COMPREHENSIVE MOCK EXAM 4

Supplemental to: Nephrology Board Review 2026-2027

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ABIM Nephrology Certification Examination — 240 questions, single-best-answer, A–E options, domain-weighted per official blueprint

Board-Review Questions

1. A 59-year-old man has an eGFR of 48 mL/min/1.73 m2 on repeated measurements over 5 months and no reversible acute illness. Which statement best describes his kidney status?

A. He has acute kidney injury because any eGFR below 60 defines an acute process

B. He does not have kidney disease unless urine albumin excretion is also elevated

C. He has kidney failure because any eGFR below 60 requires renal replacement therapy

D. He has chronic kidney disease because reduced GFR has persisted for longer than 3 months

E. He has normal kidney function because chronic kidney disease requires eGFR below 30

2. A septic patient has an increase in serum creatinine from 0.9 to 1.3 mg/dL within 36 hours. Urine output has also fallen. Which statement is correct?

A. The patient cannot have AKI until creatinine doubles from baseline

B. AKI requires at least three months of reduced kidney function by definition

C. Urine output is less informative to AKI diagnosis when creatinine is available

D. Sepsis argues against AKI because creatinine changes reflect primarily altered muscle metabolism

E. The creatinine rise meets a standard diagnostic threshold for acute kidney injury

3. A 28-year-old man develops gross hematuria within 24 hours of an upper respiratory infection. Urine microscopy shows dysmorphic red cells and RBC casts. Complement levels are normal. Which diagnosis is most likely?

A. Poststreptococcal glomerulonephritis occurring several weeks after infection with low complement

B. Membranous nephropathy causing nephrotic proteinuria without an inflammatory urine sediment

C. Minimal-change disease causing selective albuminuria and no glomerular hematuria

D. Renal papillary necrosis causing nonglomerular bleeding without dysmorphic red cells

E. IgA nephropathy with synpharyngitic glomerular hematuria

4. A kidney transplant recipient develops abrupt oliguria and graft cyanosis minutes after reperfusion. Biopsy shows widespread thrombosis. What is the most likely mechanism?

A. Acute T-cell-mediated rejection developing within minutes from newly generated donor-supportive T cells

B. BK virus nephropathy caused by immediate viral replication during graft reperfusion

C. Hyperacute rejection from preformed recipient antibodies against donor antigens

D. Chronic antibody-mediated rejection caused by years of transplant glomerulopathy

E. Calcineurin-inhibitor toxicity because tacrolimus causes instantaneous graft thrombosis at reperfusion

5. A 46-year-old man has blood pressure 168/102 mm Hg despite adherence to three antihypertensive agents, including a diuretic. Potassium is 3.1 mEq/L. Which secondary cause should be screened for first?

A. Pheochromocytoma using urinary catecholamines primarily because many resistant hypertension case is catecholamine mediated

B. Renal artery stenosis using invasive angiography before any biochemical or clinical assessment

C. Cushing syndrome using random cortisol because hypokalemia specifically argues against mineralocorticoid excess

D. Coarctation of the aorta using brain MRI because arm hypertension originates from cerebral vasoconstriction

E. Primary aldosteronism using a plasma aldosterone-to-renin assessment under appropriate conditions

6. A 55-year-old man with septic shock has pH 7.24, PaCO2 25 mm Hg, and bicarbonate 11 mEq/L. Which PaCO2 would be expected if respiratory compensation for a simple metabolic acidosis were appropriate?

A. Approximately 10 mm Hg, indicating the normal response to any degree of metabolic acidosis

B. Approximately 40 mm Hg, because metabolic acidosis should not change alveolar ventilation

C. Approximately 55 mm Hg, reflecting compensatory respiratory acidosis from bicarbonate loss

D. Approximately 25 mm Hg, consistent with the expected Winter-formula compensation

E. Approximately 70 mm Hg, because severe acidemia normally suppresses respiratory drive

7. A 68-year-old man with small-cell lung cancer has nausea and mild confusion. Serum sodium is 118 mEq/L, serum osmolality 254 mOsm/kg, urine osmolality 540 mOsm/kg, and urine sodium 68 mEq/L. He is clinically euvolemic, and thyroid and adrenal testing are normal. Which diagnosis best explains the hyponatremia?

A. Primary polydipsia with maximally dilute urine from excess water intake

B. Hypovolemic hyponatremia with sodium avidity from gastrointestinal losses

C. Hypervolemic hyponatremia caused by advanced congestive heart failure

D. Adrenal insufficiency causing cortisol deficiency and renal sodium wasting

E. Syndrome of inappropriate antidiuresis due to ectopic ADH activity

8. A patient with CKD has an eGFR of 38 mL/min/1.73 m2 and urine albumin-to-creatinine ratio of 520 mg/g. Which risk feature most strongly indicates a high likelihood of progression?

A. Severely increased albuminuria in addition to the reduced eGFR

B. A normal serum sodium concentration despite reduced glomerular filtration

C. A normal hemoglobin concentration while the patient remains clinically stable

D. A low-normal serum uric acid concentration without a history of gout

E. Absence of microscopic hematuria on a single urine sediment examination

9. A patient with albuminuric CKD starts an ACE inhibitor. Which intrarenal hemodynamic change is expected?

A. Afferent arteriolar constriction caused by direct blockade of renal prostaglandin synthesis

B. Efferent arteriolar dilation that lowers intraglomerular pressure and may cause a modest initial fall in GFR

C. Efferent arteriolar constriction that raises glomerular capillary pressure and increases albumin filtration

D. Uniform dilation of both arterioles with no change in glomerular capillary hydrostatic pressure

E. Direct mesangial contraction that increases filtration surface area and eliminates proteinuria

10. A patient with vomiting and hypotension has AKI, bland urine sediment, and fractional excretion of sodium below 1%. What is the most likely process?

A. Established acute tubular necrosis because intact tubules cannot reabsorb sodium during hypoperfusion

B. Acute interstitial nephritis because allergic interstitial inflammation often produces a low FENa

C. Bilateral cortical necrosis because irreversible vascular injury causes a bland sediment and low sodium excretion

D. Postrenal obstruction because FENa below 1% specifically strongly suggests bilateral ureteral blockage

E. Prerenal azotemia from reduced renal perfusion with avid tubular sodium reabsorption

11. A 49-year-old woman has recurrent calcium stones, serum calcium 11.2 mg/dL, low phosphate, and elevated intact PTH. Which diagnosis is most likely?

A. Secondary hyperparathyroidism from chronic kidney disease causing low or normal serum calcium

B. Humoral hypercalcemia of malignancy causing suppressed PTH and low phosphate

C. Primary hyperparathyroidism causing hypercalcemia and increased urinary calcium stone risk

D. Familial hypocalciuric hypercalcemia causing very low urinary calcium excretion and few stones

E. Vitamin D deficiency causing hypocalcemia with compensatory elevation of parathyroid hormone

12. A 63-year-old woman with diabetic CKD has persistent albuminuria and hypertension. Which antihypertensive class has a specific kidney-protective role by reducing intraglomerular pressure and albuminuria?

A. A direct vasodilator used alone without renin-angiotensin system blockade

B. A short-acting dihydropyridine calcium-channel blocker used primarily when symptoms occur

C. An alpha-1 adrenergic blocker selected primarily for urinary symptoms rather than albuminuria

D. An ACE inhibitor or angiotensin receptor blocker titrated as tolerated

E. A central alpha agonist used as first-line monotherapy specifically to reduce glomerular protein leak

13. A child develops edema, cola-colored urine, hypertension, and low C3 about 2 weeks after streptococcal pharyngitis. Which diagnosis is most likely?

A. IgA nephropathy because hematuria usually begins simultaneously with the respiratory infection

B. Minimal-change disease because low complement and RBC casts are typical nephrotic findings

C. Anti-GBM disease because preceding streptococcal infection specifically triggers linear IgG deposition

D. Poststreptococcal glomerulonephritis caused by immune-complex glomerular injury after infection

E. Alport syndrome because inherited collagen defects cause transient low complement after pharyngitis

14. A 45-year-old woman develops fever, rash, eosinophilia, pyuria, and rising creatinine 10 days after starting a proton-pump inhibitor. Which diagnosis is most likely?

A. Acute tubular necrosis caused by prolonged hypotension with muddy brown granular casts

B. Drug-induced acute interstitial nephritis causing inflammatory injury of the renal interstitium

C. Minimal-change disease causing isolated nephrotic proteinuria without interstitial inflammation

D. Postinfectious glomerulonephritis causing low complement and dysmorphic red blood cells

E. Renal artery stenosis causing hemodynamic GFR reduction without inflammatory urine findings

15. A kidney transplant recipient 3 weeks after surgery has rising creatinine. Biopsy shows interstitial inflammation and tubulitis without major vascular thrombosis. Which diagnosis is most likely?

A. Hyperacute rejection because preformed antibodies usually present several weeks after transplantation

B. Chronic antibody-mediated rejection because transplant glomerulopathy develops within the first few days

C. BK virus nephropathy because early tubulitis often strongly suggests polyomavirus infection

D. Acute T-cell-mediated rejection with interstitial inflammation and tubulitis

E. Recurrent diabetic nephropathy because nodular sclerosis develops within weeks in many diabetic recipient

16. A 36-year-old woman has severe hypertension, an abdominal bruit, and a rise in creatinine after starting an ACE inhibitor. Imaging shows a beaded right renal artery. What is the most likely diagnosis?

A. Atherosclerotic renal artery stenosis caused by ostial plaque in an elderly patient with diffuse vascular disease

B. Primary aldosteronism causing a beaded renal artery appearance through chronic vasoconstriction

C. Pheochromocytoma causing fixed structural narrowing of the renal artery by catecholamine excess

D. Takayasu arteritis because fibromuscular dysplasia rarely affects the renal circulation

E. Fibromuscular dysplasia causing renovascular hypertension in a younger woman

17. A patient with albuminuric CKD starts an ACE inhibitor. Creatinine rises from 1.5 to 1.8 mg/dL and potassium remains normal. Blood pressure improves and volume status is stable. What is the best next step?

A. Stop the ACE inhibitor immediately because any creatinine increase strongly suggests progressive structural kidney injury

B. Add an ARB to restore filtration pressure and reverse the creatinine increase

C. Begin high-dose NSAID therapy to constrict the afferent arteriole and normalize GFR

D. Start dialysis because a creatinine increase after ACE inhibition indicates irreversible kidney failure

E. Continue the ACE inhibitor and monitor because a modest creatinine rise can reflect the expected hemodynamic effect

18. A patient receiving loop diuretics develops AKI after gastrointestinal fluid loss. Which urinary index may be more useful than FENa for supporting prerenal physiology?

A. Fractional excretion of phosphate because it is strongly suggestive for prerenal AKI in most patients

B. Urinary glucose concentration because prerenal azotemia causes obligate glycosuria

C. Fractional excretion of urea, which is less directly affected by loop-diuretic natriuresis

D. Urine protein electrophoresis because volume depletion produces monoclonal light chains

E. Urinary eosinophils because they are highly supportive for prerenal physiology

19. A patient has sodium 140 mEq/L, chloride 104 mEq/L, and bicarbonate 12 mEq/L. Albumin is normal. What is the calculated anion gap?

A. 12 mEq/L, indicating a normal-anion-gap metabolic acidosis

B. 8 mEq/L, indicating isolated respiratory alkalosis rather than metabolic acidosis

C. 36 mEq/L, indicating a mixed metabolic alkalosis and respiratory acidosis

D. 48 mEq/L, indicating severe hyperchloremic metabolic acidosis

E. 24 mEq/L, indicating a high-anion-gap metabolic acidosis

20. A 24-year-old woman with schizophrenia drinks more than 8 L of water daily. Serum sodium is 125 mEq/L and serum osmolality is low. Urine osmolality is 62 mOsm/kg. Which mechanism is most likely responsible?

A. Persistent nonosmotic ADH release causing concentrated urine despite hypo-osmolality

B. Renal salt wasting with secondary ADH release from effective volume depletion

C. Excess free-water intake exceeding renal excretory capacity with suppressed ADH

D. Deficient cortisol secretion increasing ADH and impairing free-water excretion

E. Severe reduction in glomerular filtration preventing generation of dilute urine

21. A 58-year-old man with type 2 diabetes, eGFR 52 mL/min/1.73 m2, and persistent albuminuria is already taking an ARB. Which additional drug class can reduce CKD progression risk through tubuloglomerular feedback and other kidney-protective effects?

A. An SGLT2 inhibitor if no contraindication is present

B. A sulfonylurea because insulin secretion directly lowers intraglomerular pressure

C. A nonselective NSAID because afferent vasoconstriction reduces albumin filtration safely

D. A loop diuretic because increased urine volume independently prevents nephron loss

E. A proton-pump inhibitor because gastric acid suppression reduces renal inflammatory injury

22. A 70-year-old man with diabetes develops AKI, hematuria, proteinuria, and low complement during active Staphylococcus aureus bacteremia. Kidney biopsy shows proliferative immune-complex GN with IgA-dominant deposits. What is the most likely diagnosis?

A. Primary IgA nephropathy unrelated to infection because IgA deposits exclude infection-related disease

B. Infection-related glomerulonephritis associated with active staphylococcal infection

C. Anti-GBM disease because bacterial infection causes linear basement-membrane antibody deposition

D. Minimal-change disease because diabetes predisposes to podocyte effacement during bacteremia

E. Membranous nephropathy because subepithelial deposits are the usual cause of infection-associated nephritis

23. A kidney transplant recipient has rising creatinine, new donor-specific HLA antibodies, microvascular inflammation, and C4d staining in peritubular capillaries. What is the diagnosis?

A. Acute T-cell-mediated rejection because donor-supportive antibodies and C4d are unrelated to humoral injury

B. BK virus nephropathy because C4d deposition specifically identifies polyomavirus replication

C. Calcineurin-inhibitor toxicity because arteriolar vasoconstriction produces donor-supportive antibodies

D. Recurrent FSGS because podocyte injury is characterized by peritubular capillary C4d deposition

E. Antibody-mediated rejection due to donor-specific antibody injury of the allograft microcirculation

24. A patient develops AKI after prolonged hypotension. Urine microscopy shows numerous granular brown casts and renal tubular epithelial cells. Which diagnosis is most likely?

A. Prerenal azotemia with structurally intact tubules and an largely bland sediment

B. Acute tubular injury from ischemia, classically associated with muddy brown granular casts

C. Acute glomerulonephritis causing dysmorphic red cells and red-cell casts

D. Acute interstitial nephritis causing white-cell casts and hypersensitivity findings

E. Postrenal obstruction causing primarily hexagonal crystals without tubular epithelial cells

25. A patient with type 2 diabetes and albuminuric CKD has persistent albuminuria despite maximally tolerated ACE-inhibitor therapy and an SGLT2 inhibitor. Potassium is normal. Which additional therapy may further reduce kidney and cardiovascular risk?

A. A nonsteroidal mineralocorticoid receptor antagonist such as finerenone

B. A second renin-angiotensin blocker added to create complete dual-system blockade

C. Chronic high-dose NSAID therapy to lower glomerular filtration of albumin

D. A potassium-sparing epithelial sodium-channel blocker used primarily to increase albumin excretion

E. Routine glucocorticoid therapy because diabetic kidney disease is primarily immune mediated

26. A 72-year-old man with diffuse atherosclerosis has resistant hypertension and recurrent flash pulmonary edema. Bilateral renal artery stenosis is suspected. Which feature makes renovascular disease clinically important here?

A. Stable mild hypertension controlled on one medication without kidney dysfunction or volume symptoms

B. A normal creatinine after ACE-inhibitor therapy because this confirms severe bilateral stenosis

C. Isolated microscopic hematuria with normal blood pressure and no evidence of vascular disease

D. A low serum uric acid concentration because this is supportive for renal artery atherosclerosis

E. Recurrent sudden pulmonary edema with difficult blood-pressure control and possible bilateral renal ischemia

27. A patient with diarrhea has bicarbonate 15 mEq/L and a normal anion gap. Urine sodium is 25 mEq/L, urine potassium 20 mEq/L, and urine chloride 70 mEq/L. Which interpretation of the urine anion gap is most appropriate?

A. A positive urine anion gap strongly suggests distal renal tubular acidosis with impaired ammonium excretion

B. A negative urine anion gap suggests appropriate ammonium excretion and an extrarenal bicarbonate loss

C. A negative urine anion gap indicates severe proximal renal tubular acidosis from bicarbonaturia

D. The urine anion gap is useful primarily in high-anion-gap acidosis and is less useful for distinguishing renal from gastrointestinal bicarbonate loss

E. A negative urine anion gap indicates absent renal acid excretion and mandates alkali cessation

28. A patient with cirrhosis and reduced effective arterial volume develops AKI after starting ibuprofen. Which drug effect is responsible?

A. Cyclooxygenase inhibition causes selective efferent dilation similar to ACE-inhibitor therapy

B. NSAIDs activate renal prostaglandins and cause excessive afferent dilation with glomerular hyperfiltration

C. Ibuprofen blocks aldosterone receptors and produces expected renal salt wasting in cirrhosis

D. NSAIDs inhibit SGLT2 and cause glycosuric osmotic diuresis leading to isolated prerenal azotemia

E. Cyclooxygenase inhibition reduces prostaglandin-mediated afferent arteriolar dilation and renal blood flow

29. A 52-year-old man has nephrotic syndrome and a high serum anti-PLA2R antibody titer. Secondary evaluation is unrevealing. Which diagnosis is most likely?

A. Minimal-change disease because PLA2R antibodies are a marker of podocyte foot-process effacement

B. Primary FSGS because PLA2R antibodies target the slit diaphragm protein nephrin

C. Primary membranous nephropathy associated with PLA2R autoantibodies

D. Amyloidosis because PLA2R antibody is a marker of Congo-red-positive fibril deposition

E. Diabetic kidney disease because PLA2R antibody is produced by chronic hyperglycemia

30. A 71-year-old man with CKD has progressive fatigue. Hemoglobin is 9.4 g/dL, MCV is normal, reticulocyte count is low, ferritin and transferrin saturation are adequate, and there is no bleeding. What is the principal mechanism of his anemia?

A. Increased splenic sequestration caused directly by elevated serum creatinine

B. expected folate loss in urine caused by many stage of chronic kidney disease

C. Autoimmune hemolysis triggered by accumulation of urea in circulating erythrocytes

D. Reduced renal erythropoietin production causing a hypoproliferative normocytic anemia

E. Excess endogenous erythropoietin causing marrow exhaustion and reticulocytopenia

31. A patient with septic shock develops AKI despite adequate initial fluid resuscitation. Which pathophysiology best describes sepsis-associated AKI?

A. Simple bilateral renal artery occlusion that occurs in nearly many patient with septic shock

B. Complex microcirculatory, inflammatory, and cellular dysfunction that can occur even without sustained global renal ischemia

C. Pure postrenal obstruction caused by inflammatory debris in both ureters

D. An isolated immune-complex glomerulonephritis that explains most septic AKI

E. expected cortical necrosis caused whenever mean arterial pressure briefly falls below normal

32. A 58-year-old man with chronic alcohol use eats almost no solid food and drinks several liters of beer daily. He presents with sodium 116 mEq/L, urine osmolality 82 mOsm/kg, and low urine solute excretion. What is the principal cause of his hyponatremia?

A. High aldosterone levels causing renal retention of sodium-free water in the collecting duct

B. Autonomous ADH secretion maintaining a persistently hypertonic urine despite low serum osmolality

C. Renal sodium loss from cerebral salt wasting producing chronic extracellular volume depletion

D. Low daily solute intake limiting the volume of electrolyte-free water that can be excreted

E. Osmotic movement of water from cells caused by severe hyperglycemia and extracellular hypertonicity

33. A stable kidney transplant recipient develops a progressive creatinine rise and biopsy shows striped interstitial fibrosis and arteriolar hyalinosis. Tacrolimus trough levels have often been high. What is the most likely cause?

A. Acute pyelonephritis because bacterial infection produces arteriolar hyalinosis and striped fibrosis

B. Recurrent minimal-change disease because podocyte effacement causes chronic arteriolar lesions

C. Hyperacute rejection because preformed antibody injury presents years later as striped fibrosis

D. Renal vein thrombosis because venous occlusion causes chronic calcineurin-supportive histologic changes

E. Chronic calcineurin-inhibitor nephrotoxicity with arteriolar and interstitial injury

34. A patient with advanced CKD is being considered for an erythropoiesis-stimulating agent. Which treatment goal is most appropriate?

A. Normalize hemoglobin to the upper end of the healthy range in many patient regardless of risk

B. Target a hemoglobin above 15 g/dL to prevent cardiovascular disease associated with CKD

C. Use the lowest ESA exposure needed to reduce transfusion burden and symptoms rather than normalize hemoglobin

D. Use ESA therapy before checking iron status because iron availability does not affect response

E. Avoid most ESA therapy because erythropoietin deficiency does not contribute to CKD-associated anemia

35. A 43-year-old woman has episodic headaches, diaphoresis, palpitations, and paroxysmal severe hypertension. Which initial biochemical test is most appropriate?

A. Measure plasma aldosterone alone without renin because catecholamine tumors suppress most aldosterone production

B. Measure serum erythropoietin because pheochromocytoma is primarily a red-cell production disorder

C. Measure urine protein electrophoresis because monoclonal proteins cause episodic adrenergic symptoms

D. Measure serum renin primarily because pheochromocytoma often produces a strongly suggestive renin concentration

E. Measure plasma free or urinary fractionated metanephrines to screen for catecholamine excess

36. A 38-year-old man has recurrent calcium phosphate stones, urine pH 6.5, low urinary citrate, hypokalemia, and a normal-anion-gap metabolic acidosis. Which treatment is most appropriate to reduce stone recurrence?

A. Potassium citrate to correct hypocitraturia and provide alkali while replacing potassium

B. Ammonium chloride to acidify the urine despite the underlying systemic metabolic acidosis

C. High-dose sodium bicarbonate primarily because sodium loading reduces urinary calcium excretion

D. Allopurinol as the primary therapy for calcium phosphate stones with distal renal tubular acidosis

E. Fluid restriction to reduce urinary calcium delivery and decrease supersaturation of calcium salts

37. A patient with septic shock requires crystalloid resuscitation and has no specific indication for a high-chloride solution. Which fluid is often preferred to reduce hyperchloremic acid-base effects?

A. Repeated large volumes of 3% saline because hypertonic fluid is the routine sepsis resuscitation standard

B. Sterile water intravenously because it provides free water without chloride

C. Dextrose 5% water as the primary intravascular volume expander for septic shock

D. Sodium bicarbonate solution at high concentration as the standard first-line resuscitation fluid

E. A balanced isotonic crystalloid with lower chloride exposure for volume resuscitation

38. A patient with membranous nephropathy develops sudden flank pain, gross hematuria, and worsening proteinuria. Which complication should be suspected?

A. Renal vein thrombosis related to the hypercoagulable state of severe nephrotic syndrome

B. Papillary necrosis caused by expected medullary ischemia in most membranous nephropathy

C. Renal artery fibromuscular dysplasia caused by subepithelial immune deposits

D. Acute cortical necrosis caused by isolated albuminuria without systemic hypotension

E. Nephrolithiasis because many patient with membranous nephropathy develops hypercalciuria

39. A hemodialysis patient with anemia has low transferrin saturation and low ferritin. Which intervention is most likely to improve erythropoietic response?

A. Stopping iron because dialysis causes iron overload in nearly most patients

B. Immediate splenectomy because iron deficiency in dialysis is usually due to hypersplenism

C. High-dose folic acid alone because iron cannot be utilized in chronic kidney disease

D. Iron repletion, often using intravenous iron in the hemodialysis setting

E. Red-cell transfusion at many dialysis session regardless of symptoms or transplant candidacy

40. A 34-year-old woman has nephrolithiasis, non-anion-gap metabolic acidosis, urine pH 6.2, and hypokalemia. Which disorder is most likely?

A. Proximal renal tubular acidosis with intact distal acidification once bicarbonate is depleted

B. Type 4 renal tubular acidosis with hypoaldosteronism and persistent hyperkalemia

C. Chronic diarrhea with appropriate urinary acidification and a low urine pH

D. Respiratory alkalosis with compensatory bicarbonate reduction and no primary renal lesion

E. Distal renal tubular acidosis with impaired hydrogen secretion and persistently alkaline urine

41. A 62-year-old woman with decades of heavy analgesic use has slowly progressive CKD, sterile pyuria, and imaging showing small irregular kidneys with papillary calcifications. What is the most likely diagnosis?

A. Autosomal dominant polycystic kidney disease causing massively enlarged bilateral cystic kidneys

B. Minimal-change disease causing normal-sized kidneys and isolated heavy proteinuria

C. Renal amyloidosis causing nephrotic syndrome and enlarged kidneys without papillary injury

D. Acute cortical necrosis caused by obstetric catastrophe and abrupt irreversible kidney failure

E. Analgesic nephropathy with chronic tubulointerstitial injury and renal papillary necrosis

42. A kidney transplant recipient 5 months after surgery develops rising creatinine. Plasma BK viral load is high and biopsy shows tubulointerstitial nephritis with viral inclusions. What is the preferred initial strategy?

A. Increase tacrolimus and mycophenolate because BK nephropathy reflects inadequate immunosuppression

B. Begin high-dose glucocorticoids automatically because viral inclusions prove acute cellular rejection

C. Reduce overall immunosuppression to allow immune control of BK polyomavirus

D. Treat with routine acyclovir because BK polyomavirus is reliably eradicated by standard herpesvirus therapy

E. Perform immediate graft nephrectomy because BK infection rarely improves enough with immunosuppression adjustment alone

43. A patient with stage 4 CKD has phosphate retention, low-normal calcium, rising FGF23, reduced calcitriol, and elevated PTH. What process best explains the elevated PTH?

A. Secondary hyperparathyroidism driven by phosphate retention, reduced calcitriol, and disordered calcium sensing

B. Primary hyperparathyroidism from a parathyroid adenoma unrelated to kidney function

C. Autonomous tertiary hyperparathyroidism that occurs before any prolonged secondary stimulation

D. Suppressed parathyroid hormone secretion caused by reduced renal phosphate excretion

E. Increased calcitriol synthesis by the failing kidney causing excessive intestinal calcium absorption

44. A patient receives very large volumes of 0.9% saline and develops a normal-anion-gap metabolic acidosis. What is the mechanism?

