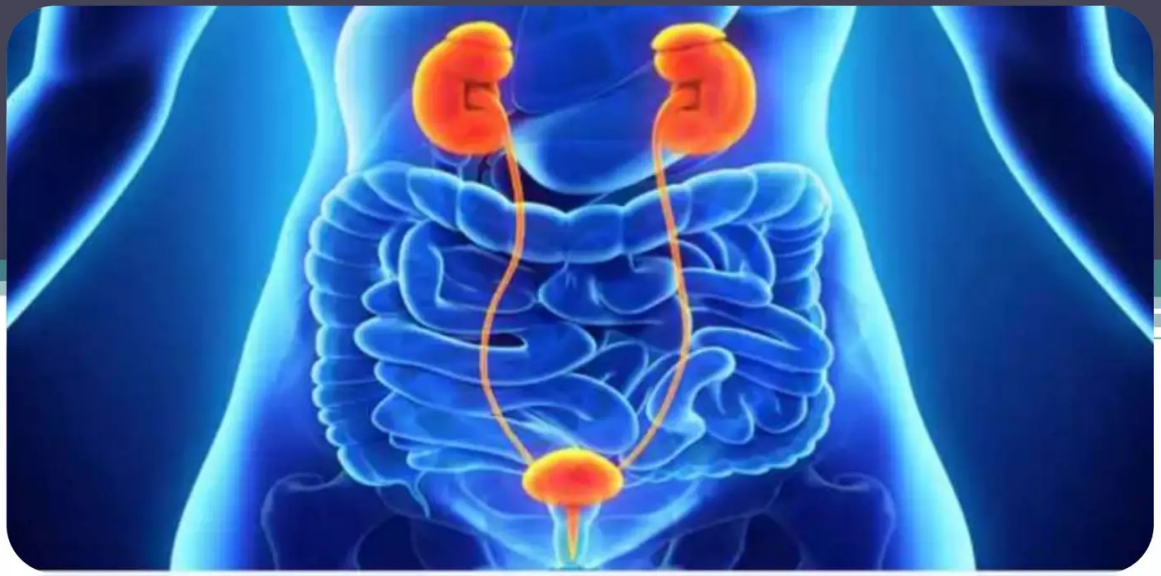
RENAL SYSTEM



Past papers divided by lecturers

Nephrology

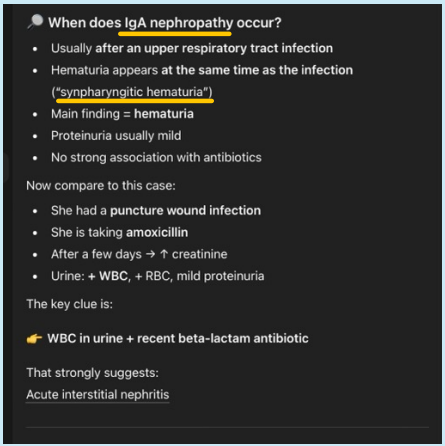
Introduction

21

78) Diabetic female patient was admitted for an infected puncture wound a couple of days ago, she is on amoxicillin, she presented with raised serum creatinine,+1 RBC, +1 WBC protienura,most likely diagnosis ? (Nephrology)

A) ATN
B) Postinfectious IgA nephropathy
C) AIN
D) RTA

Answer is C. AIN (drug induced)



34- A patient with crush injury, developed heme positive, dark urine with no RBCs, most likely:

• Rhabdomyolysis.

Rhabdomyolysis is a condition in which damaged skeletal muscle breaks down rapidly, often due to high intensity exercise over a short period. Symptoms may include muscle pains, weakness, vomiting, and confusion. There may be tea-colored urine or an irregular heartbeat.

45- Patient with lymphoma, known to excrete 1.5 gram/day protein, was found to have -ve dipstick for protein, what's your explanation:

- Dipstick detects only albumin.

(020 QUESTIONS):

56- 76 year old male with diabetes and CAD on multiple drugs including statin with diffuse muscle tenderness and muddy brown cast (uric acid high,high potassium) most likely cause

Ans: Rhabdomyolysis

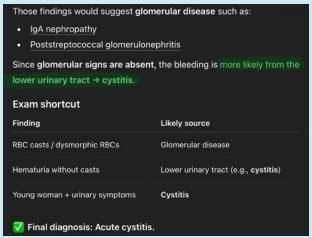
This patient developed ATN secondary to rhabdomyolysis.

?

15- A young female with hematuria, UA+ for blood and proteins (the stem doesn't mention any RBC casts or dysmorphic RBCs), Diagnosis?

• Acute Cystitis.

Acute cystitis is common in young women, presenting with hematuria, pyuria, and mild proteinuria.
The absence of fever, flank pain, or WBC casts makes pyelonephritis less likely.



Glomerulonephritis

77) Which of the following is a poor prognostic sign in IGA nephropathy ? (Nephrology)

A) IgA deposits
B) HTN at the time of diagnosis
C) Serum IgA level
D) Macroscopic hematuria

Answer is B. HTN

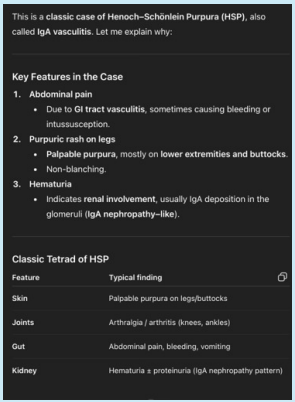
Prognostic Markers at Presentation in IgA Nephropathy

Clinical	Histopathologic
Poor Prognosis	
Hypertension	Mesangial hypercellularity
Renal impairment	Endocapillary proliferation
Severity of proteinuria	Segmental glomerulosclerosis
Hyperuricemia	Tubular atrophy
Gross obesity	Interstitial fibrosis
Duration of preceding symptoms	Capillary loop IgA deposits
Increasing age	Crescents (controversial)
Good Prognosis	
Recurrent macroscopic hematuria	
No Impact on Prognosis	
Gender	Intensity of IgA deposits
Serum IgA level	

35- Wrong about nephrotic syndrome:

• Hypertension.

39- Patient with abdominal pain, purpuric rash on legs and hematuria, diagnosis?



This is a **classic case of Henoch–Schönlein Purpura (HSP)**, also called **IgA vasculitis**. Let me explain why:

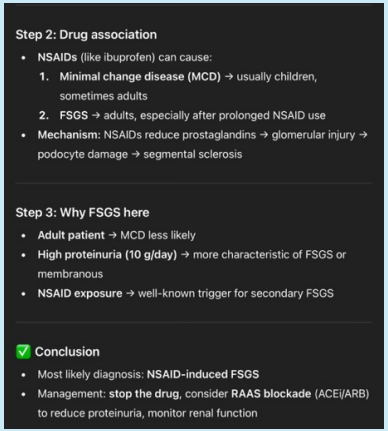
Key Features in the Case

1. **Abdominal pain**
   - Due to **GI tract vasculitis**, sometimes causing bleeding or intussusception.
2. **Purpuric rash on legs**
   - **Palpable purpura**, mostly on **lower extremities and buttocks**.
   - Non-blanching.
3. **Hematuria**
   - Indicates **renal involvement**, usually IgA deposition in the glomeruli (**IgA nephropathy–like**).

