Turk Neurosurg 34(3):429-434, 2024

Original Investigation

DOI: 10.5137/1019-5149.JTN.42249-22.3

Received: 10.09.2022
Accepted: 25.03.2023

Published Online: 16.04.2024

Microvascular Decompression for Hemifacial Spasm without the Use of Neuromonitoring and Fix Retraction: A Single-Center Experience

Serhat PUSAT[1], Efdal ERDOGAN[2], Murat ATAR[1], Yahya Cem ERBAS[2], Ersin ERDOGAN[2]

[1]University of Health Sciences Sultan Abdülhamid Han Training and Research Hospital, Department of Neurosurgery, Istanbul, Türkiye
[2]Neurospine Academy, Private Center, Ankara, Türkiye

Corresponding author: Serhat PUSAT ✉ pusatserhat@yahoo.com

ABSTRACT

AIM: To evaluate outcome of patients with hemifacial spasm surgically treated by microvascular decompression without the use of neuromonitoring and fix retraction.

MATERIAL and METHODS: Of the 78 patients with hemifacial spasm operated by the senior author of this study between 2016 and 2020, 60 patients who were followed up were included and retrospectively investigated. The female:male ratio was 32:28, and the mean age was 42.5 ± 11.5 years (range 23–71 years). All the patients were operated via the lateral retrosigmoid suboccipital infrafloccular approach, and no permanent retractor was employed during the procedures. Additionally, intraoperative neuromonitoring was not performed for any of the patients.

RESULTS: Single vessel compression was observed in 77% of the patients, whereas 16% and 6% experienced compression in two and three vessels, respectively. Immediate recovery was achieved in 70% of the patients. Half of the remaining 30% fully recovered within 6 months–2 years. Furthermore, the complaints of 5% of the patients were alleviated, whereas no improvement was observed in 10% of the patients. One patient developed a recurrence.

CONCLUSION: Microvascular decompression is a highly effective method for the treatment of hemifacial spasm. In this study, we did not resort to intraoperative neuromonitoring and observed that our postoperative complication rates were consistent with the existing literature.

KEYWORDS: Hemifacial, Neuromonitoring, Decompression, Retraction, Vessel

ABBREVIATIONS: HFS: Hemifacial spasm, **3D-TOF:** Three-dimensional time-of-flight, **CISS:** Constrictive interference at a steady state, **MRI:** Magnetic resonance imaging, **REZ:** Root entry zone, **BoNT:** Botulinum neurotoxin, **MVD:** Microvascular decompression, **CSF:** Cerebrospinal fluid, **PICA:** Posterior inferior cerebellar artery, **AICA:** Anterior inferior cerebellar artery, **AMR:** Abnormal muscle response

Serhat PUSAT : 0000-0003-2412-2320
Efdal ERDOGAN : 0000-0002-6656-0989
Murat ATAR : 0000-0001-6047-2956
Yahya Cem ERBAS : 0000-0001-7719-9271
Ersin ERDOGAN : 0000-0002-3401-940X

