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Microvascular Decompression for Hemifacial Spasm: Surgical Technical nuances and results after 300 microvascular decompression surgeries

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Microvascular Decompression for Hemifacial Spasm: Surgical Technical nuances and results after 300 microvascular decompression surgeries

Abstract

Introduction: Hemifacial spasm (HFS) is characterized by the involuntary, paroxysmal, painless, and progressive spasmodic contractions of facial muscles innervated by the ipsilateral seventh cranial nerve. To date, neuroimaging studies (Computed Tomography scan [CT] and Magnetic Resonance Imaging [MRI]) are unable to establish the diagnosis. HFS medical treatment with antiepileptic drugs, and Botulinum toxin application are temporarily effective, however, both have shown side effects and lesser cost-effective results. Currently, surgical Micro Vascular Decompression (MVD) for HFS has the highest curative rates and lower operative morbidity. We analyze the demographics, clinical manifestations, outcomes, and complications that to our knowledge, is the largest Latin-American patient's series treated for HFS through a keyhole microasterional craniectomy. **Objective:** The authors report the results of 300 Microvascular Decompression (MVD) for Hemifacial Spasm (HFS) in 265 patients due to nerve attrition by the neurovascular etiology, triggering of ectopic action potentials from the demyelinated facial nerve fibers. **Methods:** We reviewed and analyzed the clinical data from the medical records of patients treated by MVD for HFS from May 1992 to December 2018. Both preoperative MRI and audiometry studies were assessed in all patients as part of preoperatively evaluation. Patients with secondary causes of HFS such as tumors were excluded. **Results:** Among them, 168 [63.4%] were women and 97 [36.6%] males. 149 (56.2%) HFS were left-sided and with a better outcome compared to the 116 (43.8%) located on right side (p=0.22). The two main culprit vessels were AICA in 188 (70.9%) followed by PICA in 20 (7.5%). The basilar artery was identified in 14 (5.3%) and SUCA in 13 (4.9%). **Conclusions:** MVD through a retractorless microasterional approach is a very effective and a safe technique for treating HFS. Failure to HFS improvement after 1-week of MVD warrants immediate reoperation. In addition, MVD is a safe, the most effective technique and the only curative treatment for HFS.

Visual Abstract

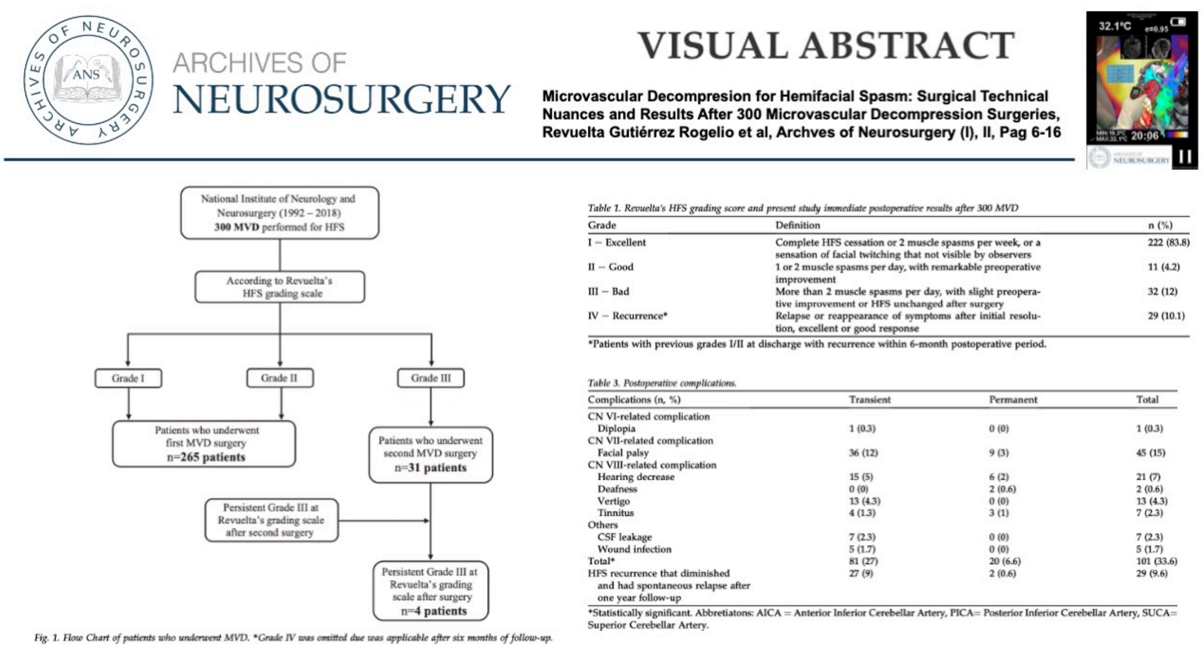

*Fig. 1. Flow Chart of patients who underwent MVD. *Grade IV was omitted due was applicable after six months of follow-up.*

Table 1. Revuelta's HFS grading score and present study immediate postoperative results after 300 MVD

Grade	Definition	n (%)
I – Excellent	Complete HFS cessation or 2 muscle spasms per week, or a sensation of facial twitching that not visible by observers	222 (83.8)
II – Good	1 or 2 muscle spasms per day, with remarkable preoperative improvement	11 (4.2)
III – Bad	More than 2 muscle spasms per day, with slight preoperative improvement or HFS unchanged after surgery	32 (12)
IV – Recurrence*	Relapse or reappearance of symptoms after initial resolution, excellent or good response	29 (10.1)

*Patients with previous grades I/II at discharge with recurrence within 6-month postoperative period.

