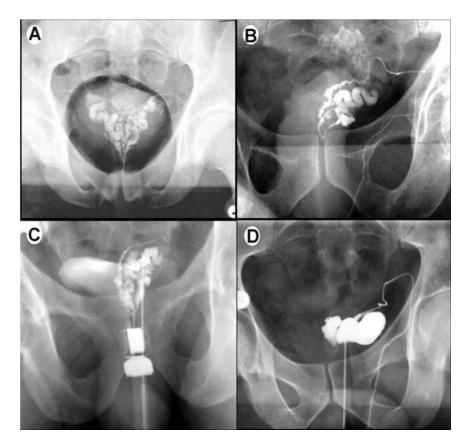
to role out paratesticular pathology & to ass. Size & morphology of lesticles



- The traditional and most commonly employed radiologic imaging study employed for the evaluation of the vasal and ejaculatory duct patency is vasography.
- The vas deferens is punctured at the level of the scrotum and injected with contrast.
- A normal test shows the passage of contrast along the vas deferens, seminal vesicles, ejaculatory duct, and into the bladder, which rules out obstruction.
- Vasography is indicated to determine the site of obstruction in azoospermic patients who have active spermatogenesis documented by testis biopsy

Vasography

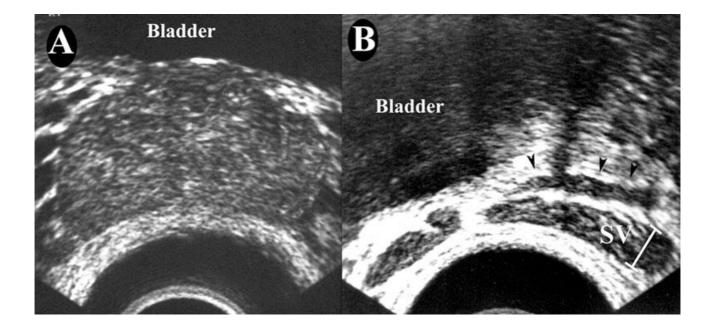
 A normal vasogram is documented when contrast agent is visualized throughout the length of the vas deferens, seminal vesicles, ejaculatory duct, and bladder



Transrectal Ultrasonography

- TRUS allows for the anatomic visualization of the prostate, seminal vesicles, and ampullary portion of the vas deferens
- TRUS is indicated in azoospermic patients suspected of having ejaculatory duct obstruction .
- TRUS is indicated for low ejaculate volumes, to investigate seminal vesicle obstruction (>1.5cm width) or absence
- The normal diameter of the seminal vesicles on transverse imaging behind the bladder is up to 1.5 cm

Transrectal Ultrasonography



Scrotal Ultrasonography

- The main application of scrotal ultrasonography in male infertility has been for the diagnosis of varicoceles
- Color duplex scrotal ultrasonography has been applied as a noninvasive alternative to internal spermatic vein venography in an attempt to objectively diagnose varicoceles
- The initial criteria developed to diagnose a varicocele include the presence of numerous large veins (>3 mm) and reversal of blood flow with Valsalva maneuver

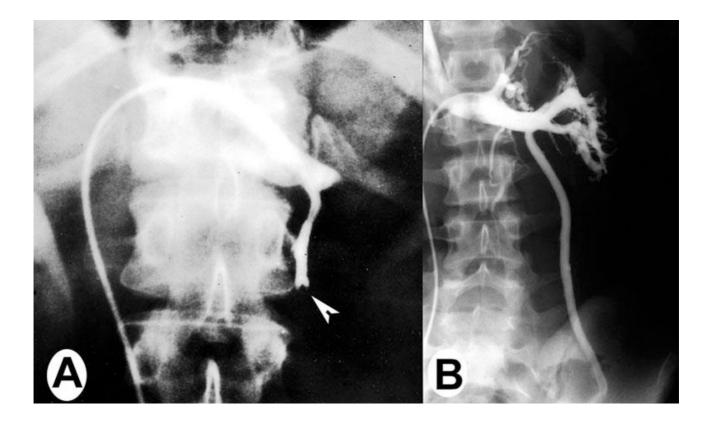
Scrotal Ultrasonography

- Scrotal ultrasonography to detect testicular tumors should be restricted to patients with suggestive histories, physical examinations, or hormonal values.
- It should not be used as a routine examination to screen all infertile men.

