Isolated Pancreatic Tuberculosis in an Immunocompetent Host

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Abstract
Despite the high prevalence of tuberculosis (TB) worldwide, pancreatic TB is rare. When present, pancreatic TB is frequently associated with miliary TB, often in immunocompromised hosts. Pancreatic TB may present as a pancreatic abscess, acute or chronic pancreatitis, and cystic or solid pancreatic masses. This is a case of isolated Tubercular infection of the pancreas in an immunocompetent patient, who presented with a discrete pancreatic abscess, and was subsequently diagnosed with isolated pancreatic TB. This case suggests that clinicians should have a heightened suspicion of pancreatic TB when faced with discrete pancreatic lesions, especially in patients from areas where the infection is endemic. Such recognition may lead to appropriate diagnostic testing, and possible resolution of pancreatic lesions with antitubercular therapy.

Introduction
Tuberculosis (TB) involving the pancreas is uncommon, especially when present in immunocompetent hosts.¹ Often occurring in the setting of miliary TB or widely disseminated disease; isolated primary pancreatic TB is extremely rare.¹ Pancreatic TB may present as a pancreatic abscess, acute or chronic pancreatitis, and cystic or solid pancreatic masses.¹ We present a case of isolated pancreatic TB presenting as two discrete pancreatic masses in an immunocompetent host.

Case Presentation
A 16-year-old adolescent male, a resident of West Midnapur district of West Bengal, presented to the outpatient department of Internal Medicine, with complaints of fever associated with abdominal pain of 2 months’ duration. He gave a history of fever which was high grade and not associated with chills or rigors, intermittent in nature, more noticeable during the evenings. He also concomitantly developed a dull, intermittent, mid epigastric pain which was neither alleviated nor aggravated by any factors. He also admitted that he had anorexia and observed a weight loss of around 5kgs in those 2 months. He denied any vomiting, hematemesis, melena, burning micturition and cough. The patient stated that he had previously been in good health and denied the use of any medications. He denied the use of illicit drugs, alcohol, and tobacco and he had never participated in any sexual activity. Family history was noncontributory, including the absence of malignancy, or gastrointestinal disorders, tuberculosis.

Physical examination revealed a thinly built individual with mild midepigastric tenderness without guarding or rigidity, on palpation. His lab tests were unremarkable and showed a normal level of hepatic transaminases and total bilirubin; serum amylase and lipase levels were also within normal range. Chest radiograph was normal. However, the sonography of the abdomen (Figure 1) showed a bulky pancreas suggestive of abscess, associated with upper abdominal lymphadenopathy and splenomegaly which prompted further evaluation by a Computed tomography (CT) of the abdomen that revealed a 3.4 x 3.3 cm cavity containing air fluid level within the mid body of the pancreas. The body and tail of the pancreas were bulky with significant peripancreatic fat stranding with multiple pathologically enlarged peripancreatic lymph nodes, the largest of which measured 18 mm in cross-section (Figure 2).

CT guided fine needle aspiration (FNA) of the pancreatic head mass was performed.

On-site cytological and histopathological evaluation of the biopsy specimen revealed the presence of karryorrhectic debris of predominantly lymphocytes and few histiocytes with granuloma / giant cells seen, suggestive of granulomatous inflammation with no evidence of...
malignancy (Figure 3). Aspirate sample tested positive for mycobacterial DNA using Gene Xpert assay. This was further categorised as a sensitive strain with no in vivo antitubercular resistance.

Given the diagnosis of pancreatic TB, he was subsequently tested for retroviral infection, which was negative. The patient was commenced on antituberculor therapy with isoniazid, rifampin, pyrazinamide, and ethambutol for 3 months and came for review after 3 months. He was found to be clinically asymptomatic and sonography revealed complete regression of pancreatic lesions ATT was continued for total duration of 6 months.

**Discussion**

Tuberculosis (TB) is a multisystemic infectious disease caused by various strains of mycobacterium, usually Mycobacterium tuberculosis. In 2013, an estimated 9.0 million people developed TB and 2.5 million died from the disease, with the highest incidence of infection occurring in Asia, South America, eastern Europe, and most sub-Saharan African countries. Although pulmonary TB is the most common presentation of disease; extra pulmonary TB (EPTB) accounts for nearly 20 percent of all cases of TB in immunocompetent hosts, and nearly 50 percent of all cases of TB in patients with human immunodeficiency virus.

