Tuberculous lymphadenopathy at the porta hepatis: a rare cause of obstructive jaundice

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Abstract: Tuberculosis is still one of the most common infectious diseases in many parts of the world. Although any part of the abdomen can be involved, lymph nodes at the porta hepatis are a rare site. A 38-year-old male presented with obstructive jaundice secondary to tuberculous lymphadenopathy at the porta hepatis. The clinical and radiological data suggested malignant obstruction. Laparoscopy was diagnostic of abdominal tuberculosis which was confirmed by histology. We emphasize the rarity of the presentation, the difficulty of establishing the diagnosis based on clinical, laboratory and/or radiological data and the need for tissue biopsy. We also highlight the central diagnostic role of laparoscopy to avoid unnecessary laparotomy.

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The incidence of tuberculosis (TB) is increasing worldwide. Abdominal TB accounts for 10-30% of extrapulmonary TB. Although TB can involve any part of the abdomen, the peritoneum and ileocecal region are the commonest sites. We report a patient with obstructive jaundice secondary to tuberculous lymphadenopathy at the porta hepatis. The unusual presentation and the rarity of TB as a cause of obstructive jaundice prompted this report. To the best of our knowledge, this is the first such reported case from Saudi Arabia.

Case report A 38-year-old Filipino male presented with progressively increasing jaundice of 7 days duration. The patient also had mild generalized abdominal pain, anorexia and recent weight loss of 5 kg. He denied drinking raw milk, and being in contact with tuberculous patients or animals. The patient had been perfectly well apart from hypertension, diagnosed one year ago, but he was not on any medication. Physical examination was unremarkable except for considerable recent weight loss and deep jaundice.

Total and differential white blood cell counts were normal. The erythrocyte sedimentation rate was 30 mm/first hour. HBsAg was reactive but other serologic tests for hepatitis A and C were negative. Sickle cell test, and serology for human immunodeficiency virus, amebiasis and hydatid disease were also negative. Carcinoembryonic antigen and α-feto protein were normal. Liver function tests showed (normal ranges between brackets) total bilirubin 24.4 mg/dl (0.1-1.0), direct bilirubin 19.1 mg/dl (<0.4), and alkaline phosphatase 317 IU/L (50-140). The prothrombin and partial thromboplastin times were moderately prolonged.

Postero-anterior chest and abdominal roentgenograms were normal. Abdominal ultrasonography showed a non-shadowing echogenic mass with calcification at the porta hepatis and the gallbladder fossa. The gallbladder was not visualized. The common bile duct was normal, but the intrahepatic ducts were moderately dilated. Computerized tomography (CT) scan of the abdomen with contrast showed multiple non-enhanced irregular hypodense lesions with dense calcification in the quadrate lobe. There was a lobulated mass at the porta hepatis (Fig. 1). A CT-guided biopsy of the liver mass was taken, but the material was inadequate for histological interpretation. Percutaneous transhepatic cholangiogram (PTC) showed normal common
bile duct, marked narrowing of the common hepatic duct due to extrinsic mass in the lateral aspect of the duct; the intrahepatic ducts appeared moderately dilated (Fig. 2). A placement of a percutaneous biliary stent was attempted but failed. Obstructive jaundice most probably due to malignancy was suspected.

Laparoscopy was then performed. There was no ascites. Multiple small tubercles were seen on the parietal peritoneum and falciform ligament. There were multiple masses in the right lobe of the liver and on the wall of the gallbladder, with multiple nodes at the porta hepatis. The small and large intestines were grossly normal. There was no apparent mesenteric lymphadenopathy. Multiple biopsies of the tubercles from the parietal peritoneum were taken. Frozen section showed granulomas with caseation and the diagnosis of TB was confirmed by paraffin section (Fig. 3). However, the Ziehl-Nielsen stains and peritoneal culture for acid fast bacilli were both negative. A triple anti-TB therapy was initiated and the patient showed a remarkable improvement after 6 weeks.

**Fig 1:** Lower cuts of CT scan of the liver showing the lobulated mass at the porta hepatitis (arrows).

**Fig 2:** PTC showing extrinsic mass effect on the lateral aspect of the common hepatic duct (arrow), with moderate dilatation of the intrahepatic ducts.

