INTRODUCTION

Hemifacial spasm (HFS) is an involuntary contraction of facial muscles caused by facial nerve compression from vascular structures. Majority of compressions (>90%) occur at the root exit zone (REZ), where central glial myelin transits to peripheral Schwann cell myelin [1]. The only treatment for HFS is microvascular decompression (MVD), and thus it is crucial to accurately specify the offending vessels and properly decompress it [2]. Preoperative magnetic resonance imaging (MRI) findings can help identify these vessels, but in complex cases, there are mismatch between MRI finding and real intraoperative offender frequently [3]. Sometimes, there is even no definite culprit vessel near the REZ. Veins contribute to neurovascular compression (NVC) in conjunction with other offending arteries to cause spasm, but sole venous offender is rare [4]. Venous offenders are difficult to identify on preoperative MRI, so it takes many efforts to confirm a real offending vessel during surgery in that case. Those efforts may damage facial nerve or vestibulocochlear nerve by vigorous cerebellar retraction. We report two cases Sole vein offenders confirmed by disappearance of the lateral spread response during microvascular decompression for hemifacial spasm

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Sole vein offenders are rare and can be difficult to identify on preoperative magnetic resonance imaging (MRI) and intraoperative microscopy. The objective of this case review was to investigate factors resulting in successful decompression in venous offender cases. Among 234 cases of microvascular decompression surgery performed for hemifacial spasm from September 2020 to April 2022 at Konkuk University Medical Center, only two cases were identified as having a definite venous offender. We reviewed preoperative MRI and intraoperative microscopic findings and studied the correlation between decompression and lateral spread response (LSR) changes. Our two patients had possibility of a small venous offender on preoperative MRI, but it was ambiguous. A definite venous offender was identified on the operative microscopic findings and decompressed successfully, referring to the disappearance of the LSR waveform. Postoperatively, spasm disappeared or significantly improved. A venous offender is difficult to identify on preoperative MRI findings or even on intraoperative microscopic findings. In such cases, changes in the LSR waveform in response to decompressing the suspected vein can be a valuable reference for successful facial outcomes.

KEY WORDS: Hemifacial spasm, Microvascular decompression, Intraoperative neurophysiological monitoring
of sole venous offenders wherein the offending vessels were not clearly seen on preoperative MRI, but were confirmed by disappearing lateral spread response (LSR) after decompression of the suspected vein.

Ethical statements

This study was exempted from the Institutional Review Board of the Konkuk University Medical Center (No. KUMC 2022-09-038). Written informed consent was obtained from the patient.

CASE REPORT

Among 234 cases of MVD surgery performed for HFS from September 2020 to April 2022, only two cases were identified as having a definite venous offender. Besides, arterial and venous offenders co-existed in four cases, and a possible venous offender without other specific offending vessels was seen in one case (Table 1). In order to examine LSR changes and surgical results after vein decompression, this review only included cases with definite sole venous offender. All patients were monitored for changes in LSR during surgery, as an indicator for confirming whether it was properly decompressed. Brainstem auditory evoked potential (BAEP) waveform was also monitored to prevent hearing loss.

Surgical procedure

The entire operation was performed by a single surgeon (KP). After retromastoid suboccipital craniectomy, the dura was opened and cerebrospinal fluid was drained to relax the cerebellum. The ninth cranial nerve and the choroid plexus were identified, which are right below the facial nerve REZ. After identifying the facial nerve, the surgeon examined the whole segment from REZ to the internal acoustic canal and 360 degrees surrounding the facial nerve to find the offender. After specifying the offending vessels, Teflon felts were inserted to decompress it. If the LSR disappeared after the decompression, the vessel was thought to be the culprit and successful decompression was made. In cases with a remnant LSR after decompression of the suspected vessel, we can look for another possible offender. However, considering the possibility of delayed LSR disappearance even with successful decompression [5], we did not vigorously retract structures to search for a missing culprit.

