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Case Report

A Recurrent Large Posterior Cerebral Artery Aneurysm Successfully Treated with Parent Artery Occlusion Using Somatosensory-Evoked Potential: A Case Report

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Objective: Treatment of large posterior cerebral artery (PCA) aneurysm involving the P1–P2 segment is difficult by both neurosurgery and endovascular treatment. Balloon occlusion test (BOT) to identify precise peripheral collateral flow is difficult prior to parent artery occlusion (PAO). Besides, PAO at the aneurysm at this location can cause peripheral cortical infarction of the occipital and temporal lobes and/or perforator infarction involving the midbrain and thalamus perfused by the perforating artery arising from the P1–P2 segment. However, detection of the perforator during PAO is difficult.

Case Presentation: The patient was a 49-year-old woman. At the age of 43 years, a right large PCA aneurysm was discovered in the right P1–P2 segment. A simple technique coiling was performed. As recurrence was identified 1 year later, embolization was performed using a same procedure. Since further recurrences were later found, a third round of treatment was planned. Somatosensory-evoked potential (SEP) was recorded as intraoperative electrophysiological monitoring. Tortuosity of the right PCA was observed at the aneurysm neck and the distal right PCA could not be secured. We could neither perform stent-assisted coil embolization nor BOT in the right PCA. Hence, we inflated the balloon in the basilar artery and checked the collateral circulation routes retrograde into the right PCA from the right middle cerebral artery via a leptomeningeal anastomosis. PAO was performed on the right P1–P2 segment at the aneurysm neck. The signal of the SEP was not decreased, and the aneurysm was not visualized. Another coil was added to strengthen the PAO to the right P1 segment, which decreased the SEP amplitude in the extremities by 3 minutes after. As the last coil was thought to be occluding the perforator branching from the right P1 segment, it was removed without detaching. The SEP amplitude began to improve and recovered by 9 minutes after. There was no postoperative deficit. No recurrence of aneurysm was observed on MRA 9 months postoperatively.

Conclusion: During PAO at the P1 segment of large PCA aneurysm involving the P1–P2 segment, SEP may be helpful to prevent perforator infarction, even if perforating artery originating from the proximal portion of the aneurysm was not detected by angiography.

Keywords aneurysm, intraoperative electrophysiological monitoring, parent artery occlusion, posterior cerebral artery, somatosensory-evoked potential

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Introduction

Treatment of large posterior cerebral artery (PCA) aneurysm involving the P1–P2 segment is difficult by both neurosurgery and endovascular therapy.[1–3] By endovascular treatment, stent-assisted coil embolization (SAC) can preserve peripheral and perforator blood supply.[2,4–6] However, in case having vessel tortuosity or access difficulty due to previously embolized coil visualization in a recurrent case, SAC is difficult. SAC requires long-term antiplatelet therapy, and it can be recurrent.

