Tuberculosis of the peripancreatic lymph nodes in an immunocompetent patient

Introduction

Tuberculosis of the pancreas is rare. Xia et al (2003) reported 16 cases of pancreatic tuberculosis in China. Of these cases, only six had peripancreatic lymph node involvement. This article reports a case of a patient who had tuberculosis of the peripancreatic lymph nodes without any pancreatic involvement, who proved to be a diagnostic challenge. This case highlights the difficulties in diagnosis of pancreatic tuberculosis because of the non-specific nature of its presentation.

Discussion

The authors were unable to find another case report of peripancreatic lymph node tuberculosis with no concomitant pancreatic involvement. Their search revealed that pancreatic tuberculosis may pose a diagnostic problem, presenting in a variety of ways. Evans et al (2000) summarized 38 cases, of which the immune status of 21 patients was undetermined and 10 known to be immunodeficient. They found that abdominal pain, malaise, fever, weight loss and transient jaundice were common presenting symptoms. Obstructive jaundice is a rare presentation of pancreatic tuberculosis (Dayananda Babu and John, 2001).

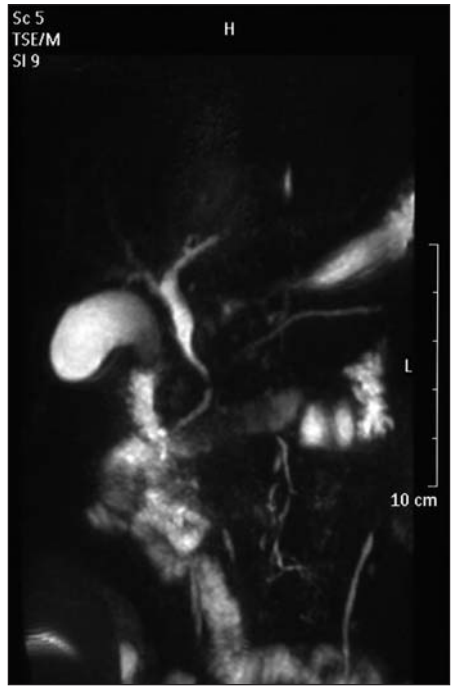
The most likely cause of abdominal tuberculosis is reactivation of previous disease. In this case, the patient did not report previous tuberculosis infection. As the presenting symptoms are non-specific, computed tomography is the investigation of choice in delineating any lesions within and around the pancreas (Sözbilen et al, 1992). However, it is sensible to obtain tissue samples to provide conclusive evidence of organ tuberculosis. Given the complexities in the diagnosis of pancreatic tuberculosis, it is not surprising that

Case Report

A 34-year-old man of Indian origin presented with a week's history of epigastric and upper abdominal pain associated with malaise and jaundice, but no fever, 2 months after returning from India. He also noticed pale stools and dark urine. He denied any weight loss, nausea and vomiting. He had no previous history of liver disease or jaundice and had had childhood bacillus Calmette–Guérin (BCG) vaccination. He was otherwise fit and well, and not on any regular medication. There was no history of recreational drug use and no family history of liver disease.

Blood tests at initial presentation showed a raised alkaline phosphatase of 1012 u/litre (range 80–260 u/litre), alanine aminotransferase 463 u/litre (range 0–37 u/litre) and bilirubin 120 μmol/litre (range 0–20 μmol/litre). Abdominal and respiratory examinations were unremarkable with no gross lymphadenopathy. At this stage, a biliary cause was suspected and an ultrasound scan was organized which showed a slightly dilated common bile duct. Magnetic resonance cholangiopancreatography showed a constant filling defect in the distal common bile duct but no obvious visible mass (*Figure 1*). Computed tomography scanning revealed no biliary dilatation but an irregular mass measuring 4x3 cm, inseparable from the upper aspect of the pancreatic head and the portal vein. There was also a 1.3 cm rounded density anterior to the pancreas close to the mesenteric vessels, showing low central attenuation (*Figure 2*). This mass was thought to be separate from the pancreas itself, even though the point of origin could not be accurately determined from the images, and enlarged lymph nodes were present. Exploratory laparotomy revealed enlarged rubbery lymph nodes in the lower omentum and around the portal triad. Pus was aspirated from these lymph nodes and sent to the laboratory, and neighbouring tissue biopsies were taken. The liver, spleen and peritoneum were normal, as was the pancreas on frozen section of trucut biopsy. Frozen section of the lymph nodes showed features consistent with tuberculosis. *Mycobacterium tuberculosis* was subsequently grown from the aspirated pus and tissue histology revealed evidence of granulomas. The patient was started on anti-tuberculosis therapy (isoniazid, rifampicin, pyrazinamide and pyridoxine) and referred to the respiratory physicians. The respiratory physicians continued the anti-tuberculosis medications, and added ethambutol, with a view to continuing this combination for a total of 2 months. After this period, the patient continued to take pyridoxine and rifampicin for 6 months. To date, the patient continues to be fit and well.

