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Case Report

Hemorrhagic cholecystitis with auto-avulsion ☆☆☆★

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ABSTRACT

Acute cholecystitis is a common condition, with varying presentations and complications, and is frequently treated in the emergency department. This case report illustrates hemorrhagic cholecystitis (HC) as a rare complication of cholecystitis. A 74-year-old woman presented to our emergency department with intermittent abdominal discomfort and continued vomiting after any oral ingestion as well as watery diarrhea. Patient history included mitral valve replacement and a daily dose of warfarin. Emergent cross-sectional abdominopelvic computed tomography (CT) and magnetic resonance imaging (MRI) revealed a large mass that occupied most of the right abdominal cavity. An exploratory laparotomy revealed a necrotizing and hemorrhagic enlarged gallbladder with cholelithiasis. This enlargement appeared adherent and fistulizing into the cecum, without signs of bowel perforation. The postoperative period was uneventful, and the patient recovered well. Owing to its non-specific presentation, imaging studies are essential in establishing a diagnosis for HC. Although CT has not been advocated as a primary imaging examination for acute right upper quadrant pain, it is a valuable tool for assessing HC, along with MRI.

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Case report

Hemorrhagic cholecystitis (HC) is a rare complication of cholecystitis. The pathophysiology of gallbladder disease involves

gallbladder wall inflammation, which causes a cascade of events leading to gallbladder wall erosion, hemorrhage, subsequent mucosal ischemic changes, and infarction [1].

There are other non-inflammatory etiologies that contribute to this rare pathology as well, such as trauma, vascular abnormalities, blood anomalies, anticoagulant usage,

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* Patient Consent: Consent to publish this case series was not obtained, as our Office of This work does not convey any personal information that would lead to the identification of the patients.

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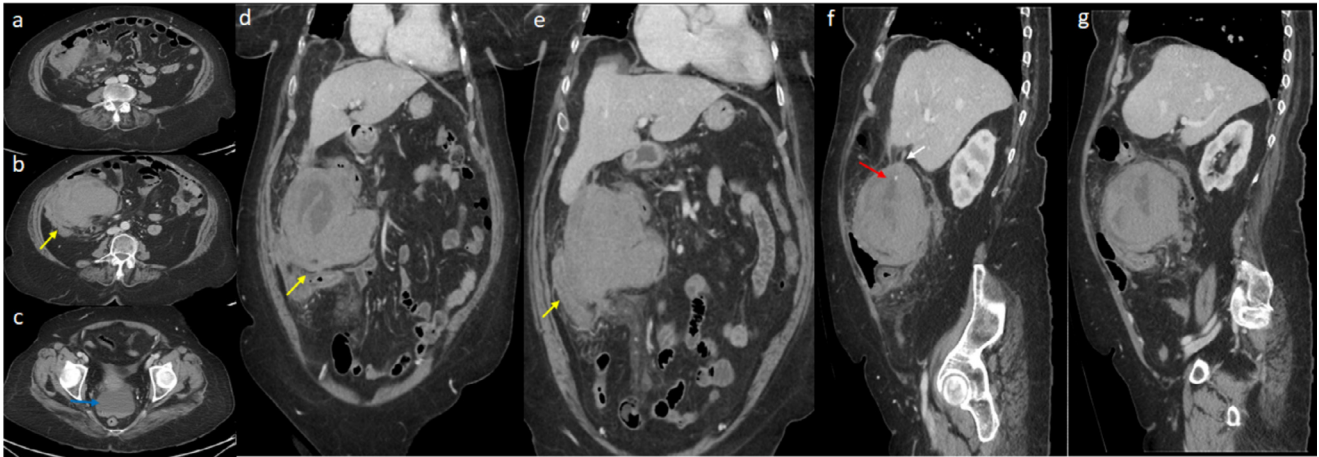


Fig. 1 – Figs. 1A-G Contrast-enhanced abdomen and pelvis CT scan obtained during the portal venous phase. Axial (1a-1c), Coronal (1d-1e), and Sagittal (1f-1g) views show a large heterogeneous predominantly dense mass with calcification (red arrow) occupying most of the right abdominal cavity. The mass was causing mass effect and was adherent to the adjacent small bowel loops, cecum and ascending colon showing edematous wall thickening and hyper dense content. This was suspect for underlying fistulation (yellow arrow). There were also mild amounts of Hemoperitoneum and fluid noted which indicated perforation (blue arrow). The cystic duct was not dilated and was tethered to the superior aspect of the mass, showing smooth distal tapering, and thus raising the possibility of biliary communication and gallbladder pathology with volvulus (Color version of figure is available online)

and iatrogenesis. Direct clinical diagnosis of HC is a challenge because of its wide presentation of symptoms, ranging from acute abdominal pain and acute biliary obstruction to hematemesis; therefore, thorough imaging is crucial for diagnosis.

