

Management of a common and proper hepatic artery aneurysm

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ABSTRACT

First-line management of hepatic artery aneurysms is via an endovascular approach. However, unfavorable anatomy may preclude this. We present a patient with an aneurysm involving most of the common hepatic artery and the entire proper hepatic artery including the emergence of the right and left hepatic artery and the gastroduodenal artery. The endovascular approach was not feasible due to unfavorable anatomy. The patient was successfully treated with an open bifurcated Dacron graft. (*J Vasc Surg Cases and Innovative Techniques* 2021;7:283-5.)

Keywords: Hepatic artery aneurysm; Asymptomatic aneurysms; Dacron graft; Aberrant anatomy

Hepatic artery aneurysms (HAA) account for 20% of splanchnic artery aneurysms.¹⁻³ HAA is associated with a 25% rupture rate with 70%-100% mortality.^{1,4} First-line management is through an endovascular approach; however, unfavorable anatomy may preclude this.⁴ Despite the lethal consequences of a spontaneous HAA rupture, there remains a paucity of treatment outcomes as well as confounding management recommendations due to the rarity of this pathology. We discuss the management of an asymptomatic HAA with anatomy not amendable to endovascular repair. The patient provided written informed consent for this publication.

CASE REPORT

A 69-year-old man with a medical history of hypertension, hyperlipidemia, and tobacco use was referred to us with an asymptomatic 2.9 cm HAA. The HAA was incidentally identified on a surveillance computed tomography (CT) with an intravenous contrast scan for a hepatic cyst. The only prior imaging was 10 years prior, in which the HAA was not present. A review of the CT suggested that endovascular repair would not be feasible given the variable sizes of the inflow and outflow branches (Fig 1). The portal vein was patent and the superior mesenteric artery was patent without evidence of stenosis. Preoperative ultrasound of the lower extremities

demonstrated a greater saphenous vein diameter of 5 mm.

The patient underwent elective repair via a midline laparotomy. His American Society of Anesthesiologists score was 3. The morphology of the aneurysm involved the entire proper hepatic artery including the emergence of the right and left hepatic artery and the gastroduodenal artery (Fig 2). The aneurysm was opened longitudinally, and the gastroduodenal artery was ligated. The luminal diameter of the proximal common hepatic artery was approximately 10 mm, with the right and left hepatic arteries at approximately 5 mm. The senior author felt that it was not appropriate to use a 5 mm autologous conduit on a 10 mm inflow in an end-to-end anastomosis. A 12 mm × 6 mm bifurcated Dacron graft was anastomosed to the common hepatic artery with each graft limb positioned in the right and left hepatic artery (Fig 3). The superior mesenteric artery was pulsatile. Total skin to skin case time was 6 hours and 47 minutes, with 35 minutes of liver ischemia time. Estimated blood loss was 150 cc.

Postoperatively, the patient was monitored in the surgical intensive care unit. He was started on 81 mg of aspirin. Liver function labs peaked on postoperative day (POD) 1 (aspartate aminotransferase: 1028 IUnit/L, alanine aminotransferase: 792 IUnit/L) and subsequently down-trended until normal. His postoperative course was complicated by an ileus that resolved on POD 4, and then he was discharged on POD 5. A 6-month postoperative CT demonstrated graft patency. The graft surveillance plan is for yearly duplex ultrasound scans.

DISCUSSION

Albeit rare clinical entities, hepatic artery aneurysms represent a potentially lethal vascular pathology. Clinical presentation ranges from incidental detection of asymptomatic aneurysms on ultrasound or CT scans to hemodynamic instability secondary to rupture. Symptomatic patients may have varying clinical presentations as

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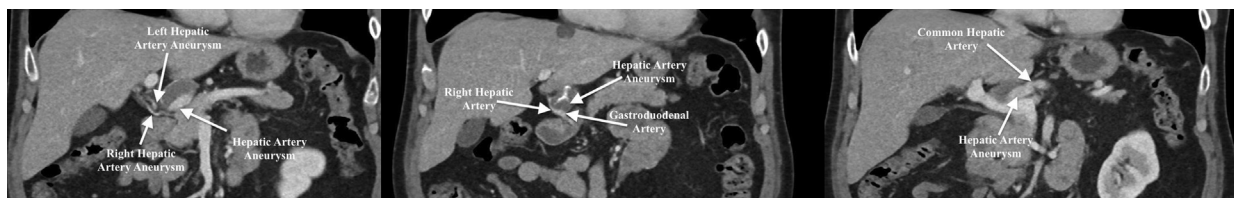


Fig 1. Preoperative computed tomography (CT) angiogram demonstrating the hepatic artery aneurysm (HAA) with its inflow and outflow branches labeled.

