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Clinical Article

Hemifacial spasm: neurovascular compressive patterns and surgical significance

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Summary

Background. The aim of this study was to report further investigation of neurovascular compression as a cause of hemifacial spasm (HFS) and to provide useful surgical guidelines by describing the compression patterns.

Material and methods. From January 2004 to February 2006, 236 consecutive patients with HFS underwent microvascular decompression (MVD) in a single centre. Based on the operation and medical records, the intraoperative findings and post-operative outcomes were obtained and analysed.

Results. We found that 95.3% of lesions had accompanying causative factors that made the neurovascular compression inevitable. Based on the contributing factors, compression patterns were categorised into six different types including: loop ($n = 11$: 4.6%), arachnoid ($n = 66$: 27.9%), perforator ($n = 58$: 24.6%), branch ($n = 18$: 7.6%), sandwich ($n = 28$: 11.9%), and tandem ($n = 52$: 22.0%). The compression patterns were significantly correlated with the compressing vessels involved. Thirty-two (86.5%) of 37 lesions where the vertebral artery was the compressing vessel involved the tandem type. Anterior inferior cerebellar artery was the compressing vessel involved in 49 (84.5%) of 58 perforator type compressions, while posterior inferior cerebellar artery was the compressing vessel involved in 8 (72.7%) of 11 loop type compressions.

Conclusions. Once the compressing vessel responsible for the neurovascular compression are identified, the probable pattern of compression can be anticipated; this knowledge could facilitate the application of the appropriate operative procedures and minimise post-operative complications.

Keywords: Cranial nerve vascular compression syndrome; microvascular decompression; compression pattern; hemifacial spasm.

Introduction

Hemifacial spasm (HFS) is mainly caused by pulsatile neurovascular contact at the root exit zone (REZ) of the facial nerve, except for a limited number of cases resulting from known underlying diseases [4] such as tumours, demyelinating disorders or infections [7, 13, 14]. Although neurovascular compression has been identified as the cause for HFS [6, 13, 14], there still are several questions that have not been sufficiently answered: (1) why do some vessels adjacent to the nerve create a neurovascular compression, while others adjacent to the nerve do not? (2) why does not every patient become symptom-free immediately after a successful microvascular decompression (MVD) procedure? (3) why do Asian people have a higher incidence of HFS than Caucasians? Clarification of these unsolved questions would contribute to the full understanding of the patho-

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physiology and, accordingly, better treatment results with less post-operative complications. This study was designed to be a step for answering one of the above questions: *what makes an adjacent vessel compress the facial nerve*? During the MVD procedures, we have realised that compression of the facial nerve is almost always influenced by other contributing factors such as thick arachnoid trabeculae, perforating arteries or adjacent collateral vessels. Here, based on the contributing factors, we describe and categorise the patterns of neurovascular compression into six types, and analyse the clinical significance for post-operative outcome and development of complications.

Patients and methods

Patient population

Between January 2004 and February 2006, 236 consecutive patients who underwent MVD for HFS were retrospectively identified into the study. All of the surgical procedures were performed by the same surgeon (Park K) at the Samsung Medical Center. The clinical records of all patients included pre-operative symptoms, intra-operative findings, post-operative outcomes and complications. In particular, microanatomical observations under the microscopic guidance were entirely dependent upon the interpretation by the senior author (Park K) to minimise inter-observer variability. To ensure complete ascertainment of patients and complete of follow up, our nurse practitioner (J. A. Lee) was responsible for these aspects. All patients underwent pre-operative evaluation by computed tomography (CT), magnetic resonance imaging (MRI) with and without the addition of a contrast agent, and three dimensional time of flight MR angiography (3-D TOF MRA). Pure tone audiometry and speech audiometry were performed pre- and post-operatively by an otologist in all patients.

Surgical technique

All of the procedures were performed via a lateral retrosigmoid suboccipital approach, which has been previously described in the literature [9, 18, 19, 26, 29]. After careful dissection of the arachnoid membrane and gentle retraction of the flocculus, the root exit zone (REZ) of the facial nerve was observed. The compressing vessel, so called as the offender, was identified near to the REZ. Several pieces of Teflon sponge were placed between the compressing vessel and the REZ. We used a Teflon sponge in two different sizes (5×15 and 2×10 mm). During manipulation of the VIIth and VIIIth nerves, BAEPs for hearing loss and facial EMG for lateral spread response phenomenon were closely monitored, which facilitated a complete decompression [18]. The dura was closed with several pieces of muscle interposed between the interrupted sutures to prevent cerebrospinal fluid leakage [18].

