INTRODUCTION
Pancreatic tuberculosis is a rare condition in immunocompetent patients and may present with protean manifestations which often makes it a diagnostic challenge for the clinician. In a review of 384 cases of abdominal tuberculosis, pancreatic involvement accounted for 32 (8.33%) of the cases.1 The nonsurgical diagnosis of this entity continues to be a challenge, as it may present as pancreatitis or may mimic cystic/mass lesion suggestive of malignancy.2-4 Performing of extensive surgical procedures for this often misdiagnosed medically treatable condition, is not uncommon and this underlies the importance of tissue diagnosis in these cases.5,6 Pancreas is an organ rarely affected by Mycobacterium tuberculosis. It is hypothesized that the pancreas is probably protected from tuberculous infection by the antibacterial effect of pancreatic enzymes including lipase and deoxyribonuclease which interfere with pancreatic seeding by Mycobacterium tuberculosis.2,6 Mechanisms of pancreatic infection have been attributed to pulmonary disease leading to lymphatic and haematogenous spread to the pancreas.5 Other mechanisms include ingestion of infected material from an active pulmonary lesion, reactivation of latent tuberculosis in the pancreatic focus, a toxic-allergic reaction of the pancreas involving an inflammatory response to mycobacterial antigens and direct extension from adjacent organs such as the lymph nodes.5,6

This case report demonstrates the possibility of managing a case of pancreatic tuberculosis as a case of pancreatic cancer despite adequate investigations again reminding that pancreatic tuberculosis can mimic pancreatic cancer.

CASE REPORT
AB is a 53 years old housewife who presented with 16 months history of recurrent epigastric pain that was burning, sometimes dull aching, and radiating straight to the back and inter-scapular area with variable response to meal and antacids. It however, was relieved by sitting and leaning forward but was usually worse in the night with poor sleep. She had no vomiting or change in bowel habit, no overt gastro-intestinal-tract (GIT) bleeding and no bleeding from any orifice. She had no jaundice, no change in the stool or skin color, no joint pains or swelling and nobody itching.

She had been loosing weight with low-grade intermittent fever and episodic excessive sweating. There was no blurring of vision, dizziness or syncopal attacks, no headaches, neck pains or stiffness. She noticed retrosternal chest pain that appears worse during the peak of the fever but no cough or difficulty in breathing. She had no palpitations, paroxysmal nocturnal dyspnoea, orthopnea or body swelling. She had noticed a gradually decreasing exercise tolerance and easy fatigue.

She had visited several hospitals for treatment during which she reacted to co-trimoxazole by cutaneous eruptions on the trunk and extremities. She never had surgery or blood transfusion, not on treatment for high blood pressure or diabetes. She had no contact with anybody with chronic cough or liver disease, no history of exposure to chemicals or ionizing radiations and no use of traditional medications or contraceptive pills. She had no history of cigarette smoking or ingestion of alcohol. Her travel history was mainly within the state...
and never outside. No family history of liver disease or malignancy. She was 3 years post-menopausal and had 13 children alive and well; last child birth was 19 years ago.

She was examined and found to be chronically ill looking, wasted with Body-Mass-Index of 23 kg/m². She was pale, anicteric with hyper-pigmented patches on the trunk, back and limbs, febrile with axillary temperature of 38°C with some dehydration.

Her pulse was 120 beats per minute; rest of the general physical, cardiovascular and neurological examination was unremarkable.

Her abdomen was distended uniformly with striae distensiae and a flat normal umbilicus. There was a mild epigastric tenderness with no rebound, definite masses, visceromegaly or demonstrable ascites. She had normal bowel sounds. Rectal and vaginal examinations were normal.

She was anemic with haemoglobin level of 8 gm/dL, WBC count of 6.5 x 10⁹/L with 42% neutrophils and 58% lymphocyte. The total platelet count was 140 x 10⁹/L and ESR was 100 mm after the first hour. Her clotting profiles were normal. She was not reactive to HIV 1 and 2 or HBsAg or HCV serologies. Her urea and electrolytes were normal, corrected calcium was 2.82 mmol/L with normal phosphate, magnesium and liver enzymes. The total serum protein was 67 g/L, serum albumin was 36 g/L with a globulin value of 31 g/L. Serum amylase was 1380 Caraway unit.

Mantoux test was negative. Her chest X-ray showed a globular heart with normal lung fields. X-ray of the dorsolumbar spine showed lytic charges in the bodies of T6 and T7 vertebrae with obliteration of the intervening disc space and paravertebral soft tissue shadows to suggest tuberculosis of the spine or a metastatic spinal disease.

