Print ISSN: 2321-6379
Online ISSN: 2321-595X

Original Article

Xanthogranulomatous Cholecystitis or Gallbladder Carcinoma – A Common Surgical Dilemma: A Prospective Study of 61 Radical Cholecystectomies in a Tertiary Care Hospital in Northern India

Kumari Sunita Bharti[1], Swati Agnohotri[2], Archana Bundela[3], Deval Brajesh Dubey[2], Rajesh Kumar Rai[4], Shilpa Ulhas Vahikar[5], Shaila Kumari Mitra[4]

[1]Assistant Professor, Department of Pathology, Baba Raghav Das Medical College, Gorakhpur, Uttar Pradesh, India, [2]Senior Resident, Department of Pathology, Baba Raghav Das Medical College, Gorakhpur, Uttar Pradesh, India, [3]Associate Professor, Department of Pathology, Baba Raghav Das Medical College, Gorakhpur, Uttar Pradesh, India, [4]Professor, Department of Pathology, Baba Raghav Das Medical College, Gorakhpur, Uttar Pradesh, India, [5]Professor and Head, Department of Pathology, Baba Raghav Das Medical College, Gorakhpur, Uttar Pradesh, India

Abstract

Introduction: Xanthogranulomatous cholecystitis (XGC) is focal or diffuse destructive inflammation followed by marked proliferative fibrosis which spreads to the adjacent organs such as the liver, small intestines, and omentum.

Aims and Objectives: The study aimed to analyze the clinical, radiological, and histomorphological features of all cases of XGC and Gallbladder carcinoma (GBC) to help clinicians and pathologists give accurate pre-operative diagnosis to avoid unnecessary radical resections.

Materials and Methods: A prospective hospital-based observational study was undertaken in the Department of Pathology, Baba Raghav Das Medical College, Gorakhpur, for 24 months which included all the cases that underwent radical cholecystectomy. Immunohistochemistry (CK, CD68) was used in all the cases for confirmation of histopathological diagnosis. Sixty-one cases were included in our study.

Results: Out of 61 patients, 45 (73.8%) were confirmed by histopathology and immunohistochemistry to be XGC, and the rest 16 (26.2%) were GBC. On analyzing clinical features, GBC patients had a significantly higher frequency of significant weight loss (62.5% vs. 2.2%), anorexia (68.8% vs. 6.7%), and clinically palpable mass (37.5% vs. 6.7%). Patients diagnosed with XGC had a significantly higher frequency of fever (75.6% vs. 43.8%). While comparing computed tomography findings, most cases of XGC had cholelithiasis (88.9% vs. 31.3%) and diffuse GB wall thickening (100% vs. 37.5%). Definite mass lesion was seen radiologically in a significant proportion of GBC (43.8% vs. 2.2%). While studying pre-operative tumor markers, carcinoembryonic antigen, and CA125 were characteristically raised in 93.8% and 62.5% cases of GBC, respectively. CD68 immunoreactivity was seen in histiocytes in all the cases of XGC which was negative in GBC. GBC showed strong and diffuse immunoreactivity for CK7.

Conclusion: Thus, integration of clinical, radiological, and pre-operative S. tumor marker findings would better help clinicians diagnose GBC preoperatively and prevent the unwanted morbidity GBC patients face routinely.

Key words: Gallbladder carcinoma, Radical cholecystectomy, Xanthogranulomatous cholecystitis

Access this article online

www.ijss-sn.com

Month of Submission : 09-2023
Month of Peer Review : 09-2023
Month of Acceptance : 10-2023
Month of Publishing : 11-2023

INTRODUCTION

Xanthogranulomatous cholecystitis (XGC) an unusual histologic variant of chronic cholecystitis is histologically defined by focal or diffuse destructive inflammation by chronic inflammatory cell infiltrate including foamy histiocytes and macrophages followed by marked

Corresponding Author: Deval Brajesh Dubey, Department of Pathology, Baba Raghav Das Medical College, Gorakhpur, Uttar Pradesh, India.