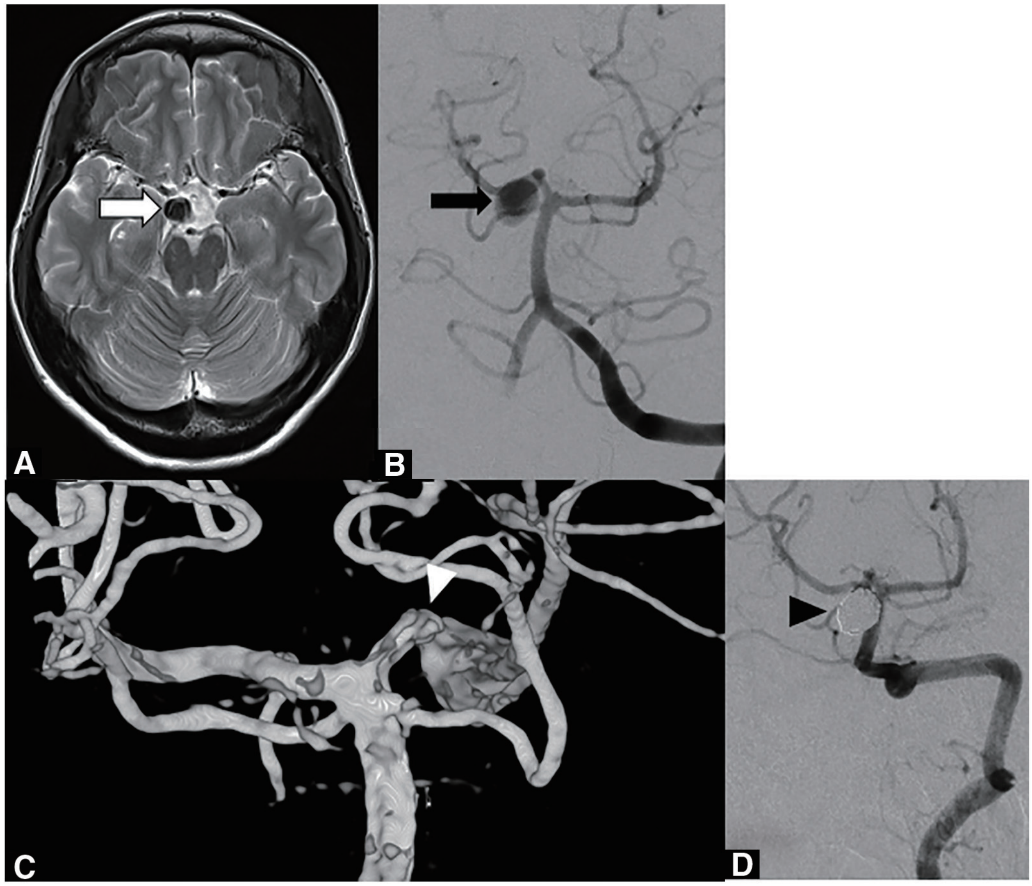

Fig. 1 (**A**) A right large PCA aneurysm with a maximum diameter of 13 mm was noted in the right P1–P2 segment on preoperative T2-weighted MRI (axial plane) (white arrow). (**B**) Preoperative angiogram (frontal view) of the left VA. Preoperative aneurysm (black arrow). (**C**) Preoperative 3D-DSA (posterior view). A perforator branching from the right P1 segment was noted (white arrowhead). (**D**) Postoperative angiogram (frontal view) of the left VA. Intra-aneurysmal coil embolization was performed using a simple technique. Postoperative aneurysm (black arrowhead). PCA: posterior cerebral artery; VA: vertebral artery

Balloon occlusion test (BOT) can determine therapeutic strategy of the distal PCA aneurysm (distal to the P2 segment). BOT is standard to evaluate peripheral collateral flow prior to occlusion of proximal aneurysmal position, and parent artery occlusion (PAO) to the aneurysm has low complication rate for hemianopia.[2,7–10] However, BOT to determine therapeutic strategy for the proximal PCA aneurysm (P1 segment and P1–P2 segment involved type) cannot be evaluated precisely because of the arterial anatomical complexities where BOT at the P1–P2 segment is difficult,[4] and BOT at the basilar artery (BA) is affected by the collateral flow from bilateral posterior communicating arteries (PComAs).

PAO for proximal PCA aneurysm has the risk of peripheral cortical infarction in the occipital and temporal lobes and/or perforator infarction involving the midbrain and thalamus perfused by the perforating artery arising from the P1–P2 segment.[6,11] However, detection of perforator ischemia during PAO is difficult. We experienced a case of unruptured large PCA aneurysm involving the P1–P2 segment, which was recurrent even though two times of coil embolization was performed. Somatosensory-evoked potential (SEP) was useful to evaluate perforator ischemia even if the perforator of the P1 segment was not detected by angiography during PAO.

Case Presentation

The patient was a 49-year-old woman. While being examined for headaches at the age of 43 years, a right large PCA aneurysm with partial thrombus formation and a maximum diameter of 13 mm was discovered in the right P1–P2 segment (**Fig. 1A** and **1B**). Due to poor development of the right PComA and a perforator branching from the right P1 segment (**Fig. 1C**), intra-aneurysmal coil embolization was performed using a simple technique (**Fig. 1D**). Multilocular recurrence was identified in the anterior direction

