CASE REPORT

Hepatic artery aneurysm: an unusual cause of upper gastrointestinal bleed

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SUMMARY

An 86-year-old woman presented to hospital with melaena. This was her third presentation with the same symptom. There was no obvious source of bleeding on her oesophagogastroduodenoscopy; however, it did show a previously clipped Dieulafoy lesion. CT angiography showed an aneurysm arising from the hepatic artery. Selective coeliac artery angiogram showed aneurysmal dilatation of the distal part of the coeliac trunk and confirmed the presence of the common hepatic artery aneurysm. The aneurysm was coiled by the interventional radiologist. Final angiogram showed good flow through the hepatic artery with obliteration of the inferior patch. The procedure was uncomplicated and the patient was discharged shortly afterwards.

BACKGROUND

Hepatic artery aneurysms (HAAs) are rare but clinically important cause of upper gastrointestinal (GI) bleed. The true incidence of HAA is not known but thought to be around 0.002% of the population.¹ They are often asymptomatic and detected incidentally; however, they may present with abdominal pain, a mass, hypovolaemia secondary to rupture or GI bleeding with a normal oesophagogastroduodenoscopy (OGD).² Causes of HAA include atherosclerosis, inflammation (such as vasculitis) or iatrogenic (for instance during angiography). They may also be idiopathic.² Delay in diagnosis may lead to life-threatening haemorrhage.³ HAA can be diagnosed by ultrasound, CT scan or CT angiography.³ In the event of a patient presenting with an upper GI bleed with normal endoscopy findings, HAA should be considered and investigated.

CASE PRESENTATION

The patient was an 86-year-old woman with multiple comorbidities, including atrial fibrillation for which she was taking apixaban. There was no history of surgical or biliary procedures. This was her third hospital admission with a bleed. Her first two OGDs had not shown any obvious source of bleeding. Her third OGD had shown what appeared to be a Dieulafoy lesion which had been clipped ([figure 1](#)). She presented to the hospital with melaena. Clinical examination revealed no significant findings. Her abdomen was soft and non-tender, and there were no palpable masses. Digital rectal examination revealed melaena. In the emergency department she was hypotensive but not tachycardic.

INVESTIGATIONS

Investigations in the emergency department revealed a haemoglobin level of 6.6 g/dL. An erect chest X-ray showed no subdiaphragmatic air. OGD showed a clip in D1 from the past Dieulafoy lesion; however, there was no luminal blood or stigmata of recent bleed. A CT angiogram was subsequently performed. This showed a 2.2×1.5 cm aneurysm ([figure 2](#)) in the common hepatic artery with a pouch from its inferior surface very close to the endoscopic clips in the duodenum, suggesting previous bleed from this point.

DIFFERENTIAL DIAGNOSIS

1. Dieulafoy lesion: this was originally thought to have been the source of bleeding; however, the bleeding was noted to persist even after the lesion had been clipped. Therefore an alternative diagnosis was sought.
2. Haemobilia: this would have been diagnosed during the CT angiogram.
3. Small intestinal telangiectasia: this would have been detected during OGD.

TREATMENT

A CT angiogram was performed with a view to embolising the aneurysm. The common hepatic artery and the aneurysm were cannulated using a microcatheter system ([figure 3](#)). Four 3 mm microcoils were deployed and packed in the inferior patch of the aneurysm. Final angiogram showed good flow through the hepatic artery with obliteration of the inferior patch ([figure 4](#)).

Figure 1 Endoscopic findings suggestive of Dieulafoy lesion.

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Figure 2 Aneurysm of the common hepatic artery (arrow).

The patient was transfused a total of four units of red blood cells during her admission. Her apixaban was withheld while there was ongoing bleeding; however, it was subsequently restarted following the procedure.

OUTCOME AND FOLLOW-UP

The patient made a good recovery following her embolisation procedure and was discharged from the hospital 3 days later. A follow-up CT scan performed 2 months later showed multiple coils obliterating the inferior recess of the common hepatic artery aneurysm. Normal enhancement was seen within the right and left hepatic artery. Mild aneurysmal dilatation of the proximal part of the celiac artery and the splenic artery was noted. The

Figure 3 Pre-embolisation selective angiogram of the common hepatic artery. (a) Aneurysm in the distal part of the common hepatic artery. (b) This inferior patch represents a defect in the aneurysm wall which causes haematemesis. (c) Endoscopic clip in the second part of the duodenum. (d) Another small aneurysm in the proximal part of the common hepatic artery.

Figure 4 Postembolisation selective angiogram of the common hepatic artery. (a) Several coils placed in the inferior defect of the aneurysm wall to prevent further bleeding. (b) Preservation of flow through the aneurysm into the right and left common hepatic arteries.

case was discussed in multidisciplinary meetings both locally and at a tertiary centre (Royal Free Hospital). The patient was felt to be at moderate risk for rebleeding. Different treatment options considered included surgery and embolisation of the hepatic artery. Overall, it was felt that surgery was not an option as the patient was likely to be at high intraoperative risk due to her comorbidities. Following a discussion with the patient, a decision was made to continue supportive measures while haemodynamically monitoring the patient rather than attempt further embolisation which would risk massive hepatic necrosis and subsequent deterioration. The patient was counselled regarding risk of further bleeding and was advised to return to the hospital immediately in the event of a bleed.

She had a further bleed 4 months later, which was managed conservatively. Following this episode, her apixaban has been withheld indefinitely because her risk of rebleeding is thought to outweigh her risk of stroke. She has now been bleed free for 2 months.

DISCUSSION

HAAs are rare with an incidence of approximately 0.002%.¹ They are usually asymptomatic and diagnosed either incidentally or on autopsy.¹⁴ However, they have been reported to present as non-specific abdominal pain, jaundice, abdominal masses or infrequently as GI haemorrhages.^{1 2 5} Causes include atherosclerosis, inflammation or iatrogenic.² The condition is associated with high morbidity and mortality unless it is treated promptly.¹³ The main diagnostic methods are by ultrasound or CT angiography; however, there are reported cases of diagnosis during surgery and also on plain radiography.¹³ Percutaneous embolisation is an increasingly popular choice of treatment for bleeding secondary to HAA.⁶⁷ This is supported by a study demonstrating good short and long-term outcomes following embolisation of visceral artery aneurysms.⁸ In cases where embolisation is unavailable or has

failed, surgical methods are a treatment option. Surgical treatments include excision of the aneurysm with saphenous vein grafting or excision and ligation of the aneurysm.¹

This case highlights successful management of a haemorrhage secondary to HAA in an elderly patient with multiple comorbidities. In the first instance, the bleeding was thought to be secondary to a Dieulafoy lesion, which is a much commoner cause of upper GI bleeding (approximately 1.5% of cases of acute upper GI bleeding).⁹ An alternative diagnosis was sought after the patient continued to bleed despite the lesion having been clipped. The patient has remained well 2 months after stopping her anticoagulation, and she is under follow-up with her local gastroenterology team.

Learning points

- ▶ Hepatic artery aneurysms are a rare but important cause of upper gastrointestinal (GI) bleeding.
- ▶ This diagnosis should be suspected in the event of a GI bleed with normal endoscopic findings.
- ▶ Diagnosis is primarily made using the modality of CT angiogram.
- ▶ Embolisation in the interventional radiology department is an effective treatment for this condition.

Contributors MF conducted the literature review, planned and wrote the case report. RP, ZA and DG treated the patient. RP and DG edited the case report. ZA provided the images included in the report.

Competing interests None declared.

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