Pancreatic Ascites: Rare Complication of a Common Disease

Sir,

Abdominal tuberculosis is known for its diverse clinical manifestations, usually affects the iliocecal region, abdominal lymph nodes and peritoneum. The pancreas is affected in 2-4.7% of cases of miliary tuberculosis. Pancreatic tuberculosis can present with abdominal pain, fever, non-specific symptoms with weight loss, obstructive jaundice and even variceal hemorrhage due to splenic vein thrombosis. We hereby report a non-alcoholic young female who was initially treated for abdominal tuberculosis, developed an uncommon complication; chronic pancreatitis resulting into pancreatic ascites. She was treated successfully, but unfortunately developed delayed complications in the form of diabetes mellitus and malabsorption.

A 28 years old female had been treated for abdominal tuberculosis, 3 years back. After a month of completion of ATT, she again developed progressive distension of abdomen. On examination she had massive ascites and splenomegaly. Routine investigations including liver function test was normal except total serum protein 5.8 g/dl (albumin 3.0 g/dl). HBsAg, anti-HCV were negative and ELISA for HIV was non-reactive. Serum amylase was 120 U/l. Ascitic fluid examination revealed total protein of 3.6 g/dL (albumin 2.4 g/dL), sugar 86 mg/dl, total leucocyte count was 1400 cells/mm³ (polymorphs 18%, lymphocytes 82%) and cytology showed predominantly lymphocytes in a proteinaceous background. Gram and acid fast bacilli stains were negative, culture was sterile and ascitic fluid was also negative for PCR for Mycobacterium tuberculosis. Ascitic fluid amylase was 32000 U/l and serum ascites albumin gradient (SAAG) was 0.6.

On imaging studies, ultrasound abdomen showed gross ascites, multiple right collaterals, normal liver with portal vein diameter 12 mm, splenic vein diameter 9 mm, bulky head of pancreas, main pancreatic duct was not visualized and splenomegaly. CECT abdomen revealed hepatomegaly, normal portal vein diameter, CBD was dilated at porta and in pancreatic head, pancreas was enlarged, irregular in outline had dilatation of main pancreatic duct and a pseudocyst was visualized anterior to the tail in lesser sac. Multiple enlarged rim enhancing lymph nodes in the mesentery and porta, marked ascites and splenomegaly were also seen. ERCP showed CBD stricture at lower end. Impression of chronic pancreatitis with pseudopancreatic cyst with pancreatic ascites with CBD stricture at lower end was made and she was treated with Roux-en-Y-jejunal loop and cystojejunostomy. Lymph node, pancreatic tissue and liver biopsy reports revealed caseating tubercular lymph node, chronic pancreatitis and mild portal and lobular inflammation respectively. Considering the biopsy report, she was also treated with category-II ATT. Patient was asymptomatic for about two years following surgery till she developed classical symptoms of diabetes for which she was started on insulin. She took insulin for 8-9 months, stopped on her own as her blood glucose levels were in normal range. Subsequently, she also developed symptoms of malabsorption and 24 hr fecal fat was 6.8 gm/24 hrs.

On present admission, she had complaints of dizziness and generalized weakness for 1 month, and 2-3 episodes of vomiting for 2 days. Examination revealed poor general condition, BP 94/70 mmHg, PR 92/min. On CNS examination she was conscious, oriented, drowsy and rest of the systemic examinations were normal. Her RBS was 624 mg/dL and urine ketones were in traces. Other routine investigations were normal. ABG analysis showed metabolic acidosis. She was treated on the line of diabetic ketoacidosis to which she showed dramatic response.

Pancreatic ascites is an uncommon clinical condition, mainly affects the alcoholic male in second to fifth decade of life. It usually develops as part of severe acute pancreatitis or as a result of pancreatic duct rupture or leakage from a pseudocyst as a complication of chronic pancreatitis. Although the exact incidence of pancreatic ascites is not known, it seems to be infrequent. It has been described in 3.5% patients with chronic pancreatitis and 6-14% patients with pseudocyst. It is characterized by a very high amylase concentration in ascitic fluid (usually over 1000 U/l) and protein concentration over 3 g/dL. Pancreatic ascites frequently is neutrocytic and also may be complicated by bacterial infection. Clinical presentation includes weight loss despite progressive increase of abdominal girth and abdominal pain or discomfort.

There are two modality for treatment of pancreatic ascites – medical and surgical. Treatment may require nasogastric suction and parenteral alimentation to decrease pancreatic secretions. In addition paracentesis may be necessary to keep the peritoneal cavity free of fluid and, it is hoped, to effect sealing of the leak. The long acting somatostatin analogue octreotide, which inhibit pancreatic secretion, is useful in cases of pancreatic ascites. If ascites continues to recur after 2-3 weeks of medical management, the patient should be operated on after pancreatography to define the anatomy of the abnormal duct.

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Fig. 1: Showing bilateral breast hematoma
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