ORIGINAL ARTICLE

Correlation of Severity of Hemifacial Spasm and MRI/MRA Brain Findings

Suntaree Thitiwichienlert, MD¹, Varalee Mingkwansook, MD², Paiboon Bawornwattanadilok, MD¹, Rungrut Manakith, MD¹, Grobgarn Wichitnark, MD¹, Sopita Anantamongkonkul, MD¹

¹ Department of Ophthalmology, Faculty of Medicine, Thammasat University, Pathum Thani, Thailand; ² Department of Radiology, Faculty of Medicine, Thammasat University, Pathum Thani, Thailand

Objective: To investigate the correlation between the clinical severity of hemifacial spasm (HFS) and brain magnetic resonance imaging (MRI)/ magnetic resonance angiography (MRA) findings.

Materials and Methods: A prospective cohort study was conducted in 47 patients who underwent MRI/MRA brain at Thammasat Hospital and follow-up between January 1, 2023 and March 31, 2024. They were divided into two groups according to the clinical severity by Samsung Medical Center (SMC) grading as mild for SMC grade I and II, and severe for SMC grade III and IV. Neurovascular compression (NVC) severity in MRI/MRA brain findings was defined as mild for facial nerve abutment, moderate for nerve indentation, and severe for nerve course deviation.

Results: The mild spasm group included 22 cases and 11 cases or 50%, had mild compression, nine cases, or 40.9%, had moderate compression, and two cases, or 9.1%, had no compression. The severe spasm group included 25 cases and 15 cases, or 60%, had mild compression, eight cases, or 32%, had moderate compression, and two cases, or 8.0%, had no compression. The present study found no significant difference in clinical severity between moderate compression (p=0.491) and severe compression (p=0.526). Similarly, subgroup analysis revealed no significant difference in clinical severity between moderate (p=0.929) and severe compression (p=0.657) within each SMC grading feature subgroup. Most cases had benign causes, but two mild spasm cases presented with non-NVC, of which, one had meningioma, and another case an unruptured aneurysm.

Conclusion: Patients diagnosed with HFS should undergo brain MRI/MRA as cases may have non-NVC causes, which can have potential life-threatening consequences.

Trial registration: The present study protocol was registered at the Thai Clinical Trials Registry (TCTR20240824001; date August 24, 2024).

Keywords: Clinical severity; Hemifacial spasm; Brain magnetic resonance imaging; Magnetic resonance angiography

Received 26 September 2024 | Revised 18 November 2024 | Accepted 25 November 2024

J Med Assoc Thai 2025;108(1):72-9

Website: http://www.jmatonline.com

A hemifacial spasm (HFS) is a subtype of a facial movement disorder characterized by involuntary spasms of the ipsilateral facial muscles due to irritation of the facial nerves, especially the nerve root exit zone (REZ), causing it to interfere with the patient's daily life. HFS can be classified into two types, primary HFS, and secondary HFS, according to its causes. Primary HFS often has benign causes, including neurovascular compression (NVC) such as dolichoectasia presses on the nerve in the REZ.

Correspondence to:

Thitiwichienlert S.

Department of Ophthalmology, Faculty of Medicine, Thammasat University, Pathum Thani 12120, Thailand. Phone: +66-2-9269957, Fax: +66-2-9869212 Email: punoipunoi@hotmail.com

How to cite this article:

Thitiwichienlert S, Mingkwansook V, Bawornwattanadilok P, Manakith R, Wichitnark G, Anantamongkonkul S. Correlation of Severity of Hemifacial Spasm and MRI/MRA Brain Findings. J Med Assoc Thai 2025;108:72-9. DOI: 10.35755/jmedassocthai.2025.1.72-79-01627

It can also be idiopathic. Secondary HFS often has various causes including aneurysms, tumors, demyelination, or trauma⁽¹⁾. Radiologic studies have found that primary HFS is more prevalent than secondary HFS⁽²⁾. Therefore, all patients with HFS should undergo magnetic resonance imaging (MRI)/ magnetic resonance angiography (MRA) of the brain to determine the cause of HFS and help in planning surgery for patients found to have NVC pressing on a nerve.

