CASE REPORT

Conversion of laparoscopic cholecystectomy to open surgical procedure due to complete fibrosis of the gallbladder

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SUMMARY

We present a case of transmural fibrotic gallbladder in a patient with previously undiagnosed gallbladder disease and describe our surgical approach to treating this patient.

BACKGROUND

Calculous biliary disease represents the most common cause of gastrointestinal illness requiring hospitalisation in young, healthy individuals.¹ Approximately 25 million Americans have gallstones which represents an estimated 10–15% of the American adult population. Although most patients remain asymptomatic from their gallstones, many can develop symptomatic disease throughout their lifetime. Most symptoms are a result of gallstone blockage of the cystic, common bile or pancreatic ducts resulting in acute inflammatory changes and clinical disease. Approximately two-thirds of these symptomatic patients present with multiple attacks, suggesting ongoing inflammation, chronic obstruction and histological changes within the gallbladder wall. Gallstones in the setting of chronic inflammation have also been linked to the development of gallbladder cancer.²

CASE PRESENTATION

A 35-year-old obese man (body mass index 42.5) presented to our care with ongoing sharp, right upper quadrant abdominal pain lasting several months. There was associated intermittent nausea and vomiting, but no associated symptoms with food intake. He described intense upper abdominal pain with radiation to the back. He had a history of gastroesophageal reflux disease, confirmed by upper endoscopy, for which he took ranitidine appropriately for symptom relief. He did not have a significant smoking, alcohol or illicit drug history, and no previous surgical history.

Examination showed a soft, non-distended abdomen with positive Murphy's sign and mild epigastric tenderness. The patient was afebrile with no increased white cell count. All liver function results were within normal limits.

INVESTIGATIONS

An abdominal ultrasound revealed circumferential wall thickening of the gallbladder, measuring up to 6 mm. There was some dependent sludge, but no acoustic shadowing calculus or pericholecystic fluid. The patient was tender to scanning over the gallbladder. The common bile duct measured

approximately 7 mm. A CT of the abdomen revealed an abnormal gallbladder with moderate gallbladder wall thickening. No definite gallstone could be identified; no pericholecystic fluid could be appreciated (figure 1). A hepatobiliary iminodiacetic acid scan was obtained, which showed non-filling of the gallbladder after 90 min.

TREATMENT

Given his presenting symptoms and imaging signs of ongoing obstruction, the decision was made to proceed with elective laparoscopic removal of the gallbladder. At the time of surgery, the working diagnosis was biliary dyskinesia with possible Mirizzi syndrome owing to the nature of the obstruction with no radiological evidence of gallstone. Intraoperatively, the patient's abdomen was entered using a standard Hassan technique. A 10 mm port was inserted for laparoscopy, followed by three 5 mm trocars in the standard subcostal positions. The abdomen was then inspected and was noted to have a white-appearing fibrotic, hard gallbladder (figure 2), bearing resemblance to a hard-boiled egg, which prevented successful laparoscopic grasping of the gallbladder wall. Aspiration of 35 cc cloudy, yellow-brown fluid was achieved and sent to pathology. An attempt to continue with laparoscopic dissection proved infeasible due to a difficult dissection, obliteration of the triangle of Calot and a very fibrotic porcelain gallbladder with extensive inflammatory reaction and dense adhesions to the peripheral tissues.

Figure 1 CT scan showing no gallstones or pericholecystic fluid.

To cite: Su LS, Burke LH, Ertl CW. *BMJ Case Rep*. Published online: [please include Day Month Year] doi:10.1136/bcr-2013-009406

Figure 2 Intraoperative photo revealing white, fibrotic gallbladder.

The decision to convert to an open procedure was made, and a right subcostal incision was undertaken with appropriate dissection down to the gallbladder. The abdomen was explored, and extensive adhesions of omentum and bowel to the region of the gallbladder were noted. The gallbladder was palpated and found to be very hard. An attempt was made to identify the cystic artery, cystic duct and Calot's triangle. However, these structures were very difficult to identify because of the surrounding fibrinous adhesions. The adventitial and peritoneal attachments were mobilised in a 'dome down' fashion. Intraoperative cholangiogram was performed and showed good bile flow into the duodenum. The gallbladder was completely excised and sent to pathology. A closed suction drain was placed.

