FULL-LENGTH PRACTICE TEST 13

Practice Test 13: Nephrology

40 Questions — Recommended Time: 40 Minutes

1. A 55-year-old man with a 15-year history of poorly controlled type 2 diabetes presents with progressive bilateral lower extremity edema, fatigue, and foamy urine. Laboratory studies reveal serum creatinine 2.8 mg/dL, BUN 42 mg/dL, hemoglobin A1c 9.2%, serum albumin 2.9 g/dL, and urinalysis showing 3+ protein. A 24-hour urine collection reveals 4.5 grams of protein. Renal ultrasound shows bilaterally enlarged kidneys. Which of the following is the most likely diagnosis?

A. Minimal change disease

B. Diabetic nephropathy

C. Membranous nephropathy

D. IgA nephropathy

2. A 6-year-old boy presents with periorbital edema, abdominal distension, and decreased urine output for the past week. He had a recent upper respiratory infection. Laboratory studies reveal serum albumin 1.8 g/dL, total cholesterol 380 mg/dL, and urinalysis showing 4+ protein with no red blood cells or casts. Serum creatinine is normal. A 24-hour urine protein is 5.2 grams. Serum complement levels (C3, C4) are normal. Which of the following is the most likely diagnosis?

A. Post-streptococcal glomerulonephritis

B. Focal segmental glomerulosclerosis

C. IgA nephropathy

D. Minimal change disease

3. A 45-year-old woman presents with flank pain, gross hematuria, and a palpable abdominal mass. She has a family history of kidney disease, with her father dying from a ruptured cerebral aneurysm at age 50 and her brother requiring dialysis at age 45. CT scan reveals bilaterally enlarged kidneys with innumerable cysts of varying sizes. Multiple hepatic cysts are also identified. Which of the following is the most likely diagnosis?

A. Autosomal dominant polycystic kidney disease (ADPKD)

B. Autosomal recessive polycystic kidney disease

C. Medullary sponge kidney

D. Simple renal cysts

4. A 70-year-old man with a history of benign prostatic hyperplasia presents with progressive difficulty urinating, weak urinary stream, urinary frequency, and nocturia. Over the past 48 hours, he has been unable to void. He presents with suprapubic fullness and discomfort. A bladder scan reveals 900 mL of retained urine. Serum creatinine is 3.2 mg/dL, up from a baseline of 1.1 mg/dL. Renal ultrasound reveals bilateral hydronephrosis. Which of the following is the most likely type of acute kidney injury?

A. Prerenal azotemia

B. Intrinsic renal (acute tubular necrosis)

C. Postrenal (obstructive) acute kidney injury

D. Rapidly progressive glomerulonephritis

5. A 60-year-old man is admitted to the ICU with septic shock from pneumonia. He is treated with aggressive IV fluid resuscitation and vasopressors. Despite initial stabilization, his urine output decreases to less than 200 mL over 12 hours. Serum creatinine rises from 1.0 to 3.5 mg/dL over 48 hours. Urinalysis reveals muddy brown granular casts. Urine sodium is 55 mEq/L and fractional excretion of sodium (FENa) is 4.2%. Which of the following is the most likely diagnosis?

A. Prerenal azotemia

B. Acute tubular necrosis (ATN)

C. Acute interstitial nephritis

D. Postrenal obstruction

6. A 28-year-old woman presents with fatigue, joint pain, malar rash, and foamy urine. Laboratory studies reveal serum creatinine 1.8 mg/dL, serum albumin 2.5 g/dL, positive ANA (1:640), positive anti-dsDNA antibody, and low complement levels (C3 and C4). Urinalysis reveals 3+ protein, dysmorphic red blood cells, and red blood cell casts. A 24-hour urine protein is 3.8 grams. Which of the following is the most likely diagnosis?

A. Lupus nephritis

B. IgA nephropathy

C. Membranous nephropathy

D. Anti-GBM disease (Goodpasture syndrome)

7. A 65-year-old man with chronic kidney disease stage IIIB (eGFR 38 mL/min) presents for routine follow-up. Laboratory studies reveal calcium 8.2 mg/dL (low), phosphorus 6.5 mg/dL (elevated), intact PTH 285 pg/mL (markedly elevated), and 25-hydroxyvitamin D 12 ng/mL (low). DEXA scan reveals low bone mineral density. Which of the following best describes the underlying pathophysiology of his bone and mineral disorder?

A. Primary hyperparathyroidism

B. Vitamin D toxicity

C. Hypoparathyroidism

D. Secondary hyperparathyroidism due to CKD-mineral bone disorder

8. A 50-year-old woman presents to the emergency department with severe muscle weakness, fatigue, nausea, and ECG changes showing peaked T waves, widened QRS complex, and shortened QT interval. Laboratory studies reveal potassium 7.2 mEq/L, creatinine 5.5 mg/dL, and BUN 68 mg/dL. She has a history of CKD stage IV and missed her last dialysis session. Which of the following is the most appropriate immediate intervention to stabilize the cardiac membrane?

A. Oral sodium polystyrene sulfonate (Kayexalate)

B. Insulin and dextrose administration

C. Intravenous calcium gluconate

D. Emergent hemodialysis

9. A 35-year-old man presents with sudden onset of colicky flank pain radiating to the groin, nausea, and gross hematuria. He is writhing in pain and unable to find a comfortable position. Non-contrast CT of the abdomen and pelvis reveals a 6-mm calculus in the left proximal ureter with mild hydronephrosis. Laboratory studies reveal serum calcium 10.1 mg/dL and uric acid 7.5 mg/dL. Stone analysis reveals calcium oxalate composition. Which of the following is the most appropriate initial management?

A. Pain control with NSAIDs or opioids, IV fluids, and medical expulsive therapy (tamsulosin) with observation for spontaneous passage

B. Immediate surgical ureteroscopy

C. Extracorporeal shock wave lithotripsy (ESWL) as the first-line intervention

D. Open surgical stone removal

10. A 72-year-old man with CKD stage V (eGFR 8 mL/min) on hemodialysis presents with progressive shortness of breath, bilateral lower extremity edema, jugular venous distension, and bibasilar crackles. His dry weight is 75 kg, and his current weight is 80 kg. He missed two dialysis sessions. Chest X-ray reveals bilateral pleural effusions and pulmonary vascular congestion. Which of the following is the most likely cause of his symptoms?

A. Pneumonia

B. Volume overload from missed dialysis

C. Acute myocardial infarction

D. Pulmonary embolism

11. A 25-year-old man presents with dark ("cola-colored") urine, periorbital edema, and hypertension 10 days after a streptococcal pharyngitis infection. Laboratory studies reveal serum creatinine 2.2 mg/dL, BUN 35 mg/dL, low C3 complement, normal C4, elevated ASO titer, and positive anti-DNase B antibody. Urinalysis reveals dysmorphic red blood cells, red blood cell casts, and mild proteinuria. Which of the following is the most likely diagnosis?

A. IgA nephropathy

B. Membranoproliferative glomerulonephritis

C. Minimal change disease

D. Post-streptococcal glomerulonephritis (PSGN)

12. A 40-year-old woman is brought to the emergency department after being found unresponsive. Arterial blood gas reveals pH 7.18, $PaCO_2$ 12 mmHg, HCO_3 5 mEq/L. Serum studies reveal sodium 138 mEq/L, chloride 100 mEq/L, BUN 15 mg/dL, and glucose 90 mg/dL. Serum osmolality is 310 mOsm/kg. Calculated serum osmolality is 285 mOsm/kg, yielding an osmolal gap of 25. Which of the following is the most likely diagnosis?

