Case Report

Successful Treatment of Bilateral Vertebral Artery Dissecting Aneurysms with Subarachnoid Hemorrhage: Report of Three Cases

Takeo Nashimoto, MD, PhD,* Tadashi Komata, MD, PhD,† Junpei Honma, MD, PhD,‡ Sinya Yamashita, MD, PhD,‡ Yasuhiro Seki, MD, PhD,* Akihiko Kurashima, MD, PhD,* and Takafumi Saito, MD, PhD*

Vertebral artery dissecting aneurysm (VADA) is a relatively rare cause of subarachnoid hemorrhage (SAH). Bilateral VADAs are even rare, and management strategies for this type of VADAs are still controversial. Here, we report 3 cases of bilateral VADAs with SAH. All 3 patients were treated conservatively under strict sedation and blood pressure control during the acute stage. During the course, rebleeding was not observed in any case. One patient underwent trapping of the ruptured VADA on day 28, because this lesion was considered to have a high tendency to rebleed, even in the chronic stage. In the other 2 patients, after conservative treatment, the VADAs spontaneously resolved on the both sides. As for the therapeutic strategy for bilateral VADAs presenting with SAH, at the acute stage, considering the difficulty of bypass surgery, we recommend conservative treatment with sedation and strict control of blood pressure. At the chronic stage, however, when the VADA is still large and growing in size, surgical treatment such as proximal occlusion or trapping of the affected VA with or without distal revascularization should be considered to avoid rebleeding. **Key Words:** Bilateral—dissecting aneurysm—subarachnoid hemorrhage—vertebral artery.

Vertebral artery dissecting aneurysm (VADA) has been considered as the cause of posterior circulation stroke, especially in young adults. In symptomatic cases of VADA, most patients suffer subarachnoid hemorrhage (SAH) or brain ischemia.[1] Among them, bilateral VADAs are significantly rare compared with unilateral VADA.[2] As a result, management strategies for those patients with bilateral VADAs who present with SAH are still controversial.

Here we describe three cases of bilateral VADAs with SAH and discuss the clinical features and the therapeutic strategy of treatment.

Case 1

A 45-year-old man whose 3-dimensional computed tomographic angiography (3D-CTA) scan 3 months before ictus showed no abnormality (Fig 1A) developed severe headache and consciousness disturbance and was referred to our hospital. A computed tomographic (CT) scan revealed massive SAH with thick hematoma in the left cerebellopontine angle (CPA) cistern (Fig 1B). 3D-CTA scan revealed fusiform dilatation of the bilateral vertebral arteries (VAs), suggesting dissecting aneurysm (DA; Fig 1C). The aneurysm on the left

From the *Departments of Neurosurgery at Nagano Red Cross Hospital, Nagano; †Sannotyou Hospital, Sanjo, Niigata; and ‡Brain Research Institute, Niigata University, Niigata, Japan.

Received June 9, 2010; revision received September 6, 2010; accepted October 18, 2010.

Address correspondence to Takeo Nashimoto, MD, PhD, Department of Neurosurgery, Nagano Red Cross Hospital, 5-22-1 Wakasato, Nagano, 380-8582, Japan. E-mail: asitake@par.odn.ne.jp.

