

## Abdominal tuberculosis presented with obstructive jaundice: A case report.

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**Sriussadaporn S, Lerdlum S. Abdominal tuberculosis presented with obstructive jaundice:  
A case report. Chula Med J 1994 Aug;38(8): 479-484**

*A case of abdominal tuberculosis presented with obstructive jaundice is reported. The tuberculous tubercles involved the liver, gall bladder, mesentery, peritoneum and lymph nodes. Obstructive jaundice was caused by external compression of the common bile duct from the surrounding enlarged lymph nodes. Surgical management included cholecystectomy and exploration with T-tube drainage of the common bile duct. Antituberculous therapy included isoniazid 300 mg/day and rifampicin 450 mg/day for 12 months, and pyrazinamide 1.5 gm/day during the first 2 months. The T-tube was removed after completion of the antituberculous therapy and when the T-tube cholangiography demonstrated free flow of contrast material into the duodenum.*

**Key words:** Abdominal tuberculosis, Obstructive jaundice.

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Received for publication: August 24, 1994.

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สุวิทย์ ศรีอัมภพร, สุกัลยา เลิศล้ำ. วัณโรคในช่องท้องที่ทำให้เกิดการอุดตันของท่อน้ำดี : รายงานผู้ป่วย 1 ราย. จุฬาลงกรณ์เวชสาร 2537 สิงหาคม; 38(8): 479-484

รายงานผู้ป่วย 1 ราย ที่เป็นวัณโรคภายในช่องท้องและมารักษาที่โรงพยาบาลจุฬาลงกรณ์ด้วย เรื่องตาเหลือง ตัวเหลือง เนื่องจากท่อน้ำดีอุดตัน อวัยวะในช่องท้องที่ติดเชื้อวัณโรคได้แก่ ตับ, ถุงน้ำดี, เยื่อ ぶช่องท้อง, mesentery และ ต่อม้ำเหลือง ภาวะท่อน้ำดีอุดตันเกิดจากต่อม้ำเหลืองที่มีขนาดโตขึ้นไปกด ท่อน้ำดีร่วม (common bile duct) ผู้ป่วยได้รับการตัดถุงน้ำดีและใส่ท่อระบายท่อน้ำดีร่วม (T-tube drainage) หลังผ่าตัดผู้ป่วยได้รับยารักษาวัณโรค isoniazid 300 มิลลิกรัม ต่อวัน และ rifampicin 450 มิลลิกรัม ต่อวัน เป็นเวลา 12 เดือน และได้รับ pyrazinamide 1.5 กรัม ต่อวัน ในระยะ 2 เดือนแรก เมื่อผู้ป่วยได้รับยา รักษาวัณโรคจนครบกำหนดและตรวจท่อน้ำดีโดยการฉีดสารทึบแสงผ่านทางท่อระบายน้ำดีร่วม (T-tube cholangiography) พบว่าการอุดตันของท่อน้ำดีหายไปแล้ว จึงค่อยดึงท่อระบายท่อน้ำดีร่วมออก หลังจาก ดึงท่อระบายท่อน้ำดีร่วมออก 1 เดือน ผู้ป่วยมารับการตรวจร่างกาย พบว่าสุขภาพสมบูรณ์แข็งแรงดี

