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Alcohol-Associated Decompensated Cirrhosis with Ascites and Suspected Hepatic Encephalopathy: A Case Report

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ABSTRACT

Introduction: Alcohol-associated cirrhosis is a preventable cause of advanced liver disease, yet it is frequently first recognized after decompensation. This case underscores the diagnostic value of integrating clinical decompensating features with non-invasive fibrosis indices and ultrasonography in a resource-limited setting.

Case Summary: A 51-year-old man with a 20-year history of daily alcohol intake presented with progressive abdominal distension, bilateral leg edema, right hypochondrial pain, nausea, jaundice, weight loss, and recent behavioral change. He had previous hematemesis and melena but had declined esophagogastroduodenoscopy. Examination revealed pallor, scleral icterus, ascites, right hypochondrial tenderness, and peripheral edema. Investigations showed anemia, thrombocytopenia, AST-predominant transaminitis, hypoalbuminemia, mild hyponatremia, and non-reactive HBsAg. APRI and FIB-4 were markedly elevated, and ultrasonography demonstrated a shrunken, coarse, irregular liver with ascites, supporting a diagnosis of presumed alcohol-associated decompensated cirrhosis. Behavioral changes without impaired consciousness were clinically suggestive of early hepatic encephalopathy; however, covert hepatic encephalopathy could not be formally confirmed because psychometric testing was not available.

Discussion: Ascites, jaundice, cytopenia, hypoalbuminemia, and neuropsychiatric change indicated advanced decompensation, while suspected portal hypertension remained incompletely characterized because endoscopy was unavailable. Management included dietary sodium and fluid restriction, nutritional support, large-volume paracentesis, albumin infusion, empiric antibiotic therapy, acid suppression, ursodeoxycholic acid, diuretic therapy, lactulose, and a non-selective beta-blocker.

Conclusion: This case emphasizes early alcohol-use assessment, abstinence-centered care, timely endoscopic risk stratification, and complication-directed management to reduce preventable deterioration in alcohol-associated decompensated cirrhosis.

Keywords: Alcohol-associated liver disease; decompensated cirrhosis; ascites; hepatic encephalopathy; case report

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Introduction

Cirrhosis represents the advanced stage of chronic liver injury, characterized by progressive fibrosis, regenerative nodules, distortion of hepatic architecture, portal hypertension, and gradual loss of hepatocellular function [1–3]. Although patients may remain clinically silent during the compensated phase, the onset of ascites, jaundice, variceal bleeding, infection, renal dysfunction, or hepatic encephalopathy marks the transition to decompensated disease and is associated with substantially poorer outcomes [4]. Recent Global Burden of Disease estimates reported approximately 58.4 million incident cases of liver cirrhosis in 2021, increasing from 36.9 million in 1990, while deaths related to cirrhosis and other chronic liver diseases exceeded 1.4 million in 2021 [5,6].

Alcohol-associated liver disease is one of the most important preventable causes of cirrhosis. Sustained alcohol exposure may produce a continuum of hepatic injury, ranging from steatosis and steatohepatitis to advanced fibrosis and decompensated cirrhosis [7–10]. Its burden is clinically relevant because many patients are diagnosed late, when complications such as ascites, jaundice, anemia, thrombocytopenia, malnutrition, and neuropsychiatric manifestations have already developed. Contemporary estimates show that alcohol-associated liver disease affects approximately 3.5% of the general population and is substantially more frequent among hazardous drinkers and individuals with alcohol use disorder [11].

In Indonesia, cirrhosis remains an important clinical problem, although national population-based data are limited. The Indonesian clinical practice guideline for adult liver cirrhosis notes a substantial national burden of cirrhosis-related mortality, with Social Security Agency on Health (BPJS Kesehatan) data documenting 2,159 deaths attributed collectively to cirrhosis and liver cancer in 2022 [12]. This combined figure does not represent cirrhosis-specific mortality. These data highlight the need for early recognition of advanced liver disease, especially in patients with long-standing risk factors such as chronic alcohol consumption.

