Xanthogranulomatous Cholecystitis — Sonographic and Computed Tomographic Findings: A Case Report

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Xanthogranulomatous cholecystitis (XGC) is an unusual but not rare disease entity that has attracted particular attention in recent years. We report a case of XGC and describe the imaging findings on ultrasound (US) and computed tomography (CT). Abdominal US demonstrated diffuse wall thickening of the gallbladder with an indistinct boundary, hypoechoic bands and nodules inside the thickened gallbladder wall, and multiple gallstones. Abdominal CT revealed similar findings. The thickened gallbladder wall was well enhanced on contrast CT scan.


KEY WORDS: • xanthogranulomatous cholecystitis • gallbladder disease • chronic inflammation • computed tomography • ultrasound

INTRODUCTION

Xanthogranulomatous cholecystitis (XGC) is an unusual but not rare disease characterized by grayish yellow nodules or streaks in the gallbladder wall, mainly caused by lipid-laden macrophages with an estimated incidence of about 1–2% of all cases of cholecystitis [1,2]. This entity was first described by Christensen and Ishak in 1970 [3], and has attracted particular attention in recent years. XGC is a chronic focal or diffuse destructive inflammatory process of the gallbladder that may occasionally be confused with a malignant neoplasm on the basis of imaging studies. XGC may lead to complications such as perforation, abscess and fistula if not treated properly. Therefore, it is important to make a correct diagnosis preoperatively, especially using medical imaging such as ultrasound (US) and computed tomography (CT). However, only a few studies have characterized the radiologic characteristics of XGC. We present a case of XGC and describe the findings of US and CT.

CASE REPORT

A 78-year-old female presented to our hospital with the chief complaints of intermittent right upper quadrant pain, nausea, vomiting and poor appetite for 3 months. Her medical history included hypertension and duodenal ulcer with bleeding; both were under regular treatment and clinical follow-up. She had lost 10 kg in body weight in the past 3 months.

Her vital signs were unremarkable. On physical examination, the lungs were clear and heartbeat was regular. There was no audible precordial murmur. There was right upper quadrant tenderness, but no
palpable mass or Murphy’s sign. Normal bowel sounds were present and there was no rebounding pain or muscle guarding. After admission, routine laboratory studies including white cell counts and differential counts were within normal limits. Chest radiograph revealed old pulmonary tuberculosis.

US of the abdomen revealed generalized wall thickening and an indistinct gallbladder boundary (Fig. 1). The gallbladder lumen was comparatively small. There were multiple echogenic structures in the gallbladder, representing gallstones (Fig. 1B). The adjacent duodenum and colon had thickened walls.

CT of the upper abdomen demonstrated similar findings (Fig. 2). The thickened gallbladder wall was enhanced at the inner surface (probably) epithelial layer on contrast CT scan (Figs. 2C, D). Hypodense bands were evident in the gallbladder wall (Figs. 2C, D). There was an indistinct boundary between the gallbladder and liver. The boundary of the gallbladder and duodenum was ill defined. There was no evidence of enlarged lymph nodes in the upper abdominal cavity. Percutaneous US-guided needle biopsy of the gallbladder wall was performed and histologic study showed xanthogranulomatous inflammatory changes without evidence of malignancy.

After antibiotic treatment for 2 weeks, she underwent selected open cholecystectomy. A severely inflamed and contracted gallbladder with small

![Fig. 1. Ultrasonography of the right upper abdomen. (A) The gallbladder wall is diffusely thickened with an indistinct boundary (arrows). (B) Layering echogenic material in the dependent portion is demonstrated in the more caudal part of the gallbladder, representing gallstones (arrows). (C, D) Color Doppler ultrasound of the gallbladder demonstrates increased flow in the wall (arrows). The hyperemic change is suggestive of inflammatory reaction or cholecystitis.](image)
gallstones was noted. There was severe adhesion between the gallbladder and the duodenum. The gallbladder was removed after adhesiolysis. Intraoperative cholangiogram showed no dilatation of the common bile duct and bilateral intrahepatic ducts without stone, stricture, or irregularity. The gross specimen showed an opened gallbladder measuring about 12 × 10 × 7 cm with a 1.2 cm thick wall. The mucosal folds were coarse and the serosa was dull. Histopathologic study revealed diffuse chronic inflammatory cell infiltration with focal lipid-laden macrophage accumulation in the gallbladder wall (Fig. 3), consistent with XGC. There was no evidence of malignant cells, abscess or bacteria in the surgical specimen.

The patient had an uneventful clinical course after surgery and was discharged in a stable condition.

