

Complex Interplay of Cardiac, Hepatic, and Renal dysfunction in a patient with Extra-Hepatic Portal Vein Obstruction and Rheumatic Mitral Stenosis: A Case Report

ABSTRACT

This case report presents a 31-year-old male with a complex interplay of cardiac, hepatic, and renal dysfunction, characterized by extra-hepatic portal venous obstruction (EHPVO), decompensated chronic liver disease (DCLD), and rheumatic heart disease (RHD) with severe mitral stenosis. The patient exhibited altered sensorium and hemodynamic instability, reflecting the intricate relationship between the heart, liver, and kidneys, often referred to as the cardio-hepato-renal axis. The initial presentation included hepatic encephalopathy, ascites, and acute kidney injury, highlighting the challenges in managing multi-organ dysfunction.

EHPVO, resulting from portal vein thrombosis, led to chronic portal hypertension and subsequent liver dysfunction, while RHD with mitral stenosis exacerbated cardiac strain and reduced renal perfusion. The clinical examination revealed significant cardiovascular findings, including elevated left atrial pressure, which contributed to pulmonary congestion and further impacted renal function. Laboratory tests indicated elevated bilirubin levels, coagulopathy, and renal impairment, consistent with the diagnosis of hepatic encephalopathy and acute kidney injury.

Management strategies included pharmacotherapy aimed at addressing hepatic encephalopathy and portal hypertension, such as lactulose and non-selective beta-blockers, along with diuretics to manage ascites. Renal support was considered to maintain adequate perfusion in light of the patient's hemodynamic instability. This case underscores the importance of a holistic and multi-disciplinary approach in managing patients with concurrent cardiac, hepatic, and renal dysfunctions, emphasizing the need for integrated strategies to address the complexities of such presentations.

Through this report, we aim to enhance understanding of the interdependencies between these organ systems and provide insights into the clinical management of complex cases involving multi-organ dysfunction.

Keywords: [Hepatic encephalopathy, EHPVO, DCLD, RHD, Portal hypertension]

1. INTRODUCTION

The intricate relationship between the heart, liver, and kidneys—often referred to as the cardio-hepato-renal axis—has garnered substantial attention in recent years due to its implications in multi-organ failure and complex management scenarios. Disruptions within this axis can precipitate a cascade of interrelated dysfunctions, where pathology in one organ amplifies the risk and severity of dysfunction in others. This interplay becomes particularly pronounced in patients with underlying chronic conditions affecting these organs, as is seen in this case involving extra-hepatic portal venous obstruction (EHPVO), decompensated chronic liver disease (DCLD), and rheumatic heart disease (RHD) with mitral stenosis (MS). Each condition poses unique pathophysiological challenges, which, in combination, create a complex clinical scenario requiring comprehensive and multi-disciplinary management.

EHPVO, primarily a disorder of the portal venous system, leads to the development of portal hypertension, often manifesting as complications such as splenomegaly, variceal bleeding, and ascites in patients from a young age. This condition, usually resulting from thrombosis of the portal vein, impairs blood flow to the liver and results in chronic portal hypertension without primary liver disease. Over time, the chronic increase in portal pressure can lead to liver dysfunction and the eventual development of DCLD, as observed in our patient. DCLD is marked by liver decompensation, including the development of hepatic encephalopathy (HE), ascites, and coagulopathy, indicating severe liver insufficiency and end-stage liver disease. Hepatic encephalopathy, characterized by altered mental status and asterixis, is often precipitated by systemic insults such as infections, gastrointestinal bleeding, or electrolyte disturbances, all of which are common in patients with portal hypertension and advanced liver disease¹⁻².

Rheumatic heart disease (RHD) remains a significant cause of valvular heart disease, particularly in developing countries, due to inadequate control of streptococcal infections³. Mitral stenosis (MS), a common consequence of RHD, leads to increased left atrial pressure and subsequent pulmonary congestion, placing additional strain on the heart and impacting systemic circulation. The resulting hemodynamic alterations can exacerbate portal hypertension and reduce renal perfusion, contributing to renal dysfunction and susceptibility to acute kidney injury (AKI)⁴. Moreover, the presence of RHD with MS complicates the hemodynamic stability of patients with concurrent liver disease, as both organs are highly sensitive to fluctuations in blood flow and pressure. This interdependence creates a vicious cycle where liver dysfunction exacerbates cardiovascular instability, further complicating renal function⁵.

