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Gallstone ileus as a complication of cholelithiasis: review of a clinical case

Íleo biliar como complicación de colelitiasis: revisión de un caso clínico

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Abstract:

Gallstone ileus is an obstructive pathology at the intestinal level, secondary to a gallstone in the intestinal lumen as a complication of cholelithiasis. It occurs when a gallbladder stone migrates through a bilioenteric fistula, with the ileum being the most common site of stone impaction. Gallstone ileus has a clinically insidious presentation, in addition to non-specific data through complementary examinations, which makes timely diagnosis difficult. Imaging studies play a relevant role in identifying this pathology, with Rigler's triad as a pathognomonic radiological finding of gallstone ileus. Hereby, we present a clinical case of this pathology in a 50-year-old male patient, along with CT scans showing the Rigler's triad, laboratory exams, and complete clinical assessment, to show the presentation of such an uncommon pathology.

Keywords:

Gallstone ileus, gallstone, fistula, Rigler's triad.

Resumen:

El íleo biliar es una patología obstructiva a nivel intestinal, secundario a la presencia de un cálculo biliar en la luz intestinal como complicación de una colelitiasis. Se produce cuando un cálculo en la vesícula biliar migra a través de una fístula bilioentérica, siendo el íleon el lugar más frecuente como sitio de impactación del cálculo. El íleo biliar tiene una presentación clínicamente insidiosa, además de datos inespecíficos a través de exámenes complementarios, lo que hace difícil el diagnóstico oportuno. Los estudios de imagen juegan un papel importante en la identificación de esta patología, siendo la tríada de Rigler un dato radiológico patognomónico de íleo biliar. En el presente trabajo mostramos un caso de esta patología en un paciente masculino de 50 años de edad, junto con imágenes de tomografía computarizada que ilustra el hallazgo de la tríada de Rigler, así como exámenes de laboratorio y características clínicas, como un medio para ilustrar la presentación de una patología tan poco común.

***Palabras Clave**:*

Íleo biliar, calculo biliar, fistula, tríada de Rigler.

INTRODUCTION

Biliary ileus (IB) is one of the less common complications of cholelithiasis (CL), described as a mechanical intestinal obstruction secondary to the presence of a gallstone (GS) in the intestinal lumen.[1,2] It is caused by the passage of a GS from the biliary ducts into the intestinal lumen through a fistula. The most frequent type of fistula is located between the gallbladder (VB) and the duodenum.[3-5.]

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These fistulas form after chronic erosion by a stone or recurrent/chronic inflammation of the gallbladder wall, that is, lithiasic cholecystitis.[6,7] Apparently, GSs larger than 2.5 cm are responsible for vesicular and intestinal impaction.[5] An impacted GS that is in close contact with inflamed mucosa first develops ischemia and then necrosis, as inflammation and pressure from the GS create erosion of the VB wall, resulting in the formation of a fistula between the VB and the adjacent segment of the gastrointestinal tract, allowing the passage of the gallstone.[8,9]

The most common sites of GS impaction are the ileum (50.0%–60.5%), jejunum (16.1%–26.9%), duodenum (3.5%–14.6%), and colon (3.0%–4.1%).[10,11] This applies to the formation of other biliary fistulas, where the gallstone can also migrate through gallbladder-jejunal fistula, gallbladder-colonic fistula, gallbladder-duodenal-colon, gallbladder-gastric, common bile duct-duodenal, or duodenum-left hepatic bile duct.[5,12]

Other mechanisms of obstruction include the passage of small stones that migrate through the ampulla of Vater, followed by *in situ* growth, small gallstones that become impacted in a narrowed intestine (for example, in Crohn's disease), or inadvertent migration of the gallstone during manipulation of the gallbladder or ducts while performing a cholecystectomy.[9,13]

BI is a surgical emergency that accounts for approximately 1% to 4% of cases of mechanical intestinal obstruction. It is more common in patients over 65 years old, being more prevalent in women, where it can reach up to 25% of small bowel obstructions.[3,4]

The mortality rate associated with BI ranges from 12% to 27%, and the morbidity rate reaches 50%, due to late diagnosis, concomitant conditions, and advanced age. BI occurs when a gallstone passes into the gastrointestinal tract through a biliary-enteric fistula.[5]

