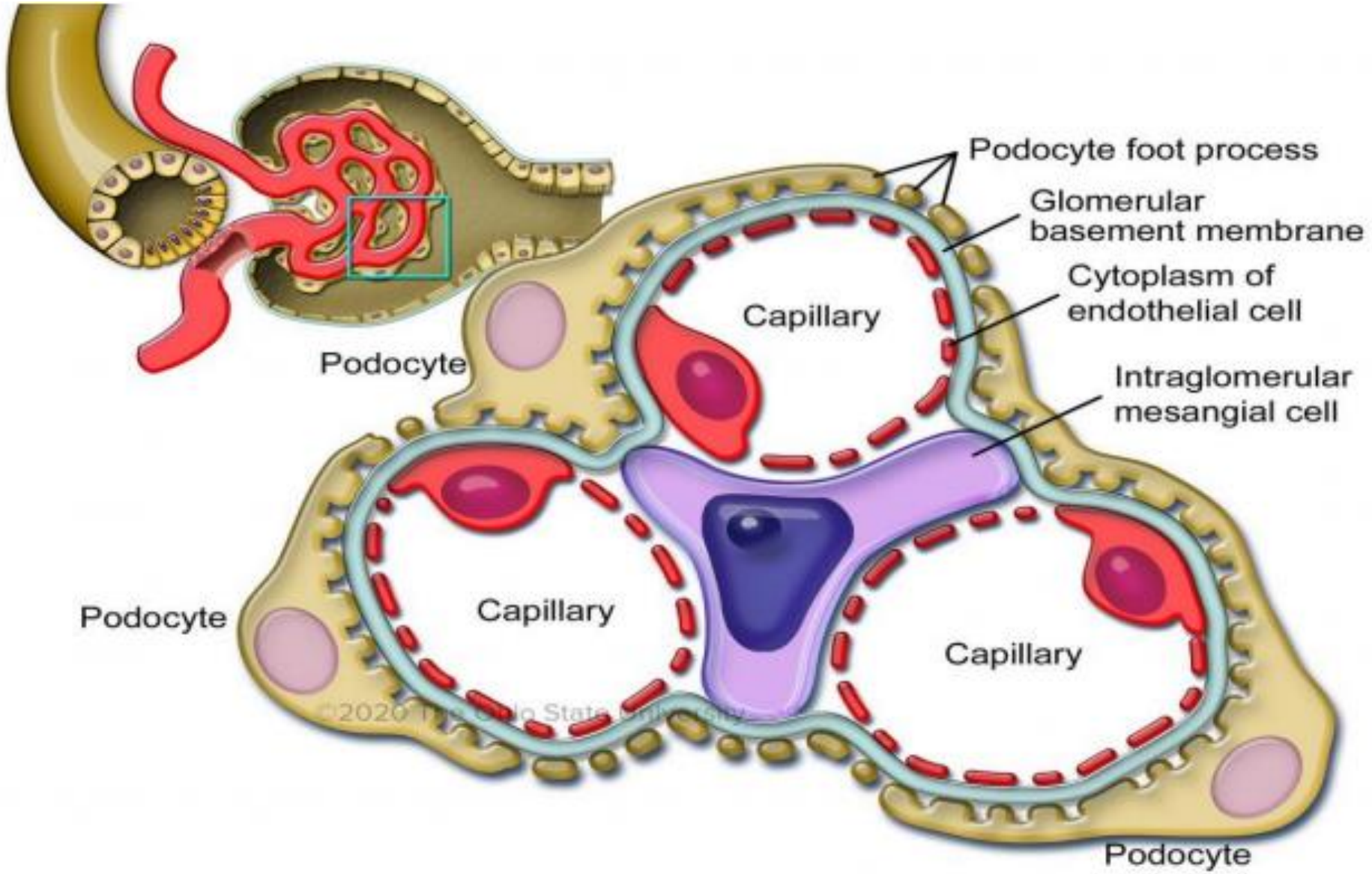
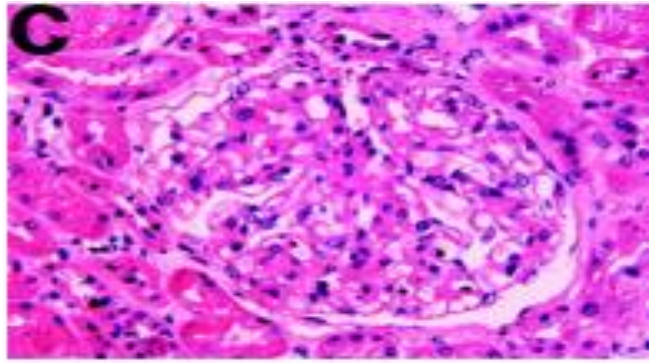
Nephrology Mini-OSCE

QMA Team

Nephrotic & Nephritic, CKD, AKI


Podocyte foot process
Glomerular basement membrane
Cytoplasm of endothelial cell
Intraglomerular mesangial cell
Capillary
Podocyte
Capillary
Podocyte
Capillary
Podocyte

Nephritic



- Glomerular disease characterized by **glomerular inflammation and bleeding**.
- It is usually due to the involvement of **GBM. (Glomerulonephritis)**
- **Characterized by**: 1. Limited proteinuria <3.5 gm/d. 2. Oliguria and azotemia = elevated BUN/SCR. 3. Salt retention with periorbital edema and hypertension. 4. **RBCS cast and dysmorphic RBC'S in urine.The major feature**
- Biopsy reveals hyper-cellular inflamed glomeruli ©.
- It is two main classes either (**Isolated Glomerular Hematuria, or Acute nephritic syndrome**).

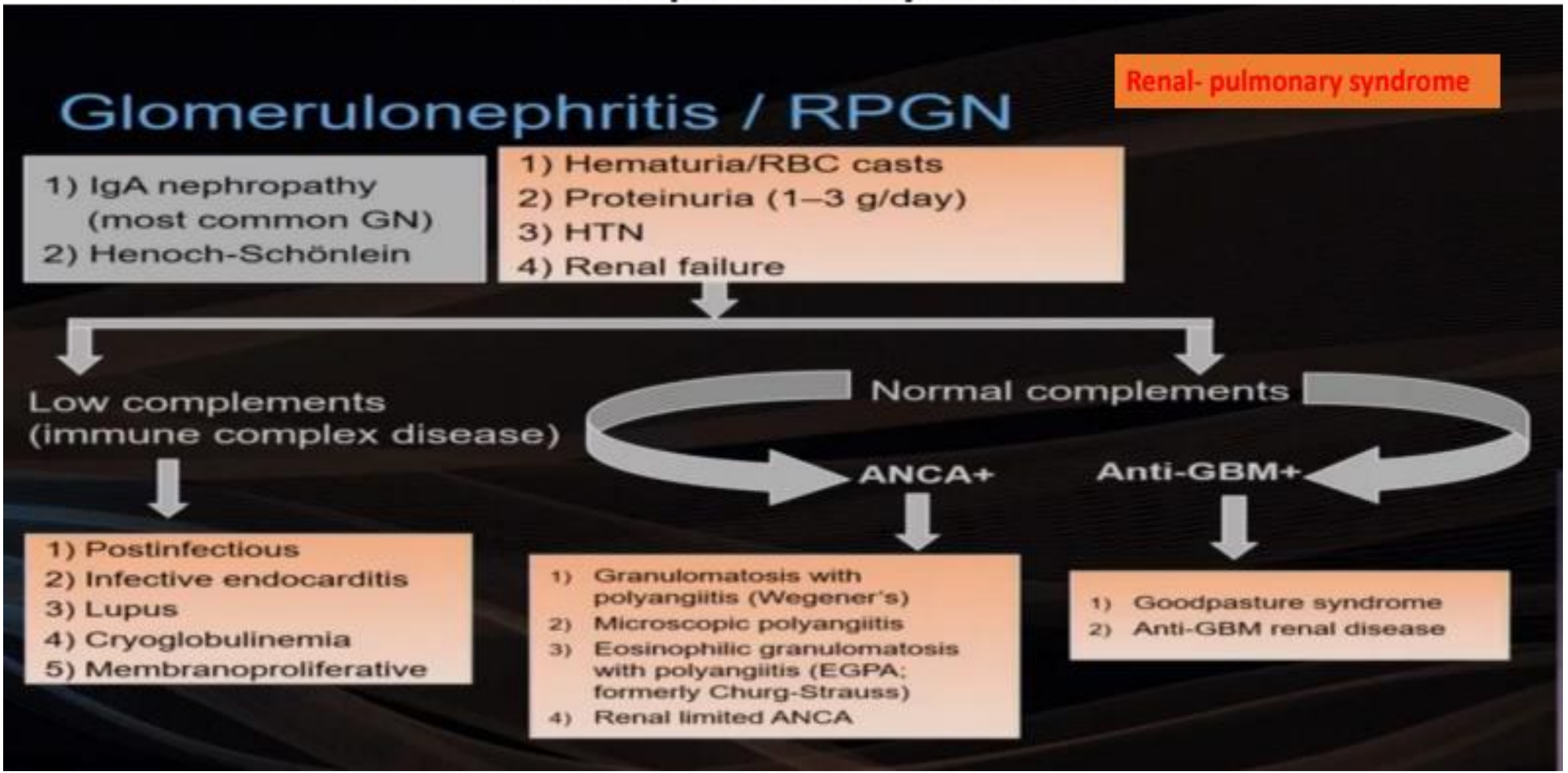
Isolated Glomerular Hematuria

- **IgA nephropathy (BERGER DISEASE)**

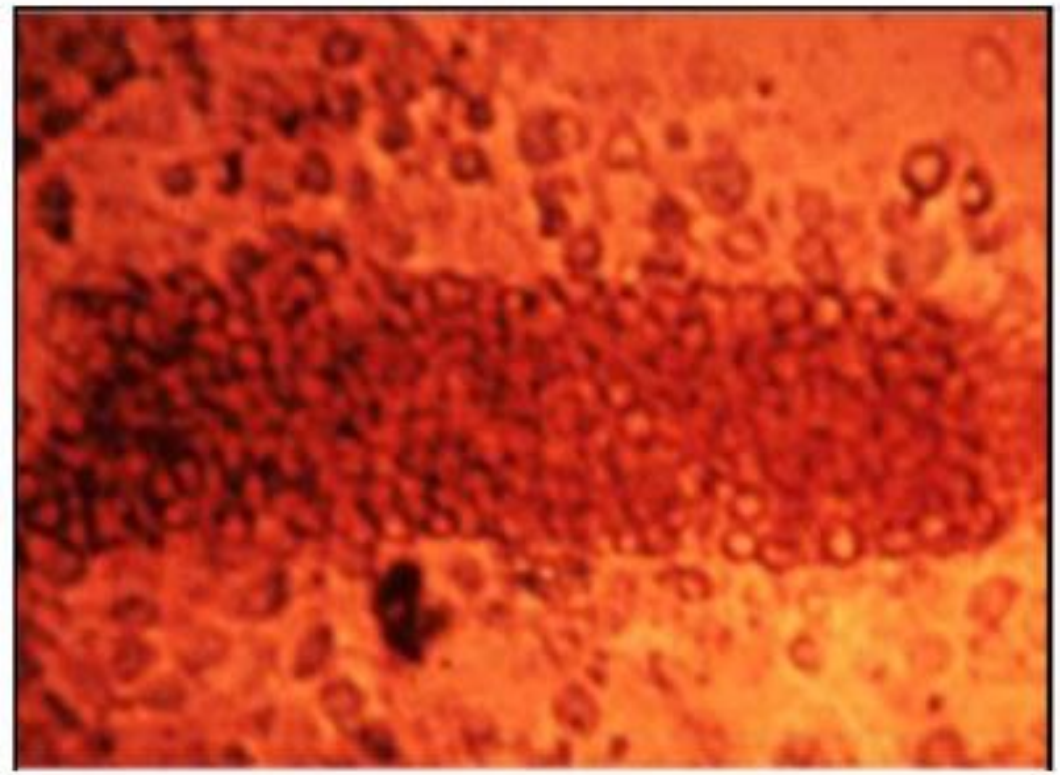
- **Alport syndrome**:

1. X-Linked – defect in type IV collagen a5 chain.
2. (Positive family history).
3. Associated with sensorineural deafness and ocular disturbances.

Acute nephritic syndrome



Q3.A pt presented with red urine. The picture shows a microscopical view of his urine sample.Mention 2 causes for this condition.



This is an RBC cast seen in nephritic syndrome.

Causes are :

1- IgA Nephropathy.

2- SLE.

3- Cryoglobulinemia.

4- Post-Strep infection.

Acute nephritic syndrome

- **IgA nephropathy is the most common primary GN worldwide**.
- Ig A complex deposits in the mesangium of glomeruli.
- **Recurrent hematuria** (Can have sub-nephrotic range of proteinuria).
- Episodes of gross/ microscopic hematuria are **precipitated by flu-like illness 2-3 days**.
- **Diagnosis is made clinically** or in percutaneous biopsy.
- Normal complement levels.
- **HSP:** Kids or young adult with (Palpable purpura, Arthritis/ Arthralgia, Abdominal pain, Renal involvement).

IgA nephropathy

Q11.A man is suffering from haematuria after 2 days of having Streptococcal infection in his throat. What's your Dx?

IgA glomerulonephritis(the

Acute postinfectious GN

- Usually occurs in children – POSTSGN.
- **Occurs after streptococcal pharyngitis or impetigo**. (GABS particularly Nephritogenic type).
- **Presents 7-10 days up to 30 days after infection as hematuria** (Cola-Colored), oliguria, hypertension and periorbital edema.
- It has a **decreased complement level** (especially C3).
- **Increased ASO titer**.

Q1.21 YO presented with SOB, fatigue, dark-colored urine, Hx of "cold" 10 days ago. On P/E: BP 140/90, puffy eyes, mild pitting lower limb edema, lung crepitations.

1-What's your Dx?

Nephritic syndrome (Post-streptococcal GN).

2-Give 2 findings in urine analysis?

Dysmorphic RBCs, RBC casts.

3-What's the most likely agent causing this ?

Group A Beta-Hemolytic strep (Streptococcus pyogenes).because he tell you Hx of cold 10 days ago

Membranoproliferative GN (MPGN)

- Can present with both **nephritic and nephrotic syndromes**.
- Low C3 level is seen in 70% of patients.
- Diagnosed by biopsy.
- Two types:

1. Type 1: Can be **associated with hepatitis B and C** (clue risk factors or elevated liver enzymes).
2. Type 2: Associated with C3 nephritic factor … **low C3**.

- Poor response to steroids and may progress to CKD.

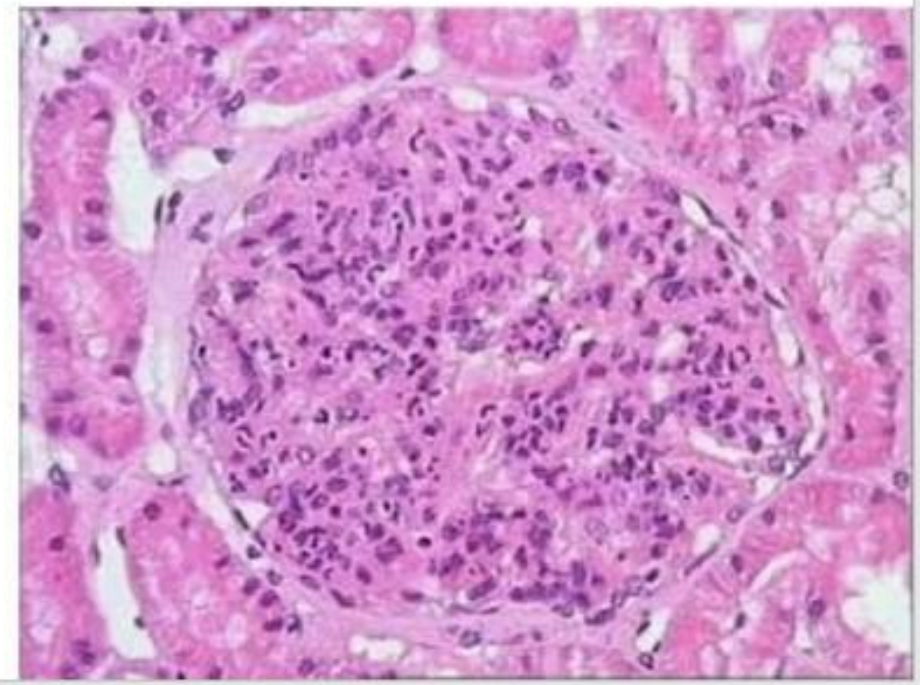
Q1 : Dx?

Diffuse proliferative GN

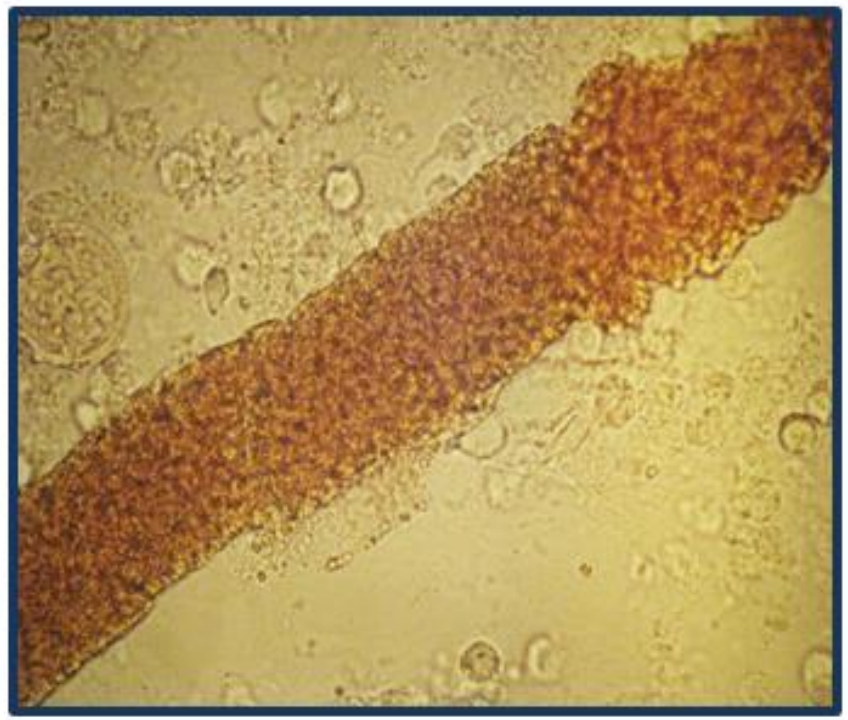
Q2 : mention 2 lines of Tx ?