Figure 1. Magnetic resonance cholangiopancreatography showing filling defect distal to the common bile duct and no obvious mass.



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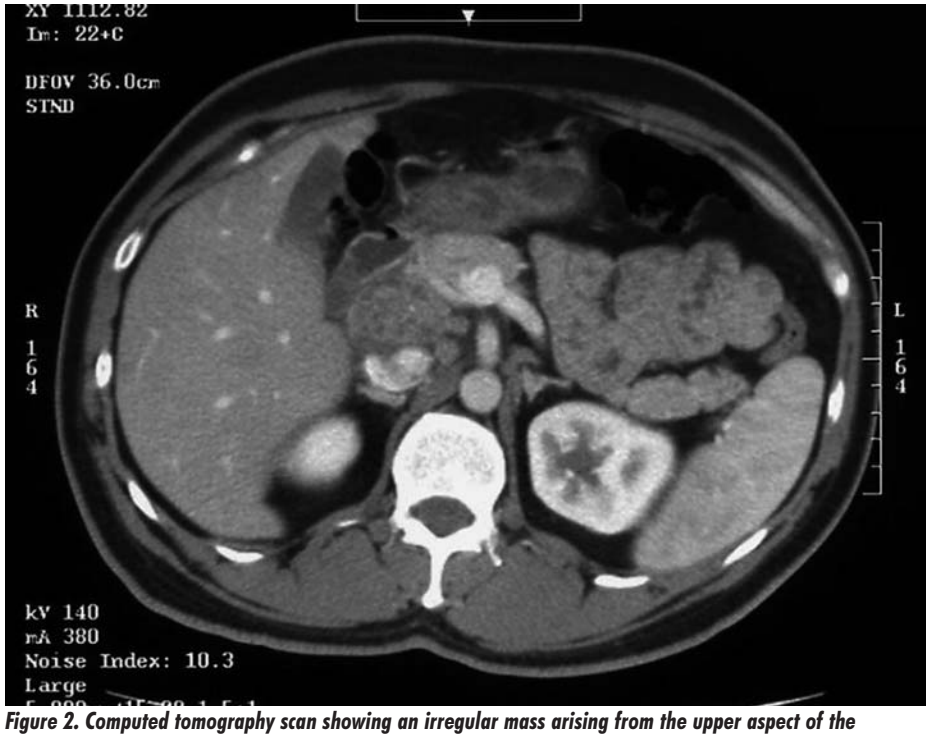

Figure 2. Computed tomography scan showing an irregular mass arising from the upper aspect of the pancreas and the portal veins with enlarged lymph nodes.

delay in diagnosis may lead to an adverse outcome (Stambler et al, 1982). Pancreatic tuberculosis is a potentially treatable cause of a mass lesion in the pancreas and rarely, obstructive jaundice. Thus, in order to maximize patient outcome and to provide appropriate treatment, one must consider pancreatic tuberculosis as a differential diagnosis in patients who present with an appropriate history. BJHM

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Fungating giant umbilical endometrioma

A 41-year-old woman presented with a periumbilical lump. On inspection it appeared to be a polypoid mass, clear of her Pfannenstiel caesarean scar. The lump bled episodically coinciding with her menses. Computed tomography demonstrated a 5 cm x 5 cm x 6.5 cm mass in the anterior abdominal wall. At surgery the mass was excised and histology confirmed the diagnosis of an endometrioma. *Figure 1* shows the lesion in situ.

Discussion

Approximately 0.5–1% of patients with endometriosis develop umbilical endometriomas. They tend to occur in women between 30 and 40 years of age. Umbilical endometriomas usually consist of a solitary firm, brown or blue nodule ranging from 0.5–3 cm in size. Symptoms of itching, bleeding or pain tend to be cyclical (Schachter et al, 2003). Cutaneous endometriomas are often associated with scars but in this case there was no prior surgical scar connected to this lesion. There is also an association with paraumbilical hernias (Khetan et al, 1999). Treatment is by local excision (Igawa et al, 1992). BJHM

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Figure 1. An umbilical endometrioma in situ.