AAVBC
AMERICAN ACADEMY OF VALUE BASED CARE

Alcoholic Hepatitis

Quick Reference Guide

2026

Table of Contents

1 CLINICAL SNAPSHOT

Definition: Alcoholic hepatitis (AH) is an acute inflammatory liver syndrome caused by prolonged heavy alcohol use, characterized by rapid-onset jaundice, hepatocellular injury, and systemic inflammation. The NIAAA 2016 consensus definition requires jaundice within the prior 8 weeks; ongoing heavy alcohol use (>40 g/day women, >60 g/day men) for **≥6** months with **<60** days of abstinence before jaundice onset; serum bilirubin >3 mg/dL; **AST >50 IU/L** with **AST/ALT ratio >1.5** (both <400 IU/L); and exclusion of competing diagnoses (drug-induced liver injury, viral hepatitis, autoimmune hepatitis, biliary obstruction).[1–3]

ICD-10 Codes: K70.10 (alcoholic hepatitis without ascites) and **K70.11** (alcoholic hepatitis with ascites) are the primary codes — both map to HCC 65 under CMS-HCC V28. **F10.xx** (alcohol use disorder) codes must be sequenced alongside **K70.1x** at every encounter — AUD is the root cause, not a secondary finding.[1] **Do NOT use Z87.891 ('history of alcoholic liver disease') when disease is active with ongoing elevated bilirubin, jaundice, or active treatment — this is the single most common AH coding error**. Avoid **K70.9** (unspecified) when **K70.10 or K70.11** can be supported[4,5]

Prevalence and Burden: Approximately 130,000 to **150,000+ U.S. hospitalizations per year** are directly attributed to AH (with primary diagnosis admissions alone exceeding 60,000 annually). True systemic incidence is even higher due to underdiagnosis of mild-to-moderate cases in the outpatient setting. **In-hospital mortality** for **severe AH (MELD >20) ranges 20–50%** without corticosteroid eligibility assessment. Severity bands: moderate (**MELD ≤20, 10–20%** one-year mortality); severe (**MELD >20, ~20%** 90-day mortality); Maddrey DF ≥32 historically identified **50% 30-day mortality untreated**.[1,2,6] In adults **≥65**, immunosenescence, polypharmacy, and CKD amplify corticosteroid-related infection and AKI risk — **15%** of U.S. adults 60–94 consume alcohol at misuse levels and binge drinking has risen sharply in adults >50 since 2000.[7]

HCC/RAF V28 Mapping

ICD-10 CODE(S)	HCC CATEGORY (V28)	RAF WEIGHT (CNA)	DOCUMENTATION REQUIREMENT
K70.10 (Alcoholic hepatitis without ascites)	**HCC 65**	0.185	Document ascites explicitly ABSENT (physical exam — no shifting dullness, no fluid wave; imaging confirming no ascites, or paracentesis if performed). Active disease supported by bilirubin, **AST/ALT ratio >1.5**, ongoing alcohol use or recent AUD treatment. Annual re-documentation required
K70.11 (Alcoholic hepatitis with ascites)	**HCC 65**	0.185	Document ascites present by physical exam (shifting dullness, fluid wave), imaging (ultrasound/CT), or paracentesis. Active disease markers as for **K70.10**. Ascites is a clinical assessment requirement, not an administrative coding formality

ICD-10 CODE(S)	HCC CATEGORY (V28)	RAF WEIGHT (CNA)	DOCUMENTATION REQUIREMENT
F10.X (Alcohol use disorder with psychotic complications)	**HCC 136**	0.424	**AUD is the root cause** — code at every encounter alongside **K70.1x**, not just at initial diagnosis. **F10.20** = active moderate AUD; **F10.21** = moderate AUD in early remission (verified sobriety only). **Avoid F10.99** (unspecified). — AUD is coded and managed as root cause
F10.X (AUD moderate/severe with non-psychotic symptoms)	**HCC 139**	0.242	**AUD is the root cause** — code at every encounter alongside **K70.1x**, not just at initial diagnosis. **F10.20** = active moderate AUD; **F10.21** = moderate AUD in early remission (verified sobriety only). **Avoid F10.99** (unspecified). — AUD is coded and managed as root cause
K70.40/K70.41 (Alcoholic hepatic failure without/with coma)	**HCC 63**	0.962	**Document HE grade** (**K70.41** — with coma); distinguish from **K70.10/K70.11** when progression to acute liver failure occurs. Hepatic failure criteria (INR >1.5, encephalopathy, coagulopathy) must be recorded. Used when AH progresses beyond hepatitis to failure
K70.31 (Alcoholic cirrhosis with ascites)/K74.60/ K74.69 (Unspecified/ other cirrhosis)	**HCC 64**	0.447	**Add when cirrhosis is documented as concurrent with AH**. **K70.3x** for documented alcoholic cirrhosis; **K74.x** for unspecified/other. AH and cirrhosis may coexist — both are coded when both are present. Add **K76.6** (portal hypertension), **I98.3** (esophageal varices) when clinically documented
Z87.891 (History of alcoholic liver disease)	Non-HCC — flat risk	No HCC	**DO NOT use when the patient has active disease** (elevated bilirubin, jaundice, ongoing treatment). This is the single most common AH coding error flagged[1] Acceptable only after sustained remission with documented sobriety and normalized liver function

ICD-10 CODE(S)	HCC CATEGORY (V28)	RAF WEIGHT (CNA)	DOCUMENTATION REQUIREMENT

ABBREVIATIONS: AH = alcoholic hepatitis; AUD = alcohol use disorder; CMS = Centers for Medicare & Medicaid Services; CNA = community non-dual aged; HCC = hierarchical condition category; HE = hepatic encephalopathy; ICD-10 = International Classification of Diseases, 10th Edition; INR = international normalized ratio; MEAT = monitor/evaluate/assess/treat; RAF = risk adjustment factor; V28 = CMS-HCC model version 28. * Annual risk value illustrative at AAVBC base rate ~$10,402/year; actual plan payment varies by county and contract year. RAF CNA represents community non-dual aged coefficient under the 2024 CMS-HCC V28 model applied at 100% for PY2026; normalization factor 1.067

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

Risk-Adjusted Care Resources per Patient/Year

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

Alcoholic hepatitis with or without ascites	Alcoholic use disorder (without psychotic features)	Alcoholic use disorder (with psychotic features)	Alcoholic hepatic failure	Alcoholic liver disease
$1.9K	**~$4.4K**	**~$2.5K**	**$10K**	**$4.6K**
HCC 65 · RAF 0.185	HCC 136 · RAF 0.424	HCC 139 · RAF 0.242	HCC 63 · RAF 0.962	HCC 64 · RAF 0.447

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

Medicare Screenings and Diagnostic Tests

The primary care setting serves as the foundational clinical portal for identifying alcohol-associated hepatitis.[1,2,8]

TEST	COVERAGE	FREQUENCY	CPT CODE	NOTES
AUDIT-C/ NIAAA single-question screener	Medicare Part B	Annually at every E/M visit; at AWV; at each hospitalization	G0442 (annual screen 15 min); 96160 (standardized instrument)	Positive screen → brief intervention + referral within 2 months (Rate 2). AUDIT-C ≥4 (men)/≥3 (women) = positive
Brief behavioral counseling for unhealthy alcohol use	Medicare Part B (USPSTF Grade B)	Up to 4 sessions per 12 months after positive screen	G0443 (15 min brief face-to- face counseling)	G0443 coverage requires a positive G0442 screen. Motivational interviewing framework; non-judgmental, non-shaming language. Document counseling content, goals, and patient response
Liver function panel (AST, ALT, bilirubin, alkaline phosphatase, albumin, INR)	Medicare Part B — as medically necessary	At AUD diagnosis; every 6–12 months if active AUD; urgently if jaundice, RUQ pain, or altered mental status	80076 (hepatic function panel); 85610 (INR)	AST/ALT ratio >1.5–2 with AST <400 IU/L + bilirubin >3 mg/dL + jaundice onset <8 weeks = AH until proven otherwise. **DO NOT simply advise 'cut back on alcohol'**
MELD/MELD-Na score calculation	Integrated into E/M; calculator-based	At every AH encounter; recalculate day 7 and with clinical change	Calculated metric — no separate CPT	MELD is the ACG 2024 preferred prognostic tool over Maddrey DF. **MELD >20 = severe AH → corticosteroid eligibility assessment, hospitalize, hepatology consult**. MELD-Na adjusts for hyponatremia — use when available. A patient with MELD 18 today can reach MELD 28 in 72 hours
Infection surveillance (blood, urine, ascitic fluid cultures; procalcitonin)	Medicare Part B — medically necessary in severe AH	At admission; with any clinical deterioration; before, during, and after corticosteroid therapy	87040 (blood culture); 87086/87088 (urine); 87070 (ascitic fluid); 84145 (procalcitonin)	Infection surveillance is mandatory before corticosteroids — corticosteroid-related infection is associated with 2.46× increased 90-day mortality. **Do NOT give prophylactic antibiotics universally** (strong rec against, ACG 2024)

TEST	COVERAGE	FREQUENCY	CPT CODE	NOTES
Renal function (creatinine, BUN, urine output, urinalysis)	Medicare Part B — as medically necessary	Daily while hospitalized; each outpatient visit in active AH; within 1–2 weeks of any nephrotoxin change	80053 (CMP); 81000/81001 (UA)	**AKI is present in up to 1/3 of severe AH** patients and is associated with a 9-fold increase in 90-day mortality. **Rising creatinine or oliguria → stop nephrotoxins, IV albumin, evaluate hepatorenal syndrome**: nephrotoxin avoidance is a life-or-death drug management decision

ABBREVIATIONS: AH = alcoholic hepatitis; AKI = acute kidney injury; ALT = alanine aminotransferase; AST = aspartate aminotransferase; AUD = alcohol use disorder; AUDIT-C = Alcohol Use Disorders Identification Test — Consumption; AWV = annual wellness visit; BUN = blood urea nitrogen; CMP = comprehensive metabolic panel; ECDS = Electronic Clinical Data Systems; E/M = evaluation and management; HCPCS = Healthcare Common Procedure Coding System; HEDIS ASF = Unhealthy Alcohol Use Screening and Follow-Up; INR = international normalized ratio; LOINC = Logical Observation Identifiers Names and Codes; MELD = Model for End-Stage Liver Disease; MELD-Na = MELD with sodium; NIAAA = National Institute on Alcohol Abuse and Alcoholism; RUQ = right upper quadrant; SNOMED = Systematized Nomenclature of Medicine; UA = urinalysis; USPSTF = US Preventive Services Task Force
*__Consult FDA labels for the most up-to-date dosage information, contraindications, and drug-drug interactions__

Subtle Early Signs in Adults

Alcoholic hepatitis does not present itself with a single symptom, it often manifests itself through a pattern of findings. The following table maps the cardinal presentations to their required clinical actions, ensuring that each red-flag finding triggers the correct diagnostic pathway, severity assessment.[9]

SIGN/SYMPTOM	CLINICAL SIGNIFICANCE
Jaundice onset (<8 weeks) with heavy alcohol use	Cardinal presentation meeting NIAAA criteria same-day evaluation required **MELD >20 mandates admission**
Tender hepatomegaly, fever, leukocytosis, and RUQ pain	**Classic triad of severe AH perform infection surveillance before starting corticosteroids**
New or worsening ascites; increased abdominal girth	Signals **K7011** upgrade paracentesis triggers SBP evaluation empiric antibiotics for fever or pain
Altered mental status, asterixis, or confusion	Suggests hepatic encephalopathy or progression to hepatic failure (**K7041**) **distinguish from withdrawal or Wernicke-Korsakoff**
GI bleeding (hematemesis, melena, or new anemia)	Contraindicates corticosteroids emergency endoscopy alters management due to high short-term mortality
Oliguria, rising creatinine, or worsening INR	Suggests Hepatorenal Syndrome (HRS) **stop nephrotoxins initiate albumin/vasoconstrictors mortality exceeds 60% without intervention**

SIGN/SYMPTOM	CLINICAL SIGNIFICANCE

ABBREVIATIONS: AH = alcoholic hepatitis; CIWA-Ar = Clinical Institute Withdrawal Assessment — Alcohol, revised; GI = gastrointestinal; HE = hepatic encephalopathy; HRS = hepatorenal syndrome; INR = international normalized ratio; PMN = polymorphonuclear leukocyte; RUQ = right upper quadrant; SBP = spontaneous bacterial peritonitis; SIRS = systemic inflammatory response syndrome

Geriatric Risk Factors

In older adults with alcoholic hepatitis, age-related immune decline, baseline renal vulnerability, and polypharmacy can interact to amplify both disease severity and treatment-related complications.[3,8,10,11]

FACTOR	RISK SIGNAL	NOTES
Older adult with AH (≥65)	Immunosenescence amplifies corticosteroid-related infection risk (**OR 1.93** (95% CI 1.27–2.92)); baseline AKI-predisposing comorbidities (CKD, diabetes, cardiovascular disease) more common; polypharmacy drives nephrotoxin exposure	Pre-corticosteroid checklist is mandatory (infection surveillance, glucose control, GI bleeding exclusion, HBV screening). Frailty assessment (Clinical Frailty Scale) guides whether transplant evaluation is appropriate
AUD pharmacotherapy in patients ≥65	naltrexone preferred first-line; baclofen *(off-label) requires lower starting dose; acamprosate limited by TID dosing and renal contraindications; disulfiram absolutely contraindicated in ALD	naltrexone 50 mg/day PO or 380 mg IM monthly — safe in advanced liver disease; check LFTs before and during. baclofen start 5 mg TID in elderly (not full 10 mg TID); titrate slowly; CNS sedation risk. **Do NOT wait for addiction specialist to initiate**
Nephrotoxin polypharmacy (NSAIDs, ACEi/ARB, IV contrast, aminoglycosides)	Elderly at higher baseline AKI risk; AKI in severe AH associated with **9-fold increase in 90-day mortality**; iatrogenic AKI is frequently preventable **AKI prevalence 27–53% in hospitalized decompensated cirrhosis**	Stop all nephrotoxins at admission; substitute acetaminophen (≤2 g/day) for pain; do not resume without hepatology/nephrology review. Medication reconciliation at every transition of care
Benzodiazepines for alcohol withdrawal in older adults	Can precipitate or worsen hepatic encephalopathy; HE and alcohol withdrawal overlap clinically — difficult to distinguish **5-fold increased risk of first-time HE during days 3–10 of new benzodiazepine use in cirrhosis**	CIWA-Ar-guided symptom-triggered dosing (not fixed schedules); shorter-acting agents (oxazepam, lorazepam) preferred over diazepam in cirrhosis; avoid topiramate for AUD if HE present

FACTOR	RISK SIGNAL	NOTES
Nutritional deficiency + sarcopenia	Sarcopenia nearly universal in severe AH; thiamine deficiency, zinc, folate, B12 common; under-nutrition worsens outcomes **Sarcopenia prevalence 80% in decompensated alcohol-associated cirrhosis (vs. ~60% other etiologies)**	Target 35–40 kcal/kg/day; 1.2–1.5 g protein/kg/day (**do NOT restrict protein — worsens sarcopenia and HE**); thiamine 100 mg IV/IM BEFORE any glucose; oral supplements first, enteral if oral intake insufficient

ABBREVIATIONS: ACEi = angiotensin-converting enzyme inhibitor; AH = alcoholic hepatitis; AKI = acute kidney injury; ALD = alcohol-associated liver disease; ARB = angiotensin receptor blocker; AUD = alcohol use disorder; CIWA-Ar = Clinical Institute Withdrawal Assessment — Alcohol, revised; CKD = chronic kidney disease; CNS = central nervous system; HE = hepatic encephalopathy; LFT = liver function test; NSAID = nonsteroidal anti-inflammatory drug; SDOH = social drivers of health; VBC = value-based care.