Classic Tetrad of HSP

Feature	Typical finding
Skin	Palpable purpura on legs/buttocks
Joints	Arthralgia / arthritis (knees, ankles)
Gut	Abdominal pain, bleeding, vomiting
Kidney	Hematuria ± proteinuria (IgA nephropathy pattern)

59- young lady took ibuprofen for shoulder pain for 1 month presents with nephrotic range proteinuria (it was very high 10g/day)
Ans: FSGS mostly



Step 2: Drug association

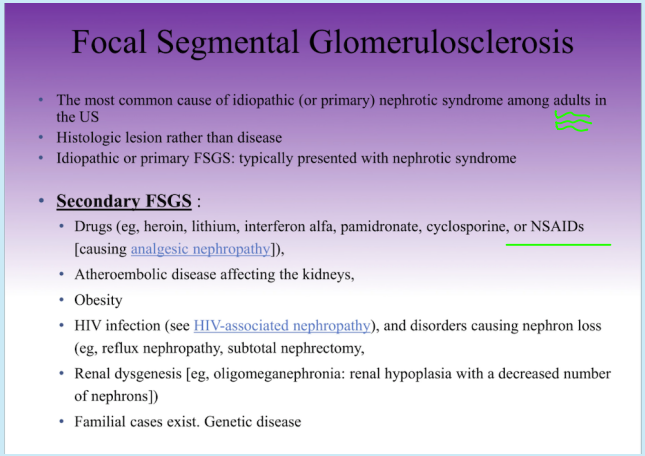
- **NSAIDs** (like ibuprofen) can cause:
  1. **Minimal change disease (MCD)** → usually children, sometimes adults
  2. **FSGS** → adults, especially after prolonged NSAID use
- **Mechanism:** NSAIDs reduce prostaglandins → glomerular injury → podocyte damage → segmental sclerosis

Step 3: Why FSGS here

- **Adult patient** → MCD less likely
- **High proteinuria (10 g/day)** → more characteristic of FSGS or membranous
- **NSAID exposure** → well-known trigger for secondary FSGS

✅ **Conclusion**

- Most likely diagnosis: **NSAID-induced FSGS**
- Management: **stop the drug**, consider **RAAS blockade** (ACEi/ARB) to reduce proteinuria, monitor renal function



Focal Segmental Glomerulosclerosis

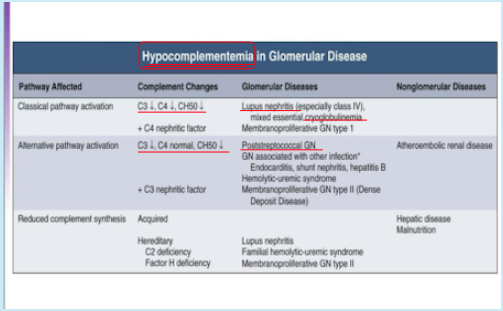
- The most common cause of idiopathic (or primary) nephrotic syndrome among adults in the US
- Histologic lesion rather than disease
- Idiopathic or primary FSGS: typically presented with nephrotic syndrome

- **Secondary FSGS** :
  - Drugs (eg, heroin, lithium, interferon alfa, pamidronate, cyclosporine, or NSAIDs [causing analgesic nephropathy]),
  - Atheroembolic disease affecting the kidneys,
  - Obesity
  - HIV infection (see HIV-associated nephropathy), and disorders causing nephron loss (eg, reflux nephropathy, subtotal nephrectomy,
  - Renal dysgenesis [eg, oligomeganephronia: renal hypoplasia with a decreased number of nephrons])
  - Familial cases exist. Genetic disease

4-Which of the following doesn't have low complement?
a) SLE nephritis
b) IgA nephropathy
c) Post streptococcal glomerulonephritis
d) Cryoglobulinemia

ANSWER: B

- Among the listed conditions, IgA nephropathy doesn't feature low serum compliment levels.
- SLE is associated with low complement levels specially C3 & C4 due to formation of immune complexes and complement consumption.
- Post streptococcal GN is chahracterized by low C3 levels in the acute phase.
- Cryoglobulinemia involves immune complex formation and consumption of complent leading to low C4 and sometimes low C3.



Hypocomplementemia in Glomerular Disease

Pathway Affected	Complement Changes	Glomerular Diseases	Nonglomerular Diseases
Classical pathway activation	C3 ↓, C4 ↓, CH50 ↓	Lupus nephritis (especially class IV), mixed essential cryoglobulinemia	
	+ C4 nephritic factor	Membranoproliferative GN type 1	
Alternative pathway activation	C3 ↓, C4 normal, CH50 ↓	Poststreptococcal GN GN associated with other infection* Endocarditis, shunt nephritis, hepatitis B Hemolytic-uremic syndrome Membranoproliferative GN type II (Dense Deposit Disease)	Atheroembolic renal disease
	+ C3 nephritic factor		
Reduced complement synthesis	Acquired		Hepatic disease Malnutrition
	Hereditary C2 deficiency Factor H deficiency	Lupus nephritis Familial hemolytic-uremic syndrome Membranoproliferative GN type II	

16- A very long case describing a nephrotic syndrome (edema, hypercholestolemia, hypoalbuminemia ... etc.), Diagnosis?

• Membranous nephropathy (all the others were nephritic diseases).

The case is describing a nephrotic syndrome and since all the other options where nephritic membranous nephropathy is the answer. However, MCD is considered the m.c cause of nephrotic syndrome in children, FSGS in adults especially in African American populations, membranous nephropathy in adults of European middle eastern or North African descent.

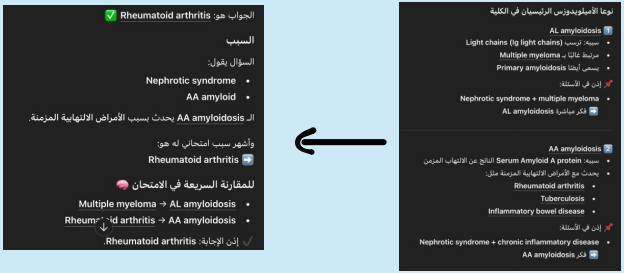
17- Patient with nephrotic syndrome and AA amyloid, most likely diagnosis?

• Rheumatoid arthritis (MM causes AL amyloid).

There are Two major types of amyloidosis affecting the kidney:

AL amyloidosis – due to light chain deposition, associated with multiple myeloma.

AA amyloidosis – caused by chronic inflammatory diseases, such as rheumatoid arthritis, tuberculosis, and inflammatory bowel disease.



8- Patient with history of cellulitis of 3 weeks, took cephalosporins. Developed SOB, bilateral lower limb edema, fever. Elevated Cr with 1-2 RBCs. Cause :
a) interstitial nephritis
b) post strep GN
c) MCD

ANSWER: B

The history of cellulitis suggests a bacterial infection, most likely caused by Streptococcus.
PSGN typically occurs 1-3 weeks after a streptococcal infection (e.g., pharyngitis or skin infection like cellulitis).

Why not the other options?
A) Interstitial Nephritis: Typically caused by drug hypersensitivity reactions (e.g., antibiotics like cephalosporins), but it presents with rash, eosinophilia, and eosinophiluria, which are absent in this case.
C) Minimal Change Disease (MCD): Presents with nephrotic syndrome (massive proteinuria, hypoalbuminemia, and severe edema) rather than hematuria and renal dysfunction seen here

12- Nephritic syndrome is associated with all of the following except:
a) hematuria
b) HTN
c) renal failure
d) edema
e) hypoalbuminemia

ANSWER: E

Nephritic syndrome is characterized by:

Hematuria (blood in urine), Hypertension (HTN), Renal failure (due to glomerular inflammation), Edema (due to sodium and water retention).
However, hypoalbuminemia is a hallmark of nephrotic syndrome, not nephritic syndrome. In nephrotic syndrome, massive proteinuria leads to a significant loss of albumin, whereas in nephritic syndrome, proteinuria is present but not as severe.

