Ruptured aneurysm of replaced left hepatic artery

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Hepatic artery aneurysm is an uncommon and potentially fatal form of vascular disease. We report the case of a 53-year-old man with an isolated, nontraumatic rupture of an aneurysm of a replaced left hepatic artery originating from the left gastric artery. This case is unusual because the ruptured aneurysm involved an hepatic artery with a rare vascular pattern. (J Vasc Surg Cases 2015;1:105-9.)

Aneurysms involving the hepatic artery are a rare but life-threatening vascular disease. Hepatic artery aneurysms (HAAs) represent ∼20% of all visceral artery aneurysms, with an estimated incidence of 0.002%, and are most commonly seen in the sixth decade of life with a male-to-female ratio of 3:2.[1]

The rupture of this type of aneurysm presents as a clinical emergency and is associated with a mortality rate as high as 20% if not recognized and treated immediately.[2] The treatment of HAAs in recent years has undergone a profound change toward endovascular procedures; indeed, coil embolization and stent graft endovascular techniques are used more frequently.[3] However, open surgery is considered the preferred treatment in most cases and especially in acute complications of these aneurysms.[4] Written informed consent was obtained from the patient for the publication of this case report.

CASE REPORT

A 53-year-old man was admitted to our hospital for hydronephrosis due to urolithiasis. He subsequently underwent the placement of a ureteral stent on the left side. The patient's medical history appeared unremarkable apart from hypertension and hypercholesterolemia. About 72 hours after the urologic procedure, the patient started to complain of a violent pain in the epigastric region associated with nausea. On physical examination, he appeared afebrile but pale, sweaty, and cold; he had a sinus tachycardia (120 beats/min) with hypotension (90/70 mm Hg). He also had a distended abdomen and moderate tenderness in the

epigastrium and left hypochondrium. Blood workup revealed anemia, thrombopenia, and a slight increase in the international normalized ratio, activated partial thromboplastin time, and alanine aminotransferase. Arterial blood gas analysis demonstrated a decompensated metabolic acidosis (Supplementary Table I). Rapid resuscitation with intravenous administration of crystalloid solutions allowed a stabilization of his hemodynamic status, which allowed us to perform a detailed imaging study.

An initial focussed assessment with sonography for trauma scan revealed free fluid in all recesses of the peritoneal cavity. An immediate contrast-enhanced triple-phase computed tomography (CT) scan of the abdomen and pelvis showed a dilated left hepatic artery with a bleeding point (aneurysm of 20 × 18 mm in diameter) that appeared to originate from the left gastric artery and was associated with a large intraperitoneal effusion. The scan also revealed evidence of heterogeneous hepatic enhancement in the left lateral liver segments (left lobe), which could suggest hepatic hypoperfusion (Figs 1 and 2).

Laparotomy revealed a massive hemoperitoneum of ∼1500 mL. At the level of the hepatogastric ligament, a ruptured aneurysm of a hepatic arterial branch was recognized. There was a fresh blood clot in the artery. Because of a diffused disruption of the vascular wall comprising the whole extrahepatic portion of the left hepatic artery, we did not consider performing revascularization of the artery. We therefore promptly clamped the aneurysm and isolated the proximal stump up to the origin of the left hepatic artery from the left gastric artery. Because no clinical sign of hepatic ischemia was observed after the cross-clamping, we performed a complete aneurysmectomy (Fig 3).

The patient spent the first 48 hours in the intensive care unit, which was the time required to correct the acidosis that occurred during surgery and to stabilize the hemodynamic conditions. He was then transferred to the surgical ward. Although we did not observe any major surgical complications, his hospital stay was prolonged due to the onset of a noninfectious fever. The patient was discharged from hospital 15 days after the operation in good clinical condition, asymptomatic, and with liver function tests within normal reference ranges.

DISCUSSION

HAA is a rare vascular disease but the second most common site for aneurysm formation within the splanchnic circulation after splenic artery aneurysms.[5] Typically this involves the extrahepatic branches of the hepatic artery in 80% of cases, whereas intrahepatic cases are related to

Fig 1. Contrast-enhanced computed tomography (CT) in the arterial-late phase demonstrates an aneurysm of a replaced left hepatic artery.
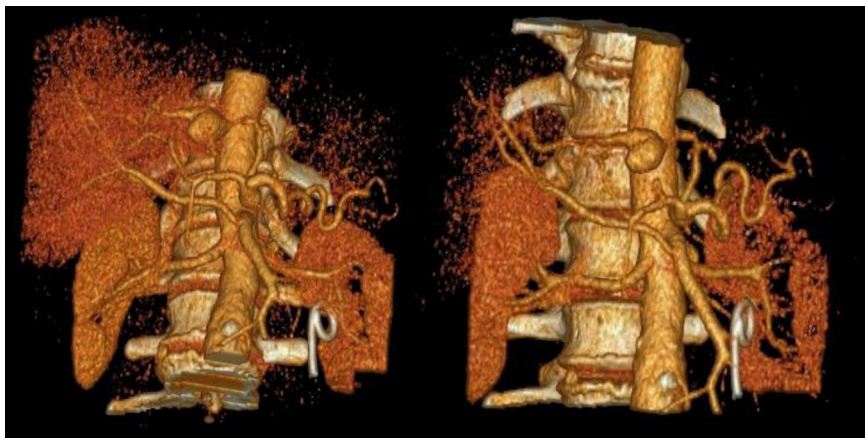