Acute kidney injury, often presenting as a form of hepatorenal syndrome in patients with DCLD, reflects the vulnerability of the kidneys in the setting of impaired liver and cardiac function. Renal failure in this context is frequently precipitated by factors such as hypoperfusion, systemic vasodilation, and neurohormonal activation, all of which are common in advanced liver disease with portal hypertension and compromised cardiac output⁶. The non-oliguric renal failure observed in this patient underscores the complexity of managing AKI in the setting of multiple concurrent organ dysfunctions, where typical signs of renal compromise may be masked by altered systemic circulation and fluid dynamics⁷.

This case report aims to illustrate the challenging clinical presentation and management of a young patient with EHPVO, DCLD, severe mitral stenosis due to RHD, hepatic encephalopathy, and acute renal failure. It highlights the interdependent pathophysiological

mechanisms linking the heart, liver, and kidneys and underscores the importance of a holistic, multi-disciplinary approach in managing such complex cases. Through this case, we aim to provide insights into the challenges and considerations involved in addressing the multi-organ dysfunctions resulting from the interplay between chronic cardiac, hepatic, and renal disease^{8 - 9}.

2. PRESENTATION OF CASE

- The patient is a 31-year-old male presenting with altered sensorium following a loss of consciousness, with a history of intermittent fever for one week, abdominal distension for two months, icterus for two months, and abdominal pain for one week.
- His medical history is significant for extra-hepatic portal venous obstruction with portal hypertension since childhood, rheumatic heart disease with mitral stenosis for the past 12 years, and decompensated chronic liver disease.
- He denies any history of hematemesis, melena, constipation, decreased urine output, or seizures. He follows a mixed diet with normal bowel and bladder habits and a regular sleep pattern. He is a known alcoholic, with his last binge a month ago.
- On examination, the patient is drowsy, does not follow verbal commands, appears oriented but febrile, tachypneic, pale, and icteric, with normal jugular venous pressure. His heart rate is 100 beats per minute, and his initial systolic blood pressure was below 80 mmHg, stabilized to 90/60 mmHg with Noradrenaline at 0.1 µg/kg/min. Respiratory rate is 22 breaths per minute, capillary blood glucose is 123 mg/dL, and sPo₂ is 96%. He also has bilateral pitting pedal edema.
- Cardiovascular examination revealed normal S1 and S2 with a loud S1 in the mitral area and a loud S2 in the pulmonary area accompanied by an ejection systolic murmur; there was a tapping apical impulse in the 4th intercostal space, 1/2 inch lateral to the midclavicular line, with visible pulsations over the pulmonary area and a palpable P2.
- Auscultation of the mitral area revealed normal S1 and S2 sounds, a loud S1, no S3 or S4, and a low-pitched, grade 3/4, rough rumbling mid-diastolic murmur best heard with the bell in the left lateral position with breath held in expiration, showing pre-systolic accentuation. In the tricuspid area, S1 and S2 were heard without any added sounds. The pulmonary area presented with S1 and S2 sounds, a loud P2, and a grade 3/6 ejection systolic murmur. In the aortic area, S1 and S2 were audible.
- Respiratory examination showed bilateral air entry.
- Neurological examination revealed bilateral pupils equal, reactive to light (3 mm), and bilateral flapping tremors.
- The abdominal examination was soft, with bowel sounds present, a distended abdomen with diffuse tenderness, a puffed down, everted umbilicus, equal movement in all quadrants with respiration, dilated veins on the sides, no hepatomegaly, splenomegaly extending 4 fingers below the costal margin, fluid thrill, and ascites.

