A Rare Complication of Enteric Fever - Case Report

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Case Report

24 yr old non diabetic non hypertensive female Staff Nurse in Delhi presented to our OPD with complaints of fever with rigor and chills of 2 weeks duration. Fever was high grade, intermittent associated with chills and rigor. She also had loose stools and vomiting but no passage of blood in stools. There was no history of cough, chest pain, dyspnoea, palpitation, dysuria, decreased urine output, abdominal pain, jaundice, seizures, swelling of legs or any bleeding manifestations.

She was prescribed Cefixime for 5 days from Delhi Hospital, but stopped after 2 days. Fever subsided but recurred after an asymptomatic interval of 5 days. Fever was high grade with 5-6 episodes of loose stools and 3-4 episodes of vomiting. She had headache and generalised myalgia also. No hematuria, dysuria or high coloured urine.

On examination, she was conscious, alert and oriented; dehydration was present. Febrile with axillary temperature 103°F. Mild pallor no icterus. PR 98/mt BP 100/70 mmHg with no postural fall. Diffuse abdominal tenderness with mild Hepatosplenomegaly. No obliteration of liver dullness. Other systems Normal. Provisionally diagnosed as enteric fever.

Investigations WBC count: 4800/cumm, Platelet count: 4.5 Lakhs/cumm, haemoglobin: 12.1 g/dL and an ESR of 55 mm. Serum electrolytes revealed Na+: 128 meq/L, K+: 3.8 meq/L. Liver function tests Bilirubin -0.9 mg%, AST: 92 U/L; ALT: 124 U/L; ALP: 325 U/L; Normal protein levels. RFT Normal. Peripheral smear was negative for Malarial Parasite. Platelets Normal. Serum CPK was normal. Serum Calcium –

*See End Note for complete author details
7.8 and S. Triglyceride was 265. Urine analysis normal.

Chest X-ray and ECG was Normal. Peripheral Smear showed Normocytic Normochromic blood picture with no immature cells or Malaria Parasite. Widal test done on the second day of fever from Delhi was negative.

Widal – repeated here showed TO 100   TH 200 AH < 50 BH<50

Widal Clot Culture — S.Typhi was isolated in Clot culture. Blood Culture isolated S.Typhi sensitive to Ampicillin/Chloramphenicol/Ceftriaxone and Sepran and Moderately Sensitive to Ciprofloxacin.

Started on antipyretics , anti emetics ,IV fluids. After repeat Widal, patient was given Inj. Ceftriaxone 2 gm IV OD.

On the fourth day of Ceftriaxone patient developed Severe epigastric pain radiating to back with associated vomiting. Epigastric tenderness elicited with sluggish bowel sounds. Suspecting perforation Abdomen X Ray abdomen erect posture was ordered but there was no gas under diaphragm.

USG abdomen No evidence of Peritonitis.

Gastroenterology consultation done. Possibility of Acute Pancreatitis. Serum amylase and lipase were significantly elevated:

S.amylase-485 U/L (normal 30-120 U/L); lipase-3578 U/L (normal 10-140 U/L). Serum lactate dehydrogenase (LDH) levels was also elevated: 556 U/L (normal 105-220 U/L).

Final diagnosis Enteric fever complicated by acute pancreatitis .Patient maintained on Ryles Tube aspiration for 4 days with IV Fluids and Oectreotide infusion for 48 hours along with Ceftriaxone which was continued for 7 days. Patient became afebrile. Abdominal pain subsided. Serum amylase and lipase levels lowered and was discharged on 17th day of admission.

**DISCUSSION**

Patients with enteric fever may 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. Enteric fever masquerading as acute pancreatitis is a rare entity. The abnormalities of pancreas that are associated with typhoid fever may include simple biochemical abnormalities such as hyperamylasemia to acute pancreatitis, chronic pancreatitis, pancreatic pseudo cyst requiring surgery.

Retrospective studies have reported a frequency of hyperamylasemia of 50% and a frequency of clinical pancreatitis ranging from 28 to 62% in patients with Salmonella infection. Two cases of acute pancreatitis by Salmonella typhi were reported.

Localized Salmonella infection of the pancreas is usually the result of Salmonella bacteraemia caused by Salmonella cholerae but may also occur after gastroenteritis by S. typhimurium and enteric fever by Salmonella typhi . Once pancreatitis develops, it may form a pancreatic abscess. Pancreatic pseudo cyst may occasionally may be infected by Salmonella typhi. Salmonella typhi is known to localize in injured or damaged tissue or in sites of malignancy.

Mechanism of pancreatitis in enteric fever is not exactly known. Typhoid pancreatitis could represent the effect of the direct pancreatic localisation of bacteria which could happen by haematogenous route, lymphatic route and transmural migration, via the biliary duct system and from the duodenum via the main pancreatic duct. This may occur, especially in patients with predisposing conditions like biliary stasis, cholelithiasis, choledocho -lithiasis, and biliary duct abnormalities, but our patient had no such predisposing conditions. The mechanisms may also be toxin induced or immune mediated pancreatitis. Isolated hyperamylasemia and hyperlipasemia without evidence of pancreatic involvement could also be the result of a reduced excretion due to either impaired renal or liver function which is common in Salmonella infections.

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**CONCLUSION**

This case highlights the importance of keeping in mind the possibility of development of acute pancreatitis in a case of enteric fever as appropriate diagnosis and treatment might lead to complete resolution of the potentially fatal condition.
END NOTE

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Conflict of Interest: None declared

Editorial Comments: Enteric fever is a common problem in the medical wards. This paper reports an uncommon complication which can occur during the course of this common disease. This uncommon association needs to be thought of in certain situations.

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