Evaluation of outcome and statistic analysis

The post-operative outcomes were evaluated by a specialised nurse practitioner (Lee JA), who did not know the patient's intra-operative findings, on post-operative day one and three and at three weeks, three months and 12 months and more frequently if needed. Telephone interview was used additionally when patients could not attend a follow-up visit. Since there is no universally approved scale system to measure the symptoms of HFS so far, we asked patients where their spasms would be on the scale of 0–10 if ten stood for the worst spasm they had ever had before the surgery in terms of both frequency and intensity. Both face-to-face ($n = 191$) and telephone interviews ($n = 45$) were carried out by the same nurse practitioner, and the data reviewed by the author. The surgeon also asked patients about post-surgical residual symptoms and any complications to validate the data gathered by the nurse practitioner. The results of the Fisher's exact test are reported with two-by-two tables. Probability values less than 0.05 were accepted as significant differences. The statistical technique was advised by a statistician (Kim SW, PhD.)

Results

Of 236 patients, 171 patients were female and 65 patients were men. The mean age was 45.4 years (ranging from 29 to 63 years). The pre-operative symptom duration was 64.1 months (ranging from 5 to 360 months) and the mean post-operative follow up period was 16.7 months (ranging from 7 to 29 months).

Microanatomical observations

The intra-operative findings included the compressing vessels along with the contributing factors. The vessel that compressed the facial nerve was recorded as the compressing vessel (Table 1), and was identified in all patients. Pathological structures that made the compression inevitable were recorded as the contributing factors,

Table 1. *Base-line characteristics of the 236 patients*

Characteristic	Value
Total – no.	236
Female: male (ratio)	171:65 (2.63:1)
Mean duration of symptoms (months)	64.1 ± 51.39 (5–360)
Mean follow up time (months)	16.7 ± 7.72 (7–29)
Compressing vessel involved – no. (%)	
Anteroinferior cerebellar artery (AICA)	122 (51.7%)
Posteroinferior cerebellar artery (PICA)	51 (21.6%)
Vertebral artery (VA)	4 (1.7%)
AICA + PICA	25 (10.6%)
AICA + VA	25 (10.6%)
PICA + VA	5 (2.1%)
AICA + PICA + VA	3 (1.3%)
Vein	1 (0.4%)

which included thick arachnoid trabeculae, perforating arteries or adjacent collateral arteries. Six different types of compression patterns were identified based on the contributing factors (Tables 2 and 3). The decision regarding the compression pattern was made by the surgeon during the procedure and written on the operation record. In cases where two or more contributing factors seemed to exist, the surgeon selected the one that made the greater influence on the compression. The loop, arachnoid and perforator types had a single compressing vessel, while the branch, sandwich and tandem types had two or more compressing vessels. A single vessel was responsible for 138 (58.5%) compressions, while two or more arteries were identified as compressing vessels in 98 (41.5%) lesions.

Table 2. *Number of compressing vessels* involved in each compression pattern*

	VA ($p < 0.001$)	AICA ($p = 0.005$)	PICA ($p = 0.003$)
Loop type	2	1	8
Arachnoid type	3	40	25
Perforator type	0	49	9
Branch type	0	14	4
Sandwich type	0	26	13
Tandem type	32	43	25
Miscellaneous	0	2	0
Total	37	175	84

* Table 2 shows the number of compressing vessels (VA, AICA or PICA) involved in each compression pattern. In cases where two or more vessels were involved in a compression, each vessel was counted individually; therefore the numbers listed in Table 2 differ from those in Table 3.

Only 11 (4.7%) of 236 patients had no contributing factor, i.e., the vascular loop itself was the only cause of the neurovascular compression (Fig. 1A). We categorised these lesions as the loop type. In 66 (28.0%), thick arachnoid trabeculae between the vessel and the brainstem caused the vessel to be tethered tightly to the nerve (Fig. 1B), and were categorised as the arachnoid type, because the thickened arachnoid trabecula was the contributing factor that forced the vessel to compress the REZ. In many cases of the arachnoid type, only the removal of arachnoid trabeculae could offer sufficient decompression. We, however, inserted a few pieces of Teflon in order to prevent a recurrence. The perforator type ($n = 58$: 24.6%) was analogous to the arachnoid type in that the perforating arteries from the compressing vessel caused compression by tethering the vessel to the brainstem similar to the thick arachnoid trabeculae in the arachnoid type (Fig. 1C). However, more caution was required to decompress the perforator type compressions, because even the slightest damage of perforating artery could create a devastating outcome.

