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CASE REPORT - FUNCTIONAL NEUROSURGERY - MOVEMENT DISORDERS

Synchronous bilateral hemifacial spasm: case-report and literature review

Tao Huang[1] · Nan-xiang Xiong[1] · Peng Fu[1] · Ahmed Abdelmaksoud[1] · Lei Wang[1] · Hong-yang Zhao[1]

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Abstract

Bilateral hemifacial spasm (biHFS) is an infrequent cranial nerve disorder that causes patients to suffer from severe psychological stress, and there are no reported cases of synchronous biHFS. In this study, a 46-year-old right-handed woman was diagnosed with a synchronous biHFS. After one unilateral microvascular decompression (MVD) surgery, the left facial twitching movements relieved immediately, and the right side twitching movements self-relieved the next day. Although there was a delayed hemorrhage, the patient achieved a satisfactory outcome defined as cessation of the twitching movements without recurrence. Based on the present case and related literature, we speculate that anatomical connections between bilateral facial nuclei and hyperactivity of facial nuclei play important roles in the biHFS, and they may, at least in some cases, be the decisive factors regarding the origin, development, and relief of the consequent contralateral spasm.

Keywords Synchronous · Bilateral · Hemifacial spasm · Facial nucleus · Mechanism

Abbreviations

HFS	Hemifacial spasm
biHFS	Bilateral hemifacial spasm
MVD	Microvascular decompression
REZ	Root exit zone
MRI	Magnetic resonance imaging
EMG	Electromyography
AICA	Anterior inferior cerebellar artery
PET	Positron emission tomography
FN	Facial nucleus
TN	Trigeminal nucleus

Introduction

Hemifacial spasm (HFS) is an infrequent cranial nerve disorder characterized by unilateral, involuntary, and irregular clonic or tonic movement of the muscles innervated by the facial nerve [20]. As we know, HFS is not a lethal disease, but it can lead to social embarrassment and be an inconvenience to the patient's lifestyle, which causes patients to suffer from severe psychological stress. According to previous studies, rare cases of bilateral HFS (biHFS) have been reported (Table 1) [2, 4–9, 15, 16, 18, 19], and the estimated incidence of bilateral symptoms is 0.6–3% in HFS patients [19]. In addition, it is generally believed that facial spasms in biHFS patients are asynchronous, even some physicians have defined this as one of the diagnostic criteria [4, 5, 18, 19]. However, in the present study, we described a case of synchronous biHFS that is rarely seen and analyzed in relative articles to explore the underlying mechanism of the symptoms.

Case report

History and presentation

This 46-year-old right-handed woman presented to our center with bilateral hemifacial spasm (biHFS). The condition started when she noted twitching movements in her left eyelid 20 years ago. The twitching movements then gradually spread to involve the lower half of her left face. Ten years later, the right eyelid and then her right lower face began twitching, and

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✉ Nan-xiang Xiong
mozhuoxiong@163.com

[1] Department of Neurosurgery, Union Hospital, Tongji Medical College, Huazhong University of Science and Technology, Jiefang Avenue 1277, Wuhan 430022, Hubei, China

