Good Surgical Outcomes of Hemifacial Spasm Patients with Obvious Facial Nerve Indentation and Color Change

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OBJECTIVES: **Hemifacial spasm results from vascular compression of the facial nerve. It remains controversial whether severe compression and subsequent nerve indentation predict a good or a poor surgical outcome. Here, to illustrate the relationship between the degree of neurovascular compression and surgical outcome, we conducted a retrospective case–cohort study focused on patients whose facial nerve was seriously compressed.**

METHODS: **This study included 2 groups. The nerve-indentation group included 48 patients with hemifacial spasm whose facial nerves had obvious indentation and color change at the site of neurovascular conflict. The control group included 48 randomly selected patients with hemifacial spasm without facial nerve indentation or color change who were surgically treated by the same team during the same period. The surgical findings, intraoperative lateral spread response results, and clinical outcomes were compared.**

RESULTS: **Single-vessel compression was found more frequently in the nerve-indentation group (87.5%) than in the control group (60.4%, $P < 0.05$). The lateral spread response (LSR) resolution rate of the nerve-indentation group was 91.7%, and that of the control group was 87.5% ($P > 0.05$). The rates at which the microvascular decompression procedure was successful were equal in the nerve-indentation and the control groups (93.8% vs. 91.7%, $P > 0.05$).**

CONCLUSIONS: **Severe vascular compression and subsequent nerve indentation were correlated with a greater possibility of single compression and a lower incidence of multiple neurovascular conflicts in patients with hemifacial spasm, making the microvascular decompression procedure more accurate and easier. Therefore nerve indentation might predict good surgical outcomes.**

INTRODUCTION

Hemifacial spasm (HFS) generally results from the vascular compression of the facial nerve.[1-5] Pathologic changes, such as demyelination, vacuolization of the myelin sheath, and partial degeneration of axons, occur at the neurovascular compression site.[6] Microvascular decompression (MVD), which detaches the offending vessels from the facial nerve,[7] has become the most reliable treatment for this disorder because it usually produces excellent results while preserving cranial nerve function.[7,8] The overall cure rate of MVD for HFS is 84%–95%.[8,9] Spasms resolve immediately after surgery in many patients, whereas others may get delayed relief in 1–2 years, and still others may not benefit from the MVD procedure.[3-5,10-14] At present, patients who will get relief from MVD cannot be identified in advance. However, the degree of vascular compression (i.e., the etiologic factor) may predict the outcome to some extent. For example, a prospective study revealed that indentation on the facial nerve caused by serious vascular compression was associated with good outcomes.[15] A recent retrospective study also indicated that intraoperative resolution of the lateral spread response (LSR) after decompression and severe indentation of the facial nerve were significant predictors of good long-term outcome after MVD for HFS. If the symptoms do not stop soon after the operation, but the surgeon confirms intraoperative resolution of the LSR and severe indentation, the chance for a

Key words
- Facial nerve
- Hemifacial spasm
- Indentation
- Microvascular decompression

Abbreviations and Acronyms
HFS: Hemifacial spasm
LSR: lateral spread response
MVD: Microvascular decompression
REZ: Root exit zone

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delayed cure is likely and a reoperation can be postponed for 3 years after MVD.[16] However, this issue remains controversial. On the basis of a retrospective review of 232 cases, Kim et al.[17] reported that severe indentations and color changes of the facial nerve may be risk factors for poor surgical outcomes. Thus we conducted this retrospective case–cohort study with an emphasis on those patients whose facial nerve was seriously compressed and consequently became indented and even grayish to illustrate the relationship between degree of neurovascular compression and surgical outcome.

PATIENTS AND METHODS

Design of Retrospective Case–Cohort Study

This study was approved by the Ethics Committee of Xinhua Hospital. The exposure factor was obvious indentation of the facial nerve, which was defined in terms of the following 3 criteria: 1) The morphologic change was located at the site of neurovascular conflict; 2) The caliber was diminished by one third or more or was ribbon shaped. The calibers of the compressed site and that of the normal site were compared visually. This criterion was subjective because it is difficult and dangerous to detect the caliber of the facial nerve with a tape measure in such a deep place. 3) This portion became grayish or even translucent. If these criteria were met, the case was assigned to the "nerve-indentation group." In contrast, the control group consisted of patients without this morphologic change.

