AAVBC
AMERICAN ACADEMY OF VALUE BASED CARE

Liver Cirrhosis

Quick Reference Guide

2026

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1 CLINICAL SNAPSHOT

Definition: Liver cirrhosis is the fibrotic replacement of normal hepatic parenchyma resulting from chronic hepatocellular injury, characterized by distortion of lobular architecture and formation of regenerative nodules.[1] Cirrhosis spans a clinical spectrum from compensated disease (with median survival >12 years) to decompensated disease, defined by ascites, variceal hemorrhage, hepatic encephalopathy (HE), or jaundice, at which point median survival falls to <1.5 years.[2] Noninvasive diagnosis is now standard using FIB-4 index followed by transient elastography (liver stiffness measurement ≥15 kPa ≈ cirrhosis); liver biopsy is rarely needed. **Major etiologies in older adults: metabolic-associated steatotic liver disease (MASLD/NASH), chronic hepatitis B (HBV) and C (HCV), and alcohol-associated cirrhosis**.

ICD-10 Codes:

Primary cirrhosis codes under **K74.x** (Fibrosis and Cirrhosis of Liver): **K74.60** (unspecified—avoid when etiology known); **K74.69** (other cirrhosis, including NASH/cryptogenic); **K70.30/K70.31** (alcoholic cirrhosis without/with ascites). Secondary stream: **K72.10/K72.11** (chronic hepatic failure without/with coma).[3]

Complication codes: G93.40 (hepatic encephalopathy); **R18.8** (ascites, non-alcoholic); **I85.01/I85.11** (esophageal varices with/without bleeding); **K65.2** (spontaneous bacterial peritonitis); **K76.7** (hepatorenal syndrome).

Associated codes critical: B18.1 (chronic HBV), **B18.2** (chronic HCV), **E11.x** (type 2 diabetes, common in MASLD), E66.01 (morbid obesity), **C22.0** (hepatocellular carcinoma). **Do NOT use Z87.19 (history of liver disease) when cirrhosis is active—this suppresses HCC mapping.**

Prevalence and Burden: Cirrhosis affects approximately **2.2 million adults** in the US, with age-adjusted mortality rising from **14.9 to 21.9 per 100,000 between 2010 and 2021**.[1] Among Medicare Advantage enrollees, cirrhosis prevalence reached **1.67%** in 2018—the highest across all insurance types.[4] Among patients ≥65 newly diagnosed with cirrhosis, NASH is the leading etiology, followed by viral hepatitis and alcohol use; all-cause mortality is significantly higher than in younger cohorts (HR 2.26, 95% CI 1.89–2.69). Comorbidity burden is substantial: **53.8% of cirrhosis patients ≥65 have ≥2 additional chronic diseases** (diabetes, CVD, CKD) compared to **35.8% in ages 40–64.**[2]

HCC/RAF V28 Mapping

ICD-10 CODE(S)	HCC CATEGORY	RAF WEIGHT (CNA)	DOCUMENTATION REQUIREMENT
K74.60 (Unspecified cirrhosis of liver)	HCC 64	0.447	**AVOID** as primary code when etiology is known. Most common coding error. Use only when etiology is undetermined after clinical workup. Requires documentation of diagnostic workup performed (imaging, serology, alcohol history assessment)

ICD-10 CODE(S)	HCC CATEGORY	RAF WEIGHT (CNA)	DOCUMENTATION REQUIREMENT
K74.69 (Other cirrhosis: NASH/ MASLD, cryptogenic, macronodular)	HCC 64	0.447	**Use for NASH/MASLD cirrhosis**. Requires documentation of contributing etiology (obesity, diabetes, hyperlipidemia) and metabolic syndrome components (**E11.x, E66.x, I10, E78.x**) coded separately FIB-4 ≥1.3 in diabetic patients triggers MASLD workup and elastography referral
K70.30 (Alcoholic cirrhosis without ascites)/K70.31 (with ascites)	HCC 64	0.447	Requires documented alcohol use disorder (F10.x) sequenced at every visit—**alcohol is the root cause,** not a comorbidity. Fifth digit changes with ascites: **K70.30** (no ascites) vs. **K70.31** (ascites present). Document alcohol quantity/frequency per AUDIT-C or similar; current use vs. remission status
K72.10/K72.11 (Chronic hepatic failure without/with coma)	HCC 63	0.962	Requires explicit documentation of end-stage liver disease, Child-Pugh C status, or MELD ≥15. 'Cirrhosis alone' does **NOT** support **K72.1x**. K72.11 (with coma) requires West Haven Grade 3–4 HE documented
B18.0, B18.1 (Hepatitis B) and B18.2 (Chronic Hepatitis C)	HCC 65	0.185	Code alongside **K74.x** to document etiology of cirrhosis. **B18.2** (HCV) + direct-acting antivirals (DAAs) may clear HCV but cirrhosis diagnosis persists; document HCV treatment status. **B18.1** (HBV) requires entecavir/tenofovir; cirrhosis diagnosis persists despite treatment. Distinguish from resolved HCV/HBV (no longer **B18.x** codes)

ABBREVIATIONS: AUDIT-C = Alcohol Use Disorders Identification Test—Concise; BCLC = Barcelona Clinic Liver Cancer; CKD = chronic kidney disease; CMS = Centers for Medicare & Medicaid Services; CNA = community non-dual aged; DAA = direct-acting antiviral; E/M = evaluation and management; F10.xx = alcohol use disorder codes; HCC = hierarchical condition category (also hepatocellular carcinoma—context-dependent); HCV = hepatitis C virus; HBV = hepatitis B virus; ICD-10 = International Classification of Diseases, 10th Edition; MELD = Model for End-Stage Liver Disease; RAF = risk adjustment factor; V28 = CMS-HCC model version 28

***RAF values represent the Community Non-Dual Eligible Aged (CNA) coefficient from the 2026 CMS-HCC model; values vary across patient populations based on eligibility and care setting. Child-Pugh C and MELD ≥15 represent distinct clinical thresholds; at least one must be explicitly documented to support K72.1x (HCC 63) rather than K74.x (HCC 64)**

Risk-Adjusted Care Resources per Patient/Year

Risk-adjusted care resource allocation — MA base rate ($10,402.34) × RAF coefficient

Cirrhosis/NASH/MASLD	Hepatic failure
$4.65K	**$10K**
HCC 64 · RAF 0.447	HCC 63 · RAF 0.962

Annual risk value illustrative at base rate ~$10,402/year; actual plan payment varies by county and contract year, RAF values represent the Community Non-Dual Eligible Aged (CNA) coefficient from the 2026 CMS-HCC model; values vary across patient populations based on eligibility and care setting

2 RECOGNITION AND DIAGNOSIS

Diagnostic Workup Covered Under Medicare Part B (older adults, at-risk population)

Every diagnostic test required to identify, stage, and monitor liver cirrhosis in older adults is covered under Medicare Part B when clinically indicated. The barrier to early detection is not coverage but consistent use of a structured screening-to-diagnosis pathway in at-risk populations.[2,5–8]

TEST	COVERAGE	FREQUENCY	CPT CODE	CLINICAL NOTES
Fib-4	Lab tests included in calculation are covered by Medicare Part B	At identification, repeat every **1–2 years** **See AAVBC stance below**	No dedicated CPT code	Screen the following population: (1) patients with **type 2 diabetes,** (2) patients with **≥2 metabolic risk factors** (central obesity, elevated triglycerides, reduced HDL, hypertension, prediabetes), and (3) patients with **incidental findings** of hepatic steatosis or elevated aminotransferases
Liver stiffness measurement (transient elastography; FibroScan)	Medicare Part B when clinically indicated	At diagnosis; repeat **every 2–3 years** if stable compensated cirrhosis	76810 (ultrasound elastography)	LSM ≥15 kPa confirms cirrhosis; avoids need for biopsy. LSM also predicts variceal presence (≥25 kPa → 90% specific for varices). Serial LSM may track disease progression; increasing stiffness signals decompensation risk

TEST	COVERAGE	FREQUENCY	CPT CODE	CLINICAL NOTES
Hepatocellular carcinoma screening: Ultrasound ± Alpha-fetoprotein (AFP)	Medicare Part B; recommended every 6 months for all cirrhotic patients	Semiannually (**every 6 months**) for compensated cirrhosis; do NOT surveil if Child-Pugh C unless transplant candidate	76700 (abdominal ultrasound); 82105 (AFP)	AASLD 2023 standard: US ± AFP every 6 months
Variceal screening: Esopha-gogastroduoden oscopy (EGD)	Medicare Part B; therapeutic endoscopy covered	**At diagnosis** (compensated cirrhosis); Baveno VI/VII criteria guide interval (avoid EGD if platelets >150k, LSM <20 kPa)	43235 (EGD); 43244 (variceal ligation if indicated)	Detects esophageal varices (risk of bleed); gastric varices; portal hypertensive gastropathy. Baveno VI/VII criteria: if platelets >150k AND LSM <20 kPa, varices unlikely (negative predictive value 92%). EGD interval: every 1–2 years if no varices; every 2–3 weeks post-variceal bleed
Laboratory tests: Platelet count, INR, albumin, bilirubin, creatinine, sodium (MELD score)	Medicare Part B; routine chemistry panels	**At diagnosis; then at least annually** (more frequently if decompensated or on diuretics)	80053 (comprehensi ve metabolic panel) + 85025 (complete blood count)	Track synthetic liver function (albumin, INR, bilirubin); renal function (creatinine, sodium) for HRS risk; platelet count (<100k signals varices, splenomegaly). Use for MELD 3.0 calculation (includes sodium, bilirubin, creatinine, albumin)
Hepatitis B/C serology (if etiology unclear)	Medicare Part B; screening when etiology undetermined	Once **at diagnosis**; repeat if new HBsAg+ or anti-HCV+ result impacts treatment (DAA eligibility)	86705 (HBsAg), 86803 (anti-HCV)	Identify HBV (HBsAg+, HBeAg) and HCV (anti-HCV+ confirmed by HCV RNA) as cirrhosis etiology. HCV RNA quantitation (viral load) guides DAA initiation; HCV treatment may clear virus but cirrhosis persists and requires surveillance

TEST	COVERAGE	FREQUENCY	CPT CODE	CLINICAL NOTES
Frailty assessment: Liver Frailty Index (LFI) or Clinical Frailty Scale (CFS)	Medicare Part B; cognitive/ functional assessment covered	**At diagnosis; repeat annually**; more frequently if decompensation suspected	96160 (standardized screening instrument), 96161 (admin)	LFI (grip strength, chair stand time) and CFS predict mortality, decompensation, transplant candidacy independent of MELD. AASLD 2021 recommends LFI for all cirrhosis patients; CFS ≥5 signals need for palliative/ geriatric comanagement

ABBREVIATIONS: AFP = alpha-fetoprotein; AASLD = American Association for the Study of Liver Diseases; CFS = Clinical Frailty Scale; CPT = Current Procedural Terminology; DAA = direct-acting antiviral; EGD = esophagogastroduodenoscopy; HCC = hepatocellular carcinoma; HBsAg = hepatitis B surface antigen; HCV = hepatitis C virus; INR = international normalized ratio; LFI = Liver Frailty Index; LSM = liver stiffness measurement; MELD = Model for End-Stage Liver Disease

AAVBC PERSPECTIVE

Guidelines suggest performing/repeating FIB-4 scoring every 1 to 2 years for patients identified as ***high-risk*** *for liver cirrhosis.*

AAVBC supports proactive screening, with patients being routinely screened every 6 months to catch liver-related changes earlier*. This includes: patients with type 2 diabetes, pre-diabetes with additional metabolic risk factors, medically complicated obesity, known hepatic steatosis on imaging, excess alcohol consumption, chronic viral hepatitis (HBV/HCV), persistently abnormal liver enzymes, first-degree relatives of patients with MASLD-related cirrhosis, and patients with hyperferritinemia or other cardiometabolic risk factors.*

Subtle Early Signs in Older Adults (>65)

Many early signs of liver cirrhosis in older adults overlap with common age-related complaints (fatigue, cognitive slowing, easy bruising, and unexplained weight loss) which makes **pattern recognition** and a **low threshold** for **FIB-4 screening** essential when these nonspecific symptoms cluster in patients with metabolic, alcohol, or viral risk factors. More severe signs of liver disease are listed below such as ascites, varices, hepatic encephalopathy, etc.[1,2,9,10]

SIGN/SYMPTOM	CLINICAL SIGNIFICANCE
Ascites (fluid in peritoneal cavity; grade 1 mild/ detectable by ultrasound; grade 2 moderate/clinical; grade 3 tense/refractory)	**Hallmark of decompensated cirrhosis**; defines transition from compensated to decompensated disease. Ascites + infection (SBP) = medical emergency. Diuretic-refractory ascites (≥2 weeks on spironolactone 400 mg + furosemide 160 mg) requires evaluation for TIPS or paracentesis **See clinical pearl below**

SIGN/SYMPTOM	CLINICAL SIGNIFICANCE
Esophageal varices with hemorrhage (hematemesis, coffee-ground emesis, melena, hypotension)	**Most common cause of death in cirrhosis**; mortality 5–15% per bleed without treatment. Acute variceal bleed = ED emergency (IV access, octreotide 50 mcg bolus then 50 mcg/hr infusion, transfusion goal Hb 7–9 g/dL, urgent EGD for ligation/sclerotherapy). Prevention: nonselective beta-blockers (carvedilol preferred) if LSM ≥25 kPa or platelets <90k
Hepatic encephalopathy (HE): covert (cognitive slowing, mood changes); overt (confusion, asterixis, altered consciousness)	**Graded by West Haven:** Grade 1 (changes in sleep, mood, concentration); Grade 2 (lethargy, personality change); Grade 3 (somnolence, confusion); Grade 4 (coma). Covert HE (40.9% worldwide; 49% age ≥65) **often misdiagnosed as dementia.** Treatment: lactulose + rifaximin for secondary prevention; identify precipitants (infection, GI bleed, AKI, electrolytes, constipation) **See clinical pearl below**
Jaundice (yellowing of skin/sclera); dark urine; pale stools	**Indicates hyperbilirubinemia (>3 mg/dL)** from hepatocellular necrosis or cholestasis. Combined with ascites/HE = decompensated cirrhosis. Bilirubin component of MELD 3.0 and Child-Pugh C criteria
Variceal bleeding history; prior decompensation (ascites, HE, variceal bleed, jaundice)	Prior decompensating event signals high mortality risk and need for escalated surveillance/prophylaxis. **Median survival <1.5 years after first decompensating event**.Secondary prophylaxis (EVL, beta-blocker, SBP prophylaxis) mandatory after first event
Weight loss; muscle wasting; sarcopenia (unintentional weight loss >5% in 6 months or grip strength <26 kg in men, <16 kg in women)	**Muscle loss independently predicts decompensation and mortality beyond MELD**; driven by ammonia, inflammation, malnutrition. High-protein supplementation (1.2 g/kg/day) and exercise are VBC priorities. LFI grip strength <26 kg (men) or <16 kg (women) = frailty

ABBREVIATIONS: ED = emergency department; EGD = esophagogastroduodenoscopy; EVL = endoscopic variceal ligation; GI = gastrointestinal; HE = hepatic encephalopathy; MELD = Model for End-Stage Liver Disease; SBP = spontaneous bacterial peritonitis; TIPS = transjugular intrahepatic portosystemic shunt; VBC = value-based care

CLINICAL PEARL: POLYPHARMACY AND DEPRESCRIBING ARE NOT ADEQUATELY ADDRESSED IN PATIENTS WITH ASCITES

Polypharmacy and the necessity of "prescribing cascades" are often inadequately addressed by standard disease-specific guidelines, which rarely provide a clear "exit strategy" (deprescribing) for older adults. Deprescribing in patients with ascites is not just about reducing pill burden; it is a critical safety intervention to prevent Acute Kidney Injury (AKI), Spontaneous Bacterial Peritonitis (SBP), and lethal falls.

