

CASE REPORT

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Accessory left gastric artery aneurysms in granulomatosis with polyangiitis: a case report and literature review

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ABSTRACT

Aneurysm formation is a potential complication of granulomatosis with polyangiitis (GPA), previously known as Wegener's granulomatosis. It is a very rare complication, but immediate diagnosis and therapy should be performed because an aneurysm can be life-threatening if it ruptures. An accessory left gastric artery (ALGA) is also a rare variant gastric artery that may obtain its blood supply from the left hepatic artery and left gastric artery. We herein describe a 57-year-old Japanese man who was diagnosed with GPA complicated by aneurysm rupture in an ALGA. Emergency surgery was performed after failure of arterial coil embolization to interrupt blood flow in the ALGA. The patient underwent partial resection of the lesser omentum, which contained all aneurysms. During partial resection of the lesser omentum, both the left gastric artery and ALGA were ligated because they were thought to be feeders of the aneurysms. Postoperative recovery was uneventful; no bleeding or recurrence of the aneurysms occurred. Immediate diagnosis and therapy should be performed for patients with GPA with symptoms of vascular ischemia or aortitis. Endovascular intervention is the first-choice therapy especially for hemodynamically stable patients with ruptured aneurysms or aneurysms located on variant arteries, which may have multiple blood supplies. In the present case, although endovascular treatment failed, the approach described herein was helpful during open surgery.

Key Words: granulomatosis with polyangiitis, aneurysm, accessory left gastric artery

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BACKGROUND

Granulomatosis with polyangiitis (GPA), previously known as Wegener's granulomatosis, is multisystemic disease characterized by systemic necrotizing and/or granulomatous vasculitis. Inflammation in GPA typically affects the small- to medium-sized arteries of the upper airways, lungs, and kidneys.¹⁾ This may lead to aneurysm formation in these arteries as well as in larger arteries. Aneurysm formation is a very rare complication of GPA, and only a few cases have been reported. This is the first report of GPA complicated by aneurysm rupture in an accessory left gastric artery (ALGA). An ALGA is also a rare variant gastric artery with an incidence of 3% to 14% in angiographic studies.^{2,3)} We herein discuss the management strategy of GPA

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complicated by aneurysms occurring in this anatomic variant.

CASE PRESENTATION

A 57-year-old Japanese man was admitted to our hospital with a 1-month history of pedal edema. His medical history included hypertension, hyperlipidemia, and atrial fibrillation, for which he had undergone atrial ablation 4 years previously. He had no history of using anticoagulant agents.

On physical examination, he was afebrile. His blood pressure was 110/68 mmHg, pulse 74 beats/minute, respiration rate 15 breaths/minute, height 168 cm, and weight 68 kg. He had pitting edema on his bilateral lower extremities. His respiratory sounds were clear, and his heart sounds were regular without any murmur. The abdomen was soft, and there were no dermatologic manifestations, nasopharyngeal abnormalities, or inflamed joints.

Laboratory data were as follows: leukocyte count, $7.5 \times 10^3/\mu\text{L}$ (80% neutrophils, 7% lymphocytes, 9% monocytes, and 4% eosinophils); hemoglobin, 7.4 g/dL; hematocrit, 23.3%; platelet count, $34.8 \times 10^3/\mu\text{L}$; C-reactive protein, 12.3 mg/dL; blood urea nitrogen, 56.7 mg/dL; serum creatinine (Scr), 3.2 mg/dL (2 years prior to the current presentation, his Scr level was 0.93 mg/dL); and albumin, 1.8 mg/dL. His electrolyte and liver enzyme levels were normal. His erythrocyte sedimentation rate was 103 mm/h. Urinalysis showed protein of 2+, occult blood of 3+, and many dysmorphic red blood cells and red blood cell casts. The urine protein-to-creatinine ratio was 1.50 mg/mg. The proteinase-3 antineutrophil cytoplasmic antibody (PR3-ANCA) level was 157 IU/mL (reference range, <3.5), while myeloperoxidase antineutrophil cytoplasmic antibody, antinuclear antibody, and antiglomerular basement antibody were negative. The serum total complement and complement 3 and 4 levels were within normal limits. Viral serology for human immunodeficiency virus, hepatitis B virus, and hepatitis C virus was unremarkable. A cryoglobulin test was negative. Serum protein electrophoresis showed that IgG, IgA, and IgM were within normal limits.

Chest X-ray revealed an infiltrative shadow in the left upper lobe (Fig. 1a). Chest computed tomography (CT) showed multiple intraparenchymal cavitate nodules in the left lung (Fig. 1b).