The most common symptoms include vomiting (87%), abdominal pain (71%), hematemesis (15%), weight loss (14%), and anorexia (13%), plus the digital signs of dehydration and abdominal distension.[13]

Due to the insidious and nonspecific presentation of signs and symptoms in patients with BI, diagnosis is often delayed. Complementary laboratory tests can assist but are not specific, typically showing leukocytosis due to obstruction.[14] Imaging studies play a crucial role in diagnosis; however, X-rays have low sensitivity and specificity, making computed tomography (CT) the preferred method.[15]

Rigler's triad, which consists of small bowel obstruction, pneumobilia, and an ectopic gallstone, is practically pathognomonic for gallstone ileus. However, it is present in conventional X-rays in only about one-third of cases of this pathology. This triad of findings is more evident in computed tomography scans.[6,13]

MATERIALS & METHODS

The analysis involved reviewing the case's physical and electronic records, collecting images from the performed computed tomography scan, laboratory data, and information derived from the clinical evaluation. The patient signed an informed consent form after the ethics committee for research at the Hospital Español de Veracruz approved the study (EXT99.SEBP2023). The study followed the principles of the Declaration of Helsinki. All the images from CT scans were adequately anonymized to respect the patient's confidentiality, and throughout the study, no data were used that could lead to his identification.

CASE REPORT

The patient is a 50-year-old male with significant medical history that includes bilateral venous insufficiency for 32 years, treated with oral anticoagulants; left knee tendinitis, treated with cortisone and diclofenac; diabetes mellitus; and a history of severe sepsis, secondary to Ludwig's angina, which required washouts and drainage of a submandibular abscess.

His surgical history includes placement of a mesh in the inferior vena cava 30 years ago and two previous saphenectomies, also 30 years ago, as well as left knee surgery due to a rupture six years ago that required multiple surgical washouts. The onset of his current condition began after consuming a copious meal, presenting with diffuse colicky abdominal pain, nausea, and multiple bilious vomiting episodes, along with abdominal distension.

Subsequently, the pain migrated to the right hypochondrium and left flank, becoming more intense. Treatment with butylscopolamine and intramuscular metoclopramide provided no relief. An abdominal ultrasound was performed, which indicated findings consistent with gallstone ileus (GI). The pain intensified further, accompanied by constipation, prompting evaluations by internal medicine and general surgery.

Upon examination, the patient appeared calm but in pain, conscious, normocephalic, with good skin coloration but dehydrated mucous membranes. Pulmonary fields showed good air entry and exit; heart sounds were rhythmic without additional phenomena. The abdomen was soft, depressible, and tender upon palpation in the right hypochondrium and left flank, positive for Murphy's sign. There was generalized tympanism and significantly decreased peristalsis in both frequency and intensity. Extremities showed immediate capillary refill without edema but displayed color changes.

Laboratory tests (Table 1) revealed normal complete blood count, but elevated C-reactive protein, indicating an acute infectious process. Initial blood chemistry showed glucose at the upper limits of normal; the renal function slightly altered with BUN at 34.9 mg/dL, urea at 74.69 mg/dL, and creatinine at 1.48 mg/dL. The lipid profile was regular, while liver function tests showed elevated bilirubin levels: total bilirubin at 4.1 mg/dL, due to indirect bilirubin at 2.8 mg/dL and direct bilirubin at 1.3 mg/dL. Transaminases and alkaline phosphatase were within normal ranges, indicating isolated hyperbilirubinemia without specific hepatic alteration patterns. The albumin/globulin relation was preserved, and there was no evidence of hydroelectrolytic imbalance.