INTRODUCTION

Microvascular decompression as a surgical procedure for hemifacial spasm (HFS) was initially proposed by Gardner and later performed by Cook and Jannetta (3,10). HFS is characterized by unilateral, involuntary, irregular, clonic, or tonic contractions of the facial muscles innervated by the facial nerve. HFS initially presents with contractions in the orbicularis oculi, which may spread to the frontal region and platysma over time (18,25). Upon the ipsilateral brow lift, the eyelid on the same side closes and is referred to as the Babinski-2 sign, a characteristic of typical HFS cases. Atypical HFS, which is less frequent, involves spasms that originate in the buccal muscles and spread upward. Moreover, in atypical HFS, vascular pressure occurred dorsally and not caudally. There was no significant difference observed in age, sex, and HFS sides between the typical and atypical HFS groups. There were cases where contractions intensified because of changes in the head position and anxiety. HFS is mostly unilateral and may be bilateral in 0.5%–6% of the cases at a rate of 9.8 per 100.000 individuals (16). Hereditary HFS cases represent 2.4% of all cases (2). HFS is more prevalent in women than in men. It is not a life-threatening disorder, but the contractions may lead to social isolation, deterioration in work performance, and depression in advanced cases (20). In severe cases, spasms may cause complete closure of the eyes, leading to impairment in 3D vision with the possibility of traffic accidents while driving. Moreover, in severe cases, the spasms can cause sleep disturbance and chronic fatigue, particularly when they occur during sleep. The diagnosis of HFS is based on conventional imaging, anamnesis, and physical examination. Three-dimensional time-of-flight (3D-TOF), 3D-constructive interference at a steady state (CISS), magnetic resonance imaging (MRI), and fast imaging employing steady state acquisition were simultaneously conducted in patients. MRI allows physicians to identify blood vessels that exhibit abnormal behavior. In-depth examination of these images can expose very small vessels that might compress the root entry zone (REZ), thereby reducing the likelihood of recurrence (15,20). Currently, HFS usually develops as a result of the pathologies in the REZ of the facial nerve. Upon examination, the etiological causes are classified as primary and secondary factors. The primary factors include primary compression of adjacent vessels. The secondary causes are a result of the damage to the facial nerve at any location from the internal auditory meatus to the stylomastoid foramen. Common secondary causes include pontocerebellar angle tumors, vascular malformations, and demyelinating brain diseases. In secondary HFS, surgery is not beneficial and should not be performed. Primary HFS often starts from abnormal contractions in the orbicularis oculi muscle, whereas secondary HFS presents with abnormal contractions in the lower facial and platysma muscles (2,6,9). Differential diagnoses include blepharospasm, oromandibular dystonia, facial tics, myokymia, and psychogenic contractions. HFS is also common in individuals with Charcot–Marie–Tooth disease and multiple sclerosis. If the symptoms are moderate, medical treatment may be considered, including anticonvulsants (carbamazepine and benzodiazepine), anticholinergics, antipsychotics, and botulinum neurotoxin (BoNT). BoNT injection is the most commonly used treatment method, and the response to treatment is highly variable (5). Microvascular decompression (MVD) is a surgical procedure performed in cases that do not respond to medical treatment. MVD is functional surgery. The surgical treatment should not damage the nerve but provide long-lasting relief of symptoms (22). MVD for HFS is performed using the lateral retrosigmoid suboccipital infrafloccular approach (7,11,18,19).

MATERIAL and METHODS

The study was approved by the Health Sciences University, Hamidiye Scientific Research Ethics Committee (Decision date: 29.03.2024, No:4/16)

Patients

Between 2016 and 2022, the senior author of this study performed surgery on 78 patients with HFS. Sixty patients who had follow-up were included and retrospectively investigated. Moreover, 32 patients were female, and 28 were male with a mean age of 42.5 ± 11.5 years (range 23–71 years). Twenty-six patients had complaints related to the left facial side, whereas 34 had complaints related to the right facial side. The spasm duration varied between 1.2 and 13 years, with an average of 3.4 years. Patient information, such as the presence of vascular pathologies, disease duration, frequency of attacks, and previous treatments were recorded preoperatively. The patients underwent T2 MRI, CISS-sequence MRI, and 3D-TOF MRI preoperatively to determine the presence of abnormal vascular structures. The bone structure of the posterior fossa and the structure of the mastoid cells were examined via thin-section computed tomography. The patients were not administered dexamethasone preoperatively or postoperatively.

