Nephrology Theme

Contents:

Medicine:

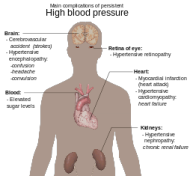
Surgery:

Hypertension

- Risk factors:

1-  2- Age (60) 3- ♂ 4-  5-  6- 

- Complications of uncontrolled hypertension:



- Diagnosis:

1- Blood pressure: unless the patient has severe HPT or evidence of end organ damage, do BP test at least 2 times on a period of 4 weeks to establish the diagnosis.

- Treatment:

Patient classification	BP Goal	Recommended managements	Note
General population >=60 y	<150/<90	Non-black→ Thiazides, ACEI/ARB or CCB **Black**→ Thiazide or CCB or in combination	Thiazides and CCB are equally first line therapy.
General population < 60 y	<140/<90		-
Diabetic no CKD	<140/<90		-
CKD + - DM	<140/<90	ACEI or ARB	-

- Life style changes are important.	- ARB→ preferred in Diabetics (Doesn't cause dry cough)
- beta-blockers → are used: in heart related conditions and depression	! = ACEI and ARB should not be combined.
- ACEI→ is preferred in diabetic patients (causes dry cough)	- most patients will eventually need more than one drug.
- alpha- blockers→ used (not first line) in BPH "prostatic" patients.	- CCB→ Dihydropyridine "most commonly used"
- Don't use Beta-blockers as first line therapy due to Cardiovascular side effects	- chlorthalidone is better than lisinopril.
- if HPT is mild and controllable patient can stop medications, but must check the BP regularly.	

- Side effects of drugs:

Drug	Side effects
Thiazides	Hypokalaemia, hyperuricemia (↑ Uric acid in urine), hyperglycaemia, metabolic alkalosis. (4)
Beta- blockers	Bradycardia, bronchospasm, sleep disturbances, fatigue, sedation, depression. (6)
ACEI	dry cough, Hyperkalaemia, Acute renal failure, skin rash. (4)
CCB	Amlodipine → peripheral edema Verampil, dilteazim→ heart block

Diabetic nephropathy

- Secondary to a glomerular disease.
- Usually manifests after 15-25 years of diagnosis of DM.
- Microalbumin urea → can't be detected by dipstick, can be detected by special dip or immunoassay.
- Microalbumin urea is the first evidence of Diabetic Nephropathy
- Microalbumin urea could develop to intermitted albumin urea or persistent protein urea.
- Kimmelstiel lesion → nodular microscopic change.
- Persistent protein urea = 5-10 years from end stage kidney disease. Which could lead to:

1- transient nephrotic syndrome 2- peripheral edema 3- hypoalbuminemia

- **End stage renal disease** (ESRD) is the last stage (stage five) of chronic kidney disease (CKD) **= Kidney Failure**

- Patients with nephropathology usually present with normocytic normochromic anemia. And ↑ Electrolyte sedimentation rate (is the rate at which red blood cells sediment in a period of one hour. It is a common haematology test "sediment = matter that settles to the bottom of a liquid").
- ↑ creatinine is a late feature that develops to renal failure eventually.
- Untreated infection in DM patient could lead to renal papillary Necrosis.

❖ Diagnosis:

- albumin: creatinine Ratio = men < 2.5. Women < 2.5

- plasma creatine level GFR - GFR - Microalbumin in young patients

- Test for protein urea at least once a year.

❖ Treatment:

- ACEI/ARB → delay the onset of protein urea

- ACEI/ARB → for hypertensive patients or Normotensive patient + Micro albumen urea.

!: Hypoglycaemic agents should be avoided. E.g. Metformin, glebelands (because it's excreted by the kidney.

- may need to Decrease insulin intake.

- Ophthalmic supervision.

* End stage management segmented islet graft → improve survival.

Acid-base balance

Objectives:

1- State the normal value for PH, PCO2, HCO3

2- Understand the basic mechanism of acid base disturbance

3- Interpret basic acid base disturbance

4- List common differential diagnosis for different acid base disorder

- **Normal values: (Objective 1)**
  PH= 7.35-7.45 PCO_2=35-45 HCO3=22-26 Anion gap=8-12

			(objective 3)		
Primary Disorder	Diseases associated	Problem (Objective 2)	pH	HCO_3	P_aCO_2
Metabolic acidosis	Lactic acidosis, Cardiac arrest.	gain of H^+ or loss of HCO_3	↓	↓	↓
Metabolic alkalosis	Loss of gastric secretions Vomiting	gain of HCO_3 or loss of H^+	↑	↑	↑
Respiratory acidosis	COPD	hypoventilation	↓	↑	↑
Respiratory alkalosis	Pregnancy	hyperventilation	↑	↓	↓

- Lung problem= Respiratory acidosis/alkalosis
- Renal problem= Metabolic acidosis/alkalosis

1- Respiratory Acidosis:

Causes: (Objective 4)

Primary mechanism→ hypoventilation

Other mechanisms→ CNS, Peripheral nerve, neuromuscular junction, chest wall, bronchial tree abnormalities.

