Cureus
Part of SPRINGER NATURE

Open Access Case Report

Xanthogranulomatous Cholecystitis After Biliary Stenting: A Case Report and Review of Diagnostic Challenges

Review began 10/25/2026
Review ended 11/15/2026
Published 11/20/2026

© **Copyright** 2026
Adhane et al. This is an open access article distributed under the terms of the Creative Commons Attribution License CC-BY 4.0., which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

DOI: 10.7759/cureus.119997

Abhishek A. Adhane [1], Satyajeet G. Pathrikar [1]

1. General Surgery, Mahatma Gandhi Mission Medical College and Hospital, Chhatrapati Sambhajinagar, IND

Corresponding author: Abhishek A. Adhane, adhaneabhishek49@gmail.com

Abstract

Xanthogranulomatous cholecystitis (XGC) is an uncommon, benign inflammatory disease of the gallbladder. Its wall thickening, adhesions, and sometimes mass-like appearance can resemble gallbladder carcinoma (GBC), so preoperative and intraoperative differentiation is difficult. We report the case of a 78-year-old woman who presented with right upper-quadrant pain one month after endoscopic retrograde cholangiopancreatography (ERCP) and common bile duct (CBD) stenting for choledocholithiasis. Contrast-enhanced computed tomography (CT) showed acute cholecystitis with a thick-walled, septated gallbladder and pericholecystic changes suggestive of a possible contained perforation, but no discrete mass, hepatic invasion, or lymphadenopathy. At open cholecystectomy, dense adhesions to the omentum and transverse colon raised concern for a locally invasive malignancy. A careful cholecystectomy with adhesiolysis was performed. Histopathology confirmed XGC with no dysplasia or carcinoma, and the patient recovered uneventfully. This case shows that dense adhesions alone are not a reliable indicator of malignancy. Recognising XGC as a benign mimic of GBC, using a measured operative approach, and relying on histopathology can help avoid overtreatment.

Categories: General Surgery, Pathology
Keywords: biliary stenting, case report, differential diagnosis, gallbladder wall thickening, open cholecystectomy, xanthogranulomatous cholecystitis

Introduction

Xanthogranulomatous cholecystitis (XGC) is an uncommon variant of chronic cholecystitis, first characterised as a distinct clinicopathological entity by Goodman and Ishak in 1981 [1]. Histologically, it is defined by a focal or diffuse destructive inflammatory process with intramural lipid-laden (foamy) macrophages, multinucleated giant cells, a mixed inflammatory infiltrate and progressive fibrosis [1,2]. Reported prevalence varies widely across series, from under 1% to 8.8% of cholecystectomy specimens, with the highest frequencies described from South Asia [2,3].

The clinical significance of XGC lies in its resemblance to gallbladder carcinoma (GBC). Wall thickening, mass formation, and infiltration of adjacent organs can occur in both conditions, and reliable preoperative or intraoperative differentiation is frequently difficult [4,5]. Patients with benign XGC may be subjected to extended resections intended for malignancy [4,6]. Recognising the features that favour each diagnosis, and the pitfalls that lead to over-resection, is therefore important for surgeons. We present a case of XGC in an elderly woman, in whom intraoperative adhesions raised concern for malignancy, and we review the diagnostic features that help distinguish the two conditions.

Case Presentation

Patient information

The patient was a 78-year-old woman with the following past history: a right middle cerebral artery aneurysm treated by craniotomy and clipping in October 2024; a previous cerebrovascular accident, maintained on lacosamide; a psychiatric disorder, maintained on nitrazepam, escitalopram-clonazepam and alprazolam; and an open appendicectomy 20 years earlier. She had no diabetes mellitus, hypertension, tuberculosis or asthma, and no known drug allergies. She was a non-smoker and non-alcoholic, and a housewife.

Presenting complaint

She presented to the outpatient clinic of our centre with right upper-quadrant pain of one month's duration. One month earlier, at an outside centre, she had undergone endoscopic retrograde cholangiopancreatography (ERCP) with common bile duct (CBD) stenting under general anaesthesia for choledocholithiasis, and she was referred to us after that procedure. She had no fever with chills, nausea, vomiting or haematemesis, and no history of abdominal trauma.

