

COMPREHENSIVE MOCK EXAM 6

Supplemental to: Transplant Hepatology Board Review 2026-2027

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ABIM Transplant Hepatology Certification Examination - 240 questions, A-E single-best-answer, domain-weighted to the 2026 ABIM blueprint

Board-Review Questions

1. A 14-month-old girl with biliary atresia had a technically successful Kasai procedure in infancy. Bilirubin is now 2.0 mg/dL, but she has had four admissions for culture-proven ascending cholangitis, progressive splenomegaly, and falling weight-for-age. What is the best next step?
- A. Repeat Kasai portoenterostomy because recurrent cholangitis after the first year is usually caused by a correctable anastomotic narrowing.
 - B. Refer for liver transplant evaluation because recurrent cholangitis and growth failure can justify transplantation before severe synthetic dysfunction.
 - C. Defer transplant referral until INR rises because preserved coagulation excludes clinically important progression of biliary atresia.
 - D. Place a TIPS to reduce portal pressure because this also prevents recurrent bacterial cholangitis in biliary atresia.
 - E. Start chronic prednisone because post-Kasai cholangitis is primarily an immune-mediated inflammatory disorder rather than an infectious complication.
2. A 5-year-old boy with biliary atresia and native liver has exertional dyspnea and clubbing. Oxygen saturation is 88% upright and 94% supine. Chest radiography is unrevealing. Which test best evaluates the suspected liver-related pulmonary complication?
- A. Right-heart catheterization to document an elevated pulmonary vascular resistance as the expected cause of orthodeoxia.
 - B. Contrast-enhanced transthoracic echocardiography to detect delayed intrapulmonary passage of microbubbles.
 - C. Ventilation-perfusion scanning to prove chronic pulmonary embolism as the usual pulmonary complication of biliary atresia.
 - D. Bronchoscopy with lavage to identify occult recurrent aspiration as the defining cause of liver-related hypoxemia.
 - E. Cardiac MRI to demonstrate an atrial septal defect because intracardiac shunting is required for hepatopulmonary syndrome.
3. An 8-month-old infant with biliary atresia awaiting liver transplantation has severe cholestasis, poor weight gain, and steatorrhea despite standard formula. Which nutritional intervention is most appropriate?

- A.** Restrict dietary protein to less than 1 g/kg/day because protein intake is the main driver of pediatric hepatic encephalopathy.
- B.** Eliminate all dietary fat because cholestasis prevents absorption of both long-chain and medium-chain triglycerides to the same degree.
- C.** Limit total calories to prevent hepatomegaly because catch-up growth before transplantation increases operative technical risk.
- D.** Use energy-dense enteral nutrition with medium-chain triglycerides and replacement of fat-soluble vitamins, adding tube feeds if needed.
- E.** Use parenteral nutrition as first-line therapy because enteral feeding is contraindicated in infants with portal hypertension.

4. A 9-year-old child has chronic cholestasis and severe pruritus. Gamma-glutamyl transferase is very low despite markedly elevated bile acids. Which diagnosis is most consistent with this biochemical pattern?

- A.** Alagille syndrome with paucity of intrahepatic bile ducts and characteristically high gamma-glutamyl transferase.
- B.** Primary sclerosing cholangitis with multifocal duct strictures and a typically elevated cholestatic enzyme profile.
- C.** Choledochal cyst causing extrahepatic bile duct obstruction and a prominent rise in gamma-glutamyl transferase.
- D.** Cystic-fibrosis cholangiopathy causing inspissated bile and a predominantly high gamma-glutamyl transferase cholestasis.
- E.** Progressive familial intrahepatic cholestasis due to a canalicular bile transport defect.

5. A 24-year-old with Wilson disease presents with jaundice, Coombs-negative hemolysis, INR 3.4, encephalopathy, and rapidly worsening renal function. Which feature most strongly affects immediate prognosis and management?

- A.** The presence of hemolysis predicts spontaneous recovery once oral zinc is started, so transplantation can usually be deferred.
- B.** A normal ceruloplasmin level would reliably exclude Wilson disease and permit routine supportive management outside a transplant center.

- C.** Neurologic symptoms make transplantation contraindicated because the hepatic copper defect remains present after graft replacement.
- D.** Chelation with penicillamine acts rapidly enough in acute liver failure that transplant evaluation should wait for a treatment response.
- E.** Wilson-associated acute liver failure has a high risk of death without urgent liver transplantation and should trigger immediate transplant-center management.

6. A patient with PiZZ alpha-1 antitrypsin deficiency undergoes successful liver transplantation for decompensated cirrhosis. Which mechanism explains why alpha-1 antitrypsin liver disease does not recur in the graft?

- A.** The recipient's bone marrow replaces donor hepatocytes, so the graft gradually acquires the recipient's normal alpha-1 antitrypsin phenotype.
- B.** Calcineurin inhibitors prevent polymerization of Z protein in hepatocytes, eliminating the inherited defect despite the recipient genotype.
- C.** Donor hepatocytes produce the donor alpha-1 antitrypsin phenotype, removing the recipient liver's production of misfolded Z protein.
- D.** Liver transplantation corrects the SERPINA1 mutation in all recipient tissues, including lung epithelium and circulating leukocytes.
- E.** Removal of portal hypertension reverses the abnormal hepatic protein folding process even though the graft retains the recipient genotype.

7. A 17-year-old with ornithine transcarbamylase deficiency has recurrent life-threatening hyperammonemic crises despite optimized scavenger therapy and dietary management. Bilirubin and INR are normal between episodes. What is the most appropriate definitive therapy to discuss?

- A.** TIPS creation, because diverting portal blood away from the liver decreases ammonia production in urea-cycle disorders.
- B.** Kidney transplantation, because the kidney is the principal site of ornithine transcarbamylase activity relevant to ammonia detoxification.
- C.** Liver transplantation, because replacing hepatocytes can correct the urea-cycle enzyme defect despite normal conventional liver synthetic tests.
- D.** Continued medical therapy until cirrhosis develops, because normal INR precludes a metabolic indication for liver transplantation.

E. Pancreas transplantation, because restoration of insulin secretion stabilizes hepatic nitrogen metabolism and prevents hyperammonemia.

8. A 6-year-old with Crigler-Najjar syndrome type I requires prolonged daily phototherapy and has had two episodes of bilirubin-induced neurologic dysfunction despite maximal treatment. Which definitive intervention is most appropriate?

A. Liver transplantation to provide functional bilirubin conjugation and prevent further kernicterus.

B. Splenectomy to reduce bilirubin production because chronic hemolysis is the primary defect in Crigler-Najjar syndrome type I.

C. TIPS placement to lower portal pressure because unconjugated hyperbilirubinemia is caused by portosystemic shunting.

D. Long-term ursodeoxycholic acid because increasing bile flow restores deficient UDP-glucuronosyltransferase activity.

E. Cholecystectomy because recurrent pigment stones are the source of the persistent unconjugated hyperbilirubinemia.

9. A 39-year-old with erythropoietic protoporphyria undergoes liver transplantation for rapidly progressive protoporphyric liver failure. Which additional issue is most important after transplantation?

A. Ongoing marrow production of protoporphyrin can injure the new liver, so hematologic strategies that reduce the source must be considered.

B. The disease is cured by liver transplantation because hepatocytes are the sole source of excess protoporphyrin in this disorder.

C. Recurrent disease is prevented by calcineurin inhibition because T-cell activation drives protoporphyrin overproduction after transplantation.

D. Long-term biliary stenting is required because recurrent disease occurs only through obstruction of the donor common bile duct.

E. Therapeutic phlebotomy is mandatory because iron overload is the principal mechanism of recurrent graft injury in protoporphyria.

10. A 28-year-old with glycogen storage disease type I has multiple hepatic adenomas, one of which has enlarged to 7 cm despite excellent metabolic control. Which concern most strongly changes management?

- A.** Adenomas in glycogen storage disease are uniformly benign, so size progression does not alter surveillance or treatment.
- B.** The main danger is portal-vein thrombosis from compression, whereas hemorrhage and malignant transformation are not clinically relevant.
- C.** Liver transplantation is contraindicated in metabolic disease because the enzyme defect will necessarily recur in donor hepatocytes.
- D.** Progressive large adenomas carry hemorrhagic and malignant-transformation risk and may prompt resection or transplant evaluation depending on disease distribution.
- E.** Any hepatic adenoma mandates immediate transplantation because local resection is never an option in glycogen storage disease.

11. A patient with hereditary transthyretin amyloidosis and progressive neuropathy is considered for liver transplantation. What is the disease-specific rationale for replacing the liver?

- A.** Transplantation removes established amyloid from peripheral nerves because donor hepatocytes secrete enzymes that dissolve existing deposits.
- B.** The procedure corrects the germline TTR mutation in neural tissue, preventing all future amyloid formation throughout the body.
- C.** Immunosuppression suppresses antibody-mediated amyloid deposition, which is the primary driver of hereditary transthyretin neuropathy.
- D.** The liver is the major source of circulating variant transthyretin, so graft replacement markedly reduces production of the amyloidogenic protein.
- E.** Portal decompression reduces transthyretin synthesis, so the benefit is equivalent to a successful TIPS without graft replacement.

12. A 31-year-old with cystic fibrosis has decompensated portal hypertension and FEV1 24% predicted despite maximal pulmonary therapy. Which transplant planning principle is most appropriate?

- A.** Proceed directly with isolated liver transplantation because lung function is expected to normalize when portal hypertension is corrected.

- B.** Perform isolated lung transplantation because advanced cystic-fibrosis liver disease reliably regresses after correction of hypoxemia.
- C.** Use TIPS as definitive therapy because cystic-fibrosis portal hypertension never progresses to a transplant indication.
- D.** Exclude transplantation entirely because cystic fibrosis is a systemic genetic disease that inevitably recurs in both donor organs.
- E.** Assess candidacy for a combined liver-lung strategy because severe pulmonary disease may make isolated liver transplantation unsafe.

13. A 44-year-old with diffuse Caroli disease has recurrent bacteremic cholangitis, intrahepatic stones throughout both lobes, and secondary biliary cirrhosis. Endoscopic drainage provides only temporary relief. What is the best definitive treatment?

- A.** Left hepatectomy because removing one lobe eliminates the congenital duct abnormality even when both lobes are extensively involved.
- B.** Permanent external biliary drainage because continuous decompression prevents progression of diffuse congenital intrahepatic duct ectasia.
- C.** Whipple pancreaticoduodenectomy because Caroli disease originates in the distal common bile duct and spreads proximally over time.
- D.** TIPS placement because reducing portal pressure directly prevents intrahepatic stone formation and recurrent cholangitis.
- E.** Liver transplantation because diffuse bilateral disease with recurrent infection and cirrhosis cannot be cured by segmental resection.

14. A 12-year-old has cholestasis, characteristic facies, butterfly vertebrae, and a peripheral pulmonary artery stenosis. Gamma-glutamyl transferase is markedly elevated. Which diagnosis best unifies the findings?

- A.** Progressive familial intrahepatic cholestasis type 1, which typically produces low GGT cholestasis without congenital cardiac lesions.
- B.** Wilson disease, which causes copper accumulation with neurologic disease rather than syndromic bile-duct paucity.
- C.** Crigler-Najjar syndrome type I, which produces unconjugated hyperbilirubinemia without cholestasis or structural cardiac findings.

- D.** Alagille syndrome caused by impaired Notch signaling and paucity of intrahepatic bile ducts.
- E.** Caroli disease, which causes saccular intrahepatic duct dilatation rather than bile-duct paucity and characteristic facies.

15. A 52-year-old woman with newly diagnosed primary biliary cholangitis has alkaline phosphatase 3 times the upper limit of normal, positive antimitochondrial antibodies, and preserved hepatic synthetic function. What is first-line disease-modifying therapy?

- A.** High-dose prednisone as long-term monotherapy because PBC is primarily a hepatocellular autoimmune disease responsive to corticosteroids.
- B.** Routine ERCP with biliary dilation because PBC is caused by dominant extrahepatic duct strictures that require mechanical treatment.
- C.** Immediate transplant listing because an elevated alkaline phosphatase alone predicts near-term liver failure despite preserved function.
- D.** Broad-spectrum antibiotics because chronic occult bacterial cholangitis is the principal mechanism of progressive small-duct injury in PBC.
- E.** Ursodeoxycholic acid at standard weight-based dosing, with later assessment of biochemical response.

16. A 49-year-old with PBC has been taking ursodeoxycholic acid reliably for 18 months. Alkaline phosphatase remains 2.5 times normal and bilirubin has begun to rise. What does this pattern imply?

- A.** Persistent alkaline phosphatase elevation has no prognostic significance once adherence to ursodeoxycholic acid is documented.
- B.** The pattern proves extrahepatic biliary obstruction and should automatically lead to repeated therapeutic ERCP sessions.
- C.** An inadequate biochemical response confers higher progression risk and should prompt reassessment and consideration of additional PBC therapy or transplant timing.
- D.** Rising bilirubin in PBC is usually caused by hemolysis and does not influence transplant-free prognosis or treatment decisions.
- E.** The only appropriate next step is permanent discontinuation of ursodeoxycholic acid because lack of normalization indicates drug toxicity.

17. A patient with decompensated PBC, prior variceal bleeding, and ascites has an incomplete response to ursodeoxycholic acid. Which principle is most important when considering second-line therapy?

- A.** Escalate automatically to the highest available dose of any second-line cholestatic agent because portal hypertension does not affect drug safety.
- B.** Stop ursodeoxycholic acid and use chronic systemic corticosteroids because decompensation proves conversion to autoimmune hepatitis.
- C.** Perform serial ERCP every three months because biochemical nonresponse in PBC reflects occult dominant biliary strictures.
- D.** Avoid agents that are contraindicated or unsafe in decompensated cirrhosis and prioritize transplant evaluation for progressive advanced disease.
- E.** Delay transplant evaluation until bilirubin exceeds 20 mg/dL because ascites and prior variceal bleeding are not transplant-relevant complications.

18. A 23-year-old woman presents with aminotransferases above 1500 U/L, IgG elevation, positive smooth-muscle antibody, INR 2.2, and no viral or drug cause. Which diagnosis is most likely?

- A.** Primary biliary cholangitis, which usually presents with isolated cholestasis and antimitochondrial antibodies rather than severe hepatitis.
- B.** Primary sclerosing cholangitis, which is defined by multifocal biliary strictures and typically produces a cholestatic laboratory pattern.
- C.** Hereditary hemochromatosis, which rarely causes abrupt aminotransferases above 1500 U/L with an autoimmune serologic profile.
- D.** Severe acute autoimmune hepatitis, which can present with marked hepatocellular injury and coagulopathy even without known chronic liver disease.
- E.** Gilbert syndrome, which causes isolated unconjugated hyperbilirubinemia without coagulopathy or marked aminotransferase elevation.

19. A 36-year-old with severe autoimmune hepatitis has INR 2.4 but no encephalopathy. Corticosteroids are started after infection is excluded. What is the most appropriate parallel strategy?

- A.** Avoid transplant referral until at least three months of corticosteroid therapy have failed because early response cannot be assessed sooner.

- B.** Add a TIPS immediately because reducing portal pressure is the principal treatment for acute severe autoimmune hepatitis.
- C.** Monitor closely at a transplant center and assess early biochemical response because failure to improve should trigger urgent transplant planning.
- D.** Begin azathioprine at full dose on day one despite severe jaundice because it acts faster than corticosteroids in acute severe disease.
- E.** Give empiric chelation therapy because severe autoimmune hepatitis is frequently caused by occult Wilson disease after age 30.

20. A liver transplant recipient whose original disease was autoimmune hepatitis develops rising aminotransferases years later. Biopsy shows interface hepatitis with plasma cells, and vascular and biliary imaging are normal. What should be high on the differential?

- A.** Ischemic cholangiopathy.
- B.** Hepatic artery thrombosis.
- C.** Recurrent autoimmune hepatitis.
- D.** Recurrent primary biliary cholangitis.
- E.** Calcineurin-inhibitor toxicity.

21. A patient with long-standing inflammatory bowel disease develops pruritus and persistent alkaline-phosphatase elevation. Cholangiography demonstrates alternating narrow segments and ectatic segments throughout both intrahepatic and extrahepatic bile ducts. Which disorder best explains this pattern?

- A.** Primary biliary cholangitis.
- B.** Autoimmune hepatitis.
- C.** Budd-Chiari syndrome.
- D.** Primary sclerosing cholangitis.
- E.** Hereditary hemochromatosis.

22. A patient with established PSC develops rapidly worsening jaundice and a new high-grade hilar stricture. What is the best next diagnostic step?

- A.** Repeat MRCP in one year because tissue sampling is not useful when PSC already explains the underlying duct abnormalities.
- B.** ERCP for targeted sampling and therapeutic assessment of the clinically relevant stricture, including cytology with ancillary testing when appropriate.
- C.** Percutaneous biopsy of the hilar mass as the first test because transperitoneal needle sampling never affects later transplant options.
- D.** Empiric high-dose prednisone because an abrupt dominant stricture in PSC is most often autoimmune hepatitis overlap.
- E.** Immediate TIPS placement because decompression of portal pressure resolves malignant and benign hilar strictures in PSC.

23. A 38-year-old with PSC and ulcerative colitis asks about colorectal cancer prevention. Which approach is most appropriate?

- A.** Follow average-risk population screening intervals because PSC does not materially change colorectal cancer risk in ulcerative colitis.
- B.** Stop colonoscopic surveillance after liver transplantation because transplant immunosuppression eliminates the colitis-associated cancer risk.
- C.** Use annual abdominal ultrasound instead of colonoscopy because PSC-related colorectal cancers arise from the extrahepatic biliary tract.
- D.** Use intensified colonoscopic surveillance because PSC with inflammatory bowel disease carries a particularly high colorectal neoplasia risk.
- E.** Perform prophylactic colectomy in every patient at PSC diagnosis because surveillance cannot detect premalignant colonic lesions reliably.

24. A 46-year-old with PSC has repeated episodes of bacterial cholangitis requiring hospitalization despite endoscopic management. MELD remains modest because renal and synthetic function are preserved. What is the best transplant-related interpretation?

- A.** Recurrent severe cholangitis can justify transplant consideration even when the laboratory MELD score underestimates disease burden.
- B.** Transplantation is never appropriate until MELD exceeds 30 because recurrent cholangitis is not a recognized manifestation of advanced PSC.

C. TIPS is definitive therapy because lowering portal pressure prevents bacterial infection of structurally abnormal bile ducts.

D. Chronic corticosteroids should replace transplant evaluation because PSC is uniformly responsive to systemic immunosuppression.

E. Recurrent cholangitis proves cholangiocarcinoma, so transplantation is absolutely contraindicated without any further oncologic evaluation.

25. A 56-year-old with obstructive jaundice has diffuse pancreatic enlargement, elevated serum IgG4, and long smooth biliary strictures. Which diagnosis best fits?

A. Primary sclerosing cholangitis with pancreatic disease.

B. Primary biliary cholangitis with pancreatic disease.

C. Caroli disease with pancreatic involvement.

D. Ischemic cholangiopathy after arterial injury.

E. IgG4-related sclerosing cholangitis with autoimmune pancreatitis.

26. A patient with IgG4-related cholangitis has symptomatic biliary obstruction and no evidence of infection. What therapy is most likely to produce a rapid disease-specific response?

A. Systemic glucocorticoid therapy after appropriate diagnostic confirmation and exclusion of competing malignancy.

B. Ursodeoxycholic acid alone because IgG4-related cholangitis is a noninflammatory cholestatic disorder that does not respond to immunosuppression.

C. Immediate liver transplantation because fibrosis progresses irreversibly despite medical therapy in nearly all patients.

D. Long-term broad-spectrum antibiotics because occult bacterial cholangitis is the dominant mechanism of the IgG4-associated strictures.

E. TIPS placement because portal decompression directly reverses the inflammatory biliary narrowing and pancreatic enlargement.

27. A 50-year-old with hepatic sarcoidosis has marked portal hypertension, splenomegaly, and recurrent variceal bleeding despite only modest aminotransferase abnormalities. Which statement is most accurate?

- A.** Portal hypertension excludes sarcoidosis because granulomatous liver disease causes cholestasis but never clinically important vascular complications.
- B.** Normal aminotransferases prove the liver is not contributing to the patient's portal hypertension and eliminate the need for hepatology follow-up.
- C.** TIPS is contraindicated solely because granulomas are present, even when portal hypertensive complications otherwise justify decompression.
- D.** Liver transplantation cannot be used for sarcoidosis because granulomatous inflammation invariably causes immediate primary graft nonfunction.
- E.** Sarcoidosis can produce severe portal hypertensive complications that are disproportionate to routine liver-test abnormalities and may ultimately require transplantation.

28. A patient with established autoimmune hepatitis develops persistent alkaline phosphatase elevation and pruritus. Antimitochondrial antibodies are positive and biopsy shows florid duct lesions in addition to interface hepatitis. What is the best interpretation?

- A.** The findings exclude autoimmune hepatitis because any bile-duct injury proves that the original diagnosis was incorrect.
- B.** Primary sclerosing cholangitis is proven because antimitochondrial antibodies are specific for large-duct beading on cholangiography.
- C.** Drug-induced liver injury is the only diagnosis that can produce simultaneous interface hepatitis and florid duct lesions.
- D.** Budd-Chiari syndrome is most likely because hepatic venous obstruction typically causes antimitochondrial antibody positivity.
- E.** An autoimmune hepatitis-PBC overlap phenotype should be considered because both hepatitic and cholangitic features are present.

29. A patient with compensated autoimmune hepatitis has been in complete biochemical remission for two years on prednisone plus azathioprine and is troubled by steroid toxicity. What is the usual maintenance strategy?

- A.** Stop all immunosuppression immediately because two years of biochemical remission guarantees durable drug-free remission in autoimmune hepatitis.
- B.** Continue high-dose prednisone indefinitely because azathioprine cannot maintain remission without full-dose corticosteroid therapy.
- C.** Taper corticosteroid exposure while continuing an effective steroid-sparing agent such as azathioprine when tolerated.
- D.** Replace immunosuppression with ursodeoxycholic acid because autoimmune hepatitis becomes a cholestatic disorder after biochemical remission.
- E.** Perform elective liver transplantation because chronic immunosuppressive medication toxicity is itself an indication for graft replacement.

30. A 34-year-old with inflammatory bowel disease has cholestatic tests but a normal high-quality MRCP. Liver biopsy shows periductal fibrosis and duct injury consistent with sclerosing cholangitis. What is the most likely diagnosis?

- A.** Primary biliary cholangitis with small-duct injury.
- B.** Autoimmune hepatitis with cholestatic features.
- C.** Caroli disease limited to microscopic ducts.
- D.** Small-duct primary sclerosing cholangitis.
- E.** IgG4-related sclerosing cholangitis.

31. A liver transplant recipient for PSC has persistent active ulcerative colitis after transplantation. Which long-term issue remains important?

- A.** Colon surveillance can stop because calcineurin inhibitors eliminate colonic inflammation and reverse prior dysplasia risk.
- B.** Only biliary imaging is needed because all post-transplant malignancy risk in PSC is confined to the transplanted liver.
- C.** Prophylactic colectomy is mandatory in every recipient because colorectal cancer becomes inevitable after transplantation for PSC.
- D.** Continued colorectal neoplasia surveillance because inflammatory bowel disease-associated cancer risk persists after liver transplantation.

E. The risk of colorectal neoplasia disappears once the native liver is removed because PSC is the direct cause of colonic dysplasia.

32. Which histologic finding most strongly supports classic autoimmune hepatitis rather than a primary cholangiopathy?

A. Concentric periductal fibrosis around medium-sized bile ducts with duct obliteration and cholestatic injury.

B. Florid destructive cholangitis centered on small interlobular bile ducts with granulomatous inflammation.

C. Interface hepatitis with a lymphoplasmacytic infiltrate rich in plasma cells.

D. Ischemic-type bile-duct necrosis with biliary casts and preservation of the hepatic arterial endothelial lining.

E. Centrilobular sinusoidal congestion with zone 3 hepatocyte atrophy and prominent hepatic venous outflow obstruction.

33. A 61-year-old with PBC has severe cholestatic pruritus despite optimized ursodeoxycholic acid. There is no biliary obstruction. Which treatment strategy is reasonable for symptomatic relief?

A. Use a stepwise antipruritic approach beginning with a bile-acid sequestrant when feasible and escalating to other agents if symptoms persist.

B. Increase dietary fat markedly because stimulating bile flow is the most effective method of reducing cholestatic pruritus.

C. Start chronic opioid agonist therapy because endogenous opioid blockade worsens pruritus in cholestatic liver disease.

D. Perform serial ERCP because pruritus in PBC usually reflects a mechanically obstructed common bile duct even when imaging is normal.

E. Stop all PBC therapy because pruritus indicates an allergic reaction to ursodeoxycholic acid rather than the underlying cholestasis.

34. A 47-year-old woman with PBC has a fragility fracture and low bone mineral density. Which management principle is most appropriate?

- A.** Avoid antiresorptive therapy because all osteoporosis in cholestatic liver disease reverses spontaneously after ursodeoxycholic acid treatment.
- B.** Restrict vitamin D because fat-soluble vitamin supplementation accelerates biliary fibrosis in primary biliary cholangitis.
- C.** Use TIPS to improve bone density because portal decompression is the definitive therapy for hepatic osteodystrophy.
- D.** Treat osteoporosis directly and ensure adequate calcium and vitamin D while continuing management of the underlying cholestatic liver disease.
- E.** Defer treatment until after liver transplantation because fracture risk does not influence management before graft replacement.

35. A liver transplant candidate with decompensated cirrhosis is HBsAg positive with detectable HBV DNA. Which antiviral approach is most appropriate before transplantation?

- A.** Defer antiviral therapy until after transplantation because treatment before graft replacement increases the risk of HBV resistance and flare.
- B.** Use interferon-based therapy because interferon is the safest and most effective antiviral strategy in decompensated cirrhosis.
- C.** Start a potent nucleos(t)ide analogue with a high barrier to resistance and continue viral suppression through the transplant period.
- D.** Treat only if aminotransferases exceed ten times normal because HBV DNA does not affect transplant or recurrence risk.
- E.** Give a short course of lamivudine only during the week before surgery because long-term suppression is unnecessary once the native liver is removed.

36. A patient with decompensated HCV cirrhosis is being evaluated for transplantation. Which principle best guides direct-acting antiviral timing?

- A.** Treat every patient before listing because viral cure always removes the need for transplantation and cannot complicate allocation decisions.
- B.** Individualize treatment before versus after transplant according to disease severity, expected wait time, donor options, and the risk that improvement may alter transplant priority.

- C.** Never treat before transplant because direct-acting antivirals are contraindicated in all patients with decompensated cirrhosis.
- D.** Delay antiviral therapy until five years after transplantation because early treatment causes universal calcineurin-inhibitor toxicity.
- E.** Use interferon and ribavirin instead because modern direct-acting antiviral regimens have no role in transplant candidates.

