CASE REPORT

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Pancreatic Tuberculosis with Reversible Exocrine Insufficiency

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Abstract

Tuberculosis (TB) continues to be a major cause of morbidity and mortality in the Indian subcontinent. However, pancreatic tuberculosis is a rare form of abdominal tuberculosis and commonly mimics pancreatic malignancy. The literature on pancreatic tuberculosis with exocrine insufficiency is extremely sparse. Herein, we report a 44-year-old male patient who presented with pain abdomen and steatorrhoea for two years and a diagnosis of pancreatic tuberculosis with exocrine insufficiency was made after imaging and histopathological confirmation. He was successfully treated with anti-tuberculous therapy with complete resolution of pancreatic lesion and exocrine insufficiency.

Key words: Abdominal tuberculosis, fecal elastase, pancreatic mass.

Introduction

Tuberculosis (TB) remains a major global health burden, with an estimated 10.6 million new cases reported worldwide annually. India continues to account for a substantial proportion of the global TB burden. Extrapulmonary tuberculosis constitutes nearly 15 - 20% of all TB cases, while abdominal tuberculosis accounts for approximately 5 - 12% of extrapulmonary disease[1]. The ileocecal region, peritoneum, and abdominal lymph nodes are the most commonly involved sites. Pancreatic tuberculosis is an exceptionally rare form of abdominal TB, even in endemic regions, accounting for less than 5% of abdominal tuberculosis cases[2].

Exocrine pancreatic insufficiency (EPI), characterised by inadequate secretion of pancreatic enzymes leading to malabsorption and steatorrhoea, is well recognised in chronic pancreatitis and pancreatic malignancy but has not been reported in pancreatic tuberculosis[3]. Faecal elastase serves as a reliable non-invasive marker of pancreatic exocrine function. We report a rare case of pancreatic tuberculosis presenting with faecal elastase-proven exocrine pancreatic insufficiency, which subsequently demonstrated clinical and biochemical reversal following anti-tubercular therapy.

Case Report

A 44-year-old male presented to our outpatient department with complaints of central epigastric pain associated with progressive weight loss, anorexia, generalised weakness, and greasy sticky floating stools for the past one year, with worsening abdominal discomfort over the preceding 7 days. On examination, the patient was thin-built with pallor and reduced muscle mass.

Laboratory investigations revealed severe microcytic anaemia with haemoglobin of 6.7 g/dL, mean corpuscular volume (MCV) - 76.2 fl. Iron profile showed serum iron of 12 µg/dL, total iron-binding capacity (TIBC) - 420 µg/dL, and serum ferritin - 72.2 ng/mL and peripheral blood smear showed microcytic hypochromic red blood cells suggesting anaemia of chronic disease. Total leukocyte count was 9,230/mm^3 with markedly elevated inflammatory markers (CRP: 107 mg/L, ESR: 28 mm/hr) and random blood glucose level was 110 mg/dL.

Viral markers including HIV, HBsAg, and anti-HCV were negative. ANA profile was negative. Serum amylase, lipase, serum ACE levels, CA 19 - 9 and serum IgG4 levels were within normal limits. Fecal elastase level was found to be markedly reduced 98 µg/g, suggesting severe exocrine pancreatic insufficiency.

Point-of-care ultrasonography performed after admission revealed a pancreatic lump with surrounding lymphadenopathy, following which contrast-enhanced computed tomography (CECT) of the abdomen was performed. CECT abdomen demonstrated a bulky heterogeneously enhancing pancreas with multiple hypodense non-enhancing areas involving the pancreatic body and peripancreatic region, associated with marked peripancreatic stranding and multiple necrotic retroperitoneal, peripancreatic and mesenteric lymph nodes with peripheral enhancement and calcification (Fig. 1).

CT-guided biopsy of retroperitoneal lymph node was subsequently performed. Histopathological examination demonstrated epithelioid granulomas with multinucleated giant cells in a reactive lymphoid background, consistent

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with granulomatous lymphadenitis. Ziehl-Neelsen staining for acid-fast bacilli was negative (Fig. 2).

Based on the clinic-radiological findings and histopathological evidence, a diagnosis of pancreatic tuberculosis with secondary exocrine pancreatic insufficiency was established. The patient was initiated on four drug anti-tubercular therapy (ATT) along with pancreatic enzyme replacement therapy.

During follow-up, the patient demonstrated marked symptomatic improvement with resolution of abdominal pain and improvement in appetite. Repeat fecal elastase after 2 months of treatment improved to 142 µg/g and normalised after six months of ATT. Serial ultrasonography over a 6-month follow-up period demonstrated complete resolution of pancreatic swelling and peripancreatic lymphadenopathy.