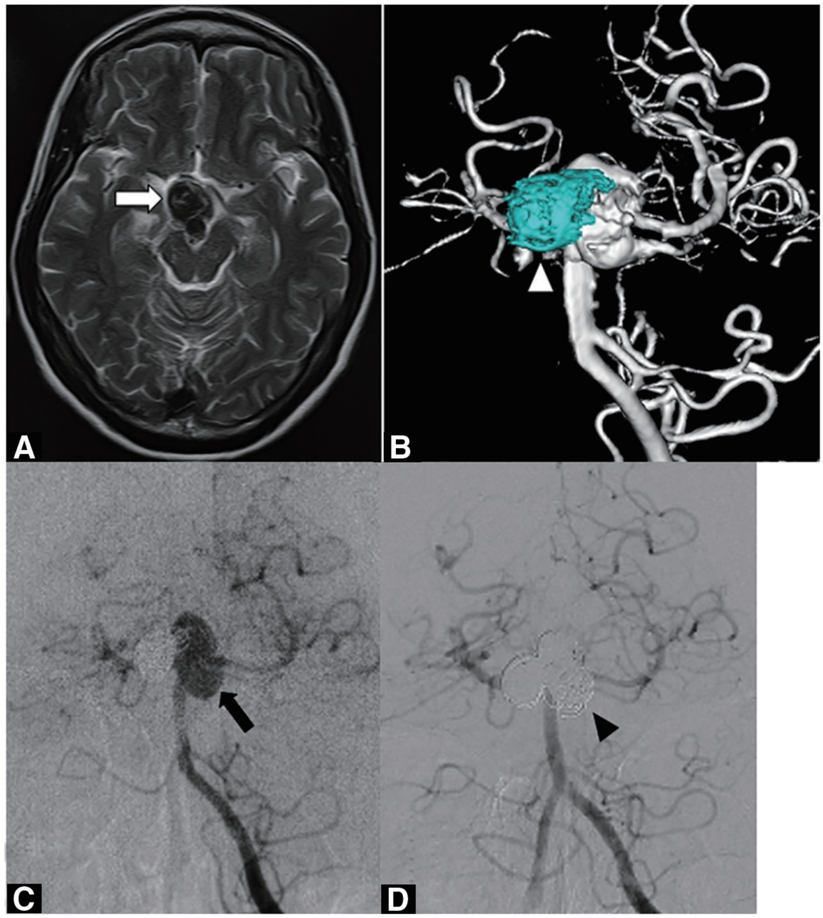

Fig. 2 (**A**) A recurrent aneurysm with a maximum diameter of 17 mm was noted on preoperative T2-weighted MRI (axial plane) (white arrow). (**B**) 3D-DSA (frontal view) before retreatment. An indwelling coil was noted (white arrowhead). (**C**) Angiogram (frontal view) of the left VA before retreatment. Aneurysm before retreatment (black arrow). (**D**) Angiogram (frontal view) of the left VA after retreatment. Recurrent aneurysm was embolized using a simple technique. Aneurysm after retreatment (black arrowhead). VA: vertebral artery

1 year later (age 44 years) (**Fig. 2A**–**2C**). SAC was considered, but the subsequent oral antiplatelet medication for long-term maintenance could complicate the patient's plans for future pregnancy. She did not consent to PAO or craniotomy, in addition. However, she opted for the simple technique of coil embolization despite the higher risk of recurrence in the long term. As the aneurysm was 17 mm in diameter, coil embolization was performed using the same procedure (**Fig. 2D**). Further recurrences were later found, which gradually expanded to fill the interpeduncular fossa and put pressure on the brainstem (**Fig. 3A** and **3B**). Since the aneurysm reached 16 mm in diameter, a third round of treatment was planned (age 49 years) (**Fig. 3C**). We decided to perform SAC but prepared for PAO as a back-up plan if SAC was unsuccessful. Though we had contemplated performing a vascular bypass in case of PAO in addition, it was difficult because of the patient's non-consent to a craniotomy. We planned not to perform PAO and to stop the operation at the simple technique of coil embolization, in case that a collateral circulation route could not be confirmed by the result of BOT. Furthermore, we planned to abort it entirely, in the event that the SEP amplitude as intraoperative electrophysiological monitoring was decreased during PAO. She consented to this surgical plan. Antiplatelet drug administration of aspirin 100 mg/day and clopidogrel 75 mg/day was initiated one week before treatment. Total intravenous anesthesia was performed. At first, general anesthesia was induced with propofol and fentanyl, and subsequently maintained with propofol and remifentanil. Inhalational anesthetic was not used. Muscle relaxant (rocuronium) was additionally administered as appropriate. SEP was recorded as intraoperative electrophysiological monitoring. Electrodes were placed in accordance with the International Federation of

