COMPREHENSIVE MOCK EXAM 5

Supplemental to: Nephrology Board Review 2026-2027

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ABIM Nephrology Certification Examination — 240 A-E single-best-answer questions, domain-weighted to the January 2026 ABIM blueprint

Board-Review Questions

1. A 71-year-old woman has 3 days of vomiting and poor intake. Serum sodium is 126 mEq/L, serum osmolality 268 mOsm/kg, urine osmolality 510 mOsm/kg, and urine sodium 8 mEq/L. She is orthostatic with dry mucous membranes. What is the most appropriate initial therapy?

A. Tolvaptan, because vasopressin-receptor antagonism is first-line treatment for symptomatic hypovolemic hyponatremia from gastrointestinal losses.

B. Desmopressin alone, because suppressing urinary water excretion corrects sodium depletion without replacing the lost extracellular volume.

C. Hypertonic saline as a continuous infusion to normal sodium, because any sodium below 130 mEq/L requires rapid normalization even without neurologic symptoms.

D. Isotonic saline, because the findings indicate hypovolemic hypotonic hyponatremia with renal sodium conservation.

E. Fluid restriction alone, because concentrated urine in hyponatremia establishes euvolemic SIADH regardless of examination findings.

2. A 66-year-old man with small-cell lung cancer has sodium 121 mEq/L, serum osmolality 258 mOsm/kg, urine osmolality 620 mOsm/kg, and urine sodium 54 mEq/L. He is clinically euvolemic, with normal thyroid and adrenal testing. Which diagnosis best explains the hyponatremia?

A. Primary polydipsia, which usually produces maximally dilute urine because water intake suppresses endogenous vasopressin release.

B. Hypovolemic hyponatremia, which usually lowers urine sodium when intact kidneys conserve sodium in response to volume depletion.

C. Pseudohyponatremia, which occurs with extreme lipid or protein elevations and is associated with a normal measured serum osmolality.

D. Syndrome of inappropriate antidiuresis, causing inappropriately concentrated urine despite hypotonic plasma in a euvolemic patient.

E. Diabetes insipidus, which causes water loss and dilute urine rather than hypotonic hyponatremia with concentrated urine.

3. A 59-year-old woman with chronic hyponatremia develops a generalized seizure. Serum sodium is 108 mEq/L. Which immediate treatment is most appropriate?

A. Give a bolus of 3% hypertonic saline and reassess symptoms and serum sodium after the initial controlled rise.

B. Correct the sodium to the normal range within several hours because seizure activity indicates that rapid full normalization is necessary.

C. Begin isotonic saline at a maintenance rate and wait for spontaneous suppression of vasopressin before repeating laboratory testing.

D. Restrict oral fluid to 800 mL/day because fluid restriction corrects severe neurologic manifestations more safely than hypertonic therapy.

E. Administer tolvaptan immediately because aquaresis is the preferred emergency therapy when seizures are caused by profound hyponatremia.

4. A malnourished man with chronic alcohol use presents with sodium 104 mEq/L. After volume resuscitation, his sodium rises from 104 to 116 mEq/L in 8 hours and urine output increases markedly. What is the best next step?

A. Start oral urea to increase osmotic water excretion because the principal danger at this stage is persistent cerebral edema from undercorrection.

B. Administer sodium bicarbonate because alkali infusion preferentially stabilizes brain cells during rapid correction of chronic hyponatremia.

C. Continue isotonic saline because the sodium remains markedly below normal and should keep rising until it exceeds 130 mEq/L.

D. Add a loop diuretic without free water because increasing electrolyte-free water excretion will safely accelerate correction of severe hyponatremia.

E. Give desmopressin and free water as needed to halt or reverse excessive correction and reduce osmotic demyelination risk.

5. A 48-year-old man drinks 10 to 12 beers daily and eats very little solid food. Sodium is 118 mEq/L, serum osmolality is low, and urine osmolality is 70 mOsm/kg. Which mechanism is most likely?

A. Mineralocorticoid deficiency causes renal sodium wasting and hyperkalemia, producing concentrated urine through effective volume depletion.

B. Low dietary solute intake limits maximal renal free-water excretion despite appropriately suppressed vasopressin.

C. Advanced kidney failure prevents any urinary dilution because the collecting duct becomes completely unresponsive to changes in vasopressin.

D. Osmotic diuresis from glucosuria causes water loss in excess of sodium and therefore produces hypernatremia rather than this pattern.

E. Persistent vasopressin secretion from SIADH keeps urine concentrated even when plasma osmolality is substantially reduced.

6. A psychiatric patient drinks more than 8 L of water daily. Sodium is 123 mEq/L and urine osmolality is 55 mOsm/kg. Kidney, thyroid, and adrenal function are normal. Which process best explains the laboratory pattern?

A. SIADH with persistent vasopressin release, which would usually produce urine osmolality well above the maximally dilute range.

B. Cerebral salt wasting with renal sodium loss, which would produce clinical volume depletion and a higher urine osmolality from vasopressin release.

C. Nephrogenic diabetes insipidus, which produces dilute urine but usually raises serum sodium when water intake cannot match urinary losses.

D. Heart failure with low effective arterial volume, which typically causes nonosmotic vasopressin release and concentrated urine despite hyponatremia.

E. Primary polydipsia with intake exceeding renal excretory capacity while vasopressin is appropriately suppressed.

7. A patient with severe systolic heart failure has edema, jugular venous distention, and sodium 126 mEq/L. Urine osmolality is 480 mOsm/kg. Which mechanism is most responsible for the hyponatremia?

A. Pseudohyponatremia from elevated plasma protein explains most cases of hyponatremia associated with decompensated systolic heart failure.

B. True extracellular volume depletion suppresses the renin-angiotensin system, leading to sodium excretion and secondary free-water retention.

C. Reduced effective arterial blood volume triggers nonosmotic vasopressin release and water retention despite total-body volume excess.

D. Primary aldosterone deficiency causes isolated water retention without affecting sodium or potassium handling in the distal nephron.

E. Excess atrial natriuretic peptide completely suppresses vasopressin and creates maximally dilute urine despite the low serum sodium.

8. A patient with cirrhosis and tense ascites has sodium 124 mEq/L but no seizures or severe confusion. Which initial strategy best addresses this dilutional hyponatremia?

A. Start desmopressin to enhance water retention because vasopressin activity is insufficient in cirrhosis with low effective arterial volume.

B. Encourage unrestricted oral water intake because the low sodium concentration is caused by total-body sodium excess rather than excess water.

C. Administer repeated 3% saline boluses until sodium exceeds 135 mEq/L because ascites protects against osmotic demyelination during correction.

D. Treat the underlying volume-status problem and restrict free water when clinically appropriate rather than rapidly normalizing sodium.

E. Give large volumes of isotonic saline because cirrhosis-associated hyponatremia primarily reflects true whole-body sodium depletion.

9. After subarachnoid hemorrhage, a patient develops sodium 121 mEq/L, high urine sodium, concentrated urine, orthostatic hypotension, and negative fluid balance. Which diagnosis is most likely?

A. Primary polydipsia, because excessive water intake produces hyponatremia with very dilute urine rather than concentrated urine and sodium wasting.

B. Pseudohyponatremia, because laboratory artifact from hyperlipidemia explains negative fluid balance and orthostatic hypotension after hemorrhage.

C. Central diabetes insipidus, because loss of vasopressin causes concentrated urine with high urine sodium and progressive hypotonic hyponatremia.

D. SIADH, because both disorders cause high urine sodium, but SIADH is usually associated with clinical euvolemia rather than ongoing volume depletion.

E. Cerebral salt wasting, because renal sodium loss is accompanied by true extracellular volume depletion after neurologic injury.

10. A 75-year-old woman with fever and poor access to water has sodium 160 mEq/L. Urine osmolality is 850 mOsm/kg. Which interpretation is most appropriate?

A. Central diabetes insipidus is likely because any hypernatremia indicates inadequate vasopressin secretion regardless of urine concentration.

B. Nephrogenic diabetes insipidus is likely because collecting-duct resistance to vasopressin usually produces urine osmolality above 800 mOsm/kg.

C. Primary hyperaldosteronism is likely because sodium retention alone commonly raises serum sodium to this degree despite intact thirst mechanisms.

D. The kidneys are appropriately concentrating urine, so the hypernatremia most likely reflects extrarenal water loss or inadequate water intake.

E. SIADH is likely because concentrated urine in a hospitalized patient always indicates excessive vasopressin even when serum sodium is elevated.

11. A patient has polyuria of 7 L/day, serum sodium 151 mEq/L, and urine osmolality 90 mOsm/kg. After desmopressin, urine osmolality rises to 520 mOsm/kg. What is the diagnosis?

A. SIADH, because patients with persistent vasopressin action commonly have polyuria, hypernatremia, and urine osmolality below 100 mOsm/kg.

B. Nephrogenic diabetes insipidus, because renal resistance to vasopressin should produce a large increase in urine osmolality after desmopressin.

C. Primary polydipsia, because chronic excess water intake typically causes hypernatremia and a dramatic desmopressin response during water deprivation.

D. Osmotic diuresis, because glucosuria or urea causes very dilute urine that becomes concentrated only after exogenous vasopressin is administered.

E. Central diabetes insipidus, because desmopressin restores a substantial urine-concentrating response.

12. A 54-year-old man taking lithium for bipolar disorder has persistent polyuria and serum sodium 149 mEq/L. Urine remains dilute after desmopressin. Which treatment is most appropriate for the renal concentrating defect if lithium cannot be stopped?

A. Amiloride, which reduces lithium entry through epithelial sodium channels and can improve lithium-induced nephrogenic diabetes insipidus.

B. Spironolactone to block mineralocorticoid receptors, because aldosterone excess is the principal mechanism of lithium-induced free-water loss.

C. Loop diuretic therapy at high dose, because inhibition of the thick ascending limb maximizes medullary concentration and reduces urine volume.

D. Desmopressin as sole therapy, because lithium-induced diabetes insipidus is primarily caused by deficient hypothalamic vasopressin production.

E. Acetazolamide to increase bicarbonate excretion, because proximal bicarbonaturia directly restores aquaporin-2 trafficking in collecting ducts.

13. A patient with uncontrolled diabetes has glucose 850 mg/dL, sodium 130 mEq/L, and measured serum osmolality 322 mOsm/kg. Which statement best explains the sodium value?

A. The low sodium is pseudohyponatremia from hyperlipidemia, because severe hyperglycemia interferes with indirect ion-selective electrode measurement.

B. SIADH is the most likely cause because hyperglycemia suppresses serum osmolality and therefore should maximally stimulate vasopressin secretion.

C. Renal salt wasting is the primary mechanism because glucose lowers proximal sodium reabsorption without changing extracellular water distribution.

D. Hyperglycemia draws water into the extracellular space, producing translocational hyponatremia even though serum tonicity is high.

E. The patient has true hypotonic hyponatremia because any serum sodium below 135 mEq/L indicates excess total-body water relative to sodium.

14. A patient with severe hypertriglyceridemia has sodium 126 mEq/L measured by an indirect ion-selective electrode, normal measured serum osmolality, and no symptoms. Which diagnosis is most likely?

A. Pseudohyponatremia caused by reduced plasma water fraction in a sample measured with an indirect dilution method.

B. SIADH, because a normal measured serum osmolality is expected when vasopressin retains electrolyte-free water in the extracellular compartment.

C. Hypovolemic hyponatremia, because sodium concentration falls when the kidney maximally conserves water after gastrointestinal sodium losses.

D. Cerebral salt wasting, because high circulating triglycerides stimulate natriuretic peptides and cause renal sodium loss without changing osmolality.

E. Primary polydipsia, because large water intake lowers sodium concentration while measured serum osmolality remains normal through compensatory urea retention.

15. A woman with chronic mild hyponatremia has stable sodium values of 128 to 131 mEq/L for years. She can dilute and concentrate urine appropriately around that range, and attempts at fluid restriction do not normalize sodium. What is the most likely explanation?

A. Reset osmostat, in which the threshold for vasopressin release and thirst is chronically shifted to a lower serum osmolality.

B. Pseudohyponatremia, in which laboratory artifact would not produce a physiologic ability to dilute and concentrate urine around a new osmotic threshold.

C. Classic SIADH, in which urine remains persistently concentrated and the defended serum sodium is not normally adjustable around a lower set point.

D. Primary polydipsia, in which ongoing water intake usually produces very dilute urine and more variable sodium rather than a stable defended level.

E. Nephrogenic diabetes insipidus, in which inability to concentrate urine tends to cause polyuria and hypernatremia rather than stable hyponatremia.

16. A patient with fatigue, weight loss, orthostatic hypotension, sodium 124 mEq/L, potassium 5.8 mEq/L, and low morning cortisol is evaluated for hyponatremia. Which mechanism is most relevant?

A. Central diabetes insipidus causes sodium loss because absent vasopressin decreases proximal sodium reabsorption, resulting in hypotonic hyponatremia.

B. Hyperaldosteronism causes sodium wasting and hyperkalemia because excess mineralocorticoid downregulates epithelial sodium channels in the distal nephron.

C. SIADH causes renal sodium wasting and hyperkalemia through direct suppression of aldosterone, making adrenal testing unnecessary in this presentation.

D. Primary polydipsia causes hyperkalemia by diluting distal tubular sodium delivery and thereby preventing potassium secretion in the collecting duct.

E. Primary adrenal insufficiency causes mineralocorticoid deficiency and cortisol deficiency, promoting sodium loss, hyperkalemia, and vasopressin-mediated water retention.

17. An older woman develops nausea, confusion, and sodium 119 mEq/L one week after starting hydrochlorothiazide. She appears euvolemic and has concentrated urine. What is the most appropriate medication action?

A. Switch immediately to desmopressin monotherapy, because the syndrome results from absolute vasopressin deficiency rather than impaired dilution.

B. Stop the thiazide, because thiazides impair urinary dilution and are a common cause of clinically important hyponatremia in susceptible patients.

C. Add a potassium-sparing diuretic only, because blocking epithelial sodium channels reverses the medullary mechanism responsible for thiazide hyponatremia.

D. Continue the thiazide unchanged and liberalize free-water intake, because mild volume contraction is protective against further decline in serum sodium.

E. Increase the thiazide dose, because greater distal sodium delivery improves free-water excretion and usually corrects thiazide-associated hyponatremia.

18. A patient with SIADH has persistent sodium 128 mEq/L despite removal of the inciting drug and modest fluid restriction. He has no severe neurologic symptoms. Which principle is most appropriate?

A. Normalize serum sodium within several hours because any persistent value below 130 mEq/L carries the same cerebral-edema risk as acute symptomatic hyponatremia.

B. Give isotonic saline routinely because sodium chloride solution reliably corrects SIADH even when urine remains more concentrated than the infused fluid.

C. Administer desmopressin to reinforce antidiuresis because persistent SIADH reflects inadequate vasopressin activity at the collecting duct receptor.

D. Encourage large amounts of free water because increasing oral intake suppresses endogenous vasopressin and therefore corrects chronic SIADH.

E. Use a gradual strategy that increases electrolyte-free water excretion or solute intake while monitoring sodium, rather than emergency hypertonic therapy.

19. A patient receiving tube feeds develops sodium 154 mEq/L and high urine osmolality after several days without scheduled water flushes. What is the most appropriate conceptual treatment?

A. Correct the entire water deficit within one to two hours because chronic hypernatremia causes cerebral edema only when correction is too slow.

B. Give hypertonic saline because a high urine osmolality indicates intracellular sodium depletion that requires concentrated sodium replacement.

C. Restrict water intake because the elevated serum sodium reflects excess total-body water rather than a deficit of water relative to body sodium.

D. Replace the calculated free-water deficit gradually while continuing to account for ongoing water losses and monitoring serum sodium.

E. Administer desmopressin regardless of urine concentration because all hypernatremia in hospitalized patients is caused by unrecognized diabetes insipidus.

20. A patient with septic shock has sodium 140 mEq/L, chloride 100 mEq/L, bicarbonate 14 mEq/L, and albumin 4.0 g/dL. Which acid-base abnormality is present?

A. A metabolic alkalosis, because a bicarbonate concentration below normal indicates renal loss of hydrogen ions from the extracellular space.

B. A high-anion-gap metabolic acidosis, because the calculated anion gap is 26 mEq/L, substantially above the normal range.

C. A chronic respiratory acidosis, because chloride and sodium values can be used to infer chronic carbon dioxide retention without blood gas measurement.

D. A normal-anion-gap metabolic acidosis, because chloride is within the laboratory reference range despite the low bicarbonate concentration.

E. A primary respiratory alkalosis, because sepsis lowers bicarbonate exclusively through renal compensation for hyperventilation.

21. A patient with diabetic ketoacidosis has sodium 138 mEq/L, chloride 100 mEq/L, and bicarbonate 8 mEq/L. After treatment, the anion gap closes but bicarbonate remains 15 mEq/L and chloride rises to 113 mEq/L. What best explains the residual acidosis?

A. New respiratory acidosis, because rising chloride is a marker of carbon dioxide retention during recovery from diabetic ketoacidosis.

B. Metabolic alkalosis from insulin therapy, because intracellular potassium uptake directly raises bicarbonate and lowers serum chloride concentration.

C. Laboratory pseudohypobicarbonatemia, because chloride-containing fluids interfere with routine serum total carbon dioxide measurements after ketoacidosis.

D. Persistent ketoacidosis despite a closed anion gap, because ketone accumulation always lowers chloride rather than altering the measured anion gap.

E. A superimposed hyperchloremic normal-anion-gap metabolic acidosis from chloride-rich fluid and urinary ketoanion losses.

22. A patient has pH 7.25, PaCO2 24 mm Hg, and bicarbonate 10 mEq/L. Which interpretation of respiratory compensation is most accurate?

A. The PaCO2 is too high for compensation, proving a concurrent primary respiratory alkalosis in addition to the metabolic acidosis.

B. The PaCO2 is too low for compensation, proving a concurrent primary respiratory acidosis from ventilatory failure in addition to the metabolic acidosis.

C. The blood gas represents isolated respiratory alkalosis because any PaCO2 below 30 mm Hg necessarily establishes a primary respiratory disorder.

D. The blood gas represents metabolic alkalosis because the low PaCO2 indicates that bicarbonate has been consumed by renal compensation for hyperventilation.

E. The PaCO2 is near the expected value by Winter's formula, so the primary metabolic acidosis has appropriate respiratory compensation.

23. A patient with profuse diarrhea has pH 7.29, bicarbonate 16 mEq/L, and a normal anion gap. Urine anion gap is negative. What is the most likely explanation?

A. Chronic respiratory alkalosis, which causes low bicarbonate through renal adaptation but does not produce a primary normal-gap metabolic acidosis.

B. Gastrointestinal bicarbonate loss, with preserved renal ammonium chloride excretion producing a negative urine anion gap.

C. Distal renal tubular acidosis, in which impaired ammonium excretion commonly produces a negative urine anion gap during systemic acidosis.

D. Type 4 renal tubular acidosis, which typically causes hypokalemia and high urinary ammonium excretion in response to low serum bicarbonate.

E. Proximal renal tubular acidosis, which always causes a strongly positive urine anion gap regardless of the current serum bicarbonate concentration.

24. A 68-year-old man with diabetic kidney disease takes lisinopril. Potassium is 5.8 mEq/L, bicarbonate 18 mEq/L, and urine pH is 5.1. Which disorder is most likely?

A. Vomiting-induced metabolic alkalosis, which would increase bicarbonate and usually lower urine chloride rather than cause hyperkalemic metabolic acidosis.

B. Chronic respiratory acidosis, which would require carbon dioxide retention and does not specifically explain the persistent hyperkalemia in this setting.

C. Type 4 renal tubular acidosis from impaired aldosterone effect, causing hyperkalemia and reduced net acid excretion.

D. Distal type 1 renal tubular acidosis, which usually causes hypokalemia and an inappropriately high urine pH during systemic acidosis.

E. Proximal type 2 renal tubular acidosis, which commonly causes hyperkalemia because proximal bicarbonate wasting directly suppresses distal sodium delivery.

25. A woman has recurrent calcium phosphate stones, potassium 3.1 mEq/L, bicarbonate 17 mEq/L, and urine pH 6.4 during systemic acidosis. Which diagnosis is most likely?

A. Proximal type 2 renal tubular acidosis, because urine must remain above pH 6.0 permanently after the serum bicarbonate reaches its new steady state.

B. Diarrheal bicarbonate loss, because intact kidneys cannot lower urine pH when systemic bicarbonate is reduced by gastrointestinal losses.

C. Chronic respiratory alkalosis, because renal bicarbonate loss causes persistently alkaline urine and recurrent nephrocalcinosis in otherwise healthy adults.

D. Distal type 1 renal tubular acidosis, because the distal nephron cannot acidify urine appropriately and nephrolithiasis is common.

E. Type 4 renal tubular acidosis, because aldosterone deficiency usually causes hypokalemia, alkaline urine, and calcium phosphate nephrolithiasis.

26. A patient with multiple myeloma develops bicarbonate 16 mEq/L, potassium 3.0 mEq/L, glucosuria despite normal serum glucose, phosphaturia, and aminoaciduria. Which acid-base disorder is present?

A. Distal type 1 renal tubular acidosis, because distal hydrogen secretion failure routinely causes glucosuria and generalized aminoaciduria.

B. Type 4 renal tubular acidosis, because aldosterone resistance causes hypokalemia and widespread proximal solute wasting in plasma-cell disorders.

C. Lactic acidosis, because multiple myeloma directly raises lactate and produces a normal anion gap with glucosuria at normal serum glucose.

D. Respiratory alkalosis, because proximal bicarbonate wasting is a compensatory response to chronic hyperventilation rather than a primary tubular lesion.

E. Proximal type 2 renal tubular acidosis as part of Fanconi syndrome from generalized proximal tubular dysfunction.

27. A patient with tinnitus, nausea, and tachypnea after an overdose has pH 7.46, PaCO2 20 mm Hg, and bicarbonate 14 mEq/L. Which acid-base pattern is most likely?

A. A mixed respiratory alkalosis and high-anion-gap metabolic acidosis, typical of significant salicylate toxicity.

B. A normal acid-base state, because opposing abnormalities can normalize pH and therefore eliminate the clinical importance of either process.

C. A pure respiratory alkalosis, because the low bicarbonate is fully explained by acute renal compensation within minutes of hyperventilation.

D. A pure metabolic acidosis, because the low PaCO2 is always an appropriate compensatory response regardless of the degree of alkalemia.

E. A mixed respiratory acidosis and metabolic alkalosis, because salicylates initially depress ventilation before causing renal bicarbonate loss.

28. A patient with diabetic ketoacidosis has potassium 5.4 mEq/L before insulin is started. Which statement about total-body potassium is most accurate?

A. Total-body potassium cannot be predicted until insulin is given because serum potassium exactly reflects intracellular potassium content before therapy.

B. Total-body potassium is increased only when bicarbonate is below 10 mEq/L because severe acidosis reverses proximal potassium secretion into the urine.

C. Total-body potassium is increased because insulin deficiency prevents renal potassium excretion and therefore causes net potassium accumulation in diabetic ketoacidosis.

D. Total-body potassium is usually depleted despite the elevated serum potassium because osmotic diuresis and secondary hyperaldosteronism cause urinary potassium losses.

E. Total-body potassium is normal because extracellular acidosis shifts potassium out of cells without changing urinary potassium losses or whole-body stores.

29. A patient with potassium 7.2 mEq/L has widened QRS complexes and peaked T waves. Which treatment should be given first to reduce immediate risk of malignant arrhythmia?

A. Loop diuretic alone, because urinary potassium excretion is the fastest way to stabilize myocardial conduction in severe hyperkalemia.

B. Insulin without glucose in every patient, because hypoglycemia is an acceptable tradeoff when electrocardiographic toxicity is present.

C. Sodium bicarbonate alone, because alkalinization reliably normalizes cardiac conduction immediately even when metabolic acidosis is absent.

D. Sodium polystyrene sulfonate alone, because gastrointestinal potassium removal acts quickly enough to reverse unstable electrocardiographic changes within minutes.

E. Intravenous calcium to stabilize the cardiac membrane while therapies that lower serum potassium are initiated.

30. A patient with severe hyperkalemia has no severe hyperglycemia. Which therapy most rapidly shifts potassium into cells while definitive potassium removal is arranged?

A. Patiromer as a single emergency therapy, because its gastrointestinal binding effect begins rapidly enough to manage life-threatening hyperkalemia within minutes.

B. Fludrocortisone as the immediate intervention, because mineralocorticoid stimulation corrects severe hyperkalemia before distal nephron transport can respond.

C. Intravenous regular insulin with dextrose, which stimulates cellular potassium uptake through the sodium-potassium ATPase.

D. Calcium gluconate as the sole potassium-lowering treatment, because membrane stabilization also transports extracellular potassium into skeletal muscle cells.

E. Oral sodium bicarbonate in the absence of acidosis, because any increase in bicarbonate produces a predictable large intracellular potassium shift.

31. A patient with potassium 6.5 mEq/L receives insulin and dextrose. Which additional therapy can provide a rapid intracellular potassium shift through beta-2 adrenergic stimulation?

A. Calcium carbonate, because gastrointestinal calcium binding creates a transcellular gradient that lowers serum potassium within several minutes.

B. Propranolol, because beta-receptor blockade enhances sodium-potassium ATPase activity and accelerates potassium entry into skeletal muscle.

C. Digoxin, because inhibition of the sodium-potassium ATPase increases intracellular potassium storage despite its known effects on membrane transport.

D. Hydrochlorothiazide, because distal sodium delivery shifts potassium into cells independently of its slower effect on urinary potassium excretion.

E. Nebulized albuterol, which can lower serum potassium by increasing cellular uptake when used at appropriate high doses.

32. A patient with CKD has recurrent potassium 5.5 to 5.9 mEq/L while taking an ACE inhibitor that is otherwise indicated. He has no ECG changes and no emergency features. Which strategy can help maintain renin-angiotensin system therapy?

A. Use repeated insulin-dextrose injections at home because intracellular shifting is a safe long-term strategy for chronic asymptomatic hyperkalemia.

B. Use dietary review, correction of reversible contributors, and a modern potassium binder when appropriate to control chronic hyperkalemia.

C. Increase potassium-rich salt substitutes because higher oral potassium intake suppresses aldosterone and improves distal potassium secretion over time.

D. Stop all renin-angiotensin system blockade permanently after any potassium value above 5.0 mEq/L because rechallenge is never appropriate in CKD.

E. Give intravenous calcium before each ACE-inhibitor dose because membrane stabilization prevents the chronic potassium rise caused by reduced aldosterone signaling.

33. A patient with leukemia has serum potassium 6.4 mEq/L but no symptoms or ECG changes. Plasma potassium obtained promptly in a heparinized tube is 4.6 mEq/L, and platelet count is 1.2 million/mm3. What is the diagnosis?

A. Type 4 renal tubular acidosis, because thrombocytosis interferes with aldosterone action and raises both serum and plasma potassium equally.

B. Pseudohyperkalemia from potassium release during clotting in the setting of extreme thrombocytosis.

C. Hemolytic hyperkalemia from intravascular red-cell destruction, because a normal plasma potassium excludes an in vitro collection artifact.

D. Familial hyperkalemic hypertension, because platelet counts above one million are a typical manifestation of altered distal chloride transport.

E. True tumor lysis syndrome, because any leukemia patient with thrombocytosis and elevated serum potassium has rapid intracellular potassium release in vivo.

34. A patient taking furosemide has potassium 2.7 mEq/L and metabolic alkalosis. A spot urine potassium is high despite hypokalemia. What is the most likely cause?

A. Remote gastrointestinal potassium loss, because the kidney should continue excreting large amounts of potassium even when hypokalemia is established.

B. Intracellular potassium shift from beta-blocker therapy, because beta blockade stimulates sodium-potassium ATPase and commonly causes marked hypokalemia.

C. Pseudohypokalemia from thrombocytosis, because high platelet counts consume potassium after blood is drawn and produce metabolic alkalosis in vivo.

D. Adrenal insufficiency, because deficient aldosterone increases distal potassium secretion and typically produces hypokalemic metabolic alkalosis.

E. Renal potassium wasting from loop-diuretic therapy, which increases distal sodium delivery and promotes potassium secretion.

35. A patient has persistent potassium 2.8 mEq/L despite repeated potassium chloride replacement. Magnesium is 1.0 mg/dL. What is the best next step?

A. Administer insulin with glucose because shifting potassium into cells corrects the underlying total-body potassium deficit more effectively than oral replacement.

B. Stop potassium replacement because persistent hypokalemia despite treatment proves that the serum result is a laboratory artifact rather than a true deficit.

C. Give sodium bicarbonate because alkalinization reduces distal potassium secretion and directly corrects magnesium-associated renal potassium wasting.

D. Start spironolactone as the only therapy because mineralocorticoid blockade immediately replenishes intracellular magnesium stores and corrects hypokalemia.

E. Replete magnesium because magnesium deficiency promotes renal potassium wasting and makes hypokalemia refractory to potassium replacement.

36. A 42-year-old patient has resistant hypertension, potassium 2.9 mEq/L, bicarbonate 31 mEq/L, suppressed renin, and elevated aldosterone. Which process best explains the acid-base abnormality?

A. The metabolic alkalosis is caused by reduced distal sodium delivery because aldosterone closes epithelial sodium channels in principal cells.

B. Mineralocorticoid excess increases distal sodium reabsorption while enhancing potassium and hydrogen secretion, causing hypokalemic metabolic alkalosis.

C. The bicarbonate elevation is pseudometabolic alkalosis caused by hypokalemia interfering with laboratory measurement of serum total carbon dioxide.

D. Aldosterone excess impairs distal hydrogen secretion and therefore produces hyperkalemic normal-anion-gap metabolic acidosis despite the elevated blood pressure.

E. Primary aldosteronism causes proximal bicarbonate wasting, which lowers serum bicarbonate and leads to a compensatory respiratory alkalosis.

37. A patient with recurrent vomiting has bicarbonate 36 mEq/L and urine chloride 8 mEq/L. Which category of metabolic alkalosis is most likely?

