Endovascular proximal artery occlusion is one of the traditional methods of treatment for ruptured vertebral artery dissecting aneurysm (rVADA). Proximal artery occlusion may be effective in the acute period, but is not enough to eradicate the risk of rebleeding (1). Mass effect usually is not a concern for a case of well occluded rVADA. However, we experienced a growing thrombosed aneurysm after rVADA occlusion, and conventional angiography did not reveal antegrade and retrograde luminal flow into the rVADA but faint contrast staining of the aneurismal periphery, which corresponded to a histological finding, proliferative vasa vasorum. We suggest that the vasa vasorum plays a crucial role in the growth of excluded rVADAs from the VA after endovascular trapping or proximal artery occlusion.

**CASE REPORT**

A 40-year-old man had a history of antihypertensive medication for one year. Twenty-five days before admission at our hospital, he had a severe headache and a brief loss of consciousness. Cerebral computed tomography (CT) images at another institution showed diffuse subarachnoid and intraventricular hemorrhage (Fig. 1A). The patient underwent cerebral angiography 2 weeks after the first symptom occurrence, which showed an 11-mm-diameter dissecting aneurysm involving the origin of the posterior inferior cerebellar artery (PICA) in the right VA, and diffuse luminal irregularities in bilateral VAs (Fig. 1B). On his first admission, he was submitted to a complete evaluation, which revealed no neurologic abnormalities. Magnetic resonance (MR) images revealed a dissecting aneurysm with subadventitial hematoma in the right distal VA (Fig. 1C). Internal trapping should have avoided due to the PICA arising from the dissecting aneurysm. Therefore, endovascular proximal artery occlusion was planned as the contralat-
eral VA was enough to be sizable to supply the entire posterior circulation. Using double-microcatheter technique, the right VA segment just proximal to the origin of the PICA was occluded with detachable coils, and a vascular plug (AGA Medical Corporation, Golden Valley, MN, USA) was placed in the upper V2 segment to reinforce the VA occlusion (Fig. 1D). At the end of the procedure, the right VA was completely occluded, and the anterior spinal artery and right PICA, and dissecting aneurysm were contrast-filled from the contralateral VA (Fig. 1E). Intravenous heparin was maintained for 24 hours postembolization, and the patient began to receive dual antiplatelet medication (aspirin and plavix). Thirteen days after treatment, follow-up angiography was performed and revealed stable occlusion of the right VA and near complete obliteration of the aneurysm; thereafter the patient was discharged in good condition without neurological deficits.

Three months after the initial treatment, whirling type dizziness and subtle headache ensued so that the patient visited the emergency room. In cerebral CT, there was no evidence of hemorrhage. But, MR images demonstrated marked enlargement of the occluded VADA indicating thrombus inside and compression of the medulla (Fig. 2A). Conventional angiography demonstrated stable occlusion of the right VADA but poorly demarcated enlarged 20-mm-diameter aneurysm with only aneurismal wall staining from unidentified feeders (Fig. 2B, C).

![Fig. 1. Preoperative and intraoperative images during the first procedure. A. CT image shows diffuse subarachnoid hemorrhage in the basal cistern and intraventricular hemorrhage in both lateral ventricles. B. 3D image of right vertebral angiography reveals a dissecting aneurysm (asterisk) in the right distal vertebral artery involving the PICA origin (arrow), and diffuse luminal irregularities in the right and left vertebral artery (not shown). C. Axial T2-weighted MR image demonstrates an aneurysm at the right distal VA, which is compressing the brain stem. D. Radiograph obtained after procedure demonstrates a coil mass in the proximal V4 segment and a vascular plug in the upper V2 (arrow). E. Left vertebral artery completion angiogram shows retrograde contrast-filling of the anterior spinal artery (arrowheads), PICA (arrows), and dissecting aneurysm (asterisk) from the left vertebral artery.](image-url)
During admission, the patient developed progressive neurological deficits, such as hoarseness, tongue deviation, and swallowing disturbance. We attempted to reduce the mass effect by surgical decompression as well as further trapping for the thrombosed growing aneurysm. The patient underwent far lateral craniotomy and partial aneurysmectomy followed by thrombectomy. During operation, we grossly ascertained the origin of the right PICA and anterior spinal artery as well as prominent vasa vasorum on the occluded VA and aneurismal neck (Fig. 3). Using surgical clips, the aneurysm was trapped between the portion immediately proximal to the PICA and the proximal end of the aneurysm. Then, decompression was performed by partial aneurysmectomy and partially organized thrombus was demonstrated in the aneurysm. Histological examination of the aneurismal wall confirmed proliferative vasa vasorum (Fig. 4).

