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Haemobilia causing cholangitis in a patient on dual anti-platelet treatment suffering from acute acalculous cholecystitis

Andreas Luhmann*, Anton Buter, Jo-Etienne Abela

Department of Surgery, Royal Alexandra Hospital, Corsebar Road, Paisley, PA2 9PN, UK

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ABSTRACT

INTRODUCTION: Haemobilia is a rare cause of upper gastro-intestinal haemorrhage which can be difficult to diagnose.
PRESENTATION OF CASE: We present the case of a patient who suffered from acute acalculous cholecystitis while on dual anti-platelet therapy with aspirin and clopidogrel. We describe the diagnostic and treatment challenges arising from the patient's complicated past history and the steps leading to the diagnosis of haemobilia causing biliary obstruction and cholangitis. Our patient did not, at any point, manifest anaemia or evidence of haemorrhage.
DISCUSSION: Haemobilia has a varied aetiology. To our knowledge there is no association with dual anti-platelet treatment in the literature to date. Diagnosis is difficult and relies on multiple modalities. In our patient the final diagnosis was only made in the course of open bile duct exploration.
CONCLUSION: In acute biliary obstruction we recommend the consideration of haemobilia in the differential diagnosis, especially in patients with a bleeding tendency.

1. Introduction

Haemobilia is a rare cause of upper gastro-intestinal haemorrhage. The classic presentation is with right upper quadrant abdominal pain, obstructive jaundice and haematemesis or malaena.[1,2] Its diagnosis can be difficult. We describe the case of a patient who developed acute acalculous cholecystitis while on dual anti-platelet treatment. After cholecystectomy he developed cholangitis. Haemobilia was eventually found to be the cause.

2. Case report

A 70-year-old man was admitted with right upper quadrant abdominal pain and fever. On examination he had a tender fullness in this region in keeping with acute cholecystitis. His history was positive for a distal gastrectomy for complicated duodenal peptic ulcer disease some 30 years previously and for recent myocardial infarction treated with primary coronary angioplasty and drug eluting stent three months previously. For this reason he was on a combination of regular aspirin and clopidogrel (prescribed for a period of 6 months). On admission he had raised inflammatory markers, a normal serum amylase and a deranged liver function test profile (with a normal bilirubin). Ultrasonography confirmed acute cholecystitis in the absence of gall stones. MRCP confirmed cholecystitis and identified an ill-defined non-occlusive filling defect in a normal calibre common hepatic duct (Fig. 1). In the absence of gall bladder stones we originally wondered whether this was indeed an artefactual effect. The patient was treated with a seven days' course of intravenous piperacillin–tazobactam 4.5 g TDS intravenously and his condition improved with resolution of his symptoms and pyrexia within 48 h and normalisation of his inflammatory markers and liver function tests (except for a mild persistent elevation of gamma-GT). Therefore, intervention was deferred until his clopidogrel was stopped three months later. As the patient was still complaining of episodic and transient biliary colic and his gamma-GT remained elevated he was offered laparoscopic cholecystectomy with intra-operative cholangiography.

Intravenous gentamicin and metronidazole were administered at induction of general anaesthesia. At laparoscopy the gall bladder and portal triad were found to be obscured in dense post-gastrectomy adhesions and early conversion to the open right sub-costal approach was made. The shrunken acalculous gall bladder was identified using the seeker needle technique and cholangiography through it was performed. Initially, a ghost outline of a filling defect in an otherwise normal common bile duct was obtained but further contrast injection resulted in a satisfactory cholangiogram with no obvious choledocholithiasis and with contrast clearance into the duodenum (Fig. 2). The duct was therefore, not formally explored. A sub-total cholecystectomy was performed with 3/0 PDS sutures applied to the gall bladder neck well clear of what was perceived to be the line of the common hepatic duct.

The patient's initial recovery was uneventful, but at 72 h he developed upper abdominal pain, jaundice (serum bilirubin of 150 μmol/L) and pyrexia. Abdominal CT was suggestive of a filling defect in the distal CBD (Fig. 3), there was no evidence of active bleeding. An endoscopic retrograde cholangiogram (ERC)

* Corresponding author. Tel.: +44 7905347445.
E-mail address: andreasluhmann@hotmail.com (A. Luhmann).