List 1 : The laboratory investigations are as follows

Parameter	Observed value			Normal range	Unit
Hematology					
WBC	14.7↑	8.7	12.9↑	4.0-11.0	x103/μl
Hb	7.8↓	7.3↓	9.7↓	14.0-18.0	g/dl
RBC	2.61↓	2.59↓	3.09↓	4.5-6.0	x106/μl
HCT	24.1↓	23.8↓	29.5↓	36-46	%
MCV	92.3	91.9	95.5	80-100	fl
MCH	29.9	28.2	31.4	27-33	pg
MCHC	32.4	30.7↓	32.9	32-36	g/dl
PLT	26	28	35	150-450	x103/μl
N	88.7↑	88.1↑	84.5↑↓	40-60	%
L	6.2↓	8.8↓	10.8↓	20-40	%
M	5.1	3.1	4.7	2-8	%
Liver function tests & electrolytes					
TB	13.9↑	16.3↑	18.6↑	0.1-1.2	mg/dl
DB	6.6↑	8.1↑	9.5↑	0.0-0.3	mg/dl
AST	90↑	70↑	53↑	10-40	U/L
ALT	49	41	37	7-56	U/L
ALP	295↑	185↑	180↑	44-147	U/L
TP	6.2	4.8	5.3	6.4-8.3	g/dl
Na	134↓	131↓	141	135-145	mEq/L
K	5.0	4.7	4.3	3.5-5.1	mEq/L
PT	27.9↑	37.3↑	26.7↑	9.5-13.8	sec
INR	2.27↑	3.15↑	2.1↑	0.8-1.2	-
Albumin	1.7↓	1.3↓		3.5-5.0	g/dl
LDH	130				IU/L
Renal function tests					
Sugar	127	94	70	70-110	mg/dl
Urea	96↑	89↑	61↑	10-50	mg/dl
Creatinine	1.4↑	1.5↑	0.8	0.6-1.2	mg/dl
Ascitic fluid analysis					
Protein	0.9			0-2.5	g/dl
Sugar	131↑			70-110	mg/dl

Albumin	0.3	<1.1	g/dl
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- HBsAg and anti-HCV were negative, and HIV was non-reactive. Ascitic fluid culture showed no pus cells or organisms.
- The ECG indicated sinus bradycardia with inverted T waves in leads V1, V2, and V3. Chest X-ray findings included right-sided pleural effusion, left atrial enlargement, and an elevated right hemidiaphragm (Fig. 1).