Table 1 Reported bilateral hemifacial spasm cases in the literature

Author, year	Number of cases	Sex	Onset age (year)	Initial site of twitching	Latency (year)	Offending arteries in MRI/A	Main treatment	Operation interval	Operative findings	Complications	Outcome	Recurrence	Commitant
Rosso, 1994 [15]	1	F	43	L eyelids	0.5	N/A	1st: L-MVD 2nd: R-MVD	8 mn	L: AICA, PICA R: AICA	L: transitory facial palsy, mild hearing impairment, and nystagmus R: transitory facial palsy and hearing impairment	L: E R: E	None	None
Kobata, 1998 [8]	2	F	56	N/A	1	N/A	L-MVD R-MVD	N/A	L: AICA R: AICA, PICA	N/A	L: E R: E	None	HT
		F	55	N/A	2	N/A	L-MVD	None	L: PICA	N/A	L: E R: N/A	N/A	HT
Schulze-Bonhage, 1998 [16]	1	M	50–60	L eyelids	N/A	VBA tortuosity	L-MVD	None	L: VBA	L: transitory slight facial palsy, permanent partial hearing loss	L: E R: N/A	None	N/A
Tan, 1999 [19]	5	F	56	L eyelids	11	VBA tortuosity	BTX	None	None	None	L: E R: E	20 wk	N/A
		F	54	L eyelids	1	Normal	BTX	None	None	None	L: E R: E	3 mn	N/A
		F	56	L eyelids	5	N/A	BTX	None	None	None	L: E R: N/A	N/A	N/A
		F	46	L eyelids	20	VBA tortuosity	1st: L-MVD 2nd: BTX	A few months	L: vascular compression	L: permanent mild facial palsy	L: RC R: N/A	6 mn	N/A
		F	63	L eyelids and angle of mouth	0.2	VBA tortuosity	N/A	N/A	N/A	N/A	N/A	N/A	Nasal herpes
Machado, 2003 [9]	1	M	55	R eyelids and angle of mouth	15	VBA tortuosity	BTX	None	N/A	N/A	L: E R: E	N/A	N/A
Tan, 2004 [18]	2	M	72	L eyelids	1	L: AICA R: AICA	BTX	None	N/A	N/A	L: E R: E	N/A	N/A
		F	39	L eyelids	1	L: VBA R: AICA	BTX	None	N/A	N/A	N/A	N/A	N/A
Felicio, 2008 [4]	10	5 M/5F	60.7	5 L and 5 R eyelids	2.8	1 VBA tortuosity	8 BTX	None	None	None	8 E, 2 N/A	4 mn	4 HT
Katz, 2007 [7]	1	F	60	L HFS	3	L: VBA, PICA R: vascular loop	BTX	None	None	None	L: E R: E	N/A	None
Han, 2009 [5]	7	F	39	L eyelids	6	L: PICA R: AICA	L-MVD	None	N/A	N/A	L: E R: E	N/A	N/A
		F	60	L eyelids	10	L: AICA R: AICA	L-MVD	None	N/A	N/A	L: E R: E	N/A	N/A
		F	35	L eyelids	7	L: none R: PICA	R-MVD	None	N/A	N/A	L: E R: E	N/A	N/A
		F	54	L eyelids	7	L: AICA R: AICA	R-MVD	None	N/A	N/A	L: E R: E	N/A	N/A
		F	51	L eyelids	4	VBA tortuosity	L-MVD R-MVD	N/A	L: VBA, PICA R: AICA	N/A	L: E R: E	N/A	N/A
		F	44	L eyelids	8	L: AICA R: none	L-MVD	None	N/A	N/A	L: E R: E	N/A	N/A
		F	69	L eyelids	0.5	L: AICA R: PICA	L-MVD	None	N/A	N/A	L: E R: E	N/A	N/A
Dou, 2016 [2]	10	M	63	R eyelids	3	L: vascular R: vascular	1st: R-MVD 2nd: L-CBZ	1 yr	L: none R: PICA	None	L: G R: E	None	N/A
		F	35	R eyelids	5	L: vascular R: vascular	R-MVD	None	R: AICA	None	L: E R: E	None	N/A
		F	13		2	L: vascular R: vascular	1st: L-MVD 2nd: R-MVD	1 yr	L: AICA R: AICA	None	L: E R: E	None	N/A

Table 1 (continued)

