Review Article

Treatment Challenges in Hemifacial Spasm: The Role of Magnetic Resonance Imaging

Mohammad Bahadoram [1,4], Esma'il Akade [2], Seyed Ehsan Mohammadianinejad [3], Shana Ahadi [1], Mohammad Davoodi [4]

1. Department of Neurology, School of Medicine, Ahvaz Jundishapur University of Medical Sciences, Ahvaz, Iran
2. Department of Medical Virology, School of Medicine, Ahvaz Jundishapur University of Medical Sciences, Ahvaz, Iran
3. Iranian Center of Neurological Research, Neuroscience Institute, Imam Khomeini Hospital Complex, Tehran University of Medical Sciences, Tehran, Iran
4. Department of Radiology, Shariati Hospital, Tehran University of Medical Sciences, Tehran, Iran

***Corresponding Author:**

Dr. Mohammad Davoodi

Shariati Hospital, Jalal-e-Al-e-Ahmad Hwy, Tehran, Iran. Postal Code: 1411713135

Email:
mohammaddavoodi47@yahoo.com

Received: 16/3/2024
Accepted: 7/11/2024

ABSTRACT

Hemifacial spasm (HFS) is an important condition for plastic surgeons to understand, as it significantly affects patients' quality of life and can complicate aesthetic and reconstructive procedures. Magnetic resonance imaging (MRI) has become vital in diagnosing HFS, assessing neurovascular relationships, and planning treatment. Transverse MRI scans often show an upward displacement of the inferior pons at the facial nerve attachment point, signaling neurovascular compression (NVC) in HFS. Important anatomical features for neurosurgeons include cerebellar atrophy and a "small posterior fossa," which can lead to HFS by narrowing fluid spaces. The posterior fossa is often more congested in HFS patients, and anatomical flatness may exacerbate lateral deviation of the vertebrobasilar arteries. Additionally, evaluating sigmoid sinus dominance and other anatomical variations is crucial for surgical planning, particularly in cases of arterial hypertension that may affect medullary compression.

KEYWORDS
Facial Nerve; Botulinum Neurotoxin; Microvascular Decompression Surgery; Neurovascular Compression Syndrome; Plastic Surgery

Please cite this paper as:
Bahadoram M., Akade E., Mohammadianinejad SE., Ahadi S., Davoodi M. Treatment Challenges in Hemifacial Spasm: The Role of Magnetic Resonance Imaging. World J Plast Surg. 2024;13(3):14-22.
doi: 10.61186/wjps.13.3.14

INTRODUCTION

In the 11th century, Persian physician Esma'il Jorjani documented conditions resembling trigeminal neuralgia, hemifacial spasm (HFS), and Bell's palsy in his work, *Treasure of the Khawarazm Shah*. He also suggested that trigeminal neuralgia could stem from an artery compressing a nerve [1]. The term "hemifacial spasm" describes involuntary contractions on one side of the face. Clinically, HFS refers to irregular, unilateral muscle contractions that can be either tonic or clonic. These spasms typically begin in the muscles around the eye and may extend to the muscles around the nose, mouth, cheek, and neck [2]. Diagnosing HFS relies largely on a patient's medical history and physical exam, though electromyographic (EMG) and imaging studies are often used as supplementary tools [3].
An epidemiological study conducted in Norway found the prevalence of HFS to be approximately 9.8 cases per 100,000 people [4]. In the United States, another study revealed that HFS affects about 7.4 per 100,000 men

[Downloaded from wjps.ir on 2026-10-31]

[DOI: 10.61186/wjps.13.3.14]

and 14.5 per 100,000 women [5]. Research indicates a male-to-female ratio of 1:2.28, with an average onset age of 52.2 years [6]. Ethnically, HFS appears to be more common in Asian populations compared to others [7]. Psychological conditions like anxiety or depression are frequently associated with HFS and are thought to impact its prognosis [8]. While a few familial cases have been documented, HFS is generally not hereditary and tends to occur mainly in adults [9].

HFS is classified as primary and secondary types according to the cause.

1. Primary HFS is believed to result from vascular compression at the root entry zone (REZ) of the facial nerve.
2. Secondary HFS arises from facial nerve damage caused by other conditions, such as facial palsy, cerebellopontine angle (CPA) tumors, Chiari I malformations, demyelinating diseases, or infections [10].

Primary HFS occurs 3–4 times more often than secondary HFS [11]. When vascular compression at the REZ is involved, the anterior inferior cerebellar artery (AICA) is typically responsible, followed by the posterior inferior cerebellar artery (PICA) and the vertebral artery (VA). Although a single artery may be the sole source of neurovascular compression, this is rare, occurring in only 4.7% of cases as noted in a prior study. Although a single artery may be the sole source of neurovascular compression, this is rare, occurring in only 4.7% of cases as noted in a prior study. Six compressive patterns of HFS have been proposed, which include loop, arachnoid, perforator, branch, sandwich, and tandem types [12]. The underlying pathophysiology of HFS is thought to involve microscopic myelin damage in or near the REZ, where the offending vessel exerts pressure. Two main theories attempt to explain the mechanism of HFS: the central hypothesis, which suggests hyperexcitability of the facial motor nucleus, and the peripheral hypothesis, which proposes ephaptic transmission between facial nerve bundles. Growing anatomical and neurophysiological research is focused on uncovering the exact mechanisms, but neither hypothesis fully accounts for all aspects of the condition without support from the other [13].

