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Surgical repair of an emergent giant hepatic aneurysm with an abdominal aortic dissection: A case report

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Abstract

BACKGROUND

Hepatic artery aneurysm (HAA) is the second most common visceral aneurysm. A significant number of hepatic aneurysms are found accidentally on examination. However, their natural history is characterized by their propensity to rupture, which is very serious and requires urgent treatment. An emergent giant hepatic aneurysm with an abdominal aortic dissection is less commonly reported.

CASE SUMMARY

We report the complicated case of a giant hepatic aneurysm with an abdominal aortic dissection. A 66-year-old female presented with the complaint of sudden upper abdominal pain accompanied by vomiting. Physical examination showed that her blood pressure was 214/113 mmHg. Her other vital signs were stable. Computed tomography found a giant hepatic proper aneurysm and dissection of the lower segment of the abdominal aorta. Furthermore, angiography showed a HAA with the maximum diameter of approximately 56 mm originating from the proper hepatic artery and located approximately 15 mm from the involved bifurcation of the left and right hepatic arteries with no collateral circulation. Therefore, we decided to use a stent to isolate the abdominal aortic dissection first, and then performed open repair. After the operation, the patient recovered well without complications, and her 3-month follow-up checkup did not reveal any late complications.

CONCLUSION

Open surgery is a proven method for treating giant hepatic aneurysms. If the patient's condition is complex, staged surgery is an option.

Key Words: Giant hepatic artery aneurysm; Abdominal aortic dissection; Open repair; Reconstruction; Good prognosis; Case report

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Core Tip: We report a relatively rare case of a giant hepatic aneurysm combined with abdominal aortic coarctation. The patient had an acute onset and was treated for abdominal aortic coarctation after blood pressure control, followed by a second stage open surgery to manage the hepatic aneurysm in a comprehensive manner. The patient's prognosis is good.

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INTRODUCTION

Hepatic artery aneurysm (HAA) is the second most common visceral aneurysm[1]. The incidence rate of HAA in 2091965 patients who visited the Mayo Clinic between 1980 and 1986 was 0.002%[2]. A total of 77% HAAs are isolated in the proximal part of the liver, of which 20% are combined with parenchymal and extraparenchymal invasion and 3% are confined to the liver[3]. Excluding traumatic aneurysms, patients most commonly suffer from HAAs during their sixth decade of life[4]. Lesions in the hepatic circulation show a ratio of approximately 3:2 in terms of sex with male predominance[2]. Risk factors for HAA include atherosclerosis, medial degeneration, infection, trauma, and vasculitis[4]. A large majority of HAAs are diagnosed incidentally *via* computed tomography (CT) scan[3]. Most patients with symptomatic aneurysms present with one or more of Quincke's classic triad of biliary bleeding (jaundice, biliary colic, and gastrointestinal bleeding)[4]. Diagnosis can be made by ultrasound scan, CT angiography (CTA), and digital subtraction angiography. CTA is recommended as the diagnostic tool of choice in patients who are thought to have HAA[5]. Despite recent advances in therapeutic techniques and diagnostic tools, the management of a visceral artery aneurysm remains clinically challenging. Rupture is the most emergent and life-threatening situation in HAA. Lumsden *et al*[4] pointed out that the HAA-related early incidence of rupture and mortality was 9.1% and 22.7%, respectively. Fibromuscular dysplasia and polyarteritis nodosa increase the risk of HAA rupture and account for 50% of HAA ruptures[5]. The majority of these lesions rupture when they are > 2 cm in diameter[3].

