DCLD with portal hypertension with grade I encephalopathy: A case report

Dr. Rahul Shil

Associate Professor, Department of MSN (Neuroscience), PES University, Bengaluru, Karnataka, India

Corresponding author: Dr. Rahul Shil

Received 11 Nov 2025; Accepted 24 Dec 2025; Published 1 Jan 2026

DOI: https://doi.org/10.64171/JAMS.2026.6.1.1-3

Abstract

Decompensated liver disease (DCLD) is a medical emergency that has a high mortality rate. DCLD is also associated with other complications such as visceral bleeding, ascites, hepatic encephalopathy, etc. In this case report, a 49-year-old male developed DCLD with portal hypertension and grade-I encephalopathy. Prognosis is generally poor in such conditions, but liver transplantation could be an option in end-stage liver failure.

Keywords: Decompensated liver cirrhosis, Hepatic encephalopathy, Chronic liver failure, Portal hypertension

Introduction

Liver cirrhosis is a major cause of mortality in the world, which constitutes around 2.4% of the global death [1]. Clinical complications such as ascites, jaundice, hepatic encephalopathy, infections, or portal-hypertensive hemorrhages are common with acute decompensation of liver cirrhosis, which marks a turning point in the prognosis [2]. Men account for almost two-thirds of all liver-related fatalities. Alcohol, non-alcoholic fatty liver disease, and viral hepatitis are the three main causes of cirrhosis in the world. The majority of acute hepatitis cases are caused by hepatotropic viruses, although a growing percentage of cases are caused by drug-induced liver injury [3, 4]. Liver disease poses serious health risks and burdens among the population. India accounted for one-fifth (18.3%) of all cirrhosis fatalities worldwide, with 259,749 liver disease deaths, or 2.95% of all deaths, according to the most recent WHO data released in 2017 [5]. A study conducted in India found that 13% of persons with alcoholic cirrhosis drink occasionally, while 52% of those with liver cirrhosis are 18 years of age or older and regularly drink [6]. With a rapidly growing economy and changes in lifestyle and food patterns, the etiological factors are changing in India, and it is not very well documented [7]. One of the major complications of end-stage liver cirrhosis is hepatic encephalopathy, which is a serious condition but can be potentially reversible. It is sometimes challenging to understand the symptoms, as there are very subtle signs that can be detected only by conducting various tests. In the case of severe encephalopathy with acute liver failure, the mortality can reach more than 50% [8]. In this case report, I present a rare and interesting case of a 49-year-old male who Developed Decompensated Chronic Liver Disease (DCLD) with portal hypertension accompanying grade I encephalopathy.

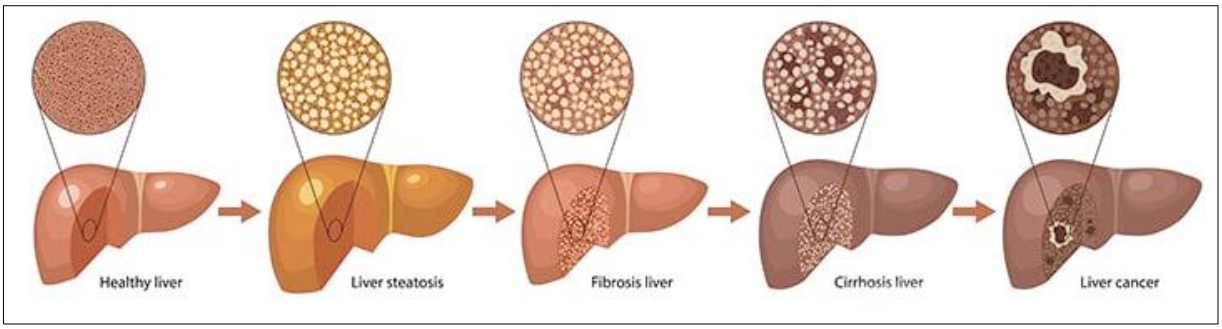

Fig 1: Stages of liver damage

Clinical Presentation

A 49-year-old male who has been a known alcoholic for 15 years came to the hospital for immediate treatment with complaints of breathlessness, low blood pressure, and swelling in the lower limb. Upon checking the patient, there was no history of fever, abdominal pain, or chest pain. However, the patient had similar complaints in the past and was admitted to the hospital 5 times. On arrival, his PR was 76 bpm, BP was

