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Infiltrating Gall Bladder Mass - Malignant or Benign

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ABSTRACT

Xanthogranulomatous (XGC) cholecystitis is a rare cause of chronic cholecystitis characterized by a focal or diffuse destructive inflammatory process with accumulation of lipid laden macrophages (Foam cells), fibrous tissue and inflammatory cells. XGC can exhibit similar imaging and intraoperative findings as those of GB Carcinoma and are easily misdiagnosed, often leading to unnecessary radical surgery. High index of suspicion at diagnosis and increased knowledge about the disease can circumvent extensive surgery.

KEYWORDS

Xanthogranulomatous cholecystitis; Infiltrating gall bladder mass; Chronic cholecystitis, Foam cells

INTRODUCTION

Xanthogranulomatous (XGC) cholecystitis is a rare disease causing chronic cholecystitis. There is focal or diffuse destructive inflammation in the gall bladder wall along with accumulation of lipid laden macrophages (Foam cells), fibrous tissue and inflammatory cells [1]. The inflammatory process may be severe and extend into adjacent organs, such as the liver, and fistulae may develop into surrounding hollow viscera (namely the duodenum and transverse colon) [2]. The incidence of XGC is variable and has been described among series of cholecystectomies to range between 0.6 % and 10 % [3]. Although XGC being a benign condition, it present a similar clinical, imaging and intraoperative findings as those of Gallbladder cancer (GBC), causing a frequent misdiagnosis [4].

Due to the rarity of the disease and its resemblance to gallbladder carcinoma (GBC) in clinical presentation and preoperative imaging, it is not uncommon that patients with XGC are taken to the operating room without a clear diagnosis [2].

We describe here a similar confusing case of Gall bladder (GB) mass which on pre-operative clinical assessment and imaging was suggestive of GBC but finally case out to be a benign disease.

CASE REPORT

A 65-year-male presented to our OPD with complaint of pain and lump in abdomen for 1 month with occasional history of post-prandial vomiting. He had no significant past medical history. On examination a gall bladder was palpable in the right hypochondrium extending into the right lumbar region. It was a non-tender hard mass with limited mobility. There was no hepatomegaly and rest of

the abdomen was normal. The blood investigations were normal except of raised serum alkaline phosphatase. The tumour marker CA19-9 was also not raised.

Ultrasound of the abdomen showed cholelithiasis asymmetrical gall bladder wall thickening with loss of mural stratification s/o malignant gall bladder (GB) wall thickening.

CECT-abdomen showed distended gall bladder with heterogeneously enhancing multifocal irregular circumferential thickening of GB wall predominantly involving its body and fundus having indistinct fat planes with segment 5 of liver. Inferiorly, lesion was abutting hepatic flexure of colon. No hilar lymphadenopathy was observed (Figure 1).

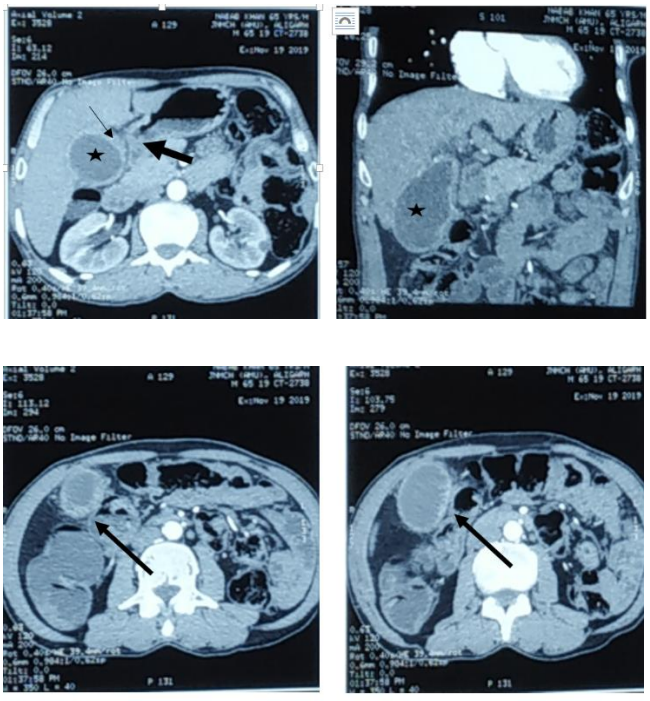

Figure 1: CECT- Abdomen: (A) Transverse section showing distended gall bladder (asterisk) with thickened wall (thin arrow) and abutment with the pylorus (thick arrow). (B) Coronal section. (C) & (D) Transverse section showing abutment of gall bladder with the hepatic flexure of colon with maintained fat plane (arrow).