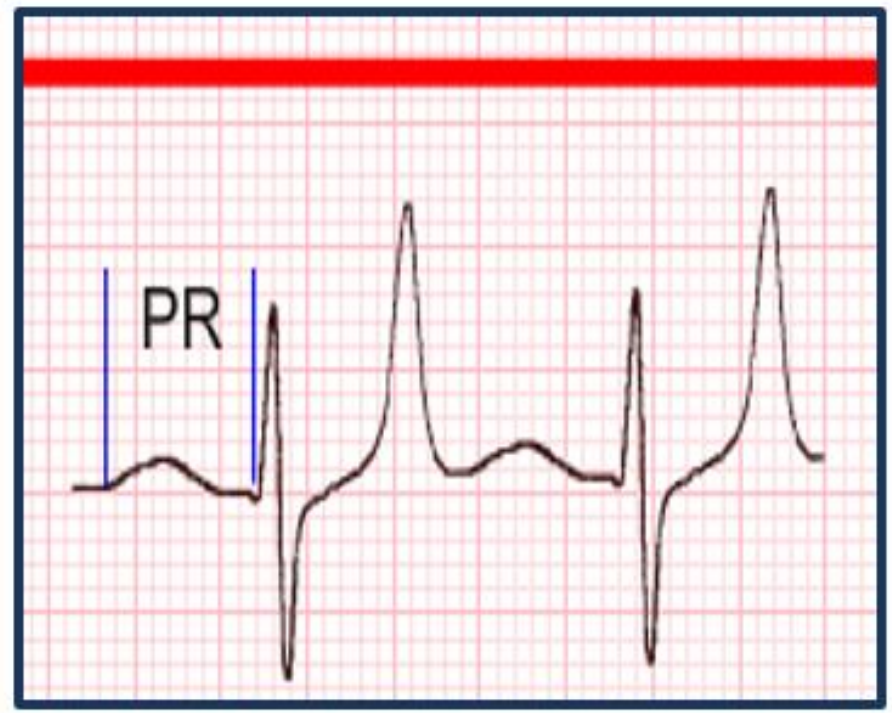
1- methylprednisolone

2- mycophenolate



45-year old patient is a known case of hepatitis C (two months ago) , he came to emergency room complaining of oliguria and palpitations , among his investigations that were done urine microscope and ECG results are shown :





Q1 : what does the urine microscope show ?

RBC cast

Q2 : what are the ECG findings ?

Peaked T wave , flattening of P wave (usually it is above this level), prolonged PR interval

Q3 : what do you suspect the most likely cause of urine microscope result in this case ?

Membranoproliferative glomerulonephritis (most common type of glomerulonephritis that associated with hepatitis C)

Q4 : what do you suspect the cause of ECG findings in this case ?

Hyperkalemia due to acute kidney impairment

Q5 : what are the investigations that you may need in this case ?

KFT , electrolytes , kidney biopsy

Q6 : what is your management in this case ?

Treat the underlying cause (hepatitis C) , steroid for glomerulonephritis , IV calcium gluconate with cardiac monitoring , IV glucose and short acting insulin , inhalational albuterol may be needed

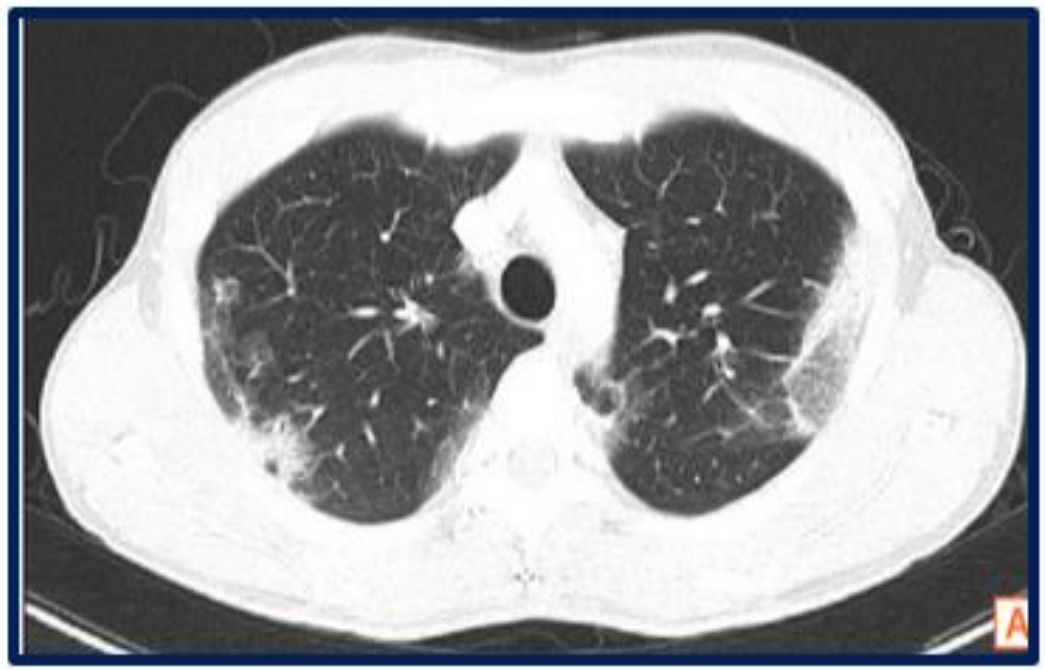
Q7 : if his ECG findings not improved upon your initial management , what is your last step in management options ?

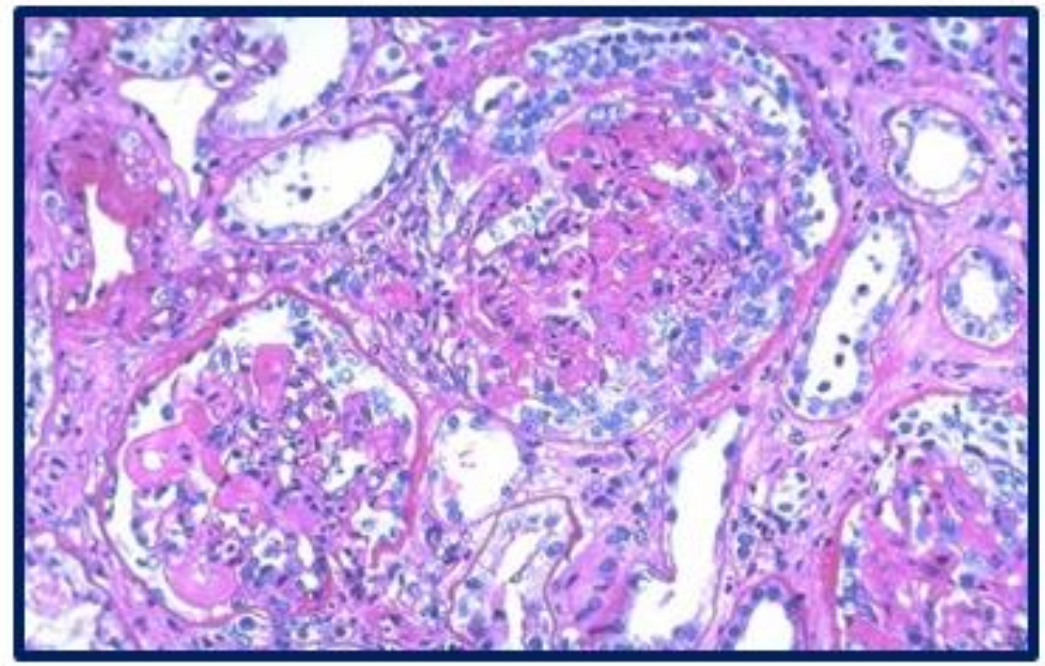
Hemodialysis

Nephritis with Normal Complements (ANCA Vasculitis & Anti-GBM)

- Granulomatosis with polyangiitis (formerly Wegener's)
  - **URTI** (sinusitis, epistaxis)
  - LRTI (infiltrates, cavitary lesions, DAH)
  - c-ANCA → anti-PR3
- Microscopic polyangiitis
  - Pulmonary hemorrhage
  - **Mononeuritis multiplex**
  - Cutaneous small vessel vasculitis (palpable purpura)
  - p-ANCA → anti-MPO
- Eosinophilic granulomatosis with polyangiitis (EGPA; formerly Churg-Strauss)
  - Asthma/Atopy
  - **Eosinophilia**

- 27 year-old female patient diagnosed with bronchial asthma and she is compliant to her treatment , but inspite of that her complains not improved , she came to emergency room complaining of SOB and chest tightness , among her investigations that were done P-ANCA is (+) chest CT and kidney biopsy are shown :





Q1 : what is the findings in patient chest CT ?

subpleural opacities

Q2 : what does renal biopsy show ?

crescent proliferation suggesting rapidly progressive glomerulonephritis

Q3 : what is the most likely diagnosis in this case ?

churg-strauss syndrome (eosinophilic granoulomatosis with poliangiitis)

Q4 : what do you suspect to see in her CBC ?

eosinophilia

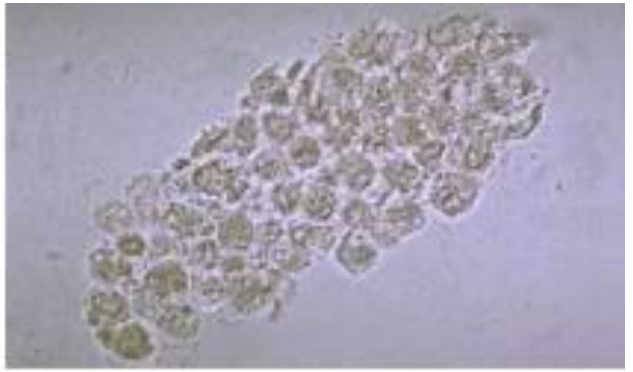
Q5 : what are the other investigations that you may need ?

urinanalysis , KFT , electrolites

Q6 : what is your management in this case ?

systemic glucocorticoids

Nephrotic syndrome



- Glomerular disorder (podocytes) characterized by **proteinuria > 3.5gm/day** resulting in:

1. **Hypoalbuminemia** – Pitting **edema.**
2. Hypogammaglobulinemia – Increased risk for infections.
3. Hypercoagulable state – due to loss of antithrombin III.
4. **Hyperlipedemia** and hypercholesterolemia – may result in fatty cast in urine = **lipiduria**.

- **Primary nephrotic disorders:** Minimal change disease, Membranous glomerulopathy, focal segmental glomerulosclerosis, membrenoproliferative GN.
- **Secondary causes**: Diabetes mellitus, systemic amyloidoisis.

Minimal change disease (MCD)

- **Minimal change disease**: most common **in children and young** adults (sudden onset of severe nephrosis).
- Patient usually normotensive, nephrotic sediment, normal renal functions.
- Secondary etiology is: **NSAIDS, Hodgkin disease**.
- **Excellent response to steroids**.

Focal segmental Glomerulosclerosis (FSGS)

- Most commonly in **African Americans**.
- Patients **usually hypertensive**, usually progress to ESRD over 5-20 years.
- Primary idiopathic.
- Secondary: (Familial, **HIV (collapsing type FSGS),** parvovirus, **Heroin**, obesity).
- Treated by: ACEI/ARBS, Steroids.

Membranous Neohropathy

- Most common cause of idiopathic nephrotic syndrome in **Caucasian adults.**
- Heavy proteinuria is common, hypertension and azotemia develops as disease progress.
- Increased incidence of **renal vein thrombosis** (Flank pain/ Hematuria/ high LDH).
- Secondary etiologies: **Carcinomas**, NSAID, Penicillamine.
- Treatment: ACEI +- immunomodulation.

Nephrotic Syndrome

	Primary*	Secondary
Minimal Change Disease **(Clue: Young adults/kids)**		**NSAIDs** Lymphoma
Focal Segmental Glomerulosclerosis **(Clue: African American)**		**Genetic** **(*APOL 1* & *MYH9* in AA)** HIV Heroin Obesity
Membranous Glomerulopathy **(Clue: Adults — cancer?)**		**Solid tumors** SLE (lupus nephritis)
Membranoproliferative GN **(Clue: Low complements)**		**Hepatitis C**

Diabetic nephropathy

- **Most common cause of nephropathy in adults.**
- **Leading cause of ESRD in US**.
- 30% with type 1 DM, 20% with type 2 DM develop diabetic nephropathy.
- Non enzymatic glycosylation **affects the efferent** more than afferent lead to high GFR.
- Initially **starts as microalbuminuria** due to hyprefiltration **followed by heavy proteinuria due to sclerosis of mesangium and decline in renal function.**
- Diagnosis is made on clinical grounds (unless there is no retinopathy).
- Treatment**: ACEI/ARBS to slow down the progression**.

Amyloidosis and Multiple myeloma

- Amyloid: Biochemical forms (AL: MM, AA: Inflammatory states (RA)).
- Multiple Myeloma:

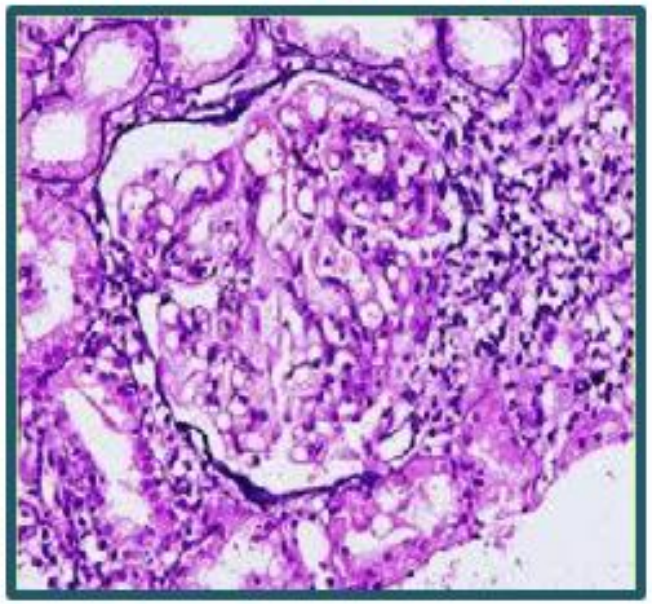
1. Renal failure with hypercalcemia.
2. Discrepancy in urine dipstick and urine prot/creat ration.
3. Low anion gap.
4. Total protein to albumin ratio > 2:1.
5. Back pain in elderly.

Q: 1. This biopsy is taken from which organ?

Kidney

2. Mention 1 indication.

Nephrotic syndrome (extereme ages, resistant to steroids .. Etc)/ Nephritic syndrome … etc

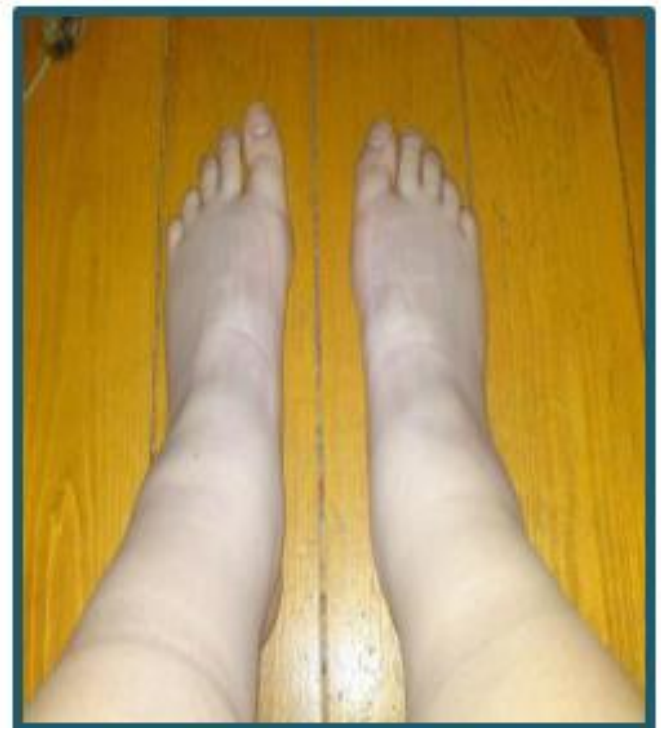


Q: This pt admitted with of bilateral lower limb pitting edema, & puffy eyes. He is a known case of Diabetes. What do you think this pt have?

nephrotic syndrome

What is the best test to start with in this case?

urinalysis, 24 hour urine collection.



A known case of diabetes presented complaining of bilateral lower limb edema & facial puffiness.

What is your diagnosis?

Nephrotic Syndrome due to Diabetic nephropathy

What is the confirmatory test?

24- hour urine protein collection

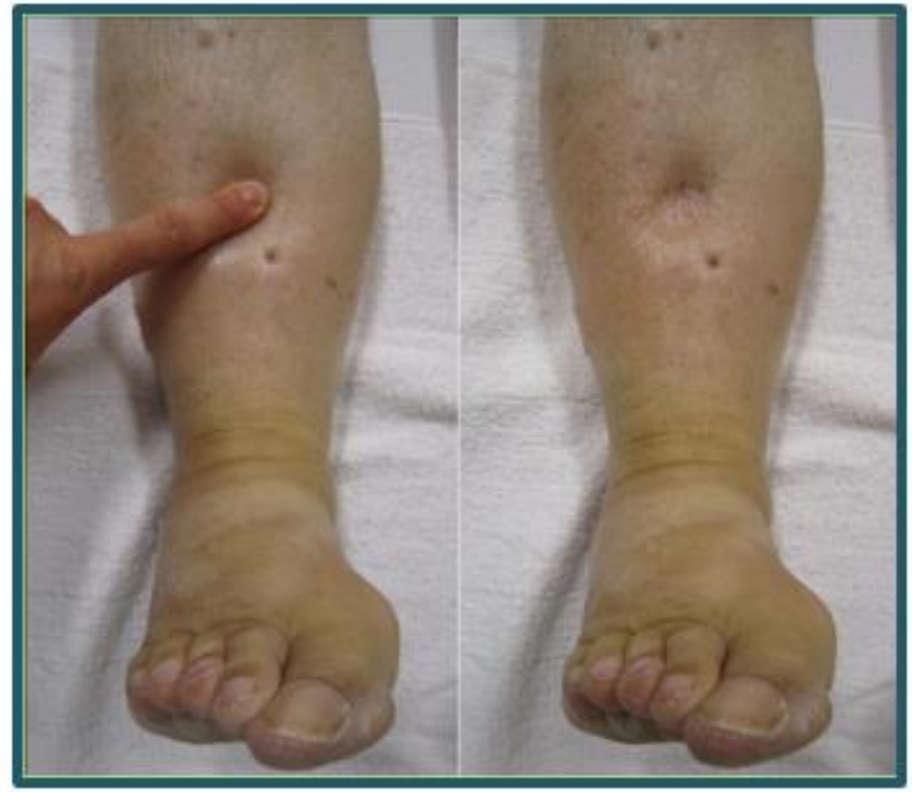
Q: A 50-year old diabetic patient developed the following.

A-What is your diagnosis?

DM nephropathy

B-What is the first lab investigation to be done?