1052-3057/$ - see front matter

doi:10.1016/j.jstrokecerebrovasdis.2010.10.003

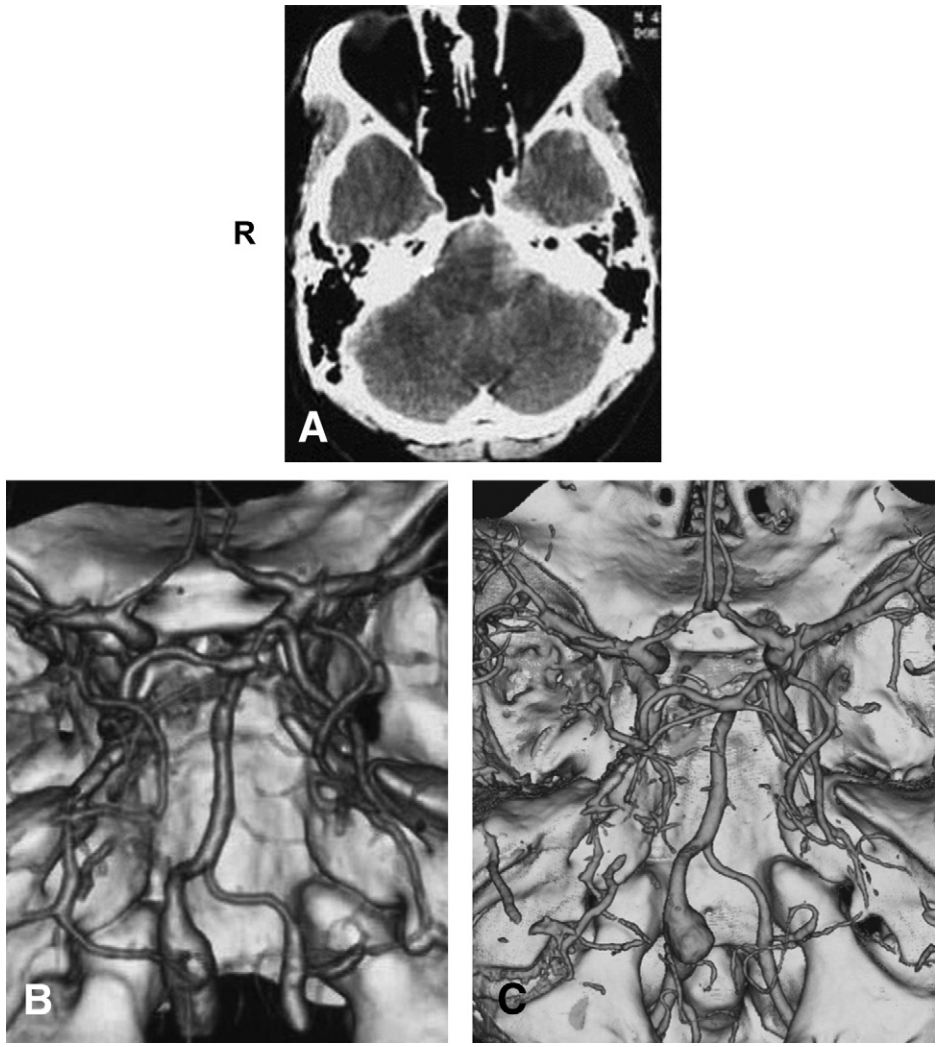

Figure 1. *(A) Head computed tomography (CT) on admission showing subarachnoid hemorrhage (SAH), mainly in the left cerebellopontine angle cistern. (B) Three-dimensional computed tomographic angiography (3D-CTA) on admission showing fusiform dilatation of the bilateral vertebral arteries (VAs) and the origin of the right posterior inferior cerebellar artery (PICA), being larger and accompanied with a bleb-like protrusion in the left VA. (C) 3D-CTA on day 22 showing improvement of the right VA dissecting aneurysm (VADA) except for the origin of the right PICA. The left VADA is slightly enlarged.*

side was larger than that on the right, and also had a bleb-like protrusion. The left posterior inferior cerebellar artery (PICA) arised from the extracranial VA. Therefore, the left one was considered to have caused the SAH. The right VADA also extended into the origin of the PICA.

During the examination, he became comatose because of recurrent SAH. We treated him conservatively using midazolam, nizofenone fumarate and pentazocine, and systolic blood pressure was strictly controlled below 140 mm Hg using nicardipine. After that episode, no bleeding was observed. A 3D-CTA scan on day 22 revealed that the left VADA was still large and growing aneurysmal dilatation, but decreased in size of the right VADA except for the origin of the PICA (Fig 1C). Because the left VADA was considered to have a high tendency to rebleed, he underwent trapping of this lesion on day 28 (Fig 2A). Thereafter, we finished sedation and strict blood pressure control. A postoperative 3D-CTA scan revealed the disappearance of the left VADA and normalization of the right VA and PICA (Fig 2B). He was transferred to a rehabilitation unit on day 213 with mild left limb ataxia, hypoesthesia, and central sleep apnea syndrome.

Case 2

A 46-year-old woman developed a sudden onset headache. Five days later, she became somnolent and was hospitalized. A CT scan revealed a massive SAH (Fig 3A and *B*). A 3D-CTA scan revealed pearl and string signs of the bilateral VAs and aneurysmal dilatation of the right VA (Fig 3C). Both posterior communicating arteries were well-developed, and retrograde blood flow supplied by these arteries would be sufficient. We treated conservatively using nizofenone fumarate and pentazocine, and systolic blood pressure was strictly controlled below 140 mm Hg using nicardipine. No rebleeding was observed during the course. On day 19, a 3D-CTA scan revealed spontaneous occlusion of the right VA but a slight increase in size of the left VADA (Fig 3D). However, the size of the left VADA had decreased until day 26 (Fig 3E), and we finished sedation and strict blood pressure control. The left VA finally occluded on day 85 (Fig 3F). Retrograde flow from the well-developed bilateral posterior communicating arteries provided continued basilar perfusion, so she was discharged with no neurologic deficits.