CASE REPORT

Haemorrhagic cholecystitis: an unusual cause of upper gastrointestinal bleeding

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SUMMARY

Haemorrhagic cholecystitis is a rare cause of upper gastrointestinal bleeding and is a difficult diagnosis to make. This case report describes an orthopaedic patient, who developed deranged liver function tests and anaemia after a hemiarthroplasty of the hip. The patient had upper abdominal pain and black stools which clinically appeared to be melaena. An ultrasound scan of the abdomen was inconclusive, and therefore a CT was performed and the potential diagnosis of haemorrhagic cholecystitis was raised. An endoscopic evaluation of the upper gastrointestinal tract showed no evidence of other causes of upper gastrointestinal bleeding. Following an emergency laparotomy and cholecystectomy, she recovered well. This report aims to increase awareness about the uncommon condition of haemorrhagic cholecystitis, and to educate regarding clinical and radiological signs which lead to this diagnosis.

BACKGROUND

This is the presentation of an interesting case from a clinical perspective in which the eventual diagnosis is a rare one. However, a number of common presentations seen in medicine, including postoperative anaemia, jaundice and melaena secondary to upper gastrointestinal (GI) bleeding were present. The case encourages the reader to think about the different causes of each of these symptoms and signs.

The case also had some management issues which have been highlighted. There was a delay in organising appropriate investigations and involving appropriate teams, which something others can learn from and try to prevent in their own practice.

CASE PRESENTATION

A frail 79-year-old woman with a history of Parkinson's disease presented to an accident and emergency department 2 weeks after a fall, and was found to have sustained an intracapsular fracture of the right femoral neck. The next day the patient underwent a right hemiarthroplasty. An estimated blood loss was approximately 300 mL. Postoperatively, haemoglobin (Hb) dropped from 110 g/L (11.0 g/dL) to 66 g/L (6.6 g/dL); therefore, she was transfused 3 units of red blood cells.

The day after transfusion, the patient was noted to be clinically jaundiced. She reported intermittent colicky abdominal pain and said she had similar episodes over the past few months. She had no other associated symptoms. Examination findings consisted of generalised abdominal tenderness with stable observations and no fever. Blood tests showed a post-transfusion Hb of 80 g/L (8 g/dL), somewhat less than expected. They also showed deranged liver function tests with a bilirubin of 147 μmol/L, alkaline phosphatase 704 iU/L and alanine transaminase 300 iU/L. The patient was discussed with the medical team who felt there was no medical issue present. Her case was then discussed with the orthopaedic registrar who was of the opinion that because it was a weekend and the patient was frail it was unlikely that the general surgeons would operate immediately, and therefore there was limited benefit from investigating further that day. The orthopaedic registrar advised hydration with intravenous fluids and for a further 2 units of red blood cells to be transfused.

The next day the patient's abdominal pain had resolved. She had passed some black stool, and digital rectal examination demonstrated black and tarry stools which clinically were thought to be melaena. There was no history of vomiting or haematemesis. An abdominal examination demonstrated a soft abdomen with right upper quadrant tenderness. Blood tests showed an improvement in liver function and a more expected rise in Hb to 103 g/dL. At this point, the most likely differential diagnosis was an upper GI bleed, and it was decided to observe her for further episodes. Unfortunately, the patient had further episodes of black tarry stools, and her Hb decreased from 103 to 81 g/dL. Treatment for an upper GI bleed was initiated. An intravenous proton pump inhibitor was started, and 2 further units of red blood cells were transfused. Aspirin and low-molecular-weight heparin (dalteparin) were stopped. An ultrasound scan and an upper GI endoscopy were arranged.

INVESTIGATIONS

Ultrasound scan:

- 8 cm mass forming heterogeneous lesion in the right upper quadrant independent of the liver, right kidney and colonic hepatic flexure;
- Difficult to assess the gallbladder, and biliary tree not dilated;
- Moderate ascites;
- Liver, pancreas, spleen and kidneys were normal.

Following the ultrasound scan, a CT was arranged by the radiology department to better assess the right upper quadrant mass.

CT (as demonstrated in figure 1):

- Distended gallbladder (cholecystitis) filled with inhomogeneous, patchy hyperdense material,

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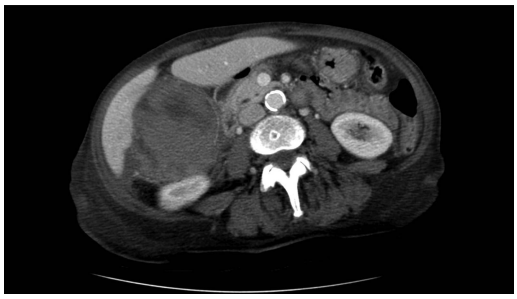

Figure 1 Cross-sectional image from CT demonstrating gallbladder findings.

which is indicative of primary spontaneous gallbladder haemorrhage;
- Common hepatic duct compressed by the gallbladder, leading to intrahepatic biliary dilatation;
- Small amount of ascites.

An oesophagogastroduodenoscopy endoscopy was performed the day after the CT scan. It showed no upper GI bleed, and no haemobilia.