24-hour urine collection for protein

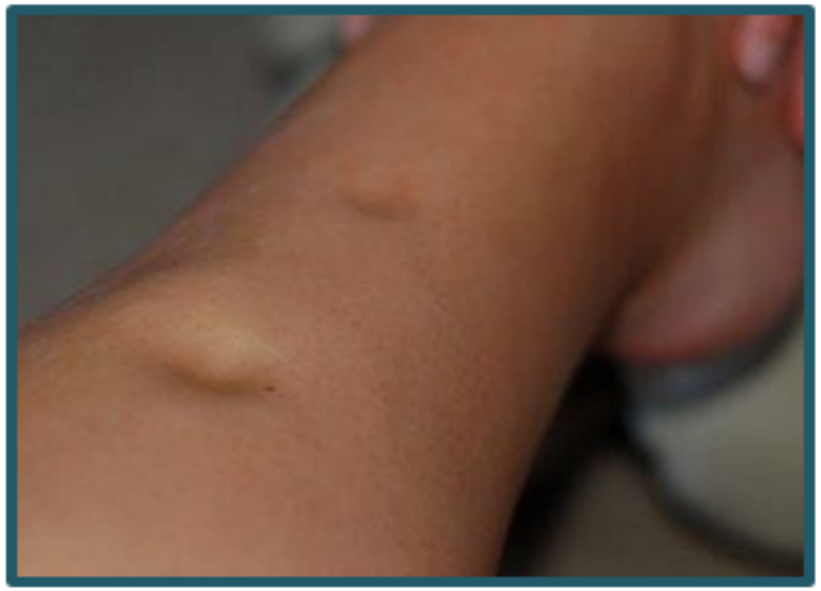


Q: A 48 year old diabetic patient presented with bilateral lower limb edema and frothy urine. What is the cause of his condition

Nephrotic Syndrome (Due to Diabetic Nephropathy)

What is the test you want to do for him?

Urine Analysis for proteinuria.



Q: 34 YO male presented with bilateral lower limb edema, puffiness of face, peri-orbital edema. 24-hour urine collection sample showed 5.4g protein

1- What other 2 findings you suspect to have in the serum of this patient ?

Hypoalbumenia/Hyperlipidemia.

2- write 2 causes that would lead to his condition.

Amyloid , Diabetic nephropathy

3- what is the diagnostic test that will give you the etiology & guide your treatment ?

Kidney biopsy

4-What is your diagnosis?

diabetic nephropathy.

5-Mention 2 other possible lab findings in this case.

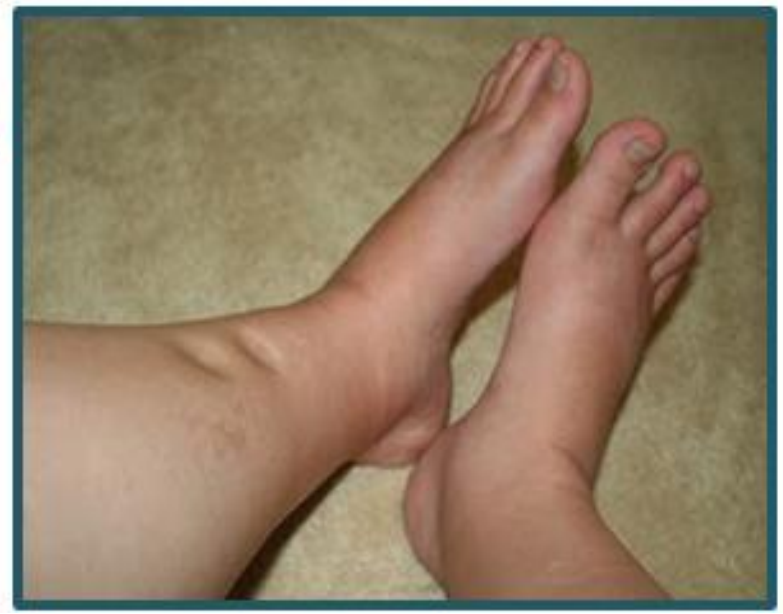
Hyperlipidemia and hypoalbuminemia

6-What is the most appropriate treatment in his case?

control HTN and diabetes , give ACEI for example

Q: A female pt visited your clinic complaining of bilateral leg swelling & peri-orbital edema. She is a known case of DM which was controlled until 3 months ago. She developed HTN 3 months ago, but was not controlled even with 2 drugs. On examination she has mild respiratory distress & large edema in her legs.

A- What is your most likely Dx?
Nephrotic Syndrome.
B- Mention 2 confirmatory tests.
24h urine collection for albumin (> 3.5 gm) / Serum albumin (dec.) /Serum lipids profile(inc.).
C- Mention 2 lines of management for this pt.
Steroids /Prophylactic Anticoagulants/ Diuresis
D- Mention 4 causes of this condition.
Heart failure
Renal failure,
Nephrotic syndrome
Liver cirrhosis
Hypo-albuminemia
Fluid overload



Q: female pt with frothy urine , DM , edema around eyes, what is your 2 lab findings?

hypoalbuminemia

Hyperlipidemia

Proteinuria

Q: 67 YO woman presents with SOB on exertion &bilateral ankle edema that she noticed just today. UA/ 24 hour urine 3+ Protein, low Albumin-3.4 g/dL (3.5-5g/dL).

Q1: What is the most likely diagnosis?

Nephrotic syndrome.

Q2: mention 2 common secondary causes of Dx?

DM, SLE , lymphoma.

Q3: mention 2 complications related to the Dx?

Increased chances of infection,

Hypercoagulability.

Q 3 , 4 , 5

Blood test result showing very high blood sugar and elevated Creatinine .

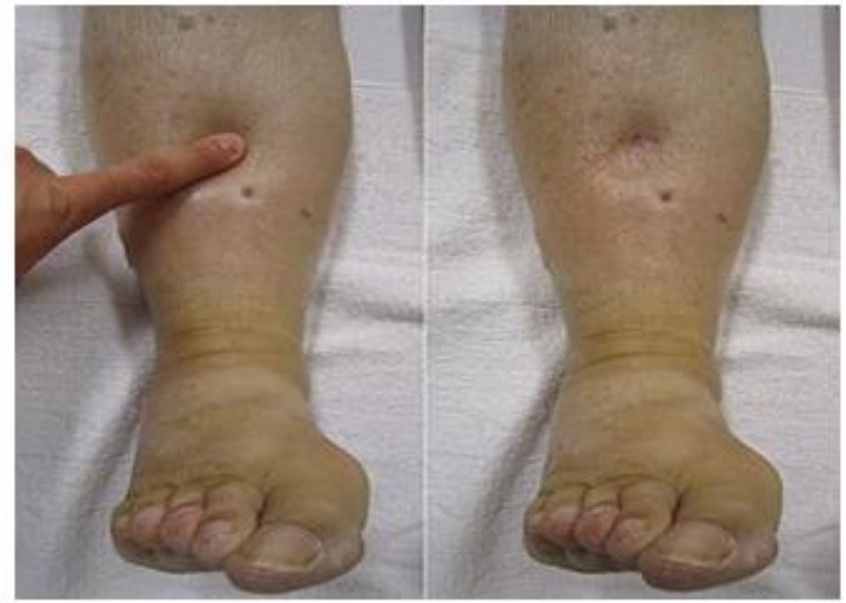
•What are abnormal findings in this test ?
Very high blood glucose and creatinine .

•What is the diagnosis ?
Diabetic nephropathy

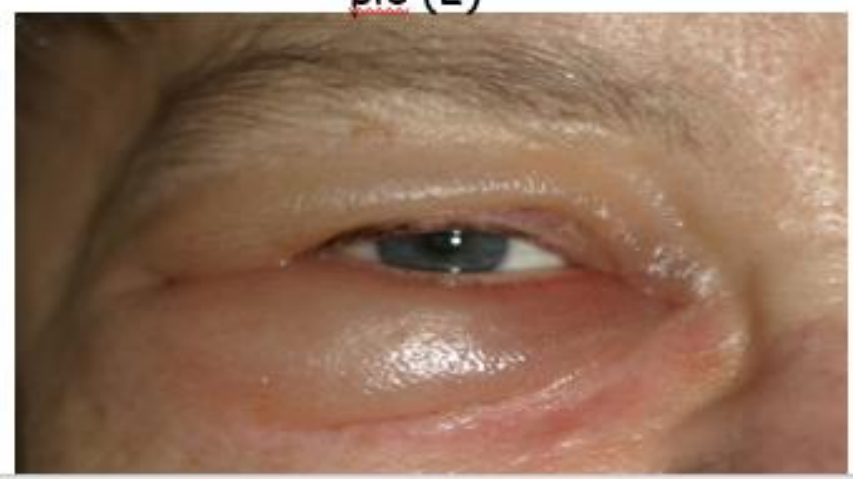
•After 10 years the patient comes with this pic (1) , what is the diagnosis ?

•After 15 years the patient comes with this pic (2) , what is the diagnosis ? And what is the treatment ?
I guess nephrotic , control DM by hypoglycemic agent and insulin , fluid restriction , diuretics , steroid and albumin

pic (1)



pic (2)



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A- What is your most likely Dx?

Nephrotic syndrome

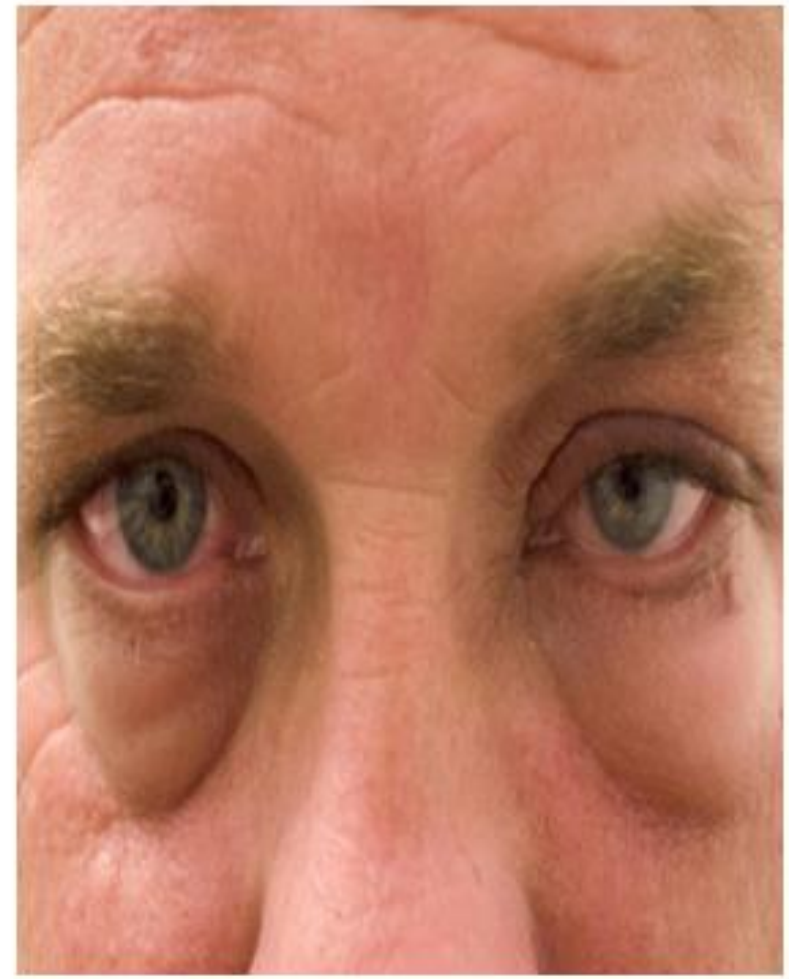
B- Mention confirmatory test:

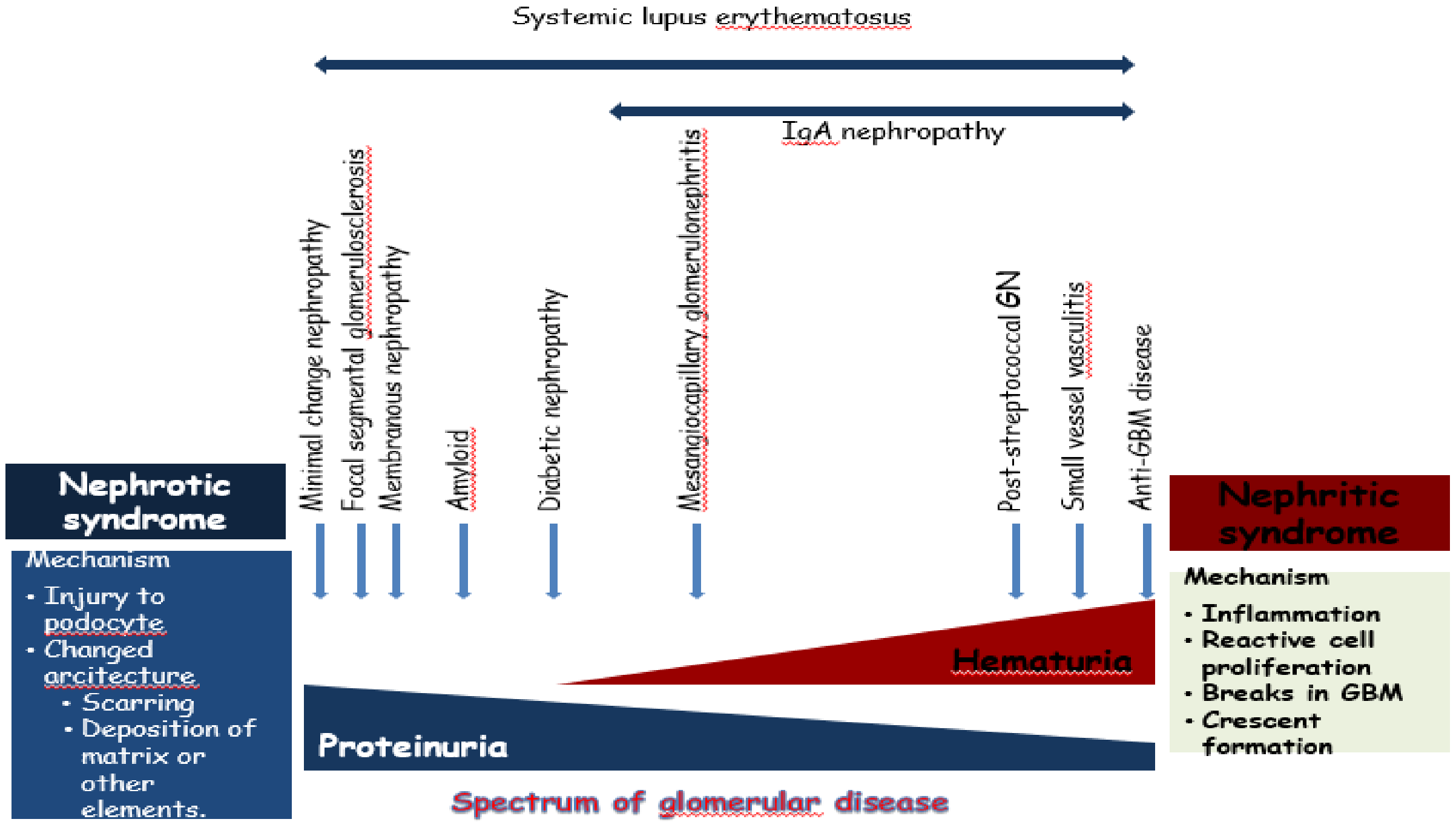
Urinalysis

NEPHROLOGY SECTIONS

Q1 : Diabetic patient , wake up with this peri-orbital edema , what is your diagnosis ? And the most possible complication ?

- Nephrotic syndrome , DVT (Hyper-coagulable status).




Systemic lupus erythematosus
IgA nephropathy
Minimal change nephropathy
Focal segmental glomerulosclerosis
Membranous nephropathy
Amyloid
Diabetic nephropathy
Mesangiocapillary glomerulonephritis
Post-streptococcal GN
Small vessel vasculitis
Anti-GBM disease
Nephrotic syndrome
Mechanism
• Injury to podocyte
• Changed arcitecture
• Scarring
• Deposition of matrix or other elements.
Nephritic syndrome
Mechanism
• Inflammation
• Reactive cell proliferation
• Breaks in GBM
• Crescent formation
Hematuria
Proteinuria
Spectrum of glomerular disease

CKD (ESRD)

- Structural/Functional kidney abnormalities X3 months, with or without decrease in GFR<60.
- May results from glomerular, tubular, inflammatory, or vascular insult.
- **Most common causes are DM,HTN, Glomerular diseases (Kidney).**
- Clinical features: **Urinary** (Polyuria to Anuria, frothy .. Etc), **Uremia** (increased BUN in blood (azotemia) results in nausea, anorexia, pericarditis, platelet dysfunctions, encephalopathy with asterixis, and deposition of urea crystals in the skin), **Salt and water retention** (Hypertension), **Hyperkalemia** with **metabolic acidosis**, **Anemia** (due to decreased EPO production), **Hypocalcemia** (Due to decrease 1a hydroxylation of vitamin d, and due to hyperphosphatemia), **Renal osteodystrophy, osteomalacia, osteoporosis**, **Infections**.

Cockroft = used to calculate drug doses.

Estimation of GFR

- Cockcroft- Gault Formula

$$\text{CrCl (ml/min)} = \frac{(140 - \text{age}) \times \text{Weight in Kg}}{72 \times \text{Serum Creat (mg/dl)}} \times (0.85 \text{ if female})$$

- MDRD Study Equation

$$\text{GFR (ml/min/1.73 m}^2) = 186 \times (S_{Cr})^{-1.154} \times (\text{age})^{-.203} \times (0.724 \text{ if female}) \times (1.210 \text{ if African American})$$

CKD stages

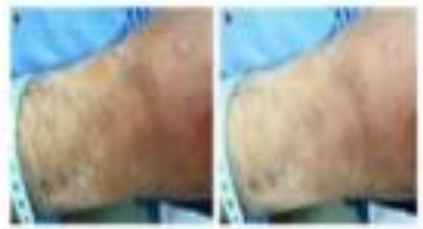
The higher albuminuria the more severe will be and the more cardiovascular disease.

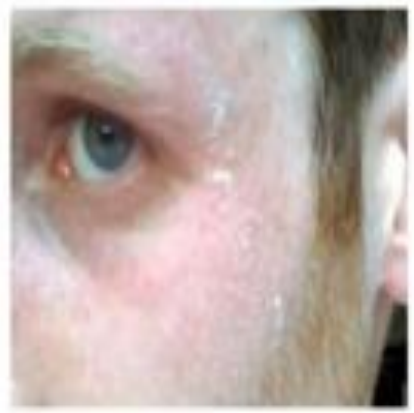
GFR categories (ml/min × 1.73 m²)			Albuminuria categories (mg/g creatinine) A1	A2	A3
			< 30	30–300	> 300
	G1	≥ 90			
	G2	60–89			
	G3a	45–59			
	G3b	30–44			
	G4	15–29			
	G5	< 15			
	G5 D/T	Kidney failure	Usually defined by KRT		

Severely albuminuria

At 3b the complications will start (Anemia).
Stage 5 is either 5D or 5 without D= Dialysis).

