COMPREHENSIVE MOCK EXAM 2

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 7-month-old infant with biliary atresia underwent Kasai portoenterostomy at 7 weeks. She now has recurrent cholangitis, progressive jaundice, splenomegaly, and growth failure despite aggressive nutritional support. Which management is most appropriate?

A. Repeat the Kasai portoenterostomy because recurrent cholangitis usually reflects a surgically correctable anastomotic obstruction.

B. Refer for liver transplantation evaluation now because progressive portal hypertension and growth failure indicate failing native-liver function.

C. Defer transplant evaluation until synthetic dysfunction develops because bilirubin and growth alone do not predict native-liver failure.

D. Start long-term corticosteroids because post-Kasai cholestasis is primarily an immune-mediated inflammatory process.

E. Perform elective TIPS because decompression of portal hypertension is the preferred bridge to transplant in infants with biliary atresia.

2. A 3-year-old child with biliary atresia has splenomegaly, thrombocytopenia, and intermittent hematemesis. Bilirubin is 1.1 mg/dL and INR is normal. Which finding most strongly supports referral for transplant evaluation despite preserved synthetic function?

A. A normal serum bilirubin after Kasai portoenterostomy despite persistent splenomegaly.

B. Isolated thrombocytopenia without bleeding, ascites, cholangitis, or impaired growth.

C. Mildly elevated gamma-glutamyl transferase with otherwise stable liver tests and growth.

D. Recurrent portal hypertensive bleeding that cannot be durably controlled with standard endoscopic therapy.

E. A single small esophageal varix found on screening endoscopy without prior hemorrhage.

3. An infant with biliary atresia is being assessed for liver transplantation. Which nutritional strategy is most appropriate while awaiting transplant?

A. Provide energy-dense nutrition with supplemental medium-chain triglycerides and fat-soluble vitamins, using enteral tube feeding when oral intake is inadequate.

B. Restrict protein to prevent encephalopathy and provide most calories as simple carbohydrates until transplantation.

C. Use a low-fat diet without vitamin supplementation because cholestasis impairs absorption of long-chain fats.

D. Avoid overnight enteral feeding because continuous nutrition increases the risk of aspiration and cholangitis.

E. Limit total calories to avoid hepatomegaly because excess weight increases technical difficulty at transplantation.

4. A 21-year-old with tremor, dysarthria, Coombs-negative hemolytic anemia, bilirubin 18 mg/dL, alkaline phosphatase 38 U/L, INR 3.1, and new confusion has no prior liver disease. Which next step is most appropriate?

A. Urgently transfer for liver transplantation because Wilson disease presenting as acute liver failure has a very poor prognosis without transplantation.

B. Begin oral zinc alone and reassess neurologic symptoms after several weeks before considering transplantation.

C. Start D-penicillamine and defer transplant referral until 24-hour urinary copper results are available.

D. Perform percutaneous liver biopsy to quantify hepatic copper before making decisions about transplantation.

E. Give high-dose corticosteroids because hemolysis with acute liver failure is most consistent with autoimmune hepatitis.

5. A 34-year-old with Wilson disease and compensated cirrhosis has stable liver tests on trientine but progressive dystonia despite neurologic therapy. Which statement best describes the role of liver transplantation?

A. Liver transplantation is contraindicated when neurologic manifestations predominate because neurologic symptoms invariably worsen after transplant.

B. Transplantation does not alter copper metabolism and therefore has no disease-specific role in Wilson disease.

C. Transplantation is reserved only for acute liver failure and cannot be considered for chronic Wilson-related cirrhosis.

D. Transplantation is indicated solely when serum ceruloplasmin becomes undetectable despite adequate chelation therapy.

E. Liver transplantation corrects the hepatic copper transport defect and may be considered for otherwise intractable neurologic disease after multidisciplinary assessment.

6. A 46-year-old with emphysema and cirrhosis has alpha-1 antitrypsin phenotype PiZZ. Which statement about liver transplantation is most accurate?

A. Liver transplantation corrects both hepatic and established pulmonary structural disease, so lung assessment is unnecessary before transplant.

B. Alpha-1 antitrypsin deficiency invariably recurs in the graft because the recipient genotype determines hepatocyte protein production.

C. Liver transplantation replaces the source of abnormal alpha-1 antitrypsin and prevents recurrent alpha-1 antitrypsin liver disease in the graft.

D. Severe emphysema is irrelevant to transplant candidacy because liver transplantation immediately normalizes pulmonary function.

E. Specific augmentation therapy reverses cirrhosis and should be used instead of transplantation for decompensated liver disease.

7. A 58-year-old with hereditary hemochromatosis, decompensated cirrhosis, insulin-requiring diabetes, and cardiomyopathy is referred for transplant evaluation. What is the most important additional assessment before listing?

A. Measure transferrin saturation monthly because only normalization below 20% determines suitability for transplantation.

B. Exclude transplantation until all extrahepatic iron has been removed by phlebotomy regardless of clinical stability.

C. Perform liver biopsy to reconfirm hepatic iron because genetic confirmation is insufficient for transplant evaluation.

D. Stop diabetes treatment during evaluation because post-transplant immunosuppression reliably reverses iron-related diabetes.

E. Define the severity and reversibility of cardiac iron-related dysfunction because significant cardiomyopathy can drive perioperative mortality.

8. A 26-year-old with ornithine transcarbamylase deficiency has recurrent hyperammonemic crises despite optimized medical and dietary therapy but has normal hepatic synthetic function. Which statement best supports liver transplantation?

A. Transplantation is inappropriate because normal INR and bilirubin exclude a liver-based indication for surgery.

B. Transplantation should be delayed until cirrhosis develops because metabolic crises alone do not justify graft replacement.

C. Transplantation can be indicated to correct the hepatic urea-cycle defect even when conventional liver synthetic function is normal.

D. Kidney transplantation is preferred because renal ammoniagenesis is the dominant cause of recurrent hyperammonemia.

E. Portosystemic shunt creation is definitive because reducing portal delivery of nitrogen corrects the enzyme deficiency.

9. A 19-year-old with cystic fibrosis has portal hypertension, preserved synthetic function, FEV1 28% predicted, and frequent pulmonary exacerbations. Which transplant approach requires particular consideration?

A. Isolated liver transplantation should proceed without pulmonary assessment because cystic-fibrosis lung disease does not influence perioperative risk.

B. Isolated lung transplantation is preferred because portal hypertension will reliably regress after correction of hypoxemia.

C. A combined liver-lung strategy may be necessary if pulmonary disease is too advanced to permit safe isolated liver transplantation.

D. TIPS should replace transplant evaluation because cystic-fibrosis portal hypertension is never associated with progressive hepatic disease.

E. Pancreas-liver transplantation is routinely required because pancreatic insufficiency predicts graft failure after isolated liver transplantation.

10. A 42-year-old with Caroli disease has recurrent bacterial cholangitis involving both hepatic lobes, secondary biliary cirrhosis, and no extrahepatic obstruction. Which therapy offers the best definitive management?

A. Long-term rotating antibiotics alone because transplantation does not prevent recurrence of congenital intrahepatic duct ectasia.

B. Whipple resection because the primary abnormality is distal common bile duct obstruction with secondary intrahepatic dilation.

C. Repeated ERCP with balloon dilation because diffuse Caroli disease is usually cured by endoscopic decompression.

D. Liver transplantation because diffuse bilateral disease with recurrent cholangitis and cirrhosis is not amenable to curative segmental resection.

E. TIPS placement because portal decompression removes the intrahepatic ductal abnormalities responsible for cholangitis.

11. A 63-year-old with hereditary transthyretin amyloidosis has progressive polyneuropathy but preserved liver tests and no cirrhosis. Why can liver transplantation be disease-modifying in selected patients?

A. The liver clears deposited amyloid from peripheral nerves, so replacing it directly reverses established axonal degeneration.

B. Transplantation corrects the germline TTR mutation in all recipient tissues and prevents extrahepatic protein production.

C. The liver produces most circulating mutant transthyretin, so replacing it markedly reduces synthesis of the amyloidogenic protein.

D. Portal hypertension drives transthyretin deposition, so transplantation works by eliminating elevated portal pressure.

E. The benefit comes primarily from lifelong calcineurin inhibition, which blocks transthyretin misfolding in peripheral tissues.

12. A 17-year-old with Crigler-Najjar syndrome type I has severe unconjugated hyperbilirubinemia controlled only with prolonged daily phototherapy. Which statement about transplantation is correct?

A. Transplantation is ineffective because bilirubin conjugation occurs predominantly in the spleen rather than hepatocytes.

B. Transplantation is indicated only after cirrhosis appears because isolated bilirubin-conjugation defects do not justify graft replacement.

C. Kidney transplantation is preferred because renal bilirubin excretion is the limiting step in Crigler-Najjar syndrome.

D. Liver transplantation is curative because the donor liver supplies functional bilirubin UDP-glucuronosyltransferase activity.

E. Phototherapy permanently induces the missing enzyme, so transplantation provides no metabolic advantage after adolescence.

13. A 35-year-old with erythropoietic protoporphyria has progressive cholestatic liver failure and very high erythrocyte protoporphyrin levels. Which principle is most important when considering liver transplantation?

A. Transplantation is curative because all clinically relevant protoporphyrin is synthesized by hepatocytes after childhood.

B. Transplantation is contraindicated because photosensitivity invariably causes immediate graft thrombosis after reperfusion.

C. Liver transplantation treats the hepatic consequence but does not correct ongoing marrow production of protoporphyrin, so recurrence remains possible.

D. Splenectomy alone is definitive because splenic protoporphyrin production is the principal source of hepatic deposition.

E. Long-term corticosteroids after transplantation suppress the genetic marrow defect and prevent recurrent protoporphyrin accumulation.

14. A 48-year-old woman has 9 months of pruritus, alkaline phosphatase 2.1 times the upper limit of normal, normal MRCP, and a strongly positive antimitochondrial antibody. Which next step is most appropriate?

A. Perform ERCP because a normal MRCP cannot exclude the large-duct obstruction typical of primary biliary cholangitis.

B. Diagnose primary biliary cholangitis and begin ursodeoxycholic acid without requiring liver biopsy for confirmation.

C. Begin prednisone and azathioprine because antimitochondrial antibody positivity indicates autoimmune hepatitis overlap.

D. Observe without therapy until bilirubin rises because early treatment does not modify the course of primary biliary cholangitis.

E. Obtain liver biopsy in every case because histology is required before primary biliary cholangitis can be diagnosed.

15. A 52-year-old with PBC has taken weight-based ursodeoxycholic acid for 14 months. Alkaline phosphatase remains 2.3 times the upper limit of normal and bilirubin is slowly rising. What is the best interpretation?

A. She has an inadequate biochemical response and should be evaluated for appropriate second-line therapy while reassessing disease stage and transplant risk.

B. This is an expected response to ursodeoxycholic acid, and treatment should be stopped because persistent cholestasis indicates drug toxicity.

C. Biochemical response is irrelevant in PBC, so no change in management is needed until ascites or variceal bleeding develops.

D. The laboratory pattern proves large-duct PSC, and ERCP should replace PBC-directed therapy despite the prior diagnostic profile.

E. Rising bilirubin excludes PBC and requires immunosuppression for seronegative autoimmune hepatitis rather than further PBC therapy.

16. A 29-year-old woman has fatigue, AST 620 U/L, ALT 710 U/L, IgG 2.2 times normal, positive ANA and smooth-muscle antibody, negative viral testing, and biopsy showing interface hepatitis rich in plasma cells. Which treatment is most appropriate?

A. Start ursodeoxycholic acid alone because positive autoantibodies with elevated aminotransferases most strongly indicate primary biliary cholangitis.

B. Start corticosteroid-based therapy and add a steroid-sparing agent such as azathioprine when appropriate after assessing contraindications.

C. Observe without treatment because autoimmune hepatitis commonly resolves spontaneously once infectious causes have been excluded.

D. Begin tacrolimus as mandatory first-line monotherapy because conventional corticosteroid regimens are no longer used for autoimmune hepatitis.

E. Perform ERCP before treatment because interface hepatitis generally results from occult extrahepatic biliary obstruction.

17. A patient with autoimmune hepatitis has cirrhosis and active inflammation on biopsy. She asks whether budesonide can replace prednisone to reduce systemic adverse effects. What is the best response?

A. Budesonide is preferred specifically in cirrhosis because reduced first-pass metabolism increases therapeutic concentrations within the liver.

B. Budesonide and prednisone are equivalent in all stages of autoimmune hepatitis, so cirrhosis does not affect drug choice.

C. Budesonide is contraindicated only when bilirubin is normal because preserved excretion leads to inadequate hepatic drug exposure.

D. Budesonide should be avoided in cirrhosis because portosystemic shunting reduces first-pass metabolism and increases systemic exposure.

E. Budesonide is used only after liver transplantation and has no role in treatment of native-liver autoimmune hepatitis.

18. A 38-year-old with autoimmune hepatitis achieved complete biochemical remission on azathioprine and low-dose prednisone. She wants to stop therapy after 8 months of normal aminotransferases and IgG. What is the best advice?

A. Continue therapy because treatment withdrawal should be considered only after a sustained period of complete biochemical remission and careful relapse-risk assessment.

B. Stop both drugs now because 6 months of normal aminotransferases is sufficient to establish permanent remission in most patients.

C. Stop azathioprine but continue prednisone indefinitely because antimetabolite therapy has no role once remission has been achieved.

D. Stop prednisone and azathioprine permanently if ANA remains positive because persistent autoantibodies predict protection from relapse.

E. Switch to ursodeoxycholic acid before withdrawal because it prevents relapse of autoimmune hepatitis after immunosuppression is stopped.

19. A 45-year-old man with ulcerative colitis has cholestatic liver tests. MRCP shows multifocal short strictures and intervening dilatation of the intrahepatic and extrahepatic ducts. Which diagnosis is most likely?

A. Primary biliary cholangitis with large-duct involvement.

B. Autoimmune hepatitis without cholangiopathy.

C. IgG4-related cholangitis isolated to the pancreas.

D. Recurrent pyogenic cholangitis from a single distal obstruction.

E. Primary sclerosing cholangitis causing multifocal inflammatory biliary strictures.

20. A 51-year-old with PSC develops worsening jaundice and a new dominant-appearing hilar stricture. CA 19-9 is elevated. What is the most appropriate next step?

A. Assume progression of PSC and increase ursodeoxycholic acid to very high doses without additional cancer evaluation.

B. Perform directed evaluation for cholangiocarcinoma with high-quality biliary imaging and endoscopic sampling of the suspicious stricture.

C. List immediately for transplantation without tumor staging because cholangiocarcinoma does not alter transplant eligibility in PSC.

D. Treat empirically with corticosteroids because a new dominant stricture in PSC is most often an inflammatory autoimmune flare.

E. Schedule routine annual follow-up because CA 19-9 and a new hilar stricture have low predictive value for clinically important disease.

21. A patient with PSC and extensive ulcerative colitis asks how colorectal cancer surveillance should differ from that of ulcerative colitis alone. Which approach is most appropriate?

A. Delay surveillance until 10 years after colitis onset because PSC does not change colorectal cancer risk or screening timing.

B. Perform colonoscopy only when rectal bleeding occurs because PSC-associated colorectal neoplasia is usually symptomatic at an early stage.

C. Use intensified colonoscopic surveillance beginning at the diagnosis of PSC-associated colitis and continue at frequent regular intervals.

D. Replace colonoscopy with annual stool DNA testing because immunosuppression and cholestasis make mucosal biopsies unreliable.

E. Stop surveillance after liver transplantation because the colorectal cancer risk associated with PSC disappears with graft replacement.

22. A 40-year-old with PSC has intractable pruritus, recurrent bacterial cholangitis requiring hospitalizations, and a MELD score of 11. Which statement about transplant referral is most accurate?

A. Referral should wait until MELD exceeds 25 because PSC-specific complications cannot support transplantation at lower calculated urgency.

B. Transplant evaluation is appropriate because recurrent cholangitis and severe quality-of-life complications may justify consideration even when MELD underestimates disease burden.

C. TIPS is the preferred definitive treatment because recurrent cholangitis in PSC is primarily caused by portal hypertension rather than biliary disease.

D. Transplantation is contraindicated until all biliary strictures are eradicated because recurrent cholangitis always predicts graft infection.

E. A low MELD score proves short-term prognosis is excellent and excludes any need for transplant-center assessment.

23. A 62-year-old has painless jaundice, diffuse pancreatic enlargement, serum IgG4 four times normal, and long smooth strictures of the common bile duct. Which feature most favors IgG4-related cholangitis over classic PSC?

A. Marked IgG4 elevation with autoimmune pancreatitis and a steroid-responsive biliary inflammatory process.

B. A strong association with ulcerative colitis and lifelong lack of response to immunosuppressive therapy.

C. Multifocal beading limited to small intrahepatic ducts with normal pancreatic imaging and normal serum IgG4.

D. Antimitochondrial antibody positivity with florid duct lesions affecting interlobular bile ducts.

E. A childhood onset pattern dominated by biliary atresia and congenital absence of the extrahepatic bile duct.

24. A 47-year-old with sarcoidosis has cholestatic liver tests, granulomas on biopsy, portal hypertension, and progressive synthetic dysfunction. Which statement is most accurate regarding transplantation?

A. Transplantation is contraindicated because systemic sarcoidosis always recurs aggressively in the liver allograft and causes early graft loss.

B. Liver transplantation is an option for decompensated hepatic sarcoidosis, although granulomatous disease may recur in the graft without necessarily causing graft failure.

C. Only corticosteroid therapy is appropriate because hepatic sarcoidosis never progresses to clinically significant portal hypertension or cirrhosis.

D. Isolated liver transplantation automatically cures extrahepatic sarcoidosis because the liver is the source of the pathogenic granulomatous immune response.

E. TIPS permanently reverses hepatic sarcoid infiltration and should be used instead of transplantation for progressive synthetic dysfunction.

25. A 31-year-old with celiac disease has persistent aminotransferase elevation despite strict gluten avoidance. IgG is elevated and smooth-muscle antibody is positive. What is the most appropriate next step?

A. Evaluate for autoimmune hepatitis, including liver biopsy when indicated, rather than attributing persistent hepatitis solely to celiac disease.

B. Assume occult gluten exposure because autoimmune liver disease is not meaningfully associated with celiac disease.

C. Start a gluten-free diet more intensively and defer any hepatic evaluation until aminotransferases exceed 20 times normal.

D. Begin ursodeoxycholic acid because celiac-associated liver disease most often represents primary biliary cholangitis despite hepatocellular tests.

E. Perform TIPS because persistent aminotransferase elevation in celiac disease reflects clinically significant portal hypertension.

26. A 43-year-old woman has alkaline phosphatase 2.5 times normal, ALT 7 times normal, positive antimitochondrial and smooth-muscle antibodies, elevated IgG, and biopsy showing both florid duct lesions and interface hepatitis. Which diagnosis best integrates these findings?

A. Primary biliary cholangitis–autoimmune hepatitis overlap syndrome.

B. Isolated primary biliary cholangitis without hepatitic features.

C. Isolated autoimmune hepatitis without cholangiopathic features.

D. Classic large-duct primary sclerosing cholangitis.

E. Drug-induced cholestasis without autoimmune features.

27. A patient with autoimmune hepatitis–PBC overlap has substantial interface hepatitis and cholestatic disease. Which treatment concept is most appropriate?

A. Treat only the cholestatic component because immunosuppression worsens all forms of autoimmune cholangiopathy.

B. Treat both components, typically combining PBC-directed therapy with immunosuppression tailored to the severity of autoimmune hepatitis activity.

C. Treat only with corticosteroids because ursodeoxycholic acid has no role once interface hepatitis is present.

D. Avoid all disease-modifying therapy because overlap syndromes are benign and rarely progress to fibrosis.

E. Use long-term antibiotics because overlap syndromes are caused by recurrent occult bacterial cholangitis.

28. A 56-year-old with decompensated cirrhosis from autoimmune hepatitis is being evaluated for transplantation. Which post-transplant issue should be discussed preoperatively?

A. Autoimmune hepatitis cannot recur because the recipient immune system is completely replaced by the donor liver at transplantation.

B. All immunosuppression should be stopped within three months because continued therapy increases rather than decreases recurrent autoimmune hepatitis.

C. Recurrence is prevented solely by ursodeoxycholic acid, allowing calcineurin inhibitors to be withdrawn early in every recipient.

D. Autoimmune hepatitis can recur in the allograft, so long-term immunosuppression and surveillance must be individualized to recurrence and rejection risk.

E. Recurrent autoimmune hepatitis is identical to biliary anastomotic stricture and is diagnosed by cholangiography rather than clinical and histologic assessment.

29. A 35-year-old with autoimmune hepatitis is 10 weeks pregnant and stable on azathioprine plus low-dose prednisone. Which management is most appropriate?

A. Stop all immunosuppression immediately because azathioprine and prednisone are absolutely contraindicated throughout pregnancy.

B. Switch to mycophenolate because it is the preferred antimetabolite during pregnancy and prevents postpartum autoimmune hepatitis flares.

C. Delay all laboratory monitoring until after delivery because aminotransferase fluctuations are physiologic during pregnancy.

D. Continue needed maintenance therapy because disease flare during pregnancy or postpartum poses substantial maternal and fetal risk, and these agents can be used when clinically indicated.

E. Schedule elective liver transplantation during the second trimester because pregnancy accelerates autoimmune hepatitis in all patients.

30. A 60-year-old with PBC, osteoporosis, and severe pruritus has no decompensation. Which ongoing complication-focused management is most appropriate in addition to disease-modifying therapy?

A. Avoid bone-density assessment because osteoporosis becomes clinically relevant only after liver transplantation.

B. Treat pruritus with escalating opioid agonists as first-line therapy because cholestatic itch reflects endogenous opioid deficiency.

C. Restrict dietary calcium because cholestasis commonly causes hypercalcemia and calcium supplementation accelerates vascular calcification.

D. Defer symptom management until bilirubin exceeds 10 mg/dL because pruritus severity parallels synthetic dysfunction and transplant urgency.

E. Assess and treat bone disease and use targeted antipruritic therapy because cholestatic symptoms and osteoporosis materially affect morbidity even before decompensation.

31. A 49-year-old with PSC has normal bilirubin but recurrent fevers, right upper quadrant pain, and bacteremia. Imaging shows a high-grade extrahepatic stricture with upstream dilation. What is the most appropriate management?

A. Begin high-dose steroids alone because bacterial cholangitis in PSC reflects sterile inflammation rather than biliary obstruction.

B. Observe because normal bilirubin excludes a clinically important stricture and endoscopic intervention would add unnecessary risk.

C. Place a TIPS to decompress the biliary tree because portal pressure is the principal driver of PSC-associated cholangitis.

D. Treat acute bacterial cholangitis and perform therapeutic endoscopic evaluation of the clinically significant stricture with sampling when indicated.

E. Proceed directly to colectomy because removal of the colon is the most effective treatment for recurrent biliary infection in PSC.

32. A 55-year-old with PBC has worsening portal hypertension despite only modest elevations in bilirubin and aminotransferases. Which concept best explains this pattern?

A. Portal hypertension in PBC always indicates acute portal vein thrombosis rather than progression of the underlying cholangiopathy.

B. A low bilirubin excludes clinically significant portal hypertension, making the splenomegaly and varices unrelated to liver disease.

C. Portal hypertension can develop before advanced synthetic failure in cholestatic liver disease, so clinical staging must not rely on bilirubin alone.

D. Portal hypertension develops only after profound hepatocyte necrosis, so PBC patients with modest aminotransferases cannot develop varices.

E. The finding proves coexisting hereditary hemochromatosis because PBC itself does not cause presinusoidal or cirrhotic portal hypertension.

33. A 44-year-old with type 1 autoimmune hepatitis has normal aminotransferases on azathioprine but persistent IgG elevation. Which statement is most accurate?

A. Normal aminotransferases alone establish complete biochemical remission, so IgG can be ignored during long-term monitoring.

B. Persistent IgG elevation suggests incomplete biochemical remission and should prompt reassessment of adherence, diagnosis, and adequacy of therapy.

C. Persistent IgG elevation proves primary sclerosing cholangitis and mandates ERCP even without cholestatic liver tests.

D. Azathioprine should be stopped because elevated IgG is a predictable drug effect rather than a marker of immune activity.

E. The finding indicates irreversible cirrhosis and makes further immunosuppressive optimization clinically futile.

34. A 58-year-old with decompensated cirrhosis is HBsAg positive, HBeAg negative, and has HBV DNA 85,000 IU/mL. Creatinine is normal. Which antiviral strategy is most appropriate?

A. Defer antiviral therapy until ALT rises above five times normal because cirrhosis alone does not justify HBV treatment.

B. Use a finite 12-week course of direct-acting antivirals because chronic HBV is treated analogously to hepatitis C.

C. Start a potent nucleos(t)ide analogue with a high barrier to resistance, such as entecavir or tenofovir, and continue long-term therapy.

D. Begin pegylated interferon because decompensated cirrhosis is the setting in which interferon has the best safety profile.

E. Treat only if HBeAg becomes positive because HBeAg-negative viremia does not contribute to hepatic decompensation.

35. A 62-year-old with HCV cirrhosis and prior variceal bleeding is being evaluated for transplantation. He has untreated genotype-independent chronic HCV infection and no HCC. Which statement is most accurate?

A. HCV treatment must always be deferred until after transplantation because pretransplant cure increases graft failure in all recipients.

B. HCV treatment is contraindicated in any patient with decompensated cirrhosis because all direct-acting antiviral regimens are hepatotoxic.

C. Direct-acting antiviral treatment should be individualized around transplant timing because cure can improve hepatic function but may also alter access to transplant and HCV-positive donor options.

D. HCV cure eliminates the need for transplant evaluation even when prior decompensation and portal hypertension remain clinically important.

E. Interferon and ribavirin remain the preferred pretransplant regimen because direct-acting antivirals cannot be used near transplantation.

36. A patient with chronic HBV has persistent ALT elevation and worsening fibrosis despite undetectable HBV DNA on tenofovir. HBsAg is positive and anti-HDV antibody is positive. Which test best determines whether active hepatitis D is present?

A. Serum HDV RNA testing.

B. Quantitative anti-HDV IgG titer alone.

C. HBV core IgM testing alone.

D. HBeAg testing despite suppressed HBV DNA.

E. Serum alpha-fetoprotein testing.

37. A liver transplant candidate with cirrhosis is nonimmune to hepatitis A and hepatitis B. What is the preferred preventive approach?

A. Delay both vaccines until after transplantation because pretransplant cirrhosis makes vaccination ineffective and unsafe.

B. Vaccinate against both hepatitis A and hepatitis B before transplantation when feasible because immune responses are generally better before immunosuppression.

C. Give only hepatitis A vaccine because hepatitis B vaccination is contraindicated in patients likely to receive immunosuppression.

D. Use live attenuated hepatitis vaccines immediately after transplantation because live vaccines provide stronger protection in immunosuppressed recipients.

E. Rely on post-transplant immunoglobulin prophylaxis rather than routine vaccination because vaccine-derived immunity cannot persist after transplant.

38. A 49-year-old transplant candidate has chronic HEV viremia after prolonged immunosuppressive therapy for another condition. Which feature distinguishes hepatitis E from typical self-limited infection in immunocompetent adults?

A. HEV invariably causes chronic infection in immunocompetent adults and usually requires lifelong nucleos(t)ide analogue therapy.

B. HEV is diagnosed by HBsAg because it requires hepatitis B surface antigen for viral assembly and transmission.

C. HEV cannot cause clinically important disease after transplantation because calcineurin inhibitors suppress its replication.

D. HEV is prevented by standard hepatitis A vaccination because the two viruses have cross-protective capsid antigens.

E. HEV can become chronic in immunosuppressed patients and may lead to progressive fibrosis, so persistent RNA requires active management.

39. A 24-year-old develops acute liver failure with fever, rapidly rising aminotransferases, coagulopathy, and encephalopathy. There are no vesicular lesions. The cause remains unclear after initial testing. Which empiric therapy should be considered while HSV testing is pending?

A. Oral valganciclovir because CMV is the usual cause of fulminant viral hepatitis in immunocompetent adults.

B. High-dose interferon because empiric antiviral immune stimulation improves survival in undifferentiated acute liver failure.

