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# Renal tumors:

Parenchymal Mass

Metanephric Blastema (embryology) Adenocarcinoma / RCC (M.C. Tumor) Radical Nephrectomy (M.C. Surgery) • Pelvi-calyceal System

Cloacal Membrane

\* Transitional Cell Carcinoma

\* Nephroureterectomy & bladder cuff

No enhancement of contrast.

No hyper vascularity

\* After knowing the origin of the mass you have to determine whether it's Benign /Malignant... there's NO specific investigation that Can tell you.. so you have to consider it *Malignant Until Proven otherwise*.

## Note that Parenchymal Masses :

# 1- Benign

a. Adenoma: very small <3cm, Asymptomatic, Originates from PCT

b. Angiomyolipoma: hamartomas that appear in child bearing female, <u>associated</u> <u>with Tuberous Sclerosis</u>.

- Found incidentally on U/S or CT, but in 10% of cases may Present with Massive retroperitoneal bleeding leading to patient collapse

- Only beign mass can be distinguished On CT scan (Fat Containing Tissue).

-It's Very vascular so **embolization** of feeding Artery Can be the treatment.

If tumor < 4 cm & Asymptomatic.  $\rightarrow$  Observe

But If > 4 cm  $\rightarrow$  excise

c- Oncocytoma: Originates from collecting ducts. (uncommon), M>F, Incidental. Treatment: Partial Nephrectomy (Indications): Bilateral tumor (VHD), Single kidney, Renal Impairment (DM) Lesion (<4 am

\* Masses can be **Solid or Cystic** (MC), screen Initially with US, confirm with Contrasted CT scan.

For cystic masses, we have Bosniak Classification:

- 1) 0% are malignant.
- 2) 0% are malignant (Cyst with minimal Separation)

2F (need follow up  $\rightarrow$  More septation. Increase suspension for Malignancy.

3) 50% are malignant

4) 100% are malignant

\* RF for renal tumors: HTN, obesity, smoking, Family Hx, Asbestos, renal dialysis, horseshoes & PCK, VHL genes (ch 3,7,17), Phenacetin Drug (Analgesia).
\* Presentation: Asymptomatic (70%), Paraneoplastic Syndrome (ectopic horm), Classical Triad: Painless hematuria, Mass, Flank Pain.

\* Investigations: u/s, Contrasted CT (gold standard). Needle Biopsy is contraindicated (fear of seeding & hemorrhage).

### Tumor Spread:

- Direct extension: adrenal gland, renal vein & IVC & Sometimes to R Atrium.
- LN; hilar & Para-aortic LN.
- Hematogenously; Lung, liver, bone, brain.

Malignant: RCC / Adenocarcinoma: M.C. renal Tumor, M>F age 60-80.

-grading  $\rightarrow$  grade 1: well differentian.

 $\rightarrow$  grade 2: moderate.

 $\rightarrow$  grade 3: Poor differentiation.

Staging /T staging
Stage 1 (confined to kidney <7 cm)</li>
Stage 2 (confined to kidney >7 cm).

Stage 3 extension to: T3a: Adrenals

T3b: renal vein or to IVC

T3c: Above diaphragm.

Stage 4 (Invading gerata's fascia & other organs)

# Prostate CA

\* **prostate anatomy:** prostate is located under the neck of the bladder, it Produces 25% of seminal fluid, It's made of glandular epithelial & Stromal tissue. It's growth maintained by hormones mainly (DHT).

- Tumor arises from Peripheral Zone (Asymptomatic In 70% of caes).

• Function: Secretion of PSA: glycoprotein that liquefy the semen &  $\uparrow$  fertilization.

**RF:** Age, Black M, FHx (x2 risk in 1st relative <55y), High fatty diet (Western), Sexual Over-activity, Vasectomy PTEN & P53 Inactivation & c-myc Activation.

• Vit. D, E, Lycopene (Anti-oxidant) & Selenium  $\downarrow$  PC growth

**Presentation** → Asymptomatic, hematuria, LUTS, Perianal discomfort, renal failure, AKI, If Mets: SOB, weight, Anorexia, Bone Pain, LL edema

Diagnosis: NO Screening Test, Most definitive Dx is histopathology. .

**1. PSA:** high suspicion for CA If...

-velocity > 0.75 -Density > 0.15, -Ratio < 20%. -Total >10

**2. DRE:** Look for consistency, size, nodularity, Symmetry.

- Normal Prostate weight is 20 g, 3-4 cm long. It's bi-lobed with median Salcus
- **Don't** Do DRE If there's Prostatitis, or Prostate Abscess

### 3. TRUS:

**Without Biopsy** Azospermia, suspected Prostate Abscess, To Assess the volume, chronic Pelvic Pain.

**↓** With Biopsy, Abnormal DRE / ↑ PSA, Abnormal Previous Biopsy To Confirm viable PC following Treatment.

**Complications**: UTI, Vasovagal Syncope, hematuria / hemato-spermia / Rectal Bleeding, Uro-sepsis or Sepsis in general (fatal).

\* Grading: Gleason score (2-10)

We take 12 samples then Put grade 1-5 for most dominant differentiation of Cells to least one then combine these 2 numbers:

- 2-6 well differentiated.
- 7 moderate.
- 8-10 Poorly differentiated.

\* Staging by DRE (size, T stage), CT &MRI (N&M stage)
T Stage
T0 N0 tumor
Tx uncertain
T1 Impalpable Tumor on DRE
T1a: 5% or less PC tissue found after TURP/Incidentally
T1b: More than 5% PC tissue Round after TURP
T1c: Tumor Round by needle biopsy → made due to 个PSA
T2 & T3 Palpable on DRE

Treatment:

A) radical Prostatectomy: confined to Prostate (T1-T2) No N or M Involvement. when PSA ≤20, moderate differentiation (Life expectancy >10, (T1-T2))

**Complications:** Urinary Incontinence, Impotence, erectile dysfunction, reduce Penis length, Lymphedema, bladder neck stenosis, obturator N Injury.

**B) Waiting & Observe:** 89 yr old Pt, 个 PSA; gleason 10, low life expectancy <10 yrs

C) Active Surveillance (DRE/6 months & Biopsy every year): waiting for

Progression, Pt with LUTS, with BPH, then biopsy comes to be malignant.

D) Hormonal, GnRH agonist with radio: If Mets give continuous dose of GnRH.

\*Open Prostatectomy is contraindicated.

\* PC is Almost Always Adenocarcinoma (glandular tissue)

\* PC extend to Iliac LN

# **Bladder CA:**

Specific gravity 1.005-1.025 + osmolality 300-1,090 MOSM/Kg

\* It's 2nd M.C urological Malignancy, M.C in white males around the age of 80.

\* RF: Smoking (most important), drugs like phenacetin & Cyclophosphamide.

Tumor spread: Direct extension: detrusor muscle, urethral orifices; prostate.

:Lymphatic infiltration Iliac & Para-aortic LN

: Hematogenous spread: Lung, liver, bone, Adrenals.

:Implantation Into wounds / Percutaneous Catheter...