Table 3. Postoperative complications.

Complications (n, %)	Transient	Permanent	Total
CN VI-related complication			
Diplopia	1 (0.3)	0 (0)	1 (0.3)
CN VII-related complication			
Facial palsy	36 (12)	9 (3)	45 (15)
CN VIII-related complication			
Hearing decrease	15 (5)	6 (2)	21 (7)
Deafness	0 (0)	2 (0.6)	2 (0.6)
Vertigo	13 (4.3)	0 (0)	13 (4.3)
Tinnitus	4 (1.3)	3 (1)	7 (2.3)
Others			
CSF leakage	7 (2.3)	0 (0)	7 (2.3)
Wound infection	5 (1.7)	0 (0)	5 (1.7)
Total*	81 (27)	20 (6.6)	101 (33.6)
HFS recurrence that diminished and had spontaneous relapse after one year follow-up	27 (9)	2 (0.6)	29 (9.6)

*Statistically significant. Abbretiatons: AICA = Anterior Inferior Cerebellar Artery, PICA= Posterior Inferior Cerebellar Artery, SUCA= Superior Cerebellar Artery.

Keywords
Hemifacial Spasm, Microvascular Decompression, Asterional approach

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Microvascular Decompression for Hemifacial Spasm: Surgical Technical Nuances and Results After 300 Microvascular Decompression Surgeries

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Abstract

Introduction: **Hemifacial spasm (HFS) is characterized by the involuntary, paroxysmal, painless, and progressive spasmodic contractions of facial muscles innervated by the ipsilateral seventh cranial nerve. To date, neuroimaging studies (Computed Tomography scan [CT] and Magnetic Resonance Imaging [MRI]) are unable to establish the diagnosis. HFS medical treatment with antiepileptic drugs and Botulinum toxin application is temporarily effective; however, both have shown side effects and lesser cost-effective results. Surgical microvascular decompression (MVD) for HFS has the highest curative rates and lower operative morbidity. We analyze the demographics, clinical manifestations, outcomes, and complications of an HFS patient's series treated through a keyhole microasterional craniectomy that, to our knowledge, is the most extensive among Latin America.**

Objective: **The authors report the results of microvascular decompression (MVD) for hemifacial spasm (HFS) due to nerve attrition by a neurovascular etiology.**

Methods: **We reviewed and analyzed the clinical data from patients' medical records treated by MVD for HFS from May 1992 to December 2018. We only included patients with a neurovascular etiology while excluded any other causes such as tumors. We assessed both preoperative MRI and audiometry studies in all patients as part of preoperative evaluation.**

Results: **Two-hundred sixty-five patients underwent three-hundred MVD for HFS; among them, 168 [63.4%] were females and 97 [36.6%] males. One-hundred forty-nine (56.2%) HFS were left-sided located and one-hundred sixteen (43.8%) right-sided. The two main culprit vessels were AICA in one-hundred eighty-eight cases (70.9%), followed by PICA in twenty (7.5%), followed by the basilar artery in fourteen (5.3%), and the SUCA in thirteen (4.9%).**

Conclusions: **MVD through a retractor-less microasterional approach is a very effective and safe technique for treating HFS. In addition, MVD is the only curative treatment for HFS; therefore, failure to improve after 1-week of MVD warrants immediate reoperation for HSF.**

Keywords: Hemifacial spasm, Microvascular decompression, Asterional approach

1. Introduction

Hemifacial spasm (HFS) is characterized by involuntary, paroxysmal, painless, and progressive spasmodic contractions of facial muscles innervated by the ipsilateral seventh cranial nerve [1–10]. HFS tends to be predominantly left-sided and to have a 2:1 preponderance ratio over the female gender [11,12]. The HFS prevalence has been

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reported in 10 cases/100,000 people [13,14] being primarily patients over the fifth decade of life [6,11].

HFS is classified as typical and atypical [11,12,15] according to the initial twitching manifestation and rendering to the occurrence in the superior or inferior half of the face, respectively. While untreated, the overall tendency is to worsen over time [4,9] with the potential to develop various grades of facial palsy [16,17]. HFS is referred to as "tic convulsive" when co-occurring with trigeminal neuralgia (TN), as Cushing's first description in 1920 [18]. Though HFS occurs unconsciously, it can be elicited by triggering maneuvers and psychogenic stimuli [4,11,19]. Even though HFS contractions decrease during rest, HFS and palatal myoclonus persist during sleep [2,20–23]. Medical treatment with carbamazepine, clonazepam, and gabapentin can only achieve poor and transient improvement [9,24,25]. Botulinum toxin type A in HFS has shown improvement of 75–100% [26,27]; however, beneficial effects are temporary and periodic applications are required [3,5,9,24]. In addition, botulinum toxin type A is not innocuous, carries potential side effects, and has a lesser cost-effective result [9,28–30].