Ultrasound sonography (US) is the standard initial test for evaluating gallbladder and biliary tree pathology due to its high sensitivity. However, computed tomography (CT) is the best radiological tool for assessing the complete spectrum, possible causes, and complications of gall bladder disease. MRI can also be useful in certain cases [2].

In this case, a 74-year-old woman presented to our emergency department (ED) with a history of 10 days of intermittent abdominal discomfort and continuous vomiting after oral ingestion along with watery diarrhea. The patient had a history of mitral valve replacement 13 years previously, and had been taking warfarin since then. She denied any history of fever, weight loss, or night sweats, and did not complain of any gastrointestinal bleeding at the time of presentation. Other HC symptoms were negative. Upon physical examination, the patient appeared fatigued, but was not in any kind of distress or pain, and was vitally stable. She did not appear pale or jaundiced. Abdominal examination revealed distension and generalized abdominal tenderness, primarily on the right side without rebound tenderness, indicating a colicky nature. No palpable masses or guarding was observed.

An emergent cross-sectional abdominopelvic CT with intravenous contrast (Figs. 1A–G) was performed, revealing a large heterogeneous predominantly dense mass with punctate calcification (Fig. 1F [red arrow]) occupying most of the abdominal cavity on the right side. This caused a mass effect and appeared adherent to the adjacent organs, including the small bowel loops, cecum, and ascending colon

(Fig. 1B,D,E [yellow arrow]), with no signs of bowel obstruction. The involved bowel demonstrated edematous wall thickening and hyperdense content, which was suggestive of possible fistulation and underlying bleeding. In addition, there were an associated perilesional inflammatory change, edema in the form of fat stranding, and reactive right lower quadrant mesenteric lymph nodes. There were also mild amounts of hemoperitoneum and fluid, which indicated perforation (Fig. 1C, [blue arrow]). A normal gallbladder appearance was not observed in the study. The cystic duct was not dilated and appeared to be tethered to the superior aspect of the mass, which raised the possibility of biliary communication and provided clues as to mass origin and pathology (Fig. 1F [white arrow]). At the site of communication between the cystic duct and the mass, luminal narrowing was observed, which was suspect for the possibility of volvulus. There were no subcapsular or perihepatic hematomas or signs of intra- or extrahepatic biliary tree dilatation. Although we did not obtain the arterial phase, no appreciable vascular abnormalities or aneurysms were identified. The general findings were suggestive of hemorrhagic acute calculus cholecystitis. To confirm the CT findings, to further evaluate the large lesion, and to exclude any underlying malignancy, additional imaging via abdominal MRI and magnetic resonance cholangiopancreatography (MRCP) was recommended. Unfortunately, US was not performed due to the misleading clinical picture, although it is normally considered the first imaging modality for evaluating gallbladder and biliary system pathology due to its high sensitivity.

The patient was kept in the ED until she was hemodynamically stabilized and was then admitted to the surgical department. MRCP was not performed due to patient status during examination time. Abdominal MRI with intravenous

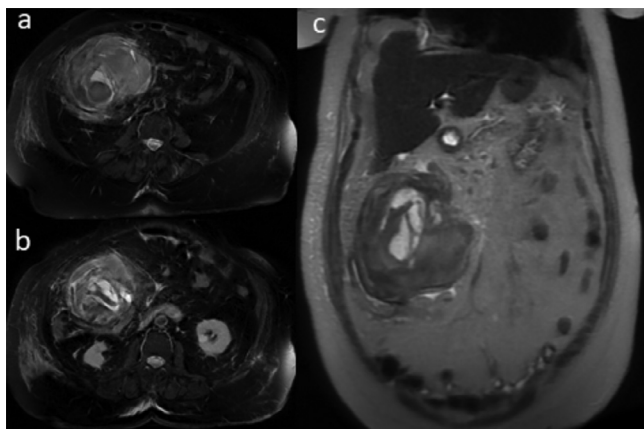


Fig. 2 – Abdominal T2- weighted MR image (Figs. 2A,B) Axial and; (2C) Coronal views show a large heterogeneous mass outside the gallbladder fossa of intermediate to high T2- weighted image signal intensity (2C), with intraluminal areas of dark T2WI signal intensity and T1WI hyper intensity dependent (Fig. 3A) suggestive of hemosiderin deposition/hematoma. The lesion also shows areas of cystic structures of bright T2- weighted image signal intensity and low T1- weighted image signal intensity without enhancement, indicating areas of necrosis

