Open Journal of
Clinical & Medical
Case Reports

Volume 7 (2021)
Issue 11

ISSN: 2379-1039

Subclinical gallbladder perforation

Mendes João*; Couto José; Martins Aires; Monteiro Conceição; Midões Alberto

***Corresponding Author: Mendes João**
Department of General Surgery, Unidade Local de Saúde do Alto Minho (ULSAM), Portugal.
Email: joao14.71@gmail.com

Abstract

Gallbladder Perforation (GBP) is a rare, but potentially fatal, disease varying in presentation and therefore is a dilemma for early diagnosis. It is frequently associated with acute calculous cholecystitis. Most perforations are subacute, causing a pericholecystic abscess. Acute free perforation with bile peritonitis and chronic perforation with an internal biliary fistula are rare. Although clear evidence is lacking, mortality has been reported to be as high as 70%. Here we report a case of subclinical GBP due to acute cholecystitis, followed by a review of the literature for similar cases and other clinical presentations of this condition.

Keywords

gallbladder; perforation; subclinical; gallstones; biliary; abdominal; surgery.

Abbreviations

GBP: Gallbladder perforation; RUQ: Right upper quadrant; CRP: C-reactive protein; ALP: Alkaline Phosphatase; GGT: Gamma-glutamyl transferase; AST: Aspartate transaminase; ALT: Alanine Transaminase; CT: Computed tomography; MRI: Magnetic resonance imaging; MRCP: Magnetic resonance cholangiopancreatography; ERCP: Endoscopic retrograde cholangiopancreatography.

Introduction

Gallstone disease is a worldwide medical problem and is often described as a major affliction in modern society, affecting up to 10% of the adult population in wealthy nations [1,3]. However, a proportion of patients are asymptomatic and remain undiagnosed until the occurrence of complications. Common complications include acute cholecystitis, biliary obstruction, acute pancreatitis and cholangitis. Severe complications include Gallbladder Perforation (GBP), Mirizzi syndrome and fistula formation, which are usually associated with significant morbidity and mortality [2]. Approximately 1% to 2% of patients with gallstones are reported to develop secondary acute cholecystitis, among which 2% to 11% will progress

to GBP, a rare but life-threatening complication. GBP represents a special diagnostic and surgical challenge because of the high mortality (as high as 70%) caused by a delay in the correct diagnosis and subsequent adequate surgical treatment. It is described that only 3% of patients with GBPs are timely and correctly diagnosed before surgery, and the remaining patients succumb to this disease due to a delayed diagnosis [3].

Perforation is thought to start with occlusion of the cystic duct, resulting in retention of intraluminal secretions. Distention of the organ with a consequent rise in intraluminal pressure impedes venous and lymphatic drainage, leading to vascular compromise and ultimately to necrosis and perforation of the gallbladder wall [1]. The fundus is the most distal part with regards to blood supply, which makes it the most common site for perforation (that can occur as early as 2 weeks or several weeks after the onset of cholecystitis) [5]. It is also known that in cases of emphysematous cholecystitis, aerogenic bacteria play a major role in inflammation and are responsible for a high perforation rate [7].

According to Niemeier (1934), perforations are classified into three categories: type I (acute), includes patients with free perforation into the peritoneal cavity; type II (subacute), describes patients with localized perforation and type III (chronic), patients with cholecysto-enteric fistulas [1]. Conditions such as cholelithiasis, infections, malignancy, diabetes, atherosclerosis and steroid therapy are predisposing risk factors for GBP [6].

The clinical presentation of GBP may not be different from uncomplicated acute cholecystitis or, at times, its presentation may perplex the clinician [4]. There are no classical symptoms or signs of perforation, thus the diagnosis is challenging. Right upper quadrant (RUQ) pain, palpable RUQ tenderness, or high fever are regarded as the cardinal symptoms of GBP. These symptoms, however, may be caused by various factors and lead to a misdiagnosis. Thus, it is of the highest importance to distinguish patients with GBP from those with acute abdominal pain [3].

Here we present a case report of a subclinical GBP, followed by a review of literature for similar cases and other clinical presentations of this condition.