In Thailand, there may be limitations in sending for an MRI/MRA of the brain in some places. Some physicians consider sending for an MRI/MRA of the brain when there are indications of 1) intractable HFS that does not respond to treatment with botulinum toxin type A (BTX-A) injections, or 2) a patient has associated neurological symptoms and signs such as a clicking sound in the ear, hearing loss, ataxia, or weakness. Previous studies have found that the severity of NVC during microvascular decompression (MVD) surgery is related to the degree of nerve indentation⁽³⁾. In the authors' practice, patients, even those having severe symptoms, refuse MVD surgery and prefer treatment with BTX-A injections first. The authors hypothesized that clinical severity might be related to radiologic nerve compression. In the present study, the authors investigated the correlation between the clinical severity of HFS and contrastenhanced brain MRI/MRA findings.

Materials and Methods

The present study design was a prospective cohort analysis. All patients older than 18 years diagnosed with HFS who underwent MRI/MRA of the brain, including base of skull protocol, between January 1, 2023 to March 31, 2024 at Thammasat University Hospital were included in the study. The diagnosis of HFS and clinical severity were made by a single neuro-ophthalmologist. The severity of radiologic nerve compression was made by a single neuroradiologist. The authors excluded female patients who were pregnant or lactating and patients who had BTX-A injections within the previous 12 weeks or had previous MVD surgery. The present study was approved by the Medical Ethics Committee of Thammasat University (MTU-EC-OP-1-245/65) and was conducted in accordance with the tenets of the Declaration of Helsinki. Prior to participation, all individuals provided written informed consents.

All patients were interviewed about their demographic data and the clinical characteristics of the HFS. The severity of HFS was defined by the Samsung Medical Center (SMC) grading scale⁽⁴⁾ as grade I for localized spasm around the periocular area, grade II for involuntary movement spreading to other parts of the ipsilateral face and affecting other muscle groups, i.e. the orbicularis oris, zygomaticus, frontalis or platysma muscle, grade III for interference with vision because of frequent tonic spasms, and grade IV for disfiguring asymmetry. ST assessed the SMC grading scale and the function of the facial nerve. The present study divided spasms into two groups according to the SMC grading scale, comprising mild spasms, which are SMC grade I and II, and severe spasms, which are SMC grade III and IV. The primary outcome measures demonstrated the correlation between the clinical severity of HFS and findings of brain MRI/MRA. The secondary outcomes included the clinical factors that affect radiologic nerve compression. Following MRI/MRA, all patients were follow-up for an average minimum of 12 weeks, as this duration corresponds to the period when the effects of the administered BTX-A start to diminish.

Table 1. Severity of neurovascular compression from MRI/MRA findings

Severity	Compression by MRI/MRA findings				
No	No NVC				
Mild	Facial nerve abutment				
Moderate	Facial nerve indentation				
Severe	Facial nerve deviation				

MRI=magnetic resonance imaging; MRA=magnetic resonance angiography; NVC=neurovascular compression

MRI/MRA technique

The eligible patients underwent MRI and MRA of the brain with cranial nerve protocol using a 1.5 T scanner (Magnetom Aera, Siemens, Erlangen, Germany) or a 3.0 T scanner (Magnetom Skyra, Siemens, Erlangen, Germany). The imaging protocol employed in the present study consisted of multiple sequences, each with specific parameters. These included coronal T1-weighted imaging (T1WI), T2weighted imaging with fat suppression (T2WI FS), and T1WI with gadolinium contrast enhancement and a field of view (FOV) of 160×160 mm, slice thickness of 3 mm, and no gap. Additionally, axial T1WI and T2WI FS with FOV of 160×160 mm, slice thickness of 3 mm, and no gap, were performed. High-resolution axial and coronal T2-weighted 3D imaging was acquired with a FOV of 160×160 mm, slice thickness of 0.8 mm, and no gap, as well as axial T1-weighted 3D imaging with fat suppression and gadolinium contrast enhancement with a FOV of 160×160 mm, slice thickness of 3 mm, and no gap. Magnetic resonance angiography (MRA) of the brain was conducted using both 3D time-offlight (TOF) imaging with a slice thickness of 0.6 mm, and a contrast-enhanced technique with a slice thickness of 0.8 mm in the axial plane. Finally, a three-dimensional reconstruction of the MRA data was performed to facilitate further analysis.

This imaging protocol was employed to assess the course of the facial nerve, from the root entry zone (REZ) to the internal auditory canal (IAC), and its relationship with the surrounding vertebrobasilar system. Radiologic evidence of nerve compression due to NVC was classified into three categories, mild, characterized by facial nerve abutment, moderate, indicated by nerve indentation, and severe, defined by deviation in the nerve's course. These assessments were conducted by VM, who was blinded to the clinical details of the cases (Table 1, Figure 1).