Elevated AKI/Fall Risk: Standard-of-care medications carry disproportionate hazards in the elderly. Diuretics (used for ascites) and Non-Selective Beta-Blockers (NSBBs) like Propranolol or Carvedilol significantly increase the risk of Acute Kidney Injury (AKI) through decreased renal perfusion and orthostatic falls due to blunted heart rate response. Other medications that need to be reduced are NSAIDS and benzodiazepines.[11]

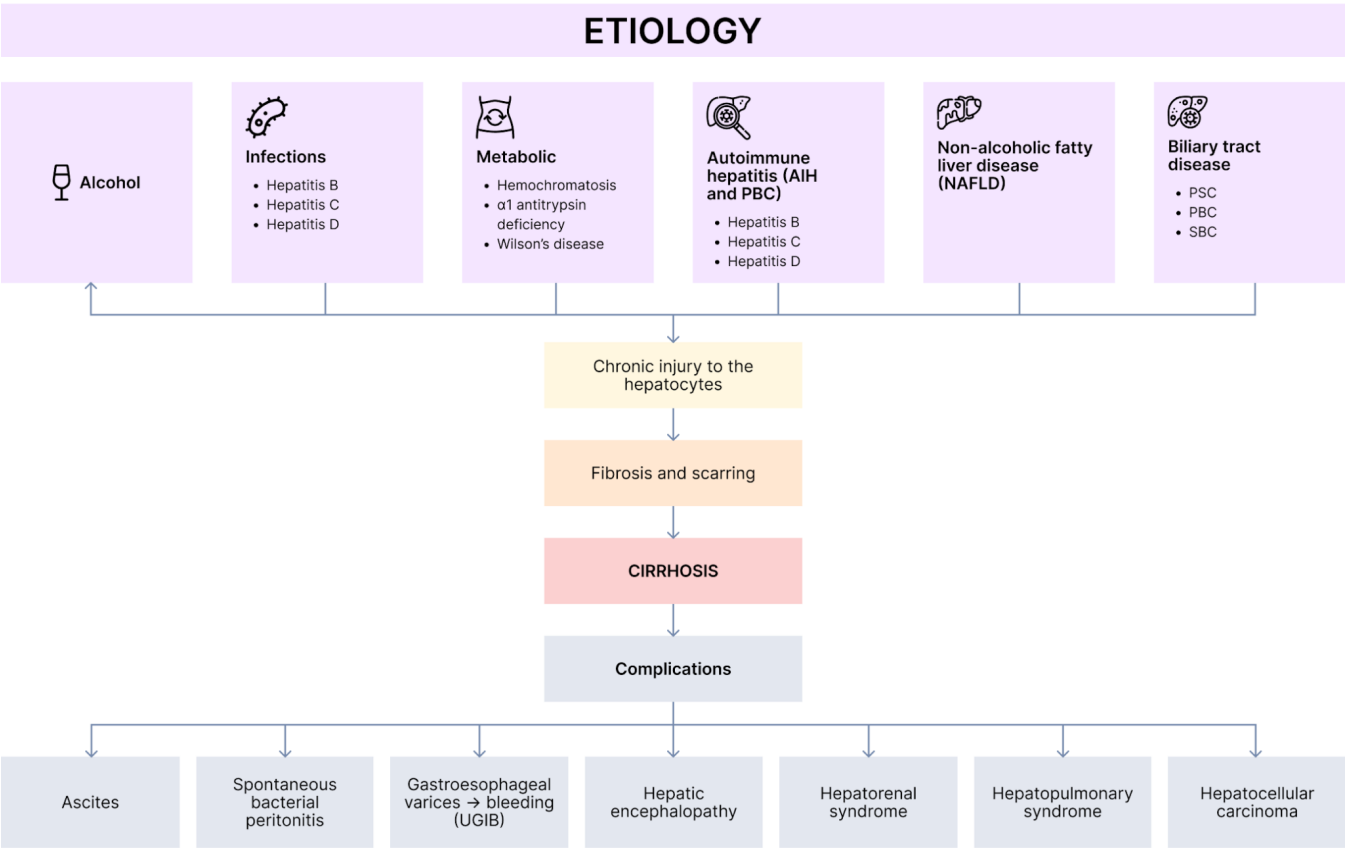
Risk Factors

Clinicians must recognize the specific metabolic, viral, and autoimmune drivers that accelerate the transition from stable liver disease to cirrhosis.[12–16]

FACTOR	RISK SIGNAL	NOTES
Type 2 Diabetes	**RR: 2.5 – 3.2**; **HR: 2.1** for cirrhosis-related hospitalization	T2DM is the strongest independent predictor of progression from MASLD to advanced fibrosis
Hepatic Steatosis (MASH)	**OR: 5.4** for advanced fibrosis; **Absolute Risk:** ~20% progress to cirrhosis over 10-15 years	Steatosis alone is common, but MASH (inflammation) drastically increases the hazard trajectory
Metabolic Syndrome	**HR: 1.8** for each additional component; **Relative Risk: ~4.0** for those with 4+ criteria	Synergistic effect: Obesity + HTN + Dyslipidemia creates a "pro-fibrotic" environment
Chronic Alcohol Use	**RR: 3.5** (for >30g/day); **Absolute Risk: 10–15%** lifetime risk for heavy drinkers	Alcohol serves as a "multiplicative" risk when combined with viral hepatitis or obesity.
Chronic Hepatitis	**Absolute Risk:** ~20% cirrhosis rate at 20 years post-infection	Modern DAAs (antivirals) can reduce this HR by >70% if treated before stage F3
Primary Biliary Cholangitis (PBC)	**HR:** 3.5 – 5.0 for cirrhosis if ALP remains >1.5x ULNA chronic autoimmune attack on small bile ducts	Treatment with UDCA can normalize this HR if started early
Primary Sclerosing Cholangitis (PSC)	**Absolute Risk: ~70% develop cirrhosis within 10–12 years**	A high-acuity condition involving scarring of both intra- and extrahepatic ducts; high risk for cholangiocarcinoma
Biliary Atresia/Chronic Obstruction	**RR: 8.0+** for rapid fibrosis progression	Mechanical obstruction causes "Pressure-Induced Fibrosis." In adults, this often stems from untreated choledocholithiasis
Gallstone Disease (Cholelithiasis)	**OR: 2.3** for secondary biliary cirrhosis (if chronic/untreated)	While common, only neglected, chronic obstructions (Secondary Biliary Cirrhosis) lead to actual liver failure
Elevated Alkaline Phosphatase (ALP)	In biliary disease, ALP is the primary "Hazard Marker" used to predict the speed of cirrhotic transformation	ALP is the primary "Hazard Marker" used to predict the speed of cirrhotic transformation.

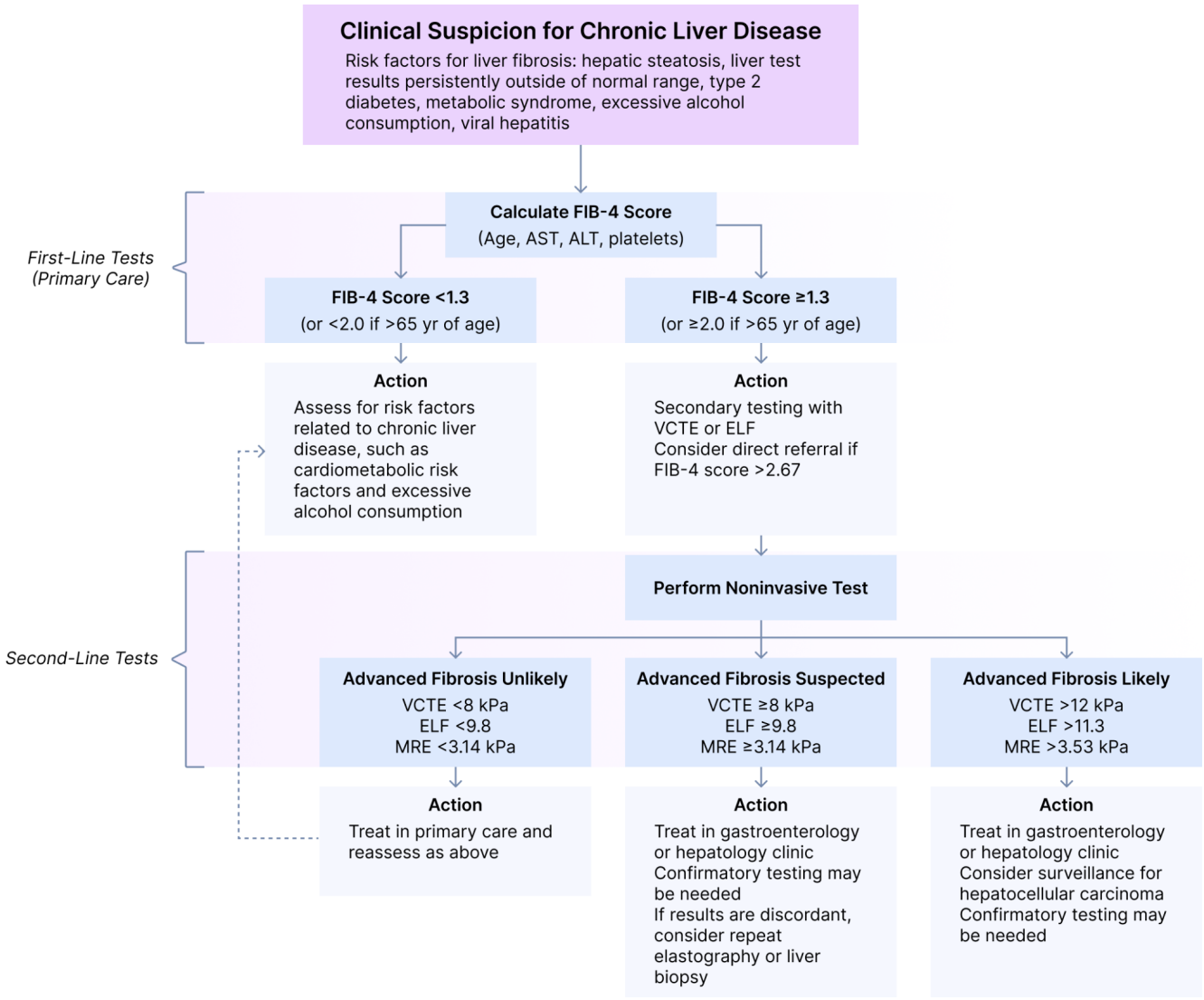
ABBREVIATIONS: ALP = alkaline phosphatase; DAA = direct-acting antiviral; HR = hazard ratio; HTN = hypertension; MASLD = metabolic dysfunction-associated steatotic liver disease; MASH = metabolic dysfunction-associated steatohepatitis; OR = odds ratio; PBC = primary biliary cholangitis; PSC = primary sclerosing cholangitis; RR = relative risk; T2DM = type 2 diabetes mellitus; UDCA = ursodeoxycholic acid; ULN = upper limit of normal

Etiology and Complications of Cirrhosis



ABBREVIATIONS: Non-alcoholic fatty liver disease (NAFLD), Primary Biliary Cholangitis (PBC), Primary Sclerosing Cholangitis (PSC) , Secondary Biliary Cirrhosis (SBC)

Screening/Diagnosis and Action Pathway



ABBREVIATIONS: AFP = alpha-fetoprotein; ALT = alanine aminotransferase; AST = aspartate aminotransferase; CLD = chronic liver disease; ELF = enhanced liver fibrosis; FIB-4 = fibrosis-4 index; kPa = kilopascals; MRE = magnetic resonance elastography; VCTE = vibration-controlled transient elastography; yr = year

Diagnostic Thresholds

The diagnosis and staging of liver cirrhosis rely on a "multimodal" approach that combines non-invasive biomarkers, imaging-based elastography, and laboratory scores.[6,17–19]

TEST/MARKER	DIAGNOSIS CRITERION	NOTES
FIB-4 Index (age × AST/ platelet count) — non-invasive fibrosis staging	FIB-4 **<1.30 = low risk** of cirrhosis (negative predictive value 95%) **≥1.30** → elastography or biopsy needed	Simple calculation; **no cost**. **FIB-4 ≥1.3 patients triggers MASLD workup per ADA 2026 standards.** Less specific for cirrhosis in older patients (age adjustments exist) but useful for risk stratification
Liver stiffness measurement (transient elastography, FibroScan)	**LSM ≥15 kPa = cirrhosis**; ≥25 kPa = 90% specific for esophageal varices; prognostic (increasing LSM = decompensation risk)	**Gold-standard noninvasive tes**t; no radiation, no contrast. Cannot perform if ascites present (technical failure). Operator-dependent; 10 measurements recommended for accuracy. Costly.
Laboratory synthetic function markers: albumin, INR, total/ direct bilirubin	**Albumin <3.5 g/dL** (protein synthesis loss) **INR >1.2** (coagulopathy) **bilirubin >2 mg/dL** = **hyperbilirubinemia**	Used in Child-Pugh score (A/B/C) and MELD 3.0. **Track over time**; acute worsening **signals decompensation or acute liver injury** (superimposed infection, alcohol relapse)
MELD 3.0 and Child-Pugh Score staging	MELD 3.0 calculation X= 10 × [0.33 ln(INR) + 0.5 ln(bili) + 0.25 ln(creatinine/2.5) – 2.88* (if male)*]; **MELD ≥15** = high-risk decompensation	**MELD 3.0 includes sex adjustment (female penalty 2.88, male 0) and sodium**; predicts short-term mortality. Child-Pugh C = albumin <2.5, INR >1.8, bilirubin >3, ascites/HE present; MELD ≥15 alternative threshold for K72.1x coding
Hepatitis B/C serology and viral load; FibroTest (if noninvasive staging needed)	HBsAg+ (chronic HBV); anti-HCV + with **detectable** HCV RNA = chronic HCV HCV RNA **<25 IU/mL** = SVR (sustained virologic response) post-DAA	HCV treatment with DAAs achieves >95% SVR; cirrhosis diagnosis persists despite viral clearance, thus surveillance and prophylaxis continue. HBV entecavir/tenofovir similarly required indefinitely

ABBREVIATIONS: AST = aspartate aminotransferase; DAA = direct-acting antiviral; HBsAg = hepatitis B surface antigen; HCV = hepatitis C virus; INR = international normalized ratio; IU = international units; MASLD = metabolic-associated steatotic liver disease; MELD = Model for End-Stage Liver Disease; RASS = Richmond Agitation-Sedation Scale; SVR = sustained virologic response; T2DM = type 2 diabetes mellitus

FIB-4

FIB-4 is the recommended first-line noninvasive test for screening liver fibrosis in primary care due to its simplicity, minimal cost, and strong rule-out performance, using only four routinely available variables: age, AST, ALT, and platelet count.[5,20,21]

$$\mathbf{FIB-4} = \frac{\text{Age (years)} \times \text{AST (U/L)}}{\text{Platelet Count } (10^9/\text{L}) \times \sqrt{\text{ALT (U/L)}}}$$

Key benefits of FIB-4 as a non-invasive screening tool:

- **Zero additional cost:** FIB-4 is calculated from labs already obtained in routine primary care (CBC and CMP); no additional blood draw, no proprietary assay, no imaging appointment required
- **High sensitivity for ruling out advanced fibrosis:** FIB-4 has dual cutoff limits. A low cutoff with >90% sensitivity to rule out advanced fibrosis, and a high cutoff with >90% specificity to rule it in
- **Endorsed across multiple societies:** The use of FIB-4 to screen for advanced fibrosis in primary care populations with liver disease risk factors is supported by several international liver, gastroenterology, and endocrine societies
- **Validated across etiologies:** Originally developed in HCV/HIV coinfection, FIB-4 has been extensively validated in MASLD (metabolic dysfunction-associated steatotic liver disease) and alcohol-related liver disease
- **Prognostic value:** High FIB-4 values are associated with all-cause and liver-related outcomes in population-based studies, and a change in FIB-4 category over time (low → intermediate → high) can track clinical progression

Core Populations for FIB-4 Testing:

- **Patients with Metabolic Risk Factors:** Anyone with Type 2 Diabetes (T2DM), obesity, or Metabolic Syndrome. These patients are at the highest risk for Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD, formerly NAFLD)
- **Patients with Known Steatosis:** If an ultrasound or CT scan incidentally shows a "fatty liver," a FIB-4 should be calculated immediately to determine if that fat has led to actual scarring
- **Patients with Elevated Liver Enzymes:** Any unexplained elevation in AST or ALT requires a FIB-4 to triage the patient for potential hepatology referral
- **Chronic Viral Hepatitis:** Patients with Hepatitis B or C are screened to assess the need for a liver biopsy or to monitor treatment efficacy

Common Oversights

Below are some common clinical oversights for clinicians to pay close attention to:[6,22]

OVERSIGHT/SHORTCUT	WHY IT MATTERS — WHAT TO DO INSTEAD
Misdiagnosing covert hepatic encephalopathy as dementia; missing up to 13% of treatable HE cases	**HE is potentially reversible** with lactulose/rifaximin; dementia is not. Use QuickStroop or SNAT-1; MRI differentiates globus pallidus hyperintensity (HE) from medial temporal atrophy (Alzheimer's)
Treating ascites or variceal risk without MELD 3.0 or Child-Pugh scoring and disease staging documentation	**Staging (MELD 3.0, Child-Pugh) guides prognosis and treatment intensity.** Diuretic dosing, NSBB target HR, and HCC surveillance intervals all depend on staging

OVERSIGHT/SHORTCUT	WHY IT MATTERS — WHAT TO DO INSTEAD
Avoiding Hepatocellular Carcinoma surveillance (ultrasound every 6 months) in compensated cirrhosis due to perceived 'low risk'	AASLD 2023: **all cirrhotic patients** (Child-Pugh A/B) **warrant 6-month US ± AFP surveillance**. Surveillance-detected hepatocellular carcinoma has better survival than symptomatic detection; skipping surveillance misses early-stage disease when curative therapies (resection, ablation) are available

ABBREVIATIONS: AASLD = American Association for the Study of Liver Diseases; HCC = hepatocellular carcinoma; HE = hepatic encephalopathy; MELD = Model for End-Stage Liver Disease; NAFLD = nonalcoholic fatty liver disease; NASH = nonalcoholic steatohepatitis; PPV = positive predictive value; RASS = Richmond Agitation-Sedation Scale; SNAT-1 = Stroop Number-Animal-Training Test

Key Differentials in Elderly

When managing liver disease in older adults, clinicians must carefully distinguish between symptoms of primary hepatic decompensation and age-related geriatric syndromes, as the clinical overlap can lead to significant diagnostic errors.[22,23]

PRESENTATION	DIFFERENTIAL	KEY TESTS
Ascites + fever + abdominal pain (acute presentation)	Spontaneous **bacterial peritonitis** (SBP); secondary bacterial peritonitis (perforated viscus); infected ascites from other source	**Diagnostic paracentesis** (CPT 49082/49083): PMN >250 cells/mm^3 = SBP; culture (+) SBP vs. (–) SBP; albumin ratio. Imaging (CT) to exclude perforation.[1] **SBP = medical emergency**; treatment: ceftriaxone 1 g daily + albumin 1.5 g/kg day 1, then 1 g/kg day 3
Hematemesis ± hypotension; melena (acute variceal bleed vs. other upper GI source)	**Esophageal varices** with hemorrhage (most common); gastric varices; portal hypertensive gastropathy; peptic ulcer disease; Mallory-Weiss tear	**ED stabilization** (IV access, type and cross, FFP/transfusion); urgent **EGD** (CPT 43235) within 12 hours. Octreotide infusion, blood products, **EVL** (CPT 43244) or sclerotherapy. Prior bleed = **permanent** secondary prophylaxis (NSBB + EVL or repeat EVL)
Acute mental status change in cirrhotic patient (HE vs. other encephalopathy vs. dementia decompensation)	**Hepatic encephalopathy** (Grade 1–4); subdural hematoma (fall risk); infection (SBP, UTI, pneumonia); hypoglycemia; hyperammonemia	**Ammonia level** (if available); mental status exam; cognitive testing (QuickStroop); head CT if trauma concern; paracentesis if ascites + fever; urine/blood cultures; glucose check. **MRI** (globus pallidus signal)

PRESENTATION	DIFFERENTIAL	KEY TESTS
Renal dysfunction (Cr rise, oliguria) in decompensated cirrhosis (HRS vs. AKI vs. CKD)	**Hepatorenal syndrome** (acute, poor prognosis); acute kidney injury (infection, NSAIDs, diuretics); chronic kidney disease (diabetes, hypertension); prerenal (volume depletion)	**Urine sodium, osmolality; urine dipstick; ultrasound** (kidney size, Doppler). **HRS** criteria: Cr rise >0.3 mg/dL in 48 h or >1.5× baseline in ≤2 weeks; oliguria; absence of improvement with fluid bolus. Prognosis poor (<50% survival without treatment); terlipressin + albumin if HRS Type 1
Jaundice (new or worsening) with/without encephalopathy (decompensation vs. acute hepatitis vs. biliary obstruction)	**Acute decompensation** (infection, alcohol relapse, medication); acute viral/autoimmune hepatitis (ALT/AST >1000); extrahepatic cholestasis (stone, malignancy); medication hepatotoxicity	**LFTs** (AST, ALT, ALP, GGT, bilirubin); **INR**; **ultrasound abdomen** (ductal dilation, stone); **autoimmune markers** (ANA, anti-smooth muscle); **medication review.** If bilirubin >3 + INR >1.8 + ascites/HE = Child-Pugh C

ABBREVIATIONS: ALT = alanine aminotransferase; ALP = alkaline phosphatase; ANA = antinuclear antibody; AST = aspartate aminotransferase; CPT = Current Procedural Terminology; Cr = creatinine; CT = computed tomography; ED = emergency department; EGD = esophagogastroduodenoscopy; EGD = esophagogastroduodenoscopy; EVL = endoscopic variceal ligation; FFP = fresh frozen plasma; GGT = gamma-glutamyl transferase; HE = hepatic encephalopathy; HRS = hepatorenal syndrome; LFT = liver function test; NSAID = nonsteroidal anti-inflammatory drug; PMN = polymorphonuclear leukocyte; SBP = spontaneous bacterial peritonitis; UTI = urinary tract infection
***Consult FDA labels for the most up-to-date dosage information, contraindications, and drug-drug interactions**

Comorbidity Screening

Beyond direct liver management, managing cirrhosis requires a multi-organ approach to identify the extrahepatic conditions that may drive decompensation.[7,24,25]

CONDITION	PREVALENCE/ASSOCIATION	SCREENING APPROACH
Hepatic encephalopathy	HE is the most under-recognized complication in elderly. Driven by hyperammonemia, inflammation, neurotoxins. **Covert 40.9% worldwide; overt 15–20%); HE + dementia coexistence up to 13%**	**Screen with validated cognitive tools** (QuickStroop, SNAT-1, MoCA) annually and at each encounter if cognitive complaints. Document West Haven grade (1–4) if present. MRI: globus pallidus hyperintensity (HE-specific); consider both HE + dementia if coexistent
Esophageal varices	**Portal hypertension** (varices form when hepatic venous pressure gradient ≥12 mmHg) is the **complication most directly preventable with prophylaxis** (NSBB, EVL). Varices correlate with LSM ≥25 kPa, platelet <90k, or prior bleed **40–50% of compensated cirrhosis; bleeding risk 5–15% per year if untreated**	**EGD at diagnosis** (CPT 43235); Baveno VI/VII criteria guide interval (if platelets >150k AND LSM <20 kPa, varices unlikely—avoid repeat EGD) **Primary prophylaxis with carvedilol 6.25 mg daily** (titrate to HR 55–60 bpm) if varices present or detected by imaging. Secondary prophylaxis: EVL (CPT 43244) after first bleed + long-term NSBB

CONDITION	PREVALENCE/ASSOCIATION	SCREENING APPROACH
Spontaneous bacterial peritonitis (SBP)	SBP is one of the most lethal complications; defined by **PMN >250 cells/mm^3** in ascitic fluid without secondary peritonitis. Renal failure during SBP treatment predicts death. Secondary prophylaxis mandatory after the first episode **13% prevalence in cirrhosis with ascites; mortality 30% even with treatment**	**Paracentesis** (CPT 49082/49083) diagnostic: PMN count, culture, albumin. If SBP documented: ceftriaxone 1 g daily × 7 days; albumin 1.5 g/kg day 1, 1 g/kg day 3. **Secondary prophylaxis:** ciprofloxacin 500 mg daily or SMX-TMP double-strength daily (indefinitely)
Hepatorenal syndrome (HRS)	HRS is **renal vasoconstriction secondary to portal hypertension + splanchnic vasodilation**; triggered by infection, hemorrhage, diuretics, NSAIDs. Defined by **acute Cr rise** (>0.3 mg/dL in 48 h or >1.5× baseline); oliguria. Type 1 (fulminant, poor prognosis); Type 2 (gradual, refractory ascites) **8–15% of decompensated cirrhosis; mortality >50% without terlipressin + albumin**	Monitor Cr, sodium, albumin at each visit (especially if ascites/diuretics). Volume expansion trial (albumin bolus) required before HRS diagnosis. If confirmed: terlipressin 0.4 mg IV bolus then 0.2 mg q4–6h (expensive; FDA 2022) + albumin 50 g daily. Nephrology comanagement; consider dialysis or TIPS
Hepatocellular carcinoma	Hepatocellular carcinoma is the **most common liver malignancy in cirrhosis**; **risk driven by etiology (HCV >HBV>NASH)**, inflammation, and cirrhosis duration. Surveillance-detected HCC has better prognosis (5-year OS 30–50%) vs. symptomatic (5-year OS <10%) **5-year cumulative incidence 5–15% in compensated; up to 30% in decompensated or cirrhosis >10 years**	**Semiannual ultrasound** (CPT 76700) + AFP (CPT 82105) per AASLD 2023.[2] Diagnostic imaging (CT/MRI) for nodules >1 cm. BCLC staging guides treatment. **Do NOT surveil if Child-Pugh C** unless transplant candidate (NCCN 2026). Refer to hepatology/oncology if HCC detected

ABBREVIATIONS: BCLC = Barcelona Clinic Liver Cancer; CPT = Current Procedural Terminology; CT = computed tomography; EGD = esophagogastroduodenoscopy; EVL = endoscopic variceal ligation; HCC = hepatocellular carcinoma; HE = hepatic encephalopathy; HRS = hepatorenal syndrome; IV = intravenous; MELD = Model for End-Stage Liver Disease; MRI = magnetic resonance imaging; NCCN = National Comprehensive Cancer Network; NSBB = nonselective beta-blocker; PMN = polymorphonuclear leukocyte; SBP = spontaneous bacterial peritonitis; SMX-TMP = sulfamethoxazole-trimethoprim
***Consult FDA labels for the most up-to-date dosage information, contraindications, and drug-drug interactions**

Staging/Severity Matrix

Child-Pugh Score (A/B/C)[26,27]

STAGING SYSTEM	CRITERIA/VALUES | ESTIMATED OUTCOMES	CLINICAL USE	DOCUMENTATION CUE
Child-Pugh Score (A/B/C)	Albumin, INR, bilirubin, ascites, HE grade → score 5–15 **A: 5–6** (compensated, low-risk) **B: 7–9** (intermediate decompensation risk) **C: ≥10** (high-risk) Short median survival, **<1.5 years** post-decompensation	**Predicts short-term mortality:** **A: 100% 1-yr survival** **B: 80%;** **C: 45%** Determines surveillance intensity and treatment eligibility (e.g. CTP C excluded from HCC surveillance unless transplant candidate)	Document each component: albumin (g/dL), INR, bilirubin (mg/dL), ascites grade (0–3), HE grade (West Haven 0–4) Calculate total score; state CTP class (A/B/C) and 1-year survival estimate

Benefits of Child-Pugh Score (A/B/C)

- **Simple bedside calculation**: Requires only three lab values (bilirubin, albumin, INR) and two clinical assessments (ascites, encephalopathy); no calculator needed
- **Strong long-term prognostic value**: 1-year survival rates of **100% (Class A)**, **80% (Class B)**, and **45% (Class C)**; 5-year survival ranges from **75% (score 5)** to **20% (score >12)**. A multicenter study of 1,047 patients found CTP had better or equivalent prognostic value compared to MELD for both short- and long-term mortality in stable cirrhosis (C-index 0.672)
- **Predicts surgical risk**: Class A patients can undergo surgical procedures with low mortality risk; originally developed for this purpose and remains the standard for perioperative risk assessment

Drawbacks of Child-Pugh Score (A/B/C)

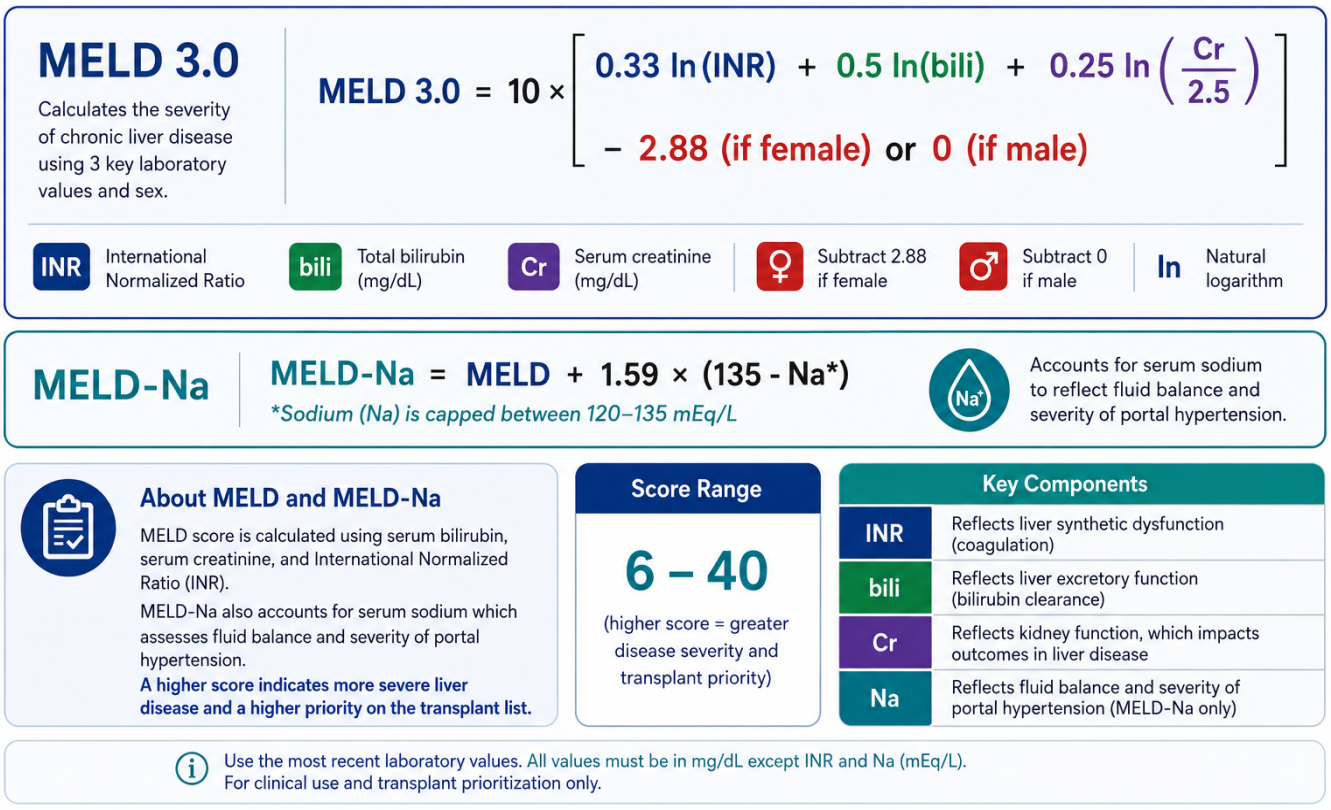
- **Omits important prognostic variables:** Does not incorporate creatinine (renal function), sodium (hyponatremia), portal pressure, spontaneous bacterial peritonitis, hepatorenal syndrome, or nutritional status (all of which independently predict mortality

