



VIRAL HEPATITIS A PRESENTING WITH ACUTE PANCREATITIS AND POLYSEROSITIS IN A OCCASIONAL ALCOHOLIC: A RARE CLINICAL CASE

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ABSTRACT

Acute viral hepatitis A is generally a self-limiting infection predominantly involving the liver. Extrahepatic manifestations such as acute pancreatitis and polyserositis are uncommon and may lead to diagnostic confusion, especially in patients with a background history of occasional alcohol consumption. We report the case of a 28-year-old male software professional with chronic alcoholism who presented with fever, bilious vomiting, abdominal pain, jaundice, ascites, bilateral pleural effusion, and biochemical evidence of pancreatic involvement. Initial diagnosis favored hepatorenal syndrome with sepsis and multiorgan dysfunction syndrome (MODS). Subsequent serological evaluation revealed acute Hepatitis A virus (HAV) infection with associated acute pancreatitis and polyserositis. Conservative management resulted in gradual clinical improvement. This case highlights the importance of considering acute viral hepatitis A as a potential etiology of pancreatitis and serosal involvement in endemic regions.

KEYWORDS

INTRODUCTION

Hepatitis A virus (HAV) infection is an enterically transmitted acute viral illness commonly encountered in developing countries. The disease is usually benign and self-limiting; however, atypical manifestations involving extrahepatic organs have increasingly been reported. Acute pancreatitis associated with HAV infection is rare, while concomitant polyserositis characterized by pleural effusion and ascites is even more uncommon. Early recognition is clinically significant because these manifestations can mimic severe sepsis, hepatobiliary pathology, or alcohol-related pancreatic disease.

We present a rare case of acute HAV infection complicated by acute edematous pancreatitis and polyserositis in a chronic alcoholic patient.

Case Presentation

A 28-year-old male employed in the IT sector in Bengaluru, presented with complaints of fever, bilious vomiting, and diffuse abdominal pain for 10 days. The patient was initially treated at local hospital, later referred here for further management.

At presentation, the patient was clinically ill with abdominal tenderness and evidence of systemic inflammatory response. Initial working diagnosis sepsis with multiorgan dysfunction syndrome (MODS), and bilateral pleural effusion, predominantly on the right side.

Initial Management

The patient received supportive treatment including:

- Intravenous fluids (5% dextrose), PPI, Anti emetics, and other supportive medications

Investigations Laboratory Findings Investigations revealed:

- Elevated total, direct, and indirect bilirubin levels
- On Admission Total bilirubin levels are. U.I, Direct bilirubin 5.U, Indirect bilirubin 3.2
- On Admission SGPT levels are G75, SGOT IU2
- Elevated serum amylase and lipase
- Elevated C-reactive protein (CRP): 2G.48
- White blood cell count within normal limits
- Anti-HAV IgM: Positive
- Anti-HEV IgM: Negative
- Scrub typhus IgM: Negative
- Malaria- negative
- Leptospirosis- negative

These findings suggested acute viral hepatitis A with pancreatic involvement.

Radiological Findings

Contrast Enhanced Computed Tomography (CECT) Abdomen and Pelvis

CECT Revealed:

- Mild hepatomegaly with fatty liver changes
- Bulky pancreas with patchy enhancement, suggestive of acute edematous pancreatitis
- Moderate ascites
- Minimal splenomegaly
- Moderate bilateral pleural effusion (right greater than left)
- Bilateral lower lobe basal consolidation

The radiological impression favored acute edematous pancreatitis with associated hepatic steatosis and serosal fluid accumulation.

Magnetic Resonance Cholangiopancreatography (MRCP)

MRCP Demonstrated:

- Hepatosplenomegaly
- Dilated portal vein (17 mm) and splenic vein (12 mm)
- Contracted gallbladder with pericholecystic edema
- Bulky pancreatic head without focal mass lesion
- Bilateral pleural effusion and ascites

The imaging findings were suggestive of focal pancreatitis in the setting of acute viral hepatitis.

Final Diagnosis

Based on clinical, serological, biochemical, and radiological correlation, the patient was diagnosed with:

1. Acute viral hepatitis A
2. Acute edematous pancreatitis secondary to HAV infection
3. Polyserositis with bilateral pleural effusion and ascites

Clinical Course

Following confirmation of HAV infection, the patient was managed conservatively with IV fluids and other supportive medications.

Broad-spectrum antibiotics were subsequently de-escalated after exclusion of active bacterial infection.

The patient demonstrated gradual symptomatic improvement with reduction in abdominal pain, vomiting, and systemic symptoms. Liver function and pancreatic enzyme levels improved progressively with supportive care.

DISCUSSION

HAV infection is a common cause of acute viral hepatitis in developing nations; however, pancreatic involvement is rare. The proposed mechanisms of pancreatitis in HAV infection include:

- Direct viral invasion of pancreatic acinar cells
- Edema of the ampulla of Vater causing pancreatic duct obstruction
- Immune-mediated inflammatory injury

Polyserositis in HAV infection is similarly uncommon and may occur secondary to hypoalbuminemia, portal hypertension, systemic inflammation, or transient immune-mediated serosal involvement.

In this patient, chronic alcohol consumption initially complicated the diagnostic evaluation because alcoholic pancreatitis was strongly considered. However, positive HAV IgM serology, absence of chronic pancreatic calcification, and imaging findings consistent with edematous pancreatitis supported HAV-associated pancreatitis.

The presence of pleural effusion, ascites, and hepatosplenomegaly further emphasized the systemic inflammatory nature of the disease process. Conservative management remains the cornerstone of therapy, with most patients showing favorable outcomes.

This case underscores the need for clinicians to consider acute viral hepatitis in patients presenting with pancreatitis and systemic serosal manifestations, especially in endemic regions.

CONCLUSION

Acute pancreatitis and polyserositis are rare but important extrahepatic manifestations of Hepatitis A virus infection. In patients presenting with abdominal pain, jaundice, elevated pancreatic enzymes, pleural effusion, and ascites, HAV infection should be considered in the differential diagnosis, even in the presence of confounding factors such as chronic alcoholism. Early diagnosis and supportive management can lead to successful clinical recovery and avoidance of unnecessary invasive interventions.

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Clinical Significance

- Acute pancreatitis should be recognized as a rare extrahepatic complication of Hepatitis A virus infection.
- Polyserositis presenting as bilateral pleural effusion and ascites may accompany acute HAV infection and mimic severe sepsis or hepatobiliary disease.
- Alcohol consumption should not preclude evaluation for viral hepatitis in patients presenting with pancreatitis and jaundice.
- Early HAV serology and appropriate imaging facilitate prompt diagnosis and prevent unnecessary invasive investigations or prolonged antibiotic therapy.
- Conservative management is usually sufficient and is associated with an excellent prognosis.

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