

Case Report

Splenic Artery Transposition Graft Usage for the Supply of the Right Hepatic Artery: A Case Report

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Hepatic artery aneurysms are responsible for 12% to 20% of all visceral arterial aneurysms. Because most patients are asymptomatic, this disease is generally diagnosed incidentally during radiologic examination. Aneurysm rupture develops in 14% to 80% of cases, depending on the aneurysmatic segment's diameter and location, as well as other etiologic factors. Mortality rates associated with rupture range between 20% and 70%. Thus, early diagnosis and timely initiation of medical interventions are critical to improve survival rates. Here, we present a male patient, age 69 years, with a hepatic artery aneurysm that was detected incidentally. The 3-cm aneurysm was detected on contrastenhanced computed tomography and extended from the common hepatic artery to the hepatic trifurcation. A laparotomy was performed using a right subcostal incision. After dissection of the hepatoduodenal ligament, the common, right, and left hepatic arteries, as well as the gastroduodenal artery, were suspended separately. Then, the aneurysmatic hepatic artery segment was resected, and the gastroduodenal artery stump was ligated. An end-to-end anastomosis was formed between the left and common hepatic arteries, followed by an end-to-end anastomosis formed between the right hepatic artery and splenic artery using a splenic artery transposition graft. Postoperative follow-up examinations showed that both hepatic arterial circulations were good, and no splenic infraction had developed.

Key words: Hepatic artery – Aneurysm – Splenic artery – Transposition graft

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Tepatic artery aneurysm (HAA) is a vascular disease of the splanchnic region, and its associated complications result in severe morbidity and mortality.^{1,2} HAA was first described by Wilson in 1809 based on his observations from a postmortem cadaveric study. Nearly 8 decades later, in 1892, Hale-White described the HAA-associated clinical symptomology known as "Quinke's triad," composed of abdominal pain, obstructive jaundice, and hemobilia; however, this profile is seen in only about one third of patients with HAA. Then, in 1903, Kehr reported the first successful hepatic artery ligation to treat a perforation into the gallbladder, which was followed almost 5 decades later, in 1951, by Paul reporting the first successful revascularization surgery.^{1,3,4} Since then, many alternative treatment methods have been developed for HAA based on our increased understanding of the etiopathogenesis of HAA, in particular its association with atherosclerosis, medial degeneration, fibromuscular dysplasia, polyarteritis nodosa, tuberculosis, trauma, invasive radiologic interventions, transplantation, and certain inflammatory diseases.

Most HAA cases are asymptomatic, and diagnosis is generally made when radiologic examinations are performed to address other indications. Rupture develops in 14% to 80% of cases, depending on the diameter of the aneurysmatic segment, location of aneurysm, and other etiologic factors, and when developed, mortality for rupture ranges between 20% and 70%.^{1-3,5,6} Therefore, despite the lack of a consensus, surgical intervention is recommended for all hepatic artery aneurysms that are symptomatic or >2 cm. The operative approaches, however, may vary according to location, diameter, collateral blood flow, and etiology of the aneurysm.^{1,5,7} Embolization, percutaneous transluminal angioplasty/stenting, or surgical repair is widely applied, whereas another less frequent option is excision with splenic artery transposition graft.^{1–10} Here, we present a case of hepatic artery aneurysm that was successfully repaired with splenic artery transposition graft.

Case Report

A 69-year-old male patient presented to the Department of Cardiology and Internal Medicine, Haydarpasa Numune Education and Research Hospital with complaints of chest and abdominal pain, and dyspepsia. The patient's medical history was unremarkable and did not include any systemic diseases, such as diabetes mellitus or hypertension. Blood chemistry analysis revealed proper functioning of the liver, thyroid, and kidney, as well as complete blood count within the normal range. Echocardiography and electrocardiography findings were also normal. Abdominal ultrasonography revealed an aneurysm in the descending aorta. Subsequent abdominal computed tomography (CT) with contrast enhancement showed the aortic aneurysm beginning at the coeliac trunk and extending to the iliac bifurcation. In addition, the CT scan incidentally revealed an aneurysm in the hepatic artery. This second aneurysm had a diameter of 3 cm and began at the common hepatic artery and extended to the region of the trifurcation formed by the right hepatic artery, left hepatic artery, and gastroduodenal artery.

