Outcomes of Microvascular Decompression for Hemifacial Spasm at the Philippine General Hospital

Gerardo D. Legaspi, MD, Lady Scarlette P. Sedano, MD and Juan Silvestre G. Pascual, MD

Division of Neurosurgery, Department of Neurosciences, College of Medicine and Philippine General Hospital, University of the Philippines Manila

ABSTRACT

Objective. To report the demographics, clinical characteristics, and surgical outcomes after microvascular decompression (MVD) for hemifacial spasm (HFS) in the Philippine General Hospital (PGH).

Methods. Between January 2018 to December 2022, the division of Neurosurgery at PGH performed thirty-four MVD operations for Primary HFS. Records were retrospectively reviewed, and pertinent demographic, intra-operative findings, outcomes, and complications were analyzed.

Results. The overall success rate is 88.2%, with a median follow-up at 15 months. Repeated measures ANOVA showed that post-op HFS and follow-up HFS grades were significantly lower than pre-op HFS grades (F=17.46, df=33, p<0.0001). HFS average age of symptom onset was early at 38.5 years, 11.8% of which were <30y/o. The M: F ratio was 1:2.4. Thirty out of 34 patients came for MVD surgery as referrals from a social media support group. Ordered logistic regression showed that years before surgery (OR:7.05, z=2.23, p=0.026) and pre-operative pharmacologic treatment (OR:48, z=2.33, p=0.02) increase the HFS grade to the next worse grade. At the same time, hypertension (OR: 0.006, z=-2.54, p=0.011) decreases pre-operative HFS grade to a lower degree. Post-operative complications were facial nerve palsy (29.4% transient and 5.9% permanent), hearing loss (17.6% transient and 5.9% permanent), CSF leak/infection (2.9%), and recurrence (8.8%).

Conclusion. MVD at PGH is a viable choice treatment option for Filipino patients with success rates comparable to internationally published studies. Besides improving surgical technique, improvement of referral systems may help make surgery more accessible.

Keywords: microvascular decompression, hemifacial spasm, retrospective review

INTRODUCTION

Hemifacial spasm (HFS) is a chronic, highly morbid unilateral movement disorder, first reported by Shultze in 1875.[1] It is characterized by twitching, tonic spasm, and synkinesis of the muscles innervated by the facial nerve.[2]

Primary HFS satisfies the following three requirements: It is (1) persistent and self-limiting, (2) not a complication of ipsilateral facial palsy, and (3) the only diagnostic finding is a vascular structure in proximity with the ipsilaterally affected facial nerve, generally in the Root Exit Zone (REZ) at imaging.[3,4] This phenomenon of misfiring of the facial nerve is due to an ephaptic transmission brought about by vascular compression. Ephaptic transmission refers to the communication of neurons via electrical conduction through the surrounding extracellular space rather than through the chemical synapses.[5]

The average annual incidence of HFS is 11 per 100,000 population.[6] Further studies show a higher incidence in Asian populations, with a prevalence of 12.63 per 100,000

Paper presentation – Academy of Filipino Neurosurgeons Resident Research Contest, November 30, 2023, EDSA Shangri-La Hotel, Philippines.

eISSN 2094-9278 (Online)
Published: September 15, 2025
https://doi.org/10.47895/amp.v59i13.11209

Corresponding author: Juan Silvestre G. Pascual, MD
Division of Neurosurgery
Department of Neurosciences
Philippine General Hospital
University of the Philippines Manila
Taft Avenue, Ermita, Manila 1000, Philippines
Email: jgpascual@up.edu.ph
ORCiD: https://orcid.org/0000-0003-4611-3011

population compared to that of Caucasians at 7.20 per 100,000 population.[7]

Medical treatment available for HFS includes using anti-convulsants such as carbamazepine, clonazepam, gabapentin, anticholinergics, baclofen, haloperidol, and botulinum toxin injections.[2,8] Microvascular decompression (MVD) of the facial nerve is the definitive surgical treatment for HFS.[1,2,8–16] It was developed by Peter Janetta in 1965 from his initial neurovascular compression theory.[11,13] Following MVD, the symptom resolution rate was over 92% at six months, and 90% of patients were still free of spasms at one year.[10] However, it carries a considerable risk of complications with the procedure. Hearing loss (7%-26%), facial nerve damage (2.8%-8.3%), and cerebrospinal fluid (CSF) leak (2%-30%) are the common morbidities associated with the procedure.[3,9,14,16–19] To improve vestibulocochlear outcomes, many surgeons advocate using intraoperative brainstem auditory evoked response (BAER) monitoring.[20,21] In the Philippine General Hospital (PGH), the largest tertiary referral hospital in the Philippines, which caters to an average of 1500 neurosurgical operations per year, intra-operative BAER monitoring is not readily available. Hence, MVD is done without this adjunct. To our knowledge, no published studies have investigated the outcomes of microvascular decompression in the Philippines. The primary objective of this retrospective review is to assess the results of MVD without using intra-operative brainstem auditory evoked response (BAER) monitoring in PGH.

METHODS

Study Procedure

After ethics approval by the University of the Philippines Manila Research Ethics Board (UPM-REB 2023-0090-01), a retrospective records review of clinical in-patient and out-patient data of all adult patients who underwent MVD for HFS operated on at PGH between 2018-2022 was done. Inclusion criteria were all patients with primary hemifacial spasms with MRI or imaging proof of a vessel in the proximity of the ipsilateral side of the patient's spasms, and this is their first MVD surgery. We excluded patients with no documented blood vessel seen on the imaging, history of prior MVD in another institution, patients who underwent MVD for trigeminal neuralgia, and post-MVD patients who were operated on before or after the specified period. Patients with history of medical management nor Botox treatment were not excluded.

The clinical data consisted of demographic characteristics, co-morbidities, symptomatology (side of HFS, pre-operative duration of symptoms), previous medical treatment done, pre-operative craniopathies, pre-operative hemifacial spasm grade, post-operative length of stay, and offending vessel recorded on the operative technique. Post-operative clinical data included the primary outcome measure: the post-operative hemifacial spasm grade, and secondary outcomes were mortality and morbidity (CSF leak or pseudo meningocele formation, new onset facial weakness, new onset ipsilateral hearing loss, re-admission, re-operation, and infection).

All the operations were done mainly by one senior consultant. Service patients were operated on by the most senior resident under the guidance of the same senior consultant. Post-operative hearing loss was not formally tested routinely, but it was consistently asked on the follow-up records of all the patients included in the study. A formal Pure-tone average (PTA) test was done if the patient noted subjective changes in hearing post-operatively. The grading scale used for the pre-and post-operative HFS grade was developed by Hyun et al. in 2010[22] and has since been utilized in other literature on HFS[23–25]. The House-Brackmann grading was used to assess facial palsy, and the WHO classification for hearing loss was used. Permanent facial nerve palsy was defined as no improvement or resolution of symptoms despite more than a year of intensive rehabilitation. Permanent hearing loss after surgery was defined as objective PTA-ST findings of hearing below normal levels accompanied by subjective complaints of hearing problems after surgery that did not improve after one year post-operatively.