Acute causes:

1- Airway obstruction
2- pneumothorax
3- trauma
4- sever pneumonia
5- residual neuromuscular blockade
6- CNS disease 'head trauma'

2- respiratory alkalosis:

Causes: (Objective4)

1- Pain
2- Pulmonary emboli
3- Sepsis
4- Fever
5- Thyrotoxicosis
6- Pregnancy
7- Overaggressive mechanical ventilation
8- Hepatic failure
9- Anxiety
10- Hypoxemia
11- Restrictive lung disease
12- Sever congestive heart failure
13- Drugs

- Anxiety (panic attack) → *Treatment:* Breath into paper bag to recycle the exhaled CO_2
- Pulmonary embolism, pneumonia and asthma
- Pregnancy
- Pain
- Sepsis
- Liver cirrhosis and hepatic failure
- Drugs (salicylates toxicity)
- Restrictive lung disease
- Overaggressive mechanical ventilation

Treatment: Treat the underlying cause No need for treatment in case of pregnancy

3- Metabolic Acidosis:

Anion gap = Na – (Cl + HCO3) OR (Na + K) – (Cl + HCO3)

Anion gap= Cation – Anion

Increase Anion Gap could be due to:

1- alcohol (Ethanol, methanol) intoxication	2- Uraemia (renal failure)	3- Lactic acidosis
4- Paraldehyde and other drugs	5- Aspirin	6- ketones

Etiologies of AG Metabolic Acidosis (Objective4)

A- Ketoacidosis	1- Alcohol 2- starvation 3- DM
B- **Lactic acidosis**	1- Type A: impairment in tissue oxygenation 2- Type B: no impairment in tissue oxygenation
C- Renal failure	Accumulation of organic anions such as phosphates, sulfates
D- GI losses of HCO_3	**Diarrhea, intestinal or pancreatic fistulas or drainage**
E- RTA	

Effects of acidosis and alkalosis

Acidosis → Right shift in oxygen-haemoglobin dissociation curve diminishes the affinity of haemoglobin for oxygen (**increasing tissue oxygen delivery**)

Alkalosis → Left shift in oxygen-haemoglobin dissociation curve increases the affinity of haemoglobin for oxygen (decreasing oxygen delivery to tissues)

- If you have for example a decrease in PH and increase in PCO2 (It's a Respiratory acidosis) you look at HCO3 if it's in the opposite direction that means there is a metabolic alkalosis (trying to compensate, but did not reach normal PH so it's not compensated "yet")
- Ranges of acute/ chronic (3-3.5 /4-5) if it's more that means there is something wrong.
- If the PH is abnormal it's Acute And Uncompensated.

Acute Kidney injury

- AKI: A rapid decline in renal function, with an increase in serum creatinine level.
- Early stages → creatine levels may be normal despite a markedly reduced GFR.
- RIFLE criteria:

	Serum creatinine	GFR	Urine out put
Risk	↑ 1.5-fold	↓ 25%	< 0.5 for 6 hours
Injury	↑ 2 folds	↓ 50%	< 0.5 for 12 hours
Failure	↑ 3 folds	↓ 75%	< 0.5 for 24 hours or anuria for 12 hours
Loss	Complete loss of kidney function for more than 4 weeks		
ESRD	Complete loss of kidney function for more than 3 months		

- most common findings are weight gain and edema.
- Azotemia (elevated BUN and Cr).
- Good prognosis 80% of patients survive and live completely normal, MOST common cause of DEATH is infection 75% of cases.
- Types of AKI:

Pre-Renal → ↓Renal blood flow

- Etiology:

1- Hypovolemia 2- CHF 3- Renal arterial obstruction
4- Cirrhosis 5- hepatorenal syndrome 6- in patient with decrease renal perfusion
- signs of volume depletion like dry mouth, hypotension, tachycardia, decrease tissue turgor, oliguria/ anuria.

- Lab findings:

1- oliguria "always"	2- ↑ BUN to serum Cr Ratio.	3- ↑ urine osmolarity (> 500 mOsm /kg H2O)
	4- ↓ urine Na (<20 mEq/L)	5- Bland urine sedimentation "No blood"

- Pre-Renal vs Intrinsic:

	Pre-Renal	Intrinsic Renal
Urinalysis	Hyaline casts	Abnormal
BUN/Cr Ratio	> 20:1	< 20:1
FENa (Fractional Excretion of Sodium)	< 1%	> 2% -3%
Urine Osmolarity	> 500mOsm	250-300 mOsm
Urine Na	< 20	> 40

Normal BUN-Creatinine ratio is (10:1-20:1)

Intrinsic → Damage to renal parenchyma

- Causes:

1- Tubular disease (ATN) → ischemia	2- Glomerular disease (acute glomerulonephritis GN)
3- Vascular disease e.g. Renal occlusion.	4- interstitial disease e.g. allergic interstitial nephritis.

- One of the differences between ATN and prerenal:

1- prerenal → Urine sedimentation is scant (lacking)

2- ATN → Full brownish pigment, granular casts with epithelial casts.

- Lab findings:

1- ↓ BUN- Cr Ratio	2- ↑ Urine Na
3- ↓ Urine Osmolarity	4- ↓ urine plasma to Cr ratio

- AKI Diagnosis is usually made by finding elevated BUN and Cr levels. The patient is asymptomatic.

Post-Renal → Urinary tract obstruction of any part with an intact kidney.

- Renal function is restored if the obstruction is relieved before renal damage.
- Postrenal obstruction can lead to ATN.
- Causes:

1- Urethral obstruction secondary to prostate (BPH)	2- Obstruction of a solitary kidney	3- Nephrolithiasis *Kidney stone*
4- Obstructing neoplasm	5- Retroperitoneal fibrosis	6- Urethral obstruction.