C. Intravenous acyclovir because HSV hepatitis can be fulminant and may occur without mucocutaneous lesions.

D. Lamivudine alone because occult HBV is the most common cause of hyperacute liver failure with fever.

E. No empiric antiviral therapy because absence of vesicles essentially excludes HSV hepatitis.

40. A 39-year-old with chronic HBV is listed for liver transplantation. Which pretransplant variable most directly increases risk of post-transplant HBV recurrence if not adequately suppressed?

A. High circulating HBV DNA at the time of transplantation.

B. A normal alkaline phosphatase immediately before transplantation.

C. A remote history of hepatitis A infection with protective IgG.

D. A low serum uric acid concentration during diuretic therapy.

E. A negative antimitochondrial antibody obtained during evaluation.

41. A 32-year-old woman presents with abdominal pain, ascites, hepatomegaly, and rapidly worsening liver tests. Doppler ultrasound shows absent flow in the hepatic veins with caudate lobe enlargement. Which diagnosis is most likely?

A. Portal vein thrombosis causing isolated prehepatic portal hypertension.

B. Primary sclerosing cholangitis causing diffuse large-duct obstruction.

C. Budd-Chiari syndrome due to hepatic venous outflow obstruction.

D. Sinusoidal obstruction syndrome caused by post-transplant biliary ischemia.

E. Hepatic artery thrombosis producing acute ischemic cholangiopathy.

42. A 40-year-old with Budd-Chiari syndrome related to a myeloproliferative neoplasm has persistent ascites despite anticoagulation and diuretics. Hepatic veins cannot be recanalized. What is the next preferred escalation in an otherwise suitable patient?

A. Consider TIPS to decompress hepatic congestion before proceeding to transplantation if anatomy and clinical status permit.

B. Stop anticoagulation because persistent ascites indicates treatment failure and continued anticoagulation has no further role.

C. Perform splenectomy because portal decompression in Budd-Chiari is achieved primarily by reducing splenic venous inflow.

D. Begin high-dose corticosteroids because refractory ascites indicates an autoimmune hepatic-vein inflammatory flare.

E. Proceed directly to colectomy because myeloproliferative-associated Budd-Chiari is commonly driven by occult inflammatory bowel disease.

43. A patient develops painful hepatomegaly, weight gain, ascites, and rising bilirubin three weeks after hematopoietic stem-cell transplantation. Doppler shows patent major hepatic veins. Which diagnosis is most likely?

A. Budd-Chiari syndrome caused by thrombosis of the major hepatic veins.

B. Primary biliary cholangitis caused by antimitochondrial antibody–mediated duct injury.

C. Sinusoidal obstruction syndrome caused by toxic injury to hepatic sinusoidal endothelial cells.

D. Hepatic artery thrombosis causing ischemic necrosis of the extrahepatic bile duct.

E. Acute portal vein thrombosis causing isolated splenomegaly without hepatocellular congestion.

44. A 24-year-old adult with prior Fontan palliation has nodular liver morphology, thrombocytopenia, and elevated liver stiffness but only mild aminotransferase abnormalities. Which mechanism primarily drives Fontan-associated liver disease?

A. Chronically elevated systemic venous pressure with reduced effective hepatic perfusion produces congestive and fibrotic liver injury.

B. Autoimmune destruction of interlobular bile ducts causes progressive cholestasis after childhood Fontan surgery.

C. Persistent hepatitis C acquired during surgery is assumed to be the dominant mechanism regardless of viral testing.

D. Excess hepatic arterial flow causes isolated portal hypertension without contribution from systemic venous congestion.

E. Congenital absence of the portal vein causes all clinically significant liver disease in patients with Fontan circulation.

45. A patient with Fontan-associated liver disease has advanced hepatic fibrosis and progressive Fontan failure with severe ventricular dysfunction. Which transplant strategy may be most appropriate?

A. Evaluate for combined heart-liver transplantation when both cardiac and hepatic disease are advanced and irreversible.

B. Perform isolated liver transplantation because replacing the liver reliably corrects the abnormal Fontan hemodynamics.

C. Perform isolated heart transplantation in every case because hepatic fibrosis always regresses completely after cardiac replacement.

D. Use TIPS as definitive therapy because reducing portal pressure corrects the elevated central venous pressure driving Fontan failure.

E. Defer all transplant evaluation until bilirubin exceeds 20 mg/dL because synthetic function is the only reliable measure of Fontan liver severity.

46. A 66-year-old with severe tricuspid regurgitation has ascites, hepatomegaly, elevated JVP, and a cholestatic-predominant liver-test pattern. Which intervention is most likely to improve the liver abnormalities?

A. Start ursodeoxycholic acid because the cholestatic tests indicate primary biliary cholangitis independent of the cardiac findings.

B. Begin corticosteroids because congestive hepatopathy is driven by immune-mediated sinusoidal inflammation.

C. Perform TIPS because increasing venous return is the preferred treatment for liver injury caused by severe right-sided heart failure.

D. Optimize the underlying cardiac disease and venous congestion because the hepatic injury is secondary to right-sided heart failure.

E. Use repeated phlebotomy because congestive hepatopathy usually reflects occult hepatic iron overload from low cardiac output.

47. A 28-year-old with sickle cell disease develops right upper quadrant pain, marked conjugated hyperbilirubinemia, coagulopathy, and progressive encephalopathy during a vaso-occlusive crisis. Which diagnosis should be considered?

A. Gilbert syndrome because unconjugated bilirubin from hemolysis is the only mechanism of jaundice in sickle cell disease.

B. Severe sickle cell hepatopathy with acute hepatic failure, requiring urgent multidisciplinary management and transplant-center involvement.

C. Primary biliary cholangitis because cholestatic liver failure in sickle cell disease is unrelated to vaso-occlusion.

D. Isolated cholelithiasis because gallstones alone explain coagulopathy and encephalopathy during a vaso-occlusive crisis.

E. Hereditary hemochromatosis because acute hepatic failure is expected after only a few red-cell transfusions.

48. A patient with decompensated congestive hepatopathy is referred for isolated liver transplantation. Which finding would most strongly argue that isolated liver transplantation is unsafe?

A. Mild thrombocytopenia from portal hypertension with otherwise stable cardiac function.

B. Severe irreversible right-ventricular failure despite optimized cardiac therapy.

C. A modest alkaline phosphatase elevation caused by chronic hepatic venous congestion.

D. Trace peripheral edema that resolves with low-dose diuretic therapy.

E. A history of atrial arrhythmia that is controlled and does not impair ventricular function.

49. A 49-year-old has varices, splenomegaly, and thrombocytopenia, but liver stiffness is low, synthetic function is preserved, and biopsy shows nodular regenerative hyperplasia without cirrhosis. Which diagnosis best fits?

A. Decompensated metabolic dysfunction–associated steatohepatitis with occult cirrhosis.

B. Primary biliary cholangitis with advanced ductopenia and cholestatic synthetic failure.

C. Budd-Chiari syndrome with complete thrombosis of the major hepatic veins.

D. Acute portal vein thrombosis without any chronic intrahepatic vascular abnormality.

E. Portosinusoidal vascular disease causing noncirrhotic portal hypertension.

50. A patient with portosinusoidal vascular disease has large esophageal varices but no ascites or encephalopathy. How should variceal risk generally be managed?

A. Avoid prophylaxis because noncirrhotic portal hypertension does not cause clinically significant variceal hemorrhage.

B. Use corticosteroids to reverse nodular regenerative hyperplasia because immune suppression is first-line prevention of bleeding.

C. Perform prophylactic splenectomy in all patients because removing splenic inflow is safer than beta-blockers or endoscopy.

D. Use standard portal-hypertension strategies for variceal prophylaxis because clinically important bleeding risk exists even without cirrhosis.

E. List for immediate transplantation solely because the presence of varices proves irreversible hepatic synthetic failure.

51. A 59-year-old with cirrhosis has an incidentally discovered nonocclusive portal vein thrombus involving 30% of the main portal vein lumen. He is a transplant candidate and has no active bleeding. Which factor most favors anticoagulation?

A. The presence of thrombocytopenia alone because low platelets prove the thrombus will spontaneously propagate without treatment.

B. A normal D-dimer because anticoagulation is indicated only when fibrinolytic markers are normal in cirrhosis.

C. Absence of varices because portal vein thrombosis cannot occur in patients who have gastroesophageal varices.

D. Transplant candidacy with risk of thrombus progression that could increase surgical complexity or preclude physiologic portal reconstruction.

E. Low MELD score because anticoagulation is used primarily to increase the MELD score and accelerate liver allocation.

52. A transplant candidate has chronic occlusive portal vein thrombosis with cavernous transformation and repeated variceal bleeding. Which intervention may restore portal inflow and improve transplantability in selected patients?

A. Portal vein recanalization with TIPS in an experienced transplant center to restore portal flow.

B. Routine splenectomy because removal of the spleen reliably reconstructs an occluded main portal vein.

C. Systemic thrombolysis months after cavernous transformation because chronic organized thrombus responds predictably to lytics.

D. Hepatic artery embolization because reducing arterial inflow causes the portal vein to reopen through pressure redistribution.

E. Long-term beta-blocker monotherapy because pharmacologic portal reduction reliably converts cavernous transformation to a normal portal vein.

53. A patient with acute portal vein thrombosis develops severe abdominal pain, lactate elevation, and CT evidence of thrombus extension into the superior mesenteric vein with bowel wall hypoenhancement. What is the most urgent concern?

A. Hepatic encephalopathy from spontaneous portosystemic shunting as the principal cause of severe abdominal pain.

B. Acute biliary obstruction because portal and mesenteric venous thrombosis usually causes ischemic cholangiopathy before bowel injury.

C. Primary sclerosing cholangitis because mesenteric venous thrombosis is a characteristic cholangiographic complication.

D. Intestinal ischemia from mesenteric venous outflow obstruction requiring urgent multidisciplinary evaluation and intervention.

E. Hepatopulmonary syndrome because lactate elevation in portal vein thrombosis most often reflects intrapulmonary vascular dilation.

54. A 23-year-old has recurrent hyperammonemic encephalopathy but normal liver synthetic function. Imaging shows a large congenital extrahepatic portosystemic shunt. Which management principle is most appropriate?

A. List immediately for liver transplantation because any congenital portosystemic shunt proves irreversible hepatic failure.

B. Begin lifelong corticosteroids because congenital shunts cause encephalopathy through immune-mediated hepatocyte inflammation.

C. Assess whether the shunt can be safely closed because diversion of portal blood can cause encephalopathy even without intrinsic liver failure.

D. Avoid shunt closure because restoring portal flow invariably causes fatal portal hypertension in all patients.

E. Treat only with diuretics because hyperammonemia in congenital shunting is caused by occult ascites rather than bypass of hepatic metabolism.

55. A transplant surgeon asks which feature of extensive portal vein thrombosis most increases operative complexity. Which is the best answer?

A. Portal vein thrombosis usually eliminates the need for portal anastomosis because the hepatic artery can provide all graft inflow.

B. Loss of a usable native portal vein may require thrombectomy, jump grafting, or nonstandard portal inflow reconstruction at transplantation.

C. Chronic thrombosis simplifies transplantation because collateral vessels provide a ready-made low-pressure portal anastomosis.

D. The main consequence is inability to construct a biliary anastomosis because portal thrombosis directly destroys the recipient common bile duct.

E. Portal vein thrombosis affects only postoperative anticoagulation and has no meaningful effect on surgical planning or graft inflow.

56. A 57-year-old with compensated cirrhosis has clinically significant portal hypertension and medium esophageal varices that have never bled. Which strategy is appropriate for primary prophylaxis?

A. Use a nonselective beta-blocker or endoscopic variceal ligation according to patient-specific factors and contraindications.

B. Give a proton-pump inhibitor indefinitely because acid suppression is more effective than portal-pressure reduction for first-bleed prevention.

C. Perform prophylactic TIPS in all patients with medium varices because shunt placement carries less morbidity than medical therapy.

D. Use selective beta-1 blockade because cardiac selectivity produces greater portal-pressure reduction than nonselective blockade.

E. Defer prophylaxis until the first variceal hemorrhage because primary prevention has not been shown to alter bleeding risk.

57. A 61-year-old with cirrhosis presents with large-volume hematemesis and hypotension. Variceal bleeding is suspected. Which immediate treatment bundle is most appropriate before endoscopic confirmation?

A. Give high-dose vitamin K alone and defer endoscopy until INR normalizes because coagulopathy is the primary cause of variceal hemorrhage.

B. Begin vasoactive therapy and prophylactic antibiotics, resuscitate judiciously, and arrange urgent endoscopy for variceal control.

C. Transfuse to a hemoglobin above 12 g/dL before any vasoactive therapy because aggressive correction lowers portal pressure.

D. Start lactulose alone because reducing intestinal ammonia rapidly stops portal-hypertensive gastrointestinal bleeding.

E. Perform diagnostic paracentesis first and delay vasoactive therapy until spontaneous bacterial peritonitis is excluded.

58. A patient survives an esophageal variceal hemorrhage treated with band ligation. Which regimen best reduces recurrent bleeding risk in the absence of contraindications?

A. Use proton-pump inhibitor monotherapy because acid suppression prevents recurrent portal-hypertensive bleeding after banding.

B. Use endoscopic surveillance alone without further therapy because successful control of the index bleed removes the recurrence risk.

C. Combine a nonselective beta-blocker with serial endoscopic variceal ligation for secondary prophylaxis.

D. Use aspirin plus anticoagulation because recurrent variceal bleeding is driven by microthrombi in the portal venous system.

E. Perform elective surgical shunt in every patient because combined pharmacologic and endoscopic therapy is inferior to surgery.

59. A 64-year-old with cirrhosis is admitted with new tense ascites. She is afebrile and has no abdominal pain. What is the most appropriate diagnostic step?

A. Perform diagnostic paracentesis promptly because hospitalized patients with new or worsening ascites should be assessed for spontaneous bacterial peritonitis even without symptoms.

B. Start high-dose diuretics without paracentesis because absence of fever makes spontaneous bacterial peritonitis unlikely.

C. Order an abdominal MRI first because paracentesis is unsafe until hepatocellular carcinoma has been excluded.

D. Correct the INR to below 1.5 with plasma before paracentesis because procedural bleeding tracks directly with INR in cirrhosis.

E. Treat empirically for hepatic encephalopathy because tense ascites is primarily a manifestation of hyperammonemia.

60. A patient with cirrhosis undergoes removal of 8 liters of ascites. Which adjunct reduces post-paracentesis circulatory dysfunction?

A. Administer intravenous albumin after large-volume paracentesis.

B. Administer fresh frozen plasma to replace coagulation factors removed with ascitic fluid.

C. Give hypertonic saline routinely to prevent all forms of post-paracentesis hyponatremia.

D. Begin high-dose beta-blockade immediately to prevent splanchnic vasodilation after fluid removal.

E. Restrict protein for 48 hours to reduce ammonia generation during intravascular volume shifts.

61. A 55-year-old with cirrhosis has recurrent tense ascites despite sodium restriction and maximally tolerated diuretics. Cardiac function is preserved and there is no uncontrolled encephalopathy. Which option should be considered?

A. Continue escalating diuretics indefinitely because refractory ascites is not an indication for transplant evaluation or portal decompression.

B. Perform peritoneovenous shunting as first-line therapy because it has lower complication rates than modern TIPS.

C. Evaluate for TIPS while simultaneously assessing candidacy for liver transplantation.

D. Start chronic systemic corticosteroids because refractory ascites reflects inflammatory failure of the peritoneal membrane.

E. Restrict fluid to 500 mL daily regardless of serum sodium because severe fluid restriction is the definitive treatment for refractory ascites.

62. Ascitic fluid from a patient with cirrhosis contains 420 polymorphonuclear leukocytes/mm3. Culture results are pending. What is the most appropriate management?

A. Wait for a positive ascitic culture because polymorphonuclear cell count alone cannot establish spontaneous bacterial peritonitis.

B. Treat immediately for spontaneous bacterial peritonitis with appropriate empiric antibiotics and albumin when indicated.

C. Repeat paracentesis in one week without antibiotics because culture-negative neutrocytic ascites is not clinically important.

D. Treat with antifungal therapy alone because bacterial infection rarely produces an ascitic neutrophil count above 250/mm3.

E. Begin corticosteroids because neutrophilic ascites in cirrhosis is usually an immune-mediated sterile peritonitis.

63. A patient with cirrhosis recovers from a first episode of spontaneous bacterial peritonitis. Which long-term strategy is appropriate?

A. No prophylaxis is needed because successful treatment of the first episode confers durable immunity to enteric organisms.

B. Use chronic intravenous albumin as the sole preventive strategy because antibiotics increase rather than decrease recurrence.

C. Perform prophylactic colectomy because colonic bacterial translocation is prevented most effectively by removing the colon.

D. Use lifelong antifungal prophylaxis because recurrent peritonitis after cirrhosis is predominantly caused by Candida species.

E. Begin secondary antibiotic prophylaxis because recurrence risk is high after an episode of spontaneous bacterial peritonitis.

64. A 63-year-old with cirrhosis is confused and has asterixis after constipation and a recent upper gastrointestinal bleed. There is no focal neurologic deficit. What is the best initial treatment approach?

A. Treat precipitating factors and give lactulose titrated to regular soft stools, adding rifaximin for recurrent episodes.

B. Restrict dietary protein to less than 20 g/day because long-term protein deprivation is the most effective way to prevent recurrence.

C. Begin chronic benzodiazepines because enhancing GABA signaling reverses the neuroinhibitory imbalance of hepatic encephalopathy.

D. Perform routine TIPS because portal decompression reduces systemic ammonia exposure in patients with recurrent encephalopathy.

E. Give intravenous mannitol for every episode because cerebral edema is the dominant mechanism of encephalopathy in chronic cirrhosis.

65. A patient with cirrhosis and recurrent encephalopathy has a large spontaneous splenorenal shunt, preserved liver function, and episodes despite optimized lactulose and rifaximin. Which intervention may help selected patients?

A. Create an additional TIPS because increasing portosystemic diversion lowers systemic ammonia exposure and prevents encephalopathy.

B. Endovascular embolization of the large spontaneous portosystemic shunt after careful portal-pressure and anatomic assessment.

C. Stop all bowel-directed therapy because encephalopathy caused by a spontaneous shunt is unrelated to intestinal ammonia production.

D. Perform splenectomy in every case because removing the spleen is equivalent to embolizing any type of portosystemic collateral.

E. Begin high-dose corticosteroids because persistent encephalopathy after rifaximin indicates autoimmune cerebral inflammation.

66. A 59-year-old with refractory hepatic hydrothorax has repeated large right pleural effusions despite sodium restriction and diuretics. There is no cardiopulmonary contraindication to shunting. Which next option is most appropriate?

A. Evaluate for TIPS and liver transplantation rather than relying on repeated chest-tube drainage.

B. Place a chronic large-bore chest tube because continuous pleural drainage is the safest long-term treatment for hepatic hydrothorax.

C. Perform pleurodesis as routine first-line therapy because the underlying portal hypertension does not affect recurrence.

D. Begin systemic corticosteroids because hepatic hydrothorax is caused by inflammatory pleuritis associated with cirrhosis.

E. Stop all diuretics permanently because sodium excretion has no effect on pleural fluid accumulation in portal hypertension.

67. A 54-year-old with cirrhosis has dyspnea, platypnea, resting oxygen saturation 86%, and contrast echocardiography showing delayed appearance of microbubbles in the left heart. Which diagnosis is most likely?

A. Portopulmonary hypertension caused by severe pulmonary arterial vasoconstriction and elevated pulmonary vascular resistance.

B. Hepatic hydrothorax because pleural fluid causes delayed intracardiac bubble transit on contrast echocardiography.

C. Cirrhotic cardiomyopathy because impaired ventricular contractility produces the characteristic delayed bubble pattern.

D. Hepatopulmonary syndrome caused by intrapulmonary vascular dilatations with abnormal oxygenation.

E. Pulmonary embolism because thromboembolic obstruction is the usual explanation for orthodeoxia in advanced cirrhosis.

68. A liver transplant candidate has mean pulmonary artery pressure 48 mm Hg and markedly elevated pulmonary vascular resistance on right-heart catheterization. Which statement is most accurate?

A. This finding is hepatopulmonary syndrome and should be treated with supplemental oxygen alone until immediate transplantation.

B. Pulmonary hypertension is expected in cirrhosis and never changes perioperative risk or transplant eligibility.

C. TIPS should be performed urgently because increasing venous return is the preferred therapy for severe portopulmonary hypertension.

D. Severe portopulmonary hypertension requires pulmonary vasodilator treatment and reassessment because uncontrolled high pulmonary vascular resistance can make transplantation unsafe.

E. Right-heart catheterization is unnecessary because echocardiography alone definitively establishes pulmonary vascular resistance and transplant risk.

69. A 60-year-old with cirrhosis develops creatinine rise from 0.8 to 2.4 mg/dL. Diuretics are stopped, nephrotoxins are removed, infection is treated, and albumin challenge does not improve renal function. Urinalysis is bland and renal ultrasound is normal. Which diagnosis is most likely?

A. Acute glomerulonephritis because bland urine sediment is typical of immune-complex renal disease in cirrhosis.

B. Hepatorenal syndrome–acute kidney injury after exclusion of structural and reversible causes of kidney injury.

C. Acute obstruction because a normal renal ultrasound strongly supports postrenal kidney injury in advanced liver disease.

D. Chronic diabetic nephropathy because a rapid creatinine rise over days is the usual first manifestation of diabetic kidney disease.

E. Calcineurin inhibitor nephrotoxicity because tacrolimus exposure is required for the diagnosis even before liver transplantation.

70. A patient with HRS-AKI remains hypotensive after albumin and has no ischemic or hypoxic contraindication. Which pharmacologic approach is preferred where available?

A. Use terlipressin with albumin while expediting liver transplant evaluation and closely monitoring respiratory status.

B. Use high-dose loop diuretics with albumin because forced diuresis reverses the renal vasoconstriction of HRS-AKI.

C. Start an ACE inhibitor because efferent arteriolar dilation restores renal blood flow in the hyperdynamic circulation of cirrhosis.

D. Use chronic nonselective beta-blockade as the sole vasoconstrictor because beta blockade directly raises renal perfusion pressure.

E. Delay vasoconstrictor therapy until dialysis is required because pharmacologic reversal does not occur before renal replacement therapy.

71. A patient with decompensated cirrhosis and HRS-AKI has increasing oxygen requirement, pulmonary edema, and SpO2 87% on supplemental oxygen. Which change in management is most important?

A. Escalate terlipressin immediately because hypoxia predicts a better renal response and is not a treatment-related safety concern.

B. Give additional large albumin boluses regardless of pulmonary edema because albumin cannot worsen respiratory failure in cirrhosis.

C. Avoid or stop terlipressin in the setting of significant hypoxia and address volume and respiratory status while using alternative hemodynamic support as appropriate.

D. Start propranolol at a high dose because beta blockade improves oxygenation and reverses HRS-associated pulmonary edema.

E. Perform TIPS emergently in every hypoxic HRS patient because TIPS is universally safe in acute respiratory failure.

72. A patient with cirrhosis has a normal resting ejection fraction but develops hypotension and poor cardiac output during stress testing. Which concept best explains this?

A. Normal resting ejection fraction excludes clinically important cirrhotic cardiac dysfunction under all physiologic conditions.

B. Cirrhotic cardiomyopathy can manifest as impaired contractile reserve and electrophysiologic abnormalities despite normal resting systolic function.

C. The finding is diagnostic of hepatopulmonary syndrome because intrapulmonary shunting directly causes impaired myocardial contractile reserve.

D. Cirrhotic cardiomyopathy results primarily from fixed epicardial coronary obstruction and is therefore synonymous with ischemic cardiomyopathy.

E. Portal hypertension protects against cardiac stress by lowering effective preload, so stress-induced dysfunction is unrelated to cirrhosis.

73. A 57-year-old with refractory ascites has serum sodium 121 mEq/L but no seizures or focal neurologic symptoms. Which strategy is most appropriate?

A. Raise serum sodium to normal within six hours with repeated hypertonic saline boluses because chronic cirrhotic hyponatremia tolerates rapid correction.

B. Increase diuretic doses aggressively because worsening effective arterial volume is the preferred method to stimulate free-water excretion.

C. Start desmopressin as chronic monotherapy because retaining additional free water corrects dilutional hyponatremia in cirrhosis.

D. Correct contributing factors gradually, limit free water when appropriate, and avoid rapid sodium correction that could cause osmotic demyelination.

E. Ignore the sodium level because hyponatremia has no prognostic significance and does not influence transplant-related risk.

74. A patient with cirrhosis has INR 2.1 and platelets 72,000/μL but no active bleeding. He needs diagnostic paracentesis. Which statement best reflects cirrhotic hemostasis?

A. INR accurately measures the net bleeding tendency in cirrhosis, so plasma should always normalize the INR before any invasive procedure.

B. Thrombocytopenia proves spontaneous bleeding is imminent because cirrhosis causes only loss of procoagulant factors without compensatory changes.

C. INR alone poorly predicts procedural bleeding in cirrhosis, and routine plasma correction before low-risk paracentesis is generally unnecessary.

D. Cirrhosis produces a purely hypercoagulable state, so no patient with an elevated INR can experience clinically important bleeding.

E. Vitamin K should be given until INR is below 1.2 because all coagulopathy in cirrhosis results from vitamin K deficiency.

75. A 63-year-old with cirrhosis has a 2.4-cm arterial phase hyperenhancing lesion with washout and capsule on multiphasic MRI. There is no vascular invasion. What is the most appropriate diagnostic interpretation?

A. The lesion is diagnostic of hepatocellular carcinoma in an at-risk cirrhotic liver when the imaging features meet accepted noninvasive criteria.

B. Biopsy is mandatory before hepatocellular carcinoma can be diagnosed in any patient with cirrhosis.

C. The lesion is most consistent with focal nodular hyperplasia because washout and capsule are benign imaging features.

D. The lesion should be considered cholangiocarcinoma until CA 19-9 is normal, regardless of the enhancement pattern.

E. The lesion cannot be classified without PET imaging because multiphasic MRI lacks specificity for hepatocellular carcinoma.

76. A 58-year-old with Child-Pugh A cirrhosis has a single 3.2-cm HCC, clinically significant portal hypertension, and no extrahepatic disease. Which curative strategy is generally favored if the patient is an acceptable candidate?

A. Major hepatic resection because portal hypertension improves postoperative liver regeneration and lowers decompensation risk.

B. Liver transplantation because it treats both the tumor and the cirrhotic liver with portal hypertension.

C. Systemic therapy alone because any portal hypertension excludes potentially curative local or transplant-based treatment.

D. Observation until the tumor exceeds 5 cm because small HCCs do not benefit from definitive therapy in cirrhosis.

E. Long-term antibiotics because HCC arising in cirrhosis is driven mainly by occult bacterial translocation.

77. A transplant candidate with cirrhosis has a single 6.5-cm HCC without vascular invasion or metastasis. Which strategy may preserve a path to transplantation?

A. List immediately with standard HCC exception because any single tumor below 8 cm automatically qualifies without treatment.

B. Exclude transplantation permanently because any lesion larger than 5 cm can never be reconsidered after locoregional therapy.

C. Use locoregional therapy for downstaging and consider listing if the tumor burden is successfully reduced to accepted transplant criteria with sustained favorable biology.

D. Begin lifelong immunosuppression before transplantation because tumor shrinkage from calcineurin inhibition predicts transplant eligibility.

E. Perform routine TIPS as the principal downstaging therapy because lowering portal pressure directly reduces HCC diameter.

78. A patient being downstaged for HCC has an AFP of 1,450 ng/mL. After locoregional therapy, AFP falls to 180 ng/mL and imaging shows viable tumor within accepted criteria. Why is this change clinically important?

A. AFP decline proves complete pathologic response, so no additional imaging or observation is needed before transplantation.

B. AFP is unrelated to post-transplant recurrence risk and is measured only to distinguish HCC from cholangiocarcinoma.

C. An initial AFP above 1,000 permanently excludes liver transplantation regardless of subsequent treatment response.

D. AFP reduction after therapy indicates worsening portal hypertension and should trigger TIPS rather than further oncologic assessment.

E. Marked AFP decline suggests more favorable tumor biology and can influence eligibility for standardized HCC exception pathways when other criteria are satisfied.

79. A 42-year-old without cirrhosis has a 9-cm solitary liver mass. Imaging suggests fibrolamellar hepatocellular carcinoma, and staging shows no metastases. Which management is most appropriate when technically feasible?

A. Liver transplantation is automatically first-line for every fibrolamellar HCC regardless of resectability or extrahepatic staging.