A. Proximal renal tubular acidosis, because bicarbonate wasting from the proximal tubule produces high serum bicarbonate after repeated emesis.

B. Chloride-responsive metabolic alkalosis from gastric acid loss and extracellular volume contraction.

C. Type 4 renal tubular acidosis, because vomiting suppresses aldosterone and causes hyperkalemic metabolic alkalosis with low urinary chloride.

D. Chronic respiratory acidosis, because renal bicarbonate retention always produces urine chloride below 10 mEq/L regardless of volume status.

E. Chloride-resistant metabolic alkalosis from autonomous mineralocorticoid excess, which usually presents with very low urine chloride despite sodium retention.

38. A patient taking high-dose loop diuretics has bicarbonate 34 mEq/L and urine chloride 58 mEq/L while the medication is active. Which interpretation is most accurate?

A. A urine chloride above 20 mEq/L excludes diuretic-induced alkalosis because loop diuretics uniformly cause complete renal chloride conservation during treatment.

B. The elevated urine chloride proves primary aldosteronism because no other cause of metabolic alkalosis can increase urinary chloride excretion.

C. Active diuretic effect can produce a chloride-rich urine despite a contraction metabolic alkalosis, so medication timing matters when interpreting urine chloride.

D. The patient has distal renal tubular acidosis because high urine chloride with alkalemia indicates impaired collecting-duct hydrogen secretion.

E. The patient has respiratory alkalosis because urine chloride is a direct surrogate for arterial carbon dioxide concentration during diuretic therapy.

39. A patient with shock has lactate 9 mmol/L, bicarbonate 12 mEq/L, and an elevated anion gap. Which management principle is most important?

A. Use chronic oral citrate as definitive therapy because high-anion-gap lactic acidosis is primarily caused by inadequate dietary base intake.

B. Restore tissue perfusion and treat the underlying cause of lactate generation rather than relying on bicarbonate to eliminate the primary problem.

C. Administer bicarbonate until serum bicarbonate normalizes because alkali directly stops cellular lactate production even if tissue hypoperfusion persists.

D. Restrict intravenous fluid and vasopressors because improving perfusion increases lactate washout and transiently worsens the metabolic acidosis.

E. Give acetazolamide to increase bicarbonate excretion because lowering serum bicarbonate accelerates hepatic metabolism of circulating lactate.

40. A patient with advanced kidney failure has bicarbonate 15 mEq/L and a high anion gap without another obvious cause. What most directly contributes to this acid-base disorder?

A. Increased distal hydrogen secretion creates systemic acidosis because secreted hydrogen ions diffuse back into the circulation when nephron number falls.

B. Hyperaldosteronism from kidney failure suppresses ammonium production but simultaneously causes a metabolic alkalosis that raises the anion gap.

C. Reduced renal excretion of fixed acids and impaired ammonium generation lead to retention of sulfate, phosphate, and other unmeasured anions.

D. Excess renal bicarbonate excretion is the dominant mechanism because failing kidneys continue filtering bicarbonate but cannot reclaim it proximally.

E. Primary respiratory alkalosis is universal in kidney failure and accounts for the high anion gap by lowering measured chloride concentration.

41. A patient with chronic COPD has pH 7.36, PaCO2 60 mm Hg, and bicarbonate 33 mEq/L. Which interpretation is most likely?

A. Chronic respiratory acidosis with renal bicarbonate retention as compensation for sustained hypercapnia.

B. Primary metabolic alkalosis with appropriate respiratory compensation, because any bicarbonate above 30 mEq/L excludes a primary respiratory disorder.

C. Mixed metabolic acidosis and respiratory alkalosis, because a near-normal pH indicates two opposing primary processes rather than chronic compensation.

D. Normal acid-base physiology, because a pH within the laboratory reference range excludes clinically important chronic hypercapnia.

E. Acute respiratory acidosis without renal compensation, because bicarbonate should remain near 24 mEq/L whenever carbon dioxide rises abruptly.

42. A patient with stage G5 CKD has phosphate 6.8 mg/dL, calcium 8.4 mg/dL, and markedly elevated parathyroid hormone. What is the most likely underlying mechanism?

A. Autonomous parathyroid adenoma is the usual first event in CKD and directly causes phosphate retention by increasing proximal tubular phosphate reabsorption.

B. Phosphate retention, reduced calcitriol production, and hypocalcemic signaling drive secondary hyperparathyroidism in advanced CKD.

C. Excess calcitriol synthesis in failing kidneys raises intestinal phosphate absorption and suppresses calcium enough to stimulate parathyroid hormone release.

D. Primary hypoparathyroidism develops as nephron mass falls, and the elevated measured parathyroid hormone reflects accumulation of inactive assay fragments only.

E. Renal phosphate wasting is the dominant abnormality in advanced CKD, causing low serum phosphate and compensatory parathyroid hormone suppression.

43. A patient with new squamous-cell carcinoma has calcium 13.2 mg/dL and suppressed intact parathyroid hormone. Which mediator is a likely cause of this hypercalcemia?

A. Erythropoietin excess, because tumor-related erythrocytosis increases serum calcium concentration by releasing calcium from mature red blood cells.

B. Calcitonin excess from the tumor, because calcitonin increases osteoclastic bone resorption and typically produces severe hypercalcemia with low PTH.

C. Fibroblast growth factor 23 deficiency, because reduced FGF23 directly causes rapid malignant hypercalcemia through increased renal calcium reabsorption.

D. Parathyroid hormone-related peptide, which can mimic PTH effects on bone and kidney while endogenous PTH is suppressed.

E. Aldosterone excess, because mineralocorticoid signaling increases distal calcium reabsorption and is a common paraneoplastic mechanism in squamous carcinoma.

44. A patient with severe secondary hyperparathyroidism undergoes parathyroidectomy and then develops profound hypocalcemia, low phosphate, and high bone-specific alkaline phosphatase. What is the most likely diagnosis?

A. Acute calcitriol toxicity, because increased intestinal calcium absorption paradoxically lowers serum calcium when parathyroid hormone falls abruptly.

B. Pseudohypoparathyroidism, because end-organ resistance to PTH begins immediately after gland removal and typically causes low phosphate with hypocalcemia.

C. Familial hypocalciuric hypercalcemia, because calcium-sensing receptor variants commonly produce severe postoperative hypocalcemia after parathyroidectomy.

D. Tumor lysis syndrome, because parathyroid surgery releases intracellular phosphate and potassium that drive calcium into bone after the operation.

E. Hungry bone syndrome from rapid skeletal uptake of calcium and phosphate after abrupt reduction in parathyroid hormone.

45. A patient with advanced CKD has weakness, hypotension, bradycardia, and magnesium 8.0 mg/dL after using large amounts of magnesium-containing laxative. What is the most appropriate initial therapy for cardiovascular toxicity?

A. Desmopressin, because severe hypermagnesemia is primarily caused by renal water loss and can be reversed by increasing collecting-duct water reabsorption.

B. Sodium bicarbonate alone, because alkalinization rapidly shifts magnesium intracellularly and eliminates the need for dialysis in advanced kidney failure.

C. Potassium chloride, because raising serum potassium directly displaces magnesium from cardiac ion channels and reverses hypotension within minutes.

D. Intravenous calcium to antagonize the membrane effects of severe hypermagnesemia while magnesium removal is arranged.

E. Intravenous magnesium sulfate, because additional magnesium stabilizes the myocardium and prevents arrhythmia during renal magnesium retention.

46. A patient taking a proton-pump inhibitor for several years has muscle cramps, magnesium 0.8 mg/dL, potassium 3.0 mEq/L, and low urinary magnesium excretion. Which mechanism is most likely?

A. Primary renal magnesium wasting from loop-diuretic physiology, because low urinary magnesium proves that the kidney is losing magnesium excessively.

B. Tumor lysis syndrome, because rapid intracellular magnesium uptake commonly causes chronic hypomagnesemia together with hypokalemia in stable outpatients.

C. Reduced intestinal magnesium absorption associated with chronic proton-pump inhibitor use, with appropriate renal magnesium conservation.

D. Hyperaldosteronism alone, because mineralocorticoid excess typically produces severe isolated magnesium depletion with low urinary magnesium excretion.

E. Distal renal tubular acidosis, because impaired urinary acidification directly blocks intestinal magnesium transport and lowers urine magnesium excretion.

47. A patient has recurrent calcium oxalate stones, normal serum calcium, low urine citrate, and no hyperoxaluria. Which preventive therapy is most appropriate?

A. Potassium citrate to increase urinary citrate and reduce calcium stone supersaturation when hypocitraturia is present.

B. Acetazolamide, because alkaline urine from carbonic anhydrase inhibition prevents calcium phosphate and calcium oxalate stones in hypocitraturia.

C. Urine acidification with ammonium chloride, because lowering urine pH increases citrate excretion and reduces calcium oxalate crystal formation.

D. High-dose vitamin C, because increasing urinary oxalate binds free calcium and prevents calcium oxalate nucleation in recurrent stone formers.

E. Calcium restriction to near zero, because eliminating dietary calcium consistently lowers urinary oxalate absorption and prevents recurrent stones.

48. A patient with gout has recurrent radiolucent uric acid stones and urine pH 5.0. Which intervention is most important for prevention?

A. Alkalinize the urine with potassium citrate, because uric acid solubility rises substantially as urine pH increases.

B. Start a loop diuretic, because increasing urinary calcium competes with uric acid and prevents crystal nucleation in acidic urine.

C. Use acetazolamide as the preferred long-term therapy, because its effect on urine pH outweighs the increased risk of calcium phosphate stones.

D. Acidify the urine with ascorbic acid, because urate remains more soluble in its protonated form at a urine pH below 5.5.

E. Restrict all dietary calcium, because uric acid stones form only when calcium complexes with urate in the gastrointestinal tract.

49. A patient has recurrent staghorn calculi and urine cultures repeatedly grow Proteus mirabilis. Which stone composition is most likely?

A. Cystine, because Proteus infection oxidizes cysteine to cystine and produces the classic infection-associated staghorn calculus.

B. Magnesium ammonium phosphate, because urease-producing organisms alkalinize urine and promote struvite stone formation.

C. Calcium oxalate monohydrate, because bacterial urease directly increases urinary oxalate concentration and forms most infection stones.

D. Drug-induced indinavir crystals, because Proteus biofilm traps protease-inhibitor metabolites and creates radiopaque branching stones.

E. Uric acid, because urease lowers urine pH and converts urate into poorly soluble uric acid that forms branching staghorn stones.

50. A young adult with recurrent stones has hexagonal crystals on urine microscopy and a positive cyanide-nitroprusside screening test. Which disorder is most likely?

A. Primary hyperoxaluria, which produces envelope-shaped calcium oxalate crystals and a positive cyanide-nitroprusside test from urinary oxalate.

B. Distal renal tubular acidosis, which produces hexagonal calcium phosphate crystals because persistently alkaline urine changes crystal geometry.

C. Cystinuria caused by defective proximal tubular reabsorption of cystine and dibasic amino acids.

D. Uric acid nephrolithiasis, which produces hexagonal urate crystals and occurs primarily when urinary cystine excretion is elevated.

E. Struvite nephrolithiasis, which produces hexagonal magnesium ammonium phosphate crystals in patients with urease-positive urinary infection.

51. A patient has recurrent nephrolithiasis, serum calcium 11.4 mg/dL, phosphate 2.1 mg/dL, and elevated parathyroid hormone. Which diagnosis best explains the findings?

A. Vitamin D deficiency, which lowers intestinal calcium absorption and therefore usually produces hypocalcemia or normal calcium rather than this profile.

B. Secondary hyperparathyroidism from CKD, which usually occurs with low or normal calcium and phosphate retention rather than hypercalcemia with hypophosphatemia.

C. Hypoparathyroidism, which causes hyperphosphatemia and hypocalcemia because renal calcium reabsorption and bone calcium mobilization are reduced.

D. Primary hyperparathyroidism, causing PTH-mediated hypercalcemia, phosphaturia, and increased calcium stone risk.

E. Familial hypocalciuric hypercalcemia as the most likely cause, because that disorder typically produces marked hypercalciuria and recurrent calcium stones.

52. A 52-year-old man has an eGFR of 54 mL/min/1.73 m2 on two measurements 2 weeks apart after an episode of gastroenteritis. Urinalysis is normal and prior creatinine values were normal. Which statement is most accurate?

A. Acute kidney injury is excluded because the eGFR values are similar on two measurements separated by more than 7 days.

B. CKD is excluded because a normal urinalysis rules out chronic kidney disease whenever eGFR remains above 45 mL/min/1.73 m2.

C. Chronic kidney disease cannot yet be diagnosed solely from these results because abnormal kidney structure or function must persist for at least 3 months.

D. Stage G3a CKD is established immediately because any single eGFR below 60 mL/min/1.73 m2 is sufficient regardless of duration.

E. Kidney failure is present because an eGFR below 60 mL/min/1.73 m2 indicates loss of more than half of normal filtration capacity.

53. A patient has eGFR 42 mL/min/1.73 m2 and urine albumin-creatinine ratio 520 mg/g for 6 months. How should the kidney disease be classified?

A. CKD category G3b with severely increased albuminuria category A3, based on an eGFR of 30–44 mL/min/1.73 m2 and UACR above 300 mg/g.

B. CKD category G4 with normal albuminuria A1 because any eGFR below 45 mL/min/1.73 m2 is classified as G4.

C. Kidney failure category G5 with A3 albuminuria because persistent albumin above 300 mg/g defines kidney failure regardless of eGFR.

D. CKD category G3a with A2 albuminuria because eGFR 30 to 59 and albuminuria 30 to 999 are each treated as single broad categories.

E. CKD category G2 with moderately increased albuminuria A2 because albuminuria determines the G stage when eGFR is below 60.

54. A patient with diabetes has a urine dipstick reported as trace protein. Which test is preferred to quantify low-level albuminuria for CKD risk assessment?

A. A random urine sodium concentration, because sodium excretion closely tracks glomerular albumin permeability in diabetic kidney disease.

B. A serum albumin concentration, because circulating albumin directly quantifies urinary albumin loss before overt nephrotic syndrome develops.

C. A urine specific gravity, because concentrated urine allows precise conversion of dipstick protein into albumin excretion per day.

D. A spot urine albumin-creatinine ratio, preferably confirmed when persistent albuminuria would change management.

E. A 24-hour urine urea nitrogen collection, because nitrogen excretion is the standard surrogate for urinary albumin loss in early CKD.

55. A muscular 32-year-old man has creatinine-based eGFR 58 mL/min/1.73 m2, no albuminuria, normal urinalysis, and no structural kidney abnormality. Kidney function classification will affect a medication decision. Which next test is most useful?

A. Use cystatin C, ideally in a combined creatinine-cystatin C eGFR equation, to improve GFR estimation when creatinine may be misleading.

B. Perform kidney biopsy, because any discrepancy between body habitus and creatinine-based eGFR requires histologic confirmation before drug dosing.

C. Repeat serum sodium, because sodium concentration is a more direct filtration marker in people with unusually high muscle mass.

D. Measure blood urea nitrogen alone, because urea is independent of protein intake and therefore gives a more accurate GFR than creatinine.

E. Order a urine eosinophil count, because occult interstitial nephritis is the most likely explanation for isolated mildly reduced creatinine eGFR.

56. A 67-year-old patient with CKD G3b and albuminuria asks about risk of needing kidney replacement therapy. Which tool is specifically designed to estimate short-term kidney failure risk using routine clinical variables?

A. The Kidney Failure Risk Equation, which incorporates variables such as age, sex, eGFR, and albuminuria to estimate kidney failure risk.

B. The Wells score, because venous thromboembolism risk closely parallels the rate of GFR loss in patients with albuminuric CKD.

C. The Framingham score alone, because cardiovascular risk and kidney failure risk are interchangeable outcomes once eGFR falls below 45.

D. The CHA2DS2-VASc score, because stroke-risk variables also accurately predict progression from moderate CKD to kidney failure.

E. The MELD score, because creatinine is included and therefore the score can be applied broadly to kidney failure prediction outside cirrhosis.

57. A patient with nondiabetic CKD has urine albumin-creatinine ratio 900 mg/g and blood pressure 142/84 mm Hg. Potassium is 4.5 mEq/L. Which drug class is especially appropriate to slow kidney disease progression?

A. A loop diuretic alone because lowering extracellular volume reduces albuminuria and has the same proven kidney-protective effect as RAAS blockade.

B. A direct renin inhibitor combined with an ACE inhibitor because dual blockade provides more complete proteinuria reduction without additional harm.

C. An alpha blocker as first-line renoprotective therapy because sympathetic blockade specifically reduces glomerular protein trafficking independent of blood pressure.

D. A nondihydropyridine calcium-channel blocker as mandatory monotherapy because RAAS blockade is contraindicated whenever eGFR is below normal.

E. An ACE inhibitor or angiotensin receptor blocker titrated as tolerated because marked albuminuria identifies a strong indication for renin-angiotensin system blockade.

58. A patient with albuminuric CKD remains hypertensive despite maximal tolerated lisinopril. Which strategy should generally be avoided because it increases adverse events without improving kidney outcomes?

A. Adding a dihydropyridine calcium-channel blocker because combination therapy is often needed to reach blood pressure targets in CKD.

B. Adding a thiazide-like diuretic when kidney function and volume status permit because sodium retention commonly contributes to resistant hypertension.

C. Reducing dietary sodium because lower sodium intake can improve blood pressure and augment the antiproteinuric effect of RAAS blockade.

D. Adding an angiotensin receptor blocker to the ACE inhibitor for dual renin-angiotensin system blockade.

E. Assessing adherence and standardized blood pressure measurement because apparent resistance is common when measurement or medication use is inconsistent.

59. A 63-year-old patient has CKD G3a, urine albumin-creatinine ratio 650 mg/g, no diabetes, and stable volume status. Which medication class can reduce CKD progression risk even in many patients without diabetes?

A. Acarbose, because delaying intestinal carbohydrate absorption lowers albuminuria independently of glycemia and is proven in nondiabetic CKD.

B. A sulfonylurea, because glucose-independent kidney protection is a class effect shared by all medications that increase pancreatic insulin secretion.

C. An SGLT2 inhibitor, provided there is no contraindication and kidney function is within an evidence-supported range.

D. A DPP-4 inhibitor, because these agents reduce intraglomerular pressure even in patients without diabetes and are established CKD disease-modifying therapy.

E. A thiazolidinedione, because fluid retention expands renal plasma flow and slows decline in eGFR across diabetic and nondiabetic kidney disease.

60. Two weeks after starting an SGLT2 inhibitor, a patient with stable CKD has an eGFR decline from 48 to 43 mL/min/1.73 m2, normal potassium, stable blood pressure, and no volume depletion. What is the best interpretation?

A. The eGFR decline demonstrates acute interstitial nephritis and requires immediate glucocorticoid therapy even without urinary or systemic findings.

B. The change proves bilateral renal artery stenosis because only renovascular disease produces an early GFR decline after medications that affect glomerular hemodynamics.

C. The patient has proven progressive intrinsic kidney injury because any eGFR decline after starting an SGLT2 inhibitor indicates nephrotoxicity.

D. A modest early eGFR dip can be an expected hemodynamic effect and does not by itself require stopping an otherwise appropriate SGLT2 inhibitor.

E. The medication should be stopped permanently because SGLT2 inhibitors are contraindicated once eGFR falls below the value present at initiation.

61. A patient with type 2 diabetes, CKD, persistent albuminuria, potassium 4.4 mEq/L, and maximally tolerated ACE-inhibitor therapy remains at high cardiorenal risk. Which additional therapy may be appropriate?

A. High-dose fludrocortisone because mineralocorticoid agonism lowers albuminuria by increasing distal sodium reabsorption and reducing renin release.

B. Chronic intravenous albumin because raising oncotic pressure directly seals glomerular filtration barriers and prevents diabetic CKD progression.

C. Long-term NSAID therapy because afferent vasoconstriction lowers intraglomerular pressure and provides the same outcome benefit as finerenone.

D. Routine dual ACE inhibitor and ARB therapy because combined blockade is the preferred next step when albuminuria persists despite one RAAS inhibitor.

E. A nonsteroidal mineralocorticoid receptor antagonist such as finerenone, with careful potassium monitoring.

62. A patient with type 2 diabetes and CKD remains above individualized glycemic goals despite metformin and an SGLT2 inhibitor. Obesity and atherosclerotic cardiovascular disease are also present. Which class is particularly attractive?

A. A long-acting GLP-1 receptor agonist with demonstrated cardiovascular benefit, which can also support weight loss and kidney-risk reduction.

B. A short-acting sulfonylurea as preferred escalation because hypoglycemia risk falls as GFR declines and weight gain improves kidney perfusion.

C. Chronic prandial insulin as the only acceptable option because all noninsulin glucose-lowering therapies are contraindicated once CKD is present.

D. A thiazolidinedione as first choice because fluid retention and weight gain are desirable hemodynamic effects in albuminuric diabetic kidney disease.

E. An alpha-glucosidase inhibitor as mandatory therapy because it provides superior cardiovascular outcome data to GLP-1 receptor agonists in CKD.

63. A 61-year-old patient with CKD G3b has no known atherosclerotic cardiovascular disease and asks about lipid therapy. Which approach is generally appropriate?

A. Use statin-based therapy because CKD substantially increases cardiovascular risk even when the primary goal is not to slow GFR decline directly.

B. Use niacin routinely because raising HDL cholesterol has consistently improved survival and reduced kidney failure in moderate to advanced CKD.

C. Avoid statins because CKD patients have predominantly nonatherosclerotic cardiovascular events and derive no meaningful benefit from LDL lowering.

D. Use fibrate monotherapy as first-line treatment regardless of triglyceride level because fibrates have the strongest cardiovascular outcome data in CKD.

E. Delay lipid therapy until dialysis begins because cardiovascular risk becomes clinically important only after kidney replacement therapy is required.

64. A patient with proteinuric CKD and hypertension consumes a high-sodium diet. Which dietary change is most likely to improve blood pressure control and augment antiproteinuric therapy?

A. Eliminate all dietary protein instead of reducing sodium because protein intake is the primary determinant of extracellular volume in albuminuric CKD.

B. Reduce dietary sodium intake because sodium restriction improves volume control and enhances the effect of RAAS blockade on blood pressure and albuminuria.

C. Increase processed-food intake because phosphate additives improve natriuresis and make loop diuretics more effective in patients with reduced GFR.

D. Increase sodium intake because higher distal sodium delivery suppresses the renin-angiotensin system and therefore reduces albumin excretion in CKD.

E. Replace sodium chloride with unlimited potassium chloride because salt substitutes are universally safe even when eGFR is low or RAAS blockade is used.

65. A patient with stable CKD G4 asks whether a very high-protein diet will preserve muscle and kidney function. Which counseling is most appropriate?

A. Eliminate nearly all dietary protein because severe protein restriction is safe without ketoacid supplementation and reliably prevents kidney failure.

B. Protein intake has no effect on kidney hemodynamics or uremic solute generation, so there is no reason to discuss dietary protein in CKD care.

C. Use only animal protein because plant protein increases acid production and phosphate absorption more than comparable animal-protein diets.

D. Avoid excessive protein intake; a moderate protein intake is generally preferred while preventing malnutrition and maintaining adequate calories.

E. Recommend a very high-protein diet because increasing renal blood flow from protein loading slows nephron loss and reduces glomerular hyperfiltration injury.

66. A patient with CKD G4 has bicarbonate values of 17 to 19 mEq/L on repeated testing and no acute illness. Which intervention may be considered after evaluating volume status and other causes?

A. Chronic acetazolamide because increasing urinary bicarbonate loss corrects acid retention and prevents CKD progression in advanced disease.

B. Long-term hydrochloric acid supplementation because low bicarbonate reflects systemic alkalosis from impaired renal chloride excretion.

C. Oral alkali therapy to correct clinically important metabolic acidosis while monitoring blood pressure, volume status, and serum bicarbonate.

D. Routine intravenous sodium bicarbonate through a central line because oral alkali is ineffective whenever eGFR is below 30 mL/min/1.73 m2.

E. No treatment can be considered until dialysis starts because metabolic acidosis is an unavoidable adaptive state in CKD and should not be modified.

67. A patient with proteinuric CKD benefits from an ACE inhibitor but develops potassium 5.6 mEq/L without ECG changes. Which principle is preferred before abandoning kidney-protective therapy?

A. Look for reversible causes and use dietary, diuretic, alkali, or potassium-binding strategies when appropriate so RAAS blockade can be continued if safely possible.

B. Increase potassium-containing salt substitutes because higher potassium intake induces renal adaptation and reliably normalizes potassium in advanced CKD.

C. Stop the ACE inhibitor permanently after the first mild potassium elevation because renoprotective benefit never justifies any hyperkalemia risk.

D. Give scheduled intravenous calcium before each ACE-inhibitor dose because membrane stabilization is a practical chronic strategy for outpatient hyperkalemia.

E. Prescribe repeated insulin-dextrose doses at home because intracellular potassium shifts are safer than modifying diet or adding a potassium binder.

68. A patient with CKD G4 has hemoglobin 9.4 g/dL. Which initial laboratory evaluation is most useful before attributing the anemia entirely to erythropoietin deficiency?

A. Assess iron availability with ferritin and transferrin saturation, together with evaluation for other common reversible causes of anemia.

B. Measure serum erythropoietin concentration because the diagnosis of renal anemia requires a low erythropoietin level before any treatment can be considered.

C. Perform bone marrow biopsy routinely because CKD-related anemia is a diagnosis that cannot be made without direct marrow assessment.

D. Measure parathyroid hormone alone because secondary hyperparathyroidism explains nearly all anemia once eGFR falls below 30 mL/min/1.73 m2.

E. Order only a reticulocyte count because iron studies are unreliable in CKD and should not influence iron replacement decisions.

69. A hemodialysis patient has anemia, low transferrin saturation, and low ferritin. Which treatment principle is most appropriate?

A. Give folate alone because low ferritin in dialysis usually reflects folate deficiency rather than depleted iron stores.

B. Increase dialysis blood-flow rate because iron deficiency is caused by inadequate solute clearance and corrects when Kt/V rises above target.

C. Transfuse red blood cells routinely because iron replacement is too slow to be useful in chronic dialysis-associated anemia.

D. Replace iron because absolute iron deficiency can limit erythropoiesis and reduce responsiveness to erythropoiesis-stimulating therapy.

E. Avoid iron because dialysis patients cannot use administered iron and treatment primarily increases infection risk without improving hemoglobin response.

70. A patient with nondialysis CKD has hemoglobin 9.1 g/dL, iron deficiency has been corrected, and fatigue remains substantial. Which principle should guide consideration of an erythropoiesis-stimulating agent?

A. Start an ESA automatically whenever hemoglobin is below 12 g/dL because normalization of hemoglobin improves cardiovascular outcomes in all CKD stages.

B. Never use an ESA before dialysis because endogenous erythropoietin remains adequate until kidney replacement therapy is initiated.

C. Individualize ESA initiation after reversible causes are addressed, balancing symptoms and transfusion avoidance against cardiovascular and thrombotic risks.

D. Use an ESA only if ferritin is above 1,000 ng/mL because high ferritin is required to prove adequate iron availability in CKD.

E. Transfuse first and defer ESA consideration indefinitely because red-cell transfusion has fewer long-term consequences than chronic erythropoiesis stimulation.

71. A dialysis patient receiving an ESA has hemoglobin increased from 9.5 to 13.2 g/dL. Which management principle is most appropriate?

A. No change is needed because ESA-associated risk depends only on iron status and is unrelated to achieved hemoglobin or dose intensity.

B. Increase ESA dosing further because hemoglobin normalization above 14 g/dL has consistently reduced stroke and vascular-access thrombosis in dialysis patients.

C. Add routine red-cell transfusions because transfusion plus ESA produces more stable hemoglobin than either strategy alone in chronic kidney failure.

D. Reduce or hold ESA dosing because targeting near-normal hemoglobin with high ESA exposure can increase cardiovascular and thrombotic risk.

E. Stop iron permanently because any hemoglobin above 12 g/dL proves iron overload even when ferritin and transferrin saturation are not elevated.

72. A patient with CKD who is being evaluated for kidney transplantation has asymptomatic anemia that can be managed without transfusion. Why is avoiding unnecessary red-cell transfusion especially important?

A. Transfusion can cause HLA sensitization, potentially making compatible kidney transplantation more difficult and prolonging waiting time.

B. Transfusion prevents future living donation because donor-specific antibodies are formed against every possible HLA type after any exposure.

C. Transfusion causes irreversible hyperkalemia in all CKD patients because stored red cells release enough potassium to prohibit later transplantation.

D. Transfusion permanently suppresses endogenous erythropoietin production, making subsequent ESA therapy ineffective even after transplantation.

E. Transfusion accelerates CKD progression by directly depositing iron in glomeruli after a single unit of packed red blood cells.

73. A patient with CKD has rising parathyroid hormone over several visits. Calcium and phosphate have also changed over time. Which approach best reflects CKD-mineral bone disorder management?

A. Use serum calcium alone because phosphate and parathyroid hormone no longer provide useful information once eGFR is below 60 mL/min/1.73 m2.

B. Use a single phosphate measurement to determine long-term therapy because phosphate varies little with diet, timing, or dialysis exposure.

C. Normalize every marker to the healthy reference range at each visit because CKD-MBD targets are identical across nondialysis and dialysis populations.

D. Interpret calcium, phosphate, and parathyroid hormone together and follow serial trends rather than treating a single isolated laboratory value.

E. Treat any parathyroid hormone value above the laboratory range immediately with parathyroidectomy regardless of CKD stage or calcium and phosphate values.

74. A hemodialysis patient has persistently elevated phosphate despite dietary counseling and adequate dialysis. Which treatment is reasonable?

A. Give calcitriol as the sole phosphate-lowering treatment because increasing intestinal phosphate absorption paradoxically lowers serum phosphate through PTH suppression.

B. Stop dialysis phosphate removal because repeated removal stimulates bone release and is the principal cause of persistent hyperphosphatemia.

C. Encourage phosphate-containing food additives because rapidly absorbed inorganic phosphate improves skeletal buffering and lowers circulating phosphate.

