

Surgical Management of Hemifacial Spasm

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ABSTRACT

Back ground: Hemifacial spasm is a syndrome of spontaneous and gradual onset that has its hallmark the intermittent twitching of the muscles of facial expression on one side of the face. Characteristically, a series of twitches, increasing in frequency and intensity are followed by a sustained spasm lasting a few seconds to a few minutes. Hemifacial spasm occurs almost exclusively in adults. It presents primarily as a problem of appearance for the affected individual, but when it becomes marked it may interfere with daily activities. A wide variety of agents have been mentioned as the cause of Hemifacial spasm but in most of the patients the cause seems to be the compression of the facial nerve by a neighbouring vessel near brain stem. Various treatment modalities have been employed with reference to the presumed cause but Micro vascular Decompression of the facial nerve in the posterior fossa gives the most promising results.

Material and methods: A series of 14 patients of Hemifacial spasm presenting to us in the Department of Neurosurgery Jinnah hospital Lahore from August 2006 to September 2009 were studied. Micro vascular decompression of the facial nerve was done in all these patients. Female were 08 and males were 06. The average age was 41 years and average conservative management period was 05 years. Out of 14 patients 06 had right sided and 08 had left sided involvement.

Results: In 11 patients an arterial loop was found responsible and venous loop pressure was in one present and both arterial and venous loops in 02 patients. There were minimal complications and only one recurrence has been noted till today.

Key words: Hemifacial spasm, twitching of muscle, sustained spasm

INTRODUCTION

Hemifacial spasm is not only a disease but it is also a social stigma as the spontaneous facial muscles contractions on one side of the face^{1,2}, more pronounced in anxiety, involves females more than the males. The world wide incidence is 14.5/100000 in females and 7.4/100000 in males⁵. There are two types of the hemifacial spasms ,Typical and Atypical. In typical variety the spasm starts from the orbicularis oculi and then involves the other muscles of the face. Atypical spasm³ involves the orbicularis oris first and then spreads to other facial muscles. This is a problem of the adult age⁴ but in can occur in extreme of ages with a mean age of 45 year. Left side of the face is more often involved. various theories have been proposed during the century and current concept suggest a vascular etiology predominantly. At time other pathologies like aneurysms ,neoplasms and osseous problems of the skull base can be the causative agents. usually the typical hemifacial spasm is due to compression at the antero-caudal aspect of the root exit zone and posterorostral vascular loop compression at the root exit zone would lead to an Atypical hemifacial spasm.

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MATERIAL AND METHODS

This is a retrospective study at Jinnah hospital Lahore from August 2006 to September 2009. Total 14 patients were operated after admission in the department. All these patients were presented in the outpatient department.

Out of these 14 patients females were 08 and males were 06.

Total No. Of patients	Females	Males
14	08	06

Left side of the face was more affected i.e. in 08 patients and right side was involved in 06 patients

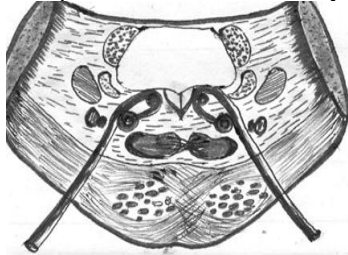
Side involved	Left	Right
	08	06

The ages range from 27 years to 60 years
Diagnosis of HFS is essentially on clinical grounds^{6,7}, while the investigations like X rays skull to rule out skull base abnormalities and diseases like paget,s disease, CT Scan brain⁸ to rule out the neoplasms of the posterior fossa, MRI Brain^{9,16} to rule out the AVM of the brain, any aneurysm of the vessels and also the brain tumours

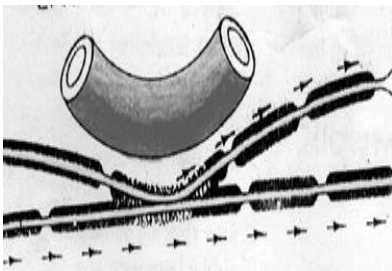
In our study, diagnosis of all the patients were made clinically and CT or MRI brain was done in all the patients to rule out the organic lesions.

HFS was differentiated clinically from other abnormal facial movements like blepharospasm (100, facial myokimias and most common Tics.

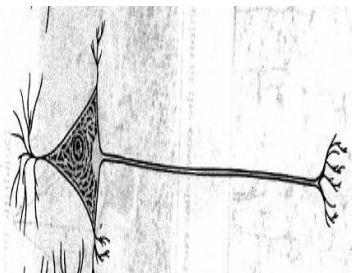
Those who believe in vascular loop compression¹¹ as the cause of the HFS explain the pathogenesis on the basis of Ephaptic transmission while abnormal stimulation of the 7th cranial nerve nucleus and aberrant regeneration of the neurons other theoretical possibilities favoured by few others.



