Case report of xanthogranulomatous cholecystitis, review of its sonographic and magnetic resonance findings, and distinction from other gallbladder pathology

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ABSTRACT

A case of xanthogranulomatous cholecystitis is presented with a brief review of its sonographic and magnetic resonance features. These imaging features are also compared to those seen in gallbladder adenomyomatosis and gallbladder carcinoma. While there are many overlapping imaging findings in these entities, it is important to recognize distinguishing characteristics so a correct surgical approach is chosen. Laparoscopic cholecystectomy attempted with existing xanthogranulomatous cholecystitis has an increased surgical complication rate compared to open cholecystectomy and often necessitates intraoperative conversion to open cholecystectomy.

CASE REPORT

CASE REPORT

A 78 year old male was undergoing inpatient work-up for gastrointestinal bleeding after colonic polypectomy. Admission lab values demonstrated elevated liver function tests and the patient reported an episode of post-prandial pain a month earlier. The patient reported a diagnosis of cholecystitis from an abdominal sonogram 17 years prior obtained for a single episode of abdominal pain. The patient denied further medical or surgical treatment for this condition. Other pertinent medical history included diet controlled diabetes mellitus, hypertension, hyperlipidemia, prostate cancer, and abdominal aortic aneurysm. Pertinent laboratory values included a mildly elevated white blood cell count (15.2 x103/mm3) and alkaline phosphatase (164 IU/L). The remainder of the patient lab values were normal.

Ultrasound (Figures 1-3) demonstrated a large gallstone and diffuse gallbladder (GB) wall thickening up to 9mm (normal thickness is less than 3mm) with cystic areas in the fundal wall. The GB lumen also contained innumerable echogenic foci also with comet tail ring down artifact. Sonographic Murphy's sign was negative. No intrahepatic or extrahepatic biliary dilatation was noted. These findings were consistent with cholelithiasis and adenomyomatosis. A recommendation was given for further evaluation with a Magnetic Resonance Imaging (MRI) to exclude GB carcinoma.

Multisequence MRI prior to and after the intravenous administration of gadolinium was preformed (Figures 4-8). Notable findings included significant thickening of the gallbladder wall, 2 cm in maximum width, with multiple cystic outpouchings in addition to a large gallstone. Additionally, there was T2 hyperintensity, in the liver parenchyma surrounding the GB fossa consistent with inflammatory change. No abnormal enhancing hepatic foci identified. Findings were felt to still be consistent with adenomyomatosis, however malignancy could not be definitely excluded.

The patient underwent a laparoscopic cholecystecomy which was converted to open cholecystecomy secondary to "extensive adhesions and inflammation" documented in the surgical report. The adhesions required liver bed resection and hepatic flexure colonic bowel resection in addition to the cholecystectomy.

The pathologic specimen showed GB adenomyomatosis (GBAM) with features of chronic xanthogranulomatous cholecystitis (XGC) including foamy histiocytes.

DISCUSSION

Although GBAM and XGC share many of the same clinical and imaging features, they are described as separate entities in the literature and are distinguishable histologically [1]. Both demonstrate GB wall thickening with sonographic intramural hypoechoic nodules. Gallstones are often seen with both conditions. Both entities represent benign inflammation, but have imaging characteristics which mimic focal GB carcinoma [1].

GBAM is a process of diffuse epithelial and smooth muscle proliferation likely in response to chronic GB obstruction. Dilated Rokitansky-Aschoff (RA) sinuses contribute to formation of intramural diverticula that may contain bile, cholesterol, sludge, or stones. The cholesterol crystals demonstrate the characteristic reverberation or V-shaped comet tail artifact on ultrasound examination [1]. RA sinuses are also reported to be visible on T2 weighted MR; "Pearl necklace sign"[2,3].

While GBAM is common, up to 8.7% of surgical specimens [4], XGC is a less common gallbladder condition. The pathological cascade of XGC is believed to begin in the setting of chronic GB outlet obstruction which results in extravasation of bile into the GB wall through mucosal ulceration or rupture of RA sinuses. GB wall abscesses with foci of xanthogranulomatous inflammation lead to the pathologic hallmark of foamy histiocytes and subsequent wall fibrosis and scar formation [1]. Scar formation leads to a higher complication rate (up to 32%) in XGC versus GBAM [5] including: GB perforation, abscess formation, fistulous tracts to the duodenum or skin, peritoneal scar formation.