Diagnostic Thresholds

Alcoholic hepatitis is not a single-severity diagnosis — it spans a spectrum from moderate disease manageable in the outpatient setting to fulminant hepatic failure with ICU-level mortality, and the difference between these trajectories is defined by the validated scoring/diagnostic systems noted below that primary care physicians should document.[2,8,10]

TEST/MARKER	DIAGNOSIS CRITERION	NOTES
NIAAA 2016 consensus diagnostic criteria	Jaundice onset within prior 8 weeks; heavy alcohol use (>40 g/d women, >60 g/d men) ≥6 months with <60 days abstinence; **Bilirubin** >3 mg/dL; **AST** >50 IU/L with AST/ALT ratio >1.5 (both <400); exclusion of competing diagnoses	If all criteria met with no competing cause, label 'definite AH' without biopsy; **liver biopsy reserved for atypical features** or trial enrollment
MELD/MELD-Na (preferred per ACG 2024)	MELD = 3.78 × ln(bilirubin) + 11.2 × ln(INR) + 9.57 × ln(creatinine) + 6.43; MELD-Na adjusts for serum sodium <135. MELD >20 = **severe AH → corticosteroid eligibility**	Calculate at admission, recalculate day 7 and with clinical change. **MELD is superior to Maddrey DF** in contemporary AH cohorts for short-term mortality prediction
Maddrey Discriminant Function (mDF)	mDF = 4.6 × (PT_patient − PT_control) + bilirubin (mg/dL); ≥32 historically = severe AH and corticosteroid candidate	AASLD 2020/AGA 2017 still reference mDF; ACG 2024 recommends **MELD as primary**. Either threshold (MELD >20 OR mDF ≥32) identifies corticosteroid candidates

TEST/MARKER	DIAGNOSIS CRITERION	NOTES
Lille score (day 7 response to corticosteroids)	Lille = function of age, albumin, bilirubin day 0 + day 7, creatinine, PT. Lille <0.45 = responder (continue steroids); Lille ≥0.45 = non-responder (discontinue steroids)	Lille day 7 is the decision point for continuation vs discontinuation — dogmatic 28-day courses are not evidence-based in non-responders. **Early transplant evaluation for non-responders with MELD >26**
Infection workup	Blood, urine, and ascitic fluid cultures at admission + with any clinical deterioration; procalcitonin to differentiate sterile SIRS from infection-associated SIRS	Rule out infection BEFORE corticosteroid initiation — corticosteroid-related infection is associated with 2.46× increased 90-day mortality. **Do NOT give prophylactic antibiotics universally**

ABBREVIATIONS: AASLD = American Association for the Study of Liver Diseases; ACG = American College of Gastroenterology; AFP = alpha-fetoprotein; AGA = American Gastroenterological Association; AH = alcoholic hepatitis; ANA = antinuclear antibody; ASMA = anti-smooth muscle antibody; AST/ALT = aspartate/alanine aminotransferase; HBcIgM = hepatitis B core IgM; HBsAg = hepatitis B surface antigen; HCC = hepatocellular carcinoma; HCV = hepatitis C virus; IgG = immunoglobulin G; INR = international normalized ratio; mDF = Maddrey discriminant function; MELD = Model for End-Stage Liver Disease; MELD-Na = MELD with sodium; NIAAA = National Institute on Alcohol Abuse and Alcoholism; PT = prothrombin time; SIRS = systemic inflammatory response syndrome
*__Consult FDA labels for the most up-to-date dosage information, contraindications, and drug-drug interactions__

CLINICAL PEARL

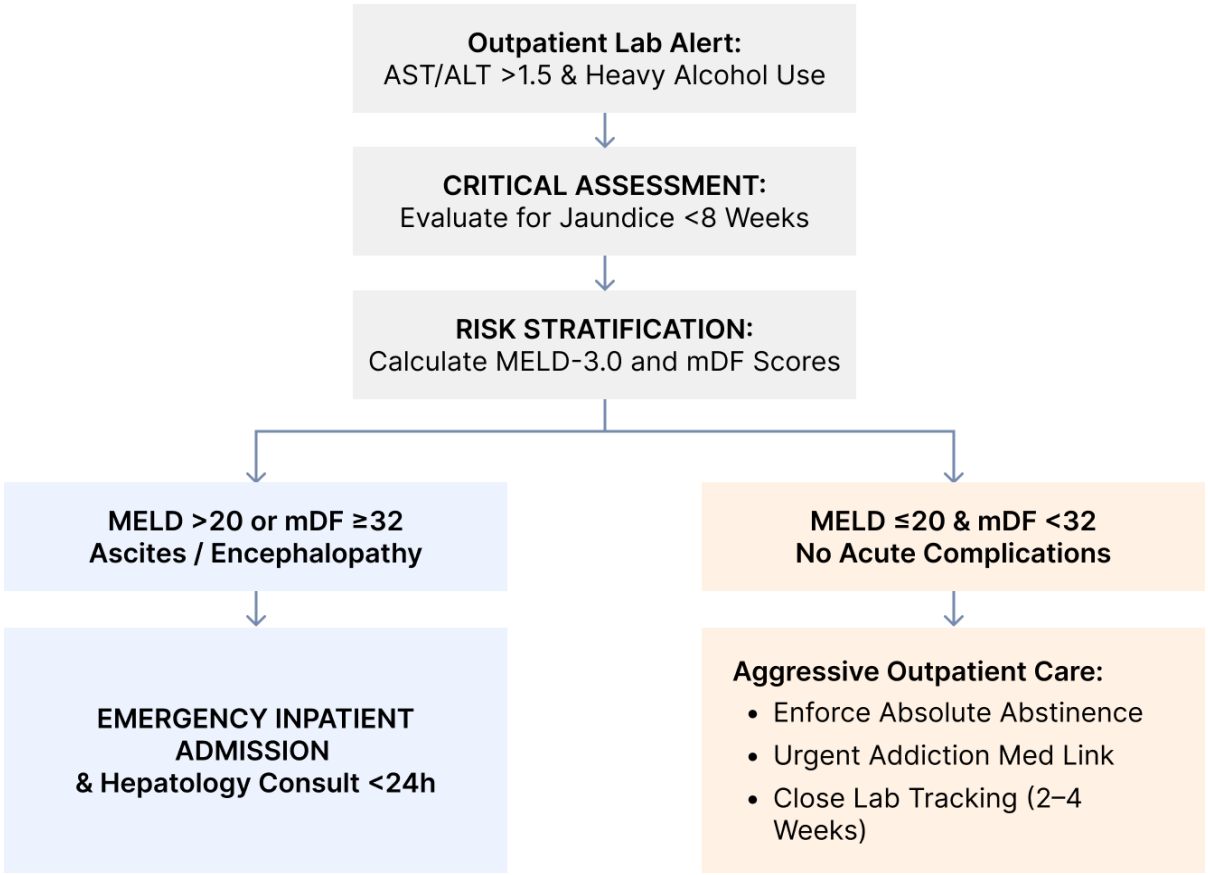
Abnormal transaminases and a high AST/ALT (2:1 or 1.5:1) ratio are merely initial indicators, not a definitive diagnosis. Counseling a patient to simply "cut back" is a dangerously inadequate response to an acute medical emergency. The PCP must recognize rapid onset of jaundice and do a thorough investigation of other signs and symptoms which may include: persistent fatigue, unexplained weight loss, itchy skin, fluid buildup in the abdomen or legs, and easy bruising or bleeding. Digestive issues like nausea, pale stools, and dark urine are also common.

AAVBC PERSPECTIVE

From a value-based care (VBC) perspective, alcoholic hepatitis represents one of the highest-impact conditions for primary care intervention. ***Primary care is uniquely positioned to intervene at every stage of the AH trajectory:***

- ***Upstream prevention:*** *AUDIT-C screening with FIB-4 coupling identifies at-risk patients before decompensation — screening with noninvasive tests for fibrosis detection is cost-effective*
- ***AUD pharmacotherapy initiation****: Naltrexone or other FDA indicated alternatives can be started in primary care without waiting for addiction specialty — pharmacotherapy in ALD cirrhosis is cost-effective through reduced decompensation and readmission*

- ***Post-discharge continuity:*** *Medication reconciliation (nephrotoxin avoidance, withdrawal management), AUD treatment continuation, nutritional rehabilitation, and hepatic surveillance coordination*
- ***Comorbidity management:*** *Diabetes, CKD, malnutrition, and metabolic syndrome — each independently coded and managed — contribute to both clinical outcomes and risk adjustment accuracy*



Common Oversights

Below are a list of common clinical oversights for clinicians to pay close attention to:[3,12]

OVERSIGHT/SHORTCUT	WHY IT MATTERS — WHAT TO DO INSTEAD
Treating abnormal LFTs with 'cut back on alcohol' counseling only, without MELD or severity assessment	AH is a clinical syndrome, not a lab abnormality. When AST/ALT >1.5 coexists with jaundice or ascites, immediate bilirubin/INR/ creatinine /MELD are required. 'Wait and watch' converts a treatable syndrome into a 50% mortality course. Calculate MELD; **MELD >20 → hospitalize, hepatology, corticosteroid eligibility assessment**

OVERSIGHT/SHORTCUT	WHY IT MATTERS — WHAT TO DO INSTEAD
Initiating corticosteroids without infection surveillance and contraindication check	**Corticosteroid-related infection is associated with 2.46× increased 90-day mortality**. Mandatory pre-initiation: blood + urine + ascitic fluid cultures, procalcitonin, HBsAg, glucose, GI bleeding history review, renal function. Active infection, uncontrolled diabetes, GI bleeding, untreated HBV, severe renal failure are contraindications
Leaving Lille score uncalculated at day 7 of corticosteroids	**Lille day 7 is the decision point** — ≥0.45 = non-responder → discontinue corticosteroids; <0.45 = responder → continue 28-day course. Dogmatic 28-day treatment in non-responders exposes patients to adverse effects without benefit. Non-responders with **MELD >26 → early transplant evaluation**
Failing to stop nephrotoxins on admission (NSAIDs, ACEi/ARB, IV contrast, aminoglycosides)	AKI in severe AH carries a 9-fold increase in 90-day mortality and is frequently iatrogenic. **Stop ALL nephrotoxins at admission; substitute acetaminophen (≤2 g/day) for analgesia**; do not resume without hepatology/nephrology review
Treating AUD as a secondary finding — waiting for addiction specialist before initiating pharmacotherapy	**Only 14% of ALD patients receive any AUD treatment; <1% receive pharmacotherapy.** AUD is the root cause — **F10.xx** codes belong at every encounter alongside **K70.1x**

ABBREVIATIONS: ACEi = angiotensin-converting enzyme inhibitor; AH = alcoholic hepatitis; AKI = acute kidney injury; ALD = alcohol-associated liver disease; ARB = angiotensin receptor blocker; AST/ALT = aspartate/alanine aminotransferase; AUD = alcohol use disorder; GI = gastrointestinal; HBsAg = hepatitis B surface antigen; HBV = hepatitis B virus; HCC = hierarchical condition category; IV = intravenous; LFT = liver function test; MELD = Model for End-Stage Liver Disease; NSAID = nonsteroidal anti-inflammatory drug; PCP = primary care provider; TID = three times daily

Key Differentials in Elderly

Several conditions may mimic the signs and symptoms present in alcoholic hepatitis, therefore the clinician must conduct a thorough investigation.[2]

PRESENTATION	DIFFERENTIAL	KEY TESTS
Jaundice + elevated transaminases in a patient with heavy alcohol use	AH **vs.** viral hepatitis (HBV, HCV), drug-induced liver injury (DILI), biliary obstruction, autoimmune hepatitis	HBsAg + anti-HCV + anti-HAV IgM; acetaminophen level + detailed drug/supplement history; RUQ ultrasound with Doppler; ANA + ASMA + IgG (autoimmune screen). AST/ALT ratio >1.5 with both <400 IU/L favors AH
Altered mental status in AH patient	Hepatic encephalopathy (**K70.41**) **vs.** alcohol withdrawal (**F10.230**) **vs.** Wernicke-Korsakoff (**E51.2**) vs benzodiazepine effect vs CNS infection/bleed	CIWA-Ar; ammonia (modest utility); **thiamine 100 mg IV BEFORE glucose**; LP/CT if focal findings or no improvement; medication reconciliation; asterixis — classic HE sign

PRESENTATION	DIFFERENTIAL	KEY TESTS
New ascites in AH patient	**K70.11 (AH with ascites)** ± SBP **vs. K70.31 (alcoholic cirrhosis with ascites)** vs portal vein thrombosis vs malignant ascites vs cardiac ascites	Diagnostic paracentesis (cell count, culture, albumin for SAAG, cytology); ultrasound with Doppler; echo if cardiac etiology suspected; **SAAG ≥1.1 = portal hypertension; PMN >250/µL = SBP**
AKI in AH patient	Pre-renal (volume depletion) **vs.** acute tubular necrosis (nephrotoxin, sepsis) vs hepatorenal syndrome (HRS) vs intrinsic renal disease	Urine sodium, FeNa, urine microscopy; volume challenge with 1 g/kg albumin × 48 h — if no response and no other cause, HRS likely; stop nephrotoxins; terlipressin (where available) or norepinephrine + albumin
GI bleeding in AH patient	Esophageal varices **vs.** gastric varices vs portal hypertensive gastropathy vs peptic ulcer vs Mallory-Weiss tear	**Emergency endoscopy within 12 h**; PPI; octreotide or terlipressin; ceftriaxone prophylaxis; band ligation for varices; corticosteroids contraindicated during active bleed