Surgery

All patients underwent surgery using the lateral retrosigmoid suboccipital infrafloccular approach. The patients were placed in two different positions: 1) the classical lateral decubitus position, in which the head slightly faces the opposite side of the lesion and fixed in a three-pin head-holder, the chin approximately two fingers from the sternum, and 2) the supine position, where the head is turned to the opposite side of the lesion by a positional angle. The value of the positional angle is calculated by measuring the angle between the line drawn from the two petrous bones to the internal auditory canal and the line drawn from the clivus dorsum (8,21,23). Before making the incision, the mastoid eminence and digastric groove were identified by palpation. A 5–6 cm curvilinear skin incision was created through the subcutaneous tissue and made parallel to the hairline from the retroauricular region with the upper border of the zygomatic arch projection. Then, after musculo-aponevrotic dissection, the cranial asterion, mastoid emissary vein, mastoid foramen, and mastoid incisura should be distinguished in the posterior fossa surgeries. In the infrafloccular approach, craniectomy involving the mastoid foramen is appropriate (Figure 1); an extension of the craniectomy up to the asterion junction is not required. Craniectomy with a diameter of ~3 cm was performed in all

patients with HFS. First, the dura mater was opened from a small point, and the cerebrospinal fluid (CSF) was released until the craniectomy was performed, facilitating cerebellar retraction. In most cases, the mastoid cells are opened, ıf so, they should be tightly occluded. Craniotomy was not performed on any patient because of its ineffectiveness as a result of a possible transverse and sigmoid sinus injury. Additionally, the surgical exposure would be incomplete, thereby complicating the procedure. After the dura mater was opened, the lateral medullary cistern was opened to release the CSF. In effectively performed craniectomies, exposure to the cranial nerve V is not required. The route used in MVD for HFS is the lateral retrosigmoid suboccipital infrafloccular route (Figure 2A). The infrafloccular route allows visualization of the supraolivary fossette. In the region posteroinferior to supraolivary fossette, the REZ of the cranial nerve VII is observed (Figure 2B). The cranial nerve complex, IX-X-XI, is initially distinguished in MVD (Figure 2C). In this region, the choroid plexus protrudes

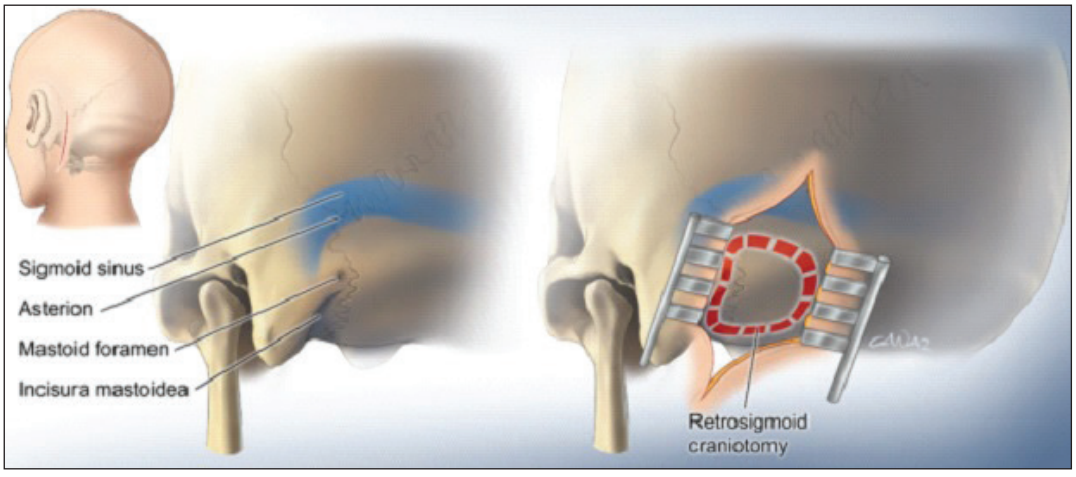

Figure 1: Retrosigmoid approach.

