CASE REPORT

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Complex aneurysm formation in the proximal segment of the posterior cerebral artery: a report of two cases

Hiroo Sasaki[1], Syuntaro Takasu[2] and Yukio Seki[2]

[1]*Department of Neurosurgery, Nagoya University Graduate School of Medicine, Nagoya, Japan*
[2]*Department of Neurosurgery, Japanese Red Cross Aichi Medical Center Nagoya Daini Hospital, Nagoya, Japan*

ABSTRACT

Posterior cerebral artery (PCA) aneurysms are rare and often fusiform. We describe two cases of complex proximal PCA aneurysm in two women in their 60's, which probably resulted from segmental arterial degeneration. Both presented with subarachnoid hemorrhage and had common angiographic and intraoperative findings: tortuous configuration of the affected P1 segment, whitish or yellowish appearance of a portion of the lesion, lesion calcification, and multiple aneurysms in the segment. Interestingly, no significant atherosclerotic changes were noted in other cerebral arteries. The ruptured aneurysm could be successfully trapped, with superficial temporal artery (STA)-PCA bypass in one and without bypass in the other, and both patients recovered well. As complex aneurysm formation in the cases described here are probably related to proximal PCA segmental degeneration, we recommend trapping the lesion, with or without STA-PCA bypass, depending on the size and patency of the posterior communicating artery.

Keywords: complex, bypass, posterior cerebral artery aneurysm, subarachnoid hemorrhage, trapping

Abbreviations:
PCA: posterior cerebral artery
PCoA: posterior communicating artery
STA: superficial temporal artery

INTRODUCTION

Posterior cerebral artery (PCA) aneurysms are rare and account for 0.7%–2.3% of all intracranial aneurysms.[1] Most are located in the precommunicating segment of the posterior cerebral artery (P1) or at the junction of P1 and the posterior communicating artery (PCoA).[2] About 24% of all PCA aneurysms are fusiform and are often present as multiple intracranial aneurysms.[2,3] Here, we describe two cases with complex proximal PCA aneurysms that were successfully treated with microsurgery. Intraoperative and angiographic findings suggest that complex aneurysm formation was probably due to proximal PCA segmental degeneration.

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Corresponding Author: Hiroo Sasaki, MD

Department of Neurosurgery, Nagoya University Graduate School of Medicine, 65 Tsurumai-cho, Showa-ku, Nagoya 466-8550, Japan

Tel: +81-52-744-2353, Fax: +81-52-744-2360, E-mail: h-sasaki@med.nagoya-u.ac.jp

CASE REPORTS

Case 1

A 66-year-old female with a past medical history of cardiac arrhythmia was transferred to our hospital with a diagnosis of subarachnoid hemorrhage. On admission, her pupils were unequal and her Hunt and Kosnik grade was 2. Computed tomography (CT) of the head showed a small amount of clot in the basal cisterns, a modest calcification in the left prepontine cistern, and hydrocephalus (Figure 1A). Digital subtraction angiography (DSA) revealed an irregularly shaped P1 associated with multiple aneurysms (Figure 1B). On the day after admission, she underwent surgical trapping of the lesion (Endovascular treatment had not been commonly performed at that time).

Specifically, under general anesthesia, the patient's head was fixed to a head holder and a left frontotemporal craniotomy was performed. A left ventricular drain was inserted, and then under an operating microscope, the Sylvian fissure was widely opened to reveal the carotid cistern. The abnormal P1 segment harboring multiple aneurysms was exposed by posterolaterally retracting the temporal lobe (Figure 2). The arterial wall appeared yellowish in color and was associated with multiple blister-like aneurysms and was visible a partially thrombosed aneurysm at the junction of the P1 and P2 segments. Aneurysms were also seen behind the PCoA. As the abnormal arterial segment had adhered to the oculomotor nerve, complete dissection was abandoned and trapping of the P1 segment was performed without disturbing blood flow to the left thalamoperforating artery. No involvement of other cerebral arteries was apparent.

Postoperatively the patient experienced transient aphasia and disorientation, which gradually resolved. Postoperative DSA showed disappearance of the aneurysms, preservation of the left thalamoperforating artery, and opacification of the distal left PCA through the PCoA (Figure 3). She received inpatient rehabilitation and was discharged home four weeks after admission