Abnormal stimulation of nucleus



Ephaptic stimulation

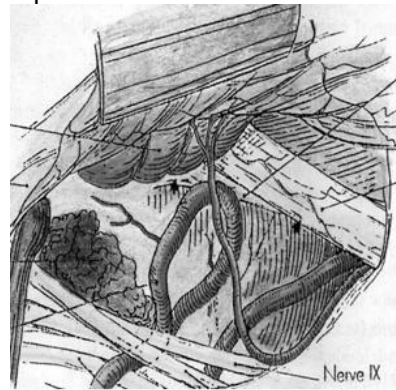


Aberant Regeneration

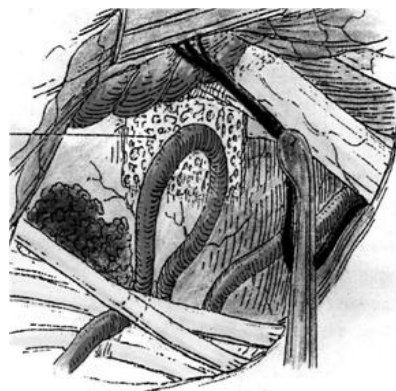
The treatment of the HFS is very confounding. Various types of treatments have been tried over the course of time during last many decades because of unclear nature of the disease. Various conservative treatments like pharmacotherapy¹⁵, massage and physiotherapy have been of an occasional help. Radiotherapy and nerve injury are of some help but without a lasting relief and they are essentially destructive procedures. Where a SOL or AVM or aneurysm has been the cause, treatment is directed towards them. The only non destructive procedure offering permanent relief significantly is micro vascular decompression of the facial nerve. All the patients in our study have taken conservative treatment like medication, physiotherapy and

massage for many years and not relieved with them. The treatment duration for conservative measures was 02 to 10 years and it was more in the female patients. Majority of these patients were social and nervous recks, meaning there by that the conservative methods of the treatment which they have been seeking during this time could not offer any benefit.

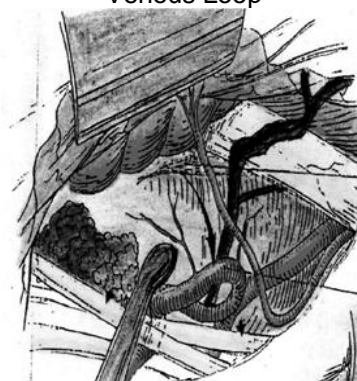
Our operative findings showed that in 11 cases arterial loops were the offending vessels and in only one case pure venous compression was the cause. Two of the patients had a combined arterial and venous compression.



Arterial Loop



Venous Loop

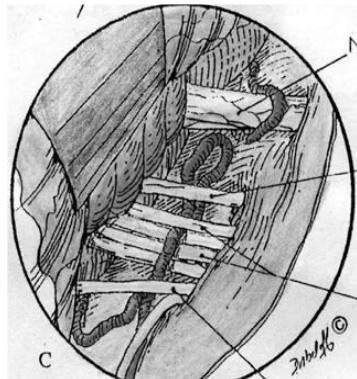


Venous Loop

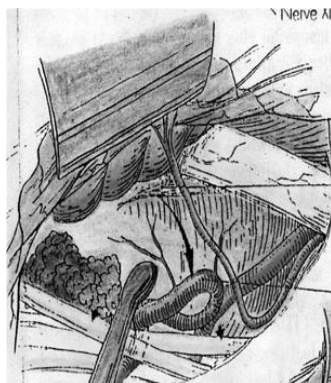
The compression site was antero-caudal in three, posterorostral in five and a combination of both was seen in six patients.



Anterocaudal



Posterorostral



Both

Procedure: In general endotracheal anaesthesia in lateral position, retro auricular retromastoid small craniectomy is done¹². The craniectomy is made more caudally and extends up to the sigmoid sinus laterally and floor of the posterior cranial fossa inferiorly. After opening the dura, cerebellum should be elevated from the floor of posterior cranial fossa. It should not

be retracted in a lateral to medial direction to avoid the damage to VIII nerve and producing hearing loss. The junction of the central and peripheral myelin in the VIII nerve is semicircular with convexity facing laterally. Any lateral to medial traction on the nerve will damage the nerve at this junction. The IX and X cranial nerves are identified and the arachnoid over them is dissected with a sharp hook. The caudal cranial nerves are followed medially up to the flocculus of cerebellum. The pons is then visualised as well as the choroid plexus at the lateral exit foramen of the IV ventricle. The VII nerve is thus approached from caudal direction in the typical HFS. The view of the VII nerve may be obstructed by the offending vessel, either single or multiple arteries, or a vein. The dissection must be carried out all around the nerve to make sure that a second compressing vessel is not missed. In the majority of the patients the offending vessel is an artery. Then a small muscle piece is placed in between the nerve and the offending vessel. The offending arteries may be the AICA, PICA, vertebral or an ectatic basilar artery. Rarely the offending artery may be situated between the VII and VIII nerve and in this situation, it is difficult to separate the artery from the nerve without producing some amount of damage to the nerves.

