Online May 17, 2022
Journal of Neuroendovascular Therapy 2022; 16: 474–480 DOI: 10.5797/jnet.cr.2021-0105

Case Report

Internal Trapping of an Acutely Ruptured Dissecting Aneurysm of a Dominant Vertebral Artery Following Balloon Test Occlusion: A Case Report

Takahiro Ogishima,[1] Osamu Tone,[2] Yohei Sato,[1] and Masashi Tamaki[1]

Objective: To report a case of an acutely ruptured vertebral artery dissecting aneurysm (VADA) with a hypoplastic contralateral vertebral artery (VA) successfully treated with internal trapping following the estimation of the collateral flow from anterior circulation.

Case Presentation: A 46-year-old woman was diagnosed with subarachnoid hemorrhage and acute hydrocephalus. Ventriculostomy was performed under general anesthesia. CTA revealed a left VADA distal to the origin of the left posterior inferior cerebellar artery (PICA). The right VA was hypoplastic, and the right posterior communicating artery (Pcom) was fetal type. We performed balloon test occlusion (BTO) of the VA proximal to the origin of the left PICA and estimated sufficient collateral blood flow via the right Pcom and basilar artery (BA) to the anterior spinal artery (ASA) and the left PICA. Internal trapping of the left VADA was then performed. The angiograms after internal trapping revealed collateral flow from the right Pcom to the BA, and the hypoplastic right VA perfused the proximal BA and ASA. She recovered without any neurological deficits following antiplatelet therapy and vasospasm treatment. She was followed up for 6 years without any neurological events occurring.

Conclusion: When BTO indicates sufficient collateral flow, internal trapping could be a useful treatment for acutely ruptured VADAs on the dominant side, given a complete understanding of the angioarchitecture and the risk of vasospasm due to subarachnoid hemorrhage.

Keywords subarachnoid hemorrhage, collateral blood circulation, posterior communicating artery, cerebral blood flow, cerebral vasospasm

Introduction

Vertebral artery dissecting aneurysms (VADAs) account for 4.5% of the autopsy cases of spontaneous subarachnoid hemorrhage (SAH).[1)] The clinical course of ruptured VADAs is characterized by frequent early rebleeding and a highly fatal outcome.[2)] Treatment strategies for VADAs depend on the location of the aneurysm, the origin of the posterior inferior cerebellar artery (PICA), and dominance of the contralateral vertebral artery (VA).[3,4)] Internal trapping is considered the first treatment choice for acutely ruptured VADAs; however, internal trapping and bypass surgery, or stent-assisted coiling (SAC), preserving the parent VA are selected for ruptured VADAs of the dominant VA.[3–6)] The contralateral VA is mainly considered in the treatment strategies for acutely ruptured VADAs.[3)] The collateral blood flow via the posterior communicating artery (Pcom) has been previously used to treat bilateral VADAs.[7)]

Herein, we report a case of an acutely ruptured VADA with an apparent hypoplastic contralateral VA, treated with internal trapping of the VADA following balloon test occlusion (BTO) proximal to the ipsilateral VA, confirming the collateral flow via the Pcom and basilar artery (BA).

[1]*Department of Neurosurgery, Japanese Red Cross Musashino Hospital, Musashino, Tokyo, Japan*
[2]*Stroke Center, Ome Municipal General Hospital, Ome, Tokyo, Japan*

Received: December 23, 2021; Accepted: April 10, 2022
Corresponding author: Osamu Tone. Stroke Center, Ome Municipal General Hospital, 4-16-5, Higashiome, Ome, Tokyo 198-0042, Japan
Email: tone_0559@yahoo.co.jp