Clinical findings

She was haemodynamically stable and afebrile. The abdomen was soft and non-distended, with tenderness localised to the right upper quadrant (right hypochondrium), no guarding or rigidity, and normal bowel sounds in all four quadrants.

Investigations

Admission laboratory investigations were largely within normal limits, with a mildly raised direct bilirubin and an alkaline phosphatase at the upper limit of normal. The haemogram, renal function, electrolytes and coagulation profile were unremarkable (Table *1*). Glycated haemoglobin was 5.5%, and HIV and hepatitis B surface antigen were non-reactive.

Investigation	Result	Reference range
Haemoglobin	12.8 g/dL	12.0–15.0 g/dL
Total leukocyte count	5920 /mm³	4000–11000 /mm³
Platelet count	164 ×10³/µL	150–410 ×10³/µL
Total bilirubin	0.70 mg/dL	0.3–1.2 mg/dL
Direct bilirubin	0.60 mg/dL	0.0–0.3 mg/dL
Aspartate aminotransferase (AST)	20 U/L	5–40 U/L
Alanine aminotransferase (ALT)	13 U/L	5–40 U/L
Alkaline phosphatase (ALP)	128 U/L	40–129 U/L
Blood urea	16 mg/dL	15–40 mg/dL
Serum creatinine	0.60 mg/dL	0.6–1.2 mg/dL
Sodium	141 mmol/L	135–145 mmol/L
Potassium	4.30 mmol/L	3.5–5.1 mmol/L
International normalised ratio (INR)	0.94	0.8–1.2
Serum albumin	4.4 g/dL	3.5–5.2 g/dL
Total protein	7.8 g/dL	6.4–8.3 g/dL
Glycated haemoglobin (HbA1c)	5.5 %	< 5.7 %

TABLE 1: On-admission investigations

Abdominal ultrasonography, performed after the ERCP and before referral to our centre, showed cholelithiasis with multiple small clumped calculi and sludge within a distended gallbladder, mild gallbladder-wall thickening, and a mildly dilated CBD containing an ill-defined calculus.

Contrast-enhanced computed tomography (CT) of the abdomen showed a thick-walled, septated, acutely inflamed gallbladder with pericholecystic changes suggestive of a possible contained perforation (Figure *1*). No discrete mass, hepatic invasion or regional lymphadenopathy was reported. The CBD stent was not visualised and was presumed to have migrated distally. Preoperative two-dimensional echocardiography showed preserved left-ventricular systolic function (ejection fraction 55%) with grade I diastolic dysfunction.

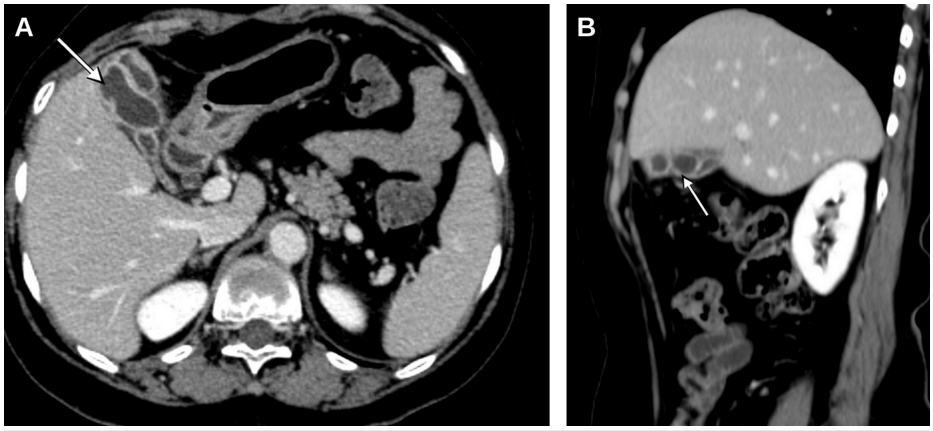


FIGURE 1: Contrast-enhanced CT of the abdomen

(A) Axial and (B) sagittal views showing a thick-walled, septated, acutely inflamed gallbladder (arrows) with pericholecystic changes suggestive of a possible contained perforation.

