Emphysematous cholecystitis in a non-diabetic patient

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A 47-year-old male was admitted to our emergency room with a 4-days history of acute abdominal pain, increased by inspiration. There was neither nausea nor vomiting. Physical examination revealed right upper quadrant and right flank tenderness. The patient had no diabetes mellitus. Laboratory tests showed a white-cell count at 15 x 10^9 cells/L (87% neutrophils), a CRP at 44.1 mg/dL, bilirubin at 1.9 mg/dL and γGT at 114 U/L.

The patient was referred to computed tomography (CT). The enhanced abdominal CT revealed numerous curvilinear gas bubbles in the gallbladder wall (Fig. A, B, C) associated with a discrete edema of the adjacent fat (Fig. B, arrows). Small gas densities in the periphery of the left lobe and the segment VIII of the liver were also detected and considered as hepatic portal venous gas (HPVG) (Fig. C, arrows). Hepatic steatosis was also identified. There were no gallstones.

Based on these imaging findings, the diagnosis of emphysematous cholecystitis was suggested and the patient underwent coelioscopic cholecystectomy 2 hours after CT. Microbiological analysis showed Clostridium Perfringens to be the causative organism. Histopathology revealed severe lesions of acute abscessed and widely gangrenous cholecystitis.

Ultrasound and plain X-ray were not performed.

The patient left the hospital 1 day after surgery. He subsequently developed a subhepatic abscess 8 days later. The abscess was drained percutaneously under echographic guidance and antibiotherapy was reintroduced for 1 month. Afterwards, follow-up was uneventful.

Comment

Emphysematous cholecystitis (EC) is a rare pathology. It imitates acute cholecystitis (AC) but has a different epidemiology: the male to female ratio is 4/1, most patients are over the age of 50 and gallstones are infrequent. Predisposing factors include diabetes, debilitated patients, abdominal trauma, abdominal surgery or any cholecystic ischemic factors. Gangrene and perforation of the gallbladder are five times higher compared to AC. The clinical presentation includes pyrexia, pain in the right abdominal region, signs of peritonitis in 50% and more rarely signs of septic shock. It is burdened with a higher mortality rate than AC. Biologically, there is a neutrophil-predominant hyperleucocytosis associated with an inflammatory syndrome, as in our case.

The diagnosis of EC is based on imaging. Although ultrasound may suggest the diagnosis, the gallbladder is often poorly visualized because of the gas. Plain X-ray of the abdomen can demonstrate a gaseous halo around the gallbladder, a gas-fluid level in the gallbladder, some gas in the pericholecystic tissues and sometimes even HPVG. The abdominal CT is the most sensible imaging modality for the diagnosis of EC and can show gas bubbles in the gallbladder wall, a gas-fluid level or intraluminal gas in the gallbladder, pericholecystic edema and HPVG.

The treatment of EC is an emergency. It is based on cholecystectomy and intravenous administration of antibiotics active on anaerobic and other digestive germs. EC should be kept in mind in front of a clinical presentation of AC, even without risk factors for EC. When ultrasound and plain X-ray are not contributory, a CT should be performed as soon as possible, since any delay in diagnosis may result in a more severe disease course. Our case is uncommon because the patient was relatively young, had neither diabetes nor cardiovascular pathology and presented HPVG, which is an infrequent finding at CT.

Reference