proliferative fibrosis which may later spread to the adjacent organs such as the liver, small intestines, and omentum. This results in uneven thickening of the gallbladder (GB) wall and the formation of yellowish-brown nodules. Christensen and Ishak were the first to recognize it in 1970 as "fibroxanthogranulomatous inflammation"[1] and later in 1976 as was renamed to XGC by McCoy *et al.*[2] XGC predominantly affects patients over 50 years of age and shows no gender predilection.[3] Its prevalence ranges from as low as 0.7% in the USA to as high as 10% in India[4] and is seen in 1.3–5.2% of resected GB specimens.[5] Patients may be asymptomatic or present with right hypochondriac pain radiating to the shoulder, fever, and nausea. GB cancer (GBC), then again, is a prognostically poor cancer and has an incidence of 3/100,000 worldwide.[6] Cholelithiasis, advanced age, sclerosing cholangitis, and porcelain sac are the best-known risk factors of GBC. Even with the advancements in the fields of pathology and radiology as low as only one-third of GBC cases can be diagnosed in the pre-operative period.[7]

The pathogenesis of XGC has not yet been clearly understood; yet, the preliminary cause is considered to be the extravasation of bile into the GB wall due to involvement of the Rokitansky-Aschoff sinuses or due to small mucosal ulcer because of constant local injury by the gallstones. This results in an extensive chronic inflammatory process and may extend to adjacent organs, forming dense adhesions with a large mass of inflammatory tissue surrounding the GB.

XGC resembles GBC both preoperatively and intraoperatively as clinical, radiological, and hematological findings are not helpful enough to distinguish XGC from GBC. These close similarities may be the reason for more than 10% of patients being treated with unnecessarily extended resection or having a missed cancer. In such cases, it is of utmost importance to cautiously assess the clinical manifestations and radiological characteristics of XGC to avoid unnecessary radical surgery and prevent morbidity. In the present study, we analyzed the clinical, radiological, and histomorphological features of 45 cases of XGC preoperatively misdiagnosed with GBC. In all these cases, clinical, radiological, and intraoperative findings were highly suggestive of malignancy, thereby leading to radical resection. However, histopathology was ultimately benign.

This study aimed to determine the pre-operative characteristics of XGC that could potentially aid in an accurate diagnosis of XGC masquerading as GBC preoperatively.

MATERIALS AND METHODS

The study aimed to analyze the clinical, radiological, and histomorphological features of all cases of XGC and GBC to help clinicians and pathologists give accurate pre-operative diagnoses so as to avoid unnecessary radical resections. It is a prospective hospital-based observational study undertaken in the Department of Pathology, Baba Raghav Das Medical College (BRDMC), Gorakhpur, for 24 months. Our study included all the cases that underwent radical cholecystectomy on the pre-operative suspicion of GBC and were received in the Department of Pathology, BRDMC for histopathological examination. Demographic details, clinical findings, tumor markers, and pre-operative computed tomography (CT) findings were collected from patients and recorded. All the specimens were grossed according to standard protocol and histopathological slides were stained with hematoxylin and eosin and reported. Immunohistochemistry (CK, CD68) was used in all the cases for confirmation of histopathological diagnosis. A total of 61 cases were included in our study. The data were transferred to the IBM SPSS Statistics program, Version 23, for analysis. When evaluating the study data, frequency distribution (number and percentages) were used for the categorical variables and descriptive statistics (median, minimum, and maximum) for the numerical variables, whereas categorical variables were compared using Fisher's exact test or the χ^2 test. A $P < 0.05$ indicated statistical significance.

RESULTS

Out of 61 patients enrolled in the study, 45 (73.8%) were confirmed by histopathology and immunohistochemistry to be XGC, and the rest 16 (26.2%) were GBC. The findings of our study are summarized in Table 1. The majority of XGC (80.0%) and all the GBC (100%) patients were aged 40–60 years. Only 5 patients (11.1%) with XGC were below 40 years and 4 (8.9%) were more than 60 years of age, though this difference was not found to be statistically significant. XGC ($n = 25$; 55.6%) and GBC ($n = 12$; 75%) both were found to be female-predominant but this difference was also not significant. While analyzing their clinical features, abdominal pain, and jaundice had comparable frequency among GBC and XGC cases. GBC patients had a significantly higher frequency of significant weight loss (62.5% vs. 2.2%), anorexia (68.8% vs. 6.7%), and clinically palpable mass (37.5% vs. 6.7%). On the other hand, patients diagnosed with XGC had a significantly higher frequency of fever (75.6% vs. 43.8%). While comparing CT findings [Figure 1], we found that most cases of XGC had cholelithiasis (88.9% vs. 31.3%) and diffuse GB wall thickening (100% vs. 37.5%). Definite mass lesion was seen radiologically in a significant