OUTCOME AND FOLLOW-UP

The pathology report found dense fibrosis and chronic inflammation within the entire thickness of the gallbladder wall. The gallbladder was measured to be 9.5×5.5×2.5 cm. The gallbladder wall was diffusely brittle and thick, to a maximum of 1.2 cm. Opening of the gallbladder revealed a shiny, grey-white mucosal aspect with scattered foci of haemorrhagic discoloration. There was a single, yellow, 1.2 cm gallstone found impacted in the gallbladder neck. No obvious mass or nodularities were found. Cytology revealed abundant macrophages with no malignant cells. Postoperatively, the patient's initial course was unremarkable and he was discharged home. However, the patient returned 2 weeks later with an abscess, which was treated with antibiotics and supportive management. His subsequent follow-up appointment was negative for any complications.

DISCUSSION

Fibrotic gallbladder has been described in the literature in reference to porcelain gallbladder, a complication of Mirizzi syndrome, xanthogranulomatous cholecystitis, gallbladder cancer and other gallbladder pathologies.^{3–10} Fibrosis itself is a marker for chronic inflammation. The presence of full-thickness fibrosis with a concomitant gallstone lodged within the neck of the gallbladder suggests that this patient suffered a component of Mirizzi syndrome causing chronic inflammatory changes within the gallbladder wall. Mirizzi syndrome is defined into two basic types by the McSherry classification: type I, which is external compression of the common hepatic duct by a stone impacted in the cystic duct of Hartmann pouch, and type II, which is associated with fistula formation between the common bile duct and gallbladder due to chronic inflammatory erosion of the

impacted stone.⁶ Interestingly, the patient did not present with jaundice or elevated liver function tests as expected from a bile obstruction, suggesting either an atypical Mirizzi syndrome with partial obstruction enough to cause chronic gallbladder inflammation but not significant blockage of the common hepatic ducts, or more likely an obstruction of the gallbladder neck only, with no compression on the hepatic ducts.

The gallbladder itself contained full thickness fibrosis with no nodularity, an uncommon finding with no associated calcifications or neoplastic associated pathology. There has been a recent literature surge of xanthogranulomatous cholecystitis, a benign condition that is often confused with gallbladder carcinoma, with the intent of correctly diagnosing the disease preoperatively to prevent larger unnecessary operations. Xanthogranulomatous disease is chronic disease of the gallbladder which is characterised by full thickness gallbladder fibrosis and infiltration of macrophage foam cells.⁸ Usually, the confusion to gallbladder carcinoma arises from associated asymmetrical nodular infiltration into the gallbladder mimicking carcinoma. There were no nodularities found in our patient's gallbladder, nor was there any neoplastic changes found on pathological examination. However, given the intense and diffuse fibrotic changes in addition to the macrophage infiltration, xanthogranulomatous cholecystitis was entertained in this case.

We postulate that this is a case of chronic cholecystitis caused directly by stone impaction into the gallbladder neck resulting in chronic inflammatory changes of the gallbladder. It is known that chronic bile stasis owing to obstruction results in inflammatory changes within the wall of the gallbladder. If given time, fibrosis and scarring occurs which will eventually affect the entire wall. The reason that this type of fibrosis has not been stated in the literature before with no associated features is because most, if not all, patients become symptomatic before dense fibrosis can occur. Consequently, the gallbladder is removed before the progression of the disease can be fully observed. Our case is particularly interesting to our practice, as we have never before observed a gallbladder with the appearance of a hard-boiled egg. The gallbladder was non-functioning, and, combined with the obliteration of surrounding anatomical features, prompted us to convert our laparoscopic procedure to open surgery.

Learning points

- ▶ Symptomatic gallstone is the usual indication for surgery.
- ▶ Chronic cholelithiasis is a risk factor for gallbladder cancer; therefore, all such gallbladders should be removed.
- ▶ The decision was made to convert to open cholecystectomy because of a difficult dissection, obliteration of the triangle of Calot, and a very fibrotic porcelain gallbladder with extensive inflammatory reaction and fibrinous adhesions to the peripheral tissues.

Contributors LSS is the primary author of the manuscript. LHB extensively revised the manuscript and prepared it for publication. CWE was the attending surgeon and primary data collector for the case.

Competing interests None.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

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