Histological grading (differentiation):

G1 (well differentiated), G2 (Moderately), G3 (Poorly, high grade).

Staging (T stage)

- -Tis / CTS (Not invading Basement mem.)
- -Ta noninvasive Papillary Carcinoma
- -T1 subepithelial Connective tissue
- -T2 Invasion to muscularis propria, Detrusor
- -T3 Invasion to perivesical fat
- -T4 invasion to other organs

### Presentation:

**Hx:** Painless Macroscopic total hematuria, LUTS, recurrent UTI & Pneumaturia (Colovesical Fistula) LL swelling (lymph. /veinous Obstruction).

**PE:** Suprapubic Mass (T4), Bimanual exam in  $\mathcal{P}$  &, DRE (mass above or Involving Prostate), Pallor (due to anemia).

**Investigations**: CT urography, Urine Cytology +ve in CIS specific, TURBT (Dx &Tx). \*\*If Biopsy-Proven Muscle-Invasive Bladder CA  $\rightarrow$  Staging Investigation (CT, MRI, bone scan)

# Types:

- 1) TCC urothelial Carcinoma (90%): Single or multifocal Superficial or muscle-Invasive, M.C. in the Floor (>Carcinogen exposure)
- a. Papillary (GI / G<sub>2</sub>) 2 Ta(Mucosa / Superficial) T1 (Sub mucosa)
- b. Solid/Mixed (G3) 50% are Muscle-Invasive.
- c. CIS/G3 (Poorly Differ.): Confined to epithelium, aggressive 100% + urine cytology.

- SCC: Solid, ulcerative (Muscle-Invasive ), Associated with Smoking, schistsoma
  - Can be due to: Bladder stones, Catheters
  - bilharzial has better prognosis than non-bilharzial.
- 3) Adenocarcinoma (Bladder& extrophy): Solid, ulcerative (Muscle Invasive).
  - G3=> Poor Prognosis: bowel Implantation or bladder extrophy: (10-20 yr)
  - Strong Association with: Cystitis glandularis .
  - 1/3 cases originate in the urachus at dome of Bladder
- 4) Others: Pheochromocytome, Melanoma, Lymphoma & Sarcoma

Treatment: According to the Type & Staging

- <T2 (Tis, Ta, T1) → TURBT + Low grade(G1, G2) → intravesical CTX (adriamycin "doxorubicine" and mitomycin)</li>
   High grade (G3, CIS) → intravesical BCG immuno Tx
- ≥ T2 (muscle-invasive: T2-4, \_\_\_\_\_\_
   SCC, Adenocarcinoma)
   In both ∂ and ♀ → Radical cystectomy+ ileal conduit
   + In ∂ → prostatectomy. So, radical cysto-prostatectomy
   + in ♀ → anterior pelvic exenteration
- TURBT Complications: Bleeding, sepsis, bladder Perforation, Incomplete resection, urethral Stricture
- Mitomycin complications: dermatitis on external genitalia, filling type LUTS.
- Intravesical BCG (Immunotherapy) at least 2 weeks post TURBT:
  - Administered via urethral Cath. & held in bladder 1 h.
  - Complicated by: high fever; requiring Anti-TB for 6 months.
  - can cause granulomatosis Prostatitis & epididymo- orchitis
  - Contraindicated In: Pregnancy, TB Pts, Immunosuppressed, Active UTI, trauma, hematological malignancy, gross hematuria, liver cirrhosis.
  - Follow up: Cystoscopy & Cytology (every 3 months For 2 years -> every 6 months for 2 years then yearly.

### **Renal Diagnostic Tests:**

### A. Laboratory Studies:

1. renal Function test: Serum Cr, Cr Clearance, Protein, BUN, Urine Casts, renal Concentration test. (Specific gravity, Osmolality of urine)

- 2. PSA (Prostate Specific Antigen) for follow up: Normally <4 ng/ml (4-10 indicates BPH)
- 3. UA: Appearance, Odor, Color, PH (Acidic) Specific gravity, osmolality.
  \* Best result taken from midstream Not Initial Flow Nor last drops

### **B. Imaging Studies:**

- 1. kub x-ray: Pt on Liquids only a day before & NPO after midnight.
- IV Pyelogram /urogram: Contrast Medium.
   \*Contraindication: renal Failure, MM, Uncontrolled DM, metformin or asthma or chronic Bronchitis, emphysema medications.
- Renal Angiogram: IV Cath through femoral / Iliac artery
  - \* Clear Liquids, midnight before , Continue oral medications.
- 4.US: Solid & cystic masses, Prostate exam (rectal Probe), Bladder (Full).

### C. Other tests:

- 1. Cystoscopy: complications: Infection, Bleeding, urinary retention (Cath), burning on voiding, Urinary frequency.
- 2. Needle Biopsy: Percutaneous US guided (for DX & Tx)of renal Diseases.

### NURSING AND PATIENT CARE CONSIDERATIONS:

**Prebiopsy:** coagulation studies to identify risk for post-biopsy bleeding and Cr serum, urinalysis, and urine culture.

- describe the procedure to the patient (including holding breath (to prevent movement of the thorax) during during insertion of the biopsy needle) and Ensure that he fasts for several hours.

**Post-biopsy:** Place the patient in a prone position immediately after -> bed rest for 8 to 24 hours to minimize bleeding.

-Take vital signs every 5-15 min in the 1<sup>st</sup> h then decrease frequency if stable to assess for hemorrhage (major complication). Watch for rise or fall in BP, anorexia, vomiting, or development of a dull, aching discomfort in abdomen, flank pain.

### Urinary tract injury:

# 1) kidney Injury / Trauma (M.C)

- a. Blunt/Closed (90%):  $\rightarrow$  > 95% managed Conservatively
  - Mechanism: Direct blow or rapid deceleration -> Hilar Injury (V, A) -Shearing force that destruct BV.
- b. **Penetrating / Open (10%):** if A to A axillary line = injury to renal vessels.
  - Gun-shot (if in high velocity it damages more than one structure due to cavitation effect, 25% treated conservatively.
  - stab wound 50% treated conservatively.

When Pt Presents to ER:

- ✓ Take vitals (Pulse rate & BP) rule out Hypotension.
- Rule out Allergy to contrast => contrast CT , INDICATION: rapid deceleration, gross/ Microscopic hematuria, Penetrating chest & Abdomen wounds, urinalysis of child showing 50 RBCs after blunt trauma.
- ✓ If unstable  $\rightarrow$  OR (Don't wait) -> Intravenous Pyelogram

**Blunt Trauma Classification after CT:** 

grade 1: Subcapsular hematoma, conservative (bed rest, take vitals).

grade 2 laceration <1 cm, conservative.

grade 3: laceration > 1 cm, conservative

grade 4: laceration to cortex, may need Nephrectomy to control Bleeding Appears as extra-vasation of contrast \*\* fascia of Gerota prevent extravasation of blood grade 5: kidney is shattered, as 4.

