COMPREHENSIVE REVIEW TEST 3: RENAL, ELECTROLYTE, AND ACID-BASE DISORDERS

1. A 68-year-old man is admitted with sepsis. Baseline creatinine was 1.0 mg/dL. On day 2, creatinine is 2.1 mg/dL and urine output has been 150 mL over 12 hours. According to KDIGO criteria, what stage of AKI is this?

A. Stage 1

B. Stage 2

C. Stage 3

D. No AKI present

2. A patient with AKI has FeNa of 0.5% and urine sodium of 8 mEq/L. What type of AKI is most likely?

A. Prerenal azotemia

B. Acute tubular necrosis

C. Post-renal obstruction

D. Acute interstitial nephritis

3. Which medication should be avoided in a patient with AKI and GFR of 20 mL/min?

A. Acetaminophen

B. NSAIDs

C. Furosemide

D. Vancomycin (with dose adjustment)

4. A patient has the following ABG: pH 7.32, $PaCO_2$ 24 mmHg, HCO_3 12 mEq/L. What is the primary acid-base disorder?

A. Respiratory acidosis

B. Respiratory alkalosis

C. Metabolic acidosis

D. Metabolic alkalosis

5. Using Winter's formula, what is the expected $PaCO_2$ for a patient with HCO_3 of 12 mEq/L?

A. 20 mmHg

B. 26 mmHg

C. 32 mmHg

D. 40 mmHg

6. A patient presents with confusion, lethargy, and sodium of 118 mEq/L. Symptoms developed over 48 hours. What is the maximum recommended sodium correction rate in the first 24 hours?

A. 4-6 mEq/L

B. 10-12 mEq/L

C. 15-18 mEq/L

D. 20-24 mEq/L

7. A patient with SIADH has sodium of 122 mEq/L with mild symptoms. What is the most appropriate initial treatment?

A. 3% hypertonic saline bolus

B. Fluid restriction

C. Demeclocycline

D. Normal saline infusion

8. Which laboratory finding is most consistent with SIADH?

A. Serum osmolality >295 mOsm/kg

B. Urine osmolality <100 mOsm/kg

C. Urine sodium >40 mEq/L with low serum osmolality

D. Elevated BUN/creatinine ratio

9. A patient has sodium of 162 mEq/L and is obtunded. Symptoms developed over 24 hours. What is the appropriate treatment?

A. Rapid correction with D5W at 500 mL/hr

B. Slow correction not exceeding 10-12 mEq/L per 24 hours

C. 3% hypertonic saline

D. Fluid restriction

10. A patient with potassium of 6.8 mEq/L has peaked T waves on ECG. After calcium gluconate, what is the next intervention to shift potassium intracellularly?

A. Kayexalate

B. Hemodialysis

C. Insulin with dextrose

D. Furosemide

11. Which ECG change is NOT associated with hyperkalemia?

A. Peaked T waves

B. Prolonged QT interval

C. Widened QRS complex

D. Sine wave pattern

12. A patient with potassium of 2.6 mEq/L has muscle weakness and U waves on ECG. What is the most appropriate potassium replacement route?

A. Oral potassium 40 mEq daily

B. IV potassium 10 mEq/hr via peripheral line

C. IV potassium 20 mEq/hr via central line with monitoring

D. No replacement needed

13. What is the most common cause of hypokalemia in hospitalized patients?

A. Poor dietary intake

B. Diuretic use

C. Vomiting

D. Diarrhea

14. A patient has calcium of 14.2 mg/dL with confusion and polyuria. What is the first-line treatment?

A. Calcitonin

B. IV bisphosphonate

C. IV normal saline

D. Furosemide

15. Which malignancy is most commonly associated with hypercalcemia of malignancy?

A. Colon cancer

B. Lung cancer (squamous cell)

C. Prostate cancer

D. Hepatocellular carcinoma

16. A patient with chronic kidney disease has calcium of 7.8 mg/dL, phosphorus of 7.2 mg/dL, and PTH of 450 pg/mL. What is the underlying disorder?

