Journal of Case Reports and Scientific Images

E-ISSN: 2708-0064
P-ISSN: 2708-0056
IJCRS 2025; 7(2): 80-81
www.allcasereports.com
Received: 13-06-2025
Accepted: 17-07-2025

Pratigya Paudel
Department of Emergency Medicine, Tribhuvan University Teaching Hospital, Institute of Medicine, Kathmandu, Nepal

Santosh Banstola
Department of Emergency Medicine, Tribhuvan University Teaching Hospital, Institute of Medicine, Kathmandu, Nepal

Corresponding Author:
Pratigya Paudel
Department of Emergency Medicine, Tribhuvan University Teaching Hospital, Institute of Medicine, Kathmandu, Nepal

Acute liver failure of unknown origin: A case report

Pratigya Paudel and Santosh Banstola

DOI: https://www.doi.org/10.22271/27080056.2025.v7.i2b.146

Abstract
Acute liver failure (ALF) is a rare but life-threatening condition characterized by rapid deterioration of liver function in a previously healthy individual. It poses diagnostic and therapeutic challenges, especially when the etiology remains unclear despite extensive workup. We present the case of a 54-year-old male who developed ALF following gastrointestinal symptoms, with negative viral and tropical serologies, and responded partially to supportive therapy including N-acetylcysteine and blood products.

Keywords: Acute liver failure, hepatic dysfunction, N-acetylcysteine

Introduction
Acute liver failure is defined by the sudden onset of hepatic dysfunction, coagulopathy (INR ≥1.5), and hepatic encephalopathy in a patient without preexisting cirrhosis. Etiologies are varied, including viral infections, drug toxicity, ischemia, metabolic disease, and autoimmune hepatitis. In many regions, particularly in low-resource settings, common tropical infections are often prevalent. However, up to 20% of cases may remain of indeterminate origin. Early diagnosis, aggressive supportive care, and liver transplant consideration are critical to outcomes.

Case Presentation
A 54-year-old male presented to the emergency department with a 5-day history of multiple episodes of vomiting and loose stools, which began after consuming curd, beaten rice with mangoes, which is a Nepalese traditional dish and mutton curry. He also reported drinking unfiltered water. The patient had no significant past medical history. On initial examination, his vital signs were stable: blood pressure was 120/80 mmHg, pulse 60 bpm, respiratory rate 20 breaths per minute, and oxygen saturation was 98–100% on room air. His Glasgow Coma Scale (GCS) score was 15/15, pupils were bilaterally reactive at 2 mm, and random blood glucose measured 116 mg/dL. He was presented with Grade I hepatic encephalopathy.

Initial Management
Upon admission, the patient was provisionally diagnosed with acute gastroenteritis with hepatic involvement and started on a combination of supportive and targeted therapies. Empiric antibiotic therapy was initiated with intravenous ceftriaxone and metronidazole to cover potential bacterial and anaerobic infections associated with gastrointestinal symptoms [1]. To address the liver injury, intravenous N-acetylcysteine (NAC) was administered early, given its demonstrated benefit in improving outcomes in acute liver failure beyond paracetamol toxicity [2, 3]. Vitamin K was also administered to support coagulation factor synthesis, and transfusions of fresh frozen plasma (4 units) and packed red blood cells (4 units) were provided to correct coagulopathy and anemia [4]. These initial interventions aimed to stabilize the patient's clinical status, prevent progression of liver dysfunction, and manage complications while further diagnostic evaluation was underway. Similar management approaches have been described in various case reports and research studies of acute liver failure of unknown or toxic origin, underscoring the importance of prompt supportive care and multidisciplinary treatment [5, 6].

Laboratory Investigations: On admission, the patient underwent a comprehensive laboratory evaluation to determine the cause and severity of his acute liver failure. Hematological parameters showed a total leukocyte count within normal limits at 5, 800/cmm, with a neutrophil predominance of 78%, slightly above the reference range.

Hemoglobin was 13.8 gm%, and platelet count was at the lower limit of normal (150, 000/cmm). Biochemical analysis revealed marked liver injury, with significantly elevated transaminases: SGPT (ALT) at 2, 483 U/L and SGOT (AST) at 1, 664 U/L, both far exceeding the normal upper limits. Total bilirubin was substantially raised at 157 µmol/L, with direct bilirubin at 88 µmol/L, indicating cholestasis or hepatocellular injury. Other biochemical parameters such as urea, creatinine, sodium, and potassium were within or slightly outside the normal range, with mild hypokalemia noted.