The clinical features of HFS at examination (e.g., facial twitching) are keys for the diagnosis; however, ancillary tests such as electromyography (EMG) can also be of help [24,25,31]. Neuroimaging studies alone cannot establish the diagnosis; nevertheless, computed tomography scan (CT-scan) [25,32,33], magnetic resonance imaging (MRI) [4,24,34–39], and hemodynamic studies [40] are valuable tools to rule out posterior fossa pathology other than vascular compression. Authors have hypothesized the mechanism of HFS to be the result of the pulsatile cross-compression over the seventh (facial-VII) within the Root Entry/Exit Zone (REZ) by 1 or 2 arterial loops [3–5,34] causing an ephaptic transmission between individual nerve cells fibers dropped the excitability threshold of the neurons in the compression zone [31,41,42]. The Anterior Inferior Cerebellar Artery (AICA) [3,43] and the Posterior Inferior Cerebellar Artery (PICA) [44–46], are considered the main culprit vessels involved in the compression, varying its frequency depending on the series.

Since Jannetta established and popularized the MVD technique in the 1970s [17,44,46–48], technical improvements have allowed other surgeons to perform MVD with minimally invasive approaches [12,20,25,38,44,49,50]. Currently, MVD for HFS has cure rates up to 90% and operative morbidity of less than 10% [11,30,51,52] being the only curative option that "treats" the cause of HFS hemifacial spasm.

Abbreviations

MVD	Micro Vascular Decompression
MRI	Magnetic Resonance Imaging
TN	Trigeminal Neuralgia
REZ	Root Entry Zone
AICA	Anterior Inferior Cerebellar Artery
PICA	Posterior Inferior Cerebellar Artery
SUCA	Superior Cerebellar Artery
EMG	Electromiography
CT	Computed Tomography
NINN	National Institute of Neurology and Neurosurgery
HB	House Brackman
CPA	Cerebello pontine angle
CSF	Cerebrospinal fluid
SD	Standard Deviation
IQR	Inter Quartile Range
BAEP	Brainstem Auditory Evoked Potentials
FIESTA	Fast Imaging Employing Steady-State Acquisition

This study aims to analyze the demographics, clinical manifestations, outcomes, and complications of an HFS patient's series treated through a keyhole microasterional craniectomy that, to our knowledge, is the most extensive among Latin America.

2. Patients and methods

We reviewed and analyzed the clinical data and medical files of patients treated with MVD for HFS from May 1992 to December 2018; we made this study in adherence to STROBE guidelines for retrospective observational cohorts. We only included patients with a neurovascular etiology while excluded any other causes such as tumors. We assessed both preoperative MRI and audiometry studies in all patients as part of preoperative evaluation. Our institutional ethics board approved this study.

2.1. Data collection and follow-up

We collected data including age, sex, previous medical history, duration of HFS, previous nonsurgical treatments, complications, and presence or absence of HFS at the time of hospital discharge. HFS outcome was set on the relief of symptoms and classified according to the previously described Revuelta's HFS score-criteria [12,53] (see Table 1): "Spasm free" group, grades I (Excellent) and II (Good); and 2) "Spasm-relapse," grades III (bad) and IV (recurrence). In addition, we also registered surgical findings such as the offending vessel, intraoperative photos, or video recordings for each case. All patients were followed for at least six months after surgery on consecutive assessments at the outpatient clinic.

Table 1. Revuelta's HFS grading score and present study immediate postoperative results after 300 MVD

Grade	Definition	n (%)
I – Excellent	Complete HFS cessation or 2 muscle spasms per week, or a sensation of facial twitching that not visible by observers	222 (83.8)
II – Good	1 or 2 muscle spasms per day, with remarkable preoperative improvement	11 (4.2)
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IV – Recurrence*	Relapse or reappearance of symptoms after initial resolution, excellent or good response	29 (10.1)

*Patients with previous grades I/II at discharge with recurrence within 6-month postoperative period.

Postoperative complications that recovered spontaneously within the first 6-months after surgery were assigned as "transient" and "permanent" when persisted after six months. We used the House- Brackmann (HB) score to establish the facial palsy grade, while we assessed hearing loss comparing to a preoperative audiological evaluation [54]. In both cases, we measured the duration of symptoms after MVD.

2.2. Technical nuances and surgical technique

All surgeries were performed by a senior Neurosurgeon (R.R.G), excluding potential bias related to surgeon skills and experience [55].

2.3. Patients position

We performed all MVDs under general anesthesia and park bench positioning with the head fixed in a Mayfield skull clamp. After ventral and caudal shoulder retraction, the head was rotated 60° opposite the HFS side with a slight lateral tilt of approximately 15° towards the floor to allow an optimal surgical corridor through the seventh nerve REZ as reported previously [56–58].

2.4. Transverse and sigmoid sinus projection

We first identified the landmarks, including the inion, the mastoid tip and notch, and a projection of the transverse sinus using Reid's baseline. We then performed a small (5 cm approximately) curvilinear rectosigmoid skin incision 3 cm behind the posterior ear (or 4.5 cm behind external acoustic meatus) over the transverse sinus projection. Next, we conducted a subperiosteal muscle dissection using sharp square periosteal elevators and monopolar (BovieTM) coagulation.

