COMPREHENSIVE MOCK EXAM 3

Supplemental to: Nephrology Board Review 2026-2027

Rasmus Broberg

ABIM Nephrology Certification Examination — 240-question MEDREV supplemental simulation; A–E single-best-answer format; domain-weighted to the official ABIM nephrology blueprint

Board-Review Questions

1. A patient with chronic hepatitis C has purpura, arthralgias, low C4, hematuria, and proteinuria. Which renal process is most likely?

A. Minimal change disease, which does not explain low complement, purpura, and circulating cryoprotein-related symptoms.

B. Anti-GBM disease, which causes linear IgG and pulmonary hemorrhage rather than low-C4 immune-complex vasculitis.

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

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

E. ADPKD, which causes cystic kidneys and hypertension rather than hypocomplementemic immune-complex disease.

2. A patient with gout has recurrent radiolucent kidney stones and urine pH 5.1. Which therapy most directly reduces recurrence?

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

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

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

D. Restrict all dietary calcium because uric acid stones form primarily from calcium-urate complexes rather than low urine pH.

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

3. A patient with a crush injury has dark urine, CK 60,000 U/L, positive dipstick for blood but few red cells, hyperkalemia, and AKI. What diagnosis best explains the kidney injury?

A. Glomerulonephritis, because dipstick heme with few red cells proves red-cell casts are being rapidly dissolved in urine.

B. Renal papillary necrosis, because muscle trauma directly causes papillary sloughing and releases myoglobin from the kidney.

C. Minimal change disease, because skeletal muscle injury causes podocyte effacement and nephrotic syndrome.

D. Postrenal obstruction from intact erythrocytes, because few red cells on microscopy indicates a large ureteral clot burden.

E. Rhabdomyolysis with myoglobin-mediated pigment nephropathy and acute tubular injury.

4. A patient is resuscitated after prolonged hemorrhagic shock and later develops oliguric AKI with granular casts. What is the most likely mechanism?

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

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

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

D. Ischemic acute tubular injury after sustained renal hypoperfusion.

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

5. A 62-year-old with CKD not requiring dialysis has no known atherosclerotic cardiovascular disease. Which lipid-management principle is most appropriate?

A. Avoid statins in all CKD because reduced GFR makes any LDL lowering more likely to cause kidney failure than cardiovascular benefit.

B. Use fibrate monotherapy for every patient because triglyceride lowering has stronger CKD outcome evidence than statin-based treatment.

C. Defer until dialysis starts before treating lipids because cardiovascular prevention has no benefit during nondialysis chronic kidney disease.

D. Treat only if nephrotic-range proteinuria develops because ordinary CKD without nephrosis does not independently increase cardiovascular risk.

E. Use statin-based therapy according to CKD and cardiovascular-risk recommendations because CKD substantially increases atherosclerotic risk.

6. A patient on peritoneal dialysis develops diffuse abdominal pain and cloudy effluent. What is the most important immediate diagnostic step?

A. Obtain a kidney biopsy because cloudy dialysate most often reflects recurrent glomerulonephritis rather than infection of the peritoneal cavity.

B. Stop all dialysis for several days without sampling the fluid because peritonitis cultures become more accurate after the effluent clears spontaneously.

C. Order only a serum procalcitonin because peritoneal fluid analysis does not distinguish infectious peritonitis from mechanical abdominal pain.

D. Send peritoneal effluent for cell count with differential and culture while promptly evaluating and treating suspected peritonitis.

E. Perform immediate nephrectomy because cloudy dialysate indicates irreversible native-kidney infection that cannot be treated through the peritoneal catheter.

7. A patient has rapidly progressive AKI, hematuria, proteinuria, and red-cell casts. Which action is most appropriate?

A. Treat as uncomplicated prerenal azotemia because red-cell casts are expected during any acute fall in renal perfusion.

B. Delay evaluation for several months because glomerular inflammation usually resolves before irreversible kidney injury develops.

C. Give empiric NSAIDs because reducing glomerular blood flow is the standard treatment for crescentic glomerulonephritis.

D. Urgently evaluate for rapidly progressive glomerulonephritis with serologies and kidney biopsy when feasible because disease-specific therapy may be time sensitive.

E. Assume obstruction because red-cell casts arise only when the ureter is completely blocked.

8. A patient with septic shock has persistent AKI despite restoration of mean arterial pressure. Why can kidney dysfunction continue?

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

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

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

9. An adult with nephrotic syndrome has circulating anti-PLA2R antibodies. Which diagnosis is most likely?

A. Minimal change disease, because anti-PLA2R antibodies identify diffuse foot-process effacement without subepithelial immune deposits.

B. IgA nephropathy, because anti-PLA2R antibodies are directed against mesangial IgA-containing immune complexes.

C. Primary membranous nephropathy driven by autoantibodies against a podocyte antigen.

D. Anti-GBM disease, because PLA2R is the principal antigen in the glomerular basement membrane targeted in pulmonary-renal syndrome.

E. Diabetic nephropathy, because anti-PLA2R levels track glycemic exposure and predict nodular mesangial sclerosis.

10. 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. What is the best interpretation?

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

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

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

D. Delayed graft function from ischemia-reperfusion injury, which does not by itself mean the allograft will never recover.

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

11. A kidney recipient develops fever and graft tenderness with pyuria and bacteriuria. What diagnosis should be considered?

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

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

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

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

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

12. A patient with CKD has fatigue, low transferrin saturation, and low ferritin. What is the most direct therapeutic issue to address?

A. Iron should be avoided because any CKD-associated anemia represents iron overload from reduced renal excretion rather than deficient available iron.

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

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

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

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

13. A patient with proteinuric CKD starts an ACE inhibitor and has a modest early rise in creatinine. Which renal hemodynamic effect explains this change?

A. Efferent arteriolar dilation lowers intraglomerular pressure and can produce a predictable initial fall in GFR.

B. Afferent arteriolar constriction is the direct pharmacologic action of ACE inhibition and is the main reason creatinine rises after every dose.

C. ACE inhibitors obstruct the ureter through smooth-muscle spasm, producing postrenal azotemia and hydronephrosis.

D. The drug causes acute immune-complex glomerulonephritis, so any creatinine increase proves an allergic renal lesion.

E. ACE inhibition increases angiotensin II signaling at the efferent arteriole and therefore raises intraglomerular pressure.

14. A patient 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. What additional process is suggested?

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

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

C. An isolated respiratory alkalosis because delta-gap analysis compares bicarbonate directly with $PaCO_2$ rather than with the serum anion gap.

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

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

15. A 48-year-old woman has recurrent nephrolithiasis, calcium 11.2 mg/dL, phosphate 2.2 mg/dL, and an inappropriately elevated parathyroid hormone level. What is the most likely diagnosis?

A. Familial hypocalciuric hypercalcemia, which usually has low urinary calcium excretion and is less commonly associated with recurrent stones.

B. Malignancy-associated hypercalcemia, which generally suppresses intact parathyroid hormone when PTH-related peptide or bone resorption is responsible.

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

D. Vitamin D intoxication, which tends to increase intestinal phosphate absorption and suppress endogenous parathyroid hormone secretion.

E. Hypoparathyroidism, which produces low calcium with high phosphate rather than hypercalcemia with an inappropriately high parathyroid hormone.

16. A dialysis patient with severe hyperkalemia receives intravenous insulin and glucose. What is the principal effect of insulin on potassium?

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

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

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

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

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

17. A kidney recipient misses immunosuppressive doses because of medication cost. What is the most appropriate clinical response?

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

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

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

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

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

18. 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. What is the best next step?

A. Continue the current fluids because rapid correction is desirable once renal perfusion and urine output have recovered.

B. Add a loop diuretic to accelerate free-water loss because preventing cerebral edema requires continued rapid sodium elevation.

C. Give sodium bicarbonate to replace intracellular sodium because malnutrition lowers the risk of osmotic demyelination syndrome.

D. Stop further overcorrection and use desmopressin with electrolyte-free water as needed to relower or stabilize the sodium trajectory.

E. Withhold all fluids and accept the rise because once correction exceeds the target there is no practical way to modify the course.

19. A dialysis patient has uncomfortable leg sensations at rest in the evening that improve with movement and disrupt sleep. What is the most likely diagnosis?

A. Uremic pericarditis, which produces positional chest discomfort and a friction rub rather than an urge to move the legs at night.

B. Calciphylaxis, which causes severe focal skin pain and ischemic plaques rather than transient discomfort relieved by movement.

C. Restless legs syndrome, which is common in advanced CKD and may be worsened by iron deficiency.

D. Dialysis disequilibrium syndrome, which causes acute neurologic symptoms during initial dialysis rather than chronic evening leg sensations.

E. Renal osteodystrophy, which can cause bone pain but does not characteristically improve immediately when the legs are moved.

20. A 22-year-old develops visible hematuria within one day of an upper respiratory infection. Complement is normal. Which disease is most likely?

A. Poststreptococcal glomerulonephritis, which usually occurs after a latent interval and often lowers complement.

B. Anti-GBM disease, which usually presents with rapidly progressive kidney injury rather than recurrent infection-linked gross hematuria alone.

C. Minimal change disease, which presents with nephrotic proteinuria rather than episodic glomerular hematuria.

D. IgA nephropathy causing synpharyngitic glomerular hematuria with mesangial IgA deposition.

E. Renal papillary necrosis, which causes nonglomerular bleeding and does not track closely with mucosal infections.

21. A 52-year-old with hypertension has repeated office readings near 152/92 mm Hg but home readings average 126/78 mm Hg. Which test best clarifies whether sustained hypertension is present?

A. Diagnose resistant hypertension immediately because any office value above goal proves persistent hypertension outside the clinic.

B. Use validated out-of-office blood-pressure measurement, preferably ambulatory monitoring, to assess for a white-coat effect.

C. Order plasma metanephrines first because discordant office and home readings are most commonly caused by pheochromocytoma.

D. Stop all antihypertensive drugs for one month because treated readings cannot be interpreted with ambulatory monitoring.

E. Use a single repeat office measurement because multiple home readings should not influence the diagnosis of sustained hypertension.

22. A patient 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. What is the best treatment principle?

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

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

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.

23. A patient with tumor lysis syndrome develops symptomatic hypocalcemia and marked hyperphosphatemia. What explains the low calcium?

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

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

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

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

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

24. A man with angiokeratomas, neuropathic pain, cardiomyopathy, and CKD has a family history consistent with X-linked inheritance. Which metabolic disease is most likely?

A. Alport syndrome, which is a collagen IV disorder and does not cause angiokeratomas from lysosomal lipid storage.

B. Primary hyperoxaluria, which causes oxalate overproduction, stones, and nephrocalcinosis rather than angiokeratomas and neuropathic pain.

C. Cystinosis, which usually presents much earlier with Fanconi syndrome and corneal cystine crystals.

D. Amyloid light-chain disease, which is acquired and is not an X-linked lysosomal enzyme deficiency.

E. Fabry disease from alpha-galactosidase A deficiency with glycosphingolipid accumulation.

25. A marathon runner drinks large amounts of hypotonic fluid and becomes confused near the finish. Sodium is 119 mEq/L. What is the most important initial principle?

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

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

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

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

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

26. A hemodialysis patient has severe generalized pruritus without cholestasis or a primary skin disorder. Which management approach is most appropriate?

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

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

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

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

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

27. A patient with CKD has hypertension and severely increased albuminuria. Which antihypertensive class is particularly useful for reducing albuminuria and slowing progression when tolerated?

A. An ACE inhibitor or angiotensin receptor blocker titrated to a tolerated evidence-based dose.

B. A direct renin inhibitor combined routinely with an ACE inhibitor because triple renin-angiotensin blockade provides greater renal protection.

C. A short-acting alpha blocker alone because reducing sympathetic tone lowers albuminuria more reliably than renin-angiotensin system blockade.

D. A nondihydropyridine calcium-channel blocker must replace all renin-angiotensin blockade because ACE inhibitors increase intraglomerular pressure.

E. Hydralazine monotherapy is preferred because it reduces protein filtration directly without affecting potassium or glomerular hemodynamics.

28. A recipient develops graft dysfunction with microvascular inflammation, circulating donor-specific antibodies, and complement deposition in peritubular capillaries. What process is most likely?

A. Isolated calcineurin inhibitor vasoconstriction, which does not by itself produce donor-specific antibodies and microvascular immune injury.

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

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

D. Recurrent minimal change disease, which causes podocyte injury and proteinuria rather than alloantibody-mediated capillaritis.

E. Postrenal obstruction, which causes hydronephrosis and pressure injury rather than donor-specific antibody formation.

29. A stable adult with CKD asks about dietary protein. Which general principle is most appropriate?

A. Use a very high-protein diet in all CKD because increased nitrogen load reduces intraglomerular pressure and slows nephron loss.

B. Eliminate nearly all dietary protein regardless of nutritional status because protein-energy wasting has little effect on outcomes in chronic kidney disease.

C. Protein intake has no effect on uremic solute generation, so dietary counseling is unnecessary even as kidney function declines.

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.

30. A patient with eGFR 7 mL/min/1.73 m2 is eating well, has controlled potassium and volume status, and has no uremic symptoms. Which statement about starting dialysis is most accurate?

