IMMEDIATE SYMPTOMATIC RELIEF AFTER MICROVASCULAR DECOMPRESSION FOR HEMIFACIAL SPASM: A CASE SERIES

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ABSTRACT

Hemifacial spasm is a movement disorder characterized by intermittent, involuntary clonic or tonic clonic contractions of muscles innervated by the ipsilateral facial nerve.it is commonly caused by vascular compression of the facial nerve at its Root Exit Zone (REZ) from the brainstem. Anterior inferior cerebellor artery, posterior inferior cerebellor artery, vertebral artery commonly compress the facial nerve. Usual symptoms are progressive involuntary facial twitching. MRI brain and MRA are advised to know about neurovascular compression.four case of hemifacial spasm are reported diagnosed on clinical grounds, and MRI brain and MRA are advised. our objective is to assess the surgical outcome of microvascular decompression (MVD) for Hemifacial spasm (HFS).

***Key Words**: Hemifacial Spasm (HFS), Microvascular decompression (MVD), Facial nerve decompression*

This case series may be cited as: Ali M, Haq N, Usman M, Ishaq M. Immediate symptomatic relief after microvascular decompression for hemifacial spasm: A Case Series. J Postgrad Med Inst 2013; 27(1): 100-4.

INTRODUCTION

Hemifacial spasm (HFS) described first by Gowers in 1884[1], is a movement disorder characterized by intermittent, involuntary clonic or tonicclonic contractions of muscles innervated by the ipsilateral facial nerve, with the contractions being asymmetrical and asynchronous[2]. HFS typically starts with intermittent twitches in the orbicularis oculi muscle. The symptoms usually progress gradually in frequency and severity and spread downward to the ipsilateral facial muscles[3]. There is now considerable evidence that primary HFS is, in almost all cases related to a vascular compression of the facial nerve at its Root Exit Zone (REZ) from the brainstem[4,5]. Hemifacial spasm may also be induced by tumor[6,7], cerebral vascular aneurysm[8,9], arteriovenous malformation (AVM)[10,11] or bony deformity[12].

A recent service-based epidemiologic study in Oslo showed a prevalence of 9.8 seeking treatment for HFS per 100,000[13]. The diagnosis is made clinically. In some cases, electromyogram may be helpful, and magnetic resonance imaging (MRI) is recommended to rule out rare causes such as tumor. Magnetic resonance angiography (MRA) provides useful information concerning the existence of offending vessels[14,15] .Microvasculor decompression is treatment of choice besides medications and botulinum toxin. There are several studies showing that microvascular decompression (MVD) is an effective and a safe treatment in HFS[16,17]. The common offending vessels are anterior inferior cerebellar artery (AICA) and posterior inferior cerebellar artery (PICA). But, in some cases main arteries such as vertebral artery and basilar artery can be the initiatives generating transmission of the pulse pressure to the nerve[18]. We conducted this case series to know about the immediate symptomatic relief after MVD for HFS.

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Date Received: May 25, 2012
Date Revised: November 1, 2012
Date Accepted: November 11, 2012

CASE REPORTS

CASE 1: A 65 years old male patient presented with 27 months history of progressive involuntary facial twitching on right side. On physical examination he had no neurological deficit. MRI brain was normal. He underwent right side suboccipital retromastoid craniectomy. AICA was found compressing the facial nerve. Vessel was dissected from the nerve and surgical prothesis was put in. Patient was observed after 24hours and symptoms were relived. On 4th post operative day patient was discharged. Patient follow up was done on 2nd and 4th week (Figure 1).

Table 1: Tabulated description of the cases

Variable	Case 1	Case 2	Case 3	Case 4
Age (in years)	65	52	42	38
Sex	Male	Male	Female	Male
Symptom Duration (in months)	27	18	36	60
Side Involved	Right	Left	Left	Right
Offending vessel	AICA	AICA	Basilar Artery	AICA

CASE 2: A 52 year old gentle man had history of typical left sided hemifacial spasm, with sensorineural hearing loss. MRI brain was normal. Left retrosigmoid craniectomy was done, AICA was found to be the offending vessel. Vessel was dissected from the nerve and surgical prosthesis was put in. Recovery from symptoms was excellent. Patient was discharged form hospital on 4th post op day. Follow up was done on 2nd and 4th week (Figure 2).

CASE 3: A 42 year old female patient had involuntary muscle contractions on left side of face from 36 months. Neurological examination was unremarkable. MRI brain was normal. Left side retrosigmoid craniectomy was done. Basilar artery was found compressing facial nerve. Basilar artery was dissected from the facial nerve and muscle patch was put in. Patient had excellent recovery from symptoms. She had sensorineural hearing loss after surgery, which improved on subsequent follow up visits. Hospital stay was 8 days (Figure 3).