Table 1: Results

Clinical finding	XGC (%)	GBC (%)	Statistical significance
Abdominal pain	30 (66.6)	10 (62.5)	χ^2=0.091; *P*=0.763
Jaundice	36 (80.0)	11 (68.8)	χ^2=0.845; *P*=0.358
Fever	34 (75.6)	7 (43.8)	χ^2=5.418; *P*=0.020
Weight loss	1 (2.2)	10 (62.5)	χ^2=5.418; *P*=0.020
Anorexia	3 (6.7)	11 (68.8	χ^2=5.418; *P*=0.020
Palpable mass	3 (6.7)	6 (37.5)	χ^2=8.922; *P*=0.003
Radiological findings			
Hypoattenuated nodule	42 (93.3)	6 (37.5)	χ^2=21.941; *P*<0.001
Loss of interface between GB and liver	21 (46.7)	10 (62.5)	χ^2=1.184; *P*=0.277
Diffuse wall thickening	45 (100.0)	6 (37.5)	χ^2=33.640; *P*<0.001
Gall stone	40 (88.9)	2 (12.5)	χ^2=32.116; *P*<0.001
Regional lymphadenopathy	17 (37.8)	12 (75)	χ^2=6.557; *P*=0.010
Mass	4 (8.9)	16 (100.0)	χ^2=44.462; *P*<0.001
Serum markers			
CEA+/(*n*)	0 (0.0)	15 (93.8)	χ^2=55.944; *P*<0.001
CA125+/(*n*)	0 (0.0)	10 (62.5)	χ^2=33.640; *P*<0.001
CA19.9+/(*n*)	30 (66.7)	14 (87.5)	χ^2=2.548; *P*=0.110
Histological findings			
Foamy histiocytes	45	0	χ^2=61.000; *P*<0.001
Giant cell	4	0	χ^2=1.522; *P*<0.217
Epithelial dysplasia in GB lining	0	16	χ^2=61.000; *P*<0.001
Immunopositivity			
CD68	45 (100.0)	0 (0.0)	χ^2=61.000; *P*<0.001
CK 7	0 (0.0)	16 (100.0)	χ^2=61.000; *P*<0.001

CEA: Carcinoembryonic antigen, GB: Gallbladder, XGC: Xanthogranulomatous cholecystitis, GBC: Gallbladder carcinoma

