DISSEMINATED TUBERCULOSIS MANIFESTING AS CHRONIC PANCREATITIS

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Summary: Pancreatic involvement in tuberculosis is known but uncommon. The clinical manifestation may vary from painless obstructive jaundice due to pancreatic mass (cyst or abscess) to fever of unknown origin. Here we report a case who initially presented as acute pancreatitis relapsing into chronic pancreatitis as an initial manifestation of disseminated tuberculosis.

Key words: Disseminated Tuberculosis, Pancreatic Tuberculosis, Acute and Chronic Pancreatitis

INTRODUCTION

Disseminated tuberculosis refers to concurrent involvement of at least two non-contiguous organ sites of the body, or involvement of the blood or bone marrow by tuberculosis process\textsuperscript{1}. Tuberculosis is an extremely common disease in developing countries, though its incidence is on the rise in the Western world too! Pancreatic tuberculosis is a rare manifestation of such a common disease possibly due to protective pancreatic enzymes. We discuss below a patient who presented as acute pancreatitis, relapsing into chronic pancreatitis as an initial manifestation of disseminated tuberculosis.

CASE REPORT

A 23-year-old non-alcoholic female was admitted previously with history of episodic abdominal pain, fever, vomiting, along with tenderness in the epigastrium three months, prior to present admission. She was managed conservatively with provisional diagnosis of acute pancreatitis based upon markedly elevated serum amylase and lipase along with imaging evidence of pancreatitis. Ultrasound abdomen at that time had revealed ill-defined bulky and hypoechoic pancreas suggestive of pancreatitis with multiple space occupying lesions at porta suggestive of lymphadenopathy and splenomegaly. Contrast enhanced computed tomography (CECT) abdomen had shown focal pancreatitis along with retroperitoneal and mesenteric lymphadenopathy and splenomegaly. Patient was relieved for a short time and was re-admitted during present episode with history of intermittent low grade fever and abdominal pain radiating to back along with distension of abdomen of three weeks’ duration. There was no history of jaundice, trauma, surgery, tuberculosis, or treatment with antitubercular drugs in past and patient also denied any history of contact with tuberculosis. On examination, her vitals were normal. There was no icterus, peripheral lymphadenopathy or pedal edema. Abdominal examination revealed tenderness in epigastrum, evidence of ascites and splenomegaly (3 cm below left subcostal margin). Respiratory examination was suggestive of left sided pleural effusion. A clinical possibility of chronic pancreatitis (in view of past evidences) along with disseminated tuberculosis was considered.

Her investigations revealed hemoglobin of 10.2 g/dl, total leucocyte count 6200/mm\textsuperscript{3}, differential leucocyte count P\textsubscript{6}L\textsubscript{32}E\textsubscript{2}M\textsubscript{2}, platelet count 3,26,000/mm\textsuperscript{3} and erythrocyte sedimentation rate (ESR) was 40 mm in 1\textsuperscript{st} hr. Other investigations including serum amylase, serum calcium, lipid profile, liver function tests with serum proteins, renal function test, blood sugar and urine examination were

