International Journal of Research and Review
[Indexed, Peer-Reviewed Journal]
Volume 12; Issue: 6; June 2025
Website: www.ijrrjournal.com
Case Report *E-ISSN: 2349-9788; P-ISSN: 2454-2237*

From Hepatitis to Heartbreak: A Rare Encounter with Myocarditis

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DOI: https://doi.org/10.52403/ijrr.20250693

ABSTRACT

Background: Symptoms of viral myocarditis include cardiogenic shock and deadly arrhythmia. In certain people, the condition can progress to chronic myocarditis and dilated cardiomyopathy. We describe a case of Hepatitis A virus-induced hepatitis with viral myocarditis.

Case presentation: A 42-year-old woman with anorexia, exhaustion, and fever was brought to our hospital. Computed CT revealed diffuse liver edema and an interior heterogeneous image, and the initial investigation revealed raised liver enzyme levels and creatine phosphokinase. Lab investigations revealed positive anti-HAV IgG by ELISA (0.68), consistent with recent Hepatitis A infection. Liver function tests showed marked hepatocellular injury with total bilirubin of 9.7 mg/dL, conjugated bilirubin of 2.4 mg/dL, AST 3849 U/L, and ALT 2024 U/L. Echocardiography revealed a dilated, spherical ventricle with decreased systolic function and pericardial effusion, while electrocardiography revealed broad ST-segment elevation. Chest X-ray was unremarkable. Abdominal ultrasound showed hepatomegaly, mild ascites, an oedematous gallbladder with sludge, and gamma-Gandy bodies in the spleen.

Conclusion: We reported a rare instance of Hepatitis A virus-induced myocarditis and hepatitis. Since hepatitis, pancreatitis, nephritis, and encephalitis can all coexist with acute myocarditis, it is essential to identify the presence of additional infectious disorders in order to treat the patient appropriately.

Keywords: Myocarditis, Hepatitis, Case report

INTRODUCTION

From moderate dyspnea to chest discomfort, cardiogenic shock, and deadly arrhythmia, myocarditis can manifest with a variety of symptoms. Myocarditis is primarily caused by recent or ongoing viral infections[1]. Hepatitis A is an RNA virus of the picornavirus family. Infection typically presents as a self-limiting acute hepatitis[2]. Clinical symptoms of anorexia, nausea and vomiting, fatigue, malaise, arthralgias, headache, cough, and low grade fever are usually found. Rarely, it can be complicated by extrahepatic manifestations[3]. Cardiac manifestations such as myocarditis and dilated cardiomyopathy are extremely rare and very sparsely reported in HAV infections[4]. Historically, enteroviruses—more especially, Coxsackievirus (CV) group B serotypes—have been thought to be the main viral cause[4], despite the fact that human hepatitis virus 6, parvovirus B19, and adenoviruses can potentially induce myocarditis[5-7]. Viral myocarditis is usually caused by RNA viruses, principally the

enteroviruses, coxsackie virus, echovirus, poliovirus and influenza virus. DNA viruses like adenovirus and hepatitis viruses are also known to be the culprit. In rare cases HIV and Hepatitis C Virus are also known to cause the same. Here we present an unusual case wherein a patient presented with post infective myocarditis after previously being infected by Hepatitis A virus.