**Fig 3:** Photomicrograph of the parietal peritoneum showing a granulomatous lesion with central caseation. (Hematoxylin & Eosin stain 40x).
Discussion

Abdominal pain is the commonest presenting feature of abdominal TB. However, in our patient, jaundice was the dominant clinical entity, with abdominal pain being mild. This is noteworthy in light of the observed extensive involvement of the peritoneum, liver and gallbladder, in addition to the grossly enlarged lymph nodes at the porta hepatis. Thus, the mild abdominal pain and the absent constitutional symptoms, such as abdominal distension, fever and night sweats, can be misleading. It is also worth mentioning that the physical examination was normal except for cachexia and deep jaundice.

Tuberculosis of the liver is of two main types: diffuse and localized. The diffuse form is a common finding in patients with miliary TB. Localized hepatic TB is rare and can present as an abscess or a macronodular lesion. TB of the gallbladder is very rare. Bergdahl and Boquist in 1972 found only 44 cases of tuberculous cholecystitis.

Lymph nodes at the porta hepatis are also rarely involved by TB. At first sight, this is surprising, since TB lymphadenopathy is the commonest form of extrapulmonary TB based on our experience and other reports. However, we speculate that it may be explained by the rarity of TB of the gallbladder and extrahepatic biliary tree which drain to the porta hepatis.

Obstructive jaundice due to TB is rare. Leader, in a review of the world literature in 1952, found jaundice to be rare in patients with TB of the liver or gallbladder. In a report of 881 cases of abdominal TB from Nigeria, Ihekwaba in 1993 did not find a single case of jaundice. In contrast with these reports, Alvarez and Carpio from the Philippines found jaundice in 33% of patients with hepatobiliary TB.

Review of the recent English literature revealed only five case reports of obstructive jaundice due to tuberculous lymphadenopathy at the porta hepatis (Table 1). In all the cases, the diagnosis of TB was not entertained before laparotomy, laparoscopy or autopsy.

Procedures required for tissue diagnosis in such cases include ultrasound- or CT-guided aspiration and biopsy, laparoscopy or laparotomy. Kawamori et al from Japan reported directly to laparotomy in a patient with a past history of pulmonary TB who presented with a macronodular lesion of the liver. They carried out segmental hepatectomy, and histology showed TB. In our patient, CT-guided biopsy was inadequate for histological diagnosis. We therefore, performed laparoscopy and found it diagnostic. The role of laparoscopy in the management of abdominal TB has been persuasively argued by others.

This report is of interest because it has three messages: (i) TB must be kept in mind as a cause of obstructive jaundice, (ii) laparoscopy should replace diagnostic laparotomy in the management of patients with obstructive jaundice in whom the diagnosis could not be established by radiology or endoscopy or both, and (iii) it underlines the necessity for tissue diagnosis in similar cases of obstructive jaundice in order to treat a potentially curable disease.

References

8. Leader SA. Tuberculosis of the liver and gall-bladder with abscess formation: A review and case report. Ann Intern


الخلاصة:

الدرن أحد الأمراض الشائعة في كثير من أنحاء العالم. على الرغم من إن إصابة أي جزء في البطن بهذا المرض أمر ممكن إلا أنه يندر إصابة العقد اللمفاوية في نفير الكبد. كان هناك مريض يبلغ من العمر 38 عامًا أصيب ببرقان إنسدادي ناجح عن تدريج لمفاوي في نفير الكبد.

كانت البيانات السريرية والإشعاعية تؤكيدًا لوجود إنسداد خبيث، غير أن الفحص المنظاري لتجويف البطن أشار إلى وجود درن بطني أكاد الفحص المجهرى للخزعة النسيجية. إذا نُرتب على ندرة هذه الحالة صعوبة تشخيصها بناءً على البيانات السريرية والمجهرية و/أو الإشعاعية وعلى أهمية خزعة النسيج. كما إذا نُرتب على الدور التشخيصي للفحص المنظاري لتجويف البطن تجنب لإجراء عملية إستكشافية للتجويف البطني.