Diagnosis of decompensated cirrhosis requires integration of clinical findings, laboratory abnormalities, non-invasive fibrosis indices, and imaging features [13]. In settings where advanced diagnostic procedures are not immediately obtained, findings such as ascites, jaundice, thrombocytopenia, hypoalbuminemia, altered behavior, APRI, FIB-4, and ultrasonography may provide a practical framework for recognizing advanced liver disease and initiating early complication-directed management [14]. We report a case of presumed alcohol-associated decompensated cirrhosis in a 51-year-old man in whom diagnosis and initial management were established primarily from clinical assessment, routinely available laboratory investigations, and abdominal ultrasonography. The educational value of this case lies in demonstrating a pragmatic approach to recognizing and managing decompensated cirrhosis when diagnostic evaluation is incomplete and more definitive assessment, particularly endoscopic evaluation of portal hypertension, has not yet been obtained.

Case Report

A 51-year-old married man presented to the emergency department with progressive abdominal distension for approximately one month, accompanied by bilateral lower-limb swelling. He also reported right-sided abdominal pain for five days, nausea, progressive yellow discoloration of the eyes and skin for one week, and unintentional weight loss over the preceding five months. During the same week, his family noticed behavioral changes, including reduced communication and irritability, raising suspicion for covert hepatic encephalopathy. He had a history of hematemesis and melena in the previous year but declined esophagogastroduodenoscopy and further inpatient evaluation at that time. He had consumed alcohol daily for approximately 20 years, although the exact amount could not be quantified, and had a 20-pack-year smoking history.

On admission, the patient was conscious and hemodynamically stable, with a blood pressure of 100/55 mmHg, pulse rate of 78 beats/minute, respiratory rate of 20 breaths/minute, temperature of 36.4°C, and oxygen saturation of 100% on room air. Relevant physical findings included pale conjunctivae, scleral icterus, a protuberant abdomen with positive shifting dullness, tenderness over the right hypochondriac region, and bilateral lower-limb edema. No clinically apparent hepatosplenomegaly was detected. Peripheral stigmata of chronic liver disease, including palmar erythema, spider angioma, and gynecomastia, were not observed, while testicular atrophy was not assessed. Tremor and asterixis were not documented. The reported reduction in communication and irritability were considered clinically suggestive of early hepatic encephalopathy; however, these findings were nonspecific, and covert hepatic encephalopathy could not be formally confirmed because psychometric testing was not available.

Laboratory evaluation showed bicytopenia characterized by anemia and thrombocytopenia, with hemoglobin 11.44 g/dL, hematocrit 35.8%, erythrocytes 4.302 $\times 10^6/\mu L$, platelets 80 $\times 10^3/\mu L$, and a leukocyte count of 8.26 $\times 10^3/\mu L$. Liver biochemistry demonstrated AST-predominant transaminitis, with AST 203.70 U/L and ALT 54.00 U/L. Serum albumin was low, with recorded values of 2.1 and 2.0 g/dL during hospitalization; the exact timing of the repeat measurement relative to intravenous albumin administration was not documented in the available record. Renal function was preserved, with BUN 7.14 mg/dL and creatinine 0.645 mg/dL. Mild hyponatremia was present, with sodium 134.80 mmol/L. HBsAg was non-reactive. Urinalysis showed protein 2+, bilirubin 2+, urobilinogen 2+, leukocytes 2+, and bacteriuria. Further etiological evaluation, including testing for hepatitis C and other causes of chronic liver disease, could not be completed because of facility and financial limitations. Moreover, serum bilirubin and coagulation parameters, including PT/INR, were not available during this admission; therefore, disease severity could not be fully assessed using the Child–Pugh and MELD-Na scores.

Non-invasive fibrosis assessment strongly supported cirrhosis, with an AST-to-platelet ratio index of 7.3 and a Fibrosis-4 index of 17.67. Abdominal ultrasonography demonstrated a shrunken liver with coarse

increased echotexture, irregular surface, and ascites (Figure 1). The working diagnosis of presumed alcohol-associated decompensated cirrhosis was made based on the integration of long-term alcohol exposure, ascites, jaundice, thrombocytopenia, hypoalbuminemia, AST-dominant aminotransferase elevation, high APRI and FIB-4 scores, and cirrhotic morphology on ultrasonography. The main clinical manifestations were ascites, jaundice, anemia, thrombocytopenia, lower-limb edema, and behavioral change suggestive of covert hepatic encephalopathy. Portal hypertension was suspected from the presence of ascites and thrombocytopenia, although varices were not confirmed because esophagogastroduodenoscopy (EGD) had not been performed as the patient had previously refused it.

