GALLBLADDER PERFORATION AND MASSIVE INTRAPERITONEAL BLEEDING SECONDARY TO HAEMORRHAGIC CHOLECYSTITIS

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ABSTRACT A 76-year-old man on apixaban presented with abdominal pain, vomiting, and signs of haemorrhagic shock. Imaging showed large-volume haemoperitoneum with active arterial extravasation in the gallbladder fossa. The patient proceeded to emergency laparotomy, where haemorrhage was thought to arise from within a perforated gallbladder. Cholecystectomy was performed, and haemostasis was achieved. Histology showed features of subacute on chronic cholecystitis, with some sections suggestive of underlying arteritis. Haemorrhagic cholecystitis is rare, and gallbladder perforation with massive intraperitoneal haemorrhage is rarer still. This article presents a case report and literature review of this entity.

KEYWORDS haemorrhagic cholecystitis, intraperitoneal haemorrhage, anticoagulation, laparotomy

Introduction

Haemorrhagic cholecystitis is rare, and gallbladder perforation with massive intraperitoneal bleeding is an even rarer complication. In the last decade, only 14 cases of this phenomenon have been described in English literature. Increasing awareness of this potentially fatal condition is critical. We present a case of haemorrhagic cholecystitis with catastrophic bleeding that was managed by emergent laparotomy and cholecystectomy.

Case Report

A 76-year-old man with a history significant for hypertension, dyslipidaemia, smoking and osteoarthritis underwent an elective right total hip replacement. Two weeks later he was found to have right leg deep vein thromboses, for which apixaban was commenced. After five days, the patient developed worsening abdominal pain associated with vomiting. In the subsequent hours, he became shocked with a systolic blood pressure of 60 mmHg and tachycardia up to 120 beats per minute. Clinically he had increasing abdominal tenderness and distension, and altered level of consciousness.

Laboratory workup showed a haemoglobin drop from 124 to 92 g/L, leukocytosis of 21.1×10⁹/L, and an international normalised ratio of 1.7. The patient also had new derangement in liver function, with a bilirubin of 57 g/L, alkaline phosphatase of 315 U/L, gamma-glutamyl transferase of 608 U/L, alanine transaminase of 803 U/L, and aspartate transaminase of 1180 U/L. Bedside ultrasound demonstrated free intraabdominal fluid. Urgent abdominal computed tomography (CT) revealed large-volume haemoperitoneum extending from the gallbladder fossa to Morison’s pouch and tracking down the right paracolic gutter and pelvis. There was active arterial extravasation in the gallbladder fossa, with a possible feeding artery arising from the right hepatic or cystic artery. The gallbladder itself was difficult to assess but cholecystitis was noted (Fig 1).

The patient was transfused packed red cells, and coagulopathy was corrected with tranexamic acid, prothrombinex and fresh frozen plasma. The haemorrhage was deemed unamenable to angioembolisation, hence the patient proceeded to emergency laparotomy. Intraoperatively, large-volume blood was found surrounding a perforated gallbladder. Clot was also identified within the lumen of the gallbladder, thought possibly to arise from an ulcer or erosion related to cholelithiasis (Fig 2). The cystic artery appeared normal. Cholecystectomy was performed, and haemostasis was achieved.

Histologically, the gallbladder showed features of subacute...
on chronic cholecystitis, with haemorrhagic disruption of the submucosa and muscularis. Intraluminal and serosal haemorrhages were noted. Interestingly, some sections showed eccentric fibrinoid necrosis affecting submucosal arteries, suggesting an underlying arteritis.

The patient made an uncomplicated recovery, and apixaban was recommenced on the second postoperative day. He was completely well on subsequent follow-up at six months.

Discussion

The pathophysiology of haemorrhagic cholecystitis is thought to involve cholelithiasis eroding the cystic artery causing pseudoaneurysm formation. Intraluminal bleeding from this pseudoaneurysm, in combination with cystic duct obstruction, leads to rupture of the hyper-pressurised gallbladder [1]. Alternatively, acute cholecystitis with perforation and bleeding from the gallbladder wall defect may be another mechanism of intraperitoneal haemorrhage. Both transepithelial perforation and free rupture into the peritoneal cavity can lead to massive haemoperitoneum [2].

This case was associated with the recent commencement of apixaban, supporting anticoagulation or antiplatelet therapy as the major risk factor for haemorrhagic cholecystitis and its complications [1, 2]. Patients with underlying bleeding diatheses also appear to be at higher risk [3, 4]. Interestingly, our histological findings suggest that an underlying vasculitis may be another predisposing factor. Cases of haemorrhagic cholecystitis associated with microscopic polyangiitis and Churg-Strauss syndrome have been reported, though none had massive intraperitoneal haemorrhage [5, 6].

Early recognition of this potentially fatal condition is crucial but challenging. Its presentation resembles that of acute calculous cholecystitis, with abdominal pain and nausea or vomiting predominating. Haemobilia and melaena may be more specific signs of haemorrhagic cholecystitis [7]. The obstructive liver function tests in our case suggest that relative biliary obstruction with haemobilia may have been present. Other supportive laboratory findings include acute anaemia and leucocytosis [1-3, 7]. The development of hypovolaemic shock should raise concern for massive intraperitoneal haemorrhage.

Imaging is usually diagnostic. While ultrasound is the first-line imaging modality for acute cholecystitis, patients presenting with gallbladder perforation and massive intraperitoneal haemorrhage almost invariably proceed to a contrast-enhanced CT scan. Our case had typical CT findings, with large-volume haemoperitoneum surrounding the gallbladder fossa, and active contrast extravasation [1, 3, 4, 7]. Cystic artery pseudoaneurysm is reportedly a specific sign [1], and disruption of the gallbladder wall may also be seen [2, 8].

Definitive management of haemorrhagic cholecystitis is cholecystectomy. Most patients with massive intraperitoneal haemorrhage undergo laparotomy like in our case, but a laparoscopic approach appears feasible in the appropriately selected patient [9].

Increasingly, there is a trend toward a two-step approach of embolisation of the culprit vessel prior to cholecystectomy [1, 8]. However, our case shows that immediate surgical intervention, without the benefits of angioembolisation, may be necessary.

Conclusion

Haemorrhagic cholecystitis is an under-recognised phenomenon that can lead to significant morbidity or mortality. In a patient who presents clinically with acute cholecystitis, the development of haemodynamic instability, particularly in the setting of anticoagulation use should trigger suspicion of massive intraperitoneal bleeding. While CT imaging can help identify a target for angioembolisation, the clinician should be mindful that immediate surgical intervention may still be required.

Consent For Publication

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Conflict of Interest
The authors declare that they have no competing interests.

Authors’ Contribution
JY was responsible for the literature review and drafting of the case report. PP was responsible for research conceptualisation and proofreading of the manuscript. JA was responsible for research supervision and manuscript revision. All authors approved the final manuscript.

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References