MELD 3.0 Scoring System

STAGING SYSTEM	CRITERIA/VALUES	CLINICAL USE	DOCUMENTATION CUE
MELD 3.0 Score (2023 update with sex adjustment, sodium)[28]	**MELD 3.0** = 10 × [0.33 ln(INR) + 0.5 ln(bili) + 0.25 ln(Cr/2.5) − 2.88 (if female) or 0 (if male)]. Range 0–40; ≥15 = high-risk	Predicts short-term (90-day, 1-year) mortality; used for transplant waitlist prioritization. Sex adjustment and sodium (lower Na = higher risk) improve predictive accuracy vs. original MELD	State recent INR, bilirubin, creatinine, sodium; calculate MELD 3.0 score; document trend (stable/improving/worsening). If MELD ≥15, document consideration of specialist referral and transplant candidacy discussion

MELD Score and MELD NA[28]

Benefits of MELD 3.0 Score



- **Widely validated across clinical settings:** Beyond transplant allocation, MELD accurately predicts mortality in variceal bleeding, hepatorenal syndrome, alcoholic hepatitis, acute liver failure, and perioperative risk for non-transplant surgery in cirrhotic patients

Drawbacks of MELD 3.0 Score

- The standard AASLD guidelines prioritizes MELD scoring for late-stage surgical interventions/ transplantation decisions; however, MELD scores do not show the full clinical picture. **Many**

geriatric patients present with low MELD scores but are functionally disabled. Frailty, muscle loss (sarcopenia), and nutritional status are all strong independent predictors of post-transplant survival

MELD Score Range

MELD-NA SCORE RANGE	SEVERITY TIER	CLINICAL STATUS	PRACTICAL CLINICAL TIMELINE AND ACTION	PRIMARY MANAGEMENT AND TRANSPLANT PRIORITY
<10	**Low Severity**	Stable hepatic baseline; low short-term mortality risk	**Routine outpatient follow-up** (Every 6–12 months)	Routine outpatient monitoring, chronic disease management, and standard surveillance
11 to 18	**Moderate Severity**	Intermediate disease status; requires closer tracking for early signs of decompensation	**Increased clinical vigilance** (Evaluate every 1–3 months as score rises)	Optimized medical management, aggressive complication prophylaxis, and consideration for initial transplant evaluation as scores approach the higher end of the tier
19 to 24	**High Severity**	Significant disease progression; high risk for acute clinical decompensation events	**Active Specialist Co-management** (Weekly to monthly lab/clinical titration)	Active specialist co-management, close lab titration frequencies, and **active listing/evaluation for liver transplantation**
25 to 40	**Very High Severity**	Critical hepatic failure; exceptionally high short-term mortality risk without intervention	**Inpatient Triage/ Intensive Care** (Continuous monitoring)	**Maximum Transplant Priority;** patients in this range are prioritized at the top of waitlists for organ allocation as compatible grafts become available

Liver Stiffness Measurement[29,30]

STAGING SYSTEM	CRITERIA/VALUES	CLINICAL USE	DOCUMENTATION CUE
Liver Stiffness Measurement (LSM) by transient elastography; Fibroscan etc.	**LSM:** <10kPa=no-cirrhosis 10–15kPa= probable cirrhosis ≥15kPa = cirrhosis. ≥25 kPa = 90% specific for esophageal varices Increasing LSM over time= decompensation risk	Serial LSM tracks disease progression; rising stiffness predicts variceal hemorrhage and decompensation. Guides EGD interval (Baveno VI/VII criteria)	Record LSM value (kPa) and date; comment on trend vs. prior (stable/increasing/decreasing). If ≥25 kPa, note initiation or continuation of NSBB prophylaxis and planned EGD interval per Baveno VI/VII

STAGING SYSTEM	CRITERIA/VALUES	CLINICAL USE	DOCUMENTATION CUE

ABBREVIATIONS: EGD = esophagogastroduodenoscopy; kPa = kilopascals; LSM = liver stiffness measurement; NSBB = nonselective beta-blocker

Benefits of LSM

- **Non-Invasive "Virtual Biopsy":** LSM eliminates the risks associated with percutaneous liver biopsy, such as pain, internal bleeding (hemorrhage), and gallbladder puncture
- **High Negative Predictive Value (NPV):** A low LSM score (typically <8 kPa for MASLD) is **exceptionally reliable for ruling out advanced fibrosis.** This allows clinicians to keep low-risk patients in primary care and avoid unnecessary specialty referrals

Drawbacks of LSM

- **"Stiffness" is Not Always "Fibrosis":** LSM measures the physical velocity of a shear wave. Several factors other than scar tissue can artificially increase liver stiffness:
- **Acute Inflammation:** A recent flare in transaminases (ALT/AST) will "stiffen" the liver and cause a false-positive high score
- **Postprandial State:** Stiffness increases significantly after eating. Patients must fast for at least 3 hours (ideally 6) before the test
- **Congestion:** Right-sided heart failure or fluid overload can lead to a "stiff" liver due to blood backup, even in the absence of fibrosis
- **Biliary Obstruction:** Acute cholestasis (blocked bile ducts) increases internal pressure and stiffness

Liver Frailty Index and Clinical Frailty Scale (CFS)[31,32]

STAGING SYSTEM	CRITERIA/VALUES	CLINICAL USE	DOCUMENTATION CUE
Liver Frailty Index (LFI) and Clinical Frailty Scale (CFS) — geriatric-specific severity	**LFI:** grip strength + chair stand time → score ≥4.27 = frail (independent of MELD) **CFS**: **1–3** robust, **4–5** vulnerable/mildly frail **6–7** moderately/severely frail **8–9** very severe	LFI and CFS independently predict mortality, decompensation, transplant candidacy, and ED utilization beyond MELD score. AASLD 2021 recommends LFI for all cirrhosis patients	Measure and document grip strength (kg) and chair stand time (seconds) at baseline and annually. Calculate LFI score. Also document CFS (1–9 scale). If frail (LFI ≥4.27 or CFS ≥5), note referral to geriatrics, palliative care, and nutrition

ABBREVIATIONS: BCLC = Barcelona Clinic Liver Cancer; CPT = Child-Turcotte-Pugh; Cr = creatinine; CTP = Child-Turcotte-Pugh; EGD = esophagogastroduodenoscopy; EVL = endoscopic variceal ligation; HCC = hepatocellular carcinoma; HE = hepatic encephalopathy; INR = international normalized ratio; LFI = Liver Frailty Index; LSM = liver stiffness measurement; MELD = Model for End-Stage Liver Disease; NSBB = nonselective beta-blocker; OS = overall survival; TACE = transarterial chemoembolization; VBC = value-based care

Benefits of Liver Frailty Index (LFI)

- **Disease-Specific Validation:** Unlike general tools, the LFI was built specifically to predict waitlist mortality and post-transplant outcomes in liver patients
- **Sensitivity to "Prehab":** Because it is a continuous scale, it can track small physical improvements when a patient starts a high-protein diet or a "pre-habilitation" exercise program
- **Superior Prediction:** It often identifies patients at high risk of death who may have a deceptively "low" MELD score

Drawbacks of LFI

- **Exclusion Bias:** It cannot be performed on patients with severe hepatic encephalopathy, significant ascites (which blocks chair stands), or physical disabilities (like severe arthritis)
- **Snapshots Only:** A patient might perform poorly due to a temporary infection (like SBP), which may not reflect their true "baseline" frailty

Benefits of CFS

- Speed and Simplicity: Can be completed in under 60 seconds during a standard physical exam without any equipment
- Holistic View: It captures the "whole person," including cognitive status and activities of daily living (ADLs), which the physical LFI might miss
- Predictive of Hospitalization: High CFS scores (>5) are strongly correlated with unplanned hospitalizations and the need for post-acute care facilities after a transplant

Drawbacks of CFS

- Lack of Liver Specificity: It was not built for liver disease. It may not be as precise as the LFI in predicting specific transplant waitlist outcomes

⚠ **RED FLAG – URGENT (SAME-DAY/ED EVALUATION)**

- **Acute variceal hemorrhage** (hematemesis, melena, hypotension, hemoglobin drop) acute SBP with fever + abdominal pain + altered mental status (sepsis)
- **Acute hepatic encephalopathy Grade 3–4** (unresponsive, risk of aspiration)
- **Acute hepatorenal syndrome** (oliguria, Cr rise >0.3 mg/dL in 48 h, BP drop)
- **Signs of acute liver failure** (jaundice + coagulopathy + encephalopathy in acute decompensation)

RED FLAG – RAPID (24–48 HOURS)

- New or worsening ascites despite diuretics (refractory ascites → TIPS evaluation)
- Grade 2 hepatic encephalopathy with cognitive decline
- AKI (Cr rise without SBP/HRS features)
- clinical suspicion of HCC on imaging or rising AFP (>400 ng/mL or doubling in <4 weeks)
- new-onset jaundice without fever
- medication toxicity (NSAID-induced AKI, benzodiazepine-induced HE)

3 MEAT DOCUMENTATION ESSENTIALS

Male patient 72 with 2-year history of NASH cirrhosis (**K74.69**) complicated by mild ascites (grade 1) and recent cognitive decline attributed to 'early dementia'. On exam: trace ascites, mild asterixis, slow speech. Recent labs: albumin 3.1, INR 1.4, bilirubin 1.8, creatinine 1.1, sodium 134. MELD 3.0 = 14 (high-risk). LSM 22 kPa. Prior EGD: small esophageal varices.

MONITOR: "NASH Cirrhosis (**K74.69**) with portal hypertension. Grade 1 Ascites (**R18.8**) present (trace on exam). Cognitive status: slow speech, inverted sleep-wake cycle, and mild asterixis noted today—suggestive of **Covert Hepatic Encephalopathy (K72.90)**. Synthetic function: Albumin 3.1, INR 1.4, Bilirubin 1.8. Renal/Electrolytes: Sodium 134, Creatinine 1.1. Adherence to diuretics confirmed; currently on Furosemide 20mg/Spironolactone 50mg daily. No GI bleeding or abdominal pain reported."

EVALUATE: "**MELD 3.0 Score: 14** (High-risk). **LSM 22 kPa** (Stiffness indicates F4/Cirrhosis). Prior EGD: small esophageal varices (non-bleeding). **West Haven Criteria:** Grade 1 HE based on clinical asterixis and psychomotor slowing. Labs: Sodium 134 indicates mild hypervolemic hyponatremia. AFP/US: No recent HCC surveillance on file; last RUQ US >12 months ago (non-compliant). **Child-Pugh Class B** (Score 8: Albumin 2, INR 2, Bili 2, Ascites 1, HE 1)."

ASSESS: "Cirrhosis with Hepatic Encephalopathy (**K74.69 + K72.90**) and Ascites (**R18.8**). **Intermediate-High Risk** — MELD 14 + low Sodium. Cognitive decline is assessed as **Covert HE**, not dementia, given the presence of asterixis and low Albumin/Sodium. This is a treatable, metabolic complication. Risk of variceal hemorrhage is present but currently stabilized (small varices). Lack of HCC surveillance represents a critical diagnostic gap."

TREAT: "**Initiate Lactulose** 30 mL BID; titrate to 2–3 soft stools daily to reduce ammonia absorption. **Initiate Rifaximin** 550mg BID for secondary HE prevention. Continue current diuretics; maintain 2g Sodium-restricted diet. **STAT Orders:** RUQ Ultrasound with Doppler + Alpha-fetoprotein (AFP) for **HCC Surveillance**. Refer to **Transplant Hepatology** (MELD >10) for evaluation of decompensation and TIPS candidacy. Refer to GI for repeat EGD (surveillance). Home monitoring: daily weights, mental status log by spouse. Follow-up 4 weeks: check MELD, reassess HE/asterixis. MEAT documented."

Clinical Documentation Elements

- **Etiology and ICD-10 Code:** Document cirrhosis etiology (NASH, HBV, HCV, alcohol, PBC, other) with diagnostic basis (imaging, serology, biopsy if done). Use specific ICD-10 code (**K74.69,**

K70.30/31, K74.3/4/5, not K74.60) with supporting comorbidity codes (**E11.x, E66.x, B18.x, F10.x** as applicable)

- **Severity Staging:** MELD 3.0 score with recent INR, bilirubin, creatinine, sodium; Child-Pugh class (A/B/C) with component scores; CFS or LFI score; LSM if available. State compensation status (compensated vs. decompensated) with date of last decompensating event if applicable
- **Complications and Surveillance:** Ascites grade (0–3) with last paracentesis date if performed; HE grade (West Haven 0–4); variceal status (screening date, presence Y/N, prior bleed Y/N); HCC surveillance schedule (last US/AFP date, next scheduled); other complications (SBP history, HRS, CKD stage)
- **Medications and Prophylaxis:** Current list with doses; note NSBB (carvedilol dose/target HR), diuretics (spironolactone + furosemide doses), lactulose/rifaximin if on board, SBP prophylaxis agent. Document high-risk meds to avoid or deprescribe; note alternatives chosen (e.g., acetaminophen instead of NSAID)
- **VBC Metrics and Care Coordination:** Weight, BMI; albumin/INR trend; CFS/LFI score; cognitive screening result (QuickStroop/MoCA if done); frailty category; referrals placed (gastroenterology, hepatology, palliative care, nutrition, PT/OT); case management involvement; palliative care discussion date and patient/family response. Annual HCC surveillance documentation and results

Reframing Common Documentation Shortcuts

INSTEAD OF...	DOCUMENT...
'Cirrhosis' (no etiology specified, coded as K74.60)	**K74.69 'NASH cirrhosis**, documented by transient elastography LSM 18 kPa, imaging showing fatty infiltration, and metabolic risk factors (diabetes E11.21, obesity **E66.01**, hypertension I10)' **OR K70.30 'Alcohol-induced cirrhosis**, history of heavy alcohol use (documented quantity per AUDIT-C score), imaging findings' **OR K74.x with specific etiology** + supporting diagnostic basis
'Confusion' or 'cognitive decline' (no HE assessment performed)	'**Mental status exam:** oriented to person/place/time; no asterixis; QuickStroop score XX [normal/abnormal]; West Haven HE Grade 0. Differential includes covert HE vs. dementia. Next: cognitive testing if decline progresses or QuickStroop abnormal.' **OR if HE present:** 'West Haven Grade 2 HE: lethargy + mild confusion + asterixis; started lactulose + rifaximin 550 mg TID for secondary prevention'
'Ascites controlled' (no grade documented, diuretic doses unclear)	'Ascites Grade 2 (moderate, clinical evidence); **current regimen:** spironolactone 200 mg + furosemide 80 mg daily; diuretics titrated weekly; weight trend [stable/decreased by X lbs in Y weeks]; urine sodium checked; renal function stable (Cr 1.0, Na 137).' **If refractory:** 'Refractory ascites (no weight loss despite max diuretics × 2+ weeks); refer to GI/hepatology for TIPS evaluation or repeated paracentesis'

INSTEAD OF...	DOCUMENT...
'No varices' (no EGD date documented; variceal bleed risk unclear)	'**EGD performed [date]:** small esophageal varices noted, no current bleeding. Started carvedilol 6.25 mg daily for primary prophylaxis; target HR 55–60 bpm; follow-up EGD planned per Baveno VI/VII criteria [dates]. **If prior bleed: '**History of variceal hemorrhage [date]; on long-term carvedilol + EVL ligation [date]. Plan repeat EVL per endoscopy interval; secondary prophylaxis in place'
'No Hepatic Cancer' (no surveillance performed in 18+ months)	'Hepatocellular carcinoma Surveillance Plan: ultrasound + AFP per AASLD 2023 (semiannually). Last surveillance ultrasound [date], AFP [value, date], both normal. Next scheduled [date]. **Compensated cirrhosis (CTP A, MELD 11) warrants continued 6-month surveillance.** If no recent imaging: 'Due for HCC surveillance: order ultrasound + AFP; patient counseled on 6-month interval importance for early detection'

