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A rare complication of cholecystolithiasis: perforation of the gallbladder

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Abstract

Common complications of cholelithiasis include gallbladder inflammation, cholangitis, and acute pancreatitis. On the other hand, rare complications include gallbladder carcinoma, gallstone ileus, Mirizzi syndrome and gallbladder perforation. Some patients with cholecystitis do not require hospitalization. It is of key importance for proper further diagnosis and treatment to remember about the possible complications of cholecystitis, especially in oligo-symptomatic patients. Therefore, ultrasound examinations in patients with a history of gallbladder stones should be performed with appropriate care. Ultrasound should be performed carefully in patients with a history of cholecystolithiasis, especially those oligosymptomatic, bearing in mind that there are some rare complications of this highly common clinical entity.

Introduction

(Acute) cholecystitis is a common reason for reporting to an Emergency Ward. Despite high sensitivity of ultrasound in this disease, the possibility of other complications should be kept in mind in the case of persisting inflammation (especially not treated one). Particular vigilance is needed in oligosymptomatic patients.

Case report

A 67-year-old patient was referred to an Ultrasound Diagnostic Unit for abdominal examination. She complained of yellowing of the skin of the abdomen and sclera, which appeared on the same day in the morning. She had already been diagnosed with gallbladder stones. Her medical history was otherwise irrelevant and she was not taking any medications on a daily basis. She admitted that moderate, intermittent pain in the right upper quadrant had appeared a few days before (2–3 days), but it resolved spontaneously.

On the day of the examination she was cardiovascularly and respiratorily stable, with C-reactive protein level elevated to 128.4 mg/L (reference: <5.00) and leukocytosis of $19.4 \cdot 10^3/\mu\text{L}$ (reference: 4.0–10.0) with a relatively high neutrophil count.

There were biochemical signs of cholestasis – a bilirubin level of 16.5 mg/dL (reference: 0.3–1.2) with predominant direct bilirubin, high activity of GGTP (gamma glutamyl transpeptidase) and LDH (lactate dehydrogenase), and mildly elevated activity of transaminases. Pancreatic parameters were normal, without signs of acute pancreatitis.

During abdominal ultrasound (Samsung RS85A, convex probe CA1-7A), the patient was not in pain, with negative sonographic Murphy's sign. Liver enlargement up to 174 mm in the AP (antero-posterior) dimension in the mid-clavicular line and diffuse hyperechogenicity, which are sonographic signs of liver steatosis, were found. The gallbladder wall was markedly thickened (up to 13 mm) and

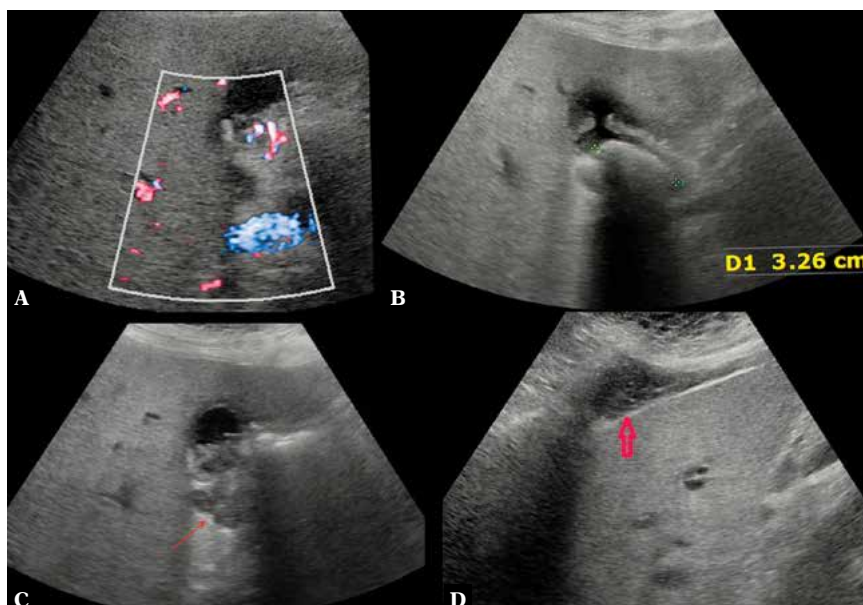


Fig. 1. US of the gallbladder presenting hypervascularization of the wall (A), with a gallstone located in the neck (B) and perforation (red arrow) (C). The largest collection of fluid in the subphrenica location (red empty arrow) (D)

hypervascular (Fig. 1A), contracted in the neck and part of the body on a 33 mm stone (Fig. 1B). The further part of the wall of gallbladder body was discontinuous – sonographic signs of perforation (Fig. 1C). Fluid was observed in the gallbladder fossa and near the round ligament. The largest fluid collection, measuring about 17 × 56 mm, was seen subphrenically (Fig. 1D), with sonographic appearance of a developing abscess. The concrement in the gallbladder neck was compressing the common bile duct (CBD). The CBD course was only possible to be tracked in the hepatic hilum, its outlet was sonographically inaccessible. The dilation of intrahepatic bile ducts of both liver lobes and of CHD (common hepatic duct) (up to 7.4 mm – Fig. 2) was noted, proximally to the site of CBD compression. Other abdominal organs were without significant abnormalities.

