Section: Miscellaneous

Case Report

ACUTE PANCREATITIS AS AN ATYPICAL INITIAL PRESENTATION OF HEPATITIS A VIRUS INFECTION IN AN ADOLESCENT MALE: A RARE CASE REPORT

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ABSTRACT

Background: Hepatitis A virus (HAV) infection is a common cause of acute viral hepatitis, particularly in developing countries. It is typically self-limiting and confined to hepatic involvement. However, extrahepatic manifestations such as acute pancreatitis are exceedingly rare and can pose a diagnostic challenge.

Case Presentation: We report an 18-year-old previously healthy male who presented with acute epigastric pain radiating to the back, accompanied by constipation and absence of flatus for two days. Laboratory investigations revealed markedly elevated serum amylase and lipase levels, consistent with acute pancreatitis. Notably, lactate dehydrogenase (LDH) was extremely elevated at 9725 IU/L, reflecting extensive tissue injury. Further evaluation for hepatic dysfunction showed raised transaminases and positive HAV IgM serology, confirming acute hepatitis A infection. Ultrasound abdomen demonstrated a bulky pancreas with mild peripancreatic fluid and no biliary obstruction. The patient was managed conservatively with bowel rest, intravenous fluids, and analgesics, leading to complete clinical and biochemical recovery.

Conclusion: This case highlights a rare but important extrahepatic manifestation of HAV infection. Acute pancreatitis may occasionally be the initial presentation of hepatitis A, and clinicians should maintain a high index of suspicion when evaluating pancreatitis of unclear etiology, especially in young, non-alcoholic patients with abnormal liver function tests. Extremely high LDH may reflect combined hepatic and pancreatic injury and warrants close monitoring.

Keywords: Hepatitis A, acute pancreatitis, extrahepatic manifestation, adolescent, viral pancreatitis, LDH.

INTRODUCTION

Hepatitis A virus (HAV) is an RNA virus transmitted via the fecal–oral route, most commonly causing acute viral hepatitis. Although hepatic involvement predominates, extrahepatic manifestations such as pancreatitis, nephritis, arthritis, and hematologic abnormalities have been reported but are rare. Among these, pancreatitis secondary to HAV infection is particularly uncommon, especially in otherwise healthy adolescents.

Although HAV primarily targets hepatocytes, extrahepatic involvement has been documented in about 10–15% of symptomatic patients, typically involving renal, hematologic, and pancreatic systems (Kumar et al., 2017). The true incidence of pancreatitis in HAV infection is uncertain, but small studies have suggested a frequency of 0.7–1.4% among hospitalized patients with acute hepatitis A (Khuroo et al., 1987; Jain et al., 2012). Because hepatic manifestations may precede or follow pancreatic inflammation, the causal link is often overlooked unless viral serology is systematically performed.

We present a rare case of HAV infection initially manifesting as acute pancreatitis, with markedly

elevated LDH, underscoring the need to consider viral etiologies in patients presenting with pancreatitis of unexplained origin.

Case Presentation

An 18-year-old male student with no significant past medical or surgical history presented to the emergency department with sudden-onset severe epigastric pain radiating to the back, associated with nausea and absolute constipation (no passage of stools or flatus) for two days. There was no history of fever, jaundice, alcohol intake, drug use, or trauma.

On examination, the patient was alert, afebrile, and hemodynamically stable. Abdominal examination revealed diffuse tenderness with sluggish bowel sounds and no guarding or rigidity.

Laboratory investigations were as follows

Parameter	Result	Reference Range	Interpretation
Serum Amylase	430 U/L	25–125 U/L	Pancreatic involvement
Serum Lipase	720 U/L	10–140 U/L	Pancreatic involvement
ALT	180 U/L	7–56 U/L	Hepatocellular injury
AST	165 U/L	5–40 U/L	Hepatocellular injury
Total bilirubin	1.8 mg/dL	0.2–1.2 mg/dL	Mild cholestasis
Alkaline phosphatase	Mildly elevated	44–147 U/L	Mild cholestasis
LDH	9725 IU/L	140–280 IU/L	Markedly elevated; reflects combined hepatic and pancreatic injury
Serum calcium	9.2 mg/dL	8.5–10.5 mg/dL	Normal
Triglycerides	110 mg/dL	<150 mg/dL	Normal
Renal function	Normal	—	—

Viral hepatitis serology revealed positive HAV IgM and negative HBsAg and anti-HCV antibodies, confirming acute hepatitis A infection.

