CASE REPORT

Ruptured aneurysm of an aberrant left hepatic artery

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SUMMARY

Although rare, the high mortality associated with visceral artery aneurysms highlights the importance of their prompt diagnosis and treatment. This report details the acute presentation, diagnosis and management of a 51-year-old man with a non-traumatic ruptured aneurysm of an aberrant left hepatic artery. This is just the second case of a rupture of this aberrant artery reported in the literature, and the only one to be treated using endovascular techniques. An up-to-date review of the investigation and surgical/endovascular management of visceral aneurysms in relation to this case is included.

BACKGROUND

Hepatic artery aneurysms are rare and ruptures have a high mortality. This case is unusual in that the rupture was spontaneous and also because it involved aberrant anatomy. The expeditious diagnosis using ultrasound and treatment using endovascular means was very successful and seems to be the ideal model for this type of case.

CASE PRESENTATION

A 51-year-old Caucasian male presented with collapse, vomiting and right upper quadrant pain radiating to the shoulder tip. The patient's medical history included hypertension and there was no history of abdominal trauma or recent surgery. He was an ex-smoker with a 40-pack-year history, and drank 40 units of alcohol/week. The patient had a body mass index of 34.

On examination, he was afebrile but pale, sweaty and cold. He had a sinus tachycardia of 140 bpm with a blood pressure of 90/40 mm Hg. This responded well to an infusion of 500 mL of gelifusin and 1 L of Hartmann's solution, subsequently rising to 110/80 mm Hg. His abdomen was distended, diffusely tender and there was guarding in the right upper quadrant. No abdominal aortic aneurysms were palpable and pulses were felt in all limbs.

INVESTIGATIONS

Initial blood tests revealed haemoglobin 13.3 g/dL (13.8–17.2 mg/L), white cell count $11.6 \times 10^9/L$ ($3.8\text{--}11.0 \times 10^9/L$), haematocrit 0.31 (0.39–0.54) and a C reactive protein of 189 mg/L (<5 mg/L). Other blood tests, including liver function tests, renal function tests, clotting and electrolytes were within the normal range. Arterial blood gas analysis demonstrated a compensated metabolic acidosis with a raised lactate of 3 mmol/L (0.3–2.3 mmol/L), pH 7.45 (7.34–7.45), base excess (BE)

–9 mmol/L (–2 to +2 mmol/L), HCO_3^- 14.4 mmol/L (22–26 mmol/L). An initial focussed assessment with sonography for trauma (FAST) scan revealed fluid in the hepatorenal recess in addition to the right and left iliac fossae (unfortunately these images were not saved). Immediate contrast-enhanced CT of the abdomen and pelvis demonstrated a dilated left hepatic artery with a bleeding point close to the porta hepatis (figures 1 and 2). It also revealed evidence of heterogeneous hepatic enhancement (which may be suggestive of hepatic hypoperfusion) and free fluid within the peritoneal cavity.

DIFFERENTIAL DIAGNOSIS

The differential diagnosis includes a hepatic artery pseudoaneurysm. A pseudoaneurysm is typically secondary to trauma or local inflammation adjacent to the vessel. Pseudoaneurysms are seen on colour Doppler imaging of duplex ultrasound as discrete vascular lesions with bidirectional colour flow, the so-called 'ying yang' sign. This pathology may coexist with evidence of acute or chronic pancreatitis (such as pseudocyst formation), liver abscess or evidence of trauma (such as free intraperitoneal fluid). On CT, a focal vascular lesion within the hepatic arterial bed may be identified which enhances in the arterial phase on contrast-enhanced CT. There may again be evidence elsewhere of concurrent/causative pathology (as described in the

Figure 1 Axial contrast-enhanced CT in the arterial phase of the upper abdomen demonstrates a focally dilated left hepatic artery (red arrow) with some mild adjacent fat stranding of the mesentery. Perisplenic and perihepatic free fluid is also seen (Sensation 16 Siemens, Erlangen, 332 mA, 120 KV, slice thickness 1–5 mm, 150 mL Imeron 300).