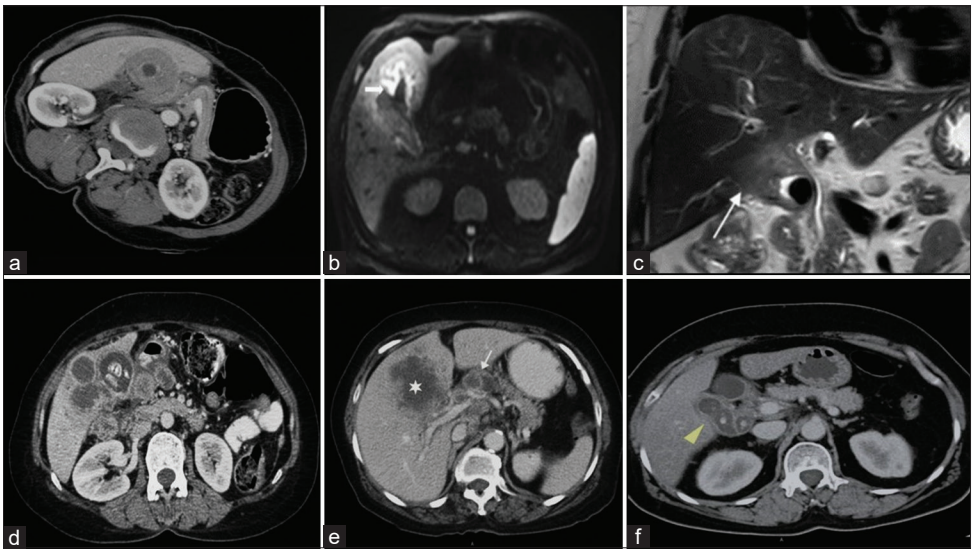

Figure 1: Radiological spectrum of xanthogranulomatous cholecystitis (XGC) and gallbladder carcinoma (GBC). (a) CT: Diffuse symmetrical gallbladder wall thickening, XGC (histopathological diagnosis: XGC). (b) CT: Asymmetrical gallbladder wall thickening with liver involvement, suspicious for malignancy (histopathological diagnosis: XGC). (c) MRI irregular mural of the gallbladder with infiltration into adjacent liver and loss of interface. Suspicious for malignancy (histopathological diagnosis: XGC). (d) CT: Diffuse irregular gallbladder wall thickening with cholelithiasis and enlarged lymph nodes suspicious for malignancy (histopathological diagnosis: XGC). (e) CT: Mass forming lesion with infiltration into liver and regional lymphadenopathy, GBC (histopathological diagnosis: XGC). (f) CT: Diffuse gallbladder wall thickening with cholelithiasis. Suspicious for malignancy (histopathological diagnosis: XGC)

proportion of GBC cases as compared to XGC (43.8% vs. 2.2%). Though hypo-attenuated nodule was seen in a higher proportion of GBC cases as compared to XGC (25.0% and 8.9%, respectively) the difference was not found to be significant statistically. While studying pre-operative tumor markers we found that carcinoembryonic antigen (CEA) and CA125 were characteristically raised in 93.8% and 62.5% cases of GBC, respectively, while being at normal levels in XGC. This association was even statistically significant. To our surprise, raised serum levels of CA19.9 were not

significantly associated with GBC. Histologically [Figure 2], foamy histiocytes and transmural chronic inflammatory cells were key features of XGC whereas epithelial dysplasia, malignant glands, and acini formation were characteristic of GBC. We found that CD68 immunoreactivity was seen in histiocytes in all the cases of XGC which was negative in GBC. GBC showed strong and diffuse immunoreactivity for CK7.

DISCUSSION

XGC is characterized by marked thickening of the GB wall and accumulation of lipid-laden macrophages. Although the exact etiology of XGC remains unknown, its postulated that XGC begins as an inflammatory process and then progresses to a granulomatous reaction that can lead to the formation of a submucosal abscess, and intramural nodules.[4] As a result of serosal perforation of the GB and the spread of inflammatory response, XGC can cause adhesions with the surrounding organs which mimic malignancy.

It is frequently misdiagnosed as GBC due to similar imaging and intraoperative findings consequently leading to extended radical surgery. Therefore, the improvement of pre-operative and intraoperative diagnosis could help to avoid this type of unnecessary extended resection in patients with benign disease.

In our study, out of a total of 61 cases preoperatively suspected of GBC who underwent radical cholecystectomy, we found that the majority of cases 73.77% (45/61 cases) were diagnosed as XGC on histopathology and (16/61 cases) 26.22% were diagnosed as GBC. The study done by Uchiyama *et al.* on 32 patients in 2009 also had 21 patients (65%) having XGC and only 6 patients (29%) having GBC,[8] which is comparable to our study.

Abdominal pain, jaundice, and fever are more frequently observed in patients with XGC as compared to patients with GBC which is similar to the findings of our study.[9] We found that only fever had a significant association with XGC when compared to GBC (75.6% vs. 43.8%). Abdominal pain and jaundice had comparable frequency among GBC and XGC cases.

Jaundice in XGC is usually secondary to stones in the common bile duct, associated with Mirrizzi's syndrome or an underlying GB carcinoma;[10] xanthogranulomatous choledochitis secondary to an inflammatory involvement of biliary tree is a rare cause of biliary stricture leading to obstructive jaundice.

In our study, weight loss (62.5% vs. 2.2%), anorexia (68.8% vs. 6.7%), and palpable mass (37.5% vs. 6.7%) were significantly associated with GBC as compared to XGC.

Thus, clinical features such as fever, anorexia, and weight loss may be used to clinically differentiate GBC from XGC, however, most of these symptoms do not present until cases of advanced malignancy.