A. Saline contains large amounts of lactate that are converted into hydrochloric acid

B. Sodium chloride directly consumes albumin and creates a high-anion-gap acidosis

C. High chloride delivery lowers the plasma strong-ion difference and produces hyperchloremic acidosis

D. The solution causes ketoacid production by preventing most insulin secretion

E. Isotonic saline contains no chloride, so the acidosis must be due to respiratory failure

45. A 72-year-old woman taking hydrochlorothiazide presents after several days of vomiting with sodium 121 mEq/L. She is orthostatic, has dry mucous membranes, and urine sodium is 8 mEq/L after the thiazide is held. What is the most appropriate initial therapy?

A. Fluid restriction alone to reduce water intake while maintaining the current sodium deficit

B. Desmopressin alone to increase collecting-duct water permeability and lower urine output

C. Tolvaptan therapy to block vasopressin receptors in a clearly hypovolemic patient

D. Isotonic saline to restore effective circulating volume and suppress nonosmotic ADH

E. Hypertonic saline infusion to rapidly normalize serum sodium to the normal range

46. A patient with confirmed pheochromocytoma is being prepared for surgery. Which pharmacologic sequence is essential?

A. Start a beta blocker first to prevent tachycardia before any alpha blockade is given

B. Use beta blockade alone because unopposed alpha stimulation lowers rather than raises blood pressure

C. Avoid most preoperative blood-pressure treatment because manipulation of the tumor suppresses catecholamine release

D. Begin an ACE inhibitor primarily because catecholamine-mediated vasoconstriction is largely angiotensin dependent

E. Establish alpha-adrenergic blockade before adding beta blockade if tachycardia persists

47. A 30-year-old adult develops abrupt nephrotic syndrome after a viral illness. Kidney biopsy shows normal glomeruli by light microscopy and diffuse podocyte foot-process effacement by electron microscopy. What is the diagnosis?

A. Minimal-change disease causing a podocytopathy with diffuse foot-process effacement

B. Membranous nephropathy causing subepithelial immune deposits and basement-membrane spikes

C. Focal segmental glomerulosclerosis causing segmental scar lesions on light microscopy

D. Amyloidosis causing Congo-red-positive fibrils within glomeruli and vessels

E. Diabetic nephropathy causing mesangial expansion and nodular glomerulosclerosis

48. A dialysis patient has severe bone pain, very high PTH, and high bone turnover. Which skeletal disorder is most consistent with these findings?

A. Adynamic bone disease caused by markedly suppressed PTH and low bone remodeling

B. Osteomalacia caused primarily by iron deficiency without any disturbance in mineralization

C. Avascular necrosis caused by uremia without relation to parathyroid hormone activity

D. Osteopetrosis caused by reduced osteoclast function from high circulating PTH levels

E. Osteitis fibrosa from high-turnover secondary hyperparathyroid bone disease

49. A child has growth failure, glucosuria with normal serum glucose, aminoaciduria, phosphaturia, hypokalemia, and non-anion-gap metabolic acidosis. Which acid-base disorder is present?

A. Distal renal tubular acidosis caused by isolated failure of collecting-duct hydrogen secretion

B. Proximal renal tubular acidosis caused by generalized proximal tubular dysfunction

C. Type 4 renal tubular acidosis caused by aldosterone deficiency and reduced ammoniagenesis

D. Respiratory alkalosis caused by chronic hyperventilation with secondary renal bicarbonate loss

E. High-anion-gap metabolic acidosis caused by retention of organic acids in advanced kidney failure

50. A patient with severe rhabdomyolysis after crush injury has dark urine, very high creatine kinase, potassium 6.2 mEq/L, and rising creatinine. Urine dipstick is strongly positive for blood but microscopy shows few red cells. What is causing the positive dipstick?

A. Intact glomerular red blood cells that are most lysed before urine microscopy

B. Free hemoglobin from intravascular hemolysis because creatine kinase elevation argues against muscle injury

C. Bilirubin because urine heme dipsticks primarily detect conjugated bile pigments

D. Uric acid because urate crystals produce a false-positive heme reaction in most acidic urine

E. Myoglobin pigment released from injured muscle and filtered into the urine

51. A patient with diabetic CKD starts an SGLT2 inhibitor and has a small early decline in eGFR. Which mechanism contributes to this expected effect?

A. Reduced distal sodium delivery suppresses tubuloglomerular feedback and maximally dilates the afferent arteriole

B. Increased distal sodium delivery restores tubuloglomerular feedback and increases afferent arteriolar tone

C. Direct efferent arteriolar constriction raises intraglomerular pressure and causes progressive hyperfiltration

D. Blockade of vasopressin V2 receptors causes an aquaresis that directly reduces glomerular capillary pressure

E. Inhibition of ENaC in the collecting duct prevents lithium uptake and lowers filtration through natriuresis

52. A CMV-seronegative recipient receives a kidney from a CMV-seropositive donor. Which strategy is commonly used because this is a high-risk pairing?

A. No CMV prevention because seronegative recipients has limited risk of donor-derived cytomegalovirus without prior recipient immunity

B. Live attenuated CMV vaccination immediately after transplantation while immunosuppression is highest

C. Routine antibacterial prophylaxis alone because CMV is prevented by trimethoprim-sulfamethoxazole

D. Increase mycophenolate dose because stronger immunosuppression lowers the risk of CMV replication

E. Antiviral prophylaxis or intensive preemptive monitoring with a CMV-active agent according to transplant protocol

53. A long-term dialysis patient has low PTH, recurrent hypercalcemia after calcium-based binders, and suspected low-turnover bone disease. Which diagnosis is most likely?

A. Osteitis fibrosa from uncontrolled secondary hyperparathyroidism and high bone turnover

B. Adynamic bone disease from excessive suppression of parathyroid-driven bone turnover

C. Tertiary hyperparathyroidism with autonomous PTH excess and severe high-turnover disease

D. Vitamin D deficiency rickets caused by high PTH and increased osteoid mineralization

E. Metastatic bone disease because low PTH argues against any renal osteodystrophy

54. A patient with obesity and reduced nephron mass has subnephrotic proteinuria and biopsy showing focal segmental glomerulosclerosis with glomerulomegaly and relatively limited foot-process effacement. Which form is most likely?

A. Primary FSGS because adaptive forms often have complete diffuse foot-process effacement

B. Secondary adaptive FSGS from glomerular hyperfiltration rather than primary circulating-factor disease

C. Minimal-change disease because glomerulomegaly argues against any form of focal segmental sclerosis

D. Membranous nephropathy because obesity causes subepithelial immune-complex deposition

E. Anti-GBM disease because nephron loss induces linear IgG binding to the basement membrane

55. A 51-year-old man has obesity, loud snoring, daytime somnolence, and resistant hypertension. Which secondary contributor is most likely?

A. Primary polydipsia causing nocturnal free-water excess and sustained sympathetic suppression

B. Central diabetes insipidus causing nocturnal polyuria and chronic extracellular volume expansion

C. Adrenal insufficiency causing cortisol deficiency with persistent sodium retention and hypertension

D. Bartter syndrome causing salt retention, hypokalemia, and increased extracellular volume

E. Obstructive sleep apnea with recurrent nocturnal hypoxemia and sympathetic activation

56. A 46-year-old woman with chronic hyponatremia from SIADH has a seizure and a serum sodium of 108 mEq/L. Which immediate treatment is most appropriate?

A. Rapid continuous 3% saline until the serum sodium reaches 140 mEq/L within several hours

B. Large-volume isotonic saline without sodium monitoring because SIADH will correct predictably

C. Oral free-water loading to prevent osmotic demyelination before treating the seizure

D. Carefully monitored boluses of 3% saline to raise sodium enough to reverse severe symptoms

E. A vasopressin antagonist as the sole emergency therapy for active hyponatremic seizures

57. A patient with ESKD develops exquisitely painful retiform purpura and necrotic plaques on the thighs. Calcium-phosphate balance has been difficult to control. What diagnosis is most likely?

A. Cholesterol embolization causing blue toes after recent arterial instrumentation

B. Warfarin skin necrosis caused primarily by transient protein C depletion in a patient without CKD

C. Calciphylaxis involving calcification and thrombosis of small dermal and subcutaneous vessels

D. Pyoderma gangrenosum caused by neutrophilic inflammation and typically painless ulceration

E. Uremic frost caused by crystallized urea on intact skin without vascular thrombosis

58. A patient with rhabdomyolysis is oliguric and develops refractory hyperkalemia, severe acidosis, and pulmonary edema. What is the next step?

A. Continue fluids alone regardless of pulmonary edema because dialysis is less effective than other strategies for potassium removal

B. Give calcium primarily because membrane stabilization permanently corrects total-body potassium excess

C. Initiate kidney replacement therapy because multiple life-threatening complications are refractory to medical management

D. Delay dialysis until creatine kinase normalizes because muscle injury makes dialysis ineffective

E. Use sodium polystyrene alone because gastrointestinal potassium removal is adequate for most severe cases

59. A 56-year-old man with gout has radiolucent stones and urine pH 5.0. Which long-term intervention most directly increases uric acid solubility?

A. Urine acidification with ammonium chloride to keep uric acid in its nonionized form

B. Urine alkalinization with potassium citrate to raise urine pH and dissolve uric acid

C. Calcium supplementation to bind urate in the gut and prevent urinary uric acid excretion

D. Thiazide therapy to lower urinary calcium as the principal treatment for pure uric acid stones

E. Restriction of oral fluids to increase urinary concentration and reduce crystal nucleation

60. A kidney transplant recipient is prescribed trimethoprim-sulfamethoxazole for several months after transplantation. What infection is this primarily intended to prevent?

A. Cytomegalovirus disease because trimethoprim directly blocks CMV DNA polymerase

B. BK polyomavirus nephropathy because sulfonamides eradicate latent polyomavirus

C. Epstein-Barr virus-associated PTLD because trimethoprim prevents B-cell transformation

D. Invasive aspergillosis because standard-dose trimethoprim is the preferred mold-active prophylaxis

E. Pneumocystis jirovecii pneumonia, with additional protection against selected bacterial infections

61. A 67-year-old man with diabetic kidney disease has potassium 6.0 mEq/L, bicarbonate 18 mEq/L, and a normal anion gap. Urine pH is 5.0. Plasma renin and aldosterone are low. What is the most likely diagnosis?

A. Type 4 renal tubular acidosis from hyporeninemic hypoaldosteronism

B. Distal renal tubular acidosis with failure to lower urine pH below 5.5

C. Proximal renal tubular acidosis with isolated bicarbonate wasting and severe hypokalemia

D. Chronic respiratory alkalosis causing compensatory bicarbonate loss and secondary hyperkalemia

E. Diarrheal bicarbonate loss with increased renal ammonium excretion and low serum potassium

62. A patient with advanced CKD has persistent metabolic acidosis attributed to reduced ammonium generation and acid excretion. What is a recognized consequence of untreated chronic metabolic acidosis?

A. Reduced protein breakdown and increased bone mineralization from chronic systemic acid retention

B. Persistent respiratory alkalosis that substantially prevents acid-mediated tissue injury

C. Increased muscle catabolism and bone buffering with potential acceleration of CKD complications

D. expected hypercalcemia caused by suppression of most skeletal buffering mechanisms

E. Elimination of potassium wasting because metabolic acidosis often lowers distal potassium secretion

63. A patient with rapidly progressive kidney failure, hematuria, and pulmonary hemorrhage has circulating anti-GBM antibodies. Which treatment is most appropriate?

A. Supportive care alone because anti-GBM antibodies usually disappear before irreversible kidney injury occurs

B. Rituximab monotherapy without antibody removal because plasma exchange has limited ability to reduce circulating antibodies quickly

C. Plasma exchange plus glucocorticoids and cyclophosphamide to remove antibody and suppress new production

D. High-dose NSAIDs to reduce glomerular inflammation while preserving basement-membrane antibody binding

E. ACE-inhibitor monotherapy because lowering intraglomerular pressure eliminates pulmonary hemorrhage

64. A patient with bulky leukemia develops tumor lysis syndrome despite prophylaxis and has severe hyperkalemia, hyperphosphatemia, symptomatic hypocalcemia, and oliguric AKI. Which therapy may be required?

A. Urgent dialysis for refractory electrolyte abnormalities and severe oliguric acute kidney injury

B. High-dose calcium supplementation alone regardless of phosphate because dialysis cannot remove phosphate

C. Fluid restriction as the sole treatment because extracellular volume expansion drives tumor lysis

D. Stop urate-lowering therapy because uric acid is protective against calcium-phosphate deposition

E. Administer potassium supplements to replace intracellular potassium lost during tumor breakdown

65. A 34-year-old pregnant woman at 30 weeks has persistent hypertension requiring treatment. Which medication is generally appropriate in pregnancy?

A. Labetalol or extended-release nifedipine as commonly used pregnancy-compatible therapy

B. An ACE inhibitor because fetal renal development is protected by renin-angiotensin system blockade

C. An angiotensin receptor blocker because placental perfusion improves when fetal RAAS signaling is inhibited

D. Direct renin inhibition because aliskiren has the best-established fetal safety data

E. Spironolactone as first-line therapy because antiandrogenic effects are beneficial for fetal development

66. A 66-year-old man with CKD has potassium 5.9 mEq/L while taking an ACE inhibitor that provides substantial albuminuria reduction. He has no ECG changes and no acute kidney injury. Which strategy can help preserve renin-angiotensin blockade?

A. Permanently stop most renin-angiotensin blockade after a single mild potassium elevation regardless of benefit

B. Address reversible causes and use dietary measures, diuretics, or a chronic potassium binder when appropriate

C. Add an ARB to the ACE inhibitor because dual blockade lowers serum potassium by increasing filtration

D. Begin high-dose potassium supplements to reduce distal secretion through feedback inhibition

E. Use an NSAID to decrease renin secretion and thereby improve renal potassium excretion

67. A patient with long-term lithium therapy develops slowly progressive CKD, nephrogenic diabetes insipidus, and multiple small renal cysts. Which diagnosis is most likely?

A. Autosomal dominant polycystic kidney disease caused by PKD1 mutation in many lithium-treated patient

B. Medullary sponge kidney causing lithium accumulation and expected nephrogenic diabetes insipidus

C. Acute interstitial nephritis because chronic lithium injury often presents with fever and rash

D. Renal amyloidosis because small cortical cysts are strongly suggestive of systemic amyloid deposition

E. Chronic lithium-associated tubulointerstitial nephropathy with collecting-duct injury

68. A patient with pulmonary edema receives intravenous furosemide. Which transporter is inhibited?

A. The NCC sodium-chloride cotransporter in the distal convoluted tubule

B. The ENaC epithelial sodium channel in principal cells of the collecting duct

C. The NKCC2 sodium-potassium-2 chloride cotransporter in the thick ascending limb

D. The sodium-glucose cotransporter SGLT2 in the proximal convoluted tubule

E. The sodium-hydrogen exchanger targeted directly by most loop diuretics in the proximal tubule

69. A kidney transplant recipient develops fever, weight loss, lymphadenopathy, and an EBV-positive B-cell lymphoma 8 months after transplant. Which diagnosis is most likely?

A. Post-transplant lymphoproliferative disorder related to immunosuppression and EBV-driven B-cell proliferation

B. Acute T-cell-mediated rejection because lymphadenopathy is a typical manifestation of graft-supportive T-cell attack

C. BK virus nephropathy because polyomavirus commonly causes systemic B-cell lymphoma

D. Calcineurin-inhibitor nephrotoxicity because tacrolimus directly creates an EBV-negative sarcoma

E. Recurrent renal-cell carcinoma because transplant immunosuppression has a much stronger association with solid tumors than lymphoid disease

70. A malnourished patient with alcoholism has chronic sodium 108 mEq/L. After volume resuscitation, sodium rises by 10 mEq/L in 6 hours and urine output becomes brisk. Which intervention best reduces the risk of further overcorrection?

A. Additional isotonic saline to sustain the water diuresis until serum sodium is fully normalized

B. Tolvaptan to accelerate free-water excretion and prevent recurrence of symptomatic hyponatremia

C. Loop diuretic monotherapy to increase urinary sodium loss while maintaining the same water clearance

D. Desmopressin with electrolyte-free water as needed to stop and reverse an excessive aquaresis

E. No intervention because correction speed is less informative once neurologic symptoms have improved

71. A patient with stage 5 CKD has progressive nausea, anorexia, pruritus, pericardial rub, and cognitive slowing despite medical therapy. Which intervention is indicated?

A. Delay dialysis until serum creatinine reaches a predetermined numeric threshold regardless of symptoms

B. Increase dietary protein substantially because uremic manifestations reflect inadequate nitrogen intake

C. Initiation of kidney replacement therapy for symptomatic uremia

D. Start an NSAID for the pericarditis because anti-inflammatory therapy removes uremic toxins

E. Treat primarily with phosphate binders because most uremic symptoms are caused by hyperphosphatemia

72. A patient with cirrhosis and ascites has rising creatinine. There is no shock, nephrotoxin exposure, or structural kidney disease, and kidney function fails to improve after appropriate volume assessment and albumin expansion. Which diagnosis is most likely?

A. Hepatorenal syndrome-AKI from severe circulatory dysfunction in advanced portal hypertension

B. Acute glomerulonephritis because cirrhosis-associated AKI often has dysmorphic red cells

C. Bilateral renal artery thrombosis because portal hypertension directly occludes renal arteries

D. Acute interstitial nephritis because bland sediment and low urine sodium are supportive for drug allergy

E. Postrenal obstruction because ascites mechanically blocks both ureters in nearly many cirrhotic patient

73. A 61-year-old man has rapidly progressive GN, pulmonary nodules, sinus disease, and positive PR3-ANCA. Kidney biopsy shows necrotizing crescentic GN with little or no immune deposition. Which diagnosis is most likely?

A. Microscopic polyangiitis because granulomatous upper-airway disease and PR3-ANCA specifically favor GPA

B. Lupus nephritis because ANCA-associated GN typically shows full-house immune-complex staining

C. Anti-GBM disease because pauci-immune biopsy argues against circulating autoantibodies

D. Granulomatosis with polyangiitis causing pauci-immune ANCA-associated glomerulonephritis

E. IgA nephropathy because PR3-ANCA is a marker of mesangial IgA deposition

74. A 29-year-old woman has recurrent vomiting. Arterial pH is 7.52 and bicarbonate is 38 mEq/L. Urine chloride is 8 mEq/L. Which treatment is most likely to correct the alkalosis?

A. Spironolactone alone because low urine chloride indicates autonomous mineralocorticoid excess

B. Acetazolamide as the preferred first treatment despite marked extracellular volume depletion

C. Hydrochlorothiazide to increase distal sodium delivery and promote bicarbonate excretion

D. Isotonic saline with potassium chloride to replace chloride and volume deficits

E. Sodium bicarbonate infusion to correct the elevated serum pH by expanding extracellular volume

75. A patient with advanced CKD is asymptomatic, euvolemic, and has stable electrolytes despite an eGFR of 8 mL/min/1.73 m2. What is the best general approach to dialysis timing?

A. Prepare for kidney replacement therapy but initiate based on symptoms or refractory complications rather than eGFR alone

B. Start emergency dialysis immediately because many eGFR below 10 is an strong indication

C. Avoid planning for access because dialysis should rarely be discussed before symptoms appear

D. Begin daily dialysis primarily to normalize serum creatinine even in the absence of complications

E. Use serum urea nitrogen alone as the standard trigger for initiation of renal replacement therapy

76. A 67-year-old man presents with blood pressure 230/130 mm Hg, pulmonary edema, and acute kidney injury. Which management principle is most appropriate?

A. Use titratable intravenous therapy with controlled initial blood-pressure reduction because acute target-organ injury is present

B. Normalize blood pressure to 120/80 mm Hg within minutes to prevent any additional vascular stress

C. Treat primarily with oral medication and discharge because the strong blood-pressure value defines no emergency

D. Withhold therapy until creatinine returns to baseline because lowering blood pressure often worsens renal perfusion

E. Use sublingual short-acting nifedipine to cause an abrupt uncontrolled pressure reduction

77. A patient with cirrhosis and hepatorenal syndrome-AKI is being treated in the ICU. Which pharmacologic approach targets the underlying circulatory disturbance?

A. A pure vasodilator to further reduce systemic vascular resistance and increase splanchnic pooling

B. High-dose NSAIDs to block renal prostaglandins and intensify renal vasoconstriction

C. An ACE inhibitor to lower efferent arteriolar tone and reduce glomerular filtration pressure

D. Tolvaptan alone because V2 blockade reverses the arterial vasodilation driving hepatorenal syndrome

E. Albumin plus a vasoconstrictor such as terlipressin or norepinephrine in appropriate settings

78. A transplant candidate is EBV seronegative. Which maintenance or co-stimulatory blockade drug is generally avoided because of increased PTLD risk in EBV-seronegative recipients?

A. Tacrolimus because calcineurin inhibition is generally contraindicated in anyone without prior EBV exposure

B. Belatacept because it carries a major PTLD risk in EBV-seronegative patients

C. Mycophenolate because inosine monophosphate inhibition causes PTLD primarily in seronegative recipients

D. Prednisone because glucocorticoids cannot be used in any EBV-seronegative transplant recipient

E. Azathioprine because purine analogs are specifically prohibited primarily by negative EBV serology

79. A 67-year-old woman has rapidly progressive GN, pulmonary capillaritis, positive MPO-ANCA, and no granulomatous upper-airway disease. Which diagnosis is most likely?

A. Microscopic polyangiitis causing pauci-immune small-vessel vasculitis

B. Granulomatosis with polyangiitis because MPO-ANCA strongly suggests granulomatous sinonasal disease

C. Polyarteritis nodosa because PAN commonly causes glomerulonephritis by involving glomerular capillaries

D. IgA vasculitis because MPO-ANCA is a characteristic marker of IgA immune-complex disease

E. Cryoglobulinemic vasculitis because serum complement is often normal in cryoglobulinemia

80. A patient with progressive CKD is likely to need hemodialysis within the next year and has suitable arm vessels. Which vascular access generally provides the best long-term outcomes when it matures successfully?

A. An autogenous arteriovenous fistula in an appropriately selected patient

B. A tunneled central venous catheter as the preferred permanent access for most patients

C. A temporary non-tunneled femoral catheter placed months before anticipated dialysis

D. A peripherally inserted central catheter because it preserves central veins for future access

E. Repeated short peripheral intravenous catheters connected together for chronic hemodialysis

81. A 48-year-old man with resistant hypertension has metabolic alkalosis, potassium 2.8 mEq/L, suppressed renin, and elevated aldosterone. What is the most likely mechanism?

A. Loss of gastric acid causing secondary hyperaldosteronism with persistently high renin activity

B. Loop-diuretic use causing increased renin and aldosterone with low distal sodium delivery

C. Distal renal tubular acidosis causing impaired hydrogen secretion and compensatory hypokalemia

D. Primary adrenal insufficiency causing sodium wasting, hyperkalemia, and metabolic acidosis

E. Autonomous mineralocorticoid activity increasing distal sodium reabsorption with potassium and hydrogen loss

82. A 70-year-old man with sodium 126 mEq/L has serum osmolality 286 mOsm/kg and glucose 650 mg/dL. Which description best characterizes the sodium abnormality?

A. True hypotonic hyponatremia caused by SIADH with inappropriate urinary concentration

B. Pseudohyponatremia caused by severe hyperlipidemia with normal plasma water sodium

C. Translocational hyponatremia from hyperglycemia causing water movement into the extracellular space

D. Hypertonic hypernatremia from osmotic diuresis despite the measured low sodium concentration

E. Hypovolemic hypotonic hyponatremia caused primarily by urinary sodium wasting from glycosuria

83. A young adult has recurrent stones beginning in childhood. Urinalysis shows hexagonal crystals. Which disorder is most likely?

A. Primary hyperoxaluria causing calcium oxalate deposition and envelope-shaped crystals

B. Struvite stone disease caused by urease-positive bacteria and alkaline infected urine

C. Uric acid nephrolithiasis caused by persistently acidic urine and hyperuricosuria

D. Cystinuria from defective proximal tubular reabsorption of cystine and dibasic amino acids

E. Distal renal tubular acidosis causing calcium phosphate stones and alkaline urine

84. A patient with severe decompensated heart failure has rising creatinine during aggressive diuresis but is still markedly congested. Which principle is most important?

A. Persistent congestion may be more harmful than a modest hemodynamic creatinine rise, so decongestion should be guided by the whole clinical picture

B. Any creatinine increase mandates immediate cessation of diuretics even when pulmonary edema persists

C. Volume overload protects renal function, so decongestion should be avoided until creatinine normalizes

D. Rising creatinine strongly suggests acute tubular necrosis and requires dialysis in many patient with heart failure

E. Loop diuretics directly scar nephrons, so any dose above a low threshold is intrinsically nephrotoxic

85. A hemodialysis patient has prolonged bleeding from needle sites after dialysis and recurrent epistaxis. Platelet count and coagulation studies are normal. Which defect best explains the bleeding tendency?

A. Vitamin K deficiency causing isolated platelet dysfunction with a normal coagulation profile

B. Disseminated intravascular coagulation causing normal platelet count and normal fibrin degradation products

C. Immune thrombocytopenia causing severe platelet depletion that is hidden by the dialysis procedure

D. Uremic platelet dysfunction causing impaired adhesion and aggregation despite a normal platelet count

E. Factor VIII inhibitor causing a normal activated partial thromboplastin time in uremic patients

86. A patient has acute aortic dissection with severe hypertension and tachycardia. Which treatment sequence is preferred?

A. Start a pure arterial vasodilator first because reflex tachycardia reduces aortic wall stress

B. Rapid intravenous beta blockade to reduce shear force, followed by vasodilator therapy if pressure remains high

C. Give a thiazide diuretic alone because gradual volume loss is adequate for an aortic emergency

D. Use hydralazine monotherapy because an increase in heart rate protects the dissected aorta

E. Delay antihypertensive treatment until definitive surgery because blood-pressure reduction obscures imaging

87. A patient with systemic lupus erythematosus develops hypertension, hematuria, proteinuria, rising creatinine, low complement, and high anti-dsDNA titers. Which renal lesion most often produces a proliferative nephritic picture requiring aggressive immunosuppression?

A. Class I lupus nephritis because minimal mesangial deposits cause severe crescentic kidney failure

B. Class III or IV lupus nephritis with focal or diffuse proliferative immune-complex injury

C. Class II lupus nephritis because mesangial proliferation usually causes rapidly progressive renal failure

D. Class VI lupus nephritis because advanced sclerosis is highly responsive to intensive induction therapy

E. Pure class V lupus nephritis because membranous disease often presents with an isolated nephritic syndrome

88. A kidney transplant recipient has persistent hematocrit 58% several months after transplantation with no hypoxia or malignancy. What therapy is often effective?

