Enlargement of Cerebellar Arteriovenous Malformation Associated with Fenestration of the Vertebral Artery

—Case Report—

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Abstract

We report a 30-year-old male case of an enlarged cerebellar arteriovenous malformation (AVM) associated with fenestration of the vertebral artery. Hemodynamic stress resulting from fenestration of the feeding system of AVM was probably an important factor in the enlargement of the small cerebellar AVM.

Key words: arteriovenous malformation, fenestration, vertebral artery

Introduction

Cerebral arteriovenous malformation (AVM) may enlarge gradually, but of 47 enlarged AVM cases reported,^{1,3-7,9,13,15-18,20,23-26)} none were associated with fenestration of the vertebral artery. Cerebral angiography shows the incidence of fenestration of the vertebral artery is 0.3-2.0%.^{11,12)} Fenestration of the vertebral artery is frequently associated with intracranial abnormalities, but only four cases were associated with AVM.^{11,12,22)}

Here, we report a patient with cerebellar hemorrhage and a small cerebellar AVM associated with fenestration of the vertebral artery. Hemorrhage recurred 13 years later, associated with enlarged cerebellar AVM. The mechanism of enlargement of AVM associated with fenestration of the vertebral artery is discussed.

Case Report

A 30-year-old male was previously admitted to the Department of Neurosurgery, Hiroshima Prefectural Hospital following the onset of severe headache, vomiting, and disturbance of consciousness on

February 26, 1976, then aged 17 years. He was comatose with marked nuchal stiffness. Cerebral angiograms showed hydrocephalus and small AVM in the right cerebellar hemisphere, associated with fenestration of the right vertebral artery (Fig. 1). Right ventricular drainage obtained bloody fluid.

Fig. 1 Right vertebral angiogram on previous admission, showing small cerebellar AVMs (arrowheads) and fenestration of the right vertebral artery (arrow).

Fig. 2 Selective right (*upper*) and left (*lower*) vertebral angiograms on present admission, showing an enlarged cerebellar AVM and fenestration of the right vertebral artery.

Cerebellar hemorrhage due to bleeding from a small cerebellar AVM was diagnosed.

His consciousness gradually improved and he became alert after 7 days. A ventriculoperitoneal shunt was emplaced to correct persistent hydrocephalus. He was discharged with mild cerebellar symptoms on May 27, 1976, and attended several times 1 year as an outpatient. Follow-up pre- and postcontrast computed tomographic (CT) scans showed no abnormal findings on January 22, 1979.

He was admitted to the Department of Neurosurgery, Hiroshima General Hospital suffering from occipital headache on July 10, 1989. He was alert with nuchal stiffness. A CT scan indicated hemorrhage into the cerebellum and fourth ventricle. Selective bilateral vertebral angiograms showed an AVM in the right cerebellar hemisphere extending to the vermis, which was fed by the right superior cerebellar, right anterior inferior cerebellar, and right posterior inferior cerebellar arteries (Fig. 2). An enlarged draining vein ran on the vermis, leading into the straight sinus.

A median suboccipital craniectomy on August 10, 1989 partially exposed the nidus along the draining vein in the vermis. The nidus was dissected from the surrounding tissue. Each feeding artery was cut after coagulation to gain access to the floor of the fourth

Fig. 3 Postoperative selective right (*upper*) and left (*lower*) vertebral angiograms, showing complete absence of AVM.

ventricle. Finally, the draining vein was cut, and the AVM totally removed.

Right abducens nerve paresis, right facial nerve paresis of the central type, and right cerebellar hemisphere syndrome appeared postoperatively, but improved gradually. After 12 days, the abducens and facial nerve pareses were completely absent. Selective bilateral vertebral angiograms 15 days (Fig. 3) and magnetic resonance images 56 days postoperatively confirmed that the AVM was completely removed. Following rehabilitation, he was discharged with right mild residual cerebellar hemisphere syndrome on October 21, 1989. He has lead a useful life during the past 2-year follow-up period.

Discussion

The mechanism of AVM enlargement may include several possible factors. 1) The AVM nidus is composed of numerous hyalinized vessels which are easily enlarged by increased intraluminal pressure.³⁾ 2) Silent hemorrhage occurs repeatedly in AVM. During the resolution of hemorrhagic degeneration and necrosis, supporting tissue decreases and increased pressure in the artery entering the AVM causes dilatation and enlargement.¹⁴⁾ 3) The presence of an arteriovenous shunt causes hemodynamic change

leading to the enlargement of vessels inside and outside the AVM.⁶⁾ 4) AVM requires more space for enlargement.^{3,26)} 5) AVM enlargement often occurs in infants, so AVM growth during infant development is possible.^{6,18)}

However, in our case, the patient was not at the age of rising arterial blood pressure and hemorrhage had occurred only twice during a 13-year period. No arteriovenous shunt was found by the first arteriograms, and the AVM was small at the onset. The site of development was the posterior fossa, and therefore limited space was available for expansion. A CT scan when he was 20 years old and growth was complete detected no AVM, and thus it developed subsequently. Thus, the previously described factors do not apply to our case. Our case also differs from previously reported enlarged AVM in that fenestration of the vertebral artery was in the arterial system feeding the enlarged AVM, not in the feeding artery itself.

There are several reports of coexisting fenestration and cerebral aneurysms. Miyazaki *et al.*¹⁰⁾ studied multiple aneurysms of the vertebrobasilar system associated with fenestration of the vertebral artery, and found 11 of 57 cases associated with cerebral aneurysm. Arai *et al.*²⁾ also reported a case of this anomaly associated with posterior inferior cerebellar artery aneurysm. In both reports, a hemodynamic factor was probably involved in the development of the cerebral aneurysm,¹⁹⁾ and fenestration of the vertebral artery was important. Kwak *et al.*⁸⁾ and Tanaka and Matsumoto²¹⁾ thought that aneurysms develop due to hemodynamic stress caused by the fenestration of the parent vessel.

In our case, we cannot be sure that hemodynamic stress caused by the fenestration of the vertebral artery enhanced the AVM enlargement. However, no other factors that could cause AVM enlargement were detected except for the fenestration of the vertebral artery in the AVM feeding system. Therefore, the pathological (hyalinized) vessels in the small AVM probably succumbed to pressure within the vessels induced by the hemodynamic stress resulting from fenestration of the vertebral artery, with consequent enlargement to a typical AVM.

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