



## Pancreatic Tuberculosis: A Case Report

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### ABSTRACT

*We report a case of pancreatic tuberculosis. A 30 year woman who presented with abdominal distention, low grade fever, night sweats and anorexia for two month. She recovered with anti tubercular therapy. Pancreatic tuberculosis, in particular is extremely rare, and only a few cases have been reported. Diagnosis of pancreatic TB has always been a challenge, but radiological investigations and image guided intervention helped in the diagnosis and prevention of unnecessary laparotomy.*

**Key Words:** *Pancreatic tuberculosis, Fine needle aspiration cytology, Polymerase chain reaction*

### INTRODUCTION

Tuberculosis is common in developing countries, but tuberculosis affecting intra-abdominal organs is relatively uncommon. The incidence varies from country to country. Pancreatic TB is an extremely rare entity.<sup>1</sup> In a classical study of 300 cases of abdominal TB carried out by Bansali,<sup>2</sup>

not a single case of pancreatic tuberculosis was reported. In 1944, Auerbach reported that pancreas was affected in 4.7% of cases of military tuberculosis.<sup>3</sup> Tuberculosis being a curable disease, every effort should be made to arrive at an early and timely diagnosis so as to avoid unnecessary interventions.

**CASE REPORT**

A 30 year old woman presented with abdominal distention, fever, night sweats, anorexia & weight loss since one month. She had lost 14 kg over a period of two months. She had enjoyed a good past health and there was no history of pulmonary tuberculosis. On clinical examination, she was poorly built with a weight of 38 kg. She was conscious, oriented with a pulse rate of 76 beats/min, blood pressure of 120/80 mm Hg. Physical examination revealed ascites, but was otherwise normal. On admission, she was found to have diabetes. Her blood sugar level (random) was 400 mg/dl with no ketones in her urine. She was diagnosed as a case of newly detected diabetes mellitus and started on insulin therapy. Her hemoglobin level was 10.8 mg/dl, WBC 7800/cu.mm, liver enzymes and kidney function tests were normal. ESR was 65mm/hr at end of one hour. HIV and Mantoux test were found to be negative. USG abdomen & pelvis revealed gross ascites. Ascitic fluid analysis was not done. Her chest x ray was normal. During hospitalisation, she had intermittent episodes of hyperglycemia & hypoglycaemia for which insulin was adjusted as per her blood sugar levels. She was diagnosed as a case of newly detected diabetes mellitus with ascites and was discharged. She came to us and was admitted after 8 days for same complaints and uncontrolled sugar levels, and suspected of abdominal kochs with a suspicious of pancreatic tuberculosis. She had brittle diabetes with large excuration and falls of blood sugar levels. Chest x ray suggestive of left sided pleural

effusion. Ultrasound of abdomen showed edematous pancreas with small hypoechoic cystic lesion in the the head of pancreas, peripancreatic and mesenteric lymphadenopathy. The pancreatic duct was normal. CT abdomen did not reveal anything more than the USG. On history, she had mild pancreatic exocrine insufficiency of recent onset. Ascitic fluid analysis was done which revealed amylase of 800 IU/l, lipase of 1200 IU/l and ADA was 63 IU/l. Further fine needle aspiration cytology & Polymerase chain reaction (PCR) was performed, USG guided FNAC revealed caseous necrosis and PCR was positive. Both the reports confirmed the clinical suspicion of pancreatic tuberculosis. She was started on anti kochs therapy and within six weeks her symptoms got relieved. She gained weight of 12 kg within a period of 2 months. At the end of 6 week, her amylase and lipase levels fell and her resolving ascites, no fever, increased appetite and increased weight gain confirm rebust response to AKT therapy. Her blood sugar level fluctuations were reduced and blood sugar level stabilized at the end of 2 months. We treated her for exocrine pancreatic insufficiency & insulin deficiency syndrome. Patient was discharged and under constant follow up after six months of anti kochs therapy, after her release from treatment of T.B. She still suffers from mild pancreatic exocrine insufficiency syndrome. USG has been found with normal pancreas and abdomen. Her weight and appetite was improved. Blood sugar levels were under control. This case has been presented with a view to highlight the entity of pancreatic tuberculosis which is extremely rare.

## DISCUSSION

Tuberculosis is a systemic disease with protean manifestations. Approximately 15% of cases involve extrapulmonary sites.<sup>4</sup> Since pancreatic tuberculosis is rare,<sup>5</sup> it poses a clinical dilemma,<sup>6</sup> as it does not commonly figure in the differential diagnosis of a pancreatic mass, or ascites.<sup>7,8</sup>

Pancreas is biologically protected from infection by mycobacterium tuberculosis because of the presence of pancreatic enzymes, however when pathogen is able to overcome the resistance, the disease pattern can be varied. The exact way in which pancreas is involved is yet to be completely understood. But two main hypotheses have been proposed. One theory suggests that by hematogenous dissemination after pulmonary disease organisms reach pancreas.<sup>9</sup> Another theory proposes that pancreatic tuberculosis can be caused by direct spread from adjacent peripancreatic lymph nodes.<sup>10</sup>

The pancreas is rarely affected by tuberculosis. In 1944, Auerbach reported pancreatic involvement in 4.7% of biopsies in cases of miliary tuberculosis.<sup>11</sup> Between 1891 and 1961, Paraf et al. reported 11 cases of pancreatic involvement in necrosis of miliary tuberculosis, with 2.1% incidence of involvement of this organ. Between 1980 and 1998, 14 cases were reported in the literature,<sup>12</sup> the majority in young adults (mean age 33 years).