The guideline, named "the Society for Vascular Surgery clinical practice guidelines on the management of visceral aneurysms", states that all hepatic artery pseudoaneurysms regardless of cause (Grade 1A) and all symptomatic HAAs regardless of size (Grade 1A) should be repaired as soon as possible; in asymptomatic patients without significant comorbidity, repair is recommended if the true HAA is > 2 cm (Grade 1A) or if the aneurysm enlarges at the rate of > 0.5 cm per year (Grade 1C); in patients with significant comorbidities, repair is recommended if the HAA is > 5.0 cm (Grade 1B); furthermore, the repair of HAA in patients with vasculopathy or vasculitis regardless of size (Grade 1C) or with positive blood cultures (Grade 1C) is recommended[5]. The clinical practice guidelines on the management of visceral aneurysms set by the Society for Vascular Surgery indicate that treatment approaches mainly include endovascular repair with covered stents, open repair, and coil embolization. The endovascular approach represents a minimally-invasive alternative with low mortality and morbidity[6]. Given the abundant collateral supply of the liver, the incidence of hepatic necrosis after disruption of the common hepatic artery is low. Percutaneous embolization is of special value in patients with intrahepatic aneurysms[5]. Endovascular therapy has become the mainstream approach. However, open repair remains a therapeutic option with definite efficacy and is mostly chosen under the conditions of HAA rupture, infeasible endovascular approach and for symptomatic patients with fibromuscular dysplasia or polyarteritis nodosa and lesions in the proper hepatic and proximal right or left hepatic branches[5].

CASE PRESENTATION

Chief complaints

A 66-year-old woman was admitted to our hospital with the chief complaint of severe abdominal pain with vomiting. Four hours before admission, the patient had a sudden onset of sharp pain in the upper

and middle abdomen with no obvious cause. The pain was unbearable and persistent without relief, which involved back pain and was accompanied by vomiting the contents of the stomach, without dizziness, headache, chest tightness, chest pain, acid reflux, heartburn, chills, fever and other symptoms.

History of present illness

The patient was found to have hypertension for more than 20 years, with the highest blood pressure reaching 220/160 mmHg. She was taking nimodipine tablets (30 mg tid) regularly, and her blood pressure was controlled at approximately 140/75 mmHg, usually without dizziness and headache.

History of past illness

The patient had no other previous illnesses.

Personal and family history

Her personal and family history was unremarkable.

Physical examination

Physical examination showed slight tenderness in the upper abdomen and no rebound pain; blood pressure of 214/139 mmHg; pulse of 64 beats/min; and temperature of 36.4°C.

Laboratory examinations

Her blood test results showed no special abnormalities.

Imaging examinations

CT revealed: (1) A giant aneurysm of the proper hepatic artery (maximum diameter approximately 56 mm); and (2) Dissection of the lower abdominal aorta (single break) (Figure 1A and B).

FINAL DIAGNOSIS

The patient was diagnosed with abdominal aortic dissection, hepatic artery aneurysm, and hypertension grade 3 (very high risk).

TREATMENT

After receiving blood pressure control, sedation and related symptomatic treatment from the coronary heart disease center of our cardiology department, the patient's symptoms disappeared and her vital signs stabilized. The patient was transferred to our department on the same day of admission due to CT findings of abdominal aortic coarctation and a hepatic aneurysm. We performed angiography, which showed that the HAA had a maximum diameter of approximately 5.6 cm and that it originated from the proper hepatic artery and was located approximately 1.5 cm from the involved bifurcation of the left and right hepatic arteries with no collaterals. Prolonged angiography revealed no communication between the HAA and superior mesenteric artery (Figure 1C). Considering the complexity of the patient's condition, the aortic dissection was repaired with a Endurant II stent graft (Medtronic, Inc.) at the first stage, and the HAA was scheduled for surgical repair at the second stage. Postoperatively, the patient was treated with antiplatelet, lipid-lowering and blood pressure control therapy.

