Primary Pancreatic Tuberculosis: A Case Report

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Abstract

Abdominal tuberculosis is common but pancreatic tuberculosis is rare. Its occurrence may pose a diagnostic problem in differentiating it from inflammation and carcinoma of the pancreas. We report a case of 52 year old male who presented with clinical picture of acute pancreatitis. Ultrasound and CT scan showed picture of pancreatitis and pseudocysts. Fluid aspiration of pseudocyst showed acid-fast bacilli. Antikoch's treatment (AKT) was started and patient responded well.

Introduction

Tuberculosis of pancreas is very rare. However, nowadays the incidence is increasing because of increase in HIV infection or any other immunocompromised state or drug resistance.[1] The nonsurgical diagnosis of this entity continues to be a challenge, as it may present as pancreatitis or may mimick mass lesion i.e. malignancy. Investigations unless specific may be misleading. The disease, though potentially curable, if left untreated might prove to be fatal.[1] We present a case of pancreatic tuberculosis.

Case Report

A 52 year old male diabetic patient presented with acute pain in abdomen radiating to back, vomiting and loose motions since 6 days. There was no history of alcoholism or gall bladder disease. Abdominal examination revealed tenderness and guarding in umbilical and epigastric region. Bowel sounds were absent. Investigations showed total counts of 14000/ cumm with polymorphocytosis. Random blood sugar was 160 mg%. Serum amylase was 453 I.U. Erythrocyte Sedimentation Rate (ESR) was 56 mm at the end of 1 hour. Ultrasound was showing bulky pancreas. Computed Tomogram (CT) scan of abdomen was showing an increased density and stranding of the peripancreatic fat in the region of tail of pancreas with focal splenic lesions probable intra splenic pseudo cysts which were unusual (Fig.1, 2). There was splenic vein thrombosis. Hence a clinical diagnosis of acute pancreatitis was established. In view of his age and no aetiological factor in history, cystic malignancy of pancreas had to be ruled out. Hence, CT guided aspiration of the pseudo cyst was done which was positive for acid fast bacilli. Anti Koch's Treatment (AKT) on five drugs regimen of Cap. Rifampicin 450 mg(R), Tab. Isoniazid 300 mg (H), Tab. Ethambutol 800 mg (E), Tab. Pyrazinamide 1500 mg (Z) along with the Inj. Streptomycin 1 gm (S) was started and patient responded well within one month. Patient gained weight and his ESR came down. A repeat CT-scan was done after 3 months which showed the previous lesion at tail of pancreas regressed in size however peripancreatic fat stranding was persistent but minimal (Fig.3). After 3 months three drug regimen (HER) was continued till 9 months of regimen. After completing the AKT for 9 months CT scan was done, this showed previous lesions decreasing in size. His ESR came back to normal limits and he had gained weight. Follow up of one year has shown him to be disease and symptom free.

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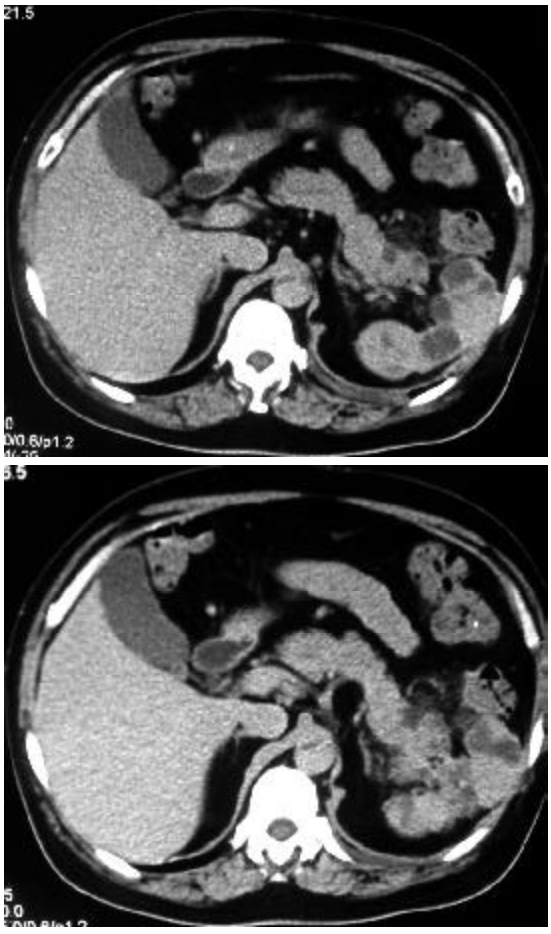
Discussion

The pathogenesis of isolated pancreatic tuberculosis is difficult in the absence of any