Case illustration

Case 1

A 71-year-old female with a 3-year history of left HFS underwent MVD surgery. Preoperative MRI revealed close approximation at the cisternal segment of the left facial nerve by the left anterior inferior cerebellar artery (AICA) and at the REZ by a small branch of petrosal vein (Fig. 1). Intraoperative inspection identified a small venous offender, which was decompressed by Teflon felt (Fig. 2). During the operation, facial electromyography (EMG) and BAEP were recorded. The LSR wave disappeared immediately after decompression of the suspected vein (Fig. 3). Postoperatively, no immediate spasm symptom was noted, but it relapsed intermittently from 2 days after surgery. At 3 months after surgery, she had a residual spasm of which an intensity was subjectively about 20% of preoperative facial spasm. However, at the last follow-up date, 9 months postoperatively, the spasm was improved with relapse fewer times per day.

Case 2

A 42-year-old female underwent MVD surgery for a 5-year history of right HFS. Preoperative MRI revealed possible NVC at the cisternal segment by the right AICA or at the REZ by the small vein (Fig. 4). Intraoperative findings confirmed a definite venous offender, which was decompressed by Teflon felt (Fig. 5). During the operation, facial EMG and BAEP waveforms were recorded. LSR disappeared right after the decompression (Fig. 6). Postoperatively, no immediate facial spasm was noted. She remained symptom-free with no residual spasm at 6 months postoperatively, the last follow-up date.

DISCUSSION

The AICA and posterior inferior cerebellar artery (PICA) are known to be the most common offending vessels in HFS. A review by Mercier and Sindou [6] found that the most common of-

| Table 1. Distribution of offenders confirmed by intraoperative field finding |
|-----------------------------|-------|------|
| Offender | Number | Ratio (%) |
| Sole offenders |
| Anterior inferior cerebellar artery (AICA) | 118 | 50.4 |
| Posterior inferior cerebellar artery (PICA) | 54 | 23.1 |
| Vertebral artery (VA) | 43 | 18.4 |
| Vein | 2 | 0.9 |
| Multiple offenders |
| AICA-PICA | 8 | 3.4 |
| AICA-VA | 4 | 1.7 |
| Vein with other arteries | 4 | 1.7 |
| Unidentified: possible venous offender | 1 | 0.4 |
| Total | 234 | 100 |

The statistics were compiled from microvascular decompression surgery performed from September 2020 to April 2022.
Fig. 1. Possible neurovascular compression (NVC) at the mid-cisternal segment of the left facial nerve by the left anterior inferior cerebellar artery (AICA), as well as at the root exit zone by a tiny petrosal vein. (A) NVC by the AICA (white arrow). (B) NVC by a tiny vein (black arrow).

Fig. 2. Gross intraoperative field findings. We confirmed the compression and indentation by a small vein at the root exit zone and decompressed it using Teflon felt. (A) Neurovascular compression by a tiny vein (white arrow). (B) After decompression by Teflon felt.

Fig. 3. Intraoperative lateral spread response (LSR) and brainstem auditory evoked potential (BAEP) monitoring. Immediately after neurovascular decompression, the LSR disappeared and was maintained until the end of surgery. The BAEP waveform was well-maintained during the entire procedure. (A) Before decompression. (B) After decompression.
Fig. 4. Possible neurovascular compression (NVC) at the mid-cisternal segment of the right facial nerve by the right anterior inferior cerebellar artery (AICA), or at the root exit zone of the facial nerve by a vein. (A) NVC by a vein (black arrow). (B) NVC by the AICA (white arrow).

Fig. 5. Gross intraoperative field findings. We confirmed the compression and indentation by a small vein at the root exit zone and decompressed it using Teflon felt. (A) Neurovascular compression by a tiny vein. (B) After decompression by Teflon felt (white arrow).