Operative Procedure

The surgery performed here is reminiscent of the standard MVD technique first published by Jannetta.[26] No neurophysiologic monitoring was used for any of the cases done. The patient on three-point head fixation is positioned supine with the neck slightly flexed with a shoulder roll on the ipsilateral side of the spasms or in a park bench position. The retromastoid area must be at the highest point of the surgical field. A linear incision over the projected intersection of the transverse sigmoid junction is done. After a thumb-sized craniotomy (2 x 2.5 cm), a linear dural incision 2-3 mm lateral to the border of the sigmoid sinus is done then the free edge is retracted up with sutures. CSF is drained via a lumbar drain inserted on induction or from the lateral pontine and medullary cisterns. The anatomy is defined, and generous, sharp arachnoid dissection must be accomplished. We do not use any form of fixed retraction system on the cerebellum as it may cause traction injury to the 8th nerve and subsequent hearing loss.[27,28] Correct positioning, thorough arachnoid dissection, and liberal CSF drainage open this surgery's corridor. Once the offending vessel and contact point/s with the facial nerve are identified (at the level of the REZ), the appropriate size of Teflon is estimated and cut. We fluff the Teflon pad by pulling the fibers apart to make its tendrils stick out. Then the artery, not the nerve, is mobilized and gently pushed away from the nerve bundle with a micro-Penfield. The Teflon pad is then insinuated between the vessel and the nerve bundle. Afterward, the area is inspected, ensuring no vessels or perforators are kinked. The field is flooded with warm saline, and watertight dural closure is done. Mastoid air cells opened intra-operatively are waxed down. The bone flap from the craniotomy is laid over the

dura, and meticulous muscle closure ensures that the bone flap is maintained in place, followed by layered closure.

Data Analysis

Continuous variables are presented as means or medians with their corresponding standard deviation or range, while categorical variables are presented as proportions. Comparing outcomes pre-, immediately post-operative, and on follow-up was done using repeated measures ANOVA with post hoc analysis. The paired t-test compared the results pre- and immediately post-operatively.

Using the different clinical factors listed: age, sex, presence of hypertension, smoking, number of co-morbidities, years of spasm, number of years before surgery, presence of pre-operative pharmacologic treatment, use of Botox, and location of spasm were tested to determine if they can predict the pre-operative HFS grade using ordered logistic regression. The rank sum test evaluated whether pre or post-operative Botox treatment impacted HFS grade.

A Kaplan Meier Curve survival analysis was done for the most commonly seen post-operative morbidity. As majority of patients were also under the Pay service (operated on by an experienced neurosurgeon as opposed to a service trainee case), a subgroup analysis of this cohort was also performed.

All comparisons were made with the alpha set at 0.05 and the beta at 0.2. All analysis was done using Microsoft Excel and STATA BE 17.

RESULTS

Patient Characteristics

After thoroughly reviewing the in-patient and out-patient hospital records, we found 34 MVD surgeries for primary HFS. The mean patient age was 46.8, and females comprised 70.6% of the population. The most common co-morbidity was hypertension at 44.1%, and patients had a median of 2 co-morbidities. Most of the patients (88.2%) found out about MVD for HFS through the internet, namely an online (Facebook) support group for HFS, and only four patients were referrals from other doctors. Twenty-five (73.5%) patients had tried at least one type of pharmacologic therapy, with 14 having been on a single medication (Clonazepam=8, Carbamazepine=5, Gabapentin=1), and 10 (29.4%) being on a combination of Clonazepam and Carbamazepine. Twenty-two (64.7%) out of thirty-four patients had tried both Botox and pharmacologic therapy before surgery. Forty-eight percent (12) of the 25 patients on Botox therapy had been using the treatment for over three years. The mean duration in years before operative intervention was 8.3 years (2-17 years). Table 1 summarizes the demographic, clinical characteristics, and intra-operative findings.

The anterior inferior cerebellar artery (AICA) was the most implicated vessel, followed by a combination of both the vertebral artery (VA) and the posterior inferior cerebellar artery (PICA). There were no cases involving venous compression, a combination of the VA, AICA, and PICA, and the AICA and PICA only. Twenty-nine (85%) of the operative techniques mentioned an indentation or a discoloration of the facial nerve at the point of contact with the offending vessel/s.

Table 1. Baseline Characteristics of HFS Patients Admitted in PGH (n=34)

Characteristic (N)	Mean (SD) Median (Range) or Percentage (%)
Age of onset of symptoms	38.5 (11.9)
Age of surgery	46.8 (11.3)
Gender	
Male (10)	29.4%
Female (24)	70.6%
Location of spasms	
Right (13)	38.2%
Left (21)	61.8%
Service admitted	
Charity (8)	23.5%
Pay (26)	76.5%
How patients were referred for surgical intervention	
Referral from other practitioners (4)	11.8%
Via social media, HFS support group (30)	88.2%
Number of Comorbidities	2 (0-6)
Hypertension (15)	44.1%
Smoking (3)	8.8%
Pre-operative HFS grade	4 (2-4)
Grade 2 (2)	5.9%
Grade 3 (10)	29.4%
Grade 4 (22)	64.7%
Pre-operative treatment	
Both Botox therapy and pharmacologic management (22)	64.7%
Proportion of patients on pre-operative Botox treatment (27)	79.4%
Duration of pre-operative Botox use (months)	12 (0-120)
Proportion of patients on pre-operative pharmacologic treatment (28)	82.4%
Number of medications taken for HFS pre-operatively	1 (0-4)
Duration of pharmacologic treatment prior to surgery (months)	6.5 (0-72)
Number of years before surgery	8.3 (4.1)
Offending vessel	
AICA (20)	58.8%
PICA + VA (4)	11.8%
AICA + labyrinthine (3)	8.8%
AICA+ VA (3)	8.8%
PICA (2)	5.9%
VA (2)	5.9%

AICA – anterior inferior cerebellar artery, PICA – posterior inferior cerebellar artery, VA – vertebral artery

Predictors of Pre-op Hemifacial Spasm Grade

Results of the clinical factors, including those subjected to ordered logistic regression, were as follows: years before surgery (OR:7.05, z=2.23, p=0.026) and presence of pre-operative pharmacologic treatment (OR:48, z=2.33, p=0.02) increase the HFS grade to the next worse grade. In contrast, hypertension(OR: 0.006, z=-2.54, p=0.011) decreases pre-operative HFS grade to a lower grade.

Pre-operative Botox therapy does not significantly influence HFS grade pre-operative (3.3 vs. 3.7, z=-0.914, p=0.38) and post-operatively (0.3 vs. 0.5, z=-0.718, p=0.65). The rest of the other clinical factors evaluated are unremarkable.

Post-operative Outcomes

The average post-operative length of stay was 3.9 (1.1) days. The overall success rate for this series is 88.2%. In all patients, the median follow-up duration was 15 months (3-64 months); 58.8% (20) of the population had been followed up for at least one year or more. This series' recurrence rate for HFS is 8.82% (three patients). All intra-operative findings for these three patients were compression from the VA (1) or the VA and the PICA (2). The median months to recurrence was 11 months (10-13 months). Considering the follow-up period, the success rate among patients who followed up for at least one year is 90%.

Twenty-one (61.76%) patients experienced complete relief of HFS symptoms immediately after the surgery. The remaining 12 patients (35.3%) experienced full spasm resolution at a later date—1-30 days post-operatively, with an average resolution at ten days post-operatively. Only one patient never experienced a complete resolution of spasms. His condition initially improved to HFS grade 1 for 13 months and slowly progressed. At this patient's most recent follow-up (46 months post-operatively), he already had grade 3 HFS. Table 2 summarizes the outcomes and complications.

Results for the repeated measures ANOVA, post-op HFS, and follow-up HFS grades were significantly lower than pre-op HFS grades (F=17.46, df=33, p<0.0001). There was no significant difference when follow-up HFS was compared to post-operative HFS (0.5 vs 0.26, p=0.18). (Figure 1).

Complications

No operative deaths, cerebellar or brainstem infarctions, post-operative hematomas, lower cranial nerve injury, or other operative complications were found. Two patients were re-admitted within 30 days post-op. The 1st was due to a pseudomeningocele formation, CSF leak, and subsequent meningitis (no isolated organism on culture). This patient was treated conservatively and, after a course of antibiotics, was discharged well. The second patient was admitted for Herpes Zoster at the L1-L2 level and was sent home after two days with an unremarkable course.