DIFFERENTIAL DIAGNOSIS

Initially, the differential diagnosis involved the causes of jaundice. Given the history of intermittent abdominal pain over the past few months and obstructive picture on liver function tests, the main differential diagnosis was of complication of gallstones, that is, cholecystitis, cholangitis or gallstone impaction in the common bile duct. Also under consideration were haemolysis secondary to recent blood transfusion, pancreatic malignancy and drug-induced jaundice given she had prophylactic flucloxacillin perioperatively during hemiarthroplasty.

However, once the patient started having episodes of melaena the differential diagnosis turned to causes of upper GI bleeding, the most likely of which were inflammation (gastritis, duodenitis), duodenal or gastric ulcers and drugs (eg, non-steroidal anti-inflammatory drugs, low molecular weight heparin and aspirin).

Hepatic causes such as cirrhosis and oesophageal varices were also considered, but given the lack of history of previous liver disease this was thought to be unlikely.

It was not until further investigations had taken place that the differential diagnosis of haemorrhagic cholecystitis was considered.

TREATMENT

Following the radiological findings, the general surgeons assessed the patient. Intravenous antibiotics were started following a discussion with the patient and her family, the outcome of which was to confirm a trial on conservative management. However, the patient deteriorated with a further episode of dark stools and became increasingly agitated. Therefore, she underwent a laparoscopy, laparotomy and an open cholecystectomy. Operative findings were consistent with haemorrhagic cholecystitis: there was an enlarged congested gallbladder with an anterior perforation. There was also a large quantity of blood within the peritoneal cavity. An estimated blood loss during the procedure was 1000 mL.

Postoperatively the patient was transferred to intensive therapy unit (ITU). Unfortunately, she had a continuous oozing from the wound postoperatively, and Hb decreased to 64 g/dL. Therefore, she was taken back to the theatre for a second-look laparotomy after 2 days. The operative findings from this laparotomy were oozing from omental adhesions, but with no active bleeding. The oozing settled with packing and surgicel.

The patient returned to ITU postoperatively. She was returned to the ward after 4 days, where she recovered well, in terms of abdominal surgery and recent hip surgery.

OUTCOME AND FOLLOW-UP

The patient recovered well after her second laparotomy. She built up to a normal diet and started to return to normal mobility (walking with a frame). She was discharged to a community rehabilitation bed after a total of 21 days in the hospital.

There was no orthopaedic or surgical follow-up required.

Histology of gallbladder was found to be consistent with haemorrhagic cholecystitis.

DISCUSSION

Haemorrhagic cholecystitis is a rare condition which should be considered in patient with a history of trauma, bleeding disorder and diathesis (eg, cirrhosis and renal failure) or findings consistent with acalculous cholecystitis.[1] When the presentation is with upper GI haemorrhage, upper abdominal pain and jaundice, it is known as Quinke's triad; however, only 22% of the cases present in this manner.[2] It more typically presents with symptoms of cholecystitis but with associated haematemesis, haemobilia on endoscopy/endoscopic retrograde cholangiopancreatography (ERCP) or melaena (as in the case of this patient) due to blood entering the GI tract. It is worth bearing in mind that a blood clot within the gallbladder may cause common bile duct obstruction and cholangitis. It is, therefore, a difficult diagnosis to make clinically as it is so similar to other gallstone-related diseases.[1]

In haemorrhagic cholecystitis, on ultrasound scan, the gallbladder usually (up to 74% of patients) has an atypical appearance including focal irregularities of the gallbladder wall and non-shadowing immobile echoes.[3] CT may indicate haemorrhagic cholecystitis by high-attenuation material within the gallbladder lumen.[4 5] Nevertheless, it is a difficult diagnosis to make radiologically. If haemobilia is seen on endoscopy or ERCP, then haemorrhagic cholecystitis should be considered.[1]

Pathologically, in the case of haemorrhage as a complication of acute cholecystitis, the transmural inflammatory process is thought to cause infarction and necrosis of the gallbladder mucosa, which results in the destruction of vessel walls and subsequent haemorrhage.[3] Haemorrhage as a result of acute cholecystitis can also be due to the development of a cystic artery pseudoaneurysm which pathologically is thought to be due to either early thrombosis of the cystic artery secondary to inflammation or erosion of the cystic artery wall by a large gallstone.[6]

In the case of this patient, as in other reported cases, it is unclear why she developed haemorrhagic cholecystitis. Other than standard clotting studies this patient was not fully investigated for a bleeding disorder. It may be that the combination of antiplatelet therapy in the form of aspirin and low molecular weight heparin was enough to result in this unusual condition. A history of intermittent colicky abdominal pain implies the patient may have had previous gallstones, and therefore may have erosions from previous stones or have developed haemorrhage as a complication of acute cholecystitis.

Learning points

- Consider haemorrhagic cholecystitis in patients with findings consistent with acalculous cholecystitis, and a drop in haemoglobin especially if there is a history of trauma or bleeding disorders.
- Quinke's triad consists of upper gastrointestinal (GI) bleeding, upper abdominal pain and jaundice. It is indicative of haemorrhagic cholecystitis.
- Haemorrhagic cholecystitis is a rare but potentially fatal complication of anticoagulation therapy. Its diagnosis and management are important in preventing the fatal outcome.

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