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Xanthogranulomatous Cholecystitis Complicated by Large Hepatic Abscess: A Rare Case Report and Literature Review

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Abstract **Case Report**

Xanthogranulomatous cholecystitis (XGC) is an uncommon, benign but locally invasive inflammatory disorder of the gallbladder that may closely resemble gallbladder cancer in clinical and radiographic evaluations. Although the majority of XGC cases are limited to the gallbladder, contiguous inflammatory expansion leading to hepatic abscess development is an infrequent, although potentially severe, consequence. We report the case of a 90-year-old male who had right upper quadrant stomach discomfort and fever, with imaging indicating a significantly thickened, dilated gallbladder and a massive, multiloculated hepatic abscess, suggesting direct inflammatory extension. The patient was well treated with percutaneous abscess drainage followed by laparoscopic cholecystectomy, which confirmed histopathologically the presence of XGC without cancer. This case emphasizes the clinical and diagnostic difficulties associated with XGC, including uncommon liver involvement, and shows the need to identify such complications for effective care.

Keywords: Xanthogranulomatous cholecystitis, Gallbladder cancer mimic, Liver abscess, CT and MRI of XGC.

INTRODUCTION

Xanthogranulomatous cholecystitis (XGC) is a rare, benign, locally aggressive gallbladder inflammatory disease with considerable wall thickening and substantial infiltration by lipid-filled macrophages, multinucleated giant cells, and chronic inflammatory cells.[1] Mostly limited to the gallbladder. XGC may invade the hepatic parenchyma and cause abscesses. This extension causes thick adhesions, gallbladder wall perforation, and intrahepatic collections, which might resemble advanced gallbladder cancer or other suppurative diseases clinically and radiologically.

Chronic inflammation and gallbladder mucosa rupture or ulceration enable the inflammatory process to infiltrate the liver via the serosa. Direct contiguous extension or localized perforation may transmit infection and cause hepatic abscesses, particularly with gallstones or biliary blockage. Imaging usually shows irregular, hypoechoic, or hypodense regions in the thickened gallbladder wall, continuous hepatic infiltration, and pericholecystic or intrahepatic abscesses. Preoperative diagnosis is difficult owing to radiologic signs overlapping with cancer; histological evaluation must show granulomatous inflammation. [2,3]

Acute or chronic cholecystitis may cause fevers, right upper quadrant discomfort, and sepsis if abscesses are large. Since extensive hepatic involvement requires more complex procedures and can complicate postoperative recovery, prompt recognition is essential for surgical planning and management. [3,4] XGC often mimics gallbladder carcinoma both clinically and radiologically; rare cases may present with contiguous spread to adjacent organs such as the liver, leading to serious complications like abscess formation.[5] Early recognition is critical to guide appropriate management and avoid unnecessary radical surgery.[6] In this case report, we present a 90-year-old Libyan male patient who was diagnosed as XGC with liver extension, with a literature review.

CASE DESCRIPTION

A 90-year-old male presented with right upper quadrant abdominal pain, fever, and malaise. Laboratory investigations revealed mild neutrophilic leukocytosis and elevated inflammatory markers. Liver function tests

showed mild cholestatic changes. Serum CA19-9A was mildly elevated. Computed tomography (CT) scan showed a markedly thickened and distended gallbladder with diffuse heterogeneous mural thickening. Hypoattenuating intramural nodules within the gallbladder wall. Adjacent liver parenchymal involvement with a large multiloculated enhancing lesion involving segments VIII and V, consistent with a hepatic abscess secondary to contiguous inflammatory extension with associated mild intrahepatic biliary ductal dilatation. **Figure 1** shows these features.