2.5. Microasterional craniectomy

The asterion is a landmark representing the junction of the lambdoid, parietomastoid, and occipitomastoid sutures. According to Rhoton's technique, we used the asterional burr-hole, as this method reduces the risk of lacerating the sinus complex in the posterior part of the parietomastoid suture, which is related to the transverse sinus's superior margin and its transition to the sigmoid sinus. From 1998 on, we used only one burr hole placed two centimeters below the asterion, two-thirds behind and one-third in front of the occipitomastoid suture. Under this technique, the microasterional keyhole craniectomy (2.5–3 cm) lays just below the inferior recess of the ipsilateral transverse sinus. Additionally, we applied wax vigorously over the edges to fill the defect when we opened mastoid air cells.

2.6. Dural opening

We performed a dural "C" shaped incision (1.5–2 cm) with the base at the transverse-sigmoid sinus junction to achieve the optimal dural angle exposure between tentorium and petrous surface [56–58]. Thus, when we correctly placed the microasterional craniectomy and correctly opened the dural, we did not need stitches to access both the CPA and infratentorial/supracerebellar corridors.

2.7. Intradural dissection

To amplify the operative field, we directed highly hydrated cottonoid patties rostrocaudally to allow drainage of the CSF. After sharp dissection of arachnoid membranes, we facilitated cisternal release by meticulous CSF depletion; then, the operative corridor is retractor-less opened using both the suction tube and bipolar electrocautery over soggy patties taking advantage of gravity. After optimal cerebellar relaxation, the cerebellum falls away from the petro-tentorial junction. Using the infrafloccular corridor, we could observe the pathway of the cisternal segment of the facial nerve from REZ to its bony entrance at the internal auditory canal.

2.8. Microvascular decompression

After visualization of neurovascular conflicts, we confirmed the diagnosis, proceeding to move the offending vessels away using microsurgical dissectors on the left and off-bipolar on the right hand. After we identified the conflict zone, we applied one to three Teflon felt pledgets. When we observed no evidence of vascular contact on the seventh-nerve (n = 12 patients), instead of Teflon threads placement, we used very subtle compression over the REZ of the facial nerve using the off-bipolar for no more than five seconds. Finally, we closed the dura mater with a watertight technique.

2.9. Statistical analysis

We analyzed categorical variables using descriptive statistics (frequencies and percentages). For continuous variables, we used dispersion measures (means and SD). In addition, we used the student T-test to compare pre and postoperative status and outcomes between groups. Lastly, we performed a factorial regression analysis to identify preoperative risk factors.

3. Results

3.1. Demographics results

From May 1992 to December 2018, we collected 265 consecutive patients with diagnosis HFS admitted to the Department of Neurosurgery at NINN. Among them, 168 [63.4%] were women and 97 [36.6%] males. One-hundred forty-nine cases of HFS (56.2%) were left-sided, and one-hundred sixteen (43.8%) were right-side. Table 2 shows other additional demographic characteristics.

3.2. Preoperative clinical findings

Only four (1.5%) patients had a preoperative HB score of 2; the remaining two hundred sixty-one (98.5%) were grade 1. Concerning their HFS symptoms onset, two hundred thirty-seven (89.4%) typical HFS began in the lower eyelid progressing downwards to oral commissure, while twenty-eight atypical HFS (10.6%) had mouth muscles initially affected with spreading upwards. All the patients received an audiometric evaluation (n = 265); two hundred thirty-one (87.2%) of them had a normal result, while thirty-five (12.8%) were abnormal (Table 2). Because our institution is public and a national reference center, we performed brainstem auditory evoked potentials (BAEP) selectively in two hundred-seven patients that could afford the extra cost involved. BAEP was normal in one hundred seventy-two (83.1%) and abnormal in thirty-five (16.9%). Table 2 shows the summary of other essential preoperative clinical findings.

Table 2. Clinical characteristics and preoperative findings of the Study Patients.

