Hemifacial Spasm with Facial Paresis: A Case Report
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Abstract
Hemifacial spasm is characterized by tonic and clonic contractions of the muscles innervated by the ipsilateral facial nerve. Magnetic resonance imaging and angiography studies frequently demonstrate vascular compression of the root exit zone of the facial nerve. Importantly, an underlying space-occupying lesion needs to be excluded in patients with associated atypical features such as facial numbness and weakness. This is a case report of a dental patient with involuntary facial movements which were noted during history recording. Thorough clinical examination was carried out and magnetic resonance imaging revealed a tortuous left vertebral artery causing mild impingement of root exit zone of left facial nerve confirming the diagnosis of hemifacial spasm. The etiology, clinical features, treatment options and differential diagnosis of hemifacial spasm are discussed.

Key Words: Facial Paresis; Facial Nerve; Hemifacial Spasm; Root Exit Zone; Vertebral Artery; Vascular Abnormalities.

Introduction
Hemifacial spasm (HFS) is a movement disorder generally as a consequence of vascular abnormalities around the root exit zone of the ipsilateral facial nerve. It is characterized by paroxysms of tonic or clonic contractions involving predominantly periorcular and perioral facial musculature. HFS may rarely be bilateral, manifested by asynchronous contractions of facial muscles on each side.

For many years, HFS was regarded as idiopathic, however, it is now recognized that vascular compression of the seventh cranial nerve, at its exit zone from the brain stem, is the primary cause. In most cases this is due to an aberrant or tortuous vessel, less commonly it may be seen with basilar artery aneurysms or arteriovenous malformations. A rare, but recognized, cause of HFS is tumour compression; it has been reported in patients with acoustic neuromas, meningiomas and cholesteotomas as well as epidermoid tumour and lipomas. It must be differentiated from other causes of involuntary facial movements, all of which can potentially affect quality of life. As facial twitchings are frequently attributed to stress and anxiety, the diagnosis of HFS may be missed.

The total prevalence of HFS is in range of 9.8 to 11 per 100,000 in the total population. HFS frequently causes social embarrassment and affects the quality of life. Facial grimacing and contortions are often thought to be a “physiologic” response in individuals suffering from pain, stress, and anxiety.

Case Report
A male patient, aged about 40 years reported to the department of oral medicine and radiology, with a complaint of pain in the left upper molar on food lodgment since 2-3 months. While recording history, asynchronous blinking of left eye and facial twitching of left cheek was noted. On further questioning, patient revealed paresthesia around the corner of the mouth on left side of the face. He also complained of food lodgment in the upper left buccal vestibule. General examination did not reveal anything significant.

Clinical examination revealed loss of buccinator function as the patient was unable to blow out the left cheek. Uncontrolled twitching of the left eye and facial muscle contraction of the left side was also noted which increased while talking. The facial muscles were relaxed in between contractions. Patient also related increased twitching during stress, fatigue, anxiety and the twitching persisted during sleep. Intra oral examination revealed chronic irreversible pulpitis in relation to 26. An intraoral periapical radiograph was taken and root canal therapy was advised. A provisional diagnosis of hemifacial spasm...
was made and referral to neurologist confirmed the diagnosis.

The magnetic resonance imaging (MRI) revealed a tortuous left vertebral artery causing mild compression on the brain stem on left side with a vascular loop causing mild impingement of root exit zone of left facial nerve. (Figure 1) Patient was treated by the neurologist with a combination of Clonazepam 0.25 mg and Benzhexol 2 mg, for a period of 1 week and recalled. The patient reported some relief from twitching. He wanted to discontinue treatment because of drowsiness. An alternative treatment plan with botulinum toxin was suggested but the patient refused the treatment.

Figure 1: Left vertebral artery (black arrow) causing compression at the root exit zone of VII cranial nerve.

Discussion

Patients with Hemifacial spasm usually present between 40 and 50 years of age. They frequently complain of involuntary eye closure. The facial spasm is spontaneous and may persist during sleep. In a large series of 158 HFS patients, the initial site of onset was the orbicularis oculi muscle in 90%, the cheek in 11% and the perioral region in less than 10% of cases. Over months to years, the spasms spread gradually to other muscles innervated by the ipsilateral facial nerve in a synchronous manner. Bilateral HFS was occasionally reported. Concomitant trigeminal neuralgia, though uncommon, has been reported with HFS. HFS is a chronic disease, with spontaneous resolution in less than 10% of patients. Clinically obvious facial weakness may be seen in long-standing cases. Mild paresis and paresthesia has been observed in the other studies.

Symptoms are frequently aggravated by stress, fatigue, anxiety and voluntary facial movements. Relaxation, alcohol intake, touching the affected areas, and exercise reportedly improve symptoms in some patients. Although detailed pathophysiological mechanisms underlying hemifacial spasm still remain unclear, the pathophysiological basis of hemifacial spasm that has been proposed is that the compression of the facial nerve by normal or abnormal vascular structures at its brainstem exit contributes to inducing abnormal excitation of motor neurons in the facial nerve nucleus.

In a study involving 366 patients with HFS, the cause was thought to be arterial in 323 patients (88%), with multiple arteries involved in 96 (26%), venous in 7 patients (2%) and both arterial and venous in 33 patients (9%). Some authors have emphasized that a vessel merely being in contact with the root exit zone of the facial nerve is not sufficient to cause symptoms, but rather that the root exit zone must be compressed or deformed by the vessel. However, pathophysiological mechanisms other than the vascular compression may contribute to the induction of hemifacial spasm. It is known that hemifacial spasm can appear as a sequel of peripheral facial nerve lesions, such as Bell’s palsy or traumatic facial nerve damage. In addition, several reports have documented familial occurrence of hemifacial spasm. Early onset post traumatic HFS has also been reported due to a hematoma formation in lower pons. A unusual case of HFS associated with Glomus jugular tumor has also been reported.

Involuntary facial movements are not uncommonly encountered in the general population. As facial twitches are frequently attributed to stress and anxiety, the diagnosis of HFS may be missed. Other aetiological causes of involuntary facial movements such as tardive dyskinesias, myokymia, tics, cranial dystonia, and psychogenic facial spasm must be differentiated from HFS, as early diagnosis allows institution of appropriate treatment.

Microvascular decompression (MVP) of the facial nerve at the cerebellopontine angle,
the most common surgical procedure carried out today, results in markedly improved HFS in the majority of patients, with success rates of 90% in some series.\(^4\) Common complications of MVP include temporary or permanent dysfunction of facial or auditory nerve, with 7–26% suffering from hearing loss in some reports.\(^5\) Other complications, such as lower cranial nerve dysfunction and intracranial infections are less common.\(^18\) Botulinum toxin injections have also been effectively used. Despite the variation in the techniques of botulinum toxin injections and the lack of a validated scale to assess treatment response, good to excellent improvement was reported in 75% to 100% of the patients.\(^4\)

Drugs such as Carbamazepine, anticholinergics, baclofen, clonazepam and haloperidol have also been used. Sedation is a common adverse effect of all these medications, particularly when high doses are used.\(^19\) Gabapentin was administered in a study, at a dose ranging from 900 to 1,600 mg daily, with rapid and clear improvement of spasms and absence of any remarkable adverse effects.\(^20\)

**Conclusion**

HFS is caused by vascular compression of seventh cranial nerve as it exits the brain stem in high percentage of cases, but it could also be caused due to other mass lesions. The dentist plays an important role in early recognition and also helps in instituting a prompt treatment.

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