COMPREHENSIVE MOCK EXAM 8

Supplemental to: Nephrology Board Review 2026-2027

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

Board-Review Questions

1. A patient taking loop diuretics has suspected prerenal AKI. Which urine index may sometimes be less affected by loop diuretics than FENa?

A. Urine albumin concentration alone

B. Fractional excretion of phosphate

C. Urine calcium-to-creatinine ratio

D. Urine protein-to-creatinine ratio

E. Fractional excretion of urea

2. A patient taking an ACE inhibitor, a loop diuretic, and an NSAID develops AKI during a gastroenteritis illness. Which combined hemodynamic effect is most important?

A. Simultaneous afferent and efferent constriction markedly increases glomerular capillary pressure

B. The three drugs cause direct tubular crystal obstruction as their shared mechanism

C. NSAIDs increase prostaglandins while ACE inhibitors increase angiotensin II

D. Reduced afferent dilation plus reduced efferent constriction in a volume-depleted patient lowers glomerular filtration pressure

E. Loop diuretics increase renal plasma flow enough to offset the other medications

3. A patient develops brisk urine output after relief of prolonged bilateral obstruction. Which complication requires monitoring?

A. Persistent hyperkalemia caused by complete cessation of distal sodium delivery

B. Immediate nephrotic syndrome from restoration of GFR

C. Volume depletion with sodium, potassium, and magnesium losses during postobstructive diuresis

D. Acute hypercalcemia from renal retention of all filtered calcium

E. Obligate hypervolemia because urine may not contain sodium after obstruction relief

4. A patient with hypernatremia from pure water loss has no ongoing losses and is hemodynamically stable. Which fluid directly replaces the free-water deficit?

A. Three-percent saline because hypernatremia reflects a deficit of extracellular sodium

B. Isotonic saline as a primary fluid until the serum sodium has completely normalized

C. Enteral water or intravenous dextrose in water once intravascular stability is adequate

D. Hypertonic sodium bicarbonate because it lowers serum tonicity without adding water

E. Colloid solution because free water may not distribute into the intracellular compartment

5. A patient with advanced CKD has recurrent gout. Which general prescribing issue is important when selecting urate-lowering therapy?

A. Dialysis is required before any urate-lowering drug can be used

B. Kidney function is not a major consideration because all urate-lowering drugs are entirely hepatically cleared

C. NSAID prophylaxis is preferred indefinitely even with kidney function or volume status

D. Drug choice and dose should account for kidney function and adverse-effect risk

E. Hyperuricemia should be treated with high-dose potassium supplementation

6. A patient with CKD wants to know why albuminuria is monitored after treatment begins. Which response is best?

A. Albuminuria is useful primarily in kidney stone disease and not in glomerular disease

B. Urine albumin is monitored solely to determine whether dialysis membranes are functioning

C. Changes in albuminuria provide information about kidney-disease activity, prognosis, and response to therapy

D. Once CKD is diagnosed, albuminuria has no further prognostic or therapeutic value

E. Albuminuria is measured primarily to estimate serum sodium concentration

7. A 52-year-old man with a catecholamine-secreting adrenal tumor begins preoperative preparation. After several days of alpha-adrenergic blockade, he remains tachycardic and is no longer orthostatic. Which next step is appropriate?

A. Stop alpha blockade and use a thiazide as primary preoperative therapy

B. Restrict sodium and fluid intake to maintain chronic volume contraction

C. Add a beta blocker now that adequate alpha blockade has been established

D. Give intravenous beta blocker and discontinue alpha blockade immediately

E. Proceed directly to surgery without further hemodynamic preparation

8. A septic patient with AKI has muddy brown granular casts and renal tubular epithelial cells on urine microscopy. Which lesion is most likely?

A. Minimal-change disease

B. Postrenal obstruction

C. Acute interstitial nephritis

D. Acute tubular injury

E. Renal-limited anti-GBM disease

9. A transplant recipient develops nephrotic-range proteinuria 18 months after transplantation. Why is kidney biopsy often necessary?

A. Glomerular disease after transplantation is more likely to arise in the native kidneys than in the allograft

B. A biopsy may not distinguish rejection from recurrent disease

C. Proteinuria after transplantation is commonly caused by recurrent diabetic nephropathy

D. The differential includes recurrent disease, de novo glomerulopathy, rejection, and other allograft injury

E. Proteinuria is expected after transplantation and has no prognostic significance

10. A patient has decades of heavy analgesic combination use and develops slowly progressive CKD, sterile pyuria, and papillary calcifications. Which diagnosis is most likely?

A. Acute poststreptococcal glomerulonephritis

B. Renal-limited ANCA vasculitis

C. Autosomal dominant polycystic kidney disease

D. Minimal-change disease from selective podocyte injury

E. Chronic analgesic nephropathy with papillary necrosis

11. A hospitalized patient receives large quantities of hypertonic sodium bicarbonate during resuscitation and develops serum sodium 160 mEq/L with weight gain. Which mechanism is most likely?

A. Central diabetes insipidus causing free-water loss with obligatory volume contraction

B. Iatrogenic sodium gain causing hypernatremia with extracellular volume expansion

C. Adrenal insufficiency causing combined sodium wasting and hyperkalemia

D. Primary polydipsia causing dilutional hyponatremia rather than hypernatremia

E. SIADH causing sodium dilution in the setting of inappropriately concentrated urine

12. A patient with severe hyperkalemia receives intravenous insulin and glucose. What is the principal effect of this treatment?

A. Rapid intracellular shift of potassium without removing potassium from the body

B. Immediate gastrointestinal binding and elimination of potassium in stool

C. Permanent renal potassium excretion independent of kidney function

D. Removal of potassium across a dialysis membrane over several hours

E. Direct stabilization of cardiac myocytes without changing extracellular potassium

13. A 67-year-old woman taking hydrochlorothiazide presents with gait instability. Serum sodium is 116 mEq/L, serum osmolality is low, urine osmolality is 510 mOsm/kg, and urine sodium is 54 mEq/L. She has no edema. What is the most appropriate immediate step?

A. Continue the thiazide and restrict dietary sodium while observing neurologic status

B. Give desmopressin alone to increase urinary water excretion and raise serum sodium

C. Begin a loop diuretic as the primary therapy despite the neurologic manifestations

D. Infuse isotonic dextrose solution to correct the low measured serum osmolality

E. Stop the thiazide and give carefully monitored hypertonic saline for severe symptoms

14. A patient with profound hypokalemia develops muscle weakness after several days of high-dose loop diuretic therapy. Which renal effect contributes most directly?

A. Reduced distal sodium delivery that suppresses potassium secretion despite volume contraction

B. Direct blockade of epithelial sodium channels that prevents potassium secretion

C. Decreased renin release causing sodium wasting without activation of distal potassium secretion

D. Inhibition of aldosterone synthesis causing a hyperkalemic metabolic acidosis

E. Increased distal sodium delivery with enhanced potassium secretion in the collecting duct

15. A hospitalized patient receives large amounts of normal saline and develops bicarbonate 17 mEq/L with a normal anion gap. Which mechanism is most likely?

A. Saline causes primary respiratory acidosis by suppressing alveolar ventilation

B. Relative chloride excess lowers bicarbonate concentration and produces hyperchloremic acidosis

C. Sodium chloride directly stimulates aldosterone enough to cause metabolic alkalosis

D. Renal generation of new bicarbonate is immediately abolished by isotonic sodium delivery

E. Lactate accumulation from saline metabolism directly creates a high anion gap

16. A patient with rhabdomyolysis is hemodynamically stable but volume depleted. Which early treatment is most important?

A. Prompt isotonic fluid administration to restore perfusion and support urine flow

B. NSAID therapy to reduce muscle inflammation and renal blood flow

C. Potassium supplementation despite hyperkalemia from muscle breakdown

D. Routine dialysis before any electrolyte or volume indication develops

E. Fluid restriction to prevent pigment from reaching the kidney

17. A kidney transplant recipient has abrupt loss of graft perfusion immediately after vascular anastomosis. Which nonimmune complication must be excluded urgently?

A. Recurrent diabetic nephropathy

B. BK virus nephropathy

C. Renal artery or vein thrombosis of the allograft

D. Late calcineurin inhibitor arteriolopathy

E. Chronic transplant glomerulopathy

18. A patient has uncontrolled blood pressure despite three appropriately dosed agents including a diuretic. Which step should occur before labeling the condition true resistant hypertension?

A. Proceed directly to renal denervation without evaluating adherence or measurement technique

B. Exclude primary aldosteronism because hypokalemia is required for the diagnosis

C. Confirm adherence, accurate measurement, and out-of-office blood pressure while reviewing interfering substances

D. Assume all elevated clinic readings represent true resistance even with home values

E. Stop the diuretic because resistant hypertension is defined by absence of diuretic therapy

19. A patient has AKI after iodinated contrast exposure but also develops livedo reticularis and eosinophilia a week later. Which diagnosis better explains the delayed systemic findings?

A. Cholesterol atheroembolic kidney disease

B. Minimal-change disease

C. Central diabetes insipidus

D. Prerenal azotemia from contrast osmolarity alone

E. Distal renal tubular acidosis

20. A patient with multiple myeloma develops glycosuria despite normal serum glucose, phosphaturia, aminoaciduria, and a normal-anion-gap metabolic acidosis. Which lesion is present?

A. Primary respiratory alkalosis causing compensatory urinary bicarbonate excretion

B. Failure of distal hydrogen secretion causing nephrocalcinosis without proximal solute wasting

C. Selective collecting-duct resistance to aldosterone causing isolated type 4 RTA

D. Excess gastric acid loss causing chloride-responsive metabolic alkalosis

E. Generalized proximal tubular dysfunction causing Fanconi syndrome and proximal RTA

21. A patient with prolonged hypotension initially has low urine sodium but later develops persistent AKI with granular casts and a higher fractional excretion of sodium. What transition has likely occurred?

A. The patient has developed central diabetes insipidus

B. The patient has developed minimal-change nephrotic syndrome

C. Prerenal physiology has progressed to structural acute tubular injury

D. The patient has converted to isolated postrenal obstruction

E. Acute tubular injury has resolved into normal kidney function

22. A 19-year-old patient develops edema and nephrotic-range proteinuria after a recent viral illness. Kidney function is normal and urine sediment is bland. Which biopsy finding would support minimal-change disease?

A. Subepithelial immune deposits with spikes along the glomerular basement membrane

B. Mesangial IgA-dominant immune deposits

C. Crescent formation with pauci-immune immunofluorescence

D. Linear IgG staining of the glomerular basement membrane

E. Diffuse podocyte foot-process effacement on electron microscopy

23. A dialysis patient with chronic atrial fibrillation asks about anticoagulation. Which principle is most appropriate?

A. Anticoagulation decisions can ignore drug clearance because dialysis removes many anticoagulants to a similar extent

B. Balance thromboembolic and bleeding risks individually because kidney failure alters both

C. All dialysis patients should receive the same anticoagulant dose used in normal kidney function

D. Bleeding risk is absent in dialysis because uremic platelet dysfunction prevents thrombosis primarily

E. Kidney failure substantially lowers stroke risk and usually makes anticoagulation unnecessary

24. A patient has an elevated aldosterone-to-renin ratio and biochemical confirmation of primary aldosteronism. CT shows a small unilateral adrenal nodule, but the patient is 58 years old. What test often helps determine whether aldosterone secretion is truly unilateral before surgery?

A. Adrenal vein sampling

B. Kidney biopsy

C. Twenty-four-hour urine protein quantification

D. Plasma free metanephrine measurement

E. Renal artery duplex ultrasonography

25. A child develops edema, hypertension, hematuria, and low C3 two weeks after impetigo. Which diagnosis is most likely?

A. Primary membranous nephropathy

B. Poststreptococcal glomerulonephritis

C. Anti-GBM glomerulonephritis

D. IgA nephropathy with synpharyngitic hematuria

E. Minimal-change nephrotic syndrome

26. A patient with resistant hypertension takes ibuprofen daily, uses pseudoephedrine, and drinks heavily. Which management principle is most appropriate?

A. Continue all substances because medication-induced hypertension may not mimic resistance

B. Start mineralocorticoid replacement because decongestants suppress aldosterone

C. Remove or reduce substances that can raise blood pressure before intensifying the regimen

D. Add a fifth antihypertensive without reviewing nonprescription agents

E. Stop all current blood-pressure drugs because NSAIDs prevent them from working entirely

27. A kidney allograft biopsy obtained for rising creatinine shows glomerulitis and peritubular capillaritis. Circulating donor-specific HLA antibodies are present. Which additional biopsy finding would most strongly support antibody interaction with the microvascular endothelium?

A. Diffuse foot-process effacement without immune deposits

B. Linear IgG staining of glomerular basement membranes

C. Isolated tubular epithelial vacuolization

D. C4d deposition in peritubular capillaries

E. Medullary urate crystal deposition

28. A patient with oliguric AKI receives furosemide and urine output increases, but serum creatinine continues to rise. What is the best interpretation?

A. Increased urine output strongly suggests the kidney injury has resolved

B. Furosemide commonly shortens the duration of established intrinsic AKI

C. A diuretic response excludes intrinsic kidney disease

D. Rising creatinine after diuresis strongly suggests bilateral obstruction

E. Diuretics can manage volume but do not by themselves reverse established tubular injury

29. A patient with severe AKI has persistent positive fluid balance, worsening oxygenation, and intra-abdominal edema despite declining vasopressor needs. Which concept is important?

A. Fluid accumulation itself can worsen organ function and should be actively reassessed after resuscitation

B. Positive fluid balance is commonly protective for the kidney once shock has resolved

C. Diuretics reverse tubular injury even when they do not improve volume status

D. Fluid removal should rarely be considered until serum creatinine normalizes

E. Pulmonary edema may not result from fluid accumulation in oliguric AKI

30. A patient with decompensated heart failure has elevated jugular venous pressure, edema, low urine sodium, and AKI. Which hemodynamic concept is most relevant?

A. Edema excludes prerenal physiology because extracellular volume is increased

B. A low urine sodium strongly favors absolute volume depletion despite clinical evidence of congestion

C. Kidney perfusion is determined primarily by systemic blood pressure and not venous pressure

D. Heart failure causes AKI primarily through direct immune-complex deposition

E. Low effective arterial blood volume and venous congestion can reduce kidney filtration despite total-body fluid excess

31. A patient chooses peritoneal dialysis. Which feature most directly enables solute and fluid removal?

A. The liver actively pumps uremic toxins across the abdominal wall into the dialysate

B. The dialysis catheter filters plasma directly through its lumen without membrane exchange

C. The bowel secretes creatinine into the dialysate through active transport

D. The peritoneal membrane acts as the semipermeable exchange surface between blood and dialysate

E. The bladder concentrates dialysate after intraperitoneal absorption

32. A patient has rapidly progressive glomerulonephritis, pulmonary nodules, sinus disease, and PR3-ANCA positivity. Which diagnosis is most likely?

A. Minimal-change disease

B. Anti-GBM disease

C. Granulomatosis with polyangiitis

D. Microscopic polyangiitis

E. Membranous nephropathy

33. A patient with leukemia has a reported potassium of 6.7 mEq/L but no ECG changes. The blood sample was difficult to draw and visibly hemolyzed. What is the best next step?

A. Start fludrocortisone because pseudohyperkalemia is caused by aldosterone deficiency

B. Repeat potassium promptly using a nonhemolyzed specimen while assessing clinical risk

C. Give intravenous potassium because hemolysis usually lowers the measured concentration

D. Administer emergent dialysis solely on the basis of the hemolyzed laboratory value

E. Ignore the result permanently because true hyperkalemia may not occur without ECG changes

34. A patient with CKD has eGFR 28 mL/min/1.73 m2 and urine albumin-to-creatinine ratio 850 mg/g. Which combination best predicts kidney and cardiovascular risk?

A. Serum sodium and potassium are sufficient for long-term CKD risk classification

B. Both reduced eGFR and severe albuminuria should be considered together

C. Risk is determined by age alone once eGFR is below 30 mL/min/1.73 m2

D. Serum creatinine should be used alone because albuminuria adds no prognostic information

E. Albuminuria should be used alone because eGFR does not predict clinical outcomes

35. A child develops bloody diarrhea followed by AKI, thrombocytopenia, and microangiopathic hemolytic anemia. Which diagnosis is most likely?

A. Shiga toxin-associated hemolytic uremic syndrome

B. Anti-GBM glomerulonephritis

C. Thrombotic thrombocytopenic purpura from severe ADAMTS13 deficiency

D. Pauci-immune ANCA-associated glomerulonephritis

E. Minimal-change nephrotic syndrome

36. A patient with persistent vomiting has bicarbonate 36 mEq/L and urine chloride 6 mEq/L. Which treatment is most likely to correct the alkalosis?

A. Sodium bicarbonate infusion to replace urinary alkali losses from the kidney

B. High-dose mineralocorticoid therapy to increase distal hydrogen and potassium secretion

C. Chloride and volume repletion with isotonic saline plus potassium as needed

D. Acetazolamide is preferred before any correction of extracellular volume depletion

E. Free-water restriction because the alkalosis reflects inappropriate antidiuretic hormone release

37. A patient with sepsis and AKI is receiving several antibiotics. Why should doses be reassessed repeatedly rather than adjusted once?

A. Recovery of kidney function commonly occurs at a fixed rate independent of illness

B. Antibiotic pharmacokinetics remain constant throughout AKI and dialysis

C. Serum creatinine changes do not alter drug exposure in critically ill patients

D. CRRT removes no antimicrobial drugs and therefore rarely affects dosing

E. Kidney function and extracorporeal clearance can change rapidly during critical illness

38. A patient with hypokalemia has a spot urine potassium concentration that remains high despite low serum potassium. What does this finding suggest?

A. Pseudohypokalemia caused by cellular uptake of potassium after blood collection

B. Ongoing renal potassium wasting rather than an exclusively extrarenal loss

C. Hyperkalemic type 4 renal tubular acidosis despite the measured low serum potassium

D. Primary respiratory alkalosis because urinary potassium may not distinguish renal from extrarenal loss

E. Appropriate renal potassium conservation indicating an extrarenal source

39. A patient has calcium 11.4 mg/dL, mildly elevated PTH, and very low 24-hour urine calcium excretion. Several relatives have lifelong mild hypercalcemia. Which diagnosis is most likely?

A. Primary hyperparathyroidism with typical increased urinary calcium excretion

B. Vitamin D intoxication with suppressed parathyroid hormone and high phosphate

C. Granulomatous hypercalcemia from excess calcitriol with suppressed parathyroid hormone

D. Familial hypocalciuric hypercalcemia from altered calcium-sensing receptor function

E. Humoral hypercalcemia of malignancy with suppressed parathyroid hormone

40. A patient with albuminuric CKD starts losartan. Serum creatinine rises from 1.8 to 2.1 mg/dL and potassium remains normal. Blood pressure improves. What is the best interpretation?

A. A modest early creatinine rise can reflect the expected hemodynamic effect of RAAS blockade

B. The dose should be doubled immediately because creatinine rise confirms therapeutic underdosing

C. The rise indicates acute interstitial nephritis from losartan and requires corticosteroids

D. The drug must be stopped because any creatinine increase strongly suggests structural kidney injury

E. The patient has renal artery thrombosis because RAAS blockers do not alter intraglomerular hemodynamics

41. A kidney transplant recipient wants to become pregnant. Which immunosuppressant is particularly important to discontinue well before conception because of teratogenicity?

A. Azathioprine

B. Prednisone

C. Mycophenolate

D. Hydroxychloroquine

E. Tacrolimus

42. A patient receiving high-dose methotrexate develops AKI with intratubular crystals. Which preventive measure is important during high-dose therapy?

A. Adequate hydration and urine alkalinization to reduce methotrexate crystal precipitation

B. Intentional dehydration to concentrate the drug and improve antineoplastic effect

C. Potassium restriction because methotrexate crystals form primarily in hyperkalemic urine

D. Urine acidification to keep methotrexate insoluble within the tubule

E. Routine NSAID administration to lower renal blood flow during methotrexate infusion

43. A patient with bilateral severe renal artery stenosis starts an ACE inhibitor and creatinine rises markedly. What mechanism explains the decline in GFR?

