Surgical Treatment of Secondary Hemifacial Spasm: Long-Term Follow-Up

Xin Zhang, Xu-Hui Wang, Hua Zhao, Yin-Da Tang, Ping Zhou, Yan Yuan, Jin Zhu, Shi-Ting Li

BACKGROUND: **Hemifacial spasm (HFS) is generally caused by the root exit zone of the facial nerve compressed by an overlying arterial loop. HFS can also be caused by various types of tumor, aneurysm, or arteriovenous malformation. We retrospectively analyzed patients to evaluate possible differences in the demographic and clinical features between primary and secondary HFS.**

METHODS: **A retrospective study of 3140 cases of HFS treated in our department between January 2009 and June 2016. Among the 3140 total cases, 26 patients had secondary HFS.**

RESULTS: **The 26 tumors of secondary HFS included 11 meningiomas, 8 epidermoid cysts, and 7 vestibular schwannomas. Compared to those with idiopathic HFS, those patients with tumor-induced HFS were significantly younger ($P < 0.05$). Secondary HFS tended to have responsible vessels, and were observed in 20 (76.92%) of these 26 patients. The long-term effective rate of operation was 84% in the secondary HFS group and was 96.45% in the primary HFS group ($P < 0.05$); the incidence rate of complication was 12% in the secondary HFS group and was 3.06% in the primary HFS group ($P < 0.05$).**

CONCLUSIONS: **In cases of secondary HFS, facial nerves of most patients were compressed by blood vessels, so microvascular decompression after tumor resection plays an important role. We should examine the entire nerve root for possible vascular compression.**

INTRODUCTION

Hemifacial spasm (HFS) is a chronic facial nerve disorder characterized by spontaneous, unilateral, and repetitive facial muscle contractions.[1] Those caused by vascular compression of the facial nerve roots were clinically regarded as primary HFS.[2-4] However, some patients with HFS have symptoms caused by tumors, trauma, infections, and so forth, which were regarded as secondary HFS.[5-7] Numerous authors had reported that various lesions may induce secondary HFS. The common etiology are tumors in the posterior fossa, posterior circulation aneurysms, vascular malformations, cysts in cerebellopontine angle and even contralateral lesions,[7] but there is still controversy about exact mechanism and pathogenesis of secondary HFS.

We retrospectively analyzed the relationship between tumor, blood vessel, and facial nerve in these patients to provide some reference for the pathogenesis of secondary HFS.

MATERIAL AND METHODS

Patients

We analyzed the records of 3140 patients with HFS who underwent microvascular decompression (MVD) surgery by Dr. Li at Xinhua Hospital between January 2009 and June 2016. Among the 3140 total cases, 26 patients were secondary HFS. These patients were analyzed in this study and their clinical features, as well as intraoperative findings and postoperative outcomes, were retrospectively investigated.

Surgery

Simple MVD for Patients of Primary HFS. All patients of secondary HFS underwent resection of intracranial tumor in the lateral

Key words
- AMR
- Hemifacial spasm
- HFS
- MVD

Abbreviations and Acronyms
AMR: Abnormal muscle response
HFS: Hemifacial spasm
MVD: Microvascular decompression

