CASE REPORT
Endovascular management of a large hepatic artery aneurysm related to type B aortic dissection

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SUMMARY
Management of visceral artery aneurysms can be challenging: there is limited evidence to determine size thresholds for intervention and it is often technically difficult to exclude the aneurysms while preserving visceral perfusion.

We present the case of a 68-year-old male with a rapidly enlarging hepatic artery aneurysm related to type B aortic dissection extending into the coeliac axis, which presented unique difficulties due to its morphology and filling via the false lumen. Endovascular treatment involved stent-graft placement from the coeliac axis into the splenic artery with the intention of excluding the coeliac supply to the common hepatic artery. Despite early stent-graft occlusion, the aneurysm was successfully excluded and adequate hepatic and splenic perfusion was maintained. The patient made a good recovery.

INTRODUCTION
True visceral artery aneurysms (VAAs) are rare, with an estimated incidence of 0.1–0.01%. The splenic and hepatic arteries are the most commonly affected vessels. Causative factors include atherosclerosis, pregnancy, infection, and connective tissue disorders. Multiple strategies for endovascular treatment have been described, which balance the need to exclude the aneurysm while also preserving visceral perfusion. We present a case of hepatic artery aneurysm with an unusual aetiology and specific anatomical challenges which was managed endovascularly with a good clinical outcome.

CLINICAL PRESENTATION
A 68-year-old male presented to his General Practitioner (GP) with severe, transient abdominal and back pain. He had a history of radical prostatectomy, hypertension, and gout. His regular medications were amlodipine, simvastatin, and allopurinol.

Initial blood tests showed mild normocytic anaemia (haemoglobin 126 g l\(^{-1}\)), normal renal function, and deranged liver enzymes (ALT 247 U l\(^{-1}\), ALP 441 U l\(^{-1}\)) with normal bilirubin (8 micromol/l). Abdominal ultrasound was reported as showing a distended gallbladder with echogenic contents, but no other significant finding. Thoracolumbar spine MRI showed no spinal pathology but a dissection flap was noted in the aorta.

On review in Vascular Surgery outpatient clinic 2 weeks after symptom onset, clinical examination revealed a blood pressure of 152/98 mmHg in the left arm, 165/98 in the right arm, a soft and non-tender abdomen, and normal lower limb pulses bilaterally.

IMAGING FINDINGS
A CT aortogram (arterial phase, Figure 1) showed a Stanford type B aortic dissection with an intimal defect 3 cm distal to the left subclavian artery origin and a dissection flap extending to the aortic bifurcation. The descending thoracic aorta was aneurysmal at 45 mm, but the other aortic segments were non-aneurysmal. The superior mesenteric artery (SMA), right renal artery, and right common iliac artery were supplied by the true lumen; left renal artery was supplied by the false lumen; coeliac axis and left common iliac artery had joint supply from true and false lumens. There was a tubular structure at the porta hepatis measuring up to 37 mm diameter, with similar density to the aortic false lumen. This was thought to correspond to the echogenic structure reported as a distended gallbladder on ultrasound. A hepatic artery aneurysm was suspected.

The CT was repeated 1 week later (Figure 2), this time with arterial and portal venous phases. This confirmed a fusiform aneurysm involving the common hepatic, proper hepatic, and left and right hepatic arteries. This had grown to 41 mm in maximal diameter. An intimal flap was demonstrated at
the origin of the common hepatic artery, suggesting aneurysm perfusion via the false lumen. The aneurysm was compressing the extrahepatic bile ducts, with mild upstream intrahepatic biliary dilatation, and the main portal vein. The gastroduodenal artery (GDA) was not clearly demonstrated, possibly reflecting compression or effacement by the aneurysm.

Repeat blood tests showed worsening liver enzymes (ALT 324 U l⁻¹, ALP 1125 U l⁻¹) and newly elevated bilirubin (152 micromol/l).