Complications: The procedure was not entirely free of problem¹³, one patient each developed CSF leakage, temporary facial weakness and deafness. There has been only one recurrence after 03 years.

Course of Improvement: The course of improvement in MVD of HFS is not uniform in all cases¹⁴. There can be immediate, delayed and late relief of spasm and same has been the case in our series where 60 % of cases had immediate relief. 30% patient relieved of their spasm in next six weeks and 20% patients improved later on after six weeks.

DISCUSSION

Hemifacial spasm is a rare entity involving one half of face by spontaneous spasmodic contraction of the face muscles. Most of the times, HFS is of typical variety, involving the orbicularis oculi first and then the other muscles of the face. International literature shows that there is female preponderance as is also evident from our study along with the involvement of the left side of the face. HFS is a disease of adults and the mean age in our study is 40 years. Different modalities of treatments are in use. People are using different drugs like carbamazepine, anti depressants, but with no satisfactory results. Botulin toxin injections are in common use but they produce severe dysaesthesias at the face and do not give permanent relief. The MVD procedure is only non destructive one which gives permanent cure.

CONCLUSION

Hemifacial spasm is a very disturbing condition for the patient. The Micro Vascular Decompression of the facial nerve is the best modality of treatment for the Hemifacial spasm, when done by an experienced surgeon, with minimal complication rate and permanent relief from the spasm.

REFERENCES

1. Digre KB, Corbett J: Hemifacial spasm, Differential diagnosis, mechanism, and treatment. *Adv Neurol* 49:151, 1988
2. Wilkins RH; Hemifacial Spasm : A Review. *Surg Neurol* 36:251, 1991
3. Jannetta PJ: Cranial Rhizopathies. P. 4169. In Youman Jr (ed) *Neurological Surgery* .3rd Ed. WB saunders, Philadelphia. 1990.
4. Ronen GM, Donat JR ,Hill A : Hemifacial spasm in childhood. *Can J Neurol Sci* 13:342, 1986
5. Jho HD, Jannetta PJ: Hemifacial spasm in young people treated with MVD of the facial nerve. *Neurosurgery* 20 : 767, 1987
6. Auger RG. Whisnant JP: hemifacial spasm in Rochester and Olmsted country, Minnesota. 1960 to 1984. *Arch neurolo* 47:1233, 1990
7. Hjorth RJ , Willison RG: the electromyogram in the facial myokymia and hemifacial spasm. *J Neurol sei* 20: 117, 1973
8. Sobel D , Norman d, yorke CH et al; radiography of trigeminal and hemifacial spasm. *AJNR* 1;251, 1980
9. Tash RR , Kier EL, chaytee D: Hemifacial Spasm caused by a tortoug vertebral artery: MR demonstration .*J compt assist tomogram* 12:492,1988l
10. Grandas f, Elston J, Quinn N et al: blepharospasm: A review of 264 patients.*J Neurol Neurosurgery Psychiatry* 51:767. 1988
11. Jannetta Pj, Rand Rw : transtentorial retrogasserian rhizotomy in trigeminal neuralgia by microsurgical technique. *Bull Los Angles neurol Soc* 31:91,1966.
12. Jannetta PJ: hemifacial spasm Part 15 P 1 . in Ransohoff J (ed) *Neurosurgery*. In *Modern technics in surgery*. Installment 2. Future, mount kisco, NY ,1980.
13. Loeser JD , chen J: hemifacial Spasm: treatment by microsurgical facial nerve decompression. *Neurosurgery* !£:141, 1983.
14. Jannetta PJ: microvascular decompression for hemifacial spasm.P 499. In May M (ed) : *The facial Nerve* . thieme, new York,1986.
15. Daniele O, Carragilious G, Marcini C et al : gabapentine in Hemifacial Spasm. *Acta Neurol Scand* 2001 Aug : 104 (2): 110-2
16. Arbab As, nishiyans Y, aoki S, et al , stimulation display of MRI and MRA : *Eur radiology* 2000. 10 (7): 1056-60