A. Primary hyperparathyroidism

B. Secondary hyperparathyroidism

C. Hypoparathyroidism

D. Vitamin D toxicity

17. A post-thyroidectomy patient develops perioral numbness and carpopedal spasm. Calcium is 6.8 mg/dL. What is the appropriate treatment?

A. Oral calcium carbonate

B. IV calcium gluconate

C. IV magnesium sulfate

D. Observation

18. What is Chvostek's sign?

A. Carpopedal spasm with BP cuff inflation

B. Facial muscle contraction with tapping of facial nerve

C. Tetany with hyperventilation

D. QT prolongation on ECG

19. A patient has magnesium of 1.0 mEq/L and is experiencing torsades de pointes. What is the appropriate treatment?

A. Amiodarone 300 mg IV

B. Magnesium sulfate 2 g IV

C. Lidocaine 100 mg IV

D. Synchronized cardioversion

20. Which condition is most likely to cause hypomagnesemia?

A. Renal failure

B. Chronic alcohol use

C. Tumor lysis syndrome

D. Rhabdomyolysis

21. A patient's ABG shows pH 7.48, $PaCO_2$ 48 mmHg, HCO_3 34 mEq/L. What is the acid-base disorder?

A. Metabolic alkalosis

B. Respiratory alkalosis

C. Respiratory acidosis

D. Mixed disorder

22. A patient with severe vomiting has pH 7.52, $PaCO_2$ 48 mmHg, HCO_3 38 mEq/L, and potassium of 2.8 mEq/L. What is the most important treatment?

A. IV sodium bicarbonate

B. IV normal saline with potassium

C. Acetazolamide

D. Intubation for respiratory support

23. What is the anion gap formula?

A. Na - (Cl + HCO3)

B. Na + K - (Cl + HCO3)

C. (Na + K) - Cl

D. Na - Cl

24. A patient has Na 140, Cl 100, HCO3 10 mEq/L. What is the anion gap?

A. 10

B. 20

C. 30

D. 40

25. Which condition causes a high anion gap metabolic acidosis?

A. Diarrhea

B. Renal tubular acidosis

C. Diabetic ketoacidosis

D. Normal saline infusion

26. The mnemonic MUDPILES represents causes of what condition?

A. High anion gap metabolic acidosis

B. Non-anion gap metabolic acidosis

C. Respiratory acidosis

D. Metabolic alkalosis

27. A patient with DKA has pH 7.18, glucose 485 mg/dL, and anion gap of 28. After treatment, anion gap normalizes but pH remains 7.28 with HCO3 of 16. What has developed?

A. Respiratory acidosis

B. Non-anion gap metabolic acidosis

C. Metabolic alkalosis

D. No additional disorder

28. What is the delta-delta ratio used for?

A. Calculating creatinine clearance

B. Identifying mixed acid-base disorders

C. Determining sodium correction rate

D. Assessing potassium deficit

29. A patient has pH 7.22, $PaCO_2$ 22 mmHg, HCO_3 8 mEq/L. Using Winter's formula (expected $PaCO_2 = 1.5 \times HCO_3 + 8 \pm 2$), what is present?

A. Pure metabolic acidosis with appropriate compensation

B. Metabolic acidosis with respiratory acidosis

C. Metabolic acidosis with respiratory alkalosis

D. Pure respiratory alkalosis

30. What is the most common cause of non-anion gap metabolic acidosis?

A. Lactic acidosis

B. Diabetic ketoacidosis

C. Diarrhea

D. Methanol ingestion

31. A patient with CKD Stage 4 has GFR of 22 mL/min. Which medication requires dose adjustment?

A. Lisinopril

B. Metformin

C. Amlodipine

D. Atorvastatin

32. What is the indication for emergent hemodialysis?

A. Creatinine >5 mg/dL

B. GFR <15 mL/min

C. Refractory hyperkalemia with ECG changes

D. BUN >80 mg/dL

33. A patient on hemodialysis develops hypotension during the session. What is the most appropriate initial intervention?