A. Toxic alcohol (methanol or ethylene glycol) ingestion

B. Diabetic ketoacidosis

C. Lactic acidosis from sepsis

D. Normal saline-induced hyperchloremic metabolic acidosis

13. A 60-year-old man with a history of chronic alcohol use presents with confusion, asterixis, and muscle twitching. Laboratory studies reveal sodium 118 mEq/L, serum osmolality 250 mOsm/kg, urine osmolality 650 mOsm/kg, and urine sodium 45 mEq/L. He is euvolemic on physical examination with no edema, ascites, or signs of dehydration. Thyroid function and cortisol level are normal. Which of the following is the most likely diagnosis?

A. Psychogenic polydipsia

B. Adrenal insufficiency

C. Syndrome of inappropriate antidiuretic hormone secretion (SIADH)

D. Cerebral salt wasting

14. A 55-year-old woman with a history of rheumatoid arthritis on chronic NSAID therapy presents with fever, diffuse maculopapular rash, and acute kidney injury. Serum creatinine has risen from 0.9 to 2.8 mg/dL over two weeks. Urinalysis reveals white blood cell casts, eosinophiluria, and mild proteinuria. CBC reveals peripheral eosinophilia. Which of the following is the most likely diagnosis?

A. Acute tubular necrosis from NSAID use

B. Acute interstitial nephritis (drug-induced)

C. NSAID-induced minimal change disease

D. Prerenal azotemia from NSAID-mediated afferent arteriolar vasoconstriction

15. A 30-year-old man presents with recurrent episodes of gross hematuria that coincide with upper respiratory infections. Between episodes, he has persistent microscopic hematuria and mild proteinuria. Serum creatinine is 1.2 mg/dL. Serum IgA level is elevated. Complement levels (C3, C4) are normal. Renal biopsy reveals mesangial IgA deposition on immunofluorescence and mesangial proliferation on light microscopy. Which of the following is the most likely diagnosis?

A. Post-streptococcal glomerulonephritis

B. Thin basement membrane disease

C. Alport syndrome

D. IgA nephropathy (Berger disease)

16. A 58-year-old man with a 20-year history of hypertension presents with progressively worsening renal function. His blood pressure is 175/100 mmHg despite three antihypertensive agents. Serum creatinine is 2.5 mg/dL, eGFR 28 mL/min. Urinalysis reveals mild proteinuria without active sediment. Renal ultrasound reveals bilaterally small kidneys (8.5 cm each) with increased echogenicity and cortical thinning. Which of the following is the most likely diagnosis?

A. Diabetic nephropathy

B. Renovascular hypertension

C. Hypertensive nephrosclerosis

D. Chronic interstitial nephritis

17. A 65-year-old woman with heart failure (EF 25%) on high-dose furosemide and lisinopril presents with worsening fatigue. Laboratory studies reveal sodium 128 mEq/L, potassium 3.0 mEq/L, chloride 85 mEq/L, bicarbonate 36 mEq/L, creatinine 1.5 mg/dL, and arterial pH 7.52. Which of the following is the most likely acid-base disturbance?

A. Metabolic alkalosis from loop diuretic use (contraction alkalosis)

B. Respiratory alkalosis

C. Metabolic acidosis from renal tubular acidosis

D. Mixed respiratory and metabolic alkalosis

18. A 45-year-old man presents with severe hypertension (blood pressure 220/130 mmHg), headache, blurred vision, and papilledema on fundoscopic examination. Laboratory studies reveal creatinine 3.5 mg/dL (baseline 1.2 mg/dL), LDH 450 U/L, and schistocytes on peripheral blood smear. Urinalysis reveals proteinuria and hematuria. Which of the following is the most likely diagnosis?

A. Hypertensive urgency

B. Hypertensive emergency with malignant hypertension (thrombotic microangiopathy)

C. Chronic hypertensive nephrosclerosis

D. Pheochromocytoma crisis

19. A 50-year-old woman presents with progressive edema, fatigue, and foamy urine. She was diagnosed with hepatitis B infection 10 years ago. Laboratory studies reveal serum albumin 2.2 g/dL, total cholesterol 350 mg/dL, creatinine 1.6 mg/dL, and 24-hour urine protein of 8.5 grams. Complement levels show low C3 and C4. Renal biopsy reveals thickened glomerular basement membranes with subepithelial "spike and dome" pattern on silver stain and granular IgG and C3 deposition along the capillary walls on immunofluorescence. Which of the following is the most likely diagnosis?

A. Minimal change disease

B. Focal segmental glomerulosclerosis

C. IgA nephropathy

D. Membranous nephropathy

20. A 70-year-old man with diabetes and CKD stage IV (eGFR 22 mL/min) is on lisinopril 20 mg daily and spironolactone 25 mg daily. He presents with generalized weakness and palpitations. ECG reveals peaked T waves and a widened QRS complex. Serum potassium is 6.8 mEq/L. Which of the following medication combinations is most likely contributing to his hyperkalemia?

A. Furosemide and hydrochlorothiazide

B. Amlodipine and metoprolol

C. ACE inhibitor (lisinopril) and potassium-sparing diuretic (spironolactone) in the setting of CKD

D. Aspirin and atorvastatin

21. A 48-year-old man presents with acute onset of flank pain and gross hematuria. He has a history of recurrent calcium oxalate kidney stones. He asks about dietary modifications to prevent future stones. Which of the following is the most appropriate dietary recommendation?

A. Increase fluid intake to maintain urine output greater than 2.5 liters per day and reduce dietary sodium and animal protein

B. Restrict calcium intake severely

C. Increase oxalate-rich foods

D. Decrease fluid intake to concentrate urine

22. A 35-year-old woman with SLE on no immunosuppressive therapy presents with worsening edema, hypertension, and foamy urine. Renal biopsy reveals diffuse proliferative changes on light microscopy with "wire-loop" lesions, subendothelial immune complex deposits on electron microscopy, and "full house" immunofluorescence pattern (IgG, IgA, IgM, C3, C1q deposition). Which class of lupus nephritis does this represent?

A. Class II (mesangial proliferative)

B. Class III (focal proliferative)

C. Class IV (diffuse proliferative — the most severe and common form requiring aggressive treatment)

D. Class V (membranous)

23. A 28-year-old man presents with hemoptysis, dyspnea, and rapidly progressive renal failure over two weeks. Serum creatinine is 4.5 mg/dL, up from 0.9 mg/dL two weeks ago. Urinalysis reveals red blood cell casts and proteinuria. Chest X-ray reveals bilateral pulmonary infiltrates. Anti-glomerular basement membrane (anti-GBM) antibodies are positive. Renal biopsy reveals crescentic glomerulonephritis with linear IgG deposition along the glomerular basement membrane on immunofluorescence. Which of the following is the most likely diagnosis?

A. Granulomatosis with polyangiitis (Wegener's)

B. Microscopic polyangiitis

C. IgA nephropathy

D. Anti-GBM disease (Goodpasture syndrome)

24. A 55-year-old man with type 2 diabetes and hypertension presents with microalbuminuria (urine albumin-to-creatinine ratio 180 mg/g) on two separate occasions. His serum creatinine is 1.1 mg/dL and eGFR is 75 mL/min. Blood pressure is 145/92 mmHg. He is currently on amlodipine 10 mg daily. Which of the following is the most appropriate medication to add for both blood pressure control and nephroprotection?

A. Hydrochlorothiazide

B. ACE inhibitor or ARB

C. Non-dihydropyridine calcium channel blocker (diltiazem)

D. Beta-blocker (metoprolol)

25. A 60-year-old woman with a history of recurrent UTIs presents with chronic bilateral flank pain, low-grade fever, and progressive renal insufficiency. Urinalysis reveals WBC casts and bacteriuria. Urine culture grows Proteus mirabilis. CT scan reveals bilateral irregular renal cortical scarring with blunted calyces. Which of the following is the most likely diagnosis?