The initial diagnostic approach therefore relied on investigations available in the documented clinical work-up, including complete blood count, liver aminotransferases, serum albumin, renal function, serum sodium, HBsAg, urinalysis, non-invasive fibrosis indices, and abdominal ultrasonography. These findings were sufficient to support advanced chronic liver disease with clinical decompensation and to guide initial treatment. Endoscopic assessment of portal hypertension was not obtained because the patient had previously declined EGD, and other investigations not documented in the available work-up were not used to establish the initial diagnosis.

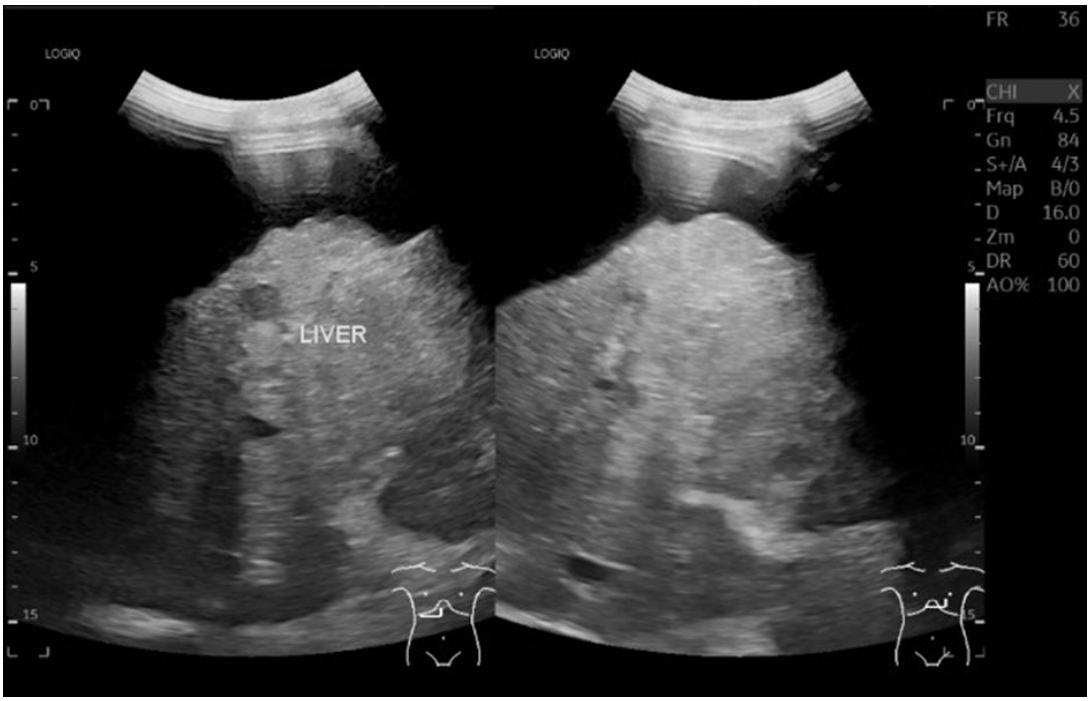

Figure 1. Abdominal Ultrasonography Showing Cirrhotic Liver Morphology with Ascites. The liver appears shrunken with coarse increased echotexture and an irregular surface, accompanied by ascites, consistent with hepatic cirrhosis.

Management focused on controlling decompensated cirrhosis and its associated complications. The patient received dietary sodium restriction as part of ascites management, together with a low-fat, high-protein diet. Therapeutic paracentesis was performed with removal of 6 L of ascitic fluid. The patient also received 100 mL of 25% intravenous albumin during hospitalization. The serum albumin level was 2.0 g/dL before albumin

administration and increased to 2.1 g/dL following the infusion. Other pharmacological treatment included intravenous cefuroxime 1 g three times daily as empiric antibiotic therapy for suspected infection, intravenous metamizole 1 g three times daily, intravenous pantoprazole 40 mg once daily (as acid-suppressive therapy in the context of gastrointestinal symptoms and the patient's previous history of hematemesis and melena), intravenous furosemide 40 mg once daily, oral spironolactone 100 mg once daily, oral lactulose 5 mL three times daily, oral propranolol 20 mg twice daily, and oral ursodeoxycholic acid 250 mg twice daily.

