Original Article

Xanthogranulomatous Cholecystitis, A Paradox in Diagnosis & Treatment : A Case Series

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Xanthogranulomatous Cholecystitis (XGC) is an enigmatic variation of Gall Bladder inflammation. Its incidence varies from 0·7% to 10% cumulatively with definite preponderance in India and in far East Countries. Because of its extensive inflammation of varying proportion unmatched with clinical presentation, surgeon more often encounters trouble ended up doing overzealous surgery and histopathology comes as relief or disappointment.

We encountered few cases of XGC during last 1 and half years in Medical College, Kolkata and analysed them retrospectively and came up with some interesting observations.

In our small yet significant case series, we were fortunate enough to experience multiplex presentations of XGC giving impression of both Cholelithiasisand carcinoma mainly during surgery planned for Cholecystectomy. There are few distinctive CT scan findings segregating XGC from carcinoma but they are yet to be authoritative. Other modalities of superior imaging also are not much encouraging. Only and best option is routine use of peroperative Frozen section.

Conclusion : Thanks to its peculiarity yet similarities with both benign and malignant disease, XGC remains to be an disturbing element for the surgeons till any algorithmic approach helped by pre-operative radiology and others comes in the horizon.

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Key words **: Xanthogranulomatous Cholecystitis, Gall Bladder carcinoma. Frozen section.**

Xanthogranulomatous Cholecystitis (XGC) is an enigma. Although for surgeons it is a known entity but the existence of this is repeatedly forgotten till the very histopathological diagnosis comes as a surprise and as a reminder[1,2]. Then, either this unanticipated diagnosis gives a sigh of relief to the patient, relatives and also to the treating surgeon when the possibility of carcinoma is looming large. Or it could be a source of disappointment when already performed radical surgery appeared as an unnecessary exercise.

The reason is although a separate entity, its clinical presentations are nothing indistinguishable from those of different spectrum of Cholelithiasis[3,4]. Rarely it may present with obstructive Jaundice or with diffuse wall thickening in Contrast Enhanced Computed Tomography (CECT) when the suspicion wavers between choledocolithiasis, Mirizzi syndrome and more sinister Gall Bladder Carcinoma (GBC) but unfortunately the thought of XGC remains elusive till the Histopathology Report (HPE) comes. This downside of this missed diagnosis is either overtreatment or undertreatment[5-7].

Editor's Comment :

- Although Xanthogranulomatous Cholecystitis will continue to create diagnostic dilemma and mixed emotion after surgery and HPE, focused radiology and then liberal frozen section still can win us considerable success.

Hence there needs to be an increased awareness of this tumour mimic, particularly in endemic areas like India[1,2]. Identifying the preoperative differences (either clinically or radiologically) between XGC and GBC is imperative, as it would help avert unnecessary morbidity especially in the form of radical surgery. Numerous study were done to search for the CT findings suggesting XGC and few algorithm were made for its early suspicion but they are yet to be of some value in larger scale.

We went through the preserved database of patients operated-upon with a pre-operative diagnosis of calculous cholecystitis with or without suspicion of Gall Bladder Carcinoma (GBC) in the Department of General Surgery of Medical College, Kolkata catering a large number people hailing from either side of river Ganga, the so-called endemic zone for GBC, between January, 2019 and December, 2020 and looked for those cases which eventually demanded more than what was planned. Either it is mere conversion from lap to open or more extensive multi visceral surgery. Of them, we segregated those cases whose final HPE revealed

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Xanthogranulomatous Cholecystitis (XGC) and analysed them in terms of symptoms, signs, laboratory data, operative findings and postoperative progress and found some interesting things which are worth sharing.

Case 1 :

A male patient of 50 came to surgery out patient department with dark yellow urine, eyes and generalised body surface for two months with occasional mild pain in upper abdomen associated with loss of appetite. The Jaundice was progressive in nature. There was no history of (H/O)associated nausea and vomiting, significant weight loss, blood vomiting and black stool or bleeding per rectum. General survey revealed only deep Jaundice and systemic local examination was unremarkable.

The blood picture showed interesting progression of Jaundice. His Liver Function Test (LFT) showed typical picture of obstructive Jaundice ; initially total bilirubin was 6.9 mg/dl (conjugated fraction was 5.5mg/dl) which rapidly progressed to 17.7mg within 3 weeks. The finding of triphasic CT scan (Fig 1),as advised with the suspicion of malignant aetiology, was equivocal showing contracted Gall Bladder with diffuse wall thickening and loss of fat plane at places. CA 19-19 level in blood was within normal range. Meanwhile, another LFT report after 2 weeks showed an interesting change, ie, diminution of total and conjugated bilirubin.