A. Discontinue dialysis permanently

B. Administer IV normal saline bolus

C. Start vasopressor infusion

D. Trendelenburg position only

34. Which renal replacement modality is preferred for hemodynamically unstable patients?

A. Intermittent hemodialysis

B. Continuous renal replacement therapy (CRRT)

C. Peritoneal dialysis

D. No dialysis until stable

35. A patient with AKI has BUN 85 mg/dL and creatinine 6.2 mg/dL. BUN/Creatinine ratio is approximately 14:1. This is most consistent with?

A. Prerenal azotemia

B. Intrinsic renal disease

C. Post-renal obstruction

D. Normal finding

36. What urinalysis finding is characteristic of acute tubular necrosis?

A. Red blood cell casts

B. Muddy brown granular casts

C. White blood cell casts

D. Hyaline casts only

37. A patient with AKI has FeNa of 3.5% and urine sodium of 45 mEq/L. What is the most likely diagnosis?

A. Prerenal azotemia

B. Acute tubular necrosis

C. Hepatorenal syndrome

D. Dehydration

38. Which medication can cause acute interstitial nephritis?

A. Acetaminophen

B. Penicillin antibiotics

C. Statins

D. Beta-blockers

39. A patient develops oliguria, rash, fever, and eosinophilia after starting a new antibiotic. Urinalysis shows WBC casts and eosinophiluria. What is the diagnosis?

A. Acute tubular necrosis

B. Acute interstitial nephritis

C. Glomerulonephritis

D. Urinary tract infection

40. What is the corrected sodium formula for hyperglycemia?

A. Measured Na + 1.6 × [(glucose - 100)/100]

B. Measured Na - 1.6 × [(glucose - 100)/100]

C. Measured Na + 2.4 × [(glucose - 100)/100]

D. Measured Na × glucose/100

41. A patient has measured sodium of 128 mEq/L and glucose of 600 mg/dL. What is the corrected sodium?

A. 128 mEq/L

B. 132 mEq/L

C. 136 mEq/L

D. 140 mEq/L

42. What is the most serious complication of overly rapid correction of chronic hyponatremia?

A. Cerebral edema

B. Osmotic demyelination syndrome

C. Seizures

D. Pulmonary edema

43. A patient with cirrhosis has sodium of 124 mEq/L and is asymptomatic. Total body sodium is actually elevated. What is the pathophysiology?

A. True sodium deficiency

B. Hypervolemic hyponatremia

C. SIADH

D. Cerebral salt wasting

44. What is the primary treatment for hypervolemic hyponatremia in cirrhosis?

A. 3% hypertonic saline

B. Fluid and sodium restriction with diuretics

C. Normal saline infusion

D. Demeclocycline

45. A patient has phosphorus of 8.5 mg/dL in the setting of CKD. Which medication is used to lower serum phosphorus?

A. Calcium acetate (phosphate binder)

B. Phosphorus supplementation

C. Vitamin D

D. Calcitonin

46. What electrolyte abnormality is characteristic of tumor lysis syndrome?

A. Hypocalcemia, hyperkalemia, hyperphosphatemia, hyperuricemia

B. Hypercalcemia, hypokalemia, hypophosphatemia

C. Hypernatremia, hypercalcemia

D. Hypomagnesemia only

47. A patient receiving chemotherapy develops potassium 7.0 mEq/L, phosphorus 9.2 mg/dL, calcium 6.5 mg/dL, and uric acid 14 mg/dL. What is the diagnosis?

