

Is it safe to be calcified? Porcelain gallbladder perforation and review of literature

Selcuk Coskun,¹ Lutfi Soyly,² Isa Kilicaslan,³ Fuat Atalay⁴

Abstract

Calcification of the gallbladder wall (porcelain gallbladder) is an intense structure and uncommon manifestation seen in chronic cholecystitis and resulting from chronic inflammation of the gallbladder wall. Patients with porcelain gallbladder are usually considered not at risk of acute cholecystitis. However, sporadic cases of cholecystitis on porcelain gallbladder have been described in literature. Gallbladder perforation is a rare entity and may complicate on acute or chronic cholecystitis in a non-calcified gallbladder. We report an unusual case of acute cholecystitis with perforation in a porcelain gallbladder.

Keywords: Gallbladder perforation, Porcelain gallbladder, Porcelain gallbladder perforation.

Introduction

Porcelain gallbladder is the end result of chronic inflammation of the gallbladder wall. In other words, it is a morphological variant of chronic cholecystitis. Its incidence is 0.06% to 0.8% in cholecystectomy specimens.^{1,2} The pathogenesis of wall calcifications is unknown. Dystrophic calcification is likely a result from inflammatory scarring in gallbladder wall.

Case Report

An 88-year-old man was admitted to the emergency department with the complaint of confusion, somnolence and abdominal pain. In his medical history, he had hypertension, type 2 diabetes mellitus (T2DM), and Alzheimer's disease, loss of vision and severe hearing loss. On physical examination, he was confused and moaned occasionally with vital signs revealing a temperature of 35.4°C, blood pressure 83/42mmHg, pulse 120 beats per minute, respiration 33 breaths per minute, and pulse oximetry 89% on room air. He had non-specific diffuse tenderness and abdominal distension on abdominal

.....
¹Department of Emergency Medicine, Ankara Ataturk Training and Research Hospital, Ankara, ²Department of General Surgery, Ankara Guven Hospital, Ankara, ³Department of Emergency Medicine, Gazi University Faculty of Medicine, Besevler-Yenimahalle, Ankara, ⁴Department of General Surgery, Division of Gastroenterologic Surgery, Ankara Guven Hospital, Ankara, Turkey.

Correspondence: Selcuk Coskun. Email: scoskun_tr@yahoo.com

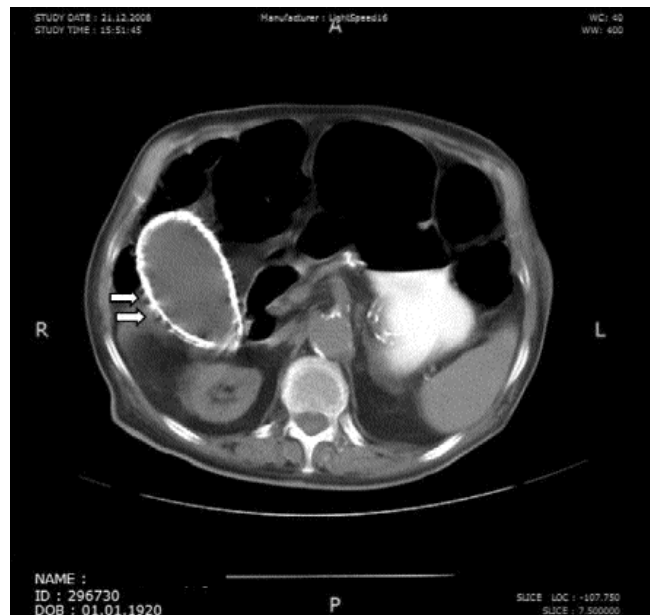


Figure-1: The transaxial enhanced abdominal CT scan of the patient revealed a large porcelain gallbladder (white arrows).

examination.

Laboratory tests revealed white blood cell count (WBC) 22,700/mm³, aspartate aminotransferase (AST) 281U/L, alanine aminotransferase (ALT) 489U/L, gamma glutamyl transferase (GGT) 640U/L, alkaline phosphatase (ALP) 979U/L, amylase 62U/l, glucose 266mg/dL, prothrombin time (PT) 19.3 seconds, activated partial thromboplastin time (APTT) 33.7 seconds, international normalised ratio (INR) 1,671, and total bilirubin 9.3mg/dL with a direct component of 8.6mg/dL. Plain radiography demonstrated a right upper quadrant pyriform opaque mass with curvilinear calcification. Other laboratory tests, electrocardiography (ECG), chest radiography and urinalysis were unremarkable. Contrast-enhanced computed tomography (CT) revealed a large porcelain gallbladder filled with multiple gallstones, and increased arterial flow suggested acute inflammation (Figure-1).

The patient was hospitalised in the intensive care unit (ICU) with the diagnosis of abdominal sepsis, cholangitis and porcelain gallbladder. On the 2nd day of follow-up, planned Endoscopic Retrograde



Figure-2: Photograph of the resected, large porcelain gallbladder specimen.

Cholangiopancreatography (ERCP) could not be performed due to patient's intolerance. On the 3rd day due to deterioration of patient's condition, emergency laparotomy was performed. In laparotomy, stained yellowish green free fluid in peritoneum and a tear over a necrotic area on the neck of the calcified gallbladder near the Hartmann's pouch were detected. Cultures were obtained, irrigation of peritoneum and cholecystectomy were performed. Pathological examination of the gallbladder confirmed the presence of a perforation of calcified gallbladder with no evidence of malignancy (Figure-2). *E. coli* was detected in bile culture. The post-operative recovery was uneventful. The patient was discharged after 6 weeks of follow-up.