Management and operative findings

Given the clinical picture and imaging, the patient underwent open cholecystectomy through a right Kocher's (subcostal) incision under general anaesthesia. The open approach was chosen preoperatively because of anticipated technical difficulty. She had a previous open appendicectomy, which was expected to be associated with dense adhesions, and CT suggested a possible contained perforation with thick-walled inflammation, which was expected to make dissection difficult. Given her age and comorbidities, a planned open procedure was preferred to a laparoscopic approach, with a probable need for conversion and suspicion of malignancy was not the basis for this decision.

Intraoperatively, dense adhesions were found between the gallbladder, omentum and transverse colon. These caused significant difficulty in dissection and raised concern for a locally invasive gallbladder carcinoma. Given the patient's age and comorbidities, and the absence of macroscopic hepatic infiltration or regional lymphadenopathy, a careful cholecystectomy rather than an extended resection was undertaken. Meticulous adhesiolysis was performed to mobilise the gallbladder, followed by dissection of Calot's triangle. The cystic artery and cystic duct were identified, clamped and ligated, and the gallbladder was separated from the liver bed and delivered intact. No gallbladder perforation or bile leak was identified. An abdominal drain was placed, and the procedure was uneventful.

Pathology

The resected gallbladder was thick-walled and fibrotic on gross examination. Histopathology showed features characteristic of XGC, including sheets of lipid-laden foamy histiocytes, multinucleated giant cells, a chronic inflammatory infiltrate and fibrosis, with no evidence of dysplasia or invasive carcinoma (Figure *2*).

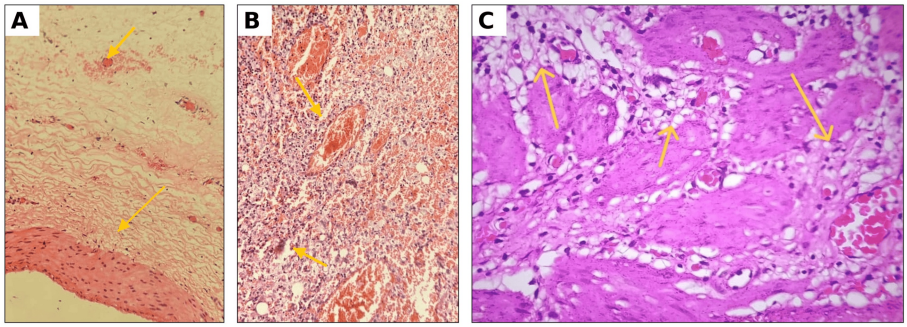


FIGURE 2: Histopathological Images of the resected gallbladder (haematoxylin and eosin)

(A) Low power: gallbladder wall showing dense collagenous fibrosis. (B) Intermediate power: dilated congested vessels with a mixed inflammatory infiltrate. Arrows indicate congested vessels and the inflammatory infiltrate. (C) High power: sheets of lipid-laden foamy histiocytes and multinucleated giant cells, with a mixed chronic inflammatory infiltrate, dilated congested vessels and dense collagenous stroma, characteristic of xanthogranulomatous cholecystitis. Arrows indicate foamy histiocytes and multinucleated giant cells. No evidence of malignancy.

Postoperative course and follow-up

The postoperative course was uneventful. The patient remained haemodynamically stable with a healthy surgical site and no abdominal pain. She was discharged on the seventh postoperative day on ursodeoxycholic acid and supportive medication, with advice for outpatient surgical follow-up.

The ill-defined CBD calculus seen on post-ERCP ultrasonography was not further characterised during admission. Because she was asymptomatic with normal serum bilirubin and alkaline phosphatase, it was managed conservatively, with surveillance planned at follow-up.

At the two-month follow-up, she remained asymptomatic with no complications. Liver function tests were: total bilirubin 0.3 mg/dL, direct bilirubin 0.1 mg/dL, aspartate aminotransferase (AST) 20 U/L, alanine transaminase (ALT) 16 U/L and ALP 48 U/L.

