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TUBERCULOSIS OF GALL-BLADDER MIMICKING MALIGNANCY – A CASE REPORT

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A correct preoperative diagnosis of gall-bladder tuberculosis is exceptionally unusual in the absence of pathognomic features both on clinical presentation and on imaging. Herein we present a case of 50 year female who was operated with a provisional diagnosis of gall-bladder malignancy and was found to have tuberculosis of gall-bladder on histopathology.
Key words: gall-bladder tuberculosis, gall-bladder cancer, hepatobiliary tuberculosis, tuberculosis

Tuberculosis is endemic in developing countries and reported prevalence of abdominal tuberculosis is 12% (1). Tuberculosis of gall-bladder (TB-GB) is extremely rare because of inhibitory action of bile on mycobacterium. First case of TB-GB was reported by Gaucher in 1870 and till now less than 150 cases have been reported in the English literature (2, 3, 4).

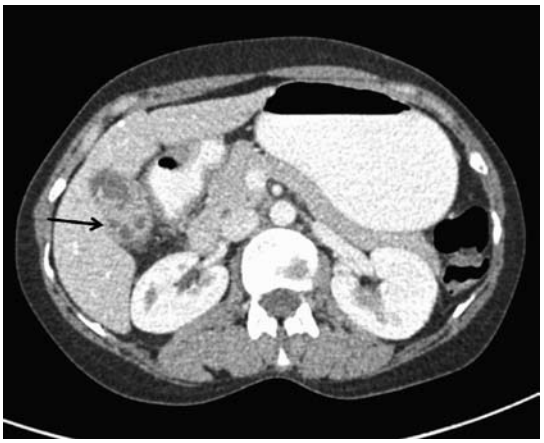
There are no pathognomic presenting symptoms. Clinical features may range from simple cholecystitis or its associated complications to malignancy. As there are no diagnostic criteria on imaging, therefore diagnosis depends solely on histopathological examination of resected specimen. Herein we report a case of TB-GB who was operated with a provisional diagnosis of gall-bladder malignancy.

CASE REPORT

A 50 year female presented with non-radiating, mild, intermittent pain in the right upper abdomen for 2 weeks. There was no history of nausea, vomiting, fever, jaundice, anorexia or weight loss. Her general physical, cardiovascular and respiratory examination was unremarkable. On abdominal examination, hard gall-bladder was palpable; there was no hepatomegaly or ascites. Per rectal examination was normal. Her biochemical, haematological parameters and Chest X-ray was normal. Ultrasound (US) abdomen revealed gall-bladder mass. Computed tomography (CT) scan abdomen (fig. 1, 2) showed asymmetrical soft tissue thickening in the fundus and body of gall-bladder, GB was abutting hepatic flexure of colon although no infiltration was appreciated. There was no ascites and no peripotal, para-aortic or mesenteric lymphadenopathy.

She was taken up for surgery with a provisional diagnosis of carcinoma gall-bladder. On exploration, there was no evidence of peritoneal or hepatic metastasis, no evidence of peritoneal tubercles, no ascites, and no peripotal, para-aortic or mesenteric lymphadenopathy. There was a hard mass in the fundus and body of gall-bladder which was densely adherent to hepatic flexure of colon with no plane of dissection in-between. Therefore, extended cholecystectomy with en-bloc resection of hepatic flexure of colon and colocolic anastomosis was done. Cystic duct margin was free of tumor on frozen section.

On cut section of gall-bladder, GB wall was one cm thick and lumen was filled with pigmented stones. Colon was firmly adhering to gall-bladder. On microscopic examination, gall-bladder showed ulcerated mucosa with trasmural inflammation extending up to serosa with serosal fibrosis. There were large areas of necrosis with multiple epithelioid cell



Ryc. 1. Komputerowa tomografia osiowa wzmocniona kontrastem – jama brzuszna pacjentki wykazująca niejednorodne wzmacniające się zgrubienie ściany obejmujące trzony i dno pęcherzyka żółciowego (strzałka)

granulomas, Langhan's giant cells and palisading histiocytes in the gall-bladder (fig. 3). Features were of granulomatous inflammation consistent with tuberculosis. Stain for AFB was negative. There was marked inflammation over the serosal aspect of colon. However, there were no inflammatory changes identified in the mucosa and muscle of colon. Choledochal and celiac lymph nodes showed features of reactive hyperplasia.

She had an uneventful postoperative recovery and was discharged on postoperative day 8. She was prescribed anti-tubercular treatment (ATT) after receiving the diagnosis of TB-GB on histopathology. She has completed her 6 months course of ATT and is doing well on follow up 2 years after surgery.

