COMPREHENSIVE MOCK EXAM 7

Supplemental to: Nephrology Board Review 2026-2027

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ABIM Nephrology Certification Examination — 240 questions, A–E single-best-answer format for this MEDREV parent book, domain-weighted to the January 2026 ABIM blueprint

Board-Review Questions

1. During inpatient nephrology rounds, a 45-year-old woman with ADPKD has chronic flank pain but suddenly develops fever, focal renal tenderness, and inflammatory markers. Urinalysis is only mildly abnormal. Given this presentation, what complication should be considered?

A. Renal artery fibromuscular dysplasia, which produces fever and local tenderness when arterial beading progresses.

B. Nephrogenic diabetes insipidus, because impaired urinary concentration routinely causes inflammatory fever and focal kidney pain.

C. Simple cyst expansion alone, because fever and inflammatory markers are expected whenever a renal cyst enlarges gradually.

D. Infection of a renal cyst, which can produce systemic infection despite limited communication with the collecting system.

E. Acute glomerulonephritis, because focal tenderness in ADPKD is most specifically caused by diffuse immune-complex glomerular inflammation.

2. At a kidney clinic follow-up visit, a 56-year-old man with multiple myeloma has sodium 124 mEq/L by an indirect ion-selective electrode, but measured serum osmolality is normal and a direct whole-blood sodium is 138 mEq/L. Given this presentation, what explains the discrepancy?

A. Pseudohyponatremia from marked expansion of the nonaqueous plasma fraction by paraproteins.

B. Cerebral salt wasting, which would cause true sodium loss, extracellular volume depletion, and concordantly low direct sodium values.

C. Primary polydipsia, which produces genuine hypotonic hyponatremia.

D. SIADH from the plasma-cell disorder, which would produce true hypotonicity and a similarly low sodium on direct measurement.

E. Hyperglycemic translocation, which would increase measured osmolality and lower sodium by both direct and indirect methods.

3. During an urgent nephrology assessment, a 67-year-old woman has eGFR 42 mL/min/1.73 m2 and urine albumin-creatinine ratio 520 mg/g. Why is albuminuria important in CKD staging?

A. Albuminuria predicts only bleeding risk during dialysis and has little relationship to future kidney function or cardiovascular outcomes.

B. Albuminuria is used only to diagnose nephrotic syndrome and does not affect CKD prognosis once eGFR has been measured.

C. Albuminuria replaces GFR in CKD classification because eGFR is no longer relevant when urine albumin exceeds 300 mg/g.

D. Albuminuria is primarily a marker of urinary infection and should not be used for risk stratification in stable chronic kidney disease.

E. Albuminuria adds prognostic information for kidney failure and cardiovascular risk beyond the GFR category alone.

4. At a hypertension clinic evaluation, a 78-year-old man arrives with blood pressure 230/130 mm Hg, confusion, papilledema, and acute kidney injury. Given this presentation, what distinguishes this from severe asymptomatic hypertension?

A. The absolute systolic pressure alone defines emergency, so symptoms and organ injury are irrelevant to treatment intensity.

B. There is acute hypertension-mediated target-organ injury, so controlled intravenous blood-pressure reduction is required.

C. Papilledema indicates chronic uncomplicated hypertension and supports outpatient oral medication adjustment without monitoring.

D. Any blood pressure above 180/120 mm Hg should be normalized to below 120/80 within minutes regardless of organ perfusion.

E. Acute kidney injury excludes hypertensive emergency because renal dysfunction cannot result from severe blood-pressure elevation.

5. During a hospital nephrology consultation, a 41-year-old woman has proliferative glomerulonephritis and persistently depressed C3 without evidence of immunoglobulin deposition. Based on these findings, which category should be considered?

A. Primary membranous nephropathy, which is characterized by dominant mesangial C3 deposition without immunoglobulin.

B. Anti-GBM disease, because linear IgG staining is absent when complement regulation is abnormal.

C. Minimal change disease, because podocyte injury causes isolated C3 deposition without immunoglobulin in glomeruli.

D. Thin basement membrane disease, because persistent complement consumption is a defining feature of collagen IV thinning.

E. C3 glomerulopathy from dysregulation of the alternative complement pathway.

6. During an outpatient nephrology consultation, a stable patient has chronic sodium values of 128-131 mEq/L. During a supervised water-loading test, urine can dilute appropriately, and sodium rises with water restriction but then settles near 130 mEq/L. Based on these findings, which concept best fits?

A. Fixed SIADH, in which urine cannot appropriately dilute because vasopressin remains inappropriately active across the tested range.

B. Nephrogenic diabetes insipidus, which causes hypernatremia tendency and persistent dilute urine.

C. A reset osmostat in which vasopressin regulation remains intact but operates around a lower sodium set point.

D. Pseudohyponatremia, which reflects laboratory methodology and should not respond physiologically to water restriction or water loading.

E. Cerebral salt wasting, which is characterized by continuing renal sodium loss, hypovolemia, and progressive.

7. At a metabolic stone clinic visit, a 63-year-old woman with Sjögren syndrome has a normal-anion-gap metabolic acidosis, urine pH 6.2 during systemic acidosis, and recurrent calcium phosphate stones. Given this presentation, what tubular disorder is present?

A. Distal renal tubular acidosis due to impaired collecting-duct hydrogen secretion.

B. Respiratory alkalosis, which lowers serum bicarbonate by compensation but does not produce the renal stone pattern described.

C. Proximal renal tubular acidosis, in which urine can acidify below 5.5 once serum bicarbonate falls sufficiently.

D. Primary hyperaldosteronism, which promotes hydrogen secretion and metabolic alkalosis.

E. Type 4 renal tubular acidosis, which is usually associated with hyperkalemia and preserved ability to lower urine pH.

8. During a dialysis-unit assessment, a 74-year-old man in septic shock requires vasopressors and has severe AKI with an indication for dialysis. Based on these findings, which modality may be favored when hemodynamic instability is profound?

A. Hemoperfusion alone is preferred because it replaces all renal solute and fluid functions without hemodynamic effects.

B. No dialysis can be performed during vasopressor therapy, so life-threatening hyperkalemia must be managed without extracorporeal removal.

C. Peritoneal dialysis is mandatory in every ICU patient because extracorporeal circuits cannot be used in shock.

D. Continuous kidney replacement therapy because slower continuous fluid and solute removal may be better tolerated.

E. High-efficiency intermittent hemodialysis with maximal ultrafiltration over one hour because rapid volume removal improves shock tolerance.

9. During an urgent nephrology assessment, a 37-year-old woman with CKD stage G3b has rising albuminuria but no symptoms. Based on these findings, which approach best estimates the need for nephrology referral and kidney replacement planning?

A. Refer only when eGFR is below 10 mL/min/1.73 m2 because earlier risk prediction does not influence treatment or preparation for kidney failure.

B. Base referral only on patient age because kidney-failure risk equations are not designed to incorporate laboratory measures or CKD severity.

C. Use the overall risk profile, including GFR, albuminuria, trajectory, and a validated kidney-failure risk equation when applicable.

D. Defer for uremic symptoms because asymptomatic CKD cannot progress rapidly enough to justify advance planning or specialist involvement.

E. Use serum creatinine alone because albuminuria and the rate of change add little information once an eGFR category has been assigned.

10. At a multidisciplinary renal conference, how do SGLT2 inhibitors alter tubuloglomerular feedback in diabetic or proteinuric CKD?

A. They prevent glucose filtration at the glomerulus, which lowers urine glucose without changing tubular sodium handling.

B. They block NKCC2 in the thick ascending limb, making their primary action identical to a loop diuretic.

C. They directly constrict the efferent arteriole by increasing angiotensin II, thereby raising intraglomerular pressure and proteinuria.

D. They reduce proximal sodium-glucose reabsorption, increase sodium delivery to the macula densa, and lower intraglomerular pressure.

E. They inhibit ENaC in the collecting duct, so their principal effect is potassium retention through an amiloride-like mechanism.

11. During a dialysis-unit assessment, a 59-year-old woman with eGFR 7 mL/min/1.73 m2 is eating well, has controlled potassium and volume status, and has no uremic symptoms. Based on these findings, which statement about starting dialysis is most accurate?

A. Dialysis initiation should be driven by symptoms, refractory complications, and overall clinical context.

B. Dialysis should never start until eGFR reaches zero because residual filtration makes extracorporeal therapy harmful before complete anuria.

C. Dialysis is mandatory as soon as eGFR falls below 10 mL/min/1.73 m2 even when the patient is asymptomatic and complications are controlled.

D. Serum creatinine alone determines the start date because symptoms and nutrition are too subjective to influence kidney replacement decisions.

E. Early dialysis at a high eGFR uniformly improves survival, so symptoms and reversible medical management should not delay treatment.

12. During an outpatient nephrology consultation, a 70-year-old man with CKD is receiving a narrow-therapeutic-index drug cleared by the kidneys. Creatinine-based eGFR may be inaccurate because of severe sarcopenia. Given this presentation, what is the best dosing principle?

A. Give the full standard dose because underdosing is always more dangerous than accumulation in patients with chronic kidney disease.

B. Use urine albumin excretion as the sole dose-adjustment variable because albuminuria directly measures clearance of small therapeutic molecules.

C. Avoid all renally cleared medications in CKD because no estimate of kidney function is reliable enough to support safe pharmacologic dosing.

D. Use the serum creatinine concentration alone because drug clearance is directly proportional to creatinine regardless of age, body size, or muscle mass.

E. Use the most appropriate validated kidney-function estimate for the drug and consider confirmatory filtration assessment when dosing consequences are high.

13. During inpatient nephrology rounds, a 61-year-old man taking lithium has persistent polyuria, sodium 149 mEq/L, and urine osmolality 130 mOsm/kg. Desmopressin produces little change. Based on these findings, which therapy is most directly useful if lithium cannot immediately be stopped?

A. Use amiloride to reduce lithium entry through epithelial sodium channels and lessen nephrogenic diabetes insipidus.

B. Use acetazolamide to produce bicarbonaturia because proximal sodium loss specifically reverses collecting-duct resistance to vasopressin.

C. Use desmopressin as sole therapy because a poor response to the test dose indicates that higher doses will reliably normalize urine concentration.

D. Use spironolactone because mineralocorticoid receptor blockade directly restores collecting-duct aquaporin responsiveness to vasopressin.

E. Give hypertonic saline to stimulate thirst because increasing serum tonicity is the preferred chronic treatment for lithium-induced polyuria.

14. At a kidney clinic follow-up visit, a 44-year-old man with severe hyperkalemia receives insulin and glucose. Given this presentation, what is the purpose of this therapy?

A. Convert potassium chemically into sodium, thereby lowering total-body potassium within minutes.

B. Remove potassium through glomerular filtration, making insulin effective only when GFR is normal.

C. Shift potassium into cells rapidly through stimulation of sodium-potassium ATPase without removing potassium from the body.

D. Bind potassium within the colon and excrete it in stool over several hours, which is insulin's principal mechanism.

E. Stabilize the cardiac membrane directly in the same way as intravenous calcium.

15. During a dialysis-unit assessment, a stable thrice-weekly hemodialysis patient has inadequate small-solute clearance because treatment sessions are repeatedly shortened. Based on these findings, which concept is most relevant to assessing dialysis dose?

A. Dialysate sodium concentration alone determines adequacy because urea clearance is independent of blood flow, dialyzer performance, and treatment time.

B. Urine albumin-creatinine ratio is the standard measure of hemodialysis adequacy even in anuric patients because albumin reflects extracorporeal clearance.

C. A urea-based adequacy measure such as Kt/V reflects delivered clearance relative to the patient's urea distribution volume.

D. Blood pressure during dialysis is the only validated adequacy metric, so a normal pressure proves that solute removal is sufficient.

E. Serum creatinine before dialysis alone fully measures delivered dialysis dose and makes treatment time or clearance calculations unnecessary.

16. At a hypertension clinic evaluation, a 44-year-old with hypertension and spontaneous hypokalemia has suppressed renin. Based on these findings, which screening test is most appropriate for primary aldosteronism?

A. Perform a water-deprivation test because aldosterone excess primarily presents by impaired urinary concentration.

B. Measure plasma aldosterone and renin and interpret the aldosterone-to-renin relationship in the appropriate clinical and medication context.

C. Measure serum cortisol only because primary aldosteronism is diagnosed by loss of diurnal cortisol variation.

D. Obtain renal biopsy because mineralocorticoid excess is confirmed by arteriolar hyalinosis on kidney histology.

E. Measure urinary catecholamines only because hypokalemic hypertension is most specific for pheochromocytoma.

17. At a transplant-nephrology follow-up visit, a 77-year-old woman with advanced CKD has an available living donor and is otherwise a good transplant candidate. Given this presentation, what strategy offers an important potential advantage?

A. Defer until severe uremic complications are irreversible because transplantation is effective only after prolonged dialysis dependence.

B. Begin dialysis solely to establish transplant eligibility because preemptive transplantation is prohibited in kidney failure.

C. Avoid living donation because deceased-donor kidneys consistently provide longer graft survival in suitable candidates.

D. Delay transplantation until many years of dialysis have passed because dialysis exposure improves subsequent graft survival.

E. Preemptive kidney transplantation before maintenance dialysis when feasible.

18. During an outpatient nephrology consultation, a 44-year-old with long-standing analgesic overuse has CKD, sterile pyuria, and papillary calcifications. Given this presentation, what pathologic process is most characteristic?

A. Diffuse crescentic glomerulonephritis, because analgesics primarily cause immune-complex rupture of glomerular capillary walls.

B. Renal amyloidosis, because papillary calcification is the typical result of systemic amyloid deposition in the medulla.

C. Chronic tubulointerstitial nephritis with renal papillary necrosis related to analgesic exposure.

D. Isolated renal-vein thrombosis, because chronic analgesic use produces papillary ischemia only through venous occlusion.

E. Minimal change disease alone, because analgesic nephropathy is defined by selective podocyte effacement without interstitial injury.

19. At a transplant-nephrology follow-up visit, a transplant recipient has slowly declining graft function years after surgery, proteinuria, transplant glomerulopathy, and persistent donor-specific antibodies. Given this presentation, what process should be considered?

A. Delayed graft function from ischemia-reperfusion injury, which is an early postoperative phenomenon and does not explain chronic transplant glomerulopathy.

B. Acute urinary retention, which can impair graft function but does not cause donor-specific antibody or glomerular basement-membrane duplication.

C. Chronic active antibody-mediated rejection with cumulative microvascular injury.

D. Primary nephrogenic diabetes insipidus, which affects water handling but does not cause chronic alloimmune glomerular injury.

E. Hyperacute rejection, which occurs at reperfusion.

20. At a transplant-nephrology follow-up visit, which kidney transplant recipient is at highest risk for primary CMV infection after transplantation?

A. A recipient and donor who are both seropositive, because preexisting immunity in both individuals eliminates all risk stratification.

B. A CMV-seronegative recipient receiving an organ from a CMV-seropositive donor.

C. CMV risk is unrelated to donor and recipient serostatus and depends only on the transplant kidney's cold ischemia time.

D. A seropositive recipient receiving from a seronegative donor, because prior recipient immunity creates the highest primary-infection risk.

E. A seronegative recipient receiving from a seronegative donor, because absence of donor virus produces the greatest viral inoculum.

21. During an urgent nephrology assessment, a 73-year-old woman with CKD has fatigue, low transferrin saturation, and low ferritin. Given this presentation, what is the most direct therapeutic issue to address?

A. Iron deficiency should be corrected using an oral or intravenous strategy appropriate to CKD stage, severity, tolerance, and treatment setting.

B. Iron should be avoided because any CKD-associated anemia represents iron overload from reduced renal excretion.

C. Chelation therapy is indicated because a low ferritin in CKD reflects iron trapped in tissues and predicts systemic iron toxicity.

D. Red-cell transfusion is preferred for stable iron deficiency because replenishing iron stores cannot improve hemoglobin in chronic kidney disease.

E. An ESA should always precede iron therapy because iron cannot support erythropoiesis unless exogenous erythropoietin has already been administered.

22. At a transplant-nephrology follow-up visit, a 69-year-old woman with a kidney transplant develops fever, weight loss, lymphadenopathy, and an EBV-associated B-cell proliferation. Given this presentation, what diagnosis is most likely?

A. Calcineurin nephrotoxicity, which causes renal vasoconstriction but not lymphadenopathy and clonal B-cell expansion.

B. Acute T-cell-mediated rejection, which is confined to graft-directed immune injury and does not explain systemic EBV-driven lymphoid proliferation.

C. Post-transplant lymphoproliferative disorder related to impaired immune control of EBV-infected B cells.

D. BK nephropathy, which affects renal tubular cells and does not characteristically produce B-cell tumors.

E. Recurrent FSGS, which presents with proteinuria.

23. During a hospital nephrology consultation, a 47-year-old woman with HIV has nephrotic proteinuria and rapidly progressive CKD. Biopsy shows segmental sclerosis with collapsing glomerular tufts. Based on these findings, which diagnosis fits best?

A. Minimal change disease, which lacks segmental sclerosis and glomerular collapse on light microscopy.

B. Collapsing focal segmental glomerulosclerosis associated with HIV and other podocyte stressors.

C. IgA nephropathy, which is defined by mesangial IgA deposition and usually presents with hematuria.

D. Acute interstitial nephritis, which is centered in the interstitium and does not produce collapsing glomerular lesions.

E. Membranous nephropathy, which is characterized by subepithelial deposits and capillary-wall thickening.

24. During an outpatient nephrology consultation, a nursing-home resident with poor access to water has sodium 166 mEq/L for several days but no seizures. Given this presentation, what is the safest correction strategy?

A. Normalize sodium within two hours because chronic brain adaptation makes cerebral edema unlikely during rapid free-water replacement.

B. Use hypertonic saline until urine becomes concentrated because adding sodium restores the medullary gradient before water can be given.

C. Replace free water gradually with frequent sodium measurements, accounting for ongoing losses and avoiding an unnecessarily rapid fall.

D. Use loop diuretics alone to lower sodium because natriuresis without water replacement corrects the free-water deficit most efficiently.

E. Withhold enteral water because gastrointestinal absorption is too unpredictable for controlled treatment of chronic hypernatremia.

25. At a hypertension clinic evaluation, a 69-year-old woman develops sudden flank pain, hematuria, severe hypertension, elevated LDH, and loss of perfusion to one kidney on imaging. Given this presentation, what diagnosis is most likely?

A. Acute interstitial nephritis, because interstitial eosinophilic inflammation causes abrupt complete loss of arterial flow.

B. Distal RTA, because alkaline urine causes renal arterial vasospasm and infarction.

C. Acute renal artery occlusion causing renal infarction and sudden loss of perfusion to the affected kidney.

D. Postrenal obstruction, because ureteral obstruction eliminates renal arterial perfusion immediately.

E. Minimal change disease, because nephrotic protein loss causes unilateral absent renal perfusion without thrombosis.

26. At a kidney clinic follow-up visit, a 80-year-old man with severe nephrotic syndrome develops sudden flank pain, hematuria, and worsening kidney function. Based on these findings, which complication should be considered?

A. Polycystic kidney rupture, because nephrotic-range proteinuria causes rapid formation of bilateral renal cysts.

B. Renal-vein thrombosis due to the hypercoagulable state of nephrotic syndrome.

C. Acute interstitial nephritis, because hypoalbuminemia directly causes eosinophilic inflammation of the renal interstitium.

D. Papillary necrosis, because urinary protein mechanically obstructs the papillae and causes sloughing in nephrotic syndrome.

E. Renal artery fibromuscular dysplasia, because urinary loss of anticoagulant proteins produces arterial beading.

27. During an urgent nephrology assessment, a 43-year-old woman with progressive CKD is likely to need kidney replacement therapy within the next year. Given this presentation, what is the best planning approach?

A. Place a tunneled dialysis catheter months in advance in every patient because catheters are the preferred permanent access for planned hemodialysis.

B. Exclude peritoneal dialysis if the patient has any prior abdominal surgery because previous surgery is an absolute contraindication to home dialysis.

C. Avoid discussing kidney replacement until the first life-threatening indication occurs because early education increases anxiety without changing outcomes.

D. Provide modality education early, discuss transplantation and conservative care, and plan access or peritoneal dialysis preparation before an emergency start.

E. Delay transplant referral until several months after dialysis begins because preemptive transplantation is not feasible in chronic kidney disease.

28. At a transplant-nephrology follow-up visit, a long-term transplant recipient develops diabetes, weight gain, osteoporosis, and skin fragility. Based on these findings, which immunosuppressant contributes to this cluster?

A. Trimethoprim-sulfamethoxazole, which causes the full chronic Cushing syndrome phenotype through mineralocorticoid receptor activation.

B. Mycophenolate alone, which most characteristically causes obesity, osteoporosis, and Cushingoid skin changes.

C. Chronic systemic glucocorticoid exposure causing metabolic, skeletal, and characteristic skin toxicity.

D. Belatacept, which directly stimulates adrenal cortisol secretion and therefore causes glucocorticoid toxicity.

E. Basiliximab induction months earlier, which causes permanent hypercortisolism and central weight gain in most recipients.

29. During a hospital nephrology consultation, a 65-year-old woman with AKI has severe metabolic acidosis with hemodynamic instability despite treatment of the cause and appropriate medical therapy. Given this presentation, what renal support may be required?

A. Start dialysis only if potassium is normal because acidosis without hyperkalemia is never an indication for extracorporeal therapy.

B. Delay intervention until uremic pericarditis develops because acid-base complications do not influence dialysis timing.

C. No renal support can correct acidosis because dialysis removes bicarbonate and always lowers blood pH.

D. Give chronic acetazolamide because bicarbonate wasting is the definitive treatment for life-threatening metabolic acidosis.

E. Kidney replacement therapy when severe metabolic acidosis remains refractory despite appropriate medical treatment.

30. At a hypertension clinic evaluation, a 76-year-old man taking ibuprofen daily for arthritis develops worsening blood pressure and edema despite previously stable therapy. Given this presentation, what is the best explanation?

A. NSAIDs increase renal prostaglandin synthesis, producing natriuresis and therefore a compensatory rise in blood pressure.

B. The blood-pressure rise is unrelated to NSAIDs because cyclooxygenase inhibition has no meaningful effect on renal sodium handling.

C. NSAIDs cause osmotic diuresis by blocking proximal glucose reabsorption, leading to reflex hypertension from free-water loss.

D. Ibuprofen acts as a mineralocorticoid receptor agonist and directly stimulates aldosterone secretion independent of kidney perfusion.

E. NSAID-mediated inhibition of renal prostaglandins promotes sodium retention and can blunt the effect of several antihypertensive drugs.

31. At a hypertension clinic evaluation, a 39-year-old woman with hypertension and hypokalemic metabolic alkalosis has suppressed renin and suppressed aldosterone. Based on these findings, which disorder is most likely?

A. Liddle syndrome from constitutive epithelial sodium channel activity causing sodium retention and potassium wasting.

B. Gitelman syndrome, which causes renal salt wasting and secondary hyperreninemic hyperaldosteronism.

C. Bartter syndrome, which causes salt wasting with increased renin and aldosterone.

D. Renovascular hypertension, which typically activates renin and secondary aldosterone.

E. Primary hyperaldosteronism, which should suppress renin but usually produces an inappropriately elevated aldosterone concentration.

32. At a transplant-nephrology follow-up visit, a 43-year-old woman with a kidney transplant develops fever and graft tenderness with pyuria and bacteriuria. Given this presentation, what diagnosis should be considered?

A. Allograft pyelonephritis, which can threaten graft function and requires prompt antimicrobial treatment.

B. Calcineurin toxicity is most likely because tacrolimus routinely causes fever and bacteriuria.

C. BK nephropathy is proven because bacterial pyuria never occurs in a denervated transplanted kidney.

D. Acute cellular rejection is proven because infection cannot cause graft tenderness after transplantation.

E. Recurrent membranous nephropathy is the usual cause of fever, pyuria, and positive urine culture after transplant.

33. During a dialysis-unit assessment, a long-term peritoneal dialysis patient develops recurrent bowel obstruction, weight loss, ultrafiltration failure, and a thickened calcified peritoneal membrane encasing bowel loops. Given this presentation, what is the diagnosis?

A. Uremic gastroenteropathy, which may cause nausea but does not create a thickened calcified peritoneal membrane with recurrent obstruction.

B. Dialysis disequilibrium syndrome, which is an acute cerebral osmotic disorder of hemodialysis.

C. Simple bacterial peritonitis, which can cause cloudy effluent but does not typically produce a chronic fibrotic cocoon around bowel loops.

D. Encapsulating peritoneal sclerosis, a rare severe complication of long-term peritoneal dialysis.

E. Nephrogenic systemic fibrosis, which is associated with certain gadolinium exposures and causes systemic skin fibrosis.

34. At a hypertension clinic evaluation, a 51-year-old has hypertension despite three medications, including a diuretic. Before labeling the condition truly resistant, what should be done?

A. Discontinue the diuretic because volume control interferes with determining whether hypertension is resistant.

B. Add a fifth drug before checking adherence because medication nonadherence does not contribute meaningfully to apparent resistance.

C. Assume secondary hypertension is present and proceed directly to adrenal vein sampling without confirming medication use or measurement quality.

D. Confirm adherence, accurate measurement, an appropriate regimen and doses, and persistent elevation with out-of-office readings.

E. Use only the highest office pressure from the last year because repeated or out-of-office measurements can obscure true resistance.

35. During a hospital nephrology consultation, a hospitalized patient develops anemia, thrombocytopenia, schistocytes, elevated LDH, and AKI. Based on these findings, which renal vascular syndrome should be considered first?

A. Renal papillary necrosis, because sloughed papillae cause systemic hemolysis and platelet consumption.

B. Acute interstitial nephritis, because interstitial inflammation characteristically causes microangiopathic hemolysis.

C. Thrombotic microangiopathy causing endothelial injury and microvascular platelet-rich thrombosis.

D. Prerenal azotemia, because reduced perfusion alone produces schistocytes and severe thrombocytopenia.

E. Minimal change disease, because podocyte injury directly fragments red cells and consumes platelets in glomerular capillaries.

36. During an outpatient nephrology consultation, a 46-year-old man with potassium 7.2 mEq/L has peaked T waves and QRS widening. Based on these findings, which intervention should be given immediately to reduce the risk of malignant arrhythmia?

A. Sodium polystyrene sulfonate alone because gastrointestinal potassium removal acts quickly enough to stabilize the myocardium within minutes.

B. Insulin without glucose in every patient because creating hypoglycemia enhances intracellular potassium uptake and is necessary for cardiac stabilization.

C. Loop diuretic alone because increasing urinary potassium excretion immediately reverses conduction abnormalities before total-body potassium changes.

D. Restrict dietary potassium and recheck the value the next morning because ECG changes do not independently increase the urgency of treatment.

E. Intravenous calcium to stabilize the cardiac membrane while potassium-lowering therapies are started.

37. During inpatient nephrology rounds, a 57-year-old woman with CKD has phosphate 6.2 mg/dL, elevated parathyroid hormone, and low-normal calcium. Given this presentation, what is the central pathophysiologic process?

A. Reduced phosphate excretion and reduced calcitriol production contribute to secondary hyperparathyroidism and CKD-mineral bone disorder.

B. Excess calcitriol production increases intestinal calcium absorption and suppresses PTH, making progressive hyperparathyroidism unlikely in CKD.

C. Renal phosphate wasting is the dominant early abnormality, so hyperphosphatemia reflects excessive dietary phosphate.

D. Autonomous parathyroid adenoma is required for any PTH elevation in CKD because kidney failure does not directly alter mineral metabolism.

E. Increased kidney production of active vitamin D raises fibroblast growth factor 23 only after dialysis begins and has no role before kidney failure.

38. At a transplant-nephrology follow-up visit, several weeks after transplant, a recipient develops leg swelling and graft dysfunction. Imaging shows a fluid collection compressing the transplant ureter and iliac vein. Given this presentation, what is the likely collection?

A. A postoperative lymphocele from disrupted lymphatic channels compressing nearby transplant structures.

B. A renal cyst inherited from the recipient, because recipient germline cysts routinely appear within the donor graft after several weeks.

C. A pancreatic pseudocyst, because kidney transplantation causes pancreatic duct disruption and peri-graft enzyme collections.

D. A urinoma is excluded because all postoperative fluid collections are lymphatic once the incision has healed.

E. An adrenal hematoma, because transplant surgery routinely places the donor adrenal gland next to the iliac vessels.

39. At a transplant-nephrology follow-up visit, a 40-year-old man with a kidney transplant on tacrolimus develops an acute rise in creatinine with high trough level and no evidence of rejection or obstruction. Given this presentation, what hemodynamic effect can contribute?

