

MICROVASCULAR DECOMPRESSION FOR IDIOPATHIC TRIGEMINAL NEURALGIA: ULTIMATE SOLUTION TO THE MANAGEMENT DILEMMA

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ABSTRACT

Idiopathic Trigeminal Neuralgia is one of the most distressing craniofacial pain syndromes and micro vascular decompression is claimed to be the only best treatment option. The objectives of the study were to analyze the theory of neurovascular conflict as the basic pathology and prove the ineffectiveness of ablative procedures.

A prospective observation study was carried out on 110 consecutive patients of idiopathic trigeminal neuralgia who went under micro vascular decompression. Clinical presentations and operative findings were recorded, analyzed and statistically correlated. Special note was made of the previous interventions. All cases of secondary trigeminal neuralgia were excluded with the help of MRI brain.

The result showed that in 90% cases superior cerebellar artery was the cause of compression on the root entry zone. It was concluded from this study that basic cause of the idiopathic trigeminal neuralgia is a vascular compression of the trigeminal nerve near its root entry zone and micro vascular decompression is the only best treatment option available at present. Moreover, correct interpretation of the symptoms is essential to avoid inappropriate intervention.

Key words: *Idiopathic trigeminal neuralgia, micro vascular decompression, neurovascular conflict, neurectomy.*

INTRODUCTION

Idiopathic Trigeminal Neuralgia is one of the most distressing and tormenting craniofacial pain syndromes characterized by its paroxysmal, lancinating pain in the distribution of one or more branches of the trigeminal nerve resulting from the compression of the sensory component of the nerve by an aberrant vascular loop.¹ Classically, the pain responds to carbamazepine therapy, at least in the initial days of treatment.^{1,2} The superior cerebellar artery is the commonest cause of compression.^{3,4} Other less common causes include anterior inferior cerebellar artery, petrosal veins and arachnoid adhesions.^{2,4,5} It is more common in female,

old age and on right side. MRI brain must differentiate it from trigeminal neuralgia secondary to space occupying lesions in Cerebellopontine (CP) angle and multiple sclerosis (MS).^{2,6,7} Clinical examination is mostly normal. Microvascular decompression is the treatment of choice for almost all cases of idiopathic trigeminal neuralgia.^{3,8,9}

METHODOLOGY

One hundred and ten consecutive patients presenting with idiopathic trigeminal neuralgia resistant to medical treatment were enlisted for microvascular decompression from July, 2003 to July, 2008. Most of

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the patients were referred from Oral & maxillofacial surgical unit Khyber college of dentistry, Peshawar, who were given different treatment modalities like carbamazepine, peripheral neurectomies and ablative injections with poor prognosis. Detailed history and clinical examination and routine work up were carried out and documented. Every patient underwent MRI of the brain with and without contrast to exclude secondary trigeminal neuralgia caused by Space Occupying Lesion(SOL) or MS. The patients and their family were counseled and written informed consent was obtained. All the patients were resistant to medical therapy at the time of intervention. All of them were treated with Microvascular Decompression (MVD). The clinical features and operative findings were recorded on an appropriate proforma, compiled and analyzed properly.

RESULTS

One hundred and ten consecutive patients underwent MVD for trigeminal neuralgia. Males were 44 (40%) and females were 66 (60%). Age ranged from 28-80 years, mean age was 58years. The duration of pain ranged from 1-15years, the median duration of being 5.5years with \pm standard deviation of 3.34 years. The disease was found more common on right side 66%, and more so in the lower branches. Both the maxillary and the mandibular divisions were involved in 44(40%), while the ophthalmic and maxillary combination in 11(10%). Table 2.

Eighty three (75.4%) of the cases had one or more teeth extracted for the same complaint with out any improvement. Peripheral neurectomies were carried out on 39(35.4%) of the cases. Pain recurred in all of these patients after the neurectomy. Seven patients had history of gasserian ganglion injections. In all of them either the pain did not relieve or recurred after few months. Table 3.

In about 98% cases a neurovasculara conflict was found, the superior cerebellar artery (SCA) being the cause of compression in 94 cases (85.4%). Less common causes were anterior inferior cerebellar artery (AICA), posterior inferior cerebellar artery (PICA), basilar artery and petrosal veins. In 2 cases no vascular loop was found. In these patients the trigeminal nerve was found encased by tight arachnoid adhesions. In (90%) cases the site of compression was the dorsal root entry

zone (DREZ). The degree of nerve compression varied from simple contact to marked indentation of the trigeminal nerve and obvious atrophy. Table 2.

