Acute pancreatitis complicating hepatitis A virus infection: A case report

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ABSTRACT

Acute pancreatitis is inflammation of pancreas associated with injury to the pancreatic exocrine parenchyma. The most common causes of acute pancreatitis are obstruction of the pancreatic duct (gall stones) and alcohol. Viral infections are also well recognized etiology for pancreatitis. Here, we report a case of 16-year-old girl with acute pancreatitis caused by hepatitis A virus. The identification of this unusual complication of hepatitis A is important. Acute pancreatitis complicating fulminant viral hepatitis has been well known; however, acute pancreatitis occurring in nonfulminant hepatitis is very rare.

Key words: *Acute pancreatitis, Hepatitis A, Viral infection*

Acute pancreatitis is a life-threatening illness characterized by acute inflammation of exocrine part of the pancreas in association with systemic inflammatory response. The most common causes for pancreatitis in adults are cholelithiasis and excessive alcohol use, accounting for 35-40% and 30% of cases, respectively. Other causes include anatomic variants of the pancreas, mechanical obstruction to pancreatic juice, hypertriglyceridemia, hypercalcemia, drug induced, toxins, trauma, ischemia, autoimmune conditions, and infections [1].

Several viral infections have been implicated as etiological factors of acute pancreatitis. The viruses most frequently thought to be responsible are mumps and coxsackie, and others include cytomegalovirus, Epstein-Barr virus, and rarely hepatitis A, B, C and E [2]. However, the association of pancreatitis with viral hepatitis is not a common clinical entity. Acute pancreatitis is not uncommon in fulminant hepatic failure and has been confirmed based on histology and serology. However, acute pancreatitis has been reported very rarely in acute (non-fulminant) viral hepatitis [3,4]. It has recently been observed that pancreatic involvement may also be present in milder forms of viral hepatitis. There are only a few case reports from India of acute pancreatitis complicating mild-to-moderate hepatitis E and hepatitis A virus infection.

CASE REPORT

A 16-year-old girl presented to our hospital with complaints of nausea and vomiting with decreased appetite since one 10 days, pain abdomen for 8 days and jaundice and dark colored urine for 4 days. There was no history of fever, loose stools, or constipation. There was no history of alcohol use, gallstone disease, chronic drug intake, or trauma. There were no similar complaints in the past, and she was not a known case of any other chronic illness.

On general physical examination, the patient was moderately built, conscious and oriented to time place and person. Vitals were within the normal limits. She was not pale but icterus was present, and there was no cyanosis, clubbing, pedal edema, or lymphadenopathy. Jugular venous pressure was not raised. On abdominal examination, there was mild distension of abdomen; tenderness was present in epigastrium and right hypochondrium with mild hepatomegaly of 2 cm below right costal margin. There was no clinically appreciable free fluid and bowel sounds were absent. Clinical examination of respiratory, cardiovascular and nervous system was unremarkable.

Laboratory investigations revealed hemoglobin of 11 g%, total leukocyte count of 7500 differential leukocyte count and platelets were normal. Erythrocyte sedimentation rate 28 mm/h, serum bilirubin: 6.1 mg/dl (normal 0.2-1.2) (conjugated 5.0 mg/dl); aspartate aminotransferase (AST) - 1808 U/L (normal 20-40); alanine aminotransferase (ALT) - 1364 U/L (normal 20-40); alkaline phosphatase: 236 U/L (normal 38-85). Serum protein was 6.4 with A:G ratio 1.1, serum amylase 5872 U/dl; serum lipase: 10,677 U/dl. Renal function tests, serum electrolytes, serum calcium; phosphorus, and serum lipids were normal. HIV, hepatitis B surface antigen, anti-hepatitis C virus antibody tests were non-reactive. Immunoglobulin M (IgM) for anti-hepatitis A virus was positive and IgM for anti-HEV was negative. Chest X-ray and electrocardiogram did not reveal any abnormality. Ultrasound examination of the abdomen revealed a bulky pancreas with hepatomegaly with no evidence of any cholelithiasis with non-dilated common bile duct with rest of the abdomen normal, and there was no free fluid. Contrast-enhanced computed tomography abdomen was done and it showed bulky pancreas however with normal enhancement, and there is peripancreatic fluid collection. Mild fluid collection was seen in the peritoneal cavity (Fig. 1). Endoscopic retrograde cholangiopancreatography was planned

in view of raised serum bilirubin in initial presentation but it was denied because of financial constraints of the patient, and there was another possible etiology, i.e., hepatitis A infection which can also causes to cholangitis. Pancreatic cancer markers were not done as our patient was young, and there was no positive family history.