Among the complications, new onset ipsilateral facial palsy was the most common. The median number of days before its occurrence was seven days (Figure 2). By Pre-Op

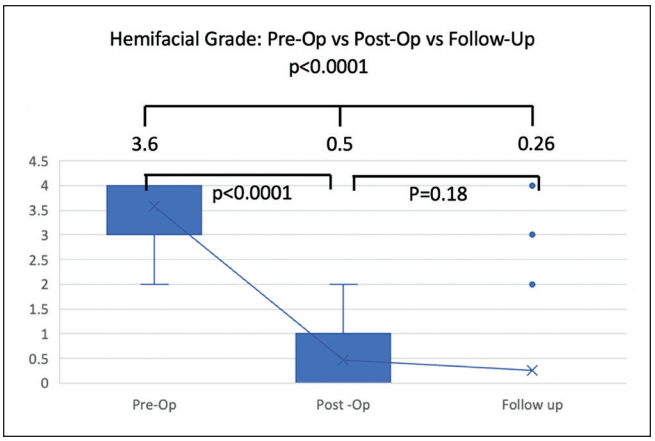

Figure 1. Pre vs. immediate post-operative HFS grade vs. HFS grade at the recent follow-up grade).

Table 2. Summary of Post-operative MVD outcomes in PGH

Characteristic (N)	Mean (SD) Median (Range) or Percentage (%)
Immediate post-op HFS grade	0 (0-2)
No spasms (21)	61.8%
Grade 1 (10)	29.4%
Grade 2 (3)	8.8%
Number of days after surgery until complete spasm resolution	0 (0-33)
Post-operative length of in-hospital stay in days	3.9 (1.1)
Median months to follow-up	15 (3-64)
HFS grade at most recent follow-up	0 (0-4)
Grade 0 (31)	91.3%
Grade 2 (1)	2.9%
Grade 3 (1)	2.9%
Grade 4 (1)	2.9%
Operative Morbidity	
Recurrence (3)	8.8%
Median months to recurrence	11 (10-13)
New onset facial weakness (10)	29.4%
Median days to new onset of facial weakness post op	7 (0-14)
Median grade of facial weakness	4 (3-5)
Median weeks till resolution of facial weakness	6 (3-24)
Permanent facial weakness (2)	5.9%
New onset unilateral hearing loss (6)	17.6%
Median days to unilateral hearing loss since post op	0.5 (0-7)
Median grade of hearing loss	1 (1-3)
Median weeks till resolution of hearing loss	2 (2)
Permanent hearing loss (2)	5.9%
Proportion of patients who developed CSF leak or pseudomeningocele formation (1)	2.9%
Post-operative infection (1)	2.9%
Re-admission rate (2)	5.9%
Combined permanent deficit rate 1 year after surgery (4)	11.8%

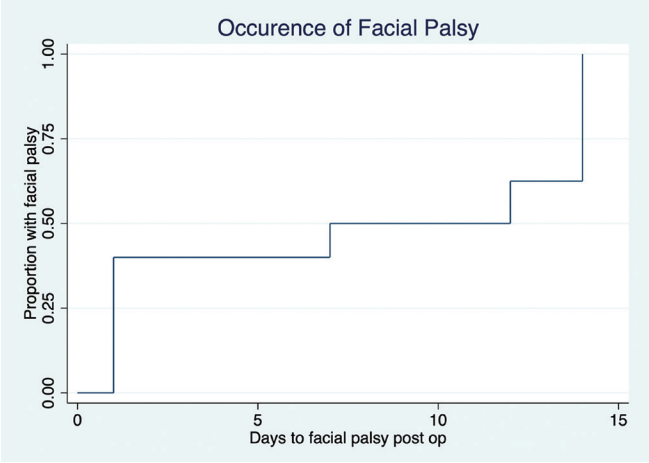

Figure 2. Kaplan Meier Curve for the occurrence of facial palsy (overall).

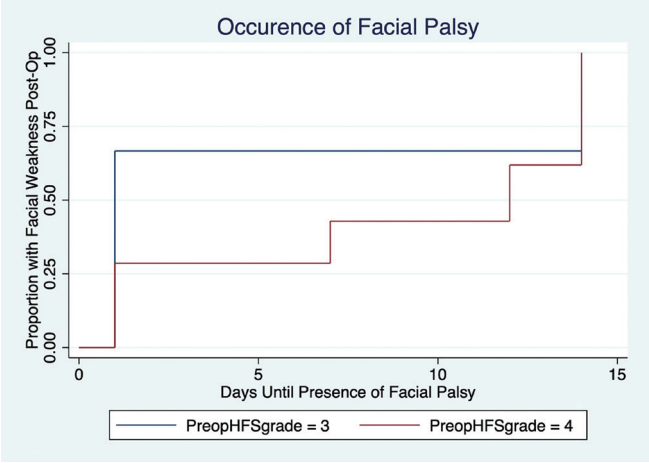

Figure 3. Kaplan Meier Curve for the occurrence of facial palsy (per pre-op HFS).

HFS grade, only patients with grades 3 and 4 developed facial palsy. Although the median number of days until occurrence differs between the two grades (grade 3: 1 day vs. grade 4: 12 days), the difference was not significant (HR: 0.82, Log likelihood= -15.6, p=0.77) (Figure 3). The rate of permanent facial nerve palsy rate was 5.9%. New onset hearing loss was seen at 17.6 %. It resolved spontaneously on an average of two weeks post-operatively, and these patients had PTA-ST findings of mild impairment. The rate of permanent hearing loss was 5.9%. These two patients had PTA-ST findings of moderately severe impairment. The permanent deficit rate for this series is 11.8%. Table 2 summarizes the other post-op complications.

Subgroup Analysis of Pay Cases

Majority (26/34, 76.5%) of patients were under the Pay service. All were operated on by a senior neurosurgeon. The mean age of this subgroup was 45.7 years, and most were female (20/26, 76.9%). Eight patients (8/26, 30.7%) had hypertension, and only three were smokers. Eighteen patients (18/26, 69.2%) had Botox treatment pre-operatively, with a mean lead time to surgery of 36.3 months. The median pre-operative HFS grade was 4. No clinical factors were found to be predictive of pre-operative HFS grade on ordered logistic regression. Intraoperatively, the AICA was the most common offending vessel found (20/26, 76.9%).

The mean length of stay was four days. All patients in this subgroup had improvement of pre-operative HFS grade immediately after surgery. Results of the repeated measures ANOVA comparing pre-, immediately post-, and last follow-up HFS grades mirrored that of the whole cohort, showing significant differences between pre- and immediately post-operative HFS grades ($F=4$, $df=50$, $p<0.0001$), and no significant differences in HFS grades between the two post-operative time periods (0.4 vs 0.3, $p=0.46$). Four patients (15.4%) had delayed facial palsy, all of which were within 30 days from surgery. These all resolved, with no patient with permanent facial nerve weakness. Two patients (7.7%) had delayed ipsilateral hearing loss, which also resolved. Two patients had spasm recurrence during the follow-up period, one of which had worsening back to pre-operative HFS grade (as discussed prior as part of the whole cohort results). The other had spasm recurrence to HFS grade from 1 during the follow-up period, but this was still improved from their pre-operative grade (3). There were no readmissions for CSF leak, pseudomeningocele, nor infection. The patient readmitted for spinal herpes zoster was from this subgroup. Overall, the success rate for this subgroup was 96.2%.