Case Report

A 58-year-old female presented at the surgical emergency with pain in the upper abdomen that had started 3 days before the current episode and with increasing intensity in the previous hours. The pain was mainly epigastric and in the right hypochondrium, accompanied by nausea. The patient denied fever, diarrhea, vomiting, choluria, acholia or any other accompanying symptoms. According to her known personal medical history the patient had hypertension, type 2 diabetes (non-insulin treated) and dyslipidemia, medicated accordingly. During examination, the patient was apyretic (36.4^{0}C), with normal blood pressure and heart rate. The patient was in good general condition, hydrated, ruddy, anicteric and eupneic. On abdominal examination, it was tender on the right hypochondrium, without guarding or other signs of peritoneal irritation. Murphy's sign was negative. The abdomen was soft, without distension or any palpable mass. Her blood tests at admission revealed a normal hemoglobin, white blood cell count and CRP value.

Bilirubin levels were elevated (total - 5.54 mg/dL; direct - 3.39 mg/dL), ALP - 267 U/L, GGT - 541 U/L, AST - 450 U/L, ALT - 705 U/L, amylase 39 U/L and lipase 45 U/L. Abdominal ultrasound showed a steatotic hepatomegaly and gallstone disease, with a 17 mm calculus in the gallbladder lumen, but no radiological signs of acute cholecystitis. The intrahepatic bile ducts were not dilated and the common bile duct was apparently normal, although it wasn't clearly observed due to some gas interposition. The rest of the examination was normal. The patient was admitted to the surgical ward for further investigation.

Investigation

During her stay at the hospital, the altered blood tests remained elevated for the first 2 days, after which they started to diminish, without any specific treatment other than analgesia and intravenous crystalloid solutions. Abdominal CT scan (Figure 1) confirmed the presence of a hepatic steatosis and gallstone disease. The gallbladder had borderline dimensions (with a transverse diameter of 38 mm), minimum wall edema and no adjacent inflammation or pericholecystic fluid. The common biliary duct and Vater papilla were slightly hyperintense, measuring 7 mm wide.

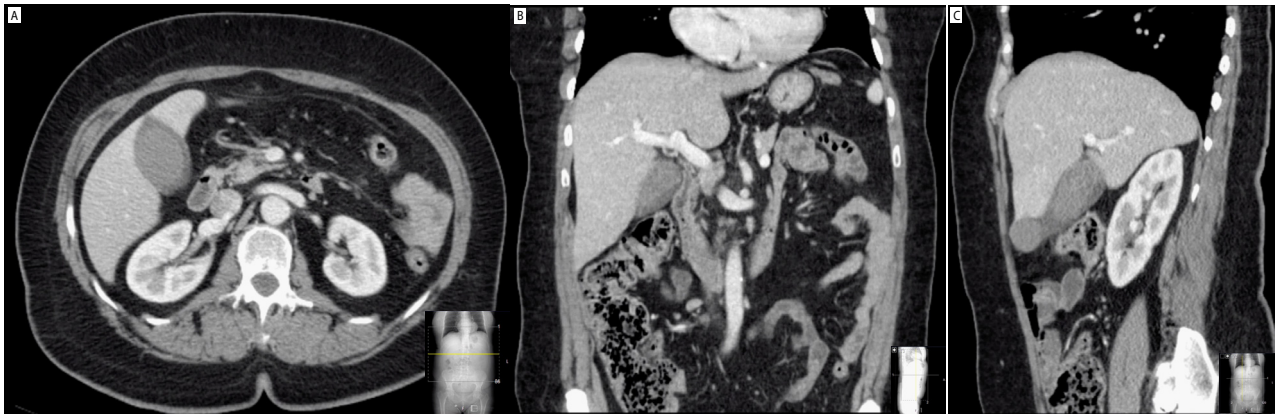

Figure 1: (A) Axial, (B) Coronal and (C) Sagittal sections of an abdominal CT scan. A dense content fills the lumen of the gallbladder, which is slightly distended (38 mm in transverse diameter). A probable large calculus can be seen at the fundus of the gallbladder in image C. Note the close relationship between the right colon, duodenum and distal portion of the stomach, in image B.

