

CASE REPORT

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A case of internal trapping to a thrombosed giant rapidly growing aneurysm at the posterior cerebral artery

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ABSTRACT

We describe a case of internal trapping including the vasa vasorum for a thrombosed giant rapidly growing posterior cerebral artery aneurysm and performing a detailed analysis. A 48-year-old woman was followed up in our hospital for a thrombosed large posterior cerebral artery aneurysm located in the P2 segment. She initially presented after experiencing a sudden headache on two occasions. Head computed tomography and magnetic resonance imaging indicated a larger aneurysm than before. Digital subtraction angiography with balloon occlusion test was assessed, and internal trapping was sequentially conducted. We detected that the vasa vasorum originated from the posterior temporal artery. Therefore, we embolized the posterior temporal artery including the vasa vasorum using *N*-butyl-2-cyanoacrylate and Lipiodol. Next, the anterior temporal artery was embolized with *N*-butyl-2-cyanoacrylate and Lipiodol, posterior temporal artery P3 segment and the aneurysm and finally the proximal P2 segment were embolized with coils. Final vertebral and internal carotid angiography showed complete obliteration of the aneurysm. On the day after the procedure her paresis worsened and she developed left upper quadrantanopia, however was finally discharged with no hemiparesis. We reported a case of a rapidly growing thrombosed giant posterior cerebral artery aneurysm treated by parent artery occlusion including the vasa vasorum with detailed image analysis.

Keywords: thrombosed giant aneurysm, posterior cerebral aneurysm, parent artery occlusion, vasa vasorum

Abbreviations:

PCA: posterior cerebral artery

MRI: magnetic resonance imaging

BOT: balloon occlusion test

PAO: parent artery occlusion

NBCA: *N*-butyl-2-cyanoacrylate

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INTRODUCTION

Posterior cerebral artery (PCA) aneurysms are rare, accounting for 0.7%–2.3% of all intracranial aneurysms.¹⁻⁴ These aneurysms have specific morphological features and clinical findings that distinguish them from those occurring elsewhere in cerebral circulation.⁵ The surgical treatment of PCA aneurysms is complicated and associated with a high morbidity rate due to the complexity of the perforating branches from the PCA and their relationship with the cranial nerves and upper brainstem.⁶⁻⁹ In addition, a large or giant partially thrombosed aneurysm often has a wide neck or calcification and can become embedded deep in the parenchyma.¹⁰⁻¹¹ Some reports suggest that vasa vasorum of the aneurysm wall plays a key role in the growth of aneurysms with intra-aneurysmal thrombosis due to proliferation.

In the present case, we report a case in which internal trapping including the vasa vasorum was performed for a thrombosed giant rapidly growing aneurysm at the PCA with a detailed analysis.

CASE PRESENTATION

A 48-year-old woman was followed up for 20 years for a partially thrombosed large aneurysm of the right PCA with a maximum dome diameter of approximately 18 mm. The aneurysm size grew larger gradually. Specifically, as the aneurysmal component, both the previous thrombosed portion and the blood flow cavity portion got larger. She experienced sudden-onset headache on two occasions and was subsequently transported and admitted to our hospital. Neurological examination on admission demonstrated a Glasgow Coma Scale of G4V5M6 and left hemiparesis. No subarachnoid hemorrhage or intracranial hemorrhage was detected on head computed tomography or magnetic resonance imaging (MRI), but the aneurysm rapidly increased in size, reaching approximately 28 mm. The thrombosed part was on the lateral side (Fig. 1). Therefore, we performed urgent digital subtraction angiography and balloon occlusion test (BOT).

Digital subtraction angiography and BOT

Initially, we performed bilateral internal carotid angiography, right vertebral angiography (Fig.2) and three-dimensional rotational angiography. Next, we evaluated multiplanar reconstruction of three-dimensional rotational angiography images (Fig.2). Three-dimensional rotational angiography volume-rendering imaging revealed an aneurysm 28 mm in size in the P2 segment of the right PCA, which was located at the bifurcation to the P3–4 segments and temporal arteries (Fig.2). In addition, a detailed analysis showed that the small vessels arising from the posterior temporal artery were suspected to be vasa vasorum, which fed the thrombosed part (Fig.2) (Fig.3).

