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Alcohol-Induced Portal Hypertension Presenting With Massive Ascites-A Case Report

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ABSTRACT

Background: Portal hypertension and ascites are serious complications of chronic liver disease. In alcohol-related cirrhosis, recurrent ascites despite optimal therapy may suggest rare vascular entities such as porto-sinusoidal vascular disease (PSVD), which can mimic cirrhosis but follow a different clinical course.

Case Presentation: A 47-year-old male with a 30-year history of chronic alcohol use and comorbid diabetes and hypertension presented with abdominal distension, pedal oedema, dyspnoea, and oliguria. He had two prior admissions for similar complaints. Examination revealed icterus, ascites, anasarca, and mild splenomegaly. Laboratory tests showed anaemia, thrombocytopenia, and elevated transaminases. Ultrasound suggested chronic liver disease with ascites. The patient was treated with intravenous furosemide, oral spironolactone, propranolol, antibiotics, and underwent large-volume paracentesis. Supportive measures included a salt-restricted, high-protein diet. Clinical improvement was noted during hospitalization. He was diagnosed with decompensated liver disease with portal hypertension, most likely due to alcoholic cirrhosis. However, recurrent ascites despite therapy raised the possibility of an overlapping vascular liver disorder such as PSVD.

Conclusion: This case highlights the need to consider alternative diagnoses, such as PSVD, in patients with alcohol-induced liver disease who experience frequent decompensations. Multidisciplinary management, alcohol cessation counselling, and long-term monitoring are crucial. Advanced diagnostics like HVPG measurement or liver biopsy may help differentiate atypical presentations and guide therapy.

Keywords: *Portal hypertension, Ascites, Chronic alcoholism, Decompensated liver disease, Cirrhosis, Case report*

1. INTRODUCTION

Cirrhosis commonly refers to irreversible hepatic fibrosis characterized by regenerative nodule formation that disrupts normal liver function and intrahepatic blood flow, ultimately impairing hepatic function. [1] Progressive scarring of the liver increases resistance to portal blood flow, resulting in portal hypertension, which is defined as a pathological elevation in portal venous pressure arising from elevated intrahepatic vascular resistance and enhanced splanchnic inflow[2]. This hemodynamic imbalance underlies the major clinical complications of cirrhosis, including the development of ascites, variceal hemorrhage, and hepatic encephalopathy, and is a major determinant of prognosis. [1,2]

Ascites is the most common form of decompensating event in cirrhosis, which typically affects approximately 50% of compensated patients within a decade. [3] Sodium and water retention due to neurohormonal activation involving the renin–angiotensin–aldosterone system, sympathetic nervous system, and vasopressin release, combined with hypoalbuminemia and splanchnic vasodilation, leading to fluid accumulation in the peritoneal cavity. [3] Once ascites refractory to sodium restriction and diuretic therapy develops, patients face a poor median survival of around six months.[4] Therapeutic strategies focus on interrupting this pathophysiological cascade. First-line medical treatment for uncomplicated ascites includes sodium restriction (<5 g/day) and combined diuretic therapy with spironolactone and furosemide in a 100 mg:40 mg ratio, titrated slowly to achieve near-neutral fluid balance.[5] In cases of refractory ascites, repeated large-volume paracentesis (LVP) is indicated, accompanied by albumin infusion (6–8 g per liter of fluid removed) to prevent post-paracentesis circulatory dysfunction. [5,6]

In recent years, pharmacologic adjuncts have gained interest. α1-agonist midodrine has demonstrated comparable efficacy to albumin in reducing renal impairment and hyponatremia post-LVP, improving urine sodium output and systemic hemodynamics at substantially lower cost. [7–9] Likewise, clonidine (an α2-agonist) has shown benefit when added to diuretics, by reducing sympathetic drive and enhancing natriuresis. [6] Vasopressin analogue terlipressin and vasoconstrictor noradrenaline have also shown potential in preventing paracentesis-induced circulatory dysfunction. [6]

Long-term albumin therapy is another emerging intervention. The ANSWER trial demonstrated that weekly infusions of 40 g albumin for 18 months significantly improved 18-month survival (77% vs 66%) and reduced paracentesis need compared with standard care.10 However, these findings contrast with the MACHT trial, which showed no survival benefit in transplant-listed patients, highlighting the need for careful patient selection. [10] For refractory cases unresponsive to medical and endoscopic measures, transjugular intrahepatic portosystemic shunt (TIPS) offers effective portal decompression and improved ascites control, albeit with the risk of hepatic encephalopathy.[2, 18] Notably, the latest AASLD guidance emphasizes early use of nonselective β-blockers in compensated advanced chronic liver disease to delay decompensation.[1] Despite these advances, liver transplantation remains the only curative option for decompensated cirrhosis with refractory ascites. Owing to donor shortages and comorbidities, many patients require optimized multidisciplinary care tailored to their clinical status. Here, we present a case of Alcohol-Induced Portal Hypertension Presenting with Massive Ascites.