A. Complete blockade of proximal sodium secretion, which produces Fanconi syndrome as the usual cause of tacrolimus AKI.

B. Renal-vein thrombosis from direct platelet activation, which is expected whenever the tacrolimus trough is above target.

C. Selective efferent arteriolar dilation identical to ACE inhibition, which is the only renal vascular effect of calcineurin inhibitors.

D. Immediate immune-complex deposition caused by tacrolimus acting as a foreign antigen in every treated recipient.

E. Calcineurin inhibitor–mediated renal vasoconstriction that can reversibly reduce graft filtration.

40. At a multidisciplinary renal conference, a 42-year-old man with AKI of unclear cause has no urinary symptoms. Based on these findings, which imaging test is commonly used to evaluate for hydronephrosis?

A. Renal ultrasonography because it can identify collecting-system dilation without nephrotoxic contrast.

B. Bone scintigraphy because skeletal tracer accumulation is specific for acute urinary obstruction.

C. Echocardiography because left ventricular filling pressure determines whether ureters are dilated.

D. Noncontrast chest CT because lung imaging is the most sensitive way to detect ureteral obstruction.

E. Renal biopsy because tissue sampling directly visualizes hydronephrosis and bladder outlet obstruction.

41. During a hospital nephrology consultation, a 51-year-old has progressive CKD, bland urine sediment, hyperuricemia, and a strong family history of early gout and kidney failure. Kidneys are small rather than cystic. Based on these findings, which inherited category is most likely?

A. Thin basement membrane disease, which usually causes isolated microscopic hematuria.

B. Autosomal dominant tubulointerstitial kidney disease, which can cause bland CKD and early hyperuricemia or gout.

C. Autosomal dominant polycystic kidney disease, which usually produces enlarged cystic kidneys and characteristic extrarenal manifestations.

D. Alport syndrome, which typically features persistent glomerular hematuria with hearing or ocular abnormalities.

E. Primary hyperoxaluria, which more often causes nephrocalcinosis and recurrent calcium oxalate stones from oxalate overproduction.

42. During an outpatient nephrology consultation, urine microscopy shows numerous dysmorphic erythrocytes and acanthocytes in a patient with hematuria. Given this presentation, what do these findings suggest?

A. Prerenal azotemia, because low renal blood flow mechanically deforms red cells as they pass through intact glomeruli.

B. Nephrogenic diabetes insipidus, because dilute urine itself creates acanthocytes and proves collecting-duct disease.

C. Renal papillary necrosis, because papillary sloughing produces predominantly dysmorphic glomerular erythrocytes.

D. Lower urinary tract bleeding, because bladder and ureteral bleeding characteristically distorts red cells into acanthocytes.

E. A glomerular source of bleeding caused by red cells traversing a damaged filtration barrier.

43. During inpatient nephrology rounds, a 75-year-old woman with acute adrenal crisis has sodium 122 mEq/L, potassium 5.8 mEq/L, hypotension, and abdominal pain. How does cortisol deficiency contribute to the hyponatremia?

A. Cortisol deficiency directly blocks collecting-duct water reabsorption, producing a free-water diuresis that secondarily lowers sodium concentration.

B. Loss of cortisol-mediated suppression of vasopressin promotes water retention, while mineralocorticoid deficiency can add renal sodium loss.

C. Low cortisol raises serum tonicity through glucosuria, causing a hypertonic form of hyponatremia similar to uncontrolled diabetes mellitus.

D. Adrenal failure suppresses vasopressin completely, so hyponatremia is explained only by excessive oral water intake in affected patients.

E. Mineralocorticoid deficiency causes avid renal sodium retention, so the low sodium results from sodium moving into cells.

44. At a transplant-nephrology follow-up visit, a 65-year-old woman with a kidney transplant develops declining urine output and hydronephrosis on ultrasound. Given this presentation, what complication is most likely?

A. Acute T-cell rejection alone, because rejection routinely dilates the collecting system and causes hydronephrosis.

B. BK viremia alone, because circulating BK virus always causes visible obstruction of the collecting system.

C. CMV syndrome, because leukopenia and viremia are required to produce post-transplant hydronephrosis.

D. Urinary outflow obstruction of the transplant ureter, requiring prompt urologic evaluation.

E. Calcineurin toxicity, because tacrolimus directly obstructs the ureter and produces mechanical hydronephrosis.

45. During an intensive-care nephrology consultation, a 49-year-old woman is resuscitated after prolonged hemorrhagic shock and later develops oliguric AKI with granular casts. Based on these findings, which pathophysiologic mechanism best explains these findings?

A. Ischemic acute tubular injury after sustained renal hypoperfusion.

B. Chronic membranous nephropathy caused by transfused blood products during resuscitation.

C. Acute interstitial nephritis from endogenous catecholamines, which produces white-cell casts after shock.

D. Bilateral renal-vein thrombosis from low flow, which is expected in nearly every episode of hemorrhage.

E. Selective immune destruction of podocytes, which is the usual consequence of hemorrhagic shock.

46. At a multidisciplinary renal conference, a 60-year-old man on loop diuretics has suspected prerenal physiology. Based on these findings, which laboratory index may be less directly affected by loop-induced natriuresis?

A. Fractional excretion of urea, although it also has limitations and should not be interpreted in isolation.

B. Fractional excretion of sodium, because loop diuretics have no effect on sodium transport in the nephron.

C. Serum chloride alone, because a normal chloride concentration reliably proves prerenal AKI.

D. Serum phosphate alone, because phosphate excretion uniquely distinguishes prerenal from tubular injury.

E. Urine protein concentration, because prerenal physiology always causes nephrotic-range proteinuria.

47. During an intensive-care nephrology consultation, an ICU patient has thrombocytopenia, schistocytes, hemolysis, and AKI. Based on these findings, which diagnostic framework is most appropriate?

A. Diagnose acute tubular necrosis alone because schistocytes and thrombocytopenia are routine consequences of tubular epithelial injury.

B. Assume disseminated minimal change disease because nephrotic podocyte injury causes microangiopathic hemolysis in the ICU.

C. Evaluate urgently for thrombotic microangiopathy, including TTP, Shiga toxin, complement-mediated disease, severe hypertension, drugs, and pregnancy-related causes.

D. Ignore the blood smear because red-cell fragmentation does not provide information about renal vascular injury.

E. Treat as isolated iron deficiency because low hemoglobin with AKI is usually nutritional.

48. During an outpatient nephrology consultation, a 34-year-old man with severe ethylene glycol poisoning has high-anion-gap acidosis, an elevated osmolar gap, and calcium oxalate crystals. Based on these findings, which therapy is appropriate in severe poisoning?

A. Block alcohol dehydrogenase with fomepizole and use hemodialysis when severe acidosis, kidney injury, or high toxic burden warrants rapid removal.

B. Give ethanol only after the toxin has fully metabolized because alcohol dehydrogenase blockade is useful only after oxalate forms.

C. Start high-dose glucocorticoids because the renal crystals are immune complexes.

D. Use sodium chloride alone because ethylene glycol toxicity is caused by volume depletion.

E. Administer desmopressin because reducing urine output prevents ethylene glycol from reaching the renal tubules.

49. During inpatient nephrology rounds, a 45-year-old woman with CKD needs iodinated contrast for an urgent procedure. Based on these findings, which preventive principle is most appropriate for a high-risk patient without pulmonary edema?

A. Use the lowest necessary contrast dose, avoid concurrent nephrotoxins, and provide appropriate isotonic volume expansion when clinically indicated.

B. Withhold needed imaging solely because CKD is present, since contrast-associated kidney injury is inevitable at any reduced eGFR.

C. Use routine high-dose loop diuretics before contrast because forced diuresis without matched hydration prevents tubular injury in most high-risk patients.

D. Give prophylactic dialysis immediately after contrast because extracorporeal removal reliably prevents contrast-associated AKI in nondialysis CKD.

E. Restrict all fluids before the procedure because intravascular volume contraction reduces delivery of contrast to the kidney and lowers nephrotoxicity.

50. At a kidney clinic follow-up visit, a 56-year-old man with a monoclonal gammopathy has proteinuria and kidney dysfunction, but does not meet hematologic criteria for overt myeloma. Why can the clone still require treatment?

A. Proteinuria proves diabetic nephropathy, so the monoclonal protein should be ignored unless lytic bone lesions develop.

B. Any monoclonal gammopathy with kidney disease is benign MGUS because glomerular injury requires a large tumor burden.

C. A small clone can produce a nephrotoxic monoclonal immunoglobulin causing monoclonal gammopathy of renal significance.

D. Kidney injury cannot be caused by a monoclonal protein until the patient meets full criteria for symptomatic multiple myeloma.

E. The kidney removes monoclonal proteins completely, so a small plasma-cell clone cannot create tissue deposits or complement dysregulation.

51. During an urgent nephrology assessment, a 67-year-old woman with hyponatremia has low serum osmolality, urine osmolality 80 mOsm/kg, and a diet consisting mainly of beer with little protein or salt. Given this presentation, what mechanism limits water excretion?

A. Very low daily osmole excretion limits the maximum volume of dilute urine that can be produced.

B. Persistent high vasopressin is required, because low-solute hyponatremia cannot occur when the urine is appropriately dilute.

C. Severe hyperglycemia shifts water intracellularly, and the low urine osmolality reflects glucose-driven osmotic diuresis.

D. Glomerular filtration must be severely reduced, because normal kidneys can excrete unlimited water regardless of dietary osmole intake.

E. Aldosterone excess causes sodium retention and obligatory water loss, which lowers serum sodium when dietary protein intake is restricted.

52. At a hypertension clinic evaluation, a 42-year-old has hypertension, flank fullness, enlarged kidneys with innumerable bilateral cysts, and a family history of subarachnoid hemorrhage. Based on these findings, which diagnosis is most likely?

A. Autosomal dominant polycystic kidney disease with risk of intracranial aneurysm in selected families.

B. Medullary sponge kidney, which causes collecting-duct ectasia and stones but not massively enlarged cystic kidneys with this inheritance pattern.

C. Simple renal cysts, which are common with age but do not explain diffuse bilateral cyst burden, hypertension, and the family history.

D. Acquired cystic kidney disease, which arises mainly in advanced CKD and produces small to normal kidneys.

E. Autosomal recessive polycystic kidney disease, which usually presents in infancy or childhood with congenital hepatic fibrosis.

53. During a hospital nephrology consultation, a hospitalized patient with sudden anuria has a distended bladder. Given this presentation, what is the fastest bedside diagnostic and therapeutic step?

A. Start loop diuretics before checking for retention because increased urine production will mechanically open any obstruction.

B. Begin hemodialysis before attempting decompression because obstruction cannot be treated until uremic solutes are removed.

C. Assess bladder retention and place a urinary catheter if appropriate to relieve lower urinary tract obstruction.

D. Perform kidney biopsy immediately because anuria is almost always caused by rapidly progressive glomerulonephritis.

E. Give hypertonic saline because bladder distention reflects severe hyponatremia.

54. During an outpatient nephrology consultation, a 52-year-old man with weakness has potassium 2.9 mEq/L. Twenty-four-hour urinary potassium excretion is high despite hypokalemia. Given this presentation, what does this indicate?

A. Pseudohypokalemia from sample handling, because falsely low serum potassium is defined by excessive urinary potassium loss in the collected specimen.

B. Normal potassium homeostasis, because urinary potassium excretion does not change in response to a low serum potassium concentration.

C. A transcellular potassium shift alone, because intracellular uptake necessarily increases urinary potassium excretion regardless of aldosterone activity.

D. Renal potassium wasting with inappropriate urinary potassium loss despite systemic potassium depletion.

E. A gastrointestinal potassium loss, because the kidney should continue excreting large amounts of potassium when total-body stores are depleted.

55. At a transplant-nephrology follow-up visit, a kidney transplant recipient taking mycophenolate plans pregnancy. Given this presentation, what medication issue is most important?

A. Mycophenolate is preferred during pregnancy because it prevents congenital malformations more effectively than other antimetabolites.

B. All immunosuppression should be stopped before conception because maintenance therapy is more dangerous to the fetus than rejection.

C. Mycophenolate is teratogenic and should be replaced with a pregnancy-compatible immunosuppressive plan before conception under transplant supervision.

D. Tacrolimus and every calcineurin inhibitor are absolutely incompatible with pregnancy, making graft nephrectomy necessary before conception.

E. Pregnancy is universally contraindicated after kidney transplantation regardless of graft stability or medication regimen.

56. During a dialysis-unit assessment, a 74-year-old man with very high blood urea nitrogen begins hemodialysis for the first time and develops headache, nausea, confusion, and seizures during treatment. Given this presentation, what is the likely diagnosis?

A. Air embolism, which causes abrupt cardiopulmonary or focal neurologic symptoms and is not specifically linked to a very high initial urea level.

B. Hemolysis from dialysate contamination, which more typically causes chest or back pain, hyperkalemia, and pink plasma.

C. Uremic pericarditis, which presents primarily with positional chest pain or a friction rub.

D. Dialysis disequilibrium syndrome from rapid reduction of plasma osmolality causing cerebral edema.

E. Calciphylaxis, which produces painful ischemic skin lesions.

57. At a transplant-nephrology follow-up visit, months after transplantation, a recipient develops worsening hypertension, graft dysfunction, and a bruit over the allograft. Based on these findings, which diagnosis should be considered?

A. Minimal change disease, which can cause proteinuria but does not produce a vascular bruit over the allograft.

B. Renal-vein thrombosis, which is usually an early acute event and does not typically cause a chronic arterial bruit.

C. BK cystitis, which causes dysuria and hematuria but not a renal-artery bruit with refractory hypertension.

D. Ureteral reflux alone, which causes recurrent infection.

E. Transplant renal artery stenosis causing renovascular hypertension and graft hypoperfusion.

58. During a dialysis-unit assessment, a 48-year-old man on chronic lithium presents with confusion, tremor, ataxia, AKI, and a markedly elevated lithium level. When is hemodialysis especially useful?

A. Only after kidney function fully recovers, because dialysis is contraindicated while lithium clearance is reduced.

B. Never, because lithium is highly protein bound and has a very large inaccessible volume of distribution, so dialysis cannot remove it.

C. Only if serum sodium is low, because dialysis removes lithium exclusively by exchanging it for extracellular sodium.

D. When severe neurologic toxicity, impaired renal clearance, or a high lithium burden makes extracorporeal removal necessary.

E. Only for isolated nephrogenic diabetes insipidus without systemic toxicity, because dialysis primarily restores collecting-duct vasopressin responsiveness.

59. During an intensive-care nephrology consultation, a 59-year-old woman with decompensated cirrhosis develops AKI, bland urine sediment, low urine sodium, and no shock or nephrotoxin exposure; kidney function does not improve after appropriate volume assessment. Given this presentation, what syndrome should be considered?

A. Nephrogenic diabetes insipidus, because low urine sodium is specific for vasopressin resistance.

B. Postrenal obstruction, because ascites mechanically blocks both ureters in most patients with decompensated cirrhosis.

C. Acute glomerulonephritis, because cirrhosis routinely produces red-cell casts even when sediment is bland.

D. Bilateral renal cortical necrosis, because any AKI in cirrhosis reflects irreversible ischemic destruction of the cortex.

E. Hepatorenal syndrome-AKI from severe circulatory dysfunction after exclusion of competing causes.

60. During an outpatient nephrology consultation, for a medically suitable patient with kidney failure, which kidney replacement strategy generally offers the best long-term survival and quality-of-life potential?

A. No kidney replacement therapy, because transplantation does not improve survival or quality of life compared with maintenance dialysis in suitable candidates.

B. Long-term tunneled catheter hemodialysis, because permanent catheter access provides better survival and fewer infections than transplantation.

C. Continuous hospital-based CRRT, because intensive 24-hour dialysis is the preferred outpatient strategy for all stable patients with kidney failure.

D. Peritoneal dialysis is always superior to transplantation because home therapy avoids immunosuppression and has the best long-term graft-independent survival.

E. Kidney transplantation, including preemptive transplantation when feasible, is generally preferred over chronic dialysis for suitable candidates.

61. During inpatient nephrology rounds, a 81-year-old woman with CKD has rising phosphate and PTH values over several visits. How should CKD-mineral bone disorder generally be managed?

A. Interpret calcium, phosphate, and PTH together, following serial trends before changing CKD-MBD therapy.

B. Treat only PTH because phosphate and calcium do not influence bone turnover or vascular calcification once chronic kidney disease is present.

C. Use vitamin D receptor activators in every patient with any PTH elevation because secondary hyperparathyroidism should be completely suppressed to zero.

D. Normalize each value immediately after one abnormal result because CKD-MBD treatment is based on isolated measurements.

E. Ignore biochemical abnormalities until fractures occur because laboratory trends cannot provide useful information about CKD-mineral bone disorder.

62. At a kidney clinic follow-up visit, a 32-year-old taking a proton-pump inhibitor develops acute kidney injury, sterile pyuria, white-cell casts, and eosinophilia. Based on these findings, which diagnosis best accounts for this presentation?

A. Minimal change disease, which primarily causes nephrotic proteinuria.

B. Prerenal azotemia, which usually has bland sediment and improves with restoration of perfusion.

C. Acute postinfectious glomerulonephritis, which more often produces hematuria, red-cell casts, and glomerular proteinuria.

D. Drug-induced acute interstitial nephritis causing inflammatory injury of the interstitium and tubules.

E. Renal artery thrombosis, which causes abrupt ischemia but not the typical sterile pyuria and white-cell casts described.

63. At a transplant-nephrology follow-up visit, a deceased-donor kidney transplant produces little urine in the first several days and dialysis is temporarily required. Doppler flow is preserved and biopsy shows acute tubular injury. Given this presentation, what is the best interpretation?

A. BK nephropathy, because BK routinely causes dialysis-dependent graft failure during the first postoperative day.

B. Hyperacute rejection, because any need for dialysis during the first week proves irreversible antibody-mediated graft loss.

C. Delayed graft function from ischemia-reperfusion injury, with temporary dialysis need despite preserved graft perfusion.

D. Recurrent diabetic nephropathy, because chronic recipient diabetes causes immediate nodular sclerosis in the donor kidney.

E. Chronic allograft fibrosis, because years of alloimmune injury develop within hours of reperfusion.

64. At a hypertension clinic evaluation, a 66-year-old man with resistant hypertension and hyperkalemia has CKD and is taking an ACE inhibitor. Based on these findings, which step should precede automatically abandoning renin-angiotensin blockade?

A. Use an NSAID to reduce renal potassium excretion because prostaglandin inhibition reliably prevents arrhythmia in hyperkalemia.

B. Review diet and interacting drugs, optimize diuretic therapy and volume status, and consider potassium-lowering strategies when clinically appropriate.

C. Stop all renin-angiotensin blockade permanently after any potassium value above the laboratory reference range, regardless of severity or reversibility.

D. Begin dialysis solely to preserve the ACE inhibitor before assessing reversible contributors to chronic hyperkalemia.

E. Add a potassium supplement because ACE inhibitors cause intracellular potassium shifts that are corrected by increasing total-body potassium.

65. During a dialysis-unit assessment, a hemodialysis patient has severe generalized pruritus without cholestasis or a primary skin disorder. Based on these findings, which management approach is most appropriate?

A. Optimize dialysis and skin care, address CKD-MBD contributors, and consider evidence-based antipruritic therapies such as gabapentinoids or kappa-opioid approaches when appropriate.

B. Stop dialysis temporarily because extracorporeal toxin removal itself causes the pruritus and symptoms usually resolve when dialysis is withheld.

C. Treat with chronic high-dose corticosteroids because CKD-associated pruritus is primarily an autoimmune dermatitis in nearly all dialysis patients.

D. Use broad-spectrum antibiotics because generalized itch in kidney failure is presumed to represent occult bacterial skin infection even without lesions.

E. Increase serum phosphate intentionally because low phosphate is the dominant cause of uremic itch and phosphate loading suppresses cutaneous nerves.

66. During an outpatient nephrology consultation, a 40-year-old man with SIADH receives 0.9% saline. Urine osmolality is 700 mOsm/kg, and the serum sodium falls further. Given this presentation, what best explains this response?

A. Saline causes pseudohyponatremia by increasing the plasma lipid fraction, so the fall in sodium is a laboratory artifact.

B. The infused chloride acutely suppresses vasopressin, producing a water diuresis that lowers sodium because urine becomes maximally dilute.

C. SIADH prevents any urinary sodium excretion, so all infused sodium remains intracellular and draws water into cells, lowering extracellular sodium.

D. Normal saline is always hypotonic to plasma and therefore directly supplies electrolyte-free water even before any renal handling occurs.

E. The kidney excretes the infused sodium in a relatively concentrated urine while retaining part of the infused water, producing net free-water gain.

67. At a transplant-nephrology follow-up visit, a 66-year-old man with a kidney transplant on mycophenolate develops persistent diarrhea and leukopenia. Based on these findings, which medication effect is most likely?

A. Belatacept deficiency, because all transplant recipients require belatacept to prevent gastrointestinal toxicity from other agents.

B. Acyclovir-induced ENaC activation, which is the usual cause of leukopenia and diarrhea after transplantation.

C. Tacrolimus withdrawal syndrome, because stopping calcineurin inhibition causes immediate diarrhea and leukopenia even while tacrolimus is continued.

D. Prednisone mineralocorticoid toxicity, which typically causes diarrhea and severe leukopenia.

E. Mycophenolate toxicity causing gastrointestinal and bone-marrow adverse effects.

68. At a hypertension clinic evaluation, a 62-year-old man with hypertension uses licorice extract daily and has hypokalemia, metabolic alkalosis, low renin, and low aldosterone. Given this presentation, what mechanism is likely?

A. Deficient epithelial sodium-channel activity, which causes salt wasting, hypotension, and hyperkalemia.

B. Activating parathyroid hormone receptor mutation, which produces hypocalcemia but not apparent mineralocorticoid excess.

C. Excess aldosterone production from an adrenal adenoma, which should raise aldosterone.

D. Inhibition of 11β-hydroxysteroid dehydrogenase type 2 allows cortisol to activate the mineralocorticoid receptor.

E. Inhibition of proximal carbonic anhydrase, which causes bicarbonaturia and metabolic acidosis.

69. During an urgent nephrology assessment, a 73-year-old woman with neurologic symptoms, thrombocytopenia, microangiopathic hemolysis, and kidney injury has severe ADAMTS13 deficiency. Based on these findings, which diagnosis is most likely?

A. Immune thrombocytopenia, which causes isolated platelet destruction without schistocytes or microangiopathic hemolysis.

B. Complement-mediated atypical HUS, which is primarily defined by severe ADAMTS13 deficiency.

C. Disseminated membranous nephropathy, which causes nephrotic proteinuria but not profound ADAMTS13 deficiency.

D. Thrombotic thrombocytopenic purpura caused by severe ADAMTS13 deficiency and microvascular platelet-rich thrombosis.

E. Hemolytic anemia from G6PD deficiency, which causes oxidative red-cell injury without severe thrombocytopenia or microvascular thrombi.

70. At a multidisciplinary renal conference, a 36-year-old man with an anion-gap metabolic acidosis has an increase in anion gap of 18 mEq/L but bicarbonate has fallen only 8 mEq/L from normal. Given this presentation, what additional process is suggested?

A. No mixed disorder, because the magnitude of bicarbonate fall has no relationship to the change in anion gap in metabolic acidosis.

B. A concurrent metabolic alkalosis because the bicarbonate fall is smaller than expected for the increase in unmeasured anions.

C. An isolated respiratory acidosis because bicarbonate retention is the expected compensation for any increase in the serum anion gap.

D. An isolated respiratory alkalosis because delta-gap analysis compares bicarbonate directly with PaCO2.

E. A concurrent normal-anion-gap metabolic acidosis because bicarbonate should fall less than the rise in anion gap when two acidoses are present together.

71. During a hospital nephrology consultation, a healthy young adult has lifelong microscopic hematuria, normal kidney function, little proteinuria, and a family history of similar benign hematuria. Given this presentation, what disorder is most likely?

A. Thin basement membrane nephropathy related to collagen IV variants, with generally favorable prognosis in this presentation.

B. Renal-vein thrombosis, which causes an acquired vascular event.

C. Rapidly progressive ANCA glomerulonephritis, which would cause active inflammation and declining kidney function.

D. Postinfectious glomerulonephritis, which is an acute process and does not explain a stable familial lifelong pattern.

E. Membranous nephropathy, which is a nephrotic proteinuric disease.

72. During an outpatient nephrology consultation, a postoperative patient has sodium 122 mEq/L. Serum osmolality is 270 mOsm/kg, urine osmolality 600 mOsm/kg, urine sodium 8 mEq/L, orthostatic hypotension, and dry mucous membranes. Given this presentation, what is the most likely physiology?

A. Pseudohyponatremia from paraproteinemia.

B. Hypertonic hyponatremia from mannitol, which raises effective serum osmolality.

C. Primary polydipsia, which suppresses vasopressin and should generate urine far more dilute than the measured value here.

D. SIADH with euvolemia, which usually has urine sodium above the low-volume range when dietary sodium intake is adequate.

E. Hypovolemic hyponatremia with nonosmotic vasopressin release and avid renal sodium conservation.

73. During inpatient nephrology rounds, a 69-year-old woman with AKI becomes progressively confused, develops myoclonus, and has no other metabolic or neurologic explanation. Given this presentation, what kidney-related complication should be considered?

A. Acute nephrogenic diabetes insipidus, because dilute urine directly causes myoclonus despite normal sodium and osmolality.

B. Uremic encephalopathy, which supports initiation of kidney replacement therapy.

C. Renal-vein thrombosis, because unilateral venous obstruction routinely causes diffuse encephalopathy without other findings.

D. Minimal change disease, because urinary albumin loss itself is neurotoxic and causes uremic symptoms at normal GFR.

E. Asymptomatic azotemia, because neurologic manifestations are unrelated to retained solutes and do not affect dialysis decisions.

74. At a transplant-nephrology follow-up visit, a transplant recipient develops unexplained rising creatinine. Ultrasound excludes obstruction and drug levels are not clearly toxic. Given this presentation, what test most directly distinguishes rejection from several competing parenchymal causes?

A. A single serum creatinine measurement, because the magnitude of creatinine rise uniquely identifies rejection without tissue.

B. Kidney allograft biopsy to define the parenchymal lesion and distinguish rejection from competing causes.

C. Chest radiography, because pulmonary findings determine whether the transplant kidney is being rejected.

D. Serum albumin alone, because hypoalbuminemia is specific for T-cell-mediated rejection.

E. Urine sodium alone, because fractional sodium excretion reliably separates acute rejection from BK infection and drug toxicity.

75. During a dialysis-unit assessment, a 42-year-old woman receiving maintenance dialysis has persistently elevated serum phosphate despite dietary counseling. Based on these findings, which treatment principle is appropriate?

A. Start calcitriol alone because vitamin D receptor activation directly removes phosphate from plasma even when kidney excretion is negligible.

B. Use a thiazide diuretic as the primary therapy in an anuric patient because thiazides markedly increase urinary phosphate excretion without residual filtration.

C. Ignore phosphate if calcium is normal because hyperphosphatemia has no relationship to secondary hyperparathyroidism or vascular calcification risk.

D. Give oral phosphate supplements with meals because intestinal phosphate loading suppresses PTH and therefore lowers serum phosphate in kidney failure.

E. Use dietary phosphate reduction, adequate dialysis, and a phosphate binder selected according to calcium balance, pill burden, and comorbidities.

76. At a multidisciplinary renal conference, a 54-year-old man taking trimethoprim develops hyperkalemia despite stable creatinine. Based on these findings, which tubular action contributes?

A. Trimethoprim inhibits epithelial sodium channels in the collecting duct, reducing the electrical drive for potassium secretion.

B. Trimethoprim activates ENaC and therefore causes hypokalemia by increasing distal potassium secretion.

C. The drug blocks NKCC2 in the loop of Henle and acts as a potent loop diuretic at usual antimicrobial doses.

D. The medication causes hyperkalemia only by massive cell lysis and has no direct distal tubular effect.

E. Trimethoprim stimulates aldosterone synthesis and always lowers potassium even when kidney function is impaired.

77. During a hospital nephrology consultation, a 65-year-old woman with fever, rash, eosinophilia, pyuria, and AKI after starting an antibiotic is suspected of acute interstitial nephritis. Given this presentation, what is the first management step?

A. Continue the drug because stopping an antibiotic worsens interstitial inflammation through immune rebound.

B. Begin high-dose loop diuretics because diuresis removes interstitial eosinophils and reverses the hypersensitivity reaction.