Late Complication of Injury:

A) scarring of kidney (Irreversible)

- B) renal A Stenosis & Aneurysm (can Cause HTN)
- c) urinoma Cleakage of urine out of kidney =>Tx By Drainage
- D) Trauma/ Fibrosis to PUJ (hydronephrosis)
- 2) Ureteral Trauma / Injury: Due to external injury, Penetrating or iatrogenic trauma during Pelvic or Abdominal Surgeries).
  - a. Simple/Perforation: latrogenic; double J insertion, uretroscopy.
  - b. Complex /Transection: due to extensive Surgery; hysterectomy... RTA

\*\*Note: in obstructed ureter Perforation can be Above or Below based on it and the degree of loss of segment we manage.

- If loss <2cm  $\rightarrow$  Direct Oblique Anastomosis. Primary ureteroureterostomy
- If segment loss >2cm -> high level: Trans-ureteroureterostomy.

L, low level: reimplantation of ureter into bladder (Directly /using Psoas hitch) or boari flap)

### **Ureteral lesion Classification:**

grade 1: Contusion, hematoma.

grade 2 transection <50% Devascularization loss of segment.

grade 3. transection >50%.

grade 4: Transection with <2 cm Loss.

grade 5: Transection with >2cm.

### 3) Bladder Trauma/Injury: (Pelvic fracture is M.C.C)

- a. Intraperitoneal (30%): Near Dome of bladder, Need Surgical management (laparotomy & Closure of Perforation to Avoid peritoneal Irritation).
- b. Extraperitoneal (60%): mild: put urethral catheter.

Moderate: urethral Catheter & suprapubic drainage. Sever: Laparotomy & Closure of Perforation.

c. Combined (10%)

Presentation: Hematuria, suprapubic Pain, Acute urine retention

**Investigation**: CT cystography with contrast, +ve = extravasation of Contrast.

4) Urethral Injury/Trauma: Penite Urethra, Prostatic Urethra.

Membranous Urethra: Sphincter Injury = worst urethra

#### \*\* land mark endoscopically is: Verumontanum (opening of ejaculatory) Duct.

- \* Due to Trauma, TURP
- \* Bleeding Per urethra, Bruises in perineal area, Acute retention.
- \* If mild Injury don't do anything.
- \* **PE:** PR exam (overriding prostate) Classic Post. urethra injury (rupture) to know If 2 segment aligned or Not By:
- 1- PR exam (If you feel the Prostate)
- 2- Ascending retrograde Urethrography (Urethral X-ray), contrast inserted through urethra If reached bladder there's Alignment.

Management

- •If 2 segment are in Line  $\rightarrow$  suprapubic Cath.
- If there's misalignment suprapubic Cath. & realignment (railroading). Follow up: Technique Impotence, Incontinence, Urethral Stricture

# Benign Prostatic Hyperplasia ( BPH ):

M.C. benign tumor in males 90% will have it with age: Increase in epithelial & stromal Cell Numbers in transitional Zone.

\* prostate size  $\uparrow$  under effect of DHT (Use 5 -alpha reductase inhibitor).

About Bladder Outlet Obstruction (BOO) causes:

Male: 1. BPH (MCC) 2. Prostate CA 3. Urethral Stricture

Females: 1. Pelvic Prolapse 2. Urethral Stricture / Diverticulum 3. Post- Surgery for Stress Incontinence 4. fowler Syndrome 5. pelvic Masses

Presentation: LUTS: obstruction or irritative, on PE, enlarged Prostate in ORE (PR)

Investigation: IPSS, PSA, TRUS, renal US (Detect hydronephrosis), Cr (Detect) ,PVR (Post - void residual urine volume-> Indicates if watch Fill waiting is safe < 350 mL).

**Complications:** chronic urinary retention, stones, UTI, progressive hydronephrosis, thickened bladder wall, diverticulation predisposes to bladder tumor.

Treatment:

If uncomplicated: watchful waiting.

If LUST bothers the patient, then:

- 1- Life-style modification
- 2- Medical:

A) alph 1 Blockers: subtype selective (Omnic, Tamsolosin) long acting (Terazos, Doxascein).

**SE:** Asthenia, headache, dizziness, Postural hypotension & retrograde ejaculation

B) **5- reductase Inhibitor:** Type 2 specific for Prostate Finasteride / Dutasterid.

-Takes several months to cause decrease in size.

SE: decrease libido, & ejaculation volume, Impotence

**3-** Surgical: A) TURP Transurethral resection of Prostate:

indications: recurrent hematuria, renal Impairment, Acute retention, Bladder stones.

\* Complications: early: bleeding, urethral Perforation.

Late: bladder neck stricture, Incontinence, retrograde ejaculation, Impotence, TURP Syndrome: glycine used in TURB affect GABA receptors & causes cardi supp effect **B) open prostatectomy** 

\* Indications: prostate >100g, failed TURP, long urethra, inguinal hernia, bladder stones.

\* **Contraindications:** small fibrous prostate, PC, abdominal hematuria.

\*Complications: bleeding, rectal perforation, UTI.

# **Urinary stones:**

1- Calcium Ca+ oxalate (80%) : MC type of stones. Radiopaque.
 RF: hypoait raturia, Crohn's and V c abuse.
 <u>Can be caused by:</u> hypercalcemia (PTH) hypercalciuria hyperoxaluria OR
 Idiopathic. <u>Treatment:</u> thiazide, citrate.

**2- Ca Phosphate:** (个 PH) can be caused by distal renal tubular acidosis type 1 due to Hypercalciuria, hypokalemin 个 PH 75.5, Metabolic Acidosis (Radiopaque mc) **Treatment:** Thiazide.

3- Ammonium magnesium Phosphate / struvite stone (15%) ↑PH (7), radiopaque.
- <u>Caused by:</u> infection Urease + Ve Bacteria proteus (mcc) klebsella, Staph.
Produce ammonia → urine alkalization and forms staghorn calculi → obstruction.
Treatment: treat underlying infection and surgical removal.

4- Uric acid: low PH, radiolucent, mcc hyperuricemia (gout) and leukemias (cell turnover)

Treatment: alkalinzation of urine and treat the cause.

5- Cystine-> hexagonal crystals (staghorn): low PH, radiolucent,

Caused by: AR disorders (PCT lose its function in cystine absorption  $\rightarrow$  cystinuria. **Treatment:** urine alkalinization, decrease Na+ intake, chelating agent if refractory.

**Presentation** is the same: flank pain, LUTS, microscopic hematuria.

**RF:**M>F, age 20-30, low water and Ca+ intake, IBD (high oxalate), high Na+ and protein, chemo (increase uric acid) steroid (high Ca+), UTI, hot climate, genes **DDx**: aortic dissection, MI, appendicitis, ectopic pregnancy, muscle spasm, testicular/ ovarian torsion.

Investigations: CBC, UA, renal US, non-contrast CT, KUB for follow up.