ABBREVIATIONS: AH = alcoholic hepatitis; AKI = acute kidney injury; ANA = antinuclear antibody; ASMA = anti-smooth muscle antibody; CIWA-Ar = Clinical Institute Withdrawal Assessment — Alcohol, revised; CNS = central nervous system; CT = computed tomography; DILI = drug-induced liver injury; FeNa = fractional excretion of sodium; HBsAg/HCV = hepatitis B surface antigen/C virus; HE = hepatic encephalopathy; HRS = hepatorenal syndrome; IgG = immunoglobulin G; LP = lumbar puncture; PMN = polymorphonuclear leukocyte; PPI = proton pump inhibitor; RUQ = right upper quadrant; SAAG = serum–ascites albumin gradient; SBP = spontaneous bacterial peritonitis; SIRS = systemic inflammatory response syndrome; UTI = urinary tract infection

Comorbidity Screening

Alcoholic hepatitis is not a standalone diagnosis, it is the acute inflammatory crisis on a foundation of chronic, interacting conditions that each independently drive mortality, readmission, and resource utilization.[8,13]

CONDITION	PREVALENCE/ ASSOCIATION	SCREENING APPROACH
Alcohol use disorder (F10.10–F10.29) — root cause	Near-universal in AH; only **14%** of ALD patients receive any AUD treatment, **<1%** pharmacotherapy. Untreated AUD drives 90-day readmission	**AUDIT-C at every visit (**HEDIS ASF); brief intervention + structured referral within 2 months of positive screen; initiate naltrexone or baclofen in primary care without waiting for specialty
Cirrhosis (K74.60/K74.69/ K70.31) — AH and cirrhosis may coexist	Approximately **50%** of severe AH patients have underlying cirrhosis; HCC surveillance mandatory in cirrhosis	**Ultrasound ± AFP every 6 months**; endoscopy for varices per Baveno; Child-Pugh and MELD for staging; code both K70.1x AND K70.31/K74.6x when present

CONDITION	PREVALENCE/ ASSOCIATION	SCREENING APPROACH
Malnutrition + sarcopenia (E44.x, M62.84)	Present in nearly all severe AH; protein-calorie malnutrition, sarcopenia, thiamine/folate/zinc deficiencies	Target 35–40 kcal/kg/day; 1.2–1.5 g protein/kg/day; **thiamine 100 mg IV BEFORE glucose**; zinc + folate + B12; enteral feeding if oral intake <35 kcal/kg; avoid protein restriction
Diabetes mellitus (E11.x)/ metabolic syndrome	Pre-existing diabetes common; corticosteroids exacerbate hyperglycemia; uncontrolled DM is a corticosteroid contraindication	Baseline HbA1c, **fasting glucose; control diabetes BEFORE corticosteroid initiation**; close glucose monitoring during therapy; adjust antidiabetic agents as needed
Chronic kidney disease (N18.x)	CKD common in elderly AH patients; amplifies AKI risk; AKI in severe AH carries 9-fold 90-day mortality	Baseline Cr/eGFR; **stop nephrotoxins;** urine microscopy if AKI; HRS protocol (albumin + vasoconstrictor) **when volume-refractory; nephrology early in severe disease**

ABBREVIATIONS: AFP = alpha-fetoprotein; AH = alcoholic hepatitis; AKI = acute kidney injury; ALD = alcohol-associated liver disease; AUD = alcohol use disorder; AUDIT-C = Alcohol Use Disorders Identification Test — Consumption; Baveno = Baveno consensus on portal hypertension; BID = twice daily; CKD = chronic kidney disease; DM = diabetes mellitus; eGFR = estimated glomerular filtration rate; HbA1c = hemoglobin A1c; HCC = hepatocellular carcinoma; HEDIS ASF = Unhealthy Alcohol Use Screening and Follow-Up; HRS = hepatorenal syndrome; MELD = Model for End-Stage Liver Disease. ***Consult FDA labels for the most up-to-date dosage information, contraindications, and drug-drug interactions**

Staging/Severity Matrix

Staging and severity assessment is the single most consequential clinical decision point in alcoholic hepatitis — it determines whether a patient receives supportive care alone or requires corticosteroid eligibility assessment, ICU-level monitoring, and hepatology consultation.[14,15]

STAGING SYSTEM	FORMULA	CLINICAL USE | ESTIMATED OUTCOMES	DOCUMENTATIO N
MELD/MELD-Na (ACG 2024 preferred)	MELD = 3.78 × ln(bili) + 11.2 × ln(INR) + 9.57 × ln(Cr) + 6.43; MELD-Na adjusts for Na <135	Admission + day 7 + with clinical change; drives corticosteroid eligibility and transplant discussion	'AH, MELD 24 at admission, MELD 28 at day 7 (K70.11); severe AH per ACG 2024 threshold’

CLINICAL PEARL: MELD SCORE AND MELD NA

MELD = 3.78 × ln(bili) + 11.2 × ln(INR) + 9.57 × ln(Cr) + 6.43
MELD-Na = MELD + 1.59 × (135 - Na),* *sodium capped between 120-135 mEq/L
MELD score is calculated using serum bilirubin, serum creatinine, and International Normalized Ratio (INR). Scores range from 6 to 40. MELD-Na also accounts for serum sodium which assesses fluid balance and severity of portal hypertension. **A higher score indicates more severe liver disease and a higher priority on the transplant list.**

MELD-Na Score Severity Staging and Clinical Priorities

The table below categorizes MELD-Na score ranges by severity tier, providing the clinical implications and primary management focuses for each level. The MELD-Na score is a highly validated, objective triage tool used primarily to predict 30-day and 90-day mortality in patients with end-stage liver disease.[16]

Estimated Mortality

- Low/Mild Severity **(MELD-Na ≤ 9): ~1.9% 90-day** mortality **| ~7% 1-year** mortality
- Moderate Severity **(MELD-Na 10–19): ~6.0%** 90-day mortalit**y | ~10–20%** 1-year mortality
- Severe Severity **(MELD-Na 20–29): ~19.6%** 90-day mortality **| ~30%** 1-year mortality
- Very Severe Severity **(MELD-Na 30–39): ~52.6%** 90-day mortality **| ~60%+** 1-year mortality
- Extreme/Maximum Severity **(MELD-Na ≥ 40): ~71.3%** 90-day mortality **| > 80%** 1-year mortality

Note: The estimated mortality figures above capture the exponential risk curve of advanced liver disease. This staging prevents the dangerous clinical underestimation of mortality that can occur when managing patients whose MELD-Na scores are rapidly rising into the 30s or 40s.

Core Clinical Indications (When to Use):

- **Chronic Liver Disease Decompensation:** Calculate MELD-Na immediately when a patient with known cirrhosis presents with new or worsening signs of decompensation: Ascites (fluid accumulation in the abdomen)
- **Hepatic Encephalopathy (HE)** (altered mental status, asterixis)
- **Variceal Bleeding (**gastrointestinal hemorrhage from portal hypertension)
- **Spontaneous Bacterial Peritonitis (SBP)Acute-on-Chronic Liver Failure (ACLF):** Use it during acute hospital admissions to track organ dysfunction over time. A rapidly rising MELD-Na score is a critical red flag for impending multi-organ failure
- **Liver Transplant Referral Timing:** The definitive threshold for initiating a formal liver transplant evaluation is a MELD-Na score >15
- **Prioritizing Organ Allocation:** Utilize the MELD-Na score as the primary metric to rank candidates on the liver transplant waitlist ("sickest first" utility model)

MELD-NA SCORE RANGE	SEVERITY TIER	CLINICAL IMPLICATION AND PROGRESSION 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

ABBREVIATIONS: MELD-Na = Model for End-Stage Liver Disease Sodium

Other Staging Criteria

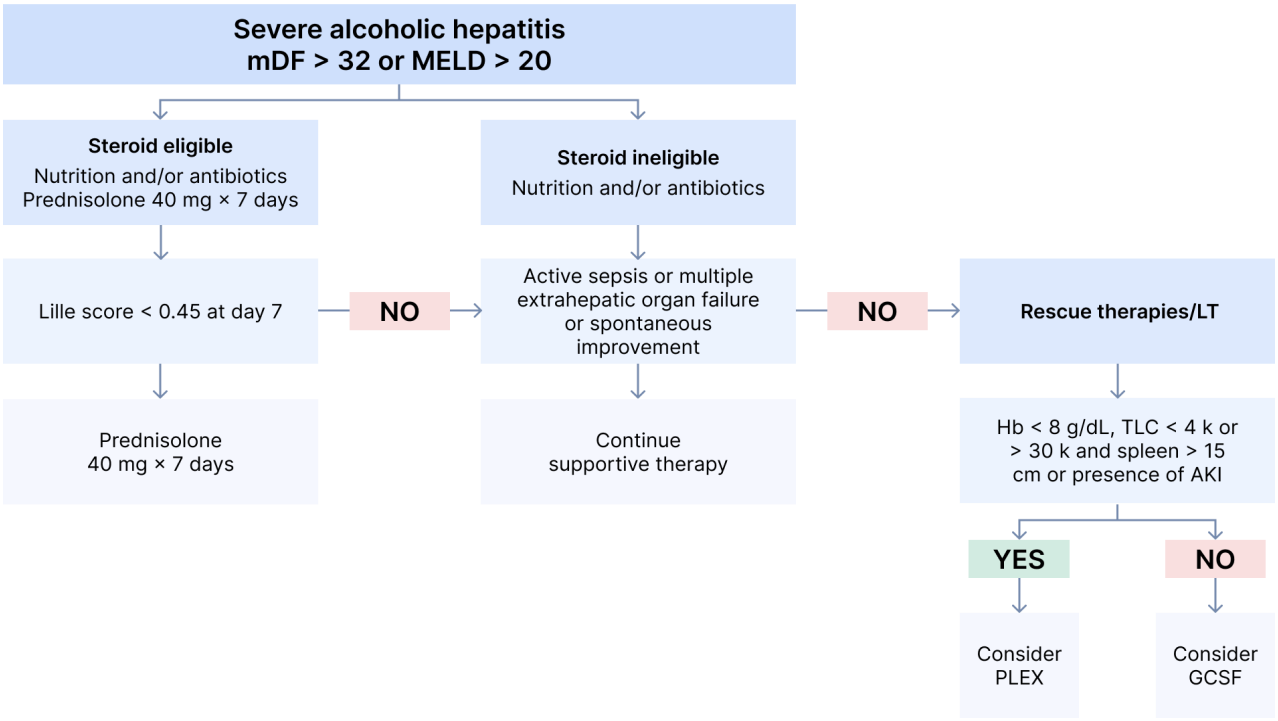
STAGING SYSTEM	CRITERIA/VALUES | ESTIMATED OUTCOMES	CLINICAL USE
Maddrey Discriminant Function[8]	mDF = 4.6 × (PT − control PT) + bilirubin (mg/dL); **≥32 = severe AH** **Estimated outcomes:** mDF ≥32 untreated 30-day mortality ~50%; MELD correlates better in contemporary cohorts	Legacy threshold; Initial severity screen **mDF ≥32 → severe AH**; consider inpatient care, infection workup, nutrition support, and hepatology input. Pair with MELD

STAGING SYSTEM	CRITERIA/VALUES | ESTIMATED OUTCOMES	CLINICAL USE
Lille score (day 7 corticosteroid response)[3]	Function of age, albumin, bilirubin day 0 + day 7, Cr, PT; **<0.45 = responder, ≥0.45 = non-responder** **Estimated outcomes:** Non-responders have 6-month mortality 70–80% without transplant	Day 4–7 of corticosteroid therapy; binary decision for continuation vs discontinuation and care plan reassessment (**MELD >26 or rising) → hepatology urgent**
Hepatorenal/ AKI staging (AASLD 2020)[17]	ICA-AKI stage 1 (Cr rise ≥0.3 within 48 h or **1.5–2× baseline**); stage 2 (**2–3× baseline**); stage 3 (**>3× baseline** or Cr ≥4 mg/dL or RRT) **Estimated outcomes:** AKI present in ~1/3 severe AH; associated with 9-fold increase in 90-day mortality	**AKI in AH patient;**Use with any creatinine rise or oliguria. Triggers nephrotoxin withdrawal, albumin/ volume assessment, infection workup, and nephrology/hepatology coordination
Clinical Frailty Scale (OA AH ≥65)[11]	CFS 1 (fit) → 9 (terminally ill) ≥5 = frail	Use in adults ≥65 to guide treatment intensity, discharge needs, nutrition/ PT/OT, caregiver support, and transplant discussions; age alone does not predict mortality and readmission
Child-Pugh (when cirrhosis concurrent)[8]	A (5–6 points)/B (7–9)/C (10–15); bili, albumin, INR, ascites, HE. **Estimated outcomes:** Child C 1-year survival ~45%; Child A ~90%	**Cirrhosis staging** when K70.31/ K74.6x concurrent with AH. Clarifies hepatic reserve, decompensation risk, surveillance needs, medication safety, and referral urgency

ABBREVIATIONS: AASLD = American Association for the Study of Liver Diseases; ACG = American College of Gastroenterology; AGA = American Gastroenterological Association; AH = alcoholic hepatitis; AKI = acute kidney injury; CFS = Clinical Frailty Scale; Child-Pugh = liver disease severity score; Cr = creatinine; HE = hepatic encephalopathy; HRS = hepatorenal syndrome; ICA-AKI = International Club of Ascites acute kidney injury; INR = international normalized ratio; mDF = Maddrey discriminant function; MELD = Model for End-Stage Liver Disease; MELD-Na = MELD with sodium; OA = older adult; PT = prothrombin time; RRT = renal replacement therapy; VBC = value-based care

Protocol for management of severe alcoholic hepatitis

Steroids are the mainstay of treatment. Response to steroids is assessed at day 7 by Lille score. Steroid non-responder or steroid-ineligible can be considered for rescue therapies.