Possible mechanisms of involvement of the pancreas are as follows:

- The first possible way<sup>13</sup> is that tubercle bacilli reach the pancreas through

haematogenous dissemination from an occult lesion in the lungs or abdomen.

- The second way<sup>14</sup> is that the route by direct spread from contiguous lymph nodes may be responsible for most of the cases with isolated pancreatic TB.
- The third possible way<sup>15</sup> is that dormant bacilli in an old tubercular lesion can re-activate in an immunosuppressive state.

Rana et al.<sup>16</sup> Isolated pancreatic tuberculosis mimicking focal pancreatitis and causing segmental hypertension. CECT revealed focal pancreatitis involving head and body of pancreas with splenic vein thrombosis. Cytological examination of image-guided aspiration revealed caseous material. Culture demonstrated growth of *Mycobacterium tuberculosis*.

Arora et al.<sup>17</sup> Patient presented with chronic abdominal pain and weight loss. CT revealed a well margined cystic lesion in the head of pancreas with upstream biliary dilatation. Endoscopic ultrasound-guided fine needle aspiration biopsy (FNAB) showed epithelioid granulomas PCR based assay confirmed *Mycobacterium tuberculosis*.

Falkowski et al.<sup>18</sup> Patient presented with pancreatitis. CT showed multi cystic solid mass with slight contrast enhancement in area of pancreatic head, located in the branching of celiac trunk and adjacent to the portal vein. Endoscopic ultrasound-guided FNAC revealed necrotising granulomatous infection and numerous acid-fast bacilli on microscopy. PCR positive for *Mycobacterium tuberculosis*.

DM is now a recognized risk factor and common complication encountered among TB patients.<sup>19, 20</sup> Recent evidence suggests that diabetic patients have an increased tendency to develop TB due to impaired cell-mediated immunity, renal failure, micronutrient deficiency, and pulmonary microangiopathy.<sup>21</sup> Chronic infections like TB are associated with reactionary hyperglycemia which occurs due to increased production of counter-regulatory stress hormones like epinephrine, glucagon, cortisol, and growth hormone that act synergistically.<sup>22</sup>

Treatment of DM among patients with TB should involve the use of insulin or oral hypoglycaemic agents (OHAs) in order to achieve the optimal goals of therapy, that is, maintaining an HbA1c of <7%, random blood sugar level <180 mg/dl or a FBG level <120 mg/dl.<sup>23</sup> However, therapeutic doses of most glucose lowering drugs may need to be increased during the initial phases of TB treatment. This is because rifampicin induces an acute transient hyperglycemia due to its effect of augmenting intestinal absorption of glucose.<sup>24</sup> Due to its cytochrome P450 enzyme inducing properties, it also augments hepatic metabolism of most OHAs.<sup>25</sup>

The common presenting features are non-specific abdominal pain, fever, anorexia and weight loss.<sup>26</sup> Less common symptoms include iron deficiency anaemia, vomiting, obstructive jaundice, upper gastro-intestinal bleeding and portal hypertension.<sup>27</sup> Patients may or may not have had other forms of tuberculosis in the past. Clinical examination is usually non-contributory.<sup>28</sup> Ultrasonographic features include

a diffusely enlarged pancreas with focal hypoechoic lesions or cystic lesions of the pancreas.<sup>29</sup> Associated findings include peripancreatic and mesenteric lymphadenopathy,<sup>30, 31</sup> bowel wall thickening (usually in the ileocaecal region), focal hepatic or splenic lesions and ascites.<sup>32</sup> CT scan most commonly reveals a mass lesion.<sup>33, 34</sup>

Therefore, to establish the diagnosis of pancreatic TB, histological, cytological as well as bacteriological confirmations are necessary. Ultrasound-guided (USG) or CT-guided FNAC has been used to confirm the diagnosis and to prevent unnecessary laprotomies.<sup>35-37</sup>

Ultrasound or CT-guided FNAC may provide the diagnosis, especially with the help of an expert cytologist experienced in the diagnosis of tuberculosis.<sup>34</sup>

The varied presentation and rare occurrence of pancreatic tuberculosis is the main reason for its diagnosis becoming difficult and a high degree of suspicion is necessary for a medical and pre or intraoperative diagnosis. USG or CT guided aspiration cytology may help in differentiating this from carcinoma, lymphoma, chronic pancreatitis or sarcoidosis.<sup>38</sup>

A recent diagnostic test is the polymerase chain reaction (PCR) based assay, which detects mycobacterium tuberculosis DNA in resected specimens. It is a highly specific assay and may give a positive result even when special staining techniques and cultures of these tissues are negative.<sup>39</sup>

The treatment of pancreatic tuberculosis comprises multi-drug anti-tuberculous

chemotherapy for between 6 and 12 months. Response to therapy is predictable and complete. These patients still need to be followed up carefully for subjective and objective response to therapy to rule out the rare possibility of tuberculosis coexisting with malignancy, especially in endemic areas.<sup>40</sup>

## CONCLUSION

The diagnosis of pancreatic tuberculosis requires a high degree of suspicion and, although is a rare condition, should be considered as a differential diagnosis in patients with pancreatic lesions. Pancreatic TB may present as SOL pancreas, peripancreatic collection as pseudocyst, peripancreatic lymphadenopathy, ascites and portal hypertension. High index of suspicion, CT/USG FNAC is extremely important to make the diagnosis of pancreatic TB. The majority of patients respond well to anti-tubercular chemotherapy and prognosis is good and surgical intervention can be overted in developing countries as like India.

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