## Stones according to location

**Kidney**: All renal stone should be removed even if there is no pain1- Can lead to mortality due to (UTI, SEPSIS, ABSCESS FORMATION)

<1.5 cm ESWL

>1.5 ESWL & double J

- If failed go for:

1- flexible uretroscopy with laser

2-PCNL ={percutaneous nephrolithotomy}

**Ureter:** fever= infection proximal to the stone, urological emergency urine & IV fluid & antibiotic, Nephrostomy drainage if fever doesn't resolve within hours. **Management:** 

-start with NSAID, opioid, alpha blockers

-watchful waiting:

if it was small pass within days- weeks.

If > 2months {alpha blocker helps in passage}

- <5 mm/ No JJ: medical therapy and wait for 1-2 weeks, If failed-> definitive Tx
- >5mm JJ or nephrostomy (pt. Unwell or obstructive pyelonephritis)

definitive Tx:

1- upper 1/3: ESWL/ flexible ureteroscopy/ lithotripsy (Pneumatic)

2- Middle 1/3: rigid ureteroscopy

3- lower 1/3: ureteroscopy then begin with ESWL

Bladder: mostly made from: struvite or uric acid, men >50 yr. (BPH)

-must be removed due to increased risk for SCC

-management if :

-<2 cm endoscopic cystolitholapaxy

->2 cm open cystolitholapaxy

Urethra: Small tone clear spontaneously

-Plugged large stone needs removal

-If at external meatus by forceps

-If up in urethra push it back to bladder (folly's) then Tx as a bladder stone.

### About definitive Tx methods

- 1) ESWL (stone fragmentation by electromagnetic method)
- Depends on stone size (<2 cm), location, degree of obesity (not used in morbid obese pts) and stone composition (shouldn't be cysteine or calcium oxalate (very hard)).
- Contraindicated in: pregnant women, morbid obese man, blood clotting disorder.
- Side effects: fxn renal damage, hematuria, AKI (in HTN and DM pts), prolonged coagulation.
- 2) Flexible ureteroscopy and laser (more effective than ESWL)
- Indication: ESWL failure, lower pole stone, obese pt, cysteine stone, stone in calyceal infundibulum or diverticulum, horseshoe kidney.
- 3) PCNL (percutaneous nephrolithotomy) = removal of stone by tract between skin and collecting system
- Indication: 1<sup>st</sup> line for staghorn calculi and if stone > 2 cm or ESWL/ ureteroscopy failure
- Contraindication: UTI/bleeding tendency
- 4) Lithotripsy (pneumatic/ laser/ mechanical/ ultrasound)
- 5) Open surgery
- Indications: non fxn kidney, complex stone, failure of endoscopic treatment
- 6) Medical dissolusion therapy
- Uric acid stone -> hydration, urine alkalization, allopurinol, low protein diet
- Cysteine stone -> diet (low methionine), alkalization, low na+ and use drugs: d-penicillamine, N-acetyl-d-pen mercaptopropionylglycine

NOTE THAT: Mg, GAGs, Inorganic phosphate, Tam-Horsfall protein can decrease crystallization of stones (In general).

# **Urinary Incontinence:**

failure to store urine due to Abnormal bladder SMCs or deficient sphincter, ectopic ureter causes total incontinence.

A. Bladder Abnormalities: filling phase defect.

**1-Detrosor Overactivity:** Neurological Problem (Spinal Cord Injury, radical hysterectomy, radiation Cystitis)

**2-Low Bladder Compliance:** decreased Volume to Pressure relationship of bladder. Caused by (chronic Catheterization, Prostatic Obstruction or Increased Collagen die to cystitis)

# **B. Sphincter Abnormalities:**

**1-Urethral hypermobility:** weakness of Pelvic floor muscle (Bladder neck will descend with Proximal urethra causing urinary leaking)

**2-Intrinsic sphincter Deficiency:** due to malfunction regardless it's Position <u>Caused by:</u> Surgery, aging, radical pelvic surgery, menopause, Child birth

\*\***RF:** F>M, Collagen Anomalies, Obesity, Smoking, UTI, Aging, decreased mobility, Prostate / Pelvic Surgery, genetic Predisposition, Neurological Disorder

## Types

 1-Stress (Recurrent UTI is not RF in it): Urine leak with exertion, Sneezing, cough; Due to hyper-mobility of the bladder and intrinsic sphincter deficiency
 2-Urge: Leakage with Sudden Strong desire to void urine; due to detrosor instability neurogenic bladder

3-Mixed: Stress and urge UT

4-Overflow: Due to chronic Urinary retention (BOO; bladder outlet obstruction)
5-Functional: Normal Voiding System but have difficulty to reach toilet
6-Total: Constant leakage during day & night

# Stages:

- $0 \rightarrow$  Incontinence without clinical sign
- 1ightarrow leakage during stress & Descend of bladder <2 am of symphysis pubis
- 2→ leakage during stress & Descend of bladder >2 am of symphysis pubis
- 3→ Bladder Neck & Proximal urethra is ofened" (During rest)

**\*\*Hx:** ask abut LUTS, tiggers for incontinence (coughing, sneezing, exercise, position, urgercy)

\*\*PE: -examine Abdomen for Palpable bladder (chronic retention) -Ask Pt to cough/sneeze & inspect for Prolapse & Urinary leakage

**\*\*Investigation:** Urinalysis, Bld test, X-ray, Cystoscopy, uroflow test Bladder Diaries (record frequency, volume of urine voided, Incontinence Episodes)

\*\*Management: Tx of Overactive bladder (OAB):

-Conservative management → Pelvic floor exercise, modify fluid Intake, avoiding Stimulants: caffeine, Alcohol

- Medication To inhibit Contraction: TCA, desmopressin (ADH), GABA agonist. -Injection of botulinum Toxin

-Surgery (Defrosor myectomy) to  $\uparrow$  functional blader capacity

# \*\*Tx of choice for stress Incontinence is Surgery (But) for urge Incontinence medical Tx usually successful

# **Urinary Tract Infection - UTI**

-protein in urine is usually < 30 mg/dl

-m.c symptom of UTI is Dysuria

-It's Inflammation of urothelium due to bacterial Invasion.

-Bacteriuria Presence of bacteria in urine (Asymptomatic)

-Pyuria; Presence of WBCs in urine in dipstick or {10 WBC /hpf}

-Complicated UTI: In M, when there's functional / Anatomical Abnormality or Immunocompromising.

-Uncomplicated: In F, No Functional /Anatomical abnormalities (NO recurrence) -Isolated: There's an interval of 6 m. (at least) btw: Infection

-recurrent: > 2 infection in 6m. of 3 within 12 months. (Due to reinfection/ Different Bacteria or due to Persistent of the same bacteria)

# \*\*M.C.C of Infection→ E-coli

**routes of Infection**: Ascending (M.C), hematogenous, lymphatic spread  $\rightarrow$  + - Defense mechanism that Prevent UTI -> (Lysozyme, Lactoferrin, IgA in urine, Flushing of urine, decreased PH of urine)

**Factors that ^ Bacterial virulence:** Adhesion factors, capsule, toxins, enzymes.

**Chronic Pyelonephritis:** Fibrosis (scarring) of renal Parenchyma (recurrent Persistent) Infection, end result of long standing reflux of Infected urine or obstruction (Affects upper & Lower Poles of kidney)

\*Scar seen on US, CT scall, IVP, renal Isotope scan

#### **Types:**

-Non-Obstructive Pyelonephritis -Obstructive Pyelonephritis.