Characteristics	Total (N = 265)	Free of HFS (n = 233)	Failure (n = 32)	P Value
Age (years)	50.4 ± 12.7	50.0 ± 12.2	53.63 ± 15.6	0.133
Sex				0.78
Male	97 (36.6%)	86 (88.7)	11 (11.3)	
Female	168 (63.4%)	147 (87.5)	21 (12.5)	
*Spasm side				*0.022
Right	116 (43.8)	108 (93.1)	8 (6.9)	
Left	149 (66.2)	125 (83.9)	24 (16.1)	
Type of Spams				0.124
Typical	237 (89.4)	211 (89.0)	26 (11.0)	
Atypical	28 (10.6)	22 (78.6)	6 (21.4)	
Botulinum Toxin				1.0
No	231 (87.2)	203 (87.9)	28 (12.1)	
Yes	34 (12.8)	30 (88.2)	4 (11.8)	
Previous Surgery				1.0
Yes	3 (1.1)	3 (100)	0 (0)	
No	262 (98.9)	230 (87.8)	32 (12.2)	
*BSAP				0.583
Normal	172 (64.9)	152 (88.4)	20 (11.6)	
Abnormal	35 (35.1)	30 (85.7)	5 (14.3)	
Audiometry				*0.044
Normal	231 (87.2)	207 (89.6)	24 (10.4)	
Abnormal	34 (12.8)	26 (76.5)	8 (23.5)	
Type of Approach				0.7
Asterional	247 (93.2)	216 (87.4)	31 (12.6)	
Jannetta	18 (6.8)	17 (94.4)	1 (0.6)	
Vessel in MRI				
No	115 (43.4)	104 (90.4)	11 (9.6)	
Yes	150 (56.6)	129 (86.0)	21 (14.0)	
Vessel				0.4
None	12 (4.5)	12 (100)	0 (0)	
SUCA	13 (4.9)	13 (100)	0 (0)	
AICA	188 (70.9)	165 (87.8)	23 (12.2)	
PICA	20 (7.5)	17 (85.0)	3 (15)	
Basilar	14 (5.3)	11 (78.6)	3 (21.4)	
More than one	18 (6.8)	15 (83.3)	3 (16.7)	
Type of Material				
Dacron	10 (3.8)	7 (70.0)	3 (30.0)	
Silastic	15 (5.7)	11 (73.3)	4 (26.7)	
Teflon	240 (90.6)	215 (89.6)	25 (10.4)	

*Statistically significant. Abbreviations: SD= Standard Deviation, IQR= Interquartile range, CBZ=Carbamazepine, PHT = phenytoin. * Tic convulsive defined as HFS concomitant with Trigeminal Neuralgia.

3.3. Preoperative MRI and neuro-radiological reports

All the two hundred sixty-five patients were evaluated with a 1.5 T MRI (Siemens) using T1/T2 weighted sequences to rule out other posterior fossa pathology rather than the suspected microvascular compression over the seventh nerve. Additionally, we performed fast imaging employing steady-state acquisition (FIESTA) sequence preoperatively to assess CSF within dural reflections of VII-VIII cranial nerves in all the patients. Brain-MRI was reported as normal in one hundred fifteen (43.4%) of the patients, and the offending vessel was presumptively identified in one hundred fifty (56.6%) of the patients (see Table 2).

3.4. Surgical results

A total of three hundred patients underwent MVDs (two-hundred sixty-five first-time surgery and thirty-five reinterventions) in two-hundred sixty-five patients in 26 years (Fig. 1).

3.5. Offending vessels and surgical findings

We identified a total of two hundred fifty-three offending vessels. The two main culprit vessels were AICA in one-hundred eighty-eight of the cases (70.9%), PICA in twenty (7.5%), the basilar artery in fourteen (5.3%), and SUCA in thirteen (4.9%). Compression by two different "loops" originated from different vessels was seen in eighteen (6.8%), while we found no offending vessel in twelve (4.5%) patients. We used Teflon two-hundred forty (90.6%) of the patients, which we consider the preferred material (after 1995) because of its ease of use, price, and maneuverability (see Table 3). Table 2 shows the remaining postoperative results.

3.6. Postoperative HFS-relief outcomes

In the whole two-hundred sixty-five patient cohort, we discontinued all conservative treatments after surgery. We performed a total of three hundred MVDs was performed, two-hundred sixty-five (88.3%) were first-time surgery; meanwhile, thirty-one (10.3%) patients needed a first reintervention, and among them, only four (1.3%) underwent a