Author, year	Number of cases	Sex	Onset age (year)	Initial site of twitching	Latency (year)	Offending arteries in MRI/A	Main treatment	Operation interval	Operative findings	Complications	Outcome	Recurrence	Commitant
				L eyelids and facial muscle									
		F	57	L eyelids	4	L: vascular R: vascular	1st: L-MVD 2nd: R-MVD	3 mn	L: AICA R: AICA	None	L: E R: E	None	N/A
		F	41	R eyelids	4.4	L: vascular R: vascular	R-MVD	None	R: AICA	None	L: E R: E	None	N/A
		F	65	R eyelids	9	L: vascular R: vascular	1st: R-MVD 2nd: CMZ	N/A	L: N/A R: AICA	None	L: poor R: E	None	N/A
		F	51	L eyelids and facial muscle	1	L: vascular R: vascular	L-MVD	None	L: AICA	None	L: E R: E	None	N/A
		M	72	L eyelids and facial muscle	4	L: vascular R: vascular	1st: L-MVD 2nd: R-MVD	1 yr	L: AICA R: AICA	None	L: E R: E	None	N/A
		F	39	L eyelids	2	L: vascular R: vascular	1st: L-MVD 2nd: R-MVD	9 mn	L: PICA R: AICA	None	L: E R: E	None	N/A
		F	54	L eyelids	3.8	L: vascular R: vascular	1st: L-MVD 2nd: R-MVD	5 mn	L: AICA R: AICA	None	L: E R: E	None	N/A
Huang, 2016 [6]	10	F	55	L	1.5	L: VBA R: AICA	N/A	N/A	N/A	N/A	N/A	N/A	N/A
		F	48	L	0.7	L: PICA R: AICA	N/A	N/A	N/A	N/A	N/A	N/A	N/A
		F	38	L	1	L: PICA R: PICA	N/A	N/A	N/A	N/A	N/A	N/A	N/A
		M	16	L	0.6	L: PICA R: PICA	N/A	N/A	N/A	N/A	N/A	N/A	N/A
		F	16	R	1	L: AICA R: AICA	N/A	N/A	N/A	N/A	N/A	N/A	N/A
		M	37	R	0.8	L: PICA R: VBA	N/A	N/A	N/A	N/A	N/A	N/A	N/A
		F	51	L	1.6	L: AICA R: PICA	N/A	N/A	N/A	N/A	N/A	N/A	N/A
		F	46	L	2.8	L: AICA R: AICA	N/A	N/A	N/A	N/A	N/A	N/A	N/A
		F	39	L	1.4	L: VBA R: PICA	N/A	N/A	N/A	N/A	N/A	N/A	N/A
		F	58	R	0.5	L: AICA R: AICA	N/A	N/A	N/A	N/A	N/A	N/A	N/A
Present	1	F	26	L eyelids	10	L: vascular R: vascular	L-MVD	None	L: AICA	Hematoma	L: E R: E	None	None

F, female; *M*, male; *R*, right; *L*, left; *yr*, year; *mn*, month; *wk*, week; *E*, excellent; *G*, good; *RC*, recurrence; *MVD*, microvascular decompression; *BTX*, botulinum toxin; *CBZ*, carbamazepine; *N/A*, data not available; *NC*, no change; *AICA*, anterior inferior cerebellar artery; *PICA*, posterior inferior cerebellar artery; *VBA*, vertebral and basilar artery; *HT*, hypertension

Literatures without detailed data were excluded

the symptoms became more severe; even during sleep, the facial muscles still twitched. Moreover, the spasms were intensified by stress and anxiety. She denied arm tremor or any relevant family history. There was no history of facial pain or weakness, facial or head trauma, prior exposure to neuroleptics, or other movement disorders. Clinical examination revealed a synchronous, involuntary, tonic-clonic muscle contractions involving bilateral facial musculature including the frontalis, corrugators, orbicularis oculi, zygomatic, and perioral muscles. The mentalis and platysma muscles were also involved. Specifically, the contractions were synchronous but not completely symmetric. Magnetic resonance imaging (MRI) scan showed direct innate contact between the root exit zone (REZ) part of the facial nerve and the adjacent small vessels on the left side, but it was not obvious on the right side (Fig. 1). Facial electromyography (EMG) revealed the burst discharges in different bilateral facial muscles are almost synchronous at various points in time (Fig. 2).