Uremic Forest





Earthy color face

Half and half nail due to uremia.

Investigation

- Urea/Cr
- Urinalysis and quantification of proteinuria;
- Electrolytes; hyperkalaemia and acidosis, Calcium, phosphate, parathyroid hormone; Albumin
- Full blood count (± Fe, ferritin, folate, B12);
- Lipids, glucose ± HbA1c;
- Renal ultrasound: size, asymmetry, cyst
- Hepatitis and HIV serology
- ECG
- SPEP, serological test

What are the Causes of CKD that cause a large kidney rather than small:
1. DM. 2. Amyloidosis. 3. APKD. 4. HIV And if there is hydronephrosis.

The difference between left and right is more than 1.5cm

Progression

Our target is <140/90 if proteinuria it is 130/80

- BP
- Glycemic control
- RAAS blocker ACEI/ ARBS
- SGLT2 inhibitors Canagliflozin …etc
- Stop smoking
- Diet: protein, salt, phosphate, K
- Exercise
- Obesity
- Acidosis HCO3 < 22
- Uric acid ?
- Dyslipidemia

Treat the cause

- GN
- Pyelonephritis
- AIN
- Obstruction
- Renovascular disease

Anemia target is 10-11.

Q: Patient x , 67 years old , with 10 years history of HTN and DM , present with bleeding gum , and epistaxis , pruritus , arrythmia , on exam has astrexis , labs indicate metabolic acidosis and hyperkalemia

Q1 what is ESRD ??

that form of kidney failure so severe as to need dialysis or renal transplantation.

- ESRD is not defined as a particular BUN or creatinine. ESRD is defined as the loss of renal function leading to a collection of symptoms and laboratory abnormalities also known as uremia.

- Uremia is a term interchangeable with the conditions for which dialysis is the answer as therapy.

Q2 what are the etiology ??

The most common causes of end-stage renal disease (ESRD) requiring dialysis are diabetes and hypertension. The next most common cause is glomerulonephritis (15% of cases), followed by cystic disease and interstitial nephritis (each 4–5%).

- ESRD usually implies disease that has been present for years; however, rapidly progressive glomerulonephritis is so named because it can lead to ESRD over weeks.

- Q3 what are the manifestations ??

anemia

hypocalcemia

hyperphosphatemia

hypermagnesemia

osteodystrophy

bleeding

infection

pruritus

- Q4 what treatment for manifestation ??
- Anemia Erythropoietin replacement and iron supplementation
- Hypocalcemia and osteomalacia Replace vitamin D and calcium
- Bleeding Desmopressin (DDAVP) increases platelet function; use only when bleeding
- Pruritus Dialysis and ultraviolet light
- Hyperphosphatemia Oral binders
- Hypermagnesemia Restriction of high-magnesium foods, laxatives, and antacids
- Atherosclerosis Dialysis
- Endocrinopathy Dialysis, estrogen and testosterone replacement.

- Q5 what are the indications for dialysis ??

1 metabolic acidosis ph < 7.1

2 symptomatic hyperkalemia , K > 6.5 mEq/L

3 ingestion of toxic alcohols salicylate lithium

4 volume overload

5 symptomatic uremia (enchephalopathy , pericarditis , bleeding)

- Q6 what are the advantages of renal transplantation ??

The advantages of renal transplantation over dialysis are: - Better survival and quality of life.

- Anemia, bone disease, and hypertension persist in spite of dialysis: these are better controlled with transplantation.
- Transplant patients have a return of normal endocrine, sexual, and reproductive functions, and enhanced energy levels; thus, returning to fulltime employment and more strenuous physical activity is possible.
- In diabetics, autonomic neuropathy persists or worsens after dialysis; whereas, it stabilizes or improves with transplantation.
- Expected survival rate after transplantation is 95% at one year and 88% at five years.

1-What's this procedure?

Hemodialysis.

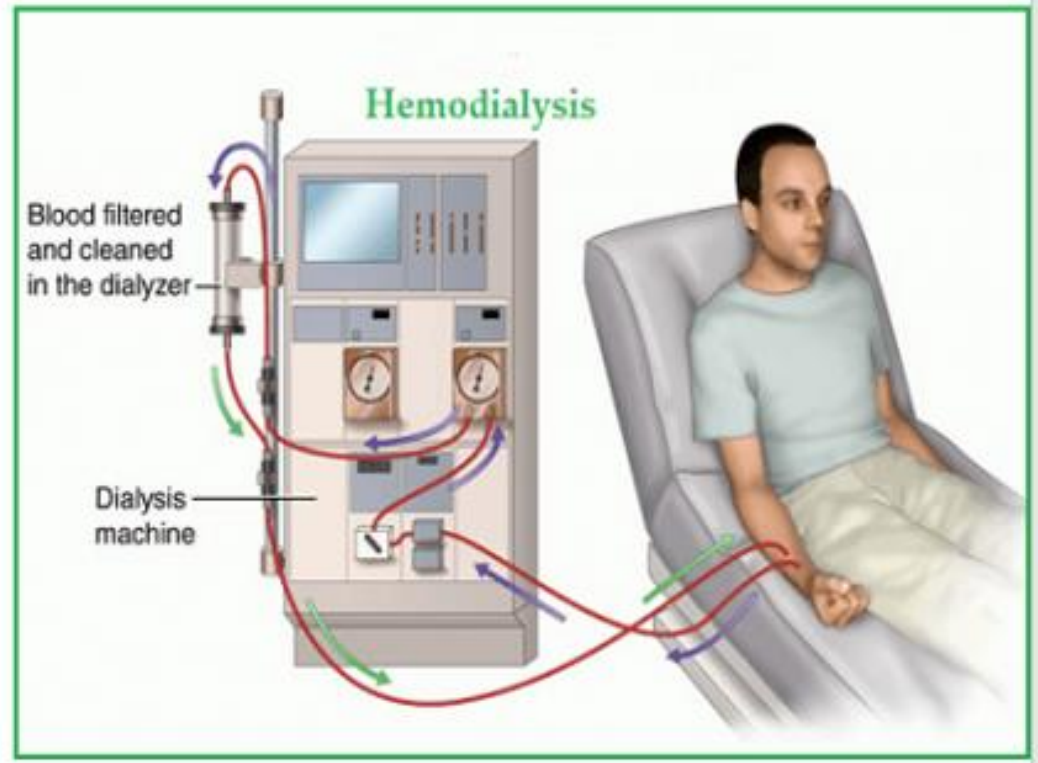
2-Mention 1 indication.

2.Renal failure (ESRD)

Note:-Dialysis Indications
AEIOU:
A:Acid-base problems(severe metabolic acidosis)
E:-Electrolyte problems(severe hyperkalemia)
I:-Intoxications
O:-Overload, fluid
U:-Uremic symptoms

Q4 : one of the following not an indication for the picture ? :

- Metabolic acidosis
- Encephalopathy
- Hyperkalemia
- **Creatinine 1000 micro.m/L**
- Pulm.edema



Which of the followings isn't an indication for hemodialysis?

A.Pulmonary edema

B.Encephalopathy

C.Creatinine =9mg/dl

D. Metabolic acidosis

E. hyperkalemia

AKI

- **Sudden and often reversible loss of renal function**, which develops over days or weeks.
- Defined as an **increase in serum creatinine by 0.3ml/dl or by 1.5 fold over baseline within 48 hrs or by oliguria (> 0.5ml/kg/hr) for at least 6 hrs.**
- AKI can be either **non-oliguria ,oligouria (<400ml/d) or anuria(<100ml/d)**.
- Elderly patients are at higher risk of developing AKI and have a worse outcome
- Cause:

1- **Pre-renal 70% :** when **perfusion to the kidney is reduced**, (If the insult is not corrected, this may lead to 'renal' injury: namely, acute tubular necrosis (ATN)).

2- **Intrinsic renal 20%: ATN** (most common cause of acute kidney injury), **Acute interstitial nephritis** (Drug induced hypersensitivity), **Renal papillary necrosis**.

3- **Post-renal:** when there is **obstruction** to urine flow at any point from the tubule to the urethra (external compression of urinary tract or intraluminal/ intratubular).

	Prerenal	Renal	Postrenal
Dipstick	0 or trace protein	Mild-moderate protein, hemoglobin, leukocytes	0 or trace protein, red and white cells
Sediment	Few hyaline casts	Granular and cellular casts[a]	Crystals and cellular casts possible
Serum BUN/Cr	20	10	10
Urine osmolality	>500	<350	<350
Urine sodium	<20	>30	
Urine/serum Cr	>40	<20	<20
Urine/serum urea	>8	<3	<3
FENa	<1%	>1%	>1%
FEUr	<35%	>50%	

FENa, fractional excretion of sodium = (urine Na/urine Cr) / (serum Na/serum Cr) %; Cr, creatinine; FEUr, fractional excretion of urea

[a]Composition of casts depends on cause of renal failure.

Adapted with permission from Thadhani R, Pasqual M, Bonventre JV. Acute renal failure. *N Engl J Med* 1996;334:1448–1460.

4. If we assume that this case is a renal, what makes you suspect that by history and examination well?

Renal			
ATN	Prolonged pre-renal state Sepsis Toxic ATN: drugs (aminoglycosides, cisplatin, tenofovir, methotrexate, iodinated contrast) Other (rhabdomyolysis, snake bite, *Amanita* mushrooms)	Vital signs Fluid assessment Limbs for compartment syndrome	Urine Na >40 mmol/L Fractional excretion Na ≥1% Dense granular ('muddy brown') casts Creatine kinase
Glomerular	Rash, weight loss, arthralgia Chest symptoms (pulmonary renal syndromes) IV drug use	Hypertension Oedema Purpuric rash, uveitis, arthritis	Proteinuria, haematuria Red cell casts, dysmorphic red cells ANCA, anti-GBM, ANA, C3 and C4 Viral hepatitis screen, HIV Renal biopsy
Tubulo-interstitial	Interstitial nephritis: drugs (PPIs, penicillins, NSAIDs) Sarcoidosis Tubular obstruction: 1. Myeloma (cast nephropathy) 2. Tubular crystal nephropathy: Drugs (aciclovir, indinavir, triamterene, methotrexate) Oxalate (fat malabsorption, ethylene glycol) Urate (tumour lysis)	Fever Rash	Leucocyturia Eosinophiluria (and a peripheral eosinophilia) White cell casts Minimal proteinuria Paraprotein Calcium (myeloma, sarcoidosis) Urine microscopy for crystals Serum urate Urine collection for oxalate
Vascular (including renal infarction, renal vein thrombosis, cholesterol emboli, malignant hypertension)	Flank pain, trauma Anticoagulation Recent angiography (cholesterol emboli) Nephrotic syndrome (renal vein thrombosis) Systemic sclerosis (renal crisis) Diarrhoea (HUS)	BP (malignant hypertension) Fundoscopy Livedo reticularis (cholesterol emboli) Sclerodactyly	Normal urinalysis or some haematuria C3 and C4 (cholesterol emboli, TMA) Doppler renal ultrasound CT angiography Platelets, haemolytic screen, LDH Consider ADAMTS13 and complement genetics (if TMA)

5.If we assume that this case is a pre-renal, what makes you suspect that by history and examination well?

15.25 Categorising acute kidney injury based on history, examination and investigations

Type of AKI	History	Examination	Investigations
Pre-renal	Volume depletion (vomiting, diarrhoea, burns, haemorrhage) Drugs (diuretics, ACE inhibitors, ARBs, NSAIDs, calcineurin inhibitors, iodinated contrast) Liver disease Cardiac failure	Low BP (including postural drop) Tachycardia Weight decrease Dry mucous membranes and increased skin turgor JVP not visible even when lying down	Urine Na < 20 mmol/L Fractional excretion Na < 1% High urea:creatinine ratio Urinalysis bland

6. If we assume that this case is a post- renal, what makes you suspect that by history and examination well?

Post-renal	Prostate cancer history Neurogenic bladder Cervical carcinoma Retroperitoneal fibrosis Bladder outlet symptoms	Rectal examination (prostate and anal tone) Distended bladder Pelvic mass	Urinalysis frequently normal (may reveal haematuria depending on cause) Renal ultrasound (hydronephrosis) Isotope renogram (delayed excretion) if ultrasound inconclusive

(ACE = angiotensin-converting enzyme; ANA = antinuclear antibody; ANCA = antineutrophil cytoplasmic antibody; ARBs = angiotensin receptor blockers; BP = blood pressure; GBM = glomerular basement membrane; HIV = human immunodeficiency virus; HUS = haemolytic uraemic syndrome; JVP = jugular venous pulse; LDH = lactate dehydrogenase; Na = sodium; NSAIDs = non-steroidal anti-inflammatory drugs; PPIs = proton pump inhibitors; TMA = thrombotic microangiopathy)

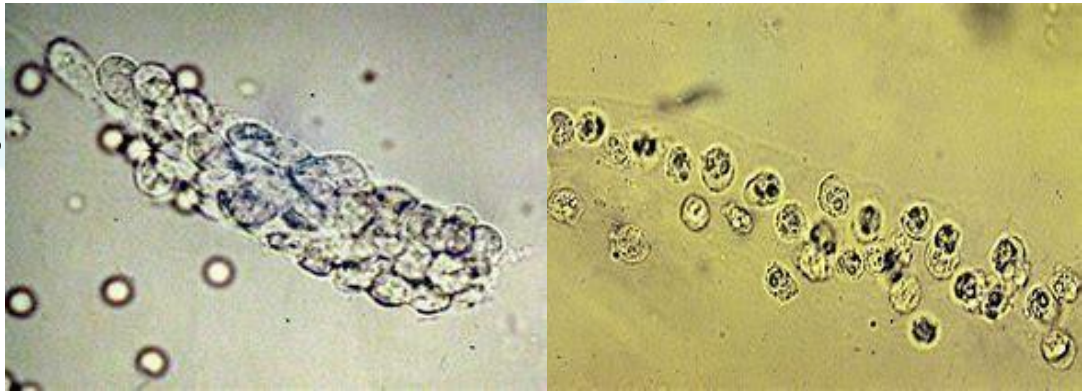
6. What the management of AKI?

15.26 Management of acute kidney injury

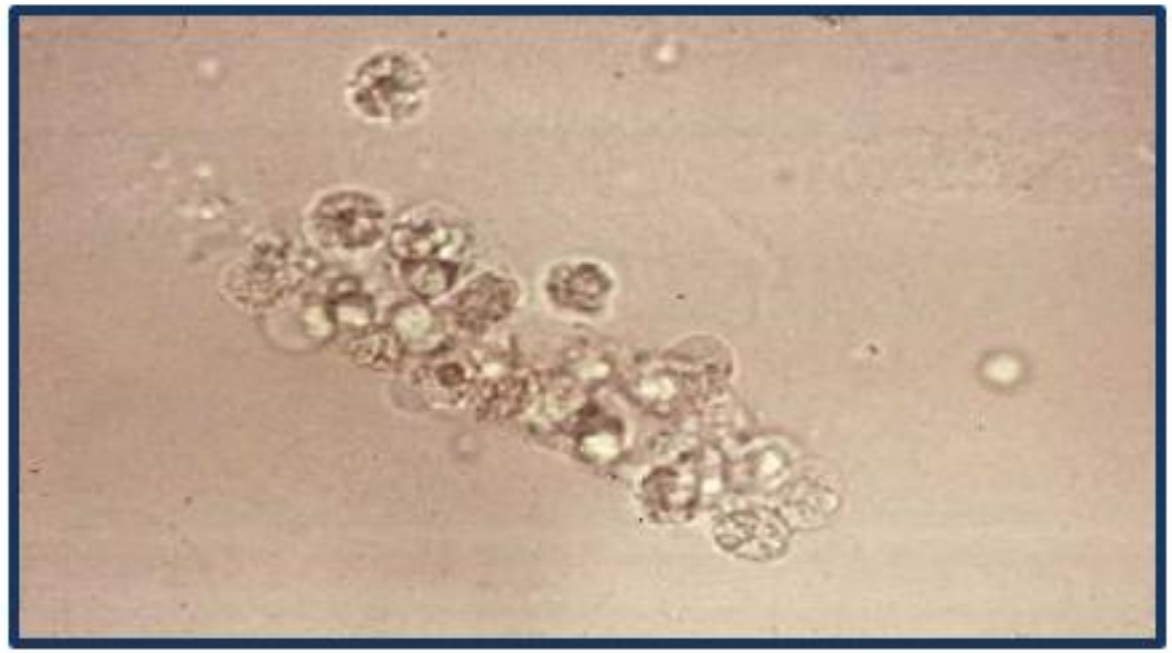
- Assess fluid status as this will determine fluid prescription:
  - If hypovolaemic: optimise systemic haemodynamic status with fluid challenge and inotropic drugs if necessary
  - Once euvolaemic, match fluid intake to urine output plus an additional 500 mL to cover insensible losses
  - If fluid-overloaded, prescribe diuretics (loop diuretics at high dose will often be required); if the response is unsatisfactory, dialysis may be required
- Administer calcium resonium to stabilise myocardium and glucose and insulin to correct hyperkalaemia if $K^+ > 6.5$ mmol/L (see Box 14.17, p. 363) as a holding measure until a definitive method of removing potassium is achieved (dialysis or restoration of renal function)
- Consider administering sodium bicarbonate (100 mmol) to correct acidosis if H^+ is >100 nmol/L (pH <7.0)
- Discontinue potentially nephrotoxic drugs and reduce doses of therapeutic drugs according to level of renal function
- Ensure adequate nutritional support
- Consider proton pump inhibitors to reduce the risk of upper gastrointestinal bleeding
- Screen for intercurrent infections and treat promptly if present
- In case of urinary tract obstruction, drain lower or upper urinary tract as necessary

Acute interstitial nephritis

- **Drug induced hypersensitivity** that involves the interstitial and tubules, Which results in pre-renal AKI.
- Causes includes: **NSAIDS, Antibiotics (Penicillin/ Cloxacilin), diuretics, and anti-epileptic drug (phenytoin).**
- Presents as: **oliguria, fever, rash** weeks after the starts of therapy which may be associated **with esonophile in the urine** (WBCS Cast), and **eosinophilia in CBC**.
- Resolves with **cessation of drug, then re-assess the Scr if it continues to increase give steroid**.
- May progress to **renal papillary necrosis**.



35-year old patient diagnosed with epilepsy 2 years ago , he came to emergency room complaining of fever and rash , the CBC was done and showed elevated WBC and elevated eosinophils , among his investigations that were done urine microscope result is shown :



Q1 : what does the urine microscope show ?

WBC cast

Q2 : what is the most likely diagnosis that interprets his complains ?

Interstitial nephritis

Q3 : what is the most likely cause of his disease ?

His anti-epileptic drug (phenytoin)

Q4 : what is the most common antibiotic that can cause his disease ?

Cloxacilin

Q5 : what are the other investigations that you may need ?