- Diagnosis:

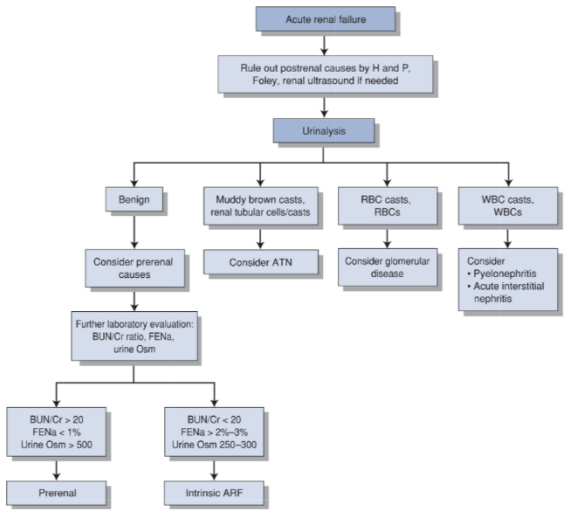


TABLE 7-2 Urinalysis Findings in Renal Failure

Cause	Urine Sediment	Protein	Blood
Prerenal	Benign sediment—few hyaline casts	Negative	Negative
Intrarenal			
Acute tubular necrosis	"Muddy brown" casts, renal tubular cells/ casts, granular casts	Trace	Negative
Acute glomerulonephritis	Dysmorphic RBCs, RBCs with casts, WBCs with casts, fatty casts	4+	3+
Acute interstitial nephritis	RBCs, WBCs, WBCs with casts, eosinophils	1+	2+
Postrenal	Benign; may or may not see RBCs, WBCs	Negative	Negative

- Complications:

1- ECF volume expiation → pulmonary edema Rx: furosemide "diuretic"

2- metabolic: hyperkalaemia, hyperphosphatemia, hyperuricemia, metabolic acidosis, Hypocalcaemia, hyponatremia.

3- uraemia

4- infection

- Treatment:

General	Prerenal	Intrinsic	Postrenal
1- avoid nephrotoxic meds 2- avoid meds that ↓ renal perfusion (NSAIDs) 3- adjust medication dosage 4- Order dialysis if symptomatic uraemia, intractable acidemia, hyperkalaemia, or volume overload develop 5- BP and CO should be in normal ranges 6- fluids and electrolytes.	1- Treat the underlying disorder. 2- Give NS to maintain euvolemia and restore blood pressure—do not give to patients with edema or ascites. May be necessary to stop antihypertensive medications. 3- Eliminate any offending agents (ACE inhibitors, NSAIDs). 4- If patient is unstable, Swan–Ganz monitoring for accurate assessment of intravascular volume.	1- Once ATN develops, therapy is supportive. Eliminate the cause/offending agent. 2- If oliguric, a trial of furosemide may help to increase urine flow. This improves fluid balance.	a bladder catheter may be inserted to decompress the urinary tract. Consider urology consultation.

Chronic Kidney Disease

CKD: either ↓ Kidney function (GFR <60) or Kidney damage for At least 3 months, regardless of the cause.

Causes:

1- DM
2- HTP
3- Chronic GN
4- interstitial nephritis
5- any of AKI causes may lead to CKD

Clinical features:

System	Symptoms/ disease	System	Symptoms / disease
A-Cardiovascular	1- HTN 2- CHF 3- Pericarditis	B- GI	1- Nausea 2- Vomiting 3- Loss of apatite
C- Neurologic	1- lethargy 2- confusion 3- weakness	D- Hematologic	1- Normocytic normochromic anemia 2- bleeding secondary to platelet dysfunction.
E- Endocrine/ metabolic	1- Ca-Phosphorus disturbance 2- Sexual/ reproductive symptoms 3- Pruritus: or itch is defined as an unpleasant sensation of the skin that provokes the urge to scratch	F- Fluid electrolyte problems:	

Treatment:

1. Diet
    a. Low protein—to 0.7 to 0.8 g/kg body weight per day
    b. Use a low-salt diet if HTN, CHF, or oliguria are present
    c. Restrict potassium, phosphate, and magnesium intake
2. ACE inhibitors—dilate efferent arteriole of glomerulus
    a. If used early on, they reduce the risk of progression to ESRD because they slow the progression of proteinuria
    b. Use with great caution because they can cause hyperkalaemia
3. BP control
    a. Strict control decreases the rate of disease progression
    b. ACE inhibitors are the preferred agents. Multiple drugs, including diuretics, may be required
4. Glycaemic control (if the patient is diabetic) prevents worsening of proteinuria
5. Smoking cessation
6. Correction of electrolyte abnormalities
    a. Correct hyperphosphatemia with calcium citrate (a phosphate binder)
    b. Patients with chronic renal disease are generally treated with long-term oral calcium and vitamin D in an effort to prevent secondary hyperparathyroidism and uremic osteodystrophy
    c. Acidosis—treat the underlying cause (renal failure). Patients may require oral bicarbonate replacement
7. Anemia—treat with erythropoietin
8. Pulmonary edema—arrange for dialysis if the condition is unresponsive to diuresis
9. Pruritus—try capsaicin cream or cholestyramine and UV light
10. Dialysis (See indications in the Dialysis section.)
11. Transplantation is the only cure

Table 7-3 Prognostic Factors in AKI

Severity of renal failure	Magnitude of increase in Cr Presence of oliguria Fractional excretion of sodium Requirement for dialysis Duration of severe renal failure Marked abnormalities on urinalysis
Underlying health of patient	Age Presence, severity, and reversibility of underlying disease
Clinical circumstances	Cause of renal failure Severity and reversibility of acute process(es) Number and type of other failed organ systems Development of sepsis and other complications

Adapted from Schrier RW, ed. *Diseases of the Kidney and Urinary Tract*. Vol II. 7th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2001:1128, Table 41-14.