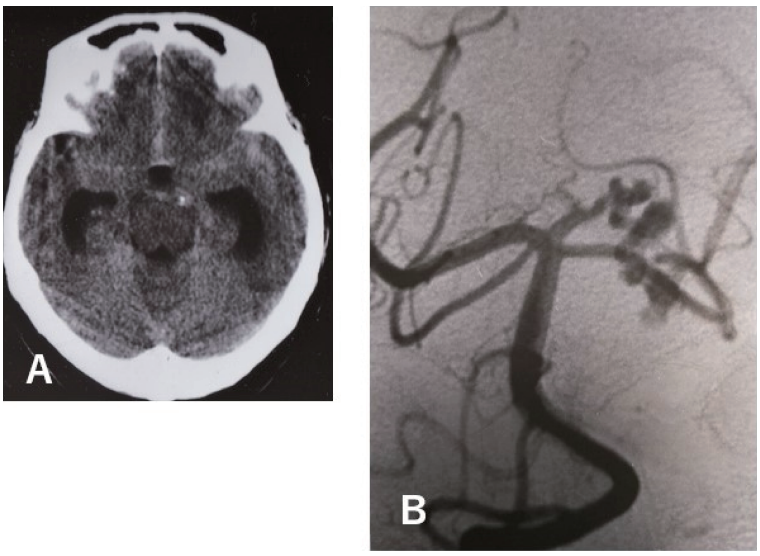

Fig. 1 Preoperative images in case 1
CT scan of case 1 on admission shows a small amount of subarachnoid hemorrhage in the basilar cisterns and a calcification of the left prepontine cistern (A). An anteroposterior view of the right vertebral angiogram shows complex aneurysms of the precommunicating segment of the left posterior cerebral artery (B).

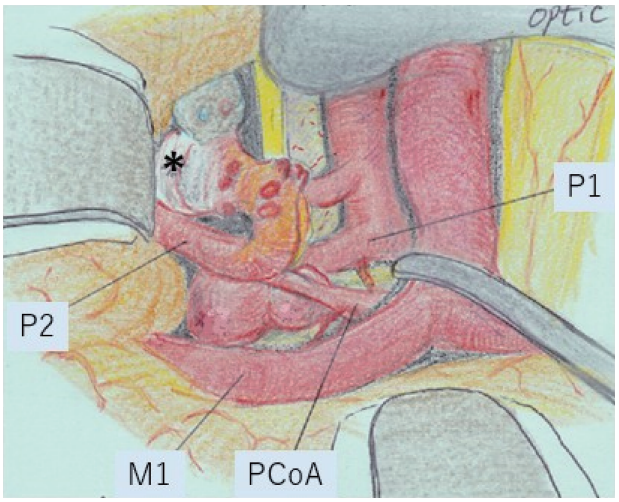

Fig. 2 Intraoperative illustration for case 1
A drawing of the intraoperative view in case 1 shows multiple aneurysms including blood blister-like aneurysms on the tortuous precommunicating segment of the left posterior cerebral artery associated with the degenerative appearance of the arterial wall.
*Partially thrombosed aneurysm
PCoA: posterior communicating artery

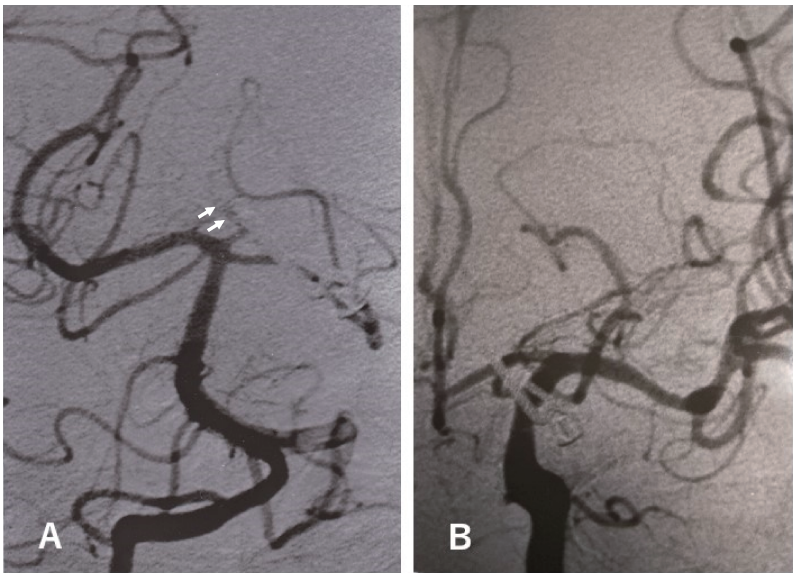

Fig. 3 Postoperative angiographies in case 1
Postoperative right vertebral angiogram (A) and left carotid angiogram (B) show complete occlusion of the lesion in the precommunicating segment of the left posterior cerebral artery (PCA) and opacification of the distal left PCA through the posterior communicating artery. The left thalamoperforating artery is preserved (arrows).

without neurological deficits. Three months later, she developed symptomatic normal pressure hydrocephalus and underwent ventriculoperitoneal shunt placement, which resolved her symptoms.