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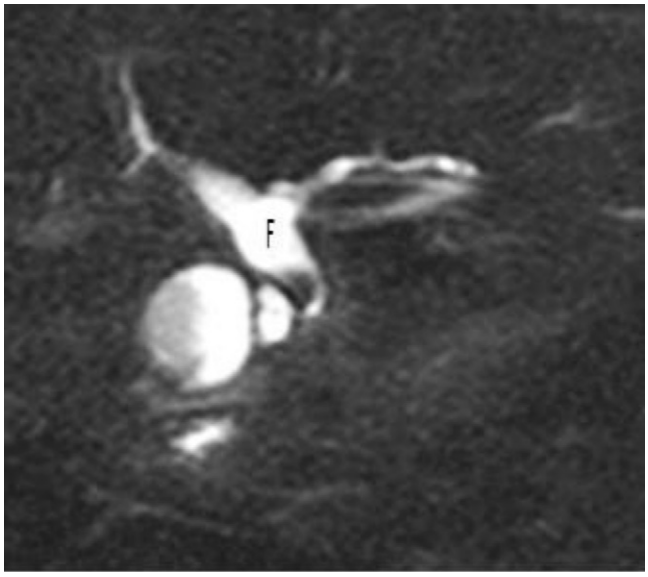

Fig. 1. MRCP view. F, filling defect.

was attempted but not surprisingly this was unsuccessful due to instrument looping due to the post-gastrectomy state.

An urgent re-laparotomy for bile duct exploration was performed. The gall bladder fossa was uncomplicated. The normal calibre common hepatic duct was again identified with the seeker needle technique and exposed with considerable difficulty. A spiral choledochotomy was performed. To our surprise we found an acute on chronic blood clot filling the whole length of the CBD and causing obstruction (Fig. 4). Following its removal and choledochoscopy, the choledochotomy was approximated over a T-tube. The patient recovered without further incident and the T-tube was removed 4 weeks later after a satisfactory tube cholangiogram was obtained. On follow up the patient remains well. It is important to note that the patient's haemoglobin level was within the normal range throughout his management and that no time did he manifest with haematemesis, malaena or indeed haemorrhage through the T-tube.

3. Discussion

Haemobilia was first described by Glisson and subsequently by Morgagni.[3] Haemorrhage may originate in the liver, extrahepatic bile ducts, gallbladder and the pancreas (52.7%, 22.5%, 23.1% and 1.7%, respectively). Iatrogenic trauma is the aetiology in 50–70% of reported cases and it may follow percutaneous needle biopsy of the liver, percutaneous biliary drainage, ERC+/− sphincterotomy, biliary stent placement, choledochoscopy, transjugular intrahepatic porto-systemic shunt, ablation of liver tumours and cholecystectomy. Other causes include liver trauma, rupture of hepatic neoplasms into the bile ducts, ruptured hepatic cyst,

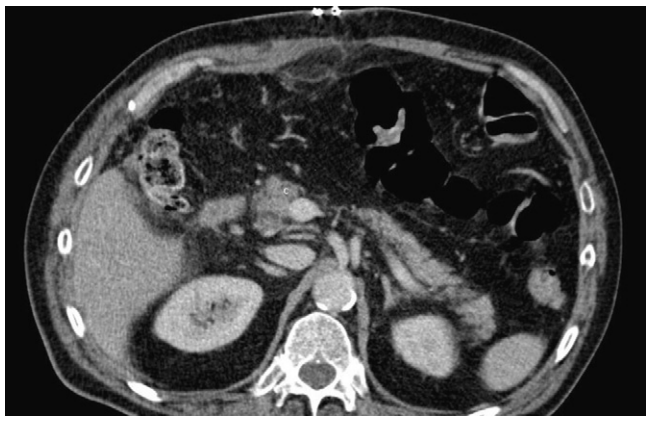

Fig. 3. Post-operative CT showing solid elements in common bile duct (C).

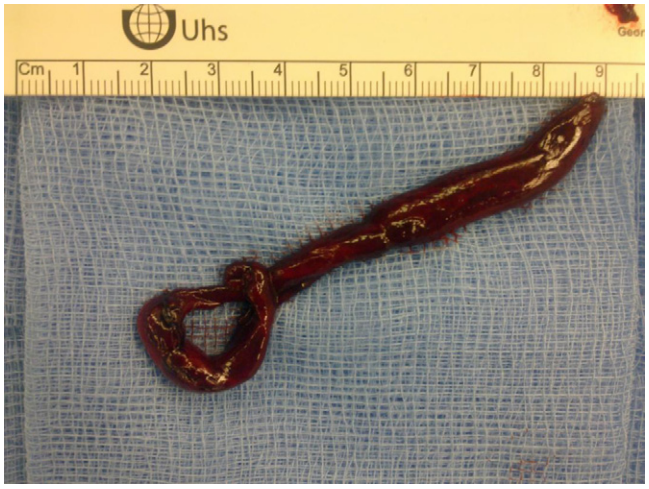

Fig. 4. Operative specimen – blood clot forming cast of common hepatic and bile ducts.