Open repair was performed six days later. A right subcostal incision was made, and the surgical approach was *via* the small omental sac. Intraoperative findings showed the following: the proper hepatic artery, which was approximately 6 cm × 6 cm in size, was located between the medial side of the descending duodenum and the anterior of the pancreatic head and bile duct (Figure 2A). We then mobilized the inflow and outflow of the proper hepatic artery. After systemic heparinization, the inflow and outflow of the HAA was clamped, and the aneurysm was directly opened. An aneurysm break approximately 2 mm in size and slight mural thrombus (Figure 2B) were found. No collateral vessel was detected in the aneurysm. The proximal part of the proper hepatic artery was anastomosed end to end with the right hepatic artery as the adjacent orifice location, and the left hepatic artery was anastomosed end to side with the proper hepatic artery (Figure 2C). The hepatic artery clamp time was 31 min. After anastomosis, ultrasound revealed the patency of the anastomotic site and the distal hepatic artery branches. The operation was performed without difficulties.

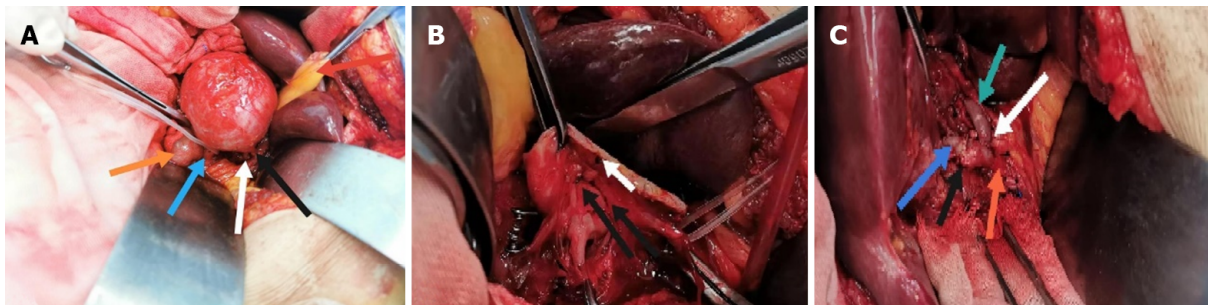
OUTCOME AND FOLLOW-UP

Postoperatively, the patient experienced no specific discomfort. Antiplatelet, blood pressure control,



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Figure 1 Computed tomography scan. A: Abdominal computed tomography (CT) three-dimensional reconstruction showed a proper hepatic artery aneurysm (black arrow) and abdominal aortic dissection (white arrow); B: The patient's abdominal CT scan showed a huge proper hepatic artery aneurysm with a maximum diameter of approximately 56 mm; C: Abdominal aortography showed a huge proper hepatic aneurysm: A bit twisted, no collaterals, originated from the proper hepatic artery (orange arrow) and involving the bifurcation of the left (black arrow) and right hepatic arteries (blue arrow).



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Figure 2 Open repair was performed six days later. A: This is a general view of the isolated hepatic aneurysm. The red arrow indicates the gallbladder; the yellow arrow the descending duodenum; the green arrow the proper hepatic artery; the white arrow the right hepatic artery and the black arrow the left hepatic artery; B: This is a general view of the cut aneurysm. The white arrow indicates the aneurysm break and the black arrow the mural thrombus; C: The general view of the proper hepatic artery (red arrow) after suturing with the left (dark green arrow) and right hepatic arteries (light green arrow), respectively. The black arrow indicates the end-to-end anastomosis of the proper hepatic artery and the right hepatic artery while the white arrow indicates the end to side anastomosis of the proper hepatic artery and the left hepatic artery.

and lipid-lowering treatments were maintained. Eleven days later, the patient was successfully discharged without surgery-related complications. The important times and events during the patient's hospitalization are shown in Table 1. The patient's 3-mo follow-up checkup did not reveal any late complications (Figure 3). She reported no specific discomfort on review and was very satisfied with her treatment.