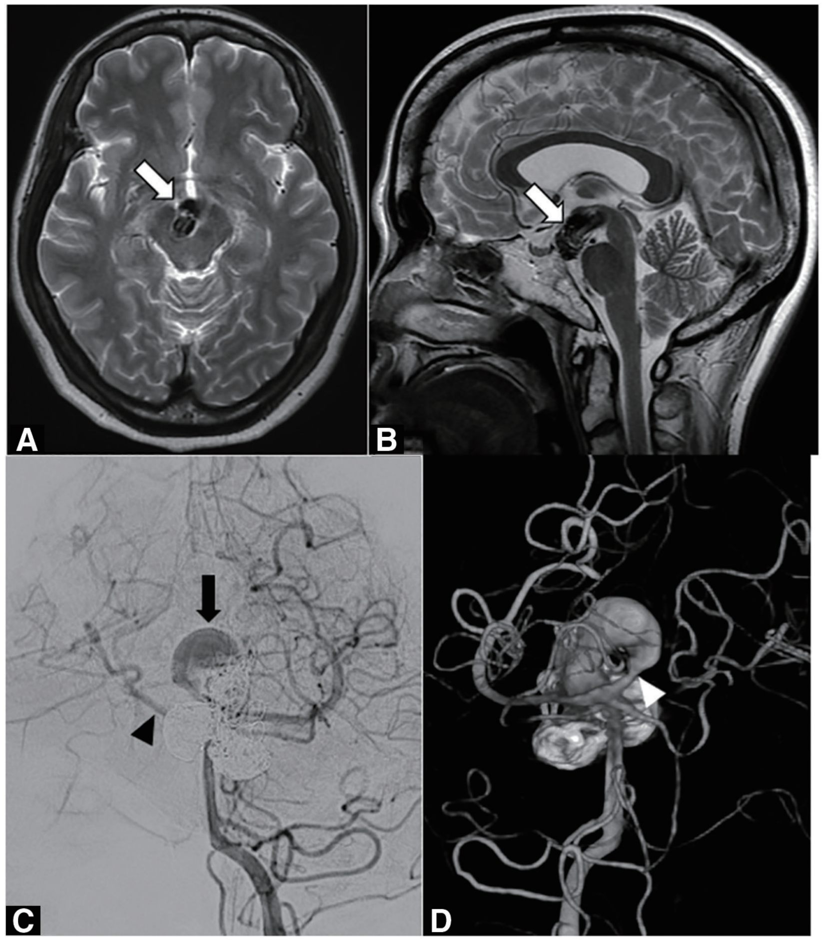

Fig. 3 (**A** and **B**) Further recurrences were found on T2-weighted MRI (axial and sagittal planes) (white arrows). (**C**) A further recurrent aneurysm with a maximum diameter of 16 mm was noted on angiogram (frontal view) of the left VA (black arrow). Right PCA (black arrowhead). (**D**) 3D-DSA (posterior view). The perforator branching from the right P1 segment (proximal to the aneurysm) was not detected by angiography (white arrowhead). PCA: posterior cerebral artery; VA: vertebral artery

Clinical Neurophysiology. To record SEP for upper extremities following median nerve stimulation, the scalp electrodes were placed 2 cm behind C4 (designated C4') and C3 (designated C3') together with Fz. To record SEP for lower extremities following posterior tibial nerve stimulation, the scalp electrodes were placed 2 cm posterior to the standard Cz (designated Cz').[12] The amplitude of the control SEP (upper extremity: N20-P25, lower extremity: P37-N46) was set to 100%, and when the amplitude was decreased to 50% or less, it was regarded as a significant change. Systemic heparinization was administered, and the activated clotting time (ACT) was set at 200–250 seconds. In the system, a 5-Fr FUBUKI Dilator Kit (Asahi Intecc, Aichi, Japan) was inserted as a guiding sheath into the V2 segment of the left vertebral artery (VA) through the right femoral artery puncture. Whether from the effect of blood flow inside the aneurysm or artifacts from an indwelling coil, the perforator branching from the right P1 segment (proximal to the aneurysm) was not detected by angiography (**Fig. 3D**). Although SAC was considered, tortuosity of the right PCA was observed at the aneurysm neck and the distal right PCA could not be secured. We could neither perform SAC nor BOT in the right PCA. Hence, we decided to inflate the balloon in the BA and checked whether blood flowed retrograde into the right PCA from the right middle cerebral artery (MCA) or right anterior cerebral artery via a leptomeningeal anastomosis. A 4-Fr FUBUKI Dilator Kit was inserted to the origin of the right internal carotid artery (ICA) through the left femoral artery puncture. Shouryu HR 4–7 mm (Kaneka Medics, Kanagawa, Japan) was guided into the BA using CHIKAI 14 (Asahi Intecc). A micro guidewire was inserted into the left PCA. And then the balloon was inflated to occlude BA (**Fig. 4A** and **4B**). Before balloon inflation, the right PCA