At discharge, neurologic deficits still remained, but the deficits gradually improved and only dysphagia for liquid food persisted at 3-month follow-up. Further radiographic follow-ups were not performed.

DISCUSSION

For VADA, proximal artery occlusion is one of the effective treatment modalities. Proximal artery occlusion can be preferred in cases in which the dissected segment appears to be too risky to cross, or too fusiform or narrow to accept coils, or in cases in which the dissected segment incorporates important branches, including the anterior inferior cerebellar artery, the PICA, or the anterior spinal artery (1, 2). However, as this treatment involves only the arterial lumen apart from the dissection, one should concern about the risk of revascularization and rebleeding.

Enlargement of intracranial aneurysm has been explained by two mechanisms: 1. repeated hemodynamic injury to the aneurysm wall (3), 2. recurrent intramural hemorrhages at the highly vascularized wall (4). As the antegrade and retrograde luminal flow to the VADA was angiographically occluded in this case, the

![Intraoperative photograph reveals the well-developed vasa vasorum (arrows) of the right VA occluded with coils near the neck of the aneurysm (An).](image)

Fig. 3. Intraoperative photograph reveals the well-developed vasa vasorum (arrows) of the right VA occluded with coils near the neck of the aneurysm (An).

![MR images obtained on readmission to the hospital after symptom aggravation.](image)

Fig. 2. MR images obtained on readmission to the hospital after symptom aggravation. 
A. Axial T2-weighted MR images demonstrates a marked enlargement of the dissecting aneurysm indicating thrombus inside. 
B. Left vertebral artery angiogram shows no filling of the aneurysm. 
C. Right vertebral artery angiogram shows a marked staining around the coils (arrows) and mainly aneurismal wall (arrowheads).
enlargement of VADA was thought not to be associated with the first mechanism. However, right vertebral angiogram showed an extraordinary finding of faint aneurysmal wall staining from unidentified feeders. We believe that these feeders, arising from the plexiform anastomotic vessels among perforators or unidentified proper feeders of the vasa vasorum, supplied the proliferative vasa vasorum and neovascularized thrombus of the occluded VADA. Enlargement of the occluded VADA in this case may be explained by repeated minor hemorrhage from the vasa vasorum, and this has been postulated to account for thrombosed growing aneurysm in previous reports (5, 6).

The exact mechanisms arousing such a transmural vascular connection still remain unknown. The aggregation of inflammatory cells was noted in the unorganized thrombus around the coils, indicating that the inflammatory process may induce neovascularization around the neck of the aneurysm (7). Therefore, the development of transmural vascular connections between the well-developed vasa vasorum and the unorganized thrombus around the coils at the neck of the aneurysm appears to be a key event in continuous aneurysm growth, probably due to recurrent minor hemorrhage after endovascular treatment (6).

The vasa vasorum is defined as the microcirculation in the walls of mid- to large-sized arteries. The vessels originate from the branches of the external carotid artery and from the proximal internal carotid artery near the bifurcation (8). Data from recent studies have shown that intracranial arteries do not have vasa vasorum except for the proximal segment of the intracranial internal carotid artery and the VA piercing the dura (9). By contrast, increased density of vasa vasorum was also reported in the proximal segment of atherosclerotic intracranial arteries (10); in case of complete atherosclerotic occlusion of the internal carotid artery, the distal internal carotid artery was revascularized from small arterial channels, and some of these channels seem to represent hypertrophied vasa vasorum. Proliferation of the vasa vasorum into atherosclerotic plaques also has been described, which indicates their potential ability to remodel the anatomy of the parent artery (5).

In conclusion, thrombosis and growth can rarely ensue from rVADA exclusion after endovascular proximal artery occlusion. The angiographic, surgical, and histologic specimen suggested that the growing mechanism should be associated with hypertrophied vasa vasorum and repeated minor hemorrhage on the aneurysm wall. Total exclusion of rVADA by endovascular trapping or proximal artery occlusion does not guarantee a durable cure in terms of aneurismal growth and ensuing mass effect.

References

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