Botulinum neurotoxin therapy and surgical approaches to HFS

Botulinum neurotoxin (BTX) injections are the most commonly chosen nonsurgical option for treating HFS, providing symptom relief in up to 85% of cases. Of the seven BTX serotypes, only types A and B are commercially available. After injection, symptoms begin to improve within 1–3 days, typically reaching their maximum effect by day 5 [14]. The duration of relief varies, typically lasting 3 to 6 months depending on the medical center. Since the effects wear off, repeated injections are necessary, and some patients may develop tolerance. However, a 10-year multicenter study found that the duration of symptom improvement remained consistent from the first to the tenth year of treatment, even with similar doses of BTX. Additionally, the study noted a reduction in BTX-related side effects over the course of treatment. Local complications such as ptosis, blurred vision, and double vision are possible but rarely permanent. Adverse effects occur in approximately 20–53% of cases, with ptosis being the most common. While BTX injections are generally effective, the need for regular treatments can place a significant emotional and financial burden on patients [15]. This is while some patients may not get satisfying results from BTX injection, leaving with some degrees of asymmetric face between the injections.

Among surgical options, microvascular decompression (MVD) stands as the only curative treatment for HFS, offering a high success rate with minimal recurrence and complication risks. A systematic review of 22 studies involving 5,700 MVD patients showed that 91.1% achieved complete symptom resolution. Recurrence was noted in only 2.4%, and temporary complications included facial palsy (9.5%), hearing loss (3.2%), and cerebrospinal fluid leakage (1.4%). Permanent complications were rarer, with 2.3% experiencing hearing loss and 0.9% developing facial palsy. The risk of stroke was 1 in 1,800, while the mortality rate was 1 in 5,500 [16].

Although the fundamental MVD technique is well-documented, specific surgical maneuvers can differ between surgeons. Typically, after a lateral retrosigmoid suboccipital craniotomy or craniectomy is performed under general anesthesia, the dura is opened to expose the cerebellar cortex. The REZ of the facial nerve is then examined, and any compressing arteries are identified and separated from the seventh nerve. This is often maintained by inserting Teflon pads. Additional methods such as vessel transposition, snare

[Downloaded from wjps.ir on 2026-10-31]

[DOI: 10.61186/wjps.13.3.14]

techniques, or vascular slings may also be used [17]. Intraoperative EMG monitoring has proven useful for improving outcomes, particularly the lateral spread response (LSR), a neurophysiological test popularized by Moller and Jannetta (Figure 1). The disappearance of LSR is often taken as a sign of successful decompression, though its persistence doesn't always predict a poor outcome, limiting its reliability for long-term prognosis [18, 19]. Additionally, intraoperative brain stem auditory evoked potentials (BAEP) are frequently employed to monitor the integrity of cranial nerve VIII, helping reduce the risk of hearing loss during surgery [6].