D. Give intravenous phosphate after each dialysis session because postdialysis phosphate rebound indicates clinically important whole-body phosphate depletion.

E. Use a phosphate-lowering strategy such as a phosphate binder, individualized to calcium balance, pill burden, and other clinical factors.

75. A nondialysis CKD G3b patient has mildly elevated parathyroid hormone, normal calcium, and normal phosphate. Which approach to active vitamin D is most appropriate?

A. Active vitamin D is contraindicated in all nondialysis CKD because it has no biologic effect until a patient starts kidney replacement therapy.

B. Do not routinely use calcitriol solely for a modest PTH elevation; first evaluate modifiable factors and reserve active vitamin D for selected progressive severe hyperparathyroidism.

C. Start high-dose calcitriol in every patient with PTH above the normal assay range because complete PTH normalization prevents fractures and cardiovascular events.

D. Use calcitriol only when serum calcium is high because active vitamin D lowers calcium while suppressing parathyroid hormone in CKD.

E. Give calcitriol as a phosphate binder because its primary therapeutic action in CKD is reducing intestinal phosphate absorption.

76. A hemodialysis patient has severe secondary hyperparathyroidism with high PTH and calcium that is already at the upper end of normal. Which medication class may lower PTH without increasing calcium?

A. Hydrochlorothiazide, because thiazide-induced hypercalcemia directly suppresses parathyroid hormone and is standard therapy in anuric dialysis patients.

B. Bisphosphonate monotherapy, because suppressing bone resorption is the primary treatment for secondary hyperparathyroidism in kidney failure.

C. Calcitriol alone, because active vitamin D reliably lowers both parathyroid hormone and serum calcium in dialysis patients with hyperparathyroidism.

D. Calcium carbonate in escalating doses, because increasing calcium exposure is preferred when baseline calcium is already high-normal.

E. A calcimimetic, which increases calcium-sensing receptor sensitivity and suppresses parathyroid hormone secretion.

77. A dialysis patient with extensive vascular calcification and recurrent hypercalcemia requires phosphate-binding therapy. Which principle is most appropriate?

A. Increase calcitriol and calcium simultaneously because raising the calcium-phosphate product slows progression of medial vascular calcification.

B. Limit unnecessary calcium loading and consider a non-calcium phosphate binder when clinically appropriate.

C. Stop all phosphate binders because vascular calcification means serum phosphate should be allowed to rise to reduce skeletal calcium release.

D. Use magnesium-containing antacids without monitoring because dialysis removes magnesium completely and prevents clinically important accumulation.

E. Use only calcium-based binders because vascular calcification is unrelated to cumulative calcium exposure in patients receiving dialysis.

78. A patient with kidney failure develops sharp pleuritic chest pain improved by sitting forward, a friction rub, and no alternative cause. Which treatment is most appropriate?

A. High-dose NSAID therapy as sole treatment because renal function no longer matters once kidney failure is established.

B. Immediate thrombolysis because pericarditic chest pain in kidney failure most commonly represents acute coronary thrombosis with atypical ECG changes.

C. Observation without dialysis because uremic pericarditis usually resolves as blood urea nitrogen rises and does not predict tamponade.

D. Urgent initiation or intensification of dialysis because uremic pericarditis is a classic indication for kidney replacement therapy.

E. Chronic colchicine alone because dialysis has no effect on retained uremic mediators responsible for pericardial inflammation.

79. A patient with kidney failure has potassium 6.8 mEq/L despite temporizing therapy and maximal medical potassium removal. Which next step is most appropriate?

A. Initiate urgent dialysis because refractory severe hyperkalemia is a life-threatening indication for kidney replacement therapy.

B. Delay intervention until muscle weakness appears because serum potassium values are not clinically important without neuromuscular symptoms.

C. Continue oral potassium binder alone for several days because definitive extracorporeal removal is unnecessary when the patient has kidney failure.

D. Give repeated calcium indefinitely because membrane stabilization removes potassium from the body and can replace dialysis when ECG changes improve.

E. Administer potassium chloride to stimulate distal potassium secretion because the kidney adapts to increased intake even in advanced kidney failure.

80. A patient with advanced CKD has pulmonary edema and severe hypoxemia despite high-dose loop diuretics and optimized medical therapy. Which intervention is indicated?

A. Give repeated hypertonic saline boluses because raising serum sodium draws water from the lungs into the intravascular compartment for renal excretion.

B. Urgent dialysis or ultrafiltration because refractory volume overload with respiratory compromise is an indication for kidney replacement therapy.

C. Use acetazolamide alone regardless of GFR because proximal bicarbonate loss is the definitive treatment for diuretic-resistant pulmonary edema in kidney failure.

D. Increase oral water intake because expanding plasma volume improves renal perfusion and will reverse pulmonary edema even when diuretics have failed.

E. Stop all diuretics and observe because pulmonary edema is expected in advanced CKD and does not by itself justify kidney replacement therapy.

81. A patient with CKD has eGFR 8 mL/min/1.73 m2 but feels well, has controlled potassium and bicarbonate, good appetite, and manageable volume status. Which principle should guide dialysis initiation?

A. Delay dialysis until eGFR reaches zero because residual filtration always prevents uremic complications until no measurable kidney function remains.

B. Begin dialysis only after serum creatinine exceeds 10 mg/dL because creatinine concentration is the validated universal trigger for kidney replacement therapy.

C. Start dialysis only when urine output stops because patients who still make urine cannot develop clinically important uremic manifestations.

D. Start dialysis immediately because every patient must begin treatment when eGFR falls below 10 mL/min/1.73 m2 regardless of symptoms or laboratory control.

E. Base the decision on symptoms, complications, and inability to maintain homeostasis rather than starting solely because eGFR crossed a fixed numeric threshold.

82. A patient with progressive CKD G4 is likely to require hemodialysis within the next year. Which access-planning principle is most appropriate?

A. Wait until the first dialysis session to discuss access because fistulas and grafts mature immediately and do not require advance planning.

B. Place a tunneled dialysis catheter preemptively in every CKD G4 patient because catheters have the lowest infection and thrombosis rates of all access types.

C. Create an arteriovenous fistula in every patient regardless of prognosis, vessels, modality preference, or likelihood of reaching kidney failure.

D. Discuss kidney replacement options early and plan permanent access in a patient-centered way before an emergency catheter-dependent start becomes necessary.

E. Avoid transplant education until dialysis has begun because preemptive transplantation is not an option for patients who have never received dialysis.

83. A hemodialysis patient repeatedly develops catheter-related bloodstream infection. Which access principle is most relevant?

A. Antibiotic prophylaxis before every dialysis session eliminates the excess infection risk of chronic catheter dependence and is recommended routinely.

B. Long-term dialysis catheters are preferred because their intravascular location prevents bacterial colonization compared with surgically created access.

C. Catheter infection risk disappears after the first month because mature fibrin sheaths prevent organisms from reaching the bloodstream.

D. Replacing the catheter every week is the preferred infection-prevention strategy even when a durable arteriovenous access can be created.

E. A functioning arteriovenous fistula or graft generally carries less bloodstream-infection risk than a chronic central venous dialysis catheter.

84. A stable thrice-weekly hemodialysis patient has persistently low delivered small-solute clearance because of shortened treatments. Which parameter is commonly used to quantify urea clearance adequacy?

A. Fractional excretion of sodium, because FENa quantifies the proportion of body urea removed by each hemodialysis treatment.

B. Creatinine clearance from a spot blood sample, because a single postdialysis creatinine concentration directly yields the delivered dialysis dose.

C. Kt/V for urea, which relates dialyzer clearance and treatment time to the patient's urea distribution volume.

D. Urine albumin-creatinine ratio, because albumin excretion directly measures extracorporeal urea removal in patients receiving maintenance hemodialysis.

E. Serum bicarbonate alone, because postdialysis bicarbonate is a complete surrogate for clearance of urea and other retained solutes.

85. A hemodialysis patient develops recurrent intradialytic hypotension after arriving 5 kg above target weight each session. What is the most useful initial strategy?

A. Reassess dry weight, interdialytic sodium and fluid intake, and ultrafiltration rate rather than repeatedly treating each episode without addressing excessive fluid gain.

B. Raise dialysate temperature because warmer dialysate increases peripheral vasoconstriction and is the standard prevention for intradialytic hypotension.

C. Give large saline boluses prophylactically throughout every session because replacing the volume being removed improves long-term hypertension control.

D. Stop all antihypertensive therapy permanently because medication timing and volume status are irrelevant once hypotension occurs during dialysis.

E. Increase the ultrafiltration rate further because faster fluid removal shortens the period of low blood pressure and improves organ perfusion.

86. A patient with very high blood urea nitrogen begins first-time hemodialysis and develops headache, nausea, restlessness, and confusion during treatment. What is the most likely diagnosis?

A. Uremic pericarditis, because pericardial inflammation during dialysis commonly presents first with headache, nausea, and acute confusion.

B. Heparin-induced thrombocytopenia, because neurologic symptoms during the first dialysis treatment are usually caused by acute platelet activation.

C. Dialysis disequilibrium syndrome from rapid reduction in plasma osmolality causing cerebral edema during initial dialysis.

D. Air embolism is certain because dialysis disequilibrium cannot occur until a patient has received several weeks of repeated treatment.

E. Acute hypernatremia, because hemodialysis rapidly raises serum sodium enough to pull water into brain cells during initial treatment.

87. A peritoneal dialysis patient reports abdominal pain and cloudy effluent. Effluent white-cell count is elevated with neutrophil predominance. What is the most appropriate initial management?

A. Give intravenous loop diuretics as sole therapy because abdominal pain results primarily from intraperitoneal volume overload rather than infection.

B. Obtain appropriate cultures and start empiric intraperitoneal antibiotics covering gram-positive and gram-negative organisms without delaying treatment.

C. Stop peritoneal dialysis permanently before obtaining cultures because cloudy effluent proves irreversible peritoneal membrane failure.

D. Treat with oral antifungal medication alone because fungi account for most first episodes of peritoneal dialysis-associated peritonitis.

E. Observe for 48 hours without antibiotics because cloudy dialysate commonly reflects dietary fat and usually resolves spontaneously.

88. A peritoneal dialysis patient has poor ultrafiltration during a long dwell and develops volume overload. Which osmotic agent can improve ultrafiltration during a prolonged dwell in selected patients?

A. Intraperitoneal albumin for every exchange, because albumin is the routine first-line osmotic agent for chronic peritoneal ultrafiltration failure.

B. Sodium bicarbonate powder, because alkalinizing dialysate creates a durable osmotic gradient and prevents glucose absorption during long dwells.

C. Free sterile water, because a hypotonic dwell pulls sodium and water across the peritoneum more effectively than an osmotic agent.

D. Isotonic saline, because sodium chloride is the principal osmotic driver of ultrafiltration in standard peritoneal dialysis solutions.

E. Icodextrin, a glucose polymer that can sustain colloid osmotic ultrafiltration during long dwell periods.

89. A peritoneal dialysis patient has purulent drainage from the catheter exit site and tenderness along the subcutaneous tunnel. Which complication is most concerning?

A. A tunnel infection associated with the peritoneal dialysis catheter, which requires antimicrobial treatment and may threaten catheter survival.

B. Uremic pericarditis, because purulent exit-site drainage is an expected manifestation of systemic serosal inflammation from inadequate dialysis.

C. Encapsulating peritoneal sclerosis, because that disorder begins as acute purulent drainage before progressing to bowel obstruction.

D. Renal colic, because stone passage can refer pain to the abdominal wall and produce sterile purulent material at a dialysis exit site.

E. Dialysis disequilibrium syndrome, because neurologic osmotic shifts commonly present as localized tenderness along the peritoneal catheter tract.

90. A patient on peritoneal dialysis for many years develops recurrent bowel obstruction, weight loss, and imaging showing peritoneal thickening and cocooning of bowel loops. Which diagnosis is most likely?

A. Simple PD peritonitis, because routine bacterial peritonitis typically causes chronic fibrotic cocooning even after infection has been treated.

B. Dialysis disequilibrium syndrome, because rapid urea shifts in peritoneal dialysis produce progressive peritoneal thickening and bowel obstruction.

C. Encapsulating peritoneal sclerosis, a rare serious complication characterized by peritoneal fibrosis and intestinal encapsulation.

D. Uremic ileus, because retained uremic toxins create a permanent fibrous membrane around the bowel without structural peritoneal disease.

E. Nephrogenic systemic fibrosis, because gadolinium exposure primarily causes intra-abdominal peritoneal encasement rather than skin and connective-tissue changes.

91. A dialysis patient still makes 800 mL of urine daily. Why is preservation of residual kidney function clinically valuable?

A. Diuretics must be stopped once dialysis starts because maintaining urine output accelerates loss of the remaining nephrons in all patients.

B. Residual kidney function contributes continuous solute and volume removal and is associated with better fluid control and patient outcomes.

C. Residual urine has no clinical value after dialysis begins because dialysis completely replaces endocrine, tubular, and clearance functions of the kidneys.

D. Urine output matters only for potassium removal and has no effect on phosphate, middle molecules, fluid balance, or quality of life.

E. Residual kidney function should be intentionally eliminated because variable native clearance makes dialysis prescription less predictable and increases mortality.

92. A patient with CKD G4 has not received hepatitis B vaccination and is likely to need hemodialysis in the future. Which preventive strategy is appropriate?

A. Use passive hepatitis B immune globulin every month instead of vaccination because active immune responses cannot be generated in advanced CKD.

B. Vaccinate only after kidney transplantation because immunosuppression improves vaccine immunogenicity compared with the predialysis period.

C. Delay vaccination until after a dialysis catheter is placed because hepatitis B vaccine is ineffective before a patient has direct blood exposure.

D. Begin hepatitis B immunization before dialysis when possible, because vaccine response is generally better earlier in CKD and exposure risk increases with dialysis.

E. Avoid hepatitis B vaccination because CKD patients have excessive vaccine-related immune activation and higher risk of glomerulonephritis.

93. A patient with eGFR 24 mL/min/1.73 m2 requires contrast-enhanced CT for a potentially life-threatening diagnosis. Which principle is most appropriate?

A. Give prophylactic loop diuretics to force high urine output because diuresis reliably prevents contrast-associated kidney injury in advanced CKD.

B. Do not withhold a clinically necessary study solely because of CKD; assess risk, avoid volume depletion and unnecessary nephrotoxins, and use appropriate contrast and hydration strategies when indicated.

C. Use oral sodium bicarbonate as the only preventive measure because intravascular volume status does not influence kidney risk around contrast exposure.

D. Substitute gadolinium-enhanced MRI automatically because gadolinium carries no kidney-related risk and is always diagnostically equivalent to CT.

E. Cancel all iodinated-contrast imaging whenever eGFR is below 30 mL/min/1.73 m2 because the risk of irreversible dialysis-dependent kidney failure is prohibitive.

94. A patient with CKD G3b takes ibuprofen daily for osteoarthritis and develops a rise in creatinine during a diarrheal illness while also taking an ACE inhibitor and diuretic. Which mechanism is most likely contributing?

A. NSAIDs dilate the afferent arteriole, causing glomerular hyperfiltration that directly raises creatinine during volume depletion.

B. ACE inhibitors constrict the efferent arteriole, so combining them with NSAIDs excessively increases intraglomerular pressure and filtration fraction.

C. Diuretics prevent volume depletion by increasing proximal sodium reabsorption, making a hemodynamic fall in GFR unlikely during diarrhea.

D. NSAID-mediated afferent arteriolar vasoconstriction combined with reduced effective volume and efferent dilation can sharply lower glomerular filtration pressure.

E. The creatinine rise proves immune-complex glomerulonephritis because medication-related hemodynamic kidney injury cannot occur in established CKD.

95. A patient with CKD needs a renally cleared antibiotic. Which principle best guides dosing?

A. Use urine albumin-creatinine ratio as the primary dosing variable because albuminuria predicts drug clearance more accurately than estimated filtration.

B. Use an appropriate estimate of kidney function together with the specific drug's labeling, indication, loading-dose needs, and dialysis status rather than reducing every dose by the same percentage.

C. Reduce all medication doses by 50% whenever eGFR is below 60 mL/min/1.73 m2 because renal clearance falls proportionally for every drug.

D. Avoid loading doses in CKD because the volume of distribution always falls in parallel with GFR, making standard loading doses toxic.

E. Base dosing only on serum creatinine concentration because age, body size, sex, and the drug's pharmacokinetic studies do not influence renal dosing.

96. A 39-year-old woman with CKD G3b and significant albuminuria is planning pregnancy. Which counseling point is most appropriate?

A. Albuminuria is protective in pregnancy because the physiologic rise in GFR reduces the risk of preeclampsia and preterm delivery.

B. CKD eliminates the normal hemodynamic changes of pregnancy, so serum creatinine and blood pressure targets remain identical to nonpregnant values.

C. Pregnancy has no additional risk until eGFR falls below 15 mL/min/1.73 m2, so preconception nephrology counseling is unnecessary in moderate CKD.

D. ACE inhibitors should be continued throughout pregnancy because fetal kidney effects occur only in patients without maternal CKD.

E. Pregnancy carries increased maternal and fetal risk in CKD, and medications such as ACE inhibitors or ARBs require preconception review because they are not used during pregnancy.

97. A patient with CKD G5 has severe generalized pruritus that persists despite skin care and correction of obvious metabolic contributors. Which approach is reasonable?

A. Use a symptom-directed therapy such as a carefully dosed gabapentinoid or other evidence-based antipruritic strategy, with renal dosing and adverse-effect monitoring.

B. Use high-dose diphenhydramine as the definitive therapy because histamine release is the primary mechanism in nearly all CKD-associated pruritus.

C. Stop dialysis because extracorporeal clearance worsens pruritus by removing endogenous antipruritic molecules from the bloodstream.

D. Start chronic systemic glucocorticoids because CKD-associated pruritus is fundamentally an autoimmune dermatitis that requires immunosuppression.

E. Increase phosphate intake because uremic pruritus is caused by systemic phosphate depletion and improves when serum phosphate rises above normal.

98. A patient with CKD has progressive edema despite a standard loop-diuretic dose. Blood pressure is stable and there is no acute kidney injury. Which strategy can improve natriuresis when loop resistance is due to distal nephron adaptation?

A. Stop the loop diuretic and add desmopressin because increasing collecting-duct water reabsorption enhances sodium excretion in diuretic-resistant edema.

B. Add an NSAID because afferent vasoconstriction increases sodium delivery to the loop of Henle and potentiates loop-diuretic natriuresis.

C. Increase dietary sodium because higher filtered sodium load overcomes distal nephron adaptation and reduces edema more effectively than combination diuretics.

D. Use sequential nephron blockade by adding a thiazide-type diuretic to the loop diuretic with close monitoring of electrolytes and volume status.

E. Use acetazolamide as lifelong monotherapy because proximal sodium blockade completely prevents distal sodium reabsorption in advanced CKD.

99. A patient with CKD and heart failure has rising creatinine after aggressive diuresis but marked improvement in congestion, blood pressure, and symptoms. Which interpretation is most appropriate?

A. The finding requires stopping all heart-failure therapies permanently because kidney function and congestion cannot be managed simultaneously.

B. A modest creatinine rise during effective decongestion can reflect hemodynamic change and should be interpreted in the context of volume status rather than automatically reversing therapy.

C. Any creatinine rise during diuresis proves tubular necrosis and requires immediate fluid loading until creatinine returns to baseline.

D. Successful decongestion should always lower creatinine, so improvement in edema with a higher creatinine indicates laboratory error.

E. The patient has bilateral renal artery stenosis because diuretic therapy cannot change creatinine through intravascular hemodynamics alone.

100. A patient with CKD G4 has repeated episodes of hypoglycemia despite an unchanged insulin regimen and stable diet. Which mechanism is most likely contributing?

A. Reduced renal insulin clearance and impaired renal gluconeogenesis can prolong insulin action and increase hypoglycemia risk as kidney function declines.

B. Albuminuria causes insulin to be lost in urine, shortening insulin half-life and producing persistent hyperglycemia rather than hypoglycemia.

C. Secondary hyperparathyroidism directly stimulates pancreatic glucagon release enough to prevent clinically important hypoglycemia in advanced CKD.

D. CKD accelerates renal insulin degradation, so insulin doses generally need to rise progressively as eGFR falls.

E. Reduced GFR eliminates peripheral insulin sensitivity, making hypoglycemia rare even when food intake is inconsistent.

101. A patient with CKD has serum potassium 5.3 mEq/L and asks about using a potassium-based salt substitute to lower sodium intake. Which counseling is most appropriate?

A. Salt substitutes contain only sodium bicarbonate and therefore have no effect on potassium balance or hyperkalemia risk.

B. Hyperkalemia risk from salt substitutes occurs only in dialysis patients and is negligible in nondialysis CKD even with RAAS blockade.

C. Potassium intake does not influence serum potassium in CKD because reduced nephron number completely prevents gastrointestinal potassium absorption.

D. Potassium-containing salt substitutes can worsen hyperkalemia in CKD, so sodium reduction should be pursued with individualized attention to potassium content.

E. Potassium salt substitutes are always preferred in CKD because potassium intake reliably lowers serum potassium by stimulating distal tubular secretion.

102. A patient with CKD G3 and recurrent gout is started on urate-lowering therapy. Which general dosing principle is most appropriate?

A. Avoid all xanthine oxidase inhibitors in CKD because reduced GFR makes any dose predictably nephrotoxic and dialysis is the only safe gout treatment.

B. Select the agent and titrate according to kidney function, comorbidities, drug interactions, and serum urate response rather than withholding urate-lowering therapy solely because CKD is present.

C. Use chronic NSAIDs at full dose for prophylaxis because kidney function does not alter the renal hemodynamic risk of anti-inflammatory therapy.

D. Target the highest tolerated serum urate because higher urate concentrations reduce crystal mobilization and lower flare risk in CKD.

E. Use probenecid as mandatory first-line therapy in advanced CKD because declining GFR increases its uricosuric efficacy.

103. A patient with CKD G4 has severe secondary hyperparathyroidism, bone pain, and fractures despite optimized medical therapy. Which additional evaluation may be appropriate when the type of renal osteodystrophy would change treatment?

A. Consider bone biopsy in selected cases because turnover and mineralization cannot always be inferred reliably from serum markers alone.

B. Give bisphosphonate therapy immediately because all fractures in CKD arise from osteoporosis rather than CKD-mineral bone disorder.

C. Use dual-energy x-ray absorptiometry alone to identify the exact histologic type of renal osteodystrophy because bone density directly measures turnover and mineralization.

D. Measure serum calcium once because a single calcium value distinguishes high-turnover from adynamic bone disease with sufficient accuracy for all treatment decisions.

E. Perform routine parathyroidectomy without further evaluation because fractures in CKD always indicate high-turnover bone disease from excess PTH.

104. A patient with CKD has persistently elevated phosphate. Which dietary counseling point is most useful?

A. Increase cola beverages because phosphoric acid is excreted unchanged and therefore lowers serum phosphate through osmotic diuresis.

B. Avoid all plant foods because plant-based phosphate is more completely absorbed than inorganic phosphate additives used in processed foods.

C. Eliminate all protein because dietary phosphate is present only in protein foods and malnutrition does not affect outcomes in advanced CKD.

D. Pay particular attention to highly absorbable inorganic phosphate additives in processed foods as well as total dietary phosphate and nutritional adequacy.

E. Favor processed foods with phosphate additives because additive phosphate is poorly absorbed compared with organic phosphate in whole foods.

105. A patient has office blood pressure 148/86 mm Hg measured immediately after walking in, with feet unsupported and no rest period. What should be done before labeling the hypertension uncontrolled?

A. Repeat blood pressure using a standardized technique after proper rest, correct cuff sizing, back and feet support, and multiple readings.

B. Use only the highest reading obtained in clinic because averaging measurements underestimates cardiovascular risk in CKD.

C. Measure blood pressure over clothing with a smaller cuff because tighter cuff compression improves accuracy in patients with vascular disease.

D. Rely on pulse pressure alone because systolic and diastolic values are not clinically interpretable unless arterial stiffness is measured invasively.

E. Increase antihypertensive therapy immediately because technique-related error is too small to affect a systolic reading above 140 mm Hg.

106. A patient has repeated clinic blood pressure around 128/76 mm Hg but ambulatory monitoring averages 148/86 mm Hg. Which phenotype is present?

A. White-coat hypertension, which requires elevated clinic pressure with normal home or ambulatory pressure outside the office.

B. Pseudohypertension, which is diagnosed whenever ambulatory pressure exceeds office pressure regardless of vascular calcification.

C. Isolated systolic hypertension, because any ambulatory systolic value above 140 mm Hg establishes that phenotype even when diastolic pressure is also elevated.

D. No hypertension, because ambulatory monitoring is less predictive of target-organ risk than a normal clinic measurement.

E. Masked hypertension, in which out-of-office blood pressure is elevated despite apparently normal clinic readings.

107. A patient takes maximally tolerated doses of an ACE inhibitor, amlodipine, and chlorthalidone but has office blood pressure 166/92 mm Hg. Before diagnosing true resistant hypertension, what should be addressed?

A. Confirm adherence, accurate measurement, out-of-office blood pressure, adequate diuretic therapy, and removal of substances that raise blood pressure.

B. Diagnose primary aldosteronism without testing because all patients taking three antihypertensive medications have autonomous aldosterone secretion.

C. Proceed directly to renal denervation because failure of three medications proves treatment-resistant sympathetic hypertension.

D. Add two more agents without reviewing adherence because medication nonadherence rarely contributes when patients report taking therapy.

E. Stop the diuretic because volume control is not relevant to resistant hypertension once an ACE inhibitor and calcium-channel blocker are used.

108. A 44-year-old patient has resistant hypertension and spontaneous potassium 2.9 mEq/L. Which screening test is most appropriate for primary aldosteronism?

A. Measure serum cortisol after an ACTH stimulation test because autonomous cortisol secretion is the defining biochemical abnormality of primary aldosteronism.

B. Order only a serum potassium level because hypokalemia below 3.0 mEq/L is diagnostic and eliminates the need for renin or aldosterone testing.

C. Measure plasma metanephrines only because hypokalemia is more characteristic of pheochromocytoma than of primary aldosteronism.

D. Measure plasma aldosterone and renin and interpret the aldosterone-to-renin relationship in the context of medications and potassium status.

E. Obtain renal biopsy because primary aldosteronism is diagnosed by juxtaglomerular-cell hyperplasia rather than hormonal testing.

109. A patient has biochemically confirmed primary aldosteronism and a 1.5-cm unilateral adrenal nodule on CT. He is a surgical candidate and wants definitive therapy. What is the best next localization step in most older adults?

A. Perform renal-artery angiography because renovascular hypertension is the only important alternative diagnosis once an adrenal nodule is present.

B. Repeat the aldosterone-renin ratio after adrenalectomy because lateralization cannot be determined before surgery.

C. Use a dexamethasone suppression test as the sole lateralization study because cortisol suppression identifies which adrenal gland secretes excess aldosterone.

D. Adrenal venous sampling to determine whether aldosterone secretion is unilateral or bilateral before adrenalectomy.

E. Proceed directly to remove the adrenal gland containing the CT nodule because imaging alone reliably identifies the aldosterone-producing side in all adults.

110. A 73-year-old patient with atherosclerosis has severe bilateral renal-artery stenosis, recurrent flash pulmonary edema, and difficult-to-control hypertension. Which management consideration is most appropriate?

A. Renal-artery stenting is mandatory for every atherosclerotic stenosis above 50%, even when blood pressure and kidney function are stable.

B. Revascularization is never appropriate because randomized trials exclude benefit in all presentations of atherosclerotic renovascular disease.

C. Revascularization may be considered because recurrent flash pulmonary edema is a high-risk clinical syndrome in which restoring renal perfusion can be beneficial.

D. Treat only with NSAIDs because afferent vasoconstriction reduces renin release and is a safe way to control flash pulmonary edema.

E. Perform bilateral nephrectomy because renovascular hypertension cannot be managed medically or endovascularly once pulmonary edema occurs.

111. A 31-year-old woman has abrupt-onset hypertension, an abdominal bruit, and imaging showing a 'string-of-beads' appearance in a renal artery. Which diagnosis is most likely?

A. Fibromuscular dysplasia causing renovascular hypertension from nonatherosclerotic renal-artery disease.

B. Primary aldosteronism, which creates the angiographic string-of-beads sign through mineralocorticoid-induced vascular remodeling.

C. Atherosclerotic renal-artery stenosis, which typically produces a string-of-beads pattern in young women without vascular risk factors.

D. Polyarteritis nodosa, which characteristically creates alternating fibrotic webs and dilations limited to the main renal artery.

E. Renal vein thrombosis, which produces beading of the renal artery because venous obstruction causes alternating arterial dilation and stenosis.

112. A patient has episodic headache, palpitations, diaphoresis, and paroxysmal hypertension. Which biochemical test is appropriate for suspected pheochromocytoma?

A. Urine vanillylmandelic acid after starting a beta blocker because modern metanephrine testing is less sensitive than older catecholamine metabolite assays.

B. Kidney biopsy because chromaffin tumors are diagnosed by renal microvascular changes rather than circulating catecholamine metabolites.

C. Random plasma renin alone because catecholamine-secreting tumors primarily cause hypertension by chronically activating renal renin release.

D. Plasma free metanephrines or appropriately collected fractionated urinary metanephrines, depending on pretest probability and testing conditions.

E. Serum aldosterone alone because pheochromocytoma is diagnosed by autonomous mineralocorticoid secretion during symptomatic spells.

113. A patient with obesity, loud snoring, daytime somnolence, and resistant hypertension is evaluated for a secondary contributor. Which condition should be strongly considered?

A. Addison disease, because cortisol and aldosterone deficiency commonly produces obesity, sleep-disordered breathing, and sustained hypertension.

B. Primary hyperparathyroidism, because mild hypercalcemia is the dominant reversible cause of resistant hypertension in obese patients.

C. Nephrotic syndrome, because proteinuria is required for sleep apnea to increase sympathetic tone and blood pressure.

D. Central diabetes insipidus, because nocturnal polyuria and hypernatremia are the usual causes of resistant hypertension in patients who snore.