Discussion

Epidemiology and pathogenesis

XGC is uncommon. It accounts for under 2% of cholecystectomy specimens in most series, but for up to 8.8% in India [2,3]. It typically affects older adults. Although the disease is benign, the dense mural fibrosis makes cholecystectomy technically demanding and is associated with a high rate of conversion from laparoscopic to open surgery [7,8]. The pathogenesis is thought to involve extravasation of bile and mucin into the gallbladder wall through ruptured Rokitansky-Aschoff sinuses or ulcerated mucosa, usually with obstructing gallstones and raised intraluminal pressure. This produces a florid granulomatous, histiocytic inflammatory reaction with lipid-laden macrophages and dense fibrosis [2]. Gallstone disease and preceding biliary obstruction requiring ERCP and stenting in our patient are consistent with this mechanism.

Differentiation from GBC

The central diagnostic challenge is differentiation from GBC [4,9]. In a series of eight XGC cases that had all been diagnosed clinico-radiologically as GBC, the two entities shared focal or diffuse wall thickening, mass formation and infiltration of adjacent organs such as the liver, duodenum and colon [4]. In a single-centre review of 1141 cholecystectomies, XGC was found in 0.87% of specimens, and only one of 10 patients was correctly diagnosed preoperatively [10]. In a series of 8213 cholecystectomies (XGC in 0.91%), no patient was suspected of malignancy preoperatively, yet malignancy was suspected intraoperatively in 10.6% [7]. The two conditions may coexist, and GBC may itself masquerade as XGC, so histological demonstration of xanthogranulomatous inflammation does not by itself exclude malignancy [3,11].

Imaging features

Several cross-sectional features may favour XGC over GBC. These include diffuse rather than focal wall thickening, intramural hypoattenuating nodules corresponding to xanthogranulomas, preservation of a continuous intact mucosal line, and the absence of macroscopic hepatic invasion or regional lymphadenopathy [12]. Diffusion-weighted MRI has been reported to help distinguish XGC from the wall-thickening type of early GBC [13]. The Gallbladder Reporting and Data System (GB-RADS) may help with risk

stratification of gallbladder wall thickening on ultrasonography [14]. The specificity of imaging remains moderate, however, and no single feature is reliable in isolation [2,12].

Interpretation of this case

Preoperative CT showed acute cholecystitis with a possible contained perforation, but did not describe a discrete mass, hepatic invasion or lymphadenopathy. The possible perforation was not confirmed at surgery. This reflects how florid inflammation in XGC can mimic complicated disease on imaging. The concern for malignancy, therefore, arose at surgery rather than on imaging.

The dense adhesions binding the gallbladder to the omentum and transverse colon caused significant difficulty in dissection and were the principal intraoperative cause of concern. Adhesions alone, however, are not a reliable indicator of malignancy, because florid inflammation in XGC can itself produce dense adhesions to adjacent organs. In this patient, the absence of hepatic infiltration or regional lymphadenopathy, the intact separation of the gallbladder from the liver bed, and the thick, fibrotic wall on gross examination were more consistent with benign disease. Histopathology confirmed this.

A high index of suspicion for malignancy has historically led to radical procedures, including wedge hepatic resection and resection of the duodenum or colon, for disease that proved to be benign XGC [4-6]. In this case, a careful cholecystectomy with adhesiolysis was undertaken instead, and the benign diagnosis was confirmed. A balanced approach is still required: avoiding overtreatment of benign disease while maintaining vigilance for coexisting carcinoma, guided by definitive histopathology [7,9].

Limitations

This report describes a single case. Serum tumour markers were not measured. Intraoperative frozen section was not sent, and the operative findings showed no hepatic infiltration or regional lymphadenopathy. The CBD calculus seen on ultrasonography was not further characterised with magnetic resonance cholangiopancreatography, which might have provided further information on the biliary tree. The status of the CBD stent after its presumed migration also remained unclear.