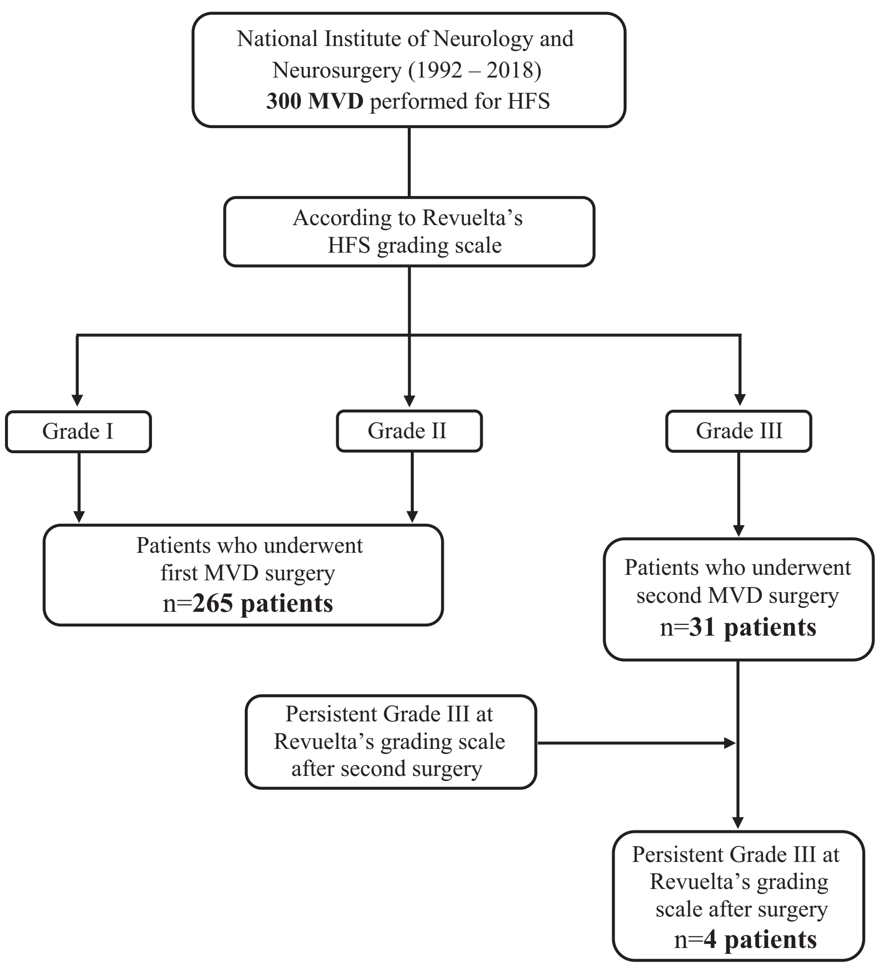

*Fig. 1. Flow Chart of patients who underwent MVD. *Grade IV was omitted due was applicable after six months of follow-up.*

Table 3. Postoperative complications.

Complications (n, %)	Transient	Permanent	Total
CN VI-related complication			
Diplopia	1 (0.3)	0 (0)	1 (0.3)
CN VII-related complication			
Facial palsy	36 (12)	9 (3)	45 (15)
CN VIII-related complication			
Hearing decrease	15 (5)	6 (2)	21 (7)
Deafness	0 (0)	2 (0.6)	2 (0.6)
Vertigo	13 (4.3)	0 (0)	13 (4.3)
Tinnitus	4 (1.3)	3 (1)	7 (2.3)
Others			
CSF leakage	7 (2.3)	0 (0)	7 (2.3)
Wound infection	5 (1.7)	0 (0)	5 (1.7)
Total*	81 (27)	20 (6.6)	101 (33.6)
HFS recurrence that diminished and had spontaneous relapse after one year follow-up	27 (9)	2 (0.6)	29 (9.6)

*Statistically significant. Abbretiatons: AICA = Anterior Inferior Cerebellar Artery, PICA= Posterior Inferior Cerebellar Artery, SUCA= Superior Cerebellar Artery.

second reintervention due to the persistence of HFS. For those who were re-operated, we saw no material migration either at first or second reintervention. Only three (1.1%) patients were previously surgically treated in other hospitals from the complete patient's sample. The immediate postoperative HFS relief according to Revuelta's HFS grading score results is displayed in Table 1. On the twelve patients whom a neurovascular conflict was not demonstrated, we obtained excellent results in 9 (75%), good in 2 (16.7%), and bad in only 1 (8.3%) patient after both the appliance of Teflon threads and very light compression with bipolar over seventh's nerve REZ for no more than 5 s.

3.7. Complications

Postoperative facial weakness was more common than hearing loss. Facial palsy was found in 45 (15%) at postoperative day 1 (HB5 and 4) with a recovery rate of 80% in the first 6-months of follow-up. We could not identify the offender vessel in 12 patients requiring light compression with the off-bipolar at seventh's nerve REZ; from them, four patients developed an HB 2, and one patient developed an HB 3 with hearing decrease with full recovery 6-months of follow up period. Hearing decrease was found in 21 patients (7%); among them, 15 patients had full hearing function recovery after 6-months of follow-up, six patients continued with a non-useful hearing by audiometry after a 1-year follow-up, and two (0.6%) with deafness. We had only one patient with diplopia who recovered spontaneously in 3 months after surgery. Table 3 demonstrates the information regarding other complications. We treated CSF leakages with external lumbar drainage showing complete resolution after the fifth day. Wound infections were entirely resolved with intravenous antibiotics demonstrating consecutive negative cultures within two weeks. Twenty-nine

Table 4. Logistic regression analysis for complications after 300 MVD for HFS.

Prognostic Factors	P Value	Odds Ratio	95% CI	
			Inferior Limit	Superior Limit
Age (years)	0.088			
Sex	.932	.973	.514	1.839
Spasm side	.387	1.305	.714	2.383
Type of Spams	.356	1.577	.600	4.147
Botulinic Toxin	.588	1.310	.493	3.478
Previous Surgery	1.000	.000	.000	.
BSAP	.185	1.859	.743	4.648
Audiometry	.803	.884	.335	2.330
**Type of Approach	.063	4.804	.921	25.060
Vessel	.54	1.698	1.202	2.399
Type of Material	.259	.483	.230	1.013
Culprit vessel in MRI*	0.003	.683	.352	1.325

*Statistically significant, ** Microasterional approach after 1998, and Retrosigmoid approach before 1998 were used.

patients (12.4%) presented recurrence of a grade IV HFS after a previous grade I or II within the initial six months to 2-years after the initial surgery. However, we performed no additional surgical intervention because spasms diminished spontaneously and attained quiescently. Table 4 shows the analysis of complications after the logistic regression; identifying a culprit vessel previous to surgery was the only factor statistically significant with a $p = 0.003$.

