Case Report

# Developmental vascular anomaly associated hemi facial spasms and botox injections therapy

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## Abstract

Hemifacial spasm (HFS) is characterized by involuntary synchronous contractions or spasms of one side of the face, usually beginning around the eye. They are typically brief, irregular clonic movements but are occasionally tonic. We present a case of a 41-year-old female who presented to the neurology clinic with complaints of recurrent right facial spasms. These involuntary spontaneous movements had affected her quality of life. The neuroimaging revealed the vascular malformation right cranial nerves (CN) VII/VIII complex. It was considered to be responsible for the patient's HFSs. The patient responded well symptomatically to the botox injections without any neurovascular decompression.

**Keywords:** Botox therapy, hemifacial spasms, neurovascular decompression

## Introduction

Hemifacial spasm (HFS) is a movement disorder characterized by involuntary, unilateral, and intermittent contractions of the facial muscles. The pulsatile compression by anterior inferior cerebellar artery (AICA) might contribute to underlying neurovascular abnormality. Patients cannot suppress movements.[1] Unlike other movement disorders, this can continue during sleep. The onset is most commonly in midlife, and complete remission is rare. Both ethical and institutional permission was taken for the case presentation. Botulinum toxin injections are the most effective treatment.[2]

## Case Presentation

We present a case of 41-year-old female presented with complaints of right facial spasms for the past 3 years. The patient also mentioned initial involuntary right upper facial and lower eyelid spasms, which started insidiously around 3 years ago. The paroxysmal involuntary contractions have gradually worsened, becoming more bothersome and have progressed to the right side of the face. These involuntary facial movements had affected her speech as well as her reading but did not typically cause any pain. There is no evidence of compulsive behavior in the current pattern of involuntary movements. She reported that the symptoms tend to become significantly worse during periods of increased psychosocial or emotional stress, as well as with activities requiring a high degree of facial muscle control, such as when attempting to put her make-up on, etc., She noticed the symptoms to be improved, albeit transiently, with sensory tricks such as wearing her glasses or trying to hold certain facial gestures. The magnetic resonance imaging (MRI) with and without contrast showed a small blood vessel traveling perpendicularly to the cranial nerve (CN) VII/VIII complex in the intra-dural space. The abnormal blood vessel was coursing between the two cranial nerves and slightly distorting the CN VII/VIII complex due to the nerve's circumduction around the

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blood vessel [Figure 1]. Although this vascular anomaly does not create a mass effect on the nerves radiographically, it may be the structural abnormality responsible for the patient's right-sided abnormal facial contractions. The frequency of HFS symptoms improved remarkably after the botox injections and did not require neurovascular decompression at the moment.

## Discussion

The HFS is a rather benign condition characterized by involuntary synchronous contractions of one side of the face. The onset of HFS is most common around ages 40–50, are typically brief, and will begin around the eye involving the orbicularis oculi muscles.[1] Over time, these spasms can spread to affect other facial muscles that are innervated solely by the facial nerve (cranial nerve VII). The underlying cause of HFS is typically by compression of the facial nerve by a blood vessel as the facial nerve exits the brainstem. The region of most common compression is at the transition zone between central and peripheral myelination, which creates the nerve's vulnerability to compression. During development, the facial nerve will split into two by the end of the fifth or sixth week of gestation, which is before any of the cerebellar arteries appear. Any change during this time may be a cause of the developmental anomaly leading to compression of the nerve by the vasculature.[2] Alternatively, there are other causes but occur at a much rarer level, such as lesions in the cerebellopontine angle, that is, Meningiomas and Schwannomas. Specific pathology that occurs in the brainstem may also lead to symptoms such as multiple sclerosis (MS), brainstem infarctions, and even rare cases of Bell's palsy. A vital distinction should be made when diagnosing HFS by ruling out any other potential causes of abnormal facial movements.[3]

There needs to be a conclusive clinical and radiographic evidence to rule out other potential causes of vascular compression. The MRI with and without contrast, is the most useful modality to visualize the cerebellopontine angle to rule out tumors or brainstem lesions.[3] The imaging can also display the potential pathologic vascular abnormalities that make up the vast majority of HFS, especially by the posterior inferior cerebellar artery (PICA) or the anterior inferior cerebellar artery (AICA), like in our case. The management modalities range from almost no treatment to microvascular decompression or botulinum toxin injection.[1] In our case, we found a vascular anomaly causing the HFS that likely occurred during the developmental period. An MRI with and without contrast showed a small blood vessel, probably the AICA, traveling perpendicularly to CN VII/VIII complex, causing slight distortion. In another study by Oh *et al.*, it was hypothesized that AICA passes through the facial nerve during developmental stages.[4] Since this abnormality is not life-threatening, all treatment options, including expectant management, surgery with microvascular decompression, botulinum toxin injection, among other various medications, need to be carefully considered. Microvascular decompression is the permanent treatment option but carries many risks involved with any surgical procedure.[5,6] The developmental vascular anomaly associated with our patient was not causing any significant mass effect.

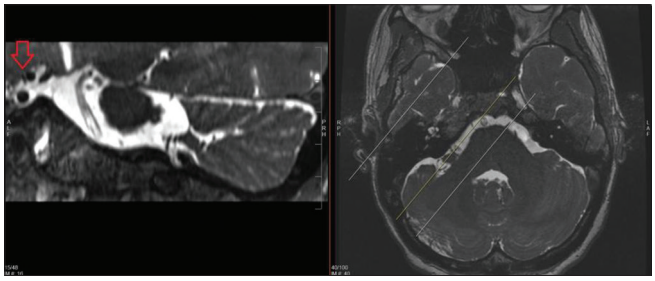

**Figure 1:** Magnetic resonance imaging (MRI) brain with and without contrast showed a small blood vessel traveling perpendicularly to the cranial nerve (CN) VII/VIII complex in the intra dural space

After weighing out the risks and benefits of surgery, we decided to treat the patient with botulinum toxin injections. However, the most significant disadvantage is due to its purely intermittent symptomatic treatment resulting in the patient having to return approximately every 3–4 months. In retrospect, this patient could have potentially benefited from performing the microvascular decompression for complete alleviation of the symptoms. However, with all things considered, our patient has been asymptomatic so far with the botulinum toxin injections. It becomes imperative to diagnose such conditions by primary care physicians and refer to Botox or microvascular decompression after brain MRI abnormality.

## Conclusion

We conclude that for all the patients presenting with complain HFSs the structural causes need to be ruled out. Refractory cases might need urgent surgical intervention for neurovascular etiology.

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### Conflicts of interest

There are no conflicts of interest.

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