61- ptn with history of Lung CA and findings of nephrotic syndrome

Ans: Membranous nephropathy

14- Patient treated with gold for 5 years, RA for 30 years presented with nephrotic syndrome, most likely Dx?
a) Renal amyloid
b) Gold induced membranous nephropathy

ANSWER: B

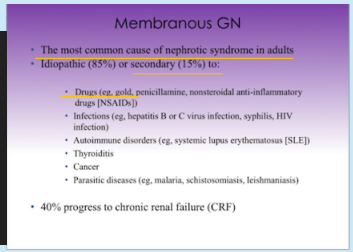
Nephropathy with proteinuria is an occasional complication of gold therapy for rheumatoid arthritis and is considered to be due to an immune hypersensitivity reaction.

3 قاعدة امتحانية سريعة 🧠

احفظ هذه العلاقات لأنها تتكرر:

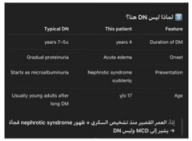
- Gold / Penicillamine / NSAIDs → Membranous nephropathy
- RA الطويل أو الأمراض الالتهابية المزمنة → Amyloidosis

لكن عندما يذكر الدواء صراحة غالبًا يريد drug-induced MN.



28- 17 years old with lower limb swelling and proteinuria, DM1 since 4 years, controlled. Most likely diagnosis:

• Minimal change disease.



29- A pt with colonic cancer, developed proteinuria and hematuria, mostly:

• Membranous glomerulonephritis.

Membranous nephropathy is the most common glomerular pathology associated with solid tumors.

50- A patient with hemoptysis, nasal mucosal ulcer, recently became oliguric, ANCA +ve:

- Wegener's granulomatosis.

31- A patient with thigh abscess, treated, developed hematuria, proteinuria, positive urine WBCs, RBCs and eosinophils, mostly:

• Drug-induced interstitial nephritis.

Drug-induced acute interstitial nephritis (DI-AIN), also called allergic acute interstitial nephritis, is the most common cause of acute interstitial nephritis (AIN) in developed countries. DI-AIN is associated with a sharp decline in renal function, often requiring dialysis, and has the potential for permanent renal insufficiency, especially if left untreated.

Post-strep GN	Drug-induced AIN	Feature
No specific drug	Yes (antibiotics)	Recent drug exposure
Cola-colored, RBC casts	Mild	Hematuria
Mild to moderate	Mild	Proteinuria
Usually absent	+	Urine WBCs
-	(hallmark) +	Urine eosinophils
Often elevated	Usually normal	BP
Common	Usually minimal	Edema
weeks post infection 3–1	Days after drug	Timing

eosinophils in urine + recent antibiotics → Drug-induced AIN :هنا
✅

38- Case of hematuria, all investigations and U/S normal, next step?

- Doppler ultrasound/ renal biopsy.

NA Disorders

6- Patient with polydipsia and polyuria and nocturia .low urine osmolarity
with no renal disease in his family history (Signs and symptoms of D.I)
what is the next step:
a) Desmopressin administration
b) Water deprivation test
c) Administer Amiloride

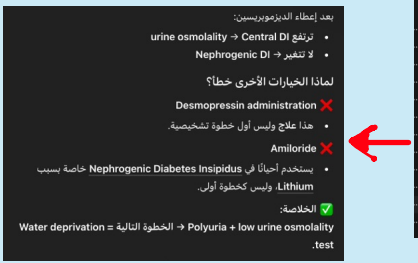
ANSWER: B

The **water deprivation test** is the gold standard for diagnosing DI. It determines whether the kidneys are capable of concentrating urine and helps differentiate between DI and primary polydipsia. It's used to determine if the body can concentrate urine in response to dehydration.

Why not the other options?

A) Desmopressin should only be given after confirming DI through water deprivation, as it is used to differentiate between central vs. nephrogenic DI.
Administering desmopressin too early could mask the true underlying issue.

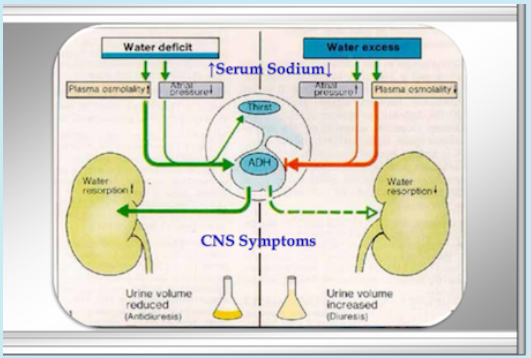
C) Amiloride is used in lithium-induced nephrogenic DI, but at this stage, we don't know if the patient even has nephrogenic DI. The first step should always be to diagnose the type of DI before treating.



19- A patient is hypovolemic (coming from a marathon), which of the following is unlikely?

• Urine osmolality less than 300 mOsm.

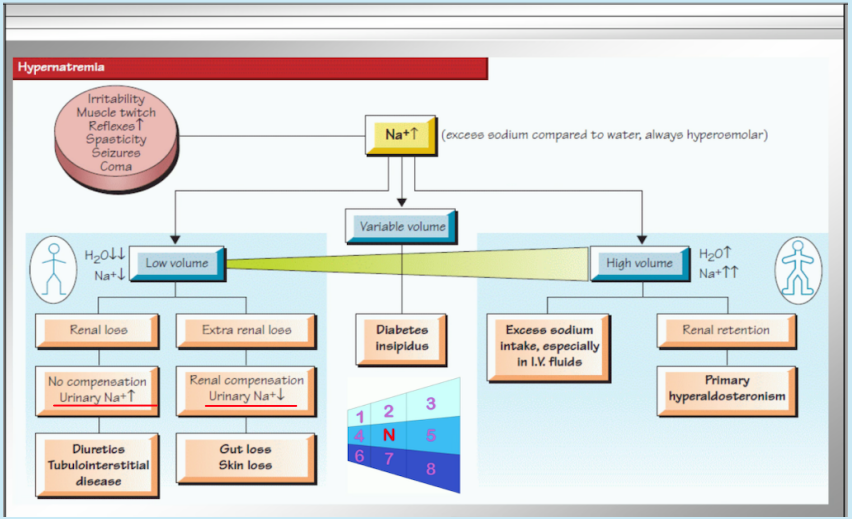
After a marathon, the patient is dehydrated and hypovolemic due to excessive sweating and fluid loss.
In response, the body activates antidiuretic hormone (ADH), which increases water reabsorption in the kidneys, making urine more concentrated (high osmolality).
The renin-angiotensin-aldosterone system (RAAS) is also activated, increasing sodium and water retention, further concentrating urine.
- A urine osmolality < 300 mOsm/kg suggests dilute urine, which is characteristic of excess hydration (e.g., primary polydipsia, diabetes insipidus).
- In hypovolemia, the kidney conserves water, leading to a high urine osmolality (> 450–500 mOsm/kg).



Orthostatic hypotension.