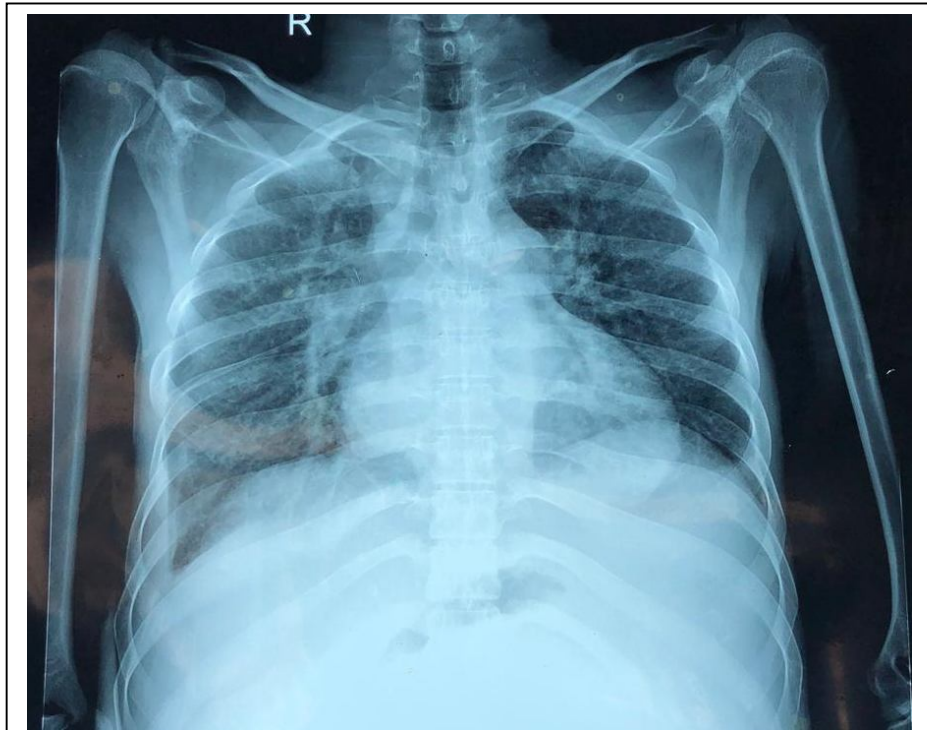


Fig. 1., Chest X-ray revealing right-sided pleural effusion, left atrial enlargement, and an elevated right hemidiaphragm.

- On assessment of diagnosis, The patient is experiencing hepatic encephalopathy (grade II-III) as evidenced by altered sensorium, drowsiness, and bilateral flapping tremors (asterixis). His history of decompensated chronic liver disease (DCLD), indicated by elevated total bilirubin (13.9 mg/dL) and direct bilirubin (6.6 mg/dL), significantly supports this diagnosis. Additionally, the prolonged prothrombin time (PT) (27.9 seconds) and elevated International Normalized Ratio (INR) (2.27), alongside low albumin levels (1.7 g/dL), confirm liver dysfunction contributing to the encephalopathy.
- The presence of extra-hepatic portal venous obstruction (EHPVO) with portal hypertension is substantiated by the distended abdomen with ascites, visible superficial abdominal veins (caput medusae), and the patient's history of portal hypertension since childhood. The ascitic fluid analysis shows low protein levels (0.9 g/dL) and a low serum-ascites albumin gradient (SAAG), indicating portal hypertension as the likely cause of the ascites.

- Rheumatic heart disease (RHD) with severe mitral stenosis is evidenced by the cardiovascular examination findings of a loud S1, a rough rumbling mid-diastolic murmur, and signs of pulmonary congestion noted on the chest X-ray. The elevated heart rate (tachycardia at 100 bpm) and initial hypotension (systolic blood pressure < 80 mmHg) suggest hemodynamic instability, indicating shock.
- The laboratory results indicate acute kidney injury (AKI) characterized by elevated creatinine levels of 1.4 mg/dL and urea levels of 96 mg/dL. These findings are consistent with renal failure, likely due to volume depletion from ascites and potential hepatorenal syndrome. Although the patient is non-oliguric, the renal function has been previously compromised, highlighting the need for ongoing monitoring and supportive management.

Overall, the correlation between the clinical presentations and laboratory findings strongly supports the assessments of hepatic encephalopathy, decompensated chronic liver disease, EHPVO with portal hypertension, severe mitral stenosis due to RHD, shock, and acute kidney injury.

Clinical management and outcomes:

The patient's treatment included a range of targeted interventions, leading to stabilization and improvement in multiple organ systems:

- Intravenous Noradrenaline 0.1 µg/kg/min – Administered to support blood pressure and stabilize hemodynamic status in response to hypotension, reducing shock risk.
- Intravenous Cefotaxime 1g thrice daily (later transitioned to Oral Cefixime 200mg 2 once daily) – Provided empirical coverage against bacterial infections that could exacerbate hepatic encephalopathy and worsen liver function. Transitioned to oral therapy as infection risk decreased.
- Intravenous Ranitidine 50 mg twice daily – Used to prevent stress ulcers and reduce gastric acid secretion, minimizing the risk of gastrointestinal bleeding, which is particularly important given the patient's coagulopathy.
- Intravenous Ondansetron 4 mg twice daily – Administered to control nausea and prevent vomiting, helping to avoid fluid losses that could exacerbate renal impairment and fluid-electrolyte imbalances.
- Intravenous Human Albumin 20% 100 ml once daily – Given to manage hypoalbuminemia, support plasma oncotic pressure, and aid in fluid balance, helping to reduce ascites and stabilize blood pressure.
- Intravenous Vitamin K 10 mg IV once daily – Used to improve coagulopathy by supporting clotting factor synthesis, essential given the prolonged PT and elevated INR.
- Oral Ursodeoxycholic acid 300 mg thrice daily – A bile acid that improves liver function by protecting liver cells, aiming to manage liver dysfunction and reduce hepatic inflammation.
- Oral Rifaximin 550 mg twice daily – Administered to reduce intestinal ammonia-producing bacteria, a key step in managing hepatic encephalopathy and improving sensorium.
- Oral Penicillin 250 mg twice daily – Given prophylactically to manage any potential infections that could complicate the patient's condition due to RHD history.
- Oral Vitamin B complex and Vitamin C once daily – Supplemented to enhance immune function and support antioxidant capacity, beneficial in combating infection and supporting overall health.
- Oral Syrup Lactulose 30 ml thrice daily and Lactulose enema at night – Used to lower ammonia levels by reducing intestinal absorption, a key measure for hepatic encephalopathy management.