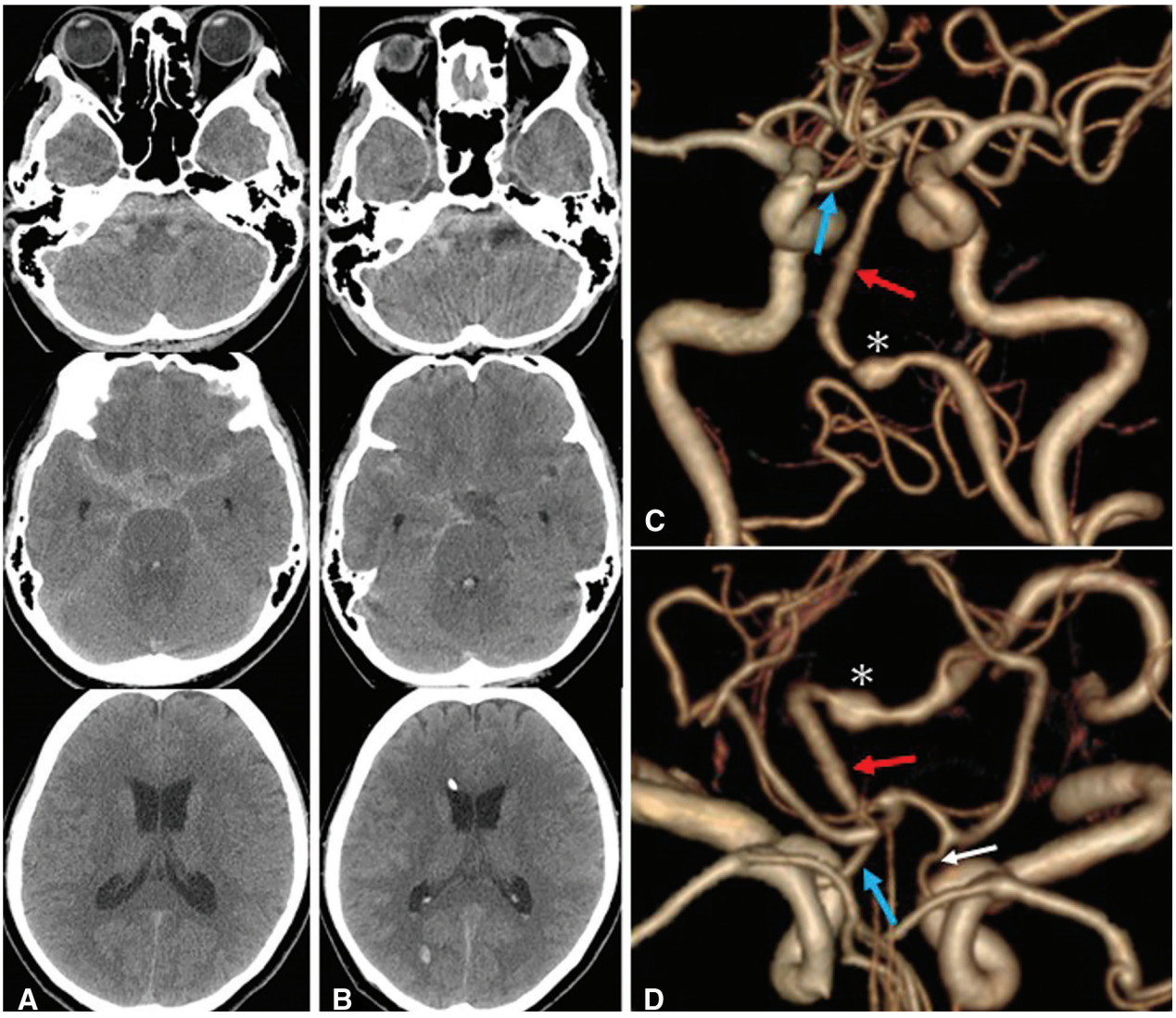

Fig. 1 CT and CTA on admission. (**A**) CT on admission indicating SAH with a predominant volume of the cisternal clot of the posterior fossa. (**B**) CT 1 day after the ventriculostomy indicating a decrease in the clot. (**C** and **D**) CTA on admission reveals the left VA dissecting aneurysm (asterisks) distal to the origin of the left PICA, showing the pearl-and-string sign, right fetal-type Pcom (blue arrows), left adult-type Pcom (white arrow), and basilar artery (red arrows). (**C**) Anteroposterior view and (**D**) superoinferior view. PICA: posterior inferior cerebellar artery; Pcom: posterior communicating artery; SAH: subarachnoid hemorrhage; VA: vertebral artery

Case Presentation

A 46-year-old woman who experienced headache 3 days prior suddenly fell into a coma and was transferred to our hospital. On admission, she was drowsy, and CT revealed SAH and acute hydrocephalus. The distribution of cisternal clots was predominant in the posterior cranial cisterns (**Fig. 1A**). She had no history of diseases except hypertension without medication, and her routine preoperative evaluation results were within normal ranges. We performed emergent ventriculostomy under general anesthesia and continued deep sedation. CT on the day after ventriculostomy indicated a decrease in the cisternal clot (**Fig. 1B**). CTA (**Fig. 1C** and **1D**) on admission revealed a left VADA, showing a pearl-and-string sign, distal to the origin of the left PICA. The V4 segment of the right VA was apparently hypoplastic, and the right Pcom was fetal type. Subsequently, we performed cerebral angiography and BTO to confirm collateral flow. Left VA angiograms (**Fig. 2A**–**2D**) showed the left VADA between the origins of the left PICA and the anterior spinal artery (ASA). The right VA angiogram showed a hypoplastic V4 segment and the BA in the anterograde flow of laminae (**Fig. 2E**). Systemic heparinization was initiated until reaching an

