

Haemorrhagic cholecystitis: a rare but life-threatening variant of acute cholecystitis

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Haemorrhagic cholecystitis (HC) is a rare complication of cholecystitis. Since its first description in the 1970s, HC remains under-recognised and may result in catastrophic intraperitoneal bleeding or gallbladder rupture if not promptly identified and treated. The condition may mimic more common biliary pathologies but is distinguished by haemorrhage into the gallbladder lumen or wall, frequently leading to necrosis and perforation. Awareness of this diagnosis is critical in the emergency, surgical and radiologic settings.

HC represents a small percentage of acute cholecystitis cases—incidence estimates range from 0.5% to 1.5% in retrospective surgical analyses, with likely overestimation in older literature of up to 5%. Primarily, it affects elderly patients and is more frequently associated with comorbidities such as coagulopathy, anticoagulant therapy, trauma, malignancy and systemic vasculitis. However, it can also occur in otherwise healthy individuals in the context of severe inflammation.

Case presentation

Mr X, an 83-year-old man, was admitted with a 3-day history of epigastric pain and nausea. He had recently been transferred to the rehabilitation hospital following an admission for open reduction and internal fixation of a periprosthetic femur fracture under the orthopaedic team 20 days prior. During this admission he developed an ultrasound-proven occlusive thrombus of his peroneal and soleal veins. Rivaroxaban was commenced to prevent upstream migration of the deep vein thrombosis. He had a background of congenital deafness, prostate cancer (grade 1, Gleason 3+3 adenocarcinoma), autoimmune hepatitis, primary biliary cirrhosis, previous total hip joint replacement and hypertension. Notable regular medications included prednisone and aspirin. Prior to admission he was fully independent, lived with his wife and was a non-smoker.

The general surgical team were contacted 72 hours post-diagnosis of deep vein thrombosis, following an onset of tachycardia and hypoxia with computed tomography (CT) pulmonary angiogram findings suggestive of HC, without evidence of pulmonary embolus. On examination, he had right upper quadrant tenderness, and a positive Murphy's sign. Biochemical analysis revealed liver function derangement with a bilirubin of 31µmol/L, alkaline phosphatase of 300IU/L and gamma-glutamyl transferase of 164IU/L. His white cell count was $11.1 \times 10^9/L$ and C-reactive protein was 116mg/L. His haemoglobin was stable at 99g/L. He had a moderate acute kidney injury with serum creatinine of 176µmol/L and estimated glomerular filtration rate of 30mL/min/1.73m². A dedicated CT abdomen–pelvis with intravenous contrast across portal venous and arterial phases was performed. It confirmed the diagnosis of HC with hyperdense material in the gallbladder and biliary tree. No active bleed was noted. Subsequent biliary ultrasonography exonerated gallstone pathology. He was commenced on cefuroxime and metronidazole and resuscitated with intravenous crystalloid fluid. He was afebrile throughout.

Given the underlying case complexity and anticoagulation requirement, hepatobiliary input was sought. The recommendation was for non-operative management given his advanced age, comorbidity burden and absence of gallstone disease. A multidisciplinary guided discussion with the patient resulted in cessation of his rivaroxaban therapy. Following initial haemodynamic and clinical improvement, the liver function deteriorated transiently. After medication reconciliation and antibiotics adjusted for renal impairment, he improved and was discharged with oral antibiotics.