- Intravenous fluid Normal saline 300 ml Bolus – Given as fluid support to stabilize blood pressure and improve renal perfusion, essential given the renal impairment and ascites.
- Diuretics withheld and then restarted with oral Furosemide 40 mg once daily and oral Spironolactone 25 mg four times a day – Diuretics were initially withheld to avoid worsening encephalopathy but were resumed to manage ascites after stabilization. Spironolactone and Furosemide together helped manage fluid retention while preserving electrolyte balance.
- Therapeutic Ascitic Fluid Tapping – Conducted to relieve abdominal distension, improve respiratory function, and aid in symptomatic relief from portal hypertension.
- Fluid Restriction (FRD - 1 L/day) – Implemented to control fluid overload and manage ascites without compromising hemodynamic stability.
- Four packs of Fresh Frozen Plasma (FFP) – Transfused to provide clotting factors, helping to manage coagulopathy and reduce bleeding risk due to prolonged PT/INR.

Overall, these interventions contributed to improvement in hepatic encephalopathy, stabilization of liver and renal function, and reduction in ascites and infection risk, providing a positive clinical outcome for the patient.

3. DISCUSSION

The interplay between cardiac, hepatic, and renal dysfunction is a complex clinical scenario, especially in a patient with extra-hepatic portal venous obstruction (EHPVO) and rheumatic mitral stenosis. Understanding this interplay is crucial for effective management strategies tailored to address multi-organ dysfunction.

1) Cardiac Dysfunction in the Context of Rheumatic Mitral Stenosis

Rheumatic heart disease (RHD), particularly in developing countries, significantly contributes to mitral stenosis (MS), leading to various cardiovascular complications. In our case, the patient's history of RHD with MS was confirmed by clinical examination findings, such as a loud first heart sound (S1) and a mid-diastolic murmur, both of which indicate increased left atrial pressure and subsequent pulmonary congestion. Elevated left atrial pressure can lead to pulmonary hypertension, increasing the workload on the right heart and contributing to right heart failure¹.

i. Pharmacotherapeutic Management:

In patients with MS, diuretics such as furosemide can help manage volume overload and pulmonary congestion, improving symptoms and cardiac output⁴. Anticoagulation therapy, particularly with warfarin or direct oral anticoagulants, may be indicated to prevent thromboembolic events associated with atrial fibrillation, which can occur due to left atrial enlargement¹⁰. Additionally, beta-blockers can help manage heart rate and reduce myocardial oxygen demand, which may be beneficial in cases of heart failure or significant arrhythmias¹¹.

ii. Surgical Options:

Surgical interventions such as balloon mitral valvuloplasty or mitral valve replacement should be considered, especially in symptomatic patients with significant stenosis, as timely intervention can significantly improve hemodynamics and mitigate complications related to right heart failure¹².

iii. Lifestyle Modifications:

Patients are advised to maintain a low-sodium diet to help manage fluid retention, engage in regular physical activity as tolerated, and avoid excessive fluid intake. Weight management can also reduce the strain on the heart, improving overall cardiovascular health.