BOT was performed continuously. A 0.014-inch microguidewire (Xpedion, ev3 Endovascular, Inc., Plymouth, MN) was used to guide a 4 mm×7 mm HyperForm microballoon catheter (ev3 Neurovascular, Inc., Irvine, CA) into the right P2 segment, just proximal to the aneurysm, which was then occluded; bilateral internal carotid angiography and vertebral angiography were subsequently performed. The right posterior temporal artery and the parieto-occipital artery were shown in retrograde in the arterial phase. In addition, the right calcarine artery presented with faint retrograde filling in the venous phase (Fig. 2). Within 30 min of occlusion, the patient experienced sensory disturbance on the left side of the body and lips but not motor disturbance. We considered that symptom of occlusion of the thalamogeniculate artery arising from the P2 segment, just proximal to the aneurysm. Although we could not determine whether there was tolerance to occlusion, we performed internal trapping rather than coil embolization because of obtaining the reduction in aneurysmal size and complete obliteration.

Internal trapping to a thrombosed giant posterior cerebral artery aneurysm

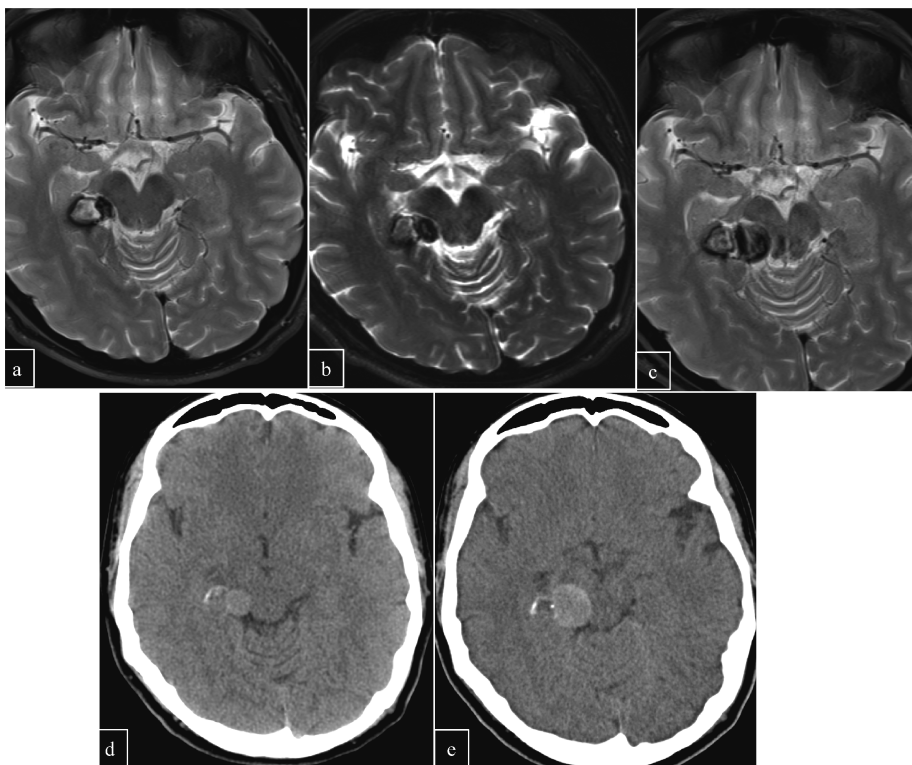


Fig. 1 MRI and CT images from 7 years ago until hospitalization for headache

Fig. 1a–c: T2-weighted MRI.

Fig. 1a: 7 years before admission. A large thrombosed aneurysm in the right PCA was 18 mm in size, and the thrombosed part was on the lateral side. The patient had no symptoms.

Fig. 1b: 2 years before admission. The aneurysm size grew larger gradually.

Fig. 1c: On admission, she had a sudden headache and rapid aneurysm growth, which was approximately 28 mm. Specifically, as the aneurysmal component, both the previous thrombosed portion and the blood flow cavity portion got larger.