DISCUSSION

We report the baseline demographics, outcomes, and complications of our MVD series for HFS at PGH spanning a 5-year duration and a total of 34 patients. To our knowledge, this is the first published paper on MVD for HFS in the Philippines.

Pre-operative Demographic Data

The demographic profile of the patients in this series is comparable to published literature—HFS is more common in females, [8,22,29–33] and has a 2:1 female-to-male ratio,[2,22,34,35] as seen in this series with a prevalence of females at 70.6% (female-to-male ratio of 2.4:1). It is more commonly unilateral,[29,30,33,36,37] and on the left side (61.8%).[29,33]

The mean age of onset was 38.5 years old, with 11.8% being 30 years old or younger. This is much less than the known average age of onset at 44-59 years old.[2,8,30,33,36] In the literature, the reported prevalence of HFS in patients ≤30 years old is 1-6%.[2,31] The previously proposed mechanisms in younger individuals are arachnoid membrane thickening in the cerebellopontine angle cistern or compression of the nerve from a venous anomaly.[35] However, three-dimensional MRI volumetric studies for posterior fossa CSF space

in patients with HFS noted a mean difference of 1,913 mm^3,[35] and is 11.8% smaller versus matched controls[34]. Further multivariate regression analysis demonstrated that younger age and the female gender are associated with a smaller posterior fossa CSF space.[35] This crowding in the posterior fossa may better explain the etiology of the vascular compression at the REZ in younger patients and its female predominance.[29,34,35] These findings have also been observed among Asian populations.[7,38] Asian skulls tend to be more brachycephalic and may predispose to HFS.[38,39] In a study by Kamiguchi et al., the petrous angle of Japanese patients with HFS was significantly smaller than controls.[38]

No bilateral cases were seen in this study, but the reported prevalence is at 0.2-5.7%.[8,29] Older age and hypertension are commonly associated with bilateral compared to unilateral HFS.[29]

Hypertension was the most common risk factor (44.1%). The relationship between hypertension and HFS has been investigated, but the results are inconsistent. The reported prevalence is 39% to 67%.[29,40,41] Others recommend a high index of suspicion for non-hypertensive HFS patients and suggest screening them for hypertension.[41] However, multiple pooled meta-analyses done, especially among Asian and Italian populations, do not show a statistically significant difference in the prevalence of hypertension in HFS patients compared to controls.[29,40,42] What these studies did note is that hypertensive patients with HFS showed a statistically significant association with a higher degree of ventrolateral medullary compression,[29,40,42,43] and has been seen to be more frequent in the population of bilateral HFS.[40,42,43] Another study even regarded ventrolateral medullary compression as contributory to hypertension and reported that after MVD, patients have better blood pressure control.[42]

Interestingly, we found that hypertension is associated with a lower HFS grade (odds ratio of 0.006). Grade 3 HFS patients comprised 10 of the 15 patients. Na et al. noted that hypertension was associated with more severe HFS but on the analysis of their data, they pooled HFS grades 3 and 4 together as severe HFS.[44] On a comprehensive search, we could not find other studies correlating HFS grade and hypertension. Of the hypertensive patients in our series, 11 had more than one co-morbidity. We hypothesize that given these multiple diagnoses, they were more likely to have been seen by a specialist where a subsequent diagnosis of HFS could have been made earlier hence the lower HFS grade. A more extensive study may be needed to confirm this hypothesis because misdiagnosis is frequent, especially in the primary care setting.[45,46] The mean time from the first symptom to HFS diagnosis was 2.64±3.8 years.[46] Another study in Canada noted that patients had to consult an average of 3.2 physicians before the correct diagnosis was made.[45]

There were no published studies regarding the referral system for MVD since a large proportion of our patients (88.2%) learned about MVD from social media (HFS online support group). Consequently, Lawrence et al. published a quality-of-life study for patients who underwent MVD, which noted that difficulty in obtaining a referral for surgery adversely affected, to very adversely affected their quality of life in 76% percent of patients.[25] Misdiagnosis and delay in referral may have contributed to the mean of 8.3 years before operative intervention in this series and higher pre-operative HFS grade (OR:7.05).

Duration of symptoms before surgery and pre-operative pharmacologic treatment were associated with a higher pre-operative HFS grade. Association between duration and severity of HFS has been observed,[8,30,44] and the definition of the clinical features of the disease includes its progressive nature[47]. One study reported the spread of muscle spasms as high as 94.1% over a mean disease duration of 10.8 years.[48] In contrast, a prospective study observing the natural history of HFS showed that the disease was either aggravated or stationary in only 49.1%, with spontaneous remission as high as 41.3% with a mean follow-up period of 12 years.[23] Prospective studies with strict patient selection are needed to clarify this question better. We found no literature on pre-operative pharmacologic therapy and HFS grade.

Intra-operative Findings

The most commonly isolated blood vessel was the AICA (58.8%); in multiple studies, the prevalence is 53.2-54.63%.[22,37] With regard to the association with morbidity, 10 out of 11 cases that developed facial palsy, both transient and permanent, were compression from the AICA, and all three recurrences in this series were due to compression from the VA alone or the VA and the PICA, however, the study is not powered enough to achieve any conclusions. The VA has been documented to produce poorer outcomes. The success rate in one series was only 77.2% (immediately post-op) for VA compression, and 6.3% of these patients had spasm progression six months post-operatively.[49] Others classify intra-operative findings more from the pattern of compression than from the offending artery.[22] A tandem type of compression has a strong predilection for VA involvement, and even preoperatively, the surgeon must be prepared to deal with two vessels instead of just one.[22] Another school of thought is that of the novel macrovascular decompression for vertebrobasilar compression, via a far lateral approach, using a Muslin or Gore-Tex sling transposition graft to pull the dolichoectatic vertebrobasilar artery towards an anteromedial direction, away from the nerve complex then the sling is anchored on the dura with an aneurysm clip. Choudhri et al. report a success rate of 100% with a mean follow-up of 6.6 years. The complications were transient hoarseness, diplopia, and dural sinus thrombosis, which resolved spontaneously. [50]

Outcomes

HFS Grade

Despite the absence of intra-operative monitoring, post-operative HFS grades and follow-up HFS grades

were significantly lower than pre-operative HFS grades ($F=17.46$, $df=33$, $p<0.0001$). These findings further support the growing evidence regarding the effectiveness of MVD for HFS. This series' overall success rate is 88.2%, comparable to internationally published extensive series data that report 82.7-99.6%.[22,44,51–54] We have no mortalities, but it is published at 0.3%.[55]

The dilemma in comparing outcomes between studies is the absence of a unified scoring system for HFS.[39] Successful outcomes are also defined differently, as some studies treat improvement as cure.[14,29,44,49,56] The possibility of delayed cure/resolution also confounds these rates.[52,57] Before labeling the surgery a failure, others prefer to watch and wait, as the documented resolution may occur even at eight months[52] to three years[57] post-operatively. In an interesting study by Kondo et al., they reported the discrepancy between the reported cure of the surgeons at 95% versus the patients themselves, that rated cure at only 81%. The complication rate reported by the surgeons was also lower at 10.3%, while their patients reported it as 14.1%.[39] This series only considered complete spasm resolution, but our follow-up period is limited, with a median of 15 months. The recurrence rate in this series is 8.8%. It is higher than the published rates at 2.4-9.5%.[24,58,59]

The dilemma of when to re-operate has been debated. Many advocate re-operation early, (within 20 days after the surgery) but warn against the higher risk of craniopathies even with monitoring.[60,61] Besides timing, age should also be considered; as younger patients (<50 y/o) seem to tolerate repeat surgeries better.[61] Re-exploration is not without risk. The series by Kureshi et al. reported a 37.5% negative exploration rate for persistent or recurrent HFS and a 75% complication rate. They concluded that they do not recommend repeat surgery.[60]

Others have devised better patient selection criteria to choose patients for surgery—pre re-operative MRI 3-dimensional time of flight angiography showing the presence of neurovascular contact showed a statistically significant favorable outcome with success rates comparable with virgin MVDs at 92.3%.[62] Two other papers reported doing a facial electromyography.[24,61] They will offer surgery if abnormal muscle response is substantial (>20% from baseline). One series reported a 0% negative exploration rate,[61] but both report a higher permanent facial palsy rate at 15.4-28.7%.[24,61] Regardless, most recommend re-operation after one year if without resolution or progression of symptoms, with MRI findings of vascular compression,[3,22,52,62,63] and Hatayama et al. even required reviewing pre-operative videos to lend a better outcome.[51]

The effect of surgical experience was also seen in our paper, with the subgroup of patients operated on by a more senior neurosurgeon showing improved surgical success, and HFS grades, with minimal morbidity. The importance of proper surgical technique and intraoperative decision-making when performing MVD cannot be underscored.