A. Excess afferent arteriolar dilation increases glomerular pressure until filtration stops

B. Direct ACE-inhibitor crystal deposition obstructs proximal tubules

C. ACE inhibition produces renal vein thrombosis in bilateral renovascular disease

D. Loss of angiotensin II-mediated efferent arteriolar constriction in kidneys dependent on low perfusion pressure

E. Increased aldosterone secretion causes intense afferent vasoconstriction

44. A patient with advanced cirrhosis has edema, ascites, serum sodium 126 mEq/L, and low effective arterial blood volume. Which physiologic response is central to the hyponatremia?

A. Primary renal sodium retention without any change in water handling or thirst

B. Complete suppression of vasopressin causing excretion of maximally dilute urine

C. Loss of medullary concentrating ability causing obligatory electrolyte-free water excretion

D. Excess atrial natriuretic peptide causing isolated sodium loss without neurohormonal activation

E. Nonosmotic vasopressin release causing retention of water out of proportion to sodium

45. A malnourished patient begins nutritional rehabilitation and develops weakness, rhabdomyolysis, and serum phosphate 1.0 mg/dL. What is the most likely cause?

A. Insulin-driven intracellular phosphate uptake during refeeding

B. Acute renal failure causing impaired phosphate excretion and extracellular accumulation

C. Hypoparathyroidism causing increased proximal phosphate reabsorption

D. Cell lysis causing release of intracellular phosphate into the circulation

E. Vitamin D intoxication causing increased intestinal phosphate absorption

46. A patient with CKD is scheduled for an elective procedure and takes several medications that can affect renal perfusion or volume. Which principle is best?

A. Continue most medications routinely because peri-procedural kidney risk is minimally affected by medication choices

B. Begin NSAIDs prophylactically because they preserve renal autoregulation during anesthesia

C. Review nephrotoxic and hemodynamically active drugs in the peri-procedural setting and individualize temporary holds

D. Use a fixed universal medication-hold schedule independent of volume status or kidney function

E. Stop all antihypertensive and diabetes therapies for one month even with the procedure

47. A transplant recipient develops rising creatinine 10 days after transplantation. Doppler ultrasound excludes obstruction and vascular compromise. Biopsy shows interstitial inflammation with tubulitis. Which diagnosis is most likely?

A. Recurrent focal segmental glomerulosclerosis

B. BK virus nephropathy

C. Chronic antibody-mediated rejection

D. Calcineurin inhibitor toxicity without inflammatory lesions

E. Acute T-cell-mediated rejection

48. A 35-year-old woman has palpable purpura, abdominal pain, arthralgias, hematuria, and proteinuria. Kidney biopsy shows mesangial IgA deposition. Which diagnosis is most likely?

A. Cryoglobulinemic vasculitis

B. Anti-GBM disease

C. IgA vasculitis with nephritis

D. Thrombotic thrombocytopenic purpura

E. Minimal-change disease

49. A dialysis patient has persistently high phosphate despite dietary counseling. Which approach is reasonable?

A. Use a phosphate binder with meals and optimize dialysis and dietary phosphate management

B. Take phosphate binders primarily between meals because they act independently of intestinal phosphate intake

C. Increase dietary processed-food phosphate because it is poorly absorbed compared with plant phosphate

D. Give routine high-dose calcitriol solely to lower serum phosphate by reducing intestinal absorption

E. Stop all dialysis because extracorporeal phosphate removal worsens hyperphosphatemia

50. A patient on CRRT develops recurrent hypophosphatemia. Which management is appropriate?

A. Ignore the finding because CRRT may not remove phosphate

B. Give calcium alone because hypophosphatemia is caused by calcium deficiency

C. Stop all nutrition because phosphate enters primarily through feeding

D. Provide phosphate replacement or use phosphate-containing CRRT solutions when appropriate

E. Increase citrate dose because citrate directly raises serum phosphate

51. A patient with malignant hypertension has AKI, schistocytes, and thrombocytopenia. Which pathologic process is most likely in the kidney?

A. Mesangial IgA deposition causing synpharyngitic hematuria

B. Subepithelial immune-complex deposition with podocyte foot-process effacement

C. Isolated proximal tubular bicarbonate wasting without vascular injury

D. Linear anti-GBM antibody deposition as the expected lesion

E. Severe arteriolar endothelial injury producing a secondary thrombotic microangiopathy

52. A patient has hypertension, hypokalemia, bicarbonate 34 mEq/L, suppressed renin, and elevated aldosterone. Which acid-base disturbance is expected?

A. Metabolic alkalosis from enhanced distal hydrogen and potassium secretion

B. Primary respiratory acidosis from suppression of medullary respiratory drive

C. High-anion-gap metabolic acidosis from increased lactic acid generation

D. Primary respiratory alkalosis caused by direct aldosterone stimulation of ventilation

E. Normal-anion-gap metabolic acidosis from impaired distal hydrogen secretion

53. A patient with CKD has low 25-hydroxyvitamin D and elevated PTH. Which initial principle is reasonable?

A. Target a low-normal PTH concentration across CKD stages without considering bone turnover

B. Ignore vitamin D status because it has no role in CKD mineral metabolism

C. Administer high-dose calcium even with serum calcium or vascular calcification risk

D. Treat a single mildly elevated PTH value without considering longitudinal trends

E. Correct vitamin D deficiency and evaluate phosphate, calcium, and trends in PTH together

54. A patient has purpura, arthralgias, neuropathy, low complement, hepatitis C infection, and membranoproliferative glomerulonephritis. Which diagnosis is most likely?

A. Thin-basement-membrane nephropathy

B. Minimal-change nephrotic syndrome

C. Anti-GBM glomerulonephritis

D. HCV-associated cryoglobulinemic glomerulonephritis

E. Primary focal segmental glomerulosclerosis

55. A patient with eGFR 18 mL/min/1.73 m2 is asymptomatic, potassium and bicarbonate are controlled, and volume status is stable. Which approach to dialysis timing is best?

A. Use serum creatinine alone to determine the exact calendar date dialysis must begin

B. Avoid access planning because stable patients rarely progress to kidney failure

C. Start dialysis immediately because eGFR below 20 is an absolute universal indication

D. Wait until cardiac arrest from hyperkalemia before discussing kidney replacement therapy

E. Continue close follow-up and preparation rather than starting dialysis solely because of the eGFR number

56. A patient with severe hyperkalemia, pulmonary edema, and refractory metabolic acidosis has oliguric AKI. Which management is indicated?

A. Urgent kidney replacement therapy for life-threatening complications

B. Avoid dialysis because acidosis prevents membrane solute transfer

C. Use a phosphate binder as the primary therapy for all three complications

D. Observation until creatinine reaches a predetermined universal level

E. Potassium-rich fluid administration to stimulate renal excretion

57. A hemodialysis patient develops severe headache and chest pain with very high blood pressure between treatments. Which contributing factor should be assessed?

A. Chronic extracellular volume excess and high sodium burden

B. Low dialysate calcium as a primary possible explanation for hypertension

C. Persistent hypovolemia because dialysis hypertension is caused primarily by underfilling

D. Loss of all renin activity because kidney failure prevents any vasoactive signaling

E. Excess removal of sodium during missed dialysis despite increasing body weight

58. A patient with vomiting is hypotensive and hyponatremic. Urine sodium is 8 mEq/L and urine osmolality is 620 mOsm/kg. Which process best accounts for the laboratory pattern?

A. Primary renal salt wasting with failure to conserve sodium during extracellular volume depletion

B. SIADH with euvolemia and persistent urinary sodium excretion despite normal perfusion

C. Primary polydipsia with complete suppression of antidiuretic hormone and dilute urine

D. Pseudohyponatremia from hyperproteinemia with a normal measured serum osmolality

E. Extrarenal salt loss with renal sodium conservation and baroreceptor-mediated antidiuresis

59. A patient with cancer develops flank pain, gross hematuria, enlarged kidney, and nephrotic-range proteinuria. Imaging shows renal vein thrombosis. Which mechanism can reduce GFR?

A. The lesion increases filtration by lowering Bowman-space pressure

B. Venous thrombosis directly causes central diabetes insipidus

C. Enhanced renal venous drainage increases glomerular hydrostatic pressure

D. Renal vein thrombosis causing isolated tubular bicarbonate wasting without a hemodynamic effect

E. Impaired renal venous outflow raises intrarenal pressure and reduces effective filtration

60. A patient with ADPKD has rapidly progressive disease and preserved enough kidney function for disease-modifying therapy. Which medication may slow cystic kidney growth in appropriately selected adults?

A. Lithium to reduce collecting-duct cyclic AMP signaling

B. Tolvaptan, a vasopressin V2-receptor antagonist

C. Desmopressin, a vasopressin V2-receptor agonist

D. High-dose NSAID therapy to reduce renal blood flow

E. Fludrocortisone to expand extracellular volume

61. A patient is recovering from bilateral urinary obstruction after catheter placement and produces 7 L of urine during the next 24 hours. Which management principle is most appropriate?

A. Administer hypertonic saline routinely because the urine is necessarily sodium-free

B. Replace a portion of urinary water and electrolyte losses while monitoring volume and serum chemistries

C. Start desmopressin immediately because all postobstructive polyuria represents central diabetes insipidus

D. Give no fluid because postobstructive diuresis is commonly a benign physiologic response

E. Replace an equal volume of urine with free water to prevent any fall in urine output

62. A kidney transplant recipient develops dyspnea, diffuse pulmonary infiltrates, elevated LDH, and hypoxemia several months after transplant without appropriate prophylaxis. Which opportunistic infection is a major concern?

A. Pneumocystis jirovecii pneumonia

B. BK virus nephropathy

C. Acute uric acid nephropathy

D. Recurrent IgA nephropathy

E. Transplant renal artery stenosis

63. A kidney transplant recipient has rising creatinine and plasma BK viral load. Biopsy shows viral cytopathic changes in tubular epithelial cells. Which diagnosis is most likely?

A. BK polyomavirus nephropathy

B. Cytomegalovirus colitis

C. Transplant renal artery stenosis

D. Acute T-cell-mediated rejection

E. Recurrent IgA nephropathy

64. A dialysis patient misses several treatments and presents with dyspnea, edema, potassium 6.6 mEq/L, and bicarbonate 14 mEq/L. Which intervention is most definitive?

A. Potassium supplementation because acidosis lowers total-body potassium

B. Stopping all antihypertensives as definitive treatment for the missed dialysis

C. Oral fluid loading to increase edema and dilute serum potassium

D. Urgent dialysis to remove potassium, acid, and excess volume

E. A single phosphate binder dose because phosphate binding corrects hyperkalemia and acidosis

65. A patient with AKI has bilateral hydronephrosis and a distended bladder. Which immediate intervention is most appropriate if bladder outlet obstruction is suspected?

A. Give high-dose diuretics before relieving the obstruction

B. Start immunosuppression because hydronephrosis indicates interstitial nephritis

C. Restrict fluids and wait for spontaneous ureteral dilation

D. Begin plasma exchange because obstruction causes anti-GBM antibodies

E. Decompress the bladder with urinary catheterization when feasible

66. A patient with CKD has repeated hyperkalemia while taking an ACE inhibitor that is providing substantial albuminuria benefit. Which strategy may allow continuation when clinically appropriate?

A. Address dietary and medication contributors and consider diuretics or a potassium binder with close monitoring

B. Add routine potassium supplements to improve the antiproteinuric effect

C. Ignore potassium values because RAAS blockade prevents cardiac effects of hyperkalemia

D. Combine an ACE inhibitor and ARB to offset hyperkalemia through dual blockade

E. Stop all diuretics because they increase urinary potassium excretion

67. A patient on hemodialysis develops thrombocytopenia and repeated clotting of the dialysis circuit after recent heparin exposure. Which diagnosis should be considered?

A. Iron deficiency because reduced ferritin causes acute circuit thrombosis

B. Uremic platelet dysfunction because it causes thrombosis rather than bleeding

C. Secondary hyperparathyroidism because elevated PTH directly activates the heparin antibody

D. Heparin-induced thrombocytopenia with a prothrombotic immune response

E. Dialysis disequilibrium syndrome because it lowers platelets during rapid urea removal

68. A patient with acute leukemia develops hyperkalemia, hyperphosphatemia, hypocalcemia, hyperuricemia, and AKI after chemotherapy. Which syndrome is present?

A. Distal renal tubular acidosis

B. Tumor lysis syndrome

C. SIADH

D. Nephrotic syndrome

E. Hepatorenal syndrome

69. A patient with severe COPD has a chronic PaCO2 of 60 mm Hg and stable renal function. Which renal compensation is expected over several days?

A. An increase in serum bicarbonate from enhanced renal acid excretion and bicarbonate retention

B. No bicarbonate change because the kidney does not compensate for respiratory disorders

C. Immediate urinary loss of ammonium that lowers systemic bicarbonate

D. Persistent hyperchloremic acidosis caused by compensatory sodium retention

E. A marked fall in serum bicarbonate from renal bicarbonate wasting

70. A pregnant patient at 28 weeks develops hypertension, proteinuria, thrombocytopenia, and elevated liver enzymes. Which diagnosis is most likely?

A. Primary aldosteronism

B. Preeclampsia with severe features

C. White-coat hypertension

D. Liddle syndrome

E. Renal artery fibromuscular dysplasia

71. A healthy potential living donor has newly discovered persistent albuminuria. What is the best principle?

A. Ignore albuminuria because removal of one kidney eliminates protein excretion

B. Treat with immunosuppression before donation to normalize the urine

C. Accept the donor routinely if serum creatinine is within the laboratory reference range

D. Proceed with donation because long-term CKD risk after nephrectomy is negligible in a healthy donor candidate

E. Investigate the albuminuria carefully because donor evaluation prioritizes the donor's long-term kidney health

72. A dialysis patient with severe secondary hyperparathyroidism remains uncontrolled despite medical therapy. Which treatment may be required?

A. Routine adrenalectomy because aldosterone is the primary driver of PTH excess

B. Permanent cessation of dialysis because dialysis causes all parathyroid disease

C. Parathyroidectomy for refractory severe hyperparathyroidism

D. Total thyroidectomy because thyroid hormone drives the parathyroid response

E. Long-term high-dose phosphate supplementation to suppress PTH secretion

73. A hypertensive patient has hypokalemic metabolic alkalosis, suppressed renin, suppressed aldosterone, and a family history of early hypertension. Which disorder is most likely?

A. Liddle syndrome from constitutive epithelial sodium-channel activity

B. Bartter syndrome from impaired sodium chloride transport in the thick ascending limb

C. Renovascular hypertension from unilateral renal artery stenosis

D. Primary aldosteronism from an aldosterone-producing adrenal adenoma

E. Type 4 renal tubular acidosis from reduced aldosterone effect

74. A patient with CKD and hyperkalemia is advised to modify diet. Which counseling principle is best?

A. Ignore processed-food potassium additives because they are not absorbed

B. Use dietary restriction as a primary treatment even when severe hyperkalemia is recurrent

C. Eliminate all fruits and vegetables even with potassium level or nutritional needs

D. Increase potassium-containing salt substitutes because they lower sodium without risk

E. Target high-potassium foods and additives in the context of overall nutrition rather than imposing indiscriminate restriction

75. A patient with kidney failure has platelet dysfunction and mucosal bleeding despite a normal platelet count. Which mechanism is typical of uremia?

A. Autoimmune destruction of platelets caused by secondary hyperparathyroidism

B. Vitamin K deficiency caused directly by low glomerular filtration rate

C. Qualitative platelet dysfunction from retained uremic toxins

D. Excess fibrinogen production leading to isolated hypercoagulability without bleeding

E. Bone marrow hypoproduction commonly causes profound thrombocytopenia in patients with uremia

76. A kidney transplant recipient has recurrent nephrolithiasis but reports little pain with ureteral obstruction. Why can symptoms be atypical?

A. Transplant ureters are larger and may not become obstructed

B. Calcineurin inhibitors commonly suppress pain signaling from the transplanted kidney

C. The transplanted kidney and ureter are denervated, so classic renal colic may be absent

D. Immunosuppression blocks stone formation but increases pain perception

E. Allograft stones are painless primarily when they contain uric acid

77. A patient with severe AKI has no urgent dialysis indication and begins producing more urine with falling creatinine. Which finding best supports renal recovery?

A. Persistent hyperkalemia despite increasing urine output

B. Rising BUN and creatinine with worsening acidosis

C. Ongoing anuria with increasing fluid overload

D. Sustained improvement in filtration and urine output without escalating dialysis support

E. A single large urine volume after a high-dose diuretic even with creatinine trend

78. A patient with stage G5 CKD has persistent hyperkalemia despite dietary changes and optimized medical therapy. Which consideration is appropriate?

A. Refractory hyperkalemia is an indication to consider kidney replacement therapy

B. RAAS blockade should commonly be increased despite repeated life-threatening potassium elevations

C. Hyperkalemia is expected in CKD and does not influence dialysis planning

D. Dialysis should be avoided because potassium may not cross dialysis membranes

E. Increasing potassium intake can enhance renal secretion and correct the problem

79. A patient treated with amphotericin B develops rising creatinine, hypokalemia, and renal magnesium wasting. Which mechanism is most likely?

A. Direct tubular membrane toxicity with renal vasoconstriction

B. Immune-complex glomerulonephritis with isolated heavy proteinuria

C. Bilateral renal artery emboli from fungal organisms

D. Pure prerenal azotemia without tubular electrolyte losses

E. Epithelial sodium-channel activation causing hypertension and alkalosis

80. A patient with advanced CKD reports anorexia, pruritus, nausea, and progressive cognitive slowing despite optimized medical therapy. Which finding most strongly supports starting dialysis?

A. An isolated eGFR value below 15 mL/min/1.73 m2 in an entirely asymptomatic patient

B. A stable serum creatinine above the laboratory reference range without complications

C. Mild proteinuria alone in a patient with preserved volume and electrolyte control

D. A single elevated BUN value without symptoms, volume overload, or metabolic complications

E. Persistent uremic symptoms attributable to kidney failure despite medical management

81. A patient with rapidly progressive AKI has dysmorphic red cells and red-cell casts. Which category of injury is most likely?

A. Glomerulonephritis

B. Pure prerenal azotemia

C. Isolated acute tubular injury

D. Lower urinary tract obstruction

E. Central diabetes insipidus

82. A 48-year-old man has resistant hypertension and spontaneous potassium 3.0 mEq/L. Which screening test is most appropriate for primary aldosteronism?

A. Plasma cortisol alone because aldosterone excess may not occur with a low potassium level

B. A dexamethasone suppression test as the first test in all hypokalemic hypertension

C. Plasma aldosterone concentration interpreted with renin to calculate an aldosterone-to-renin ratio

D. Random urinary metanephrines without considering adrenergic symptoms

E. Renal biopsy to look for juxtaglomerular-cell hyperplasia

83. A critically ill patient has increasing abdominal distention, high ventilator pressures, oliguria, and worsening hypotension. Bladder pressure is markedly elevated. Which diagnosis is most likely?

A. Abdominal compartment syndrome

B. Minimal-change disease

C. Acute poststreptococcal glomerulonephritis

D. Renal artery fibromuscular dysplasia

E. Central diabetes insipidus

84. A patient with profuse diarrhea has pH 7.28, bicarbonate 15 mEq/L, chloride 114 mEq/L, and a normal anion gap. Which mechanism is most likely?

A. Primary mineralocorticoid excess causing hydrogen loss and metabolic alkalosis

B. Primary respiratory acidosis with renal bicarbonate retention as compensation

C. Accumulation of lactate producing a high-anion-gap metabolic acidosis

D. Gastrointestinal bicarbonate loss producing a hyperchloremic metabolic acidosis

E. Loss of gastric hydrochloric acid producing chloride-responsive metabolic alkalosis

85. A patient with diabetic CKD remains albuminuric despite an ACE inhibitor and an SGLT2 inhibitor. Potassium is 4.4 mEq/L and eGFR is adequate for therapy. Which additional drug may reduce kidney and cardiovascular risk?