### **UTI classification:**

### 1- Upper UTT:

A. Acute Pyelonephritis:

Hx: Flank Pain and tenderness, fever, chills and high WBC (one or both kidneys)
-Causes: 80% due to E-coli -> Strep fecalic, Klebsiella, Proteus, Pseudom
-RF: DM, Pregnancy, Tract Onstruction, Catheter, VUR
-Investigation: Urinalysis, CBC, Cr, Bld Culture (Immuno decreased in Pt)
-Tx:
1-Fever /Not Systemic ill, give Flauroquinolones, Bacterin (10-14 D.)
2-Systemic ill: IV Fluid & Antibiotics then switch to oral agents for 14 D.

Used for: Complicated Infection, Persistent V, extremes of age, failure of OPT.

**Emphysematous Pyelonephritis:** rare severe form, usually occur in DM Patient & Precipitated by obstruction, characterized by high Fever & Abd. Pain (Intrarenal gas seen on CT scan) Pts. are very unwell with ↑ mortality rate.

**\*\*TX:** IV Fluid & IV Antibiotic, Percutaneous Drainage (emergent Nephrectomy when sepsis occur)

#### Xanthogranulomatus Pyelonephritis: (by E.coli)

-Severe renal Infection (Due to renal Stone /Obstruction) kidney is grossly enlarged, contains yellowish Nodule with pus & hemorrhagic Necrosis, -Acute flank Pain, fever, flank mass. (renal calcification on CT). -Around the Abscesses (within Parenchyma) there's fat-filled macrophages.

Tx: IV Fluid & Antibiotic

### 2- Lower UTI:

A-Cystitis: (cloudy urine>> bacterial cystitis)

-Infectious: GLUTS, Suprapubic Pain I fever, bad odor urine.

### - Non-Infectious.

-**Types:** Interstitial, follicular hemorrhagic cystitis, glandular Cystica. -**Tx:** oral Antibiotic (Trimethofrem / sulfameth.)

## **B-Urethritis**

-Hx: LUTS, Discharge (No Irritation symptoms)

-Causes: N. gonorrhea (M.C.C) & Chlamydia Trach.

-Dx: culture from urethral swab (large Amount of yellow Dish),

-Tx: Azithromycin $\rightarrow$  chlamydia

Fluoroquinolone  $\rightarrow$  n.gonorhea (-ve Difc)

C-Prostatitis: Due to reflux of Infected urine into Prost. Duct

-RF: UTI, wethral Cath.

-Caused: by E-coli, Proteus & Klebsiella.

**-Types:** Acute, chronic, Chronic Pelvic Pain Syndrome, Asymptomatic Inflammatory.

# **Scrotal Pathologies:**

## 1-Testicular Torsion:

-Mostly in Left testis, gold period to refer is 6 hs (cause Irreversible Ischemia) -Hx: sudden testicular pain.

-PE: Doppler US, Radio-Isotope scan (Most sensitive).

**Tx:** surgical exploration and detorsion within 6 hs.

# 2-epididymo-orchitis (Inflammation of testis & epididymis):

-Acute <6w. If chronic there's Pain but no swelling.

-In Male <35 yrs  $\rightarrow$  caused by; N. gonorrhea, C. Trachomatic.

-In children & Older men caused by  $\rightarrow$  E-coli, Proteus, Klebsiella, Pseudom.

- -DX: Doppler US & Radio-Isotope scan.
- -Tx: Analgesia & scrotal elevation, then according to grade:

Low  $\rightarrow$  Oral Antibiotic high  $\rightarrow$  IV Abx. (Flouroquinilone, cephalosporin))

- Complication: Infertility, Acute hydrocele, recurrence

Testicular Torsion	Epididymo-Orchitis
-Age btw 10-30 (Peak 14)	-In rexually-active men from uti
-Sudden Pain (hemiscrotum)	- Sudden /gradual Pain
** Associated with Nausea	** No Nausea
** Wakes Pt from sleep	** less severe Pain
-Hx of Minor trauma to testis	-Hx of wethritis, LUTS, STD
-Swollen, tender, high riding testis	-epididymis swollen, tender, Painful
-Abscent Cremasteric reflex	-Relieved by elevation
-Not relieved by scrotal elevation.	

**3-Varicocele:** (Dilation of Veins of Pampiniform Plexus due to Incompetent Valves in the Internal Spermatic veins) -M.C.C of Infertility (Lt > R).

-majority Asymptomatic but large varicocele may cause pain, heaviness in scrotam. -PE: Appears while standing with Valsalva Maneuver.

-Dx: Doppler, Semen Analysis (oligoasthenoteratospermia, reduce count &motility)
 -management: Surgical ligation if: Severe Pain, small testis on same side, Infertility.
 - grading:

grade 1 (small, Palpable on Valsalva).

grade 2 (Moderate, Palpable in Standing position).

grade 3 (large, palpable & Visible through skin).

4-hematocele (After Trauma): Painful collection of Blood in tunica vaginalis 5-Hydrocele:

Due to Inflammation, trauma, tumor, defective Absorption, congenita.

-Lies Anterior to testis (small ones) but large ones surround it.

-types: Communicating (Patent Processus vaginalis), Non-communicating.

-Dx: Trans-Illumination (you can get Above it while hernia you can't), U/S

-Tx: Surgical excision of hydrocele sac.

-Complications: long standing hydrocele Infection, testis Atrophy

### 6-Epididymal Cyst / Spermatocele / Vasa Efferentia:

-Derived from collecting tubules of epididymis, contains clear fluid.

-Lies (Above & P) to testis, often multiple, multiloculated.

-Dx: US, Trans-Illuminates.

-Tx: Spermatocelectomy.

# **Erectile Dysfunction**

\*If there is an early morning erection, then we rule out organic causes.

\*An organic cause usually has a gradual onset.

\*Erection is caused by Parasympathetic Stimulation.

\*Ejaculation and detumescence (loss of erection) caused by stimulation.

-Mechanism of erection:

-expansion of sinusoidal spaces against tunica Albugica (in corpus Covernosum) this will decrease Venous outflow & trapping blood within the erect Penis.

-this happens under effect of NO, CGMP -> vasodilation & muscle relaxation.

\* During ejaculation; alkaline prostatic secretion is discharged 1<sup>st</sup> followed by Spermatozoa & finally seminal vesicle secretions, ejaculate Volume is 2-5 mL

erectile Impotence: Persistent Inability to achieve or maintain an erection.

-Causes:

\*Inflammatory: Prostatitis.

\*Mechanical: Peyronie's disease.

\*Psychological: Depression, anxiety, stress & mcc.

\*Occlusive: HTN, Smoking, DM, DVD, hyperlipidemia.

\*Trauma: Pelvic fracture, spinal cord Injury, Penile trauma.

\*Neurogenic: MS, Parkinson, multisystem atrophy, Alcohol related.

\*Chemicals: Antidepressant. Statin, Anxiolytic, Anti-Parkinson.

