CASE REPORT - AN INTERESTING CASE OF ENTERIC FEVER MASQUERADING AS ACUTE PANCREATITIS

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ABSTRACT
Enteric fever is a common infectious disease worldwide, especially in developing countries like India. Patients with enteric fever may present with pancreatitis and the presence of pancreatitis as a complication of enteric fever increases the mortality rate in untreated enteric fever due to salmonella typhi. We report a case of a 35-year-old patient in this case report who presented with fever and abdominal pain. Enteric fever presenting as pancreatitis is a rare occurrence and has to be considered in patients in endemic areas, presenting with abdominal pain and concomitant fever.

Key Words: Salmonella Typhi, Pancreatitis, Typhoid, Enteric Fever

INTRODUCTION
Enteric fever remains a serious health threat in developing countries including India (Dutta et al., 2006). Initial presentation as pancreatitis is a rare manifestation of enteric fever (Parmar et al., 2000). The gastrointestinal complications of enteric fever are intestinal haemorrhage and perforation, acute cholecystitis, acute pancreatitis, hepatic abscess, splenic rupture & typhoid hepatitis (Bhan et al., 2005). There have been few case reports (Kune et al., 1972; Kadappu et al., 2002) that have identified Salmonella as a causative agent of acute pancreatitis. Furthermore, two retrospective studies (Renner et al., 1991; Hermans et al., 1991) have reported a frequency of hyperamylasemia of 50% and a frequency of clinical pancreatitis ranging from 28 to 62% in patients with Salmonella infection.

CASES
This 35 year old male patient got admitted with the chief complaints of fever and yellowish discoloration of eye/urine of 20 days duration. He had fever which was high grade, intermittent and associated with chills and rigor patient also had jaundice, abdominal pain, loose stools and vomiting. He also developed breathlessness for the past 2 days and was admitted to our hospital. There was no history of cough, chest pain, palpitation, sweating, giddiness, decreased urine output, swelling of legs and no bleeding manifestations. The patient was not an alcoholic.

On examination, patient was conscious, oriented, dehydrated and febrile. He also had pallor, icterus and was tachypneic with a respiratory rate of 40/min and his saturation in room air was 87% and he had minimal respiratory distress. Examination of the respiratory system revealed decreased breath sounds and crepitations over both the bases of the lungs. Examination of the abdomen revealed generalised, diffuse tenderness and bowel sounds were sluggish. Examination of the other systems did not reveal any abnormalities.

Investigations revealed leucopenia, thrombocytopenia, normocytic normochromic anemia and an elevated ESR. Peripheral smear showed a left myeloid shift and pancytopenia. Chest X-ray showed bilateral pleural effusion (right > left). ABG showed evidence of type 1 respiratory failure and respiratory alkalosis. Serum electrolytes revealed hyponatremia and hypocalcemia. Liver function tests showed direct hyperbilirubinemia, elevated liver enzymes and hypoalbuminemia. Serum amylase and lipase were significantly elevated. Serum LDH levels were found to be elevated. Serum creatinine was mildly elevated. Urine routine showed proteinuria and hematuria. USG abdomen revealed mild hepato-splenomegaly/ascites, edematous/thickened gall bladder wall (possibly reactive) and
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a heterogenous head and body of pancreas with the possibility of pancreatitis. Urine culture, stool routine and culture were normal. Blood culture grew salmonella typhi which was resistant to nalidixic acid.

At the time of admission, a provisional diagnosis of viral syndrome with muti-organ dysfunction was made, the patient continued to have abdominal pain and greenish coloured loose stools. Imaging showed evidence of pancreatitis and serum amylase & lipase were significantly elevated and a diagnosis of pancreatitis was made. Patient also had evidence of acute hepatitis, hypoalbuminemia, pancytopenia and ARDS. Patient’s blood culture grew salmonella typhi and hence a final diagnosis of enteric fever was made. The patient was started on ceftriaxone (3rd generation cephalosporin) for 2 weeks based on the sensitivity pattern. Then the patient’s general condition improved and he was discharged in a well condition.

DISCUSSION

Forty percent of patients with enteric fever present with abdominal pain and the causes for abdominal pain in a patient with enteric fever are intestinal haemorrhage and perforation, acute cholecystitis, typhoid hepatitis, hepatic abscess, splenic rupture and acute pancreatitis (Bhan et al., 2005). Enteric fever masquerading as acute pancreatitis is a rare entity. Localised salmonella infection of the pancreas is usually the result of salmonella bacteraemia caused by S. choleraesuis but may also occur after gastroenteritis by S. typhimurium and enteric fever by S. typhi (Cohen et al., 1987). The abnormalities of pancreas that are associated with typhoid fever may include simple biochemical abnormalities such as hyperamylasemia, gallbladder involvement (Cohen et al., 1987) to acute pancreatitis, pancreatic abscess(Kune et al., 1972), chronic pancreatitis pancreatic pseudocyst (Koshi et al., 1976) requiring surgery.

Mechanism of pancreatitis in Typhoid fever is not exactly known but various explanations have been postulated. The mechanisms are direct pancreatic localisation of bacteria, toxin induced or immune mediated pancreatitis (Arendt et al., 1993 and Schmid et al., 1999). Theoretically this could occur by hematogenous or lymphatic or transmural migration via the biliary duct system or form the duodenum via the main pancreatic duct. This is considered further in view of the increased incidence in patients with cholelithiasis, choledocholithiasis and biliary duct anomalies. This patient had a BISAP score > 3 and a Ranson’s score (Ranson et al., 1974) which was also suggestive of acute pancreatitis. Treatment led to resolution of this patient’s symptoms and the patient was discharged in a well condition. Though the patient was asymptomatic at the time of discharge, both serum lipase & amylase continued to be elevated and normalised only after a period of two months at the time of review.

Sensory neuronal hearing loss which was also documented in this patient has also been reported in patients with enteric fever (Baek et al., 1997). The other important infectious agents known to cause sensorineural hearing loss are Chickenpox and smallpox. The incidence is reported to be about 14% with typhoid (Kapur et al., 1965). It usually resolves with treatment of typhoid.

This case highlights the importance of including enteric fever in the differential diagnosis of acute pancreatitis as appropriate diagnosis and treatment might lead onto resolution of the disease.

REFERENCES


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