A. A high-dose NSAID to reduce glomerular prostaglandin synthesis

B. A direct renin inhibitor added to dual RAAS blockade as routine therapy

C. A potassium supplement to prevent mineralocorticoid receptor activation

D. A loop diuretic solely to suppress albumin excretion in the absence of volume overload

E. A nonsteroidal mineralocorticoid receptor antagonist such as finerenone

86. A patient loses substantial muscle mass during a prolonged ICU stay. Serum creatinine returns near baseline, but kidney recovery is uncertain. Why can creatinine be misleading?

A. Dialysis permanently suppresses creatinine assay detection

B. Reduced creatinine generation can make GFR appear better than it is

C. Muscle loss increases creatinine production and therefore underestimates kidney function

D. Low muscle mass causes severe albuminuria that falsely lowers serum creatinine

E. Creatinine becomes completely independent of filtration during critical illness

87. A transplant recipient with stable creatinine develops new donor-specific antibodies but no overt dysfunction. Why is follow-up important?

A. The antibodies indicate BK infection rather than alloimmunity

B. Donor-specific antibodies are commonly benign once the first posttransplant year has passed

C. Alloantibodies are unlikely to affect the graft until serum creatinine has already doubled

D. The antibodies prove immediate graft failure and require nephrectomy

E. Donor-specific antibodies can precede clinically apparent antibody-mediated allograft injury

88. A 54-year-old man with poorly controlled diabetes has serum sodium 128 mEq/L, glucose 720 mg/dL, and measured serum osmolality 318 mOsm/kg. Which interpretation is most accurate?

A. The sodium confirms true hypotonic hyponatremia requiring immediate free-water restriction

B. The low sodium strongly suggests renal salt wasting and requires mineralocorticoid replacement

C. The low sodium is largely translocational hyponatremia from severe hyperglycemia

D. The finding represents pseudohyponatremia from excess plasma lipids rather than glucose

E. The patient has classic SIADH because the sodium is low despite a high serum osmolality

89. A 79-year-old nursing-home resident with limited access to water has serum sodium 158 mEq/L and urine osmolality 820 mOsm/kg. Which mechanism is most likely?

A. Lithium-induced nephrogenic diabetes insipidus producing resistance to vasopressin

B. Extrarenal or inadequate water intake with preserved renal concentrating ability

C. Primary polydipsia causing persistent ingestion of electrolyte-free water

D. Central diabetes insipidus causing failure to concentrate urine during hypernatremia

E. Osmotic diuresis from glucose producing a urine that is maximally dilute

90. A patient with septic AKI has a fractional excretion of sodium below 1%. Which interpretation is best?

A. A low FENa does not exclude intrinsic AKI in sepsis and must be interpreted in context

B. A low FENa strongly suggests kidney function will normalize within hours

C. FENa is diagnostic of obstruction when below 1%

D. FENa below 1% strongly suggests prerenal azotemia with no tubular injury

E. FENa is unaffected by diuretics, CKD, or sepsis

91. A patient with advanced CKD is receiving several drugs and develops confusion and myoclonus after gabapentin dose escalation. What is the likely problem?

A. Drug accumulation from inadequate renal dose adjustment

B. Increased hepatic clearance caused by reduced GFR

C. Loss of gabapentin through glomerular hyperfiltration

D. An allergic reaction that is unrelated to kidney function or dosing

E. Direct stimulation of erythropoietin causing neurologic toxicity

92. A patient on citrate-anticoagulated CRRT develops metabolic alkalosis with stable ionized calcium and no evidence of citrate accumulation. Which mechanism is possible?

A. Citrate directly causes chloride retention and normal-gap acidosis

B. Regional citrate anticoagulation may not alter systemic acid-base status

C. Excess citrate metabolism generates bicarbonate equivalents

D. The alkalosis strongly suggests dialysate contains no bicarbonate

E. Citrate metabolism consumes bicarbonate and commonly causes acidosis

93. A patient at very high risk for tumor lysis syndrome has a markedly elevated uric acid concentration. Which agent rapidly degrades existing uric acid?

A. Rasburicase

B. Desmopressin

C. Hydrochlorothiazide

D. Spironolactone

E. Allopurinol

94. A muscular 34-year-old man has a creatinine-based eGFR of 58 mL/min/1.73 m2 but no albuminuria or structural kidney abnormality. The clinician suspects creatinine is misleading. Which test can improve GFR estimation?

A. A serum urea concentration interpreted without clinical context

B. A spot urine sodium concentration without a timed collection

C. A urine dipstick for glucose as a direct measure of filtration

D. A serum albumin concentration used as a substitute for GFR

E. A cystatin C-based estimate, alone or combined with creatinine

95. A kidney transplant recipient develops delayed graft function and requires dialysis during the first week. Which injury is a common cause in a deceased-donor graft?

A. Recurrent membranous nephropathy as a primary cause of early graft dysfunction

B. Chronic antibody-mediated rejection that develops before implantation

C. BK virus nephropathy before viral replication can occur

D. Tertiary hyperparathyroidism causing acute tubular obstruction

E. Ischemia-reperfusion acute tubular injury

96. A patient with potassium 7.1 mEq/L has peaked T waves and QRS widening. Which therapy should be given first to reduce immediate arrhythmic risk?

A. Intravenous calcium to stabilize the cardiac membrane

B. Fludrocortisone because mineralocorticoid replacement acts within minutes

C. Dietary potassium restriction because ECG toxicity resolves promptly with intake reduction

D. A loop diuretic before any cardiac membrane stabilization

E. Oral sodium polystyrene sulfonate as the primary emergency intervention

97. A patient with anti-GBM disease has pulmonary hemorrhage and rapidly progressive kidney failure. Which treatment strategy is standard?

A. RAAS blockade as a primary therapy for active crescentic nephritis

B. Supportive care alone because anti-GBM antibodies disappear without treatment

C. Long-term antibiotics because the disease is caused by persistent bacterial infection

D. Plasma exchange plus immunosuppression to remove antibody and stop further production

E. High-dose diuretics without immunosuppression or antibody removal

98. A patient with active proliferative lupus nephritis is starting therapy. Which approach reflects contemporary management?

A. Stop hydroxychloroquine in all patients because it worsens kidney outcomes

B. Use glucocorticoids with an effective immunosuppressive backbone, with selected add-on agents based on disease and patient factors

C. Treat primarily with an ACE inhibitor because immunosuppression has no role in proliferative disease

D. Delay therapy until irreversible scarring is established on repeat biopsy

E. Use chronic NSAIDs as the principal therapy for active glomerular inflammation

99. A patient receiving cisplatin has persistent hypomagnesemia despite oral supplementation. Which mechanism is most likely?

A. Cisplatin-induced renal magnesium wasting from tubular injury

B. Pseudohypomagnesemia caused by marked hyperlipidemia in the laboratory sample

C. Impaired renal filtration causing magnesium retention rather than loss

D. Redistribution of magnesium into bone caused by acute parathyroid hormone excess

E. Reduced intestinal magnesium absorption caused by blockade of vitamin D receptors

100. A 16-year-old boy has persistent glomerular hematuria. His mother has microscopic hematuria, and several maternal male relatives reached kidney failure early in adulthood. He also has high-frequency sensorineural hearing loss. Pathogenic variation in which gene is most likely?

A. PKD1

B. SLC12A3

C. NPHS1

D. COL4A5

E. UMOD

101. Eight months after kidney transplantation, a patient has biopsy-proven monomorphic EBV-positive post-transplant lymphoproliferative disorder. The graft is functioning and there is no immediately life-threatening organ compromise. Which management principle is generally part of initial therapy?

A. Increase mycophenolate to restore antiviral T-cell function

B. Give high-dose calcineurin inhibitor monotherapy

C. Treat with plasma exchange for presumed alloantibody injury

D. Reduce immunosuppression when feasible and add lymphoma-directed therapy as indicated

E. Remove the allograft as the routine first intervention

102. A patient with licorice overuse has hypertension, hypokalemia, metabolic alkalosis, low renin, and low aldosterone. Which mechanism is responsible?

A. Inhibition of 11-beta-hydroxysteroid dehydrogenase permits cortisol to activate mineralocorticoid receptors

B. Loss of epithelial sodium channels causing renal sodium wasting and secondary hyperaldosteronism

C. Impaired thick ascending-limb transport causing prostaglandin-mediated salt wasting

D. Excess aldosterone secretion from the adrenal gland despite suppression of renin

E. Deficient cortisol production causing increased ACTH without mineralocorticoid receptor activation

103. A patient with decompensated systolic heart failure has edema and serum sodium 124 mEq/L. Which urine finding is most likely before diuretics are given?

A. A relatively concentrated urine due to persistent nonosmotic vasopressin activity

B. A urine osmolality near plasma because heart failure abolishes medullary concentration gradients

C. Profound bicarbonaturia from proximal tubular dysfunction caused by venous congestion

D. Persistent glucosuria causing osmotic diuresis despite a normal plasma glucose level

E. A maximally dilute urine because arterial underfilling fully suppresses vasopressin release

104. A patient with long-standing inflammatory arthritis develops nephrotic syndrome. Biopsy shows Congo red-positive deposits with apple-green birefringence. Which diagnosis is most likely?

A. Minimal-change disease

B. Inflammation-associated AA amyloidosis

C. Thin basement membrane nephropathy

D. Anti-GBM nephritis

E. AL amyloidosis from plasma-cell dyscrasia

105. A 49-year-old man with chronic heavy beer intake and very poor food consumption has serum sodium 119 mEq/L. Urine osmolality is 85 mOsm/kg. Which factor most limits his ability to excrete free water?

A. Marked hyperglycemia creates a transcellular water shift despite a normal blood glucose concentration

B. Extremely low daily solute intake limits the maximal volume of dilute urine that can be produced

C. Severe autonomous ADH secretion prevents urine dilution despite a low serum osmolality

D. Renal failure prevents any urine formation even though measured urine output is preserved

E. Primary hyperaldosteronism causes renal sodium retention and directly lowers serum sodium

106. A patient with CKD has eGFR 25 mL/min/1.73 m2 and asks whether creatinine alone reflects kidney health. Which response is best?

A. Imaging may not provide information relevant to chronic kidney disease

B. The cause of CKD does not influence prognosis or management

C. Serum creatinine is a principal clinically useful measure of kidney disease severity

D. CKD assessment also includes albuminuria, urine findings, imaging, cause, complications, and trajectory

E. Urine albumin is unnecessary once serum creatinine is elevated

107. A patient with multiple myeloma develops AKI and very high circulating free light chains. Which intrarenal lesion is a major concern?

A. Distal renal tubular acidosis

B. Renal artery stenosis

C. Papillary necrosis from sickling

D. Minimal-change disease

E. Light-chain cast nephropathy

108. A young adult has persistent microscopic hematuria, normal kidney function, and several relatives with similar benign findings. Biopsy shows diffusely thin glomerular basement membranes without other lesions. Which diagnosis is most likely?

A. Minimal-change disease

B. Thin-GBM nephropathy

C. Membranous nephropathy

D. Anti-GBM disease

E. C3 glomerulopathy

109. A patient with severe symptomatic hyponatremia receives a small bolus of 3% saline. What is the immediate therapeutic goal?

A. Complete elimination of urinary sodium to prevent subsequent osmotic demyelination

B. A modest early rise in serum sodium sufficient to reduce cerebral edema and severe symptoms

C. Normalization of serum sodium within the first hour even with chronicity or risk factors

D. Rapid correction of all associated potassium abnormalities before any sodium change is allowed

E. A fall in serum sodium to restore intracellular brain water before neurologic recovery

110. A patient whose native-kidney disease was primary membranous nephropathy develops rising proteinuria after transplantation. Which concern is appropriate?

A. Calcineurin-inhibitor toxicity as the predominant cause of posttransplant proteinuria

B. Hyperacute rejection can first appear several years after transplantation

C. BK nephropathy commonly causes isolated nephrotic syndrome without tubular injury

D. Recurrence is unlikely because disease-associated antibodies generally do not injure a donor kidney

E. Recurrent membranous nephropathy causing proteinuria in the renal allograft

111. A patient with a functioning arteriovenous fistula has loss of the previously continuous thrill. What should be suspected?

A. Peritoneal membrane failure causing reduced fistula vibration

B. Acute access thrombosis or severe flow-limiting dysfunction

C. Normal maturation of the fistula after repeated dialysis sessions

D. Improved access flow because a strong thrill disappears when resistance falls

E. Dialysis disequilibrium syndrome producing transient loss of the bruit

112. A 35-year-old patient develops polyuria after pituitary surgery. Serum sodium rises to 151 mEq/L, urine osmolality is 95 mOsm/kg, and urine osmolality increases markedly after desmopressin. What is the diagnosis?

A. Central diabetes insipidus due to deficient vasopressin secretion

B. Osmotic diuresis from glucosuria causing a high rather than very low urine osmolality

C. Nephrogenic diabetes insipidus due to renal resistance to circulating vasopressin

D. Mineralocorticoid excess causing renal sodium retention without a water diuresis

E. Primary polydipsia with intact concentrating ability after prolonged water loading

113. A woman with systemic sclerosis develops abrupt severe hypertension, AKI, and microangiopathic hemolytic anemia. Which treatment is most important?

A. Avoid all RAAS blockade because creatinine elevation is a contraindication

B. Mineralocorticoid replacement because aldosterone is deficient

C. High-dose glucocorticoid escalation as the primary renal treatment

D. Prompt ACE inhibitor therapy for scleroderma renal crisis

E. Immediate nephrectomy because the injury is caused by unilateral renal ischemia

114. A 72-year-old man with metastatic lung cancer has serum sodium 123 mEq/L and serum osmolality 262 mOsm/kg. Urine osmolality is 610 mOsm/kg and urine sodium 72 mEq/L. He appears euvolemic and has normal thyroid and morning cortisol testing. Which initial chronic strategy is most appropriate if symptoms are mild?

A. Increase free-water intake because concentrated urine indicates a primary water deficit

B. Give fludrocortisone because euvolemic hyponatremia usually reflects mineralocorticoid deficiency

C. Start rapid continuous hypertonic saline until the serum sodium reaches the normal range

D. Administer hypotonic saline because renal sodium excretion is inappropriately increased

E. Restrict free-water intake and address the underlying cause of persistent SIADH

115. A patient has thrombocytopenia, schistocytes, elevated LDH, low haptoglobin, neurologic symptoms, and kidney injury. Which syndrome is present?

A. Acute interstitial nephritis

B. Thrombotic microangiopathy

C. Distal renal tubular acidosis

D. Minimal-change disease

E. Nephrotic syndrome

116. A patient with CKD and heart failure has persistent edema. Why can serum creatinine transiently rise during effective decongestion without necessarily indicating tubular injury?

A. A rise in creatinine strongly suggests occult glomerulonephritis and mandates kidney biopsy

B. Hemoconcentration and hemodynamic changes can lower filtration while venous congestion improves

C. Creatinine becomes unrelated to GFR during diuresis because muscles stop producing it

D. Diuretics commonly cause acute tubular necrosis whenever creatinine rises by any amount

E. Improved venous congestion commonly increases creatinine through direct nephron toxicity

117. A patient with AKI has fever, rash, arthralgias, eosinophilia, and pyuria after starting an antibiotic. Which diagnosis is most likely?

A. Acute tubular injury from ischemia

B. Bilateral renal artery stenosis

C. Prerenal azotemia

D. Acute interstitial nephritis

E. Nephrotic syndrome

118. A kidney biopsy shows a membranoproliferative pattern with intense C3 staining but little immunoglobulin. Serum C3 remains depressed months after an infectious illness has resolved. Which pathophysiologic process best explains this pattern?

A. Selective podocyte injury without activation of complement

B. Autoantibodies directed against the glomerular basement membrane

C. Persistent dysregulation of the alternative complement pathway

D. Mesangial trapping of polymeric IgA as the dominant process

E. Deposition of Congo red-positive immunoglobulin fibrils

119. A patient with severe salicylate intoxication has confusion, pulmonary edema, metabolic acidosis, and rising salicylate concentration despite treatment. Which therapy should be strongly considered?

A. Isotonic saline alone because salicylate is not dialyzable

B. Hemodialysis to rapidly remove salicylate and correct acid-base and volume abnormalities

C. Immediate intubation without attention to maintaining compensatory hyperventilation

D. Potassium supplementation as a primary definitive method of salicylate removal

E. Activated charcoal alone even when severe systemic toxicity is progressing

120. A patient with severe diarrhea has bicarbonate 12 mEq/L and an arterial PaCO2 of 40 mm Hg. Using Winter's formula, which acid-base interpretation is most accurate?

A. The measured PaCO2 is appropriate for a simple metabolic acidosis

B. A concurrent metabolic alkalosis is present

C. A concurrent respiratory alkalosis is present

D. The data indicate a simple chronic respiratory acidosis

E. A metabolic acidosis with superimposed respiratory acidosis is present

121. A patient with diabetic CKD asks why smoking cessation matters for kidney care. What is the best response?

A. Tobacco exposure affects lung disease but has no meaningful relationship to CKD risk

B. Smoking is recommended if blood pressure is controlled because nicotine reduces albuminuria

C. Smoking cessation is relevant primarily after dialysis has been initiated

D. Smoking improves renal perfusion and therefore slows CKD progression

E. Smoking increases cardiovascular and kidney risk, so cessation is part of comprehensive CKD care

122. A young patient has hypertension, low renin, low aldosterone, hypokalemia, and metabolic alkalosis. Which drug is most mechanistically appropriate for suspected Liddle syndrome?

A. Amiloride to directly block the epithelial sodium channel responsible for Liddle syndrome

B. Hydrocortisone because cortisol deficiency causes the sodium-retaining phenotype

C. Furosemide because loop blockade corrects the inherited epithelial sodium-channel defect

D. Desmopressin because vasopressin excess drives distal sodium reabsorption

E. Spironolactone because aldosterone receptor blockade is reliably effective despite low aldosterone

123. A postpartum patient after massive hemorrhage and disseminated intravascular coagulation remains anuric. Imaging shows absent cortical perfusion with preserved medullary flow. Which diagnosis is most likely?

A. Central diabetes insipidus

B. Minimal-change disease

C. Acute cystitis

D. Prerenal azotemia

E. Renal cortical necrosis

124. A patient with CKD works in a battery-recycling facility and has abdominal pain, hypertension, anemia, and gout. Which exposure should be considered?

A. Radon exposure causing selective glomerular basement membrane thinning

B. Carbon monoxide exposure causing chronic renal phosphate wasting

C. Chronic lead exposure causing tubulointerstitial kidney injury

D. Asbestos exposure causing primarily cystic kidney disease

E. Silica exposure causing isolated nephrotic syndrome without hypertension

125. A kidney transplant recipient has persistent hyperparathyroidism and hypercalcemia months after transplantation despite good graft function. Which diagnosis is most likely?

A. Tertiary hyperparathyroidism from autonomous parathyroid hyperplasia

B. Hypoparathyroidism from restoration of renal calcitriol production

C. Primary renal phosphate wasting causing low parathyroid hormone

D. BK virus infection causing parathyroid hormone suppression

E. Acute rejection causing calcium release from the graft

126. A dialysis patient has functional iron deficiency with low transferrin saturation but ferritin that is not low. Which mechanism can explain this pattern?

A. Hyperparathyroidism increases transferrin synthesis enough to mask complete iron depletion

B. Inflammation-driven hepcidin elevation limits iron release and availability for erythropoiesis

C. Renal erythropoietin excess traps iron inside mature red cells before marrow uptake

D. Dialysis removes hepcidin completely, so functional iron deficiency may not occur

E. Low hepcidin causes excessive iron export from macrophages and profound serum iron excess

127. A patient in shock receives multiple vasopressors but remains oliguric after adequate volume assessment. Which statement about norepinephrine is most accurate?