\*Endocrine: hypogonadism, hyper Prolactin, hyper /hypo-thyroid.

-not a normal aging process >> complete erectile dysfunction

-PE: DRE (Assess Prostate), external genitalia exam. (Penile lesion, testicular exam) Bulbocavernous reflex (test Integrity of spinal Segment).

-Investigations: PSA, glu, testosterone, FSH, LH, Prolactin, TFT, color Doppler US. -Treatment:

# [1] First Line:

a-PDE5 Inhibitor (Sildanafil)

•Blocks breakdown of CGMP (maintains erection)

•Contra. in Nitrates, stroke Pt recent MI

**b**-Vacuum erection device (↑ Bld flow to Corpora Cavernosuim)

**c**-Intraurethral Therapy  $\rightarrow$  Injection of PGE5 (cGMP)

[2] Second Line: intracavernosal therapy of PGE 1

[3] Third Line: Penile Prosthesis (Implanted into corpora to provide Penile rigidity)

**Retrograde ejaculation:** Failure of adequate bladder neck contraction resulting in Propulsion of sperm, back into the bladder on ejaculation.

-Presentation: Dry ejaculation, Claudy urine containing sperm after 1st Void (following intercourse)

- **Causes:** Neurological, DM, Spinal Cord Injury, &-Blocker (tamsulosin), TURB or Prostatectomy

-Investigation: Presence of >10-15 sperm Per hpf in urine

-Tx: Oral Adrenergic (ephedrine),  $\uparrow$  Sympathetic. tone of Bladder neck SMCs

# Testicular cancer (TC):

- It's relatively rare, 1-1.5% of M cancers.
- More in **white**, less in African Americans.
- Primary are the MC solid malignant tumor in young men 20 to 35 years old.
- Germ cell tumors (90-95%) all are malignant.
- Interstitial tumors (1-2%) usually benign.
- Testicular cancer is highly curable and has good prognosis.
- Lymphatic spread: testicular lymphatic drainage is to para-aortic LN
- Involvement of the epididymis, spermatic cord, and scrotum may lead to pelvic or inguinal node metastasis
- Inguinal LN enlargement indicates:
- Advanced stage of testicular cancer.
- It may be unrelated to cancer, infection for example.
- Testicular cancer with scrotal involvement
- The patient had a previous scrotal surgery which caused disruption of lymphatics.
- Blood-borne metastasis to the lungs, liver, and bones is more likely once the disease reaches the tunica albuginea.

# Factors that determine if testicular cancer will be diagnosed early or late: Patient factors:

1- Embracement

2- Cultural and sexual education of the patient; some patient may consider the enlargement in testicular size (which occurs in testicular cancer) as a part of normal sexual development.

3- Scrotal self-exam is not well-known in the society.

### • **Physician factors:**

1) incomplete physical examination

2) Wrong diagnosis: mistaking the cancer for a hydrocele. Avoid this by perform US

- Risk factors:
- **Cryptorchidism** \*\* (orchidopexy doesn't prevent cancer development)
- Contra lateral testicular cancer
- Germ cell neoplasia in situ(GCNIS)
- Family history of testicular cancer (Genetic factors)
- Klinefelter syndrome, trisomy21(increased risk for germ cell tumors)
- white people
- HIV, recurrent infections / Maternal estrogen ingestion / Trauma / Gonadal dysgenesis
- Presentation:
- It is usually painless
- Can be painful: dull pain or acute pain that occurs due to intratumoral **necrosis or hemorrhage** (rapid growth)
- Unilateral> bilateral, Right > left.
- Approximately 5% of men have gynecomastia, resulting from elevated serum HCG levels >> mostly in choriocarcinoma.

DDx: hydrocele, spermatocele, hematoma, hernia, epididymitis, TB.

PE:

- Asymmetry or slight scrotal skin discoloration
- Hard, non-tender, irregular, non-trans-illuminating mass, non-reducible
- Supraclavicular lymphadenopathy, hepatomegaly, LL edema, Gynecomastia

Dx: Scrotal US, Image contralateral testis (2% of patients will have it bilateral).

- Abdominal and chest CT: for staging purposes
- Serum tumor markers are very useful in staging and follow up.

- Any solid testicular mass should be managed as malignancy until proven otherwise and <u>histopathology should always be done</u>.

All are treated by inguinal radical orchiectomy to prevent seeding. Also, a diagnostic tool to determine T stage (biopsy is not allowed from fear of seeding).
 Radical orchiectomy involves excision of the testis, epididymis and spermatic cord, with their coverings. Sperm cryopreservation should be offered for both single and bilateral orchiectomy patients.

-If you suspect cancer in patients with single testes you can use frozen section approach and do partial orchiectomy if possible.

- serum markers are measured 1-2 weeks after radical orchiectomy and during follow-up to assess response to treatment and residual disease.

- Normal markers prior to orchiectomy do not exclude metastatic disease (some TC present with normal levels of serum markers). Normalization of markers post-orchiectomy does not exclude absence of disease. Persistent elevation of markers post-orchiectomy usually indicates metastatic disease.



Seminoma: MC germ cell tumor -25-35 y/o, rarely bilateral.

- Appears pale and homogenous (NSGCTs are heterogeneous and sometimes contain tissues such as cartilage or hair)
- Pure seminomas never secrete AFP. 10% of them secrete  $\beta$ -hCG
- At diagnosis, it is usually confined to the testis
- Treatment: radical inguinal orchiectomy + has excellence response to RTX
- Stage 1: bilateral inguinal orchiectomy + retroperitoneal LN dissection +/- RTX
- Stage 2a/2b: bilateral inguinal orchiectomy + RPLND +/- CTX
- Stage 2c/3: bilateral inguinal orchiectomy + high dose CTX

### Embryonal:

Usually occurs as component of mixed tumor Pure embryonal carcinoma rare (2% testicular GCTs) Key distinctions from seminoma: Mass with hemorrhage and necrosis =painful May have syncytiotrophoblast tissue, secretes β-hCG Tx: Poor response to CTX and RTX

### Yolk sac tumor:

-Infants and children <3 years old.

-Secretes AFP (AFP normally derives from yolk sac).

-Hematogenous spread.

• Hallmark: Schiller-Duval bodies (Glomerular-like structures ("glomeruloid")

## Chroricocarcinoma:

- $\bullet$  Syncytiotrophoblast and cytotrophoblast cells (always secretes  $\beta\text{-hCG}$  )
- Hematogenous spread, especially to lungs and liver very early.
- No LN involvement (the only one)
- WORST PROGNOSIS

# Teratoma:

- Cells from all three germ layers: Ectoderm (skin, hair follicles), Endoderm, Mesoderm (cartilage)
- Large mass: Neural tissue, muscle, cartilage
- Often part of a mixed tumor in adults
- Pure teratoma usually seen in young children: Mean age: 20 months, before 4.