ABBREVIATIONS: AUDIT-C = Alcohol Use Disorders Identification Test—Concise; CTP = Child-Turcotte-Pugh; eGFR = estimated glomerular filtration rate; EVL = endoscopic variceal ligation; GI = gastrointestinal; HE = hepatic encephalopathy; HCC = hepatocellular carcinoma; INR = international normalized ratio; LSM = liver stiffness measurement; MELD = Model for End-Stage Liver Disease; NSBB = nonselective beta-blocker; TIPS = transjugular intrahepatic portosystemic shunt; VBC = value-based care. ***Consult FDA labels for the most up-to-date dosage information, contraindications, and drug-drug interactions**

DOCUMENTATION IS THE FOUNDATION OF CIRRHOSIS MANAGEMENT IN VBC: WHAT EVERY NOTE SHOULD INCLUDE

(1) **Etiology** with specific ICD-10 code (not **K74.60**)
(2) **Severity staging** (MELD 3.0, Child-Pugh class, CFS/LFI)
(3) **Complication assessment** (ascites grade, HE grade, varices status, HCC surveillance dates);
(4) **Medication reconciliation** (NSBB dose/target, diuretics, lactulose, SBP prophylaxis, high-risk meds to avoid)
(5) **Frailty and cognitive screening** results (LFI/CFS, QuickStroop/MoCA)
(6) **Advance care planning** discussion (documented if MELD ≥15, CTP C, or recurrent decompensation)
(7) **Specialist coordination** (referrals placed and status). This documentation directly supports HCC mapping, decompensation prediction, quality metric reporting, and appropriate resource allocation in VBC populations

4 TREATMENT AND REFERRAL QUICK GUIDE

Therapy Escalation Criteria

Therapy escalation in liver cirrhosis follows a stage-specific framework driven by the transition from compensated to decompensated disease, with each complication having its own stepwise escalation pathway. The critical inflection point is the development of clinically significant portal hypertension (CSPH, HVPG ≥10 mmHg), after which **median survival drops** from **>12 years to <1.5 years** once decompensation occurs.[6,26,28,31]

TRIGGER	ACTION
MELD ≥15 or Child-Pugh B/C; clinical decompensation (new ascites, HE, variceal bleed, jaundice, renal dysfunction)	**Refer to hepatology/gastroenterology urgently (within 1 week)** for disease staging confirmation, transplant candidacy evaluation, and intensive management. Coordinate with case management, social work, palliative care
Refractory ascites (no weight loss despite spironolactone 400 mg + furosemide 160 mg × 2+ weeks) or tense ascites with respiratory compromise	**Refer to interventional radiology for TIPS evaluation** or repeat large-volume paracentesis. Discuss fluid and sodium restriction more rigorously; consider albumin infusions post-paracentesis
Suspected or confirmed hepatorenal syndrome (acute Cr rise >0.3 mg/dL in 48 h or >1.5× baseline with oliguria despite volume expansion)	**ED transfer urgently.** Initiate terlipressin 0.4 mg IV bolus then 0.2 mg IV q4–6h (expensive; FDA 2022 approval) + albumin 50 g daily. Nephrology comanagement. MELD ≥15 likely; discuss transplant urgency
Hepatocellular Carcinoma detected on imaging (nodule >1 cm) or AFP rising >20 ng/mL/month	**Refer to hepatology/oncology + transplant surgery urgently.** Staging (BCLC stage). BCLC 0/A: curative intent (resection, ablation, transplant if eligible). BCLC B: TACE. BCLC C/D: sorafenib ± atezolizumab or supportive care
CFS ≥5 (moderate–severe frailty) or LFI ≥4.27 (frail) with repeated hospitalizations or acute decompensation	**Refer to geriatrics + palliative medicine + nutrition + PT/OT + social work + case management.** Discuss goals of care, advance directives, hospice eligibility. Intensify home-based support, medication reconciliation, symptom management

ABBREVIATIONS: CPT = Current Procedural Terminology; CFS = Clinical Frailty Scale; ED = emergency department; HCC = hepatocellular carcinoma; IR = interventional radiology; LFI = Liver Frailty Index; MELD = Model for End-Stage Liver Disease; PT/OT = physical/occupational therapy; TIPS = transjugular intrahepatic portosystemic shunt; TACE = transarterial chemoembolization
***Consult FDA labels for the most up-to-date dosage information, contraindications, and drug-drug interactions**

AASLD/ACG/AAFP Aligned Recommendations

CATEGORY	RECOMMENDED AGENT/ APPROACH[22,33–35]	NOTES[22,33–35]
Variceal Prophylaxis (NSBB First-Line) — Paradigm Shift 2024: Early NSBB Initiation When CSPH Identified	**carvedilol** 6.25 mg daily (preferred), titrate to target HR 55–60 bpm; propranolol 20–40 mg BID; nadolol 40 mg daily. Initiate when LSM ≥25 kPa, platelets <90k, or varices detected at EGD. **Continue indefinitely post-bleed**	AASLD 2024 portal hypertension guidance now recommends early NSBB when clinically significant portal hypertension (CSPH) identified, even before varices develop (paradigm shift from prior reactive approach). **carvedilol shows superior outcomes vs. propranolol** (lower rebleeding rate); preferred agent. Monitor HR and BP; avoid if SBP <90, HR <55, or severe asthma/COPD

CATEGORY	RECOMMENDED AGENT/ APPROACH[22,33–35]	NOTES[22,33–35]
Ascites Management: Diuretics + Sodium Restriction	**spironolactone** 100 mg daily (max 400 mg) + furosemide 40 mg daily (max 160 mg); sodium <2 g/day (goal weight loss 0.5 kg/day if ascites, 1 kg/day if ascites + edema)	AASLD 2021 guidance: first-line diuretics (spironolactone preferred over furosemide monotherapy). **Monitor renal function, potassium, and sodium closely** (risk of hyponatremia, hyperkalemia, AKI).Refractory ascites (no weight loss ≥2 weeks at max doses) → TIPS or repeated paracentesis
Hepatic Encephalopathy Prevention and Treatment	**lactulose** (titrate to 2–3 soft bowel movements/day; start 15–30 mL BID); rifaximin 550 mg TID (nonabsorbed antibiotic; reduces ammonia-producing bacteria). Identify and treat precipitants (infection, GI bleed, hypokalemia, dehydration, AKI, medications)	ACG 2026 HE guideline: l**actulose + rifaximin combination for secondary prevention** (superior to either agent alone). O**pioids, benzodiazepines, NSAIDs worsen HE and should be avoided.** Early recognition of covert HE (QuickStroop, MoCA) enables treatment before overt encephalopathy develops
SBP Prophylaxis (After First Episode or Albumin <1.5 g/dL)	**ciprofloxacin** 500 mg daily OR sulfamethoxazole-trimethoprim (double-strength) daily; continue indefinitely or until liver transplant/ disease reversal	AASLD 2021 guidance: **secondary prophylaxis mandatory after first SBP episode** (reduces recurrence from 69% to 26% at 1 year). Also consider prophylaxis if albumin <1.5 g/dL even without prior SBP (reduces spontaneous SBP risk)
HCC Surveillance and Early Detection	**Ultrasound** (CPT 76700) + alpha-fetoprotein (CPT 82105) every 6 months in Child-Pugh A/B compensated cirrhosis. Do **NOT** surveil if CTP C unless transplant candidate (NCCN 2026)	AASLD 2023 HCC guidance: **semiannual surveillance is the standard**. Surveillance-detected HCC (typically earlier stage) has 5-year OS 30–50% vs. symptomatic HCC <10%. Refer to hepatology/oncology if nodule >1 cm or AFP rising

ABBREVIATIONS: ACG = American College of Gastroenterology; AASLD = American Association for the Study of Liver Diseases; BID = twice daily; CPT = Current Procedural Terminology; CSPH = clinically significant portal hypertension; CTP = Child-Turcotte-Pugh; HCC = hepatocellular carcinoma; HE = hepatic encephalopathy; LSM = liver stiffness measurement; MELD = Model for End-Stage Liver Disease; NCCN = National Comprehensive Cancer Network; NSBB = nonselective beta-blocker; OS = overall survival; SBP = spontaneous bacterial peritonitis; TID = three times daily; TIPS = transjugular intrahepatic portosystemic shunt

***Consult FDA labels for the most up-to-date dosage information, contraindications, and drug-drug interactions**

Non-Pharmacologic Treatment and Lifestyle Modification

While pharmacotherapy and procedural interventions stabilize the physiological manifestations of cirrhosis, long-term survival and the prevention of complications related to cirrhosis are primarily dictated by the patient's nutritional and physical reserve.[33–35]

INTERVENTION	TARGET/RECOMMENDATION	NOTES
Nutritional Counseling and Dietary Modification	**Sodium <2 g/day** (critical for ascites); **high-protein (1.2 g/kg/day** minimum, higher if sarcopenic); **small frequent meals**; avoid alcohol (absolute); limit fat if steatorrhea	**RDN consult essential.** BCAA supplementation if sarcopenia/malnutrition. Monitor weight, albumin, prealbumin quarterly. High-protein diet prevents sarcopenia; restriction (old myth) worsens HE. **Nutritional status is powerful frailty predictor**
Alcohol Cessation (If Alcohol-Associated Cirrhosis)	**Motivational interviewing;** alcohol support groups (AA, SMART Recovery); naltrexone 50 mg daily (preferred in older adults); acamprosate 666 mg TID (renally dosed); avoid disulfiram	**Only evidence-based intervention to slow alcohol-induced cirrhosis progression**· Naltrexone is safer than disulfiram in older adults. Addiction medicine or psychiatry comanagement is recommended. Document AUDIT-C or CAGE screening + intervention at every visit
Exercise and Physical Activity	Walking, swimming, or gentle yoga as tolerated; PT/OT if frail (CFS ≥4) for fall prevention and strengthening. **Goal: preserve muscle mass and mobility**	Exercise reduces frailty and mortality in cirrhosis; improves quality of life. PT/OT referral is especially important in sarcopenic or frail patients. Avoid strenuous exercise if variceal bleed risk or hemodynamic instability
Vaccination (All Cirrhosis Patients)	**Hepatitis A** (if anti-HAV negative): 2-dose series; **Hepatitis B** (if anti-HBs negative): 3-dose series (double-dose formulations may be needed); **pneumococcal** (Pneumovax 23 + Prevnar 20 or 15); **influenza** (annual); **COVID-19** (per current CDC guidelines)	AASLD 2021 recommends vaccination for all cirrhosis patients (immunocompromised hosts). Response may be lower in advanced cirrhosis (CTP C); vaccine response testing may guide need for booster
Social Support, Case Management and Early Palliative Care	Link to case management if hospitalization, complex medications, or polypharmacy. Mental health, social work, and caregiver support. Palliative care discussion early (MELD ≥15, CTP C, recurrent decompensation, frailty)	**Early palliative care** (not just end-stage) improves symptom control, quality of life, reduces ED/ICU utilization, and supports goals-of-care conversations. **VBC-critical intervention for high-cost populations**

ABBREVIATIONS: AUDIT-C = Alcohol Use Disorders Identification Test—Concise; BCAA = branched-chain amino acid; CAGE = Cut down, Annoyed, Guilty, Eye-opener screening tool; CDC = Centers for Disease Control and Prevention; CFS = Clinical Frailty Scale; CTP = Child-Turcotte-Pugh; ED = emergency department; ICU = intensive care unit; MELD = Model for End-Stage Liver Disease; PT/OT = physical/occupational therapy; RDN = Registered Dietitian Nutritionist; VBC = value-based care

AAVBC PERSPECTIVE ON EARLY PALLIATIVE

Palliative care in cirrhosis should begin at the point of prognostic awareness, not at the point of terminal decline. For the majority of geriatric patients, liver transplantation is likely not a viable option due to age, frailty, or comorbidity burden, ***making proactive symptom management and advance***

care planning the primary pathway to quality of life. *Standard hepatology guidelines position palliative care late in the disease trajectory, value-based care models encourage earlier integration and address undertreated symptoms such as pruritus, refractory muscle cramps, fatigue, and sleep disruption alongside structured goals-of-care conversations and realistic treatment targets calibrated to the individual patient's functional trajectory and transplant candidacy.*

Non-Pharmaceutical Prevention Strategies

Building on the non-pharmacologic interventions above, the table below summarizes the core prevention strategies and their clinical rationale.[6,7,12,36]

STRATEGY/ INTERVENTION	CLINICAL GOAL AND RATIONALE
Alcohol Cessation	Only intervention proven to slow/reverse disease progression in alcohol-associated cirrhosis
Hepatocellular Carcinoma Screening	Semiannual Ultrasound (US) + Alpha-fetoprotein (AFP)
Variceal Prophylaxis	Endoscopic surveillance (EGD) for high-risk varices; use of NSBBs or EVL
Sodium Restriction	**Strict <2 g/day** diet to manage/prevent ascites and peripheral edema
Frailty and Sarcopenia Prevention	High-protein diet (1.2–1.5 g/kg) and 'late-night snack' to prevent muscle wasting
Metabolic Control	Aggressive management of T2DM, lipids, and obesity in MASLD/MASH
Immunization & Viral Control	Full series for HAV, HBV, Pneumococcal, Flu, COVID-19; Viral suppression for HCV/HBV
Medication Stewardship	**STRICT AVOIDANCE of NSAIDs** (risk of HRS) and Benzodiazepines (risk of HE)
Early Palliative Care	Integrated symptom management (pain, pruritus, sleep) to reduce ED utilization

ABBREVIATIONS: AFP = alpha-fetoprotein; COVID-19 = coronavirus disease 2019; ED = emergency department; EGD = esophagogastroduodenoscopy; EVL = endoscopic variceal ligation; HAV = hepatitis A virus; HBV = hepatitis B virus; HCC = hepatocellular carcinoma; HCV = hepatitis C virus; HE = hepatic encephalopathy; HRS = hepatorenal syndrome; MASLD = metabolic-associated steatotic liver disease; MASH = metabolic-associated steatohepatitis; NSAIDs = nonsteroidal anti-inflammatory drugs; NSBBs = nonselective beta-blockers; T2DM = type 2 diabetes mellitus; US = ultrasound

Medication Safety and Dose Adjustments

Achieving optimal symptom management requires balancing therapeutic efficacy with clinical vigilance, making rigorous medication safety and proactive dose adjustments a foundational element of the treatment plan.[22,37,38]

AGENT/CLASS	INTERACTION/ MECHANISM	ADJUSTMENT	MONITORING
Nonselective Beta-Blockers (carvedilol, propranolol) + diuretics/ACEi	Hypotension; hyperkalemia if combined with spironolactone; bradycardia; fall risk in elderly	Target HR 55–60 bpm (not lower); **avoid if SBP <90 mmHg.** Monitor K, Cr, BP at baseline and 2 weeks. **Reduce dose if HR <55 or SBP <90**	Check HR, BP, K, Cr at each visit while titrating. **Stop NSBB if decompensation (refractory ascites, HRS criteria) develops**; restart post-stabilization if possible
Diuretics (spironolactone + furosemide) + NSAIDs or ACEi	Acute kidney injury; hyperkalemia (spironolactone); hyponatremia; volume depletion → HRS	**Avoid NSAIDs entirely if eGFR <30**; if ≥30 and necessary, use lowest dose × shortest duration. **Preferred pain relief: acetaminophen <2 g/day.** Hold diuretics if Cr rising or dehydration	Baseline and monthly Cr, K, Na. Monitor weight and urine output weekly while titrating diuretics. Stop diuretics if oliguria or Cr >2× baseline develops
lactulose + rifaximin (HE treatment) + opioids or anticholinergics	Severe constipation (opioids block bowel function); delayed GI transit (anticholinergics) → reduced lactulose efficacy; worsening HE	**Minimize or avoid opioids if possible**. If opioid required, add docusate + senna prophylactically. Avoid anticholinergic medications (diphenhydramine, promethazine)	Assess bowel function weekly (goal 2–3 soft stools/day on lactulose). If constipation, increase lactulose dose; deprescribe opioid if possible
Benzodiazepines (lorazepam, diazepam) + Any Sedating Agent	Worsening HE (benzodiazepines metabolized by liver, accumulate); oversedation; falls; aspiration risk	**Avoid benzodiazepines in cirrhosis;** use alternatives (propofol for sedation in ICU, hydroxyzine for anxiety if short-term needed, SSRIs for chronic anxiety)	If benzodiazepine is absolutely required (seizure prophylaxis post-variceal bleed, acute agitation), use lorazepam (shorter half-life) × minimal dose. Do **NOT** taper abruptly (seizure risk); plan slow taper over weeks

