IDIOPATHIC TRIGEMINAL NEURALGIA: CLINICAL PRESENTATIONS AND OPERATIVE FINDINGS ON MICROVASCULAR DECOMPRESSION

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ABSTRACT

Objective: *To analyze the clinical presentations of idiopathic trigeminal neuralgia and its operative findings on microvascular decompression.*

Material and Methods: *This descriptive case series study was conducted in the Department of Neurosurgery, PGMI, Govt. Lady Reading Hospital, Peshawar from July, 2003 to November, 2007. A total of 110 consecutive patients of idiopathic trigeminal neuralgia undergoing microvascular decompression in the Department of Neurosurgery, Lady Reading Hospital were included in the study. Clinical presentations and operative findings were recorded and analyzed. All cases of secondary trigeminal neuralgia were excluded with the help of MRI brain.*

Results: *Ratio of Male: female was 2:3. Age ranged from 28-80 years, mean age being 57 years. Right side was affected in 60% cases. Maxillary and mandibular divisions in combination were involved in majority of cases. Pain-free period varied from few hours to years. The common triggering stimuli were chewing and touching. A trigger point was found in (77%) cases. Superior cerebellar artery was the cause of compression in 90% of cases. In 90% of cases compression was on the root entry zone. The nerve compression was found on superomedial aspect in 66(60%) of cases.*

Conclusion: *Almost all cases of idiopathic trigeminal neuralgia are caused by a vascular compression of the trigeminal nerve, superior cerebellar artery being the commonest cause 90%.*

Key Words: *Trigeminal nerve, idiopathic trigeminal neuralgia, microvascular decompression, neurovascular conflict, trigger point.*

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 trigeminal nerve by an aberrant vascular loop. Classically, the pain responds to crabamazipine therapy, at least in the initial days of treatment. The superior cerebellar artery is the commonest cause of compression. Other less common causes include anterior inferior cerebellar artery, petrosal veins and arachnoid adhesions. It is more common in female, old age and on right side. MRI brain must differentiate it form trigeminal neuralgia secondary to space occupying lesions in CP angle and multiple sclerosis. Clinical examination is mostly normal. Microvascular decompression is the treatment of choice for almost all cases of idiopathic trigeminal neuralgia.

MATERIAL AND METHODS

One hundred and ten consecutive patients presenting with idiopathic trigeminal neuralgia resistant to medical treatment were enlisted for microvascular decompression from July, 2003 to November, 2007. All patients got admitted through the OPD. 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 lesions or multiple sclerosis. The

TOPOGRAPHY 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 1

DEGREE OF NERVE COMPRESSION (n: 110)

Degree of compression	Number of Cases	Percentage
Contact	30	27.2
Displacement	38	34.5
Indentation	35	31.8
Encasement	2	01.8
Atrophy	6	05.4

Table 2

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 underwent Microvascular Decompression (MVD) for Trigeminal Neuralgia. The clinical features and operative findings were recorded on an appropriate proforma, compiled and analyzed properly.

RESULTS

Total 110 consecutive patients underwent MVD for trigeminal neuralgia. Males were 44 (40%) and females were 66 (60%). The Male: Female 2:3. Age ranged from 28-80 years, 60% of cases in the 6th decade. Mean age 58years.

Right side was involved in 66 (60%) and left side in 43 (39.%) cases while only one patient 0.9% had bilateral pain.

The duration of pain ranged from 1-15years, majority 50 (45.45%) being in the 4-6 years group. Maximum patients were having pain for more than 4 years. The mean duration of symptoms was 5.5years with ± standard deviation of 3.34 years.

The lower divisions were affected much more than the upper ones. Both the maxillary and the mandibular divisions were involved 44(40%), while the ophthalmic and maxillary combination in 11(10%) cases. Table I. Touch, chewing, washing of face and mouth, shaving and brushing were the common pain-precipitating stimuli. A trigger point was found in 63.6% (70).

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). Arterial compression was on superomedial aspect of the trigeminal nerve in 61 (55.3%) of cases. The degree of nerve compression varied from simple contact to marked indentation of the trigeminal nerve and obvious atrophy. Table II.

Superolteral aspect compression gave pain more commonly in the ophthalmic and maxillary divisions, while the lower division pain was more commonly associated with compression from superomedial aspect of the trigeminal nerve. Table III. Similarly, a positive trigger point was more indicative of indentation of the trigeminal nerve by the compressing loop, most commonly the superior cerebellar artery. Table IV.

CORRELATION BETWEEN DISTRIBUTION OF PAIN AND DIRECTION OF CONFLICT (N=110)

Branch of TN	SM	SL	INF	Ar. Ad'n.	Total
V1	9	4	1	0	14
V2	4	2	1	0	07
V3	14	7	2	0	22
V1 &V2	5	6	0	0	11
V2 & V3	23	12	8	1	44
V1,V2,V3	6	2	3	1	12

V1: ophthalmic division; V2: maxillary division; V3: mandibular division.
SM: superomedial; SL: superolateral; INF: inferior; Ar. Ad'n: arachnoid adhesions.