ABBREVIATIONS: SAH: Severe alcoholic hepatitis; LT: Liver transplant; MELD: Model for end stage liver disease; mDF: Maddrey's discriminant function; TLC: Total leucocyte count; AKI: Acute kidney injury; PLEX: Plasma exchange; GCSF: Granulocyte colony stimulating factor

⚠ RED FLAG – EMERGENCY | CALL 911 OR ACTIVATE ED TRANSFER

- Active variceal hemorrhage (hematemesis, hemodynamic compromise)
- Grade III–IV hepatic encephalopathy with airway compromise
- Acute liver failure (INR >1.5 + encephalopathy + bilirubin rapidly rising)
- Severe sepsis with hypotension despite fluid
- Hepatorenal syndrome with oliguria
- Complete obstructive jaundice with cholangitis
- Acute alcohol withdrawal with seizures or delirium tremens

⚠ RED FLAG – URGENT/RAPID EVALUATION (WITHIN 24–72 HOURS)

- New jaundice + heavy alcohol use + bilirubin >3 mg/dL (calculate MELD same day)
- MELD >20 or mDF ≥32 (hospitalize + hepatology)
- New ascites (paracentesis + SBP workup)
- Rising creatinine or oliguria (nephrotoxin stop + albumin)
- Lille ≥0.45 at day 7 (discontinue steroids + transplant evaluation if MELD >26)
- Fever + leukocytosis on corticosteroids (pan-culture + procalcitonin + reassess steroids)
- Discharge from hospitalization for AH without AUD pharmacotherapy (initiate naltrexone or baclofen before discharge)
- Alcohol relapse after period of abstinence

3 MEAT DOCUMENTATION ESSENTIALS

Case Vignette: A 58-year-old man with a 20-year history of heavy alcohol use (8–10 drinks/day) presents with 3 weeks of progressive jaundice, RUQ pain, and increasing abdominal girth. He was seen at urgent care 10 days ago and told to "cut back on drinking." Labs: AST 220, ALT 98 (ratio 2.2), total bilirubin 9.4, INR 1.8, creatinine 1.3, sodium 131, albumin 2.6. AUDIT-C score 10. Abdominal ultrasound shows hepatomegaly with moderate ascites and no biliary obstruction. MELD-Na 26. No prior AUD pharmacotherapy"

MONITOR: Alcohol use: active heavy use, 8–10 drinks/day × 20 years; AUDIT-C 10 (positive); last drink 48 hours ago. CIWA-Ar 8 — symptom-triggered benzodiazepine protocol initiated (lorazepam preferred given liver disease). Weight: 94 kg (baseline for nutritional targets). Vitals: HR 104, BP 108/62, T 38.1°C. MELD-Na 26 (severe AH) — recalculate at 48–72 hours and day 7. Infection surveillance: blood cultures, urine culture, diagnostic paracentesis (cell count, culture, albumin) obtained before any corticosteroid consideration. Procalcitonin pending. Glucose: fasting 142 — **monitor closely if corticosteroids initiated.**

EVALUATE: Hepatic function panel (date): AST 220, ALT 98 (AST/ALT ratio 2.2), total bilirubin 9.4, alkaline phosphatase 188, albumin 2.6. INR 1.8. Creatinine 1.3, sodium 131. **MELD-Na calculated:** 26 (severe). **Maddrey DF calculated:** 42 (≥32 = severe). **NIAAA criteria met:** jaundice onset <8 weeks, heavy alcohol use >60 g/day for ≥6 months, bilirubin >3, AST >50 with AST/ALT >1.5 (both <400), no competing diagnosis identified. **Viral hepatitis panel:** HBsAg negative, anti-HCV negative, anti-HAV IgM negative. **Acetaminophen level:** undetectable. ANA/ASMA: negative. **RUQ ultrasound with Doppler (date):** hepatomegaly, moderate ascites, patent portal vein, no biliary obstruction, no focal liver lesion. Diagnostic paracentesis (date): SAAG 1.8 (portal hypertension); PMN 82/μL (SBP excluded); culture pending.

ASSESS: Severe alcoholic hepatitis with ascites, MELD-Na 26, Maddrey DF 42 — **K70.11** (AH with ascites). Alcohol use disorder, severe, active — **F10.20**. **Corticosteroid eligibility:** pending infection surveillance results, GI bleeding assessment (guaiac negative), HBsAg negative, glucose manageable. **Pre-existing:** no prior cirrhosis diagnosis on imaging — evaluate for concurrent cirrhosis after acute episode resolves. Malnutrition — **E44.1** (mild-moderate protein-calorie). **AKI risk:** creatinine 1.3 (baseline unknown) — hold nephrotoxins, monitor urine output.

TREAT: "Prednisolone 40 mg/day PO pending negative infection surveillance; IV NAC × 5 days per ACG 2024. Lille at day 7 — <0.45: complete 28 days; ≥0.45: stop steroids, supportive care, **early transplant discussion if MELD >26**. Nutrition: 35–40 kcal/kg/day, 1.2–1.5 g protein/kg/day; thiamine 100 mg IV before glucose; zinc, folate, B12; RD consult; enteral feeding if oral intake insufficient. AUD: naltrexone 50 mg/day PO before discharge; peer recovery referral. Withdrawal: CIWA-Ar-guided lorazepam (symptom-triggered). **Nephrotoxin avoidance: hold NSAIDs/ACEi/ARB**; acetaminophen ≤2 g/day; no IV contrast without hepatology/nephrology review. Glucose: fingerstick QID on steroids; sliding scale if >180. Surveillance: MELD-Na q48–72h; daily Cr/lytes/CBC; procalcitonin with any deterioration. Referrals: **hepatology (within 24h)**, addiction medicine (parallel), social work. ACP (CPT 99497): surrogate and code status documented while decisional capacity intact."

Clinical Documentation Elements

- **Specify severity:** State MELD (preferred per ACG 2024) AND mDF; Lille at day 7 when corticosteroids given; ICA-AKI stage; ascites status **(K70.10 vs K70.11)**. 'AH with ascites **(K70.11)**, severe per MELD 24, Maddrey DF 42; Lille 0.38 at day 7 (responder) — continue prednisolone 40 mg/d × 28 days'
- **Include current data:** Current bilirubin, AST/ALT ratio, INR, creatinine, albumin, WBC, glucose, MELD; culture results when drawn; Lille score when applicable. 'Bilirubin 8 mg/dL (admission 10); MELD 24 → 22 at day 7; cultures negative; Lille 0.38 — responder.' Data is the evidence of active disease management
- **Link comorbidities:** Explicitly link AH to AUD **(F10.xx — root cause)**, cirrhosis (**K70.31/K74.6x**), HE (**K72.x**), AKI (**N17.x**), varices (**I98.3**), portal HTN (**K76.6**), diabetes (**E11.x**), CKD (**N18.x**). 'AH with ascites (**K70.11**), alcoholic cirrhosis with ascites (**K70.31**), hepatic encephalopathy grade II (**K72.90**), active moderate AUD (**F10.20**) — naltrexone 50 mg/d initiated'
- **Document chronicity/active vs remission:** Active AH uses **K70.10/K70.11**. Do NOT use **Z87.891** while disease is active.[1] Record onset of current episode (jaundice onset), duration of alcohol use, prior episodes, prior treatments. When sustained sobriety and normalized function: then Z87.891 may apply — not before
- **Associated conditions: List all active codes: K70.10** or **K70.11** (primary); **F10.20** or **F10.21** (AUD); **K70.31/K74.6x** (cirrhosis when concurrent); **K72.x** (HE); **N17.x** (AKI); **I98.3** (varices); **K76.6** (portal HTN); **E11.x** (DM); **N18.x** (CKD); **Z87.891** (ONLY after sustained remission). Each carries independent clinical and documentation significance

Reframing Common Documentation Shortcuts

INSTEAD OF…	DOCUMENT…
'Elevated LFTs — advised to cut back on alcohol; recheck 2 months.'	'Alcoholic hepatitis with ascites (**K70.11**), severe per MELD 24 and Maddrey DF 42. Active moderate alcohol use disorder (**F10.20**). Admit; hepatology consult; hold NSAIDs and ACEi; thiamine 100 mg IV before glucose; blood/urine/ascitic cultures; procalcitonin; glucose control; pre-corticosteroid checklist. Naltrexone 50 mg/d planned at discharge'
'History of alcoholic hepatitis' (during active disease)	'**Alcoholic hepatitis with ascites (K70.11)** — active disease with bilirubin 8 mg/dL, AST/ALT 2.1, ongoing alcohol use. Z87.891 NOT applicable'
'Alcoholic liver disease, unspecified' (K70.9)	'Alcoholic hepatitis without ascites (**K70.10**) — physical exam without shifting dullness or fluid wave; ultrasound without ascites; bilirubin 6 mg/dL, AST 180/ALT 85. Active moderate AUD (**F10.20**)'
'Will refer to addiction medicine'	'Active moderate AUD (**F10.20**) — naltrexone 50 mg/d started today. Baclofen as alternative if intolerance. Peer recovery support referral. Addiction medicine consult placed in parallel — AUD pharmacotherapy initiated without waiting'
'Steroids started — will check Lille'	'**Prednisolone 40 mg/d day 1 of 28** — pre-treatment checklist: cultures negative, procalcitonin 0.12, HBsAg negative, glucose controlled on basal-bolus, no active GI bleed, creatinine 1.1. Lille score at day 7 planned for continuation decision'

ABBREVIATIONS: ACEi = angiotensin-converting enzyme inhibitor; AH = alcoholic hepatitis; AST/ALT = aspartate/alanine aminotransferase; AUD = alcohol use disorder; AUDIT-C = Alcohol Use Disorders Identification Test — Consumption; HBsAg = hepatitis B surface antigen; HCC = hierarchical condition category; HEDIS ASF = Unhealthy Alcohol Use Screening and Follow-Up; IV = intravenous; LFT = liver function test; mDF = Maddrey discriminant function; MELD = Model for End-Stage Liver Disease; NSAID = nonsteroidal anti-inflammatory drug; PCP = primary care provider
*__Consult FDA labels for the most up-to-date dosage information, contraindications, and drug-drug interactions__

DOCUMENTATION REMINDER

Every AH encounter should state the diagnosis with severity and ascites status in the A&P ('Alcoholic hepatitis with ascites (**K70.11**), severe per MELD 24 and mDF 42'), sequence AUD as root cause ('active moderate alcohol use disorder (**F10.20**)'), document current labs (bilirubin, AST/ALT, INR, Cr, albumin), note corticosteroid eligibility and Lille score when applicable, list comorbidity linkages, and describe the active plan (nephrotoxin withdrawal, nutrition, AUD pharmacotherapy, follow-up). Avoid **Z87.891** while active; avoid **K70.9** when **K70.10/.**[11] is supported.

4 TREATMENT AND REFERRAL QUICK GUIDE

Therapy Escalation Criteria

Alcoholic hepatitis management operates as a time-sensitive escalation sequence rather than a single treatment decision — each 48–72 hour interval requires reassessment and potential course correction.[1,2,8]

TRIGGER	ACTION
New jaundice + heavy alcohol use (bilirubin >3 mg/dL, AST/ALT >1.5, jaundice <8 weeks)	Same-day evaluation; calculate MELD + mDF; check INR, Cr, glucose, WBC; obtain HBsAg, anti-HCV, ultrasound with Doppler; rule out competing causes. **If MELD >20 or mDF ≥32 or ascites or HE → hospitalize and hepatology consult**
MELD >20 or Maddrey DF ≥32 (severe AH)	Hospitalize; hepatology consult within 24 h; infection **surveillance (blood + urine + ascitic cultures + procalcitonin) BEFORE corticosteroid decision**; nephrotoxin withdrawal; nutritional support; glucose control; pre-corticosteroid contraindication checklist
Corticosteroid candidacy met (no active infection, no uncontrolled DM, no GI bleed, no untreated HBV, no severe renal failure)	Prednisolone 40 mg/d PO × 28 days if Lille <0.45 at day 7; add IV N-acetylcysteine × 5 days as adjunct. Methylprednisolone 32 mg/d IV if oral route unavailable. **Pentoxifylline NOT recommended (strong rec against, ACG 2024)**
Lille ≥0.45 at day 7 (non-responder)	**Discontinue corticosteroids**. Early liver transplant evaluation (if MELD >26 and AUD insight and social support present). Palliative-hepatology plan if transplant not appropriate. **DO NOT continue dogmatic 28-day courses in non-responders**
AKI in severe AH (creatinine rise ≥0.3 within 48 h or 1.5× baseline)	**Stop ALL nephrotoxins (NSAIDs, ACEi/ARB, contrast, aminoglycosides)**; IV albumin 1 g/kg × 48 h; rule out sepsis, GI bleed, volume depletion; nephrology consult; if volume-refractory and no alternative cause → HRS protocol with terlipressin (where available) or norepinephrine + albumin

ABBREVIATIONS: ACEi = angiotensin-converting enzyme inhibitor; AH = alcoholic hepatitis; AKI = acute kidney injury; ARB = angiotensin receptor blocker; AST/ALT = aspartate/alanine aminotransferase; AUD = alcohol use disorder; DM = diabetes mellitus; GI = gastrointestinal; HBV = hepatitis B virus; HBsAg = hepatitis B surface antigen; HCC = hepatocellular carcinoma; HE = hepatic encephalopathy; HRS = hepatorenal syndrome; IM = intramuscular; IV = intravenous; mDF = Maddrey discriminant function; MELD = Model for End-Stage Liver Disease; PO = by mouth; TID = three times daily

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

ACG/AASLD/NIAAA/USPSTF/AAFP Aligned Recommendations

Alcohol-associated hepatitis (AH) is an acute inflammatory liver injury that can progress rapidly, particularly in patients with severe jaundice, coagulopathy, kidney injury, infection, malnutrition, or

underlying cirrhosis. Primary care clinicians play an important role in early recognition, alcohol use disorder (AUD) treatment initiation, nutrition support, medication safety, and timely escalation to hepatology when severe disease is suspected. Management should prioritize clinical stabilization, identification of corticosteroid eligibility, close monitoring for infection and acute kidney injury, and integrated support for sustained alcohol cessation.[2,8,12]

CATEGORY	RECOMMENDED AGENT/ APPROACH	NOTES
Severe AH — corticosteroids	Prednisolone 40 mg/d PO × 28 days if Lille <0.45 at day 7; alternative prednisone 40 mg/d PO; methylprednisolone 32 mg/d IV if oral unavailable	NNT = 7 for 28-day survival in severe AH (MELD >20 or mDF ≥32) with eligible contraindication profile. No benefit beyond 28 days. **Pentoxifylline strong recommendation AGAINST**
Corticosteroid adjunct — IV N-acetylcysteine	IV NAC × 5 days added to corticosteroids; strong rec, moderate evidence	Improves 28-day survival and reduces infection vs corticosteroids alone. **No benefit at 6 months**
Supportive care — infection surveillance, nephrotoxin avoidance, nutrition	Blood + urine + ascitic cultures at admission and with any deterioration; procalcitonin to differentiate SIRS; stop NSAIDs, ACEi/ARB, contrast; target 35–40 kcal/kg/d and 1.2–1.5 g protein/kg/d; thiamine 100 mg IV before glucose	**Prophylactic antibiotics NOT recommended universally** (strong rec against, ACG 2024). Enteral nutrition if oral intake inadequate
AUD pharmacotherapy — integrated	Naltrexone 50 mg/d PO or 380 mg IM monthly (preferred elderly per ACG 2025 geriatric hepatology)	Only 14% of ALD patients receive any AUD treatment, <1% pharmacotherapy.[7] Initiate in primary care — do not wait for specialty **Disulfiram absolutely contraindicated in ALD**
Non-responders + liver transplant	Non-responders (Lille ≥0.45) with MELD >26 → early transplant evaluation with demonstrated AUD insight and social support[3]	Conditional rec, low evidence. Integrated multidisciplinary models (hepatology + addiction medicine) improve outcomes