During the five-day hospitalization, no active gastrointestinal bleeding, renal failure, or overt deterioration in consciousness was documented. Clinical improvement was observed, with reduced abdominal distension and lower-limb edema, improvement in abdominal pain, and better communication and behavior. Renal function remained preserved, with adequate urine output, and no significant treatment-related adverse events were documented. The patient was considered clinically stable for discharge and was discharged with dietary advice, oral medications, counseling regarding alcohol abstinence, and scheduled internal medicine follow-up. However, he did not return for the scheduled follow-up; therefore, longer-term clinical outcomes and completion of further diagnostic evaluation could not be assessed.

Discussion

This case illustrates presumed alcohol-associated decompensated cirrhosis presenting with ascites, jaundice, anemia, thrombocytopenia, hypoalbuminemia, and behavioral changes suggestive of covert hepatic encephalopathy. The diagnosis was supported by a long history of daily alcohol consumption, AST-predominant aminotransferase elevation, thrombocytopenia, hypoalbuminemia, markedly elevated APRI and FIB-4 scores, and ultrasonographic evidence of a shrunken liver with coarse echotexture, irregular surface, and ascites. These findings are consistent with advanced chronic liver disease complicated by clinical decompensation.

The bicytopenia in this patient consisted of anemia and thrombocytopenia with a preserved leukocyte count. These hematologic abnormalities were likely multifactorial, with possible contributions from portal hypertension and hypersplenism, nutritional deficiency, alcohol-associated bone marrow suppression, and the previous history of gastrointestinal bleeding. Further hematologic evaluation was not available to determine the relative contribution of each mechanism.

The clinical importance of this case lies in the late recognition of alcohol-associated cirrhosis after the development of multiple decompensating features. Cirrhosis may remain asymptomatic during the compensated phase, whereas decompensation is commonly marked by ascites, variceal bleeding, jaundice, or hepatic encephalopathy and is associated with more rapid progression toward death or liver transplantation [15]. In this patient, ascites was the dominant presentation, accompanied by jaundice, peripheral edema, hypoalbuminemia, thrombocytopenia, and neuropsychiatric change. This constellation supports a transition

from chronic compensated liver injury to clinically evident decompensated cirrhosis.

Alcohol-associated liver disease was considered the most likely etiology in this patient. He had consumed alcohol daily for approximately 20 years; however, the exact amount of alcohol intake could not be reliably quantified. The available etiological evaluation was limited, with HBsAg being non-reactive, while hepatitis C and other causes of chronic liver disease could not be fully excluded because of facility and financial limitations. Therefore, considering the long-term alcohol exposure together with the clinical, biochemical, and ultrasonographic findings, the diagnosis was regarded as presumed alcohol-associated decompensated cirrhosis.

Sustained alcohol exposure can progress from steatosis and steatohepatitis to fibrosis and cirrhosis, and long-standing intake of more than 40 g/day has been associated with cirrhosis in a substantial proportion of exposed individuals [9,16]. Recent epidemiological data also show that alcohol-associated liver disease remains a major global problem, with an estimated prevalence of 3.5% in the general population, 26.0% among hazardous drinkers, and 55.1% among individuals with alcohol use disorder [11]. In the present case, the negative HBsAg result and the absence of stronger alternative etiologies in the available work-up further supported alcohol-associated liver disease as the leading cause.

Ascites and thrombocytopenia suggested clinically significant portal hypertension, although this could not be fully characterized because EGD was not performed. This limitation is clinically relevant because the patient had a prior history of hematemesis and melena, which raises concern for previous variceal bleeding. Endoscopic evaluation would have been important for detecting gastroesophageal varices and guiding primary or secondary prophylaxis. Current guidance emphasizes complication-directed management in decompensated cirrhosis, including ascites control, infection prevention, evaluation for varices, and treatment of hepatic encephalopathy when clinically indicated [1,2,17].