Postoperative recovery following MVD is not

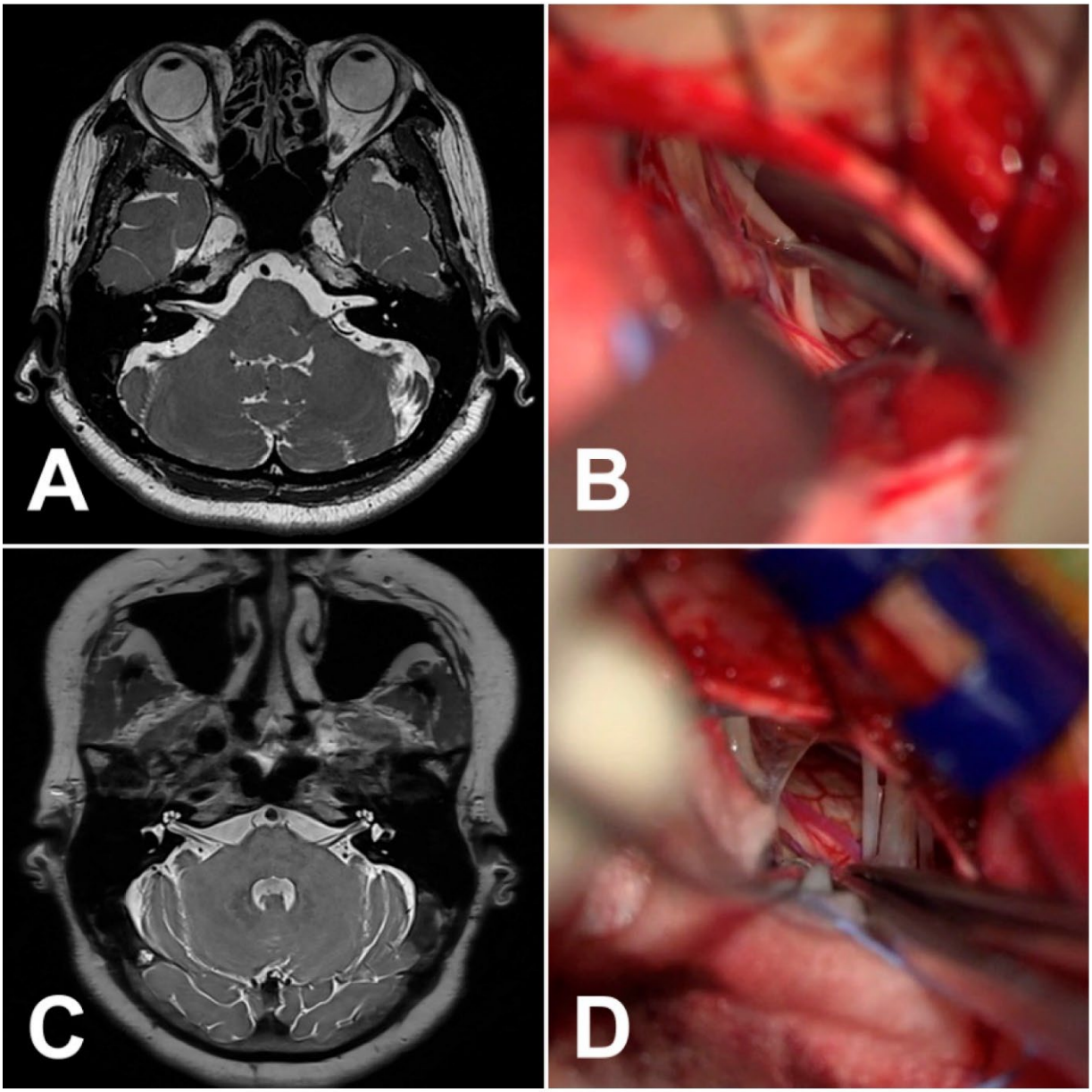


Figure 1: (A) A 3D MR cisternography (MRC) scan using 3D VISTA for a case with right hemifacial spasm appears normal. (B) During surgery, no neurovascular conflict (NVC) was observed at the root exit zone. Decompression was performed on a venous-arterial sandwich-type compression at the cisternal part of the facial nerve. The lateral spread response (LSR) disappeared immediately after decompressing the vein, following the initial decompression of a small arteriole. (C) A 3D MRC using 3D VISTA for another case with right hemifacial spasm also showed normal findings. (D) In surgery, no NVC was seen at the root exit zone. After a 360-degree exploration of the root exit zone, the LSR vanished after decompressing a small anterior inferior cerebellar artery (AICA), which was causing arachnoid-type compression in the distal cisternal part of the facial nerve, close to the internal auditory canal (Image from an article by Chiman Jeon et al. published in Life, 2023) [20]

[Downloaded from wjps.ir on 2026-10-31]

[DOI: 10.61186/wjps.13.3.14]

uniform. In one study, 92.8% of 807 patients were either completely or nearly spasm-free two years after surgery. However, 19.0% of those who eventually became symptom-free experienced residual spasms for over a month, and in some cases, for more than a year. There is no widely accepted explanation for this variability in recovery, underscoring the need for further microanatomic and electrophysiologic research [6].

Lastly, MVD requires thorough and precise preoperative evaluation, typically involving imaging techniques, to ensure proper diagnosis and planning.

The importance of magnetic resonance imaging in HSF

While many conditions are classified as neurovascular compression syndromes based on the cranial nerves involved and the resulting symptoms, a clear cause-and-effect relationship has only been firmly established for three specific disorders. These include trigeminal neuralgia, linked to compression of the trigeminal nerve; HFS, associated with the facial nerve; and vago-glossopharyngeal neuralgia, which affects the vagus and glossopharyngeal nerves [21].

With the development of magnetic resonance imaging (MRI), it quickly replaced conventional angiography and computed tomography (CT) in evaluating patients with HFS and other neurovascular compression syndromes like trigeminal neuralgia. MRI's excellent contrast resolution allows for the simultaneous visualization of both cranial nerves and blood vessels, providing a detailed view of their anatomical relationships [22]. Today, MRI, often combined with magnetic resonance angiography (MRA), is the preferred imaging technique for diagnosing and planning treatment for HFS. Advanced 3D imaging methods, such as fast spin-echo (FSE) or fast gradient-echo (FGE) sequences, enable a precise understanding of the neurovascular interactions that could be causing symptoms [23].

High-resolution 3D MRI has proven highly effective and accurate in the preoperative assessment of both primary and secondary HFS. Advances in MRI hardware and software now allow for high-quality imaging in a relatively short time. Transverse axial images acquired with 3D heavily T2-weighted fast imaging sequences, known as magnetic resonance cisternography (MRC), are particularly suited for evaluating the complex nerve-vessel interactions. In these images, nerves and vessels appear as low-signal areas against the bright signal of cerebrospinal fluid (CSF). Using 3D techniques, isotropic images can be reconstructed in any plane through multiplanar reformation (MPR), enhancing evaluation accuracy [24-26]. Various 3D steady-state FGE and FSE sequences are utilized across MRI platforms, such as FIESTA-C, CISS, and bTFE for FGE, and CUBE, SPACE, and VISTA for FSE [23].