A. Chronic pyelonephritis (reflux nephropathy)

B. Acute pyelonephritis

C. Renal cell carcinoma

D. Polycystic kidney disease

26. A 72-year-old man on hemodialysis for ESRD secondary to diabetic nephropathy develops worsening pruritus, bone pain, and a calcium-phosphorus product of 72 mg^2/dL^2 (markedly elevated). His PTH is 950 pg/mL. X-rays reveal subperiosteal bone resorption of the phalanges and vascular calcifications. Which of the following is the most serious complication of this condition?

A. Osteomalacia

B. Adynamic bone disease

C. Aluminum bone disease

D. Calciphylaxis (calcific uremic arteriolopathy)

27. A 40-year-old woman presents with recurrent episodes of severe hypokalemia (potassium 2.5 mEq/L), metabolic alkalosis, hypomagnesemia, and normal blood pressure. She denies diuretic use or vomiting. Urine potassium is elevated (greater than 30 mEq/day), urine calcium is low, and renin and aldosterone levels are elevated. She has a family history of a similar condition diagnosed in her brother during childhood. Which of the following is the most likely diagnosis?

A. Primary hyperaldosteronism (Conn syndrome)

B. Gitelman syndrome

C. Liddle syndrome

D. Bartter syndrome

28. A 68-year-old man with multiple myeloma presents with acute kidney injury. Serum creatinine has risen from 1.0 to 4.8 mg/dL over 3 weeks. Serum calcium is 13.5 mg/dL. Urine dipstick is negative for protein, but a sulfosalicylic acid (SSA) test is strongly positive. Serum free light chain ratio is markedly abnormal. Renal biopsy reveals eosinophilic casts within the tubular lumen surrounded by multinucleated giant cells. Which of the following is the most likely cause of his acute kidney injury?

A. Hypercalcemic nephropathy alone

B. Amyloidosis (AL type)

C. Myeloma cast nephropathy (myeloma kidney)

D. Light chain deposition disease

29. A 25-year-old woman is found to have persistent isolated microscopic hematuria on routine urinalysis. She is asymptomatic with normal blood pressure. Serum creatinine is 0.8 mg/dL. Urine protein-to-creatinine ratio is normal. She has a family history of hematuria in her mother and maternal grandmother, neither of whom developed kidney failure. Renal biopsy reveals diffuse thinning of the glomerular basement membrane on electron microscopy with no immune complex deposits. Which of the following is the most likely diagnosis?

A. Thin basement membrane disease (benign familial hematuria)

B. IgA nephropathy

C. Alport syndrome

D. Minimal change disease

30. A 55-year-old man with recently diagnosed small cell lung cancer presents with confusion, nausea, and lethargy. Laboratory studies reveal sodium 119 mEq/L, serum osmolality 245 mOsm/kg, urine osmolality 550 mOsm/kg, and urine sodium 60 mEq/L. He is euvolemic with normal thyroid function and cortisol level. Which of the following is the most appropriate initial management for his symptomatic hyponatremia?

A. Aggressive fluid resuscitation with normal saline

B. Oral demeclocycline as first-line therapy

C. Fluid restriction alone

D. Hypertonic saline (3% NaCl) with careful monitoring to avoid correction exceeding 10-12 mEq/L in 24 hours

31. A 50-year-old man with a history of type 2 diabetes presents with nephrotic-range proteinuria. His serum creatinine is 2.0 mg/dL. He has been diabetic for 18 years but has no diabetic retinopathy on fundoscopic examination. Renal biopsy is performed due to the atypical presentation and reveals Congo red-positive deposits with apple-green birefringence under polarized light. Mass spectrometry confirms AL (immunoglobulin light chain) amyloid. Which of the following is the most likely diagnosis?

A. Diabetic nephropathy

B. Membranous nephropathy

C. AL amyloidosis with renal involvement

D. Focal segmental glomerulosclerosis

32. A 30-year-old woman at 34 weeks gestation presents with headache, visual changes, right upper quadrant pain, and blood pressure of 170/110 mmHg. Urinalysis reveals 3+ proteinuria. Laboratory studies reveal platelet count 85,000/μL, AST 280 U/L, ALT 310 U/L, creatinine 1.4 mg/dL, and LDH 750 U/L. Which of the following is the most appropriate definitive treatment?

A. IV magnesium sulfate alone

B. Delivery of the fetus (definitive treatment for preeclampsia with severe features/HELLP syndrome)

C. Oral labetalol and continued observation until term

D. Plasma exchange therapy

33. A 45-year-old man presents with recurrent kidney stones. A 24-hour urine collection reveals urine pH consistently below 5.5, low urine volume, and elevated uric acid excretion. Stone analysis reveals uric acid composition. He has a history of gout and metabolic syndrome. Which of the following is the most appropriate pharmacologic intervention to prevent recurrent uric acid stones?

A. Hydrochlorothiazide

B. Calcium supplementation

C. Increased dietary purine intake

D. Potassium citrate (urinary alkalinization to pH 6.0-6.5) and allopurinol if hyperuricosuria persists

34. A 58-year-old man with CKD stage IIIA (eGFR 52 mL/min) secondary to hypertension is started on lisinopril 10 mg daily. Two weeks later, his serum creatinine rises from 1.5 to 2.0 mg/dL (a 33% increase), and serum potassium rises from 4.5 to 5.4 mEq/L. He is asymptomatic. Which of the following is the most appropriate management?

A. Continue lisinopril with close monitoring — a creatinine rise up to 30-35% is expected and acceptable after ACE inhibitor initiation

B. Immediately discontinue lisinopril permanently

C. Add spironolactone

D. Switch to a higher dose of lisinopril

35. A 42-year-old woman with no significant past medical history presents with bilateral flank pain, fever of 103°F, and oliguria for 2 days. She recently completed a 10-day course of trimethoprim-sulfamethoxazole for a UTI. Serum creatinine is 4.2 mg/dL. Urinalysis reveals sterile pyuria, white blood cell casts, and eosinophiluria. Peripheral blood reveals eosinophilia. She has a new maculopapular rash on her trunk. Which of the following is the most appropriate treatment?

A. Broad-spectrum IV antibiotics

B. Emergent hemodialysis

C. Discontinue the offending drug and consider corticosteroids if no improvement

D. Cyclophosphamide pulse therapy

36. A 68-year-old man with ESRD on hemodialysis presents with pericardial chest pain that worsens with inspiration and improves with leaning forward. Physical examination reveals a pericardial friction rub. ECG reveals diffuse ST elevation with PR depression. He missed his last two dialysis sessions. Which of the following is the most likely diagnosis?

A. Acute myocardial infarction

B. Uremic pericarditis

C. Viral pericarditis

D. Constrictive pericarditis

37. A 55-year-old man with recently diagnosed stage IV non-small cell lung cancer begins his first cycle of cisplatin-based chemotherapy. Forty-eight hours later, he develops nausea, muscle cramps, oliguria, and ECG changes. Laboratory studies reveal potassium 7.0 mEq/L, phosphorus 8.5 mg/dL, calcium 6.2 mg/dL, uric acid 15 mg/dL, LDH 1,500 U/L, and creatinine 4.0 mg/dL (baseline 1.0). Which of the following is the most likely diagnosis?