37. A patient with chronic hepatitis D asks why control of hepatitis B remains important. Which statement is correct?

- A.** Hepatitis D is a fully independent DNA virus, so hepatitis B vaccination has no effect on the risk of acquiring HDV infection.
- B.** Hepatitis D replicates only in hepatocytes infected with hepatitis C virus, making HCV eradication the primary preventive strategy.
- C.** Hepatitis D is acquired only after transplantation from donor leukocytes and is unrelated to preexisting viral hepatitis status.
- D.** Hepatitis D depends on Epstein-Barr virus envelope proteins, explaining its association with post-transplant lymphoproliferative disorder.
- E.** Hepatitis D virus requires hepatitis B surface antigen for assembly and propagation, so HBV prevention and control remain central.

38. A solid-organ transplant recipient has persistent aminotransferase elevation for six months. HBV, HCV, CMV, and EBV studies are negative. HEV RNA is detectable. What is the most likely diagnosis?

- A.** Acute self-limited hepatitis E infection.
- B.** Autoimmune hepatitis after transplantation.
- C.** Chronic hepatitis E infection during immunosuppression.
- D.** Chronic ischemic hepatitis after transplantation.
- E.** Chronic hepatitis D infection after transplantation.

39. A patient with suspected HSV acute liver failure has fever, very high aminotransferases, coagulopathy, and rapidly evolving encephalopathy. Mucocutaneous lesions are absent. What is the best immediate management?

- A.** Withhold antiviral therapy until skin vesicles appear because HSV hepatitis cannot occur without visible mucocutaneous disease.
- B.** Begin oral valganciclovir because CMV is the only herpesvirus that causes fulminant hepatitis in immunocompetent adults.
- C.** Start intravenous acyclovir promptly while pursuing diagnostic testing and urgent liver-transplant evaluation.
- D.** Treat with high-dose corticosteroids because HSV-associated acute liver failure is primarily an immune-mediated process.
- E.** Wait for liver biopsy confirmation before therapy because empiric acyclovir worsens the prognosis of viral acute liver failure.

40. A cirrhotic transplant candidate lacks immunity to hepatitis A and B. Which preventive strategy is most appropriate?

- A.** Avoid both vaccines because inactivated viral vaccines precipitate hepatic decompensation in patients with cirrhosis.
- B.** Give only hepatitis A vaccine because hepatitis B vaccination is ineffective and contraindicated in chronic liver disease.
- C.** Wait until the first month after transplantation because vaccine responses are stronger under high-dose immunosuppression.
- D.** Use prophylactic antivirals instead of vaccination because neither HAV nor HBV vaccines are recommended for transplant candidates.
- E.** Vaccinate against both hepatitis A and hepatitis B before transplantation whenever feasible.

41. A patient with chronic HBV cirrhosis has undetectable HBV DNA on tenofovir. Which statement about hepatocellular carcinoma risk is correct?

- A.** Effective viral suppression lowers but does not eliminate HCC risk in cirrhosis, so surveillance should continue.

- B.** Undetectable HBV DNA eliminates HCC risk, allowing surveillance to stop once viral suppression has been sustained for one year.
- C.** HCC surveillance is unnecessary in HBV because liver cancer occurs only after HCV coinfection or heavy alcohol exposure.
- D.** Antiviral therapy increases HCC risk by delaying viral clearance, so surveillance should be intensified only while treatment is active.
- E.** HCC develops only when HBV DNA is detectable, making serial viral load measurement an adequate substitute for imaging surveillance.

42. A 29-year-old woman has hepatic-vein thrombosis, ascites, painful hepatomegaly, and a myeloproliferative neoplasm. She is hemodynamically stable and has no active bleeding. What is the initial disease-specific therapy for Budd-Chiari syndrome?

- A.** Immediate liver transplantation in every case because anticoagulation cannot improve hepatic venous outflow in Budd-Chiari syndrome.
- B.** Therapeutic anticoagulation while treating the underlying prothrombotic condition and assessing need for step-up intervention.
- C.** Long-term dual antiplatelet therapy instead of anticoagulation because venous thrombosis is platelet mediated in myeloproliferative disease.
- D.** Routine corticosteroids because hepatic-vein thrombosis in young adults is usually caused by autoimmune venulitis.
- E.** No antithrombotic therapy because portal hypertensive varices make anticoagulation absolutely contraindicated in Budd-Chiari syndrome.

43. A patient with Budd-Chiari syndrome remains ascitic with worsening liver function despite therapeutic anticoagulation and technically unsuitable focal lesions for angioplasty. Which next intervention is most appropriate before transplantation if anatomy permits?

- A.** Repeated large-volume phlebotomy because reducing blood volume is the primary treatment for persistent hepatic outflow obstruction.
- B.** ERCP with biliary stenting because cholestasis from Budd-Chiari is caused by secondary common bile duct narrowing.
- C.** TIPS to decompress the congested hepatic circulation and reduce portal hypertension.

D. Splenectomy because removal of splenic inflow restores hepatic venous patency and reverses ascites in most patients.

E. Permanent discontinuation of anticoagulation before any intervention because anticoagulation prevents shunt creation from succeeding.

44. An adult with repaired congenital heart disease and longstanding Fontan circulation has nodular liver morphology, thrombocytopenia, and splenomegaly. Which principle best applies to evaluation of Fontan-associated liver disease?

A. Use aminotransferase levels alone because they accurately stage fibrosis and portal hypertension in Fontan-associated liver disease.

B. Assume all nodularity represents hepatocellular carcinoma because benign regenerative nodules do not occur after Fontan surgery.

C. Assess both hepatic disease severity and Fontan hemodynamics because transplant planning may require coordinated heart-liver evaluation.

D. Exclude liver transplantation from consideration because elevated central venous pressure always causes immediate graft failure.

E. Perform TIPS routinely because creating an additional portosystemic shunt corrects the underlying Fontan hemodynamic problem.

45. Which hepatic hemodynamic pattern is most characteristic of chronic right-sided heart failure causing congestive hepatopathy?

A. Isolated presinusoidal portal hypertension with completely normal hepatic venous pressures and no sinusoidal congestion.

B. Primary obstruction of the common bile duct with secondary elevation of right atrial pressure and centrilobular necrosis.

C. Severe portal inflow reduction caused by splenic-artery vasoconstriction as the dominant mechanism of chronic hepatic congestion.

D. Elevated hepatic venous pressures with centrilobular congestion from transmission of high right-sided filling pressure.

E. Diffuse autoimmune destruction of hepatic venules mediated by antimitochondrial antibodies and independent of cardiac filling pressures.

46. On day 18 after allogeneic hematopoietic stem-cell transplantation, a patient has gained 8% body weight and develops tender hepatomegaly, tense ascites, rising bilirubin, and renal dysfunction. Large hepatic veins remain patent on Doppler. What disease-directed therapy should be considered for this severe syndrome?

- A.** Therapeutic anticoagulation with warfarin because the disorder is caused by macroscopic hepatic-vein thrombosis despite a normal Doppler study.
- B.** High-dose ursodeoxycholic acid alone because severe sinusoidal obstruction is a benign cholestatic reaction that does not cause multiorgan failure.
- C.** Defibrotide with intensive supportive care for severe sinusoidal obstruction syndrome.
- D.** Immediate ERCP because bilirubin elevation reflects sloughed biliary casts from conditioning-related common duct injury.
- E.** TIPS as routine first-line therapy because portal decompression reverses the endothelial injury responsible for all cases of sinusoidal obstruction syndrome.

47. A patient with sickle cell disease develops abrupt right-upper-quadrant pain, rapidly rising conjugated bilirubin, coagulopathy, and worsening encephalopathy during a vaso-occlusive crisis. Which urgent therapy should be considered for severe sickle cell hepatopathy?

- A.** High-dose iron supplementation because acute hepatic failure in sickle cell disease is usually caused by iron-deficiency ischemia.
- B.** TIPS placement because reducing portal pressure corrects the microvascular sickling that causes acute intrahepatic cholestasis.
- C.** Prednisone monotherapy because severe sickle-cell hepatopathy is primarily an autoimmune hepatitis triggered by vaso-occlusion.
- D.** Observation alone because hepatic complications of sickle cell disease are self-limited and do not progress to multiorgan failure.
- E.** Exchange transfusion to rapidly reduce the proportion of sickled erythrocytes while providing critical supportive care.

48. An adult with failing Fontan physiology is referred for transplant planning. Liver assessment shows chronic congestion. Which hepatic finding would most strongly favor a combined heart-liver strategy over isolated heart transplantation?

- A.** Mild aminotransferase elevation during diuretic therapy because any liver-test abnormality proves irreversible cirrhosis.
- B.** A transient bilirubin rise during an episode of acute heart failure that completely normalizes after decongestion.
- C.** Isolated hepatomegaly without fibrosis, portal hypertension, or impaired hepatic function because size alone predicts graft failure.
- D.** Normal platelet count and absence of varices because these findings establish severe portal hypertension requiring a liver graft.
- E.** Advanced irreversible hepatic disease with clinically significant portal hypertension rather than congestion alone.

49. A 45-year-old has splenomegaly, thrombocytopenia, large esophageal varices, and preserved albumin and INR. Imaging shows a patent portal vein and no cirrhotic morphology. Liver biopsy shows nodular regenerative hyperplasia without bridging fibrosis. What is the best diagnosis?

- A.** Portosinusoidal vascular disease with noncirrhotic portal hypertension.
- B.** Cryptogenic cirrhosis with portal hypertension.
- C.** Budd-Chiari syndrome with hepatic-vein obstruction.
- D.** Primary sclerosing cholangitis with portal hypertension.
- E.** Hepatorenal syndrome with advanced portal hypertension.

50. A patient with noncirrhotic presinusoidal portal hypertension has large varices. Which hemodynamic observation may occur despite clinically important portal pressure elevation?

- A.** The hepatic venous pressure gradient must exceed 20 mm Hg because all variceal formation requires severe sinusoidal resistance.
- B.** Right atrial pressure is always markedly elevated because presinusoidal portal hypertension is a manifestation of occult right-heart failure.
- C.** Portal pressure is normal because varices in portosinusoidal vascular disease arise from primary systemic venous obstruction.
- D.** Wedged hepatic venous pressure becomes zero because portal venous blood bypasses the liver completely in all affected patients.

E. The hepatic venous pressure gradient can be normal or only mildly elevated because the resistance is primarily presinusoidal.

51. A patient with portosinusoidal vascular disease has recurrent variceal bleeding despite optimized endoscopic and pharmacologic therapy, with preserved liver function and favorable vascular anatomy. Which intervention is reasonable?

A. Proceed directly to liver transplantation because noncirrhotic portal hypertension cannot be treated with portal decompression.

B. Consider TIPS for refractory portal hypertensive complications when anatomy and overall clinical status are appropriate.

C. Start chronic corticosteroids because obliterative portal venopathy is uniformly driven by autoimmune inflammation responsive to steroids.

D. Perform biliary sphincterotomy because portal hypertensive bleeding in this disorder usually arises from occult common bile duct obstruction.

E. Avoid all shunt procedures because preserved synthetic function makes decompression ineffective and predictably causes liver failure.

52. A patient with cirrhosis is found to have a recent, occlusive main portal-vein thrombosis extending into the proximal superior mesenteric vein. There is no active bleeding. What is the best management principle?

A. Consider therapeutic anticoagulation because recent extensive thrombosis can progress and may complicate future transplantation.

B. Observe without therapy because anticoagulation is never used in cirrhosis when gastroesophageal varices may be present.

C. Perform splenectomy because portal-vein thrombosis in cirrhosis is driven primarily by excessive splenic venous inflow.

D. Start dual antiplatelet therapy because platelet aggregation rather than venous stasis is the dominant mechanism of portal thrombosis.

E. List immediately for transplantation because portal-vein thrombosis itself is an absolute indication regardless of hepatic disease severity.

53. A transplant candidate with long-standing portal-vein thrombosis has numerous serpiginous periportal collaterals replacing the expected portal-vein lumen. What does this finding represent?

- A.** Acute hepatic-artery thrombosis with collaterals.
- B.** Cavernous transformation from chronic portal-vein thrombosis.
- C.** Budd-Chiari syndrome with venous collaterals.
- D.** Hepatic hemangiomas with portal channels.
- E.** Sinusoidal obstruction syndrome with collateralization.

54. A liver transplant candidate has chronic portal-vein thrombosis that threatens a conventional portal anastomosis. Which pretransplant strategy may restore portal inflow in selected patients?

- A.** Transjugular hepatic-vein embolization before transplant.
- B.** Permanent cessation of anticoagulation before transplant.
- C.** Repeated ERCP for portal-vein decompression.
- D.** Splenic irradiation to recreate portal inflow.
- E.** Portal-vein recanalization with TIPS when technically feasible.

55. A patient with pancreatitis develops isolated gastric varices but no esophageal varices. Liver tests and portal-vein imaging are normal. Which vascular lesion is most likely?

- A.** Main portal-vein thrombosis causing generalized portal hypertension.
- B.** Hepatic-vein thrombosis causing diffuse portal hypertension.
- C.** Inferior vena cava thrombosis causing systemic venous congestion.
- D.** Splenic-vein thrombosis causing left-sided portal hypertension.
- E.** Hepatic-artery thrombosis causing ischemic biliary injury.

56. A patient with cirrhosis has recurrent hepatic encephalopathy despite adherence to lactulose and rifaximin. Imaging reveals a very large spontaneous splenorenal shunt, and liver function is otherwise relatively preserved. What intervention may help?

- A.** Embolization or occlusion of the dominant spontaneous portosystemic shunt in a carefully selected patient.
- B.** Increase the shunt diameter with TIPS because additional portosystemic diversion improves ammonia clearance in refractory encephalopathy.
- C.** Start chronic benzodiazepine therapy because enhanced GABA signaling reverses the neuroinflammatory component of hepatic encephalopathy.
- D.** Perform splenectomy because removal of the spleen is required before a spontaneous splenorenal shunt can be closed safely.
- E.** Stop lactulose and rifaximin because persistent encephalopathy in the presence of a large shunt is no longer ammonia mediated.

57. A patient with compensated cirrhosis has clinically significant portal hypertension and small esophageal varices without prior bleeding. Which therapy can reduce the risk of decompensation and variceal bleeding?

- A.** A selective beta-1 blocker such as metoprolol because portal-pressure reduction requires isolated cardiac beta-1 blockade.
- B.** Long-term proton-pump inhibitor therapy because suppression of gastric acid prevents variceal rupture and hepatic decompensation.
- C.** A nonselective beta blocker such as carvedilol when there is no contraindication.
- D.** Prophylactic TIPS because all patients with small varices benefit from immediate portosystemic decompression before a first bleed.
- E.** Daily vitamin K because correction of INR is the main strategy for preventing spontaneous bleeding from esophageal varices.

58. A patient with cirrhosis presents with hematemesis and shock from suspected variceal hemorrhage. Which treatment bundle should begin promptly before definitive endoscopy?

- A.** Aggressive crystalloid transfusion to a hemoglobin above 12 g/dL before any vasoactive drug or endoscopic intervention is attempted.
- B.** High-dose corticosteroids and proton-pump inhibitor monotherapy because variceal bleeding is primarily caused by mucosal inflammation.

- C.** Immediate TIPS in every patient without first attempting endoscopic therapy or assessing the risk profile for early shunt placement.
- D.** Routine correction of INR to normal with plasma before endoscopy because endoscopic banding is unsafe until coagulation tests normalize.
- E.** Resuscitation plus vasoactive therapy and short-course antibiotic prophylaxis, followed by urgent endoscopic control.

59. A patient with cirrhosis and acute esophageal variceal bleeding is Child-Pugh class C but not beyond the range generally considered for shunt therapy. Bleeding is controlled endoscopically. Which strategy should be considered because of the high risk of early rebleeding and death?

- A.** Delay TIPS until a third recurrent bleed because successful initial band ligation eliminates the early mortality benefit of portal decompression.
- B.** Use aspirin after hemostasis because platelet inhibition reduces portal-vein pressure and prevents recurrent variceal rupture.
- C.** Stop all vasoactive therapy immediately and avoid further intervention because Child-Pugh C disease makes any shunt absolutely contraindicated.
- D.** Preemptive TIPS within the early post-bleed window in an appropriately selected high-risk patient.
- E.** Perform elective splenectomy because removing splenic inflow is superior to TIPS for preventing early recurrent esophageal variceal bleeding.

60. A patient with cirrhosis has tense ascites causing dyspnea. Diagnostic paracentesis is negative for infection. What is the most appropriate immediate treatment for symptom relief?

- A.** Escalate oral diuretics for several weeks before drainage because therapeutic paracentesis is contraindicated in uncomplicated tense ascites.
- B.** Large-volume paracentesis with albumin replacement when a substantial volume is removed.
- C.** Place a TIPS emergently in every patient with tense ascites because paracentesis worsens portal hypertension and accelerates renal failure.
- D.** Administer fresh frozen plasma until INR normalizes before paracentesis because bleeding risk is determined by the INR in cirrhosis.

E. Start broad-spectrum antibiotics for five days because all tense ascites should be presumed infected even when neutrophil count is low.

61. A patient with cirrhotic ascites has preserved kidney function and no severe hyponatremia. Which outpatient diuretic strategy is commonly used?

A. Aldosterone antagonism with spironolactone, often combined with a loop diuretic such as furosemide.

B. Loop diuretic monotherapy at very high dose because secondary hyperaldosteronism is not important in cirrhotic sodium retention.

C. Acetazolamide monotherapy because proximal tubular bicarbonate loss is the principal mechanism of sodium retention in portal hypertension.

D. Thiazide monotherapy because distal sodium blockade is safest and most effective in decompensated cirrhosis with ascites.

E. Chronic intravenous mannitol because osmotic diuresis prevents hepatorenal syndrome while mobilizing portal hypertensive ascites.

62. A patient has refractory ascites requiring weekly large-volume paracentesis despite sodium restriction and maximally tolerated diuretics. There is no severe pulmonary hypertension, heart failure, or recurrent encephalopathy. What should be considered?

A. Permanent tunneled peritoneal drainage as standard first-line therapy.

B. High-dose NSAID therapy to promote renal sodium excretion.

C. Routine surgical portacaval shunting before transplant evaluation.

D. TIPS while liver transplant candidacy is concurrently assessed.

E. Discontinue transplant evaluation despite refractory ascites.

63. Ascitic fluid from a patient with cirrhosis contains 420 polymorphonuclear leukocytes/mm³. The patient has mild abdominal discomfort but is hemodynamically stable. What is the diagnosis?

A. Sterile portal-hypertensive ascites because spontaneous bacterial peritonitis requires a positive ascitic fluid culture in addition to neutrophilia.

- B.** Secondary bacterial peritonitis because any neutrophil count above 250/mm³ proves a surgically treatable perforated viscus.
- C.** Hepatorenal syndrome because neutrophilic ascites is a nonspecific consequence of renal vasoconstriction in advanced cirrhosis.
- D.** Spontaneous bacterial peritonitis because an ascitic neutrophil count of at least 250/mm³ is diagnostic regardless of culture result.
- E.** Chylous ascites because neutrophils above 250/mm³ are the defining laboratory criterion for lymphatic leakage into the peritoneum.

64. A patient with tense ascites is ready for discharge after successful treatment of spontaneous bacterial peritonitis. Beyond management of the underlying cirrhosis, which discharge strategy most directly lowers the likelihood of another SBP episode?

- A.** Secondary antibiotic prophylaxis after recovery from SBP.
- B.** No preventive therapy after a successfully treated first episode.
- C.** Chronic corticosteroid therapy to suppress peritoneal inflammation.
- D.** Elective splenectomy to prevent recurrent bacterial translocation.
- E.** Daily proton-pump inhibitor therapy to prevent bacterial translocation.

65. A patient with decompensated cirrhosis develops acute kidney injury. Diuretics and nephrotoxins are stopped, volume status is addressed, urinalysis is bland, and renal imaging shows no obstruction. Creatinine continues to rise. Which diagnosis should be considered?

- A.** Acute glomerulonephritis, which is expected to have a bland urine sediment and no proteinuria or hematuria in cirrhosis.
- B.** Obstructive nephropathy, which remains the most likely cause despite normal renal imaging and absence of hydronephrosis.
- C.** Hepatorenal syndrome-acute kidney injury after exclusion of structural and reversible causes.
- D.** Chronic calcineurin-inhibitor nephrotoxicity, which is the usual cause of renal failure before any organ transplantation has occurred.
- E.** Acute cortical necrosis, which should be assumed whenever creatinine rises in a patient with portal hypertension and ascites.

66. A hospitalized patient with cirrhosis meets criteria for hepatorenal syndrome-AKI and has no contraindication to vasoconstrictor therapy. Which treatment is preferred?

- A.** High-dose loop diuretics with fluid restriction because forced diuresis restores renal perfusion in hepatorenal syndrome.
- B.** ACE-inhibitor therapy because reducing efferent arteriolar tone reverses the renal vasoconstriction of advanced cirrhosis.
- C.** NSAID therapy because renal prostaglandin blockade improves glomerular filtration in the low-effective-volume state of cirrhosis.
- D.** Terlipressin with albumin, with careful monitoring for ischemic and respiratory complications.
- E.** Long-term dialysis alone because vasoconstrictor therapy does not produce renal recovery and has no role before transplantation.

67. A cirrhotic patient has progressive dyspnea, platypnea, and oxygen saturation of 86% when standing. Contrast echocardiography shows delayed bubbles in the left atrium. What is the diagnosis?

- A.** Portopulmonary hypertension.
- B.** Acute pulmonary embolism.
- C.** Hepatic hydrothorax.
- D.** Cirrhotic cardiomyopathy.
- E.** Hepatopulmonary syndrome.

68. A patient with portal hypertension has exertional dyspnea and an echocardiogram estimating high pulmonary artery pressure. Which test is required to confirm portopulmonary hypertension and characterize transplant risk?

- A.** Contrast echocardiography alone because delayed bubbles establish elevated pulmonary vascular resistance and make catheterization unnecessary.
- B.** Pulmonary function testing alone because portopulmonary hypertension is defined by a restrictive ventilatory defect in cirrhosis.
- C.** Ventilation-perfusion scanning alone because chronic thromboembolic disease is required for the diagnosis of portopulmonary hypertension.

D. Arterial blood gas alone because the severity of hypoxemia directly determines pulmonary vascular resistance and transplant candidacy.

E. Right-heart catheterization to measure pulmonary pressures, wedge pressure, cardiac output, and pulmonary vascular resistance.

69. A liver transplant candidate has moderate-to-severe portopulmonary hypertension on right-heart catheterization. What is the best management principle?

A. Proceed directly to liver transplantation because removal of the cirrhotic liver immediately normalizes pulmonary vascular resistance during surgery.

B. Place TIPS because increasing portal-systemic shunting reliably reduces pulmonary artery pressure in portopulmonary hypertension.

C. Use supplemental oxygen alone because pulmonary vasodilator therapy is contraindicated in all patients with portal hypertension.

D. Treat the pulmonary vascular disease and reassess hemodynamics before transplantation because uncontrolled disease markedly increases perioperative risk.

E. Exclude the patient permanently from transplantation because pulmonary hemodynamics can never improve enough to alter candidacy.

70. A patient with recurrent hepatic encephalopathy is taking lactulose but still has frequent episodes despite two to three soft stools daily. What is the next best preventive therapy?

A. Add rifaximin to lactulose for prevention of recurrent overt hepatic encephalopathy.

B. Stop lactulose and begin chronic benzodiazepines because excessive bowel movements increase cerebral ammonia production.

C. Add a high-dose proton-pump inhibitor because gastric acid suppression reduces intestinal ammonia production more effectively than antibiotics.

D. Start chronic opioid therapy because mu-receptor activation stabilizes astrocyte function and prevents recurrent encephalopathy.

E. Perform prophylactic TIPS because additional portosystemic shunting improves hepatic clearance of ammonia from portal blood.

71. A patient with cirrhosis has a large right pleural effusion that recurs despite sodium restriction and diuretics. Pleural fluid is a transudate, and there is no primary cardiac or pulmonary cause. What is the most likely management pathway?

- A.** Insert a chronic chest tube as routine first-line therapy because continuous pleural drainage has low infection and protein-loss risk in cirrhosis.
- B.** Perform repeated pleurodesis before transplant evaluation because diaphragmatic defects are always permanently cured by chemical sclerosis.
- C.** Start high-dose corticosteroids because hepatic hydrothorax is caused by inflammatory pleuritis from portal-hypertensive cytokines.
- D.** Increase dietary sodium because expanding plasma volume reduces movement of ascites through diaphragmatic defects into the pleural space.
- E.** Treat hepatic hydrothorax, consider TIPS in selected patients, and assess for liver transplantation when the effusion is refractory.

72. A stable cirrhotic patient with INR 2.0 and platelets 62,000/mm³ is scheduled for diagnostic paracentesis. There is no disseminated intravascular coagulation or active bleeding. What is the best approach?

- A.** Transfuse plasma until INR is below 1.3 because the INR directly measures procedural bleeding risk in cirrhosis.
- B.** Proceed without routine correction of INR or platelets because conventional coagulation tests poorly predict bleeding from low-risk paracentesis.
- C.** Transfuse platelets to above 150,000/mm³ because any thrombocytopenia makes bedside paracentesis unsafe.
- D.** Cancel paracentesis and treat empirically for spontaneous bacterial peritonitis because coagulation abnormalities prohibit ascitic sampling.
- E.** Administer vitamin K and wait one week because portal-hypertensive coagulopathy always represents nutritional vitamin K deficiency.

73. A patient with refractory ascites has gradually fallen to a serum sodium of 121 mEq/L while remaining alert and without seizures. Which interpretation of this laboratory abnormality is most appropriate in advanced cirrhosis?

- A.** The sodium should be corrected to normal within six hours because rapid correction prevents hepatic encephalopathy without neurologic risk.
- B.** Hyponatremia is unrelated to portal-hypertensive circulatory dysfunction and has no prognostic or transplant significance in cirrhosis.
- C.** Routine hypertonic saline is indicated for all asymptomatic cirrhotic patients with sodium below 125 mEq/L regardless of volume status.
- D.** Severe dilutional hyponatremia reflects advanced circulatory dysfunction, worsens prognosis, and requires cautious correction rather than rapid normalization.
- E.** Stopping all dietary protein is the most effective treatment because amino-acid metabolism is the primary determinant of sodium concentration in cirrhosis.

74. A patient with cirrhosis has a 2.4-cm liver lesion showing nonrim arterial-phase hyperenhancement, washout, and enhancing capsule on high-quality multiphasic imaging. What is the best interpretation?

- A.** Biopsy is mandatory because imaging cannot establish hepatocellular carcinoma in a cirrhotic liver regardless of lesion size or enhancement pattern.
- B.** The lesion is most consistent with focal nodular hyperplasia because arterial enhancement plus washout is characteristic of benign hepatocellular lesions.
- C.** The lesion should be ignored until it exceeds 5 cm because smaller tumors have no transplant or oncologic significance in cirrhosis.
- D.** The imaging pattern is diagnostic of cholangiocarcinoma because HCC typically lacks arterial enhancement and delayed washout.
- E.** The lesion has imaging features diagnostic of hepatocellular carcinoma in an at-risk cirrhotic liver and may not require biopsy for diagnosis.