KFT (cr , urea) , electrolytes (K , na) , biopsy (not done usually)

Q6 : what is your management in this case ?

Stop phenytoin and choose other anti-epileptic drug , and then re-assess the cr if it continues to increase give steroid

Q: Hx of a hospitalized patient with HTN , DM underwent cardiac catheterization , taking multiple medications , a contrast CT was done to him , presented with Acute kidney injury .

1. Mention 3 causes of hospital induced renal failure.

ATN (ischemia), Contrast nephropathy , acute interstitial nephritis (AIN) (drugs: PPIs is the most common cause)

2. True or False about Kidney Injury Molecule 1 (KIM-1)

1- novel biomarker for human renal proximal tubule injury. True

2-not affected by UTI or chronic kidney failure. True ?????

3- not affected by cardiac catheterization. False

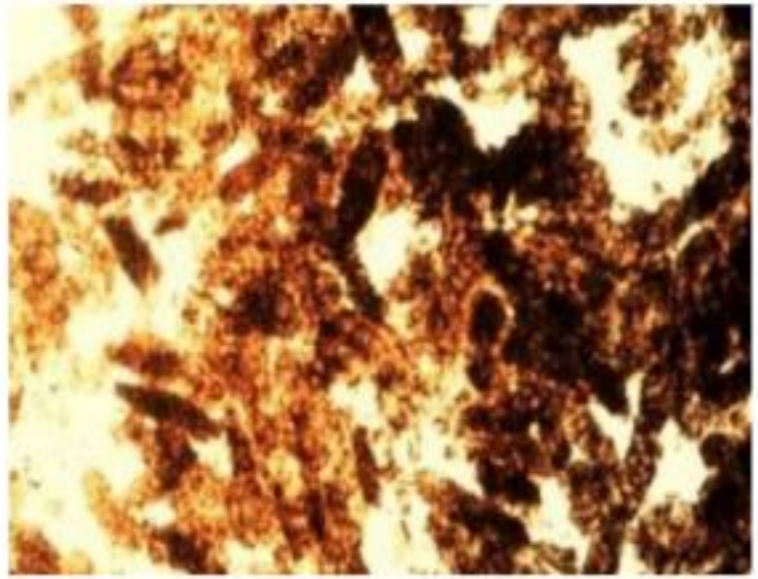
ACUTE TUBULAR NECROSIS

A. Injury and necrosis of tubular epithelial cells (Fig. 12.5); most common cause of acute renal failure (intrarenal azotemia)
B. Necrotic cells plug tubules; obstruction decreases GFR.
    1. Brown, granular casts are seen in the urine.
C. Dysfunctional tubular epithelium results in decreased reabsorption of BUN (serum BUN:Cr ratio < 15), decreased reabsorption of sodium (FENa > 2%), and inability to concentrate urine (urine osm < 500 mOsm/kg).
D. Etiology may be ischemic or nephrotoxic.
    1. Ischemia—Decreased blood supply results in necrosis of tubules.
        i. Often preceded by prerenal azotemia
        ii. Proximal tubule and medullary segment of the thick ascending limb are particularly susceptible to ischemic damage.
    2. Nephrotoxic—Toxic agents result in necrosis of tubules.
        i. Proximal tubule is particularly susceptible.
        ii. Causes include aminoglycosides (most common), heavy metals (e.g., lead), myoglobinuria (e.g., from crush injury to muscle), ethylene glycol (associated with oxalate crystals in urine), radiocontrast dye, and urate (e.g., tumor lysis syndrome).
        iii. Hydration and allopurinol are used prior to initiation of chemotherapy to decrease risk of urate-induced ATN.

Q12.a pt had this finding on microscope for his urine after he went cardiac cath

A.What is the finding?

Muddy brown cast



B.What is the ddx?

Acute tubular necrosis (ATN)

Causes is either 1. Ischemic cause (Prerenal prolonged/ after cath). 2. Toxic causes (Myoglobin/ aminoglycoside ….).

Q15.A 25-year old man is undergoing a physical examination to become a firefighter. He must carry a 200-pound bag up a flight of stairs, followed by push-ups and a walk across a balance beam. He becomes very week afterward and is brought to the emergency department with painful muscles and dark urine.

1. What is the cause of his urine color?

Myoglobinuria .

2. What is the diagnosis?

Rhabdomyolysis

3. Do you predict having RBCs in urinalysis?

No

4. What is the cause for low serum Calcium level?

Hyperphosphatemia & Deposition of CA+2 in the injured muscles .

5. What is the first line of management ?

Hydration .

Thank You

Nephrology Mini-OSCE (2)

QMA Team

ABG’S, UTI, Secondary HTN (Renal causes)

Acid-Base balance disorders

ACID-BASE REGULATION

- Maintenance of an acceptable pH range in the extracellular fluids is accomplished by three mechanisms:

1- Chemical Buffers: Phosphate Buffer Protein Buffer Bicarbonate Buffer System

- React very rapidly (less than a second)

2- Respiratory Regulation: Hyperventilation Hypoventilation

- Reacts rapidly (seconds to minutes)

3- Renal Regulation: reabsorption or excretion of filtered (HCO_3^-). Formation of titratable acid. Excretion of NH_4^+ in the urine.

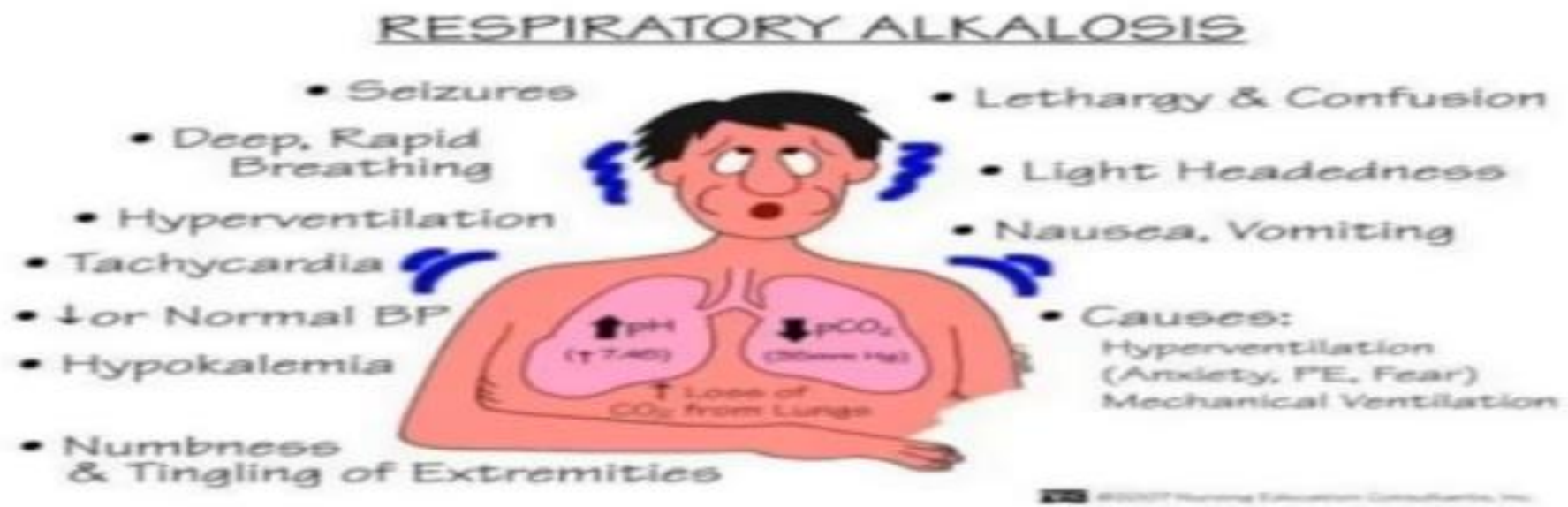
- Reacts slowly (minutes to hours)

TABLE 1

Normal ABG Figures	
PH	7.35-7.45
PO2	80-100
PC02	35-45
HC03	22-28

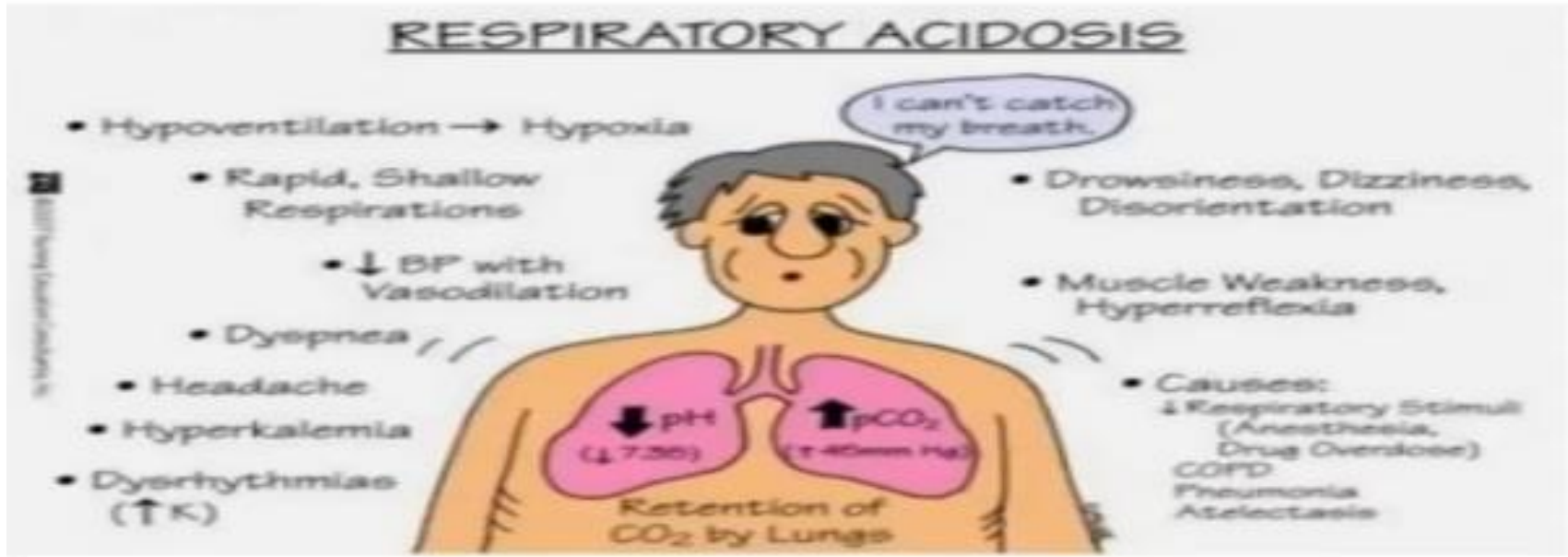
Respiratory alkalosis rapid RR

- CNS hyperactivity (pain, fever)
- CNS trauma, CVA, infection
- Hypoxia: increase demand (anemia, hyperthyroidism)
- Hypoxemia: Pneumonia, PE, pulmonary edema
- Liver insufficiency, pregnancy



Respiratory acidosis: low RR

- CNS depression (acute stroke, head trauma, narcotic overdose)
- Increase ICP
- Neurological: GBS
- Neuromuscular disease: MG
- Obstructive lung disease: COPD, OSA, foreign body



Metabolic acidosis

- Again, process add proton (lower pH)
- Is this extra proton because we losing HCO_3 adding acid
- This lead to Anion gap

Anion gap

- Anion = Cations
- Na + K + unmeasured cations (Mg + Ca + globulin) = Cl + HCO_3 + unmeasured anions (PO_4 + SO_4 + albumin + acid)
- Na — (Cl + HCO_3) = unmeasured anion — unmeasured cation

AGMA

G	Glycols (ethylene and propylene)
O	5-Oxoproline (pyroglutamic acid) chronic paracetamol use, EtOH, poor nutrition, vegetarian diet, renal failure, infection, flucloxacillin/dicloxacillin/netilmicin, Vigabatrin
L	Lactate
D	D-lactic acid Associated with short bowel syndrome
M	Methanol and other toxins (ethanol, Aldehyde)
A	Aspirin, salicylates
R	Renal failure
K	Ketoacidosis

EtOH, ethyl alcohol,.

NGMA

Gastrointestinal losses of HCO_3
- Diarrhea
- Enteric fistula
- Pancreatic fistula

Ureteral diversions
- Uretero-sigmoidostomy
- Ileal bladder
- Ileal ureter

Renal tubular acidosis
- Proximal
- Distal
- Buffer deficiency (phosphate, ammonia)

Medications
- Carbonic anhydrase inhibitors (i.e., acetazolamide)
- Amphotericin B

Urine AG

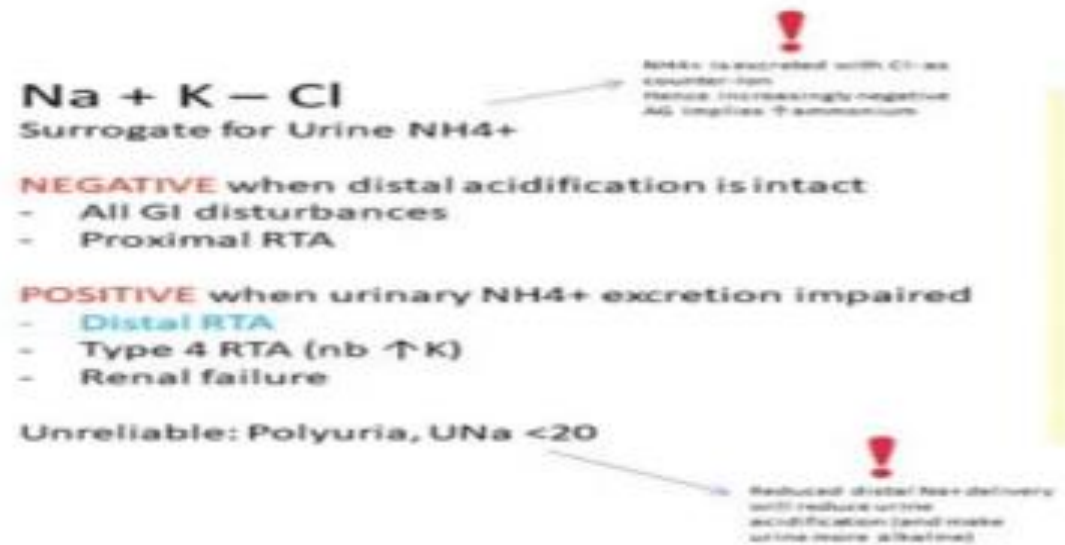
Na + K − Cl

Surrogate for Urine NH4+

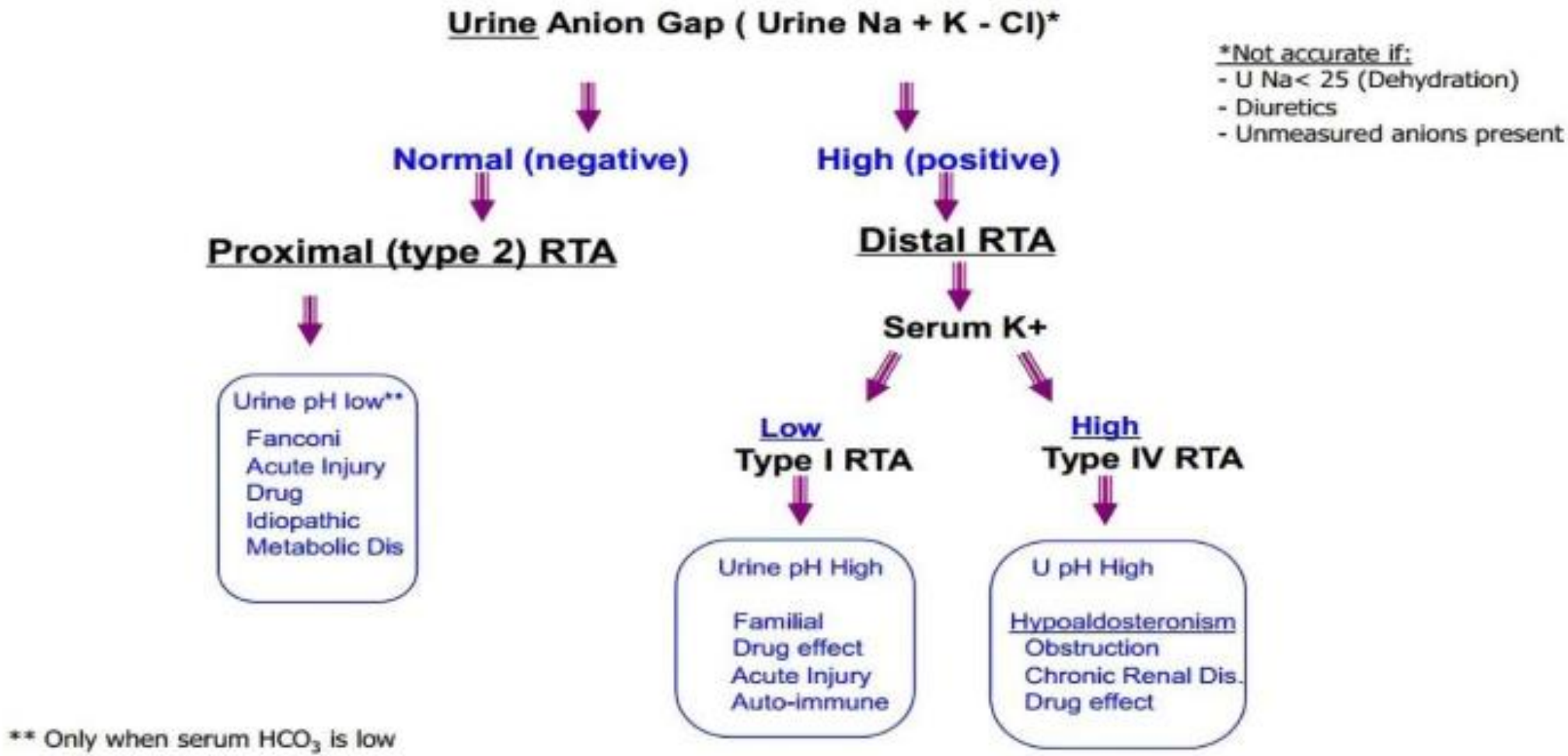
NEGATIVE when distal acidification is intact
- All GI disturbances
- Proximal RTA

POSITIVE when urinary NH4+ excretion impaired
- Distal RTA
- Type 4 RTA (nb ↑K)
- Renal failure

Unreliable: Polyuria, UNa <20



RTA



*Not accurate if:
- U Na< 25 (Dehydration)
- Diuretics
- Unmeasured anions present