Glomerular Disease

- Normal urine should **NOT** Have:

1- Protein 2- Fat 3- Sugar
4- RBC 5- HEME 6- Cell cast

	Nephrotic	Nephritic
Site affected	Podocyte	Mesangial, Endothelium, GBM
Urine finding	Proteinuria	Hematuria
Microscopic finding	Foot processes are lost, but the body is intact	---
Lab finding	1- Hypoalbuminemia <30 2- Heavy protein urea >3.5 3- Hyperlipidemia 4- Peripheral edema	---
Complications	1- infection, sepsis. 2- AKI 3- Thrombosis 4- ESRD	---
Urine analysis	1- Proteinuria 2- NO RBC 3- NO RBC cast 4- Fat 5- No WBCs	1- RBC 2-RBC cast 3- Dysmorphic RBC 4- Protein
Clinical presentation	1- edema 2- Fatigue 3- Frothy urine 4- Anorexia 5- Nausea, vomiting 6- SOB 7- Abdominal pain 8- Weight gain due to fluid retention	1- AKI 2- ↓ urine out-put 3- edema 4- High BP
Glomerular diseases that present as	(FSGS)→ Focal segmental Glomerulosclerosis (MCD)→ Minimal change disease (MN) → membranous nephropathy	1- IgA Nephropathy 2- Post streptococcal GN 3- Membranoproliferative GN 4- ANCA Vasculitis 5- Anti-GBM (Goodpasture disease) 6- Lupus Nephritis

- You need a renal biopsy to diagnose any suspected primary glomerular disease.
- Primary glomerular disease is mostly caused by immune system dysfunction.

❖ **Nephrotic syndrome:**

	FSGS	MCD	MN
Primary	**Sudden onset heavy protein urea**	**Idiopathic** **(Main cause of nephrotic in children)**	**Idiopathic** **Most common cause of Nephrotic in Adults.**
Microscope	**All glomeruli whether it's affected or not foot processes are going to be diffused.**	**Light → normal** **Electron→ diffuse effacement of the epithelial cells foot processes.**	----------
Diagnosis	------- Biopsy	**Biopsy**	---------- Biopsy
Rx of primary	**First line: Corticosteroids** **Second line: cyclosporine or tacrolimus (Immuno…)**	**First line: corticosteroids** **Second line: cyclophosphamide, cyclosporine.**	**Corticosteroids + cyclophosphamide or cyclosporine.** **May be Rituximab**
Secondary	**Proteinuria is less heavy**	----	---------
Causes of secondary	**1- obesity** **2- nephron loss** **3- Reflux nephropathy** **4- healing GN** **5- Anabolic steroids** **6- Drugs: interferon, pamidronate, heroin.** **7- Infection HIV** **8- sever preeclampsia**	**1- Drugs: NSAIDs, Lithium, sulfasalazine, pamidronate** **2 Neoplasms: Hodgkin/ non, leukemia.** **3- Infection (TB, syphilis)** **4- Allergy.**	**1- SLE** **2- Drugs: Penicillamine** **3- Infections: hep B, C, Syphilis** **4- Malignancy: solid tumors prostate, lung, or GI tract**
Secondary Rx	**Not treated with Immono… treat primary cause**	---------	**Mainly treat what caused MN**

- The most important difference between FSGS and MCD is that only FSGS has glomerular sclerosis.
- Important secondary causes of nephrotic syndrome in adults:

1- DM **2- Amyloidosis** 3- IgA Nephropathy 4- MPGN

❖ Nephritic syndrome:

Go read last 10 slides

Non-traumatic	Info	Symptoms	Diagnosis	Management	History	Differential
Hematuria	- **Types**: Microscopic and gross - **Causes**: pre-renal: s, s, h, anti… Renal: T, TB, S, GN, RS. Post renal: T (B, U, U), Urethral (S, P), B, PP.	- Risk factor for TCC: (5) smoker, 40<, LUTS irritation, pelvis radiation, Bilharzias	Full work up CT Urography (Gold standard)	3-way catheter	Age (TCC only adult)	If it's painful suspect: (stones, UTI, Trauma, Renal vein Thrombosis)
Renal Colic	**indications of surgery**: 1- to relieve obstruction and remove stone 2- Impairment of renal function 3- unrelieved obstruction for >4 weeks. 4- Pain no response to analgesia 5- associated with fever 6- personal reasons.	- Sudden onset sever pain (one of the worst) - Intermittent - relieved by analgesia - nothing aggravates it - Associated with N/V - Location of pain may change 1- upper ureteric calculi → costovertebral angle / flank pain. 2- mid ureteric →pain radiating from the loin to the groin. 3- lower ureteric calculi → cause pain radiating to the testicle or labia majora 3- vesical calculus (bladder) → suprapubic pain. 4- renal stone → could be silent even if it's large enough to fill the pelvis 5- seminal vesicle stone → extremely rare cause haematospermia	- Physical Exam: patient want to move around to relief the pain *In appendicitis Can't move Fever indicate infection (needs extra hydration) - Investigation: 1- Pregnancy test 2- midstream urine analysis 3- Urea electro lights. CT without contrast MRI→ very accurate for stones in the ureter, for pregnant ladies.	- NSAIDs, Opiates analgesics - Hyper hyperhydration - wait 95% of stones (5mm) will go out on their own. **Types of surgery:** **A- temporary relieve:** 1- JJ stent 2- percutaneous nephrostomy tube. **B- Definitive treatment:** 1- extracorporeal shock waves 2- percutaneous nephrolithotomy. 3- ureteroscopy (laser) 4- Laparoscopic extraction 5- open surgery		1- Radiculitis 2- Pneumonia and MI 3- Abdomen (rupture of aortic aneurism) 4- ectopic pregnancy 5- testicular torsion