B. No treatment is indicated because fibrolamellar HCC is a benign variant that rarely recurs or metastasizes.

C. Surgical resection is the preferred potentially curative therapy because fibrolamellar HCC often arises without cirrhosis and may be resectable.

D. TIPS is preferred because the disease is caused by portal hypertension even in a noncirrhotic liver.

E. Ursodeoxycholic acid is first-line because fibrolamellar HCC arises from autoimmune injury to intrahepatic bile ducts.

80. A 49-year-old with unresectable perihilar cholangiocarcinoma has no metastases and meets a specialized transplant-center protocol. Which statement is most accurate?

A. Cholangiocarcinoma is an absolute contraindication to liver transplantation in all anatomic locations and clinical circumstances.

B. Transplantation should proceed without neoadjuvant therapy because pretransplant radiation universally worsens graft survival.

C. Selected patients can undergo neoadjuvant therapy followed by liver transplantation under strict protocols with careful staging.

D. TIPS is the definitive oncologic treatment because reducing portal pressure prevents local progression of hilar cholangiocarcinoma.

E. Routine systemic chemotherapy alone provides equivalent curative outcomes to protocol-based transplantation in strictly selected perihilar disease.

81. A 36-year-old woman has a 6-cm hepatic adenoma that enlarged during estrogen-containing contraceptive use. Which management is most appropriate?

A. Continue estrogen and observe because hepatic adenomas usually regress only while exogenous estrogen is maintained.

B. Treat with lifelong nonselective beta-blockade because adenoma hemorrhage is primarily a consequence of portal hypertension.

C. List for liver transplantation immediately because any adenoma larger than 5 cm requires graft replacement rather than local therapy.

D. Stop estrogen exposure and pursue definitive treatment because a large adenoma carries hemorrhage and malignant-transformation risk.

E. Start corticosteroids because hepatic adenoma enlargement reflects an autoimmune hepatocellular proliferation.

82. A 44-year-old man is found to have a 3.5-cm hepatic adenoma on MRI. He is asymptomatic and has no cirrhosis. Which feature most strongly changes management compared with a similarly sized lesion in a woman?

A. Male sex increases concern for malignant transformation and generally favors resection even for smaller adenomas.

B. Male sex lowers malignant-transformation risk, so observation is preferred until the lesion exceeds 10 cm.

C. Men do not develop true hepatic adenomas, so the lesion should be assumed to be focal nodular hyperplasia without further evaluation.

D. Male sex makes the lesion an automatic indication for liver transplantation rather than surgical resection.

E. The lesion should be treated with androgen supplementation because low androgen levels are the usual cause of male hepatic adenomas.

83. A 52-year-old has a 4-cm peripheral hepatic mass with classic discontinuous peripheral nodular enhancement and progressive centripetal fill-in. She is asymptomatic. What is the most appropriate management?

A. Reassure and observe because the imaging is classic for a benign hemangioma and no treatment is needed in an asymptomatic patient.

B. Biopsy the lesion because vascular imaging features are nonspecific and tissue is required before a hemangioma can be diagnosed.

C. Resect urgently because every hemangioma larger than 3 cm has a high short-term risk of spontaneous rupture.

D. List for transplantation because benign vascular tumors frequently transform into hepatocellular carcinoma in normal liver.

E. Start anticoagulation because slow blood flow within a hemangioma is a common cause of portal vein thrombosis.

84. A 61-year-old with compensated cirrhosis has no focal lesion on ultrasound and a normal AFP. What is the appropriate HCC surveillance strategy?

A. Stop surveillance permanently because normal ultrasound and AFP establish negligible future HCC risk.

B. Continue surveillance at approximately 6-month intervals because a normal current study does not remove the ongoing HCC risk of cirrhosis.

C. Repeat imaging every month because six-month intervals allow most HCCs to become metastatic before detection.

D. Use PET-CT as the routine surveillance test because ultrasound has no accepted role in cirrhotic HCC screening.

E. Perform annual liver biopsy because microscopic HCC is more reliably detected histologically than by imaging surveillance.

85. A 57-year-old with a well-differentiated neuroendocrine tumor has liver-dominant metastatic disease that is unresectable but otherwise controlled, with no extrahepatic progression. Which statement about liver transplantation is most accurate?

A. Any metastatic neuroendocrine tumor is an absolute contraindication to transplantation regardless of biology or distribution.

B. Transplantation is routine first-line therapy for all liver metastases because immunosuppression prevents extrahepatic progression.

C. A high-grade rapidly progressive neuroendocrine carcinoma is the ideal transplant indication because aggressive tumors benefit most from graft replacement.

D. Transplantation may be considered only in highly selected patients with favorable tumor biology and controlled extrahepatic disease at experienced centers.

E. TIPS is the preferred definitive therapy because neuroendocrine liver metastases are driven by portal venous hypertension.

86. A 38-year-old has multifocal hepatic epithelioid hemangioendothelioma confined to the liver and not amenable to resection. Which statement is most accurate?

A. Liver transplantation can be an accepted option in selected patients because this rare vascular tumor may have favorable post-transplant outcomes despite multifocality.

B. Transplantation is contraindicated because all vascular liver tumors behave like angiosarcoma and universally recur within months.

C. Observation is mandatory because epithelioid hemangioendothelioma never causes progressive liver disease or metastasis.

D. Systemic corticosteroids are curative because the tumor is an inflammatory pseudotumor rather than a vascular neoplasm.

E. TIPS is definitive therapy because portal hypertension is the primary driver of tumor growth and multifocal spread.

87. A 55-year-old with cirrhosis has a new 2-cm indeterminate nodule that does not meet definitive imaging criteria for HCC. What is the most appropriate next step?

A. Assume HCC and award transplant exception priority because any nodule above 1 cm in cirrhosis is diagnostic of cancer.

B. Ignore the lesion until symptoms develop because small indeterminate nodules do not require short-interval reassessment.

C. Perform TIPS because lowering portal pressure converts indeterminate nodules into benign regenerative lesions.

D. Use guideline-based repeat high-quality imaging or biopsy according to the lesion category and how the result would affect management.

E. Start systemic HCC therapy because imaging uncertainty is itself evidence of microscopic vascular invasion.

88. A 58-year-old with decompensated cirrhosis has a MELD score of 14, recurrent ascites, and one hospitalization for encephalopathy. When should transplant evaluation begin?

A. Only after the MELD score exceeds 25 because lower scores do not justify transplant-center evaluation.

B. Only after dialysis is required because renal failure is the first decompensation that meaningfully predicts waitlist mortality.

C. Now, because clinical decompensation warrants early transplant referral even when the MELD score is not very high.

D. After two years of stable abstinence from all medications because pharmacologic therapy must be exhausted before referral.

E. After a first episode of hepatocellular carcinoma because nonmalignant decompensation is not a recognized indication for evaluation.

89. A 67-year-old with MASH cirrhosis is being evaluated for transplant. He has diabetes, obesity, and exertional chest pressure. Which evaluation is especially important?

A. No cardiac evaluation is needed if resting ejection fraction is normal because cirrhosis protects against coronary atherosclerosis.

B. Coronary disease is irrelevant after transplant because calcineurin inhibitors reduce long-term cardiovascular event rates.

C. Comprehensive cardiovascular risk assessment because occult coronary and structural heart disease can substantially affect transplant candidacy and perioperative risk.

D. Only pulmonary function testing is required because metabolic liver disease does not confer excess cardiovascular risk.

E. TIPS should be performed before cardiac testing because portal decompression eliminates ischemic symptoms in most MASH candidates.

90. A 50-year-old with cirrhosis and well-controlled HIV has undetectable HIV RNA, CD4 count 420/μL, and no opportunistic infection. Which statement about liver transplantation is most accurate?

A. Any history of HIV infection is an absolute contraindication because post-transplant immunosuppression causes universal uncontrolled viremia.

B. HIV infection is acceptable only if all antiretroviral therapy can be stopped permanently after liver transplantation.

C. A CD4 count above 200 eliminates the need to evaluate drug interactions between antiretrovirals and calcineurin inhibitors.

D. HIV-positive candidates can receive only living-donor grafts because deceased-donor allocation prohibits transplantation in this population.

E. Well-controlled HIV infection is not by itself a contraindication to liver transplantation when immunologic, infectious, and adherence criteria are acceptable.

91. A candidate for liver transplantation has active uncontrolled bacterial sepsis with hemodynamic instability. What is the most appropriate listing decision?

A. Proceed immediately because replacing the liver is the most reliable method to eradicate any systemic bacterial infection.

B. Permanently exclude the patient because any bacterial infection at any time is an absolute lifetime contraindication to transplantation.

C. Begin induction immunosuppression before infection treatment because T-cell suppression improves antibiotic penetration into infected tissues.

D. Defer transplantation until the infection is adequately controlled because uncontrolled sepsis creates prohibitive perioperative and immunosuppression risk.

E. Perform TIPS instead because uncontrolled sepsis is a specific indication for portal decompression in all transplant candidates.

92. A 64-year-old transplant candidate has severe frailty, low grip strength, sarcopenia, and frequent falls but no fixed neurologic disease. Which approach is most appropriate?

A. Remove the patient permanently from transplant consideration because any frailty measure predicts certain graft failure after transplantation.

B. Restrict dietary protein to reduce fall risk because muscle catabolism improves when nitrogen intake is minimized in cirrhosis.

C. Treat frailty as a dynamic risk factor with nutrition, physical rehabilitation, and repeated assessment rather than using frailty alone as an automatic permanent exclusion.

D. Ignore frailty because only MELD score predicts waitlist mortality and postoperative outcomes in liver transplantation.

E. Use bed rest to conserve calories because exercise before transplantation accelerates sarcopenia and hepatic decompensation.

93. A patient with decompensated cirrhosis has a healthy adult sibling interested in living donation. Which principle is essential during donor evaluation?

A. Recipient urgency should override donor risk because living donation is justified whenever it shortens time to transplantation.

B. The donor evaluation should be performed only by the recipient's hepatologist to keep medical decisions aligned with recipient benefit.

C. A biologically related donor can waive formal psychosocial assessment because family relationships establish voluntary consent.

D. The donor must undergo independent medical and psychosocial evaluation focused on minimizing donor risk and ensuring voluntary informed consent without coercion.

E. Potential donors should be promised zero long-term risk because modern partial hepatectomy has no meaningful morbidity or mortality.

94. A cirrhotic patient has chronic kidney disease with eGFR 24 mL/min for more than three months and evidence that renal dysfunction is unlikely to recover after isolated liver transplantation. Which transplant question should be addressed?

A. Whether simultaneous liver-kidney transplant is mandatory for every patient whose eGFR ever falls below 60 mL/min.

B. Whether kidney transplantation should always precede liver transplantation because kidney grafts tolerate portal hypertension better than native kidneys.

C. Whether the patient meets current medical and policy criteria for simultaneous liver-kidney transplantation rather than assuming renal recovery after liver transplant alone.

D. Whether renal dysfunction can be ignored because all chronic kidney disease reverses promptly once portal hypertension is corrected by transplantation.

E. Whether TIPS automatically restores chronic intrinsic kidney disease enough to eliminate any need for renal assessment.

95. A transplant candidate has treated HCC within accepted criteria but also a newly diagnosed extrahepatic colon cancer with metastatic disease. How should this affect liver transplant candidacy?

A. Proceed with liver transplantation because the HCC indication automatically overrides all other active malignancies.

B. Active extrahepatic metastatic malignancy generally precludes liver transplantation because immunosuppression and limited prognosis make transplant benefit unacceptable.

C. List urgently because immunosuppression usually controls metastatic colon cancer after transplantation.

D. Ignore the colon cancer if the MELD score is high because allocation rules prohibit considering nonhepatic prognosis.

E. Perform TIPS first because portal decompression reduces metastatic progression and restores transplant eligibility.

96. A 45-year-old with acute liver failure has INR 2.3 and new encephalopathy but no known chronic liver disease. Which definition best fits this syndrome?

A. Acute liver failure requires ascites for at least six months before encephalopathy develops.

B. Acute liver failure is defined by aminotransferases above 10,000 U/L regardless of INR or mental status.

C. Acute liver failure requires biopsy-proven cirrhosis plus a new precipitating infection.

D. Acute liver failure is synonymous with acute-on-chronic liver failure and requires a prior history of portal hypertension.

E. Acute liver failure is acute hepatic injury with coagulopathy and hepatic encephalopathy in a patient without established cirrhosis.

97. A 27-year-old presents 24 hours after a large acetaminophen ingestion with AST 5,800 U/L, INR 2.0, and confusion. Which treatment should be started immediately?

A. Activated charcoal alone because N-acetylcysteine is ineffective once aminotransferases have risen above 1,000 U/L.

B. Intravenous N-acetylcysteine while arranging intensive care and early transplant-center evaluation.

C. High-dose corticosteroids because acetaminophen toxicity causes immune-mediated centrilobular hepatitis.

D. Hemodialysis as the sole therapy because extracorporeal removal replaces the need for antidotal treatment after 24 hours.

E. Ursodeoxycholic acid because enhancing bile flow is the most effective way to prevent acetaminophen-related hepatic necrosis.

98. A patient with non-acetaminophen acute liver failure of indeterminate cause has early encephalopathy and no contraindication to therapy. Which supportive treatment may improve transplant-free survival in selected patients?

A. Chronic lactulose infusion is mandatory because ammonia reduction alone reverses the hepatic necrosis of acute liver failure.

B. Intravenous N-acetylcysteine can be considered even when acetaminophen toxicity is not established.

C. High-dose vitamin K is curative because acute liver failure is primarily a vitamin K deficiency syndrome.

D. Routine prophylactic plasma transfusion should normalize the INR because serial INR is otherwise useless for prognostication.

E. Nonselective beta-blockade should be started because portal pressure reduction prevents cerebral edema in acute liver failure.

99. A patient with acute liver failure develops grade III encephalopathy, serum ammonia 190 µmol/L, and acute kidney injury. Which ICU strategy may help control ammonia and cerebral-edema risk?

A. Use intermittent large-volume paracentesis because removal of ascites is the most efficient extracorporeal method for clearing ammonia.

B. Induce profound hypernatremia above 170 mEq/L because extreme sodium elevation reliably prevents intracranial hypertension without complications.

C. Use continuous renal replacement therapy when indicated because it can support renal function and enhance ammonia clearance while avoiding large hemodynamic shifts.

D. Begin chronic benzodiazepine sedation as monotherapy because GABA agonism protects the brain from ammonia-mediated astrocyte swelling.

E. Delay renal replacement until potassium exceeds 7 mEq/L because ammonia concentration has no role in cerebral-edema risk.

100. A 36-year-old with acute liver failure has rapidly worsening encephalopathy and signs concerning for intracranial hypertension. Which general management principle is most appropriate?

A. Transfer can wait until fixed pupillary dilation develops because early neurologic changes do not influence transplant timing.

B. Perform routine lumbar puncture to directly measure intracranial pressure because coagulopathy does not increase procedural risk in acute liver failure.

C. Give large volumes of hypotonic fluid because lowering serum osmolality reduces astrocyte swelling and intracranial pressure.

D. Use sedating benzodiazepines without neurologic monitoring because serial mental-status assessment does not contribute to prognosis.

E. Manage in an experienced intensive care and transplant center with measures to prevent cerebral edema and expedite transplant assessment.

101. A 58-year-old in shock from massive gastrointestinal bleeding develops AST 9,000 U/L, ALT 7,500 U/L, LDH markedly elevated, and only modest bilirubin elevation. Which cause of acute hepatic injury is most likely?

A. Hypoxic (ischemic) hepatitis from profound hepatic hypoperfusion during shock.

B. Primary biliary cholangitis because cholestatic autoimmune disease commonly causes aminotransferases above 5,000 U/L during shock.

C. Chronic hepatitis C because untreated HCV typically causes abrupt aminotransferase spikes above 7,000 U/L with high LDH.

D. Hemochromatosis because iron overload produces hyperacute hepatocyte necrosis immediately after a hypotensive episode.

E. Gilbert syndrome because reduced bilirubin conjugation explains the extreme aminotransferase elevation during hemodynamic collapse.

102. A patient with acute liver failure has negative viral studies and no drug exposure history. IgG is markedly elevated, ANA is strongly positive, and biopsy shows severe interface hepatitis with plasma cells. Which etiology is most likely?

A. Acetaminophen toxicity despite no exposure and a biopsy dominated by immune interface activity.

B. Budd-Chiari syndrome despite normal hepatic venous imaging and no sinusoidal congestion.

C. Autoimmune hepatitis causing acute liver failure with immune-mediated interface injury.

D. Primary sclerosing cholangitis despite normal cholangiography and absence of duct-centered injury.

E. Hereditary hemochromatosis despite no iron overload and an abrupt inflammatory presentation.

103. A 41-year-old with acute liver failure from Amanita phalloides ingestion has worsening coagulopathy and encephalopathy despite supportive therapy. What is the most important next step?

A. Discharge once gastrointestinal symptoms improve because hepatic toxicity ends when the initial vomiting and diarrhea resolve.

B. Start chronic beta-blockade because portal-pressure reduction is the only intervention that changes amatoxin-associated prognosis.

C. Urgent transfer to a liver transplant center because amatoxin poisoning can cause rapidly progressive hepatic failure requiring transplantation.

D. Delay transplant referral until bilirubin exceeds 30 mg/dL because early coagulopathy has little prognostic significance in mushroom poisoning.

E. Perform elective TIPS because decompression of portal pressure removes amatoxin from the systemic circulation.

104. A pregnant patient at 35 weeks develops nausea, abdominal pain, hypoglycemia, rising bilirubin, INR 2.0, and encephalopathy. Platelets are mildly reduced. Which management is most appropriate?

A. Continue pregnancy to term because delivery does not affect the maternal course of acute fatty liver of pregnancy.

B. Suspect acute fatty liver of pregnancy and expedite delivery with intensive maternal supportive care and transplant-center involvement if liver failure progresses.

C. Start long-term azathioprine because acute fatty liver of pregnancy is primarily an autoimmune hepatitis flare.

D. Perform TIPS before delivery because portal decompression is the definitive treatment for pregnancy-related acute liver failure.

E. Treat with ursodeoxycholic acid alone because the syndrome is a benign form of intrahepatic cholestasis of pregnancy.

105. A patient with acute liver failure from suspected HSV has rapidly progressive coagulopathy while PCR is pending. Which therapeutic principle is most appropriate?

A. Do not delay intravenous acyclovir when HSV hepatitis is reasonably suspected because early treatment can be lifesaving.

B. Wait for liver biopsy confirmation because antiviral treatment before histology obscures the diagnosis and worsens transplant outcomes.

C. Use oral acyclovir because intravenous therapy increases rather than decreases mortality in fulminant HSV hepatitis.

D. Treat with hepatitis B immune globulin because HSV hepatitis depends on surface-antigen expression for viral entry.

E. Avoid antiviral therapy in acute liver failure because drug clearance is reduced and all nucleoside analogues are contraindicated.

106. A patient with acute liver failure is being considered for the highest urgency transplant status. Which general principle best describes such status designations?

A. They are assigned to every patient with cirrhosis once the MELD score exceeds 20 regardless of chronicity or clinical stability.

B. They are based only on serum bilirubin and do not incorporate encephalopathy, ventilation, dialysis, or timing of liver failure.

C. They are reserved for narrowly defined patients with a very high risk of imminent death from acute graft or native-liver failure and require specific policy criteria.

D. They are optional center labels that do not affect allocation priority and are not governed by national transplant policy.

E. They are used primarily for stable HCC candidates because tumor progression creates the highest immediate risk of death within days.

107. A 49-year-old with alcohol-associated hepatitis has jaundice, tender hepatomegaly, bilirubin 18 mg/dL, INR 2.1, and no active infection or gastrointestinal bleeding. The Maddrey discriminant function indicates severe disease. Which treatment is most appropriate?

A. Begin high-dose azathioprine because steroid-sparing therapy improves short-term survival in acute alcohol-associated hepatitis.

B. Begin prednisolone after confirming that major contraindications are absent and reassess early response with a validated score.

C. Use pentoxifylline as first-line therapy because it is superior to corticosteroids for 28-day survival.

D. Start prophylactic broad-spectrum antibiotics as the sole disease-specific therapy even when no infection is identified.

E. Delay all disease-specific therapy until six months of abstinence have been documented because treatment response cannot be assessed earlier.

108. A patient receiving prednisolone for severe alcohol-associated hepatitis has a high Lille score after one week. What is the best interpretation?

A. The patient has an excellent corticosteroid response and should receive a higher dose for at least six months.

B. The score demonstrates occult portal vein thrombosis and mandates immediate anticoagulation.

C. The score establishes alcoholic cirrhosis and eliminates the need for further transplant evaluation.

D. The score measures lifetime relapse risk and should be used to determine candidacy independent of clinical assessment.

E. The patient is a corticosteroid nonresponder, so prolonged corticosteroid treatment is unlikely to provide benefit.

109. A 38-year-old hospitalized with severe alcohol-associated hepatitis is being considered for early liver transplantation. He has no prior rehabilitation attempts, strong family support, insight into his

alcohol use disorder, and a low predicted chance of medical recovery. Which principle is most appropriate?

A. Transplantation is categorically prohibited until exactly six months of abstinence have elapsed regardless of mortality risk.

B. Alcohol-associated liver disease is an absolute contraindication to transplantation because recurrence is universal.

C. Only the serum ethanol level at admission should determine candidacy because psychosocial assessment is unreliable.

D. Transplant candidacy should use individualized psychosocial and medical assessment rather than an inflexible six-month abstinence requirement.

E. Candidacy should be decided solely by MELD score because relapse risk and social support are irrelevant after transplantation.

110. A 56-year-old with decompensated alcohol-associated cirrhosis asks what intervention most improves long-term prognosis while awaiting transplant evaluation. Which recommendation is most important?

A. Routine therapeutic phlebotomy to reduce hepatic iron stores regardless of ferritin or transferrin saturation.

B. Sustained alcohol abstinence supported by evidence-based treatment of alcohol use disorder.

C. Long-term systemic corticosteroids to suppress inflammatory injury from alcohol exposure.

D. A high-dose vitamin A regimen because fat-soluble vitamin replacement reverses portal hypertension.

E. Elective portocaval shunt surgery before transplant evaluation to prevent further hepatic decompensation.

111. A patient with cirrhosis from alcohol-associated liver disease has macrocytosis, AST 160 U/L, ALT 70 U/L, and bilirubin 2.4 mg/dL. Which laboratory pattern is most characteristic of ongoing alcohol-associated hepatic injury?

A. ALT is usually more than ten times the AST because alcohol selectively injures periportal hepatocytes.

B. Alkaline phosphatase is always greater than 2,000 U/L while aminotransferases remain completely normal.

C. AST is typically higher than ALT, often with an AST-to-ALT ratio greater than 2, while aminotransferases are usually below the thousands.

D. Aminotransferases routinely exceed 10,000 U/L in uncomplicated chronic alcohol exposure.

E. Isolated unconjugated hyperbilirubinemia with normal AST and ALT is the defining biochemical pattern of alcohol-associated hepatitis.

112. A malnourished patient with decompensated alcohol-associated cirrhosis is admitted with ascites and sarcopenia. Which nutritional approach is most appropriate?

A. Restrict protein to less than 20 g/day indefinitely because dietary protein is the principal cause of hepatic encephalopathy.

B. Use prolonged fasting between meals because hepatic glycogen stores are increased in cirrhosis.

C. Avoid enteral nutrition because it increases portal pressure and precipitates variceal bleeding.

D. Provide only intravenous dextrose because dietary fat and protein cannot be metabolized in decompensated cirrhosis.

E. Provide adequate calories and protein with frequent meals and a late-evening snack rather than routinely restricting protein.

113. A 44-year-old with alcohol-associated cirrhosis has been abstinent for four months and shows improving bilirubin and INR. What is the best implication for transplant timing?

A. List immediately at the highest urgency status because improvement after abstinence predicts sudden irreversible failure.

B. Remove the patient permanently from transplant consideration because any improvement proves transplantation can never be needed.

C. Ignore changes in bilirubin and INR because alcohol abstinence does not alter liver-related prognosis once cirrhosis is established.

D. Proceed directly to TIPS solely to test whether the patient can remain abstinent after an invasive procedure.

E. Continue close reassessment because hepatic function may recover with abstinence, potentially changing urgency or need for transplantation.

114. A 52-year-old with obesity, type 2 diabetes, hypertension, and elevated aminotransferases has hepatic steatosis on imaging. Viral and autoimmune studies are negative. Which diagnosis best fits the current metabolic framework?

A. Wilson disease because metabolic syndrome is a typical phenotypic expression of copper accumulation in midlife.

B. Primary sclerosing cholangitis because steatosis plus diabetes constitutes a cholangiographic disorder even with normal bile ducts.

C. Metabolic dysfunction-associated steatotic liver disease when steatosis coexists with qualifying cardiometabolic risk factors and another dominant cause is absent.

D. Autoimmune hepatitis because obesity and insulin resistance are diagnostic markers of immune-mediated interface hepatitis.

E. Budd-Chiari syndrome because hepatic steatosis in a patient with hypertension implies hepatic venous outflow obstruction.

115. A 61-year-old with MASLD has platelets 210,000/mm3, AST 32 U/L, ALT 36 U/L, and a low FIB-4 score. What is the most appropriate next step for fibrosis assessment?

A. Perform liver biopsy immediately in every patient with imaging-confirmed steatosis regardless of noninvasive risk.

B. List for liver transplantation because the presence of steatosis alone establishes decompensated cirrhosis.

C. Manage metabolic risk factors and repeat noninvasive fibrosis assessment rather than proceeding directly to biopsy.

D. Obtain ERCP because fibrosis in MASLD is best staged by direct cholangiography.

E. Start nonselective beta-blocker therapy because a low FIB-4 score indicates clinically significant portal hypertension.

116. A patient with MASLD has an indeterminate FIB-4 score. Which test is most useful as a next noninvasive step to refine the probability of advanced fibrosis?

A. Diagnostic paracentesis because ascitic fluid protein directly stages fibrosis before ascites develops.

B. Vibration-controlled transient elastography or another validated liver-stiffness assessment.

C. Serum ammonia concentration because ammonia correlates linearly with collagen deposition in asymptomatic MASLD.

D. Routine ERCP because biliary pressure is the standard surrogate for fibrosis in metabolic liver disease.

E. Serum alpha-fetoprotein alone because AFP accurately distinguishes F2 from F4 fibrosis.

117. A 58-year-old with MASH cirrhosis loses weight through diet and exercise and has improved glycemic control. Which statement is most accurate?

A. Weight reduction is contraindicated in compensated MASH because any caloric deficit accelerates portal hypertension.

B. Glycemic control has no relevance to MASH because insulin resistance is unrelated to hepatic fat accumulation.

C. Only liver transplantation can alter the natural history of MASH once fibrosis is present.

D. A high-fructose diet is preferred because fructose bypasses insulin signaling and decreases hepatic triglyceride synthesis.

E. Sustained weight reduction and control of cardiometabolic risk factors are central treatment goals and can improve steatosis and steatohepatitis.

118. A patient with MASLD and type 2 diabetes is being evaluated for liver transplantation. Which comorbidity deserves especially careful assessment because it materially affects perioperative and long-term outcomes?

A. Seasonal allergic rhinitis because it is the dominant cause of perioperative mortality in metabolic liver disease.

B. Remote appendectomy because prior appendicitis predicts recurrent steatohepatitis after transplantation.

C. Cardiovascular disease, including occult coronary artery disease and heart failure risk.

D. Myopia because refractive error is a surrogate marker for portal venous thrombosis.

E. Lactose intolerance because disaccharidase deficiency is the principal driver of post-transplant graft loss.

119. A 55-year-old with obesity and diabetes drinks approximately 30 g of alcohol daily and has steatotic liver disease. Which concept best captures the interaction of these exposures?

A. Alcohol exposure protects against metabolic fibrosis whenever diabetes is present, so abstinence counseling is unnecessary.

B. The presence of metabolic risk factors excludes any contribution from alcohol by definition.

C. Metabolic and alcohol-related risk can coexist and act synergistically, so both cardiometabolic factors and alcohol use should be addressed.

D. Alcohol can be ignored below a level that causes intoxication because fibrosis risk begins only after withdrawal symptoms develop.

E. Metabolic steatosis and alcohol-associated liver disease cannot occur in the same patient and should never be considered together.

120. A 63-year-old with MASH cirrhosis is listed for transplantation. Which factor most directly indicates decompensation rather than compensated advanced chronic liver disease?

