

# Management of a large hepatic artery aneurysm

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We present a rare case of a giant hepatic artery aneurysm in a 61-year-old man that was successfully treated by aneurysmectomy with prosthesis bypass grafting. Because the gastroduodenal artery was occluded, an adequate collateral circulation was not ensured after simple ligation, so a direct arterial flow to the liver was restored to avoid the risk of significant liver or biliary tract ischemia. A computed tomography scan at 1 month showed occlusion of the bypass. The patient remained asymptomatic, despite the supposed lack of adequate collateral circulation. The unpredictable blood supply to the liver is discussed. (*J Vasc Surg Cases* 2015;1:97-9.)

Hepatic artery aneurysms (HAAs) may remain asymptomatic until life-threatening complications occur. Although HAAs were predominantly of mycotic origin in past reports, mycotic HAAs are seldom seen today because of earlier antibiotic treatment of infections.

Although atherosclerosis seems to be the most common factor, arterial fibrodysplasia, vasculitis, polyarteritis nodosa, and systemic lupus erythematosus have been related to HAAs. Less frequently, HAAs have been associated with Takayasu arteritis, Kawasaki disease, von Recklinghausen neurofibromatosis, and Wegener granulomatosis. Long-term oral amphetamine use has also been associated with HAA formation. Congenital causes of HAAs include Marfan syndrome, Ehlers-Danlos syndrome and hereditary hemorrhagic telangiectasia.<sup>1,2</sup>

The treatment strategy should be determined individually, based on the anatomy of the aneurysm, the wide variability of hepatic blood supply, and of course, the risks of intervention. The patient consented to publication of this report.

## CASE REPORT

A 61-year-old man was transferred from another hospital to our department with a diagnosis of a giant HAA. The patient complained of vague but continuous right upper quadrant abdominal pain that had arisen in the last 2 weeks. Risk factors included smoking and hypertension. He had no history of abdominal trauma or surgery, and laboratory results were within normal reference

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ranges. The physical examination revealed a pulsatile mass in the right flank and iliac fossa.

A computed tomography (CT) examination showed a tortuous celiac anatomy and a 14-cm × 13-cm fusiform aneurysm involving the common and proper hepatic artery and compressing the surrounding structures. The CT also showed occlusion of the gastroduodenal artery (GDA; Fig 1).

Open surgery was preferred to an endovascular approach because of the size of the aneurysm and the lack of a proximal and distal neck and was performed in a conventional operating room. A median laparotomy was done (Fig 2). The size of the lesion did not allow conventional supraceliac clamping. Therefore, via a transfemoral access, a balloon (NuMED, Hopkinton, NY) was introduced through a 12F × 30-cm sheath (W. L. Gore and Associates, Flagstaff, Ariz) and inflated at the level of the celiac trunk ostium to achieve a temporary proximal aortic endoclamping. After the aneurysm incision, we achieved proximal control with a Foley catheter proximally and with a Fogarty catheter distally. We considered that the occlusion of the GDA would not have guaranteed the adequacy of collateral pathways after exclusion of the aneurysm by ligation.

After systemic heparinization, an aneurysmectomy, followed by revascularization, was performed. Considering the inadequacy of the saphenous veins, an 8-mm Dacron (DuPont, Wilmington, Del) bypass graft was used.

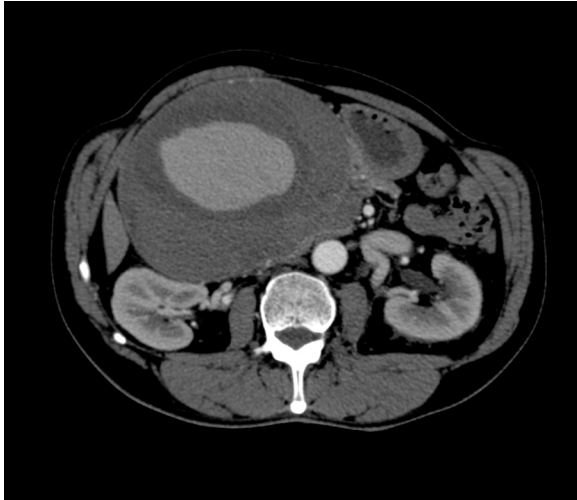
The patient's postoperative course was uneventful, and he was discharged home on postoperative day 6. The patient received clopidogrel (75 mg/d) and aspirin (100 mg/d).

A postoperative duplex scan on the day of discharge did not reveal liver ischemia and showed patency of the bypass graft, without kinking or anastomotic flaws.

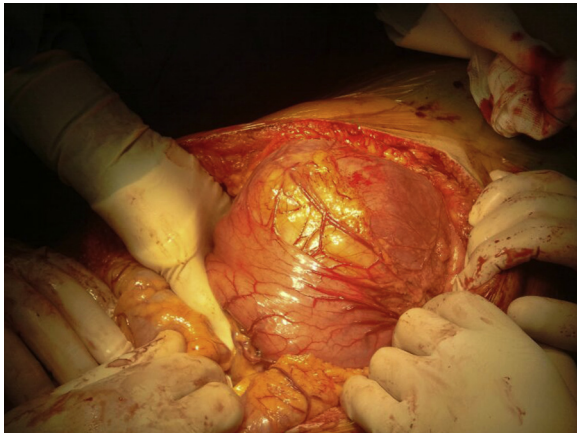
A follow-up abdominal CT at 1 month showed occlusion of the bypass, probably caused by discontinuation of dual-antiplatelet therapy by the patient on his own initiative. Occlusion occurred without complaints.

