Bypass surgery for ruptured dissecting aneurysms of the proximal posterior cerebral artery: illustrative case

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BACKGROUND Posterior cerebral artery (PCA) dissecting aneurysms commonly occur in the proximal PCA and are considered rare. The treatment of proximal PCA dissecting aneurysms is challenging because of the existence of perforators supplying the vital neural structures. Recently, endovascular intervention has been used; however, concerns for ischemic or hemorrhagic complications exist.

OBSERVATIONS A 54-year-old woman presented with subarachnoid hemorrhage due to dissecting aneurysm rupture at the P1-P2 junction of the PCA. The thalamoperforating artery (TPA) and medial posterior choroidal artery (MPchA) originated from the proximal end and the distal end of the aneurysm, respectively. Additionally, the posterior communicating artery (PcomA) connected with the dissected segment. To preserve these perforators, we performed surgical trapping combined with superficial temporal artery (STA) PCA anastomosis. Clips were applied for trapping the proximal and distal end of the aneurysm, with preservation of the TPA and MPchA origin. PcomA was left open for blood flow preservation to the perforators directly arising from the aneurysm. The postoperative course was uneventful, and the patient was discharged.

LESSONS Surgical trapping using STA-PCA bypass could be a treatment of choice for proximal PCA dissecting aneurysms, considering its potential for cure and prevention of ischemic complications.

https://thejns.org/doi/abs/10.3171/CASE22341

KEYWORDS posterior cerebral artery; subarachnoid hemorrhage; dissecting aneurysm; bypass

Posterior cerebral artery (PCA) dissecting aneurysms are rare, accounting for less than 1% of intracranial dissecting aneurysms, with most being localized in the proximal segment.1-5 To prevent rebleeding, intervention is necessary for ruptured cases. With the advent of endovascular devices and sophisticated techniques, most cases are treated via endovascular intervention.1 Access to the deep lesion location with minimal invasion is among the advantages of endovascular treatment. However, concerns for ischemic or hemorrhagic complications associated with endovascular treatment exist.

Herein, we report a case of a ruptured dissecting aneurysm at the P1-P2 junction successfully treated with surgical trapping with superficial temporal artery (STA) PCA bypass.

Illustrative Case

A 54-year-old woman with an unremarkable medical history was admitted to the emergency department with sudden headache and vomiting. Computed tomography showed diffuse subarachnoid hemorrhage (SAH), predominantly in the interpeduncular cistern (Fig. 1A). Digital subtraction angiography (DSA) of the left vertebral artery revealed a fusiform aneurysm, 3.5 x 3.4 mm in size, at the P1-P2 junction of the right PCA. DSA showed faint filling of the posterior communicating artery (PcomA) connected with the dissected segment. To preserve these perforators, we performed surgical trapping of the aneurysm.
combined with STA-PCA anastomosis through a subtemporal approach (Video 1). The STA parietal branch was prepared, and temporal craniotomy was performed. The ambient cistern was opened through the subtemporal space. Adjacent to the oculomotor nerve, the aneurysm was exposed. Some perforators originated directly from the dissecting aneurysm (Fig. 2A). After completing the STA-PCA bypass in an end-to-side fashion at the P2 segment, the aneurysm clip trapped the proximal and distal end of the aneurysm (Fig. 2B). Clips were applied so as not to occlude the TPA and MPchA origin. The PcomA was left open to preserve circulation to the perforators arising from the aneurysm, which incompletely trapped the aneurysm. Indocyanine green videoangiography demonstrated patency of the STA-PCA bypass and all the perforators with disappearance of the aneurysm. Transient right oculomotor nerve palsy was observed after surgery, which completely resolved after 3 months. Postoperative magnetic resonance imaging revealed no ischemic complications (Fig. 3A). Postoperative angiography showed disappearance of the aneurysm with preservation of the TPA, MPchA, and PcomA (Fig. 3B). The patient was discharged 3 weeks after surgery.