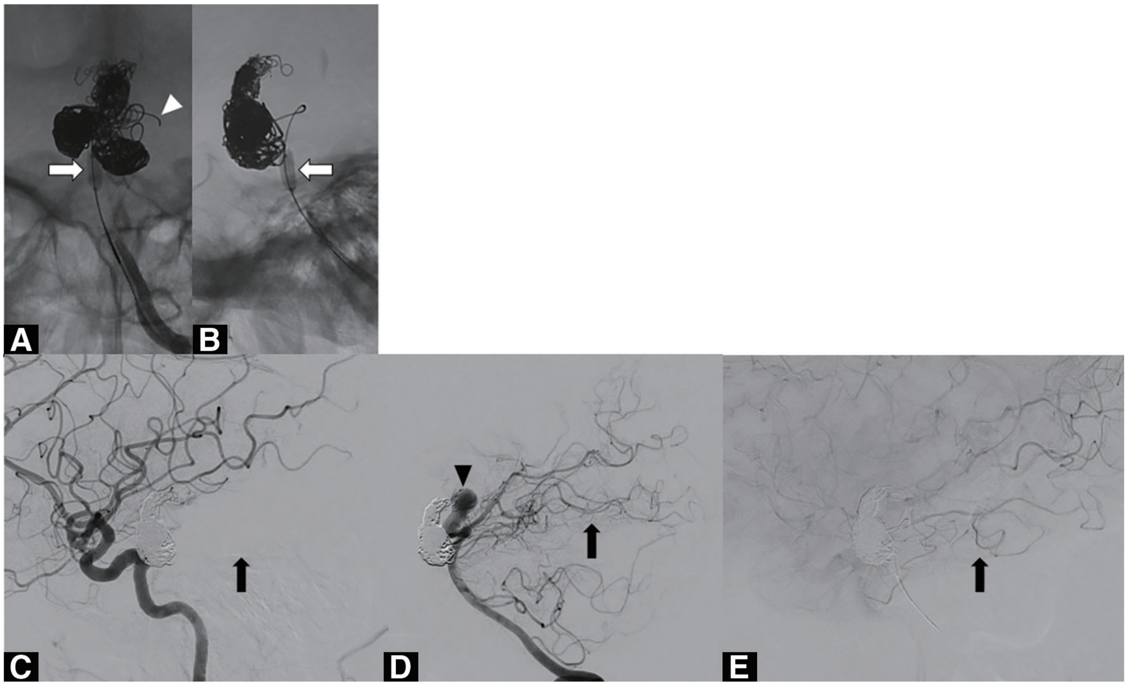

Fig. 4 (**A** and **B**) Angiogram (frontal and lateral views) of the left VA during the balloon inflation in the BA (white arrows). The balloon was inflated to check the collateral circulation routes retrograde into the right PCA from the right MCA via a leptomeningeal anastomosis. A micro guidewire was inserted into the left PCA (white arrowhead). (**C**) Angiogram (lateral view, arterial phase) of the right ICA before the balloon inflation. The right PCA and aneurysm were not depicted (black arrow). (**D**) Angiogram (lateral view, arterial phase) of the left VA before the balloon inflation. The right PCA (black arrow) and preoperative aneurysm (black arrowhead) were detected. (**E**) Angiogram (lateral view, venous phase) of the right ICA during the balloon inflation. Retrograde depiction of the right PCA was obtained from the right MCA via the leptomeningeal collaterals (black arrow). No retrograde depiction of the aneurysm was noted. BA: basilar artery; ICA: internal carotid artery; MCA: middle cerebral artery; PCA: posterior cerebral artery; VA: vertebral artery

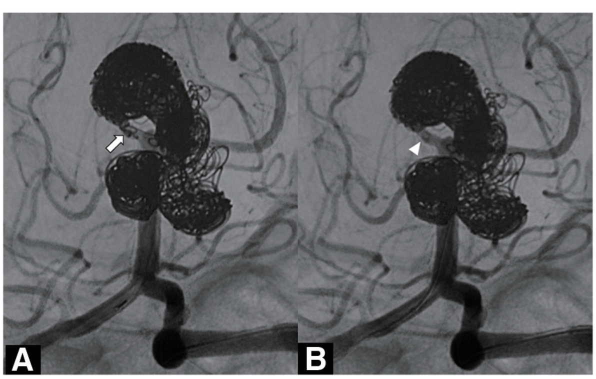

Fig. 5 (**A**) Angiogram (frontal view) of the left VA. Another coil was added to strengthen the PAO to the right P1 segment (white arrow). (**B**) The coil was removed without detaching (white arrowhead). PAO: parent artery occlusion; VA: vertebral artery

and aneurysm were not depicted on angiogram of the right ICA and no blood flow was observed from the right PComA (**Fig. 4C**), and aneurysm was only detected by angiogram of the VA (**Fig. 4D**). Further, blood flow was not observed from the right PComA during balloon inflation, but retrograde depiction of the right PCA was obtained in the venous phase from the right MCA via the leptomeningeal anastomosis. There was no retrograde depiction of the aneurysm (**Fig. 4E**). Since balloon inflation had confirmed the collateral circulation route from the right MCA, the balloon was deflated immediately after the angiogram. The signal of the SEP did not change during balloon inflation. Thus, we speculated that extensive infarction in the right occipital lobe would be avoided after PAO at proximal PCA. Due to the higher risk of further recurrences because of the partial thrombus formation of the aneurysm, we decided to perform PAO with full patient's consent, considering the risks of hemianopsia as a symptom of cortical infarction even if collateral circulation route was present. Excelsior SL-10 (STR; Stryker, Kalamazoo, MI, USA) was guided into the aneurysm using CHIKAI 14. Using Target XL 360 standard 14×50 (Stryker) as the first coil, embolization was started from inside the aneurysm and PAO was performed on the right P1–P2 segment at the aneurysm neck with 14 coils in total. The signal of the SEP was not decreased, and the aneurysm was not visualized. Another coil was added to strengthen the PAO to the right P1 segment (**Fig. 5A**), which decreased the SEP amplitude in the extremities by 3 minutes after its placement. The SEP amplitude of the upper-left, upper-right, lower-left,