Surgical procedure

The procedure was performed according to technical notes described before in previous literature [11]. Described briefly, under general anesthesia, the patient was placed in the right lateral decubitus position with the head rotated about 10° away from the affected site, and the vertex was dropped 15° toward the floor. The procedure was performed via keyhole lateral retrosigmoid suboccipital approach. After the cerebellopontine angle cisterns were fenestrated, the arachnoid membrane around the lower cranial nerves was dissected. Retracting the flocculus cerebelli gently with an aspirator, the left anterior inferior cerebellar artery (AICA) was identified as the compressing vessel near the REZ. The operator isolated the AICA away from the REZ and placed a piece of a correctly sized Teflon sponge into the space between the AICA and the medulla oblongata to push the AICA away from the facial nerve. Space was maintained between the Teflon sponges and the REZ. Finally, the layers from the dura mater to the skin were gradually sutured layer by layer.

Clinical outcome

After the procedure, this patient reported an immediate relief of the left facial twitching movement, and the facial spasm on the right side resolved the next day too. No intracranial hematoma or pneumatosis was discovered in the routine postoperative CT scan. After 5 days of medical treatment, the patient recovered and was discharged from the hospital. Unfortunately, the patient complained of a mild headache at the outpatient department in the third-week post-operation, and her brain CT scan indicated approximately 10 ml in size located the exterior cerebellar lobe and the bone defect site. Because the mass effect and patient's symptom were not severe, we selected a conservative treatment for management. After inpatient rehabilitation for 2 weeks, her symptoms improved, and the CT imaging showed approximately 90% of