3D time-of-flight (TOF) MRA is another useful tool to visualize problematic arteries, though it may struggle with smaller vessels. This technique is also beneficial for identifying high-flow vascular malformations [27]. Combining MRC and 3D TOF MRA through image fusion has been reported to enhance the visualization of neurovascular relationships in HFS patients [28, 29]. Additionally, 3D contrast-enhanced T1-weighted imaging is effective for evaluating HFS, particularly for ruling out secondary causes like tumors, inflammation, or demyelinating diseases. Venous lesions contributing to HFS are also better depicted with this technique [27].

For secondary HFS cases, especially in patients with demyelinating diseases such as multiple sclerosis, fluid-attenuated inversion recovery (FLAIR) sequences are valuable. By suppressing the fluid signal, FLAIR highlights lesions as areas of high signal intensity [27]. The fusion of 3D FLAIR and 3D TOF MRA has shown superiority in delineating neurovascular relationships when the root exit zone of the facial nerve is hard to visualize on MRC. This approach has also been beneficial in patients with trigeminal neuralgia [29, 30].

Diffusion-weighted imaging (DWI) is another useful tool for assessing HFS, particularly in identifying lesions with restricted diffusion, such as brainstem infarctions or epidermoid cysts in the cerebellopontine angle (CPA) cistern, which can cause secondary HFS. Additionally, diffusion tensor imaging with tractography has recently been reported as helpful, particularly in patients with trigeminal neuralgia [27, 31].

Normal MRI findings of the facial nerve

MRI offers detailed high-resolution images of the facial nerve outside the brainstem, but these findings must always be interpreted alongside clinical symptoms, as neurovascular contacts are

[Downloaded from wjps.ir on 2026-10-31]

[DOI: 10.61186/wjps.13.3.14]

often present in people without symptoms. Most of these asymptomatic neurovascular contacts do not involve nerve indentation. However, in some cases, significant displacement of the facial nerve caused by dolichoectatic vertebral arteries, located further from the REZ, can be seen in asymptomatic individuals (Figure 2). A recent study involving 100 MRI scans of people without HFS was analyzed by two independent observers, who identified neurovascular contact in 37% and 53% of cases, respectively. These contacts were typically more peripheral, away from the REZ, and less severe than what is seen in patients with HFS [32].

Differentiating primary and secondary causes of HFS

The initial step in interpreting MRI scans of patients with HFS is to rule out secondary causes, such as tumors, vascular anomalies, demyelinating diseases, or ischemic changes. If none of these conditions are present, attention should shift to the facial nerve's path to locate potential vascular compression. For diagnosing primary HFS, several key aspects must be included in the MRI report: the type of vessel causing the compression, the exact location of the neurovascular compression, and its severity [13].

Regarding the vessel type, primary HFS is most commonly caused by arterial compression, with the anterior inferior cerebellar artery (AICA) being responsible in 43–53.2% of cases, followed by the posterior inferior cerebellar artery (PICA) in 30.9–31%, and the vertebral artery in 1.1–23% [13, 34]. Multiple arteries may contribute to HFS; for example, Hyun et al.'s review of 1,174 patients found multiple vessels involved in 14.1% of cases [34], while