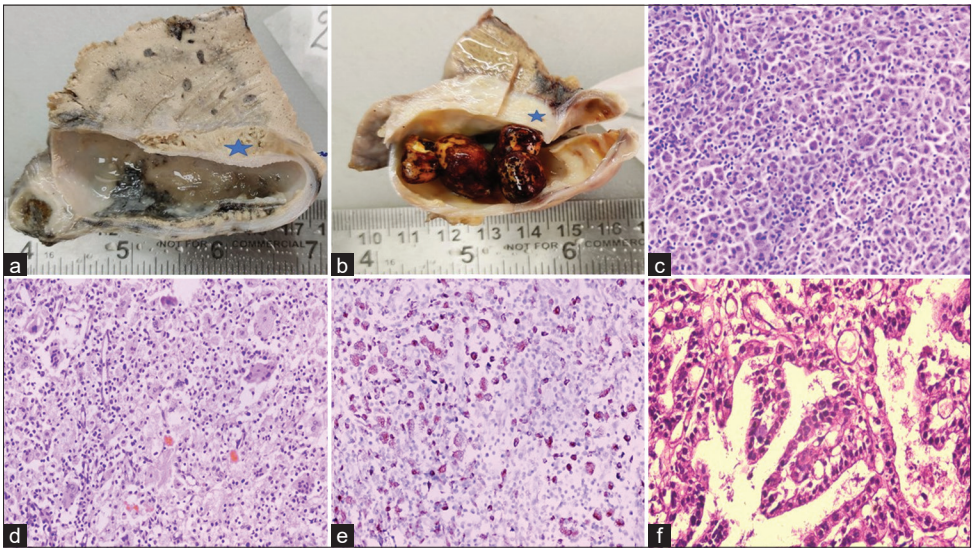

Figure 2: Pathological findings of xanthogranulomatous cholecystitis (XGC) and gallbladder carcinoma (GBC). (a) Diffuse wall thickening of the gallbladder in a radical cholecystectomy specimen (*) (histopathological diagnosis: XGC). (b) Diffuse wall thickening of the gallbladder with cholelithiasis in a radical cholecystectomy specimen (*) (histopathological diagnosis: XGC). (c) Hematoxylin and eosin-stained sections showing sheets of histiocytes along with chronic inflammatory cell infiltrates (×200×). (d) Hematoxylin and eosin-stained sections showing giant cells, and histiocytes along chronic inflammatory cell infiltrates (×200×). (e) CD68 immunopositivity in histiocytes in XGC (×200×). (f) Hematoxylin and eosin stained section showing malignant epithelial neoplasm (GBC) forming glands and acini (×400×)

Raised tumor markers such as CEA, CA125, and CA 19.9 should raise suspicions of GBC. Some studies showed that the increased CA19.9 level (>20 U/mL) had a 79.4% sensitivity and a 79.2% specificity, and the increased CEA level (>4.0 mg/mL) had a 93% specificity, but only a 50% sensitivity.[11,12] Our study showed that CEA (93.8% vs. 0%) and CA125 (62.5% vs. 0%) were significantly raised in patients with GBC as compared to XGC. However, the increased CA19.9 levels were also present in 30 XGC cases (66.7%), which proved to be of no clinical significance in the differential diagnosis of XGC from GBC. Thus, raised levels of CEA and CA125 proved to be a better tool to clinically suspect GBC over XGC.

Usually, radiological findings are helpful in differentiating GBC and XCG. The findings in XGC are diffuse thickening of the wall, hypo-attenuated nodules occupying >60% of the GB wall thickness, severe proliferative fibrosis along with scarring. Although typically considered characteristic of XGC, hypo-attenuated nodules may also be seen in well-differentiated GBCs with abundant mucin production.[13] In our study, hypoattenuated nodules on radiology were significantly associated with XGC (93.3%) as compared to GBC (37.5%). The presence of gallstones on imaging in our study was significantly associated with XGC (88.9%) compared to GBC (12.5%) which was confirmed on subsequent cholecystectomy. This was comparable to similar studies where 84–100% of cases of XGC were associated with gallstone disease.[14-16] European studies have reported the incidence of gallstones in XGC to vary between 92% and 100%.[17]

Goshima *et al.* suggested that diffuse wall thickening, continuity of the mucosal line, presence of intramural hypoattenuating nodules, and absence of the invasion of the adjacent liver parenchyma, and IHBD dilatation confirm the diagnosis of XGC with sensitivity and specificity rates of 83–100%.[18] On imaging, the findings in our study that were indicative of malignancy included mass lesion, loss of interface between GB and liver, along with lymphadenopathy out of which mass lesion (100% vs. 8.9%) and regional lymphadenopathy (75% vs. 37.8%) were found to be significantly associated with GBC. There is too much overlap of the radiological findings between XGC and GBC to reliably differentiate between the two as the former findings may also be found in florid XGC. Therefore, synthesis of all available clinical, serological, and radiological information is necessary to diagnose GBC preoperatively.