A. Development of clinically significant ascites requiring diuretic therapy and repeated paracentesis.

B. Hepatic steatosis on ultrasound without portal hypertension or synthetic dysfunction.

C. Type 2 diabetes treated with metformin while liver tests remain stable.

D. An elevated LDL cholesterol concentration without liver-related symptoms.

E. A mildly elevated ALT concentration with normal bilirubin, INR, and platelet count.

121. A patient with MASH cirrhosis and BMI 44 kg/m2 is referred for transplant evaluation. Which approach is most appropriate?

A. Assess obesity together with frailty, body composition, cardiometabolic risk, technical considerations, and center-specific criteria rather than using BMI alone as a complete measure of candidacy.

B. Exclude the patient permanently because any BMI above 40 kg/m2 is a universal statutory contraindication to liver transplantation.

C. Ignore obesity entirely because body habitus has no influence on surgical or cardiometabolic outcomes after transplantation.

D. Require rapid starvation-level weight loss before any other evaluation because loss of lean mass improves transplant outcomes.

E. Replace MELD with BMI for wait-list priority because obesity is the principal determinant of short-term mortality in cirrhosis.

122. A patient with MASH cirrhosis undergoes liver transplantation but later develops obesity, diabetes, and recurrent hepatic steatosis. Which preventive strategy is most appropriate?

A. Stop all immunosuppression because calcineurin inhibitor withdrawal is the only proven way to prevent recurrent metabolic disease.

B. Aggressively manage weight, diabetes, blood pressure, lipids, diet, and physical activity after transplantation.

C. Use chronic corticosteroids specifically to suppress recurrent steatosis despite their metabolic adverse effects.

D. Avoid statins in all liver transplant recipients because any post-transplant statin exposure causes graft failure.

E. Recommend unrestricted weight gain because post-transplant adiposity protects against recurrent fibrosis.

123. A 59-year-old with cirrhosis from MASH has normal aminotransferases. Which statement is most accurate?

A. Normal ALT excludes clinically important MASH with near-perfect sensitivity.

B. Advanced metabolic fibrosis always causes AST and ALT levels above 500 U/L.

C. Normal aminotransferases prove that portal hypertension cannot be present.

D. Aminotransferase normalization means hepatocellular carcinoma surveillance can be stopped.

E. Normal aminotransferase levels do not exclude advanced fibrosis or cirrhosis in MASLD.

124. A pregnant patient at 34 weeks has intense pruritus of the palms and soles, elevated serum bile acids, and mild aminotransferase elevation without synthetic dysfunction. Which diagnosis is most likely?

A. Acute fatty liver of pregnancy with impending hepatic encephalopathy.

B. Intrahepatic cholestasis of pregnancy with pruritus and elevated maternal bile acids.

C. HELLP syndrome despite normal blood pressure, platelet count, and absence of hemolysis.

D. Budd-Chiari syndrome despite patent hepatic veins and no ascites.

E. Primary biliary cholangitis newly defined solely by pregnancy-associated pruritus.

125. A 30-year-old at 33 weeks' gestation develops hypertension, right upper quadrant pain, hemolysis, AST elevation, and platelets of 55,000/mm3. Which diagnosis is most likely?

A. HELLP syndrome with hemolysis, elevated liver enzymes, and thrombocytopenia.

B. Intrahepatic cholestasis of pregnancy because thrombocytopenia and hemolysis are defining features of that disorder.

C. Gilbert syndrome because unconjugated hyperbilirubinemia causes hypertension and microangiopathic hemolysis.

D. Primary sclerosing cholangitis because platelet consumption is a typical manifestation of large-duct obstruction.

E. Wilson disease because pregnancy uniquely converts chronic copper overload into a microangiopathic syndrome.

126. A 47-year-old starts a new herbal supplement and two months later develops ALT 1,100 U/L, bilirubin 8 mg/dL, and negative viral and autoimmune testing. Imaging shows no obstruction. What is the best next step?

A. Continue the supplement to see whether aminotransferases plateau because withdrawal prevents accurate causality assessment.

B. Perform prophylactic ERCP because hepatocellular drug injury is usually caused by occult common bile duct obstruction.

C. Start lifelong corticosteroids in every suspected case because all idiosyncratic drug injury is autoimmune.

D. Rechallenge with a higher dose after enzymes decline because recurrence confirms safety for future use.

E. Stop the suspected agent and perform a structured evaluation for drug-induced liver injury while excluding competing causes.

127. A patient develops ALT 900 U/L and alkaline phosphatase 160 U/L after starting an antibiotic. The ALT is 15 times the upper limit of normal and alkaline phosphatase is 1.3 times the upper limit. Which injury pattern is most consistent with the R ratio?

A. Pure cholestatic injury because alkaline phosphatase elevation, however small, always defines cholestasis.

B. Mixed injury because any simultaneous ALT and alkaline phosphatase elevation is classified as mixed.

C. Isolated bilirubin transport failure because the R ratio is calculated from bilirubin alone.

D. Hepatocellular drug-induced liver injury, based on the strongly ALT-predominant R ratio.

E. Extrahepatic obstruction because an R ratio cannot be used when an antibiotic is involved.

128. A patient with cirrhosis develops a new acute kidney injury, jaundice, INR elevation, hepatic encephalopathy, and vasopressor-requiring circulatory failure after bacterial pneumonia. Which concept best describes this syndrome?

A. Compensated cirrhosis because infection-associated organ dysfunction does not alter the stage of chronic liver disease.

B. Isolated hepatorenal syndrome because simultaneous respiratory, circulatory, and neurologic failures are excluded from that diagnosis.

C. Acute liver failure because any encephalopathy with INR elevation is classified as acute liver failure even when cirrhosis is established.

D. Stable portal hypertension because organ failures are unrelated to short-term mortality in chronic liver disease.

E. Acute-on-chronic liver failure with acute decompensation and extrahepatic organ failure.

129. Which feature most strongly distinguishes acute-on-chronic liver failure from uncomplicated acute decompensation of cirrhosis?

A. A serum bilirubin above the laboratory upper limit of normal, even without organ dysfunction.

B. A history of cirrhosis longer than five years, regardless of the current clinical state.

C. The absence of any precipitating infection because infection excludes acute-on-chronic liver failure.

D. A single episode of mild pedal edema that resolves without therapy.

E. The presence and severity of organ failures that confer a high short-term risk of death.

130. A patient with end-stage liver disease has refractory ascites, recurrent admissions, severe frailty, and uncertain transplant candidacy. Which approach best integrates palliative care?

A. Delay palliative care until transplantation has been formally ruled out because palliative involvement precludes listing.

B. Stop all hepatology treatment once palliative care is consulted because the two approaches cannot coexist.

C. Introduce palliative care alongside disease-directed treatment to address symptoms, goals, caregiver needs, and advance care planning.

D. Use palliative care only for opioid prescribing because goals-of-care discussions are outside its role.

E. Avoid discussing prognosis until the final hours of life because earlier discussion increases liver-related mortality.

131. A patient listed for liver transplantation has repeated hospitalizations and tells the team that avoiding prolonged mechanical ventilation if recovery is unlikely is a priority. What is the best next step?

A. Remove the patient from the wait list because any treatment limitation is incompatible with transplantation.

B. Document the patient's values and surrogate decision-maker and incorporate them into an advance care plan that remains compatible with ongoing transplant candidacy.

C. Ignore the preference because transplant candidates cannot participate in advance care planning.

D. Ask the family to decide without involving the patient while decision-making capacity is intact.

E. Wait until cardiac arrest occurs before clarifying treatment preferences because earlier documentation has no clinical value.

132. A transplant candidate with cirrhosis asks which routine preventive intervention should generally be optimized before transplantation when feasible. Which is most appropriate?

A. Live vaccines should routinely be started immediately after transplantation while immunosuppression is highest.

B. All vaccination should be deferred indefinitely because cirrhosis prevents an effective immune response.

C. Age- and risk-appropriate vaccination, including non-live vaccines and indicated hepatitis immunization, should be updated before transplantation when possible.

D. Only tetanus vaccination matters because transplant immunosuppression does not increase susceptibility to vaccine-preventable infection.

E. Vaccines are contraindicated once a patient has any history of hepatic decompensation.

133. A 62-year-old candidate with high MELD is offered a liver from a 52-year-old donation-after-circulatory-death donor. Which counseling point is most accurate?

A. DCD grafts cannot be used for liver transplantation because warm ischemia invariably causes primary nonfunction.

B. DCD grafts can provide excellent survival but carry greater risk of ischemic cholangiopathy than comparable donation-after-brain-death grafts.

C. DCD grafts eliminate biliary complications because the bile ducts are protected during circulatory arrest.

D. DCD grafts are suitable only for recipients with hepatocellular carcinoma and cannot be allocated by medical urgency.

E. DCD grafts require lifelong anticoagulation because hepatic artery thrombosis is universal after circulatory-death donation.

134. A transplant program is considering a donor liver with marked macrovesicular steatosis. Which principle is most appropriate?

A. Macrovesicular steatosis has no effect on graft function because hepatic fat is removed during reperfusion.

B. Any steatosis is an absolute contraindication to transplantation regardless of recipient urgency.

C. Macrovesicular steatosis increases ischemia-reperfusion susceptibility, so graft risk should be assessed in donor-recipient context.

D. Steatotic grafts are preferred for recipients with severe portal hypertension because they resist reperfusion injury.

E. Microvesicular and macrovesicular steatosis have identical effects and never require pathologic distinction.

135. An HCV-uninfected candidate is offered an organ from an HCV-viremic donor. Which approach best reflects contemporary transplantation practice?

A. The organ must always be discarded because HCV transmission makes cure impossible after transplantation.

B. The organ may be considered with informed consent and a plan for prompt post-transplant direct-acting antiviral therapy and virologic monitoring.

C. The recipient should receive interferon for six months before transplantation to prevent donor-derived infection.

D. The organ can be used without discussing transmission because HCV RNA positivity has no implication for the recipient.

E. The recipient should remain untreated unless cirrhosis develops because antiviral therapy is ineffective under immunosuppression.

136. A liver from an HBsAg-negative, anti-HBc-positive donor is offered to an HBV-naive recipient. Which concern is most important?

A. Occult donor HBV can reactivate in the recipient, so prophylaxis and post-transplant HBV monitoring should be planned.

B. The donor liver cannot transmit HBV because HBsAg negativity proves complete viral eradication from hepatocytes.

C. Only hepatitis A can be transmitted from anti-HBc-positive donors, so HBV prophylaxis has no role.

D. The donor organ must be rejected in all circumstances because no antiviral prophylaxis reduces transmission risk.

E. The recipient should receive HCV direct-acting antivirals because anti-HBc positivity predicts occult HCV rather than HBV.

137. A 68-year-old donor has otherwise favorable characteristics but advanced age. What is the best principle for donor selection?

A. Any liver donor older than 60 years is absolutely prohibited by national policy.

B. Older donor age improves graft tolerance to prolonged cold ischemia and should be paired with the sickest recipient regardless of other factors.

C. Chronologic donor age is a risk modifier rather than an automatic exclusion, and acceptance should reflect graft quality, preservation, and recipient context.

D. Donor age is irrelevant once blood type matches because age does not affect graft outcomes.

E. Older livers can be used only as split grafts because whole-organ transplantation is contraindicated.

138. A donor liver has an unexpected mass discovered during procurement. Which action is most appropriate before implantation?

A. Implant the liver immediately because donor tumors cannot metastasize after transplantation.

B. Discard the liver automatically without attempting characterization because all donor lesions carry identical transmission risk.

C. Excise the lesion after implantation and avoid informing the recipient because donor findings are confidential from recipients.

D. Administer empiric chemotherapy to the recipient before transplantation because that eliminates all donor-derived cancer risk.

E. Characterize the lesion urgently and assess donor-derived malignancy risk with pathology and transplant-team input before deciding whether the organ is acceptable.

139. A medically urgent candidate is offered a higher-risk donor liver that is expected to function adequately but has a greater-than-average chance of graft complications. What is the central decision principle?

A. Balance the individualized risk of accepting the organ against the candidate's risk of death or deterioration while waiting for another offer.

B. Decline every higher-risk graft because donor quality should never be considered in relation to recipient urgency.

C. Accept every offered graft because wait-list mortality eliminates the need for informed consent.

D. Use donor age alone to determine acceptance because no recipient characteristic modifies organ-offer decisions.

E. Base acceptance exclusively on the candidate's chronological age and ignore disease severity or likely waiting time.

140. A healthy 34-year-old is being evaluated to donate a right hepatic lobe to her brother. Which ethical requirement is essential?

A. The donor must provide voluntary informed consent free of coercion and undergo an independent assessment focused on donor welfare.

B. The recipient's medical urgency overrides donor autonomy once the donor is a first-degree relative.

C. The donor may be accepted without psychosocial evaluation because family relationships eliminate coercion risk.

D. The transplant team should conceal donor surgical risks to prevent anxiety from interfering with altruistic intent.

E. Living donation is permissible only when the donor receives direct financial compensation for lost organ function.

141. During living donor evaluation, CT volumetry predicts an inadequate future liver remnant for the donor after the proposed right hepatectomy. What is the best next step?

A. Proceed because the recipient's need outweighs the donor's postoperative liver-failure risk.

B. Do not proceed with the proposed donation unless an alternative plan can provide an adequate donor remnant and acceptable safety.

C. Increase the recipient's immunosuppression because that compensates for a small donor remnant.

D. Perform preoperative TIPS in the donor because portal decompression guarantees remnant hypertrophy after hepatectomy.

E. Remove additional donor liver tissue because a smaller remnant regenerates faster and reduces donor risk.

142. Which potential living liver donor requires particular concern for occult liver disease before donation?

A. A donor with a healed ankle fracture from adolescence and normal current mobility.

B. A donor with corrected myopia and no systemic disease.

C. A donor with obesity, diabetes, and imaging evidence of hepatic steatosis.

D. A donor with remote tonsillectomy and normal laboratory testing.

E. A donor with blood type identical to the recipient but no metabolic risk factors.

143. A recipient of a partial living-donor graft develops severe cholestasis, coagulopathy, ascites, and persistent portal hypertension despite patent hepatic vessels. The graft-to-recipient size is very low. What is the most likely syndrome?

A. Hyperacute antibody-mediated rejection caused solely by recipient-donor height mismatch.

B. Small-for-size syndrome from inadequate functional graft mass relative to recipient demand.

C. Primary sclerosing cholangitis recurrence within hours of implantation.

D. Hepatic artery steal syndrome caused by an oversized graft compressing the portal vein.

E. Chronic ductopenic rejection, which typically develops during the first postoperative day.

144. A living donor asks how liver regeneration affects risk after partial hepatectomy. Which statement is most accurate?

A. The remnant liver can regenerate substantially, but regeneration does not eliminate the real perioperative risks of major hepatectomy.

B. The liver returns instantly to its preoperative mass, so donor liver failure cannot occur.

C. Regeneration occurs only in the transplanted graft and not in the donor remnant.

D. Regeneration prevents biliary, thrombotic, and infectious complications after donation.

E. Regeneration is impossible after right-lobe donation, which is why donors require lifelong liver replacement therapy.

145. A potential living donor reports that family members have threatened to withdraw housing support if she refuses donation. What is the best response by the donor team?

A. Proceed because pressure from relatives is acceptable when the intended recipient is critically ill.

B. Pause the donation process and address coercion confidentially, prioritizing the donor's right to decline without penalty.

C. Inform the recipient immediately that the donor is reluctant so the family can persuade her more effectively.

D. Allow donation if the donor signs a surgical consent form because a signature alone proves voluntariness.

E. Exclude the donor permanently from all medical care because ambivalence violates the ethics of living donation.

146. A small adult recipient is being considered for a split deceased-donor liver graft. What is the major technical principle?

A. The segmental graft must provide adequate functional mass with reliable arterial, portal, venous, and biliary anatomy for the intended recipient.

B. Split grafts can be used without regard to recipient size because all segments rapidly become full-sized before reperfusion.

C. Only the common bile duct matters because vascular anatomy does not affect segmental graft survival.

D. Split transplantation is contraindicated in all adults and can be performed only in neonates.

E. The two split grafts must always be transplanted into recipients with identical MELD scores regardless of anatomy or size.

147. A child with fulminant liver failure has no compatible deceased donor immediately available. An ABO-incompatible graft is being considered. Which principle is most accurate?

A. ABO incompatibility has no immunologic consequence because hepatocytes do not express blood-group antigens.

B. ABO-incompatible liver transplantation is universally fatal and is never performed under any circumstance.

C. ABO incompatibility affects only red-cell transfusion and has no association with biliary or antibody-mediated complications.

D. ABO incompatibility increases immunologic risk but may be considered in selected urgent situations using center-specific desensitization and immunosuppression strategies.

E. ABO-incompatible grafts require no additional monitoring once the donor operation is complete.

148. Preoperative imaging of a donor shows a replaced right hepatic artery arising from the superior mesenteric artery. What is the best interpretation?

A. This proves the donor has portal hypertension and the organ must be discarded.

B. This variant means the right lobe has no arterial blood supply and cannot survive transplantation.

C. Hepatic arterial variants are irrelevant because the liver receives all graft oxygen from the portal vein.

D. The finding establishes hepatic artery thrombosis before procurement and requires systemic thrombolysis in the donor.

E. This is an important anatomic variant that must be recognized for procurement and arterial reconstruction planning but is not automatically prohibitive.

149. A candidate has chronic complete portal vein thrombosis with cavernous transformation. Which statement about transplantation is most accurate?

A. Portal vein thrombosis increases technical complexity, but transplantation may still be feasible using thrombectomy or alternative portal inflow strategies in experienced centers.

B. Any portal vein thrombosis is an absolute contraindication to transplantation regardless of extent or available inflow.

C. Portal thrombosis improves surgical outcomes by reducing portal pressure before implantation.

D. The portal vein is unnecessary for graft perfusion because hepatic arterial flow alone supplies adequate nutrient inflow.

E. Cavernous transformation guarantees spontaneous recanalization during reperfusion, so preoperative planning is unnecessary.

150. Immediately after reperfusion of a liver graft, the recipient develops abrupt hypotension, bradycardia, decreased systemic vascular resistance, and hyperkalemia. What is the most likely phenomenon?

A. Chronic ductopenic rejection because bile-duct loss causes an immediate vasodilatory shock state.

B. Recurrent primary biliary cholangitis because antimitochondrial antibodies trigger bradycardia within seconds.

C. Late hepatic artery stenosis because arterial intimal hyperplasia occurs during the first minute after reperfusion.

D. Portopulmonary hypertension because pulmonary vascular remodeling begins only after graft reperfusion.

E. Postreperfusion syndrome causing abrupt cardiovascular instability immediately after graft reperfusion.

151. Within hours of transplantation, a recipient has refractory acidosis, severe coagulopathy, minimal bile production, profound hemodynamic instability, and rapidly rising aminotransferases. Doppler shows patent major vessels. Which diagnosis is most concerning?

A. Chronic rejection because progressive ductopenia commonly presents within the first postoperative hours.

B. Recurrent autoimmune hepatitis because autoantibody-mediated graft failure is immediate after implantation.

C. Anastomotic biliary stricture because fibrotic narrowing develops before postoperative day 1.

D. Post-transplant lymphoproliferative disorder because EBV-driven tumors commonly cause immediate graft failure.

E. Primary nonfunction of the liver graft causing severe early synthetic and metabolic failure.

152. A recipient has marked aminotransferase elevation and coagulopathy during the first postoperative week but then steadily improves without retransplantation. Which concept best fits this course?

A. Chronic antibody-mediated rejection because spontaneous normalization in the first week is characteristic.

B. Recurrent hepatitis C because viral fibrosis develops immediately and resolves without antiviral therapy.

C. Early allograft dysfunction from perioperative graft injury rather than irreversible primary nonfunction.

D. Post-transplant lymphoproliferative disorder because early tumor lysis causes transient aminotransferase elevation.

E. Anastomotic cholangiocarcinoma because biliary malignancy typically resolves as ischemia improves.

153. On postoperative day 2 after liver transplantation, AST rises abruptly and Doppler ultrasonography shows absent hepatic arterial flow. What is the most urgent next step?

A. Observe for several weeks because collateral arterial flow usually develops before biliary ischemia occurs.

B. Begin ursodeoxycholic acid alone because absent arterial flow is primarily a cholestatic medication-responsive process.

C. Perform colonoscopy because hepatic artery thrombosis most often reflects occult inflammatory bowel disease.

D. Reduce tacrolimus and wait for spontaneous recanalization because calcineurin toxicity is the usual cause of absent flow.

E. Obtain immediate confirmatory vascular imaging or operative assessment and pursue urgent revascularization when feasible.

154. Why can hepatic artery thrombosis cause particularly severe biliary injury after liver transplantation?

A. The bile ducts receive oxygen only from the portal vein after transplantation and therefore fail when portal flow increases.

B. Biliary epithelium has no vascular supply and is injured only by immunologic rejection.

C. Hepatic artery thrombosis directly blocks bile flow mechanically by filling the common bile duct with clot.

D. Bile ducts are supplied by lymphatic vessels rather than blood vessels, so arterial thrombosis cannot affect them.

E. The transplanted biliary epithelium depends predominantly on hepatic arterial perfusion through the peribiliary vascular plexus.

155. On postoperative day 3, a liver transplant recipient develops abdominal pain and rapidly accumulating bilious fluid in a surgical drain. What is the most likely complication?

A. Chronic ductopenic rejection because progressive bile-duct loss presents with external bile drainage in the first three days.

B. An early postoperative bile leak, often from an anastomosis or cut surface.

C. Recurrent primary sclerosing cholangitis because multifocal strictures commonly recur before hospital discharge.

D. Portal vein stenosis because reduced portal inflow causes bile to exit directly through surgical drains.

E. Calcineurin inhibitor nephrotoxicity because renal vasoconstriction produces bilious ascites.

156. A recipient develops acute kidney injury in the first 48 hours after a prolonged transplant operation with vasopressor exposure and major blood loss. Which mechanism is most likely?

A. Chronic calcineurin arteriolopathy because fixed vascular fibrosis develops before immunosuppression is started.

B. Recurrent hepatorenal syndrome because renal vasoconstriction cannot improve after successful graft implantation.

C. Glomerulonephritis from donor-specific antibodies because it is the universal cause of immediate postoperative oliguria.

D. Multifactorial perioperative acute tubular injury from hemodynamic instability, ischemia, and nephrotoxic exposures.

E. Obstructive nephropathy from the biliary anastomosis because bile-duct edema compresses both ureters after transplantation.

157. During liver transplantation, the recipient develops severe hyperkalemia just before reperfusion. Which action is most appropriate?

A. Ignore the potassium level because reperfusion invariably lowers serum potassium through hepatic uptake.

B. Treat hyperkalemia promptly and optimize acid-base status before reperfusion because potassium can rise further when graft effluent enters the circulation.

C. Give potassium chloride prophylactically because the graft consumes most circulating potassium after implantation.

D. Delay treatment until ventricular arrhythmia occurs because preemptive therapy interferes with graft function.

E. Use spironolactone as the fastest intraoperative treatment because its onset is immediate.

158. A recipient has persistent hypotension and diffuse surgical bleeding immediately after reperfusion despite adequate volume replacement. Which perioperative principle is most appropriate?

A. Normalize the INR with unlimited plasma regardless of bleeding because the INR alone precisely measures global hemostasis in cirrhosis.

B. Evaluate and correct surgical bleeding, hypothermia, acidosis, fibrinolysis, and coagulation-factor deficits using targeted hemostatic support.

C. Avoid all blood products because coagulation abnormalities after liver transplantation are always self-correcting within minutes.

D. Give aspirin immediately because diffuse operative bleeding is usually caused by platelet hyperactivity rather than coagulopathy.

E. Treat only with vitamin K because transplantation-associated coagulopathy never involves platelets, fibrinogen, or fibrinolysis.

159. A liver transplant recipient develops hypoxemia and pulmonary edema shortly after a massive transfusion, with bilateral infiltrates and no clear evidence of volume overload. Which complication should be considered?

A. Hepatopulmonary syndrome recurrence caused by immediate formation of new intrapulmonary shunts.

B. Chronic rejection because ductopenia directly causes noncardiogenic pulmonary edema.

C. TIPS dysfunction because every liver transplant contains a functioning transjugular portosystemic shunt.

D. Recurrent portopulmonary hypertension because pulmonary edema is the defining feature of pulmonary arterial vasoconstriction.

E. Transfusion-related acute lung injury causing acute noncardiogenic pulmonary edema after transfusion.

160. A recipient has rising lactate and transaminases after transplantation. Doppler shows absent portal venous flow while hepatic arterial flow is present. Which complication is most likely?

A. Isolated biliary anastomotic stricture because strictures abolish portal flow on Doppler.

B. Portal vein thrombosis causing loss of major graft inflow and acute graft dysfunction.

C. Chronic cellular rejection because T-cell activity mechanically occludes the portal vein within hours.

D. Calcineurin inhibitor toxicity because tacrolimus selectively stops portal flow while preserving arterial flow.

E. Post-transplant lymphoproliferative disorder because lymphoid proliferation typically occludes the portal vein on postoperative day 1.

161. After a technically difficult transplant, Doppler shows very high velocity at the portal anastomosis with a focal caliber change, and the patient has impaired graft function. What is the most likely issue?

A. Normal postoperative physiology because focal portal velocity acceleration always indicates excellent graft inflow.

B. Hepatic vein thrombosis because venous outflow obstruction cannot affect portal Doppler findings.

C. Hemodynamically significant portal vein stenosis at the anastomosis.

D. Bile-duct leak because biliary fluid directly accelerates blood through the portal anastomosis.

E. Acute cellular rejection because rejection produces a fixed focal narrowing visible within the portal lumen.

162. A recipient with substantial preoperative pulmonary hypertension becomes hemodynamically unstable during transplantation. Which physiologic concern is most important?

A. Severe pulmonary hypertension protects the right ventricle from volume changes by increasing coronary perfusion automatically.

B. Acute right-ventricular failure can occur when large shifts in preload and pulmonary vascular resistance exceed right-heart reserve.

C. Right-ventricular function is irrelevant during liver transplantation because the graft receives only portal venous blood.

D. Pulmonary vascular resistance always falls to zero after reperfusion, eliminating right-heart risk.

E. The principal risk is isolated left-atrial rupture rather than right-ventricular failure.

163. A recipient is receiving a liver from a donor with known bacteremia that was treated with active antibiotics before procurement and has no evidence of uncontrolled metastatic infection. What is the best principle?

A. Any donor bacteremia absolutely prohibits organ use because transmission cannot be prevented with antibiotics.

B. Donor blood-culture results are irrelevant because bacteria cannot be transmitted in vascularized organs.

C. The recipient needs no antimicrobial therapy if the liver appears normal at procurement.

D. Selected organs from bacteremic donors may be used when organism, treatment response, graft quality, and a recipient antimicrobial plan are carefully assessed.

E. Only viral donor infections matter because bacterial transmission never causes post-transplant sepsis.

164. A donor from an endemic area has unrecognized Strongyloides infection. Why is this important for transplant recipients?

A. Strongyloides remains confined to the donor intestine and cannot be transmitted with a liver graft.

B. Immunosuppression prevents Strongyloides dissemination by suppressing eosinophilic inflammation.

C. The infection causes only a self-limited rash and never produces pulmonary or gastrointestinal disease in recipients.

D. Strongyloides transmission is prevented by hepatitis B vaccination, making donor screening unnecessary.

E. Donor-derived Strongyloides can cause rapidly progressive hyperinfection under immunosuppression, so epidemiologic risk assessment and prompt treatment are important.

165. A donor is found to have active invasive fungal infection with bloodstream dissemination at the time of organ recovery. What is the most appropriate general approach?

A. Proceed routinely because systemic fungal infections are not transmissible through vascularized organs.

B. Use the organ without informing the recipient because donor infection data are not relevant to consent.

C. Treat this as a major transmission hazard and avoid use of the organ unless an exceptional, specifically assessed circumstance justifies the risk.

D. Rely on a single postoperative fluconazole dose because it eradicates all possible donor-derived fungi.

E. Prefer the organ specifically for the most immunosuppressed recipient because higher immunosuppression reduces fungal invasion.

166. A recipient develops fever and septic shock four days after transplantation, and another recipient from the same donor develops a similar unusual infection. What should the transplant team suspect?