AGENT/CLASS	INTERACTION/ MECHANISM	ADJUSTMENT	MONITORING
Antidiabetic Agents (metformin, GLP-1 RA, insulin) in cirrhosis + CKD/HRS Risk	**metformin:** lactic acidosis risk if eGFR <30 or HRS. GLP-1 RA: dehydration (diuretics + GLP-1) → AKI **Insulin:** hypoglycemia especially in sarcopenia/ malnutrition; HE + hypoglycemia = severe altered mental status	**Avoid metformin if eGFR <30.** GLP-1 RA: safe in cirrhosis (MASLD benefit) but monitor volume status; hold if dehydration/HRS suspected. Insulin: target HbA1c 7–8% in elderly to avoid hypoglycemia	Baseline and ongoing eGFR, electrolytes, volume status. If GLP-1 RA, monitor for dehydration (thirst, dark urine, orthostasis). Glucose logs for insulin-treated patients; educate on hypoglycemia recognition
NSAIDs (ibuprofen, naproxen, ketorolac) + Diuretics in Decompensated Cirrhosis	**Acute kidney injury** (HRS criteria); GI bleeding (in varices risk); sodium/fluid retention → worsening ascites	**Avoid NSAIDs entirely if decompensated, ascites, or eGFR <30**. Compensated (no ascites, eGFR >45): acetaminophen <2 g/day preferred; if NSAID absolutely needed, lowest dose × shortest duration, monitor Cr closely	Baseline Cr; recheck at 2 weeks and 2 months if NSAID used. Educate on NSAID avoidance; offer acetaminophen alternatives. If Cr rises >0.3 mg/dL in 48 h, stop NSAID immediately
Sodium-Glucose Cotransport Inhibitor (SGLT2i: empagliflozin) — Emerging Benefit in MASLD	**Dehydration risk;** DKA; Fournier gangrene (rare). In cirrhosis, **SGLT2i may reduce liver fibrosis progression**; benefit data emerging (trial data pending)	If SGLT2i used in MASLD cirrhosis (pending stronger evidence), ensure adequate fluid intake, monitor volume status closely, hold if dehydration/AKI suspected	Volume status assessment weekly; Cr, Na, glucose at baseline and 2 weeks. **DKA risk counseling** (rare; educate on sick-day management). **Monitor for signs of genital infection**

ABBREVIATIONS: ACEi = angiotensin-converting enzyme inhibitor; AKI = acute kidney injury; CKD = chronic kidney disease; CPT = Current Procedural Terminology; Cr = creatinine; DKA = diabetic ketoacidosis; ED = emergency department; eGFR = estimated glomerular filtration rate; GI = gastrointestinal; GLP-1 RA = glucagon-like peptide-1 receptor agonist; HE = hepatic encephalopathy; HRS = hepatorenal syndrome; ICU = intensive care unit; MASLD = metabolic-associated steatotic liver disease; MELD = Model for End-Stage Liver Disease; Na = sodium; NSAID = nonsteroidal anti-inflammatory drug; NSBB = nonselective beta-blocker; SGLT2i = sodium-glucose cotransport inhibitor 2
***Consult FDA labels for the most up-to-date dosage information, contraindications, and drug-drug interactions**

When to Refer

Specific clinical thresholds and emergent findings mandate immediate referral to specialized hepatology or surgical services.[6,7,36]

CRITERION	SPECIALIST	URGENCY
Cirrhosis diagnosis confirmation (unclear etiology, atypical imaging, risk stratification for transplant candidacy)	**Hepatology** or **Gastroenterology**	**ROUTINE (2–4 weeks)** for uncomplicated compensated; URGENT (1 week) if decompensation (ascites, HE, variceal bleed, jaundice) or MELD ≥15 suspected
Carcinoma detected on imaging (nodule >1 cm) or rising AFP (>400 ng/mL or doubling in <4 weeks)	**Hepatology** + Medical Oncology + Transplant Surgery (multidisciplinary HCC consultation)	**URGENT (within 3–5 days)** to establish BCLC stage and treatment plan (curative vs. palliative)
Variceal hemorrhage (active bleeding or recent bleed with planned repeat EGD)	**Gastroenterology** with endoscopy capability (for EGD + EVL/sclerotherapy)	**IMMEDIATE (ED)** if active hemorrhage; URGENT (next day) if recent bleed with recurrent hemodynamic instability; **ROUTINE** (within 2 weeks) for primary prophylaxis EGD in known varices
Malnutrition, sarcopenia, or unresponsive to PERT-type nutritional interventions	Registered **Dietitian Nutritionist** (RDN); consider home health for in-home assessment if homebound	**ROUTINE (1–2 weeks)** for malnutrition; **URGENT** (same week) if albumin <2.5 or rapid weight loss (>10% in <3 months)
Refractory ascites (no weight loss despite max diuretics × 2+ weeks) or tense ascites with respiratory compromise	**Interventional Radiology** (TIPS evaluation) or **Gastroenterology** (repeated paracentesis management)	**ROUTINE (1–2 weeks)** for uncomplicated; URGENT (same day) if respiratory distress from tense ascites
Advanced Frailty (CFS ≥5 or LFI ≥4.27) with multiple comorbidities, repeated hospitalizations, or acute decompensation	**Multidisciplinary and individualized:** Geriatrics, Palliative Medicine,Case Management,Social Work,PT/OT,Nutrition; Psychiatry if depression/anxiety	**ROUTINE (within 1 week)** for stable frailty; URGENT (same day/next day) if acute decompensation, suicidal ideation, or acute functional decline

ABBREVIATIONS: BCLC = Barcelona Clinic Liver Cancer; CPT = Current Procedural Terminology; CFS = Clinical Frailty Scale; ED = emergency department; EVL = endoscopic variceal ligation; HCC = hepatocellular carcinoma; LFI = Liver Frailty Index; MELD = Model for End-Stage Liver Disease; PT/OT = physical/occupational therapy; RDN = Registered Dietitian Nutritionist; TIPS = transjugular intrahepatic portosystemic shunt

Follow-Up Timing

Following immediate clinical decision points for specialist referral, long-term success for the patient requires a structured follow-up schedule tailored to the patient's disease stage and stability.[35,39]

INTERVAL/SCENARIO	VISIT FOCUS	KEY ACTIONS/DOCUMENTATION
Compensated cirrhosis, MELD <15, no complications (stable baseline)	Annual wellness visit + chronic disease management; or **every 6 months if on diuretics/NSBBs**	**Repeat MELD 3.0** (labs: INR, bili, Cr, Na); check for ascites, HE, jaundice, weight change; review HCC surveillance schedule (last US/AFP date); renew diuretics/NSBB prescriptions; annual HCC surveillance due; check CFS/frailty score; address medication adherence
Decompensated cirrhosis (ascites, HE, variceal bleed history, or MELD ≥15)	**Every 2–4 weeks initially**; then monthly if stable on medications; sooner if clinical change	**MEAT** (Monitor: ascites grade, HE grade, weight, labs; Evaluate: MELD, imaging if new ascites/jaundice; Assess: frailty, comorbidities, medication reconciliation; Treat: adjust diuretics, NSBB, lactulose, prophylaxis). **HCC surveillance every 6 months.** Variceal surveillance per EGD interval (Baveno VI/VII). Consider transplant evaluation
Post-acute event (variceal bleed, SBP, HE episode, HRS, or hospital discharge)	**2–3 days post-discharge; then weekly × 4 weeks;** then as clinically indicated	**Assess for persistent complications** (bleeding, ascites, altered mental status, renal dysfunction); medication reconciliation (deprescribe high-risk agents; add prophylaxis if indicated); check labs (CBC, CMP, INR, Cr, Na, ammonia if HE concern); arrange specialist follow-up (GI, hepatology, nephrology as needed); assess psychosocial needs (depression, social support, caregiver burden); reinforce discharge instructions
Frailty (CFS ≥5 or LFI ≥4.27) or low albumin (<3.0) on nutritional support	**Every 4 weeks** for nutrition/RDN; PT/OT assessment; case management; social work + palliative care	Nutrition labs (albumin, prealbumin) monthly; weight weekly (goal stable or gradual gain); assess medication adherence and side effects; review advance directives and goals of care; document CFS/LFI score and trend; escalate palliative care involvement
Hepatocellular Carcinoma detected (nodule >1 cm confirmed)	**Urgent multidisciplinary clinic (within 3–5 days)**; then per oncology treatment plan (every 2–4 weeks if on systemic therapy or during/after curative intent treatment)	**BCLC staging**; treatment plan documentation (curative vs. palliative); baseline performance status; imaging (CT/MRI per protocol); alpha-fetoprotein trend; assess goals of care (curative intent, palliative, hospice); link to oncology, transplant surgery, palliative care. Restaging imaging per treatment protocol
Post-HCV cure (sustained virologic response achieved after DAA therapy) or post-HBV treatment optimization	**Every 6–12 months**; HCC surveillance continues (cirrhosis diagnosis persists despite viral clearance)	**Reassess MELD/CTP; continue HCC surveillance** (US + AFP every 6 months per AASLD 2023); may consider deprescribe/reduce frequency of HBV antivirals if HBsAg clears + anti-HBs develops (rare); assess for portal hypertension complications (ascites, varices); **continue variceal prophylaxis if indicated**

INTERVAL/SCENARIO	VISIT FOCUS	KEY ACTIONS/DOCUMENTATION

ABBREVIATIONS: BCLC = Barcelona Clinic Liver Cancer; CBC = complete blood count; CMP = comprehensive metabolic panel; CPT = Current Procedural Terminology; CFS = Clinical Frailty Scale; CT = computed tomography; CTP = Child-Turcotte-Pugh; DAA = direct-acting antiviral; ED = emergency department; EGD = esophagogastroduodenoscopy; HCC = hepatocellular carcinoma; HCV = hepatitis C virus; HE = hepatic encephalopathy; HRS = hepatorenal syndrome; LFI = Liver Frailty Index; MELD = Model for End-Stage Liver Disease; MRI = magnetic resonance imaging; MEAT = Monitor/Evaluate/Assess/Treat; PT/OT = physical/occupational therapy; RDN = Registered Dietitian Nutritionist; SBP = spontaneous bacterial peritonitis; US = ultrasound

Comorbidity Management

Effective long-term success in cirrhosis management depends on aggressive treatment of the co-existing conditions that contribute to decompensation.[5,22,30]

COMORBIDITY	INTERACTION WITH CIRRHOSIS	MANAGEMENT NOTES (CIRRHOSIS-SPECIFIC ADJUSTMENTS)
Type 2 Diabetes Mellitus (E11.x; 56% in MASLD cirrhosis)	**HbA1c unreliable in Child-Pugh** B/C (anemia, hemolysis); glycemic control brittle if sarcopenic; diuretics + NSBBs affect glucose stability	**Prefer CGM over HbA1c in advanced cirrhosis.** Avoid metformin if eGFR <30; GLP-1 RAs safe (MASLD benefit) but monitor for dehydration. Avoid glinides (hypoglycemia risk). Target HbA1c 7–8% to avoid hypoglycemia in elderly. Monitor glucose 2–4× daily; educate on hypoglycemia recognition
Chronic Kidney Disease Stage 3–5 (eGFR <60; 45–46% of cirrhosis patients)	**CKD + cirrhosis = rapid progression risk** (dual organ stress). Diuretics, NSBBs, NSAIDs, ACEi/ARB all modulate renal function; monitoring critical	**Avoid NSAIDs, ACEi/ARB if eGFR <30 or AKI risk.** Monitor Cr, Na, K at baseline and every 3–6 months (more frequently if diuretics). Distinguish CKD from HRS (acute Cr rise vs. chronic baseline). **Nephrology comanagement if Stage 4–5. Avoid renally cleared drugs** (adjust pregabalin, metformin, fluoroquinolones)
Hypertension (I10; 45% of cirrhosis >65)	NSBBs (carvedilol, propranolol) **serve dual purpose** (varices + BP control); diuretics lower BP. Overtreatment → hypotension → HRS risk	**BP target in cirrhosis: systolic 120–130 mmHg** (not too low; avoid HRS). NSBB (carvedilol preferred) primary agent. **Avoid ACEi/ARB if eGFR <30 or HRS risk.** Monitor BP at each visit; hold diuretics/NSBB if SBP <90 mmHg. **Calcium channel blockers (amlodipine) safe alternative** if NSBB contraindicated

COMORBIDITY	INTERACTION WITH CIRRHOSIS	MANAGEMENT NOTES (CIRRHOSIS-SPECIFIC ADJUSTMENTS)
Cardiovascular Disease (CVD; 45% overall, higher in age ≥65)	Dual comorbidity (CVD + cirrhosis): mortality risk additive; HR 2.5 × adjusted mortality. Cirrhosis-specific treatments (NSBBs, diuretics) manage CVD but carry HRS/AKI risk	**NSBBs provide cardioprotection + portal HTN benefit.** Avoid NSAIDs (GI bleed + AKI risk); use acetaminophen for pain. ACEi/ARB contraindicated if eGFR <30 or ascites. **Aspirin: avoid if varices or thrombocytopenia**. Statins: safe but no proven cirrhosis-specific benefit; avoid aggressive lipid targets. Coordinate cardiology + hepatology comanagement
Obesity/Metabolic Syndrome (E66.01 BMI ≥40; I10, E78.x dyslipidemia)	MASLD/NASH **cirrhosis driven by obesity + diabetes + dyslipidemia**	GLP-1 RA (e.g., semaglutide) shows MASLD benefit; safe in cirrhosis but monitor hydration. Weight loss goal modest (5–10% if overweight); aggressive weight loss → malnutrition risk. **High-protein diet during weight loss.** Metabolic components must be coded (**E11.x, E66.x, E78.x, I10**) for HCC mapping
Sarcopenia/Frailty (30–70% in ESLD)	**Muscle loss independently predicts decompensation** + mortality beyond MELD. Inflammation, ammonia, malnutrition, physical inactivity drive sarcopenia	High-protein diet (1.2 g/kg/day); BCAA supplementation if sarcopenic; PT/OT for resistive exercise. LFI grip strength / CFS score track sarcopenia. Avoid protein restriction (counterproductive). Nutritionist involvement essential. Monitor weight, albumin, prealbumin monthly

ABBREVIATIONS: ACEi = angiotensin-converting enzyme inhibitor; BCLC = Barcelona Clinic Liver Cancer; BCAA = branched-chain amino acid; CGM = continuous glucose monitoring; CKD = chronic kidney disease; CVD = cardiovascular disease; E11.x = type 2 diabetes codes; E66.01 = morbid obesity code; E78.x = dyslipidemia codes; ED = emergency department; ESLD = end-stage liver disease; eGFR = estimated glomerular filtration rate; GLP-1 RA = glucagon-like peptide-1 receptor agonist; HAS = hepatic artery stenosis; HCC = hepatocellular carcinoma; HRS = hepatorenal syndrome; I10 = hypertension code; MASLD = metabolic-associated steatotic liver disease; MELD = Model for End-Stage Liver Disease; NASH = nonalcoholic steatohepatitis; NSAID = nonsteroidal anti-inflammatory drug; NSBB = nonselective beta-blocker; PT/OT = physical/occupational therapy; RDN = Registered Dietitian Nutritionist

***Consult FDA labels for the most up-to-date dosage information, contraindications, and drug-drug interactions**

Cost-Smart Options

Optimizing treatment adherence also requires a focus on cost-effective, generic therapeutic options.