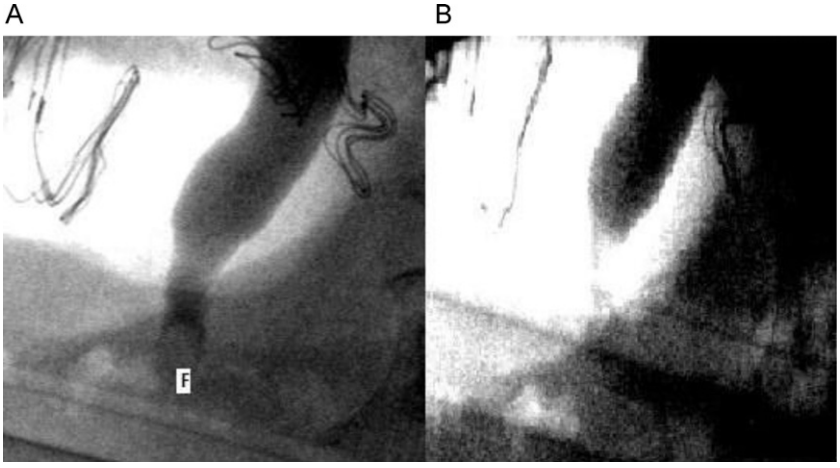

Fig. 2. Intra-operative cholangiography; (A) slow contrast injection with a ghost outline of a filling defect (F) which disappeared on further contrast injection in (B).

infectious/parasitic causes such as liver abscesses or ascaris infestation and sequelae of acute cholecystitis like cystic artery or right hepatic artery aneurysms. Conversely, haemobilia has been reported to cause cholecystitis, cholangitis and pancreatitis. Haemobilia in the setting of bleeding diathesis secondary to anti-coagulation has been described.[4,5] To our knowledge there is no association with dual anti-platelet treatment to date.

Haemobilia may be classified into mild and major cases. The former tends to be relatively benign presentations which settle with conservative measures within 48 h. Major haemobilia, however, results in haemodynamic instability and may become rapidly life-threatening in the absence of adequate resuscitations.[1,2,5] Management depends on the likely aetiology. Upper gastrointestinal endoscopy confirms bleeding in only 10% of cases and these are usually complications after endoscopic sphincterotomy. Despite a range of emerging imaging modalities, CT angiography is probably the gold standard investigation in all other major scenarios.[4,5] Whereas, conservative management has been reported to be effective in 43% of cases, selective angiographic embolisation has become the standard of care in major haemorrhage, wherein its success rate is between 80 and 100%.[6,7] Laparotomy has in effect become the treatment of last resort.[8]

Our patient suffered from mild haemobilia while on both aspirin and clopidogrel and thus formed a clot in his extrahepatic biliary tree. It is impossible to determine whether this was the cause or indeed the effect of the observed acalculous cholecystitis. In retrospect bile duct exploration should have been performed during the first operation, given the suspicions raised by the MRCP and initial intra-operative cholangiogram. At the very least and with the benefit of hindsight, we should probably have requested a repeat MRCP just before the cholecystectomy. This case highlights the danger of overfilling the biliary tree with contrast during operative cholangiography and thus obscuring subtle filling defects. Possibly, the dense post-gastrectomy adhesions may have predisposed us to be conservative in order to avoid injury to the portal triad. This case is even more unique as ERCP was not possible in view of his previous gastrectomy. Therefore, re-laparotomy was inevitable.

4. Conclusion

In acute biliary obstruction we recommend the consideration of haemobilia in the differential diagnosis, especially in patients with a bleeding tendency when gall stones are not confirmed and imaging is equivocal.

Conflict of interest

None.

Funding

None.

Ethical approval

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

Author contributions

The first author collected clinical data and images with the help of the 3rd author. He also did the bulk of the writing. All 3 authors were involved in the clinical care of the patient and both the 2nd and 3rd author provided invaluable input reviewing the paper.

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