A. An ACE inhibitor or angiotensin receptor blocker for post-transplant erythrocytosis

B. An erythropoiesis-stimulating agent because post-transplant erythrocytosis reflects insufficient erythropoietin

C. High-dose iron supplementation because increased hematocrit is usually caused by iron deficiency

D. Splenectomy because splenic sequestration is the principal cause of high hematocrit after transplant

E. Tolvaptan because aquaresis is the definitive treatment for excessive red-cell production

89. A patient with uremic platelet dysfunction requires an urgent invasive procedure. Which therapy can provide a rapid but temporary improvement in platelet function?

A. Warfarin to increase vitamin K-dependent clotting factor activity before the procedure

B. Aspirin to enhance platelet aggregation through irreversible cyclooxygenase inhibition

C. Clopidogrel to increase platelet adhesion by blocking the P2Y12 receptor

D. Tissue plasminogen activator to restore fibrin formation in uremic bleeding

E. Desmopressin to increase release of von Willebrand factor and improve platelet adhesion

90. A ventilated patient with severe abdominal distension has oliguria, rising creatinine, high bladder pressure, and worsening airway pressures. Which diagnosis should be considered?

A. Central diabetes insipidus because high intra-abdominal pressure causes free-water loss and hypernatremia

B. Renal vein thrombosis because bladder pressure directly measures renal venous clot burden

C. Minimal-change disease because elevated airway pressure causes acute podocyte effacement

D. Abdominal compartment syndrome causing renal venous congestion and reduced renal perfusion

E. Acute interstitial nephritis because abdominal distension is a classic allergic manifestation

91. A patient taking hydrochlorothiazide develops hyponatremia and hypokalemia. Which transporter is blocked by thiazide diuretics?

A. The NKCC2 cotransporter in the thick ascending limb used by loop diuretics

B. The ENaC channel in the cortical collecting duct blocked by amiloride

C. The SGLT2 cotransporter in the proximal tubule blocked by gliflozin drugs

D. The NCC sodium-chloride cotransporter in the distal convoluted tubule

E. The Na-K ATPase on the basolateral membrane selectively blocked by thiazides

92. A 39-year-old man has hypertension, enlarged kidneys with innumerable bilateral cysts, hepatic cysts, and a family history of kidney failure. What is the most likely diagnosis?

A. Autosomal dominant polycystic kidney disease due to a pathogenic PKD1 or PKD2 variant

B. Autosomal recessive polycystic kidney disease presenting primarily in infancy with no adult cases

C. Acquired cystic kidney disease caused by long-term dialysis and usually small native kidneys

D. Medullary sponge kidney causing primarily collecting-duct ectasia without enlarged cortical cystic kidneys

E. Simple renal cysts because numerous bilateral cysts plus hepatic cysts do not suggest inherited disease

93. A long-term hemodialysis patient develops shoulder pain, carpal tunnel syndrome, and destructive arthropathy. Which retained protein is associated with this complication?

A. Albumin deposited as the dominant fibril protein in dialysis-related arthropathy

B. Beta-2 microglobulin deposited as dialysis-related amyloid

C. Transthyretin deposited primarily because hemodialysis accelerates hereditary amyloidosis

D. Immunoglobulin A deposited in joints as the usual cause of dialysis-related carpal tunnel syndrome

E. Fibrinogen deposited because dialysis membranes selectively retain this clotting protein

94. A 22-year-old man develops weakness after a high-carbohydrate meal. Potassium is 2.2 mEq/L, acid-base status is normal, and urinary potassium excretion is low. Which mechanism is most likely?

A. Renal potassium wasting from primary hyperaldosteronism causing persistent hypertension and alkalosis

B. Gastrointestinal potassium loss from secretory diarrhea causing a normal-gap metabolic acidosis

C. Rapid intracellular potassium shift causing hypokalemic periodic paralysis rather than true body depletion

D. Distal renal tubular acidosis causing renal potassium wasting with an inappropriately high urine pH

E. Loop-diuretic exposure causing sodium chloride loss with secondary hyperaldosteronism and alkalosis

95. A 35-year-old woman develops polyuria after transsphenoidal surgery. Serum sodium is 151 mEq/L and urine osmolality is 95 mOsm/kg. After desmopressin, urine osmolality rises to 520 mOsm/kg. What is the diagnosis?

A. Nephrogenic diabetes insipidus caused by renal resistance to circulating vasopressin

B. Central diabetes insipidus due to deficient vasopressin secretion after pituitary surgery

C. Primary polydipsia with chronic suppression of vasopressin and excessive water intake

D. Osmotic diuresis from glucosuria causing an inappropriately concentrated urine

E. Cerebral salt wasting causing renal sodium loss and secondary free-water retention

96. A patient with lupus has nephrotic-range proteinuria but preserved kidney function and biopsy showing diffuse subepithelial immune deposits without proliferative lesions. Which class is most likely?

A. Class II mesangial proliferative lupus nephritis

B. Class V membranous lupus nephritis

C. Class III focal proliferative lupus nephritis

D. Class IV diffuse proliferative lupus nephritis

E. Class VI advanced sclerosing lupus nephritis

97. A patient has blood pressure 182/112 mm Hg discovered in clinic but no symptoms, no acute target-organ injury, and repeat measurements remain similar. Which approach is most appropriate?

A. Adjust oral therapy and arrange close follow-up rather than rapidly lowering pressure with intravenous drugs

B. Admit for immediate IV therapy because any pressure above 180/110 is by definition a hypertensive emergency

C. Lower mean arterial pressure by more than 50% in the first hour to prevent future organ injury

D. Use sublingual nifedipine to achieve a rapid unpredictable reduction before the patient leaves clinic

E. Withhold most treatment because severe asymptomatic hypertension rarely increases long-term cardiovascular risk

98. An older woman with CKD develops AKI after a sodium phosphate bowel preparation. Kidney biopsy later shows tubular calcium-phosphate deposits. What is the diagnosis?

A. Acute phosphate nephropathy from phosphate loading and intratubular calcium-phosphate deposition

B. Oxalate nephropathy because sodium phosphate is metabolized into oxalate crystals

C. Urate nephropathy because phosphate preparations cause massive nucleic acid breakdown

D. Acute interstitial nephritis because phosphate crystals often trigger a systemic allergic syndrome

E. Renal cortical necrosis because bowel preparation causes diffuse cortical infarction rather than tubular deposits

99. A patient on maintenance hemodialysis develops headache, nausea, and confusion during the first dialysis sessions after presenting with very high urea levels. Which complication is most likely?

A. Air embolism caused by many rapid decrease in blood urea nitrogen during dialysis

B. Uremic encephalopathy that necessarily worsens whenever dialysis removes nitrogenous solutes

C. Dialysis disequilibrium syndrome from rapid reduction in plasma osmolality and cerebral edema

D. Calciphylaxis caused by acute shifts in calcium during the first hemodialysis treatment

E. Hemolysis from normal dialysate temperature causing isolated neurologic symptoms

100. A kidney transplant recipient develops abrupt nephrotic-range proteinuria within days of transplantation. The native disease was primary FSGS. What is the most likely diagnosis?

A. Recurrent diabetic nephropathy because nodular glomerulosclerosis develops within days of transplant

B. Chronic antibody-mediated rejection because transplant glomerulopathy is immediate after reperfusion

C. Calcineurin-inhibitor toxicity because tacrolimus directly causes massive proteinuria within hours in most recipients

D. Recurrent ADPKD because cyst formation in the transplanted kidney causes immediate nephrotic syndrome

E. Recurrent primary FSGS in the allograft, which can occur very early after transplantation

101. A patient receiving peritoneal dialysis has cloudy effluent and diffuse abdominal pain. Peritoneal fluid shows neutrophil-predominant leukocytosis. What is the most likely diagnosis?

A. Normal peritoneal membrane adaptation that requires no treatment when dialysate becomes cloudy

B. Encapsulating peritoneal sclerosis presenting acutely with neutrophilic dialysate in many case

C. Uremic gastritis causing referred abdominal pain without infection of the peritoneal cavity

D. Peritoneal dialysis-associated peritonitis requiring prompt intraperitoneal antimicrobial therapy

E. Dialysis disequilibrium syndrome caused by rapid osmotic shifts across the peritoneal membrane

102. A patient has potassium 6.8 mEq/L and peaked T waves. Which treatment should be given first to reduce the immediate risk of ventricular arrhythmia?

A. Intravenous insulin alone because membrane stabilization is unnecessary when potassium can be shifted

B. Intravenous calcium to stabilize the cardiac membrane before potassium-shifting therapies

C. Sodium polystyrene sulfonate alone because gastrointestinal potassium removal is the fastest intervention

D. Loop diuretic alone because urinary potassium excretion immediately reverses ECG toxicity

E. Oral sodium bicarbonate alone because correction of acidemia instantly stabilizes the myocardium

103. A patient with short-bowel syndrome develops recurrent calcium oxalate stones and progressive kidney dysfunction. Which mechanism is responsible?

A. Enteric hyperoxaluria from increased intestinal oxalate absorption when fatty acids bind luminal calcium

B. Reduced oxalate absorption because unabsorbed fatty acids bind oxalate and prevent renal excretion

C. Primary hyperparathyroidism because bowel resection directly stimulates autonomous parathyroid secretion

D. Cystinuria because malabsorption creates an inherited defect in dibasic amino acid transport

E. Struvite formation because short-bowel syndrome causes chronic infection with urease-positive bacteria

104. A patient has purpura, arthralgias, abdominal pain, and glomerular hematuria after an upper respiratory infection. Kidney biopsy shows mesangial IgA deposition. What is the diagnosis?

A. Cryoglobulinemic vasculitis because IgA deposition is not seen in IgA-mediated disease

B. Microscopic polyangiitis because ANCA-associated GN shows dominant mesangial IgA staining

C. IgA vasculitis with renal involvement from mesangial IgA immune-complex deposition

D. Polyarteritis nodosa because medium-vessel vasculitis commonly causes glomerular immune deposits

E. Anti-GBM disease because purpura and abdominal pain indicate basement-membrane antibody injury

105. A patient has normal office blood pressure but elevated home and ambulatory readings. What is this pattern called?

A. White-coat hypertension, in which out-of-office pressure is lower than the clinic measurement

B. Pseudohypertension, caused by incompressible arteries and normal ambulatory blood-pressure values

C. Orthostatic hypotension, defined by higher out-of-office pressure without positional symptoms

D. Resistant hypertension, which requires failure of multiple medications regardless of actual pressure

E. Masked hypertension, which may carry cardiovascular risk despite normal clinic measurements

106. A kidney transplant recipient with prior IgA nephropathy develops microscopic hematuria and proteinuria several years after transplantation. Biopsy shows mesangial IgA deposits. What is the diagnosis?

A. Recurrent IgA nephropathy in the renal allograft with mesangial IgA deposition

B. Hyperacute rejection because mesangial IgA deposits are caused by preformed donor-supportive antibodies

C. BK nephropathy because polyomavirus produces dominant mesangial IgA immune complexes

D. Calcineurin toxicity because arteriolar vasoconstriction deposits IgA in the mesangium

E. Transplant renal artery stenosis because ischemia creates recurrent glomerular IgA deposition

107. A peritoneal dialysis patient has recurrent peritonitis caused by the same organism shortly after completing apparently successful therapy. What complication should be considered?

A. Dialysis disequilibrium because recurrent infection indicates overly rapid urea removal

B. Adynamic bone disease because low-turnover bone directly causes recurrent peritoneal infection

C. Renal artery stenosis because reduced renal perfusion promotes bacterial persistence in dialysate

D. Relapsing infection from a persistent catheter-associated biofilm or inadequately eradicated focus

E. Nephrogenic systemic fibrosis because gadolinium exposure causes neutrophilic peritoneal inflammation

108. A 62-year-old man taking lithium for bipolar disorder has polyuria, sodium 149 mEq/L, and urine osmolality 130 mOsm/kg. Desmopressin produces minimal change. Which therapy most directly targets the cause?

A. Hydrochlorothiazide to block sodium-chloride cotransport as the primarily mechanism-supportive therapy

B. Desmopressin replacement to correct deficient pituitary vasopressin secretion after lithium exposure

C. Spironolactone to block mineralocorticoid receptors and restore collecting-duct aquaporin expression

D. Amiloride to reduce lithium entry through epithelial sodium channels in collecting-duct cells

E. Acetazolamide to increase bicarbonate excretion and directly restore vasopressin receptor signaling

109. A woman with recurrent urinary tract infections has a staghorn calculus and urine pH 8.2. Which organism is most likely to promote this stone type?

A. Escherichia coli that acidifies urine through lactate production and forms pure uric acid stones

B. A urease-producing organism such as Proteus that generates alkaline urine and struvite

C. Enterococcus faecalis that consistently produces cystine stones through amino acid transport defects

D. Candida albicans that directly precipitates calcium oxalate by secreting oxalic acid in most infections

E. Staphylococcus epidermidis that causes stones primarily through hypercalciuria without urinary infection

110. A patient with multiple myeloma develops abrupt AKI, very high serum free light chains, and urine protein with relatively little albumin. Which lesion is most likely?

A. AL amyloidosis because amyloid fibrils usually cause abrupt oliguric AKI with little albuminuria

B. Light-chain cast nephropathy from filtered monoclonal light chains obstructing and injuring tubules

C. Minimal-change disease because monoclonal light chains produce selective albuminuria without tubular casts

D. Acute cortical necrosis because myeloma proteins cause diffuse cortical infarction rather than tubular injury

E. Renal artery stenosis because high free light chains constrict the main renal artery

111. A patient with Liddle syndrome has hypertension, hypokalemia, low renin, and low aldosterone. Which medication most directly treats the underlying channel abnormality?

A. Spironolactone, because excessive aldosterone secretion is the primary defect in Liddle syndrome

B. Furosemide, because NKCC2 activation is the inherited molecular lesion in Liddle syndrome

C. Hydrochlorothiazide, because NCC gain of function causes the low-renin hypertension

D. Amiloride, which blocks the epithelial sodium channel in the collecting duct

E. Tolvaptan, because vasopressin V2-receptor activation is the cause of ENaC overactivity

112. A peritoneal dialysis patient has progressive loss of ultrafiltration and recurrent volume overload despite adherence. Which membrane change is a recognized long-term cause?

A. Increased peritoneal solute transport with rapid dissipation of the osmotic gradient during long dwells

B. Complete loss of peritoneal capillaries causing inability of glucose to enter the systemic circulation

C. Suppression of aquaporin channels by most standard dialysate solutions within the first week of therapy

D. Formation of an AV fistula that diverts osmoles away from the peritoneal cavity

E. Reduced lymphatic drainage that often increases rather than decreases net ultrafiltration

113. A patient with chronic hepatitis C has purpura, neuropathy, low C4, rheumatoid factor positivity, and MPGN on kidney biopsy. Which process is most likely?

A. Anti-GBM disease causing linear IgG staining and isolated pulmonary-renal syndrome

B. Minimal-change disease caused by hepatitis C with normal complement and no systemic vasculitis

C. Mixed cryoglobulinemia causing immune-complex small-vessel disease and membranoproliferative GN

D. Primary FSGS because hepatitis C causes segmental sclerosis without circulating immune complexes

E. Alport syndrome because low complement and cryoglobulins are inherited collagen abnormalities

114. A patient with thrombocytosis has serum potassium 6.1 mEq/L but normal plasma potassium, no ECG changes, and no symptoms. What is the most likely explanation?

A. True hyperkalemia from reduced renal potassium excretion despite a normal plasma measurement

B. Intracellular potassium shift caused by insulin deficiency with selective elevation primarily in serum

C. Pseudohyperkalemia from potassium release during clotting in the serum sample

D. Familial hyperkalemic periodic paralysis causing episodic serum potassium elevation after exercise

E. Laboratory hypokalemia masked by hemolysis because platelet counts do not affect measured potassium

115. A patient has repeatedly elevated clinic blood pressure but normal validated home and ambulatory measurements. Which diagnosis is most likely?

A. Masked hypertension because ambulatory readings are lower than clinic readings

B. Malignant hypertension because office measurements are often the most accurate values

C. Pheochromocytoma because normal home blood pressure argues against most other causes

D. White-coat hypertension caused by a clinic-associated blood-pressure rise

E. Pseudohypotension because cuff measurements underestimate arterial pressure in the clinic

116. A hemodialysis patient develops recurrent intradialytic hypotension without evidence of infection or bleeding. Which nonpharmacologic adjustment is most appropriate?

A. Increase ultrafiltration aggressively to remove more fluid before hypotension becomes severe

B. Reassess dry weight and ultrafiltration rate, avoiding excessive fluid removal over a short session

C. Raise dialysate temperature because warming reliably increases peripheral vascular resistance

D. Give a large meal during dialysis to increase splanchnic blood flow and maintain arterial pressure

E. Use the highest possible sodium exposure chronically regardless of interdialytic weight gain

117. A kidney transplant recipient has delayed graft function and requires dialysis during the first week. Biopsy shows acute tubular injury without rejection. What is the most likely cause?

A. Hyperacute rejection because biopsy would show primarily bland acute tubular injury without vascular thrombosis

B. Recurrent membranous nephropathy because subepithelial deposits cause immediate anuria in most grafts

C. BK virus nephropathy because viral inclusions usually appear during the first hours after transplant

D. Chronic calcineurin toxicity because striped fibrosis develops before the graft is reperfused

E. Ischemia-reperfusion acute tubular injury causing delayed graft function

118. A patient with lymphoma receives high-dose methotrexate and develops AKI with methotrexate crystalluria despite hydration and urine alkalinization. Which rescue therapy may be indicated for severe delayed clearance?

A. Allopurinol because xanthine oxidase inhibition directly degrades methotrexate in plasma

B. Glucarpidase to rapidly metabolize circulating methotrexate when severe toxicity and delayed elimination are present

C. Desmopressin because V2-receptor activation increases methotrexate solubility in acidic urine

D. Fomepizole because alcohol dehydrogenase converts methotrexate into nephrotoxic metabolites

E. Tolvaptan because aquaresis enzymatically cleaves methotrexate into inactive compounds

119. A patient with ADPKD asks about screening for intracranial aneurysm. Which circumstance most strongly supports screening with noninvasive cerebrovascular imaging?

A. many asymptomatic patient with a single renal cyst regardless of family history or diagnosis

B. A normal neurologic examination because this alone predicts a high prevalence of aneurysm rupture

C. A personal history of aneurysm or a strong family history of intracranial aneurysm or subarachnoid hemorrhage

D. Absence of hypertension because normal blood pressure is the principal aneurysm risk factor

E. Age under 30 years with no family history because aneurysms occur primarily in young patients

120. A hemodialysis patient has persistent volume overload and hypertension despite reaching the prescribed postdialysis weight. Which measure best helps assess whether the target weight is too high?

A. Integrate serial physical findings, blood pressure trends, weight trajectory, and objective volume assessment when available

B. Use a single predialysis blood pressure because it precisely measures total-body extracellular volume

C. Use serum sodium alone because sodium concentration directly equals total-body sodium content

D. Use one chest radiograph regardless of symptoms because radiographic congestion is often present

E. Use serum creatinine because higher creatinine directly indicates excess extracellular fluid volume

121. A patient has hematuria, proteinuria, low C3, and biopsy showing a C3-dominant glomerulopathy with little immunoglobulin. What mechanism is most likely?

A. Anti-GBM autoantibodies causing linear IgG deposition without complement pathway dysregulation

B. T-cell podocyte injury causing minimal-change disease and selective albuminuria

C. Primary aldosterone excess causing mesangial C3 trapping as a consequence of hypertension

D. Defective type IV collagen synthesis causing hereditary basement-membrane thinning

E. Dysregulation of the alternative complement pathway causing persistent C3 activation in the glomerulus

122. A 79-year-old nursing-home resident with limited access to water has sodium 162 mEq/L and hypotension. Which fluid strategy is most appropriate initially?

A. Begin pure free-water replacement immediately and defer treatment of circulatory shock until sodium normalizes

B. Use hypertonic saline first because extracellular sodium concentration is markedly elevated

C. Restrict most fluids to avoid cerebral edema during spontaneous correction of the hypernatremia

D. Restore intravascular volume with isotonic crystalloid before replacing the remaining free-water deficit

E. Administer a vasopressin antagonist to create an aquaresis and reduce total-body sodium concentration

123. A patient receiving intravenous acyclovir develops flank discomfort and AKI shortly after high-dose therapy. Urine contains needle-shaped crystals. Which mechanism is most likely?

A. Intraluminal acyclovir crystal precipitation causing obstructive tubular injury

B. Immune-complex glomerulonephritis caused by antibodies against acyclovir-protein conjugates

C. Selective renal artery vasospasm that produces no tubular or urinary findings

D. Osmotic nephrosis caused by intracellular glycogen accumulation from acyclovir metabolism

E. Thrombotic microangiopathy caused by direct inhibition of ADAMTS13 in most high-dose recipients

124. A kidney transplant recipient 2 weeks after surgery has rising creatinine and hydronephrosis of the allograft. What complication should be suspected?

A. Acute T-cell-mediated rejection because hydronephrosis is a supportive histologic sign of cellular rejection

B. Calcineurin toxicity because tacrolimus causes mechanical ureteral obstruction through vasoconstriction

C. BK virus nephropathy because polyomavirus routinely causes early hydronephrosis in the first week

D. Recurrent glomerulonephritis because glomerular immune deposits directly dilate the collecting system

E. Ureteral obstruction from edema, stricture, clot, or extrinsic compression of the transplant ureter

125. A patient with ESKD and severe pruritus has no primary rash other than excoriations. Phosphate control is reasonable. Which approach is appropriate?

A. Assume the symptom is psychogenic because uremia does not cause generalized pruritus

B. Optimize dialysis and skin care and consider targeted antipruritic therapy for persistent uremic pruritus

C. Stop dialysis because removal of uremic solutes directly worsens pruritus in most patients

D. Treat primarily with systemic antibiotics because occult skin infection is the usual cause

E. Increase calcium-based phosphate binder exposure until serum calcium becomes elevated

126. A patient with apparent resistant hypertension takes five prescribed drugs but pharmacy records show frequent gaps in refills. What is the most important next step before extensive secondary-cause testing?

A. Proceed directly to renal denervation because nonadherence does not affect the definition of resistant hypertension

B. Assess and address medication adherence using nonjudgmental discussion and objective data when possible

C. Add several more antihypertensives because refill history is unrelated to blood-pressure control

D. Stop measuring home blood pressure because out-of-office readings cannot identify pseudoresistance

E. Order adrenal surgery because medication nonadherence specifically indicates primary aldosteronism

127. A patient with diabetic ketoacidosis has pH 7.20, bicarbonate 10 mEq/L, and PaCO2 38 mm Hg. What additional acid-base disorder is present?

A. Concurrent respiratory acidosis because PaCO2 is higher than expected for the metabolic acidosis

B. Concurrent respiratory alkalosis because PaCO2 should rise during metabolic acidosis

C. Metabolic alkalosis because bicarbonate is below the normal range during ketoacidosis

D. No additional disorder because PaCO2 of 38 mm Hg is normal in severe metabolic acidosis

E. Chronic respiratory alkalosis because renal bicarbonate retention is increasing the serum bicarbonate

128. A 65-year-old patient has nephrotic syndrome, restrictive cardiomyopathy, peripheral neuropathy, and monoclonal lambda light chains. Kidney biopsy shows Congo-red-positive deposits with apple-green birefringence. What is the diagnosis?

A. Light-chain cast nephropathy because casts produce Congo-red-positive glomerular fibrils

B. Diabetic nephropathy because nodular sclerosis is often Congo-red positive

C. Fibrillary glomerulonephritis because its fibrils are characteristically Congo-red positive in routine cases

D. Minimal-change disease because podocyte effacement produces apple-green birefringence

E. AL amyloidosis from monoclonal immunoglobulin light-chain fibril deposition

129. A patient with CKD is scheduled for colonoscopy and is at risk for volume depletion. Which bowel preparation is generally avoided because of risk for phosphate nephropathy and severe electrolyte disturbance?

A. Polyethylene glycol-based lavage used with appropriate volume monitoring

B. Oral sodium phosphate preparations in susceptible patients with kidney disease

C. A split-dose isotonic polyethylene glycol regimen in a patient without obstruction

D. Low-residue dietary preparation followed by an established nonphosphate regimen

E. Standard bowel cleansing using a kidney-safe preparation selected for comorbidities

130. A patient receiving tenofovir disoproxil fumarate develops AKI, hypophosphatemia, normoglycemic glycosuria, and proteinuria. Which lesion is most likely?

A. Distal tubular toxicity causing isolated type 1 renal tubular acidosis without phosphate loss

B. Acute glomerular immune-complex disease causing selective albuminuria and low complement

C. Renal papillary necrosis because tenofovir primarily injures the medullary papillae

D. Postrenal obstruction because tenofovir precipitates as large stones in both ureters

E. Proximal tubular toxicity causing a Fanconi-type syndrome

131. A patient taking acetazolamide develops a normal-anion-gap metabolic acidosis. Which mechanism explains the effect?

A. Carbonic anhydrase inhibition reduces proximal bicarbonate reabsorption and causes bicarbonaturia

B. ENaC blockade increases distal hydrogen secretion and causes bicarbonate retention

C. NKCC2 blockade directly increases proximal bicarbonate reabsorption and metabolic alkalosis

D. V2-receptor antagonism causes selective chloride loss and severe contraction alkalosis

E. Mineralocorticoid receptor activation suppresses proximal bicarbonate reclamation

132. A patient presents after antifreeze ingestion with high-anion-gap metabolic acidosis, AKI, and calcium oxalate crystals in the urine. Which toxic alcohol is responsible?

A. Ethylene glycol, metabolized to organic acids including oxalate that can injure renal tubules

B. Methanol, metabolized primarily to formate and classically causing optic nerve toxicity

C. Isopropanol, metabolized to acetone and typically producing ketosis without high-gap acidosis

D. Propylene glycol, which rarely causes lactic acidosis or osmolar-gap abnormalities

E. Ethanol, which directly forms calcium oxalate crystals through hepatic metabolism

133. A 52-year-old woman with chronic hypernatremia from impaired thirst has sodium 158 mEq/L but no neurologic symptoms. Why should correction generally be gradual?