DRUG CLASS	BRAND NAME	BRAND (EST. COST)	GENERIC AVAILABILITY	TYPICAL MONTHLY EST. COST (GENERIC)
HE Prophylaxis	Xifaxan	$1,000+	Yes (rifaximin) (Released 2026)	$115 – $250

DRUG CLASS	BRAND NAME	BRAND (EST. COST)	GENERIC AVAILABILITY	TYPICAL MONTHLY EST. COST (GENERIC)
HE Management	Enulose	$150+	Yes (lactulose)	$15 – $35
Variceal Risk	Coreg	$250+	Yes (carvedilol) *Carvedilol preferred over propranolol (superior portal pressure reduction; also treats hypertension)*	$10 – $50
SBP Prevention	Bactrim DS	$100+	Yes (SMX-TMP DS)	$8 – $25
Edema/Ascites	Aldactone	$120+	Yes (spironolactone)	$15 – $30
Edema/Ascites	Lasix	$100+	Yes (furosemide)	$10 – $20
Biliary Disease	Urso/Actigall	$350 – $600	Yes (ursodiol)	$30 – $80

***Consult FDA labels for the most up-to-date dosage information, contraindications, and drug-drug interactions**

Patient Education and Adherence

While cost-smart therapeutic choices improve access, achieving long-term outcomes and preventing readmissions ultimately depends on consistent patient adherence, which is facilitated by targeted education.[22,27]

FOCUS AREA	CLINICAL INTERVENTION AND STRATEGY	RATIONALE
Volume Control	Low-Sodium Diet (<2 g/day) + Diuretic Adherence	Prevents ascites/edema; **reduces preventable 30-day readmissions**
Alcohol Cessation	Sobriety is mandatory. Use Naltrexone, AA/SMART Recovery	Only intervention proven to arrest or reverse fibrosis in ALD
Nutritional Care	High Protein (1.2–1.5 g/kg/day) + Gentle exercise	Prevents Sarcopenia. Muscle helps clear ammonia; loss increases HE risk
HCC Surveillance	Semiannual US + AFP (**every 6 months**)	Shift from palliative to curative intent through early detection
Medication Safety	**STRICT AVOIDANCE of NSAIDs, Benzos, and Narcotics**	Prevents iatrogenic AKI/Hepatorenal Syndrome and HE triggers

FOCUS AREA	CLINICAL INTERVENTION AND STRATEGY	RATIONALE
Preventive Health	Annual Screens (DEXA, Vit D) + Vaccines (HAV, HBV, Flu)	Patients are immunocompromised; prevents Acute-on-Chronic Failure
Care Coordination	Early Palliative Care Integration (MELD ≥ 15 or CFS ≥ 5)	Focuses on symptom relief; reduces futile ICU use and improves QoL
Paracentesis	A dedicated outpatient paracentesis clinic (**FL**uid **AS**Piration [**FLASP**] model)	**ED paracentesis is the most expensive and least efficient setting.** A dedicated outpatient paracentesis clinic can **reduce ED visits for paracentesis** with low complication rates and high patient satisfaction

ABBREVIATIONS: AA = Alcoholics Anonymous; AFP = alpha-fetoprotein; AKI = acute kidney injury; ALD = alcohol-associated liver disease; CFS = Clinical Frailty Scale; DEXA = dual-energy X-ray absorptiometry; FLASP = **Fl**uid **Asp**iration; HAV = hepatitis A virus; HBV = hepatitis B virus; HCC = hepatocellular carcinoma; HE = hepatic encephalopathy; ICU = intensive care unit; MELD = Model for End-Stage Liver Disease; NSAIDs = nonsteroidal anti-inflammatory drugs; QoL = quality of life; SMART = Self-Management and Recovery Training; US = ultrasound; Vit D = vitamin D

Quality Metrics Tie-In

Below is the table mapping HEDIS, CMS Star, and MIPS quality measures to their applicability in liver cirrhosis care:[40]

MEASURE (MY2026)	STANDARD	APPLICABILITY TO LIVER CIRRHOSIS
HEDIS COA: Care for Older Adults — Medication Review	**Denominator**: MA enrollees ≥66, continuously enrolled **Numerator:** 2 sub-measures documented annually: (1) functional status assessment, (2) medication review **Exclusions:** hospice, ESRD	**Polypharmacy is common in decompensated cirrhosis** — medication review should reconcile nephrotoxins (NSAIDs, ACEi/ARBs, aminoglycosides), lactulose/rifaximin for HE, diuretics (spironolactone/furosemide), and beta-blockers for portal hypertension. Beers Criteria review is high-yield: **benzodiazepines can precipitate HE; anticholinergics worsen cognition in covert HE**. AASLD quality measures include medication review as a core process measure for all cirrhosis patients[27]
HEDIS COA: Care for Older Adults — Functional Status Assessment	**Denominator:** enrollees ≥66 **Numerator:** functional status assessment documented annually **Exclusions:** hospice, ESRD	AASLD Practice Metrics Committee rates frailty assessment as a high-importance quality measure (median importance 8/9) for all cirrhosis patients. Clinical Frailty Scale, Liver Frailty Index, or chair stand test directly satisfy this sub-measure; document at every visit to track functional trajectory and guide transplant evaluation vs. palliative care discussions[35]

MEASURE (MY2026)	STANDARD	APPLICABILITY TO LIVER CIRRHOSIS
HEDIS COA: Care for Older Adults — Advance Care Planning	**Denominator:** enrollees ≥66 **Numerator:** advance care planning documented annually; CPT 99497/99498	**Cirrhosis patients face 27% 30-day readmission rates and high mortality with decompensation**. Advance care planning should be initiated during stable disease — document goals of care, transplant candidacy discussion, and code status. Particularly critical before MELD >15 threshold when transplant evaluation is recommended[41]
HEDIS ASF: Unhealthy Alcohol Use Screening and Follow-Up (Rate 1 + Rate 2)	Rate 1 **Denominator:** enrollees ≥18 **Numerator:** screened for unhealthy alcohol use with validated instrument (AUDIT-C, SASQ) during measurement year Rate 2 **Denominator:** positive screens from Rate 1 **Numerator:** brief intervention or referral within 2 months. ECDS measure — requires structured data documentation (LOINC, ICD-10-CM, CPT, SNOMED, HCPCS); unstructured narrative notes do **NOT** satisfy the measure	Directly applicable to all cirrhosis patients regardless of etiology — alcohol use worsens outcomes in MASLD, viral hepatitis, and ALD alike. AUDIT-C ≥4 (men) / ≥3 (women) = positive. Only 14% of ALD patients receive any AUD treatment[36], 1% pharmacotherapy. AASLD rates substance abuse counseling/referral within 2 months as importance 9/9. G0442 (screen) + G0443 (counseling) billable under Medicare Part B[8]
HEDIS TSC-E: Tobacco Use Screening and Cessation Intervention	**Denominator** 1: enrollees ≥12 **Numerator 1:** screened with documented tobacco status **Denominator 2:** positive tobacco users **Numerator 2:** received cessation counseling or pharmacotherapy; Exclusions: death, hospice	**Tobacco use accelerates fibrosis progression and increases HCC risk in cirrhosis**.[6] Document tobacco status at every visit; active smoking is an independent risk factor for decompensation. USPSTF Grade A recommendation. Particularly relevant for ALD patients where dual substance use (alcohol + tobacco) is common
HEDIS TRC: Transitions of Care	**Denominator:** enrollees ≥18 with inpatient discharge **Numerator:** 4 sub-measures within 30 days: (1) notification of inpatient admission, (2) receipt of discharge information, (3) patient engagement after discharge, (4) medication reconciliation post-discharge **Exclusions:** hospice	**Cirrhosis patients have 27–53% readmission rates within 30–90 days — among the highest of any chronic disease**.[41] AASLD recommends clinic visit within 4 weeks of discharge (importance 8/9). Medication reconciliation is critical: nephrotoxin avoidance, diuretic dose adjustment, lactulose titration, and crisis-trigger medication review. HE is the strongest predictor of readmission (OR 1.77)[41]

MEASURE (MY2026)	STANDARD	APPLICABILITY TO LIVER CIRRHOSIS
HEDIS MRP: Medication Reconciliation Post-Discharge	**Denominator:** enrollees ≥18 discharged from inpatient facility **Numerator:** medication reconciliation conducted by prescribing practitioner, clinical pharmacist, or RN within 30 days of discharge	**Preventable readmissions in cirrhosis are often related to paracentesis timeliness, diuretic adjustment monitoring, and HE treatment failures**.[33] Post-discharge medication reconciliation should specifically address: nephrotoxin removal (NSAIDs, ACEi/ARBs), lactulose/rifaximin adherence, diuretic electrolyte monitoring, and AUD pharmacotherapy initiation
HEDIS DAE: Use of High-Risk Medications in Older Adults	**Denominator:** enrollees ≥67 **Numerator:** received ≥1 high-risk medication per Beers Criteria; Lower rate = better performance	**High-yield in cirrhosis:** benzodiazepines precipitate/worsen HE; NSAIDs cause AKI (present in up to 1/3 of severe AH, 9-fold 90-day mortality); anticholinergics worsen covert HE. Beta-blocker review is important — while indicated for portal hypertension, dose adjustment is critical in decompensated disease with hypotension[39]
CMS Star: Controlling Blood Pressure (CBP)	**Denominator:** enrollees 18–85 with HTN diagnosis **Numerator:** most recent BP 140/90 mmHg	**HTN is common in cirrhosis patients with metabolic comorbidities (MASLD, diabetes)**. ACEi/ARBs should be avoided in decompensated cirrhosis with ascites (AASLD 2021) — use alternative agents. Beta-blockers for portal hypertension may contribute to BP control but require dose reduction or discontinuation if SBP 90 or refractory ascites develops[39]
CMS Star: Plan All-Cause Readmissions (PCR)	30-day readmission rate; lower is better; risk-adjusted	**Cirrhosis has among the highest 30-day readmission rates of any chronic disease**. Cirrhosis as a comorbidity increases readmission in HRRP-targeted conditions, and readmission rates have **NOT** improved under HRRP for cirrhosis patients unlike non-cirrhosis populations. Targeted interventions: post-discharge clinic within 4 weeks, medication reconciliation, HE prophylaxis, AUD treatment initiation before discharge[41]
CMS Star: Statin Therapy for Patients with Cardiovascular Disease (SPC)	**Denominator:** enrollees with clinical ASCVD **Numerator:** received statin therapy of any intensity during measurement year	Many cirrhosis patients have concurrent ASCVD (especially MASLD-related). Statins are generally safe in compensated cirrhosis and may have antifibrotic properties. Discontinue or dose-reduce only in decompensated cirrhosis (Child-Pugh C) with significantly elevated transaminases. Document clinical rationale if statin is held[7]

MEASURE (MY2026)	STANDARD	APPLICABILITY TO LIVER CIRRHOSIS
CMS Star: Diabetes Care — HbA1c Control (8%)	**Denominator:** enrollees 18–75 with diabetes **Numerator:** most recent HbA1c 8.0%	Diabetes is present in **30–50%** of cirrhosis patients and is an independent predictor of readmission (OR in multicenter data). HbA1c may underestimate glycemia in cirrhosis due to shortened RBC lifespan and anemia — fructosamine or continuous glucose monitoring may be more accurate. Metformin is safe in compensated cirrhosis but contraindicated in decompensated disease; SGLT-2 inhibitors require caution with volume status[42]
HEDIS AIS: Adult Immunization Status	Pneumococcal, influenza, HZV per age-based schedule	AASLD rates hepatitis B vaccination documentation as importance 8/9 with reach 9/9 for all cirrhosis patients. Hepatitis A vaccination is also recommended if non-immune. Influenza and pneumococcal vaccines reduce infection-related decompensation. Inactivated vaccines only during immunosuppression (post-transplant)

ABBREVIATIONS: AASLD = American Association for the Study of Liver Diseases; ACEi = angiotensin-converting enzyme inhibitor; AH = alcoholic hepatitis; AIS = Adult Immunization Status; AKI = acute kidney injury; ALD = alcohol-associated liver disease; ARB = angiotensin receptor blocker; ASCVD = atherosclerotic cardiovascular disease; ASF = Unhealthy Alcohol Use Screening and Follow-Up; AUD = alcohol use disorder; AUDIT-C = Alcohol Use Disorders Identification Test–Concise; AWV = Annual Wellness Visit; BP = blood pressure; CBP = Controlling Blood Pressure; CMS = Centers for Medicare & Medicaid Services; COA = Care for Older Adults; CPT = Current Procedural Terminology; DAE = Use of High-Risk Medications in Older Adults; ECDS = Electronic Clinical Data Systems; ESRD = end-stage renal disease; HAV = hepatitis A virus; HBV = hepatitis B virus; HbA1c = hemoglobin A1c; HCC = hepatocellular carcinoma; HCPCS = Healthcare Common Procedure Coding System; HE = hepatic encephalopathy; HEDIS = Healthcare Effectiveness Data and Information Set; HRRP = Hospital Readmissions Reduction Program; HTN = hypertension; HZV = herpes zoster vaccine; ICD-10-CM = International Classification of Diseases, 10th Revision, Clinical Modification; LOINC = Logical Observation Identifiers Names and Codes; MA = Medicare Advantage; MASLD = metabolic-associated steatotic liver disease; MELD = Model for End-Stage Liver Disease; MRP = Medication Reconciliation Post-Discharge; MY = measurement year; NSAIDs = nonsteroidal anti-inflammatory drugs; OR = odds ratio; PCR = Plan All-Cause Readmissions; RBC = red blood cell; RN = registered nurse; SASQ = Single Alcohol Screening Question; SBP = systolic blood pressure; SGLT-2 = sodium-glucose cotransporter-2; SNOMED = Systematized Nomenclature of Medicine; SPC = Statin Therapy for Patients with Cardiovascular Disease; TRC = Transitions of Care; TSC-E = Tobacco Use Screening and Cessation Intervention; USPSTF = United States Preventive Services Task Force

QUALITY OUTCOME: SPECIALIST AND SUPPORTIVE CARE INTEGRATION

Strategic Intervention: Hepatology and Palliative Care referral at **MELD 3.0 >15** or first decompensation event (Ascites/HE) reduces ICU utilization and increases the likelihood of curative transplant evaluation.[1]

Documentation Requirement: Document the referral date, the specific clinical trigger (e.g., "MELD score 17" or "Grade 2 Ascites"), and the management plan in the **MEAT** section to satisfy **HEDIS PCR** (Readmission) and **MIPS 047** (Advance Care Planning) metrics.