A. Rhabdomyolysis

B. Tumor lysis syndrome

C. DKA

D. Adrenal insufficiency

48. What is the prophylactic treatment to prevent tumor lysis syndrome?

A. Calcium supplementation

B. IV hydration and allopurinol or rasburicase

C. Phosphate binders alone

D. Potassium supplementation

49. A patient with rhabdomyolysis has CK of 45,000 U/L. What is the most important intervention to prevent AKI?

A. Sodium bicarbonate infusion

B. Aggressive IV normal saline hydration

C. Mannitol infusion

D. Immediate hemodialysis

50. Which urine finding is characteristic of rhabdomyolysis?

A. Positive blood on dipstick with few RBCs on microscopy

B. Heavy proteinuria

C. Glucosuria

D. Bacteriuria

51. A patient has pH 7.38, PaCO2 24 mmHg, HCO3 14 mEq/L. What acid-base disorder is present?

A. No acid-base disorder

B. Metabolic acidosis with respiratory compensation

C. Respiratory alkalosis with metabolic compensation

D. Mixed metabolic acidosis and respiratory alkalosis

52. What is the expected compensation for acute respiratory acidosis?

A. HCO3 increases 1 mEq/L per 10 mmHg rise in PaCO2

B. HCO3 increases 3.5 mEq/L per 10 mmHg rise in PaCO2

C. HCO3 decreases 2 mEq/L per 10 mmHg rise in PaCO2

D. No metabolic compensation in acute setting

53. A patient with chronic COPD has pH 7.36, PaCO2 60 mmHg, HCO3 33 mEq/L. What does this represent?

A. Acute respiratory acidosis

B. Chronic compensated respiratory acidosis

C. Metabolic alkalosis

D. Mixed disorder

54. Which IV fluid is most appropriate for a patient with hyperchloremic metabolic acidosis?

A. Normal saline (0.9% NaCl)

B. Lactated Ringer's solution

C. D5W

D. 3% hypertonic saline

55. A patient receiving large volume normal saline resuscitation develops pH 7.28, HCO3 18 mEq/L, Cl 115 mEq/L. What is the diagnosis?

A. Lactic acidosis

B. Dilutional acidosis/hyperchloremic metabolic acidosis

C. Renal tubular acidosis

D. DKA

56. What is the renal threshold for glucose (plasma glucose at which glucosuria occurs)?

A. 100 mg/dL

B. 180 mg/dL

C. 250 mg/dL

D. 400 mg/dL

57. A patient with AKI has urine output of 80 mL in 6 hours despite adequate fluid resuscitation. According to KDIGO, this meets criteria for which AKI stage based on urine output alone?

A. Stage 1

B. Stage 2

C. Stage 3

D. Does not meet AKI criteria

58. What is the mechanism of contrast-induced nephropathy?

A. Direct glomerular damage

B. Renal vasoconstriction and tubular toxicity

C. Immune-mediated injury

D. Urinary obstruction

59. What is the best preventive strategy for contrast-induced nephropathy in high-risk patients?

A. Pre-procedure diuresis with furosemide

B. IV hydration with isotonic saline before and after contrast

C. N-acetylcysteine alone

D. Prophylactic hemodialysis

60. A patient has potassium of 2.2 mEq/L that is refractory to IV potassium replacement. Magnesium level returns at 0.8 mEq/L. What is the next step?

A. Increase potassium infusion rate

B. Replace magnesium first

C. Add spironolactone

D. Check for GI losses

Answer Key with Explanations

1. B - KDIGO Stage 2 AKI is defined by creatinine 2.0-2.9 times baseline OR urine output <0.5 mL/kg/hr for ≥12 hours. This patient has creatinine 2.1× baseline (from 1.0 to 2.1) meeting Stage 2 criteria. Stage 1 is 1.5-1.9× baseline; Stage 3 is ≥3× baseline or creatinine ≥4.0 mg/dL.

2. A - FeNa <1% and urine sodium <20 mEq/L indicate prerenal azotemia where kidneys are avidly retaining sodium due to decreased perfusion. In ATN, tubular damage impairs sodium reabsorption resulting in FeNa >2% and urine sodium >40 mEq/L.

3. B - NSAIDs inhibit prostaglandin-mediated afferent arteriolar vasodilation, reducing renal blood flow and GFR. They can worsen AKI and should be avoided in renal impairment. Furosemide, acetaminophen, and dose-adjusted vancomycin can be used cautiously.

4. C - Low pH (7.32) with low HCO3 (12 mEq/L) indicates metabolic acidosis. The low PaCO2 (24 mmHg) represents respiratory compensation (hyperventilation to blow off CO2). Primary respiratory disorders would show PaCO2 as the initial abnormality.