A. Norepinephrine commonly causes renal ischemia and should rarely be used in septic shock

B. Renal perfusion is independent of systemic pressure once sepsis develops

C. Restoring adequate mean arterial pressure with norepinephrine can support renal perfusion in vasodilatory shock

D. Dopamine is preferred solely because it selectively increases kidney blood flow at low doses

E. All vasopressors should be stopped whenever urine output falls below 0.5 mL/kg/h

128. A patient with advanced kidney failure takes magnesium-containing laxatives and develops lethargy, hypotension, and bradycardia. Which abnormality is most likely?

A. Hypophosphatemia from renal phosphate wasting caused by magnesium excess

B. Hypermagnesemia from impaired renal excretion combined with increased magnesium intake

C. Hypomagnesemia from gastrointestinal binding of magnesium by the laxative

D. Hypernatremia from osmotic water shifts generated by serum magnesium

E. Hypercalcemia from magnesium-induced parathyroid hormone stimulation

129. A patient with kidney failure needs a potent opioid for severe pain. Which agent is often favored because it lacks clinically important renally cleared active metabolites?

A. Meperidine

B. Fentanyl

C. Propoxyphene

D. Morphine

E. Codeine

130. A hemodialysis patient has fever and purulent drainage from a tunneled dialysis catheter. What is the key concern?

A. Normal exit-site inflammation that does not require cultures or antibiotics

B. Dialysis disequilibrium syndrome caused by the presence of the catheter

C. Access steal syndrome because catheters divert arterial blood from the limb

D. Catheter-related bloodstream infection with risk of bacteremia and metastatic infection

E. Secondary hyperparathyroidism because PTH causes purulent catheter drainage

131. A 62-year-old man has repeated office blood pressures near 155/92 mm Hg but normal home readings and no target-organ damage. Which next step best clarifies the diagnosis?

A. Begin evaluation for pheochromocytoma before confirming out-of-office blood pressure

B. Start four antihypertensive drugs without confirming whether hypertension persists outside clinic

C. Assume the home device is inaccurate because office readings are commonly definitive

D. Diagnose resistant hypertension because office readings are above goal

E. Obtain ambulatory or validated home blood-pressure measurements to assess for white-coat hypertension

132. A patient with systemic lupus erythematosus develops proteinuria, hematuria, low complement, and active urine sediment. What is the most appropriate next diagnostic step when kidney involvement is clinically significant?

A. Begin long-term antibiotics because low complement strongly suggests postinfectious nephritis

B. Delay evaluation until nephrotic syndrome or dialysis dependence develops

C. Use serum creatinine alone because urine abnormalities do not affect lupus classification

D. Assume minimal-change disease and treat without defining the lesion

E. Kidney biopsy to classify lupus nephritis and guide treatment

133. A patient with severe hypertension develops confusion, seizures, papilledema, and acute kidney injury. Which management principle is most appropriate?

A. Treat primarily the kidney injury because neurologic findings do not affect urgency

B. Use controlled intravenous blood-pressure reduction because hypertensive emergency has acute target-organ injury

C. Normalize blood pressure immediately to a low-normal level within minutes in most cases

D. Use primarily oral therapy because intravenous agents worsen renal autoregulation

E. Delay treatment until the creatinine returns to baseline

134. A 48-year-old man has nephrotic-range proteinuria. Kidney biopsy shows diffuse capillary-wall thickening, subepithelial immune deposits, and granular IgG along the glomerular basement membrane. Which target antigen is most commonly associated with primary membranous nephropathy?

A. Myeloperoxidase

B. Alpha-3 chain of type IV collagen

C. Phospholipase A2 receptor

D. Double-stranded DNA

E. Proteinase 3

135. A patient has blood pressure 185/110 mm Hg but no symptoms, normal neurologic examination, no pulmonary edema, and no evidence of acute kidney injury. Which statement is most accurate?

A. Intravenous vasodilators are required even without acute target-organ injury

B. The patient has a hypertensive emergency solely because the systolic pressure exceeds 180

C. The absence of symptoms strongly suggests the blood pressure measurement is false

D. Blood pressure should be reduced to below 120/80 within one hour

E. Severe asymptomatic hypertension usually does not require rapid intravenous normalization

136. An adult with nephrotic syndrome has a history of Hodgkin lymphoma. Which glomerular lesion is classically associated?

A. Scleroderma renal crisis

B. Minimal-change disease

C. Anti-GBM nephritis

D. C3 glomerulopathy

E. Alport syndrome

137. A transplant recipient three months after surgery develops fever, malaise, leukopenia, and diarrhea. CMV PCR is positive. Which diagnosis is most likely?

A. CMV syndrome after transplantation

B. Acute antibody-mediated rejection

C. BK nephropathy

D. Recurrent FSGS

E. Tertiary hyperparathyroidism

138. A 59-year-old woman with type 2 diabetes has eGFR 42 mL/min/1.73 m2 and albuminuria despite maximally tolerated RAAS blockade. Which medication class has strong evidence for slowing CKD progression independent of glucose lowering?

A. A short-acting nitrate because venodilation prevents glomerular hyperfiltration

B. A nonselective NSAID because afferent vasoconstriction reduces protein filtration

C. A thiazolidinedione because edema improves kidney perfusion in albuminuric CKD

D. An SGLT2 inhibitor when no contraindication is present

E. A sulfonylurea because insulin secretion directly lowers intraglomerular pressure

139. A dialysis patient receiving gabapentin develops somnolence and myoclonus. Which pharmacokinetic property is most relevant?

A. Gabapentin is highly protein bound and is minimally removed during hemodialysis

B. Gabapentin is renally eliminated and requires substantial dose adjustment in kidney failure

C. Kidney failure converts gabapentin into a nephrotoxic metabolite in the liver

D. Gabapentin is cleared primarily through bile and does not require renal dosing

E. Hemodialysis increases gabapentin half-life by preventing hepatic metabolism

140. During hemodialysis a patient develops sudden dyspnea, chest pain, and a sense of impending doom within minutes of starting a new dialyzer. Which complication is most likely?

A. Secondary hyperparathyroidism causing acute bronchospasm at dialysis initiation

B. Calciphylaxis causing painful skin ischemia during ultrafiltration

C. Chronic aluminum toxicity causing progressive neurologic decline over months

D. Dialysis disequilibrium syndrome from gradual cerebral edema late in a first treatment

E. Acute hypersensitivity reaction to the dialysis membrane or circuit

141. A patient with diabetic ketoacidosis has serum potassium 5.6 mEq/L before insulin is given. Which statement best explains this finding?

A. Total-body potassium is depleted despite extracellular potassium elevation from insulin deficiency and acidosis

B. Hyperkalemia strongly suggests that potassium replacement will rarely be needed during treatment

C. The elevated potassium is caused primarily by pseudohyperkalemia from platelet release in the tube

D. Insulin deficiency reduces potassium movement out of cells and therefore lowers serum potassium

E. Total-body potassium is increased because osmotic diuresis prevents urinary potassium loss

142. A transplant recipient years after surgery has slowly worsening kidney function, proteinuria, donor-specific antibodies, and biopsy showing transplant glomerulopathy. Which process is most likely?

A. Hyperacute rejection

B. Immediate surgical vascular thrombosis

C. Chronic active antibody-mediated rejection

D. Isolated acute pyelonephritis

E. Early ischemia-reperfusion injury

143. A patient with CKD and diabetes has glycemic values that appear improved as kidney function declines, despite no change in medication. Which factor can contribute?

A. Reduced renal insulin clearance can increase insulin exposure and hypoglycemia risk

B. Albuminuria directly destroys circulating insulin before it reaches tissues

C. CKD prevents hypoglycemia because renal gluconeogenesis becomes excessive

D. The kidney becomes the dominant source of insulin as GFR falls

E. Reduced GFR accelerates insulin degradation and raises insulin requirements in all patients

144. A long-term peritoneal dialysis patient has increasing volume overload despite stronger glucose solutions. Peritoneal testing suggests high transport and reduced ultrafiltration. What complication is present?

A. Uremic pericarditis from initial dialysis

B. Dialysis disequilibrium syndrome

C. Peritoneal ultrafiltration failure

D. Arteriovenous fistula outflow stenosis

E. Hemodialysis water contamination

145. A patient with severe ethylene glycol poisoning has high-anion-gap acidosis, AKI, and calcium oxalate crystals. Which treatment combination is appropriate in severe toxicity?

A. Desmopressin plus loop diuretics because the primary lesion is water intoxication

B. Urine acidification and observation because toxic metabolites are harmless

C. Flumazenil plus charcoal because ethylene glycol is a benzodiazepine analogue

D. Alcohol dehydrogenase blockade with fomepizole plus hemodialysis when indicated

E. High-dose calcium supplements as the primary therapy to dissolve oxalate crystals

146. A 76-year-old patient has very high cuff blood-pressure readings but no expected symptoms or retinal findings. The radial artery remains palpable despite cuff inflation above systolic pressure. Which phenomenon may be present?

A. Pseudohypertension from markedly noncompressible calcified arteries

B. Renovascular hypertension caused by a unilateral renal infarct

C. White-coat hypertension caused by anxiety during office blood-pressure measurement

D. Masked hypertension caused by normal office readings

E. Isolated diastolic hypertension caused by increased arterial compliance

147. A patient on intermittent hemodialysis for AKI repeatedly becomes hypotensive during ultrafiltration. Which prescription change may help while fluid removal remains necessary?

A. Increase the target fluid removal even with intradialytic symptoms

B. Reduce the ultrafiltration rate by extending treatment time or using a slower modality

C. Use a smaller blood-pressure cuff because the hypotension is usually measurement artifact

D. Lower sodium intake after dialysis while leaving the same rapid ultrafiltration prescription unchanged

E. Shorten the treatment and remove the same volume faster

148. An adult has severe thrombocytopenia, microangiopathic hemolytic anemia, confusion, and relatively modest kidney injury. ADAMTS13 activity is severely deficient. Which diagnosis is most likely?

A. Scleroderma renal crisis

B. Renal vein thrombosis

C. Thrombotic thrombocytopenic purpura

D. Shiga toxin-associated HUS

E. Complement-mediated atypical HUS

149. A patient with kidney failure has a willing but ABO-incompatible living donor. Which strategy can expand transplantation opportunities?

A. Delay transplantation until the recipient has completed ten years of dialysis

B. ABO incompatibility generally precludes living donation because desensitization is ineffective

C. Conversion of the donor to a deceased-donor allocation pathway

D. Routine transplantation without immunologic assessment because living donors do not cause rejection

E. Kidney paired donation or another compatible exchange pathway

150. A patient with severe AKI is hemodynamically unstable on multiple vasopressors and requires dialysis. Which modality is often better tolerated?

A. No kidney replacement therapy because vasopressor use is an absolute contraindication

B. Continuous kidney replacement therapy with slower fluid and solute shifts

C. Peritoneal lavage without dialysate because solute removal is unnecessary

D. Daily contrast administration to increase osmotic clearance

E. A brief high-efficiency intermittent treatment with maximal ultrafiltration

151. A 61-year-old man taking lithium has persistent polyuria, serum sodium 149 mEq/L, and urine osmolality 130 mOsm/kg that changes minimally after desmopressin. Which therapy is most specific for this mechanism?

A. Mannitol to create an osmotic diuresis and thereby reduce the existing urine volume

B. Spironolactone solely to block aldosterone-mediated sodium reabsorption in the distal nephron

C. Desmopressin escalation because the kidneys remain fully responsive to exogenous vasopressin

D. Acetazolamide to increase bicarbonate delivery and thereby enhance collecting-duct water uptake

E. Amiloride to reduce lithium entry through epithelial sodium channels in collecting ducts

152. A hemodialysis patient repeatedly becomes lightheaded and hypotensive during the final hour of treatment after gaining 4 to 5 kg between sessions. There is no fever, bleeding, or arrhythmia. Which strategy most directly addresses the recurring mechanism?

A. Raise the prescribed ultrafiltration rate to reach target weight earlier

B. Increase interdialytic sodium intake to support blood pressure

C. Lower the ultrafiltration rate by reducing interdialytic weight gain or extending treatment time

D. Shorten dialysis sessions while removing the same fluid volume

E. Administer routine fluid boluses before most dialysis sessions even when volume overloaded

153. A long-term dialysis patient with acquired cystic kidney disease develops painless gross hematuria. Which complication is particularly important to exclude?

A. Renal tubular acidosis

B. Renal cell carcinoma

C. Minimal-change disease

D. Liddle syndrome

E. Primary polydipsia

154. A patient with kidney failure has a severe lithium intoxication with neurologic symptoms and a very high serum concentration. Which extracorporeal therapy is effective?

A. Peritoneal lavage because lithium is stored primarily in the peritoneal cavity

B. Hemodialysis because lithium is small, water soluble, and poorly protein bound

C. Plasmapheresis because lithium is almost completely albumin bound

D. No extracorporeal therapy works because lithium has an extremely large protein-bound fraction

E. Hemoperfusion is required because lithium may not cross dialysis membranes

155. A kidney transplant recipient has BK viremia with biopsy-proven BK nephropathy. What is the main treatment principle?

A. Remove the graft immediately because BK nephropathy may not improve

B. Give valganciclovir because it reliably eradicates BK virus

C. Begin high-dose glucocorticoids as routine first-line therapy without reconsidering immunosuppression

D. Intensify T-cell immunosuppression because BK disease reflects under-immunosuppression

E. Carefully reduce immunosuppression while monitoring viral load and graft function

156. A patient with biopsy-proven IgA nephropathy has persistent proteinuria despite optimized supportive therapy and remains at high risk for progression. Which general treatment concept is current?

A. No therapy beyond observation is appropriate once kidney biopsy confirms IgA deposition

B. Escalate disease-specific therapy in selected high-risk patients after optimized supportive care

C. Use chronic high-dose NSAIDs to reduce glomerular filtration and protein excretion

D. Stop RAAS blockade because proteinuria is expected and should not be treated

E. Begin plasma exchange routinely in all stable patients with IgA nephropathy

157. A patient taking lithium develops tremor, confusion, and worsening kidney function after starting a thiazide diuretic. What explains the interaction?

A. The interaction is caused by direct precipitation of lithium with hydrochlorothiazide in urine

B. Thiazides induce hepatic lithium metabolism and produce a toxic intermediate

C. Thiazide-induced sodium loss increases proximal lithium reabsorption and raises lithium levels

D. Lithium blocks thiazide absorption from the intestine and causes diuretic withdrawal

E. Thiazides increase distal lithium secretion and therefore lower serum lithium

158. A patient on continuous venovenous hemofiltration is anticoagulated with regional citrate and develops rising total calcium relative to ionized calcium, metabolic acidosis, and escalating calcium infusion needs. What complication is most likely?

A. Acute phosphate toxicity

B. Hypermagnesemia from dialysate contamination

C. Citrate accumulation from impaired metabolism

D. Dialysis disequilibrium syndrome

E. Heparin-induced thrombocytopenia

159. A dialysis patient has very high PTH, bone pain, and subperiosteal bone resorption. Which renal osteodystrophy pattern is most likely?

A. Osteomalacia from defective mineralization with no relationship to PTH

B. Avascular necrosis caused solely by phosphate retention

C. Normal bone turnover because dialysis prevents parathyroid stimulation

D. Adynamic bone disease with suppressed bone formation and low PTH

E. High-turnover osteitis fibrosa from secondary hyperparathyroidism

160. A child has growth impairment, polyuria, hypophosphatemia, glycosuria with normal serum glucose, aminoaciduria, and bicarbonate wasting. Which nephron segment is primarily dysfunctional?

A. Proximal tubular epithelium

B. Thick ascending limb

C. Collecting-duct principal cells

D. Thin descending limb

E. Distal convoluted tubule

161. A patient has hypertension, hypokalemia, low renin, and high cortisol production that does not suppress appropriately. Which secondary cause is most likely?

A. Central diabetes insipidus

B. Bartter syndrome

C. Gitelman syndrome

D. Cushing syndrome

E. Primary polydipsia

162. A patient with advanced CKD asks about pregnancy. Which statement is most accurate?

A. All antihypertensive drugs are safe to continue without medication review before conception

B. Pregnancy in advanced CKD carries increased maternal and fetal risk and benefits from preconception multidisciplinary counseling

C. Pregnancy reliably improves kidney function and lowers proteinuria in advanced CKD

D. Pregnancy after kidney transplantation is exceedingly uncommon, so reproductive counseling has little clinical value

E. CKD has no effect on pregnancy outcomes if serum creatinine is stable

163. A 43-year-old marathon runner drinks large volumes of water during a race and develops confusion. Serum sodium is 121 mEq/L and urine osmolality is 70 mOsm/kg. Which mechanism best explains the hyponatremia?

A. Marked hyperlipidemia displaced plasma water and produced pseudohyponatremia with normal tonicity

B. Persistent autonomous ADH secretion caused highly concentrated urine despite water loading

C. Water intake exceeded renal excretory capacity while antidiuresis was relatively suppressed

D. Hyperglycemia shifted water from cells and created hypertonic rather than hypotonic hyponatremia

E. Renal sodium wasting from mineralocorticoid deficiency produced sustained volume contraction

164. A 27-year-old man develops gross hematuria two days after an upper respiratory infection. Complement is normal and kidney function is preserved. Which diagnosis is most likely?

A. IgA nephropathy

B. Anti-GBM disease

C. Poststreptococcal glomerulonephritis

D. Minimal-change disease

E. Membranous nephropathy

165. A malnourished patient with chronic hyponatremia has serum sodium corrected from 108 to 121 mEq/L during the first 14 hours of treatment. He is now clinically improved. What is the most important next concern?

A. Continue correction at the same rate because neurologic recovery excludes treatment toxicity

B. Lower the sodium to below 100 mEq/L because any increase after chronic hyponatremia is harmful

C. Prevent further rapid correction because he is at high risk for osmotic demyelination

D. Give a large isotonic saline bolus because chronic hyponatremia commonly reflects hypovolemia

E. Begin potassium restriction because osmotic demyelination is driven primarily by hyperkalemia

166. A patient with an ileostomy has recurrent radiolucent stones and persistently acidic urine. Which stone composition is most likely?

A. Struvite

B. Uric acid

C. Cystine

D. Calcium phosphate

E. Magnesium silicate

167. A patient is found confused after ingesting an unknown toxic alcohol. Laboratory testing shows a high-anion-gap metabolic acidosis, an elevated osmolal gap, and calcium oxalate crystals in the urine. Which antidotal therapy is indicated while definitive management is arranged?

A. Fomepizole to inhibit alcohol dehydrogenase

B. N-acetylcysteine to replenish glutathione

C. Methylene blue to reduce methemoglobin

D. Atropine to reverse muscarinic excess

E. Hydroxocobalamin to bind cyanide

168. A 69-year-old smoker has abrupt worsening hypertension, recurrent flash pulmonary edema, and asymmetric kidney size. Which cause should be strongly considered?

A. Primary polydipsia

B. Central diabetes insipidus

C. Renal artery atherosclerosis

D. Gitelman syndrome

E. Minimal-change disease

169. A patient with subarachnoid hemorrhage develops marked hypertension. Which principle is most appropriate?

A. Use the usual chronic outpatient blood-pressure target immediately in most patients with subarachnoid hemorrhage

B. Induce profound hypotension to reduce renal perfusion and prevent vasospasm

C. Treat primarily if chronic kidney disease is also present

D. Ignore blood pressure because cerebral perfusion is independent of systemic pressure

E. Blood-pressure goals should account for the neurologic condition and risk of rebleeding or impaired cerebral perfusion

170. A patient with advanced CKD has pruritus without a primary skin eruption. Which statement is most accurate?

A. The symptom strongly suggests cholestatic liver disease and may not be related to dialysis

B. The presence of pruritus means dialysis adequacy is commonly normal

C. Uremic pruritus is common in advanced kidney disease and often requires multifactorial management

D. Uremic pruritus is caused solely by high serum sodium and resolves with free water

E. Pruritus excludes kidney failure because uremia affects primarily the gastrointestinal tract

171. A patient with multiple myeloma has serum sodium 126 mEq/L measured by an indirect ion-selective electrode, but measured serum osmolality is normal and there are no neurologic symptoms. What is the most likely explanation?