Table 3

CORRELATION BETWEEN THE PRESENCE OF TRIGGER POINT AND THE DEGREE OF COMPRESSION (n=110)

Degree of compression	Trigger point present	Trigger point absent	Total
Contact	10	20	29
Displacement	21	17	38
Indentation	31	4	35
Encasement	2	0	2
Atrophy	6	0	6

Table 4

DISCUSSION

Trigeminal neuralgia can be caused by a variety of conditions and can present in different clinical patterns. 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 the cerebellopontine (CP) angle such as tumours or multiple sclerosis (MS). The diagnosis is purely based on history and clinical examination. The classical characteristic is a lancinating type of neurogenic pain which occurs in brief paroxysms in the distribution of one or more branches of the fifth cranial nerve. Each attack lasts for a few seconds to a couple of minutes. However, some times patients may present in persistent irretractable pain, a condition known as status trigeminus. The pain typically responds well to carbamazipine therapy in the initial stages of treatment but this therapeutic effect is partially or completely lost sooner or latte. Clinical examination is usually unremarkable except for an impaired corneal reflex in rare cases. MRI of the brain with and without contrast is mandatory to rule out any other pathologies especially SOL in the CP angle or MS.

The idiopathic type of the disease is considered to be caused by a neurovascular conflict. The concept of neurovascular conflict was postulated by Dandy in 1930's. Since then, MVD has been the procedure of choice for the management of idiopathic trigeminal neuralgia. Inspite of the popularity of the concept of neurovascular conflict, the issue of the aetiology of "idiopathic" trigeminal neuralgia is still a matter of great debate. The only way to find out the most likely cause of the disease and to establish the relationship between the clinical presentation and the underlying pathology is to study the various presentations and anatomical observations during MVD. This study was focused on the clinical presentations and operative findings on MVD in patients of idiopathic trigeminal neuralgia to get some valuable clues to the resolution of this crux.

A total of 110 consecutive patients were included in the study. They all had lost their initial response to medical therapy at the time of surgery. All the patients underwent MVD. The disease affected females more than males with a ratio of Male: Female 2:3 according to our findings. In other local studies there is slight male preponderance[ ]. In the international studies, however, females are more commonly found affected.

The disease is 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 pain in the bilateral disease was more severe in the left side which was operated while the other side was treated with medications.

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 resulting in transient pain relief. 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.

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 outcome.

In majority of our cases 98%, the cause of compression of the trigeminal nerve, was an arterial loop. The Superior Cerebellar Artery 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. Arachniod adhesions were probably the cause of compression in these 2 cases. Several local and international data are very close to our findings.

We noted that in the maximum majority (90%), 99of 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 studies.

The relationship between the distribution of pain and the direction of the compression was also carefully analyzed. Compression from superolteral aspect gave pain more commonly in the upper 2 branches, while the lower face pain was commonly caused by compression along the superomedial aspect of the trigeminal nerve. An inferior compression gave pain exclusively in the distribution of the lower branches. All these findings were well explaining the arrangement of the fibers from all the three branches of the trigeminal nerve in the sensory root.

The relationship of the presence of trigger point and the cause of compression was observed. Where a trigger point was present, in majority of the cases, the nerve was found indented by the SCA. Similarly, the topography of the pain was correlated to the direction of the compression on the nerve.

CONCLUSION

Our study strongly suggests the neurovascular conflict theory as the aetiology of primary trigeminal neuralgia. The commonest cause of compression is the superior cerebellar artery. The clinical presentation of the disease can be correlated with the operative findings and the most likely anatomical changes can be predicted. A vascular compression being the established cause of idiopathic trigeminal neuralgia, Microvascular Decompression is the most logical and curative procedure of treatment.

REFERENCES

1. Greenberg MS. Pain: Hand Book of Neurosurgery. 6th ed. NewYork: Thieme, 2005: 378-9.
2. Tanrikulu L, Hastreiter P, Troescher R, Buchfelder M, Naraghi R. Intraoperative three-dimensional visualization in microvascular decompression. J Neurosurg 2007; 107:1137-43.
3. Jannetta PJ, Levy EI. Trigeminal neuralgia: microvascular decompression of the trigeminal nerve for tic douloureux. In: Winn HR, ed. Youman's Neurological Surgery. Philadelphia: WB Saunders, 2004; 3005–15.
4. Mouregela S, Sakellaropoulos A, Anagnostopoulou S. Middle cranial fossa ndoscopy using a rigid endoscope. Minim Invasive Ther Allied Technol 2007;16:355-9.
5. Elias HK, Jeffrey W, Burchiel KJ, Kim J. Microvascular Decompression. Clin J Pain 2002;18:35-41.
6.. Vohra AH, Abbasi KH, Kasuri MN. Microvascular Decompression and Glycerol Rhizotomy for Trigeminal Neuralgia. Pak J Neurol 2000; 6: 13-5.
7. Lehari IA, Baloch MR. Trigeminal Neuralgia - distribution of pain and nerve involvement. Med Channel 2004; 10:21-3.
8. Ali M, Khan KM, Khanzada KA, Ayub S, Khan H. Significance of trigger point in trigeminal neuralgia. J Postgrad Med Inst 2007; 21: 183-6.
9. Ayub S, Ilyas M, Ali M. Surgical management of Trigeminal Neuralgia by microvascular decompression. J Postgrad Med Inst 2004; 18:507-11.
10. Sindou M.Anatomical findings in idiopathic trigeminal neuralgias. In: Kanno T, ed. Proceedings of 5th Meeting of the Society for Microvascular Decompression Surgery, Matsumoto, Japan:2002:23-35.
11. El-Garem HF, Badr-El-Dine M, Talaat AM, Magnan J. Endoscopy as a tool in minimally invasive trigeminal neuralgia surgery. Otol Neurotol 2002;23:132-5.
12. Yoshino N, Akimoto H, Yamada I, Nagaoka T, Tetsumura A, Kurabayashi T, et al. Trigeminal Neuralgia: Evaluation of Neuralgic Manifestation and Site of Neurovascular Compression with 3D CISS MR Imaging and MR Angiography. Radiology 2003; 228:539-42.

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