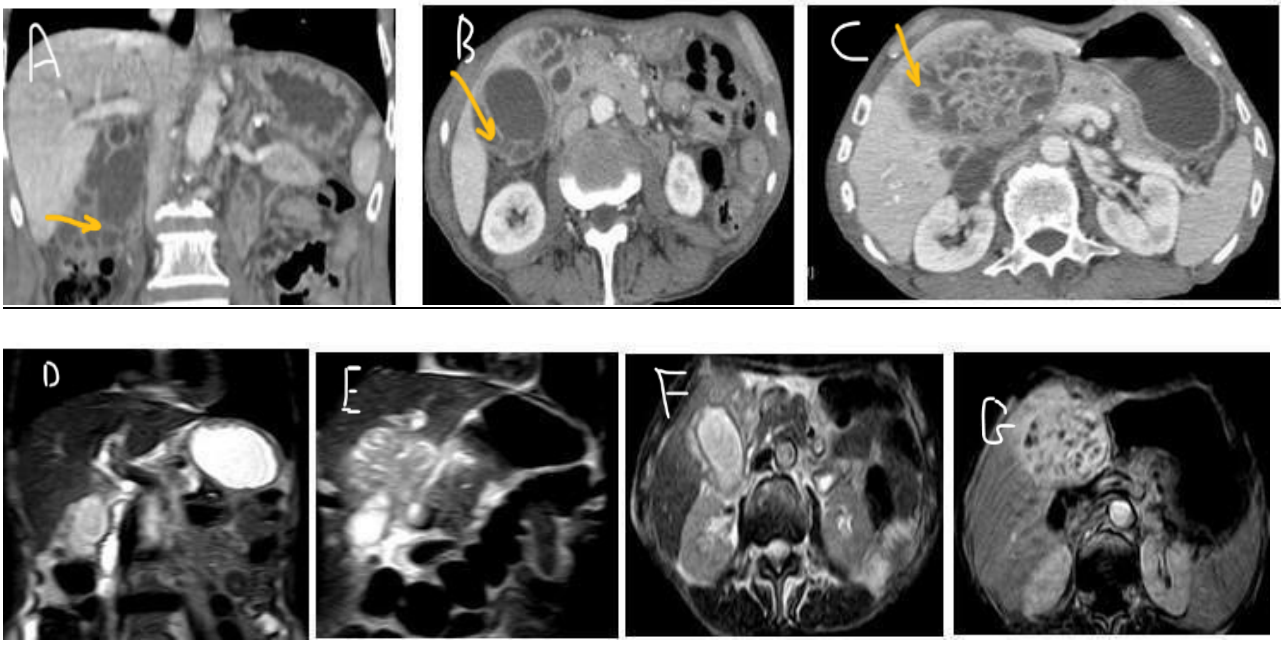

Figure 1: CT findings

Contrast-enhanced CT scan of the abdomen (axial view) demonstrates a markedly thickened and distended gallbladder with diffuse heterogeneous mural thickening and multiple hypoattenuating intramural nodules (image A-B). The adjacent hepatic parenchyma, particularly segments V and VIII, shows a large, multiloculated, peripherally enhancing low-attenuation lesion consistent with a hepatic abscess secondary to direct contiguous inflammatory extension from the gallbladder wall (image C).

Coronal, axial T2-weighted, and axial post-contrast T1-weighted magnetic resonance images demonstrated gallbladder wall thickening with multiple high-signal intramural nodules (Image D, F). Delayed enhancement of the gallbladder and a multiloculated large contrast-enhanced hepatic abscess (image E, G).

Management and Outcome

The patient underwent percutaneous drainage of the liver abscess followed by laparoscopic cholecystectomy and antibiotic coverage. Intraoperatively, the gallbladder was densely adherent to the liver with extensive inflammatory changes. Histopathology confirmed xanthogranulomatous cholecystitis with no evidence of malignancy.

DISCUSSION

Xanthogranulomatous cholecystitis (XGC) is a benign, locally aggressive inflammatory disease with approximately 1.3 -5.2% incidence in cholecystectomy specimens.[7] XGC diagnosis and therapy are a dilemma for diagnostic radiologists, surgeons, and physicians.[8] Hepatic abscess formation due to contiguous spread from XGC is rare and can create a diagnostic challenge.[9] Features favoring XGC over carcinoma include: A) Presence of intramural hypoattenuating nodules, B) Preservation of the mucosal line, C) Diffuse gallbladder wall thickening, and D) Lack of overt discrete mass or distant metastasis.[10] In XGC cases complicated by liver abscess, timely intervention with drainage and appropriate surgical management is critical for patient recovery. Awareness of this rare complication can prevent misdiagnosis and guide optimal treatment.[11]