A. Cisplatin-induced acute tubular necrosis

B. Prerenal AKI from dehydration

C. Acute interstitial nephritis from chemotherapy

D. Tumor lysis syndrome

38. A 62-year-old man with a history of renal artery stenosis from atherosclerosis presents with flash pulmonary edema and severe hypertension (blood pressure 210/120 mmHg). He has a history of recurrent episodes of acute pulmonary edema with rapid onset and resolution. Renal artery duplex ultrasound reveals greater than 70% stenosis of both renal arteries. Serum creatinine is 2.5 mg/dL. Which of the following is the most likely mechanism of his recurrent flash pulmonary edema?

A. Bilateral renal artery stenosis causing sodium and water retention with pressure-dependent natriuresis failure

B. Acute myocardial infarction

C. Mitral valve stenosis

D. Chronic obstructive pulmonary disease exacerbation

39. A 48-year-old man presents with acute kidney injury following a marathon in hot weather. He reports dark brown urine and severe muscle pain and cramping. Serum creatinine is 4.5 mg/dL, CK is 45,000 U/L (markedly elevated), potassium 6.2 mEq/L, phosphorus 7.8 mg/dL, calcium 6.8 mg/dL, and uric acid 12 mg/dL. Urinalysis reveals 4+ blood on dipstick but no red blood cells on microscopy. Which of the following is the most likely diagnosis?

A. Acute post-streptococcal glomerulonephritis

B. Rhabdomyolysis-induced acute kidney injury

C. Acute tubular necrosis from dehydration

D. Thrombotic thrombocytopenic purpura

40. A 65-year-old woman with a history of breast cancer treated with chemotherapy 5 years ago presents with progressive peripheral edema, proteinuria (5.5 g/day), and hypoalbuminemia. Serum complement levels are normal. Renal biopsy reveals segmental sclerosis of the glomerular tuft with hyalinosis, involving some but not all glomeruli. Electron microscopy reveals diffuse podocyte foot process effacement. She is obese with a BMI of 35. Which of the following is the most likely diagnosis?

A. Minimal change disease

B. Membranous nephropathy

C. Focal segmental glomerulosclerosis (FSGS)

D. Diabetic nephropathy

PRACTICE TEST 13: ANSWER KEY WITH EXPLANATIONS

Nephrology

1. B. Diabetic nephropathy. Diabetic nephropathy is the leading cause of end-stage renal disease in the United States, developing in approximately 30-40% of patients with diabetes after 15-25 years of disease. The natural history progresses through hyperfiltration (enlarged kidneys with elevated GFR), microalbuminuria (30-300 mg/day), overt proteinuria (greater than 300 mg/day), declining GFR, and eventually ESRD. Bilaterally enlarged kidneys early in diabetic nephropathy (from hyperfiltration and glomerular hypertrophy) distinguish it from most other causes of CKD, which produce small, shrunken kidneys. Nephrotic-range proteinuria (greater than 3.5 g/day) with poor glycemic control (HbA1c 9.2%) and hypoalbuminemia are characteristic. Renal biopsy shows Kimmelstiel-Wilson nodules (nodular glomerulosclerosis) pathognomonic for diabetic nephropathy. Treatment includes strict glycemic control, ACE inhibitors or ARBs for nephroprotection, SGLT2 inhibitors, blood pressure control, and finerenone.

2. D. Minimal change disease. Minimal change disease (MCD) is the most common cause of nephrotic syndrome in children (approximately 80% of pediatric cases), typically presenting between ages 2-6 years. It produces the full nephrotic syndrome — heavy proteinuria (greater than 3.5 g/day in adults or greater than 40 mg/m²/hour in children), hypoalbuminemia, hyperlipidemia, and edema — with preserved renal function and normal complement levels. Urinalysis reveals heavy proteinuria without hematuria or red blood cell casts (bland sediment). Light microscopy appears normal, and immunofluorescence is negative — the only abnormality is diffuse podocyte foot process effacement on electron microscopy. MCD is highly responsive to corticosteroids (prednisone), with approximately 90% of children achieving complete remission within 8 weeks. Relapse is common, occurring in approximately 50-75% of cases.

3. A. Autosomal dominant polycystic kidney disease (ADPKD). ADPKD is the most common hereditary renal disease, caused by mutations in PKD1 (chromosome 16, approximately 85% of cases, more severe) or PKD2 (chromosome 4, approximately 15%, milder course). It is characterized by progressive bilateral renal cyst formation leading to massive kidney enlargement and eventual renal failure (mean age of ESRD approximately 54 years for PKD1, 74 years for PKD2). Extrarenal manifestations include hepatic cysts (most common extrarenal manifestation), intracranial berry aneurysms (risk of subarachnoid hemorrhage, explaining the father's death), mitral valve prolapse, and colonic diverticula. The family history showing autosomal dominant inheritance pattern is characteristic. Tolvaptan (vasopressin V2 receptor antagonist) slows cyst growth and disease progression. Screening of at-risk family members with renal ultrasound is recommended.

4. C. Postrenal (obstructive) acute kidney injury. Postrenal AKI results from obstruction of urinary flow at any level from the renal pelvis to the urethra. Bilateral obstruction or obstruction of a solitary functioning kidney is required to produce significant renal failure. In this patient, benign prostatic hyperplasia causes bladder outlet obstruction (the most common cause of postrenal AKI in elderly men), leading to urinary retention (900 mL on bladder scan), bilateral hydronephrosis from back-pressure, and rising creatinine. Immediate management is urinary catheter placement to relieve the obstruction, followed by monitoring for post-obstructive diuresis (massive polyuria from impaired concentrating ability and osmotic diuresis that can cause hypovolemia and electrolyte derangements). Renal function typically recovers significantly after obstruction relief if intervention is timely.

5. B. Acute tubular necrosis (ATN). ATN is the most common cause of intrinsic renal AKI, resulting from ischemic or nephrotoxic injury to the renal tubular epithelial cells. In this septic shock patient, despite fluid resuscitation, prolonged renal hypoperfusion progressed from prerenal azotemia to established ATN. The hallmark urinalysis finding is muddy brown granular casts (composed of sloughed tubular epithelial cells and cellular debris). Laboratory findings distinguishing ATN from prerenal azotemia include elevated urine sodium (greater than 40 mEq/L, reflecting impaired tubular sodium reabsorption), FENa greater than 2% (ATN) versus less than 1% (prerenal), and BUN-to-creatinine ratio less than 20:1 (versus greater than 20:1 in prerenal). ATN is typically self-limited with recovery expected in 1-3 weeks if the underlying cause is corrected, though supportive care including dialysis may be required during the recovery period.

6. A. Lupus nephritis. Lupus nephritis occurs in approximately 50-60% of SLE patients and is one of the most serious manifestations of the disease. The combination of nephritic features (dysmorphic RBCs, RBC casts indicating glomerular inflammation) with nephrotic features (heavy proteinuria, hypoalbuminemia) is characteristic of a mixed nephritic-nephrotic presentation. Positive ANA, anti-dsDNA antibodies, and low complement levels (C3, C4) confirm active SLE with complement consumption from immune complex deposition. Renal biopsy is essential for classification using the ISN/RPS system (Class I-VI), which guides treatment intensity. Class IV (diffuse proliferative) is the most common and severe form, treated with aggressive immunosuppression (mycophenolate mofetil or cyclophosphamide induction, followed by mycophenolate maintenance). ACE inhibitors or ARBs are added for proteinuria reduction and nephroprotection.