An exploratory Laparotomy was planned with keeping option of per operative Frozen Section. There was a conglomerated mass in the Liver bed, containing part of transverse Colon, Duodenum and Stomach burying the GB inside. The adhesion appeared dense but separable and with painstaking dissection, the Gall Bladder was separated from the rest, pus aspirated from within and Cholecystectomy was done. The specimen was sent to pathology department and its frozen section soon revealed Xanthogranulomatous Cholecystitis. We closed the abdomen after all form of haemostasis and placing a drain inside.

Case 2 :

A male case of calculous cholecystitis was planned for Laparoscopic Cholecystectomy. The relevant history is an episode of acute cholecystitis which needed hospitalisation and conservative management. During interval cholecystectomy, gross adhesions between Gall Bladder with the adjacent organs was noted and a suspected duodenal injury during an attempt to separate GB from duodenum prompted us to convert. On conversion and on attaining further clarity, the Gall Bladder was separated from adjacent viscera. It was thick walled, grossly inflammed and

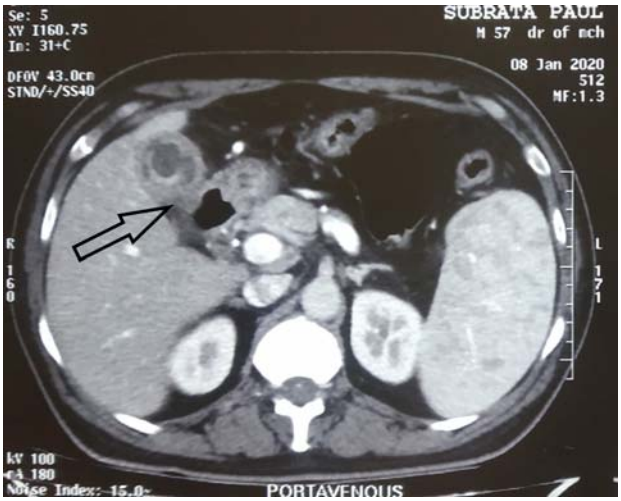

Fig 1 — Showing finding of triphasic CT scan

there was stony hardness at the neck probably due to impacted large stone. Cholecystostomy was done to express out multiple small calculi but hardness at the neck was not due large impacted stone but due to a homogeneous mass at the neck, almost completely obliterating the lumen. There was no apparent lymphadenopathy elsewhere. Partial cholecystectomy with removal of part of GB bearing the suspicious mass and leaving a stump of 1cm length was completed. The leak at the second part of duodenum was repaired. But postoperative drain collection was bilious and unusually high and CECT revealed intact duodenum. An MRCP showed a leak at proximal part of extrahepatic bile duct. ERCP was required to put a stent beyond the leak. Drain collection soon became minimal, patient tolerated semisolid diet well. No further definitive radical surgery, as anticipated was required as HPE revealed XGC.

Case 3 :

The next case was a 76 years old female with H/O of vague upper abdominal pain, vomiting off and on for a duration of 1 month, her abdominal sonography (USG) showed cholelithiasis with thick GB wall. To evaluate further, triphasic CT and MRCP followed. CT abdomen revealed thick walled distended GB but fat plain between GB liver maintained, suspicious lymph nodes at porta, while in MRCP ,CBD was found mildly dilated. A 3.5 mm filling defect within CBD warranted Endoscopic Retrograde Cholangiopancreatography (ERCP), followed by sphincterotomy and Endoscopic Papillary Balloon Dilatation (EPBD).

An open exploration with Frozen Section facility was planned in anticipation difficult surgery and suspicious pathology. A conglomerated mass as discovered demanded extensive adhesionolysis to

segregate grossly distended, very thick walled GB with calculus impacted at the neck from the adjacent viscera eg, the omentum, transverse Colon, Stomach and Duodenum all appeared to be part of gross inflammatory process . Lymph nodes at lesser omentum and hepatoduodenal ligaments (LN 8,12,13) were found enlarged, but there was no ascites or metastasis in liver bed or elsewhere. After cholecystectomy, the GB was cut open to reveal 3 suspicious areas of fatty infiltration, two in body & one in neck. As Frozen biopsy commented only suspicious pathology without any definitive conclusion, surgery was further advanced to removal of liver bed and clearance of lymph nodal basin. Final HPE gave a diagnosis of acute on chronic cholecystitis with xanthomatous changes with no evidence of malignancy in any part of specimen.

