Case Report

Aneurysms of Gastroepiploic Artery and Vein with an Arteriovenous Fistula after Partial Gastrectomy in a Patient Presenting with Abdominal Aortic Aneurysm—Report of a Case

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A case of asymptomatic right gastroepiploic artery and vein aneurysms with an arteriovenous (AV) fistula coexisting with an abdominal aortic aneurysm (AAA) is presented. A 68-year-old man was referred for treatment of AAA (6 cm in diameter) and he was incidentally diagnosed as having right gastroepiploic artery and venous aneurysms (3 cm and 8 cm in diameter, respectively) with an AV fistula. Resection of the aneurysms and closure of the fistula were successfully completed with AAA repair. He had a history of gastrectomy, and these gastroepiploic aneurysms with an AV fistula were considered a late complication due to mass ligation of vessels during gastrectomy.

Key words: gastroepiploic artery aneurysm, gastroepiploic venous aneurysm, arteriovenous fistula

INTRODUCTION

Visceral artery aneurysms are unlike aneurysms of the peripheral arterial system. The etiology of visceral artery aneurysms is multifactorial, and they can arise secondary to trauma, medial degeneration, or periarterial inflammation; whereas peripheral artery aneurysms have been proved to be caused mainly by degenerative change of arterial wall.¹⁾ Visceral artery aneurysms are relatively rare but are associated with high mortality due to rupture.²⁻⁴⁾ Of all splanchnic artery aneurysms, less than 4% are located in the gastric and gastroepiploic arteries (GEA).³⁾ The incidence of GEA aneurysms is only about one tenth of that of gastric artery aneurysms.³⁾

A previous study showed that 90% of GEA aneurysms were found to be ruptured on discovery, with gastrointestinal bleeding or intraperitoneal hemorrhage.^{3,4)}

We present a case of an asymptomatic right GEA aneurysm and arteriovenous (AV) fistula to the right gastroepiploic venous aneurysm following partial gastrectomy, coexisting with abdominal aortic aneurysm (AAA).

CASE

A 68-year-old man with a history of partial gastrectomy 30 years ago was referred to our hospital for treatment of an AAA. There was no history of weight loss, appetite loss, hematemesis, or epigastralgia, but he had hypertension. He has no remarkable family history. On admission his general condition was good, and a physical examination revealed no abnormal findings apart from a pulsatile AAA and a bruit and thrill in the abdomen. Laboratory investigation showed no abnormalities except for renal insufficiency with serum creatinine level of 2.48 mg/dl. Ultrasonographic (USG) examination as well as computed tomography (CT) scanning showed an infrarenal AAA with a diameter of 6 cm and a large cystic lesion beneath the liver, which had turbulent blood flow in connection with an aneurysmal splanchnic artery. (**Fig. 1A**) Magnetic resonance image (MRI) showed a connec-

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Fig. 1 Preoperative ultrasonography and MRI findings.

A: USG showed a cystic lesion with a diameter of 8 cm beneath the lateral segment of the liver. The right GEA was located next to the cystic lesion and showed tortuosity and aneurysmal dilatation 3 cm in diameter. USG with color Doppler detected arterial blood flow in the aneurysmal right GEA and also detected turbulent blood flow in the huge cystic lesion.

B: MRI examination showed a connection between the cystic lesion and superior mesenteric vein (white arrow).

tion between this large cystic lesion and superior mesenteric vein (SMV) (**Fig. 1B**). A selective celiac and superior mesenteric arteriogram revealed an aneurysm that connected into the right GEA, extending through to the gastroduodenal artery (**Fig. 2**). The posterior pancreaticoduodenal artery was delineated and a circular calcification was seen adjacent to the right GEA just beneath the liver.

All aneurysms were explored through a midline incision. The right GEA was aneurysmal and measured 3 cm in diameter and 7 cm in length, and was located between the greater omentum and the pancreas. The extended venous segment connecting to the right GEA aneurysm showed aneurysmal change, measured 8×6×6 cm in size, and was connected to the SMV. Turbulent blood flow could be seen through the wall. No pulsation but a slight

Fig. 2 Selective celiac and superior mesenteric arteriographic findings.

Selective celiac (**A**) and superior mesenteric (**B**) arteriograms showed a tortuous aneurysm, which continued into the GEA through the gastroduodenal artery and the posterior superior pancreaticoduodenal artery. A large cystic lesion was seen connected to the aneurysmal right GEA in the late phase.

thrill was detected on the venous aneurysm, which indicated there was an arterial communication to the portal system. After opening the greater omentum, the origin of the right GEA was exposed and a communication between the right GEV and superior mesenteric vein was found (**Fig. 3A**). The portal venous pressure was 14 cm H₂O, which decreased to 13 cm H₂O after clamping of the right GEA. The right GEA and GEV aneurysms with AV fistula were resected en bloc. The origin of the right GEA was sutured and ligated with polypropylene sutures and the right GEV stump was closed. The infrarenal AAA was saccular in shape and measured 6 cm in diameter. The aneurysm involved both common iliac arteries and had a diameter of 2 cm. A bifurcated prosthetic graft was chosen for arterial reconstruction. The postoperative course was uneventful and the patient was discharged on

Fig. 3 Operative and histological findings.

A: The origin of the right GEA was exposed and controlled by a vascular tape. The cystic mass was found to be the venous aneurysm connected to the GEV. A communication between the SMV and the GEV was exposed and controlled by a vascular tape.