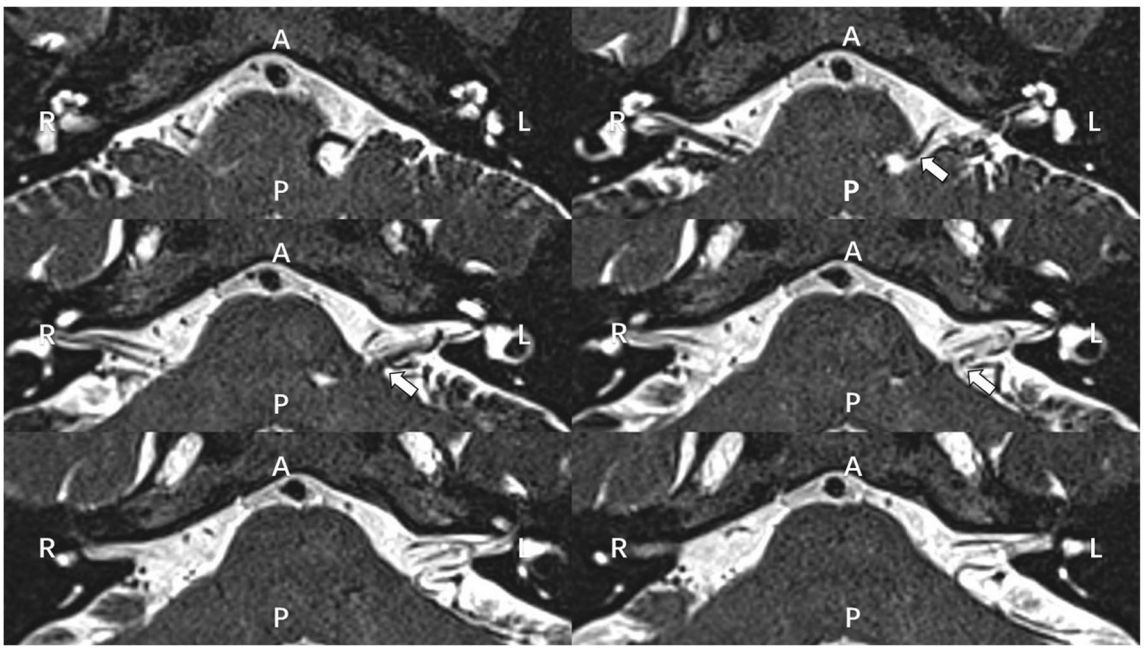

Fig. 1 Preoperative serial axial MRI imaging of the posterior cranial fossa. Axial images of 3D-CISS sequence (TR 12, TE 6, FA 70, 2 NEX, 282 × 512 matrix, 0.5 mm partitions, 56 slices) clearly revealed bilateral facial and vestibulocochlear nerves with excellent CSF-nerve contrast. The vessels around the nerves appeared to have lower signal intensities. It is clearly delineated that close contact is between the REZ of the facial nerve and adjacent tortuous AICA (white arrow) on the left side, whereas neurovascular compression is not apparent on the right side. *A*, anterior; *P*, posterior; *L*, left; *R*, right

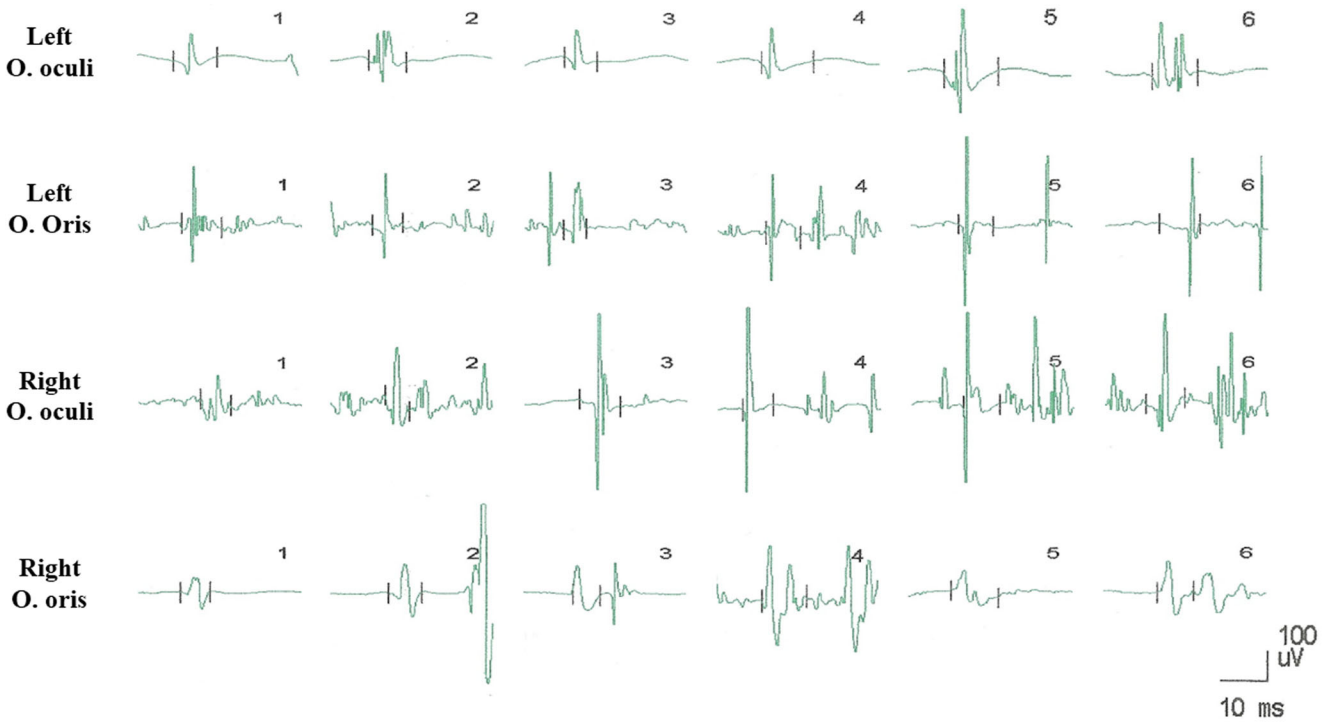

Fig. 2 Facial electromyography (EMG) of various muscles. It revealed that bursts discharged in different bilateral facial muscles are almost synchronous at various points, especially in the left orbicularis oris (O. Oris), orbicularis oculi (O. Oculi), and the right O. Oris. Importantly, in the fifth segment, four facial muscles were totally synchronous

the hematoma was absorbed. During 2 years of follow-up, no recurrence of an abnormal facial movement or a headache was detected.