No subarachnoid or intracerebral hemorrhage was noted.

Fig. 1d, e: Plain computed tomography.

Fig. 1d: 1 year before admission. The aneurysm size was 18 mm.

Fig. 1e: On admission, the aneurysm grew rapidly.

MRI: magnetic resonance image, PCA: posterior cerebral artery.

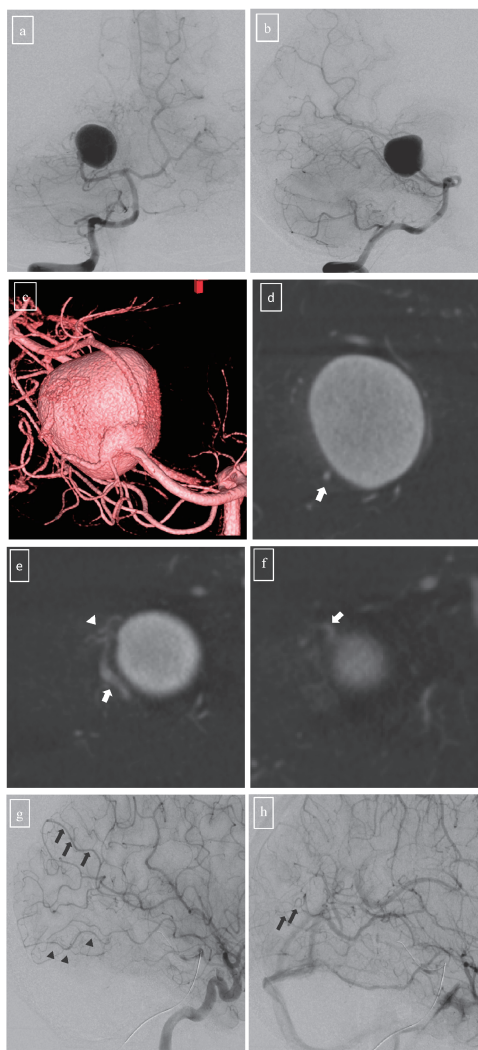


Fig. 2 VAG and BOT images on admission

Fig. 2ab: The aneurysm was 28 mm and located in the P2 segment of the right PCA at the bifurcation to the P3–4 segments and the temporal arteries.

Fig. 2a: AP view of right VAG.

Fig. 2b: Lateral view of right VAG.

Fig. 2c: VR image of right vertebral 3D-RA.

Fig. 2d-f: Coronal view of the MPR images on right vertebral 3D-RA.

From a to d, front to back view, we suspected that the small vessels arising from the posterior temporal artery were suspected to be vasa vasorum, which fed the thrombosed part and subsequently the posterior temporal artery was formed once and ran backward.

Arrow: posterior temporal artery, Arrowhead: small vessel suspected of being vasa vasorum

Fig. 2gh: These images are from the balloon occlusion test.

Fig. 2g: Right ICAG in arterial phase lateral view on BOT. A parieto-occipital artery and posterior temporal artery were demonstrated in retrograde.

Black arrow: parieto-occipital artery, Black arrowhead: posterior temporal artery.

Fig. 2h: Right ICAG in venous phase lateral view on BOT. A calcarine artery was demonstrated faintly in retrograde.

Black double-headed arrow: calcarine artery, PCA: posterior cerebral artery, AP: antero-posterior,

MPR: multiplanar reconstruction, 3D-RA: 3-dimensional rotational angiography, VAG: vertebral angiography,

VR: volume-rendering, BOT: balloon occlusion test, ICAG: internal carotid angiography.