2) Hepatic Dysfunction and Its Management

In patients with EHPVO, portal hypertension leads to various hepatic complications, including ascites and hepatic encephalopathy (HE). The patient presented with ascites, asterixis, and altered sensorium, indicative of HE, which has been substantiated by previous studies emphasizing the need for early recognition and management of HE in patients with liver dysfunction⁸.

i. Pharmacotherapeutic Management:

Management strategies for HE typically include addressing precipitating factors such as infections, electrolyte imbalances, and gastrointestinal bleeding. Lactulose is commonly used to reduce ammonia levels, while rifaximin can be added to prevent recurrence of HE⁶. Additionally, for portal hypertension, non-selective beta-blockers (such as propranolol or nadolol) can be employed to reduce portal pressure by decreasing cardiac output and splanchnic blood flow, thereby lowering the risk of variceal bleeding¹³.

ii. Management of Portal Hypertension:

- a) Non-Selective Beta-Blockers: These are first-line agents for preventing variceal bleeding in portal hypertension, effective in reducing portal pressure¹⁴.
- b) Diuretics: Agents like spironolactone can be used to manage ascites, helping to decrease fluid retention and reduce abdominal pressure¹⁵.
- c) Endoscopic Procedures: Variceal ligation or sclerotherapy may be necessary for patients with significant varices to prevent bleeding¹⁶.
- d) Surgical Options: For patients with refractory ascites or significant portal hypertension, procedures such as transjugular intrahepatic portosystemic shunt (TIPS) may be indicated to reduce portal pressure and improve liver function⁷.

iii. Lifestyle Modifications:

Dietary modifications should include a low-protein diet to reduce ammonia production, while also monitoring for malnutrition due to hepatic dysfunction. Regular follow-ups with a hepatologist are vital for managing liver health and monitoring for complications⁹.

3) Renal Dysfunction in the Context of Hepatic and Cardiac Failure

Acute kidney injury (AKI) in this patient, reflected by elevated creatinine and urea levels, underscores the renal vulnerability associated with hepatic dysfunction and cardiac instability. The concept of hepatorenal syndrome, as detailed by Boyer and Sanyal, demonstrates that renal impairment in liver disease often stems from splanchnic vasodilation and reduced renal perfusion¹⁷. This patient's non-oliguric renal failure complicates the clinical picture, as hemodynamic stability must be restored to improve renal outcomes.

i. Pharmacotherapeutic Management:

Maintaining adequate renal perfusion is crucial. Administering intravenous fluids and avoiding nephrotoxic agents such as nonsteroidal anti-inflammatory drugs (NSAIDs) is essential¹⁸. In some cases, vasopressors may be required to support renal blood flow in the setting of severe hypotension. Additionally, managing hyperkalemia may be necessary, particularly if renal function continues to decline, and can be achieved through the use of agents such as calcium gluconate, insulin with glucose, and sodium bicarbonate¹⁹.

ii. Supportive therapy:

In cases of acute kidney injury secondary to hepatorenal syndrome, continuous renal replacement therapy (CRRT) may be indicated to support renal function while addressing underlying hepatic issues ⁵.

iii. Lifestyle Modifications:

Encouraging patients to maintain a balanced diet with adequate hydration, while monitoring for signs of fluid overload, is critical. Regular exercise and avoidance of alcohol can help protect renal function.

Interconnectedness of Multi-Organ Dysfunction:

The complexities of multi-organ dysfunction in this case demonstrate how dysfunction in one organ can lead to cascading failures in others. This phenomenon is particularly evident in patients with portal hypertension and RHD, where the combined effects exacerbate renal insufficiency and HE.

Supporting this view, Vilstrup et al. highlight that management of hepatic encephalopathy requires a holistic approach that considers the overall clinical status, integrating cardiac and renal parameters to optimize outcomes ⁹.

Summary of Management Strategies (Table. 1)

Cardiac Management:

- Early intervention in mitral stenosis through balloon valvuloplasty or surgical repair should be considered, especially in patients with concurrent liver disease to optimize hemodynamics ¹².
- Pharmacotherapy: Use of diuretics for fluid management, anticoagulants for thromboembolic prevention, and beta-blockers for rate control.
- Lifestyle: Low-sodium diet and regular exercise.