### Leydig cell tumor:

- Most common non-germ cell tumor
- Strongly associated with cryptochordism
- Management: bilateral orchiectomy + RPLND

### Sertoli: Usually

- do not produce hormones
- Most are benign
- -Management: bilateral orchiectomy + RPLND

### Lymphoma:

- Non-Hodgkin lymphoma may involves testes
- Diffuse large B-cell lymphoma most common subtype
- 5% testicular cancers = lymphoma
- Most common testicular tumor men > 60 years old
- often bilateral aggressive malignant cancer
   Tx. Radical orchiectomy + chemo

#### Gonadoblastoma:

- Seen in patients with gonadal dysgenesis
- Management: radical orchiectomy + gonadectomy of the contralateral gonad (50% bilateral)

### **RPLND (retroperitoneal LN dissection)**

Retrograde ejaculation is a major complications: damage to sympathetic innervation, avoidable by modified RPLND: spare one side of sympathetic innervation. \*\*Limits for right-sided tumors consist of ureter (lateral), midpoint of aorta (medial), bifurcation of iliac vessels (inferior) and renal hilum (superior). \*\*Limits for left-sided tumors consist of ureter (lateral), midpoint of vena cava (medial), bifurcation of iliac vessels (distal) and renal hilum (superior).

### Pediatric Urology

- *1. Testicular Maldescent:* In 7th month of gestation, the testicles normally descend into the scrotum. Continued descent of the testis may progress after birth, but descent comes to a halt before 2 years of age.
- In up to 4% of normal full-term newborn males, one or both testes have failed to reach the scrotum, and this percentage increases with prematurity (30%).
- By the age of one year, full descent will occur in most boys, leaving about 0.3% with maldescended testes. (A testicle is considered truly undescended at one)
- The cause of most cases of cryptorchidism is unknown, but a deficiency of androgen production by the fetal testes is an important factor.
- The maldescended testes may be arrested at any point on its path of descent from the posterior abdominal wall to the scrotum: Abdominal, Inguinal canal, At the external ring (mc), Perineal, Superficial inguinal pouch, High retractile
- The right testis is affected in 45% of cases, the left in 30%, and both testis in 25%.

**Complications:** 

- Testicular malignancy (especially seminoma) X 30, Surgical correction doesn't reduce this risk; however, a testicular tumor is more likely to be discovered early if the testis is in the scrotum.
- Subfertility: Neonatal spermatogenesis requires scrotum cooler temperature. all bilaterally cryptorchid adult males become sterile if untreated.
- Traumatic injury
- $\circ$  Torsion.
- Patent processus vaginal is present in 95% of patients with cryptorchidism, and approximately 25% develop a clinical hernia.
- Anomalies associated with cryptorchidism occur in about 15% of cases: Klinefelter syndrome, hypogonadotopic hypogonadism, renal agenesis, horse-shoekidneys, extrophy of the bladder, ureteral reflux and others.

PE: empty hemiscrotum with absent rugae.

**Treatment:** orchidopexy -best performed before 2. The usual technique involves mobilizing the spermatic cord and placing the testes in a subcutaneous scrotal pouch outside the dartosmuscle.

### Retractile Testes:

- Here, the testes don't appear to be fully descended, and can be palpated in the scrotal neck and gentlymanipulated into its correct position.
- It is due to the very active cremasteric muscle in children under 3 years of age and small testis. It is avariant of normal.
- no treatment required; testes become less retractile as the boy grows.

### Vesicoureteric Reflux

- The main function of the ureterovesical junction is to permit free drainage of the ureter and simultaneously prevent urine from refluxing back from the bladder.
- The ureteral musculature continues uninterrupted into the base of the bladder to form the superficial trigone, and this direct continuity offers an efficient, muscularly active valvular function. Thus, any stretch of the trigone (with bladder filling) or any trigonal contraction (with voiding) leads to firm occlusion of the intravesical ureter, preventing retrograde flow.

Causes:

- Primary VUR
- Developmental uretero-trigonal weakness
- anomalies: Ectopic ureter, Duplex ureter, Congenital megaureter, Ureterocele.
- Secondary VUR
- Bladder outlet or urethral obstruction
- Neuropathic dysfunction
- latrogenic causes
- Infection. e.g.: TB
- Stones and foreign body

Complications: Pyelonephritis, UTI, ESRD.

**Clinical findings:** 

- Infants and young children: Non-specific (due to UTI), Fever, Failure to thrive.
- Older children: Incontinence, Frequency, Dysuria, Abdominal pain
- With acute pyelonephritis: Fever and chills, Costovertebral angle tenderness

**Diagnosis:** 

- Urinalysis and urine cultures: Evidence of infection; pyuria and bacteriuria.
- Abnormal renal function tests
- Radionuclide voiding studies
- Voiding cystourethrogram: This is the standard investigation. It demonstrates the grade of the reflux and the urethral anatomy. It involves injecting contrast medium into the bladder using a urinary catheter. The catheter is then removed and x-rays are taken while the child is voiding;

Grade I	reflux into a non-dilated distal ureter
Grade II	reflux into the upper collecting system without dilation
Grade III	reflux into a dilated collecting system without blunting of calyces
Grade IV	reflux into a dilated system with blunting of calyces
Grade V	massive reflux with gross dilation and distortion of the ureter and collecting system

### Management:

When there is no ureteral dilation, there is an 85% chance of spontaneous resolution as the child grows. In the meantime, the urinary tract must be kept free of infection, and this is done by:

- Regular voiding.
- High fluid intake.
- Avoiding constipation.
- Maintaining perineal hygiene.
- Anti -bacterial chemotherapy.
- Regular follow up, with charting of growth and development.

\*\*In obstructive secondary reflux (e.g. posterior urethral valves), release of obstruction may cure reflux.

\*\*In neuropathic reflux, intermittent catheterization for control of infection may allow return of valvular competence.

**Treatment:** Surgical correction by reimplanting the ureter in the bladder wall so that a length of it liesdeep to the bladder mucosa. This is indicated for:

- severe reflux with dilated ureters
- For other anatomical abnormalities
- For children who fail to progress on conservative management

#### **Ureteral anomalies**

- Congenital obstruction of the ureter
- Duplication of ureters
- Ectopic ureteral orifice
- Ureterocele

### **Bladder anomalies**

- Bladder agenesis
- Bladder duplication:
- Complete; with separate urethral openings drained by duplicated urethras
- Incomplete; with a septum deformity
- Urachal anomalies:
- Urachal diverticulum
- Urachal cyst.
- Extrophy of the bladder: complete ventral defect of the urogenital sinus and the overlying inferior abdominal wall musculature.

**Presentation:** 

- The lower central portion is devoid of skin and muscles.
- A bladder wall is absent, and P wall is contiguous with the surrounding skin
- The rami of the pubic bone are widely separated, and the open pelvic ring may affectgait
- Urine drains onto the abdominal wall
- In males, the penis is shortened, and the urethra is epispadiac
- The exposed bladder mucosa tends to be chronically inflamed.
   Treatment:
- Closure of the bladder in the newborn period
- Urethral closure and penile reconstruction
- Ureteral re-implantation

### Penile and urethral anomalies

- A. Hypospadias: results from failure of fusion of the urethral folds on the undersurface of the genital tubercle.
- Incidence: 1 in 400 male births
- The urethral meatus is ventrally displaced on the glans, shaft, the level of the scrotum, or perineum.
- The remnant of urethral tissue distal to the meatus is fibrotic, causing the penis to bend downwards or be erect (chordee). The more proximal the urethral meatus, the worse the chordee is.
- The ventral part of the foreskin is absent giving rise to a hooded appearance.
- In hypospadias with the meatus positioned proximal to the corona, circumcision shouldn't be done, as the prepuce can be used later in surgical repair.