A. 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.

B. Dialysis initiation should be driven by symptoms, refractory complications, and overall clinical context rather than a fixed eGFR threshold alone.

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

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.

31. A kidney recipient on mycophenolate develops persistent diarrhea and leukopenia. Which medication effect is most likely?

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

B. Prednisone mineralocorticoid toxicity, which typically causes diarrhea and severe leukopenia rather than glucose intolerance and bone disease.

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

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

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

32. A dialysis patient with obesity, diabetes, warfarin exposure, and severe secondary hyperparathyroidism develops exquisitely painful retiform plaques that become necrotic. What is the most likely diagnosis?

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

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

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

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

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

33. A patient with suspected pheochromocytoma is being prepared for surgery. Which pharmacologic sequence is safest?

A. Begin a nonselective beta blocker first because unopposed alpha stimulation lowers blood pressure by reducing cardiac output.

B. Use beta blockade alone because catecholamine-mediated vasoconstriction is primarily beta-2 receptor dependent.

C. Establish adequate alpha-adrenergic blockade and volume repletion before adding beta blockade if needed for tachycardia.

D. Avoid alpha blockade because it increases the risk of intraoperative hypertensive crisis from catecholamine release.

E. Start a thiazide as the sole preoperative therapy because chronic volume contraction protects against catecholamine surges.

34. An 86-year-old with advanced CKD, metastatic cancer, severe frailty, and repeated hospitalizations asks whether dialysis is mandatory. Which approach is most appropriate?

A. Use shared decision-making that compares dialysis with comprehensive conservative kidney management in light of goals, prognosis, symptoms, and treatment burden.

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. Tell the patient that dialysis will certainly prolong life and preserve independence regardless of metastatic cancer and severe frailty.

E. Delay discussion until the patient loses decision-making capacity because advance planning can bias patients against life-prolonging kidney replacement therapy.

35. A hemodialysis patient with a tunneled catheter develops fever, rigors during dialysis, and positive blood cultures. What complication should be presumed?

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

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

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

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

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

36. An ICU patient has thrombocytopenia, schistocytes, hemolysis, and AKI. Which diagnostic framework is most appropriate?

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

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

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

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 rather than hemolytic.

37. A patient with CKD needs iodinated contrast for an urgent procedure. Which preventive principle is most appropriate for a high-risk patient without pulmonary edema?

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

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

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

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

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

38. A patient with severe salicylate toxicity has tinnitus, tachypnea, confusion, pulmonary edema, and worsening acidemia. What is the role of hemodialysis?

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

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

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

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

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

39. A patient has recurrent episodes of hypertension after taking a monoamine oxidase inhibitor and eating aged cheese. What is the mechanism?

A. Excess catecholaminergic activity from impaired tyramine metabolism can trigger an acute hypertensive crisis.

B. Tyramine blocks alpha receptors and causes profound vasodilation, so the hypertension reflects compensatory renal sodium retention over months.

C. Aged cheese inhibits epithelial sodium channels, producing Liddle-like sodium retention independent of sympathetic activity.

D. Monoamine oxidase inhibition prevents aldosterone synthesis, and the resulting salt wasting paradoxically produces catecholamine-independent hypertension.

E. The interaction causes direct renal artery thrombosis, making abrupt renovascular hypertension the expected mechanism.

40. A patient has potassium 2.7 mEq/L, hypertension, metabolic alkalosis, suppressed renin, and elevated aldosterone. Which diagnosis is most likely?

A. Liddle syndrome, which causes the same phenotype but suppresses aldosterone because ENaC activity is independent of mineralocorticoid signaling.

B. Renal artery stenosis, which produces secondary hyperaldosteronism with elevated rather than suppressed renin activity.

C. Gitelman syndrome, which causes hypokalemic alkalosis with low or normal blood pressure and activation of renin and aldosterone.

D. Vomiting, which causes chloride-responsive alkalosis but does not typically suppress renin while producing autonomous aldosterone elevation.

E. Primary aldosteronism causing renal sodium retention, potassium wasting, and hydrogen ion secretion.

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

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

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

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

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

42. A patient receiving amphotericin B develops rising creatinine, hypokalemia, and hypomagnesemia. What renal toxicity is characteristic?

A. Selective podocyte injury causing nephrotic syndrome without tubular electrolyte abnormalities.

B. Renal vasoconstriction plus direct tubular membrane injury causing reduced GFR and distal electrolyte wasting.

C. Exclusive proximal glucose transporter blockade producing isolated glucosuria with no effect on potassium or magnesium.

D. Immune-complex crescentic glomerulonephritis as the universal mechanism of amphotericin nephrotoxicity.

E. Renal artery thrombosis from drug-induced platelet aggregation, which explains nearly all creatinine rises during treatment.

43. A dialysis patient has persistently low PTH, low bone turnover on biopsy, and repeated calcium loading. Which bone disorder is most likely?

A. Osteitis fibrosa cystica, which is a high-turnover lesion driven by marked secondary hyperparathyroidism rather than chronically suppressed PTH.

B. Osteomalacia from severe vitamin C deficiency, which is the dominant cause of low-turnover renal bone disease in modern dialysis populations.

C. Paget disease, which causes focal high-turnover bone lesions and is unrelated to PTH suppression or CKD-mineral bone management.

D. Adynamic bone disease from excessive suppression of PTH signaling and skeletal bone turnover.

E. Primary hyperparathyroid bone disease, which requires autonomous high PTH and does not fit a persistently low circulating PTH concentration.

44. A septic patient develops AKI with granular 'muddy brown' casts and renal tubular epithelial cells in the urine. What lesion is most likely?

A. Acute tubular injury from ischemic and inflammatory stress with tubular epithelial damage.

B. Minimal change disease, which causes nephrotic proteinuria but not muddy granular casts from injured tubular cells.

C. Acute interstitial nephritis, which more often produces pyuria and white-cell casts rather than classic muddy brown casts.

D. Postrenal obstruction, which may cause hydronephrosis but does not characteristically produce abundant tubular epithelial casts.

E. Renal-vein thrombosis, which is a vascular event and does not explain the typical tubular sediment.

45. A patient with AKI has severe metabolic acidosis with hemodynamic instability despite treatment of the cause and appropriate medical therapy. What renal support may be required?

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

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

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

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

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

46. A young adult has recurrent stones and hexagonal crystals on urine microscopy. Which disorder is most likely?

A. Primary hyperoxaluria, which produces calcium oxalate crystallization rather than the classic hexagonal cystine crystal morphology.

B. Distal renal tubular acidosis, which favors calcium phosphate stones in alkaline urine rather than inherited cystine transport failure.

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

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

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

47. A child develops cola-colored urine, edema, and hypertension two weeks after streptococcal pharyngitis. C3 is low. What is the most likely diagnosis?

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. Minimal change disease, which produces nephrotic syndrome and usually lacks low complement or red-cell casts.

D. Alport syndrome, which is inherited and does not characteristically cause transient hypocomplementemia after pharyngitis.

E. Renal-vein thrombosis, which can cause hematuria but does not explain low complement after streptococcal infection.

48. A 51-year-old with long-standing analgesic overuse has CKD, sterile pyuria, and papillary calcifications. What pathologic process is most characteristic?

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

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

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

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

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

49. A patient with chronic hypercapnic COPD has PaCO2 60 mm Hg and bicarbonate 33 mEq/L. Which interpretation is most likely?

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

B. Acute respiratory acidosis, because bicarbonate rises by roughly 8 to 10 mEq/L immediately after any acute PaCO2 increase of 20 mm Hg.

C. Primary metabolic alkalosis with no respiratory component, because an elevated bicarbonate fully explains the high PaCO2 through hypoventilation.

D. Mixed respiratory alkalosis and metabolic acidosis, because chronic lung disease lowers PaCO2 and bicarbonate together rather than raising both.

E. Normal acid-base physiology, because PaCO2 and bicarbonate can rise in parallel without reflecting a primary respiratory disorder.

50. A patient with tinnitus, nausea, tachypnea, and salicylate overdose has pH 7.44, PaCO2 22 mm Hg, and bicarbonate 14 mEq/L. What acid-base disorder is present?

A. A pure compensated metabolic acidosis, because salicylates do not directly stimulate the respiratory center early in toxicity.

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

C. A mixed respiratory acidosis and metabolic alkalosis, because tinnitus is associated with hypoventilation and gastric acid retention.

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

E. A normal acid-base state, because a near-normal pH excludes clinically significant simultaneous metabolic and respiratory disorders.

51. A 37-year-old has hypertension, flank fullness, enlarged kidneys with innumerable bilateral cysts, and a family history of subarachnoid hemorrhage. Which diagnosis is most likely?

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

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

C. Acquired cystic kidney disease, which arises mainly in advanced CKD and produces small to normal kidneys rather than inherited enlargement.

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

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

52. 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 thick ascending limb, which would mainly impair sodium-potassium-chloride transport and urinary concentrating ability.

C. The collecting-duct principal cell, which primarily regulates sodium, potassium, and water rather than bulk nutrient reabsorption.

D. The glomerular filtration barrier, because selective protein permeability defects cause glucosuria and aminoaciduria without hyperglycemia.

E. The renal pelvis, where defects cause hydronephrosis but not generalized loss of filtered solutes.

53. A dialysis patient has severe secondary hyperparathyroidism with hypercalcemia that limits vitamin D receptor activator use. Which medication class can lower PTH without adding calcium?

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

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

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

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

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

54. A patient with profound hypokalemia remains refractory to potassium replacement. Magnesium is 0.9 mg/dL. Why is magnesium replacement important?

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

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

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

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

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

55. A patient with diabetic ketoacidosis has serum potassium 5.3 mEq/L before treatment. What is the most accurate statement about total-body potassium?

A. Total-body potassium is increased because the elevated serum concentration proves that intracellular stores are also expanded before insulin therapy.

B. Total-body potassium is normal because acidosis prevents urinary potassium loss until insulin has been administered and glucose begins to fall.

C. Total-body potassium cannot change in ketoacidosis because potassium is an intracellular ion and therefore is not lost in urine during hyperglycemia.

D. The serum potassium concentration directly measures exchangeable body potassium and can be used without considering redistribution or urinary losses.

E. Total-body potassium is usually depleted despite a normal or high initial serum potassium because osmotic diuresis causes substantial urinary losses.

56. A patient with SIADH receives 0.9% saline. Urine osmolality is 700 mOsm/kg, and the serum sodium falls further. What best explains this response?

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

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

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

D. Saline causes pseudohyponatremia by increasing the plasma lipid fraction, so the fall in sodium is a laboratory artifact rather than physiology.

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

57. A 29-year-old woman has severe hypertension and an abdominal bruit. Imaging shows alternating stenosis and dilation of a renal artery. What is the likely diagnosis?

A. Atherosclerotic renal artery stenosis, which typically causes smooth ostial plaque in older patients with diffuse vascular disease.

B. Polyarteritis nodosa, which usually produces diffuse microaneurysms rather than the classic medial fibroplasia pattern described.

C. Renal-vein thrombosis, which causes venous filling defects rather than alternating arterial stenosis and dilation.

D. Fibromuscular dysplasia causing a characteristic beaded appearance of the renal artery.

E. Autosomal dominant polycystic kidney disease, which causes renal cysts but not a beaded renal artery morphology.

58. A patient with severe AKI develops a pericardial friction rub and chest discomfort without another explanation. What complication is most concerning?

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

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

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

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

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

59. A patient with weakness has potassium 2.9 mEq/L. Twenty-four-hour urinary potassium excretion is high despite hypokalemia. What does this indicate?

A. Renal potassium wasting rather than an appropriate renal response to an extrarenal potassium loss.

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

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

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

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

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

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

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

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

61. A patient with severe AKI continues metformin and develops shock with profound lactic acidosis. Why can kidney failure increase concern about the drug?

A. Metformin is metabolized entirely by the liver and kidney function has no effect on exposure or toxicity.

B. The drug directly generates ketone bodies in the kidney, so lactic acidosis is unrelated to accumulation during AKI.

C. Metformin blocks distal hydrogen secretion and causes a pure normal-anion-gap renal tubular acidosis rather than lactic acidosis.

D. Kidney failure accelerates metformin elimination, so toxicity becomes less likely as GFR falls.

E. Metformin is renally cleared, so severe acute loss of kidney function can promote accumulation when other causes of lactate generation are present.

62. A patient with potassium 7.2 mEq/L has peaked T waves and QRS widening. Which intervention should be given immediately to reduce the risk of malignant arrhythmia?

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

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

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

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

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

63. A patient with resistant hypertension is taking lisinopril, amlodipine, and hydrochlorothiazide. Kidney function and potassium permit additional therapy. Which class is commonly useful as a fourth agent after true resistance is confirmed?

A. A second ACE inhibitor, because combining two agents from the same class improves pressure control without increasing adverse effects.

B. An NSAID, because renal prostaglandin inhibition potentiates diuretic natriuresis and lowers blood pressure in resistant hypertension.

C. A mineralocorticoid receptor antagonist, with potassium and kidney function monitored closely.

D. A short-acting beta agonist, because peripheral beta-2 stimulation is a standard chronic treatment for resistant hypertension.

E. A potassium supplement regardless of baseline potassium, because improving potassium stores directly reverses all causes of resistant hypertension.

