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Beyond the Bottle: A Practical Guide to Recognizing Problem Drinking, Identifying Liver Fibrosis and Knowing When to Refer

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Abstract

Alcohol use and maladaptive coping behaviors, including excessive consumption of ultra-processed foods, are increasingly recognized as important contributors to both psychiatric illness and chronic liver disease. Many patients self-treat anxiety, insomnia, trauma, chronic pain, or mood disorders with alcohol without recognizing hazardous drinking patterns. In the setting of the global rise in Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD), even modest alcohol intake may accelerate progression to Alcohol-Associated Liver Disease (ALD), advanced fibrosis, cirrhosis, and hepatocellular carcinoma. These risks are particularly concerning given the marked rise in alcohol use disorder and alcohol-related liver disease among women. Because advanced fibrosis is often clinically silent, subtle laboratory abnormalities, incidental imaging findings, or mild cognitive and functional decline may be the earliest indicators of liver injury. Mental health clinicians, who often follow patients longitudinally, are uniquely positioned to identify these red flags and facilitate timely referral for further evaluation. This practical commentary highlights the intersection of psychiatric illness, alcohol use, metabolic dysfunction, and chronic liver disease while providing clinicians with key clinical clues that warrant referral to primary care, gastroenterology, or hepatology, where early intervention may prevent progression and even allow regression of liver fibrosis.

Keywords: MASLD, Metabolic dysfunction associated steatotic liver disease, Alcoholic liver disease, Liver fibrosis, Cirrhosis, Hepatocellular carcinoma, Liver function tests, FIB-4 index

Who is at Higher Risk (Even If They Deny, "Heavy Drinking")? Mental Health Settings Often See These Profiles First

- **Alcohol used as a medication**: Nightly use for sleep; "as-needed" use for panic/anxiety; escalating dose over time; or morning use to steady nerves or reduce nausea.
- **History or current alcohol use disorder**
- **Metabolic syndrome / MASLD[*] risk** (central obesity, type 2 diabetes, dyslipidemia, and hypertension): MASLD increases baseline fibrosis risk; alcohol can accelerate progression even at amounts the patient considers "moderate" [1]. [*]MASLD is the new nomenclature for previously NAFLD (Nonalcoholic fatty liver disease).
- **History of bariatric surgery** or rapid weight changes (altered alcohol pharmacokinetics and higher vulnerability to alcohol-related harm).
- **Psychiatric comorbidity plus episodic binges** (weekend/"reset" drinking, blackout history, recurrent ED/urgent care visits), even if average weekly intake seems low.
- **Polysubstance exposure and medication interactions** (e.g., alcohol plus acetaminophen-containing products, sedative-hypnotics, or other hepatically metabolized medications).
- **Unexplained functional decline** (Fatigue, reduced concentration, daytime sleepiness) that is attributed to depression/anxiety but persists despite adequate psychiatric treatment.
- **Women are more vulnerable to effects of alcohol due to**:
    - First-pass metabolism resulting in decreased gastric oxidation of ethanol compared to men [2].

- Lower gastric mucosal alcohol dehydrogenase activity [2].
- Lower levels of cytochrome P4502E1, important enzyme for alcohol metabolism [3],
- Increase Kupffer cell (specialized liver cells) activation by estrogens leading to cytokine release and inflammation [3].
- Theory that women have less water in their bodies than men, leading to smaller volume of distribution and higher concentrations of alcohol in blood with harmful levels of exposure to organs [4].

Referral Red Flags: When to Involve Hepatology (Even If the Patient Looks "Well")

- **Any evidence of impaired synthetic function:** Low albumin, rising INR or Protime and low platelet (<150k including low normal values). The liver is the site of production of albumin, vitamin K clotting factors and thrombopoietin, the hormone that induces platelet production from the bone marrow
- **Persistently abnormal liver transaminases:** (e.g., AST: ALT>1, elevated GGT) for>6 months, or any unexplained mixed hepatocellular–cholestatic pattern (elevated total bilirubin, elevated alkaline phosphatase and elevated transaminases).
- **High-risk noninvasive fibrosis screen (FIB-4):** >2.67 suggests higher likelihood of advanced fibrosis; 1.3–2.67 is indeterminate and should prompt additional assessment (e.g., FibroScan elastography), especially when combined with metabolic syndrome or ongoing alcohol exposure. (See Quick Tool below for details).
- **New or disproportionate neuropsychiatric change:** (Reversal of sleep-wake cycle, fluctuating attention, slowed processing, new irritability/apathy) in a patient with liver disease risk factors including metabolic syndrome, consider covert hepatic encephalopathy and coordinate medical evaluation urgently.
- **Any decompensation features:** (Ascites, GI bleeding/hematemesis/melena, jaundice, confusion) are urgent referral/emergency evaluation triggers.