Non-traumatic	Info	Symptoms	Diagnosis	Management	History	Differential
Urinary Retention	Acute urinary retention Cause: ♂: 1- Benign prostatic hyperplasia 2- Carcinoma of prostate 3- Abscess in the prostate 4- Urethral stricture ♀: 1-pelvic organ prolapse. 2- Urethral stricture. 3- post surgery stress. 4- pelvic masses. Chronic: Develop slowly and the bladder is stretched	- Could be associated with ↓ renal function OR renal failure - upper track dilation - hydronephrosis - Pain is NOT a feature * present with 1- urinary dribbling 2- overflow incontinence 3- palpable bladder with NO pain	The cause is usually neurological	1- renal support 2- treat electro light imbalance. 3- SLOW rate drainage of bladder to avoid decompression (Hematuria) 4- treatment of underlying cause. - 3-way catheter OR Foleys catheter. - Give analgesia to prevent spasm. - suprapubic catheter (used when the urethra is inaccessible) * definitive Treatment is treatment of the underlying cause.		
Acute scrotum	Also known as testicular pain or scrotal pain. Torsion of the cord. Epididymo-orchitis. Could be caused by Brucella.	- Acute onset, sharp and sever - Referred to the ipsilateral lower quadrant of the abdomen - Children may present with abdominal pain little or No pain Gradual not sudden, get sever toward the end. Dysuria, fever. Common with patients with STD (gonorrhoea) or UTI	* So, any child with abdominal pain should have a genital examination. *Crimastic reflex: - Absent in testicular torsion - Present in epididymo-orchitis *scrotal support: - painful in testicular torsion - relieve pain in epididymo-orchitis. * tests: 1- sound doppler (high false -ve) 2- Color doppler US (gold standard) 3- Radionuclide imaging (assess blood flow and helpful with hematoma and hydrocele) 4- Surgical exploration Physical: swollen hemi-scrotum Epidydimal tenderness Elevated WBC and bacteriuria	True surgical emergency of highest order. Could cause irreversible ischemia of as soon as 4hours. - usually patient is sent to the Operating Room immediately without investigation because this is an emergency. - Bed rest 1-3 days - scrotal elevation - antibiotics if UTI is - suspected or documented * avoid urethral instruments to reduce risk of more infection.	* Common in teenagers, possible in neonates, unlikely above 25. *Majority have a history of prior episodes. * When there is a high possibility of testicular torsion in the history and exam take the patient to OR without waiting.	1- epididymitis (most common cause) Or epididymo-orchitis. 2- torsion of spermatic cord (most serious complication)

Non-traumatic	Info	Symptoms	Diagnosis	Management	History	Differential
Priapism	Persistent erection for more than 4 hours. Types: 1- ischemic. (veno-occlusive or low flow) 2-Non-ischemic (arterial or high flow) - The persistence of priapism will cause clotting which leads to healing by fibrosis in the corpora and this will damage it and the patent will damage it and the patient will lose the ability of erection. - In general, the causes are either 1- primary:(idiopathic) 30-50% 2- secondary: drug trauma, neurological, hematological,	1- painful. - most common. - Pathophysiology: thrombosis of venous system causing congestion and engorgement which leads to the erection. Causes: 1- sickle cell disease. 2- malignancy that infiltrated the corpora cavernosa 3- drugs like prostaglandin injection. 1- painless. Pathophysiology: perineal trauma will cause arteriovenous fistula which fills the corpora.	History: Erection>4h, painful/painless, previous history. Examination: - Tender→ in low/high blood flow - Corpora is rigid and gland is flaccid. - evidence of malignancy. - perirectal exam Investigation: - CBC - haemoglobin electrophoresis for SCD. - Urinalysis for toxicology. - Blood gases taken from either corpora. - color doppler in cavernous arteries: A- ischemic → if flow is low or non-existent. B- Non-ischemic: inflow is normal to high	Depend on the type. 1- conservative: Ask the patient to clime the stairs so the vein channels open. 2- Drugs: bicarbonate, high O2 and cold enema. 3- surgical treatment: aspiration and saline wash of corpora.	-	-

Variable	Low flow (ischemia/occlusive)	High-flow (non-ischemic/Fistula)
Blood color	Dark blood	Bright red blood (similar arterial blood at room temperature)
Ph	<7.25 (acidosis)	= 7.4 normal
pO2	<30 mmHg (hypoxia)	>90 mmHg (normal)
Pco2	>60 mmHg (hypercapnia)	<40 mmHg (normal)