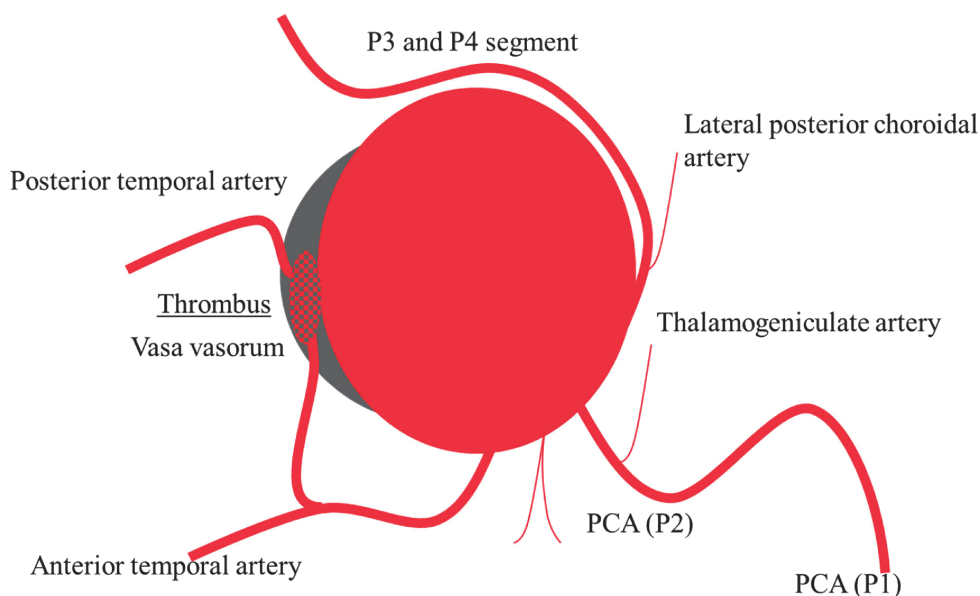


Fig. 3 A schema: 3D-RA VR image of VAG

Small vessels arising from the posterior temporal artery were vasa vasorum, which fed the thrombosed part. The aneurysm was located at the bifurcation to the P3–4 segments and the temporal arteries. A thalamogeniculate artery arose from the P2 segment, just proximal to the aneurysm.

3D-RA: 3-dimensional rotational angiography, VAG: vertebral angiography, VR: volume-rendering.

Endovascular treatment

The patient was diagnosed with a partially thrombosed giant P2 aneurysm, and we performed internal trapping. Considering the presence of the vasa vasorum arising from the posterior temporal artery, we performed internal trapping including the vasa vasorum. The vasa vasorum fed the thrombus, therefore, we considered that it possibly caused the aneurysm to continue growing.

Embolization was performed as the treatment strategy in the following order: (1) posterior temporal artery and vasa vasorum embolization using a mixture of *N*-butyl-2-cyanoacrylate (NBCA) (Histoacryl; B. Braun Melsungen AG, Melsungen, Germany) and Lipiodol (Lipiodol Ultra Fluide; Guerbet, Roissy, France) (2) anterior temporal artery embolization using a mixture of NBCA and Lipiodol and (3) coil embolization of the P3 segment and (4) intraneurysmal coil embolization and proximal P2 occlusion.

Under local anaesthesia, a 4-Fr diagnostic catheter was placed in the left vertebral artery, and a 7-Fr Roadmaster 90 cm (GOODMAN, Aichi, Japan) guiding catheter was advanced into the right vertebral artery.

[Posterior temporal artery and vasa vasorum embolization] (Fig.4)

Using a 0.014-inch ASAHI CHIKAI microguidewire (Asahi Intecc Co., Ltd., Aichi, Japan), an Excelsior SL-10 microcatheter (Stryker Neurovascular, CA, USA) was advanced into the P2 segment, followed by a Tactics 3.4-Fr catheter (Technocrat Corporation, Aichi, Japan) advanced coaxially into the PCA P1 segment. Embolization including the vasa vasorum was performed using a 25% mixture of NBCA and Lipiodol. Good penetration to the vasa vasorum and posterior temporal artery was achieved.

[Anterior temporal artery embolization] (Fig.4)

Similarly, an Excelsior SL-10 microcatheter was advanced into the origin of the anterior temporal artery and embolization was performed using the same mixture of NBCA and Lipiodol. Good penetration was achieved and anterior temporal artery was embolization including the origin.