ABBREVIATIONS: ACEi = angiotensin-converting enzyme inhibitor; ACG = American College of Gastroenterology; AASLD = American Association for the Study of Liver Diseases; AH = alcoholic hepatitis; ALD = alcohol-associated liver disease; ARB = angiotensin receptor blocker; AUD = alcohol use disorder; AUDIT-C = Alcohol Use Disorders Identification Test — Consumption; HEDIS ASF = Unhealthy Alcohol Use Screening and Follow-Up; IM = intramuscular; IV = intravenous; mDF = Maddrey discriminant function; MELD = Model for End-Stage Liver Disease; NAC = N-acetylcysteine; NIAAA = National Institute on Alcohol Abuse and Alcoholism; NNT = number needed to treat; NSAID = nonsteroidal anti-inflammatory drug; PO = by mouth; TID = three times daily; USPSTF = US Preventive Services Task Force
***Consult FDA labels for the most up-to-date dosage information, contraindications, and drug-drug interactions**

Non-Pharmacologic Treatment and Lifestyle Modification

Surviving the acute episode of alcoholic hepatitis is only the first milestone — long-term survival is determined largely by what happens after discharge, where sustained alcohol abstinence, nutritional rehabilitation, integrated addiction treatment, social determinants of health intervention,

and ongoing hepatic surveillance collectively define whether the patient returns to functional recovery or cycles through preventable readmissions and progressive liver failure.[13,18,19]

INTERVENTION	TARGET/RECOMMENDATION	NOTES
Absolute alcohol abstinence	Complete cessation — the single most impactful intervention for long-term survival; **even partial reduction improves outcomes** but abstinence is the goal	Counsel at every visit with non-judgmental motivational interviewing; document in structured EHR fields (HEDIS ASF). **Alcohol dependence prompts withdrawal support and surveillance**
Nutritional rehabilitation	35–40 kcal/kg/d; 1.2–1.5 g protein/kg/d; thiamine 100 mg IV or IM **BEFORE** glucose; zinc, folate, B12 supplementation; enteral feeding if oral <35 kcal/kg	**Do NOT restrict protein** — worsens sarcopenia and hepatic encephalopathy. Small frequent meals reduce fasting catabolism. RD consultation when available
Peer recovery support + addiction medicine integration	Integrated multidisciplinary care (hepatology + addiction medicine + peer recovery + primary care)	Integrated models improve long-term abstinence and outcomes; do not wait for addiction specialist — initiate pharmacotherapy in PCP + hepatology setting while specialty referral pending
SDOH intervention (housing, isolation, financial)	**Screen SDOH at every visit**; connect to community resources, Area Agency on Aging, transportation, housing, food security	**Untreated SDOH drive relapse and readmission**. Social isolation correlates with treatment failure; involve family/chosen family where appropriate
HCC and varices surveillance (when cirrhosis concurrent)	**Ultrasound ± AFP every 6 months**; endoscopy for varices per Baveno; band ligation + non-selective beta-blocker for large varices	AH and cirrhosis often coexist; HCC incidence 2–4% per year in cirrhosis — **surveillance is mandatory**

ABBREVIATIONS: AFP = alpha-fetoprotein; AH = alcoholic hepatitis; Baveno = Baveno consensus on portal hypertension; CKD = chronic kidney disease; EHR = electronic health record; HCC = hepatocellular carcinoma; HEDIS ASF = Unhealthy Alcohol Use Screening and Follow-Up; HTN = hypertension; IM = intramuscular; IV = intravenous; PCP = primary care provider; RD = registered dietitian; SDOH = social drivers of health

Non-Pharmaceutical Prevention Strategies

INTERVENTION STRATEGY	BEST PRACTICES
Behavioral and Care Integration	Establish **structural** addiction medicine consults, **co-managed** psychiatry, and direct warm handoffs to peer recovery support networks (e.g., AA, SMART Recovery)

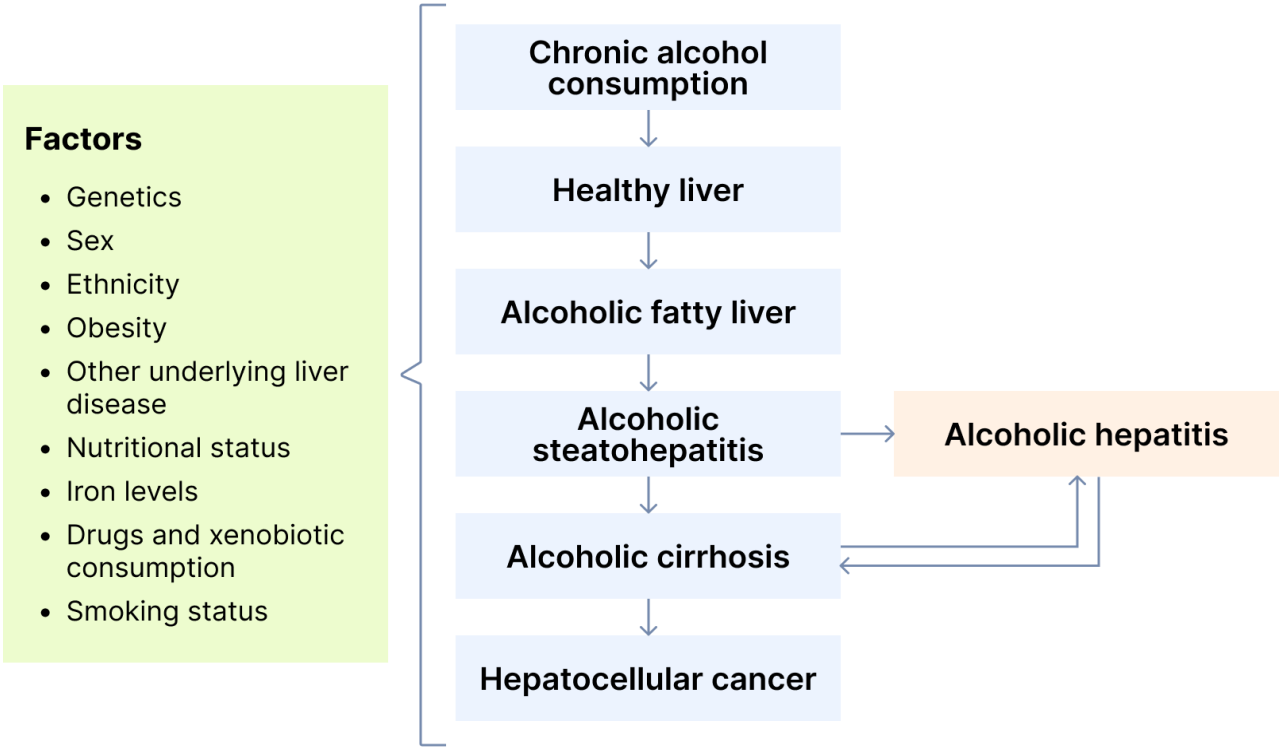
INTERVENTION STRATEGY	BEST PRACTICES
Social Determinants of Health (SDOH)	**Systematically screen** for and document structural barriers such as housing instability, social isolation, and financial insecurity that directly drive relapse
Metabolic and Cellular Support	Prescribe **high-protein**, calorie-dense diets coupled with aggressive micronutrient repletion (Thiamine, Folate) to reverse underlying malnutrition
Organ Protection	**Enforce strict "zero tolerance" guardrails against known nephrotoxins** (especially NSAIDs) and hepatotoxins to shield compromised renal and hepatic vascular beds
Advanced Longitudinal Monitoring	In cases of concurrent, established cirrhosis, hardcode standard Hepatocellular Carcinoma (HCC) surveillance (ultrasound + AFP every 6 months) and variceal screening protocols into the EMR
Definitive Rescue Therapy	Expeditiously escalate to an early liver transplant evaluation when guideline-directed medical therapy fails and the patient satisfies foundational psychosocial prerequisites

ABBREVIATIONS: AA = Alcoholics Anonymous; AFP = alpha-fetoprotein; EMR = electronic medical record; HCC = hepatocellular carcinoma; NSAID = nonsteroidal anti-inflammatory drug; SDOH = social determinants of health; SMART = Self-Management and Recovery Training

CLINICAL PEARL: DO NOT WATCH & WAIT

Suspected severe alcohol-associated hepatitis — **jaundice, ascites, or hepatic encephalopathy in the setting of heavy alcohol use — requires immediate multidisciplinary escalation**, not observation. Delayed action in this clinical scenario is associated with increased mortality, and current guidelines from the ACG and AASLD position urgent hepatology involvement, infection surveillance, and prognostic scoring (MELD-Na, mDF) as the standard of care from the point of recognition.

Factors Influencing Alcoholic Hepatitis



Medication Safety and Dose Adjustments

Alcoholic hepatitis affects the pharmacokinetic and pharmacodynamic profile of commonly prescribed medications. Medications that are routine in general practice become high-lethality exposures in the context of splanchnic vasodilation, impaired renal autoregulation, and coagulopathy.[1,20]

DRUG/ COMBINATION	INTERACTION CONTEXT	MECHANISM I RISK	ACTION	MONITORING
NSAIDs (ibuprofen, naproxen, ketorolac, celecoxib) + Active AH	Active alcoholic hepatitis	NSAIDs impair renal autoregulation via prostaglandin inhibition **Risk: HIGH**	**STOP all NSAIDs at admission**. Cautiously use acetaminophen ≤2 g/day for pain. Do not resume without hepatology/ nephrology review	Creatinine daily while hospitalized; urine output; urine microscopy if AKI develops; AKI stage per ICA-AKI criteria

DRUG/ COMBINATION	INTERACTIO N CONTEXT	MECHANISM I RISK	ACTION	MONITORING
ACE inhibitors/ ARBs + Active AH, particularly with ascites	Active AH with portal hypertension and/or ascites	ACEi/ARBs reduce effective arterial volume and renal perfusion in the setting of splanchnic vasodilation → hypotension, AKI, and hepatorenal syndrome risk. NSAIDs, ACEi, and ARBs should be avoided in patients with cirrhosis and ascites **Risk: HIGH**	**Hold** in active AH and decompensated cirrhosis with ascites; reassess once AH resolved and hemodynamics stable. Use spironolactone + furosemide for ascites control with close electrolyte and creatinine monitoring	Creatinine, potassium, sodium daily during active disease; BP; hydration status
Aminoglycosides + IV contrast + Active AH	Active AH with high AKI risk	Direct nephrotoxicity superimposed on AH-associated AKI risk **Risk: HIGH**	**Avoid entirely** in active AH; substitute ceftriaxone or other non-nephrotoxic antibiotics. Defer contrast imaging when alternatives are available	Creatinine; urine output; reassess imaging necessity with radiology
Disulfiram in any ALD	Any stage of alcohol-associated liver disease	Hepatotoxicity risk including fulminant liver failure **Risk: ABSOLUTE CONTRAINDICATION**	**Do NOT initiate under any circumstance**s. Use acamprosate (renally excreted, no hepatic metabolism), or naltrexone (accumulating safety data in compensated cirrhosis) instead	**Discontinue immediately if present**; LFTs

DRUG/ COMBINATION	INTERACTIO N CONTEXT	MECHANISM I RISK	ACTION	MONITORING
Corticosteroids (prednisolone, prednisone, methylprednisolone) + Active infection, uncontrolled DM, GI bleed, untreated HBV, severe renal failure	Severe AH with contraindications present	Prednisolone increases susceptibility to serious infection **Risk: HIGH**	Pre-initiation checklist mandatory. **Active infection is absolute contraindication** — treat infection first. Uncontrolled DM — control first. HBsAg positive — antiviral prophylaxis before initiation	**Daily infection surveillance during therapy**; glucose daily; Lille score at day 7; stool guaiac; HBV DNA if HBsAg+
Benzodiazepines (lorazepam, diazepam) + Any sedating agent in cirrhosis	Cirrhosis with or at risk for hepatic encephalopathy	Benzodiazepines are metabolized by the liver and accumulate in cirrhosis; associated with a **24% increased risk of incident HE and 23% increased risk of HE hospitalization Risk: HIGH**	**Avoid benzodiazepines in cirrhosis.** If alcohol withdrawal requires treatment, use lorazepam or oxazepam at minimal dose. Alternatives: hydroxyzine 25 mg for anxiety/ insomnia; SSRIs for chronic anxiety	If absolutely required, use lorazepam at minimal dose. **Do NOT taper abruptly (seizure risk)**; plan slow taper. Monitor mental status continuously
Naltrexone + Opioid medications	Concurrent prescribing in AUD treatment	Naltrexone is a competitive opioid receptor antagonist — concurrent opioid use renders analgesia ineffective and can precipitate acute opioid withdrawal (agitation, vomiting, diarrhea, piloerection, tachycardia) **Risk: MODERATE**	**Confirm opioid-free status before initiating naltrexone (7–10 day washout from short-acting opioids; 10–14 days from methadone).** Coordinate with pain management. Document opioid status. If patient requires opioid analgesia, use baclofen or acamprosate for AUD instead	Pain plan documented; opioid use history; urine drug screen (UDS) before initiation and when clinically appropriate; monitor for precipitated withdrawal symptoms if opioid status uncertain

DRUG/ COMBINATION	INTERACTIO N CONTEXT	MECHANISM I RISK	ACTION	MONITORING

ABBREVIATIONS: ACEi = angiotensin-converting enzyme inhibitor; AH = alcoholic hepatitis; AKI = acute kidney injury; ALD = alcohol-associated liver disease; ARB = angiotensin receptor blocker; BP = blood pressure; ICA-AKI = International Club of Ascites – Acute Kidney Injury (criteria); IV = intravenous; LFTs = liver function tests; NSAIDs = nonsteroidal anti-inflammatory drugs
*__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.[1]

CRITERION	SPECIALIST	URGENCY
Severe AH (MELD >20, mDF ≥32), ascites, HE, variceal bleed, or bilirubin >3 mg/dL	Hepatology (hospitalized) + addiction medicine in parallel	**Immediate — within 24 h of admission**. Do not delay corticosteroid eligibility assessment for specialty visit scheduling. Addiction medicine in parallel, not sequentially
AKI in AH, volume-refractory, HRS suspected	Nephrology + hepatology urgent	**Within 12–24 h.** Stop nephrotoxins; IV albumin 1 g/kg × 48 h; if no response, HRS protocol with terlipressin or norepinephrine + albumin
AH discharge without AUD pharmacotherapy initiated	Addiction medicine + PCP	Before discharge or within 1 week. **Do NOT discharge without pharmacotherapy**; naltrexone or alternatives initiated in-hospital; peer recovery support arranged
Concurrent cirrhosis or hepatocellular carcinoma (HCC) suspicion	Hepatology (outpatient)	**Within 2–4 weeks**. HCC surveillance (ultrasound ± AFP every 6 months), varices surveillance (endoscopy per Baveno); transplant evaluation discussion
Older adult with AH + frailty, cognitive decline, or polypharmacy concerns	Geriatric hepatology + PCP; palliative care consult if appropriate	**Within 1–2 weeks.** Frailty-guided treatment intensity; medication reconciliation with Beers Criteria; goals-of-care conversation