Sample size calculation

The sample size was calculated on the result of

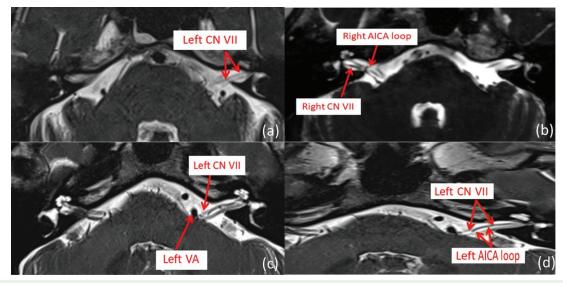


Figure 1. The MRI and MRA brain findings demonstrated radiologic nerve compression of the cranial nerve VII (CN VII); (a) no neurovascular compression (NVC), (b) CN VII abutment from the anterior inferior cerebellar artery (AICA) loop, (c) CN VII indentation from the left vertebral artery (VA), (d) CN VII deviation from the left AICA (arrow).

Banerjee et al.'s study, which showed that the rate of facial nerve abnormality from MRI/MRA in patients diagnosed with HFS was found to be $78.22\%^{(5)}$. In the present study, the error value allowed to occur was 15% of the p-value, equal to $0.782 \times 0.15 = 0.1173$. The sample size was 48 by using the formula of estimating a single proportion⁽⁶⁾. With an expected loss of data collection, a sample size of 50 was needed.

Statistical analysis

The data were analyzed using IBM SPSS Statistics, version 23.0 (IBM Corp., Armonk, NY, USA). Baseline characteristics were described as number (percentage), median (range), and mean with standard deviation (SD). Chi-square or Fisher's exact test was employed to compare categorical data including gender, underlying disease, presence of facial palsy, and MRI/MRA findings between the two groups. The independent t-test was used to compare the mean values of continuous quantitative data between the two groups, and the Mann-Whitney U test was used for non-nominal distributions. All reported p-values were 2-sided with p<0.05 set as the threshold for statistical significance.

The present study protocol was registered in the Thai Clinical Trails Registry (TCTR) and approved on August 24, 2024, identification number TCTR20240824001. Data of all cases in the analyses are available on request. The trial protocol can be accessed at: https://www.thaiclinicaltrials.org/show/ TCTR20240824001.

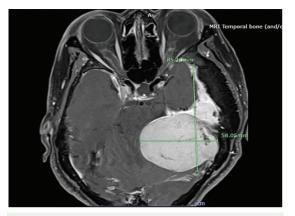


Figure 2. An example case of secondary HFS with mild spasms from a large meningioma with a tumor 85.10×58.05 mm in initial size (dimension lines).

Results

Initially, fifty patients were enrolled in the present study, but two lost to follow-up and one had claustrophobia during the scanning process. Thus, forty-seven patients were included in the study, comprising 34 females (72.3%), and 13 males (27.7%). The mean age was 62.13 ± 11.92 years, with a range of 27 to 86 years. Twenty-two patients were in the mild spasm group, and 25 patients were in the severe spasm group. After MRI/MRA testing, it was found that 45 (95.74%) had primary HFS, and two (4.26%) had secondary HFS causes. In patients with primary HFS, 41 cases of NVC were found, along with four cases of idiopathic causes. The anterior

Table 2. Patient's demographic data and baseline characteristics (n=47)

