Tuberculous lymphadenitis as a cause of obstructive jaundice: A case report and literature review

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Abstract

Obstructive jaundice secondary to tuberculosis (TB) is extremely rare. It can be caused by TB enlargement of the head of the pancreas, TB lymphadenitis, TB stricture of the biliary tree, or a TB mass of the retroperitoneum. A 29-year-old man with no previous history of TB presented with abdominal pain, obstructive jaundice, malaise and weight loss. Ultrasonography (US), computer tomography (CT) scan and endoscopic retrograde cholangiopancreatography (ERCP) were suggestive of a stenosis of the distal common bile duct (CBD) caused by a mass in the posterior head of the pancreas. Tumor markers, CEA and CA19-9 were within normal limits. HBsAg and HCV were negative. Abdominal ultrasonography (US) revealed a semi-solid hypoechoic lesion 39 mm × 40 mm in size around the head of the pancreas, with two enlarged lymph nodes lying above this, and a common bile duct measuring 10 mm in diameter. Computer tomography (CT) scan showed a low density mass on the posterior aspect of the head of the pancreas with a contrast enhancing solid-rim (Figure 1). Pancreatography was normal, however severe narrowing of the distal common bile duct (CBD) was seen on endoscopic retrograde cholangiopancreatography (ERCP) (Figure 2A and B).

INTRODUCTION

Abdominal tuberculosis (ATB) is rare and obstructive jaundice caused by tuberculosis (TB) is extremely rare. ATB can mimic more common noninfectious abdominal syndromes and is often overlooked because of its low incidence. The mechanisms by which ATB causes bile duct obstruction are varied. We describe a patient with biliary obstruction caused by enlarged tuberculous lymph nodes.

CASE REPORT

A 29-year-old man presented to our unit with epigastric pain and tenderness on examination, and jaundice, steatorrhea, malaise and weight loss of 7 kg over the preceding 6 mo. Total bilirubin was 163 µmol/L and direct bilirubin 88 µmol/L; SGOT, SGPT, gamma GT and alkaline phosphatase were moderately elevated. Other laboratory tests including the tumor markers CEA and CA19-9 were all within normal limits. HBsAg and HCV were negative. Abdominal ultrasonography (US) revealed a semi-solid hypoechoic lesion 39 mm × 40 mm in size around the head of the pancreas, with two enlarged lymph nodes lying above this, and a common bile duct measuring 10 mm in diameter. Computer tomography (CT) scan showed a low density mass on the posterior aspect of the head of the pancreas with a contrast enhancing solid-rim (Figure 1). Pancreatography was normal, however severe narrowing of the distal common bile duct (CBD) was seen on endoscopic retrograde cholangiopancreatography (ERCP) (Figure 2A and B).
The patient underwent open surgery, and at operation the liver was found to be slightly firm, gallbladder moderately dilated, Lund’s lymph node enlarged (about 1.5 cm), common bile duct moderately dilated and two lymph nodes close to the common hepatic artery also enlarged (2 cm and 3 cm). After mobilizing the duodenum and the head of the pancreas, an enlarged (4 cm) soft lymph node adherent to the distal CBD, was removed. The lymph node had a solid surface with a soft and caseous centre, and had a fistulous connection with the posterior aspect of the CBD. Frozen section histology of the lymph node revealed chronic granulomatous inflammation. The gallbladder, Lund’s lymph node, the two other enlarged lymph nodes lying close to the common hepatic artery, and a specimen of liver was removed and sent for histology. The narrowed distal CBD was resected, the distal end over sewn, and the proximal end anastomosed with a Roux-en-Y jejunal limb. The resected specimen included the fistulous opening on the posterior wall of the CBD (Figure 3).

The patient had an uneventful postoperative recovery, and bilirubin levels normalised within two weeks. Histology of the liver and gallbladder was normal. The resected CBD showed epithelial ulceration and inflammation with a number of necrotizing granulomata. The lymph nodes had a chronic granulomatous appearance with large merged necrotic areas, and smaller epitheloid-type granulomata with occasional multinuclear giant cells (Figure 4A and B) suggestive of tuberculous lymphadenitis. The diagnosis was confirmed with a polymerase chain reaction (PCR) using automated analyzer Cobas/Roche/, with the Amplicor Mycobacterium tuberculosis assay. No previous specific risk for TB was found in the patient. He was treated with anti-tuberculous quadruple therapy and achieved gradual clinical improvement, with resolution of pain and malaise, and a weight gain of 10 kg over the next 6 mo. He remained well at 2.5 years postoperatively.

**DISCUSSION**

Obstructive jaundice secondary to abdominal TB is extremely rare. Four mechanisms have been described: TB of the pancreas itself may cause pseudoneoplastic obstructive jaundice; it may be secondary to TB lymphadenitis causing compression and inflammation of the lymph nodes and the CBD, as in our case, with caseation of the lymph node causing fistulation into the CBD; biliary TB itself may lead to single or multiple
structures, mimicking cholangiocarcinoma, and TB can create a retroperitoneal mass leading to biliary tree obstruction.

The diagnosis of abdominal TB should be considered in the context of a mass in the head of the pancreas in the immunocompromised patients and in countries with endemic TB, after the exclusion of malignancy and other biliary inflammation. TB lymphadenitis can be suspected when a contrast-enhanced CT scan demonstrates low density masses surrounded by an enhancing solid rim, or when ERCP demonstrates a normal pancreatogram with a smooth narrowing of the CBD, as were seen in our patient. FDG-PET scanning has not been shown to be useful in distinguishing TB from pancreatic malignancy, as both conditions have an increased uptake of the FDG metabolite. US or CT-guided percutaneous fine needle aspiration (FNA) of the enlarged lymph nodes may be useful, but is often not definitive. Cytology of CBD aspirate, however, obtained by ERCP, may be confirmatory in the presence of the acid-fast bacillus (Mycobacterium tuberculosis); alternatively PCR of the aspirate may be diagnostic. However, in the case of a periportal lymphadenopathy causing obstructive jaundice, as in our patient, these FNA tests are only positive if a fistula exists between the TB lymph node and the CBD, allowing bacilli to pass into the CBD. Other potential diagnostic methods include obtaining tissue specimens by laparoscopy or endoscopic ultrasound with FNA. Though in practice, the diagnosis is often established at operation or even after surgery by histology or PCR-based assay, as was the case in our patient.

The great benefit of a preoperative diagnosis of TB causing the obstruction is that a more conservative path could be followed, involving removal of the obstructing lymph node alone, followed by anti-TB medications. In our case more elaborate CBD resective surgery was undertaken for presumed malignancy.

However, even though TB lymphadenitis was suspected in our patient after intraoperative frozen section, resection of the involved part of the CBD was necessary as the bile duct was already strictured, and eventual closure of the fistula would probably result in additional stenosis or even complete obstruction of the CBD. Thus inexplicable stenosis of the CBD should be taken into consideration in the context of pancreatic or TB lymphadenitis associated with obstructive jaundice and be treated by biliary bypass surgery in addition to anti-TB medication.

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