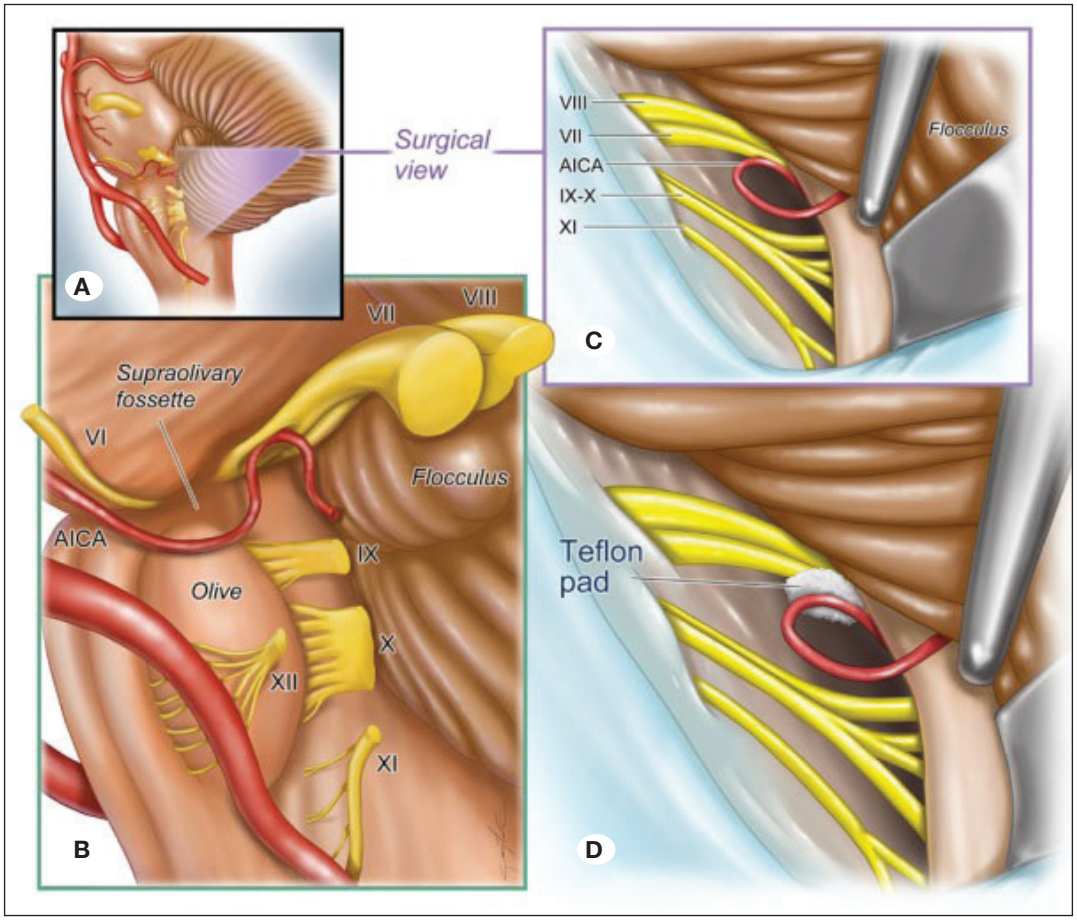

Figure 2: Operative nuance.

