Case Report

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Pancreatic tuberculosis: A rare case

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Abstract

Pancreatic tuberculosis is an extremely rare condition, even in an endemic region like Nepal, and confirming the diagnosis is even more difficult due to the difficulty in obtaining the pathological specimen. In this case report, we present a young male who was diagnosed with pancreatic tuberculosis and inadvertent surgery avoided following proper workup of the patient.

Key words: tuberculosis, pancreas, ascites

Case profile

A 22 year-old gentle man presented with abdominal pain for 1 year and abdominal distention for 1 month. Pain was in the epigastric region, it was dull in nature and radiating to right upper quadrant and decreased after taking analgesics. Pain was associated with nausea.

Distension of abdomen used to increase after taking food but distension and discomfort continued since 1 month and was gradually increasing. There was history of weight loss. He had good health in the past and did not have any history of pulmonary tuberculosis in the past.

Physical examination showed ascites, but was otherwise normal. His haemoglobin level was 13.8g/dl; white blood cell count 7.9 × 10^9L; liver enzymes and renal function tests normal; and erythrocyte sedimentation rate was 32mm/h Wintrobe. Abdominal tapping yielded a straw-colored fluid which contained red blood cell and white blood cell and 10% neutrophils and 90% lymphocytes and no atypical cells. On gram staining no micro organism was seen. No acid fast bacilli (AFB) was seen. Protein was 5.8g%, LDH was 535 U/l in ascetic fluid. Ascetic fluid ADA was 92 U/L.

Ultrasonography of abdomen showed encysted fluid collection and calcification in the area of head of pancreas and ascites. A computed tonography scan of the abdomen showed a hetetogeneous mass measuring 43.6 × 23.3 mm and demonstrating central cystic area measuring 22.6× 23.6 mm in head of the pancreas with moderate amount of ascites. There was lymphadenopathy in the hepatoduodenal ligament. A computed tomography scan of his lungs showed evidence of pulmonary tuberculosis.USG guided fine needle aspiration (FNAC) yieded few scattered epithelial calls along with acute and chronic inflammatory cells and negative for malignant cells.

On Peritoneoscopic examination there was multiple peritoneal adhesion and peritoneal cavity was stucked with nodules. The biopsy taken revealed multiple necrotizing epithelial cells, granulomas and multinucleated langhans gaint cells which was consistent with tuberculosis. A ZN stain did not show acid fast bacilli.

A diagnosis of tuberculous infection of the pancreas was made and he is being treated with ethambutol. Pyrezinamide, Isoniazid and rifampicin.

Discussion

Tuberculosis is common in developing countries, but tuberculosis affecting intraabdominal organs is relatively uncommon. Even though Tuberculosis can involve any part of the gastrointestinal tract and is the sixth most frequent site of extra pulmonary involvement. Abdominal tuberculosis mainly involves abdominal lymph nodes and the ileocecal junction, other organs that are uncommonly involved the rest of gastrointestinal tract, peritoneum, liver spleen[12]. Tuberculosis bacteria reach the gastrointestinal tract via haematogenous spread, ingestion of infected sputum, or direct spread form infected contiguous lymph nodes and fallopian tub es.

Peritoneal tuberculosis occurs in three forms: wet type with ascitis, dry with adhesions, and fibrotic type with omental thickening and loculated ascites. The most common site of involvement of the gastrointestinal tuberculosis is the ileocaecal region. Tuberculosis (TB) can involve any part of the gastrointestinal tract form mouth to anus, the peritoneum and pancreatobiliary system.

The postulated mechanisms by which the tubercle bacilli reach the gastrointestinal tract are: (i) haematogenous spread form the primary lung focus in childhood, with later reactivation; (ii) ingestion of bacilli in sputum form active pulmonary focus; (iii) direct spread form adjacent organs; and (iv)and through lymph channels form infected nodes.

The pancreas is biologically protected form infection by Mycobacterium tuberculosis, probably because of the presence of pancreatic enzymes that interfere with the seeding of M tuberculosis. However, when pathogens are able to overcome the resistance, they can have diverse presentations, such as pancreatic masses that can mimic carcinoma, obstructive jaundice, pancreatitis, and gastrointestinal bleeding.

Pancreatic involvement of TB is extremely rare. It is very difficult to confirm the diagnosis because of difficulty in obtaining the pathological evidence, and

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lack of high index suspicion always misled by pancreatic neoplasm's. Similarly the wide spectrum of presentation makes the abdominal tuberculosis a difficult disease to recognize[3]. Therefore, pancreatic tuberculosis should be kept in mind among the differential diagnosis of solitary masses in the pancreas, especially in young people in developing countries. The development of abdominal tuberculosis is independent of pulmonary disease in most patients. Auerbach reported in 1944 that 4.7% of 297 autopsy cases of miliary tuberculosis involved the pancreas[4]. Paraf et al in 1966 reviewed autopsy studies of miliary tuberculosis between 1891 and 1961 and found only an incidence of 2.1%(11/526) of pancreatic or peripancreatic involvment. In 1977, Bhansali did not report a single case involvement of the pancreas in 300 patients with abdominal tuberculosis from India[6].

Tuberculosis although rare, should be considered as a differential diagnosis in patients with a pancreatic cystic lesion, especially in those with constitutional symptoms[7]. Several factors have made doctors overlook the diagnosis of pancreatic tuberculosis: (1) the disease is rare; (2) tuberculosis is a stigma of the poor; (3) and abdominal tuberculosis may not always be associated with active pulmonary tuberculosis.

A diagnosis of tuberculosis that can be suggested only in the presence of ancillary findings like pulmonary tuberculosis, pleural effusion, enlarged celiac lymph nodes, lesions in other solid viscera, ascites, mural thickening in the ileocecal region or positive tuberculin test[8, .9,10].

The ascetic fluid has a high protein content (>2.5-3 mg/dl), with a predominance of lymphocytes and presence of neutrophils and monocytes. Adenosine deaminase (ADA) an enzyme found in many cells types-macrophages, lymphocytes and erythrocytes. It is a marker host immune response in case of abdominal tuberculosis. Serum ADA activity values of more than 42 U/1 have significant. ADA levels >33U/1 100% and 95% sensitivity and specificity respectively[11]. Serum lactate dehydroginase levels are elevated to over 90 U/1 in patients with intestinal tuberculosis[12].

Abdominal CT and US may show an enlarged pancreas with focal hypodense or hypoechoic lesion, usually in the head region, sometimes with irregular multilobular cyst arising form pancreas. However, these findings are nonspecific and simulate solid and/or cystic pancreatic neoplasms. US or CT-guided percutaneous FNAC of tumor and to obtain proof of the bacilli by the Ziehl-Neelsen stain or by culture is one diagnostic option, however, the aspiration of material form peripancreatic tumors or lymph nodes may be difficult, even if acid-fast stain for exact sampling, it showed to be positive only in 33-41% of cases of abdominal tuberculosis[13],[14]. The polymerase chain reaction (PCR) of ascites was reported sometimes useful for those the bacterial cultures were negative[15]. Another diagnostic option involves initiating treatment with antituberculous drugs and evaluating the response of a tumor to this treatment.

Conclusion

Pancreatic tuberculosis should be kept in mind among the differential diagnosis of solitary masses in the pancreas, especially in young people in developing countries. Ascitic fluid ADA and LDH is useful and reliable biochemical test. Diagnostic laparoscopic examination and biopsy are useful for conformation of diagnosis and avoids unnecessary surgery.

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