By definition, EPTB describes the occurrence of TB at sites other than the lung. The term must not be confused with miliary TB, as this refers to pulmonary involvement with EPTB, and not EPTB in isolation. EPTB can occur in almost any organ system, with the most common sites of infection being the lymph nodes, pleura, genitourinary system, and bone. Abdominal TB is the sixth most common site for EPTB and includes infection anywhere in the gastrointestinal tract, peritoneum, and intra-abdominal organs such as the spleen, liver, and pancreas.

Pancreatic TB is rare, with an incidence reported to be less than 4.7 percent worldwide. Isolated pancreatic TB is extremely uncommon, with pancreatic involvement usually occurring in the setting of miliary or widely disseminated TB; often in immunocompromised hosts. Infection of the pancreas is thought to occur by direct extension to the organ via lymphatic or hematogenous spread, or by reactivation of previous TB infection. Pancreatic TB may present as pancreatic abscesses, acute or chronic pancreatitis, gastrointestinal bleeding, and in rare cases, discrete pancreatic masses mimicking malignancy.

The clinical presentation of pancreatic TB is often insidious, with nonspecific constitutional symptoms occurring frequently. In a study by Saluja et al., the three most common presenting complaints in patients found to have pancreatic TB were abdominal pain, jaundice, and weight loss. Individuals infected with pancreatic TB may also present with fever, gastrointestinal hemorrhage secondary to splenic vein thrombosis, and anorexia. If pancreatic TB is suspected, preliminary testing such as tuberculin skin testing and an interferon-γ release assay for TB may be negative in patients. Sharma et al. suggest that the sensitivity of tuberculin skin testing in patients with abdominal tuberculosis may range from 58 to 100 percent. With the wide-ranging sensitivities of TB screening modalities and an often nonspecific and varied clinical presentation of pancreatic TB; diagnosis of infection relies heavily on radiologic and histopathologic findings.

Ultrasonography or computed tomography (CT) are often first-line diagnostic modalities in patients presenting with signs of pancreatic pathology. Ultrasonography and CT may reveal both hypo dense and hyper echoic lesions, typically found in the head of the pancreas. The findings of these solid or cystic lesions are however nonspecific, as pancreatic adenocarcinomas, cystadenocarcinomas, and pancreatic pseudocysts often have similar appearances. D’cruz et al. further suggest that there is no radio graphical difference between cystic neoplasm of the pancreas and pancreatic TB abscess formation, as both present as septated masses with surrounding hypo dense lymphadenopathy. As the initial radiographic and clinical presentation of isolated pancreatic TB may mimic malignancy; histologic evaluation of the lesion is essential for diagnosis of pancreatic TB.

Techniques for pancreatic biopsy include CT or ultrasound-guided percutaneous biopsy, surgical biopsy, or endoscopic ultrasound- (EUS-) guided fine needle aspiration (FNA). The presence of on-site cytology is imperative in the diagnosis of pancreatic TB, as the immediate interpretation of the specimen will allow clinicians to request appropriate cultures. The presence of on-site cytology has also been shown to increase the diagnostic yield of FNA by up to 15 percent and may also decrease potential complications by avoiding the need for multiple needle passes once initial diagnostic tissue is procured. Cytologic interpretation of biopsy specimens may reveal the presence of granulomatous inflammation, with the presence of aggregates of epithelioid histiocytes, plasma cells, and lymphocytes. Acid fast bacilli are commonly not seen with FNA. In a study by Farar et al., nearly 40 percent of patients with abdominal TB had staining that was negative for acid fast bacilli. Clinicians should be cognizant of the relatively low yield of FNA specimens to reveal acid fast bacilli and thus culture the specimen for evidence of Mycobacterium tuberculosis. Bacterial culture, although requiring a prolonged incubation, has proven to be the most specific diagnostic modality to reveal pancreatic TB.

Once the diagnosis of pancreatic TB has been made, standard antituberculin therapy appears to be successful in management of this infection. A minimum of 6 months of antituberculin therapy is often indicated to achieve resolution of pancreatic lesions and alleviation of symptoms. Follow-up CT imaging after treatment may reveal the complete resolution of pancreatic lesions secondary to tuberculosis and may guide clinicians regarding duration of therapy.
Conclusion

Isolated pancreatic TB is extremely rare and may present as discrete pancreatic abscess. Clinicians should have heightened suspicion of infectious processes such as TB as a potential etiology of such abscess. Furthermore, TB should be considered as a cause of any suspicious pancreatic lesion, especially in patients from areas where the infection is endemic. Clinical awareness of pancreatic TB may guide clinicians to appropriate diagnostic studies and management; which may lead to alleviation of symptoms and possible resolution of pancreatic masses with antituberculin therapy.

References