ERCP revealed a normal common bile duct. There was no outgoing calculus after sphincterotomy and exploration with the Dormia basket.

The patient was discharged after normalization of blood tests and disappearance of abdominal pain. An abdominal MRI/MRCP was requested in order to assess the existence of residual lithiasis in common biliary duct. The patient was reassessed in a General Surgery consultation on the following week.

Abdominal MRI/MRCP scan (Figure 2) exhibited very suggestive radiological signs of acute cholecystitis. The gallbladder was distended, multilithiasic, with a thickened wall and surrounding edema. The liver exhibited perfusion alterations in segment IV due to contiguous inflammation from the gallbladder. There was no evidence of GBP or abscess in the surrounding area. The intra and extrahepatic bile ducts were normal, with a common bile duct measuring 7 mm, without evidence of biliary obstruction. The remaining exam was normal.

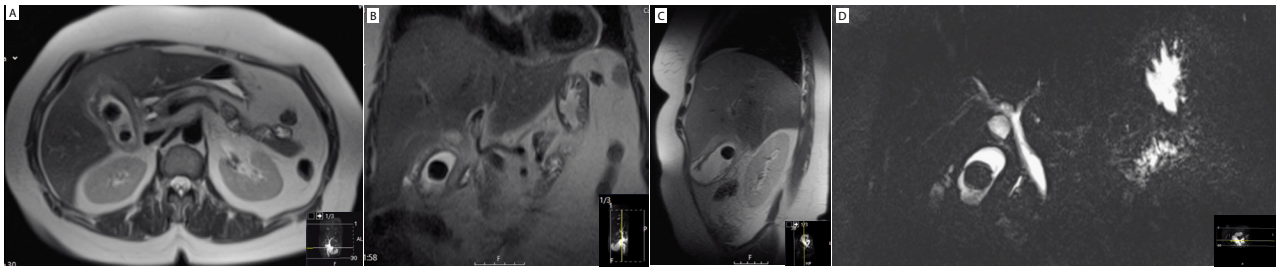

Figure 2: (A) Axial, (B) Coronal and (C) Sagittal sections of an abdominal MRI scan. A large calculus can be seen at the fundus of the gallbladder, which is distended, with a thickened wall and surrounding edema. (D) MRCP reveals an unobstructed common bile duct, with passage of contrast to the small bowel.

Despite these changes on the MRI scan the patient was asymptomatic, so no medical treatment was given. The patient was booked for surgery. During this period, the patient remained asymptomatic.

Surgery and histology

The patient was admitted for elective laparoscopic cholecystectomy about 3 weeks after the consultation. At first inspection of the abdominal cavity, the gallbladder wasn't visible due to an extensive inflammatory process in the area, with involvement of the transverse colon, distal part of the stomach and omentum. After thorough dissection, a GBP was spotted. The perforation was located in the fundus of the gallbladder, with a large calculus within the perforation (Figure 3 A and B). After careful judgement of the potential risks and benefits, the procedure was converted to open cholecystectomy and carried out without complications.

The histological evaluation was compatible with acute cholecystitis. A 2.2 cm calculus (Figure 3 C) was observed.

