OPEN ACCESS

Saudi Journal of Medical and Pharmaceutical Sciences
Abbreviated Key Title: Saudi J Med Pharm Sci
ISSN 2413-4929 (Print) | ISSN 2413-4910 (Online)
Scholars Middle East Publishers, Dubai, United Arab Emirates
Journal homepage: https://saudijournals.com

Case Report **Gastroenterology**

Delayed Diagnosis of Autoimmune Hepatitis Unmasked by Acute Hepatitis A: A Case Report and Literature Review

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DOI: https://doi.org/10.36348/sjmps.2025.v11i06.008 | **Received:** 15.05.2025 | **Accepted:** 21.06.2025 | **Published:** 24.06.2025

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Abstract

Introduction: Autoimmune hepatitis (AIH) is a chronic inflammatory liver disease of unknown etiology that can lead to cirrhosis and liver failure if left untreated. Environmental triggers, particularly viral infections, have been implicated in disease onset. ***Case Presentation:*** We report the case of a 24-year-old woman who presented with abdominal pain, nausea, and cholestatic jaundice. Initial serological workup revealed acute hepatitis A (HAV) infection. Despite conservative management, liver function continued to deteriorate. Autoimmune screening showed high-titer antinuclear antibodies (ANA), and liver biopsy revealed interface hepatitis with portal lymphoplasmacytic infiltrates and fibrosis (A3F1), consistent with AIH. ***Conclusion:*** This case highlights the potential role of HAV infection as a trigger for autoimmune hepatitis. In cases of persistent liver dysfunction after acute viral hepatitis, clinicians should maintain a high index of suspicion for evolving autoimmune liver disease.

Keywords: Jaundice, Viral Hepatitis A, Autoimmune Hepatitis, Hepatotocix Exposure, Rubia Tinctorum.

INTRODUCTION

Autoimmune hepatitis (AIH) is a chronic inflammatory liver disease that can lead to cirrhosis and liver failure.

AIH can present in all ages, races, and ethnicities, but it predominantly affects women. As a heterogeneous disease, AIH presents variably in different patients, making diagnosis and treatment a challenge [1]. Previous case reports indicate that various forms of viral hepatitis, including hepatitis A, may act as triggers for autoimmune hepatitis.

We report a particular case of severe acute viral hepatitis A revealing an autoimmune hepatopathy.

OBSERVATION

Patient Information

A 24-year-old woman with a history of untreated iron-deficiency anemia presented with two weeks of cholestatic jaundice and abdominal pain.

There was no history of hepatotoxic medication use or herbal remedies at the initial presentation.

Clinical Findings and Initial Workup

On examination, the patient was alert, afebrile, and visibly jaundiced. Laboratory investigations showed severe hepatocellular injury with AST 15× ULN and ALT 12× ULN. Total bilirubin was 106 mg/L (predominantly conjugated), with mild cholestasis (GGT 1.3× ULN; ALP 2.7× ULN). Coagulation was impaired with a prothrombin time (PT) of 48%. Renal function and albumin levels were normal. Complete blood count was within normal limits.

Abdominal ultrasound was unremarkable. Serology confirmed acute hepatitis A (positive HAV IgM); hepatitis B, C, E, and other viral markers (EBV, CMV, HSV, HIV) were negative.

Clinical Course and Diagnostic Challenge

The patient initially received supportive care with symptomatic treatment and vitamin

supplementation. Although liver enzymes and jaundice began to improve, complete resolution was not achieved.

Two weeks after initial presentation, while jaundice had only partially regressed, the patient reported self-medicating with *madder* (Rubia tinctorum), a plant known for its potential hepatotoxicity. Shortly after, she experienced worsening jaundice and a resurgence of liver cytolysis, although her mental status remained intact. Laboratory testing showed a marked increase in liver enzymes (AST 20× ULN, ALT 32× ULN) and worsening liver function, with a prothrombin time falling below 50%.

Given the lack of clinical resolution, the known hepatotoxic exposure, and the absence of other infectious, metabolic, or drug-related causes, the possibility of an autoimmune process was considered. As autoimmune hepatitis remains a diagnosis of exclusion, a comprehensive autoimmune workup was performed, which revealed high-titer antinuclear antibodies (ANA at 1:640). A liver biopsy was also performed, demonstrating interface hepatitis with periportal necrosis (figure 1) and septal fibrosis (A3F1) (figure 2), highly suggestive of autoimmune hepatitis.