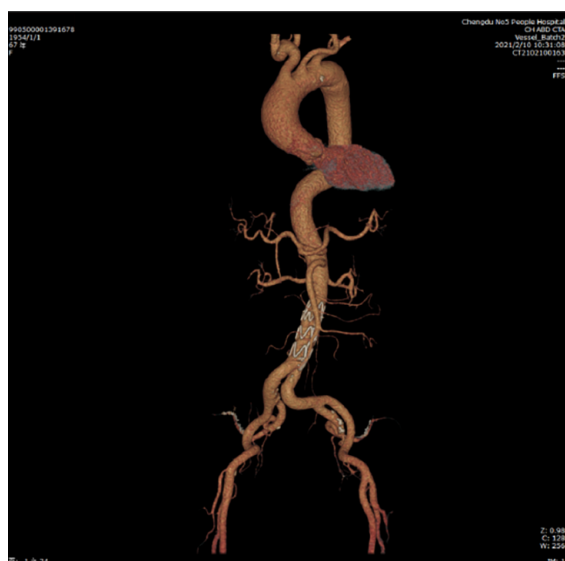
DISCUSSION

Visceral aneurysms, despite their rare incidence of 0.01%-0.2%, are of clinical importance, especially if we consider their natural history which is characterized by their propensity to rupture, with HAA accounting for approximately 20% of visceral aneurysms and a rupture rate of 44% [5]. They are usually asymptomatic and difficult to detect until they rupture and cause abdominal pain and hypovolemic shock. As a result, most visceral aneurysms are found incidentally. The mortality rate following ruptured visceral aneurysms remains high (30% reported in the last decade) [7].

The timing of the intervention for hepatic aneurysms has been mentioned above. The treatment of a hepatic aneurysm is mainly as follows: Covered stent, open repair, and embolization [2,3,5]. The ideal surgical option should be to remove the aneurysm while maintaining the hepatic circulation. Therefore, the primary treatment of hepatic aneurysms varies by site. The main treatments for common HAAs include open surgical ligation, endovascular embolization, resection/reconstruction, aneurysmorrhaphy, and a covered stent; those for the proper hepatic artery are resection with arterial reconstruction and endovascular repair with a covered stent; those for the proximal right or left hepatic branches are resection with arterial reconstruction and endovascular stent grafting; and finally, those for an

Table 1 Important events and dates during this patient's hospitalization

Date	Events
December 8, 2020	(1) The patient was admitted to the emergency department with acute abdominal pain and widespread pulling pain in the back with a blood pressure of 214/139 mmHg at the time of the emergency; (2) Computed tomography (CT) suggested abdominal aortic coarctation with intramural hematoma, hepatic artery aneurysm, bilateral common iliac artery and calcified plaque in the internal iliac artery; and (3) The patient was transferred to our department due to CT findings of abdominal aortic coarctation and hepatic aneurysm
December 14, 2020	Ultrasound showed no special abnormalities in the renal artery and bilateral carotid and vertebral arteries
December 23, 2020	Abdominal aortogram + endoluminal isolation of abdominal aortic coarctation (non-emergency) was performed
December 29, 2020	Hepatic intrinsic aneurysm resection+ hepatic artery reconstruction (non-emergency) was performed
January 9, 2021	The patient was successfully discharged with a good prognosis and without any associated complications

**DOI:** 10.12998/wjcc.v10.i17.5798 **Copyright** ©The Author(s) 2022.**Figure 3** The patient was reexamined 3 mo after surgery and showed no complications. The anastomotic end of the proper hepatic artery was unobstructed. The abdominal aortic dissection was well isolated.

intrahepatic aneurysm are endovascular embolization and resection of the lobe in which the aneurysm is located[5,8]. However, the specific choice of treatment should be based on the patient's specific circumstances.