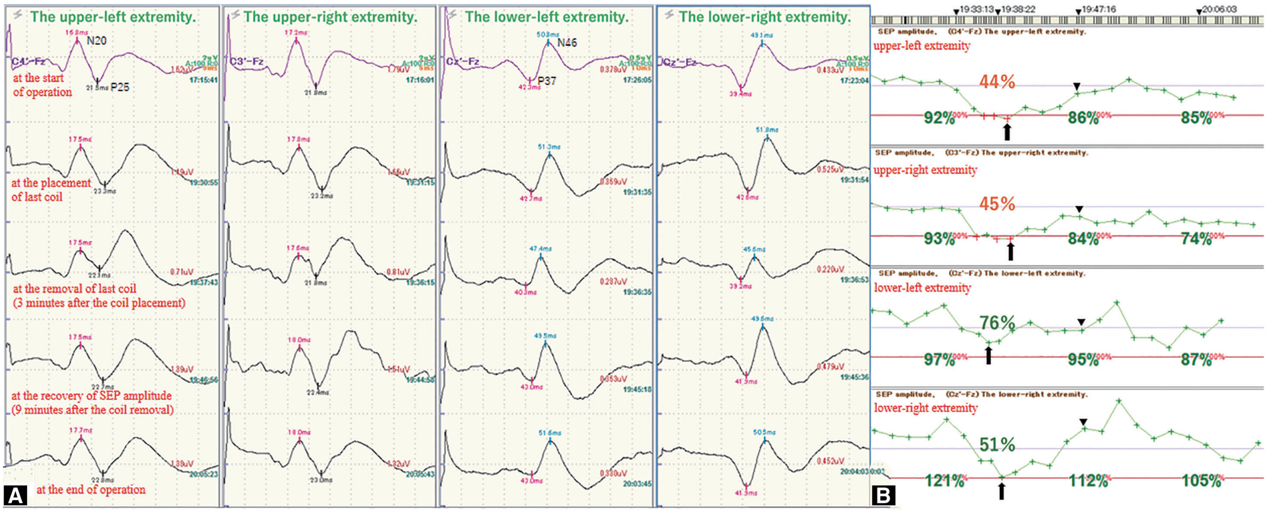

Fig. 6 (**A**) SEP latency and amplitude: at the start of operation, at the placement of last coil, at the removal of last coil (3 minutes after the coil placement), at the recovery of SEP amplitude (9 minutes after the coil removal), and at the end of operation. (**B**) The trend graph of SEP amplitude over time. The SEP amplitude of the upper-left, upper-right, lower-left, and lower-right extremities was decreased to 44%, 45%, 76%, and 51%, respectively, 3 minutes after the placement of last coil (black arrow). After the last coil was removed without detaching, the SEP amplitude began to improve and was recovered by 9 minutes after (black arrowhead). SEP: somatosensory-evoked potential

and lower-right extremities was decreased to 44%, 45%, 76%, and 51%, respectively (**Fig. 6A** and **6B**). As the last coil was thought to be occluding the perforator branching from the right P1 segment, the coil was removed without detaching (**Fig. 5B**). The SEP amplitude began to improve and had recovered by 9 minutes after the coil was removed (**Fig. 6A** and **6B**). Immediately after angiogram, the parent artery was occluded and the aneurysm was no longer depicted (**Fig. 7A**). Postoperative arousal was good, there were no neurological symptoms, and no infarction was observed on diffusion-weighted imaging the following day (**Fig. 7B**). After the treatment, continuous infusion of argatroban was administered at 60 mg/day for 2 days. The patient's course was positive and she was discharged 3 days after treatment at a modified Rankin Scale of 0. The antiplatelet drugs were withdrawn one by one and discontinued after 2 months. No recurrence of aneurysm was observed on MRA 9 months postoperatively, and shrinkage of the aneurysm had reduced the pressure on the brain stem (**Fig. 7C** and **7D**). The patient provided consent for the submission and publication of this case report.

Discussion

PCA aneurysms are rare lesions that account for only 0.7%–2.3% of all intracranial aneurysms.[9,11,13–15] PCA aneurysms occur more frequently in young patients and mostly arise from the P2 segment, and they are more likely to be fusiform or giant in comparison with other intracranial aneurysms.[7,11,13,15,16] The most common clinical presentation of PCA aneurysms is the subarachnoid hemorrhage (80%).[10,11] They can present with signs of mass effect on the surrounding tissues, resulting in oculomotor palsy and hemianopsia.[11] Further, they may present with even a thalamic infarction caused by perforator occlusion.[9,10,14,15]

Surgery of PCA aneurysms is technically challenging and often associated with high rates of morbidity owing to the complexity of the perforating branches from the PCA and their relationship with cranial nerves and the upper brain stem.[4,9,13,17,18] PCA aneurysms are deeply located, where it is difficult to reach the target vessel through a pterional approach. In addition, the subtemporal approach can cause a retraction injury of temporal lobe.[3]

In contrast, endovascular treatment is not associated with manipulation of the surrounding tissues. Therefore, the risks of brain infarction due to retraction or removal are reduced. As a result, surgery of PCA aneurysms is being replaced gradually by endovascular therapy. Advances in devices for endovascular therapy and intraoperative electrophysiological monitoring have improved the results of treatment. The role of endovascular therapy is highly important. Adjunctive techniques make it possible to embolize wide neck or fusiform aneurysms with patency of the parent artery. However, a simple technique, balloon-assisted coil embolization, and SAC for these types of aneurysm are limited due to their etiology or morphology in some cases.[5,6,10] It is not uncommon to perform PAO for