Therapeutic Intervention and Follow-up

The patient was started on prednisolone (1 mg/kg/day), followed by azathioprine for maintenance and a slow steroid taper. Clinical improvement was rapid, with normalization of liver enzymes, bilirubin, and coagulation parameters. She remained stable without relapse during follow-up (Table 1).

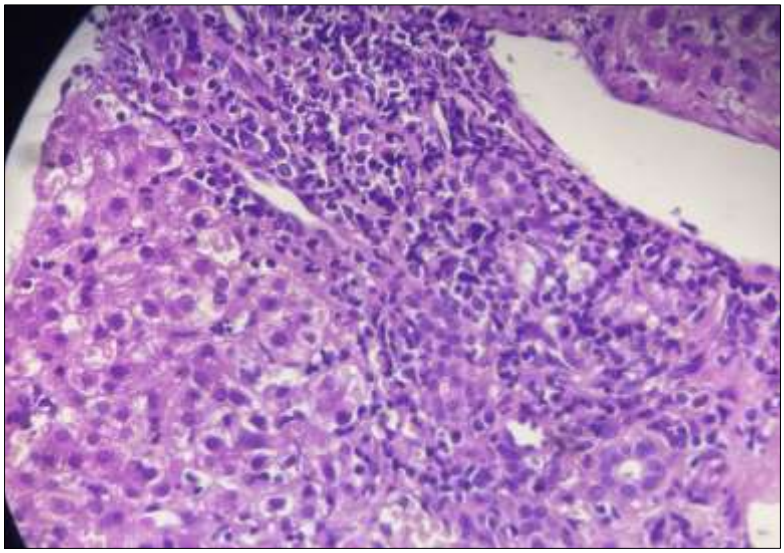

Figure 1: Liver biopsy showing interface hepatitis with dense portal lymphoplasmacytic infiltrates extending into the periportal hepatocytes (H&E stain, ×200)

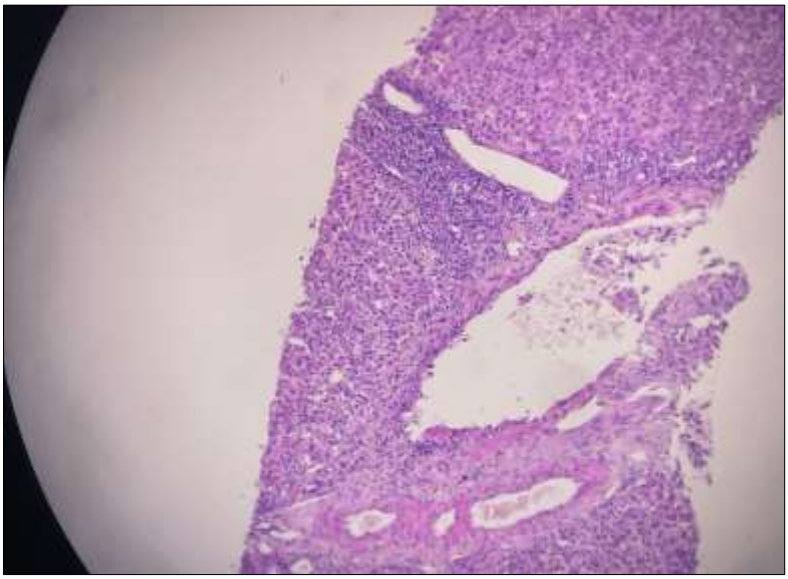

Figure 2: Fibrous expansion of portal tracts with early septal fibrosis (stage F1) and ongoing interface activity (H&E stain, ×400)

