

A Case of Hemifacial Spasm Caused by Venous Compression

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Abstract

Hemifacial spasm (HFS) is often caused by arterial compression of the root exit zone of the facial nerve. HFS caused by venous compression is rare. In this report, we describe a case of microvascular decompression (MVD) for hemifacial spasm caused by venous compression, with excellent results. The patient was a 68-year-old man with right HFS for 3 years (2019~2022). Preoperative magnetic resonance imaging (MRI) demonstrated that the facial nerve was compressed by a vein rather than an artery. We performed MVD of the facial nerve. Interposition of the vein led to sufficient nerve decompression. The HFS disappeared immediately after surgery. Preoperative diagnosis of venous compression is possible with MRI. During surgery, it is necessary to be careful not to cause venous circulation disorders because veins, unlike arteries, are sometimes difficult to move.

Keywords: Vein; Hemifacial Spasm; Microvascular Decompression; Magnetic Resonance Imaging

Introduction

Hemifacial spasm (HFS) is a paroxysmal involuntary movement of the face on one side, often beginning around the eyelid in the early stages of a disease and gradually spreading to the corners of the mouth [1,2]. It is often caused by arterial compression at the root exit zone (REZ) of the facial nerve [1,2]. HFS caused by venous compression is extremely rare [3-6]. In this study, we report a case of HFS in which the vein was the only responsible vessel, along with the surgical findings and diagnostic imaging associated with the case.

Case Presentation

A 62-year-old man visited the outpatient unit of our department for the examination and treatment of right HFS. HFS began 3 years ago (2019~2022) in the right eyelid area and gradually spread to the corners of the mouth. Tinnitus was not observed. Bilateral otoscopic examination was unremarkable. No associated sensory loss, facial palsy, or other neurological signs were observed on clinical examination.

His medical history included well-controlled diabetes mellitus.

A neuroradiological diagnosis was made using magnetic resonance imaging (MRI). There were no brain tumor lesions in the cerebellar-pontine angle. Spoiled gradient recalled echo (SPGR) sequencing (Figure 1A) failed to identify any obvious vessels at the REZ of the facial nerve, indicating that no arteries were involved, but constructive interference in the steady state (CISS) sequencing (Figure 1B) identified a compression vessel at the same site, which was identified as a vein. With a diagnosis of HFS due to venous compression, informed consent was obtained, and microvascular decompression (MVD) was performed.

During surgery, the patient was placed in the left lateral decubitus position and a right-sided suboccipital retrosigmoid craniotomy was performed with continuous intraoperative monitoring of the auditory brainstem response. An 8 cm linear skin incision was made in the retroauricular region, and a craniotomy procedure was

Figure 1

performed until the sigmoid sinus was partially exposed. The dura mater was opened, cerebrospinal fluid was aspirated, the cerebellum was gently retracted, and the arachnoid membrane around the lower cranial nerves was dissected. After sufficient dissection, the REZ of the facial nerve was observed. There was no obvious artery in the REZ of the facial nerve, but severe compression by the transverse pontine vein from the lateral side was noted (Figure 2A). Dissection was performed between the facial nerve and the vein. In addition, dissection between the brainstem and vein was performed, but the extent of dissection was limited to what could be safely performed due to severe adhesions. Next, a Teflon felt was inserted and fixed between the facial nerve and vein responsible for the facial nerve compression (Figure 2B).

Figure 2

Following surgery, the patient had an uneventful postoperative recovery, and the HFS resolved immediately with no additional neurological deficits. At the 6-month follow up, no further HFS occurred.

Discussion

HFS is typically caused by arterial compression of the REZ of the facial nerve. It can also be caused by compression by a brain tumor [7] or vein [3-6] but this is quite rare (veins are responsible for 0.1-2.9% [3-6] of all cases). On the other hand, trigeminal neuralgia (TN), which is also a neurovascular compression syndrome,

is thought to have a different pathophysiology, with 12.5-26.8% of TN [8,9] of the compression vessels involving veins. In 138 cases of HFS operated on in the past 5 years in our department, the vein was the responsible vessel in the present case (0.7%).

Fifteen cases of HFS involving veins have previously been reported, including 13 cases of recurrence [10]. All the patients were female, and 60% had clinical symptoms of platyzma. Our patient was a male and had no symptoms of platyzma. In eight cases, the vein was cut as part of a surgical procedure. Although symptoms improved in all patients, hearing loss, facial paralysis, lower cranial nerve symptoms, and cerebellar symptoms appeared in 80% of the patients as a result of surgical complications, suggesting the need for caution when managing these cases surgically [10].

