Hemifacial Spasm Caused by Veins Confirmed by Intraoperative Monitoring of Abnormal Muscle Response

Wenlei Yang¹, Yasuhiro Kuroi², Suguru Yokosako², Hidenori Ohbuchi², Shigeru Tani², Hidetoshi Kasuya²

BACKGROUND: Hemifacial spasm (HFS) is a benign disease caused by the hyperexcitement of facial nerves owing to vessel compression. The offending vessels are usually arteries, such as anterior and posterior inferior cerebellar or vertebral arteries, but there are few reports of vein involvement cases.

OBJECTIVE: The aim of this study was to investigate veins as offending vessels in patients with HFS confirmed by abnormal muscle response (AMR).

METHODS: We analyzed 5 patients with HFS caused by veins among 78 patients with HFS over the past 10 years. All patients underwent microvascular decompression (MVD) with AMR monitoring, whereas 3 of them underwent a second MVD. The mean follow-up time was 97 months.

RESULTS: Arteries were thoroughly decompressed in 3 patients with a failed first MVD surgery who received a second surgery, during which veins at the root exit point (RExP) were decompressed with the disappearance or a significant decrease in the amplitude of AMR. Two patients showed spasm resolution after the first surgery when veins were decompressed together with the disappearance of AMR. The location of veins was RExP and the cisternal portion. All patients had excellent outcomes within 3 months, and no complications were observed.

CONCLUSIONS: Veins can be offending vessels in HFS patients. AMR is useful to determine the endpoint in these cases. Once arteries are decompressed thoroughly with residual AMR, surrounding veins at unusual sites, such as the RExP or the cisternal portion, must be checked to prevent persistent HFS. Complete decompression of veins leads to a good clinical outcome.

INTRODUCTION

Hemifacial spasm (HFS) is a hyperactive symptom mainly caused by vessel compression at the root exit zone of facial nerves. The offending vessels are usually arteries; such as anterior (AICA) or posterior (PICA) inferior cerebellar artery, or vertebral artery, but veins are rarely reported, and estimated at fewer than 5%. Microvascular decompression (MVD) is widely accepted as a first choice for HFS, which leads to spasm resolution in over 90% of patients. An abnormal muscle response (AMR) is a typical abnormal muscle reaction in patients with HFS, which is particularly valuable when AMR disappears just after the decompression of facial nerves that is important to help surgeons confirm whether adequate decompression has been achieved. We performed this study applying AMR monitoring during MVD to confirm vein compression in HFS patients.

MATERIAL AND METHODS

Patient Data
We reviewed 78 patients who underwent MVD for typical HFS in the Department of Neurosurgery, Tokyo Women’s Medical University Medical Center East from 2007—2018. Among them, 5

Key words
- Abnormal muscle response
- Hemifacial spasm
- Vein

Abbreviations and Acronyms

AICA: Anterior inferior cerebellar artery
AMR: Abnormal muscle response
AS: Attached segment
CP: Cisternal portion
HFS: Hemifacial spasm
MVD: Microvascular decompression
PICA: Posterior inferior cerebellar artery
RDP: Root detachment point
RExP: Root exit point
patients were confirmed to have veins as offending vessels according to intraoperative findings and AMR monitoring. Patients consisted of 4 women and 1 man, whose mean age was 55.6 years old (ranging from 43–72 years old). Three patients had a failed first MVD surgery and received a second surgery; the mean interval between the 2 operations was 16.3 months (4–36 months). The mean follow-up period was 97 months (74–140 months). The patients’ clinical characteristics are presented in Table 1. The study was approved by the institutional review board of Tokyo Women’s Medical University (IRB #4879).

Surgical Procedure
One dose of short-acting muscle blockage agent was administered before intubation; total intravenous anesthesia is maintained during the whole operation. The patients were operated on in the lateral decubitus position, using a 3-skullpin head holder to fix the head. Craniotomy was performed using a routine retro-sigmoid approach. After the edge between the transverse and sigmoid sinuses was explored, we opened the dura and arachnoid tissue, gradually exposed the lower cranial nerves, looked upward through the infra-flocculus, and exposed the root exit point (RExP), attached segment (AS), root detachment point (RDP) transition zone, or cisternal portion (CP) of the facial nerve. When the offending vessels were identified, we used a transposition method for decompression for arteries; for veins, we performed dissection and coagulation, or inserted a Teflon prosthesis to achieve decompression.

Intraoperative Monitoring
The auditory brain stem response was monitored in all cases during MVD to preserve hearing. The AMR was used to identify the recognition of offending vessels and to evaluate the effectiveness of decompression. We stimulated the zygomatic branches and recorded the AMR wave from the mentalis muscle.