Figs. 1 and 2 : Computed Tomogram (CT) scan of abdomen showing irregularly marginated homogenously enhancing bulky pancreas with peripancreatic fat stranding in the region of tail of pancreas with multiple hypodense cystic lesions in the spleen.

detectable lesions in the other parts of the body. It is speculated that the tubercle bacilli reach the pancreas through haematogenous dissemination from an occult lesion in the lungs or abdomen. Secondly, the route by direct spread from contiguous lymph nodes may be responsible for most of the cases with isolated pancreatic tuberculosis. The third possible way is that dormant bacilli in an old tubercular lesion can reactivate in an immunosuppressive state.[1, 2]

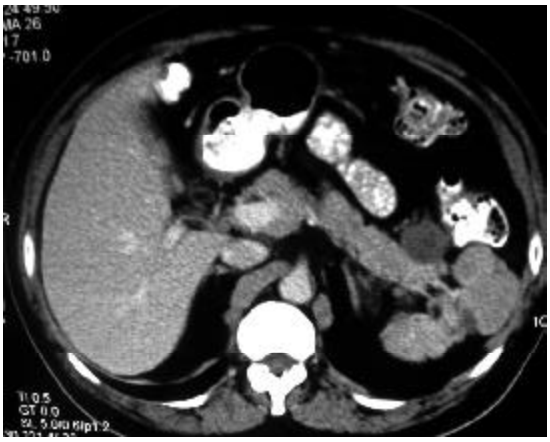


Fig. 3 : Computed Tomogram (CT) scan of abdomen showing normal pancreas with minimal fat stranding and resolving cystic lesions in the spleen.

In 1944, Auerbach reported that the pancreas is affected in 4.7% of cases of miliary tuberculosis, although the low incidence may have been due to under-diagnosis.[3] Paraf *et al* in 1966 reviewed autopsy studies of miliary tuberculosis between 1891 and 1961 and found only an incidence of 2.1% of pancreatic or peripancreatic lymph nodes.[4] Both the studies were done in patients with miliary tuberculosis. Isolated tuberculosis of pancreas is rare. Because of difficulty of obtaining the pathological evidence, it is very difficult to confirm the diagnosis, which is always misled by pancreatic neoplasms.[5, 6]

Pancreatic tuberculosis can present with symptoms of upper abdominal pain, pyrexia of unknown origin, ascites, abdominal mass, obstructive jaundice, acute or chronic pancreatitis, and upper gastrointestinal haemorrhage due to splenic vein thrombosis, diarrhoea and weight loss.[1,7] This makes it difficult to be diagnosed clinically.

Abdominal CT and ultrasound may show an enlarged pancreas with focal hypodense or hypoechoic lesion, usually in the head region, sometimes with irregular

multilobular cyst arising from pancreas.[7] However, these findings are nonspecific and simulate solid or cystic pancreatic neoplasms. The nonspecific clinical and laboratory investigations combination with pancreatic masses on CT scan frequently lead to an erroneous primary diagnosis of a carcinoma, cystadenocarcinoma or pseudocyst.[1, 5-7] In fact, there are no cardinal differences between radiologic appearances of cystic neoplasm and tuberculous abscess of pancreas; both of them have septa within mass, cyst with internal echos, and nearby hypodense lympha-denopathy.[7] To establish diagnosis image guided pancreatic biopsy is indicated. Endoscopic ultrasound with fine needle aspiration (FNA) for cytology is likely to become the preferred technique.[6] However, the aspiration of material from peripancreatic tumours or lymph nodes may be difficult; even if we get a biopsy the acid-fast stain for exact sampling is positive only in 33-41% of cases of abdominal tuberculosis. In view of age, of our patient and CT findings there was a need to rule out malignancy so FNA was needed. In our patient the CT guided aspiration of pseudocyst was positive for acid-fast bacilli (AFB).[1]

Early diagnosis will lead us to start with the Anti Koch's treatment which includes either four (Cap. Rifampicin 450 mg (R), Tab. Isoniazid 300 mg(H), Tab. Ethambutol 800 mg(E), Tab. Pyrizanamide 1500 mg(Z)) or five drug (RHZE and Inj Streptomycin 1 gm(S)) depending on the patient's status. Usually patient responds well and clinicoradiologically improves within 3-4 weeks. The period may vary between 6-12 months.[1,7-9] If patient does not respond then patient has to undergo surgery for aspiration or drainage of abscesses.[1, 6-9] Sometimes resection of a part of pancreas may be required. However, this must be the last resort. Usually patients respond well and need of surgery is rare.[9] Our patient also responded well to the AKT within 3-4 weeks on five drug regimen which was given for 3 months and remaining 6 months three drug regimen of RHE was given.