A. Central diabetes insipidus causing urinary water loss and an increased serum sodium

B. SIADH causing hypotonic hyponatremia with an inappropriately concentrated urine

C. Primary polydipsia causing excess electrolyte-free water with maximally dilute urine

D. Cerebral salt wasting causing true sodium depletion and extracellular volume contraction

E. Pseudohyponatremia from a reduced plasma-water fraction caused by marked paraproteinemia

172. A patient with hypertension develops a rise in creatinine and hyperkalemia after adding spironolactone to an ACE inhibitor. Which principle is most appropriate?

A. Continue dose escalation even with repeated potassium above 6 mEq/L

B. Hyperkalemia is unlikely when mineralocorticoid receptors are blocked

C. Add potassium supplements routinely to prevent a treatment-related potassium fall

D. Kidney function does not influence spironolactone adverse effects

E. Monitor potassium and kidney function closely when combining therapies that reduce aldosterone effect

173. A kidney transplant recipient develops nephrotic proteinuria within days of transplantation and had end-stage kidney disease from primary FSGS. What is a major concern?

A. De novo Alport syndrome caused by the donor kidney

B. Early recurrence of primary FSGS in the allograft

C. Recurrent diabetic nephropathy developing within several days

D. BK virus nephropathy presenting before immunosuppression begins

E. Chronic transplant glomerulopathy requiring years to develop

174. A transplant candidate has a high panel-reactive antibody level after prior pregnancies and transfusions. What does this finding imply?

A. She is protected from donor-specific antibodies by prior antigen exposure

B. She is broadly sensitized to HLA antigens and may have fewer compatible donor options

C. She has active antibody-mediated rejection before receiving a transplant

D. She has an obligate indication for combined kidney-liver transplantation

E. Kidney transplantation is generally not offered during pregnancy under any circumstances

175. A patient starts a proton-pump inhibitor and several weeks later develops AKI, sterile pyuria, white-cell casts, and mild eosinophilia. Which diagnosis is most likely?

A. Drug-induced acute interstitial nephritis

B. Postinfectious glomerulonephritis

C. Ischemic acute tubular injury

D. Renal artery thrombosis

E. Minimal-change disease

176. A young adult has hypertension and a bruit over the upper abdomen. Imaging shows a beaded appearance of the renal artery. Which disorder is most likely?

A. Renal vein thrombosis

B. Atherosclerotic renal artery stenosis

C. Fibromuscular dysplasia

D. Minimal-change disease

E. Polyarteritis nodosa limited to glomerular capillaries

177. A stable hemodialysis patient consistently gains 5 kg between sessions. Which counseling target is most directly relevant?

A. Avoid protein intake because urea generation is the principal cause of weight gain

B. Increase dietary sodium so thirst falls through expansion of extracellular volume

C. Stop all antihypertensive therapy because blood pressure control increases thirst

D. Drink extra free water before dialysis to improve ultrafiltration tolerance

E. Reduce dietary sodium and fluid intake to limit interdialytic weight gain

178. A pregnant patient with chronic hypertension is taking lisinopril when pregnancy is discovered. What is the best medication principle?

A. Stop all blood-pressure treatment because hypertension is physiologic in pregnancy

B. Discontinue ACE inhibitor therapy and use a pregnancy-compatible antihypertensive regimen

C. Add an ARB to reduce fetal exposure to a single RAAS blocker

D. Switch to direct renin inhibition because it is safer for fetal kidney development

E. Continue lisinopril because RAAS blockers are preferred throughout pregnancy

179. A patient with CKD and metabolic acidosis has bicarbonate 17 mEq/L on repeated measurements. Why is correction considered?

A. Low bicarbonate prevents hyperkalemia by increasing cellular potassium uptake

B. Bicarbonate therapy is used primarily to normalize urine pH and has no systemic rationale

C. Chronic acidosis reliably increases serum albumin and improves nutritional status

D. Chronic acidosis contributes to muscle catabolism, bone buffering, and adverse CKD outcomes

E. Acidosis protects bone by preventing calcium release from the skeleton

180. A 39-year-old patient has bilateral enlarged cystic kidneys, hypertension, and a family history of early kidney failure. Which extrarenal complication deserves targeted consideration based on personal and family history?

A. Aortic coarctation

B. Intracranial aneurysm

C. Achalasia

D. Addison disease

E. Myasthenia gravis

181. A patient with obesity, reduced nephron mass, and heavy proteinuria undergoes biopsy showing focal segmental glomerulosclerosis. Which mechanism is most likely?

A. Anti-GBM antibody deposition along the basement membrane

B. Secondary adaptive FSGS from glomerular hyperfiltration

C. Primary circulating-factor FSGS as the usual explanation in patients with obesity

D. Mesangial IgA deposition from mucosal immune activation

E. Subepithelial immune complexes caused by PLA2R autoantibodies

182. A patient with CKD and established atherosclerotic cardiovascular disease is not taking lipid-lowering therapy. Which approach is generally appropriate?

A. Use statin-based therapy unless there is a specific contraindication

B. Use niacin primarily because statins predictably accelerate CKD progression

C. Use fibrate monotherapy as the default even with triglyceride concentration

D. Wait until dialysis initiation because lipid treatment has no role before kidney failure

E. Avoid statins because reduced GFR makes LDL lowering ineffective

183. A patient with psychogenic polydipsia has serum sodium 129 mEq/L and urine osmolality 55 mOsm/kg. What does the urine osmolality indicate?

A. Persistent SIADH with failure of the collecting duct to excrete electrolyte-free water

B. Appropriate suppression of vasopressin with near-maximal urinary dilution

C. Severe nephrogenic diabetes insipidus despite the presence of hypotonic hyponatremia

D. Advanced kidney failure because a dilute urine is specific for severely reduced glomerular filtration

E. Renal salt wasting with marked activation of vasopressin from extracellular volume depletion

184. A CMV-seronegative kidney recipient receives a kidney from a CMV-seropositive donor. How is this pairing classified?

A. Equivalent risk to a donor-negative recipient-positive pairing in all circumstances

B. No CMV risk because donor organs do not transmit latent infection

C. Protective because donor antibodies transfer durable CMV immunity with the kidney

D. High risk for primary CMV infection after transplantation

E. Lowest-risk CMV pairing because the recipient has no prior immunity to reactivate

185. A patient with severe pancreatitis develops symptomatic hypocalcemia. Which process contributes?

A. Acute FGF23 deficiency producing isolated urinary calcium wasting

B. Calcium sequestration in areas of fat necrosis through saponification

C. Increased calcitriol production causing renal calcium loss despite intestinal absorption

D. Enhanced albumin synthesis binding ionized calcium and causing total hypercalcemia

E. Excess PTH secretion causing acute movement of calcium into necrotic tissue

186. A patient with AKI has urine output 0.2 mL/kg/h for 10 hours. What does this indicate?

A. AKI requires complete anuria for at least 24 hours

B. This urine output excludes AKI because some urine is still produced

C. The finding strongly suggests bilateral renal artery thrombosis

D. Urine output is not a major consideration when serum creatinine is available

E. The patient meets an oliguria criterion used in AKI staging

187. A patient with metabolic acidosis has bicarbonate 12 mEq/L. Which PaCO2 is closest to appropriate respiratory compensation by Winter's formula?

A. About 52 mm Hg

B. About 68 mm Hg

C. About 26 mm Hg

D. About 40 mm Hg

E. About 12 mm Hg

188. A patient with nephrotic syndrome has positive anti-PLA2R antibodies and biopsy showing subepithelial immune deposits. Which diagnosis is most likely?

A. IgA nephropathy

B. Anti-GBM disease

C. C3 glomerulopathy

D. Membranous nephropathy

E. Minimal-change disease

189. A peritoneal dialysis patient presents with abdominal pain and cloudy effluent. Dialysate cell count shows neutrophilic leukocytosis. What is the most likely diagnosis?

A. Peritoneal dialysis-associated peritonitis

B. Normal peritoneal equilibration during a long dwell

C. Dialysate glucose intolerance causing sterile turbidity without leukocytes

D. Hemodialysis access infection despite absence of vascular access

E. Encapsulating peritoneal sclerosis without active inflammation

190. A peritoneal dialysis patient has persistent hyperglycemia and weight gain after starting therapy. Which treatment feature contributes?

A. Loss of glucose exclusively into the dialysate causing chronic hypoglycemia

B. Systemic absorption of dialysate protein causing direct insulin resistance

C. Absorption of glucose from conventional dextrose-containing dialysate

D. Suppression of appetite caused by removal of all circulating glucose

E. Complete absence of carbohydrate in standard peritoneal dialysis solutions

191. A stable kidney transplant recipient starts clarithromycin and several days later tacrolimus level becomes markedly elevated with tremor and AKI. What explains the interaction?

A. Clarithromycin induces tacrolimus metabolism and causes under-immunosuppression

B. Tacrolimus blocks renal clearance of clarithromycin without changing tacrolimus levels

C. Macrolides activate P-glycoprotein and lower calcineurin-inhibitor absorption

D. The two drugs form insoluble crystals that obstruct the transplant ureter

E. Macrolide inhibition of CYP3A metabolism increases tacrolimus exposure

192. A patient with sickle cell disease develops gross hematuria and papillary necrosis. Which mechanism contributes?

A. Excess vasopressin causing cortical infarction

B. Renal vein thrombosis presenting predominantly with nephrotic-range proteinuria

C. Immune-complex deposition limited to glomerular capillaries

D. Medullary ischemia from sickling in the hypoxic, hypertonic renal medulla

E. Hyperaldosteronism causing papillary calcium deposition

193. A transplant candidate has a positive complement-dependent cytotoxic crossmatch against a prospective donor. What is the major concern?

A. The crossmatch primarily measures cytomegalovirus immunity rather than alloantibody

B. The donor kidney will have inadequate nephron mass even with antibody status

C. Preformed donor-reactive antibodies create a high risk of immediate antibody-mediated graft injury

D. The recipient has T-cell tolerance and therefore a lower rejection risk

E. The result predicts recurrent diabetic nephropathy rather than rejection

194. A patient with CKD is being prepared for possible dialysis. Why should upper-extremity veins be preserved when possible?

A. Central venous catheters improve future fistula maturation by enlarging veins

B. Peripheral IV placement permanently lowers GFR in the ipsilateral kidney

C. Venous anatomy is not a major consideration because all chronic dialysis uses arterial catheters

D. Future arteriovenous access may depend on intact peripheral and central venous anatomy

E. Vein preservation prevents diabetic nephropathy by increasing renal blood flow

195. A teenager has salt craving, low-normal blood pressure, hypokalemic metabolic alkalosis, and hypercalciuria. Which disorder is most consistent with this profile?

A. Bartter syndrome causing impaired sodium chloride reabsorption in the thick ascending limb

B. Apparent mineralocorticoid excess causing hypertension with suppressed renin and aldosterone

C. Liddle syndrome causing epithelial sodium-channel activation and hypertension

D. Type 4 renal tubular acidosis causing hyperkalemia and metabolic acidosis

E. Gitelman syndrome causing distal convoluted-tubule dysfunction with hypocalciuria

196. A patient has recurrent calcium stones, hematuria, and imaging showing medullary nephrocalcinosis with dilated collecting ducts. Which diagnosis is most likely?

A. Primary membranous nephropathy

B. Acute cortical necrosis

C. Medullary sponge kidney

D. Autosomal dominant polycystic kidney disease

E. Renal artery fibromuscular dysplasia

197. A patient with Sjogren syndrome has recurrent calcium phosphate stones, potassium 3.0 mEq/L, bicarbonate 16 mEq/L, and urine pH 6.2. Which tubular disorder is most likely?

A. Bartter syndrome from thick ascending-limb salt wasting

B. Type 4 renal tubular acidosis from hypoaldosteronism

C. Proximal renal tubular acidosis from isolated bicarbonate wasting

D. Gitelman syndrome from distal convoluted-tubule salt wasting

E. Distal renal tubular acidosis from impaired distal hydrogen secretion

198. A patient with advanced CKD receives morphine and develops prolonged sedation and respiratory depression. Which prescribing principle is most relevant?

A. Hyperkalemia as the principal determinant of opioid accumulation in CKD

B. Active morphine metabolites can accumulate in kidney failure, so an alternative opioid may be safer

C. Kidney failure accelerates morphine clearance and requires larger routine doses

D. Morphine and its active metabolites are predominantly hepatically eliminated and therefore do not accumulate in CKD

E. Morphine is preferred because dialysis removes no opioids or opioid metabolites

199. A patient with CKD has edema resistant to a loop diuretic despite adherence. Which strategy can overcome distal nephron compensation?

A. Increase dietary sodium to enhance delivery to the distal nephron

B. Add a thiazide-type diuretic for sequential nephron blockade with close electrolyte monitoring

C. Add an NSAID to increase renal prostaglandins and sodium excretion

D. Add desmopressin to increase free-water retention and relieve edema

E. Stop the loop diuretic and use potassium chloride as the primary natriuretic

200. A patient with monoclonal gammopathy has kidney dysfunction and biopsy showing nonfibrillar monoclonal light-chain deposits along glomerular and tubular basement membranes. Which diagnosis is most likely?

A. IgA nephropathy

B. Thin basement membrane nephropathy

C. Minimal-change disease

D. Poststreptococcal glomerulonephritis

E. Light-chain deposition disease

201. A patient with a high-anion-gap metabolic acidosis has an anion gap of 20 mEq/L and bicarbonate 8 mEq/L. Using a normal anion gap of 12 and normal bicarbonate of 24, what additional disorder is suggested by the delta comparison?

A. A concurrent normal-anion-gap metabolic acidosis

B. A concurrent metabolic alkalosis

C. A concurrent chronic respiratory acidosis

D. No additional metabolic process is suggested

E. A concurrent acute respiratory alkalosis

202. A young adult has hypokalemia, metabolic alkalosis, normal blood pressure, hypomagnesemia, and low urinary calcium. Which inherited tubulopathy is most likely?

A. Type 4 renal tubular acidosis causing hyperkalemia and normal-anion-gap acidosis

B. Bartter syndrome involving thick ascending-limb salt transport and hypercalciuria

C. Distal renal tubular acidosis causing hypokalemia, acidosis, and calcium phosphate stones

D. Gitelman syndrome involving the thiazide-sensitive sodium-chloride cotransporter

E. Liddle syndrome causing sodium retention with hypertension and suppressed aldosterone

203. A home hemodialysis patient performs more frequent treatments and asks why this may improve volume control. Which explanation is best?

A. More frequent dialysis substantially reduces the need for sodium and fluid awareness in most patients

B. Shorter intervals between treatments reduce interdialytic fluid accumulation and allow gentler ultrafiltration

C. The main benefit comes from intentionally increasing the ultrafiltration rate each session

D. Home dialysis uses no dialysate and therefore may not change sodium balance

E. Frequent dialysis removes all residual kidney function within the first week

204. A 39-year-old woman has resistant hypertension despite three medications, including a thiazide-type diuretic. Potassium is 3.0 mEq/L and bicarbonate is 31 mEq/L. Which screening test is most appropriate for the suspected endocrine cause?

A. Plasma free metanephrines

B. Twenty-four-hour urinary free cortisol as the initial test for all resistant hypertension

C. Plasma aldosterone concentration interpreted with renin to obtain an aldosterone-renin ratio

D. Serum catecholamine concentration measured during a hypertensive episode

E. Renal-vein renin sampling as the first screening study

205. A patient with sarcoidosis develops hypercalcemia and suppressed PTH. Which mechanism is most likely?

A. Renal calcium wasting triggers secondary hyperparathyroidism and increases serum calcium

B. Extrarenal macrophage 1-alpha-hydroxylase activity increases calcitriol production

C. Calcitonin deficiency causes unregulated osteoclast activity in granulomatous tissue

D. Autonomous parathyroid secretion increases calcium while suppressing vitamin D activation

E. FGF23 deficiency raises phosphate excretion and directly produces hypercalcemia

206. A patient with an acute panic attack has PaCO2 25 mm Hg. Which primary acid-base disorder is present?

A. Acute respiratory acidosis from impaired alveolar ventilation

B. Metabolic alkalosis from chloride depletion

C. High-anion-gap metabolic acidosis from organic acid accumulation

D. Acute respiratory alkalosis from hyperventilation

E. Normal-anion-gap metabolic acidosis from bicarbonate loss

207. A patient with hypertension has diminished femoral pulses and higher blood pressure in the arms than the legs. Which diagnosis is most likely?

A. Primary hyperparathyroidism

B. Apparent mineralocorticoid excess

C. Renal artery fibromuscular dysplasia

D. Bilateral renal vein thrombosis

E. Coarctation of the aorta

208. A patient with CKD has standardized office systolic blood pressure consistently above the recommended target despite tolerable therapy. Which measurement issue is most important before intensifying treatment?

A. Rely on a wrist cuff over clothing to avoid white-coat effects

B. Use any single triage blood pressure because technique does not affect treatment targets

C. Average the highest reading primarily because lower measurements are less predictive

D. Ensure blood pressure was obtained with a standardized technique

E. Ignore cuff size because arm circumference does not affect measurement accuracy

209. A patient with recurrent urinary tract infections has a large branching staghorn calculus and alkaline urine. Which process most likely produced the stone?

A. Cystine transporter deficiency causing hexagonal crystals and acidic urine

B. Hyperuricemia causing uric acid precipitation despite alkaline urine

C. Primary hyperparathyroidism causing primarily small nonbranching calcium stones

D. Marked hypocitraturia causing calcium oxalate deposition in acidic urine

E. Urease-positive bacterial infection generating magnesium ammonium phosphate

210. A patient receives gentamicin for a severe gram-negative infection and develops nonoliguric AKI several days later. Which nephrotoxic lesion is classic?

A. Linear anti-GBM antibody deposition

B. Selective podocyte effacement causing minimal-change disease

C. Distal ureteral obstruction from drug crystals in many patients

D. Renal artery thrombosis from endothelial antibody formation

E. Proximal tubular accumulation causing acute tubular injury

211. A patient beginning dialysis for severe azotemia develops headache, nausea, confusion, and seizures during an aggressive first hemodialysis session. Which mechanism is most likely?

A. Heparin exposure causes immune thrombocytopenia and neurologic symptoms within minutes

B. Phosphate removal causes immediate cerebral calcification during the first dialysis session

C. Rapid sodium gain causes acute neuronal dehydration and hypernatremic encephalopathy

D. Rapid urea removal creates an osmotic gradient that promotes cerebral edema

E. Dialyzer calcium loss causes isolated tetany without cerebral water shifts

212. A transplant recipient develops hypertension, a bruit over the graft, and worsening kidney function. Doppler imaging suggests high velocity in the transplant renal artery. Which diagnosis is most likely?

A. Acute pyelonephritis

B. T-cell-mediated rejection

C. BK nephropathy

D. Recurrent diabetic nephropathy

E. Transplant renal artery stenosis

213. A patient on dialysis has persistent hypoalbuminemia. Which interpretation is most appropriate?

A. Hypoalbuminemia can reflect inflammation, illness, protein loss, and inadequate nutrition rather than intake alone

B. Low albumin strongly suggesting nephrotic syndrome in most dialysis patients

C. Albumin has no prognostic value in kidney failure and should not be evaluated

D. Increasing dietary sodium is the most specific treatment for low serum albumin

E. Serum albumin is a pure dietary-protein marker unaffected by inflammation or illness

214. A patient with pelvic malignancy has worsening AKI and bilateral ureteral obstruction above the bladder. Which kidney-directed strategy may be necessary?

A. Use ACE inhibitor therapy to increase pressure across the obstruction

B. Relieve upper-tract obstruction with ureteral stents or percutaneous nephrostomy

C. Start NSAIDs as definitive therapy for bilateral malignant obstruction

D. Give desmopressin to reduce urine flow into the obstructed system

E. Observe until anuria persists for several weeks before decompressing

215. A patient with CKD has persistent metabolic acidosis despite adequate bicarbonate therapy and stable kidney function. Which issue should be reassessed?