64. A nursing-home resident with poor access to water has sodium 166 mEq/L for several days but no seizures. 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. Withhold enteral water because gastrointestinal absorption is too unpredictable for controlled treatment of chronic hypernatremia.

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

65. A patient with recurrent calcium phosphate stones has bicarbonate 17 mEq/L, potassium 3.1 mEq/L, and urine pH 6.4 despite systemic metabolic acidosis. Which disorder is most likely?

A. Proximal renal tubular acidosis, in which distal acidification remains intact once serum bicarbonate falls below the reabsorptive threshold.

B. Distal renal tubular acidosis with impaired collecting-duct hydrogen secretion and failure to acidify the urine.

C. Type 4 renal tubular acidosis, which generally produces hyperkalemia and can still lower urine pH despite reduced ammonium excretion.

D. Chronic diarrhea, which causes extrarenal bicarbonate loss and should lead to increased renal ammonium excretion with acidic urine.

E. Respiratory alkalosis, which lowers serum bicarbonate through compensation but does not explain persistently alkaline urine and nephrocalcinosis.

66. A patient with resistant hypertension snores loudly, has witnessed apneas, and daytime sleepiness. What secondary contributor should be addressed?

A. Central diabetes insipidus, because nocturnal polyuria is the usual mechanism linking sleep symptoms to persistent hypertension.

B. Chronic respiratory alkalosis, because hypocapnia during sleep directly causes daytime resistant hypertension.

C. Renal tubular acidosis, because bicarbonate loss is the principal cause of snoring and nocturnal blood-pressure surges.

D. Nephrotic syndrome, because proteinuria itself produces recurrent upper-airway obstruction during sleep.

E. Obstructive sleep apnea, which is strongly associated with difficult-to-control hypertension and nocturnal sympathetic activation.

67. A long-term transplant recipient develops diabetes, weight gain, osteoporosis, and skin fragility. Which immunosuppressant contributes to this cluster?

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

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

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

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

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

68. 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 directly measures renal blood flow, so it is unaffected by inflammation, corticosteroids, thyroid disease, or other non-GFR factors.

B. Cystatin C is secreted only by the proximal tubule and therefore rises exclusively in tubular disease rather than with reduced filtration.

C. Cystatin C can improve GFR estimation when creatinine is misleading because creatinine generation is strongly influenced by muscle mass.

D. Cystatin C should replace creatinine in every patient because combining filtration markers never improves GFR estimation accuracy.

E. Cystatin C is useful only in dialysis patients because it cannot estimate kidney function when measured GFR exceeds 15 mL/min/1.73 m2.

69. A patient with persistent vomiting has pH 7.52, bicarbonate 38 mEq/L, and urine chloride 6 mEq/L. Which treatment is most appropriate?

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

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

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

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

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

70. A patient with advanced CKD needs iodinated contrast for an urgent, potentially lifesaving vascular study. Which approach best balances renal and clinical risk?

A. Use contrast when the diagnostic benefit is important, minimize avoidable nephrotoxic exposures, and optimize volume status rather than withholding necessary imaging automatically.

B. Never give iodinated contrast at low GFR because the risk of permanent dialysis is certain regardless of indication or preventive measures.

C. Delay all urgent imaging until creatinine normalizes because missing a vascular emergency carries less risk than any contrast exposure.

D. Give prophylactic high-dose NSAIDs because afferent vasoconstriction protects tubules from contrast-associated oxidative stress.

E. Use repeated contrast doses to precondition the kidney because more exposure reduces rather than increases risk.

71. A patient receiving trimethoprim develops potassium 6.0 mEq/L without a major change in GFR. Which mechanism best explains the hyperkalemia?

A. Trimethoprim stimulates mineralocorticoid receptors, increasing distal sodium reabsorption and causing potassium to leave cells through sodium-potassium ATPase.

B. Trimethoprim inhibits proximal bicarbonate reabsorption, producing severe alkalosis that directly prevents distal potassium secretion.

C. Trimethoprim causes obligate potassium release from skeletal muscle by uncoupling oxidative phosphorylation at standard therapeutic concentrations.

D. Trimethoprim blocks epithelial sodium channels in the collecting duct, reducing the lumen-negative potential that drives potassium secretion.

E. Trimethoprim raises renin and aldosterone markedly, but paradoxical aldosterone excess causes potassium retention in the cortical collecting duct.

72. Months after transplantation, a recipient develops worsening hypertension, graft dysfunction, and a bruit over the allograft. Which diagnosis should be considered?

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

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

E. Ureteral reflux alone, which causes recurrent infection rather than a hemodynamic bruit and renovascular hypertension.

73. A patient with oliguric AKI receives repeated large fluid boluses despite no evidence of fluid responsiveness and develops worsening edema. Why is this harmful?

A. Fluid overload improves every form of AKI by increasing renal interstitial pressure and therefore accelerating glomerular filtration.

B. Positive fluid balance can worsen pulmonary gas exchange, tissue edema, and venous congestion without restoring renal perfusion.

C. Edema is harmless in critical illness because only intravascular volume affects organ function.

D. Additional crystalloid always increases urine output in intrinsic tubular injury, regardless of hemodynamic responsiveness.

E. Fluid accumulation lowers venous pressure and therefore protects the kidney from congestion.

74. A patient with advanced CKD has recurrent mild hyperkalemia but no ECG changes. Which chronic strategy is most appropriate?

A. Give intravenous calcium at home every day because calcium removes potassium from the body and is the standard chronic treatment for stable hyperkalemia.

B. Stop all vegetables and fruits permanently because dietary potassium restriction should ignore nutritional quality and the patient's actual contributors.

C. Review dietary potassium, constipation, acidosis, and potassium-raising drugs, and use diuretics or potassium binders when appropriate to preserve beneficial therapies.

D. Use insulin injections without glucose between meals because repeated intracellular shifting is the safest long-term method of potassium control.

E. Avoid correcting metabolic acidosis because raising bicarbonate always increases serum potassium by driving potassium out of skeletal muscle cells.

75. A patient with decompensated cirrhosis has edema, ascites, and sodium 126 mEq/L. What is the main mechanism of the hyponatremia?

A. Complete suppression of vasopressin causes excessive renal water loss that secondarily lowers serum sodium in edematous states.

B. Primary renal sodium wasting from absent aldosterone activity is the dominant mechanism in most patients with advanced cirrhosis.

C. Pseudohyponatremia from high plasma proteins is expected because cirrhosis usually produces extreme hyperproteinemia rather than hypoalbuminemia.

D. Reduced effective arterial blood volume causes nonosmotic vasopressin release and water retention despite total-body volume expansion.

E. Hypertonicity from retained urea draws water out of cells and is the principal explanation for low sodium in uncomplicated cirrhosis.

76. A patient has a staghorn calculus, alkaline urine, and recurrent infections with Proteus species. What is the stone composition most likely to be?

A. Uric acid, which is favored by persistently acidic urine and is unrelated to urease activity or infection-associated alkalinization.

B. Cystine, which reflects an inherited amino acid transport disorder and typically forms recurrent stones without requiring infection.

C. Magnesium ammonium phosphate formed in alkaline urine during infection with a urease-producing organism.

D. Calcium oxalate, which is the most common stone overall but does not characteristically form infection-driven staghorn calculi from urease.

E. Xanthine, which is a rare stone type associated with purine metabolism or xanthine oxidase inhibition rather than alkaline infected urine.

77. A patient with type 2 diabetes, albuminuric CKD, and persistent albuminuria despite optimized ACE inhibitor and SGLT2 inhibitor therapy has normal potassium. Which additional class may reduce kidney and cardiovascular risk in appropriate patients?

A. Chronic fludrocortisone, because mineralocorticoid receptor activation reduces albuminuria and lowers the risk of hyperkalemia in diabetic CKD.

B. A long-term NSAID, because afferent arteriolar vasoconstriction improves diabetic glomerular hyperfiltration without increasing kidney injury risk.

C. Routine dual ACE inhibitor and ARB therapy, because combined blockade has superior hard kidney outcomes and less hyperkalemia than either drug alone.

D. High-dose acetazolamide, because chronic bicarbonaturia suppresses diabetic glomerular injury and substitutes for mineralocorticoid receptor blockade.

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

78. A patient with membranous nephropathy has albumin 1.8 g/dL and develops unilateral leg swelling. What complication requires urgent evaluation?

A. Venous thromboembolism, because severe nephrotic syndrome is a hypercoagulable state.

B. Acute arterial vasculitis, because loss of albumin selectively causes inflammatory thrombosis of medium arteries rather than veins.

C. Compartment syndrome from renal salt wasting, because nephrotic syndrome lowers total-body sodium and causes intramuscular edema.

D. Calciphylaxis, because low albumin alone causes painful calcified plaques in patients with normal kidney function.

E. Deep soft-tissue infection is the only important cause because nephrotic syndrome protects against venous thrombosis.

79. A patient treated with cisplatin has AKI and persistent hypomagnesemia. Which mechanism is most likely?

A. Cisplatin stimulates aldosterone receptors and therefore causes magnesium retention rather than urinary loss.

B. The drug causes isolated glomerular hyperfiltration, so electrolyte abnormalities must be unrelated to kidney effects.

C. Cisplatin blocks ADH release from the pituitary and causes pure central diabetes insipidus without tubular injury.

D. Direct tubular toxicity with renal magnesium wasting, often involving proximal and distal tubular segments.

E. The medication causes urinary magnesium precipitation as obstructing stones, making crystal nephropathy the only renal lesion.

80. A patient on chronic lithium presents with confusion, tremor, ataxia, AKI, and a markedly elevated lithium level. When is hemodialysis especially useful?

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

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

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

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

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

81. A kidney recipient develops declining urine output and hydronephrosis on ultrasound. What complication is most likely?

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

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

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

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

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

82. Which kidney transplant recipient is at highest risk for primary CMV infection after transplantation?

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

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

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

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

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

83. A patient with CKD is receiving a narrow-therapeutic-index drug cleared by the kidneys. Creatinine-based eGFR may be inaccurate because of severe sarcopenia. What is the best dosing principle?

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

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

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

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

84. A nondiabetic patient with proteinuric CKD and eGFR 38 mL/min/1.73 m2 is already receiving maximally tolerated renin-angiotensin system blockade. 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. A dual ACE inhibitor plus ARB regimen, because combining both arms of renin-angiotensin blockade improves kidney outcomes without added harm.

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

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

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

85. A patient with multiple myeloma develops glucosuria with normal serum glucose, phosphaturia, aminoaciduria, hypouricemia, and bicarbonate wasting. What renal tubular disorder is present?

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

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

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

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

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

86. A young patient has early severe hypertension, hypokalemic metabolic alkalosis, suppressed renin, and suppressed aldosterone. Which inherited disorder is most likely?

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

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

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

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

E. Type 4 renal tubular acidosis, which causes hyperkalemia and metabolic acidosis rather than hypokalemic alkalosis.

87. A dialysis patient has persistently elevated serum phosphate despite dietary counseling. Which treatment principle is appropriate?

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

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

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

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

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

88. A young adult has hemoptysis and rapidly progressive glomerulonephritis. Immunofluorescence shows linear IgG along glomerular capillary walls. What is the diagnosis?

A. IgA nephropathy, which shows mesangial IgA rather than linear capillary-wall IgG staining.

B. Anti-glomerular basement membrane disease producing linear IgG staining and a pulmonary-renal syndrome.

C. C3 glomerulopathy, which shows dominant C3 deposition from complement dysregulation rather than linear IgG.

D. Membranous nephropathy, which shows granular capillary-wall immune deposits rather than a smooth linear pattern.

E. FSGS, which is a pattern of sclerosis and does not produce linear glomerular immunoglobulin deposition.

89. A patient arrives with blood pressure 230/130 mm Hg, confusion, papilledema, and acute kidney injury. 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. Papilledema indicates chronic uncomplicated hypertension and supports outpatient oral medication adjustment without monitoring.

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

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

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

90. A patient with recurrent calcium oxalate stones has low urine volume and low urinary citrate. Which preventive strategy is most appropriate?

A. Restrict dietary calcium severely because lower intestinal calcium always reduces oxalate absorption and prevents calcium oxalate stones.

B. Increase sodium intake because higher sodium delivery lowers urinary calcium excretion and improves the effectiveness of citrate therapy.

C. Increase fluid intake to raise urine volume and use potassium citrate when appropriate to increase urinary citrate and reduce calcium stone supersaturation.

D. Use chronic loop diuretic therapy because loop blockade lowers urinary calcium and is preferred over thiazide-type agents for stone prevention.

E. Acidify the urine aggressively because lowering urine pH increases citrate excretion and reduces calcium oxalate crystallization in most patients.

91. A biopsy report uses the term 'membranoproliferative pattern of injury.' What does this phrase imply?

A. A single inherited disease caused only by collagen IV mutations, making serologic evaluation unnecessary.

B. A synonym for minimal change disease because both have normal light microscopy and podocyte effacement.

C. A diagnosis that excludes monoclonal gammopathy, infection, autoimmune disease, and complement dysregulation.

D. A purely vascular lesion confined to renal arteries and unrelated to glomerular immune or complement injury.

E. A histologic pattern that can arise from several immune-complex or complement-mediated diseases rather than a single etiology.