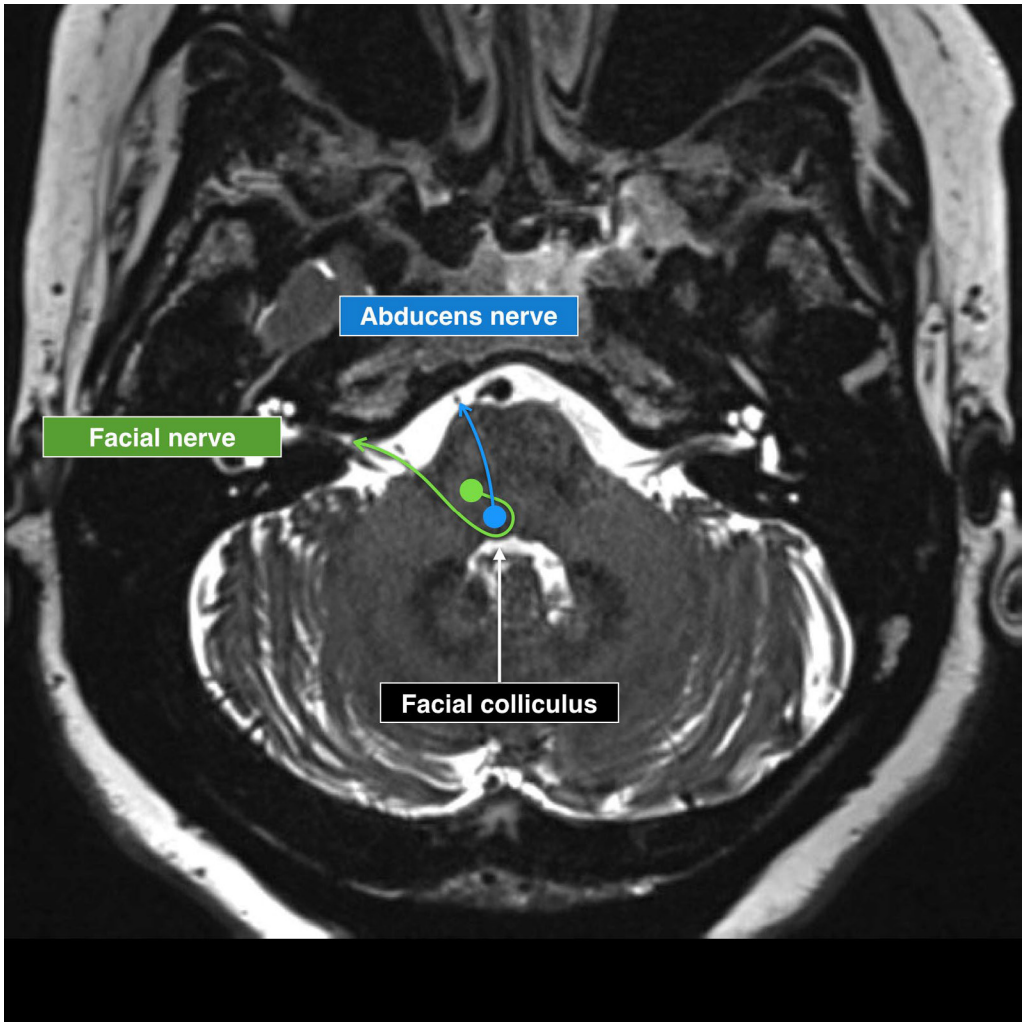


Figure 2: An axial view at the level of the facial and abducens nerves shows the positioning of their nuclei and fibers. Notably, the facial nerve fibers pass behind the abducens nucleus, forming the elevation known as the facial colliculus (Image courtesy of Dr. Frank Gaillard, Radiopaedia.org, rID: 50811) [33]

[Downloaded from wjps.ir on 2026-10-31]

[DOI: 10.61186/wjps.13.3.14]

Campos Benitez and Kaufmann reported a 38% incidence of multiple vessels [13]. Venous compression is less frequent, occurring in only 0.3–3% of cases [34]. The site of compression should be detailed, especially whether it affects the REZ or a more distal part of the facial nerve, such as the cerebellopontine angle. In primary HFS, the REZ is involved in almost 97–100% of cases [13, 35]. For more precise guidance for surgeons, it's recommended to describe the specific points of compression within the REZ, as more proximal compression suggests a higher likelihood that the vessel is a true culprit [35].

The severity of neurovascular compression should also be noted, categorized as simple contact, indentation, or displacement. As the severity increases, so does the likelihood that the vessel is the cause of the HFS (Figure 3). When multiple vessels are responsible, the report should specify the site and degree of compression for each vessel individually to aid in surgical planning [36].

Secondary or symptomatic HFS refers to cases where HFS is linked to various conditions that