ABBREVIATIONS: AH = alcoholic hepatitis; AKI = acute kidney injury; AUD = alcohol use disorder; Beers = American Geriatrics Society Beers Criteria; HCC = hepatocellular carcinoma; HE = hepatic encephalopathy; HRS = hepatorenal syndrome; IV = intravenous; mDF = Maddrey discriminant function; MELD = Model for End-Stage Liver Disease; PCP = primary care provider *__Consult FDA labels for the most up-to-date dosage information, contraindications, and drug-drug interactions__

Follow-Up Timing

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

STAGE/CATEGORY	FREQUENCY	LABS/ASSESSMENTS TO MONITOR
Initial AH diagnosis (outpatient, not yet severe)	**Same-day labs**; **hepatology within 1–2 weeks; PCP within 1 week**	Bilirubin, AST/ALT, INR, Cr, albumin, WBC, glucose; HBsAg, anti-HCV; ultrasound with Doppler; MELD ± mDF; AUDIT-C; naltrexone or baclofen initiated
Severe AH — hospitalized	**Daily labs**; MELD at admission + day 7 + with clinical change; **Lille at day 7** of corticosteroids	Bilirubin, AST/ALT, INR, Cr, WBC, glucose, electrolytes daily; cultures at admission + with deterioration; procalcitonin; MELD; Lille; nephrotoxin review
Post-discharge — acute phase (weeks 1–4)	**PCP within 1 week; hepatology within 1–2 weeks; addiction medicine within 2 weeks**	LFTs, INR, Cr weekly × 4; adherence to naltrexone/baclofen; AUDIT-C; peer recovery engagement; SDOH reassessment; HEDIS ASF Rate 2 documentation
Stabilization (months 1–6)	**Every 4 weeks** × 3; then every 2–3 months if stable	LFTs, INR, Cr; AUDIT-C; adherence; metabolic panel (HbA1c, lipids); weight; varices endoscopy if cirrhosis; ultrasound every 6 months if cirrhosis (HCC)
Long-term maintenance (beyond 6 months)	**Every 3–6 months**	LFTs; AUDIT-C; medication adherence; HCC + varices surveillance if cirrhosis (ultrasound ± AFP every 6 months; endoscopy per Baveno); annual depression screen (AUD comorbid); annual comprehensive metabolic

ABBREVIATIONS: AFP = alpha-fetoprotein; AH = alcoholic hepatitis; AST/ALT = aspartate/alanine aminotransferase; AUDIT-C = Alcohol Use Disorders Identification Test — Consumption; Baveno = Baveno consensus on portal hypertension; Beers = American Geriatrics Society Beers Criteria; CFS = Clinical Frailty Scale; Cr = creatinine; HbA1c = hemoglobin A1c; HCC = hepatocellular carcinoma; HEDIS ASF = Unhealthy Alcohol Use Screening and Follow-Up; INR = international normalized ratio; LFT = liver function test; mDF = Maddrey discriminant function; MELD = Model for End-Stage Liver Disease; MoCA = Montreal Cognitive Assessment; PCP = primary care provider; SDOH = social drivers of health

Comorbidity Management

Effective long-term success in cirrhosis management depends on aggressive treatment of the co-existing conditions that contribute.[8]

COMORBIDITY	APPROACH	CAUTION
Alcohol use disorder (F10.xx) — root cause	Naltrexone 50 mg/d PO or 380 mg IM monthly (preferred elderly); acamprosate TID when renal function permits; NOT disulfiram	Only 14% of ALD patients receive any AUD treatment, <1% pharmacotherapy. **Initiate in PCP setting — do not wait for an addiction specialist. Disulfiram absolutely contraindicated**
Cirrhosis (K70.31/K74.6x) + portal hypertension	HCC surveillance (ultrasound ± AFP every 6 months); endoscopy per Baveno for varices; non-selective beta-blocker + band ligation for large varices	Code BOTH AH (**K70.1x**) AND cirrhosis (**K70.31 or K74.6x**) when both present. Child-Pugh and MELD for staging
Diabetes mellitus (E11.x)/ pre-diabetes	Metformin if eGFR permits; SGLT-2/ GLP-1 per CV/renal indication; insulin during corticosteroid therapy; **control diabetes BEFORE corticosteroid initiation**	Uncontrolled DM is a corticosteroid contraindication; close glucose monitoring during prednisolone therapy; **adjust antidiabetic agents**
Chronic kidney disease (N18.x)	eGFR at baseline and each visit; stop nephrotoxins; dose-adjust medications; nephrology when stage 3b or worse	CKD amplifies AKI risk; AKI in severe AH carries 9-fold 90-day mortality. Acamprosate requires TID dosing and is limited by CKD
Hepatic encephalopathy (K72.x)	Lactulose titrated to 2–3 soft stools/ day; rifaximin 550 mg BID when recurrent; rule out precipitants (infection, GI bleed, electrolyte, medication)	**Benzodiazepines can precipitate/ worsen HE** — use CIWA-Ar-guided shorter-acting agents. Avoid topiramate for AUD if HE present
Malnutrition + sarcopenia (E44.x, M62.84)	35–40 kcal/kg/d; 1.2–1.5 g protein/kg/ d; thiamine 100 mg IV before glucose; zinc, folate, B12; enteral if oral <35 kcal/kg	**Do NOT restrict protein**. RD consultation when available. Small frequent meals reduce fasting catabolism

ABBREVIATIONS: AFP = alpha-fetoprotein; AH = alcoholic hepatitis; AKI = acute kidney injury; ALD = alcohol-associated liver disease; AUD = alcohol use disorder; Baveno = Baveno consensus on portal hypertension; BID = twice daily; CIWA-Ar = Clinical Institute Withdrawal Assessment — Alcohol, revised; CKD = chronic kidney disease; CV = cardiovascular; DM = diabetes mellitus; eGFR = estimated glomerular filtration rate; GLP-1 = glucagon-like peptide-1; HCC = hepatocellular carcinoma; HE = hepatic encephalopathy; IM = intramuscular; IV = intravenous; PCP = primary care provider; RD = registered dietitian; SGLT-2 = sodium-glucose cotransporter-2; TID = three times daily
***Consult FDA labels for the most up-to-date dosage information, contraindications, and drug-drug interactions**

Cost-Smart Options

Effective long-term success in cirrhosis management depends on aggressive treatment of the co-existing conditions that contribute to liver damage and functional decline.

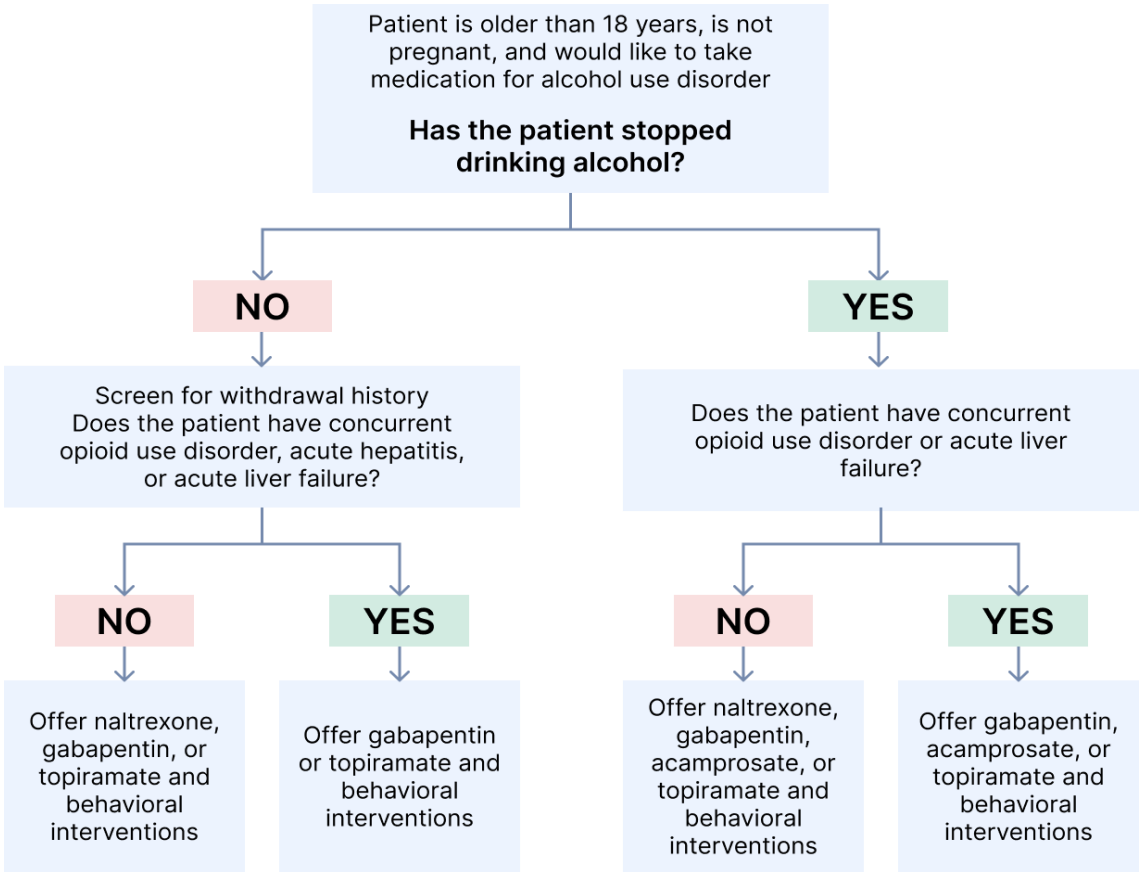
THERAPEUTIC AGENT	GENERIC AND COST	BRAND AND COST	COST SAVINGS	COST SMART TIPS AND NOTES
Prednisolone /Prednisone	Generic prednisolone/ prednisone 40 mg daily (~$4–$15/28-day course)	Millipred/ Orapred (~$50–$150+/ course)	~$35–$135/ course	Generic 40 mg/day costs ~$4–15/ course; cost is essentially zero as a barrier — the limiting factor is the infection/contraindication checklist, not price Lille score at day 7 identifies non-responders early; stopping futile steroid therapy avoids infectious complications and unnecessary inpatient days
N-Acetylcysteine (NAC)	Generic IV Acetylcysteine (200 mg/mL solution): ~$30–$60 per 30mL vial ~$150–$300 (5-day regimen)	Acetadote(Intravenous Injection)~$120 – $180 per vial ~$600 – $900+	~$300 – $600/ course	Generic IV acetylcysteine saves ~$300–600/course vs brand Acetadote; identical formulation 5-day fixed protocol limits total cost; must pair with corticosteroids (no monotherapy benefit)
Spironolactone	Generic spironolactone 25–100 mg (~$5–$10/ month; Medicare OOP median ~$2/ month)	Aldactone (~$45–$90/ month)	~$35–$80/ month	Co**-**prescribed in tandem with to manage ascites and peripheral edema Combined generic cost ~$9–16/ month; **among the cheapest chronic medications in any formulary** If gynecomastia develops, switch to amiloride (~$15/month) before eplerenone (~$200+/month) Electrolyte-driven AKI from poor monitoring generates far greater costs than the diuretics themselves

THERAPEUTIC AGENT	GENERIC AND COST	BRAND AND COST	COST SAVINGS	COST SMART TIPS AND NOTES
Furosemide	Generic furosemide 20–80 mg (~$3–$5/ month; Medicare OOP median ~$1/ month)	Lasix(~$30–$60/month)	~$25–$55/ month	Co**-**prescribed in tandem with to manage ascites and peripheral edema Combined generic cost ~$9–16/ month; **among the cheapest chronic medications in any formulary** If gynecomastia develops, switch to amiloride (~$15/month) before eplerenone (~$200+/month) Electrolyte-driven AKI from poor monitoring generates far greater costs than the diuretics themselves
Naltrexone	Generic naltrexone 50 mg daily (~$15–$35/month)	ReVia (~$40–$60/ month)	~$20–$30/ month	Generic oral naltrexone ~$15–35/ month; reserve Vivitrol IM (~$1,300/injection) for documented oral non-adherence where compliance benefit justifies premium For active liver disease or advanced cirrhosis, acamprosate (no hepatic metabolism) is the safer alternative; disulfiram is contraindicated in any ALD stage
Lactulose	Generic lactulose solution (~$15/473mL bottle, ~$15–$50/ month at HE dosing)	Enulose/ Kristalose(~$50–$200+/ month)	~$35–$150/ month	Generic PEG 3350 (~$10–15/ month) is an evidence-based alternative if lactulose causes intolerable bloating
Rifaximinsecondary HE prevention	No generic currently available in US	Xifaxan (rifaximin) 550 mg BID ~$1,800–$2,200/month —Copay)	—	**No generic available** in the US (~$1,800–2,200/month); **reserve for secondary prophylaxis only** — add after a breakthrough HE episode on lactulose, not as first-line prevention

THERAPEUTIC AGENT	GENERIC AND COST	BRAND AND COST	COST SAVINGS	COST SMART TIPS AND NOTES
Acamprosate	Generic acamprosate 666 mg TID (~$30–$80/ month)	Campral (~$100–$250/ month)	~$20–$170/ month	safe in liver disease (renally excreted, no hepatic metabolism) — the preferred AUD medication when naltrexone is contraindicated due to active hepatitis or cirrhosis **TID dosing and large tablet size are the primary adherence barriers**, not cost; best suited for highly motivated, already-abstinent patients not deterred by dosing frequency

ABBREVIATIONS: AKI = acute kidney injury; ALD = alcohol-associated liver disease; BID = twice daily; HE = hepatic encephalopathy; IM = intramuscular; IV = intravenous; NAC = N-acetylcysteine; OOP = out-of-pocket; PEG = polyethylene glycol. ***Consult FDA labels for the most up-to-date dosage information, contraindications, and drug-drug interactions**

Medication Decision Pathway for Alcohol Use Disorder



Patient Education and Adherence

1. Alcohol Abstinence — The Single Most Important Intervention

Abstinence from alcohol is the cornerstone of treatment for alcohol-associated hepatitis (AH). Without treatment and abstinence, 90-day mortality reaches **40–50%**.[12]

- Sustained abstinence is associated with long-term survival in both severe and moderate AH
- Critically, patients should be encouraged to abstain completely but offered support and encouragement even if they fail to achieve full abstinence, as even a reduction in alcohol consumption improves survival

Key patient-facing messages:

- Any amount of alcohol can worsen liver inflammation and increase the risk of liver failure
- Jaundice should be reported immediately if it develops or worsens — it is a harbinger of serious liver injury
- Recovery is possible with sustained abstinence; the liver has some capacity to heal when the ongoing insult is removed

2. Behavioral and Psychosocial Interventions

The ACG guideline strongly recommends integrated multidisciplinary care incorporating behavioral and/or pharmacotherapy for AUD treatment.[8]

Evidence-supported modalities include:

- **Brief Interventions (5 A's model):** Ask, Advise, Assess, Assist, Arrange — shown to reduce alcohol consumption by ~20 g/week in primary care settings across a meta-analysis of >33,000 participants
- **Motivational Enhancement Therapy (MET):** Particularly effective for patients who are ambivalent about change; has supporting evidence specifically in alcohol-related liver disease
- **12-Step Facilitation/Alcoholics Anonymous:** A Cochrane review found this approach had the most promise for decreasing drinking behavior compared to CBT or no treatment
- **Cognitive Behavioral Therapy (CBT):** Helps identify relapse triggers and improve coping strategies; can be integrated with family/couples therapy

DOCUMENTATION REMINDER: PATIENT EDUCATION

Document the following education topics, the patient and caregiver(s) present, materials provided, and patient understanding (teach-back method):

- **Alcohol use disorder and abstinence:** Document alcohol use history (quantity, frequency, duration, last drink), AUDIT-C score, diagnosis of AUD, and counseling on absolute abstinence as the single most important modifiable factor for survival; abstinence reduces mortality at all stages of alcohol-associated liver disease
- **Alcohol withdrawal risk and monitoring:** Explain CIWA-Ar symptom-triggered protocol, timeline of withdrawal symptoms (6–96 hours post-last drink), seizure and delirium tremens warning signs, and importance of reporting symptoms immediately rather than minimizing intake history
- **Corticosteroid therapy (if initiated):** Purpose, expected 28-day course, Lille score reassessment at day 7 to determine continuation vs. discontinuation, infection risk during treatment, glucose monitoring requirements, and signs of infection to report (fever, worsening mental status, abdominal pain)
- **AUD pharmacotherapy and recovery support:** Naltrexone or acamprosate options post-stabilization (naltrexone contraindicated in acute hepatitis/advanced cirrhosis); peer recovery specialist referral; mutual support groups; relapse is expected and does not preclude ongoing care
- **Caregiver assessment:** Document caregiver present, their understanding of care responsibilities (lactulose administration, encephalopathy monitoring, alcohol-free home environment, medication reconciliation, appointment adherence), and burden screening

Quality Metrics Tie-In

MEASURE (MY2026)	STANDARD	APPLICABILITY TO ALCOHOLIC HEPATITIS[21]
HEDIS ASF: Unhealthy Alcohol Use Screening and Follow-Up	**Denominator:** enrollees ≥18 with an eligible encounter **Numerator:** Rate 1 — screened for unhealthy alcohol use using validated instrument (AUDIT-C or NIAAA single question); Rate 2 — positive screen followed by brief intervention or referral within 2 months **Data source:** ECDS (requires structured data — LOINC, ICD-10-CM, CPT, SNOMED, HCPCS)	The single highest-yield measure for AH prevention. AUDIT-C ≥4 (men)/≥3 (women) = positive; unstructured narrative notes do NOT satisfy the ECDS measure. **Only 37.8% of adults report being asked about binge-level consumption at checkups**. Positive screen should trigger FIB-4 coupling to identify hepatic fibrosis early[22]
HEDIS IET: Initiation and Engagement of Substance Use Disorder Treatment	**Denominator:** enrollees ≥13 with newly diagnosed with SUD **Numerator:** Initiation — treatment within 14 days of diagnosis; Engagement — ≥2 additional services within 34 days of initiation **Exclusions:** hospice	**Only 14% of ALD patients receive any AUD treatment, 1% pharmacotherapy**. AH hospitalization is a critical opportunity to initiate AUD treatment — early alcohol rehabilitation within 30 days of discharge reduced readmission (AOR 0.16), relapse (AOR 0.11), and death (AHR 0.20). Naltrexone or baclofen should be initiated before discharge[8]
HEDIS COA: Care for Older Adults — Medication Review	**Denominator:** Medicare SNP enrollees ≥66, continuously enrolled **Numerator:** 4 sub-measures documented annually: (1) functional status assessment, (2) medication review **Exclusions:** hospice, ESRD	Nephrotoxin polypharmacy (NSAIDs, ACEi/ARBs, aminoglycosides) is the leading preventable cause of iatrogenic AKI in AH — **AKI is present in up to one-third of severe AH patients and carries 9-fold increase in 90-day mortality**. Medication review must reconcile all nephrotoxins, benzodiazepines (HE risk), and corticosteroid interactions at every encounter[1]

MEASURE (MY2026)	STANDARD	APPLICABILITY TO ALCOHOLIC HEPATITIS[21]
HEDIS COA: Care for Older Adults — Functional Status Assessment	**Denominator:** enrollees ≥66 **Numerator:** functional status assessment documented annually **Exclusions:** hospice, ESRD	**Sarcopenia and malnutrition are nearly universal in severe AH**; frailty assessment (Clinical Frailty Scale) guides whether transplant evaluation is appropriate and informs corticosteroid risk-benefit. Document functional status at every visit to track trajectory[10]
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	**AH 30-day readmission rates are 25-30%**, with annual costs reaching $164 million nationally. Readmission predictors include cirrhosis, ascites, AKI, and high comorbidity burden — each addressable through structured post-discharge medication reconciliation (nephrotoxin avoidance, AUD pharmacotherapy continuation, withdrawal management)[23]
HEDIS DMS-E: Utilization of the PHQ-9 to Monitor Depression Symptoms	**Denominator:** enrollees ≥12 with diagnosis of major depression or dysthymia AND elevated PHQ-9 ≥10 **Numerator:** follow-up PHQ-9 documented within 4–8 months of index elevated score **Exclusions:** hospice	Depression and anxiety are highly prevalent in AUD/ALD populations; untreated depression drives relapse and readmission. PHQ-9 at every visit; follow-up plan if positive; AH diagnosis, functional decline, and social isolation are independent depression risk factors
HEDIS ACP: Advance Care Planning	**Denominator: Adults ages 66–80** who have an advanced illness, a documented frailty indicator, or are receiving palliative care; all adults **ages 81 and older** **Numerator:** advance care plan or surrogate decision-maker documented, OR documentation that ACP was discussed but patient declined **Exclusions:** hospice	**Severe AH (MELD >20) carries 20–50% short-term mortality** — ACP should begin at diagnosis, not end-of-life. Billable as CPT 99497/99498 during AWV with no copay. Document surrogate, code status, and goals of care while decisional capacity is intact (before hepatic encephalopathy progression)

MEASURE (MY2026)	STANDARD	APPLICABILITY TO ALCOHOLIC HEPATITIS[21]
CMS Star PCR: Plan All-Cause Readmissions	**Denominator:** enrollees ≥18 with acute inpatient discharge **Numerator:** unplanned readmission within 30 days (lower is better) **Exclusions:** planned readmissions, hospice, psychiatric	**AH 30-day readmission rates (22.8–30%) are among the highest for any liver condition**; primary drivers include AH recurrence, alcoholic cirrhosis, and hepatic encephalopathy. Early alcohol rehabilitation within 30 days of discharge reduced readmission odds by 84% (AOR 0.16). **PCP post-discharge follow-up within 7 days reduces readmission risk**[23]
AASLD Cirrhosis Quality Measures: Post-Discharge Follow-Up	**Process measure:** recently discharged patients with cirrhosis should have a clinic visit within 4 weeks of discharge	Early post-discharge care was associated with lower all-cause mortality (HR 0.57) in a national VA cohort of 121,129 cirrhosis patients. **Readmissions after 30 days (HR 1.53) and 90 days (HR 1.88) were associated with higher mortality**[24]
AASLD Cirrhosis Quality Measures: Frailty Assessment	**Process measure:** patients with cirrhosis should be assessed for frailty using a systematic screening method	AASLD PMC rated importance 8/9 with performance gap 7.5/9. Frailty guides transplant candidacy, corticosteroid risk-benefit, and treatment intensity in elderly AH patients. Clinical Frailty Scale or Liver Frailty Index recommended
MIPS #431: Preventive Care and Screening — Unhealthy Alcohol Use	**Denominator:** patients ≥18 with ≥2 visits or ≥1 preventive visit **Numerator:** screened for unhealthy alcohol use using validated instrument AND received brief counseling or referral if positive **Exclusions:** hospice	Platform for integrating AH prevention into routine primary care; aligns with HEDIS ASF; documents the screening-to-intervention pathway that is the foundation of ALD prevention

MEASURE (MY2026)	STANDARD	APPLICABILITY TO ALCOHOLIC HEPATITIS[21]

ABBREVIATIONS: AA = Alcoholics Anonymous; ACEi = angiotensin-converting enzyme inhibitor; ACP = advance care planning; AH = alcoholic hepatitis; AHR = adjusted hazard ratio; AKI = acute kidney injury; ALD = alcohol-associated liver disease; AOR = adjusted odds ratio; ARB = angiotensin receptor blocker; ASCVD = atherosclerotic cardiovascular disease; AUD = alcohol use disorder; AUDIT-C = Alcohol Use Disorders Identification Test–Concise; AWV = Annual Wellness Visit; CGA = comprehensive geriatric assessment; CMS = Centers for Medicare & Medicaid Services; COA = Care for Older Adults; CPT = Current Procedural Terminology; DAE = dangerous/high-risk medications in the elderly; DMS-E = Depression Monitoring and Follow-Up; ECDS = Electronic Clinical Data Systems; EMR = electronic medical record; ESRD = end-stage renal disease; FIB-4 = Fibrosis-4 Index; HCPCS = Healthcare Common Procedure Coding System; HE = hepatic encephalopathy; HEDIS = Healthcare Effectiveness Data and Information Set; HR = hazard ratio; ICD-10-CM = International Classification of Diseases, 10th Revision, Clinical Modification; IET = Initiation and Engagement of Substance Use Disorder Treatment; LOINC = Logical Observation Identifiers Names and Codes; MA = Medicare Advantage; MELD = Model for End-Stage Liver Disease; MIPS = Merit-based Incentive Payment System; NIAAA = National Institute on Alcohol Abuse and Alcoholism; NSAID = nonsteroidal anti-inflammatory drug; PCR = Plan All-Cause Readmissions; PDC = proportion of days covered; PHQ-9 = Patient Health Questionnaire 9-item; PMC = Practice Metrics Committee; SASQ = Single Alcohol Screening Question; SNOMED = Systematized Nomenclature of Medicine; SUD = substance use disorder; TRC = Transitions of Care; TSC-E = Tobacco Screening and Cessation; USPSTF = United States Preventive Services Task Force; VA = Veterans Affairs.

DOCUMENTATION REMINDER

- **Lack of specificity regarding ascites status:** Loses the clinical complexity that distinguishes **K70.10** from **K70.11**
- **Failure to link Alcohol Use Disorder (AUD) to alcoholic hepatitis:** Severs the etiologic link that drives diagnosis, treatment, and recurrence prevention
- **Missing alcohol consumption pattern, quantity, and duration:** Documentation lacks the specific intake history needed to support the AH diagnosis
- **Documenting “history of alcoholic hepatitis” while disease is active:** Misrepresents active critical illness (high bilirubin, high LFTs, jaundice, active treatment) as a resolved condition
- **Overlooking AUD as the root cause:** Misses the cornerstone of preventing disease progression and high-cost readmission

5 CODING REMINDERS AND CASE EXAMPLES

Documentation Specificity

- **Stage/Severity:** Explicitly state MELD/MELD-Na and Maddrey DF with ascites status (e.g., "Severe AH with ascites, **K70.11**, MELD 24"). Add Lille score at day 7 of steroids and Clinical Frailty Scale for older adults. Accurate ascites specificity is critical

- **Etiology:** Always sequence AUD (**F10.xx**) as the root cause, specifying severity and remission status. Avoid unspecified codes (**K70.9, F10.99**) when clinical details are available to ensure proper risk documentation and treatment alignment
- **Current Status:** Document recent labs (bilirubin, INR, Cr), prognostic scores (MELD, Lille), AUD pharmacotherapy, AUDIT-C, and nephrotoxin review. For steroid patients, track course duration, infection surveillance, and glucose trends
- **Associated Conditions:** Explicitly link all comorbidities to AH in the assessment, including cirrhosis, portal hypertension, encephalopathy, AKI, and metabolic or social drivers (SDOH)
- **Chronicity:** AH is an active syndrome; do not use "history of" (**Z87.891**) while disease is active or under treatment. Reserve history codes for sustained remission with documented sobriety and normalized function

Annual Clinical Review and Confirmation

- **Annual review: K70.10 and K70.11 both map to HCC 65 (RAF ~0.185) and must be** reassessed face-to-face or via synchronous audio-video telehealth each payment year while disease remains active. Update severity (MELD, Maddrey DF where applicable), ascites status, AUD treatment status and AUDIT-C, nephrotoxin review, concurrent cirrhosis/varices/HCC (cancer) surveillance, functional status (CFS in ≥65), and SDOH
- **Visit modality: In-person or video telehealth with meaningful evaluation qualifies**. Document current labs (bilirubin, AST/ALT, INR, Cr, albumin), MELD, AUD severity and pharmacotherapy, recent hospitalizations, adherence/tolerability, nutritional status, and suicide risk assessment where indicated. HEDIS ASF Rate 1 (structured AUDIT-C, AUDIT, or NIAAA) and Rate 2 (G0443 brief counseling within 2 months of positive screen) should be satisfied each year[11]
- **Clinical context:** Under CMS-HCC V28, **K70.10/K70.11** both map to **HCC 65 at RAF ~0.185** . **Z87.891** (history of alcoholic liver disease) is acceptable only after sustained remission with normalized liver function — explicitly prohibits **Z87.891** during active disease. [1] H-1 (ascites specificity), H-2 (AUD root cause), H-4 (urgency not observation), and H-5 (nephrotoxin avoidance) are all central to annual AH review. Continuity of the clinical record matters more than any individual code
- **Avoid rollover:** Do not copy last year's AH note forward without updating MELD, ascites status, AUD treatment and AUDIT-C, nephrotoxin review, cirrhosis assessment, functional status, SDOH, and goals of care. Severity, frailty, caregiver availability, and alcohol use all shift — reassess at each annual visit and after any hospitalization. Do **NOT** default to **Z87.891** simply because the patient's AST/ALT have improved; check bilirubin, alcohol use, and sobriety duration before considering the history code