75. A transplant candidate has a solitary 4.2-cm HCC with no macrovascular invasion or extrahepatic disease. Which statement best describes transplant eligibility from an oncologic standpoint?

- A.** Any HCC larger than 3 cm is outside accepted transplant criteria and cannot be considered for liver transplantation at any center.
- B.** The tumor burden is within conventional Milan morphologic criteria and can be compatible with liver transplantation if other requirements are met.

C. A solitary HCC qualifies only when it is smaller than 2 cm because larger lesions have an unacceptably high recurrence risk after transplantation.

D. Transplant eligibility depends only on AFP level and not on tumor size, number, vascular invasion, or extrahepatic spread.

E. Macrovascular invasion is irrelevant to transplant oncology as long as the total tumor diameter remains below 5 cm.

76. A patient with HCC initially exceeds standard transplant tumor burden but meets an accepted downstaging entry framework. After locoregional therapy, imaging shows sustained viable tumor burden within transplant criteria and AFP falls substantially. What is the significance?

A. Successful durable downstaging can permit transplant consideration because response provides information about tumor biology as well as size.

B. Downstaging never changes transplant eligibility because only the tumor dimensions at first diagnosis can be used for any later decision.

C. Any fall in AFP automatically guarantees transplant candidacy even if macrovascular invasion and extrahepatic spread remain present.

D. Locoregional therapy is contraindicated before liver transplantation because treatment obscures tumor biology and increases post-transplant recurrence.

E. Transplantation should occur immediately after the first treatment session because a period of response assessment has no clinical value.

77. A patient with HCC has a marked AFP elevation that remains very high despite technically successful locoregional treatment. Why is this important before transplantation?

A. AFP has no prognostic value once the lesion has been treated because only the current largest tumor diameter predicts recurrence.

B. Persistently extreme AFP can signal aggressive tumor biology and a higher risk of post-transplant recurrence even when imaging burden appears controlled.

C. A high AFP proves cholangiocarcinoma and excludes hepatocellular carcinoma regardless of imaging or histology.

D. AFP elevation is caused only by portal hypertension, so it should fall after TIPS rather than after tumor-directed therapy.

E. AFP is useful only for diagnosing acute hepatitis and has no role in transplant oncology or response assessment.

78. A patient listed for liver transplantation with HCC is expected to wait many months. The tumor remains within transplant criteria and liver function allows intervention. What is the purpose of locoregional bridge therapy?

- A.** Replace transplantation permanently because any tumor responding to locoregional therapy no longer benefits from removal of the cirrhotic liver.
- B.** Increase AFP to improve allocation priority because treatment-related inflammation is used as a favorable tumor-biology marker.
- C.** Create macrovascular invasion that facilitates complete surgical removal of the portal vein at the time of transplantation.
- D.** Eliminate the need for surveillance imaging because a single technically successful treatment proves complete pathologic tumor eradication.
- E.** Reduce the risk of tumor progression beyond transplant criteria while the patient remains on the waiting list.

79. A patient with unresectable perihilar cholangiocarcinoma has no metastatic disease and meets a center's strict transplant-oncology protocol. Which strategy can be appropriate?

- A.** Immediate transplantation without staging or neoadjuvant therapy because perihilar cholangiocarcinoma has minimal recurrence risk in an untreated graft recipient.
- B.** Routine transperitoneal tumor biopsy before referral because needle-track seeding does not affect transplant eligibility in perihilar disease.
- C.** Protocol-based neoadjuvant therapy followed by liver transplantation in carefully selected patients.
- D.** TIPS followed by observation because portal decompression is the definitive oncologic treatment for hilar cholangiocarcinoma.
- E.** Permanent exclusion from transplantation because cholangiocarcinoma is never treated with liver transplantation under any circumstance.

80. A child with hepatoblastoma has multifocal disease involving all four liver sectors but no unresectable extrahepatic metastases after systemic therapy. Which surgical strategy should be considered?

- A.** Liver transplantation when complete conventional hepatic resection is not feasible and oncologic criteria are satisfied.
- B.** TIPS followed by chemotherapy because portal decompression converts diffuse hepatoblastoma into a resectable localized tumor.
- C.** Observation after chemotherapy because multifocal hepatoblastoma has no risk of progression once AFP begins to decline.
- D.** Radiofrequency ablation alone because complete ablation of multifocal pediatric liver tumors is superior to transplantation.
- E.** Whipple resection because hepatoblastoma originates from the extrahepatic biliary epithelium rather than hepatoblasts.

81. A 35-year-old man with no cirrhosis has an 8-cm hepatic adenoma. Which management is most appropriate?

- A.** Observation with imaging every five years because hepatic adenomas in men have lower malignant potential than those in women.
- B.** TIPS placement because reducing portal pressure causes hepatocellular adenomas to regress even in patients without portal hypertension.
- C.** Liver transplantation as the routine first treatment for any solitary adenoma larger than 5 cm in an otherwise healthy liver.
- D.** Systemic corticosteroids because most hepatic adenomas are inflammatory lesions that regress with immunosuppression.
- E.** Surgical resection because adenomas in men carry meaningful malignant-transformation risk regardless of symptoms.

82. A healthy 27-year-old woman has a 4-cm liver lesion with homogeneous arterial enhancement and a central scar, and hepatobiliary-phase imaging favors focal nodular hyperplasia. She has no symptoms. What is the best management?

- A.** Immediate liver transplantation because focal nodular hyperplasia has a high rate of malignant transformation once larger than 3 cm.

- B.** Reassurance without intervention when imaging is characteristic and the patient remains asymptomatic.
- C.** Percutaneous biopsy every year because imaging cannot distinguish focal nodular hyperplasia from HCC in a noncirrhotic liver.
- D.** TACE because all hypervascular liver lesions require arterial treatment to prevent rupture and metastatic progression.
- E.** Stop all estrogen exposure and resect the lesion because focal nodular hyperplasia behaves biologically like a hepatic adenoma.

83. A 62-year-old has a 6-cm hepatic lesion with classic peripheral discontinuous nodular enhancement and progressive centripetal fill-in. He is asymptomatic. What is the best next step?

- A.** Perform urgent needle biopsy because vascular tumors cannot be diagnosed by imaging and histology is required before observation.
- B.** Treat with anticoagulation because a hemangioma represents chronic organized hepatic-vein thrombosis rather than a vascular malformation.
- C.** List for transplantation because hemangiomas larger than 5 cm inevitably rupture and undergo malignant transformation.
- D.** Begin systemic chemotherapy because the enhancement pattern is diagnostic of hepatic angiosarcoma rather than hemangioma.
- E.** Recognize a typical hemangioma and avoid percutaneous biopsy when imaging is diagnostic.

84. A patient has multifocal hepatic epithelioid hemangioendothelioma that cannot be completely resected but remains confined to the liver without aggressive extrahepatic disease. Which option may be appropriate?

- A.** Transplantation is absolutely contraindicated because every vascular liver tumor behaves like angiosarcoma after immunosuppression.
- B.** TIPS is definitive therapy because reducing portal pressure causes epithelioid hemangioendothelioma to involute spontaneously.
- C.** No therapy is indicated because multifocality proves the tumor is benign and incapable of progression or metastasis.

D. Routine Whipple resection is preferred because the tumor arises from distal bile-duct epithelium and spreads secondarily to the liver.

E. Liver transplantation in a carefully selected patient because this rare vascular tumor can have favorable transplant outcomes.

85. A patient with well-differentiated neuroendocrine tumor has liver-dominant metastatic disease that is stable on systemic therapy, with the primary tumor completely controlled and no extrahepatic spread. Which statement is most accurate?

A. Liver transplantation is standard first-line therapy for all neuroendocrine liver metastases regardless of primary control or extrahepatic spread.

B. Liver transplantation is an uncommon but possible option in highly selected patients with favorable tumor biology and liver-limited disease.

C. Any metastatic malignancy has identical biology after transplantation, so tumor grade and treatment stability are irrelevant to selection.

D. TIPS is preferred because portal decompression directly reduces neuroendocrine tumor hormone secretion and metastatic growth.

E. Transplantation can be considered only when the primary neuroendocrine tumor remains untreated so that immunosuppression can shrink both sites.

86. A patient with decompensated cirrhosis is referred for transplant evaluation. Which group of laboratory values contributes directly to MELD 3.0 calculation?

A. Bilirubin, INR, creatinine, sodium, and albumin, with additional demographic adjustment incorporated into the model.

B. AST, ALT, alkaline phosphatase, platelet count, and ammonia because these directly measure hepatocyte necrosis and portal hypertension severity.

C. AFP, albumin, lactate, sodium, and ferritin because tumor biology and systemic inflammation are the main determinants of MELD 3.0.

D. Bilirubin, ammonia, platelet count, variceal size, and ascites volume because clinical portal-hypertensive findings replace renal function in MELD 3.0.

E. INR, factor VIII, fibrinogen, D-dimer, and thromboelastography because coagulation balance is the principal focus of modern MELD scoring.

87. Two transplant candidates have similar MELD scores, but one has severe sarcopenia, slow gait speed, repeated falls, and dependence in activities of daily living. Why is this important?

- A.** Frailty has no independent prognostic value because all mortality risk in cirrhosis is fully captured by bilirubin, INR, creatinine, and sodium.
- B.** Severe frailty is automatically a permanent contraindication to transplantation and should not be reassessed after rehabilitation or nutrition therapy.
- C.** Frailty provides prognostic information beyond MELD and can identify a potentially modifiable risk that should be addressed during candidacy assessment.
- D.** Frailty is relevant only after transplantation because pretransplant physical performance does not influence wait-list or perioperative outcomes.
- E.** Sarcopenia increases MELD directly by elevating serum creatinine, so no separate functional assessment is needed in transplant candidates.

88. A listed patient is called in for a liver offer but is currently receiving vasopressors for uncontrolled gram-negative bloodstream infection. How should the transplant team approach the immediate organ offer?

- A.** Proceed immediately because immunosuppression after transplantation reliably clears bacteremia by reducing the inflammatory response.
- B.** Ignore the infection if MELD is above 30 because allocation priority supersedes all reversible medical contraindications.
- C.** Treat and control the infection before proceeding because uncontrolled sepsis creates prohibitive perioperative risk.
- D.** Use TIPS as definitive infection treatment because portal decompression removes the source of bloodstream infection in cirrhosis.
- E.** Permanently exclude the patient because any episode of bacteremia is a lifelong absolute contraindication to liver transplantation.

89. A patient with well-controlled HIV infection, undetectable viral load, and preserved CD4 count has decompensated cirrhosis. Which statement is most accurate?

- A.** Controlled HIV infection is not by itself a contraindication to liver transplantation, although drug interactions and opportunistic-infection risk require careful planning.
- B.** Any HIV infection permanently excludes liver transplantation because immunosuppression inevitably causes uncontrolled viral replication and graft loss.
- C.** HIV infection automatically requires simultaneous kidney transplantation even when kidney function is normal before transplant.
- D.** Antiretroviral therapy must be permanently stopped after liver transplantation because all regimens are incompatible with calcineurin inhibitors.
- E.** Only living-donor transplantation can be performed in HIV-positive recipients because deceased-donor organs are prohibited by transplant policy.

90. A cirrhotic patient has sustained advanced chronic kidney disease and meets accepted criteria for simultaneous liver-kidney transplantation. What is the key rationale for the combined approach?

- A.** The renal dysfunction is sufficiently severe and unlikely to recover reliably with liver transplantation alone.
- B.** Every patient with any creatinine elevation should receive a kidney graft because post-transplant calcineurin inhibitors always cause end-stage kidney disease.
- C.** Simultaneous liver-kidney transplantation is based only on liver disease etiology and not on the duration or severity of renal dysfunction.
- D.** A kidney graft is required whenever ascites is present because diuretic use makes renal function impossible to assess before transplantation.
- E.** Combined transplantation is used only to improve the liver MELD score and does not depend on an independent renal indication.

91. A potential living liver donor is medically healthy but states that family members are threatening to cut off financial support if donation is refused. What is the most appropriate response?

- A.** Proceed because family pressure is acceptable when the recipient's mortality risk is high and the donor has no medical contraindication.
- B.** Allow the recipient's transplant surgeon to counsel the donor alone because an independent donor advocate creates unnecessary conflict of interest.

- C.** Exclude the recipient from all forms of transplantation because evidence of donor coercion is automatically misconduct by the recipient.
- D.** Ask the donor to sign an additional surgical consent form because written consent eliminates concerns about undue influence or coercion.
- E.** Pause the donation process and ensure an independent donor evaluation focused on voluntariness and freedom from coercion.

92. A transplant candidate has a history of poor medication adherence related to unstable housing and inability to afford prescriptions. Which approach best reflects a fair candidacy assessment?

- A.** Deny candidacy automatically because any prior nonadherence proves an immutable personal trait and predicts graft loss regardless of circumstances.
- B.** Identify modifiable barriers and build a realistic support plan rather than treating socioeconomic disadvantage itself as a categorical exclusion.
- C.** Ignore adherence history completely because social and behavioral factors should never influence transplant outcomes or post-transplant planning.
- D.** Require the candidate to demonstrate a fixed number of years of perfect adherence even if housing and medication access are now stabilized.
- E.** Transfer the decision entirely to the insurance company because financial coverage is the only ethically relevant determinant of post-transplant adherence.

93. A patient presents 18 hours after a large acetaminophen ingestion with aminotransferases rapidly rising and INR 2.1. The serum acetaminophen level is now low. What treatment should be given?

- A.** Withhold N-acetylcysteine because treatment is useful only when the acetaminophen concentration remains above the treatment nomogram line.
- B.** Start intravenous N-acetylcysteine despite the low measured drug level because hepatic injury is already evolving.
- C.** Give activated charcoal as the only therapy because gastrointestinal decontamination remains effective long after hepatic injury has begun.
- D.** Start high-dose corticosteroids because acetaminophen acute liver failure is mediated primarily by autoimmune hepatocyte destruction.

E. Wait for encephalopathy before any antidotal therapy because coagulopathy alone does not represent clinically important toxicity.

94. A patient with acetaminophen acute liver failure develops severe lactic acidosis, worsening coagulopathy, renal failure, and high-grade encephalopathy despite resuscitation. What is the key next step?

A. Transfer out of intensive care because aminotransferases often fall late in acetaminophen injury and this reliably indicates hepatic recovery.

B. Delay transplant evaluation until bilirubin exceeds 30 mg/dL because bilirubin is the only validated prognostic marker in acetaminophen toxicity.

C. Urgent transplant-center assessment using validated prognostic criteria because the probability of spontaneous recovery may be low.

D. Perform elective TIPS because portal decompression is the definitive treatment for cerebral edema in acute liver failure.

E. Begin chronic dialysis as definitive therapy because renal replacement removes the hepatic necrosis that drives multiorgan failure.

95. A patient with non-acetaminophen acute liver failure has early encephalopathy and no contraindication to therapy. Which treatment has potential benefit even when acetaminophen toxicity is not proven?

A. Intravenous N-acetylcysteine can be considered in early-stage non-acetaminophen acute liver failure while etiology-specific treatment proceeds.

B. Oral lactulose is the only therapy shown to restore hepatic synthetic function in non-acetaminophen acute liver failure.

C. Routine prophylactic plasma transfusion improves survival by correcting INR and should be used before any transplant assessment.

D. TIPS is indicated because all acute liver failure is driven by portal hypertension that requires immediate decompression.

E. Chronic interferon therapy should be started empirically because occult viral hepatitis causes nearly all non-acetaminophen acute liver failure.

96. A patient develops acute liver failure after eating foraged wild mushrooms. Severe vomiting occurred several hours after ingestion, followed by a brief improvement and then massive hepatocellular injury. Which toxin is most likely?

- A.** Muscarine toxicity from *Inocybe* species.
- B.** Amatoxin poisoning from *Amanita* species.
- C.** Gyromitrin toxicity from false morels.
- D.** Psilocybin toxicity from hallucinogenic mushrooms.
- E.** Orellanine toxicity from *Cortinarius* species.

97. A pregnant patient at 35 weeks develops nausea, abdominal pain, hypoglycemia, jaundice, coagulopathy, and encephalopathy. Acute fatty liver of pregnancy is suspected. What is the most important immediate treatment?

- A.** Prompt delivery with intensive maternal supportive care and transplant-center involvement if liver failure does not reverse.
- B.** Continue the pregnancy until fetal lung maturity because delivery worsens the maternal mitochondrial fatty-acid oxidation defect.
- C.** Start ursodeoxycholic acid and observe because acute fatty liver of pregnancy is a benign cholestatic disorder without maternal mortality.
- D.** Perform TIPS before delivery because portal decompression is required to correct the coagulopathy and hypoglycemia.
- E.** Use high-dose corticosteroids as definitive therapy because acute fatty liver of pregnancy is primarily an autoimmune hepatitis of late gestation.

98. A patient with acute liver failure has fever, splenomegaly, cytopenias, ferritin 40,000 ng/mL, and evidence of hemophagocytosis. Which systemic syndrome should be considered?

- A.** Primary biliary cholangitis, which commonly presents with pancytopenia, extreme hyperferritinemia, and acute liver failure in young adults.
- B.** Hereditary hemochromatosis, because any ferritin above 10,000 ng/mL proves chronic iron overload as the cause of fulminant hepatic failure.

- C.** Hepatorenal syndrome, which produces cytopenias and fever through renal vasoconstriction and macrophage activation.
- D.** Gilbert syndrome, which causes isolated unconjugated hyperbilirubinemia and is a common cause of hyperinflammatory acute liver failure.
- E.** Hemophagocytic lymphohistiocytosis causing severe immune-mediated hepatic injury.

99. A patient with profound cardiogenic shock develops AST and ALT above 8000 U/L, high lactate, and modest bilirubin elevation. Hepatic vasculature is patent. What is the most likely cause?

- A.** Acute viral hepatitis from hepatitis C.
- B.** Hypoxic or ischemic hepatitis from systemic hypoperfusion.
- C.** Primary sclerosing cholangitis with acute necrosis.
- D.** Wilson disease causing acute hepatic necrosis.
- E.** Budd-Chiari syndrome with hepatic venous obstruction.

100. A previously healthy adult develops indeterminate acute liver failure with rapidly worsening encephalopathy and cerebral edema despite intensive supportive care. What is the most important definitive option?

- A.** Elective outpatient observation because an unknown etiology predicts a high chance of spontaneous recovery once aminotransferases begin to fall.
- B.** TIPS because cerebral edema in acute liver failure is caused by portal hypertension and improves after portal decompression.
- C.** Long-term corticosteroids in all cases because indeterminate acute liver failure should be assumed to be autoimmune until proven otherwise.
- D.** Urgent liver transplantation if prognostic assessment indicates poor likelihood of spontaneous recovery.
- E.** Permanent mechanical ventilation without transplant evaluation because cerebral edema makes transplantation contraindicated once encephalopathy is severe.

101. A 19-year-old with acute liver failure has Coombs-negative hemolysis, low alkaline phosphatase relative to bilirubin, and acute kidney injury. Which diagnosis should be urgently evaluated?

- A.** Primary biliary cholangitis causing acute liver failure.
- B.** Wilson disease presenting as acute liver failure.
- C.** Hereditary hemochromatosis causing acute liver failure.
- D.** Autoimmune hepatitis causing acute liver failure.
- E.** Biliary atresia presenting as acute liver failure.

102. A patient with acute liver failure from autoimmune hepatitis develops worsening INR and encephalopathy despite an initial corticosteroid trial. What should happen next?

- A.** Do not prolong ineffective medical therapy; proceed with urgent transplant decision-making because continued deterioration predicts poor spontaneous recovery.
- B.** Increase corticosteroids indefinitely because autoimmune etiology guarantees eventual response even after encephalopathy and multiorgan failure develop.
- C.** Delay transplantation until six months of immunosuppression have been completed because acute liver failure cannot be listed during initial treatment.
- D.** Perform TIPS because the main cause of worsening INR in autoimmune acute liver failure is untreated portal hypertension.
- E.** Switch to ursodeoxycholic acid monotherapy because failure of steroids indicates that the disease has converted to primary biliary cholangitis.

103. A patient with severe alcohol-associated hepatitis receives corticosteroids after infection and gastrointestinal bleeding are excluded. Which tool is used during the first treatment week to assess response and decide whether steroids should continue?

- A.** The Milan criteria, which estimate transplant benefit from alcohol-associated hepatitis using tumor size and number.
- B.** The PELD score, which was designed to assess corticosteroid response in adult alcohol-associated hepatitis.
- C.** The Child-Pugh score alone, because it directly measures steroid responsiveness after seven days of treatment.
- D.** The Maddrey discriminant function repeated hourly, because short-term changes in prothrombin time determine whether steroids are effective.

E. The Lille score, which incorporates the early bilirubin response and other clinical variables.

104. A patient with first-episode severe alcohol-associated hepatitis has a very high predicted short-term mortality and is a steroid nonresponder. He has strong family support, insight into alcohol use disorder, and no prior rehabilitation failures. Which statement is most accurate?

A. Early liver transplantation may be considered at experienced centers after rigorous psychosocial and medical selection rather than requiring a universal fixed abstinence interval.

B. Transplantation is prohibited until exactly six months of abstinence have elapsed because this duration is a universal national allocation rule.

C. Steroid nonresponse makes transplantation futile because the same inflammatory process invariably recurs immediately in the donor liver.

D. Living-donor transplantation is the only ethical option because deceased-donor organs cannot be allocated to alcohol-associated liver disease.

E. Transplant evaluation should be deferred until complete resolution of jaundice because severe alcohol-associated hepatitis is never an urgent indication.

105. A patient with severe alcohol-associated hepatitis has marked protein-calorie malnutrition but no refractory encephalopathy. What nutritional strategy is most appropriate?

A. Provide adequate calories and protein, using enteral supplementation when oral intake is insufficient.

B. Restrict protein to less than 0.5 g/kg/day because protein feeding worsens hepatic encephalopathy and increases mortality in alcohol-associated hepatitis.

C. Use total parenteral nutrition routinely because the gastrointestinal tract should be rested during all episodes of severe alcohol-associated hepatitis.

D. Provide calories primarily as alcohol-containing supplements because ethanol withdrawal accelerates hepatic catabolism and muscle loss.

E. Delay nutrition until bilirubin begins to fall because early feeding increases portal pressure and the risk of variceal hemorrhage.

106. During transplant evaluation for alcohol-associated cirrhosis, which factor is more clinically useful than relying on a fixed abstinence duration alone?

- A.** A single blood alcohol concentration measured at the first clinic visit because it predicts all future post-transplant drinking behavior.
- B.** The severity of portal hypertension alone because alcohol relapse risk has no relationship to psychosocial or addiction-related variables.
- C.** The candidate's socioeconomic status alone because income is the strongest validated predictor of post-transplant alcohol recurrence.
- D.** A structured assessment of relapse risk, treatment engagement, psychiatric comorbidity, insight, and recovery supports.
- E.** A universal six-month abstinence threshold because relapse prediction does not improve with multidisciplinary psychosocial assessment.

107. A liver transplant recipient with prior alcohol-associated liver disease reports recurrent heavy drinking. What is the best management approach?

- A.** Immediately withdraw all immunosuppression because alcohol relapse is best managed by allowing rejection to remove the graft.
- B.** Permanently discharge the patient from transplant follow-up because relapse proves that further medical treatment has no benefit.
- C.** Use only punitive monitoring because pharmacologic and behavioral treatment for alcohol use disorder is ineffective after transplantation.
- D.** Treat alcohol use disorder as a chronic relapsing disease with prompt addiction care while evaluating graft injury and medication adherence.
- E.** Ignore the drinking if liver tests are normal because alcohol exposure cannot injure a transplanted liver until cirrhosis has developed.

108. A patient has obesity, diabetes, and ongoing heavy alcohol use with steatohepatitis. Which concept best reflects current understanding of disease risk?

- A.** Metabolic dysfunction and alcohol exposure can act together to accelerate steatotic liver disease progression rather than representing mutually exclusive categories.
- B.** Metabolic risk eliminates the effect of alcohol, so only obesity and diabetes should be considered when estimating fibrosis progression.

- C.** Alcohol exposure protects against metabolic steatohepatitis by increasing hepatic fatty-acid oxidation in patients with diabetes.
- D.** The presence of alcohol use excludes any contribution from insulin resistance or obesity to steatohepatitis and fibrosis.
- E.** Steatotic liver disease progresses only when one isolated cause is present, because combined metabolic and alcohol exposure has no additive effect.

109. A patient with MASLD asks which histologic feature most strongly predicts long-term liver-related outcomes. Which is most important?

- A.** Percentage of macrovesicular hepatic steatosis.
- B.** Stage of hepatic fibrosis, especially advanced fibrosis.
- C.** Single-visit serum alanine aminotransferase level.
- D.** Presence and severity of obesity alone.
- E.** Absolute liver size measured by ultrasonography.

110. A 54-year-old with obesity and type 2 diabetes has mildly elevated ALT and a high FIB-4 score. What is the best next step for fibrosis risk stratification?

- A.** Reassure the patient because aminotransferases are only mildly elevated and advanced fibrosis cannot occur without marked enzyme abnormalities.
- B.** Perform ERCP because fibrosis in MASLD is caused by occult large-duct obstruction that is not visible on routine ultrasound.
- C.** Start transplant evaluation immediately because a high FIB-4 alone is equivalent to decompensated cirrhosis and proves transplant need.
- D.** Use a second-line noninvasive fibrosis assessment such as elastography and refer for specialist evaluation when advanced fibrosis is suspected.
- E.** Repeat ALT yearly without additional testing because noninvasive fibrosis scores are useful only after cirrhosis is already clinically apparent.

111. A patient with MASH cirrhosis is being evaluated for liver transplantation. Which extrahepatic assessment is especially important?

- A.** No cardiac assessment is needed because cirrhotic vasodilation protects patients with MASH from atherosclerotic cardiovascular disease.
- B.** Only pulmonary testing is required because cardiovascular risk after transplantation is unrelated to diabetes, obesity, or dyslipidemia.
- C.** Comprehensive cardiovascular risk evaluation because metabolic syndrome and occult coronary disease can drive perioperative morbidity and mortality.
- D.** Elective coronary angiography is mandatory in every candidate regardless of age, symptoms, renal function, or noninvasive risk assessment.
- E.** Cardiovascular disease becomes irrelevant after transplantation because immunosuppression reverses insulin resistance and dyslipidemia.

112. A patient with compensated MASH cirrhosis has obesity and poorly controlled type 2 diabetes. Which management principle is most appropriate?

- A.** Avoid weight loss because any caloric reduction accelerates sarcopenia and hepatic decompensation in all patients with compensated cirrhosis.
- B.** Treat only the liver disease because diabetes and obesity no longer influence outcomes once cirrhosis has developed.
- C.** Use an integrated metabolic strategy emphasizing sustained weight loss, diabetes control, cardiovascular risk reduction, and cirrhosis-specific surveillance.
- D.** Use high-dose corticosteroids because MASH is primarily an autoimmune inflammatory hepatitis that improves with immunosuppression.
- E.** Stop HCC surveillance if diabetes improves because metabolic control eliminates the carcinogenic risk of established cirrhosis.