Utilizing sonography and computed tomography (CT)/MRI to differentiate XGC from GBAM is important for clinical management. Inflammatory changes outside the gallbladder should raise suspicion for XGC or GB carcinoma, both requiring open cholecystecomy to address potential complications resulting from inflammatory changes and scarring. Laparoscopic cholecystectomy, in the presence of XGC, is frequently unsuccessful secondary to unclear surgical anatomy from intense fibrosis leading to higher complication rates [6].

Attempts to distinguish XGC from GB carcinoma have been undertaken in case series but have been unsuccessful secondary to difficulty differentiating carcinoma when severe inflammatory changes are present [7]. An XGC case series of 26 patients found intramural hypoechoic nodules or bands in the presence of a thickened GB wall with cholelithiasis/chronic cholecystitis was highly suggestive of XGC [8]. Another case series comparing XGC and GB carcinoma proposed that reliable findings for XGC included ultrasound findings of hypoechoic nodules occupying greater than 60% of a thickened wall with CT findings of an intact mucosal line. Disruption of the mucosal line was associated with GB carcinoma [9]. Recently, an XGC case series with 19 cases, demonstrated hypoechoic nodules in 100% of XGC cases and an intact mucosal line in 79% [10]. Cases of coexisting XGC and GB carcinoma have also been reported [6]. Additional, a recent case report demonstrated that XGC had fluorodeoxyglucose uptake on positron emission tomography mimicking that of GB carcinoma [11].

TEACHING POINT

Xanthogranulomatous cholecystitis is a rare entity, and even though its sonographic, CT and MRI features can mimic gallbladder adenomyomatosis, differentiation is important for guiding surgical management. Sonographic, CT, and MRI differentiation of xanthogranulomatous cholecystitis from gallbladder carcinoma has proven problematic and requires further investigation.

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FIGURES

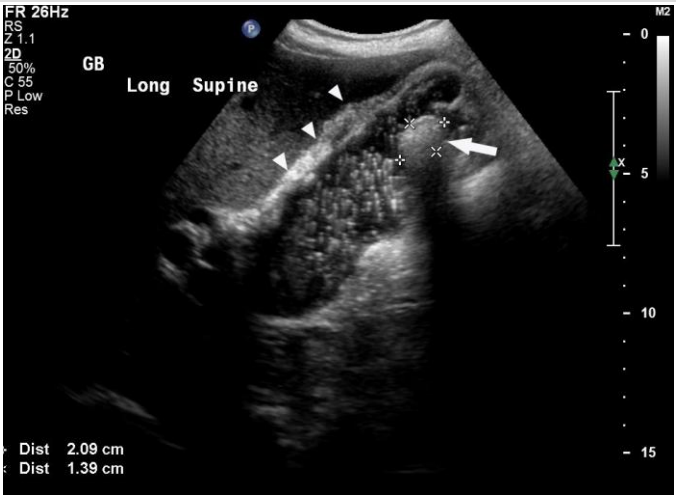


Figure 1: 78 year old male with xanthogranulomatous cholecystitis. Longitudinal sonogram of the gallbladder demonstrates a 2.1 cm echogenic focus with posterior acoustic shadowing consistent with a gallstone (arrow) and diffuse GB echogenic contents, ringdown artifact from intramural nodules within a diffusely thickened gallbladder wall (arrow heads) representing cholesterol-filled cysts. [Phillips, curvilinear 5 Mhz transducer]

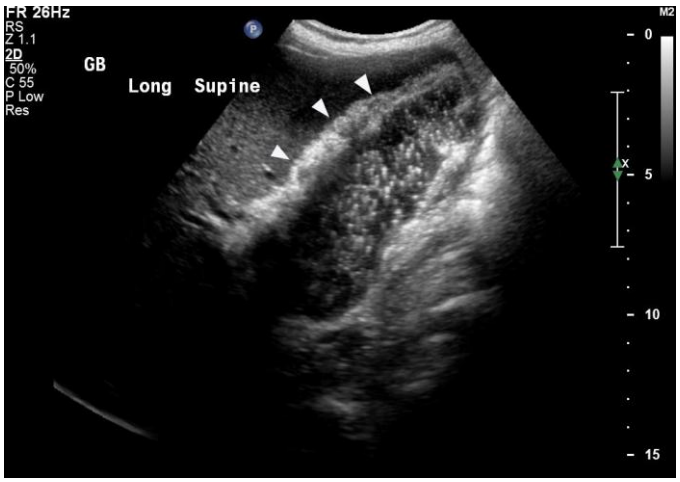


Figure 2: 78 year old male with xanthogranulomatous cholecystitis. Longitudinal sonogram of the gallbladder demonstrates diffuse GB echogenic contents, ringdown artifact from intramural nodules within a diffusely thickened gallbladder wall (arrow heads) representing cholesterol-filled cysts. [Phillips, curvilinear 5 Mhz transducer]