**DISCUSSION**

According to the clinical manifestation in our patient, the complex nature of the gallbladder, the adjacent abnormality, and the history of weight loss, the differential diagnoses included acute cholecystitis, chronic cholecystitis, XGC, gallbladder carcinoma, adenomatosis, and metastasis. All these disorders are associated with gallstones and are more common in women. These gallbladder diseases cannot be reliably distinguished from each other preoperatively using clinical or imaging modalities. Moreover, in a minority of patients, they may even coexist [4].

The exact etiology of XGC is uncertain. It may be caused by chronic infection and calculi formation in association with bile stasis, since recurrent inflammation and calculi provoke degeneration and
necrosis of the gallbladder wall and subsequent intramural microabscess formation. These intramural microabscesses are occupied by histiocytes in reaction to the extravasated bile, eventually replaced by xanthogranulomatous nodules [5].

The presence of gallstones, moderate-to-marked thickening of the gallbladder wall and a complex poorly marginated mass on US and CT is suggestive of carcinoma [6]. Itai et al reported gallbladder wall thickening on CT in six of 27 cases (22.2%) with gallbladder carcinoma, but wall thickening was focal in most cases [7]. However, the gallbladder wall can sometimes be diffusely thickened in malignancy and focally thickened in XGC and simple cholecystitis, so the usefulness of this finding is limited. Chun et al concluded that carcinoma was more probable if there were enlarged regional lymph nodes or heterogeneous adenopathy or if there were multiple masses or a large heterogeneous mass extending to the liver [8]. Four groups have reported that the presence of hypoechoic nodules and bands in a thickened gallbladder wall, together with calculi in a patient with chronic gallbladder disease, is highly suggestive of XGC [5,9–11], as in our patient. The nodules and bands can behave as xanthogranulomatous nodules [11]. A more recent study by Shuto et al concluded that luminal surface enhancement (LSE) of the gallbladder wall represents an intact epithelial layer, and intramural low-attenuation areas correspond to xanthogranulomatous lesions or abscesses on CT [12]. They also found that very high signal intensity on T2-weighted magnetic resonance images were helpful in differentiating XGC from gallbladder cancer. Although we did not use magnetic resonance imaging for our patient, the presence of LSE and intramural low-attenuation areas on CT are highly suggestive of XGC (Figs. 2C, D).

The pathologic features of XGC parallel xanthogranulomatous pyelonephritis, and the clinical presentation is usually one of chronic cholecystitis [2]. An association with diabetes and obesity is suggested in some series [10]. Most patients present in their sixth or seventh decade of life [13]. Patients with XGC may present with a high comorbid factor such as perforated gallbladder, abscess formation, or enterobiliary fistula, which were seen in 23% of patients with XGC [1,11]. A high rate of postoperative morbidity has also been reported [14]. Fortunately, these did not happen in our patient.

The role of needle biopsy in the diagnosis of XGC is controversial. The diagnosis may not be reliably established with fine needle aspiration biopsy (FNAB) or core biopsy and, if carcinoma is also present, there is concern that the needle track may be seeded with tumor [10,13]. Demonstration of foamy histiocytes on percutaneous FNAB should be helpful in surgical planning. However, as XGC has the potential for fistula formation, some investigators believe that it is probably contraindicated. It has

Fig. 3. Histopathologic study of a representative specimen. (A) Diffuse chronic inflammatory cell infiltration of the gallbladder wall is associated with focal areas of lipid-laden macrophage accumulation (arrows). (B) In some areas, the lipid-laden macrophages disperse diffusely in the wall.
been suggested that the benign nature of the lesion should be confirmed by intraoperative frozen section for a preliminary histologic diagnosis [14]. Two cases of XGC were diagnosed by FNAB without complication [10,15]. In our patient, there were only transient fever and mild leukocytosis with left shift. These symptoms and signs disappeared in a few days. Further complicating matters, the incidence of carcinoma in XGC is up to 10% [11]. Although preoperative FNAB in our patient demonstrated XGC, there was still some concern about occult malignancy. A negative result on percutaneous FNAB of the gallbladder mass should be interpreted with caution, as almost 50% of cases with negative results in one large series had malignancy [16]. Therefore, if a neoplasm is proximal to the body of the gallbladder and is not infiltrating the wall, a secondarily inflamed gallbladder could be misdiagnosed as XGC [10]. There is general consensus that percutaneous FNAB can be used in patients with suspicious advanced gallbladder carcinoma to avoid unnecessary laparotomy [10].

In conclusion, XGC has certain characteristic US and CT findings, which reflect the histopathologic findings. Knowledge of these findings is helpful in the diagnosis of XGC.

REFERENCES