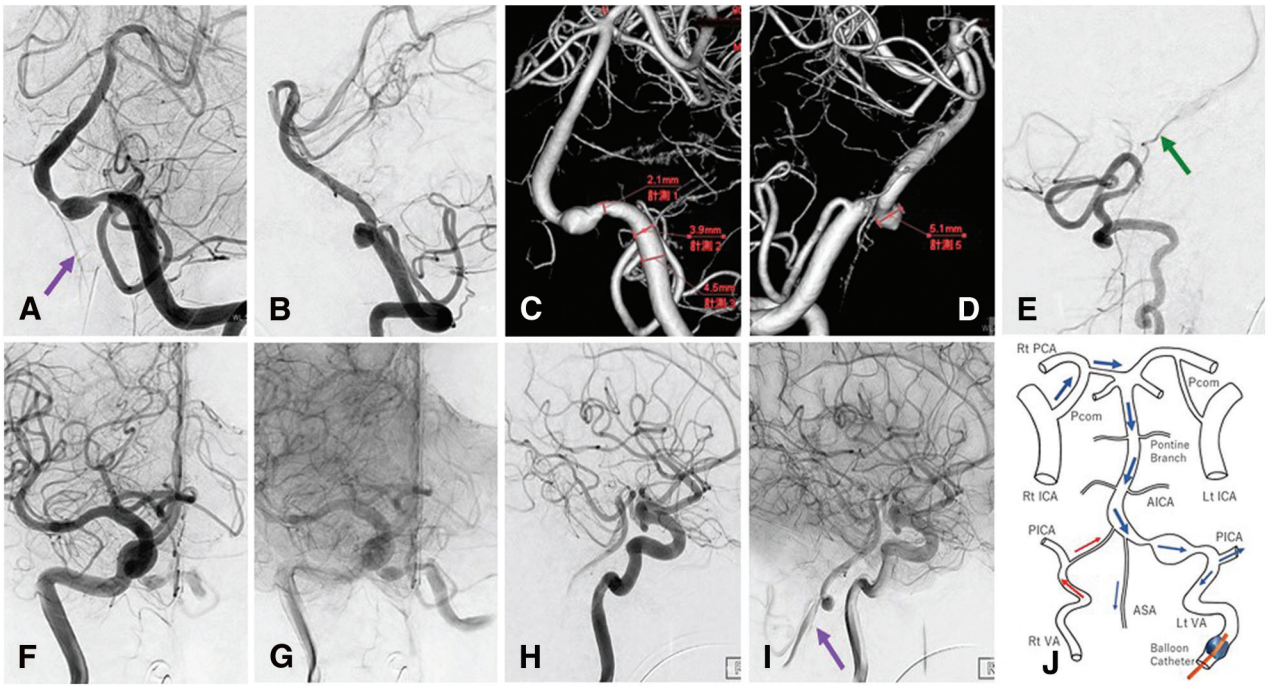

Fig. 2 VA angiographies and BTO of the left proximal VA before internal trapping. (**A**–**D**) Conventional (**A** and **B**) and three-dimensional (**C** and **D**) left VA angiograms indicate a left VA dissecting aneurysm between the origin of the left PICA and the ASA (purple arrow). (**E**) Right VA angiogram indicates the hypoplastic V4 segment (green arrow). (**F**–**I**) Right internal carotid angiograms following the BTO of the left V2 segment indicate collateral flow via the right Pcom and the basilar artery to the ASA, the VA aneurysm, and the left PICA. (**J**) The collateral flow patterns following the BTO. (**A**, **C**, **F**, and **G**) Anteroposterior view and (**B**, **D**, **E**, **H**, and **I**): lateral view. AICA: anterior inferior cerebellar artery; ASA: anterior spinal artery; BTO: balloon test occlusion; ICA: internal carotid artery; Lt: left; PCA: posterior cerebral artery; Pcom: posterior communicating artery; PICA: posterior inferior cerebellar artery; Rt: right; VA: vertebral artery

activated clotting time (ACT) longer than twice that during pre-heparinization, from 98 s to 219 s. Selecon MP Catheter II 5.2 F (Terumo Clinical Supply, Gifu, Japan) was introduced to the left VA (V2 segment), and BTO was performed by the occlusion of the left VA proximal to the origin of the left PICA. The right internal carotid angiogram revealed the ASA, VADA, and left PICA via the right Pcom and BA (**Fig. 2F**–**2I**). We estimated the collateral blood flow via right Pcom as enough to perfuse the BA, ASA, and the branches of these arteries. Therefore, we did not perform the angiograms of the right VA and left internal carotid artery following BTO of the left VA. After the balloon deflation, systemic heparin was reversed using protamine sulfate. The occlusion time of the left V2 segment was 1 min and 20 s. **Figure 2J** shows the collateral flow patterns following BTO.