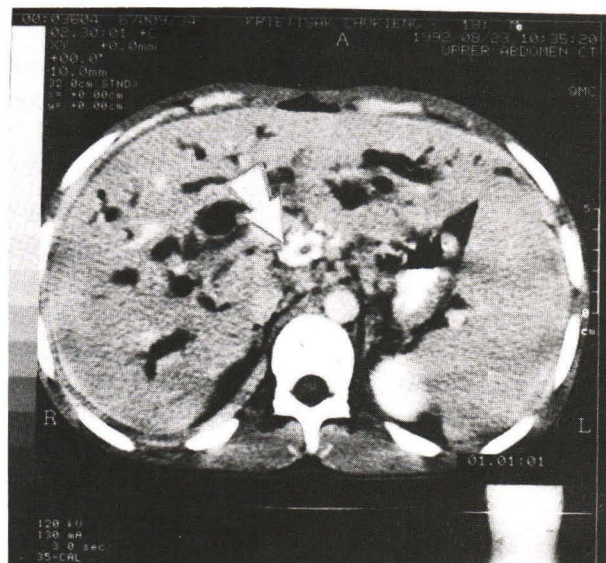
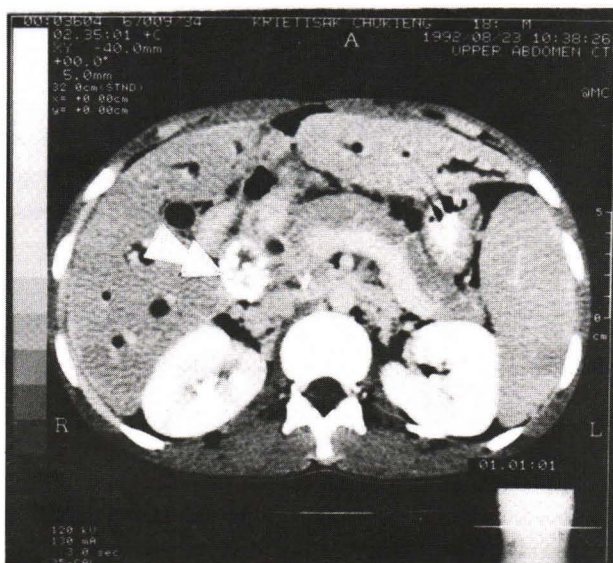
Abdominal tuberculosis may be presented with a wide variety of clinical spectra. Prolonged fever, gastrointestinal obstruction and malabsorption are among the common manifestations. Presentation with obstructive jaundice is rare. We report a case of obstructive jaundice from abdominal tuberculosis which was successfully treated by T-tube drainage of the common bile duct and a complete course of antituberculous drug therapy.

### A case report

An 18-year old man was admitted to Chulalongkorn Hospital on 25 August 1992 because of fever and progressive jaundice for 1 month. On physical examination, the blood pressure was found to be 110/70 mmHg, the pulse rate was 100 beats/min and the body temperature was 38° c. He had markedly icteric sclera. The heart and lungs were within normal limits. Abdominal examination re-

vealed ascites. The liver was enlarged 2 fingers breadth below the right costal margin with smooth surface, rubbery in consistency and slightly tender. Rectal examination was normal.

Laboratory tests were as follows: hemoglobin 11.5 gm/dL, white blood cell count 8,900/mm<sup>3</sup> and platelet count 460,000/mm<sup>3</sup>; urinalysis:pH 6, specific gravity 1.015, sugar(-), protein(-), ketone(-), bilirubin(+), urobilinogen (-); urine sediment: red blood cell negative, white blood cell negative, bacteria(1+); blood chemistries: total bilirubin 11.7 mg/dL, direct bilirubin 6.7 mg/dL, aspartate aminotransferase 480 u/L, alkali phosphatase 5,620 u/L (normal 98-279 u/L), albumin 3.4 gm/dL, globulin 4.6 gm/dL, BUN 7 mg/dL, creatinine 0.7 mg/dL, prothrombin time 13.2 seconds (control 11.9 seconds), activated partial thromboplastin time 34 seconds (control 22.6 seconds), HBs-antigen(-), Anti HAV IgM (-).



**Figure 1 and 2.** Computed tomography of the abdomen reveals dilatation of the intrahepatic bile ducts and proximal common bile duct. Calcification of the lymph nodes at portahepatis and celiac regions (arrows) are noted.

Chest X-ray was normal. Sputum examination revealed no tubercle bacilli. Hemoculture was negative for microorganisms. Computed tomography of the abdomen showed hepatosplenomegaly with dilatation of the intrahepatic bile ducts and proximal common bile duct. There was multiple calcification at the portahepatis, celiac and mesenteric regions suggesting calcified lymph nodes (figures 1 and 2). An upper gastrointestinal study revealed a deformity of the post-bulba part of duodenum with nodular lesions. Faint calcification was noted in the soft tissue posterolateral to the duodenal lesion, thought to be calcified lymph nodes (figure 3). Gastroscopy disclosed multiple ulcerating lesions at the second and third parts of the duodenum. Histopathology of the duodenal biopsy showed lymphoid follicles in the lamina propria compatible with chronic inflammation.