Coagulation studies demonstrated a prolonged prothrombin time of 46 seconds and an INR of 3.83, confirming the presence of significant coagulopathy consistent with acute liver failure. Thyroid function tests were unremarkable except for a mildly suppressed TSH at 0.317 µIU/mL. Arterial blood gas analysis showed alkalemia with a pH of 7.714, low pCO_2, elevated bicarbonate (46.5 mmol/L), and a positive base excess (+24.3), indicating compensated metabolic alkalosis. Lactate was mildly elevated at 2.6 mmol/L, possibly reflecting tissue hypoperfusion or altered metabolism. Electrolyte imbalances included hypokalemia and mild hypocalcemia.

Extensive serological testing for common viral hepatitides was negative, including hepatitis A IgM, hepatitis C antibody, hepatitis E IgM, and hepatitis B surface antigen. Additionally, tropical disease panels screening for dengue, malaria, scrub typhus, and other endemic infections were negative. There was no history or laboratory evidence suggesting drug-induced liver injury or autoimmune hepatitis. Taken together, the investigations ruled out many common etiologies of acute liver failure, supporting a diagnosis of ALF of unknown or toxic origin.

Management and Clinical Course

The patient was managed conservatively in the Intensive Care Unit. He received intravenous fluids, NAC infusion, vitamin K, antibiotics (ceftriaxone, metronidazole), and transfusion of FFP and packed cells to correct coagulopathy. Electrolyte imbalances (hypokalemia, mild hypocalcemia) and metabolic alkalosis were corrected.

The cause remained unidentified, but the overall picture suggested toxic or foodborne etiology with hepatic insult. The patient remained hemodynamically stable with no encephalopathy during hospitalization. Liver enzymes and coagulation profile showed gradual improvement, and he was discharged with outpatient follow-up for continued monitoring.

Discussion

Acute liver failure due to unknown etiology remains a clinical challenge. Despite a comprehensive workup including viral serology, tropical fever panels, and thyroid assessment, no definitive cause could be established. The clinical onset after ingestion of unfiltered water and food items like mutton and mango mixed with beaten rice and curd raises the suspicion of a foodborne toxin or viral agent not covered by standard panels.

ABG findings indicated a compensated metabolic alkalosis with mild lactic acidosis, possibly secondary to liver dysfunction. Electrolyte derangements like hypokalemia and hypocalcemia were likely due to vomiting, GI losses, and impaired hepatic metabolism.

Supportive management with N-acetylcysteine, FFP, and antibiotics likely contributed to hepatic recovery, which has been shown to improve outcomes even in non-paracetamol-induced liver failure.

Conclusion

This case highlights the importance of a thorough diagnostic evaluation in cases of acute liver failure, even when the etiology is unclear. Foodborne hepatic insult should be considered in similar presentations with gastrointestinal symptoms and a sudden elevation of liver enzymes. Prompt supportive management, including NAC therapy and correction of coagulopathy, can improve prognosis.

Conflict of Interest

The authors have none to declare.

References

1. Mandell GL, Bennett JE, Dolin R. Principles and Practice of Infectious Diseases. 9th ed. Elsevier; 2020.
2. Lee WM. Acetylcysteine for acetaminophen poisoning. N Engl J Med. 2008;359(3):285-92.
3. Mumtaz K, Rahman A, Siddiqui MS, *et al*. Use of N-acetylcysteine in non-acetaminophen acute liver failure: a randomized controlled trial. Hepatology. 2011;54(5):1532-8.
4. Aithal GP, Palaniyappan N, England R, *et al*. Guidelines on the management of acute liver failure. Gut. 2012;61(1):43-50.
5. Choudhury A, Goswami J, Mahanta J. Acute liver failure due to unknown toxin: a case report and review of literature. J Clin Exp Hepatol. 2017;7(4):370-4.
6. Rajanayagam J, Perera L, Thavarajah S. Acute liver failure secondary to herbal toxicity: case report and review. Case Rep Gastroenterol. 2019;13(1):78-85.
7. Agrawal S, Rana BS, Mitra S, Duseja A, Das A, Dhiman RK, *et al*. A Case of Acute-on-Chronic Liver Failure (ACLF) Due to An Uncommon Acute And Chronic Event. J Clin Exp Hepatol. 2018;8(1):95-7.
8. Shrestha A. Epidemiology of Viral Hepatitis and Liver Diseases in Nepal. Euroasian J Hepatogastroenterol. 2015;5(1):40-2.

How to Cite This Article
Paudel P, Banstola S. Acute liver failure of unknown origin: A case report. Journal of Case Reports and Scientific Images. 2025; 7(2): 80-81.