In this case, we did not select coil embolization mainly for the following reasons: First, the endovascular repair of extrahepatic HAA depends on the collaterals and location of the HAA. Given that the maintenance of distal organ perfusion is important, embolization is usually discouraged in patients with HAAs in the proper hepatic artery due to the risk of liver ischemia[5]. Furthermore, in this case, the location of the HAA in the proper hepatic artery involved the bifurcation of the left and right hepatic arteries with no collateral circulation and thus increased the risk. Second, the HAA was so large that a large parenchymal lesion would be created if we performed embolization; this lesion might compress the biliary tract and duodenum and thus cause jaundice, gastrointestinal obstruction, and even duodenum fistula[5,9].

Another main option for HAA repair is endovascular stent grafting. The endovascular repair of visceral aneurysms with stent implantation can simultaneously enable aneurysm exclusion and vascular preservation, and therefore minimize the risk of ischemic complications[10]. Nearly all retrospective case series have shown that although the outcomes for visceral artery aneurysms after open or endovascular repair share similar long-term results, morbidity is significantly worse with open repair than with the endovascular approach[5,8]. The scope of aneurysm morphology suitable for endovascular repair is expanding with the accumulation of experience and improvements in equipment. The anatomical complexity of aneurysms is generally believed to affect the technical difficulty of repair with the development of the application of endovascular covered grafts; this belief is the main reason why we did not choose the approach of endovascular covered grafting. The main complications of

endovascular stent grafting include occlusion[9,11]. However, the patency rate of hepatic artery stenting is rarely reported. Künzle *et al*[12] reported that the 2-year patency of the endovascular stent grafting of visceral artery aneurysms is approximately 81%.

Open surgery, which is usually known as open surgical revascularization, is another common method for the treatment of HAA. Considering the possibility of central liver necrosis despite adequate collateral flow by endovascular exclusion, open repair is recommended in low-risk patients if endovascular stent graft exclusion is not possible[5]. In addition, open surgery has its unique role in aneurysm rupture.

The main methods of vascular reconstruction include direct vascular anastomosis and bypass of the artificial vascular or saphenous vein and vascular patch[11]. The main complications of open surgical revascularization are infection and occlusion. Erben *et al*[11] reported that in open surgical revascularization, the incidence of occlusion is 12%, with saphenous veins and artificial vessels sharing 6% and 6% equally, and the incidence of infection is 6%.

In this case, deploying the covered stent was difficult considering the tortuosity of the delivery route. Therefore, the proper hepatic artery was anastomosed end to end with the right hepatic artery, and the left hepatic artery was anastomosed end to side with the proper hepatic artery without an artificial blood vessel or saphenous vein. This approach was riddled with the considerations discussed above. First, we anastomosed the blood vessels directly because the ends were highly adjacent, and the tension was low after direct anastomosis with no need for the use of artificial blood vessels or saphenous veins, so that the patient could reduce the subsequent anticoagulant burden. Second, we did not first anastomose the left and right hepatic arteries and then anastomose them with the proper hepatic artery as during the operation, we found that the patient's right hepatic artery was thick and large, so that we could prevent complications in one of the left and right hepatic arteries from affecting the other artery to the greatest extent. Moreover, we did not completely isolate the whole aneurysm, thus reducing the damage to the surrounding tissue and the incidence of postoperative complications. During the entire operation, the hepatic artery occlusion time was 31 min, which reduced the probability of hepatic ischemia.

CONCLUSION

Diagnosing huge hepatic aneurysms in time and choosing the best treatment are very challenging. When other serious diseases, such as Stanford type B aortic dissection, are found at the same time, the complexity of the patient's condition and the difficulty of treatment double. Although endovascular therapy is the first choice in most cases, open surgery still has a unique role. We should not only strictly understand the indications of various surgical procedures, but also make clinical decisions in accordance with the specific conditions of patients.

FOOTNOTES

Author contributions: Wen X was responsible for collecting the information and writing the article; Yao ZY was involved in surgery and communication with the patient; Zhang Q participated in surgery and data collection; Wei W participated in surgery; Chen XY revised the article; Huang B designed the surgical plan and participated in the surgery; all authors have read and approved the final manuscript.

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