48- Female patient was running in a marathon and came later in the day (mainly with signs of dehydration), you expect to find all the following except:

60-schizophrenic ptn with hypernatremia with low urine Na and osmolarity
Ans: Diabetes insipidus



Patient profile:
Schizophrenic patient (often psychogenic polydipsia is considered in psychotic patients, but let's see labs)
Hypernatremia
Low urine sodium
Low urine osmolarity
Step 1: Think about hypernatremia causes
Hypernatremia occurs when water loss exceeds sodium loss, or rarely sodium gain.
Key lab clues:
Low urine sodium → kidneys are trying to retain sodium (not losing it)
Low urine osmolarity → urine is very dilute, cannot concentrate urine
This combination tells us:
Kidneys are unable to concentrate urine → water is lost freely → hypernatremia develops.
Step 2: Differential
Central Diabetes Insipidus (CDI)
ADH deficiency → cannot concentrate urine
Labs: hypernatremia, low urine osmolarity, low urine sodium
Nephrogenic Diabetes Insipidus (NDI)
Kidneys resistant to ADH → similar labs, often history of lithium or CKD
Psychogenic polydipsia
Usually hyponatremia (dilutional), not hypernatremia
Urine is very dilute, but patient drinks a lot → prevents hypernatremia
Step 3: Why Diabetes Insipidus
Hypernatremia despite low urine sodium and dilute urine = water loss due to ADH deficiency
In a psychotic patient, this can be central (ADH deficiency) or rarely nephrogenic
Key differentiator: psychogenic polydipsia usually causes low sodium, not high
✅ Conclusion
Hypernatremia + low urine osmolarity + low urine sodium → Diabetes Insipidus
Next step: water deprivation test ± desmopressin to distinguish central vs nephrogenic DI

K Disorders

21

50) A patient who is hyperkalemic for some reason, right order of intervention : (Nephrology)

A) Calcium gluconate – bicarbonate- insulin/glucose
B) Insulin/glucose – bicarbonate
C) Calcium gluconate – insulin/ glucose- bicarbonate
D) Dialysis

Answer is C

52) pH 7.31, potassium 6, high calcium, low bicarbonates, acidic urine (nephrology)

A) Distal tubular acidosis

B) RTA IV

C) RTA II

D) CKD

E) Proximal renal acidosis

Answer is B. RTA IV

Feature	Type 1 RTA(Distal)	Type 2 RTA(Proximal)	Type 4 RTA(Hyperkalemic)	CKD(Chronic Kidney Disease)
Site of defect	Distal tubule	Proximal tubule	Collecting duct / aldosterone	Global nephron loss
Primary defect	↓ H^+ secretion	↓ HCO_3^- reabsorption	↓ Aldosterone or resistance	↓ GFR, ↓ waste excretion
Serum K^+	↓ (Hypokalemia)	↓ (Hypokalemia)	↑ (Hyperkalemia)	↑ or ↓ depending on stage/meds
Urine pH	> 5.5	< 5.5	< 5.5	Usually < 5.5 (acidified unless late)
Serum HCO_3^-	↓	↓↓↓	↓	↓ (as GFR falls)
Urine anion gap	Positive	Variable	Positive	Often positive (late stage)
Nephrolithiasis	Common	Rare	Rare	Possible (esp. with hyperparathyroidism)
GFR	Normal	Normal	Normal to mildly ↓	↓↓↓ progressively
Creatinine	Normal	Normal	Normal to slightly ↑	↑ chronically
				Diabetes, hypertension, GN, aging

21

96) Female psychogenic came to ER, fatigue, looks obese, psychiatric issues, BP 105/??, ...pH: 7.48 , hypokalemia 3.1, mg 1.6 , Cl ~40, urine k ~100, ... High urine Calcium (nephrology)

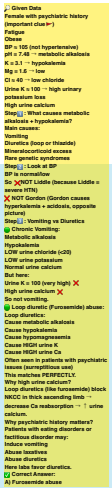
A) Furosemide abuse

B) Chronic vomiting

C) Liddle

D) Gordon syndrome

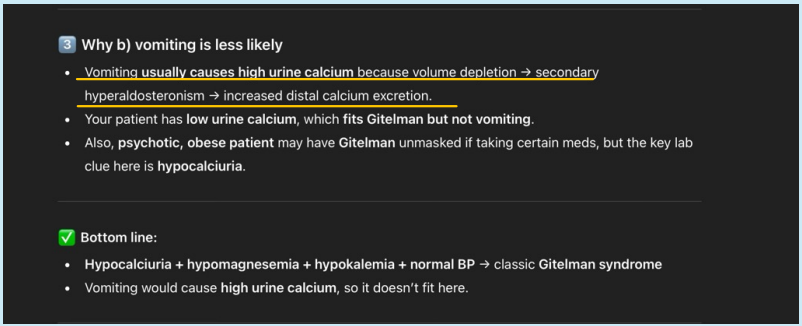
Answer is A. Furosemide abuse



11- Obese psychotic patient with Low Ca in urine, hypomagnesemia, no HTN, hypokalemia:
a) gitelman's
b) Excessive vomiting

ANSWER: A

Gitelman syndrome is an autosomal recessive kidney tubule disorder characterized by low blood levels of potassium and magnesium, decreased excretion of calcium in the urine, and elevated blood pH. It is the most frequent hereditary salt-losing tubulopathy.



57- Psychotic patient with hyponatremia,hypokalemia,normal urinary potassium and hypocalciuria

Ans: Thiazide abuse

Psychotic patient → may have access to or abuse medications

Labs:

Hyponatremia

Hypokalemia

Normal urinary potassium

Hypocalciuria

Step 1: Think "who causes hyponatremia + hypokalemia"

Diuretics are a top cause.

5- Patient with CKD, DM, HTN. on B blockers ACEI and statin. Blood glucose >240, k+=7. CPK =300. Which one of the following doesn't contribute to hyperkalemia in her condition?

a) Beta blockers
b) CKD
c) Use of ACEI
d) Hyperglycemia
e) Rhabdomyolysis (although statins cause rhabdomyolyis, but in this case the rise in CPK is not in range of frank rhabdomyolysis which should be in thousands).

ANSWER: E

- Rhabdomyolysis Would cause a marked increase in serum potassium due to massive release from damaged muscle cells if it were severe. However, CPK (Creatine Phosphokinase) in rhabdomyolysis is in the thousands or even higher (e.g., >5,000).Here, CPK = 300, which is not indicative of significant muscle breakdown.Mildly elevated or near-normal CPK does not contribute meaningfully to hyperkalemia in this scenario.

Why not the other options?

A) Blocking β2-adrenergic receptors that normally promote potassium uptake into cells results in increased serum K^+ (contributes to hyperkalemia).
B) Reduced kidney function impairs potassium excretion and results in increased serum K^+ (contributes to hyperkalemia).
C) ACE Inhibitors (ACEI) decrease aldosterone production. Ending up in reduced renal potassium excretion (contributes to hyperkalemia).
D) Hyperglycemia is often associated with insulin deficiency or reduced insulin effect.
Insulin normally drives potassium into cells.
Result: Less K^+ uptake into cells → higher serum K^+ (contributes to hyperkalemia).

18- In vomiting, what's the mechanism of hypokalemia?

- Loss of potassium in urine.

Vomiting leads to loss of gastric acid (HCl), resulting in metabolic alkalosis.
This triggers the kidneys to excrete bicarbonate, leading to increased urinary potassium loss.
Additionally, dehydration from vomiting activates the renin-angiotensin-aldosterone system (RAAS), which increases sodium reabsorption at the expense of potassium and hydrogen ion excretion in urine.