C. Start chronic dialysis without withdrawing the agent because drug-induced AIN is uniformly irreversible once creatinine rises.

D. Stop the suspected offending drug and assess the severity and need for further diagnostic or anti-inflammatory treatment.

E. Give a potassium supplement as definitive therapy because AIN is primarily a disorder of total-body potassium depletion.

78. During an outpatient nephrology consultation, a 56-year-old with bilateral renal artery stenosis begins an ACE inhibitor and creatinine rises markedly within several days. Based on these findings, which mechanism best accounts for the fall in GFR?

A. ACE inhibition acutely deposits immune complexes in glomeruli, producing proliferative glomerulonephritis and obstruction.

B. Loss of angiotensin II–mediated efferent arteriolar constriction reduces glomerular pressure when renal perfusion is already compromised.

C. ACE inhibition constricts the afferent arteriole directly, eliminating renal blood flow despite normal upstream perfusion.

D. ACE inhibition causes renal-vein thrombosis by increasing intraglomerular pressure and activating platelets.

E. ACE inhibition blocks proximal glucose reabsorption, causing osmotic diuresis severe enough to collapse GFR in every patient.

79. At a transplant-nephrology follow-up visit, a recipient develops graft dysfunction with microvascular inflammation, circulating donor-specific antibodies, and complement deposition in peritubular capillaries. Given this presentation, what process is most likely?

A. Isolated calcineurin inhibitor vasoconstriction.

B. Acute pyelonephritis, which can inflame the interstitium but does not explain donor-specific antibody with complement-associated microvascular injury.

C. Recurrent minimal change disease, which causes podocyte injury and proteinuria.

D. Antibody-mediated allograft rejection driven by donor-specific alloantibodies and microvascular injury.

E. Postrenal obstruction, which causes hydronephrosis and pressure injury.

80. At a kidney clinic follow-up visit, a 50-year-old man with high-grade lymphoma develops hyperuricemia, hyperphosphatemia, hyperkalemia, hypocalcemia, and AKI after chemotherapy. Given this presentation, what syndrome is present?

A. Refeeding syndrome, which is characterized by hypophosphatemia.

B. Tumor lysis syndrome causing abrupt release of intracellular solutes and crystal-related kidney injury.

C. Nephrotic syndrome, which causes hypoalbuminemia and edema but not acute urate and phosphate release from tumor cells.

D. SIADH, which causes water retention and hyponatremia without the characteristic cell-lysis electrolyte pattern.

E. Distal RTA, which causes normal-anion-gap acidosis and stones but not rapid hyperuricemia and hyperphosphatemia after chemotherapy.

81. At a metabolic stone clinic visit, a young adult has recurrent stones and hexagonal crystals on urine microscopy. Based on these findings, which disorder is most likely?

A. Struvite stone disease, which requires urease-producing infection and often forms coffin-lid crystals in alkaline urine.

B. Hyperuricosuria from gout, which produces uric acid crystals with different morphology and strong dependence on acidic urine.

C. Primary hyperoxaluria, which produces calcium oxalate crystallization.

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

E. Distal renal tubular acidosis, which favors calcium phosphate stones in alkaline urine.

82. At a hypertension clinic evaluation, a young patient has early severe hypertension, hypokalemic metabolic alkalosis, suppressed renin, and suppressed aldosterone. Based on these findings, which inherited disorder is most likely?

A. Gitelman syndrome, which causes renal salt wasting with low or normal blood pressure.

B. Primary aldosteronism, which characteristically produces an inappropriately elevated aldosterone concentration.

C. Bartter syndrome, which usually activates renin and aldosterone because of salt wasting in the loop of Henle.

D. Liddle syndrome from gain-of-function epithelial sodium channels in collecting-duct principal cells.

E. Type 4 renal tubular acidosis, which causes hyperkalemia and metabolic acidosis.

83. During a dialysis-unit assessment, why might a non-calcium phosphate binder be preferred in a dialysis patient with hyperphosphatemia and extensive vascular calcification?

A. Non-calcium binders eliminate the need for dietary phosphate management because they bind every ingested phosphate molecule regardless of dose.

B. Non-calcium binders increase intestinal phosphate absorption intentionally, allowing dialysis to remove a larger phosphate load during the next session.

C. Avoiding additional calcium load may be desirable when calcium balance and vascular calcification are already major concerns.

D. Calcium-containing binders cannot bind dietary phosphate at all, so they are pharmacologically ineffective despite their common historical use.

E. Vascular calcification in CKD is caused only by low calcium intake, so non-calcium binders prevent it by increasing serum calcium indirectly.

84. During an outpatient nephrology consultation, a 46-year-old man with severe diarrhea has normal-gap acidosis. The urine anion gap is strongly negative. Given this presentation, what does this finding imply?

A. Urinary ammonium excretion is low, confirming distal renal tubular acidosis as the cause of the systemic acidosis.

B. The patient has a high-anion-gap acidosis because a negative urine anion gap indicates unmeasured organic anions in plasma.

C. Urine contains no chloride, proving that the kidneys are retaining acid.

D. Urinary ammonium excretion is appropriately increased, supporting an extrarenal bicarbonate loss.

E. The result establishes proximal renal tubular acidosis because bicarbonaturia is the only mechanism that can make the urine anion gap negative.

85. During inpatient nephrology rounds, a 57-year-old woman with severe AKI develops a pericardial friction rub and chest discomfort without another explanation. Given this presentation, what complication is most concerning?

A. Acute interstitial nephritis, because pericardial rub is a specific manifestation of renal interstitial eosinophilia.

B. Uremic pericarditis, which is an urgent indication for intensified kidney replacement therapy.

C. Minimal change disease, because hypoalbuminemia commonly produces a friction rub without pericardial inflammation.

D. Nephrogenic diabetes insipidus, because hypernatremia causes sterile pericardial inflammation before polyuria develops.

E. Renal papillary necrosis, because sloughed papillae directly irritate the pericardium through circulating debris.

86. At a kidney clinic follow-up visit, a 68-year-old man with profound hypokalemia remains refractory to potassium replacement. Magnesium is 0.9 mg/dL. Why is magnesium replacement important?

A. Magnesium replacement directly converts potassium into an intracellular salt, correcting hypokalemia without any need for potassium administration.

B. Magnesium deficiency prevents gastrointestinal potassium absorption, so intravenous potassium is ineffective even when renal function is normal.

C. Low magnesium blocks distal sodium delivery, so potassium wasting occurs only through stool and is unrelated to renal potassium handling.

D. Magnesium deficiency promotes renal potassium wasting, so potassium may not correct until magnesium is repleted.

E. Low magnesium suppresses aldosterone completely, causing potassium retention that falsely lowers the serum potassium concentration by redistribution.

87. During an urgent nephrology assessment, a 79-year-old woman with persistent vomiting has pH 7.52, bicarbonate 38 mEq/L, and urine chloride 6 mEq/L. Based on these findings, which treatment is most appropriate?

A. Give acetazolamide as first-line therapy despite marked volume depletion because chloride-responsive alkalosis should not be treated with saline.

B. Restrict chloride intake because urinary chloride is low, indicating that additional chloride will worsen the alkalemia.

C. Administer sodium bicarbonate because raising filtered bicarbonate promotes renal alkali excretion and is preferred in chloride depletion.

D. Begin hydrochlorothiazide to increase distal sodium delivery because thiazide-induced contraction corrects vomiting-related alkalosis.

E. Restore extracellular volume and chloride with isotonic saline and replace potassium as needed.

88. At a multidisciplinary renal conference, a 42-year-old man with cerebral edema and severe AKI requires renal replacement therapy. Why might slower continuous therapy be preferred?

A. More gradual osmotic and fluid shifts can reduce abrupt changes in intracranial pressure compared with rapid intermittent solute removal.

B. Continuous therapy raises blood urea rapidly, which pulls water out of the brain and always resolves cerebral edema.

C. CRRT eliminates the need to monitor sodium because continuous therapy cannot change plasma tonicity.

D. Cerebral edema is unaffected by dialysis-related osmotic shifts, so modality choice has no neurologic implications.

E. Intermittent dialysis cannot remove urea, so continuous therapy is chosen solely because it provides greater solute clearance per minute.

89. During a hospital nephrology consultation, a 75-year-old woman taking hydrochlorothiazide presents with nausea and confusion. Serum sodium is 116 mEq/L, serum osmolality is low, urine osmolality is 520 mOsm/kg, and urine sodium is 62 mEq/L. She has no edema. Given this presentation, what is the most likely cause of the hyponatremia?

A. Primary polydipsia, which usually produces maximally dilute urine when water intake is the dominant problem.

B. Hyperglycemia-mediated translocational hyponatremia, which would be accompanied by elevated effective serum osmolality.

C. Thiazide-associated hyponatremia with impaired urinary dilution and continued renal sodium loss.

D. Pseudohyponatremia from severe hyperlipidemia, which leaves measured serum osmolality near normal with indirect assays.

E. Advanced heart failure, which generally causes low effective arterial volume with edema and avid renal sodium retention.

90. During an outpatient nephrology consultation, a 64-year-old man with multiple myeloma develops glucosuria with normal serum glucose, phosphaturia, aminoaciduria, hypouricemia, and bicarbonate wasting. Given this presentation, what renal tubular disorder is present?

A. Type 4 renal tubular acidosis, which is driven by hypoaldosteronism or aldosterone resistance and usually causes hyperkalemia.

B. Fanconi syndrome with generalized proximal tubular dysfunction causing proximal renal tubular acidosis.

C. Gitelman syndrome, which affects the distal sodium-chloride cotransporter and characteristically causes hypomagnesemia and hypocalciuria.

D. Distal renal tubular acidosis, which primarily impairs hydrogen secretion and does not cause generalized proximal solute wasting.

E. Bartter syndrome, which affects thick ascending limb salt transport and does not produce glucosuria or aminoaciduria with normal serum glucose.

91. During inpatient nephrology rounds, an older man with prostate enlargement develops anuria and bilateral hydronephrosis. Based on these findings, which category of AKI is present?

A. Acute interstitial nephritis, because prostate enlargement triggers eosinophilic inflammation of both kidneys.

B. Intrinsic glomerular AKI, because hydronephrosis indicates inflammatory capillary-wall injury.

C. Prerenal AKI, because urinary obstruction lowers intake and therefore affects GFR only through volume depletion.

D. Renal vascular AKI, because bladder outlet obstruction directly thromboses both renal arteries.

E. Postrenal acute kidney injury from bilateral urinary tract obstruction and increased collecting-system pressure.

92. At a kidney clinic follow-up visit, a 38-year-old man with acute decompensated heart failure has pulmonary edema, venous congestion, and worsening creatinine. Based on these findings, which mechanism can contribute to cardiorenal AKI?

A. Elevated venous pressure and renal congestion can reduce effective filtration in addition to reduced forward perfusion.

B. Heart failure affects the kidney only through low cardiac output, so right-sided venous pressure has no renal consequence.

C. Venous congestion increases the renal perfusion gradient and therefore always raises GFR despite heart failure.

D. Diuretic exposure proves tubular necrosis because any creatinine increase during decongestion represents structural kidney damage.

E. Pulmonary edema causes direct immune-complex deposition in glomeruli and is the main cause of AKI in heart failure.

93. During an urgent nephrology assessment, a 49-year-old woman on chronic lithium develops polyuria with low urine osmolality despite hypernatremia. Based on these findings, which nephron effect is most relevant?

A. Lithium blocks aquaporin release from the posterior pituitary, producing central.

B. Lithium activates V2 receptors continuously, causing SIADH and concentrated urine during hypernatremia.

C. Lithium inhibits the sodium-chloride cotransporter in the distal convoluted tubule, making its effect identical to a thiazide.

D. Lithium selectively blocks glomerular filtration of water while preserving solute filtration, which directly causes dilute urine.

E. Lithium enters collecting-duct principal cells through ENaC and impairs vasopressin signaling, producing nephrogenic diabetes insipidus.

94. At a transplant-nephrology follow-up visit, a stable kidney recipient asks whether a live attenuated vaccine can be given routinely while receiving maintenance immunosuppression. Given this presentation, what is the general principle?

A. Live vaccines are generally avoided after transplantation because immunosuppression can permit uncontrolled replication of the vaccine organism.

B. All vaccines are contraindicated after transplantation, including inactivated products, because any immune response triggers rejection.

C. Vaccine decisions depend only on graft function, not on the intensity or type of immunosuppression.

D. Live vaccines are preferred after transplantation because they produce stronger immunity and cannot replicate in immunosuppressed hosts.

E. Only bacterial live vaccines are avoided; live viral vaccines are routinely safe under full immunosuppression.

95. During a hospital nephrology consultation, a 71-year-old woman has recurrent complement-mediated thrombotic microangiopathy without severe ADAMTS13 deficiency or Shiga toxin. Based on these findings, which pathway is implicated?

A. Excess vasopressin secretion, which directly produces platelet-rich glomerular thrombi and hemolysis.

B. Autoantibodies to PLA2R, which cause thrombotic microangiopathy.

C. Loss of proximal bicarbonate transport, which activates complement and causes systemic microangiopathic hemolysis.

D. Deficient renin release from juxtaglomerular cells, which is the defining molecular lesion in complement-mediated HUS.

E. Dysregulated alternative complement activation causing complement-mediated HUS.

96. During an outpatient nephrology consultation, a 34-year-old man with chronic hepatitis C has purpura, arthralgias, low C4, hematuria, and proteinuria. Based on these findings, which renal process is most likely?

A. Minimal change disease.

B. Thin basement membrane disease, which causes isolated hematuria without systemic vasculitic manifestations.

C. ADPKD, which causes cystic kidneys and hypertension.

D. Cryoglobulinemic immune-complex glomerulonephritis, often with an MPGN pattern.

E. Anti-GBM disease, which causes linear IgG and pulmonary hemorrhage.

97. During inpatient nephrology rounds, a 45-year-old woman with tumor lysis syndrome develops symptomatic hypocalcemia and marked hyperphosphatemia. Given this presentation, what explains the low calcium?

A. Released intracellular phosphate binds calcium and promotes calcium-phosphate deposition, lowering the ionized calcium concentration.

B. Potassium released from tumor cells enters bone and displaces calcium into urine, causing isolated renal calcium wasting without phosphorus changes.

C. Lactate from tumor metabolism suppresses parathyroid hormone secretion permanently, making hypocalcemia independent of kidney function and phosphate.

D. Cell lysis releases calcitonin in large quantities, and this hormone is the dominant cause of acute hypocalcemia during tumor lysis syndrome.

E. Uric acid directly chelates calcium in plasma as the principal mechanism, independent of the large phosphate load released from tumor cells.

98. At a kidney clinic follow-up visit, a 56-year-old man has 5.8 g/day proteinuria, albumin 2.1 g/dL, edema, and hyperlipidemia. Based on these findings, which syndrome is present?

A. Renal tubular acidosis, because bicarbonate wasting produces edema and hyperlipidemia through loss of serum albumin.

B. Nephritic syndrome, because nephritic disease requires heavy proteinuria above 3.5 g/day as its defining feature.

C. Prerenal azotemia, because reduced renal perfusion directly causes nephrotic-range urinary protein losses.

D. Nephrotic syndrome with heavy proteinuria, hypoalbuminemia, edema, and lipid abnormalities.

E. Acute interstitial nephritis, because interstitial inflammation routinely causes massive albuminuria and severe hypoalbuminemia.

99. During a dialysis-unit assessment, a 70-year-old woman receiving maintenance dialysis has severe secondary hyperparathyroidism with hypercalcemia that limits vitamin D receptor activator use. Based on these findings, which medication class can lower PTH without adding calcium?

A. A calcium-containing phosphate binder at high dose because increasing calcium intake is the preferred way to treat hypercalcemic secondary hyperparathyroidism.

B. A loop diuretic in an anuric patient because increasing urinary calcium excretion is the main mechanism for suppressing PTH during dialysis.

C. An ACE inhibitor because angiotensin blockade directly activates parathyroid calcium-sensing receptors and lowers PTH independent of calcium.

D. Erythropoietin because correction of anemia directly suppresses autonomous parathyroid cell proliferation in advanced CKD.

E. A calcimimetic that increases calcium-sensing receptor activity and suppresses parathyroid hormone secretion.

100. During a dialysis-unit assessment, a 78-year-old man with severe metformin-associated lactic acidosis has shock, AKI, and very low pH despite resuscitation. Why can dialysis be considered?

A. Metformin is covalently bound to plasma proteins and cannot cross any dialysis membrane.

B. Dialysis is avoided because bicarbonate delivery during treatment invariably worsens lactic acidosis.

C. Metformin toxicity resolves only after hepatic transplantation, so renal replacement therapy has no mechanistic role.

D. Only peritoneal dialysis can remove metformin because the drug is too large for hemodialysis membranes.

E. Extracorporeal therapy can remove metformin and correct profound acidosis when toxicity is severe and renal clearance is impaired.

101. At a hypertension clinic evaluation, a 41-year-old woman with CKD has serum bicarbonate 18 mEq/L and is also volume overloaded with uncontrolled hypertension. Given this presentation, what is an important consideration before prescribing sodium bicarbonate?

A. Alkali should be given in unlimited doses until bicarbonate exceeds normal because overcorrection does not affect blood pressure or volume status.

B. The best treatment is hydrochloric acid infusion because correcting sodium overload requires acidifying the extracellular fluid directly.

C. Volume overload makes metabolic acidosis harmless, so low bicarbonate should be ignored until the patient becomes clinically dehydrated.

D. Alkali may be helpful, but the sodium load and volume status should be considered and therapy individualized with monitoring.

E. Sodium bicarbonate is absolutely contraindicated in every patient with edema because no form of alkali therapy can be used in chronic kidney disease.

102. At a transplant-nephrology follow-up visit, a 51-year-old woman with a kidney transplant misses immunosuppressive doses because of medication cost. Given this presentation, what is the most appropriate clinical response?

A. Double every immunosuppressive medication indefinitely without checking levels because missed doses require permanent dose escalation.

B. Ignore the access problem until creatinine rises because adherence has no relation to donor-specific antibody or rejection risk.

C. Address adherence nonjudgmentally, identify financial and access barriers, and restore a sustainable immunosuppressive plan promptly.

D. Stop immunosuppression completely because intermittent adherence is more harmful than no therapy after kidney transplantation.

E. Immediately label the patient ineligible for further transplant care because any missed dose proves intentional rejection of medical advice.

103. During inpatient nephrology rounds, a 63-year-old woman with panic-induced hyperventilation has $PaCO_2$ 25 mm Hg shortly after symptom onset. Based on these findings, which renal compensation is expected if the respiratory alkalosis persists for several days?

A. The kidneys cannot compensate for respiratory alkalosis, so bicarbonate remains fixed regardless of the duration of hypocapnia.

B. Renal ammonium excretion increases markedly, generating new bicarbonate and restoring $PaCO_2$ toward normal without any change in ventilation.

C. Renal bicarbonate reabsorption increases, producing a progressive metabolic alkalosis that amplifies the initial rise in pH.

D. Renal bicarbonate excretion increases, lowering serum bicarbonate more than in an acute episode.

E. Potassium secretion stops completely because respiratory alkalosis directly blocks distal sodium reabsorption and aldosterone signaling.

104. At a transplant-nephrology follow-up visit, minutes after reperfusion of a kidney allograft, the graft becomes mottled and produces no urine. Pretransplant testing reveals preformed donor-specific antibody. Given this presentation, what process is most likely?

A. Acute T-cell-mediated rejection, which generally develops over days to weeks and is not driven by preformed circulating antibody at reperfusion.

B. Chronic calcineurin inhibitor toxicity, which evolves over months to years.

C. Recurrent diabetic nephropathy, which requires prolonged exposure and cannot cause immediate graft failure.

D. Hyperacute antibody-mediated rejection from preexisting anti-donor antibodies causing immediate vascular injury.

E. BK polyomavirus nephropathy, which occurs under sustained immunosuppression and does not cause immediate graft thrombosis at reperfusion.

105. During an urgent nephrology assessment, a nondiabetic patient with proteinuric CKD and eGFR 38 mL/min/1.73 m2 is already receiving maximally tolerated renin-angiotensin system blockade. Based on these findings, which medication class has evidence for slowing CKD progression in many such patients?

A. A nonsteroidal anti-inflammatory drug, because lowering intraglomerular prostaglandins chronically preserves filtration and reduces albuminuria safely.

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

C. A dual ACE inhibitor plus ARB regimen, because combining both arms of renin-angiotensin blockade improves kidney outcomes without added harm.

D. A chronic high-dose loop diuretic solely to raise GFR, because increasing urine volume directly prevents progressive nephron loss.

E. An aminoglycoside antibiotic, because mild tubular stress activates protective pathways that slow proteinuric chronic kidney disease.

106. At a transplant-nephrology follow-up visit, a stable tacrolimus-treated recipient starts voriconazole and soon develops tremor, hyperkalemia, and rising creatinine. Given this presentation, what explains the change?

A. Voriconazole chelates tacrolimus in the intestine and prevents absorption, making toxicity pharmacologically impossible.

B. The combination invariably causes immune-complex nephritis independent of either drug concentration.

C. CYP3A inhibition can markedly increase tacrolimus exposure, requiring close level monitoring and dose adjustment.

D. Tacrolimus is cleared exclusively by glomerular filtration, so hepatic enzyme interactions cannot alter its blood concentration.

E. Voriconazole induces CYP3A and therefore causes tacrolimus underexposure and rejection.

107. During a dialysis-unit assessment, a 59-year-old woman has severe AKI but stable potassium, manageable volume status, improving acidosis, and no uremic symptoms. Creatinine is 7.0 mg/dL. Given this presentation, what is the best dialysis principle?

A. Do not start dialysis solely for a creatinine threshold; follow clinical complications and trajectory.

B. Avoid dialysis until creatinine exceeds 15 mg/dL because earlier kidney replacement therapy cannot treat electrolyte or volume complications.

C. Initiate dialysis automatically whenever creatinine exceeds 5 mg/dL because serum creatinine is an absolute indication independent of symptoms or trends.

D. Use serum urea alone because creatinine and clinical manifestations should not influence dialysis timing.

E. Start dialysis only after complete anuria persists for one month because urine output is the sole determinant of renal replacement need.

108. During an outpatient nephrology consultation, a 33-year-old woman develops polyuria after transsphenoidal surgery. Serum sodium is 151 mEq/L and urine osmolality is 90 mOsm/kg. After desmopressin, urine osmolality rises to 520 mOsm/kg. Given this presentation, what is the diagnosis?

A. SIADH, which causes water retention and concentrated urine.

B. Nephrogenic diabetes insipidus, in which the kidney remains poorly responsive and urine osmolality changes little after desmopressin.

C. Primary polydipsia, which generally lowers.

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

E. Osmotic diuresis from glucosuria, which usually produces urine containing substantial solute.

109. During inpatient nephrology rounds, a 81-year-old woman has nephrotic syndrome, cardiomyopathy, and neuropathy. Kidney biopsy shows Congo-red-positive deposits with apple-green birefringence. Given this presentation, what process is present?

A. Diabetic nephropathy, because Kimmelstiel-Wilson nodules routinely display apple-green birefringence under polarized light.

B. Fibrillary glomerulonephritis is excluded because all organized glomerular deposits are Congo-red positive by definition.

C. Renal amyloidosis with extracellular beta-pleated-sheet fibril deposition.

D. Anti-GBM disease, because linear antibody binding creates amyloid fibrils within the glomerular basement membrane.

E. Minimal change disease, because podocyte effacement produces Congo-red-positive mesangial material in nephrotic patients.

110. At a transplant-nephrology follow-up visit, a 53-year-old woman with a kidney transplant is receiving standard opportunistic-infection prophylaxis. Based on these findings, which drug is commonly used to prevent Pneumocystis jirovecii pneumonia when tolerated?

A. Trimethoprim-sulfamethoxazole prophylaxis, which is commonly first-line for Pneumocystis prevention when tolerated.

B. Fluconazole alone, because yeast-directed prophylaxis reliably prevents Pneumocystis pneumonia.

C. Metronidazole, because anaerobic coverage is the standard preventive strategy for post-transplant Pneumocystis infection.

D. Oseltamivir, because influenza neuraminidase inhibition prevents Pneumocystis replication in alveoli.

E. Amoxicillin alone, because Pneumocystis is a typical extracellular bacterial pathogen susceptible to beta-lactams.

111. During an urgent nephrology assessment, why is routine combination therapy with an ACE inhibitor and an ARB generally avoided in CKD?

A. Dual therapy consistently causes severe hypercalcemia because angiotensin blockade directly stimulates parathyroid hormone secretion in CKD.

B. Combining the drugs eliminates albuminuria completely, but this is avoided only because current assays cannot measure urine protein afterward.

C. ACE inhibitors neutralize ARBs in the intestine, making both medications pharmacologically inactive while leaving blood pressure uncontrolled.

D. The two drugs have identical molecular structures, so combining them causes allergic cross-linking.

E. Dual blockade can increase hyperkalemia, hypotension, and acute kidney injury without providing enough outcome benefit to justify the added risk.

112. During a dialysis-unit assessment, a 66-year-old man with a new upper-arm AV fistula develops hand pain, pallor, and coolness that worsens during dialysis, with reduced distal pulses. Given this presentation, what is the most likely complication?

A. Dialysis access steal syndrome from diversion of arterial flow through the fistula causing distal ischemia.

B. High-output heart failure, which can occur with high-flow access but usually causes dyspnea and systemic volume symptoms.

C. Central venous stenosis, which more typically causes ipsilateral arm or breast edema and venous collaterals.

D. Catheter-related bacteremia, which causes fever and rigors.

E. Dialysis disequilibrium syndrome, which causes cerebral edema symptoms during rapid solute removal and is unrelated to access-side hand perfusion.

113. At a transplant-nephrology follow-up visit, a recipient develops a new donor-specific HLA antibody despite stable creatinine. Why does this finding matter?

A. It can signal alloimmune risk and warrants clinical assessment because donor-specific antibodies may precede antibody-mediated graft injury.

B. It is always clinically irrelevant unless serum creatinine has already doubled, because antibodies cannot injure graft endothelium before advanced dysfunction.

C. It establishes BK nephropathy because HLA antibodies are generated by polyomavirus replication in tubular cells.

D. It confirms ureteral obstruction because donor-specific antibodies form only when urine flow from the graft is impaired.

E. It proves hyperacute rejection is occurring, which can only develop years after transplantation when antibodies first appear.

114. During an outpatient nephrology consultation, a 40-year-old man with AKI has potassium 7.2 mEq/L and new QRS widening. Given this presentation, what is the immediate first priority?

A. Administer a thiazide as the sole emergency therapy because distal sodium delivery immediately reverses cardiotoxicity.

B. Give a potassium supplement because QRS widening indicates intracellular potassium depletion despite elevated serum potassium.

C. Delay treatment until a repeat potassium returns because electrocardiographic changes make pseudohyperkalemia more likely.

D. Give sodium polystyrene alone because gastrointestinal potassium removal acts quickly enough to reverse QRS widening within minutes.

E. Give intravenous calcium to stabilize the cardiac membrane while simultaneously arranging potassium-shifting and removal therapies.

115. During a dialysis-unit assessment, a hemodialysis patient with a tunneled catheter develops fever, rigors during dialysis, and positive blood cultures. Given this presentation, what complication should be presumed?

A. AV fistula steal syndrome, because reduced hand perfusion commonly presents with rigors and sustained positive blood cultures.

B. Dialysis disequilibrium syndrome, because fever and positive blood cultures are expected osmotic manifestations of rapid urea removal.

C. Catheter-related bloodstream infection requiring prompt antimicrobial therapy and source-control assessment.

D. Uremic pericarditis, because catheter manipulation causes sterile pericardial inflammation that commonly produces bacteremia during dialysis.

E. Calciphylaxis, because vascular calcification releases bacteria into the bloodstream even when there are no painful skin lesions.

116. At a kidney clinic follow-up visit, a 62-year-old man with severe AKI after iodinated contrast has no obstruction and urine sediment is consistent with tubular injury. Given this presentation, what is the most appropriate general management?

A. Start high-dose glucocorticoids because contrast-associated tubular injury is an immune-complex glomerulonephritis.

B. Provide supportive care, avoid additional renal insults, optimize hemodynamics, and monitor for indications for kidney replacement therapy.

C. Induce dehydration to reduce tubular flow because concentrating contrast prevents further epithelial exposure.

D. Begin dialysis solely to remove all residual contrast even when electrolytes, volume, and symptoms are controlled.

E. Give repeated contrast doses because recurrent exposure accelerates tubular adaptation and shortens the duration of AKI.