GU-Oncology

Tumor →	Renal	Bladder	Prostate	Testicular
Types	1- Renal cell carcinoma (Adenocarcinoma) → arise from proximal tubules → sub type clear cell carcinoma 2- Oncocytoma commonest benign tumor 3- papillary cell carcinoma runs in families	1- Transitional cell carcinoma 90% 2- squamous carcinoma 5% 3- A denocarcinoma 2%	Adenocarcinoma → peripheral zone of the prostate	
Prognosis	- Benign tumors are RARE. - All neoplasms should be considered malignant - patient will have reoccurrence even after removal of tumor. Early stage → 5years Late stage → 3-6 months	1- **TCC→** **- 80%** superficial without muscle invasion **good prognosis** above muscle layer **Higher reoccurrence rate** **- 20%** → high grade and muscle invasion **poor prognosis** **2- Squamous carcinoma: worst prognosis**	- More men die with prostate cancer Than from prostate cancer.	- In those with disease localized to the testis → 95% 5year survival Risk factors: 1- cryptorchidism. (absence of one testis "birth defect") 2- testicular maldescent. 3- Klinefelter's syndrome 4- testicular torsion.
Metastasis / Grading	A- extend→ IVC, Renal vein, heart. (only if it's localized in the heart surgery) B- blood born→ canon ball pulmonary metastasis. C- Tumor thrombosis → could block IVC. *Grading system for kidney cancers is called **Fuhrman** system.	Tis → in situ disease Ta → epithelium only T1→ Lamina propria invasion T2→superficial muscle invasion T3a→Deep muscle invasion T3b → peri vesical fat invasion T4 → prostate or contiguous muscle invasion G1: well differentiated G2; moderately differentiated G3: poorly differentiated	# Adenocarcinoma: - spread into peri-neural spaces, bladder neck, pelvic wall and rectum. - Lymph spread is common - hematogenous spread occurs to axial skeleton # malignant prostate tumors arise in the peripheral zone. # Benign prostatic hyperplasia arises in the transitional zone. - tumors are classified by **Gleeson** classification	**Staging:** Stage 1 → disease confined to testis Stage 2 → Abdominal Lymphadenopathy A → < 2cm B → 2-5 cm C → >5 cm Stage 3 → supra-diaphragmatic disease.
Symptoms	- incidental finding 1- gross hematuria 2- loin pain 3- palpable mass 4- pyrexia of unknown origin 5- HPT 6- polycythaemia 7- Hypercalcemia + Stauffer's syndrome in paraneoplastic syndrome.	80% with painless hematuria (Terminal hematuria) Also present with treatment resistant infection Or: bladder irritability and sterile pyuria.	- Incidental finding. - could present with bone pain, cord compression, leucoerythroblastic anemia. - renal failure can occur due to bilateral ureteric obstruction.	- Commonest presentation is an ipsilateral painless testicular swelling.
Investigation	- US: to confirm - CT: staging - Echocardiogram if clot in IVC extends above the diaphragm	*Painless hematuria is considered cancer till proven otherwise. Investigation of painless hematuria: 1- urinalysis 2- KUB (X-ray) 3- US (bladder and kidney) 4- Cystoscopy (Must) 5- Urine Cytology 6- IVU (consider)	Screening in north America: 1-PSA 2- Perirectal exam. If any of them is positive indication for biopsy. - Rectal exam → confirm Transrectal biopsy - MRI → staging - Bone scanning → for metastasis - Unlikely to be abnormal if asymptomatic and PSA<10	**- Testicular US →** confirmation **- Inguinal orchidectomy →** pathological diagnosis. **- Thoracoabdominal CT:** for staging. **#Tumor markers:** **- α-FP →** produced by yolk sac elements **- α-FP→** NOT by seminoma **- β-hCG →** Trophoblastic elements, elevated in teratoma and seminoma **- LDH**
Treatment	1- Unless extensive metastasis Rx will involve surgery (partial or complete) + adrenal, perinephric fat should be removed. # Laparoscopic nephrectomy is the Gold standard 2- Lymph node removal is not effective only for staging 3- immunotherapy #- Never use Radiotherapy or Chemotherapy unless symptomatic bone metastasis for Pain	- T2 and above need to remove the hall bladder - Carcinoma in situ consider immunotherapy if fails may need radical cystectomy **1- superficial TCC**: transurethral resection + cystoscopic follow-up + consider Chemo if high risk of invasion (multiple tumors, Big tumors, **carcinoma in situ**) + consider immune if M.bovis, **2- Invasive TCC:** - Radical cystectomy 5% death in OR - Urinary diversion: A- ileal conduit (incontinent) B- neo-bladder (continent) C- content cutaneous reservoir - Radiotherapy is NO good	Treatment depend on tumors stage and the patients age: **1- Local disease:** - observation. - Radical Radiotherapy. - Radical prostatectomy. **2- Locally advanced disease:** - Radical radiotherapy. - hormonal therapy. **3- Metastatic disease:** - Hormonal therapy. Hormonal therapy: Cense 80-90% of prostate cancers are androgen dependent for their growth, the therapy involves androgen depletion. Can be achieved by: 1- orchidectomy 2- LHRH agonist e.g. goseraline 3- complete androgen blockade	**#Seminoma:** Radiosensitive **Stage 1,2 →** inguinal orchidectomy + Radiotherapy to ipsilateral abdominal and pelvic nodes (dog led) + surveillance. **Stage 2c→** and above are treated with chemotherapy # Non-seminoma: Not-Radiosensitive Stage 1→ orchidectomy + RPLVD + chemotherapy. **Chemotherapy→ BEP** **B**leomycin (pulmonary fibrosis) * **E**topside **P**isplatin