Figure 3: 78 year old male with xanthogranulomatous cholecystitis. Transverse sonogram of the gallbladder demonstrates diffuse GB echogenic contents, ringdown artifact from intramural nodules within a diffusely thickened gallbladder wall (arrow heads) representing cholesterol-filled cysts. [Phillips, curvilinear 5 Mhz transducer]

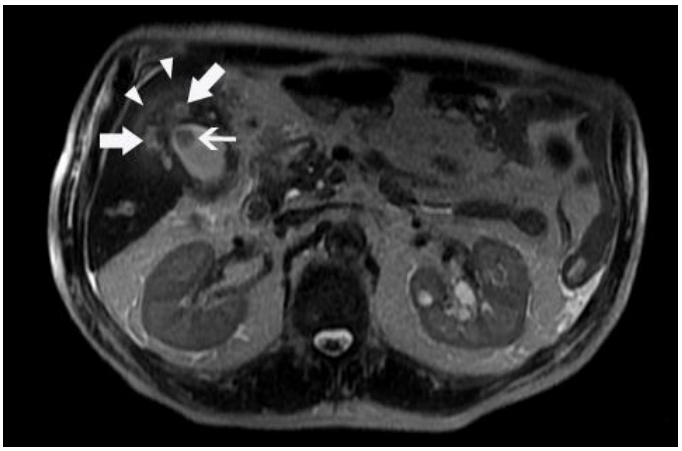


Figure 4: 78 year old male with xanthogranulomatous cholecystitis. Axial T2 weighted image demonstrates GB wall thickening with hyperintense intramural cysts (thick arrows), hypointense gallstone (thin arrow), and adjacent hepatic T2 hyperintensity, representing inflammation (arrowheads). [GE Signa Excite 1.5 Tesla magnet, 8 channel body coil, 6 mm slices, TE=90, TR=907, no contrast]

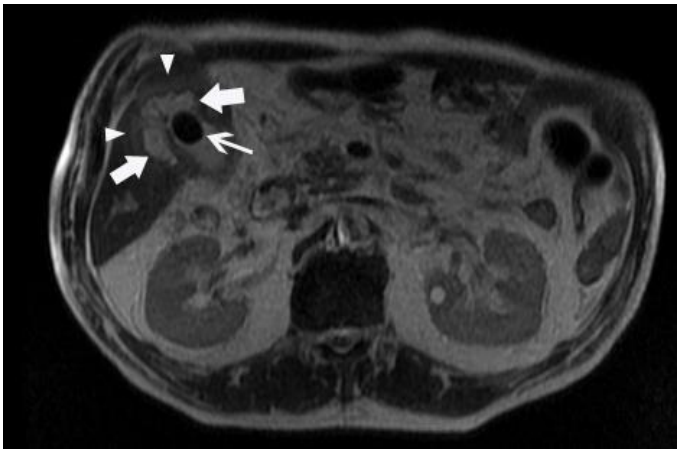


Figure 5: 78 year old male with xanthogranulomatous cholecystitis. Axial T2 weighted image demonstrates GB wall thickening with hyperintense intramural cysts (thick arrows), hypointense gallstone (thin arrow), and adjacent hepatic T2 hyperintensity, representing inflammation (arrowheads). [GE Signa Excite 1.5 Tesla magnet, 8 channel body coil, 6 mm slices, TE=90, TR=907, no contrast]

Figure 8 (right): 78 year old male with xanthogranulomatous cholecystitis. Axial spoiled gradient post-contrast image demonstrates GB wall thickening (thick arrows) and hypointense gallstone (thin arrow). No areas of abnormal enhancement. [GE Signa Excite 1.5 Tesla magnet, 8 channel body coil, 6 mm slices, TE=2, TR=4.2, 30 cc intravenous gadodiamide]