5. B - Winter's formula: Expected PaCO2 = 1.5 × [HCO3] + 8 (±2) = 1.5 × 12 + 8 = 26 mmHg (range 24-28). This predicts appropriate respiratory compensation for metabolic acidosis. If actual PaCO2 differs significantly, a mixed disorder exists.

6. B - For symptomatic hyponatremia developing over >48 hours (chronic), correct sodium by no more than 10-12 mEq/L in 24 hours to prevent osmotic demyelination syndrome. More rapid correction is acceptable only for acute (<48 hours) severe symptomatic hyponatremia.

7. B - Mild symptomatic SIADH with sodium 122 mEq/L is initially treated with fluid restriction (typically <1-1.5 L/day). Hypertonic saline is reserved for severe symptoms (seizures, obtundation). Demeclocycline and vaptans are second-line options for refractory cases.

8. C - SIADH shows inappropriately concentrated urine (urine osmolality >100 mOsm/kg) despite low serum osmolality, with urine sodium >40 mEq/L indicating continued renal sodium excretion. Serum osmolality is low (<275 mOsm/kg), not elevated.

9. B - Hypernatremia should be corrected slowly, not exceeding 10-12 mEq/L per 24 hours to prevent cerebral edema. Rapid correction causes water to shift into brain cells that have accumulated osmolytes. Use hypotonic fluids (D5W or 0.45% saline) for free water replacement.

10. C - Insulin (regular 10 units IV) with dextrose (25-50g) shifts potassium intracellularly within 15-30 minutes by stimulating Na-K-ATPase. Kayexalate removes potassium from body but works slowly; dialysis is definitive but takes time to arrange. Calcium stabilizes membrane but doesn't lower potassium.

11. B - Prolonged QT interval is associated with hypocalcemia and hypomagnesemia, not hyperkalemia. Hyperkalemia causes peaked T waves (earliest sign), PR prolongation, QRS widening, loss of P waves, sine wave pattern, and ultimately VF/asystole.

12. C - Severe hypokalemia (2.6 mEq/L) with symptoms and ECG changes requires IV replacement. Central line allows faster infusion (20 mEq/hr) with cardiac monitoring. Peripheral IV maximum is 10 mEq/hr due to vein irritation. Oral replacement is too slow for severe symptomatic hypokalemia.

13. B - Diuretics (especially loop and thiazide) are the most common cause of hypokalemia in hospitalized patients. They increase potassium excretion by increasing distal tubular flow and sodium delivery. Always check potassium when initiating or increasing diuretic therapy.

14. C - IV normal saline is first-line for hypercalcemia, restoring intravascular volume and promoting calciuresis. Give 200-300 mL/hr initially. Bisphosphonates (zoledronic acid) are added for malignancy-related hypercalcemia but take 2-4 days to work. Calcitonin provides rapid but transient effect.

15. B - Squamous cell lung cancer commonly produces PTHrP (parathyroid hormone-related peptide) causing humoral hypercalcemia of malignancy. Breast cancer and multiple myeloma also frequently cause hypercalcemia through different mechanisms (bone metastases, osteoclast activation).

16. B - Secondary hyperparathyroidism occurs in CKD due to phosphate retention, decreased calcitriol production, and hypocalcemia, which stimulate PTH secretion. The pattern shows low calcium, high phosphorus, and markedly elevated PTH—classic for CKD-mineral bone disorder.

17. B - Acute symptomatic hypocalcemia (tetany, seizures, cardiac effects) requires IV calcium gluconate 1-2g over 10-20 minutes. Calcium gluconate is preferred over calcium chloride for peripheral administration as it's less irritating. Oral calcium is insufficient for acute severe hypocalcemia.

18. B - Chvostek's sign is facial muscle twitching (contraction of facial muscles) elicited by tapping the facial nerve anterior to the ear. It indicates neuromuscular irritability from hypocalcemia. Trousseau's sign is carpopedal spasm induced by BP cuff inflation (more specific).

19. B - Magnesium sulfate 2g IV is first-line for torsades de pointes regardless of serum magnesium level. It suppresses early afterdepolarizations that trigger torsades. This patient also needs magnesium replacement for the underlying hypomagnesemia causing the arrhythmia.