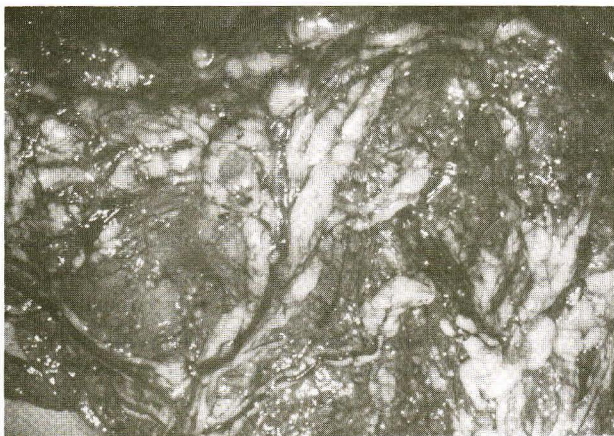
Exploratory laparotomy revealed hepatosplenomegaly with multiple yellowish nodules of 0.5 to 1 cm. in diameter distributed over the liver surface, omentum, mesentery and parietal peritoneum (figures 4,5 and6). The gall bladder was contracted. The hepatoduodenal ligament was shortened and thickened, and surrounded with inflammatory exudative reactions. The duodenum appeared normal. The celiac and paraortic lymph nodes were also enlarged. A cholecystectomy was performed. Common bile duct exploration revealed obstruction at the distal end due to the external compression of the enlarged lymph nodes. A 16 french-T-tube was subsequently inserted into the common bile duct and the abdomen was closed after biopsies of the liver, lymph nodes, omentum and peritoneum were performed.



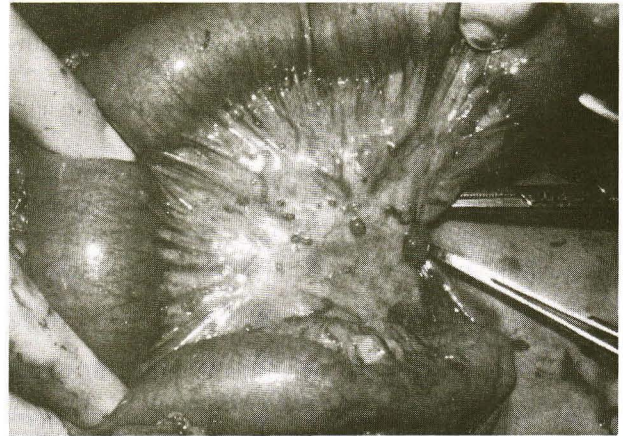
**Figure 3.** Upper gastrointestinal study reveals deformity of the post-bulba part of duodenum (arrows). Faint calcification is noted in the soft tissue posterolateral to the duodenal lesion (arrows).

The histopathology revealed multiple non-caseating granulomas with giant cells of the lymph nodes and gall bladder. Acid-fast bacilli (AFB) were not detected on specially stained sections. The diagnosis of abdominal tuberculosis was established. The treatment planning was 300 mg/day of isoniazid and 450 mg/day of rifampicin for 12 months and 1.5 gm/day of pyrazinamide during the first 2 months.

The patient recovered well. A T-tube cholangiography performed on day 7 post-operation revealed an extrinsic pressure effect causing incomplete obstruction of the common bile duct (figure 7). T-tube cholangiography performed at 4 months post-operation disclosed near normal intrahepatic bile ducts, but partial obstruction of the common bile duct was still noted (Figure



