

Acalculous Cholecystitis Secondary to Giant Hepatic Abscess. Case Report and Literature Review

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Abstract

Alithiasic cholecystitis (AC) occurs in 5% of cases of acute cholecystitis, typically in severe patients, treatment of liver abscesses according to size is usually antibiotic therapy and radiological drainage, in refractory cases it may be considered surgical.

Clinical case: A 75-year-old male patient with an 11-day history of nonspecific abdominal pain, evidenced by computed axial tomography anhepatic lesion of 134 mm diameter, was approached laparoscopically in which evidence of cholecystitis and liver abscess was evident and resolved. Results: The patient probably presented a simple hepatic cyst, a lesion from 10 previous years, that was infected with *E. coli*, with subsequent development of AC due to the infection. The resolution of the primary pathology's complications by laparoscopic was feasible.

Conclusions: The treatment of the primary cause and of the AC is indispensable for the clinical improvement of the patient, the laparoscopic treatment is considered as a safe option to approach the two entities with less morbidity than open surgery.

Keywords: Acute Alithiasic cholecystitis, liver abscess, geriatric

1. CASE REPORT

A 75-year-old male patient suffering from abdominal pain 11 days prior to admission, presenting with diffuse abdominal pain, intensity 6/10, a visual analogue scale, exacerbated by the intake of any food, early postprandial fullness, abdominal bloating, and intermittent fever without hourly predominance.

Personal history of arterial hypertension managed with losartan, acute myocardial infarction 9 years ago with reperfusion treatment by balloon catheterization with stenting, chronic smoker, when myocardial infarction protocol, a simple hepatic cyst of 2 cm was identified. No prior abdominal surgery.

On physical examination, mild dehydration, lung fields suggestive of right pulmonary

effusion, cardiac intensity decreased, abdomen globose at the expense of painful abdominal distension pain at deep palpation on epigastrium and right hypochondrium, positive Murphy and Boas sign.

A tomography was performed in which a giant liver lesion was observed in segments IVA and VIII estimated at 134 mm in diameter (image 1) with a density compatible with a simple cyst. Laboratories: hemoglobin 10.8 g/dL, platelets 202×10^3 U/L, leukocytes 10.2×10^3 U/L, neutrophils 79%. Prothrombin time 17.4 secs, INR 1.55. Activated thromboplastin partial time 46 secs. creatinine 1.55 mg/dL, total protein 5.8 g/dL, albumin 2.3 g/dL, globulins 3.4 g/dL, total bilirubin 0.63 mg/dL, CA 19-9 antigen 28.1 U/mL.



Image1. Giant liver lesion observed in segments IVA and VIII estimated at 134 mm in diameter by computed axial tomography

It is approached laparoscopically identifying fibrinopurulent peri vesicular and vesicular plastron (image 2), a cholecystectomy is performed, when it is not possible to perform a Strasberg critical vision of safety due to short cystic, an antrograde gallbladder dissection with placement of two Vicryl endoloops on the cystis performed, changes in liver parenchyma are observed (image 3), an incision is made with ultrasonic energy and 950 cc of purulent material are drained. After surgery due to risk

factors, it is managed in intensive care, it presents a residual collection of 20% (image 4) with respect to the initial image and it goes on laparoscopic unroofing and drainage. The culture was positive for Gram negative bacilli E. coli, ESBL negative, sensitive to ertapenem. After this, the patient shows significant improvement and is discharged after 10-day hospitalization. Follow-up is performed at 6 months with adequate evolution.