Follow-Ups
We checked the symptoms on the first postoperative day, at discharge, and in the outpatient clinic later with a face-to-face inquiry.

RESULTS
All 5 patients diagnosed with HFS completed preoperative exams in the outpatient clinic and received scheduled MVD by 1 doctor. None had received Botox treatment previously, and none had been clearly diagnosed with vein compression preoperatively (Table 1). Three patients had a failed first MVD and received a second surgery. Among their first procedures, PICA was revealed as the offending vessel in 2 patients, and AICA in 1 patient. AMRs persisted in 2 patients, although they disappeared during the procedure in 1 patient. In their second surgeries, the original decompressed arteries remained transposed from the facial nerve, but veins were found to have adhered or formed girdles around the nerve root. Finally, we coagulated veins or inserted a Teflon prosthesis to complete the decompression. All veins were located at RExP. AMRs disappeared in 2 of the patients, although the peak wave decreased to one third in 1 patient (Figure 1). In the fourth case, PICA was found to compress AS. The AMR did not change when PICA was transposed, which disappeared immediately when the vein under the facial nerve at CP was freed using a Teflon prosthesis (Figure 2). In the fifth case, we first alleviated AICA compression but AMR showed no change. We finally found a small branch of the transversospontine vein crossing at RExP, which was carefully coagulated, and AMR fully resolved.

Outcomes
HFS of the 5 patients disappeared within 3 months. Up to the present, HFS has not recurred in these patients. None of the patients experienced any complications, such as hearing loss, facial palsy, or cerebrospinal fluid leakage. The intraoperative findings and outcomes are detailed in Table 1.

Table 1. Summary of 5 Patients with Facial Spasm Caused by Veins

| Patient Number | Sex | Age (years) | Side | Interval Between MVD | Offending Vessels at First MVD | AMR Findings | Outcome | Offending Vessels at Second MVD | AMR Findings | Outcome |
|----------------|-----|-------------|------|----------------------|-------------------------------|--------------|---------|---------------------------------|--------------|---------|
| 1              | F   | 57          | Rt   | 15 Month             | PICA                          | No change     | Persist | Vein (RExP), Teflon prosthesis | Disappeared after decompression | Immediate relief |
| 2              | F   | 69          | Rt   | 35 Month             | AICA                          | No change     | Persist | Vein (RExP), coagulate         | Decreased to one third | Relief within 3 months |
| 3              | F   | 47          | Lt   | 4 Month              | PICA                          | Disappeared during procedure | Persist | Vein (RExP), coagulate         | Disappeared after decompression | Relief within 3 months |
| 4              | F   | 60          | Rt   | N/A                  | PICA/vein (CP), Teflon prosthesis | Disappeared after decompression of vein | Immediate relief | N/A | N/A | N/A |
| 5              | M   | 48          | Lt   | N/A                  | AICA/vein (RExP), coagulate | Disappeared after decompression of vein | Relief within 3 months | N/A | N/A | N/A |

MVD, microvascular decompression; AMR, abnormal muscle response; F, female; Rt, right; PICA, posterior inferior cerebellar artery; RExP, root exit point; AICA, anterior inferior cerebellar artery; Lt, Left; N/A, not available; CP, cisternal portion; M, male.
DISCUSSION

HFS is caused by neurovascular conflicts involving facial nerves, especially the root exit zone. MVD, which was standardized by Gardner7 and Jannetta et al.,8 is widely accepted as the first

Figure 1. Patient with repeated microvascular decompression (No. 2 in Table 1). Operative view at the first surgery of a 69-year-old woman (A) shows an anterior inferior cerebellar artery (white arrow) compressing the attached segment of the facial nerve, which was transposed and fixed to the posterior surface of the petrous bone using fibrin glue. In the second surgery (B), the arteries were left transposed and there were no arterial compressions. A small vein (black arrowhead) crossed at the root exit point of the facial nerve (black arrows), which was coagulated. The abnormal muscle response decreased to one third of the initial amplitude (C), serial change from the bottom to second top.