Facial Nerve Palsy

Among the complications seen, 29.4% had new onset ipsilateral facial palsy. It was the most common complication and is higher than other published series (2.7-22.5%).[14,37,44,64] Only patients with HFS grades 3 and 4 developed facial palsy with a median day of occurrence at seven days post-operative, but no statistical significance was seen between the two. Other studies have shown a statistically significant association between the severity of HFS pre-operatively and the occurrence of facial palsy.[30,44]

Delayed facial palsy (DFP) occurred 7-14 days from surgery and resolved entirely by 24 weeks post-operatively except for two out of the thirty-four patients. The course is similar to other academic papers, with onset at 7-10 days after surgery and resolution at 2-3 months post-operatively, regardless of the severity of the House-Brackmann grade.[37,65] The permanent facial palsy rate in this series was higher at 5.9% than other reports at 1.2-2.8%.[56,64] These two patients presented with grade 4-5 facial palsy immediately after surgery. Despite extensive rehabilitation, they have only recovered to grades 2 and 4 (>12 months post-operatively). These results agree with the study conclusions by Huh et al. that stated—the earlier and more severe the degree of facial palsy, the more permanent the deficit remained.[64] Immediate facial palsy (occurs within 24 hours post-operatively) is likely due to direct injury or heat from coagulating close to the nerve, or compression or angulation of the Teflon pad.[58] The mechanism of DFP remains unclear. Some attribute it to direct trauma to the nerve,[18] but that may not explain why it occurs 7-10 days after surgery. Re-activation of varicella zoster,[65,66] or herpes simplex virus (HSV) dormant at the geniculate ganglion is another theory.[10,18,65] However, Han et al. did serologic HSV testing for all their patients pre-operatively, and none of the patients positive for HSV developed DFP.[67] Hengstman et al. documented two patients with DFP whose CSF was positive for HSV on polymerase chain reaction.[68] Major surgery itself is an immunosuppressant event and may contribute to viral reactivation. More studies are needed to elucidate these theories.[65,68]

Unplanned Re-admission, CSF Leak, and Surgical Site Infection

Our unplanned re-admission rate was 5.9%, similar to the re-admission rate of Cote et al. at 6.8%.[55] One patient was re-admitted due to pseudomeningocele formation, CSF leak (2.9%), and subsequent meningitis (2.9%). She was treated conservatively and, after a course of antibiotics, was discharged well. No organisms were grown on CSF culture. In another paper, surgical site infection was the most common reason for re-admission.[55] CSF leak rate post-operatively is 0.2-12%.[10,14,22,32,57] The rate of surgical site infection post-MVD is 0.93% to 2.6%.[37,55,61]

The second patient was admitted for herpes zoster at the L1-L2 level (and no associated DFP). He was sent home after two days with an unremarkable course.

Hearing Loss

As with facial palsy, it can be classified as temporary and permanent. Our transient hearing loss prevalence was 17.6%, which was permanent in two patients (5.9%). Some scholars reported that hearing loss was present (1.9-20%),[3,37] and 0.5-2.3%[3,57] were permanent. Unlike facial palsy after MVD, this complication presents readily within 24 hours post-operatively. In our series, recovery took two weeks. Theories on the mechanisms for this phenomenon are similar to immediate facial palsy—direct nerve injury from manipulation, compression from the Teflon pad, and heat injury from coagulation.[3,58] Another possibility is stretching the VII-VIII nerve complex from excessive cerebellar retraction.[28]

Other possible reported complications we did not demonstrate in this series were post-operative hemorrhage, cerebellar bleeding or infarction, diplopia, lower cranial nerve deficits (hoarseness and dysphagia), re-operation, vertebral artery injury or sacrifice that can subsequently lead to a lateral medullary syndrome and death.

Impact of Social Media Use on MVD for HFS

Our study has also elucidated on the impact of social media on acceptance of MVD as a treatment for HFS. Majority (88.2%) of patients in this series sought neurosurgical treatment via a social media support group for HFS patients. The use of this group is twofold: for patients and for physicians. On the one hand, patients have used the group for community building and empowerment, allowing for offhand treatment advice from fellow sufferers of HFS as well as shared psychosocial support. In a socio-cultural perspective, there is still disease stigma for sufferers of HFS, and having a social media group allows for acceptance and alleviation of the stigma, enabling patients to seek more timely physician consultations. This further broadens the treatment paradigms to not just medications or adjunctive treatments, but to more invasive surgical management such as MVD; which has been illustrated in our paper. On the other hand, physicians may use this social media group to promulgate patient education directives, allowing for a credible information source. Through this engagement, a more streamlined referral system may be created, which will lead patients open to surgical treatment to proper channels for neurosurgical consultation. With the clinical results of this paper, the use of social media may then in turn increase those who avail of neurosurgical treatment of HFS, lessening disease sufferers.

Limitations

There are several limitations to this study. This is a single-center retrospective evaluation in a tertiary referral hospital that does about 1500 surgeries yearly; results may not be generalizable to all institutions. Another limitation is that we did not determine temporal relationships of pharmacologic and Botox treatment to surgical management. These adjunctive treatments have varying times to action which may influence effectiveness of surgery. Future studies are recommended to determine temporal relationships between treatments types to determine the exact effects of each modality.

The population size, follow-up duration, and study design are also significant limitations. Given that this is retrospective, the data in the charts are less complete than we would have wanted. Population-based epidemiologic studies can help clarify the link between earlier age of onset and the prevalence of HFS in Filipino patients. Larger prospective control studies with a more extended follow-up period with data collected pre- and post-operatively on CSF posterior fossa volume, hearing tests, quality of life assessments, as well as intra-operative findings—the type of vascular compression and grade of facial nerve compression, would help better describe the demographics of HFS and better evaluate outcomes for MVD and Botox therapy in the Philippines.

CONCLUSION

The series consisted of homogenously Filipinos with a mean age of onset at 38.5 years (younger than published data) and a 2.4:1 female-to-male ratio. Most referrals come from an online hemifacial support group (88.2%) rather than from other doctors. Duration before surgery and pre-operative medical management is associated with the worst pre-operative HFS scores. Hypertension was the most prevalent co-morbidity and had a negative odds ratio for pre-operative HFS severity. The recurrence rate was 8.8%, for which all cases involved VA compression. Permanent facial nerve palsy and permanent hearing loss were both at 5.9%. Microvascular decompression for HFS at PGH has an overall success rate of 88.2%, comparable to internationally published rates. It should give us the confidence to offer MVD as a viable option to offer our HFS patients in the Philippines. In addition, the data for the surgical morbidities in this study should be a driver to improve the outcomes further.

