

Pancreatic tuberculosis: An unusual presentation

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ABSTRACT

Tuberculosis (TB) of pancreas is a rare presentation in both immune-competent and immune-suppressed patients. Its presenting clinical features are usually vague and non-specific, while radiological features mimic other common pancreatic conditions such as malignancy, so it is often misdiagnosed. It commonly involves the head and the uncinate process of the pancreas. We report a middle-aged immune-compromised man who presented with left-sided tubercular pleural effusion and later was diagnosed as pancreatitis by clinical presentation and TB of pancreas on computed tomography of the abdomen.

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INTRODUCTION

Tuberculosis (TB) is a serious health problem worldwide. About 12.5% of all TB cases are extrapulmonary, of which 11% to 16% account for abdominal TB.^{1,2}

Extrapulmonary TB is seen in nearly 50% of HIV-positive patients. In isolated extrapulmonary TB cases (without pulmonary TB), pancreatic TB occurs especially in acquired immunodeficiency syndrome patients (0.46%).^{1,3}

TB of pancreas with or without peri-pancreatic lymphadenitis is not a common occurrence in both immune-competent and immune-suppressed patients.

THE CASE

A 40-year-old man presented to the emergency department with complaints of shortness of breath, low-grade fever, weight loss and loss of appetite for 1 month. There were no significant comorbid conditions or any addictions. On admission, vital parameters were stable, and general physical examination revealed anaemia and tachypnoea. Chest examination revealed decreased left-sided air entry with dullness on percussion, and chest X-ray showed blunting of the left costophrenic angle, suggestive of pleural effusion. Pleural tap was done, and pleural fluid was suggestive of tubercular effusion. Blood investigation showed microcytic hypochromic anaemia (haemoglobin 8.3 g/dl) with normal total leucocyte count, raised erythrocyte sedimentation rate (106 mm/hour) and positive Mantoux test (20 mm×22 mm). Renal function test and liver function test (LFT) were normal. Acid-fast bacilli stain on sputum was non-contributory. Carbohydrate antigen 19-9 was not elevated. The patient was started on antitubercular treatment (ATT); 2 days

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after starting ATT, the patient developed epigastric pain (non-radiating) with nausea and vomiting. The liver function tests, serum amylase and serum lipase level were normal (82 and 17 μ /L, respectively). The patient was managed symptomatically and conservatively, and a diagnosis of isoniazid-induced pancreatitis was made and ATT was modified accordingly; meanwhile, other investigations such as HIV turned out to be positive with CD4 count of 44. Computed tomography (CT) scan of the abdomen was also done which showed hepatosplenomegaly with multiple splenic granulomas and two well-defined lesions in the body and tail of the pancreas, with surrounding lymphadenopathy likely to be of tubercular aetiology. He tolerated the treatment well. Repeat LFT was normal. His condition improved symptomatically, and he was discharged on day 23 with advice to follow-up regularly.

DISCUSSION

Abdominal TB is the sixth most common type of extrapulmonary TB, but pancreatic TB is rare.

Pancreatic tissue resists invasion by *Mycobacterium tuberculosis*, as it is a retroperitoneal organ, and is protected from direct environmental exposure and also due to antimycobacterial effects of pancreatic enzymes (lipase and

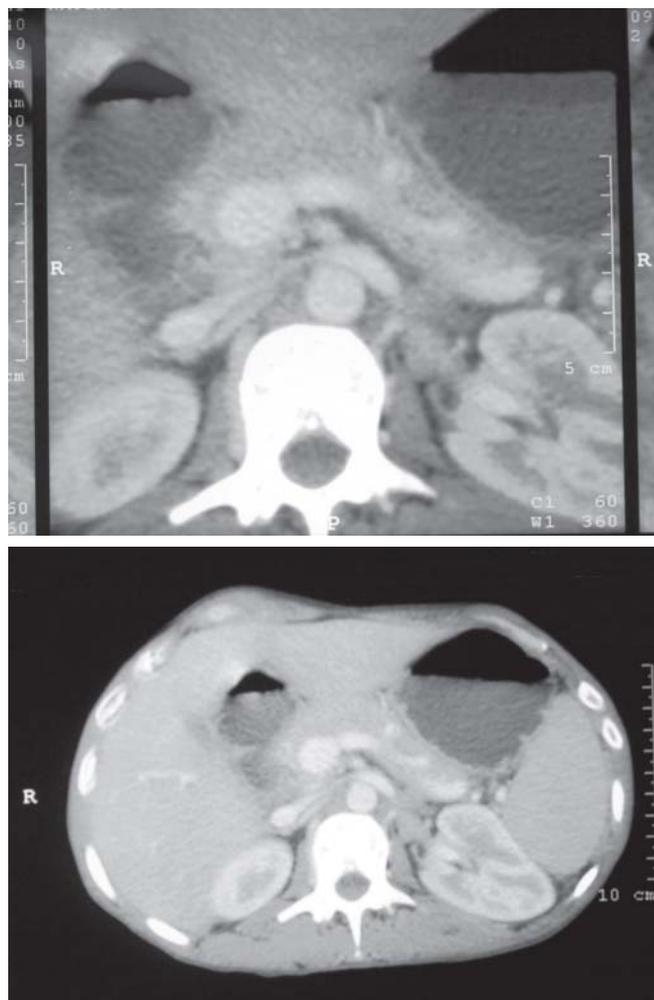


FIG 1. CT scan of abdomen showing two well-defined lesions in the body and tail of pancreas with surrounding lymphadenopathy

deoxyribonuclease extracts).⁴ The possible mechanisms by which pancreas is involved are:⁵

- Haematogenous dissemination of tubercle bacilli from lungs or abdomen (occult lesion) to pancreas
- Direct spread from contiguous lymph nodes (in cases of isolated pancreatic TB)
- Reactivation of dormant tubercular bacilli in an old lesion, in an immunosuppressive state.

Mycobacterial infection of the pancreas can be of three types: (i) generalized/miliary TB (the agent is *M. tuberculosis*); (ii) pancreatic spread from coeliac and other retroperitoneal lymph nodes (the main agent to be considered is *M. bovis*); and (iii) primary localized pancreatic TB (origin from the intestinal tract).⁶

Auerbach in 1944 reported that only 4.7% (14 of 1656 autopsies of tuberculous patients) cases had pancreatic involvement.⁷ However, in an Indian study (1999–2004), pancreatic TB was detected in 8.3% of patients of diagnosed abdominal TB.⁸ Pancreatic TB is predominantly seen in patients who reside in areas endemic for TB, in areas of widespread TB dissemination (as in developing countries) and in immunocompromised patients.

The predominant symptoms include abdominal pain (75%–100%), anorexia and weight loss (69%), fever and night sweats (50%), anaemia (50%) and obstructive jaundice (30%).⁴

The most important factor in diagnosing a case of pancreatic TB is having a high index of suspicion, as the symptoms and imaging findings may be subtle or confusing. Direct histopathological examination is the best way of diagnosing TB. Ultrasonography/CT/endosonography-guided biopsy is the recommended diagnostic technique.

Pancreatic TB is difficult to diagnose, but once diagnosed,

it responds well with conventional ATT and the treatment remains the same as for any other extrapulmonary TB.⁹ Standard ATT involving the use of at least four drugs remains the cornerstone of treatment.^{2,5}

Conclusion

Abdominal TB has diverse and non-specific symptomatology. Pancreatic TB is usually difficult to diagnose, requiring a high index of clinical suspicion. Hence, TB should always be considered in the differential diagnosis of any pancreatic masses as it usually responds well to ATT.

Conflicts of interest. None declared

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