Figure 2. Patient with both posterior inferior cerebellar artery (PICA) and vein compression (No. 4 in Table 1). Operative view of a 60-year-old woman (A) shows typical PICA (black arrow) compressing the attached segment of the facial nerve. The abnormal muscle response (AMR) did not disappear after decompression. A branch of the vein of the cerebellopontine fissure (black arrow) crossed under the cisternal portion of the facial nerve (white arrows). A Teflon prosthesis was inserted between them (B). AMR disappeared after these procedures (C), serial change from the bottom to second top.
Some reasons for failure included misidentification of the actual offending vessels, displacement of an inserted prosthesis, or recompression owing to vessel displacement. Some authors reviewed the second MVD, and offending arteries were found rostral to the VII nerve, between VII and VIII nerves, or in the cisternal segment of the VII root. The inability to identify these arteries or small arterioles led to the inefficiency of MVD, but vein compression was not mentioned as a cause of reoperation. Others found veins as offending vessels in significantly more patients who underwent a second MVD compared to the first MVD. Similar to our study of 3 patients with a second MVD, the arterial vessels were fully decompressed and the main cause of failure was the overlooking of vein compression.

Li et al. reported 13 early repeat MVD (several days after the first surgery) based on the following inclusion criteria: (1) The first MVD failed, the spasm was not resolved at all or became more severe, and (2) AMR persisted during the first MVD or disappeared once but re-emerged after the closure of the dural incision with the same or even at a higher amplitude. All 13 patients had good or excellent spasm resolutions immediately after the reoperation, which involved whole-range exploration and intraoperative AMR monitoring. One patient received a second MVD within a 4-month interval (No. 3 in Table 1) who completely failed the first MVD. However, once patients show partial relief, the outcome is different. We question the necessity of early repeat surgeries. According to our experience, many patients show delayed relief—symptoms disappearing after 3 months or longer is quite common. Jo et al. found that among 70 patients with residual or recurrent spasms >1 year after surgery, 11 showed gradual improvement over 3 years, and one showed delayed improvement >3 years after surgery. Intraoperative resolution of AMR after decompression and severe indentation were significant predictors of a good long-term outcome after MVD for HFS. Chang et al. reported a probability of delayed

| Table 2. Distribution of Conflicting Veins in Patients with Hemifacial Spasm |
|---------------------------------|-----------------|-----------------|-----------------|-----------------|
| Author (year)                  | Total           | Veins with Arteries (%) | Veins Alone (%) | Location of Compression | AMR Monitoring |
| Huang et al., 1992             | 310             | 2 (0.6)            | Not mentioned   | Not mentioned       |
| Barker et al., 1995            | 648             | 132 (20.4)         | 19 (2.4)        | Not mentioned       |
| Samii et al., 2002             | 145             | 2 (1.4)            | 6 (4.1)         | Not mentioned       |
| Park et al., 2008              | 13              | 1 (7.7)            | 1: RExP         | Not mentioned       |
| Park et al., 2008              | 236             | 1 (0.4)            | Not mentioned   | Not mentioned       |
| Campos-Benitez and Kaufmann 2008 | 115            | 4 (3.4)            | 3: AS, 1:RDP    | Not mentioned       |
| Sindou and Keravel 2009        | 147             | 13 (13)            | (0.7)           | Not mentioned       |
| Zhong et al., 2012             | 1327            | 3 (3)              | 0               | Not mentioned       |
| Wang et al., 2013              | 33              | 19 (57.58)         | Not mentioned   | Detailed            |
| Present study (2018)           | 78              | 5 (6.4)            | 4: RExP, 1: CP  | Detailed            |

AMR, abnormal muscle response; RExP, root exit point; AS, attached segment; RDP, root detached point; CP, cisternal portion.
*Not mentioned: no data or analysis of AMR; not detailed: only brief data for AMR monitoring.
Judging from figure.
Focus on second surgery.
Including second surgery.
recurrence of 1.0%, 1.7%, and 2.9% at 1 year, 2 years, and 5 years after surgery. Our policy is observation, especially in patients whose symptoms show partial relief. We were concerned about missing responsible vessels, and discussed the possibility of a second MVD.

We list 9 reports including our series that focused on veins (Table 2). Among them 0%–6.4% of facial spasms were caused by veins. 4,5,9,10,12-27 In the second surgery, the number of veins increased. Campos-Benitez and Kaufmann4 determined the nature of neurovascular compression in 115 consecutive patients undergoing their first MVD for HFS. The primary culprit location was at RExP in 10%, AS in 64%, RDP in 22%, and CP in 3%. They found 3 cases of venous compression in AS, and 1 case in RDP. In our series, 4 cases of venous compression were found in RExP, and 1 in CP, which is not a common location. We would like to stress that we should carefully look for vessels not only in common locations but also at RExP and CP at the second surgery or when AMR persists.

CONCLUSIONS

Veins can be offending vessels in HFS patients, although this is not very common (6.4% in our database). AMR is useful to determine the endpoint in these cases. Once arteries are decompressed thoroughly with residual AMR, surrounding veins at unusual sites, such as RExP or CP, must be checked to prevent persistent HFS. Complete decompression of veins leads to a good clinical outcome.

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