MVD for HFS involves moving the responsible vessel to within a safe range and obtaining sufficient decompression of the facial nerve. There are two main methods: transposition, in which the responsible vessel is moved significantly, and interposition, in which a prosthesis is inserted between the facial nerve and responsible vessel. While transposition is recommended to prevent recurrence; there is no reported difference in the occurrence of complications between the two techniques [11]. These techniques are applied when the responsible vessel is an artery. During surgery, unlike when the responsible vessel is an artery, migration of a vein can be difficult because of its adhesions to the brain stem and nerves. Dissection of veins with their thinner wall entails significant risks, including impaired venous return to the brainstem, facial paralysis, and hearing loss [6]. Before incising a vein, its circulation territory should be determined. There have been reports of impaired venous return to the brainstem due to venous transection; therefore, care must be taken during these procedures [6]. HFS is a functional disease, and efforts should be made to prevent its complications as much as possible. In our case, the responsible vein was a transverse pontine vein with a large diameter and was determined to be involved in the venous return to the brainstem; therefore, it was dissected as much as possible, and a prosthesis was inserted between the nerve and responsible vein for decompression. Fortunately, the patient's HFS disappeared without complications, and good results were obtained. However, there have been reports of recurrent cases, and we believe that a close ongoing follow-up is necessary for all patients.

When veins are the responsible vessels, unlike arteries, they are usually difficult to visualize on MRI. The use of contrast media has been proposed to better identify responsible veins [12]. In our case, contrast was not utilized because of mild renal dysfunction. Although an artery could be evaluated on SPGR sequence images, no artery could be seen around the right facial nerve in our case, and the responsible vessel was determined to be a vein because a vessel could be seen on the CISS sequence. However, evaluation of small veins may be difficult, and this is a potential research avenue for the future.

Conclusion

It should be kept in mind that facial spasm due to venous compression can occur. MRI requires further advances to provide detailed preoperative information when the responsible vessel is the vein. Veins have thinner walls and are more easily damaged than arteries. Therefore, care must be taken when moving them.

Conflicts of Interest

The authors declare that they have no conflicts of interest.

Ethical Approval

The Ethical Committee of the International University of Health and Welfare approved all the procedures used in this research.

Submission Statement

This original manuscript has not been submitted elsewhere in part or whole.

Bibliography

- Hyun SJ, *et al.* "Microvascular decompression for treating hemifacial spasm: lessons learned from a prospective study of 1,174 operations". *Neurosurgical Review* 33.3 (2010): 325-334.
- Jannetta PJ. "Observations on the etiology of trigeminal neuralgia, hemifacial spasm, acoustic nerve dysfunction and glossopharyngeal neuralgia. Definitive microsurgical treatment and results in 117 patients". *Neurochirurgia (Stuttg)* 20.5 (1977): 145-154.
- Mercier P and Sindou M. "The conflicting vessels in hemifacial spasm: Literature review and anatomical-surgical implications". *Neurochirurgie* 64.2 (2018): 94-100.
- El Refaee E, *et al.* "Arachnoid bands and venous compression as rare causes of hemifacial spasm: analysis of etiology in 353 patients". *Acta Neurochirurgica (Wien)* 162.1 (2020): 211-219.
- Eun J, *et al.* "Hemifacial Spasm Caused by a Vein: A Case Report". *Asian Journal of Neurosurgery* 13.3 (2018): 786-788.
- Dumot C and Sindou M. "Veins of the Cerebellopontine Angle and Specific Complications of Sacrifice, with Special Emphasis on Microvascular Decompression Surgery. A Review". *World Neurosurgery* 117 (2018): 422-432.
- Liu J, *et al.* "Hemifacial Spasm as Rare Clinical Presentation of Vestibular Schwannomas". *World Neurosurgery* 116 (2018): e889-e894.
- Kondo A. "Follow-up results of microvascular decompression in trigeminal neuralgia and hemifacial spasm". *Neurosurgery* 40.1 (1997): 46-51.
- Sabourin V, *et al.* "Internal Neurolysis with and without Microvascular Decompression for Trigeminal Neuralgia: Case Series". *World Neurosurgery* 143 (2020): e70-e77.
- Wang X, *et al.* "The role of vein in microvascular decompression for hemifacial spasm: a clinical analysis of 15 cases". *Neurological Research* 35.4 (2013): 389-394.
- Chai S, *et al.* "Microvascular decompression for trigeminal neuralgia caused by vertebrobasilar dolichoectasia: interposition technique versus transposition technique". *Acta Neurochirurgica (Wien)* 162.11 (2020): 2811-2821.
- Inoue T, *et al.* "Diagnosis and management for trigeminal neuralgia caused solely by venous compression". *Acta Neurochirurgica (Wien)* 159.4 (2017): 681-688.