Statement of Authorship

All authors certified fulfillment of ICMJE authorship criteria.

Author Disclosure

All authors declared no conflicts of interest.

Funding Source

None.

REFERENCES

1. Herrero-Infante Y, Rodríguez-Sanz A, Máñez-Miró J, Vivancos-Matellano F. Hemifacial spasm through the last three decades: From etiology to efficacy and safety of long-term botulinum toxin treatment. Clin Neurol Neurosurg. 2021 Apr;203:106555. doi: 10.1016/j.clineuro.2021.106555. Epub 2021 Feb 10. PMID: 33662742.
2. Chaudhry N, Srivastava A, Joshi L. Hemifacial spasm: The past, present and future. J Neurol Sci. 2015 Sep 15;356(1-2):27-31. doi: 10.1016/j.jns.2015.06.032. Epub 2015 Jun 16. PMID: 26111430.

3. Sindou M, Mercier P. Microvascular decompression for hemifacial spasm: Surgical techniques and intraoperative monitoring. Neurochirurgie. 2018 May;64(2):133-143. doi: 10.1016/j.neuchi.2018.04.003. Epub 2018 May 18. PMID: 29784430.
4. Yaltho TC, Jankovic J. The many faces of hemifacial spasm: differential diagnosis of unilateral facial spasms. Mov Disord. 2011 Aug 1;26(9):1582-92. doi: 10.1002/mds.23692. Epub 2011 Apr 5. PMID: 21469208.
5. Bennett MV. Gap junctions as electrical synapses. J Neurocytol. 1997 Jun;26(6):349-66. doi: 10.1023/a:1018560803261. PMID: 9278865.
6. Rosenstengel C, Matthes M, Baldauf J, Fleck S, Schroeder H. Hemifacial spasm: conservative and surgical treatment options. Dtsch Arztebl Int. 2012 Oct;109(41):667-73. doi: 10.3238/arztebl.2012.0667. Epub 2012 Oct 12. PMID: 23264807; PMCID: PMC3487151.
7. Wu Y, Davidson AL, Pan T, Jankovic J. Asian over-representation among patients with hemifacial spasm compared to patients with cranial-cervical dystonia. J Neurol Sci. 2010 Nov 15;298(1-2):61-3. doi: 10.1016/j.jns.2010.08.017. PMID: 20864122.
8. Wang A, Jankovic J. Hemifacial spasm: clinical findings and treatment. Muscle Nerve. 1998 Dec;21(12):1740-7. doi: 10.1002/(sici)1097-4598(199812)21:12<1740::aid-mus17>3.0.co;2-v. PMID: 9843077.
9. Lee MH, Jee TK, Lee JA, Park K. Postoperative complications of microvascular decompression for hemifacial spasm: lessons from experience of 2040 cases. Neurosurg Rev. 2016 Jan;39(1):151-8; discussion 158. doi: 10.1007/s10143-015-0666-7. Epub 2015 Sep 18. PMID: 26382646.
10. Samii M, Günther 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. 2002 Apr;50(4):712-8; discussion 718-9. doi: 10.1097/00006123-200204000-00005. PMID: 11904020.
11. Barker FG 2nd, Jannetta PJ, Bissonette DJ, Shields PT, Larkins MV, Jho HD. Microvascular decompression for hemifacial spasm. J Neurosurg. 1995 Feb;82(2):201-10. doi: 10.3171/jns.1995.82.2.0201. PMID: 7815147.
12. Auger RG, Piepgras DG, Laws ER Jr. Hemifacial spasm: results of microvascular decompression of the facial nerve in 54 patients. Mayo Clin Proc. 1986 Aug;61(8):640-4. doi: 10.1016/s0025-6196(12)62028-x. PMID: 3724243.
13. Kaufmann AM, Price AV. A history of the Jannetta procedure. J Neurosurg. 2019 Feb 1;132(2):639-646. doi: 10.3171/2018.10.JNS181983. PMID: 30717044.
14. 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. 2008 Sep;109(3):410-5. doi: 10.3171/JNS/2008/109/9/0410. PMID: 18759569.
15. Heuser K, Kerty E, Eide PK, Cvancarova M, Dietrichs E. Microvascular decompression for hemifacial spasm: postoperative neurologic follow-up and evaluation of life quality. Eur J Neurol. 2007 Mar;14(3):335-40. doi: 10.1111/j.1468-1331.2006.01670.x. PMID: 17355557.
16. Ray DK, Bahgat D, McCartney S, Burchiel KJ. Surgical outcome and improvement in quality of life after microvascular decompression for hemifacial spasms: a case series assessment using a validated disease-specific scale. Stereotact Funct Neurosurg. 2010;88(6):383-9. doi: 10.1159/000319883. Epub 2010 Oct 15. PMID: 20948243.
17. Hanakita J, Kondo A. Serious complications of microvascular decompression operations for trigeminal neuralgia and hemifacial spasm. Neurosurgery. 1988 Feb;22(2):348-52. doi: 10.1227/00006123-198802000-00012. PMID: 3352885.
18. Lovely TJ, Getch CC, Jannetta PJ. Delayed facial weakness after microvascular decompression of cranial nerve VII. Surg Neurol. 1998 Nov;50(5):449-52. doi: 10.1016/s0090-3019(97)00314-5. PMID: 9842870.
19. Compagnon C, Labrousse M, Brenet E, Chays A, Bazin A, Kleiber JC, Dubernard X. Efficacy and Complications of Microsurgical Neurovascular Decompression in 55 Patients With Hemifacial Spasm. Otolaryngol Head Neck Surg. 2021 Jun;164(6):1299-1306. doi: 10.1177/0194599820969168. Epub 2020 Nov 17. PMID: 33198570.
20. Nugroho SW, Perkasa SAH, Gunawan K, Manuhutu YN, Rahman MA, Rizky A. Predicting outcome of hemifacial spasm after microvascular decompression with intraoperative monitoring: A systematic review. Heliyon. 2021 Feb 2;7(2):e06115. doi: 10.1016/j.heliyon.2021.e06115. PMID: 33644443; PMCID: PMC7889992.
21. Liu M, Xia L, Zhong J, Li B, Dou N, Li S. A comparative study of intraoperative findings and postoperative outcomes between atypical and typical hemifacial spasms. Neurosurg Rev. 2018 Apr;41(2):593-597. doi: 10.1007/s10143-017-0898-9. Epub 2017 Aug 22. PMID: 28831592.
22. Hyun SJ, Kong DS, Park K. Microvascular decompression for treating hemifacial spasm: lessons learned from a prospective study of 1,174 operations. Neurosurg Rev. 2010 Jul;33(3):325-34; discussion 334. doi: 10.1007/s10143-010-0254-9. Epub 2010 Mar 27. PMID: 20349099.
23. Lee JA, Kim KH, Park K. Natural History of Untreated Hemifacial Spasm: A Study of 104 Consecutive Patients over 5 Years. Stereotact Funct Neurosurg. 2017;95(1):21-25. doi: 10.1159/000453276. Epub 2017 Jan 14. PMID: 28088801.
24. Li S, Hong W, Tang Y, Ying T, Zhang W, Li X, Zhu J, Zhong J, Hua X, Xu S, Wan L, Wang X, Yang M, Li Y, Zheng X. Re-operation for persistent hemifacial spasm after microvascular decompression with the aid of intraoperative monitoring of abnormal muscle response. Acta Neurochir (Wien). 2010 Dec;152(12):2113-8. doi: 10.1007/s00701-010-0837-9. Epub 2010 Oct 10. PMID: 20936485.
25. Lawrence JD, Frederickson AM, Chang YF, Weiss PM, Gerszten PC, Sekula RF. An investigation into quality of life improvement in patients undergoing microvascular decompression for hemifacial spasm. J Neurosurg. 2018 Jan;128(1):193-201. doi: 10.3171/2016.9.JNS161022. Epub 2017 Feb 10. PMID: 28186450.
26. Jannetta PJ, McLaughlin MR, Casey KF. Technique of microvascular decompression. Technical note. Neurosurg Focus. 2005 May 15;18(5):E5. PMID: 15913281.
27. Amagasaki K, Watanabe S, Naemura K, Nakaguchi H. Microvascular decompression for hemifacial spasm: how can we protect auditory function? Br J Neurosurg. 2015 Jun;29(3):347-52. doi: 10.3109/02688697.2014.1003033. Epub 2015 Feb 9. PMID: 25665598.
28. Lee MH, Lee HS, Jee TK, Jo KI, Kong DS, Lee JA, Park K. Cerebellar retraction and hearing loss after microvascular decompression for hemifacial spasm. Acta Neurochir (Wien). 2015 Feb;157(2):337-43. doi: 10.1007/s00701-014-2301-8. Epub 2014 Dec 18. PMID: 25514867.
29. Kobata H, Kondo A, Iwasaki K, Nishioka T. Combined hyperactive dysfunction syndrome of the cranial nerves: trigeminal neuralgia, hemifacial spasm, and glossopharyngeal neuralgia: 11-year experience and review. Neurosurgery. 1998 Dec;43(6):1351-61; discussion 1361-2. doi: 10.1097/00006123-199812000-00052. PMID: 9848849.
30. Batla A, Goyal C, Shukla G, Goyal V, Srivastava A, Behari M. Hemifacial spasm: clinical characteristics of 321 Indian patients. J Neurol. 2012 Aug;259(8):1561-5. doi: 10.1007/s00415-011-6376-3. Epub 2012 Jan 6. PMID: 22222858.
31. Tan EK, Chan LL. Young onset hemifacial spasm. Acta Neurol Scand. 2006 Jul;114(1):59-62. doi: 10.1111/j.1600-0404.2006.00650.x. PMID: 16774629.
32. McLaughlin MR, Jannetta PJ, Clyde BL, Subach BR, Comey CH, Resnick DK. Microvascular decompression of cranial nerves: lessons learned after 4400 operations. J Neurosurg. 1999 Jan;90(1):1-8. doi: 10.3171/jns.1999.90.1.0001. PMID: 10413149.
33. 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. 2006 Mar;63(3):441-4. doi: 10.1001/archneur.63.3.441. Erratum in: Arch Neurol. 2006 Sep;63(9):1241. Avanzino, Lucio [corrected to Avanzino, Laura]; Marinelli, Laura [corrected to Marinelli, Lucio]. PMID: 16533973.