Table 1: Kinetics of liver function tests in our patient

Phase / Day	AST (U/L)	ALT (U/L)	Bilirubin (mg/dL)	GGT (U/L)	ALP (U/L)	PT (%)	Albumin (g/dL)	Remarks
Day 0 – Initial diagnosis (HAV IgM+)	~600 (15× ULN)	~670 (12× ULN)	10.6	Mild ↑ (1.3× ULN)	2.7× ULN	48%	Normal	Acute hepatitis A, predominantly conjugated hyperbilirubinemia
Day 10 – Partial improvement	↓ ~300	↓ ~350	~7	Stable	↓ ~1.8× ULN	~55%	Normal	Clinical improvement but incomplete normalization
Day 14 – After Rubia tinctorum intake	↑ ~800 (20× ULN)	↑ ~1800 (32× ULN)	↑ >12	NA	NA	<50%	Normal	Worsening cytolysis; suspicion of drug-induced injury
Day 20 – Further workup	~750	~1600	~11	↑	↑	45%	Slightly low	Diagnosis of autoimmune hepatitis confirmed (ANA/SMA positive, high IgG)
Day 21 – Start of corticosteroids	—	—	—	—	—	—	—	Initiation of immunosuppressive therapy
Day 35 – Clinical improvement	↓ ~200	↓ ~400	↓ ~5	↓	↓	~60%	Normalizing	Biochemical response to corticosteroids
Day 60 – Near normalization	~60	~70	~1.8	Normal	Normal	~80%	Normal	Good response; close to full remission

DISCUSSION

Hepatitis A virus (HAV) is a non-enveloped RNA Picornavirus, responsible annually for almost 1.5 million cases of acute hepatitis. It is the most common cause of acute viral hepatitis worldwide, causing substantial morbidity [2]. Hepatitis A virus is mainly transmitted through fecal-oral route either by direct contact of a susceptible person with an infectious person (generally through contaminated hands) or by ingestion of contaminated food or water. It may occasionally progress to severe disease, especially among elderly population [3, 4].

The infection occurs more frequently in populations of less economically-developed regions with little education and poor hygiene. Case series and small retrospective studies have linked viruses such as Epstein-Barr virus, varicella zoster virus, viral hepatitis A to AIH [5].

Unfortunately, these potentially important triggers are seldom considered systematically in clinical practice. Further evidence supports an environmental role in AIH pathogenesis, as factors such as microbiome diversity [6], and psychosocial stress have also been associated with disease.

In our case, autoimmune hepatitis was identified on the basis of an IAIHG score of 18, and viral infection A was the triggering factor, based on a serological profile indicating a transitional phase, attesting to the originality of this case.

In the literature, we find an autoimmune hepatitis triggered by an E viral infection and an HAI triggered by an EBV viral infection. These case reports should encourage practitioners to be on the lookout for the onset of an autoimmune disease and, more specifically, autoimmune hepatitis following EBV infection, particularly in predisposed subjects, i.e. females and patients with a family history of dysimmune disease. Subsequent prognosis would depend on the evolutionary history of the underlying liver disease. Biological remission in our patient was achieved on corticosteroids after 4 weeks of corticosteroids, followed by a switch to azathioprine.

A similar case was recently reported by Lee *et al*., [7], in which a 55-year-old woman developed autoimmune hepatitis after an acute HAV infection. Like our patient, she had persistent elevation of liver enzymes and positive autoimmune markers, with biopsy findings confirming AIH. However, the patient in their report improved spontaneously without immunosuppressive therapy. In contrast, our patient experienced worsening liver function—potentially exacerbated by hepatotoxic plant exposure—and required corticosteroids and azathioprine to achieve remission. This difference underscores the clinical variability of HAV-induced AIH and the importance of individualized treatment based on disease severity.

Our case adds to the growing evidence suggesting that acute HAV infection can act as a trigger for autoimmune hepatitis, especially in predisposed

individuals (e.g., young women). Clinicians should remain vigilant for autoimmune liver disease in cases of unexplained or prolonged liver dysfunction following viral hepatitis.

CONCLUSION

Given the lack of concrete scientific data corroborating the hypotheticals implicating HAV in the triggering of autoimmune hepatitis and the contribution of environmental factors in AIH pathogenesis. This is a challenging situation whereas there are not many cases described in the literature. Replication of these findings and prospective examination may provide new insight into AIH onset and outcomes.

Declaration of Conflicting Interests: The author(s) declared no potential conflicts of interest with respect to the research, authorship and/or publication of this article

Funding: The author(s) received no financial support for the research, authorship and/or publication of this article

Ethical Approval: Our institution does not require ethical approval for reporting individual cases or case series

Informed Consent: Written informed consent was obtained from the patient(s) for their anonymized information to be published in this article

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