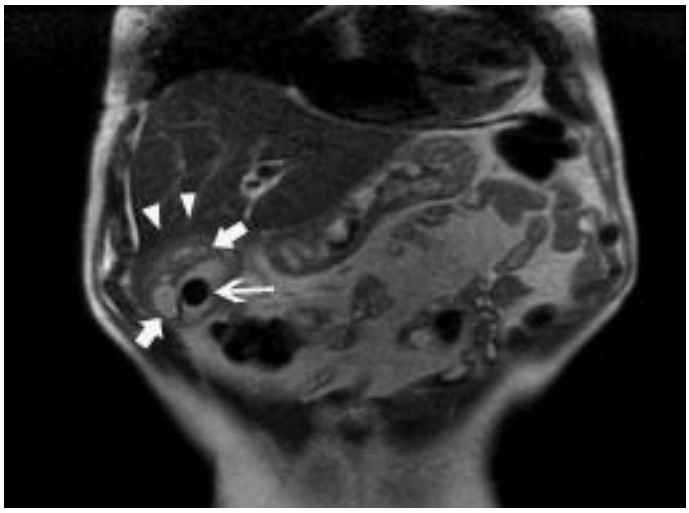
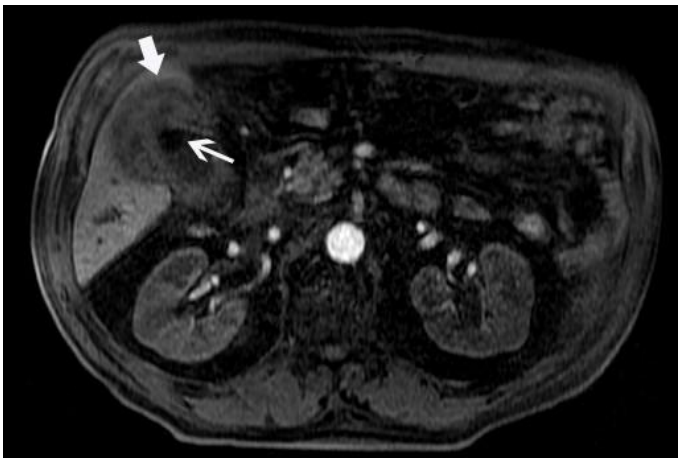


Figure 6: 78 year old male with xanthogranulomatous cholecystitis. Coronal T2 weighted image demonstrates GB wall thickening with hyperintense intramural cysts (thick arrows), hypointense gallstone (thin arrow), and adjacent hepatic T2 hyperintensity, representing inflammation (arrowheads). [GE Signa Excite 1.5 Tesla magnet, 8 channel body coil, 6 mm slices, TE=89, TR=1235, no contrast]

Figure 7: 78 year old male with xanthogranulomatous cholecystitis. Axial spoiled gradient pre-contrast image demonstrates GB wall thickening (thick arrows) and hypointense gallstone (thin arrow). [GE Signa Excite 1.5 Tesla magnet, 8 channel body coil, 6 mm slices, TE=2, TR=4.2, no contrast]



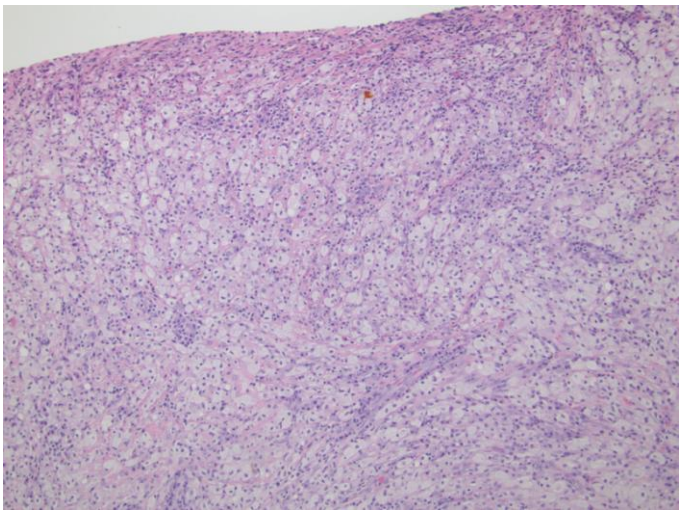


Figure 9: 78 year old male with xanthogranulomatous cholecystitis. Photomicrograph at low power of denuded gallbladder wall demonstrating sheets of neutrophils, areas of mixed inflammation, and sheets of foamy histiocytes. (Stain: hematoxylin and eosin; Magnification: 20x)