TREATMENT
After multidisciplinary discussion involving specialists in interventional radiology, hepatobiliary surgery and vascular surgery, it was determined that urgent intervention was warranted due to the rapid enlargement of the aneurysm (4 mm in 7 days) and the deteriorating blood tests. It was decided to place a stent–graft from the coeliac axis into the splenic artery, with the intention of excluding coeliac supply to the common hepatic artery while maintaining splenic perfusion. (Figure 3)
Via right common femoral artery access under local anaesthetic, the left gastric artery was embolised with coils to reduce the risk of endoleak. Stable access to the splenic artery could not be achieved via the femoral sheath; therefore left brachial artery access was gained and a long 6-French sheath was advanced into the proximal splenic artery. A 7 × 37 mm balloon-mounted stent-graft (BeGraft, Bentley, Germany) was deployed from the coeliac artery to the proximal splenic artery, covering the common hepatic artery origin. The segment of stent-graft within the coeliac artery was overdilated with a 9 × 20 mm balloon. A final angiogram confirmed occlusion of the hepatic artery origin, with no filling of the aneurysm from the coeliac artery. Flow into the splenic artery through the stent was sluggish – this was thought to be due to splenic artery spasm downstream of the stent-graft. The original procedural plan was to perform angiography of the GDA via the pancreaticoduodenal arcade from the SMA to assess for retrograde aneurysm perfusion following stent-graft deployment and collateral supply to the liver. However, the patient became restless and it was decided to stop the procedure and reassess the aneurysm and hepatic arterial supply with early post-operative CT instead. It was decided not to institute antiplatelet or anticoagulant therapy following the procedure, as the risk of haemorrhage from the aneurysm was thought to outweigh any benefits of optimising stent-graft patency.

In the early post-operative period, the patient experienced mild abdominal discomfort and a low-grade fever. CT imaging (Figure 4) on day 3 showed that the stent-graft was occluded, but the majority of the spleen was perfused via collaterals. No contrast filling of the aneurysm was demonstrated, confirming successful exclusion of the common hepatic artery. There was infarction in hepatic segment IV, but multiple arterial branches in both hepatic lobes were filling via collaterals and the majority of the liver appeared to be enhancing normally. After a short course...
DISCUSSION

While it is generally accepted that all visceral artery pseudoaneurysms require intervention, regardless of size or location, there is some uncertainty around thresholds for intervention for true VAAs. The most widely accepted practice is to treat true aneurysms with a diameter of 2 cm or more, in the absence of symptoms. While in certain situations it can be difficult to distinguish between true and pseudo-aneurysms on imaging alone, in our patient, we considered that the imaging findings were more characteristic of a true aneurysm, given that the aneurysm extended beyond the proper hepatic artery bifurcation into the left and right hepatic arteries, and there was no surrounding haematoma. In any case, given the rapid enlargement to 4.1 cm and associated biliary obstruction and portal vein compression, urgent intervention was clearly mandated. Given the increased perioperative mortality risk associated with surgical repair of VAAs, endovascular repair is preferable in the majority of cases. In our patient, surgery was felt to involve unacceptable risks, especially given the anatomy of the aneurysm. The proposed surgical approach under consideration would potentially have required ligation of the common hepatic artery and the left and right hepatic arteries, with a high risk of associated ischaemic complications in addition to the risks of major laparotomy and retroperitoneal dissection.

A variety of different endovascular strategies have been described involving coils, vascular plugs, liquid embolic agents, and stent–grafts. Usually treatment decisions relate to aneurysm anatomy with the requirement to exclude the aneurysm being balanced with the need to maintain visceral perfusion. In general, assuming a stent–graft cannot be placed through an entire aneurysm, or across the neck of a pseudo-aneurysm, it is recommended in non-terminal vascular territories (such as the hepatic circulation) to occlude both upstream and downstream of the aneurysm to prevent both antegrade and retrograde perfusion. In our patient, it was not possible to place a stent–graft through the whole aneurysm given its length and extension beyond the bifurcation of the proper hepatic artery. The normal rules of upstream and downstream occlusion, which would have mandated the embolisation of the common hepatic artery at its origin as well as the left and right hepatic arteries, did not apply in this case given that the aneurysm was perfused via the false lumen. Our judgement was that occlusion of the common hepatic artery origin alone would be sufficient as there would be a low risk of retrograde perfusion of the false lumen aneurysm via normal collateral pathways (such as the GDA), especially given the SMA origin was supplied exclusively by the true lumen. Furthermore, embolisation of the left and right hepatic arteries downstream of the aneurysm would present an unacceptable risk of hepatic ischaemia, especially given the presence of portal vein compression. Our treatment strategy of stent–graft placement from coeliac axis to splenic artery was intended to prevent antegrade perfusion of the common hepatic artery false lumen (and therefore exclude the aneurysm), while allowing true-lumen collateralisation from the GDA to preserve hepatic perfusion. Thankfully, the strategy was successful: upstream occlusion alone proved to be sufficient to treat the aneurysm while adequate hepatic arterial supply was preserved to prevent clinically significant hepatic ischaemia; and despite early stent–graft occlusion, collateral supply also prevented major splenic infarction.