A. Coincidental community infection only, because donor-derived infections cannot affect multiple recipients.

B. Acute cellular rejection, because rejection is routinely transmitted from one donor to multiple recipients.

C. A donor-derived infection and the need for urgent communication with organ-procurement and transplant safety networks.

D. Calcineurin inhibitor toxicity, because tacrolimus contamination produces identical sepsis syndromes across centers.

E. Recurrent cirrhosis, because chronic liver disease typically recurs synchronously in all recipients from one donor.

167. A donor had a remote, completely treated low-grade malignancy with a long disease-free interval. Which statement best describes organ-use decision making?

A. Any prior malignancy in a donor carries identical transmission risk and absolutely prohibits all organ donation.

B. A disease-free interval guarantees zero transmission risk, so recipient counseling is unnecessary.

C. Only donor age matters once cancer treatment is complete because tumor biology no longer affects transmission.

D. Organs from donors with prior malignancy are automatically allocated only to pediatric recipients regardless of urgency.

E. Transmission risk depends on tumor type, stage, treatment, and disease-free interval and should be balanced against recipient urgency with informed discussion.

168. A donor liver has been preserved for a prolonged cold ischemia time. Which graft process is most directly worsened by prolonged cold storage?

A. Ischemia-reperfusion injury with greater risk of early graft dysfunction.

B. Recipient autoantibody production before implantation because cold storage directly stimulates adaptive immunity in the recipient.

C. Chronic ductopenic rejection before reperfusion because bile-duct loss occurs only during static storage.

D. Recurrent metabolic liver disease because cold time determines the recipient's future insulin resistance.

E. Post-transplant lymphoproliferative disorder because EBV-driven B-cell proliferation begins in the preservation solution.

169. A transplant center uses normothermic machine perfusion for a marginal donor liver. What is a major potential advantage?

A. It eliminates the need for donor-recipient blood-group assessment because perfusion removes all endothelial antigens.

B. It guarantees zero biliary complications regardless of warm ischemia or graft anatomy.

C. It replaces the need for vascular anastomoses because the graft remains connected to the perfusion circuit after implantation.

D. It permanently prevents cellular rejection by washing donor leukocytes out of the organ.

E. It can provide physiologic perfusion that permits functional assessment and may reduce preservation-related injury in selected grafts.

170. A recipient becomes profoundly hypotensive immediately after unclamping the vena cava during transplantation. Which initial management principle is most appropriate?

A. Assume acute rejection and administer lymphocyte-depleting therapy before checking hemodynamics.

B. Treat only with diuretics because hypotension after unclamping always reflects intravascular volume excess.

C. Ignore the blood pressure for 30 minutes because postreperfusion hypotension is harmless and self-limited in every patient.

D. Rapidly assess preload, bleeding, cardiac function, vascular resistance, electrolytes, and reperfusion physiology while providing targeted hemodynamic support.

E. Clamp the hepatic artery permanently because arterial inflow is the usual cause of systemic hypotension after reperfusion.

171. A recipient develops severe lactic acidosis during the anhepatic phase of liver transplantation. Which explanation is most appropriate?

A. Loss of hepatic lactate clearance together with tissue hypoperfusion can cause lactate accumulation until graft function and hemodynamics improve.

B. The anhepatic phase increases hepatic lactate metabolism because the native liver is temporarily more efficient after explantation.

C. Lactate is generated only by the liver, so levels should fall to zero when the native liver is removed.

D. Lactic acidosis proves irreversible graft rejection before the graft has been implanted.

E. The finding is caused exclusively by biliary obstruction and should be treated with ERCP during the operation.

172. Three weeks after liver transplantation, a recipient develops rising aminotransferases. Doppler vascular flow is normal and biliary imaging shows no obstruction. Biopsy demonstrates portal inflammation, bile-duct injury, and venous endotheliitis. What is the most likely diagnosis?

A. Chronic ductopenic rejection because irreversible bile-duct loss is required within the first month.

B. Acute T-cell-mediated rejection with portal inflammation, bile-duct injury, and venous endotheliitis.

C. Hepatic artery thrombosis because normal Doppler flow excludes a histologic rejection pattern.

D. Recurrent MASH because metabolic steatohepatitis develops with endotheliitis within weeks.

E. Calcineurin inhibitor nephrotoxicity because renal arteriolar injury produces portal endotheliitis in the liver.

173. A liver transplant recipient has biopsy-proven moderate acute T-cell-mediated rejection with no uncontrolled infection. What is a typical initial treatment?

A. Increase immunosuppression, commonly with a short course of high-dose corticosteroids while ensuring maintenance therapy is therapeutic.

B. Stop all immunosuppression so donor-reactive lymphocytes become exhausted.

C. Treat with direct-acting antivirals alone because all acute rejection is caused by occult hepatitis C.

D. Perform urgent retransplantation before attempting medical therapy because acute cellular rejection is uniformly irreversible.

E. Use plasmapheresis alone because T-cell-mediated rejection is driven exclusively by circulating antibody.

174. A recipient has severe biopsy-proven T-cell-mediated rejection that does not improve after adequate corticosteroid therapy. Which next strategy is most appropriate?

A. Stop tacrolimus and steroids simultaneously because persistent rejection indicates excessive immunosuppression.

B. Begin ursodeoxycholic acid as the sole therapy because steroid resistance proves a biliary stricture.

C. Treat with lactulose because hepatic encephalopathy reverses the immune infiltrate.

D. Escalate immunosuppression with a lymphocyte-depleting agent such as antithymocyte globulin after reassessing infection risk and the diagnosis.

E. Observe without therapy because steroid-resistant rejection always resolves spontaneously within days.

175. A recipient several years after transplantation develops progressive cholestasis. Biopsy shows loss of interlobular bile ducts in many portal tracts and obliterative arteriopathy. What is the most likely diagnosis?

A. Acute uncomplicated T-cell-mediated rejection because ductopenia is not part of chronic graft injury.

B. Recurrent hepatitis A because HAV produces progressive bile-duct disappearance years after infection.

C. Calcineurin inhibitor nephrotoxicity because renal arteriolar hyalinosis causes hepatic ductopenia.

D. Simple anastomotic stricture because a focal mechanical lesion produces diffuse obliterative arteriopathy on biopsy.

E. Chronic rejection with progressive bile-duct loss (ductopenia) and vascular injury.

176. A liver transplant recipient has graft dysfunction, strong donor-specific HLA antibodies, microvascular injury on biopsy, and diffuse C4d staining after other causes are excluded. Which diagnosis should be considered?

A. Pure calcineurin inhibitor toxicity because donor-specific antibodies and C4d are unrelated to humoral immunity.

B. Recurrent Wilson disease because copper accumulation causes donor-specific HLA antibodies.

C. Hepatic artery stenosis because arterial narrowing itself generates diffuse C4d deposition.

D. Antibody-mediated rejection supported by donor-specific antibodies, C4d, and microvascular injury.

E. Post-transplant diabetes because hyperglycemia produces microvascular C4d staining in the graft.

177. A stable liver transplant recipient asks whether maintenance immunosuppression can be stopped because the liver is relatively tolerogenic. What is the best response?

A. Routine complete withdrawal is not standard care; any attempt at operational-tolerance protocols should occur only in carefully selected patients under specialized supervision.

B. All liver recipients should stop immunosuppression after one year because rejection cannot occur beyond that time.

C. Tacrolimus should be stopped whenever liver tests are normal because normal aminotransferases prove immunologic tolerance.

D. Immunosuppression is needed only for the first postoperative week because donor lymphocytes then disappear completely.

E. Complete withdrawal is required to prevent chronic rejection because maintenance drugs are the primary cause of ductopenia.

178. An adolescent liver transplant recipient develops repeated late rejection episodes, and tacrolimus troughs fluctuate widely. Which issue should be assessed urgently?

A. Universal graft senescence because late rejection in adolescents is unrelated to medication exposure.

B. Occult hemochromatosis because iron overload is the main cause of fluctuating tacrolimus concentrations.

C. Excess dietary protein because protein intake directly triggers donor-specific T-cell activation.

D. Prior childhood vaccination because vaccine memory is the leading cause of late graft rejection.

E. Medication nonadherence, including developmental, psychosocial, access, and family-system barriers.

179. A liver transplant recipient has mild unexplained aminotransferase elevation. Tacrolimus level is therapeutic and ultrasound is normal. What is the best way to establish whether rejection is present?

A. Diagnose rejection from any ALT elevation because biopsy adds no useful information after transplantation.

B. Exclude rejection if the tacrolimus trough is therapeutic because rejection cannot occur at target levels.

C. Perform splenectomy because histologic confirmation is unnecessary when ultrasound is normal.

D. Obtain a liver biopsy when clinically appropriate because rejection cannot be diagnosed reliably from liver tests or drug levels alone.

E. Use serum ammonia as the definitive rejection biomarker because it directly measures portal inflammation.

180. A recipient develops late acute rejection after independently reducing immunosuppression. Why can late rejection be clinically important?

A. Late rejection is harmless because the graft becomes completely immune privileged after the first year.

B. Late rejection can be harder to reverse and can progress to chronic rejection and graft loss.

C. Late rejection affects only kidney function and does not injure the liver graft.

D. Late rejection always represents recurrent viral hepatitis rather than alloimmunity.

E. Late rejection cannot occur unless the recipient is ABO-incompatible with the donor.

181. Biopsy of a liver allograft shows prominent plasma-cell-rich portal inflammation and interface hepatitis several years after transplant. Autoimmune hepatitis was not the original disease. Which consideration is most appropriate?

A. The finding proves recurrent autoimmune hepatitis even when the native liver never had autoimmune disease.

B. The finding excludes alloimmune injury because plasma cells cannot participate in transplant rejection.

C. The biopsy is diagnostic of biliary obstruction because interface hepatitis occurs only with mechanical cholestasis.

D. Plasma-cell-rich rejection should be considered after excluding recurrent or de novo autoimmune hepatitis, viral infection, and other causes of interface injury.

E. No treatment is needed because plasma-cell-rich inflammation is a normal histologic feature of long-term graft adaptation.

182. Six months after transplantation, a recipient develops cholestatic liver tests. MRCP shows a short focal narrowing at the duct-to-duct biliary anastomosis with upstream dilation. What is the most likely diagnosis?

A. Diffuse ischemic cholangiopathy because that typically presents as a single short anastomotic narrowing only.

B. Chronic rejection because bile-duct loss produces a focal surgically localized narrowing with upstream dilation.

C. Recurrent hepatitis B because HBV causes mechanical narrowing at the duct-to-duct anastomosis.

D. An anastomotic biliary stricture causing a short focal narrowing at the surgical bile-duct connection.

E. Tacrolimus nephrotoxicity because renal vasoconstriction produces extrahepatic bile-duct stenosis.

183. A recipient of a DCD liver develops recurrent cholangitis and cholestasis. Cholangiography shows multiple intrahepatic and extrahepatic nonanastomotic strictures with casts despite a patent hepatic artery. What is the most likely diagnosis?

A. Simple anastomotic stricture because a single surgical narrowing causes diffuse intrahepatic duct destruction.

B. Acute cellular rejection because rejection routinely creates multiple biliary casts with a completely normal biopsy.

C. Ischemic cholangiopathy from peribiliary ischemic injury with nonanastomotic strictures and casts.

D. Recurrent alcohol-associated liver disease because alcohol exposure causes multifocal biliary strictures soon after transplant.

E. Gilbert syndrome because impaired bilirubin conjugation produces biliary casts and cholangitis.

184. A liver transplant recipient develops progressive chronic kidney disease while graft function is excellent and tacrolimus troughs have been consistently high. What is the best management principle?

A. Increase tacrolimus because calcineurin inhibitors dilate renal arterioles and slow chronic kidney disease.

B. Reduce calcineurin inhibitor exposure when safely possible and optimize other renal risk factors, often using a renal-sparing immunosuppressive strategy.

C. Stop all antihypertensive therapy because blood-pressure control accelerates calcineurin nephrotoxicity.

D. Use repeated iodinated contrast studies to stimulate renal recovery through osmotic diuresis.

E. Ignore kidney function because chronic renal disease does not affect long-term survival after liver transplantation.

185. A liver transplant recipient develops new hypertension after tacrolimus initiation. Which mechanism contributes to this complication?

A. Tacrolimus causes obligatory systemic vasodilation and sodium wasting, producing chronic hypotension rather than hypertension.

B. Hypertension after transplant is caused only by recurrent portal hypertension and is unrelated to immunosuppression.

C. Calcineurin inhibitors suppress sympathetic tone so completely that hypertension cannot develop during therapy.

D. Post-transplant hypertension occurs only when hepatic artery thrombosis is present.

E. Calcineurin-inhibitor-mediated renal vasoconstriction and sodium retention can increase blood pressure.

186. A recipient develops marked hyperglycemia after transplantation while receiving tacrolimus and corticosteroids. Which statement is most accurate?

A. Immunosuppressive drugs cannot affect glucose metabolism, so all post-transplant hyperglycemia is laboratory error.

B. Post-transplant diabetes is multifactorial and can be promoted by calcineurin inhibitors, corticosteroids, weight gain, and preexisting metabolic risk.

C. Post-transplant diabetes should be untreated because hyperglycemia reduces rejection risk.

D. Only recurrent hepatitis C can cause diabetes after transplantation, regardless of obesity or medications.

E. Diabetes resolves permanently when corticosteroids are tapered, so long-term metabolic follow-up is unnecessary.

187. A recipient has severe hypertriglyceridemia after conversion to an mTOR inhibitor. What is the best approach?

A. Ignore the lipid level because post-transplant dyslipidemia has no cardiovascular consequence.

B. Treat dyslipidemia and reassess the immunosuppressive regimen because mTOR inhibitors can worsen lipid abnormalities.

C. Increase the mTOR inhibitor dose because these agents lower triglycerides in a dose-dependent manner.

D. Stop all lipid-lowering drugs because statins are absolutely contraindicated in every transplant recipient.

E. Perform TIPS because portal decompression is the definitive treatment for mTOR-associated hyperlipidemia.

188. A liver transplant recipient develops tremor, headache, confusion, hypertension, and seizures shortly after a rise in tacrolimus concentration. MRI shows posterior vasogenic edema. What is the most likely syndrome?

A. Recurrent hepatic encephalopathy because the transplanted liver normally produces severe hyperammonemia despite good graft function.

B. Chronic rejection because ductopenia directly causes posterior vasogenic cerebral edema.

C. Post-transplant lymphoproliferative disorder because PTLD always presents as symmetric posterior edema within days.

D. Biliary anastomotic stricture because cholestasis selectively causes occipital seizures.

E. Calcineurin-inhibitor neurotoxicity with posterior reversible encephalopathy syndrome.

189. A long-term liver transplant recipient has atraumatic vertebral compression fractures. Which factors commonly contribute to post-transplant bone disease?

A. Tacrolimus-induced excess bone formation is the dominant mechanism, so osteoporosis screening is unnecessary.

B. Preexisting cirrhosis-related bone loss, corticosteroid exposure, vitamin D deficiency, hypogonadism, and post-transplant metabolic factors.

C. Liver transplantation permanently eliminates fracture risk once bilirubin normalizes.

D. Only hereditary hemochromatosis causes osteoporosis in transplant recipients.

E. Weight-bearing activity is contraindicated after transplant because mechanical loading accelerates bone resorption.

190. A liver transplant recipient with preserved graft function is planning pregnancy. Which principle is most appropriate?

A. Pregnancy is absolutely prohibited after liver transplantation because graft rejection is inevitable.

B. Stop all immunosuppression at conception because every maintenance agent is teratogenic.

C. Coordinate conception with transplant and maternal-fetal specialists after graft stability, and change teratogenic drugs beforehand.

D. Continue mycophenolate without change because it is preferred during pregnancy.

E. Pregnancy requires prophylactic retransplantation because the graft cannot tolerate increased blood volume.

191. A recipient has anemia, leukopenia, and thrombocytopenia while receiving valganciclovir, mycophenolate, and trimethoprim-sulfamethoxazole. What is the best next step?

A. Diagnose chronic rejection because pancytopenia is a specific marker of graft ductopenia.

B. Increase all three medications because cytopenias indicate underexposure to immunosuppression.

C. Perform splenectomy immediately because post-transplant cytopenias are always caused by hypersplenism.

D. Ignore the blood counts because marrow suppression cannot occur with antiviral or antiproliferative therapy.

E. Evaluate for drug-related marrow suppression and infection, then adjust contributing medications carefully.

192. A long-term liver transplant recipient has increasing obesity, hypertension, diabetes, and chronic kidney disease. Which outcome is most directly threatened by this cluster?

A. Only early hepatic artery thrombosis, which is determined by metabolic syndrome years after surgery.

B. Immediate primary nonfunction because metabolic risk factors cause graft failure only on postoperative day 1.

C. Biliary anastomotic leak because hypertension and diabetes mechanically disrupt the bile duct years later.

D. ABO incompatibility because cardiometabolic disease changes the recipient's blood group over time.

E. Long-term cardiovascular and renal morbidity and mortality despite good liver-graft function.

193. A liver transplant recipient has severe chronic kidney disease and asks whether kidney transplantation may ever be appropriate. Which principle is most accurate?

A. Kidney transplantation is never permitted after liver transplantation because the liver graft precludes a second solid-organ transplant.

B. Evaluate persistent advanced kidney failure for kidney-after-liver transplantation when allocation criteria are met.

C. All post-liver kidney dysfunction resolves spontaneously, so renal transplant referral should never occur.

D. Kidney transplantation is indicated for any transient creatinine rise during the first postoperative day.

E. Only recipients with recurrent liver disease can receive a later kidney transplant regardless of renal function.

194. Six weeks after transplantation, a CMV-seronegative recipient of a CMV-seropositive donor develops fever, malaise, leukopenia, and detectable CMV DNA. What is the most likely diagnosis?

A. Acute cellular rejection because donor-recipient CMV serostatus has no relevance after transplant.

B. Primary nonfunction because CMV causes graft failure only in the first 24 hours.

C. Recurrent hepatitis A because HAV typically causes leukopenia and prolonged viremia after transplantation.

D. Calcineurin inhibitor toxicity because tacrolimus produces CMV DNA in blood without infection.

E. CMV syndrome in a high-risk donor-positive/recipient-negative transplant pairing.

195. A liver transplant recipient has CMV DNAemia plus odynophagia and endoscopy shows large esophageal ulcers. What is the best way to establish tissue-invasive CMV disease?

A. Obtain biopsy of the involved tissue for histopathologic evidence of CMV while treating the clinically significant infection.

B. Use the tacrolimus trough because elevated calcineurin exposure is diagnostic of CMV esophagitis.

C. Rely on a normal chest radiograph because tissue-invasive CMV is excluded when the lungs are clear.

D. Diagnose CMV solely from donor serostatus because tissue confirmation is never useful.

E. Perform ERCP because cholangiography is the definitive test for all forms of tissue-invasive CMV.

196. A recipient on high-dose immunosuppression presents with subacute dyspnea, fever, diffuse bilateral ground-glass opacities, and hypoxemia three months after transplant. Which opportunistic infection should be strongly considered if prophylaxis was interrupted?

A. Primary biliary cholangitis because autoimmune cholestasis produces diffuse pulmonary ground-glass disease.

B. Hepatic artery thrombosis because arterial occlusion primarily presents with subacute interstitial pneumonia.

C. Pneumocystis jirovecii pneumonia causing hypoxemic diffuse opportunistic lung infection.

D. Recurrent Wilson disease because copper accumulation causes opportunistic fungal pneumonia.

E. Gilbert syndrome because unconjugated hyperbilirubinemia produces hypoxemic alveolitis.

197. A liver transplant recipient develops dermatomal vesicular pain and rash without dissemination. Which general treatment principle is most appropriate?

A. Withhold antiviral therapy because varicella-zoster virus cannot reactivate after solid-organ transplantation.

B. Stop all immunosuppression permanently because any dermatomal zoster proves graft tolerance.

C. Use antibacterial therapy alone because vesicular eruptions in transplant recipients are never viral.

D. Treat herpes zoster promptly with an active antiviral and assess carefully for dissemination because immunosuppression increases complication risk.

E. Perform liver biopsy first because zoster is diagnosed by portal inflammation rather than skin findings.

198. Two months after transplantation, a recipient has fever, pleuritic chest pain, pulmonary nodules with surrounding ground-glass halo, and profound immunosuppression. Which infection is most concerning?

A. Uncomplicated CMV syndrome because CMV does not produce focal pulmonary nodules in heavily immunosuppressed patients.

B. Recurrent MASH because metabolic steatosis causes angioinvasive pulmonary lesions.

C. Anastomotic biliary stricture because focal bile-duct narrowing produces pulmonary fungal-like nodules.

D. Acute T-cell-mediated rejection because portal endotheliitis creates cavitating lung nodules.

E. Invasive pulmonary aspergillosis causing angioinvasive nodular lung disease under intense immunosuppression.

199. A transplant recipient with a biliary anastomotic stricture develops fever, jaundice, and hypotension. What is the most important management principle?

A. Treat acute cholangitis with antibiotics and urgent biliary decompression when obstruction is present.

B. Increase immunosuppression immediately because fever and jaundice after transplantation always indicate rejection.

C. Delay antibiotics until liver biopsy results are available because infection cannot coexist with a biliary stricture.

D. Use lactulose alone because biliary sepsis is caused by ammonia accumulation.

E. Observe without intervention because transplant recipients do not mount clinically important cholangitis.

200. A transplant recipient develops EBV DNAemia, fever, lymphadenopathy, weight loss, and a mass lesion. Which diagnosis is most concerning?

A. Acute T-cell-mediated rejection because lymphadenopathy and tumor masses are typical graft-limited findings.

B. Recurrent PSC because biliary strictures produce systemic EBV-driven lymphoid masses.

C. Primary nonfunction because PTLD occurs only during the first 24 hours after implantation.

D. Post-transplant lymphoproliferative disorder, often EBV-associated under intense immunosuppression.

E. Calcineurin nephrotoxicity because renal vasoconstriction causes clonal B-cell tumors.

201. An HCV-viremic patient undergoes liver transplantation without having received antiviral therapy. What is expected without post-transplant treatment?

A. HCV infection of the new graft is expected, so direct-acting antiviral therapy should be used rather than allowing progressive recurrent hepatitis.

B. The donor liver is permanently resistant to HCV because transplantation removes all recipient virus.

C. HCV cannot infect a transplanted liver while tacrolimus is present.

D. Recurrent HCV should be observed until decompensated graft cirrhosis develops because earlier therapy is ineffective.

E. Interferon is required because direct-acting antivirals do not work after solid-organ transplantation.

202. A recipient transplanted for HBV cirrhosis has undetectable HBV DNA on potent nucleos(t)ide therapy. Which long-term principle is most appropriate?

A. Stop all HBV therapy after transplantation because the new liver cannot be infected by circulating HBV.

B. Use hepatitis C antivirals instead because HBV recurrence is caused by HCV coinfection.

C. Permit high-level HBV viremia because immunosuppression prevents graft inflammation.

D. Maintain effective HBV suppression after transplantation, with prophylaxis individualized to recurrence risk and program strategy.

E. Treat only when graft cirrhosis recurs because prophylaxis does not alter post-transplant HBV outcomes.

203. A patient transplanted for primary biliary cholangitis develops gradual cholestatic liver tests years later. Biopsy and serologic evaluation suggest recurrence, while biliary imaging is normal. Which statement is most accurate?

A. PBC never recurs because transplantation removes the recipient's entire adaptive immune system.

B. Primary biliary cholangitis can recur in the allograft despite successful transplantation.

C. Any post-transplant cholestasis in a prior PBC patient is automatically chronic rejection without need for evaluation.

D. Recurrence occurs only in the first postoperative week and resolves spontaneously.

E. Recurrent PBC is caused by hepatic artery thrombosis and is diagnosed solely by Doppler ultrasound.

204. A patient transplanted for PSC develops multifocal biliary strictures several years later. Hepatic artery flow is normal. Which issue is especially important before diagnosing recurrent PSC?

A. Exclude alternative causes of nonanastomotic biliary injury, particularly ischemia, infection, and chronic rejection.

B. Assume recurrence immediately because all biliary strictures after PSC transplantation represent recurrent disease.

C. Exclude recurrent PSC if inflammatory bowel disease persists because PSC recurrence requires colectomy.

D. Diagnose acute cellular rejection because cholangiography cannot show recurrent biliary disease.

E. Use serum ammonia alone because recurrent PSC is defined by hyperammonemia rather than biliary findings.

205. A patient transplanted for autoimmune hepatitis develops rising aminotransferases, IgG elevation, and interface hepatitis years later. Which diagnosis should be considered?

A. Recurrent Wilson disease because interface hepatitis with high IgG is specific for copper overload.

B. Anastomotic biliary stricture because focal obstruction causes diffuse plasma-cell-rich interface hepatitis.

C. Recurrent autoimmune hepatitis after excluding rejection, viral infection, and drug injury.

D. Primary nonfunction because graft failure years after transplant is still classified as primary nonfunction.

E. Hepatic artery thrombosis because arterial occlusion is the only cause of post-transplant aminotransferase elevation.

206. A recipient transplanted for alcohol-associated cirrhosis resumes heavy alcohol use. Which statement is most accurate?

A. The transplanted liver is resistant to alcohol because donor hepatocytes lack alcohol-metabolizing enzymes.

B. Sustained harmful alcohol use can cause recurrent alcohol-associated liver injury and is associated with adverse graft and patient outcomes.

C. Any single drink causes immediate graft failure and requires automatic retransplantation.

D. Alcohol use affects only psychosocial outcomes and cannot cause histologic graft injury.

E. Immunosuppression completely prevents alcohol-associated steatohepatitis after transplantation.

207. A recipient transplanted for MASH develops obesity, diabetes, and steatosis in the graft. Which statement is most accurate?

A. MASH cannot recur because the metabolic syndrome is removed with the native liver.

B. Recurrent or de novo metabolic steatotic liver disease can occur after transplantation and may progress to steatohepatitis and fibrosis.

C. Any graft steatosis proves acute rejection and should be treated with steroid pulses.

D. Post-transplant weight gain protects against fibrosis by increasing hepatic energy stores.

E. Tacrolimus completely prevents hepatic steatosis by increasing insulin sensitivity.

208. A long-term liver transplant recipient asks about skin-cancer prevention. Which recommendation is most appropriate?

A. Use regular sun protection and periodic dermatologic screening because chronic immunosuppression increases nonmelanoma skin-cancer risk.

B. Avoid dermatologic evaluation because skin cancers are less common under immunosuppression.

C. Stop all immunosuppression whenever a benign nevus appears because any skin lesion predicts graft tolerance.

D. Use tanning beds to increase vitamin D because ultraviolet exposure reduces squamous-cell carcinoma risk.

E. Screen only if liver tests become abnormal because skin malignancy tracks directly with graft dysfunction.

209. Which vaccination principle is generally appropriate for a stable liver transplant recipient on maintenance immunosuppression?

A. Avoid all vaccines because immunosuppression makes every vaccine dangerous and ineffective.

B. Give live vaccines preferentially because replicating vaccines work best during intense immunosuppression.

C. Use indicated non-live vaccines and generally avoid live-attenuated vaccines during significant immunosuppression.

D. Vaccinate only against hepatitis A because other vaccine-preventable infections do not affect transplant recipients.

E. Stop immunosuppression for one month before every vaccine because otherwise no antibody response can occur.

210. A liver transplant recipient with ulcerative colitis and prior PSC asks about colorectal cancer surveillance. Which principle is most appropriate?

A. Stop colonoscopy after transplantation because removal of the native liver eliminates colorectal cancer risk.

B. Screen only with serum CEA because immunosuppression makes colonoscopy unsafe.

C. Perform colonoscopy only when liver tests rise because colorectal neoplasia causes graft cholestasis before intestinal symptoms.

D. Continue high-intensity colonoscopic surveillance because inflammatory bowel disease and prior PSC confer substantial colorectal neoplasia risk after transplantation.

E. Use stool occult blood testing alone at standard population intervals because PSC history no longer matters.

211. A transplant recipient is a current smoker. Which counseling is most appropriate?

A. Encourage continued smoking because nicotine reduces acute cellular rejection.

B. Strongly support smoking cessation to reduce cardiovascular and malignancy risks after transplantation.

C. Smoking is harmless after transplantation as long as liver enzymes remain normal.

D. Smoking affects only lung recipients and has no relevance after liver transplantation.

E. Recommend switching to cigars because noninhaled tobacco does not contribute to transplant-related cancer risk.

212. A 67-year-old liver transplant recipient has never had age-appropriate cancer screening since transplantation. What is the best approach?