Management:

- If the opening is glandular or coronal (85%), the penis is usually functional, and repair isdone primarily for cosmetic reasons
- More proximal openings require correction (surgical plastic repair).
- Complications of surgery include meatal stenosis and fistula formation.

### **B. Epispadius:**

- The urethra opens on the dorsum of the penis, with deficient corpus spongiosum and looselyattached corpora cavernosa.
- If the defect is extensive, it may extend to the bladder neck causing incontinence.
- The pubic bones are separated, as in extrophy
- Marked dorsiflexion of the penis is usually present
- It is commonly associated with bladder extrophy, and if present alone is considered as a milddegree of the extrophy complex.

Management:

- Correction of penile curvature
- Reconstruction of the urethra and bladder neck

### **Urethral strictures**

Most common in the fossa navicularis, just proximal to the meatus, and in the bulbo-membranaous urethra. They are thin diaphragms that respond to simple dilation or direct-vision internal urethrotomy. Open surgery is rarely required.

### Posterior urethral valve

The most common obstructive urethral lesions in newborn and infant males, and the most common cause of end-stage renal disease in boys.

They are obstructive mucosal folds, seen only in males, which originate at the veru montanum at the prostatic urethra.

**Clinical manifestations:** 

- Difficult voiding
- Weak stream
- A lower abdominal mass that represents a distended bladder
- Palpable kidneys with signs of acidosis and uremia
- Urinary incontinence and UTI
- Up to 70% have VUR

Laboratory findings:

- Elevated BUN and creatinine
- Evidence of UTI
- U/S shows evidence of bladder thickening and trabeculations, hydroureter and hydronephrosis
- Voiding cystourethrogram demonstrating the urethral valves establishes the diagnosis.
- Treatment: Endoscopic destruction of the valves as soon as possible.

# Hematuria:

**Gross hematuria** - observable through direct visual inspection of the urine. Overall urine color may be pink, red, brown.

Microscopic hematuria - only detectable by microscopic examination of the urine (five or more RBC/HPF).

#### symptoms:

- Amount of blood and Colur of urine/blood
- Relationship of blood to stream: Initial -> urethral cause
  - Terminal -> prostate or bladder neck, bladder stone
  - Total -> bladder and above.
- Presence of clots (pathology is in the UT rather than from the renal parenchmya)
- Irritative symptoms: Frequency, urgency and nocturia.
- risk factors for malignancy: WL, anorexia, smoking and family history.
  - flank pain that radiates to the groin: suggestive of a uretric stone.
- Concurrent pyuria + dysuria = UTI
- Recent URTI = infection-related glomerulonephritis positive
- FH of kidney disease: hereditary nephritis, polycystic kidney, or sickle cell disease.

- Unilateral flank pain, which may radiate to the groin, suggests ureteral obstruction
- Symptoms of prostatic obstruction in old M (hesitancy& dribbling) suggests BPH
- History of a bleeding disorder or bleeding from multiple sites due to excessive anticoagulant therapy.
- Cyclic hematuria in women that is most prominent during and shortly after menstruation, suggesting endometriosis of the urinary tract
- LL swelling or rash is suggestive of a glomerular origin.

**PE:** Vitals, abdominal examination for tenderness (renal angle), palpation for a masses or urinary retention, careful examination of genitalia.

\*\* DRE in M to assess for enlargement (prostate cancer) or tenderness (prostatitis).

### Diagnosis:

Cytology, cystoscopy, CT with contrast Labs: KFT, CBC, UA, culture, INR.

**Urine analysis:** detects heme in urine (high sensitivity, low specificity. + does not = hematuria because the test does not distinguish between the presence of RBCs, hemoglobin, and myoglobin.

**Urine sediment:** Confirm hematuria with microscopy (≥ 5 RBCs/HPF).

- If RBC casts and proteinuria: Evaluate for glomerular diseases.
- If the morphology of RBCs is normal: Evaluate for non-glomerular causes (e.g., coagulation disorders, kidney stones, malignancy).

\*\*urine remains pigmented after centrifugation because the pigments are dissolved in the urine and do not settle at the bottom like RBCs in hematuria.

Culture: if clinical signs of infection or + WBCs dipstick (pyuria) and/or leukocyte esterase.

**US**: Urinary tract neoplasm, stone disease, inflammatory processes, congenital abnormalities, vascular lesions, and obstruction.

Not likely to show non-obstructing ureteral stones or small urothelial abnormalities.

Cystoscopy:

- > All patients with gross hematuria without evidence of glomerular disease or UTI.
- If the patient has evidence of glomerular disease but there is blood clots.
- Gross painless hematuria in a patient > 35 should be considered as cancer until proven otherwise-> CYSTOSCOPY.
- 4 85% of patients with bladder cancer and 40% of patients with RCC present with gross hematuria.

\*\*uninfected patients (UTI), subsequent evaluation depends upon whether the hematuria is gross or microscopic:

- 1. gross hematuria with visible blood clots: CTU, Cystoscopy.
- 2. gross hematuria without visible blood clots in the urine
  - A. Patients with AKI or findings suggestive of glomerular bleeding-> nephrology.
  - B. Nonpregnant patients without AKI: CTU Cystoscopy.
  - C. Pregnant patients: kidney and bladder US.
- 3. microscopic hematuria:
  - A. Patients with AKI or findings suggestive of glomerular bleeding-> nephrology.
  - B. Pregnant patients: kidney and bladder US.
  - C. Nonpregnant patients who have risk factors for malignancy: CTU, Cystoscopy.
  - D. Nonpregnant patients who have no risk factors for malignancy: do not require imaging studies or cystoscopy.

**Transient hematuria:** Up to 50% of patients with microscopic hematuria have no apparent cause after thorough evaluation.

In these patients' prognosis is excellent and it is recommended to monitored with annual urine analysis. After two negative urinalyses we can stop the follow up.

### Treatment:

- ✓ Asymptomatic (isolated) hematuria generally does not require treatment.
- Microscopic hematuria in a woman during her menses, or in a patient shortly after vigorous exercise or acute trauma, confirmed by repeating urinalysis.
- ✓ In UTI, urinalysis should be repeated approximately 6 weeks after completion of antibiotic therapy in order to determine if it is persistent.
- ✓ If abnormal clinical, Labs, or imaging, treat the cause.
- Surgical intervention in certain anatomic abnormalities (ureteropelvic junction obstruction, tumor, or significant urolithiasis).
- ✓ persistent microscopic hematuria monitored every 6-12 months for the appearance of signs or symptoms indicative of progressive renal disease.