20. B - Chronic alcohol use causes hypomagnesemia through poor dietary intake, increased urinary losses, and GI losses. Alcoholism is the most common cause of hypomagnesemia. Renal failure, tumor lysis syndrome, and rhabdomyolysis cause hypermagnesemia, not hypomagnesemia.

21. A - Elevated pH (7.48) with elevated HCO_3 (34 mEq/L) indicates primary metabolic alkalosis. The elevated $PaCO_2$ (48 mmHg) represents appropriate respiratory compensation (hypoventilation to retain CO_2). If this were primary respiratory acidosis, pH would be low.

22. B - Metabolic alkalosis from vomiting causes volume depletion and hypokalemia. Treatment requires volume repletion with normal saline and potassium replacement. Chloride-responsive metabolic alkalosis (from vomiting, NG suction) corrects with saline; kidneys excrete excess bicarbonate once volume is restored.

23. A - Anion gap = Na - (Cl + HCO_3). Normal anion gap is 8-12 mEq/L. It represents unmeasured anions (albumin, phosphate, sulfate, organic anions). Some formulas include potassium [Na + K - (Cl + HCO_3)] with normal 12-16, but the simpler formula is more commonly used.

24. C - Anion gap = 140 - (100 + 10) = 30 mEq/L. This is significantly elevated (normal 8-12), indicating high anion gap metabolic acidosis. The unmeasured anions causing this could include lactate, ketones, uremic toxins, or toxic alcohols.

25. C - DKA causes high anion gap metabolic acidosis due to accumulation of ketoacids (beta-hydroxybutyrate, acetoacetate). Diarrhea and RTA cause non-anion gap (hyperchloremic) acidosis due to bicarbonate loss. Normal saline causes hyperchloremic acidosis, not anion gap acidosis.

26. A - MUDPILES represents causes of high anion gap metabolic acidosis: Methanol, Uremia, DKA, Propylene glycol, Isoniazid/Iron, Lactic acidosis, Ethylene glycol, Salicylates. Some versions use GOLDMARK: Glycols, Oxoproline, L-lactate, D-lactate, Methanol, ASA, Renal failure, Ketoacidosis.

27. B - When anion gap normalizes but acidosis persists, non-anion gap metabolic acidosis has developed. In DKA treatment, ketones are cleared (lowering anion gap) but aggressive saline causes hyperchloremic acidosis. This is common and usually resolves with continued treatment.

28. B - Delta-delta ratio compares the change in anion gap to change in bicarbonate: (AG - 12)/(24 - HCO3). Ratio <1 suggests concurrent non-AG acidosis; ratio >2 suggests concurrent metabolic alkalosis. It identifies mixed disorders when anion gap elevation doesn't match bicarbonate decrease.

29. A - Expected PaCO2 = 1.5(8) + 8 = 20 mmHg (range 18-22). Actual PaCO2 is 22 mmHg, within expected range. This represents pure metabolic acidosis with appropriate respiratory compensation. If PaCO2 were higher, concurrent respiratory acidosis would be present.

30. C - Diarrhea causes non-anion gap (hyperchloremic) metabolic acidosis through direct bicarbonate loss in stool. The kidneys retain chloride to maintain electroneutrality, hence "hyperchloremic." Other causes include RTA, carbonic anhydrase inhibitors, and ureteral diversions.

31. B - Metformin is contraindicated in CKD with GFR <30 mL/min due to risk of lactic acidosis from drug accumulation. It should be used cautiously with GFR 30-45 mL/min. Lisinopril, amlodipine, and atorvastatin can be used with appropriate monitoring in CKD.

32. C - Emergent dialysis indications (mnemonic AEIOU): Acidosis (severe, refractory), Electrolyte abnormalities (refractory hyperkalemia with ECG changes), Ingestion (toxic alcohols, lithium, salicylates), Overload (fluid overload refractory to diuretics), Uremia (encephalopathy, pericarditis, bleeding).

33. B - Intradialytic hypotension is common, occurring in 20-30% of sessions. Initial management is IV normal saline bolus (100-250 mL), reducing ultrafiltration rate, and Trendelenburg position. Dialysis can often continue with these measures. Vasopressors are rarely needed.