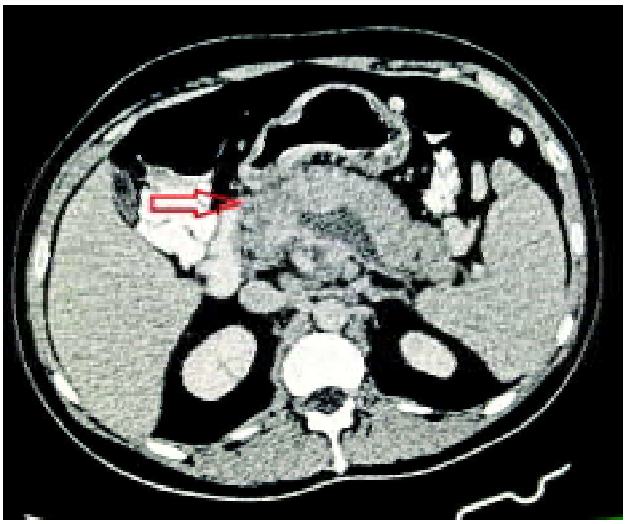


Fig. 1: *CECT abdomen showing pancreatic enlargement with necrotic peripancreatic lymphadenopathy.*

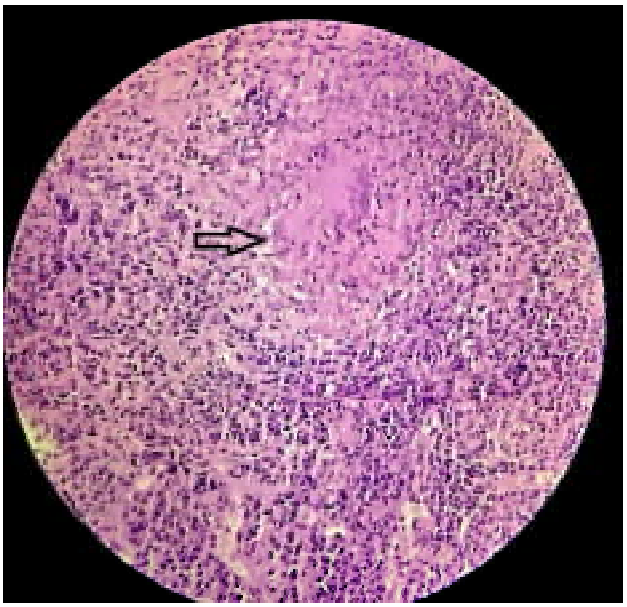


Fig. 2: *Retroperitoneal lymph node biopsy showing epithelioid granulomas and multinucleated giant cells.*

Discussion

Pancreatic tuberculosis remains an uncommon manifestation of extrapulmonary TB despite the high prevalence of tuberculosis in endemic regions. The rarity of pancreatic involvement has been attributed to the bactericidal effect of pancreatic enzymes, lipase and deoxyribonuclease, which provide relative resistance to Mycobacterium tuberculosis. Pancreatic TB may occur through haematogenous dissemination, lymphatic spread, or direct extension from adjacent lymph nodes[4].

The clinical presentation of pancreatic TB is non-specific and frequently overlaps with pancreatic malignancy. Abdominal pain, anorexia, weight loss, fever, jaundice, and pancreatic masses are commonly reported features[5]. Clinically and radiologically, pancreatic tuberculosis frequently mimics pancreatic malignancy, like in our case. Radiological modalities such as ultrasonography and contrast-enhanced computed tomography (CECT) aid in identifying pancreatic lesions; however, tissue diagnosis remains essential. Endoscopic ultrasound (EUS)-guided aspiration or image-guided biopsy demonstrating granulomatous inflammation plays a crucial role in establishing the diagnosis and preventing unnecessary surgery[6,7].

Exocrine pancreatic insufficiency secondary to pancreatic tuberculosis is exceedingly rare and poorly documented in literature. Faecal elastase is a reliable non-invasive marker of pancreatic exocrine function, with levels below 100 µg/g indicating severe insufficiency[8,9]. In the present case, reduced faecal elastase levels correlated with symptoms of steatorrhoea and malabsorption, providing objective evidence of pancreatic exocrine dysfunction.

More importantly, serial follow-up demonstrated gradual improvement and eventual normalisation of faecal elastase levels following anti-tubercular therapy, accompanied by radiological resolution of pancreatic lesion. This strongly suggests that pancreatic exocrine dysfunction in tuberculosis may be reversible with timely diagnosis and treatment.

Previous reports of pancreatic tuberculosis have primarily focused on its resemblance to malignancy and diagnostic difficulties[10]. However, to the best our knowledge this is the first case of reversible exocrine pancreatic insufficiency in pancreatic tuberculosis.

This case highlights pancreatic tuberculosis as a rare differential diagnosis of pancreatic masses and a potentially

reversible cause of secondary exocrine pancreatic insufficiency.

Conclusion

Pancreatic tuberculosis is a rare but important differential diagnosis in patients presenting with pancreatic masses and constitutional symptoms. This case highlights an unusual presentation with faecal elastase-proven exocrine pancreatic insufficiency, which demonstrated clinical, biochemical, and radiological reversal following anti-tubercular therapy. Recognition of this potentially reversible entity is crucial, particularly in endemic regions, as timely diagnosis and tissue confirmation can prevent unnecessary surgical procedures and result in excellent clinical outcomes.

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