**VIDEO 1.** Clip showing surgical trapping of the aneurysm combined with STA-PCA anastomosis through a subtemporal approach. The STA parietal branch was prepared, and temporal craniotomy was performed. The ambient cistern was opened through the subtemporal space. Adjacent to the oculomotor nerve, the aneurysm was exposed. Some perforators originated directly from the dissecting aneurysm. After completing STA-PCA bypass in an end-to-side fashion at the P2 segment, the aneurysm clip trapped the proximal and distal end of the aneurysm. Clips were applied not to occlude the TPA and MPchA origin. PcomA was left open to preserve circulation to the perforators arising from the aneurysm. Indocyanine green videoangiography demonstrated patency of the STA-PCA bypass and all the perforators with disappearance of the aneurysm. Click here to view.

**Results of the Literature Review**

We reviewed previous English-language literature regarding proximal PCA ruptured dissecting aneurysms to elucidate a variety of treatment strategies and their corresponding outcomes. PubMed publications were searched using key terms such as “posterior cerebral artery dissection” and “dissecting aneurysm.” There were 33 cases of ruptured dissecting aneurysms of the proximal PCA, including the present case, as shown in Table 1.1,2,4–16 The mean age was 41.3 years, and 21 patients (63.6%) were male. The main location of the aneurysms was the P2 segment (60.6%), followed by the P1-P2 junction (33.3%). Endovascular methods (30/33) were used for most patients, including parent artery occlusion (PAO) (21/30), stent-assisted coil embolization (3/30), aneurysm sac coil embolization (3/30), and flow diverter (2/30). Surgical trapping was performed in three cases (3/33): two cases with bypass and one case without bypass. No cases treated by direct surgery showed hemorrhagic or ischemic complications. Conversely, infarctions were detected in 5 of 30 (16.7%) cases after endovascular treatment. Furthermore, rebleeding occurred in 3 cases (10%) after endovascular treatment. Among them, 2 cases were treated using endovascular PAO and 1 case was treated using stent-assisted coil embolization. The latter case was treated via endovascular PAO after rebleeding, which caused perforator territory infarction. Nonhemorrhagic recurrence was detected in 5 cases after endovascular treatment (16.6%). For these recurrent cases, reconstructive therapies were performed in 4 cases, and endovascular PAO was performed in 1 case. The last patient experienced rebleeding 3 hours after endovascular PAO and died.

**Discussion**

**Endovascular Treatment for PCA Dissecting Aneurysm**

A PCA dissecting aneurysm is rare, accounting for less than 1% of intracranial dissecting aneurysms, which causes SAH, ischemic stroke, or cranial nerve palsy due to their mass effect.1,3,7,18 Moreover, 54% of PCA dissecting aneurysms presented with SAH, 25% presented with focal neurological deficits due to infarcts or mass effect, and 21% were asymptomatic.19 PCA dissecting aneurysm treatment depends on its clinical presentation. Furthermore, conservative therapy is acceptable for cases of unruptured aneurysms.20 Contrarily, surgical or endovascular treatment is recommended for cases of ruptured aneurysms. With the advent of endovascular devices, most of the recent cases were treated endovascularly.1
Most of the PCA dissecting aneurysms are located in the proximal PCA: 22.6% in the P1 segment, 12% in the P1-P2 junction, 46.7% in the P2 segment, and 18.6% in the P3 segment. Risk for complication of endovascular treatment for PCA depends on the existence of perforating arteries. The perforating branches of the proximal PCA include TPA, short circumflex artery (SCxA), long circumflex artery (LcxA), and MPchA. In the cadaveric study, all TPAs arose from the posterosuperior aspect of the P1 segment and supplied the medial part of cerebral peduncle and thalamus. The SCxAs and the LCxAs were observed in 42.8% and 57.1% of the P1 segment, respectively, supplying the lateral part of the mesencephalon and diencephalon. The MPChAs were observed in 35.7% of the P1 segment, originating from the most distal part of the P1 segment. Although not always angiographically visible, these perforators should be preserved in the treatment of proximal PCA dissecting aneurysms.

Deconstructive and reconstructive methods are among the surgical and endovascular treatment strategies for PCA dissecting aneurysms.

FIG. 2. A: Preprocedural schema showing the arterial structure, dissecting aneurysms, and blood flow (blue arrows). B: Postprocedural schema demonstrating blood flow direction changes after bypass and trapping. Note that the perforator arising from the dissected segment provided blood flow from the PcomA.