Fig 2. Abdominal axial and three-dimensional reconstruction image contrast computed tomography (CT) scan shows a replaced left hepatic artery arising from the left gastric artery and a replaced right hepatic artery arising from the superior mesenteric artery (type VIII according to the Michels classification). The left ureteral stent is also visible.

interventional procedures or to abdominal trauma. Specifically, these aneurysms occur mainly in the proper or common hepatic artery (40%) or in the right branch of the hepatic artery (50%) and very rarely in the left hepatic artery. Our patient had a rare anatomic variation in the vascularity of the liver because he had a replaced left hepatic artery from the left gastric artery and a replaced right hepatic artery arising from the superior mesenteric artery (type VIII according to Michels' classification[6]; Supplementary Table II). A systematic search through the medical literature showed that this is only the third reported case of rupture of the hepatic artery in this location.[7,8]

The etiology of HAAs includes atherosclerosis, medial degeneration, infection (mycotic), and traumatic and iatrogenic inflammation; other rare causes include polyarteritis nodosa, Wegener granulomatosis, Takayasu arteritis, and Kawasaki disease. However, due to a significant increase in interventional procedures on the liver, cases of hepatic artery pseudoaneurysms have increased progressively over time,

now accounting for 50% of the aneurysms observed.[9] The most likely etiology in our patient was atherosclerosis due to his risk factors of hypertension and hypercholesterolemia.

Although most small HAAs are usually asymptomatic, the natural history of this disease involves a progressive enlargement with an increased risk of rupture, which is estimated at ∼20% to 30%. The high mortality associated with the spontaneous rupture of the HAA (30%) means prompt evaluation and management are essential.[10] Rupture into the hepatobiliary tree or gastrointestinal system is a little more common than rupture into the peritoneal cavity. Most symptomatic patients present with one or more of the Quincke classic triad of hemobilia (jaundice, biliary colic, and gastrointestinal hemorrhage).[11] An HAA that is sufficiently calcified may be found incidentally on plain radiography of the abdomen. Ultrasound imaging can identify a fusiform or round hypoechoic lesion, and Doppler imaging can help to differentiate an HAA from other lesions such as fluid collection or cystic lesions.
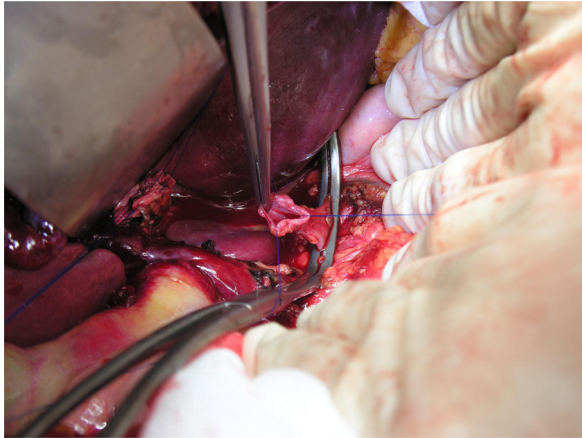
Fig 3. Intraoperative view after placement of a clamp at the junction of left hepatic and left gastric arteries.

Ultrasound imaging has a low sensitivity in detecting small aneurysms because identification can be compromised by overlying gas and obesity.[12]

Currently, CT angiography is the ideal tool for diagnosis because it also identifies other aneurysms and associated diseases and clarifies the anatomical variations of the hepatic artery. Selective catheter angiography is considered a useful option for diagnosis as well as for planning and performing therapeutic interventions. However, multislice CT angiography has been demonstrated to be the most powerful tool in the noninvasive evaluation of vascular disorders.[13,14]

All authors have advocated treatment in patients with symptomatic HAA or in those who are identified to be at high risk of rupture. According to published literature, all symptomatic HAA, multiple aneurysms or any asymptomatic aneurysm of nonatherosclerotic origin >2 cm in diameter should be treated.[15]

The treatment of a ruptured aneurysm of the hepatic artery may be surgical or endovascular; however, the best type of treatment is still debated[3,16] (Supplementary Table III). Open surgical treatment includes simple ligation and exclusion of the aneurysm, excision, or revascularization. All of these can be taken into consideration when the patient is in shock or in unstable condition and if the aneurysm is extrahepatic.[17] Ligation of the hepatic artery was first performed by Kehr,[18] and this treatment has traditionally been proposed for unstable patients. Owing to the perfusion provided by the gastroduodenal and right gastric arteries, lesions of the common hepatic artery can usually be ligated or resected without reconstruction, whereas surgical treatment of more distal extrahepatic lesions that involve the proper hepatic or proximal right or left arteries requires direct arterial reconstruction to reduce the risk of hepatic ischemia.[19] Although the right and left hepatic arteries as well as the replaced hepatic arteries are considered end-arteries in cadavers, that intrahepatic anastomoses exist in vivo has long been known.[20] In our patient, the replaced left hepatic artery ligation did not create any anatomical or functional consequence to the liver because the left lobe was revascularized by the branch artery from the fourth segment.