Characteristics	Total (n=47); n (%)	SMC clinical	p-value	
		Mild spasm group (n=22)	Severe spasm group (n=25)	
Sex				0.478
Male	13 (27.7)	5 (22.7)	8 (32.0)	
Female	34 (72.3)	17 (77.3)	17 (68.0)	
Age (years)				0.221
<60	19 (40.4)	6 (27.3)	13 (52.0)	
60 to 69	18 (38.3)	10 (45.5)	8 (32.0)	
≥70	10 (21.3)	6 (27.3)	4 (16.0)	
Mean±SD	62.13±11.92	64.95 ± 11.68	59.64 ± 11.80	0.129
Underlying diseases				0.491
Yes	21 (44.7)	11 (50.0)	10 (40.0)	
No	26 (55.3)	11 (50.0)	15 (60.0)	
• HT	16 (34.0)	8 (36.4)	8 (32.0)	0.753
• DLP	14 (29.8)	8 (36.4)	6 (24.0)	0.355
• DM	5 (10.6)	2 (9.1)	3 (12.0)	1.000
• IHD	1 (2.1)	0 (0.0)	1 (4.0)	1.000
• CVA	2 (4.3)	1 (4.5)	1 (4.0)	1.000
• CKD	1 (2.1)	0 (0.0)	1 (4.0)	1.000
Duration (months)				0.778
≥12	13 (27.7)	5 (22.7)	8 (32.0)	
13 to 36	14 (29.8)	7 (31.8)	7 (28.0)	
>36	20 (42.6)	10 (45.5)	10 (40.0)	
Median (IQR)	24.0 (12 to 60)	35.5 (21 to 60)	24.0 (12 to 72)	0.659
Facial palsy				1.000
Yes	6 (12.8)	3 (13.6)	3 (12.0)	
No	41 (87.2)	19 (86.4)	22 (88.0)	
Laterality				0.391
Right	18 (38.3)	7 (31.8)	11 (44.0)	
Left	29 (61.7)	15 (68.2)	14 (56.0)	
Primary HFS				
Negative	4 (8.5)	2 (9.1)	2 (8.0)	1.000
AICA	34 (72.3)	17 (77.3)	17 (68.0)	0.478
PICA	4 (8.5)	0 (0.0)	4 (16.0)	0.112
VA	4 (8.5)	3 (13.6)	1 (4.0)	0.328
PCA	1 (2.1)	0 (0.0)	1 (4.0)	1.000
Secondary HFS				
Others	2 (4.3)	2 (9.1)	0 (0.0)	0.214

SMC=Samsung Medical Center; HT=hypertension; DLP=dyslipidemia; DM=diabetes mellitus; IHD=ischemic heart disease; CVA=cerebral vascular accident; CKD=chronic kidney disease; HFS=hemifacial spasm; AICA=anterior inferior cerebellar artery; PICA=posterior inferior cerebellar artery; VA=vertebral artery; PCA=posterior cerebral artery; SD=standard deviation; IQR=interquartile range

p-value for mean data were calculated with the use of independent t-test or Mann-Whitney U-test, for percentages with the use of chi-square test or Fisher's exact test

inferior cerebellar artery (AICA) was the most common vessel causing NVC at 72.3%, followed by the posterior inferior cerebellar artery (PICA) at 8.5%, vertebral artery (VA) at 8.5%, and posterior cerebral artery (PCA) at 2%. Two patients had both AICA and VA compressions of their nerves. In the two patients with secondary HFS, both had mild spasms (SMC grade II), but one had multiple meningiomas and the other had an unruptured basilar tip aneurysm (Figure 2).

The baseline clinical characteristics, including demographic data, underlying vascular disease, duration of disease, presence of facial palsy, and vessel causing NVC were not significantly different

Table 3. Primary outcomes

Total (n=47)	Clinical severity by SMC grading scale; n (%)		Proportion difference (95% CI)	p-value
n (%)	Mild spasm (grade I-II) (n=22)	Severe spasm (grade III-IV) (n=25)		
				0.823
4 (8.5)	2 (9.1)	2 (8.0)	1.1 (-17.09 to 20.70)	1.000
26 (55.3)	11 (50.0)	15 (60.0)	10 (-17.25 to 35.44)	0.491
17 (36.2)	9 (40.9)	8 (32.0)	8.9 (-17.47 to 34.07)	0.526
	n (%) 4 (8.5) 26 (55.3)	n (%) Mild spasm (grade I-II) (n=22) 4 (8.5) 2 (9.1) 26 (55.3) 11 (50.0)	n (%) Mild spasm (grade I-II) (n=22) Severe spasm (grade III-IV) (n=25) 4 (8.5) 2 (9.1) 2 (8.0) 26 (55.3) 11 (50.0) 15 (60.0)	n (%) Mild spasm (grade I-II) (n=22) Severe spasm (grade III-IV) (n=25) 4 (8.5) 2 (9.1) 2 (8.0) 1.1 (-17.09 to 20.70) 26 (55.3) 11 (50.0) 15 (60.0) 10 (-17.25 to 35.44)