Fiberbronchoscopic biopsy was performed, and periodic Schiff-methenamine staining showed no granulomas; however, numerous neutrophils and macrophages were infiltrating the interstitium. Ziehl-Neelsen and Grocott staining were negative.

Based on renal insufficiency, chest radiographic abnormalities, and PR3-ANCA positivity, the patient was diagnosed with GPA and began pulse therapy with methylprednisolone at 500 mg/day for 3 days followed by maintenance with prednisolone at 60 mg/day.

Eleven days after starting steroid therapy, he complained of sudden-onset persistent epigastric pain. His blood pressure was 102/70 mmHg and pulse was 57 beats/minute. His abdomen was mildly tender in the epigastric quadrant without rigidity.

Laboratory data revealed a hemoglobin level of 7.2 mg/dL, which was 2 mg/dL lower than that 1 day prior. The leukocyte count was $12.8 \times 10^3/\mu\text{L}$ (90% neutrophils, 5.9% lymphocytes, 3.0% monocytes, and 0.8% eosinophils), C-reactive protein was 0.76 mg/dL, BUN was 70.7 mg/dL, Scr was 3.14 mg/d, and PR3-ANCA was 161 IU/mL. Abdominal contrast-enhanced CT confirmed the presence of stringing 5- to 10-mm-diameter aneurysms located on a branch of the left hepatic artery. A medium- to high-density area was noted around the aneurysms (Fig. 2).

Rupture of these aneurysms with continuous bleeding was suspected, and angiography of the common hepatic artery was performed on an emergency basis. Angiography showed four stringing aneurysms located on the ALGA arising from the left hepatic artery (Fig. 3). Arterial coil

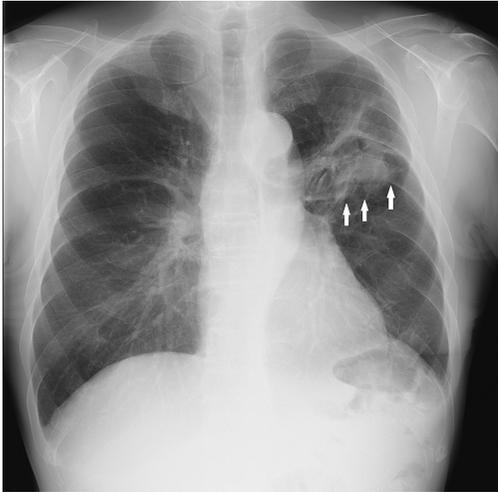


Fig. 1 (a) Chest X-ray on admission, showing an infiltrative shadow in the left upper lobe.

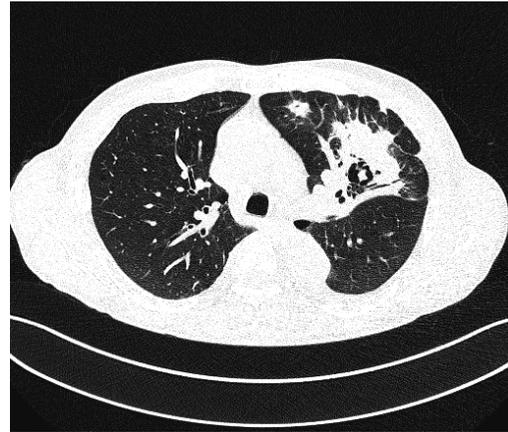
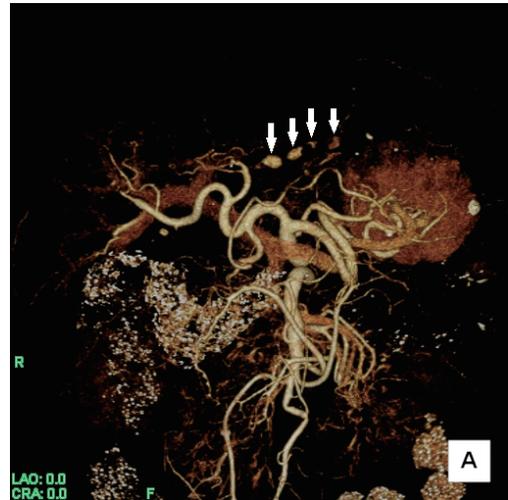


Fig. 1 (b) Chest computed tomography scan on admission, showing multiple intraparenchymal cavitate nodules in the left lung.



(a)



(b)

Fig. 2 Abdominal contrast-enhanced computed tomography scan performed at the onset of persistent epigastric pain. (a) Edema and a hematoma are present in the lesser omentum, and accumulation of fluid is present in the abdominal cavity. (b) Three-dimensional image reconstruction shows the presence of stringing aneurysms located on a branch of the left hepatic artery.

embolization to interrupt blood flow in the ALGA failed, and emergency surgery was performed.

