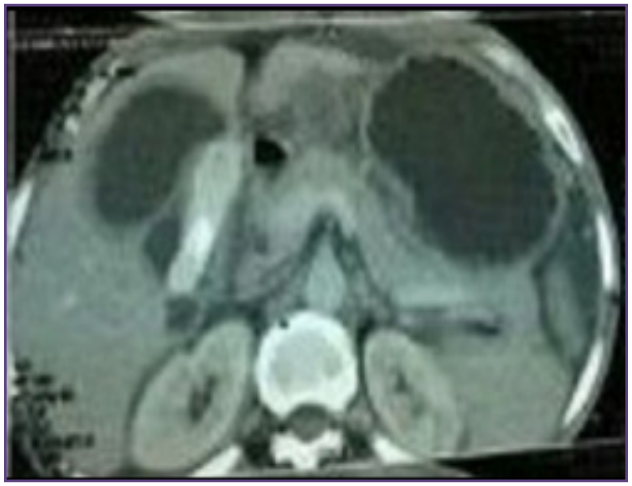

Fig. 1: CT abdomen showing distended sludge filled gall bladder with a calculus of 3.5cm diameter.

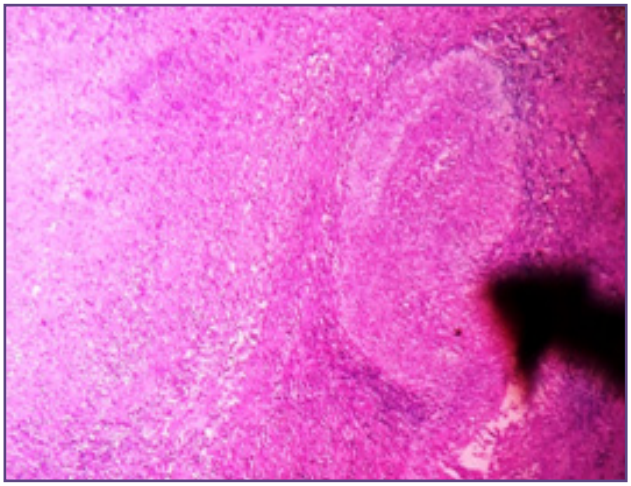

Fig. 2A (H & E stain, 10X): thickened fibrotic wall with granuloma (black arrow) comprising of caseous necrosis.

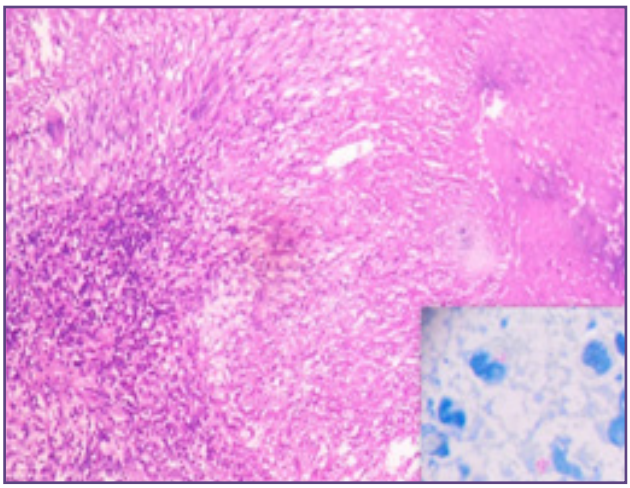

Fig. 2B (H & E stain, 40X): Granuloma comprising of caseous necrosis, epithelioid cells, giant cells and lymphocytic infiltrate. Inset (ZN stain, 100X) shows acid fast tubercular bacilli.

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, even in areas with a high incidence of tuberculosis [3]. Gastrointestinal tuberculosis is commonly seen in the form of tuberculous peritonitis or involvement of the abdominal lymph nodes. Gallbladder tuberculosis is very rare worldwide, with only 50 cases reported in the literature till 2003 [4]. It occurs most commonly in women over 30 years of age [5,6] and cholelithiasis is associated in more than 70% of cases [4]. According to Sir BOA Moynihan, "a gall stone is a tomb-stone erected to the memory of the organism within it". Infecting organism reaches gallbladder via blood stream or lymphatics from a focus nearby [2]. Mycobacteria can also be a cause of cholelithiasis and/or cholecystitis, particularly when tuberculosis is disseminated to the peritoneum and lymph nodes in the vicinity. The rarity of tuberculous involvement of gallbladder could be due possibly to hypovascularity of gallbladder sac and high alkalinity of concentrated bile inside it. Isolated tuberculosis of gallbladder has also been reported but without any direct evidence of its primary involvement [2]. Our case had no evidence of tuberculosis elsewhere with no any past history of tuberculosis and can be assumed to be primary gallbladder tuberculosis. When the bacilli travel by ascending infection through the biliary passages, they form a nidus for the stone formation. Around the bacilli, multiple layers are laid down which leads to the formation of the stone. Also, the bile becomes infected with the presence of the bacilli. It has been suggested that cystic duct obstruction leads to the disappearance of the

bile acids from the gallbladder and to a lowered resistance against tuberculous bacilli [7]. Cholelithiasis and cystic duct obstruction are considered the most important factors in the development of gallbladder tuberculosis[1] Isolated gallbladder tuberculosis has been reported on a few occasions, mostly in adult patients [8,9]. It has also been shown to occur following military tuberculosis [10]. It may be due to the secondary spread from the distant foci or direct extension from neighborhood lesions. Absence of tubercles on gallbladder mucosa indicates hematogenous or lymphatic spreading, whereas tubercles mainly located on the mucosa suggest canalicular dissemination. Tubercles scattered over the serous layer of the gallbladder suggest spreading from peritoneal cavity [11,12]. Review of the role of ultrasound in the detection of hepatobiliary disease showed that the presence of gallbladder wall thickening and sludge, when coupled with right upper quadrant pain and normal liver function tests, correlates with a significant incidence of intrinsic pathology of the gallbladder [13,14].

Conclusion

The incidence of abdominal tuberculosis is increasing and the familiarity with its clinical presentation shortens its diagnostic time and improves its management. As tuberculous cholecystitis is difficult to diagnose and most cases are diagnosed post cholecystectomy or at post-mortem, all resected cholecystectomy specimens should be sent for histopathological examination for evidence of tuberculosis.

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