Table 1. *Complementary exams.*

	Day 1	Day 2	Day 10
Hemoglobin (gr/dL)	17.2	12.8	12.4
Hematocrit (%)	53.6	38.4	32.8
Platelets (/mm^3)	203,000	90,000	158,000
Leucocytes (u/L)	7330	5900	7300
Total neutrophils (%)	68	74	80
Segmented neutrophils (%)	68	74	80
CRP (mg/L)	25.2	101	145.9
Glucose (mg/dl)	120	71.9	83.15
BUN (mg/dl)	34.9	12.5	10.11
Urea (mg/dl)	74.69	26.74	21.64
Creatinine (mg/dl)	1.48	1.1	1.0
Uric acid (mg/dl)	1.48	4.7	3.9
Cholesterol (mg/dl)	157	77.03	73.69
Triglycerides (mg/dl)	79	82.76	88.14
Total bilirubin (mg/dl)	4.1	0.7	0.6
Direct bilirubin (mg/dl)	1.3	0.6	0.4
Indirect bilirubin (mg/dl)	2.8	0.1	0.2
Amylase (U/L)	94	36.3	32.1
Lipase (U/L)	26	39.5	30.6
ALP (U/L)	121	111.1	119.9
GOT (U/L)	27	26.2	16.2
GPT (U/L)	21	20.2	11.1
LDH (U/L)	252	183.5	186.9
GGT (U/L)	29	56.9	55.5
Total protein (g/dl)	9.7	5.8	5.5
Albumin (g/dl)	5.4	3.25	2.93
Globulin (g/dl)	4.3	2.55	2.57
Albumin/globulin relation	1.26	1.27	1.14
Na (mEq/L)	141	141	135.9
K (mEq/L)	3.58	3.78	3.37
Cl (mEq/L)	108.1	115	107.9
Ca (mEq/L)	10.8	7.97	7.58
P (mEq/L)	2.7	2.65	2.68
Mg (mEq/L)	2.0	1.68	1.43

CRP, c-reactive protein; BUN, blood-urea nitrogen; ALP, alkaline phosphatase; GOT, glutamic-oxaloacetic transaminase; GPT, glutamic-pyruvic transaminase; LDH, lactic-dehydrogenase; GGT, gamma-glutamil transaminase.

The abdominal ultrasound findings indicate hepatosplenomegaly of undetermined origin, a collapsed gallbladder, likely intrahepatic versus hypoplastic, and non-obstructive right renal lithiasis, along with abundant gas in the colon. Due to these findings, hospitalization is recommended for specialist evaluation. Additionally, the patient will begin parenteral rehydration with crystalloid fluids, empirical antibiotic coverage, and treatment with an antispasmodic and antiemetic.

On the third day after admission, a plain and contrast-enhanced abdominal CT scan was requested, revealing findings consistent with a cholecystoduodenal fistula (Fig. 1), as well as signs of intestinal obstruction (Fig. 2) and nonspecific findings such as intrahepatic pneumobilia (Fig. 3). However, no alterations justified urgent surgery; but, due to the insidious nature of the condition. The possibility of surgery cannot be ruled out. . Therefore, physicians requested a follow-up consultation with general surgery.

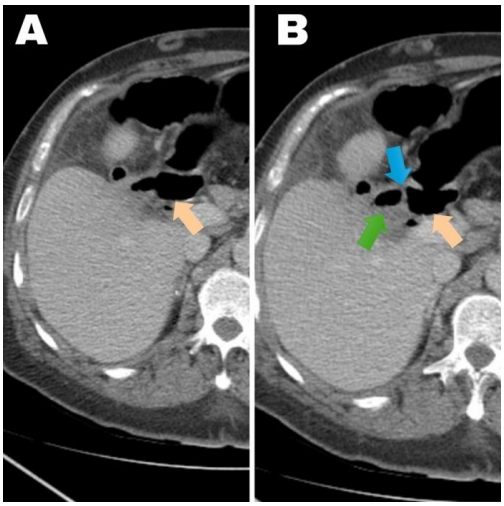

Figure 1. Abdominal CT, axial cut. Rigler's triad: A) Duodenal bulb (brown arrow) in vesicular topography. B) Biliary duct (green arrow) not distended by gas; it has apparent communication (blue arrow) with the duodenal bulb (brown arrow).

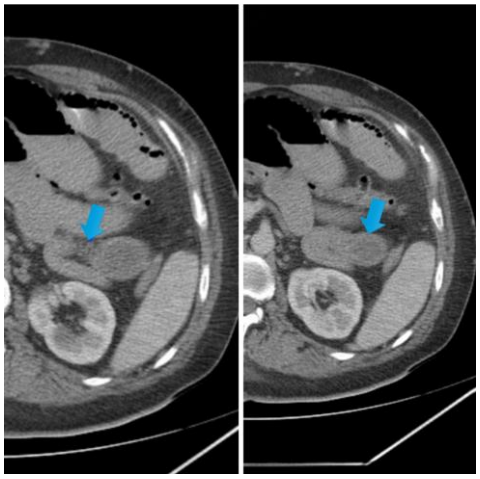

Figure 2. Abdominal CT scan in venous series, axial cut: Loops of the small intestine with signs of intestinal obstruction, with a transition zone in the jejunal loops (blue arrow) and an oval hypodense image suggestive of a non-radiopaque gallstone.