The branch, sandwich and tandem type compressions involved two or more vessels. Eighteen (7.6%) patients had the branch type compression (Fig. 1D). When a

Table 3. *Compression pattern and surgical outcome*

	No. of cases	Mean follow-up time (months)	Post-operative response-no.		Complication-no.	
			Cure	No response	Facial palsy*	Hearing loss
Loop type	11	11.8 ± 8.86	11	0	0	0
Arachnoid type	66	18.3 ± 7.65	61	5	4	1*
Perforator type	58	17.5 ± 7.40	55	3	6	0
Branch type	18	14.2 ± 8.70	17	1	1	0
Sandwich type	28	16.4 ± 7.35	26	3	3	1†
Tandem type	52	16.1 ± 7.45	49	3	8	0
Miscellaneous	3	13.7 ± 5.51	3	0	0	0
Total	236	16.7 ± 7.72	221 (93.64%)	15 (6.36%)	22 (9.32%)	2 (0.85%)
p-value			$p = 0.58$		$p = 0.59$	$p = 0.55$

* Temporary complication (resolved within six months).
† Permanent complication.

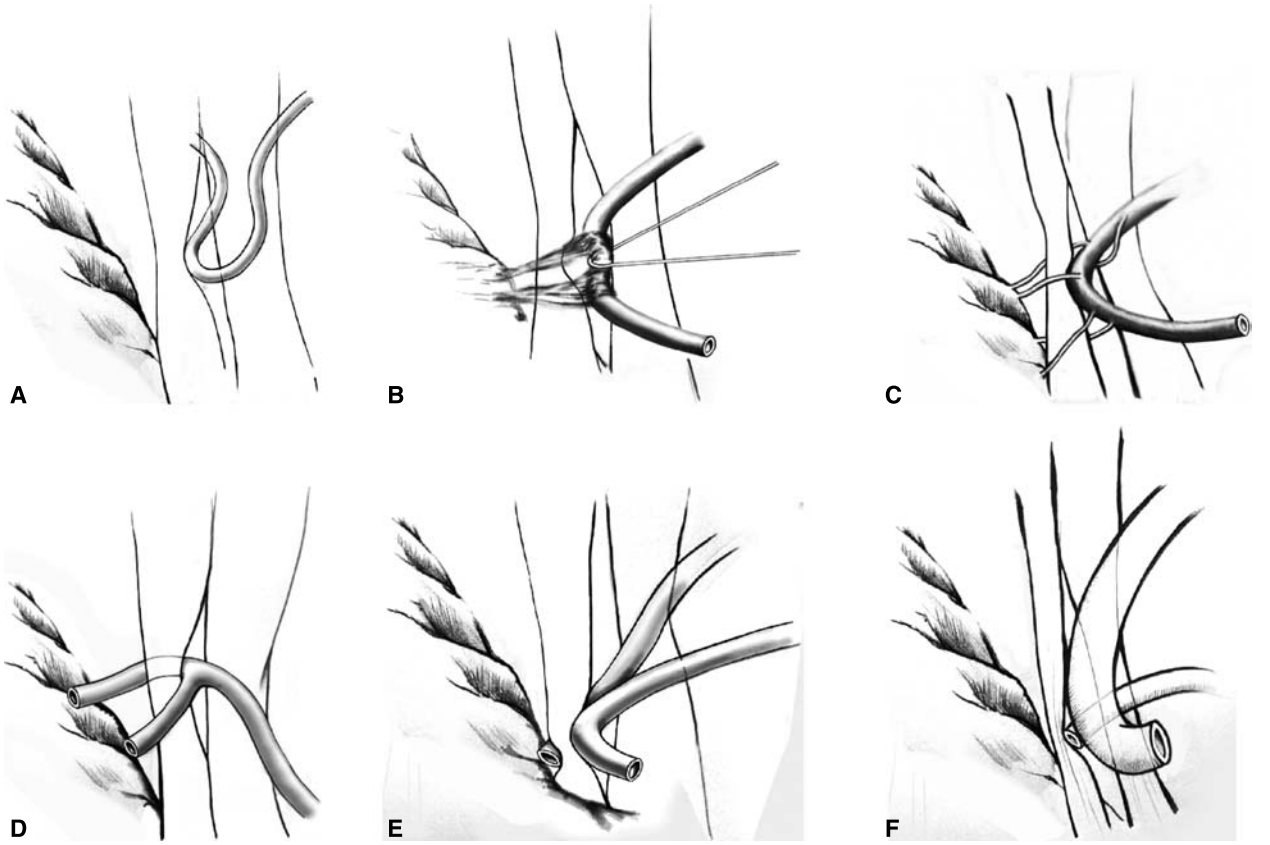

Fig. 1. Patterns of neurovascular compression. (*A*) Loop type, the vascular loop itself without any contributing factors creates the compression. (*B*) Arachnoid type, thick arachnoid trabeculae between the vessel and the brainstem cause the vessel to be tethered tightly to the nerve. (*C*) Perforator type, the perforating arteries from the compressing vessel cause compression by tethering the vessel to the brainstem. (*D*) Branch type, the nerve is caught in between the compressing vessel and its branch. (*E*) Sandwich type, the nerve is sandwiched in between two different vessels independently. (*F*) Tandem type, one vessel compresses another vessel which, in turn, compresses the nerve. *VII* Facial nerve; *VIII* vestibulocochlear nerve; *BS* brain stem; *Cbll* cerebellum, retracted; *R* tapered retractor; *CV* compressing vessel; *Solid arrow head* thick arachnoid trabeculae; *Open arrow* a string that detaches the compressing vessel from the cranial nerve; *Open arrow head* perforating arteries from the compressing vessel to the brain stem

compressing vessel branched off near the REZ, the nerve could be caught in between the two branches. Given that the contributing factor in such cases was the two branches, those compressions were categorised as the branch type. The sandwich type compressions were observed in 28 (11.9%) patients (Fig. 1E). Where the nerve was sandwiched in between two different vessels independently, we called them sandwich type compressions. In order to achieve a complete decompression of sandwich type lesions, a careful inspection of the ventromedial aspect of the REZ was critical, because the decompression of only one vessel on the dorsal aspect of the REZ could leave the patient susceptible to a recurrence. The tandem type ($n = 52$: 22.0%) defined the situation where one vessel compressed another vessel which, in turn, compressed the REZ (Fig. 1F). The ''miscellaneous'' compression group included three atypical examples: two patients who individually had a vessel (AICA in both cases) held between the seventh and eighth nerve and one who had a compressing vein instead of as artery (Tables 2 and 3).