Case 2

A 63-year-old woman with a past medical history of hypertension was referred to our hospital for severe headache. Her initial Glasgow Coma Scale score was 13 (E3 V4 M6). No limb weakness was detected on neurological examination. A head CT scan showed diffuse subarachnoid hemorrhage (Figure 4A), and a DSA acquired the next day showed complex fusiform and saccular aneurysms in a tortuous right P1 segment (Figure 4 B, C). The right PCoA could not be visualized and was thought to be hypoplastic. As stenosis was present within the lesion, an endovascular approach was ruled out. As a superficial temporal artery (STA)-PCA bypass was required, right frontotemporal craniotomy and subtemporal approach were performed.

Intraoperative inspection revealed a thin-walled aneurysm with a daughter sac located at the PCoA-P2 junction, the latter was thought to be the ruptured aneurysm (Figure 5A), and the junction appeared whitish or yellowish. STA-P2 bypass followed by trapping was performed as clipping the aneurysm neck was considered difficult because of its shape (Figure 5B). As the thalamoperforating arteries arose from the P1 fusiform aneurysm and the walls of the P1 aneurysms were not reddish, the anatomical connection between P1 and the PCoA was intentionally maintained to avoid thrombosis at the origin of the perforators. Surgery was uneventful but right oculomotor nerve palsy was observed after the procedure. Postoperative DSA showed obliteration of the aneurysm at the PCoA-P2 junction and patency of the STA-P2 bypass (Figure 6 A, B).

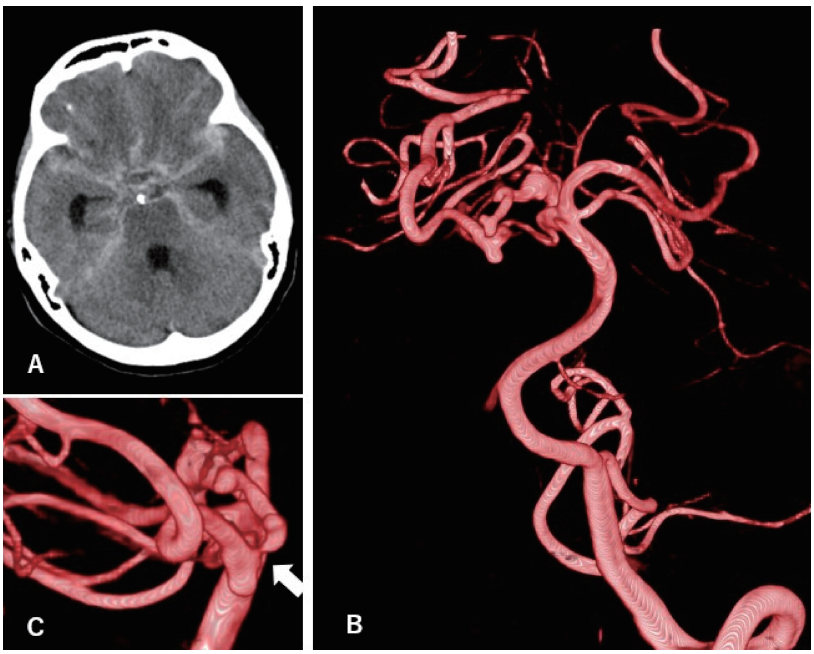

Fig. 4 Preoperative images of case 2
CT scan at admission shows diffuse subarachnoid hemorrhage and a calcification of the prepontine cistern (A). Preoperative left vertebral angiograms, antero-posterior view (B) and lateral view (C), show a tortuous precommunicating segment of the right posterior cerebral artery that is associated with multiple complex aneurysms and an intralesional stenosis (arrow).

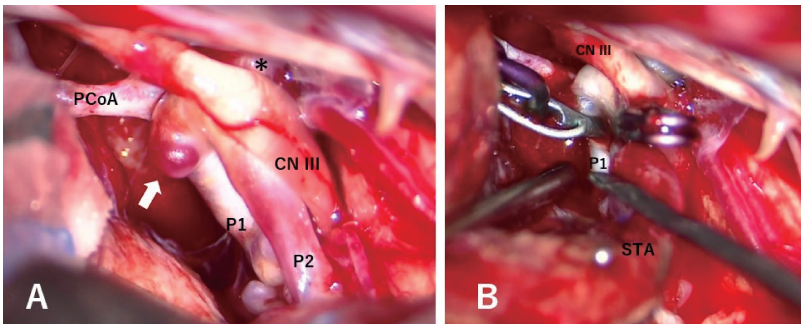

Fig. 5 Intraoperative images of case 2
Intraoperative view (right subtemporal approach) shows the whitish appearance of the P1 segment and multiple aneurysms (A). The thin-walled aneurysm (arrow) located just distal to the PCoA-P2 junction had ruptured, and the point of rupture was located behind the tentorium cerebelli (*). Following STA-P2 bypass, the aneurysm was trapped and collapsed (B).
CN III: oculomotor nerve
PCoA: posterior communicating artery
STA: superficial temporal artery