The patient was planned for open surgical exploration. Intraoperatively, gall bladder was enlarged distended and thickened. The hepatic colon and pylorus was adhered to the body of the GB. However, there were no enlarged lymph node or liver infiltration/nodule and also the extrahepatic bile ducts were normal (Figure 2).

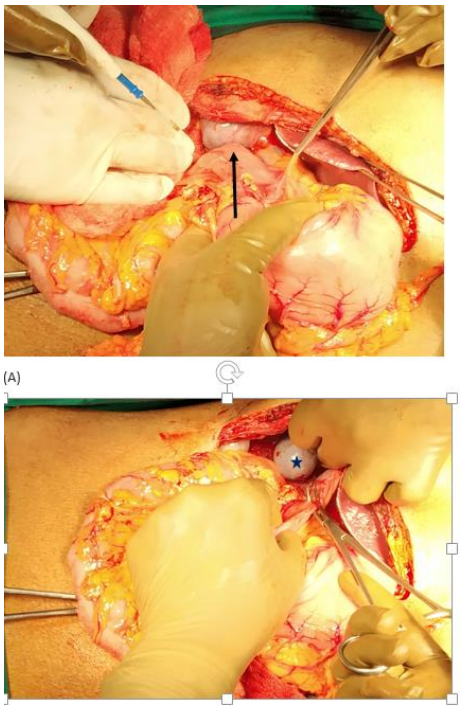

Figure 2: Per-operative photograph: (A) showing adhesion of pylorus and hepatic colon with the gallbladder (arrow). (B) After removing the adhesion, the gall bladder fundus (Star) can be seen.

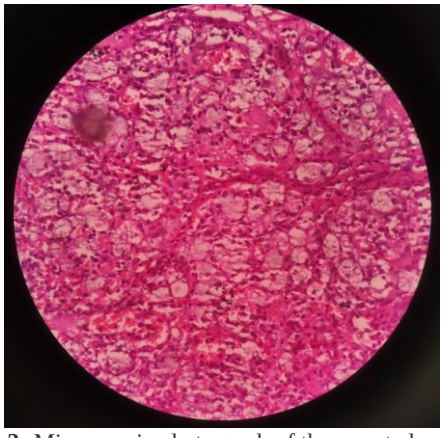

Figure 3: Microscopic photograph of the resected specimen showing foamy cells and inflammatory cells consistent with xanthogranulomatous cholecystitis.

The per-operative finding and previous experience of similar cases raised the suspicion of XGC so we sent tissue for frozen section examination and in it identified only inflammatory cells, no malignant cell was seen. Thus we proceeded for simple cholecystectomy and the adhesion with bowel were removed gently. The post-operative

period was uneventful and the patient was orally allowed on post-operative day 2 and discharged after 5 days of surgery.

Pathological analysis of the surgical specimen revealed hypertrophic fibromuscular layer with inflammatory infiltrate containing foamy macrophages suggesting xanthogranulomatous cholecystitis and absence of any malignant cell (Figure 3). With over four months of follow-up, the patient remains well and asymptomatic.

DISCUSSION

XGC was first reported and named by McCoy in 1976 and described as a distinct pathological condition by Goodman and Ishak in 1981 [5]. It is a variant of cholecystitis characterized by intense acute or chronic inflammation, severe proliferative fibrosis with formation of multiple yellow-brown intramural nodules and foamy histiocytes. It is speculated that XGC may result from extravasation of bile into the gallbladder wall from rupture of occluded Rokitansky-Aschoff sinuses or through small ulceration in the mucosa. Extravasated bile provokes inflammation, and macrophages phagocytose bile lipids and cholesterol to form ceroid-laden and foamy histiocytes (xanthoma cells). The macroscopic appearance is of a poorly defined, nodular yellow mass that infiltrates the wall of the gallbladder. The inflammatory process often extends into neighbouring organs, such as the liver, omentum, duodenum and colon [2,6,7].

XGC can behave similar to GBC in terms of imaging and intra-operative features, thus leading to unnecessary radical surgery. An increased awareness combined with high index of suspicion could help avoid extensive surgery. It has been repeatedly suggested that intraoperative frozen-section analysis may be useful when diagnosis is in doubt, in order to avoid an unnecessarily aggressive/“mutilating” intervention [2, 3,7-10].

In our case, clinical and radiological features clued the diagnosis of carcinoma gall bladder, but frozen section examination ruled out malignancy. So, going only by radiological imaging and clinical features would have resulted in treatment by extensive surgical resection. But intra-op frozen section guided in planning of surgical procedure and open cholecystectomy was performed. Biopsy confirmed the diagnosis of XGC.

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