A. Metabolic acidosis excludes diarrhea because bowel losses cause alkalosis

B. A low bicarbonate value strongly suggests respiratory alkalosis without need for blood gas assessment

C. Ongoing gastrointestinal bicarbonate loss, medications, or renal tubular disorders may be contributing

D. All medications raise bicarbonate and therefore may not contribute

E. CKD is a principal possible cause of low bicarbonate once eGFR is reduced

216. A patient with HIV receives tenofovir and develops glycosuria with normal serum glucose, hypophosphatemia, and metabolic acidosis. Which complication is most likely?

A. Distal renal tubular acidosis with nephrocalcinosis

B. Renal artery stenosis from antiviral vasoconstriction

C. Minimal-change disease from podocyte injury

D. Tenofovir-associated proximal tubular dysfunction

E. Type 4 renal tubular acidosis from isolated aldosterone resistance

217. A patient has rapidly progressive glomerulonephritis and pulmonary capillaritis without granulomatous upper-airway disease. MPO-ANCA is positive. Which diagnosis is most likely?

A. Granulomatosis with polyangiitis

B. Alport syndrome

C. Microscopic polyangiitis

D. Membranous nephropathy

E. IgA nephropathy

218. A patient has recurrent thrombotic microangiopathy without Shiga toxin and without severe ADAMTS13 deficiency. Complement dysregulation is suspected. Which disease category is most likely?

A. Complement-mediated HUS

B. Minimal-change disease

C. Primary membranous nephropathy

D. Thin basement membrane nephropathy

E. Poststreptococcal glomerulonephritis

219. A patient with CKD has anemia, normal iron stores, no bleeding, and an inappropriately low reticulocyte response. Which mechanism is central?

A. Increased erythropoietin production causing ineffective erythrocytosis

B. Reduced renal erythropoietin production with impaired marrow stimulation

C. High calcitriol concentrations directly inhibiting erythroid progenitors

D. Renal loss of mature red cells through intact glomeruli as the dominant mechanism

E. Excess platelet production suppressing red-cell precursors in the bone marrow

220. A kidney transplant recipient has tremor, hypertension, hyperkalemia, and a rise in creatinine after tacrolimus levels increase. Which mechanism is most likely?

A. Immune-complex deposition caused directly by tacrolimus antibodies

B. BK viral replication caused by low rather than high immunosuppression

C. Obstruction from tacrolimus crystals in the ureter

D. Calcineurin-inhibitor vasoconstriction causing a reversible hemodynamic fall in GFR

E. Complete loss of afferent arteriolar tone causing hyperfiltration

221. A recurrent calcium-stone former has hypercalciuria, normal serum calcium, and low urine citrate. Which medication can reduce recurrence by lowering urinary calcium?

A. A loop diuretic that increases calcium delivery into the urine

B. High-dose vitamin D that increases intestinal calcium absorption and urinary calcium

C. Acetazolamide that raises urine pH and promotes calcium phosphate supersaturation

D. Sodium bicarbonate loading that increases sodium and calcium excretion together

E. A thiazide-type diuretic to reduce urinary calcium excretion

222. A patient with kidney failure is considering stopping maintenance dialysis because of advanced terminal illness and declining quality of life. Which principle is appropriate?

A. The nephrologist should make the decision without assessing the patient's preferences or capacity

B. Dialysis can rarely be discontinued once started because it is legally preferred

C. Palliative-care involvement is generally deferred until all dialysis treatments have stopped

D. An informed patient may choose dialysis withdrawal after goals, prognosis, and symptom management are discussed

E. Withdrawal is allowed primarily if serum potassium is already normal without dialysis

223. A patient receiving peritoneal dialysis develops progressive dyspnea and a right-sided pleural effusion soon after therapy begins. Pleural fluid glucose is unusually high. What is the likely diagnosis?

A. Pulmonary embolism because it raises pleural-fluid glucose above serum

B. Pleuroperitoneal communication with dialysate leak into the chest

C. Bacterial empyema caused by a fistula between the colon and pleural space

D. Uremic pleuritis caused by inadequate solute clearance alone

E. Heart failure because pleural glucose is typically higher than dialysate glucose

224. A 55-year-old woman has normal office blood pressures but repeated home readings of 150/88 mm Hg. Which phenomenon is most likely?

A. Masked hypertension

B. Isolated nocturnal normotension

C. White-coat hypertension

D. Orthostatic hypotension

E. Pseudohypertension from noncompressible arteries

225. A patient with severe cerebral edema and AKI requires kidney replacement therapy. Which principle is important?

A. Choose modality solely on serum creatinine and ignore intracranial physiology

B. Use the highest-efficiency intermittent dialysis possible to maximize rapid urea removal

C. Withhold all dialysis even for life-threatening hyperkalemia

D. Avoid rapid osmotic shifts that could worsen intracranial pressure

E. Induce hypernatremia and rapid urea clearance without neurologic monitoring

226. A 22-year-old patient has recurrent stones. Urine microscopy shows characteristic hexagonal crystals. Which diagnosis is most likely?

A. Distal renal tubular acidosis causing calcium phosphate stone formation

B. Hyperuricosuria causing rhomboid or rosette-shaped uric acid crystals

C. Urease-positive infection causing magnesium ammonium phosphate crystals

D. Primary hyperoxaluria causing calcium oxalate envelope-shaped crystals

E. Cystinuria causing recurrent cystine stones with hexagonal crystals

227. A transplant recipient develops multiple aggressive cutaneous squamous-cell carcinomas. Which long-term complication of immunosuppression is illustrated?

A. Skin cancer risk falls below that of the general population after transplantation

B. Markedly increased skin-cancer risk after transplantation

C. Predominantly hematologic cancers are increased by transplant immunosuppression

D. Immunosuppression protects against ultraviolet-induced malignancy

E. Cutaneous squamous-cell carcinoma excludes a role for chronic immunosuppression

228. A patient with severe ANCA-associated vasculitis is being treated for remission induction. Which general principle reflects current therapy?

A. Avoid immunosuppression because pauci-immune disease resolves spontaneously

B. Use potent immunosuppression such as rituximab or cyclophosphamide with a glucocorticoid strategy tailored to severity

C. Use antibiotics alone because ANCA vasculitis is caused by occult bacterial infection

D. Use chronic plasma exchange as maintenance therapy in many patients

E. Treat primarily with RAAS blockade because proteinuria is the main driver of injury

229. A transplant recipient is several years posttransplant with stable graft function. Which preventive-care principle remains important?

A. Screen primarily for graft rejection because nonrenal complications are uncommon

B. Avoid all vaccines permanently because none are safe after transplantation

C. Continue age-appropriate cancer screening, vaccination planning, cardiovascular risk reduction, and infection surveillance

D. Stop general preventive care because transplant follow-up replaces primary care

E. Discontinue cardiovascular risk management once kidney function normalizes

230. A patient on peritoneal dialysis has clear effluent but poor drain volume and constipation. Which issue is most likely?

A. Bacterial peritonitis because clear effluent excludes mechanical causes

B. Arteriovenous fistula stenosis causing reduced peritoneal drainage

C. Systemic hyperkalemia causing automatic closure of the dialysis catheter

D. Hemodialysis water contamination affecting intraperitoneal flow

E. Mechanical catheter outflow impairment from bowel distention or catheter malposition

231. A patient with septic shock requires fluid resuscitation. Which general principle is most appropriate for kidney protection?

A. Target oliguria alone with repeated fluid boluses even with perfusion or congestion

B. Avoid all fluids because sepsis-associated AKI is rarely hemodynamic

C. Use goal-directed balanced crystalloid resuscitation to restore perfusion while avoiding persistent fluid overload

D. Use hypertonic saline as the default resuscitation fluid for many patients with septic shock

E. Give unlimited crystalloid because positive fluid balance has no renal consequences

232. A patient with a monoclonal gammopathy has nephrotic proteinuria and biopsy showing randomly arranged fibrils that are Congo red positive. Which process is most likely?

A. C3 glomerulopathy from complement dysregulation

B. AL amyloidosis from monoclonal immunoglobulin light chains

C. Minimal-change disease from podocyte effacement

D. Alport syndrome from type IV collagen mutation

E. Postinfectious glomerulonephritis from subepithelial humps

233. A 58-year-old man with uncontrolled diabetes has polyuria. Serum glucose is 640 mg/dL and urine osmolality is 690 mOsm/kg. Which type of polyuria is present?

A. Pure water diuresis from severe nephrogenic diabetes insipidus

B. Pure water diuresis from complete central diabetes insipidus

C. Solute diuresis driven by urinary glucose excretion

D. Primary polydipsia with maximally suppressed urinary solute concentration

E. Postobstructive water diuresis occurring despite absence of recent urinary obstruction

234. A patient with rhabdomyolysis after prolonged immobilization has creatine kinase 40,000 U/L, hyperkalemia, and AKI. Urine dipstick is strongly positive for blood but microscopy shows few red cells. What does this indicate?

A. Hemoglobinuria from autoimmune hemolysis as a primary explanation

B. Glomerular hematuria with red-cell casts

C. Renal papillary necrosis causing isolated intact erythrocytes

D. False-negative microscopy caused by uric acid crystals

E. Myoglobinuria causing pigment-associated tubular injury

235. A patient receives intravenous acyclovir without adequate hydration and develops AKI with needle-shaped crystals in the urine. Which lesion is most likely?

A. Acyclovir crystal nephropathy

B. Minimal-change disease

C. Type 4 renal tubular acidosis

D. Anti-GBM glomerulonephritis

E. Renal artery thrombosis

236. A patient presents with hemoptysis, rapidly progressive glomerulonephritis, and linear IgG staining along glomerular basement membranes. Which diagnosis is most likely?

A. C3 glomerulopathy

B. Anti-GBM disease

C. Minimal-change disease

D. Membranous nephropathy

E. IgA nephropathy

237. A patient develops severe hypertension after blunt abdominal trauma. CT shows a subcapsular renal hematoma compressing the kidney. Which mechanism is most likely?

A. SIADH caused by renal capsular pressure

B. Liddle syndrome triggered by renal trauma

C. Central sympathetic failure causing low-renin hypertension

D. Primary aldosteronism from traumatic adrenal hemorrhage

E. Page kidney with renin activation from external renal compression

238. A patient with CKD has a persistently rising PTH but normal calcium and phosphate. Which interpretation is best?

A. Management should consider trends and modifiable factors rather than reacting to one target PTH value

B. A rising PTH commonly means adynamic bone disease from oversuppression

C. PTH is clinically not a major consideration in CKD unless serum calcium is above 12 mg/dL

D. A normal phosphate level makes secondary hyperparathyroidism unlikely in advanced CKD

E. Any PTH above the laboratory normal range mandates parathyroidectomy

239. A 70-year-old man with diabetic kidney disease takes lisinopril and has potassium 5.8 mEq/L, bicarbonate 18 mEq/L, and a normal anion gap. Which mechanism is most likely?

A. Vomiting-induced metabolic alkalosis with secondary renal potassium conservation

B. Distal RTA with severe potassium wasting from enhanced collecting-duct secretion

C. Proximal RTA with generalized urinary glucose and phosphate wasting

D. Lactic acidosis from tissue hypoperfusion producing a large anion gap

E. Reduced aldosterone effect causing hyperkalemic type 4 renal tubular acidosis

240. A patient with cirrhosis and suspected hepatorenal syndrome-AKI is treated after volume assessment. Which therapy targets the underlying circulatory abnormality?

A. Routine hypotonic saline to expand intracellular volume

B. ACE inhibitor therapy to reduce efferent arteriolar tone

C. Immediate nephrectomy because renal vasoconstriction is irreversible

D. High-dose NSAIDs to constrict the afferent arteriole

E. Vasoconstrictor therapy with albumin support in an appropriate monitored setting

Answers

1. E. Because urea handling occurs largely in proximal segments, fractional excretion of urea may be less affected by loop diuretics than FENa. It is still imperfect and should not replace clinical assessment.

2. D. NSAIDs inhibit prostaglandin-mediated afferent dilation, while ACE inhibitors reduce angiotensin II-mediated efferent constriction. Combined with diuretic-related volume depletion, this can sharply lower intraglomerular pressure and precipitate AKI.

3. C. Postobstructive diuresis may be physiologic but can become pathologic, causing large water and electrolyte losses. Fluid replacement is individualized and serum chemistries are monitored closely.

4. C. After hemodynamic stabilization, hypernatremia from water deficit is corrected with electrolyte-free water, delivered enterally or as intravenous dextrose solution. Isotonic saline is reserved for restoring perfusion when needed, not for replacing the entire free-water deficit.

5. D. CKD changes the pharmacokinetics and adverse-effect profile of several gout therapies. Management should be individualized, with attention to renal dosing, drug interactions, and safer approaches to flare prophylaxis.

6. C. Albuminuria is both a marker of kidney damage and a prognostic variable. Reductions with effective therapy often accompany lower long-term kidney risk, while rising albuminuria can signal progression or a change in disease activity.

7. C. Beta blockade may be added for persistent tachycardia only after adequate alpha-adrenergic blockade has been established. Starting beta blockade before alpha blockade can permit unopposed alpha-mediated vasoconstriction and provoke severe hypertension.

8. D. Muddy brown granular casts and tubular epithelial cells strongly support acute tubular injury. In sepsis, tubular injury often coexists with complex hemodynamic and inflammatory disturbances.

9. D. Significant new proteinuria in an allograft has a broad differential and can signal recurrent or de novo glomerular disease, chronic antibody-mediated injury, or other pathology. Histology often guides diagnosis and treatment.

10. E. Chronic high-dose analgesic exposure can cause chronic tubulointerstitial nephritis and papillary necrosis. Imaging may show papillary calcification or irregular renal contours in advanced disease.

11. B. Hypernatremia can rarely result from sodium gain rather than water loss. Hypertonic sodium administration produces both hypernatremia and extracellular volume expansion, distinguishing it from most water-loss states.

12. A. Insulin stimulates cellular potassium uptake and lowers serum potassium rapidly, but total-body potassium is unchanged. Definitive therapy must also stop ongoing potassium input and enhance removal when necessary.

13. E. Severe symptomatic hypotonic hyponatremia requires prompt but controlled hypertonic saline while the precipitating drug is stopped. Thiazides impair urinary dilution and are a common cause of profound hyponatremia in older adults; correction must be monitored to avoid overcorrection.

14. E. Loop diuretics increase distal sodium delivery and produce volume contraction with secondary aldosterone activation. Both effects enhance distal potassium secretion and can cause substantial hypokalemia.

15. B. Large chloride loads can reduce the strong-ion difference and produce a normal-anion-gap hyperchloremic metabolic acidosis. This is common after substantial 0.9% saline administration.

16. A. Early volume resuscitation helps correct hypovolemia and supports renal perfusion and tubular flow in rhabdomyolysis. Electrolytes must be monitored closely, and dialysis is reserved for standard indications.

17. C. Immediate anuria or loss of graft perfusion after surgery can result from vascular thrombosis, kinking, or technical complications. These require urgent imaging and often surgical intervention.

18. C. Apparent resistant hypertension may reflect poor adherence, inaccurate measurement, white-coat effect, suboptimal regimen, excess sodium, or interfering drugs. These should be addressed before concluding that blood pressure is pharmacologically resistant.

19. A. Contrast-associated creatinine changes typically occur relatively soon and do not cause blue toes, livedo, or eosinophilia. Delayed kidney injury with embolic skin findings after angiography is more consistent with cholesterol embolization.

20. E. Fanconi syndrome reflects generalized proximal tubular reabsorptive failure. Bicarbonate, glucose, phosphate, amino acids, and uric acid can be lost in urine, producing proximal RTA plus other proximal-tubule abnormalities.

21. C. Early hypoperfusion can produce avid sodium retention, but sustained ischemia damages tubular cells and impairs sodium reabsorption. Urine indices then become less prerenal and the sediment may show granular casts.

22. E. Minimal-change disease typically has normal or nearly normal light microscopy, negative immunofluorescence, and diffuse podocyte foot-process effacement on electron microscopy.

23. B. Patients with kidney failure can have both high thrombotic and bleeding risks, and drug pharmacokinetics differ substantially. Anticoagulation decisions require individualized assessment rather than automatic treatment or avoidance.

24. A. Adrenal nodules are common and may be nonfunctioning. In many surgical candidates, adrenal vein sampling is used to distinguish unilateral aldosterone production from bilateral disease before adrenalectomy.

25. B. Poststreptococcal glomerulonephritis typically follows streptococcal pharyngitis or skin infection after a latent period and causes nephritic syndrome with complement consumption. C3 usually returns toward normal over subsequent weeks.

26. C. NSAIDs, sympathomimetic decongestants, excess alcohol, stimulants, and several other substances can increase blood pressure or blunt antihypertensive efficacy. Medication and substance review is part of resistant-hypertension evaluation.

27. D. In the appropriate clinicopathologic setting, C4d deposition in peritubular capillaries is evidence of antibody interaction with the allograft microcirculation and supports antibody-mediated rejection when integrated with allograft injury and donor-specific antibody findings.

28. E. Loop diuretics are useful for volume control but have not been shown to repair acute tubular injury or improve survival solely by increasing urine output. Kidney recovery must be assessed independently.

29. A. After initial resuscitation, persistent fluid accumulation can worsen pulmonary gas exchange, venous congestion, tissue edema, and kidney function. De-resuscitation with diuretics or kidney replacement therapy may be appropriate when perfusion is adequate.

30. E. Heart failure can produce kidney dysfunction through reduced forward perfusion, neurohormonal activation, and renal venous congestion. A patient may be globally volume overloaded yet have low effective arterial filling.

31. D. Peritoneal dialysis uses the patient's peritoneal capillary bed and peritoneal membrane as the dialysis interface. Diffusion removes solutes and osmotic gradients, commonly from glucose, drive ultrafiltration.

32. C. Granulomatosis with polyangiitis commonly involves the upper and lower respiratory tracts and kidneys and is often associated with PR3-ANCA. Kidney biopsy typically shows pauci-immune necrotizing crescentic glomerulonephritis.

33. B. Hemolysis can release intracellular potassium and create pseudohyperkalemia. A repeat properly collected specimen is appropriate when the clinical picture is discordant, although unstable patients or those with ECG toxicity still require immediate treatment.

34. B. CKD risk assessment integrates GFR category and albuminuria category. Lower eGFR and higher albuminuria each independently identify greater risk, and their combination is especially prognostic.

35. A. Shiga toxin-producing enteric infection can cause a typical HUS syndrome after a diarrheal prodrome. Supportive care is central, with dialysis as needed for severe kidney failure.

36. C. Vomiting causes chloride depletion and contraction alkalosis. A low urine chloride identifies a chloride-responsive process in which isotonic saline and potassium repletion restore distal chloride delivery and allow bicarbonate excretion.

37. E. AKI evolves dynamically, and dialysis modalities can add variable extracorporeal drug clearance. Doses may need repeated adjustment to avoid both toxicity and underexposure.

38. B. When hypokalemia is present, the kidney should reduce potassium excretion. Inappropriately high urinary potassium suggests renal loss from diuretics, mineralocorticoid activity, tubular disorders, or other renal mechanisms.

39. D. Familial hypocalciuric hypercalcemia is characterized by lifelong mild hypercalcemia, nonsuppressed PTH, and low urinary calcium excretion. This distinction is important because parathyroid surgery does not correct the underlying calcium-sensing receptor defect.

40. A. ACE inhibitors and ARBs reduce efferent arteriolar constriction, so GFR may fall modestly after initiation. A limited stable rise in creatinine can be acceptable if potassium and volume status are appropriate and no major renovascular problem is present.

41. C. Mycophenolate is associated with pregnancy loss and a characteristic pattern of congenital malformations and must be avoided in pregnancy. Preconception planning allows transition to a safer immunosuppressive regimen while protecting graft function.