113. A patient with MASH cirrhosis has a normal ALT. Which statement is correct?

- A.** Normal aminotransferases do not exclude advanced fibrosis, cirrhosis, or clinically important MASH-related risk.
- B.** Normal ALT excludes steatohepatitis-related advanced fibrosis and allows all cirrhosis surveillance to be discontinued.
- C.** ALT becomes normal only after complete histologic reversal of fibrosis, so the patient no longer has meaningful liver disease.

D. Normal ALT proves the cirrhosis is caused by alcohol rather than metabolic dysfunction and should be reclassified automatically.

E. A liver biopsy showing cirrhosis should be disregarded when ALT is normal because enzyme levels are more specific than histology.

114. A patient listed for transplant with MASH has severe sarcopenic obesity. Which pretransplant goal is most appropriate?

A. Use a very-low-calorie diet until body mass index is normal because muscle loss does not influence transplant outcomes in obesity.

B. Avoid exercise because resistance training increases portal pressure and is contraindicated in all patients with decompensated cirrhosis.

C. Use chronic fasting to reduce graft-size requirements because prolonged fasting improves hepatic synthetic function before transplantation.

D. Improve functional capacity and body composition through individualized nutrition and resistance-focused activity rather than pursuing rapid weight loss alone.

E. Delay all nutrition intervention until after transplantation because sarcopenia cannot improve while portal hypertension is present.

115. A patient undergoes liver transplantation for MASH cirrhosis and later develops obesity, diabetes, and dyslipidemia. Which graft complication should remain on the long-term radar?

A. Recurrent Wilson disease in the liver allograft.

B. Post-transplant sclerosing cholangitis from obesity.

C. Recurrent or de novo steatotic liver disease in the allograft.

D. Post-transplant Budd-Chiari syndrome from insulin resistance.

E. Acquired bilirubin-conjugation deficiency in the allograft.

116. A transplant candidate with obesity and compensated cirrhosis asks about bariatric surgery. Which principle is most appropriate?

A. Bariatric surgery is always safe before liver transplantation because obesity-related procedures do not interact with portal hypertension or cirrhosis.

- B.** Timing and procedure choice require individualized multidisciplinary assessment because portal hypertension and decompensation materially affect surgical risk.
- C.** All bariatric procedures are absolutely contraindicated for life in any patient with a history of cirrhosis, even after successful transplantation.
- D.** TIPS should routinely precede bariatric surgery because portal decompression eliminates all bleeding and decompensation risk from the operation.
- E.** Only body mass index matters when choosing timing; liver disease severity and transplant trajectory do not affect bariatric surgical planning.

117. A patient with MASH and type 2 diabetes is prescribed a medication that promotes meaningful weight loss and improves glycemic control. Which broader treatment goal remains essential even if liver enzymes improve?

- A.** Stop all fibrosis assessment because improved glycemia proves that cirrhosis, if present, has regressed completely.
- B.** Continue management of cardiovascular risk and fibrosis surveillance because metabolic improvement does not instantly reverse established advanced liver disease.
- C.** Discontinue statin therapy because lipid lowering becomes unsafe once a weight-loss medication is used in chronic liver disease.
- D.** Avoid resistance exercise because pharmacologic weight loss provides complete protection against sarcopenia and frailty.
- E.** End HCC surveillance immediately because improved liver enzymes eliminate cancer risk in a patient with prior cirrhosis.

118. A woman at 34 weeks has hypertension, right-upper-quadrant pain, platelets 62,000/mm³, elevated AST, and evidence of hemolysis. What is the definitive management for HELLP syndrome?

- A.** Stabilize the mother and proceed with delivery, with timing guided by maternal and fetal status.
- B.** Treat with ursodeoxycholic acid and continue pregnancy to term because HELLP is a cholestatic disorder that resolves without delivery.
- C.** Perform TIPS before delivery because portal decompression reverses thrombocytopenia and hemolysis in pregnancy-associated liver disease.

D. Begin long-term azathioprine because HELLP is an autoimmune hepatitis variant triggered by pregnancy and requires maintenance immunosuppression.

E. Delay delivery until platelet count normalizes spontaneously because delivery during thrombocytopenia worsens hepatic injury and maternal mortality.

119. A woman in the third trimester has intense pruritus of the palms and soles, elevated serum bile acids, and mild cholestatic liver-test abnormalities without synthetic failure. What is the most likely diagnosis?

A. Acute fatty liver of pregnancy.

B. HELLP syndrome.

C. Intrahepatic cholestasis of pregnancy.

D. Budd-Chiari syndrome during pregnancy.

E. Primary sclerosing cholangitis.

120. A patient taking a new medication develops hepatocellular injury with ALT 12 times normal and total bilirubin 5 mg/dL, with no biliary obstruction or alternative explanation. Why is this pattern concerning?

A. Jaundice makes drug-induced injury unlikely because clinically important DILI causes isolated aminotransferase elevation without bilirubin increase.

B. The pattern proves chronic extrahepatic obstruction because any bilirubin rise with ALT elevation is diagnostic of common bile duct stones.

C. Hepatocellular drug injury accompanied by jaundice can signal a higher risk of severe or fatal DILI and requires close monitoring.

D. This is a benign adaptation response that should be ignored as long as the patient has no pruritus or abdominal pain.

E. The medication should be continued at double dose because worsening bilirubin reflects undertreatment of the underlying disease rather than hepatic toxicity.

121. A patient develops worsening coagulopathy and encephalopathy from suspected idiosyncratic drug-induced liver injury despite withdrawal of the offending agent. What is the most important next step?

- A.** Restart the suspected medication at a lower dose because rechallenge is the fastest way to confirm DILI before considering transplantation.
- B.** Urgent transfer to a liver transplant center for acute liver failure management and assessment of transplant need.
- C.** Perform elective TIPS because drug-induced acute liver failure is caused by severe portal hypertension rather than hepatocyte loss.
- D.** Wait for a liver biopsy to prove the drug cause before transfer because transplant evaluation should not begin without histologic confirmation.
- E.** Use chronic broad-spectrum antibiotics alone because idiosyncratic DILI is usually caused by occult bacterial infection of the biliary tree.

122. A patient with cirrhosis develops bacterial sepsis followed by renal failure, encephalopathy, circulatory shock, and respiratory failure. Which feature most strongly drives short-term prognosis in acute-on-chronic liver failure?

- A.** The peak ALT alone, because aminotransferase height directly determines mortality regardless of renal, respiratory, or circulatory failure.
- B.** The presence of ascites alone, because organ failures outside the liver do not materially change short-term mortality in ACLF.
- C.** The number and severity of extrahepatic organ failures rather than the liver-test abnormalities alone.
- D.** The serum AFP level, because tumor-marker elevation is the main determinant of recovery from infection-triggered ACLF.
- E.** The duration of cirrhosis in years, because chronicity predicts outcome more strongly than the current organ-failure burden.

123. A previously stable cirrhotic patient develops infection-triggered ACLF with multiple organ failures but no irreversible contraindication to transplantation. Which management principle is most appropriate?

- A.** Treat the precipitating event aggressively and initiate early transplant evaluation because the window for successful transplantation may be short.
- B.** Delay transplant referral until all organ failures have resolved because transplantation cannot be considered during acute-on-chronic liver failure.

- C.** Use TIPS routinely during shock because portal decompression reverses systemic inflammation and multiorgan failure in ACLF.
- D.** Stop antibiotics once liver failure is recognized because infection-directed therapy no longer influences outcome after ACLF has developed.
- E.** Provide palliative care only because multiple organ failures make meaningful recovery or transplantation impossible in every case.

124. A patient with decompensated cirrhosis is listed for transplantation but has severe symptom burden, repeated hospitalizations, and anxiety about dying before an organ becomes available. What is the best role for palliative care?

- A.** Wait until the patient is removed from the transplant list because palliative care is incompatible with active life-prolonging therapy.
- B.** Provide concurrent symptom management, coping support, and goals-of-care discussions while transplant-directed treatment continues.
- C.** Replace hepatology care entirely because involvement of palliative specialists means the patient has chosen hospice and stopped transplantation.
- D.** Avoid discussing prognosis because realistic goals-of-care conversations reduce adherence and make transplant candidates lose hope.
- E.** Use palliative care only for cancer pain because nonmalignant end-stage liver disease is outside the scope of specialty palliative services.

125. A patient with end-stage cirrhosis who is not a transplant candidate has refractory dyspnea, weakness, and recurrent hospitalizations. He wants to avoid future intensive care. What is the most appropriate next step?

- A.** Continue full escalation by default because advance care planning is inappropriate in nonmalignant liver disease until cardiac arrest occurs.
- B.** Avoid hospice because cirrhosis has an unpredictable course and therefore cannot be managed with comfort-focused services under any circumstances.
- C.** Clarify goals, document treatment preferences, and discuss hospice when the patient's priorities and prognosis are consistent with that level of care.
- D.** Create a TIPS solely to satisfy the wish to avoid hospitalization even if the patient has severe contraindications to the procedure.

E. Stop all symptom medication because comfort-oriented treatment can mask deterioration and interfere with prognostic assessment.

126. A patient with advanced cirrhosis has painful muscle cramps, insomnia, and severe pruritus that impair quality of life despite stable laboratory values. What is the best general principle?

A. Avoid symptom-directed medication because only interventions that lower MELD are appropriate before transplantation.

B. Treat symptom burden directly because quality of life is clinically important even when MELD does not change.

C. Assume the symptoms are psychiatric because clinically important cirrhosis symptoms always correlate with worsening bilirubin and INR.

D. Use sedating opioids and benzodiazepines as first-line treatment for all symptoms because encephalopathy risk is not relevant in stable cirrhosis.

E. Remove the patient from transplant consideration because symptom burden despite stable MELD proves irreversible frailty and poor adherence.

127. A transplant candidate has repeatedly stated that if catastrophic neurologic injury leaves no meaningful chance of recovery, she would not want prolonged life support. Which document or discussion most directly helps preserve this preference if she later loses decision-making capacity?

A. A transplant listing agreement, because listing automatically defines the patient's preferences for all future intensive-care decisions.

B. A MELD exception request, because allocation documents function as legally binding instructions about mechanical ventilation and resuscitation.

C. A donor-recipient consent form, because surgical consent governs all later decisions about life support after transplantation.

D. An insurance authorization, because payer approval identifies the appropriate surrogate when a transplant candidate loses capacity.

E. Advance care planning with designation of a surrogate and clear documentation of the patient's values and treatment preferences.

128. A cirrhotic patient is completing evaluation for liver transplantation. Which vaccination strategy is preferred?

- A.** Give indicated age- and risk-based vaccines before transplantation whenever feasible because immune responses are generally stronger before immunosuppression.
- B.** Delay all vaccines until the first postoperative month because immunosuppression improves vaccine immunogenicity and eliminates adverse reactions.
- C.** Avoid inactivated vaccines because chronic liver disease converts them into live vaccines with a risk of disseminated infection.
- D.** Give only vaccines required for international travel because routine adult immunizations have no role in transplant candidates.
- E.** Use long-term prophylactic antibiotics instead of vaccination because vaccine responses are absent in all patients with cirrhosis.

129. A patient with cirrhosis is found to have osteoporosis on DEXA during transplant evaluation. What is the best approach?

- A.** Ignore osteoporosis until after transplantation because pretransplant fractures do not affect morbidity or recovery from major surgery.
- B.** Use chronic high-dose corticosteroids to increase bone formation because glucocorticoids counteract hepatic osteodystrophy in cirrhosis.
- C.** Treat bone disease before transplantation when indicated and optimize vitamin D, calcium, fall prevention, and modifiable risk factors.
- D.** Avoid all antiresorptive therapy permanently because chronic liver disease makes osteoporosis medications uniformly hepatotoxic.
- E.** Perform TIPS because reducing portal pressure reliably normalizes bone density and replaces standard osteoporosis treatment.

130. A 58-year-old transplant candidate with cirrhosis has never undergone age-appropriate colorectal cancer screening. Why should this be addressed before listing?

- A.** Cancer screening is unnecessary before transplant because immunosuppression eliminates preexisting premalignant lesions and reduces cancer mortality.

- B.** Only HCC screening matters in cirrhosis because extrahepatic malignancy never influences candidacy for liver transplantation.
- C.** Unrecognized malignancy can affect transplant candidacy, and standard preventive screening should be completed when clinically feasible.
- D.** Colonoscopy should always be delayed until after transplantation because portal hypertension makes all pretransplant endoscopy contraindicated.
- E.** Screening is relevant only in living donors because recipient malignancy has no effect on graft allocation or post-transplant outcomes.

131. A woman of childbearing potential with decompensated liver disease is being evaluated for transplantation. Which counseling issue should be addressed explicitly?

- A.** Reproductive counseling is unnecessary because fertility is permanently lost in all women with decompensated cirrhosis and never returns after transplantation.
- B.** Contraception and reproductive planning, including how pregnancy timing and post-transplant medications may affect maternal and fetal risk.
- C.** Pregnancy should be encouraged immediately after transplantation because graft rejection risk is lowest during the first three postoperative months.
- D.** All immunosuppressive drugs have identical pregnancy safety profiles, so medication choice never influences reproductive planning.
- E.** Permanent sterilization is required for transplant listing because pregnancy after liver transplantation is universally contraindicated.

132. A frail transplant candidate with cirrhosis has poor oral intake and sarcopenia. Which nutritional recommendation is most appropriate?

- A.** Provide adequate protein and calories with frequent meals and a late-evening snack to reduce prolonged fasting and muscle catabolism.
- B.** Use severe protein restriction because muscle breakdown is preferable to dietary nitrogen intake in patients at risk for hepatic encephalopathy.
- C.** Recommend prolonged overnight fasting because glycogen depletion increases hepatic fat oxidation and preserves skeletal muscle in cirrhosis.

D. Avoid resistance activity because any increase in muscle mass raises ammonia production and worsens portal hypertension.

E. Limit calories aggressively because weight gain before transplantation is always more dangerous than sarcopenia or malnutrition.

133. A deceased-donor liver biopsy shows substantial macrovesicular steatosis. Which feature is most relevant when estimating early graft risk?

A. Microvesicular steatosis alone determines graft discard because it is the strongest independent predictor of primary nonfunction in all recipients.

B. Any hepatic steatosis guarantees primary nonfunction, so steatotic donor grafts should never be used under any clinical circumstances.

C. Macrovesicular steatosis can increase susceptibility to ischemia-reperfusion injury and should be interpreted with donor and recipient risk together.

D. Steatosis affects only late metabolic recurrence and has no influence on early allograft function after deceased-donor transplantation.

E. Recipient disease severity is irrelevant once donor steatosis is identified because graft histology alone determines transplant outcome.

134. A liver from a donation-after-circulatory-death donor is offered to a candidate. Which graft complication is relatively more concerning than with a standard donation-after-brain-death graft?

A. Recurrent autoimmune hepatitis caused by transfer of donor lymphocytes during the circulatory-death process.

B. De novo Wilson disease because warm ischemia alters copper transport proteins in donor hepatocytes after reperfusion.

C. Ischemic cholangiopathy related to warm ischemic injury of the biliary microcirculation.

D. Primary sclerosing cholangitis because DCD procurement directly creates the autoimmune phenotype responsible for multifocal strictures.

E. Portal hypertensive gastropathy because circulatory-death donors transmit systemic venous congestion to the recipient after transplantation.

135. A transplant program is considering an HCV-viremic donor liver for an HCV-negative recipient. Which principle best reflects contemporary practice?

- A.** HCV-viremic donor livers are absolutely prohibited for HCV-negative recipients because post-transplant infection cannot be cured under immunosuppression.
- B.** The recipient should receive interferon for six months before transplantation because preemptive interferon prevents transmission from a viremic graft.
- C.** No antiviral planning is needed because HCV transmission from a viremic donor liver is uncommon and usually clears spontaneously after transplantation.
- D.** Only recipients with hepatocellular carcinoma may accept HCV-viremic grafts because viral transmission is permitted solely under oncology allocation rules.
- E.** The graft can be used in selected recipients when informed consent and reliable access to prompt direct-acting antiviral therapy are in place.

136. A donor is anti-HBc positive and HBsAg negative. The intended recipient has no evidence of active HBV infection. What is the main transplant concern?

- A.** The graft cannot transmit HBV because absence of donor HBsAg proves that no viral genome remains in hepatocytes.
- B.** The donor liver will develop hepatitis D without HBV because anti-HBc positivity is a marker of autonomous HDV replication.
- C.** Only the donor kidneys carry HBV risk because core antibody positivity in a liver donor does not affect hepatic transplantation.
- D.** The recipient should receive live HBV vaccine during surgery because immediate vaccination alone reliably prevents donor-derived infection under induction immunosuppression.
- E.** Occult HBV in the donor liver can reactivate in the recipient, so recipient immunity and antiviral prophylaxis strategy must be considered.

137. What is a major rationale for ex vivo machine perfusion of donor livers?

- A.** It permanently eliminates all donor-derived pathogens because perfusion fluid sterilizes hepatocytes and biliary epithelium before transplantation.
- B.** It removes donor HLA antigens from the liver, making ABO and HLA compatibility irrelevant after implantation.

- C.** It converts a deceased-donor graft into an immunologically autologous organ by replacing donor leukocytes with recipient cells.
- D.** It can reduce cold ischemic stress and provide functional assessment or reconditioning before implantation.
- E.** It guarantees that any steatotic or injured graft will function normally, eliminating the need for donor and recipient risk assessment.

138. During normothermic machine perfusion, which type of observation is most useful for assessing graft viability?

- A.** Donor blood type alone because ABO group predicts metabolic viability more accurately than any measurement made during perfusion.
- B.** A single preperfusion aminotransferase value because dynamic functional changes during perfusion do not correlate with graft performance.
- C.** Recipient MELD score alone because donor-organ function cannot be assessed outside the body before implantation.
- D.** The color of the perfusion circuit tubing because gross appearance is the validated sole criterion for transplantable graft function.
- E.** A composite of metabolic and perfusion behavior, such as lactate clearance, bile production or characteristics, flow, and biochemical stability.

139. A marginal donor liver has advanced donor age, moderate macrosteatosis, and anticipated prolonged cold ischemia. Which allocation principle is most appropriate?

- A.** Offer the graft only to the sickest possible recipient because higher recipient acuity neutralizes donor-related ischemic and metabolic risk.
- B.** Ignore cold ischemia time because donor age and steatosis have no interaction with preservation injury in liver transplantation.
- C.** Avoid stacking multiple donor and recipient risk factors when possible because graft risk is cumulative rather than determined by one feature alone.
- D.** Discard the organ automatically because any two nonstandard donor features guarantee primary nonfunction despite recipient selection or machine perfusion.

E. Use the graft only in pediatric recipients because children are uniformly protected from the effects of donor age, steatosis, and prolonged preservation.

140. A healthy adult wishes to donate a right hepatic lobe to a sibling. Volumetric imaging predicts an inadequate remnant liver volume for the donor. What is the best response?

A. Proceed if the recipient signs a waiver because recipient urgency can ethically override a predictable risk of donor liver failure.

B. Reduce the donor remnant further because a smaller remnant hypertrophies faster and therefore lowers the risk of postoperative liver failure.

C. Use preoperative TIPS in the donor because portal decompression allows safe removal of nearly the entire liver in healthy adults.

D. Do not proceed with that planned donation because donor safety takes priority over recipient need.

E. Proceed only if the donor accepts financial compensation because payment changes the acceptable medical threshold for donor risk.

141. A living-donor graft is predicted to be very small relative to the recipient's metabolic needs and portal inflow. What complication is of greatest concern?

A. Primary sclerosing cholangitis because graft size directly induces autoimmune large-duct inflammation after living-donor transplantation.

B. Small-for-size syndrome with impaired graft function, cholestasis, ascites, and portal hyperperfusion.

C. Hepatitis B recurrence because smaller grafts contain fewer hepatocytes and therefore cannot respond to nucleos(t)ide analogue prophylaxis.

D. Graft-versus-host disease because a small graft necessarily contains a higher absolute number of immunocompetent donor lymphocytes.

E. Budd-Chiari syndrome because recipient portal inflow selectively thromboses the donor hepatic veins whenever graft volume is reduced.

142. Which preoperative study is essential for a prospective living liver donor because it defines both graft and remnant anatomy?

- A.** Screening endoscopy alone because variceal anatomy predicts donor hepatic arterial and biliary branching more accurately than cross-sectional imaging.
- B.** Bone-density scanning alone because skeletal mass determines graft volume and donor postoperative liver regeneration.
- C.** Diagnostic ERCP in every donor because invasive duct cannulation is the safest routine method for screening all healthy volunteers.
- D.** Liver biopsy in every donor regardless of imaging or risk because histology replaces the need to assess vascular anatomy and remnant volume.
- E.** High-quality cross-sectional imaging with vascular and biliary mapping plus volumetric analysis.

143. A prospective living donor asks about risk. Which counseling statement is most accurate?

- A.** Living liver donation has a small but real risk of serious morbidity and death, so consent must include complications and alternatives without minimizing risk.
- B.** Donation is risk free in healthy adults because hepatic regeneration prevents all major postoperative complications after partial hepatectomy.
- C.** The only meaningful donor risk is a temporary scar because bile leaks, thrombosis, bleeding, and liver failure occur only in transplant recipients.
- D.** Long-term health cannot be discussed because no postoperative follow-up is needed once the donor liver volume has regenerated.
- E.** Donor risk becomes ethically irrelevant when the intended recipient has a high MELD score and no deceased-donor offer.

144. A potential living donor has mild hepatic steatosis related to recent weight gain but no fibrosis or metabolic disease. Which approach is most appropriate?

- A.** Proceed without further assessment because any degree of steatosis is harmless in living donors with normal aminotransferases.
- B.** Permanently exclude the donor because even minimal steatosis cannot improve and always causes donor postoperative liver failure.
- C.** Start corticosteroids to remove steatosis rapidly because living-donor fatty liver is driven by immune-mediated hepatocyte inflammation.

D. Perform TIPS before donation because portal decompression reverses hepatic steatosis and increases the donor's future remnant volume.

E. Use individualized risk assessment and consider weight-loss intervention with repeat evaluation if steatosis is potentially reversible.

145. A deceased donor liver is anatomically suitable for splitting. What is the principal potential benefit of split-liver transplantation?

A. Splitting eliminates rejection because each partial graft contains too few donor leukocytes to stimulate recipient alloimmunity.

B. Split grafts cannot develop biliary complications because division of the liver removes the peribiliary vascular plexus from both grafts.

C. The technique is used only to transplant two adult recipients of identical body size because pediatric use is contraindicated.

D. One donor liver can provide grafts for two recipients when anatomy, donor quality, recipient selection, and center expertise are appropriate.

E. Splitting doubles the total functional hepatocyte mass by stimulating immediate regeneration before either graft is implanted.

146. During living-donor hepatectomy planning, CT angiography identifies two separate arterial inflow branches to the right lobe, including a branch arising from the superior mesenteric artery. Why must this anatomy be defined before procurement and implantation?

A. The variant makes transplantation impossible because any accessory hepatic artery is an absolute contraindication to organ donation.

B. The artery must be preserved or reconstructed appropriately because unrecognized arterial anatomy can cause graft ischemia and biliary injury.

C. The variant is irrelevant because the liver has no arterial contribution to the biliary tree after transplantation.

D. The artery should be intentionally ligated because accessory arterial inflow increases the risk of hepatic-artery thrombosis after implantation.

E. The finding predicts recipient portal-vein thrombosis rather than arterial or biliary complications and therefore requires portal anticoagulation only.

147. Why does hepatic-artery compromise after liver transplantation frequently produce biliary ischemia even when portal-vein flow is preserved?

- A.** The portal vein normally supplies all oxygen to the bile ducts, so arterial thrombosis causes biliary injury only by reducing portal pressure.
- B.** The transplanted biliary epithelium depends predominantly on the hepatic arterial peribiliary vascular plexus for its blood supply.
- C.** Bile ducts receive their blood exclusively from lymphatic channels that collapse when the hepatic artery thromboses.
- D.** The hepatic artery carries bile rather than blood after transplantation, so thrombosis directly obstructs biliary drainage.
- E.** Portal venous blood is diverted away from the liver after transplantation, leaving the artery as the sole source of all hepatocyte perfusion.

148. An ABO-incompatible liver graft is being considered because no compatible organ is available. What is the major immunologic concern?

- A.** Preformed isohemagglutinins can injure the graft vasculature and bile ducts, increasing antibody-mediated complications without appropriate mitigation.
- B.** ABO incompatibility causes only T-cell-mediated rejection because anti-blood-group antibodies do not recognize liver endothelium.
- C.** The only risk is recipient hemolysis from donor red cells, whereas the liver graft itself is immunologically protected from ABO antibodies.
- D.** ABO incompatibility is identical to HLA mismatch and therefore has no additional relevance in liver transplantation.
- E.** ABO-incompatible liver transplantation is impossible under any circumstance because no desensitization or antibody-management strategy can reduce risk.

149. A child with fulminant liver failure has only an ABO-incompatible donor option. Which principle best guides the decision?

- A.** ABO-incompatible grafting should never be considered even when death is otherwise imminent because incompatibility causes universal hyperacute graft loss.

- B.** Delay transplantation until the child reaches adulthood because ABO antibodies disappear permanently with age and make later grafting risk free.
- C.** Use no immunosuppression after surgery because ABO incompatibility is mediated only by complement and not by adaptive immunity.
- D.** The urgency of liver failure may justify ABO-incompatible transplantation with an experienced desensitization and monitoring protocol.
- E.** Perform TIPS instead because portal decompression can replace transplantation in fulminant hepatic failure while waiting for a compatible donor.

150. Within hours of transplantation, a recipient has massive aminotransferase release, severe acidosis, persistent coagulopathy, hemodynamic instability, and no meaningful bile production despite patent major vessels. Which diagnosis is most concerning?

- A.** Primary nonfunction of the liver allograft.
- B.** Mild early allograft dysfunction with spontaneous recovery.
- C.** Hyperacute chronic rejection of the liver allograft.
- D.** Rapid recurrent cirrhosis in the liver allograft.
- E.** Post-transplant lymphoproliferative disorder.

151. On postoperative day 3, a transplant recipient has elevated bilirubin, INR, and aminotransferases but remains hemodynamically stable, and Doppler flow is satisfactory. The laboratory pattern does not meet catastrophic primary nonfunction. What is the broader syndrome?

- A.** Chronic rejection, because any bilirubin and INR elevation within the first week proves irreversible ductopenic rejection.
- B.** Recurrent autoimmune hepatitis, because native-disease recurrence is the usual cause of abnormal tests during the first postoperative week.
- C.** Early allograft dysfunction, which describes impaired early graft function and requires monitoring for recovery and reversible causes.
- D.** Post-transplant cirrhosis, because fibrosis can develop rapidly enough to explain postoperative day-3 synthetic dysfunction.

E. Hepatic adenoma recurrence, because donor hepatocytes develop benign tumors immediately when exposed to postoperative immunosuppression.

152. On postoperative day 2, a liver transplant recipient develops abrupt aminotransferase elevation and loss of hepatic arterial Doppler signal. What is the most urgent concern?

A. Acute cellular rejection, because rejection selectively eliminates hepatic arterial flow while leaving portal venous and biliary flow unchanged.