Selective treatments with endovascular coil embolization or covered stent are both available resources and alternatives to surgery and can be used with good success in elective cases and also in emergency settings. The main advantages of the endovascular techniques are avoidance of a general anesthetic, reduction of the morbidity associated with open surgery, and also a shorter hospital stay. Complications after endovascular repair include incomplete aneurysmal exclusion, distal thromboembolic events, coil migration, and end-organ infarction. That endovascular techniques have been associated with up 25% of morbidity related to transient postembolization syndrome or an incompletely excluded aneurysm has also been reported.[21]

CONCLUSIONS

Aneurysms involving the hepatic artery are an uncommon and potentially fatal form of vascular disease. A high index of suspicion and prompt diagnosis are key elements for the effective management of patients with HAAs. Although HAAs can be treated with good success through endovascular techniques, surgical open repair remains the gold standard for satisfactory long-term results, especially in cases of ruptured aneurysms or in those involving the hepatic hilum.

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Supplementary Table I. Preoperative results of patient's blood analysis

Parameter	Value	Normal range
Hemoglobin, g/dL	8.6	13-17
White cell count, $\times 10^3$	9.9	4-10
Platelet count, $\times 10^3$	47	140-400
International normalized ratio	1.9	0.8-1.2
APTT	1.31	0.8-1.2
Alanine aminotransferase, IU/L	95	<41
pH	7.24	7.34-7.45
HCO_3, mmol/L	17.3	22-26
Base excess, mmol/L	−9.9	−2 to 2

APTT, Activated partial thromboplastin time.

Supplementary Table II. Classification of hepatic artery anatomy

Type	Description	Percent
Classification of hepatic arterial types according to Michels' classification[1]		(N = 200)
1	Normal anatomy	55
2	Replaced LHA from LGA	10
5	Accessory LHA	8
3	Replaced RHA from SMA	11
6	Accessory LHA	7
4	Replaced RHA + LHA	1
7	Accessory RHA + LHA	1
8	Replaced RHA + accessory LHA or replaced LHA + accessory RHA	2
9	CHA from SMA	2.5
10	CHA from LGA	0.5
Classification of hepatic arterial types according to Hiatt's classification[2]		(N = 1000)
1	Normal	75.7
2	Replaced or accessory LHA	9.7
3	Replaced or accessory RHA	10.6
4	Replaced or accessory RHA + replaced or accessory LHA	2.3
5	CHA from SMA	1.5
	CHA from Aorta	0.2

CHA, Common hepatic artery; *LGA,* left gastric artery; *LHA,* left hepatic artery; *RHA,* right hepatic artery; *SMA,* superior mesenteric artery.

Supplementary Table III. Literature review of patients with hepatic artery aneurysms and pseudoaneurysms treated with endovascular or surgical techniques (small series of <5 patients and patients with nonoperative management were not considered for this review)

First author	Patients, No.	Surgery	Endovascular	Mean size, cm	Technical complication	30-day mortality	Overall complications (%)
Abbas[3]	14	2 excisions 12 reconstructions	2	5.7	3 graft occlusions	1 in OG	2 pneumonia, 1 duodenal leak, 1 urinary infection, 1 Paralytic ileus, 1 catheter sepsis
Chiesa[4]	7	4 reconstructions 1 excision	2 Coil embolizations	—	1 failure of embolization	None	1 liver abscess
Fankhauser[5]	56	—	Coil embolization	—	2	None	8 liver infarcts
Huang[6]	14	2 excisions	12 coil embolizations	—	—	2 in EG	2 rebleeding in EG
Kasirajan[7]	11	none	11 coil embolizations	2	3 failures of embolization	None	None
Lumdsen[8]	21	5 excisions 5 reconstructions 1 hepatic resection		4.3 ± 3.6	1 migration of embolic material, 1 failure of embolization	1 EG 1 OG	2 hepatic abscesses
Marone[9]	17	11 reconstructions 1 excision	3 coil embolizations 2 stent grafts		1 stent graft occlusion, 1 failure of embolization	None	—
Pulli[10]	7	1 excision 6 excisions 1 reconstruction	—	—	None	None	None
Sachdev[11]	22	3 excisions 5 reconstructions 1 hepatic resection 1 OLT	10 coil embolizations 2 stent grafts	3.28 ± 1.6 EG 4.35 ± 1.98 SG			
Tulsyan[12]	12	—	12 coil embolizations	3.45	None	None	None

EG, Endovascular group; *excision,* aneurysmectomy or simple ligation; *reconstruction,* vascular reconstruction with graft interposing or end-to-end anastomosis; *SG,* surgery group.

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