Deconstructive Endovascular Treatment of Ruptured Serpentine Basilar Artery Aneurysm by Mid-Basilar Occlusion

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Key words

- Basilar artery aneurysm
- Endovascular internal trapping
- Interventional neurosurgery
- Medullary infarction
- Parent artery embolization
- Tortuous aneurysm

Abbreviations and Acronyms

BA: Basilar artery CT: Computed tomography PComA: Posterior communicating artery SCA: Superior cerebellar artery

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INTRODUCTION

Direct surgery on aneurysms of the main part of the basilar artery (BA) is difficult to perform, and endovascular treatment is therefore often preferred. Recently, reports on treatments that preserve the parent artery with a stent are increasing.¹ However, in cases in which stent placement is difficult, occlusion of the parent vessel can be an alternative but it has a high risk of serious damage due to infarction caused by a perforator branching from the parent artery.²⁻⁴ In this study, we performed a careful anatomic evaluation of a BA aneurysm with subarachnoid hemorrhage finding it to be a large cerebral serpentine aneurysm. In this case, we performed parent artery embolization after hemodynamic evaluation obtaining positive outcomes.

BACKGROUND: No clear treatment course for cerebral aneurysms in the main trunk of the basilar artery (BA) has been established thus far.

CASE DESCRIPTION: A 69-year-old man with a history of hypertension presented to the emergency department with a sudden headache followed by decreased consciousness. Head computed tomography revealed a subarachnoid hemorrhage from the front of the brain stem to the basal cistern, and a lump-like dilation localized in the main trunk of the BA in the front of the midbrain. A rotated 360° large cerebral serpentine aneurysm was observed from the upper BA to the proximal part of the superior cerebellar artery. Endovascular treatment was performed under general anesthesia. To avoid a perforator infarction, shortsegment parent artery embolization was performed only at the rupture point and the proximal part of the aneurysm. A magnetic resonance imaging scan 1 day after surgery revealed only microinfarction in the pons just below the embolus. The patient developed mild left hemiparesis postoperatively but soon improved and was discharged from the hospital.

CONCLUSIONS: Parent vessel sacrifice carries a risk of perforator injury due to extension of thrombosis from the blind pouch into eloquent perforator vessels. Minimizing this risk requires careful evaluation of anatomy and strategic occlusion, which preserves runoff into relatively large vessels, such as the superior cerebellar artery, the anterior inferior cerebellar artery, or the penetrating branch from the blind pouch, as much as possible.

CASE DESCRIPTION

This study was approved by our institutional review board, which waived the need for informed consent owing to the retrospective nature of the report. A 69-year-old man was transported to the emergency department and exhibited impaired consciousness with a Glasgow Coma Scale score of 14 (E3V5M6). Physical examination revealed 3-mm diameter pupils and a blood pressure of 245/168 mm Hg, but he experienced no paralysis. The patient had a history of hypertension although no significant familial medical history of arterial disease.

A head computed tomography (CT) scan revealed subarachnoid hemorrhage in the pontocerebellar cistern, suprasellar cistern, and interpeduncular cistern. CT angiography revealed a 13-mm-sized aneurysm-like dilation projecting to the left in the main BA trunk (Figure 1A-C).

The patient was diagnosed with subarachnoid hemorrhage classified as Hunt and Kosnik grade III or World Federation of Neurological Surgeons grade II due to a ruptured cerebral aneurysm of the main BA trunk. Cerebral angiography was performed under general anesthesia on the first day of hospitalization.

A large serpentine aneurysm rotated 360° from the upper BA to the proximal part of the superior cerebellar artery (SCA) was observed (Figure 2A). A balloon occlusion test performed near the aneurysm showed retrograde circulation from the anterior circulation to the bilateral SCAs and into the aneurysm via the bilateral posterior communicating arteries (PComAs) (Figure 2B). As sufficient collateral blood flow was expected, and parent vessel occlusion was planned.

To avoid perforator infarction at the blind end, short-segment parent artery



Figure 1. (A) Computed tomography scan on admission showing subarachnoid hemorrhage in basal cistern. (B, C) A 13-mm-sized

aneurysm-like dilation projecting to the left in the main part of the basilar artery.

embolization was performed only at the bleb that appeared to be the rupture point and the proximal part of the aneurysm, and the distal part of the aneurysm was left without embolization (Figure 2C-E). After embolization, we confirmed that the SCA and the distal part of the aneurysm could be imaged from the anterior circulation.

There was no postoperative bleeding. A magnetic resonance imaging scan I day after surgery revealed a microinfarction in the pons just below the embolus (Figure 3A and B). Mild left hemiparesis appeared but soon improved. The patient was discharged from the hospital with a modified Rankin Scale score of one 27 days after admission.