E. Obstructive sleep apnea, which is common in resistant hypertension and can sustain sympathetic activation and nocturnal blood pressure elevation.

114. A patient has hypertension, proximal muscle weakness, easy bruising, facial rounding, and new diabetes. Which secondary cause of hypertension is most likely?

A. Primary adrenal insufficiency, because cortisol deficiency commonly causes central obesity, bruising, diabetes, and sustained hypertension.

B. Renal salt wasting, because chronic sodium loss stimulates enough renin to produce the cushingoid phenotype and proximal myopathy.

C. Pheochromocytoma, because catecholamine excess typically causes purple striae and chronic glucocorticoid-type skin thinning.

D. Coarctation of the aorta, because upper-body hypertension directly causes cortisol excess and the characteristic metabolic phenotype.

E. Hypercortisolism, which can cause sodium retention, vascular sensitivity to catecholamines, diabetes, and characteristic catabolic features.

115. A 24-year-old patient has arm blood pressure 168/86 mm Hg, weak delayed femoral pulses, and lower leg blood pressure. Which diagnosis is most likely?

A. Liddle syndrome, which causes congenital obstruction of the descending aorta through epithelial sodium-channel overactivity.

B. Primary aldosteronism, which produces a consistent arm-leg pressure gradient by selectively constricting lower-extremity arteries.

C. Coarctation of the aorta, producing upper-extremity hypertension with reduced lower-extremity perfusion.

D. Renal-artery stenosis, which causes delayed femoral pulses because renal hypoperfusion reduces blood flow to both iliac arteries.

E. Pheochromocytoma, which produces fixed upper-extremity hypertension and absent femoral pulses between catecholamine surges.

116. A patient with blood pressure 230/130 mm Hg has confusion, papilledema, and acute kidney injury. What defines this presentation?

A. A hypertensive emergency because severe blood pressure elevation is accompanied by acute target-organ injury.

B. Resistant hypertension because target-organ damage is not relevant once more than three antihypertensive agents are needed.

C. Pseudohypertension because papilledema indicates noncompressible arteries rather than true elevation in intra-arterial pressure.

D. Hypertensive urgency because any systolic pressure above 220 mm Hg is an urgency even when neurologic and kidney injury are present.

E. White-coat hypertension because acute anxiety can produce papilledema and kidney injury without requiring intravenous treatment.

117. A patient with acute aortic dissection has blood pressure 210/110 mm Hg and heart rate 112/min. Which initial intravenous strategy is most appropriate?

A. Give a long-acting oral thiazide and reassess in 24 hours because gradual outpatient blood pressure reduction is safest in aortic dissection.

B. Rapid beta blockade to reduce heart rate and shear stress, followed by additional vasodilator therapy if blood pressure remains elevated.

C. Use pure alpha stimulation because increasing systemic vascular resistance reduces the pressure gradient across the dissection flap.

D. Avoid beta blockers because lowering heart rate increases left-ventricular ejection force and worsens propagation of the dissection.

E. Start hydralazine alone before controlling heart rate because isolated arterial vasodilation reduces aortic shear even when reflex tachycardia develops.

118. A patient with severe hypertension develops acute pulmonary edema with marked dyspnea and diffuse crackles. Which intravenous therapy can rapidly reduce preload and afterload when blood pressure permits?

A. Intravenous normal saline boluses because increasing preload is the fastest way to reverse hydrostatic pulmonary edema caused by severe hypertension.

B. Phenylephrine infusion because raising systemic vascular resistance unloads the left ventricle and lowers pulmonary capillary pressure.

C. Intravenous nitroglycerin, often at appropriately titrated doses, together with noninvasive ventilation and diuresis as clinically indicated.

D. Oral clonidine alone because rapid pulmonary edema should be managed with a delayed centrally acting antihypertensive rather than titratable intravenous therapy.

E. Desmopressin because water retention expands effective arterial volume and suppresses the sympathetic surge that causes hypertensive pulmonary edema.

119. A patient with blood pressure 240/140 mm Hg has thrombocytopenia, schistocytes, elevated LDH, and AKI. ADAMTS13 activity is not severely deficient. Which mechanism is most likely?

A. Severe hypertension causing endothelial injury and thrombotic microangiopathy rather than primary TTP.

B. Iron deficiency anemia, because red-cell fragmentation and low platelets are typical consequences of chronic iron-restricted erythropoiesis.

C. Immune TTP is proven because any schistocytes with thrombocytopenia require severe ADAMTS13 deficiency by definition.

D. Disseminated intravascular coagulation is certain because severe hypertension directly consumes fibrinogen and prolongs coagulation tests in every case.

E. Warm autoimmune hemolytic anemia, because hypertensive endothelial injury produces a positive direct antiglobulin test and spherical red cells.

120. A pregnant patient at 34 weeks has persistent blood pressure 172/114 mm Hg. Which agent is appropriate for acute severe hypertension in pregnancy?

A. Sodium nitroprusside as routine first-line therapy because fetal cyanide exposure is negligible and the drug is safer than labetalol.

B. Spironolactone because antiandrogen effects are beneficial for fetal development and potassium monitoring is unnecessary in pregnancy.

C. Lisinopril because ACE inhibition rapidly lowers blood pressure and has no fetal renal effect after the first trimester.

D. Intravenous labetalol, intravenous hydralazine, or immediate-release oral nifedipine are accepted options depending on the clinical setting.

E. Aliskiren because direct renin inhibition is preferred in pregnancy when rapid blood pressure reduction is needed.

121. A patient with nondialysis CKD has standardized office systolic pressure 138 mm Hg and tolerates therapy without orthostasis. Which general blood pressure principle is consistent with modern CKD guidance?

A. Use the same numeric target from casual triage blood pressure because standardized measurement does not affect interpretation of treatment thresholds.

B. Avoid RAAS blockers when albuminuria is present because lowering intraglomerular pressure worsens long-term CKD outcomes.

C. Treat only diastolic pressure because systolic blood pressure has little relationship to cardiovascular events in older adults with CKD.

D. Aim for intensive systolic blood pressure control using standardized measurement when tolerated, while individualizing for frailty, symptoms, and comorbidity.

E. Maintain systolic pressure above 150 mm Hg in all CKD patients because lower values accelerate kidney failure regardless of cardiovascular risk.

122. An 86-year-old patient with CKD has seated blood pressure 126/64 mm Hg but recurrent falls and standing pressure 98/52 mm Hg. Which approach is most appropriate?

A. Intensify therapy until seated systolic pressure is below 110 mm Hg because orthostatic symptoms do not alter CKD blood pressure goals.

B. Add an alpha blocker because vasodilation improves standing blood pressure and reduces falls in older patients with CKD.

C. Ignore standing measurements because only seated blood pressure predicts cardiovascular outcomes and treatment harm.

D. Stop all fluids because lower intravascular volume reliably corrects orthostatic hypotension by increasing sympathetic tone.

E. Individualize antihypertensive intensity and reduce treatment if necessary to address symptomatic orthostatic hypotension and fall risk.

123. A patient with bilateral renal-artery stenosis has recurrent sudden pulmonary edema despite preserved left-ventricular systolic function. What syndrome does this suggest?

A. Cerebral salt wasting, because renal sodium loss after neurologic injury is the classic cause of sudden pulmonary edema in patients with renovascular disease.

B. Nephrotic syndrome, because heavy proteinuria is required for every episode of flash pulmonary edema in bilateral renal-artery stenosis.

C. Pickering syndrome, in which severe renovascular disease can cause recurrent flash pulmonary edema.

D. Cardiorenal syndrome type 1, because preserved systolic function excludes any renovascular contribution to pulmonary edema.

E. Liddle syndrome, because epithelial sodium-channel activation causes a characteristic association with bilateral renal-artery stenosis and flash edema.

124. A kidney transplant recipient develops hypertension, tremor, hyperkalemia, and a gradual creatinine rise while tacrolimus trough levels are high. Which mechanism is likely contributing to the hypertension?

A. Tacrolimus lowers tubular sodium reabsorption in the distal nephron, producing volume depletion that secondarily raises blood pressure.

B. The hypertension is caused by acute adrenal insufficiency because calcineurin inhibitors block cortisol synthesis as their principal adverse effect.

C. Calcineurin-inhibitor vasoconstriction and sodium retention can raise blood pressure while also causing kidney dysfunction and hyperkalemia.

D. Tacrolimus causes systemic vasodilation and renal salt wasting, so hypertension argues against calcineurin-inhibitor toxicity.

E. Calcineurin inhibitors suppress sympathetic tone and aldosterone, making persistent hypertension unlikely unless a pheochromocytoma is present.

125. A patient with severe resistant hypertension is started on minoxidil. Which additional medications are commonly needed to counter predictable adverse effects?

A. A loop diuretic and a beta blocker because minoxidil can cause marked sodium retention and reflex tachycardia.

B. An ACE inhibitor and potassium supplement because minoxidil directly suppresses renin and aldosterone, causing hyperkalemia and bradycardia.

C. Acetazolamide and nitrates because minoxidil causes metabolic alkalosis and coronary vasospasm as its dominant dose-limiting toxicities.

D. No adjunctive therapy because minoxidil lowers heart rate and promotes natriuresis, making beta blockers and diuretics unnecessary.

E. Desmopressin and potassium chloride because minoxidil causes nephrogenic diabetes insipidus with severe potassium wasting.

126. A patient abruptly stops high-dose clonidine and develops agitation, tachycardia, and severe rebound hypertension. What is the mechanism?

A. Clonidine withdrawal causes acute aldosterone deficiency, leading to sodium loss and compensatory vasopressin-mediated hypertension.

B. Stopping clonidine blocks peripheral beta receptors, causing unopposed alpha vasoconstriction identical to untreated pheochromocytoma.

C. The rebound is caused by abrupt inhibition of renin secretion, which raises angiotensin II concentration within several hours.

D. Clonidine withdrawal causes direct renal sodium wasting, and the resulting volume depletion is the primary cause of severe hypertension.

E. Sudden withdrawal of central alpha-2 agonism causes a sympathetic surge with rebound catecholamine release.

127. A patient has hypertension, hypokalemic metabolic alkalosis, suppressed renin, suppressed aldosterone, and daily consumption of large amounts of licorice. Which mechanism is most likely?

A. Licorice inhibits renal potassium secretion, so hyperkalemia rather than hypokalemia is the defining electrolyte finding.

B. Inhibition of 11-beta-hydroxysteroid dehydrogenase type 2 allows cortisol to activate mineralocorticoid receptors, producing apparent mineralocorticoid excess.

C. Licorice directly stimulates aldosterone synthesis, so aldosterone should be markedly elevated despite suppressed renin in this syndrome.

D. Licorice blocks epithelial sodium channels, causing renal sodium wasting and secondary hyperaldosteronism with hypokalemic alkalosis.

E. The syndrome is caused by cortisol deficiency, which removes mineralocorticoid activity and therefore produces sodium retention with low renin.

128. A young patient has early severe hypertension, hypokalemic metabolic alkalosis, low renin, and low aldosterone. A sibling has the same disorder. Which diagnosis is most likely?

A. Gitelman syndrome, because NCC loss causes severe early hypertension with low renin, low aldosterone, and hypokalemic alkalosis.

B. Addison disease, because mineralocorticoid deficiency presents with low aldosterone, hypokalemia, metabolic alkalosis, and hypertension.

C. Liddle syndrome from gain-of-function epithelial sodium channels causing autonomous distal sodium reabsorption.

D. Primary aldosteronism, because autonomous aldosterone secretion produces low renin and low aldosterone after chronic sodium retention.

E. Bartter syndrome, because NKCC2 dysfunction causes hypertension and low renin through excessive sodium reabsorption in the thick ascending limb.

129. A patient develops fever, rash, eosinophilia, sterile pyuria, white-cell casts, and AKI 10 days after starting a beta-lactam antibiotic. Which diagnosis is most likely?

A. Postinfectious glomerulonephritis, because glomerular immune complexes usually produce sterile pyuria without hematuria or red-cell casts.

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

C. Renal-artery thrombosis, because cortical ischemia commonly presents with eosinophilia and an allergic rash after beta-lactam therapy.

D. Minimal change disease, because podocyte injury from beta-lactams primarily produces pyuria and eosinophilic interstitial inflammation rather than proteinuria.

E. Acute tubular necrosis, because ischemic tubular injury typically causes fever, rash, eosinophilia, and white-cell casts after antibiotic exposure.

130. A patient with decades of heavy analgesic use has CKD, sterile pyuria, and imaging showing small irregular kidneys with papillary calcifications. Which diagnosis is most likely?

A. ADPKD, because chronic analgesic exposure causes bilateral renal enlargement with innumerable cortical and medullary cysts.

B. Acute pyelonephritis, because papillary calcification and small irregular kidneys are typical after a single episode of ascending bacterial infection.

C. Membranous nephropathy, because NSAID exposure causes papillary necrosis through subepithelial immune deposits in glomeruli.

D. Analgesic nephropathy with chronic tubulointerstitial injury and papillary necrosis.

E. Renal amyloidosis, because analgesics induce amyloid deposition that primarily destroys the renal papillae and causes sterile pyuria.

131. A patient has taken lithium for 20 years and develops slowly progressive CKD with polyuria. Biopsy shows chronic tubulointerstitial fibrosis and small tubular cysts. What is the most likely cause?

A. Membranous nephropathy, because chronic lithium exposure primarily produces subepithelial immune complexes with nephrotic syndrome and preserved concentration.

B. IgA nephropathy, because lithium increases mesangial IgA deposition and causes tubular cysts as a secondary glomerular phenomenon.

C. Renal amyloidosis, because lithium is converted to amyloid fibrils that deposit in the medulla and cause nephrogenic diabetes insipidus.

D. Chronic lithium nephrotoxicity producing tubulointerstitial nephritis and collecting-duct dysfunction.

E. Acute uric acid nephropathy, because long-term lithium therapy raises uric acid enough to cause repeated crystal obstruction and cortical cysts.

132. A 35-year-old patient has hypertension, enlarged kidneys with numerous bilateral cysts, and a parent who required dialysis at age 58. Which diagnosis is most likely?

A. Autosomal dominant polycystic kidney disease, given the cystic imaging pattern and vertical family history.

B. Medullary sponge kidney, because collecting-duct ectasia routinely causes huge cortical cysts, progressive kidney failure, and dominant inheritance.

C. Simple renal cysts, because multiple large bilateral cysts with family history and progressive hypertension are expected incidental findings in young adults.

D. Autosomal recessive polycystic kidney disease, because adult-onset bilateral enlarged cystic kidneys with an affected parent usually follow recessive inheritance.

E. Acquired cystic kidney disease, because acquired dialysis-related cysts typically produce massively enlarged kidneys before kidney failure develops.

133. A 42-year-old patient with ADPKD has preserved eGFR but imaging and clinical features indicate rapid disease progression. Which disease-modifying therapy may be appropriate after discussing liver monitoring and aquaretic effects?

A. High-dose loop diuretics, because chronic medullary volume depletion suppresses cyst epithelial proliferation and has proven disease-modifying benefit.

B. Spironolactone, because mineralocorticoid blockade directly inhibits polycystin signaling and is approved to slow kidney enlargement in ADPKD.

C. Calcitriol, because vitamin D receptor activation prevents intracranial aneurysms and slows renal cyst growth independently of blood pressure.

D. Tolvaptan, a vasopressin V2-receptor antagonist that can slow kidney growth and GFR decline in selected adults at risk of rapid progression.

E. Desmopressin, because increasing V2-receptor stimulation reduces cyst fluid secretion and is the preferred disease-modifying therapy in rapidly progressive ADPKD.

134. A patient with ADPKD has a parent and sibling who both had subarachnoid hemorrhage from intracranial aneurysms. The patient has no neurologic symptoms. Which approach is most appropriate?

A. Do not screen because intracranial aneurysm risk in ADPKD is identical to the general population regardless of family history.

B. Perform annual lumbar puncture because cerebrospinal fluid xanthochromia is the preferred screening test before an aneurysm ruptures.

C. Start prophylactic anticoagulation because preventing thrombosis within an aneurysm is more effective than imaging surveillance.

D. Offer screening for intracranial aneurysm because the strong family history places the patient in a higher-risk group.

E. Remove both native kidneys because nephrectomy eliminates the systemic vascular defect responsible for intracranial aneurysm formation.

135. A patient taking tenofovir develops hypophosphatemia, normal-anion-gap metabolic acidosis, glucosuria with normal serum glucose, and proteinuria composed largely of low-molecular-weight proteins. What syndrome is present?

A. Fanconi syndrome from proximal tubular dysfunction causing urinary loss of bicarbonate, phosphate, glucose, and other solutes.

B. Minimal change disease, because podocyte foot-process effacement causes selective loss of glucose and phosphate without marked albuminuria.

C. Distal renal tubular acidosis, because collecting-duct acidification failure causes glucosuria and phosphaturia as defining distal tubular findings.

D. Type 4 renal tubular acidosis, because hypoaldosteronism causes proximal glucose and phosphate wasting together with hyperkalemia.

E. Nephrogenic diabetes insipidus, because impaired aquaporin response produces glucosuria and bicarbonate wasting in addition to polyuria.

136. A child has hypokalemic metabolic alkalosis, high renin and aldosterone, normal blood pressure, hypercalciuria, and nephrocalcinosis. Which inherited tubular disorder is most likely?

A. Bartter syndrome, reflecting impaired sodium chloride transport in the thick ascending limb with secondary hyperaldosteronism.

B. Type 4 renal tubular acidosis, because aldosterone deficiency produces hypokalemic alkalosis and hypercalciuria despite normal blood pressure.

C. Distal renal tubular acidosis, because failure of hydrogen secretion causes metabolic alkalosis and secondary hyperaldosteronism with nephrocalcinosis.

D. Gitelman syndrome, because distal convoluted tubule dysfunction usually causes hypercalciuria, nephrocalcinosis, and presentation in infancy with severe salt wasting.

E. Liddle syndrome, because constitutive ENaC activity causes high renin, high aldosterone, normal blood pressure, and renal calcium wasting.

137. An adult has chronic muscle cramps, potassium 2.9 mEq/L, metabolic alkalosis, low magnesium, low urinary calcium, and normal blood pressure. Which diagnosis is most likely?

A. Liddle syndrome, because epithelial sodium-channel activation produces low blood pressure, high renin, and hypocalciuria in young adults.

B. Primary aldosteronism, because autonomous aldosterone secretion commonly causes low magnesium and low urinary calcium with normal renin and blood pressure.

C. Gitelman syndrome from loss of thiazide-sensitive sodium chloride transport in the distal convoluted tubule.

D. Type 1 renal tubular acidosis, because distal acidification failure produces metabolic alkalosis with hypocalciuria rather than acidosis and stones.

E. Bartter syndrome, because thick ascending limb dysfunction usually causes hypocalciuria and hypomagnesemia with a milder adult presentation.

138. A patient develops edema, hypertension, creatinine rise, dysmorphic red blood cells, and red-cell casts. Which syndrome best describes this presentation?

A. An acute nephritic syndrome caused by glomerular inflammation with hematuria and impaired filtration.

B. A pure nephrotic syndrome, which is defined by red-cell casts and glomerular hematuria rather than heavy proteinuria and hypoalbuminemia.

C. Prerenal azotemia, because reduced perfusion characteristically produces dysmorphic erythrocytes and red-cell casts in the urine sediment.

D. Acute interstitial nephritis, because interstitial inflammation usually causes red-cell casts without pyuria or white-cell casts.

E. Lower urinary tract bleeding, because bladder or urethral blood commonly becomes dysmorphic and forms red-cell casts while traversing the nephron.

139. A patient has 7 g/day proteinuria, serum albumin 2.1 g/dL, edema, and hyperlipidemia. Which complication is especially associated with this syndrome?

A. Respiratory alkalosis from urinary bicarbonate retention, which develops as albumin loss lowers the plasma anion gap.

B. Severe neutropenia from urinary loss of mature neutrophils, which is the principal infectious complication of nephrotic syndrome.

C. Hypocoagulability from urinary fibrinogen loss, making spontaneous bleeding more common than thrombosis when serum albumin is low.

D. Venous thromboembolism from urinary loss of anticoagulant proteins, increased hepatic procoagulant synthesis, and other nephrotic changes.

E. Hypermagnesemia from increased proximal magnesium reabsorption, which is the defining electrolyte complication of nephrotic-range proteinuria.

140. A 28-year-old man develops gross hematuria within 24 hours of an upper respiratory infection. Complement levels are normal, and biopsy shows mesangial IgA deposition. What is the diagnosis?

A. Anti-GBM disease, which produces mesangial IgA deposition and recurrent mild hematuria rather than rapidly progressive crescentic nephritis.

B. IgA nephropathy, classically causing synpharyngitic hematuria with mesangial IgA-dominant immune deposits.

C. Minimal change disease, which causes synpharyngitic hematuria because podocyte foot-process effacement releases IgA into the mesangium.

D. Poststreptococcal glomerulonephritis, which typically causes hematuria within a day of pharyngitis and usually has normal complement levels.

E. Membranous nephropathy, which characteristically causes episodic gross hematuria linked to mucosal infections and mesangial IgA deposits.

141. A patient with biopsy-proven IgA nephropathy has persistent proteinuria despite optimized blood pressure control. Which management foundation is appropriate?

A. Avoid ACE inhibitors and ARBs because lowering intraglomerular pressure increases mesangial IgA deposition and accelerates progression.

B. Use long-term high-dose NSAIDs because cyclooxygenase inhibition reduces proteinuria and has proven kidney-protective effects in IgA nephropathy.

C. Maximize supportive kidney care with RAAS blockade, sodium reduction, and other indicated kidney-protective therapies such as SGLT2 inhibition before or alongside disease-targeted treatment.

D. Restrict dietary sodium only when nephrotic syndrome is present because sodium intake does not influence proteinuria or blood pressure in IgA nephropathy.

E. Begin chronic cyclophosphamide for every patient because IgA nephropathy is primarily a crescentic vasculitis regardless of proteinuria or GFR trajectory.

142. A patient with IgA nephropathy remains at high risk of progression despite optimized supportive care. Which newer disease-targeted option may be considered in an appropriate patient?

A. Indefinite high-dose systemic glucocorticoids for every patient because toxicity does not increase when eGFR is reduced or infection risk is high.

B. Routine plasma exchange because removing circulating IgA permanently eliminates mesangial deposition in nonrapidly progressive disease.

C. Targeted-release budesonide or another evidence-based IgA nephropathy therapy selected according to current risk, kidney function, and contraindications.

D. Chronic broad-spectrum antibiotics because occult bacterial infection is the primary driver of mesangial IgA deposition in all adults.

E. Bilateral tonsillectomy as universal first-line treatment because it has consistently replaced pharmacologic therapy across all regions and risk groups.

143. A patient has palpable purpura, abdominal pain, arthralgia, hematuria, and kidney biopsy showing IgA-dominant mesangial deposits. Which diagnosis is most likely?

A. Polyarteritis nodosa, because medium-vessel necrotizing vasculitis commonly causes glomerulonephritis with IgA-dominant immune deposits.

B. Microscopic polyangiitis, because ANCA-associated vasculitis typically causes palpable purpura together with IgA-dominant mesangial deposits on kidney biopsy.

C. Cryoglobulinemic vasculitis, because mixed cryoglobulins usually produce isolated IgA deposits without complement consumption or membranoproliferative features.

D. IgA vasculitis with kidney involvement, a systemic small-vessel IgA-mediated disease that can produce purpura, abdominal symptoms, arthralgia, and glomerulonephritis.

E. Anti-GBM disease, because linear IgG deposition along glomerular basement membranes is clinically indistinguishable from mesangial IgA deposition.

144. A patient has nephrotic syndrome and strongly positive anti-PLA2R antibodies. Kidney biopsy shows diffuse subepithelial immune deposits. Which diagnosis is most likely?

A. C3 glomerulopathy, because alternative complement activation produces subepithelial PLA2R-containing deposits with nephrotic syndrome.

B. Diabetic nephropathy, because advanced glycation exposes PLA2R antigens and commonly produces high circulating anti-PLA2R titers.

C. Primary membranous nephropathy associated with phospholipase A2 receptor autoantibodies.

D. Minimal change disease, because anti-PLA2R antibodies are directed against podocyte foot processes and define steroid-responsive minimal change nephrosis.

E. Primary FSGS, because subepithelial immune deposits and circulating PLA2R antibodies are typical causes of segmental sclerosis.

145. A 68-year-old patient develops membranous nephropathy but is negative for PLA2R antibodies and has unintentional weight loss and iron-deficiency anemia. What is the most important next consideration?

A. Diagnose minimal change disease because negative PLA2R testing excludes membranous nephropathy despite biopsy findings.

B. Ignore systemic symptoms because membranous nephropathy is never associated with cancer, infections, autoimmune disease, or medications.

C. Perform bilateral nephrectomy because secondary membranous nephropathy cannot remit unless the kidneys producing the target antigen are removed.

D. Start high-dose glucocorticoids immediately because malignancy screening is unnecessary when nephrotic syndrome is severe.

E. Evaluate for a secondary cause, including age-appropriate malignancy assessment, rather than assuming all membranous nephropathy is primary.

146. A patient develops sudden nephrotic syndrome after starting an NSAID. Biopsy shows normal glomeruli by light microscopy with diffuse podocyte foot-process effacement. What is the diagnosis?

A. Membranous nephropathy, because subepithelial immune deposits are required to produce podocyte effacement after NSAID exposure.

B. Minimal change disease, which can be associated with NSAID exposure and causes diffuse podocyte foot-process effacement.

C. FSGS, because segmental scars must be visible by light microscopy in every case before nephrotic-range proteinuria can occur.

D. Amyloidosis, because Congo-red-positive fibrils are the expected biopsy finding in abrupt NSAID-associated nephrotic syndrome.

E. Diabetic nephropathy, because diffuse podocyte effacement without mesangial expansion is the classic earliest lesion of diabetes-related nephrosis.

147. An adult has nephrotic syndrome with diffuse foot-process effacement but no secondary cause. Proteinuria falls rapidly after glucocorticoid therapy. Which diagnosis is most likely?

A. Alport syndrome, because collagen IV defects are immune mediated and respond rapidly to systemic glucocorticoids in adult nephrotic syndrome.

B. Primary membranous nephropathy, because membranous lesions usually disappear rapidly after glucocorticoid monotherapy and never require antibody testing.

C. Amyloidosis, because amyloid fibrils are highly steroid responsive and cause complete proteinuria remission within days.

D. Diabetic nephropathy, because longstanding diabetic nodular sclerosis typically produces abrupt complete remission with glucocorticoids.

E. Primary minimal change disease, which is typically steroid responsive even in adults, although response may take longer than in children.

148. A patient has nephrotic syndrome, diffuse foot-process effacement, and focal segmental glomerular scars without obesity, reduced nephron mass, viral infection, or drug exposure. Which pathophysiologic category is most likely?

A. Membranous nephropathy, because segmental scars are the defining late stage of subepithelial immune-complex deposition.

B. C3 glomerulopathy, because complement dysregulation is the usual cause of segmental scars with nephrotic syndrome and diffuse effacement.

C. Adaptive secondary FSGS, because primary forms usually have subnephrotic proteinuria and only patchy foot-process effacement.

D. Primary FSGS, particularly when nephrotic syndrome and diffuse foot-process effacement suggest a circulating podocyte injury process.

E. Minimal change disease, because any diffuse foot-process effacement excludes focal segmental sclerosis seen on light microscopy.

149. A patient with severe obesity and reduced nephron mass has 2.5 g/day proteinuria, focal segmental sclerosis, and only patchy foot-process effacement. What is the best classification?

A. Minimal change disease because patchy foot-process effacement proves the absence of any true segmental sclerotic lesion.

B. Membranous nephropathy because subnephrotic proteinuria with obesity is usually caused by anti-PLA2R autoantibodies.

C. Anti-GBM disease because reduced nephron mass causes linear IgG deposition and segmental scars without hematuria.

D. Primary FSGS requiring high-dose glucocorticoids because obesity and reduced nephron mass are not recognized secondary causes of segmental sclerosis.

E. Secondary adaptive FSGS from glomerular hyperfiltration, for which treatment focuses on the underlying hemodynamic stress rather than routine immunosuppression.

150. A patient of recent African ancestry has FSGS and progressive CKD without a clear secondary cause. Which genetic factor may influence risk and prognosis?

A. PKD1 variants, because polycystin-1 mutations are the most common genetic cause of primary FSGS in adults of African ancestry.

B. CFTR variants, because chloride-channel dysfunction produces podocyte sclerosis and nephrotic syndrome before pulmonary disease develops.

C. HFE variants, because iron-loading mutations are the principal inherited determinant of FSGS progression in populations with African ancestry.

D. Factor V Leiden, because inherited hypercoagulability directly causes collapsing FSGS and determines response to glucocorticoids.

E. High-risk APOL1 variants, which are associated with increased risk of several nondiabetic kidney diseases including FSGS in people with relevant ancestry.

151. A child develops cola-colored urine and edema 2 weeks after streptococcal pharyngitis. C3 is low and biopsy shows granular immune deposits with subepithelial humps. What is the diagnosis?

A. Membranous nephropathy, because subepithelial humps are persistent PLA2R-containing deposits and complement is typically low after infection.

B. IgA nephropathy, which classically begins after a 1- to 3-week latent period and usually causes low C3 with subepithelial humps.

C. Poststreptococcal glomerulonephritis, an infection-related immune-complex nephritis with transient complement consumption.

D. Anti-GBM disease, because linear antibodies against basement membrane form granular subepithelial humps after streptococcal infection.

E. Minimal change disease, because podocyte effacement after pharyngitis produces low complement and red-cell casts as the defining features.

152. An older patient with diabetes has active Staphylococcus aureus infection, AKI, hematuria, low C3, and biopsy showing IgA-dominant immune-complex glomerulonephritis. Which diagnosis is most likely?

A. Staphylococcus-associated infection-related glomerulonephritis, which can be IgA dominant in older adults with ongoing infection.

B. Minimal change disease, because staphylococcal superantigens cause podocyte effacement with low complement and nephritic sediment.

C. Alport syndrome, because chronic collagen IV defects can first present during bacteremia with low complement and IgA-dominant deposits.