The patient was admitted to the General and Vascular Surgery Department. Computed tomography of the abdomen with intravenous administration of contrast agent revealed a concrement measuring about 19 × 30 mm in



Fig. 2. US of dilated CHD up to 7.4 mm due to compression of CBD

the gallbladder neck (Fig. 3A). The gallbladder wall was partially contracted on the stone and thickened (up to 18 mm in the fundus) (Fig. 3B); stranding in the surrounding fat was noted. A suspicion of covered perforation of the gallbladder accompanied by fluid collection or abscess was raised, even though the fat tissue around the gallbladder fundus was without other abnormalities or signs of free gas (Fig. 3C). CHD was dilated up to 7.2 mm (Fig. 3D).

Open cholecystectomy was performed, during which the inflammatory changes of the gallbladder fundus and body were found. Perforation and necrosis at the base of gallbladder due to 3–4 cm concrement in the gallbladder neck were seen. What is more, due to compression, the gallstone caused CHD and CBD necrosis. The gallbladder and the necrotic part of the bile tract was resected, but the reconstruction of the biliary tract was postponed due to patient's condition (presence of inflammation and pus). A drain was placed in the subhepatic region to collect bile from the biliary tract and possible purulent content from the inflamed region. If the conditions are favorable and the operating team has experience in biliary tract reconstructions, it is performed simultaneously with cholecystectomy. Unexperienced operators may put a drain in the subhepatic area under the damaged bile duct and later refer the patient to a high-volume center for biliary damage. In our case, due to pronounced inflammation, necrosis and purulent content, simultaneous reconstruction was postponed.

On the eighth day after the operation, the patient underwent an endoscopic retrograde cholangiopancreatography, which revealed a post-surgical bile leak. Distal CBD was selectively catheterized; cholangiography showed amputation about 3 cm from the ampulla, without concrement-related filling defects. After sphincterotomy, it was attempted to go further to the proximal bile ducts, but without success. Occlusive cholangiogram was performed.

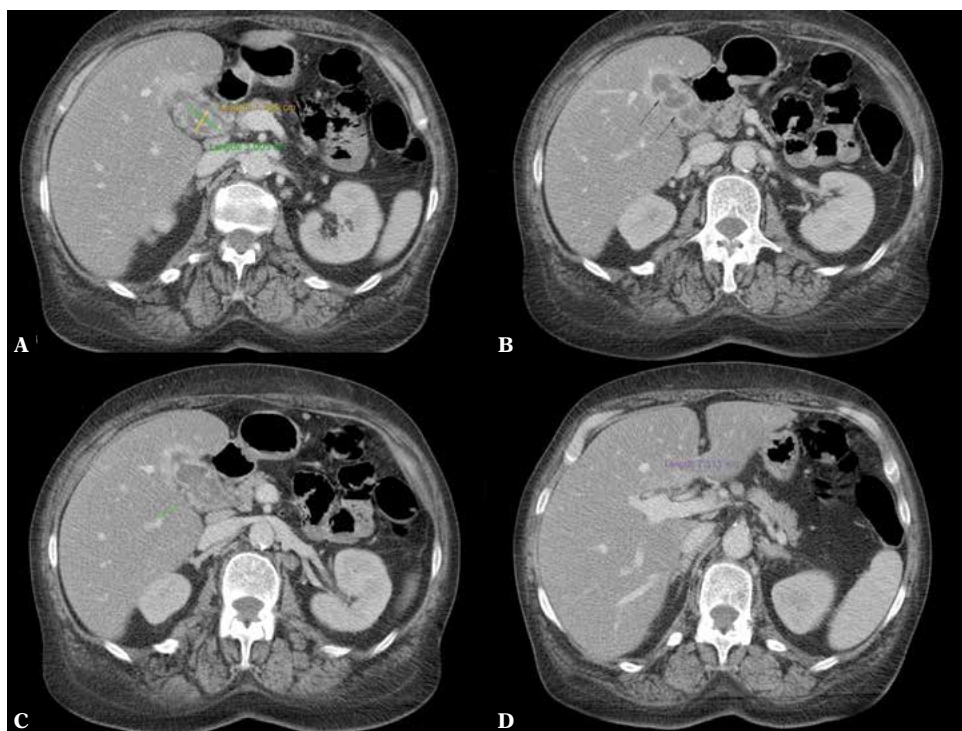


Fig. 3. CT in coronal view, portal phase presenting poorly calcified concrement in the gallbladder neck measuring 18×30 mm (A), contracted gallbladder wall with strictures (purple arrows) (B) and location of covered perforation (green arrow) (C). Dilated CHD measuring 7.5 mm (D)

On the tenth day the patient was re-operated and underwent biliary reconstruction (left and right hepatic ducts) with Roux-en-Y hepaticojejunostomy.