from the foramen of Luschka and is an extremely important anatomical landmark. After the flocculus and choroid plexus are elevated and the pia-arachnoid filaments of the cistern divided, the pontomedullary fissure is reached with the REZ of the eight complex posteriorly, the ventrocaudal aspect of the facial REZ together with the adjacent brainstem anteriorly, and flocculus was superiorly retracted to expose the REZ of the cranial nerve VII and compressing artery (often the posterior inferior cerebellar artery [PICA] or anterior inferior cerebellar artery [AICA]) 1–3 mm above the cranial nerve IX (23). Subsequently, cranial nerve VII was detached from the surrounding tissue. Some arachnoid dissection of the cerebello-medullary fissure is particularly important in arterial mobilization. Here the pulsatile vessel contact on the REZ should be examined right from the point where it exits the brain stem. During decompression, the small feeding arteries coursing at the level of the nerve complex VII–VIII on the AICA observed carefully. These arteries are the internal auditory artery, subarcuate artery, meatal loop, and recurrent perforating arteries, and their course around the internal auditory canal makes decompression difficult (12,17,18). Following the completion of this surgical step, the dissection of neural structures and manipulation of vessels require an adapted microsurgical instrument that utilizes Teflon as a synthetic barrier between the nerve and vessel (Figure 2D). The inserted material used to maintain the offending vessel(s) away must not be neo-compressive. To prevent the migration of the Teflon barrier, some surgical tissue adhesive was used at this stage. The correct use of the Teflon barrier is important to prevent any potential hearing loss. As we did not employ neuromonitoring or examine the lateral spread response, we stopped placing the Teflon barriers once we confirmed the surgical separation of the nerve was complete. The purpose of using neuromonitoring in HFS microvascular surgery is to reveal abnormal muscle response (AMR). It is important to demonstrate the disappearance of EMG-guided AMR during MVD surgery in HFS, and it has been reported in many studies (11,12). Intraoperative EMG was not performed in this group of patients due to lack of adequate technical support. The characteristic of this group is that MVD is performed only by the observation of the surgeon. Intraoperative EMG has been performed to indicate AMR for the last 7 months (4,27). To preserve hearing, we focused on clearly revealing the lower cranial nerves, Most importantly, the permanent retraction was not performed on any patient during the surgery, making a sharp arachnoid dissection without opening the arachnoid above cranial nerve VIII. After watertight dural closure was achieved, the surgery was completed by performing *in situ* cranioplasty without suturing the bone tissue (17).

RESULTS

The right side was affected in 34 (57%) of 60 patients who were investigated (Table I). AICA and PICA were the most common compressing arteries. Facial paralysis was not observed in any patient postoperatively. Three patients developed partial hearing loss but had their serviceable hearing capacity preserved. Furthermore, a wound closure problem was observed in three patients, and no patient was detected with rhinorrhea (Table II).

Single vessel compression was noted in 46 (77%) patients. Additionally, 20 (33%) patients had AICA compression, 18 (30%) had PICA compression, four (8%) had vertebral artery compression, and four (8%) had basilar artery compression. Ten (16%) patients had compression in two vessels and four (6%) had compression in three vessels (Table III).

There were immediate improvements in 42 (70%) patients. Of the remaining 18 patients (30%), nine (15%) completely recovered within 6 months–2 years. The complaints of three patients (5%) were alleviated over time. There were no improvements in six (10%) patients. One patient with relapse was followed-up for 1 year postoperatively and will undergo reoperation since no improvement has been observed (Table IV). Concerning postoperative complications that developed in the study group, one patient had partial facial paralysis (House–Brackmann Grade 2), and three patients had partial hearing loss. Wound site-related problems were observed in three patients.

DISCUSSION

HFS is a condition characterized by uncontrolled and continuous muscle contractions that can cause social isolation over time. This disease is not known to be hereditary. Symptoms of HFS usually appear around the age of 40 years, and majority of the patients in this study were women. Treatment modalities for HFS include medical treatment, and in cases that do not respond to medical treatment, surgical intervention is considered. The typical approach to medical treatment involves administering carbamazepine, benzodiazepine, and BoNT. However, despite being a popular option nowadays,

Table I: Patient Population

	Female Patients	Male Patients	Total
No. of Cases	32	28	60
Mean Age (years)	47.12	44.66	45.89
Age Range (years)	25-65	26-71	23-71
No. on Left Side	16	10	26
No. on Right Side	15	19	34
Mean Duration of Syptoms (years)	3.4	4.8	40

Table II: Operative Findings

Vessel	Patients (n)
AICA	18
PICA	18
VA	4
LA	2
BA	4
VA+PICA	4
VA+AICA	2
PICA+AICA	4
VA+AICA+PICA	4