[PCA P3 segment embolization] (Fig.4)

In addition, an Excelsior SL-10 microcatheter was advanced into the P3 segment using a 0.014-inch GT microguidewire with a double angle tip (Terumo Corporation, Tokyo, Japan) and coil embolization was performed using a total of 11cm coils.

[Intra-aneurysmal and proximal P2 segment embolization] (Fig.5)

Likewise, an Excelsior SL-10 microcatheter was advanced into the intra-aneurysm using a 0.014-inch ASAHI CHIKAI microguidewire. We performed rough packing with 189cm coils and the P2 segment was embolized with 17cm coils to prevent occlusion of the thalamogeniculate artery (Fig.5).

Finally, vertebral angiography did not indicate an aneurysm, and internal carotid angiography identified the parieto-occipital artery and posterior temporal artery in the delayed phase. (Fig.5)

Post-procedure clinical course

The day after the procedure, left paresis worsened, and left upper quadrantanopia developed. MRI showed no change in the size of the aneurysm, and subarachnoid hemorrhage was not detected. However, cerebral infarction developed in the right temporal lobe, and midbrain edema was noted, which appeared to exacerbate the left hemiparesis. After conservative treatment, her paresis fully recovered but the quadrantanopia remained. On post-procedure day 10, she was discharged without paresis. MRI findings at 2-month and 1-year follow-up revealed the disappearance of the edema and a decreased aneurysm size. The high signal intensity of the aneurysm at 2 months appeared to indicate thrombosis, and iso-intensity at 1 year appeared to show an old change of aneurysm. The aneurysm appeared to be embolized completely (Fig.5).

DISCUSSION

PCA aneurysms are rather rare, accounting for 0.7%–2.3% of all intracranial aneurysms.¹⁻⁴ In addition, 49% of PCA aneurysms are large, and 45% are located at the P2 segment.¹²

In the treatment of PCA aneurysms, Xuanfeng et al¹² reported parent artery occlusion (PAO) in 75% of P2 or distal segment fusiform/dissecting aneurysms, resulting in 88% complete occlusions. However, they also reported that cerebral infarctions occurred as a complication of PAO to PCA aneurysms in 10% of cases, with the primary symptoms being hemiparesis and hemianopsia. Likewise, several authors have reported hemianopsia in 5%–10% of cases.¹³⁻¹⁵

Although BOT can be used to prevent such cerebral infarctions, its value in evaluating tolerance to PAO remains debatable. Previous studies have reported both false-positive and -negative test results. Moreover, the use of BOT in the tortuous PCA is technically challenging, thus increasing the risk of procedure-related complications. In our case, BOT was performed; however, tolerance to PAO was not clarified. Although we were afraid to cause PCA territory infarcts, we performed PAO and coil embolization because of obtaining the reduction in aneurysmal size and prevent recanalization. Although it was safer to perform coil embolization with stent, we chose PAO hoping it more shrinking because symptom of mass effect is severe and aneurysm pressed the brainstem. As a result, cerebral infarction was induced, and BOT might have yielded a false-negative result. On the basis of the risk of false-negative and -positive results, BOT is not always recommended for PCA aneurysms. Certainly, occipital artery – posterior cerebral artery bypass was considered to prevent infarction before internal trapping. However, the procedure was