Discussion

HC comprises approximately 0.5–1.5% of

acute cholecystitis cases in surgical series and is relatively under-represented in surgical literature.¹ It primarily affects elderly patients and is more frequently associated with comorbidities such as coagulopathy, anticoagulant therapy, trauma, malignancy and systemic vasculitis.² Zhang et al.³ suggest that the inhibition of platelet and prostaglandin function of non-steroidal anti-inflammatories and aspirin therapy may be overlooked at time of admission. Other pharmaceutical agents with haemorrhagic risk should be considered, including methotrexate (risk of thrombocytopenia and hepatotoxicity), and systemic glucocorticoids (impaired clotting ability and mucosal injury).⁴ Nonetheless, HC can also occur in otherwise healthy individuals in the context of severe inflammation. The proposed pathophysiological mechanism is of mucosal ischaemia and necrosis secondary to gallbladder distension and inflammation, which leads to erosion of small blood vessels or the cystic artery itself.^{4,5} This is exacerbated by infection-related inflammatory mediators and can be worsened by mucosal erosion by gallstones, reperfusion injury, coagulopathy, malignancy or vascular disease.^{4,5} The resultant intraluminal haemorrhage may increase gallbladder pressure, perpetuating ischaemic injury and potentially leading to wall necrosis, haemobilia and perforation. Perforation occurs in 2–15% of cases and is predominantly at the fundus.^{2,6} Symptoms and signs are often indistinguishable from uncomplicated acute cholecystitis and, in some cases, hypotension or shock may be the presenting feature, especially in patients with massive haemorrhage or rupture and can at times present with pain like aortic dissection.⁷

Imaging plays a crucial role in diagnosis. Ultrasonography may reveal echogenic intraluminal material (clots) without acoustic shadowing, suggestive of intraluminal haemorrhage with thrombosis. CT is superior in detecting high-attenuation intraluminal contents suggestive of blood, gallbladder wall thickening and active extravasation.^{4,5,8} Magnetic resonance imaging and magnetic resonance cholangiopancreatography (MRCP) can further delineate haemobilia or differentiate blood from bile or exudate and also intraluminal and intramural haemorrhage.⁹ Endoscopic evaluation (e.g., endoscope retrograde cholangiopancreatography [ERCP]) may demonstrate blood draining from the ampulla of Vater in cases of haemobilia.² Laboratory findings often include leucocytosis, elevated liver enzymes,

anaemia and coagulopathy, particularly in anticoagulated patients.⁵

Management of HC depends on multiple factors, including the patient's overall stability, comorbidities, anticoagulation status, risk of further bleeding and underlying aetiology. Initial treatment includes fluid resuscitation, reversal of coagulopathy and broad-spectrum antibiotics. Laparoscopic cholecystectomy remains the mainstay in patients with acceptable operative risk.¹⁰ In high-risk patients or those unfit for surgery, percutaneous cholecystostomy may be a temporising measure.⁴ In cases of active bleeding, interventional radiology-guided embolisation of the cystic artery can be life-saving.¹⁰ In those that are medically comorbid, a trial of antibiotics with avoidance of interventional measures is a reasonable alternative. Overall, timely intervention is essential, as delayed treatment is associated with increased risk of gallbladder rupture, peritonitis and death. Mortality rates for HC are reported between 10% and 20%, particularly in elderly or anticoagulated patients.⁷

In this case, our patient was elderly, with a significant comorbidity burden including biliary cirrhosis and autoimmune hepatitis, which may have further complicated surgical decision making. His recent orthopaedic surgery and concurrent venous thrombosis necessitated anticoagulation therapy. The multidisciplinary decision to pursue non-operative management was guided by the absence of gallstones, the lack of evidence of ongoing haemorrhage or perforation on CT angiogram and the patient's relative haemodynamic stability. Conservative management involved intravenous antibiotics, close monitoring and temporary cessation of anticoagulation, with plans to reassess the need for cholecystectomy should his clinical condition have deteriorated. Were anticoagulation deemed necessary, surgical management may have been more strongly considered to prevent recurrent haemorrhage and complications from a potential re-bleed. However, in patients where anticoagulation can be safely withheld or reversed, conservative management may allow the inflammation and haemorrhage to resolve, especially in the absence of gallstones.

This nuanced approach reflects the need to balance surgical risks with the potential benefits of definitive management in complex patients. Ultimately, each case of HC must be managed on an individual basis, considering the unique interplay of patient factors, disease severity and

resource availability. Multidisciplinary collaboration between surgeons, medical specialists and radiologists is essential to determine the most appropriate course of action. Continued vigi-

lance and early surgical consultation remain key in optimising patient outcomes in this rare but life-threatening condition.

COMPETING INTERESTS

Nil.

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