42. A. High-dose methotrexate and its metabolites can precipitate in renal tubules, especially in acidic concentrated urine. Hydration, urine alkalinization, leucovorin rescue, and drug-level monitoring are key components of prevention and treatment.

43. D. With severe bilateral renal artery stenosis, glomerular filtration may depend on angiotensin II-mediated efferent constriction. RAAS blockade can therefore produce a large hemodynamic fall in GFR.

44. E. Cirrhosis lowers effective arterial blood volume despite total-body sodium and water excess. Baroreceptor-mediated vasopressin release promotes disproportionate water retention, producing hypervolemic hyponatremia.

45. A. Refeeding raises insulin, which drives phosphate, potassium, and magnesium into cells while increasing ATP synthesis. Profound hypophosphatemia can cause weakness, respiratory failure, hemolysis, and rhabdomyolysis.

46. C. Medication stewardship is important around acute illness and procedures. Drugs that alter renal hemodynamics, volume status, glucose handling, or toxicity risk may need individualized temporary adjustment rather than a blanket rule.

47. E. Interstitial inflammation and tubulitis are characteristic features of T-cell-mediated rejection. Biopsy is important because the differential diagnosis of early allograft dysfunction is broad.

48. C. The combination of small-vessel purpura, gastrointestinal and joint symptoms, and IgA-dominant kidney disease is characteristic of IgA vasculitis. Kidney involvement can resemble IgA nephropathy histologically.

49. A. Phosphate binders work by binding dietary phosphate in the gut, so they are taken with meals. Management also considers dietary sources and dialysis adequacy rather than relying on a single intervention.

50. D. CRRT can remove substantial phosphate, especially during prolonged high-dose therapy. Hypophosphatemia can impair respiratory and muscle function and should be prevented or treated with supplementation or phosphate-containing fluids when available.

51. E. Extreme hypertension can damage arteriolar endothelium and produce a TMA pattern with fibrinoid necrosis, thrombosis, hemolysis, and kidney injury. Blood-pressure control is central while other TMA causes are considered.

52. A. Aldosterone increases sodium reabsorption in the collecting duct while enhancing potassium and hydrogen secretion. Mineralocorticoid excess therefore commonly produces hypokalemia and metabolic alkalosis.

53. E. CKD-MBD management is based on serial assessment of phosphate, calcium, PTH, and vitamin D status rather than a single isolated value. Correctable factors such as vitamin D deficiency and phosphate excess should be addressed.

54. D. Mixed cryoglobulinemia, often associated with hepatitis C, can cause systemic small-vessel vasculitis and an immune-complex glomerulonephritis with low complement, especially low C4.

55. E. Modern practice emphasizes symptoms, refractory metabolic complications, nutrition, volume status, and patient goals rather than an eGFR threshold alone. Preparation should occur early even when dialysis is not yet indicated.

56. A. Life-threatening electrolyte disturbance, acidemia, and volume overload that cannot be controlled medically are classic indications for urgent dialysis in AKI. The decision is driven by clinical complications, not creatinine alone.

57. A. Hypertension in dialysis is often driven by extracellular volume and sodium excess, although RAAS activation, sympathetic activity, medications, and other factors also contribute. Accurate dry-weight assessment is fundamental.

58. E. Vomiting causes extracellular volume depletion. Intact kidneys avidly retain sodium, producing a low urine sodium, while effective-volume depletion stimulates vasopressin and concentrates the urine.

59. E. Renal vein thrombosis can impair venous drainage, increase interstitial pressure, and reduce renal perfusion and filtration. It is associated with nephrotic syndrome and hypercoagulable states.

60. B. Tolvaptan can slow kidney-volume growth and eGFR decline in selected adults with ADPKD at risk for rapid progression. Its aquaretic effects and potential hepatotoxicity require careful counseling and monitoring.

61. B. Postobstructive diuresis can cause clinically important water and electrolyte losses after relief of obstruction. Management requires close monitoring and partial replacement tailored to hemodynamics and laboratory trends, not automatic one-for-one replacement.

62. A. Pneumocystis jirovecii can cause severe hypoxemic pneumonia in immunosuppressed transplant recipients. Prophylaxis with trimethoprim-sulfamethoxazole is commonly used during the highest-risk period.

63. A. BK polyomavirus can reactivate under immunosuppression and cause tubulointerstitial nephritis with progressive graft dysfunction. Viral load testing and biopsy help distinguish it from rejection.

64. D. Severe hyperkalemia, metabolic acidosis, and volume overload after missed dialysis are classic indications for urgent kidney replacement therapy. Temporizing measures may be used while dialysis is arranged.

65. E. Bladder outlet obstruction is a reversible cause of postrenal AKI. Prompt decompression relieves pressure and is followed by monitoring for hematuria, electrolyte shifts, and postobstructive diuresis.

66. A. When RAAS blockade is beneficial, clinicians often first correct reversible contributors to hyperkalemia and use strategies that increase potassium excretion or binding. Persistent severe hyperkalemia may still require dose reduction or discontinuation.

67. D. Heparin-induced thrombocytopenia is an immune-mediated prothrombotic disorder characterized by a platelet fall and new thrombosis after heparin exposure. Dialysis circuits can clot, and heparin must be stopped while alternative anticoagulation is considered.

68. B. Rapid malignant-cell breakdown releases potassium, phosphate, and nucleic acids, producing hyperkalemia, hyperphosphatemia, secondary hypocalcemia, hyperuricemia, and acute kidney injury.

69. A. Chronic respiratory acidosis allows time for renal adaptation, including increased net acid excretion and generation of bicarbonate. Serum bicarbonate therefore rises more than it does during acute respiratory acidosis.

70. B. New hypertension after midpregnancy with proteinuria and maternal end-organ abnormalities is consistent with preeclampsia. Thrombocytopenia and hepatic involvement are severe features requiring urgent obstetric assessment.

71. E. Living-donor evaluation is designed primarily to protect the donor. Persistent albuminuria may indicate underlying kidney disease and requires careful assessment before nephrectomy is considered.

72. C. Severe secondary or tertiary hyperparathyroidism that remains refractory to medical therapy can require surgical parathyroidectomy, especially when accompanied by complications such as bone disease or uncontrolled mineral abnormalities.

73. A. Liddle syndrome causes constitutive ENaC activity, sodium retention, hypertension, hypokalemia, and metabolic alkalosis. Both renin and aldosterone are suppressed because the sodium-retaining defect is downstream of aldosterone.

74. E. Dietary potassium management should be individualized. Bioavailable potassium additives, portion size, constipation, acidosis, medications, and overall dietary quality all matter, and excessive restriction can undermine nutrition.

75. C. Uremia can impair platelet adhesion and aggregation despite a normal platelet count. Dialysis, correction of severe anemia, and selected hemostatic therapies may improve bleeding in urgent settings.

76. C. Denervation of the transplanted kidney and ureter can blunt classic flank pain. Obstruction may therefore present primarily as graft dysfunction, reduced urine output, hematuria, or infection.

77. D. Recovery is reflected by improving endogenous kidney clearance, stable electrolytes and acid-base status, manageable volume, and adequate urine output without dependence on kidney replacement therapy. Diuretic-stimulated urine alone is not proof of recovery.

78. A. When hyperkalemia remains uncontrolled despite appropriate medical therapy, kidney replacement therapy may be required. The timing of dialysis is based on refractory complications and symptoms, not a creatinine threshold alone.

79. A. Conventional amphotericin B can cause renal vasoconstriction and direct distal tubular injury, leading to AKI, potassium wasting, magnesium wasting, and impaired urinary acidification. Lipid formulations generally reduce nephrotoxicity.

80. E. Dialysis initiation is driven by symptoms and complications that cannot be controlled medically rather than by an eGFR threshold alone. Uremic manifestations, refractory volume overload, hyperkalemia, or acidosis are common indications.

81. A. Dysmorphic erythrocytes and red-cell casts indicate glomerular bleeding and strongly suggest an inflammatory glomerular process. Serologic evaluation and often urgent kidney biopsy are warranted.

82. C. Resistant hypertension and spontaneous or diuretic-induced hypokalemia are classic indications to screen for primary aldosteronism. The aldosterone-to-renin ratio is widely used, with attention to medications, potassium status, and confirmatory testing when appropriate.

83. A. Marked intra-abdominal hypertension can reduce renal blood flow, increase renal venous pressure, and decrease cardiac output, causing oliguria and multiorgan dysfunction. Decompression may be required in true abdominal compartment syndrome.

84. D. Diarrheal fluid contains bicarbonate. Its loss lowers serum bicarbonate, while chloride rises to maintain electroneutrality, producing a normal-anion-gap or hyperchloremic metabolic acidosis.

85. E. Finerenone can reduce kidney and cardiovascular risk in selected patients with type 2 diabetes and persistent albuminuric CKD despite optimized standard therapy. Potassium must be monitored because hyperkalemia is an important adverse effect.

86. B. Creatinine concentration depends on both generation and excretion. Critical-illness muscle wasting can substantially reduce creatinine production, so a deceptively low serum value may overestimate recovered kidney function.

87. E. De novo donor-specific antibodies can signal increased risk of antibody-mediated injury before major creatinine changes occur. Clinical context, adherence, proteinuria, and sometimes biopsy help define significance.

88. C. Marked hyperglycemia raises extracellular tonicity and pulls water out of cells, lowering the measured sodium concentration. This is hypertonic translocational hyponatremia rather than a primary excess of electrolyte-free water.

89. B. In hypernatremia, a strongly concentrated urine demonstrates an intact renal response to vasopressin. Limited water access or extrarenal water loss is therefore more likely than diabetes insipidus.

90. A. Urine indices are imperfect. Sepsis, early tubular injury, glomerular disease, contrast exposure, and other settings can produce low FENa despite intrinsic injury, while diuretics can raise FENa despite prerenal physiology.

91. A. Gabapentin is substantially renally eliminated and can accumulate in advanced CKD, causing sedation, confusion, and myoclonus. Dose and interval should be adjusted to kidney function and dialysis schedule.

92. C. When citrate is adequately metabolized, it generates bicarbonate. A high citrate load relative to clearance and metabolic needs can therefore contribute to metabolic alkalosis without evidence of citrate accumulation.

93. A. Rasburicase converts uric acid to a more soluble metabolite and can rapidly reduce existing hyperuricemia. Allopurinol prevents formation of new uric acid but does not remove uric acid already present.

94. E. Creatinine is influenced by muscle mass and other non-GFR determinants. Cystatin C, especially when combined with creatinine, can improve estimation when creatinine-based eGFR may be inaccurate and the result will affect management.

95. E. Delayed graft function commonly reflects ischemia-reperfusion injury and acute tubular injury, particularly with deceased-donor organs. Rejection and other causes must still be considered when recovery is atypical.

96. A. With ECG manifestations of severe hyperkalemia, intravenous calcium is given immediately to stabilize the myocardium. Insulin with glucose, beta-agonists, bicarbonate in selected acidemic patients, and potassium removal address the serum potassium itself.

97. D. Anti-GBM disease is treated urgently with plasma exchange to remove circulating antibody and immunosuppression to suppress new antibody production, alongside supportive care. Prognosis depends heavily on kidney function and biopsy severity at presentation.

98. B. Proliferative lupus nephritis requires prompt immunosuppressive therapy in addition to supportive care. Current regimens use glucocorticoids with agents such as mycophenolate or cyclophosphamide, and selected patients may receive add-on therapies including belimumab or a calcineurin-pathway agent.

99. A. Cisplatin can injure renal tubules and produce prolonged renal magnesium wasting. The urine may remain inappropriately rich in magnesium despite a low serum concentration.

100. D. An X-linked pedigree with glomerular hematuria, progressive kidney disease, and sensorineural hearing loss is characteristic of X-linked Alport syndrome, which is caused by pathogenic variants in COL4A5 encoding the alpha-5 chain of type IV collagen.

101. D. Reduction of immunosuppression, when clinically feasible, is a foundational initial step in many cases of post-transplant lymphoproliferative disorder because it can restore antiviral and antitumor immune surveillance. Additional therapy, often including rituximab for CD20-positive disease, is guided by histology, burden, and response.

102. A. Glycyrrhetinic compounds in licorice inhibit 11-beta-hydroxysteroid dehydrogenase type 2, allowing cortisol to stimulate mineralocorticoid receptors. The phenotype resembles mineralocorticoid excess but both renin and aldosterone are suppressed.

103. A. Reduced effective arterial blood volume in advanced heart failure activates vasopressin despite hypotonicity. The kidney therefore retains water and typically produces urine that is not maximally dilute.

104. B. Chronic inflammatory disorders can drive sustained serum amyloid A production and secondary AA amyloid deposition. Renal involvement commonly presents with heavy proteinuria and progressive kidney dysfunction.

105. B. Water excretion requires enough urinary solute to carry water out at the kidney's minimum urine osmolality. With very low protein and salt intake, even appropriately dilute urine may be insufficient to excrete a large water load.

106. D. Kidney care integrates filtration, markers of kidney damage, disease cause, rate of change, and complications. Creatinine is important but does not capture the full clinical picture.

107. E. In myeloma cast nephropathy, filtered monoclonal light chains combine with Tamm-Horsfall protein and form obstructing toxic casts in distal nephron segments. Rapid control of the plasma-cell disorder is central to kidney recovery.

108. B. Thin basement membrane nephropathy commonly presents with persistent glomerular hematuria and preserved kidney function. Family history can be present, although collagen-related disease spans a spectrum and prognosis depends on associated proteinuria and other features.

109. B. For seizures or other severe manifestations, the immediate objective is a small controlled increase in sodium that reduces brain swelling, not immediate normalization. Subsequent correction should remain limited, particularly in chronic high-risk patients.

110. E. Several glomerular diseases, including membranous nephropathy, can recur after kidney transplantation. Monitoring proteinuria and disease-specific biomarkers can help detect recurrence.

111. B. A sudden loss of thrill or bruit suggests thrombosis or severe access dysfunction and requires prompt evaluation. Early intervention can sometimes salvage the access.

112. A. A water diuresis with hypernatremia and a large increase in urine osmolality after desmopressin indicates deficient endogenous vasopressin production. The postoperative setting is typical for central diabetes insipidus.

113. D. Scleroderma renal crisis is treated urgently with ACE inhibition even when creatinine is rising. Delayed RAAS blockade can worsen outcomes; blood pressure is lowered carefully while kidney function and potassium are monitored.

114. E. This pattern is classic for SIADH: hypotonic hyponatremia, inappropriately concentrated urine, urine sodium that is not low, euvolemia, and exclusion of thyroid and adrenal failure. Mild chronic SIADH is commonly managed first with fluid restriction plus treatment of the cause.

115. B. Microangiopathic hemolytic anemia plus thrombocytopenia and organ injury indicates thrombotic microangiopathy. The differential includes TTP, Shiga toxin-associated HUS, complement-mediated HUS, severe hypertension, pregnancy-related syndromes, drugs, and other triggers.

116. B. During aggressive but effective decongestion, GFR can fall modestly from hemodynamic shifts while congestion and outcomes improve. Clinical context, urine findings, perfusion, and trajectory help distinguish this from true intrinsic kidney injury.

117. D. Drug-induced AIN can present with systemic hypersensitivity findings and a tubulointerstitial urine sediment. The full triad is uncommon, but this classic presentation strongly supports the diagnosis.

118. C. C3-dominant glomerular disease with persistent hypocomplementemia points to dysregulation of the alternative complement pathway. Acquired autoantibodies or genetic abnormalities affecting complement regulation can sustain activation.

119. B. Salicylate is dialyzable, and severe neurologic toxicity, pulmonary edema, kidney failure, refractory acidosis, or a rising high concentration despite treatment are indications for urgent extracorporeal removal.

120. E. For metabolic acidosis, expected PaCO2 is approximately 1.5 × bicarbonate + 8 ± 2. With bicarbonate 12 mEq/L, expected PaCO2 is about 26 ± 2 mm Hg. A measured PaCO2 of 40 mm Hg is substantially higher, indicating superimposed respiratory acidosis.

121. E. CKD care is cardiovascular as well as kidney focused. Tobacco use compounds vascular risk and is associated with worse kidney outcomes, making cessation an important component of risk reduction.

122. A. Liddle syndrome results from constitutively active ENaC channels in the collecting duct. Direct ENaC blockade with amiloride or triamterene is effective, whereas mineralocorticoid-receptor antagonists are less useful because aldosterone is already suppressed.

123. E. Renal cortical necrosis is a rare, severe ischemic injury associated with obstetric catastrophes, shock, and DIC. Persistent anuria and imaging evidence of cortical nonperfusion support the diagnosis.

124. C. Chronic lead exposure can cause tubulointerstitial nephropathy, hypertension, hyperuricemia, gout, and anemia. Occupational history is therefore important in otherwise unexplained CKD.

125. A. Long-standing secondary hyperparathyroidism can become autonomous and persist after transplantation, producing hypercalcemia and low phosphate. Medical therapy or parathyroidectomy may be required depending on severity and persistence.

126. B. Inflammation and reduced kidney clearance can increase hepcidin in CKD, restricting intestinal iron absorption and iron release from stores. Ferritin may be normal or high even when iron available to the marrow is inadequate.

127. C. In vasodilatory shock, norepinephrine is a standard first-line vasopressor and can improve organ perfusion by restoring arterial pressure. Persistent oliguria does not mean vasopressors are intrinsically nephrotoxic when they are needed for hemodynamic support.

128. B. The kidney is the major route of magnesium excretion. In advanced kidney failure, magnesium-containing antacids or laxatives can cause clinically significant hypermagnesemia with neuromuscular and cardiovascular depression.

129. B. Fentanyl is primarily hepatically metabolized and lacks active renally cleared metabolites that commonly accumulate in kidney failure. Dosing still requires careful titration because any opioid can cause respiratory and central nervous system toxicity.

130. D. Central venous dialysis catheters carry a high infection risk. Fever with purulent drainage should prompt evaluation for catheter-related bloodstream infection and timely antimicrobial and catheter management.

131. E. Discordance between office and out-of-office readings raises the possibility of white-coat hypertension. Ambulatory or validated home monitoring helps determine the patient's usual blood pressure and prevents overtreatment.

132. E. Kidney biopsy is central to classifying clinically important lupus nephritis because histologic class and activity/chronicity influence treatment and prognosis. Serologies and urine findings support the diagnosis but do not replace tissue classification in appropriate patients.

133. B. Hypertensive emergency is defined by severe blood pressure elevation with acute target-organ injury. Blood pressure is lowered promptly but in a controlled fashion to avoid ischemia from excessive reduction.

134. C. Autoantibodies to the M-type phospholipase A2 receptor (PLA2R) account for most cases of primary membranous nephropathy in adults. The characteristic lesion is subepithelial immune-complex deposition with granular capillary-wall staining.

135. E. The urgency of severe hypertension depends on acute target-organ injury rather than the pressure value alone. In asymptomatic patients without such injury, blood pressure is usually reduced more gradually with oral therapy and close follow-up.

136. B. Minimal-change disease can occur as a paraneoplastic glomerulopathy, particularly with Hodgkin lymphoma. Treating the underlying lymphoma may improve the nephrotic syndrome.

137. A. CMV commonly causes fever, constitutional symptoms, cytopenias, and gastrointestinal or other tissue-invasive disease in immunosuppressed transplant recipients. Risk depends on donor-recipient serostatus and prophylaxis strategy.

138. D. SGLT2 inhibitors reduce CKD progression and cardiovascular events across broad groups of patients with CKD, including many with and without diabetes. Their kidney benefit is not explained solely by glucose lowering.

139. B. Gabapentin is eliminated largely unchanged by the kidneys. Without dose reduction and attention to dialysis timing, accumulation can cause confusion, sedation, ataxia, and myoclonus.

140. E. Acute dialyzer reactions can occur soon after extracorporeal circulation begins and may cause dyspnea, chest discomfort, hypotension, or anaphylactoid features. The treatment should be stopped and supportive measures given while the cause is investigated.

141. A. DKA causes major urinary potassium losses, so total-body potassium is depleted even when serum potassium is initially high. Insulin deficiency, hyperosmolality, and acidemia shift potassium out of cells; treatment reverses these shifts and can rapidly lower serum potassium.