2. CASE PRESENTATION:

In July 2024, a 47-year-old male was admitted to the General Medicine unit with complaints of abdominal distension, progressive shortness of breath, swelling in both lower limbs, reduced urine output, and disturbed sleep cycle for the past three days. These symptoms had begun approximately a week before admission and had gradually worsened in severity. He was a known case of decompensated liver disease, type 2 diabetes mellitus, and hypertension, but had been noncompliant with his medications for several months. His prior medication history included Amlodipine 5 mg twice daily, Metformin 500 mg twice daily, and Ursodeoxycholic acid (Udiliv) 300 mg once daily.

The patient reported a significant history of chronic alcohol use spanning over 30 years, although he denied any history of tobacco use. He consumed a mixed diet and had been hospitalized twice before for similar complaints, indicating a pattern of recurrent decompensation. On presentation, he was conscious, alert, and oriented. General physical examination revealed bilateral pitting pedal edema, icterus, ascites, and anasarca. There was no clinical evidence of clubbing, cyanosis, or lymphadenopathy.

On vital sign assessment, his blood pressure was 150/100 mmHg, and his pulse rate was 100 beats per minute. Respiratory rate was slightly elevated at 22 breaths per minute, and oxygen saturation was 96% on room air. Systemic examination showed a distended abdomen with mild to moderate ascites, evidenced by a positive fluid thrill and dilated superficial abdominal veins. Cardiovascular examination revealed normal first and second heart sounds (S1 and S2) with no audible murmurs, and no abnormalities were noted on respiratory or central nervous system evaluations.

Laboratory investigations revealed anemia with a hemoglobin level of 8.1 g/dL and thrombocytopenia with a platelet count of 1.25 lakhs/cu.mm. Liver function tests showed elevated transaminases, with SGOT (AST) at 140 IU/L and SGPT (ALT) at 65 IU/L. Other biochemical parameters, including renal function and electrolytes, were within normal limits, though INR and serum bilirubin were mildly elevated. An abdominal ultrasound revealed features of chronic liver parenchymal disease

with mild splenomegaly and mild to moderate ascites. Exfoliative cytology from the ascitic fluid was negative for malignant cells, ruling out peritoneal carcinomatosis. The detailed laboratory investigations on the day of admission are listed in Table 1.

Table 1: Laboratory and Imaging Investigations

Parameter	Result
Hemoglobin	8.1 g/dL
Platelet Count	1.25 lakhs/cu.mm
SGOT (AST)	140 IU/L
SGPT (ALT)	65 IU/L
INR	Mildly elevated
Serum Bilirubin	Mildly elevated
Renal Function & Electrolytes	Within normal limits
Abdominal Ultrasound	CLD, mild splenomegaly, ascites
Ascitic Fluid Cytology	Negative for malignancy

The patient was initiated on intravenous Furosemide 20 mg twice daily and oral Spironolactone 25 mg once daily for ascitic fluid management. He also received intravenous Ranitidine, oral Propranolol 25 mg twice daily for portal hypertension, Alprazolam 0.25 mg at bedtime for sleep disturbances, and multivitamin supplements. A salt-restricted diet and high-protein intake were advised to support nutritional status and manage fluid retention.

A large-volume paracentesis (2 liters) was performed for symptomatic relief, and prophylactic antibiotics—Ciprofloxacin 500 mg twice daily and Metronidazole 400 mg three times daily—were initiated to prevent spontaneous bacterial peritonitis. The patient's fluid input and output were closely monitored during hospitalization. The detailed therapeutic management of the patient is listed in Table 2.

Table 2: Therapeutic Management

Intervention/Medication	Dosage/Route
Furosemide	20 mg IV BID
Spironolactone	25 mg orally OD
Ranitidine	1 amp IV
Propranolol	25 mg orally BID
Alprazolam	0.25 mg orally HS
Ciprofloxacin	500 mg orally BID
Metronidazole	400 mg orally TID
Large Volume Paracentesis	2 liters
Multivitamins	Oral

Over the course of his hospital stay, the patient demonstrated clinical improvement. His abdominal distension subsided, dyspnoea decreased, and urine output normalized. The pedal oedema showed marked regression, and there were no new complications. The patient received detailed counselling on alcohol cessation, and the potential long-term complications of chronic liver disease and portal hypertension were explained to him and his family.

The final diagnosis was confirmed as decompensated chronic liver disease with portal hypertension, most likely secondary to alcoholic cirrhosis. The patient was discharged after two weeks in a stable condition with advice for strict medication adherence, nutritional support, abstinence from alcohol, and follow-up with a hepatologist for long-term disease management

and transplant consideration if needed.

3. DISCUSSION

Portal hypertension and subsequent ascites are hallmark complications of liver cirrhosis, signalling a decline in hepatic function and an increased risk of decompensation. While cirrhosis is the predominant cause of portal hypertension, rare aetiologies may present in similar clinical contexts and confound diagnosis and management.

