A Ruptured Large Thrombosed True Posterior Communicating Artery Aneurysm Treated with Endovascular Treatment Three Times

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Objective: We report a case of ruptured large thrombosed true posterior communicating artery (PCoA) aneurysm and consider its treatment.

Case Presentation: A 71-year-old male patient had a left ruptured large thrombosed true PCoA aneurysm (maximum diameter 23 mm) with a small neck. Intra-aneurysmal coil embolization via the internal carotid artery was performed to preserve the premammillary artery (PMA). The adjunctive technique could not be used because the diameter of the PCoA was 1.5 mm. The result was a neck remnant and the aneurysm was recanalized. After 14 months, similar treatment was performed, and the aneurysm was recanalized again. The acute and twisted angle of the PCoA origin and the thinness of the PCoA were considered as factors for incomplete embolization. Because the distance between the origin of the PMA and aneurysmal neck was 5 mm, short-segment internal trapping of the aneurysm was performed 13 months after the second embolization. As a result, the PMA was no longer visualized on DSA; however, he had no neurologic deficit. The aneurysm remained obliterated after 7 months.

Conclusion: Making a tight intra-aneurysmal coil embolization of a large thrombosed true PCoA aneurysm is difficult. If there is a certain distance between the PMA and the aneurysm neck, short-segment internal trapping might be useful to treat it.

Keywords ▶ true posterior communicating artery aneurysm, large and giant aneurysm, thrombosed aneurysm, parent artery occlusion, premammillary artery

Introduction

Posterior communicating artery (PCoA) aneurysms can occur at the junction of the internal carotid artery (ICA) or PCoA itself. The latter are called true PCoA aneurysms. Their treatment is difficult because their parent artery is small and has important perforators. We report a case of large thrombosed ruptured true PCoA aneurysm, which was treated by intra-aneurysmal coiling twice, but was recanalized each time, and successfully treated by using short-segment internal trapping.

Case Presentation

A 71-year-old male patient with histories of diabetes mellitus, hypertension, hyperlipidemia, and smoking 20 cigarettes per day for 50 years experienced sudden headache and vomiting while he was working. He was misdiagnosed as having cold and was treated but did not improve. Two days later, MRI revealed a subarachnoid hemorrhage (SAH: Hunt and Kosnik grade II, World Federation of Neurosurgical Societies grade I) caused by a ruptured large thrombosed aneurysm (maximum diameter 23 mm) on the left anterior surface of the brainstem at the level of between the lower midbrain and upper pons (Fig. 1A–1C).
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Therefore, he was transferred to our hospital and underwent DSA on hospital day 1. Left internal carotid angiography (ICAG) revealed an irregular-shaped aneurysm arising from the left fetal-type PCoA itself (Fig. 1D). Left vertebral angiography (VAG) showed left posterior inferior cerebellar artery (PICA), but not left PCoA. Left VAG with ipsilateral carotid compression showed left PCoA and a small residual lumen of the thrombosed aneurysm (Fig. 1E). Therefore, we presumed that the aneurysm was mostly thrombosed and awaited conservative therapy. Repeat DSA on hospital day 22 revealed an enlarged non-thrombosed space. 3D-rotational angiography (3D-RA) fusion imaging with left ICAG and left VAG with carotid compression effectively identified the PCoA-P1 junction (Fig. 2A and 2B). The length of the left PCoA was approximately 33 mm, whereas the diameter of the left PCoA near the aneurysmal neck was 1.5 mm. The position of the aneurysm was closer to the PCA, and the distance between the aneurysm neck and the PCA was 1.5 mm. The size of the non-thrombosed aneurysmal space was $7.0 \times 6.1 \times 5.2$ mm, and the neck size was $2.7 \times 2.3$ mm.

To treat the aneurysm with the preservation of PCoA perforators, we considered either direct neck clipping or intra-aneurysmal embolization. Direct clipping is a reliable and radical treatment, but it would be difficult because of the small and deep operative field due to the large size and low mobility of the aneurysm. Hence, we considered the approach route to the aneurysm for intravascular treatment. First, we considered using the approach of the posterior circulation because the aneurysm was very close to PCoA–P1 junction. However, the dominant left VA origin was tortuous, and the non-dominant right VA was very thin after the branching of the posterior inferior cerebellar artery. In contrast, the aneurysm was well shown in the left ICA angiography; hence, we selected the intra-aneurysmal embolization using the approach of the anterior circulation.