142. C. Transplant glomerulopathy and chronic microvascular injury in the setting of donor-specific antibodies are characteristic of chronic antibody-mediated alloimmune injury. Management focuses on immunologic assessment and modifiable graft-risk factors.

143. A. The kidney contributes to insulin clearance and gluconeogenesis. Advanced CKD can reduce insulin requirements and increase hypoglycemia risk, so glycemic therapy often needs reassessment as kidney function changes.

144. C. Peritoneal membrane characteristics can change over time, and ultrafiltration may become inadequate despite prescription adjustments. Persistent volume overload in this setting can require changes in PD regimen or dialysis modality.

145. D. Fomepizole blocks alcohol dehydrogenase and prevents formation of toxic glycolate and oxalate metabolites. Severe acidosis, kidney failure, high toxin burden, or clinical deterioration are common reasons to add hemodialysis.

146. A. Severe arterial calcification can make a cuff overestimate intra-arterial pressure because the vessel is difficult to compress. Pseudohypertension is uncommon but should be considered when measured values are implausibly high for the clinical picture.

147. B. Hemodynamic intolerance often reflects a fluid-removal rate that exceeds vascular refill. Longer treatments, lower ultrafiltration rates, or continuous/prolonged modalities can improve tolerance.

148. C. Severe ADAMTS13 deficiency strongly supports TTP, a medical emergency requiring urgent plasma exchange and disease-specific immunosuppressive therapy. Kidney injury can occur but is often less dominant than in some HUS presentations.

149. E. Kidney paired donation matches incompatible donor-recipient pairs with other pairs so that each recipient can receive a compatible kidney. It can substantially expand living-donor transplantation.

150. B. Continuous kidney replacement therapy provides slower solute and fluid removal and is often selected for patients with marked hemodynamic instability. Modality choice also depends on resources, neurologic status, and treatment goals.

151. E. Lithium can enter collecting-duct principal cells through epithelial sodium channels and impair the vasopressin-aquaporin pathway, producing nephrogenic diabetes insipidus. Amiloride reduces lithium entry and can lessen the concentrating defect.

152. C. Recurrent intradialytic hypotension in a patient with large interdialytic gains often reflects an ultrafiltration rate that exceeds plasma refilling. Lowering the fluid-removal burden through sodium/fluid management and, when needed, longer treatment time reduces the required ultrafiltration rate.

153. B. Acquired cystic kidney disease becomes common with prolonged kidney failure and dialysis and is associated with an increased risk of renal cell carcinoma. New hematuria warrants appropriate structural evaluation.

154. B. Lithium is readily dialyzable because it is small, water soluble, and minimally protein bound. Severe poisoning, kidney failure, or significant neurologic toxicity may warrant hemodialysis, with monitoring for postdialysis rebound.

155. E. The cornerstone of BK nephropathy management is a controlled reduction in immunosuppression to allow antiviral immunity to recover. The process must be balanced against rejection risk and monitored closely.

156. B. Modern IgA nephropathy management begins with optimized supportive care, including blood-pressure and proteinuria reduction. Selected patients who remain at high risk may be candidates for disease-specific therapies based on current guideline criteria and individual risk.

157. C. Volume and sodium depletion increase proximal sodium and lithium reabsorption. Thiazides can therefore raise lithium concentrations substantially, as can NSAIDs and RAAS blockers in susceptible patients.

158. C. Citrate chelates calcium and is normally metabolized to bicarbonate. In severe liver dysfunction or shock, citrate can accumulate, causing a rising total-to-ionized calcium ratio, ionized hypocalcemia, and metabolic abnormalities.

159. E. Severe secondary hyperparathyroidism can produce high-turnover bone disease, classically osteitis fibrosa, with increased osteoclastic and osteoblastic activity and characteristic radiographic changes.

160. A. The proximal tubule reabsorbs filtered glucose, amino acids, phosphate, uric acid, and most bicarbonate. Generalized proximal dysfunction produces Fanconi syndrome with loss of multiple solutes.

161. D. Cortisol excess can increase blood pressure through mineralocorticoid and vascular effects and may cause hypokalemia in severe cases. Endocrine testing is guided by the clinical phenotype.

162. B. Reduced kidney function, hypertension, and proteinuria increase risks such as preeclampsia, preterm delivery, fetal growth restriction, and maternal kidney decline. Medication review and coordinated nephrology-obstetric care are important.

163. C. A very dilute urine in acute hypotonic hyponatremia points to appropriate suppression of ADH. Excess free-water intake can still overwhelm maximal renal dilution, especially during endurance exercise when intake is extreme.

164. A. Synpharyngitic gross hematuria with normal complement is characteristic of IgA nephropathy. Postinfectious glomerulonephritis more often follows infection after a latent period and commonly has low complement.

165. C. A rapid rise in serum sodium in chronic hyponatremia, especially in malnutrition, alcoholism, hypokalemia, or liver disease, increases the risk of osmotic demyelination. Further correction should be prevented and, when necessary, actively relowered under close monitoring.

166. B. Chronic gastrointestinal alkali loss and low urine volume after ileostomy can produce acidic urine that favors uric acid precipitation. Uric acid stones are radiolucent on plain radiographs but visible on noncontrast CT.

167. A. The combination of a high-anion-gap metabolic acidosis, elevated osmolal gap, and urinary calcium oxalate crystals is characteristic of ethylene glycol poisoning. Fomepizole inhibits alcohol dehydrogenase and prevents formation of toxic metabolites; severe poisoning may also require hemodialysis.

168. C. Atherosclerotic renovascular disease can present with resistant or abruptly worsening hypertension, asymmetric kidneys, and recurrent flash pulmonary edema. The clinical context guides imaging and management.

169. E. Acute neurologic emergencies require condition-specific blood-pressure management. Excessive pressure can worsen bleeding, whereas overly aggressive reduction can impair cerebral perfusion.

170. C. CKD-associated pruritus is common, especially in dialysis patients, and its pathophysiology is multifactorial. Management includes optimizing kidney-failure care and using targeted symptomatic therapies when needed.

171. E. Severe hyperproteinemia can cause pseudohyponatremia when sodium is measured with methods that assume a normal plasma-water fraction. Tonicity remains normal because the sodium concentration in plasma water is not actually reduced.

172. E. RAAS blockade and mineralocorticoid receptor antagonism can both reduce distal potassium excretion. CKD and other risk factors increase the chance of clinically important hyperkalemia, so laboratory monitoring is essential.

173. B. Primary FSGS can recur rapidly after transplantation, consistent with a circulating permeability factor in susceptible patients. Abrupt nephrotic-range proteinuria soon after transplant is a classic presentation.

174. B. Broad HLA sensitization increases the likelihood of reacting to potential donors and can prolong waiting time. Detailed antibody testing and allocation strategies help identify acceptable donors and avoid clinically important donor-specific antibody.

175. A. Acute interstitial nephritis commonly follows medication exposure and can present with AKI, pyuria, white-cell casts, hematuria, or systemic hypersensitivity findings. The classic fever-rash-eosinophilia triad is neither sensitive nor required.

176. C. Fibromuscular dysplasia is a nonatherosclerotic, noninflammatory arteriopathy that often affects renal arteries and can produce renovascular hypertension. A string-of-beads appearance is classic for medial fibroplasia.

177. E. Interdialytic weight gain largely reflects sodium and water intake. Dietary sodium reduction decreases thirst and can make fluid restriction and volume control more achievable.

178. B. ACE inhibitors and ARBs can injure the developing fetal kidneys and are avoided in pregnancy. Chronic hypertension should instead be treated with agents with established pregnancy safety.

179. D. Persistent metabolic acidosis in CKD promotes protein catabolism, bone buffering, and other complications. Alkali therapy may be used when appropriate, while avoiding excessive sodium load or alkalosis.

180. B. ADPKD is associated with intracranial aneurysms as well as polycystic liver disease and selected cardiovascular abnormalities. Screening decisions are individualized based on factors such as family history, prior rupture, high-risk occupation, and patient preference.

181. B. Obesity and reduced nephron number can produce adaptive glomerular hyperfiltration and secondary FSGS. Management emphasizes treatment of the driver and reduction of intraglomerular pressure rather than reflexive immunosuppression.

182. A. CKD markedly increases cardiovascular risk, and statin-based therapy is recommended for many adults with non-dialysis CKD according to age and cardiovascular risk. New initiation in established dialysis is a different evidence question, but existing indications before dialysis remain important.

183. B. A urine osmolality near the physiologic minimum indicates that vasopressin is suppressed and the kidney is attempting to excrete free water. In primary polydipsia the problem is water intake that exceeds excretory capacity, not inappropriate antidiuresis.

184. D. A CMV-positive donor to a CMV-negative recipient is a high-risk combination because the recipient lacks prior immunity and can acquire primary CMV infection from the graft.

185. B. Acute pancreatitis can cause hypocalcemia as free fatty acids generated by fat necrosis bind calcium and form soaps. Severe illness may add other contributors, but saponification is the classic mechanism.

186. E. AKI definitions incorporate both serum creatinine change and urine output. Sustained oliguria can identify clinically important AKI even before a large creatinine rise is apparent.

187. C. Winter's formula estimates $PaCO_2$ as 1.5 times bicarbonate plus 8, plus or minus 2. With bicarbonate 12, the expected $PaCO_2$ is about 26 mm Hg; a substantially different value implies an additional respiratory disorder.

188. D. PLA2R autoantibodies are strongly associated with primary membranous nephropathy. The pathology is characterized by subepithelial immune deposits and granular capillary-wall immunofluorescence.

189. A. Cloudy effluent and abdominal pain with an elevated dialysate white-cell count and neutrophil predominance strongly suggest PD-associated peritonitis. Cultures should be obtained promptly and empiric intraperitoneal antibiotics started according to local protocols.

190. C. Conventional PD fluids often use dextrose to create an osmotic gradient. Some glucose is absorbed systemically, contributing to caloric load, hyperglycemia, dyslipidemia, and weight gain in susceptible patients.

191. E. Tacrolimus is metabolized by CYP3A and transported by P-glycoprotein. Strong inhibitors such as clarithromycin and azole antifungals can sharply raise tacrolimus concentrations and toxicity.

192. D. The renal medulla is hypoxic, acidic, and hypertonic, conditions that promote sickling. Recurrent medullary ischemia can impair concentrating ability and cause papillary necrosis and hematuria.

193. C. A strongly positive cytotoxic crossmatch indicates clinically significant preformed antibodies against donor cells. Proceeding without an appropriate desensitization strategy can risk hyperacute or severe early antibody-mediated rejection.

194. D. Repeated venipuncture and central venous catheterization can damage veins or cause stenosis, compromising future arteriovenous access. Vessel preservation is therefore important in patients likely to need hemodialysis.

195. A. Bartter syndromes impair thick ascending-limb salt transport, producing renal salt wasting, secondary hyperaldosteronism, hypokalemic alkalosis, and often hypercalciuria. The physiology resembles chronic loop-diuretic exposure.

196. C. Medullary sponge kidney is characterized by cystic dilation of collecting ducts in the renal medulla and is associated with nephrocalcinosis, hematuria, urinary tract infection, distal acidification defects, and recurrent stones.

197. E. Autoimmune tubulointerstitial disease, including Sjogren syndrome, can cause distal RTA. The combination of non-anion-gap acidosis, hypokalemia, inappropriately high urine pH, hypocitraturia, and calcium phosphate stones is characteristic.

198. B. Morphine metabolites are renally cleared and can accumulate when kidney function is poor, prolonging analgesic and respiratory effects. Opioid selection and dosing should account for renal clearance and active metabolites.

199. B. Chronic loop-diuretic therapy increases distal sodium reabsorption. Adding a thiazide-type diuretic can provide sequential nephron blockade, but careful monitoring is required for volume depletion, hyponatremia, and hypokalemia.

200. E. Light-chain deposition disease is a monoclonal immunoglobulin deposition disorder characterized by nonamyloid deposits, often along glomerular and tubular basement membranes. It can cause proteinuria, hematuria, and kidney failure.

201. A. The rise in anion gap is 8 mEq/L, whereas bicarbonate has fallen by 16 mEq/L. Because the bicarbonate decrease substantially exceeds the rise in anion gap, an additional normal-anion-gap metabolic acidosis is suggested.

202. D. Gitelman syndrome resembles chronic thiazide exposure: salt wasting, secondary hyperaldosteronism, hypokalemic alkalosis, hypomagnesemia, and hypocalciuria. Bartter syndromes more closely resemble loop-diuretic physiology and often have higher urinary calcium.

203. B. More frequent or longer dialysis schedules can reduce interdialytic fluid gains per treatment and permit lower ultrafiltration rates. This can improve blood pressure and volume control in selected patients.

204. C. Resistant hypertension with hypokalemia and metabolic alkalosis should raise suspicion for primary aldosteronism. Screening is performed with aldosterone and renin measurements, typically interpreted as an aldosterone-renin ratio under appropriate testing conditions.

205. B. Activated macrophages in granulomatous disease can express 1-alpha-hydroxylase and generate excess calcitriol outside normal renal feedback control. Increased intestinal calcium absorption can then cause hypercalcemia with suppressed PTH.

206. D. Hyperventilation lowers $PaCO_2$ and produces primary respiratory alkalosis. In an acute process, renal bicarbonate loss has not yet reached the magnitude seen with chronic hypocapnia.

207. E. Coarctation classically causes upper-extremity hypertension with lower-extremity pulse delay or lower pressures. It is an important secondary cause in younger patients.

208. D. Modern CKD blood-pressure recommendations depend on standardized office measurement. Rest period, positioning, appropriate cuff size, repeated readings, and validated equipment materially influence the values used for treatment decisions.

209. E. Urease-producing organisms split urea to ammonia, raising urine pH and promoting struvite precipitation. These infection stones can grow rapidly into branching staghorn calculi.

210. E. Aminoglycosides accumulate in proximal tubular cells and can cause dose- and duration-related acute tubular injury, often with nonoliguric kidney dysfunction. Therapeutic drug monitoring and dose adjustment reduce risk.

211. D. Dialysis disequilibrium syndrome is linked to rapid reduction in plasma urea and other osmoles, leaving the brain relatively hyperosmolar and promoting cerebral edema. Slow, gentle initial dialysis reduces risk in markedly azotemic patients.

212. E. Transplant renal artery stenosis can cause resistant hypertension, graft dysfunction, fluid retention, and a bruit over the allograft. Doppler ultrasonography is a common initial diagnostic study.

213. A. Serum albumin in dialysis patients is influenced by inflammation, comorbidity, protein losses, volume status, and nutritional intake. A low value should trigger a broader clinical assessment rather than a simple instruction to eat more protein.

214. B. Bilateral ureteral obstruction from malignancy can cause severe postrenal AKI. Upper-tract decompression with stents or nephrostomy may be needed when lower-tract catheterization cannot relieve the obstruction.

215. C. A patient with CKD can have additional causes of metabolic acidosis, including diarrhea, tubular disorders, ketoacidosis, lactic acidosis, or medication effects. Persistent or disproportionate acidosis deserves a broader evaluation.

216. D. Tenofovir can injure proximal tubular cells and cause a Fanconi-like syndrome with phosphate, glucose, uric acid, and bicarbonate wasting. Kidney function and tubular abnormalities should be monitored in at-risk patients.

217. C. Microscopic polyangiitis is a pauci-immune small-vessel vasculitis often associated with MPO-ANCA. It frequently causes necrotizing glomerulonephritis and may cause pulmonary capillaritis without granulomatous ENT disease.

218. A. Complement-mediated HUS results from dysregulated alternative complement activity and can cause recurrent or severe TMA. Complement inhibition is a key disease-specific therapy when the diagnosis is established.

219. B. Loss of functioning renal interstitial cells reduces erythropoietin production and contributes substantially to the hypoproliferative anemia of CKD. Iron deficiency, inflammation, blood loss, and shortened red-cell survival can add to the anemia.

220. D. Calcineurin inhibitors can cause dose-related renal vasoconstriction, hypertension, hyperkalemia, neurotoxicity, and acute kidney dysfunction. Drug levels and interacting medications should be reviewed.

221. E. Thiazide-type diuretics reduce urinary calcium excretion and can help prevent recurrent calcium stones in appropriately selected hypercalciuric patients. Sodium restriction enhances the hypocalciuric effect.

222. D. Dialysis is a medical treatment that may be declined or withdrawn by an informed patient. Careful communication, assessment of decision-making capacity, symptom control, and palliative support are essential.

223. B. A hydrothorax with glucose-rich pleural fluid in a PD patient suggests movement of dextrose-containing dialysate through a diaphragmatic defect. The right side is affected more often.

224. A. Masked hypertension is present when clinic blood pressure appears normal but out-of-office readings are elevated. It carries cardiovascular risk and is detected by ambulatory or reliable home monitoring.

225. D. Rapid reductions in plasma osmolality can worsen cerebral edema. In patients with intracranial pathology, slower and carefully prescribed kidney replacement may be favored to minimize osmotic gradients.

226. E. Hexagonal urinary crystals are strongly suggestive of cystine. Cystinuria is an inherited defect in proximal tubular reabsorption of dibasic amino acids and can cause recurrent stones at a young age.

227. B. Chronic immunosuppression markedly increases the risk and aggressiveness of certain malignancies, especially nonmelanoma skin cancers. Prevention includes sun protection, dermatologic surveillance, and individualized immunosuppression review.

228. B. Remission induction for severe ANCA-associated vasculitis uses rituximab or cyclophosphamide with glucocorticoids, with modern regimens aiming to limit steroid toxicity. Plasma exchange is reserved for selected clinical circumstances rather than routine use in all patients.

229. C. Long-term transplant care extends beyond graft function. Recipients face increased risks of infection, malignancy, cardiovascular disease, metabolic complications, and medication toxicity and need coordinated preventive care.

230. E. Constipation can alter catheter position or compress drainage pathways and is a common reversible cause of poor PD outflow. Mechanical causes should be assessed when effluent is clear and infection is not suggested.

231. C. Kidney perfusion requires adequate circulation, but excessive fluid accumulation is also associated with harm in critical illness. Resuscitation is guided by perfusion, responsiveness, and ongoing reassessment rather than by urine output alone.

232. B. AL amyloidosis is caused by misfolded monoclonal light chains that form Congo red-positive fibrils. Kidney disease often manifests as heavy albuminuria or nephrotic syndrome.

233. C. Glucosuria increases urinary osmole excretion and water loss, so the urine is relatively concentrated despite a high urine volume. This distinguishes osmotic diuresis from a pure water diuresis such as diabetes insipidus.

234. E. A heme-positive dipstick with few or no red blood cells suggests myoglobin or free hemoglobin. In rhabdomyolysis, myoglobin contributes to tubular toxicity and obstruction, especially with hypovolemia and aciduria.

235. A. High-dose intravenous acyclovir can precipitate in renal tubules, especially with rapid infusion or volume depletion. Hydration and appropriate dosing reduce risk.

236. B. Anti-GBM disease can cause a pulmonary-renal syndrome and crescentic glomerulonephritis. Linear IgG staining along the glomerular basement membrane is the defining immunopathologic pattern.

237. E. External compression of renal parenchyma can reduce perfusion and activate the renin-angiotensin-aldosterone system, causing Page kidney hypertension. Causes include subcapsular hematoma and other compressive lesions.

238. A. In non-dialysis CKD, the optimal PTH level is not defined by a single universal target. Serial trends and correctable abnormalities such as phosphate load, calcium balance, and vitamin D deficiency are important.

239. E. Diabetic hyporeninemic hypoaldosteronism and RAAS blockade both reduce distal potassium and hydrogen secretion. The typical pattern is hyperkalemia with a mild normal-anion-gap metabolic acidosis.

240. E. Hepatorenal syndrome reflects severe splanchnic vasodilation with secondary renal vasoconstriction. Vasoconstrictor therapy plus albumin can improve effective arterial circulation in selected patients while liver transplantation addresses the underlying disease.