TABLE 1: DISTRIBUTION OF PAIN (n: 110)

Branch of "V" nerve	Number of cases	Percentage
V1	14	12.70
V2	7	06.30
V3	22	20.00
V1 & V2	11	10.00
V2 & V3	44	40.00
V1, V2, V3	12	10.90

TABLE 2: DEGREE OF NERVE COMPRESSION (n: 110)

Degree of compression	Number of cases	Percentage
Contact	30	27.2
Displacement	38	34.5
Indentation	34	30.9
Encasement	2	01.8
Atrophy	6	05.4

TABLE 3: PREVIOUS INTERVENTIONS (n: 110)

Procedure	No of patients	Percentage
Tooth Extraction	83	75.4
Neurectomy	39	35.4
Alcohol injections	7	06.3

DISCUSSION

Trigeminal neuralgia can be caused by a variety of conditions and can present in different clinical patterns⁴. Primary or idiopathic type is the classical type of trigeminal neuralgia while the secondary or atypical type is caused by some disease in the brainstem or CP angle such as tumours or MS. The diagnosis is purely based on history and clinical examination. The classical characteristic is a lanciating type of neurogenic pain which occurs in brief paroxysms in the distribution of one or more branches of the fifth cranial nerve. The pain typically responds well to carbamazepine therapy in the initial stages of treatment but this therapeutic

effect is partially or completely lost sooner or latter. Clinical examination is usually unremarkable. MRI of the brain with and without contrast is mandatory to rule out any other pathology such as MS or SOL in the CP angle.

Idiopathic trigeminal neuralgia is caused by a neurovascular conflict. In spite of the popularity of the concept of neurovascular conflict, the issue of the etiology of "idiopathic" trigeminal neuralgia is still a matter of great debate, and so different treatment options are advocated by different people. This includes the ablative procedures such as peripheral neurectomies, alcohol injections and gasserian ganglionectomy etc. The only way to find out the most likely underlying pathology is to study the various presentations and anatomical observations during MVD. This study was focused on the clinical presentations and operative findings in patients of idiopathic trigeminal neuralgia to get some valuable clues to the resolution of this crux.

The disease was found to be more common in the elderly people. This may be because of the age related changes that occur in the blood vessels. In this study, the commonest age group affected was between 50 and 60 years of life. These findings are pretty close to the local and international studies. In the younger patients one must be very cautious to make a meticulous search for secondary type of trigeminal neuralgia.

Like our findings, multiple other studies show a right sided predilection of the symptoms. The duration of pain was variable over a long range of time. In the ones with a short period of history either the response to medical therapy was poorer from the very beginning or the disease was unbearably severe in nature, compelling the patients for earlier intervention. Patients with a very long history usually have undergone several ablative procedures or inappropriate interventions. As majority of the patients have pain in the mandibular division in isolation or in combination with other branches, so it is apt to be misdiagnosed⁴ as a dental problem culminating in tooth extraction. In our patients 83 (75.45%) had one or multiple teeth extractions. Thirty nine patients were tried to be treated with peripheral neurectomies resulting in transient pain relief but ultimately the pain recurred.

The pain of trigeminal neuralgia is typically brought on by a physical stimulus applied to the affected area of

the face. There can be very highly sensitive point which when touched precipitates the pain. A trigger point was found in 70(63.6%) cases. The usual sites were near the angle of the mouth, in front of the tragus or over the cheek. The presence of the trigger point is of clinical importance because of its relationship to significant compression on the nerve and good post operative outcome⁴.

The interval between the consecutive attacks varied widely. Some patients had months or years completely free of pain. In others, there were several attacks every day. They complain of the typical lancinating attacks superadded to the dull aching background. In our study, pain-free interval ranged from few hours to 2 years.

In majority of our cases (98%), the cause of compression of the trigeminal nerve, was an arterial loop. The SCA was responsible for 94(85.4%). AICA, PICA and basilar artery were seen compressing the nerve in 5, 3 and 2 cases respectively. Three cases were involving some unnamed vessels. Only one case was having compression due to the superior petrosal veins. In the remaining 2 cases no vascular loop was found. Arachnoid adhesions were probably the cause of compression in these 2 cases. Several local and international data are very close to our findings^{4,5}.

We noted that in the maximum majority, 99 (90%), of the cases, the site of compression was the dorsal root entry zone (DREZ). It is here that the persistent pressure and pulsating effect of the loop causes demyelination of the sensory axons and result in pain. Two patients, who had no vascular cause, the whole length of the nerve was encased in thick arachnoid bands. These findings are close to those of the international studies⁴.

CONCLUSION

This study strongly supports the neurovascular conflict theory as the etiology of primary trigeminal neuralgia. The commonest cause of compression is the superior cerebellar artery. Detailed history and thorough clinical examination are most essential to avoid wrong diagnosis and inappropriate interventions. A vascular compression being the established cause of idiopathic trigeminal neuralgia, MVD is the most logical and curative procedure of treatment!

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