AICA: *Anterior inferior cerebellar artery,* ***PICA:*** *Posterior inferior cerebellar artery,* ***LA:*** *Labyrinthine artery,* ***BA:*** *Basiler artery,* ***VA:*** *Vertebral artery.*

Table III: Postoperative Complications (60 Cases)

Symptoms	At Discharge	Follow-up
Deafness	3	1
Facial Weakness	2	0
Cerebrospinal Fluid Leak	3	0

Table IV: Results (60 Cases)

	No. of Cases		
	At Discharge	Follow-up (6 mo-2 years)	Follow-up (3 years)
Spasm- free	42 (70%)	51 (85%)	54 (90%)
Further spasm	18 (30%)	9 (15%)	6 (10%)
Recurrence		1	1

these treatments have proven to be ineffective. BoNT acts by blocking calcium-mediated acetylcholine release at the synaptic junction. Blurred vision, diplopia, and ptosis may develop after BoNT use, but these complications improve within days. Studies reported alleviation of up to 85% of the symptoms because of BoNT use. Repeated injections may lead to atrophy of the target muscles and result in facial asymmetry. Furthermore, doses should be gradually increased in each injection to obtain the same efficiency of treatment with repeated injections. The side effects that may develop due to BoNT use are paralysis of the orbicularis oculi muscle, ptosis, and lagophthalmos. Studies also reported that anticonvulsants and GABAergic agents may also be used as alternatives to BoNT (7,16). The success rate of treatment with BoNT is between 76% and 95%, and the symptom-free period differs (16,20). Surgical treatment is the best method to achieve long-term symptom-free results. The retrosigmoid suboccipital infrafloccular approach is used for surgical treatment. The most important aspect of the surgery is craniectomy and should be performed with complete efficiency. Craniectomy is performed until the sinus is visible, and upon initiation of craniectomy, after the first burr hole is opened, the dura mater is opened with a small incision to drain the CSF and relax the cerebellum. It is important not to use retraction during surgery to minimize possible auditory and brainstem complications. Cranial nerve VII is frequently compressed by AICA or PICA. After the arteries and nerves are detached, a Teflon barrier is placed between them (Figure 3). In this study, the severity of vascular compression and a symptom-free postoperative period were not correlated. Additionally, the disappearance of the lateral spread response during neuromonitoring was not associated with the symptom-free postoperative period. This finding supports our method where surgery was performed without using a neuromonitor (9,24). In a meta-analysis that evaluated the outcomes of MVD surgeries for HFS, the majority of patients (91%) remained asymptomatic during a mean follow-up period of 2.9 years (18). In a study conducted by Samii et al., the researchers reported no variance in the rates of asymptomatic cases during a mean follow-up period of 9 years (22). In the literature, the symptom-free period had no statistical correlation with operative time, age, sex, spasm side, and spasm frequency (1,13). The recurrence rate after MVD was 2.4%, whereas the reoperation rate was 1.2% in the group of patients who underwent surgery (18,27).

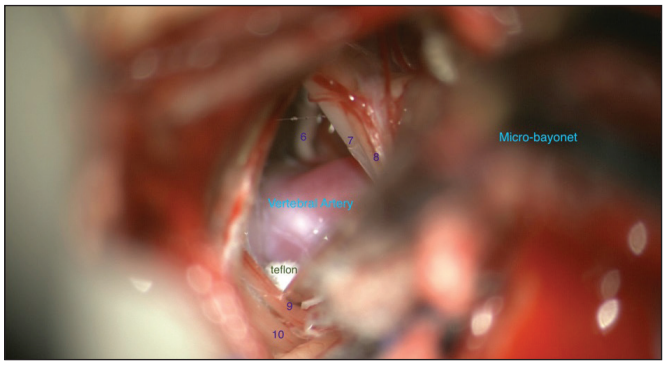

Figure 3: Intraoperative imaging.