gadolinium contrast was performed. The intermediate to high T2- weighted image signal intensity (Fig. 2C) revealed a large heterogeneous enhancing mass outside the gallbladder fossa measuring $10.1 \times 12.2 \times 11$ cm. The coronal precontrast T1-weighted gradient-echo LAVA image [Fig. 3A] revealed an irregular hyperintense thick gallbladder wall, and a $2.6 \times 4.4 \times 7.3$ cm hyperintense dependent intraluminal content appeared as a region with low and dark signal intensity on the spin-echo T2-weighted image [Fig. 2A] within the mass, thereby suggesting hemosiderin deposition in the hematoma. The intraluminal hematoma did not show any enhancement on the T1-weighted LAVA dynamic gadolinium-enhanced image (Fig. 3B,C). The contrast-enhanced T1-weighted image showed heterogeneous diffuse enhancement of the thickened irregular gallbladder wall. The lesion also revealed cystic-like structures of bright T2- weighted image signal intensity and low T1- weighted image signal intensity without enhancement, indicating areas of necrosis. Although an MRCP image was not obtained, the cystic duct appeared to arise from the superior aspect of the mass, indicating gallbladder origin (Fig. 3B, arrowhead).

Exploratory laparotomy, cholecystectomy, right hemicolectomy, and ileocolic side-to-side anastomosis were performed, revealing a necrotizing and hemorrhagic enlarged gallbladder with cholelithiasis occupying the right abdominal cavity. This enlargement appeared to be adherent and fistulizing into the cecum, with no signs of bowel perforation. Auto-avulsion of the cystic duct was present, and the cystic stump was identified and stitched. The pathologic examination revealed a large distended hemorrhagic gallbladder weighing 615.1 grams and measuring $16.0 \times 14.0 \times 7.0$ cm. Sectioning revealed that the gallbladder was distended and entirely filled with a large blood

clot measuring $10.0 \times 9.0 \times 8.5$ cm. There was a wide gallbladder wall defect measuring 6.5×5.5 cm. Upon opening specimens from the cecum and ascending colon, their lumen were also filled with blood clots, and the mucosal surface and wall both appeared hemorrhagic. Pathological examination did not reveal any abnormal cells indicating malignancy. The post-operative period was uneventful, and the patient recovered well.

Discussion

Acute cholecystitis is a well-known entity commonly seen in EDS with varying presentations and complications. Since it can be a life-threatening condition, early diagnosis is crucial. One rare complication of acute cholecystitis is HC, which is known to be associated with anticoagulant therapy, now considered an important predisposing factor due to widespread use [13]. Cholelithiasis is also a leading cause of HC in up to 50% of cases [3,4]. Other reported non-lithiasic etiologies include hematological issues related to the use of anticoagulation therapy; vascular anomalies and aneurysms; and increased bleeding tendency disorders such as hemophilia, renal failure, and cirrhosis [5–9]. There are also non-inflammatory conditions linked to HC, such as trauma, malignancy, and iatrogenic causes [10,11].

The pathophysiology of HC occurs in the context of gallbladder inflammation and mural damage, leading to a cascade of unfortunate events in the form of gallbladder wall ischemia, necrosis, and erosions that eventually evolve into intraluminal bleeding and hematoma formation, perforation [14], and hemoperitoneum. The gallbladder fundus is the most frequent site of perforation because of its poor vascularity [4] which occurs as a sequel to acute gangrenous cholecystitis, with an estimated prevalence of 8%–12% and mortality of 24.1% [12]. Other reported predisposing factors that increase the risk of gallbladder perforation are male sex, advanced age, systemic disease, and the chronic use of corticosteroids [15].