** Only when serum HCO_3 is low

Q : calculate anion gap

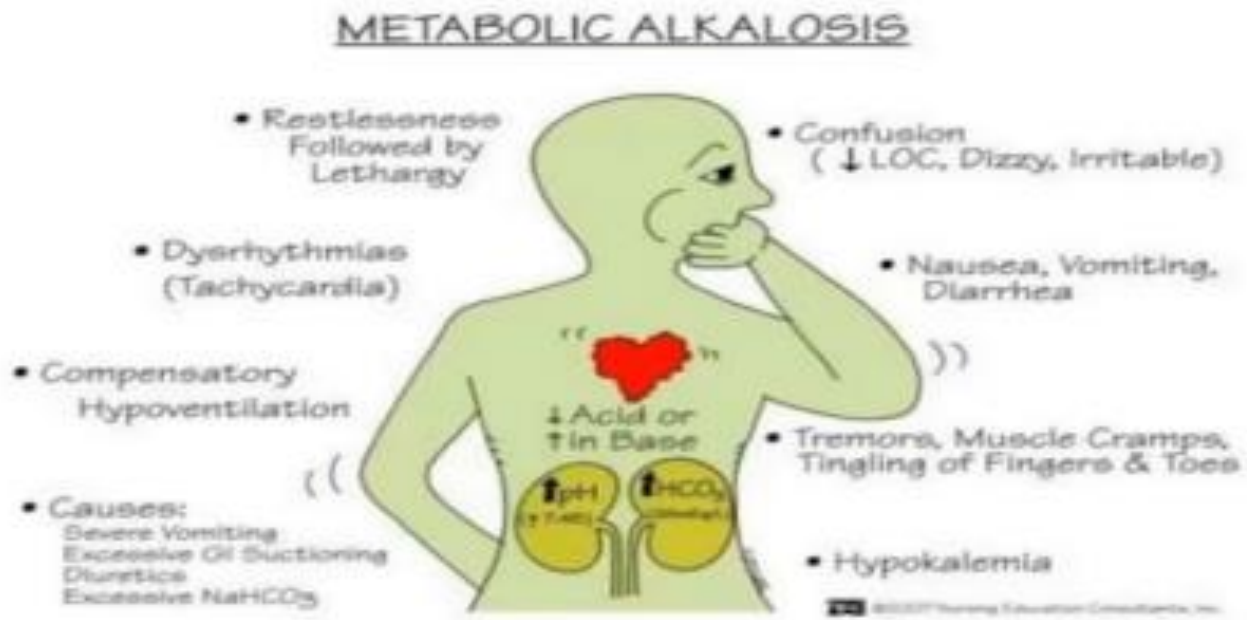
- **ABG :**
  - Na : 150
  - K : 5
  - Cl : 110
  - Hco3 :25

150+5-110 -25 = 20

METABOLIC ALKALOSIS

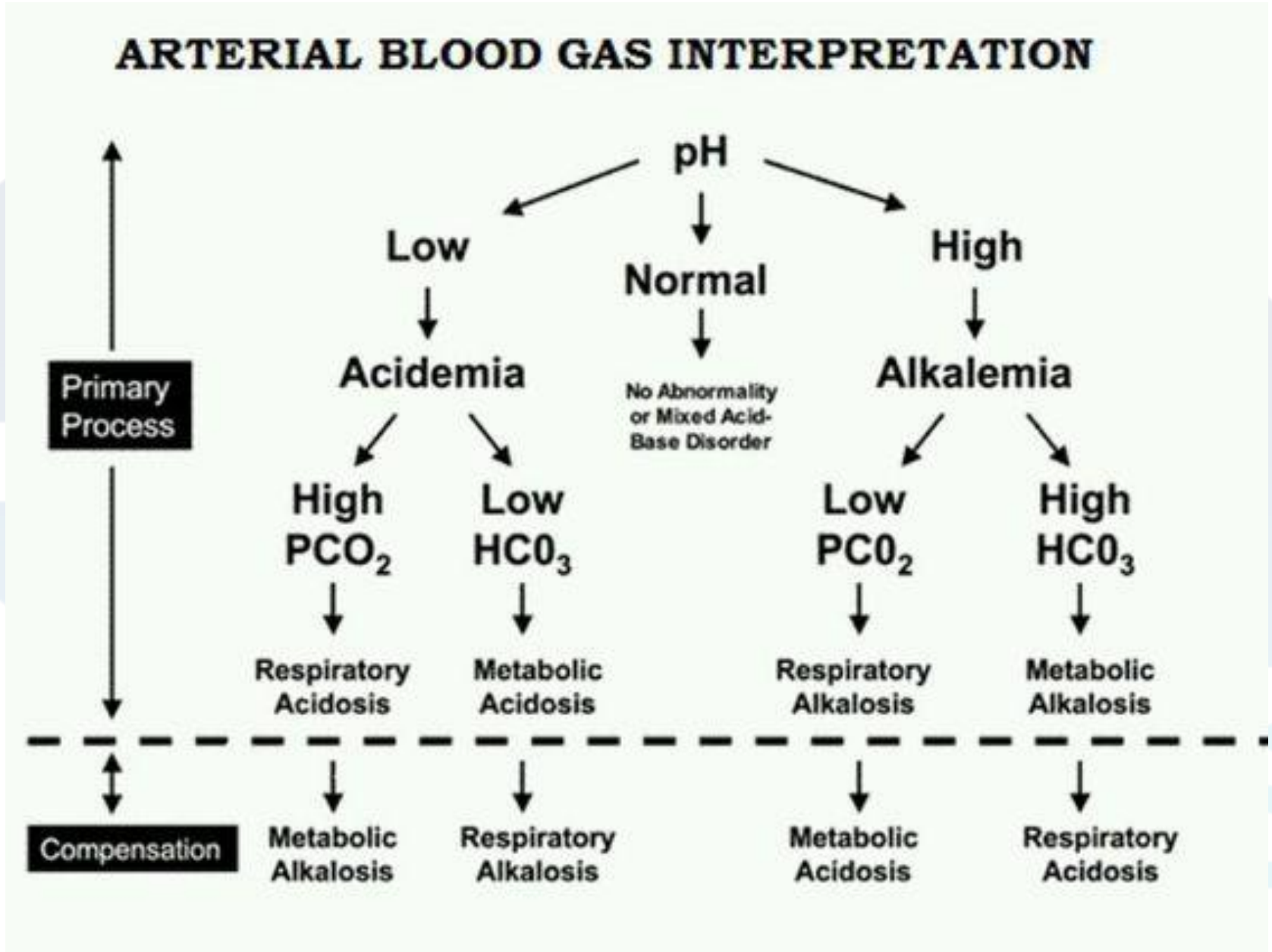
↑ HCO_3^- & ↑ pH

- Chloride responsive (responds to NaCl or KCl therapy): contraction alkalosis, diuretics, corticosteroids, gastric suctioning, vomiting
- Chloride resistant: any hyperaldosterone state (e.g., Cushing's syndrome, Bartter's syndrome, severe K^+ depletion)



Causes of Acid-Base Balance

Metabolic Acidosis Diabetic ketoacidosis Diarrhea Renal failure Shock Aspirin overdose Sepsis	**Metabolic Alkalosis** Loss of gastric secretions Overuse of antacids K+ wasting diuretics
Respiratory Acidosis Hypoventilation COPD Airway obstruction Drug overdose Chest trauma Pulmonary edema Neuromuscular disease	**Respiratory Alkalosis** Hyperventilation Hypoxia Anxiety High altitude Pregnancy Fever


ARTERIAL BLOOD GAS INTERPRETATION
pH
Low
Normal
High
Acidemia
No Abnormality or Mixed Acid-Base Disorder
Alkalemia
Primary Process
High PCO_2
Low HCO_3
Low PCO_2
High HCO_3
Respiratory Acidosis
Metabolic Acidosis
Respiratory Alkalosis
Metabolic Alkalosis
Compensation
Metabolic Alkalosis
Respiratory Alkalosis
Metabolic Acidosis
Respiratory Acidosis

Clinical approach

1- validity

2- acidemia or alkalemia

3- primary disorders

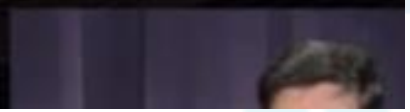
4- compensation

5- AG

6- delta/delta

Six-Step Approach to Acid-Base Analysis

1. Is the patient acidemic or alkalemic?
2. Is the overriding disturbance metabolic or respiratory?
3. If respiratory … is it acute or chronic based on compensation?
4. If metabolic … is the respiratory system compensating appropriately?
5. Is there a high anion gap? (***Always check an anion gap!!)
6. In cases of increased anion gap —
   Is there a second metabolic disturbance?
   (check corrected bicarbonate)



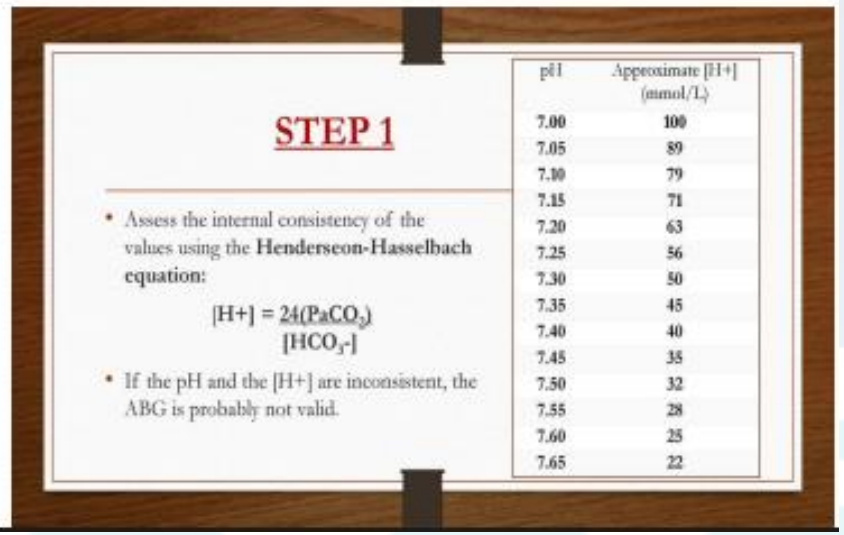
Validity

- According to the equation:

7.80- PH = PaCO2 X 24 / HCO3-

If they are equal it is valid, but if not it is not valid.

- pH = 7.30
- PO_2 = 80 mm Hg
- BE = - 14
- Na^+ = 139 m mol / L
- Cl^- = 100 m mol / L
- PCO_2 = 30 mm Hg
- HCO_3 = 10 mmol / L
- SaO_2 = 95 %
- K^+ = 4.1 m mol / L
- Anion Gap = 29



Determine Acidemia from Alkalemia

- If ph > 7.40 it is alkalemia , if ph < 7.40 it is acidemia.

pH = 7.50	PCO_2 = 50 mm Hg
PO_2 = 75 mm Hg	HCO_3 = 40 mmol / L
BE = + 16	SaO_2 = 95 %
Na^+ = 132 m mol / L	K^+ = 3.1 m mol / L
Cl^- = 88 m mol / L	Anion Gap = 4

Alkalemia

pH = 7.32	PCO_2 = 70 mm Hg
PO_2 = 62 mm Hg	HCO_3 = 32 m mol / L
BE = + 8	SaO_2 = 90 %
Na^+ = 136 m mol / L	K^+ = 3.5 m mol / L
Cl^- = 96 m mol / L	

Acidemia

Identify the primary disorder

Acid Base Disorders

Disorder	pH	$[H^+]$	Primary disturbance	Secondary response
Metabolic acidosis	↓	↑	↓ $[HCO_3^-]$	↓ pCO_2
Metabolic alkalosis	↑	↓	↑ $[HCO_3^-]$	↑ pCO_2
Respiratory acidosis	↓	↑	↑ pCO_2	↑ $[HCO_3^-]$
Respiratory alkalosis	↑	↓	↓ pCO_2	↓ $[HCO_3^-]$

- pH = 7.50
- PCO_2 = 50 mm Hg
- PO_2 = 75 mm Hg
- HCO_3 = 40 mmol / L
- BE = + 16
- SaO_2 = 95 %
- Na^+ = 132 m mol / L
- K^+ = 3.1 m mol / L
- Cl^- = 88 m mol / L
- Anion Gap = 4

Metabolic Alkalosis

- pH = 7.32
- PCO_2 = 70 mm Hg
- PO_2 = 62 mm Hg
- HCO_3 = 32 m mol / L
- BE = + 8
- SaO_2 = 90 %
- Na^+ = 136 m mol / L
- K^+ = 3.5 m mol / L
- Cl^- = 96 m mol / L

Respiratory acidosis

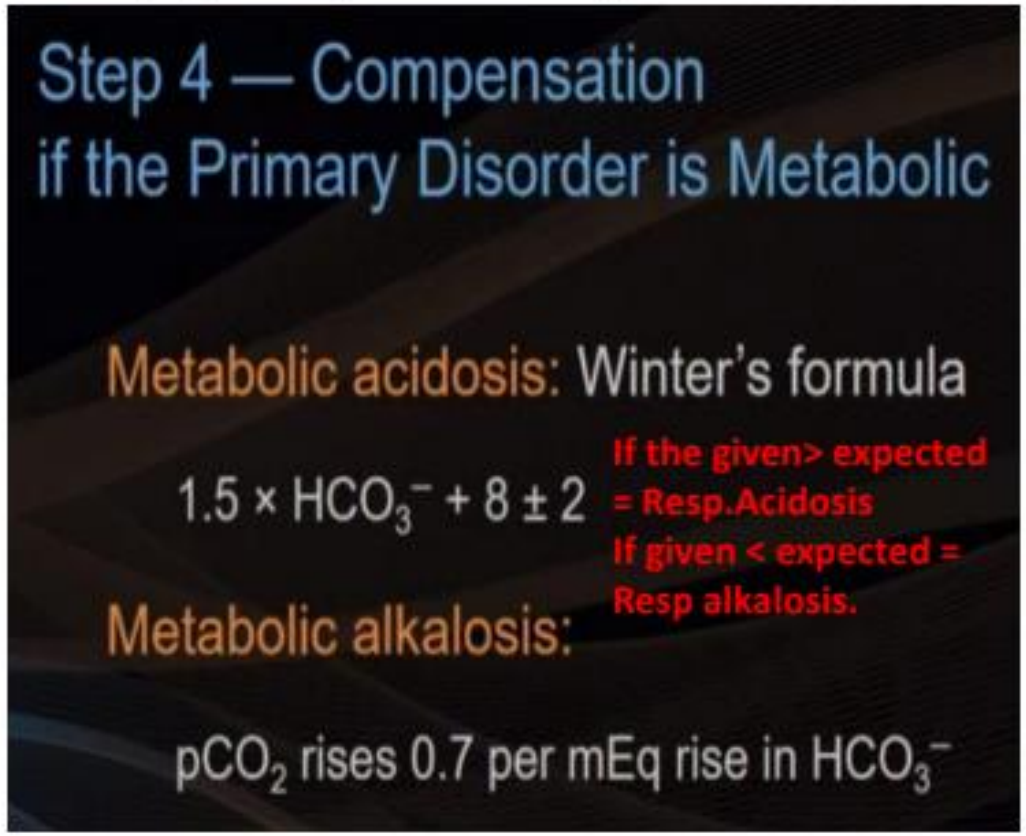
If it is Respiratory … dose the compensation acute or chronic?

ΔPCO_2 :	Respiratory acidosis	Respiratory alkalosis
Acute	10:1	10:2
Chronic	10:3	10:5
	For every rise of 10 in the pCO_2 the HCO_3^- will rise by 1 or 3	For every fall of 10 in pCO_2 the HCO_3^- will fall by 2 or 4

- pH = 7.32
- PO_2 = 62 mm Hg
- BE = +8
- Na^+ = 136 m mol / L
- Cl^- = 96 m mol / L
- PCO_2 = 70 mm Hg
- HCO_3 = 32 m mol / L
- SaO_2 = 90 %
- K^+ = 3.5 m mol / L

PCO2 Increases 30 so the expected [HCO3-] will rise 3 in acute and 9 in chronic.
[HCO3-]= 24+ 3 = 27 (A) XX
[HCO3-]= 24+ 9 = 33 (C)

If it is metabolic …. dose the respiratory appropriately compensate or not?



- pH = 7.50
- PO_2 = 75 mm Hg
- BE = + 16
- Na^+ = 132 m mol / L
- Cl^- = 88 m mol / L
- PCO_2 = 50 mm Hg
- HCO_3 = 40 mmol / L
- SaO_2 = 95 %
- K^+ = 3.1 m mol / L
- Anion Gap = 4

1. **Valid.**
2. **Metabolic alkalosis.**
3. **Respiratory compensation**???

[PaCO2]E= 16 X 0.7 = 11.2 + 40 = 51.2

E> R …….. Respiratory alkaloisis

Is there a high anion gap?

• pH = 7.32	• PCO_2 = 70 mm Hg
• PO_2 = 62 mm Hg	• HCO_3 = 32 m mol / L
• BE = + 8	• SaO_2 = 90 %
• Na^+ = 136 m mol / L	• K^+ = 3.5 m mol / L
• Cl^- = 96 m mol / L	

Anion gap = 136- 96-32= 8 < 12 so there is normal anion gap.

If there is anion gap determine what was the bicarbonate before it?

- Corrected bicarbonate = △anion gap +HCO3-
- △ anion gap = Anion gap – 12

• pH = 7.30	PCO_2 = 30 mm Hg
• PO_2 = 80 mm Hg	HCO_3 = 10 mmol / L
• BE = - 14	SaO_2 = 95 %
• Na^+ = 139 m mol / L	K^+ = 4.1 m mol / L
• Cl^- = 100 m mol / L	Anion Gap = 29

Respiratory and metabolic acidosis

29 – 12 = 17

17 + 10 = 27

So there is a metabolic alkalosis prior to the current condition.

Q8.Given the following lab results, Na = 145 K = 3.7 Cl =100 Ca = 2.5 Glucose = 143 HCO3 =10 Creatinine = 2.1.What's the anion gap?

Anion gap= Na – (Cl + HCO3) 145- (100 + 10) = 35

Q9.Fill the table with the suitable arrow

(1)--->↓decrease

(2)---->↑↑ increase

Type of Disorder	pH	PaCO2	[HCO3]
Metabolic Acidosis	↓	↓	(1)
Metabolic Alkalosis	↑	↑	↑
Acute Respiratory Acidosis	↓	↑	↑
Chronic Respiratory Acidosis	↓	↑	(2)
Acute Respiratory Alkalosis	↑	↓	↓
Chronic Respiratory Alkalosis	↑	↓	↓↓

case 6 :what is your interpretation of this ABG

high anion gap metabolic acidosis with respiratory compensation

one of these can cause this disturbance

lactic acidosis was the answer

- ABG Case
- Ph: 7.29
- Co2: 22
- hco3: 10
- Cl: 100
- Na: 145
- + other labs , normal values was given

Q10.A 39 YO woman was admitted with a Hx of generalized weakness, dyspnea, continuous nausea & diarrhea. Bowel motions were frequent & watery.

- **ABG: pH 7.29, $PaCO_2$ 25.6, PaO_2 98**
- **Na^+=125, K^+=2.8, Cl^-=101, HCO_3=14**

What is the abnormal electrolyte imbalance in this pt?