B. Recurrent hepatitis C, because viral recurrence commonly causes complete arterial occlusion during the first 48 hours after transplantation.

C. Post-transplant lymphoproliferative disorder, because early EBV proliferation typically presents with absent arterial Doppler flow.

D. Calcineurin-inhibitor nephrotoxicity, because tacrolimus-induced vasoconstriction causes irreversible hepatic-artery thrombosis before kidney injury occurs.

E. Early hepatic-artery thrombosis requiring immediate confirmatory imaging and potential revascularization or retransplantation.

153. A transplant recipient develops abdominal pain, ascites, and worsening graft function. Doppler ultrasound shows absent portal venous flow. What is the most likely early vascular complication?

A. Portal-vein thrombosis causing loss of portal inflow.

B. Hepatic-artery stenosis with preserved portal flow.

C. Acute T-cell-mediated rejection with graft inflammation.

D. Anastomotic biliary stricture causing cholestasis.

E. Post-transplant diabetes causing metabolic graft dysfunction.

154. A recipient of a small living-donor graft develops persistent cholestasis, ascites, coagulopathy, and poor graft function with marked portal hyperperfusion. Which mechanism best explains small-for-size syndrome?

A. Excess portal inflow relative to graft mass causes sinusoidal injury and impaired hepatic recovery.

- B.** Insufficient portal inflow is universal in small grafts, causing ischemia that is corrected by increasing portal pressure as much as possible.
- C.** The syndrome is caused by recipient antibodies against donor ABO antigens even when the transplant is fully ABO compatible.
- D.** Small grafts contain too few bile ducts, so extrahepatic mechanical obstruction is the sole mechanism of postoperative cholestasis.
- E.** The syndrome results from immediate recurrence of the recipient's original cirrhosis because smaller grafts fibrose faster after implantation.

155. A liver transplant recipient is CMV-seronegative and receives a graft from a CMV-seropositive donor. Which preventive strategy is typically appropriate?

- A.** No CMV prevention is needed because the absence of recipient antibodies prevents donor-derived viral replication after transplantation.
- B.** Antiviral prophylaxis or a rigorously structured prevention strategy because this donor-positive/recipient-negative pairing carries high CMV risk.
- C.** Use only acyclovir at herpes-simplex dosing because CMV is fully suppressed by low-dose anti-HSV prophylaxis.
- D.** Give a live CMV vaccine immediately after transplantation because live vaccines are most effective during induction immunosuppression.
- E.** Stop immunosuppression entirely for the first three months because avoiding calcineurin inhibitors is the standard method of preventing CMV disease.

156. A liver transplant recipient is receiving high-intensity immunosuppression early after surgery. Which medication is commonly used to prevent *Pneumocystis jirovecii* pneumonia?

- A.** Oral vancomycin prophylaxis.
- B.** Oseltamivir prophylaxis.
- C.** Fluconazole prophylaxis.
- D.** Rifaximin prophylaxis.
- E.** Trimethoprim-sulfamethoxazole.

157. A liver transplant recipient has prolonged surgery, massive transfusion, renal replacement therapy, and reoperation for abdominal bleeding. Which infectious risk is particularly increased?

- A.** Measles, because transfusion and reoperation specifically eliminate prior vaccine immunity and create a unique early measles risk.
- B.** Chronic hepatitis D, because surgical complexity promotes autonomous HDV infection even in the absence of HBV.
- C.** Rabies, because prolonged surgery is the principal determinant of donor-derived rabies transmission after liver transplantation.
- D.** Invasive fungal infection, supporting consideration of targeted antifungal prophylaxis in high-risk recipients.
- E.** *Clostridioides difficile* only, because invasive fungal risk is unrelated to renal replacement therapy, transfusion, or reoperation.

158. A liver transplant recipient with prior HBV has undetectable HBV DNA at surgery. Which principle should guide post-transplant recurrence prevention?

- A.** Stop all HBV therapy once the native liver is removed because extrahepatic viral reservoirs cannot infect donor hepatocytes.
- B.** Use interferon as the preferred lifelong prophylaxis because it has fewer rejection interactions than nucleos(t)ide analogues.
- C.** Rely only on HBsAg testing once yearly because prophylactic antivirals increase rather than decrease graft reinfection risk.
- D.** Maintain potent antiviral suppression, with additional hepatitis B immune globulin use tailored to recurrence risk and center strategy.
- E.** Use lamivudine for one week after surgery and then stop because modern grafts are intrinsically resistant to HBV infection.

159. Three days after transplant, a recipient on tacrolimus develops tremor, confusion, hypertension, and seizures. MRI shows posterior vasogenic edema. What is the most likely diagnosis?

- A.** Hepatic encephalopathy from early graft dysfunction.
- B.** Progressive multifocal leukoencephalopathy.

- C. Posterior reversible encephalopathy syndrome related to tacrolimus.
- D. Acute bacterial meningitis after transplantation.
- E. Recurrent Wilson disease causing acute neurotoxicity.

160. A new liver transplant recipient develops rising creatinine with high tacrolimus exposure, bland urinalysis, and no hemodynamic cause. What is the best initial management principle?

- A. Increase tacrolimus because higher trough levels reverse renal vasoconstriction by saturating intrarenal calcineurin receptors.
- B. Reduce calcineurin-inhibitor exposure and reassess interacting medications while maintaining adequate rejection prophylaxis.
- C. Stop all immunosuppression permanently because calcineurin nephrotoxicity cannot improve after dose reduction or regimen adjustment.
- D. Begin NSAID therapy because prostaglandin inhibition protects the kidney from tacrolimus-induced afferent vasoconstriction.
- E. Perform TIPS because portal decompression is the primary treatment for calcineurin-inhibitor nephrotoxicity after liver transplantation.

161. A recipient of an HCV-viremic donor liver has confirmed early post-transplant HCV viremia. What is the appropriate next step when the planned medication access is available?

- A. Observe for spontaneous clearance for one year because donor-derived HCV usually resolves without therapy under post-transplant immunosuppression.
- B. Start pegylated interferon because direct-acting antivirals are contraindicated during the first year after liver transplantation.
- C. Initiate a compatible direct-acting antiviral regimen promptly, accounting for renal function and immunosuppressant interactions.
- D. Withdraw all immunosuppression because restoration of T-cell function is the standard treatment for donor-derived acute HCV infection.
- E. Retransplant immediately because HCV transmission from a donor graft cannot be cured once viremia is detectable in the recipient.

162. A donor lived for years in an area endemic for *Strongyloides* and has positive serology. What is the major concern for an immunosuppressed liver recipient?

- A.** Donor-derived *Strongyloides* can cause severe hyperinfection, so donor or recipient treatment planning is warranted.
- B.** *Strongyloides* cannot be transmitted by organ transplantation because the parasite remains permanently confined to the donor intestine.
- C.** The only risk is mild self-limited diarrhea because immunosuppression prevents larval dissemination into the lungs and bloodstream.
- D.** Positive donor serology means the organ must always be discarded because effective antiparasitic prevention is impossible after transplantation.
- E.** The parasite causes only chronic cholangiopathy and cannot produce systemic hyperinfection in solid-organ transplant recipients.

163. After transplantation, a donor culture obtained before procurement grows an organism resistant to many antibiotics. The recipient is currently asymptomatic. What is the best principle?

- A.** Ignore all donor cultures obtained after procurement because they cannot affect a recipient who has already received the graft.
- B.** Remove the graft immediately because any donor bacterial culture positivity mandates retransplantation regardless of organism or clinical context.
- C.** Urgently review donor and recipient cultures with transplant infectious-disease experts and provide targeted therapy when transmission risk is meaningful.
- D.** Stop immunosuppression completely because immune reconstitution alone is the standard prophylaxis for all donor-derived bacterial infections.
- E.** Give the same empiric antibiotic to every recipient because organism susceptibility and the site of donor infection do not influence treatment.

164. Several recipients from one deceased donor develop rapidly progressive encephalitis shortly after transplantation. What possibility should be considered immediately?

- A.** Independent calcineurin-inhibitor neurotoxicity in each recipient.
- B.** Recurrent hepatic encephalopathy in each organ recipient.

- C. Acute cellular rejection occurring in each organ recipient.
- D. Donor-derived copper overload causing Wilson-like disease.
- E. A donor-derived neuroinvasive infection affecting multiple recipients.

165. A potential deceased donor previously had melanoma that was treated many years ago. Why is this history especially concerning?

- A. Melanoma never metastasizes through transplanted organs because malignant melanocytes cannot survive cold preservation.
- B. Remote cancer history is irrelevant if current imaging is negative because no malignancy can remain dormant longer than five years.
- C. The only risk is recipient skin pigmentation because donor melanocytes repopulate the recipient epidermis after transplantation.
- D. Melanoma can recur after long latency and donor-derived transmission under immunosuppression can be aggressive.
- E. Prior melanoma increases bacterial infection risk but does not affect donor-derived malignancy risk in immunosuppressed recipients.

166. A donor is found after procurement to have an unexpected malignant tumor with meaningful transmission potential. What is the best immediate systems response?

- A. Inform only the liver recipient because other transplanted organs cannot transmit donor malignancy from the same donor.
- B. Wait until a recipient becomes symptomatic because notifying teams before clinical disease appears creates unnecessary surveillance and anxiety.
- C. Destroy all donor records because the recipient's privacy makes cross-organ communication impermissible after transplantation.
- D. Stop immunosuppression in every recipient without further evaluation because this uniformly eradicates all donor-derived malignant cells.
- E. Notify all recipient teams and relevant transplant oversight networks promptly so recipients can undergo coordinated risk assessment and management.

167. On postoperative day 12 after liver transplantation, unexplained graft-enzyme elevation prompts biopsy after Doppler confirms patent vessels and imaging excludes biliary obstruction. Histology shows a portal mixed inflammatory infiltrate, lymphocytic bile-duct damage, and subendothelial inflammation of portal and hepatic venules. Which diagnosis best fits?

- A.** Chronic ductopenic rejection with progressive cholestasis.
- B.** Acute T-cell-mediated rejection of the liver allograft.
- C.** Hepatic-artery thrombosis with ischemic biliary injury.
- D.** Recurrent steatotic liver disease in the allograft.
- E.** Post-transplant lymphoproliferative disorder involving the graft.

168. A clinically significant acute T-cell-mediated rejection episode is confirmed on biopsy in a stable liver transplant recipient. What is the usual initial therapy?

- A.** Stop all immunosuppression because withdrawal allows donor-specific tolerance to develop and reverses T-cell-mediated rejection.
- B.** Perform urgent retransplantation in every case because acute cellular rejection is never reversible with medical therapy.
- C.** Increase immunosuppression, commonly with a corticosteroid pulse while optimizing the maintenance regimen.
- D.** Begin antiviral therapy alone because acute T-cell-mediated rejection is caused by occult CMV replication rather than alloimmune injury.
- E.** Place a TIPS because portal decompression directly suppresses lymphocyte trafficking and is first-line treatment for graft rejection.

169. A liver transplant recipient has cholestatic graft dysfunction. Biopsy shows microvascular injury, and testing reveals strong donor-specific HLA antibodies with compatible complement staining. What immune process should be considered?

- A.** Acute T-cell-mediated rejection without antibody involvement.
- B.** Antibody-mediated rejection of the liver allograft.
- C.** Recurrent primary biliary cholangitis in the allograft.
- D.** Ischemic cholangiopathy with a patent hepatic artery.

E. Graft-versus-host disease caused by donor lymphocytes.

170. A liver recipient has biopsy-supported antibody-mediated rejection with high donor-specific antibody levels and progressive graft dysfunction. Which treatment principle is most appropriate?

A. Treat only with lactulose because antibody-mediated rejection is driven by ammonia accumulation rather than alloantibodies.

B. Stop immunosuppression because donor-specific antibody levels fall most rapidly when recipient lymphocytes are fully activated.

C. Use TIPS as definitive therapy because portal decompression removes donor-specific antibodies from the hepatic circulation.

D. Use a multimodal strategy aimed at reducing antibody production or effect while optimizing baseline immunosuppression.

E. No therapy is indicated because antibody-mediated rejection cannot cause clinically meaningful liver graft dysfunction.

171. Years after transplantation, a recipient develops progressive cholestasis. Biopsy shows loss of interlobular bile ducts and chronic vascular changes without a mechanical biliary obstruction. What is the most likely diagnosis?

A. Acute T-cell-mediated rejection with portal endopheliitis.

B. Anastomotic biliary stricture with focal obstruction.

C. Recurrent chronic hepatitis E in the liver allograft.

D. Calcineurin-inhibitor nephrotoxicity causing graft cholestasis.

E. Chronic rejection with progressive ductopenic injury.

172. Which history most increases concern for chronic rejection in a liver transplant recipient?

A. Repeated or inadequately treated rejection episodes and persistent under-immunosuppression or nonadherence.

B. Consistently therapeutic immunosuppression with no prior rejection because chronic rejection is most common in fully tolerant recipients.

- C.** Isolated post-transplant weight gain because metabolic syndrome directly causes ductopenia and obliterative arteriopathy.
- D.** Successful vaccination before transplantation because vaccine-induced antibodies cross-react with bile ducts and cause chronic rejection.
- E.** Remote appendectomy because prior abdominal surgery is the principal predictor of late ductopenic graft injury.

173. A liver recipient develops graft dysfunction with plasma-cell-rich interface hepatitis years after transplant. The original disease was not autoimmune hepatitis. Viral causes and biliary obstruction are excluded. What should be considered?

- A.** Recurrent autoimmune hepatitis, because any plasma cells in the graft prove that the recipient's native disease was autoimmune despite the original diagnosis.
- B.** Hepatic-artery thrombosis, because arterial ischemia typically produces plasma-cell-rich interface hepatitis without biliary abnormalities.
- C.** Post-transplant lymphoproliferative disorder, because plasma-cell-rich portal infiltrates always represent monoclonal EBV-driven lymphoma.
- D.** Primary nonfunction, because this syndrome routinely first appears years after transplant with interface hepatitis and normal vascular imaging.
- E.** Plasma cell-rich rejection, an alloimmune graft hepatitis that can resemble autoimmune hepatitis.

174. Six weeks after liver transplantation, a recipient develops fever, diffuse rash, profuse diarrhea, and pancytopenia. Liver tests are only mildly abnormal. What diagnosis is most concerning?

- A.** Acute T-cell-mediated rejection, which is caused by recipient lymphocytes attacking the graft and rarely produces rash, diarrhea, and profound pancytopenia together.
- B.** CMV hepatitis alone, which cannot cause gastrointestinal symptoms or cytopenias in a transplant recipient.
- C.** Hepatic-artery stenosis, which primarily causes biliary ischemia and does not explain fever, rash, diarrhea, and marrow suppression.
- D.** Graft-versus-host disease from immunocompetent donor lymphocytes attacking recipient tissues.
- E.** Recurrent alcohol-associated hepatitis, which commonly causes diffuse rash and abrupt pancytopenia during the first two months after transplant.

175. Which cellular direction of immune attack distinguishes liver-transplant graft-versus-host disease from ordinary allograft rejection?

- A.** Recipient neutrophils attack donor red cells in graft-versus-host disease, whereas donor antibodies attack recipient bile ducts in conventional rejection.
- B.** Both disorders are identical directions of recipient-against-donor immunity and differ only in the timing after transplantation.
- C.** Graft-versus-host disease is caused by donor hepatocytes attacking recipient kidneys, whereas rejection is caused only by recipient antibodies against donor leukocytes.
- D.** Conventional rejection occurs without adaptive immunity, whereas graft-versus-host disease requires only mechanical vascular obstruction and no lymphocytes.
- E.** Donor immune cells attack recipient tissues in graft-versus-host disease, whereas recipient immune cells attack the donor organ in conventional rejection.

176. A transplant recipient repeatedly misses tacrolimus doses because of medication cost and now has biopsy-proven rejection. What is the most important long-term intervention in addition to treating the rejection episode?

- A.** Increase the prescribed tacrolimus dose without addressing cost because a larger prescription guarantees adequate exposure despite missed doses.
- B.** Remove the patient from all transplant follow-up because nonadherence is an immutable behavior and cannot be improved with system support.
- C.** Stop tacrolimus permanently and use no maintenance immunosuppression because adherence problems are best solved by eliminating all medications.
- D.** Correct the access barrier and build a sustainable adherence plan because recurrent underexposure can lead to further rejection and graft loss.
- E.** Switch to corticosteroid monotherapy indefinitely because prednisone requires no monitoring and prevents all forms of chronic rejection despite missed doses.

177. Four months after liver transplantation, a patient develops cholestatic liver tests. MRCP shows a short narrowing at the duct-to-duct anastomosis with upstream dilation. What is the most likely complication?

- A.** Chronic rejection causing diffuse ductopenic graft injury.
- B.** Hepatic-artery thrombosis causing diffuse ischemic biliary injury.
- C.** Recurrent primary sclerosing cholangitis with multifocal strictures.
- D.** An anastomotic biliary stricture with upstream dilation.
- E.** Post-transplant lymphoproliferative disorder causing duct obstruction.

178. A recipient with duct-to-duct biliary reconstruction has a symptomatic anastomotic stricture. What is the usual first-line therapeutic approach?

- A.** Endoscopic therapy with dilation and stent-based management when anatomy permits.
- B.** Immediate retransplantation because anastomotic strictures do not respond to endoscopic or percutaneous interventions.
- C.** High-dose corticosteroids because focal biliary anastomotic narrowing is an alloimmune lesion that reverses with treatment of rejection.
- D.** TIPS placement because reducing portal pressure mechanically enlarges the biliary anastomosis and restores bile flow.
- E.** Long-term anticoagulation because anastomotic biliary strictures are caused by portal-vein thrombosis even when portal flow is normal.

179. A recipient of a DCD liver develops recurrent cholangitis. Cholangiography shows multiple intrahepatic nonanastomotic strictures and biliary casts despite a patent main hepatic artery. What is the likely diagnosis?

- A.** A focal postoperative anastomotic biliary stricture.
- B.** Ischemic cholangiopathy from injury to the biliary microcirculation.
- C.** Acute cellular rejection with inflammatory bile-duct injury.
- D.** Recurrent primary biliary cholangitis in the allograft.
- E.** Tacrolimus nephrotoxicity causing indirect biliary injury.

180. A liver recipient develops new ascites and abnormal graft tests months after transplantation. Doppler suggests focal narrowing of the portal vein with increased velocity across the lesion. What is the likely problem?

- A.** Biliary anastomotic stricture, which produces ductal dilation but does not create focal high-velocity portal venous flow.
- B.** Portal-vein stenosis causing impaired portal inflow and recurrent portal hypertensive physiology.
- C.** Acute rejection, which causes portal inflammation but not a mechanical high-velocity stenosis on vascular imaging.
- D.** Post-transplant diabetes, which can cause ascites only through osmotic diuresis and does not affect portal-vein velocities.
- E.** Pneumocystis pneumonia, which explains hypoxemia but cannot cause focal portal-vein narrowing and graft dysfunction.

181. A transplant recipient has significant hepatic-artery stenosis with threatened biliary perfusion but no complete thrombosis. What is the best management principle?

- A.** Observe until bilirubin exceeds 20 mg/dL because arterial stenosis does not affect the graft while hepatocyte portal flow remains present.
- B.** Treat with lactulose because reducing ammonia lowers hepatic arterial resistance and reverses the mechanical stenosis.
- C.** Use corticosteroids because all post-transplant arterial narrowing is caused by acute T-cell-mediated rejection of the arterial wall.
- D.** Prompt endovascular or surgical correction should be considered because untreated arterial stenosis can progress to thrombosis and ischemic biliary injury.
- E.** Perform TIPS because increased portal inflow automatically restores hepatic arterial diameter through the arterial buffer response.

182. A patient without prior diabetes develops persistent fasting hyperglycemia three months after liver transplantation while taking tacrolimus and corticosteroids. What is the most likely diagnosis?

- A.** Post-transplant diabetes mellitus related to baseline metabolic risk and diabetogenic immunosuppression.

- B.** Hepatorenal syndrome, which presents with persistent hyperglycemia because renal vasoconstriction impairs insulin clearance.
- C.** Acute rejection, because all rejection episodes primarily cause diabetes before causing liver-test abnormalities.
- D.** Chronic hepatitis E, which is the usual direct cause of hyperglycemia in the first year after liver transplantation.
- E.** Biliary stricture, because cholestasis causes persistent fasting hyperglycemia through impaired bile-mediated insulin secretion.

183. A liver transplant recipient gains 18 kg in the first year and develops hypertension, diabetes, and dyslipidemia. What is the best long-term approach?

- A.** Accept the changes as inevitable because post-transplant metabolic syndrome cannot be modified without causing graft rejection.
- B.** Stop all immunosuppression because weight gain and diabetes are more dangerous than rejection once the first transplant year is complete.
- C.** Use chronic corticosteroids to improve lipid metabolism because glucocorticoids reduce post-transplant cardiovascular risk.
- D.** Treat the full cardiometabolic syndrome with lifestyle intervention, appropriate medications, and immunosuppressive optimization when feasible.
- E.** Focus only on liver tests because cardiovascular disease does not materially affect long-term survival after successful liver transplantation.

184. A liver transplant recipient has progressive chronic kidney disease with long-term tacrolimus exposure. Which factor makes this clinically important beyond kidney function itself?

- A.** Chronic kidney disease increases cardiovascular morbidity and mortality and may progress to end-stage kidney disease after liver transplantation.
- B.** Chronic kidney disease is benign after liver transplantation because the liver graft compensates for lost renal filtration and prevents cardiovascular effects.
- C.** Renal impairment lowers cardiovascular risk by reducing intravascular volume, so aggressive kidney preservation is unnecessary after transplantation.

D. Kidney disease affects only tacrolimus dosing and has no independent relationship to survival or quality of life in liver recipients.

E. Progressive renal dysfunction proves recurrent liver failure because isolated kidney disease cannot occur after a successful liver transplant.

185. A stable liver recipient has worsening chronic kidney disease attributed partly to calcineurin-inhibitor exposure. What is a reasonable immunosuppressive strategy?

A. Stop all immunosuppression immediately because kidney preservation always takes priority once chronic graft rejection risk has fallen.

B. Increase tacrolimus exposure because higher levels improve renal perfusion by suppressing intrarenal inflammatory cytokines.

C. Add chronic NSAID therapy because prostaglandin blockade protects glomerular filtration from calcineurin vasoconstriction.

D. Switch to high-dose prednisone monotherapy indefinitely because corticosteroids have no metabolic, bone, or infectious toxicity after transplantation.

E. Use a renal-sparing regimen that safely reduces calcineurin-inhibitor exposure while maintaining adequate rejection prevention.

186. A liver transplant recipient has osteoporosis and a new vertebral compression fracture. Which management is appropriate?

A. Observe only because fractures after transplantation heal spontaneously once liver synthetic function is normal.

B. Provide standard osteoporosis therapy plus calcium, vitamin D, fall-risk reduction, and review of steroid exposure.

C. Increase corticosteroids because glucocorticoids stimulate osteoblast activity and prevent further post-transplant bone loss.

D. Stop calcium and vitamin D because improved bile flow after transplantation makes supplementation toxic and causes graft calcification.

E. Perform TIPS because portal decompression is required for osteoporosis treatment even after a successful liver transplantation.

187. A transplant recipient remains sarcopenic despite normalized liver tests. Which intervention is most appropriate?

- A.** Protein restriction because a functioning liver graft still cannot metabolize dietary nitrogen and high protein causes recurrent encephalopathy.
- B.** Prolonged bed rest because physical training increases graft oxygen demand and risks chronic rejection in the first years after transplant.
- C.** Structured rehabilitation with resistance exercise and adequate protein and energy intake.
- D.** Chronic parenteral nutrition because enteral nutrition cannot restore muscle after liver transplantation.
- E.** High-dose corticosteroids because glucocorticoid-induced protein catabolism paradoxically stimulates long-term skeletal muscle growth.

188. Which cause of late morbidity deserves routine attention in long-term liver transplant survivors even when graft function is excellent?

- A.** Acute primary nonfunction, which remains the dominant late cause of death decades after a stable graft has functioned normally.
- B.** Cardiovascular disease related to age, metabolic syndrome, kidney disease, and immunosuppressive effects.
- C.** Biliary atresia recurrence, which develops in most adult donor livers and eventually causes late mortality regardless of native diagnosis.
- D.** Acute acetaminophen toxicity, which is the inevitable long-term consequence of calcineurin inhibitor metabolism.
- E.** Wilson disease in the donor graft, which commonly develops over time even when the donor did not carry a pathogenic ATP7B genotype.

189. A liver recipient who was EBV-seronegative before transplantation develops fever, lymphadenopathy, weight loss, and a rapidly enlarging abdominal mass six months after transplant. EBV DNA is high. What is the most concerning diagnosis?

- A.** Acute cellular rejection with systemic lymphadenopathy.
- B.** Hepatic-artery stenosis with ischemic graft injury.

- C. Recurrent hepatocellular carcinoma after transplantation.
- D. Pneumocystis jirovecii pneumonia after transplantation.
- E. Post-transplant lymphoproliferative disorder.

190. A liver transplant recipient has biopsy-confirmed EBV-positive monomorphic PTLN. What is an important initial treatment principle?

- A. Increase immunosuppression because PTLN is caused by recipient immune overactivity and improves when T-cell function is suppressed further.
- B. Reduce immunosuppression when feasible and use subtype-directed therapy such as rituximab-based treatment for appropriate B-cell disease.
- C. Treat only with valganciclovir because established monomorphic lymphoma is eradicated by suppressing EBV replication alone.
- D. Perform TIPS because portal decompression reduces EBV-driven lymphoid proliferation in abdominal lymph nodes and graft tissue.
- E. Observe without treatment because PTLN is a benign lymphoid response that never progresses once the liver graft is stable.

191. A long-term liver transplant recipient asks why dermatology follow-up is recommended. What is the main reason?

- A. Skin cancer risk falls below that of the general population because calcineurin inhibition suppresses ultraviolet-induced inflammation.
- B. Dermatologic surveillance is used mainly to detect acute cellular rejection because rejection first appears as malignant-appearing skin lesions.
- C. Chronic immunosuppression markedly increases the risk of cutaneous malignancy, especially keratinocyte cancers.
- D. Only melanoma risk increases, whereas squamous and basal cell skin cancers are less common after transplantation than before transplant.
- E. Skin examinations are unnecessary if liver tests remain normal because cancer risk is directly proportional to graft dysfunction.

192. A liver recipient develops dyslipidemia and has high atherosclerotic cardiovascular risk. Which principle is appropriate when prescribing a statin?

- A.** Avoid all statins because liver transplantation makes HMG-CoA reductase inhibition universally hepatotoxic even with normal graft function.
- B.** Stop immunosuppression before starting a statin because any concurrent calcineurin inhibitor makes lipid treatment impossible.
- C.** Treat only with bile-acid sequestrants because no systemic lipid-lowering drug can be used safely after liver transplantation.
- D.** Ignore dyslipidemia because post-transplant cardiovascular events are unrelated to serum lipids once the native cirrhotic liver is removed.
- E.** Use indicated lipid-lowering therapy while checking for interactions with the recipient's immunosuppressive regimen and monitoring toxicity.