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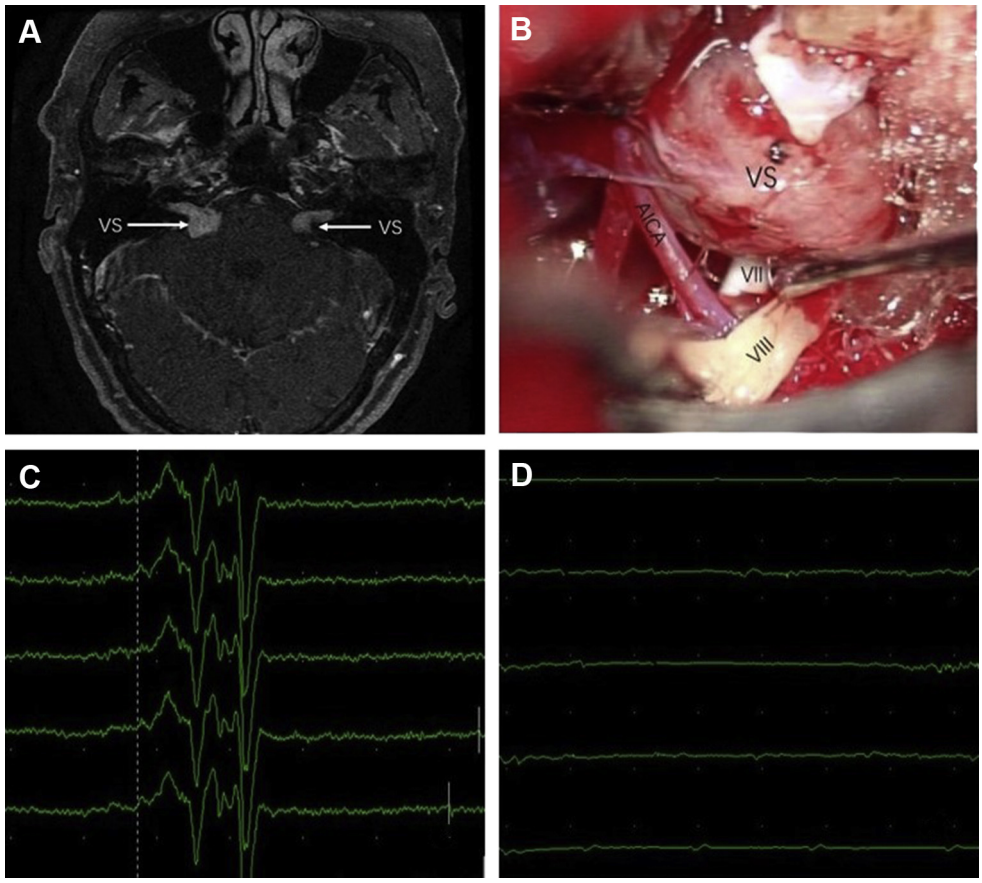

Figure 1. A secondary left hemifacial spasm caused by vestibular schwannoma. (**A**) Preoperative T1+weighted magnetic resonance imaging showed bilateral vestibular schwannoma. (**B**) During the operation, we can see the anterior inferior cerebellar artery between the facial nerve and the vestibular nerve pushed by the tumor. (**C**) Before operation, the abnormal muscle responses were positive. (**D**) Abnormal muscle responses disappeared after the operation. AICA, anterior inferior cerebellar artery; VII, facial nerve; VIII, auditory nerve; VS, vestibular schwannoma.

decubitus position via a standard retrosigmoid craniotomy and an infrafloccular approach. After the tumor resection was completed, we conducted a full range exploration of the facial nerve to identify any offending vessels. The offending vessel(s) was shifted off the facial nerve by inserting small pieces of shredded Teflon felt between the vessels and the brainstem or flocculus (Figure 1).

Intraoperative Monitoring

Abnormal muscle responses (AMRs) were recorded by electrical stimulation of the zygomatic and marginal mandibular branches of the facial nerve. Paired stainless-steel needle electrodes were subdermally inserted into both the lower edge of the zygomatic bone (zygomatic branch stimulation) and the lower edge of the mandibular bone (marginal mandibular branch) ~3 cm apart from the midline. AMR was continuously recorded and printed out at 1-minute intervals via amplifiers using 50 times summation; responses were filtered to the frequency band of 5 Hz–3 kHz. Stimulation (0.1 ms, rectangular wave, 2 Hz) was adjusted to supramaximal strength.[8] If any AMR waveform appeared, we considered the AMR to be positive. If the AMR disappeared completely, or the amplitude decreased to <50% compared to the baseline level, the AMR response was considered negative (Figure 1).