D. Primary IgA nephropathy, because concurrent staphylococcal infection and low complement strongly favor the primary mesangial disease rather than infection-related GN.

E. Anti-GBM disease, because bacterial infection commonly triggers linear IgG deposition and IgA-dominant immune complexes in glomeruli.

153. A patient has hematuria, proteinuria, low C3, and a membranoproliferative pattern on biopsy. Which principle is most appropriate?

A. The pattern proves diabetic nephropathy because mesangial expansion from diabetes is the only cause of double-contour basement membranes.

B. Low complement excludes complement-mediated disease because complement consumption occurs only in immune-complex disorders.

C. All MPGN requires immediate cyclophosphamide before etiologic evaluation because the biopsy pattern predicts rapid crescentic progression in every patient.

D. MPGN is a single disease with one treatment, so serologic, infectious, and hematologic evaluation is unnecessary once the pattern is seen.

E. The membranoproliferative pattern is a pattern of injury, so evaluation should seek immune-complex causes, monoclonal gammopathy, infection, and complement dysregulation.

154. A patient has persistent low C3, glomerular C3-dominant deposits with little immunoglobulin, and a membranoproliferative pattern. Which mechanism is most likely?

A. Alternative complement pathway dysregulation causing C3 glomerulopathy, which fits persistent hypocomplementemia and C3-dominant glomerular deposits.

B. Anti-GBM autoantibodies, because linear IgG binding activates alternative complement and leaves only C3 deposits after immunoglobulin is cleared.

C. Primary membranous nephropathy, because PLA2R antibodies produce C3-dominant mesangial deposits with little immunoglobulin.

D. Minimal change disease, because podocyte foot-process effacement activates alternative complement and causes low C3 with dense deposits.

E. Diabetic nephropathy, because advanced glycation chronically consumes C3 and produces C3-dominant immune deposits in the mesangium.

155. A patient has rapidly progressive glomerulonephritis and pulmonary hemorrhage. Kidney biopsy shows crescentic GN with linear IgG along the glomerular basement membrane. What is the diagnosis?

A. Membranous nephropathy, because subepithelial immune complexes form a continuous linear IgG band and rapidly progressive crescentic nephritis.

B. ANCA-associated vasculitis, because pauci-immune GN is defined by intense linear IgG staining along the glomerular basement membrane.

C. Anti-GBM disease, causing antibodies against basement-membrane antigens in kidney and sometimes lung.

D. Lupus nephritis, because full-house immune-complex staining typically appears as smooth linear IgG without mesangial or granular deposits.

E. IgA vasculitis, because mesangial IgA disease commonly produces linear IgG and pulmonary hemorrhage in adults.

156. A patient has hematuria, AKI, pulmonary nodules, positive PR3-ANCA, and kidney biopsy showing necrotizing crescentic GN with minimal immune deposits. Which diagnosis is most likely?

A. Postinfectious GN, because bacterial immune complexes rapidly disappear from biopsy and leave a pauci-immune crescentic pattern with PR3-ANCA.

B. Membranous nephropathy, because subepithelial PLA2R deposits commonly cause pulmonary nodules and necrotizing glomerular capillary lesions.

C. Anti-GBM disease, because anti-basement-membrane antibodies typically produce pauci-immune biopsy findings without linear immunoglobulin deposition.

D. Lupus nephritis, because immune-complex deposition is usually absent and PR3-ANCA is the defining serologic marker of proliferative lupus disease.

E. ANCA-associated pauci-immune glomerulonephritis as part of granulomatosis with polyangiitis or a related small-vessel vasculitis.

157. A patient with new ANCA-associated vasculitis has active kidney disease and is at high risk from glucocorticoid toxicity. Which newer agent can reduce glucocorticoid exposure in an appropriate induction regimen?

A. Belimumab as sole induction therapy, because B-cell activating factor blockade has replaced standard remission-induction regimens for pauci-immune GN.

B. Avacopan, a C5a receptor inhibitor used as an alternative to part of the glucocorticoid burden in selected ANCA-associated vasculitis.

C. Tolvaptan, because vasopressin V2 antagonism reduces crescent formation and is approved to spare glucocorticoids in ANCA vasculitis.

D. Finerenone, because nonsteroidal mineralocorticoid blockade directly inhibits neutrophil C5a signaling in ANCA-associated vasculitis.

E. Eculizumab as routine first-line monotherapy, because terminal complement blockade replaces rituximab or cyclophosphamide in all ANCA-associated vasculitis.

158. A patient with ANCA-associated vasculitis has severe kidney failure and pulmonary hemorrhage. Which statement about plasma exchange is most accurate?

A. Plasma exchange is mandatory for all ANCA-positive patients regardless of organ involvement because ANCA concentration directly determines kidney recovery.

B. Plasma exchange is contraindicated whenever creatinine is elevated because removing plasma worsens kidney perfusion and increases dialysis dependence.

C. Plasma exchange replaces immunosuppressive induction therapy and therefore eliminates the need for rituximab, cyclophosphamide, or glucocorticoid-sparing regimens.

D. Plasma exchange is useful only in isolated upper-airway granulomatosis because severe kidney and lung disease do not respond to antibody removal.

E. Plasma exchange is not routine for every patient but may be considered in selected severe presentations, including certain patients with advanced kidney disease or hypoxemic pulmonary hemorrhage.

159. A patient with systemic lupus erythematosus has active urine sediment, proteinuria, low complement, and kidney biopsy showing class IV diffuse proliferative lupus nephritis. Which treatment principle is appropriate?

A. Use calcitriol monotherapy because vitamin D receptor activation is the established first-line induction treatment for class III and IV lupus nephritis.

B. Use induction with glucocorticoids plus a proven immunosuppressive regimen such as mycophenolic acid analogs or low-dose intravenous cyclophosphamide, with selected add-on agents when appropriate.

C. Treat with an ACE inhibitor alone because proliferative lupus nephritis is a hemodynamic proteinuric disorder and does not require immunosuppression.

D. Use high-dose NSAIDs as primary therapy because prostaglandin inhibition suppresses immune-complex deposition more effectively than mycophenolate or cyclophosphamide.

E. Perform immediate nephrectomy because diffuse proliferative lesions are irreversible and immunosuppressive therapy cannot preserve kidney function.

160. A patient with lupus has pure class V membranous lupus nephritis and nephrotic-range proteinuria despite optimized supportive care. Which principle is most appropriate?

A. Immunosuppressive therapy is considered when class V lupus nephritis causes significant proteinuria, especially nephrotic syndrome, while RAAS blockade and other supportive care continue.

B. Stop hydroxychloroquine routinely because it increases proteinuria and thrombosis risk in lupus nephritis.

C. Use plasma exchange as universal first-line therapy because circulating immune complexes must be removed permanently in every class V case.

D. Pure class V lupus nephritis never requires immunosuppression because membranous immune deposits cannot respond to therapy.

E. Treat only with antibiotics because class V disease is caused by occult infection rather than systemic autoimmunity.

161. A patient with chronic hepatitis C has purpura, arthralgia, neuropathy, low C4, positive rheumatoid factor, and membranoproliferative glomerulonephritis. Which diagnosis is most likely?

A. Anti-GBM disease, because hepatitis C commonly causes linear IgG deposition together with cryoglobulin precipitation in small vessels.

B. Mixed cryoglobulinemic vasculitis with immune-complex glomerulonephritis related to hepatitis C.

C. Minimal change disease, because viral infection triggers podocyte effacement that explains low C4, purpura, and rheumatoid factor positivity.

D. ADPKD, because chronic hepatitis C promotes cyst growth and systemic vasculitis through polycystin-mediated complement activation.

E. ANCA-associated vasculitis, because low C4 and rheumatoid factor are typical markers of pauci-immune PR3-ANCA disease in hepatitis C.

162. A patient has nephrotic proteinuria and biopsy showing randomly arranged fibrils that are Congo red negative and composed of immunoglobulin. Which diagnosis is most likely?

A. AL amyloidosis, because amyloid fibrils are characteristically Congo red negative and lack apple-green birefringence.

B. Minimal change disease, because podocyte effacement produces visible immunoglobulin fibrils on electron microscopy without Congo red staining.

C. Diabetic nephropathy, because advanced mesangial matrix forms randomly arranged immunoglobulin fibrils that are Congo red negative.

D. Fibrillary glomerulonephritis, an immune-deposit disease with Congo-red-negative fibrils in the glomeruli.

E. Anti-GBM disease, because linear IgG deposits polymerize into fibrils and produce nephrotic proteinuria without hematuria.

163. A patient has nephrotic syndrome, restrictive cardiomyopathy, macroglossia, and biopsy deposits with apple-green birefringence after Congo red staining. Which diagnosis is most likely?

A. Postinfectious glomerulonephritis, because subepithelial humps become Congo-red positive as infection resolves and can infiltrate the myocardium.

B. Fibrillary glomerulonephritis, because fibrillary GN is defined by Congo-red-positive deposits with apple-green birefringence and systemic macroglossia.

C. Minimal change disease, because podocyte effacement produces Congo-red-positive basement membranes when proteinuria is severe.

D. Systemic amyloidosis involving the kidney, with fibril type requiring further characterization because treatment depends on the precursor protein.

E. IgA nephropathy, because mesangial IgA deposits form amyloid fibrils and commonly cause restrictive cardiomyopathy.

164. A patient with a small plasma-cell clone has proteinuria and kidney biopsy showing monoclonal light-chain deposits causing glomerular injury, but criteria for multiple myeloma are not met. Which concept applies?

A. Monoclonal gammopathy of renal significance, in which a small clone causes clinically important kidney disease and may require clone-directed therapy.

B. Minimal change disease, because small plasma-cell clones produce podocyte injury without any monoclonal deposits in the kidney.

C. Diabetic nephropathy, because monoclonal deposits in a patient with a plasma-cell clone should be ignored if diabetes is also present.

D. Monoclonal gammopathy of undetermined significance only, because kidney injury cannot be attributed to a clone unless full multiple-myeloma criteria are met.

E. Primary membranous nephropathy, because monoclonal light-chain deposition is a benign incidental finding in patients with nephrotic proteinuria.

165. A patient has fever, neurologic changes, thrombocytopenia, microangiopathic hemolytic anemia, and AKI. ADAMTS13 activity is severely deficient. Which diagnosis is most likely?

A. Immune thrombotic thrombocytopenic purpura, which is strongly associated with severe ADAMTS13 deficiency.

B. Shiga toxin-associated HUS, because severe ADAMTS13 deficiency is caused by bacterial toxin and is expected after bloody diarrhea.

C. Malignant hypertension alone, because hypertensive TMA routinely lowers ADAMTS13 activity to severely deficient levels.

D. Disseminated intravascular coagulation, because consumption of coagulation factors selectively destroys ADAMTS13 while leaving other proteases intact.

E. Complement-mediated atypical hemolytic uremic syndrome, because severe ADAMTS13 deficiency is the defining laboratory abnormality of complement-mediated TMA.

166. A patient has recurrent thrombotic microangiopathy, AKI, thrombocytopenia, and hemolysis without severe ADAMTS13 deficiency or Shiga toxin. Genetic testing reveals complement-regulatory dysfunction. Which therapy is most specific?

A. Plasma exchange alone as definitive lifelong therapy because complement dysregulation cannot be treated pharmacologically.

B. High-dose aspirin monotherapy because platelet cyclooxygenase inhibition blocks the complement cascade responsible for endothelial injury.

C. Terminal complement inhibition with a C5-targeted agent in complement-mediated atypical hemolytic uremic syndrome.

D. Desmopressin because raising von Willebrand factor release corrects the underlying complement regulatory defect and improves kidney recovery.

E. Tolvaptan because vasopressin V2-receptor antagonism prevents terminal complement assembly on glomerular endothelial cells.

167. Minutes after reperfusion of a kidney allograft, the graft becomes mottled and anuric. Biopsy shows widespread microvascular thrombosis and neutrophils, and a pretransplant crossmatch is retrospectively found to have been strongly positive. What is the most likely diagnosis?

A. Recurrent focal segmental glomerulosclerosis, which causes immediate graft mottling and diffuse arterial thrombosis rather than isolated heavy proteinuria.

B. Acute T-cell-mediated rejection, which usually develops within minutes because recipient T cells directly thrombose the graft microcirculation at reperfusion.

C. Hyperacute antibody-mediated rejection caused by preformed recipient antibodies against donor antigens.

D. Delayed graft function from ischemic acute tubular injury, which characteristically causes immediate diffuse microvascular thrombosis in a strongly positive crossmatch setting.

E. BK polyomavirus nephropathy, which typically appears at reperfusion because latent donor virus rapidly destroys the graft microvasculature.

168. Three months after kidney transplantation, serum creatinine rises from 1.2 to 2.1 mg/dL. Ultrasound shows no obstruction, tacrolimus trough is therapeutic, and biopsy demonstrates interstitial inflammation with tubulitis but no microvascular inflammation. What is the best initial treatment?

A. High-dose corticosteroid therapy for acute T-cell-mediated rejection, with escalation if the rejection is steroid resistant.

B. Reduce all immunosuppression, because acute T-cell-mediated rejection is most effectively treated by allowing endogenous immune regulation to recover.

C. Start antiviral therapy for BK virus without further testing, because tubulitis by itself is diagnostic of BK nephropathy even when viral evidence is absent.

D. Plasma exchange alone, because interstitial inflammation and tubulitis without antibody-mediated features indicate circulating donor-specific antibody as the primary mechanism.

E. Immediate graft nephrectomy, because biopsy-proven acute cellular rejection is generally irreversible once serum creatinine has doubled from baseline.

169. Six months after kidney transplantation, a patient develops rising creatinine and proteinuria. Biopsy shows glomerulitis and peritubular capillaritis with C4d staining, and circulating donor-specific HLA antibodies are detected. Which process is most likely?

A. Calcineurin-inhibitor toxicity, because tacrolimus exposure produces donor-specific antibodies and diffuse peritubular capillary C4d deposition.

B. Active antibody-mediated rejection driven by donor-specific antibodies and microvascular endothelial injury.

C. Recurrent IgA nephropathy, because mesangial IgA deposition is expected to produce transplant glomerulitis with donor-specific HLA antibodies.

D. BK polyomavirus nephropathy, because viral tubular injury routinely causes C4d-positive peritubular capillaries and newly formed donor-specific antibodies.

E. Acute T-cell-mediated rejection, because C4d deposition and donor-specific antibodies are defining features of isolated cellular tubulitis.

170. A kidney transplant recipient has slowly progressive graft dysfunction and proteinuria several years after transplantation. Biopsy shows transplant glomerulopathy with duplication of glomerular basement membranes and chronic microvascular injury. Donor-specific antibodies are present. Which diagnosis best fits?

A. Isolated acute tubular injury, because tubular epithelial damage alone produces double contours of glomerular basement membranes and persistent donor-specific antibodies.

B. Acute pyelonephritis, because bacterial infection typically creates transplant glomerulopathy and chronic donor-specific antibody-mediated endothelial remodeling.

C. Recurrent minimal change disease, because podocyte foot-process effacement produces the characteristic double-contour glomerular capillary walls seen here.

D. Chronic active antibody-mediated rejection causing progressive allograft microvascular remodeling.

E. Tacrolimus withdrawal syndrome, because stopping a calcineurin inhibitor directly duplicates glomerular basement membranes without an alloimmune mechanism.

171. A kidney transplant recipient develops an abrupt rise in creatinine after several days of diarrhea. Tacrolimus trough is markedly elevated, potassium is 5.9 mEq/L, and magnesium is low. Urinalysis is bland. What is the most likely cause of the kidney dysfunction?

A. Acute calcineurin-inhibitor nephrotoxicity from excessive tacrolimus exposure causing renal vasoconstriction.

B. Transplant renal artery thrombosis, because vascular occlusion usually produces a high tacrolimus trough together with magnesium wasting and preserved urine sediment.

C. Acute T-cell-mediated rejection, because a high tacrolimus level directly proves that alloimmune cellular rejection is breaking through adequate immunosuppression.

D. BK nephropathy, because BK virus predictably causes abrupt hyperkalemia and hypomagnesemia whenever tacrolimus levels rise during diarrhea.

E. Recurrent glomerulonephritis, because a bland urinalysis with supratherapeutic tacrolimus is typical of recurrent immune-complex glomerular disease.

172. A kidney transplant recipient taking tacrolimus begins voriconazole for an invasive fungal infection. Two days later the tacrolimus level is markedly elevated. What explains this interaction?

A. Tacrolimus accelerates voriconazole clearance but is not itself affected, so no change in tacrolimus monitoring is needed when the drugs are combined.

B. The drugs interact only by increasing urine potassium loss, so tacrolimus blood levels are not expected to change despite combined administration.

C. Voriconazole induces tacrolimus metabolism, so tacrolimus dosing must generally be increased to prevent abrupt underimmunosuppression.

D. Voriconazole blocks gastrointestinal tacrolimus absorption, so the elevated trough must represent laboratory interference rather than a true pharmacokinetic interaction.

E. Voriconazole inhibits tacrolimus metabolism, so the tacrolimus dose generally must be reduced with close trough-level monitoring.

173. Eight months after kidney transplantation, creatinine rises gradually. Plasma BK viral load is persistently high and biopsy shows tubulointerstitial inflammation with viral cytopathic changes and positive polyomavirus staining. What is the cornerstone of management?

A. Reduce the intensity of immunosuppression in a structured fashion while monitoring BK viral load and graft function.

B. Begin high-dose corticosteroids alone, because viral cytopathic changes indicate an immune-mediated rejection syndrome that responds to augmented glucocorticoids.

C. Treat with prolonged broad-spectrum antibacterial therapy, because BK polyomavirus is eradicated reliably by agents used for bacterial pyelonephritis.

D. Perform urgent graft nephrectomy in every case, because established BK viremia cannot improve after adjustment of immunosuppressive therapy.

E. Increase tacrolimus and mycophenolate immediately, because BK nephropathy reflects insufficient suppression of alloimmune tubulitis rather than viral replication.

174. Four months after kidney transplantation, a recipient has fever, malaise, leukopenia, and transaminitis. CMV PCR is strongly positive. Which treatment is appropriate for clinically significant CMV disease in a patient who can take oral medication and has no life-threatening disease?

A. Acyclovir at standard herpes-simplex dosing, because CMV replication is reliably suppressed by the same oral doses used for mucocutaneous herpes infections.

B. Trimethoprim-sulfamethoxazole, because CMV syndrome is a Pneumocystis-spectrum opportunistic infection responsive to folate antagonism.

C. Valganciclovir with dose adjustment for kidney function, while reassessing the net state of immunosuppression.

D. High-dose methylprednisolone alone, because leukopenia and transaminitis after transplantation are presumptive manifestations of cellular rejection rather than viral disease.

E. No antiviral therapy, because detectable CMV DNA after transplantation represents asymptomatic latency even when fever and cytopenias are present.

175. A kidney transplant recipient is discharged on tacrolimus, mycophenolate, and prednisone. Which medication is commonly used to prevent Pneumocystis jirovecii pneumonia during the high-risk post-transplant period?

A. Rifampin daily, because mycobacterial prophylaxis also prevents Pneumocystis while lowering calcineurin-inhibitor exposure.

B. Trimethoprim-sulfamethoxazole, which also provides protection against several other opportunistic infections.

C. Oral nystatin alone, because suppression of Candida in the oropharynx prevents Pneumocystis infection in the lung.

D. Valganciclovir alone, because CMV-active antivirals are also first-line prophylaxis against Pneumocystis jirovecii.

E. Azithromycin once weekly, because macrolide prophylaxis is the standard preventive regimen for Pneumocystis after kidney transplantation.

176. A kidney transplant recipient develops fever, lymphadenopathy, weight loss, and an extranodal mass. EBV DNA is high, and biopsy shows a monomorphic B-cell proliferation. What is the most likely diagnosis?

A. Calcineurin-inhibitor nephrotoxicity, because elevated immunosuppression directly causes clonal B-cell lesions without an oncogenic viral contribution.

B. Recurrent renal-cell carcinoma, because EBV DNA elevation and B-cell histology are expected findings when native-kidney cancer recurs after transplantation.

C. Post-transplant lymphoproliferative disorder, often driven by EBV under the pressure of immunosuppression.

D. BK polyomavirus nephropathy, because BK infection primarily produces extranodal B-cell tumors rather than tubular injury in kidney allografts.

E. Acute T-cell-mediated rejection, because rejection typically presents with generalized lymphadenopathy and a monoclonal B-cell mass outside the graft.

177. An EBV-seronegative adult is being considered for belatacept-based maintenance immunosuppression after kidney transplantation. What is the key safety issue?

A. Belatacept must be avoided in all living-donor recipients because costimulation blockade is effective only after deceased-donor transplantation.

B. Belatacept is contraindicated whenever tacrolimus has been used previously because prior calcineurin-inhibitor exposure permanently eliminates its immunologic effect.

C. Belatacept should not be used in an EBV-seronegative recipient because of the markedly increased risk of post-transplant lymphoproliferative disorder.

D. Belatacept can be used regardless of EBV status if prednisone is stopped, because glucocorticoid withdrawal removes the excess PTLD risk.

E. Belatacept is contraindicated only in CMV-seronegative recipients because CMV status, rather than EBV status, determines lymphoma risk with costimulation blockade.

178. A kidney transplant candidate has high immunologic risk because of prior transplants and strong sensitization. Which induction approach is generally more potent than an interleukin-2 receptor antagonist?

A. A lymphocyte-depleting induction agent such as rabbit antithymocyte globulin, selected after balancing immunologic and infectious risks.

B. Basiliximab is always more potent than lymphocyte-depleting induction because it eliminates mature T cells rather than blocking interleukin-2 signaling.

C. No induction therapy, because sensitized recipients have the lowest rejection risk and therefore benefit least from perioperative immunosuppression.

D. Mycophenolate alone as induction, because antimetabolite maintenance therapy provides immediate lymphocyte depletion equivalent to antithymocyte globulin.

E. Prednisone monotherapy, because glucocorticoids alone provide the same rejection prevention as biologic induction in highly sensitized kidney recipients.

179. A stable kidney transplant recipient requires long-term maintenance immunosuppression. Which combination is a common contemporary backbone when no contraindication exists?

A. A calcineurin inhibitor, usually tacrolimus, plus an antimetabolite such as mycophenolate, with corticosteroid use individualized.

B. Belatacept plus live-virus vaccination, because live vaccines enhance tolerance and are required to make costimulation blockade effective after transplantation.

C. Acyclovir plus trimethoprim-sulfamethoxazole without an immunosuppressive agent, because antimicrobial prophylaxis provides sufficient protection from allograft rejection.

D. High-dose prednisone alone indefinitely, because combination immunosuppression adds toxicity without reducing acute or chronic rejection risk.

E. Sirolimus plus amphotericin B as standard therapy, because antifungal nephrotoxicity is needed to counter the hyperfiltration caused by mTOR inhibition.

180. A deceased-donor kidney transplant produces little urine during the first postoperative week, and dialysis is needed for hyperkalemia. Doppler ultrasound shows preserved perfusion and no obstruction. What is the most common explanation?

A. Chronic antibody-mediated rejection is the expected cause of oliguria in the first days because transplant glomerulopathy develops immediately after reperfusion.

B. BK nephropathy is the most common explanation because BK virus routinely causes severe tubular injury within hours of transplantation.

C. Hyperacute rejection is proven by any dialysis requirement during the first week, even when perfusion is preserved and crossmatch testing was negative.

D. Delayed graft function from ischemia-reperfusion injury and acute tubular injury, while rejection and other causes still require consideration.

E. Recurrent IgA nephropathy is the usual cause of immediate post-transplant oliguria and typically requires dialysis before mesangial deposits can be detected.

181. Six months after kidney transplantation, a patient develops difficult-to-control hypertension, a bruit over the allograft, hypokalemia, and a rise in creatinine after starting an ACE inhibitor. Which diagnosis is most likely?

A. BK nephropathy, because viral tubular injury directly activates renin and produces a focal vascular bruit over the transplant.

B. Ureteral obstruction, because hydronephrosis commonly causes hyperreninemic hypertension and a systolic bruit over the iliac fossa graft.

C. Recurrent minimal change disease, because podocyte injury causes renovascular hypertension with a bruit even in the absence of nephrotic proteinuria.

D. Renal vein thrombosis, because chronic venous obstruction typically causes an audible arterial bruit, hypokalemia, and an ACE-inhibitor-associated creatinine rise.

E. Transplant renal artery stenosis causing renovascular hypertension and functionally important reduction in graft perfusion.

182. Two weeks after kidney transplantation, creatinine rises and urine output falls. Ultrasound shows new hydronephrosis with a dilated transplant ureter, while perfusion is intact. What is the best next principle?

A. Relieve the urinary obstruction promptly and identify the mechanical cause, such as ureteral edema, stricture, clot, or leak-related compression.

B. Increase tacrolimus because hydronephrosis is a classic imaging manifestation of underimmunosuppression and acute cellular rejection.

C. Observe without intervention because early transplant hydronephrosis is physiologic and cannot compromise graft function during the first postoperative month.

D. Begin plasma exchange because urinary tract dilation indicates antibody-mediated swelling of the peritubular capillaries rather than a mechanical problem.

E. Perform graft nephrectomy immediately because any early ureteral obstruction implies irreversible ischemic injury of the transplanted kidney.

183. A patient with primary focal segmental glomerulosclerosis receives a kidney transplant. Within days, urine protein excretion rises to 12 g/day despite good graft perfusion. What is the most likely explanation?

A. Transplant renal artery stenosis, because reduced perfusion characteristically produces nephrotic-range proteinuria within hours of transplantation without severe hypertension.

B. Recurrent diabetic nephropathy, because diabetic nodular glomerulosclerosis typically recurs within days and produces immediate massive proteinuria after transplantation.

C. Chronic calcineurin-inhibitor toxicity, because arteriolar hyalinosis develops immediately after transplantation and is the usual cause of abrupt nephrotic syndrome.

D. BK nephropathy, because BK infection primarily presents as sudden massive proteinuria during the first several postoperative days.

E. Early recurrence of focal segmental glomerulosclerosis in the allograft, often attributed to a circulating permeability factor.

184. A kidney transplant recipient with a history of IgA nephropathy develops microscopic hematuria and proteinuria several years after transplantation. Biopsy shows dominant mesangial IgA deposits. What is the most likely diagnosis?

A. Recurrent IgA nephropathy in the kidney allograft, supported by the prior diagnosis plus new hematuria, proteinuria, and dominant mesangial IgA deposition.

B. Calcineurin-inhibitor toxicity, because chronic tacrolimus exposure produces dominant mesangial IgA deposits rather than arteriolar and striped interstitial changes.

C. De novo membranous nephropathy, because subepithelial immune deposits are routinely reported as dominant mesangial IgA on immunofluorescence.

D. Acute antibody-mediated rejection, because mesangial IgA is a defining alloimmune marker when donor-specific antibodies are absent.

E. BK nephropathy, because polyomavirus infection characteristically deposits IgA in the mesangium and causes recurrent hematuria years later.

185. A kidney transplant recipient develops new diabetes several months after transplantation. Which immunosuppressive agents are especially associated with this complication?

A. Tacrolimus and glucocorticoids, which can both impair glucose regulation after transplantation.

B. Belatacept and valganciclovir, because costimulation blockade and antiviral therapy routinely induce severe insulin resistance in most recipients.

C. Azathioprine and trimethoprim-sulfamethoxazole, because antimetabolite and Pneumocystis prophylaxis are the dominant diabetogenic exposures after transplantation.

D. Sirolimus and nystatin only, because calcineurin inhibitors and corticosteroids have no clinically important effect on post-transplant glycemia.

E. Mycophenolate and basiliximab, because both directly cause pancreatic beta-cell destruction and are the principal drivers of post-transplant diabetes.

186. A kidney transplant recipient taking tacrolimus has persistent mild hyperkalemia and hypomagnesemia despite stable graft function. Which medication effect best explains this pattern?

A. Tacrolimus activates distal sodium-potassium exchange so strongly that it predictably causes hypokalemia together with hypermagnesemia.

B. Mycophenolate blocks renal magnesium excretion and therefore causes hypermagnesemia rather than the electrolyte pattern described.

C. Calcineurin-inhibitor effects on distal tubular electrolyte handling can promote hyperkalemia and renal magnesium wasting.

D. Prednisone selectively suppresses aldosterone secretion and is the sole explanation for hyperkalemia in otherwise stable kidney transplant recipients.

E. Valganciclovir directly stimulates renal magnesium reabsorption, so hypomagnesemia indicates that the drug is not being absorbed.

187. A kidney transplant recipient has had several actinic keratoses and one cutaneous squamous-cell carcinoma. Which long-term care principle is most appropriate?

A. Begin chronic ultraviolet-light therapy because additional UV exposure lowers the incidence of squamous-cell carcinoma in immunosuppressed patients.

B. Intensify sun protection and dermatologic surveillance, and reassess the immunosuppressive regimen in light of the recipient's malignancy risk.

C. Stop all immunosuppression permanently after the first skin cancer because rejection risk disappears once the allograft has functioned for more than one year.

D. Avoid dermatology follow-up because immunosuppression reduces inflammatory skin changes and makes recurrent cutaneous malignancy uncommon.

E. Use routine live-virus vaccination to stimulate antitumor immunity, because live vaccines are the preferred cancer-prevention strategy in transplant recipients.

188. A stable kidney transplant recipient asks about vaccination. Which recommendation is most appropriate?

A. Give live vaccines whenever tacrolimus trough is therapeutic because calcineurin inhibition prevents vaccine-strain dissemination.

B. Delay all inactivated vaccines until immunosuppression has been completely discontinued, even if that requires waiting for graft failure.

C. Avoid all vaccines after transplantation because any antigen exposure predictably triggers acute allograft rejection.

D. Use indicated inactivated or non-live vaccines, while generally avoiding live attenuated vaccines during ongoing post-transplant immunosuppression.

E. Prefer live attenuated vaccines because replication is necessary to overcome the reduced immune response caused by maintenance immunosuppression.

189. A kidney transplant recipient taking mycophenolate wants to become pregnant. Which medication issue is most important during preconception planning?