B: The aneurysmal wall showed moderate atherosclerosis with marked intimal thickening and attenuation of the media with calcification and myxoid degeneration.

the 14th postoperative day.

Pathologic examination confirmed a macroscopic AV fistula between the right GEA and GEV aneurysms. The aneurysmal wall of the right GEA showed marked intimal thickening and attenuation and destruction of the media with calcification and myxoid change. Mild fibrosis was observed in the adventitia, but inflammatory reaction was slight (**Fig. 3B**).

DISCUSSION

Aneurysms of the GEA are reported to comprise only 0.4% of all splanchnic aneurysms.³⁾ Due to a rupture rate of nearly 90%, these aneurysms have great clinical importance.^{2,4)} Previous studies suggested that most cases of ruptured gastric artery and GEA aneurysms caused life threatening gastrointestinal bleeding and hemoperitone-

um.^{2,4,5)} In patients who are found to have a splanchnic aneurysm, a complete vascular examination should be considered because of the 15–40% reported frequency of multiple aneurysms.^{2,6,7)} For the proper diagnosis, selective angiography has been the most widely advocated approach. Now, however, CT scanning,^{3,5,7)} MRI and USG with color Doppler⁴⁾ are also available as effective and noninvasive modalities, and provided good images for the proper diagnosis in the present case. It is reported that all multiple splanchnic aneurysms should be resected during the same operation when technically feasible because of their high rupture rate and prohibitive mortality following emergency operation.⁸⁾ In our present case, three aneurysms, the right GEA and GEV aneurysms with an AV fistula coexisting with AAA, were found and we resected all aneurysms at the same time.

Recently transcatheter coil embolization has been shown to be an effective and less invasive alternative to open surgery for the management of visceral aneurysms.⁹⁾ In the present case, however, the GEA aneurysm was connected to the SMV and portal system via an AV fistula, and coil dislocation to the portal system had to be avoided. Therefore, we chose surgical repair for the GEA and GEV aneurysms in consideration of the necessity of AAA repair.

Stanley et al. argued that the common histological findings of arteriosclerosis represented a secondary event rather than initiating cause of most visceral artery aneurysms, which occur due to trauma, medial dysplasia, gestational alteration, connective tissue disorders, and atherosclerosis.^{10,11)} They also reported that calcific arteriosclerotic changes were found pathologically in aneurysms alone, without involving adjacent vessels.^{10,11)} In our case, histological findings of the aneurysmal wall of the GEA showed moderate atherosclerosis with an attenuated media with calcification and myxoid degeneration. Regarding the etiology of the right GEA aneurysm in our case, it was not clear whether the atherosclerotic change was primary, or secondary to some degenerative change of arterial wall. Stanley et al. reported that coexistence of visceral and aortic aneurysms was found in 20% of cases.¹⁰⁾ Graham and his colleagues also reported that 28% of patients with a splanchnic artery aneurysm had a concomitant AAA.¹²⁾ However, the association between visceral and aortic aneurysms is not clear, other than that the arterial wall was found to be degenerative in both lesions. This might suggest that degenerative changes of the arterial wall cause visceral artery aneurysms as well as AAA, and that this could be a candidate for the cause

of the right GEA aneurysm in the present case.

The trauma during previous gastrectomy as well as degeneration of the arterial wall might also have been contributing factors for the GEA aneurysm formation in the present case. However, aneurysms that followed vascular trauma show a saccular form and might be pseudoaneurysms in many cases. In our case, the GEA aneurysm was found to be fusiform and not a pseudoaneurysm from the histopathological findings. Brachial artery aneurysms are sometimes found in patients with an AV fistula for hemodialysis.¹³⁾ In patients with AV fistulas for hemodialysis, blood flow in the running-in artery of an AV fistula is increased and this increased blood flow results in tortuosity and aneurysmal dilatation of the proximal artery.¹³⁾ In our case, this mechanism may also have contributed to the formation of the right GEA aneurysm.

Major vascular injury following partial gastrectomy is rare. However, one of the late complications following gastrectomy is reported to be an AV fistula between the GEA and GEV caused by en masse or suture ligation of an artery and an adjacent vein that subsequently develops into an enlarging fistula.¹⁴⁾ Out of patients with an AV fistula after gastrectomy, only 20% were asymptomatic and the other 80% had some symptoms, which were not life threatening.¹⁴⁾ Although our case had no symptoms, he also had no history of abdominal trauma but had undergone partial gastrectomy, and the AV fistula in our case was considered to be due to the previous gastrectomy procedures.

Portal hypertension is reportedly one of the clinical features of the arteriportal fistula.¹⁵⁾ In the present case, however, preoperative examinations by CT scanning and USG showed no splenomegaly or hepatic cirrhosis. The portal venous pressure was 14 cm H₂O, which decreased a little to 13 cm H₂O after clamping the right GEA. These data suggested that the arterial pressure through an AV fistula would play a part in forming and enlarging the venous aneurysm without raising the venous pressure of run-off vessels and this huge venous aneurysm, in which turbulent blood flow could be seen, was considered to be a pressure buffer in this AV fistula following no portal hypertension.

In summary, both the en masse ligation of right GEA and GEV during gastrectomy with a subsequent AV fistula, and vascular degeneration were suggested to play an important role in the pathogenesis of the GEA and GEV aneurysms with AV fistula in the present case. The case suggested that during gastrectomy great care should be taken to ligate vessels after sufficient identification

and isolation, especially in younger patients who might suffer vascular degeneration in their older years, leading to aneurysmal diseases.

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