Surgery revealed accumulation of a large amount of blood in the abdominal cavity and a hematoma in the lesser omentum; other bleeding sources were not identified. Four stringing aneurysms of the ALGA were confirmed in the lesser omentum. The patient was therefore diagnosed with lesser omental hemorrhage caused by ruptured aneurysms of the ALGA and

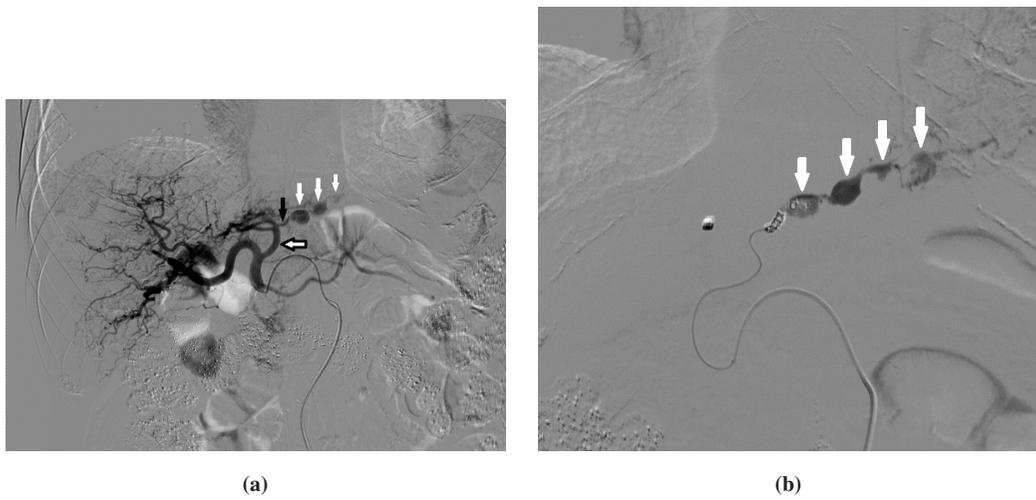


Fig. 3 Common hepatic artery angiography. **(a)** Stringing aneurysms (white arrows) are located on an ALGA (black arrow) arising from the left hepatic artery(void arrow). **(b)** After coil embolization, the stringing aneurysms are still enhanced.



Fig. 4 (a) Resected lesser omentum containing all four aneurysms.

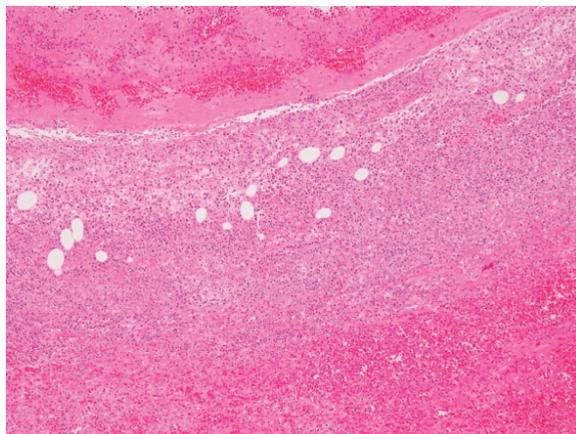


Fig. 4 (b) The aneurysmal wall is infiltrated by numerous neutrophils and macrophages, indicating necrotizing inflammation. Hematoxylin–eosin staining, $\times 200$.

underwent partial resection of the lesser omentum, which contained all four aneurysms (Fig. 4a). During partial resection of the lesser omentum, both the left gastric artery and ALGA were ligated because they were thought to be feeders of the aneurysms. Histopathological examination showed no granulomas, but necrotizing inflammation of the aneurysmal wall was present (Fig. 4b). This was consistent with the diagnosis of GPA and thought to be the main factor involved in the pathogenesis of this aneurysmal rupture.

Postoperative recovery was uneventful, and prednisolone was restarted at 60 mg/day from the first postoperative day. From postoperative day 14, 900-mg cyclophosphamide pulses were administered intravenously six times on a monthly basis. The daily prednisone dose was gradually tapered to 10 mg for 6 months. Treatment for GPA was continued, and a follow-up CT scan showed no new signs of vascular inflammation or other aneurysms.