117. At a metabolic stone clinic visit, a 73-year-old woman with gout has recurrent radiolucent kidney stones and urine pH 5.1. Based on these findings, which intervention most directly reduces recurrence?

A. Acidify the urine further because uric acid becomes more ionized and soluble as urinary pH falls below 5.5.

B. Restrict all dietary calcium because uric acid stones form primarily from calcium-urate complexes.

C. Start a loop diuretic to increase urinary calcium because calcium complexes with uric acid and prevents urate crystallization.

D. Alkalinize the urine, usually with potassium citrate, to increase uric acid solubility and reduce crystal formation.

E. Use ammonium chloride to lower urine pH because urinary acidification is the main treatment for radiolucent uric acid stones.

118. At a multidisciplinary renal conference, a 36-year-old man with advanced CKD develops pleuritic chest pain relieved by sitting forward and a pericardial friction rub. ECG lacks the typical diffuse ST elevations of viral pericarditis. Given this presentation, what is the best treatment principle?

A. Recognize uremic pericarditis as an indication for urgent initiation or intensification of dialysis while assessing for tamponade.

B. Treat only with high-dose NSAIDs because dialysis does not influence uremic pericardial inflammation and kidney function is no longer relevant.

C. Begin thrombolysis because chest pain with a friction rub in CKD is most often an atypical presentation of acute coronary thrombosis.

D. Observe without changing kidney replacement therapy because uremic pericarditis usually resolves spontaneously despite continued toxin accumulation.

E. Start chronic anticoagulation because uremic pericardial effusions are caused primarily by fibrin thrombi in the pericardial microcirculation.

119. During a hospital nephrology consultation, a 47-year-old woman with AKI is taking a loop diuretic. Why can fractional excretion of sodium be misleading?

A. FENa is unaffected by any medication because sodium excretion depends only on GFR and not tubular transport.

B. Diuretics prevent creatinine filtration, making the mathematical formula for FENa impossible to calculate.

C. Loop diuretics increase urinary sodium excretion, so FENa may be elevated even when reduced effective perfusion contributed to AKI.

D. Loop diuretics make urine sodium zero, causing FENa to be falsely low in all forms of intrinsic kidney injury.

E. FENa directly measures renal blood flow, so it remains perfectly accurate regardless of tubular drug effects.

120. During an outpatient nephrology consultation, a 58-year-old man with CKD has rapidly worsening edema and rising albuminuria despite stable eGFR. Why should this change trigger reassessment?

A. A major increase in albuminuria can signal active kidney disease and higher progression risk even before a substantial fall in eGFR occurs.

B. Stable eGFR excludes progression because structural kidney injury cannot worsen until creatinine has already risen by at least twofold.

C. Only hematuria predicts active glomerular disease; albuminuria magnitude has no relationship to renal prognosis or treatment decisions.

D. Albuminuria cannot change independently of GFR, so the result is necessarily laboratory error if serum creatinine remains stable.

E. Edema proves the patient has heart failure.

121. During an intensive-care nephrology consultation, a 69-year-old woman with septic shock has persistent AKI despite restoration of mean arterial pressure. Why can kidney dysfunction continue?

A. The kidney receives no sympathetic or microvascular regulation during sepsis, making organ injury independent of systemic inflammation.

B. Persistent AKI proves bilateral ureteral obstruction because inflammatory pathways cannot alter renal microcirculation.

C. Sepsis causes only glomerular immune-complex deposition, so tubular and endothelial mechanisms are not relevant.

D. Sepsis-associated AKI involves inflammatory, endothelial, microcirculatory, and metabolic tubular stress in addition to macroscopic perfusion pressure.

E. Once blood pressure is restored, any remaining creatinine elevation must be laboratory error because sepsis affects kidneys only through hypotension.

122. At a kidney clinic follow-up visit, a 80-year-old man has hypernatremia, polyuria, urine osmolality 70 mOsm/kg, and no access to water because of delirium. Based on these findings, which combination most directly explains development of severe hypernatremia?

A. Failure of urinary concentration combined with impaired thirst or inability to obtain enough free water.

B. High vasopressin activity with unrestricted water access, because concentrated urine and preserved thirst are the usual prerequisites for hypernatremia.

C. Isolated loss of aldosterone action, because mineralocorticoid deficiency typically causes sodium retention and water loss without hyperkalemia.

D. SIADH with preserved renal function, because persistent water retention raises sodium when oral intake is limited for several hours.

E. Primary polydipsia with intact access to fluids, because excessive water intake is a common cause of severe hypernatremia in hospitalized adults.

123. During a dialysis-unit assessment, a 63-year-old woman receiving maintenance dialysis with obesity, diabetes, warfarin exposure, and severe secondary hyperparathyroidism develops exquisitely painful retiform plaques that become necrotic. Based on these findings, which diagnosis best accounts for this presentation?

A. Cholesterol embolization, which can cause livedo and blue toes but is usually triggered by vascular manipulation and has different systemic clues.

B. Warfarin skin necrosis alone, which usually occurs soon after initiation and does not capture the broader CKD-mineral bone risk phenotype.

C. Cellulitis, which causes infection and erythema but does not typically produce multifocal retiform necrosis in the classic dialysis risk profile.

D. Uremic pruritus, which causes diffuse itching but not intensely painful retiform ischemic plaques with progressive tissue necrosis.

E. Calciphylaxis with calcification and thrombosis of small dermal and subcutaneous vessels.

124. At a hypertension clinic evaluation, a 54-year-old man with chronic hypertension has an arm pressure of 150/88 mm Hg and ankle pressure of 210/96 mm Hg with diminished femoral pulses. Based on these findings, which diagnosis should be considered?

A. Pheochromocytoma, which characteristically lowers ankle systolic pressure while preserving arm pressure between spells.

B. Liddle syndrome, which produces aortic narrowing through chronic epithelial sodium-channel activation.

C. Primary aldosteronism, which produces an arm-leg pressure gradient by selectively constricting lower-extremity arteries.

D. Coarctation of the aorta causing upper-extremity hypertension with lower-extremity pulse and pressure abnormalities.

E. White-coat hypertension, which causes persistent femoral pulse delay and fixed lower-extremity arterial obstruction.

125. At a hypertension clinic evaluation, a 65-year-old woman has severe asymptomatic hypertension discovered during a routine visit but no neurologic, cardiac, retinal, or renal evidence of acute injury. Given this presentation, what is the general management principle?

A. Begin thrombolysis because severe hypertension without symptoms is usually caused by an occult ischemic stroke.

B. Lower mean arterial pressure by more than half within one hour because chronic autoregulation protects organs from rapid reductions.

C. Use an intravenous vasodilator immediately because any severe pressure reading represents a hypertensive emergency even without organ injury.

D. Confirm the pressure and intensify oral therapy with close follow-up, avoiding rapid intravenous lowering without acute organ injury.

E. Withhold all treatment because asymptomatic severe hypertension cannot contribute to long-term cardiovascular or kidney risk.

126. During an outpatient nephrology consultation, a 72-year-old cachectic patient has creatinine 0.8 mg/dL but multiple signs of chronic kidney disease. Given this presentation, what is the key limitation of creatinine-based eGFR in this setting?

A. Low muscle mass can lower creatinine generation and make kidney function appear better than it actually is.

B. Creatinine is fully reabsorbed by the proximal tubule in malnutrition, making serum values unrelated to filtration in all cachectic patients.

C. Low muscle mass causes pseudohypercreatininemia from assay interference, so a low measured creatinine indicates severe laboratory error.

D. Serum creatinine becomes independent of GFR after age 75, so no filtration estimate can be made in an older patient.

E. Cachexia increases creatinine generation so markedly that creatinine-based equations always underestimate GFR in frail older adults.

127. During inpatient nephrology rounds, a 39-year-old woman with subarachnoid hemorrhage develops sodium 125 mEq/L, high urine sodium, concentrated urine, hypotension, and a negative fluid balance. Based on these findings, which diagnosis is favored over SIADH?

A. Primary polydipsia, because neurologic injury causes compulsive water intake with urine osmolality remaining markedly elevated.

B. Cerebral salt wasting with renal sodium loss and true extracellular volume depletion.

C. SIADH, because SIADH typically produces hypotension and a persistently negative sodium balance from major extracellular volume depletion.

D. Pseudohyponatremia, because intracranial hemorrhage commonly increases plasma protein enough to create an indirect assay artifact.

E. Hypertonic hyponatremia, because subarachnoid hemorrhage usually raises serum osmolality sufficiently to shift water out of cells.

128. At a kidney clinic follow-up visit, a marathon runner drinks large amounts of hypotonic fluid and becomes confused near the finish. Sodium is 119 mEq/L. Based on these findings, which is the most important initial principle?

A. Treat symptomatic exercise-associated hyponatremia with hypertonic saline while avoiding additional hypotonic fluid intake.

B. Start a thiazide diuretic to enhance sodium retention because distal sodium blockade reduces urinary sodium losses during endurance exercise.

C. Give large volumes of plain water because exertional heat production makes free-water replacement the priority despite the measured sodium.

D. Delay treatment until urine osmolality returns to normal because acute symptomatic hyponatremia rarely causes clinically important cerebral edema.

E. Use isotonic dextrose solution as the sole therapy because glucose rapidly enters cells and leaves sodium-rich extracellular fluid behind.

129. During a dialysis-unit assessment, a 61-year-old woman with severe salicylate toxicity has tinnitus, tachypnea, confusion, pulmonary edema, and worsening acidemia. Given this presentation, what is the role of hemodialysis?

A. Dialysis worsens toxicity by acidifying blood and driving salicylate into the brain in every patient.

B. Hemodialysis can rapidly remove salicylate and correct acid-base and volume complications in severe toxicity.

C. Extracorporeal therapy is useful only when the patient has isolated metabolic alkalosis without neurologic or pulmonary manifestations.

D. Dialysis removes potassium but not salicylate, so it has no role in severe poisoning.

E. Hemodialysis is contraindicated because salicylate is completely irreversibly protein bound even at toxic concentrations.

130. At a multidisciplinary renal conference, a 72-year-old man with diarrhea has bicarbonate 15 mEq/L, normal anion gap, and urine ammonium excretion is appropriately high. Given this presentation, what is the primary acid-base process?

A. Distal renal tubular acidosis, which would be expected to impair net acid excretion and reduce ammonium generation despite systemic acidosis.

B. Type 4 renal tubular acidosis, which usually occurs with hyperkalemia and reduced ammonium excretion.

C. High-anion-gap lactic acidosis, because stool bicarbonate loss generates unmeasured organic anions that accumulate in the extracellular fluid.

D. Proximal renal tubular acidosis, because diarrhea directly inhibits proximal bicarbonate reclamation even when kidney function is otherwise normal.

E. Gastrointestinal bicarbonate loss causing a normal-anion-gap metabolic acidosis with intact renal acid excretion.

131. During a dialysis-unit assessment, a 49-year-old woman receiving maintenance dialysis with severe hyperkalemia receives intravenous insulin and glucose. Based on these findings, which effect best describes of insulin on potassium?

A. It blocks epithelial sodium channels in the collecting duct, causing rapid kaliuresis even in patients with minimal residual kidney function.

B. It increases glomerular filtration acutely, allowing filtered potassium excretion to rise independently of residual nephron function.

C. It chelates extracellular potassium in plasma, forming a nonconductive complex that directly protects the myocardium from depolarization.

D. It increases gastrointestinal potassium binding, thereby removing potassium from the body through stool within several minutes.

E. It stimulates sodium-potassium ATPase activity and shifts potassium from the extracellular fluid into cells without removing it from the body.

132. During an outpatient nephrology consultation, a 46-year-old man on an aminoglycoside develops nonoliguric AKI with granular casts after several days of therapy. Given this presentation, what is the likely lesion?

A. Proximal tubular epithelial toxicity causing aminoglycoside-associated acute tubular injury.

B. Renal-vein thrombosis, because aminoglycosides primarily injure venous endothelium and cause thrombotic occlusion.

C. Minimal change disease, because the characteristic toxicity is isolated podocyte effacement with nephrotic proteinuria.

D. Obstructive crystal nephropathy, because aminoglycosides routinely precipitate as crystals in the distal ureter.

E. Acute anti-GBM glomerulonephritis, because aminoglycosides bind type IV collagen and produce linear IgG staining.

133. During inpatient nephrology rounds, a hospitalized patient with sodium 130 mEq/L has measured serum osmolality 306 mOsm/kg and glucose 620 mg/dL. Given this presentation, what type of hyponatremia is present?

A. Hypovolemic hypotonic hyponatremia, because osmotic diuresis always lowers effective tonicity despite severe hyperglycemia.

B. Hypertonic translocational hyponatremia caused by glucose drawing water from cells into the extracellular space.

C. Reset osmostat, because a stable lower sodium set point is defined by high measured osmolality during marked glucose elevation.

D. Pseudohyponatremia from excess lipid, because pseudohyponatremia characteristically raises measured serum osmolality above normal.

E. Hypotonic SIADH, because hyperglycemia does not materially affect serum tonicity or the measured sodium concentration.

134. At a kidney clinic follow-up visit, a 68-year-old man with severe CKD requires a renally eliminated medication with a narrow therapeutic index. Given this presentation, what prescribing principle is most important?

A. Adjust dose or dosing interval to estimated kidney clearance and monitor drug levels or clinical toxicity when available.

B. Assume dialysis removes every drug completely and therefore give the full daily dose only before each treatment.

C. Double the dose to overcome reduced renal blood flow because lower GFR reduces delivery of drug to target tissues.

D. Base all dosing on serum creatinine concentration alone without considering age, body size, trajectory, or whether kidney function is changing.

E. Use the standard dose because kidney failure affects only endogenous solutes and has no effect on medication clearance.

135. During an urgent nephrology assessment, a child develops abrupt edema and nephrotic-range proteinuria after a viral illness. Complement is normal and kidney function is preserved. Given this presentation, what lesion is most likely?

A. Membranoproliferative glomerulonephritis, which usually has immune or complement abnormalities and a more inflammatory presentation.

B. Poststreptococcal glomerulonephritis, which typically causes a nephritic syndrome with hematuria and low complement.

C. Minimal change disease with diffuse podocyte foot-process effacement on electron microscopy.

D. Anti-GBM disease, which causes rapidly progressive nephritis and may involve pulmonary hemorrhage.

E. Acute tubular necrosis, which produces granular casts and AKI.

136. During a dialysis-unit assessment, a 49-year-old man receiving maintenance dialysis has very high PTH, bone pain, subperiosteal resorption, and elevated alkaline phosphatase. Based on these findings, which renal osteodystrophy pattern is most likely?

A. Paget disease, which is unrelated to kidney failure and would not be expected to track with severe secondary hyperparathyroidism.

B. Osteomalacia from isolated vitamin C deficiency, which causes impaired mineralization without the characteristic hyperparathyroid high-turnover pattern.

C. High-turnover bone disease from severe secondary hyperparathyroidism, classically osteitis fibrosa.

D. Adynamic bone disease, which is characterized by suppressed remodeling and typically low.

E. Osteoporosis alone, which can reduce bone mass but does not specifically explain marked PTH elevation and subperiosteal resorption.

137. During an intensive-care nephrology consultation, a 53-year-old woman with severe AKI continues metformin and develops shock with profound lactic acidosis. Why can kidney failure increase concern about the drug?

A. Metformin is renally cleared, so severe acute loss of kidney function can promote accumulation when other causes of lactate generation are present.

B. Metformin is metabolized entirely by the liver and kidney function has no effect on exposure or toxicity.

C. Kidney failure accelerates metformin elimination, so toxicity becomes less likely as GFR falls.

D. The drug directly generates ketone bodies in the kidney, so lactic acidosis is unrelated to accumulation during AKI.

E. Metformin blocks distal hydrogen secretion and causes a pure normal-anion-gap renal tubular acidosis.

138. At a hypertension clinic evaluation, a 64-year-old man with CKD starts an SGLT2 inhibitor and eGFR falls modestly during the first two weeks while blood pressure and volume status remain stable. Given this presentation, what is the best interpretation?

A. The only safe response is aggressive intravenous fluid administration until eGFR returns above the pretreatment value, even without hypovolemia.

B. The fall reflects progressive diabetic nephropathy regardless of diabetes status and is unrelated to changes in tubuloglomerular feedback.

C. Any fall in eGFR after an SGLT2 inhibitor proves acute tubular necrosis and requires permanent discontinuation of the drug.

D. A small early hemodynamic dip in eGFR can be expected and does not by itself require stopping therapy if the patient is otherwise stable.

E. The decline means the drug has caused bilateral renal artery thrombosis because SGLT2 inhibition should immediately increase measured GFR.

139. During inpatient nephrology rounds, a 75-year-old woman with advanced uremia has persistent mucosal bleeding despite a normal platelet count and coagulation studies. Based on these findings, which short-term therapy can improve platelet function before an urgent procedure?

A. Aspirin should be given because inhibiting platelet cyclooxygenase restores adhesion when uremic toxins interfere with von Willebrand factor binding.

B. Desmopressin can transiently improve uremic platelet dysfunction while definitive management includes dialysis and correction of anemia when appropriate.

C. Protamine is first-line therapy even without heparin exposure because uremia causes endogenous heparin accumulation in all patients with kidney failure.

D. Recombinant erythropoietin acts within minutes and is the preferred sole emergency treatment for active uremic mucosal bleeding.

E. Warfarin should be started because uremic bleeding is caused by excess vitamin K-dependent clotting factor activity.

140. At a kidney clinic follow-up visit, a 38-year-old man with chronic toluene exposure has weakness, hypokalemia, and a normal-anion-gap metabolic acidosis with a high urinary potassium loss. Based on these findings, which mechanism is most likely?

A. Toluene generates a persistent pure metabolic alkalosis because hippurate excretion obligately retains bicarbonate in the extracellular space.

B. Toluene directly blocks aldosterone receptors, producing the hyperkalemic type 4 renal tubular acidosis pattern.

C. The solvent causes pseudohypokalemia by interfering with ion-selective electrodes, while whole-body potassium stores remain unchanged.

D. Renal loss of potassium and ammonium salts during excretion of organic acid metabolites can produce hypokalemic hyperchloremic acidosis.

E. Toluene causes isolated respiratory acidosis by depressing ventilation, so the low bicarbonate reflects compensation.

141. During an urgent nephrology assessment, a 49-year-old woman with diabetic ketoacidosis has bicarbonate 8 mEq/L and PaCO2 20 mm Hg. Based on these findings, which interpretation is most accurate?

A. The respiratory compensation is appropriate for a primary high-anion-gap metabolic acidosis by Winter's relationship.

B. There is a primary metabolic alkalosis because bicarbonate below normal indicates renal retention of acid.

C. The values cannot be interpreted without urine chloride because serum bicarbonate and PaCO2 do not permit assessment of respiratory compensation.

D. There is a primary respiratory alkalosis without metabolic acidosis because the low PaCO2 fully explains the low serum bicarbonate.

E. There is a superimposed respiratory acidosis because any PaCO2 below 40 mm Hg is inadequate compensation for metabolic acidosis.

142. At a transplant-nephrology follow-up visit, a 60-year-old man whose native kidney disease was IgA nephropathy asks whether the disease can return after transplantation. Given this presentation, what is correct?

A. IgA nephropathy cannot recur because the donor kidney lacks the recipient’s immune system and therefore cannot receive circulating IgA complexes.

B. Recurrence is prevented completely by any standard maintenance immunosuppressive regimen.

C. IgA recurrence affects only the native kidneys and cannot involve an allograft under any circumstances.

D. IgA nephropathy can recur in the allograft, although recurrence severity and effect on graft survival vary.

E. Recurrence always occurs within the first 24 hours and causes immediate irreversible graft thrombosis.

143. At a hypertension clinic evaluation, a 36-year-old woman has severe hypertension and an abdominal bruit. Imaging shows alternating stenosis and dilation of a renal artery. Given this presentation, what is the likely diagnosis?

A. Atherosclerotic renal artery stenosis, which typically causes smooth ostial plaque in older patients with diffuse vascular disease.

B. Fibromuscular dysplasia causing a characteristic beaded appearance of the renal artery.

C. Renal-vein thrombosis, which causes venous filling defects.

D. Polyarteritis nodosa, which usually produces diffuse microaneurysms.

E. Autosomal dominant polycystic kidney disease, which causes renal cysts but not a beaded renal artery morphology.

144. During an outpatient nephrology consultation, a biopsy report uses the term 'membranoproliferative pattern of injury.' Given this presentation, what does this phrase imply?

A. A synonym for minimal change disease because both have normal light microscopy and podocyte effacement.

B. A purely vascular lesion confined to renal arteries and unrelated to glomerular immune or complement injury.

C. A single inherited disease caused only by collagen IV mutations, making serologic evaluation unnecessary.

D. A histologic pattern that can arise from several immune-complex or complement-mediated diseases.

E. A diagnosis that excludes monoclonal gammopathy, infection, autoimmune disease, and complement dysregulation.

145. At a hypertension clinic evaluation, a child develops cola-colored urine, edema, and hypertension two weeks after streptococcal pharyngitis. C3 is low. Based on these findings, which diagnosis best accounts for this presentation?

A. IgA nephropathy, which typically causes hematuria at the time of infection and usually has normal complement.

B. Poststreptococcal infection-related glomerulonephritis with a nephritic syndrome and transient complement consumption.

C. Alport syndrome, which is inherited and does not characteristically cause transient hypocomplementemia after pharyngitis.

D. Minimal change disease, which produces nephrotic syndrome and usually lacks low complement or red-cell casts.

E. Renal-vein thrombosis, which can cause hematuria but does not explain low complement after streptococcal infection.

146. At a kidney clinic follow-up visit, a man with angiokeratomas, neuropathic pain, cardiomyopathy, and CKD has a family history consistent with X-linked inheritance. Based on these findings, which metabolic disease is most likely?

A. Cystinosis, which usually presents much earlier with Fanconi syndrome and corneal cystine crystals.

B. Amyloid light-chain disease, which is acquired and is not an X-linked lysosomal enzyme deficiency.

C. Fabry disease from alpha-galactosidase A deficiency with glycosphingolipid accumulation.

D. Primary hyperoxaluria, which causes oxalate overproduction, stones, and nephrocalcinosis.

E. Alport syndrome, which is a collagen IV disorder and does not cause angiokeratomas from lysosomal lipid storage.

147. During a dialysis-unit assessment, a 56-year-old woman receiving maintenance dialysis has persistently low PTH, low bone turnover on biopsy, and repeated calcium loading. Based on these findings, which bone disorder is most likely?

A. Osteomalacia from severe vitamin C deficiency, which is the dominant cause of low-turnover renal bone disease in modern dialysis populations.

B. Paget disease, which causes focal high-turnover bone lesions and is unrelated to PTH suppression or CKD-mineral bone management.

C. Adynamic bone disease from excessive suppression of PTH signaling and skeletal bone turnover.

D. Osteitis fibrosa cystica, which is a high-turnover lesion driven by marked secondary hyperparathyroidism.

E. Primary hyperparathyroid bone disease, which requires autonomous high PTH and does not fit a persistently low circulating PTH concentration.

148. At a multidisciplinary renal conference, a 78-year-old man with type 2 diabetes, albuminuric CKD, and persistent albuminuria despite optimized ACE inhibitor and SGLT2 inhibitor therapy has normal

potassium. Based on these findings, which additional class may reduce kidney and cardiovascular risk in appropriate patients?

A. High-dose acetazolamide, because chronic bicarbonaturia suppresses diabetic glomerular injury and substitutes for mineralocorticoid receptor blockade.

B. Chronic fludrocortisone, because mineralocorticoid receptor activation reduces albuminuria and lowers the risk of hyperkalemia in diabetic CKD.

C. Routine dual ACE inhibitor and ARB therapy, because combined blockade has superior hard kidney outcomes and less hyperkalemia than either drug alone.

D. A nonsteroidal mineralocorticoid receptor antagonist such as finerenone with close potassium monitoring.

E. A long-term NSAID, because afferent arteriolar vasoconstriction improves diabetic glomerular hyperfiltration without increasing kidney injury risk.

149. During a dialysis-unit assessment, an 89-year-old with advanced CKD, metastatic cancer, severe frailty, and repeated hospitalizations asks whether dialysis is mandatory. Based on these findings, which approach is most appropriate?

A. Tell the patient that dialysis will certainly prolong life and preserve independence regardless of metastatic cancer and severe frailty.

B. Start dialysis automatically because age, comorbidity, prognosis, and patient values should never influence treatment once eGFR reaches kidney-failure range.

C. Withhold all symptom treatment if dialysis is declined because conservative kidney management means no active medical care for complications of kidney failure.

D. Use shared decision-making that compares dialysis with comprehensive conservative kidney management in light of goals, prognosis, symptoms, and treatment burden.

E. Delay discussion until the patient loses decision-making capacity because advance planning can bias patients against life-prolonging kidney replacement therapy.

150. During an outpatient nephrology consultation, a 52-year-old man taking an ACE inhibitor and diuretic develops AKI shortly after adding high-dose ibuprofen. Based on these findings, which drug interaction is most important?

A. NSAID-induced afferent vasoconstriction combines with reduced effective volume and efferent dilation to lower glomerular filtration pressure.

B. ACE inhibitors and NSAIDs together block tubular creatinine secretion without altering true GFR, so the AKI is always laboratory artifact.

C. Ibuprofen dilates the afferent arteriole, while ACE inhibition constricts the efferent arteriole, producing excessive glomerular hyperfiltration.

D. The three drugs precipitate nephrotic syndrome by causing immediate podocyte foot-process fusion in nearly all patients.

E. NSAIDs increase prostaglandin production and therefore amplify diuretic natriuresis until renal perfusion becomes excessive.

151. During inpatient nephrology rounds, a young adult has hemoptysis and rapidly progressive glomerulonephritis. Immunofluorescence shows linear IgG along glomerular capillary walls. Given this presentation, what is the diagnosis?

A. IgA nephropathy, which shows mesangial IgA.

B. C3 glomerulopathy, which shows dominant C3 deposition from complement dysregulation.

C. FSGS, which is a pattern of sclerosis and does not produce linear glomerular immunoglobulin deposition.

D. Membranous nephropathy, which shows granular capillary-wall immune deposits.

E. Anti-glomerular basement membrane disease producing linear IgG staining and a pulmonary-renal syndrome.

152. At a kidney clinic follow-up visit, a 74-year-old man with chronic hypercapnic COPD has PaCO2 60 mm Hg and bicarbonate 33 mEq/L. Based on these findings, which interpretation is most likely?

A. Mixed respiratory alkalosis and metabolic acidosis, because chronic lung disease lowers PaCO2 and bicarbonate together.

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

C. Acute respiratory acidosis, because bicarbonate rises by roughly 8 to 10 mEq/L immediately after any acute PaCO2 increase of 20 mm Hg.

D. Normal acid-base physiology, because PaCO2 and bicarbonate can rise in parallel without reflecting a primary respiratory disorder.

E. Primary metabolic alkalosis with no respiratory component, because an elevated bicarbonate fully explains the high PaCO2 through hypoventilation.

153. During a dialysis-unit assessment, a 63-year-old woman receiving maintenance dialysis has uncomfortable leg sensations at rest in the evening that improve with movement and disrupt sleep. Based on these findings, which diagnosis best accounts for this presentation?

A. Dialysis disequilibrium syndrome, which causes acute neurologic symptoms during initial dialysis.

B. Calciphylaxis, which causes severe focal skin pain and ischemic plaques.

C. Renal osteodystrophy, which can cause bone pain but does not characteristically improve immediately when the legs are moved.

D. Restless legs syndrome, which is common in advanced CKD and may be worsened by iron deficiency.

E. Uremic pericarditis, which produces positional chest discomfort and a friction rub.

154. During a dialysis-unit assessment, a long-term peritoneal dialysis patient has progressively poor ultrafiltration and increasing transport of small solutes on peritoneal equilibration testing. Given this presentation, what mechanism can contribute?

A. The peritoneum becomes completely impermeable to glucose, causing glucose to remain intravascular and eliminating all osmotic water movement.

B. Residual kidney function directly closes aquaporin channels in the peritoneum, so loss of ultrafiltration proves native GFR has improved substantially.

C. Small-solute transport falls to zero, trapping urea in blood and causing a stronger glucose gradient that paradoxically lowers ultrafiltration.

D. Changes in the peritoneal membrane can increase solute transport and dissipate the glucose osmotic gradient more rapidly, reducing ultrafiltration.

E. Peritoneal dialysis failure is caused only by catheter migration; membrane transport characteristics never change with long treatment duration.