Venography

- Internal spermatic vein venography is used to both detect and potentially treat varicoceles
- The femoral vein approach is generally preferred, but the internal jugular approach is superior if embolization of bilateral varicoceles is being observed.

Venography



Treatment options for male factor infertility

General: aim to identify and treat reversible causes of subfertility and improve semen quality. Advice on modification of lifestyle factors (i.e. reduce alcohol consumption, avoid hot baths). *improvement of psycholic conditions of placement*.

Medical treatment:

• Antibiotics:

Treat any positive semen, urine, or urethral cultures with the appropriate antibiotics.

- Hormonal:
- 1. Secondary hypogonadism (pituitary intact): may respond to administration of human chorionic gonadotrophin (HCG) which stimulates an increase in testosterone and testicular size.

-- If the patient remains azoospermic after 6 months of treatment, FSH is added (human recombinant FSH or human menopausal gonadotrophin). Alternatively, pulsatile LHRH can be administered subcutaneously via a minipump (used for treating Kallman's syndrome).

2. - Hyperprolactinaemia: is treated with dopamine agonists. Arrange an MRI to rule out a pituitary tumour.

• Antioestrogens (clomiphene citrate 25mg od):

are used empirically to increase LHRH which stimulates endogenous gonadotrophin secretion. Used for idiopathic oligospermia.

Antioxidants:

Vitamin E supplements have been shown to improve sperm function and IVF success rates; zinc and folic acid may increase sperm concentrations.

Erectile and ejaculatory dysfunction

Erectile dysfunction may be treated conventionally (oral, intraurethral, intracavernosal drugs; vacuum devices or prostheses).

Ejaculatory failure may respond to sympathomimetic drugs (desipramine) or electroejaculation (used in spinal cord injury), where an electrical stimulus is delivered via a rectal probe to the postganglionic sympathetic nerves that innervate the prostate and seminal vesicles.

Antisperm antibodies

Corticosteroids have been used, but assisted conception methods are usually required.

Surgical treatment

- Genital tract obstruction

- **1. Epididymal obstruction:** can be overcome by microsurgical anastomosis between the epididymal tubule and vas (vasoepididymostomy).
- 2. Vas deferens obstruction: is treated by microsurgical reanastomosis of ends of the vas (vasovasotomy) and is used for vasectomy reversal. Highest success rates for finding viable sperm occur in the first 8y postvasectomy (80–90%); overall pregnancy rates are 750%. Patency rates are better than pregnancy rates; success rates drop to 30% if >15y.
- **3. Post-vasectomy:** Ejaculatory duct obstruction: requires transurethral resection of the ejaculatory ducts (TURED).
- 4. Varicocele: can be treated by embolization or open or laparoscopic surgical ligation.

#if failed (all measurement to achive fertility) Assisted reproductive techniques (ART)

Sperm extraction

- sperm are removed directly from the epididymis by *PESA* or *MESA*.
- If these methods fail, *TESE* by conventional biopsy or microsurgical techniques, or aspiration (*TESA*) may be tried.
- Sperm undergo cryopreservation until required.
- Later, they are separated from seminal fluid by dilution and centrifuge methods, with further selection of motile sperm and normal forms using Percoll gradient techniques.

Assisted conception

• Intrauterine insemination (IUI): Following ovarian stimulation, sperm are placed directly into the uterus.

• In vitro fertilization (IVF): Controlled ovarian stimulation produces oocytes that are then retrieved under transvaginal ultrasound guidance. Oocytes and sperm are placed in a Petri dish for fertilization to occur. Embryos are incubated and cultured for 2–3 days and then transferred to the uterine cavity. Pregnancy rates are 20–30% per cycle.

• Gamete intrafallopian transfer (GIFT): Oocytes and sperm are mixed and deposited into the fallopian tubes via laparoscopy. Variations include zygote intrafallopian transfer (ZIFT) and tubal embryo transfer (TET).

• Intracytoplasmic sperm injection (ICSI): A single spermatozoa is injected directly into the oocyte cytoplasm (through the intact zona pellucida). The advantage is that fewer sperm are needed. ICSI is always combined with IVF and the clinical pregnancy rate is 28–40% per cycle.