FIG. 3. A: Postoperative diffusion-weighted imaging revealed the absence of a cerebral infarction. B: Postoperative DSA demonstrating the patency of the bypass (asterisk), PcomA (arrow), TPA (double arrows), and MPchA (arrowhead) and disappearance of the aneurysm.
TABLE 1. Cases of ruptured dissecting aneurysms of proximal PCA

| Authors & Year       | Case No. | Age (yrs)/Sex | Location                | Treatment          | Outcome      | Recurrence | Outcome (mRS Score) |
|---------------------|----------|---------------|-------------------------|--------------------|--------------|------------|---------------------|
| Park et al., 2015   | 1        | 66/F          | P1-P2 junction          | PAO w/ coils       | Un-eventful  | –          | 0                   |
|                     | 2        | 42/F          | P1-P2 junction          | PAO w/ coils       | Un-eventful  | –          | 0                   |
|                     | 3        | 47/M          | P1-P2 junction          | PAO w/ coils       | Rebleeding +| 6          |                     |
|                     | 4        | 33/F          | P2                      | Flow diversion     | Un-eventful  | –          | 0                   |
|                     | 5        | 61/M          | P2                      | Coiling            | Un-eventful  | +          | 2                   |
|                     | 6        | 22/M          | P2                      | PAO w/ coils       | Infarction of PCA – | 2          |
|                     | 7        | 72/M          | P1                      | PAO w/ coils       | Infarction of PCA – | 4          |
|                     | 8        | 41/F          | P2                      | Trapping w/ bypass | Un-eventful  | –          | 0                   |
| Wang et al., 2012   | 9        | 53/M          | P2                      | PAO w/ coils       | Infarction of PCA – | 1          |
|                     | 10       | 48/M          | P2                      | PAO w/ coils       | Un-eventful  | –          | 0                   |
|                     | 11       | 15/F          | P2                      | PAO w/ coils       | Un-eventful  | –          | 0                   |
|                     | 12       | 39/M          | P1                      | PAO w/ coils       | Un-eventful  | –          | 5                   |
| Wael Osman et al., 2017 | 13      | 49/F          | P2                      | SAC                | Un-eventful  | +          | 0                   |
| Lv et al., 2020     | 14       | 40/M          | P1-P2 junction          | PAO w/ coils       | Rebleeding   | –          | 0                   |
|                     | 15       | 62/M          | P1-P2 junction          | SAC                | Infarction of perforators – | 2          |
| Horie et al., 2008  | 16       | 36/F          | P1-P2 junction          | Coiling            | Un-eventful  | +          | 0                   |
| Liu et al., 2011    | 17       | 31/M          | P2                      | PAO w/ coils       | Un-eventful  | –          | 0                   |
|                     | 18       | 54/F          | P2                      | PAO w/ coils       | Un-eventful  | –          | 0                   |
|                     | 19       | 73/M          | P2                      | PAO w/ coils       | Un-eventful  | –          | 0                   |
|                     | 20       | 56/M          | P1-P2 junction          | PAO w/ coils       | Un-eventful  | –          | 0                   |
|                     | 21       | 16/M          | P2                      | Proximal PAO w/ coils | Un-eventful  | –          | 0                   |
| Lee et al., 2011    | 22       | 15/M          | P2                      | SAC*               | Rebleeding & infarction of perforators + | 3          |
| Lobo et al., 2011   | 23       | 62/M          | P1-P2 junction          | Flow diversion     | Un-eventful  | –          | 0                   |
| Vilela & Goulão, 2006 | 24      | 2/M           | P2P                     | PAO w/ coils       | Un-eventful  | –          | 0                   |
| Ciceri et al., 2001 | 25       | 13/M          | P1-P2 junction          | Coiling            | Un-eventful  | –          | 0                   |
| Hallaq et al., 2002 | 26       | 47/M          | P2                      | PAO w/ coils       | Un-eventful  | –          | 0                   |
| Hamada et al., 2005 | 27       | 33/F          | P2                      | Surgical trapping  | Un-eventful  | –          | 0                   |
| Lv et al., 2009     | 28       | 37/F          | P2                      | PAO w/ coils       | Un-eventful  | –          | 0                   |
|                     | 29       | 48/M          | P2                      | PAO w/ coils       | Un-eventful  | –          | 0                   |
|                     | 30       | 31/M          | P2                      | PAO w/ coils       | Un-eventful  | –          | 0                   |
|                     | 31       | 5/M           | P2                      | PAO w/ coils       | Un-eventful  | –          | 0                   |