4. Discussion

The estimated incidence of HFS is 0.8 cases per 100,000 individuals with a 2:1 female preponderance ratio and a prevalence reported in 14.5 women per 100,000 cases and 7.4 males per 100 000 cases [4,6,11,25]. Since the first case of HFS described in the literature by F. Shültze in 1875, the hallmark of this entity has been the involuntary, paroxysmal, left-sided, and painless contractions of the muscles innervated by the facial nerve in middle-aged women [3,11,13,24,25].

Contrastingly to its unilateral nature (hemi = one side), there exist bilateral HFS cases [10,15,34,61,62] and HFS in patients under 30-year, as the so-called "young-onset HFS" [6,63,64]. In our study, the afflicted patients were predominantly females (63.4%) over the fourth and fifth decade of life, and with a slightly left-side apparition (56.2%), these results are consistent with the HFS current literature reported [3,4,7,9,11,46].

Typical HFS symptoms start on the lower eyelid and periorbital musculature but later involve the ipsilateral facial, perioral, and platysma muscles over months to years [1,11]. On the other hand, patients with atypical syndrome start instead on the lower half of the face [11,12,15]. Like other series, we found the "typical" syndrome onset as the most common HFS in two-hundred thirty-seven (89.4%), while finding "atypical" syndrome in twenty-eight (10.6%). We found only twelve (4.5%) patients with "tic convulsif" or "tic dolorous," as the initially Cushing's description at 1920 when HFS is coexisting with TN [18,59].

Although in practice, the underlying HFS etiology is the nerve attrition due to an atypically aberrant blood vessel, two main theories explain the pathophysiology of HFS. The "peripheral" hypothesis stands that the neurovascular interface triggers ephaptic and ectopic action potentials from the locally demyelinated facial nerve fibers by surrounding arterial loops, which compresses the facial nerve within or close to the REZ [3–5,23,34]. The opposite "central" hypothesis stands that hyperexcitability arises from the facial motor nucleus in the brainstem [3]. HFS diagnosis is easy to ascertain clinically by simple observation of the patient's face; however, it is frequently confused with other facial movement disorders. Neurodiagnostic studies are rarely helpful in HFS. Recent evidence suggests that MRI should be performed when possible using a 3T magnet due to signal-to-noise and contrast-to-noise ratios better at 3T than lower field strengths [35]. However, neuroimaging studies have also found nerve conflicts at VII/VIII nerve complex in asymptomatic patients [36,37]. The anatomic conspicuity of the posterior fossa and the poor delineation of cranial nerves on image studies complicate small vessels' assessment. Surprisingly, the interobserver discrepancy and differences between image studies concerning the conflict vessels reported preoperatively in HFS on MRI studies correlate purely with the actual vessels seen at surgery compressing the VII nerve [5]. From one hundred fifty patients' studies with an abnormal MRI, we found that ninety-eight (37%) of them reported a non-specific vascular loop, and forty-seven (17.7%) reported a dolichoectatic basilar artery; while five (1.9%) only reported an "abnormal MRI." MRI-angiography may demonstrate the vascular features around the root entry zone [11]. Although a CT or MRI screening imaging study may not be justified in all cases of HFS, it is prudent to employ these diagnostic studies in all atypical cases to rule out tumoral pathology rather than an accurate compression over a specified vessel.

Our experience with these patients has led us to think that long-standing disease and delay in surgical treatment frequently lead to social isolation. As the diagnosis can be difficult, a psychogenic stimulus such as emotional stress, repetitive face movements, and even speaking can elicit and trigger spasms, helping in differentiate them from other facial movements disorders such as blepharospasm, oromandibular dystonia, facial tic, hemimasticatory spasm, myokymia, and facial myorhythmia [4,10,19,27]. HFS, along with palatal myoclonus, is the only two-movement disorder that persists during sleep [2,20–23].

Rather than surgery, other therapeutic options consider oral medications, acupuncture, and botulinum toxin type A injections [9,24,27]. The subcutaneous injections with botulinum toxin type A on the facial affected muscles produce complete or almost complete relief of symptoms in 76–100% of patients [28,65]; however, the improvement is only temporary (mean duration, 3–6 months). In addition, despite the effectiveness of botulinum toxin injections can be maintained for years to alleviate the symptoms of HFS, it represents a less cost-effective treatment due to dosage that may need to be gradually increased to achieve its desired effect [9,28]. In this series, we found the main alternative conservative treatment received was botulinum toxin type A in thirty-four (12.8) patients, followed by CBZ and PHT in twenty-one (7.9%). Furthermore, we saw a mean of three years delay before MVD as definitive treatment among those who received botulinum toxin type A.

In Mexico, applying a single botulinum toxin injection for HFS cost between 5500 and 7300 Mexican pesos (275–365 US Dollars). This cost includes medical fees, botulinum toxin bottle (AllerganTM) at each session. Nevertheless, one patient with HFS needs approximately four sessions, summing a total of 22,000–29,200 Mexican pesos (1100–1460 US Dollars) for optimal relief for three months. On the other side, in our hospital, the mean individual payment for MVD for HFS is 3000 Mexican pesos (150 USD), including surgery, doctor fees, and in-hospital care for three postoperative days.