34. Cheng J, Fang Y, Zhang H, Lei D, Wu W, You C, Mao B, Mao K. Quantitative Study of Posterior Fossa Crowdedness in Hemifacial Spasm. World Neurosurg. 2015 Oct;84(4):920-6. doi: 10.1016/j.wneu.2015.04.040. Epub 2015 Sep 1. PMID: 26341431.
35. Chan LL, Ng KM, Fook-Chong S, Lo YL, Tan EK. Three-dimensional MR volumetric analysis of the posterior fossa CSF space in hemifacial spasm. Neurology. 2009 Sep 29;73(13):1054-7. doi: 10.1212/WNL.0b013e3181b9c8ce. PMID: 19786697; PMCID: PMC2754328.
36. Lu AY, Yeung JT, Gerrard JL, Michaelides EM, Sekula RF Jr, Bulsara KR. Hemifacial spasm and neurovascular compression. ScientificWorldJournal. 2014;2014:349319. doi: 10.1155/2014/349319. Epub 2014 Oct 28. PMID: 25405219; PMCID: PMC4227371.
37. Li F, Liu R. Clinical analysis of microvascular decompression in patients with hemifacial spasm: a retrospective study. Ann Palliat Med. 2020 Mar;9(2):318-323. doi: 10.21037/apm.2020.01.11. Epub 2020 Feb 25. PMID: 32156128.
38. Kamiguchi H, Ohira T, Ochiai M, Kawase T. Computed tomographic analysis of hemifacial spasm: narrowing of the posterior fossa as a possible facilitating factor for neurovascular compression. J Neurol Neurosurg Psychiatry. 1997 May;62(5):532-4. doi: 10.1136/jnnp.62.5.532. PMID: 9153617; PMCID: PMC486879.
39. Kondo A, Date I, Endo S, Fujii K, Fujii Y, Fujimaki T, Hasegawa M, Hatayama T, Hongo K, Inoue T, Ishikawa M, Ito M, Kayama T, Kohmura E, Matsushima T, Munemoto S, Nagahiro S, Ohno K, Okamura T, Ryu H, Shigeno T, Shirane R, Tagusagawa Y, Tanabe H, Yamada K, Yamakami I. A proposal for standardized analysis of the results of microvascular decompression for trigeminal neuralgia and hemifacial spasm. Acta Neurochir (Wien). 2012 May;154(5):773-8. doi: 10.1007/s00701-012-1277-5. Epub 2012 Feb 11. PMID: 22327325.
40. Defazio G, Martino D, Aniello MS, Masi G, Logroscino G, Manobianca G, La Stilla M, Livrea P. Influence of age on the association between primary hemifacial spasm and arterial hypertension. J Neurol Neurosurg Psychiatry. 2003 Jul;74(7):979-81. doi: 10.1136/jnnp.74.7.979. PMID: 12810800; PMCID: PMC1738545.
41. Leong JL, Li HH, Chan LL, Tan EK. Revisiting the link between hypertension and hemifacial spasm. Sci Rep. 2016 Feb 19;6:21082. doi: 10.1038/srep21082. PMID: 26891766; PMCID: PMC4759578.
42. Rudzińska M, Wójcik-Pędziwiatr M, Malec-Litwinowicz M, Grabska N, Hartel M, Flak M, Szczudlik A. Is hypertension a risk factor of hemifacial spasm? Neurol Neurochir Pol. 2016;50(2):69-74. doi: 10.1016/j.pjnns.2015.11.002. Epub 2015 Nov 25. PMID: 26969561.
43. Oliveira LD, Cardoso F, Vargas AP. Hemifacial spasm and arterial hypertension. Mov Disord. 1999 Sep;14(5):832-5. doi: 10.1002/1531-8257(199909)14:5<832::aid-mds1017>3.0.co;2-7. PMID: 10495046.
44. Na BS, Cho JW, Park K, Kwon S, Kim YS, Kim JS, Youn J. Severe Hemifacial Spasm is a Predictor of Severe Indentation and Facial Palsy after Microdecompression Surgery. J Clin Neurol. 2018 Jul;14(3):303-309. doi: 10.3988/jcn.2018.14.3.303. Epub 2018 Apr 27. PMID: 29856152; PMCID: PMC6031990.
45. Jog M, Chouinard S, Hobson D, Grimes D, Chen R, Bhogal M, Simonyi S. Causes for treatment delays in dystonia and hemifacial spasm: a Canadian survey. Can J Neurol Sci. 2011 Sep;38(5):704-11. doi: 10.1017/s0317167100012270. PMID: 21856572.
46. Martinez AR, Nunes MB, Immich ND, Piovesana L, França M Jr, Campos LS, D'Abreu A. Misdiagnosis of hemifacial spasm is a frequent event in the primary care setting. Arq Neuropsiquiatr. 2014 Feb;72(2):119-22. doi: 10.1590/0004-282X20130227. PMID: 24604365.
47. Abbruzzese G, Berardelli A, Defazio G. Hyperkinectic Disorders. In: Weiner W, Tolosa E, eds. Handbook of Clinical Neurology Vol. 100, 3rd ed. Amsterdam: Elsevier B.V., 2011. pp. 675-680.
48. Conte A, Falla M, Diana MC, Bologna M, Suppa A, Fabbrini A, Colosimo C, Berardelli A, Fabbrini G. Spread of Muscle Spasms in Hemifacial Spasm. Mov Disord Clin Pract. 2014 Nov 4;2(1):53-55. doi: 10.1002/mdc3.12106. PMID: 30363856; PMCID: PMC6183182.
49. Kim JP, Park BJ, Choi SK, Rhee BA, Lim YJ. Microvascular decompression for hemifacial spasm associated with vertebrobasilar artery. J Korean Neurosurg Soc. 2008 Sep;44(3):131-5. doi: 10.3340/jkns.2008.44.3.131. Epub 2008 Sep 30. PMID: 19096662; PMCID: PMC2588300.
50. Choudhri O, Connolly ID, Lawton MT. Macrovascular Decompression of the Brainstem and Cranial Nerves: Evolution of an Anteromedial Vertebrobasilar Artery Transposition Technique. Neurosurgery. 2017 Aug 1;81(2):367-376. doi: 10.1093/neuros/nyx110. PMID: 28402528.
51. Hatayama T, Kono T, Harada Y, Yamashita K, Utsunomiya T, Hayashi M, Nakajima H, Hatanaka R, Shimada 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). 2015;55(8):663-8. doi: 10.2176/nmc.ra.2014-0386. Epub 2015 Jul 28. PMID: 26226977; PMCID: PMC4628157.
52. Ishikawa M, Nakanishi T, Takamiya Y, Namiki J. Delayed resolution of residual hemifacial spasm after microvascular decompression operations. Neurosurgery. 2001 Oct;49(4):847-54; discussion 854-6. doi: 10.1097/00006123-200110000-00013. PMID: 11564245.
53. Li CS. Varied patterns of postoperative course of disappearance of hemifacial spasm after microvascular decompression. Acta Neurochir (Wien). 2005 Jun;147(6):617-20; discussion 620. doi: 10.1007/s00701-005-0492-8. Epub 2005 Apr 7. PMID: 15806333.
54. Kong DS, Park K, Shin BG, Lee JA, Eum DO. Prognostic value of the lateral spread response for intraoperative electromyography monitoring of the facial musculature during microvascular decompression for hemifacial spasm. J Neurosurg. 2007 Mar;106(3):384-7. doi: 10.3171/jns.2007.106.3.384. PMID: 17367059.
55. Cote DJ, Dasenbrock HH, Gormley WB, Smith TR, Dunn IF. Adverse Events After Microvascular Decompression: A National Surgical Quality Improvement Program Analysis. World Neurosurg. 2019 Aug;128:e884-e894. doi: 10.1016/j.wneu.2019.05.022. Epub 2019 May 11. PMID: 31082546; PMCID: PMC6825526.
56. Zhao H, Zhang X, Tang YD, Zhang Y, Ying TT, Zhu J, Li ST. Operative Complications of Microvascular Decompression for Hemifacial Spasm: Experience of 1548 Cases. World Neurosurg. 2017 Nov;107:559-564. doi: 10.1016/j.wneu.2017.08.028. Epub 2017 Aug 18. PMID: 28823667.
57. Jo KW, Kong DS, Park K. Microvascular decompression for hemifacial spasm: long-term outcome and prognostic factors, with emphasis on delayed cure. Neurosurg Rev. 2013 Apr;36(2):297-301; discussion 301-2. doi: 10.1007/s10143-012-0420-3. Epub 2012 Sep 2. PMID: 22940822.
58. Miller LE, Miller VM. Safety and effectiveness of microvascular decompression for treatment of hemifacial spasm: a systematic review. Br J Neurosurg. 2012 Aug;26(4):438-44. doi: 10.3109/02688697.2011.641613. Epub 2011 Dec 15. PMID: 22168965.
59. Lee JA, Kim KH, Kong DS, Lee S, Park SK, Park K. Algorithm to Predict the Outcome of Microvascular Decompression for Hemifacial Spasm: A Data-Mining Analysis Using a Decision Tree. World Neurosurg. 2019 May;125:e797-e806. doi: 10.1016/j.wneu.2019.01.172. Epub 2019 Feb 7. PMID: 30738936.
60. Kureshi SA, Wilkins RH. Posterior fossa reexploration for persistent or recurrent trigeminal neuralgia or hemifacial spasm: surgical findings and therapeutic implications. Neurosurgery. 1998 Nov;43(5):1111-7. doi: 10.1097/00006123-199811000-00061. PMID: 9802855.
61. Engh JA, Horowitz M, Burkhart L, Chang YF, Kassam A. Repeat microvascular decompression for hemifacial spasm. J Neurol Neurosurg Psychiatry. 2005 Nov;76(11):1574-80. doi: 10.1136/jnnp.2004.056861. PMID: 16227555; PMCID: PMC1739385.
62. Park CK, Lee SH, Park BJ. Surgical Outcomes of Revision Microvascular Decompression for Persistent or Recurrent Hemifacial Spasm After Surgery: Analysis of Radiologic and Intraoperative Findings. World Neurosurg. 2019 Nov;131:e454-e459. doi: 10.1016/j.wneu.2019.07.191. Epub 2019 Aug 2. PMID: 31382068.

