Spontaneous Healthy Gallbladder Perforation, Case Report and Literature Review

Rotura espontánea de vesícula biliar sana, reporte de caso y revisión de literatura

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Summary
Spontaneous gallbladder perforation is a rare medical condition and it is primarily related to acalculous cholecystitis; however, it has a high mortality rate compared to deaths caused by calculous cholecystitis, which only represents 1%. Clinical and paraclinical data are not specific, so it is important to provide a timely diagnosis and immediate treatment to avoid complications and to improve patients' prognosis.

Keywords: Gallbladder, Acalculous Cholecystitis, Cholecystectomy

Resumen
La perforación espontánea de la vesícula biliar es una entidad poco común y está relacionada principalmente con la colecistitis alitiáscica; sin embargo, tiene una alta tasa de mortalidad en comparación con las muertes causadas por colecistitis litiásica, que solo representan 1%. Los datos clínicos y paraclínicos no son específicos, por lo que el diagnóstico oportuno es importante para proporcionar un tratamiento inmediato y evitar complicaciones con el propósito de mejorar el pronóstico del paciente.

Palabras clave: vesícula biliar, colecistitis alitiáscica, colecistectomía

Introduction
Spontaneous gallbladder perforation is a rare illness (3 to 10%), usually found as a complication of acute acalculous cholecystitis, yet has a high mortality rate of up to 42%.1-5 It is considered a diagnostic challenge because the pathogenesis is not well known, clinical evolution may be confused with uncomplicated acute cholecystitis data, there are no pathognomonic clinical data, blood tests show no difference or any specific data leading to an accurate diagnosis and even the information obtained through different imaging techniques do not always provides concrete data, so the diagnosis is accurate only until surgery. Early diagnosis and management are crucial to patients' prognosis, which can become fatal if not detected in a timely manner.

Below is the case of a patient who went to the emergency room with a clinical picture of pain and with image studies compatible with a liver hematoma, however, because of its unfavorable evolution laparoscopy was performed that concluded in cholecystectomy by seemingly healthy gallbladder perforation.

Case Report
Female patient of 46 years, with no apparent important medical history, fasting of only eight hours, with clinical picture of sudden pain, located in the right hypochondrium of moderate or mild intensity to 3 of 10, on the visual analog scale (vas), with ten days of evolution, initiated after a pool dive, accompanied by nausea without vomiting. She was examined in a private medical unit, where image studies were performed, an abdominal cat that reported subcapsular hematoma. She was managed symptomatically and released due to improvement after 48 hours, however pain persisted so she returned to the hospital five days after her hospital discharge. At her admission, new studies were conducted including a new cat that reported subcapsular hematoma (figure 1), her gallbladder was reported normal (figure 2). Blood tests showed high leukocytosis and bilirubin; and after three days there was no improvement (table 1), so it was decided to perform diagnostic-therapeutic laparoscopy finding a non-inflamed gallbladder, with rupture in the bottom, without gallstones ( lithos), the encapsulated bile (biloma) of approximately 100cc (figure 3) wrapped by omentum, for which cholecystectomy and bile drainage were performed without eventualities; the evolution was satisfactory and the patient was discharged after 48 hours of the surgery.

Table 1

<table>
<thead>
<tr>
<th>Day</th>
<th>Clinical Evolution</th>
<th>Blood Tests Evolution</th>
<th>Imaging Evolution</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Pain. Mild in the right hypochondrium</td>
<td>Complete Blood Count (CBC): leukocytes 10500 neutrophils 75% hematoglobin 12 Liver function Total Bilirubin 2.1 Direct bilirubin 1.7 Indirect bilirubin 0.4</td>
<td>Abdominal cat: normal gallbladder Subcapsular hematoma of 3x5 cm right liver lobe</td>
</tr>
<tr>
<td>2</td>
<td>Pain. Mild in the right hypochondrium</td>
<td>Complete Blood Count (CBC): leukocytes 10500 neutrophils 75% hematoglobin 11 Liver function Total bilirubin 2.1 Direct bilirubin 1.7 Indirect bilirubin 0.4</td>
<td>Abdominal cat: normal gallbladder Subcapsular hematoma of 4x5 cm right liver lobe</td>
</tr>
<tr>
<td>3</td>
<td>Pain. Mild in the right hypochondrium</td>
<td>Complete Blood Count (CBC): leukocytes 11000 neutrophils 80% hematoglobin 11 Total bilirubin 1.5 Direct bilirubin 0.5 Indirect bilirubin 1.0</td>
<td>Abdominal cat: normal gallbladder Subcapsular hematoma of 4x5 cm right liver lobe</td>
</tr>
</tbody>
</table>