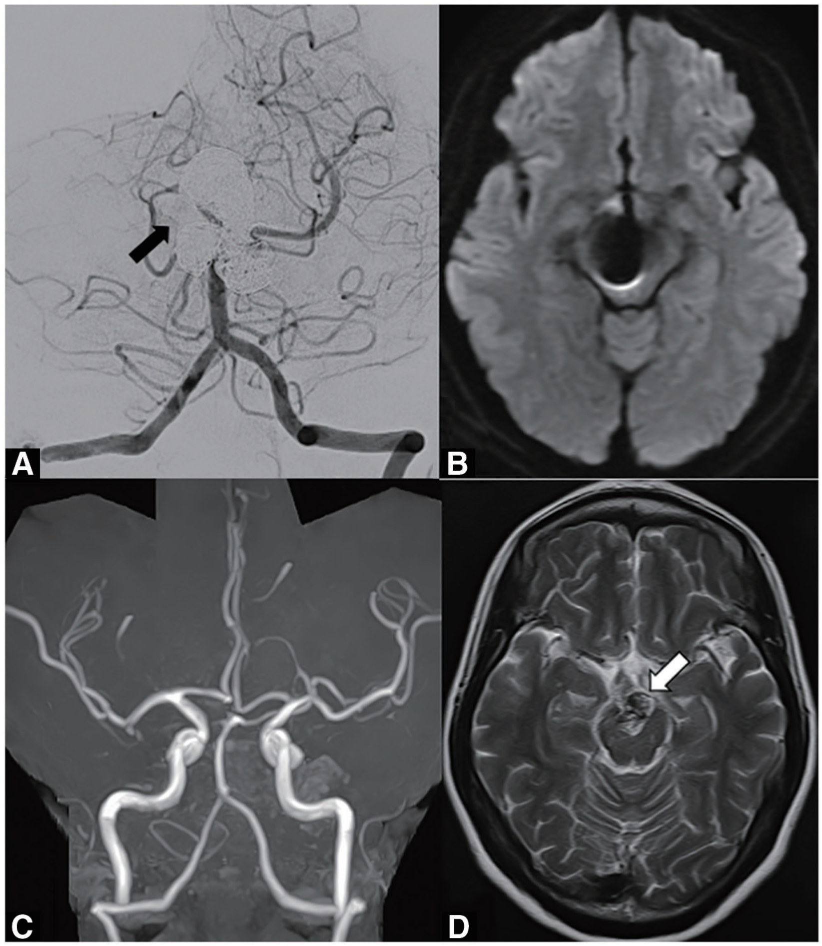

Fig. 7 (**A**) Angiogram (frontal view) of the left VA. The parent artery was occluded and aneurysm was not visualized (black arrow). (**B**) No abnormality was noted on postoperative DWI (axial plane). (**C**) No recurrence of aneurysm was observed on MRA 9 months postoperatively. (**D**) Marked shrinkage of the aneurysm was noted on T2-weighted MRI (axial plane) 9 months postoperatively (white arrow). DWI: diffusion-weighted imaging; VA: vertebral artery

large PCA aneurysm.[1,3,7,9–11] This is because of the presence of many collateral circulation routes, including 1) anastomosis of the lateral posterior choroidal artery and anterior choroidal artery, 2) anastomosis of the long circumflex branch arising from the P1 segment and the superior cerebellar artery, 3) anastomosis of the splenial artery and posterior pericallosal artery, and 4) anastomosis of inferior temporal artery of the PCA and the posterior temporal artery of the MCA.[11,15] Collateral circulation routes are often checked using BOT prior to PAO. BOT is widely used for evaluating patient tolerance of PAO with neurological monitoring, but its value remains controversial.[3,9,11,15] Although angiography during BOT can show a collateral circulation route of the PCA territory, for perforator, both false-positive and false-negative results have been reported previously.[2,3] Furthermore, as far as aneurysms of P1 to P2a segment are concerned, the proximal space of PCA is not enough for balloon, and BOT is not always reliable, especially in the evaluation of perforators.[4] Also, BOT of the planned occlusion site may be attempted, but it is not always as straightforward as in the present case, because collateral flow from BA via the left PComA was not evaluated. Moreover, BOT in the tortuous PCA is often technically difficult in delivering the balloon to the target vessel, increasing the risk of procedure-related complications.[2,3] In the present case, because BOT was not performed on the PCA, only retrograde depiction of the right PCA was possible via the leptomeningeal anastomosis from the right MCA.

