Endovascular Treatment of Vertebral Artery Aneurysm Manifesting as Progressive Hemifacial Spasm
—Case Report—

Keigo MATSUMOTO, Satoshi KIMURA*, and Kiyohito KAKITA*

Department of Neurosurgery, Shakaihoken Kobe Central Hospital, Kobe, Hyogo;
*Department of Neurosurgery, Kyoto First Red Cross Hospital, Kyoto

Abstract
A 62-year-old woman presented with right hemifacial spasm persisting for 6 months. Brain magnetic resonance imaging and digital subtraction angiography showed a wide-neck aneurysm of the intracranial portion of the right vertebral artery. The patient underwent endovascular trapping of the aneurysm by coil embolization of the parent vessel on both sides of the aneurysm. The patient experienced gradual disappearance of the hemifacial spasm within 3 months. No relapses occurred during a follow-up period of 3 years. Magnetic resonance imaging revealed shrinkage of the vertebral artery aneurysm which had compressed the facial nerve. Endovascular trapping of a vertebral artery aneurysm can be used to treat hemifacial spasm caused by an aneurysm instead of surgical microvascular decompression.

Key words: endovascular treatment, hemifacial spasm, vertebral artery aneurysm

Introduction
Hemifacial spasm (HFS) is characterized by involuntary intermittent twitching of portions of the muscles innervated by the facial nerve. Facial nerve irritation or compression by vascular structures, most frequently elongated arteries, is a widely accepted cause of HFS, but pathological lesions such as tumors, angiomas, and arteriovenous malformations may also be involved. Aneurysms in the vertebrobasilar system rarely cause HFS. Magnetic resonance (MR) imaging can demonstrate the microanatomical structures with high sensitivity and good correlation with surgical findings. Microvascular decompression of the facial nerve is performed in patients with severe symptoms and has been successful in a high percentage of cases. Here we report a case of vertebral artery aneurysm causing progressive HFS which was successfully treated with endovascular coil embolization.

Case Report
A 62-year-old female developed right hemifacial spasm which gradually worsened for 6 months. She consulted a neurosurgical clinic where MR imaging suggested a right vertebral artery aneurysm as a flow void in the right cerebellopontine angle (Fig. 1). Digital subtraction angiography showed a wide-neck aneurysm of the right vertebral artery proximal to the posterior inferior cerebellar artery (Fig. 2). The left vertebral artery was also patent and had the same caliber as the right vertebral artery. We planned to perform endovascular trapping of aneurysm to decompress the aneurysm dome, which had compressed the facial nerve resulting in HFS.

The endovascular procedure was performed under general anesthesia. A sheath introducer was inserted into the right femoral artery, then a guiding catheter (Envoy 6 Fr; Cordis, Miami, Fla., U.S.A.) was introduced into the right vertebral artery. The aneurysm was trapped by occlusion of the right vertebral artery on both sides of the aneurysm. Arterial occlusion was performed with Guglielmi detachable coils (Target Therapeutics, Fremont, Calif., U.S.A.) passed through a microcatheter (Transit; Cordis). Postembolization, the basilar artery and right
Fig. 1 Axial T₂-weighted magnetic resonance image showing an aneurysmal dilatation (arrow) of the right vertebral artery in the cerebellopontine angle.

Fig. 2 Right vertebral angiogram showing a wide-neck aneurysm in the right vertebral artery proximal to the posterior inferior cerebellar artery.

Fig. 3 Right vertebral angiogram after trapping of the right vertebral artery aneurysm showing no opacification of the aneurysm and the deposited coils (left). Left vertebral angiogram showing the basilar artery and the right posterior inferior cerebellar artery were fed by the left vertebral artery (right).

Fig. 4 Axial T₂-weighted magnetic resonance image at 3 months after embolization showing shrinkage of the right vertebral artery aneurysm (arrow).

Endovascular treatment for vascular compression syndrome of the cranial nerves has been performed in a limited number of cases, such as parent vessel occlusion for a dissecting vertebral artery aneurysm in a patient presenting with torticollis, and transvenous embolization for a cerebral arteriovenous malformation in a patient presenting with hemifacial spasm. Parent vessel occlusion by surgery or endovascular techniques is sometimes used for the treatment of fusiform or dissecting aneurysms because the parent vessel is difficult to reconstruct.

Discussion

Endovascular treatment for vascular compression syndrome of the cranial nerves has been performed in a limited number of cases, such as parent vessel occlusion for a dissecting vertebral artery aneurysm in a patient presenting with torticollis, and transvenous embolization for a cerebral arteriovenous malformation in a patient presenting with hemifacial spasm. Parent vessel occlusion by surgery or endovascular techniques is sometimes used for the treatment of fusiform or dissecting aneurysms because the parent vessel is difficult to reconstruct.
using clip application in such aneurysms. Recently, endovascular trapping was introduced as a treatment for serpentine aneurysm, which can completely occlude the channel to an aneurysm in some cases. Thrombus formation of a large or giant aneurysm following parent vessel occlusion results in the remission of cranial nerve disturbances such as cavernous sinus syndrome.

The complete remission of HFS in our patient was probably related to thrombosis of the aneurysm sac and relief of facial nerve compression, although this conclusion is based on circumstantial evidence. This hypothesis is supported by a recent report that selective catheterization of the posterior inferior cerebellar artery resulted in transient improvement of HFS, probably by weakening the arterial pulsation and straightening the arterial loop that separates the artery and nerve. Although the long-term results of vertebral artery coil embolization remain to be evaluated, endovascular trapping of a vertebral artery aneurysm can be used to treat HFS caused by an aneurysm instead of surgical microvascular decompression.

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References


Address reprint requests to: K. Matsumoto, M.D., Department of Neurosurgery, Shakaiboken Kobe Central Hospital, 2–1–1 Souyama-cho, Kita-ku, Kobe 651–1145, Japan. e-mail: keigom@msc.biglobe.ne.jp