Literature reviews

The PubMed online database was searched for publications related to patients who have suffered biHFS. Search terms included bilateral hemifacial spasm and bilateral HFS. When different terms were found, these were searched further to find more studies. Furthermore, references for all search selected articles were also reviewed for potential cases. The following types of studies were excluded: literature reviews, repeated cases, articles not available in full text, and articles with incomplete clinical information. The following information, if reported on, was collected from eligible articles: (1) sex, (2) onset age, (3) clinical presentations, (4) imaging description, (5) treatment, (6) surgical findings, (7) outcome, and (8) complications.

A summary of the medical literature available can be seen in Table 1 [2, 4–9, 15, 16, 18, 19]. The study sample comprised 51 patients with biHFS, 12 men and 39 women, having a mean ± SD ages at onset of 48.6 ± 14.1 years, and having a mean ± SD symptom latency between two sides of 4.1 ± 4.3 years. Except for the present case, all the patients were classified as asynchronous bilateral HFS. The ratio of the left/right side of onset was 36/13 (data was not available in two cases). As to the initial site of onset, eyelids were the initial site in 33 cases (86.8%), while other five patients' onset location were multiple muscles. Patients with bilateral HFS and the total number of patients with HFS had similar mean ± SD ages at onset (48.6 ± 14.1 vs 46.6 ± 11.5 years); however, biHFS patients have a higher female-male ratio of the total number of patients with HFS (39:12 vs 1.8:1) [21]. Vascular loop compressing or an abnormality was found in 46 (97.9%) patients. As proved by the intraoperative observations described, the offending vessels were AICA (17, 54.8%), artery of uncertain origin (7, 22.6%), posterior inferior cerebellar artery (3, 9.7%), multiple arteries (3, 9.7%), and vertebral and basilar artery (1, 3.2%). Among patients without improvement of the contralateral symptoms, most of them underwent another MVD on the contralateral side within 1 year, and none of them received a second operation due to the failure of the first surgery. All of the differences need to be researched further, which will help us to understand the mechanism of HFS.

Discussion

Bilateral hemifacial spasm (biHFS) cases are rarely reported due to their low morbidity, and thus the mechanism of the disease has not been clearly clarified. Additionally, lack of curative experience and theoretical research causes difficulty

of neurosurgeons in confirming the diagnosis and selecting an effective therapeutic strategy [19]. In the present case, the common clinical characteristics included unilateral onset and bilateral synchronous contractions of the facial muscles, and this pattern was confirmed electrophysiologically by the facial EMG (Fig. 2). The rudimentary mechanism is a vascular compression of the left facial nerve, which was supported by MR scan findings (Fig. 1) and intraoperative observation.

In this case, several similar diseases need to be distinguished from biHFS. Blepharospasm is a rare disorder that consists of bilateral, usually symmetric and synchronous contractions of the orbicularis oculi [19], which is easily confused and therefore difficult to differentiate due to the synchronous bilateralism. However, the simultaneous nature of the bilateral contractions is the decisive diagnostic criterion in blepharospasm patients [19]. Thus, the long latency between the right and left sides in the present case, 10 years, helps us to exclude the diagnosis of blepharospasm. In addition, based on affected muscles and past history, it is easy to exclude some common differential diagnoses, including oromandibular dystonia, facial tics, hemimasticatory spasm, and facial myokymia. Above all, a combination of clinical features and auxiliary examination results, especially synchronous bust discharges in the EMG (Fig. 2), in the diagnosis of synchronous bilateral HFS is most reliable.