The first coil embolization of the aneurysm was performed on hospital day 29. No antiplatelet drug was provided before embolization. With the patient under general anesthesia, a 6 Fr catheter (Roadmaster; Goodman, Aichi, Japan) was placed in the left ICA via the right femoral artery. We attempted to guide a pre-shaped angle 45 degrees microcatheter (Headway 17; MicroVention Terumo, Tustin, CA, USA) to the left PCoA by using a micro guidewire (Transend 0.014 Soft Tip; Stryker, Kalamazoo, MI, USA).
The derivation of the microcatheter was difficult due to the acute and twisted angle of the PCoA origin. We planned to use a double catheter technique, but the PCoA was too thin to insert an additional microcatheter. We used a 4 × 12 mm coil (Orbit Galaxy Complex Fill; Codman & Shurtleff, Johnson & Johnson, Raynham, MA, USA) to make a coil frame and filled it with another nine coils (total 43 cm). At the end of the first treatment, the residual space around the neck was very small (Fig. 2C and 2D). We did not administer an antithrombotic drug after treatment.

However, regular follow-up X-ray revealed deformation of the coil mass, and the second coil embolization was performed 14 months after the first procedure. We administered 75 mg clopidogrel and 100 mg of aspirin once daily from 1 week before embolization. The recanalized space was 6.6 × 6.2 × 4.6 mm (Fig. 3A–3C). Its size was almost the same as that before the first procedure. To improve the trackability and stability of the microcatheter, we placed a 6 Fr CGuiding Sheath (Asahi Intecc, Aichi, Japan) into the left ICA. The 6 Fr Cerulean DD6 catheter (Medikit, Tokyo, Japan) was inserted coaxially; however, the blood flow was stagnated. Therefore, we exchanged the DD6 catheter with a 4 Fr Cerulean G catheter (Medikit) and derived it to the carotid siphon. After a Headway 17 microcatheter (steam shaped to 45 degrees) was placed in the aneurysm, a 4 mm × 8 cm coil (HydroFrame 10; MicroVention Terumo) was used to make a coil frame. Subsequently, six fine coils (total 18 cm) were packed into the aneurysm, then the catheter deviated outside the aneurysm, and we were unable to put it back into the aneurysm again. The final angiography showed a small residual space along the aneurysmal wall (Fig. 3D–3F). After the second session, clopidogrel alone was administered for 3 weeks.

Subsequent regular follow-up X-ray revealed deformation of the coil mass again. We performed diagnostic DSA 12 months after the second session, and the recanalized space was 5.1 × 6.1 × 3.5 mm on DSA (Fig. 4A). The distance between the pre mammillary artery (PMA) and the aneurysmal neck was 5 mm; therefore, we decided to perform short-segment internal trapping of the aneurysm with preservation of PMA. The third endovascular procedure was performed a month after the previous DSA (13 months after the second embolization). We again administered 75 mg clopidogrel and 100 mg of aspirin once daily from 1 week before embolization. The recanalized space rapidly enlarged to 10.0 × 8.4 × 4.2 mm (Fig. 4B). We guided a 6 Fr FUBUKI Guiding Sheath (Asahi Intecc) and 6 Fr Cerulean DD6 catheter coaxially into the left ICA. The 3.4 Fr catheter (Tactics; Technocrat Corporation, Aichi, Japan) was guided to the carotid siphon coaxially. After an Excelsior SL-10...
A true PCoA aneurysm originates from the PCoA itself. He et al.\(^1\) retrospectively reviewed 46 published studies of true PCoA aneurysms (all but three were small case reports) and true PCoA aneurysms represented approximately 1.3% of all intracranial aneurysms and 6.8% of all PCoA aneurysms. The natural history of true PCoA aneurysms is unclear. In a large study comparing 67 junctional and 10 true PCoA aneurysms, aneurysm size was statistically larger in the junctional aneurysms; however, the prevalence of ruptured aneurysms was similar in both groups.\(^2\) Therefore, true PCoA aneurysms have been presumed to be a higher risk of rupture at small sizes compared with junctional PCoA aneurysms. However, three giant true PCoA aneurysms and less than 10 cases of large true PCoA aneurysms, including our case, have been reported.\(^3\)–\(^7\) Before the treatment of true PCoA aneurysms, it is necessary to evaluate the distance between the neck of them and ICA or PCA. Most true PCoA aneurysms arise within 2–3 mm from the origin of the PCoA near the ICA.\(^2\) Koga et al.\(^8\) reviewed 17 cases of true PCoA aneurysms, and 9 arose within the anterior, 5 within the middle, and 3 within posterior thirds of the PCoA. Exceptionally, the aneurysm neck in our case was very close to the P1–P2 junction. The microcatheter (Stryker; steam shaped to 90 degrees) was placed in the aneurysm, a 9 mm × 31 cm coil (HydroFrame 18; MicroVention Terumo) was used to make a coil frame. Subsequently, 13 coils (total 121 cm) were inserted in the aneurysm, and then, the tip of the microcatheter was placed out of the aneurysm (Fig. 4C). Subsequently, two thin coils (total 6 cm) were inserted in the PCoA at the aneurysm neck. After the next very small coil was inserted, the left PCoA was not contrasted; therefore, we removed the last coil. We kept his activated clotting time at ≥250 seconds and injected 60000-unit urokinase from the microcatheter. DSA injection via the microcatheter within the PCoA showed the PCoA proximal to the aneurysm (Fig. 4D); however, DSA injection via the Tactics catheter did not show the PCoA (Fig. 4E). The PMA was no longer visualized on DSA; however, we considered that the PCoA and PMA flows might remain. After embolization, the patient emerged from anesthesia quickly with no neurologic deficit. Argatroban was injected continuously for 4 days. MRI conducted the next day did not show cerebral infarction (Fig. 4F). Both antiplatelet drugs were administered for a month, and after that only clopidogrel was continued. He returned to work one month later. Seven months later, DSA was performed, and left ICAG and left VAG did not show the PCoA and the aneurysm.
Treatment of a Ruptured Large Thrombosed True PCoA Aneurysm