Internal trapping to a thrombosed giant posterior cerebral artery aneurysm

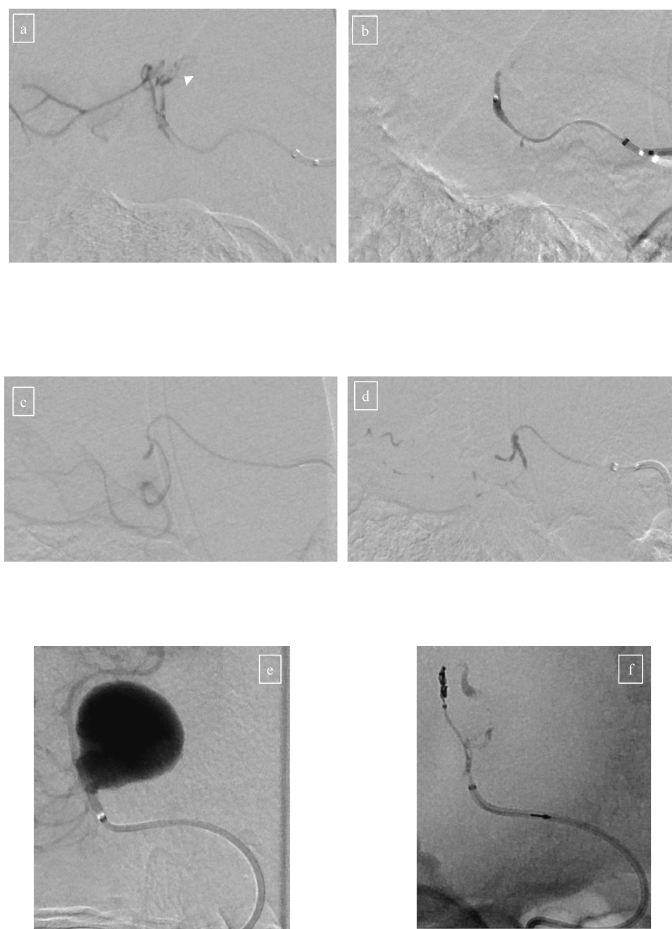


Fig. 4 Embolization of the posterior temporal artery including vasa vasorum, anterior temporal artery, and P3 segment.

Fig. 4ab: Embolization of the posterior temporal artery and vasa vasorum using a mixture of *N*-butyl-2-cyanoacrylate and Lipiodol.

Fig. 4a: Lateral view of angiography of right posterior temporal artery. Small vessels resembling vasa vasorum arising from the posterior temporal artery and feeding the thrombosed part of the aneurysm. Arrow head: vasa vasorum.

Fig. 4b: The vasa vasorum and posterior temporal artery were embolized using a 25% mixture of *N*-butyl-2-cyanoacrylate and Lipiodol.

Fig. 4cd: Anterior temporal artery embolization.

Fig. 4c: Lateral view of angiography of right anterior temporal artery. An Excelsior SL-10 microcatheter was advanced into the origin of the anterior temporal artery.

Fig. 4d: Anterior temporal artery embolization was performed using a 25% mixture of NBCA and Lipiodol.

Fig. 4ef: P3 segment embolization.

Fig. 4e: Anteroposterior view of the posterior cerebral angiography before embolization. An Excelsior SL-10 was advanced into the P3 segment using a 0.012 GT microguidewire with a double angle tip.

Fig. 4f: Coil embolization was performed using a total of 11cm coils.

