Ischemic acute cholecystitis: a case report

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Abstract

Acute cholecystitis is the sudden inflammation of the gallbladder. Symptoms include right upper abdominal pain, nausea, vomiting, and occasionally fever. In most cases, it is caused by the blockage of the cystic duct by a gallstone which causes bile stasis in the gallbladder and results in secondary bacterial infection.

It occurs without evidence of gallstones or cystic duct obstruction in 2 to 15% of the cases [1]. This condition is called acalculous acute cholecystitis (AAC). The etiology of AAC is multifactorial and likely results from bile stasis or ischemia (or both) [1-3]. It is mostly diagnosed in critically ill patients, particularly those with cardiovascular diseases.

In this article, we report a case of ischemic acute cholecystitis.

KEYWORDS: Cholecystitis, Acalculous, Gallbladder Wall, Ischemia, Bile Stasis

INTRODUCTION

Acute cholecystitis (AC) is the sudden inflammation of the gallbladder (GB). It is a prevalent surgical condition and a frequent cause of abdominal pain.

In most cases, it is due to the obstruction of the cystic duct by a gallstone (GS), which exists for a long period before this condition occurs in asymptomatic patients or those having neglected biliary colic.

Symptoms include right upper abdominal pain in most cases. Fever, nausea, and vomiting are less frequent. Paralytic ileus was also described in some patients. Physical examination generally finds a Murphy's sign.

Abdominal ultrasound can confirm the diagnosis. However normal ultrasound findings don't exclude the diagnosis of AC. These symptoms are found in patients with no radiological or surgical evidence of gallstones or cystic duct obstruction in approximately 10% of the cases. This condition is called acalculous acute cholecystitis (AAC).

The etiology of AAC is multifactorial. Its pathophysiology includes bile stasis, ischemia, or both [1-3].

We report a case of an ischemic AAC diagnosed in our emergency department.

Case report

A 50-year-old male patient with a medical history of diabetes, hypertension, ischemic stroke, and kidney failure presented to our emergency department

suffering from right upper abdominal pain, asthenia, and vomiting starting 4 days before admission, without intestinal abdominal disorders. No chest pain was reported.

On initial physical examination, he was conscious, with a normal respiratory rate and a pulse oximetry of 100 %. He had a heart rate of 135 beats per minute and a normal blood pressure of 130/80 mmHg. No signs of circulatory impairment or right heart failure were found. The temperature was 36.9^{c} and the blood sugar level was > 5g/l. The abdominal examination found tenderness with guarding in the epigastric, the right hypochondriac, the right lumbar, and the right iliac region. An electrocardiogram showed a sinus tachycardia.

Blood panel showed: white blood cells elevation but normal CRP level (WBC=14610 el/mm3; CRP = 7.4 ng/l), normal liver enzymes (AST=26 u/l; ALT =40 u/l), elevated creatinine and urea levels (715 µmol /l; 15.6 mmol/l), normal lipase (33u/l) and normal ultra-sensible troponin level (12.2 ng/l). The lactate was 5.8 mmol/l.

The abdominal ultrasound showed a distended gallbladder with normal wall thickness associated with hyperechogenic, and heterogeneous content without GS.

DISCUSSION

AAC remains challenging because of its complex multifactorial pathogenesis and nonspecific presentation.

AAC risk factors include severe trauma requiring blood transfusions, heavy surgery, shock of any kind, large burn injuries (with an incidence between 0.4 and 3.5%), and critically ill patients requiring ICU care and sepsis [1, 4, 5].

Less frequently described risk factors are endoscopic retrograde cholangiopancreatography, cardiovascular risk factors (diabetes mellitus, hypertension, atherosclerotic, obesity), end-stage renal failure, vasculitis (Churg–Strauss, giant cell arteritis, Henoch–Schoenlein (purpura, polyarteritis nodosa, lupus, Takayasu's syndrome), Immunodeficiency conditions (AIDS, Transplant) [1,4,6].

Two other rare risk factors are worth mentioning:

-ACC after transarterial chemoembolization of hepatocellular cancer, which is reported with an incidence between 0.3% and 10% [7].