If symptoms recur after a long symptom-free period, the patients should be regularly monitored. In HFS, the symptoms are sometimes temporary and may improve. Sometimes, there is compression of an abnormal vessel that cannot be observed during surgery, or the Teflon barrier migrates. The complications of MVD surgeries for HFS treatment include temporary or permanent facial nerve palsy (9.3%), hearing loss (3.2%), ischemia (0.1%), occipital numbness, intracranial hemorrhage, epidural hemorrhage, meningitis, abducens nerve palsy, wound site infection, and CSF leakage (11,13,18,20,24,26). The incidence rate of serious complications after MVD was <1% (18,27). Through this study, we intend to emphasize that we did not utilize intraoperative neuromonitoring and none of our patients experienced total auditory loss (14). For experienced surgeons performing posterior fossa surgery, MVD is an effective treatment method and has a high success rate (16).

CONCLUSION

It should be kept in mind that repetitive medical treatments increase the cost of HFS treatment. Surgeries performed by competent and experienced surgeons are usually highly cost-effective and safe. Permanent complications are extremely rare. Therefore, surgical treatment should be the treatment of choice for patients with symptomatic HFS.

AUTHORSHIP CONTRIBUTION

Study conception and design: SP
Data collection: EfE
Analysis and interpretation of results: YCE, MA
Draft manuscript preparation: GC
Critical revision of the article: ErE
Other (study supervision, fundings, materials, etc...): ErE
All authors (SP, EfE, MA, YCE, ErE) reviewed the results and approved the final version of the manuscript.