7. D. Secondary hyperparathyroidism due to CKD-mineral bone disorder. CKD-mineral bone disorder (CKD-MBD) results from progressive loss of functioning nephrons causing impaired phosphorus excretion (hyperphosphatemia), decreased 1-alpha-hydroxylation of 25-hydroxyvitamin D to active 1,25-dihydroxyvitamin D (calcitriol), and resultant hypocalcemia. These derangements stimulate compensatory PTH secretion (secondary hyperparathyroidism), which initially maintains serum calcium and phosphorus but eventually causes renal osteodystrophy. The markedly elevated PTH (285 pg/mL) with low calcium, high phosphorus, and low vitamin D in the setting of CKD confirms secondary hyperparathyroidism. Treatment includes phosphate binders (calcium-based or non-calcium-based such as sevelamer), calcitriol or active vitamin D analogs (paricalcitol), calcimimetics (cinacalcet) for persistent hyperparathyroidism, and dietary phosphorus restriction.

8. C. Intravenous calcium gluconate. Severe hyperkalemia (potassium greater than 6.5 mEq/L) with ECG changes is a medical emergency requiring immediate treatment. The priority is cardiac membrane stabilization with IV calcium gluconate (or calcium chloride through a central line), which acts within 1-3 minutes to antagonize the membrane effects of hyperkalemia by restoring the normal resting membrane potential, but does not lower serum potassium. After cardiac stabilization, potassium-lowering measures are initiated — insulin with dextrose (shifts potassium intracellularly within 15-30 minutes, most effective temporizing measure), sodium bicarbonate (for concurrent acidosis), inhaled beta-2 agonists (albuterol), and potassium elimination strategies including loop diuretics, sodium polystyrene sulfonate or patiromer (GI potassium binders), and hemodialysis (definitive treatment for refractory or severe hyperkalemia in ESRD patients).

9. A. Pain control with NSAIDs or opioids, IV fluids, and medical expulsive therapy with observation. Urolithiasis presents with acute onset of severe colicky flank pain radiating to the groin from ureteral peristalsis against the obstructing stone. Non-contrast CT is the imaging study of choice (sensitivity greater than 95%). For stones 5-10 mm in the proximal ureter, initial conservative management includes pain control (ketorolac is often first-line; opioids for refractory pain), IV hydration, and medical expulsive therapy with tamsulosin (alpha-1 blocker that relaxes ureteral smooth muscle, increasing spontaneous passage rates by approximately 30%). Stones less than 5 mm have approximately 90% spontaneous passage rate, while 5-10 mm stones have approximately 50% passage rate. Indications for urgent intervention (ureteroscopy or ESWL) include persistent obstruction, infection (obstructed infected system is a urologic emergency), intractable pain, solitary kidney, or failure to pass within 4-6 weeks.

10. B. Volume overload from missed dialysis. ESRD patients on hemodialysis are anuric or severely oliguric and depend entirely on dialysis for fluid and solute removal. Missing dialysis sessions leads to progressive fluid accumulation, electrolyte derangements (hyperkalemia, hyperphosphatemia), and uremic toxin accumulation. This patient's 5 kg weight gain above dry weight (approximately 5 liters of excess fluid) explains his symptoms of volume overload — dyspnea, bilateral lower extremity edema, JVD, bibasilar crackles, and chest X-ray findings of pleural effusions and pulmonary vascular congestion. Treatment is urgent hemodialysis with ultrafiltration to remove excess fluid and restore dry weight. Patient education regarding dialysis adherence and fluid restriction (typically 1-1.5 liters daily) between sessions is essential.

11. D. Post-streptococcal glomerulonephritis (PSGN). PSGN is the prototypical post-infectious glomerulonephritis, occurring 1-3 weeks after streptococcal pharyngitis or 3-6 weeks after streptococcal skin infection (impetigo). It results from immune complex deposition in the glomeruli, activating complement and causing glomerular inflammation. The nephritic presentation includes hematuria (dark "cola-colored" urine from dysmorphic RBCs), RBC casts (pathognomonic for glomerulonephritis), hypertension, edema, and oliguria with mild-moderate proteinuria. Low C3 with normal C4 (indicating alternative complement pathway activation) is characteristic. Elevated ASO titer and anti-DNase B confirm recent streptococcal infection. PSGN is a clinical diagnosis that typically does not require biopsy. Electron microscopy, when performed, reveals "subepithelial humps" (immune complex deposits). Treatment is supportive, and prognosis is excellent in children with greater than 95% complete recovery.

12. A. Toxic alcohol (methanol or ethylene glycol) ingestion. This patient presents with severe high anion gap metabolic acidosis (anion gap = 138 - 100 - 5 = 33) and a significant osmolal gap (measured osmolality 310 minus calculated osmolality 285 = 25, elevated above normal of less than 10). The combination of high anion gap metabolic acidosis with an elevated osmolal gap is classic for toxic alcohol ingestion — methanol (metabolized to formic acid, causing blindness) or ethylene glycol (metabolized to glycolic and oxalic acid, causing calcium oxalate crystal deposition and renal failure). The osmolal gap results from the unmeasured parent alcohol, while the anion gap results from the toxic metabolites. Normal glucose excludes DKA, and the osmolal gap excludes most other causes of high anion gap acidosis. Treatment includes fomepizole (alcohol dehydrogenase inhibitor) to block toxic metabolite production, hemodialysis for severe poisoning, and sodium bicarbonate for acidosis.

13. C. Syndrome of inappropriate antidiuretic hormone secretion (SIADH). SIADH is characterized by euvolemic hyponatremia from excessive ADH secretion causing inappropriate water retention and dilutional hyponatremia. Diagnostic criteria include hypo-osmolar hyponatremia (serum osmolality less than 280 mOsm/kg), inappropriately concentrated urine (urine osmolality greater than 100 mOsm/kg, typically above serum osmolality), elevated urine sodium (greater than 40 mEq/L), euvolemic status, and normal thyroid and adrenal function. Common causes include CNS disorders, pulmonary disease, malignancies (particularly small cell lung cancer), medications (SSRIs, carbamazepine, cyclophosphamide), and chronic alcohol use. Chronic alcohol use can cause SIADH through both direct CNS effects and nutritional deficiency. Treatment for chronic SIADH includes fluid restriction (first-line, 800-1000 mL/day), salt tablets, and vasopressin receptor antagonists (tolvaptan) for refractory cases.

14. B. Acute interstitial nephritis (drug-induced). Acute interstitial nephritis (AIN) is an immune-mediated inflammatory condition of the renal interstitium, most commonly caused by drugs (NSAIDs, antibiotics — penicillins, cephalosporins, sulfonamides, rifampin — PPIs, and allopurinol). The classic triad (present in only approximately 10% of cases) includes fever, maculopapular rash, and eosinophilia. Key laboratory findings include rising creatinine, eosinophiluria (detected by Hansel stain, more sensitive than Wright stain), white blood cell casts, sterile pyuria, and mild proteinuria. This presentation — new rash, fever, eosinophilia, eosinophiluria, and AKI temporally related to NSAID use — is characteristic of drug-induced AIN. Treatment requires immediate discontinuation of the offending agent, which alone leads to renal recovery in most cases. Corticosteroids may hasten recovery if no improvement occurs within 3-7 days of drug discontinuation.

15. D. IgA nephropathy (Berger disease). IgA nephropathy is the most common primary glomerulonephritis worldwide. The hallmark presentation is episodic gross hematuria occurring simultaneously with (synpharyngitic) or within 1-2 days of an upper respiratory infection — this timing distinguishes it from PSGN, which occurs 1-3 weeks after pharyngitis. Between episodes, persistent microscopic hematuria and mild proteinuria are typical. Serum IgA levels are elevated in approximately 50% of patients. Complement levels are characteristically normal (distinguishing IgA nephropathy from PSGN and lupus nephritis, which have low complement). The gold standard diagnosis is renal biopsy showing mesangial IgA deposition on immunofluorescence (pathognomonic) and mesangial

hypercellularity and matrix expansion on light microscopy. Electron microscopy reveals mesangial electron-dense deposits. Prognosis varies — approximately 30-40% progress to ESRD over 20-25 years.