DISCUSSION

GPA is a rare disease; in Japan, its estimated annual incidence is 2.3 cases per million individuals.⁴⁾ This rate is much lower than that observed in northern European countries (Norway, 14.4 per million⁵⁾; Sweden, 11.9 per million⁶⁾). However, the number of patients with GPA in Japan has been gradually increasing and doubled in the past 10 years.⁷⁾

GPA is a multisystemic disease characterized by systemic necrotizing and/or granulomatous vasculitis. The pathogenesis of GPA begins with expression of glycoprotein enzymes such as PR3 on the surface of cytokine-primed neutrophils.⁸⁾ PR3-ANCA then binds to this antigen, activating neutrophils and resulting in cytotoxicity to vascular endothelial cells via release of superoxides, lytic enzymes, and proinflammatory cytokines.^{9,10)} PR3-ANCA is observed in 96% of patients with GPA and helps to establish the diagnosis of GPA.¹¹⁾ Neutrophil extracellular traps may also contribute to the pathogenesis.¹²⁾ GPA tends to affect the medium and small arteries of the respiratory tract and kidneys.¹⁾ This inflammation can also lead to aneurysm formation in these and larger arteries, although aneurysm formation is a very unusual feature.

A MEDLINE search of the medical literature revealed that 20 published cases described medium- and large-vessel aneurysms in patients with GPA (Table 1).¹³⁻³¹⁾ The mean age of the 21 patients (including the patient in the present case) was 44.9 years (range, 22–67 years). Arterial aneurysms were observed more commonly in men (n = 17) and diagnosed within 1 month from disease onset (n = 12). Most patients had symptoms of vascular ischemia or aortitis (n = 16), such as abdominal pain (n = 8) and back pain (n = 5). Aneurysmal rupture occurred in nine patients, three of whom died. Four of these nine patients had begun prednisone and other immunosuppressants, but rupture occurred in one patient as long as 22 years after beginning prednisone; the remaining five patients had never used such drugs when aneurysmal rupture occurred. Aneurysms were commonly diagnosed by ultrasound (n = 5), CT (n = 12), angiography (n = 8), or their combination. With respect to site, 7 cases were large-vessel aneurysms and 14 cases were medium-vessel aneurysms: branches of the celiac axis (n = 8), branches of the renal artery (n = 5), craniocervical artery (n = 2), coronary artery (n = 1), and superficial femoral artery (n = 1). Ours is the first reported aneurysm of an ALGA in a patient with GPA.

An ALGA is a variant gastric artery that arises from the left hepatic artery and supplies the cardia and fundus of the stomach. The reported incidence of this anatomic variant is 3% to 14% in angiographic studies.^{2,3)} Ishigami *et al.*³²⁾ stated that this vessel tends to be seen more frequently in Japanese than European individuals. An accessory or replaced left hepatic artery arises from the left gastric artery and supplies the lateral segment of the left hepatic lobe. These two arteries are similar in embryonic development.

Table 1 Summary of large- and medium-sized vessel aneurysms in patients with GPA: literature review

References	Gender/ Age (y)	Aneurysmal symptoms	Aneurysmal symptoms from diagnosis of GPA	Diagnostic tools for aneurysms	Involved Artery	Therapy	Rupture	Outcome
Den Bakker (13)	M/55	Right upper quadrant pain	15 days after	US, autopsy	Hepatic	Immuno- suppression	yes	Death
Baker (14)	M/24	Right flank pain, shock	Concomitant	Angiography	Renal	Endovascular, immuno- suppression	yes	Recovery
Moutsopoulos (15)	M/30	NS	4 months after	Angiography	Renal	Immuno- suppression	no	NS
Moutsopoulos (15)	F/53	NS	1 month after	Angiography	Renal	Immuno- suppression	no	NS
Sieber (16)	M/59	Abdominal pain	9 months before	CT, laparotomy	Aorta	Surgery	no	Recovery
Aoki (17)	M/56	Shock	Concomitant	Autopsy	Left gastric	Resuscitation	yes	Death
Blockmans (18)	M/42	Abdominal pain	3 weeks before	US, CT	Aorta	Surgery, Immuno- suppression	no	Recovery
Shirit (19)	M/58	Ischemic painful hand	9 days after	Angiography	Subclavian	Endovascular	no	NS
Senf (20)	M/35	Left flank pain, shock	1 month after	US, CT	Hepatic, renal, splanchnic	Immuno- suppression	yes	Recovery
Famularo (21)	M/67	Abdominal pain, shock,	1 month after	laparotomy	PD	Surgery	yes	Death
Takei (22)	M/34	Sever headache	1 month before	CT, angiography	Intracranial	Endovascular	yes	Recovery
Carels (23)	M/63	Low back pain, fever	Concomitant	CT	Aorta	Surgery, Immuno- suppression	no	Recovery
Arlet (24)	M/29	Abdominal pain, vomiting	5 years after	CT	PD, hepatic, renal	Endovascular, immuno- suppression	no	Recovery
Minnee (25)	F/53	Low back pain	2 months before	CT	Aorta	Immuno- suppression	yes	Recovery
Durai (26)	F/33	Abdominal discomfort	3 weeks before	CT	Aorta	Surgery, immuno- suppression	no	Recovery
Luebke (27)	M/67	Left knee and foot pain, fever	5 years after	US, CT	Superficial femoral	Surgery, immuno- suppression	no	Recovery
Unlu (28)	M/43	Abdominal pain	11 years after	US, CT	Aorta	Surgery, immuno- suppression	no	Recovery
Musuruana (29)	M/25	Ventricular tachycardia	Concomitant	Angiography	Coronary	Immuno- suppression	no	Recovery
Onodera (30)	F/22	No complaint	7 months after	MRI	Internal carotid	Endovascular	no	Recovery
Ohta (31)	M/38	Back pain, syncope	22 years after	CT	Aorta	Surgery, immuno- suppression	yes	Recovery
Present case	M/57	Abdominal pain	11 days after	CT, angiography	Accessory left gastric	Endovascular, surgery, immuno- suppression	yes	Recovery

