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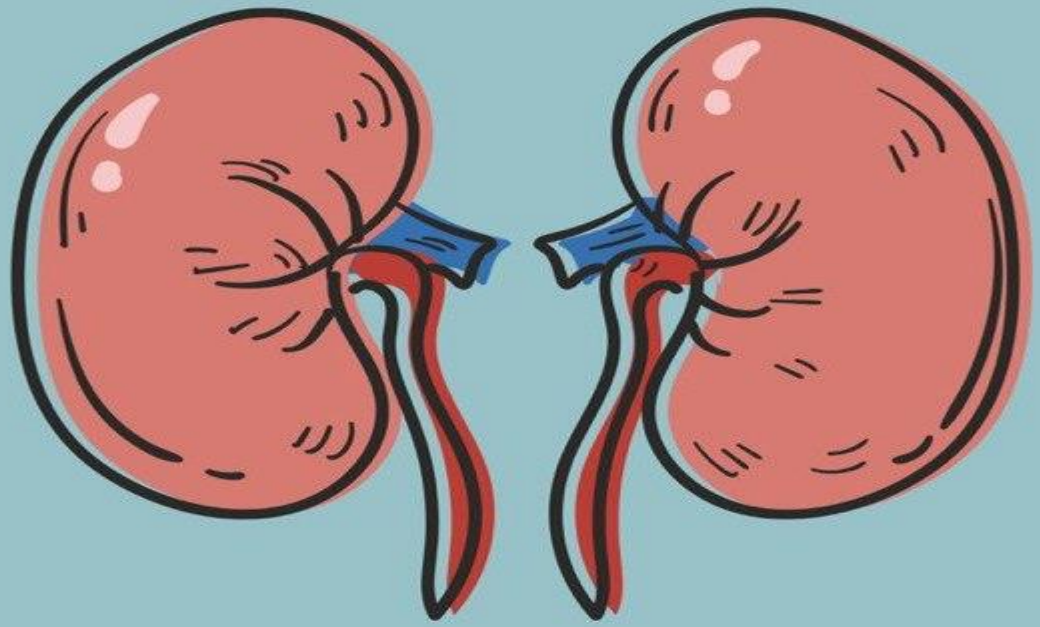

By Dania AbuSamha

Nephrology Test Bank

Collected by: Mona Moubarak

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

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.

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.

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.

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).

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.

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.

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

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.

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.

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.

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.

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.

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.

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.

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.

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.

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).

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.

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).

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.

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.

25- Hypokalemia, hyperchloremia, low bicarbonate, normal BP, urine pH is 6.5, most likely cause is:

• RTA.

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.

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.

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.

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

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.

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

• BP of 155/95.

Hypertension accelerates diabetic nephropathy progression.

33- An intubated patient with normal BP, JVP and no swellings, has hypernatremia, urine osmolality is 350, most likely cause:

• Diabetes Insipidus.

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.

35- Wrong about nephrotic syndrome:

• Hypertension.

36- True about kidney blood supply:

• NSAIDs cause constriction of the afferent arteriole.

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

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

• Doppler ultrasound/ renal biopsy.

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

40- Not a cause of hyponatremia?
• Lithium.

41- Patient with renal stones, urine Ph=7, hypokalemia:
• Distal RTA.

42- Aldosterone:
• Increase Na-k channels.

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.

44- Which is wrong about HTN:

• Target of reduction in diabetic nephropathy is < 140/90 mmHg.

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.

46- acid-base case, ph=7.6, HCO3=45, Na=133, Cl=75, PO2=60, PCO2=59:
• Metabolic alkalosis only.

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

50- A patient with hemoptysis, nasal mucosal ulcer, recently became oliguric,
ANCA +ve:
• Wegener’s granulomatosis.

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

52- Wrong about angiotensin 2:
• Decrease ADH.

53- ABGs and electrolytes results [PCO2=38, pH=7.12, HCO3- =12] dx?
• High anion gap metabolic acidosis and respiratory acidosis.

54- Blood hydrostatic pressure 55, blood oncotic pressure 30, bowman's capsule hydrostatic pressure 15, the net filtration pressure is:
• 10.

55- Goal for BP in DM:
• 130/80.

(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

57- Psychotic patient with hyponatremia,hypokalemia,normal urinary potassium and hypocalciuria
Ans: Thiazide abuse

58- ph 7.3, pCO2 38 ,HCO3- 13
Ans: HAGMA with respiratory acidosis

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

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

61- ptn with history of Lung CA and findings of nephrotic syndrome
Ans: Membranous nephropathy

62- CKD patient with hip fracture likely cause of hypocalcemia:
Ans: Decreased vit d hydroxylation

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

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

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 ?

66- A diabetic patient takes metformin,he has CKD, when is it indicated to stop Metformin?

1. Creatinine > 1.5
2. Creatinine > 1.5 for males and > 1.4 for females
3. GFR < 30
4. GFR < 15
5. When starting HD

Ans: 3