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Case Study
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A CASE STUDY OF DECOMPENSATED CHRONIC LIVER DISEASE WITH PORTAL HYPERTENSION IN A MIDDLE-AGED MALE

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Abstract

Decompensated chronic liver disease (DCLD) represents the advanced stage of cirrhosis characterized by the development of complications such as ascites, jaundice, portal hypertension, variceal bleeding, and hepatic encephalopathy. Alcohol remains one of the leading causes of chronic liver disease worldwide. We report a case of a 41-year-old male with a long history of alcohol consumption who presented with abdominal distension, jaundice, and gastrointestinal bleeding. Clinical evaluation, laboratory investigations, and ultrasonographic findings confirmed the diagnosis of decompensated chronic liver disease with portal hypertension. The patient was managed with diuretics, non-selective beta-blockers, lactulose, rifaximin, proton pump inhibitors, and supportive therapy, resulting in clinical stabilization. This case highlights the importance of early recognition, comprehensive evaluation, and multidisciplinary management of DCLD to prevent life-threatening complications.

Keywords: *Decompensated chronic liver disease; Portal hypertension; Alcoholic liver disease; Ascites; Cirrhosis.*

INTRODUCTION

Chronic liver disease is a progressive condition characterized by irreversible hepatic fibrosis and nodular regeneration leading to cirrhosis. Decompensation occurs when the liver fails to maintain metabolic, synthetic, and detoxification functions, resulting in complications such as ascites, jaundice, variceal hemorrhage, and hepatic encephalopathy [1]. Portal hypertension is a major consequence of cirrhosis and contributes significantly to morbidity and mortality [2]. Alcohol-related liver disease remains a major etiological factor, especially in middle-aged males [3]. This case report describes a patient with alcoholic cirrhosis presenting with features of decompensation and portal hypertension.

CASE STUDY

A 41-year-old male was admitted to the hospital on 23 October 2025 with complaints of progressive abdominal distension and yellowish discoloration of the eyes for the past two months. The patient also reported the passage of blood in stools one week prior to admission and gave a significant history of melena occurring approximately two months earlier. According to the patient, he was apparently asymptomatic and in his usual state of health one month before the onset of these symptoms, indicating a relatively acute decompensation of an underlying chronic condition.

The patient had a known medical history of **hypertension for the past ten years**, for which he was on regular treatment with **metoprolol**. He also had a significant history of **chronic alcohol consumption for nearly fifteen years**, which is a well-recognized risk factor for the development of chronic liver disease. There was no documented history suggestive of viral hepatitis, autoimmune liver disease, or other chronic systemic illnesses, making alcohol the most likely etiological factor in this case.

On clinical examination at the time of admission, the patient was found to be **icteric**, with visible yellowish discoloration of the sclera and skin. Abdominal examination revealed **distension with features suggestive of ascites**, indicating hepatic decompensation. Vital parameters recorded during hospitalization showed relatively stable blood pressure values, although **initial episodes of tachycardia** were noted, which gradually improved with medical management. Oxygen saturation remained within normal limits throughout the hospital stay, indicating preserved respiratory function.

Laboratory investigations revealed clear evidence of **hepatic dysfunction and portal hypertension**. Serum bilirubin was markedly elevated at **8.3 mg/dL**, correlating with the clinical findings of jaundice. Liver enzyme analysis demonstrated a **disproportionate elevation of AST (112 IU/L) compared to ALT (23 IU/L)**, a biochemical pattern commonly associated

with alcoholic liver injury. Hematological parameters showed **significant thrombocytopenia with a platelet count of 53,000/cumm**, along with **anemia**, as indicated by a reduced red blood cell count of **2.99 million/cumm** and a hematocrit of **32.3%**. These findings were consistent with **hypersplenism secondary to portal hypertension**. Renal function tests, including blood urea and serum creatinine, were within normal limits, suggesting preserved renal function at the time of evaluation.

Ultrasonography of the abdomen revealed classical imaging features of **advanced liver cirrhosis**. The liver parenchyma appeared coarse and heterogeneous, with an irregular and nodular surface. Structural changes such as **right lobe atrophy with relative hypertrophy of the caudate and left lobes** were noted, strongly suggestive of chronic cirrhotic changes. Additional findings included **splenomegaly, a dilated portal vein, and the presence of portosystemic collaterals**, all of which are hallmark features of portal hypertension. Doppler evaluation demonstrated **markedly reduced portal flow velocity**, indicating severe portal hypertension and a high risk of complications such as variceal bleeding. The presence of **free anechoic ascitic fluid** further confirmed the decompensated state of liver disease and was associated with a poorer prognosis.

Based on the patient's clinical presentation, laboratory abnormalities, and characteristic imaging findings, a diagnosis of **decompensated chronic liver disease with portal hypertension**, most likely secondary to chronic alcohol use, was established.

The patient was managed with a comprehensive medical regimen aimed at treating complications of cirrhosis and preventing further deterioration. **Diuretic therapy with furosemide and spironolactone** was initiated to control ascites. **Lactulose and rifaximin** were administered to reduce ammonia levels and prevent hepatic encephalopathy. **Propranolol**, a non-selective beta-blocker, was started to lower portal pressure and reduce the risk of variceal hemorrhage. Supportive measures included **proton pump inhibitors, ursodeoxycholic acid, thiamine supplementation**, and **tranexamic acid** as clinically indicated. The patient responded favorably to treatment, with gradual clinical stabilization, and was subsequently discharged on **30 October 2025** with appropriate medications, strict advice on alcohol abstinence, and recommendations for regular follow-up.

MANAGEMENT

The patient received a combination of pharmacological and supportive therapy aimed at controlling ascites, preventing variceal bleeding, reducing ammonia levels, and providing hepatoprotective support. Diuretics such as furosemide and spironolactone were used to manage ascites. Lactulose and rifaximin were administered to prevent hepatic encephalopathy. Propranolol was initiated as a non-selective beta-blocker to reduce portal pressure and prevent variceal hemorrhage. Proton pump inhibitors, ursodeoxycholic acid, thiamine supplementation, and tranexamic acid were also administered as per clinical indication. The patient showed clinical improvement and was discharged on **30-10-2025** with appropriate medications and follow-up advice.

DISCUSSION

Decompensated cirrhosis represents a critical stage in chronic liver disease with significantly reduced survival. Alcoholic liver disease is a major contributor, particularly in patients with prolonged alcohol exposure [3]. Portal hypertension plays a central role in the development of ascites, splenomegaly, thrombocytopenia, and variceal bleeding [2]. Early identification of decompensating events and prompt initiation of standard therapy can improve outcomes and quality of life. Long-term abstinence from alcohol and regular follow-up are essential components of management.

CONCLUSION

This case underscores the clinical and radiological hallmarks of decompensated chronic liver disease with portal hypertension. Comprehensive assessment and timely intervention are crucial in preventing fatal complications. Patient education regarding alcohol cessation and adherence to therapy remains fundamental in improving prognosis.

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