CASE PRESENTATION

We report the case of a 42-year-old female with no known comorbidities who presented with intermittent low-grade fever for two weeks, followed by chest discomfort, diffuse epigastric pain, respiratory distress, and multiple episodes of vomiting over one week. She had a recent diagnosis of acute Hepatitis A infection a few weeks prior. On admission, her blood pressure was 120/60 mmHg, heart rate was 82 beats per minute (bpm), respiratory rate was 21 breaths per minute, and temperature was 98.6°F. All serological investigation performed at admission were listed in table 1. She was without clinical signs of respiratory distress, palpitations, or abdominal tenderness. Auscultation revealed normal bilateral breath sounds, and cardiovascular examination was unremarkable. However, she reported persistent nausea, vomiting, and mild arthralgia. Relevant investigations pertaining to her condition were sent. Lab investigations revealed positive anti-HAV IgG by ELISA (0.68), consistent with recent Hepatitis A infection. Liver function tests showed marked hepatocellular injury with total bilirubin of 9.7 mg/dL, conjugated bilirubin of 2.4 mg/dL, AST 3849 U/L, and ALT 2024 U/L. Serum proteins were mildly reduced (total protein 6.2 g/dL, albumin 3.5 g/dL, globulin 2.7 g/dL), while alkaline phosphatase was 131 U/L. Lactate dehydrogenase was significantly elevated at 6033 U/L. Muscle enzymes showed mildly raised CPK (112 U/L) and CPK-MB (19 U/L). Lactate dehydrogenase (LDH) levels were markedly elevated at 6033 U/L. Renal parameters were within normal limits with blood urea 47 mg/dL and serum creatinine 0.8 mg/dL. Electrolytes were normal (Na^+ _135 mEq/L, K^+ _3.9 mEq/L). Coagulation profile was deranged, with PT 29.1 seconds, APTT 35.9 seconds, and INR 2.45. Lipid profile showed hypocholesterolemia: total cholesterol 74 mg/dL, LDL 46 mg/dL, and triglycerides 83 mg/dL. Inflammatory markers were elevated, including CRP (24 mg/L) and ESR (47 mm/hr). Hemogram revealed normocytic anemia (Hb 9.6 g/dL, hematocrit 31.7%), leukocytosis (TLC 11,500/cumm), and mild thrombocytopenia (platelets 1.40 lakh/cumm). Iron studies showed elevated serum iron (295 mcg/dL), ferritin (801 ng/mL), and transferrin saturation (80.4%) with normal TIBC (367 mcg/dL). ECG demonstrated QS complexes in V1/V2 with nonspecific ST-T changes. Transthoracic Echocardiogram revealed a mildly dilated left ventricle with global hypokinesia, reduced ejection fraction (LVEF 36%), severe pulmonary arterial hypertension, and mild pericardial effusion (Figure 1). Liver Function Test (LFT) trends were documented in table 2 and LDH with Liver Function trends were illustrated in figure 3. Chest X-ray was unremarkable. Abdominal ultrasound showed hepatomegaly, mild ascites, an oedematous gallbladder with sludge, and gamma-Gandy bodies in the spleen. A CT angiography of her coronaries revealed normal coronary arteries, aorta, and pericardium. The patient was managed conservatively with supportive measures, including intravenous fluids, ceftriaxone, ursodeoxycholic acid, and statins, alongside close monitoring of hepatic and cardiac function. As the patient started to improve, liver function tests and LDH started to trend down. A follow-up echocardiogram revealed an improved ejection fraction of 48%. Our patient was discharged home with a plan for follow-up to continue monitoring labs.

Table 1: Laboratory data upon admission

Parameter	Recorded Value
Hemoglobin (Hb) (gm/dl)	9.6
Hematocrit (%)	31.7
Total Leukocyte Count (TLC) (L/cumm)	11500
Platelets (L/cumm)	1.40
Erythrocyte Sedimentation Rate (ESR) (mm/hr)	47
Serum Iron (mcg/dl)	295
Ferritin (mg/dl)	801
Total iron-binding capacity (TIBC) (mcg/dl)	367
Transferrin Saturation (%)	80.4
Total bilirubin (mg/dl)	9.7
Conjugate bilirubin (mg/dl)	2.4
Total protein (g/dl)	6.2
Albumin (g/dl)	3.5
Globulin (mg/dl)	2.7
Aspartate aminotransferase (AST) (U/L)	3849
Alanine transaminase (ALT) (U/L)	2024
Alkaline Phosphatase (U/L)	131
Lactate Dehydrogenase (U/L)	6033
Creatine phosphokinase (CPK) (U/L)	112
Creatine kinase-myocardial band (CK-MB) (U/L)	19
Blood Urea (mg/dl)	47
Serum Creatinine (mg/dl)	0.8
Na^+ (mEq/L)	135
K^+ (mEq/L)	3.9
Haptoglobin (mg/dl)	57.5
Activated Partial Thromboplastin Clotting Time (APTT)(Seconds)	35.9
Prothrombin time (PT)	29.1
International normalised ratio (INR)	2.45
Total Cholesterol (mg/dl)	74
LDL Cholesterol (mg/dl)	46
Triglyceride (mg/dl)	83