CONTINUED ON PAGE 5
With the advent of endovascular devices and sophisticated techniques, endovascular treatment has become dominant, especially for posterior circulation aneurysms. Endovascular PAO as a deconstructive method is considered safe for distal PCA lesions because of the rich collateral circulation. Conversely, endovascular PAO of the P1 segment has potential risks of ischemic complications in the occipital cortex or perforator territories.\(^1,2,4\) PAO of the P2 segment is safe except for fetal-type PCA, which has the risk of perforator territory infarcts after PAO.\(^5\) Balloon test occlusion (BTO) could be useful in evaluating PAO tolerance of the proximal PCA. The incidence of infarction following PAO decreases from 60% to 20% by BTO.\(^6\) However, because of the risk of rebleeding and inaccuracy of the examination, BTO should be used with caution in the acute stage of SAH. Reconstructive endovascular therapy, including stent-assisted coil embolization or flow diverter therapy, is an alternative option, especially in cases without PAO tolerance.\(^7\) These reconstructive endovascular treatments preserve circulation not only to the cortex but also to the perforators. However, some risks of rebleeding after the treatment may be present.\(^1,9\) Recent reports described that perforator territory ischemia and in-stent stenosis or occlusion could occur in case of flow diverter therapy.\(^2,22\) Both the stent-assisted coil embolization and flow diverter therapy require dual antplatelet therapy during the periprocedural period, which may increase postoperative rebleeding risk. Moreover, stent use, including flow diversion for the acute stage of SAH, is not approved in Japan because of its risk for thromboembolic complications.

**Observations**

Surgical treatment of proximal PCA dissecting aneurysms is used with caution because of its technically challenging deep location. However, surgical trapping with revascularization therapy is still the safest strategy to prevent rebleeding\(^8\) because perforator occlusion can be avoided under direct microscopic view. In this case, because the perforators originated from the dissected segment, trapping was incompletely performed. However, intraaneurysmal pressure was insufficiently decreased to prevent rebleeding because only the blood flow from the tiny PcomA was left after the trapping. STA-PCA bypass was effective in preventing the risk of PCA territory infarctions. Surgical treatment is considered the treatment of choice.

**Lessons**

Surgical trapping with STA-PCA bypass could be the treatment of choice for proximal PCA dissecting aneurysms, considering its curability and prevention of ischemic complications.

### TABLE 1. Cases of ruptured dissecting aneurysms of proximal PCA

| Authors & Year | Case No | Age (yrs)/Sex | Location | Treatment | Outcome | Recurrence | Outcome (mRS Score) |
|---------------|---------|--------------|----------|-----------|---------|------------|-------------------|
| Nistri et al., 2007\(^16\) | 32 | 58/M | P1-P2 junction | Conservative | Uneventful | – | 0 |
| Present case | 33 | 54/F | P1-P2 junction | Trapping w/ bypass | Transient oculomotor palsy | – | 0 |

*CONTINUED FROM PAGE 4*

+ = yes; – = no; PAO = parent artery occlusion; SAC = stent-assisted coil.

* Although SAC had been performed, rebleeding occurred and PAO was performed.

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Disclosures
The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

Author Contributions
Conception and design: Endo, Hayashi. Acquisition of data: Endo, Hayashi. Analysis and interpretation of data: Endo, Hayashi. Drafting the article: Hayashi, Kanoke. Critically revising the article: Endo. Reviewed submitted version of manuscript: Endo, Kawaguchi. Approved the final version of the manuscript on behalf of all authors: Endo. Administrative/technical/material support: Kawaguchi. Study supervision: Tominaga.

Supplemental Information
Video
Video 1. https://vimeo.com/744614120.

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