An important practical aspect of this case was that the initial diagnosis did not depend on liver biopsy, transient elastography, or endoscopic confirmation. Instead, the clinical diagnosis was established by combining a long history of alcohol exposure with recognizable decompensating features, routine laboratory abnormalities, markedly elevated APRI and FIB-4 values, and ultrasonographic evidence of cirrhotic morphology. This approach is particularly relevant in settings where the diagnostic work-up is incomplete or where advanced investigations cannot be obtained immediately. In such circumstances, primary and general-care physicians should recognize that the presence of ascites, jaundice, thrombocytopenia, hypoalbuminemia, and compatible ultrasonographic findings may be sufficient to identify probable decompensated cirrhosis, initiate appropriate supportive and complication-directed treatment, and arrange referral or further evaluation for portal hypertension and other complications.

The suspected hepatic encephalopathy should be interpreted cautiously. The patient's family reported reduced communication and irritability, while he remained compos mentis and asterixis was not documented.

These behavioral changes were clinically suggestive of early hepatic encephalopathy but were nonspecific. Psychometric testing was not available; therefore, covert hepatic encephalopathy could not be formally confirmed. Other potential contributors to behavioral change, including infection, electrolyte disturbance, gastrointestinal bleeding, medication-related effects, and alcohol withdrawal, should also be considered. In this patient, mild hyponatremia and suspected infection represented possible alternative or precipitating factors, while no active gastrointestinal bleeding was documented during hospitalization. This distinction is important because hepatic encephalopathy spans a wide neuropsychiatric spectrum and requires careful clinical correlation and exclusion of alternative causes of altered mental status [18].

Management in this case included dietary salt and fluid restriction, nutritional support, therapeutic paracentesis, albumin infusion, empiric antibiotic therapy, acid suppression, diuretic therapy with furosemide and spironolactone, lactulose, non-selective beta-blocker therapy with propranolol, and ursodeoxycholic acid. In terms of the use of antibiotics, spontaneous bacterial peritonitis could not be microbiologically or cytologically confirmed because ascitic fluid analysis was unavailable; therefore, antibiotic therapy was administered empirically for suspected infection.

For alcohol-associated cirrhosis, sustained abstinence remains a central disease-modifying intervention, with recent meta-analytic evidence showing that abstinence is associated with improved overall survival [19]. More importantly, this case demonstrates that early recognition and initial management of advanced liver disease can be guided by careful clinical assessment and a limited but appropriately selected diagnostic work-up, while definitive evaluation of portal hypertension and other complications should be pursued when feasible.

This case has several limitations related to the incomplete diagnostic work-up in a resource-limited clinical setting. As detailed in the Case Report, incomplete laboratory data precluded formal disease-severity scoring. Comprehensive etiological evaluation, including testing for hepatitis C and other causes of chronic liver disease, was also limited. Ascitic fluid analysis was unavailable; therefore, spontaneous bacterial peritonitis could not be confirmed. In addition, psychometric testing was not available to formally confirm covert hepatic encephalopathy. Endoscopic evaluation for portal hypertension and gastroesophageal varices was not completed because the patient had previously declined esophagogastroduodenoscopy. Despite these limitations, the integration of clinical findings, available laboratory results, non-invasive fibrosis indices, and ultrasonographic features strongly supported the working diagnosis of presumed alcohol-associated decompensated cirrhosis and allowed initial complication-directed management. Long-term outcome assessment was also limited because the patient did not return for scheduled follow-up after discharge.

Conclusion

This case highlights the practical importance of recognizing a late presentation of presumed alcohol-associated decompensated cirrhosis using clinical findings, routinely available laboratory tests, non-invasive

fibrosis indices, and ultrasonography when the diagnostic work-up is incomplete. In this patient, ascites, jaundice, thrombocytopenia, hypoalbuminemia, AST-predominant transaminitis, markedly elevated APRI and FIB-4 scores, and cirrhotic morphology on ultrasonography supported the working diagnosis and initial management despite the absence of endoscopic characterization of portal hypertension. Behavioral changes were clinically suggestive of early hepatic encephalopathy, although covert hepatic encephalopathy could not be formally confirmed because psychometric testing was unavailable. The main practical lesson is that clinicians should not delay recognition and complication-directed management of probable decompensated cirrhosis while awaiting more advanced investigations. Alcohol abstinence, nutritional optimization, control of decompensating events, appropriate referral for endoscopic assessment, and close follow-up remain essential to reduce further deterioration.

Patient Consent

Written informed consent for publication of this case report was obtained from the patient.

Conflicts of Interest

The authors declare that they have no conflicts of interest related to the publication of this case report.

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