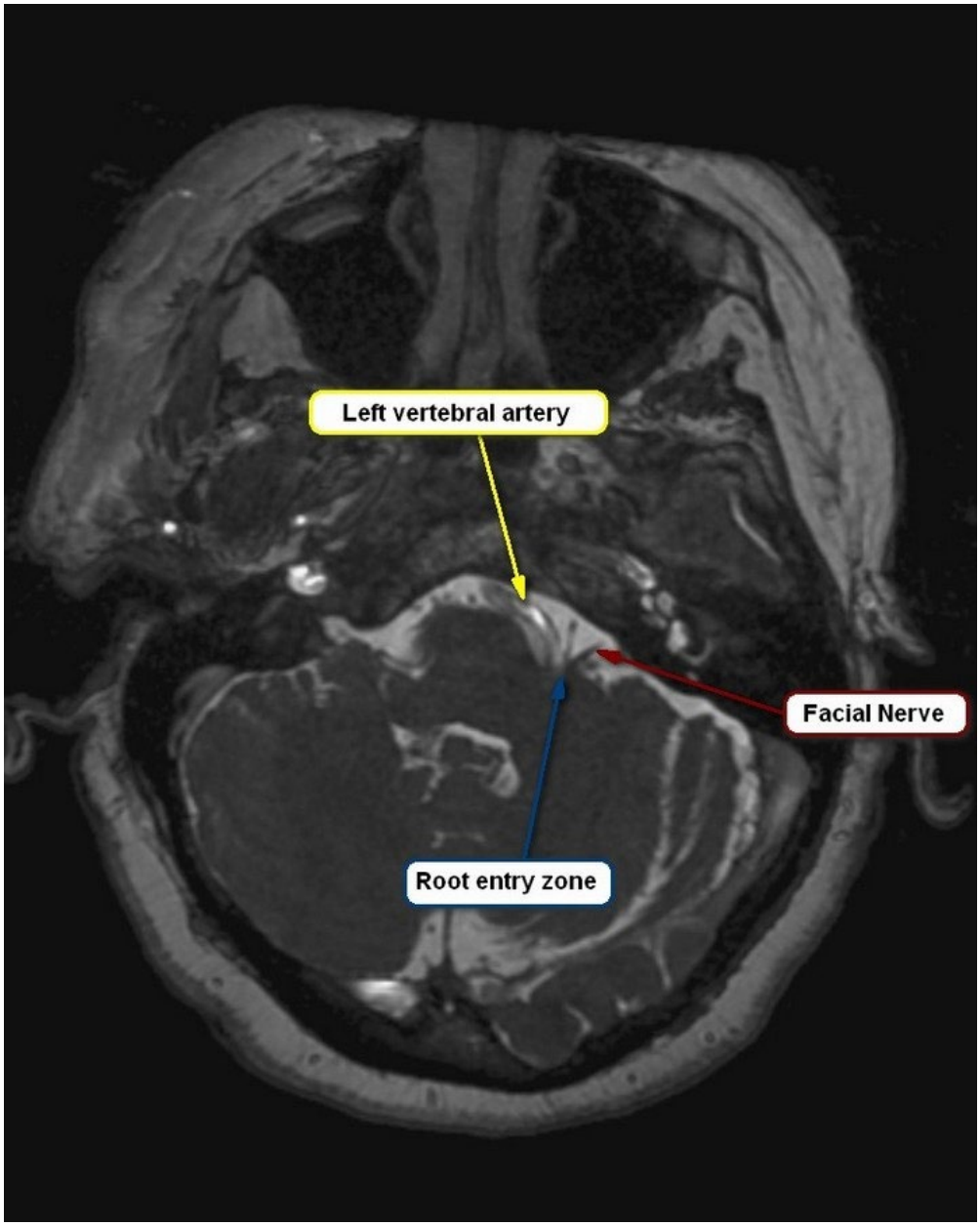


Figure 3: An annotated image of a case of left hemifacial spasm. The left vertebral artery contacting the facial nerve at the nerve root entry zone appears to be the cause of the condition (Image courtesy of Dr. Frank Gaillard, Radiopaedia.org, rID: 2617) [37]

[Downloaded from wjps.ir on 2026-10-31]

[DOI: 10.61186/wjps.13.3.14]

damage the facial nerve along its pathway. MRI, with its diverse pulse sequences, plays a crucial role in diagnosing these conditions and precisely assessing the extent of the lesions, helping clinicians plan the most effective treatment [27]. The incidence of secondary HFS is about a quarter of that of primary HFS [28]. Among the causes, CPA tumors are relatively rare, with a reported incidence of 0.3–2.5% [27, 38]. In a study of 2,050 HFS patients, only nine (0.44%) cases were attributed to CPA tumors, including vestibular schwannoma (2 cases), meningioma (5 cases), and epidermoid cyst (2 cases). DWI is especially helpful in diagnosing epidermoid cysts [38]. Although HFS associated with demyelinating diseases like multiple sclerosis has been documented, its actual incidence is unclear due to its rarity [39].

Vascular abnormalities can also cause HFS, including vertebrobasilar artery dolichoectasia, developmental venous anomalies, arteriovenous malformations, and pial arteriovenous fistulas. Less common causes of secondary HFS include vascular injury, trauma, infection, and inflammation affecting the facial nerve [27].

Advice regarding MRI evaluation of HFS

Transverse MRI images of patients with HFS often show an upward displacement of the lower pons at the point where the facial nerve attaches, near the area of vascular conflict. This is considered an indirect sign of neurovascular compression [40]. Beyond identifying the type, location, and severity of neurovascular compression, certain anatomical factors are important for neurosurgeons. These include cerebellar atrophy or, conversely, a "small posterior fossa," as narrow fluid spaces near the facial nerve's exit zone can increase the likelihood of HFS development [41]. Studies suggest that patients with HFS tend to have a more "congested" posterior fossa compared to those without the condition [42].

Additionally, the flatness of the infratentorial space's superior-inferior dimension may cause lateral deviation of the vertebrobasilar arteries, particularly in patients with arteriosclerosis, intensifying space competition between vessels and cranial nerves. These anatomical variations can not only make vascular compression more likely but also complicate surgical interventions. Several techniques have been proposed to quantify these anatomical features via MRI or CT scans [43].

Other considerations during surgery include any left or right dominance of the sigmoid sinus, arachnoid granulations, or hyperdeveloped mastoid cells, as these may push the surgical approach backward. Developmental venous anomalies or rare bridging veins in the surgical path should also be noted. In patients with arterial hypertension associated with HFS, special attention should be given to ventrolateral medullary compression [44, 45].

CONCLUSION

HFS is crucial for plastic surgeons, given its impact on facial aesthetics and patient well-being. HFS presents unique challenges, as the involuntary muscle contractions can distort facial appearance and complicate surgical interventions. MRI allows for detailed visualization of neurovascular interactions and facilitates informed treatment decisions. The examination begins by ruling out secondary causes, such as tumors or demyelinating diseases, before focusing on the facial nerve's path to locate any vascular compression. Key findings include identifying the type of vessel causing compression—most commonly the anterior inferior cerebellar artery (AICA)—and the exact location and severity of the neurovascular contact.