A. Tacrolimus must always be stopped before conception because calcineurin inhibitors are absolutely contraindicated in every pregnancy after transplantation.

B. Prednisone is the only immunosuppressive drug that can ever be used in pregnancy, so all other maintenance agents should be discontinued abruptly.

C. Mycophenolate is teratogenic and should be replaced with a pregnancy-compatible alternative before conception under transplant-specialist supervision.

D. No medication changes are needed before conception because placental transfer of antimetabolites does not occur during the first trimester.

E. Mycophenolate is preferred throughout pregnancy because it lowers congenital-malformation risk compared with azathioprine in transplant recipients.

190. A highly sensitized kidney transplant candidate has a potential donor. The candidate has a positive complement-dependent cytotoxic crossmatch against donor lymphocytes. What does this finding imply?

A. The donor has antibodies against the recipient, which predicts graft-versus-host disease but does not increase the risk of kidney allograft rejection.

B. The crossmatch proves that the recipient and donor are HLA identical, so immunosuppression can be omitted after transplantation.

C. The result reflects only ABO incompatibility and cannot be caused by HLA antibodies or prior sensitizing events such as pregnancy or transfusion.

D. A positive crossmatch is expected in all successful kidney transplants because complement activation is required for immunologic tolerance.

E. The recipient has clinically important donor-reactive antibodies, creating a high risk of immediate antibody-mediated graft injury if transplantation proceeds without an effective strategy.

191. A kidney transplant candidate has an untreated active bacterial infection with persistent bacteremia. What is the best transplantation principle?

A. Permanently exclude the patient from transplantation because any prior bacterial infection is an irreversible contraindication even after cure.

B. Proceed immediately because induction immunosuppression improves bacterial clearance by reducing the inflammatory response to bacteremia.

C. Defer transplantation until the active infection is adequately treated because intensified immunosuppression can convert uncontrolled infection into a life-threatening complication.

D. Proceed if a living donor is available, because active infection is relevant only to deceased-donor transplantation and not to elective living donation.

E. Proceed after a single antibiotic dose because sterility of blood cultures is unnecessary when graft function is expected to be immediate.

192. A stable kidney transplant recipient develops a slowly rising creatinine. Ultrasound is normal, tacrolimus level is not excessive, and there is no clear reversible cause. What is the best next diagnostic principle?

A. Start broad-spectrum antibiotics for several months because occult pyelonephritis is the default explanation whenever ultrasound is normal.

B. Stop all immunosuppression to clarify the diagnosis, because withdrawal reliably distinguishes immune from nonimmune causes without risking irreversible injury.

C. Assume chronic rejection without tissue evaluation because all late allograft dysfunction has the same mechanism and treatment.

D. Increase tacrolimus empirically before any diagnostic testing because higher calcineurin-inhibitor exposure improves all common causes of graft dysfunction.

E. Obtain an allograft biopsy when clinically appropriate to distinguish rejection, recurrent disease, drug toxicity, infection, and other parenchymal causes.

193. A patient with CKD takes ibuprofen for several days and develops a rise in creatinine during an episode of volume depletion. Which renal hemodynamic effect of NSAIDs is most relevant?

A. NSAIDs stimulate renin release and markedly increase angiotensin II formation, producing isolated efferent arteriolar constriction without affecting afferent tone.

B. NSAIDs increase renal prostaglandin production, causing excessive afferent vasodilation and hyperfiltration that directly produces acute tubular necrosis.

C. Inhibition of renal prostaglandin synthesis reduces afferent arteriolar vasodilation and can lower glomerular perfusion in susceptible patients.

D. NSAIDs block proximal tubular creatinine secretion without changing true GFR, so a creatinine rise during volume depletion is always a benign laboratory artifact.

E. NSAIDs selectively dilate the efferent arteriole, lowering intraglomerular pressure through the same mechanism as ACE inhibitors.

194. A patient with albuminuric CKD starts an ACE inhibitor. Serum creatinine rises modestly over the next week and potassium is 4.8 mEq/L. Which mechanism explains the expected hemodynamic change?

A. Afferent arteriolar constriction is the principal action of ACE inhibition and directly increases intraglomerular pressure despite lowering systemic blood pressure.

B. ACE inhibition blocks proximal creatinine secretion without affecting glomerular hemodynamics, so any creatinine increase is purely an assay artifact.

C. Efferent arteriolar constriction raises filtration pressure, so a creatinine rise indicates that the medication has failed to block angiotensin II signaling.

D. ACE inhibition causes osmotic diuresis through proximal glucose wasting, and the early creatinine rise is therefore caused by the same mechanism as an SGLT2 inhibitor.

E. Efferent arteriolar dilation lowers intraglomerular pressure, which can produce a limited early fall in GFR while reducing proteinuria.

195. A patient taking trimethoprim-sulfamethoxazole has a small creatinine rise without change in cystatin C or urine output, and potassium increases to 5.6 mEq/L. Which drug effect best explains these findings?

A. Trimethoprim causes nephrogenic diabetes insipidus, and free-water loss raises creatinine solely through hypernatremia while potassium remains unaffected.

B. Trimethoprim can inhibit tubular creatinine secretion and act like an epithelial sodium-channel blocker in the distal nephron, producing pseudo-AKI and hyperkalemia.

C. Sulfamethoxazole directly increases glomerular filtration and drives potassium into cells, so the combination should lower creatinine and cause hypokalemia.

D. Trimethoprim causes selective efferent arteriolar thrombosis, producing true ischemic AKI while preserving cystatin C at the baseline level.

E. The combination stimulates aldosterone release and distal potassium secretion, making hyperkalemia evidence that the medication is not pharmacologically active.

196. A hospitalized patient receiving gentamicin develops nonoliguric AKI after several days. Urine sediment shows granular casts and there is mild magnesium wasting. Which mechanism is most likely?

A. Renal papillary necrosis from cyclooxygenase inhibition, because aminoglycosides share the same renal mechanism as chronic analgesic combinations.

B. Acute renal artery vasculitis, because aminoglycoside accumulation occurs in vascular endothelium rather than tubular epithelial cells.

C. Immune-complex deposition in glomerular capillaries, because aminoglycosides primarily cause a proliferative glomerulonephritis with low complement.

D. Aminoglycoside accumulation in proximal tubular cells causing toxic acute tubular injury.

E. Selective blockade of distal vasopressin receptors, because gentamicin nephrotoxicity is a pure nephrogenic diabetes-insipidus syndrome without tubular cell injury.

197. A patient receiving amphotericin B develops rising creatinine, hypokalemia, and hypomagnesemia. Which mechanism best explains this pattern?

A. Amphotericin B increases collecting-duct water permeability and therefore causes hyponatremia without tubular electrolyte losses or creatinine elevation.

B. The drug causes isolated glomerular immune-complex disease, so tubular potassium and magnesium losses argue strongly against amphotericin toxicity.

C. Amphotericin B inhibits creatinine secretion only, making the rise in creatinine spurious and unrelated to the simultaneous electrolyte abnormalities.

D. Amphotericin B selectively blocks distal sodium channels, producing hyperkalemia and metabolic acidosis without any effect on renal perfusion.

E. Amphotericin B causes renal vasoconstriction and direct tubular membrane injury, producing reduced GFR and renal potassium and magnesium wasting.

198. A patient receiving high-dose intravenous acyclovir becomes oliguric with flank discomfort and an abrupt creatinine rise. Urinalysis shows needle-shaped crystals. Which preventive measure is most appropriate when high-dose therapy is required?

A. Restrict fluid intake to concentrate the urine, because higher tubular acyclovir concentrations prevent precipitation of the drug in the collecting system.

B. Ensure adequate hydration and appropriate renal dose adjustment, and avoid unnecessarily rapid intravenous administration.

C. Add an NSAID to dilate the renal microcirculation, because prostaglandin inhibition protects against intratubular acyclovir crystallization.

D. Acidify the urine aggressively, because lower urine pH reliably dissolves acyclovir crystals and prevents obstructive tubular injury.

E. Combine acyclovir with a high-dose aminoglycoside, because proximal tubular competition prevents acyclovir entry into the urine.

199. A patient receiving high-dose methotrexate for leukemia develops AKI and markedly delayed methotrexate clearance despite intravenous hydration, urine alkalinization, and leucovorin rescue. Which drug can rapidly lower circulating methotrexate in this setting?

A. Mesna, because binding toxic methotrexate metabolites in the urinary tract directly accelerates systemic methotrexate elimination during AKI.

B. Rasburicase, because enzymatic oxidation of uric acid also hydrolyzes methotrexate and reverses delayed antifolate clearance.

C. Glucarpidase, which enzymatically converts extracellular methotrexate to inactive metabolites when toxic delayed clearance accompanies kidney dysfunction.

D. Amiloride, because epithelial sodium-channel blockade prevents proximal tubular uptake of methotrexate and rapidly lowers its serum concentration.

E. Desmopressin, because increasing collecting-duct water permeability enhances renal methotrexate clearance despite severe acute kidney injury.

200. A patient receiving cisplatin develops AKI with persistent hypomagnesemia despite replacement. Which renal adverse effect is characteristic of this drug?

A. Nephrogenic diabetes insipidus as the sole kidney toxicity, with magnesium retention rather than urinary magnesium wasting.

B. Direct tubular toxicity with renal magnesium wasting, which can persist even after the acute kidney injury improves.

C. Immune-complex crescentic glomerulonephritis, because cisplatin commonly produces low complement and active urinary sediment.

D. Selective glomerular podocyte injury causing isolated nephrotic syndrome without tubular electrolyte abnormalities.

E. Pure prerenal azotemia from systemic vasodilation, because cisplatin does not enter or injure renal tubular epithelial cells.

201. A patient treated with ifosfamide develops hypophosphatemia, glycosuria despite normal plasma glucose, and bicarbonate wasting. What is the most likely renal complication?

A. Acute uric acid nephropathy, because glycosuria and phosphate wasting are caused by urate crystals obstructing distal collecting ducts.

B. Minimal change nephrotic syndrome, because urinary glucose and bicarbonate loss are consequences of severe albuminuria rather than tubular transport defects.

C. Ifosfamide-induced proximal tubular injury causing Fanconi syndrome, which accounts for phosphate, glucose, and bicarbonate wasting.

D. Renal artery vasospasm, because ifosfamide causes a hemodynamic fall in GFR without direct tubular toxicity.

E. Ifosfamide-induced type IV renal tubular acidosis, because the drug selectively suppresses aldosterone and causes hyperkalemia without proximal solute wasting.

202. A patient taking acetazolamide for idiopathic intracranial hypertension develops bicarbonate 17 mEq/L, chloride 112 mEq/L, and persistently alkaline urine. Which renal pharmacologic effect best explains the acid-base disorder?

A. Carbonic anhydrase inhibition reduces proximal bicarbonate reabsorption, producing bicarbonaturia and a hyperchloremic normal-anion-gap metabolic acidosis.

B. Mineralocorticoid-receptor blockade suppresses distal potassium and hydrogen secretion, producing hyperkalemia with an acidic urine rather than proximal bicarbonate loss.

C. Epithelial sodium-channel blockade in collecting-duct principal cells causes isolated potassium retention without increasing urinary bicarbonate delivery.

D. Na-K-2Cl cotransporter inhibition in the thick ascending limb produces contraction alkalosis rather than sustained bicarbonaturia from proximal carbonic anhydrase inhibition.

E. Thiazide-sensitive NaCl cotransporter inhibition in the distal convoluted tubule causes hypokalemic alkalosis and does not directly block proximal bicarbonate reclamation.

203. A patient receiving a VEGF-pathway inhibitor for metastatic cancer develops new severe hypertension, proteinuria, thrombocytopenia, and AKI. Kidney biopsy shows glomerular endothelial swelling and thrombotic microangiopathy. Which drug effect best explains the renal syndrome?

A. VEGF inhibition selectively activates podocyte glucocorticoid receptors, causing minimal change disease without endothelial or blood-pressure effects.

B. The medication directly blocks aldosterone receptors, so the expected syndrome is isolated hyperkalemia and hypotension rather than endothelial injury.

C. VEGF inhibition causes obstructive crystal nephropathy because the drug precipitates in collecting ducts and forms calcium oxalate crystals.

D. VEGF-pathway inhibition can injure the glomerular endothelium, producing hypertension, proteinuria, and a renal thrombotic microangiopathy pattern.

E. The renal toxicity is limited to nephrogenic diabetes insipidus, because VEGF signaling is unrelated to glomerular endothelial integrity.

204. A patient with type 2 diabetes and CKD taking an SGLT2 inhibitor is scheduled for major surgery and will have prolonged fasting. Which medication-safety principle is most important?

A. Replace the SGLT2 inhibitor with an NSAID during fasting because afferent vasoconstriction preserves the medication's kidney-protective hemodynamic effect.

B. Stop all renin-angiotensin system blockade permanently instead, because perioperative ketoacidosis is caused by ACE inhibitors rather than SGLT2 inhibition.

C. Continue the SGLT2 inhibitor through prolonged fasting because its glucose-lowering effect requires hyperglycemia and therefore cannot contribute to ketoacidosis with a normal glucose level.

D. Temporarily withhold the SGLT2 inhibitor before the perioperative fasting period because of the risk of euglycemic ketoacidosis, then restart when clinically stable and eating.

E. Double the SGLT2 inhibitor dose on the morning of surgery because osmotic diuresis protects against perioperative volume shifts and acute kidney injury.

205. A hospitalized patient has a baseline creatinine of 0.9 mg/dL. Forty hours later the creatinine is 1.3 mg/dL, with no prior CKD. Which statement is correct?

A. AKI can be diagnosed only when urine output falls below 0.5 mL/kg/hour, so creatinine changes alone are insufficient.

B. The change represents CKD because any creatinine elevation lasting more than 24 hours defines chronic kidney disease.

C. The patient meets creatinine criteria for acute kidney injury because serum creatinine increased by at least 0.3 mg/dL within 48 hours.

D. AKI requires an absolute creatinine increase of at least 1.0 mg/dL within 7 days, regardless of the baseline value.

E. The patient does not have AKI because the creatinine must at least double before the diagnosis can be made.

206. A patient with AKI requires hemodialysis for refractory hyperkalemia. How is the AKI severity classified under the traditional KDIGO staging system?

A. The patient remains stage 1 unless serum creatinine has tripled, because dialysis requirement does not affect AKI staging.

B. The patient is stage 2 because hyperkalemia is an electrolyte complication rather than a criterion relevant to AKI severity.

C. Kidney replacement therapy places the patient in stage 3 AKI regardless of the contemporaneous serum creatinine value.

D. AKI staging cannot be assigned once dialysis begins because kidney replacement therapy makes serum creatinine uninterpretable.

E. The patient has end-stage kidney disease by definition because any dialysis treatment permanently reclassifies AKI as chronic kidney failure.

207. A patient develops AKI after septic shock. Urine microscopy shows numerous renal tubular epithelial cells and coarse granular 'muddy brown' casts. Which lesion is most likely?

A. Prerenal azotemia alone, which typically causes diffuse tubular epithelial necrosis and numerous muddy brown casts before structural injury occurs.

B. Acute interstitial nephritis, which is defined by muddy brown casts and lacks pyuria or white-cell abnormalities.

C. Minimal change disease, which causes coarse granular casts from podocyte loss even when albuminuria is absent.

D. Acute tubular injury, which commonly produces granular casts and tubular epithelial cells in the urine sediment.

E. Renal artery stenosis, which produces abundant tubular epithelial casts despite preserved tubular integrity and a bland sediment.

208. A patient has vomiting, orthostatic hypotension, rising creatinine, urine sodium 8 mEq/L, and a bland sediment with hyaline casts. Kidney function improves promptly after appropriate volume replacement. Which process best explains the AKI?

A. Acute interstitial nephritis, because gastrointestinal volume loss triggers eosinophilic interstitial inflammation that resolves with saline alone.

B. Bilateral renal cortical necrosis, because cortical infarction is rapidly reversible after extracellular-volume repletion.

C. Acute tubular injury, because intact tubules characteristically waste sodium and fail to improve after restoration of circulating volume.

D. Prerenal azotemia from reduced effective renal perfusion with intact tubular sodium conservation.

E. Acute glomerulonephritis, because hyaline casts and low urine sodium are specific for inflammatory glomerular capillary injury.

209. A patient receiving loop diuretics develops AKI. The fractional excretion of sodium is 2.5%, but the examination suggests volume depletion. How should the FENa be interpreted?

A. FENa is unaffected by diuretics because sodium handling in the loop of Henle does not influence urinary sodium concentration.

B. A FENa above 2% proves glomerulonephritis because inflammatory glomerular disease is the only cause of increased fractional sodium excretion.

C. FENa should replace bedside assessment because fractional excretion indices remain perfectly specific across sepsis, CKD, diuretic use, and obstruction.

D. A FENa above 1% proves acute tubular necrosis even when a loop diuretic was given immediately before urine sampling.

E. Loop diuretics can increase urinary sodium and make FENa less reliable, so the result must be interpreted with the clinical context and urine sediment.

210. An older man with prostatic enlargement develops anuria and rapidly rising creatinine. Bladder scan shows marked retention and ultrasound demonstrates bilateral hydronephrosis. What is the priority?

A. Begin high-dose glucocorticoids because bilateral hydronephrosis with anuria is most consistent with immune-mediated interstitial nephritis.

B. Delay decompression until a renal biopsy is performed because obstruction cannot cause severe AKI without histologic confirmation.

C. Promptly decompress the urinary tract and address the site and cause of obstruction.

D. Administer an ACE inhibitor to reduce glomerular pressure because postrenal AKI is best treated by lowering filtration before relieving the blockage.

E. Restrict all fluids for several days because restoration of urine flow is dangerous until serum creatinine returns to baseline spontaneously.

211. A patient with a solitary functioning kidney develops sudden anuria and severe flank pain. Ultrasound shows hydronephrosis of that kidney from an obstructing ureteral stone. What is the most appropriate principle?

A. Treat with corticosteroids because hydronephrosis in a solitary kidney is primarily an inflammatory process rather than a mechanical emergency.

B. Observe for several weeks because unilateral obstruction cannot cause clinically important AKI even when the contralateral kidney is absent.

C. Give an NSAID continuously at high dose because afferent vasoconstriction improves filtration across an acutely obstructed solitary kidney.

D. Urgent urinary decompression is required because obstruction of the solitary functioning kidney threatens total kidney function.

E. Begin chronic dialysis without relieving the obstruction because recovery of kidney function is impossible once anuria has developed.

212. A patient with septic shock has AKI and requires intravascular volume resuscitation. Which fluid approach is preferred for initial resuscitation in the absence of a special indication for another fluid?

A. Use intravenous crystalloids, with balanced crystalloids generally favored over 0.9% saline for many adults with sepsis or septic shock.

B. Use hypertonic saline exclusively because high sodium concentration prevents septic acute tubular injury and eliminates the need for vasopressors.

C. Use loop diuretics instead of resuscitation fluid because forcing urine output reverses renal hypoperfusion even when the patient remains in shock.

D. Use hydroxyethyl starch as first-line resuscitation because synthetic colloids reduce AKI and improve survival compared with crystalloids.

E. Avoid crystalloid because isotonic fluids are contraindicated whenever serum creatinine is elevated during septic shock.

213. A patient with septic shock remains hypotensive after initial fluid resuscitation and has evidence of ongoing hypoperfusion. Which vasopressor is preferred first line?

A. Phenylephrine in all patients, because pure alpha-agonism has superior survival and renal outcomes compared with norepinephrine in septic shock.

B. Terlipressin, because current sepsis guidance recommends it as the universal first-line vasopressor for adults with septic shock.

C. No vasopressor until urine output normalizes, because pressors worsen renal perfusion whenever serum creatinine is elevated.

D. Low-dose dopamine, because it is preferred specifically for kidney protection and reliably prevents progression of septic AKI.

E. Norepinephrine, titrated to restore adequate arterial pressure and perfusion while the underlying infection is treated.

214. A patient with septic shock is receiving large-volume resuscitation and has worsening AKI. Which resuscitation fluid should generally be avoided because it increases kidney-related harm?

A. Balanced crystalloid, because buffered isotonic crystalloids are contraindicated once septic AKI has developed.

B. 0.9% saline, because any exposure to chloride-containing crystalloid is absolutely prohibited in all patients with kidney injury.

C. Human albumin, because albumin universally causes osmotic nephrosis and is never considered after substantial crystalloid administration.

D. Hydroxyethyl starch, a synthetic colloid that should not be used for routine resuscitation in sepsis.

E. Oral water, because enteral free water is the principal cause of septic acute tubular injury in critically ill adults.

215. A patient with severe cholestatic liver failure has bilirubin 38 mg/dL and progressive AKI. Urine sediment contains pigmented casts, and kidney biopsy shows bile-stained tubular casts with acute tubular injury. Which diagnosis is most likely?

A. Hepatorenal syndrome alone, because functional renal vasoconstriction characteristically produces abundant bile-stained tubular casts on kidney biopsy.

B. Bile cast nephropathy, in which severe hyperbilirubinemia contributes to tubular toxicity and intratubular bile-cast obstruction.

C. Acute interstitial nephritis, because bilirubin directly recruits eosinophils and produces an allergic interstitial infiltrate rather than tubular casts.

D. Myoglobin pigment nephropathy, because bilirubin is converted to myoglobin in renal tubules during severe cholestasis.

E. Cholesterol crystal embolization, because jaundice causes atheroemboli that form pigmented intratubular casts without vascular cholesterol clefts.

216. A patient is found after prolonged immobilization with crush injury. Creatine kinase is 45,000 U/L, potassium is 6.1 mEq/L, phosphate is elevated, and urine dipstick is strongly positive for blood with few red cells on microscopy. What is the renal diagnosis?

A. Acute interstitial nephritis, because interstitial inflammation releases myoglobin from skeletal muscle into the urine.

B. Rhabdomyolysis with myoglobin-mediated pigment nephropathy causing acute tubular injury.

C. Prerenal azotemia alone, because myoglobin does not directly injure tubules and therefore cannot contribute to AKI.

D. Acute glomerulonephritis, because a positive urine heme test with few red cells indicates glomerular bleeding from ruptured capillary loops.

E. Renal papillary necrosis, because papillary sloughing causes massive creatine kinase elevation and hyperphosphatemia after muscle compression.

217. A patient with bulky lymphoma develops AKI, hyperkalemia, hyperphosphatemia, hypocalcemia, and marked hyperuricemia after chemotherapy. Which diagnosis is most likely?

A. Acute interstitial nephritis, because allergic inflammation classically produces simultaneous severe hyperkalemia, hypocalcemia, and tumor-cell breakdown.

B. Renal artery thrombosis, because abrupt loss of kidney perfusion directly releases phosphate and uric acid from malignant lymphocytes.

C. Minimal change disease, because nephrotic syndrome after lymphoma treatment causes the characteristic combination of hyperuricemia and hyperphosphatemia.

D. Tumor lysis syndrome causing acute kidney injury from rapid release of intracellular solutes and uric acid/phosphate burden.

E. Hepatorenal syndrome, because liver-mediated renal vasoconstriction produces hyperuricemia and hyperphosphatemia immediately after cytotoxic chemotherapy.

218. A hospitalized patient with decompensated cirrhosis has progressive AKI despite stopping diuretics, treating infection, and receiving appropriate albumin expansion. There is no shock, nephrotoxin exposure, obstruction, or active urine sediment. Which therapy is appropriate for suspected hepatorenal syndrome-AKI in a suitable patient?

A. Administer an NSAID to constrict the afferent arteriole because reduced renal blood flow reverses the circulatory physiology of hepatorenal syndrome.

B. Use vasoconstrictor therapy such as terlipressin together with albumin while urgently addressing liver-transplant candidacy and monitoring for adverse effects.

C. Escalate loop diuretics until creatinine normalizes because forced natriuresis directly corrects the intense renal vasoconstriction of hepatorenal syndrome.

D. Start an ACE inhibitor to dilate the efferent arteriole because lower intraglomerular pressure restores filtration in advanced cirrhosis.

E. Treat with corticosteroids as definitive therapy because hepatorenal syndrome is an immune-complex glomerulonephritis without structural liver dependence.

219. A patient with septic shock has received initial crystalloid resuscitation but remains hypoperfused. Central venous pressure is 14 mm Hg, and the team is uncertain whether more fluid will improve cardiac output. Which approach is most appropriate?

A. Continue crystalloid until central venous pressure exceeds 20 mm Hg because a fixed static filling-pressure target reliably identifies adequate renal perfusion.

B. Stop all further fluid permanently once an initial weight-based bolus has been given because no patient with septic shock can remain fluid responsive afterward.

C. Use a dynamic assessment such as a passive-leg-raise or small fluid challenge with a measured stroke-volume response to guide additional fluid rather than relying on a static pressure alone.

D. Use urine output alone to decide each additional bolus because oliguria is a specific real-time marker of preload responsiveness in septic AKI.

E. Administer hydroxyethyl starch as the diagnostic fluid challenge because synthetic colloid response predicts cardiac output without kidney risk.

220. A patient with severe pancreatitis is mechanically ventilated and develops oliguria, rising peak airway pressures, a tense abdomen, and escalating vasopressor requirements. Bladder pressure is markedly elevated. Which mechanism is most likely contributing to the AKI?

A. Abdominal compartment syndrome causing increased intra-abdominal pressure, reduced renal perfusion, and venous outflow obstruction.

B. Renal artery embolism, because unilateral arterial occlusion typically creates a tense abdomen and globally elevated bladder pressure during pancreatitis.

C. Acute interstitial nephritis, because drug allergy is the defining cause of increased intra-abdominal pressure and reduced lung compliance in critical illness.

D. Isolated nephrogenic diabetes insipidus, because loss of collecting-duct water reabsorption causes oliguria and increased intra-abdominal pressure.

E. Minimal change disease, because acute podocyte injury raises airway pressures and bladder pressure through selective albuminuria without edema.

221. A patient with massive intravascular hemolysis after a mismatched transfusion develops dark urine and AKI. Urine is heme positive but contains few intact red blood cells. Which mechanism is most likely?

A. Acute uric acid nephropathy, because hemolysis releases enough uric acid to explain heme-positive urine in the absence of red cells.

B. Acute interstitial nephritis, because circulating free hemoglobin triggers an eosinophilic allergic infiltrate as the primary kidney lesion.

C. Renal cortical necrosis, because cortical infarction specifically produces heme-positive urine without red cells through urinary myoglobin release.

D. Glomerular capillary rupture, because free hemoglobin in urine requires intact red cells to cross the glomerular basement membrane.

E. Hemoglobin-mediated pigment nephropathy causing tubular toxicity and obstruction after intravascular hemolysis releases filtered free hemoglobin.

222. A patient with CKD undergoes bowel preparation with an oral sodium-phosphate product and later develops AKI. Biopsy shows abundant calcium-phosphate deposits in distal tubules and collecting ducts. What is the diagnosis?

A. Oxalate nephropathy, because oral phosphate is metabolized to oxalate crystals that deposit exclusively in proximal tubules.

B. Acute phosphate nephropathy from a large phosphate load with intratubular calcium-phosphate deposition.

C. Urate nephropathy, because phosphate-containing bowel preparations cause tumor lysis and massive uric acid release from intestinal mucosa.

D. Acute interstitial nephritis, because the characteristic lesion is eosinophilic inflammation without crystal deposition.

E. Minimal change disease, because sodium phosphate causes isolated podocyte effacement and nephrotic syndrome rather than tubular mineral deposition.

223. A postpartum patient with severe hemorrhage, shock, and disseminated intravascular coagulation develops persistent anuria. Imaging later shows patchy nonenhancement of the renal cortex with relative medullary sparing. Which diagnosis is most likely?

A. Renal vein thrombosis, because venous occlusion from nephrotic syndrome routinely affects both kidneys symmetrically after postpartum hemorrhage.

B. Acute papillary necrosis, because papillary ischemia selectively removes cortical enhancement while preserving the renal medulla.

C. Minimal change disease, because podocyte injury after pregnancy causes bilateral cortical nonenhancement and irreversible anuria.

D. Acute interstitial nephritis, because drug hypersensitivity produces geographic cortical infarction with preserved medullary blood flow.

E. Renal cortical necrosis from severe and prolonged cortical ischemic injury, a recognized complication of catastrophic hemorrhage, shock, and DIC.

224. After relief of severe bilateral urinary obstruction, a patient produces 7 liters of urine in 24 hours and becomes orthostatic with falling potassium and magnesium levels. What is the best management principle?

A. Clamp the urinary catheter for several hours at a time because postobstructive diuresis is caused by excessive decompression and is best treated by recreating the obstruction.

B. Recognize postobstructive diuresis and replace fluid and electrolytes judiciously with close monitoring while allowing the kidneys to excrete retained salt and water.

C. Replace every milliliter of urine with an equal or larger volume of hypotonic fluid automatically, because matching output exactly prevents all electrolyte disturbances.

D. Give high-dose desmopressin routinely because postobstructive diuresis is always caused by permanent central diabetes insipidus after bladder decompression.

E. Stop all oral and intravenous intake because continued urine production proves that total-body volume remains excessive even when orthostatic hypotension develops.

225. A patient with oliguric AKI has arterial pH 6.98 from severe metabolic acidosis that persists despite treatment of the underlying shock and appropriate medical therapy. Which management is most appropriate?

A. Administer an acetazolamide infusion because bicarbonate wasting is the preferred treatment for profound metabolic acidosis in oliguric AKI.

B. Initiate kidney replacement therapy for severe refractory acidemia that persists despite treatment of the underlying cause and appropriate medical measures.

C. Give hydrochlorothiazide to increase distal acid secretion because thiazide diuretics reliably correct life-threatening acidemia in anuric patients.

D. Restrict ventilation to raise carbon dioxide because superimposed respiratory acidosis will reduce the renal generation of organic acids.

E. Wait for serum creatinine to reach 10 mg/dL because acidemia is not an independent reason to begin kidney replacement therapy.

226. A patient with oliguric acute tubular injury is euvolemic and oxygenating well. A colleague proposes high-dose furosemide solely to convert the patient to nonoliguric AKI and prevent dialysis. Which statement is most accurate?