After obtaining written informed consent from the legal representatives, we administered aspirin 200 mg, clopidogrel 150 mg, and ozagrel sodium 80 mg. We then performed internal trapping of the VADA, including the short proximal portion of the left VA (**Fig. 3A** and **3B**) using Target coils (total length of 84 cm; Stryker, Fremont, CA, USA) under general anesthesia and systemic heparinization (ACT from 117 s to 254 s) on the next day of BTO. Following internal trapping of the VADA, a right internal carotid angiogram revealed slow retrograde BA flow (**Fig. 3C**–**3E**). A left internal carotid angiogram revealed the left P1 segment but not the BA (not shown), and a right VA angiogram revealed the proximal BA and ASA (**Fig. 3F**–**3H**). **Figure 3I** shows the collateral flow patterns following the internal trapping of the aneurysm.

Postoperatively, we administered intravenous heparin 15000 U/day, ozagrel sodium 160 mg/day for 2 days, oral clopidogrel 75 mg/day for 20 days, and aspirin 100 mg/day for more than 6 years. For the treatment of vasospasm, fasudil hydrochloride hydrate 90 mg/day for 2 weeks, nicardipine 3 mg–6 mg/hr for 3 weeks, dobutamine for 11 days, and low-molecular-weight dextran for 14 days were administered.

Although diffusion-weighted MRI on the day after internal trapping indicated some ischemic spots of the cerebellar hemisphere and the time-of-flight MRA did not show the BA, no ischemic lesions of the brainstem were detected (**Fig. 4A** and **4B**). The MRA findings of the BA were consistent until 5 years later (**Fig. 4C**). Single-photon emission CT using ^{99m}Tc-ethyl cysteinate dimer performed