The macroscopic findings of XGC include abnormal thickening of the GB wall with poorly demarcated soft-to-firm, yellow–brown intramural nodules of various sizes with cholecystitis.

In our study, gross findings that were found to be significantly associated with XGC were cholelithiasis (88.9% in XGC vs. 12.5% in GBC), diffuse GB wall thickening (100% in XGC vs. 37.5% in GBC), and presence of a definite growth (2.2% in XGC vs. 43.8% in GBC). However, intramural nodules were found in only 8.9% of cases of XGC as compared to 25% of cases of GBC showing no significant association between the two.

On histology, the key features of XGC were foamy histiocytes (100% cases) and inflammatory infiltrate (84.4% cases) indicating a significant association. A few cases of XGC also showed cholesterol clefts and giant cell reaction but it did not show any significant association with XGC. Epithelial dysplasia in GB lining was found to be the key feature of GBC (100% cases) and significantly associated with it.

The diagnosis of GBC and XGC were further confirmed on IHC. CD68 was positive in histiocytes in 100% of cases of XGC while CK was only positive in epithelial lining. In cases of GBC, CK was positive in tumor cells in 100% of cases of GBC while CD68 was negative. This indicates a significant association of CD68 positivity with XGC and CK positivity with GBC.

The features that involve adjacent organs such as GB perforation, abscess formation, fistulous tracts to the duodenum, and extension of the inflammatory process to adjacent abdominal organs such as the liver and transverse colon indicate that XGC develops aggressively like advanced GB cancer. Therefore, it is important to differentiate XGC from advanced GB cancer preoperatively to avoid unnecessary surgical treatment.

In cases of utter confusion, an intraoperative frozen section examination is recommended. However, in daily practice, the possibility of conducting this type of analysis may be limited in some medical centers, especially in cases where cholecystectomies are performed under emergency conditions. Endoscopic ultrasound-guided fine-needle aspiration cytology (EUS-FNAC) can be a convenient and safe method for sampling GB lesions that cannot be differentiated radiologically.[19]

For the ideal approach in XGC, the patient's history, physical examination findings, radiological findings, and pre-operative cytology, if any, should be carefully evaluated.

A simple cholecystectomy is often enough for XGC. Contiguous organ involvement may necessitate extensive resection, despite knowing preoperatively that the underlying disease process is entirely benign. This could have been prevented by a frozen section preoperatively.

Therefore, radical resection done in all our cases is justifiable, although the final diagnosis was benign in the majority of cases.

CONCLUSION

Past acute cholecystitis episodes, acute cholangitis, and choledocholithiasis with CT/MRI findings of diffuse and regular wall thickening along with gall stones and normal serum markers, appear to be more common in XGC patients. Patients with a history of weight loss, anorexia, palpable mass, mass forming lesion on radiology, and regional lymphadenopathy along with raised S. tumor markers preoperatively had significantly higher chances of being diagnosed as GBC on histopathology. The most accurate pre-operative diagnosis is potentially possible by integrating all of these factors. We also recommend the use of EUS-FNAC preoperatively and frozen sections intraoperatively in cases with extremely overlapping symptoms to get an accurate pre-operative diagnosis.

ACKNOWLEDGMENT

We would like to thank all the staff and technicians of the Department of Pathology, BRDMC for helping us block cutting, grossing, and immunohistochemistry without which the article would never have been possible.