64- ptn with diabetes and CAD who takes amlodipine and metoprolol,presented with acidosis and hyperkalemia,urinary pH 5.7
Ans: Type IV RTA

Step 1: Recognize the acidosis type
The patient has acidosis + hyperkalemia.
First, determine if it's high anion gap (HAGMA) or normal anion gap (NAGMA / non-anion gap acidosis).
High anion gap acidosis → MUDPILES (e.g., ketoacidosis, lactic acidosis, toxins)
Normal anion gap acidosis → usually renal or GI bicarbonate loss
In this patient:
Likely non-anion gap acidosis → points away from DKA or lactic acidosis as the primary cause.
Step 2: Check renal contribution
Acidosis can be renal (RTA) or extra-renal (GI losses like diarrhea).
****Clues suggesting renal cause (RTA):**
Hyperkalemia → unlike diarrhea-induced acidosis, which usually causes hypokalemia.
Urine pH → 5.7, not alkalotic. Type IV RTA can acidify urine mildly despite acidosis, but the kidney is still the source of the problem.
Step 3: Identify type of RTA
There are 3 classic RTAs that cause NAGMA:
Type I (distal) → hypoaldosteronism not typical, usually hypokalemia, urine pH > 5.5
Type II (proximal) → hypokalemia, urine pH can be <5.5 once bicarbonate wasting stabilizes
Type IV (hyperkalemic) → hyperkalemia, often in diabetics, sometimes due to drugs (ACEi, ARBs, β-blockers slightly).
This patient fits Type IV because:
Hyperkalemia ✅
Mild non-anion gap acidosis ✅
Diabetic ✅
Urine pH mildly acidic (compatible) ✅

26- Not associated with hypokalemia:

• Addison's disease.

Addison's disease (primary adrenal insufficiency) typically causes hyperkalemia due to low aldosterone, which reduces renal K+ excretion.

23- A case of AML and hyperkalemia and the patient is not on treatment, which one of them can be the cause?

• Shift from intracellular to extracellular (spontaneous tumor lysis syndrome).

AML has a high tumor burden, and even without chemotherapy, spontaneous TLS can occur, releasing potassium, phosphate, and uric acid into the bloodstream.

احتمال آخر حسب صيغة السؤال

في مريض عنده **Acute Myeloid Leukemia (AML)** وظهر عنده **Hyperkalemia** وهو لم يبدأ العلاج بعد، أهم سبب نفكر فيه عادة هو:

1️⃣ **Pseudohyperkalemia (الأكثر شيوعًا في AML)**

- في AML يكون عدد الـ **WBC مرتفع جدًا**.
- أثناء **سحب الدم أو تخزين العينة** قد تتكسر الخلايا البيضاء.
- تكسر الخلايا يطلق **البوتاسيوم داخل الأنبوب** → فيظهر في التحليل **مرتفع كاذب**.

📌 لذلك النتيجة في المختبر مرتفعة، لكن **البوتاسيوم الحقيقي في جسم المريض طبيعي**.

دلائل تساعدك في السؤال:

- WBC عالي جدًا (leukocytosis).
- المريض **لا توجد لديه أعراض hyperkalemia**.
- ECG طبيعي.

إذا تتكلم عن **AML قبل أي علاج**:

- **Pseudo-hyperkalemia** → **الأكثر شيوعًا**، خصومنا إذا WBC عالي جدًا (100,000s).
  - السبب: تكسر خلايا الدم البيضاء أثناء سحب أو معالجة العينة.
  - المريض عادة **لا أعراض**، ECG طبيعي، البوتاسيوم في الدم الحقيقي طبيعي.
- **Spontaneous TLS** → أقل شيوعًا قبل العلاج، لكنه ممكن ويحدث خصومًا في **AML مع خلايا عالية الانقسام أو Burkitt lymphoma**.
  - المريض قد يكون عنده **أعراض Hyperkalemia حقيقية**، ↑ uric acid، ↓ Ca، ↑ phosphate، وAKI أحيانًا.

💡 **خلاصة الامتحان:**

- شوف **WBC وعدد الخلايا** → إذا عالي جدًا، وفحصك طبيعي → غالبًا **pseudo**.
- شوف **أعراض حقيقية، تغيّر في uric acid/phosphate، ↑ ECG، ↑ ارفع TLS** في ذهنك.

Tumor Lysis Syndrome (TLS) يمكن أن يحدث حتى **قبل بدء العلاج**، ويسمى:

Spontaneous Tumor Lysis Syndrome.

لماذا يحدث في AML؟

في **Acute Myeloid Leukemia** يكون هناك:

- **عدد كبير جدًا من الخلايا السرطانية**
- معدل تكاثر وموت خلايا سريع

أحيانًا **الخلايا تتكسر تلقائيًا** بدون علاج، فيحدث تحللها داخل الدم.

ماذا يحدث عند تحلل الخلايا؟

عند تحطم الخلايا تطلق محتوياتها إلى الدم:

- **Potassium** → **Hyperkalemia** ↑
- Phosphate ↑
- Uric acid ↑
- Calcium ↓ (لأن الفوسفات يرتبط بالكالسيوم)

القاعدة الامتحانية

إذا جاء:

- **Leukemia / lymphoma**
- **Hyperkalemia**
- **High uric acid / AKI**

فكر مباشرة في TLS حتى لو المريض لم يبدأ العلاج.

خلاصة السؤال

المريض:

- AML
- Hyperkalemia

24- AKI and hyperkalemia, least likely cause:

• Vomiting.

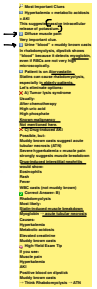
Vomiting primarily leads to hypokalemia, not hyperkalemia, because of loss of gastric acid and subsequent metabolic alkalosis.

AKI

21

51) A 70 years old patient who is known to have epilepsy and HTN comes to the ER with reduced consciousness, hypertension and diffuse hand and legs pain, he's on Carbamezebiene, propnalol, valsartan, captopril,aterovastatin, Ka 7, bicarb was low I think, creatinene 3, urine 1ml/kg/h, ++ Protein in urine, +++ blood in urine, 20 WBC/powerfield in urine. granular muddy brown casts in urine What is the most likely diagnosis (nephrology)

A) tumor lysis syndrome
B) Rhabdomyolisis
C) Drug induced acute kidney injury

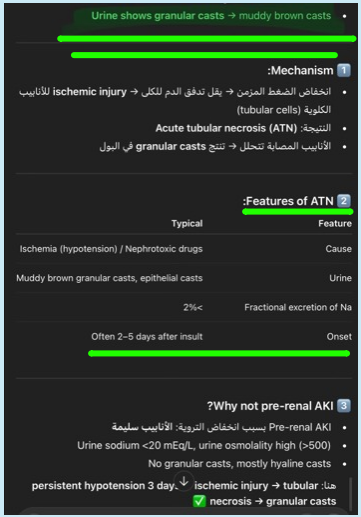


Answer is B Rhabdomyolysis

hands and legs pain, signs of ATN, hyperkalemia, all signs of severe rhabdomyolysis, the combination of Valsartan and Carbamezebiene -two myopathic drugs- supports the diagnosis, why not A ? There is no indication of malignancy or chemotherapy or Uric acid levels.

30- A patient with MI, persistent hypotension for 3 days, developed AKI with granular deposits in urine, mostly:

• Acute tubular necrosis.



36- True about kidney blood supply:

NSAIDs cause constriction of the afferent arteriole.

47- A patient with cholecystectomy, which issupportive for pre-renal failure:

• Orthostatic hypotension.

4️⃣ **Why post-cholecystectomy?**

- Surgery → **blood loss, fasting, third-spacing** → hypovolemia
- Orthostatic hypotension is a clinical sign of **low intravascular volume** → pre-renal AKI

✅ **Bottom line:**

> **Orthostatic hypotension in a post-surgical patient strongly supports pre-renal AKI due to volume depletion.**

CKD

62- CKD patient with hip fracture likely cause of hypocalcemia:

Ans: Decreased vit d hydroxylation

65- True about CKD:
A. 90% of patients with late stage CKD have anemia
B. Anemia causes significant morbidity and mortality in CKD patients
C. CKD can rarely cause Iron deficiency
D. Criteria for Iron deficiency in patients with CKD is similar to normal patients
E. erythropoietin secretion is not controlled by hypoxia inducible factor
Ans:A ?