There were statistically significant correlations between the compression patterns and the compressing vessels involved in each pattern (Table 2). The involvement of the VA had a strong predilection for the tandem type, while that of AICA and PICA were associated with the perforator and loop types. No statistically significant correlations, however, existed between the compression pattern and mean symptom duration (64.9 ± 51.37 months, $p = 0.76$).

Outcome of surgery and complications

The results of surgery are shown in Table 3. Complete or near complete resolution of HFS was observed in 221 patients (93.64%) at the three-month follow up. Patients

with no spasm at all were included in the complete resolution group, and those whose residual symptoms were minimal were categorised as a near complete resolution group. Both groups were considered cured.

The different compression patterns had no statistically significant difference in relation to post-operative outcome and development of post-operative complications including facial palsy or hearing loss; however, the tandem type had the highest incidence rate (8 of 52, 15.4%) of facial palsy. All facial palsies ($n = 22$: 9.32%) were temporary and none lasted for more than six months. Among 236 patients, only two hearing losses (0.85%) were noted: one with the arachnoid pattern and the other with the sandwich pattern. While the hearing loss from the arachnoid pattern was temporary, that from the sandwich pattern was a high-frequency sensorineural type and permanent. The loop type, which had no contributing factors other than the vascular loop itself by definition, had a 100% cure rate with no post-operative complications; however, this finding lacked statistical significance.

Discussion

Neurovascular compression has been accepted as the aetiology and has allowed for the development of MVD. Dandy [5] described a conflict between the fifth cranial nerve and the vessel, and Gardner and Sava [7] suggested that there were some similarities between trigeminal neuralgia and HFS. Influenced by those studies, Jannetta and colleagues [13, 23] pioneered the concept of MVD for both trigeminal neuralgia and HFS. Even though there remains controversy over the pathogenesis and management of HFS [1, 12, 30, 31], many investigators have reported relatively high success rates following MVD, ranging from 79 to 97% [2, 10, 11, 20, 24, 27, 28]. We performed MVDs with a similar success rate of 93.64%; however, the ratio of multiple compressing vessels to single compressing vessel (98:138, 0.71:1) was significantly higher than of previously reported (0.07:1–0.18:1) [3, 25]. In order to minimise the possibility of recurrence, we focused on finding other factors that made the neurovascular compression inevitable, even after seemingly successful decompression was performed; therefore, more vessels were found and recognised as the compressing vessels.