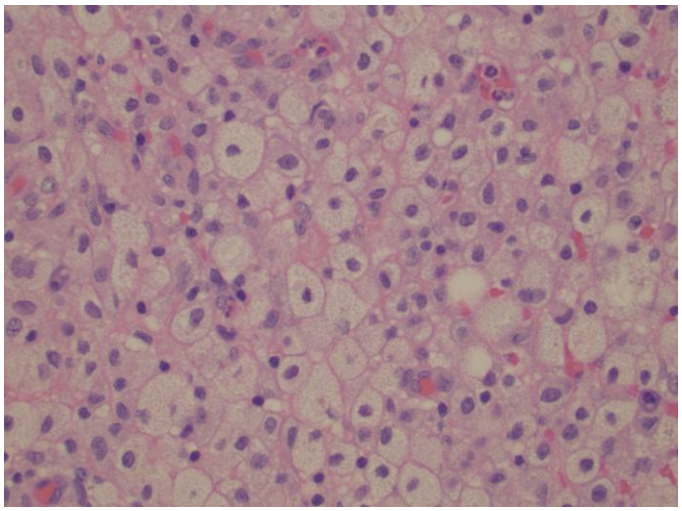


Figure 10: 78 year old male with xanthogranulomatous cholecystitis. Photomicrograph at high power of denuded gallbladder wall demonstrating sheets of neutrophils, areas of mixed inflammation, and sheets of foamy histiocytes. (Stain: hematoxylin and eosin; Magnification: 400x)

Etiology	Not firmly established, but extravasation of bile into the gallbladder wall is thought have a role in the inflammatory process.
Incidence	1%-2% of surgically resected gallbladders
Gender Ratio	Occurs more commonly in females
Age Predilection	6^{th}-7^{th} Decade
Risk Factors	None identified
Treatment	Open cholecystecomy
Prognosis	Complications include gallbladder perforation, abscess formation, fistulous tracts to duodenum or skin, extension of the inflammatory process to adjacent abdominal organs. In uncomplicated cases, prognosis is excellent following surgical management.
Findings on Imaging	Ultrasound: Focal/diffuse GB wall thickening with hypoechoic bands or nodules with comet tail artifact. Possible gallstones and disruption of the mucosal line with indistinct liver margin. Pericholecystic inflammation. CT: Focal/diffuse GB wall thickening with non-enhancing wall hypodense nodules. Possible gallstones and disruption of the mucosal line. Pericholecystic inflammation. MRI: GB wall thickening with T2 bright cystic spaces and hepatic/pericholecystic inflammation.

Table 1. Summary table for xanthogranulomatous cholecystitis

Differential Diagnosis	Ultrasound	CT	MRI
Chronic Cholecystitis	Diffuse GB wall thickening with contracted GB. Gallstones. No pericholecystic inflammation.	Diffuse GB wall thickening with contracted GB. Gallstones. No pericholecystic inflammation.	Diffuse GB wall thickening with contracted GB. Gallstones. No pericholecystic inflammation.
Gallbladder Adenomyomatosis	Focal/diffuse GB wall thickening with hypoechoic nodules with comet tail artifact. Possible gallstones. No pericholecystic inflammation.	Focal/diffuse GB wall thickening with non-enhancing wall hypodense nodules. Possible gallstones. No pericholecystic inflammation.	GB wall thickening with T2 bright cystic spaces in (string of pearls). No pericholecystic inflammation. No contrast enhancement.
Xanthogranulomatous cholecystitis (XGC)	Focal/diffuse GB wall thickening with hypoechoic bands or nodules with comet tail artifact. Possible gallstones and disruption of the mucosal line with indistinct liver margin. Pericholecystic inflammation.	Focal/diffuse GB wall thickening with non-enhancing wall hypodense nodules. Possible gallstones and disruption of the mucosal line. Pericholecystic inflammation.	GB wall thickening with T2 bright cystic spaces and hepatic/pericholecystic inflammation.
GB Carcinoma	Echogenic GB wall polyp or mass with infiltrating thickening of GB wall with gallstones. Often disruption of mucosal line with liver infiltration and pericholecystic inflammation.	Hypovascular GB fossa mass infiltrating liver. Disruption of the mucosal line. Calcified gallstones or porcelain GB.	Infiltrating GB wall mass with increased T1 and T2 signal in respect to liver. Poor contrast enhancement. Pericholecystic inflammation.

Table 2. Differential diagnosis table for xanthogranulomatous cholecystitis

ABBREVIATIONS

GB = Gallbladder
GBAM = Gallbladder Adenomyomatosis
XGC = Xanthogranulomatous Cholecystitis
RA = Rokitansky-Aschoff
CT = Computed Tomography
MRI = Magnetic Resonance Imaging

KEYWORDS

Xanthogranulomatous cholecystitis; adenomyomatosis; gallbladder MRI

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