MRI=magnetic resonance imaging; MRA=magnetic resonance angiography; NVC=neurovascular compression; SMC=Samsung Medical Center; CI=confidence interval

p-value for chi-square test or Fisher's exact test

Table 4. Analysis within specific categories of primary outcomes

	Total (n=47); n (%)	Clinical severity by SMC grading scale; n (%)				p-value
		SMC grade I (n=2)	SMC grade II (n=20)	SMC grade III (n=16)	SMC grade IV (n=9)	
MRI/MRA findings						0.898
No NVC	4 (8.5)	0 (0.0)	2 (10.0)	2 (12.5)	0 (0.0)	0.847
Abutment	26 (55.3)	1 (50.0)	10 (50.0)	10 (62.5)	5 (55.6)	0.929
Indentation	17 (36.2)	1 (50.0)	8 (40.0)	4 (25.0)	4 (44.4)	0.657

SMC=Samsung Medical Center; MRI=magnetic resonance imaging; MRA=magnetic resonance angiography; NVC=neurovascular compression p-value from chi-square test or Fisher's exact test

Table 5. Secondary outcomes

	Total (n=47); n (%)	Radiologic nerve compression	on by MRI/MRA findings; n (%)	Proportion difference (95% CI)	p-value
		Mild compression (n=30)	Severe compression (n=17)		
Facial palsy	6 (12.8)	4 (13.3)	2 (11.7)	1.6 (-22.34 to 19.99)	1.000
Clicking sound in the ear	5 (10.6)	2 (6.7)	3 (17.6)	10.94 (-7.67 to 34.82)	0.336
Hearing loss	1 (2.1)	0 (0.0)	1 (5.9)	5.88 (-6.46 to 26.98)	0.362
Neurological signs	1 (2.1)	0 (0.0)	1 (5.9)	5.88 (-6.46 to 26.98)	0.362

 $MRI = magnetic \ resonance \ imaging; \ MRA = magnetic \ resonance \ angiography; \ CI = confidence \ interval \ resonance \$

p-value for chi-square test or Fisher's exact test

in both groups (Table 2). In the mild spasm group, which was 22 cases, there were 11 cases (50%) of mild compression, nine cases (40.9%) of moderate compression, and two cases (9.1%) of no compression. In the severe spasm group, which included 25 cases, there were 15 cases (60%) of mild compression, eight cases (32%) of moderate compression, and two cases (8.0%) of no compression. The present study found no significant difference in clinical severity between moderate compression (p=0.491) and severe compression (p=0.526), as shown in Table 3. Similarly, analysis within specific categories revealed no significant difference in clinical severity between moderate (p=0.929) and severe compression (p=0.657) within each SMC grading feature, detailed in Table 4. For the clinical factors that might affect radiologic nerve compression, the authors found facial palsy in six patients (12.77%), hearing loss in one patient (2.13%), and hemiplegia in one patient (2.13%). These factors were not correlated with radiological nerve compression severity (Table 5).

Discussion

Few studies have investigated the relationship between the clinical severity of HFS and the severity of NVC on neuroimaging^(7,8). Most studies have focused on comparing the severity of compression found on imaging with the severity seen during MVD surgery^(9,10). Previous studies have retrospectively analyzed patients with HFS and undergone MVD surgery to reduce the pressure of NVC. They found that severe spasms are a strong predictor of nerve indentation and facial palsy in those undergoing surgery. Their results showed that there was a correlation between intraoperative compression severity and clinical severity⁽¹¹⁾.

In the present study, the authors hypothesized that there will usually be normal neuroimaging findings or only slight NVC, if the symptoms were mild. Furthermore, MRI/MRA is easier to perform than surgery and most patients refuse surgery. Therefore, the authors investigated the clinical severity in patients with HFS who underwent MRI/MRA of the brain. This study used the SMC grading system, which was based on a study by Lee et al. that found this method to be useful in indicating quality of life and quantifying spasms⁽⁴⁾. The clinical assessor was not blinded, but the neuroradiologist would not know who had severe spasm to reduce bias in the study. All patients had similar characteristics at baseline and patients who received BTX-A injection would be assessed at 12 weeks post-injection, which is a similar period for the effects of the drug to wear off.