-ACC is associated with an aortic dissection which is rarely described in the literature. Only 5 cases of AAC associated with an aortic dissection have been reported previously [8-11].

Our patient had an AAC which is related to the superior mesenteric artery stenosis. To the best of our knowledge, no similar cases have been described before in the literature.

AAC pathophysiology is complex, but studies found that it involves two major phenomena: bile stasis and ischemia [1, 4].

Bile stasis can be caused by fasting, obstruction, postsurgical/procedural irritation, ileus (total parenteral nutrition), or opioid analgesia (spasm of the sphincter of Oddi causing elevated bile duct pressure). This stasis modifies the chemical composition of bile, resulting in gallbladder mucosal lesions [1, 4].

The decrease of the gallbladder perfusion pressure, resulting in ischemia, may be the result of many of the risk factors stated above and it results in heavy injuries directly to all layers of its wall [1, 4].

Bacterial invasion of the ischemic tissue is a secondary phenomenon. The immunity response to this invasion may produce or contribute to GB tissue damage [4].

These pathophysiological mechanisms are well described in the literature, such as the histopathological data provided by Laurila et al [12], through comparing histologic findings of the gallbladders with and without ACC. Histological abnormalities reported in this study were : (1) an increased leukocyte margination (suggesting involvement of ischemia and reperfusion-mediated injury) ; (2) capillary thrombosis and increased focal lymphatic dilation (ischemia related) and (3) increased and deeper bile infiltration in the GB mucosa (suggesting that bile stasis and increased epithelial permeability exist, leading to epithelial damage).

As for the clinical presentation, there are no symptoms nor signs that are specific to AAC. Fever may be the only sign present in critically ill patients, unable to communicate their symptoms. For this kind of patient, AAC is a diagnosis among many others to suspect facing sepsis or a septic shock. It is sufficiently common and should be discussed in every critically ill patient with a clinical presentation of sepsis or icterus and no other obvious cause [1, 4].

The clinical presentation may also include right upper abdominal pain, fever, nausea, and vomiting.

Physical findings show fever, and tenderness with or without guarding in the epigastric region and/or the right hypochondriac. Clinically, AAC is indistinguishable from acute calculous cholecystitis [1]. Laboratory evaluation is also unreliable. Leukocytosis and CRP elevation are common but not specific. Lactate elevation is common.

Normal inflammatory markers with elevated lactates may be an early sign of an ischemic disorder but it is not specific.

The confirmation of the diagnosis rests on imaging. Abdominal ultrasound and computerized tomography (CT) scans are usually sufficient with an excellent reported sensibility and specificity (for the US: 92 % sensitivity and 96 % specificity; For CT scan: 100 % sensitivity and specificity) [13].

Other exams such as HIDA (hepatobiliary iminodiacetic acid scan) are used but they are expensive and not immediately available.

Diagnostic criteria were defined for both US and abdominal CT scans in many radiologic studies [1, 4, 13-16]:

Criteria for abdominal US: 2 major or 1 major and 2 minor

Major: 3.5- to 4-mm (or more) thick wall

Striated gallbladder.

Intramural gas Pericholecystic fluid

Sloughed mucosal membrane

Minor: Gallbladder distention (>5 cm in transverse diameter)

Echogenic bile (sludge)

Criteria for abdominal CT: 2 major or 1 major and 1 minor

Major: Gallbladder wall thickening >3 to 4 mm

Subserosal halo sign

Pericholecystic infiltration of fat

Pericholecystic fluid

Mucosal sloughing

Intramural gas

Minor: Gall bladder distention (>5 cm in transverse diameter)

High-attenuation bile (sludge)

AAC complications are numerous. The prevalence of gallbladder gangrene in AAC exceeds 50% [4]. Gallbladder perforation, abscess formation, generalized peritonitis, or severe sepsis with multiple organ dysfunction syndrome are commonly reported.

CONCLUSION

Although ACC is mostly described in critically ill patients, it remains a possible diagnosis in patients with multiple cardiovascular risk factors.

Clinical presentation is not specific, the laboratory assessment, is as well.

Abdominal US and abdominal CT scan criteria are the key to the diagnosis with excellent sensitivity and specificity.

An early accurate assessment is essential for the management to avoid poor outcomes.

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