Case 4 :

Sonology of a 46 years male patient with pain upper abdomen, being investigated for suspected cholelithiasis, came as a surprise as USG detected thickened and irregular GB wall along multiple calculi, with indistinct fat plane between GB and liver. Suggestion was of CA GB with contiguous extension to Liver.

CT substantiated the USG findings, but also added that the diffusely thickened wall was irregular and appeared discontinuous at places (Fig 2). Fat planes between Liver and GB fundus were indistinct. No metastasis seen. No definite diagnosis was given. CA 19-9 was raised.

Extended cholecystectomy with removal of hepatoduodenal lymph nodes. GB specimen showed impacted large stone at the neck.

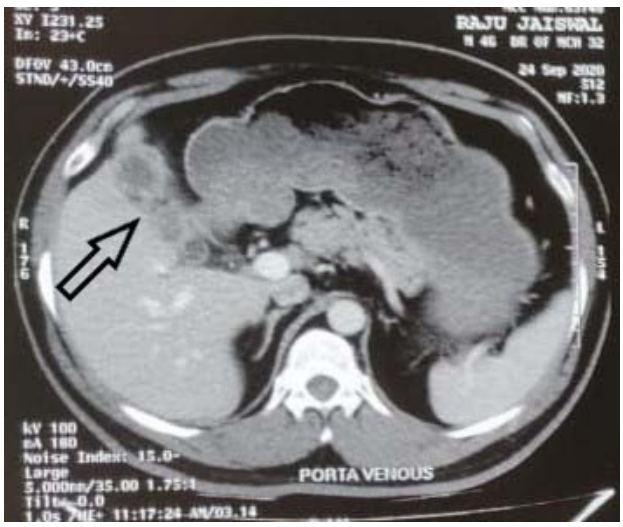

Fig 2 — Showing CT substantiated the USG findings

Second surprise came in the form of HPE which was compatible with XGC with no feature of malignancy. Lymph nodes were reactive.

Case 5 :

Next case is a female patient of 50 with H/O pain abdomen, single bout of melaena ,anorexia and weight loss in last 2-3months.USG revealed GB calculus with irregular thickening of GB wall at fundus. MDCT showed irregular wall thickening at fundus of GB and an oval lesion in the lumen in body region measuring 2.2 x 1.5 cm. There was significant lymphadenopathy in and around porta. In open surgery, the GB was distended, thickening of wall was obvious at fundus. Cholecystectomy was completed and the specimen was readily sent for Frozen Section which reported inconclusively with no suggestion of malignancy. The liver bed of 2 cm margin and few suspicious lymph nodes were removed. HPE came as XGC.

Case 6 :

A planned laparoscopic cholecystectomy was soon converted for difficult anatomy with transverse colon seen occupying the Liver Bed burying the GB underneath. Any attempt to separate and pull the colon up was thought to be beset with danger of colonic injury. On conversion, the overlying part of transverse colon was pulled up carefully from underlying GB, which was small, contracted and part of it seen densely adhered to colon and to structures in the lesser omentum. The part of colon, adhered to GB was suspiciously thickened but rest were apparently okay. At a point of dissection, proximal CBD got injured while separating the GB from it . As there was no provision of frozen section, a multivisceral surgery had to be done in suspicion of malignancy (primary source is either GB or colon) with radical cholecystectomy and hepatico jejunostomy and transverse colectomy and colo-colic anastomosis. The postoperative period was expectedly eventful but patient recovered well, tolerated semi solid in due time and was discharged eventually. The HPE report of GB specimen was of XGC while the suspicious part of transverse colon revealed nonspecific fibrosis. There was no evidence of malignancy.

Case 7 (7A,7B,7C) :

Three female cases had almost similar course of disease and similar per-operative findings and outcome. All were operated in three separate occasions for the diagnosis of cholelithiasis, no H/O acute attack and hospitalization, slated for lap surgery, had to be converted into open surgery because of considerable adhesions and suspected CBD injury.

Two had partial right lateral wall tear while the other one had complete transection of CBD just distal to hilum. Roux-en-Y hepatico jejunostomy (side to side and end to side respectively) along with cholecystectomy was done .In all cases ,the HPE were XGC (Table 1).