Table 2: Liver Function Test (LFT) trends

Liver Function Test	Day 1	Day 4	Day 7
Total Bilirubin (mg/dL)	9.7	5.2	4.9
Conjugated Bilirubin (mg/dL)	2.4	1.2	2.9
AST/SGOT (U/L)	3849	1311	365
ALT/SGPT (U/L)	2024	812	565
Alkaline Phosphatase (U/L)	131	132	126
Total Protein (g/dL)	6.2	6.2	7.1
Albumin (g/dL)	3.5	3.4	3.2
Globulin (g/dL)	2.7	2.8	3.9

Figure 1: ECG showing QS pattern in V1/V2 along with Non specific ST-T changes

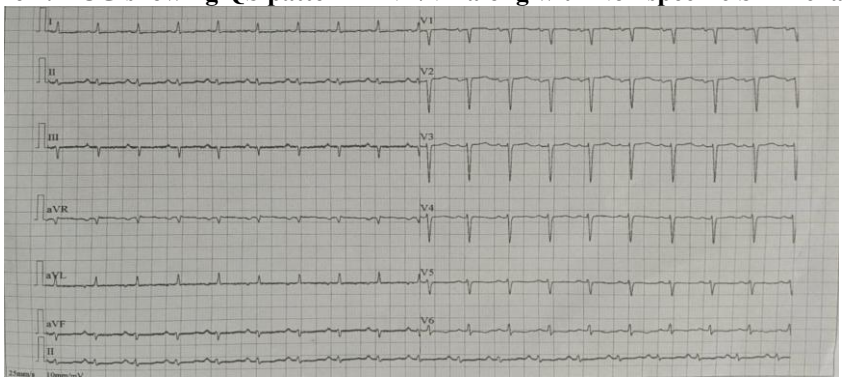
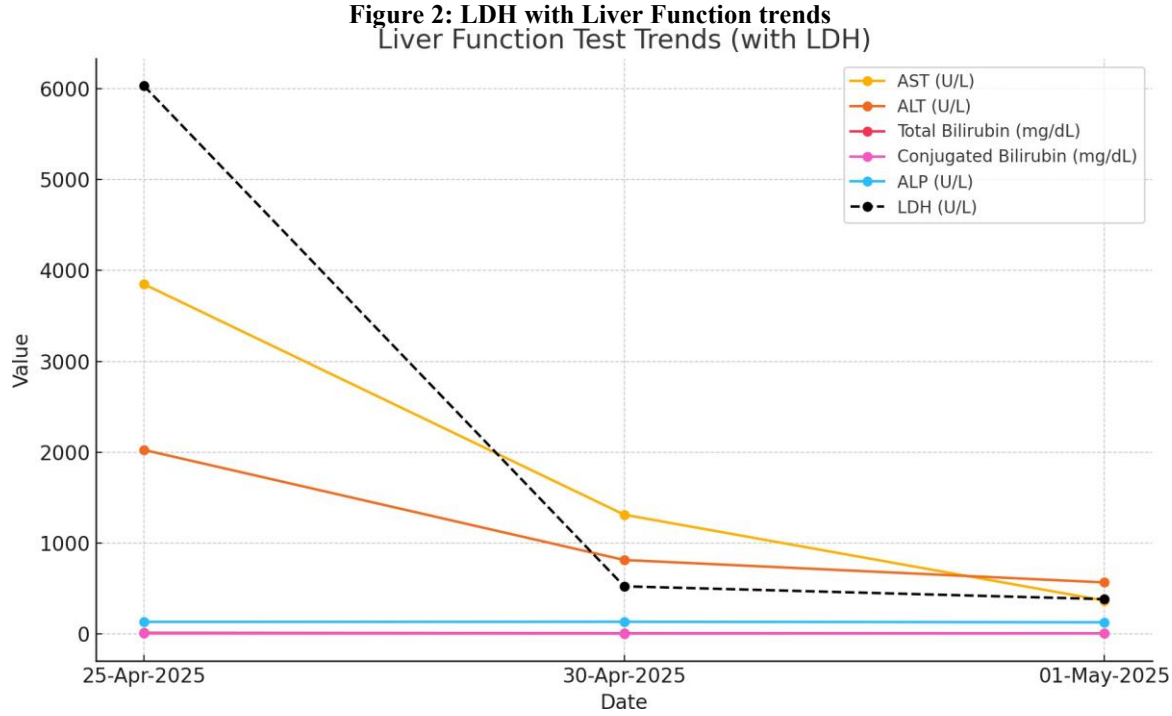