193. A liver transplant recipient has persistent hypertension while taking tacrolimus. What is the best management principle?

- A.** Leave hypertension untreated because higher systemic pressure is required to maintain portal flow through a transplanted liver.
- B.** Stop all immunosuppression because hypertension proves irreversible calcineurin toxicity and makes graft rejection risk irrelevant.
- C.** Use chronic NSAID therapy because prostaglandin inhibition lowers calcineurin-induced renal vasoconstriction and blood pressure.
- D.** Treat blood pressure to standard cardiovascular goals while considering calcineurin-inhibitor contribution and drug interactions.
- E.** Perform TIPS because hypertension after transplant is caused by recurrent portal hypertension even when the graft has no fibrosis.

194. Two months after transplant, a recipient develops fever, malaise, leukopenia, and rising liver tests. CMV PCR is high. What is the most likely syndrome?

- A.** Acute cellular rejection, because leukopenia and high CMV DNA are expected biomarkers of T-cell-mediated graft injury.

- B.** Hepatic-artery thrombosis, because vascular occlusion routinely produces CMV viremia as a direct ischemic consequence.
- C.** Pneumocystis pneumonia, because Pneumocystis infection is diagnosed by high circulating CMV DNA rather than respiratory testing.
- D.** Recurrent PBC, because primary biliary cholangitis after transplant commonly causes fever, leukopenia, and high CMV viral load.
- E.** CMV infection with systemic or tissue-invasive disease, depending on organ involvement.

195. A liver recipient has symptomatic CMV disease with stable renal function and no major cytopenia precluding therapy. What treatment is appropriate?

- A.** Use acyclovir at standard HSV doses because CMV is equally sensitive to low-dose acyclovir after solid-organ transplantation.
- B.** Use ganciclovir or valganciclovir at treatment dosing, adjusted for renal function, and reduce immunosuppression when clinically feasible.
- C.** Stop all antiviral therapy and increase immunosuppression because CMV disease is caused by excessive recipient immune activation.
- D.** Treat with trimethoprim-sulfamethoxazole because Pneumocystis and CMV share the same folate-dependent replication pathway.
- E.** Use corticosteroid pulse therapy alone because CMV hepatitis is indistinguishable from rejection and improves with stronger immunosuppression.

196. A liver transplant recipient has fever and right-upper-quadrant pain with cholestatic tests. Imaging shows biliary dilation above a known anastomotic stricture. What infectious complication is most likely?

- A.** Pneumocystis jirovecii pneumonia.
- B.** Cytomegalovirus retinitis.
- C.** Cryptococcal meningitis.
- D.** Ascending bacterial cholangitis.
- E.** Graft-versus-host disease with hepatobiliary involvement.

197. A liver recipient receiving high-dose corticosteroids for rejection develops pleuritic chest pain, hemoptysis, and nodular pulmonary infiltrates with surrounding ground-glass change. Which opportunistic infection is most concerning?

- A.** Invasive pulmonary aspergillosis.
- B.** Chronic hepatitis E infection.
- C.** Primary cytomegalovirus infection.
- D.** *Pneumocystis jirovecii* pneumonia.
- E.** *Clostridioides difficile* infection.

198. A liver recipient has chronic unexplained aminotransferase elevation. HEV RNA remains positive for more than three months. What is the most likely diagnosis?

- A.** Resolved hepatitis E with persistent noninfectious RNA fragments.
- B.** Acute T-cell-mediated rejection with false-positive HEV RNA.
- C.** Chronic hepatitis E infection in an immunosuppressed host.
- D.** Recurrent hepatitis D with cross-reactive HEV testing.
- E.** Hepatic-artery stenosis with persistent false-positive HEV RNA.

199. A stable liver recipient has chronic hepatitis E with persistent viremia. What is an appropriate management strategy?

- A.** Increase immunosuppression because stronger T-cell suppression is the most reliable way to clear chronic HEV infection.
- B.** Reduce immunosuppression when safely possible and consider ribavirin if viremia persists or disease is clinically important.
- C.** Use lifelong valganciclovir because HEV is a herpesvirus and responds to inhibition of viral DNA polymerase.
- D.** Perform immediate retransplantation because chronic HEV cannot be cleared medically and always causes rapid graft failure.
- E.** Use interferon routinely in all recipients because interferon has no risk of precipitating graft rejection after liver transplantation.

200. A liver transplant recipient develops symptomatic influenza during community circulation. Which principle is most appropriate?

- A.** Avoid antivirals because transplant recipients clear influenza more effectively than immunocompetent adults and have lower complication rates.
- B.** Stop all immunosuppression immediately because immune reconstitution alone is sufficient treatment for respiratory viral infections after transplantation.
- C.** Use live intranasal influenza vaccine as treatment because it generates a faster immune response than antiviral medication during active infection.
- D.** Treat promptly with an active antiviral and assess for complications because immunosuppressed recipients are at increased risk of severe disease.
- E.** Assume any respiratory symptoms represent rejection because community respiratory viruses do not cause clinically important disease in liver recipients.

201. Years after transplantation for PBC, a recipient develops slowly rising alkaline phosphatase. Biopsy shows portal inflammation and bile-duct injury compatible with the original disease, with no obstruction or rejection. What is most likely?

- A.** Delayed primary nonfunction of the liver allograft.
- B.** Late hepatic-artery thrombosis with cholestatic graft injury.
- C.** Recurrent Wilson disease in donor hepatocytes.
- D.** Post-transplant diabetes causing chronic bile-duct injury.
- E.** Recurrent primary biliary cholangitis in the liver allograft.

202. A recipient transplanted for PSC asks whether the disease can return in the graft. Which statement is correct?

- A.** PSC cannot recur because transplantation permanently removes the immune and intestinal factors that contribute to the disease.
- B.** Recurrence occurs only if the donor also had PSC because the disease is transmitted exclusively through donor bile-duct genetics.

C. All post-transplant biliary strictures represent recurrent PSC, making vascular imaging and anastomotic assessment unnecessary.

D. PSC can recur after transplantation and may again cause multifocal biliary strictures and progressive graft disease.

E. Recurrence is prevented completely by standard calcineurin-inhibitor therapy, so long-term biliary follow-up is not required.

203. Five years after transplantation for autoimmune hepatitis, maintenance immunosuppression is reduced. The patient subsequently develops elevated IgG and hepatocellular enzymes, and biopsy again shows plasma-cell-rich interface hepatitis with no vascular or biliary lesion. What is the leading diagnosis?

A. Recurrent autoimmune hepatitis in the liver allograft.

B. Recurrent primary sclerosing cholangitis in the allograft.

C. Late hepatic-artery thrombosis causing graft hepatitis.

D. Post-transplant lymphoproliferative disorder involving the graft.

E. Tacrolimus nephrotoxicity causing secondary graft hepatitis.

204. A patient transplanted for HCC had microvascular invasion in the explant. Why is this finding important during post-transplant follow-up?

A. Microvascular invasion is a benign explant finding that lowers recurrence risk because it shows the tumor remained confined to the liver.

B. The finding affects only early hepatic-artery thrombosis risk and has no relationship to post-transplant malignancy recurrence.

C. Microvascular invasion guarantees recurrence, so surveillance is unnecessary because outcome is predetermined regardless of other tumor features.

D. The finding is relevant only to cholangiocarcinoma and has no prognostic meaning in hepatocellular carcinoma after transplantation.

E. Microvascular invasion indicates more aggressive tumor biology and increases the risk of HCC recurrence after transplantation.

205. Several years after liver transplantation for MASH, a recipient has gained substantial weight and developed poorly controlled diabetes. Biopsy of the graft now shows steatohepatitis with fibrosis. Which long-term management strategy is most appropriate?

- A.** Ignore graft steatosis because recurrent MASLD after transplantation never progresses to steatohepatitis or fibrosis.
- B.** Increase corticosteroids because recurrent MASH is an alloimmune inflammatory process that improves with stronger immunosuppression.
- C.** Perform TIPS because portal decompression is the definitive treatment for post-transplant metabolic steatosis.
- D.** Aggressively treat metabolic risk factors and assess graft fibrosis because recurrent MASLD can progress despite normal synthetic function.
- E.** Stop diabetes treatment because tighter glycemic control paradoxically accelerates graft fibrosis after transplantation.

206. A recipient transplanted for alcohol-associated cirrhosis has returned to sustained heavy alcohol use but no current graft decompensation. What is the best response?

- A.** Stop transplant medications to accelerate graft failure because relapse represents forfeiture of further medical treatment.
- B.** Take no action until bilirubin rises because alcohol cannot damage a transplanted liver before advanced fibrosis develops.
- C.** Provide prompt alcohol-use-disorder treatment and evaluate for graft injury rather than waiting for recurrent cirrhosis.
- D.** Use only punitive abstinence contracts because evidence-based addiction therapy has no role after organ transplantation.
- E.** Retransplant immediately because any alcohol use after transplantation inevitably causes irreversible graft failure within weeks.

207. A liver transplant recipient for HBV-related cirrhosis has detectable HBV DNA after a lapse in antiviral therapy. What is the best next step?

- A.** Observe because donor hepatocytes cannot be infected by HBV once the recipient's native liver has been removed.

- B.** Use interferon as the automatic first therapy because it has no rejection risk and is superior to nucleos(t)ide analogues after transplant.
- C.** Stop all immunosuppression because immune reconstitution alone is the preferred way to eliminate HBV from the graft.
- D.** Restart or optimize potent antiviral suppression and assess for resistance, adherence barriers, and graft injury.
- E.** Perform immediate retransplantation because any post-transplant HBV DNA detection indicates irreversible graft failure.

208. A stable liver transplant recipient asks about routine immunizations. Which principle is correct?

- A.** Avoid all vaccines permanently because any immune stimulation inevitably causes acute cellular rejection of the liver graft.
- B.** Prefer live vaccines because immunosuppression prevents vaccine strains from replicating and makes them safer than inactivated vaccines.
- C.** Use recommended inactivated vaccines, timed to maximize response, while generally avoiding live-attenuated vaccines under significant immunosuppression.
- D.** Vaccinate only if liver tests become abnormal because immunizations have no role in a recipient with stable graft function.
- E.** Stop immunosuppression for two weeks before every vaccine because full immune reconstitution is required for safe vaccination.

209. Which cancer-screening issue deserves heightened attention in a long-term liver transplant recipient?

- A.** Cancer screening can be reduced because immunosuppression suppresses malignant transformation and lowers overall cancer incidence.
- B.** Skin cancer surveillance because chronic immunosuppression greatly increases keratinocyte cancer risk.
- C.** Only recurrent HCC screening matters because immunosuppression does not increase the risk of any de novo extrahepatic malignancy.
- D.** Skin examinations are unnecessary if the recipient uses sunscreen because ultraviolet exposure becomes irrelevant after transplantation.

E. Colorectal and cervical screening should stop because chronic immunosuppression eliminates dysplasia caused by oncogenic viruses or inflammation.

210. A woman with a stable liver graft wishes to become pregnant. She takes tacrolimus, low-dose prednisone, and mycophenolate. What medication issue requires action before conception?

A. Tacrolimus must be permanently stopped because calcineurin inhibitors are absolutely contraindicated in every pregnancy after liver transplantation.

B. Mycophenolate is teratogenic and should be replaced with a pregnancy-compatible regimen well before conception.

C. Prednisone must be replaced by mycophenolate because mycophenolate is the safest antimetabolite during pregnancy.

D. All immunosuppression should be stopped at conception because pregnancy itself induces complete immunologic tolerance of the liver graft.

E. No medication change is needed because all maintenance immunosuppressants have equivalent fetal safety profiles.

211. A young adult who received a pediatric liver transplant is transferring to adult care. Which intervention best reduces transition-related graft risk?

A. An abrupt transfer at age 18 without overlap because independence develops most reliably when pediatric clinicians stop all involvement immediately.

B. Stopping therapeutic-drug monitoring because adult recipients should manage immunosuppression without laboratory reminders.

C. Reducing immunosuppression during the transition year because adolescents have lower rejection risk than children or older adults.

D. Using parent-only communication because direct patient involvement increases anxiety and worsens adherence during transition.

E. A structured transition process that verifies medication knowledge, refill skills, follow-up ownership, and support before transfer.

212. A stable liver recipient has low bone mineral density but no fracture. Which prevention strategy is appropriate?

- A.** Avoid exercise because mechanical loading increases hepatic venous pressure and accelerates chronic rejection in a stable transplant recipient.
- B.** Optimize calcium and vitamin D, encourage weight-bearing activity, minimize unnecessary steroids, and treat osteoporosis when standard thresholds are met.
- C.** Increase glucocorticoids because long-term steroid exposure strengthens cortical bone in post-transplant patients.
- D.** Stop vitamin D because a functioning donor liver produces enough vitamin D to eliminate skeletal risk from immunosuppression.
- E.** Wait for the first fragility fracture before any intervention because bone density has no predictive value after liver transplantation.

213. A long-term liver transplant recipient smokes cigarettes and has hypertension and diabetes. What is the best preventive priority?

- A.** Focus only on tacrolimus trough levels because conventional cardiovascular risk factors no longer affect survival after transplantation.
- B.** Use comprehensive cardiovascular risk reduction, including smoking cessation, blood-pressure and diabetes control, lipid management, diet, and activity.
- C.** Encourage continued smoking because nicotine reduces appetite and prevents the post-transplant weight gain that drives cardiovascular disease.
- D.** Avoid antihypertensive therapy because lower systemic pressure reduces graft perfusion and causes chronic rejection in stable recipients.
- E.** Stop statins and diabetes therapy because drug interactions make cardiometabolic prevention more dangerous than cardiovascular disease itself.

214. A liver transplant recipient has excellent graft tests but reports depression, sexual dysfunction, fatigue, and difficulty returning to work. Which statement is most appropriate?

- A.** These concerns are unrelated to transplant care and should be ignored unless they are accompanied by abnormal bilirubin or INR.
- B.** Normal graft tests prove the symptoms are fabricated because transplantation reliably normalizes all physical and psychological functioning.

- C.** The only appropriate intervention is higher immunosuppression because depression and fatigue are early manifestations of under-immunosuppression.
- D.** Return to work should be mandatory for all recipients with normal liver tests because social function is determined entirely by graft biochemistry.
- E.** Quality-of-life outcomes require active assessment because successful graft function does not guarantee psychosocial or functional recovery.

215. A liver transplant candidate has preformed antibodies against donor HLA antigens. Which term best describes these antibodies?

- A.** Isohemagglutinins, which are defined specifically as antibodies against HLA class I and II rather than ABO blood-group antigens.
- B.** Autoantibodies, because any antibody detected after transplantation must recognize the recipient's own tissue rather than donor antigens.
- C.** Heterophile antibodies, which are the principal antibodies responsible for chronic liver rejection and donor endothelial injury.
- D.** Donor-specific antibodies, which can participate in antibody-mediated allograft injury when clinically significant.
- E.** Anti-mitochondrial antibodies, because PBC-associated antibodies are the standard marker of HLA sensitization before transplantation.

216. Compared with several other transplanted solid organs, selected liver recipients can sometimes be maintained on relatively lower immunosuppressive intensity. Which feature of hepatic immunobiology best helps explain this observation?

- A.** The liver expresses no HLA molecules, so recipient T cells and antibodies cannot recognize donor tissue after transplantation.
- B.** Portal blood contains no lymphocytes, preventing adaptive immune cells from reaching the liver graft after implantation.
- C.** Its large antigen-presenting cell network and distinctive hepatic immune environment can dampen some alloimmune responses, although clinically important rejection still occurs.
- D.** Hepatocytes permanently delete all recipient B cells during reperfusion, making antibody-mediated rejection biologically impossible.

E. The liver is immunologically privileged because it lacks endothelium and therefore cannot present donor antigens to recipient immune cells.

217. How does tacrolimus suppress T-cell activation?

A. It inhibits inosine monophosphate dehydrogenase, selectively blocking de novo guanine synthesis in proliferating lymphocytes.

B. It binds the glucocorticoid receptor and directly alters nuclear transcription of hundreds of inflammatory genes.

C. It binds FK-binding protein and inhibits calcineurin, reducing NFAT activation and interleukin-2 transcription.

D. It blocks the interleukin-2 receptor alpha chain on activated T cells without entering the cell or affecting calcineurin signaling.

E. It inhibits mTOR complex signaling downstream of growth-factor receptors and thereby prevents cell-cycle progression without calcineurin inhibition.

218. A liver recipient develops tremor, diabetes, and kidney dysfunction on tacrolimus. Which adverse-effect profile is most consistent with this drug?

A. Neurotoxicity, nephrotoxicity, and diabetogenic effects are important dose-related toxicities of tacrolimus.

B. Severe gingival hyperplasia and hirsutism are uniquely characteristic of tacrolimus and never occur with cyclosporine.

C. Myelosuppression and teratogenicity are the dominant toxicities because tacrolimus is an antimetabolite that blocks purine synthesis.

D. Pulmonary fibrosis and delayed wound healing are universal because tacrolimus is an mTOR inhibitor rather than a calcineurin inhibitor.

E. Cytokine-release syndrome is expected with every dose because tacrolimus is a T-cell-depleting polyclonal antibody preparation.

219. A stable liver recipient on tacrolimus starts voriconazole and one week later has tremor and acute kidney injury with a markedly elevated tacrolimus trough. What is the mechanism?

- A.** Voriconazole induces CYP3A, so the high tacrolimus level reflects accelerated absorption rather than reduced metabolism.
- B.** Tacrolimus inhibits voriconazole metabolism and the measured tacrolimus concentration is falsely elevated by cross-reactive fungal metabolites.
- C.** The interaction occurs because both drugs chelate magnesium in the gut, increasing tacrolimus renal clearance and intracellular accumulation.
- D.** Voriconazole inhibits CYP3A-mediated tacrolimus metabolism, increasing tacrolimus exposure.
- E.** Voriconazole directly converts tacrolimus into cyclosporine, causing the assay to measure both calcineurin inhibitors as one concentration.

220. A liver recipient on tacrolimus starts rifampin for a compelling infectious indication and then develops subtherapeutic tacrolimus levels. What is the explanation?

- A.** Rifampin inhibits calcineurin directly, so tacrolimus levels fall because the two drugs compete for the same intracellular binding site.
- B.** Rifampin blocks tacrolimus intestinal absorption only by causing cholestasis, and the interaction disappears when bilirubin is normal.
- C.** Rifampin increases tacrolimus assay degradation but has no effect on actual drug exposure or rejection risk.
- D.** The low trough is expected because rifampin converts tacrolimus to an active metabolite with identical immunosuppressive potency and no monitoring requirement.
- E.** Rifampin strongly induces drug-metabolizing pathways, increasing tacrolimus clearance and risking under-immunosuppression.

221. How does mycophenolate suppress lymphocyte proliferation?

- A.** It binds FKBP and inhibits calcineurin, preventing NFAT activation and interleukin-2 transcription in the same way as tacrolimus.
- B.** It inhibits inosine monophosphate dehydrogenase and impairs de novo guanine nucleotide synthesis used heavily by activated lymphocytes.
- C.** It blocks the interleukin-2 receptor alpha chain and therefore acts only on activated T cells expressing CD25.

D. It alkylates lymphocyte DNA directly and produces permanent T-cell depletion after a single dose.

E. It inhibits mTOR complex 1 and therefore causes delayed wound healing through the same molecular target as sirolimus.

222. A stable liver transplant recipient taking tacrolimus and mycophenolate requests preconception counseling. Which medication-related point must be addressed before she attempts pregnancy?

A. Mycophenolate is preferred during pregnancy because it prevents congenital malformations caused by calcineurin inhibitors.

B. The only reproductive effect is temporary male infertility, so no medication change is needed for a woman planning pregnancy.

C. Mycophenolate is safe once the first trimester begins because teratogenicity is limited to the week immediately after transplantation.

D. Pregnancy requires stopping all maintenance immunosuppression because fetal tolerance eliminates maternal graft rejection risk.

E. Mycophenolate is strongly teratogenic and must be replaced with a pregnancy-compatible regimen before conception.

223. A transplant recipient taking azathioprine develops profound pancytopenia after allopurinol is added for gout. What is the mechanism?

A. Allopurinol inhibits xanthine oxidase and can markedly increase active azathioprine metabolite exposure unless the azathioprine dose is reduced.

B. Allopurinol induces CYP3A and lowers azathioprine clearance, causing accumulation of tacrolimus rather than thiopurine metabolites.

C. Azathioprine blocks uric-acid excretion, so allopurinol causes marrow failure by precipitating uric acid directly inside hematopoietic cells.

D. The interaction is caused by folate deficiency because both drugs inhibit dihydrofolate reductase and therefore mimic trimethoprim toxicity.

E. Allopurinol converts azathioprine to mycophenolate in the liver, producing overlapping antimetabolite toxicity and immediate pancytopenia.

224. What is the principal immunosuppressive target of sirolimus and everolimus?

- A.** Calcineurin phosphatase, thereby preventing NFAT activation through an FKBP complex identical to tacrolimus.
- B.** Inosine monophosphate dehydrogenase, thereby blocking guanine synthesis in the same manner as mycophenolate.
- C.** The CD25 interleukin-2 receptor alpha chain, thereby depleting all activated T cells through complement fixation.
- D.** mTOR signaling, thereby inhibiting growth-factor-driven lymphocyte cell-cycle progression.
- E.** DNA topoisomerase II, thereby producing nonspecific lymphocyte apoptosis through direct cytotoxic chemotherapy.

225. After conversion from a calcineurin inhibitor to sirolimus, a liver recipient develops new hyperlipidemia, painful oral ulcers, proteinuria, and delayed wound healing. Which adverse-effect pattern is most characteristic of the drug class now being used?

- A.** Severe gingival hyperplasia and hirsutism are the defining mTOR toxicities and distinguish the class from all calcineurin inhibitors.
- B.** Profound hyperacute T-cell depletion and cytokine-release syndrome occur with each oral dose because mTOR inhibitors are polyclonal antibodies.
- C.** Isolated cholestasis without metabolic or hematologic effects is expected because mTOR is expressed only in bile-duct epithelial cells.
- D.** Permanent adrenal insufficiency is universal because sirolimus acts by irreversibly blocking the glucocorticoid receptor.
- E.** Dyslipidemia, cytopenias, mouth ulcers, proteinuria, and impaired wound healing are important toxicities.

226. Basiliximab is used for induction in some liver transplant recipients. What is its main target?

- A.** The interleukin-2 receptor alpha chain (CD25) on activated T lymphocytes.
- B.** The calcineurin catalytic subunit inside resting T cells, producing the same intracellular blockade as tacrolimus.
- C.** The CD20 antigen on B cells, producing prolonged B-cell depletion similar to rituximab.

- D.** mTOR complex 1, preventing lymphocyte proliferation through the same pathway as sirolimus.
- E.** The glucocorticoid receptor, producing broad genomic suppression of inflammatory cytokine transcription.

227. A liver transplant recipient receives antithymocyte globulin for severe steroid-resistant rejection. Which complication requires close monitoring during treatment?

- A.** Profound lymphocyte depletion with infusion reactions, cytopenias, and increased opportunistic-infection risk.
- B.** Predictable severe hyperlipidemia without infection risk because antithymocyte globulin acts only on hepatic mTOR signaling.
- C.** Isolated gingival hyperplasia because the antibody binds calcineurin in gingival fibroblasts rather than immune cells.
- D.** Immediate fetal malformations in all adult recipients because the principal toxicity is teratogenicity rather than immune depletion.
- E.** Irreversible portal-vein thrombosis because T-cell depletion directly activates coagulation in every treated liver transplant recipient.

228. How do corticosteroids exert broad immunosuppressive effects after transplantation?

- A.** They selectively inhibit inosine monophosphate dehydrogenase and therefore affect only proliferating B lymphocytes.
- B.** They bind CD25 on activated T cells and prevent interleukin-2 signaling without any genomic effects.
- C.** They inhibit mTOR complex 1 directly and have no effects on innate immune signaling or cytokine transcription.
- D.** They bind intracellular glucocorticoid receptors and alter gene transcription, suppressing multiple inflammatory cytokines and immune-cell pathways.
- E.** They irreversibly deplete circulating T cells by complement-mediated lysis, producing the same mechanism as antithymocyte globulin.

229. A competent potential living donor tells the independent donor advocate in private that she no longer wants to donate but fears disappointing her family. What is the ethically appropriate response?

- A.** Protect the donor's right to withdraw and provide a medically neutral explanation to the family if needed without revealing confidential reasons.
- B.** Tell the family exactly what the donor said because recipient need overrides donor confidentiality during living-donor evaluation.
- C.** Proceed with donation because the donor signed the original consent and cannot ethically withdraw once recipient surgery is being planned.
- D.** Require the donor to confront the recipient directly because the transplant team should not help conceal a voluntary decision to withdraw.
- E.** List the donor as medically unsuitable only if a new physical contraindication can be found, because psychological withdrawal alone is not valid.

230. A transplant program notices that candidates from rural areas have lower listing rates because travel, lodging, and repeated evaluation visits are difficult. Which ethical principle is most relevant?

- A.** Utility requires excluding rural candidates because longer travel makes every rural transplant predictably produce worse graft survival.
- B.** Equity requires identifying and mitigating structural barriers so access is not determined unnecessarily by geography or socioeconomic resources.
- C.** Autonomy requires ignoring structural barriers because only the candidate, not the transplant program, is responsible for transportation and lodging.
- D.** Confidentiality requires the program not to collect data on geography or socioeconomic barriers because such variables can never be used for quality improvement.
- E.** Nonmaleficence requires listing only candidates who can independently fund all travel because financial support creates conflicts of interest.

231. A candidate with alcohol use disorder has engaged in treatment, demonstrates insight, and has strong recovery support. Which ethical approach is most appropriate?

- A.** Assess relapse risk and treatment engagement using the same clinically relevant standards applied to other chronic diseases rather than moral judgment.
- B.** Deny transplantation because self-inflicted disease has lower ethical priority than any condition not influenced by behavior.

- C.** Ignore alcohol use entirely because substance-use history has no possible relationship to medication adherence or recurrent graft injury.
- D.** Require a universal lifetime abstinence guarantee because uncertainty about relapse can be eliminated before transplantation by signed contracts.
- E.** Allocate organs based on whether clinicians personally believe the candidate deserves treatment rather than on medical benefit and transparent criteria.

232. A transplant center invites a candidate to enroll in a clinical trial testing a new preservation strategy. The candidate believes declining will reduce the chance of receiving an organ. What should the research team do?

- A.** Confirm that refusal will lower allocation priority because clinical trials require strong incentives to recruit critically ill transplant candidates.
- B.** Ask the treating surgeon to obtain consent immediately before anesthesia because time pressure improves understanding of complex research tradeoffs.
- C.** Withhold standard-care alternatives because discussing them can discourage enrollment and threaten the scientific validity of the trial.
- D.** Use the transplant listing consent as sufficient research consent because separate disclosure of experimental risks is unnecessary once a patient is listed.
- E.** Make clear that participation is voluntary and that declining research will not reduce access to standard transplant care or allocation priority.

233. A candidate has limited English proficiency and struggles to understand complex transplant instructions. What is the best ethical response?

