

TUBERCULOUS GALLBLADDER – A RARE PRESENTATION OF TUBERCULOSIS

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ABSTRACT

Tuberculosis is one of the most widespread diseases worldwide but gallbladder tuberculosis (GT) is an extremely rare disease, and very few cases have been reported in the literature. The first case of GT was described in 1870 by Gaucher. The unusual case reported here showed features highly indicative of cholelithiasis with tuberculous disease of the gallbladder – as evidenced by the clinical, laboratory and ultrasonographic and CT scan findings, and confirmed by histopathological examination.

Key Words: Chronic Cholecystitis, Gallbladder Tuberculosis; Cholelithiasis

Introduction

Abdominal tuberculosis is common in developing countries but gallbladder tuberculosis (GT) is an extremely rare disease^[1,2], and very few cases have been reported in the literature.^[1] An unusual case of a female with cholelithiasis and tuberculous disease is hereby presented.

Case Report

A 30-year-old female presented with a 6 months history of recurrent attacks of right hypochondrial pain that was localized and intermittent with increasing intensity on eating spicy and fatty food. She suffered from loss of appetite, occasional non-bilious vomiting, and low grade fever throughout the day. Two days prior to admission, she had severe pain, fever, abdominal distension and frequent excessive vomiting of yellow contents. There were no other associated symptoms. The patient was of low socio-economic status. On examination, the patient looked ill, wasted and pale. She was jaundiced, febrile and there was no noticeable skin rash. The chest, cardiovascular and central nervous systems showed no abnormality. The abdomen was distended, soft and tender in the right hypochondrium. The liver was palpable 2 cm below the costal margin. Bowel sounds were frequent and digital rectal examination was unremarkable.

A provisional diagnosis of chronic cholecystitis with cholelithiasis was made. Possible diagnostic consideration here would include atypical appendicitis, pancreatitis, colitis, ascending cholangitis, and hepatitis due to viral or other etiology. Initial investigations showed a haemoglobin level of 9.2 gm/dl, PCV 29%, WBC

count 9900/cmm with relative neutrophilia. The ESR was 65mm/hr. The RBCs showed mixed hypochromia, micro- and anisocytosis, and normal platelet count. Reticulocyte count was not raised. Sickling test was negative. No malarial parasites were seen on a blood film. The blood urea and electrolytes were within normal limit. Serum bilirubin was normal, total proteins 8.1mg/dl, albumin 4.7mg/dl and the alkaline phosphatase level was 13 KAU. ALT, AST and clotting profile were within normal limit.

Urine analysis showed no abnormality. The stool contained mucus, but no evidence of a parasitic or bacterial infection.

An abdominal ultrasound scan revealed multiple enlarged lymph nodes at porta hepatis and gallbladder calculi. Liver, spleen, pancreas, kidney, urinary bladder, stomach, small and large bowels appear normal. CT scan shows same finding of enlarged lymph nodes in porta hepatis region, and there was no ascites, or pleural effusion.

Histopathology from gallbladder shows granuloma comprising of caseating necrosis, epithelioid cells, giant cells, lymphocytes and fibroblast. So Histopathological features are of tubercular lesion.

Discussion

The first case of tuberculosis of gall bladder in the world literature was described in 1870 by Gaucher.^[2] In 1908, Simmonds^[7] collected 8 cases from the literature including two of his own cases. The first case from Kashmir was reported by Akhter et al and the second by Misgar et al.^[4]

The incidence of gall bladder tuberculosis is very low^[6], although the frequency of cholecystitis has increased during the past decades.^[3] Various reasons are given for the low incidence of gall bladder tuberculosis - including failure to recognize the condition or a special resistance of the gall bladder to the tubercle bacillus.^[5] It is not clearly known whether the infection can occur in a normal gall bladder. Kettler^[3] has proposed that the absence of tubercles from the mucosa indicates a haematogenous or lymphogenic spread of infection, whereas tubercles mainly localized in the mucosa denote canalicular dissemination. Tubercles scattered over the serous layer of the gall bladder might indicate dissemination via the peritoneal cavity.^[2]

The gallbladder is highly resistant to tubercular infection, and the presence of cholelithiasis and cystic duct obstruction is essential for the development of GT. About 70% of GT cases are accompanied by gallstones. Four types of GT have been described - according to whether only the gallbladder is involved, or there is generalized tuberculosis, and whether the gallbladder mucosa is histologically involved. GT often occurs together with other intra-abdominal tuberculosis, usually in women over 30 years of age. However, in the present case, it was restricted to the gallbladder, because the duodenum was only locally involved. The route of infection for GT may be peritoneal, haematogenous or lymphatic. Histologically, the lesion may be in the form of a localized lesion. A wide spectrum of symptoms have been described in patients with GT, including abdominal pain in right hypochondrium, weight loss, fever, anorexia, diarrhea, nausea, vomiting and a palpable abdominal mass. Anaemia, elevated ESR and positive tuberculin test are usually found on laboratory examinations. The correct diagnosis of GT is difficult, and it is usually made after a cholecystectomy. The differential diagnosis of GT includes acute and chronic cholecystitis, polypoid lesions and gallbladder carcinoma. The presence of a mass that fills the gallbladder associated with cholelithiasis is indistinguishable from carcinoma of the gallbladder.

Moreover, both GT and carcinoma can give rise to regional lymph nodes. The presence of liver metastasis or liver infiltration suggests the presence of a gallbladder carcinoma. On the other hand, lung lesions or mesenteric thickening is common in children. The symptom may be caused by infection, dyskinesia or malformation of the gallbladder and the cystic duct. In the present case, there was no pathognomonic presentation of this condition.

Usually, symptoms can vary from non-specific symptoms of fever, anorexia, abdominal pain and jaundice; to even gallbladder perforation. An unusual presentation of a persistent port-site sinus in a patient after laparoscopic cholecystectomy (due to cholelithiasis) has also been recorded. Imaging findings, though often, describe features of chronic cholecystitis and cholelithiasis or intraluminal mass lesion, such findings are nonspecific and a histological confirmation is absolutely essential.

Conclusion

It is important to have a high index of suspicion of tuberculosis in such patients, particularly in endemic region, even if their clinical diagnosis shows a case of chronic cholecystitis or malignancy, to avoid unnecessary delay in treatment.

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