Discussion

Xanthogranulomatous Cholecystitis (XGC) is a chronic inflammatory disease of the Gall Bladder characterized by focal or diffuse destructive inflammation with marked proliferative fibrosis along with infiltration of macrophages and foamy cells[5,7]. Surgeons encounter them off and on either in operating theatre or when the HPE report comes to hand. Latter is the commonest. Its incidence ranges from 0.7 to 10%[5,7,8]. Ofcourse, the incidence wise, marked geographical and ethnic variations were obvious in past studies.

In the largest European series to date together with all previously published series in order to gain a worldwide perspective of XGC, 42 (0.9%) were diagnosed as XGC on pathological review out of 4773 cholecystectomies performed at SJUH during the study period[9]. Rather, the cumulative data from India histopathology a far East and America revealed overall incidence of XGC 1.3-1.9%, with the exception of India where it was 8.8% and incidence of GBCa is higher in Indian and Far Eastern populations than in Western populations. There were no patients with XGC associated with GBCa[9].

The male-to-female ratio appears to be equal with little geographical influence although, Indian study reflected a female preponderance in more than one occasion.

The pathogenesis is speculative but reported opinions mostly favoured stasis being the common final pathway, which results increased intra GB pressure, ruptured Aschoff's sinuses, formation bile lake and a final intense inflammatory response to this extravasated bile[10-12]. Occasional wall dehiscence results fistulisation. The association of Gall Bladder Ca with XGC makes the scenario further complicated.

Talking of Indian perspective, Vinodh Dixit *et al* in their study,the so called first study over XGC way back in 1993 reported a surprising incidence of 9.34% among

Table 1 — *All cases with pre-operative and intraoperative findings with final HPE of XGC*

	Any clue	CT Finding	Open/Lap	Frozen, If asked	Operative	Iatrogenic Injury	Excess Surgery/ hazards	HPE
					Adh / Mass / Fistula			
1	Jaundice	Diffusely thick W/FP lost at places	Open	Yes Finding-XGC	Mass of GB/ colon/duodenum			XGC
2	Nil	Not done	Converted	No	Thick Wall GB, Hardness at Neck, Cholecysto-duodenal fistula	Duodenal & CBD (missed)	Duodenal Repair ERCP & Stenting	XGC
3	USG	Thick wall GB, Nodes at porta		Yes Inconclusive	Thick wall GB, Conglomerated mass/nodes		Extended Choli Nodal Clearance	XGC
4	USG	Thick, irregular wall with discontinuity at places	Open	No	Simulates GBC		Do	XGC
5	USG	Thick irregular fundus/Nodes at porta	Open	Yes inconclusive	Thick fundus, Oval lesion in body		Liver bed excision/ Nodal Clearance	XGC
6	Nil	Not done	Converted	No	Mass with Cholecysto-Colic fistula	CBD	Rad Choli+ HJ+ Tr-colectomy	XGC
7A	Nil	Not done	Converted		Gross Adhesion	CBD,lat wall	HJ	XGC
7B	Nil	Not done	Converted		Do	CBD,lat wall	HJ	XGC
7C	Nil	Not done	Converted		Do	CBD transection	HJ	XGC

routine cholecystectomy specimens[13]. By then,no parallel series existed for comparison[13]. Another high incidence of 9% was reported from Japan very soon[12]. As time progressed, XGC continued to make its presence felt in numerous Indian studies with notable geographical variability (eg, Northern *versus* Southern India) quite analogous to the comparable variation in incidence of GBC. Krishnani *et al* in their Indian series of XGC, found a staggering coexistence of carcinoma gallbladder with XGC (19.6% of cases)[14]. A carcinomatous growth can obstruct the cystic duct raising the intra-GB pressure and can trigger the same thing what stones do in other cases. Moreover, malignant process causing breach in mucosa may further facilitate the growing inflammation in the stroma and further sequela[14]. However we did not find any such association in our small series.

The most frequent clinical presentation of patients in the pooled cohort was non-specific abdominal pain followed by other symptoms and signs typical of cholecystitis demonstrating that XGC is typically indistinguishable from cholecystitis on clinical assessment[5,7]. Gall stone is of course the strongest association but not present in every patient, indicating a role for additional aetiological factors[9]. Expectedly, those cases never demanded something extra ordinary unless presentation of Jaundice or lump distorted the picture. In our small series, not a single case has compelled us to think of advising any superior imaging like CECT other than routine sonology just on the basis of clinical grounds except in Case 1. Deep Jaundice in a 50 year old person at presentation and its sharp progression in Case 1 alerted us of malignant aetiology unless proved otherwise prospectively.