A. Chronic hypernatremia causes persistent cerebral sodium loss, so rapid lowering triggers osmotic demyelination

B. Rapid sodium lowering causes abrupt potassium influx into neurons and produces malignant hyperkalemia

C. Hypernatremia suppresses cerebral glucose uptake, so rapid correction precipitates severe hypoglycemia

D. Slow correction is required because the kidney cannot excrete sodium when vasopressin is present

E. Brain cells accumulate osmolytes during chronic hypernatremia, so rapid lowering can cause cerebral edema

134. A patient with stage 4 CKD asks whether a gadolinium-enhanced MRI can ever be performed. Which statement is most accurate?

A. Risk depends on the gadolinium agent and kidney function, and lower-risk macrocyclic agents may be used when clinically justified

B. most gadolinium agents are generally forbidden at any eGFR below 60 because nephrogenic systemic fibrosis is likely

C. Gadolinium is low risk in kidney failure because it is not renally excreted

D. primarily linear high-risk gadolinium agents should be used because they clear faster during CKD

E. Hemodialysis must be initiated primarily to permit any single gadolinium exposure in a nondialysis patient

135. A kidney transplant recipient develops a painless fluid collection near the graft several weeks after surgery that compresses the ureter and causes hydronephrosis. Fluid creatinine is similar to serum. What is the most likely diagnosis?

A. Urinoma because urinary leaks typically have fluid creatinine similar to serum rather than much higher

B. Perinephric abscess because sterile lymph fluid often contains abundant neutrophils and bacteria

C. Renal vein thrombosis because venous occlusion forms a clear lymphatic fluid collection

D. Acute rejection because rejection creates an extrarenal fluid cavity around the graft

E. Lymphocele causing extrinsic compression of the transplant ureter

136. A patient with true resistant hypertension is taking an ACE inhibitor, a long-acting calcium-channel blocker, and an appropriate diuretic. Kidney function and potassium permit additional therapy. Which drug is often an effective fourth agent?

A. A short-acting nitrate used primarily when the patient feels that pressure is elevated

B. A second ACE inhibitor because duplicate renin-angiotensin blockade increases efficacy without toxicity

C. An NSAID because prostaglandin inhibition improves sodium excretion in resistant hypertension

D. A mineralocorticoid receptor antagonist such as spironolactone

E. A sympathomimetic decongestant because alpha stimulation lowers systemic vascular resistance

137. A patient with salicylate toxicity has tinnitus, tachypnea, pH 7.46, PaCO2 20 mm Hg, and bicarbonate 14 mEq/L. Which acid-base pattern is present?

A. Simple respiratory alkalosis with fully appropriate renal compensation and no metabolic process

B. Simple high-anion-gap metabolic acidosis with expected respiratory compensation primarily

C. Mixed metabolic alkalosis and respiratory acidosis caused by reduced central respiratory drive

D. Normal-anion-gap metabolic acidosis with superimposed chronic respiratory acidosis

E. Mixed respiratory alkalosis and high-anion-gap metabolic acidosis

138. A patient develops AKI after several days of aminoglycoside therapy. Which pattern is most typical?

A. Acute glomerulonephritis with low complement and red-cell casts as the usual toxicity

B. Bilateral ureteral obstruction from large aminoglycoside crystals as the dominant mechanism

C. Nonoliguric acute tubular injury from proximal tubular accumulation of aminoglycoside

D. Hyperacute interstitial nephritis within minutes of the first dose in nearly many patient

E. Renal vein thrombosis due to direct aminoglycoside activation of coagulation

139. A patient has nephrotic-range proteinuria and kidney biopsy shows randomly arranged fibrils larger than amyloid fibrils, negative Congo red staining, and glomerular staining for DNAJB9. Which diagnosis is most likely?

A. AL amyloidosis because Congo red is usually strongly positive in fibrillary glomerulonephritis

B. Immunotactoid glomerulopathy because DNAJB9 is a supportive marker of organized microtubules in parallel arrays

C. Minimal-change disease because podocyte effacement produces fibrils visible on electron microscopy

D. Fibrillary glomerulonephritis with DNAJB9-positive, Congo-red-negative fibrils

E. Anti-GBM disease because linear antibody deposition creates extracellular fibrils in the mesangium

140. A patient with stable CKD and diabetes needs iodinated contrast for an urgent coronary intervention. Which strategy is most appropriate for kidney protection?

A. Cancel most contrast studies because contrast exposure is generally contraindicated in any chronic kidney disease

B. Use the lowest necessary contrast dose, avoid volume depletion and nephrotoxins, and provide appropriate isotonic volume support when indicated

C. Give high-dose NSAIDs before contrast to maintain renal perfusion through afferent vasoconstriction

D. Induce forced diuresis with severe volume depletion because low urine flow prevents contrast entry into tubules

E. Begin prophylactic hemodialysis primarily to remove contrast immediately after many exposure

141. A patient with ADPKD has evidence of rapid disease progression and preserved enough kidney function to consider disease-modifying therapy. Which drug may slow cyst growth and GFR decline in selected patients?

A. Desmopressin, a V2-receptor agonist that suppresses cyst growth by increasing intracellular cAMP

B. Tolvaptan, a vasopressin V2-receptor antagonist, with monitoring for liver toxicity and aquaresis

C. High-dose NSAID therapy because prostaglandin inhibition directly prevents cyst epithelial proliferation

D. Fludrocortisone because mineralocorticoid activation reduces total kidney volume in ADPKD

E. Acetazolamide because urinary alkalinization has been proven to halt PKD1-mediated cyst formation

142. A kidney transplant recipient develops sudden graft swelling, pain, hematuria, oliguria, and loss of venous flow on Doppler ultrasound during the first week. Which diagnosis is most likely?

A. Chronic antibody-mediated rejection because transplant glomerulopathy causes sudden venous occlusion in the first week

B. BK nephropathy because viral tubulitis produces absent venous flow on Doppler examination

C. Renal allograft vein thrombosis causing acute venous outflow obstruction

D. Calcineurin toxicity because vasoconstriction selectively eliminates renal venous flow while preserving arterial perfusion

E. Recurrent IgA nephropathy because mesangial deposits cause immediate graft swelling and venous thrombosis

143. A patient with CKD has progressive loss of appetite, weight loss, and muscle wasting. Which nutritional principle is most appropriate before dialysis?

A. Use severe calorie restriction because obesity is the primarily nutritional risk in advanced CKD

B. Eliminate nearly most dietary protein regardless of nutritional status to suppress blood urea nitrogen

C. Encourage unrestricted sodium intake because extracellular volume does not affect nutritional status

D. Provide adequate calories and avoid excessive protein restriction that worsens protein-energy wasting

E. Avoid dietitian involvement because CKD dietary recommendations are too variable to be clinically useful

144. A patient with severe metformin-associated lactic acidosis has shock, pH 6.9, very high lactate, and kidney failure. Which extracorporeal therapy is most appropriate when toxicity is severe?

A. Peritoneal dialysis primarily because metformin is too protein bound for hemodialysis removal

B. Plasma exchange because metformin is confined to circulating immunoglobulins

C. No extracorporeal therapy because metformin has insufficient extracorporeal clearance to justify dialysis

D. Ultrafiltration without dialysate because toxin removal requires primarily convective water extraction

E. Hemodialysis to remove metformin and correct severe acidosis and electrolyte abnormalities

145. A 58-year-old man with a monoclonal gammopathy has proteinuria and kidney biopsy showing nodular glomerulosclerosis with linear deposition of a single light chain along glomerular and tubular basement membranes. What is the diagnosis?

A. AL amyloidosis because monoclonal light chains often form Congo-red-positive fibrils rather than linear deposits

B. Diabetic nephropathy because monoclonal light-chain restriction is a nonspecific feature of diabetes

C. Minimal-change disease because linear basement-membrane staining indicates isolated podocyte injury

D. Monoclonal immunoglobulin deposition disease, such as light-chain deposition disease

E. Postinfectious GN because monoclonal deposits are expected after bacterial infection

146. A patient with advanced CKD and resistant hypertension remains volume overloaded despite a thiazide at a low dose. Which change may improve volume and blood-pressure control?

A. Stop most diuretics because reduced GFR makes sodium retention impossible

B. Replace the diuretic with chronic NSAID therapy to increase renal sodium excretion

C. Increase dietary sodium to enhance delivery of antihypertensive drugs to the nephron

D. Use an effective loop diuretic regimen, with combination nephron blockade in selected resistant cases

E. Use desmopressin to increase free-water retention and lower systemic vascular resistance

147. A hospitalized patient has sodium 119 mEq/L, serum osmolality 258 mOsm/kg, urine osmolality 110 mOsm/kg, and urine sodium 45 mEq/L. Sodium remains stably between 118 and 122 mEq/L despite changes in fluid intake, and a water-loading test shows preserved ability to dilute urine below the lower defended osmolality. Which diagnosis is most likely?

A. Reset osmostat with a lower defended threshold for vasopressin release and thirst

B. Classic SIADH with fixed inability to dilute urine at any degree of hypo-osmolality

C. Primary adrenal insufficiency causing persistent sodium loss and mineralocorticoid deficiency

D. Psychogenic polydipsia causing continuously maximally dilute urine and progressive sodium decline

E. Renal failure causing obligate isosthenuria and inability to vary urine concentration

148. A patient with advanced CKD develops progressive hyperphosphatemia despite dietary counseling. Which therapy is appropriate?

A. Take phosphate binders primarily on an empty stomach because they work by increasing renal phosphate excretion

B. Use intravenous phosphate supplementation because CKD causes total-body phosphate depletion

C. Use a phosphate binder with meals when persistent phosphate elevation requires treatment

D. Stop most dietary protein because phosphate binders cannot affect intestinal phosphate absorption

E. Use a loop diuretic as the sole therapy because dialysis patients excrete most phosphate in urine

149. A patient with severe hypoalbuminemia has a measured anion gap of 11 mEq/L despite suspected lactic acidosis. Which principle is most important when interpreting the result?

A. The anion gap should be corrected downward for low albumin because albumin is a major unmeasured cation

B. The anion gap should be corrected upward for low albumin because albumin is a major unmeasured anion

C. Albumin concentration has no effect on the anion gap and should rarely alter interpretation

D. Lactate lowers the anion gap, so a normal value confirms the absence of tissue hypoperfusion

E. The anion gap is invalid whenever serum albumin is below the laboratory reference range

150. A patient with chronic lithium use presents with confusion, ataxia, tremor, kidney dysfunction, and a very high lithium concentration. Which feature favors hemodialysis?

A. Mild asymptomatic elevation with normal kidney function because most lithium levels require dialysis

B. A low lithium concentration after a single missed dose because rebound toxicity is likely

C. Isolated nephrogenic diabetes insipidus without neurologic symptoms because dialysis cures the chronic concentrating defect

D. Normal serum lithium with chronic tremor because extracorporeal removal is required despite no circulating drug

E. Severe neurologic toxicity with impaired renal clearance and a high or rising lithium burden

151. A patient with SIADH receives tolvaptan. What is the principal renal action?

A. Activation of vasopressin V2 receptors, increasing aquaporin insertion and water retention

B. Blockade of ENaC, producing natriuresis that is similar to the action of amiloride

C. Blockade of vasopressin V2 receptors in collecting ducts, producing electrolyte-free water excretion

D. Inhibition of NKCC2, collapsing the medullary gradient through loop-diuretic activity

E. Activation of mineralocorticoid receptors, increasing sodium retention and potassium secretion

152. A dialysis patient has persistent hyperphosphatemia and recurrent hypercalcemia while taking large doses of calcium carbonate. Which change is most reasonable?

A. Increase the calcium carbonate dose because hypercalcemia strongly suggests inadequate intestinal calcium exposure

B. Add high-dose vitamin D primarily to raise calcium further and suppress most parathyroid hormone secretion

C. Stop phosphate control largely because hyperphosphatemia is low risk once dialysis has started

D. Use oral phosphate supplements to prevent vascular calcification from low intracellular phosphate stores

E. Switch to or add a noncalcium phosphate binder to reduce further calcium loading

153. A kidney transplant recipient develops refractory hypertension and rising creatinine several months after surgery. A bruit is heard over the graft. Which diagnosis is most likely?

A. Transplant renal artery stenosis causing renovascular hypertension and allograft dysfunction

B. Ureteral leak because urinary extravasation commonly causes an arterial bruit and severe hypertension

C. BK nephropathy because viral replication produces a focal arterial bruit over the graft

D. Post-transplant erythrocytosis because increased hematocrit directly narrows the transplant renal artery

E. Acute pyelonephritis because infection creates fixed renal artery stenosis on Doppler imaging

154. A young man has persistent microscopic hematuria, progressive proteinuria, sensorineural hearing loss, and ocular abnormalities. Which inherited disease is most likely?

A. Alport syndrome from pathogenic variants affecting type IV collagen in basement membranes

B. Thin-basement-membrane nephropathy because hearing and ocular abnormalities are typical benign features

C. IgA nephropathy because type IV collagen mutations cause mesangial IgA deposition

D. Fabry disease because alpha-galactosidase deficiency primarily causes sensorineural deafness without other systemic signs

E. Autosomal dominant polycystic kidney disease because glomerular basement-membrane splitting is caused by renal cysts

155. A 28-year-old man has early-onset severe hypertension, hypokalemia, metabolic alkalosis, low renin, and low aldosterone. Which inherited disorder is most likely?

A. Primary aldosteronism from adrenal aldosterone overproduction with elevated aldosterone levels

B. Bartter syndrome from NKCC2 dysfunction causing salt wasting, high renin, and high aldosterone

C. Gitelman syndrome from NCC dysfunction causing salt wasting, low blood pressure, and high renin

D. Liddle syndrome from constitutive activation of the epithelial sodium channel

E. Addison disease from mineralocorticoid deficiency causing hyperkalemia and volume depletion

156. A patient with persistent hypomagnesemia also has hypokalemia that does not correct despite potassium supplementation. Which mechanism best explains the refractory hypokalemia?

A. Low magnesium directly blocks aldosterone receptors and prevents any urinary potassium excretion

B. Low intracellular magnesium increases distal renal potassium secretion through ROMK channels

C. Magnesium deficiency shifts potassium from cells into plasma, masking rather than causing hypokalemia

D. Hypomagnesemia closes epithelial sodium channels and eliminates the electrical gradient for potassium secretion

E. Low magnesium causes pseudohypokalemia through an artifact in routine serum chemistry measurement

157. A patient on dialysis has severe secondary hyperparathyroidism despite correction of phosphate and use of vitamin D therapy. Which medication can suppress PTH by activating the calcium-sensing receptor?

A. A loop diuretic that directly activates the parathyroid calcium-sensing receptor

B. A sodium-glucose cotransporter inhibitor that blocks PTH secretion from the parathyroid gland

C. An ACE inhibitor that suppresses PTH through efferent arteriolar vasodilation

D. A calcimimetic such as cinacalcet or etelcalcetide

E. A xanthine oxidase inhibitor that lowers PTH by reducing uric acid production

158. A patient with salicylate poisoning has altered mental status, pulmonary edema, severe acidemia, and rising salicylate levels despite treatment. What is the definitive next step?

A. Intubation with permissive hypoventilation before any alkalinization because higher $PaCO_2$ reduces CNS salicylate entry

B. Stop bicarbonate because urine alkalinization increases salicylate reabsorption from the kidney

C. Urgent hemodialysis because severe salicylate toxicity is rapidly treatable with extracorporeal removal

D. Give calcium as the definitive antidote because calcium binds salicylate and neutralizes its acid-base effects

E. Use peritoneal dialysis primarily because salicylate is too highly protein bound for hemodialysis in poisoning

159. A marathon runner drinks large amounts of hypotonic fluid during a race and develops headache, vomiting, and sodium 122 mEq/L. Which mechanism most likely contributes to this condition?

A. Isolated renal sodium wasting from exercise-induced aldosterone suppression with fully suppressed vasopressin

B. Excess hypotonic intake combined with nonosmotic vasopressin release during prolonged exercise

C. Loss of pure water through sweat causing hypertonicity despite the measured reduction in serum sodium

D. Primary mineralocorticoid deficiency caused by transient adrenal ischemia during endurance exercise

E. Persistent osmotic diuresis from glycogen breakdown causing hypernatremia and intracellular dehydration

160. A 33-year-old woman with a history of surreptitious diuretic use has hypokalemia and metabolic alkalosis. Urine chloride is 62 mEq/L while she is actively taking the drug. Which finding would best support loop-diuretic exposure rather than vomiting?

A. Persistently high urine chloride during active diuretic effect despite volume contraction

B. Very low urine chloride because loop diuretics substantially prevent urinary chloride excretion

C. Marked hyperkalemia because loop diuretics reduce distal sodium delivery to the collecting duct

D. Normal renin and aldosterone because loop diuretics do not activate the renin-angiotensin system

E. Metabolic acidosis because loop diuretics directly inhibit proximal bicarbonate reabsorption

161. A patient with long-standing dialysis-dependent kidney failure develops persistent hypercalcemia with markedly elevated PTH despite medical therapy. Which diagnosis best describes the parathyroid disorder?

A. Primary hypoparathyroidism caused by dialysis-related destruction of most parathyroid tissue

B. Adynamic bone disease because autonomous PTH secretion often suppresses bone turnover

C. Tertiary hyperparathyroidism from autonomous secretion after prolonged secondary hyperparathyroidism

D. Pseudohypoparathyroidism caused by renal resistance to PTH with low circulating hormone levels

E. Isolated vitamin D deficiency because this typically causes both hypercalcemia and autonomous PTH excess

162. A woman has lifelong isolated microscopic hematuria, normal kidney function, minimal proteinuria, and a family history of similar benign hematuria. Biopsy shows diffusely thin glomerular basement membranes. Which diagnosis is most likely?

A. Anti-GBM disease because a thin basement membrane causes circulating basement-membrane antibodies

B. Membranous nephropathy because subepithelial deposits make the basement membrane uniformly thin

C. Thin basement membrane nephropathy related to heterozygous type IV collagen variants

D. Postinfectious GN because complement activation creates a lifelong inherited thin-membrane pattern

E. Amyloidosis because fibril deposition causes a diffusely thin rather than thickened basement membrane

163. A woman with a kidney transplant plans pregnancy. Which immunosuppressive medication should be discontinued and replaced before conception because of major teratogenic risk?

A. Azathioprine because it is generally contraindicated in many transplant pregnancy

B. Tacrolimus because calcineurin inhibitors cause standard fetal renal agenesis

C. Prednisone because any glucocorticoid exposure causes severe congenital anomalies

D. Mycophenolate, which is associated with pregnancy loss and characteristic congenital malformations

E. Hydroxychloroquine because it is the principal transplant antimetabolite and is highly teratogenic

164. A patient ingests antifreeze and has high-anion-gap metabolic acidosis, osmolar gap, hypocalcemia, and AKI. Which antidote should be given promptly?

A. Naloxone to block opioid receptors and prevent oxalate crystal formation

B. Flumazenil to inhibit benzodiazepine receptors and reduce glycolate generation

C. Fomepizole to inhibit alcohol dehydrogenase and prevent toxic ethylene glycol metabolite formation

D. N-acetylcysteine to replenish glutathione and block alcohol dehydrogenase

E. Atropine to inhibit muscarinic receptors and prevent metabolism to oxalic acid

165. A patient with hypertension, hypokalemia, low renin, and low aldosterone consumes large amounts of black licorice. Which treatment is most important?

A. Stop licorice exposure and correct potassium while allowing mineralocorticoid-receptor physiology to recover

B. Increase licorice intake to restore 11-beta-hydroxysteroid dehydrogenase activity

C. Begin fludrocortisone because the syndrome is caused by true mineralocorticoid deficiency

D. Use high-dose potassium restriction because total-body potassium is elevated in this disorder

E. Add an ACE inhibitor primarily to increase aldosterone synthesis and worsen potassium loss

166. A dialysis patient develops sudden weakness after missing several treatments. Potassium is 7.1 mEq/L with QRS widening despite temporizing therapy. What is the definitive treatment?

A. Urgent hemodialysis to remove potassium after temporizing therapies have failed

B. Repeat intravenous calcium indefinitely because calcium directly removes potassium from plasma

C. Give insulin alone because intracellular shifting provides permanent total-body potassium removal

D. Administer oral potassium supplements to prevent rebound after membrane stabilization

E. Use sodium bicarbonate as the sole definitive therapy regardless of the degree of kidney failure

167. A patient taking trimethoprim-sulfamethoxazole develops a mild creatinine rise without other evidence of kidney injury and new hyperkalemia. Which pharmacologic effects explain these findings?

A. Sulfamethoxazole directly raises GFR and stimulates ENaC, causing potassium retention through increased filtration

B. Trimethoprim activates aldosterone receptors and increases distal potassium secretion while lowering creatinine

C. The drug blocks vasopressin receptors, causing nephrogenic diabetes insipidus and pseudohyperkalemia

D. The medication inhibits proximal glucose uptake and causes an isolated glycosuric osmotic diuresis

E. Trimethoprim inhibits tubular creatinine secretion and has an amiloride-like ENaC-blocking effect

168. A patient with chronic hypercapnic respiratory failure has PaCO2 60 mm Hg and bicarbonate 32 mEq/L. Which renal adaptation is expected?

A. Rapid renal bicarbonate loss causing bicarbonate to fall below 20 mEq/L within minutes

B. Suppressed ammonium production because chronic hypercapnia makes urinary acid excretion unnecessary

C. Complete normalization of arterial pH because renal compensation fully corrects respiratory acidosis

D. Increased chloride retention without any change in renal bicarbonate handling

E. Increased renal bicarbonate retention and ammonium excretion over several days

169. A patient has thrombocytopenia, microangiopathic hemolytic anemia, neurologic symptoms, fever, and kidney injury. ADAMTS13 activity is severely deficient. What is the diagnosis?

A. Complement-mediated atypical hemolytic uremic syndrome because severe ADAMTS13 deficiency is its defining lesion

B. Malignant hypertension because ADAMTS13 activity is often profoundly deficient in hypertensive emergencies

C. Disseminated intravascular coagulation because severe ADAMTS13 deficiency is caused by consumptive coagulopathy

D. Thrombotic thrombocytopenic purpura due to severe ADAMTS13 deficiency

E. Immune thrombocytopenia because isolated platelet destruction produces schistocytes and renal microthrombi

170. A patient with ESKD has refractory pulmonary edema despite maximal diuretic therapy and severe hypoxemia. Which intervention is indicated?

A. Continue escalating oral fluid intake because dialysis is contraindicated during pulmonary edema

B. Delay kidney replacement therapy until peripheral edema becomes absent

C. Urgent ultrafiltration through dialysis for refractory volume overload

D. Give intravenous sodium bicarbonate as the primarily therapy because pulmonary edema is caused by metabolic acidosis

E. Use an NSAID to reduce renal prostaglandins and improve diuretic delivery to the nephron

171. A patient has visual blurring, high-anion-gap acidosis, and an osmolar gap after ingesting windshield-washer fluid. Which toxic alcohol is most likely?

A. Ethylene glycol, whose hallmark toxicity is isolated optic neuritis without kidney injury

B. Isopropanol, which produces severe high-anion-gap acidosis through formic acid formation

C. Methanol, whose formate metabolite causes optic nerve and retinal toxicity

D. Ethanol, which routinely causes retinal infarction through oxalate crystal deposition

E. Propylene glycol, which uniquely causes optic nerve toxicity with no lactate elevation

172. A kidney transplant recipient asks about vaccination. Which vaccine type is generally avoided after transplantation while significant immunosuppression continues?

A. Inactivated influenza vaccine because killed vaccines routinely cause disseminated infection

B. Recombinant zoster vaccine because nonlive recombinant vaccines are prohibited after transplantation

C. Pneumococcal vaccines because polysaccharide and conjugate vaccines replicate in immunosuppressed hosts

D. Live attenuated vaccines because uncontrolled replication can occur in immunosuppressed recipients

E. Hepatitis B vaccine because most nonlive vaccines are generally contraindicated after transplant

173. A patient with subarachnoid hemorrhage develops sodium 124 mEq/L, high urine sodium, concentrated urine, orthostatic hypotension, and negative fluid balance. Which diagnosis best fits?

A. SIADH causing euvolemic water retention without a sustained negative sodium balance

B. Primary polydipsia causing hypotonic urine and expansion of extracellular fluid volume

C. Cerebral salt wasting with renal sodium loss causing true extracellular volume depletion

D. Pseudohyponatremia from hyperproteinemia without a reduction in measured plasma osmolality

E. Nephrogenic diabetes insipidus causing free-water loss and progressive hypernatremia

174. A young patient has nephrocalcinosis, recurrent stones, hematuria, and cystic dilation of collecting ducts in the renal medulla with preserved kidney function. What is the most likely diagnosis?

A. Autosomal dominant polycystic kidney disease with diffuse cortical and medullary cyst enlargement

B. Medullary sponge kidney with congenital ectasia of medullary collecting ducts

C. Nephronophthisis causing corticomedullary cysts, salt wasting, and progressive childhood kidney failure

D. Acquired cystic kidney disease caused by years of dialysis and increased renal-cell carcinoma risk

E. Simple renal cyst disease because collecting-duct ectasia rarely causes nephrocalcinosis or stones

175. A patient receiving intermittent hemodialysis has recurrent muscle cramps late in treatment. Which factor commonly contributes?

A. Rapid ultrafiltration causing intravascular volume contraction during dialysis

B. Excessive retention of urea because hemodialysis does not remove small solutes

C. High serum bicarbonate caused by failure of the dialyzer to exchange electrolytes

D. expected hypercalcemia from most bicarbonate-based dialysate solutions

E. Progressive polycythemia caused by endogenous erythropoietin excess during dialysis

176. A young adult has upper-extremity hypertension, delayed femoral pulses, and lower-extremity systolic pressure substantially below arm pressure. Which diagnosis is most likely?

A. Primary aldosteronism causing equal hypertension in most extremities without pulse delay

B. Pheochromocytoma causing fixed femoral pulse delay from catecholamine-mediated vasospasm

C. Coarctation of the aorta producing proximal hypertension and reduced lower-extremity perfusion

D. Renal artery stenosis causing a persistent arm-leg pressure gradient from renal ischemia

E. Obstructive sleep apnea causing differential blood pressure primarily in the upper extremities

177. A hemodynamically unstable ICU patient with severe AKI requires kidney replacement therapy and cannot tolerate rapid fluid shifts. Which modality is most appropriate?