ABG

21

70) Female 24, Type one diabetes , sweaty, unconscious,acidosis severe low bicarb, glucose 500, anion gap 22, best next step:

A) Measure ketone bodies in urine

B) Measure delta gap

C) Measure serum lactate

Answer is B. Measure delta gap

The patient has classic symptoms of DKA, delta gap helps in determining if there is mixed acid base disorder, A. Is good for diagnosis however not management

ph 7.3, pCO2 38 ,HCO3- 13 .?..

Ans: HAGMA with respiratory acidosis

ABGs and electrolytes results
[PCO2=38, pH=7.12, HCO3- =12] dx?

High anion gap metabolic acidosis
and respiratory acidosis

20- A case of DKA and hypoventilation (ABGs given)

• HAGMA + respiratory acidosis (after you calculate it for sure – don't depend on signs and symptoms mentioned in the stem).

HAGMA (High Anion Gap Metabolic Acidosis): DKA leads to a high anion gap due to the accumulation of ketones.

Respiratory Acidosis: The patient may also have hypoventilation, leading to CO_2 retention, which causes respiratory acidosis.

Diabetic Nephropathy & lupus nephritis

1- The most important predictor for a diabetic to develop a nephropathy is:
a) Duration of diabetes
b) The development of retinopathy
c) Proteinuria

ANSWER: B

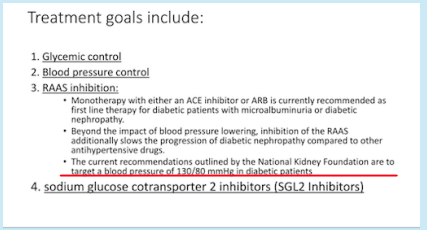
- Diabetic retinopathy and nephropathy are both caused by damage to small blood vessels due to chronic hyperglycemia. If a patient has diabetic retinopathy, it strongly suggests that microvascular damage is already occurring in the kidneys as well.
- Retinopathy often develops before significant kidney damage occurs, making it useful early warning sign.
- More than 90% of patients with T1DM and nephropathy have diabetic retinopathy.
- Absence of retinopathy in T1DM patients with proteinuria should prompt consideration of a diagnosis other than diabetic nephropathy.

Why not the other options?

A) Duration of Diabetes: While a longer duration of diabetes increases the risk of nephropathy, it is not the most specific predictor. Some patients may have diabetes for many years without developing nephropathy, depending on glycemic control and other factors.

C) Proteinuria: Proteinuria is a marker of existing nephropathy, not a predictor. By the time proteinuria appears, kidney damage has already occurred

4- Which is wrong about HTN:
Target of reduction in diabetic nephropathy is < 140/90 mmHg.


Treatment goals include:

1. Glycemic control
2. Blood pressure control
3. RAAS inhibition:
   - Monotherapy with either an ACE inhibitor or ARB is currently recommended as first line therapy for diabetic patients with microalbuminuria or diabetic nephropathy.
   - Beyond the impact of blood pressure lowering, inhibition of the RAAS additionally slows the progression of diabetic nephropathy compared to other antihypertensive drugs.
   - The current recommendations outlined by the National Kidney Foundation are to target a blood pressure of 130/80 mmHg in diabetic patients
4. sodium glucose cotransporter 2 inhibitors (SGL2 Inhibitors)

55- Goal for BP in DM:

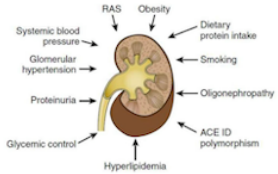
- 130/80.

32- A bad prognostic factor for a patient with diabetic nephropathy

• BP of 155/95.

Hypertension accelerates diabetic nephropathy progression.

Factors contributing to progression of DN



63- 40 y.o female with diabetic nephropathy, which one is true?
A. Absence of retinopathy excludes it.
B. Most patients develop nephropathy after 10-20 years after diagnosis
Ans: B

RRT

27- Absolute indication for dialysis in stage-V CKD patient:

• Pericarditis.

In end-stage kidney disease (ESKD), waste products (e.g., urea, creatinine, inflammatory mediators) accumulate, leading to pericardial inflammation.

Dialysis clears these toxins, reducing inflammation and preventing fluid accumulation in the pericardial sac.

Outside slides

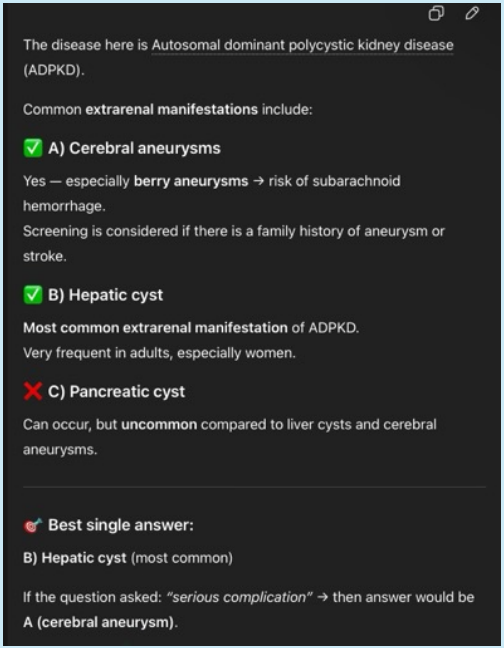
Mostly about polycystic kidney disease &
RTA

21

76) Adult patient with ADPKD, what of the following is a common extrarenal manifestation ?

A) Cerebral aneurysms
B) Hepatic cyst
C) Pancreatic cyst

Answer is B. Hepatic cyst according to chat gpt



The disease here is Autosomal dominant polycystic kidney disease (ADPKD).

Common **extrarenal manifestations** include:

✅ **A) Cerebral aneurysms**

Yes — especially **berry aneurysms** → risk of subarachnoid hemorrhage.
Screening is considered if there is a family history of aneurysm or stroke.

✅ **B) Hepatic cyst**

Most common extrarenal manifestation of ADPKD.
Very frequent in adults, especially women.

❌ **C) Pancreatic cyst**

Can occur, but **uncommon** compared to liver cysts and cerebral aneurysms.

🎯 **Best single answer:**

B) Hepatic cyst (most common)

If the question asked: *"serious complication"* → then answer would be **A (cerebral aneurysm)**.

3-A 45 year old man presented with sudden onset headache and loss of consciousness. He has a history of hypertension and CKD. His father and grandfather died of intracranial hemorrhages. What is the most likely diagnosis:

a) Medullary sponge kidney
b) Polycystic kidney disease
c) Renal cell carcinoma

ANSWER: B

- We have combination of CKD, family hx of fatal intracranial hemorrhages and a sudden onset of headache and loss of consciousness which is classic foe Autosomal Dominant Polycystic Kidney Disease (ADPKD).

ADPKD is known to cause:

- Multiple bilateral renal cysts leading to chronic kidney disease.
- Hypertension (often an early manifestation).
- A high risk of **berry aneurysms** in the cerebral vasculature. When these aneurysms rupture, they can cause subarachnoid hemorrhages, which often present as a sudden, severe headache ("worst headache of my life") and can lead to loss of consciousness or even death.
- The family history of intracranial hemorrhages (father and grandfather) strongly suggests an inherited condition that predisposes to these vascular aneurysms—again pointing toward ADPKD.