155. During a hospital nephrology consultation, a 59-year-old woman on CRRT with regional citrate anticoagulation develops rising total calcium, falling ionized calcium, and increasing calcium infusion needs. Given this presentation, what complication should be suspected?

A. Hyperparathyroidism, because PTH suppresses ionized calcium only during CRRT and raises total calcium through citrate binding.

B. Acute phosphate nephropathy, because phosphate crystals within the filter consume ionized calcium while increasing serum total calcium.

C. Heparin-induced thrombocytopenia, because citrate directly forms antibodies against platelet factor 4 and causes calcium chelation.

D. Dialysis disequilibrium, because rapid urea removal selectively increases the total-to-ionized calcium ratio.

E. Citrate accumulation with impaired citrate metabolism and systemic calcium chelation.

156. During an outpatient nephrology consultation, a 59-year-old woman with nephrotic syndrome has circulating anti-PLA2R antibodies. Based on these findings, which diagnosis is most likely?

A. Diabetic nephropathy, because anti-PLA2R levels track glycemic exposure and predict nodular mesangial sclerosis.

B. Anti-GBM disease, because PLA2R is the principal antigen in the glomerular basement membrane targeted in pulmonary-renal syndrome.

C. Primary membranous nephropathy driven by autoantibodies against a podocyte antigen.

D. Minimal change disease, because anti-PLA2R antibodies identify diffuse foot-process effacement without subepithelial immune deposits.

E. IgA nephropathy, because anti-PLA2R antibodies are directed against mesangial IgA-containing immune complexes.

157. During inpatient nephrology rounds, a 81-year-old woman with decompensated cirrhosis has edema, ascites, and sodium 126 mEq/L. Given this presentation, what is the main mechanism of the hyponatremia?

A. Pseudohyponatremia from high plasma proteins is expected because cirrhosis usually produces extreme hyperproteinemia.

B. Reduced effective arterial blood volume causes nonosmotic vasopressin release and water retention despite total-body volume expansion.

C. Primary renal sodium wasting from absent aldosterone activity is the dominant mechanism in most patients with advanced cirrhosis.

D. Hypertonicity from retained urea draws water out of cells and is the principal explanation for low sodium in uncomplicated cirrhosis.

E. Complete suppression of vasopressin causes excessive renal water loss that secondarily lowers serum sodium in edematous states.

158. At a kidney clinic follow-up visit, a 44-year-old man with chronic SIADH has sodium 118 mEq/L and a generalized seizure. Based on these findings, which immediate treatment is most appropriate?

A. Restrict water alone and wait for spontaneous correction because chronic hyponatremia makes urgent neurologic therapy unnecessary.

B. Infuse isotonic saline rapidly until sodium normalizes because normal saline reliably corrects SIADH without worsening water retention.

C. Administer desmopressin alone to lower urine output before any sodium-raising therapy because neurologic symptoms do not alter urgency.

D. Give hypertonic saline in controlled boluses to achieve a prompt small rise in serum sodium while monitoring closely.

E. Start oral urea as the only emergency therapy because gradual osmole loading is preferred even during active hyponatremic seizures.

159. During an intensive-care nephrology consultation, a 55-year-old woman with CKD has hypertension and severely increased albuminuria. Based on these findings, which antihypertensive class is particularly useful for reducing albuminuria and slowing progression when tolerated?

A. A short-acting alpha blocker alone because reducing sympathetic tone lowers albuminuria more reliably than renin-angiotensin system blockade.

B. An ACE inhibitor or angiotensin receptor blocker titrated to a tolerated evidence-based dose.

C. A nondihydropyridine calcium-channel blocker must replace all renin-angiotensin blockade because ACE inhibitors increase intraglomerular pressure.

D. Hydralazine monotherapy is preferred because it reduces protein filtration directly without affecting potassium or glomerular hemodynamics.

E. A direct renin inhibitor combined routinely with an ACE inhibitor because triple renin-angiotensin blockade provides greater renal protection.

160. At a hypertension clinic evaluation, a 66-year-old man with resistant hypertension snores loudly, has witnessed apneas, and daytime sleepiness. Given this presentation, what secondary contributor should be addressed?

A. Obstructive sleep apnea, which is strongly associated with difficult-to-control hypertension and nocturnal sympathetic activation.

B. Renal tubular acidosis, because bicarbonate loss is the principal cause of snoring and nocturnal blood-pressure surges.

C. Central diabetes insipidus, because nocturnal polyuria is the usual mechanism linking sleep symptoms to persistent hypertension.

D. Nephrotic syndrome, because proteinuria itself produces recurrent upper-airway obstruction during sleep.

E. Chronic respiratory alkalosis, because hypocapnia during sleep directly causes daytime resistant hypertension.

161. During a hospital nephrology consultation, a 65-year-old man has eGFR 52 mL/min/1.73 m2 on one outpatient measurement and no other kidney abnormalities. Given this presentation, what is required before labeling this chronic kidney disease on the basis of low GFR alone?

A. Defer until eGFR falls below 30 mL/min/1.73 m2 because stages above that threshold are considered normal aging.

B. Repeat the creatinine after 24 hours because two abnormal values on consecutive days establish chronicity regardless of clinical context.

C. Demonstrate that the reduced GFR or another marker of kidney damage persists for at least three months.

D. Document nephrotic-range proteinuria because reduced GFR without heavy proteinuria cannot meet criteria for chronic kidney disease.

E. Obtain a kidney biopsy because histologic confirmation is required whenever eGFR is below 60 mL/min/1.73 m2.

162. At a hypertension clinic evaluation, a 51-year-old has abrupt worsening of hypertension, recurrent flash pulmonary edema, and asymmetric kidneys. Based on these findings, which secondary cause is most concerning?

A. Hemodynamically important renal artery stenosis, particularly atherosclerotic renovascular disease.

B. Isolated nephrogenic diabetes insipidus, because polyuria produces recurrent pulmonary edema through sodium retention.

C. Distal renal tubular acidosis, because chronic acid retention commonly causes flash pulmonary edema and renal artery narrowing.

D. Primary polydipsia, because excess water intake produces renal asymmetry and episodic severe hypertension.

E. Essential hypertension alone, because flash pulmonary edema is a typical benign manifestation of uncomplicated primary hypertension.

163. During inpatient nephrology rounds, a 51-year-old woman with mild lifelong hypercalcemia has a family history of similar findings, normal renal function, and very low fractional urinary calcium excretion. Based on these findings, which diagnosis is most likely?

A. Multiple myeloma, which causes acquired hypercalcemia with other systemic findings.

B. Sarcoidosis, which increases calcitriol production and typically suppresses PTH.

C. Primary hyperparathyroidism, which usually produces greater urinary calcium excretion and is more likely to cause kidney stones or skeletal complications.

D. Milk-alkali syndrome, which is an acquired disorder linked to calcium and alkali intake and does not produce inherited hypocalciuria.

E. Familial hypocalciuric hypercalcemia from altered calcium-sensing receptor function and reduced urinary calcium excretion.

164. At a metabolic stone clinic visit, a 45-year-old woman has recurrent nephrolithiasis, calcium 11.2 mg/dL, phosphate 2.2 mg/dL, and an inappropriately elevated parathyroid hormone level. Based on these findings, which diagnosis best accounts for this presentation?

A. Malignancy-associated hypercalcemia, which generally suppresses intact parathyroid hormone when PTH-related peptide or bone resorption is responsible.

B. Vitamin D intoxication, which tends to increase intestinal phosphate absorption and suppress endogenous parathyroid hormone secretion.

C. Hypoparathyroidism, which produces low calcium with high phosphate.

D. Familial hypocalciuric hypercalcemia, which usually has low urinary calcium excretion and is less commonly associated with recurrent stones.

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

165. At a hypertension clinic evaluation, an older patient with isolated systolic hypertension has a wide pulse pressure and stiff arteries. Based on these findings, which vascular change is most responsible?

A. Markedly increased aortic compliance stores more stroke volume and therefore raises systolic pressure while lowering diastolic pressure.

B. Reduced large-artery compliance increases systolic pressure and pulse pressure as the aorta loses its buffering function.

C. Decreased stroke volume is the defining cause because less ejected blood always produces a larger arterial pulse pressure.

D. Loss of venous capacitance alone determines isolated systolic hypertension without any contribution from large-artery stiffness.

E. Reduced systemic vascular resistance from generalized arteriolar dilation is sufficient to create persistent isolated systolic hypertension.

166. At a multidisciplinary renal conference, a 36-year-old man with advanced CKD needs iodinated contrast for an urgent, potentially lifesaving vascular study. Based on these findings, which approach best balances renal and clinical risk?

A. Give prophylactic high-dose NSAIDs because afferent vasoconstriction protects tubules from contrast-associated oxidative stress.

B. Delay all urgent imaging until creatinine normalizes because missing a vascular emergency carries less risk than any contrast exposure.

C. Use contrast when the diagnostic benefit is important, minimize avoidable nephrotoxic exposures, and optimize volume status.

D. Use repeated contrast doses to precondition the kidney because more exposure reduces.

E. Never give iodinated contrast at low GFR because the risk of permanent dialysis is certain regardless of indication or preventive measures.

167. During a hospital nephrology consultation, a stable adult with CKD asks about dietary protein. Based on these findings, which general principle is most appropriate?

A. Protein intake has no effect on uremic solute generation, so dietary counseling is unnecessary even as kidney function declines.

B. Eliminate nearly all dietary protein regardless of nutritional status because protein-energy wasting has little effect on outcomes in chronic kidney disease.

C. Use a very high-protein diet in all CKD because increased nitrogen load reduces intraglomerular pressure and slows nephron loss.

D. Avoid excessive protein intake and individualize a moderate protein target while maintaining adequate calories and preventing malnutrition.

E. Restrict calories together with protein because negative energy balance prevents urea production and protects against CKD-related muscle loss.

168. During a dialysis-unit assessment, a 58-year-old man on peritoneal dialysis develops diffuse abdominal pain and cloudy effluent. Based on these findings, which is the most important immediate diagnostic step?

A. Order only a serum procalcitonin because peritoneal fluid analysis does not distinguish infectious peritonitis from mechanical abdominal pain.

B. Send peritoneal effluent for cell count with differential and culture while promptly evaluating and treating suspected peritonitis.

C. Obtain a kidney biopsy because cloudy dialysate most often reflects recurrent glomerulonephritis.

D. Stop all dialysis for several days without sampling the fluid because peritonitis cultures become more accurate after the effluent clears spontaneously.

E. Perform immediate nephrectomy because cloudy dialysate indicates irreversible native-kidney infection that cannot be treated through the peritoneal catheter.

169. During inpatient nephrology rounds, a 69-year-old woman with CKD and anemia has hemoglobin 9.4 g/dL. Before attributing the anemia entirely to erythropoietin deficiency, what evaluation is essential?

A. Perform bone marrow biopsy in every patient because peripheral blood and iron testing cannot identify treatable causes of anemia in chronic kidney disease.

B. Assess iron status and other reversible causes of anemia such as blood loss, inflammation, nutritional deficiencies, and hemolysis when indicated.

C. Start an erythropoiesis-stimulating agent immediately without iron studies because all anemia below 10 g/dL in CKD is caused solely by erythropoietin deficiency.

D. Ignore the anemia until dialysis begins because anemia complications do not occur and treatment has no role during nondialysis chronic kidney disease.

E. Give repeated red-cell transfusions before laboratory evaluation because transfusion is the safest first-line correction for any asymptomatic CKD anemia.

170. At a hypertension clinic evaluation, a 80-year-old man with chronic severe hypertension presents with hypertensive encephalopathy. Why should blood pressure not be normalized immediately?

A. High pressure must be maintained because hypertensive encephalopathy is caused by inadequate cerebral perfusion.

B. Only diastolic pressure affects autoregulation, so systolic pressure can be reduced to normal immediately without ischemic risk.

C. Antihypertensive treatment has no role until papilledema resolves spontaneously because lowering pressure worsens vasogenic edema in every case.

D. Normalizing pressure quickly is always safe because organ blood flow is independent of mean arterial pressure in chronic hypertension.

E. Chronic hypertension shifts autoregulation, so an excessive abrupt fall can reduce cerebral, coronary, and renal perfusion.

171. During an urgent nephrology assessment, a 43-year-old woman with proteinuric CKD starts an ACE inhibitor and has a modest early rise in creatinine. Based on these findings, which renal hemodynamic effect explains this change?

A. Afferent arteriolar constriction is the direct pharmacologic action of ACE inhibition and is the main reason creatinine rises after every dose.

B. ACE inhibitors obstruct the ureter through smooth-muscle spasm, producing postrenal azotemia and hydronephrosis.

C. The drug causes acute immune-complex glomerulonephritis, so any creatinine increase proves an allergic renal lesion.

D. Efferent arteriolar dilation lowers intraglomerular pressure and can produce a predictable initial fall in GFR.

E. ACE inhibition increases angiotensin II signaling at the efferent arteriole and therefore raises intraglomerular pressure.

172. At a multidisciplinary renal conference, a 54-year-old man with CKD and diabetes develops vomiting and poor oral intake while taking an SGLT2 inhibitor and diuretic. Given this presentation, what medication-safety principle is most appropriate?

A. Stop all medications permanently after any intercurrent illness because resuming kidney-protective therapy after recovery is unsafe in chronic kidney disease.

B. Continue every chronic medication unchanged because acute illness never alters the risk-benefit balance of SGLT2 inhibitors, diuretics, or RAS blockade.

C. Temporarily reassess medications that can worsen volume depletion or ketoacidosis risk during acute illness and restart them when clinically stable.

D. Add an NSAID to preserve GFR during dehydration because prostaglandin inhibition improves afferent arteriolar blood flow in volume depletion.

E. Double the SGLT2 inhibitor dose because reduced oral intake increases the need for glucosuria to prevent stress hyperglycemia and kidney injury.

173. During a hospital nephrology consultation, a 65-year-old woman with tinnitus, nausea, tachypnea, and salicylate overdose has pH 7.44, PaCO2 22 mm Hg, and bicarbonate 14 mEq/L. Given this presentation, what acid-base disorder is present?

A. A normal acid-base state, because a near-normal pH excludes clinically significant simultaneous metabolic and respiratory disorders.

B. A mixed respiratory acidosis and metabolic alkalosis, because tinnitus is associated with hypoventilation and gastric acid retention.

C. A pure compensated metabolic acidosis, because salicylates do not directly stimulate the respiratory center early in toxicity.

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

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

174. During a dialysis-unit assessment, a 76-year-old man with advanced CKD on dialysis develops multiple acquired renal cysts over years of therapy. Why is surveillance attention sometimes warranted?

A. The cysts indicate unrecognized ADPKD in every case and therefore require intracranial aneurysm screening for all dialysis patients.

B. Acquired cystic kidney disease is associated with an increased risk of renal cell carcinoma compared with the general population.

C. The only complication is severe nephrotic syndrome because acquired cysts uniformly disrupt the glomerular filtration barrier.

D. The cysts inevitably transform into urothelial carcinoma, so prophylactic bilateral nephrectomy is required in every dialysis patient.

E. Acquired cystic disease reverses kidney failure by increasing nephron number, so the main concern is unexpected recovery of GFR.

175. During inpatient nephrology rounds, an older diabetic patient with active Staphylococcus aureus infection develops AKI, hematuria, proteinuria, and low C3. Based on these findings, which glomerular disease should be considered?

A. Anti-GBM disease because low C3 during active staphylococcal infection is specific for linear anti-basement-membrane antibodies.

B. Infection-related glomerulonephritis associated with ongoing bacterial infection.

C. FSGS because infection-related immune complexes routinely cause segmental scars without proliferative glomerular inflammation.

D. Minimal change disease because bacterial infection usually causes isolated podocyte effacement without complement activation.

E. Primary IgA nephropathy solely because IgA deposition can occur, making the active bacterial infection clinically irrelevant.

176. At a kidney clinic follow-up visit, a 50-year-old man treated with cisplatin has AKI and persistent hypomagnesemia. Based on these findings, which mechanism is most likely?

A. The drug causes isolated glomerular hyperfiltration, so electrolyte abnormalities must be unrelated to kidney effects.

B. The medication causes urinary magnesium precipitation as obstructing stones, making crystal nephropathy the only renal lesion.

C. Cisplatin stimulates aldosterone receptors and therefore causes magnesium retention.

D. Cisplatin blocks ADH release from the pituitary and causes pure central diabetes insipidus without tubular injury.

E. Direct tubular toxicity with renal magnesium wasting, often involving proximal and distal tubular segments.

177. During a dialysis-unit assessment, a 65-year-old with CKD not requiring dialysis has no known atherosclerotic cardiovascular disease. Based on these findings, which lipid-management principle is most appropriate?

A. Defer until dialysis starts before treating lipids because cardiovascular prevention has no benefit during nondialysis chronic kidney disease.

B. Treat only if nephrotic-range proteinuria develops because ordinary CKD without nephrosis does not independently increase cardiovascular risk.

C. Use fibrate monotherapy for every patient because triglyceride lowering has stronger CKD outcome evidence than statin-based treatment.

D. Avoid statins in all CKD because reduced GFR makes any LDL lowering more likely to cause kidney failure than cardiovascular benefit.

E. Use statin-based therapy according to CKD and cardiovascular-risk recommendations because CKD substantially increases atherosclerotic risk.

178. At a multidisciplinary renal conference, a 72-year-old man with a crush injury has dark urine, CK 60,000 U/L, positive dipstick for blood but few red cells, hyperkalemia, and AKI. Based on these findings, which diagnosis best explains the kidney injury?

A. Minimal change disease, because skeletal muscle injury causes podocyte effacement and nephrotic syndrome.

B. Rhabdomyolysis with myoglobin-mediated pigment nephropathy and acute tubular injury.

C. Glomerulonephritis, because dipstick heme with few red cells proves red-cell casts are being rapidly dissolved in urine.

D. Postrenal obstruction from intact erythrocytes, because few red cells on microscopy indicates a large ureteral clot burden.

E. Renal papillary necrosis, because muscle trauma directly causes papillary sloughing and releases myoglobin from the kidney.

179. During a hospital nephrology consultation, a 35-year-old woman with CKD stage 4 has persistent bicarbonate 17 mEq/L without respiratory disease. Why is chronic metabolic acidosis clinically important?

A. It can contribute to bone buffering, muscle catabolism, and progression of kidney disease, so reversible causes and alkali therapy should be considered.

B. Acidosis eliminates hyperkalemia risk by forcing potassium permanently into cells, making potassium monitoring unnecessary in advanced CKD.

C. Metabolic acidosis is protective in CKD because chronic acid retention suppresses protein breakdown and prevents bone mineral loss.

D. Low bicarbonate in CKD reflects only respiratory compensation and therefore should not influence nutrition, bone health, or kidney management.

E. Bicarbonate therapy is never used in CKD because any alkali administration predictably causes immediate pulmonary edema regardless of volume status.

180. During an outpatient nephrology consultation, a child has bloody diarrhea followed by microangiopathic hemolysis, thrombocytopenia, and AKI. Based on these findings, which syndrome is most likely?

A. Minimal change disease, which causes nephrotic syndrome without a thrombotic microangiopathy.

B. Shiga toxin–associated hemolytic uremic syndrome producing a diarrheal thrombotic microangiopathy.

C. Anti-GBM disease, which causes nephritis and pulmonary hemorrhage but not microangiopathic hemolytic anemia.

D. IgA nephropathy, which may follow infection but does not cause schistocytes and thrombocytopenia.

E. TTP from severe ADAMTS13 deficiency, which is less typical after a diarrheal prodrome in a child.

181. During inpatient nephrology rounds, a 57-year-old woman with eGFR 18 mL/min/1.73 m2 requires contrast-enhanced MRI. Based on these findings, which principle best reflects current gadolinium safety?

A. Linear high-risk gadolinium agents are preferred in advanced CKD because weaker chelation allows faster dialysis removal and less tissue deposition.

B. Give repeated high doses to overcome reduced filtration because lower GFR decreases tissue exposure and therefore reduces gadolinium-related risk.

C. Use a lower-risk macrocyclic or group II gadolinium agent only when clinically justified, at the lowest effective dose, with attention to severe CKD risk.

D. Gadolinium has no relationship to nephrogenic systemic fibrosis, so agent selection and kidney function never affect MRI contrast decisions.

E. All gadolinium agents are absolutely contraindicated at any eGFR below 60 because nephrogenic systemic fibrosis risk is identical across agents.

182. At a kidney clinic follow-up visit, a 68-year-old man receiving amphotericin B develops rising creatinine, hypokalemia, and hypomagnesemia. Given this presentation, what renal toxicity is characteristic?

A. Exclusive proximal glucose transporter blockade producing isolated glucosuria with no effect on potassium or magnesium.

B. Renal vasoconstriction plus direct tubular membrane injury causing reduced GFR and distal electrolyte wasting.

C. Immune-complex crescentic glomerulonephritis as the universal mechanism of amphotericin nephrotoxicity.

D. Renal artery thrombosis from drug-induced platelet aggregation, which explains nearly all creatinine rises during treatment.

E. Selective podocyte injury causing nephrotic syndrome without tubular electrolyte abnormalities.

183. During an intensive-care nephrology consultation, a 79-year-old woman with septic shock has lactate 8 mmol/L, bicarbonate 13 mEq/L, and an increased anion gap. Based on these findings, which is the most important treatment principle?

A. Give large sodium bicarbonate doses solely to normalize serum bicarbonate because correcting the number directly reverses cellular hypoxia.

B. Restrict oxygen delivery because high inspired oxygen worsens anaerobic metabolism and therefore increases lactate production in shock.

C. Restore tissue perfusion and treat the underlying cause of shock with appropriate resuscitation and source control.

D. Begin chronic oral alkali therapy because septic lactic acidosis is primarily caused by an isolated renal bicarbonate reabsorption defect.

E. Treat with acetazolamide to increase bicarbonate excretion because lowering serum bicarbonate improves oxygen unloading and lactate clearance.

184. At a multidisciplinary renal conference, a 42-year-old man with membranous nephropathy has albumin 1.8 g/dL and develops unilateral leg swelling. Given this presentation, what complication requires urgent evaluation?

A. Deep soft-tissue infection is the only important cause because nephrotic syndrome protects against venous thrombosis.

B. Venous thromboembolism, because severe nephrotic syndrome is a hypercoagulable state.

C. Compartment syndrome from renal salt wasting, because nephrotic syndrome lowers total-body sodium and causes intramuscular edema.

D. Acute arterial vasculitis, because loss of albumin selectively causes inflammatory thrombosis of medium arteries.

E. Calciphylaxis, because low albumin alone causes painful calcified plaques in patients with normal kidney function.

185. At a hypertension clinic evaluation, a 53-year-old woman with hypertension and episodic muscle weakness has potassium 2.7 mEq/L and bicarbonate 34 mEq/L. Renin is low and aldosterone is high. Based on these findings, which mechanism best fits?

A. Hypoaldosteronism increases distal potassium secretion and generates metabolic alkalosis despite reduced sodium reabsorption.

B. Autonomous mineralocorticoid activity increases distal sodium reabsorption and potassium and hydrogen secretion.

C. Renal salt wasting activates neither renin nor aldosterone and therefore produces hypertension with low potassium and alkalosis.

D. Loss of ENaC function causes sodium retention and suppressed renin while increasing potassium secretion in principal cells.

E. Carbonic anhydrase inhibition increases bicarbonate retention and suppresses potassium excretion, causing this low-renin phenotype.

186. At a hypertension clinic evaluation, a 64-year-old man has potassium 2.7 mEq/L, hypertension, metabolic alkalosis, suppressed renin, and elevated aldosterone. Based on these findings, which diagnosis is most likely?

A. Vomiting, which causes chloride-responsive alkalosis but does not typically suppress renin while producing autonomous aldosterone elevation.

B. Gitelman syndrome, which causes hypokalemic alkalosis with low or normal blood pressure and activation of renin and aldosterone.

C. Primary aldosteronism causing renal sodium retention, potassium wasting, and hydrogen ion secretion.

D. Liddle syndrome, which causes the same phenotype but suppresses aldosterone because ENaC activity is independent of mineralocorticoid signaling.

E. Renal artery stenosis, which produces secondary hyperaldosteronism with elevated.

187. During a dialysis-unit assessment, a hemodialysis patient receiving an erythropoiesis-stimulating agent has hemoglobin rising toward the normal range. Why should therapy not simply target a normal hemoglobin in every patient?

A. Normal hemoglobin always causes severe iron overload in CKD because each new red cell releases iron.

B. The only reason to limit ESA therapy is cost; clinical outcome studies show no difference in cardiovascular events across hemoglobin targets.

C. ESAs become ineffective once hemoglobin exceeds 10 g/dL because erythropoietin receptors disappear from erythroid precursors at that concentration.

D. Higher hemoglobin targets with ESAs can increase cardiovascular and thrombotic risk, so treatment balances symptoms, transfusion avoidance, and safety.

E. A normal hemoglobin worsens kidney filtration directly by increasing tubular oxygen tension, so anemia must be maintained to preserve nephron function.

188. At a transplant-nephrology follow-up visit, a 63-year-old woman with a kidney transplant develops rising creatinine two weeks after transplant. Biopsy shows interstitial inflammation and tubulitis without dominant antibody-mediated features. Given this presentation, what is the diagnosis?

A. Recurrent ADPKD.

B. Hyperacute rejection, which presents immediately after reperfusion with severe vascular injury.

C. BK nephropathy, which commonly shows viral cytopathic changes and usually occurs later during sustained immunosuppression.

D. Acute T-cell-mediated allograft rejection characterized by interstitial inflammation and tubulitis.

E. Chronic antibody-mediated rejection, which is characterized by chronic microvascular and basement-membrane remodeling.

189. During an urgent nephrology assessment, a 49-year-old woman with proteinuric CKD develops potassium 5.4 mEq/L after an ARB, but the drug has substantially reduced albuminuria and there are no ECG changes. Given this presentation, what is the best management principle?

A. Add an ACE inhibitor to the ARB because dual blockade lowers potassium by increasing distal sodium delivery and aldosterone secretion.

B. Permanently stop all renin-angiotensin system therapy at the first potassium value above normal because recurrent hyperkalemia cannot be mitigated.

C. Address reversible contributors and use dietary, diuretic, bicarbonate, or potassium-binding strategies when appropriate before abandoning beneficial RAS blockade.

D. Increase dietary potassium to stimulate renal potassium secretion because higher intake is the preferred adaptation strategy in proteinuric CKD.

E. Give chronic intravenous calcium because membrane stabilization lowers total-body potassium and allows the ARB to be continued indefinitely.

190. At a multidisciplinary renal conference, a malnourished patient with chronic sodium 105 mEq/L begins a brisk water diuresis after treatment of hypovolemia, and sodium rises 9 mEq/L in 5 hours. Based on these findings, which next step is most appropriate?

A. Add a loop diuretic to accelerate free-water loss because preventing cerebral edema requires continued rapid sodium elevation.

B. Continue the current fluids because rapid correction is desirable once renal perfusion and urine output have recovered.

C. Withhold all fluids and accept the rise because once correction exceeds the target there is no practical way to modify the course.

D. Give sodium bicarbonate to replace intracellular sodium because malnutrition lowers the risk of osmotic demyelination syndrome.

E. Stop further overcorrection and use desmopressin with electrolyte-free water as needed to relower or stabilize the sodium trajectory.

191. During a hospital nephrology consultation, a 71-year-old woman with vomiting and poor intake has AKI, orthostatic hypotension, bland urine sediment, and rapid improvement after isotonic fluid. Given this presentation, what category of AKI is most likely?

A. Prerenal AKI from reduced effective renal perfusion without established structural tubular injury.

B. Postrenal obstruction is most likely because vomiting directly compresses both ureters and causes hydronephrosis.

C. Acute tubular necrosis is proven because any creatinine rise during volume depletion represents structural tubular cell death.

D. Renal cortical necrosis is expected because brief orthostasis causes irreversible ischemic destruction of the cortex.

E. Acute glomerulonephritis is most likely because volume depletion characteristically causes red-cell casts and complement consumption.

192. At a metabolic stone clinic visit, a 34-year-old man after Roux-en-Y gastric bypass develops recurrent calcium oxalate stones and high urinary oxalate. Given this presentation, what mechanism is most likely?

A. Fat malabsorption binds intestinal calcium, leaving more free oxalate available for colonic absorption and urinary excretion.

B. Reduced intestinal sodium absorption lowers serum calcium and causes secondary cystinuria, leading to mixed cystine-calcium oxalate stones.

C. Bile acid depletion causes the kidney to synthesize oxalate directly, so intestinal absorption plays little role after bariatric surgery.