34. B - CRRT (continuous venovenous hemofiltration/hemodialysis) is preferred for hemodynamically unstable patients. It provides gradual fluid and solute removal over 24 hours, causing less hemodynamic instability than intermittent HD which removes large volumes over 3-4 hours.

35. B - BUN/Creatinine ratio of 14:1 (approximately 10-15:1) suggests intrinsic renal disease. Prerenal azotemia typically shows ratio >20:1 due to increased urea reabsorption with preserved creatinine clearance. Post-renal obstruction has variable ratios.

36. B - Muddy brown granular casts are pathognomonic for ATN, representing sloughed tubular epithelial cells and cellular debris. RBC casts indicate glomerulonephritis; WBC casts suggest pyelonephritis or interstitial nephritis; hyaline casts are nonspecific.

37. B - FeNa >2% and urine sodium >40 mEq/L indicate tubular dysfunction with impaired sodium reabsorption, characteristic of ATN. The tubules cannot appropriately conserve sodium despite decreased perfusion. Prerenal states have FeNa <1% with avid sodium retention.

38. B - Penicillins, cephalosporins, sulfonamides, NSAIDs, and PPIs commonly cause acute interstitial nephritis (AIN). AIN is an allergic/immune-mediated reaction causing inflammation in the renal interstitium. Classic triad: fever, rash, eosinophilia (present in only 10-30% of cases).

39. B - The classic triad of fever, rash, and eosinophilia with AKI after new medication exposure indicates acute interstitial nephritis. WBC casts and eosinophiluria (>1% urine eosinophils) support the diagnosis. Treatment involves stopping the offending drug; steroids may hasten recovery.

40. A - Corrected Na = Measured Na + 1.6 × [(glucose - 100)/100]. Hyperglycemia causes osmotic water shift from cells to extracellular space, diluting sodium. For every 100 mg/dL glucose above normal, sodium decreases by approximately 1.6 mEq/L. Some use 2.4 for severe hyperglycemia.

41. C - Corrected Na = 128 + 1.6 × [(600-100)/100] = 128 + 1.6 × 5 = 128 + 8 = 136 mEq/L. The true sodium is 136 mEq/L, indicating the low measured sodium is dilutional from hyperglycemia-induced water shift, not true hyponatremia.

42. B - Osmotic demyelination syndrome (central pontine myelinolysis) results from overly rapid correction of chronic hyponatremia. Brain cells adapt to hyponatremia by losing osmolytes; rapid correction causes cell shrinkage and demyelination, particularly in the pons. Symptoms appear 2-6 days after correction.

43. B - Cirrhosis causes hypervolemic hyponatremia. Despite total body sodium excess (edema, ascites), effective arterial volume is low due to splanchnic vasodilation. This triggers ADH release and water retention, diluting sodium. Total body water increases more than sodium.

44. B - Hypervolemic hyponatremia in cirrhosis is treated with fluid restriction (1-1.5 L/day), sodium restriction, and diuretics (spironolactone preferred as it blocks aldosterone). Hypertonic saline is dangerous as it worsens fluid overload. Vaptans may be considered for severe cases.

45. A - Phosphate binders (calcium acetate, sevelamer, lanthanum) are used to lower serum phosphorus in CKD by binding dietary phosphate in the gut and preventing absorption. Dietary phosphorus restriction is also important. Treating hyperphosphatemia helps prevent secondary hyperparathyroidism and vascular calcification.

46. A - Tumor lysis syndrome causes hyperkalemia (cell lysis releases K^+), hyperphosphatemia (released from cells), hypocalcemia (calcium-phosphate precipitation), and hyperuricemia (nucleic acid breakdown). This constellation is pathognomonic for TLS.

47. B - The combination of hyperkalemia, hyperphosphatemia, hypocalcemia, and hyperuricemia after chemotherapy is diagnostic of tumor lysis syndrome. It occurs when rapidly proliferating tumor cells (leukemia, lymphoma) are destroyed, releasing intracellular contents. Can cause fatal arrhythmias and AKI.

