Isolated Tuberculosis of the Gallbladder

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Citation

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Abstract

A 26-year-old HIV-positive female patient presented with acute acalculous cholecystitis. An abdominal CT scan showed a thickened gallbladder wall, pericholecystic fluid and porta hepatis lymphadenopathy. A laparoscopic cholecystectomy was done and histology of the gallbladder revealed granulomatous inflammation, and Ziehl-Neelson staining was positive for acid-fast bacilli. In the HIV/AIDS era, tuberculosis of the gallbladder should be a differential diagnosis in patients presenting with acute or chronic cholecystitis in the presence or absence of gallstones. An accurate pre-operative diagnosis cannot be made, and the literature review highlights that most cases are diagnosed post cholecystectomy or at post-mortem. The routine histopathological examination of all cholecystectomy specimens should therefore be standard practice.

INTRODUCTION

The resurgence of tuberculosis (TB) in South Africa has largely paralleled the HIV/AIDS pandemic with extrapulmonary TB being a common presentation to the general surgeon. Notwithstanding the advances in imaging techniques, the uncommon sites of abdominal tuberculosis continue to present diagnostic challenges[1]. Not uncommonly, the diagnosis is made following surgery. We report a rare case of tuberculosis of the gallbladder to highlight the protean presentation of this multisystemic disease and the clinical challenges this condition presents.

CASE PRESENTATION

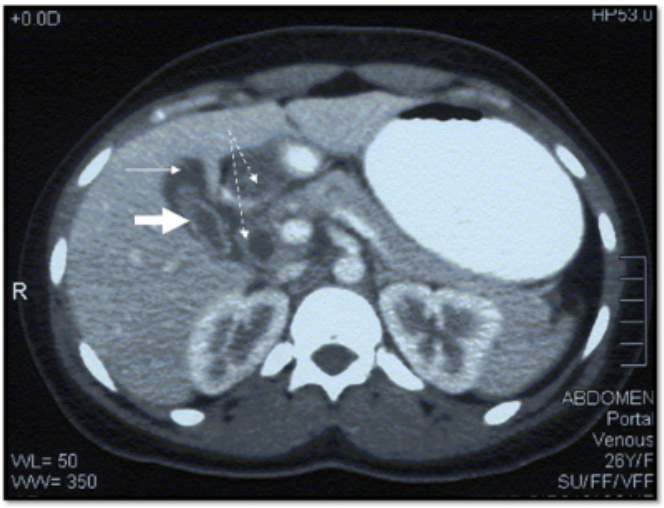
A 26-year-old known HIV-positive patient presented with a two-week history of right upper quadrant pain and dyspepsia. The patientâ™s previous history was non contributory, with no prior history of TB. On examination, the patient was noted to be well nutritioned, afebrile and mildly jaundiced. The cardiopulmonary examination was normal. The abdominal examination revealed mild right upper quadrant tenderness without peritonism.

The hemoglobin concentration was 12.4g/dL, the white cell count was $6.1x10^9$/L and the platelet count $247x10^9$/L. The electrolytes and renal function were normal. The liver function tests showed the serum bilirubin to be 59Âµmol/L (normal up to 17Âµmol/L) and alkaline phosphatase 379 U/L (normal up to 150 U/L). The CD4 count was unknown. The chest radiograph was normal. An abdominal ultrasonography revealed a thickened gallbladder wall with pericholecystic fluid. Necrotic lymph nodes were found in the porta hepatis. A contrast enhanced CT scan of the abdomen confirmed thickening of the gallbladder wall with pericholecystic fluid. In addition, multiple ring enhancing hypodensities in the porta hepatis suggesting lymphadenopathy were noted. The patient was diagnosed as acalculous cholecystitis and treated with antibiotics and antispasmodics. Over a 10-day period the symptoms improved; serial liver function tests undertaken during this period demonstrated a normalizing trend.

On day 14 following admission the patient underwent a laparoscopic cholecystectomy. The gallbladder was distended with easy identification of structures in Calotâ™s triangle. Apart from a dense inflammatory reaction in the porta hepatis region, there were no other features to suggest abdominal TB. Histological examination revealed granulomatous inflammation with caseation necrosis suggesting TB; Ziehl Neelson staining was positive for acid-fast bacilli suggesting TB of the gallbladder.

Figure 1

Fig. 1: Thick arrow showing thickened gallbladder wall; thin arrow showing pericholecystic fluid; broken arrows showing porta hepatis lymph nodes.



DISCUSSION

Tuberculosis of the gallbladder was originally described by Gaucher in 1870. Over a century later, Bergdahl reported 41 cases of this condition which remains uncommon[2], even in areas with a high incidence of tuberculosis. Usually, TB of the gallbladder occurs in women over 30 years old, presenting with protean symptoms such as right upper quadrant pain, weight loss, fever, diarrhea, nausea, vomiting and a palpable abdominal mass[3].

The gallbladder has been regarded as resistant to TB infection owing to its thick wall and natural resistance conferred by bile[4]. The high alkalinity of bile inhibits the growth of the mycobacteria. The role of extracellular pH on immune function is well established. An acidic environment has been shown to impair macrophage, lymphocyte and natural killer cell function[5]. It is postulated that the alkaline environment in bile may be responsible for the innate resistance. Cholelithiasis and cystic duct obstruction are considered the most important factors in the development of TB of the gallbladder with cholelithiasis being present in 70% of cases[6]. It has been suggested that tuberculous bacilli, having negotiated the biliary passages, form a nidus for calculus formation. In this report, while there was no cholelithiasis, obstruction to the biliary tree could have been predisposed by enlarged lymph nodes at the porta hepatis. It is a moot point whether the porta hepatis lymph nodes developed secondary to tuberculous involvement of the gallbladder.

Traditionally, TB of the gallbladder follows either haematogenous or lymphatic spread, by contiguous involvement from a recognized focus, or following ascending/descending infection along the biliary tree. An immunocompromised state is an important clinical predisposition to this condition[7]. In the HIV/AIDS era, TB of the gallbladder should be a differential diagnosis in patients presenting with acute or chronic cholecystitis in the presence or absence of gallstones. Gulati et al.[8] emphasized that there are no pathognomonic diagnostic imaging features, but the condition may mimic acute cholecystitis, chronic cholecystitis and a gallbladder mass like gallbladder cancer. The presence of TB in other organs, namely liver, spleen, peritoneum and mesenteric/para-aortic lymph nodes increases the suspicion of TB of the gallbladder when there are radiological findings of gallbladder involvement[9]. An accurate pre-operative diagnosis cannot be made and the literature review highlights that most cases are diagnosed post cholecystectomy or at post-mortem. The routine histopathological examination of all cholecystectomy specimens should therefore be standard practice.

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