- A.** Exclude the candidate because inability to communicate in English independently predicts graft loss and cannot be mitigated by interpreters.
- B.** Use minor family members as the default interpreters because this is more efficient and equally accurate for complex informed consent.
- C.** Ask the candidate to sign English-language consent forms without interpretation because written consent is valid regardless of comprehension.
- D.** Transfer all decisions to family members because limited English proficiency automatically removes the candidate's decision-making capacity.

E. Provide qualified language services and accessible education rather than treating language difference itself as evidence of nonadherence.

234. What is the primary role of OPTN policy in United States transplantation?

- A. It establishes operational rules for member transplant hospitals, organ procurement organizations, and histocompatibility laboratories, including allocation and reporting requirements.
- B. It replaces all federal regulation and allows each transplant center to create independent organ-allocation rules without national oversight.
- C. It governs only private insurance reimbursement and has no role in organ matching, data reporting, or transplant-program operations.
- D. It applies only to living donation because deceased-donor allocation is determined solely by individual organ procurement organizations.
- E. It is an optional educational guideline that members may disregard whenever local practice differs from national policy.

235. A transplant program believes a candidate's calculated MELD score substantially underestimates near-term mortality from a recognized complication. What mechanism can address this mismatch?

- A. The center may simply increase the laboratory MELD score in its own medical record because allocation scores are determined locally by transplant programs.
- B. A MELD exception request using standardized policy criteria or National Liver Review Board review when appropriate.
- C. The candidate must wait until the calculated MELD rises because the national system has no mechanism for disease-specific urgency not captured by MELD.
- D. The organ procurement organization can award an informal score increase without documentation because exceptions are outside OPTN oversight.
- E. A living donor must be found because MELD exceptions are permitted only for kidney allocation and not for liver transplantation.

236. Which scenario best illustrates the purpose of a standardized liver exception rather than a discretionary increase in score?

- A.** A candidate requests extra points solely because the local waiting time is long despite having no diagnosis or urgency outside the calculated score.
- B.** A center wants to reward perfect clinic attendance even though adherence is not a diagnosis-specific source of wait-list mortality.
- C.** An organ offer is inconvenient for the surgical team, so the candidate's score is raised temporarily to secure a later donor.
- D.** A candidate has a high calculated MELD already, so the score is lowered to improve donor-recipient size matching and operative scheduling.
- E.** A defined diagnosis meets published policy criteria showing medical urgency not adequately represented by the calculated MELD or PELD score.

237. Why do HCC exception policies incorporate tumor size, number, imaging characteristics, treatment response, and markers of tumor biology rather than relying only on liver synthetic function?

- A.** HCC exception points are intended to measure renal failure because creatinine becomes unreliable in patients with liver tumors.
- B.** Tumor features are used only to predict surgical bleeding and have no relationship to wait-list dropout or post-transplant recurrence.
- C.** Laboratory MELD already measures tumor growth directly, so imaging criteria are included only for billing and documentation purposes.
- D.** The allocation problem is risk of cancer progression beyond transplantability, which is not captured by the laboratory MELD score.
- E.** HCC exceptions exist because all patients with liver cancer have identical mortality and recurrence risk regardless of tumor burden or treatment response.

238. A cirrhotic candidate has severe hepatopulmonary syndrome but a relatively low calculated MELD score. Why can an exception pathway be appropriate?

- A.** Hepatopulmonary syndrome is a cosmetic symptom that does not alter mortality, but exception points are used to shorten travel distance to the transplant center.
- B.** The degree of hypoxemia can confer substantial mortality risk that is not reflected by bilirubin, INR, and creatinine alone.

- C.** The exception compensates for low serum sodium because hypoxemia directly lowers laboratory sodium and artificially reduces MELD.
- D.** All patients with any dyspnea automatically receive identical exception priority without objective documentation of abnormal oxygenation.
- E.** The exception is intended to treat pulmonary arterial hypertension because hepatopulmonary syndrome and portopulmonary hypertension are the same disorder.

239. An organ is offered according to the OPTN match sequence. A program wishes to bypass higher-priority candidates for convenience rather than a policy-defined reason. Which principle applies?

- A.** Centers may reorder offers freely because allocation policy is advisory and convenience is an accepted reason to bypass higher-priority candidates.
- B.** Only organ procurement organizations are subject to allocation rules, so transplant hospitals have no obligation to follow the match sequence.
- C.** A lower-priority candidate may always be chosen if the center believes that person is more deserving, even when policy criteria are not met.
- D.** Match sequences apply only to kidneys and lungs because liver allocation is determined entirely by local surgeon preference.
- E.** Allocation should follow OPTN policy and the match sequence; bypassing candidates outside permitted circumstances undermines fairness and system accountability.

240. A transplant center is worried that accepting medically complex candidates could worsen publicly reported outcomes. What is the ethically and clinically appropriate principle?

- A.** Programs should avoid all high-risk candidates because protecting observed survival metrics is more important than individual transplant benefit.
- B.** Outcome measurement has no role in transplantation because centers should never be held accountable for graft or patient survival.
- C.** Only the sickest candidates should be transplanted because higher mortality automatically improves risk-adjusted program performance.
- D.** Quality metrics should support safety and improvement without creating incentives to deny appropriate high-risk candidates solely to protect program statistics.

E. Programs may alter diagnostic coding to improve reported outcomes because public metrics are administrative rather than patient-safety tools.

Answers

1. **B.** Children with biliary atresia can need transplantation for recurrent cholangitis, portal hypertensive complications, or growth failure even when bilirubin and INR are not profoundly abnormal. Waiting for advanced synthetic failure can worsen nutritional status and transplant risk.
2. **B.** Orthodeoxia and clubbing in chronic liver disease suggest hepatopulmonary syndrome. A contrast echocardiogram showing delayed microbubble appearance in the left heart supports intrapulmonary vascular dilatation; right-heart catheterization is used for suspected portopulmonary hypertension instead.
3. **D.** Cholestatic infants have increased caloric needs and impaired fat and fat-soluble vitamin absorption. Energy-dense feeds, medium-chain triglycerides, vitamin A/D/E/K replacement, and enteral tube supplementation are standard strategies while awaiting transplant.
4. **E.** Several forms of PFIC present with intense pruritus and cholestasis despite low or normal GGT, reflecting canalicular transport defects rather than large-duct obstruction. Alagille syndrome and obstructive cholangiopathies more often produce high-GGT cholestasis.
5. **E.** Wilson disease causing acute liver failure is a transplant emergency. Hemolysis, coagulopathy, encephalopathy, and renal deterioration are ominous; chelation is too slow to substitute for urgent transplant evaluation in this setting.
6. **C.** Alpha-1 antitrypsin is synthesized predominantly by hepatocytes. A donor liver brings the donor SERPINA1 genotype and therefore does not reproduce the recipient's hepatic Z-protein accumulation, although established emphysema may persist.
7. **C.** Selected metabolic liver diseases are transplant indications even without cirrhosis or impaired INR. Replacing the liver supplies functional urea-cycle activity and can prevent recurrent catastrophic hyperammonemia.
8. **A.** Crigler-Najjar type I results from severe absence of bilirubin UDP-glucuronosyltransferase activity. Transplantation replaces the defective hepatic enzyme system and is definitive when phototherapy cannot reliably prevent neurotoxicity.
9. **A.** In erythropoietic protoporphyria, excess protoporphyrin originates from erythroid tissue and is excreted by the liver. Liver transplantation treats hepatic failure but does not remove the hematopoietic source, so recurrence in the graft is possible.

- 10. D.** Glycogen storage disease can be complicated by multiple adenomas. Growth, large size, hemorrhage risk, or concern for malignant transformation can justify surgical management, including transplant evaluation when lesions are multifocal or otherwise unsuitable for resection.
- 11. D.** Variant transthyretin is produced mainly in the liver. Liver transplantation substantially lowers production of the mutant protein, although established disease and deposition of wild-type transthyretin can still influence long-term outcomes.
- 12. E.** Advanced cystic-fibrosis lung disease materially affects perioperative risk and may require combined organ planning. Isolated liver transplantation is inappropriate when pulmonary reserve is insufficient for major surgery.
- 13. E.** Localized Caroli disease may be resectable, but diffuse bilateral disease complicated by recurrent cholangitis and cirrhosis is a classic setting for transplant consideration. Endoscopic or antibiotic therapy cannot remove the diffuse structural abnormality.
- 14. D.** Alagille syndrome combines cholestatic liver disease from bile-duct paucity with characteristic cardiac, skeletal, ocular, and facial features. The GGT is usually elevated, helping distinguish it from low-GGT PFIC disorders.
- 15. E.** Ursodeoxycholic acid is first-line treatment for PBC and improves transplant-free outcomes in responders. Biochemical response is assessed after therapy, with additional agents considered when response is inadequate.
- 16. C.** Biochemical nonresponse to ursodeoxycholic acid is clinically meaningful in PBC. Persistent cholestasis and especially rising bilirubin signal greater risk and should trigger treatment escalation, complication assessment, and attention to transplant timing when disease is advanced.
- 17. D.** Advanced decompensated cirrhosis changes the safety profile of several PBC therapies. In a patient with portal hypertensive decompensation, treatment decisions must account for those restrictions and should not delay appropriate transplant evaluation.
- 18. D.** Autoimmune hepatitis may present acutely or as acute liver failure. Marked hepatocellular enzyme elevation, high IgG, compatible autoantibodies, and exclusion of competing causes support the diagnosis.
- 19. C.** Corticosteroids can benefit selected patients with acute severe autoimmune hepatitis, but nonresponse can be rapid and dangerous. Close monitoring and early transplant-center involvement are essential rather than waiting for prolonged medical failure.
- 20. C.** Autoimmune hepatitis can recur after liver transplantation. Interface hepatitis with plasma cells, compatible serology or IgG changes, and exclusion of rejection and structural graft complications support recurrence.

- 21. D.** PSC is strongly associated with inflammatory bowel disease and classically causes multifocal biliary strictures with intervening normal or dilated segments. MRCP is generally the preferred noninvasive diagnostic study.
- 22. B.** A new clinically significant stricture with worsening cholestasis in PSC raises concern for cholangiocarcinoma as well as benign obstruction. ERCP permits decompression and directed sampling; diagnostic strategy should preserve potential transplant options when hilar cholangiocarcinoma is suspected.
- 23. D.** PSC plus inflammatory bowel disease confers substantially increased colorectal neoplasia risk and warrants close colonoscopic surveillance. The risk persists after liver transplantation and is not replaced by hepatobiliary imaging.
- 24. A.** PSC can cause disabling recurrent cholangitis and other complications that are not captured well by MELD. Transplant candidacy therefore depends on the full clinical course, not only the laboratory score.
- 25. E.** IgG4-related cholangitis often accompanies autoimmune pancreatitis and can mimic PSC or cholangiocarcinoma. The combination of pancreatic findings, elevated IgG4, and compatible biliary strictures should prompt this diagnosis.
- 26. A.** IgG4-related disease is typically steroid responsive. Before treatment, clinicians should establish the diagnosis carefully and exclude malignancy or uncontrolled infection, since both can mimic obstructive disease.
- 27. E.** Hepatic sarcoidosis may lead to portal hypertension through fibrosis and vascular mechanisms even when routine enzymes are not striking. Management follows the severity of complications, and selected advanced cases can require transplantation.
- 28. E.** Autoimmune liver diseases can overlap. Concurrent interface hepatitis and characteristic PBC cholangitic features should prompt an overlap assessment rather than forcing all findings into a single pure phenotype.
- 29. C.** Long-term control of autoimmune hepatitis often uses a steroid-sparing maintenance regimen to minimize corticosteroid toxicity. Complete withdrawal is possible only in carefully selected patients after sustained remission and requires close follow-up.
- 30. D.** Small-duct PSC is characterized by a compatible clinical setting and histology despite a normal cholangiogram. Patients require longitudinal follow-up because some later develop large-duct disease.
- 31. D.** Transplantation treats the liver disease but does not erase the colitis-associated colorectal cancer risk. Ongoing guideline-based surveillance remains important, particularly when inflammatory bowel disease remains active.

- 32. C.** Autoimmune hepatitis classically features interface hepatitis with lymphoplasmacytic inflammation, often including many plasma cells. Periductal fibrosis suggests PSC, whereas florid duct lesions support PBC.
- 33. A.** Cholestatic pruritus is treated symptomatically with a stepwise strategy. Bile-acid sequestrants are commonly used first, followed by other agents such as rifampin or opioid-pathway modulators when needed, while obstruction and drug causes are excluded.
- 34. D.** Bone disease is an important complication of chronic cholestatic liver disease. Fragility fracture or established osteoporosis warrants standard bone-directed treatment plus correction of calcium and vitamin D deficits rather than waiting for transplantation.
- 35. C.** Potent high-barrier nucleos(t)ide analogues are central to HBV management in transplant candidates. Suppressing HBV DNA before transplant reduces recurrence risk and is safer than interferon in decompensated cirrhosis.
- 36. B.** HCV therapy is highly effective, but timing around transplantation is individualized. In some candidates, treatment can improve liver function; in others, post-transplant treatment or acceptance of HCV-viremic organs may better serve transplant access and timing.
- 37. E.** HDV is a defective RNA virus that uses HBsAg for its envelope. Preventing HBV infection prevents HDV acquisition, and suppressing HBV is part of comprehensive management even though nucleos(t)ide analogues do not directly eradicate HDV.
- 38. C.** HEV can become chronic in immunosuppressed transplant recipients. Persistent HEV RNA with otherwise unexplained hepatitis should prompt management that may include careful reduction of immunosuppression and antiviral therapy in selected cases.
- 39. C.** HSV hepatitis can be rapidly fatal and may occur without typical lesions. Empiric IV acyclovir is appropriate when suspicion is high, while transplant-center management proceeds because some patients progress despite antiviral therapy.
- 40. E.** Patients with chronic liver disease should receive indicated HAV and HBV vaccination, ideally before transplant when immune responses are more robust. Preventing superimposed viral hepatitis is particularly important in advanced liver disease.
- 41. A.** Potent HBV therapy reduces hepatic inflammation, decompensation, and HCC risk, but cirrhosis remains a major carcinogenic substrate. Standard HCC surveillance therefore continues despite complete virologic suppression.
- 42. B.** Budd-Chiari management is typically stepwise, beginning with anticoagulation and treatment of the thrombophilic disorder. Angioplasty, TIPS, or transplantation is added when medical therapy does not adequately control disease.

- 43. C.** When anticoagulation and focal venous interventions are insufficient, TIPS can bypass the hepatic outflow obstruction and control portal hypertensive complications. Transplantation is reserved for progressive failure or cases not controlled by less invasive therapy.
- 44. C.** Fontan-associated liver disease results from chronic venous congestion and low-flow physiology. Liver tests can underestimate severity, so evaluation integrates portal hypertensive findings, fibrosis and tumor assessment, and the function of the Fontan circulation when considering isolated versus combined transplantation.
- 45. D.** Congestive hepatopathy reflects passive transmission of elevated right-sided venous pressure to the hepatic veins and sinusoids, producing zone 3 congestion and fibrosis. The primary problem is cardiac hemodynamics rather than a presinusoidal portal process.
- 46. C.** Sinusoidal obstruction syndrome is a toxic endothelial injury of hepatic sinusoids and small venules after transplantation or chemotherapy. Severe disease with organ dysfunction can be treated with defibrotide in addition to meticulous supportive management.
- 47. E.** Severe sickle-cell intrahepatic cholestasis can be life threatening. Exchange transfusion is used to reduce sickling burden, alongside intensive supportive care and transplant-center discussion in refractory hepatic failure.
- 48. E.** Many hepatic abnormalities from cardiac congestion can improve after correction of hemodynamics. Combined transplantation is generally considered when there is convincing advanced, irreversible liver disease or clinically important portal hypertension rather than congestion alone.
- 49. A.** Portosinusoidal vascular disease can produce marked portal hypertension despite absent cirrhosis and relatively preserved synthetic function. Nodular regenerative hyperplasia and portal venopathy are characteristic histologic patterns.
- 50. E.** HVPG reflects sinusoidal pressure and can underestimate presinusoidal portal hypertension. Thus a patient can have major portal hypertensive complications despite a normal or modest HVPG.
- 51. B.** Management of portosinusoidal vascular disease focuses on its portal hypertensive complications. TIPS can be effective in selected patients with refractory bleeding or ascites, while transplantation is reserved for uncommon cases with otherwise unmanageable disease or liver failure.
- 52. A.** Recent extensive portal-vein thrombosis, especially with mesenteric extension or transplant relevance, is a common reason to anticoagulate after individualized bleeding-risk assessment. Chronic cavernomatous thrombosis is managed differently.
- 53. B.** Chronic portal-vein thrombosis can lead to cavernous transformation, in which a network of collateral venous channels develops around the obstructed portal vein. This has important surgical implications for transplantation.

- 54. E.** Portal-vein recanalization combined with TIPS can re-establish portal flow in selected candidates with chronic thrombosis and improve transplant technical options. The decision depends on anatomy and center expertise.
- 55. D.** Splenic-vein thrombosis can cause segmental or left-sided portal hypertension with isolated gastric varices, especially after pancreatitis. The liver and main portal vein may remain normal.
- 56. A.** Large spontaneous portosystemic shunts can drive recurrent encephalopathy. In selected patients with sufficient hepatic reserve, shunt embolization can reduce episodes, although portal hypertension may worsen and must be considered.
- 57. C.** Nonselective beta blockers reduce portal pressure through beta-1 and beta-2 effects and are used in appropriate patients with clinically significant portal hypertension. Carvedilol also has alpha-1 blocking activity and can provide additional portal-pressure reduction.
- 58. E.** Acute variceal bleeding is managed with careful resuscitation, vasoactive therapy, antibiotic prophylaxis, and urgent endoscopic treatment. A restrictive transfusion strategy is generally favored, and selected high-risk patients benefit from early TIPS.
- 59. D.** Selected high-risk patients with acute variceal hemorrhage have improved outcomes with early or preemptive TIPS after initial endoscopic control. The benefit depends on appropriate selection and timing.
- 60. B.** Large-volume paracentesis rapidly relieves symptomatic tense ascites. Albumin is used with larger-volume removal to reduce post-paracentesis circulatory dysfunction.
- 61. A.** Cirrhotic ascites is driven in part by secondary hyperaldosteronism. Spironolactone is therefore foundational therapy, commonly paired with a loop diuretic to improve natriuresis and potassium balance.
- 62. D.** Refractory ascites is a major decompensating event and should prompt transplant evaluation. TIPS can reduce paracentesis requirements in appropriately selected patients without major cardiac, pulmonary, or encephalopathy contraindications.
- 63. D.** An ascitic PMN count of 250/mm³ or more is sufficient to diagnose and treat SBP, even if cultures are negative. Secondary peritonitis should be considered when clinical or fluid features suggest a surgically treatable source.
- 64. A.** A prior episode of SBP confers a high recurrence risk. Secondary prophylaxis is therefore standard while the patient is managed for advanced liver disease and transplant candidacy.
- 65. C.** HRS-AKI is a functional renal failure syndrome in advanced cirrhosis and is diagnosed after addressing volume status and excluding shock, nephrotoxins, obstruction, and intrinsic renal disease. A bland urine sediment supports but does not by itself prove the diagnosis.

- 66. D.** HRS-AKI is treated with splanchnic vasoconstriction plus albumin when appropriate. Terlipressin is an established option, while definitive management still depends on recovery of liver function or transplantation.
- 67. E.** Hepatopulmonary syndrome combines liver disease or portal hypertension, abnormal oxygenation, and intrapulmonary vascular dilatation. Delayed left-sided appearance of agitated-saline microbubbles supports intrapulmonary rather than intracardiac shunting.
- 68. E.** Echocardiography is a screening tool, but right-heart catheterization is needed to confirm pulmonary arterial hemodynamics and assess treatment response. Severity and reversibility are crucial to liver-transplant risk assessment.
- 69. D.** Significant portopulmonary hypertension is a major transplant risk. Pulmonary arterial hypertension therapy can improve hemodynamics in some patients, allowing reassessment for transplantation when targets are achieved.
- 70. A.** Rifaximin reduces recurrence of overt hepatic encephalopathy when added to lactulose in patients with repeated episodes. Precipitating factors and large spontaneous shunts should also be sought when episodes continue.
- 71. E.** Hepatic hydrothorax results from movement of ascitic fluid across diaphragmatic defects. Standard ascites measures are first-line; refractory cases may be bridged with TIPS and should prompt transplant evaluation, while chronic chest tubes carry substantial risk.
- 72. B.** Cirrhosis produces a rebalanced hemostatic state, and INR does not accurately predict procedural bleeding. Diagnostic paracentesis is a low-risk procedure and generally does not require prophylactic correction of these laboratory abnormalities.
- 73. D.** Hyponatremia in advanced cirrhosis reflects impaired free-water excretion from severe circulatory dysfunction and is associated with worse outcomes. Management is cause-directed and correction must be cautious to avoid osmotic demyelination.
- 74. E.** In an appropriate at-risk population, characteristic multiphasic imaging can diagnose HCC noninvasively. Biopsy is reserved for lesions with indeterminate or atypical imaging when tissue diagnosis will change management.
- 75. B.** Conventional Milan criteria include a solitary HCC up to 5 cm or up to three tumors each up to 3 cm, without macrovascular invasion or extrahepatic spread. Additional biologic factors still influence candidacy and recurrence risk.
- 76. A.** Downstaging can bring selected patients into acceptable transplant criteria. Sustained imaging response and favorable tumor-marker behavior help identify tumors with more favorable biology, while progression or vascular invasion is concerning.

- 77. B.** Tumor biology matters in transplant selection. Persistently very high AFP or poor AFP response despite treatment is associated with more aggressive HCC and increased recurrence risk, prompting careful candidacy assessment.
- 78. E.** Bridge therapy aims to maintain transplant eligibility during a meaningful wait by preventing progression. Response also provides information about tumor behavior, but treated patients still require imaging surveillance until transplantation.
- 79. C.** Selected patients with unresectable perihilar cholangiocarcinoma can achieve meaningful outcomes with strict staging, neoadjuvant therapy, and transplantation at experienced centers. Diagnostic procedures must be planned carefully because tumor seeding can affect eligibility.
- 80. A.** Transplantation is an established option for selected children with unresectable hepatoblastoma confined to the liver after appropriate systemic therapy. Extrahepatic disease must be controlled and oncologic staging is essential.
- 81. E.** Hepatic adenomas in men have a greater risk of malignant transformation and are generally resected when feasible. In women, size, subtype, growth, symptoms, and hormonal exposure help guide management.
- 82. B.** Focal nodular hyperplasia is a benign hyperplastic lesion with essentially no malignant potential. Characteristic imaging in an asymptomatic patient usually requires neither biopsy nor treatment.
- 83. E.** A classic hemangioma has characteristic enhancement and is usually managed conservatively when asymptomatic. Biopsy is generally avoided when imaging is diagnostic because of bleeding risk and lack of clinical benefit.
- 84. E.** Hepatic epithelioid hemangioendothelioma is biologically distinct from angiosarcoma. Selected unresectable liver-confined cases can be considered for transplantation after careful staging.
- 85. B.** Transplantation for neuroendocrine liver metastases is reserved for highly selected patients with favorable histology, controlled primary disease, liver-dominant or liver-limited tumor burden, and sustained stability. It is not routine therapy for metastatic disease.
- 86. A.** MELD 3.0 incorporates bilirubin, INR, creatinine, sodium, and albumin, together with model-specific demographic adjustment and interaction terms. Aminotransferases, ammonia, and tumor markers are not direct components of the score.
- 87. C.** Frailty and sarcopenia are important predictors of wait-list and post-transplant outcomes that are not fully captured by MELD. Prehabilitation, nutrition, and treatment of reversible contributors can be incorporated into candidacy planning.
- 88. C.** Active uncontrolled infection is a major contraindication to immediate transplantation. Once infection is adequately treated and the patient is stabilized, candidacy can be reassessed rather than permanently denied.

- 89. A.** Patients with well-controlled HIV can achieve good transplant outcomes. Evaluation includes viral control, immune status, opportunistic-infection history, malignancy risk, and antiretroviral-immunosuppressant interactions.
- 90. A.** The purpose of simultaneous liver-kidney transplantation is to address kidney disease unlikely to recover after liver transplant alone. Candidacy depends on the severity and chronicity of renal dysfunction and relevant allocation criteria, not on transient creatinine changes alone.
- 91. E.** Living donation must be voluntary and free from coercion. Independent donor evaluation protects the donor's interests and may require pausing or ending donation without disclosing private reasons to the recipient.
- 92. B.** Transplant programs must evaluate adherence risk while distinguishing modifiable structural barriers from persistent behavior. Addressing housing, medication access, transportation, literacy, and support can improve candidacy and promotes equitable assessment.
- 93. B.** N-acetylcysteine is indicated in suspected acetaminophen hepatotoxicity even when a late serum level is low or undetectable. Benefit is greatest early, but treatment should not be delayed when hepatic injury is present.
- 94. C.** Acute liver failure can deteriorate rapidly, and transplantation may be lifesaving when prognostic features predict poor spontaneous survival. Trends in lactate, acidosis, encephalopathy, coagulopathy, and organ failure are more important than aminotransferase decline alone.
- 95. A.** N-acetylcysteine may improve outcomes in selected early non-acetaminophen acute liver failure and is often used because of its safety profile. It complements, rather than replaces, etiology-specific therapy and transplant evaluation.
- 96. B.** Amatoxin poisoning classically causes delayed severe gastrointestinal illness, a deceptive latent phase, and then hepatic necrosis with possible acute liver failure. Early poison-center and transplant-center involvement is essential.
- 97. A.** Acute fatty liver of pregnancy can cause rapidly progressive maternal liver failure. Delivery is the cornerstone of therapy, with aggressive supportive management; transplantation is rarely required when hepatic failure does not recover.
- 98. E.** HLH can present with fever, cytopenias, splenomegaly, extreme hyperferritinemia, and multiorgan dysfunction including acute liver failure. Recognition matters because management targets the hyperinflammatory syndrome while transplant decisions remain individualized.
- 99. B.** Hypoxic hepatitis follows severe cardiac, respiratory, or circulatory failure and can produce dramatic aminotransferase elevations. Treatment focuses on correcting the underlying hemodynamic cause rather than a liver-specific antidote.

100. D. Indeterminate acute liver failure may have poor spontaneous survival, especially with advanced encephalopathy and multiorgan failure. Early transplant-center management and timely listing are critical when recovery appears unlikely.

101. B. Wilson acute liver failure often has a distinctive combination of hemolysis, severe coagulopathy, renal dysfunction, and relatively low alkaline phosphatase compared with bilirubin. It is a transplant emergency.

102. A. In autoimmune acute liver failure, a short trial of corticosteroids may be reasonable in selected patients, but worsening coagulopathy or encephalopathy should trigger prompt transplantation rather than prolonged ineffective immunosuppression.

103. E. The Lille model is used after several days of corticosteroid therapy to identify nonresponders with poor prognosis who are unlikely to benefit from continued steroids. It complements baseline severity assessment rather than replacing it.