Hepatic Management:

- Close monitoring for precipitating factors of HE and utilizing lactulose or rifaximin for symptomatic relief are essential ⁶.
- Portal Hypertension Management: Use of non-selective beta-blockers, diuretics, and endoscopic interventions to manage complications ⁷.
- Surgical Options: Consideration of TIPS for refractory cases.
- Lifestyle: Dietary modifications and regular hepatology follow-ups.

Renal Management:

- Maintaining fluid balance and considering CRRT in severe AKI can help manage renal complications arising from liver dysfunction ⁵.
- Pharmacotherapy: Avoidance of nephrotoxins, administration of fluids, and managing electrolyte imbalances.
- Lifestyle: Hydration management and monitoring for fluid overload.

Table. 1, Summary of Management Strategies

Management Area	Medication/Intervention	Purpose
Cardiac Management	Diuretics (e.g., Furosemide), Anticoagulants (e.g., Warfarin), Beta-blockers	To manage fluid overload, prevent thromboembolism, and reduce myocardial oxygen demand.
Cardiac Surgical Options	Balloon Mitral Valvuloplasty or Mitral Valve Replacement	Improves hemodynamics by addressing mitral stenosis, reducing strain on the heart.
Liver Support and Hepatic Encephalopathy Management	Lactulose, Rifaximin	Reduces ammonia levels to manage hepatic encephalopathy and improve mental status.
Portal Hypertension Management	Non-Selective Beta-Blockers (e.g., Propranolol), Diuretics (e.g., Spironolactone), Endoscopic Variceal Ligation	Decreases portal pressure to prevent variceal bleeding and manage ascites.
Renal Support in Hepatorenal Syndrome	IV Fluids, Avoidance of NSAIDs, CRRT if needed	Maintains renal perfusion, avoids nephrotoxicity, and supports renal function in severe AKI cases.
Coagulopathy Management	Vitamin K, Fresh Frozen Plasma (FFP)	Improves clotting factor levels, reducing bleeding risks due to prolonged PT/INR.
Lifestyle Modifications	Low-Sodium Diet, Protein Restriction, Regular Follow-Ups	Manages fluid retention, prevents ammonia build-up and supports ongoing health monitoring.

Holistic Approach:

- A multi-disciplinary approach integrating cardiology, hepatology, and nephrology expertise is crucial for managing such complex cases effectively⁹.

Future Research Implications:

This case suggests multiple future research directions to improve the management of complex cardio-hepato-renal interactions, especially in patients with concurrent conditions like EHPVO, RHD, and DCLD. Studies could refine multi-organ management protocols, focusing on pharmacologic combinations and fluid management to balance heart, liver, and kidney needs¹. Research on minimally invasive cardiac interventions, like transcatheter mitral valve procedures, might provide safer options for patients unfit for traditional surgery, while the potential of TIPS in alleviating portal hypertension and improving organ function warrants further exploration¹². Additionally, examining the impact of lifestyle modifications, particularly in patients with alcohol-related complications, could reveal preventive strategies and long-term benefits¹³. Further research could also explore biomarkers to identify early multi-organ deterioration and investigate genetic predispositions affecting the cardio-hepato-renal axis, adding a personalized medicine aspect²⁰. These insights emphasize the importance of an integrated, multidisciplinary approach for multi-organ dysfunction⁹.

4. CONCLUSION

This case underscores the critical need for integrated management strategies in patients with complex multi-organ dysfunction, emphasizing that a thorough understanding of the interrelated pathophysiological mechanisms is essential for optimal patient outcomes.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

CONSENT

The participation was on a voluntary basis and written consent was obtained from the individual who participated in this case report.

ETHICAL APPROVAL (WHERE EVER APPLICABLE)

As per international standards or university standards written ethical approval has been collected and preserved by the author(s).

Disclaimer (Artificial intelligence)

Author(s) hereby declare that NO generative AI technologies such as Large Language Models (ChatGPT, COPILOT, etc.) and text-to-image generators have been used during the writing or editing of this manuscript.

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