Common Labs You May Already See (Or Can Request) That Should Prompt Targeted Alcohol + Fibrosis Screening

- **AST: ALT ratio>1** (and particularly >1.5) with modest absolute elevations (often both <400 IU/L) can suggest alcohol-associated liver injury, and may also be seen in advanced fibrosis/cirrhosis from multiple etiologies including hemochromatosis, hepatitis B, untreated hepatitis C, autoimmune hepatitis.
- **Disproportionate GGT elevation** (especially if persistent) may reflect hepatobiliary inflammation and is commonly seen with chronic alcohol exposure and MASLD; it may also rise with cholestasis and certain medications, thus, persistence and context matter [5].
- **Thrombocytopenia** as discussed above. In outpatient mental health settings, this is easy to miss if only AST/ALT are reviewed.
- **Low albumin and/or rising INR** as discussed above and should prompt evaluation for advanced liver disease rather than attribution to "poor nutrition"
- **Macrocytosis (elevated MCV)** with or without anemia may be associated with alcohol use (and/or folate deficiency, hypothyroidism, marrow disorders). When paired with sleep/anxiety "self-medication," falls, or liver enzyme abnormalities, it strengthens suspicion for ongoing alcohol exposure.
- **Unexplained hyperbilirubinemia** (especially direct/conjugated) or a mixed hepatocellular–cholestatic pattern should trigger assessment for alcoholic hepatitis, biliary disease, and advanced fibrosis.
- **Elevated triglycerides** (and/or poorly controlled diabetes) may point to metabolic syndrome, which increases baseline fibrosis risk and can also confound pancreatitis risk, particularly when combined with alcohol use.
- **Elevated HDL-C** can point to alcohol use with magnitude of elevation in linear association with number of drinks per day [6].

Normal or near-normal ALT and AST do not exclude advanced fibrosis; enzyme levels can fall as hepatocyte mass declines. When the above patterns are present (or when metabolic risk is high), a brief, practical approach is to (1) review trends in platelets, albumin, INR, bilirubin, and MCV (not just AST/ALT), (2) ask about alcohol in concrete terms (quantity, frequency, binges, and morning use), and (3) calculate a noninvasive score such as FIB-4 using age, AST, ALT, and platelets. If risk is indeterminate or high—or if there are any synthetic function abnormalities or portal hypertension features—coordinate prompt medical evaluation (PCP/urgent care as appropriate) and refer to hepatology for elastography and etiologic workup.

Quick Tool: FIB-4 to Screen for Probable Advanced Fibrosis

FIB-4 is a simple, noninvasive score that uses age, AST, ALT, and platelet count to estimate the likelihood of clinically significant liver fibrosis. It is most useful in patients with liver risk factors (e.g., alcohol exposure and/or metabolic syndrome/MASLD) and can help you decide who needs elastography and/or hepatology evaluation. FIB-4 calculators are available on the internet for ease of calculations. One site recommended by this author is from the Liver Foundation.

Formula: FIB-4 = (Age × AST) ÷ (Platelets × √ALT) [Age in years; AST/ALT in IU/L; platelets in 10^9/L]

- **FIB-4 <1.3 (typical adult cut-point):** Lower likelihood of advanced fibrosis. Guidance for primary care providers is to repeat periodically for individuals with ongoing alcohol exposure and diabetes/obesity.
- **FIB-4 1.3–2.67**: Indeterminate. Consider additional evaluation (e.g., transient elastography) and coordinate with primary care; refer to hepatology if other red flags are present (platelets trending down, low albumin/rising INR, abnormal imaging, or concerning symptoms).
- **FIB-4 > 2.67**: Higher likelihood of advanced fibrosis—generally warrants elastography and hepatology referral.
- **Age caveat:** In adults >65, baseline FIB-4 tends to run higher; some pathways use a higher "rule-out" threshold (e.g., 2.0) to reduce false positives. When in doubt—especially with any synthetic dysfunction or portal hypertension features refer.