Ischemic complications have been reported in 12%–17% with PAO for treating PCA aneurysm.[7–9,11,13,15] Permanent homonymous hemianopsia after PAO as a symptom of cortical infarction was reported even in a case in which the PCA was depicted retrogradely via the leptomeningeal anastomosis.[11,15,19] The risk has been 0%–12.5%.[7,9,11,14] In the present case, BOT was originally planned under general anesthesia, but because we prepared for PAO as a back-up plan, monitoring the visual-evoked potential (VEP) would likely have provided even more useful information for predicting cortical infarction. Nonetheless, a thorough literature review revealed no reports of VEP use in endovascular treatment. Furthermore, due to the impact of periorbital light stimulation device and wiring for VEP monitoring on X-ray view, intraoperative manipulation could become difficult. Therefore, the use of VEP during endovascular therapy does not appear to be practical. However, the major problems are hemiparesis and sensory deficit due to occlusion of the perforator.[7,9,10] Known perforators from the PCA to the brain stem include the direct perforating branch and circumflex branch of the P1 segment and the thalamogeniculate artery and peduncular perforating artery of the P2 segment.[16] PAO may cause infarctions in areas such as the cerebral peduncle and thalamus. The collateral circulation route among perforators is not well defined.[7] Although cortical infarction can be predicted to some extent using BOT, perforator infarction caused by PAO is relatively difficult to predict with BOT as noted above. PAO may compromise flow of perforator near the aneurysm. PAO at proximal PCA will be more likely to cause perforator infarction involving the midbrain and thalamus perfused by the perforating artery arising from the P1–P2 segment. In general, PAO will be avoided at the P1–P2 segment with the rich vascular supply to the midbrain and thalamus. PCA aneurysm involving the P1–P2 segment remains challenging.

There has also been a report of measuring cerebral blood flow before and during BOT using single-photon emission computed tomography (SPECT) to evaluate tolerance of PAO.[14] While SPECT can assess ischemia in the cortical region, doing so in the perforator region is difficult. Although there have been reports of PAO in bypass surgery, predicting the appearance of ischemia in the perforator region is seen as difficult even if blood flow in the cortical region can be maintained.[13,19]

In the present case, early and rapid diagnosis and treatment of perforator occlusion was crucial for the patient's outcome. During the PAO procedure, we adopt a wait-and-see strategy, i.e. observation of intraoperative electrophysiological monitoring for a few minutes after coil placement without detaching. When perforator occlusion occurred during the wait-and-see period, it was immediately identified by intraoperative electrophysiological monitoring. Then, the coil was removed without detaching. SEP was useful for detecting the perforator occlusion in the present case. Motor-evoked potential (MEP) would also have been useful for evaluating a perforator to the cerebral peduncle. However, only SEP equipment was available for intraoperative monitoring of endovascular therapy at our facility at that time. Furthermore, coil embolization was performed near the perforator branching from the right P1 segment, yet it decreased the SEP amplitude in bilateral upper and lower extremities. Approximately in 21%, all the paramedian thalamic and superior paramedian mesencephalic arteries originated from a single P1 segment, thus supplying the entire paramedian artery territory. In addition, in 10.7%, the paramedian arteries originated from only one trunk, meaning that occlusion would always cause bilateral thalamopeduncular infarction.[20] It seems plausible that this perforator was occluded in the present case. Unfortunately, this perforator was not recognizable on our routine angiography and had not been adequately evaluated prior to PAO. Oishi et al. reported that selective injection from a 4-Fr. distal access catheter (DAC) was useful to identify perforating arteries arising from the PCA.[21] It may have been possible to identify the perforating branch through selective injection from DAC prior to PAO. If the perforating branch had been identified, this would have enabled a closer evaluation of the detailed anatomy of PCA where the coil should be placed during PAO. However, in the present case, selective injection from DAC was not possible during P1 segment occlusion.

As a coil may directly occlude the perforator, one method for preventing ischemic symptoms in the perforator region has been reported to be to avoid placing the coil in the location of a perforator to the brain stem.[11] There may be multiple perforators and collateral circulation routes in a small area, which requires the most effective PAO in the short segment so as not to occlude the perforators and collateral circulation routes.[11,15] Therefore, it is important that PAO should be limited to the aneurysm neck, particularly because a single coil can occlude the perforator or a collateral circulation route as in the present case. Unnecessarily adding a coil must be strictly refrained from. The perforator occlusion would be detected timely according to the intraoperative electrophysiological monitoring. Thus, it is probably a feasible method to ensure early and rapid diagnosis of perforator occlusion. PAO should be performed using intraoperative electrophysiological monitoring such as SEP, MEP, and VEP. The intraoperative electrophysiological monitoring is indispensable to endovascular therapy to evaluate the risk of cortical or perforator infarction.

Conclusion

During PAO at the P1 segment of large PCA aneurysm involving the P1–P2 segment, SEP may be helpful to prevent perforator infarction, even if perforating artery originating from the proximal portion of the aneurysm was not detected by angiography.

Disclosure Statement

We declare no conflict of interest.

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