16. C. Hypertensive nephrosclerosis. Hypertensive nephrosclerosis is a chronic progressive kidney disease resulting from long-standing poorly controlled hypertension causing arteriolar and glomerular damage. It is the second leading cause of ESRD in the United States (after diabetic nephropathy) and is more prevalent in African Americans. Pathologic features include arteriolar hyaline arteriolosclerosis (thickening and hyalinization of afferent arteriolar walls), intimal fibrosis, glomerulosclerosis, and tubular atrophy with interstitial fibrosis. Renal ultrasound reveals bilaterally small, echogenic kidneys with cortical thinning reflecting chronic parenchymal damage. Urinalysis shows mild proteinuria without active sediment (no RBC casts or dysmorphic RBCs). Treatment focuses on aggressive blood pressure control (target less than 130/80 mmHg), preferably with ACE inhibitors or ARBs, which provide additional renal protective effects by reducing intraglomerular pressure.

17. A. Metabolic alkalosis from loop diuretic use (contraction alkalosis). Loop diuretics (furosemide) cause metabolic alkalosis through multiple mechanisms — volume contraction concentrates existing bicarbonate (contraction alkalosis), increased distal sodium delivery stimulates sodium reabsorption in exchange for potassium and hydrogen ion secretion (generating new bicarbonate), and hypokalemia shifts hydrogen ions intracellularly and stimulates renal ammoniagenesis. The elevated arterial pH (7.52) with elevated bicarbonate (36 mEq/L) confirms metabolic alkalosis. The low potassium (3.0 mEq/L) and low chloride (85 mEq/L) reflect chloride-responsive metabolic alkalosis from diuretic use. This is classified as "chloride-responsive" because urine chloride is typically low (less than 20 mEq/L) except during active diuretic effect. Treatment includes volume repletion with normal saline, potassium chloride replacement, and reduction of diuretic dose if possible.

18. B. Hypertensive emergency with malignant hypertension. Hypertensive emergency is defined as severe hypertension (typically greater than 180/120 mmHg) with evidence of acute target organ damage. Malignant hypertension is characterized by severe hypertension with fibrinoid necrosis of arterioles producing a thrombotic microangiopathy — evidenced by schistocytes on peripheral smear, elevated LDH from microangiopathic hemolysis, and acute kidney injury from renal arteriolar damage. Papilledema (hypertensive retinopathy grade IV) is a defining feature of malignant hypertension. Other target organs include the brain (hypertensive encephalopathy, stroke), heart (acute heart failure, ACS), and aorta (dissection). Treatment requires immediate IV antihypertensive therapy (nicardipine, clevidipine, sodium nitroprusside, or labetalol) with a target of reducing MAP by no more than 25% in the first hour to avoid cerebral hypoperfusion from impaired autoregulation.

19. D. Membranous nephropathy. Membranous nephropathy is the most common cause of nephrotic syndrome in white adults and is characterized by immune complex deposition in the subepithelial space of the glomerular basement membrane. Light microscopy reveals diffuse GBM thickening. Silver stain (Jones methenamine silver) shows the classic "spike and dome" pattern — basement membrane projections (spikes) between and around subepithelial immune deposits (domes). Immunofluorescence reveals granular IgG and C3 deposition along the capillary walls. Membranous nephropathy can be

primary (anti-PLA2R antibody-mediated, approximately 70% of cases) or secondary to hepatitis B, SLE, malignancy, or medications (NSAIDs, gold, penicillamine). The hepatitis B association makes this a secondary membranous nephropathy. Low complement levels support the immune complex-mediated etiology. Treatment includes addressing the underlying cause (antiviral therapy for HBV) plus immunosuppression for severe nephrotic syndrome.

20. C. ACE inhibitor (lisinopril) and potassium-sparing diuretic (spironolactone) in the setting of CKD. Hyperkalemia is a potentially life-threatening electrolyte abnormality. The combination of an ACE inhibitor (which reduces aldosterone-mediated potassium excretion) and a potassium-sparing diuretic (spironolactone, which directly blocks the mineralocorticoid receptor and aldosterone-mediated potassium excretion) in a patient with CKD (which independently impairs potassium excretion due to reduced nephron mass) creates a triple risk for severe hyperkalemia. Additional risk factors in this patient include diabetes (hyporeninemic hypoaldosteronism/type IV RTA) and likely dietary potassium intake. ECG changes (peaked T waves, widened QRS) indicate cardiac toxicity requiring emergent treatment. Management includes discontinuing or dose-reducing the offending medications and implementing the hyperkalemia treatment protocol (IV calcium, insulin-dextrose, and potassium elimination strategies).

21. A. Increase fluid intake to maintain urine output greater than 2.5 liters per day and reduce dietary sodium and animal protein. Dietary and lifestyle modifications are the cornerstone of kidney stone prevention. For calcium oxalate stones (the most common type, approximately 80% of all stones), evidence-based recommendations include increasing fluid intake (the single most important intervention, targeting greater than 2.5 L of urine output daily to dilute stone-forming solutes), reducing dietary sodium (high sodium increases urinary calcium excretion), reducing animal protein (increases uric acid, decreases urinary citrate, and produces acid load that promotes calcium mobilization from bone), and maintaining normal calcium intake (1000-1200 mg/day — calcium restriction paradoxically increases stone risk by allowing more free oxalate absorption from the gut). Dietary oxalate restriction (spinach, nuts, chocolate, tea) is also beneficial.

22. C. Class IV (diffuse proliferative lupus nephritis). Class IV lupus nephritis is the most common (approximately 40-60% of cases) and most severe form, affecting greater than 50% of glomeruli with diffuse endocapillary and/or extracapillary proliferation. Pathognomonic features include "wire-loop" lesions on light microscopy (representing massive subendothelial immune complex deposition thickening the capillary walls), subendothelial deposits on electron microscopy, and "full house" immunofluorescence (simultaneous deposition of IgG, IgA, IgM, C3, and C1q — virtually diagnostic of lupus nephritis as no other glomerulonephritis shows this pattern). Class IV requires aggressive immunosuppressive therapy — induction with mycophenolate mofetil or IV cyclophosphamide plus corticosteroids, followed by maintenance immunosuppression with mycophenolate mofetil or azathioprine. Without treatment, Class IV lupus nephritis progresses rapidly to ESRD.

23. D. Anti-GBM disease (Goodpasture syndrome). Anti-GBM disease is caused by autoantibodies directed against the alpha-3 chain of type IV collagen in the glomerular and alveolar basement membranes, producing a pulmonary-renal syndrome with rapidly progressive glomerulonephritis (RPGN) and diffuse

alveolar hemorrhage. The term Goodpasture syndrome specifically refers to anti-GBM disease with both pulmonary and renal involvement. Circulating anti-GBM antibodies are detected by ELISA. Renal biopsy is the gold standard — linear IgG deposition along the GBM on immunofluorescence is pathognomonic (distinguishing it from the granular pattern of immune complex diseases and the pauci-immune pattern of ANCA vasculitis). Crescentic glomerulonephritis (crescents composed of proliferating parietal epithelial cells and macrophages in Bowman space) indicates RPGN. Treatment requires emergent plasmapheresis (to remove circulating anti-GBM antibodies), cyclophosphamide, and high-dose corticosteroids.