In most instances, chronic alcohol-induced cirrhosis leads to portal hypertension through fibrotic remodelling of hepatic sinusoids and microvascular changes. [1–3] The hepatic venous pressure gradient (HVPG) threshold for clinically significant portal hypertension is well established at ≥10–12 mm Hg, above which complications such as ascites, variceal bleeding, and hepatic encephalopathy commonly ensue. [4–6] In our patient, a 47-year-old male with longstanding alcohol use and decompensated cirrhosis, the typical pathophysiological trajectory was observed, such as progressive ascites, oedema, and laboratory evidence of hepatic insufficiency. Yet, what makes this case notably rare is the multiple admissions for ascites over a short period, despite compliance-oriented in-hospital management. This echoes features seen in idiopathic non-cirrhotic portal hypertension (INCPH) or porto-sinusoidal vascular disease (PSVD), a condition characterized by portal hypertension in the absence of overt cirrhosis. [7–9,19] Recent reviews show that, although infrequent, these vascular disorders can present with ascites, variceal bleeding, and splenomegaly, mimicking cirrhosis, yet histologically and hemodynamically differ, often showing near-normal liver stiffness and lower HVPG readings. [8,17] INCPH frequently occurs in middle-aged males in Asia, with an incidence of around 10–13 cases per 100,000. A recent international cohort highlights that approximately 50% of INCPH patients develop ascites, often in conjunction with anticoagulable states or exposure to toxins, and these patients can progress to portal vein thrombosis if untreated. [7,10–12] In contrast to the natural history of alcoholic cirrhosis, INCPH may preserve synthetic liver function longer, though outcomes worsen significantly once ascites ensues. [12,13]

Although our patient had biochemical and sonographic evidence consistent with alcoholic cirrhosis, the recurrent nature of his ascites, despite large-volume paracentesis, diuretics, β-blockers, and dietary measures, raises the possibility of an overlapping vascular component to his portal hypertension. Structural changes in hepatic microcirculation from decades of alcohol use could overlap with PSVD, predisposing him to more labile fluid accumulation. [7, 20] Therapeutically, guidelines for INCPH align with those for cirrhosis: nonselective β-blockers and diuretics remain central. However, trans jugular intrahepatic portosystemic shunt (TIPS) may be considered earlier in refractory cases, given its relative efficacy in both conditions, though encephalopathy risk persists. [8,10] Recent Asian series report improved ascites control post-TIPS in INCPH, although data on cirrhotic patients with mixed histology remain limited. [11,12, 18, 20] Moreover, a small but growing body of literature suggests that early identification of a vascular pattern could alter outcomes. In a European cohort of idiopathic portal hypertension patients, five-year transplant-free survival was higher (86%) when ascites was absent initially, but dropped sharply once fluid retention developed.10 This underscores the need for vigilance and perhaps histological confirmation when decompensation is unusual or erratic.

In our patient, while invasive measurements (e.g., HVPG) and liver biopsy were not performed, persistent ascites despite standard management may reflect a mixed etiological profile, involving both alcoholic cirrhosis and PSVD. This nexus between chronic alcohol toxicity and vascular remodelling represents a rare but plausible phenotype, demanding a tailored approach. Future assessment, such as transient elastography, HVPG measurement, or, if feasible, liver biopsy, might clarify portal pressure dynamics and tissue architecture, guiding decisions such as early TIPS placement.

This case highlights several important strengths, including a thorough clinical evaluation of a patient with decompensated liver disease and portal hypertension, managed through a multidisciplinary approach involving diuretics, large-volume paracentesis, beta-blockers, antibiotics, and dietary modifications. It underscores the real-world challenge of recurrent ascites in chronic alcoholism and raises the rare but clinically relevant possibility of overlapping portal vascular disorders such as porto-sinusoidal vascular disease (PSVD). [14-16] The case also emphasizes patient counseling for alcohol cessation, reflecting holistic management. However, the absence of histopathological confirmation via liver biopsy and the lack of hepatic venous pressure gradient (HVPG) measurement are notable limitations, which restrict diagnostic certainty regarding the etiology of portal hypertension. Additionally, long-term follow-up data, including outpatient compliance and recurrence of symptoms, are unavailable. Imaging modalities were limited to ultrasonography, and advanced vascular studies could have enhanced diagnostic clarity. As a single case, generalizability is inherently limited, though the presentation encourages clinicians to maintain diagnostic vigilance in atypical or recurrent ascitic decompensation.

4. CONCLUSION

Recurrent ascites in chronic alcoholic liver disease is a marker of disease progression and significant portal hypertension. In cases where ascites persists despite standard therapy, rare vascular conditions such as porto-sinusoidal vascular disease (PSVD) should be considered. Early diagnosis supported by appropriate imaging and hemodynamic studies, along with individualized pharmacologic and interventional care—including diuretics, large-volume paracentesis, and beta-blockers—can improve clinical outcomes. Patient education, nutritional support, and sustained alcohol cessation counseling are

essential to reduce morbidity, enhance quality of life, and potentially delay the need for liver transplantation. A multidisciplinary approach remains vital for long-term management and prognostic improvement.

Conflicts of Interest:

None declared.

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Patient Consent:

Informed consent was received from the patient for publication. Identifiers have been removed.

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