The operation and postoperative treatment were the same for patients in both groups. The operative findings, intraoperative LSR resolution results, and surgical outcomes of the 2 groups were compared. The study was retrospective and observational, and no intervention was provided (i.e., when the study began, all surgical treatments had been completed. We just observed the relationship between the exposure factor [obvious indentation] and results.).

Patients

We performed the MVD procedure for 895 patients with hemifacial spasm during January 2009–December 2010. Of these, 48 patients with hemifacial spasm whose facial nerves were obviously indented and had changed color at the site of neurovascular compression were included in the "nerve-indentation group." Another 48 patients with HFS, who were surgically treated by the same team during the same period but had no facial nerve indentation, were selected randomly to serve as the control group. The clinical features, surgical findings, intraoperative LSR results, and spasm resolution data of the 2 groups were compared.

Operative Procedure

MVD was performed under general anesthesia via a lateral retrosigmoid suboccipital approach with the patient in the contralateral decubitus position.[18] The arachnoid membrane was opened sharply, and the facial nerve was fully exposed. Dissection began from the caudal cranial nerves, and then the arachnoid membranes between the VII and VIII and caudal cranial nerves were opened sharply. The cerebellum and flocculus were raised gradually until the pontomedullary sulcus was exposed. Next, the arachnoid membrane between the acoustic nerve and cerebellum and that rostral to the VII–VIII nerves were dissected. At this stage, the entire arachnoid membrane surrounding VII–VIII and the caudal nerves were opened thoroughly, and the VII–VIII nerves from the brainstem to the internal meatus were exposed sufficiently. The entire course of the facial nerve was thoroughly explored, as reported previously.[1] Tracing the offending vessel began from the attached segment (AS), proceeded to the root exit zone (REZ) and cisternal portion, and ended at the root exit point. This full-course exploration principle was applied to both groups of patients (i.e., even if obvious neurovascular conflict was found at the REZ, the entire facial nerve root was exposed and scrutinized to identify offending vessels). After identifying the offending vessel, a Teflon sponge was placed between the vessel and brainstem to transpose the course of the offending vessel. If transposition was impossible, thin pieces of Teflon felt were interposed between the culprit vessel and the facial nerve.

Intraoperative Electrophysiological Monitoring

The LSR was monitored using an evoked potential system (Medtronic Keypoint 4; Dantec Dynamics, Skovlunde, Denmark). The LSR was recorded from either the mentalis muscle while stimulating the temporal branch of the facial nerve or from the orbicularis oculi muscle while stimulating the marginal mandibular branch. After the dura mater was opened, the LSR was monitored continuously until the operation was finished. The LSR was induced by 10–15-mA electrical stimulation. During the decompression stage, the LSR often disappeared intermittently or decreased in amplitude or frequency, so we increased the stimulation intensity. If the LSR disappeared and never reemerged when the stimulation intensity was increased to a maximum value of 100 mA, we considered the LSR to be completely resolved.

Surgical Outcome Evaluation

Surgical outcomes were evaluated as reported previously[19] using the following ratings: "excellent" if HFS was absent; "good" if HFS was >90% resolved; "fair" if HFS was >50% resolved; "poor" if HFS was <50% resolved; and "failure" for no resolution or even deterioration. "Immediate resolution" was assigned when the spasm was relieved the day after the operation. Otherwise, if the symptoms diminished gradually over time, it was defined as "delayed resolution." All operative complications were recorded. Postoperative follow-up was done primarily by telephone interviews and at least 1 outpatient interview. The follow-up period was 5–7 years.

RESULTS

In the nerve-indentation group, a single offending vessel was present in 42 cases (87.5%) and multiple vessel compression was present in 6 cases (12.5%). The anterior inferior cerebellar artery was the most frequently offending artery in the nerve-indentation group. In contrast, only 60.4% patients (29 cases) in the control group had a single offending vessel, and 39.6% patients (19 cases) in this group had multiple vascular compressions. Arterioles were the culprit vessels in 6.3% of patients (3 cases) in the control group, but it was the culprit in no patients in the nerve-indentation group. Thus the difference in the incidence of single-vessel compression between the 2 groups was significant ($P < 0.05$, chi-square).