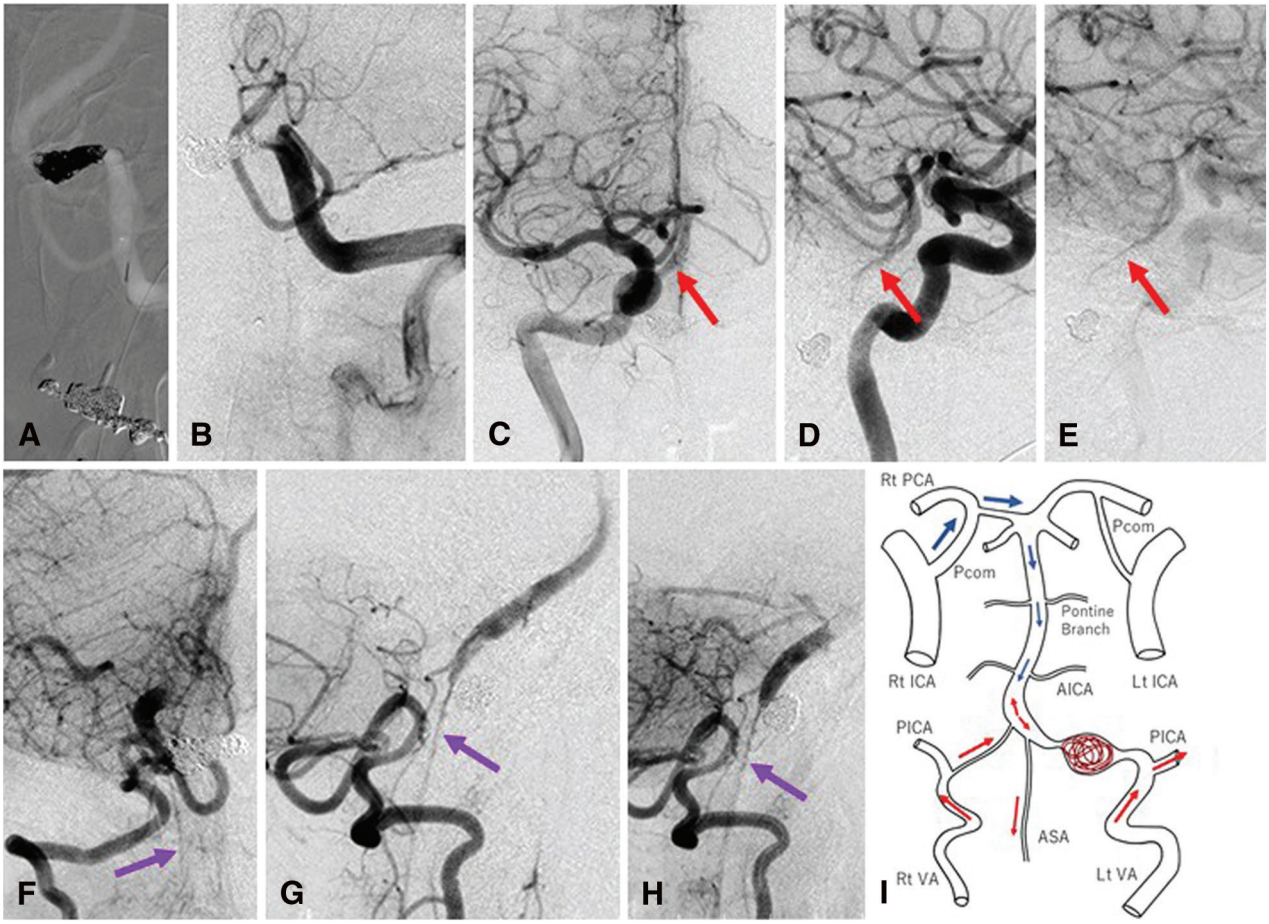

Fig. 3 Internal trapping of the VADA and the altered collateral flow. (**A**, road map) and (**B**, subtracted): Left VA angiograms showing internal trapping of the aneurysm. (**C**–**E**) Right carotid angiograms of the arterial (**C** and **D**) and capillary (**E**) phases indicate a slow flow of the basilar artery (red arrows) but not the ASA. (**F**–**H**) Right VA angiograms following internal trapping of the aneurysm of the arterial (**F** and **G**) and capillary (**H**) phases showing the ASA (purple arrows). (**I**) The collateral flow patterns following internal trapping of the aneurysm. (**A** and **B**) Right anterior oblique view, (**C** and **F**) anteroposterior view, and (**D**, **E**, **G**, and **H**) lateral view. AICA: anterior inferior cerebellar artery; ASA: anterior spinal artery; ICA: internal carotid artery; Lt: left; PCA: posterior cerebral artery; Pcom: posterior communicating artery; PICA: posterior inferior cerebellar artery; Rt: right; VA: vertebral artery; VADA: vertebral artery dissecting aneurysm

3 days, 24 days, and 6 years later (**Fig. 4D**–**4F**) indicated normal cerebral blood flow (CBF), including in the brainstem. She recovered without any neurological deficits and was followed up for 6 years without any neurological events occurring. The CTA 6 years later (**Fig. 4G** and **4H**) indicated a narrowed BA and a slightly dilated right Pcom and P1 segment.

Discussion

In this case report, an acutely ruptured dissecting aneurysm of a dominant VA with an apparently hypoplastic contralateral VA was successfully treated with internal trapping of the aneurysm following BTO of the proximal dominant VA. The postoperative administration of anticoagulants and antiplatelets and the treatment of vasospasm resulted in no neurological deficits. Furthermore, long-term follow-up (more than 6 years) revealed complete ischemic tolerance of the internal trapping of the dominant VADA. This is the first report of acutely ruptured VADA treated by internal trapping, confirmed by sufficient collateral flow from anterior circulation, and followed up for many years, to the best of our knowledge.

Endovascular treatment strategies for ruptured VADAs have been reported to select the deconstructive techniques, e.g., internal trapping with or without bypass surgeries, and the reconstructive techniques, e.g., SAC. The ruptured VADA, in this case, was located on the V4 segment of the dominant side, between the origins of PICA and ASA. Therefore, reconstructive treatment will be selected, barring an ischemic tolerance.[3,4]

Although SAC is a treatment to preserve the flow of the parent artery, it is technically more demanding than internal trapping, especially during the period of acute rupture.