			4- Anti androgens (e.g. cyproterone acetate, flutamide, bicutamide)	
Extra	1- von Hipple-lindau syndrome (multiple cancers RCC could be one of them) 2- paraneoplastic syndrome (secretes ADH and EPO) 3- Stauffer's syndrome (part of paraneoplastic; non-metastatic hepatic dysfunction) * no hepatic metastasis= no jaundice	TCC causes: 1- smoking 2- schistosoma 3- factory 4- analgesics: phenacetin 5- pelvic irritation for carcinoma of the cervix Radical cystectomy involves the removal of 1- bladder 2- prostate 3- distal ureter 4- Lymph node + in females: 1- uterus 2- cervix 3- anterior vaginal wall	PSA: prostate specific antigen: - 4ng/ml is the upper limit of normal - more than 10 is suggestive of prostatic carcinoma. - can be significantly raised in BPH - Useful in monitoring response to treatment Rare before 50	Classifications: 1- seminoma (50%) → radiosensitive 2- Non-seminoma (50%)→ radio-resistant - Teratomas. - Yolk sac tumor. - Embryonal. - Mixed germ cell tumor. Radical orchidectomy is done through the groin. Peak age for teratoma → 25 Peak age for seminoma → 35

Summary (urology + Atherosclerosis)

	Symptoms	Modality of choice	Treatment
Hematuria	-	CTU	3-way catheter
Renal colic	-	CT	Temporary relieve (2) definitive treatment (5) "3 radiations 2 surgeries"
Acute scrotum	-	Color doppler	OR
Renal trauma	-	CT + Contrast	Conservative unless there is An expanding/pulsatile hematoma, tachycardia, hypotension.
Ureteral injury	-	Intra-operative To confirm: water soluble solution then urethrogram (x-ray)	Move the kidney or adjacent structures to improve the situation. If the patient after an injury passes clean urine = bladder and urethra are intact. If there is blood at the external meatus urethral injury is suspected.
Bladder injury	-	Intra-operative To confirm: water soluble catheter then x-ray.	Drain then start repairing
Urethral injury	-	Retrograde Urethrography	No catheter, mostly conservative relay on waiting or diversion of urine.
PAD	1- intermitted claudication 2- pain at rest	Angiogram	1- risk factor modification (7) 2- improve limb circulation (1) 3- major/ minor amputation (2/2)
Carotid AD	1- (TIA) less than 24h 2- Stroke 3- Asymptomatic		1- risk factor modification (same as PAD) 2-impove brain circulation Carotid endarterectomy.
ALI	6Ps	- If complete ischemia→ OR. - if incomplete do an imaging test.	ABC, IV heparin, thromboembolectomy. Ca^{+} Glutamate, Bicarbonate, Lots of fluids. (any patient with ischemia) # depending on the cause 1- Embolus→ if it's an elderly (conservative) brachial If he is young it's better to intervein brachial If the legs are affected intervention is IM 2- Thrombus → (usually due to sepsis) initial treatment should be with drugs, if they don't work go to surgery.
Renal Adenoma (RCC)	1- pain 2- hematuria 3- mass	- US - Staging: CT + contrast.	Never use Chemo-radio therapy unless bone metastasis for pain Removing the kidney or a part of it.
Acute pyelonephritis		Urine culture Imaging sometimes needed (CT, US)	Mainly to treat the infection
Chronic pyelonephritis		CT+ contrast	Treatments + drainage if they don't workout surgery is indicated
Bladder tumors	80% present with painless hematuria (should be considered tumor until proven otherwise)	Biopsies are taken from the area or any other suspected areas.	- Transurethral resection and the detrusor muscle. - Chemotherapy is useful. - Cystectomy if there is reoccurrence or not responding to treatment.
BPH	Usually affects >40 years 1- frequency 2- nocturia 3- urgency 4- dysuria 5- poor stream.	1- DRE ← If any one is positive indicate biopsy 2- PSA ← 3- biopsy 4- US	1- minimal symptoms→ watchful waiting 2- intermediate → Medical management: α-blockers 5α reductase inhibitor

Priapism	1-Self-injected drug 2-Leukemia 3- disorder of coagulation 4 -renal dialysis 5- sickle cell	-	Injection intra-cavernosal vasoconstrictor (phenylephrine) especially in self-injected cases.
Testicular torsion	1- Sudden onset 2- teenager 3- testicular swelling 4- history of minor trauma. 5- previous episodes.	cremasteric reflex scrotal support	Surgical emergency
Epididymo-orchitis	1- Both testis 2-acute inflammatory action 3- discharge	testis alone is a feature of viral (mumps) if both testis and epididymis is a feature of bacterial spread either from infected urine or gonococcal urethritis. Discharge should be cultured	Antibiotics depending on the organism.

Paediatric inguinal and scrotal conditions

- common groin condition in infants and children:

1- Inguinal hernia → is a protrusion of abdominal-cavity contents through the inguinal canal. into the scrotum (male) or via the canal of Nuck to the labium (female)

Risk Factors:

Undescended testis.	ventriculo-peritoneal shunt (VP shunt).
prematurity.	Ascites (any conditions causes an increase intra-abdominal pressure).
connective tissue disorders.	peritoneal dialysis(PD).

Findings:

1- Inguinal Pain is rare unless hernia gets complicated

2- Provocative maneuver such as standing, coughing, laughing or jumping are required to elicit

Treatment:

1- Uncomplicated: IH will not resolve spontaneously and surgery is only the treatment. Open inguinal herniotomy (more common approach)

2- complicated: The presence of peritonitis or septic shock is an absolute contraindication to attempted reduction.