A final learning point from this case is the need to assess the visceral arteries carefully when reporting CT imaging, especially in the setting of aortic dissection. Although VAA related to aortic dissection is rare, there is a recognised association. It is important not to miss opportunities to diagnose VAA, given the potential to intervene prophylactically to prevent aneurysm rupture.

LEARNING POINTS

Although rare, there is an association between aortic dissection and VAA. Therefore it is important to assess carefully the visceral arteries on imaging performed for aortic dissection. When technically feasible, endovascular therapy is generally preferred to open surgery for VAA. Any intervention must exclude the aneurysm while maintaining adequate perfusion to the major viscera. Treatment options include coil embolisation, vascular plugs and stent–graft placement, depending on aneurysm anatomy.

NORMAL RANGES

- Alanine aminotransferase (ALT): 0–40 units/l
- Alkaline phosphatase (ALP): 30–120 units/l
- Bilirubin: 0–21 micromol/l
- Haemoglobin: 135–170 g dl⁻¹
REFERENCES

1. Juntermanns B, Bernheim J, Karaindros K, Walensi M, Hoffmann JN. Visceral artery aneurysms. Gefasschirurgie 2018; 23(Suppl 1): 19–22. doi: https://doi.org/10.1007/s00772-018-0384-x

2. Hemp JH, Sabri SS. Endovascular management of visceral arterial aneurysms. Tech Vasc Interv Radiol 2015; 18: 14–23. doi: https://doi.org/10.1053/j.tvir.2014.12.003

3. Cavalcante RN, Couto VAP, da Fonseca AV, de Miranda RB, Costa AJV, Correa JA. Endovascular treatment of a giant hepatic artery aneurysm with Amplatzer vascular plug. J Vasc Surg 2014; 60: 500–2. doi: https://doi.org/10.1016/j.jvs.2013.06.077

4. Loffroy R, Favelier S, Pottecher P, Genson P-Y, Estivalet L, Gehin S, et al. Endovascular management of visceral artery aneurysms: when to watch, when to intervene? World J Radiol 2015; 7: 143–8. doi: https://doi.org/10.4329/wjr.v7.i7.143

5. Belli A-M, Markose G, Morgan R. The role of interventional radiology in the management of abdominal visceral artery aneurysms. Cardiovasc Intervent Radiol 2012; 35: 234–43. doi: https://doi.org/10.1007/s00270-011-0201-3

6. Huang Y-K, Hsieh H-C, Tsai F-C, Chang S-H, Lu M-S, Ko P-J. Visceral artery aneurysm: risk factor analysis and therapeutic opinion. European Journal of Vascular and Endovascular Surgery 2007; 33: 293–301. doi: https://doi.org/10.1016/j.ejvs.2006.09.016

7. Masuda K, Takenaga S, Morikawa K, Ashida H, Ojiri H. A case of giant common hepatic artery aneurysm successfully treated by transcatheter arterial embolization with isolation technique via pancreatic-duodenal arcade. Radiol Case Rep 2019; 14: 195–9. doi: https://doi.org/10.1016/j.radcr.2018.10.029

8. Nosher JL, Chung J, Brevetti LS, Graham AM, Siegel RL. Visceral and renal artery aneurysms: a pictorial essay on endovascular therapy. Radiographics 2006; 26: 1687–704 Nov-Dec. doi: https://doi.org/10.1148/rg.266055732