USG has its own limitations but surely can guide clinician towards need for further superior imaging, ie, CT scan, MRCP or EUS. Unsuspected thickening of GB wall, presence of mass or polyp in USG can open up the whole conundrum of differential diagnosis like Mirizzi syndrome or GB ca but prediction of XGC hardly arose in the horizon. In case module 1, 3, 4 & 5, unusual thickening of GB wall demanded CECT as next line of imaging.

No doubt, triphasic CT scan is still considered to be the best option around for dispelling the confusion and in making an pre-operative suggestion of XGC. Emphasis is usually made on wall thickening (focal or diffuse), Luminal Surface Enhancement (LSE), presence of intramural hypoattenuating nodules in thickened walls etc[15]. In XGC, Gall Bladder wall thickening can range from 4.0 mm to 18.5 mm and is usually diffuse in nature as observed in 88.9% and 87.8% of patients by two independent researchers Goshima *et al* and Zhao *et al* respectively[16,17]. Focal thickening is less commonly seen in XGC and is more likely to be associated with carcinoma of Gall Bladder[15]. The intramural nodules detected on imaging studies (85.7% and 61.1% by Zhao *et al* and Goshima *et al* respectively) usually represent either xanthogranulomas or abscesses[15-17]. Occupation of a large area of the thickened Gall Bladder wall by intramural nodules is highly suggestive of XGC[6].

XGC is pathology of gallbladder wall where mucosal surface mostly remains intact or hardly focally denuded[15]. On the contrary, carcinoma of Gall Bladder arises from the gallbladder epithelium and causes mucosal disruption in majority of the cases. Mucosal line disruption has been observed in 82.2% cases of carcinoma of Gall Bladderr. A continuous mucosal lining is more often observed with XGC (66.7% of cases) compared to a disrupted mucosal lining (33.3%)[17]. Luminal Surface Epithelium(LSE),defined as enhancement of the Gall Bladder wall predominantly at the luminal surface, was noted in 85.7% of cases by Zhao *et al* and 70% cases by Shuto *et al*[16,18].

Goshima *et al* set out five CT findings (eg diffuse Gall Bladder wall thickening, continuous mucosal lining, intramural hypoattenuating nodules in the thickened walls, absence of macroscopic hepatic invasion and absence of intrahepatic bile duct dilatation) to segregate XGC from carcinoma of Gall Bladder and interestingly found that diagnostic accuracy of XGC increases with the presence of three or more of the above mentioned findings[17].

Next comes the nature and extent of lymph nodal involvement where the observations of different researchers were varied. While Zhao *et al* have described an incidence of 10.2%, Goshima et alfound an incidence of 90%[16,17]. However, presence of regional lymphadenopathy is more prevalent in carcinoma compared to XGC[16]. While 58.9% cases with Gall Bladderr carcinoma had retroperitoneal lymph nodes enlargement, only 10.2% cases of XGC had mild lymph node enlargement (1-1.5 cm in diameter)[16]. In our series, case module 3,4 and 5 showed significant lymphadenopathy at porta but eventual diagnosis was XGC. Finally, CT findings like mass replacing Gall Bladder, intra-luminal mass or polypoidal mass-like thickening were yet to be seen in XC of different studies and same was the case in our series.

Despite those above mentioned distinctive CT features, the preoperative diagnosis remains ambiguous at its best which continues to be so or rather often gets further compounded in operation

invariably resulting iatrogenic injuries or surgical excess.

Now, decision of going forward with laparoscopic method when the possibility of XGC is there, is a matter of debate[19,20]. The intense chronic inflammation in XGC surely make the procedure hazardous and risky demanding painstaking dissection and good tissue respect. Multiple series of XGC has supported the safety of laparoscopy, but there is definitely higher incidence of conversion or iatrogenic injuries[5,20,21]. For us, as there is no luxury of suspecting XGC beforehand, the question of choosing laparoscopy or avoiding it never arose. Rather we went ahead with open method right from the beginning either in anticipation of sinister diagnosis (Case 1&4) or ambiguous pathology (Case 5). However, it is always better to adopt a low threshold for conversion, which enables a better assessment of the lesion and ensures superior outcomes with regard to mortality and morbidity[5,7,19,20].

In our series, the reasons behind the conversion from laparoscopy to open method are either lack of free access to abdomen via umbilicus or difficult anatomy (Case 2,5) or some iatrogenic injury (Case 2) prompted it. In Case 2, the stigma of past documented acute attack of cholecystitis is the likely explanation while in other cases of conversion and further misadventures, undocumented or missed history of acute cholecystitis or pancreatitis was readily blamed because of its potential to result inseparable adhesion. Whatsoever, the thought of XGC never tickled our imagination.