24. B. ACE inhibitor or ARB. ACE inhibitors (enalapril, lisinopril, ramipril) and ARBs (losartan, valsartan, irbesartan) are the first-line antihypertensive agents for diabetic patients with albuminuria due to their dual benefit of blood pressure reduction and nephroprotection. They reduce intraglomerular pressure by preferentially dilating the efferent arteriole (which is constricted by angiotensin II), decreasing proteinuria and slowing the progression of diabetic kidney disease. Multiple landmark trials (RENAAL, IDNT, HOPE, MICRO-HOPE) demonstrated significant renal protective effects independent of blood pressure lowering. SGLT2 inhibitors (empagliflozin, dapagliflozin) have emerged as additional nephroprotective agents in diabetic kidney disease. Target blood pressure in diabetic kidney disease is less than 130/80 mmHg. ACE inhibitors and ARBs should not be combined due to increased risk of hyperkalemia and AKI without additional benefit.

25. A. Chronic pyelonephritis (reflux nephropathy). Chronic pyelonephritis results from recurrent or persistent renal infections causing progressive tubulointerstitial inflammation, scarring, and fibrosis. The hallmark imaging findings are irregular cortical scarring overlying blunted (clubbed) calyces, best seen on CT or intravenous pyelography. Vesicoureteral reflux (VUR) is the most common predisposing factor (hence the term "reflux nephropathy"), allowing infected urine to reflux from the bladder to the kidneys. Proteus mirabilis is a urease-producing organism that alkalinizes urine and promotes struvite (magnesium ammonium phosphate) stone formation, perpetuating infection. WBC casts indicate renal parenchymal inflammation. Chronic pyelonephritis can progress to CKD and ESRD if untreated. Management includes prolonged antibiotic courses, correction of anatomic abnormalities (ureteral reimplantation for VUR), and suppressive antibiotic prophylaxis for recurrent infections.

26. D. Calciphylaxis (calcific uremic arteriolopathy). Calciphylaxis is a rare but devastating complication of severe CKD-MBD characterized by systemic arteriolar medial calcification, intimal fibrosis, and thrombotic occlusion, leading to ischemic skin necrosis. Risk factors include ESRD on dialysis, markedly elevated calcium-phosphorus product (above 55-70 mg^2/dL^2), severe hyperparathyroidism, warfarin use, obesity, and female sex. Clinical presentation includes excruciatingly painful violaceous skin lesions that progress to black eschars and non-healing ulcers, typically on adipose-rich areas (abdomen, thighs, buttocks). Mortality is extremely high (60-80% at 1 year), primarily from sepsis from infected wounds. Treatment includes intensive dialysis, sodium thiosulfate (calcification inhibitor), surgical debridement of necrotic tissue, aggressive wound care, lowering calcium-phosphorus product, and cinacalcet or parathyroidectomy for uncontrolled hyperparathyroidism.

27. B. Gitelman syndrome. Gitelman syndrome is an autosomal recessive tubulopathy caused by loss-of-function mutations in the SLC12A3 gene encoding the thiazide-sensitive sodium-chloride cotransporter (NCC) in the distal convoluted tubule. It mimics the effects of chronic thiazide diuretic use, producing hypokalemia, metabolic alkalosis, hypomagnesemia, hypocalciuria, and normal-to-low blood pressure despite elevated renin and aldosterone levels (secondary hyperaldosteronism from volume contraction). Hypocalciuria is the key feature distinguishing Gitelman syndrome from Bartter syndrome (which has normal-to-elevated urinary calcium). Gitelman syndrome typically presents in adolescents or adults with milder symptoms than Bartter syndrome. Treatment includes oral magnesium supplementation, potassium supplementation (KCl), potassium-sparing diuretics (amiloride or spironolactone), and liberal salt intake.

28. C. Myeloma cast nephropathy (myeloma kidney). Myeloma cast nephropathy is the most common cause of renal failure in multiple myeloma, resulting from precipitation of monoclonal free light chains (Bence Jones proteins) with Tamm-Horsfall protein in the distal tubular lumen, forming obstructing casts that incite a foreign body giant cell reaction. The discrepancy between negative urine dipstick protein (which detects albumin) and strongly positive sulfosalicylic acid test (which detects all proteins including light chains) is a classic clue — light chains are not detected by standard dipstick. Markedly abnormal serum free light chain ratio confirms excess monoclonal light chain production. Renal biopsy shows characteristic eosinophilic, fractured-appearing intratubular casts surrounded by multinucleated giant cells. Treatment includes chemotherapy targeting the underlying myeloma, aggressive IV hydration to reduce cast formation, correction of hypercalcemia, and avoidance of nephrotoxins.

29. A. Thin basement membrane disease (benign familial hematuria). Thin basement membrane disease (TBMD) is one of the most common causes of persistent microscopic hematuria, affecting approximately 1% of the population. It is an autosomal dominant condition caused by heterozygous mutations in COL4A3 or COL4A4 genes (encoding type IV collagen alpha-3 and alpha-4 chains of the GBM). The hallmark finding is isolated microscopic hematuria with normal renal function, minimal or no proteinuria, and a family history of hematuria without progressive renal disease. Renal biopsy reveals uniformly thin GBM (less than 250 nm, compared to normal 300-400 nm) on electron microscopy with no immune deposits. TBMD must be distinguished from Alport syndrome (X-linked or autosomal recessive), which also has thin GBM but features progressive renal failure, sensorineural hearing loss, and ocular abnormalities (anterior lenticonus). Prognosis of TBMD is excellent.

30. D. Hypertonic saline (3% NaCl) with careful monitoring. Symptomatic severe hyponatremia (sodium less than 120 mEq/L with neurologic symptoms including confusion, seizures, or obtundation) is a medical emergency requiring urgent treatment with hypertonic saline (3% NaCl). The critical safety principle is controlled correction — sodium should not be raised more than 10-12 mEq/L in the first 24 hours and no more than 18 mEq/L in 48 hours to prevent osmotic demyelination syndrome (ODS, formerly called central pontine myelinolysis), which can cause irreversible quadriplegia, pseudobulbar palsy, and locked-in syndrome. An initial bolus of 100-150 mL of 3% NaCl over 10-20 minutes can be repeated up to three times for severe symptoms. Fluid restriction alone is appropriate for chronic, asymptomatic SIADH but is inadequate for severe symptomatic hyponatremia. The underlying cause (small cell lung cancer producing ectopic ADH) should be treated concurrently.

31. C. AL amyloidosis with renal involvement. AL (primary) amyloidosis is a plasma cell dyscrasia in which monoclonal immunoglobulin light chains misfold and deposit as insoluble amyloid fibrils in tissues, causing progressive organ damage. Renal involvement (the most commonly affected organ) presents with nephrotic-range proteinuria and progressive renal insufficiency. The gold standard diagnosis is tissue biopsy demonstrating Congo red-positive deposits with apple-green birefringence under polarized light — this finding is pathognomonic for amyloidosis regardless of type. Mass spectrometry identifies the amyloid type (AL vs. AA vs. ATTR). The absence of diabetic retinopathy in a long-standing diabetic raises suspicion for an alternative diagnosis — virtually all patients with diabetic nephropathy severe enough to cause nephrotic-range proteinuria have concurrent retinopathy. Treatment targets the underlying plasma cell clone with bortezomib-based chemotherapy and consideration for autologous stem cell transplantation.

32. B. Delivery of the fetus. Preeclampsia with severe features (blood pressure greater than 160/110, thrombocytopenia, elevated liver enzymes, renal insufficiency, or cerebral/visual symptoms) and HELLP syndrome (hemolysis, elevated liver enzymes, low platelets) at 34 or more weeks gestation requires prompt delivery as the definitive treatment. The only cure for preeclampsia is delivery of the fetus and placenta, as the placenta is the underlying cause of the disease. IV magnesium sulfate is administered for seizure prophylaxis (prevention of eclampsia) but is not definitive treatment alone. IV labetalol or hydralazine is given for acute blood pressure control. For pregnancies less than 34 weeks, a brief period of expectant management with corticosteroids for fetal lung maturation may be considered if the mother is stable, but delivery should not be delayed if the clinical condition deteriorates.