Evaluation and Statistical Analysis

A comprehensive analysis of the surgical outcomes at 1 day, 7 days, 1 month, 3 months and 1 year after surgery, including complications, was performed based on the analysis described by Kondo et al.[9] The efficacy of MVD was categorized as poor (no improvement), fair (moderate spasm, apparently persisting), good (occasional slight spasm), or excellent (complete disappearance of spasm). "Excellent" and "good" were defined as effective. Statistical analyses were performed using SAS software version 9.1.3 (SAS Institute Inc., Cary, North Carolina, USA). Continuous variables were presented as mean ± SD, and categorical variables were presented as

Table 1. Summary of the Secondary Hemifacial Spasm Patients

Case	Age (Years)/Sex	Side	Pathology	Offending Vessels	Outcome	Complications
1	23/F	L	Meningioma	AICA	Excellent	None
2	54/F	R	Epidermoid	None	Poor	None
3	28/M	R	Meningioma	AICA, PICA	Good	Facial palsy
4	62/F	L	Meningioma	AICA	Excellent	None
5	36/F	L	Epidermoid	None	Excellent	None
6	58/F	L	Vestibular schwannomas	AICA, PICA	Excellent	Facial palsy
7	42/M	R	Meningioma	None	Poor	None
8	38/F	L	Epidermoid	AICA	Good	None
9	64/F	R	Vestibular schwannomas	PICA	Excellent	Facial palsy
10	71/F	L	Meningioma	AICA	Fair	None
11	35/F	L	Epidermoid	None	Excellent	None
12	48/F	L	Vestibular schwannomas	AICA	Excellent	Hearing disturbance
13	66/M	R	Meningioma	AICA	Excellent	None
14	68/F	R	Meningioma	PICA	Excellent	None
15	72/F	L	Epidermoid	None	Poor	None
16	80/F	L	Vestibular schwannomas	AICA, PICA	Excellent	None
17	55/M	L	Meningioma	AICA	Good	None
18	61/F	R	Epidermoid	AICA	Excellent	Facial palsy
19	63/M	R	Meningioma	VA, AICA	Excellent	None
20	38/F	L	Meningioma	AICA	Excellent	None
21	29/F	R	Vestibular schwannomas	AICA, PICA	Good	None
22	35/F	L	Epidermoid	AICA	Excellent	None
23	67/M	R	Meningioma	VA, AICA	Good	None
24	45/F	R	Vestibular schwannomas	AICA	Excellent	Hearing disturbance
25	54/F	L	Epidermoid	None	Fair	None
26	48/F	L	Vestibular schwannomas	None	Good	None

F, female; L, left; AICA, anterior inferior cerebellar artery; R, right; M, male; PICA, posterior inferior cerebellar artery; VA, vertebral artery.

frequency (%). Age, sex, side of lesion, and surgical outcomes were compared across groups using the χ^2 tests or unpaired t tests. A P value < 0.05 was considered to indicate significant between-group differences.

RESULTS

The incidence of secondary HFS accounted for 0.83% (26 of 3140) of the total number of HFS in the study, which were caused by 11 meningiomas, 8 epidermoid cysts, and 7 vestibular schwannomas (Tables 1 and 2).

Compared to those with primary HFS, it was not significantly different in the affected side. However, in the secondary HFS group, women were dominant ($P = 0.04$) and the age was a bit younger than those of the primary patients ($P = 0.03$). During the operation, AMR were detected in 19 patients (73.08%) in the secondary HFS group, and in 2996 patients (96.21%) in the primary HFS group ($P < 0.001$) (Table 2).

At 1 day, 7 days, 1 month, 3 months, and 1 year after surgery, the success rate for the secondary HFS group was 80.77%, 80.77%, 84.62%, 84.00%, and 84.00%, respectively. The primary complication had incidence rates of 23.08%, 23.08%, 19.23%, 20.00%, and 12.00%, respectively. The success rates in the primary HFS group were 95.76%, 96.21%, 96.45%, 96.76%, and 96.45%, respectively; the incidence rates of complication were 15.00%, 10.47%, 7.99%, 5.42%, and 3.06%, respectively.