The high risk of rupture and the high mortality and morbidity associated with hepatic artery aneurysms >2 cm led to the patient being referred to our surgery department for operative management. Following completion of the preoperative and intraoperative preparations, a laparotomy was performed via a right subcostal incision. After the hepatoduodenal ligament was turned, the area was dissected up to the coeliac trunk. As a result of the dissection, the right and left hepatic arteries, the gastroduodenal artery, and the common hepatic artery were able to be suspended with vessel



Fig. 1 Perioperative view of the patient's aneurysm at the common, right, and left hepatic arteries, and the gastroduodenal artery. The aneurysm was at least 3 cm in diameter.

suspenders distinguished by different colors (Fig. 1). After excision of the aneurysmatic segment, the gastroduodenal artery stump was ligated. Then, an end-to-end anastomosis was formed without using any graft material between the left hepatic artery and the common hepatic artery. At this point during the surgery, the operative team briefly discussed the potential anastomosis techniques appropriate for right hepatic artery anastomosis (allogenic or artificial graft use). It was agreed that splenic artery transposition was the most appropriate option. The gastrocolic ligament was opened and the splenic artery was dissected up to the hilus by following along its course on the superior edge of the pancreas. Next, an end-to-end anastomosis was formed between the right hepatic artery and the splenic artery transposition graft (Fig. 2). No ischemia was observed in the spleen, and the organ was preserved. At the end of the first postoperative month, examination by CT angiography revealed patency of both the hepatic artery and the transposed splenic artery (Fig. 3).

Discussion

HAA accounts for 0.01% to 0.2% of all arterial aneurysms and 12% to 20% of all visceral aneurysms involving the splanchnic region.^{1,5,8,9} Among the different forms of splanchnic vascular aneurysms,^{1,2,4,8–10} only splenic artery aneurysms (60%–75%) are more frequent than HAA. Pseudoaneurysms of hepatic arteries in particular have been increasing in recent decades, and the rate of increase



Fig. 2 View of the anastomosis between the right hepatic artery and the transposed splenic artery.



Fig. 3 CT angiography image with contrast enhancement taken at the end of the first postoperative month. Both the left hepatic artery and the splenic artery transposition graft were patent.

has paralleled the increase in interventional biliary procedures, such as biopsies, and nonoperative liver trauma (such as that caused by car accidents).³ The actual incidence of HAA is unknown, but a study by the Mayo Clinic reported it to be approximately 0.002%.^{1,9}

In 75% to 80% of cases, the HAA is extrahepatic; only about 20% of cases are intrahepatic, and <5%involve both locations.^{2,3,5,9} It has been reported that extrahepatic aneurysms are located in the common hepatic artery in 63% of reported cases, in the right hepatic artery in 28% of cases, in the left hepatic artery in 5% of cases, and in both the right and left hepatic arteries in 4% of cases.^{3–5,8,10} Furthermore, a general trend has been noted in which intrahepatic aneurysms tend to be located in the right hepatic artery.⁴ The aneurysmal diameters reported have ranged between 1.5 and 14 cm, with the mean diameter being calculated at 3.6 cm.9 HAAs are solitary in 92% of cases and multiple in 8% of cases, $2^{2,8-10}$ and about one third of HAA patients present with other visceral artery aneurysms.^{3,5,8,10} Although HAA has been diagnosed in all age groups and both sexes, most patients are adults (age range, 20-80 years) and male.^{1,2,8,10}

The exact etiology of HAA remains to be elucidated, and the ratio of possible risk factors has certainly changed during the past century. At the beginning of the 20th century, most HAAs were considered to result from endocarditis-associated infection or inflammation. However, the widespread use of antibiotics has been accompanied by a decreased ratio of mycotic aneurysms (currently <5% among all HAAs).^{1,3–5,8,10} In the last quarter century, the main etiologic factors have been

atherosclerosis (30%–50%) and medial degeneration (20%). In addition, pathogenic conditions (such as fibromuscular dysplasia, polyarteritis nodosa, systemic lupus erythematosus, tuberosclerosis, and periarterial inflammation secondary to cholecystitis or pancreatitis) and traumatic physical events (such as blunt abdominal trauma, surgical procedures including liver transplantation, hepatic tumor embolization pancreaticoduodenectomy, and percutaneous interventional procedures of the biliary tract) may cause frank or pseudo HAA.^{3–8,10}