92. A kidney recipient had FSGS as the cause of native kidney failure and develops massive proteinuria within days of transplantation. What should be suspected?

A. Recurrent ADPKD, because cyst formation from recipient genes develops within days in a genetically normal donor kidney.

B. Early recurrence of primary FSGS in the allograft, likely mediated by circulating podocyte-injuring factors.

C. Chronic calcineurin fibrosis, because years of drug exposure are not required for extensive interstitial scarring and immediate nephrotic syndrome.

D. Acute pyelonephritis, because bacterial infection is the usual cause of abrupt nephrotic-range proteinuria immediately after transplant.

E. Transplant renal artery stenosis, because renovascular disease selectively causes massive protein loss within the first postoperative days.

93. A patient 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. What is the best management principle?

A. Permanently stop all renin-angiotensin system therapy at the first potassium value above normal because recurrent hyperkalemia cannot be mitigated.

B. Add an ACE inhibitor to the ARB because dual blockade lowers potassium by increasing distal sodium delivery and aldosterone secretion.

C. Give chronic intravenous calcium because membrane stabilization lowers total-body potassium and allows the ARB to be continued indefinitely.

D. Address reversible contributors and use dietary, diuretic, bicarbonate, or potassium-binding strategies when appropriate before abandoning beneficial RAS blockade.

E. Increase dietary potassium to stimulate renal potassium secretion because higher intake is the preferred adaptation strategy in proteinuric CKD.

94. An older diabetic patient with active Staphylococcus aureus infection develops AKI, hematuria, proteinuria, and low C3. Which glomerular disease should be considered?

A. Primary IgA nephropathy solely because IgA deposition can occur, making the active bacterial infection clinically irrelevant.

B. Infection-related glomerulonephritis associated with ongoing bacterial infection.

C. Minimal change disease because bacterial infection usually causes isolated podocyte effacement without complement activation.

D. Anti-GBM disease because low C3 during active staphylococcal infection is specific for linear anti-basement-membrane antibodies.

E. FSGS because infection-related immune complexes routinely cause segmental scars without proliferative glomerular inflammation.

95. A hemodialysis patient repeatedly becomes hypotensive near the end of treatment after large interdialytic weight gains. Which strategy most directly addresses the problem?

A. Increase the ultrafiltration rate further so the treatment finishes earlier and the circulation has less cumulative exposure to dialysis.

B. Give routine large saline boluses before every treatment without changing fluid goals because chronic volume expansion prevents future hypotension safely.

C. Reduce excessive ultrafiltration stress by reassessing target weight, treatment time, sodium intake, and the required fluid-removal rate.

D. Raise dialysate temperature substantially because warming improves vascular tone and reduces intradialytic vasodilation during rapid fluid removal.

E. Stop all antihypertensive therapy permanently because blood-pressure medications are the sole cause of intradialytic hypotension in volume-overloaded patients.

96. A 66-year-old man taking lithium has persistent polyuria, sodium 149 mEq/L, and urine osmolality 130 mOsm/kg. Desmopressin produces little change. Which therapy is most directly useful if lithium cannot immediately be stopped?

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

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

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

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

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

97. A long-term peritoneal dialysis patient has progressively poor ultrafiltration and increasing transport of small solutes on peritoneal equilibration testing. 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. Changes in the peritoneal membrane can increase solute transport and dissipate the glucose osmotic gradient more rapidly, reducing ultrafiltration.

D. Small-solute transport falls to zero, trapping urea in blood and causing a stronger glucose gradient that paradoxically lowers ultrafiltration.

E. Peritoneal dialysis failure is caused only by catheter migration; membrane transport characteristics never change with long treatment duration.

98. How do SGLT2 inhibitors alter tubuloglomerular feedback in diabetic or proteinuric CKD?

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

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

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

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

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

99. A 79-year-old cachectic patient has creatinine 0.8 mg/dL but multiple signs of chronic kidney disease. What is the key limitation of creatinine-based eGFR in this setting?

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

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

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

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

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

100. A patient with a new upper-arm AV fistula develops hand pain, pallor, and coolness that worsens during dialysis, with reduced distal pulses. What is the most likely complication?

A. Central venous stenosis, which more typically causes ipsilateral arm or breast edema and venous collaterals rather than a cool ischemic hand.

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

C. Catheter-related bacteremia, which causes fever and rigors rather than reproducible distal ischemia linked to fistula blood flow.

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

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

101. Several weeks after transplant, a recipient develops leg swelling and graft dysfunction. Imaging shows a fluid collection compressing the transplant ureter and iliac vein. What is the likely collection?

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

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

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

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

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

102. A patient with CKD has phosphate 6.2 mg/dL, elevated parathyroid hormone, and low-normal calcium. What is the central pathophysiologic process?

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

B. Renal phosphate wasting is the dominant early abnormality, so hyperphosphatemia reflects excessive dietary phosphate rather than reduced nephron excretion.

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

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

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

103. A patient with diarrhea has bicarbonate 15 mEq/L, normal anion gap, and urine ammonium excretion is appropriately high. What is the primary acid-base process?

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

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

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

D. Type 4 renal tubular acidosis, which usually occurs with hyperkalemia and reduced ammonium excretion rather than an appropriate ammonium response.

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

104. A patient 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 is used only to diagnose nephrotic syndrome and does not affect CKD prognosis once eGFR has been measured.

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

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

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

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

105. 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. What is the most likely physiology?

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

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

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

D. Pseudohyponatremia from paraproteinemia, which does not produce low measured serum osmolality or clinical volume depletion.

E. Hypertonic hyponatremia from mannitol, which raises effective serum osmolality rather than producing true hypotonicity.

106. A patient with HIV has nephrotic proteinuria and rapidly progressive CKD. Biopsy shows segmental sclerosis with collapsing glomerular tufts. Which diagnosis fits best?

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

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

C. Membranous nephropathy, which is characterized by subepithelial deposits and capillary-wall thickening rather than collapse.

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

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

107. A patient on an aminoglycoside develops nonoliguric AKI with granular casts after several days of therapy. What is the likely lesion?

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

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

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

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

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

108. A patient with panic-induced hyperventilation has $PaCO_2$ 25 mm Hg shortly after symptom onset. Which renal compensation is expected if the respiratory alkalosis persists for several days?

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

B. Renal ammonium excretion increases markedly, generating new bicarbonate and restoring PaCO2 toward normal without any change in ventilation.

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

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

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

109. A patient 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. What is the best explanation?

A. True tumor lysis syndrome, which would produce concordant hyperkalemia in plasma along with other biochemical evidence such as hyperphosphatemia.

B. Type 4 renal tubular acidosis, which causes genuine plasma hyperkalemia and does not normalize with a different specimen type.

C. Pseudohyperkalemia from potassium release during sample clotting or handling in the setting of extreme cell counts.

D. Insulin deficiency, which causes extracellular potassium shift that should remain present in both serum and plasma samples obtained correctly.

E. Digitalis toxicity, which inhibits sodium-potassium ATPase and would produce physiologic hyperkalemia rather than a specimen-dependent discrepancy.

110. A patient with hypertension uses licorice extract daily and has hypokalemia, metabolic alkalosis, low renin, and low aldosterone. What mechanism is likely?

A. Excess aldosterone production from an adrenal adenoma, which should raise aldosterone rather than suppress it.

B. Deficient epithelial sodium-channel activity, which causes salt wasting, hypotension, and hyperkalemia rather than this phenotype.

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

D. Inhibition of proximal carbonic anhydrase, which causes bicarbonaturia and metabolic acidosis rather than alkalosis.

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

111. A patient has severe AKI but stable potassium, manageable volume status, improving acidosis, and no uremic symptoms. Creatinine is 7.0 mg/dL. What is the best dialysis principle?

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

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

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

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

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

112. A patient with neurologic symptoms, thrombocytopenia, microangiopathic hemolysis, and kidney injury has severe ADAMTS13 deficiency. Which diagnosis is most likely?

A. Complement-mediated atypical HUS, which is primarily defined by severe ADAMTS13 deficiency rather than complement dysregulation.

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

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

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

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

113. A patient receiving cisplatin develops tremor and ventricular ectopy. Magnesium is 0.8 mg/dL and potassium is 3.0 mEq/L. What is the best management principle?

A. Replace potassium alone because hypomagnesemia does not affect renal potassium handling or cardiac electrical stability.

B. Give intravenous calcium as the only treatment because magnesium depletion is corrected indirectly once serum calcium concentration rises.

C. Restrict magnesium intake because low serum magnesium in cisplatin therapy usually reflects intracellular magnesium excess rather than depletion.

D. Replete magnesium and potassium while evaluating ongoing renal magnesium wasting from nephrotoxic tubular injury.

E. Use a loop diuretic to retain magnesium because loop blockade increases paracellular magnesium reabsorption in the thick ascending limb.

114. A kidney recipient on tacrolimus develops an acute rise in creatinine with high trough level and no evidence of rejection or obstruction. What hemodynamic effect can contribute?

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

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

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

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

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

115. A patient taking trimethoprim develops hyperkalemia despite stable creatinine. 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. Trimethoprim stimulates aldosterone synthesis and always lowers potassium even when kidney function is impaired.

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

116. A patient with fever, rash, eosinophilia, pyuria, and AKI after starting an antibiotic is suspected of acute interstitial nephritis. What is the first management step?

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

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

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

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

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

117. A patient has a mildly elevated urine albumin-creatinine ratio on a random sample after strenuous exercise. What is the best next step before diagnosing persistent albuminuria?

A. Start immunosuppression immediately because any albuminuria above the reference range establishes chronic glomerulonephritis.

B. Repeat the measurement, preferably with a first-morning specimen when transient confounders have resolved.

C. Order a 24-hour urine every time because spot albumin-creatinine ratios cannot be used to assess chronic kidney disease.

D. Ignore the result permanently because exercise-related albuminuria proves that future albumin measurements will be unreliable.

E. Begin dialysis planning because a single elevated albumin-creatinine ratio predicts imminent kidney failure regardless of eGFR.

118. A patient with progressive CKD develops rising PTH before marked hyperphosphatemia appears. 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. Kidney failure increases calcitriol production, raising serum calcium and directly stimulating rather than suppressing parathyroid hormone secretion.

C. FGF23 falls early in CKD, causing unopposed renal phosphate excretion and therefore chronic hypophosphatemia as the trigger for PTH release.

D. The parathyroid glands lose calcium-sensing receptors only after dialysis begins, so PTH elevation before kidney failure is unrelated to CKD physiology.

E. Secondary hyperparathyroidism requires a parathyroid adenoma and therefore cannot develop as an adaptive response to reduced nephron function.

119. A patient with high-grade lymphoma develops hyperuricemia, hyperphosphatemia, hyperkalemia, hypocalcemia, and AKI after chemotherapy. What syndrome is present?

A. Refeeding syndrome, which is characterized by hypophosphatemia rather than hyperphosphatemia after rapid cellular uptake.

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

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

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

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

120. A patient with long-standing diabetes has albuminuria, declining GFR, and biopsy showing mesangial expansion with nodular glomerulosclerosis. What is the likely diagnosis?

A. Minimal change disease, which does not produce nodular mesangial sclerosis on light microscopy.

B. Postinfectious glomerulonephritis, which more often shows diffuse proliferative injury and subepithelial humps.

C. Diabetic kidney disease with Kimmelstiel-Wilson nodular mesangial sclerosis.

D. Anti-GBM disease, which produces crescentic nephritis and linear IgG staining rather than diabetic nodules.

E. Acute uric acid nephropathy, which is a tubular crystal process rather than chronic glomerular sclerosis.

121. A patient with ADPKD has chronic flank pain but suddenly develops fever, focal renal tenderness, and inflammatory markers. Urinalysis is only mildly abnormal. What complication should be considered?

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

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

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

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

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

122. A patient with eGFR 18 mL/min/1.73 m2 requires contrast-enhanced MRI. Which principle best reflects current gadolinium safety?

A. All gadolinium agents are absolutely contraindicated at any eGFR below 60 because nephrogenic systemic fibrosis risk is identical across agents.

B. Linear high-risk gadolinium agents are preferred in advanced CKD because weaker chelation allows faster dialysis removal and less tissue deposition.

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. Give repeated high doses to overcome reduced filtration because lower GFR decreases tissue exposure and therefore reduces gadolinium-related risk.

123. A patient with chronic severe hypertension presents with hypertensive encephalopathy. Why should blood pressure not be normalized immediately?

A. Chronic hypertension shifts autoregulation, so an excessive abrupt fall can reduce cerebral, coronary, and renal perfusion.

B. High pressure must be maintained because hypertensive encephalopathy is caused by inadequate cerebral perfusion rather than autoregulatory failure.

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. Only diastolic pressure affects autoregulation, so systolic pressure can be reduced to normal immediately without ischemic risk.

124. How can chronic metabolic acidosis worsen nutritional status in CKD?

A. Acidemia stimulates sustained muscle protein synthesis, so low bicarbonate is usually associated with increased lean mass rather than catabolism.

B. Acidemia promotes muscle protein breakdown and negative nitrogen balance, contributing to weakness and protein-energy wasting.