In the present study, we consider that the phenomenon of synchronous bilateral spasms is plausible, although it is not consistent with the intrinsic experience of most neurosurgeons [4, 5, 18, 19]. It is widely accepted that the main cause of HFS is the compression of the offending artery on the facial nerve at the REZ, and MRI scan findings (Fig. 1) are consistent with this finding. Additionally, both chronic vascular conflict and facial movements could enhance excitability of the facial nerve neurons, including the facial nucleus [3, 12, 13], and many researchers believe that hyperactivity of facial nucleus also plays an important role in the occurrence of HFS [12, 22]. Some studies have been indicated that there were anatomical connections between bilateral facial nuclei, but whether trigeminal nuclei is involved is not clear [1, 10, 14]. Furthermore, Shimizu et al. using positron emission tomography (PET) proved that a functional network existed between bilateral thalami in HFS patients, in particular, bilateral hyperactive status was synchronous and symmetric [17]. Therefore, we speculate that a single vascular compression can stimulate the facial nerve and antidromically activate the bilateral facial nuclei through the anatomical connections between them, then synchronous bilateral facial spasms can be induced due to hyperactivity of bilateral facial nuclei. Associated with the presentation of synchronous bilateral facial contractions and synchronous burst discharges of different muscles in the facial EMG (Fig. 2), we have reasons to believe that the emergence of synchronous bilateral spasms, in at least some cases, is possible.

In this patient, only one unilateral MVD surgery was carried out on the more severely affected side, and contralateral symptoms were relieved afterward. Similar cases were reported on in other studies [2, 5]. There are several possible explanations for this situation. Firstly, the hyperactivity of bilateral facial nuclei could be caused by an antidromic afferent input from the facial nerve [17, 20]. With the completion of the surgery, stimulation from the offending artery stopped and the hyperactivity of the contralateral nucleus decreased or stopped then the symptoms. Secondly, the facial skin or muscle undergoing contraction could have initiated and conducted an afferent volley via the trigeminal nerve to the bilateral facial nuclei and enhanced their excitability [1, 10, 12, 14, 22]. With the disappearance of movements on the operated side, the afferent signal could not sustain hyperactivity of the facial nuclei, and the contralateral symptoms ceased. Thirdly, there might have been bilateral offending arteries. With the ipsilateral artery removed in the operation, the contralateral artery could have been stretched by the arachnoid or the vertebrobasilar arteries away from the facial nerves [2, 5]. However, in our opinion, we do not think that the third hypothesis is applicable to some cases, because if it is correct, bilateral contractions should have ceased simultaneously and immediately after the surgery. Nevertheless, some patients experience the delayed contralateral relief [2], and this may be due to attenuation of the facial nucleus hyperactivity. In summary, we are inclined to the hypothesis of hyperactivity of the facial nuclei, and we suggest that this may be the decisive factor regarding the origin, the development, and the alleviation of the consequent contralateral spasm.

Although we tried to complete our research as perfectly as we can, this study still has a number of limitations. Firstly, the rarity of bilateral HFS cases makes reporting of large case studies difficult. Secondly, due to a lack of preparation in collecting perioperative data, we cannot present more concise records, such as video material. Thirdly, because of economic factors, we cannot use some more precise and advanced equipment to confirm our diagnoses, such as PET and high-field MRI. The research will be of a higher standard and the findings of a higher value if these limitations could be overcome.

Conclusion

We reported a patient with synchronous biHFS who experienced one unilateral MVD surgery and obtained bilateral symptoms relieved successfully. We speculate the fact that the connections between the bilateral facial nuclei are an important cause of synchronicity. Additionally, we present the hypothesis that hyperactivity of the facial nuclei may be the decisive factor regarding the origin, development, and relief of the consequent contralateral spasm, at least in some cases.

Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

Ethical approval All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards.

Informed consent Informed consent was obtained from the participant included in the study.

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