Alcohol-Associated Liver Cirrhosis

Diagnosis, staging, complications & management

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From alcohol exposure to cirrhosis

Alcohol-associated liver disease (ALD) is a spectrum; cirrhosis may be clinically silent until decompensation.

Disease spectrum

Steatosis → alcohol-associated steatohepatitis/inflammation → fibrosis → cirrhosis

Progression is influenced by alcohol dose/duration, sex, genetics, nutrition and coexisting liver disease.

Continued alcohol exposure increases risk of decompensation and mortality.

Clinical phenotype

Compensated: no ascites, variceal hemorrhage, hepatic encephalopathy or jaundice attributable to liver failure.

Decompensated: ascites, variceal bleeding, hepatic encephalopathy and/or other major complications.

Acute-on-chronic liver failure can occur with infection, bleeding, alcohol-associated hepatitis or other precipitants.

Diagnosis: history & examination

History

Quantify alcohol exposure: pattern, quantity, duration, binge episodes and recent change.

Screen for AUD and withdrawal risk; assess prior treatment attempts.

Look for viral hepatitis, metabolic risk, autoimmune disease, iron overload and drug/supplement exposure.

Ask about ascites, edema, GI bleeding, confusion/sleep reversal, pruritus and weight loss.

Examination

Stigmata of chronic liver disease: jaundice, spider angiomas, palmar erythema, muscle wasting.

Portal hypertension: splenomegaly, thrombocytopenia, ascites, caput medusae.

Decompensation: edema/ascites, asterixis or encephalopathy, hypotension, infection signs.

Assess frailty, sarcopenia and nutritional status.

Diagnosis: laboratory & imaging work-up

Laboratory assessment

CBC, AST/ALT, ALP, GGT, bilirubin, albumin, INR/PT, creatinine, sodium.

Calculate severity scores: Child–Pugh and MELD-Na/MELD where appropriate.

Screen alternative/coexisting etiologies: HBV/HCV, iron studies ± autoimmune/metabolic tests guided by context.

Evaluate nutrition and common deficiencies; consider infection work-up when clinically indicated.

Imaging / fibrosis

Ultrasound with Doppler: liver morphology, portal vein, splenomegaly and ascites.

Transient elastography or other non-invasive liver disease assessment can support fibrosis/portal hypertension assessment.

CT/MRI when complications, focal lesions or another diagnosis is suspected.

Liver biopsy is not routine; consider when diagnosis is uncertain or competing etiologies/atypical features remain.

Assess portal hypertension & variceal risk

Look for clinically significant portal hypertension (CSPH): thrombocytopenia/splenomegaly, collaterals, ascites and/or validated non-invasive criteria.

Current guidance supports non-invasive risk stratification; selected low-risk patients may avoid screening EGD.

For patients who need endoscopic assessment, evaluate for esophageal/gastric varices and stigmata of bleeding risk.

Non-selective beta-blockers (especially carvedilol in appropriate patients) may reduce risk of decompensation in compensated cirrhosis with CSPH and are also used for variceal prophylaxis when tolerated.

Always individualize beta-blocker use in hypotension, renal dysfunction, refractory ascites or other clinical instability.

Core management: treat the cause and the whole patient

Alcohol abstinence is the cornerstone: diagnose and actively treat AUD with behavioral therapy, addiction-medicine support and pharmacotherapy when appropriate.

Nutrition: adequate calories and protein; avoid unnecessary protein restriction. Address sarcopenia and micronutrient deficiencies.

Vaccination and prevention: HAV, HBV, influenza, pneumococcal, COVID-19 and other age/risk-appropriate vaccines.

Avoid **hepatotoxic drugs** and unnecessary sedatives; review all OTC/herbal products.

Screen/manage **metabolic**, cardiovascular and psychiatric comorbidities; smoking cessation and physical activity are beneficial when feasible.

Coordinate hepatology, addiction medicine, nutrition and primary care.

Ascites & spontaneous bacterial peritonitis (SBP)

Ascites

Diagnostic paracentesis for new-onset clinically significant ascites and when hospitalized or infection/SBP is suspected.

Salt restriction; avoid routine fluid restriction unless significant hyponatremia.

Diuretics (spironolactone ± furosemide) for appropriate patients; monitor weight, renal function, Na/K.

Large-volume paracentesis for tense/refractory ascites; albumin replacement is used with large-volume removal.

SBP

Ascitic fluid PMN ≥250 cells/mm^3 is diagnostic in the appropriate clinical context.

Treat promptly with appropriate antibiotics; obtain cultures before antibiotics when feasible.

Albumin is indicated in selected high-risk SBP, particularly with renal dysfunction/rising creatinine.