Discussion
The first gallbladder perforation report was described by Duncan in 1840.1,2 In 1890 Courvoisier described 499 cases of gallbladder perforation, of which 169 showed as cholecystocutaneous fistula.3 It is most common in men and the average age is 60 years.4,6,7 While there is no specific cause, there are a number of factors that predispose to this pathology, including systemic diseases such as type 2 diabetes mellitus (dm2), atherosclerotic heart disease, systemic high blood pressure (since they are factors for the development of peripheral angiopathy), infections (enteric fever), corticosteroid therapy; in younger patients, immune commitment may be associated to hemodynamic instability and critical illnesses.6,7 Based on its origin it can be classified in: spontaneous, iatrogenic and...
Type I: Acute perforation of the free gallbladder (no adhesions) with generalized biliary peritonitis

Type II: Subacute perforation with pericholecystic abscess with localized peritonitis

Type III: Chronic perforation with cholecystoenteric fistula formation

The most common anatomical site is the bottom (40.5%), \(^1\), \(^2\), \(^5\) as it is the least vascularized area, \(^4\), \(^5\) combined with vascular and ischemic changes, which are crucial for the perforation pathogenesis; it can also occur in the neck in relation to impacted lithos or infection.\(^5\)

The explanation that underpin gallbladder perforation in acalculous cholecystitis is the presence of peripheral angiopathy that triggers disorders such as coronary atherosclerosis and capillary injury, this decreases resistance in the gallbladder and its terminal segment. The terminal blood vessels of the hepatic artery that are responsible for the irrigation of the gallbladder are likely to develop embolism and ischemia, causing necrosis and perforation of the vesicular wall.\(^7\)

The most common mechanism involves obstruction of the cystic duct, by increasing the intraluminal pressure of the bile duct, which causes gallbladder distension, vascular involvement, ischemia, necrosis and perforation. Bloat-free gallbladder perforation is related to infection of the Rokitansky-Aschoff’s sinuses with subsequent necrosis and rupture.\(^6\)
Immersion in water results in a redistribution of blood flow caused by external hydrostatic pressure from the water, causing it to increase at the central level and decrease in other peripheral areas, such as the skin and intestines, including the gallbladder, which generates ischemia.10

Initially, it may occur with data resembling acute cholecystitis; there are no classic or pathological symptoms of gallbladder perforation, so diagnosis is a challenge.2,4,6,7

Perforation usually develops during cholecystitis (one to two days, even weeks later). It has been noted that perforation should be suspected in patients with acute cholecystitis that deteriorate rapidly; once vesicular perforation has been instituted there may be peritoneal irritation data.1,4,5 It is suggested that when the perforation occurs at the bottom there is less chance of it being covered by omentum, so bile drains into the peritoneal cavity, on the other hand, when drilling occurs at a site other than the bottom, it is easily sealed by omentum or intestine, so it will remain limited to the right upper quadrant with formation of pericholecystic fluid.1,4

Since it is an unusual medical condition and has clinical similarities with uncomplicated acute cholecystitis, diagnosis is a challenge; in addition to the clinical course, medical history and physical examination, it is important to highlight the need of using imaging techniques for clarification of diagnosis.

Ultrasound is the initial examination, however, sometimes it does not show perforation due to increased intestinal gas; it shows data such as edema and vesicular wall thickening.2,4,7

A cat has increased sensitivity and shows a clearer gallbladder with thick walls and collections.2-4,9

The unique radiological sign of the vesicular rupture is called the “hole sign”, in which the wall defect can be observed, however, it is not always evident.1,2,5

Due to clinical and even imaging similarities with cholecystitis, the diagnosis is not accurate until the patient undergoes a surgery.

Management will always be surgical through cholecystectomy, however, it will depend on the general condition of the patient; when it comes to a critical patient minimally invasive procedures are chosen, such as percutaneous collection drainage, antibiotic handling and a plan for definitive surgery, once the patient is stable. In clinically stable patients laparoscopic cholecystectomy is used, but in case of difficulty it can become an open surgery.2 The main complication is abdominal sepsis that subsequently ends in septic shock, so it is important to intentionally seeking systemic inflammatory response data.6

Conclusion
A perforated acalculous cholecystitis was found so it was decided to do a laparoscopic examination due to the clinical evolution of the patient. It is likely that vesicular perforation may have been related to the pool submersion by the redistribution of blood flow.

Perforated acalculous cholecystitis is a difficult medical condition to diagnose in patients without prior pathology; scenarios such as the case described should be taken into account to avoid multiple life-threatening complications.

References