Good Documentation is Comprehensive Coding

EHR SHORTCUT/RISK	PREFERRED DOCUMENTATION LANGUAGE
Carrying 'alcoholic hepatitis' into the A&P without ascites status or severity	'Alcoholic hepatitis with ascites (**K70.11**), severe per MELD 24 and Maddrey DF 42; active moderate alcohol use disorder (**F10.20**).' Never just 'alcoholic hepatitis'
Using K70.9 when K70.10 or K70.11 is documentable	'Alcoholic hepatitis without ascites (**K70.10**) — physical exam without shifting dullness, ultrasound without ascites, bilirubin 6 mg/dL, AST/ALT 2.1.' **K70.9** does not map
Coding Z87.891 during active disease	**DO NOT Use K70.10 or K70.11 (active) with supporting labs. Z87.891** only applies after sustained sobriety with normalized liver function
Omitting F10.xx sequence (AUD as root cause)	'Active moderate alcohol use disorder (**F10.20**) — naltrexone 50 mg/d initiated today.' Sequence **F10.xx** with **K70.1x** at every encounter. ’
'Doing better — continue plan' as the only entry	'AH with ascites (**K70.11**), severe AH improving — bilirubin 8 → 5 mg/dL, MELD 24 → 19, Lille 0.38 (responder) on prednisolone day 14 of 28. Naltrexone 50 mg/d continued; AUDIT-C 0. Hepatology follow-up in 1 week.'
No AUD pharmacotherapy at AH discharge	'Discharge: Naltrexone 50 mg/d PO initiated. Addiction medicine within 2 weeks; PCP within 1 week; peer recovery support arranged. AUDIT-C at next visit (HEDIS ASF Rate 1); G0443 counseling scheduled.'[1]

ABBREVIATIONS: ACG = American College of Gastroenterology; AH = alcoholic hepatitis; ALD = alcohol-associated liver disease; AST/ALT = aspartate/alanine aminotransferase; AUD = alcohol use disorder; AUDIT-C = Alcohol Use Disorders Identification Test — Consumption; A&P = assessment and plan; CFS = Clinical Frailty Scale; CKD = chronic kidney disease; DM = diabetes mellitus; HCC = hierarchical condition category; HEDIS ASF = Unhealthy Alcohol Use Screening and Follow-Up; mDF = Maddrey discriminant function; MELD = Model for End-Stage Liver Disease; NSAID = nonsteroidal anti-inflammatory drug; PCP = primary care provider; PO = by mouth
***Consult FDA labels for the most up-to-date dosage information, contraindications, and drug-drug interactions**

EHR Workflow Tips

- **[EHR TIP — Workflow] MELD-3.0 and Severity Staging:** The MELD-3.0 calculator must auto-populate from the patient's latest Bilirubin, Sodium, INR, Creatinine, and Albumin. Confirm that a MELD-3.0 > 20 or mDF >32 triggers an immediate inpatient admission alert and a hepatology consult flag
- **[EHR TIP — Alert] Corticosteroid Candidacy and The Day 7 Lille Stop:** Configure a dynamic "Steroid Safety Checklist" alert to fire when an order for Prednisolone 40 mg is initiated for severe AH. The alert must parse the chart to verify the absence of hard exclusions: active infection, uncontrolled diabetes, or active GI bleeding. Furthermore, the EHR must hardcode an automated "Day 7 Lille Stop" task. If the Day 7 Lille score calculates at >0.45 (indicating a steroid non-responder), the system should block automatic refills,

prompt corticosteroid discontinuation, and launch an urgent liver transplant/palliative care evaluation pathway

- **[EHR TIP — Workflow] Relapse Prevention and Care Integration Loop:** EHR entry for acute AH must auto-embed a validated AUDIT-C or CAGE questionnaire directly into the intake flow. A positive score should trigger a mandatory, automated Addiction Medicine or Psychiatry co-management referral within 48 hours of evaluation. Prior to discharge, the system should surface a preference order panel for evidence-based anti-craving pharmacotherapy (e.g., Naltrexone or renally adjusted Acamprosate), capturing key value-based care (VBC) metrics aimed at preventing readmissions
- **[EHR TIP — Workflow] Acute Frailty and Transplant Readiness:** The Liver Frailty Index (LFI) and Clinical Frailty Scale (CFS) calculators should be embedded directly into the urgent inpatient/hepatology referral order set for alcohol-associated hepatitis (AH)

Brief Case Examples

SUCCESS: COMPREHENSIVE DOCUMENTATION

Scenario: 68-year-old with alcohol-associated hepatitis superimposed on alcoholic cirrhosis and Type 2 Diabetes. MELD-3.0 is 16; labs show Albumin 3.1, INR 1.4, Sodium 134, Bilirubin 1.8, Creatinine 1.1. 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. Alcohol use disorder is documented; abstinence counseling and nutritional optimization (35 kcal/kg/day, 1.2–1.5 g/kg/day protein) have been initiated.

Documentation: "Decompensated alcoholic cirrhosis with superimposed alcohol-associated hepatitis and moderate ascites (**K70.31 + K70.11**), Alcohol Use Disorder (**F10.20**), MELD-3.0 score 16 (Bili 1.8, INR 1.4, Na 134, Cr 1.1 on 5/20/26), Child-Pugh Class B. MELD ≤20 consistent with moderate AH; corticosteroids not indicated at this severity per ACG guidelines. Type 2 Diabetes (**E11.9**) managed—insulin preferred given decompensated cirrhosis per ADA Standards of Care. HCC surveillance (US/AFP) completed 5/2026—negative for malignancy; semiannual surveillance continued per AASLD guidance. Stable on Carvedilol 6.25 mg BID for portal hypertension (HR 62); Spironolactone used cautiously for volume control with close AKI monitoring given heightened renal risk in AH. Nutritional plan (35 kcal/kg/day, 1.2–1.5 g/kg protein) and thiamine/B-vitamin supplementation in place. Abstinence counseling reinforced; liver transplant evaluation to be considered if MELD remains >17 after sustained abstinence. MEAT documented."

Outcome: HCC 27 (Alcoholic Hepatic Failure – 0.902) + HCC 18 (Diabetes with Chronic Complications – 0.166) + HCC 139 (Alcohol Use Disorder – 0.242), for a combined RAF of 1.310, representing approximately $13,627/year in risk-adjusted care resources at a base rate of $10,402.

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 28 but fails to document decompensation or etiology. Chronic hepatic failure (**K72.10/K72.11**), which maps to HCC 27 (RAF 0.902), is never triggered because ascites, hepatic encephalopathy, and hepatic failure are not coded as active diagnoses. Using **Z87.19** ("history of liver disease") instead of an active cirrhosis

code zeroes out the HCC entirely. Documenting unspecified cirrhosis (**K74.60**) rather than alcoholic cirrhosis with ascites (**K70.31**) obscures the etiology and decompensation status. Alcohol use disorder (**F10.X**) maps to HCC 139 and must be coded separately from alcohol-associated hepatitis.

RAF Impact:Potential loss of HCC 27 (0.902), coded instead as HCC 28 (0.362) or missed entirely. If downgraded from HCC 27 to HCC 28, the RAF differential of 0.540 represents ≈$5,617/year. If missed entirely, the full HCC 27 loss equals ≈$9,383/year. Additional loss of HCC 139 (0.242) from uncoded Alcohol Use Disorder adds ≈$2,517/year. Combined potential gap of ≈$8,134–$11,900/year in unrepresented care resources depending on segment.

Fix: "Decompensated alcoholic cirrhosis with superimposed alcohol-associated hepatitis and moderate ascites (**K70.31 + K70.11**). Alcohol Use Disorder, moderate, in early remission (**F10.20**). MELD-3.0 score 16 (Bili 1.8, INR 1.4, Na 134, Cr 1.1 on 5/20/26), Child-Pugh Class B. MELD ≤20 — corticosteroids not indicated per ACG guidelines at this severity. Type 2 Diabetes with chronic complications (**E11.9**) managed; insulin preferred given decompensated cirrhosis. Ascites evaluated; Carvedilol titrated for portal hypertension prophylaxis. Spironolactone continued with close AKI monitoring given heightened renal risk in AH. Nutritional plan (35 kcal/kg/day, 1.2–1.5 g/kg protein) and thiamine supplementation in place. Abstinence counseling reinforced; transplant evaluation pathway discussed. MEAT documented."

REFERENCES

1.Bataller R, Arab JP, Shah VH. Alcohol-associated hepatitis. *N Engl J Med*. 2022;387(26):2436-2448.

2.Crabb DW, Im GY, Szabo G, Mellinger JL, Lucey MR. Diagnosis and treatment of alcohol-associated liver diseases: 2019 practice guidance from the American Association for the Study of Liver Diseases. *Hepatology*. 2020;71(1):306-333.

3.Singal AG, Llovet JM, Yarchoan M, et al. AASLD Practice Guidance on prevention, diagnosis, and treatment of hepatocellular carcinoma. *Hepatol Baltim Md*. 2023;78(6):1922-1965. doi:10.1097/HEP.0000000000000466

4.Geng CX, Gudur AR, Patterson DR, Stotts MJ. Concordance of ICD-10 Codes and the Clinical Diagnosis of Alcoholic Hepatitis. *Am J Gastroenterol*. 2022;117(10):1706-1708. doi:10.14309/ajg.0000000000001886

5.ICD-10 | CMS. Accessed April 20, 2026. https://www.cms.gov/medicare/coding-billing/icd-10-codes

6.Arnold MJ. Management of Rheumatoid Arthritis: Update From ACR. *Am Fam Physician*. 2022;106(3):340-342.

7.Brown CL, Bajaj JS. Considerations in the Diagnoses and Management of Hepatic Disease in Older Adults. *Off J Am Coll Gastroenterol ACG*. 2025;120:S56-S66.

8.Jophlin LL, Singal AK, Bataller R, et al. ACG Clinical Guideline: Alcohol-Associated Liver Disease. *Am J Gastroenterol*. 2024;119(1):30-54. doi:10.14309/ajg.0000000000002572

9.Alcohol-Related Liver Disease - Hepatology. Merck Manual Professional Edition. Accessed June 7, 2026. https://www.merckmanuals.com/professional/hepatic-and-biliary-disorders/alcohol-related-liver-disease/alcohol-related-liver-disease

10.Brown CL, Bajaj JS. Considerations in the Diagnoses and Management of Hepatic Disease in Older Adults. *Off J Am Coll Gastroenterol ACG*. 2025;120:S56-S66.

11.Lai JC, Tandon P, Bernal W, et al. Malnutrition, Frailty, and Sarcopenia in Patients With Cirrhosis: 2021 Practice Guidance by the American Association for the Study of Liver Diseases. *Hepatol Baltim Md*. 2021;74(3):1611-1644. doi:10.1002/hep.32049

12.Mitchell MC, McClain CJ. Medical Management of Severe Alcoholic Hepatitis. *Clin Gastroenterol Hepatol Off Clin Pract J Am Gastroenterol Assoc*. 2017;15(1):. doi:10.1016/j.cgh.2016.08.047

13.Singal AK, Zhang W, Shetty A, et al. Treatment of alcohol use disorder in alcohol-associated liver disease: A meta-analysis. *Hepatol Commun*. 2025;9(5):e0686. doi:10.1097/HC9.0000000000000686

14.Wijdicks EFM. Hepatic Encephalopathy. Longo DL, ed. *N Engl J Med*. 2016;375(17):1660-1670. doi:10.1056/NEJMra1600561

15.Karvellas CJ, Bajaj JS, Kamath PS, et al. AASLD Practice Guidance on Acute-on-chronic liver failure and the management of critically ill patients with cirrhosis. *Hepatology*. 2024;79(6):1463-1502. doi:10.1097/HEP.0000000000000671

16.Kim WR, Mannalithara A, Heimbach JK, et al. MELD 3.0: the model for end-stage liver disease updated for the modern era. *Gastroenterology*. 2021;161(6):1887-1895.

17.Biggins SW, Angeli P, Garcia-Tsao G, et al. Diagnosis, Evaluation, and Management of Ascites, Spontaneous Bacterial Peritonitis and Hepatorenal Syndrome: 2021 Practice Guidance by the American Association for the Study of Liver Diseases. *Hepatol Baltim Md*. 2021;74(2):1014-1048. doi:10.1002/hep.31884

18.Altamirano J, López-Pelayo H, Michelena J, et al. Alcohol abstinence in patients surviving an episode of alcoholic hepatitis: Prediction and impact on long-term survival. *Hepatol Baltim Md*. 2017;66(6):1842-1853. doi:10.1002/hep.29338

19.Simonetto DA, Shah VH, Kamath PS. Outpatient management of alcohol-related liver disease. *Lancet Gastroenterol Hepatol*. 2020;5(5):485-493. doi:10.1016/S2468-1253(19)30415-7

20.Tyson LD, Atkinson S, Hunter RW, et al. In severe alcohol-related hepatitis, acute kidney injury is prevalent, associated with mortality independent of liver disease severity, and can be predicted using IL -8 and micro-RNAs. *Aliment Pharmacol Ther*. 2023;58(11-12):1217-1229. doi:10.1111/apt.17733

21.Centers for Medicare & Medicaid Services. Medicare 2026 Part C & D Star Ratings Technical Notes. Published online 2025. https://www.cms.gov/medicare/health-drug-plans/part-c-d-performance-data

22.Arnold MJ. Alcohol-Associated Liver Disease: Guidelines From the American College of Gastroenterology. *Am Fam Physician*. 2025;111(3):284-286.

23.Adejumo AC, Cholankeril G, Iqbal U, et al. Readmission Rates and Associated Outcomes for Alcoholic Hepatitis: A Nationwide Cohort Study. *Dig Dis Sci*. 2020;65(4):990-1002. doi:10.1007/s10620-019-05759-4

24.Serper M, Kaplan DE, Shults J, et al. Quality Measures, All-Cause Mortality, and Health Care Use in a National Cohort of Veterans With Cirrhosis. *Hepatol Baltim Md*. 2019;70(6):2062-2074. doi:10.1002/hep.30779

Medical Disclaimer

The information provided by the American Academy of Value-Based Care (AAVBC) in this document, including but not limited to coding guidance, and related content, is intended for educational and informational purposes only. This content is designed to assist healthcare providers, organizations,

and professionals in understanding and applying Value-Based Care principles, practices, and regulatory compliance standards.

AAVBC does not provide legal, clinical, or medical advice. The content presented should not be interpreted or relied upon as specific legal, medical, clinical, or professional guidance. While efforts are made to ensure the accuracy and currency of the information provided, AAVBC does not guarantee that the materials presented are complete, comprehensive, or without error.

Providers and healthcare professionals must independently evaluate all content and guidance presented herein in the context of applicable laws, regulations, clinical guidelines, payer policies, patient-specific conditions, and contraindications. Any treatments, procedures, diagnostic approaches, medications, or strategies discussed are illustrative and should not be directly applied without a thorough and independent review of current, evidence-based clinical resources, and regulatory requirements.

For coding, documentation, utilization management, quality measures (including STAR ratings), and compliance matters, users are responsible for consulting official guidelines issued by regulatory bodies, including but not limited to the Centers for Medicare and Medicaid Services (CMS), the American Medical Association (AMA), relevant state agencies, and other authoritative entities.

AAVBC expressly disclaims liability for any loss, claim, or damages arising directly or indirectly from the use or reliance on information provided in this document. Users should consult their organization's legal counsel, compliance officer, or qualified professional advisor for advice specific to their situation. By accessing and using this document, you agree to AAVBC's terms and acknowledge that the use of the content is at your own risk and discretion.