D. Gastric bypass increases urinary citrate markedly, and citrate itself is converted into oxalate in the collecting duct during alkaline urine formation.

E. The procedure raises urine pH enough to precipitate uric acid, and uric acid crystals then transform chemically into calcium oxalate.

193. During an intensive-care nephrology consultation, a 45-year-old woman receiving cisplatin develops tremor and ventricular ectopy. Magnesium is 0.8 mg/dL and potassium is 3.0 mEq/L. Given this presentation, what is the best management principle?

A. Restrict magnesium intake because low serum magnesium in cisplatin therapy usually reflects intracellular magnesium excess.

B. Give intravenous calcium as the only treatment because magnesium depletion is corrected indirectly once serum calcium concentration rises.

C. Replete magnesium and potassium while evaluating ongoing renal magnesium wasting from nephrotoxic tubular injury.

D. Use a loop diuretic to retain magnesium because loop blockade increases paracellular magnesium reabsorption in the thick ascending limb.

E. Replace potassium alone because hypomagnesemia does not affect renal potassium handling or cardiac electrical stability.

194. During an obstetric-nephrology consultation, a postpartum patient after catastrophic hemorrhage and disseminated intravascular coagulation remains anuric. Imaging suggests diffuse cortical nonperfusion. Given this presentation, what lesion is most concerning?

A. Nephrogenic diabetes insipidus, because cortical ischemia causes inability to concentrate urine and therefore profound polyuria.

B. Renal cortical necrosis from severe prolonged ischemic and microvascular injury.

C. Poststreptococcal glomerulonephritis, because postpartum DIC is a typical nephritogenic streptococcal trigger.

D. Uncomplicated prerenal azotemia, because persistent anuria with absent cortical perfusion indicates preserved renal parenchyma.

E. Minimal change disease, because obstetric hemorrhage causes selective podocyte effacement and abrupt anuria.

195. During an urgent nephrology assessment, a 67-year-old woman with diabetic ketoacidosis has serum potassium 5.3 mEq/L before treatment. Given this presentation, what is the most accurate statement about total-body potassium?

A. The serum potassium concentration directly measures exchangeable body potassium and can be used without considering redistribution or urinary losses.

B. Total-body potassium is usually depleted despite a normal or high initial serum potassium because osmotic diuresis causes substantial urinary losses.

C. Total-body potassium is increased because the elevated serum concentration proves that intracellular stores are also expanded before insulin therapy.

D. Total-body potassium is normal because acidosis prevents urinary potassium loss until insulin has been administered and glucose begins to fall.

E. Total-body potassium cannot change in ketoacidosis because potassium is an intracellular ion and therefore is not lost in urine during hyperglycemia.

196. At a hypertension clinic evaluation, a 78-year-old man with malignant hypertension has AKI, retinal hemorrhages, and arteriolar onion-skin thickening. Given this presentation, what vascular lesion is present?

A. Hyaline arteriolosclerosis only, which is the exclusive lesion in rapidly progressive malignant hypertension.

B. Amyloid deposition, which forms beta-pleated fibrils.

C. Hyperplastic arteriolosclerosis from severe hypertension-mediated vascular injury.

D. Cholesterol clefts, which indicate atheroembolization.

E. Fibrinoid glomerular crescents, which describe extracapillary proliferation.

197. At a hypertension clinic evaluation, a 41-year-old woman with suspected pheochromocytoma is being prepared for surgery. Based on these findings, which pharmacologic sequence is safest?

A. Begin a nonselective beta blocker first because unopposed alpha stimulation lowers blood pressure by reducing cardiac output.

B. Establish adequate alpha-adrenergic blockade and volume repletion before adding beta blockade if needed for tachycardia.

C. Start a thiazide as the sole preoperative therapy because chronic volume contraction protects against catecholamine surges.

D. Avoid alpha blockade because it increases the risk of intraoperative hypertensive crisis from catecholamine release.

E. Use beta blockade alone because catecholamine-mediated vasoconstriction is primarily beta-2 receptor dependent.

198. At a hypertension clinic evaluation, a 52-year-old man with CKD is taking ibuprofen daily for chronic back pain and develops a gradual rise in creatinine and worsening blood pressure. Given this presentation, what medication change is most appropriate?

A. Stop or minimize chronic NSAID exposure and use safer analgesic strategies because NSAIDs can reduce renal perfusion, retain sodium, and worsen CKD.

B. Add a second NSAID from another class because combining cyclooxygenase inhibitors reduces the renal adverse effects of each individual drug.

C. Continue ibuprofen unchanged because NSAIDs affect kidney function only in patients with normal GFR and are protective once CKD is established.

D. Switch to a nephrotoxic aminoglycoside for pain control because tubular injury lowers glomerular pressure and slows progression of chronic kidney disease.

E. Increase the NSAID dose because stronger prostaglandin inhibition dilates the afferent arteriole and improves GFR in chronic kidney disease.

199. During inpatient nephrology rounds, how can chronic metabolic acidosis worsen nutritional status in CKD?

A. Acidosis increases insulin sensitivity enough to prevent muscle loss, so correcting bicarbonate can paradoxically worsen protein-energy wasting.

B. Acidemia promotes muscle protein breakdown and negative nitrogen balance, contributing to weakness and protein-energy wasting.

C. Acidemia stimulates sustained muscle protein synthesis, so low bicarbonate is usually associated with increased lean mass.

D. Low bicarbonate causes only respiratory symptoms and has no meaningful effect on skeletal muscle metabolism or nutritional status.

E. Metabolic acidosis prevents amino acid release from muscle, making dietary protein restriction unnecessary in advanced chronic kidney disease.

200. At a kidney clinic follow-up visit, a 51-year-old man with small-cell lung cancer has sodium 121 mEq/L, low serum osmolality, urine osmolality 480 mOsm/kg, urine sodium 74 mEq/L, normal thyroid function, and a normal morning cortisol. He appears euvolemic. Based on these findings, which diagnosis best fits?

A. Primary adrenal insufficiency, which can mimic SIADH but should be supported by cortisol deficiency and often hyperkalemia.

B. Cirrhosis with arterial underfilling, which generally presents with clinical volume expansion and avid sodium retention.

C. Syndrome of inappropriate antidiuretic hormone secretion causing inappropriately concentrated urine despite hypotonicity.

D. Renal salt wasting from osmotic diuresis, which requires an ongoing filtered osmole load and usually a different volume picture.

E. Psychogenic polydipsia, in which suppression of vasopressin usually produces urine osmolality below about 100 mOsm/kg.

201. At a hypertension clinic evaluation, a 37-year-old woman with resistant hypertension is taking lisinopril, amlodipine, and hydrochlorothiazide. Kidney function and potassium permit additional therapy. Based on these findings, which class is commonly useful as a fourth agent after true resistance is confirmed?

A. A mineralocorticoid receptor antagonist, with potassium and kidney function monitored closely.

B. A second ACE inhibitor, because combining two agents from the same class improves pressure control without increasing adverse effects.

C. A potassium supplement regardless of baseline potassium, because improving potassium stores directly reverses all causes of resistant hypertension.

D. A short-acting beta agonist, because peripheral beta-2 stimulation is a standard chronic treatment for resistant hypertension.

E. An NSAID, because renal prostaglandin inhibition potentiates diuretic natriuresis and lowers blood pressure in resistant hypertension.

202. At a hypertension clinic evaluation, a 48-year-old man with diabetes and CKD has potassium 5.9 mEq/L, bicarbonate 17 mEq/L, a normal anion gap, and low renin and aldosterone activity. Based on these findings, which diagnosis best fits?

A. Distal type 1 renal tubular acidosis, which more often causes hypokalemia and an inappropriately high urine pH during systemic acidosis.

B. Respiratory acidosis, which raises bicarbonate through renal compensation.

C. Type 4 renal tubular acidosis from hyporeninemic hypoaldosteronism with impaired potassium and ammonium excretion.

D. Lactic acidosis, which would generally raise the anion gap.

E. Proximal type 2 renal tubular acidosis, which causes bicarbonate wasting but usually not persistent hyperkalemia from aldosterone deficiency.

203. During a hospital nephrology consultation, a 59-year-old woman with advanced CKD has recurrent mild hyperkalemia but no ECG changes. Based on these findings, which chronic strategy is most appropriate?

A. Avoid correcting metabolic acidosis because raising bicarbonate always increases serum potassium by driving potassium out of skeletal muscle cells.

B. Review dietary potassium, constipation, acidosis, and potassium-raising drugs, and use diuretics or potassium binders when appropriate to preserve beneficial therapies.

C. Give intravenous calcium at home every day because calcium removes potassium from the body and is the standard chronic treatment for stable hyperkalemia.

D. Use insulin injections without glucose between meals because repeated intracellular shifting is the safest long-term method of potassium control.

E. Stop all vegetables and fruits permanently because dietary potassium restriction should ignore nutritional quality and the patient's actual contributors.

204. During an outpatient nephrology consultation, a 70-year-old man has rapidly progressive glomerulonephritis, pulmonary hemorrhage, positive MPO-ANCA, and little immune deposition on biopsy. Given this presentation, what pattern is present?

A. Anti-GBM nephritis, which would show strong linear IgG staining.

B. Pauci-immune necrotizing crescentic glomerulonephritis associated with ANCA vasculitis.

C. Immune-complex membranous nephropathy, which is a nephrotic subepithelial-deposit disease.

D. Minimal change disease, which shows podocyte effacement without necrotizing crescents or pulmonary hemorrhage.

E. Diabetic glomerulosclerosis, which produces nodular mesangial expansion and chronic proteinuria.

205. At a transplant-nephrology follow-up visit, a 72-year-old man with a kidney transplant had FSGS as the cause of native kidney failure and develops massive proteinuria within days of transplantation. Given this presentation, what should be suspected?

A. Chronic calcineurin fibrosis, because years of drug exposure are not required for extensive interstitial scarring and immediate nephrotic syndrome.

B. Recurrent ADPKD, because cyst formation from recipient genes develops within days in a genetically normal donor kidney.

C. Transplant renal artery stenosis, because renovascular disease selectively causes massive protein loss within the first postoperative days.

D. Acute pyelonephritis, because bacterial infection is the usual cause of abrupt nephrotic-range proteinuria immediately after transplant.

E. Early recurrence of primary FSGS in the allograft, likely mediated by circulating podocyte-injuring factors.

206. At a hypertension clinic evaluation, a 44-year-old man with hypertension develops ankle edema on a dihydropyridine calcium-channel blocker but has clear lungs and stable weight. Given this presentation, what mechanism is most likely?

A. Dihydropyridines directly increase lymphatic permeability by causing inflammatory capillary destruction in most treated patients.

B. The drug causes severe venoconstriction and lowers capillary hydrostatic pressure, forcing fluid into the interstitium.

C. The edema results from complete inhibition of aldosterone synthesis and renal sodium wasting, leading to tissue fluid accumulation.

D. Calcium-channel blockade always causes acute nephrotic-range proteinuria, and the edema therefore proves new glomerular disease.

E. Preferential arteriolar dilation raises capillary hydrostatic pressure and can cause dependent edema without generalized sodium overload.

207. During an urgent nephrology assessment, a child has glucosuria despite normal serum glucose, phosphaturia, aminoaciduria, and bicarbonate wasting. Where is the principal defect?

A. A proximal tubular defect producing generalized Fanconi syndrome with loss of multiple filtered solutes.

B. The glomerular filtration barrier, because selective protein permeability defects cause glucosuria and aminoaciduria without hyperglycemia.

C. The renal pelvis, where defects cause hydronephrosis but not generalized loss of filtered solutes.

D. The thick ascending limb, which would mainly impair sodium-potassium-chloride transport and urinary concentrating ability.

E. The collecting-duct principal cell, which primarily regulates sodium, potassium, and water.

208. At a multidisciplinary renal conference, a 66-year-old man with oliguric AKI has severe pulmonary edema and hypoxemia despite appropriate diuretic therapy. Based on these findings, which next step is most appropriate?

A. Give an NSAID to reduce renal prostaglandins because afferent vasoconstriction improves pulmonary capillary fluid clearance.

B. Use hypertonic saline as definitive treatment because increasing serum osmolality draws lung water directly into the circulation for renal excretion.

C. Initiate kidney replacement therapy for refractory volume overload with respiratory compromise.

D. Defer for creatinine to exceed a fixed numeric threshold because dialysis cannot be started for volume indications alone.

E. Continue escalating fluid administration because pulmonary edema in AKI usually reflects intravascular depletion.

209. At a transplant-nephrology follow-up visit, a long-term kidney transplant recipient asks about cancer prevention. Based on these findings, which malignancy risk is particularly increased by chronic immunosuppression and sun exposure?

A. Cutaneous squamous-cell carcinoma and other nonmelanoma skin cancers promoted by chronic immunosuppression and UV exposure.

B. Pituitary adenoma, because glucocorticoid therapy directly causes monoclonal pituitary neoplasia.

C. Retinoblastoma, because adult transplant immunosuppression reactivates congenital retinal tumors in most recipients.

D. Testicular germ-cell cancer, because allograft immunosuppression specifically increases seminoma incidence more than skin cancer.

E. Osteosarcoma, because calcineurin inhibitors selectively transform osteoblasts and make bone cancer the dominant post-transplant malignancy.

210. During an obstetric-nephrology consultation, a pregnant patient at 29 weeks has new hypertension, proteinuria, headache, and elevated liver enzymes. Based on these findings, which diagnosis best integrates the findings?

A. Primary aldosteronism, because pregnancy-related endothelial disease does not cause proteinuria or neurologic symptoms.

B. White-coat hypertension, because proteinuria and liver abnormalities are expected consequences of anxiety during pregnancy.

C. Isolated gestational hypertension without severe features, because headache and hepatic injury do not alter the classification.

D. Renal artery stenosis, because any hypertension after 20 weeks of gestation is caused by renovascular disease.

E. Preeclampsia with severe features, requiring obstetric evaluation and management.

211. During inpatient nephrology rounds, a 51-year-old woman starts a proton-pump inhibitor and develops AKI with sterile pyuria and white-cell casts. Given this presentation, what drug-associated lesion should be suspected?

A. Acute uric acid nephropathy, because gastric acid suppression causes urate crystals to precipitate throughout the tubules.

B. Renal cortical necrosis, because proton-pump inhibitors routinely cause diffuse cortical ischemia through arterial thrombosis.

C. Acute interstitial nephritis from a drug-triggered hypersensitivity-type interstitial inflammatory reaction.

D. Minimal change disease, because white-cell casts are a defining feature of isolated podocyte injury.

E. Membranous nephropathy, because the typical renal toxicity is subepithelial immune-complex deposition with nephrotic syndrome.

212. At a metabolic stone clinic visit, a 62-year-old man has a staghorn calculus, alkaline urine, and recurrent infections with Proteus species. Given this presentation, what is the stone composition most likely to be?

A. Cystine, which reflects an inherited amino acid transport disorder and typically forms recurrent stones without requiring infection.

B. Xanthine, which is a rare stone type associated with purine metabolism or xanthine oxidase inhibition.

C. Magnesium ammonium phosphate formed in alkaline urine during infection with a urease-producing organism.

D. Uric acid, which is favored by persistently acidic urine and is unrelated to urease activity or infection-associated alkalinization.

E. Calcium oxalate, which is the most common stone overall but does not characteristically form infection-driven staghorn calculi from urease.

213. During an urgent nephrology assessment, a 73-year-old woman with sickle cell disease develops gross hematuria and sloughed papillary tissue. Based on these findings, which lesion is most likely?

A. Membranous nephropathy, because sickling selectively deposits immune complexes under podocytes and causes papillary sloughing.

B. Acute uric acid nephropathy, which causes crystal obstruction but does not characteristically produce sloughed papillae.

C. Renal cortical necrosis, which is confined to the outer cortex and typically follows obstetric catastrophe.

D. Renal papillary necrosis from medullary ischemia in the hypoxic, hypertonic renal medulla.

E. Polycystic kidney rupture, because sickle cell disease routinely produces acquired bilateral renal cysts that bleed into the collecting system.

214. At a multidisciplinary renal conference, a 27-year-old develops visible hematuria within one day of an upper respiratory infection. Complement is normal. Based on these findings, which disease is most likely?

A. Minimal change disease, which presents with nephrotic proteinuria.

B. IgA nephropathy causing synpharyngitic glomerular hematuria with mesangial IgA deposition.

C. Anti-GBM disease, which usually presents with rapidly progressive kidney injury.

D. Poststreptococcal glomerulonephritis, which usually occurs after a latent interval and often lowers complement.

E. Renal papillary necrosis, which causes nonglomerular bleeding and does not track closely with mucosal infections.

215. During a hospital nephrology consultation, a 47-year-old woman with psychogenic polydipsia has sodium 128 mEq/L. Based on these findings, which urine finding is most expected before any treatment?

A. Urine containing broad granular casts because water excess produces acute tubular epithelial injury before hyponatremia develops.

B. Urine osmolality fixed near plasma osmolality because psychogenic polydipsia causes an acquired inability to dilute or concentrate urine.

C. Urine osmolality above 500 mOsm/kg because excessive water intake directly stimulates vasopressin release in otherwise healthy kidneys.

D. Very dilute urine with osmolality near the physiologic minimum because vasopressin is appropriately suppressed.

E. Urine sodium consistently below 10 mEq/L because primary polydipsia necessarily activates intense renin-angiotensin signaling.

216. During an outpatient nephrology consultation, a 58-year-old man has a mildly elevated urine albumin-creatinine ratio on a random sample after strenuous exercise. Given this presentation, what is the best next step before diagnosing persistent albuminuria?

A. Begin dialysis planning because a single elevated albumin-creatinine ratio predicts imminent kidney failure regardless of eGFR.

B. Repeat the measurement, preferably with a first-morning specimen when transient confounders have resolved.

C. Ignore the result permanently because exercise-related albuminuria proves that future albumin measurements will be unreliable.

D. Start immunosuppression immediately because any albuminuria above the reference range establishes chronic glomerulonephritis.

E. Order a 24-hour urine every time because spot albumin-creatinine ratios cannot be used to assess chronic kidney disease.

217. At a hypertension clinic evaluation, a 69-year-old woman with edema, hypertension, rising creatinine, hematuria, and red-cell casts has which general renal syndrome?

A. An acute nephritic syndrome caused by inflammatory glomerular injury with an active urine sediment.

B. An isolated tubular concentrating defect, which causes polyuria without glomerular hematuria or red-cell casts.

C. A pure nephrotic syndrome, which is dominated by heavy proteinuria and hypoalbuminemia.

D. Postrenal obstruction, which may cause AKI but does not characteristically generate dysmorphic red cells and red-cell casts.

E. Prerenal azotemia, which usually has a bland sediment and lacks glomerular blood and casts.

218. At a kidney clinic follow-up visit, a 80-year-old man has rapidly progressive AKI, hematuria, proteinuria, and red-cell casts. Based on these findings, which action is most appropriate?

A. Delay evaluation for several months because glomerular inflammation usually resolves before irreversible kidney injury develops.

B. Urgently evaluate for rapidly progressive glomerulonephritis with serologies and kidney biopsy when feasible because disease-specific therapy may be time sensitive.

C. Give empiric NSAIDs because reducing glomerular blood flow is the standard treatment for crescentic glomerulonephritis.

D. Assume obstruction because red-cell casts arise only when the ureter is completely blocked.

E. Treat as uncomplicated prerenal azotemia because red-cell casts are expected during any acute fall in renal perfusion.

219. During an urgent nephrology assessment, a 43-year-old woman with oliguric AKI receives repeated large fluid boluses despite no evidence of fluid responsiveness and develops worsening edema. Why is this harmful?

A. Positive fluid balance can worsen pulmonary gas exchange, tissue edema, and venous congestion without restoring renal perfusion.

B. Additional crystalloid always increases urine output in intrinsic tubular injury, regardless of hemodynamic responsiveness.

C. Fluid overload improves every form of AKI by increasing renal interstitial pressure and therefore accelerating glomerular filtration.

D. Fluid accumulation lowers venous pressure and therefore protects the kidney from congestion.

E. Edema is harmless in critical illness because only intravascular volume affects organ function.

220. At a multidisciplinary renal conference, a 54-year-old man with long-standing diabetes has albuminuria, declining GFR, and biopsy showing mesangial expansion with nodular glomerulosclerosis. Given this presentation, what is the likely diagnosis?

A. Postinfectious glomerulonephritis, which more often shows diffuse proliferative injury and subepithelial humps.

B. Acute uric acid nephropathy, which is a tubular crystal process.

C. Anti-GBM disease, which produces crescentic nephritis and linear IgG staining.

D. Minimal change disease.

E. Diabetic kidney disease with Kimmelstiel-Wilson nodular mesangial sclerosis.

221. During a hospital nephrology consultation, a 65-year-old woman receiving trimethoprim develops potassium 6.0 mEq/L without a major change in GFR. Based on these findings, which mechanism best explains the hyperkalemia?

A. Trimethoprim blocks epithelial sodium channels in the collecting duct, reducing the lumen-negative potential that drives potassium secretion.

B. Trimethoprim causes obligate potassium release from skeletal muscle by uncoupling oxidative phosphorylation at standard therapeutic concentrations.

C. Trimethoprim inhibits proximal bicarbonate reabsorption, producing severe alkalosis that directly prevents distal potassium secretion.

D. Trimethoprim stimulates mineralocorticoid receptors, increasing distal sodium reabsorption and causing potassium to leave cells through sodium-potassium ATPase.

E. Trimethoprim raises renin and aldosterone markedly, but paradoxical aldosterone excess causes potassium retention in the cortical collecting duct.

222. At a transplant-nephrology follow-up visit, a 43-year-old woman with a kidney transplant has rising creatinine and increasing BK viral load. Biopsy shows viral cytopathic changes in tubular cells. Given this presentation, what is the core management principle?

A. Remove the transplant immediately because BK viremia cannot be controlled without nephrectomy.

B. Increase immunosuppression because BK nephropathy is caused by under-immunosuppression and is treated like acute cellular rejection.

C. Reduce net immunosuppression carefully to permit antiviral immune control while monitoring for rejection.

D. Use antibiotics targeting gram-negative bacteria because BK is a bacterial urinary pathogen.

E. Give high-dose glucocorticoids as definitive antiviral therapy because steroids directly inhibit BK viral replication in tubular cells.

223. During inpatient nephrology rounds, a 39-year-old woman with progressive CKD develops rising PTH before marked hyperphosphatemia appears. Based on these findings, which early factor contributes to secondary hyperparathyroidism?

A. Declining calcitriol availability and changes in phosphate and FGF23 signaling reduce calcium-mediated suppression of the parathyroid glands.

B. FGF23 falls early in CKD, causing unopposed renal phosphate excretion and therefore chronic hypophosphatemia as the trigger for PTH release.

C. The parathyroid glands lose calcium-sensing receptors only after dialysis begins, so PTH elevation before kidney failure is unrelated to CKD physiology.

D. Secondary hyperparathyroidism requires a parathyroid adenoma and therefore cannot develop as an adaptive response to reduced nephron function.

E. Kidney failure increases calcitriol production, raising serum calcium and directly stimulating.

224. At a metabolic stone clinic visit, a 50-year-old man with recurrent calcium phosphate stones has bicarbonate 17 mEq/L, potassium 3.1 mEq/L, and urine pH 6.4 despite systemic metabolic acidosis. Based on these findings, which disorder is most likely?

A. Distal renal tubular acidosis with impaired collecting-duct hydrogen secretion and failure to acidify the urine.

B. Proximal renal tubular acidosis, in which distal acidification remains intact once serum bicarbonate falls below the reabsorptive threshold.

C. Type 4 renal tubular acidosis, which generally produces hyperkalemia and can still lower urine pH despite reduced ammonium excretion.

D. Respiratory alkalosis, which lowers serum bicarbonate through compensation but does not explain persistently alkaline urine and nephrocalcinosis.

E. Chronic diarrhea, which causes extrarenal bicarbonate loss and should lead to increased renal ammonium excretion with acidic urine.

225. During an intensive-care nephrology consultation, a septic patient develops AKI with granular 'muddy brown' casts and renal tubular epithelial cells in the urine. Given this presentation, what lesion is most likely?

A. Postrenal obstruction, which may cause hydronephrosis but does not characteristically produce abundant tubular epithelial casts.

B. Renal-vein thrombosis, which is a vascular event and does not explain the typical tubular sediment.

C. Acute interstitial nephritis, which more often produces pyuria and white-cell casts.

D. Minimal change disease, which causes nephrotic proteinuria but not muddy granular casts from injured tubular cells.

E. Acute tubular injury from ischemic and inflammatory stress with tubular epithelial damage.

226. At a multidisciplinary renal conference, a 72-year-old man with suspected methanol ingestion has visual symptoms and severe high-anion-gap acidosis. Based on these findings, which toxic metabolite is primarily responsible for ocular and metabolic toxicity?

A. Oxalic acid, which is the principal toxic metabolite of ethylene glycol.

B. Acetone, which is produced in isopropanol metabolism and generally does not cause a high-anion-gap acidosis.

C. Lactic acid generated directly from methanol without metabolism, making alcohol dehydrogenase blockade ineffective.

D. Formic acid produced after alcohol dehydrogenase metabolism of methanol.

E. Uric acid, which causes tumor lysis nephropathy but is not the toxic metabolite of methanol.

227. During a hospital nephrology consultation, a 35-year-old woman with marked leukocytosis from leukemia has potassium 6.8 mEq/L in a serum tube, no ECG changes, and a repeat plasma potassium drawn atraumatically is 4.3 mEq/L. Given this presentation, what is the best explanation?

A. Digitalis toxicity, which inhibits sodium-potassium ATPase and would produce physiologic hyperkalemia.

B. Pseudohyperkalemia from potassium release during sample clotting or handling in the setting of extreme cell counts.

C. Type 4 renal tubular acidosis, which causes genuine plasma hyperkalemia and does not normalize with a different specimen type.

D. True tumor lysis syndrome, which would produce concordant hyperkalemia in plasma along with other biochemical evidence such as hyperphosphatemia.

E. Insulin deficiency, which causes extracellular potassium shift that should remain present in both serum and plasma samples obtained correctly.

228. During an outpatient nephrology consultation, a 46-year-old man with systemic lupus erythematosus has active urine sediment, rising creatinine, proteinuria, and low complement. Given this presentation, what is the best next diagnostic step?

A. Measure urine sodium alone because fractional sodium excretion establishes the histologic class of lupus nephritis.

B. Diagnose chronic pyelonephritis because low complement and active sediment are specific for tubulointerstitial infection in lupus.

C. Kidney biopsy to classify lupus nephritis and guide prognosis and treatment when no contraindication exists.

D. Assume minimal change disease and begin therapy without tissue because lupus serology cannot be associated with multiple renal lesions.

E. Delay evaluation until creatinine doubles again because kidney biopsy is useful only after advanced irreversible kidney failure develops.

229. At a hypertension clinic evaluation, a 28-year-old has episodic headache, palpitations, diaphoresis, and paroxysmal severe hypertension. Given this presentation, what is the preferred initial biochemical evaluation?

A. Measure serum aldosterone alone because episodic adrenergic spells are produced by mineralocorticoid excess.

B. Measure renin after intense sodium loading because catecholamine tumors are diagnosed from suppressed renin activity.

C. Perform renal angiography first because episodic sweating and palpitations localize the lesion to the renal arteries.

D. Obtain a dexamethasone suppression test only because pheochromocytoma is excluded by normal cortisol regulation.

E. Measure plasma free or urinary fractionated metanephrines to evaluate for catecholamine-secreting tumor.

230. At a hypertension clinic evaluation, a 68-year-old man has recurrent episodes of hypertension after taking a monoamine oxidase inhibitor and eating aged cheese. Given this presentation, what is the mechanism?

A. Excess catecholaminergic activity from impaired tyramine metabolism can trigger an acute hypertensive crisis.

B. Aged cheese inhibits epithelial sodium channels, producing Liddle-like sodium retention independent of sympathetic activity.

C. Monoamine oxidase inhibition prevents aldosterone synthesis, and the resulting salt wasting paradoxically produces catecholamine-independent hypertension.

D. Tyramine blocks alpha receptors and causes profound vasodilation, so the hypertension reflects compensatory renal sodium retention over months.

E. The interaction causes direct renal artery thrombosis, making abrupt renovascular hypertension the expected mechanism.

231. During an intensive-care nephrology consultation, several days after an angiographic procedure, a patient develops AKI, livedo reticularis, eosinophilia, and low complement. Based on these findings, which diagnosis best accounts for this presentation?