HC has a clinical presentation similar to that of acute cholecystitis, such as abdominal pain, fever, nausea, and vomiting, which are generally poor. In addition, since bleeding occurs, some patients may present with anemia and hypotension, leading to shock. Additionally, blood clot formation is likely. Blood clots are eventually evacuated through the cystic duct and pass into the gastrointestinal tract, causing biliary obstruction, jaundice, hematemesis, and melena. Considering its non-specific and wide presentation, a specific clinical diagnosis is difficult for HC; therefore, imaging studies play a major role in establishing a diagnosis and assessing alternate diagnoses [16]. CT is most often performed in patients with acute abdominal pain. Although CT has not been advocated as a primary imaging examination for acute right upper quadrant pain, it can confirm or at least rule out the diagnosis of acute cholecystitis complications with nearly 90% negative predictive value. CT is also often preferred over MRI mainly because of its speed [1]. Notably, the sensitivity for gallstone detection on CT is only approximately 75% and is dependent on the density of the stone relative to bile [3].

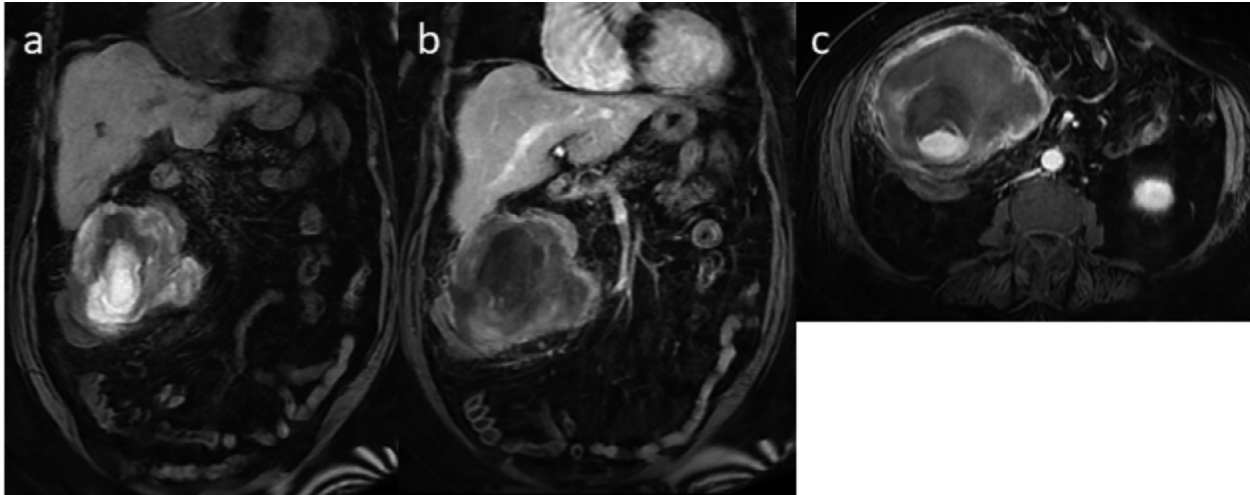


Fig. 3 – (Fig. 3A) Coronal pre-Gadolinium fat suppressed T1- weighted MR image of the abdomen shows irregular hyper intense thick gallbladder wall. Hyper intense dependent intraluminal content appeared as a region of dark signal intensity on the T2-weighted image (Fig. 2A) suggestive of hemosiderin deposition/hematoma. (Figs. 3B and C) Coronal and axial dynamic post-Gadolinium enhanced fat suppressed T1- weighted MR image of the abdomen shows diffuse heterogeneous and thick wall enhancement of the mass. The intraluminal hematoma did not show any post-contrast enhancement. Although MRCP images were not obtained, the cystic duct appeared to arise from the superior aspect of the mass, indicating a gallbladder origin (Fig. 3B)

As with CT, MRI is not advocated as a primary imaging examination to evaluate acute right upper quadrant pain; however, several studies have suggested that abdominal MRI is a reliable alternative and can be particularly helpful in patients who are difficult to examine with US [17–19]. Although factors such as longer acquisition times limit its use in the emergency setting, more consistent visualization of the extrahepatic biliary tree is an important advantage of its use [20–21]. MRI also has a higher sensitivity than US for the diagnosis of acute cholecystitis [12].

Compliance with Ethical Standards

Ethical approval: This article does not contain any studies with human participants or animals performed by any of the authors. **Informed Consent:** The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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