Why not the other options?

A) Medullary sponge kidney typically presents with recurrent kidney stones and hematuria rather than severe hypertension or a strong association with cerebral aneurysms.

C) Renal cell carcinoma could explain some kidney-related issues and can be familial in certain syndromes, but it does not commonly cause multiple aneurysms or the characteristic pattern of familial subarachnoid hemorrhages seen here.

37- Case scenario most likely APKD, which of the following goes with the diagnosis?

• Cerebral aneurysm.

ADPKD is associated with intracranial berry aneurysms, especially in the circle of Willis.
Can lead to subarachnoid hemorrhage

10- Patient with Chronic renal failure developed osteitisfibrosacystica, all the following may be associated except

a) HyperPTH
b) Hypocalcemia
c) Aluminum toxicity
d) Hyperphosphatemia
e) Metabolic Acidosis

ANSWER:C

Osteitis fibrosa cystica (OFC) is a bone disorder commonly associated with secondary hyperparathyroidism (secondary hyperPTH) in chronic kidney disease (CKD). It results from excessive parathyroid hormone (PTH) secretion, leading to increased bone resorption and characteristic bone lesions.

A) Secondary hyperparathyroidism occurs in CKD due to hypocalcemia and hyperphosphatemia, which stimulate excessive PTH secretion.

B) CKD leads to impaired vitamin D activation, reducing calcium absorption and causing hypocalcemia, which triggers PTH release.

D) In CKD, the failing kidneys cannot excrete phosphate, leading to hyperphosphatemia, which further stimulates PTH secretion.

E) CKD leads to reduced acid excretion, causing metabolic acidosis, which worsens bone demineralization and contributes to osteodystrophy.

Aluminum toxicity is NOT a direct cause of osteitis fibrosa cystica.

While aluminum toxicity can cause bone disease (adynamic bone disease or osteomalacia) in dialysis patients (due to aluminum-containing phosphate binders), it does not specifically cause osteitis fibrosa cystica, which is due to high PTH levels.

13- Most common diuretic to cause hyponatremia?
a) Furosemide
b) Thazide
c) Amiloride
d) No difference between them

ANSWER: B

Thiazide diuretics (e.g., hydrochlorothiazide, chlorthalidone) are the most common cause of diuretic-induced hyponatremia. They work by inhibiting the Na^+/Cl^- cotransporter in the distal convoluted tubule, leading to sodium loss and water retention, which can dilute plasma sodium levels.

Why not the other options?
A) Loop diuretics (e.g., furosemide) also cause sodium loss but are less likely to induce severe hyponatremia because they disrupt the medullary concentration gradient, limiting water reabsorption.
C) Amiloride is a potassium-sparing diuretic that blocks the epithelial sodium channel (ENaC) in the collecting duct, but it is not a common cause of hyponatremia.

21- A patient with features of GN + fresh blood per rectum + colicky abdominal pain, most appropriate thing to do is?

• Blood film to see schistocytes (this describes HUS following E.coli hemorrhagic diarrhea).

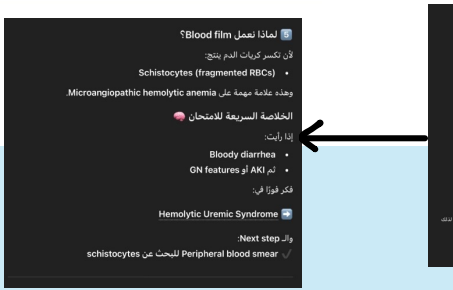
Hemolytic uremic syndrome (HUS) is a condition that can occur when small blood vessels become damaged and inflamed. This damage can cause clots to form in the vessels all through the body. The clots can damage the kidneys and other organs. Hemolytic uremic syndrome can lead to kidney failure, which can be life-threatening.

Anyone can get hemolytic uremic syndrome. But it's most common in young children. Most often, infection with certain strains of Escherichia coli (E. coli) bacteria is the cause.

The first symptoms of hemolytic uremic syndrome caused by E. coli bacteria might include:

- **Diarrhea, which is often bloody.**
- **Pain, cramping or bloating in the stomach area.**
- **Fever.**
- **Vomiting.**

Next Step: Blood film to look for schistocytes, which are fragmented RBCs seen in microangiopathic hemolytic anemia (MAHA), a hallmark of HUS.



22- A patient had cardiac cath, then developed acute decline in renal function, + livedo reticularis, Dx?

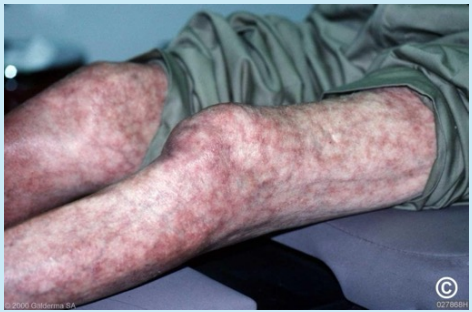
• Cholesterol emboli (this distinguishes cholesterol emboli from contrast-induced nephropathy).

This occurs when cholesterol crystals dislodge from atherosclerotic plaques during catheterization and embolize to small vessels, leading to renal ischemia and skin findings.

Key Differentiation: Unlike contrast-induced nephropathy (which presents with transient renal impairment), cholesterol embolism involves systemic signs like livedo reticularis and eosinophilia.

(Livedo reticularis is a common skin finding consisting of a mottled reticulated vascular pattern that appears as a lace-like purplish discoloration of the skin).

Livedo reticularis is a skin condition characterized by a net-like, mottled, reddish-blue discoloration, usually on the legs, caused by reduced blood flow or vessel spasms. Often triggered by cold, it is commonly benign but can indicate underlying conditions like autoimmune diseases, medication reactions, or blood clots.



40- Not a cause of hyponatremia?

- Lithium.

📊Not a cause of hyponatremia: Lithium
Lithium → actually causes nephrogenic diabetes insipidus → polyuria and hypernatremia, not hyponatremia.
Hyponatremia causes:
SIADH
Thiazides
Heart failure / cirrhosis
Hypothyroidism / adrenal insufficiency
✅Answer: Lithium is NOT a cause of hyponatremia.

42- Aldosterone:

- Increase Na-k channels.

Aldosterone → Increase Na-K channels
Aldosterone effect in distal nephron:
Increases ENaC channels → Na^+ reabsorption
Stimulates Na^+/K^+ ATPase → K^+ secretion into urine
Overall effect: Na^+ retention, K^+ excretion
✅ Answer: True — aldosterone increases Na-K channels and activity.

41- Patient with renal stones, urine Ph=7, hypokalemia:

- Distal RTA.

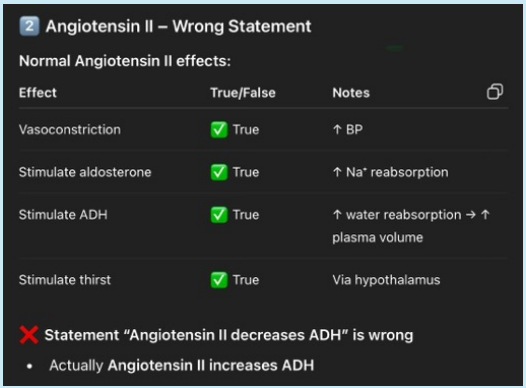
43- Patient with hypokalemia, HTN, metabolic alkalosis, hyernatemia, low aldosterone levels:

• Liddle syndrome.