A. Provide routine age- and sex-appropriate cancer screening plus transplant-specific surveillance when indicated.

B. Avoid all cancer screening because immunosuppression makes biopsy and treatment impossible.

C. Screen only for hepatocellular carcinoma because nonhepatic malignancies do not increase after transplant.

D. Wait for symptoms because screening has no role once a patient has received a solid-organ transplant.

E. Use PET scanning annually for every cancer because targeted screening recommendations no longer apply after transplantation.

213. A recipient transplanted for hepatocellular carcinoma asks about post-transplant surveillance. Which principle is most appropriate?

A. No surveillance is needed because transplantation guarantees that hepatocellular carcinoma cannot recur extrahepatically.

B. Use an individualized recurrence-surveillance plan based on the pretransplant tumor burden, pathology, and recurrence risk.

C. Perform daily alpha-fetoprotein testing because recurrence occurs only as an abrupt serum marker rise.

D. Use colonoscopy as the sole surveillance test because HCC recurrence occurs primarily in the colon.

E. Stop immunosuppression completely because that is the standard method of preventing all HCC recurrence.

214. A long-term recipient has stable graft function but has not seen a primary-care clinician in years. Which care model is best?

A. Use transplant clinic as a substitute for all preventive care because nonhepatic disease is unrelated to long-term outcomes.

B. Coordinate transplant-specialty follow-up with comprehensive primary and preventive care for cardiovascular, metabolic, bone, infectious, and cancer risks.

C. Follow only tacrolimus levels because normal levels exclude cardiovascular and malignant complications.

D. Discontinue primary care after one year because transplantation reverses age-related disease risk.

E. Limit follow-up to annual liver enzymes because graft chemistry is the only meaningful long-term health metric.

215. Which mechanism most directly initiates T-cell alloimmunity after liver transplantation?

A. Recipient erythrocytes recognize donor bile acids and then differentiate into cytotoxic lymphocytes.

B. Hepatocytes eliminate all donor HLA molecules at reperfusion, preventing adaptive immune recognition.

C. Complement activation alone permanently replaces the need for T-cell receptor recognition of alloantigen.

D. Portal hypertension converts recipient neutrophils into donor-specific plasma cells without antigen presentation.

E. Recipient T cells recognize donor alloantigens by direct and indirect antigen-presentation pathways.

216. Why is the liver sometimes described as relatively tolerogenic compared with several other transplanted organs?

A. The liver expresses no HLA molecules, so recipient lymphocytes cannot recognize the graft.

B. Portal venous blood contains no immune cells, making hepatic alloimmunity anatomically impossible.

C. Kupffer cells permanently destroy every recipient T cell during the first pass through the graft.

D. The liver's specialized immune environment can promote tolerance, but it does not eliminate alloimmune rejection.

E. Tolerance is guaranteed after one year, which is why long-term immunosuppression is unnecessary in all recipients.

217. Tacrolimus prevents rejection primarily through which mechanism?

A. It binds FKBP and inhibits calcineurin, reducing IL-2 transcription and T-cell activation.

B. It blocks inosine monophosphate dehydrogenase selectively in B cells and prevents guanosine synthesis.

C. It irreversibly alkylates donor DNA and directly deletes all graft antigen-presenting cells.

D. It neutralizes circulating TNF-alpha with a monoclonal antibody and has no intracellular target.

E. It activates mTOR to increase T-cell proliferation while suppressing antibody production.

218. A liver transplant recipient taking tacrolimus begins clarithromycin and several days later develops tremor and acute kidney injury. What is the most likely explanation?

A. Clarithromycin induced CYP3A and rapidly reduced tacrolimus to undetectable concentrations.

B. Tacrolimus caused immune hemolysis because macrolides convert it into an oxidizing metabolite.

C. The combination obligatorily causes hepatic artery thrombosis independent of tacrolimus concentration.

D. CYP3A inhibition increased tacrolimus exposure, causing calcineurin-inhibitor toxicity.

E. Clarithromycin blocks mycophenolate absorption, which specifically produces tacrolimus neurotoxicity.

219. A transplant recipient with a stable tacrolimus dose starts rifampin and subsequently has a very low trough concentration. What is the most likely mechanism?

A. Rifampin induces drug-metabolizing pathways and can markedly lower tacrolimus exposure.

B. Rifampin directly inhibits calcineurin and therefore makes tacrolimus levels appear falsely low while exposure increases.

C. Rifampin blocks renal tacrolimus excretion and invariably raises tacrolimus concentrations.

D. Rifampin converts tacrolimus into cyclosporine, so trough monitoring becomes invalid.

E. Rifampin has no clinically relevant interaction with calcineurin inhibitors.

220. Which adverse-effect profile is most characteristic of calcineurin inhibitors?

A. Profound irreversible bone-marrow aplasia in every recipient without renal or neurologic effects.

B. Severe pulmonary fibrosis as the dominant class toxicity with complete renal protection.

C. Nephrotoxicity, hypertension, neurotoxicity, hyperkalemia, and metabolic complications such as diabetes, particularly with tacrolimus.

D. Universal hemorrhagic cystitis and bladder cancer caused by acrolein metabolites.

E. Isolated cholestatic pruritus with no systemic or renal adverse effects.

221. Mycophenolate mofetil suppresses lymphocyte proliferation primarily by which mechanism?

A. Binding FKBP to inhibit calcineurin and block NFAT activation.

B. Inhibition of inosine monophosphate dehydrogenase, limiting de novo guanosine nucleotide synthesis in lymphocytes.

C. Covalent inhibition of cyclooxygenase to suppress prostaglandin-mediated rejection.

D. Direct antagonism of the IL-2 receptor without any effect on nucleotide synthesis.

E. Activation of mTOR to arrest hepatocytes in the G2 phase of the cell cycle.

222. A woman taking mycophenolate after liver transplantation wants to become pregnant. What is the most appropriate management principle?

A. Continue mycophenolate unchanged because it has no fetal effects and prevents preeclampsia.

B. Stop every immunosuppressive drug immediately and attempt conception the same day.

C. Double the mycophenolate dose during the first trimester because pregnancy accelerates graft rejection in all patients.

D. Add isotretinoin because it counteracts the teratogenic metabolites of mycophenolate.

E. Replace mycophenolate with a pregnancy-compatible immunosuppressive strategy before conception because mycophenolate is strongly teratogenic.

223. A liver transplant recipient taking azathioprine is prescribed allopurinol for gout. What is the major concern?

A. Allopurinol accelerates azathioprine clearance and predictably causes acute rejection from underexposure.

B. Allopurinol inhibits thiopurine metabolism and can cause severe azathioprine myelotoxicity.

C. The combination causes isolated tacrolimus nephrotoxicity even when tacrolimus is not being used.

D. Azathioprine inactivates allopurinol, making gout the only clinical concern.

E. There is no clinically important interaction between thiopurines and xanthine oxidase inhibition.

224. A recipient receiving everolimus develops marked hyperlipidemia, oral ulcers, edema, and proteinuria. Which statement is most accurate?

A. They prove acute cellular rejection because mTOR inhibitors do not have metabolic or renal adverse effects.

B. The findings are expected only with corticosteroids and exclude everolimus toxicity.

C. Everolimus should be increased because proteinuria indicates inadequate mTOR blockade.

D. The findings mandate stopping all immunosuppression permanently because no alternative regimen is possible.

E. These are recognized adverse effects of mTOR inhibition and may require dose adjustment, supportive treatment, or a change in immunosuppressive strategy.

225. Why are mTOR inhibitors often avoided immediately after major transplant surgery in patients with significant wound concerns?

A. They cause obligatory hyperacute rejection whenever given within the first month.

B. They dissolve surgical sutures by direct chemical action in the bloodstream.

C. They completely suppress hepatic regeneration, making all post-transplant use permanently contraindicated.

D. They increase calcineurin activity and thereby produce immediate T-cell activation at the incision.

E. They can impair wound healing, so timing of initiation should account for surgical healing and other patient-specific risks.

226. A transplant team uses basiliximab as induction therapy in a recipient in whom early calcineurin-inhibitor exposure is being minimized. What is its principal target?

A. CD20 on mature B lymphocytes, causing broad B-cell depletion.

B. Calcineurin within renal tubular cells, producing direct nephroprotection.

C. The alpha chain of the IL-2 receptor on activated T lymphocytes.

D. The glucocorticoid receptor on hepatocytes, producing high-dose steroid effects.

E. C5 complement, completely preventing all antibody-mediated graft injury.

227. Antithymocyte globulin is most accurately described as which type of immunosuppressive therapy?

A. A nonabsorbable antibiotic that prevents bacterial translocation without altering lymphocytes.

B. A selective mTOR activator used mainly to treat dyslipidemia after transplantation.

C. A polyclonal T-cell-depleting antibody preparation used for potent induction or steroid-resistant rejection.

D. A hepatitis B neutralizing antibody with no effect on human T cells.

E. A calcineurin inhibitor metabolized identically to tacrolimus and monitored with the same trough assay.

228. A stable recipient has worsening chronic kidney disease on tacrolimus. Which immunosuppressive principle is most appropriate?

A. Use the lowest effective calcineurin-inhibitor exposure and add renal-sparing agents when appropriate.

B. Increase tacrolimus until creatinine doubles because nephrotoxicity predicts stronger graft protection.

C. Stop immunosuppression abruptly because kidney disease proves that the liver graft has become tolerant.

D. Replace tacrolimus with high-dose corticosteroid monotherapy indefinitely in every patient.

E. Ignore renal decline because immunosuppression should never be modified once the first discharge regimen is chosen.

229. A competent transplant candidate declines a donor liver after hearing the organ-specific risks, even though the physician believes acceptance is medically advisable. Which ethical principle is most directly involved?

A. Beneficence requires the physician to override every informed refusal when survival may be improved.

B. Justice permits the physician to conceal donor risk so that scarce organs are not declined.

C. Nonmaleficence requires removal from the wait list whenever a patient asks questions about an organ offer.

D. Confidentiality allows the transplant team to obtain consent from the family instead of the competent patient.

E. Respect for patient autonomy after adequate disclosure and assessment of decision-making capacity.

230. A transplant candidate's adult child asks the team for the exact medical reason another living donor was declined. The potential donor has not authorized disclosure. What is the best response?

A. Provide the complete donor medical record because the recipient's need automatically overrides donor privacy.

B. Tell the recipient that the donor was psychologically unsuitable even if that was not the reason, because confidentiality permits fabrication.

C. Release the donor's test results to all family members because evaluation for altruistic donation eliminates privacy rights.

D. Post the donor's reasons in the medical chart visible to the recipient so the family can reconsider donation together.

E. Protect the potential donor's medical confidentiality and disclose only information the donor has authorized.

231. A wealthy candidate offers to pay another person directly for a liver lobe outside an authorized transplant program. Which principle is most appropriate?

A. Organ purchase is prohibited; living donation must remain voluntary and protected from commercial exploitation.

B. Direct payment is preferred because market pricing is the fairest way to allocate living donor risk.

C. Commercial organ purchase is acceptable whenever the recipient's MELD score is high enough.

D. Payments are required for valid informed consent because altruistic donation is considered coercive.

E. The transaction is permissible if both parties sign a private contract without transplant-program review.

232. Two transplant candidates have similar medical urgency, but one has greater financial resources and offers a large donation to the hospital. How should organ-allocation priority be determined?

A. Give priority to the wealthier candidate because ability to pay is a validated predictor of graft survival.

B. Give priority to the candidate with the most prestigious occupation because societal contribution should determine organ access.

C. Let the transplant surgeon privately choose whichever candidate has the strongest personal relationship with the center.

D. By established allocation policy and medically relevant criteria rather than wealth, social worth, or financial contribution.

E. Auction the organ between candidates because market competition is the standard method of distributive justice.

233. Why does liver allocation use objective severity models such as MELD-based systems for many adult candidates?

A. They predict long-term alcohol relapse with perfect accuracy and therefore replace psychosocial evaluation.

B. They provide a standardized estimate of short-term mortality risk that supports prioritization by medical urgency rather than local subjective judgment alone.

C. They measure donor graft quality and are calculated from donor rather than recipient variables.

D. They guarantee identical waiting times across every blood group and geographic area.

E. They eliminate the need for exceptions because every clinically important liver disease is fully represented by laboratory values.

234. A candidate has a liver condition associated with substantial wait-list mortality that is poorly reflected by the standard laboratory severity score. What is the purpose of an exception process?

A. To allow each center to assign any desired priority without documentation or review.

B. To let candidates purchase additional allocation points when insurance coverage is available.

C. To bypass national allocation rules for every patient whose physician prefers earlier transplantation.

D. To provide policy-governed additional priority when validated disease-specific risk is not adequately represented by the calculated score.

E. To replace medical urgency with first-come, first-served waiting time for all candidates.

235. A patient with hepatocellular carcinoma has preserved synthetic function but a substantial risk of tumor progression while waiting. Why can tumor-specific allocation exceptions be ethically justified?

A. Because every liver tumor has identical biology and therefore deserves the highest possible transplant priority immediately.

B. Standardized tumor exceptions can account for progression risk that laboratory liver scores underestimate.

C. Because cancer candidates should always outrank patients with acute liver failure regardless of tumor stage.

D. Because exception points guarantee cure and remove the need to consider tumor burden or biology.

E. Because tumor exceptions are based solely on ability to pay for locoregional therapy rather than medical risk.

236. A transplant candidate repeatedly misses appointments because of unstable housing and transportation barriers. Which approach best reflects equitable transplant evaluation?

A. Exclude the patient automatically because unstable housing proves unwillingness to follow medical advice.

B. Identify and address modifiable structural barriers and assess the patient's ability to follow the treatment plan rather than equating poverty itself with nonadherence.

C. Ignore missed visits entirely because adherence has no relationship to post-transplant medication safety.

D. Require a large cash deposit because financial resources are the most ethical measure of transplant readiness.

E. Use employment status as the sole predictor of adherence because employed patients never miss medications.

237. A patient with alcohol-associated cirrhosis has a history of relapse but is now engaged in treatment, attends appointments, and has strong support. Which ethical approach to candidacy is most appropriate?

A. Exclude every patient with alcohol-associated liver disease because self-inflicted disease has lower moral worth.

B. Ignore alcohol use disorder completely because relapse cannot affect graft or patient outcomes.

C. List only patients who can prove that they have never consumed alcohol in their lifetime.

D. Determine candidacy solely by family income because economic stability is the best measure of addiction recovery.

E. Use individualized relapse-risk and treatment-engagement assessment rather than categorical exclusion.

238. A listed patient with metastatic extrahepatic malignancy develops rapid clinical decline and no longer has a reasonable expectation of transplant benefit. What is the most appropriate program response?

A. Keep the patient active indefinitely because wait-list status can never be changed after initial listing.

B. Proceed with transplantation solely because the patient has accumulated waiting time, regardless of new contraindications.

C. Reassess candidacy transparently and consider inactivation or delisting if transplantation is no longer expected to provide acceptable benefit under program and policy criteria.

D. Delete all documentation of the new cancer because candidacy decisions must use only information available at the original evaluation.

E. Transfer the patient to the highest urgency category because metastatic cancer increases overall mortality risk.

239. A candidate is offered an organ associated with a specific increased infectious transmission risk. Which consent standard is most appropriate?

A. Disclose material donor risk, expected benefit, alternatives, and consequences of accepting or declining.

B. Withhold donor-related risk because discussing transmission might reduce organ utilization.

C. Obtain consent only after transplantation because preoperative discussion delays surgery.

D. Tell the patient that every donor organ has identical risk and therefore no organ-specific information matters.

E. Ask a hospital administrator to consent on behalf of every competent recipient because organ offers are allocation decisions rather than medical choices.

240. A transplant clinician has a serious concern that an allocation rule is producing inequitable effects. What is the most appropriate response?

A. Secretly alter candidate data to obtain higher priority because perceived unfairness justifies falsifying allocation information.

B. Follow current rules while using established policy-review channels to advocate transparent evidence-based change.

C. Ignore the concern because transplant-allocation policies cannot be revised once implemented.

D. Create a private center-specific priority system that overrides national allocation requirements.

E. Offer organs outside the regulated network to whichever patients the clinician personally believes deserve them most.

Answers

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1. B. Biliary atresia can progress despite an initially successful Kasai procedure. Recurrent cholangitis, persistent cholestasis, portal hypertension, and poor growth are major signals that native-liver survival is failing and should prompt transplant evaluation before critical deterioration. Repeat Kasai or TIPS is not standard rescue therapy for this pattern, and corticosteroids do not reverse the underlying obliterative cholangiopathy.

2. D. Children with biliary atresia may develop severe portal-hypertensive complications while bilirubin and INR remain relatively preserved. Recurrent or difficult-to-control variceal bleeding is an important indication to consider transplantation because clinical decompensation, not only synthetic failure, determines native-liver adequacy. Stable thrombocytopenia or an incidental small varix alone is less compelling.

3. A. Cholestatic infants have high caloric needs and impaired fat and fat-soluble vitamin absorption. Energy-dense feeds, medium-chain triglycerides, aggressive vitamin replacement, and tube feeding when necessary are standard strategies to support growth. Protein restriction and calorie limitation worsen sarcopenia and growth failure.

4. A. The combination of acute liver failure, Coombs-negative hemolysis, relatively low alkaline phosphatase, and neuropsychiatric findings strongly suggests Wilsonian acute liver failure. This presentation can deteriorate rapidly and requires urgent transplant evaluation; chelation is not a reliable substitute for transplantation in fulminant Wilson disease. Biopsy should not delay definitive management and may be hazardous with coagulopathy.

5. E. The transplanted liver restores functional ATP7B-mediated copper handling and is definitive therapy for hepatic Wilson disease. Advanced chronic liver disease is a standard indication, and selected patients with severe refractory neurologic disease may also be considered after careful multidisciplinary review. Neurologic outcomes are variable, so transplantation is not automatic for isolated neurologic symptoms.

6. C. Alpha-1 antitrypsin is produced primarily by hepatocytes, so donor hepatocytes produce normal protein and the hepatic disease does not recur from the recipient genotype. However, established lung disease may persist and can materially affect transplant candidacy and perioperative risk. Intravenous augmentation therapy is directed at lung disease and does not reverse advanced cirrhosis.

7. E. In hereditary hemochromatosis, extrahepatic disease—particularly cardiomyopathy—may determine transplant risk more than hepatic iron concentration. Careful cardiac assessment is essential because severe fixed cardiac dysfunction can make isolated liver transplantation unsafe. Diabetes may persist after transplant, and complete iron depletion is not a prerequisite when decompensated cirrhosis requires timely evaluation.

8. C. Some inherited metabolic diseases are transplant indications because the liver contains the missing enzyme, not because the liver is structurally cirrhotic. Liver transplantation can prevent recurrent hyperammonemic crises in urea-cycle disorders by restoring functional ureagenesis. Waiting for synthetic failure or cirrhosis would expose the patient to preventable neurologic injury.

9. C. Cystic fibrosis can produce clinically important liver disease together with severe pulmonary disease. When lung function is profoundly impaired, isolated liver transplantation may be unsafe and combined organ transplantation may need consideration at an experienced center. The decision depends on the severity of both organ systems rather than liver synthetic function alone.

10. D. Localized Caroli disease may sometimes be treated by hepatic resection, but diffuse bilobar disease complicated by recurrent cholangitis and secondary cirrhosis is a classic indication for transplantation. Endoscopic therapy may manage episodes or stones but does not eliminate widespread congenital duct ectasia. The abnormality is intrahepatic rather than a distal obstructing lesion.

11. C. Most circulating transthyretin is synthesized in hepatocytes. In selected hereditary TTR amyloidosis phenotypes, liver replacement sharply reduces production of variant TTR and can slow disease progression, although existing neurologic or cardiac amyloid may not fully regress and wild-type deposition can continue. The germline mutation in other tissues is not changed.

12. D. Crigler-Najjar type I results from absent or nearly absent hepatic UGT1A1 activity. A normal donor liver restores bilirubin conjugation and can be curative even though the native liver is not cirrhotic. This is another example of transplantation for a hepatic metabolic defect rather than structural end-stage liver disease.

13. C. In erythropoietic protoporphyria, excess protoporphyrin is produced largely in erythroid cells and excreted through the liver. Transplantation can rescue liver failure but does not eliminate the upstream hematopoietic defect, so recurrent graft injury can occur. Management therefore requires coordinated metabolic and hematologic planning rather than assuming the transplant is etiologically curative.

14. B. PBC can be diagnosed when compatible cholestatic liver tests coexist with PBC-specific autoantibodies, most commonly antimitochondrial antibody. In a typical presentation with no competing diagnosis, biopsy is not mandatory. Ursodeoxycholic acid is first-line disease-modifying therapy and should not be delayed until bilirubin rises.

15. A. Persistent alkaline phosphatase elevation and rising bilirubin after an adequate trial of ursodeoxycholic acid indicate higher-risk PBC and inadequate biochemical response. Such patients need reassessment for additional disease-modifying options, complications, and timely transplant referral when appropriate. Stopping ursodeoxycholic acid simply because the response is incomplete is not the usual strategy.

16. B. The serologic pattern, elevated IgG, and compatible biopsy establish autoimmune hepatitis after exclusion of competing causes. Corticosteroid induction with a steroid-sparing maintenance strategy is standard in patients with active disease. Untreated AIH can progress to cirrhosis or acute liver failure, so observation is inappropriate when significant inflammatory activity is present.

17. D. Budesonide depends on extensive hepatic first-pass metabolism to limit systemic glucocorticoid exposure. In cirrhosis and significant portosystemic shunting, that pharmacologic advantage is lost, and systemic exposure may increase. Prednisone or prednisolone-based regimens are therefore favored when cirrhosis is present.

18. A. Relapse is common after premature withdrawal of immunosuppression in autoimmune hepatitis. Withdrawal is generally reserved for carefully selected patients with a prolonged period of complete biochemical remission, followed by close monitoring. Eight months of normal tests is too short to assume durable drug-free remission.

19. E. A beaded pattern of multifocal stricturing involving intrahepatic and extrahepatic bile ducts in a patient with inflammatory bowel disease is characteristic of PSC. PBC is a small-duct autoimmune cholangiopathy and does not produce this classic cholangiographic pattern. IgG4-related disease can mimic PSC but usually has additional serologic, pancreatic, or morphologic clues.

20. B. A new clinically relevant stricture, progressive cholestasis, and elevated tumor marker in PSC require prompt evaluation for cholangiocarcinoma. Cross-sectional imaging and endoscopic assessment

with appropriate sampling are central to the workup. Assuming benign PSC progression can delay diagnosis of a malignancy that profoundly affects treatment and transplant strategy.

21. C. PSC substantially increases colorectal neoplasia risk in patients with inflammatory bowel disease. Surveillance therefore begins earlier and is performed more frequently than in average-risk colitis. The risk does not disappear after liver transplantation, so ongoing colonoscopic surveillance remains important.

22. B. MELD may not capture important PSC morbidity such as recurrent cholangitis, severe pruritus, or selected cancer-related indications. Transplant centers can evaluate such patients even when the calculated score is modest. Referral permits assessment of complications, exception pathways where applicable, and timing rather than waiting for profound synthetic failure.

23. A. IgG4-related cholangitis is commonly associated with autoimmune pancreatitis, can produce substantial serum IgG4 elevation, and often responds to glucocorticoid therapy. PSC more commonly coexists with inflammatory bowel disease and has a different cholangiographic and therapeutic profile. The distinction is crucial because IgG4-related disease is treatable with immunosuppression.

24. B. A minority of patients with hepatic sarcoidosis progress to severe portal hypertension or liver failure and can be considered for transplantation. Granulomatous recurrence in the graft can occur, but it does not universally lead to graft failure. Extrahepatic disease must be assessed because transplantation does not cure systemic sarcoidosis.

25. A. Mild liver-test abnormalities related to untreated celiac disease often improve with a gluten-free diet. Persistent hepatocellular injury, elevated IgG, and disease-specific autoantibodies should trigger evaluation for coexisting autoimmune hepatitis. Celiac disease and autoimmune liver diseases can coexist, so continued abnormalities should not simply be attributed to dietary exposure.

26. A. The patient has strong features of both PBC and AIH: cholestasis, antimitochondrial antibodies, florid duct injury, marked aminotransferase elevation, elevated IgG, smooth-muscle antibodies, and interface hepatitis. Recognizing overlap matters because treatment often needs to address both cholangiopathy and hepatitic inflammation rather than treating either component alone.

27. B. Overlap syndromes contain clinically meaningful features of more than one autoimmune liver disease. When PBC and AIH features coexist, therapy is generally individualized to treat both cholestatic and hepatitic components, commonly using ursodeoxycholic acid plus immunosuppression when interface hepatitis is important. Ignoring one component risks undertreatment.

28. D. Autoimmune hepatitis may recur after liver transplantation and can overlap diagnostically with rejection or other causes of graft dysfunction. Maintenance immunosuppression therefore requires thoughtful long-term management, especially in patients transplanted for autoimmune disease. Transplantation replaces the diseased liver but does not eliminate the recipient's immunologic predisposition.

29. D. Maintaining control of autoimmune hepatitis is important during pregnancy, and flares can occur particularly postpartum. Azathioprine and corticosteroids may be continued when required for disease control, whereas mycophenolate is teratogenic and should not be used in pregnancy. Close biochemical monitoring is appropriate rather than routine withdrawal of therapy.

30. E. PBC management includes more than biochemical disease control. Pruritus and metabolic bone disease are common and should be actively assessed and treated, with bone-density surveillance and appropriate osteoporosis therapy when indicated. Symptom burden can be severe even when synthetic function is preserved.

31. D. A symptomatic high-grade stricture with cholangitis requires antibiotics and relief of clinically relevant obstruction, generally through expert endoscopic management. Suspicious strictures often require sampling for malignancy because PSC confers cholangiocarcinoma risk. A normal bilirubin does not exclude a dangerous focal biliary problem.

32. C. Cholestatic liver diseases can produce portal-hypertensive complications before dramatic changes in conventional synthetic markers. Transplant assessment and complication management therefore depend on the whole clinical picture, including varices, ascites, splenomegaly, and quality of life—not bilirubin alone.

33. B. Complete biochemical remission in autoimmune hepatitis includes normalization of aminotransferases and IgG. Persistent IgG elevation may signal ongoing immune activity, imperfect adherence, under-treatment, or an alternative diagnosis. It should not be dismissed simply because AST and ALT have normalized.

34. C. Patients with cirrhosis and detectable HBV DNA require potent suppressive antiviral therapy, and decompensated cirrhosis is a particularly strong indication. Entecavir or tenofovir-based therapy is preferred because of potency and resistance profile. Interferon is unsafe in decompensated disease, and HBV treatment is generally suppressive rather than a short curative course.

35. C. Modern direct-acting antivirals can cure HCV before or after transplantation, but timing should be individualized. In decompensated patients, cure may improve function enough to delay transplant, yet some patients remain clinically decompensated despite a lower MELD and may lose access to HCV-viremic donors. The transplant team should integrate disease severity, expected wait time, and donor strategy.

36. A. Anti-HDV establishes exposure but does not prove ongoing viral replication. Detectable HDV RNA confirms active hepatitis D and is required to assess current infection. HDV can drive progressive disease even when HBV DNA is suppressed because it depends on HBsAg rather than high-level HBV replication.

37. B. Candidates with chronic liver disease should receive indicated inactivated vaccines before transplantation whenever possible, including hepatitis A and B vaccination when nonimmune. Vaccine

responses often diminish after transplantation because of immunosuppression. Live vaccines generally require special timing and are not routinely administered after solid-organ transplantation.

38. E. Most HEV infections in immunocompetent hosts are self-limited, but chronic infection is well recognized in immunosuppressed patients, including transplant recipients. Persistent HEV RNA can lead to chronic hepatitis and fibrosis. Diagnosis and management therefore depend on recognizing this different natural history rather than treating HEV as uniformly transient.

39. C. HSV hepatitis is uncommon but highly lethal and can present without characteristic skin or mucosal lesions. In unexplained acute liver failure, especially with compatible clinical features, empiric intravenous acyclovir is reasonable while PCR testing is pending because delay in therapy can be catastrophic. The absence of vesicles does not reliably exclude the diagnosis.

40. A. Active HBV replication at transplantation historically conferred a high risk of graft reinfection. Potent nucleos(t)ide analogues and appropriately selected prophylactic strategies have dramatically reduced recurrence. The relevant virologic factor is HBV replication, not unrelated biochemical or autoimmune markers.