33. D. Potassium citrate and allopurinol if hyperuricosuria persists. Uric acid stones account for approximately 5-10% of all kidney stones and are the only common stone type that can be dissolved medically. They form in persistently acidic urine (pH less than 5.5), as uric acid is relatively insoluble at low pH. The cornerstone of prevention and dissolution is urinary alkalinization with potassium citrate, targeting urine pH 6.0-6.5 (at which uric acid solubility increases dramatically). Risk factors include gout, metabolic syndrome, obesity, type 2 diabetes, chronic diarrhea (bicarbonate loss producing acidic urine), and myeloproliferative disorders. Allopurinol (xanthine oxidase inhibitor) is added if hyperuricosuria persists despite alkalinization and dietary modification (reducing purine-rich foods — red meat, organ meats, shellfish, beer). Adequate fluid intake to maintain dilute urine is essential for all stone types.

34. A. Continue lisinopril with close monitoring. An initial rise in serum creatinine of up to 30-35% after starting an ACE inhibitor or ARB is expected and acceptable, reflecting the hemodynamic effect of reduced intraglomerular pressure from efferent arteriolar dilation. This reduction in glomerular hyperfiltration is actually the mechanism of long-term nephroprotection. The creatinine rise typically stabilizes within 1-2 weeks. The medication should be continued with close monitoring of creatinine and potassium. ACE inhibitor or ARB should be discontinued if creatinine rises more than 30-35% above baseline, as this suggests bilateral renal artery stenosis or severe volume depletion. The potassium elevation to 5.4 mEq/L warrants monitoring but does not require drug discontinuation. Dietary potassium restriction and possible addition of a loop diuretic may help manage mild hyperkalemia while preserving the nephroprotective benefits of RAAS blockade.

35. C. Discontinue the offending drug and consider corticosteroids if no improvement. This presentation is classic for drug-induced acute interstitial nephritis — the temporal relationship to TMP-SMX exposure, classic triad of fever, rash, and eosinophilia, plus characteristic urinalysis findings (sterile pyuria, WBC casts, eosinophiluria) confirm the diagnosis. The most important step is immediate discontinuation of the offending drug, which alone leads to renal recovery in the majority of cases. If renal function does not improve within 3-7 days of drug discontinuation, a course of corticosteroids (prednisone 1 mg/kg/day for 1-2 weeks with gradual taper) may accelerate recovery and reduce the risk of chronic interstitial fibrosis. Observational studies and a recent randomized trial suggest that early corticosteroid therapy (within 7-14 days of AIN diagnosis) improves renal recovery outcomes. Renal biopsy can confirm the diagnosis when clinical suspicion is uncertain.

36. B. Uremic pericarditis. Uremic pericarditis is a serious complication of advanced uremia (inadequately dialyzed ESRD or severe acute kidney injury), occurring when uremic toxins irritate the pericardial surfaces causing fibrinous pericarditis. Clinical features include pleuritic chest pain relieved by sitting forward, pericardial friction rub (most specific physical finding), and diffuse ST elevation with PR depression on ECG. Uremic pericarditis differs from other causes of pericarditis in several important ways — it often occurs without significant pericardial effusion initially, responds to intensive dialysis rather than NSAIDs or colchicine (which are used for viral/idiopathic pericarditis), and carries a risk of hemorrhagic pericardial effusion due to uremic platelet dysfunction and heparin use during dialysis. Treatment is intensive daily hemodialysis (heparin-free to reduce hemorrhagic risk) until symptoms resolve, usually within 1-2 weeks.

37. D. Tumor lysis syndrome. Tumor lysis syndrome (TLS) is an oncologic emergency resulting from massive release of intracellular contents following rapid destruction of tumor cells (spontaneously or after chemotherapy). The classic metabolic derangements include hyperuricemia (from nucleic acid catabolism), hyperkalemia (from cellular potassium release), hyperphosphatemia (from cellular phosphorus release), and hypocalcemia (from calcium-phosphorus binding and precipitation). Acute kidney injury results from uric acid crystal deposition in the renal tubules (urate nephropathy) and calcium-phosphate precipitation. TLS is most common with rapidly proliferating, highly chemo-sensitive malignancies (Burkitt lymphoma, ALL, high-grade lymphomas) but can occur with any bulky tumor. Prevention includes aggressive IV hydration, allopurinol or rasburicase (recombinant urate oxidase), and close metabolic monitoring. Treatment of established TLS includes IV hydration, rasburicase, correction of electrolytes, and hemodialysis for severe renal failure or refractory hyperkalemia.

38. A. Bilateral renal artery stenosis causing sodium and water retention with pressure-dependent natriuresis failure. Bilateral renal artery stenosis (or stenosis of a solitary functioning kidney) creates a unique pathophysiology leading to recurrent flash pulmonary edema — a clinical scenario described as "Pickering syndrome." When both renal arteries are significantly stenotic, the kidneys cannot excrete sodium and water at normal perfusion pressures, becoming "pressure-dependent" for natriuresis. The kidneys respond to perceived hypoperfusion by activating the RAAS and retaining sodium and water, leading to volume overload that manifests as rapid-onset (flash) pulmonary edema with severe hypertension. The episodes are characteristically rapid in onset and resolution (distinguishing from

cardiogenic pulmonary edema from systolic dysfunction). Renal artery revascularization (stenting for atherosclerotic disease) can dramatically reduce episodes by restoring normal renal perfusion pressure and natriuretic capacity.

39. B. Rhabdomyolysis-induced acute kidney injury. Rhabdomyolysis is the breakdown of skeletal muscle releasing intracellular contents — myoglobin, CK, potassium, phosphorus, and uric acid — into the circulation. Causes include crush injury, extreme exertion (marathon running in heat), seizures, statins, and drug/alcohol intoxication. Myoglobin is directly nephrotoxic through tubular obstruction (myoglobin cast formation), oxidative injury (free radical generation), and renal vasoconstriction. The classic laboratory clue is a positive urine dipstick for blood (dipstick detects both hemoglobin and myoglobin peroxidase activity) with absent red blood cells on microscopy — indicating myoglobinuria rather than hematuria. Massively elevated CK (greater than 5 times normal, often exceeding 10,000-100,000 U/L) confirms rhabdomyolysis. Treatment is aggressive IV normal saline (target urine output 200-300 mL/hour) to flush myoglobin from the tubules and prevent cast formation. Electrolyte abnormalities (hyperkalemia, hyperphosphatemia, hypocalcemia) require monitoring and treatment.

40. C. Focal segmental glomerulosclerosis (FSGS). FSGS is the most common cause of nephrotic syndrome in African American adults and the second most common overall in adults (after membranous nephropathy). It is characterized by segmental (affecting part of the glomerular tuft) sclerosis and hyalinosis affecting some but not all glomeruli (focal distribution) on light microscopy, with diffuse podocyte foot process effacement on electron microscopy. FSGS can be primary (idiopathic, from circulating permeability factor) or secondary (adaptive — from obesity, reduced nephron mass, reflux nephropathy, or drug toxicity). This patient's obesity (BMI 35) is a significant risk factor for adaptive FSGS through glomerular hyperfiltration. Primary FSGS is treated with high-dose corticosteroids (though response rates are lower than MCD at approximately 50%), while secondary FSGS focuses on treating the underlying cause (weight loss, RAAS blockade, addressing the secondary factor). SGLT2 inhibitors have shown benefit in reducing proteinuria in FSGS.