At 1 month, 3 months, and 1 year after surgery, the number of patients who were lost to follow-up of the secondary HFS group were respectively 0 cases, 1 case, and 1 case; of the primary HFS group were respectively 12 cases, 90 cases, and 238 cases.

Compared to the secondary HFS group, success rates in the primary HFS group were significantly higher at 1 day ($P = 0.0009$),

Table 2. Characteristics of Patients in the Secondary and Primary Hemifacial Spasm Groups

Characteristics	Secondary HFS	Primary HFS	*P* Value
Patient, number	26	3114	
Mean age (years)	50.4 ± 14.3	63.5 ± 13.8	0.03
Sex (male:female)	6:20	1366:1748	0.04
Location (right:left)	11:15	1246:1868	0.81
AMR not detected in OP	7 (26.9%)	118 (3.79%)	<0.001

HFS, hemifacial spasm; AMR, abnormal muscle response; OP, operation.

7 days (P = 0.0004), 1 month (P = 0.004), 3 months (P = 0.002), and 1 year (P = 0.003) after the operation (Figure 2).

Compared to the secondary HFS group, complication rates in the primary HFS group were significantly lower at 7 days (P = 0.04), 1 month (P = 0.04), 3 months (P = 0.004), and 1 year (P = 0.02) after surgery; there were no significant differences between groups at 1 day (P = 0.26) (Table 1 and Figure 3).

DISCUSSION

HFS is a hyperactive cranial rhizopathy mostly owing to a vascular compression at the root exit zone of the facial nerve.[10] However, HFS can also be caused by factors other than vascular compression itself. Posterior circulation aneurysms, tumors, vascular malformations, and cysts have been reported to be secondary causes of HFS.[4,11-14] The incidence of tumor-induced HFS is reported as 0.3%–2.5% of all HFSs in the literature.[11] In our study, the incidence of secondary HFS was 26 out of 3140 cases (0.32%). The 26 tumors of secondary HFS included 11 meningiomas, 8 epidermoid cysts, and 7 vestibular schwannomas (Table 1).

The pathogenesis of secondary HFS is still controversial. With respect to tumor, many authors have suggested that it is the vascular compression under the tumor that causes HFS.[11,13] Other experts have suggested that tumors may compress or distort either the root exit zone of the nerve or the facial nucleus.[12,14] In our study, after the tumors were resected, we could find the responsible vessels in up to 76.92% (20 out of 26) of patients. We believe that the cause of secondary HFS begins as the tumor grows and pushes the blood vessels in the tumor, then the displaced blood vessels contact the facial nerve and produce compression of the facial nerve. The 20 tumors of secondary HFS included 10 meningiomas, 3 epidermoid cysts, and 7 vestibular schwannomas. We analyzed the data and found that epidermoid cysts were the main tumors (83.33%) without obvious vessels around the facial nerve after tumor resection. This may be related to the growth pattern of epidermoid cysts.

How can symptoms arise when there is no vascular compression of the facial nerve in patients with secondary HFS? This may