The patient was treated with fluid, electrolyte replacement, and other supportive care. She was improved gradually over 2 weeks, relieved of her symptoms and recovered without any complications. On discharge her, serum bilirubin was 1.2 mg/dl, AST was 95 U/l, ALT - 58 U/I, and alkaline phosphatase: 119 U/L. The patient reported to outpatient department after 2 weeks of discharge with normal liver function tests and ultrasound showing normal pancreas and liver.

DISCUSSION

Alcohol abuse and biliary disease are thought to be the main etiological factors in the development of pancreatitis, idiopathic acute, and chronic pancreatitis constitutes a high percentage of the total number of cases. Evidence supporting this concept of a viral cause derives from serological studies, case reports, and animal studies.

The association of pancreatitis with viral hepatitis, though known, is not a common clinical entity. What leads to pancreatic injury in viral hepatitis is still a debatable question. The cytopathic effect may be direct or it may be mediated through the patient's immune response. It has also been suggested that viral infections cause edema of Vater's ampulla and pancreatic ducts leading to pancreatitis as result of obstruction to the flow of pancreatic fluid [5]. The hepatitis viruses might injure the pancreatic acinar cell membrane, resulting in the leakage of intracellular enzymes, and/or precipitate a network of intracellular events culminating in cell death by a mechanism analogous to hepatocyte necrosis [6]. Another mechanism for acute pancreatitis associated with viral etiology is direct inflammation and destruction of the pancreatic acinar cell by the virus; this hypothesis is supported by the autopsy finding of hepatitis B virus antigens within the cytoplasm of pancreatic acinar cells of patients with hepatitis B surface antigenemia [7,8]. It is showed that the severity of pancreatitis is related to the magnitude of exposure of pancreatic acinar cells to the hepatitis virus [9].

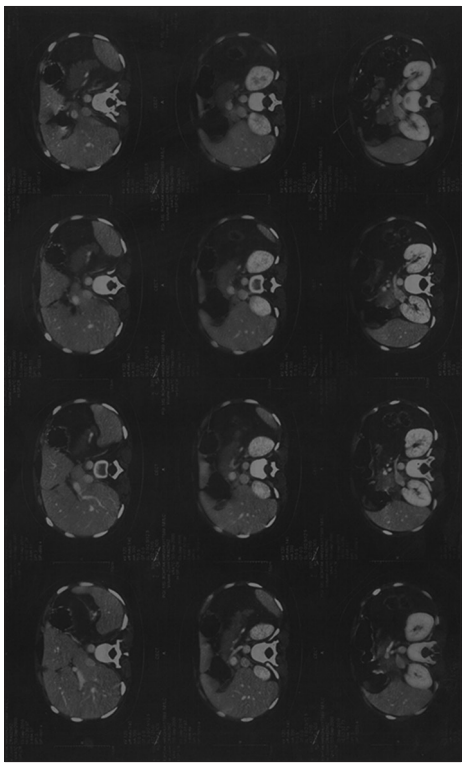

Figure 1: CECT Abdomen showing bulky pancreas with normal enhancement, and peripancreatic fluid collection

From the whole spectrum of hepatitis viruses, HBV is the infectious agent most implicated in acute pancreatitis in most cases pancreatitis associated with benign forms of acute viral hepatitis resolve with little morbidity in conjunction with the recovery from hepatitis. However, chronic relapsing forms of pancreatitis in association with viral hepatitis have been hypothesized and patient needs proper follow-up. Severe abdominal pain in the patients with early course of acute hepatitis should alert the clinician for the possibility of complications including acute pancreatitis and investigate in an appropriate way. However, the prognosis of patients with acute pancreatitis in the setting of acute viral hepatitis is usually good, and most patients recover with conservative treatment [8].

CONCLUSION

Although a rare etiology, viral agents including hepatitis A need to be considered as a causative agent for acute pancreatitis, especially when another common cause of the disease (mechanical, toxic, metabolic, or traumatic) is evident.

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