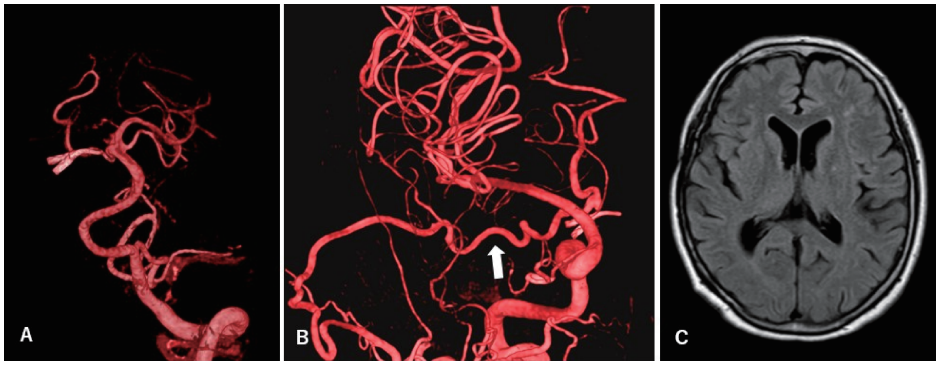

Fig. 6 Postoperative images of case 2
Postoperative anteroposterior views of the left vertebral angiogram (A) and right carotid angiogram (B) show obliteration of the ruptured aneurysm and opacification of the right posterior cerebral artery through the bypass (arrow). Postoperative fluid-attenuated inversion recovery magnetic resonance imaging (C) shows no infarction in the posterior cerebral artery distribution.

The right P1 segment had not opacified but thalamic infarction was not visualized on magnetic resonance imaging, suggesting the presence of patent perforators (Figure 6C). The patient was discharged home one month after surgery with modified Rankin Scale score of 2, and her oculomotor nerve palsy improved three months after discharge.

DISCUSSION

Multiple aneurysms are found in 53% of patients who harbor a PCA aneurysm; among these, another aneurysm is located on the PCA in 12%.[4] However, the multiple PCA aneurysms of the patients in this report clearly differ from those previously reported. Based on angiographic and

intraoperative findings, proximal PCA segmental degeneration seemed to be a background of complex aneurysm formation. Common characteristics of this disorder included tortuous configuration of the affected P1 segment, whitish or yellowish appearance of a portion of the lesion, lesion calcification, and multiple aneurysms of the segment; in addition, no significant atherosclerotic changes of other cerebral arteries were noted. In case 2, significant stenosis was also noted within the affected P1 segment. The similar age of presentation (7th decade), intraoperative findings, and presence of calcification in these cases suggest that the lesion is focal, degenerative, and progressive. The presence of multiple aneurysms, including the blood blister-like aneurysms of case 1, also suggests a predisposing structural failure in the affected arterial segment.

The cause and pathological mechanisms leading to focal degeneration limited to the P1 segment are unclear. Considering the focality, it is likely that the initiating change occurred during the stage of embryologic development of the vertebrobasilar system when the newly formed P1 segments complete the circle of Willis.[5] This change then might have been promoted to form multiple aneurysms by systemic factors such as hypertension and aging.

However, this report is limited by a lack of pathological findings of the lesion that could elucidate the mechanism of degeneration.

Surgical trapping and bypass of fusiform PCA aneurysms has been previously reported.[6-8] The optimal surgical management of complex aneurysms formed on the degenerated proximal segment of PCA is also trapping of the affected segment that involves the ruptured aneurysm while preserving the thalamoperforating arteries. STA-P2 bypass is necessary if the PCoA is hypoplastic or the PCoA-P2 junction is within the trapped segment. Endovascular trapping of the lesion would be another option if cannulation of the tortuous P1 segment is possible.

CONCLUSION

Two cases of a unique form of cerebrovascular disorder, complex aneurysm formation on the proximal PCA segment associated with the segmental parent artery degeneration, are presented. It should be managed by trapping of the lesion with or without STA-P2 bypass, depending on the size and patency of the PCoA.

CONFLICTS OF INTEREST DISCLOSURE

The authors declare they have no conflicts of interest and no commercial relationships and received no support from pharmaceutical or other companies.

INFORMED CONSENT

We received consent from all patients for the publication of this case report.

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