## DISCUSSION

Visceral artery aneurysms are rare, comprising 0.1% to 2% of all arterial aneurysms. HAAs account for ~20% to 40% of these aneurysms<sup>3-5</sup> and are the second most frequent visceral aneurysm after splenic artery aneurysms.<sup>2-4</sup> Approximately 80% of HAAs are extrahepatic and 63% of these involve the common hepatic artery (CHA).<sup>6,7</sup>



**Fig 1.** Preoperative computed tomography (CT) scan demonstrates the large hepatic artery aneurysm (HAA).



**Fig 2.** Hepatic artery aneurysm (HAA): Intraoperative finding.

The disease is often asymptomatic, so patients usually present late to medical attention. Most HAAs are discovered incidentally in an asymptomatic state during diagnostic imaging procedures performed for other conditions.

When symptomatic (10%-15%),<sup>8</sup> the clinical manifestations are nonspecific and variable, depending mostly upon the size of the aneurysm. The typical findings at presentation include epigastric or right upper quadrant pain, followed by gastrointestinal hemorrhage and obstructive jaundice. All of these symptoms (Quinke triad) are rarely reported at the same time. Like all aneurysms, the natural evolution is to increase in size. The risk of rupture is highly variable (20%-80%), with mortality rates ranging from 9.1% to 40%.<sup>2,5</sup> Giant HAAs are extremely rare, with very few cases reported.<sup>2,3,9-12</sup>

Although there is no consensus about size criteria for surgical treatment, most authors consider surgery for patients with asymptomatic aneurysms >2 cm in diameter

or rapidly growing, pseudoaneurysms, symptomatic aneurysms regardless of size,<sup>2,3,6</sup> and for women of child-bearing age.<sup>6</sup>

Currently, indications regarding the type of intervention are still controversial. The choice of treatment should be tailored to the patient, depending on clinical presentation, aneurysm type, and anatomic location, as well as associated risk factors. Treatment options include open or endovascular techniques. Open surgery for extra-HAAs is mainly by complete excision or exclusion by ligation, with direct revascularization in selected cases. Endovascular techniques, including coil embolization, stent grafting, and multilayer stenting,<sup>2-5</sup> offer a minimally invasive approach but are restricted to lesions with favorable morphologic characteristics.

A review by Berceci et al<sup>1</sup> reported that open surgical options are mainly determined by aneurysm anatomic location. Lesions of the CHA usually do not need vascular reconstruction. A bypass with autogenous conduit has been suggested in good-risk patients, especially when the GDA is small.

Pulli et al<sup>13</sup> recently reaffirmed that evaluation of the blood supply provided by the GDA is crucial to avoid liver ischemia. In cases of aneurysms of the CHA, the presence of an anastomotic arcade between the celiac axis and the superior mesenteric arteries via the GDA and right gastric arteries can usually protect the liver from ischemia after ligation of the proximal CHA. In these cases, aneurysms may be simply excluded by ligation, once assured that there is a patent GDA or pancreaticoduodenal artery.<sup>5,12</sup> Otherwise, when collateral circulation is insufficient after the interruption of hepatic arterial flow, a graft replacement should be applied to maintain a direct antegrade flow.<sup>4,6</sup> This is also true for aneurysms distal to the origin of the GDA, which require, as a rule, direct arterial reconstruction to minimize the risk of liver ischemia.

The GDA occlusion in our patient led us to restore a direct arterial flow to the liver. This approach seemed more appropriate than simple ligation to avoid the risk of significant liver or biliary tract ischemia. The asymptomatic occlusion of the bypass, despite the occlusion of the GDA and the expected lack of a collateral blood supply, may highlight the unpredictable response of liver parenchyma to hepatic dearterialization.

Most patients in this situation develop a compensatory increase of the portal vein flow and of the flow through normal collateral arteries.<sup>12</sup> As reported in a study of 200 dissections, there are at least 26 possible routes of a collateral blood supply to the liver other than the typical celiac blood supply made by a single common hepatic trunk.<sup>14,15</sup>

Similarly, several reports of successful ligation of HAAs distal to the GDA suggest that restoration of arterial blood flow may not always be necessary.<sup>5,6,12</sup> Collateral circulation via the right inferior phrenic and subcostal arteries was the most common. Other possible routes included the GDA and its branches, superior and inferior pancreaticoduodenal, and at the splenic hilum, the left gastric, and splenic arteries.<sup>16</sup>

## CONCLUSIONS

The blood supply to the liver is unpredictable. Proximal and distal ligation of the hepatic artery provides a low-risk solution; however, evaluation of the blood supply provided by the GDA on the basis of the color changes of the liver parenchyma is crucial. Our patient, being relatively young, presumably had patent collateral vessels and a resilient vascular system, so collateral arterial perfusion to the liver may have been guaranteed by some of these collateral pathways.

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