The contributing factors that influence the neurovascular compression have been discussed by several authors. The incidence of HFS in middle aged or older patients could be associated with focal atherosclerosis or hypertension, while that in young people could be associated with thick arachnoid membranes or a cerebellopontine angle arachnoid cyst [8, 15, 16]. Based on the previously mentioned studies and our surgical experiences, we assumed that a single vessel compressing the facial nerve could not explain the full mechanism of neurovascular compression [2, 20, 28]. For the categorisation of the compression pattern, we utilised the contributing factors, such as thick arachnoid trabeculae, branching artery, perforating arteries and other adjacent arteries, because only 4.7% (loop type) of all cases had no contributing factors other than the vascular loop itself (Table 3).

A strong correlation was noted between the involved vessel (compressing vessel, offender) and the compression pattern (Table 2). Thirty-two of 37 (86.5%) compressions involving the VA were the tandem type. If pre-operative MRI with 3D TOF MRA shows that the VA would be involved, surgeons should consider the possibility of a tandem type and expect a second vessel compressed by the VA to be involved. Forty-nine of 58 (84.5%) compressions of the perforator type involved the AICA as the compressing vessel, while eight of 11 (72.7%) compressions of the loop type involved the PICA (Table 2). This difference might be explained by the anatomy of the perforating artery [21, 22] or the proximity of the compression site on the offender; however, this remains to be proven. However, the fact that 83.5% of compressions of the perforator type involved the AICA would help surgeons to be more careful not to injure perforating arteries when they decompress lesions involving the AICA.

As a result of surgery with the knowledge of variable compression patterns, we have acquired some information useful for the operative procedure. The arachnoid type compression, which has thick arachnoid trabeculae between the facial nerve and the brainstem, is usually associated with a thick arachnoid membrane surrounding the cerebellopontine cistern. Therefore, careful arachnoid dissection is crucial to achieve a successful post-operative result. Both sandwich and tandem types involve two or more compressing vessels; surgeons should pay more attention to the possibility of an undetected vessel when they decompress the lesion in patients with one of these types. Decompressing the branch type lesion demands more careful manoeuvre with more pieces of Teflon felt than other types because the nerve is unmovable between the branches; however, identifying the lesion is less demanding. We recommend the transposition of the compressing vessel for the decompression of the branch type lesion. The loop type has been defined as a compression without any other con-

tributing factors. However, the vascular loop appears to cause compression especially when it is transposed; five out of 11 loop type compressions required the re-transposition of vessels during MVD. We assume that the excellent post-operative results (100% of cure rate without any complications) after decompressing the loop type lesion might be derived from the simplicity of operative procedure (re-transposition) and the absence of other structural obstacles such as perforating arteries or thick arachnoid membrane.

In terms of the post-operative outcomes and complications, no significant differences were identified among the different compression patterns (Table 3). Since not all the patients become spasm-free immediately after the MVD, an appropriate follow-up period is critical to evaluate the surgical outcome. Samii [28] reported that 108 (92.3%) out of 117 patients became spasm-free six months after surgery; Kondo [18] showed that the symptoms due to manipulation of cranial nerve during the surgery might be masked for a period of up to six to 12 months after surgery. In our series, the mean follow-up time was 16.7 months; however, 19 (8.1%) patients had a follow-up period for less than six months. We plan to chronologically analyse the recovery pattern to find out the optimal time for evaluation of the surgical outcome.

Conclusions

We have categorised the patterns of neurovascular compression into six different types. The compression patterns are correlated with the compressing vessel involved. Since the involved artery can be anticipated by the use of preoperative MRI, surgeons can assume the most probable pattern of compression; and this knowledge of the compression pattern could be helpful to perform more appropriate surgical procedures with fewer post-operative complications. In terms of the post-operative outcome or the development of complications, there are no significant differences among the different compression patterns; however, we assume that there are specific risk factors. Therefore, we plan to focus further attention on identifying the specific risk factors that contribute to post-operative outcome in future studies.

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Comment

This article, from an important series of hemifacial spasm (HFS) treated with micro-vascular decompression (MVD), provides interesting anatomical date. Classification of the vascular compression(s) types into six subgroups is useful in that it has practical implications for decompression techniques. Knowing this classification should help neurosurgeons to better understand-pre-operatively on 3D-TOF-AngioMR-what they will be confronted with at surgery and to make decompression safer and more effective.

Like other published series, including ours, the authors found multiple compressions in 24.6% of the cases. The authors found it easy to clearly identify at surgery the offending vessel(s). This was not as easy for us; we sometimes had difficulty knowing whether the loop(s) was (were) arising from: AICA or PICA.

It would have been interesting if the authors correlated the findings of an arachnoidal type with the age of the patients, to confirm their hypothesis that such a factor would explain genesis of HFS in non-arteriosclerotic patients.

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