A. High-dose loop diuretics should be routine in all oliguric AKI because conversion to nonoliguria directly accelerates tubular regeneration and improves mortality.

B. Loop diuretics are absolutely contraindicated in AKI because any increase in urine flow worsens tubular oxygen consumption and inevitably increases kidney injury.

C. Furosemide is indicated only when serum creatinine exceeds 5 mg/dL because creatinine, rather than volume status, determines whether diuresis is beneficial.

D. Loop diuretics replace kidney replacement therapy for refractory hyperkalemia because kaliuresis remains dependable even when urine output is minimal.

E. Loop diuretics can be useful to manage volume overload, but forcing urine output does not reverse established tubular injury or reliably prevent dialysis or improve survival.

227. A patient with advanced kidney failure has active mucosal bleeding and a prolonged bleeding time attributed to uremic platelet dysfunction. An urgent invasive procedure is needed. Which short-term therapy can improve platelet function?

A. Warfarin, because vitamin K antagonism corrects the platelet adhesion defect caused by retained uremic solutes before an invasive procedure.

B. Aspirin, because irreversible cyclooxygenase inhibition restores platelet granule release and is the preferred treatment for uremic bleeding.

C. Clopidogrel, because P2Y12 inhibition increases platelet aggregation when bleeding is caused by advanced kidney failure.

D. Desmopressin, which can transiently improve primary hemostasis in uremia by increasing release of von Willebrand factor and factor VIII.

E. Tissue plasminogen activator, because systemic fibrinolysis removes the uremic toxins that interfere with platelet-vessel-wall interaction.

228. A patient with severe AKI, refractory hyperkalemia, and septic shock requires kidney replacement therapy but is receiving high-dose vasopressors. Which modality is often preferred initially?

A. No kidney replacement therapy can be provided during shock, so refractory hyperkalemia should be managed only with repeated intracellular shifts.

B. Isolated plasma exchange because it removes potassium and corrects acidosis more efficiently than dialysis without affecting blood pressure.

C. High-efficiency intermittent hemodialysis with the fastest possible ultrafiltration, because rapid fluid shifts improve blood pressure in vasopressor-dependent shock.

D. Peritoneal dialysis is mandatory because continuous extracorporeal therapies are contraindicated whenever vasopressors are required.

E. Continuous kidney replacement therapy because slower solute and fluid removal may be better tolerated in marked hemodynamic instability.

229. A patient on continuous venovenous hemodiafiltration for AKI is clinically stable. What delivered effluent dose is generally targeted based on established KDIGO guidance?

A. At least 80 to 100 mL/kg/hour for every patient, because high-intensity CRRT has consistently improved survival compared with standard dosing.

B. Exactly 1 mL/kg/hour, because CRRT dose should match normal urine output rather than extracorporeal solute clearance.

C. Approximately 20 to 25 mL/kg/hour of delivered effluent, recognizing that the prescribed dose may need to be higher to account for interruptions.

D. No dose target is needed because effluent rate is unrelated to solute clearance in continuous hemodialysis or hemofiltration.

E. Approximately 2 to 5 mL/kg/hour, because very low effluent rates provide the same small-solute clearance as standard CRRT doses.

230. A patient on CRRT has a high bleeding risk and no contraindication to citrate. Which anticoagulation strategy can minimize systemic anticoagulant exposure?

A. Full-dose systemic warfarin, because vitamin K antagonism provides immediate circuit anticoagulation and is easily titrated during CRRT.

B. Dual antiplatelet therapy, because platelet inhibition is preferred to circuit-based anticoagulation for preventing filter clotting in CRRT.

C. Regional citrate anticoagulation of the extracorporeal circuit with protocolized calcium replacement and metabolic monitoring.

D. No calcium monitoring, because citrate does not bind ionized calcium and therefore cannot cause clinically important metabolic complications.

E. Systemic thrombolysis, because continuous infusion of a fibrinolytic agent is safer than regional anticoagulation in patients at high bleeding risk.

231. A patient receiving regional citrate anticoagulation for CRRT develops worsening high-anion-gap metabolic acidosis, falling systemic ionized calcium, and a rising total-calcium-to-ionized-calcium ratio despite increasing calcium infusion. What complication should be suspected?

A. Acute phosphate nephropathy, because calcium-phosphate crystals in the kidney cause extracorporeal citrate to accumulate in the bloodstream.

B. Heparin-induced thrombocytopenia, because citrate directly activates platelet factor 4 and causes the same syndrome as unfractionated heparin.

C. Dialysis disequilibrium syndrome, because rapid urea removal specifically raises the total-to-ionized calcium ratio during continuous therapy.

D. Citrate accumulation from impaired citrate metabolism, causing systemic calcium chelation and metabolic derangement.

E. Hypermagnesemia from citrate, because citrate is metabolized into magnesium and therefore increases total calcium measurements artifactually.

232. During hemodialysis, a patient suddenly develops chest and back pain, dyspnea, nausea, and dark red-brown blood in the extracorporeal circuit. Serum potassium rises abruptly. What is the best immediate response?

A. Continue dialysis at a higher blood-flow rate because rapid clearance of free hemoglobin prevents hyperkalemia and makes returning the circuit blood safer.

B. Suspect acute intravascular hemolysis, stop dialysis, clamp the blood lines, and do not return the potentially hemolyzed circuit blood to the patient while the cause is investigated.

C. Diagnose dialysis disequilibrium syndrome and give additional dialysate sodium because visible red-brown circuit blood is caused by cerebral osmotic shifts.

D. Return all circuit blood immediately before stopping dialysis because hemolyzed blood is harmless once it has passed through the dialyzer membrane.

E. Treat only with an oral potassium binder because sudden hyperkalemia during dialysis is expected and does not suggest a mechanical or dialysate emergency.

233. A patient with severe salicylate poisoning has altered mental status, pulmonary edema, persistent acidemia, and rising salicylate levels despite bicarbonate therapy. What extracorporeal treatment is preferred?

A. Intermittent hemodialysis, which rapidly removes salicylate and corrects acid-base and electrolyte abnormalities in severe poisoning.

B. Plasma exchange alone, because salicylate is confined to plasma proteins and cannot be cleared effectively across a hemodialysis membrane.

C. No extracorporeal therapy, because bicarbonate alkalinization is sufficient even when neurologic deterioration and pulmonary edema are present.

D. Ultrafiltration without dialysate, because isolated water removal provides the same salicylate clearance as diffusive hemodialysis.

E. Peritoneal dialysis, because it removes salicylate more rapidly than high-efficiency intermittent hemodialysis in life-threatening toxicity.

234. A patient with chronic lithium therapy presents with confusion, tremor, seizures, AKI, and a markedly elevated lithium concentration. Which extracorporeal treatment is preferred?

A. Plasma exchange, because lithium is highly protein bound and is removed most effectively by exchanging the plasma compartment.

B. Hemoperfusion only, because lithium cannot cross standard hemodialysis membranes due to its large molecular size and high volume of distribution.

C. No extracorporeal therapy, because lithium is not dialyzable and severe neurologic toxicity must resolve only through renal excretion.

D. Isolated ultrafiltration, because convective water removal without substantial solute clearance eliminates lithium faster than hemodialysis.

E. Intermittent hemodialysis, with serial lithium levels afterward because redistribution can cause a rebound increase.

235. A patient drinks windshield-washer fluid and develops visual blurring, severe anion-gap metabolic acidosis, and an elevated osmolar gap. Which treatment is appropriate in severe methanol poisoning?

A. Treat with insulin and glucose because intracellular methanol shift is the most effective way to prevent optic-nerve injury.

B. Give an alcohol-dehydrogenase inhibitor such as fomepizole and use intermittent hemodialysis when severe features such as visual toxicity or major acidemia are present.

C. Give acetazolamide to acidify the blood because conversion of methanol to formate is slowed as systemic pH falls.

D. Use bicarbonate alone and avoid alcohol-dehydrogenase blockade because methanol itself, rather than formate, is the principal toxic metabolite.

E. Administer calcium gluconate as the specific antidote because methanol toxicity is mediated by oxalate binding of ionized calcium.

236. A patient ingests antifreeze and develops high-anion-gap metabolic acidosis, hypocalcemia, AKI, and calcium oxalate crystals in the urine. What is the best diagnosis and treatment principle?

A. Isopropanol poisoning; give bicarbonate and hemodialysis routinely because isopropanol produces severe anion-gap acidosis with oxalate crystals.

B. Lithium poisoning; give calcium to bind lithium in the gut because renal oxalate deposition is the characteristic target-organ injury of lithium.

C. Methanol poisoning; treat with thiamine alone because oxalate formation is prevented by vitamin replacement without an alcohol-dehydrogenase inhibitor.

D. Ethylene glycol poisoning; inhibit alcohol dehydrogenase with fomepizole and use hemodialysis when poisoning is severe or metabolic and kidney complications warrant it.

E. Salicylate poisoning; acidify the urine because calcium oxalate crystallization is caused by alkaline salicylate metabolites.

237. A patient with kidney failure taking metformin develops profound lactic acidosis, shock, and altered mental status. Standard supportive measures are failing. What is the kidney-specific treatment principle?

A. Avoid dialysis because metformin has a molecular size and protein binding pattern that prevents clinically meaningful extracorporeal removal.

B. Consider urgent extracorporeal treatment, preferably intermittent hemodialysis when feasible, for severe metformin-associated lactic acidosis with shock or profound acidemia.

C. Use isolated ultrafiltration without dialysate because removal of plasma water corrects lactic acidosis more effectively than diffusive solute clearance.

D. Treat only with an oral alkali preparation because severe shock and impaired kidney function do not alter the need for extracorporeal support.

E. Give additional metformin to suppress hepatic gluconeogenesis because lowering glucose production directly consumes lactate and reverses severe acidemia.

238. A patient with severe AKI is receiving multiple renally cleared antimicrobials. Which medication-management principle is most appropriate?

A. Use the usual full outpatient doses throughout AKI because dose reduction is unnecessary until permanent end-stage kidney disease has been diagnosed.

B. Stop every antimicrobial once creatinine rises because renally cleared drugs cannot be administered safely during AKI under any circumstances.

C. Reassess drug doses frequently using the evolving kidney function and the type and intensity of kidney replacement therapy, rather than relying on a single admission creatinine.

D. Dose all medications according to urine color because serum creatinine and kidney replacement modality do not affect drug clearance in critical illness.

E. Use a fixed eGFR reported by the laboratory as an exact measure of clearance during rapidly changing AKI, because steady-state assumptions remain fully valid.

239. A patient recovers from dialysis-requiring AKI and is discharged with creatinine near the pre-illness baseline. What follow-up principle is most appropriate?

A. Arrange post-AKI reassessment of kidney function and albuminuria because survivors remain at increased risk for recurrent AKI, CKD, cardiovascular events, and medication-related complications.

B. Measure serum creatinine only if edema develops because asymptomatic decline in kidney function cannot occur after apparent recovery from AKI.

C. No kidney follow-up is needed once creatinine returns near baseline because recovered AKI has no association with later CKD or recurrent kidney injury.

D. Permanently avoid all renin-angiotensin system blockers because any prior AKI episode is an absolute lifelong contraindication to these medications.

E. Begin chronic dialysis access planning in every survivor because dialysis-requiring AKI inevitably progresses to permanent kidney failure.

240. A patient has stage 3 AKI with creatinine 5.0 mg/dL but is hemodynamically stable, potassium is normal, acid-base status is acceptable, volume can be managed medically, and there are no uremic manifestations. What should determine whether dialysis is started?

A. Start dialysis solely because the patient meets stage 3 criteria, since all stage 3 AKI requires immediate extracorporeal treatment even when stable.

B. Never use dialysis in potentially reversible AKI because kidney replacement therapy is reserved exclusively for irreversible chronic kidney failure.

C. Start dialysis immediately because any creatinine of 5.0 mg/dL is an absolute indication independent of symptoms, trajectory, or complications.

D. Delay dialysis until creatinine reaches 10 mg/dL because a universal numerical threshold is safer than assessing the patient's clinical condition.

E. The overall clinical context and presence of refractory complications or inability to meet metabolic and volume needs, not the creatinine value alone.

Answers

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1. D. The low urine sodium, orthostasis, and gastrointestinal losses support hypovolemic hyponatremia. Isotonic saline restores effective arterial volume, suppresses nonosmotic vasopressin release, and allows excretion of excess free water.

2. D. Euvolemic hypotonic hyponatremia with urine that remains concentrated and urine sodium above 30 mEq/L, after excluding thyroid and adrenal disease, is classic for SIADH. Small-cell lung cancer is a well-known cause through ectopic vasopressin production.

3. A. Seizure from severe hyponatremia is an indication for prompt 3% saline bolus therapy, with close monitoring. The goal is an early modest rise sufficient to relieve cerebral edema, not rapid normalization, because overcorrection can cause osmotic demyelination.

4. E. This high-risk patient has already corrected too rapidly. Desmopressin can stop the water diuresis, and electrolyte-free water can be used to relower sodium when necessary, limiting the risk of osmotic demyelination.

5. B. Beer potomania is a low-solute hyponatremic state. Even with maximally dilute urine, the kidney cannot excrete large amounts of water when daily osmole excretion is very low.

6. E. Very dilute urine in a hyponatremic patient indicates effective suppression of vasopressin. With excessive water intake and otherwise normal physiology, primary polydipsia is the best explanation.

7. C. In heart failure, arterial underfilling activates sympathetic tone, the renin-angiotensin-aldosterone system, and vasopressin. Water retention can exceed sodium retention, producing dilutional hyponatremia despite edema.

8. D. Cirrhosis commonly causes hypervolemic hyponatremia through arterial underfilling and vasopressin-mediated water retention. Without severe neurologic symptoms, management is gradual and directed at the underlying cirrhotic physiology rather than rapid sodium correction.

9. E. Cerebral salt wasting and SIADH may both show high urine sodium after neurologic injury, but the differentiating clinical feature is volume status. This patient is hypovolemic with negative balance, supporting cerebral salt wasting.

10. D. A urine osmolality of 850 mOsm/kg shows a robust renal concentrating response. In hypernatremia, this argues against diabetes insipidus and points toward insufficient intake or extrarenal hypotonic fluid loss.

11. E. A large increase in urine osmolality after desmopressin indicates that the kidney can respond to vasopressin but endogenous hormone activity was deficient. That pattern is characteristic of central diabetes insipidus.

12. A. Lithium enters principal cells through ENaC and impairs vasopressin signaling and aquaporin expression. Amiloride blocks lithium entry and is a preferred targeted therapy when lithium must be continued.

13. D. Severe hyperglycemia increases extracellular tonicity and shifts water from cells into extracellular fluid, lowering measured sodium concentration. This is hypertonic or translocational hyponatremia, not hypotonic hyponatremia.

14. A. With extreme lipids or proteins, indirect measurement can underestimate sodium because the nonaqueous fraction of plasma is increased. Direct ion-selective electrode measurement would show the true sodium in plasma water.

15. A. A reset osmostat is a variant of altered osmoregulation in which sodium is stably defended at a lower level. Patients retain appropriate urinary dilution and concentration responses around that lower set point.

16. E. Primary adrenal insufficiency produces both cortisol and aldosterone deficiency. Sodium wasting lowers effective arterial volume, while cortisol deficiency favors vasopressin release; hyperkalemia strongly supports primary rather than secondary adrenal failure.

17. B. Thiazides reduce sodium chloride reabsorption in the distal convoluted tubule, a major diluting segment, and can provoke severe hyponatremia. The offending drug should be stopped and correction monitored carefully.

18. E. Chronic, mildly symptomatic or asymptomatic SIADH is generally managed gradually with removal of causes, fluid restriction, increased solute strategies, or selected aquaretic approaches. Hypertonic saline is reserved for severe neurologic symptoms or other urgent situations.

19. D. Inadequate free-water provision with intact renal concentration is a common cause of hospital-acquired hypernatremia. The deficit and ongoing losses should be replaced in a controlled fashion, avoiding overly rapid correction when hypernatremia is chronic.

20. B. The anion gap is 140 - (100 + 14) = 26 mEq/L, which is elevated with a normal albumin concentration. In septic shock, lactic acidosis is a common cause.

21. E. During DKA treatment, the high anion gap may resolve while bicarbonate remains low because ketoanions are lost in urine and chloride-rich fluids increase serum chloride. The result is a non-anion-gap acidosis.

22. E. Winter's formula predicts PaCO2 of about 1.5 x 10 + 8 = 23 mm Hg, plus or minus 2. The measured value of 24 mm Hg is appropriate compensation for metabolic acidosis.

23. B. Diarrhea causes direct bicarbonate loss. The kidney responds by increasing ammonium excretion with chloride, making the urine anion gap negative, which argues against impaired renal acid excretion.

24. C. Type 4 RTA is common in diabetic kidney disease and with renin-angiotensin system blockade. Hypoaldosteronism or aldosterone resistance reduces distal potassium and hydrogen secretion, producing hyperkalemic normal-gap acidosis.

25. D. Distal RTA causes inability to lower urine pH appropriately during systemic acidosis. Hypokalemia, hypocitraturia, nephrocalcinosis, and calcium phosphate stones are typical associated findings.

26. E. Fanconi syndrome causes generalized proximal reabsorptive failure, including bicarbonate, phosphate, glucose, uric acid, and amino acids. Bicarbonate wasting produces proximal RTA.

27. A. Salicylates stimulate the respiratory center, causing respiratory alkalosis, while organic acid accumulation produces high-gap metabolic acidosis. The combination often yields a near-normal or alkalemic pH despite serious toxicity.

28. D. DKA causes extracellular potassium shift, but urinary losses from osmotic diuresis and volume depletion deplete total-body stores. Serum potassium may therefore be normal or high before treatment even though replacement will be needed.

29. E. When hyperkalemia causes ECG changes, intravenous calcium is the urgent membrane-stabilizing therapy. It does not lower potassium, so intracellular shifting and potassium removal therapies must follow.

30. C. Insulin rapidly activates cellular potassium uptake and is a mainstay of emergency temporizing therapy. Dextrose is usually given unless significant hyperglycemia makes it unnecessary, and potassium must still be removed from the body.

31. E. Beta-2 agonists stimulate sodium-potassium ATPase and shift potassium intracellularly. They are adjunctive temporizing therapy; they do not remove total-body potassium.

32. B. Chronic hyperkalemia should be managed by addressing diet, drugs, acidosis, volume status, and other contributors. Newer potassium binders can permit continuation of prognostically beneficial RAAS blockade in selected patients.

33. B. Serum potassium may be falsely elevated when platelets release potassium during clot formation. A normal promptly processed plasma potassium supports pseudohyperkalemia rather than true hyperkalemia.

34. E. A high urinary potassium during hypokalemia indicates inappropriate renal loss. Loop diuretics increase distal sodium delivery and activate RAAS, producing both potassium wasting and contraction alkalosis.

35. E. Hypomagnesemia removes inhibition of renal outer medullary potassium channels and increases urinary potassium loss. Magnesium repletion is often necessary before potassium can be corrected effectively.

36. B. Aldosterone stimulates ENaC-mediated sodium uptake, creates a lumen-negative potential, and promotes potassium and hydrogen secretion. Primary aldosteronism therefore often produces hypokalemia and metabolic alkalosis.

37. B. Vomiting causes hydrogen chloride loss and volume contraction. A low urine chloride indicates avid renal chloride retention and identifies a chloride-responsive alkalosis that typically improves with chloride and volume repletion.

38. C. Urine chloride is useful in metabolic alkalosis but must be interpreted in context. Active loop or thiazide diuresis can keep urine chloride elevated even when the overall physiology is volume-contracted and chloride-depleted.

39. B. Lactic acidosis from shock reflects impaired oxygen delivery or utilization and adrenergic stress. Definitive therapy is correction of the hemodynamic and etiologic problem; bicarbonate has limited selected roles and does not reverse the cause.

40. C. As GFR declines, the kidney's capacity to excrete daily acid load as ammonium and titratable acids decreases. Retained unmeasured anions can produce a high-gap component in advanced kidney failure.

41. A. In chronic hypercapnia, the kidney increases bicarbonate reabsorption and net acid excretion over several days. A markedly elevated bicarbonate with near-normal pH therefore supports chronic respiratory acidosis.

42. B. Advanced CKD reduces phosphate excretion and calcitriol production. These changes, along with altered calcium sensing and skeletal resistance, stimulate parathyroid hyperplasia and secondary hyperparathyroidism.

43. D. Humoral hypercalcemia of malignancy is commonly mediated by PTHrP, especially in squamous-cell cancers. PTHrP produces PTH-like effects while native PTH is appropriately suppressed.

44. E. After parathyroidectomy in severe hyperparathyroid bone disease, mineralized bone avidly takes up calcium, phosphate, and magnesium. This can cause prolonged postoperative hypocalcemia requiring aggressive replacement.

45. D. Severe symptomatic hypermagnesemia can depress neuromuscular and cardiac function. Intravenous calcium provides temporary physiologic antagonism; patients with kidney failure may also require dialysis for magnesium removal.

46. C. Chronic proton-pump inhibitor therapy can impair gastrointestinal magnesium absorption. Low urinary magnesium during hypomagnesemia shows that the kidney is conserving magnesium appropriately, supporting an extrarenal cause.

47. A. Citrate complexes urinary calcium and inhibits crystal formation. Potassium citrate is effective for hypocitraturic calcium stone disease, while extreme dietary calcium restriction can paradoxically increase oxalate absorption.

48. A. Acidic urine is the dominant driver of many uric acid stones. Raising urine pH with potassium citrate can dissolve uric acid and prevent recurrence; xanthine oxidase inhibition is added when hyperuricosuria or gout warrants it.

49. B. Urease-positive bacteria split urea into ammonia, raising urine pH and increasing ammonium. Magnesium ammonium phosphate and carbonate apatite can then form large struvite stones, often with staghorn morphology.

50. C. Hexagonal urine crystals are characteristic of cystine. Cystinuria is an inherited defect in proximal tubular transport of cystine and dibasic amino acids, leading to recurrent stones at a young age.

51. D. Primary hyperparathyroidism increases serum calcium, reduces proximal phosphate reabsorption, and often increases urinary calcium load. Recurrent calcium nephrolithiasis is a classic complication.

52. C. CKD requires abnormalities of kidney structure or function present for at least 3 months. Two low eGFR values over only 2 weeks may still reflect unresolved acute kidney disease rather than established CKD.

53. A. An eGFR of 30 to 44 mL/min/1.73 m2 is G3b, and urine albumin-creatinine ratio above 300 mg/g is A3. Both dimensions are used because prognosis depends on GFR and albuminuria.

54. D. Spot UACR corrects albumin concentration for urine concentration and is the preferred practical measure of albuminuria. Persistent abnormalities should generally be confirmed because exercise, infection, fever, and other factors can transiently increase albumin excretion.

55. A. Creatinine generation depends on muscle mass. Cystatin C is less influenced by muscle mass, and a combined creatinine-cystatin C estimate often improves accuracy when treatment or diagnosis depends on a more reliable GFR estimate.

56. A. The Kidney Failure Risk Equation is validated for estimating the probability of treated kidney failure over defined horizons in many patients with CKD. It can help guide referral intensity, education, and access planning.

57. E. ACE inhibitors and ARBs reduce intraglomerular pressure and albuminuria and are central therapies for albuminuric CKD when tolerated. Creatinine and potassium should be monitored after initiation or dose escalation.

58. D. ACE inhibitor plus ARB therapy can further reduce albuminuria but increases hyperkalemia, hypotension, and acute kidney injury without a net outcome benefit. Dual blockade is therefore generally avoided.

59. C. SGLT2 inhibitors have kidney-protective effects that extend beyond glucose lowering and benefit many patients with proteinuric CKD without diabetes. They reduce intraglomerular pressure and slow long-term GFR decline.

60. D. SGLT2 inhibitors commonly cause a small initial fall in eGFR from tubuloglomerular feedback and reduced intraglomerular pressure. In a stable patient, this is usually not a reason to discontinue therapy.

61. E. Finerenone reduces kidney and cardiovascular events in appropriately selected patients with type 2 diabetes and albuminuric CKD already receiving maximally tolerated RAAS blockade. Hyperkalemia risk requires baseline and follow-up potassium monitoring.

62. A. When additional glucose lowering is needed in type 2 diabetes with CKD, long-acting GLP-1 receptor agonists with proven cardiovascular benefit are often preferred. They can reduce cardiovascular events, support weight loss, and may provide kidney benefit.

63. A. CKD is a high cardiovascular-risk state, and statin-based therapy is recommended for many adults with nondialysis CKD according to age and risk profile. The principal benefit is reduction of atherosclerotic events rather than direct preservation of GFR.

64. B. High sodium intake worsens hypertension and blunts the antiproteinuric effect of ACE inhibitors and ARBs. Moderating sodium intake is a core component of CKD blood pressure and volume management.

65. D. Excessive protein intake can increase glomerular hyperfiltration and uremic solute production, whereas severe restriction risks malnutrition. Most patients with stable CKD are counseled toward a moderate, individualized protein intake.

66. C. Chronic metabolic acidosis is common in advanced CKD and contributes to bone and muscle consequences. Oral alkali can be used in selected patients, balanced against sodium load and volume or blood pressure concerns.

67. A. Mild chronic hyperkalemia should prompt review of diet, medications, acidosis, constipation, and volume status. Potassium-lowering strategies can often permit continuation of therapies with proven kidney and cardiovascular benefit.

68. A. Iron deficiency is common in CKD and is treatable. Ferritin and transferrin saturation help assess iron stores and availability, while the broader evaluation should consider bleeding, B12 or folate deficiency, inflammation, hemolysis, and other causes.

69. D. Low ferritin with low transferrin saturation supports absolute iron deficiency. Iron replacement is a foundational component of anemia management and may improve hemoglobin or reduce ESA requirements.

70. C. ESA decisions in CKD are individualized rather than triggered by a single universal hemoglobin value. Symptoms, rate of decline, transfusion risk, cardiovascular history, malignancy, thrombosis risk, and patient preferences all matter.

71. D. Trials of aggressive hemoglobin normalization with ESAs have shown increased harm without commensurate benefit. ESA therapy is generally adjusted to avoid unnecessarily high hemoglobin levels and excessive dosing.

72. A. Red-cell transfusions can expose patients to nonself HLA antigens and increase sensitization. In transplant candidates, avoiding nonessential transfusion may preserve access to compatible donors and shorten transplant waiting.

73. D. CKD-MBD is a dynamic disorder. Management is based on trends and the interaction among phosphate, calcium, PTH, vitamin D status, bone disease, and treatment rather than a single number in isolation.

74. E. Persistent hyperphosphatemia is managed with dietary phosphate reduction, dialysis removal, and phosphate binders when needed. Binder choice should account for calcium load, vascular calcification risk, adverse effects, and adherence.

75. B. In nondialysis CKD, modest PTH elevations may be adaptive. Routine calcitriol or vitamin D analog use is not favored for mild elevations because of hypercalcemia risk; modifiable factors should be addressed first.

76. E. Calcimimetics suppress PTH by activating the calcium-sensing receptor and often lower serum calcium. This can be useful when PTH is high and additional calcium loading or active vitamin D would be undesirable.

77. B. In patients with hypercalcemia or substantial calcification, excess calcium exposure may be undesirable. Non-calcium binders can reduce phosphate without adding additional calcium load.

78. D. Uremic pericarditis is a strong indication to start or intensify dialysis. The goal is removal of uremic toxins while monitoring closely for effusion and tamponade.

79. A. Severe hyperkalemia that persists despite appropriate medical therapy requires definitive potassium removal. Dialysis rapidly removes potassium and is indicated when kidney excretion is inadequate and the disturbance is refractory.

80. B. Refractory pulmonary edema is a classic urgent indication for kidney replacement therapy. Extracorporeal or peritoneal ultrafiltration can remove volume when medical diuresis is inadequate.

81. E. Dialysis initiation is individualized. Symptoms of uremia, refractory electrolyte or acid-base disorders, uncontrolled volume, nutrition decline, or other complications matter more than a single eGFR threshold.

82. D. Modern access planning follows an individualized life-plan rather than a one-size-fits-all fistula rule. Early education allows time for modality choice, transplant evaluation, and creation of durable access when appropriate.

83. E. Central venous dialysis catheters have the highest infection risk among standard hemodialysis access types. Reducing catheter dependence is an important safety goal when a durable access is feasible.

84. C. Kt/V is a standard metric for urea clearance in hemodialysis, although adequacy is broader than any single number. Treatment time, volume control, symptoms, nutrition, and other factors also matter.

85. A. Large interdialytic weight gains force high ultrafiltration rates, which can exceed plasma refill and provoke hypotension. Sodium and fluid management, target-weight reassessment, and safer ultrafiltration rates address the cause.

86. C. Dialysis disequilibrium is most likely during initial treatment of severe azotemia when urea is removed rapidly from plasma. Slower, gentler initial dialysis reduces the osmotic gradient that can produce cerebral edema.

87. B. Cloudy effluent with abdominal pain and neutrophilic dialysate strongly suggests PD peritonitis. Cultures should be obtained promptly, but empiric intraperitoneal antibiotics should not be delayed while awaiting results.

88. E. Icodextrin is used for long dwells because it provides sustained colloid osmotic pressure and can improve ultrafiltration in appropriate PD patients, especially when rapid glucose absorption limits long-dwell fluid removal.

89. A. Exit-site infection can extend along the subcutaneous catheter tunnel. Tunnel involvement increases the risk of peritonitis and often requires more intensive therapy than superficial exit-site inflammation alone.

90. C. Encapsulating peritoneal sclerosis is an uncommon but severe long-term PD complication involving inflammation and fibrosis that can encase the bowel and cause obstruction, malnutrition, and abdominal symptoms.

91. B. Residual kidney function provides continuous clearance and fluid removal between treatments and often improves volume management and quality of life. Avoiding unnecessary nephrotoxins and severe volume depletion can help preserve it.

92. D. Vaccination is an important part of CKD preventive care. Hepatitis B vaccination is ideally completed before dialysis when feasible because immune response may be stronger and future blood exposure risk is greater.

93. B. When contrast imaging is clinically important, the diagnostic benefit may outweigh kidney risk. Risk mitigation includes appropriate volume management, avoiding concurrent nephrotoxins, and using the lowest necessary dose of modern contrast rather than reflexively withholding needed imaging.