CASE 4: A 38 years old male patient presented with 60 months history of progressive involuntary facial twitching on right side. On physical examination he had no neurological deficit. MRI brain was unremarkable. He underwent right side suboccipital retromastoid craniectomy. AICA was found compressing the facial nerve. Vessel was dissected from the nerve and surgical prothesis was put in. Patient was observed after 24 hours and symptoms were relieved. Despite repeated request, this man refused to allow his pictures to be published.

The tabulated description of the cases is given in Table 1.

DISCUSSION

Few studies have been undertaken to elucidate the prevalence of HFS in the general population. An US study revealed an average prevalence of 11 per 100,000 population, while a Norwegian study reported a prevalence rate of 9.8 per 100,000 people. Both studies demonstrated an increased prevalence in women with an average female to male ratio of approximately 2:1[19]. Though no apparent cause may be found in some cases, the most common finding is a focal compression of the root exit zone of the facial nerve by an aberrant, atherosclerotic or ectatic vessel, commonly the posterior inferior cerebellar artery or anterior inferior cerebellar artery, or a combination of anterior inferior cerebellar artery and posterior inferior cerbellar artery, or the vertebral artery[20]. It is therefore not surprising that HFS has also been termed a hyperactive dysfunction syndrome of the facial nerve [21].

The differential diagnosis includes blepharospasm, facial myokymia, habitual tic, tardive dyskinesia, etc. Typical HFS begins in the eyelids and spreads to the lower facial muscles. The intensity of symptom can vary from the intermittent mild spasm to sustained tonic muscle contraction. There is a tendency that the longer the spasm continues, the higher the frequency and the intensity become[22]. MRI brain and MRA are advised to know about neurovascular conflict.

Medical treatment of HFS, using carbamazepine, clonazepam, baclofen, anti-cholinergic, gabapentin or orphenedrine effect is often transient. Treatment with botulinum A toxin for facial dyskinesiae including HFS is well known. This technique requires multiple injections into the muscles and is effective for two to three months, when it must be repeated. Thus injections are for an indefinite time. The quoted success rate for significant relief of symptoms is 70-75%. The procedure can give rise to complications including ptosis, exposure keratitis, diplopia, epiphora, drooling, and strabismus. Excess dosage at the time of injection can result in temporary facial paralysis[23,24].

Neurovascular compression has been accepted as the etiology of HFS, Janneta popularized the concept of MVD as an effective form of treatment, and MVD is currently the mainstay of treatment for HFS[25]. The most frequently reported complications of MVD include

Figure 1: Pre and Post-operative pictures of Case 1

Figure 2: Pre and Post-operative pictures of Case 2

Figure 3: Pre and Post-operative pictures of Case 3

hearing loss, diplopia, facial palsy, brainstem or cerebellar infarct, hematoma, ataxia, CSF leak, meningitis and hydrocephalus[26].

In our case series we had 4 patients with typical HFS. Out of 4, 3 were males and 1 was female. We had follow-up of all patients for upto 4 weeks as, at 1 week, 2 weeks and then at 4 weeks. Three patients had complete symptom relief within 24 hour of surgery, while 1 patient after 2 days and the same patient continued to have minor problem in hearing (developed sensorineural hearing loss), which improved after 4 weeks. All of them were satisfied with the procedure. A causative vessel was found on the root exit zone of all the patients, 3 had anterior inferior cerebellar artery and 1 had basilar artery. There was no mortality in the series. Success rate of MVD in our case series is 100 % and is comparable with published series, which is 94.6% reported by Ryong et al and 98.2 % reported by Jun et al[27]. Dibyendu K. Ray etal conducted study on Surgical Outcome and Improvement in Quality of Life after Microvascular Decompression for Hemifacial Spasms, had success rate of 90%, determined by complete res-olution of symptoms[28]. Moffat et al noticed that they dropped from 93.3% at the 3-month follow-up to 80% at 16 months of follow-up[29].we had success rate of 100%, However, given the small sample size of our study, it was not possible to determine with convincing statistical accuracy whether the degree of severity of preoperative symptoms had any influence on either the success of surgery or the time taken for resolution of symptoms. Another limitation of our case series is that, we had follow up of only 1 month.

CONCLUSION

All the patients of hemifacial spasm got symptomatic relief with Microvascular decompression, but minor complication (partial hearing loss) occurred in single patient.

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CONTRIBUTORS

All the authors contributed significantly to the collection of cases and writing of manuscript.