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Figure 2 Coronal reformatted image of the contrast-enhanced CT scan of the abdomen in the arterial phase demonstrates an aberrant left hepatic artery arising from the left gastric artery (red arrow). Note how the artery appears focally dilated and prominent. Right subdiaphragmatic, subhepatic and intraperitoneal free fluid is also present (Sensation 16 Siemens, Erlangen, 332 mA, 120 KV, slice thickness 1–5 mm, 150 mL Imeron 300).

ultrasonographic findings). Arterial aneurysms may also be secondary to vasculitic processes such as the antineutrophil cytoplasmic antibody-associated vasculitides, or those associated with rheumatological disease. These are usually immune-mediated processes that start in the vasa vasorum. In this case a vasculitic screen was negative.

TREATMENT

A 6 Fr right common femoral artery approach was undertaken for coil embolisation. Selective angiogram of the coeliac axis confirmed the presence of an aneurysmal left hepatic artery originating from the left gastric artery (figure 3). A 2.7 Fr microcatheter (Progreat, Treumo) and 5 mm microcoils (Tornado, Cook) were used to embolise the left hepatic artery (coils were placed successfully distal and then proximal to the aneurysm)

Figure 3 Selective digital subtraction angiography of the coeliac axis confirms the presence of an aneurysmal left hepatic artery (red arrow) originating from the left gastric artery. A poorly defined lateral margin with a blush of contrast in this region is indicative of rupture. A 2.7 Fr microcatheter (Progreat, Treumo) and 5 mm microcoils (Tornado, Cook) are used to embolise the left hepatic and left gastric arteries.

Figure 4 Portovenous phase axial contrast-enhanced CT of the abdomen showed satisfactory embolisation with exclusion of the aberrant left hepatic artery aneurysm. Liver segments II and III demonstrate features consistent with ischaemic hepatitis (Sensation 16 Siemens, Erlangen, 332 mA, 120 KV, slice thickness 1–5 mm, 150 mL Imeron 300) arteries.

and left gastric artery. The time between FAST scanning and emergency coil embolisation of the left gastric artery was 2.5 h. This short interval allowed for resuscitation (four units of packed red cells were transfused during this interval) and CT imaging to determine the appropriateness of the angiographic intervention.

OUTCOME AND FOLLOW-UP

One day postprocedure there was derangement of the liver function tests: alkaline phosphatase 171 IU/L (32–110 IU/L), aminotransferase 1897 IU/L (7–56 IU/L), bilirubin 11 μ mol/L (2–12 μ mol/L). A triple-phase contrast CT of the abdomen showed satisfactory embolisation with exclusion of the aberrant left hepatic artery aneurysm with low attenuation in liver segments II and III, unchanged from the preembolisation imaging and consistent with ischaemic hepatitis (figure 4).

The patient continued to improve clinically, with some amelioration of the liver function tests. He was discharged from the hospital on day 7. On outpatient review at 3 months, the patient remained well with restoration to normal of his liver function tests.

DISCUSSION

An aneurysm is defined as the localised dilation of an artery including all the layers of its wall to more than 150% its normal diameter. Hepatic artery aneurysms are more commonly seen in males (in a ratio of 3 : 2) in the fifth to sixth decade of life and have an incidence of 0.002–0.4%.¹ They occur most frequently in the common or proper hepatic (40%) and the right hepatic artery branches (50%), and less commonly in the left artery branches.¹ Our patient demonstrated a variation of normal vascular anatomy: the left hepatic artery arises from the left gastric artery in 15% of cases. A search of the MEDLINE and EMBASE databases revealed just one other case of a ruptured artery in this location, and we describe the only case to be treated using endovascular techniques.^{2 3}

The aetiology of hepatic arterial aneurysms includes atherosclerosis (most commonly), mediointimal degeneration, trauma,

infection and iatrogenic (following biliary procedures or cholecystectomy). The most likely aetiology in our patient is atherosclerosis due to his risk factors (hypertension, smoking and alcohol usage) and the exclusion of other causes. Patients may present as an emergency or via an incidental finding on routine imaging. Although small hepatic artery aneurysms are often asymptomatic, the natural history is for progressive enlargement with increasing risk of rupture and death. The high mortality associated with the spontaneous rupture of hepatic artery aneurysms, 21%, means prompt evaluation and management are essential.¹ Typical symptoms after rupture include back, chest and abdominal pain. Rupture into the hepatobiliary tree or gastrointestinal system is more common than into the peritoneal cavity, as in our case.