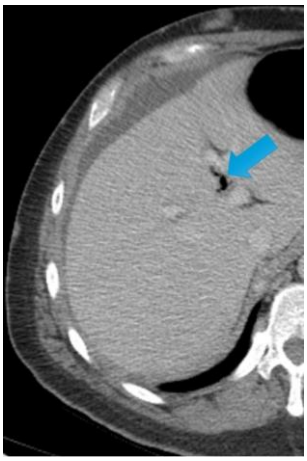

Figure 3. Abdominal CT on venous series, axial cut: intrahepatic pneumobilia (blue arrow).

The management plan continued with fasting, general care, antibiotic therapy, antiemetics, analgesics, a gastric regulator, a proton pump inhibitor, anticoagulants, and laxatives.

On day 4 after hospital admission, the evaluation by general surgery revealed alterations during the examination, such as a globular abdomen due to gas and retained intestinal contents, absent peristalsis, and isolated decantation sounds in the lower quadrants. The CT scan findings also indicated widespread diverticular disease, leading to the recommendation of exploratory laparotomy, which the patient and family accepted. During the intraoperative phase, the following findings were noted: the site of obstruction was 30 cm from the ileocecal valve, corresponding to a stone from the biliary tree impacted at this distance. An enterotomy was performed, extracting a stone approximately 3x2 cm in size.

Twenty-four hours post-surgery, the patient experienced a vomiting episode of 450 ml with an intestinal appearance, despite still lacking peristalsis. This situation was secondary to the surgical event; peristalsis was expected to begin 24 to 36 hours after surgery. Therefore, ambulation was encouraged to aid recovery. By day 7 after hospital admission, the patient started to show mild peristalsis without other significant changes in clinical status or laboratory results recorded in Table 1.

Pharmacological management continued as mentioned, along with electrolyte replenishment, the same management employed for 3 days; by day 10 post-hospitalization, the patient presented semi-liquid evacuations and gas discharge for the first time, leading to the initiation of a well-tolerated soft diet, resulting in discharge from the general surgery service. The next day, as the patient showed improvement in the clinical condition and no postoperative complications or issues with the wound itself, the patient was discharged home with instructions and outpatient pharmacological management.

DISCUSSION

GI is an uncommon cause of intestinal obstruction, primarily associated with advanced age and comorbidities. It presents as an insidious condition that is often difficult to diagnose due to nonspecific symptoms. Typically, it arises as a complication of CL, where gallstones migrate from the gallbladder into the intestinal lumen through a bilioenteric fistula, leading to intestinal obstruction.[5]

Laboratory tests and simple radiographs generally exhibit low sensitivity and specificity for diagnosing gallstone ileus. The most reliable diagnostic tool is CT, which can reveal significant imaging findings, including the Rigler's triad: small bowel obstruction, pneumobilia, and the presence of a gallstone.[8]

The treatment for gallstone ileus is surgical, usually involving an exploratory laparotomy. However, laparoscopic techniques have shown successful outcomes and offer the advantage of earlier recovery.[6]

In the present case report, we show the importance of CT scans in diagnosing the pathology through Rigler's triad, which is a patognomonic sign of BI.[16] In such circumstances, current recommendations in diagnosing acute abdomen-type pathologies include using CT scan when available, which, as we show here, was of enhanced utility in the diagnostic procedure for this patient's presentation.[17]

CONCLUSION

GI is a cause of intestinal obstruction resulting from an impacted gallstone in the intestinal lumen. It typically occurs in elderly patients, associated with a high frequency of comorbidities and significant surgical risk. Due to its challenging diagnosis, it is essential to understand the pathology to maintain a high index of suspicion and achieve a timely diagnosis, which can improve postoperative prognosis. The treatment is surgical; however, it is at the discretion and judgment of the surgeon, who must tailor it according to the patient's specific conditions.

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