94. D. Prostaglandins support afferent vasodilation during low perfusion states, while angiotensin II supports efferent tone. NSAIDs, RAAS blockers, and volume depletion can therefore combine to produce a marked hemodynamic drop in GFR.

95. B. Drug dosing in CKD is medication-specific. Loading doses often depend more on volume of distribution, while maintenance dosing depends on renal and nonrenal clearance; dialysis can add substantial extracorporeal clearance for some agents.

96. E. CKD increases risks of preeclampsia, preterm birth, fetal growth problems, and maternal kidney deterioration, with risk rising as CKD and albuminuria worsen. Preconception medication and blood pressure planning is essential.

97. A. CKD-associated pruritus is multifactorial and may persist despite adequate dialysis and skin care. Treatments can include renally dosed gabapentinoids and other targeted agents, while avoiding sedation and accumulation.

98. D. Chronic loop therapy increases distal sodium reabsorption, contributing to diuretic resistance. Adding a thiazide-type diuretic can block this compensatory pathway, but electrolyte depletion and volume contraction require close monitoring.

99. B. Creatinine can rise transiently during decongestion because of changes in renal perfusion and hemoconcentration. Clinical congestion, blood pressure, electrolytes, and trajectory help distinguish acceptable hemodynamic change from true kidney injury.

100. A. The kidney contributes to insulin clearance and gluconeogenesis. Advanced CKD can therefore increase insulin exposure and reduce counterregulatory glucose production, making hypoglycemia more likely.

101. D. Many salt substitutes replace sodium chloride with potassium chloride. In patients with impaired potassium excretion or medications that raise potassium, these products can cause clinically important hyperkalemia.

102. B. CKD changes the choice and dosing of gout therapies but is not itself a reason to leave hyperuricemia with gout untreated. Drug-specific renal dosing, interactions, hypersensitivity risk, and flare prophylaxis should be individualized.

103. A. Bone biopsy is not routine, but it can clarify turnover, mineralization, and volume when the diagnosis is uncertain and the result would materially alter therapy, especially before potent bone-active treatment in complex CKD-MBD.

104. D. Inorganic phosphate additives are highly bioavailable and can contribute substantially to phosphate burden. Dietary counseling should reduce avoidable additive phosphate without causing protein-energy malnutrition.

105. A. Blood pressure technique materially affects the result. Standardized office measurement reduces misclassification and should precede treatment escalation based on a single poorly obtained reading.

106. E. Masked hypertension can be missed by office readings and is associated with cardiovascular and kidney risk. Ambulatory or validated home monitoring is useful when office values do not fit the clinical picture.

107. A. Apparent resistant hypertension often reflects poor measurement, white-coat effect, nonadherence, excess sodium, suboptimal diuresis, or pressor drugs. These should be addressed before labeling the condition truly resistant.

108. D. Primary aldosteronism should be considered in resistant hypertension, hypokalemia, adrenal incidentaloma, or selected high-risk settings. Screening uses aldosterone and renin, while medications and hypokalemia can affect interpretation.

109. D. Nonfunctioning adrenal nodules are common, especially with age. Adrenal venous sampling is usually needed to distinguish unilateral from bilateral aldosterone excess before potentially curative surgery.

110. C. Most stable atherosclerotic renal-artery stenosis is managed medically, but recurrent flash pulmonary edema, rapidly declining kidney function in selected bilateral disease, or refractory hypertension may justify revascularization consideration.

111. A. Fibromuscular dysplasia is a nonatherosclerotic, noninflammatory arterial disorder often affecting renal and cervicocephalic arteries. Medial fibroplasia classically produces a beaded angiographic appearance.

112. D. Metanephrines are continuously produced within catecholamine-secreting tumors and are sensitive biochemical markers. Proper sampling conditions and awareness of interfering medications reduce false-positive results.

113. E. OSA is strongly associated with resistant hypertension, obesity, nondipping nocturnal blood pressure, and sympathetic activation. Diagnosis and treatment can improve overall cardiovascular risk and may aid blood pressure control.

114. E. The phenotype is classic for Cushing syndrome. Cortisol excess can activate mineralocorticoid pathways at high levels and enhance catecholamine responsiveness, producing hypertension along with metabolic and catabolic findings.

115. C. Coarctation should be considered in young patients with hypertension, radiofemoral delay, upper-lower extremity pressure differences, or a characteristic murmur. It may be associated with bicuspid aortic valve and intracranial aneurysm risk.

116. A. Hypertensive emergency is defined by severe hypertension with acute target-organ injury, such as encephalopathy, retinal injury, myocardial ischemia, pulmonary edema, aortic dissection, or AKI. Controlled intravenous reduction is usually required.

117. B. Aortic dissection requires rapid reduction of impulse and pressure. Intravenous beta blockade is used first to reduce heart rate and dP/dt, with vasodilator therapy added if needed after heart-rate control.

118. C. Hypertensive acute pulmonary edema is driven by high afterload and volume redistribution. Rapid titratable vasodilation, especially nitrates when appropriate, plus respiratory support and diuresis can quickly improve hemodynamics.

119. A. Malignant hypertension can cause arteriolar endothelial injury and secondary TMA with microangiopathic hemolysis, thrombocytopenia, and AKI. Severe ADAMTS13 deficiency instead supports immune TTP.

120. D. Acute severe hypertension in pregnancy should be treated promptly with agents that have established obstetric use, including IV labetalol, IV hydralazine, or immediate-release oral nifedipine. RAAS blockers are avoided in pregnancy.

121. D. KDIGO blood pressure guidance supports a lower standardized systolic target for many adults with CKD when tolerated. The measurement method and the patient's frailty, falls, orthostasis, and overall goals are essential to applying that target safely.

122. E. Blood pressure targets must be individualized. Symptomatic orthostasis, frailty, falls, limited life expectancy, and medication burden can justify less intensive treatment even when lower targets are recommended for many healthier patients.

123. C. Recurrent flash pulmonary edema with bilateral atherosclerotic renal-artery stenosis is often called Pickering syndrome. It is one of the clinical scenarios in which revascularization may be considered more strongly.

124. C. Calcineurin inhibitors can constrict renal arterioles, activate sodium-retentive pathways, and impair potassium excretion. High levels may produce hypertension, hyperkalemia, neurologic effects, and nephrotoxicity.

125. A. Minoxidil is a potent arteriolar vasodilator that can trigger sympathetic tachycardia and substantial fluid retention. A beta blocker and potent diuretic are usually paired with it when used for severe resistant hypertension.

126. E. Chronic clonidine suppresses central sympathetic outflow. Abrupt discontinuation can produce marked rebound sympathetic activation, so the drug should generally be tapered rather than stopped suddenly.

127. B. Glycyrrhetinic acid in licorice inhibits 11-beta-HSD2, allowing cortisol to stimulate mineralocorticoid receptors in the kidney. The resulting sodium retention, potassium loss, and hypertension suppress both renin and aldosterone.

128. C. Liddle syndrome is caused by constitutively active ENaC, producing sodium retention, hypertension, hypokalemia, and metabolic alkalosis with suppressed renin and aldosterone. Amiloride or triamterene targets the abnormal channel.

129. B. The classic triad is not always present, but fever, rash, eosinophilia, sterile pyuria, and WBC casts after a new medication strongly support acute interstitial nephritis. Stopping the offending drug is essential.

130. D. Chronic analgesic nephropathy is a tubulointerstitial process historically associated with heavy combination analgesic exposure. Papillary necrosis and small scarred kidneys are characteristic late findings.

131. D. Lithium can cause nephrogenic diabetes insipidus and, after long exposure, chronic tubulointerstitial nephropathy with fibrosis and microcyst formation. Kidney function and lithium levels should be monitored over time.

132. A. ADPKD is an autosomal dominant systemic cystic disease with bilateral renal cysts, progressive enlargement, hypertension, and variable age at kidney failure. Family history is often present but can be absent with de novo disease.

133. D. Tolvaptan reduces vasopressin-driven cAMP signaling and can slow ADPKD progression in selected adults at risk for rapid progression. Polyuria and thirst are expected, and liver safety monitoring is required.

134. D. ADPKD increases intracranial aneurysm risk, especially with a personal or strong family history of aneurysm or subarachnoid hemorrhage. Screening decisions are individualized and commonly use noninvasive angiographic imaging.

135. A. Fanconi syndrome is generalized proximal tubular reabsorptive failure. Drugs such as tenofovir can cause bicarbonaturia, phosphaturia, glycosuria despite normal plasma glucose, uricosuria, aminoaciduria, and tubular proteinuria.

136. A. Bartter syndromes mimic loop-diuretic physiology in the thick ascending limb. They cause salt wasting, high renin and aldosterone, hypokalemic alkalosis, and often hypercalciuria; some forms present early and severely.

137. C. Gitelman syndrome resembles chronic thiazide exposure: renal salt wasting causes secondary hyperaldosteronism and hypokalemic alkalosis, while hypomagnesemia and hypocalciuria are characteristic.

138. A. Dysmorphic erythrocytes and red-cell casts localize bleeding to the glomerulus. When accompanied by reduced GFR, edema, and hypertension, the pattern is an acute nephritic syndrome.

139. D. Nephrotic syndrome causes heavy proteinuria, hypoalbuminemia, edema, and often hyperlipidemia. Hypercoagulability is clinically important, especially in membranous nephropathy and when serum albumin is very low.

140. B. IgA nephropathy often presents with gross hematuria at the time of a mucosal infection, in contrast to the latent period of postinfectious GN. Diagnosis is established by kidney biopsy showing dominant or codominant mesangial IgA.

141. C. Supportive care remains central in IgA nephropathy and includes optimized blood pressure, RAAS blockade for proteinuria, sodium restriction, and modern CKD disease-modifying therapy. Disease-specific options are added according to risk and eligibility.

142. C. The 2025 IgA nephropathy guideline incorporates disease-targeted therapies in addition to optimized supportive care. Selection depends on progression risk, eGFR, proteinuria, safety profile, and local availability rather than a universal immunosuppressive regimen.

143. D. IgA vasculitis combines systemic small-vessel features such as purpura, arthralgia, and gastrointestinal involvement with an IgA nephropathy-like glomerular lesion. Adults can have substantial kidney morbidity.

144. C. PLA2R is the major target antigen in many cases of primary membranous nephropathy. Antibody level can help with diagnosis and disease monitoring when interpreted in the clinical context.

145. E. Membranous nephropathy can be secondary to malignancy, infections, autoimmune disease, or medications. A negative PLA2R result plus concerning systemic features increases the importance of a secondary-cause evaluation.

146. B. Minimal change disease shows little or no abnormality on light microscopy and diffuse foot-process effacement on electron microscopy. NSAIDs are a recognized secondary trigger and may also cause interstitial nephritis.

147. E. Primary minimal change disease often responds to glucocorticoids. Adults may require a longer course to achieve remission than children, and relapse patterns guide subsequent steroid-sparing therapy.

148. D. Primary FSGS is suggested by full nephrotic syndrome and diffuse foot-process effacement after secondary and genetic causes are considered. Secondary adaptive forms often have less diffuse effacement and proteinuria related to hyperfiltration.

149. E. Adaptive FSGS occurs when remaining nephrons hyperfilter, as in obesity, reflux nephropathy, or reduced nephron mass. Treating the driver, controlling pressure, and reducing proteinuria are key; immunosuppression is not routinely indicated.

150. E. APOL1 high-risk genotypes are associated with increased susceptibility to FSGS and other kidney disease in individuals with recent African ancestry. Genetic information can aid counseling and etiologic classification in selected cases.

151. C. Poststreptococcal GN follows a latent period after infection and often has low C3, nephritic urine sediment, and classic subepithelial hump-shaped deposits. Treatment is generally supportive while infection is addressed when still present.

152. A. Infection-related GN can occur during active staphylococcal infection, especially in older adults and patients with diabetes. IgA-dominant deposits and low complement may mimic primary IgA nephropathy, but the infection context is decisive.

153. E. MPGN describes a morphologic pattern rather than one disease. Etiologic classification into immune-complex or complement-mediated processes guides the workup and treatment.

154. A. C3 glomerulopathy is driven by dysregulation of the alternative complement pathway and shows dominant C3 staining relative to immunoglobulins. Evaluation may include complement studies and assessment for monoclonal proteins.

155. C. Anti-GBM disease causes rapidly progressive crescentic GN and may cause pulmonary hemorrhage. Linear IgG staining along the GBM is characteristic, and treatment is urgent because kidney recovery declines with advanced irreversible injury.

156. E. ANCA-associated vasculitis causes necrotizing crescentic GN with little or no immune deposition. PR3-ANCA is commonly associated with granulomatosis with polyangiitis, while MPO-ANCA is common in microscopic polyangiitis.

157. B. Avacopan blocks the C5a receptor and can reduce glucocorticoid exposure when used with standard remission-induction therapy in selected patients. Rituximab or cyclophosphamide remains central for organ-threatening disease.

158. E. Current guidance is more selective about plasma exchange than older practice. It may be considered in some patients with very severe kidney disease, rapidly rising creatinine, dialysis dependence, or severe diffuse alveolar hemorrhage.

159. B. Class III or IV lupus nephritis is an immune-complex proliferative disease requiring immunosuppression. Modern regimens combine glucocorticoids with mycophenolic acid analogs or low-dose cyclophosphamide, with belimumab or calcineurin-inhibitor strategies in selected patients.

160. A. Class V lupus nephritis may be managed conservatively when proteinuria is low, but nephrotic-range disease generally warrants immunosuppressive therapy because of risks from persistent proteinuria and nephrotic syndrome.

161. B. Hepatitis C is a classic cause of mixed cryoglobulinemia. Low C4, rheumatoid factor activity, purpura, neuropathy, and an MPGN pattern strongly support cryoglobulinemic immune-complex disease.

162. D. Fibrillary GN shows organized fibrillar deposits that are generally Congo red negative, distinguishing it from amyloidosis. Modern immunostaining such as DNAJB9 is highly useful in diagnosis.

163. D. Congo-red positivity with apple-green birefringence identifies amyloid. Kidney involvement often causes heavy proteinuria, and systemic features such as restrictive cardiomyopathy or macroglossia can suggest AL amyloidosis, but definitive fibril typing is essential.

164. A. MGRS describes kidney disease caused by a nephrotoxic monoclonal immunoglobulin produced by a clone that may be too small to meet conventional malignancy criteria. Kidney-directed diagnosis can therefore change hematologic treatment.

165. A. Severe ADAMTS13 deficiency strongly supports immune TTP, a medical emergency requiring immediate plasma exchange and immunosuppressive therapy. Kidney injury can occur, but prominent neurologic and hematologic findings are common.

166. C. Complement-mediated aHUS results from uncontrolled alternative complement activation on endothelium. C5 inhibition can stop ongoing complement-mediated TMA and improve hematologic and kidney outcomes.

167. C. Hyperacute rejection occurs within minutes to hours when preformed donor-reactive antibodies activate complement and injure the allograft endothelium. A positive crossmatch and immediate thrombosis are classic clues.

168. A. Interstitial inflammation and tubulitis are typical of acute T-cell-mediated rejection. Corticosteroids are standard first-line therapy, while lymphocyte-depleting treatment may be used for severe or steroid-resistant rejection.

169. B. Antibody-mediated rejection is supported by evidence of microvascular injury, antibody-endothelium interaction such as C4d, and circulating donor-specific antibodies. Treatment is individualized and often combines intensified immunosuppression with antibody-directed strategies.

170. D. Transplant glomerulopathy is a major morphologic feature of chronic antibody-mediated injury. In the presence of donor-specific antibodies and chronic microvascular changes, chronic active antibody-mediated rejection is strongly favored.

171. A. Tacrolimus and cyclosporine can acutely reduce GFR through afferent arteriolar vasoconstriction. High drug exposure, hyperkalemia, hypomagnesemia, and a bland sediment support calcineurin-inhibitor toxicity.

172. E. Tacrolimus is metabolized through CYP3A pathways. Azole antifungals such as voriconazole inhibit this metabolism and can markedly increase tacrolimus exposure, requiring anticipatory dose adjustment and frequent monitoring.

173. A. BK polyomavirus nephropathy is promoted by intense immunosuppression. The main evidence-based management principle is stepwise reduction of immunosuppression while balancing rejection risk and following viral load and kidney function.

174. C. CMV syndrome or tissue-invasive disease after transplantation requires antiviral treatment. Oral valganciclovir is appropriate for many nonsevere cases, whereas intravenous ganciclovir is favored for severe or life-threatening disease or unreliable absorption.

175. B. Trimethoprim-sulfamethoxazole is preferred Pneumocystis prophylaxis in most kidney transplant recipients and can additionally reduce risk of toxoplasmosis, nocardiosis, and some urinary infections.

176. C. PTLD encompasses lymphoid proliferations arising in immunosuppressed transplant recipients and is often associated with EBV. Initial management commonly includes reduction of immunosuppression when feasible, with rituximab and other oncologic therapy according to subtype and severity.

177. C. Belatacept carries a specific risk of PTLD, particularly involving the central nervous system, in EBV-seronegative recipients. It is indicated only for recipients with evidence of prior EBV exposure.

178. A. Induction intensity is individualized. Lymphocyte-depleting agents such as antithymocyte globulin are commonly favored for higher immunologic risk, whereas basiliximab is often used in lower-risk recipients.

179. A. Tacrolimus plus mycophenolate is a widely used maintenance backbone, often with corticosteroids depending on center protocol and patient risk. Regimens are individualized to rejection, infection, malignancy, and toxicity risks.

180. D. Delayed graft function, commonly defined operationally by dialysis need in the first week, most often reflects ischemia-reperfusion acute tubular injury in deceased-donor grafts. It does not exclude rejection, obstruction, vascular complications, or other causes.

181. E. Transplant renal artery stenosis can cause resistant hypertension, graft dysfunction, and an allograft bruit. A fall in filtration after ACE inhibition supports hemodynamically significant renal artery narrowing.

182. A. Post-transplant urinary obstruction is a reversible cause of allograft dysfunction. Hydronephrosis with preserved perfusion should prompt rapid urologic evaluation and decompression when clinically significant.

183. E. Primary FSGS can recur very early after transplantation and may present with abrupt nephrotic-range proteinuria. The timing is much faster than many immune-complex recurrences.

184. A. IgA nephropathy can recur in the allograft. Hematuria and proteinuria with dominant mesangial IgA deposition support recurrence rather than a nonspecific transplant injury.

185. A. Post-transplant diabetes is multifactorial, but tacrolimus and glucocorticoids are important medication contributors. Age, obesity, family history, infection, and other metabolic risk factors also matter.

186. C. Calcineurin inhibitors can cause hyperkalemia through distal tubular effects and are well known to cause renal magnesium wasting. These abnormalities may occur even when overall allograft function is stable.

187. B. Solid-organ transplant recipients have a substantially increased skin-cancer risk. Preventive counseling, sun protection, and regular dermatologic surveillance are important; immunosuppression may be adjusted in selected patients with recurrent malignancy.

188. D. Transplant recipients should receive appropriate non-live vaccines, although responses may be attenuated. Live attenuated vaccines are generally avoided during significant immunosuppression because of the risk of uncontrolled vaccine-organism replication.

189. C. Mycophenolate exposure is associated with miscarriage and characteristic congenital malformations. Pregnancy should be planned with the transplant team, with conversion to a compatible maintenance regimen before conception.

190. E. A positive crossmatch indicates recipient antibodies that react with donor cells, often donor-specific HLA antibodies. This finding signals substantial risk for hyperacute or early antibody-mediated rejection and requires specialized evaluation.

191. C. Active serious infection is generally a reason to delay transplantation until it is controlled. The decision is individualized, but proceeding with potent immunosuppression during uncontrolled bacteremia is unsafe.

192. E. Persistent unexplained allograft dysfunction often requires biopsy because many clinically similar processes demand very different treatment. Tissue diagnosis can identify rejection, infection, toxicity, recurrent disease, and other pathology.

193. C. Prostaglandins help preserve afferent arteriolar dilation when renal perfusion is threatened. Cyclooxygenase inhibition can remove this compensation, reducing GFR in volume depletion, heart failure, cirrhosis, and other prostaglandin-dependent states.

194. E. ACE inhibitors reduce angiotensin II-mediated efferent arteriolar tone, lowering intraglomerular pressure. A modest early creatinine rise can be hemodynamic and acceptable, whereas a large rise should prompt evaluation for volume depletion, renal artery stenosis, NSAID exposure, or other causes.

195. B. 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 effect on epithelial sodium channels.

196. D. Aminoglycosides are filtered and taken up by proximal tubular cells, where they can cause dose- and exposure-related acute tubular injury. Nonoliguric AKI, granular casts, and electrolyte wasting are typical clues.

197. E. Conventional amphotericin B is nephrotoxic through renal vasoconstriction and direct tubular effects. Potassium and magnesium wasting can accompany the decline in GFR; lipid formulations generally reduce but do not eliminate nephrotoxicity.

198. B. Acyclovir can precipitate in renal tubules, especially with high intravenous doses, rapid infusion, volume depletion, and inadequate renal adjustment. Hydration, dosing for kidney function, and controlled infusion reduce risk.

199. C. High-dose methotrexate can precipitate or injure renal tubules, creating a cycle of impaired clearance and systemic toxicity. When toxic delayed clearance occurs with kidney dysfunction, glucarpidase rapidly hydrolyzes circulating methotrexate; leucovorin rescue continues according to specialist protocols.

200. B. Cisplatin is a classic cause of tubular nephrotoxicity and magnesium wasting. Prevention centers on appropriate hydration, dose considerations, and avoidance of additional nephrotoxins when possible.

201. C. Ifosfamide can cause proximal tubular toxicity, particularly with cumulative exposure, producing Fanconi syndrome and sometimes chronic kidney dysfunction.

202. A. Acetazolamide inhibits proximal tubular carbonic anhydrase, decreasing bicarbonate reclamation. The resulting bicarbonaturia raises urine pH and can cause a hyperchloremic normal-anion-gap metabolic acidosis.

203. D. VEGF signaling is important for glomerular endothelial health. VEGF-pathway inhibitors can cause hypertension, proteinuria, and kidney-limited or systemic thrombotic microangiopathy, making drug exposure an important clue when these findings occur together.

204. D. SGLT2 inhibitors can precipitate euglycemic diabetic ketoacidosis during prolonged fasting, acute illness, or surgery. Temporary withholding around these stressors is an important medication-stewardship practice.

205. C. KDIGO AKI criteria include an increase in serum creatinine of at least 0.3 mg/dL within 48 hours, an increase to at least 1.5 times baseline within 7 days, or qualifying oliguria. The creatinine change in this case therefore meets AKI criteria.

206. C. Under KDIGO staging, initiation of kidney replacement therapy qualifies as stage 3 AKI. This staging convention does not imply irreversible kidney failure or long-term dialysis dependence.

207. D. Muddy brown granular casts and renal tubular epithelial cells strongly support acute tubular injury. Prerenal physiology more often has a bland sediment or hyaline casts unless ischemia progresses to structural tubular damage.

208. D. Volume depletion, low urine sodium, bland sediment, and rapid response to volume restoration support prerenal AKI. Persistent ischemia can eventually progress to structural acute tubular injury.

209. E. FENa is a physiologic clue rather than a definitive diagnostic test. Diuretics, CKD, sepsis, pigment injury, and other conditions can alter urinary sodium handling, so microscopy and clinical context remain essential.

210. C. Bilateral urinary tract obstruction can cause severe postrenal AKI and requires timely decompression. After relief, patients may develop postobstructive diuresis and need close monitoring of volume and electrolytes.

211. D. Obstruction of a solitary functioning kidney is physiologically equivalent to bilateral obstruction and can produce abrupt anuric AKI. Rapid decompression may preserve or restore function.

212. A. Current sepsis guidance favors crystalloids as first-line resuscitation fluid and generally prefers balanced crystalloids over normal saline. Fluid should be individualized with frequent reassessment to avoid both underresuscitation and overload.

213. E. Norepinephrine is the preferred first-line vasopressor for septic shock. 'Renal-dose' dopamine does not prevent or treat AKI and exposes patients to arrhythmic and other adverse effects.

214. D. Hydroxyethyl starch solutions have been associated with kidney harm and are recommended against in sepsis resuscitation. Crystalloids remain first-line, with fluid choice and volume tailored to the patient.

215. B. Bile cast nephropathy, also called cholemic nephropathy, can occur with marked hyperbilirubinemia. Bile acids and pigments may injure tubular epithelium and form obstructing casts, often superimposed on other hemodynamic insults in liver failure.

216. B. Rhabdomyolysis releases myoglobin, potassium, phosphate, and other intracellular contents. Heme-positive urine with few red cells suggests pigment, and myoglobin can cause tubular obstruction and oxidant injury; early volume resuscitation and electrolyte management are central.

217. D. Tumor lysis syndrome releases potassium, phosphate, and nucleic-acid metabolites, producing hyperkalemia, hyperphosphatemia, hypocalcemia, hyperuricemia, and AKI. Prevention and treatment include hydration, urate-lowering strategies, and dialysis when severe complications are refractory.

218. B. HRS-AKI is a functional kidney failure syndrome in advanced cirrhosis after alternative causes are addressed. Vasoconstrictor therapy with albumin can improve kidney function in selected patients, but liver transplantation remains definitive therapy for eligible patients.

219. C. After initial resuscitation in sepsis, additional fluid should be individualized. Dynamic measures such as the stroke-volume response to passive leg raise or a small fluid bolus are more informative about fluid responsiveness than static filling pressures alone.

220. A. Marked intra-abdominal hypertension can reduce renal perfusion, compress renal veins, impair ventilation, and worsen shock. In the appropriate clinical context, decompression and treatment of the underlying abdominal process may be kidney-saving.

221. E. Intravascular hemolysis releases free hemoglobin, which can appear as urine heme positivity without many intact red cells. Filtered heme pigment can cause oxidative tubular injury and cast formation, especially with volume depletion and acidemia.

222. B. Large phosphate loads can cause hyperphosphatemia and calcium-phosphate precipitation in renal tubules, especially in susceptible patients with CKD, older age, volume depletion, or medications affecting renal perfusion.

223. E. Renal cortical necrosis is a rare but severe ischemic lesion associated with catastrophic obstetric events, shock, sepsis, and DIC. Persistent anuria and cortical nonenhancement with medullary sparing are characteristic.

224. B. After obstruction is relieved, patients may excrete large amounts of retained sodium and water and can have a transient concentrating defect. Significant postobstructive diuresis requires frequent monitoring and tailored replacement of volume, potassium, magnesium, and other losses without simply matching output indiscriminately.

225. B. Severe metabolic acidosis that remains refractory to correction of the underlying cause and appropriate medical therapy is an accepted indication for urgent kidney replacement therapy.

226. E. Diuretics can help control fluid overload in AKI but are not disease-modifying therapy for acute tubular injury. Increasing urine volume does not by itself improve renal recovery, survival, or the need for kidney replacement therapy.

227. D. Uremia impairs platelet adhesion and aggregation. Desmopressin can provide a rapid but temporary improvement in hemostasis, useful for active bleeding or urgent procedures while definitive management of uremia, including dialysis when indicated, is addressed.

228. E. Both intermittent and continuous therapies can be effective, but CRRT is commonly chosen for patients with major hemodynamic instability because fluid and solute removal occur more gradually.

229. C. KDIGO recommends delivering an effluent dose in the range of 20 to 25 mL/kg/hour for CRRT in AKI. Prescriptions often exceed the desired delivered dose because downtime and interruptions reduce actual therapy.

230. C. Regional citrate chelates calcium within the circuit and can reduce systemic bleeding risk compared with systemic heparin. Safe use requires protocolized citrate delivery, calcium replacement, and monitoring of acid-base status and calcium indices.

231. D. Citrate accumulation can occur when metabolism is impaired, particularly in severe shock or liver dysfunction. A rising total-to-ionized calcium ratio, increasing calcium requirement, ionized hypocalcemia, and unexplained acidosis are important warning signs.

232. B. Acute hemolysis during hemodialysis can result from overheated or hypotonic dialysate, chemical contamination, or mechanical obstruction. Dialysis should be stopped immediately and the circuit blood should not be returned because it may deliver a large potassium and free-hemoglobin load.

233. A. Severe salicylate poisoning with neurologic symptoms, pulmonary compromise, refractory acidemia, high concentrations, or failure of standard therapy is an indication for extracorporeal treatment. Intermittent hemodialysis is preferred when available.

234. E. Lithium is a small, dialyzable ion. Severe poisoning, especially with neurologic toxicity or impaired kidney function, can require hemodialysis; rebound after treatment occurs because lithium redistributes from tissues.

235. B. Methanol is metabolized by alcohol dehydrogenase to toxic formate. Fomepizole blocks toxic metabolite formation, and hemodialysis is indicated in severe poisoning to remove methanol/formate and correct profound acidosis.

236. D. Ethylene glycol is converted to toxic organic acids including glycolate and oxalate, producing anion-gap acidosis, hypocalcemia, crystal deposition, and AKI. Fomepizole is the preferred antidote; severe poisoning may require hemodialysis.

237. B. Severe metformin poisoning or metformin-associated lactic acidosis can warrant extracorporeal treatment, particularly with profound acidemia, high lactate, shock, impaired kidney function, or failure of supportive measures. Intermittent hemodialysis provides effective metformin and lactate clearance and bicarbonate delivery.

238. C. Drug clearance can change quickly during AKI and is further altered by intermittent dialysis or CRRT. Dosing should be individualized to indication, pharmacokinetics, kidney-function trajectory, extracorporeal clearance, and when available therapeutic drug monitoring.

239. A. AKI is a risk marker for subsequent CKD, recurrent AKI, cardiovascular disease, and death even when creatinine improves. Follow-up should reassess kidney function, albuminuria, blood pressure, medications, and recovery trajectory.

240. E. Kidney replacement therapy in AKI is initiated for clinical indications such as refractory hyperkalemia, severe acidosis, pulmonary edema or other unmanageable volume overload, selected intoxications, or uremic complications. No single creatinine or BUN threshold should dictate initiation in an otherwise stable patient.