A. Continuous kidney replacement therapy to allow slower solute and fluid removal

B. High-efficiency intermittent hemodialysis because the fastest fluid removal is safest during shock

C. Peritoneal dialysis primarily because continuous extracorporeal therapy cannot be used with vasopressors

D. No dialysis until vasopressors are stopped because kidney replacement therapy is contraindicated in shock

E. Ultrafiltration without any extracorporeal circuit because continuous therapy is less effective than other strategies for potassium removal

178. A patient develops severe AKI, thrombocytopenia, and microangiopathic hemolysis after a diarrheal illness caused by Shiga-toxin-producing E. coli. Which syndrome is most likely?

A. Shiga toxin-associated hemolytic uremic syndrome after enterohemorrhagic infection

B. TTP because diarrheal HUS is defined by severe ADAMTS13 deficiency

C. Anti-GBM disease because Shiga toxin triggers linear IgG deposition in glomeruli

D. IgA vasculitis because Shiga toxin causes mesangial IgA deposition and palpable purpura

E. Renal vein thrombosis because endothelial injury from diarrhea produces isolated venous occlusion

179. A patient with a mature AV fistula has a cold painful hand, paresthesias, and diminished distal pulses that worsen during dialysis. Which complication is most likely?

A. Central venous stenosis causing isolated facial edema without distal hand ischemia

B. Dialysis disequilibrium syndrome causing cerebral edema and symmetric hand pain

C. Peritoneal dialysis peritonitis causing referred pain to the vascular access arm

D. Uremic neuropathy that appears abruptly primarily when the fistula is cannulated

E. Hemodialysis access-induced distal ischemia from diversion of arterial flow through the fistula

180. A kidney transplant recipient on tacrolimus begins clarithromycin and soon develops tremor, hyperkalemia, and rising creatinine with a very high tacrolimus trough. What is the mechanism?

A. CYP3A induction accelerates tacrolimus clearance and causes acute rejection from subtherapeutic levels

B. CYP3A inhibition decreases tacrolimus metabolism and raises calcineurin-inhibitor exposure

C. Inhibition of renal tubular creatinine secretion causes an isolated creatinine rise without tacrolimus toxicity

D. Chelation of tacrolimus in the gut prevents absorption and lowers the trough concentration

E. Activation of P-glycoprotein exclusively increases tacrolimus excretion into urine

181. A patient with panic-induced hyperventilation has an acute $PaCO_2$ decrease from 40 to 20 mm Hg. Which bicarbonate response is expected initially?

A. A modest fall in bicarbonate from acute intracellular and extracellular buffering before renal adaptation

B. A large rise in bicarbonate because renal compensation begins immediately during respiratory alkalosis

C. No change in bicarbonate because respiratory disorders rarely alter serum bicarbonate concentration

D. A severe fall in bicarbonate from immediate renal bicarbonaturia occurring within a few minutes

E. A rise in bicarbonate caused by lactic acid consumption during hyperventilation

182. A 65-year-old man with squamous cell carcinoma has calcium 13.4 mg/dL, phosphate 2.1 mg/dL, suppressed intact PTH, and elevated PTH-related peptide. Which diagnosis is most likely?

A. Primary hyperparathyroidism with autonomous intact PTH secretion from a parathyroid adenoma

B. Vitamin D intoxication causing increased intestinal calcium and phosphate absorption

C. Granulomatous disease causing excess calcitriol with increased serum phosphate

D. Humoral hypercalcemia of malignancy mediated by PTH-related peptide

E. Familial hypocalciuric hypercalcemia caused by altered calcium-sensing receptor function

183. A 60-year-old woman with chronic SIADH cannot maintain an acceptable sodium concentration with fluid restriction. She is alert and clinically stable. Which oral therapy can increase electrolyte-free water excretion by increasing urinary solute load?

A. Oral desmopressin to increase collecting-duct water reabsorption and concentrate the urine further

B. High-dose fludrocortisone to increase sodium retention as the primary treatment for euvolemic SIADH

C. Oral urea to increase osmole excretion and permit greater renal water clearance

D. Isotonic saline alone because its sodium content reliably corrects persistent SIADH

E. Free-water supplementation to lower urine osmolality and restore normal vasopressin physiology

184. A hemodialysis patient has progressive arm and facial swelling on the side of a functioning AV access and a history of multiple prior central venous catheters. What is the most likely cause?

A. Central venous stenosis causing venous hypertension in the access arm

B. Access steal syndrome causing arterial insufficiency and reduced venous pressure

C. Dialysis disequilibrium syndrome causing localized venous congestion around the fistula

D. Acute glomerulonephritis causing unilateral edema limited to the catheter side

E. Peritoneal leak causing isolated upper-extremity swelling during hemodialysis

185. A patient on continuous kidney replacement therapy receives regional citrate anticoagulation and develops rising total calcium with low ionized calcium and increasing calcium infusion requirements. What complication is most concerning?

A. Heparin-induced thrombocytopenia because citrate directly activates platelet factor 4 antibodies

B. Citrate accumulation, often from impaired citrate metabolism, causing systemic calcium chelation

C. Hypermagnesemia because citrate selectively binds magnesium but rarely calcium

D. Dialysis disequilibrium syndrome because citrate causes cerebral urea accumulation

E. Metabolic acidosis from direct hydrochloric acid infusion inherent to many citrate protocol

186. A patient with severe hypertension develops headache, seizures, visual symptoms, and MRI findings of posterior vasogenic edema. What is the most likely syndrome?

A. Osmotic demyelination syndrome caused by rapid correction of chronic hypernatremia

B. Dialysis disequilibrium syndrome occurring primarily during first hemodialysis and rarely with hypertension

C. Posterior reversible encephalopathy syndrome associated with abrupt blood-pressure elevation and endothelial dysfunction

D. Subarachnoid hemorrhage because posterior vasogenic edema is strongly suggestive of aneurysmal rupture

E. Central pontine myelinolysis caused by untreated severe hypertension without sodium change

187. A patient has recurrent complement-mediated thrombotic microangiopathy without severe ADAMTS13 deficiency or Shiga-toxin infection. Which targeted therapy may be indicated?

A. Long-term high-dose aspirin alone because platelet cyclooxygenase is the primary complement regulator

B. Desmopressin because von Willebrand factor release corrects complement pathway mutations

C. Plasma exchange as the primarily effective chronic therapy regardless of the complement mechanism

D. ACE-inhibitor monotherapy because efferent dilation directly stops terminal complement activation

E. Complement C5 inhibition for complement-mediated atypical hemolytic uremic syndrome

188. A patient with advanced CKD is approaching dialysis and has poor superficial veins but needs durable hemodialysis access. Which access may be preferred over a chronic tunneled catheter if a fistula is unlikely to mature?

A. A non-tunneled femoral catheter maintained permanently because it has the lowest infection risk

B. A peripherally inserted central catheter because it preserves veins needed for later access creation

C. Repeated temporary jugular catheters changed weekly as the preferred long-term strategy

D. A peripheral intravenous cannula used for high-flow hemodialysis three times each week

E. An arteriovenous graft as a durable access with lower infection risk than chronic catheter use

189. A kidney transplant recipient receiving an mTOR inhibitor develops wound dehiscence and new proteinuria. Which drug effect is most likely contributing?

A. mTOR inhibition can impair wound healing and may worsen or provoke proteinuria

B. mTOR inhibition universally accelerates wound healing by stimulating fibroblast proliferation

C. mTOR inhibitors prevent most proteinuria by directly restoring the glomerular basement membrane

D. These findings are supportive for belatacept and do not occur with sirolimus or everolimus

E. The medication causes primarily hyperkalemia and has no recognized effect on wound repair or protein excretion

190. A patient with AKI is being considered for dialysis solely because creatinine is 6.5 mg/dL. He is asymptomatic, potassium and acid-base status are controlled, and volume status is manageable. Which principle is correct?

A. A creatinine above 6 mg/dL is an strong standard indication for immediate dialysis

B. Dialysis is contraindicated until creatinine exceeds 10 mg/dL regardless of symptoms

C. Serum creatinine directly measures uremic toxin concentration and therefore dictates the exact start time

D. Kidney replacement therapy should begin as soon as creatinine rises above baseline even without complications

E. Dialysis should be initiated for clinical indications and complications rather than a creatinine threshold alone

191. A patient receiving tacrolimus develops hypertension, hyperkalemia, tremor, and a rise in creatinine. Which renal mechanism is characteristic of acute calcineurin-inhibitor toxicity?

A. Reversible renal arteriolar vasoconstriction that reduces renal blood flow and GFR

B. Irreversible bilateral ureteral obstruction caused by crystal precipitation in the collecting system

C. Selective afferent vasodilation that produces severe glomerular hyperfiltration and polyuria

D. Direct V2-receptor blockade causing nephrogenic diabetes insipidus as the primary lesion

E. Inhibition of proximal bicarbonate reabsorption causing isolated type 2 renal tubular acidosis

192. A 41-year-old woman has hypertension, hypokalemic metabolic alkalosis, low renin, low aldosterone, and chronic licorice ingestion. Which mechanism explains the findings?

A. Direct stimulation of aldosterone synthesis causing high aldosterone with suppressed renin

B. Blockade of epithelial sodium channels producing renal sodium wasting and hyperkalemia

C. Inhibition of 11-beta-hydroxysteroid dehydrogenase allowing cortisol to activate mineralocorticoid receptors

D. Inhibition of sodium-chloride cotransport in the distal convoluted tubule causing Gitelman physiology

E. Activation of vasopressin V2 receptors causing isolated water retention without potassium wasting

193. A patient on hemodialysis has persistently inadequate solute clearance despite completing prescribed session times. The AV fistula has a strong thrill. Which issue should be evaluated?

A. Excessive dietary calcium because calcium intake is the main determinant of urea clearance

B. Low serum albumin because albumin concentration directly sets dialyzer membrane surface area

C. Access recirculation or insufficient delivered blood flow reducing effective dialysis dose

D. High hemoglobin because erythrocytes prevent most urea movement across the dialysis membrane

E. Urine supportive gravity because residual concentrating ability determines dialyzer clearance

194. A patient develops blue toes, livedo reticularis, eosinophilia, and AKI several days after aortic angiography. Complement is low. Which diagnosis is most likely?

A. Contrast-associated tubular injury because blue toes and eosinophilia are classic isolated contrast effects

B. Acute renal vein thrombosis because cholesterol crystals preferentially obstruct large veins

C. Anti-GBM disease because angiography stimulates antibodies against vascular basement membranes

D. Minimal-change disease because aortic instrumentation causes podocyte effacement and nephrotic syndrome

E. Cholesterol crystal embolization to small renal and systemic arteries after vascular instrumentation

195. A patient with malignant hypertension has AKI, thrombocytopenia, hemolytic anemia with schistocytes, and very high blood pressure. Which renal vascular lesion is characteristic?

A. Minimal-change podocytopathy without vascular injury as the defining renal lesion

B. Isolated renal vein thrombosis causing most findings through venous hypertension

C. Membranous nephropathy with subepithelial immune deposits and no arteriolar changes

D. Amyloidosis causing Congo-red-positive deposits as the primary mechanism of hypertensive crisis

E. Hyperplastic arteriolosclerosis with endothelial injury and thrombotic microangiopathy

196. A patient with psychogenic polydipsia has sodium 120 mEq/L and urine osmolality 55 mOsm/kg. Which finding most strongly argues against SIADH?

A. A urine sodium concentration above 30 mEq/L while dietary sodium intake is adequate

B. A normal thyroid-stimulating hormone concentration during evaluation of hyponatremia

C. Absence of peripheral edema or clinically apparent ascites on physical examination

D. Urine that is nearly maximally dilute despite the low serum osmolality

E. A low serum uric acid concentration caused by increased urate clearance

197. A patient with AKI develops severe hyperkalemia with QRS widening. After intravenous calcium, which treatment most rapidly lowers serum potassium concentration?

A. Oral potassium chloride to restore transcellular potassium equilibrium

B. Spironolactone to block aldosterone and increase renal potassium retention

C. A nonselective beta blocker because beta-2 blockade drives potassium into skeletal muscle

D. Intravenous insulin with glucose to shift potassium into cells

E. Normal saline alone because extracellular volume expansion immediately shifts potassium intracellularly

198. A patient with ESKD chooses conservative kidney management rather than dialysis after informed discussion. Which statement is most appropriate?

A. Conservative management means no medical treatment should be offered once dialysis is declined

B. Dialysis must be initiated against the patient's wishes whenever eGFR falls below a fixed threshold

C. Palliative symptom treatment is incompatible with ongoing management of anemia, edema, or acidosis

D. Kidney transplantation should be performed without consent because conservative care is medically invalid

E. Conservative management is an active treatment pathway focused on symptoms, goals, and quality of life without dialysis

199. A transplant recipient has rising creatinine. Tacrolimus level is therapeutic, ultrasound shows no obstruction, and urinalysis is bland. What is the best next diagnostic step if dysfunction remains unexplained?

A. Increase immunosuppression empirically without tissue diagnosis because most unexplained dysfunction is rejection

B. Stop most immunosuppression because a bland urine sediment argues against rejection

C. Begin dialysis permanently because any unexplained creatinine rise indicates irreversible graft failure

D. Perform native-kidney biopsy because the allograft cannot be safely sampled after transplantation

E. Kidney allograft biopsy to distinguish rejection, drug toxicity, infection, and other intrinsic causes

200. An adolescent has polyuria, salt wasting, anemia, growth impairment, bland urine sediment, and progressive kidney failure. Imaging shows small kidneys with corticomedullary cysts. What inherited disorder is most likely?

A. Autosomal dominant polycystic kidney disease causing enlarged kidneys and adult-onset hypertension in many case

B. Nephronophthisis, a ciliopathy causing chronic tubulointerstitial disease and corticomedullary cysts

C. Medullary sponge kidney causing preserved function and isolated nephrolithiasis without progression

D. Alport syndrome causing sensorineural hearing loss from a glomerular basement-membrane collagen defect

E. Minimal-change disease causing nephrotic syndrome and normal renal imaging

201. A patient has hypokalemia despite aggressive potassium replacement. Magnesium is 0.9 mg/dL. What is the most important next step?

A. Stop potassium replacement because magnesium deficiency makes potassium administration contraindicated

B. Administer bicarbonate to shift potassium out of cells and correct the low serum concentration

C. Replete magnesium because magnesium deficiency promotes renal potassium wasting and refractory hypokalemia

D. Restrict dietary potassium because magnesium deficiency causes pseudohypokalemia rather than true depletion

E. Begin a loop diuretic to reduce distal potassium secretion by decreasing sodium delivery

202. A patient with CKD has progressive decline in eGFR and persistent proteinuria. Which measure best improves longitudinal assessment rather than relying on a single creatinine?

A. Base prognosis on one serum creatinine value because kidney function is static in chronic disease

B. Ignore albuminuria after CKD is diagnosed because it no longer provides prognostic information

C. Use serum sodium trends as the primary marker of nephron loss instead of eGFR

D. Follow serial eGFR and albuminuria trends and investigate changes that exceed expected biologic variability

E. Measure urine color because darker urine quantitatively tracks chronic GFR decline

203. A patient with atrial fibrillation develops sudden severe flank pain, hematuria, elevated LDH, and a wedge-shaped perfusion defect in one kidney. What is the most likely diagnosis?

A. Renal papillary necrosis because papillary ischemia produces wedge-shaped cortical perfusion defects

B. Acute pyelonephritis because bacterial infection causes abrupt renal arterial occlusion

C. Renal vein thrombosis because venous thrombosis typically produces a sharply wedge-shaped arterial defect

D. Minimal-change disease because nephrotic proteinuria causes isolated renal cortical infarction without thrombosis

E. Acute renal infarction from arterial thromboembolism related to atrial fibrillation

204. A patient with severe AKI has metabolic acidosis with pH 7.05 despite treatment of shock. Which consideration is most appropriate?

A. Acidemia should rarely be treated because low pH is an adaptive response that cannot harm the cardiovascular system

B. Severe refractory acidemia is a potential indication for bicarbonate therapy in selected patients or kidney replacement therapy

C. Sodium bicarbonate often eliminates the need for dialysis regardless of volume status or ongoing acid production

D. Dialysis is indicated primarily for alkalemia because extracorporeal therapy cannot correct metabolic acidosis

E. Acid-base status is less informative in AKI because primarily potassium and creatinine determine treatment

205. A patient receiving chemotherapy for bulky lymphoma develops potassium 6.3 mEq/L, phosphate 7.2 mg/dL, uric acid 12 mg/dL, calcium 6.8 mg/dL, and rising creatinine. What syndrome is present?

A. Hungry bone syndrome caused by rapid skeletal uptake of calcium and phosphate after parathyroidectomy

B. Refeeding syndrome caused by insulin-mediated intracellular shifts of phosphate, potassium, and magnesium

C. Primary hyperparathyroidism causing hypercalcemia, hypophosphatemia, and recurrent nephrolithiasis

D. Tumor lysis syndrome with release of intracellular potassium, phosphate, and nucleic acid metabolites

E. Rhabdomyolysis without tumor lysis because uric acid is not affected by cell breakdown

206. A patient with hypertension develops an abrupt rise in creatinine after starting an ACE inhibitor. Which finding would most strongly suggest bilateral renal artery stenosis?

A. A small stable creatinine increase with improved albuminuria and normal potassium

B. A low urine albumin concentration with substantially normal vascular examination

C. Isolated microscopic hematuria that resolves after vigorous exercise

D. Marked polyuria with very dilute urine responding fully to desmopressin

E. A large creatinine increase accompanied by diffuse atherosclerotic disease or recurrent flash pulmonary edema

207. A patient with CKD has eGFR 24 mL/min/1.73 m2 and worsening albuminuria. Which care step is appropriate?

A. Avoid nephrology referral until dialysis is required because earlier care does not affect CKD complications

B. Stop monitoring anemia and mineral metabolism because these occur primarily after dialysis begins

C. Place a central venous catheter immediately even if dialysis is not expected for years

D. Discontinue cardiovascular risk reduction because advanced CKD eliminates benefit from preventive care

E. Nephrology follow-up with preparation for complications and future kidney replacement options as risk increases

208. A kidney transplant recipient has progressive proteinuria, declining function, circulating donor-specific antibodies, and biopsy showing double contours of glomerular capillary walls. What process is most likely?

A. Acute tubular injury because ischemia produces double-contour glomerular capillary walls years later

B. Chronic antibody-mediated rejection with transplant glomerulopathy

C. Minimal-change recurrence because foot-process effacement creates chronic donor-supportive antibodies

D. Ureteral obstruction because hydronephrosis leads to transplant glomerulopathy on biopsy

E. Calciphylaxis because vascular calcification causes donor-supportive HLA antibody formation

209. A patient with severe hypernatremia has a total-body water deficit. Which variable is essential for estimating the deficit using the usual bedside formula?

A. The urine sodium concentration alone without any estimate of body water stores

B. The serum potassium concentration as the sole determinant of extracellular tonicity

C. An estimate of total-body water based on body weight, age, and sex

D. The measured creatinine clearance because free-water deficit is independent of body size

E. The plasma bicarbonate concentration because acid-base status directly defines water deficit

210. A patient with oliguric AKI has pulmonary edema despite escalating loop diuretics. Which statement about diuretics is correct?

A. Loop diuretics reliably shorten the duration of acute tubular injury by regenerating tubular epithelial cells

B. High urine output after furosemide strongly suggests that intrinsic tubular injury is absent

C. Diuretics are contraindicated in most AKI because any natriuresis worsens tubular necrosis

D. Diuretics can manage volume overload but do not convert established acute tubular injury into faster renal recovery

E. Failure to respond to a loop diuretic argues against the need for dialysis in pulmonary edema

211. A patient with CKD is taking metformin, gabapentin, and several antihypertensives. Why is medication review especially important as eGFR declines?

A. most medications become more rapidly cleared as GFR declines, causing standard treatment failure

B. primarily antibiotics require renal dose adjustment because other drug classes bypass the kidney

C. Serum creatinine affects drug absorption but rarely drug elimination or metabolite accumulation

D. CKD prevents most hepatic metabolism, so renal function is less informative to medication selection

E. Renal clearance of many drugs and metabolites falls, increasing toxicity unless doses or agents are adjusted

212. A patient with infective endocarditis develops hematuria, proteinuria, low complement, and AKI. Which renal process is most likely?

A. Immune-complex glomerulonephritis related to active endocardial infection

B. Primary membranous nephropathy because endocarditis causes PLA2R autoantibody formation

C. Anti-GBM disease because bacterial vegetations induce linear basement-membrane antibody deposition

D. Minimal-change disease because complement levels are typically low in isolated podocyte disorders

E. Alport syndrome because infection unmasks inherited type IV collagen abnormalities

213. A patient on lithium develops nephrogenic diabetes insipidus. Which pharmacologic intervention reduces lithium entry into collecting-duct principal cells?

A. Desmopressin blockade of V2 receptors to prevent lithium uptake from the tubular lumen

B. Furosemide blockade of NKCC2 because lithium enters principal cells primarily through loop transporters

C. Spironolactone activation of mineralocorticoid receptors to increase lithium excretion

D. Acetazolamide stimulation of carbonic anhydrase to restore aquaporin expression

E. Amiloride blockade of ENaC channels through which lithium enters principal cells

214. A patient with profuse diarrhea has potassium 2.7 mEq/L and bicarbonate 16 mEq/L. Which acid-base and potassium pattern is most expected?

A. Normal-anion-gap metabolic acidosis with total-body potassium depletion from gastrointestinal losses

B. High-anion-gap metabolic acidosis with total-body potassium excess from retained organic acids

C. Metabolic alkalosis with potassium retention because stool contains little bicarbonate or potassium

D. Respiratory alkalosis with intracellular potassium release and increased total-body potassium stores

E. Type 4 renal tubular acidosis with hyperkalemia caused by reduced aldosterone action

215. A patient with hypertension has a clinic reading of 170/96 mm Hg obtained immediately after walking in, with an undersized cuff. Repeat measurement after 5 minutes using an appropriate cuff is 146/88 mm Hg. What principle does this illustrate?

A. The first blood-pressure reading is often the primarily valid measurement in a clinical encounter

B. Cuff size does not affect blood pressure and should be chosen for clinician convenience

C. Accurate standardized measurement is essential before diagnosing or escalating treatment for hypertension

D. Rest before measurement lowers pressure artifactually and therefore should be avoided

E. Automated and home measurements are invalid because primarily immediate manual readings reflect true risk

216. A patient with advanced CKD is taking an NSAID for chronic back pain and develops worsening edema and creatinine. Which renal effect of NSAIDs contributes?

A. Stimulation of prostaglandin synthesis causing excessive afferent dilation and glomerular hyperfiltration

B. Selective efferent arteriolar dilation similar to the hemodynamic effect of ACE inhibitors

C. Direct activation of SGLT2 causing increased sodium-glucose reabsorption and osmotic diuresis

D. Inhibition of prostaglandin-mediated afferent arteriolar dilation, reducing renal perfusion in susceptible states

E. Blockade of mineralocorticoid receptors causing expected natriuresis and hyperkalemia without GFR change

217. A postoperative patient becomes anuric. Bladder scan shows a markedly distended bladder. Which intervention should occur first?

A. Prompt bladder catheterization to relieve lower urinary tract obstruction and assess urine output

B. Administer high-dose furosemide before evaluating obstruction because anuria often reflects tubular injury

C. Begin corticosteroids because postoperative anuria is most often acute interstitial nephritis

D. Start hemodialysis immediately without relieving the obstruction because decompression cannot restore GFR

E. Give desmopressin because urinary retention is caused by deficient vasopressin secretion

218. A transplant candidate has a living donor with an incompatible ABO blood group. Which statement is most accurate?

A. ABO incompatibility makes kidney transplantation impossible under most circumstances

B. Some centers can perform ABO-incompatible transplantation using desensitization and careful immunologic management

C. ABO compatibility is less informative because kidney endothelial cells do not express blood-group antigens

D. primarily HLA identity matters, so ABO mismatch carries no risk of antibody-mediated rejection

E. ABO-incompatible grafts rarely require antibody reduction because natural isohemagglutinins do not bind the kidney

219. A patient with hepatitis B infection develops nephrotic syndrome and biopsy showing membranous nephropathy. Which principle is most important?

A. Assume the membranous lesion is primary because hepatitis B cannot cause immune-complex glomerular disease

B. Use high-dose glucocorticoids alone because viral replication is unaffected by immunosuppression

C. Stop antiviral therapy because reducing viral antigen increases immune-complex formation

D. Treat the underlying hepatitis B infection and consider its role before using immunosuppression

E. Treat primarily with diuretics because secondary membranous nephropathy rarely improves when the trigger is controlled

220. A patient with CKD and heart failure is taking an ACE inhibitor, diuretic, and NSAID and develops abrupt AKI. Which interaction best explains the risk?

A. Combined reduction in afferent flow and efferent tone with volume depletion markedly lowers glomerular filtration pressure

B. most three drugs constrict both afferent and efferent arterioles, producing severe glomerular hypertension

C. The NSAID dilates the afferent arteriole while the ACE inhibitor constricts the efferent arteriole

D. The diuretic expands plasma volume, increasing hydrostatic pressure and directly injuring podocytes

E. The combination causes isolated postrenal obstruction by precipitating crystals in both ureters

221. A 40-year-old patient with hypernatremia has polyuria of 6 L/day. Urine osmolality is 650 mOsm/kg and urine glucose is markedly elevated. Which process is most likely driving the polyuria?

A. Central diabetes insipidus causing absent vasopressin and a persistently very dilute urine

B. Nephrogenic diabetes insipidus causing renal vasopressin resistance and urine osmolality below plasma

C. Primary polydipsia causing excessive water intake with low serum sodium rather than hypernatremia

D. Osmotic diuresis from unreabsorbed solute causing water loss despite preserved concentrating ability

E. Low-solute intake limiting urine volume and preventing excretion of ingested free water

222. A patient with severe hypokalemia has muscle weakness and new U waves on ECG. Which complication is most concerning?

A. Acute cerebral edema due to rapid intracellular water entry caused directly by low potassium

B. Hypercalcemic crisis due to potassium-mediated activation of parathyroid hormone secretion

C. Pulmonary edema due to expected renal sodium retention caused by many episode of hypokalemia

D. Ventricular arrhythmia due to impaired cardiac repolarization from severe potassium depletion

E. Hemolytic anemia due to direct potassium depletion of circulating erythrocytes

223. A patient has bilateral ureteral obstruction relieved after several days. Urine output rises to 7 L/day. Which complication is most important to monitor?