C. Metabolic acidosis prevents amino acid release from muscle, making dietary protein restriction unnecessary in advanced chronic kidney disease.

D. Low bicarbonate causes only respiratory symptoms and has no meaningful effect on skeletal muscle metabolism or nutritional status.

E. Acidosis increases insulin sensitivity enough to prevent muscle loss, so correcting bicarbonate can paradoxically worsen protein-energy wasting.

125. A patient with hypertension and hypokalemic metabolic alkalosis has suppressed renin and suppressed aldosterone. Which disorder is most likely?

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

B. Renovascular hypertension, which typically activates renin and secondary aldosterone rather than suppressing both hormones.

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

D. Bartter syndrome, which causes salt wasting with increased renin and aldosterone rather than volume-expanded suppression of both.

E. Gitelman syndrome, which causes renal salt wasting and secondary hyperreninemic hyperaldosteronism rather than low renin and aldosterone.

126. A patient with suspected methanol ingestion has visual symptoms and severe high-anion-gap acidosis. Which toxic metabolite is primarily responsible for ocular and metabolic toxicity?

A. Oxalic acid, which is the principal toxic metabolite of ethylene glycol rather than methanol.

B. Formic acid produced after alcohol dehydrogenase metabolism of methanol.

C. Acetone, which is produced in isopropanol metabolism and generally does not cause a high-anion-gap acidosis.

D. Uric acid, which causes tumor lysis nephropathy but is not the toxic metabolite of methanol.

E. Lactic acid generated directly from methanol without metabolism, making alcohol dehydrogenase blockade ineffective.

127. A 33-year-old has episodic headache, palpitations, diaphoresis, and paroxysmal severe hypertension. What is the preferred initial biochemical evaluation?

A. Measure serum aldosterone alone because episodic adrenergic spells are produced by mineralocorticoid excess rather than catecholamines.

B. Measure plasma free or urinary fractionated metanephrines to evaluate for catecholamine-secreting tumor.

C. Obtain a dexamethasone suppression test only because pheochromocytoma is excluded by normal cortisol regulation.

D. Measure renin after intense sodium loading because catecholamine tumors are diagnosed from suppressed renin activity.

E. Perform renal angiography first because episodic sweating and palpitations localize the lesion to the renal arteries.

128. A patient with CKD starts an SGLT2 inhibitor and eGFR falls modestly during the first two weeks while blood pressure and volume status remain stable. What is the best interpretation?

A. A small early hemodynamic dip in eGFR can be expected and does not by itself require stopping therapy if the patient is otherwise stable.

B. Any fall in eGFR after an SGLT2 inhibitor proves acute tubular necrosis and requires permanent discontinuation of the drug.

C. The decline means the drug has caused bilateral renal artery thrombosis because SGLT2 inhibition should immediately increase measured GFR.

D. The fall reflects progressive diabetic nephropathy regardless of diabetes status and is unrelated to changes in tubuloglomerular feedback.

E. The only safe response is aggressive intravenous fluid administration until eGFR returns above the pretreatment value, even without hypovolemia.

129. A transplant recipient develops unexplained rising creatinine. Ultrasound excludes obstruction and drug levels are not clearly toxic. 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. Urine sodium alone, because fractional sodium excretion reliably separates acute rejection from BK infection and drug toxicity.

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

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

130. A patient with vomiting and poor intake has AKI, orthostatic hypotension, bland urine sediment, and rapid improvement after isotonic fluid. What category of AKI is most likely?

A. Acute tubular necrosis is proven because any creatinine rise during volume depletion represents structural tubular cell death.

B. Acute glomerulonephritis is most likely because volume depletion characteristically causes red-cell casts and complement consumption.

C. Postrenal obstruction is most likely because vomiting directly compresses both ureters and causes hydronephrosis.

D. Renal cortical necrosis is expected because brief orthostasis causes irreversible ischemic destruction of the cortex.

E. Prerenal AKI from reduced effective renal perfusion without established structural tubular injury.

131. A patient with AKI becomes progressively confused, develops myoclonus, and has no other metabolic or neurologic explanation. What kidney-related complication should be considered?

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

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

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

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

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

132. A 68-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. What is the most likely cause of the hyponatremia?

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

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

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

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

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

133. A patient taking ibuprofen daily for arthritis develops worsening blood pressure and edema despite previously stable therapy. What is the best explanation?

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

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

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

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

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

134. A 44-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. Which inherited category is most likely?

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

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

C. Alport syndrome, which typically features persistent glomerular hematuria with hearing or ocular abnormalities rather than a bland sediment.

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

E. Thin basement membrane disease, which usually causes isolated microscopic hematuria rather than early gout with progressive tubulointerstitial fibrosis.

135. A patient with sickle cell disease develops gross hematuria and sloughed papillary tissue. Which lesion is most likely?

A. Renal papillary necrosis from medullary ischemia in the hypoxic, hypertonic renal medulla.

B. Membranous nephropathy, because sickling selectively deposits immune complexes under podocytes and causes papillary sloughing.

C. Renal cortical necrosis, which is confined to the outer cortex and typically follows obstetric catastrophe rather than chronic sickling.

D. Acute uric acid nephropathy, which causes crystal obstruction but does not characteristically produce sloughed papillae.

E. Polycystic kidney rupture, because sickle cell disease routinely produces acquired bilateral renal cysts that bleed into the collecting system.

136. A patient with septic shock has lactate 8 mmol/L, bicarbonate 13 mEq/L, and an increased anion gap. What 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. Restore tissue perfusion and treat the underlying cause of shock rather than attempting to normalize lactate with bicarbonate alone.

C. Restrict oxygen delivery because high inspired oxygen worsens anaerobic metabolism and therefore increases lactate production in shock.

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.

137. A young man has persistent microscopic hematuria, progressive kidney disease, sensorineural hearing loss, and ocular abnormalities. Which diagnosis is most likely?

A. Thin basement membrane nephropathy alone, which usually lacks progressive kidney failure, hearing loss, and characteristic ocular disease.

B. Minimal change disease, which causes nephrotic proteinuria and has no collagen-related hearing manifestations.

C. Alport syndrome from a type IV collagen basement-membrane disorder with renal and extrarenal manifestations.

D. Acute interstitial nephritis, which is acquired and does not produce inherited hearing and ocular abnormalities.

E. Renal papillary necrosis, which may cause hematuria but does not cause a hereditary sensorineural hearing syndrome.

138. A 63-year-old with bilateral renal artery stenosis begins an ACE inhibitor and creatinine rises markedly within several days. What mechanism best explains the fall in GFR?

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

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

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

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

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

139. A patient with advanced uremia has persistent mucosal bleeding despite a normal platelet count and coagulation studies. Which short-term therapy can improve platelet function before an urgent procedure?

A. Warfarin should be started because uremic bleeding is caused by excess vitamin K-dependent clotting factor activity rather than platelet dysfunction.

B. Desmopressin can transiently improve uremic platelet dysfunction while definitive management includes dialysis and correction of anemia when appropriate.

C. Aspirin should be given because inhibiting platelet cyclooxygenase restores adhesion when uremic toxins interfere with von Willebrand factor binding.

D. Recombinant erythropoietin acts within minutes and is the preferred sole emergency treatment for active uremic mucosal bleeding.

E. Protamine is first-line therapy even without heparin exposure because uremia causes endogenous heparin accumulation in all patients with kidney failure.

140. A pregnant patient at 29 weeks has new hypertension, proteinuria, headache, and elevated liver enzymes. Which diagnosis best integrates the findings?

A. White-coat hypertension, because proteinuria and liver abnormalities are expected consequences of anxiety during pregnancy.

B. Primary aldosteronism, because pregnancy-related endothelial disease does not cause proteinuria or neurologic symptoms.

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 rather than treatment as chronic essential hypertension alone.

141. Why might a non-calcium phosphate binder be preferred in a dialysis patient with hyperphosphatemia and extensive vascular calcification?

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

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

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

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

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

142. A patient with CKD stage 4 has persistent bicarbonate 17 mEq/L without respiratory disease. Why is chronic metabolic acidosis clinically important?

A. Metabolic acidosis is protective in CKD because chronic acid retention suppresses protein breakdown and prevents bone mineral loss.

B. Low bicarbonate in CKD reflects only respiratory compensation and therefore should not influence nutrition, bone health, or kidney management.

C. Acidosis eliminates hyperkalemia risk by forcing potassium permanently into cells, making potassium monitoring unnecessary in advanced CKD.

D. It can contribute to bone buffering, muscle catabolism, and progression of kidney disease, so reversible causes and alkali therapy should be considered.

E. Bicarbonate therapy is never used in CKD because any alkali administration predictably causes immediate pulmonary edema regardless of volume status.

143. A patient taking an ACE inhibitor and diuretic develops AKI shortly after adding high-dose ibuprofen. 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. Ibuprofen dilates the afferent arteriole, while ACE inhibition constricts the efferent arteriole, producing excessive glomerular hyperfiltration.

C. The three drugs precipitate nephrotic syndrome by causing immediate podocyte foot-process fusion in nearly all patients.

D. NSAIDs increase prostaglandin production and therefore amplify diuretic natriuresis until renal perfusion becomes excessive.

E. ACE inhibitors and NSAIDs together block tubular creatinine secretion without altering true GFR, so the AKI is always laboratory artifact.

144. Why is routine combination therapy with an ACE inhibitor and an ARB generally avoided in CKD?

A. The two drugs have identical molecular structures, so combining them causes allergic cross-linking rather than any hemodynamic or electrolyte effect.

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

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

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

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

145. A 54-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. Which diagnosis best fits?

A. Syndrome of inappropriate antidiuretic hormone secretion causing inappropriately concentrated urine despite hypotonicity.

B. Primary adrenal insufficiency, which can mimic SIADH but should be supported by cortisol deficiency and often hyperkalemia.

C. Psychogenic polydipsia, in which suppression of vasopressin usually produces urine osmolality below about 100 mOsm/kg.

D. Cirrhosis with arterial underfilling, which generally presents with clinical volume expansion and avid sodium retention.

E. Renal salt wasting from osmotic diuresis, which requires an ongoing filtered osmole load and usually a different volume picture.

146. A kidney transplant recipient taking mycophenolate plans pregnancy. 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. Tacrolimus and every calcineurin inhibitor are absolutely incompatible with pregnancy, making graft nephrectomy necessary before conception.

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

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

147. Minutes after reperfusion of a kidney allograft, the graft becomes mottled and produces no urine. Pretransplant testing reveals preformed donor-specific antibody. What process is most likely?

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

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

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

D. Chronic calcineurin inhibitor toxicity, which evolves over months to years rather than minutes after transplantation.

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

148. A patient with edema, hypertension, rising creatinine, hematuria, and red-cell casts has which general renal syndrome?

A. An isolated tubular concentrating defect, which causes polyuria without glomerular hematuria or red-cell casts.

B. A pure nephrotic syndrome, which is dominated by heavy proteinuria and hypoalbuminemia rather than an inflammatory sediment.

C. Postrenal obstruction, which may cause AKI but does not characteristically generate dysmorphic red cells and red-cell casts.

D. An acute nephritic syndrome caused by inflammatory glomerular injury with an active urine sediment.

E. Prerenal azotemia, which usually has a bland sediment and lacks glomerular blood and casts.

149. A patient on chronic lithium develops polyuria with low urine osmolality despite hypernatremia. Which nephron effect is most relevant?

A. Lithium blocks aquaporin release from the posterior pituitary, producing central rather than nephrogenic diabetes insipidus.

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

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

150. A hospitalized patient develops anemia, thrombocytopenia, schistocytes, elevated LDH, and AKI. Which renal vascular syndrome should be considered first?

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

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

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

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

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

151. A patient with CKD and diabetes develops vomiting and poor oral intake while taking an SGLT2 inhibitor and diuretic. What medication-safety principle is most appropriate?

A. Continue every chronic medication unchanged because acute illness never alters the risk-benefit balance of SGLT2 inhibitors, diuretics, or RAS blockade.

B. Double the SGLT2 inhibitor dose because reduced oral intake increases the need for glucosuria to prevent stress hyperglycemia and kidney injury.

C. Add an NSAID to preserve GFR during dehydration because prostaglandin inhibition improves afferent arteriolar blood flow in volume depletion.

D. Stop all medications permanently after any intercurrent illness because resuming kidney-protective therapy after recovery is unsafe in chronic kidney disease.

E. Temporarily reassess medications that can worsen volume depletion or ketoacidosis risk during acute illness and restart them when clinically stable.

152. A patient with mild lifelong hypercalcemia has a family history of similar findings, normal renal function, and very low fractional urinary calcium excretion. Which diagnosis is most likely?

A. Familial hypocalciuric hypercalcemia from altered calcium-sensing receptor function and reduced urinary calcium excretion.

B. Primary hyperparathyroidism, which usually produces greater urinary calcium excretion and is more likely to cause kidney stones or skeletal complications.

C. Sarcoidosis, which increases calcitriol production and typically suppresses PTH rather than producing a familial lifelong low-calcium-excretion pattern.

D. Multiple myeloma, which causes acquired hypercalcemia with other systemic findings rather than stable familial hypercalcemia from childhood onward.

E. Milk-alkali syndrome, which is an acquired disorder linked to calcium and alkali intake and does not produce inherited hypocalciuria.