NS, not stated; US, ultrasound; PD, pancreaticoduodenal

Some variant arteries have been reported around the superior mesenteric artery and celiac axis, which contains the left gastric artery, hepatic artery, and splenic artery.³³⁾ These vessels provide a rich blood supply to aid in the digestive process and protect the stomach and liver from potential ischemia or infarction. These variations result from diversity of embryonic development. Tandler³⁴⁾ stated that four primitive vitelline arteries arise from the dorsal abdominal aorta in the fetus and are interconnected through a ventral anastomotic channel. Regression or persistence of these vitelline segments or their ventral anastomosis causes the development of variation around the superior mesenteric artery and celiac axis.

Based on these reports, Song *et al.*³⁵⁾ hypothesized that an anatomic channel connects the left gastric artery and hepatic artery and that an identical embryonic remnant of this channel is the original form of an ALGA and an accessory or replaced left hepatic artery. Both arteries run through the fissure of the ligamentum venosum,³⁶⁾ supporting the hypothesis. Considering this hypothesis, we ligated not only the ALGA but also the left gastric artery to block multiple blood supplies to the aneurysms in the present case.

Treatment of aneurysmal vasculitis in patients with GPA should involve the combination of surgical or endovascular interventions with immunosuppressive agents such as high-dose prednisone, cyclophosphamide,³⁷⁾ and rituximab³⁸⁾ to prevent aneurysm rupture or control hemorrhage. Our patient was treated by both surgical and endovascular interventions combined with high-dose prednisone and cyclophosphamide, resulting in a good prognosis. Hybrid therapy combining both surgical and endovascular interventions for aneurysms in patients with GPA has never been reported, but such therapy would be a good approach especially for patients with ruptured aneurysms or aneurysms located on variant arteries, as in our case. Detecting the site of the rupture in a massive hematoma and recognizing anatomical blood supplies to aneurysms is sometimes difficult in open surgery and may be much more complicated in patients with variant arteries. In the present case, we failed to interrupt the blood flow to the aneurysms by an endovascular approach, but this approach was helpful to restrain the blood supply to the aneurysms, detect the site of rupture, and determine which artery to ligate in open surgery. In fact, we could immediately detect the aneurysms with the massive hematoma in the lesser omentum by touching the embolized coils.

In conclusion, we have presented a rare case of GPA complicated by aneurysm rupture in an ALGA. Aneurysm formation is a rare complication of GPA but can be life-threatening if rupture occurs. Immediate diagnosis and therapy should be performed for patients with symptoms of vascular ischemia or aortitis. Endovascular intervention is the first-choice therapy especially for hemodynamically stable patients with ruptured aneurysms or aneurysms located on variant arteries; notably, however, variant arteries may have multiple blood supplies. In the present case of aneurysm rupture in an ALGA, although we failed to interrupt the blood flow to the aneurysms by an endovascular approach, this approach was helpful to restrain the blood supply to the aneurysms, detect the site of rupture in a lesser omental hematoma, and determine the efficacy of ligating not only the ALGA but also the left gastric artery to block retrograde blood flow to the aneurysms during open surgery. This may have prevented postoperative bleeding or recurrence of the aneurysms.

CONFLICTS OF INTEREST

All authors certify that they have no personal financial or institutional interest in the subject matter, materials, or drugs in this article.

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