The primary outcome showed no significant correlation between clinical severity and NVC (p=0.823), which is different from a previous study that hypothesized that lower clinical severity may result in normal MRI/MRA brain findings or minimal NVC. The numbers of patients in the subgroup analysis in the SMC grade I and grade IV groups were small, which may explain why the results of this study did not find that severe symptoms were associated with severe compression. However, most patients with HFS responded well to BTX-A treatment. Only a few cases require MVD surgery, and most studies have pointed out the use of modern MRI brain protocols, which could visualize cranial nerves in the cisterna area. This is also a good indicator for assessing facial nerve compression compared to intraoperative findings^(12,13). Therefore, the authors believe that radiologic nerve compression from MRI/MRA brain findings may be useful in looking for indirect compressive effects, although it is not as definite as intraoperative findings during MVD surgery.

Studies have also discovered that HFS patients do not show NVC or other causes of facial nerve compression^(14,15). In the present study, four cases (8.5%) were found where NVC was not observed. Studies also found that NVC did not necessarily lead to clinical symptoms, which might indicate that other factors such as cortical adaptation, nerve sensitivity, genetic susceptibility, stress, and emotional factors were involved in determining the clinical symptoms and their severity⁽¹⁶⁻¹⁸⁾. The present study found only two cases having non-NVC that were caused by large multiple meningiomas and vascular aneurysms, which correlated with the previous studies indicating the low incidence of non-NVC causes⁽¹⁴⁾.

The secondary outcome did not show that the

presence of facial palsy correlates with radiological nerve compression severity, which is different from the previous studies, which found secondary HFS had significantly more hearing loss and facial palsy than primary HFS, and that the presence of facial palsy is related to the severity of the disease⁽¹⁴⁾. In the present study, there were only three secondary HFS patients. The higher proportion of patients with primary HFS than secondary HFS may explain that the present study had different results from the previous studies. The low incidence of clinical factors may affect radiologic nerve compression including the presence of facial palsy, clicking sound in the ear, hearing loss, and neurological signs. Statistical interpretation of secondary outcomes aimed at investigating the clinical factors that influence or correlate the compression severity in neuroimaging is limited in the current study.

The present study faced limitations. The small sample size limited the subgroup analysis for patients with SMC grade I and grade IV, as did the variety of non-NVC causes, with only two patients without NVC. There are many factors involved in the clinical severity assessment, including the assessor and the timing of post-treatment. Ideally, the assessor should be independent of the research team to minimize bias. For continuity in evaluation, measurements are taken at 12 weeks post-injection to coincide with the period during which the effect of the BTX-A begins to decrease. It has been reported that the BTX-A effect can extend beyond 12 weeks, which could lead to an underestimation of clinical severity. Moreover, an individual's response to BTX-A varies as patients may experience effects lasting longer than 12 weeks, which may affect the assessment of clinical severity. Patients had MRI/MRA brain scans performed at different institutions. However, the same protocol was established for every case, and a single neuroradiologist was used to interpret and analyze the imaging results to ensure consistency. The neuroradiologist was blinded to the affected side and clinical severity to diminish bias. Furthermore, the study's reliability was compromised by the presence of only one clinical assessor and one neuroradiologist, without any intraobserver variability, leading to potential methodological bias. Additionally, the sample size for each group should be increased to allow for proper characterization and differentiation in future studies.

Conclusion

In summary, the present study demonstrated that

there was no correlation between clinical severity and compression severity from the findings of MRI/ MRA of the brain. Despite most causes being benign, patients diagnosed with HFS should undergo brain MRI/MRA as cases with mild clinical severity may have non-NVC causes, which can have potentially life-threatening consequences. In the future, research should be conducted that considers both clinical severity and MRI findings with a larger sample size as well as a greater variety of clinical severity to demonstrate whether there is a correlation.

What is already known about this topic?

Previous studies found that severe spasms are a strong predictor of nerve indentation and facial palsy in those undergoing surgery. The greater the intraoperative compression severity, the greater the clinical severity.

What does this study add?

This study found that there was no correlation between clinical severity and compression severity from the findings of MRI/MRA of the brain. However, the authors think that the results of this study are still useful in looking at compression effects indirectly from neuroimaging, although they may not be as definitive as intraoperative findings during MVD surgery. Not only can imaging identify the cause of HFS, but also helps in MVD planning, which is considered a definitive treatment.

Acknowledgement

The authors would like to thank Miss Manussanun Tanavikrankoon for her support with the statistical analyses.