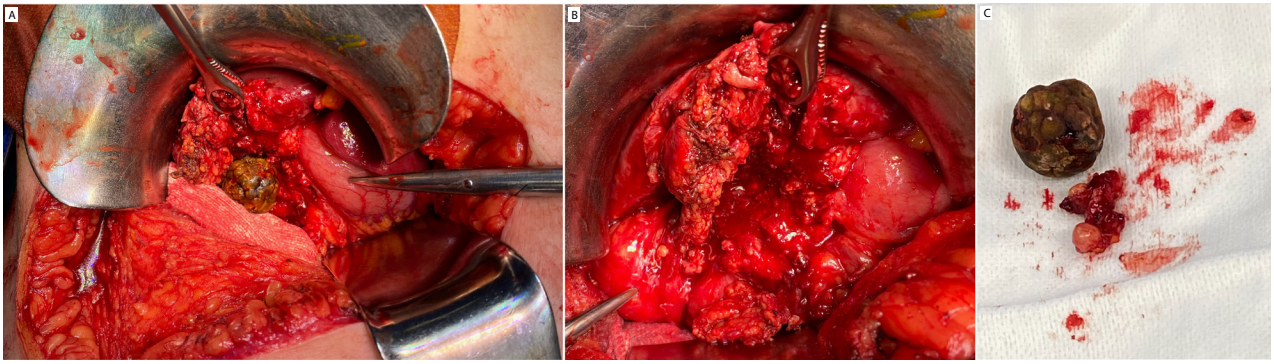

Figure 3: Intraoperative images showing a perforated gallbladder and a bulky gallstone (C). Note the intimate relationship with the stomach (A) and colon (B).

Outcome and follow-up

The postoperative period was uneventful. The patient was discharged 5 days after surgery and reassessed in consultation. She remained stable, with a good overall status. No long-term complications from the surgery were acknowledged.

Discussion

We felt that this was a very unusual case due to its presentation and clinical course. This case of GBP presented without any major symptoms that would have led the patient to resort to a hospital facility.

Instead, the patient remained asymptomatic or with minor symptoms, at home. Additionally, none of the examinations performed were able to demonstrate or suspect an eminent perforation.

At first, the patient presented at the emergency with abdominal pain and obstructive jaundice. It was found that the patient had fatty liver and gallstone disease, but there was no evidence of acute cholecystitis, cholangitis, pancreatitis, perforation or other complication of the gallstone disease. The changes found in the blood tests could be explained by the migration of a gallstone and impaction on the common hepatic duct, transiently. The presence of a slightly dilated bile duct (7 mm) favors this option. Another explanation, although less likely, would be an intermittent Mirizzi syndrome, caused by impaction of a large calculus in the cystic duct or neck of the gallbladder, followed by mechanical obstruction of the common hepatic duct (by the calculus itself or by secondary inflammation). The presence of a large calculus (2.2 cm) in the histologic evaluation and extensive inflammation in the MRI scan, extending from the gallbladder into the liver, could justify this scenario. After the spontaneous resolution of the patient's clinical condition and hospital discharge, it was uncovered that the patient's MRI scan was very suggestive of acute cholecystitis, although the patient was asymptomatic at the time. An elective laparoscopic cholecystectomy was performed 3 weeks later, with an unexpected GBP verified intraoperatively. One can assume that this unique course can be justified by a subacute onset along with the patient being diabetic which, on one hand, confers an increased risk for perforation, and on the other, can be responsible for some extent of neuropathy or reduced threshold for pain.

After searching the literature, we discovered very few similar cases. There are several case reports of spontaneous and acalculous perforations of the gallbladder, but all of them attended with severe abdominal pain and/or signs of peritoneal irritation. In a single center retrospective study of 32 cases of GBP, 7 cases of type III (chronic) perforations were diagnosed while the patients were undergoing laparoscopic cholecystectomy for symptomatic cholelithiasis. Most of type I GBPs were diagnosed intraoperatively (during exploratory laparotomy for perforation peritonitis) and type II perforations were diagnosed by enhanced abdominal CT [5]. In another study, a single center 2-year prospective study of 16 cases of GBP, the clinical presentation included general symptoms like abdominal pain, fever and vomiting. The authors reported 2 cases with atypical presentation with anterior abdominal wall abscess [4].

Clinical diagnosis of GBP (idiopathic or secondary) is very difficult and often delayed or missed because there are no classical symptoms and signs of this condition. Some authors suggest that a sudden decrease in pain intensity caused by the relief of high intracholecystic pressure might herald the perforation [1]. When the gallbladder is perforated at the fundus, it generally results in generalized peritonitis (type I). In our case, the perforation was at the fundus, but the surrounding omentum coated the perforation, avoiding a peritonitis. If the perforation site is other than the fundus, it is easily sealed by the omentum or the intestines and the condition remains limited to the right hypochondrium with the formation of a plastron and pericholecystic fluid or abscess (type II). The fistulous tract forms (type III) arise from the gradual erosion of the chronically inflamed and densely adherent wall of the gallbladder and stomach [4].