Fig. 6. Intraoperative lateral spread response (LSR) and brainstem auditory evoked potential (BAEP) monitoring. Immediately after neurovascular decompression, the LSR disappeared and was maintained until the endpoint of surgery. The BAEP waveform was well-maintained during the entire procedure. (A) Before decompression. (B) After decompression.
fending vessels were PICA (47.2%), AICA (45.9%), vertebral artery (VA; 17.5%), and veins (4.9%). Their review included cases with multiple offending vessels. Notably, venous offenders associated with other arteries are seen in 0.7% to 7.9% (4.9% on average) of cases, while cases with solely venous offender are seen in 0.4% to 5.5% (1.4% on average) [6-14]. In addition, El Refaee et al. [15] reported a sole venous offender in just one patient among 353 cases (0.28%), while Dumot and Sindou [16] similarly reported an incidence of 0.1%. Among MVD surgeries for HFS performed at our clinic, the most offending vessels, in descending order, were AICA, PICA, and VA, followed by definite sole venous offenders, seen only in two cases (0.9%) (Table 1).

Whether LSR monitoring is a confirmative indicator of successful MVD surgery remains controversial. Some authors assert that it is a major reference to predicting facial outcome, while others argue that it is still questionable whether LSR monitoring is correlated with long-term facial outcome [17]. Some papers argue the usefulness of LSR monitoring say that even if the LSR waveform doesn’t completely disappear but the amplitude is just reduced, it is associated with favorable facial outcome [18,19]. Some papers argue that persistent LSR during surgery is not directly related to poor prognosis because it may take months to years for the motor nucleus hyperexcitability to normalize [5]. The disappearance of LSR immediately after decompression doesn’t always mean a successful outcome because LSR could relapse.

Nevertheless, considering these LSR changes and favorable facial outcome, it can usually be a good reference indicator for successful decompression. Even if the LSR did not completely disappeared, clinical symptom improved when sufficient decompression was achieved based on MRI and intraoperative microscopic field findings. In some our patients whose LSR did not completely disappeared during surgery, facial spasm successfully disappeared or at least lessened in intensity or frequency. In these cases, LSR monitoring could not function as a confirmative indicator of successful decompression. However, even in these cases, changes such as amplitude reduction of LSR were observed at the moment of decompression, and this could be helpful to ongoing decompression procedure. These results of LSR monitoring and patient’s facial outcome in our clinic are in line with many other reports, asserting the LSR as the major confirmative reference [4,17-19].

Venous offenders are rare; theoretically, these might not affect the facial nerve because of their lower pressure than arteries. Furthermore, vascular conditions affected by atherosclerosis or hypertension could contribute to NVC [20], but veins are less affected by these factors. Arachnoid membrane tightness or arachnoid bands, which strangulate the facial nerve at the REZ can also be a causative factor [15], but these factor affect both arteries and veins near the facial nerve. Therefore, the existence of venous offenders has had numerous controversies. If there were no arterial offenders in the surgical field and only a vein near the REZ, we cannot be confident of specifying the vein would be a real culprit. However, the immediate disappearance of LSR after vein decompression could be a useful indicator for ascertaining the real culprit for appropriate decompression. Besides, the changes of LSR resulted from vein decompression would suggest the existence of a venous offender.

If LSR monitoring is performed while paying attention to the aforementioned considerations, changes in LSR can be a good reference for successful decompression. Nevertheless, it is more important to pay attention to MRI findings and intraoperative microscopic findings between facial nerve and vascular structures. In our both cases, although a definite venous offender was not identified on preoperative MRI findings, there remained the possibility of a small venous offender. If LSR persists after decompression of suspected arterial offenders confirmed on preoperative MRI, we should keep in mind the possibility of small vessel offenders, which were not seen/ambiguous on MRI. Importantly, during the small vessel decompression, LSR changes should be monitored carefully. Furthermore, if it a possible venous offender that cannot be definitely confirmed by preoperative MRI or intraoperative microscopic finding, LSR waveform changes after suspected vein decompression can serve as a major reference for identifying a real culprit.

CONCLUSION

Venous offender is difficult to specify on preoperative MRI findings or even on intraoperative microscopic findings. In addition, when the MRI or intraoperative finding doesn’t show compression or indentation by arterial offender, the possibility of a venous offender should always be considered. In such cases, changes in LSR waveform by decompressing the suspected vein can be a major reference for successful facial outcomes.

CONFLICTS OF INTEREST

No potential conflict of interest relevant to this article was reported.

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