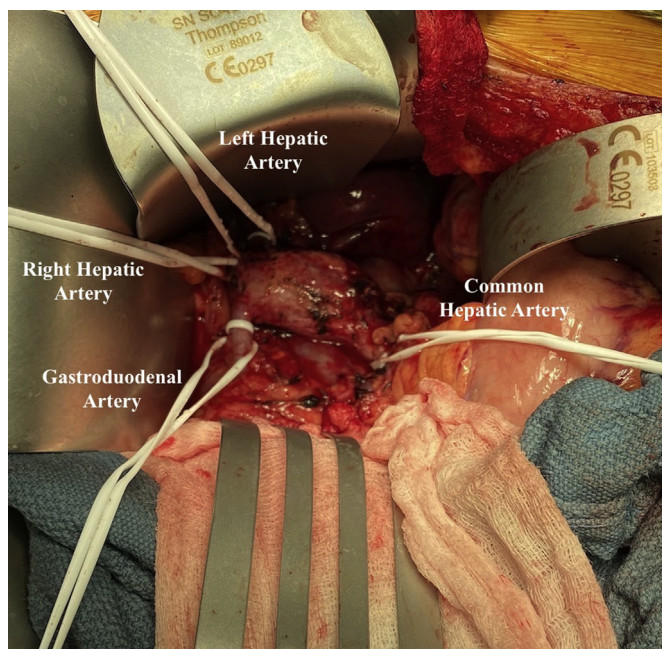


Fig 2. Hepatic artery aneurysm (HAA) with arteries labeled adjacent to each vessel loop.

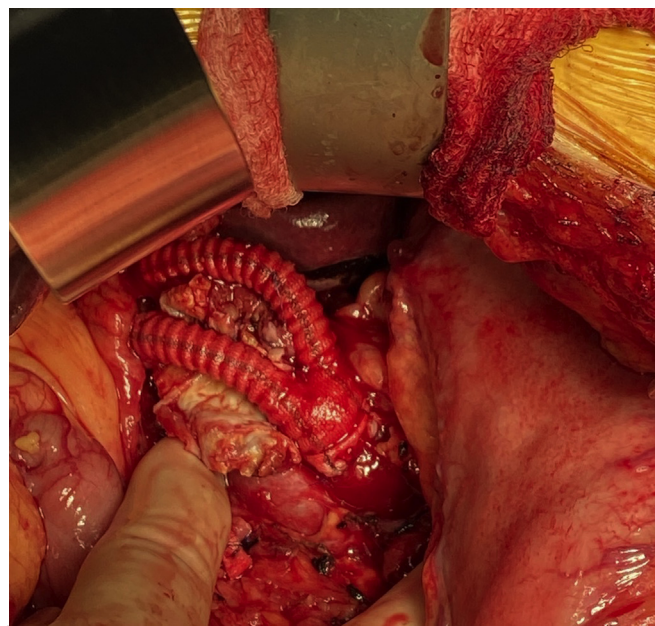


Fig 3. Dacron graft anastomosed to the common hepatic with each limb anastomosed to the right and left hepatic artery.

extrahepatic aneurysms rupture into the peritoneum and intrahepatic aneurysms tend to rupture into the biliary tract resulting in approximately 25% of symptomatic patients presenting with Quinke's triad of pain, jaundice, and hemobilia.^{5,6} Hepatic artery pseudoaneurysms most commonly arise from blunt abdominal trauma or iatrogenic trauma from biliary tree interventions.⁷ True HAA can arise from atherosclerosis, fibromuscular dysplasia, portal hypertension, hypertension, vasculitis, polyarteritis nodosa, and systemic lupus erythematosus.^{1,3,8} HAA of nonatherosclerotic origins are at a higher risk for rupture and therefore warrant more immediate repair.³

Previously, it was recommended that on the diagnosis of HAA regardless of the presence of symptoms, immediate surgical or endovascular repair was warranted.⁴ However, the current Society for Vascular Surgeons guidelines recommend intervention in the following: (1) all hepatic

artery pseudoaneurysms; (2) all symptomatic HAA regardless of size; (3) asymptomatic patients without significant comorbidity in true HAA >2 cm, if the aneurysms enlarge by 0.5 cm/y or if a patient with significant comorbidities has an aneurysm greater than 5 cm.⁴ Society for Vascular Surgeons guidelines also recommend a one-time screening CT angiogram of the head, neck, and chest for nonatherosclerotic causes of HAA. This was not performed in our patient as the etiology was due to atherosclerosis.

A variety of treatment options for HAA exist including ligation, excision and repair, arterial grafting and reconstruction, hepatic resections, and endovascular approaches.³ Although endovascular repair is first-line treatment for HAA, the location of the aneurysm, the presence of collateral flow, and the clinical status of the patient have important implications on the ideal therapy.³ Anatomical variation can limit the feasibility of

endovascular repair, as seen in our patient, given the luminal diameter differences between the proximal common hepatic artery compared to the right and left hepatic arteries. Also, each hepatic artery originated individually from the aneurysm sac; therefore, it was felt that endovascular therapy would have left an unacceptably high risk of failure. In addition, the patient was a good surgical candidate based on comorbidities and risk factors. Although outcomes are limited to small studies and individual case reports, the 30-day mortality rate for both urgent and elective open or endovascular procedures ranges between 6% and 14%.⁹ Postintervention complications occur in 24%⁹ to 29%¹ of patients and include hemorrhage, graft thrombosis, common bile duct stricture, duodenal perforation, pancreatitis, enterocutaneous fistula, and infection. Despite the risk of thrombosis, 5-year graft patency rates have been reported as 79%,³ 86%,¹ and 100%,¹⁰ demonstrating promising results for HAA repair with synthetic or venous grafting.

CONCLUSION

We present a successful operative management of an HAA not amendable to endovascular repair to demonstrate to the reader a feasible option when encountering a similar patient. This case illustrates the importance of creating an individualized surgical approach to HAA repair based on patient anatomy and surgical risk.

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