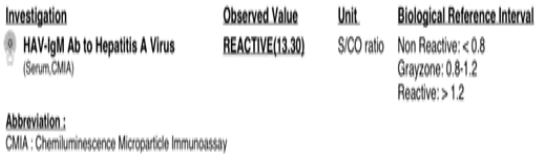

Figure 1: Positive HAV IgM antibodies

Ultrasonography of the abdomen showed a bulky pancreas with mild peripancreatic fluid and no biliary obstruction or gallstones. A diagnosis of acute pancreatitis secondary to HAV infection was made. A CECT Whole Abdomen was also done which showed Acute Pancreatitis with A CTSI Score 4/10 and mild hepatomegaly.

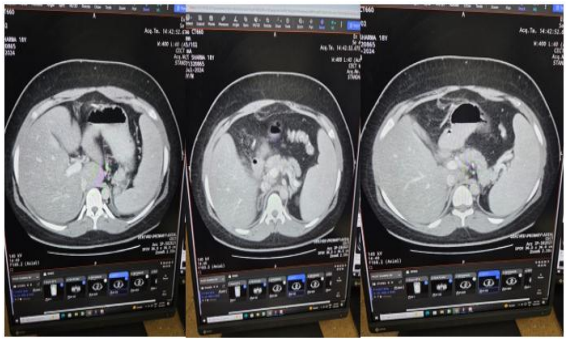

Figure 2: Axial views of contrast enhanced computed tomography scans of abdomen showing bulky head of pancreas with adjacent peri pancreatic fat stranding s/o acute pancreatitis

The patient was managed conservatively with intravenous fluids, analgesics, and bowel rest. Over the next five days, his symptoms improved, and serum enzyme levels normalized. He was discharged in stable condition with advice for follow-up.

DISCUSSION

HAV infection is a common cause of acute viral hepatitis in developing countries but is rarely associated with pancreatitis. Pancreatic involvement in viral hepatitis is believed to occur due to direct viral cytopathic effect or immune-mediated injury, though the exact pathogenesis remains unclear (Yadav et al., 2013).

Proposed mechanisms include

1. Direct viral invasion of pancreatic acinar cells leading to cytolysis.
2. Immune cross-reactivity between HAV antigens and pancreatic tissue causing inflammation.
3. Ampullary edema from biliary tract inflammation, leading to transient pancreatic duct obstruction (Vasudevan et al., 2015).

Experimental data have detected HAV RNA in pancreatic tissue, supporting a direct cytopathic mechanism (Sagnelli et al., 2006).

In our patient, LDH was extremely elevated (9725 IU/L), far higher than typically observed in isolated viral hepatitis. This rise likely reflects combined hepatocellular and pancreatic injury. LDH elevation is also incorporated in Ranson's criteria for assessing acute pancreatitis severity, with levels >350 IU/L indicating more severe disease. The exceptionally high LDH in our case underscores the systemic impact of the combined hepatic and pancreatic injury, even though the patient responded well to conservative management. Clinicians should recognize that markedly raised LDH in this setting signals significant cellular damage and warrants close monitoring for complications.

A literature review by Rawla et al. (2018) identified fewer than 25 reported cases of HAV-associated pancreatitis worldwide. Most occurred in children or young adults and followed a mild, self-limited course. Similarly, Bhatti et al. (2014) reported a young male presenting with pancreatitis as the first

manifestation of HAV, who recovered fully with conservative management.

Compared to pancreatitis associated with hepatitis B, C, or E viruses, which may lead to necrotizing pancreatitis or multi-organ failure, HAV-related pancreatitis is generally benign and reversible with supportive therapy (Gupte et al., 2014).

Our case highlights the importance of screening for viral causes, especially HAV, in idiopathic pancreatitis—particularly in young, non-alcoholic, non-gallstone patients with deranged liver enzymes and markedly raised LDH.

Learning Points

- Hepatitis A can rarely present with pancreatitis before hepatic symptoms, leading to diagnostic delay.
- Viral serology should be part of the work-up in acute pancreatitis with raised liver enzymes.
- Markedly elevated LDH may indicate combined hepatic and pancreatic injury and warrants close monitoring.
- The condition usually resolves with supportive management, and early recognition avoids unnecessary invasive testing.

CONCLUSION

Acute pancreatitis is an uncommon but noteworthy extrahepatic manifestation of HAV infection. Recognition of this association is crucial for appropriate diagnosis and management. Clinicians should consider HAV in the differential diagnosis of idiopathic pancreatitis, especially in young patients presenting with concurrent liver enzyme elevation and markedly raised LDH.

Patient Consent

Written informed consent was obtained from the patient for publication of this case report. Efforts have been made to ensure complete anonymity.

Declarations:

Competing Interests: Authors declare that no financial or non-financial interests are directly or indirectly related to the work submitted for publication.

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