153. Several days after an angiographic procedure, a patient develops AKI, livedo reticularis, eosinophilia, and low complement. What is the most likely diagnosis?

A. Contrast-associated tubular injury alone, which does not typically cause livedo, eosinophilia, or systemic cholesterol emboli.

B. Anti-GBM disease, which causes pulmonary-renal findings and linear IgG rather than livedo after arterial manipulation.

C. Minimal change disease, which causes nephrotic syndrome without embolic skin findings.

D. Renal-vein thrombosis, which does not produce distal livedo from showering arterial cholesterol crystals.

E. Cholesterol crystal atheroembolic kidney disease from showering arterial plaque debris after vascular instrumentation.

154. A patient with severe diarrhea has normal-gap acidosis. The urine anion gap is strongly negative. What does this finding imply?

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

B. Urine contains no chloride, proving that the kidneys are retaining acid rather than excreting ammonium chloride during diarrhea.

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

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

E. Urinary ammonium excretion is appropriately increased, supporting an extrarenal bicarbonate loss rather than renal acidification failure.

155. A patient with resistant hypertension and hyperkalemia has CKD and is taking an ACE inhibitor. Which step should precede automatically abandoning renin-angiotensin blockade?

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

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

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

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

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

156. Why are avoidable red-cell transfusions particularly undesirable in a kidney-transplant candidate with CKD anemia?

A. Transfusions permanently suppress endogenous erythropoietin production, so the patient can never respond to an ESA after receiving donor red cells.

B. Transfusions always cause acute antibody-mediated rejection even before transplantation because donor erythrocytes contain a functioning kidney graft.

C. Transfused red cells are rapidly filtered into urine in CKD, causing iron nephrotoxicity and accelerating progression to kidney failure.

D. Transfusions can expose the patient to HLA antigens and increase sensitization, potentially making compatible transplantation more difficult.

E. Blood transfusion eliminates the possibility of ABO-compatible transplantation because the recipient's inherited blood group changes after donor exposure.

157. A patient with severe AKI after iodinated contrast has no obstruction and urine sediment is consistent with tubular injury. What is the most appropriate general management?

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

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

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

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

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

158. A patient has recurrent complement-mediated thrombotic microangiopathy without severe ADAMTS13 deficiency or Shiga toxin. Which pathway is implicated?

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

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

C. Autoantibodies to PLA2R, which cause thrombotic microangiopathy rather than membranous nephropathy.

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

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

159. A patient with hyponatremia has low serum osmolality, urine osmolality 80 mOsm/kg, and a diet consisting mainly of beer with little protein or salt. 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. Glomerular filtration must be severely reduced, because normal kidneys can excrete unlimited water regardless of dietary osmole intake.

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

E. Severe hyperglycemia shifts water intracellularly, and the low urine osmolality reflects glucose-driven osmotic diuresis rather than low solute.

160. A patient after Roux-en-Y gastric bypass develops recurrent calcium oxalate stones and high urinary oxalate. What mechanism is most likely?

A. Bile acid depletion causes the kidney to synthesize oxalate directly, so intestinal absorption plays little role after bariatric surgery.

B. Gastric bypass increases urinary citrate markedly, and citrate itself is converted into oxalate in the collecting duct during alkaline urine formation.

C. Reduced intestinal sodium absorption lowers serum calcium and causes secondary cystinuria, leading to mixed cystine-calcium oxalate stones.

D. The procedure raises urine pH enough to precipitate uric acid, and uric acid crystals then transform chemically into calcium oxalate.

E. Fat malabsorption binds intestinal calcium, leaving more free oxalate available for colonic absorption and urinary excretion.

161. An older patient with isolated systolic hypertension has a wide pulse pressure and stiff arteries. 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. Loss of venous capacitance alone determines isolated systolic hypertension without any contribution from large-artery stiffness.

D. Decreased stroke volume is the defining cause because less ejected blood always produces a larger arterial pulse pressure.

E. Reduced systemic vascular resistance from generalized arteriolar dilation is sufficient to create persistent isolated systolic hypertension.

162. A mechanically ventilated patient with massive fluid resuscitation develops tense abdominal distention, oliguria, rising airway pressures, and elevated bladder pressure. What should be suspected?

A. SIADH, because high bladder pressure directly stimulates vasopressin and produces oliguria with a tense abdomen.

B. Acute glomerulonephritis, because glomerular inflammation is the usual cause of increased airway pressure after fluid resuscitation.

C. Nephrogenic diabetes insipidus, because elevated abdominal pressure causes polyuria before renal perfusion falls.

D. Abdominal compartment syndrome causing reduced renal perfusion and venous outflow from markedly increased intra-abdominal pressure.

E. Renal papillary necrosis, because papillary sloughing produces a rigid abdomen and elevated plateau pressures.

163. A patient with systemic lupus erythematosus has active urine sediment, rising creatinine, proteinuria, and low complement. What is the best next diagnostic step?

A. Kidney biopsy to classify lupus nephritis and guide prognosis and treatment when no contraindication exists.

B. Assume minimal change disease and begin therapy without tissue because lupus serology cannot be associated with multiple renal lesions.

C. Measure urine sodium alone because fractional sodium excretion establishes the histologic class of lupus nephritis.

D. Delay evaluation until creatinine doubles again because kidney biopsy is useful only after advanced irreversible kidney failure develops.

E. Diagnose chronic pyelonephritis because low complement and active sediment are specific for tubulointerstitial infection in lupus.

164. A patient with acute decompensated heart failure has pulmonary edema, venous congestion, and worsening creatinine. 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. Venous congestion increases the renal perfusion gradient and therefore always raises GFR despite heart failure.

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

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

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

165. A kidney recipient is receiving standard opportunistic-infection prophylaxis. Which drug is commonly used to prevent Pneumocystis jirovecii pneumonia when tolerated?

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

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

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

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

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

166. A patient with cerebral edema and severe AKI requires renal replacement therapy. Why might slower continuous therapy be preferred?

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

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

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

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

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

167. A patient with very high blood urea nitrogen begins hemodialysis for the first time and develops headache, nausea, confusion, and seizures during treatment. What is the likely diagnosis?

A. Uremic pericarditis, which presents primarily with positional chest pain or a friction rub rather than acute neurologic deterioration during dialysis.

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

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

D. Calciphylaxis, which produces painful ischemic skin lesions rather than acute headache and seizures during a first hemodialysis session.

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

168. A patient with hypertension and episodic muscle weakness has potassium 2.7 mEq/L and bicarbonate 34 mEq/L. Renin is low and aldosterone is high. Which mechanism best fits?

A. Renal salt wasting activates neither renin nor aldosterone and therefore produces hypertension with low potassium and alkalosis.

B. Loss of ENaC function causes sodium retention and suppressed renin while increasing potassium secretion in principal cells.

C. Hypoaldosteronism increases distal potassium secretion and generates metabolic alkalosis despite reduced sodium reabsorption.

D. Autonomous mineralocorticoid activity increases distal sodium reabsorption and potassium and hydrogen secretion.

E. Carbonic anhydrase inhibition increases bicarbonate retention and suppresses potassium excretion, causing this low-renin phenotype.

169. A child develops abrupt edema and nephrotic-range proteinuria after a viral illness. Complement is normal and kidney function is preserved. What lesion is most likely?

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

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

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

D. Acute tubular necrosis, which produces granular casts and AKI rather than selective nephrotic protein loss.

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

170. A hospitalized patient with sudden anuria has a distended bladder. 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. Perform kidney biopsy immediately because anuria is almost always caused by rapidly progressive glomerulonephritis.

C. Give hypertonic saline because bladder distention reflects severe hyponatremia rather than mechanical urinary retention.

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

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

171. A patient with Sjögren syndrome has a normal-anion-gap metabolic acidosis, urine pH 6.2 during systemic acidosis, and recurrent calcium phosphate stones. What tubular disorder is present?

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

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

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

D. Primary hyperaldosteronism, which promotes hydrogen secretion and metabolic alkalosis rather than systemic acidosis.

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

172. A patient with diabetes and CKD has potassium 5.9 mEq/L, bicarbonate 17 mEq/L, a normal anion gap, and low renin and aldosterone activity. Which diagnosis best fits?

A. Type 4 renal tubular acidosis from hyporeninemic hypoaldosteronism with impaired potassium and ammonium excretion.

B. Distal type 1 renal tubular acidosis, which more often causes hypokalemia and an inappropriately high urine pH during systemic acidosis.

C. Proximal type 2 renal tubular acidosis, which causes bicarbonate wasting but usually not persistent hyperkalemia from aldosterone deficiency.

D. Lactic acidosis, which would generally raise the anion gap rather than produce an isolated hyperchloremic acidosis.

E. Respiratory acidosis, which raises bicarbonate through renal compensation rather than causing a primary low-bicarbonate hyperchloremic state.

173. A patient with chronic SIADH has sodium 118 mEq/L and a generalized seizure. 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. Start oral urea as the only emergency therapy because gradual osmole loading is preferred even during active hyponatremic seizures.

D. Give hypertonic saline in controlled boluses to achieve a prompt small rise in serum sodium while monitoring closely.

E. Administer desmopressin alone to lower urine output before any sodium-raising therapy because neurologic symptoms do not alter urgency.

174. A patient with chronic toluene exposure has weakness, hypokalemia, and a normal-anion-gap metabolic acidosis with a high urinary potassium loss. Which mechanism is most likely?

A. Renal loss of potassium and ammonium salts during excretion of organic acid metabolites can produce hypokalemic hyperchloremic acidosis.

B. Toluene causes isolated respiratory acidosis by depressing ventilation, so the low bicarbonate reflects compensation rather than a metabolic process.

C. Toluene directly blocks aldosterone receptors, producing the hyperkalemic type 4 renal tubular acidosis pattern rather than potassium wasting.

D. The solvent causes pseudohypokalemia by interfering with ion-selective electrodes, while whole-body potassium stores remain unchanged.

E. Toluene generates a persistent pure metabolic alkalosis because hippurate excretion obligately retains bicarbonate in the extracellular space.

175. A patient with advanced CKD on dialysis develops multiple acquired renal cysts over years of therapy. Why is surveillance attention sometimes warranted?

A. The cysts inevitably transform into urothelial carcinoma, so prophylactic bilateral nephrectomy is required in every dialysis patient.

B. Acquired cystic disease reverses kidney failure by increasing nephron number, so the main concern is unexpected recovery of GFR.

C. The cysts indicate unrecognized ADPKD in every case and therefore require intracranial aneurysm screening for all dialysis patients.

D. The only complication is severe nephrotic syndrome because acquired cysts uniformly disrupt the glomerular filtration barrier.

E. Acquired cystic kidney disease is associated with an increased risk of renal cell carcinoma compared with the general population.

176. A stable thrice-weekly hemodialysis patient has inadequate small-solute clearance because treatment sessions are repeatedly shortened. Which concept is most relevant to assessing dialysis dose?

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

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

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

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

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

177. A patient has severe asymptomatic hypertension discovered during a routine visit but no neurologic, cardiac, retinal, or renal evidence of acute injury. What is the general management principle?

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

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

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

D. Confirm the pressure and intensify oral therapy with close follow-up rather than using rapid intravenous normalization solely because the number is high.

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

178. A patient whose native kidney disease was IgA nephropathy asks whether the disease can return after transplantation. 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 always occurs within the first 24 hours and causes immediate irreversible graft thrombosis.

C. Recurrence is prevented completely by any standard maintenance immunosuppressive regimen.

D. IgA recurrence affects only the native kidneys and cannot involve an allograft under any circumstances.

E. IgA nephropathy can recur in the allograft, although recurrence severity and effect on graft survival vary.

179. A patient has 5.8 g/day proteinuria, albumin 2.1 g/dL, edema, and hyperlipidemia. Which syndrome is present?

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

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

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

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

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

180. A patient 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. What explains the discrepancy?

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

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

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

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

E. Primary polydipsia, which produces genuine hypotonic hyponatremia rather than a method-dependent laboratory artifact.

181. A patient with hypertension develops ankle edema on a dihydropyridine calcium-channel blocker but has clear lungs and stable weight. What mechanism is most likely?

A. The drug causes severe venoconstriction and lowers capillary hydrostatic pressure, forcing fluid into the interstitium.

B. Calcium-channel blockade always causes acute nephrotic-range proteinuria, and the edema therefore proves new glomerular disease.

C. The edema results from complete inhibition of aldosterone synthesis and renal sodium wasting, leading to tissue fluid accumulation.

D. Dihydropyridines directly increase lymphatic permeability by causing inflammatory capillary destruction in most treated patients.

E. Preferential arteriolar dilation raises capillary hydrostatic pressure and can cause dependent edema without generalized sodium overload.

182. A transplant recipient has slowly declining graft function years after surgery, proteinuria, transplant glomerulopathy, and persistent donor-specific antibodies. What process should be considered?

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

B. Hyperacute rejection, which occurs at reperfusion rather than years later.

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

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

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

183. A patient has proliferative glomerulonephritis and persistently depressed C3 without evidence of immunoglobulin deposition. Which category should be considered?

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

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

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

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

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

184. A patient with CKD has rising phosphate and PTH values over several visits. How should CKD-mineral bone disorder generally be managed?