Follow up of patient includes monitoring patient's weight, ESR and ultrasound or CT scan to review for the changes in the lesions of pancreas. In our patient after completion of AKT of nine months, he gained weight and is asymptomatic.

Conclusion

Pancreatic tuberculosis should be considered in the differential diagnosis of focal pancreatic lesions, especially for people in developing countries and those with constitutional symptoms. The tuberculin skin test can be of diagnostic value. The clinician's high index of suspicion of tuberculosis and application of Fine needle aspiration (FNA) to obtain pathological evidence are extremely important to a correct diagnosis especially in young. Even radiological appearance may be simulating mass so a biopsy or culture is important. Provided diagnosis is established, anti-tuberculous chemotherapy together with aspiration or pigtail drainage may be sufficient. Surgery should be reserved either to establish diagnosis or to deal with associated complications.

References

1. Quanda Liu, Zhenping He, Ping Bie. Solitary pancreatic tuberculous abscess mimicking pancreatic cystadenocarcinoma: a case report. *Gastroenterology* 2003; 3 : 1.
2. Gupta MM, Wig JD, Suri AS. Tuberculosis of pancreas: a case report. *Ind J Tub* 1992; 39 : 247.
3. Auerbach O. Acute generalized military tuberculosis. *Am J Pathol* 1944; 20 : 121-36.
4. Paraf A, Menager C, Texier J, La Tuberculose du pancreas, *et al*. Tuberculose des ganglions de L'etage superior de L'abdomen. *Rev Med-Chir Mal Foie* 1966; 41 : 101-26.
5. Heitmann P, Kratsch B, Löschke S, Klempa I. Tuberculosis of the pancreas—a clinical rarity

Chirurg. 2001; 72 (6) : 739-41.

6. Woodfield, John C, Windsor A, Godfrey C, David A, Neil M. Diagnosis and Management of isolated Pancreatic Tuberculosis: Recent Experience and literature Review. *ANZ J Surgery* 2004; 74 (5) : 368-71.
7. D'cruz S, Sachdev A, Kaur L, Handa U, Bhalla A, Lehl SS. Fine needle aspiration diagnosis of isolated pancreatic tuberculosis: a case report and review of literature. *JOP J Pancreas (Online)* 2003; 4 (4) : 158-62.
8. Ioannis B, Athanasios S. Isolated Tuberculosis of pancreas. *JOP J Pancreas (Online)* 2004; 5 (3) : 155-58.
9. Feng Xia, *et al*. Tuberculosis of pancreas and peripancreatic lymph nodes in immuno-compromised states: Experience from China. *World J Gastroenterology* 2003; 9 (6) : 1361-64.

INITIAL COMBINATION THERAPY IN EARLY CROHN'S DISEASE

Hypothesis that combination immunosuppression with azathioprine and infliximab was superior to conventional therapy in patients with early Crohn's disease.

Patients intolerant of azathioprine in either group received methotrexate instead. At week 26, 60% of patients in the combined immunosuppression group were in clinical remission without corticosteroids or surgical resection (which was the primary endpoint) compared with 36% in the conventional group (a difference of 24.1%, 95% CI 7.3-40.8%).

Episodic therapy with infliximab leads to immunogenicity and is thus not an appropriate strategy. Recent practice guidelines suggest that azathioprine should be started with the first course of corticosteroids, and thus the conventional therapy group in D'Haens' trial may have underestimated the benefit of conventional therapy. However, the reality is that few, if any, clinicians routinely start azathioprine with the first course of corticosteroids.

These findings also emphasise the futility of corticosteroid therapy for most patients in the long term.

In patients with early rheumatoid arthritis, initial combination therapy with tapered high-dose prednisone and methotrexate or initial combination therapy with infliximab and methotrexate was more effective than sequential monotherapy or step-up combination therapy for preventing disease progression.

A definitive trial should compare azathioprine, a tumour-necrosis-factor antagonist such as infliximab, and the combination of the two, continued for at least 1 year in enough patients to assess safety, typical clinical endpoints (e.g., clinical response and remission, and steroid sparing), endoscopic healing, and hospitalization and surgery rates. The SONIC trial (Study of Biologic and Immunomodulator Naïve Patients in Crohn's Disease) has enrolled 500 patients with early Crohn's disease and randomly assigned them to azathioprine, infliximab monotherapy, and combination therapy with azathioprine and infliximab. If the preliminary data on initial combination therapy in early Crohn's disease reported by D'Haens and colleagues are confirmed, the treatment algorithm for patients with Crohn's disease will change.

William J Sandborn, The Lancet, 2008; 371 : 635-36.