The only curative treatment and HFS long-term control that treats the cause of HFS has been the nerve decompression of the culprit's vessel on REZ [17,38,45]. This zone, also called the Obersteiner-Redlich zone, corresponds with a transitional zone between central and peripheral myelin. HFS has excellent clinical results after MVD when performed by a neurosurgeon who has experience in the nuances of the operative procedure; however, newer MVD technique has been strengthened over time by the endoscope integration in the so-called endoscope-assisted (EA) surgery. When combined, EA procedures bring inherent endoscope's advantage in "looking around the corner." Other advantages in using EA are avoidance of cerebellar retraction in patients with complicated or abnormal posterior fossa anatomy where the microscope's view alone limits identification of neurovascular conflicts [66,67].

In the present series, we found the anterior inferior cerebellar artery (AICA) as the primary culprit vessel in one hundred eighty-eight (70.9%) [3,9,12,43,68]; however, others have reported the posterior inferior cerebellar artery (PICA) as the most common vessel responsible [17,40,44,45,69] (PICA twenty (7.5%) in our series). More rarely, vein compressions as etiology for HFS have also been described [69,70]. Interestingly, although most nerve–vascular complexes responsible for HFS are either rostrocaudally or ventrocaudal at the REZ or brainstem, a blood vessel on the posterior or rostral side of the nerve could cause the atypical HFS; however, these findings were not statistically significant in this case series.

Sindou et al., in 2018 reported a variety of grading scales for assessing the outcome after MVD for HFS [52]; however, returning to the patient's self-assessed previous everyday life is the best indicator that impacts the final outcome. Therefore, the patient's HFS relief appreciation is the best indicator that the surgeon could achieve (RRG).

The overall consensus for reoperation in recurrent HFS after MVD has been set to 12 months [52]. However, the senior surgeon's opinion (RRG) is that patients with recurrent symptoms in the first four days after surgery require immediate exploration, and those with later recurrence without facial paresis are likely to improve within the next six months to 2 years. Although HFS is painless and non-life-threatening, HFS affects facial appearance; the cosmetic and aesthetic discomfort becomes socially and frequently economically disabling [46]. In severe cases, the facial disfigurement generates high anxiety levels with potentially psychiatric complications that could directly impact self-image and satisfaction [9]; this severe condition requires timely diagnosis and therapy [24]. In the MVD, as in the rest of Neurosurgical procedures, high precision preoperative planning is mandatory.

This series is the largest to date on a Hispanic population. In high-volume centers with experienced Neurosurgeons, the MVD for HFS through a microasterional craniectomy approach constitutes a reliable surgical treatment at which it is possible to achieve optimal exposure of the central aspect of the CPA. The main challenge is the limited microsurgical working space to achieve a successful decompression and optimal brain stem structures [45]. In addition, the microasterional craniectomy approach is minimally invasive, nondestructive, and achieves good long-term results with minor morbidity.

This study has two limitations. First, we did not use intraoperative EMG and BAEP due to the cost involved. Electrophysiological monitoring is a valuable tool in MVD surgeries, such as monitoring the abnormal muscle response (AMR) while evaluating the offending vessels and ensuring adequate decompression, or by BAEP monitoring to reduce the incidence of hearing impairment [71]. Unfortunately, this circumstance prevented us from observing an association between neurophysiological studies and postoperative deficits. Second, the inherent limitations of retrospective, single-center investigations. The main delay in receiving surgical treatment was over-population at our institution and the surgical cost involved. We did not compare MVD effectiveness over patients only treated with botulinum toxin in a control group. Extensive studies are lacking that compare the possible therapeutic options in randomized and controlled trials. Despite these limitations, we recommend that surgery be deliberately decided, especially in the early stages, and immediate reoperation in spasm relapse within 1-week of surgery.

5. Conclusions

Due to the recent technological advancements in microsurgery, extensive craniotomies have been replaced by less invasive keyhole craniotomies. We have found that the patients' age, gender, pathology side, and the time from onset of symptoms are non-related factors in achieving a good outcome after MVD in HFS after surgical treatment.

The use of alternative treatments, different from surgical, tends to worsen HFS over a variable period. While non-life-threatening, HFS can significantly affect the quality of life.

In well-trained hands, MVD through a retractorless microasterional approach is an effective and safe technique for treating HFS; in addition, MVD is the only curative treatment for HFS. Therefore, failure to HFS improvement after 1-week of MVD warrants immediate reoperation.

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Reviewer Comment 1:

Microvascular Decompression for Hemifacial Spasm: Surgical Technical Nuances and Results After 300 Microvascular Decompression Surgeries
This paper shows the outstanding results of case series of microvascular decompression for hemifacial spasms. The authors also report the demographic characteristics of the patients and the most frequent etiology causing the compression. This referral center shows a significant concentration of these patients, which determined a large sample of collected cases.

The author also describes the technique dominated by this group of microasterional craniotomy, which provides a minimally invasive approach to the middle cerebellar complex and excellent exposure to the structures involved.

The results obtained in improving spasms are satisfactory and preserve facial and auditory function. This group recommends performing a new intervention if there is no improvement in the spasm in the first week after surgery. On the opposite, other studies in similar patients suggest waiting longer. It will be worthwhile to conduct prospective and comparative studies between various hospital centers to contrast the results.

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