Case Report:

Pancreatic tuberculosis mimicking malignancy in an immunocompetent individual

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ABSTRACT

Tuberculosis of pancreas in an immunocompetent individual is rare even in endemic regions. We report here one such case where in a 53-year-old man presented with jaundice, weight loss and cervical lymphadenopathy. Base line liver functions were deranged. Imaging studies of abdomen and chest revealed a pancreatic mass and miliary filtrates. Fine needle aspiration cytology of cervical lymph nodes was suggestive of tuberculosis. In view of deranged baseline liver functions the patient was started on a modified daily self-supervised antituberculosis treatment and oral corticosteroid treatment made an uneventful recovery with complete resolution of mass lesion.

Key words: Pancreas, Tuberculosis, Malignancy, Jaundice, Acute kidney injury

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INTRODUCTION

Pancreatic tuberculosis (TB) is extremely rare in immunocompetent individuals, even in those with military TB.¹ A high index of suspicion needs to be exercised especially in endemic areas as diagnosis of pancreatic TB is both elusive and rare.^{2,3} As this condition is often mistaken for malignancy, patients are subjected to avoidable radical surgery. We report the clinical presentation, method of diagnosis and treatment of pancreatic TB in an immunocompetent patient.

CASE REPORT

A 53-year-old man, a chronic smoker presented to us with two months history of dyspepsia, loss of appetite, significant weight loss and yellowish discolouration of eyes. Subsequently nausea and vomiting further worsened with ensuing poor oral intake. On clinical examination he was dehydrated, had jaundice, cervical and supraclavicular lymphadenopathy. Received: 22 September, 2013. Abdominal examination was unremarkable. Laboratory findings were as follows: haemoglobin 12.7 gm/dL, total leukocyte count 6210/mm³, erythrocyte sedimentation rate 60 mm at the end of the first hour; serum total and direct bilirubin 6.5 mg/dL and 2.9mg/dL respectively, serum alkaline phosphatase 460 U/L (normal 40-140 U/L), serum gammaglutamyl transferase 313 U/L (normal 10-55), serum alanine aminotransferase (ALT) 244 U/ L (normal 0-37 U/L), serum aspartate aminotransferase (AST) 210 U/L (normal 8-48 U/L), serum albumin 3.6 gms/dL, blood urea 65 mg/dL, serum creatinine 2.1 mg/dL, serum CA19-9 6 U/mL (normal 0-37 U/mL). He was human immunodeficiency virus seronegative. Urine examination revealed a bland sediment. Chest radiograph was normal. Abdominal ultrasonography revealed focal hypoechoic lesions in the region of pancreatic head and uncinate process extending to peripancreatic region with extra hepatic biliary obstruction and

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peripancreatic lymphadenopathy; kidneys were normal sized normal sized. Nephrology consultation was sought for renal failure and contrast-imaging was planned. Treatment of pre-renal azotaemia with intravenous fluids resulted in normalization of renal function. Contrast enhanced computed tomography (CECT) of the abdomen with intravenous and oral contrast was then done with appropriate precautions. CECT revealed a multilobulated, heterogeneously enhancing hypodense mass with peripheral ring enhanced lesion arising from head of pancreas and uncinate process measuring $62 \times 55 \times 73$ mm. The hepatic artery was encased within the above mass, the portal vein was found to be compressed and displaced anteriorly (Figures 1A and 1B). No pancreatic duct dilatation or calcification observed. Periportal, peripancreatic and para aortic lymphadenopathy was seen. Duodenum was compressed and displaced outwards by the mass. High resolution computed tomography (HRCT) of chest revealed confluence of small less than 2 mm sizes infiltrates suggesting miliary infiltrates in right upper lobe¹ with a nodular density in apical segment of right upper lobe suggesting pulmonary tuberculosis (Figure 2). Fine needle aspiration cytology (FNAC) of the cervical lymphnodes (Figure 3) was suggestive of tuberculosis; acid-fast bacilli