70/50 mm/Hg, and SpO_2 was 96%. ABG shows pH 7.31, pO_2 190.4, pCO_2 16.3, and HCO_3^- 12.3. The 2-D echo report shows normal cardiac chambers with adequate LV systolic function along with IVC: 17 mm, partially collapsing. Peripheral smear tests were also done, which show severe dimorphic anemia with neutrophilia & thrombocytopenia. The x-ray of the chest was clear. Other biochemistry results show ADA: 0.78 U/L, CRP: 41.01 mg/L, HB: 4.9 g/dL, RBC count: 1.39 million/cumm, PCV: 15.4%, platelet count: 0.27 lacks/cumm, WBC: 30660 cells/cumm, neutrophils: 85%, lymphocytes: 07%, eosinophils: 00%, and LDH: 224 U/L. RFT test shows urea: 128 mg/dl, serum creatinine: 2.6 mg/dl, serum sodium: 127 mmol/L, serum potassium: 6.4 mmol/L, and serum chloride: 110 mmol/L. Furthermore, the LFT test shows that total bilirubin is 11.2 mg/dl, direct bilirubin is 9.0 mg/dl, total protein is 3.9 g/dl, and AST or SGOT is 71. The serology report shows that the C-reactive protein was 71.35. Following the diagnostic tests, the patients were prescribed inj imipenem + cilastatin (250+250) IV in 100 ml NS, inj azithral 500 mg IV 1-0-0, inj H. albumin 20% 100 ml IV, 10 ml/hr, inj Mpraz 40 mg IV 1-0-0, inj optineron 1 amp in 100 ml NS, IV 0-1-0, inj Octreotide 100 mg in 50 ml NS @ 5 ml/hr, Tab Ursosan 400 mg 1-0-1, Tab Liveril forte 1-0-1, Tab Ursocol 300 mg 1-1-1, Tab Cipmido 10 mg 1-1-1, Tab Lobun forte 1-0-1, Tab Nefrosave 1-0-1, and Tab Nodosus 1 gm 1-1-1. Also, he was advised to inject Actrapid 1U in 25 IU in 100 ml over 1 hour. However, the patient took discharge against medical advice (DAMA) and left the hospital for further consultation.

Discussion

Decompensated chronic liver disease is a complex disease and requires proper management protocol. As liver cirrhosis patients can be severely immunocompromised, infections are more common along with ascites in the abdomen due to portal hypertension [9]. DCLD with portal hypertension remains clinically challenging, which can ultimately lead to acute on-chronic liver failure (ACLF). In the year 2015, the Food and Drug Administration (FDA) focused on the study related to endpoints and biomarkers. Therefore, the BEST (Biomarkers, Endpoints, and Other Tools) resource was published [10]. A study done by Ariza *et al*. found that urine NGAL (neutrophil gelatinase-associated lipocalin) could be a factor in developing ACLF [11]. So the BEST framework can guide novel therapies in ACLF. Furthermore, some of the other recent recommended therapies are gut-derived systemic inflammation and CAID (cirrhosis-associated immune dysfunction) as a targeted therapy, use of human albumin as medicine, and targeting hepatic regeneration as a therapy, etc [10]. Patients with DCLD have an increased risk of developing portal hypertension. Sepsis is a major problem in DCLD cases, which should be detected early by blood and urine culture, X-ray, and corrections of renal failure and electrolyte abnormalities. In the case of renal impairment, an assessment of the reversible factors such as dehydration, sepsis, and intrinsic renal disease has to be done. In the case of ascites, a salt-restricted diet is important, along with diuretics; prophylaxis and antibiotics need to be given as required [12]. In the case of hepatic encephalopathy, a low-protein diet is no longer recommended due to its ineffectiveness. Lactose can be administered orally, and an enema can be used to achieve loose stool in case of severe acute encephalopathy [13].