The gut and extra-gut endothelial cells synthesize CA19-9. Various factors, including enzyme deficiencies, ethnicity, diet, and comorbidities, influence the expression and metabolism. Overexpression is more frequently observed in the context of pancreaticobiliary pathology, particularly in pancreaticobiliary malignancies. The epigenetic silencing factor associated with carcinogenesis is responsible for the malignant overexpression of CA9-9, as it is regarded as a tumor marker. Nonetheless, the emerging terminology is now tumor-associated due to its substantial correlation with extensive benign pathology. Extremely high levels of CA19-9 are typically observed in advanced cases of pancreaticobiliary malignancy, although this is not exclusive.[12]

In biliary conditions, acute cholangitis, choledocholithiasis, and cholecystitis are the most frequently reported cases associated with significantly elevated CA 19-9 levels. This suggests that obstructive inflammation is likely the cause of elevated CA 19-9 levels. The exact cause of extremely high levels of CA 19-9 in benign conditions remains unknown. However, several theories have been proposed. The first theory is that elevated inflammatory cytokines result in the overexpression of CA 19-9. The second theory is that significant inflammation causes ductal obstruction and extensive cellular destruction, leading to increased production and hematogenous leakage [13,14].

In XGC, CA 19-9 plays a significant role in assessment alongside clinical and radiological evaluations to distinguish between gallbladder adenocarcinoma and xanthogranulomatous cholecystitis. However, emerging evidence indicates extremely high levels of CA 19-9 in xanthogranulomatous cholecystitis, raising questions about its significance. An elevated CA 19-9 level was reported in 45.95% of patients with xanthogranulomatous cholecystitis.[15] CA19-9 levels are significantly elevated in gallbladder carcinoma; however, it is not a reliable marker for differentiation between gallbladder cancer and XGC.[16] In our case, the serum levels of CA 19-9 were mildly increased; however, in the follow-up period, they improved

Computed Tomography is a valuable investigation in XGC, and its findings usually reveal diffuse or focal wall thickening, intramural hypodense nodules within thickened walls, and luminal surface enhancement accompanied by continuous or focal disruption of mucosal lines. Gallbladder calculi and pericholecystic fluid accumulation are frequently linked with XGC, with infiltration into adjacent liver parenchyma or bowel may also occur.[17] CT may also reveal additional complications such as perforation, abscess, or fistula formation. The CT scoring system evaluates five components: diffuse wall thickening, absence of polypoid lesions, intramural nodules or bands, pericholecystic infiltration, and pericholecystic abscess collection. This approach is likely to enhance the sensitivity and specificity of CT diagnosis for xanthogranulomatous cholecystitis. The presence of three or more findings demonstrated a sensitivity of 77% and a specificity of 94%, accompanied by a 95% Confidence Interval. In a study of 49 confirmed cases of XGC, CT findings revealed diffuse wall thickening in 87.8% of cases and intramural hypodense nodules in the thickened wall in 85.7% of cases. Luminal surface enhancement accompanied by a continuous mucosal line was observed in 85.7% of cases. Gallbladder stones were observed in 69.4% of the patient population. CT findings were identified in 40% of cases, with 80% exhibiting four or more of these features. Coexistent malignancy of the gallbladder has been documented in the literature. [18,19] A separate study indicated that 19.6% of XGC cases were associated with gallbladder carcinoma. The CT scan of the patient revealed irregular diffuse thickening of the gallbladder wall, accompanied by several hypoattenuating mural nodules, multiple hyperdense calculi, pericholecystic fluid collection, and diminished fat planes adjacent to the liver parenchyma.[18] Another case report revealed irregular diffuse gallbladder mural thickening with a few hypoattenuating mural nodules, multiple calculi beside pericholecystic fluid collection, and poor fat planes at the nearby hepatic parenchyma.[3]

CONCLUSION

Xanthogranulomatous cholecystitis must be included in the differential diagnosis for individuals with gallbladder wall thickening and concomitant hepatic diseases, particularly when imaging reveals characteristic characteristics while malignancy remains a possibility. Increased CA 19-9 levels may be seen in XGC, requiring meticulous interpretation with radiological and clinical evidence. Radiological modalities, particularly computed tomography, may indicate a diagnosis; nonetheless, a conclusive differentiation from cancer needs histopathological examination. Recognizing unusual consequences, such as hepatic abscess development, is essential for diagnostic precision and to inform appropriate treatment, hence preventing overtreatment or unwarranted severe surgery and enhancing patient outcomes.

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