5 CODING REMINDERS AND CASE EXAMPLES

Good Documentation is Comprehensive Coding

SHORTCUT/RISK	PREFERRED DOCUMENTATION LANGUAGE
Coding K74.60 (unspecified cirrhosis) when etiology is known	Specify etiology: "Alcoholic cirrhosis of liver without ascites (**K70.30**)" or "MASLD-related cirrhosis (**K74.69 + K75.81**)." **K74.60** (unspecified) has lower PPV in administrative databases and suppresses etiology-specific quality tracking
Coding K74.60 when patient has decompensated disease (ascites, HE, varices)	Document each decompensation event explicitly: "Decompensated alcoholic cirrhosis with ascites (**K70.31**), hepatic encephalopathy grade II (**K72.11**), and esophageal varices without bleeding (**I85.00**); portal hypertension (**K76.6**); MELD-Na 22 (Bili 3.2, INR 1.6, Na 131, Cr 1.4 on [date]); Child-Pugh Class C." Decompensation reduces survival — coding must reflect this severity shift
Using R18.8 (ascites) alone without linking to liver disease	Always pair ascites with the cirrhosis code: "Alcoholic cirrhosis with ascites (**K70.31**)" or "Other cirrhosis with ascites (**K74.69 + R18.8**)." Pairing with a liver disease code is required for accurate identification
Using Z87.891 ("history of alcoholic liver disease") when disease is active	Use active disease codes while cirrhosis is present, bilirubin is elevated, or patient is on active treatment: "Active alcoholic cirrhosis with ascites (**K70.31**); MELD-Na 18; on spironolactone/furosemide; HCC surveillance current." Reserve **Z87.891 ONLY** for resolved disease with no evidence of active liver injury
Not documenting hepatic encephalopathy grade or coma status	Specify grade and coma status: "Chronic hepatic failure with coma (**K72.11**)" vs. "without coma (**K72.10**)." West Haven Criteria grade should be documented (grade 0–4). Overt HE (grades ≥2) presents with asterixis, disorientation, lethargy; covert HE (grades ≤1) may present as executive function deficits or sleep disorder
"Cirrhosis, follow up with hepatology" without MELD, Child-Pugh, or complication status	"Decompensated MASLD cirrhosis (**K74.69**), Child-Pugh Class B (score 8), MELD-Na 16 (Bili 1.8, INR 1.4, Na 134, Cr 1.1 on [date]). Moderate ascites on spironolactone 100 mg + furosemide 40 mg daily. HCC surveillance (US + AFP) completed [date] — negative. Variceal screening endoscopy [date] — no high-risk varices. Carvedilol 6.25 mg BID for portal hypertension prophylaxis"
"HCC screening done" without specifying modality, result, or interval	"HCC surveillance: abdominal ultrasound + AFP performed [date]; US negative, AFP [value]; next surveillance in 6 months per AASLD/NCCN. US visualization quality: adequate/limited." Document LI-RADS category if applicable. Surveillance adherence is quite low nationally — structured documentation improves tracking

SHORTCUT/RISK	PREFERRED DOCUMENTATION LANGUAGE
Not coding concurrent AUD (F10.xx) alongside alcoholic cirrhosis (K70.3x)	Always sequence AUD alongside ALD codes: "Alcohol use disorder, moderate, in remission (**F10.20**); alcoholic cirrhosis with ascites (**K70.31**). AUDIT-C [score] at today's visit; naltrexone 50 mg/day initiated; brief intervention documented (**G0443**)." Only 14% of ALD patients receive any AUD treatment — coding AUD triggers quality measure tracking (HEDIS ASF)
"On diuretics" without dose, response, or monitoring plan	"Ascites managed with spironolactone 100 mg daily + furosemide 40 mg daily (100:40 ratio maintained); weight [value], down [value] kg from [date]; creatinine [value] (stable); sodium [value]; potassium [value]. No AKI. Paracentesis not required this visit. Next labs in [timeframe]"
Not documenting variceal screening or prophylaxis status	"Variceal screening: EGD [date] — small varices, no red signs; carvedilol 6.25 mg BID for primary prophylaxis per Baveno/AASLD. Heart rate [value]. Next endoscopy in [2–3 years if compensated/1 year if decompensated]. LSM [value] kPa; platelets [value] — Baveno VI criteria [met/not met] for endoscopy deferral"
Coding K76.0 (fatty liver) when patient has progressed to cirrhosis	**K76.0** is for steatosis without fibrosis or cirrhosis. Once cirrhosis is established, use **K74.69** (other cirrhosis) + **K75.81** (MASH) to document both the etiology and the fibrotic stage. "MASLD-related cirrhosis (**K74.69 + K75.81**), compensated, LSM 18 kPa on VCTE [date]; FIB-4 [value]"
Not documenting nutritional status or sarcopenia in decompensated cirrhosis	"Malnutrition, moderate (**E44.0**); sarcopenia (**M62.84**). BMI [value]; grip strength [value]; Liver Frailty Index [value]. Nutrition plan: 35–40 kcal/kg/day, 1.2–1.5 g protein/kg/day — protein **NOT** restricted. RD consultation [date]. Thiamine, zinc, folate supplemented"

ABBREVIATIONS: AUD = alcohol use disorder; BCLC = Barcelona Clinic Liver Cancer; CFS = Clinical Frailty Scale; CTP = Child-Turcotte-Pugh; DAA = direct-acting antiviral; EGD = esophagogastroduodenoscopy; EVL = endoscopic variceal ligation; FIB-4 = fibrosis-4 index; HCC = hepatocellular carcinoma (also CMS-HCC risk category); HCV = hepatitis C virus; HE = hepatic encephalopathy; ICD-10 = International Classification of Diseases, 10th revision; INR = international normalized ratio; LFI = Liver Frailty Index; MASLD = metabolic dysfunction-associated steatotic liver disease; MELD = Model for End-Stage Liver Disease; RAF = risk adjustment factor; SVR = sustained virologic response; US = ultrasound; VBC = value-based care

Documentation Specificity

- **Stage/Severity:** Specify LC etiology (**K74.69** MASLD vs. **K70.30/31** Alcohol vs. **K74.3–5** Biliary) and current MELD 3.0 + Child-Pugh Class (A/B/C). Accurate staging (e.g., MELD >15 or CTP B/C) is a clinical imperative for VBC risk stratification and transplant referral thresholds
- **Current Status:** Document the "Big Four" complications: Ascites (Grade 0–3), HE (West Haven 0–4), Variceal status (date of last EGD/prophylaxis), and HCC Surveillance (date of last US+AFP). Current status anchors the clinical trajectory and justifies high-intensity management
- **Associated Conditions:** Explicitly code active comorbidities—CKD Stage (**N18.x**), Diabetes (**E11.x**), and Sarcopenia/Frailty (CFS 1–9). Under V28, capturing these independent risk factors is essential for accurate risk-adjustment and identifying patients at high risk for 30-day readmission

- **Medication Stewardship:** List active VBC-critical meds: NSBBs (Carvedilol dose/HR target), Diuretics, and HE/SBP prophylaxis (Lactulose/Rifaximin/SMX-TMP). Confirm avoidance of nephrotoxins (NSAIDs) to demonstrate active renal protection and safety monitoring
- **Chronicity:** Confirm persistence via serial labs or imaging — "confirmed >3 months" — to distinguish chronic cirrhosis from acute liver failure. Use active codes; never use **Z87.19** (History of) for active LC, as it suppresses HCC mapping and underrepresents patient complexity

Annual Clinical Review and Confirmation

Confirm cirrhosis remains active, etiology-defined, and clinically staged at least once per calendar year:

- **Annual Review:** Cirrhosis must be reassessed once per calendar year via face-to-face or synchronous audio-video encounter, with **MEAT** (Monitor, Evaluate, Assess, Treat) documented by 12/31
- **Visit Modality:** In-person or video telehealth encounters qualify when they support meaningful evaluation of liver synthetic function, portal hypertension markers, and frailty (LFI/CFS)
- **Clinical Context:** Under **HCC V28**, risk weighting is highly sensitive to disease severity Documenting "Cirrhosis without mention of alcohol" (**HCC K72.1**) vs. "Chronic Hepatic Failure with Complications" (HCC **K72.1**) significantly changes the patient's risk profile. Staging severity via **MELD 3.0** and **Child-Pugh** is a clinical imperative to ensure resource allocation matches patient risk
- **Complication Specificity:** VBC accuracy requires documenting active complications. If a patient has **Ascites** (**K76.81**) or **Esophageal Varices** (**I85.10**), these must be coded alongside the cirrhosis to captivate the true complexity of portal hypertension
- **Avoid "Rollover" Documentation:** Do not copy forward a prior year's hepatology note without updating current labs (INR, Bilirubin, Creatinine, Albumin), current MELD score, and the date of the most recent **HCC Surveillance (US/AFP)**

Good Documentation is Comprehensive Coding

INSUFFICIENT DOCUMENTATION	COMPREHENSIVE DOCUMENTATION
"Liver cirrhosis, stable. Continue current medications." (No MELD components, no Child-Pugh class, no frailty score documented)	**MELD 3.0 score** (calculated from INR, bilirubin, creatinine, sodium, sex); **Child-Pugh class (A/B/C); CFS or LFI frailty score; LSM if available (kPa)**; compensation status (compensated vs. decompensated with specific complication: ascites grade, HE grade, recent bleed, etc.)

INSUFFICIENT DOCUMENTATION	COMPREHENSIVE DOCUMENTATION
"NAFLD-related cirrhosis. On appropriate management." (No ICD-10 specificity, no metabolic comorbidity codes, no diagnostic basis noted)	**Document etiology with ICD-10 code: K74.69 + supporting metabolic factors** (**E11.x, E66.x, I10, E78.x**) for MASLD; **K70.30/31** + **F10.x** + documented alcohol quantity/AUDIT-C for alcohol; **K74.3/4/5** for biliary/other; **B18.1/B18.2** for viral. Specify diagnostic basis (imaging findings, serology, clinical history, FIB-4 result, elastography)
"Patient on diuretics and beta-blocker for liver disease. Surveillance ongoing." (No doses, no HCC surveillance date, no variceal or ascites status)	HCC surveillance (**last US + AFP date, results**); **variceal status** (prior EGD date, varices presence, current prophylaxis: carvedilol dose or EVL status); ascites management (diuretic regimen spironolactone/furosemide doses, Na restriction, response); HE: lactulose + rifaximin doses and response (bowel frequency target); SBP prophylaxis (agent + dose if applicable); viral suppression (HCV RNA result, HBV treatment, etc.)
"Diabetes and kidney disease also present. Will monitor." (No ICD-10 codes, no complication staging, no interaction with cirrhosis management documented)	**All comorbidities coded** (CKD stage, T2DM with complications, CVD, obesity, etc.). **Note interaction with cirrhosis management** (e.g., CKD affects diuretic dosing, T2DM affects glucose goals, CVD affects NSBB tolerance). **List medications with high-risk flags** (NSAIDs, benzodiazepines, narcotics — document reason for use or deprescribe plan)
"Patient has had cirrhosis for several years. No significant change in status." (No diagnosis date, no MELD trend, no decompensation timeline, no hospitalization frequency)	**Date of cirrhosis diagnosis** (or 'presumed cirrhosis' if diagnosis predates current record); **etiology onset date if known** (e.g., HCV diagnosis year, alcohol use onset, NASH timeline). MELD trend (is it stable, worsening, or improving?). **Decompensation timeline** (when did ascites appear? first variceal bleed? HE? jaundice?) **Hospitalizations in past 12 months (**count and reasons) — frequency of hospitalization signals high VBC cost/risk

ABBREVIATIONS: CFS = Clinical Frailty Scale; AUDIT-C = Alcohol Use Disorders Identification Test—Concise; CVD = cardiovascular disease; CKD = chronic kidney disease; EGD = esophagogastroduodenoscopy; E11.x = type 2 diabetes codes; E66.x = obesity codes; E78.x = dyslipidemia codes; EVL = endoscopic variceal ligation; F10.x = alcohol use disorder codes; FIB-4 = fibrosis index based on 4 factors; HCC = hepatocellular carcinoma; HCV = hepatitis C virus; HE = hepatic encephalopathy; HRS = hepatorenal syndrome; I10 = hypertension code; ICD-10 = International Classification of Diseases, 10th Edition; INR = international normalized ratio; K74.x = cirrhosis codes; K70.x = alcohol-associated cirrhosis; LFI = Liver Frailty Index; LSM = liver stiffness measurement; MELD = Model for End-Stage Liver Disease; NSAID = nonsteroidal anti-inflammatory drug; NSBB = nonselective beta-blocker; RNA = ribonucleic acid; SBP = spontaneous bacterial peritonitis; T2DM = type 2 diabetes mellitus; VBC = value-based care

EHR Workflow Tips

- **[EHR TIP — Workflow] MELD-3.0 and Staging** The **MELD-3.0 calculator** should auto-populate from the latest Bilirubin, Sodium, INR, Creatinine, and Albumin. Confirm that the mapped Child-Pugh Class (A/B/C) and MELD score match the documented diagnosis before signing to ensure accurate **HCC V28** risk-weighting

- **[EHR TIP — Alert] Surveillance Gap** No **Ultrasound + AFP** resulted in 6 months — configure a hard-stop alert to fire at the point of scheduling for patients with a cirrhosis diagnosis. Surveillance gaps are the leading cause of late-stage HCC detection
- **[EHR TIP — Best Practice] Prophylaxis Advisory** Advisory flags an **NSBB (Carvedilol)** initiation or titration opportunity when a patient has documented varices or a Liver Stiffness Measurement (LSM) >20 kPa and no NSBB is on the med list. Include a secondary check to hold if SBP <90 or HR <55
- **[EHR TIP — Workflow] Frailty Integration Liver Frailty Index (LFI)** calculator embedded in the hepatology referral order — auto-populate grip strength and chair-stand data from nursing intake to generate the 3-month waitlist mortality risk and support transplant referral timing
- **[EHR TIP — Auto-prompt] Etiology Specificity** Trigger an **"Etiology + Status"** prompt when cirrhosis is added to the problem list — reduces unspecified **K74.60** coding by forcing a choice between MASLD (**K74.69**), Alcoholic (**K70.30**), or Viral (**B18.x**) at the source
- **[EHR TIP — Best Practice] HE Adherence Link** Link **Lactulose and Rifaximin** refill requests to an automated **Bristol Stool Scale** patient-portal survey — closing the "HE Adherence Gap" by allowing real-time titration based on stool frequency before the next clinical encounter
- **[EHR TIP — Alert] Nephrotoxin Hard Stop Nephrotoxin Check** — flag and block new **NSAID** orders (Ibuprofen/Naproxen) when a cirrhosis diagnosis with ascites is present. Include a clinical rationale regarding the 4.5x increased risk of **Hepatorenal Syndrome (AKI)** in this population
- **[EHR TIP — Workflow] Pre-Visit Population Health Pre-visit prep:** Pull patients with Cirrhosis + no HCC surveillance or MELD labs in 6 months into a population health worklist. Schedule labs and imaging **2 weeks before** the encounter so results are available for real-time **MEAT** documentation and staging

Brief Case Examples

SUCCESS: SUFFICIENT DOCUMENTATION

Scenario: 68-year-old with MASLD-related cirrhosis and Type 2 Diabetes. MELD-3.0 is 16; labs show Albumin 3.1, INR 1.4, Sodium 134. Most recent imaging shows moderate ascites and a stable 1.8 cm regenerative nodule. Currently on Carvedilol 6.25 mg BID and Spironolactone 100 mg daily.

Documentation: "Decompensated MASLD cirrhosis with moderate ascites (**K74.69 + K76.81**), MELD-3.0 score 16 (Bili 1.8, INR 1.4, Na 134, Cr 1.1 on 5/20/26), Child-Pugh Class B. Type 2 Diabetes (**E11.9**) and Obesity (**E66.01**) managed. HCC surveillance (US/AFP) completed 5/2026—negative for malignancy. Stable on Carvedilol for portal hypertension (HR 62). Refilled Spironolactone for volume control; monitored for AKI risk. MEAT documented."

Outcome: HCC 63 (Chronic Hepatic Failure - 0.962) + HCC 18 (Diabetes with Chronic Complications* - 0.166) Combined RAF 1.128 ≈ $11,730/year in risk-adjusted care resources.

PITFALL: INSUFFICIENT DOCUMENTATION

Documentation as written: "Cirrhosis, unspecified (**K74.60**). History of liver disease. Follow up with Hepatology. Labs reviewed; MELD 16. Continue current medications."

Consequence: K74.60 (unspecified cirrhosis) maps to HCC 64 but fails to document decompensation. Chronic hepatic failure (**K72.10/K72.11**), which maps to HCC 63 (RAF 0.962), is never triggered because ascites, hepatic encephalopathy, and hepatic failure are not coded as active diagnoses. Using Z87.19 instead of an active cirrhosis code zeroes out the HCC entirely. Unlinked MASLD (**K76.0**) and unspecified diabetes (**E11.9**) miss the metabolic complexity — the patient's true acuity remains invisible to the risk model.

RAF Impact: Potential loss of HCC 63 (0.962), coded instead as HCC 64 (0.447) or missed entirely ≈$6,000–$12,000/year in unrepresented care resources depending on segment.

Fix: Decompensated MASLD cirrhosis with moderate ascites (**K74.69 + K76.81**), MELD-3.0 score 16 (Bili 1.8, INR 1.4, Na 134, Cr 1.1 on 5/24/26). Type 2 Diabetes with metabolic complications managed. Ascites evaluated; Carvedilol titrated for portal hypertension prophylaxis. MEAT documented."

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