Conclusion

It is important for patients with decompensated liver diseases who are listed for suitable liver transplantation to have an extensive management plan for survival until the transplantation is done. DCLD is a serious problem with an increased mortality rate. Interprofessional team professionals such as specially trained renal nurses, nephrologists, urologists, neurologists, radiologists, pathologists, etc., are required for the proper hospital management of the patients.

Acknowledgements

We thank the patients for allowing us to share her case.

Ethical Approval

Ethical approval was taken as per international standards.

Conflicts of interests

No conflicts of interest were reported among the authors.

Funding: None declared.

Reference

1. Jagdish RK, Roy A, Kumar K, Premkumar M, Sharma M, Rao PN, *et al*. Pathophysiology and management of liver cirrhosis: from portal hypertension to acute-on-chronic liver failure. Frontiers in Medicine, 2023, 10.
2. Gülcicegi DE, Goeser T, Kasper P. Prognostic assessment of liver cirrhosis and its complications: current concepts and future perspectives. Frontiers in Medicine, 2023, 10.
3. Devarbhavi H, Asrani SK, Arab JP, Nartey YA, Pose E, Kamath PS. Global burden of liver disease: 2023 update. Journal of Hepatology. 2023;79(2):516-537.
4. Das M, Shil R. Awareness and influencing lifestyle modification factors regarding hepatitis B among non-medical students – efficacy of an educational package. International Journal of Advances in Nursing Management. 2022;10(4):285-291.
5. Mishra D, Dash KR, Khatua C, Panigrahi S, Parida PK, Behera SK, *et al*. Temporal trends in the etiology of cirrhosis of liver in coastal eastern Odisha. Euroasian Journal of Hepato-Gastroenterology. 2020;10(1):1–6.
6. Sinha S, Shil R. Knowledge and attitude towards liver cirrhosis due to alcoholism among commercial auto drivers in Bengaluru south region through VAT program: a pre-experimental study. International Journal of Advanced Research. 2023;11(2):118–128.
7. Cleveland Clinic. Hepatic encephalopathy, 2024. Available from: https://my.clevelandclinic.org/health/diseases/21220-hepatic-encephalopathy

8. Mandiga P, Kommu S, Bollu PC. Hepatic encephalopathy. In: StatPearls. Treasure Island (FL): StatPearls Publishing, 2025 Jan. Updated 2024 Mar 7. Available from: https://www.ncbi.nlm.nih.gov/books/NBK430869/
9. Mansour D, McPherson S. Management of decompensated cirrhosis. Clinical Medicine (London). 2018;18(Suppl 2):s60-s65.
10. Trebicka J, Hernaez R, Shawcross DL, Gerbes AL. Advances in prevention and treatment of decompensated cirrhosis and acute-on-chronic liver failure and the role of biomarkers. Gut. 2024;73(6):1015-1024.
11. Ariza X, Graupera I, Coll M, Solà E, Barreto R, García E, *et al*. Neutrophil gelatinase-associated lipocalin as a biomarker of acute-on-chronic liver failure and prognosis in cirrhosis. Journal of Hepatology. 2016;65(1):57-65.
12. GGC Medicines. Management of decompensated liver disease. Available from: https://handbook.ggcmedicines.org.uk/guidelines/gastrointestinal-system/management-of-decompensated-liver-disease/
13. Harrison PM. Management of patients with decompensated cirrhosis. Clinical Medicine. 2015;15(2):201-203.