Intravenous access and rehydration.

Monitored conscious sedation.

Firm and continuous pressure is applied around the incarceration.

Successful reduction is usually confirmed by sudden pop of contents back to abdominal cavity.

Over 90-95% of incarcerated IH can be successfully reduced.

Once hernia is reduced, a delay of 24-48h is allowed before herniotomy (resolution of edema and inflammation)

Urgent operation (Herniotomy) is necessary if reduction fails.

2- Congenital Hydrocele → is a type of swelling in the scrotum that occurs when fluid collects in the thin sheath surrounding a testicle

Clinical presentation:

1- Painless scrotal or groin swelling, but mostly scrotal.

2- Increase in size following viral infection.

3- On examination, tense, overlying skin is often has a blue tinge. Not reducable, transilluminte , difficult to palpate the testis separately.

Management:

1- Expectant management(observation) in the first two years of age.

2- By the age of 2 years 90% of hydoceles will have resolved.

3- Surgery (hydrocelectomy /high ligation of PPV) is indicated if the hydrocele fails to resolve by age of 2 years.

3- Undescended testis (CRYPTORCHDISM): arrest along the normal path. ((occur more on the right side))

Retractile testis: move back and fourth

Ectopic testis: located out-side the normal path of descent

Management: Surgical treatment (orchidopexy) the treatment of choice. The best timing is between 6-12 months of age

4- Acute scrotum: Acute scrotal pain with or without swelling and erythema.

Atherosclerosis

* Risk factors:

A- NON-modifiable: Male, Advanced age, Family history.

B- Modifiable: Major: Smoking, hyperlipidemia, diabetes, hypertension.

Minor: Homocystenemia, obesity, inactivity, hypercoagulable states.

* It's a systemic disease that affects all the body

* It's an inflammation that is caused by the rupture of the wall followed by accumulation of fat, fibrous plaque and calcification of arterial wall.

Peripheral Artery Disease (PAD):

It's a marker for systemic atherosclerosis.

Coexisting diseases coronary artery disease and cerebrovascular disease.

6-fold increase in risk of cardiovascular disease in PAD patients, even in asymptomatic patient.

Symptoms: 1- intermittent claudication pain in legs relieved by rest (like angina).

2- critical limb ischemia pain at rest, tissue loss, gangrene, limb threatening condition.

Diagnosis: 1- ABI (ankle Brachial index)

2- non-invasive: - Arterial duplex (doppler + US) good for anatomical view

- CTA = CT+ Contrast.

- MRA

3- Invasive: - Angiogram (gold standard) very accurate in mapping out the arteries but the duplex is better in assessing dynamic view.

Asymptomatic patients you should scan groups with high risk factors like 50< years, male, family history.

ABI: ankle systolic/Brachial systolic (highest of each)

Ankle you can use either Dorsalis pedis (DP) or Posterior tibial (PT)

0.9 is normal, 0.8 mild, 0.5-0.8 moderate, 0.5> severe, 0.25 very sever.

Treatment of PAD:

1- Risk factor modification: Diet- exercise -anti platelet- HTN- DM- Lipid control- smoking. (7)

2- improve limb circulation:

- Conservative: Exercise-

- Interventional: revascularisation→ angioplasty, surgical bypass.

3- last strategy in treating PAD:

- Major amputation: affects function whole leg amputation

A- primary amputation: we start with amputation

B- secondary amputation: we start with angioplasty or bypass but the patient does not respond.

- Minor amputation: Doesn't affect the function:

- BKA: Below Knee Amputation.

- AKA: Above Knee Amputation.

Carotid Artery Disease (CAD)

Symptoms:

1- (TIA) Transient Ischemic Attack loss of motor or sensory function for less than 24h

2- Stroke

3- Asymptomatic

Treatment:

Goals of treatment:

1- prevention of strokes. 2- prolong survival.

Strategies in treating patients with CAD:

1- Risk factor modification: Diet, antiplatelet, exercise, HTN, DM, Lipid control, smoking.

2- improving brain circulation: revascularization with Carotid Endarterectomy (best method) and standard of care

- angioplasty with or without stenting

Acute Limb Ischemia (ALI)

#Could be caused by:

1- Embolus 2- Thrombosis 3- Trauma 4- Iatrogenic (doctor caused it). 5- Arterial dissection

#Possible sources of an emboli:

Spontaneous 80%: cardiac cause → Arrhythmia, MI, prosthetic valve, endocarditis.

Non-cardiac → Proximal plaque, aneurysm, paradoxical emboli.

Iatrogenic 20%: Angiographic manipulation, surgical manipulation.

commonest site is the Femoral Artery.

#Presentation of Acute limb ischemia:

Sudden poorly localized leg pain

6Ps:

1- Paraesthesia (pins and needles) 2- Pain 3- Poikilothermia (inability to control temperature
4- Pulselessnes 5- Pallor 6- Paralysis

#Investigations: in clinic, angiography if possible.

#Treatment: Golden time is 6h

1- ABC is the most Important step

2- IV heparin

3- Rapid surgical thromboembolectomy:

+/- surgical bypass.

+/- Thrombolytic therapy.

+/- primary amputation.

 # perfusion injury: due to rise in pressure in the compartment which leads to edema and more ischemia → treated with fasciotomy

#you should give a patient with ischemia:

1- Ca+ glutamate → prevent cardiac arrest 2- Bicarbonate → prevent acidosis 3- Lots of fluids