Figure 2: LDH with Liver Function trends



DISCUSSION

Hepatitis A virus (HAV) is transmitted almost exclusively via the fecal-oral route, with outbreaks linked to poor hygiene, overcrowding, and contaminated food or water—common sources include shellfish, produce, and milk. Person-to-person spread is common within families and institutions. It is classically considered a self-limiting illness, predominantly affecting the liver. Hepatic manifestations are well recognized, with high transaminases (AST>ALT), presenting as a self-limiting illness characterized by fever, malaise, anorexia, nausea, vomiting, and jaundice[8]. While hepatic involvement is the primary feature, extrahepatic manifestations are rare and generally underreported[9]. Among these, cardiac involvement such as acute myocarditis is exceptionally uncommon, with only a handful of cases described in the literature[10-13]. Viral myocarditis is most frequently associated with RNA viruses, particularly enteroviruses such as coxsackievirus B, echovirus, and poliovirus. Other recognized viral causes include adenovirus, parvovirus B19, influenza virus, human hepatitis B viruses, HIV, and hepatitis C virus[14]. The pathogenesis typically involves direct viral invasion of cardiac myocytes, followed by immune-mediated myocardial injury. However, HAV is not generally considered cardiotropic, and its role in myocardial inflammation is poorly understood[15]. Proposed mechanisms include immune-mediated injury secondary to systemic inflammation, cross-reactive antibodies, or cytokine-induced damage. Our patient demonstrated classical signs of acute HAV infection with markedly elevated transaminases and bilirubin[16]. The concurrent development of chest discomfort, ECG abnormalities, and echocardiographic evidence of global left ventricular hypokinesia and reduced ejection fraction pointed to myocarditis[17]. In contrast to earlier publications, there were very few reports of obtaining a pathology specimen throughout the patient's lifespan. Despite our inability to prove HAV by histopathological examination, it was evident from serology that HAV was the infection's source. The exclusion of other cardiotropic viruses and the temporal relationship with HAV support a post-infectious or immune-mediated etiology. This case is clinically significant because it highlights a rare but potentially serious extrahepatic complication of a common viral infection. With improved sanitation and widespread vaccination in

endemic countries, adults now represent a larger proportion of susceptible individuals[18]. Given that myocarditis can progress to heart failure or arrhythmias if unrecognized, early cardiac evaluation in patients with HAV and unexplained cardiovascular symptoms is warranted. This report contributes to the limited literature on HAV-associated myocarditis and underscores the importance of maintaining a broad differential diagnosis when managing viral hepatitis.

It is frequently assumed that ischemic hepatitis is the cause of liver enzyme increase in myocarditis patients. Although hepatitis and myocarditis are sometimes coupled, we are generally unable to do liver biopsies due to the patient's systemic state and coagulation issue. Determining whether other infectious lesions are present is crucial since acute myocarditis can coexist with hepatitis, pancreatitis, nephritis, and encephalitis.

CONCLUSION

Although Hepatitis A is typically a self-limiting hepatic illness, this case highlights its potential to cause rare and serious extrahepatic complications such as acute myocarditis. The temporal association, clinical presentation, and echocardiographic findings support an immune-mediated cardiac involvement secondary to HAV infection. Given the rarity of such presentations, heightened clinical awareness is essential, especially in adult patients presenting with cardiovascular symptoms following HAV. Early recognition and supportive management can significantly improve outcomes. This case adds to the limited literature on HAV-associated myocarditis and underscores the importance of considering atypical complications in common viral infections.

Consent For Publication: Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor of this journal.

Competing Interests: The authors declare that they have no competing interests.

Authors Contribution: Roshmi Bag managed the case and redaction and correction of the manuscript. Rohit Das also assisted with clinical management of the case and correction of the manuscript. Rishad Ahmed assisted with manuscript correction and redaction of comments from the illustrations. All authors read and approved the final manuscript.

Declaration by Authors

Ethical Approval: Ethics approval and consent for this case report were waived.

Acknowledgement: Author gratefully appreciate all departmental staff of for supporting throughout the research and the study participants for their meticulous information. Author also thankful to intigent research for their help in medical writing of this case reports.

Source of Funding: None

Conflict of Interest: The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

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How to cite this article: Roshmi Bag, Rohit Das, Rishad Ahmed. From hepatitis to heartbreak: a rare encounter with myocarditis. *International Journal of Research and Review*. 2025; 12(6): 828-833. DOI: *10.52403/ijrr.20250693*

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