Simple metabolic acidosis.

Q28: This is ABG of A 15 years old girl presented to ER with hyperventilation.

PH 7.50
pCO2 20
HCO3 15
O2sat 96%
Na 140
Cl 103

1. what's the acid-base disturbance ?
2. what's the diagnosis ?

1- Respiratory Alkalosis with high anion gap metabolic acidosis
2- Salicylate overdose

Q1 :Patient with this ABG Results :

The ABGs interpretation ?

-Partial compensated respiratory acidosis

*One of the following can't cause this case ?

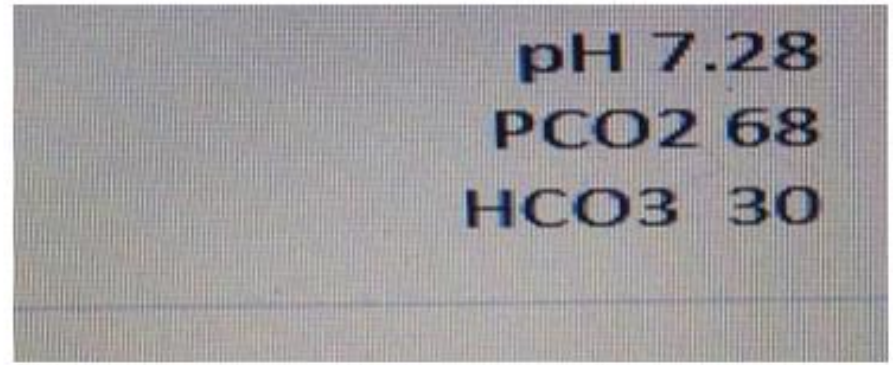
A . COPD

B. Pulmonary edema

C. guillain barre syndrome

D. Respiratoy muscle paralysis

E. Pulmonary Infarction



Q18.A 54 YO male pt complaining of severe abdominal pain, nausea, vomiting. He is a known case of DM. 3 days before he came he had URTI. On P/E; there is tenderness in the epigastric area: RR: 33. investigations: Blood Sugar: 620 mg/dl, PH: 7.2, PaCO2 : 22, HCO3: 11.

1) What is your diagnosis?

DKA.

2) What type of acid-base disorder is this ?

Metabolic acidosis.

3) what are the most common causes of this condition? What is it in this case ?

Infection, stress.

4) Give 2 lines of treatment in such cases.

IV fluid -IV glucose - IV insulin.

Q20.pH=7.3, Na=136, HCO_3 =16, Cl=110

A.Calculate the Anion gap?

Anion gap= Na – (Cl + HCO3)

=136 - (110 + 16) = 10 (normal anion gap=3-11)

B.Mention one cause.

Diarrhea or renal tubular acidosis (cause metabolic acidosis with normal anion gap)

Station 5

Medical student female came to ER

ANALYTE	Value
PH	7.50
PCO2	20mm Hg`
HCO3	24meq/L normal
SaO2	%88
PO2	70mm Hg`

Q1 : the oxygenation and acid base status ?

Respiratory Alkalosis with hypoxemia

Q2 : 2 causes for her condition ?

Panic attack ,

• A case of hepatic failure has persistent vomiting

pH 7.54 HCO3 38 mEq/L PaCO2 44 mmhg

• **What is the ABG showed ?**

Metabolic alkalosis

▪ **Expected compensation (rise in PaCO2) will be**

Every 1 mEq change in Hco3 will change PaCo2 0.6

Rise in PaCO2= 0.6 X rise in HCO3= 0.6 X (38-24) =0.6 X14=8.4

▪ **So expected PaCO2 will be 40+8.4 =48.4 mmhg. But actual value of PaCO2 is lesser than expected PaCO2 (44 vs 48.4 mmhg) which suggests presence of additional respiratory disorder (respiratory alkalosis … actual value of PaCO2 is lesser than expected PaCO2 , if its higher >> respiratory acidosis) SO pateint have Mixed disorder metabolic alkalosis and respiratory alkalosis ****

- Following sleeping pills ingestion, patient presented in drowsy state with sluggish respiration with respiratory rate 4/min.

pH 7.1 HCO3 28 mEq/L PaCO2 80 mmhg PaO2 42 mmhg

Respiratory acidosis

- **Is it Acute OR chronic respiratory disorder???**

Acute: Every 10 mmHg change in PaCo2 leads to change pH 0.08.

Chronic: Every 10 mmHg change in PaCo2 leads to change pH 0.03.

Δ pH = 7.4 – 7.1 = 0.3……. So It is Acute Disorder

**** Expected Hco3 : Every increase Co2 (10 mmHg) leads to increase Hco3 (1 mEq) = 24 + 4 = 28 mEq/ L which matches with actual HCO3, which is 28mEq/l, suggestive of simple ABD.**

- **So, the patient has primary respiratory acidosis due to respiratory failure, due to sleeping pills.**

**** If HCO3 lower than expected >> associated with metabolic acidosis , if it higher than expected >> metabolic alkalosis**

- A 15 year old boy is brought from examination hall in apprehensive state with complain of tightness of chest.

pH 7.54 HCO3 21 mEq/L PaCO2 21 mm of hg

** Respiratory alkalosis

- Is it Acute OR chronic respiratory disorder???

Acute: Every 10 mmHg change in PaCo2 leads to change pH 0.08.

Chronic: Every 10 mmHg change in PaCo2 leads to change pH 0.03.

Δ pH = 7.54 – 7.40 = 0.14… So It is Acute Disorder

** Expected Hco3 : Every ↓ Co2 (10 mmHg) leads to ↓ Hco3 (2 mEq)

= 24 – 4 = 20 mEq/ L which almost matches with actual HCO3, which is 21 mEq/l, suggestive of simple ABD

** So the patient has primary respiratory alkalosis due to anxiety….

If HCO3 lower than expected >> associated with metabolic acidosis , if it higher than expected >> metabolic alkalosis

Urinary Tract Infection

General: Infection of the lower genitourinary tract, synonymous with cystitis

Risk: Women, sexual activity, urinary catheterization, diabetes, pregnancy

Micro:

(1) *E. Coli* (most common)
(2) Enterobacteriaceae (*Proteus, Klebsiella*)
(3) *S. saprophyticus*
(4) *Pseudomonas* (if healthcare exposure)

Clinical: Dysuria, increased frequency/urgency, suprapubic pain

Diagnosis: Clinical (symptoms above) sufficient
- Urinalysis (+ nitrites, leuk esterase) can be supportive
- Culture generally not required, but used in those with persistent symptoms /high risk for drug resistant organism

Management	Criteria	Management
Simple Cystitis (uncomplicated)	- Infection confined to bladder in non-pregnant woman or man - Lacks systemic symptoms below	- First line: TMP-SMX, Nitrofurantoin, Fosfomycin
Complicated	(1) Systemic signs (ie Temp > 100°F) (2) Flank pain/CVA tenderness	See next page
Pregnancy	- [See: OB-GYN]	
Prophylaxis	- Recurrent UTI (≥ 2 infections in six months or ≥ 3 UTIs in one year) - Risks: Frequent sexual activity, spermicide use, post-menopause	- Behavioral modification (post-coital voiding, stop spermicides) - Pharm: TMP-SMX or other drug can be used. Use can be daily, post-coital, or intermittent self treatment.

Interstitial Cystitis/Bladder Pain Syndrome

General: Chronic bladder pain and discomfort for > 6 weeks without clear underlying medical cause

Risk: More common in women, psychiatric history

Clinical: Dysuria, increased urinary frequency, dyspareunia, relief with voiding, pelvic pain (with palpation)

Diagnosis: Diagnosis of exclusion. Urinalysis (rule out UTI).

Management:

(1) Behavioral Modification (trigger avoidance)
(2) Pharm: Amitriptyline
(3) Analgesics (Phenazopyridine, Methenamine) for short term relief
(4) Surgical Interventions (bladder hydrodistention)

Complicated UTI/Pyelonephritis

General: Infection of the upper urinary tract (extending past the bladder), most commonly from ascending lower urinary tract infection

Micro: *E. coli*, enterobacteriaceae (*Proteus, Klebsiella*), other gram negative (*Pseudomonas*), *Enterococcus*, fungi (*Candida*)

Clinical:

- UTI symptoms, plus systemic signs (fever, chills, flank pain), CVA tenderness
- Urinalysis (pyuria/bacteriuria, WBC casts)
- Gram stain and culture often positive

Diagnosis:

- Clinical diagnosis (systemic symptoms plus pyuria and bacteriuria)
- Imaging (CT) reserved for cases when patient is not improving

Management:

	General	Empiric Management
Outpatient	- Young, otherwise healthy patients can receive ER care with close follow up	- Fluoroquinolone (Ciprofloxacin) OR - IM Dose of Ceftriaxone plus TMP-SMX, Amox-Clav, or 3rd gen PO cephalosporin
Inpatient	- Septic/critically ill patients - Urinary hardware/obstruction	- Ceftriaxone, fluoroquinolone - Carbapenem (if risk factor for MDR organism)

Complications:

Renal/Perinephric Abscess

- Walled off cavity of necrosis. Can be
  - (1) Renal or
  - (2) Perinephric (perirenal fat to Gerota's fascia)
- Generally occurs as a complication of pyelo, but can be due to seeding
- Dx: CT or US
- Tx: Antibiotics +/- percutaneous drainage (> 5 cm renal and all perinephric)

Chronic Pyelonephritis

- Chronic interstitial disease due to recurrent/chronic infection
- Causes: Vesicoureteral reflux, chronic urinary obstruction (ie stone)

Xanthogranulomatous pyelonephritis

- Subtype of chronic pyelonephritis (generally from obstructive stone)
- Massive kidney damage from granulomatous inflammation and foamy macrophages

UTI

CASE1

You are called to see a 19-year-old woman in complaining of a 2-day history of frequency, dysuria and urgency. She has a temperature of 39.8°C with some right loin pain.Yesterday she had a rigor. She tells you that this is her first episode of an UTI. She has no vaginal discharge and has never had a history of sexually transmitted diseases

Q1:what is the diagnosis,and most common organism?

Uncomplicated pyelonephritis,E.coli.

Q2:what are the initial investigations for this patient?

Urinanalysis,gram stain+urine culture,CBCwith differential ,RFT.

Q3:what is the treatment and for how long?

Single parenteral dose of ceftriaxone, or of gentamicin, followed by oral fluorquinolones orTMP\SMX for gram-ev and amoxicillin for gram+ev, for 10-14day.

Q4:Mention Prophylactic measures to prevent further UTIs that should be advised.

- A 2 L daily fluid intake.
- Voiding before bedtime and after intercourse.
- Avoidance of spermicidal jellies and bubble baths and other chemicals.
- In bathwater.
- Avoidance of constipation.

CASE2

An 84-year-old woman, a nursing home resident with Alzheimer disease,is brought to the emergency room for agitation and confusion. She is found to be febrile, tachycardic, and hypotensive. Examination shows flat neck veins, clear lung fields, and no cardiac murmur or gallops; her limbs are warm and well perfused.Her hemodynamic status has improved with a fluid bolus. Laboratory examination shows evidence of a urinary tract infection (UTI).

Q1:what is the diagnosis?

Shock, most likely as a consequence of urosepsis.

Q2:what is the initial the management of this patient?

- Intravenous (IV) fluids or vasopressors as necessary. Broad-spectrum antibiotics should be started as soon as possible.

Q3:which investigations should you order?

Urinanalysis,gram stain+urine culture,CBCwith differential ,RFT,blood culture

Q4:which antbiotics could be used for her UTI, and for how long?

IV Ampicillin+gentamicin OR fluorquinolones (cipro or levo)for 2-3weeks

CASE3

a 45 -year -old woman who presents with a 4 -day history of urinary frequency and dysuria. On examination, her temperature is 38 ° C, pulse 90 bpm and BP 125/75 mmHg. She is mildly tender in the right flank and suprapubically but there is no rebound or guarding. This is the third UTI in 9 months.

Q1:what is the most likely dignosis?

complicated UTI

Q2:what are the initial tests for this patients?

urinanalysis,gram stain+urine culture,CBCwith differential ,RFT

Q3:mention three causes for her recurrent UTI.

Diabetes mellitus· Immunosuppression· Pregnancy(or others)

Q4:how would you further investigate her ?

screen for diabetes,KUB X-RAY,US,IVP,CT...etc

Q5:how would you prevent her recurrent UTIs?

- Single dose of TMP/SMX after intercourse or at first signs of symptoms.
- Alternative low-dose prophylactic antibiotics (e.g., TMP/SMX) for 6 months.

Urine analysis:

protein –ve , Glucose +2

RBC 8 cells/uL , leukocytes 25/uL

60 years old male complaining from abdominal pain and dysuria the most likely diagnosis is :

A. UTI

B. Bladder stone

C. Bladder tumor

D. Rapidly progressive GN

E. Tubular necrosis

Secondary HTN (CONN'S)

Case 1

Clinical case scenario

26-year-old female previously healthy, her weight 45kg and use a dighragm as a method of contraception on routine physical exam :

blood pressures was 166/100 mmHg another reading was taken and bp was 158/94

Pulse 72 beat per minute regular ,good volume no radio-radial or radio – femoral delay

Abdominal and chest exam was unremarkable

What to do next for her ?

Consider it as hypertension but you need to confirm that by 2 measurement in the next visit

Note :diagnosis of hypertension require two or more properly measured, seated BP readings On each of two or more office visits.

On the next visit the blood pressure measured twice and still elevated so hypertension is confirmed and because she is a young take it seriously (secondary hypertension).

What investigation to do for her ?

1. investigation for effect of HTN on organs (ecg for LVH ,urinalysis ,fudal exam for retinopathy)
2. Routine screening lab :glucose ,electrolytes ,creatinine,GFR ,total cholestrol ,HDL , TFT)

The previous investigations was normal except for stage 2 hypertensive retinopathy on fundal exam K: 3 meq/l NA :145

What further investigation you will order ?

Serum level of renin , aldosterone , renin / aldosterone ratio , CT SCAN

Result : aldosterone elevated , low plasma renin

so you think of primary hyperaldosteronism(conns or bilateral adrenal hyperplasia).

How to differentiate between them ?

Saline infusion test (its called suppression test) or stimulation test (captopril).

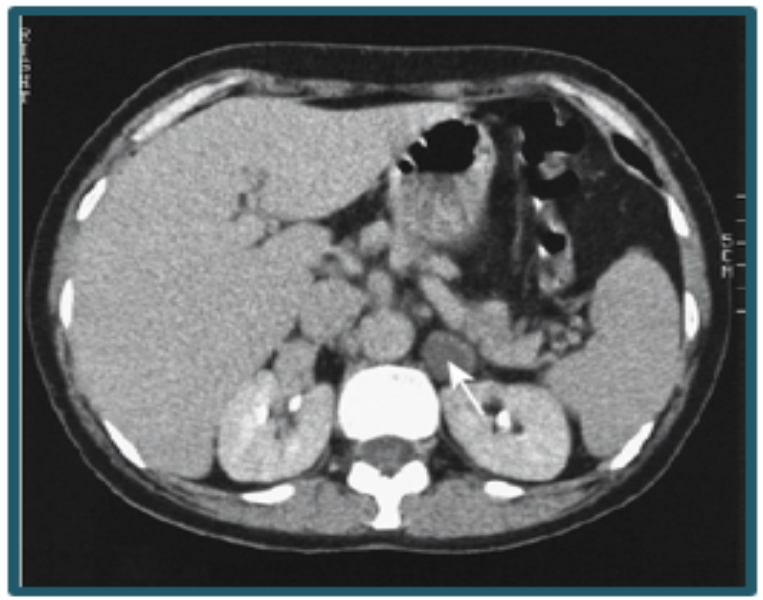
Result of saline suppression :

persistent elevation of aldosterne so its conns (aldosternoe secreting adenoma) order CT SCAN to localize tumor.

Note if the cause of primary hyperaldosteronism is bilateral adrenal hyperplasia saline suppression test will decrease the serum aldosterone while in conns not (because its autonomous secretion).

Q: Result of CT scan

adenoma in left adrenal gland



Q: Treatment for this patient ???

1. Mineralocorticoid receptor anagonist prior to surgery(to correct electrolyte abnormality)
2. Adrenal vein catheterization and adrenalectomy
3. after surgery 70% has persisitent HTN so you should give minralocorticoid receptor antagonist

Notes

**Mineralocorticoid receptor antagonist like :spironolactone ,eplerenone

**adrenal vein cathetarization is important during surgery to confirm site of adenoma right or left because CT may visualize non functioning adenoma as aldosterone secreting adenoma (false positive) .

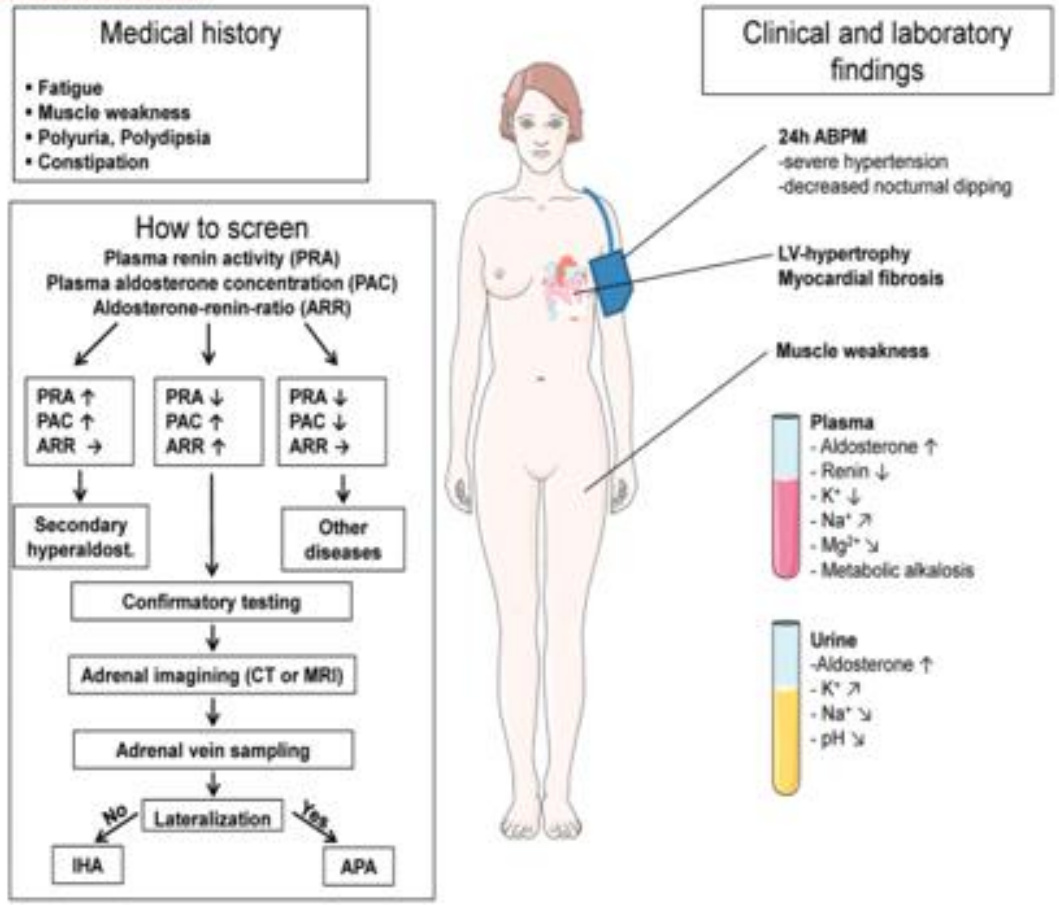
**Dr mdallal said that :in primary hyperaldosteronism potassium not always low it could be at lower normal limit .