REFERENCES

1. Barker FG 2nd, Janetta PJ, Bıssonette DJ, Shields PT, Larkins MV, Jho HD: Microvascular decompression for hemifacial spasm. J Neurosurg 82:201-210, 1995
2. Colosimo C, Bologna M, Lamberti S, Avanzino L, Marinelli L, Fabbrini G, Abbruzzese G, Defazio G, Berardelli A: A comparative study of primary and secondary hemifacial spasm. Arch Neurol 63:441-444, 2006
3. Cook BR, Jannetta PJ: Tic convulsif: Results in 11 cases treated with microvascular decompression of the fifth and seventh cranial nerves. J Neurosurg 61:949-951, 1984
4. Dannenbaum M, Lega BC, Suki D, Harper RL, Yoshor D: Microvascular decompression for hemifacial spasm: Long-term results from 114 operations performed without neurophysiological monitoring. J Neurosurg 109:410-415, 2008
5. Defazio G, Abbruzzese G, Girlanda P, Vacca L, Curro A, Salvia R, Marchese R, Raineri R, Roselli F, Livrea P, Berardalli A: Botulinum toxin A treatment for primary hemifacial spasm: A 10-year multicenter study. Arch Neurol 59:418-420, 2002
6. Dou NN, Zhong, J, Zhou QM, Zhu J, Wang YN, Xia L, Yang XS, Ying TT, Zheng XS, Li ST: The mechanism of hemifacial spasm: A new understanding of the offending artery. J Neurol Res 37:184-188, 2015
7. Erbas YC, Pusat S, Atac GK, Erdogan E: In-situ cranioplasty after microvascular decompression: A technical note. Turk Neurosurg 27:479-481, 2017
8. Erdogan E, Civelek E, Onal MB, Solmaz I, Kural C, Yakupoğlu H: A new method of patient's head positioning in suboccipital retrosigmoid approach. Neurol India 57:777-779, 2009
9. Fukuda H, Ishikawa M, Okumura R: Demonstration of neurovascular compression in trigeminal neuralgia and hemifacial spasm with magnetic resonance imaging: Comparison with surgical findings in 60 consecutive cases. Surg Neurol 59:93-99, 2003
10. Gardner WJ: Concerning the mechanism of trigeminal neuralgia and hemifacial spasm. J Neurosurg 19:947-958, 1962
11. Gardner WJ, Miklos MV: Response of trigeminal neuralgia to decompression of sensory root: Discussion of the cause of trigeminal neuralgia. J Am Med Assoc 170:1773-1776, 1959
12. Hatayama T, Kono T, Harada Y, Yamashita K, Utsunamiya T, Hayashi M, Nakajima H, Hataraka R, Shimado D, Takemura A, Tabata H, Tobishima H: Indications and timings of re-operation for residual or recurrent hemifacial spasm after microvascular decompression: Personal experience and literature review. Neurol Med Chir (Tokyo) 55:663-668, 2015
13. Holste K, Sahyouni R, Teton Z, Chan YA, Englot DJ, Rolston JD: Spasm freedom following mıcrovascular decompression for hemifacial spasm: Systematıc review and Meta-Analysis. World Neurosurg 139:383-390, 2020
14. Iijima K, Horiguchi K, Yoshimoto Y: Microvascular decompression of the root emerging zone for hemifacial spasm: Evaluation by fusion magnetic resonance imaging and technical considerations. Acta Neurochir (Wien) 155:855-862, 2013
15. Jia JM, Guo H, Huo WJ, Hu SW, He F, Sun XD, Lin GJ: Preoperative evaluation of patients with hemifacial spasm by three-dimensional time-of-flight (3D-TOF) and three-dimensional constructive interference in steady state (3D-CISS) sequence. Clin Neuroradiol 26:431-438, 2016
16. Lu AY, Yeung JT, Gerrard JL, Michaelides EM, Sekula RF Jr, Bulsara KR: Hemifacial spasm and neurovascular compression. Scientific World J 6:1-7, 2014
17. Matsushima T, Hitotsumatsu T, Inamura T, Natori Y, Inoue T, Fukui M: Pitfalls associated with MVD for hemifacial spasm and their overcome. Jpn J Neurosurg 10:164-172, 2001
18. Miller LE, Miller VM: Safety and effectiveness of microvascular decompression for treatment of hemifacial spasm: A systematic review. Br J Neurosurg 26:438–444, 2012
19. Mizobuchi Y, Nagahiro S, Kondo A, Arita K, Date I, Fujii Y, Fujimaki T, Hanoya R, Hasegawa M, Hatayama T, Onoue T, Kasuya H, Konayashi M, Kahmura E, Matsushima T, Masuoka J, Morita A, Nizhizawa S, Okayama Y, Shigeno T, Shimano H, Tokeshima H, Yamakami I: Microvascular decompression for trigeminal neuralgia: A prospective, multicenter study. Neurosurgery 89:557-564, 2021
20. Pawlowski M, Gess B, Evers S: The Babinski-2 sign in hemifacial spasm. Mov Disord 28:1298-1300, 2013
21. Pusat S, Erbaş YC, Geyik M, Erdoğan E: Trigeminal nevraljide mikrovasküler dekompresyon: Tek merkezin deneyimi. Türk Nöroşir Derg 26:120-124, 2016
22. Samii M, Gunther T, Iaconetta G, Muehling M, Vorkapic P, Samii A: Microvascular decompression to treat hemifacial spasm: Long-term results for a consecutive series of 143 patients. Neurosurgery 50:712-718, 2002
23. Sindau M, Mercier P: Microvascular decompression for hemifacial spasm: Surgical techniques and intraoperative monitoring. Neurochirurgie 64:133-143, 2018
24. Tan EK, Chan LL: Clinico-radiologic correlation in unilateral and bilateral hemifacial spasm. J Neurol Sci 222:59–64, 2004
25. Tan EK, Chan LL: Young-onset hemifacial spasm. Acta Neurol Scand 114:59–62, 2006
26. Tash R, DeMerritt J, Sze G, Leslie D: Hemifacial spasm: MR imaging features. AJNR Am J Neuroradiol 12:839-842, 1991
27. Wang A, Jankovic J: Hemifacial spasm: Clinical findings and treatment. Muscle Nerve 21:1740-1747, 1998