41. C. Budd-Chiari syndrome results from obstruction of hepatic venous outflow and classically causes abdominal pain, hepatomegaly, ascites, and characteristic Doppler or cross-sectional imaging findings. Portal vein thrombosis affects inflow rather than hepatic venous drainage. Recognition is important because management may include anticoagulation, treatment of the prothrombotic cause, endovascular therapy, TIPS, or transplantation.

42. A. Management of Budd-Chiari is stepwise: anticoagulation and treatment of the prothrombotic disorder, recanalization when possible, and portal decompression with TIPS for persistent congestion or complications. Liver transplantation is reserved for fulminant disease or failure of less invasive strategies. Anticoagulation often remains necessary because the thrombophilic tendency persists.

43. C. Sinusoidal obstruction syndrome typically follows conditioning regimens or other endothelial toxins and presents with weight gain, painful hepatomegaly, ascites, and jaundice. Major hepatic veins can remain patent because the obstruction is at the sinusoidal and terminal venular level. This distinguishes it from classic Budd-Chiari syndrome.

44. A. The Fontan circulation exposes the liver to chronically elevated venous pressure and often reduced pulsatile cardiac output, producing sinusoidal congestion, fibrosis, portal-hypertensive features, and eventually advanced liver disease. Conventional liver tests may underestimate severity. Assessment therefore needs to integrate cardiac physiology, imaging, portal-hypertensive findings, and malignancy surveillance.

45. A. In Fontan patients, liver and heart disease arise from shared abnormal hemodynamics and can both become irreversible. When advanced hepatic disease coexists with severe cardiac failure, combined

heart-liver transplantation may offer the best strategy. Isolated organ decisions require multidisciplinary assessment because correcting one organ does not automatically reverse established disease in the other.

46. D. Congestive hepatopathy is driven by elevated right-sided pressures and impaired hepatic venous drainage. Treatment focuses on improving the cardiac lesion and volume status. TIPS can worsen right-sided failure by increasing venous return and is not a routine treatment for congestive hepatopathy caused by severe cardiac disease.

47. B. Sickle cell disease can produce several hepatic syndromes, including severe intrahepatic sickling with extreme cholestasis and acute liver failure. When synthetic failure and encephalopathy develop, the condition is life-threatening and may require exchange transfusion, intensive supportive care, and early transplant-center involvement. Simple hemolysis or gallstones do not explain the full syndrome.

48. B. The success of isolated liver transplantation in congestive hepatopathy depends heavily on the heart's ability to tolerate major shifts in preload and afterload. Severe fixed right-sided failure can make isolated liver transplantation prohibitive and may instead prompt consideration of cardiac intervention or combined organ transplantation. Minor manifestations of congestion do not carry the same implication.

49. E. Portosinusoidal vascular disease can cause clinically significant portal hypertension despite the absence of cirrhosis and may show nodular regenerative hyperplasia or obliterative portal venopathy. Preserved synthetic function and discordantly low liver stiffness can be clues. Patients still require management of portal-hypertensive complications and evaluation for associated conditions.

50. D. The complications of portal hypertension—especially varices—are clinically important whether portal pressure arises from cirrhosis or a noncirrhotic vascular disorder. Standard prophylactic approaches are therefore used when indicated. Transplantation is not automatically required when liver function is preserved and portal-hypertensive complications can be managed.

51. D. Management of portal vein thrombosis in cirrhosis depends on acuity, extent, symptoms, progression, bleeding risk, and transplant candidacy. In a transplant candidate, preserving portal vein patency can be particularly valuable because progressive thrombosis may complicate or jeopardize transplant surgery. Thrombocytopenia alone neither proves progression nor absolutely prohibits anticoagulation.

52. A. In selected transplant candidates with chronic portal vein thrombosis, portal vein recanalization combined with TIPS can restore physiologic portal flow and facilitate standard portal anastomosis at transplantation. This is technically specialized and not appropriate for every patient. Chronic cavernous transformation is generally not reversed by simple systemic thrombolysis.

53. D. Extension of acute splanchnic thrombosis into mesenteric veins can compromise bowel drainage and produce intestinal ischemia or infarction. Severe pain, rising lactate, and abnormal bowel enhancement are alarm features. This requires urgent assessment rather than routine outpatient anticoagulation alone.

54. C. Large congenital portosystemic shunts can bypass hepatic detoxification and cause encephalopathy, pulmonary vascular complications, or hepatic nodules despite preserved synthetic function. When anatomy and portal venous capacity permit, shunt closure may be definitive. Transplantation is reserved for selected cases in which closure is not feasible or serious liver-related complications coexist.

55. B. Physiologic portal inflow is important for graft function. Progressive or extensive portal vein thrombosis can remove a straightforward anastomotic target and force complex surgical or interventional reconstruction. This is why preventing progression or restoring portal patency can matter greatly in transplant candidates.

56. A. Patients with varices at meaningful risk of bleeding should receive primary prophylaxis. Nonselective beta-blockers reduce portal inflow and, depending on the agent, intrahepatic resistance; endoscopic ligation is an alternative when appropriate. Routine prophylactic TIPS is not indicated for uncomplicated primary prevention.

57. B. Suspected acute variceal hemorrhage is treated immediately with vasoactive therapy, short-course antibiotic prophylaxis, careful resuscitation, and urgent endoscopic hemostasis. Overtransfusion can increase portal pressure, and correction of INR is not the central strategy. Treatment should not wait for definitive endoscopic proof when the clinical suspicion is high.

58. C. After a first variceal bleed, the recurrence risk is high. Standard secondary prophylaxis combines nonselective beta-blockade with repeated endoscopic ligation unless contraindicated. TIPS is used for selected patients, including failures of standard therapy or certain high-risk presentations, rather than automatically for every survivor.

59. A. Spontaneous bacterial peritonitis can be clinically subtle. Diagnostic paracentesis is recommended in hospitalized patients with cirrhosis and new, worsening, or otherwise concerning ascites. Routine correction of INR before low-risk paracentesis is generally unnecessary because INR does not accurately quantify bleeding risk in cirrhosis.

60. A. Large-volume paracentesis can worsen effective arterial underfilling and precipitate circulatory and renal dysfunction. Intravenous albumin is used after removal of large volumes to reduce this risk. Plasma replacement, routine hypertonic saline, and protein restriction do not address the underlying circulatory physiology.

61. C. Refractory ascites marks advanced portal hypertension and poor prognosis. TIPS can improve ascites control in carefully selected patients with acceptable cardiac function, bilirubin, and encephalopathy risk, while liver transplantation addresses the underlying disease. Simply escalating diuretics despite failure increases renal and electrolyte toxicity.

62. B. An ascitic polymorphonuclear leukocyte count of at least 250/mm3 is diagnostic of spontaneous bacterial peritonitis in the appropriate clinical setting and warrants prompt treatment, even if cultures are

negative. Delaying therapy for culture confirmation can be dangerous. Albumin is added in patients at higher risk of renal dysfunction or death.

63. E. A prior episode of spontaneous bacterial peritonitis confers a substantial risk of recurrence. Secondary antibacterial prophylaxis is therefore standard while the patient is assessed for definitive management of advanced liver disease, including transplantation when appropriate. Prophylaxis does not eliminate the need to reassess new symptoms with paracentesis.

64. A. Overt hepatic encephalopathy in cirrhosis is treated by correcting precipitants and reducing intestinal nitrogen load, most commonly with lactulose; rifaximin reduces recurrence when added in appropriate patients. Severe protein restriction worsens sarcopenia and is not recommended. TIPS can worsen encephalopathy by increasing portosystemic shunting.

65. B. Large spontaneous portosystemic shunts can drive refractory encephalopathy by bypassing hepatic detoxification. In carefully selected patients with adequate hepatic reserve, embolization can reduce recurrent episodes, but it may increase portal pressure and worsen varices or ascites. TIPS would generally increase, not reduce, portosystemic diversion.

66. A. Hepatic hydrothorax reflects movement of ascitic fluid through diaphragmatic defects and often signals advanced portal hypertension. Refractory cases warrant transplant evaluation and may improve with TIPS in appropriately selected patients. Chronic chest tubes carry major risks, including infection, renal dysfunction, protein loss, and electrolyte disturbance.

67. D. Hepatopulmonary syndrome consists of liver disease or portal hypertension, abnormal oxygenation, and intrapulmonary vascular dilatations. Delayed left-sided bubble appearance on contrast echocardiography supports intrapulmonary shunting. Liver transplantation is the definitive therapy in appropriately selected patients.

68. D. Portopulmonary hypertension is pulmonary arterial hypertension associated with portal hypertension and is confirmed hemodynamically. Severe elevations in pulmonary vascular resistance and pressure substantially increase perioperative right-heart failure risk. Treatment aims to improve hemodynamics enough to permit safe transplantation when possible, with repeat invasive assessment guiding decisions.

69. B. HRS-AKI is a functional renal failure syndrome in advanced cirrhosis characterized by intense renal vasoconstriction in the setting of systemic and splanchnic circulatory dysfunction. Diagnosis requires exclusion and treatment of other common causes such as hypovolemia, infection, nephrotoxins, obstruction, and structural renal disease. A bland sediment and lack of improvement after appropriate volume expansion support the diagnosis.

70. A. HRS-AKI is treated with splanchnic vasoconstrictor therapy plus albumin, with terlipressin an established option where available. Response is more likely when treatment begins before extreme renal

failure. Respiratory status must be monitored because terlipressin can worsen respiratory failure in susceptible patients, and transplantation remains the definitive treatment for appropriate candidates.

71. C. Terlipressin can precipitate or worsen serious respiratory failure, particularly in patients with hypoxia or volume overload. Significant oxygenation deterioration should prompt reassessment and discontinuation or avoidance of the drug, along with careful management of albumin and volume status. HRS therapy must balance renal benefit against cardiopulmonary safety.

72. B. Cirrhotic cardiomyopathy may be occult at rest and emerge during physiologic or pharmacologic stress, when the heart fails to augment output appropriately. Diastolic dysfunction and electrophysiologic abnormalities may also occur. This matters during transplantation, a period of major hemodynamic stress.

73. D. Hyponatremia in advanced cirrhosis usually reflects impaired free-water excretion driven by nonosmotic vasopressin release. Management focuses on reversible factors, medication review, volume status, and selective fluid restriction, while avoiding overly rapid correction. Severe hyponatremia also carries prognostic and transplant implications.

74. C. Cirrhosis creates a rebalanced but fragile hemostatic system in which both procoagulant and anticoagulant pathways are altered. Conventional INR measures only part of this system and does not reliably predict bleeding risk. Low-risk procedures such as paracentesis generally do not require routine correction of an abnormal INR in the absence of other specific concerns.

75. A. In a patient at risk for HCC, characteristic multiphasic imaging can establish the diagnosis noninvasively without biopsy. Arterial phase hyperenhancement with later washout and other major features supports HCC when size and technical criteria are satisfied. Biopsy is reserved for lesions with indeterminate or atypical imaging or when pathology would change management.

76. B. For early HCC in cirrhosis, transplant candidacy depends on tumor burden, liver function, portal hypertension, comorbidity, and organ availability. Significant portal hypertension makes resection less attractive because of postoperative decompensation risk. Transplantation can cure the tumor while replacing the diseased liver and reducing the risk of new primary tumors in the native cirrhotic liver.

77. C. Selected patients beyond conventional transplant criteria can undergo structured downstaging with locoregional therapy. Successful reduction to accepted tumor burden, absence of vascular invasion or metastasis, and a period demonstrating favorable tumor biology can restore transplant eligibility. Downstaging is an oncologic process, not simply an automatic exception based on initial size.

78. E. AFP is a biomarker of HCC biology and recurrence risk. Very high AFP can limit standardized exception eligibility, while a substantial treatment-associated decline may support reconsideration when the residual tumor meets accepted criteria and there is no metastatic or macrovascular disease. AFP response does not by itself prove complete tumor necrosis.

79. C. Fibrolamellar HCC commonly occurs in younger patients without cirrhosis and can present as a large solitary mass. Complete surgical resection is the main curative approach when feasible. Transplantation may be considered in selected unresectable cases at specialized centers but is not automatically preferred over resection.

80. C. Perihilar cholangiocarcinoma was historically a contraindication to transplantation, but carefully selected patients treated with neoadjuvant protocols and rigorous staging can achieve meaningful long-term survival after transplant. This strategy is specialized and should not be generalized to all cholangiocarcinoma presentations.

81. D. Hepatic adenomas are associated with estrogen exposure and other metabolic or genetic factors. Larger lesions, symptomatic lesions, male sex, and certain molecular subtypes carry greater hemorrhagic or malignant potential. Withdrawal of hormonal drivers is important, but a lesion of this size commonly requires surgical or other definitive management rather than simple observation.

82. A. Hepatic adenomas in men have a higher malignant-transformation risk than in women, and management is therefore more aggressive even at smaller sizes. Resection is generally favored when feasible. Transplantation is reserved for highly selected circumstances rather than being the routine response to a solitary resectable adenoma.

83. A. Typical imaging can confidently diagnose hepatic hemangioma. Asymptomatic lesions generally require no intervention, and biopsy is avoided when imaging is classic because of bleeding risk and lack of benefit. Treatment is reserved for unusual complications, diagnostic uncertainty, or significant symptoms attributable to the lesion.

84. B. Cirrhosis confers persistent HCC risk, so surveillance continues even after a negative examination. A roughly six-month interval balances tumor growth kinetics with the ability to detect disease at a potentially curable stage. Alternative imaging may be needed when ultrasound quality is inadequate, but routine PET or serial biopsy is not standard surveillance.

85. D. Transplant oncology has expanded in carefully selected settings, including a small subset of patients with liver-dominant neuroendocrine metastases. Selection depends on tumor grade, stability, extent, primary-tumor control, age and comorbidity, and absence of uncontrolled extrahepatic disease. It remains a specialized indication rather than routine treatment of metastatic cancer.

86. A. Hepatic epithelioid hemangioendothelioma is biologically distinct from aggressive hepatic angiosarcoma. In selected patients with unresectable liver-dominant disease, transplantation can produce durable survival. Careful staging and tumor-specific selection are essential because not all vascular hepatic malignancies are suitable for transplant.

87. D. Not every nodule in cirrhosis is HCC. Indeterminate lesions require structured follow-up with appropriate cross-sectional imaging and, in selected cases, biopsy when histology would alter

management. Prematurely labeling an indeterminate lesion as HCC can expose the patient to unnecessary treatment and inappropriate transplant decisions.

88. C. Modern candidate evaluation emphasizes timely referral when decompensation develops rather than waiting for an arbitrary high MELD threshold. Ascites and encephalopathy mark a major change in prognosis and quality of life. Early evaluation allows time to address frailty, cardiopulmonary disease, psychosocial barriers, living-donor options, and other modifiable factors before the patient becomes critically ill.

89. C. Cardiovascular disease is a major concern in candidates with metabolic risk factors and can determine both perioperative and long-term outcomes. Evaluation should be individualized to symptoms and baseline risk rather than relying on resting ejection fraction alone. MASH candidates often need particularly careful coronary, structural, and functional assessment.

90. E. Outcomes in carefully selected people with controlled HIV can be acceptable after liver transplantation. Evaluation focuses on virologic suppression, immune status, opportunistic infection history, malignancy, adherence, and drug-drug interactions. HIV status alone should not be used as an automatic exclusion.

91. D. Active uncontrolled infection is a major contraindication to proceeding with transplantation because surgery and immunosuppression can be fatal in the setting of sepsis. The key distinction is uncontrolled infection, not a remote or adequately treated infectious history. Reassessment is appropriate once infection has been treated and clinical stability restored.

92. C. Frailty is associated with waitlist and post-transplant risk, but it can be modifiable and should be incorporated into a broader assessment rather than treated as a simplistic binary contraindication. Prehabilitation, adequate protein and calories, and management of reversible contributors can improve functional reserve. Serial assessment helps determine trajectory and treatment response.

93. D. Living donation exposes a healthy person to major surgery solely for another's benefit, so donor autonomy and safety are central. Evaluation should be independent, confidential, medically rigorous, and attentive to coercion, financial pressure, comprehension, and psychosocial readiness. Recipient benefit never justifies minimizing or misrepresenting donor risk.

94. C. The decision between liver-alone and simultaneous liver-kidney transplantation requires careful assessment of duration, severity, and reversibility of renal dysfunction and adherence to current allocation criteria. The goal is to avoid unnecessary use of a kidney when recovery is likely while protecting patients with established irreversible kidney disease. No single transient creatinine or eGFR value determines the decision.

95. B. Candidate evaluation includes assessment for active malignancy because transplantation must offer a reasonable expectation of survival and immunosuppression can accelerate some cancers. Active extrahepatic metastatic malignancy is generally incompatible with proceeding to liver transplantation.

Eligibility can be reconsidered in some cancer histories after successful treatment and an appropriate disease-free assessment.

96. E. Acute liver failure is characterized by rapid development of severe hepatic dysfunction, coagulopathy, and hepatic encephalopathy in someone without preexisting cirrhosis. The exact time window can vary by convention, but encephalopathy and impaired coagulation are central. Very high aminotransferases are neither required nor sufficient for the diagnosis.

97. B. N-acetylcysteine is the key antidotal therapy for acetaminophen toxicity and should be given even when presentation is delayed and hepatic injury is established. It replenishes glutathione and has additional hemodynamic and antioxidant benefits. Patients with encephalopathy and coagulopathy require intensive monitoring and early transplant-center involvement.

98. B. N-acetylcysteine is standard for acetaminophen toxicity and may also improve transplant-free survival in selected patients with early-stage non-acetaminophen acute liver failure. Supportive care does not replace etiologic evaluation or transplant assessment. Routine plasma correction can obscure INR trends and is reserved for bleeding or procedures rather than simply normalizing laboratory values.

99. C. Severe hyperammonemia is associated with cerebral edema in acute liver failure. Continuous renal replacement can support kidney function and remove ammonia while providing more stable hemodynamics than intermittent dialysis in critically ill patients. Neuroprotective ICU management also includes careful ventilation, sodium, temperature, glucose, and avoidance of factors that increase intracranial pressure.

100. E. High-grade encephalopathy in acute liver failure can progress to cerebral edema, intracranial hypertension, and herniation. These patients require specialized ICU care, frequent neurologic assessment, correction of physiologic triggers, and rapid transplant evaluation. Management should avoid maneuvers that lower serum osmolality or unnecessarily increase bleeding risk.

101. A. Ischemic hepatitis produces massive aminotransferase and LDH elevations after a period of reduced hepatic oxygen delivery, often with relatively modest bilirubin early in the course. Improvement depends on restoring hemodynamics and treating the underlying shock. The laboratory pattern differs from most chronic hepatitides and cholestatic diseases.

102. C. Autoimmune hepatitis can present dramatically, including with acute liver failure. Marked IgG elevation, compatible autoantibodies, and plasma-cell-rich interface hepatitis support the diagnosis after exclusion of other causes. Steroids may be considered in selected patients, but treatment must not delay transplant evaluation when prognostic features are poor or response is inadequate.

103. C. Amanita poisoning can have a deceptive interval after early gastrointestinal symptoms followed by severe hepatic necrosis. Progressive coagulopathy and encephalopathy require aggressive supportive care and early transplant-center involvement. Waiting for extreme bilirubin elevation or relying on portal-decompressive procedures can miss the narrow window for successful rescue transplantation.

104. B. Acute fatty liver of pregnancy can cause acute liver failure with hypoglycemia, coagulopathy, renal dysfunction, and encephalopathy. Prompt delivery is the key disease-specific intervention, accompanied by intensive supportive care. Most patients improve after delivery, but severe cases may require transplantation if hepatic failure does not reverse.

105. A. HSV hepatitis is a rare but devastating cause of acute liver failure, often without mucocutaneous lesions. When the syndrome is plausible, empiric intravenous acyclovir should be started while diagnostic testing proceeds because waiting for definitive confirmation can be fatal. Transplant evaluation should occur in parallel if hepatic failure is advanced.

106. C. Highest-urgency liver allocation categories are tightly defined by national policy and target patients at imminent risk of death, such as selected acute liver failure or very early graft-failure scenarios. They are not synonymous with a high MELD score in chronic liver disease. Transplant teams must document that the specific criteria are met.

107. B. Selected patients with severe alcohol-associated hepatitis may receive corticosteroid therapy after infection and other major contraindications are assessed. Early reassessment, commonly with the Lille score, identifies nonresponders in whom continued corticosteroids are unlikely to help. Abstinence treatment and nutritional support remain essential regardless of steroid use.

108. E. The Lille model uses early changes during corticosteroid therapy to estimate response in severe alcohol-associated hepatitis. A high score indicates poor response and supports stopping ineffective corticosteroid therapy rather than exposing the patient to continued infection risk. It is not a thrombosis, fibrosis, or relapse-prediction score.

109. D. Modern transplant evaluation for carefully selected patients with severe alcohol-associated hepatitis does not rely on a universal fixed abstinence interval. Programs assess medical urgency, prior treatment history, insight, social support, psychiatric comorbidity, and relapse risk in an integrated manner. Ongoing addiction treatment is part of transplant care rather than a one-time screening hurdle.

110. B. Complete abstinence can improve liver function, reduce decompensation risk, and remains central before and after transplantation. Treatment should include behavioral and pharmacologic approaches to alcohol use disorder when appropriate. Portal decompression or empiric anti-inflammatory therapy does not substitute for treating the underlying alcohol exposure.

111. C. Alcohol-associated hepatic injury often produces AST predominance, partly reflecting mitochondrial injury and relative pyridoxal phosphate deficiency. Extremely high aminotransferases should prompt evaluation for another or additional cause such as ischemia or toxin injury. Laboratory findings support but do not independently establish the diagnosis.

112. E. Protein-calorie malnutrition and sarcopenia worsen outcomes in cirrhosis. Adequate protein and energy intake, meal spacing, and a late-evening snack help reduce accelerated starvation; routine protein restriction is generally harmful. Enteral feeding is preferred when the gastrointestinal tract can be used.

113. E. Some patients with alcohol-associated decompensation improve substantially with sustained abstinence and optimized care. Transplant teams therefore reassess trajectory rather than assuming that the initial severity is fixed. Recovery can reduce urgency, whereas persistent or recurrent decompensation may still justify listing.

114. C. MASLD describes hepatic steatosis associated with cardiometabolic risk factors after appropriate clinical assessment. Fibrosis stage, rather than aminotransferase height alone, is the strongest liver-related prognostic determinant. Competing or coexisting causes of liver disease still require evaluation when clinically indicated.

115. C. Simple noninvasive scores such as FIB-4 are useful first-line tools for excluding advanced fibrosis in many patients. Low-risk patients generally undergo longitudinal reassessment while cardiometabolic disease is treated. Higher or indeterminate risk prompts second-line elastography or specialist evaluation rather than automatic biopsy.

116. B. Patients with indeterminate or elevated first-line fibrosis scores commonly need a second noninvasive test such as elastography. Sequential testing improves risk stratification and helps identify patients who merit hepatology referral or further evaluation. Ammonia, AFP, and cholangiography do not stage metabolic fibrosis.

117. E. Lifestyle treatment remains foundational in MASLD and MASH. Clinically meaningful, sustained weight loss can improve steatosis and inflammatory activity, with greater weight reduction generally needed for fibrosis benefit. Cardiovascular risk reduction is also crucial because cardiometabolic disease is a major source of morbidity and mortality.

118. C. MASLD frequently accompanies diabetes, obesity, dyslipidemia, and cardiovascular disease. Pretransplant evaluation therefore requires careful cardiopulmonary risk assessment and optimization, not merely characterization of liver disease. Cardiovascular complications remain important after transplantation as well.

119. C. Steatotic liver disease nomenclature recognizes that metabolic dysfunction and alcohol exposure can overlap. Alcohol can amplify liver injury in patients with cardiometabolic risk, and treatment should address both exposures rather than forcing a false single-cause classification. Accurate quantification of alcohol use is therefore clinically important.

120. A. Ascites, variceal hemorrhage, and overt hepatic encephalopathy are classic manifestations of hepatic decompensation. Their appearance changes prognosis and usually prompts transplant consideration when appropriate. Steatosis or isolated metabolic abnormalities do not by themselves define decompensated cirrhosis.

121. A. Obesity complicates transplant assessment, but BMI can be distorted by ascites and does not capture sarcopenia, frailty, or fat distribution. Evaluation should integrate cardiovascular risk, functional

status, technical feasibility, nutritional health, and program criteria. Aggressive weight loss that worsens sarcopenia can be counterproductive.

122. B. Metabolic risk commonly persists or worsens after transplantation, and steatotic liver disease can recur or arise de novo. Long-term care includes active management of obesity, diabetes, dyslipidemia, hypertension, and lifestyle. Immunosuppression may be individualized to reduce metabolic toxicity, but it should not simply be stopped.

123. E. Aminotransferase levels correlate poorly with fibrosis stage in individual patients with MASLD. Advanced fibrosis and even cirrhosis can be present despite normal AST and ALT. Risk assessment therefore relies on clinical context, noninvasive fibrosis tests, imaging, and complications rather than enzyme values alone.

124. B. Intrahepatic cholestasis of pregnancy classically presents in later pregnancy with pruritus and elevated bile acids, often with modest aminotransferase abnormalities. Maternal hepatic failure is unusual, but fetal risks increase with disease severity. Management is coordinated with obstetrics and includes symptom treatment and delivery planning based on bile-acid burden and gestational age.

125. A. HELLP is defined by hemolysis, elevated liver enzymes, and low platelets in the setting of a hypertensive pregnancy disorder. Maternal stabilization and timely delivery are central to management. Severe hepatic complications such as hematoma or rupture require urgent multidisciplinary care.

126. E. DILI is a diagnosis of careful attribution and exclusion. Prompt withdrawal of a suspected hepatotoxic agent is fundamental, while timing, biochemical injury pattern, competing diagnoses, and known drug signatures are assessed. Deliberate rechallenge is generally avoided when the initial injury was clinically important.

127. D. The R ratio compares ALT and alkaline phosphatase elevations as multiples of their upper limits of normal. A markedly predominant ALT elevation yields a high R ratio and a hepatocellular pattern. The pattern helps organize the differential and causality assessment but does not identify a specific drug by itself.

128. E. ACLF refers to acute deterioration of chronic liver disease accompanied by organ failure and high short-term mortality. Infection is a common precipitant, although no trigger is found in some cases. Management requires aggressive treatment of precipitants, organ support, and early transplant consideration in appropriate candidates.

129. E. The prognostic hallmark of ACLF is organ failure, not simply worsening liver tests or fluid retention. The number and severity of failed organ systems strongly influence short-term mortality and transplant urgency. Acute decompensation without organ failure generally has a different immediate prognosis.

130. C. Palliative care can be concurrent with transplant evaluation and other life-prolonging treatment. It supports symptom control, communication, caregiver needs, and preference-sensitive decisions in a

disease with substantial symptom burden and prognostic uncertainty. Referral does not itself determine transplant eligibility.

131. B. Advance care planning is appropriate even for patients pursuing transplantation because clinical trajectories can change quickly. Clarifying values and identifying a surrogate improves goal-concordant care during periods when the patient may lose decision-making capacity. Preferences can be revisited if circumstances or goals change.

132. C. Pretransplant care includes preventive health measures because vaccine responses are often better before intensive immunosuppression. Immunization plans are individualized for age, exposures, prior immunity, and transplant timing; live vaccines require special timing considerations. Preventive care is part of transplant readiness rather than an optional add-on.

133. B. Donation after circulatory death expands the donor pool and can yield good outcomes with careful selection and preservation. The biliary tree is particularly vulnerable to warm ischemia, increasing the risk of nonanastomotic strictures and ischemic cholangiopathy. Risk is influenced by donor, recipient, ischemic-time, and preservation factors.

134. C. Macrovesicular steatosis can reduce graft tolerance to ischemia and is associated with greater early dysfunction when severe. It is therefore considered within a broader donor-recipient matching decision rather than as a binary characteristic. Preservation strategy and recipient urgency also matter.

135. B. Highly effective direct-acting antiviral therapy permits selected HCV-viremic organs to be transplanted into HCV-negative recipients under structured protocols. Informed consent, reliable access to antivirals, and post-transplant monitoring are essential because transmission is expected in most such transplants. Delaying treatment until advanced graft disease develops is not appropriate.

136. A. Anti-HBc positivity can indicate prior HBV infection with persistent intrahepatic viral templates despite negative HBsAg. Immunosuppression can permit donor-derived HBV infection or reactivation in a susceptible recipient. Antiviral prophylaxis and surveillance substantially mitigate this risk.