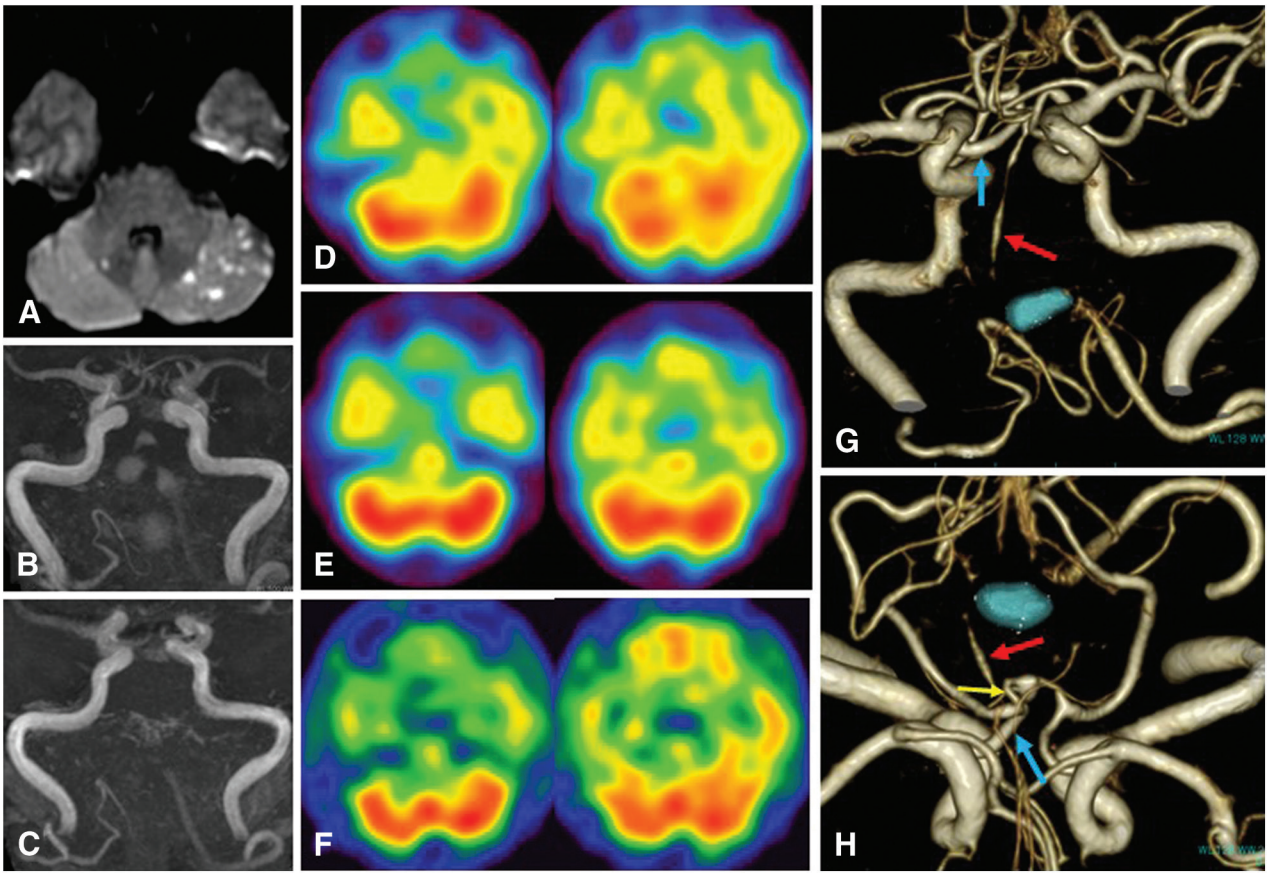

Fig. 4 MRI, SPECT, and CTA following internal trapping. Diffusion-weighted MRI (**A**) and time-of-flight MRA (**B**) images, 1 day after internal trapping, showing some thromboembolic findings of the left cerebellar hemisphere and no opacification of the BA. The findings of MRA of the BA were consistent until 5 years later (**C**). CBF measurements using ^{99m}Tc-ECD SPECT 3 days (**D**), 24 days (**E**), and 6 years (**F**) following internal trapping, showing no abnormal findings of the brainstem and cerebellum. (**G** and **H**) CTA 6 years later indicates the narrowed BA (red arrows) and the slightly dilated right Pcom (blue arrows) and right P1 segment (yellow arrows). BA: basilar artery; CBF: cerebral blood flow; ECD: ethyl cysteinate dimmer; Pcom: posterior communicating artery; SPECT: single-photon emission computed tomography

In our case, the diameter of the string portion of VADA was 2.1 mm. The risk of thromboembolic complication of SAC is the main reason for the off-label use of SAC for acutely ruptured aneurysms, and the small diameter of the parent artery is not suitable for stenting because of the risk of in-stent thrombosis.[8] Recently, flow diverter treatment of ruptured VADAs has been reported.[9–11] Although flow diverter is also an off-label treatment for acutely ruptured aneurysms as SAC, flow diverter could become an alternative treatment of acutely ruptured VADAs in the future.[10,11]

Sönmez et al.[12] reported a meta-analysis of long-term outcomes between internal trapping and SAC of VADA. The long-term complete occlusion rate of deconstructive techniques (88%) was significantly higher than that of reconstructive techniques (81%). Furthermore, SAC has been reported to be inferior in preventing aneurysmal rebleeding compared to internal trapping. Madaelil et al.[4] reviewed case series of ruptured VADAs from the literature with a comparison of the treatment types, internal trapping (197 cases), SAC (31 cases), and proximal occlusion (26 cases), and indicated the frequency of recurrent hemorrhage as 3.1%, 6.4%, and 19%, respectively.