A. Normalize each value immediately after one abnormal result because CKD-MBD treatment is based on isolated measurements rather than longitudinal patterns.

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

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

D. Interpret calcium, phosphate, and PTH together and follow trends rather than reacting to a single isolated laboratory value.

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

185. A patient starts a proton-pump inhibitor and develops AKI with sterile pyuria and white-cell casts. What drug-associated lesion should be suspected?

A. Renal cortical necrosis, because proton-pump inhibitors routinely cause diffuse cortical ischemia through arterial thrombosis.

B. Membranous nephropathy, because the typical renal toxicity is subepithelial immune-complex deposition with nephrotic syndrome.

C. Acute interstitial nephritis from a drug-triggered hypersensitivity-type interstitial inflammatory reaction.

D. Acute uric acid nephropathy, because gastric acid suppression causes urate crystals to precipitate throughout the tubules.

E. Minimal change disease, because white-cell casts are a defining feature of isolated podocyte injury.

186. A patient with CKD and anemia has hemoglobin 9.4 g/dL. Before attributing the anemia entirely to erythropoietin deficiency, what evaluation is essential?

A. Start an erythropoiesis-stimulating agent immediately without iron studies because all anemia below 10 g/dL in CKD is caused solely by erythropoietin deficiency.

B. Perform bone marrow biopsy in every patient because peripheral blood and iron testing cannot identify treatable causes of anemia in chronic kidney disease.

C. Give repeated red-cell transfusions before laboratory evaluation because transfusion is the safest first-line correction for any asymptomatic CKD anemia.

D. Ignore the anemia until dialysis begins because anemia complications do not occur and treatment has no role during nondialysis chronic kidney disease.

E. Assess iron status and other reversible causes of anemia such as blood loss, inflammation, nutritional deficiencies, and hemolysis when indicated.

187. A patient with subarachnoid hemorrhage develops sodium 125 mEq/L, high urine sodium, concentrated urine, hypotension, and a negative fluid balance. Which diagnosis is favored over SIADH?

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

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

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

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.

188. An older man with prostate enlargement develops anuria and bilateral hydronephrosis. Which category of AKI is present?

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

B. Intrinsic glomerular AKI, because hydronephrosis indicates inflammatory capillary-wall injury rather than increased collecting-system pressure.

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

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

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

189. A healthy young adult has lifelong microscopic hematuria, normal kidney function, little proteinuria, and a family history of similar benign hematuria. What disorder is most likely?

A. Rapidly progressive ANCA glomerulonephritis, which would cause active inflammation and declining kidney function rather than stable isolated hematuria.

B. Membranous nephropathy, which is a nephrotic proteinuric disease rather than a typical cause of lifelong isolated hematuria.

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

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

E. Renal-vein thrombosis, which causes an acquired vascular event rather than inherited glomerular hematuria.

190. A postpartum patient after catastrophic hemorrhage and disseminated intravascular coagulation remains anuric. Imaging suggests diffuse cortical nonperfusion. What lesion is most concerning?

A. Minimal change disease, because obstetric hemorrhage causes selective podocyte effacement and abrupt anuria.

B. Poststreptococcal glomerulonephritis, because postpartum DIC is a typical nephritogenic streptococcal trigger.

C. Renal cortical necrosis from severe prolonged ischemic and microvascular injury.

D. Nephrogenic diabetes insipidus, because cortical ischemia causes inability to concentrate urine and therefore profound polyuria rather than anuria.

E. Uncomplicated prerenal azotemia, because persistent anuria with absent cortical perfusion indicates preserved renal parenchyma.

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

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

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

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

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

192. A patient with oliguric AKI has severe pulmonary edema and hypoxemia despite appropriate diuretic therapy. What is the most appropriate next step?

A. Initiate kidney replacement therapy for refractory volume overload with respiratory compromise.

B. Continue escalating fluid administration because pulmonary edema in AKI usually reflects intravascular depletion rather than excess volume.

C. Defer for creatinine to exceed a fixed numeric threshold because dialysis cannot be started for volume indications alone.

D. Give an NSAID to reduce renal prostaglandins because afferent vasoconstriction improves pulmonary capillary fluid clearance.

E. Use hypertonic saline as definitive treatment because increasing serum osmolality draws lung water directly into the circulation for renal excretion.

193. A stable tacrolimus-treated recipient starts voriconazole and soon develops tremor, hyperkalemia, and rising creatinine. What explains the change?

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

B. Voriconazole induces CYP3A and therefore causes tacrolimus underexposure and rejection rather than toxicity.

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

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

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

194. A patient with AKI is taking a loop diuretic. Why can fractional excretion of sodium be misleading?

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

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

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

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

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

195. A patient with chronic hypertension has an arm pressure of 150/88 mm Hg and ankle pressure of 210/96 mm Hg with diminished femoral pulses. Which diagnosis should be considered?

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

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

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

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

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

196. A patient with CKD has serum bicarbonate 18 mEq/L and is also volume overloaded with uncontrolled hypertension. What is an important consideration before prescribing sodium bicarbonate?

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

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

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

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

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

197. A long-term peritoneal dialysis patient develops recurrent bowel obstruction, weight loss, ultrafiltration failure, and a thickened calcified peritoneal membrane encasing bowel loops. What is the diagnosis?

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

B. Dialysis disequilibrium syndrome, which is an acute cerebral osmotic disorder of hemodialysis rather than a structural peritoneal complication.

C. Nephrogenic systemic fibrosis, which is associated with certain gadolinium exposures and causes systemic skin fibrosis rather than bowel encapsulation.

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

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

198. A patient 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. What syndrome should be considered?

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

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

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

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

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

199. A stable kidney recipient asks whether a live attenuated vaccine can be given routinely while receiving maintenance immunosuppression. What is the general principle?

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

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

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

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

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

200. A patient with psychogenic polydipsia has sodium 128 mEq/L. Which urine finding is most expected before any treatment?

A. Urine osmolality above 500 mOsm/kg because excessive water intake directly stimulates vasopressin release in otherwise healthy kidneys.

B. Urine sodium consistently below 10 mEq/L because primary polydipsia necessarily activates intense renin-angiotensin signaling.

C. Urine containing broad granular casts because water excess produces acute tubular epithelial injury before hyponatremia develops.

D. Very dilute urine with osmolality near the physiologic minimum because vasopressin is appropriately suppressed.

E. Urine osmolality fixed near plasma osmolality because psychogenic polydipsia causes an acquired inability to dilute or concentrate urine.

201. A patient with diabetic ketoacidosis has bicarbonate 8 mEq/L and PaCO2 20 mm Hg. Which interpretation is most accurate?

A. There is a superimposed respiratory acidosis because any PaCO2 below 40 mm Hg is inadequate compensation for metabolic acidosis.

B. There is a primary respiratory alkalosis without metabolic acidosis because the low PaCO2 fully explains the low serum bicarbonate.

C. There is a primary metabolic alkalosis because bicarbonate below normal indicates renal retention of acid rather than systemic acid gain.

D. The values cannot be interpreted without urine chloride because serum bicarbonate and PaCO2 do not permit assessment of respiratory compensation.

E. The respiratory compensation is appropriate for a primary high-anion-gap metabolic acidosis by Winter's relationship.

202. A patient with severe ethylene glycol poisoning has high-anion-gap acidosis, an elevated osmolar gap, and calcium oxalate crystals. Which therapy is appropriate in severe poisoning?

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

B. Use sodium chloride alone because ethylene glycol toxicity is caused by volume depletion rather than toxic metabolites.

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

D. Start high-dose glucocorticoids because the renal crystals are immune complexes rather than calcium oxalate.

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

203. A kidney recipient develops rising creatinine two weeks after transplant. Biopsy shows interstitial inflammation and tubulitis without dominant antibody-mediated features. What is the diagnosis?

A. Hyperacute rejection, which presents immediately after reperfusion with severe vascular injury.

B. BK nephropathy, which commonly shows viral cytopathic changes and usually occurs later during sustained immunosuppression.

C. Chronic antibody-mediated rejection, which is characterized by chronic microvascular and basement-membrane remodeling rather than acute tubulitis alone.

D. Recurrent ADPKD, which cannot recur in a normal donor kidney because the recipient's germline cyst mutation is not present in the graft.

E. Acute T-cell-mediated allograft rejection characterized by interstitial inflammation and tubulitis.

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

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

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

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

205. A child has bloody diarrhea followed by microangiopathic hemolysis, thrombocytopenia, and AKI. Which syndrome is most likely?

A. Shiga toxin–associated hemolytic uremic syndrome producing a diarrheal thrombotic microangiopathy.

B. TTP from severe ADAMTS13 deficiency, which is less typical after a diarrheal prodrome in a child.

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. Minimal change disease, which causes nephrotic syndrome without a thrombotic microangiopathy.

206. A patient with CKD has rapidly worsening edema and rising albuminuria despite stable eGFR. Why should this change trigger reassessment?

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

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

C. Edema proves the patient has heart failure rather than kidney disease, so further renal evaluation is unnecessary when eGFR is unchanged.

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

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

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

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

D. Mineralocorticoid deficiency causes avid renal sodium retention, so the low sodium results from sodium moving into cells rather than urinary loss.

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

208. A patient with malignant hypertension has AKI, retinal hemorrhages, and arteriolar onion-skin thickening. 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 rather than concentric smooth-muscle and basement-membrane thickening.

C. Hyperplastic arteriolosclerosis from severe hypertension-mediated vascular injury.

D. Fibrinoid glomerular crescents, which describe extracapillary proliferation rather than the arteriolar lesion.

E. Cholesterol clefts, which indicate atheroembolization rather than pressure-mediated onion-skin narrowing.

209. A 39-year-old with hypertension and spontaneous hypokalemia has suppressed renin. Which screening test is most appropriate for primary aldosteronism?

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

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

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

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

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

210. A dialysis patient has very high PTH, bone pain, subperiosteal resorption, and elevated alkaline phosphatase. Which renal osteodystrophy pattern is most likely?

A. Adynamic bone disease, which is characterized by suppressed remodeling and typically low rather than very high PTH and bone turnover.

B. Osteoporosis alone, which can reduce bone mass but does not specifically explain marked PTH elevation and subperiosteal resorption.

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

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

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

211. A patient with progressive CKD is likely to need kidney replacement therapy within the next year. What is the best planning approach?

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

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

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

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

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

212. A patient with CKD and heavy albuminuria needs antihypertensive therapy. 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. Dual ACE inhibitor plus ARB therapy because complete renin-angiotensin blockade improves outcomes without added kidney or potassium risk.

D. An ACE inhibitor or angiotensin receptor blocker because lowering intraglomerular pressure and albuminuria can slow progression.

E. Chronic NSAID therapy because afferent vasoconstriction reduces albuminuria while preserving long-term kidney function.

213. A kidney recipient develops fever, weight loss, lymphadenopathy, and an EBV-associated B-cell proliferation. What diagnosis is most likely?

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

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

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

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

E. Recurrent FSGS, which presents with proteinuria rather than systemic lymphadenopathy and EBV-associated proliferation.

214. Urine microscopy shows numerous dysmorphic erythrocytes and acanthocytes in a patient with hematuria. What do these findings suggest?

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

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

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

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

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

215. A patient on CRRT with regional citrate anticoagulation develops rising total calcium, falling ionized calcium, and increasing calcium infusion needs. What complication should be suspected?

A. Heparin-induced thrombocytopenia, because citrate directly forms antibodies against platelet factor 4 and causes calcium chelation.

B. Citrate accumulation with impaired citrate metabolism and systemic calcium chelation.

C. Hyperparathyroidism, because PTH suppresses ionized calcium only during CRRT and raises total calcium through citrate binding.

D. Dialysis disequilibrium, because rapid urea removal selectively increases the total-to-ionized calcium ratio.

E. Acute phosphate nephropathy, because phosphate crystals within the filter consume ionized calcium while increasing serum total calcium.

216. 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. ESAs become ineffective once hemoglobin exceeds 10 g/dL because erythropoietin receptors disappear from erythroid precursors at that concentration.

B. Normal hemoglobin always causes severe iron overload in CKD because each new red cell releases iron rather than incorporating it into hemoglobin.

C. A normal hemoglobin worsens kidney filtration directly by increasing tubular oxygen tension, so anemia must be maintained to preserve nephron function.

D. Higher hemoglobin targets with ESAs can increase cardiovascular and thrombotic risk, so treatment balances symptoms, transfusion avoidance, and safety.

E. The only reason to limit ESA therapy is cost; clinical outcome studies show no difference in cardiovascular events across hemoglobin targets.

217. A patient with severe hyperkalemia receives insulin and glucose. What is the purpose of this therapy?

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

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

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

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

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

218. A hospitalized patient with sodium 130 mEq/L has measured serum osmolality 306 mOsm/kg and glucose 620 mg/dL. What type of hyponatremia is present?

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

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

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

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

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

219. A patient with AKI of unclear cause has no urinary symptoms. Which imaging test is commonly used to evaluate for hydronephrosis?

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

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

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

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

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

220. A 46-year-old has hypertension despite three medications, including a diuretic. Before labeling the condition truly resistant, what should be done?