137. C. Older donor age can increase vulnerability to ischemic and biliary complications, especially when combined with other risk factors. Nonetheless, many older donor livers function well when appropriately selected and matched. Donor assessment is multidimensional rather than governed by a single age threshold.

138. E. Unexpected donor lesions require rapid risk stratification because transmission risk varies greatly by tumor type, stage, and certainty of diagnosis. Pathology, imaging, procurement findings, and informed risk-benefit discussion guide acceptance. Neither automatic implantation nor indiscriminate discard is appropriate for every lesion.

139. A. Organ-offer decisions involve competing risks: graft-related complications versus continued waiting. A graft that is inappropriate for a stable low-risk candidate may be reasonable for a patient with

substantial wait-list mortality. Shared decision-making and center expertise are important in this individualized assessment.

140. A. Living liver donation exposes a healthy person to major surgery solely for another person's benefit. Evaluation must therefore prioritize donor autonomy, comprehension, voluntariness, psychosocial context, and medical safety. Independent donor advocacy helps separate donor interests from recipient urgency.

141. B. Donor safety is the dominant constraint in living donation. An insufficient future liver remnant increases the risk of donor hepatic failure and is unacceptable without a safer alternative strategy. Recipient benefit cannot justify disproportionate donor risk.

142. C. Steatosis and metabolic liver disease matter because donation removes substantial hepatic mass from an otherwise healthy person and because steatosis can impair graft and donor remnant function. Metabolic risk factors therefore trigger careful imaging, laboratory assessment, and sometimes biopsy. Remote nonhepatic conditions generally have much less relevance.

143. B. A graft that is too small for the recipient's metabolic and portal-flow demands may develop small-for-size syndrome, characterized by poor synthetic function, cholestasis, ascites, and portal hyperperfusion. Prevention depends on graft sizing, inflow/outflow optimization, and donor-recipient selection. Vascular patency does not exclude the syndrome.

144. A. Both the donor remnant and partial graft can regenerate, enabling living donor transplantation. However, regeneration takes time and does not remove the immediate risks of hemorrhage, bile leak, infection, thrombosis, or hepatic insufficiency. This distinction is central to informed consent.

145. B. Voluntariness is indispensable in living donation. The donor must be able to withdraw without coercion, and transplant programs protect confidentiality around a decision not to proceed. Family or financial pressure requires careful independent evaluation rather than accelerated surgery.

146. A. Split liver transplantation can expand access by serving two recipients from one donor when anatomy and graft volume are suitable. Successful use requires careful matching of functional graft mass and vascular/biliary structures. Recipient size and technical feasibility are central considerations.

147. D. The liver is relatively tolerant compared with some organs, but ABO-incompatible transplantation still carries increased antibody-mediated and biliary risk. It can be lifesaving in selected urgent or pediatric settings when appropriate protocols are available. The risk-benefit calculation is individualized.

148. E. Hepatic arterial variants are common and influence procurement and reconstruction. Failure to identify them can lead to injury or compromised inflow, but many variants can be safely managed by experienced surgical teams. Detailed donor vascular mapping is therefore important.

149. A. Portal vein thrombosis can complicate liver transplantation, particularly when extensive, but it is not uniformly prohibitive. Preoperative imaging defines the extent and helps the surgical team plan thrombectomy, jump grafts, or other inflow reconstruction. Outcomes depend on anatomy and center expertise.

150. E. Reperfusion can cause abrupt hemodynamic instability through release of cold, acidotic, potassium-rich blood and vasoactive mediators from the graft. Management includes anticipatory correction of electrolytes and acid-base abnormalities plus rapid hemodynamic support. It is an intraoperative event, not chronic rejection.

151. E. Primary nonfunction is catastrophic early graft failure without another reversible explanation such as major vascular occlusion. Severe synthetic failure, acidosis, hemodynamic instability, and absent functional recovery prompt urgent evaluation for retransplantation. It must be distinguished from potentially reversible early dysfunction.

152. C. Early allograft dysfunction describes suboptimal early graft performance manifested by abnormalities such as bilirubin, INR, or aminotransferases. Unlike primary nonfunction, many grafts recover with supportive care. The distinction is clinically important because primary nonfunction may require urgent retransplantation.

153. E. Early hepatic artery thrombosis threatens both hepatocytes and, critically, the arterial blood supply of the bile ducts. Prompt diagnosis and revascularization can sometimes salvage the graft; otherwise retransplantation may be required. Delay risks graft necrosis, sepsis, and ischemic cholangiopathy.

154. E. The liver parenchyma has dual arterial and portal inflow, but the bile ducts are much more dependent on arterial perfusion. Loss of hepatic arterial flow can therefore produce bile-duct necrosis, leaks, nonanastomotic strictures, and infected bilomas. This vascular-biliary relationship is a core post-transplant concept.

155. B. Bilious drain output soon after transplantation strongly suggests leakage from the biliary reconstruction or a cut surface in partial grafts. Imaging and cholangiographic evaluation define the site, and management may involve endoscopic stenting, drainage, or surgical repair. Untreated leaks can lead to biloma and infection.

156. D. Early post-transplant AKI is common and often multifactorial. Intraoperative hypotension, blood loss, ischemia-reperfusion, baseline kidney dysfunction, infection, and nephrotoxins can all contribute. Evaluation should not automatically attribute immediate AKI to calcineurin inhibitors alone.

157. B. Hyperkalemia may worsen with reperfusion as potassium-rich, acidotic graft effluent enters the circulation. Anticipatory correction and standard rapid hyperkalemia therapies reduce arrhythmia risk. Mineralocorticoid antagonists do not provide immediate treatment in this setting.

158. B. Transplant-associated bleeding is complex and reflects surgical sources plus dynamic changes in coagulation, platelets, fibrinogen, fibrinolysis, temperature, and acid-base status. Management is guided by clinical bleeding and, when available, viscoelastic testing rather than INR normalization alone. Overtransfusion also has important harms.

159. E. TRALI presents with acute hypoxemic noncardiogenic pulmonary edema temporally related to transfusion. It must be distinguished from transfusion-associated circulatory overload and other perioperative pulmonary causes. Management is supportive, with avoidance of unnecessary transfusion exposure.

160. B. Portal vein thrombosis can cause severe graft dysfunction by compromising a major source of hepatic blood flow. Early recognition with Doppler and confirmatory imaging is critical because thrombectomy, revision, or other revascularization may salvage the graft. Extensive thrombosis can threaten graft survival.

161. C. A focal anastomotic narrowing with marked velocity increase suggests portal vein stenosis, particularly when accompanied by graft dysfunction or portal-hypertensive findings. Cross-sectional or invasive imaging can confirm significance. Selected lesions can be treated with angioplasty or stenting.

162. B. Significant portopulmonary hypertension increases perioperative risk because the right ventricle may not tolerate abrupt changes in preload, afterload, hypoxia, or reperfusion physiology. Pretransplant hemodynamic assessment and optimization are therefore essential. Severe uncontrolled pulmonary vascular disease can preclude safe transplantation.

163. D. Some donor bloodstream infections can be managed safely when the pathogen is susceptible, donor therapy is adequate, there is no uncontrolled focus, and the recipient receives appropriate post-transplant treatment. Transmission risk varies by organism and clinical context. This is a structured risk-benefit decision rather than a universal exclusion.

164. E. Strongyloides can persist for years through autoinfection and may disseminate catastrophically during immunosuppression. Donor or recipient epidemiologic risk should prompt screening or empiric management according to transplant-infectious-disease protocols. Early recognition is critical because hyperinfection can be fatal.

165. C. Uncontrolled disseminated invasive fungal infection in a donor poses a serious risk of donor-derived disease. Organ acceptance depends on organism, site, control of infection, and urgency, but disseminated infection is generally a major contraindication. Transplant-infectious-disease expertise is crucial for borderline scenarios.

166. C. Clusters of unusual early infections among recipients from a common donor are a classic signal of possible donor-derived transmission. Rapid reporting and communication can trigger testing and preemptive therapy in other exposed recipients. The priority is coordinated safety action while treating the ill patient.

167. E. Donor malignancy risk is heterogeneous. Some treated tumors carry negligible or low transmission risk, whereas others remain unacceptable even after treatment. Acceptance decisions therefore integrate tumor biology, certainty of cure, recipient urgency, and informed consent.

168. A. Prolonged cold ischemia depletes cellular energy and primes endothelial and hepatocyte injury that becomes amplified on reperfusion. The effect is especially important in marginal grafts such as steatotic or older livers. Preservation strategies aim to reduce this injury while maintaining organ viability.

169. E. Machine perfusion technologies can support metabolism outside the body and provide additional information about graft function before implantation. They may expand use of selected marginal organs and mitigate preservation injury, but they do not abolish immunologic, vascular, or biliary risk. Acceptance remains a clinical decision.

170. D. Severe intraoperative hypotension can have multiple simultaneous causes, including bleeding, preload shifts, postreperfusion syndrome, myocardial dysfunction, pulmonary hypertension, and metabolic abnormalities. Management is rapid and physiology-directed. Assuming a single diagnosis without assessment can miss a reversible life-threatening cause.

171. A. The liver is a major site of lactate clearance, so removal of the native liver reduces clearance while surgical stress and hypoperfusion may increase production. Lactate trends after reperfusion also provide information about systemic perfusion and early graft metabolism. Interpretation must remain contextual rather than absolute.

172. B. Acute T-cell-mediated rejection is diagnosed histologically by a characteristic triad that includes portal inflammation, bile-duct injury, and venous endothelial inflammation. Liver tests are nonspecific, so competing vascular and biliary causes should be excluded. Most clinically significant episodes respond to intensified immunosuppression, often corticosteroids.

173. A. Clinically important acute T-cell-mediated rejection is commonly treated with corticosteroid pulses plus optimization of baseline immunosuppression. Infection, medication adherence, drug levels, and alternative graft injuries should be assessed in parallel. Retransplantation is rarely the first response to a typical treatable episode.

174. D. Steroid-resistant acute T-cell-mediated rejection may require lymphocyte-depleting therapy after histologic confirmation and exclusion of mimics. Escalation substantially increases infectious and hematologic risk, so treatment is individualized and monitored closely. Persistent severe rejection can threaten graft survival.

175. E. Chronic rejection is characterized by progressive bile-duct loss and vascular injury and can lead to irreversible graft dysfunction. It may follow inadequately controlled or recurrent rejection, including episodes related to nonadherence. Early disease may respond to optimization of immunosuppression, whereas advanced graft failure may require retransplantation.

176. D. Antibody-mediated rejection is less common in liver than in some other solid organs but is recognized when graft injury occurs with compatible histology and evidence of donor-specific antibody, often with supportive C4d findings. Diagnosis requires exclusion of other causes. Treatment may combine intensified immunosuppression with antibody-directed approaches in severe cases.

177. A. Some liver recipients may achieve operational tolerance, but reliable prediction is difficult and rejection can be clinically silent. Standard practice therefore uses individualized long-term immunosuppression at the lowest effective exposure. Research withdrawal protocols require careful selection and biopsy-based monitoring.

178. E. Nonadherence is a major and potentially modifiable cause of late rejection, particularly during adolescence and transition to adult care. Assessment should be nonjudgmental and address routines, understanding, mental health, access, family support, and medication adverse effects. Repeated rejection can culminate in chronic graft injury.

179. D. Abnormal liver tests after transplantation have a broad differential that includes rejection, biliary disease, vascular complications, infection, drug injury, and recurrent disease. When noninvasive evaluation does not identify the cause, biopsy is often necessary to establish rejection and its severity. Drug levels inform management but are not diagnostic tests for rejection.

180. B. Rejection can occur long after transplantation, especially when immunosuppression is reduced or adherence is poor. Repeated or severe late episodes can cause progressive duct and vascular injury and contribute to chronic rejection. Long-term adherence and thoughtful minimization are therefore essential.

181. D. Plasma-cell-rich inflammatory graft injury can represent an alloimmune phenotype and overlaps histologically with autoimmune hepatitis. Interpretation requires the original diagnosis, serologies, immunoglobulins, medication history, and exclusion of infections and other graft insults. Management usually involves adjustment of immunosuppression based on the integrated diagnosis.

182. D. Anastomotic strictures are usually short, focal lesions at the surgical connection and commonly present with cholestatic laboratory abnormalities. Endoscopic dilation and stenting are effective in many duct-to-duct reconstructions. Multifocal nonanastomotic disease raises different concerns such as ischemic injury.

183. C. Nonanastomotic strictures and biliary casts after transplantation can reflect ischemic cholangiopathy, particularly after DCD grafts or prior arterial compromise. The bile ducts are highly dependent on arterial microcirculation, and injury can persist even when current major-vessel flow is patent. Severe cases may require repeated interventions or retransplantation.

184. B. Chronic kidney disease is a major long-term complication after liver transplantation. Calcineurin inhibitors can contribute through renal vasoconstriction and chronic vascular injury, but diabetes,

hypertension, prior AKI, and other causes also matter. Management aims to preserve graft protection while minimizing nephrotoxic exposure and controlling conventional renal risks.

185. E. Hypertension is common after transplantation and is promoted by calcineurin inhibitors, corticosteroids, kidney disease, weight gain, and preexisting cardiovascular risk. Treatment follows standard cardiovascular principles while accounting for drug interactions and kidney function. Uncontrolled hypertension adds to renal and cardiovascular morbidity.

186. B. New or worsened diabetes is common after transplant and contributes to cardiovascular, renal, and infectious risk. Management combines standard diabetes therapy, lifestyle measures, and immunosuppression optimization when feasible. Some cases improve as steroid exposure falls, but persistent disease requires long-term treatment.

187. B. Dyslipidemia is common after transplantation and may be exacerbated by specific immunosuppressants, including mTOR inhibitors. Cardiovascular risk reduction includes diet, exercise, pharmacologic lipid lowering, and thoughtful medication adjustment. Drug-drug interactions should be reviewed when lipid-lowering therapy is selected.

188. E. Calcineurin inhibitors can cause neurotoxicity ranging from tremor and headache to seizures and PRES, especially with high exposure, hypertension, renal dysfunction, or interacting drugs. Management includes blood-pressure control, seizure treatment when needed, and reduction or modification of the offending immunosuppressant. PRES is often reversible when recognized promptly.

189. B. Bone disease often predates transplantation and can worsen early afterward because of corticosteroids, inactivity, nutritional deficits, and endocrine factors. Risk assessment includes bone-density testing when indicated, vitamin D and calcium optimization, exercise, and pharmacologic osteoporosis therapy for appropriate patients. Fracture prevention remains part of long-term transplant care.

190. C. Successful pregnancy is possible after liver transplantation, but timing and medication review are crucial. Mycophenolate is strongly teratogenic and requires preconception replacement, whereas selected calcineurin inhibitors and azathioprine may be used with monitoring. Maternal hypertension, renal function, rejection, and fetal growth need coordinated surveillance.

191. E. Cytopenias after transplantation are often multifactorial. Valganciclovir, mycophenolate, and trimethoprim-sulfamethoxazole can suppress marrow, while CMV and other infections may contribute. Management balances the seriousness of cytopenia against the consequences of reducing antiviral prophylaxis or immunosuppression.

192. E. As graft survival improves, nonhepatic disease becomes increasingly important. Metabolic syndrome, cardiovascular disease, and chronic kidney disease are major determinants of long-term outcomes. Prevention and aggressive risk-factor modification are therefore core components of transplant follow-up.

193. B. Some liver recipients develop irreversible advanced kidney disease despite renal-sparing strategies. Selected patients may benefit from subsequent kidney transplantation, with eligibility determined by the severity and persistence of renal dysfunction and applicable allocation policy. Early reversible AKI should not be confused with chronic kidney failure.

194. E. CMV risk is highest in seronegative recipients who receive organs from seropositive donors. CMV may cause a systemic syndrome with fever, malaise, cytopenias, and viremia or tissue-invasive disease. Prevention and treatment strategies depend on donor-recipient serostatus, immunosuppression, prophylaxis, and viral monitoring.

195. A. Blood CMV testing supports the diagnosis but does not by itself prove tissue invasion at a symptomatic site. Histopathologic demonstration in involved tissue is important when feasible, particularly for gastrointestinal disease. Treatment generally uses systemic anti-CMV therapy with attention to drug toxicity and immunosuppression.

196. C. Pneumocystis pneumonia is an important opportunistic infection in heavily immunosuppressed solid-organ recipients, particularly without prophylaxis. The presentation often includes progressive dyspnea, hypoxemia, and diffuse ground-glass infiltrates. Trimethoprim-sulfamethoxazole is commonly used for both prophylaxis and treatment when tolerated.

197. D. VZV reactivation can be more severe in immunocompromised hosts and may disseminate beyond a dermatome. Prompt antiviral therapy and assessment of disease extent are important, with intravenous treatment for severe or disseminated disease. Prevention includes appropriate pretransplant vaccination when feasible.

198. E. Invasive aspergillosis is a life-threatening opportunistic infection in heavily immunosuppressed recipients and may present with nodules, infarct-like lesions, or cavitation. Diagnosis uses imaging, microbiology, antigen testing, and sometimes tissue. Prompt mold-active therapy is essential while drug interactions with immunosuppressants are managed.

199. A. Biliary obstruction predisposes transplant recipients to ascending bacterial cholangitis and sepsis. Management requires prompt antimicrobial therapy and relief of the obstructed biliary system, usually by endoscopic or percutaneous means depending on anatomy. Rejection and infection can coexist, but septic obstruction is an emergency.

200. D. PTLD ranges from polyclonal proliferations to aggressive lymphoma and is often linked to EBV, particularly in seronegative recipients with primary infection under intense immunosuppression. Diagnosis requires tissue when feasible. Treatment commonly begins with reduction of immunosuppression and may include rituximab or lymphoma-directed therapy.

201. A. If viremia is present at transplantation, HCV can infect the allograft. Modern direct-acting antiviral regimens achieve high cure rates in transplant recipients and have transformed the natural history of recurrent HCV. Drug interactions and graft/renal function guide regimen selection.

202. D. HBV can persist outside the removed native liver and recur under immunosuppression. Potent nucleos(t)ide analogues are central to prevention, with use of hepatitis B immune globulin tailored to risk and center practice. Maintaining viral suppression protects the graft from recurrent HBV disease.

203. B. PBC can recur after transplantation, usually over years, and must be distinguished from rejection, biliary obstruction, infection, and drug injury. Histology is often important because antimitochondrial antibodies may persist regardless of graft disease. Recurrence does not usually threaten graft survival early but can become clinically important over time.

204. A. Recurrent PSC is a diagnosis that requires compatible cholangiographic or histologic disease after exclusion of other transplant-specific causes of biliary injury. Ischemic cholangiopathy, arterial problems, infection, and chronic rejection can mimic it. Clinical timing and the original disease context help interpretation.

205. C. Autoimmune hepatitis can recur after transplantation and may resemble plasma-cell-rich rejection. The diagnosis integrates original disease, autoantibodies, IgG, histology, adherence, and exclusion of competing graft injuries. Treatment generally involves optimization of immunosuppression.

206. B. Alcohol exposure can injure the transplanted liver and contributes to medical, psychiatric, adherence, and social complications. Long-term transplant care should include nonstigmatizing surveillance and access to evidence-based treatment for alcohol use disorder. Relapse management is a clinical treatment issue rather than merely a punitive one.

207. B. The cardiometabolic drivers of MASLD often persist or worsen after transplantation because of weight gain, diabetes, dyslipidemia, and immunosuppressive effects. Steatosis can therefore recur in patients transplanted for MASH or develop de novo. Prevention and treatment focus on long-term metabolic risk reduction.

208. A. Chronic immunosuppression substantially increases the incidence and aggressiveness of cutaneous malignancy, particularly squamous-cell carcinoma. Prevention includes sun avoidance/protection, self-awareness, and professional skin surveillance. Immunosuppression may be modified in patients with recurrent malignancy when graft safety permits.

209. C. Vaccination remains an important preventive strategy after transplantation, although immune responses may be reduced. Inactivated vaccines are generally safe, whereas live vaccines are usually avoided during significant immunosuppression except in carefully selected settings. Whenever possible, immunization is optimized before transplantation.

210. D. Transplantation does not remove the elevated colorectal neoplasia risk associated with PSC and colitis. Ongoing colonoscopic surveillance remains important, often at shorter intervals than average-risk screening. Immunosuppression may further increase malignancy concerns over time.

211. B. Long-term transplant survival is strongly influenced by cardiovascular disease and malignancy. Tobacco exposure worsens both and should be addressed with evidence-based cessation support. Preventive care remains important even when graft function is excellent.

212. A. Cancer prevention after transplantation includes routine population-based screening plus attention to transplant-specific risks such as skin cancer, PTLD, and recurrence of selected pretransplant malignancies. Screening should reflect age, sex, prior disease, exposures, and immunosuppression. A one-size-fits-all annual whole-body scan is not appropriate.

213. B. HCC can recur after transplantation, usually because of occult micrometastatic disease present before surgery. Surveillance intensity is tailored to recurrence risk using clinical and explant features and typically includes cross-sectional imaging with or without tumor markers. The balance between cancer risk and graft rejection also informs immunosuppressive strategy.

214. B. Modern transplant survivorship requires more than monitoring the liver graft. Cardiovascular disease, kidney disease, metabolic syndrome, bone disease, infection, and malignancy contribute substantially to long-term morbidity. Shared care between transplant specialists and primary-care teams improves comprehensive prevention and management.

215. E. Allorecognition occurs through direct and indirect pathways. In direct recognition, recipient T cells react to intact donor HLA-peptide complexes; in indirect recognition, recipient antigen-presenting cells process donor proteins and present donor-derived peptides. These pathways contribute to acute and chronic alloimmune injury.

216. D. The liver has distinctive immunologic properties that can favor tolerance, including abundant antigen exposure and specialized innate and adaptive cell interactions. Nevertheless, acute, chronic, and antibody-mediated rejection occur, and most recipients require long-term immunosuppression. Relative tolerogenicity is therefore a biologic tendency rather than complete immune privilege.

217. A. Tacrolimus is a calcineurin inhibitor. The tacrolimus-FKBP complex prevents calcineurin-dependent activation of NFAT and reduces transcription of IL-2 and other cytokines required for T-cell activation. Its major toxicities include nephrotoxicity, neurotoxicity, hypertension, and metabolic effects.

218. D. Tacrolimus has a narrow therapeutic index and is metabolized by CYP3A pathways. Macrolides such as clarithromycin and many azole antifungals can raise concentrations substantially, producing nephrotoxicity or neurotoxicity. Medication reconciliation and timely trough monitoring are essential when interacting drugs are started or stopped.

219. A. Potent enzyme induction can reduce calcineurin-inhibitor concentrations and create rejection risk if not recognized. Rifampin is a classic strong inducer. When interacting therapy is unavoidable, close drug-level monitoring and coordinated dose adjustment are required.

220. C. Tacrolimus and cyclosporine share calcineurin inhibition and several class toxicities, especially renal vasoconstriction and chronic nephrotoxicity, hypertension, neurotoxicity, and electrolyte disturbances. Tacrolimus is more strongly associated with post-transplant diabetes, whereas cyclosporine more often causes gingival hyperplasia and hirsutism. Toxicity management requires balancing graft protection against cumulative organ injury.

221. B. Mycophenolate inhibits inosine monophosphate dehydrogenase and therefore de novo purine synthesis, a pathway on which activated lymphocytes depend heavily. This limits T- and B-cell proliferation. Important adverse effects include gastrointestinal toxicity, leukopenia, infection risk, and teratogenicity.

222. E. Mycophenolate exposure in pregnancy is associated with pregnancy loss and characteristic congenital malformations. Patients planning pregnancy should transition to an appropriate alternative before conception under transplant supervision. Immunosuppression should not be stopped indiscriminately because maternal graft rejection also threatens pregnancy.

223. B. Azathioprine metabolites are partly cleared through xanthine oxidase. Allopurinol or febuxostat can markedly increase active thiopurine exposure and cause life-threatening marrow toxicity unless expert dose modification is used. Medication interaction review is therefore essential.

224. E. mTOR inhibitors such as sirolimus and everolimus can cause dyslipidemia, edema, mouth ulcers, cytopenias, proteinuria, impaired wound healing, and other toxicities. They may permit calcineurin-inhibitor minimization in selected recipients, but adverse effects can limit use. Monitoring must include renal and metabolic parameters.

225. E. mTOR signaling is important for cellular proliferation and tissue repair. Pharmacologic inhibition can therefore impair wound healing and contribute to wound dehiscence or related complications in susceptible patients. The clinical value of renal-sparing or antineoplastic effects must be balanced against this timing-dependent toxicity.

226. C. Basiliximab is a monoclonal antibody directed against CD25, the IL-2 receptor alpha chain expressed on activated T cells. It is used as nondepleting induction in selected solid-organ transplant strategies. Its use can facilitate delayed or reduced early calcineurin-inhibitor exposure in recipients at renal risk.

227. C. Antithymocyte globulin produces profound lymphocyte depletion and can rescue severe steroid-resistant T-cell-mediated rejection. Its potency also increases risks such as infusion reactions, cytopenias, opportunistic infection, and malignancy. Use therefore requires careful prophylaxis and monitoring.

228. A. Long-term immunosuppression is individualized to balance rejection against drug toxicity. In recipients with chronic kidney disease, calcineurin exposure may be minimized and other agents used to

maintain adequate immunosuppressive potency. Changes should be gradual, monitored, and tailored to rejection history and comorbidity.

229. E. A competent patient may accept or refuse an offered organ after receiving material information about benefits, risks, alternatives, and consequences. Clinicians should ensure understanding and address reversible misconceptions, but they do not simply substitute their own preferences. Respect for autonomy coexists with beneficence and stewardship.

230. E. Living donors retain independent confidentiality rights. Programs often provide a neutral explanation that donation will not proceed without revealing private medical or psychosocial details. Protecting confidentiality also helps potential donors withdraw safely from coercive situations.

231. A. Transplant systems distinguish reimbursement of legitimate donor expenses from buying organs. Commercialization creates risks of coercion, exploitation, inequity, and hidden medical harm. Living donation requires regulated evaluation, voluntariness, informed consent, and donor protection.

232. D. Scarce organs should be allocated according to transparent, consistently applied medical and policy criteria. Financial status or perceived social worth should not purchase priority. Justice in transplantation requires both equitable access and stewardship of scarce donated organs.

233. B. Severity models support transparent urgency-based allocation by estimating short-term mortality risk from objective recipient variables. They improve consistency but do not capture every disease-specific risk, which is why carefully governed exception pathways exist. Allocation remains a policy framework rather than a bedside prognostic score alone.

234. D. Some liver diseases create mortality or progression risk that standard laboratory scores underestimate. Exception pathways are intended to recognize defined circumstances using standardized criteria and review, balancing individual need with fairness to other candidates. They are not unrestricted discretionary points.

235. B. HCC illustrates the limitation of laboratory-based mortality scores: a patient may have compensated liver function yet face loss of transplant candidacy from tumor progression. Standardized criteria seek to balance this risk against the urgency of non-HCC candidates. Tumor stage, treatment response, and biology therefore remain relevant.

236. B. Transplantation requires complex lifelong care, so adherence capacity matters, but social disadvantage should not be treated as a moral failing. Programs should distinguish modifiable access barriers from persistent behavior that threatens safety and should mobilize social support where possible. Equity requires avoiding unsupported proxies such as wealth or employment.

237. E. Alcohol use disorder is a treatable medical condition, not a basis for assigning lesser social worth. Evaluation should use evidence-informed relapse-risk assessment, engagement in treatment, support, insight, and the expected medical benefit of transplantation. The same principles of stewardship and fairness apply as in other transplant indications.

238. C. Transplant candidacy is dynamic. New medical conditions can change perioperative risk, recurrence risk, life expectancy, or the likelihood of meaningful graft benefit, requiring reassessment. Decisions should be documented, communicated, and consistent with transparent program and allocation standards.

239. A. Informed consent for transplantation includes risks that are material to the specific organ offer as well as the risk of declining and continuing to wait. The purpose is not merely to obtain a signature but to support a voluntary, informed decision under time pressure. Disclosure should be understandable and clinically relevant.

240. B. Allocation systems require integrity and consistent application, but they also require ongoing evaluation and revision as evidence and values evolve. Clinicians can participate in policy development, public comment, data review, and professional advocacy without falsifying records or bypassing rules. Transparency is essential to maintaining public trust in donation and transplantation.