A. Permanent SIADH because relief of obstruction often causes persistent water retention

B. Postobstructive diuresis causing volume depletion and urinary electrolyte losses

C. Hypervolemia from immediate complete reabsorption of most retained sodium and water

D. Severe hyperkalemia because postobstructive diuresis prevents urinary potassium excretion

E. Nephrotic syndrome because decompression acutely increases glomerular albumin permeability

224. A patient with CKD and chronic edema is advised to reduce dietary sodium. What is the main physiologic benefit?

A. Lower sodium intake reduces extracellular volume expansion and improves blood-pressure and edema control

B. Sodium restriction directly increases nephron number and reverses established renal fibrosis

C. Lower sodium intake eliminates the need for most antihypertensive and diuretic therapy in advanced CKD

D. Sodium restriction raises intraglomerular pressure and intentionally increases albuminuria

E. Dietary sodium has no effect on extracellular volume once glomerular filtration falls below normal

225. A patient with difficult-to-control hypertension is taking ibuprofen daily, an oral decongestant, and an estrogen-containing contraceptive. What is the best next step?

A. Assume the hypertension is genetic because common medications cannot meaningfully affect blood pressure

B. Review and remove medications or substances that can raise blood pressure before labeling treatment failure

C. Add a sixth antihypertensive without changing contributory drugs because interactions are clinically less informative

D. Stop most antihypertensive drugs and continue the pressor medications to clarify the baseline pressure

E. Begin fludrocortisone because NSAIDs and sympathomimetics cause hypertension by mineralocorticoid deficiency

226. A kidney transplant recipient has multiple cutaneous squamous-cell carcinomas. Which principle is appropriate?

A. Reduce modifiable ultraviolet exposure and reassess immunosuppression intensity with transplant and dermatology teams

B. Increase immunosuppression because stronger T-cell suppression lowers the risk of skin cancer recurrence

C. Avoid dermatologic surveillance because transplant-associated skin cancer rarely recurs

D. Use live viral vaccines to stimulate antitumor immunity while maintaining full immunosuppression

E. Stop most immunosuppression immediately because any skin cancer requires graft abandonment

227. A family has autosomal dominant CKD with bland urine sediment, early hyperuricemia and gout, and small to normal-sized kidneys without many cysts. Which diagnosis is most likely?

A. Autosomal dominant polycystic kidney disease because numerous enlarged bilateral cysts are often present

B. Autosomal dominant tubulointerstitial kidney disease related to UMOD or another tubular gene

C. IgA nephropathy because recurrent gross hematuria is required for inherited tubulointerstitial disease

D. Alport syndrome because sensorineural hearing loss is expected in UMOD-associated disease

E. Membranous nephropathy because nephrotic-range proteinuria is the typical presentation of ADTKD

228. A patient with nephrotic syndrome has marked edema, serum albumin 1.8 g/dL, and hyperlipidemia. Which mechanism contributes to edema formation?

A. Increased plasma oncotic pressure draws water into the interstitium despite low serum albumin

B. Complete suppression of renal sodium reabsorption causes expected intravascular volume expansion

C. Loss of albumin raises effective circulating volume in many nephrotic patient and eliminates RAAS activity

D. Hyperlipidemia directly increases urine osmolality enough to produce isolated free-water loss

E. Renal sodium retention combined with reduced plasma oncotic pressure promotes expansion of interstitial fluid

229. A patient with CKD has secondary hyperparathyroidism and asks why calcitriol levels fall as kidney function declines. Which explanation is correct?

A. The failing kidney converts excess calcitriol to PTH, depleting vitamin D stores directly

B. Reduced GFR increases 1-alpha hydroxylase activity, so calcitriol usually rises in advanced CKD

C. Calcitriol is synthesized primarily in the liver, so kidney function cannot affect its concentration

D. Reduced renal 1-alpha hydroxylation and increased FGF23 activity lower production of active vitamin D

E. Phosphate retention directly converts calcitriol into inactive parathyroid hormone within plasma

230. A patient with recurrent calcium oxalate stones has hypercalciuria despite normal serum calcium. Which medication is most likely to reduce urinary calcium excretion and stone recurrence?

A. A loop diuretic because inhibition of NKCC2 increases calcium reabsorption in the thick ascending limb

B. A thiazide or thiazide-like diuretic combined with dietary sodium restriction

C. Acetazolamide because alkaline urine and reduced citrate excretion prevent calcium phosphate precipitation

D. High-dose sodium chloride tablets because sodium loading decreases renal calcium excretion

E. An SGLT2 inhibitor because glycosuria directly chelates urinary calcium and dissolves calcium oxalate crystals

231. A woman with placental abruption and hemorrhagic shock develops persistent anuria and severe AKI. Imaging shows poor cortical perfusion, and kidney recovery is minimal. Which diagnosis is most likely?

A. Prerenal azotemia because prolonged anuria with absent cortical perfusion indicates intact renal parenchyma

B. Acute renal cortical necrosis from severe obstetric ischemic and microvascular injury

C. Minimal-change disease because obstetric hemorrhage causes isolated podocyte effacement

D. Acute interstitial nephritis because placental abruption triggers a delayed allergic reaction in renal interstitium

E. Postrenal obstruction because bilateral hydronephrosis is the usual cause of obstetric cortical ischemia

232. A patient with hyperphosphatemia from advanced CKD takes sevelamer with meals. What is its mechanism?

A. Increasing renal phosphate filtration by directly dilating the afferent arteriole

B. Stimulating PTH secretion to drive phosphate into bone independently of intestinal absorption

C. Activating vitamin D receptors to increase intestinal phosphate uptake and lower serum phosphate

D. Binding dietary phosphate in the gastrointestinal tract to reduce phosphate absorption

E. Inhibiting proximal tubular SGLT2 to increase urinary phosphate excretion as its primary action

233. A patient with stage 4 CKD has a rising creatinine over several weeks and new bilateral hydronephrosis on ultrasound. What is the most important next step?

A. Assume the decline is likely CKD progression because chronic disease argues against superimposed obstruction

B. Increase ACE-inhibitor dose immediately because hydronephrosis reflects excessive efferent constriction

C. Evaluate and relieve urinary tract obstruction because a reversible postrenal component may be present

D. Begin phosphate binders because hydronephrosis is caused by calcium-phosphate deposition in most cases

E. Wait three months before acting because any renal abnormality must persist that long before treatment

234. A patient with chronic SIADH has sodium 124 mEq/L. After a new medication is started, urine output rises sharply and sodium increases rapidly. Which medication class is most likely responsible?

A. Mineralocorticoid receptor antagonist causing natriuresis and increased potassium retention

B. Epithelial sodium-channel blocker causing natriuresis through the collecting duct

C. Carbonic anhydrase inhibitor causing bicarbonaturia and proximal sodium loss

D. Alpha-1 adrenergic antagonist lowering vascular tone without a direct aquaretic effect

E. Vasopressin V2-receptor antagonist producing aquaresis without major sodium loss

235. A patient with toluene exposure develops weakness, hypokalemia, and a normal-anion-gap metabolic acidosis after prolonged solvent abuse. Which renal finding is most likely?

A. Absent urinary ammonium excretion from classic distal renal tubular acidosis in many case

B. Severe glucosuria and aminoaciduria from an expected Fanconi syndrome caused by most solvent exposure

C. Markedly elevated serum bicarbonate due to metabolism of toluene into alkali equivalents

D. High urinary ammonium excretion with hippurate loss that can produce a negative urine anion gap

E. Persistent hyperkalemia caused by hypoaldosteronism and reduced distal sodium delivery

236. A patient with CKD has hypertension, albuminuria, and no contraindication to renin-angiotensin system blockade. Why is blood-pressure control especially important?

A. It eliminates the need to monitor albuminuria because blood pressure is unrelated to glomerular injury

B. It reduces cardiovascular risk and can slow kidney disease progression, particularly when albuminuria is present

C. It reverses most established nephron loss once systolic pressure is below the treatment target

D. It prevents many episode of acute kidney injury regardless of volume status or nephrotoxin exposure

E. It makes dietary sodium intake less informative because hypertension in CKD is rarely volume dependent

237. A kidney transplant recipient develops severe diarrhea and leukopenia while taking mycophenolate mofetil. Which medication is the most likely cause?

A. Tacrolimus because calcineurin inhibitors most characteristically cause profound chronic diarrhea and leukopenia

B. Prednisone because low-dose glucocorticoids usually cause severe marrow suppression and neutropenia

C. Belatacept because co-stimulatory blockade predictably causes acute secretory diarrhea in nearly most recipients

D. Basiliximab because induction therapy months earlier is the usual cause of persistent gastrointestinal toxicity

E. Mycophenolate toxicity causing gastrointestinal injury and bone-marrow suppression

238. A patient has nephrotic syndrome with very low serum albumin and asks why thrombosis risk is increased. Which change contributes?

A. Urinary loss of fibrinogen causing a profound bleeding tendency rather than hypercoagulability

B. Suppression of platelet function similar to uremic bleeding in most nephrotic patients

C. Urinary loss of anticoagulant proteins together with increased procoagulant factors and platelet activation

D. Severe thrombocytopenia caused by urinary platelet loss through the glomerular filtration barrier

E. Decreased hepatic synthesis of clotting factors because hypoalbuminemia turns off protein production

239. A patient with severe AKI has uremic pericarditis with chest pain and a pericardial friction rub. What is the most appropriate treatment?

A. High-dose NSAID therapy alone because dialysis worsens uremic pericardial inflammation

B. Delay dialysis until cardiac tamponade develops because pericarditis alone is not a uremic indication

C. Urgent kidney replacement therapy to treat the uremic complication

D. Give anticoagulation routinely because uremic pericarditis is caused by coronary thrombosis

E. Use colchicine alone and ignore kidney failure because uremic toxin removal does not affect pericarditis

240. A patient with advanced CKD is being evaluated for kidney transplantation before dialysis is required. What is a major potential advantage of preemptive transplantation for an eligible patient?

A. Avoiding or shortening dialysis exposure while providing kidney replacement with a functioning allograft

B. Eliminating the need for lifelong immunosuppression because preemptive grafts are immunologically tolerated

C. Preventing most future cardiovascular disease because transplantation normalizes risk to that of healthy controls

D. Guaranteeing permanent graft survival because transplantation before dialysis prevents rejection

E. Removing the need for donor evaluation because preemptive transplantation uses no living or deceased donor

Answers

1. D. CKD is defined by abnormalities of kidney structure or function present for at least 3 months and associated with health implications. An eGFR persistently below 60 mL/min/1.73 m2 meets that definition even if albuminuria is absent. Kidney failure is generally reserved for much more advanced loss of function or need for kidney replacement therapy.

2. E. AKI is diagnosed by an acute rise in serum creatinine or reduction in urine output using standardized criteria. A rise of at least 0.3 mg/dL within 48 hours meets a creatinine criterion. Staging then depends on the magnitude of creatinine change and the duration and severity of oliguria.

3. E. IgA nephropathy classically causes recurrent gross hematuria that begins during or within a day or two of a mucosal infection. Complement is usually normal, and urine sediment is glomerular. Persistent proteinuria and reduced GFR are important prognostic features.

4. C. Hyperacute rejection occurs within minutes to hours when preformed antibodies react with donor ABO or HLA antigens, activating complement and thrombosis. Modern crossmatching makes it uncommon. The graft is usually unsalvageable once fulminant hyperacute rejection occurs.

5. E. Resistant hypertension with spontaneous or diuretic-associated hypokalemia is a classic indication to screen for primary aldosteronism. The aldosterone-to-renin ratio is the usual first biochemical step, with attention to medications, potassium status, and sampling conditions. Confirmatory testing and subtype evaluation follow when appropriate.

6. D. Winter's formula estimates expected PaCO2 as 1.5 × bicarbonate + 8 ± 2. With bicarbonate 11 mEq/L, the expected PaCO2 is about 24 to 27 mm Hg, so 25 represents appropriate respiratory compensation. A value substantially higher suggests concomitant respiratory acidosis; a much lower value suggests additional respiratory alkalosis.

7. E. This is classic SIADH: hypotonic hyponatremia with inappropriately concentrated urine, urine sodium above 30 mEq/L, and clinical euvolemia after excluding thyroid and adrenal disease. Small-cell lung cancer is a well-known ectopic source of ADH. Primary polydipsia would usually produce very dilute urine, whereas hypovolemia and heart failure would have different volume findings.

8. A. Both GFR category and albuminuria category independently predict CKD progression and cardiovascular risk. An albumin-to-creatinine ratio above 300 mg/g represents severely increased albuminuria and materially raises risk. Combining eGFR and albuminuria provides better prognostic information than eGFR alone.

9. B. Angiotensin II preferentially constricts the efferent arteriole, so ACE inhibition relaxes efferent tone and lowers intraglomerular pressure. This can reduce albuminuria and slow progression in appropriate CKD while producing a small early creatinine rise. The effect becomes hazardous when filtration is highly dependent on angiotensin II, such as severe bilateral renal artery stenosis or marked volume depletion.

10. E. Prerenal physiology from volume depletion often produces avid sodium and water reabsorption, a low FENa, concentrated urine, and bland sediment. FENa is not perfect and can be misleading with diuretics, CKD, pigment injury, or early intrinsic AKI. Clinical context and urine microscopy remain important.

11. C. Hypercalcemia with an inappropriately elevated PTH indicates primary hyperparathyroidism. Increased filtered calcium can promote nephrolithiasis. Familial hypocalciuric hypercalcemia is distinguished by low urinary calcium excretion and generally does not require parathyroid surgery.

12. D. Renin-angiotensin system blockade reduces efferent arteriolar tone, intraglomerular pressure, and albuminuria and is foundational therapy for many patients with albuminuric CKD. Blood pressure, creatinine, and potassium should be monitored after initiation or dose escalation. ACE inhibitors and ARBs should not be routinely combined because dual blockade increases adverse events.

13. D. Poststreptococcal GN typically follows streptococcal infection after a latent period and presents with nephritic urine, edema, and hypertension. C3 is commonly low and usually recovers. Supportive therapy is the mainstay while persistent hypocomplementemia or atypical features should prompt reconsideration of the diagnosis.

14. B. The timing after a new medication and the combination of pyuria, systemic hypersensitivity features, and AKI suggest acute interstitial nephritis. The classic fever-rash-eosinophilia triad is not always present. Management begins with withdrawal of the offending drug, with corticosteroids considered in selected cases after diagnostic assessment.

15. D. Acute T-cell-mediated rejection commonly presents with an otherwise unexplained rise in creatinine and biopsy evidence of interstitial inflammation and tubulitis, with or without arteritis. Treatment often begins with high-dose glucocorticoids, while severe or steroid-resistant cases may

require T-cell-depleting therapy. Biopsy is important because clinical findings overlap with drug toxicity and infection.

16. E. Fibromuscular dysplasia classically affects younger women and can produce a string-of-beads appearance from alternating stenosis and aneurysmal dilation. Renal involvement can cause severe renovascular hypertension. Percutaneous angioplasty is often effective when intervention is indicated.

17. E. A modest rise in creatinine after starting renin-angiotensin system blockade can occur because efferent arteriolar dilation lowers intraglomerular pressure. In a stable patient without major hyperkalemia or a large creatinine increase, therapy is generally continued with monitoring. A larger rise should prompt assessment for volume depletion, NSAID use, or bilateral renal artery stenosis.

18. C. Loop diuretics increase urinary sodium excretion and can make FENa less useful. A low fractional excretion of urea may support prerenal physiology in this setting, although it also has limitations and should not replace clinical assessment. Urine sediment and response to hemodynamic correction are often more informative.

19. E. The anion gap is sodium minus chloride minus bicarbonate: 140 − 104 − 12 = 24 mEq/L. With normal albumin, this is elevated and indicates accumulation of unmeasured anions. Causes include lactic acidosis, ketoacidosis, advanced kidney failure, and several toxic ingestions.

20. C. Markedly dilute urine in a patient with extreme water intake points to primary polydipsia. ADH is appropriately suppressed, but water intake exceeds the kidney's maximal ability to excrete free water. SIADH, adrenal insufficiency, and hypovolemia generally produce a urine osmolality well above 100 mOsm/kg.

21. A. SGLT2 inhibitors reduce CKD progression and cardiovascular events in many patients with diabetic and nondiabetic CKD, particularly when albuminuria is present. Their early small eGFR dip is hemodynamic and often stabilizes. They are not simply glucose-lowering drugs; kidney benefit extends beyond glycemic effects.

22. B. Staphylococcus-associated infection-related GN often occurs in older adults, especially those with diabetes, during an active infection. It can have IgA-dominant immune deposits and low complement. Treating the infection is central; indiscriminate immunosuppression can be harmful when infection remains active.

23. E. Antibody-mediated rejection is supported by evidence of allograft injury, antibody-endothelial interaction such as C4d or validated molecular findings, and donor-specific antibodies in the appropriate setting. Treatment commonly uses combinations of plasma exchange, IVIG, and other immunomodulatory therapies depending on severity and center practice. Chronic forms may lead to transplant glomerulopathy.

24. B. Ischemic acute tubular injury produces tubular epithelial cell injury and characteristic muddy brown granular casts. The same pattern can follow nephrotoxins. Management is supportive, including

hemodynamic optimization, avoidance of further injury, and treatment of complications while the tubules recover.

25. A. Finerenone can reduce kidney and cardiovascular events in appropriately selected patients with type 2 diabetes and persistent albuminuric CKD despite standard renin-angiotensin blockade. Potassium must be monitored because hyperkalemia can occur. Combining an ACE inhibitor with an ARB is not recommended because harm outweighs benefit.

26. E. Atherosclerotic renal artery stenosis is common, but many lesions are incidental. Recurrent flash pulmonary edema, refractory hypertension, and otherwise unexplained progressive kidney dysfunction are features that make hemodynamically important renovascular disease more likely. Revascularization is not routinely beneficial for every asymptomatic stenosis.

27. B. The urine anion gap is Na + K − Cl, which is negative here. In normal-gap acidosis, a negative value implies robust ammonium chloride excretion and therefore intact renal acidification, favoring gastrointestinal bicarbonate loss. A positive or insufficiently negative value suggests impaired renal ammonium excretion, as in some RTAs.

28. E. In low-perfusion states, local prostaglandins help preserve afferent arteriolar dilation. NSAIDs block prostaglandin synthesis and can precipitate a hemodynamic fall in GFR. They also promote sodium retention and can worsen edema, hypertension, and hyperkalemia.

29. C. PLA2R autoantibodies are strongly associated with primary membranous nephropathy and can help with diagnosis, risk assessment, and longitudinal monitoring in the proper context. Secondary causes still need consideration when clinical features suggest them. Biopsy shows subepithelial immune deposits and basement-membrane remodeling.

30. D. Anemia of CKD is primarily a hypoproliferative anemia from inadequate erythropoietin production relative to the degree of anemia, with additional contributions from inflammation and shortened red-cell survival. Iron status and other reversible causes should be assessed before or during ESA therapy. The typical pattern is normocytic with a low reticulocyte response.

31. B. Sepsis-associated AKI is multifactorial and involves altered microcirculation, inflammation, endothelial and tubular-cell stress, and hemodynamic changes. It is not explained solely by prolonged global ischemia. Management focuses on infection control, appropriate hemodynamics, avoidance of nephrotoxins, and supportive care.

32. D. Beer potomania is a low-solute hyponatremia. Even when the kidney can dilute urine, excretion of a large water load requires enough osmoles to carry that water out; very low protein and salt intake sharply reduces this capacity. The low urine osmolality and dietary history distinguish it from SIADH and salt-wasting states.

33. E. Calcineurin inhibitors can cause acute functional vasoconstriction and chronic structural injury including arteriolar hyalinosis and striped tubulointerstitial fibrosis. The findings are not perfectly

specific, so biopsy interpretation is integrated with exposure and alternative diagnoses. Dose reduction or regimen modification may be considered while maintaining adequate rejection prophylaxis.

34. C. ESA therapy can reduce transfusion needs and improve selected symptoms, but aggressive normalization of hemoglobin increases cardiovascular and thrombotic risk. Decisions are individualized based on symptoms, transfusion risk, comorbidity, and iron status. The objective is not to restore hemoglobin to a normal population value.

35. E. The classic symptomatic triad with paroxysmal hypertension raises concern for pheochromocytoma or paraganglioma. Plasma free metanephrines or 24-hour urinary fractionated metanephrines are sensitive screening tests. Biochemical confirmation generally precedes tumor localization imaging.

36. A. Distal RTA promotes calcium phosphate stones through alkaline urine and hypocitraturia. Potassium citrate corrects potassium depletion and raises urinary citrate, which complexes calcium and reduces crystallization. Adequate fluid intake is also important; deliberate fluid restriction would worsen stone risk.

37. E. Balanced crystalloids contain less chloride and a more physiologic strong-ion composition than 0.9% saline. Large chloride loads can produce hyperchloremic metabolic acidosis and may affect renal perfusion. Fluid choice still depends on the clinical context, and saline remains appropriate in selected conditions.

38. A. Nephrotic syndrome increases thrombotic risk through urinary loss of anticoagulant proteins, increased procoagulant factors, platelet activation, and hemoconcentration. Membranous nephropathy has a particularly strong association with venous thrombosis. Sudden flank pain and hematuria should raise concern for renal vein thrombosis.

39. D. Absolute iron deficiency is common in hemodialysis because of blood loss, phlebotomy, and increased requirements during ESA treatment. Intravenous iron is often more effective than oral therapy in this setting. Iron indices should be monitored to guide therapy and avoid both deficiency and excessive iron exposure.

40. E. Distal RTA causes impaired distal hydrogen secretion, non-anion-gap acidosis, urine pH that remains inappropriately high, and often hypokalemia. Calcium phosphate stones and nephrocalcinosis are characteristic because alkaline urine and hypocitraturia promote calcium phosphate precipitation. Proximal RTA can acidify urine once serum bicarbonate falls below the reabsorptive threshold.

41. E. Chronic heavy analgesic exposure can cause chronic tubulointerstitial nephritis and papillary necrosis, with small scarred kidneys and papillary calcifications. The urine sediment may show sterile pyuria and modest proteinuria. Removing the offending analgesics and managing CKD complications are central.

42. C. BK polyomavirus reactivation can cause allograft tubulointerstitial nephritis and graft dysfunction. The cornerstone of management is stepwise reduction of immunosuppression while monitoring viral load and graft function. Distinguishing BK nephropathy from rejection is crucial because intensifying immunosuppression can worsen viral injury.

43. A. CKD-mineral and bone disorder begins with phosphate retention and increased FGF23, followed by reduced calcitriol and disturbances in calcium-phosphate homeostasis. These changes stimulate parathyroid hyperplasia and PTH secretion. Long-standing secondary hyperparathyroidism can eventually become autonomous, producing tertiary hyperparathyroidism.

44. C. Large chloride loads from 0.9% saline can increase serum chloride relative to sodium and lower bicarbonate, producing a hyperchloremic normal-gap acidosis. This is a predictable physicochemical effect rather than accumulation of unmeasured organic acids. Balanced crystalloids reduce chloride exposure.

45. D. The patient has hypovolemic hypotonic hyponatremia, so restoring effective arterial volume with isotonic saline removes the nonosmotic stimulus for ADH. Fluid restriction and vaptans are inappropriate for hypovolemia. Hypertonic saline is reserved for severe neurologic symptoms and should not be used to rapidly normalize sodium.

46. E. Alpha blockade is established first to control catecholamine-mediated vasoconstriction and allow volume expansion. Beta blockade may then be added for persistent tachyarrhythmia. Giving a beta blocker first risks unopposed alpha-adrenergic stimulation and severe hypertension.

47. A. Minimal-change disease is a podocytopathy characterized by diffuse foot-process effacement with essentially normal glomeruli on light microscopy. It can occur in adults and may be associated with NSAIDs, Hodgkin lymphoma, or other triggers. Most patients respond to glucocorticoids, although adults may respond more slowly than children.

48. E. Severe secondary hyperparathyroidism increases osteoclastic and osteoblastic activity and produces high-turnover renal osteodystrophy, classically termed osteitis fibrosa. Adynamic bone disease is the opposite pattern, with low turnover often associated with excessive PTH suppression, diabetes, or calcium loading. Bone biopsy is the definitive way to distinguish uncertain forms of renal osteodystrophy.

49. B. The combination of bicarbonaturia with glucosuria, aminoaciduria, and phosphaturia indicates Fanconi syndrome and proximal RTA. The proximal tubule cannot reclaim filtered bicarbonate and other solutes. Distal RTA lacks this generalized proximal wasting pattern.

50. E. The urine heme dipstick detects peroxidase activity from both hemoglobin and myoglobin. In rhabdomyolysis, strong heme positivity with few RBCs suggests myoglobinuria. Management includes prompt volume resuscitation when appropriate and treatment of dangerous complications such as hyperkalemia.

51. B. SGLT2 inhibition decreases proximal sodium-glucose reabsorption, increasing sodium chloride delivery to the macula densa. Tubuloglomerular feedback then reduces glomerular hyperfiltration, producing a small early eGFR dip that usually stabilizes. This hemodynamic effect is part of the kidney-protective mechanism.

52. E. CMV donor-positive/recipient-negative transplantation carries high risk because the recipient lacks prior immunity and may acquire donor-derived infection. Valganciclovir-based prophylaxis or intensive preemptive strategies are used depending on protocol. CMV can cause fever, cytopenias, tissue-invasive disease, and indirect effects on the graft.

53. B. Adynamic bone disease is a low-turnover form of renal osteodystrophy associated with low or inappropriately normal PTH, diabetes, older age, and excessive calcium or vitamin D exposure. The skeleton has reduced ability to buffer calcium, so hypercalcemia can occur. Management focuses on avoiding excessive PTH suppression and calcium loading.

54. B. Secondary adaptive FSGS occurs with reduced nephron mass, obesity, reflux, or other hyperfiltration states. Proteinuria may be less dramatic and electron microscopy often shows less diffuse foot-process effacement than primary FSGS. Treatment emphasizes the underlying cause and reduction of intraglomerular pressure rather than routine immunosuppression.

55. E. Obstructive sleep apnea is strongly associated with resistant hypertension through sympathetic activation, intermittent hypoxemia, and neurohormonal effects. Screening and treatment are appropriate when clinical features are present. Weight management and continuous positive airway pressure may improve overall cardiovascular risk and blood-pressure control.