Discussion

Cholecystolithiasis is a common disease, especially in western countries⁽¹⁾. It is the leading reason for admissions to hospital for gastroenterological reasons, and its frequency is estimated at around 20% in Europe⁽²⁾.

The factors predisposing to the formation of gallstones include: older age, female sex⁽³⁾, genes, pregnancy, diabetes, overweight and rapid weight loss⁽⁴⁾. Some groups of drugs may also increase the risk of gallstone formation: fibrates, ceftriaxone, somatostatin analogs⁽⁵⁾, oral contraceptive therapy⁽⁶⁾. Other factors include cirrhosis, Crohn's disease, and hyperbilirubinaemia⁽⁷⁾.

Asymptomatic patients constitute the majority of patients with cholecystolithiasis, which is found accidentally during imaging examinations, such as abdominal ultrasound. Approximately 15–25% of asymptomatic patients may develop symptoms after 10–15 years of follow-up⁽⁸⁾. Symptomatic patients most often report symptoms of biliary colic before other manifestations and complications of gallstone disease (cholecystitis, pancreatitis or cholangitis) occur. Patients with uncomplicated disease have a risk of complications of approximately 2–3% per year as opposed to patients with previous complications, where the risk of developing further, often more severe

complications is about 30% per year⁽⁹⁾. Asymptomatic or oligosymptomatic patients with complications of cholelithiasis without previous episodes of biliary colic are very rarely encountered. In this specific group of patients, risk factors include diabetes mellitus (DM), male sex, older age, elevated white blood cell count and coronary heart disease^(10,11). In our case, the patient complained of yellowing of the skin of the abdomen and sclera that appeared on the same day in the morning, without typical symptoms of cholecystolithiasis.

In symptomatic disease, typical complaints include dull pain in the right upper quadrant, upper abdomen or (less commonly) lower abdomen, which may radiate to the back (especially the right shoulder blade). The pain is often accompanied by profuse sweating, nausea and vomiting⁽¹²⁾. Among the unusual symptoms, the following should be distinguished: belching, early satiety, bloating, epigastric or retrosternal heartburn, chest pain, nausea and vomiting, non-specific abdominal pain.

Complications of cholelithiasis include inflammation of the gallbladder, which is the most common complication characterized by a set of symptoms: pain in the right upper quadrant, fever, leukocytosis, which are associated with inflammation of the gallbladder. On ultrasound examination acute cholecystitis typically demonstrates gallbladder wall thickening (greater than 4 to 5 mm) with hypervascularization or edema (double wall sign corresponding with pericholecystic fluid), a positive sonographic Murphy sign, and the presence of single or multiple gallstones. Choledocholithiasis, with or without inflammation of bile

ducts, refers to the presence of deposits in the bile ducts, for example in the common bile duct. Cholangitis is characterized by a triad of symptoms (triad of Charcot): fever, jaundice and pain in the upper abdomen (that may radiate to the right shoulder blade). Acute pancreatitis might be caused by gallstone passage into CBD and result in a reflux into the pancreatic duct, either due to impeded outflow from the pancreatic duct or because of obstruction at the level of papilla. Patients with acute pancreatitis related to gallstone impaction in CBD outlet may additionally present with elevated hepatic parameters (bilirubin, alkaline phosphatase, transaminases). Less common causes of cholecystolithiasis include gallbladder carcinoma, gallstone ileus, Mirizzi syndrome (obstruction of CHD due to a concrement in a gallbladder neck or a cystic duct) and gallbladder perforation. In the presented case, the patient was oligosymptomatic, both before and on the day of initial diagnosis. Careful sonographic scrutiny helped to make the initial diagnosis of cholecystitis with perforation and abscess in the right subphrenic space, which was later confirmed by

CT and intraoperatively. Neither US nor CT showed the signs of CHD and CBD necrosis, which were seen during the surgery. The results of laboratory tests reflected the diagnostic imaging findings.

Conclusions

The presented case indicates that, even if the patient with cholelithiasis is oligosymptomatic, one should always be alert and consider not only the common, but also the rare complications of this disease.

Conflict of interest

Authors do not report any financial or personal connections with other persons or organizations, which might negatively affect the contents of this publication and/or claim authorship rights to this publication.

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