In treating true PCoA aneurysms, preserving the perforators of the PCoA is key to success. The anterior thalamoperforating arteries (ATPAs) are almost synonyms of perforators arising from the PCoA. According to several cadaveric studies, the number of ATPAs was 1 to 14 (average, approximately 5–7). Vicentelli et al. divided the PCoA perforators into inner and outer groups. The former consisted of 3–11 tiny branches supplying the hypothalamus and the latter consisted of 2–7 branches. These perforators do not arise from PCoA at equal intervals. Saeki et al. reported that more branches arose on the anterior half of the PCoA in 54%, the posterior half in 25%, and equal halves in 21% of cases. Kim et al. examined 80 cadaveric PCoAs, dividing them into three parts, and found that the number of perforators arising from each part was 2.66, 3.03, and 1.67, respectively. Beumer et al. examined the length of the perforator-free zone (PFZ) of the PCoA, and 80% of the longest PFZs were located closer to the PCA than to the ICA. In general, more ATPAs arose on the anterior half of PCoA. The most common perfusion areas of ATPAs are the posterior hypothalamus, anterior thalamus, subthalamus, optic tract, tuber cinereum, mammillary body, posterior limb of the internal capsule, and cerebral peduncle. Damage of ATPAs can lead to amnesia, subcortical cognitive disturbances, endocrine dysfunction, visual field defects, vegetative disorders, hyperthermia, motor weakness, and sensory and personality changes.

The largest ATPA terminates between the mammillary bodies and the optic tract, such as the premammillary area, and the perforator is called the PMA or thalamotuberal artery. Rarely, the PMA originated from the P1 or P2 segment. The PMA is not always single. Some reports showed double or triple PMAs. Gibo et al. reported fusion 3D-RA image of the ICAG and VAG was very useful to evaluate the complex anatomical structures around the aneurysm and identify the P1–P2 junction.

Fig. 4 (A) Left ICAG, 1 month before the third embolization shows the recanalization of the aneurysm. The distance between the PMA (arrowheads) and aneurysmal neck was 5 mm. (B) At the third embolization, initial left ICAG shows the rapid enlargement of the recanalized space. (C) Intraoperative left ICAG shows that the aneurysm was almost occluded with 14 coils. (D) After 16 coils were inserted, DSA injection via the microcatheter within the PCoA showed the PCoA proximal to the aneurysm. (E) After 16 coils were inserted, DSA injection via the Tactics catheter did not show the PCoA. (F) DWI performed the day after treatment reveals no acute cerebral infarction. DWI: diffusion weighted image; ICAG: internal carotid artery angiography; PCoA: posterior communicating artery; PMA: premammillary artery.
that the PMA tends to arise on the posterior half of the PCoA. Other cadaveric studies reported that almost half of PMAs tend to arise on the middle third of the PCoA.\textsuperscript{9,13,16} The PMA supplies very important regions of the diencephalon (posterior hypothalamus, subthalamus and anterior thalamus), mammillothalamic tract, as well as certain parts of the rostral brainstem and occasionally anteromedial part of the optic tract, head of the caudate nucleus, genu, and part of the posterior limb of the internal capsule.\textsuperscript{14,16} Occlusion of the PMA can lead to contralateral motor weakness, changes in the superficial modalities of sensation, apathy, lack of spontaneity, disorientation, memory disturbances, and thalamic aphasia.\textsuperscript{14,16}

However, PCoA occlusion did not always cause cerebral infarction. Such cases were thought to have abundant anastomosis between ATPAs and neighboring arteries. In a detailed report on PMA anastomosis, anastomoses involving the PMA were present in 35.9\% of the cases.\textsuperscript{14} The anastomotic channels may interconnect the branches of the PMA itself, or they may connect the PMA and its branches to adjacent vessels. Rich anastomosis with perforating branches of the PCoA, AChA, and PCA may be present.\textsuperscript{14}\textsuperscript{Kim et al.}\textsuperscript{19} reported a case with anastomoses between the PMA and perforators from the PCA and from the posterior segment of the PCoA. He also reported on three cases with an anastomosis between perforators from the middle segment of the PCoA.