Figure 1A: CT abdomen showing multiple hypodense lesions (arrow) with peripheral rim enhancement in periportal, peripancreatic celiac axis regions suggestive of lymphadenopathy

were not seen. FNAC from the pancreatic lesion was deferred as it was considered as a highrisk procedure by the radiologist. As baseline hepatic function was deranged a daily, selfsupervised antituberculosis treatment with a modified intensive phase for 3 months consisting of isoniazid, ethambutol and ofloxacin was started. Oral prednisolone was started at a dosage 60 mg/ day for 1 week, 20 mg/day for 1 week and rapidly tapered of to 5 mg/day for 1 week and stopped. Rifampicin was introduced subsequently to above combination after 4 weeks on normalization of liver function. It was followed by maintenance phase of 9 months with isoniazid and rifampicin. Patient was under regular followup for the entire duration of the therapy and was compliant with therapy. He responded well to antituberculosis treatment. There was complete resolution of the pancreatic lesion on subsequent follow-up CT of the abdomen.

DISCUSSION

Obstructive jaundice is attributed to various lesions as calculi, strictures and malignancies arising from gall bladder, common bile duct, ampulla of Vater, pancreas and duodenum. Clinically the nature of the lesion has been approached traditionally through the time tested Courvoisier's law with few rare exceptions.^{4,5} Modern imaging techniques as computed



Figure 1B: CT Abdomen showing multilobulated heterodense mass lesion with peripheral rim enhancement and central non-enhancing hypodensity in the region of head and uncinate process of pancreas (arrow)



Figure 2: CT chest (coronal view) showing a wedge shaped soft tissue nodular density abutting the mediastinal pleura in the apical segment of right upper lobe (thick arrow). Miliary pattern is also evident

tomography (CT), magnetic resonance cholangiopancreatography (MRCP) have greatly increased the diagnostic accuracy. Pancreatic cancer is the fourth leading cause of death in the United States of America (USA) and usually presents with obstructive jaundice, abdominal pain, back pain or other constitutional symptoms. By the time of presentation the disease is usually at an advanced stage with poor prognosis.⁵

Clinical presentation of pancreatic TB closely mimics that of a pancreatic carcinoma and hence poses a diagnostic dilemma.^{6,7} Pancreas enjoys high degree of protection from TB owing to the pancreatic enzymes which interfere with seeding of pathogenic bacilli.^{8,9} The theories proposed in the TB involvement of the pancreas are haematogenous spread from a pulmonary source and direct lymphatic spread from adjacent peripancreatic lymph nodes. Clinical presentation and laboratory evaluation usually fail to differentiate pancreatic TB from malignancy often subjecting the patient to avoidable radical surgery.⁷⁻⁹ Diagnosis is established by histopathology and direct identification of the organism from the tissue by microbiological methods. However, diagnosis can still be difficult as bacteriological confirmation is possible only in 57% cases of extra pulmonary TB.8 Diagnostic tests based



Figure 3: Photomicrograph of fine needle aspiration from supraclavicular lymph node, showing clusters of epithelioid granulomas and Langhans' giant cells, casetion necrosis and scattered lymphocytes (Haematoxylin and eosin, \times 200)

on polymerase chain reaction (PCR) in the tissue samples have been proposed to be highly specific even when tissue samples are negative for bacteriological studies.⁶ Chest radiograph may be normal as is the case in our patient, efforts to identify a possible active TB lesions in the lung by high resolution computed tomography (HRCT) may be helpful as primary TB of pancreas by itself is extremely rare.⁹ Pancreatic TB seems to have a predilection to head and uncinate process of pancreas owing to rich vascular supply. Various lesions described on CT of abdomen are focal hypodense masses, pancreatic nodules, multiloculated, cystic lesions, low attenuation peripancreatic and periportal adenopathies with peripheral rim enhancement.^{6,7,9} In acquired immunodeficiency syndrome (AIDS) patients diffusely hypoechoic enlarged gland and cystic mass lesion have been described on abdominal utorasonography.7

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