Authors' contributions

ST: Conceptualization, methodology, data curation, investigation, resources, writing-original draft, writing-review & editing, formal analysis, software, supervision, project administration, funding acquisition. VM: Conceptualization, methodology, investigation, resources, writing-review & editing, supervision. PB: Conceptualization, methodology, resources, formal analysis. RM: Conceptualization, methodology, investigation, resources, writingoriginal draft. GW: Conceptualization, methodology, investigation, resources. SA: Conceptualization, methodology, investigation, resources.

Funding disclosure

The present study was funded by the Faculty of

Medicine, Thammasat University.

Conflicts of interest

The authors declare that there is no conflict of interest.

References

- Girard B, de Saint Sauveur G, Tatry M, Abdellaoui M, Tassart M. Spasmes hémifaciaux. Étiologies et conduite à tenir. [Hemifacial spasm. Etiology and management]. J Fr Ophtalmol 2021;44:382-90.
- Colosimo C, Bologna M, Lamberti S, Avanzino L, Marinelli L, Fabbrini G, et al. A comparative study of primary and secondary hemifacial spasm. Arch Neurol 2006;63:441-4.
- Kim HR, Rhee DJ, Kong DS, Park K. Prognostic factors of hemifacial spasm after microvascular decompression. J Korean Neurosurg Soc 2009;45:336-40.
- 4. Lee JA, Jo KW, Kong DS, Park K. Using the new clinical grading scale for quantification of the severity of hemifacial spasm: correlations with a quality-of-life scale. Stereotact Funct Neurosurg 2012;90:16-9.
- Banerjee P, Alam MS, Koka K, Pherwani R, Noronha OV, Mukherjee B. Role of neuroimaging in cases of primary and secondary hemifacial spasm. Indian J Ophthalmol 2021;69:253-6.
- Daniel WW. Biostatistics: A foundation of analysis in the health sciences. 6th ed. New York: John Wiley and Sons; 1995.
- Tan EK, Chan LL. Clinico-radiologic correlation in unilateral and bilateral hemifacial spasm. J Neurol Sci 2004;222:59-64.
- Traylor KS, Sekula RF, Eubanks K, Muthiah N, Chang YF, Hughes MA. Prevalence and severity of neurovascular compression in hemifacial spasm patients. Brain 2021;144:1482-7.
- Campos-Benitez M, Kaufmann AM. Neurovascular compression findings in hemifacial spasm. J Neurosurg 2008;109:416-20.
- Fukuda H, Ishikawa M, Okumura R. Demonstration of neurovascular compression in trigeminal neuralgia and hemifacial spasm with magnetic resonance imaging: comparison with surgical findings in 60 consecutive cases. Surg Neurol 2003;59:93-9.
- Na BS, Cho JW, Park K, Kwon S, Kim YS, Kim JS, et al. Severe hemifacial spasm is a predictor of severe indentation and facial palsy after microdecompression surgery. J Clin Neurol 2018;14:303-9.
- Finger G, Wu KC, Vignolles-Jeong J, Godil SS, McGahan BG, Kreatsoulas D, et al. A new finding on magnetic resonance imaging for diagnosis of hemifacial spasm with high accuracy and interobserver correlation. Brain Sci 2023;13:1434. doi: 10.3390/ brainsci13101434.
- 13. Haller S, Etienne L, Kövari E, Varoquaux AD, Urbach

H, Becker M. Imaging of neurovascular compression syndromes: Trigeminal neuralgia, hemifacial spasm, vestibular paroxysmia, and glossopharyngeal neuralgia. AJNR Am J Neuroradiol 2016;37:1384-92.

- Batla A, Goyal C, Shukla G, Goyal V, Srivastava A, Behari M. Hemifacial spasm: clinical characteristics of 321 Indian patients. J Neurol 2012;259:1561-5.
- Pandey S, Jain S. Clinical features and response to botulinum toxin in primary and secondary hemifacial spasm. Neurol India 2018;66:1036-42.
- Kotterba S, Tegenthoff M, Malin JP. Hemifacial spasm or somatoform disorder--postexcitatory inhibition after transcranial magnetic cortical stimulation as a diagnostic tool. Acta Neurol Scand 2000;101:305-10.
- 17. Tan EK, Jankovic J. Psychogenic hemifacial spasm. J Neuropsychiatry Clin Neurosci 2001;13:380-4.
- Carter JB, Patrinely JR, Jankovic J, McCrary JA 3rd, Boniuk M. Familial hemifacial spasm. Arch Ophthalmol 1990;108:249-50.