CASE REPORT

Haemoperitoneum secondary to intrahepatic gallbladder perforation

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SUMMARY

A 70-year-old man presented to the accident and emergency department with a 1-day history of right upper quadrant pain and nausea. Examination revealed mild tenderness in the right upper quadrant but no evidence of peritonism or haemodynamic instability. The patient was admitted to the general surgical ward with a diagnosis of cholecystitis and remained stable overnight. In the morning, he developed acute severe pain in the upper abdomen. Examination found him to be tachycardic, tachypnoeic and to have peritonism in the upper abdomen. An urgent CT scan demonstrated cholecystitis but also a small amount of fluid in the pelvis, with Hounsfield units suggestive of blood. The patient proceeded to laparotomy and was found to have massive haemoperitoneum secondary to intrahepatic gallbladder perforation causing liver capsule tear. Cholecystectomy was performed and the liver packed until haemostasis was achieved. The patient made an uneventful recovery.

BACKGROUND

Gallbladder perforation is a rare complication of acute calculous cholecystitis, occurring in approximately 2–11% of cases.¹ The Niemeier classification of gallbladder perforations describes perforations as follows: type I includes patients with free perforation into the peritoneal cavity, type II describes patients with localised perforation and type III describes patients with cholecystoenteric fistulas.² The mortality ranges from 12% to 42%.^{3–4} The literature shows examples of acute gallbladder perforation mostly managed surgically, but on rare occasions managed conservatively.^{3–5} Owing to the acute deterioration that occurs, this case highlights the importance of having a high index of suspicion for atypical cases of cholecystitis and the need for prompt surgical intervention in such cases.

CASE PRESENTATION

A 70-year-old man presented to accident and emergency with a 1-day history of right upper quadrant pain and nausea. Medical history included rheumatoid arthritis, psoriatic arthritis, hereditary motor polyneuropathy and previous tuberculosis. The patient reported no significant family history, alcohol intake or smoking history. Initial examination revealed mild tenderness in right upper quadrant but no evidence of peritonism. Observations were stable and haematological investigations revealed normal liver function tests and mildly raised inflammatory markers. The patient was

admitted to the general surgical ward and remained stable overnight. Antibiotics and intravenous fluids were given. The working diagnosis was cholecystitis. Early the following morning, the patient suddenly deteriorated with acute onset of severe pain in the upper abdomen. On examination, he was found to be tachycardic and tachypnoeic with upper abdominal peritonism. An urgent CT was organised, which demonstrated a distended gallbladder with pericholecystic inflammation, in keeping with acute cholecystitis. There was also some free fluid noted in the pelvis, of which the Hounsfield units were felt to be possibly in keeping with blood, and an irregularity to the inferior angle of the liver thought possibly to be a subcapsular collection. The patient proceeded straight to laparotomy due to his acute deterioration. He was found to have massive haemoperitoneum secondary to an intrahepatic gallbladder perforation causing a liver capsule tear. The gallbladder had perforated into the liver and bile; gallstones and blood were found exiting from the liver. The findings were in keeping with a Niemeier type I perforation of the gallbladder, as previously described.² The gallbladder was drained and then excised retrogradely. The liver was packed and PerClot (CryoLife) applied until haemostasis was achieved. The patient was transferred to intensive care for 24 h where he received blood transfusions. After transfer back to the ward, he made an uneventful recovery and was discharged home day 10 postoperatively.

DISCUSSION

From studying the literature, it is clear that gallbladder perforation is a rare complication of gallstone disease.¹ Furthermore, the findings of a haemoperitoneum secondary to gallbladder perforation was only seen approximately 18 times in the literature, highlighting the unusual nature of this case.

The sequence of events leading to gallbladder perforation is thought to be due to transmural inflammation leading to necrosis and eventually perforation.⁶

The factors thought to contribute to spontaneous gallbladder perforation in the absence of trauma include acute cholecystitis, infection or malignancy. It is thought that the fundus is the most likely place for a perforation to occur.⁷

In a study of 46 patients with gallbladder perforation by Derici *et al*,¹ advanced age, male gender, temperature over 38°C, raised white cell count and presence of cardiovascular comorbidity were found to be risk factors that predisposed patients to

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gallbladder perforation, and therefore Derici concluded that these patients should be carefully investigated to avoid missing this diagnosis. Our patient had many of these risk factors including male gender, advanced age and raised white cell count.

Other factors mentioned in the literature that are thought to contribute are use of steroids, anticoagulants and immunosuppressant therapy.⁶ None of these factors were deemed to be present in this case at the time of admission.

Symptoms of perforation are often similar to those of an acute episode of cholecystitis. The only clear difference here was the apparent sudden deterioration. This could initially have been mistaken for severe sepsis, however, bloods, imaging and a high level of clinical suspicion all pointed to the possible diagnosis of gallbladder perforation and possible associated haemorrhage.

Initially, the diagnosis of most gallbladder pathology is via ultrasound. However, the rapid clinical deterioration in this case prompted a CT for added detail and efficiency. There are no pathognomonic signs for gallbladder perforation on CT. A 'hole-sign' finding on ultrasound has been described, but this is thought to be uncommon.⁸ If CT findings suggest cholecystitis associated with pericholecystic fluid collection, high-attenuation intraluminal density, gallbladder wall defect, streaky omentum, gallbladder fossa haematoma, intrahepatic haemorrhage or the presence of extraluminal gallstones, all of these should raise suspicion of gallbladder perforation with possible haemorrhage.^{6,9}

Ultimately, in this case, the diagnosis was made at surgery. However, suspicious findings were already present on the emergency CT.

Many cases of acute cholecystitis can be managed conservatively in hospital with intravenous antibiotics, followed by an elective cholecystectomy, either during the acute admission or on a subsequent planned admission.

In some cases, conservative management of gallbladder perforation may be the only option due to the premorbid state of the patient.¹⁰ However, in this case, given the patient's satisfactory premorbid state and the acute nature of his deterioration, emergency laparotomy was the most reasonable method of intervention and, as such, led to a successful outcome.

Rarely is a cholecystectomy performed as an absolute emergency. This case highlights a significant and potentially fatal complication from cholecystitis and the need to always consider a cause other than sepsis to account for a patient's acute clinical

deterioration. A delay in diagnosis or surgery in such a case could have resulted in significant morbidity or mortality.

Learning points

- ▶ A diagnosis of gallbladder perforation should be considered when a patient with presumed cholecystitis deteriorates or behaves atypically.
- ▶ Hounsfield units can assist in the differentiation of fluids on CT scanning.
- ▶ If the deterioration is particularly acute, the possibility of haemorrhage should be excluded.

Competing interests None declared.

Patient consent Obtained.

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