56. D. Seizure from profound hyponatremia is an indication for hypertonic saline, generally given in controlled boluses with frequent sodium checks. The goal is a modest initial rise sufficient to improve life-threatening symptoms, not immediate normalization. Overcorrection risks osmotic demyelination, especially in chronic hyponatremia.

57. C. Calciphylaxis is a life-threatening syndrome of painful ischemic skin lesions caused by calcification and thrombosis of small vessels, most often in advanced CKD or dialysis. Risk factors include disordered mineral metabolism, obesity, diabetes, female sex, and warfarin exposure. Management is multidisciplinary and commonly includes wound care, pain control, correction of mineral abnormalities, and sodium thiosulfate.

58. C. Dialysis is indicated when severe complications of AKI—such as refractory hyperkalemia, acidosis, volume overload, or uremic manifestations—cannot be controlled medically. The decision is based on clinical complications rather than creatinine or creatine kinase alone. Rhabdomyolysis itself is not an automatic dialysis indication.

59. B. Uric acid is much more soluble at higher urine pH, so alkalinization is the cornerstone of prevention and can dissolve existing pure uric acid stones. Potassium citrate is commonly used.

Allopurinol may be added when hyperuricosuria persists, but raising urine pH is the most direct intervention.

60. E. Trimethoprim-sulfamethoxazole is standard prophylaxis against Pneumocystis in many kidney transplant programs and also reduces some urinary and other bacterial infections. It does not prevent CMV, BK virus, or PTLD. Renal function, potassium, and cytopenias should be monitored.

61. A. Type 4 RTA is characterized by hyperkalemia and a mild normal-gap acidosis, commonly in diabetic kidney disease with hyporeninemic hypoaldosteronism. Hyperkalemia itself suppresses renal ammoniagenesis, contributing to acidosis. Urine can still be acidic, so a low urine pH does not exclude type 4 RTA.

62. C. Chronic metabolic acidosis in CKD contributes to muscle protein catabolism, bone buffering, and other adverse metabolic effects and may be associated with faster CKD progression. Alkali therapy is considered when clinically meaningful acidosis persists, while sodium load and volume status are monitored. The exact threshold and target should be individualized.

63. C. Anti-GBM disease can cause a pulmonary-renal syndrome and crescentic GN. Standard treatment combines plasma exchange to remove circulating antibody with immunosuppression to stop further production. Prognosis depends strongly on kidney function and the extent of crescentic injury at presentation.

64. A. Severe tumor lysis can produce life-threatening hyperkalemia, phosphate accumulation, hypocalcemia, urate nephropathy, and volume problems. Dialysis may be required when these complications are severe or refractory, especially with oliguria. Continuous modalities can be useful when phosphate and potassium rebound rapidly or the patient is unstable.

65. A. Labetalol and extended-release nifedipine are commonly used for hypertension in pregnancy, with methyldopa another established option. ACE inhibitors, ARBs, and direct renin inhibitors are contraindicated because fetal renin-angiotensin system blockade can cause serious fetal kidney injury and oligohydramnios.

66. B. Hyperkalemia management should first address reversible contributors such as diet, acidosis, constipation, medication interactions, and volume status. Diuretics or newer potassium binders can sometimes permit continuation of kidney-protective renin-angiotensin blockade. Immediate discontinuation may be necessary for severe or refractory hyperkalemia, but it is not always required for a manageable isolated elevation.

67. E. Chronic lithium exposure can cause a chronic tubulointerstitial nephropathy with distal and collecting-duct injury, nephrogenic diabetes insipidus, and characteristic small tubular cysts. Risk relates to cumulative exposure and episodes of toxicity. Kidney function and lithium concentration should be monitored during long-term therapy.

68. C. Loop diuretics inhibit NKCC2 in the thick ascending limb, producing potent natriuresis and reducing the medullary concentration gradient. They increase urinary calcium and magnesium excretion and can cause hypokalemic metabolic alkalosis. Their efficacy makes them central for significant volume overload in advanced CKD and heart failure.

69. A. PTLD ranges from polyclonal proliferations to aggressive lymphoma and is often EBV related, especially in high-risk seronegative recipients. Initial management commonly includes reduction of immunosuppression when feasible, with rituximab and chemotherapy used according to histology and severity. Tissue diagnosis is important.

70. D. High-risk chronic hyponatremia can overcorrect when the ADH stimulus disappears and a brisk water diuresis begins. Desmopressin can halt the aquaresis, and D5W can relower sodium when needed. Alcoholism and malnutrition increase the risk of osmotic demyelination, making strict correction limits especially important.

71. C. Dialysis initiation is based on symptoms and complications that are refractory to medical therapy, not a single creatinine or eGFR threshold. Uremic pericarditis, encephalopathy, refractory volume overload, severe acid-base or electrolyte problems, and progressive uremic symptoms are classic indications. Early dialysis in an otherwise asymptomatic patient has not shown benefit simply based on eGFR.

72. A. Hepatorenal syndrome-AKI is a functional renal failure caused by severe splanchnic vasodilation and renal vasoconstriction in advanced cirrhosis after other causes of AKI are excluded and volume status is addressed. Urine sediment is usually bland. Treatment includes albumin plus a vasoconstrictor strategy and evaluation for liver transplantation.

73. D. Granulomatosis with polyangiitis commonly involves the upper and lower respiratory tracts and kidneys and is often associated with PR3-ANCA. Renal biopsy shows pauci-immune necrotizing crescentic GN. Induction therapy uses glucocorticoids plus rituximab or cyclophosphamide, with regimen selection based on severity and patient factors.

74. D. Vomiting causes chloride depletion, volume contraction, and a chloride-responsive metabolic alkalosis. A low urine chloride supports this diagnosis. Repletion with chloride-containing isotonic fluid and potassium allows the kidney to excrete bicarbonate and correct the alkalosis.

75. A. The decision to start dialysis is clinical and incorporates symptoms, nutrition, volume control, electrolyte and acid-base status, and patient preferences. A low eGFR should prompt preparation but is not, by itself, an automatic indication in an asymptomatic stable patient. Advance planning helps avoid unplanned catheter-dependent dialysis.

76. A. Severe hypertension with acute pulmonary edema and AKI is a hypertensive emergency. Blood pressure is reduced in a controlled fashion with titratable IV agents, generally avoiding an excessive

initial fall that could compromise autoregulated organ perfusion. The specific drug depends on the involved organ system and comorbidities.

77. E. HRS-AKI reflects marked splanchnic vasodilation with reduced effective arterial volume and intense renal vasoconstriction. Albumin supports plasma volume while vasoconstrictors improve effective arterial pressure and renal perfusion. Terlipressin is used where appropriate, and norepinephrine is an ICU alternative.

78. B. Belatacept is contraindicated in EBV-seronegative or unknown-status recipients because of increased risk of PTLD, particularly involving the central nervous system. EBV serostatus must therefore be established before use. Other immunosuppressants also influence infection and malignancy risk but do not carry this same absolute EBV-serostatus restriction.

79. A. Microscopic polyangiitis is an ANCA-associated small-vessel vasculitis often associated with MPO-ANCA. It can cause pulmonary capillaritis and pauci-immune crescentic GN but lacks the granulomatous upper-airway lesions typical of GPA. Therapy parallels other severe ANCA-associated vasculitides.

80. A. When feasible and likely to mature, an autogenous AV fistula has lower long-term infection and thrombosis rates than catheters. Access choice should be individualized within an ESKD life-plan because not every patient benefits from a fistula-first approach. Central venous catheters are useful for urgent access but carry substantial infection and stenosis risks.

81. E. Primary aldosteronism produces sodium retention, hypertension, suppressed renin, and increased distal secretion of potassium and hydrogen, causing hypokalemic metabolic alkalosis. The elevated aldosterone distinguishes it from mimics such as Liddle syndrome or licorice effect, in which aldosterone is suppressed.

82. C. Marked hyperglycemia raises extracellular tonicity and draws water out of cells, lowering the measured serum sodium by dilution. This is hypertonic or translocational hyponatremia rather than true hypotonic hyponatremia. The sodium should rise as hyperglycemia is corrected.

83. D. Hexagonal urine crystals are characteristic of cystine. Cystinuria is an inherited defect in reabsorption of cystine and dibasic amino acids, leading to recurrent stones at a young age. Management emphasizes very high fluid intake, urine alkalinization, sodium restriction, and thiol drugs for refractory disease.

84. A. In acute decompensated heart failure, venous congestion and high renal interstitial pressure contribute to kidney dysfunction. A modest creatinine rise during effective decongestion may reflect hemodynamic change rather than structural injury. Treatment should balance perfusion, congestion, symptoms, and objective response rather than reacting to creatinine in isolation.

85. D. Uremia causes qualitative platelet dysfunction, including impaired adhesion and aggregation, so mucocutaneous bleeding can occur despite a normal platelet count. Dialysis may improve the defect, and

desmopressin can transiently enhance hemostasis in urgent situations. Anemia can also worsen uremic bleeding by reducing platelet interaction with the vessel wall.

86. B. Aortic dissection requires rapid reduction of impulse and shear stress. An IV beta blocker such as esmolol is used first to reduce heart rate and contractility; a vasodilator can then be added if needed. Vasodilation before beta blockade can provoke reflex tachycardia and increase aortic wall stress.

87. B. Class III and IV lupus nephritis are proliferative forms that can cause active urinary sediment, reduced GFR, hypertension, and significant proteinuria. They typically require induction immunosuppression followed by maintenance therapy. Kidney biopsy is important because clinical findings alone do not reliably define histologic class.

88. A. Post-transplant erythrocytosis can occur in patients with a well-functioning graft and persistent native-kidney erythropoietin signaling. ACE inhibitors and ARBs often lower hematocrit effectively. Other causes of erythrocytosis, including hypoxia and malignancy, should be excluded when appropriate.

89. E. Desmopressin can transiently improve uremic platelet function by increasing circulating von Willebrand factor and factor VIII and enhancing platelet adhesion. The effect begins quickly but is short-lived and tachyphylaxis can occur. Definitive management includes adequate dialysis and correction of contributing anemia.

90. D. Intra-abdominal hypertension can reduce renal perfusion pressure, increase renal venous pressure, and sharply reduce urine output. Abdominal compartment syndrome also impairs ventilation and hemodynamics. Measuring bladder pressure helps support the diagnosis, and decompression may be necessary in severe cases.

91. D. Thiazide and thiazide-like diuretics block NCC in the distal convoluted tubule. They reduce urinary calcium excretion but can cause hyponatremia, hypokalemia, metabolic alkalosis, and hyperuricemia. Their effect on urinary dilution helps explain the particular risk of hyponatremia.

92. A. Bilateral enlarged cystic kidneys, hepatic cysts, hypertension, and an affected first-degree relative are typical of ADPKD. PKD1 generally causes earlier progression than PKD2, although individual course varies widely. Extrarenal manifestations include intracranial aneurysms, valvular disease, and abdominal wall hernias.

93. B. Dialysis-related amyloidosis is caused by accumulation and tissue deposition of beta-2 microglobulin, historically after many years of dialysis. It can cause carpal tunnel syndrome, bone cysts, and destructive arthropathy. Modern high-flux dialysis and transplantation have reduced its incidence.

94. C. Hypokalemic periodic paralysis is caused by a shift of potassium into cells, often triggered by carbohydrate intake, rest after exercise, or adrenergic stimulation. Low urinary potassium and normal acid-base status support redistribution rather than total-body depletion. Therapy includes cautious potassium replacement and treatment of an underlying cause such as thyrotoxicosis when present.

95. B. A large increase in urine osmolality after desmopressin indicates that the kidney can respond to vasopressin, so the defect is deficient hormone secretion. Pituitary surgery is a classic cause of central diabetes insipidus. Nephrogenic diabetes insipidus shows little or no concentrating response to desmopressin.

96. B. Class V lupus nephritis is a membranous pattern with subepithelial immune deposits and commonly presents with nephrotic proteinuria. It can occur alone or with proliferative lesions. Treatment decisions depend on proteinuria severity, kidney function, complications, and whether a proliferative component is present.

97. A. Severe asymptomatic hypertension without acute target-organ injury is not a hypertensive emergency. Rapid IV lowering can cause ischemia and is generally unnecessary. The focus is confirmation, assessment of adherence and secondary factors, adjustment of oral therapy, and timely outpatient follow-up.

98. A. Oral sodium phosphate can cause acute phosphate nephropathy, especially in older patients, CKD, volume depletion, or those taking medications that alter renal perfusion. Calcium-phosphate crystals deposit in distal tubules and collecting ducts, and kidney recovery may be incomplete. Safer bowel preparations are preferred in susceptible patients.

99. C. Dialysis disequilibrium occurs when plasma urea and osmolality fall faster than brain osmoles, creating an osmotic gradient that favors cerebral edema. It is most likely during initial treatment of severe azotemia. Prevention uses gentler, shorter dialysis with slower solute removal in high-risk patients.

100. E. Primary FSGS can recur rapidly after transplantation, sometimes within hours to days, supporting a circulating permeability-factor mechanism. Sudden heavy proteinuria in a recipient whose native kidney failed from primary FSGS is a major clue. Plasma exchange and additional immunomodulatory strategies are commonly used.

101. D. Cloudy dialysate, abdominal pain, and a neutrophil-predominant peritoneal cell count are classic for PD-associated peritonitis. Empiric therapy should cover gram-positive and gram-negative organisms and is usually given intraperitoneally after cultures are obtained. Delayed treatment increases the risk of catheter loss and morbidity.

102. B. When hyperkalemia causes ECG changes, intravenous calcium is given promptly to stabilize myocardial membranes. Insulin with glucose and beta-agonists then shift potassium into cells, while diuretics, binders, or dialysis remove potassium from the body. Calcium does not lower the serum potassium concentration but reduces the immediate arrhythmic risk.

103. A. Fat malabsorption leaves fatty acids in the intestinal lumen, where they bind calcium and free oxalate for absorption. Increased oxalate delivery to the kidney can cause stones, nephrocalcinosis, and

oxalate nephropathy. Treatment includes hydration, dietary oxalate reduction, calcium with meals, and management of fat malabsorption.

104. C. IgA vasculitis is a systemic small-vessel immune-complex disease characterized by palpable purpura, arthralgia, gastrointestinal involvement, and IgA-mediated glomerulonephritis. The renal lesion resembles IgA nephropathy. Severity of kidney involvement determines prognosis and treatment intensity.

105. E. Masked hypertension refers to normal clinic readings with elevated out-of-office blood pressure. It can be detected by home or ambulatory monitoring and is associated with increased cardiovascular risk. White-coat hypertension is the reverse pattern.

106. A. IgA nephropathy frequently recurs histologically after kidney transplantation and can eventually affect graft function in some patients. Recurrence is generally later and more gradual than the dramatic early recurrence seen with primary FSGS. Management emphasizes blood-pressure and proteinuria control and individualized immunosuppression decisions.

107. D. Recurrence with the same organism soon after treatment suggests relapse rather than a new unrelated infection and raises concern for persistent catheter biofilm or another un-eradicated source. Repeated relapses may require catheter removal depending on organism and clinical course. Organism identification and timing are therefore important in PD peritonitis management.

108. D. Lithium commonly causes nephrogenic diabetes insipidus by entering principal cells through ENaC and impairing aquaporin-mediated water reabsorption. Amiloride blocks ENaC and therefore decreases lithium uptake into these cells. Thiazides may reduce urine volume, but amiloride most directly addresses lithium entry.

109. B. Struvite stones are composed of magnesium ammonium phosphate and form in alkaline urine infected with urease-producing organisms such as Proteus. They can grow rapidly into staghorn calculi. Definitive management generally requires complete stone removal plus control of infection.

110. B. Myeloma cast nephropathy occurs when large quantities of filtered monoclonal free light chains interact with Tamm-Horsfall protein and form obstructing, inflammatory casts. It often presents with severe AKI and a discrepancy between total urine protein and albumin. Rapid control of the plasma-cell clone and supportive kidney care are essential.

111. D. Liddle syndrome is caused by constitutive ENaC activation, so amiloride or triamterene directly blocks the abnormal sodium entry pathway. Aldosterone is suppressed, making spironolactone less mechanistically effective. Sodium restriction also helps reduce volume expansion.

112. A. High peritoneal transport status can lead to rapid glucose absorption and loss of the osmotic gradient during long dwells, reducing ultrafiltration. Prescription changes may include shorter dwells or use of icodextrin for long dwells. Persistent ultrafiltration failure requires evaluation for mechanical and membrane-related causes.

113. C. Hepatitis C can drive mixed cryoglobulinemia, producing palpable purpura, arthralgia, neuropathy, low complement—especially C4—and MPGN. Management includes direct-acting antiviral therapy and, for severe organ-threatening disease, selected immunosuppressive or plasma-exchange strategies. The systemic findings support cryoglobulinemic vasculitis.

114. C. Pseudohyperkalemia can occur with marked thrombocytosis because platelets release potassium during clotting, raising serum but not plasma potassium. The normal plasma value and absence of ECG changes support an artifact rather than true hyperkalemia. Similar artifacts can occur with severe leukocytosis or specimen hemolysis.

115. D. White-coat hypertension is present when office blood pressure is elevated but out-of-office measurements are normal. Ambulatory or validated home monitoring helps distinguish it from sustained hypertension and can prevent unnecessary medication escalation. Patients still require periodic follow-up because some progress to sustained hypertension.

116. B. Excessive ultrafiltration relative to plasma refill is a common cause of intradialytic hypotension. Reassessing target weight, limiting interdialytic sodium and fluid gain, and reducing the ultrafiltration rate by extending or modifying treatment are key strategies. Cool dialysate and selected medications may also help in recurrent cases.

117. E. Delayed graft function is commonly due to ischemia-reperfusion acute tubular injury, especially in deceased-donor kidneys with prolonged ischemic time. Dialysis may be needed temporarily. Biopsy is often required when graft dysfunction persists because rejection and other surgical complications can present similarly.

118. B. High-dose methotrexate can precipitate in tubules and cause AKI, especially with acidic urine or volume depletion. Prevention includes vigorous hydration and urine alkalinization, with leucovorin rescue. Glucarpidase rapidly hydrolyzes methotrexate and is used for severe toxicity with delayed clearance due to kidney dysfunction.

119. C. Routine universal aneurysm screening in ADPKD is not necessary for every patient. Screening is particularly considered with a personal or strong family history of intracranial aneurysm or subarachnoid hemorrhage, certain high-risk occupations, or before selected major procedures depending on circumstances. Magnetic resonance angiography is commonly used.

120. A. Dialysis dry weight is a clinical estimate rather than a single laboratory value. Serial examination, blood pressure, symptoms, interdialytic weight change, and adjunctive measures such as lung ultrasound or bioimpedance can help refine volume assessment. Persistent hypertension and edema may indicate that target weight remains above true euvolemia.

121. E. C3 glomerulopathy is driven by alternative complement pathway dysregulation from acquired or genetic abnormalities. Biopsy shows dominant C3 staining with little immunoglobulin and can have an

MPGN pattern. Evaluation includes complement studies and assessment for monoclonal gammopathy in appropriate patients.

122. D. In hypernatremia with hemodynamic instability, resuscitation of the circulation takes priority. Isotonic crystalloid is used until perfusion is restored; then the free-water deficit is corrected gradually with enteral water or hypotonic fluid. Treating shock with hypotonic water alone is unsafe and ineffective.

123. A. Acyclovir can crystallize in renal tubules, particularly with rapid IV administration, high doses, or inadequate hydration. Prevention includes dose adjustment, hydration, and appropriate infusion rates. Withdrawal and supportive care usually lead to improvement when recognized early.

124. E. Post-transplant ureteral obstruction can cause graft dysfunction and hydronephrosis and may result from edema, technical issues, stricture, clot, stone, or external compression. Because the allograft may be denervated, pain can be absent. Prompt imaging and decompression protect graft function.

125. B. Uremic pruritus is common in advanced kidney failure and may persist despite apparently adequate dialysis. Management includes optimizing dialysis adequacy, correcting contributing mineral abnormalities, aggressive emollient use, and selected pharmacologic therapies for persistent symptoms. A primary dermatologic or systemic cause should also be excluded when the presentation is atypical.

126. B. Apparent resistant hypertension may reflect pseudoresistance from nonadherence, inaccurate measurement, white-coat effect, or suboptimal regimens. Confirming adherence and blood-pressure technique is essential before labeling true resistant hypertension. A nonjudgmental approach improves the chance of identifying barriers and finding workable solutions.

127. A. With bicarbonate 10 mEq/L, Winter's formula predicts a $PaCO_2$ near 23 ± 2 mm Hg. A measured $PaCO_2$ of 38 is substantially higher, indicating inadequate ventilatory compensation and a superimposed respiratory acidosis. This can signal fatigue, CNS depression, or pulmonary disease and is clinically important in DKA.

128. E. AL amyloidosis results from a plasma-cell clone producing amyloidogenic light chains that deposit as Congo-red-positive fibrils. Renal disease commonly causes heavy proteinuria, and systemic involvement can include cardiomyopathy and neuropathy. Identifying and treating the underlying plasma-cell disorder is essential.

129. B. Oral sodium phosphate preparations can cause acute phosphate nephropathy and major electrolyte disturbances, especially in older patients, those with CKD, volume depletion, or medications affecting renal perfusion. Polyethylene glycol-based preparations are generally preferred in patients at kidney risk. Adequate hydration and medication review remain important.

130. E. Tenofovir disoproxil fumarate can injure proximal tubular cells, causing Fanconi features such as phosphate wasting, glycosuria despite normal glucose, aminoaciduria, and bicarbonate loss. Risk is

affected by kidney function and interacting drugs. Tenofovir alafenamide generally has lower systemic renal exposure.

131. A. Acetazolamide inhibits carbonic anhydrase in the proximal tubule, impairing bicarbonate reabsorption and causing bicarbonaturia and a hyperchloremic metabolic acidosis. It also alkalinizes urine and can promote hypokalemia. Uses include glaucoma, altitude illness, and selected cases of metabolic alkalosis.

132. A. Ethylene glycol is metabolized to glycolate and oxalate, producing high-gap acidosis, hypocalcemia, and calcium oxalate crystal deposition in the kidney. Methanol more characteristically causes visual toxicity from formate. Treatment includes inhibition of alcohol dehydrogenase and, in severe cases, hemodialysis.

133. E. The brain adapts to chronic hypernatremia by generating intracellular osmoles that restore cell volume. If extracellular tonicity falls too quickly, water shifts into adapted brain cells and can cause cerebral edema. This is distinct from osmotic demyelination, which is classically associated with overly rapid correction of chronic hyponatremia.

134. A. Nephrogenic systemic fibrosis risk is strongly related to agent stability and severe kidney dysfunction. Modern lower-risk group II agents have a very low NSF risk and can be used when the diagnostic benefit warrants it, with appropriate institutional practice. Dialysis should not be started solely to clear gadolinium in a patient who otherwise does not need kidney replacement therapy.

135. E. Lymphoceles arise from disrupted lymphatics and often appear weeks after transplant. They may be asymptomatic or compress the ureter, bladder, or iliac veins. A urinoma generally has a much higher fluid creatinine than serum, helping distinguish the two.

136. D. Mineralocorticoid receptor antagonists are highly effective for many patients with resistant hypertension, even without proven primary aldosteronism. Kidney function and potassium must be monitored because hyperkalemia risk rises in CKD. The background regimen should include an effective diuretic and long-acting agents at tolerated doses.

137. E. Salicylates directly stimulate the respiratory center, causing respiratory alkalosis, while also producing organic acid accumulation and a high-anion-gap metabolic acidosis. A near-normal or alkalemic pH can therefore hide severe metabolic acidosis. Recognizing the mixed disorder is a classic clue to salicylate poisoning.

138. C. Aminoglycosides accumulate in proximal tubular cells and can cause nonoliguric acute tubular injury after several days of exposure. Risk increases with higher exposure, prolonged courses, older age, and other nephrotoxins. Dosing strategies and drug-level monitoring can reduce toxicity.

139. D. Fibrillary GN features nonbranching fibrils that are usually Congo-red negative and larger than amyloid fibrils. DNAJB9 is a useful diagnostic marker. It can present with proteinuria, hematuria, hypertension, and reduced kidney function.

140. B. Contrast-associated AKI risk rises with lower kidney function and concurrent hemodynamic risk, but necessary procedures should not be withheld reflexively. Minimizing contrast, avoiding other nephrotoxins, and maintaining appropriate intravascular volume are key. Prophylactic dialysis after contrast has not been shown to prevent AKI and is not routinely indicated.

141. B. Tolvaptan blocks V2 receptors and can slow progression in appropriately selected adults with ADPKD at risk for rapid progression. Aquaresis is expected, and liver tests require monitoring because clinically important hepatotoxicity can occur. Patient selection balances potential benefit, age, kidney function, progression risk, and tolerability.

142. C. Renal vein thrombosis is an early surgical vascular complication that can present with sudden graft dysfunction, swelling, pain, hematuria, and abnormal Doppler flow. It is a surgical emergency and often results in graft loss. Renal artery thrombosis is another catastrophic early vascular complication.

143. D. Protein-energy wasting is an important complication of advanced CKD. Dietary protein may be moderated in selected nondialysis patients, but adequate energy intake and preservation of nutritional status are essential. Overly restrictive diets can worsen frailty and outcomes, so individualized renal dietitian guidance is valuable.

144. E. Severe metformin-associated lactic acidosis with profound acidemia, shock, or kidney failure may require hemodialysis. Metformin is small and not highly protein bound, so it is dialyzable, and dialysis also corrects acidosis. Ongoing supportive care addresses shock and the precipitating illness.

145. D. Light-chain deposition disease is a monoclonal gammopathy of renal significance characterized by nonfibrillar monoclonal deposits, often along glomerular and tubular basement membranes. It can produce nodular sclerosis and proteinuria. Therapy targets the pathogenic plasma-cell or B-cell clone.

146. D. As GFR declines, higher-dose loop diuretics are often needed for adequate natriuresis, and carefully selected combination therapy with a thiazide-like agent can overcome distal nephron adaptation. Volume excess is a common driver of resistant hypertension in CKD. Electrolytes and kidney function require close monitoring during intensified diuresis.

147. A. A reset osmostat behaves like SIADH around a lower set point but retains the ability to dilute urine when osmolality falls sufficiently below that new threshold. Sodium tends to remain mildly and stably low rather than falling progressively. This distinction is useful because aggressive attempts to normalize sodium may be ineffective and unnecessary.