Nearly 75% of HAAs are asymptomatic and generally are detected incidentally by radiologic tests performed to address other indications.^{3,8} For 50% to 55% of symptomatic cases, the most frequent symptom is abdominal pain beginning at the right upper quadrant and epigastrium, and radiating to the back.3,5,8 The most common symptoms after abdominal pain are gastrointestinal bleeding and jaundice⁴; however, as stated in the introduction of the current study, only one third of cases demonstrate the Quinke triad symptomology.3,4,6,8,9 In most cases, the physical examination provides findings within normal limits, although a pulsatile mass may be palpable in some cases of giant aneurysm.^{5,8} Sudden-onset abdominal pain and obstructive jaundice may take place in some HAA patients upon development of intrahepatic rupture. Patients with extrahepatic rupture may also present with sudden-onset abdominal pain, but in conjunction with signs of hypovolemic shock.⁵

The most common complications of HAAs are intraperitoneal or intrahepatic rupture, retroperitoneal hemorrhage, arteriovenous fistula, and mesenteric ischemia. Furthermore, hemobilia and obstructive jaundice result from fistulization of the aneurysm to the biliary system.5,8 Reports have stated that aneurysm rupture occurs in 14% to 80% of all HAA patients.^{3-6,8,10} Although there are no robust data, the risk factors for a rupture include aneurysmal diameter >2 cm, multiple aneurysm, and certain etiologies (such as nonatherosclerotic).^{1,8,10} Mortality associated with rupture can be as high as 70%,^{1-3,5,6} making early diagnosis and timely initiation of medical intervention critical for preserving the life of these patients. For the current case reported herein, the main indicator for operative treatment was the large aneurysmal diameter (3 cm).

The most commonly employed imaging methods for diagnosis of HAA are ultrasonography, CT/CT angiography, magnetic resonance (MR)/MR angiography, and conventional angiography.^{5,8} Conventional angiography is considered the gold standard for diagnosis because it provides precise information about the lesion's size, shape, and location. The main advantage of conventional angiography is the ability of endovascular embolization or endovascular stent application to occur simultaneously in suitable patients. Endoscopic retrograde cholangiopancreatography and percutaneous transhepatic cholangiography are effective adjunctive methods for diagnosis of intrahepatic aneurysm rupture and are able to indicate the relationship of the biliary system with the aneurysm.

The current dominant view among surgeons is to operate on all aneurysms >2 cm, even when they are asymptomatic; however, no consensus has vet been reached in the literature about the approach to treat hepatic artery aneurysms.^{3,10} Typically, the mode of treatment may vary according to the location and diameter of the aneurysm, the presence of collateral flow, the general condition of the patient, the presence of any comorbid diseases, whether complications have developed due to the aneurysm, and other etiologic factors.^{3,5,8} The most popular treatment methods include endovascular embolization, endovascular stent, excision with end-to-end anastomosis, excision with autologous or prosthetic graft interposition, ligation, aortohepatic artery bypass, interposition grafting with gastroduodenal artery reimplantation, and excision with splenic artery transposition graft.^{1–10} Ligation or embolization with no adjuvant reconstruction may be performed on aneurysms located in the common hepatic artery, including aneurysms that develop proximally to the origin of the gastroduodenal artery. In such cases, the collateral circulation supplied by the superior mesenteric artery provides blood flow to the liver via the gastroduodenal artery.^{3,4} Aneurysms involving the proper hepatic artery (right and/or left hepatic arteries), such as aneurysms that develop distally to the origin of the gastroduodenal artery, should be treated by excision in conjunction with vascular reconstruction.^{3–} ^{5,8} Finally, embolization is recommended for treating intrahepatic HAA in patients considered to be at high risk for rupture.³

The HAA case presented herein had a large hepatic artery aneurysm involving all arteries supplying the liver. The most appropriate approach was excision with vascular reconstruction. Because the left hepatic artery was close to the common hepatic artery, a direct end-to-end anastomosis was carried out. However, a graft of at least 5 to 6 cm long was necessary for reconstruction of the right hepatic artery. Autologous saphenous vein graft, polytetrafluoroethylene graft, and splenic artery transposition graft options were all considered viable options for this particular reconstruction, but the splenic artery transposition graft was selected according to its long-term patency rate. The grafting procedure included isolation and clamping of the splenic artery so that splenic perfusion remained intact (as observed approximately 5 minutes later), after which the splenic artery transposition graft was applied. Although at least 2 anastomoses are required for other vascular graft materials, only a single anastomosis is necessary for splenic artery transposition. Given the postanastomotic stenoses, a single anastomosis is much more beneficial.

In conclusion, the splenic artery transposition graft, which is a natural grafting material, was successfully used in the surgical treatment of an HAA patient.

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