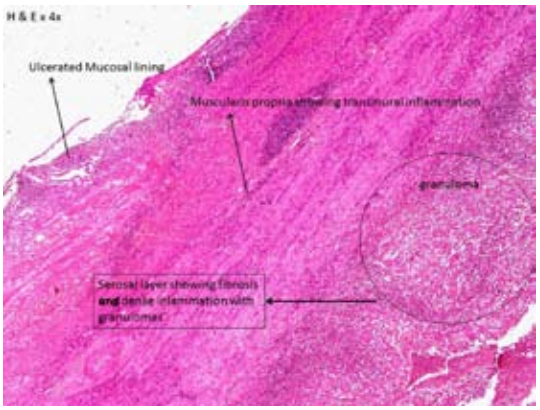


Fig. 3. H&E staining on histopathology (4x) showing ulcerated gall-bladder mucosa with transmural inflammation with granulomas and giant cells extending upto serosa

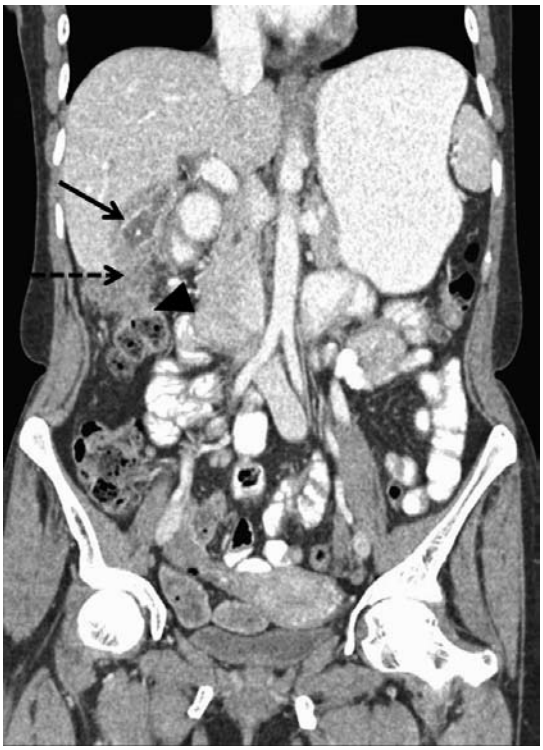


Fig. 2. 3-D coronal reformatted CECT image showing cholelithiasis (arrow) with a heterogeneous soft tissue growing exophytically from the fundus of gall-bladder (dotted arrow) infiltrating into the hepatic flexure of colon (arrow head)

DISCUSSION

Isolated TB-GB is an extremely rare entity. Gall-bladder is relativery resistant to tubercular infection due to its highly alkaline bile which has inhibitory action on mycobacterium. Gall-stones have been isolated in 70% of patients with TB-GB (5). Clinical studies have shown the pathogenic significance of gall stones and development of TB-GB. Gall-stones may cause obstruction of cystic duct which leads to loss of bile acids from the gall-bladder which in turn decreases the natural innate resistance to mycobacterium (6). Gall-stones may also cause damage to gall-bladder wall because of persistent irritation between stone and mucosa which is a risk factor for the development of both cholecystitis and carcinoma.

Routes of infection to gall-bladder may be peritoneal, hematogenous, lymphatic or due to contiguous spread from adjacent organs or adjacent caseating lymph nodes. Kettler has proposed that the absence of tubercles from the mucosa indicates a hematogenous or lymphatic spread; whereas presence of tubercles on the serosa indicates spread via the perito-

neal cavity (7, 8). Four distinct clinical varieties of TB-GB has been documented: 1) as a component of miliary tuberculosis, 2) as a component of disseminated abdominal tuberculosis, 3) isolated TB-GB, 4) in anergic states like uremia, AIDS, or malignancy. In this case there were no tubercles on the mucosal or serosal aspect of gall-bladder, there was no evidence of miliary or peritoneal tuberculosis; there was primary involvement of gall-bladder and because of intense inflammatory response colon was densely adherent to gall-bladder. Therefore, we think index case might have resulted from hematogenous spread of infection causing isolated TB-GB.

It usually occurs in women over 30 years of age. Clinical presentation is usually non-specific. Patients may present with variable symptoms like abdominal pain, weight loss, low grade fever, anorexia, nausea, vomiting, diarrhoea, jaundice and rarely awareness of lump in the abdomen. It is not feasible to correctly diagnose and differentiate isolated TB-GB from clinical features mimicking cholelithiasis or carcinoma gall-bladder. Differential diagnosis of TB-GB includes acute or chronic cholecystitis, gall-bladder polyp, and gall-bladder cancer. The presence of mass in association with cholelithiasis raises the suspicion of gall-bladder malignancy. There are few reported cases in the English literature where TB-GB was masquerading as gall-bladder cancer (9, 10). As US and CT findings are non-specific, therefore, preoperative diagnosis is difficult to make. If there is associated liver infiltration or multiple lesions in the liver, it suggests malignancy. But if there is mesenteric thickening or lung lesions it is more in suggestion of tuberculosis. PET scan also cannot rule out malignancy as increased glycolysis in macrophages results in high FDG uptake value (9, 10). Definitive diagnosis of tuberculosis is made only after histopathology examination of resected specimen or presence of acid fast bacilli. Therefore, tuberculosis should be kept as one of the differential diagnosis in endemic areas in patients with biliary malignancy.

CONCLUSION

To diagnose isolated TB-GB preoperatively is not feasible as its clinical features and radiological findings mimic cholelithiasis or malignancy. Therefore, tuberculosis should be kept as one of the differential diagnosis in endemic areas in patients with biliary malignancy and every specimen should be subjected to histopathological evaluation.

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