148. C. Phosphate binders act within the gut and should be taken with meals to reduce intestinal phosphate absorption. Choice of calcium-containing versus noncalcium binders depends on calcium balance, vascular calcification risk, pill burden, cost, and other patient factors. Dietary phosphate source and dialysis clearance also matter.

149. B. Albumin contributes substantially to the normal anion gap. When albumin is low, the baseline anion gap falls, so a seemingly normal measured value can conceal accumulation of unmeasured acids. Correcting for hypoalbuminemia improves detection of high-gap metabolic acidosis.

150. E. Lithium is highly dialyzable because it is small and minimally protein bound. Severe neurologic manifestations, markedly elevated concentrations, and impaired renal clearance are major considerations for hemodialysis. Rebound can occur after intermittent dialysis because lithium redistributes from tissues, so serial levels are required.

151. C. Tolvaptan antagonizes V2 receptors and reduces aquaporin-2 insertion, producing aquaresis. It can raise serum sodium and is also used in selected ADPKD patients to slow disease progression. Sodium and volume status require monitoring, and liver toxicity is an important concern during chronic ADPKD therapy.

152. E. In a patient with hypercalcemia or substantial calcium loading, a noncalcium phosphate binder such as sevelamer or lanthanum can reduce phosphate absorption without adding more calcium. Calcium balance and vascular calcification risk are important in CKD-MBD management. Therapy should be based on serial trends rather than a single isolated value.

153. A. Transplant renal artery stenosis can cause difficult hypertension, graft dysfunction, volume overload, and a bruit. It often occurs near the anastomosis or in areas of arterial injury. Doppler ultrasound is a common screening test, with angiographic studies used when intervention is contemplated.

154. A. Alport syndrome is caused by defects in type IV collagen and can produce hematuria, progressive CKD, sensorineural hearing loss, and ocular findings. X-linked COL4A5 disease is most common, but autosomal forms occur. Family history and genetic testing help define inheritance and prognosis.

155. D. Liddle syndrome is a gain-of-function ENaC disorder that causes sodium retention, hypertension, hypokalemia, and metabolic alkalosis with suppressed renin and aldosterone. It is treated with ENaC blockers such as amiloride or triamterene rather than spironolactone, because aldosterone itself is not the driver.

156. B. Magnesium deficiency promotes renal potassium wasting, partly by increasing ROMK-mediated potassium secretion. As a result, potassium replacement may be ineffective until magnesium is also repleted. This relationship is especially important in patients receiving diuretics, aminoglycosides, or platinum chemotherapy.

157. D. Calcimimetics increase the sensitivity of the parathyroid calcium-sensing receptor to extracellular calcium and lower PTH. They are useful in dialysis patients with significant secondary hyperparathyroidism, particularly when hypercalcemia limits vitamin D analogs. Hypocalcemia and gastrointestinal adverse effects can occur.

158. C. Severe salicylate poisoning with CNS manifestations, pulmonary edema, severe acid-base disturbance, kidney failure, or worsening levels is an indication for urgent hemodialysis. Alkalinization of serum and urine helps reduce tissue penetration and increase renal elimination before dialysis. Intubation is hazardous if post-intubation ventilation fails to match the patient's high minute ventilation.

159. B. Exercise-associated hyponatremia usually reflects excessive hypotonic fluid intake plus impaired water excretion because vasopressin remains elevated from pain, nausea, stress, or exertion. Sweat losses may contribute, but water excess is central. Symptomatic cases require prompt assessment and, when severe, hypertonic saline.

160. A. Active loop or thiazide diuretic use increases urinary chloride, so urine chloride may remain high even though the patient is volume depleted and alkalotic. Vomiting usually produces a chloride-responsive alkalosis with low urine chloride. Timing matters because urine chloride can fall after the diuretic effect wears off.

161. C. Tertiary hyperparathyroidism develops after prolonged secondary hyperparathyroid stimulation, with nodular hyperplasia and autonomous PTH secretion. Hypercalcemia in the setting of very high PTH is a key clue. Refractory severe disease may require parathyroidectomy.

162. C. Thin basement membrane nephropathy commonly presents with persistent microscopic hematuria and preserved kidney function. It is often related to heterozygous COL4A3 or COL4A4 variants, so the spectrum overlaps genetically with Alport disease. Significant proteinuria or declining GFR warrants closer evaluation because not all collagen-IV disease is benign.

163. D. Mycophenolate is strongly teratogenic and must be stopped with an appropriate washout period before conception, using a pregnancy-compatible alternative such as azathioprine when needed. Selected calcineurin inhibitors and glucocorticoids can be used with monitoring. Pregnancy should be planned when graft function and immunologic status are stable.

164. C. Fomepizole blocks alcohol dehydrogenase and prevents conversion of ethylene glycol to nephrotoxic and acid-generating metabolites. Severe poisoning may also require hemodialysis to remove parent alcohol and metabolites and correct acidosis. Calcium oxalate crystalluria and hypocalcemia are classic clues.

165. A. Licorice inhibits 11-beta-hydroxysteroid dehydrogenase type 2, allowing cortisol to activate mineralocorticoid receptors. The resulting apparent mineralocorticoid excess suppresses renin and aldosterone. Removing the exposure is central, with supportive management of blood pressure and potassium.

166. A. Severe hyperkalemia with ECG abnormalities requires immediate membrane stabilization and intracellular shifting, but these measures do not remove much potassium from the body. In dialysis-dependent kidney failure, urgent dialysis is the definitive way to remove potassium when hyperkalemia is severe or refractory. Ongoing sources of potassium should also be addressed.

167. E. Trimethoprim can raise serum creatinine by inhibiting proximal tubular creatinine secretion without a true fall in GFR. It also reduces distal potassium secretion through an amiloride-like ENaC effect and can cause clinically important hyperkalemia, especially with CKD or renin-angiotensin blockade.

168. E. In chronic respiratory acidosis, the kidney increases hydrogen secretion, ammoniagenesis, and bicarbonate retention over days. This raises serum bicarbonate more than occurs in acute respiratory acidosis and partially restores pH. Renal compensation does not completely normalize pH while the primary hypercapnia persists.

169. D. Severe ADAMTS13 deficiency strongly supports TTP, an emergency requiring plasma exchange plus immunosuppressive therapy and often caplacizumab. Kidney injury can occur but severe renal failure may be more prominent in other TMAs. Treatment should not wait for the ADAMTS13 result when clinical suspicion is high.

170. C. Refractory volume overload with pulmonary edema is a classic indication for kidney replacement therapy when diuretics cannot achieve adequate decongestion. Ultrafiltration removes isotonic fluid and can rapidly improve pulmonary congestion. The overall prescription should account for hemodynamic stability and the rate of fluid removal.

171. C. Methanol is metabolized to formic acid, which causes high-gap acidosis and visual toxicity. Fomepizole is used to block alcohol dehydrogenase, and severe cases require hemodialysis. Folate therapy enhances formate metabolism.

172. D. Live attenuated vaccines are generally contraindicated after solid-organ transplantation because immunosuppression can permit uncontrolled vaccine-strain replication. Inactivated and recombinant vaccines are typically safe, although immune responses may be reduced. Whenever possible, vaccination status is optimized before transplantation.

173. C. The key discriminator is true volume depletion with renal sodium wasting after intracranial disease. Cerebral salt wasting and SIADH can share high urine sodium and concentrated urine, but SIADH is generally euvolemic. Treatment therefore differs: salt wasting requires volume and sodium replacement rather than simple fluid restriction.

174. B. Medullary sponge kidney is characterized by dilation of medullary collecting ducts and is associated with nephrocalcinosis, hematuria, stones, and sometimes distal acidification defects. Kidney function is often preserved. Management focuses on stone prevention and treatment of urinary infection when present.

175. A. Dialysis-related cramps often occur when ultrafiltration exceeds vascular refill, producing relative intravascular depletion and tissue hypoperfusion. Reducing the ultrafiltration rate, reassessing target weight, and preventing excessive interdialytic weight gain may help. Other causes of cramps should be considered when symptoms are atypical or persistent.

176. C. Coarctation causes hypertension proximal to the narrowing and reduced or delayed femoral pulses distal to it. An arm-leg pressure gradient and radiofemoral delay are classic findings. Adults may also have collateral vessel findings or associated bicuspid aortic valve.

177. A. Continuous kidney replacement therapy provides gradual solute and volume removal and is often better tolerated in patients with severe hemodynamic instability or cerebral edema risk. Intermittent dialysis remains appropriate for many stable patients and may remove certain toxins more rapidly. Modality is selected based on clinical goals and resources.

178. A. Shiga toxin-associated HUS is a thrombotic microangiopathy characterized by AKI, thrombocytopenia, and microangiopathic hemolytic anemia after enteric infection. Supportive care is central. Routine antibiotics and antimotility agents are generally avoided during suspected Shiga-toxin diarrhea because of concern for toxin release or prolonged exposure.

179. E. Access-induced distal ischemia, often called steal syndrome, occurs when fistula flow compromises distal hand perfusion. Findings include pain, coolness, neurologic symptoms, tissue loss, or reduced pulses. Severe cases require prompt vascular evaluation because limb-threatening ischemia can occur.

180. B. Tacrolimus is metabolized through CYP3A pathways and transported by P-glycoprotein. Macrolides such as clarithromycin can inhibit metabolism and markedly increase tacrolimus exposure, causing neurotoxicity, hyperkalemia, hypertension, and nephrotoxicity. Medication interaction review is essential in transplant care.

181. A. Acute respiratory alkalosis produces a modest immediate decrease in bicarbonate through buffering; renal bicarbonate loss develops over the next several days if hypocapnia persists. The distinction between acute and chronic compensation helps identify mixed disorders. Renal compensation is not instantaneous.

182. D. PTH-related peptide mimics many renal and skeletal actions of PTH, causing hypercalcemia and phosphaturia while suppressing endogenous intact PTH. Squamous carcinomas are classic causes. Vitamin D-mediated hypercalcemia more often raises phosphate rather than lowering it.

183. C. Oral urea increases urinary osmole excretion and can enhance water clearance in chronic SIADH. Desmopressin would worsen water retention, and isotonic saline may fail or even aggravate hyponatremia when urine remains highly concentrated. Therapy should still address the underlying cause whenever possible.

184. A. Prior central venous catheters can cause central venous stenosis. High flow through an ipsilateral AV access then produces venous hypertension, manifested by arm, breast, neck, or facial swelling and prominent collateral veins. Imaging and endovascular management are often required.

185. B. Citrate chelates ionized calcium in the circuit and is normally metabolized, generating bicarbonate equivalents. When citrate accumulates—such as in severe shock or liver dysfunction—the

total-to-ionized calcium ratio rises and calcium requirements increase. Acid-base disturbances can vary depending on citrate delivery and metabolism.

186. C. Posterior reversible encephalopathy syndrome presents with seizures, encephalopathy, headache, and visual symptoms, often in the setting of severe hypertension, eclampsia, cytotoxic drugs, or kidney disease. Imaging typically shows posterior-predominant vasogenic edema. Management includes control of the precipitating cause and careful blood-pressure reduction.

187. E. Complement-mediated aHUS results from dysregulated alternative complement activation and can respond dramatically to terminal complement blockade such as eculizumab or ravulizumab. Vaccination and infection precautions are critical because terminal complement inhibition increases meningococcal risk. Secondary TMAs and TTP must be distinguished from complement-mediated disease.

188. E. An AV graft can provide durable access when native vessels are unsuitable for a fistula or when timely access is needed. Grafts have higher infection and thrombosis rates than mature fistulas but generally lower infectious risk than long-term central venous catheters. Access planning should be individualized to life expectancy, anatomy, and treatment goals.

189. A. Sirolimus and everolimus can impair wound healing and are associated with mouth ulcers, dyslipidemia, cytopenias, and proteinuria among other adverse effects. Timing around surgery and baseline proteinuria therefore matter when choosing an mTOR inhibitor. Immunosuppression is individualized to immunologic risk and toxicity profile.

190. E. No single creatinine or BUN value determines the need for kidney replacement therapy in AKI. Indications include refractory hyperkalemia, acidosis, volume overload, selected toxin removal, and uremic complications. Clinical trajectory, urine output, catabolic state, and expected recovery also inform timing.

191. A. Calcineurin inhibitors cause dose-related renal vasoconstriction that can acutely reduce GFR, along with hypertension and hyperkalemia. Chronic exposure can produce arteriolar hyalinosis and tubulointerstitial fibrosis. Drug interactions that increase tacrolimus or cyclosporine levels can precipitate toxicity.

192. C. Glycyrrhetinic acid in licorice inhibits 11-beta-hydroxysteroid dehydrogenase type 2, which normally protects the mineralocorticoid receptor from cortisol. Cortisol then acts as a mineralocorticoid, producing hypertension, hypokalemia, and metabolic alkalosis. Both renin and aldosterone are suppressed.

193. C. Delivered dialysis dose can be reduced by access recirculation, low blood flow, shortened treatment, dialyzer problems, or prescription mismatch. A strong thrill does not exclude stenosis or recirculation. Evaluation includes review of machine parameters, access function, and adequacy metrics such as Kt/V or urea reduction ratio.

194. E. Cholesterol embolization can follow angiography or anticoagulation in patients with severe atherosclerosis. Findings include AKI, livedo or blue toes with preserved pulses, eosinophilia, and sometimes hypocomplementemia. Kidney biopsy may show cholesterol clefts in small arteries and arterioles.

195. E. Malignant hypertension can cause severe arteriolar endothelial injury, fibrinoid necrosis, and hyperplastic arteriolosclerosis with a thrombotic microangiopathy picture. Microangiopathic hemolysis and thrombocytopenia can occur. Blood-pressure control is essential, while alternative TMAs should be considered if the presentation is atypical or fails to improve.

196. D. In SIADH, vasopressin activity prevents maximal urinary dilution, so urine osmolality is usually above about 100 mOsm/kg. A urine osmolality near 55 mOsm/kg indicates appropriate suppression of ADH and strongly supports excess water intake. Other findings listed can occur in SIADH and are not exclusionary.

197. D. After membrane stabilization with calcium, insulin rapidly stimulates cellular potassium uptake and lowers serum potassium within minutes. Nebulized beta-2 agonists can provide additional shifting. These therapies are temporary; potassium must ultimately be removed from the body through urine, gastrointestinal binders, or dialysis.

198. E. For selected patients, especially those with high comorbidity or strong preferences against dialysis, comprehensive conservative kidney management is a legitimate active pathway. It includes treatment of symptoms and complications, advance care planning, and support for patients and families. Shared decision-making is central.

199. E. Many causes of allograft dysfunction present with nonspecific creatinine elevation. When hemodynamic, obstructive, and obvious drug-level issues do not explain the change, biopsy is often essential to distinguish rejection, BK nephropathy, recurrent disease, and chronic injury. Treatment differs markedly among these entities.

200. B. Nephronophthisis is an inherited ciliopathy that causes chronic tubulointerstitial nephritis, concentrating defects, salt wasting, and progressive kidney failure, often in childhood or adolescence. Kidneys may be small or normal sized with corticomedullary cysts. Extrarenal findings can occur depending on the genetic syndrome.

201. C. Hypomagnesemia increases renal potassium loss, in part by disinhibiting potassium secretion through ROMK channels. Potassium can remain refractory to replacement until magnesium is corrected. Both deficits should therefore be treated when clinically significant.

202. D. CKD progression is assessed by repeated measurements over time, not a single value. Both eGFR trajectory and albuminuria provide prognostic information, and abrupt changes should prompt evaluation for superimposed AKI, medication effects, obstruction, or other reversible causes. Trends also guide timing of referral and kidney replacement planning.

203. E. Acute renal infarction should be considered with sudden flank pain, hematuria, and elevated LDH in a patient with an embolic source such as atrial fibrillation. Contrast-enhanced imaging commonly shows wedge-shaped perfusion defects. Early diagnosis matters because anticoagulation or selected reperfusion strategies may be considered.

204. B. Severe acidemia can impair cardiovascular function and may become an indication for kidney replacement therapy when refractory. Bicarbonate may be useful in selected patients, especially when bicarbonate loss or severe acidemia with AKI is present, but it adds sodium and CO2 and does not correct ongoing causes. Therapy should address the underlying shock or toxin as well.

205. D. Tumor lysis releases potassium and phosphate and increases uric acid production from nucleic acids. Hyperphosphatemia can lower calcium and promote calcium-phosphate deposition, while uric acid and calcium phosphate contribute to AKI. Prevention and treatment emphasize hydration, urate-lowering therapy, electrolyte management, and dialysis when indicated.

206. E. ACE inhibitors lower efferent arteriolar tone. In bilateral severe renal artery stenosis, GFR may depend heavily on angiotensin II-mediated efferent constriction, so blockade can cause a substantial fall in filtration. A large creatinine rise should prompt assessment for renovascular disease, volume depletion, or NSAID use.

207. E. Advanced CKD warrants nephrology involvement for diagnostic review, slowing progression, complication management, medication adjustment, and timely education about transplant, dialysis, and conservative pathways. Preparation should be risk based rather than triggered only by an emergency. Avoiding unnecessary central venous access helps preserve future dialysis options.

208. B. Transplant glomerulopathy is a chronic microvascular injury pattern strongly associated with chronic antibody-mediated rejection, particularly when donor-specific antibodies are present. It can cause proteinuria, hypertension, and progressive graft dysfunction. Prevention and early control of alloimmune injury are important because established chronic lesions are difficult to reverse.

209. C. The standard water-deficit estimate uses total-body water multiplied by the fractional increase of measured sodium above a reference sodium. Total-body water is estimated from body weight with adjustment for age and sex. The calculation is only an estimate and must be paired with serial sodium measurements and ongoing-loss assessment.

210. D. Loop diuretics are useful for treating volume overload in AKI when there is residual diuretic responsiveness. They do not improve intrinsic tubular recovery or survival simply by increasing urine volume. Refractory pulmonary edema remains an indication for kidney replacement therapy.

211. E. Declining kidney function reduces elimination of many medications and active metabolites, increasing risks such as neurotoxicity, hypoglycemia, bleeding, and electrolyte abnormalities. Dose adjustments should use appropriate kidney-function estimates and consider dialysis clearance when relevant. Medication reconciliation also identifies nephrotoxins and harmful combinations.

212. A. Endocarditis can cause an infection-related immune-complex GN with active urine sediment and low complement. Treating the infection is essential. ANCA may occasionally be positive in endocarditis, so clinical context and biopsy can prevent inappropriate immunosuppression for presumed primary vasculitis.

213. E. Lithium enters principal cells through ENaC and disrupts vasopressin signaling and aquaporin-2 expression. Amiloride blocks ENaC and can reduce lithium uptake and polyuria. Thiazides can also reduce urine volume but work through a different hemodynamic mechanism and require careful monitoring of lithium levels.

214. A. Diarrheal fluid contains bicarbonate and potassium, so sustained losses cause a hyperchloremic normal-gap metabolic acidosis and potassium depletion. The kidney should increase ammonium excretion appropriately, often making the urine anion gap negative. This contrasts with renal tubular acidosis, in which renal acid excretion is impaired.

215. C. Technique matters: the patient should rest, the arm should be supported at heart level, and an appropriately sized cuff should be used. Poor technique can create pseudoresistant hypertension and lead to unnecessary medication escalation. Repeated standardized readings or out-of-office monitoring improve diagnostic accuracy.

216. D. Renal prostaglandins help preserve afferent arteriolar dilation when renal perfusion is threatened. NSAIDs inhibit prostaglandin synthesis and can reduce GFR, promote sodium retention, worsen edema and hypertension, and precipitate AKI. Risk is higher in CKD, heart failure, cirrhosis, or volume depletion.

217. A. A distended bladder in an anuric patient suggests lower urinary tract obstruction, a rapidly reversible cause of postrenal AKI. Catheterization both relieves obstruction and provides diagnostic information. After decompression, patients should be monitored for postobstructive diuresis and electrolyte losses.

218. B. ABO-incompatible kidney transplantation is feasible at experienced centers using protocols that reduce or control anti-blood-group antibodies. It carries added complexity and immunologic risk compared with compatible transplantation. Paired kidney exchange may provide another option for incompatible living-donor pairs.

219. D. Hepatitis B can cause secondary membranous nephropathy through immune-complex mechanisms. Management prioritizes effective antiviral therapy and careful consideration of immunosuppression, which can reactivate or worsen viral infection. The cause of membranous nephropathy therefore materially changes treatment.

220. A. The so-called triple-whammy interaction combines diuretic-associated volume depletion, NSAID-mediated loss of afferent prostaglandin dilation, and ACE inhibitor or ARB-mediated efferent

dilation. Together these changes can sharply reduce intraglomerular pressure and GFR. Medication review is therefore critical in patients with CKD or intercurrent illness.

221. D. A urine osmolality of 650 mOsm/kg shows that the kidney can concentrate urine, arguing against diabetes insipidus. Marked glucosuria creates an osmotic diuresis that carries water into the urine and can produce hypernatremia if intake does not keep pace. The treatment targets both the hyperglycemia and the water deficit.

222. D. Severe hypokalemia alters myocardial repolarization and can produce U waves, QT-appearing prolongation, ectopy, and life-threatening ventricular arrhythmias. Risk is greater with heart disease, digoxin, or concomitant hypomagnesemia. Prompt but controlled potassium replacement is indicated.

223. B. After relief of significant obstruction, accumulated salt and water and impaired concentrating ability can produce a brisk postobstructive diuresis. Excessive losses can cause hypovolemia, hypokalemia, hypernatremia, or other electrolyte abnormalities. Fluid replacement should be guided by output and clinical status rather than matching every milliliter automatically.

224. A. Sodium retention contributes to hypertension and edema in CKD. Dietary sodium reduction improves volume control and can enhance the antiproteinuric effect of renin-angiotensin system blockers. The specific target should be individualized to nutritional status and comorbidities.

225. B. Medication and substance review is a core part of resistant-hypertension evaluation. NSAIDs, sympathomimetics, some hormonal therapies, stimulants, calcineurin inhibitors, erythropoiesis-stimulating agents, and others can raise blood pressure. Removing or replacing contributors may substantially improve control.

226. A. Solid-organ transplant recipients have a markedly increased risk of cutaneous squamous-cell carcinoma because of chronic immunosuppression and UV exposure. Prevention includes sun protection and regular skin surveillance. In patients with recurrent cancers, immunosuppression minimization or regimen modification may be considered while balancing rejection risk.

227. B. Autosomal dominant tubulointerstitial kidney disease produces progressive CKD with bland sediment and relatively little proteinuria. UMOD-associated disease often features hyperuricemia and gout. The absence of enlarged polycystic kidneys helps distinguish it from ADPKD.

228. E. Nephrotic edema reflects a combination of low plasma oncotic pressure and renal sodium retention, with the relative contribution varying among patients. Some are intravascularly underfilled, whereas others are primarily overfilled from intrinsic renal sodium retention. Diuretic therapy therefore must account for hemodynamics and kidney function.

229. D. The kidney converts 25-hydroxyvitamin D to active 1,25-dihydroxyvitamin D through 1-alpha hydroxylase. CKD reduces this capacity, and elevated FGF23 further suppresses calcitriol production. Lower calcitriol contributes to hypocalcemic signaling and secondary hyperparathyroidism.

230. B. Thiazide-type diuretics enhance distal calcium reabsorption and reduce urinary calcium excretion, especially when dietary sodium intake is limited. Loop diuretics increase calciuria. Acetazolamide can increase calcium phosphate stone risk by alkalinizing urine and lowering citrate.

231. B. Renal cortical necrosis is a rare but severe ischemic injury associated with obstetric catastrophes, septic shock, and disseminated intravascular coagulation. It often causes prolonged anuria and incomplete recovery because much of the cortex is irreversibly damaged. This differs from typical acute tubular injury, which has greater recovery potential.

232. D. Sevelamer is a noncalcium phosphate binder that binds phosphate in the gut when taken with food. It is useful when phosphate remains elevated and additional calcium loading is undesirable. Because the action is gastrointestinal, timing with meals is important.

233. C. Patients with CKD can develop superimposed reversible causes of kidney function decline. Bilateral hydronephrosis should prompt urgent evaluation for obstruction and decompression when indicated. Labeling every creatinine rise as chronic progression risks missing a treatable postrenal process.

234. E. V2-receptor antagonists block vasopressin-mediated water reabsorption in collecting ducts and produce aquaresis. They can raise serum sodium quickly, so close monitoring is required. The other agents primarily alter sodium handling or vascular tone rather than selectively increasing electrolyte-free water excretion.

235. D. Toluene is metabolized to hippuric acid; after substantial renal excretion of hippurate with sodium and potassium, patients may present with hypokalemic normal-gap acidosis. Renal ammonium excretion can be high, giving a negative urine anion gap. The acid-base pattern depends on timing and renal function, so a high-gap component may also occur earlier.

236. B. Hypertension both contributes to CKD progression and is a major cardiovascular risk factor in CKD. Effective treatment, including renin-angiotensin blockade for appropriate albuminuric patients, can reduce proteinuria and slow progression. Volume control, sodium intake, and overall cardiovascular risk management remain important.

237. E. Mycophenolate commonly causes diarrhea, abdominal symptoms, and leukopenia because it inhibits lymphocyte purine synthesis and affects rapidly dividing cells. Dose reduction, temporary interruption, formulation changes, or alternative immunosuppression may be needed after excluding infection and rejection-related concerns. Cytopenias can also be multifactorial in transplant recipients.

238. C. Nephrotic syndrome creates a hypercoagulable state through urinary loss of natural anticoagulants, increased hepatic procoagulant synthesis, platelet activation, and sometimes hemoconcentration. Venous thromboembolism risk is particularly high in severe membranous nephropathy. Prophylactic anticoagulation decisions require individualized assessment of thrombosis and bleeding risks.

239. C. Uremic pericarditis is a classic indication for urgent dialysis or intensification of dialysis. Anticoagulation and NSAIDs may be problematic because of bleeding risk and kidney dysfunction. Patients should also be assessed for pericardial effusion and tamponade physiology.

240. A. Preemptive kidney transplantation can avoid the morbidity of dialysis initiation and is associated with favorable outcomes in appropriately selected candidates. It still requires donor evaluation, surgery, lifelong immunosuppression, and ongoing monitoring for rejection and other complications. Early referral allows time for evaluation and living-donor consideration.