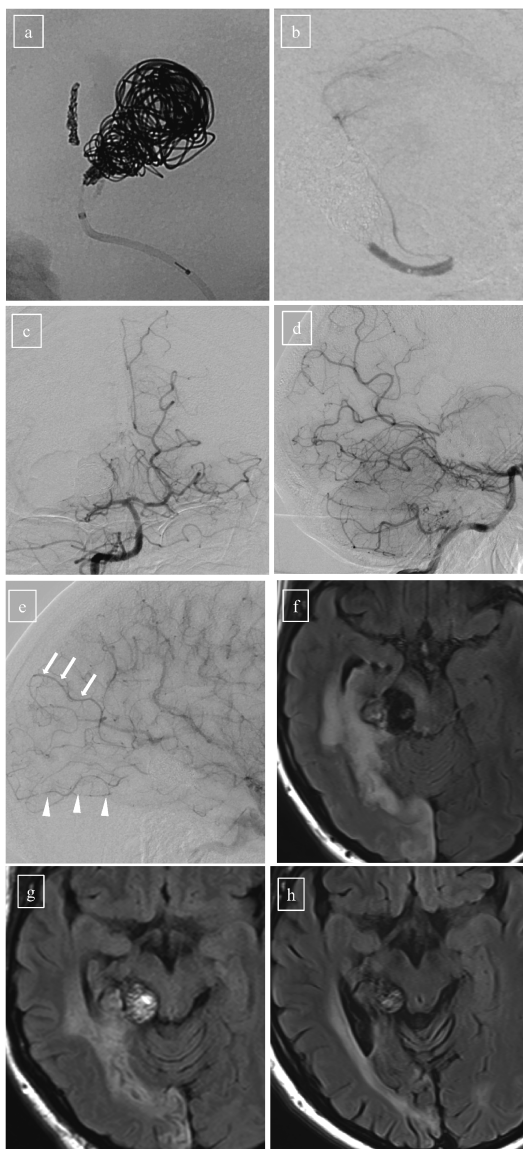


Fig. 5 Embolization of intra-aneurysm and proximal P2 segment

Fig. 5a: The aneurysm was roughly packed with 189cm coils, and the proximal P2 segment was embolized using 17cm coils to prevent occlusion of the thalamogeniculate artery.

Fig. 5b: Image after aneurysm and proximal P2 segment embolization, indicating a thalamogeniculate artery.

Fig. 5c: Final right vertebral angiography, anteroposterior view.

Fig. 5d: Final right vertebral angiography, lateral view.

Fig. 5e: Final right internal carotid angiography, lateral view, which indicated a parieto-occipital artery and a posterior temporal artery on delayed phase.

Arrow: Parieto-occipital artery, Arrow head: Posterior temporal artery.

Fig. 5f-h: Fluid-attenuated inversion recovery image.

Fig. 5f: 2 days after the procedure. No change in aneurysm size was seen, and subarachnoid hemorrhage was not detected.

However, cerebral infarction developed in the right temporal lobe, and midbrain edema was noted.

Fig. 5g: 2 months after the procedure.

Fig. 5h: 1 year after the procedure. Midbrain edema disappeared, and the aneurysm size decreased.

The high signal intensity of the aneurysm at 2 months indicated thrombosis and iso-intensity at 1 year demonstrated

challenging and we did not choose it considering the condition of the patient.

Partially thrombosed aneurysms are a diverse collection of complex aneurysms with an organized intraluminal thrombus.¹⁶ They might grow normally and exhibit new neurological symptoms related to the mass effect and subarachnoid hemorrhage due to rupture.¹⁷ In addition to conventional clipping, neurosurgical approaches frequently involve thrombectomy with clip reconstruction and are commonly associated with complications. Bypass surgery may generate good outcomes but is not feasible in all cases, particularly in those involving posterior circulation. However, although the surgical management of these aneurysms is complex, endovascular treatment with proximal vessel occlusion or selective coil embolization, with or without a stent, is generally straightforward.¹⁶

The recanalization rate after coil embolization was reported to be 30%–77.8%, whereas the complete obliteration rate after PAO was 77%–100%.^{18–21} In fact, PAO is typically considered the first-line treatment for thrombosed large or giant aneurysms. However, cases in which the aneurysm continued to grow after PAO have been reported, attributed to vascular channel involvement and vasa vasorum.^{16,22,23} Iihara et al²² described a partially thrombosed giant vertebral artery aneurysm initially treated with PAO using coils; however, the aneurysm continued to grow after treatment, although angiography did not give evidence of an aneurysm and parent artery filling. Then, the same author reported a marked development of the vasa vasorum on the occluded VA and the neck of the aneurysm, and it was visualized at the time of surgery. Moreover, when the occluded vertebral artery was cut, blood oozed through the coils packed within its lumen on the aneurysm side; therefore, the vasa vasorum could be a potential source of recanalization of a thrombosed aneurysm.²² The present case noted the vasa vasorum arising from the posterior temporal artery, which supplied blood to the thrombosed segment of the aneurysm. Therefore, we embolized the vasa vasorum in addition to the parent artery. Certainly, this aneurysmal major growing part was vessel cavity of the component and the effect of embolizing vasa vasorum is debatable. However, we also feared of the possibility of the growth after PAO. As a result, we considered embolization of the vasa vasorum might cause the aneurysm to shrink.

CONCLUSION

We reported a case of a rapidly growing thrombosed giant posterior cerebral artery aneurysm treated by parent artery occlusion including the vasa vasorum with detailed image analysis.

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CONFLICT OF INTEREST

Declaration of interest: none.

This summary statement ultimately will be published if the article is accepted.

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