Preoperative BTO of the VA has been reported for treating unruptured giant aneurysms of the BA.[7,13,14] Complete BTO is reported to function by BTO with induced hypotension, CBF measurement, and neurophysiologic monitoring.[14,15] Because the patient was emergent and under deep sedation, we estimated BTO mainly with angiographic findings. However, combining the angiography with neurophysiologic monitoring, such as the auditory brainstem response, motor evoked potential, or somatosensory evoked potential, could help estimate the ischemic tolerance in more detail.

In this case, because the VADA was located distal to the origin of the left PICA, BTO was performed proximal to the PICA origin. Therefore, the demanded flow from the Pcom decreased following internal trapping of the VADA, and the right hypoplastic VA started to perfuse the ASA. The mechanism of this diversion of the roles of collateral flow is not fully understood. We thought the collateral flow via the right Pcom and the BA as sufficient. Therefore, we did not perform the angiograms of the right VA and the left internal carotid artery following BTO. To estimate complete collateral flows, these angiograms should be performed following BTO.

The precise balloon location to estimate the collateral flows following aneurysmal internal trapping is considered to be the left V4 segment between the aneurysmal proximal end and the origin of the left PICA. However, we hesitated to inflate a micro balloon to occlude this segment close to the dissecting aneurysm for the risk of aneurysmal rebleeding.

Because the diameters of bilateral anterior inferior cerebellar arteries and the perforators of BA were apparently smaller than the diameters of bilateral PICAs and SCAs, the blood flow perfusion of the posterior fossa mainly depended on bilateral PICAs and SCAs. These angioarchitectures of blood flow could be essential to estimate ischemic tolerance following internal trapping of VADAs.

In performing internal trapping of a dominant VA to treat acutely ruptured VADAs, vasospasm following SAH must be considered. In this case, ventriculostomy on admission rapidly drained the cisternal clot; other medical treatments using fasudil and nicardipine are also considered effective.[16,17]

An important ischemic complication of internal trapping of VADAs is medullary infarction. In this case, the aneurysmal morphology enabled tight internal trapping inside the aneurysmal pearl portion and the proximal short VA segment. The origin of the perforators of the V4 segment is reported to range mainly from 14 mm below to the vertebrobasilar junction.[18] Internal trapping of the short segment of the VA could be a strategy to prevent medullary infarction because the internal trapping of the long segment has been reported to be a risk factor for medullary infarction.[19,20] To avoid medullary infarction due to the direct occlusion of perforating arteries of VA, proximal occlusion could be one of the treatments. However, according to the literature review, the frequencies of symptomatic ischemic complication and favorable outcome of internal trapping vs. proximal occlusion were reported as 5.3% vs. 17% and 75% vs. 50%, respectively, indicating the poor results of proximal occlusion.[4]

In this case, postoperative diffusion-weighted MRI indicated the ischemic lesions of the left cerebellar hemisphere. We performed both procedures, the preoperative BTO and the internal trapping, under systemic heparinization. Dual antiplatelet loading was also started before internal trapping. The anticoagulant therapy was continued for 2 days and antiplatelet therapy for years, to prevent the occlusion of the branches and perforators of the BA and the VA following internal trapping and flow reduction. However, the asymptomatic cerebellar ischemic lesions could not be avoided.

Conclusion

When BTO indicates sufficient collateral flow, internal trapping could be a useful treatment for acutely ruptured VADAs on the dominant side, with a hypoplastic contralateral VA, given a complete understanding of the angioarchitecture of anterior and posterior circulation and the risk of vasospasm due to SAH.

Acknowledgments

The authors thank Dr. Shuichi Tomida for his careful follow-up of the patient and Ms. Yoko Tone for illustrations.

Disclosure Statement

The authors declare that they have no conflicts of interest.