Conclusions

XGC is a benign inflammatory disease that can closely resemble GBC on imaging and at operation. Surgeons should consider XGC in the differential diagnosis of gallbladder wall thickening and dense adhesions. Dense adhesions alone should not prompt radical resection, and imaging features such as an intact mucosal line, diffuse wall thickening and the absence of hepatic invasion or lymphadenopathy favour benign disease. In this patient, a careful cholecystectomy with adhesiolysis guided by definitive histopathology achieved a good outcome without overtreatment. Surgeons should also remain alert to the possibility of coexisting carcinoma.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Abhishek A. Adhane, Satyajeet G. Pathrikar

Acquisition, analysis, or interpretation of data: Abhishek A. Adhane

Drafting of the manuscript: Abhishek A. Adhane

Critical review of the manuscript for important intellectual content: Satyajeet G. Pathrikar

Supervision: Satyajeet G. Pathrikar

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

References

1. Goodman ZD, Ishak KG: Xanthogranulomatous cholecystitis. Am J Surg Pathol. 1981, 5:653-9. 10.1097/00000478-198110000-00007
2. Giudicelli X, Rode A, Bancel B, Nguyen AT, Mabrut JY: Xanthogranulomatous cholecystitis: diagnosis and management. J Visc Surg. 2021, 158:326-36. 10.1016/j.jviscsurg.2021.02.004
3. Hale MD, Roberts KJ, Hodson J, Scott N, Sheridan M, Toogood GJ: Xanthogranulomatous cholecystitis: a European and global perspective. HPB (Oxford). 2014, 16:448-58. 10.1111/hpb.12152
4. Khan S, Abeer I, Husain M, Hassan MJ, Jetley S: Xanthogranulomatous cholecystitis mimicking advanced gallbladder carcinoma-analysis of 8 cases. J Cancer Res Ther. 2021, 17:969-75. 10.4103/jcrt.JCRT_1180_19
5. Kishore R, Nundy S, Mehrotra S, Metha N, Mangla V, Lalwani S: Strategies for differentiating gallbladder carcinoma from xanthogranulomatous cholecystitis-a tertiary care centre experience. Indian J Surg Oncol. 2017, 8:554-9. 10.1007/s13193-017-0677-7
6. Frountzas M, Schizas D, Liatsou E, et al.: Presentation and surgical management of xanthogranulomatous cholecystitis. Hepatobiliary Pancreat Dis Int. 2021, 20:117-27. 10.1016/j.hbpd.2021.01.002
7. Şimşek G, Şahin A, Metin ŞH, Ulutaş ME, Arslan K: The management of xanthogranulomatous cholecystitis. Turk J Surg. 2021, 37:242-6. 10.47717/turkjsurg.2021.5020
8. Feng L, You Z, Gou J, Liao E, Chen L: Xanthogranulomatous cholecystitis: experience in 100 cases . Ann Transl Med. 2020, 8:1089. 10.21037/atm-20-5836
9. Chauhan SS, Kumar N, Rana F: Xanthogranulomatous cholecystitis mimicking gall bladder cancer: a diagnostic dilemma and review of literature. Indian J Surg Oncol. 2023, 14:796-9. 10.1007/s13193-023-01778-w
10. Alotaibi AM, Almasoudi E, Ahmed H, Alzwaihiri A: The incidental finding of xanthogranulomatous cholecystitis: a report of 10 cases. J Surg Case Rep. 2022, 2022:443.
11. Deng X, Yang CY, Tian W, et al.: Gallbladder cancer masquerading as xanthogranulomatous cholecystitis: a case report and literature review. Front Oncol. 2024, 14:1409347. 10.3389/fonc.2024.1409347
12. Uchiyama K, Ozawa S, Ueno M, et al.: Xanthogranulomatous cholecystitis: the use of preoperative CT findings to differentiate it from gallbladder carcinoma. J Hepatobiliary Pancreat Surg. 2009, 16:333-8. 10.1007/s00534-009-0067-9
13. Kang TW, Kim SH, Park HJ, Lim S, Jang KM, Choi D, Lee SJ: Differentiating xanthogranulomatous cholecystitis from wall-thickening type of gallbladder cancer: added value of diffusion-weighted MRI. Clin Radiol. 2013, 68:992-1001. 10.1016/j.crad.2013.03.022
14. Gupta P, Dutta U, Rana P, et al.: Gallbladder reporting and data system (GB-RADS) for risk stratification of gallbladder wall thickening on ultrasonography: an international expert consensus. Abdom Radiol (NY). 2022, 47:554-65. 10.1007/s00261-021-03360-w