Hemifacial Spasm: A Case Report and Review of Literature

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ABSTRACT A hemifacial spasm is characterized by the presence of tonic or clonic, intermittent and involuntary unilateral contractions of the muscles supplied by the facial nerve, usually around the eyes, cheeks, mouth and neck. A case is presented in a patient with twitching in the right lower eyelid with irradiated pain in the temple and upper lid edema. Causative factors and management strategies are emphasized to enhance the recognition and understanding of this condition.

A hemifacial spasm is a condition characterized by the presence of tonic or clonic, intermittent and involuntary unilateral contractions of the muscles supplied by the facial nerve, usually starting around the eyes before progressing to the bottom of the cheek, mouth and neck. Although it affects mostly females, there are reports of the condition in males. The condition has been reported mainly on the left side with a progressive evolution starting in the orbicularis oculi and orbicularis oris muscles.2-3 These symptoms can be motor or nonmotor, producing movements and autonomic signs. The intensity of this disorder can vary and intensify with factors such as stress, intense light and masticatory movements. On average it affects people around the age of 44 and incidence has been reported in 9.8–11/100,000 persons.4,5

The cause seems to be of neuronal origin and is most often attributed to compression of the facial nerve or damage to the brain stem. In cases where there is a confirmed lesion, treatment is decompression or removal of structures. However, sometimes no specific cause is identified. This complicates the therapeutic approach, which should be directed to the recovery of function and aesthetics.6 Other conditions reported as causative agents are the aneurysm spasm,7 occupying masses8 and neuronal hyperactivity.9-10

Dentists have a role in the diagnostic process due to the possibility that the initial symptoms are related to discomfort and dysfunction of the jaws. Additionally, episodes of spasm can cause damage to oral tissue, making the dentist an important part of the treatment plan. The purpose of this article is to review the signs and symptoms, as well as management of hemifacial spasms. This case of a nonspecific cause is presented to enhance understanding of hemifacial spasms and the treatment approach.
Case Report

E.C.F is a 62-year-old male who was seen at the Orofacial Pain Clinic, Dentus Group in Costa Rica. His chief complaint was a twitching in the right lower eyelid (FIGURES 1A, 1B), with irradiated pain in the temple and upper lid edema that he had experienced for four years but that had worsened the last two years. Imaging studies (CAT scan and MRI) were noncontributory. The cranial nerves examination showed a CNII with smooth extraocular movements, no double vision and CNIII, CNIV and CNVI within normal limits. There was equal pupil reaction to light. CNV had symmetrical and normal response to light touch and pinprick with normal motor function. Regarding facial expression, there was lacrimation and ptosis in the right eye and asymmetrical pull of the upper lip on the right side. The patient’s forehead was flat. There was a reduction of hearing of the right side, which was confirmed by audiometry. Palatal elevation was equal and the uvula was located at the midline. His finger-nose coordination was normal. His masticatory muscles were not tender to palpation, range of jaw motion was normal and there were no intraoral lesions. The differential diagnosis included benign essential blepharospasm, Bell’s palsy, trigeminal neuralgia, damage in the facial nerve and cluster-tic syndrome. However, based on the clinical presentation and evolution, a diagnosis of hemifacial spasm was favored because he had had episodic pain accompanied by ptosis and twitching of the eye.

He had been treated by a neurologist and an otolaryngologist and had pharmacotherapy (carbamazepine 200mg qid, pregabalin 150 mg hs, sodium valproate 500 mg hs, clonazepam 0.5 mg hs, lorazepam 1 mg hs), but the medications were discontinued either because of the side effects at the indicated doses or because the patient got no relief. He also received a series of three botulinum toxin injections four months apart in the temporalis and orbicularis oculi. No significant changes with these approaches were reported.

Considering the possible contribution of the autonomous nervous system, a calcium channel blocker (verapamil 160 mg) was prescribed together with a muscle relaxant (cyclobenzaprine, 10 mg hs) to address the stress-triggered contraction. A series of four physical therapy sessions were performed, including the use of electric stimulation, ultrasound and facial massage. None of the described treatments produced a change in the symptoms.

The case was presented in a roundtable session of neurologists, and a decompression surgery was recommended considering the possibility of compression of the trigeminal root not evident in the MRI of the cranial region. However, considering the risks of the surgery and the uncertainty of the results, the patient declined this procedure. In order to help the patient with the episodic twitching, biofeedback intervention was suggested with the hope of him being able to control his musculature. The patient is now under the care of the psychologist.