A. Anti-GBM disease, which causes pulmonary-renal findings and linear IgG.

B. Cholesterol crystal atheroembolic kidney disease after recent vascular instrumentation.

C. Minimal change disease, which causes nephrotic syndrome without embolic skin findings.

D. Contrast-associated tubular injury alone.

E. Renal-vein thrombosis.

232. At a metabolic stone clinic visit, a 42-year-old man with recurrent calcium oxalate stones has low urine volume and low urinary citrate. Based on these findings, which preventive strategy is most appropriate?

A. Increase sodium intake because higher sodium delivery lowers urinary calcium excretion and improves the effectiveness of citrate therapy.

B. Restrict dietary calcium severely because lower intestinal calcium always reduces oxalate absorption and prevents calcium oxalate stones.

C. Use chronic loop diuretic therapy because loop blockade lowers urinary calcium and is preferred over thiazide-type agents for stone prevention.

D. Increase fluid intake to raise urine volume and use potassium citrate when appropriate to increase urinary citrate and reduce calcium stone supersaturation.

E. Acidify the urine aggressively because lowering urine pH increases citrate excretion and reduces calcium oxalate crystallization in most patients.

233. At a hypertension clinic evaluation, a 59-year-old with hypertension has repeated office readings near 152/92 mm Hg but home readings average 126/78 mm Hg. Based on these findings, which test best clarifies whether sustained hypertension is present?

A. Use a single repeat office measurement because multiple home readings should not influence the diagnosis of sustained hypertension.

B. Stop all antihypertensive drugs for one month because treated readings cannot be interpreted with ambulatory monitoring.

C. Diagnose resistant hypertension immediately because any office value above goal proves persistent hypertension outside the clinic.

D. Use validated out-of-office blood-pressure measurement, preferably ambulatory monitoring, to assess for a white-coat effect.

E. Order plasma metanephrines first because discordant office and home readings are most commonly caused by pheochromocytoma.

234. During an outpatient nephrology consultation, a mechanically ventilated patient with massive fluid resuscitation develops tense abdominal distention, oliguria, rising airway pressures, and elevated bladder pressure. Given this presentation, what should be suspected?

A. Abdominal compartment syndrome causing reduced renal perfusion and venous outflow from markedly increased intra-abdominal pressure.

B. Renal papillary necrosis, because papillary sloughing produces a rigid abdomen and elevated plateau pressures.

C. Acute glomerulonephritis, because glomerular inflammation is the usual cause of increased airway pressure after fluid resuscitation.

D. Nephrogenic diabetes insipidus, because elevated abdominal pressure causes polyuria before renal perfusion falls.

E. SIADH, because high bladder pressure directly stimulates vasopressin and produces oliguria with a tense abdomen.

235. During inpatient nephrology rounds, a 75-year-old woman with CKD and heavy albuminuria needs antihypertensive therapy. Based on these findings, which drug class has a specific kidney-protective role when tolerated?

A. A direct alpha blocker as monotherapy because alpha blockade has the strongest evidence for reducing glomerular protein leak independent of pressure.

B. Short-acting nifedipine used intermittently because rapid pressure swings protect glomerular capillaries from chronic injury.

C. Chronic NSAID therapy because afferent vasoconstriction reduces albuminuria while preserving long-term kidney function.

D. An ACE inhibitor or angiotensin receptor blocker because lowering intraglomerular pressure and albuminuria can slow progression.

E. Dual ACE inhibitor plus ARB therapy because complete renin-angiotensin blockade improves outcomes without added kidney or potassium risk.

236. At a kidney clinic follow-up visit, two patients have eGFR 45 mL/min/1.73 m2, but one has urine albumin-creatinine ratio 900 mg/g and the other 20 mg/g. Based on these findings, which patient is at higher risk of progression?

A. The patient with low albuminuria because lower urinary protein proves greater glomerular scarring and faster nephron loss at any fixed eGFR.

B. Their risks are identical because CKD prognosis is determined entirely by GFR category and not by urinary albumin excretion.

C. Risk cannot be compared unless both patients are receiving dialysis because albuminuria has prognostic meaning only after kidney failure.

D. The patient with severe albuminuria because albuminuria powerfully stratifies kidney and cardiovascular risk even at the same eGFR.

E. Neither patient is at meaningful risk because an eGFR above 30 mL/min/1.73 m2 excludes progressive chronic kidney disease.

237. During an urgent nephrology assessment, a muscular bodybuilder and a frail older adult have the same serum creatinine. Why can cystatin C be useful in selected patients?

A. Cystatin C can improve GFR estimation when creatinine is misleading because creatinine generation is strongly influenced by muscle mass.

B. Cystatin C directly measures renal blood flow, so it is unaffected by inflammation, corticosteroids, thyroid disease, or other non-GFR factors.

C. Cystatin C should replace creatinine in every patient because combining filtration markers never improves GFR estimation accuracy.

D. Cystatin C is useful only in dialysis patients.

E. Cystatin C is secreted only by the proximal tubule and therefore rises exclusively in tubular disease.

238. During a dialysis-unit assessment, a hemodialysis patient repeatedly becomes hypotensive near the end of treatment after large interdialytic weight gains. Based on these findings, which strategy most directly addresses the problem?

A. Give routine large saline boluses before every treatment without changing fluid goals because chronic volume expansion prevents future hypotension safely.

B. Increase the ultrafiltration rate further so the treatment finishes earlier and the circulation has less cumulative exposure to dialysis.

C. Raise dialysate temperature substantially because warming improves vascular tone and reduces intradialytic vasodilation during rapid fluid removal.

D. Reduce excessive ultrafiltration stress by reassessing target weight, treatment time, sodium intake, and the required fluid-removal rate.

E. Stop all antihypertensive therapy permanently because blood-pressure medications are the sole cause of intradialytic hypotension in volume-overloaded patients.

239. During a hospital nephrology consultation, a young man has persistent microscopic hematuria, progressive kidney disease, sensorineural hearing loss, and ocular abnormalities. Based on these findings, which diagnosis is most likely?

A. Alport syndrome from a type IV collagen basement-membrane disorder with renal and extrarenal manifestations.

B. Minimal change disease, which causes nephrotic proteinuria and has no collagen-related hearing manifestations.

C. Acute interstitial nephritis, which is acquired and does not produce inherited hearing and ocular abnormalities.

D. Thin basement membrane nephropathy alone, which usually lacks progressive kidney failure, hearing loss, and characteristic ocular disease.

E. Renal papillary necrosis, which may cause hematuria but does not cause a hereditary sensorineural hearing syndrome.

240. At a transplant-nephrology follow-up visit, why are avoidable red-cell transfusions particularly undesirable in a kidney-transplant candidate with CKD anemia?

A. Blood transfusion eliminates the possibility of ABO-compatible transplantation because the recipient's inherited blood group changes after donor exposure.

B. Transfusions permanently suppress endogenous erythropoietin production, so the patient can never respond to an ESA after receiving donor red cells.

C. Transfusions can expose the patient to HLA antigens and increase sensitization, potentially making compatible transplantation more difficult.

D. Transfused red cells are rapidly filtered into urine in CKD, causing iron nephrotoxicity and accelerating progression to kidney failure.

E. Transfusions always cause acute antibody-mediated rejection even before transplantation because donor erythrocytes contain a functioning kidney graft.

Answers

1. D. Cyst infection is an important complication of ADPKD and may be difficult to diagnose because infected cysts may not communicate freely with the urinary tract. Persistent fever and focal pain should prompt targeted imaging and appropriate antimicrobial selection.

2. A. Indirect sodium methods assume a normal plasma-water fraction and can read falsely low when lipids or proteins occupy an unusually large fraction of plasma volume. Normal measured osmolality and a normal direct sodium establish pseudohyponatremia.

3. E. KDIGO classification incorporates both GFR and albuminuria because each independently predicts adverse kidney and cardiovascular outcomes. A patient with substantial albuminuria is at higher risk than a patient with the same eGFR and little or no albuminuria.

4. B. Hypertensive emergency is defined by severe blood-pressure elevation with acute target-organ injury, not by a number alone. Management uses titratable therapy and careful reduction to limit ongoing injury while avoiding ischemia from excessive rapid lowering.

5. E. C3 glomerulopathy is characterized by dominant C3 deposition and often reflects alternative-pathway dysregulation. Persistent hypocomplementemia supports ongoing complement activation and helps distinguish it from transient postinfectious changes.

6. C. A reset osmostat preserves normal osmoregulation but around a lower threshold for vasopressin release and thirst. Patients can dilute and concentrate appropriately, yet the sodium is defended at a lower steady state.

7. A. Distal RTA causes an inability to acidify urine appropriately during systemic acidosis. Chronic alkaline urine, hypocitraturia, and hypercalciuria predispose to calcium phosphate stones and nephrocalcinosis.

8. D. CRRT is commonly selected for hemodynamically unstable patients because fluid and solute shifts are slower and continuous. Modality choice also depends on local expertise, neurologic status, toxin characteristics, and treatment goals.

9. C. Modern CKD care emphasizes absolute risk, not a single creatinine threshold. GFR, albuminuria, progression rate, complications, and validated kidney-failure risk models can help identify patients who need earlier specialist care and preparation.

10. D. SGLT2 inhibition increases distal sodium delivery and restores tubuloglomerular feedback, reducing glomerular hyperfiltration. A small early eGFR dip may occur, followed by slower chronic decline in appropriate patients.

11. A. Kidney replacement therapy is started for clinical indications such as refractory volume overload, hyperkalemia, acidosis, pericarditis, encephalopathy, malnutrition, or other uremic manifestations. A low eGFR is important for planning but is not by itself an absolute start trigger.

12. E. Medication dosing depends on the kidney-function metric used in the drug's evidence and labeling, and large errors matter most for narrow-therapeutic-index drugs. When creatinine is clearly unreliable, cystatin C or measured clearance may improve clinical decision-making.

13. A. Lithium enters principal cells through epithelial sodium channels and interferes with vasopressin signaling. Amiloride reduces lithium uptake and is a favored treatment for lithium-associated nephrogenic diabetes insipidus when the drug cannot simply be discontinued.

14. C. Insulin rapidly lowers extracellular potassium by driving it into cells. Because total-body potassium is unchanged, definitive removal or correction of the underlying cause is still required when potassium burden remains high.

15. C. Kt/V is a widely used measure of hemodialysis small-solute dose, incorporating dialyzer urea clearance, treatment time, and the patient's urea distribution volume. Repeatedly shortened sessions predictably reduce delivered clearance.

16. B. Hypertension with hypokalemia and suppressed renin is a classic setting for primary aldosteronism. Screening uses aldosterone and renin, followed by confirmatory and subtype evaluation when appropriate.

17. E. Preemptive transplantation can avoid dialysis-related morbidity and is associated with favorable outcomes in appropriate candidates. Evaluation should begin early enough to allow donor assessment, cardiovascular risk assessment, and logistical planning.

18. C. Chronic heavy analgesic exposure has been associated with papillary necrosis and chronic interstitial nephritis. The urine sediment may be relatively nonspecific, and imaging can show small irregular kidneys or papillary calcification.

19. C. Chronic antibody-mediated injury can produce transplant glomerulopathy, proteinuria, and progressive graft dysfunction. Donor-specific antibodies support an alloimmune mechanism, although biopsy is essential to characterize the lesion.

20. B. Donor-positive/recipient-negative pairing carries the highest risk of primary CMV infection because the recipient lacks prior immunity and receives latent virus with the graft. Prevention strategies depend on serostatus and immunosuppressive intensity.

21. A. Absolute iron deficiency is a common and treatable contributor to CKD anemia. Repletion strategy depends on dialysis status, severity, prior response, tolerance, and the need for rapid correction, but the underlying deficiency should not be ignored.

22. C. PTLD ranges from polyclonal proliferation to aggressive lymphoma and is associated with the intensity of immunosuppression and EBV risk. Treatment commonly begins with reduction of immunosuppression when feasible and may require rituximab or lymphoma-directed therapy.

23. B. Collapsing FSGS is a severe podocytopathy classically associated with HIV and certain genetic risk backgrounds. It commonly presents with heavy proteinuria and rapid loss of kidney function.

24. C. Chronic hypernatremia is accompanied by cerebral osmotic adaptation, so overly rapid correction can cause cerebral edema. Treatment replaces the calculated and ongoing water deficit gradually while monitoring the sodium trajectory.

25. C. Renal infarction can result from embolism, thrombosis, dissection, or vascular injury and may cause flank pain, hematuria, hypertension, and elevated LDH. Early recognition matters because selected patients may benefit from revascularization or anticoagulation depending on the mechanism and timing.

26. B. Nephrotic syndrome increases thrombosis risk through urinary loss and altered synthesis of anticoagulant and procoagulant proteins. Renal-vein thrombosis is a classic complication, particularly in some membranous nephropathy patients.

27. D. Advance planning supports informed choice and reduces urgent catheter-dependent dialysis starts. Evaluation for preemptive transplantation, dialysis modality selection, access planning, and conservative kidney management should be individualized and begun before kidney failure becomes an emergency.

28. C. Glucocorticoids contribute to hyperglycemia, weight gain, bone loss, infection risk, cataracts, and skin changes. Immunosuppression is individualized to maintain graft protection while limiting cumulative toxicity.

29. E. Severe refractory acidosis is an accepted indication for dialysis or continuous kidney replacement therapy. Treatment also targets the underlying cause, perfusion, ventilation, and toxin exposure when relevant.

30. E. Cyclooxygenase inhibition reduces vasodilatory prostaglandins, can lower renal blood flow, and favors sodium and water retention. NSAIDs can therefore raise blood pressure and antagonize diuretics, ACE inhibitors, ARBs, and other therapies.

31. A. Liddle syndrome mimics mineralocorticoid excess but aldosterone is suppressed because ENaC is constitutively active. The resulting sodium retention, hypertension, distal potassium secretion, and alkalosis distinguish it from salt-wasting tubulopathies.

32. A. Urinary tract infection is common after kidney transplantation and can present with systemic illness, graft dysfunction, or relatively subtle urinary symptoms. Culture-guided treatment and evaluation for obstruction or recurrent infection are important when indicated.

33. D. Encapsulating peritoneal sclerosis is an uncommon but serious fibrosing process associated with long PD exposure and recurrent inflammation. Progressive obstruction, ultrafiltration failure, and characteristic imaging findings are key clues.

34. D. Apparent treatment-resistant hypertension can result from poor technique, white-coat effect, undertreatment, interfering substances, or nonadherence. These factors should be addressed before extensive secondary-cause testing or additional medications are pursued.

35. C. Microangiopathic hemolytic anemia, thrombocytopenia, and organ injury are hallmarks of thrombotic microangiopathy. The cause can include TTP, complement-mediated HUS, Shiga toxin, severe hypertension, pregnancy-related syndromes, drugs, and other triggers.

36. E. ECG changes from severe hyperkalemia require immediate membrane stabilization with intravenous calcium. Insulin with glucose, beta-agonists, bicarbonate in selected acidemic patients, diuresis, binders, or dialysis are then used to shift or remove potassium.

37. A. As kidney function declines, phosphate retention, increased FGF23, reduced calcitriol availability, and altered calcium balance stimulate secondary hyperparathyroidism. CKD-MBD is therefore a systemic disorder involving mineral metabolism, bone, and vascular calcification risk.

38. A. Lymphoceles are common postoperative fluid collections and can compress the ureter, bladder, or iliac vessels when large. Symptomatic collections may require drainage or surgical management.

39. E. Calcineurin inhibitors can cause dose-related renal vasoconstriction and acute functional decline in GFR. Chronic exposure can also contribute to vascular and tubulointerstitial injury.

40. A. Ultrasound is a practical first test for obstruction in many patients with unexplained AKI. Early obstruction may occasionally lack marked dilation, so clinical context still matters.

41. B. ADTKD encompasses inherited disorders with slowly progressive tubulointerstitial fibrosis and relatively bland urine findings. UMOD-associated disease often features hyperuricemia and gout before kidney failure.

42. E. Dysmorphic red cells, especially acanthocytes, support glomerular hematuria. Red-cell casts provide even stronger evidence that bleeding originates within the nephron.

43. B. Cortisol normally restrains vasopressin release, so deficiency can cause inappropriate water retention. In primary adrenal failure, aldosterone deficiency also promotes sodium loss and hyperkalemia, making the clinical picture distinct from isolated SIADH.

44. D. Hydronephrosis with low urine output points toward mechanical obstruction. Post-transplant causes include ureteral edema, stricture, kinking, stones, or external compression.

45. A. Prolonged severe hypoperfusion can cause structural tubular injury after an initial prerenal phase. Recovery may include a diuretic phase as tubular function improves.

46. A. Urea handling may be less immediately altered by loop diuretics than sodium handling, so FEurea can sometimes support the assessment. Like all fractional excretion indices, it is imperfect and must be integrated with the clinical picture.

47. C. Microangiopathic hemolysis plus thrombocytopenia and organ injury is a TMA until proven otherwise. Rapid testing and treatment can be critical, especially when TTP is possible.

48. A. Ethylene glycol is metabolized to toxic organic acids, including oxalate-generating metabolites. Fomepizole blocks further metabolism, and hemodialysis removes parent alcohol and toxic metabolites while correcting severe acidosis.

49. A. Risk mitigation includes avoiding unnecessary contrast load and nephrotoxins and maintaining appropriate intravascular volume when safe. Prophylactic dialysis and forced diuresis are not routine preventive strategies.

50. C. MGRS describes kidney disease caused by a monoclonal immunoglobulin from a clone that may be too small to meet malignancy criteria. Because the protein itself can progressively damage the kidney, clone-directed therapy may be appropriate after defining the lesion.

51. A. The amount of water that can be excreted depends on both the minimum achievable urine osmolality and the amount of osmole available for excretion. With extremely low solute intake, even maximally dilute urine may be insufficient to excrete a large water load.

52. A. ADPKD causes progressive bilateral renal cyst formation, hypertension, and enlarged kidneys. Important extrarenal associations include hepatic cysts and intracranial aneurysms, especially when there is a relevant family history or other risk indication for screening.

53. C. A distended bladder with anuria suggests lower urinary tract obstruction and can often be confirmed and relieved quickly with catheterization. Persistent obstruction above the bladder may require imaging and urologic intervention.

54. D. During true potassium depletion, healthy kidneys should reduce urinary potassium excretion. Continued high urine potassium therefore points to renal losses from diuretics, mineralocorticoid activity, tubular disorders, or other renal mechanisms.

55. C. Mycophenolate exposure is associated with pregnancy loss and congenital malformations and requires preconception planning. Successful pregnancy is possible in selected stable transplant recipients with coordinated transplant and maternal-fetal care.

56. D. Dialysis disequilibrium is most likely during initial treatment of severe azotemia when urea is removed from plasma faster than cerebral osmoles equilibrate. Prevention uses gentler initial dialysis with slower solute removal in high-risk patients.

57. E. Transplant renal artery stenosis can present with resistant hypertension, declining graft function, and sometimes a bruit. Doppler imaging and further vascular evaluation are used when suspicion is high.

58. D. Lithium is a small ion with low protein binding and is dialyzable. Severe neurologic manifestations and impaired kidney clearance are major considerations, and post-dialysis rebound can occur as lithium redistributes from tissues.

59. E. HRS-AKI is a functional renal failure syndrome driven by advanced splanchnic vasodilation and renal vasoconstriction. Diagnosis requires exclusion of shock, structural kidney disease, nephrotoxins, and other reversible causes.

60. E. For appropriately selected patients, kidney transplantation generally provides better survival and quality of life than remaining on dialysis. Preemptive transplantation can avoid many complications of dialysis and is an important planning goal when possible.

61. A. CKD-MBD is dynamic, and treatment decisions should integrate serial phosphate, calcium, PTH, alkaline phosphatase, and clinical context. Overcorrection can be harmful, particularly if PTH is suppressed excessively and adynamic bone disease develops.

62. D. Acute interstitial nephritis is commonly drug related and may present with AKI, pyuria, white-cell casts, rash, fever, or eosinophilia, although the full triad is uncommon. Removing the offending agent is central to management.

63. C. Delayed graft function is common after deceased-donor transplantation and often reflects ischemic acute tubular injury. It increases subsequent risks but is not synonymous with irreversible primary nonfunction.

64. B. RAS blockade can be kidney- and cardiovascular-protective, particularly with albuminuria. Mild or moderate hyperkalemia should prompt identification of reversible contributors and appropriate potassium management before reflexive permanent discontinuation when the drug remains indicated.

65. A. CKD-associated pruritus is multifactorial and requires exclusion of other causes. Management combines adequate dialysis and skin care with targeted therapies for persistent symptoms; gabapentinoids and newer kappa-opioid receptor approaches have evidence in selected patients.

66. E. When urine is very concentrated in SIADH, sodium from isotonic saline can be excreted while water is retained, a phenomenon sometimes called desalination. Thus normal saline may fail to correct and can even worsen hyponatremia in strongly antidiuretic states.

67. E. Mycophenolate commonly causes diarrhea and cytopenias through antiproliferative effects. Management balances toxicity against rejection risk and includes evaluation for infectious causes of diarrhea or leukopenia.

68. D. Glycyrrhetinic compounds in licorice inhibit renal conversion of cortisol to cortisone. Cortisol then activates mineralocorticoid receptors, producing sodium retention, hypokalemic alkalosis, hypertension, and suppression of both renin and aldosterone.

69. D. Severe ADAMTS13 deficiency supports TTP, usually from an acquired autoantibody in adults. Rapid recognition matters because plasma exchange and immunosuppressive therapy can be lifesaving.

70. B. In a simple high-gap acidosis, the rise in anion gap and fall in bicarbonate are often of similar magnitude. A much smaller bicarbonate fall suggests an offsetting metabolic alkalosis, such as vomiting accompanying ketoacidosis.

71. A. Thin basement membrane nephropathy often presents as persistent microscopic hematuria with preserved kidney function. Some collagen IV variants can carry greater long-term risk, so significant proteinuria or declining GFR should prompt reassessment.

72. E. Volume depletion triggers vasopressin despite hypotonicity, explaining the concentrated urine. The low urine sodium and clinical signs of depletion indicate intact renal sodium conservation rather than SIADH.

73. B. Uremic encephalopathy can present with cognitive change, asterixis, myoclonus, seizures, or progressive obtundation. When attributable to kidney failure, it is a major indication for dialysis.

74. B. Graft dysfunction has a broad differential including rejection, drug toxicity, infection, recurrent disease, and ischemic injury. Biopsy provides direct evidence of the renal lesion and guides immunosuppressive treatment.

75. E. Hyperphosphatemia management combines control of intake, removal by dialysis, and intestinal phosphate binders when needed. Binder choice should account for calcium loading, iron status, medication interactions, pill burden, and patient preference.

76. A. Trimethoprim has an amiloride-like effect on ENaC and can reduce distal potassium secretion. Risk is greater with CKD, RAS blockade, mineralocorticoid antagonists, or other potassium-raising factors.

77. D. Drug withdrawal is the key initial intervention in suspected AIN. The diagnosis may require biopsy when uncertain, and corticosteroids are considered in selected cases after weighing severity, timing, and alternative diagnoses.

78. B. When kidney perfusion depends on angiotensin II to maintain efferent tone, blocking the renin-angiotensin system can cause a substantial fall in filtration pressure. A large creatinine rise should prompt evaluation for volume depletion, NSAID exposure, or bilateral/solitary-kidney renovascular disease.

79. D. Antibody-mediated rejection is supported by evidence of graft microvascular injury, circulating donor-specific antibody, and antibody-endothelial interaction such as complement deposition in appropriate contexts. The diagnosis is integrated with current pathologic criteria.

80. B. Tumor lysis releases potassium, phosphate, and nucleic-acid metabolites, producing hyperkalemia, hyperphosphatemia, secondary hypocalcemia, and uric acid burden. Prevention and treatment include hydration, urate-lowering strategies, frequent laboratory monitoring, and dialysis when severe complications are refractory.

81. D. Hexagonal crystals are highly characteristic of cystine. Cystinuria is an inherited defect in proximal reabsorption of cystine and dibasic amino acids and often causes recurrent stones beginning at a young age.

82. D. Liddle syndrome causes constitutive ENaC activity and avid distal sodium reabsorption. Volume expansion suppresses renin and aldosterone, distinguishing it from primary aldosteronism; amiloride or triamterene targets the defect.

83. C. Calcium-based binders can contribute to positive calcium balance, particularly when used heavily. In a patient with hypercalcemia or substantial calcification burden, limiting extra calcium exposure can be a rational part of binder selection.

84. D. A negative urine anion gap is a surrogate for substantial urinary ammonium chloride excretion. In hyperchloremic acidosis, that response indicates intact renal acid excretion and favors gastrointestinal bicarbonate loss.

85. B. Uremic pericarditis is a serious complication of advanced kidney failure and supports urgent dialysis. Anticoagulation and ultrafiltration strategies may require special attention if a significant pericardial effusion is present.

86. D. Hypomagnesemia disinhibits renal outer medullary potassium channels and favors ongoing distal potassium secretion. Refractory hypokalemia therefore often requires magnesium repletion in addition to potassium replacement.

87. E. Vomiting produces chloride depletion, volume contraction, and often hypokalemia, leading to a chloride-responsive metabolic alkalosis. A low urine chloride supports treatment with saline and potassium rather than further diuresis.

88. A. Rapid reduction of plasma urea and osmolality can promote water movement into the brain in vulnerable patients. Slower therapy allows tighter control of tonicity and volume when intracranial pressure is a major concern.

89. C. Thiazides impair dilution in the distal nephron and can produce severe hypotonic hyponatremia, especially in older adults. The concentrated urine and urine sodium above the expected low-volume range fit persistent renal sodium loss rather than polydipsia or a nonhypotonic process.

90. B. Fanconi syndrome is a generalized proximal reabsorptive defect, so glucose, phosphate, uric acid, amino acids, and bicarbonate can all be lost in urine. Monoclonal light chains are an important acquired cause in adults.

91. E. Obstruction raises hydrostatic pressure proximal to the blockage and can reduce GFR. Bilateral obstruction or obstruction of a solitary functioning kidney can cause severe AKI and requires prompt decompression.

92. A. Cardiorenal physiology includes both forward-flow and backward-pressure effects. Venous congestion can impair renal perfusion gradients, raise interstitial pressure, and contribute to worsening filtration.

93. E. Lithium can enter principal cells via ENaC and disrupt aquaporin-2 regulation and collecting-duct responsiveness to vasopressin. Amiloride can reduce lithium entry when therapy must continue.

94. A. Immunization should be optimized before transplant when possible. Inactivated vaccines are generally usable after transplantation, while live attenuated vaccines are usually contraindicated during significant immunosuppression.

95. E. Complement-mediated HUS reflects uncontrolled alternative-pathway activity due to genetic or acquired abnormalities. Complement inhibition can be central to treatment after other major TMA causes are assessed.

96. D. Mixed cryoglobulinemia is strongly associated with chronic hepatitis C and can cause systemic small-vessel vasculitis. Kidney involvement often shows immune-complex MPGN with low complement, especially C4.

97. A. Rapid cell breakdown releases phosphate, which complexes with calcium and can precipitate in tissues and kidneys. The resulting fall in ionized calcium may cause neuromuscular or cardiac symptoms, especially when kidney function is impaired.

98. D. Nephrotic syndrome is defined by heavy proteinuria and is accompanied by hypoalbuminemia, edema, and often hyperlipidemia. The syndrome also increases thrombotic and infectious risk depending on severity and cause.

99. E. Calcimimetics increase the sensitivity of the parathyroid calcium-sensing receptor and lower PTH, often lowering calcium as well. They are useful when secondary hyperparathyroidism persists and hypercalcemia limits other therapies.

100. E. Metformin is dialyzable, and severe lactic acidosis with shock or kidney failure may require extracorporeal support. Management also addresses hypoperfusion, sepsis, hypoxemia, and other contributors to lactate accumulation.

101. D. Treating chronic metabolic acidosis can be beneficial, but sodium-containing alkali may worsen edema or hypertension in some patients. Dose and formulation should therefore be individualized and monitored alongside volume status and serum bicarbonate.

102. C. Nonadherence is a major modifiable risk for rejection and graft loss and often reflects complex barriers. Effective care identifies the cause, supports access, monitors graft status, and uses collaborative strategies to improve long-term adherence.

103. D. Chronic respiratory alkalosis is compensated by decreased renal bicarbonate reabsorption and reduced net acid excretion, producing a larger fall in serum bicarbonate than occurs acutely. This renal adaptation partially normalizes pH but does not correct the primary hypocapnia.