Operative findings in all our cases were varied, quite comparable with multiplex complications of XGC found in other studies, namely perforation, abscess formation, fistulous connection to duodenum or skin, and extension of the inflammatory process to the liver, colon, or surrounding soft tissues[22]. They all contribute to the confusing picture either resembling cases of calculous cholecystitis complicated with repeated attack or acute pancreatitis or simulating that of CA GB. Almost all our cases had gross inflammatory process,some limited to Gall Bladder only while few invading to surrounding viscera often culminating into fistulous connection like duodenum (Case 2), colon (Case 5). Often, it was a conglomerated mass of GB with surrounding viscera (Case 1,3 & 5). Or thanks to wall thickening and local destructive spread of inflammation or a conglomerated mass as in Case 1,3,4 & 5 it appeared as an advanced Gall Bladder carcinoma. Interestingly, it is also reported that both XGC and GBC may coexist, but latter may be masked by the masquerading features of the former[21].

In this scenario of overlapping features and the resultant ambiguity, the frozen section is expected to bring some clarity and help us fix the surgical strategy and avoid those unwanted exercise. Our experience was mixed as in Case module 1, frozen section helped us avoid unwanted excess surgery while in Case 3 & 5, the frozen section failed to comment conclusively hence forcing overzealous surgery for intention of oncological adequacy.

Now if we look back and judge all our cases in comparison with other databases from different studies, some interesting observations came up. Not a single case presented with clinically palpable lump. Jaundice never distorted the clinical picture except Case 1. Except Case 2,not a single case had documentable H/O acute attack of neither cholecystitis nor pancreatitis. So it can be said, unexplained gross and dense inflammatory process, which is typical of XGC ,is mostly a pathological phenomenon but clinically silent. In other way round, any operative field showing considerable adhesions and further sequelae in the absence of explainable past (eg, prior attack of cholecystitis or pancreatitis) should always raise the suspicion of XGC. Secondly, when a conversion (Laparoscopic to open Cholecystectomy) is followed by unplanned multivisceral surgery taking prolonged operating time, XGC must come in the differential diagnoses[21]. We also noted generally longer hospital stay than the usual.

Last but not the least, although XGC is a variant or aberration of Cholecystitis, it's more a radiologically (and of course Histopathologically) appreciable phenomena. Naturally, a senior radiologist has a portend role to play in minimising the misdiagnosis and associated hazards by putting particular stress on certain CT findings like the thickness of the Gall Bladder wall, patterns of wall thickening (focal versus diffuse), continuity of mucosal line (continuous versus disrupted), enhancement characteristics of mucosa (homogeneous versus heterogeneous), presence of submucosal hypo-attenuated nodules or bands and presence or absence of enlarged lymph nodes[8,15].

Talking of other advanced imaging in identifying XGC, Endoscopic Ultrasound (EUS) has shown remarkable accuracy of 93% in segregating GB Ca in a study of patients with suspected XGC and/or GB Ca[23]. But a negative sample cannot be conclusive. Secondly, sampling errors in EUS-FNA is another deterrent factor which limits its widespread applicability in XGC[21,23]. Fluorine-18 Fluorodeoxyglucose Positron Emission Tomography (FDG-PET) also may be effective in diagnosing GB Ca,however, but it should

be interpreted with caution as XGC because of its inflammatory nature may yield a false-positive result[24,25].

So as the above mentioned CT findings are yet to considered conclusive in larger scale and EUS or PET scan still searching for encouraging note, intra-operative frozen section analysis is still considered our best bet in diagnosing XGC. So its liberal use will surely rule out simultaneous occurrence of GBC/XGC, thereby guiding optimum surgery and minimise unnecessary surgery in XGC patients[1,7,8].

Conclusion

Because of few confounding factors, eg, its allegiance to chronic Cholecystitis and GBC, otherwise two extremes of a spectrum, XGC will continue to be an puzzle for surgeons and surgical misadventures is unavoidable. Case series like ours, however small, will have some significant role to play in forcing certain issues. An increased awareness from the part of radiologist with focus on certain features combined with high degree of suspicion of surgeon combined liberal application of intra operative Frozen section can contribute to an algorithmic approach to XGC, thus ensuring appropriateness both to diagnosis and surgery.

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