Ultrasonography is usually the initial radiological investigation done in most of the cases but fin-

dings are very non-specific for GBP and mimic those seen in acute uncomplicated cholecystitis. Distention of the gallbladder and edema of its wall may be the earliest detectable signs of imminent perforation. CT scan is the most sensitive tool to diagnose GBP, with a reported sensitivity in the detection of GBP and biliary calculi between 88% and 89%. The gallbladder can show direct signs of perforations, such as calculi outside the gallbladder or a ruptured segment of the gallbladder wall. Indirect indicators include the presence of an abscess outside the gallbladder and the presence of gallstones together with thickening of gallbladder wall [6]. The advantage of MRCP is its superb ability to detect stones in the bile ducts, biliary dilatation, and the relationship of a pericholecystic fluid collection to the abdominal wall and gallbladder [5].

Conclusion

In conclusion, GBP represents a special diagnostic and surgical challenge. Patients usually present with general symptoms commonly shared by different abdominal conditions (cholecystitis, pancreatitis, cholangitis, etc.) therefore, it is difficult to diagnose this condition at once. Radiological confirmation of the diagnosis is required, especially in cases of type II and III perforation. Nowadays, improved diagnostic and therapeutic modalities (endoscopic, laparoscopic or percutaneous) represent a major step forward on managing gallbladder disease and its complications.

Disclosure: The authors declare no conflict of interest.

References

1. Chiapponi C, Wirth S, and Siebeck S. Acute Gallbladder Perforation with Gallstones Spillage in a Cirrhotic Patient. World Journal of Emergency Surgery. 2010; 5: 7–10.

2. Başara I and Mustafa S. Spontaneous Asymptomatic Gallbladder Perforation. Quantitative imaging in medicine and surgery. 2014; 4: 212–13.

3. Shi X et al. Gallbladder Perforation in a Patient with Alcoholic Liver Cirrhosis and Asymptomatic Gallstones. Medicine (United States). 2018; 97: 9–12.

4. Patel G et al. Gallbladder Perforation: A Prospective Study of Its Divergent Appearance and Management. Euroasian Journal of Hepato-Gastroenterology. 2019; 9: 14–19.

5. Gunasekaran G et al. Gallbladder Perforation: A Single Center Experience of 32 Cases. Korean Journal of Hepato-Biliary-Pancreatic Surgery. 2015; 19: 6.

6. Sheoran S, Sahai R, Indora J and Biswal U. Spontaneous Perforation of Gallbladder: Case Report. Gastroenterology Research. 2016; 9: 61–63.

7. Yokoe, Masamichi et al. "Tokyo Guidelines 2018: Diagnostic Criteria and Severity Grading of Acute Cholecystitis (with Videos)." Journal of Hepato-Biliary-Pancreatic Sciences. 2018; 25: 41–54.

8. Sheridan D, Qazi A, Lisa S and Vashisht R. Spontaneous Acalculous Gallbladder Perforation. BMJ Case Reports 2014: 1–2.

9. Kim H et al. A Case of Spontaneous Gallbladder Perforation. Korean Journal of Internal Medicine. 2004; 19: 128–31.

10. Sharma R, Goyal S, Mittal A and Singhal K. Spontaneous Perforation of Acalculous Gallblader: A Case Report. Indian Journal of Surgery. 2011; 73: 316–17.

Manuscript Information: Received: May 13, 2021; Accepted: August 17, 2021; Published: August 31, 2021

Authors Information: Mendes João*; Couto José; Martins Aires; Monteiro Conceição; Midões Alberto
Department of General Surgery, Unidade Local de Saúde do Alto Minho (ULSAM), Portugal.

Citation: João M, José C, Aires M, Conceição M, Alberto M. Subclinical gallbladder perforation. Open J Clin Med Case Rep. 2021; 1783.

About the Journal: Open Journal of Clinical and Medical Case Reports is an international, open access, peer reviewed Journal focusing exclusively on case reports covering all areas of clinical & medical sciences.
Visit the journal website at www.jclinmedcasereports.com
For reprints and other information, contact info@jclinmedcasereports.com