Blood tests may reveal evidence of bleeding manifested by low-haemoglobin concentration and falling haematocrit. FAST scan evaluation may be the initial imaging modality and reveals the presence of free intraperitoneal fluid. This case highlights the benefits of FAST scanning in revealing the pathology early and thereby expediting further investigations and treatment. Note must be made, however, that FAST is usually performed by non-radiologists and is very operator dependent. A negative scan does not indicate a lack of free peritoneal fluid, and it is certainly true that this patient could be stabilised enough for a CT scan to be undertaken rapidly.

Hepatic artery aneurysms may sometimes be seen on plain abdominal films as a thin rim of calcification in the right upper quadrant. Formal ultrasound may identify a discrete fusiform or round hypoechoic lesion in the right upper quadrant. Pulsed wave and colour Doppler imaging of duplex ultrasound can help to differentiate this from other lesions such as fluid collections or cystic tumours. Ultrasound is useful for the follow-up of patients. Vessel wall calcification and hypodense thrombotic deposits within the vessel lumen may be seen on the unenhanced CT.⁴ Contrast-enhanced abdominal CT usually provides the initial diagnosis (although magnetic resonance angiography may also be used). The widened lumen can be clearly visualised and measured after intravenous contrast, and attenuation of 70 HU in the surrounding tissues indicates fresh bleeding.

CT angiography is the ideal modality for diagnosis and additionally identifies other aneurysms (there are commonly more than one) and associated pathology (such as arterioportal fistulas, feeding vessels) and elucidates variant anatomy.⁴

The treatment of ruptured hepatic artery aneurysms may be surgical or endovascular. Open surgical treatment includes ligation and aneurysm exclusion, excision or revascularisation. Ligation of the hepatic artery for aneurysm was first performed successfully by Kehr⁵ and this has traditionally been the treatment for haemodynamically unstable patients. This case, however, illustrates the opportunity of the endovascular approach. Complications of open surgery include hepatic infarction, bleeding (which may be massive), wound infection, paralytic ileus and damage to adjacent structures.⁶ Selective endovascular coil embolisation or covered stenting are both viable alternatives to surgery and may be used in the elective or emergency setting.^{7, 8} The restoration of liver function tests in our patient to baseline demonstrates the safety of left gastric artery embolisation. Endovascular techniques avoid a general anaesthetic and the morbidity associated with open surgery; it also makes the procedure possible in those who would be unfit for surgery due to comorbidities. Hospital stay is shorter.⁹ Access is usually transarterial but a percutaneous approach is also possible for patients with unfavourable anatomy. The technical success of the endovascular procedure is very high (98% in

one series).¹⁰ Other than failure and hepatic infarction, the complications include arterial dissection, embolism, pseudoaneurysms and contrast-induced nephropathy. Recanalisation is not uncommon, however, necessitating repeat embolisation. Usage of microcoils for embolisation has a lower incidence of recanalisation than Gelfoam.⁹ Stent migration has also been observed.¹¹ In a retrospective study of 59 patients by Sachdev *et al*,¹² the endovascular technique was associated with a shorter length of stay (2.4 days compared with 6.6 days), but with no differences in mortality, complications or reintervention. Interestingly, of the 11% who experienced primary treatment failure secondary embolisation was successful in all patients.

The common hepatic artery may be ligated with impunity provided there is a patent gastroduodenal artery or pancreaticoduodenal artery providing collateral blood supply to the liver.⁶ Although there is evidence of intrahepatic anastomoses between the left and right hepatic arteries, revascularisation of the arteries distal to the common hepatic is generally preferable to reduce the risk of hepatic ischaemia. Inlay grafts are used for open surgery and covered stents for the endovascular approach. Polytetrafluoroethylene may be preferable to vein grafts as it is less likely to become compressed by anatomical structures, and is less prone to eventual dilatation. Covered stents are usually reserved for arteries 6 mm or larger in diameter because of the risk of thrombosis in smaller vessels.⁹

Learning points

- ▶ Focussed assessment with sonography for trauma (FAST) scanning is useful for expediting the investigation and treatment of seriously ill patients even without a history of trauma; it must be remembered, however, that FAST is operator dependent and negative scans do not provide evidence of lack of pathology.
- ▶ Endovascular coil embolisation is a valid treatment option for even ruptured hepatic artery aneurysms; however, the interventional radiologist must be aware of variant anatomy before embolisation.
- ▶ If detected and treated early, the prognosis for hepatic artery aneurysm rupture is favourable.

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