LETTER TO THE EDITOR

Hemifacial Spasm Developing After the Adhesion of the Facial Nerve and Vertebral Artery

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Hemifacial spasm is a movement disorder characterized by involuntary contractions of the facial muscles. Although the mechanism of hemifacial spasm (HFS) is not yet well established, vascular compression of the facial nerve root exit zone (REZ) is one of the most likely mechanisms given the effectiveness of microvascular decompression (MVD) in the treatment of the disorder. We present a case of HFS in which the etiology was the facial nerve adherence of the VA. To our knowledge, this is the first report to this situation. Except for the compression at the facial REZ, the vascular compression mechanism supplied another possible etiology: adhesion of the facial nerve to the vertebral artery (VA).

A 55-year-old woman presenting with a 12-month history of right hemifacial spasm was admitted to our hospital in October 2011. Preoperative computed tomography angiography and magnetic resonance imaging showed that the left VA deflected to the right and flexed abnormally and was closely associated with the facial and statoacoustic auditory nerves. The patient underwent MVD surgery with electrophysiological monitoring in October 2011.

During the MVD operation, after removing the arachnoid, the left VA was gross and flexed abnormally; it was compressed against the right facial and statoacoustic auditory nerves. No vascular compression was found at the REZ of the facial nerve, but the VA was closely associated with the distal cisternal portion of the facial and auditory nerves. One section of the facial nerve was severely adhered to the VA (Figure 1). The abnormal muscle response observed on the electrophysiological monitor immediately disappeared after the facial nerve was separated from the VA [1]. The VA was moved aside, and a Teflon pad was used to physically separate the VA and facial nerve in the cisternal portion. The spasm completely disappeared immediately postoper-

Figure 1 An intraoperative photograph of the relationship of the facial nerve (FN), vertebral artery (VA), and the cochlear nerve (CN) (Video S1).

ative, without facial palsy or hearing disturbance, and did not recur during 15 months of follow-up.

Although the mechanism of HFS is not clearly defined, vascular compression of the facial nerve REZ is generally considered the primary etiology of HFS [2]. The offending vessels are most often found on preoperative radiologic evaluations or during surgery. MVD of these offending vessels has yielded successful outcomes in large serial studies [3,4]. The area of the REZ is the root of the facial nerve exiting from the brain stem. This 2–3-mm area, considered synonymous with the Obersteiner–Redlich zone, is a transition zone between central and peripheral axonal myelination that is situated at the site of nerve detachment from the pons. Further proximally, however, many authors have observed vascular compression of the distal cisternal portion of the facial nerve as one of the etiologies of HFS [5]. MVD of this portion has been shown to be effective, and the culprits were clear.

In our case, we had to consider the adhesion of the facial nerve and VA as the cause of this HFS. The spasm completely disappeared immediately, and the abnormal muscle response on the

electrophysiological monitor immediately disappeared after we separated the facial nerve from the offending VA. Therefore, we concluded that hemodynamic stress from the VA applied pressure to the facial nerve and triggered the onset of the HFS in this patient as a result of the adhesion.

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Supporting Information

The following supplementary material is available for this article:

Video S1. A video of posterior cranial fossa surgical exploration for microvascular decompression for a 55-year-old woman with

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right hemifacial spasm. The neurovascular conflict was the facial nerve severely adhesion to the VA and the facial nerve was separated from the VA gently.