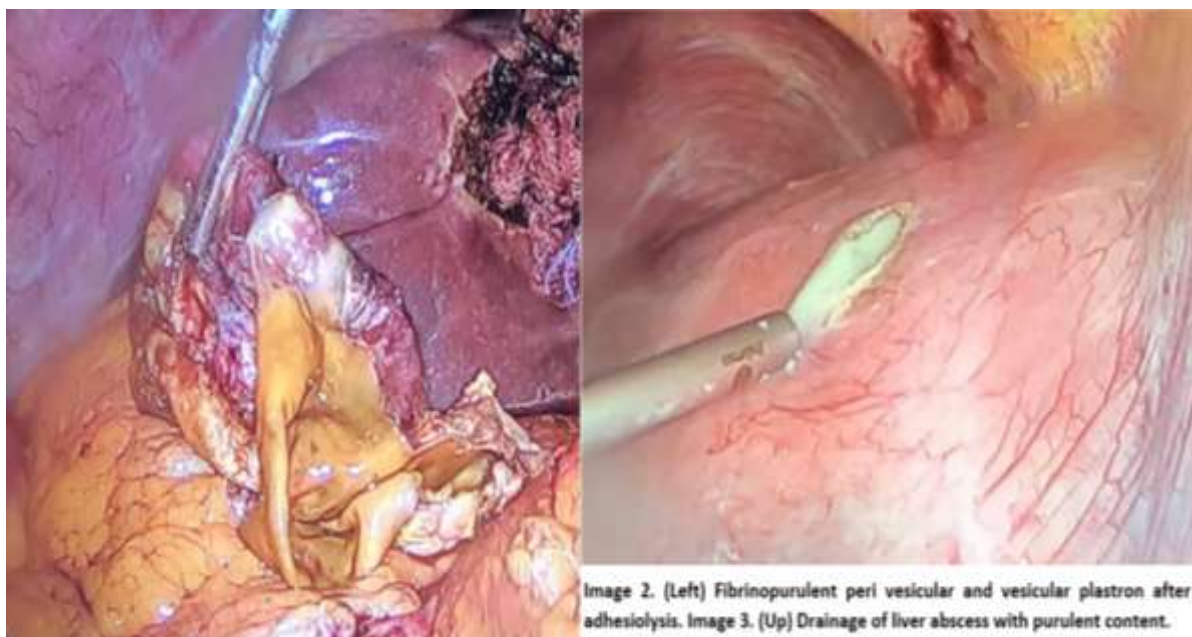


Image2. (Left) Fibrinopurulent peri vesicular plastron after adhesiolysis, **Image3.** (Up) Drainage of liver abscess with purulent content



Image4. Control tomographic image of liver abscess before second intervention

2. DISCUSSION

Acalculous cholecystitis is the inflammation of the gallbladder without evidence of gallstones; it accounts for the 10% of all cases of acute cholecystitis; its presentation is insidious and may induce high morbi-mortality.[1] Typically seen in critically ill patients (sepsis, recent trauma, burn injury, surgery, or hemodynamic instability) and on specific infectious conditions.[1-4] Results from gallbladder stasis and ischemia, which then cause a local inflammatory response in the gallbladder wall[5] Characterized by unexplained fever, leukocytosis, hyperamylasemia, high aminotransferases, and may or may not right upper quadrant tenderness with a palpable mass.[1, 5, 6] Diagnosis of acalculous cholecystitis is based upon a constellation of symptoms and signs in the setting of supportive imaging findings (abdominal ultrasound or contrast abdominal CT), and the exclusion of differential diagnoses. Ultrasound imaging findings include 3.5- to 4-mm (or more) thick wall; sonographic Murphy's sign, pericholecystic fluid/subserosal edema, intramural gas, echogenic bile, hydrops (dimensions greater than 8x5cm. CT findings include gallbladder wall thickening (>3 mm), subserosal edema, pericholecystic fluid, intramural gas, gallbladder distention (>5 cm). [6-8] Management of acalculous cholecystitis includes supportive care with intravenous fluids, pain control, initiation of antibiotics, and definitive therapy with either cholecystectomy or gallbladder drainage. Patients without indications for emergency cholecystectomy

(gallbladder necrosis, emphysematous cholecystitis and/or gallbladder perforation), who are critically ill or in poor health, or unfit for general anesthesia should be treated with gallbladder drainage. [6, 9-11]

Liver abscess is a differential diagnosis of acalculous cholecystitis, there is low evidence of coexistent conditions, the most of them presented as a complication of acalculous cholecystitis. [12-15] When pyogenic liver abscesses develop, it is most commonly following peritonitis due to leakage of intraabdominal bowel contents with subsequent spread to the liver via the portal circulation or in the setting of biliary infection via direct spread. Enteric gram-negative bacilli, particularly *Escherichia coli* and *K. pneumoniae*, are generally the most identified pathogens. Clinical manifestations are fever and abdominal pain. [16, 17] Other common symptoms include nausea, vomiting, anorexia, weight loss, and malaise. Laboratory abnormalities may include elevated bilirubin and/or liver enzymes. Evaluation of suspected pyogenic liver abscess includes imaging (usually computed tomography or ultrasound), blood cultures, followed by aspiration and culture of the abscess material [16, 18]. The diagnosis is confirmed in a patient with a liver lesion on imaging that is purulent on aspiration and/or has bacteria identified on Gram stain or culture of the aspirate material or blood.[19,20, 21] Drainage of liver abscess, either surgically (either open or laparoscopic), percutaneously, or via endoscopic retrograde cholangiopancreatography, is standard.[21] A single unilocular abscess,

percutaneous drainage if ≤ 5 cm in diameter; if > 5 cm in diameter, percutaneous catheter drainage rather than needle aspiration.[22] Surgical drainage is appropriate when there is an underlying disease that requires primary surgical management, when there is an inadequate response to catheter drainage, or if the abscess has viscous contents that preclude successful percutaneous drainage.[18, 23]

3. CONCLUSION

The patient probably initially presented with a simple hepatic cyst, a lesion from the previous 10 years, which was infected with E. coli, with subsequent development of AC due to the infection. The treatment allowed the resolution of the primary pathology and its complications.

The treatment of the primary cause and of the AC is indispensable for the clinical improvement of the patient, the laparoscopic treatment is considered as a safe option to approach the two entities with less morbidity than open surgery

FUNDING AND CONFLICTS OF INTEREST

None

ETHICAL APPROVAL

There was no ethics approval required for this case report

CONSENT

Written informed consent was obtained from the patient for publication of this case report and accompanying images

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