**Figure 4.** Tuberculous tubercles at the omentum.

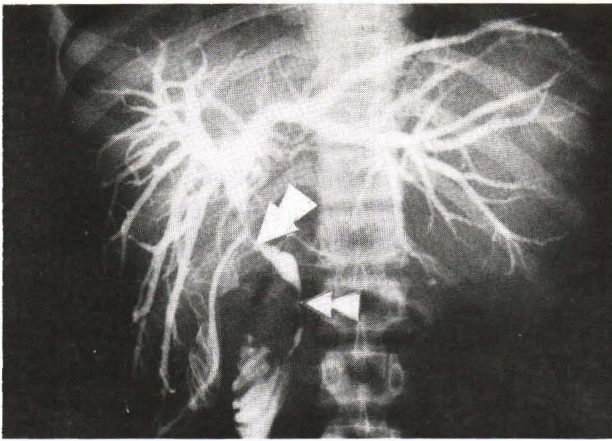


**Figure 5.** Tuberculous tubercles at the mesentery.

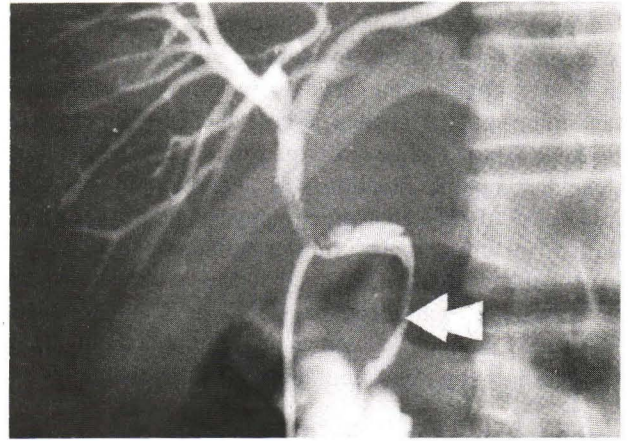


**Figure 6.** Tuberculous tubercles at the liver.

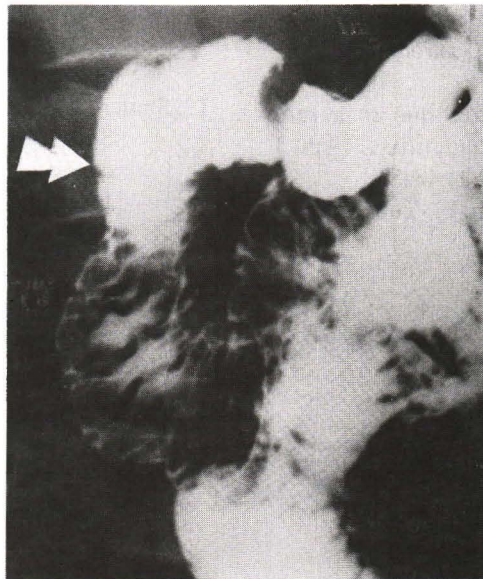
8). An upper gastrointestinal study performed at 8 months post-operation revealed a marked improvement of the duodenal lesions compared to previous observations, however, a fixed contraction and deformity of the post-bulbar region was still observed (figure 9). At 13 months post-operation (1 month after cessation of antituberculous drugs), T-tube cholangiography revealed normal appearance of the intrahepatic bile duct with free flow of the contrast material into the duodenum. The liver function test was normal and the patient was in good condition. The T-tube was then clamped and subsequently removed two days later. One month after that the patient was seen at the out-patient clinic and was in normal condition.



**Figure 7.** T-tube cholangiography performed 7 days post-operation shows extrinsic pressure effect upon the common bile duct (arrows).



**Figure 8.** T-tube cholangiography performed 4 months post-operation shows near normal intrahepatic bile ducts, partial obstruction of the common bile duct is still noted (arrows).



**Figure 9.** Upper gastrointestinal study 8 months post-operation reveals a markedly improvement of the duodenal lesion compared to previous study (arrows).

## Discussion

Intraabdominal tuberculosis presented with clinical signs of obstructive jaundice is rare. Extrahepatic biliary obstruction from tuberculous infection may be due to lymphadenopathies in the portahepatis or hepatoduodenal ligament,<sup>(1-3)</sup> or tuberculosis of the pancreas.<sup>(4,5)</sup> The diagnosis is often established from histopathology obtained during the operation for relieving obstructive jaundice. Intraoperatively, the findings may not be able to differentiate from carcinomatosis. Histopathologic demonstration of caseous necrosis and/or mycobacterium tuberculosis are diagnostic. Before the chemotherapy era, 70 per cent of patients with advanced pulmonary tuberculosis developed gastrointestinal tuberculosis due to the infectious secre-

tions which they swallowed. Although it is assumed that present-day cases are also due to swallowed infectious secretions, coexistent evidence of pulmonary tuberculosis is actually present in less than 50 per cent of the cases and in some series is as little as 25 per cent.<sup>(6-8)</sup>

Decompression of the biliary tract must be done to prevent further damage to the liver. If the common bile duct is obstructed at the distal end by the enlarged lymph nodes or pancreas, biliary-enteric bypass may be done by either cholecystojejunostomy or choledochojejunostomy.<sup>(4-5)</sup> However, when the lesions involve the portahepatis or hepatoduodenal ligament, biliary-enteric bypass may be a dangerous procedure or even impossible due to the dense inflammatory process in these areas. Common bile duct

drainage with a T-tube is recommended because it is a simple procedure which can shorten the operation time in a chronically ill patient. In addition, biliary drainage with a T-tube obviates the need to perform biliary-enteric anastomosis of the infected tissues which poses high risk of leakage. The usefulness of T-tube drainage of the common bile duct in this situation was supported by previous reports.<sup>(3,9)</sup> T-tube cholangiography should be performed at 2 to 3 month intervals during the antituberculous therapy. The T-tube should be clamped and removed a few days later when the antituberculous therapy is completed (usually 9 to 12 months)<sup>(10-12)</sup> and when T-tube cholangiography reveals no further obstruction of the biliary tract.

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