48. B - TLS prevention includes aggressive IV hydration (maintain urine output >2 mL/kg/hr) and allopurinol (prevents uric acid formation) or rasburicase (enzymatically degrades uric acid). Rasburicase is preferred for high-risk patients as it actively lowers uric acid rather than just preventing formation.

49. B - Aggressive IV hydration with normal saline (200-300 mL/hr targeting urine output >200-300 mL/hr) is the cornerstone of rhabdomyolysis treatment. This prevents myoglobin precipitation in tubules. Sodium bicarbonate to alkalinize urine is controversial; early hydration is most important.

50. A - Rhabdomyolysis shows positive blood on urine dipstick (detects heme) but few or no RBCs on microscopy. The dipstick detects myoglobin (released from damaged muscle) which contains heme. This discrepancy between dipstick and microscopy is characteristic.

51. D - Normal pH with low $PaCO_2$ and low HCO_3 suggests two opposing processes: metabolic acidosis (low HCO_3) and respiratory alkalosis (low $PaCO_2$). If this were simple metabolic acidosis with compensation, pH would still be low. Normal pH with abnormal values indicates mixed disorder.

52. A - Acute respiratory acidosis: HCO_3 increases by 1 mEq/L for every 10 mmHg rise in $PaCO_2$ (minimal buffering). Chronic (3-5 days): HCO_3 increases by 3.5 mEq/L per 10 mmHg $PaCO_2$ rise (renal compensation). The difference helps distinguish acute from chronic respiratory acidosis.

53. B - Chronic compensated respiratory acidosis shows elevated $PaCO_2$ with proportionally elevated HCO_3 (renal compensation) returning pH toward normal. Expected $HCO_3 = 24 + 3.5 \times [(60-40)/10] = 24 + 7 = 31$ mEq/L. Actual HCO_3 (33) matches expected chronic compensation.

54. B - Lactated Ringer's or other balanced crystalloids are preferred for hyperchloremic acidosis because they contain less chloride than normal saline. Normal saline (154 mEq/L chloride) can worsen or cause hyperchloremic acidosis. LR contains 109 mEq/L chloride with lactate metabolized to bicarbonate.

55. B - Large volume normal saline (0.9% NaCl) causes hyperchloremic non-anion gap metabolic acidosis. NS contains 154 mEq/L chloride (supraphysiologic), leading to chloride accumulation and bicarbonate displacement. This is increasingly recognized, promoting use of balanced crystalloids.

56. B - Renal threshold for glucose is approximately 180 mg/dL. Above this plasma level, tubular reabsorption capacity (Tm) is exceeded and glucose appears in urine. In diabetes, glucosuria indicates plasma glucose >180 mg/dL. SGLT2 inhibitors lower this threshold therapeutically.

57. B - KDIGO Stage 2 AKI by urine output criteria is <0.5 mL/kg/hr for ≥12 hours. This patient has 80 mL/6 hours, which if weight is ~70 kg equals ~0.19 mL/kg/hr, meeting Stage 2 criteria. Stage 1 is <0.5 mL/kg/hr for 6-12 hours; Stage 3 is <0.3 mL/kg/hr for ≥24 hours or anuria for ≥12 hours.

58. B - Contrast-induced nephropathy results from direct tubular toxicity of iodinated contrast and renal medullary vasoconstriction causing ischemia. Risk factors include pre-existing CKD, diabetes, volume depletion, and contrast volume. Creatinine typically rises 24-48 hours after contrast exposure.

59. B - IV isotonic saline hydration before and after contrast administration is the most effective preventive strategy. Typically 1-1.5 mL/kg/hr for 6-12 hours pre-procedure and 6-12 hours post-procedure. N-acetylcysteine has not shown consistent benefit. Use lowest contrast volume possible.

60. B - Hypokalemia refractory to potassium replacement often indicates concurrent hypomagnesemia. Magnesium is required for Na-K-ATPase function and renal potassium retention. Potassium replacement will be ineffective until magnesium is repleted. Always check magnesium in refractory hypokalemia.