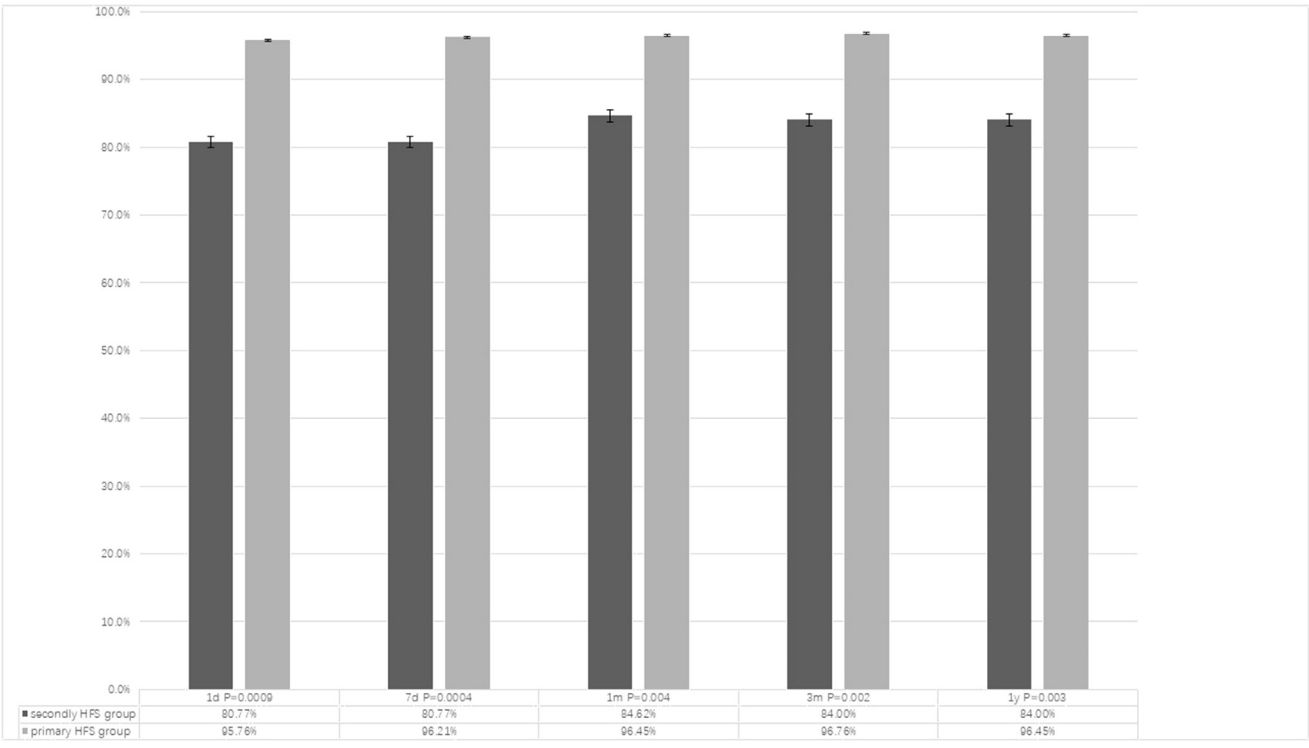

Figure 2. Comparison of the efficiency of the operation between the secondary hemifacial spasm group and the primary hemifacial spasm group. d, day; HFS, hemifacial spasm; m, month; y, year.