Liddle's syndrome, also called Liddle syndrome, is a genetic disorder inherited in an autosomal dominant manner that is characterized by early, and frequently severe, high blood pressure associated with low plasma renin activity, metabolic alkalosis, low blood potassium, and normal to low levels of aldosterone.

52- Wrong about angiotensin 2:

- Decrease ADH.



2 Angiotensin II – Wrong Statement

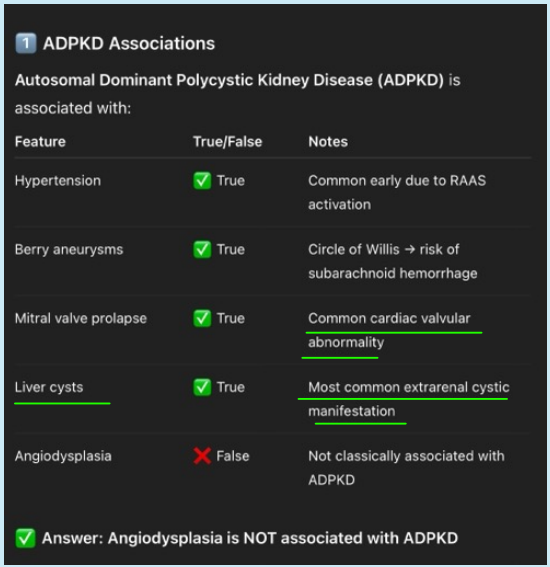
Normal Angiotensin II effects:

Effect	True/False	Notes
Vasoconstriction	✅ True	↑ BP
Stimulate aldosterone	✅ True	↑ Na^+ reabsorption
Stimulate ADH	✅ True	↑ water reabsorption → ↑ plasma volume
Stimulate thirst	✅ True	Via hypothalamus

❌ Statement "Angiotensin II decreases ADH" is wrong

- Actually **Angiotensin II increases ADH**

51- AD-PKD is associated with all of the following except:
• Angiodysplasia.



1️⃣ ADPKD Associations

Autosomal Dominant Polycystic Kidney Disease (ADPKD) is associated with:

Feature	True/False	Notes
Hypertension	✅ True	Common early due to RAAS activation
Berry aneurysms	✅ True	Circle of Willis → risk of subarachnoid hemorrhage
Mitral valve prolapse	✅ True	Common cardiac valvular abnormality
Liver cysts	✅ True	Most common extrarenal cystic manifestation
Angiodysplasia	❌ False	Not classically associated with ADPKD

✅ **Answer: Angiodysplasia is NOT associated with ADPKD**

2-Distinctive for distal RTA?
a) Kidney stones
b) fanconi syndrome
c) hypokalemia
d) Hypercalceuria

ANSWER: A

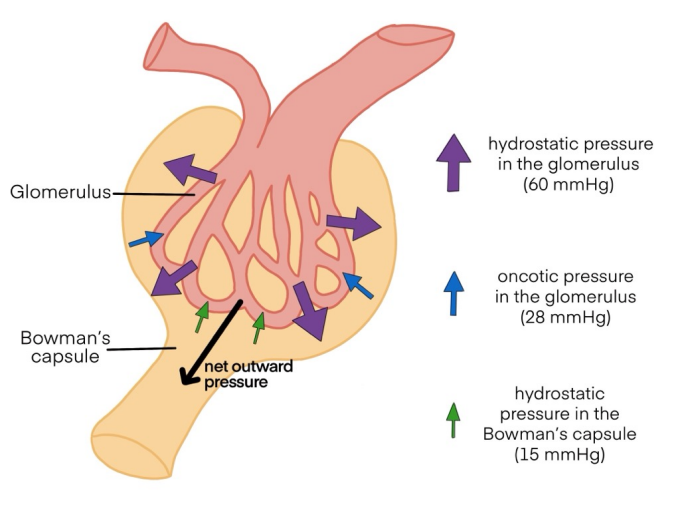
- Distal renal tubular acidosis (dRTA) is characterized by an inability of the distal tubules to acidify urine properly, leading to chronic metabolic acidosis. This results in various complications, including kidney stones.
- **Why are kidney stones distinctive for distal RTA?**
- Alkaline Urine (pH > 5.5): In dRTA, hydrogen ion (H^+) secretion is impaired, leading to persistently alkaline urine, which promotes calcium phosphate stone formation.
- Hypercalciuria: Increased calcium excretion in the urine contributes to nephrocalcinosis and kidney stones.
- Reduced Citrate Excretion (Hypocitraturia): Citrate normally binds calcium and prevents stone formation. In dRTA, citrate excretion is low, increasing the risk of stones.
- Nephrocalcinosis: Chronic acidemia leads to calcium deposition in the kidneys, which can further promote stone formation.

Why Not the Other Options?

B) Fanconi Syndrome: Fanconi syndrome is associated with proximal RTA (Type 2), not distal RTA.
C) Hypokalemia: While hypokalemia is a feature of distal RTA, it is not distinctive, as it also occurs in other types of RTA.
D) Hypercalciuria: While common in dRTA, hypercalciuria alone is not distinctive. It is the combination of hypercalciuria, hypocitraturia, and alkaline urine that makes kidney stones a hallmark of dRTA.

54- Blood hydrostatic pressure 55, blood oncotic pressure 30, bowman's capsule hydrostatic pressure 15, the net filtration pressure is:

• 10.



Step 1: Formula for Net Filtration Pressure (NFP)

$$\text{NFP} = P_{\text{hydrostatic (glomerular)}} - P_{\text{oncotic (plasma)}} - P_{\text{Bowman's capsule}}$$

$$\text{NFP} = P_{\text{G}} - \pi_{\text{B}} - P_{\text{BC}}$$

Seminar

7-Which of the following causes CKD with enlarged kidneys:
a) Amyloidosis
b) HTN
c) Glomerulonephritis
d) Hepatitis

ANSWER: A

CKD typically leads to shrunken, fibrotic kidneys, except in a few conditions that cause kidney enlargement like amyloidosis which is a systemic disorder in which misfolded proteins (amyloid) deposit in various organs, including the kidneys. In renal amyloidosis, amyloid fibrils accumulate in the glomeruli, tubules, and interstitium, leading to enlarged kidneys with nephrotic syndrome and progressive CKD.

Why not the other options?
B) Chronic HTN leads to nephrosclerosis, which causes small, atrophic kidneys due to ischemia and fibrosis.
C) Most types of chronic glomerulonephritis result in shrunken kidneys over time due to scarring and fibrosis.
D) Hepatitis itself does not directly cause CKD with enlarged kidneys.

9-A patient with renal failure is expected to have hypocalcemia due to :
a) Decreased hydroxylation of vitamin D
b) Decreased absorption of vitamin D

ANSWER:A

Vitamin D is obtained from diet and sunlight exposure as cholecalciferol (D3) or ergocalciferol (D2).
The liver converts it to 25-hydroxyvitamin D [25(OH)D], an inactive form.
The kidneys hydroxylate 25(OH)D into 1,25-dihydroxyvitamin D (calcitriol), the active form. Calcitriol increases calcium absorption from the intestines and promotes bone resorption

In renal failure, Kidneys fail to hydroxylate 25(OH)D into $1,25(OH)_2D$ (calcitriol).
Without calcitriol, intestinal calcium absorption decreases.
Hypocalcemia develops, leading to secondary hyperparathyroidism (renal osteodystrophy).

Why not the other option?
Option B (decreased absorption of vitamin D) refers to conditions affecting the gut (e.g., celiac disease, Crohn's), not renal failure.
In renal failure, vitamin D absorption is normal, but its activation is impaired.

﴿هَٰذَا مِن فَضْلِ رَبِّي﴾