104. A. Selected patients with severe alcohol-associated hepatitis and high predicted mortality may undergo early transplantation without a rigid universal abstinence period. Careful psychosocial assessment of relapse risk and commitment to long-term addiction treatment is essential.

105. A. Malnutrition is common and prognostically important in alcohol-associated hepatitis. Adequate energy and protein intake is recommended, with enteral support when needed; unnecessary protein restriction worsens sarcopenia.

106. D. Modern transplant evaluation emphasizes individualized relapse-risk assessment and addiction treatment rather than a single arbitrary time threshold. Multiple psychosocial, behavioral, and clinical variables inform risk and support planning.

107. D. Alcohol relapse requires active addiction treatment and assessment for graft injury, adherence problems, and psychosocial stressors. A chronic-disease approach improves safety and avoids delaying care until irreversible recurrent liver disease develops.

108. A. Alcohol and metabolic risk factors can synergistically worsen steatosis, inflammation, fibrosis, and liver-related outcomes. Clinical assessment should quantify both rather than assigning all risk to a single category.

109. B. Fibrosis stage is the major histologic predictor of liver-related outcomes in MASLD. Aminotransferases and steatosis burden can fluctuate and do not substitute for fibrosis-risk assessment.

110. D. Risk stratification for MASLD often uses a staged noninvasive approach. A high initial score should prompt more specific testing such as elastography rather than relying on aminotransferase magnitude alone.

- 111. C.** MASH transplant candidates often have diabetes, obesity, dyslipidemia, and cardiovascular disease. Careful cardiovascular risk assessment is essential because cardiac events are an important cause of perioperative and long-term morbidity.
- 112. C.** Management of MASH cirrhosis combines cirrhosis care with aggressive treatment of metabolic disease. Weight loss should preserve muscle mass, and HCC and portal-hypertension surveillance continue despite metabolic improvement.
- 113. A.** Aminotransferases can be normal in advanced MASLD. Fibrosis stage, imaging, portal hypertensive findings, and clinical outcomes are more informative than a single ALT value.
- 114. D.** Sarcopenic obesity combines excess adiposity with low muscle mass and functional reserve. Prehabilitation should focus on adequate protein, safe activity, and preservation or restoration of muscle rather than aggressive weight loss that worsens frailty.
- 115. C.** The metabolic drivers of steatotic liver disease often persist or worsen after transplantation. Weight gain, diabetes, dyslipidemia, and immunosuppressive effects can lead to recurrent or de novo graft steatosis and fibrosis.
- 116. B.** Bariatric surgery around transplantation is complex. Compensated disease, portal pressure, transplant timing, nutritional consequences, and center expertise all influence whether surgery is performed before, during, or after transplantation.
- 117. B.** Metabolic therapies can improve weight, glycemia, and steatotic liver disease, but established fibrosis and cardiovascular risk require ongoing management. Cirrhosis-specific surveillance continues unless regression is convincingly established by appropriate longitudinal assessment.
- 118. A.** HELLP is a severe hypertensive pregnancy complication. Maternal stabilization and delivery are central; liver transplantation is rarely needed unless catastrophic hepatic failure or irreversible injury occurs.
- 119. C.** Intrahepatic cholestasis of pregnancy typically presents with pruritus and elevated bile acids in late pregnancy. It is distinct from acute fatty liver of pregnancy and HELLP, which produce more severe systemic and synthetic abnormalities.
- 120. C.** The combination of substantial hepatocellular injury and jaundice is a classic warning pattern for serious DILI. The suspected drug should be stopped when appropriate, alternative causes excluded, and the patient monitored for worsening coagulopathy or encephalopathy.
- 121. B.** Once DILI progresses to acute liver failure, management follows acute liver failure principles. Early transplant-center involvement is essential because deterioration can be rapid and no specific antidote exists for most idiosyncratic reactions.

- 122. C.** ACLF is characterized by acute decompensation with systemic inflammation and organ failure. Short-term mortality rises sharply with increasing number and severity of organ failures, making early intensive care and transplant assessment important.
- 123. A.** ACLF management includes rapid treatment of triggers, organ support, and early transplant assessment. Some patients can recover or be transplanted successfully, but the trajectory can deteriorate quickly, so delayed referral may lose the opportunity.
- 124. B.** Palliative care can be integrated with transplant evaluation and active treatment. It addresses symptoms, caregiver burden, communication, advance care planning, and uncertainty without requiring abandonment of transplantation.
- 125. C.** Advance care planning should reflect the patient's goals and expected course. When transplantation is not an option and the patient prioritizes comfort and avoiding hospitalization, hospice may be appropriate alongside thoughtful symptom treatment.
- 126. B.** Symptom burden in cirrhosis can be substantial and poorly reflected by MELD. Palliative principles support active treatment of pruritus, cramps, sleep disturbance, pain, and psychological distress while avoiding medications that worsen encephalopathy or renal function.
- 127. E.** Advance care planning documents goals and identifies who should speak for the patient if capacity is lost. It is especially useful in serious illness with unpredictable deterioration and can coexist with full transplant intent.
- 128. A.** Pretransplant evaluation is an important opportunity to update immunizations. Inactivated vaccines are generally safe, and pretransplant administration often produces better immune responses than vaccination during intense post-transplant immunosuppression.
- 129. C.** Bone loss and fracture risk are common before and after transplantation. Evaluation and treatment should begin before transplant when possible, including correction of vitamin deficiencies and standard osteoporosis therapy when appropriate.
- 130. C.** Transplant evaluation includes age- and risk-appropriate cancer screening because active or recently treated malignancy can change candidacy and because immunosuppression can amplify future cancer risk.
- 131. B.** Fertility can improve after transplantation, making proactive reproductive counseling important. Pregnancy is often possible after stable graft function is established, but timing and medication selection require coordinated specialist planning.
- 132. A.** Cirrhosis promotes accelerated starvation physiology and muscle loss. Adequate protein, sufficient energy, frequent intake, and a late-evening snack help preserve lean mass and functional reserve.

- 133. C.** Substantial macrovesicular steatosis increases vulnerability to preservation and reperfusion injury. Organ acceptance therefore integrates steatosis with donor age, ischemia time, recipient severity, and center experience rather than applying a single isolated variable.
- 134. C.** DCD grafts have improved substantially with modern practice, but warm ischemic injury can increase the risk of ischemic-type biliary complications. Donor, procurement, preservation, and recipient factors all contribute to risk.
- 135. E.** Highly effective direct-acting antivirals have enabled use of HCV-viremic organs in selected HCV-negative recipients. Programs must ensure informed consent, post-transplant testing, medication access, and prompt antiviral treatment.
- 136. E.** Anti-HBc-positive donor livers can harbor latent HBV and transmit infection, especially to nonimmune recipients. Modern antiviral prophylaxis and assessment of recipient HBV immunity allow many such grafts to be used safely.
- 137. D.** Machine perfusion preserves the organ in a more physiologic state than static cold storage and can permit viability assessment, treatment, and potentially safer use of higher-risk grafts. It does not remove immunologic or infectious risk.
- 138. E.** Normothermic perfusion creates an opportunity to observe graft metabolism and hemodynamics dynamically. Viability decisions use multiple parameters rather than a single laboratory or visual finding.
- 139. C.** Donor risk factors interact with ischemia and recipient illness. Thoughtful donor-recipient matching aims to avoid compounding several high-risk features while preserving access to transplantation.
- 140. D.** Living-donor transplantation exposes a healthy person to major surgery, so donor safety is paramount. Inadequate predicted remnant volume is a reason to reject the planned graft or pursue an alternative strategy.
- 141. B.** A graft that is too small can face excessive portal inflow relative to mass, leading to sinusoidal injury, poor synthetic function, cholestasis, and ascites. Graft size and recipient physiology are therefore central to living-donor planning.
- 142. E.** Living-donor evaluation requires detailed anatomic mapping and volumetry to plan safe resection and implantation. Additional tests such as biopsy are used selectively rather than replacing imaging.
- 143. A.** Living donation can be lifesaving for the recipient but exposes a healthy donor to major abdominal surgery. Ethical consent requires clear discussion of short- and long-term complications, uncertainty, alternatives, and the right to withdraw.

- 144. E.** Living-donor steatosis affects both donor and graft safety. Mild reversible steatosis may improve with metabolic intervention, after which anatomy and liver quality can be reassessed rather than applying an automatic permanent exclusion.
- 145. D.** Split-liver transplantation can expand the donor pool by using one liver for two recipients, often an adult and a child. Success depends on suitable donor anatomy and quality, appropriate graft sizing, and surgical expertise.
- 146. B.** Hepatic arterial variants are common and must be recognized during procurement and implantation. Bile ducts depend heavily on arterial blood flow after transplantation, so arterial compromise can produce major biliary complications.
- 147. B.** The biliary tree is particularly vulnerable to arterial injury because its microcirculation is supplied largely by branches of the hepatic artery. Arterial thrombosis or severe stenosis can therefore cause biliary necrosis and nonanastomotic strictures.
- 148. A.** ABO-incompatible liver transplantation can be lifesaving in selected settings but carries increased antibody-mediated and biliary risk. Modern protocols may use B-cell depletion, antibody removal, and careful immunosuppression to mitigate that risk.
- 149. D.** In urgent settings, particularly pediatrics, ABO-incompatible liver transplantation may be appropriate when a compatible graft is unavailable. Outcomes depend on center expertise and strategies to control antibody-mediated injury.
- 150. A.** Primary nonfunction is catastrophic failure of the graft to sustain metabolic and synthetic function after implantation, after excluding correctable vascular causes. Urgent retransplantation is often required.
- 151. C.** Early allograft dysfunction is a recognized early postoperative syndrome defined by impaired biochemical and synthetic recovery. It is distinct from primary nonfunction, which is more severe and often requires emergency retransplantation.
- 152. E.** Early hepatic-artery thrombosis is a surgical emergency. The biliary tree is especially vulnerable to arterial ischemia, and rapid diagnosis can determine whether revascularization is possible or retransplantation is needed.
- 153. A.** Early portal-vein thrombosis can compromise graft inflow and cause acute dysfunction or portal hypertensive complications. Doppler is an important screening test, followed by urgent confirmatory imaging and intervention when needed.
- 154. A.** Small-for-size syndrome reflects a mismatch between graft mass and recipient demand, often amplified by excessive portal inflow. Sinusoidal stress and impaired arterial buffering contribute to graft dysfunction.

- 155. B.** CMV donor-positive/recipient-negative status is a high-risk combination. Prevention generally uses valganciclovir-based prophylaxis or a carefully monitored preemptive approach depending on organ and center protocol.
- 156. E.** Trimethoprim-sulfamethoxazole is preferred PJP prophylaxis after solid-organ transplantation when tolerated. Alternative agents are used for allergy, cytopenias, or other contraindications.
- 157. D.** Complex surgery, reoperation, heavy transfusion, renal replacement, and other markers of critical illness increase invasive fungal risk. Many centers use risk-targeted rather than universal systemic antifungal prophylaxis.
- 158. D.** Modern HBV recurrence prevention relies on potent nucleos(t)ide analogue therapy. Hepatitis B immune globulin use varies by virologic risk and center protocol, but stopping antiviral suppression after transplant can permit recurrence.
- 159. C.** Tacrolimus and cyclosporine can cause neurotoxicity, including posterior reversible encephalopathy syndrome, especially with hypertension or high exposure. Management includes blood-pressure control and adjustment of the offending immunosuppressant.
- 160. B.** Calcineurin inhibitors can cause dose-related renal vasoconstriction and chronic nephrotoxicity. Management often reduces exposure, addresses drug interactions, and uses adjunctive immunosuppressants to preserve rejection protection.
- 161. C.** Donor-derived HCV is expected when an HCV-viremic organ is used. Modern direct-acting antivirals are highly effective, so programs arrange prompt treatment and monitor for medication interactions and hepatic recovery.
- 162. A.** Strongyloides may persist for decades and can cause lethal hyperinfection under immunosuppression. Screening based on epidemiologic risk and appropriate ivermectin treatment can allow safe organ use in many circumstances.
- 163. C.** Unexpected donor culture results require rapid communication and organism-specific risk assessment. Some findings represent contamination, while others require targeted recipient therapy or surveillance to prevent donor-derived infection.
- 164. E.** Clusters of severe unexplained illness in multiple recipients from one donor should trigger concern for donor-derived infection and immediate notification of transplant and public-health networks. Rapid coordination can identify the pathogen and protect other recipients.
- 165. D.** Certain donor malignancies, including melanoma, can recur after long latency and have high transmission consequences. Donor cancer history must be evaluated carefully even when treatment was remote.

- 166. E.** Potential donor-derived disease requires rapid communication across all affected transplant programs and oversight systems. Management depends on tumor type, organ, transmission risk, recipient status, and feasibility of graft removal or oncologic therapy.
- 167. B.** Acute T-cell-mediated rejection is diagnosed histologically, classically with portal inflammation, bile-duct damage, and venous endopheliitis. Structural vascular and biliary causes should be excluded because liver-test patterns alone are not specific.
- 168. C.** Most acute T-cell-mediated rejection responds to intensified immunosuppression, often corticosteroids plus adjustment of baseline therapy. Steroid-resistant or severe cases require additional immunosuppressive strategies after diagnostic reassessment.
- 169. B.** Antibody-mediated rejection is less common in liver than in some other organs but can occur. Diagnosis integrates graft dysfunction and histology with donor-specific antibodies and supportive complement or microvascular findings.
- 170. D.** Treatment of significant antibody-mediated rejection is individualized and may include plasmapheresis, IVIG, B-cell or plasma-cell-directed therapy, and adjustment of maintenance immunosuppression. Diagnosis should be secure because therapies carry substantial risk.
- 171. E.** Chronic rejection classically causes progressive cholestatic dysfunction, ductopenia, and chronic vascular injury. Early cases may respond to immunosuppressive optimization, whereas advanced duct loss can be irreversible.
- 172. A.** Chronic rejection is associated with ongoing alloimmune injury, including recurrent rejection and inadequate immunosuppressive exposure. Medication adherence and correct interpretation of prior graft dysfunction are therefore important preventive issues.
- 173. E.** Plasma cell-rich rejection is a late graft hepatitis characterized by plasma-cell-rich inflammation and alloimmune features. It overlaps histologically with autoimmune hepatitis, so the recipient's original diagnosis and broader immune evaluation matter.
- 174. D.** Liver-transplant GVHD usually occurs weeks after transplantation and classically presents with fever, rash, diarrhea, and bone-marrow failure. Donor lymphocytes attack recipient tissues, making the syndrome biologically opposite to conventional rejection.
- 175. E.** GVHD occurs when viable donor lymphocytes recognize recipient antigens and attack recipient skin, gut, and marrow. Conventional rejection is recipient immune recognition of donor tissue.
- 176. D.** Treatment of rejection must be paired with correction of the cause of under-immunosuppression. Financial access, health literacy, transportation, mental health, and regimen complexity are modifiable contributors to adherence and graft outcomes.

177. D. Anastomotic strictures are common biliary complications after liver transplantation and usually present with cholestatic tests and focal narrowing at the biliary anastomosis. Endoscopic therapy is often effective in duct-to-duct reconstructions.

178. A. Most duct-to-duct anastomotic strictures are approached endoscopically with balloon dilation and stent therapy. Surgery or retransplantation is reserved for refractory complex disease.

179. B. Ischemic cholangiopathy produces diffuse nonanastomotic strictures, casts, and recurrent cholangitis, particularly after ischemic injury such as DCD procurement. Main arterial patency does not exclude prior microvascular biliary ischemia.

180. B. Portal-vein stenosis can produce portal hypertension, ascites, splenomegaly, or graft dysfunction. Doppler screening followed by cross-sectional or interventional imaging helps confirm the lesion and guide angioplasty or stenting.

181. D. Hepatic-artery stenosis threatens the sole arterial supply of the transplanted biliary tree and can progress to thrombosis. Timely angioplasty, stenting, or surgical revision may preserve graft and biliary function.

182. A. Post-transplant diabetes is common and reflects both recipient metabolic risk and medication effects, particularly corticosteroids and calcineurin inhibitors. It contributes to cardiovascular and graft risk and should be treated actively.

183. D. Metabolic syndrome is a major long-term complication after liver transplantation and increases cardiovascular, renal, and graft risk. Management includes weight, blood pressure, glycemic, lipid, and medication strategies.

184. A. Chronic kidney disease is a major determinant of long-term outcomes after liver transplantation. Calcineurin-inhibitor exposure, diabetes, hypertension, and pretransplant renal injury all contribute.

185. E. Renal-sparing approaches may use lower calcineurin exposure with adjunctive agents such as mycophenolate or mTOR inhibitors in appropriate recipients. Changes must balance renal benefit against rejection and drug-specific adverse effects.

186. B. Bone loss may persist or worsen early after transplant, especially with corticosteroids and preexisting hepatic osteodystrophy. Fragility fracture warrants active osteoporosis treatment and risk-factor modification.

187. C. Sarcopenia may not resolve automatically after transplantation. Rehabilitation and adequate nutrition are central to recovery of muscle mass, function, and quality of life.

188. B. As transplant survival improves, cardiovascular disease becomes a major long-term threat. Weight, diabetes, blood pressure, lipids, renal function, smoking, and exercise therefore require active management.

- 189. E.** PTLD ranges from reactive EBV-driven proliferation to aggressive lymphoma. Risk is highest with intense immunosuppression and primary EBV infection in a previously seronegative recipient.
- 190. B.** Management of PTLD starts with defining histology and disease extent. Reduction of immunosuppression can restore immune control, and many B-cell PTLDs require rituximab with or without chemotherapy depending on response and severity.
- 191. C.** Immunosuppression increases skin-cancer incidence and can make tumors more aggressive. Sun protection, regular skin examination, and early treatment are important parts of long-term transplant care.
- 192. E.** Statins are often appropriate after liver transplantation, but drug-drug interactions and myopathy risk vary by agent and immunosuppressant. Cardiovascular prevention should not be neglected solely because the patient is a transplant recipient.
- 193. D.** Hypertension is common after transplantation due to preexisting risk and medication effects. It contributes to cardiovascular and renal disease and should be actively treated with attention to kidney function and immunosuppressant interactions.
- 194. E.** CMV can cause a febrile leukopenic syndrome and tissue-invasive disease affecting the graft, gut, lungs, or other organs. Viral load supports the diagnosis, but tissue confirmation may be required for end-organ disease.
- 195. B.** Ganciclovir-class therapy is standard for CMV disease, with renal dose adjustment and blood-count monitoring. Immunosuppression is often reduced when safe because excessive immune suppression contributes to uncontrolled viral replication.
- 196. D.** Structural biliary disease predisposes to ascending bacterial infection after liver transplantation. Management requires appropriate antibiotics plus relief of the obstruction when feasible.
- 197. A.** Intense immunosuppression increases invasive mold risk. *Aspergillus* can invade pulmonary vessels, causing nodules, infarction, cavitation, and hemoptysis; rapid diagnostic testing and mold-active therapy are important.
- 198. C.** Solid-organ transplant recipients can develop chronic HEV infection, which may progress to fibrosis. Persistent HEV RNA rather than serology alone is important in immunosuppressed patients.
- 199. B.** Initial management often includes cautious reduction of immunosuppression to permit viral clearance. Ribavirin is used for persistent chronic infection in selected transplant recipients, with monitoring for anemia and response.
- 200. D.** Transplant recipients are vulnerable to severe respiratory viral infection. Early antiviral treatment, supportive care, and infection-control measures are appropriate; live vaccines are generally avoided in immunosuppressed recipients.

- 201. E.** PBC can recur after liver transplantation, usually over years. Diagnosis requires exclusion of biliary obstruction and rejection and relies on compatible histology and clinical context.
- 202. D.** PSC can recur in the graft, but biliary strictures after transplantation also have ischemic, anastomotic, and infectious causes. Diagnosis therefore requires careful structural and clinical evaluation.
- 203. A.** Autoimmune hepatitis may recur after liver transplantation, especially with reduced immunosuppression. Compatible histology and IgG elevation support recurrence, while rejection and viral hepatitis remain important alternatives.
- 204. E.** Explant pathology can reveal tumor biology not fully captured before transplant. Microvascular invasion, poor differentiation, and high tumor burden are associated with higher post-transplant HCC recurrence risk and can inform surveillance intensity.
- 205. D.** The metabolic drivers of MASH remain active after transplantation and may be amplified by immunosuppression. Weight, diabetes, lipids, blood pressure, and fibrosis risk all require longitudinal management.
- 206. C.** Post-transplant alcohol use is clinically important even before abnormal graft tests appear. Early addiction treatment and assessment of alcohol-related graft injury can reduce progression and address associated adherence or psychosocial problems.
- 207. D.** HBV can recur in the graft if viral suppression is interrupted. Potent nucleos(t)ide analogue therapy is central, with attention to adherence, prior resistance, HBV serology, and graft injury.
- 208. C.** Vaccination remains an important preventive strategy after transplantation. Inactivated vaccines are generally safe, whereas live vaccines are usually avoided during significant immunosuppression because of risk of vaccine-derived infection.
- 209. B.** Transplant recipients have increased risk for multiple malignancies, including skin cancer and virus-associated tumors. Screening should follow transplant-specific and age-appropriate recommendations rather than being reduced after surgery.
- 210. B.** Pregnancy after liver transplantation is often possible when graft function is stable, but mycophenolate is strongly teratogenic and must be discontinued with an appropriate washout and replacement plan. Immunosuppression should not simply be stopped.
- 211. E.** Adolescents and young adults are vulnerable to medication nonadherence and care discontinuity. Structured transition programs build self-management skills and provide overlap between pediatric and adult systems.
- 212. B.** Bone health remains important after transplantation because preexisting osteodystrophy, corticosteroids, renal disease, and inactivity increase fracture risk. Prevention and treatment follow standard principles adapted to the recipient's comorbidities.

213. B. Cardiovascular disease is a leading long-term threat after liver transplantation. Conventional risk factors remain important and are often compounded by immunosuppressive therapy.

214. E. Transplant success includes survival, graft function, and quality of life. Mood disorders, sexual health, fatigue, cognition, employment, and caregiver strain may persist and should be addressed directly.

215. D. Donor-specific antibodies recognize HLA epitopes present in the donor and can contribute to antibody-mediated rejection. Their significance depends on strength, persistence, complement activity, histology, and the clinical setting.

216. C. The liver has unique immunobiology that can promote immune regulation and allows some recipients to tolerate lower immunosuppressive burdens than recipients of other organs. This does not eliminate T-cell- or antibody-mediated rejection.

217. C. Tacrolimus forms a complex with FKBP that inhibits calcineurin phosphatase activity. This prevents NFAT-dependent transcription of IL-2 and other cytokines needed for T-cell activation.

218. A. Tacrolimus can cause renal vasoconstriction and chronic nephrotoxicity, neurologic effects such as tremor or seizures, and post-transplant diabetes. Drug exposure and interactions should be reviewed when these toxicities appear.

219. D. Tacrolimus is metabolized by CYP3A enzymes and transported by P-glycoprotein. Azole antifungals can markedly increase tacrolimus concentrations, requiring preemptive dose adjustment and close therapeutic-drug monitoring.

220. E. Rifampin is a potent inducer of CYP3A and related pathways and can substantially reduce calcineurin-inhibitor exposure. Alternative antimicrobials are often preferred when possible; otherwise intensive drug-level adjustment is required.

221. B. Mycophenolate inhibits IMP dehydrogenase, limiting de novo guanosine synthesis. Activated T and B lymphocytes rely heavily on this pathway, making the drug an effective antimetabolite immunosuppressant.

222. E. Mycophenolate exposure during pregnancy is associated with pregnancy loss and characteristic congenital malformations. Reproductive counseling and planned conversion to a safer regimen are essential before conception.

223. A. Azathioprine is inactivated in part through xanthine oxidase. Allopurinol or febuxostat can raise active thiopurine exposure dramatically, so combination therapy requires major dose reduction and close blood-count monitoring.

224. D. Sirolimus and everolimus bind FKBP but inhibit mTOR rather than calcineurin. The result is impaired cellular response to growth signals and reduced lymphocyte proliferation.

- 225. E.** mTOR inhibitors have a distinct toxicity profile that includes dyslipidemia, cytopenias, oral ulcers, proteinuria, edema, pneumonitis, and impaired wound healing. These effects influence timing and recipient selection.
- 226. A.** Basiliximab is a monoclonal antibody directed at CD25, the alpha chain of the IL-2 receptor on activated T cells. It is nondepleting and can facilitate delayed or reduced calcineurin-inhibitor exposure in selected recipients.
- 227. A.** Antithymocyte globulin is a potent T-cell-depleting polyclonal antibody preparation. Cytokine-related infusion reactions, leukopenia, thrombocytopenia, and infections are important treatment risks.
- 228. D.** Glucocorticoids act through intracellular receptors that modify transcription of numerous inflammatory and metabolic genes. Their broad effects explain both their usefulness for rejection and their substantial infectious, metabolic, bone, and neuropsychiatric toxicity.
- 229. A.** Living donation must remain voluntary throughout evaluation. Donors may withdraw without penalty, and programs should protect confidentiality so that family pressure does not undermine autonomy.
- 230. B.** Equitable transplant evaluation distinguishes medically relevant risk from remediable barriers such as transportation, lodging, language, and financial access. Programs should avoid turning social disadvantage itself into an unnecessary exclusion criterion.
- 231. A.** Substance use disorders are chronic medical conditions. Ethical transplant assessment focuses on prognosis, relapse risk, treatment engagement, support, and the ability to protect the graft, not moral blame for disease causation.
- 232. E.** Valid research consent requires voluntariness, understanding, and freedom from undue influence. Candidates must know that declining a trial does not jeopardize ordinary transplant evaluation, allocation, or clinical care.
- 233. E.** Language access is part of informed consent and equitable care. Qualified interpretation and tailored education can address communication barriers without wrongly equating language difference with incapacity or nonadherence.
- 234. A.** OPTN policies are binding operational rules for OPTN members and address allocation, donor and recipient safety, data, and other transplant-system requirements. They operate within the federal statutory and regulatory framework rather than replacing it.
- 235. B.** Exception pathways exist because calculated MELD or PELD does not capture every source of wait-list mortality. Standardized exceptions and National Liver Review Board processes aim to improve consistency and equity.

236. E. Standardized exceptions are designed for specific clinical conditions in which calculated laboratory scores do not reflect true transplant urgency. They use transparent criteria rather than ad hoc local preference.

237. D. HCC creates a different source of transplant urgency: progression can make a patient no longer transplantable even when bilirubin, INR, and creatinine remain favorable. Allocation policy therefore considers both tumor burden and biology.

238. B. Hepatopulmonary syndrome can cause progressive severe hypoxemia and mortality while laboratory MELD remains modest. Standardized criteria use objective oxygenation and diagnostic evidence to identify candidates whose urgency is underestimated.

239. E. National allocation depends on transparent rules and sequential offers to protect fairness and public trust. Deviations outside policy-defined circumstances require scrutiny because they can deprive higher-priority candidates of an opportunity.

240. D. Transplant outcomes should be monitored for quality and safety, but metrics can create unintended risk-avoidance incentives. Appropriate risk adjustment and thoughtful oversight aim to protect patients without discouraging justified transplantation of complex candidates.