REFERENCES

1. Christensen AH, Ishak KG. Benign tumors and pseudotumors of the gallbladder. Report of 180 cases. Arch Pathol 1970;90:423-32.
2. McCoy JJ Jr., Vila R, Petrossian G, McCall RA, Reddy KS. Xanthogranulomatous cholecystitis. Report of two cases. J S C Med Assoc 1976;72:78-9.
3. Giudicelli X, Rode A, Bancel B, Nguyen AT, Mabrut JY. Xanthogranulomatous cholecystitis: Diagnosis and management. J Visc Surg 2021;158:326-36.
4. Guzmán-Valdivia G. Xanthogranulomatous cholecystitis: 15 years' experience. World J Surg 2004;28:254-7.
5. Suzuki H, Wada S, Araki K, Kubo N, Watanabe A, Tsukagoshi M, *et al.* Xanthogranulomatous cholecystitis: Difficulty in differentiating from gallbladder cancer. World J Gastroenterol 2015;21:10166-73.
6. Steinert R, Nestler G, Sagynaliev E, Müller J, Lippert H, Reymond MA. Laparoscopic cholecystectomy and gallbladder cancer. J Surg Oncol 2006;93:682-9.
7. Varshney S, Butturini G, Gupta R. Incidental carcinoma of the gallbladder. Eur J Surg Oncol 2002;28:4-10.
8. Uchiyama K, Ozawa S, Ueno M, Hayami S, Hirono S, Ina S, *et al.* Xanthogranulomatous cholecystitis: The use of preoperative CT findings to differentiate it from gallbladder carcinoma. J Hepatobiliary Pancreat Surg 2009;16:333-8.
9. Poddar E, Mainali P, Shrestha S, Gautam P, Twayana A, Pathak N, *et al.* Xanthogranulomatous cholecystitis mimicking carcinoma gallbladder. Case Reports Hepatol 2023;2023:2507130.
10. Corvera CU, Blumgart LH, Darvishian F, Klimstra DS, DeMatteo R, Fong Y, *et al.* Clinical and pathologic features of proximal biliary strictures masquerading as hilar cholangiocarcinoma. J Am Coll Surg 2005;201:862-9.
11. Misra S, Chaturvedi A, Misra NC, Sharma ID. Carcinoma of the gallbladder. Lancet Oncol 2003;4:167-76.
12. Strom BL, Maislin G, West SL, Atkinson B, Herlyn M, Saul S, *et al.* Serum CEA and CA 19-9: Potential future diagnostic or screening tests for gallbladder cancer? Int J Cancer 1990;45:821-4.
13. Nacif LS, Hessheimer AJ, Rodríguez Gómez S, Montironi C, Fondevila C. Infiltrative xanthogranulomatous cholecystitis mimicking aggressive gallbladder carcinoma: A diagnostic and therapeutic dilemma. World J Gastroenterol 2017;23:8671-8.
14. Goldar-Najafi A, Khettry U. Xanthogranulomatous choledochitis: A previously undescribed mass lesion of the hepatobiliary and ampullary region. Semin Liver Dis 2003;23:101-6.
15. Pantanowitz L, Goldar-Najafi A, Nasser I. Xanthogranulomatous choledochitis mimicking cholangiocarcinoma. Pathol Int 2004;54:213-5.
16. Pottakkat B, Saxena R, Nag HH, Kumari N, Krishnani N. Ampullary xanthogranulomatous inflammation mimicking periampullary cancer: Report of a case. JOP 2006;7:222-5.
17. 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.
18. Goshima S, Chang S, Wang JH, Kanematsu M, Bae KT, Federle MP. Xanthogranulomatous cholecystitis: Diagnostic performance of CT to differentiate from gallbladder cancer. Eur J Radiol 2010;74:e79-83.
19. Akkurt G, Birben B, Çoban S, Akgül Ö, Kulaçoğlu S, Doğanay M. Xanthogranulomatous cholecystitis and gallbladder cancer: Two diseases with difficult differential diagnoses. Turk J Gastroenterol 2021;32:694-701.

How to cite this article: Bharti KS, Agnohotri S, Bundela A, Dubey DB, Rai RK, Vahikar SU, Mitra SK. Xanthogranulomatous Cholecystitis or Gallbladder Carcinoma – A Common Surgical Dilemma: A Prospective Study of 61 Radical Cholecystectomies in a Tertiary Care Hospital in Northern India. Int J Sci Stud 2023;11(8):41-46.

Source of Support: Nil, **Conflicts of Interest:** None declared.