Discussion

As in this case and many others, the causes of the disease are not clear and may be multiple, including vascular compression, movement disorders, neuronal demyelination and the presence of other diseases. One of the most common theories is that the facial nerve is pressed by blood vessels at the level of output from the nerve to the central nervous system. This etiology is also supported by other studies. Castiglione et al. state the facial nerve compression is by the lower anterior cerebellar artery, although one must rule out a pathological condition. Another possible cause of facial nerve compression has been linked to a blood defect in the sensory root of the nerve. An alternative theory is the possibility of artery and vein compression at the spinal cord level. Genetic and psychological factors have also been linked to hemifacial spasms, and in this case there was no family history reported. In cases with atypical features, psychogenic etiologies should be considered. Although the patient in this case reported no psychogenic conditions, the psychological impact of this...
condition is very important because the patient tends to move away from daily activities as a result of functional and aesthetic alterations that occur with the spasms. Interference with social life has been reported in up to 90 percent of the cases, leading to isolation and depression. In this case, the patient reported an impact on social and daily activities because of the spasms that could occur at any moment. For example, if a spasm occurred when he was driving he would have to pull over because his vision was affected.

There was a documented coexistence of hemifacial spasms with migraine headaches among the cases reported in the literature. Spasms can behave as activators thus complicating the management of headaches; however, they are not a causal element if one has predisposing features. The patient in this case did not have migraine or other headache issues.

As with any medical condition, the diagnosis process should include history, clinical examination and the use of additional diagnostic tools. In this case, a bone scan and neuroimaging enabled us to rule out potential underlying conditions that were unremarkable in the findings. In the case of a hemifacial spasm, other imaging tools can be useful. Functional magnetic resonance imaging can detect changes in neuronal activity. Voxel-based morphometry can assess changes in gray matter, thalamus, putamen and dorsolateral spinal region, which are areas that have an impact on motor control.

Treatment depends on the diagnosis and identification of causal or etiological elements. In general, hemifacial spasms are addressed with surgical options, radio frequency, pharmacotherapy, a behavioral approach and complementary or alternative techniques. In this case, pharmacotherapy was one of the first lines of treatment, although medications tend not to be effective long-term treatments of hemifacial spasms. Baclofen, clonazepam, carbamazepine, gabapentin, phenytoin or orphenadrine are considered first-line medications that may be used to provide transient relief. Membrane stabilizers or drugs related to gamma aminobutyric acid (such as carbamazepine and gabapentin), which can be used as both monotherapy and complementary to other techniques, have been described as an alternative therapy.

Botulinum toxin is also reported as an alternative therapy, but its effectiveness is limited in controlling symptoms. It has been indicated that after application of botulinum toxin, there is an improvement ranging from eight days to 14.8 weeks in approximately 73.7 percent of patients. The most common adverse effects reported were erythema (5 percent), facial asymmetry (3.6 percent), ptosis (3.4 percent) and diplopia (3.2 percent), which are conditions that sometime accompanied hemifacial spasms from the start. It is important to point out that the botulinum toxin effect is temporary; the procedure must be repeated periodically. In this case, pharmacotherapy has been ineffective in managing the patient’s symptoms.

Microvascular decompression is the surgical treatment of choice for hemifacial spasm. It is considered a safe and effective alternative, although complications as a result of the procedure include facial paralysis and hearing loss. Other techniques have been reported that separate the vessels, such as placing Teflon between the vein and facial nerve. This procedure generally improves the condition, but there is a lengthy recovery time of up to 10 months. Sometimes it is necessary to repeat these procedures because of small vessels that are not identified in the first approach. Improvement because of these surgical procedures can last up to five years. The use of radiofrequency is another alternative that damages the nerves directly by reducing their firepower. This approach is considered conservative and has been described for refractory spasms and in cases where decompression surgery has not been efficient.

Complementary therapies, such as acupuncture, biofeedback and facial massage, have also shown benefits. Biofeedback interventions using electromyography have suggested that the length of the spasms are reduced but not the frequency. Although there are insufficient data on its effectiveness, it has been observed that individuals whose symptoms have greater severity are the ones who tend to seek these alternatives. However, in this case physical therapy, including the use of electric stimulation, ultrasound and facial massage, was unsuccessful. The biofeedback and psychological intervention results in this case are too early to tell.
Conclusions

In the case presented, the patient had experienced hemifacial spasms for more than four years. The necessity of implementing a detailed history and comprehensive examination with the adjunctive diagnostic instruments, such as radiographic imaging, cannot be overemphasized. Imaging studies have been noncontributory and treatments to date unsuccessful. The cause in this case, although not clear, appears to be of neuronal origin. In these cases, it is important that dentists have knowledge of the treatment and diagnostic processes to correctly guide patients.

REFERENCES

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