Her abdominal CT showed enlargement in the region of the pancreatic tail with an irregular superior margin measuring 20 mm. There was a non-enhancing hypodense focus (1.9 x 1.0 cm) in the pancreatic tail. There was a simple renal cyst in the left kidney measuring 20 mm in diameter.

The patient was admitted with a diagnosis of pancreatic cancer. After several reviews, exploratory laparotomy was not carried out due to the possible vertebral involvement. Instead, she was given seven courses of Gemcitabine at 1000 mg/meter of body surface area.

During the course of treatment she developed a swelling at the back around T6,7 level which was progressive, painful and limited her movements. The swelling was soft but tender and was later aspirated and drained of 300 mLs of pus with a cavity that was packed. The aspirate did not grow any organism and no AFB was seen. Cytologically, it consisted of degenerated polymorphs and mono-nuclear cells in a necrotic background. Another similar swelling was also noted few weeks after the first and was similarly drained with same result, only that there were viable and degenerating polymorphs and debris but no malignant cells seen.

Following this lack of improvement, a review of the chest radiograph and CT was done with a strong suspicion of tuberculosis as the diagnosis. She was commenced on anti-TB medication for 12 months with complete healing and resolution of symptoms.

She had since fully recovered and is attending the follow-up clinic regularly after a total of 6 months of hospital admission.

**DISCUSSION**

The highest incidence of abdominal tuberculosis occurs in the gastrointestinal tract (GIT) and the peritoneum, followed by the mesenteric lymph nodes. Within the GIT, the ileocaecal area is the most common site of involvement. Disease affecting the upper GIT, liver and pancreas are uncommon.7 Patients with abdominal tuberculosis may have many symptoms which may mimic any disease.7 Therefore, if it is not clinically suspected, it may result in important morbidity and mortality and a high index of suspicion is required, especially in the high-risk population. In a study by Cho, the most common anatomic locations for pancreatic tuberculosis were the head (72.7%) followed by the tail (18.2%), and body (9.1%).8 In the patient, the lesion was located in the tail of pancreas.

The clinical presentation of pancreatic tuberculosis is slow and insidious, with non-specific symptoms and signs.5,8 As described by Xia et al., pancreatic tuberculosis typically occurs in young females who reside in tuberculosis endemic areas or who have a past history of pulmonary tuberculosis. This index case happened to be an older woman residing in a place with very high incidence of tuberculosis.9 The predominant symptoms consisted of abdominal pain (75%), anorexia/weight loss (69%), malaise/weakness (64%), fever and night sweats (50%), back pain (38%) and jaundice (31%), most of the patient presented with swelling of the head of the pancreas with heterogeneous attenuation echo was detected with ultrasound in 75% of patients.10 Computerized tomographic scan showed pancreatic mass with heterogeneous hypodense appearance in all patients, and calcification in 56% of patients,5 and peripancreatic nodules in 38% patients.9

Ultrasound of patients with pancreatic tuberculosis may show an enlarged pancreas with focal hypoechoic lesions and irregular borders, most commonly in the head region or enlarged pancreatic tissue and peripancreatic lymph nodes on CT.1,9 However, it should be noted that these findings are non-specific and may be seen in focal pancreatitis or pancreatic carcinoma.

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Recently, endoscopic ultrasound (EUS)-guided fine needle aspiration cytology (FNAC) has become the preferred method for histologic and bacteriologic confirmation. The microscopic features suggestive of tuberculosis are the presence of caseating granulomatous inflammation on histology and positive Ziehl Neelson stain or culture for acid fast bacilli.

Once the tissue diagnosis has been made, the management of TB rests on the medical treatment. The treatment of pancreatic tuberculosis comprises combination anti-tuberculous chemotherapy with rifampicin, isoniazid, pyrazinamide and ethambutol for 6 or 12 months depending on the response. The DOTS guidelines recommend only 6 months of therapy even for severe forms of tuberculosis.

Pancreatic involvement in abdominal tuberculosis or disseminated tuberculosis should be sought for early by careful clinical and laboratory evaluations. Attempt should be made to delay the treatment of pancreatic cancer to allow for exclusion of a tuberculous process because of the potential cure that can be obtained and not the exposure to the side effects of cytotoxics which may not be tolerated by some patients. A tissue diagnosis should be sought for early to aid in the diagnosis of pancreatic tuberculosis.

REFERENCES