Subtle Clinical Signs and History Cues (Often Present Before Decompensation)

Decompensation would likely be very apparent, and moot to the substance of this commentary

- **Soft stigmata of chronic liver disease:** Scattered spider telangiectasias, palmar erythema, easy bruising, or mild lower-extremity edema that is otherwise unexplained.
- **Medication and symptom patterns consistent with self-treatment:** Escalating use of alcohol for sleep, anxiety, grief, chronic pain, or appetite; morning nausea, "shakes," or needing a drink to feel normal.
- **Recurrent injuries or falls:** Episodic hypertension or tachycardia, or ED visits for gastritis, vomiting, or nonspecific abdominal pain.
- **Neurocognitive or sleep-wake changes that don't fit the psychiatric picture:** new daytime somnolence, reversal of sleep schedule, fluctuating attention, slowed processing, irritability/apathy, or a "brain fog" that is out of proportion to depression/anxiety severity—particularly when accompanied by liver-risk labs/imaging—should raise concern for covert hepatic encephalopathy or alcohol-related neurotoxicity and prompt medical evaluation.
- **Psychopharmacology "tolerance" or adverse effects at low doses:** unexpected sedation, confusion, falls, or medication sensitivity (e.g., to benzodiazepines, sedative-hypnotics, certain antipsychotics) can be a clue to impaired hepatic metabolism or early encephalopathy, especially when paired with thrombocytopenia, low albumin, or abnormal imaging.
- **Imaging "incidentalomas" that are easy to dismiss:** Fatty liver, nodular contour, splenomegaly, enlarged portal vein, or recanalized umbilical vein, features that may indicate portal hypertension even in a patient who feels well.

These patterns are particularly important in patients with metabolic syndrome (central obesity, type 2 diabetes, hypertension, and dyslipidemia). MASLD already confers risk for progressive fibrosis; when alcohol is added, even if the patient views intake as "moderate" or therapeutic, the trajectory can accelerate, and clinicians may encounter significant underlying fibrosis or compensated cirrhosis earlier than expected [7]. For mental health clinicians, this means that a patient with depression/anxiety plus diabetes/obesity and "nightly drinks for sleep" can already be in a high-risk category despite minimal symptoms. Because hepatocellular carcinoma (HCC) risk rises substantially once cirrhosis is present, identifying combined metabolic + alcohol exposure is a key reason to initiate fibrosis staging (FIB-4/elastography) and refer to hepatology when screens or labs are concerning.

A Quick, Scriptable Checklist for Psychiatry/Psychology Visits

- **Ask concretely:** "In the past 30 days, how many days per week did you drink?" "On a typical drinking day, how many drinks?" "Any binges?" "Any morning drinking to feel normal?" "Do you use alcohol specifically for sleep or anxiety?"
- **Look for silent advanced disease:** Platelets trend, albumin, INR, MCV; don't be reassured by normal AST/ALT alone.
- **Review prior imaging comments (CT/US/MRI):** "Fatty liver," "nodular," "splenomegaly," "portal vein enlargement," "varices."

- **Calculate the FIB-4 index:** If over 1.3, consider referring. If >2, refer.
- **Escalate care:** If any referral red flags are present (synthetic dysfunction, thrombocytopenia with risk factors, portal hypertension features, concerning fibrosis score, or new fluctuating cognition/somnolence) or FIB-4 index is >2.67.

Don't Miss Concomitant Pancreatic Disease in Alcohol Self-Treatment

Alcohol self-treatment can also mask or delay recognition of pancreatic injury. Patients may normalize recurrent epigastric discomfort, nausea, or intermittent vomiting, and clinicians may attribute symptoms to reflux, gastritis, or "stress." Recurrent alcohol exposure is a leading cause of both relapsing acute pancreatitis and chronic pancreatitis, with downstream risks including malnutrition, chronic pain, diabetes, and pancreatic pseudocysts [8].

- **Recurrent epigastric pain** (often radiating to the back), particularly if episodic and associated with nausea or vomiting.
- **Intermittent or persistent lipase/amylase elevations**, especially if previously documented during ED/urgent care visits.
- **Unexplained weight loss, steatorrhea, or fat-soluble vitamin deficiencies**, which may indicate exocrine pancreatic insufficiency in chronic pancreatitis.
- **Worsening glycemic control** or new diabetes in a patient with abdominal symptoms, suggesting endocrine dysfunction from pancreatic injury.
- **Hypertriglyceridemia** (common in metabolic syndrome) which can independently trigger pancreatitis and may compound alcohol-related risk.

In practice, a brief, routine set of steps can reduce missed disease: ask specifically about alcohol use as a coping strategy (quantity, frequency, binges, and morning use), review trends in platelets, albumin, INR, and MCV—not just ALT/AST—and calculate a simple fibrosis score when risk factors or unexplained abnormalities are present. Early identification of advanced fibrosis or compensated cirrhosis enables targeted counseling on alcohol cessation, metabolic risk modification, timely hepatology referral, and consideration of appropriate surveillance for complications such as portal hypertension and HCC. Similarly, recognizing recurrent abdominal symptoms or enzyme elevations in this context may identify occult pancreatitis before irreversible complications develop.

Disclaimer

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