63. Park JS, Kong DS, Lee JA, Park K. Chronologic analysis of symptomatic change following microvascular decompression for hemifacial spasm: value for predicting midterm outcome. Neurosurg Rev. 2008 Oct;31(4):413-8; discussion 418-9. doi: 10.1007/s10143-008-0150-8. Epub 2008 Jul 30. PMID: 18665406.
64. Huh R, Han IB, Moon JY, Chang JW, Chung SS. Microvascular decompression for hemifacial spasm: analyses of operative complications in 1582 consecutive patients. Surg Neurol. 2008 Feb;69(2):153-7; discussion 157. doi: 10.1016/j.surneu.2007.07.027. PMID: 18261641.
65. Liu LX, Zhang CW, Ren PW, Xiang SW, Xu D, Xie XD, Zhang H. Prognosis research of delayed facial palsy after microvascular decompression for hemifacial spasm. Acta Neurochir (Wien). 2016 Feb;158(2):379-85. doi: 10.1007/s00701-015-2652-9. Epub 2015 Dec 11. PMID: 26659255.
66. Lee JH, Kang J, Seo YD, Kim HC, Shin KJ. Delayed Facial Palsy in a Patient With Varicella Zoster Virus Encephalitis. J Clin Neurol. 2021 Oct;17(4):604-606. doi: 10.3988/jcn.2021.17.4.604. PMID: 34595877; PMCID: PMC8490894.
67. Han JS, Lee JA, Kong DS, Park K. Delayed cranial nerve palsy after microvascular decompression for hemifacial spasm. J Korean Neurosurg Soc. 2012 Oct;52(4):288-92. doi: 10.3340/jkns.2012.52.4.288. Epub 2012 Oct 22. PMID: 23133714; PMCID: PMC3488634.
68. Hengstman GJ, Gons RA, Menovsky T, Lunel FV, van de Vlasakker CJ, de Vries J. Delayed cranial neuropathy after neurosurgery caused by herpes simplex virus reactivation: report of three cases. Surg Neurol. 2005 Jul;64(1):67-9; discussion 69-70. doi: 10.1016/j.surneu.2004.08.066. PMID: 15993188.