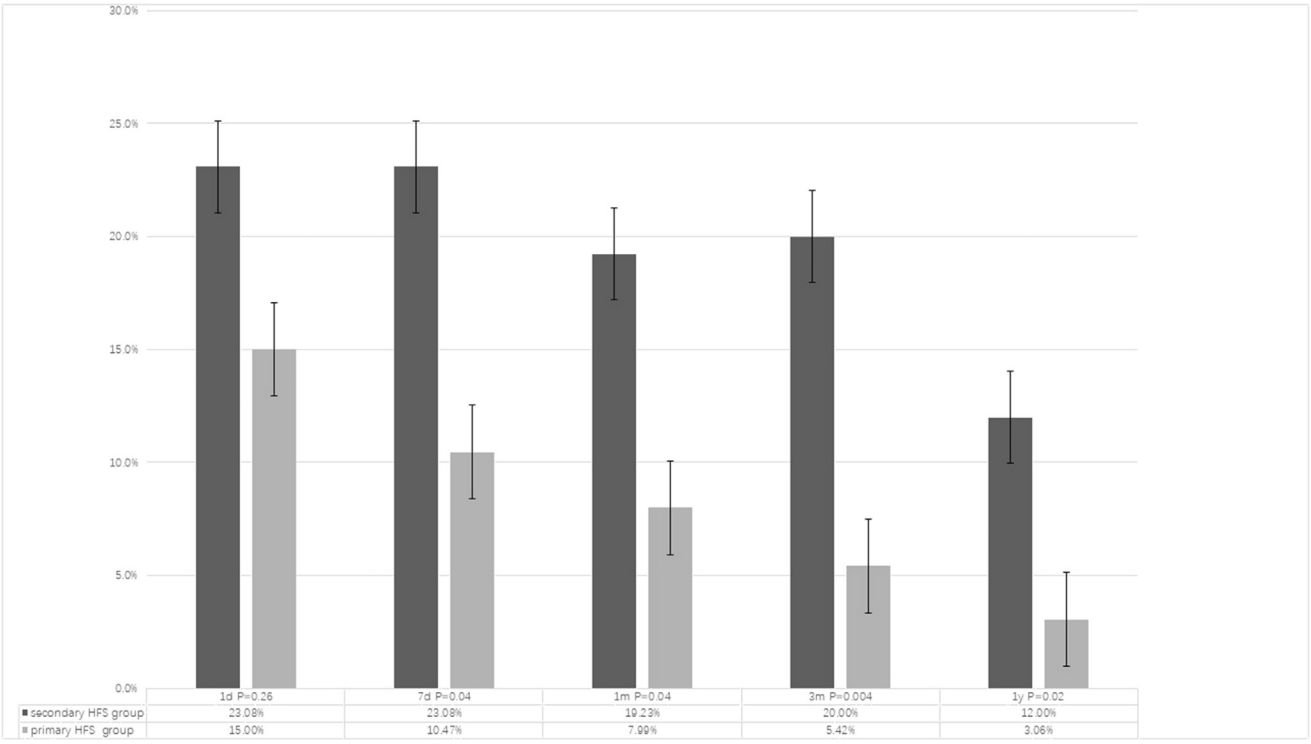

Figure 3. Comparison of the incidence of complication between the secondary hemifacial spasm group and the primary hemifacial spasm group. d, day; HFS, hemifacial spasm; m, month; y, year.

be owing to the presence of tiny blood vessels around the facial nerve that have been damaged during tumor resection. It may also be that vascular compression exists in the V area (the internal auditory canal segment) of the facial nerve, which is difficult to detect, or it may be related to the pathophysiological changes of the facial nerve caused by tumors, which need further analysis.

AMR indicates that there is abnormal conduction of nerve impulse in different branches of the facial nerve. During the operation, AMR were detected in 19 patients (73.08%) in the secondary HFS group and in 2996 patients (96.21%) in the primary HFS group ($P < 0.001$) (Table 2). Of these 7 negative AMR, we found no responsible vessels for compression of the facial nerve during the operation in 5 patients. This also suggests that the occurrence of secondary HFS is related not only to tumor but also to facial nerves compressed by vessels.

Compared to the secondary HFS group, success rates in the primary HFS group were significantly higher after the operation (Figure 2). Unlike primary HFS, secondary HFS is caused by both tumor and vascular factors, and the facial nerve in patients with secondary HFS is often heavily pushed or even wrapped by the tumor. This may also be the cause of facial nerve degeneration and symptoms of HFS, even after tumor resection and MVD of the facial nerve, but the degeneration of the facial nerve is difficult to recover.

Compared to the secondary HFS group, complication rates in the primary HFS group were significantly lower (Table 1 and Figure 3). This is because in the treatment of patients with secondary HFS, it is often necessary to resect the tumor before probing and decompressing the facial nerve, which increases the probability of involvement and injury of brain tissue and cranial nerve around the tumor. Patients with vestibular schwannomas, for example, often have a marked loss of hearing before surgery, and with the removal of the tumor, the operative side of the hearing loss is often an inevitable complication.

To summarize, we suggest exploring the entire facial nerve in patients with secondary HFS after tumor resection. Facial nerve compressed by vessels can be found in most patients during surgery, so tumor resection combined with MVD of the facial nerve may be of value for patients with secondary facial spasm.

CONCLUSIONS

In cases of secondary HFS, facial nerves of most patients were compressed by blood vessels, so MVD after tumor resection plays an important role. We suggest exploring the entire nerve root for possible vascular compression.

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