DISCUSSION

Cerebral aneurysms occurring in the main part of the BAs account for approximately 2.1% of all aneurysms. Compared with other aneurysms, the incidence of poor outcome (modified Rankin Scale score 3–6) during treatment (85.7%) and perforator branch infarction (78.6%) is high; hence cerebral aneurysm is recognized as a difficult condition to treat.⁵ In our case, the BA had a significantly twisted and rotated structure, which is associated with dolichoectatic and serpentine aneurysms, but the definitions of these conditions are often confusing. The former is called vertebrobasilar dolichoectasia when the vertebral/BA is elongated, distended, and tortuous over a wide area,⁶ and dolichoectatic aneurysm if a giant fusiform aneurysm is included, which is thought to be caused by arterial wall dissection or arteriosclerosis. In contrast, the latter has a thin, "snakelike" vascular structure that penetrates thrombosed giant aneurysms, and the most common site is the middle cerebral arterv.7,8 This case differs from dolichoectatic aneurysm because the dilation is confined to part of the BAs, and the proximal vertebrobasilar artery is unaffected. Moreover, although the evaluation of the thrombotic areas was ambiguous, this case also differs from typical serpentine aneurysm cases on the low embolus volume; thus it seemed to be a unique case in terms of onset location and morphology.

Current treatment methods are direct surgery and endovascular treatment. In direct surgery, neck clipping is often difficult, especially for large, thrombosed aneurysms. For such special aneurysms, in addition to palliative treatment, such as wrapping, clipping after thrombus removal with bypass, proximal occlusion with bypass, and distal occlusion with bypass, can be considered.^{9,10} However, in addition to involving a surgical intervention, these methods are difficult because of the deep aneurysm location, the dense cranial nerves, and the perforating branch of the brain stem. Further, including bypass surgery makes the whole procedure difficult to complete without complications.^{TI-15} However, endovascular treatments involve internal trapping, stent-assisted coiling, multiple stenting, and the use of flow diverter devices; however, the approach itself is much easier than direct surgery, ^{I4-16} therefore the number of endovascular treatments has significantly increased.^{T7-19}

In this case, the aneurysm-like dilated portion of the vessel was twisted, and thus preoperative CT angiography was not sufficient to differentiate it from a saccular aneurysm. The morphology could be clearly observed through cerebral angiography, which added information on the time axis. Although the possibility of stent-assisted coiling was examined before angiography, placing the stent was hardly possible because of the significantly twisted and rotated vascular structure, so we finally decided to perform parent artery occlusion. The procedure was performed after confirming retrograde blood flow from the PComA to the distal aneurysm by occluding the balloon at the proximal part of the aneurvsm.

Ischemic complications associated with parent artery occlusion of the posterior circulation are reported to occur in 13%–17% of proximal basilar or bilateral vertebral artery occlusions ("hunterian ligation") by



Figure 2. Intraoperative findings. (A) Balloon indwelling site (vertebral angiography, front view): a balloon was introduced proximal to the aneurysm. (B) Balloon occlusion test findings (internal carotid angiography, lateral view): retrograde imaging from the anterior circulation through posterior communicating artery (PComA) into the aneurysm. (\rightarrow PComA,

← intraaneurysmal imaging site). (C) Postembolic cerebral angiography (vertebral angiography, front view). (D) Simple photography of the embolus (front view). (E) Schema of the embolus (front view). AICA, anterior inferior cerebellar artery; PCA, posterior cerebral artery; SCA, superior cerebellar artery.

direct surgery.^{20,21} However, there is no comprehensive report on endovascular treatments. Although it is difficult to completely predict the occurrence of ischemia after surgery associated with parent artery occlusion in endovascular treatment, it is possible to make some estimations. In this study, we used I) collateral blood circulation of the PComA, and 2) avoidance of blind ends.

First, the major contributor to the onset of ischemia after BA occlusion is the presence or absence of collateral blood flow, in other words, the degree of development of the PComA, which is the largest collateral blood flow in the BAs.²⁰ Ischemic complications are recognized if 4%-8%of bilateral PComAs are ≥ 1 mm, o-26%of one PComA is ≥ 1 mm, and 43%-45%of bilateral PComAs are <1 mm. In this case, bilateral PComAs had a lumen of $>_{I}$ mm. In addition, balloon occlusion test was effective because the retrograde blood flow from PComA could be confirmed angiographically.

Second, the perforator branch infarction occurs at a high rate due to delayed thrombosis of the blind end of the obstruction during parent artery occlusion. Therefore it is important to consider





trapping in which the high-flow blood vessels serve as flow outlets and the blind end is shortened as much as possible. In this case, the bilateral SCAs and anterior inferior cerebellar arteries, which are highflow blood vessels, were left and the blind end shortened. Although a part of the BA and the aneurysm inevitably became the blind end. Therefore there were concerns about perforator infarction occurring from the site. Although it is not clear how thick it should be to become a flow outlet and avoid thrombosis, a study showed that thrombosis does not occur even if the branch is thin.^{3,4,22-25} Therefore efforts should be made for short-distance occlusion maintaining the perforator branch as intact as possible. In this case, to preserve as much as possible the penetrating branch generated from the aneurysm, a short-segment parent artery embolization centered on the bleb was performed, which seemed to be the rupture point. Although a microinfarction appeared postoperatively, retrograde blood flow was confirmed from the anterior circulation to the distal aneurysm even I week after embolization, suggesting that thrombosis due to blind end could be avoided.

CONCLUSIONS

Parent vessel sacrifice carries a risk of perforator injury due to extension of

thrombosis from the blind pouch into eloquent perforator vessels. Minimizing this risk requires careful evaluation of anatomy and strategic short-segment occlusion, which preserves runoff into relatively large vessels or the penetrating branch from the blind pouch as much as possible.

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