Discussion

Evaluation of geriatric patients is always difficult. Because of several factors, including a high frequency of coexisting disease, a variability of symptoms, accompanying cognitive impairment and the inability of some patients, it is difficult to acquire an accurate history. Therefore historical information and physical examination findings are often unreliable.^{3,4} In addition to general problems of elderly patients, our patient had communication problems due to Alzheimer's, as well as loss of hearing and vision. All these factors led to a delay in the diagnosis.

Several studies have reported the incidence of cholelithiasis to be greater than 50% in patients older than 70 years, and acute cholecystitis is the most common cause of acute abdominal disease in the elderly, ranging from 25% to 41% in various series.⁵ Besides, 95% of patients with porcelain gallbladder have cholelithiasis and it is more frequent in women. In our case, the patient was male and in addition to cholelithiasis, cholecystitis, choledocholithiasis and sepsis were present.

Acute cholecystitis may lead to serious complications such as sepsis, pericholecystic abscess, or bilious peritonitis secondary to gallbladder perforation (GBP). Biliary tract disease is the most common indication for intra-abdominal surgery in the elderly.⁵ Biliary pathology accounts for nearly 25% of all suspected abdominal sepsis sources in the elderly. Moreover, perforation and abscess formation should be suspected in those patients with acute cholecystitis whose conditions deteriorate rapidly or who become increasingly toxic for unexplained reasons. In our case, due to the rapid deterioration and elevation of liver function tests (LFT), investigation of sepsis was headed to abdomen and in particular to biliary tract.

It can be difficult to distinguish porcelain gallbladder from entirely-filled cholelithiasis by ultrasonography. This differential diagnosis can be made with an abdominal radiograph, as was done in our case. The appearance on abdominal radiographs consists of tiny curvilinear calcifications in the right hypochondrium.

Approximately 8% to 12% of cases of acute cholecystitis result in GBP, carry a mortality rate of 20%.^{1,5} The relations between the site and the type of GBP have not been elucidated yet. Niemeier, in 1934, classified free perforation of the gallbladder and generalised biliary peritonitis as acute or type I GBP, a localised peritonitis and pericholecystic abscess/collection as sub-acute or type II GBP, and cholecystoenteric fistula as chronic or type III GBP.⁶ Acute or type I perforation was detected in our patient in the operating room.

The fundus of the gallbladder is the most common site of perforation because of its poor vascular supply.⁷ In our case, the perforation site of the porcelain gallbladder was the neck of the gallbladder near the Hartmann's pouch, and it was an unexpected region for perforation. This condition may be fortuitous, or more likely due to the calcification pattern of the gallbladder. Typically, the ostium of the gallbladder is unaffected from calcification. Therefore, in our patient inflammation and ischaemia probably progressed in non-calcified area and eventually resulted in necrosis and perforation of the gallbladder.

Porcelain gallbladder can be classified into complete type and incomplete type. In the complete type, the gallbladder wall is completely replaced by dense fibrosis or calcification. In the incomplete type, calcification of the gallbladder wall is milder. On histopathological examination, multiple dense calcification layers were seen in non-perforated part of porcelain gallbladder. Contrarily, delaminated segments of calcification were detected around the perforation site. Infection had probably contributed to the formation of perforation.

There is an association of porcelain gallbladder and carcinoma, varying between 12% and 61% (8-10). But in our case, pathological examination revealed no evidence of malignancy.

Conclusion

Although patients with porcelain gallbladder are usually considered not at risk of acute cholecystitis and its complications, but cholecystitis and its complication perforation can be seen. Due to a possible calcification pattern, perforation can be seen in an unexpected region.

References

1. Liang HP, Cheung WK, Su FH, Chu FY. Porcelain gallbladder. *J Am Geriatr Soc* 2008; 56: 960-1.
 2. Lee TC, Liu KL, Lai IR, Wang HP. Diagnosing porcelain gallbladder. *Am J Med* 2005; 118: 1171-2.
 3. Kauvar DR. The geriatric acute abdomen. *Clin Geriatr Med*. 1993; 9: 547-58.
 4. Chang C-C, Wang S-S. Acute Abdominal Pain in the Elderly. *Int J Gerontol* 2007; 1: 77-82.
 5. Balsano N, Cayten CG. Surgical emergencies of the abdomen. *Emerg Med Clin North Am* 1990; 8: 399-410.
 6. Niemeier OW. Acute Free Perforation of the Gall-Bladder. *Ann Surg*. Jun 1934; 99: 922-4.
 7. Isch JH, Finneran JC, Nahrwold DL. Perforation of the gallbladder. *Am J Gastroenterol* 1971; 55: 451-8.
 8. Khan ZS, Livingston EH, Huerta S. Reassessing the need for prophylactic surgery in patients with porcelain gallbladder: case series and systematic review of the literature. *Arch Surg* 2011; 146: 1143-7.
 9. Wong SM, Weissglas IS. Gallbladder wall calcification and gallbladder cancer. *J Am Coll Surg* 2013; 216: 1223-4.
 10. Smith PW, Farrar RA, Evangelista RS. Intrahepatic porcelain gallbladder: coexistence of pathologies. *Am Surg* 2012; 78: E414-5.
-