**Saline suppression test and stimulation test (captopril ,furesemide) used to differentiate causes of primary hyperaldosteronism is it conns or bilateral adrenal hyperplasia .

If there is decrease in aldosterone after suppression test it means that secretion not autonomous (not conns) so its bilateral adrenal hyperplasia

If the cause of hyperaldosteronism is bilateral adrenal hyperplasia we don't do bilateral adrenalectomy we give only mineralocorticoid receptor antagonist

Figure 5 Medical history, clinical findings, and screening work-up in patients with suspected primary aldosteronism. ...



European Heart Journal, Volume 35, Issue 19, 14 May 2014, Pages 1245–1254,
https://doi.org/10.1093/eurheartj/eht534

New case

- A 15-year-old gypsy girl student with a history of migraines diagnosed 2 years before the current clinical picture, began having symptoms of non-pulsatile frontal headaches 9 months before hospital admission, with worsening symptoms in the last 2 months associated with palpitations. She turned to the emergency room (ER) where she presented AHT (blood pressure (BP) 160/123 mm Hg) for which she was then treated, discharged and referred to her treating physician (without medication).
- On 9 February 2012, she returned to the ER because of worsening headaches and new visual symptoms (blurred vision of the left eye). She was evaluated by ophthalmology, which observed the following changes in visual acuity: right eye 9/10 and left eye 4/10. Funduscopy and angiography revealed small venous occlusions with sparing of the macula. The patient was medicated (eye drops) and referred to ophthalmology, paediatrics and neurology consults, which she failed to attend.
- On 10 February 2012, she turned to the ophthalmology ER because of worsening visual symptoms: bilateral 'cloudy' vision most pronounced in the left eye, presenting aggravated retinopathy with macular detachment and superficial peripapillary haemorrhages. She was referred to a consult and observed 6 days after with worsening visual acuity (right eye: 1/10, left eye with the capability of only counting fingers). Funduscopy with cotton wool spots, haemorrhages and macular oedema were most pronounced in the left eye, without oedema of the optic disc. The patient was transferred to the general ER and referred to Internal Medicine with the diagnosis of hypertensive crisis. The patient denied relevant pathological medical history including smoking, alcohol use or drug consumption, and had had no previous hospital admissions. She was on no regular medication, including oral contraceptives.
- Regarding her family history, the patient's mother was diagnosed with HTN at the age of 38 years and chronic kidney disease. No familial hereditary diseases were known.

Physical exam

Medical examination showed BMI of 17.5 kg/m^2 and no skin lesions. BP was 187/139 mm Hg—overlapping values in all four limbs with no asymmetric pulse. Heart rate was 130 bpm—rhythmic and without heart murmurs, abdomen without murmurs or palpable masses and without oedema. Funduscopy with grade II retinopathy, was without papillary oedema. The remaining neurological examination was normal.

Labs

Blood work with complete blood count, renal function and hepatic enzymes, was normal. Urine was without proteinuria. ECG showed sinus tachycardia, HR of 126 bpm and signs of left ventricular hypertrophy. Chest radiograph and cerebral CT were normal.

The patient was admitted with the diagnosis of severe HTN and retinopathy. She began perfusion of labetalol until BP control was achieved, with progressive clinical improvement, and regression of headaches and visual symptoms.

The clinical picture of recurrent HTN in a young patient made us suspect underlying secondary hypertension.

According to history and physical exam what's the most likely diagnosis ?

Pheochromocytoma

Findings support diagnosis in history and physical exam :

1-episodic attack of HTN

2- sweating

3- tachycardia

What further lab investigation you order to confirm the diagnosis?

- Serum, urinary catecholamines metanephrines
- parathyroid hormone
- Renal u/s
- Thyroid , parathyroid u/s (maybe men syndrome)
- CT scan (chest ,abdomen to exclude extradrenal sites of pheochromocytoma)

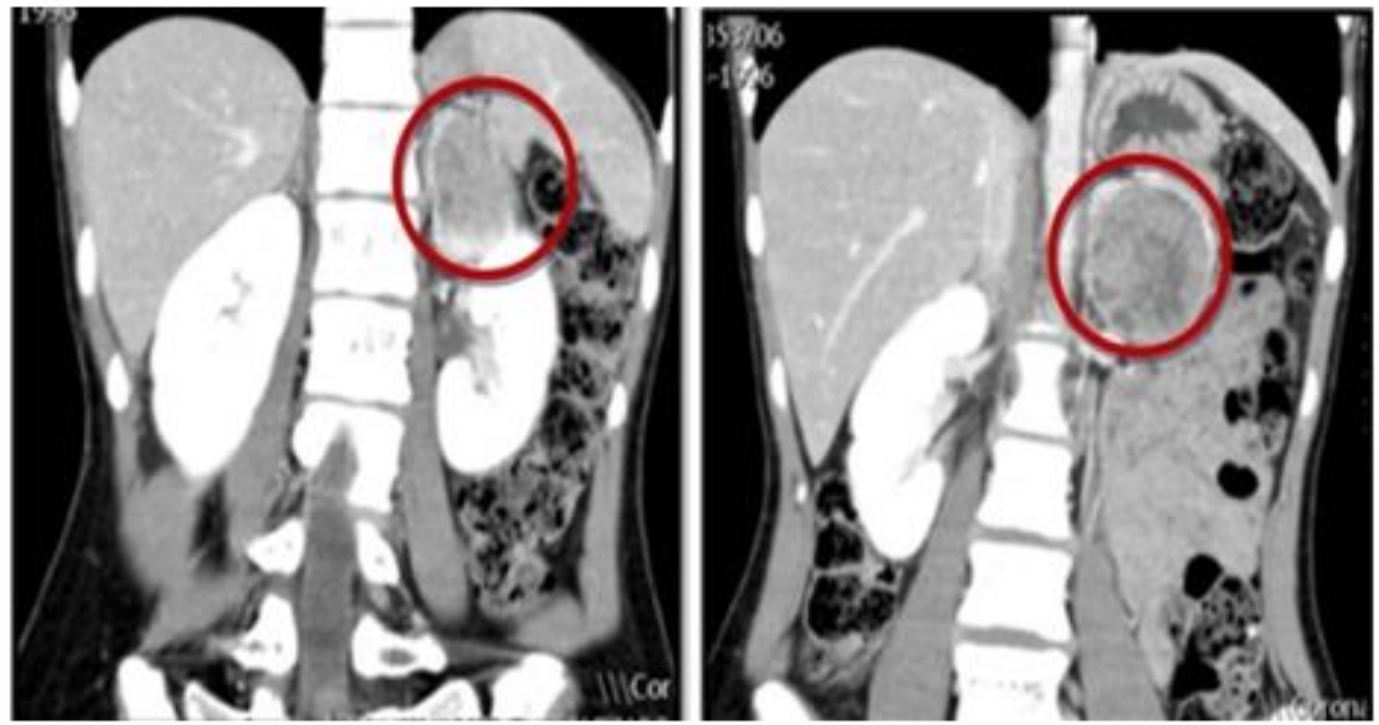
Lab result

Table 1

Laboratory tests

		Ref.
Fasting glucose (mg/dL)	97	74–106
PTHi (pg/mL)	111.5	7.8–53.8
Renin (pg/dL)	16.7	1.1–16.5
Aldosterone (ng/dL)	11.8	5–14.5
Cortisol (µg/dL)	11.8	8.7–25
ACTH (pg/mL)	22.8	<46
Calcitonin (pg/mL)	<2	<5
Phosphorus (mg/dL)	5.5	2.8–4.8
Calcium corrig (mg/dL)	9.8	8.4–10.3
Chromogranin A (nmol/L)	47.7	<6
Serum catecholamines (ng/L)		
Total	22 326	<598
Norepinephrine	20 591	<420
Epinephrine	1318	<84
Urinary metanephrines (µg/24 h)		
Metanephrines	2570	<90
Normetanephrines	>4000	<180

- Renal Doppler ultrasound revealed a solid nodule in left adrenal gland and normal permeability of the renal arteries.
- Abdominal CT scan confirmed the presence of a heterogeneous nodular 6.5×5.3×6.0 cm mass in the left adrenal gland, compatible with a pheochromocytoma

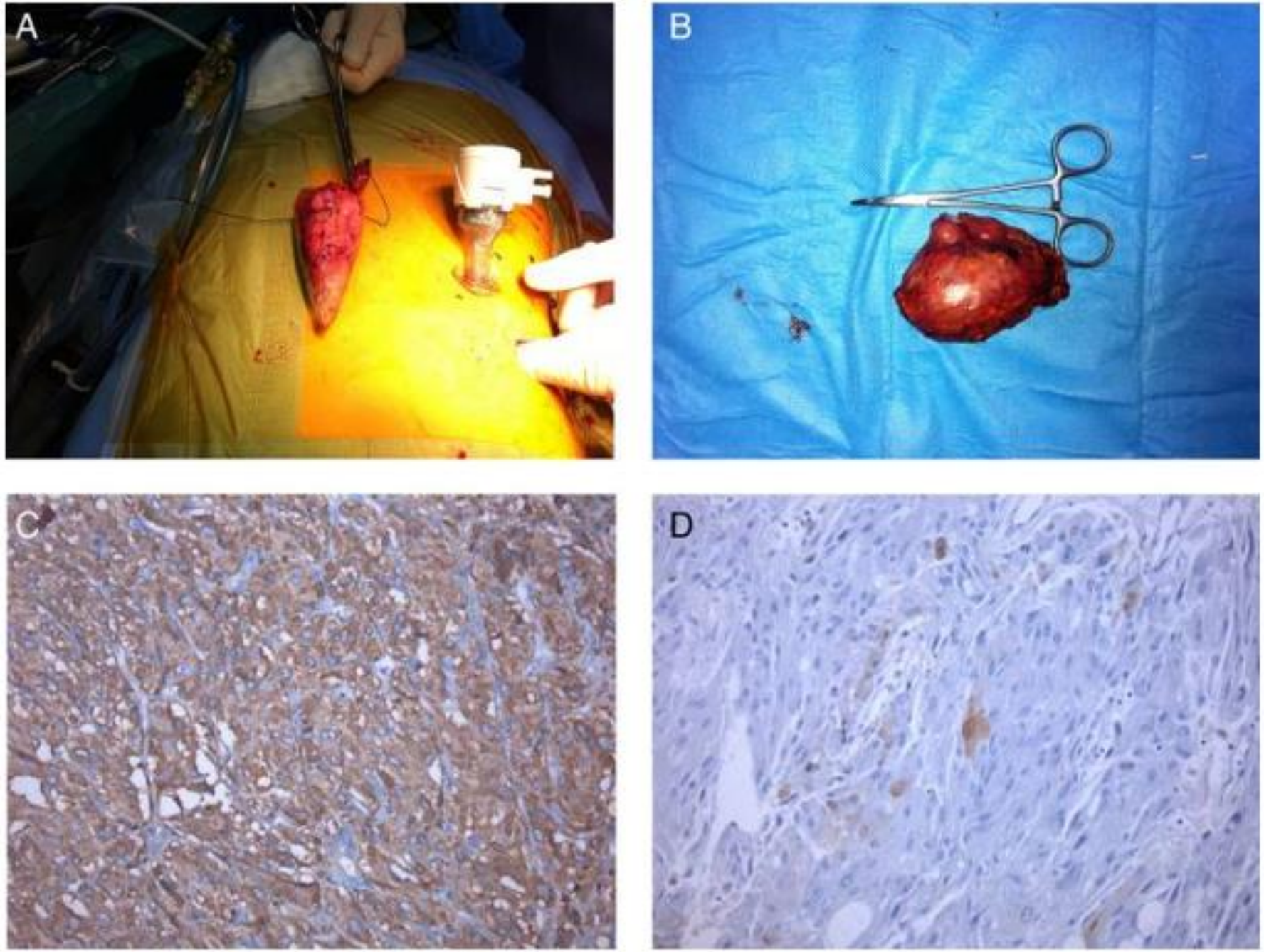


Thyroid and parathyroid ultrasound revealed a small nodular 6×9×4 mm formation posteroinferior to the left lobe, suggestive of a probable parathyroid.

With the diagnoses of pheochromocytoma and associated hyperparathyroidism in a young patient, although no lesions suggestive of thyroid carcinoma existed, possible MEN was admitted, so genetic testing was performed, which revealed a negative RET gene.

Treatment ??

- Therapy with α and posteriorly β-adrenergic blockers was started (intradermal phenoxybenzamine 10 mg and propranolol 20 mg) with adequate BP control.
- The patient was submitted to laparoscopic left adrenalectomy from a retroperitoneal approach on 28 March 2012.
- A 6 cm tumour was removed and the pathological examination confirmed the diagnosis of pheochromocytoma


A
B
C
D

Adult polycystic kidney disease

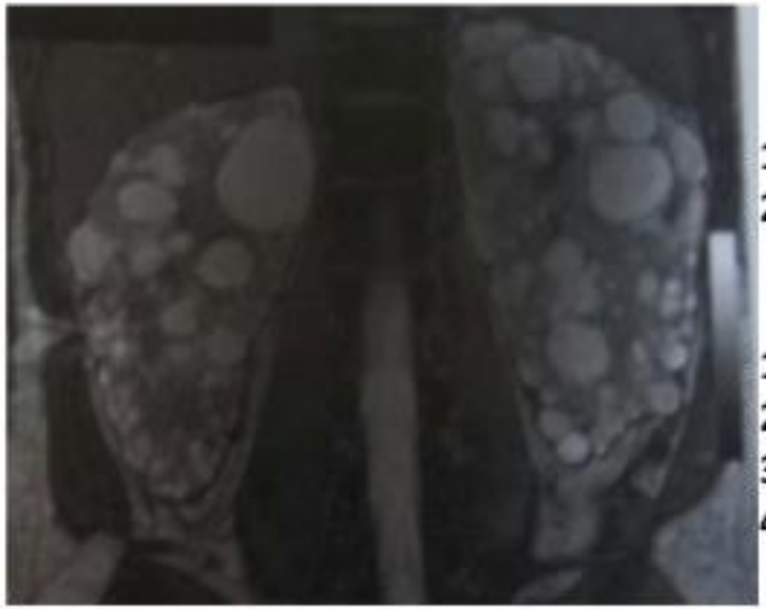
Q21.This abdomen MRI is for a 40 year old man.

1.what is the diagnosis?

Adult Onset Polycystic Kidney Disease

2.what is the neurological complication of this case?

Uremia



- **Autosomal dominant inheritance.**
- **No family history in 40%.**
- **Two gene are identified:**

1. **PKD1 85%.**
2. **PKD2 15%.**

PKD1 Is worse.

- **Complications:**

1. **Hypertension.**
2. **Hemorrhage in the cyst.**
3. **Infected cyst.**
4. **Stones.**

- **Systemic manifestations:**

1. **Cysts in liver (75%), in pancreas.**
2. **Cardiac valve abnormalities like (M.Prolapse/ M.R or T.R).**
3. **Intracranial aneurysms.**

Don't screen unless there is a FHX of aneurysms in the first degree or High risk occupations.

Q1

Q1:whats your Dx?

Polycystic kidney disease

Q2: investigation :

Ultrasound

Q3:pattern of inheritance :

Autosomal Dominant

Others

14-A19 year old female patient is presented to the emergency department with abdominal pain of 1 day duration along with nausea , vomiting and generalized Weakness . Her mother reports 3 days of dysuria and suprapubic pain treated At home by amoxicillin without improvement . The underlying cause of her presentation is most likely ?

a. Extensive hyperosmolarity

b. Constipation

c. Starvation

d. Urinary tract infection

e. **Missed insulin dose**

Physical exam is remarkable for very rapid breathing.

Bp 100/60 pulse 120 SpO_2 99% T36.5
RR 28

pH 7.20 pCO_2 22 HCO_3 11 pO2 93

Na 135 K 4.2 Cr 100

Glucose 40 mmol/L

Q17 : ABG question , the date given with two different units for each parameter , **Note that** we use the Unit mmHg for (PCO2 & PO2) and meq/L for (HCO3-) in the interpretation we used to !

The answer was :

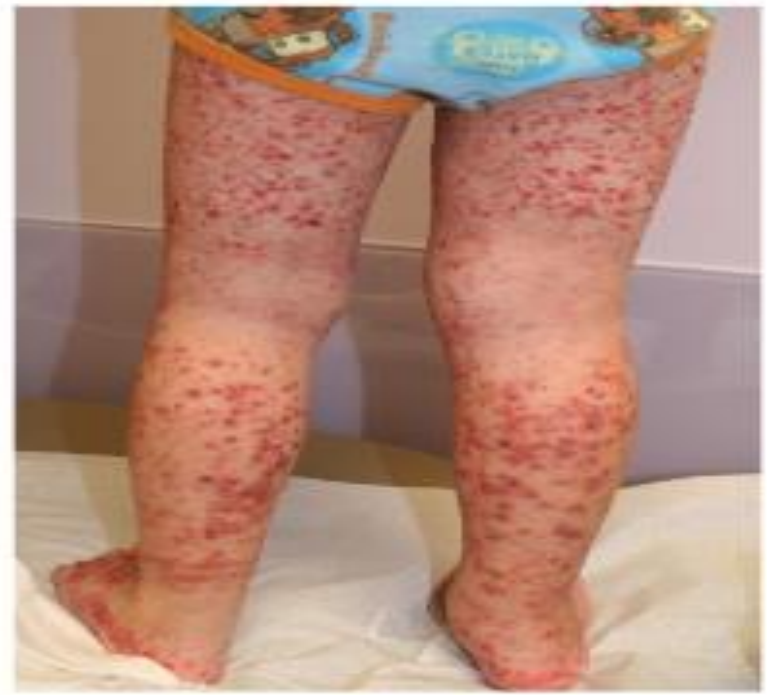
(**partialy compensated respiratory acidosis**)

So : PH ⬇ and PCo2 ⬆ HCO3- ⬆

10) A 12 years old boy.

a) What is your diagnosis? Henoch schonlein purpura

b) What's the major cause of morbidity and mortality in this patient? Renal failure



Thank You