References

1) Sasaki O, Ogawa H, Koike T, et al. A clinicopathological study of dissecting aneurysms of the intracranial vertebral artery. *J Neurosurg* 1991; 75: 874–882.
2) Yamaura A, Watanabe Y, Saeki N. Dissecting aneurysms of the intracranial vertebral artery. *J Neurosurg* 1990; 72: 183–188.
3) Lim SH, Shin HS, Lee SH, et al. Endovascular treatment of vertebral artery dissecting aneurysms that cause subarachnoid hemorrhage: Consideration of therapeutic approaches relevant to the angioarchitecture. *J Korean Neurosurg Soc* 2015; 58: 175–183.

4) Madaelil TP, Wallace AN, Chatterjee AN, et al. Endovascular parent vessel sacrifice in ruptured dissecting vertebral and posterior inferior cerebellar artery aneurysms: clinical outcomes and review of the literature. *J Neurointerv Surg* 2016; 8: 796–801.
5) Manabe H, Hatayama T, Hasegawa S, et al. Coil embolisation for ruptured vertebral artery dissection distal to the origin of the posterior inferior cerebellar artery. *Neuroradiology* 2000; 42: 384–387.
6) Kurata A, Ohmomo T, Miyasaka Y, et al. Coil embolization for the treatment of ruptured dissecting vertebral aneurysms. *AJNR Am J Neuroradiol* 2001; 22: 11–18.
7) Omahen DA, Findlay JM. A giant fusiform basilar aneurysm treated by bilateral vertebral artery occlusion. *J Clin Neurosci* 2004; 11: 324–328.
8) Chung J, Lim YC, Suh SH, et al. Stent-assisted coil embolization of ruptured wide-necked aneurysms in the acute period: incidence of and risk factors for periprocedural complications. *J Neurosurg* 2014; 121: 4–11.
9) Cerejo R, Bain M, Moore N, et al. Flow diverter treatment of intracranial vertebral artery dissecting pseudoaneurysms. *J Neurointerv Surg* 2017; 9: 1064–1068.
10) Maus V, Mpotsaris A, Dorn F, et al. The use of flow diverter in ruptured, dissecting intracranial aneurysms of the posterior circulation. *World Neurosurg* 2018; 111: e424–e433.
11) Schob S, Becher A, Bhogal P, et al. Segment occlusion vs. reconstruction. A single center experience with endovascular strategies for ruptured vertebrobasilar dissecting aneurysms. *Front Neurol* 2019; 10: 207.
12) Sönmez Ö, Brinjikji W, Murad MH, et al. Deconstructive and reconstructive techniques in treatment of vertebrobasilar dissecting aneurysms: A systematic review and meta-analysis. *AJNR Am J Neuroradiol* 2015; 36: 1293–1298.
13) Horie N, Kitagawa N, Morikawa M, et al. Giant thrombosed fusiform aneurysm at the basilar trunk successfully treated with endovascular coil occlusion following bypass surgery: a case report and review of the literature. *Neurol Res* 2007; 29: 842–846.
14) Jung YJ, Kim MS, Choi BY, et al. Fusiform aneurysm on the basilar artery trunk treated with intra-aneurysmal embolization with parent vessel occlusion after complete preoperative occlusion test. *J Korean Neurosurg Soc* 2013; 53: 235–240.
15) Standard SC, Ahuja A, Guterman LR, et al. Balloon test occlusion of the internal carotid artery with hypotensive challenge. *AJNR Am J Neuroradiol* 1995; 16: 1453–1458.
16) Haley EC, Kassell NF, Torner JC. A randomized trial of nicardipine in subarachnoid hemorrhage: angiographic and transcranial Doppler ultrasound results. A report of the Cooperative Aneurysm Study. *J Neurosurg* 1993; 78: 548–553.
17) Zhao J, Zhou D, Guo J, et al. Efficacy and safety of fasudil in patients with subarachnoid hemorrhage: final results of a randomized trial of fasudil versus nimodipine. *Neurol Med Chir (Tokyo)* 2011; 51: 679–683.
18) Mahmood A, Dujovny M, Torche M, et al. Microvascular anatomy of foramen caecum medullae oblongatae. *J Neurosurg* 1991; 75: 299–304.
19) Ikeda H, Imamura H, Mineharu Y, et al. Effect of coil packing proximal to the dilated segment on postoperative medullary infarction and prognosis following internal trapping for ruptured vertebral artery dissection. *Interv Neuroradiol* 2016; 22: 67–75.
20) Aihara M, Naito I, Shimizu T, et al. Predictive factors of medullary infarction after endovascular internal trapping using coils for vertebral artery dissecting aneurysms. *J Neurosurg* 2018; 129: 107–113.