Secondary prophylaxis is indicated after an episode of SBP; primary prophylaxis is reserved for selected high-risk patients.

Variceal hemorrhage: emergency management

Resuscitate carefully; protect airway when indicated. Avoid over-transfusion—use a restrictive RBC strategy in most patients.

Start vasoactive therapy promptly (e.g., octreotide/terlipressin according to local protocols) and prophylactic antibiotics.

Urgent upper endoscopy with endoscopic variceal ligation for esophageal variceal bleeding.

Consider early/pre-emptive TIPS in selected high-risk patients; rescue TIPS for uncontrolled bleeding.

After control: secondary prophylaxis with non-selective beta-blocker + repeat band ligation as indicated.

Hepatic encephalopathy (HE)

Recognize & precipitant search

Diagnosis is clinical; ammonia level alone does not establish or grade HE.

Look for infection, GI bleeding, constipation, dehydration, electrolyte disorders, renal failure and sedatives.

Exclude alternative causes of altered mental status (hypoglycemia, stroke, intoxication, etc.).

Treatment

Lactulose is first-line for overt HE; titrate to about 2–3 soft stools/day.

Rifaximin can be added for recurrent overt HE despite lactulose.

Do not chronically restrict protein; adequate protein intake supports muscle and ammonia handling.

Educate caregivers about recurrence and medication adherence.

Hepatorenal syndrome-AKI & renal protection

Monitor creatinine, sodium, urine output and volume status closely in decompensated cirrhosis.

Identify and reverse precipitants: infection, hypovolemia, GI bleeding, nephrotoxins and excessive diuresis.

Avoid NSAIDs and other nephrotoxins; use diuretics cautiously during AKI.

HRS-AKI: albumin plus a vasoconstrictor is standard pharmacologic therapy; terlipressin is preferred where available, with norepinephrine as an ICU alternative.

Consider renal replacement therapy as a bridge in selected patients, particularly when transplant is feasible.

Hepatocellular carcinoma surveillance

All patients with cirrhosis should undergo HCC surveillance.

Standard surveillance: abdominal ultrasound every 6 months, with AFP commonly included under current AASLD guidance.

If ultrasound is inadequate or a lesion is detected, proceed to appropriate contrast-enhanced CT or MRI according to HCC diagnostic algorithms.

Do not use routine CT/MRI as the surveillance test for every patient unless specific circumstances warrant it.

When to consider liver transplantation

Refer early when cirrhosis becomes decompensated, MELD-based risk rises, or complications become recurrent/refractory.

Major triggers: refractory ascites, recurrent variceal bleeding, recurrent HE, HRS-AKI, severe frailty/sarcopenia or HCC within transplant criteria.

Transplant evaluation should include medical, psychosocial and addiction assessment.

Modern practice emphasizes **individualized assessment** rather than relying on a fixed duration of abstinence alone; sustained recovery and ability to maintain post-transplant care are key.

Practical follow-up checklist

At routine visits

Alcohol use / AUD treatment engagement

Weight, nutrition, frailty and sarcopenia

CBC, CMP/liver panel, INR and renal function as clinically indicated

Medication review, vaccination status and infection prevention

Ascites/edema, HE symptoms and GI bleeding history

Surveillance

HCC: ultrasound ± AFP every 6 months

Portal hypertension/varices: follow current non-invasive/endoscopic strategy

Renal function and sodium in decompensated disease

Bone health and fall risk where appropriate

Reassess transplant candidacy as disease trajectory changes

Key take-home messages

1. Confirm cirrhosis and exclude/co-manage other liver diseases.
2. Treat alcohol use disorder aggressively—abstinence changes the disease trajectory.
3. Stage severity and portal hypertension; distinguish compensated from decompensated disease.
4. Prevent complications with evidence-based surveillance and prophylaxis.
5. Treat ascites, SBP, variceal bleeding, HE and HRS-AKI promptly when they occur.
6. Refer for transplant evaluation early in clinically significant decompensation.

Selected references & guidance

AASLD. Alcohol-Associated Liver Disease Practice Guidance. Updated July 2019.

AASLD. Practice Guidance on Risk Stratification and Management of Portal Hypertension and Varices in Cirrhosis. 2023/2024 guidance.

AASLD. Practice Guidance on the Use of TIPS, Variceal Embolization, and Retrograde Transvenous Obliteration in Variceal Hemorrhage. Updated 2023.

AASLD. HCC guidance: surveillance of adults with cirrhosis using ultrasound ± AFP every 6 months.

EASL. Clinical Practice Guidelines: Management of alcohol-related liver disease. Journal of Hepatology, 2018.

Note: This teaching deck is for educational use and should be adapted to local protocols, formularies and specialist guidance.

Thank you