Advanced MRI techniques, such as 3D imaging and MRA, enhance visualization of both cranial nerves and blood vessels, allowing for a comprehensive assessment of their anatomical relationships. Techniques like fluid-attenuated inversion recovery (FLAIR) sequences are useful for highlighting lesions in secondary HFS cases. Diffusion-weighted imaging (DWI) helps identify lesions causing secondary HFS. Importantly, MRI findings must be interpreted alongside clinical symptoms, as neurovascular contacts can also be present in asymptomatic individuals. In cases of primary HFS, a detailed MRI report should include the type, location, and degree of compression to assist in treatment planning, particularly for surgical interventions.

ACKNOWLEDGMENTS

None.

CONFLICTS OF INTEREST

None.

[Downloaded from wjps.ir on 2026-10-31]

[DOI: 10.61186/wjps.13.3.14]

REFERENCES

1. Shoja MM, Tubbs RS, Khalili M, Khodadoost K, Loukas M, Cohen-Gadol AA. Esmail Jorjani (1042-1137) and his descriptions of trigeminal neuralgia, hemifacial spasm, and bell's palsy. *Neurosurgery* 2010 Aug;**67**(2):431-4.
2. Jankovic J, Brin MF. Botulinum toxin: historical perspective and potential new indications. *Muscle Nerve Suppl* 1997;**6**:S129-45.
3. 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.
4. Nilsen B, Le KD, Dietrichs E. Prevalence of hemifacial spasm in Oslo, Norway. *Neurology* 2004 Oct 26;**63**(8):1532-3.
5. Auger RG, Whisnant JP. Hemifacial spasm in Rochester and Olmsted County, Minnesota, 1960 to 1984. *Arch Neurol* 1990 Nov;**47**(11):1233-4.
6. Park JS, Lee S, Park SK, Lee JA, Park K. Facial motor evoked potential with paired transcranial magnetic stimulation: prognostic value following microvascular decompression for hemifacial spasm. *J Neurosurg* 2019 Dec 1;**131**(6):1780-7.
7. Jankovic J. Peripherally induced movement disorders. *Neurol Clin* 2009 Aug;**27**(3):821-32, vii.
8. Jin Y, Zhao C, Su S, Zhang X, Qiu Y, Jiang J. Residual hemifacial spasm after microvascular decompression: prognostic factors with emphasis on preoperative psychological state. *Neurosurg Rev* 2015 Jul;**38**(3):567-72; discussion 72.
9. Wilkins RH. Hemifacial spasm: a review. *Surg Neurol* 1991 Oct;**36**(4):251-77.
10. Colosimo C, Bologna M, Lamberti S, et al. A Comparative Study of Primary and Secondary Hemifacial Spasm. *Archives of Neurology* 2006;**63**(3):441-4.
11. 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.
12. Park JS, Kong DS, Lee JA, Park K. Hemifacial spasm: neurovascular compressive patterns and surgical significance. *Acta Neurochir (Wien)* 2008 Mar;**150**(3):235-41; discussion 41.
13. Campos-Benitez M, Kaufmann AM. Neurovascular compression findings in hemifacial spasm. *J Neurosurg* 2008 Sep;**109**(3):416-20.
14. Dutton JJ, Buckley EG. Long-term results and complications of botulinum A toxin in the treatment of blepharospasm. *Ophthalmology* 1988 Nov;**95**(11):1529-34.
15. Ramirez-Castaneda J, Jankovic J. Long-term efficacy and safety of botulinum toxin injections in dystonia. *Toxins (Basel)* 2013 Feb 4;**5**(2):249-66.
16. 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.
17. Kurokawa Y, Maeda Y, Toyooka T, Inaba K. Microvascular decompression for hemifacial spasm caused by the vertebral artery: a simple and effective transposition method using surgical glue. *Surg Neurol* 2004 Apr;**61**(4):398-403.
18. Møller AR, Jannetta PJ. Physiological abnormalities in hemifacial spasm studied during microvascular decompression operations. *Exp Neurol* 1986 Sep;**93**(3):584-600.
19. Lee SH, Park JS, Ahn YH. Bioglue-Coated Teflon Sling Technique in Microvascular Decompression for Hemifacial Spasm Involving the Vertebral Artery. *J Korean Neurosurg Soc* 2016 Sep;**59**(5):505-11.
20. Jeon C, Kim M, Lee H-S, Kong D-S, Park K. Outcomes after Microvascular Decompression for Hemifacial Spasm without Definite Radiological Neurovascular Compression at the Root Exit Zone. Life; 2023.
21. Guclu B, Sindou M, Meyronet D, Streichenberger N, Simon E, Mertens P. Cranial nerve vascular compression syndromes of the trigeminal, facial and vago-glossopharyngeal nerves: comparative anatomical study of the central myelin portion and transitional zone; correlations with incidences of corresponding hyperactive dysfunctional syndromes. *Acta Neurochir (Wien)* 2011 Dec;**153**(12):2365-75.
22. Tanrikulu L, Scholz T, Nikoubashman O, Wiesmann M, Clusmann H. Preoperative MRI in neurovascular compression syndromes and its role for microsurgical considerations. *Clin Neurol Neurosurg* 2015 Feb;**129**:17-20.
23. Donahue JH, Ornan DA, Mukherjee S. Imaging of Vascular Compression Syndromes. *Radiol Clin North Am* 2017 Jan;**55**(1):123-38.
24. Tash R, DeMerritt J, Sze G, Leslie D. Hemifacial spasm: MR imaging features. *AJNR Am J Neuroradiol* 1991 Sep-Oct;**12**(5):839-42.
25. Yamakami I, Kobayashi E, Hirai S, Yamaura A. Preoperative assessment of trigeminal neuralgia and hemifacial spasm using constructive interference in steady state-three-dimensional Fourier transformation magnetic resonance imaging. *Neurol Med Chir (Tokyo)* 2000 Nov;**40**(11):545-55; discussion 55-6.
26. Lee MS, Kim MS, Hong IS, Whang K, Han YP. Clinical usefulness of magnetic resonance cisternography in patients having hemifacial spasm. *Yonsei Med J* 2001 Aug;**42**(4):390-4.
27. Chen SR. Neurological Imaging for Hemifacial Spasm. *Int Ophthalmol Clin* 2018 Winter;**58**(1):97-109.
28. Lu AY, Yeung JT, Gerrard JL, Michaelides EM, Sekula RF, Jr., Bulsara KR. Hemifacial spasm and