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within normal limits. HBsAg, anti-HCV were negative and ELISA for HIV 1 and 2 was non-reactive. Mantoux test was positive (12x20 mm). Chest radiograph showed left sided pleural effusion. Ultrasound abdomen showed hepatosplenomegaly, dilated common bile duct (CBD) with narrowing at lower end, bulky head of pancreas and peripancreatic lymph nodes and ascites. Spiral CT abdomen showed bulky head of pancreas (Fig.) with prominent pancreatic duct with retroperitoneal and mesenteric lymphadenopathy, splenomegaly, left sided pleural effusion, ascites and omental thickening and likely radiological diagnosis of tuberculosis was suggested. CECT chest had revealed left-sided pleural effusion without any evidence of miliary tuberculosis/adenopathy. Magnetic resonance cholangiopancreatography (MRCP) revealed irregular bulky head of pancreas with cystic spaces suggestive of chronic pancreatitis with dilated tortuous main pancreatic duct, dilated CBD with stricture at lower end, mild dilatation of intrahepatic biliary radicals, splenomegaly, left pleural effusion and ascites. Upper GI endoscopy did not show any varices. Her ascitic fluid had total protein of 4 gm%, albumin 2.6 gm% and serum ascites albumin grading (SAAG) was 0.5 gm%. Total leucocyte count was 1800/mm³ with 85% lymphocytes. On Gram stain, no organisms were seen, AFB stain, for mycobacterium tuberculosis was negative and culture was sterile. But polymerase chain reaction (PCR) for M. tuberculosis in ascitic fluid was positive. Pleural fluid was also exudative in nature. Diagnosis of disseminated tuberculosis with chronic pancreatitis was made and she was put on antitubercular therapy (RHZE) along with conservative treatment for chronic pancreatitis as there was no history of jaundice in past or at present episode, no symptoms of mal-absorption, liver function tests and blood sugar were normal. She showed gradual response in the form of marked

Fig.: Spiral CT abdomen showing bulky head of pancreas (arrow)
improvement with reduction in both intensity and frequency of epigastric pain, resolution of fever, ascites and pleural effusion over next three months. Ultrasound abdomen done during her follow-up had also shown gradual reduction in size of pancreatic head and she was doing well on follow-up.

**DISCUSSION**

This patient who had been previously diagnosed as acute pancreatitis presented as chronic pancreatitis along with hepatosplenomegaly, lymphadenopathy and polyserositis of tubercular etiology.

Abdominal tuberculosis affecting gastrointestinal tract, peritoneum, omentum, mesentery and its node and other solid intra-abdominal organs like liver, spleen is a common form of extra-pulmonary tuberculosis occurring in about 11-16%. Tubercular peritonitis constitutes 4-10% of all patients with extra-pulmonary tuberculosis. Abdominal tuberculosis affecting gastrointestinal tract, peritoneum, omentum, mesentery and its node and other solid intra-abdominal organs like liver, spleen is a common form of extra-pulmonary tuberculosis occurring in about 11-16%. Tubercular peritonitis constitutes 4-10% of all patients with extra-pulmonary tuberculosis.

Pancreatic tuberculosis is extremely uncommon and occurs more often in immunocompromised patients and miliary tuberculosis where the disease pattern can be variable. Pancreas is supposed to be biologically protected from infection by *Mycobacterium tuberculosis* because of pancreatic enzymes. Pancreatic involvement is thought to be caused by direct spread from adjacent peripancreatic lymph nodes and also by hematogenous dissemination. Isolated case reports, which are available on presentation of involvement of pancreas in tuberculosis, include upper abdominal pain, pyrexia of unknown origin, obstructive jaundice mimicking pancreatic carcinoma, acute pancreatitis, pancreatic abscess refractory to antibiotics, massive gastrointestinal hemorrhage due to duodenal wall erosion, splenic vein thrombosis and non-specific symptoms with weight loss. Mass lesion in pancreas due to tuberculosis are infrequently described. Additional features of low attenuation peripancreatic and peri-portal adenopathies with peripheral rim enhancement on CT along with fine needle aspiration cytology wherever feasible usually supplement the diagnosis. Though a review of literature has revealed that a majority of cases are diagnosed on laparotomy either for constitutional symptoms along with mass or obstructive jaundicethree.

The advent of PCR has simplified the diagnosis of tuberculosis in situations where fluid or tissue samples can be obtained easily and it obviates the need for laparotomy. The polymerase chain reaction for *Mycobacterium tuberculosis* (TB-PCR) is a rapid and reliable method for the diagnosis of both pulmonary and extra-pulmonary tuberculosis with an overall sensitivity of 78.3% and a specificity of 100%. Because of the relatively low sensitivity of TB-PCR, clinical judgment remains the ultimate decision in the management of tuberculosis.

**REFERENCES**