Therefore, the treatment of true PCoA aneurysms is as follows: Surgical therapy includes neck clipping, proximal clipping, and trapping, whereas interventional therapy includes intra-aneurysmal coil embolization and internal trapping. Only surgical treatment was performed in early cases; however, technical advances achieved in endovascular therapy have permitted the successful treatment of true PCoA aneurysms.\textsuperscript{5,6} To preserve perforators of PCoA, direct clipping or intra-aneurysmal embolization is initially considered. In our case, we selected embolization as the therapy, and we found two disadvantages of the therapy. One was the acute and twisted angle of PCoA origin from ICA. That did not allow appropriate stability of the microcatheter for tight packing and resulted in difficulty of microcatheter reinsertion when the microcatheter went out of the aneurysm. Another was the thinness of PCoA as a parent artery. Stent-assisted coiling is good at preserving perforators and making tight embolization; however, the smallest stent available for cerebral aneurysm in Japan can be used in cerebral arteries of \geq2.0\ mm in caliber. The PCoA in our case had a loop, and the PCoA diameter around the aneurysm was 1.5 mm; therefore, we could not use a stent. The double catheter technique was also unable to be performed due to the small caliber of the PCoA. In contrast, it is an advantage that direct clipping is able to confirm the preservation of PCoA visually. If the aneurysm is small, direct clipping might be better than embolization.

If the PCA will have to be sacrificed, the point and length of occlusion are important. If the PCoA is obliterated near the P1–P2 segment, ATPAs including PMA will presume to be preserved. However, if the PCoA is obliterated near the ICA, the reverse flow of the PCoA from the PCA will perfuse ATPAs, and the diameter of the P1 segment is important.\textsuperscript{17} A super-selective balloon test occlusion of the PCoA is seemed to be considered.\textsuperscript{18} In our case, the aneurysmal neck was small, very close to the P1–P2 segment, and more than 5 mm away from PMA; therefore, we planned a short-segment internal trapping as a third session. At the end of the treatment, angiography via the microcatheter within left PCoA visualized the PCoA; hence, the PCoA was patent at that time. Consequently, the PMA became invisible on DSA, but the patient experienced no neurologic deficit. In the large review by He\textsuperscript{15}, only 2 of 7 cases trapped by craniotomy had neurologic deficits. In a recent report, a ruptured true PCoA aneurysm was treated using short-segment interventional parent artery occlusion, and the postoperative course was uneventful.\textsuperscript{19} As a matter of course, we must make efforts to preserve PMA, but not a few persons might have enough anastomotic flow between PMA and AChA and/or PCA.

In all cases, surgical treatment of true PCoA aneurysms \geq20\ mm would be rather difficult. Among the three aforementioned giant true PCoA aneurysms, one was a fusiform type with a diameter of 26 mm, and five fenestrated clips were used for neck-plastic clipping\textsuperscript{20}; one was a saccular type with a diameter of 25 mm, but the patient refused treatment\textsuperscript{21}; and one was a saccular type with a diameter of 35 mm treated with trapping and thrombectomy of the aneurysm.\textsuperscript{7} And our case was treated with endovascular procedure three times. Fortunately, all the treated cases recovered, treatment of these aneurysms is still challenging.

**Conclusion**

We experienced a case of ruptured large thrombosed true PCoA aneurysm. To preserve ATPAs definitively, we attempted intra-aneurysmal coil embolization twice; however, the aneurysm recanalized both times. The acute and
twisted angle of the PCoA origin and the thinness of the PCoA were considered as factors for incomplete embolization. For the third treatment, we performed short-segment internal trapping without any complication. Although it was difficult to prove by vascular imaging, two factors were presumed for successful treatment. First, the aneurysmal neck was very close to P1–P2 junction, and the distance between the aneurysmal neck and the PMA origin was enough for preservation of the PMA. Second, the potential anastomosis among perforating branches of the PCoA, AChA, and PCA was abound.

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Neither the first author nor any of the coauthors have any conflicts of interest.

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