[Downloaded from wjps.ir on 2026-10-31]

[DOI: 10.61186/wjps.13.3.14]

neurovascular compression. *ScientificWorldJournal* 2014;**2014**:349319.
29. Satoh T, Onoda K, Date I. Fusion imaging of three-dimensional magnetic resonance cisternograms and angiograms for the assessment of microvascular decompression in patients with hemifacial spasms. *J Neurosurg* 2007 Jan;**106**(1):82-9.
30. Cha J, Kim ST, Kim HJ, et al. Trigeminal neuralgia: assessment with T2 VISTA and FLAIR VISTA fusion imaging. *Eur Radiol* 2011 Dec;**21**(12):2633-9.
31. Lutz J, Linn J, Mehrkens JH, et al. Trigeminal neuralgia due to neurovascular compression: high-spatial-resolution diffusion-tensor imaging reveals microstructural neural changes. *Radiology* 2011 Feb;**258**(2):524-30.
32. Deep NL, Fletcher GP, Nelson KD, et al. Magnetic Resonance Imaging Assessment of Vascular Contact of the Facial Nerve in the Asymptomatic Patient. *J Neurol Surg B Skull Base* 2016 Dec;**77**(6):503-9.
33. Gaillard F. Abducens and facial cranial nerves and nuclei. Radiopaediaorg: Radiopaedia.org; 2017.
34. 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 34.
35. Naraghi R, Tanrikulu L, Troescher-Weber R, et al. Classification of neurovascular compression in typical hemifacial spasm: three-dimensional visualization of the facial and the vestibulocochlear nerves. *J Neurosurg* 2007 Dec;**107**(6):1154-63.
36. 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 Feb;**59**(2):93-9; discussion 9-100.
37. Gaillard F. Hemifacial spasm. Radiopaediaorg: Radiopaedia.org; 2008.
38. Lee SH, Rhee BA, Choi SK, Koh JS, Lim YJ. Cerebellopontine angle tumors causing hemifacial spasm: types, incidence, and mechanism in nine reported cases and literature review. *Acta Neurochir (Wien)* 2010 Nov;**152**(11):1901-8.
39. Marin Collazo IV, Tobin WO. Facial Myokymia and Hemifacial Spasm in Multiple Sclerosis: A Descriptive Study on Clinical Features and Treatment Outcomes. *Neurologist* 2018 Jan;**23**(1):1-6.
40. Hermier M. Imaging of hemifacial spasm. *Neurochirurgie* 2018 May;**64**(2):117-23.
41. Parise M, Acioly MA, Ribeiro CT, Vincent M, Gasparetto EL. The role of the cerebellopontine angle cistern area and trigeminal nerve length in the pathogenesis of trigeminal neuralgia: a prospective case-control study. *Acta Neurochir (Wien)* 2013 May;**155**(5):863-8.
42. Cheng J, Fang Y, Zhang H, et al. Quantitative Study of Posterior Fossa Crowdedness in Hemifacial Spasm. *World Neurosurg* 2015 Oct;**84**(4):920-6.
43. Hamasaki T, Yamada K, Kitajima M, Kuratsu J. Flatness of the infratentorial space associated with hemifacial spasm. *Acta Neurochir (Wien)* 2016 Jul;**158**(7):1405-12.
44. Nakamura T, Osawa M, Uchiyama S, Iwata M. Arterial hypertension in patients with left primary hemifacial spasm is associated with neurovascular compression of the left rostral ventrolateral medulla. *Eur Neurol* 2007;**57**(3):150-5.
45. Sindou M, Mahmoudi M, Brînzeu A. Hypertension of neurogenic origin: effect of microvascular decompression of the CN IX-X root entry/exit zone and ventrolateral medulla on blood pressure in a prospective series of 48 patients with hemifacial spasm associated with essential hypertension. *J Neurosurg* 2015 Dec;**123**(6):1405-13.

[Downloaded from wjps.ir on 2026-10-31]

[DOI: 10.61186/wjps.13.3.14]