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

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

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

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

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

221. A long-term kidney transplant recipient asks about cancer prevention. Which malignancy risk is particularly increased by chronic immunosuppression and sun exposure?

A. Retinoblastoma, because adult transplant immunosuppression reactivates congenital retinal tumors in most recipients.

B. Cutaneous squamous-cell carcinoma and other nonmelanoma skin cancers promoted by chronic immunosuppression and UV exposure.

C. Osteosarcoma, because calcineurin inhibitors selectively transform osteoblasts and make bone cancer the dominant post-transplant malignancy.

D. Testicular germ-cell cancer, because allograft immunosuppression specifically increases seminoma incidence more than skin cancer.

E. Pituitary adenoma, because glucocorticoid therapy directly causes monoclonal pituitary neoplasia.

222. A patient with CKD stage G3b has rising albuminuria but no symptoms. Which approach best estimates the need for nephrology referral and kidney replacement planning?

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

B. 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.

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

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

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

223. A patient in septic shock requires vasopressors and has severe AKI with an indication for dialysis. Which modality may be favored when hemodynamic instability is profound?

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

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

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

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

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

224. A patient has nephrotic syndrome, cardiomyopathy, and neuropathy. Kidney biopsy shows Congo-red-positive deposits with apple-green birefringence. What process is present?

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

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

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

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

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

225. A patient with severe nephrotic syndrome develops sudden flank pain, hematuria, and worsening kidney function. Which complication should be considered?

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

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

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

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

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

226. A patient with advanced CKD has an available living donor and is otherwise a good transplant candidate. What strategy offers an important potential advantage?

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

B. Preemptive kidney transplantation before maintenance dialysis when feasible.

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

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

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

227. A 40-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. What is the diagnosis?

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

B. Primary polydipsia, which generally lowers rather than raises serum sodium and does not require absent pituitary vasopressin secretion.

C. Osmotic diuresis from glucosuria, which usually produces urine containing substantial solute rather than extremely dilute urine.

D. SIADH, which causes water retention and concentrated urine rather than hypernatremia with a large volume of dilute urine.

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

228. A 58-year-old man has eGFR 52 mL/min/1.73 m2 on one outpatient measurement and no other kidney abnormalities. What is required before labeling this chronic kidney disease on the basis of low GFR alone?

A. Repeat the creatinine after 24 hours because two abnormal values on consecutive days establish chronicity regardless of clinical context.

B. Demonstrate that the reduced GFR or another marker of kidney damage persists for at least three months.

C. Obtain a kidney biopsy because histologic confirmation is required whenever eGFR is below 60 mL/min/1.73 m2.

D. Document nephrotic-range proteinuria because reduced GFR without heavy proteinuria cannot meet criteria for chronic kidney disease.

E. Defer until eGFR falls below 30 mL/min/1.73 m2 because stages above that threshold are considered normal aging rather than CKD.

229. A patient has rapidly progressive glomerulonephritis, pulmonary hemorrhage, positive MPO-ANCA, and little immune deposition on biopsy. What pattern is present?

A. Immune-complex membranous nephropathy, which is a nephrotic subepithelial-deposit disease rather than pauci-immune crescentic nephritis.

B. Anti-GBM nephritis, which would show strong linear IgG staining rather than minimal immune deposition.

C. Diabetic glomerulosclerosis, which produces nodular mesangial expansion and chronic proteinuria rather than pulmonary capillaritis.

D. Minimal change disease, which shows podocyte effacement without necrotizing crescents or pulmonary hemorrhage.

E. Pauci-immune necrotizing crescentic glomerulonephritis associated with ANCA vasculitis.

230. A patient develops sudden flank pain, hematuria, severe hypertension, elevated LDH, and loss of perfusion to one kidney on imaging. What diagnosis is most likely?

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

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

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

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

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

231. A patient with CKD is taking ibuprofen daily for chronic back pain and develops a gradual rise in creatinine and worsening blood pressure. What medication change is most appropriate?

A. Increase the NSAID dose because stronger prostaglandin inhibition dilates the afferent arteriole and improves GFR in chronic kidney disease.

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. Stop or minimize chronic NSAID exposure and use safer analgesic strategies because NSAIDs can reduce renal perfusion, retain sodium, and worsen CKD.

232. 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. 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 rather than a defended low sodium level.

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 rather than defended hyponatremia.

233. A patient with AKI has potassium 7.2 mEq/L and new QRS widening. What is the immediate first priority?

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

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

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

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

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

234. A kidney recipient has rising creatinine and increasing BK viral load. Biopsy shows viral cytopathic changes in tubular cells. What is the core management principle?

A. Increase immunosuppression because BK nephropathy is caused by under-immunosuppression and is treated like acute cellular rejection.

B. Reduce net immunosuppression carefully to permit antiviral immune control while monitoring for rejection.

C. Give high-dose glucocorticoids as definitive antiviral therapy because steroids directly inhibit BK viral replication in tubular cells.

D. Remove the transplant immediately because BK viremia cannot be controlled without nephrectomy.

E. Use antibiotics targeting gram-negative bacteria because BK is a bacterial urinary pathogen rather than a polyomavirus.

235. A patient on loop diuretics has suspected prerenal physiology. Which laboratory index may be less directly affected by loop-induced natriuresis?

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

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

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

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

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

236. A patient has hypernatremia, polyuria, urine osmolality 70 mOsm/kg, and no access to water because of delirium. Which combination most directly explains development of severe hypernatremia?

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

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

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

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

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

237. A 58-year-old has abrupt worsening of hypertension, recurrent flash pulmonary edema, and asymmetric kidneys. Which secondary cause is most concerning?

A. Hemodynamically important renal artery stenosis, particularly atherosclerotic renovascular disease.

B. Essential hypertension alone, because flash pulmonary edema is a typical benign manifestation of uncomplicated primary hypertension.

C. Primary polydipsia, because excess water intake produces renal asymmetry and episodic severe hypertension.

D. Distal renal tubular acidosis, because chronic acid retention commonly causes flash pulmonary edema and renal artery narrowing.

E. Isolated nephrogenic diabetes insipidus, because polyuria produces recurrent pulmonary edema through sodium retention.

238. A 35-year-old taking a proton-pump inhibitor develops acute kidney injury, sterile pyuria, white-cell casts, and eosinophilia. What is the most likely diagnosis?

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

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

C. Minimal change disease, which primarily causes nephrotic proteinuria rather than an inflammatory interstitial urine sediment.

D. Prerenal azotemia, which usually has bland sediment and improves with restoration of perfusion rather than drug withdrawal alone.

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

239. 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. 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. The patient with severe albuminuria because albuminuria powerfully stratifies kidney and cardiovascular risk even at the same eGFR.

D. Neither patient is at meaningful risk because an eGFR above 30 mL/min/1.73 m2 excludes progressive chronic kidney disease.

E. Risk cannot be compared unless both patients are receiving dialysis because albuminuria has prognostic meaning only after kidney failure.

240. A patient with severe CKD requires a renally eliminated medication with a narrow therapeutic index. 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. Use the standard dose because kidney failure affects only endogenous solutes and has no effect on medication clearance.

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

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

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

Answers

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1. C. 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.

2. A. 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.

3. E. 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.

4. D. Prolonged severe hypoperfusion can cause structural tubular injury after an initial prerenal phase. Recovery may include a diuretic phase as tubular function improves.

5. 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.

6. D. 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.

7. D. 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.

8. E. 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.

9. 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.

10. D. 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.

11. C. 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.

12. D. 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.

13. A. 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.

14. 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.

15. C. Primary hyperparathyroidism causes autonomous PTH secretion, increasing serum calcium while lowering phosphate through renal phosphaturia. Hypercalciuria and recurrent calcium stones are common renal manifestations.

16. A. 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.

17. B. 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.

18. D. 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.

19. C. 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.

20. D. IgA nephropathy commonly causes hematuria concurrently with or shortly after a mucosal infection. Mesangial IgA deposition is the defining pathologic feature.

21. B. 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.

22. B. 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.

23. C. 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.

24. E. 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.

25. E. 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.

26. D. 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.

27. A. 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.

28. C. 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.

29. 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.

30. B. 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.

31. D. 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.

32. C. 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.

33. C. 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.

34. A. 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.

35. B. 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.

36. B. 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.

37. D. 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.

38. D. 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.

39. A. MAO inhibition impairs metabolism of dietary sympathomimetic amines. Large tyramine exposure can increase catecholamine release and precipitate severe acute hypertension.

40. E. 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.

41. B. 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.

42. 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.

43. D. 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.

44. A. 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.

45. 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.

46. 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.

47. 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.

48. E. 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.

49. A. 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.

50. 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.

51. E. 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.

52. 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.

53. A. 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.

54. E. 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.

55. E. 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.

56. B. 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.

57. D. 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.

58. D. 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.

59. A. 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.

60. B. 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.

61. E. 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.

62. A. 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.

63. C. 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.

64. 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.

65. B. 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.

66. E. 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.

67. B. 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.

68. C. 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.

69. A. 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.

70. A. 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.

71. D. 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.

72. A. 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.

73. B. 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.

74. C. 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.

75. D. Cirrhosis reduces effective arterial filling because of systemic and splanchnic vasodilation. Baroreceptor-mediated vasopressin release then promotes disproportionate water retention, producing hypervolemic hypotonic hyponatremia.

76. 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.

77. E. 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.

78. A. 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.

79. D. 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.

80. B. 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.

81. B. Hydronephrosis with low urine output points toward mechanical obstruction. Post-transplant causes include ureteral edema, stricture, kinking, stones, or external compression.

82. A. 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.

83. D. 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.

84. D. 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.

85. E. 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.

86. C. 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.

87. A. 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.

88. B. 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.

89. D. 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.

90. C. 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.

91. E. 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.

92. B. 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.

93. D. 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.

94. 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.

95. C. 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.

96. B. 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.

97. C. 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.

98. B. 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.

99. B. 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.

100. D. 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.

101. E. 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.

102. D. 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.

103. A. 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.

104. C. 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.

105. C. 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.

106. A. 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.

107. 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.

108. E. 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.

109. C. 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.

110. E. 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.

111. E. 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.

112. B. Severe ADAMTS13 deficiency supports TTP, usually from an acquired autoantibody in adults. Rapid recognition matters because plasma exchange and immunosuppressive therapy can be lifesaving.

113. D. 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.

114. C. Calcineurin inhibitors can cause dose-related renal vasoconstriction and acute functional decline in GFR. Chronic exposure can also contribute to vascular and tubulointerstitial injury.

115. 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.

116. B. 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.

117. 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.

118. 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.

119. D. 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.

120. C. 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.

121. A. 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.

122. 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.

123. A. 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.

124. 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.

125. C. 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.

126. B. 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.

127. B. 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.

128. A. 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.

129. 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.

130. E. 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.

131. D. 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.

132. A. 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.

133. 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.

134. 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.

135. A. 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.

136. B. 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.

137. C. 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.

138. D. 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.

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. 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.

141. 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.

142. D. 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.

143. 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.

144. C. 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.

145. A. 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.

146. E. 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.

147. A. Hyperacute rejection occurs when preformed antibodies bind donor endothelium immediately after reperfusion, activating complement and thrombosis. Modern crossmatching has made this event uncommon.

148. D. 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.

149. D. 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.

150. B. 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.

151. E. 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.

152. A. 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.

153. E. 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.

154. E. 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.

155. C. 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.

156. D. 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.

157. 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.

158. B. 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.

159. 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.

160. E. 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.

161. 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.

162. D. 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.

163. A. 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.

164. 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.

165. E. 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.

166. C. 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.

167. E. 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.

168. D. 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.

169. E. 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.

170. D. 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.

171. E. 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.

172. A. 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.

173. 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.

174. A. 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.

175. E. 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.

176. A. 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.

177. 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.

178. E. 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.

179. C. 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.

180. B. 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.

181. 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.

182. A. 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.

183. C. 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.

184. D. 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.

185. 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.

186. E. 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.

187. 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.

188. D. 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.

189. C. 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.

190. C. 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.

191. D. 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.

192. A. 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.

193. A. 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.

194. B. 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.

195. A. 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.

196. E. 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.

197. E. 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.

198. C. 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.

199. C. 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.

200. 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.

201. E. 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.

202. C. 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.

203. E. 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.

204. 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.

205. A. 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.

206. B. 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.

207. E. 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.

208. 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.

209. C. 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.

210. E. 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.

211. C. 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.

212. 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.

213. A. 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.

214. D. Dysmorphic red cells, especially acanthocytes, support glomerular hematuria. Red-cell casts provide even stronger evidence that bleeding originates within the nephron.

215. B. 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.

216. 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.

217. B. 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.

218. 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.

219. D. 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.

220. C. 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.

221. B. 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.

222. 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.

223. C. 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.

224. E. 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.

225. C. 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.

226. B. 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.

227. E. 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.

228. B. 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.

229. E. ANCA-associated vasculitis classically causes pauci-immune necrotizing and crescentic glomerulonephritis. Pulmonary capillaritis can produce a pulmonary-renal syndrome.

230. A. 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.

231. E. 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.

232. 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.

233. 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.

234. B. 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.

235. C. 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.

236. D. 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.

237. 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.

238. E. 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.

239. C. 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.

240. 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.