104. D. Hyperacute rejection occurs when preformed antibodies bind donor endothelium immediately after reperfusion, activating complement and thrombosis. Modern crossmatching has made this event uncommon.

105. B. SGLT2 inhibitors reduce kidney-disease progression and cardiovascular events across important CKD populations, including many patients without diabetes. They are added to foundational blood-pressure and renin-angiotensin system management when appropriate.

106. C. Tacrolimus has a narrow therapeutic index and is metabolized through CYP3A pathways. Potent inhibitors such as azole antifungals can raise levels substantially and precipitate neurotoxicity, hyperkalemia, and nephrotoxicity.

107. A. Kidney replacement therapy in AKI is initiated for refractory electrolyte, acid-base, volume, toxin, and uremic complications rather than a single creatinine value. Close monitoring is essential because the need can change quickly.

108. D. Markedly dilute urine despite hypernatremia indicates impaired antidiuretic action. A strong rise in urine osmolality after desmopressin demonstrates preserved renal responsiveness and therefore supports central rather than nephrogenic diabetes insipidus.

109. C. Congo-red positivity with apple-green birefringence is characteristic of amyloid. Renal involvement commonly produces heavy proteinuria, and systemic manifestations depend on the underlying amyloid type.

110. A. TMP-SMX is commonly used for Pneumocystis prophylaxis and also provides protection against several other opportunistic pathogens. Alternative regimens are used when allergy, cytopenia, or other toxicity prevents its use.

111. E. More complete renin-angiotensin blockade lowers albuminuria but also increases adverse events such as hyperkalemia, hypotension, and AKI. Clinical outcome trials have not supported routine ACE inhibitor plus ARB therapy for CKD protection.

112. A. A functioning AV access can divert arterial blood from the distal limb, particularly in patients with vascular disease. Pain, coolness, neurologic symptoms, or tissue threat distal to the access suggest steal and require vascular evaluation.

113. A. De novo donor-specific antibodies are associated with increased risk of antibody-mediated rejection and graft loss. Their significance depends on the clinical setting, antibody characteristics, adherence, graft function, and biopsy findings.

114. E. ECG changes from severe hyperkalemia require immediate membrane stabilization with intravenous calcium. Insulin with glucose, beta-agonist therapy, bicarbonate in selected acidemic patients, diuretics if appropriate, potassium binders, and dialysis address serum potassium by shifting or removal.

115. C. Central venous dialysis catheters carry substantial bloodstream infection risk. Fever or rigors during dialysis with positive cultures requires prompt treatment and a decision about catheter removal or exchange based on organism, severity, and complications.

116. B. Once contrast-associated or multifactorial acute tubular injury has occurred, treatment is primarily supportive. Recovery depends on avoiding further insults and managing fluid, electrolyte, acid-base, and uremic complications.

117. D. Uric acid solubility rises sharply as urine pH increases, so urinary alkalinization is central to both dissolution and prevention. Low urine volume and hyperuricosuria can also contribute, but acidic urine is the dominant risk factor in many patients.

118. A. Uremic pericarditis is a classic urgent indication for dialysis, and patients also require assessment for effusion and tamponade. ECG findings may be less stereotyped than in idiopathic inflammatory pericarditis.

119. C. FENa reflects tubular sodium handling and is influenced by diuretics, CKD, sepsis, glomerular disease, and other factors. It should be interpreted with the history, sediment, hemodynamics, and sometimes alternative indices such as fractional urea excretion.

120. A. Albuminuria and GFR are complementary markers. A large change in albumin excretion may reflect active glomerular injury, treatment failure, or a new process and can materially change risk even before eGFR changes substantially.

121. D. Sepsis-associated AKI is multifactorial and cannot be reduced to simple global renal ischemia. Even with adequate systemic pressure, microcirculatory dysfunction and cellular stress may persist.

122. A. The defense against hypernatremia requires both renal water conservation and thirst-driven water intake. Severe hypernatremia often occurs when a concentrating defect is combined with impaired consciousness, impaired thirst, or lack of access to water.

123. E. Calciphylaxis is a high-mortality syndrome of painful ischemic skin lesions in advanced CKD, associated with diabetes, obesity, disordered mineral metabolism, and warfarin exposure among other risks. Management is multidisciplinary and includes wound care, pain control, correction of mineral abnormalities, and often sodium thiosulfate.

124. D. A substantial arm-leg pressure discrepancy with weak or delayed femoral pulses suggests aortic coarctation. Adult presentations may include resistant hypertension, headaches, murmurs, and collateral vessels.

125. D. In the absence of acute target-organ injury, severe hypertension usually does not require rapid intravenous lowering. Measurement should be confirmed, adherence and contributing factors assessed, and chronic therapy adjusted with timely follow-up.

126. A. Creatinine is a product of muscle metabolism, so a low creatinine may reflect reduced production rather than excellent filtration. In frail or sarcopenic patients, alternative or combined filtration markers can provide a more realistic estimate of GFR.

127. B. Both SIADH and cerebral salt wasting can show high urine sodium and concentrated urine after brain injury. Objective volume depletion and ongoing negative sodium balance favor cerebral salt wasting.

128. A. Exercise-associated hyponatremia usually reflects excess hypotonic intake plus persistent vasopressin. Neurologic symptoms indicate cerebral edema risk and warrant hypertonic saline rather than further free water.

129. B. Severe salicylate poisoning can deteriorate rapidly as acidemia increases tissue penetration, including into the CNS. Hemodialysis provides efficient toxin removal and correction of acid-base, electrolyte, and fluid abnormalities.

130. E. Diarrhea removes bicarbonate from the body and typically causes a hyperchloremic normal-gap acidosis. If the kidneys are intact, ammonium excretion rises appropriately, helping distinguish extrarenal bicarbonate loss from impaired renal acidification.

131. E. Insulin lowers serum potassium rapidly by driving potassium into cells through stimulation of sodium-potassium ATPase. This is redistribution rather than elimination, so definitive potassium removal may still be required.

132. A. Aminoglycosides accumulate in proximal tubular cells and can cause dose- and exposure-related acute tubular injury. Toxicity is often nonoliguric and may be accompanied by electrolyte wasting during recovery.

133. B. Severe hyperglycemia raises extracellular tonicity, moving water out of cells and lowering the measured sodium concentration. This is true translocational hyponatremia rather than SIADH or pseudohyponatremia.

134. A. Reduced kidney clearance can substantially increase exposure to renally eliminated medications. Dose adjustment should reflect the best available kidney-function estimate, the drug's therapeutic window, changing AKI trajectory, and dialysis clearance when relevant.

135. C. Minimal change disease is the most common cause of nephrotic syndrome in children and often follows an immune trigger. Light microscopy can appear nearly normal, while electron microscopy shows diffuse foot-process effacement.

136. C. Severe secondary hyperparathyroidism accelerates bone resorption and formation, producing high-turnover osteitis fibrosa. Markedly elevated PTH, high alkaline phosphatase, and characteristic skeletal changes support this diagnosis.

137. A. Metformin-associated lactic acidosis is uncommon but can occur when drug accumulation coincides with severe illness, hypoperfusion, hypoxemia, or advanced kidney dysfunction. In severe toxic presentations, supportive care and extracorporeal removal may be considered.

138. D. SGLT2 inhibition alters tubuloglomerular feedback and can produce a modest initial reduction in eGFR. In a stable patient this hemodynamic effect is expected and often precedes long-term kidney protection, though larger declines require evaluation for volume depletion or other causes.

139. B. Uremia impairs platelet adhesion and aggregation despite a normal platelet count and routine coagulation tests. Desmopressin can produce a rapid but temporary improvement, while dialysis and management of anemia address underlying contributors.

140. D. Toluene metabolism produces hippurate and can generate a complex acid-base picture, often with substantial renal potassium loss and a hyperchloremic acidosis after urinary excretion of organic anions with cations. The associated hypokalemia is an important clue.

141. A. Winter's formula predicts an expected PaCO2 of about 1.5 x 8 + 8, or roughly 20 mm Hg. The measured PaCO2 is therefore appropriate compensation for the metabolic acidosis rather than evidence of an additional respiratory disorder.

142. D. Several primary glomerular diseases, including IgA nephropathy, can recur after kidney transplantation. Recurrence may be clinically silent, cause hematuria or proteinuria, or contribute to graft dysfunction over time.

143. B. Fibromuscular dysplasia often affects younger women and commonly involves the mid-to-distal renal artery. Medial fibroplasia produces the classic string-of-beads appearance and can cause renovascular hypertension.

144. D. Membranoproliferative glomerulonephritis is best viewed as a pattern characterized by mesangial and capillary-wall remodeling. Determining whether immune complexes, monoclonal proteins, infection, autoimmune disease, or complement dysregulation are driving the pattern is essential.

145. B. Poststreptococcal glomerulonephritis follows a latent period after nephritogenic infection and commonly causes low C3. Most children recover, although kidney function and volume status require monitoring during the acute phase.

146. C. Fabry disease is an X-linked lysosomal storage disorder that can affect kidneys, heart, peripheral nerves, skin, and brain. Proteinuria and progressive CKD are common renal manifestations.

147. C. Adynamic bone disease is characterized by very low bone turnover and may occur when PTH is over-suppressed, particularly with high calcium exposure. Avoiding excessive calcium and reconsidering PTH-suppressing therapy are important management steps.

148. D. Finerenone has demonstrated kidney and cardiovascular benefit in selected patients with type 2 diabetes and albuminuric CKD already receiving renin-angiotensin system blockade. Hyperkalemia risk requires attention to baseline and follow-up potassium.

149. D. Dialysis is not obligatory when its burdens outweigh likely benefits according to an informed patient's goals. Conservative kidney management actively treats symptoms and complications while avoiding dialysis, and it should be discussed as a legitimate option in appropriate patients.

150. A. The combination of RAS blockade, a diuretic, and an NSAID can sharply reduce filtration pressure in susceptible patients. NSAIDs remove prostaglandin-mediated afferent support while ACE inhibition reduces efferent tone and diuretics may lower effective circulating volume.

151. E. Anti-GBM antibodies bind a basement-membrane collagen epitope and produce linear IgG staining. Pulmonary involvement occurs because the target antigen is also present in alveolar basement membranes.

152. B. Sustained hypercapnia leads the kidneys to increase bicarbonate reabsorption and net acid excretion over several days. The degree of bicarbonate elevation here is consistent with chronic rather than purely acute respiratory acidosis.

153. D. Restless legs syndrome is defined by an urge to move the legs with unpleasant sensations that begin or worsen at rest, improve with movement, and are worse in the evening. Iron deficiency and uremia are important contributors in CKD.

154. D. Long-term exposure can alter the peritoneal membrane, increasing small-solute transport in some patients. Rapid glucose absorption then erodes the osmotic gradient and can reduce net ultrafiltration despite adequate catheter function.

155. E. Citrate binds ionized calcium in the circuit and is normally metabolized to bicarbonate. In severe liver dysfunction or shock with impaired metabolism, citrate can accumulate, producing a rising total-to-ionized calcium ratio and escalating calcium requirements.

156. C. PLA2R is the major target antigen in many cases of primary membranous nephropathy. Antibody levels can support diagnosis and provide information about immunologic activity in the appropriate clinical context.

157. B. Cirrhosis reduces effective arterial filling because of systemic and splanchnic vasodilation. Baroreceptor-mediated vasopressin release then promotes disproportionate water retention, producing hypervolemic hypotonic hyponatremia.

158. D. Seizure from severe hyponatremia is an emergency requiring hypertonic saline to raise sodium promptly by a limited amount and reduce cerebral edema. The goal is symptom control, not normalization, because excessive correction increases the risk of osmotic demyelination.

159. B. ACE inhibitors and ARBs reduce intraglomerular pressure and albuminuria and are foundational therapy in proteinuric CKD when tolerated. Monitoring creatinine and potassium is important after initiation or dose escalation.

160. A. Obstructive sleep apnea is common in resistant hypertension and promotes sympathetic activation, intermittent hypoxemia, and neurohormonal changes. Evaluation and treatment are part of comprehensive blood-pressure management.

161. C. CKD requires abnormalities of kidney structure or function present for at least three months with implications for health. A single low eGFR can reflect acute illness, biologic variation, or measurement error and does not establish chronicity by itself.

162. A. Abrupt or resistant hypertension with asymmetric kidneys and recurrent flash pulmonary edema should raise suspicion for renovascular disease. The clinical question is whether the stenosis is hemodynamically important and contributing to high-risk presentations.

163. E. Familial hypocalciuric hypercalcemia is usually benign and reflects reduced calcium-sensing receptor activity. The key clue is mild familial hypercalcemia with inappropriately low urinary calcium excretion, helping distinguish it from primary hyperparathyroidism.

164. E. Primary hyperparathyroidism causes autonomous PTH secretion, increasing serum calcium while lowering phosphate through renal phosphaturia. Hypercalciuria and recurrent calcium stones are common renal manifestations.

165. B. With aging and vascular disease, large arteries stiffen and buffer pulsatile ejection less effectively. Systolic pressure rises, reflected waves return earlier, and pulse pressure widens.

166. C. Contrast-associated AKI risk is greatest in patients with severe CKD, AKI, hemodynamic instability, or multiple insults, but needed imaging should not be reflexively withheld. Risk mitigation includes avoiding unnecessary exposure and maintaining appropriate intravascular volume when not contraindicated.

167. D. CKD nutrition requires balance: excessive protein can increase nitrogenous waste and glomerular workload, but overrestriction can cause protein-energy wasting. Dietary advice should be individualized to CKD stage, comorbidity, and nutritional status.

168. B. Cloudy effluent with abdominal pain is peritoneal dialysis peritonitis until proven otherwise. Dialysate cell count, differential, and culture are central to diagnosis, and empiric antimicrobial therapy should not be unnecessarily delayed.

169. B. CKD commonly causes a hypoproliferative anemia, but iron deficiency and other reversible processes remain frequent. Contemporary anemia care begins with a structured evaluation rather than assuming every low hemoglobin value reflects erythropoietin deficiency alone.

170. E. In chronic hypertension the autoregulatory curve shifts to higher pressures. Controlled rather than precipitous reduction is used in most hypertensive emergencies to reduce injury without compromising organ perfusion.

171. D. Angiotensin II preferentially supports efferent arteriolar tone. Blocking its formation lowers glomerular pressure, which is part of the kidney-protective effect but can also reduce GFR, especially with volume depletion or renovascular disease.

172. C. Acute illness with reduced intake can increase risks from volume-depleting or hemodynamically active drugs and can increase euglycemic ketoacidosis risk with SGLT2 inhibitors. Temporary holding and structured restart after recovery is safer than either automatic continuation or permanent discontinuation.

173. D. Salicylates directly stimulate ventilation and also generate organic acids, producing the characteristic combination of respiratory alkalosis and high-gap metabolic acidosis. A near-normal or slightly alkalemic pH can conceal the severity of the mixed disorder.

174. B. Acquired cystic kidney disease becomes more common with dialysis duration and is linked to renal cell carcinoma. Management is individualized, but new hematuria, a solid lesion, or suspicious imaging deserves evaluation.

175. B. In adults, infection-related GN can occur during an active infection and may be IgA-dominant, particularly with staphylococcal disease. Treatment centers on infection control plus management of kidney and volume complications.

176. E. Cisplatin is a well-recognized nephrotoxin that can cause acute tubular injury and renal magnesium wasting. Hydration and exposure-limiting strategies are used to reduce risk when cisplatin is necessary.

177. E. CKD is a major cardiovascular risk state, and statin-based therapy is recommended for many nondialysis adults based on age and risk. The approach changes once a patient is already established on maintenance dialysis, where initiating statins de novo has less clear benefit.

178. B. The urine dipstick detects heme pigment and therefore can be positive from myoglobin even when microscopy shows few erythrocytes. Myoglobin, volume depletion, acidosis, and tubular obstruction contribute to AKI in rhabdomyolysis.

179. A. Reduced net acid excretion can cause chronic metabolic acidosis as GFR falls. Persistent acidosis has adverse effects on muscle, bone, and possibly CKD progression, so treatment may include dietary measures and alkali when appropriate.

180. B. Shiga toxin–producing enteric infection can damage endothelium and trigger HUS, especially in children. Supportive care is central, with close management of volume, electrolytes, anemia, and kidney failure complications.

181. C. The risk of nephrogenic systemic fibrosis is strongly agent-dependent and is very low with more stable group II agents. In severe CKD, contrast should still be used only when the expected diagnostic benefit justifies exposure and the lowest effective dose should be chosen.

182. B. Amphotericin can cause renal vasoconstriction and tubular toxicity, leading to AKI, potassium and magnesium wasting, and impaired concentrating ability. Lipid formulations reduce but do not eliminate nephrotoxicity.

183. C. Lactic acidosis in shock is a marker of impaired perfusion and altered metabolism. Definitive therapy is hemodynamic resuscitation and source control; bicarbonate has limited, context-dependent roles and does not replace treatment of the cause.

184. B. Severe nephrotic syndrome increases venous thromboembolic risk, with membranous nephropathy among the higher-risk etiologies. New unilateral swelling or cardiopulmonary symptoms should prompt assessment for thrombosis.

185. B. Primary aldosteronism produces sodium retention and volume expansion, suppressing renin while aldosterone remains inappropriately elevated. Enhanced distal sodium delivery through ENaC creates a lumen-negative potential that favors potassium and hydrogen secretion.

186. C. Primary aldosteronism causes volume expansion and sodium retention, suppressing renin while aldosterone remains inappropriately high. Distal sodium reabsorption then promotes potassium and hydrogen secretion, leading to hypokalemic metabolic alkalosis.

187. D. ESA therapy reduces transfusion needs and can improve anemia-related symptoms, but trials of near-normal hemoglobin targets have shown harm in some CKD populations. Dosing should be individualized rather than aiming to normalize hemoglobin universally.

188. D. Tubulitis and interstitial inflammation are classic features of acute T-cell-mediated rejection. Biopsy is important because rejection, drug toxicity, obstruction, infection, and other causes of graft dysfunction can present similarly.

189. C. Mild hyperkalemia does not automatically require withdrawal of a kidney-protective ACE inhibitor or ARB. Clinicians should review diet, medications, acidosis, volume status, diuretic use, and potassium binders while balancing the benefits and risks of continued therapy.

190. E. A sudden aquaresis can cause dangerous overcorrection after the stimulus for vasopressin disappears. High-risk patients such as those with malnutrition are especially vulnerable to osmotic demyelination, so desmopressin and free water can be used to halt or reverse excessive correction.

191. A. Prerenal AKI reflects reduced perfusion with initially preserved parenchymal structure and may reverse quickly when the hemodynamic problem is corrected. Persistent or severe ischemia can progress to acute tubular injury.

192. A. Enteric hyperoxaluria occurs when unabsorbed fatty acids bind calcium, decreasing calcium available to bind oxalate in the intestinal lumen. More free oxalate is then absorbed and excreted in urine, increasing calcium oxalate supersaturation.

193. C. Cisplatin can cause renal magnesium wasting, and severe hypomagnesemia increases neuromuscular and arrhythmic risk. Magnesium deficiency also promotes refractory hypokalemia, so both deficits should be corrected while the cause is addressed.

194. B. Renal cortical necrosis is a rare but severe complication of profound ischemia and obstetric catastrophes associated with DIC. Unlike reversible prerenal AKI, extensive cortical necrosis can lead to permanent kidney failure.

195. B. Insulin deficiency and hypertonicity shift potassium out of cells, so serum potassium may initially be normal or high. At the same time, osmotic diuresis causes large urinary potassium losses, making total-body depletion typical and creating a risk of rapid hypokalemia during treatment.

196. C. Severe hypertension can cause hyperplastic arteriolosclerosis with concentric narrowing and sometimes fibrinoid necrosis. The resulting ischemia can amplify renin activation and further worsen blood pressure.

197. B. Beta blockade before alpha blockade can leave alpha-mediated vasoconstriction unopposed and precipitate severe hypertension. Preoperative management therefore establishes alpha blockade and restores volume before treating residual tachycardia.

198. A. NSAIDs inhibit renal prostaglandins, predisposing susceptible patients to afferent vasoconstriction, sodium retention, hypertension, hyperkalemia, and AKI. Chronic use can also contribute to analgesic nephropathy and should be avoided or minimized in CKD when alternatives exist.

199. B. Chronic acid retention activates catabolic pathways in skeletal muscle and contributes to protein breakdown. Correcting persistent metabolic acidosis can therefore be part of preserving muscle and nutritional status when clinically appropriate.

200. C. This is classic SIADH: hypotonic hyponatremia with inappropriately concentrated urine, relatively high urine sodium, and clinical euvolemia after exclusion of thyroid and adrenal disease. Small-cell lung cancer is a well-known source of ectopic vasopressin.

201. A. After confirmation of true resistant hypertension and optimization of the core regimen, mineralocorticoid receptor blockade is often effective. Hyperkalemia and worsening kidney function are important limitations, especially in CKD.

202. C. Type 4 RTA is characterized by hyperkalemia and a normal-gap metabolic acidosis due to deficient aldosterone effect and reduced ammonium excretion. Diabetic kidney disease is a classic setting for hyporeninemic hypoaldosteronism.

203. B. Chronic hyperkalemia management is multifactorial and should target reversible causes rather than relying on emergency shifting therapies. Newer potassium binders, diuretics, treatment of acidosis, and medication review can help maintain kidney- and heart-protective drugs.

204. B. ANCA-associated vasculitis classically causes pauci-immune necrotizing and crescentic glomerulonephritis. Pulmonary capillaritis can produce a pulmonary-renal syndrome.

205. E. Primary FSGS can recur rapidly after transplantation and may present with abrupt heavy proteinuria. Early recognition is important because treatment strategies differ from those for rejection or ischemic injury.

206. E. Dihydropyridine edema reflects an imbalance between precapillary arteriolar and postcapillary venous tone. It may improve with dose adjustment or RAS blockade and is not necessarily evidence of heart failure or major volume expansion.

207. A. The proximal tubule reabsorbs filtered glucose, phosphate, amino acids, bicarbonate, uric acid, and other solutes. Generalized proximal dysfunction produces Fanconi syndrome and may lead to type 2 RTA and metabolic bone disease.

208. C. Refractory pulmonary edema is a classic urgent indication for kidney replacement therapy when medical management cannot safely restore oxygenation. Dialysis decisions are driven by complications and clinical trajectory, not creatinine alone.

209. A. Nonmelanoma skin cancer risk is markedly increased after solid-organ transplantation. Sun protection, self-examination, and regular dermatologic surveillance are important components of long-term care.

210. E. New hypertension after 20 weeks accompanied by proteinuria or specific end-organ findings supports preeclampsia. Neurologic symptoms and hepatic involvement are concerning severe features and require urgent obstetric management.

211. C. PPIs are among many medications associated with acute interstitial nephritis. The presentation can be subtle, so medication history and urine findings are important, and definitive diagnosis may require biopsy in selected cases.

212. C. Urease-positive organisms split urea, raising urine pH and generating ammonium that favors struvite precipitation. These stones can enlarge rapidly into staghorn calculi and require both infection control and definitive stone management.

213. D. The renal medulla is especially vulnerable to sickling because it is hypoxic, acidic, and hypertonic. Recurrent ischemia can cause papillary necrosis, hematuria, and impaired urinary concentration.

214. B. IgA nephropathy commonly causes hematuria concurrently with or shortly after a mucosal infection. Mesangial IgA deposition is the defining pathologic feature.

215. D. In primary polydipsia, water intake exceeds excretory capacity while vasopressin is suppressed, so urine is typically maximally or nearly maximally dilute. A concentrated urine would suggest persistent vasopressin effect from SIADH, hypovolemia, or another stimulus.

216. B. Albuminuria can be transient with exercise, fever, infection, or marked hyperglycemia. Persistent abnormality should be confirmed, and a first-morning urine helps reduce variation from posture and activity.

217. A. Red-cell casts strongly localize bleeding to the nephron and, with hematuria and impaired filtration, point toward glomerulonephritis. Nephritic syndromes commonly include hypertension and edema from sodium retention.

218. B. Active glomerular sediment with rapid loss of kidney function is a nephrologic emergency. ANCA vasculitis, anti-GBM disease, lupus, infection-related GN, and other causes require prompt differentiation and targeted treatment.

219. A. Once intravascular volume is adequate, indiscriminate fluid administration can cause substantial harm. Reassessment of perfusion and fluid responsiveness helps separate needed resuscitation from avoidable fluid accumulation.

220. E. Diabetic nephropathy can progress from glomerular basement-membrane thickening and mesangial expansion to nodular sclerosis. Albuminuria and progressive loss of filtration are common clinical manifestations.

221. A. Trimethoprim has an amiloride-like effect on ENaC, decreasing distal sodium uptake and the electrical gradient for potassium secretion. The effect is especially important in CKD or when combined with other potassium-raising drugs.

222. C. BK polyomavirus reactivates under immunosuppression and can cause progressive tubulointerstitial nephropathy. Reducing immunosuppressive burden is the main strategy, balanced against the risk of precipitating rejection.

223. A. CKD alters phosphate handling, FGF23, calcitriol, and calcium balance long before overt kidney failure. These changes stimulate parathyroid hyperplasia and PTH secretion as an initially adaptive but eventually maladaptive response.

224. A. Distal RTA causes an inability to lower urine pH during systemic acidosis. Hypokalemia, alkaline urine, hypocitraturia, and calcium phosphate stones or nephrocalcinosis are classic associated features.

225. E. Muddy brown granular casts and tubular epithelial cells are classic urine findings in acute tubular injury. Sepsis can produce AKI through hemodynamic, inflammatory, endothelial, and microcirculatory mechanisms even without sustained severe hypotension.

226. D. Methanol is metabolized to formaldehyde and then formic acid, which causes severe acidosis and optic toxicity. Fomepizole and, in severe cases, hemodialysis are key therapies.

227. B. Extreme leukocytosis or thrombocytosis can cause artifactual potassium elevation during specimen processing. A normal carefully obtained plasma value without compatible ECG or clinical findings supports pseudohyperkalemia.

228. C. Lupus can produce several histologic patterns with different treatment implications. Significant proteinuria, active sediment, or unexplained loss of GFR generally warrants biopsy to define the class and activity/chronicity when feasible.

229. E. The classic episodic triad with paroxysmal hypertension warrants biochemical testing for pheochromocytoma or paraganglioma. Metanephrines are sensitive because catecholamines are continuously metabolized within the tumor.

230. A. MAO inhibition impairs metabolism of dietary sympathomimetic amines. Large tyramine exposure can increase catecholamine release and precipitate severe acute hypertension.

231. B. Atheroembolization can follow vascular instrumentation and produce delayed kidney injury with skin findings, eosinophilia, and complement consumption. Cholesterol clefts may be seen on tissue biopsy.

232. D. High urine volume is a foundational stone-prevention measure, and hypocitraturia can often be treated with potassium citrate. Severe dietary calcium restriction can increase oxalate absorption, while high sodium intake tends to increase urinary calcium.

233. D. Discordance between office and home readings raises concern for white-coat hypertension or a white-coat effect. Ambulatory or carefully obtained home measurements better characterize the patient's usual blood pressure and can prevent unnecessary intensification.

234. A. Abdominal compartment syndrome can reduce renal blood flow and increase renal venous pressure while impairing ventilation and other organ systems. Recognition and pressure-relieving interventions can be kidney-saving and lifesaving.

235. D. RAS blockade is particularly useful in proteinuric CKD because it reduces systemic and intraglomerular pressure and usually lowers albuminuria. Kidney function and potassium should be monitored, and dual ACE inhibitor–ARB therapy is generally avoided because harms outweigh benefit.

236. D. Albuminuria is a major independent predictor of CKD progression and cardiovascular events. At a given eGFR, substantially higher albuminuria generally identifies a much higher-risk phenotype.

237. A. Creatinine generation varies with muscle mass and diet, so serum creatinine can underestimate impairment in sarcopenia and overestimate impairment in very muscular people. Cystatin C provides an additional filtration marker and combined equations can improve accuracy in selected settings.

238. D. High ultrafiltration rates can exceed plasma refill and provoke intradialytic hypotension and ischemic stress. Prevention focuses on limiting interdialytic sodium and fluid gain, extending or individualizing treatment, and reassessing target weight and medications.

239. A. Alport syndrome results from pathogenic variants affecting type IV collagen and causes glomerular basement-membrane abnormalities. Hearing loss and ocular findings are important extrarenal clues.

240. C. Red-cell transfusion can lead to HLA sensitization, increasing panel-reactive antibodies and complicating donor matching. When clinically safe, anemia management seeks to reduce unnecessary transfusion exposure in transplant candidates.