CASE REPORT

Acute hepatitis E complicated by acute pancreatitis and multiorgan dysfunction

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SUMMARY

We report this rare case of a 27-year-old man who presented with acute hepatitis E and went on to develop acute epigastric pain. He was diagnosed to have acute severe pancreatitis with shock and acute renal failure due to hepatitis E. Such a phenomenon has rarely been reported in the literature, with patients following a benign course and complete recovery after conservative management and analgesia. Awareness of this potentially life-threatening complication, especially in young men from endemic areas with acute hepatitis E presenting with abdomen pain has been highlighted.

BACKGROUND

In the developing world epidemics of hepatitis E virus (HEV) are witnessed periodically, especially in countries like India, China and Somalia. The spectrum of infection ranges from asymptomatic disease to fulminant hepatic failure with 0.2–4% mortality, reaching 10–20% in pregnant women.[1] While acute pancreatitis is a recognised complication of non-fulminant hepatitis A[2] (HAV) and hepatitis B (HBV),[3] association with HEV is rare. We report a rare case of severe acute pancreatitis with shock and acute renal failure following acute HEV infection.

CASE PRESENTATION

We report a case of a 27-year-old Indian male agriculturist who presented with a 20 day history of moderate grade, intermittent fever without chills. One week later he noticed jaundice and had symptoms of itching, nausea and fatigue. He denied a history of consumption of alcohol, substance abuse, any medications, abdominal trauma, gall stones, hypercalcaemia or family history of pancreatitis. There was no abdominal pain or decreased urine output. He was afebrile at presentation to us. Clinical examination revealed an icterus and a tender, mildly enlarged palpable liver.

Blood investigations revealed gross conjugated hyperbilirubinaemia (total bilirubin: 23.3 mg/dL, direct bilirubin: 16.6 mg/dL). The rest of the liver function test revealed the following: aspartate serum transaminase (AST) of 125 IU/L, alanine serum transaminase (ALT) of 80 IU/L, alkaline phosphatase (ALP) of 80 IU/L, total protein of 7 g/dL and serum albumin of 4.3 g/dL. Ultrasonography of the abdomen showed mild hepatosplenomegaly, with no obstruction in the biliary duct and normal pancreatic architecture. Anti-HEV IgM was positive for HEV (titres 1 : 10 000). Anti-HEV IgG and PCR for HEV were positive. Genotyping was not performed due to its non-availability in our laboratory. Tests for HAV (anti-HAV IgM), HBV (antibodies to surface antigen: HBsAg and antibodies to core antigen: anti-HBc IgM) and hepatitis C (anti-HCV) were negative. HIV (ELISA test), malaria (immunochromatography and quantitative Buffy Coat testing), leptospirosis, enteric fever, scrub typhus (IgM ELISA), dengue (IgM by ELISA) cytomegalovirus CMV (by PCR) and Epstein-Barr virus (EBV) (Paul-Bunnel test and ELISA for antibodies against EBV) were ruled out. Following symptomatic treatment, the patient was on a recovering trend with clinical and biochemical improvement. On the 6th day he developed acute epigastric abdominal pain, nausea, vomiting and hypotension (blood pressure: 80/60 mm Hg). The patient was anuric.

INVESTIGATIONS

Serum amylase was 5214 IU/L and lipase was 11207 IU/L. Total leucocyte count was elevated (TC: 31 600/mm^3) and renal parameters were deranged (blood urea nitrogen: 131 mg/dL). Total leucocyte count was elevated (TC: 31 600/mm^3) and renal parameters were deranged (blood urea nitrogen: 131 mg/dL; Creatinine: 5.5 mg/dL; potassium: 6 meq/L). An arterial blood gas analysis showed severe metabolic acidosis (pH 7.06; bicarbonate: 3 meq/L). His lipid profile was normal.

DIFFERENTIAL DIAGNOSIS

Acute severe pancreatitis with shock and acute renal failure due to HEV infection.

TREATMENT

The patient received renal replacement therapy, inotropic support (intravenous dopamine), intravenous antibiotic (meropenem after renal dose modification) and analgesia. After 2 weeks of renal replacement therapy there was gradual improvement in renal parameters.

OUTCOME AND FOLLOW-UP

At discharge there was normalisation of amylase levels and reduction of bilirubin (total bilirubin: 5.2 mg/dL). Investigations at a 6 week follow-up were normal. Eendoscopic retrograde cholangiopancreatography showed no evidence of pancreatic divisum or chronic pancreatitis.

DISCUSSION

Acute pancreatitis is most often associated with alcohol, gall stones, trauma, hyperlipidaemia and ulcer disease. Viruses namely rubella, CMV, EBV and varicella zoster virus have been implicated in pancreatitis, with mumps being the commonest.[4]

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Fulminant viral hepatitis has long been linked to acute pancreatitis. In these patients, the associated pancreatitis was most often not severe and mortality depended on the severity of hepatitis rather than pancreatitis.[5] In recent years, however, there are increasing reports of its association with non-fulminant HCA and HCB.[2 3] The mechanism is postulated to be due to either an immune response or direct cytotoxicity against the infected acinar cells.[6] Oedema of the ampulla of vater with obstruction of pancreatic fluid flow[7] and release of lysosomal enzymes from the damaged acinar cells into the circulation[8] are other possible mechanisms of pancreatic damage.

Only 14 cases of HEV causing acute pancreatitis have been reported in the literature.[9–12] The majority of these cases developed pancreatitis during the second to third weeks of hepatitis with complete resolution of illness with conservative management. Our case differs from previous reports. The patient developed pancreatitis in the fourth week of hepatitis. As opposed to the benign course of the remaining cases he suffered from severe pancreatitis leading to shock, requiring inotropic support. In addition, he developed acute renal failure and severe metabolic acidosis requiring haemodialysis for 2 weeks. One such case has been reported by Maity *et al*[13] of an 18-year-old boy where pancreatitis was complicated by sepsis and acute renal failure. The boy recovered over a period of 5 weeks and underwent only three sessions of haemodialysis. Fatalities following pancreatitis due to HEV have not been reported in immunocompetent adults. Sinha *et al* described fatal pancreatitis following HEV in a renal transplant recipient which had a multifactorial causation including the immunosuppressive agents the patient received, rather than hepatitis alone.[14] The majority of cases were young men, which is in accordance with the pattern of HEV epidemics, clinically affecting men two to five times more than women,[15] HEV genotype 1a being the main infective strain in India. Risk factors for infection in our patient were male gender, his agriculturist profession making him prone to exposure, and an epidemic of HEV in his area at the time. In conclusion, we report the second case in the literature of severe life-threatening pancreatitis complicated by multiorgan dysfunction in an immunocompetent young man. In cases of acute HEV, especially in young males presenting with abdominal pain, the diagnosis of acute pancreatitis should be considered. The patient should be closely monitored for life-threatening complications.

Learning points

- Acute hepatitis E causing acute life-threatening pancreatitis with multiorgan dysfunction especially in an immunocompetent person is extremely rare.
- Especially in young men with acute hepatitis E presenting with abdominal pain, the diagnosis of acute pancreatitis should be considered.
- Awareness of such complications allow physicians to closely monitor patients with acute hepatitis E for potential life-threatening complications and prompt treatment.

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Competing interests None.

Patient consent Obtained.

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