Short Communication

NEURO-VASCULAR CONFLICT AS CAUSATIVE FACTOR IN IDIOPATHIC TRIGEMINAL NEURALGIA

Mumtaz Ali[(1)], Hamzullah Khan[(2)]

***ARYA Atherosclerosis Journal* 2007, 3(3): 175-177**
Date of submission: 25 Jul 2007, ***Date of acceptance:*** 01 Oct 2007

Introduction

Trigeminal neuralgia is one of the most unbearable pain syndromes in one or more branches of trigeminal nerve. The basic pathology is still poorly understood [1]. Two divergent view points, central versus peripheral have been presented to explain the possible mechanism [2]. In spite of numerous favorable reports, the neurovascular conflict theory remains contravertical [3]. Nevertheless, whether or not, neurovascular compression is accessory or predominant in the mechanism of trigeminal neuralgia is not yet determined. Although neurovascular compression and global atrophy of the root, a focal arachnoid thickening and angulated root on crossing over the petrous ridge have been observed. Yet, neurovascular conflict has made responsible as the main cause of this neuralgia [4]. This lead to focal demylination of the nerve due to its pulsatile compression. Demylination result in short circuiting of neuronal flow and hence trigeminal neuralgia [5].

Present study was therefore designed as to appreciate neurovascular conflict as causative agent in idiopathic trigeminal neuralgia.

Material and Methods

This prospective observational study was conducted in department of Neurosurgery Government Lady Reading Hospital Peshawar where microvascular decompression is performed as a primary procedure of choice for patients with trigeminal neuralgia. The duration of this study was from May 2003 – to June 2007. Total number of patients operated was 86. Drug resistant cases of trigeminal neuralgia that were willing for operation was selected and proper clinical record was documented. MRI was done in all patients to exclude secondary causes of trigeminal Neuralgia. Under general Anesthesia in lateral position, small 2.5x2.5cm retro-mastoid craniotomy was performed. All these cases were operated by one surgeon with a team of associate's doctors. Microscopic per-operative anatomical findings were recorded. Any possible per-operative complications were also documented.

Operative Technique

All these patients were operated in park bench position. The effected side was made up neck veins were kept without compression. Pinna of operative Ear was stitched anteriorly to expose the retro-mastoid site properly, about 5 –7cases incision was made after proper cleaning and dropping the operative site. Incision was 1/3 above and 2/3 below the mastoid tip.

About 2.5x2.5cm craniotomy was made transverse sigmoid venous was dissected and emissary vein coming from this site was coagulated. Dura was opened through a C- shaped incision and converted into inverted "T" by extending toward sigmoid transverse junction.

Supra-cerebellar infratentorial approach was made and cerebrospinal fluid (CSF) was drained. Petrotentorial corridor was seen. Petrosal vein was dissected and coagulated carefully to avoid rupture it was cut and arachnoid was opened. Search was made for offending arterial and loop and abnormal veins. Exploration up to DREZC dorsal post entering zone

1) Assistant Professor, Department of Neurosurgery, Postgraduate Medical Institute, Lady Reading Hospital, Peshawar, Pakistan.
2) MBBS, Department of Neurosurgery, Postgraduate Medical Institute, Lady Reading Hospital, Peshawar, Pakistan.
e-mail: hamza_kmc@yahoo.com

Corresponding author: Hamzullah Khan

was done. Any contact of vessel either casual or grooming was noted. Muscle piece taken from neck muscles drainage exploration was put around nerve. Head end was left down to heart level and valsalva manure was performed to see any venues ooze. Dura was closed water lightly and wound was closed layer by layer. Recovery from anesthesia was smoothly. All these patients were kept in neuro intensive care unit (neuro-ICU) for 24 –48 hours.

Finally information's collected were analyzed and association of neurovascular mechanisms in development of trigeminal neuralgia was studied.

Results

A total of 86 patients with idiopathic trigeminal neuralgia were operated. There were 54 male and 32 female patients. Their age ranged from 43 to 78 years with mean age of 59 years. Bilateral trigeminal neuralgia was seen in two female patients. Right sided trigeminal neuralgia was noted in 46 and left sided in 38 cases. 63 patients had maxillary division (V2) involvement and 7 patients had both maxillary and mandibular divisions (V2 and V3). Four patients were having only mandibular involvement (V3) while in 2 patients all three branches i.e. ophthalmic, maxillary and mandibular divisions (V1, V2 and V3) were involved.

Superior cerebellar artery was involved in 61 cases, anterior and inferior cerebellar artery in 20 cases, basilar artery in one and superior, anterior and inferior cerebellar arteries in 4 cases.

Alteration of the whole trigeminal nerve was observed in 62 patients, vascular groove on the superio lateral aspect by vessel in 16 and from inferior aspect in 5 cases.

No mortality was noted. Injury to transverse venous sinus during dural opening in 4 cases peroperative rephir of petrosal vein in 5 cases and cerebellar contusion in one patient. Post operatively, nausea, vomiting and dizziness in 16 cases, diplopia in 3 cases, slight deafness in two capsular infarct in one and CSF leakage in three, prolong discharge from wound due to Osteomyelitis of bone in one patient.

Discussion

As the poshilahed theory of arterial compression over the trigeminal nerve as the cause of trigeminal neurologia is well popularized. Therefore, the operative treatment is based on the neurovascualr conflict. [6] Only microvascular decompression directly modifies the well known etiology of vascular compression while as all other treatment rely on damage of the trigeminal nerve to relieve pain.

It has been found that idiopathic trigeminal neuralgia is actually caused by an offending vessel in 96.9% of cases where as without a compression factor has been reported in 3.1 to 17% of cases [7]. Segment of superior cerebellar artery on the superior and supero-medial aspect of nerve in the form of loop is the most common findings in 80 –88% of cases. Anterio-inferior cerebellar artery in 12 –20% of cases with offending arterial loop while venous compression has been reported in 9% to 25% of cases [8].

We observed superior cerebellar artery in 55 cases 63% and anterior inferior cerebellar artery in 16 cases, while both combined compression in 5% cases. Venous compression was seen in 3 cases in my series while reported incidence is 9 – 25%.

It is difficult to define the precise etiology of trigeminal neuralgia in patients where no abnormality is found. Arachnoid thickening or adhesion can modify the normal direction of the trigeminal nerve. Sever arachnoidal thickening may involve directly the root entry zone (obersteiner Redelinck line), causing the pain, we found this pathology in 3 cases of our patient. R-Revuelta – Gutierrez etal has reported 5 cases of arachnoid adhesion in his 668 patients series. Marc sindon has reported 12.6% patients with local thickening of arachnoid membrane in 560cases series [9 & 10].

Distortion of nerve and a marked indentation by an offending loops based on degree of severity of this main conflict, we observed alteration of the whole trigeminal nerve in 62% patients, vascular groove (marked indentation by arterial loop) in 21% cases. Our finding matches with that of Guevara N et al[11]. Idiopathic trigeminal neuralgia is commonly the result of offending loop in the vicinity of trigeminal nerve. Superior cerebellar and anterior inferior cerebellar arteries are common causative agent responsible for this conflict. The abscence or presence of this causative agent can be defined with difficulty, preoperatively. The preferred option of treatment is standard micro vascular decompression.

References

1. Revuelta-Gutierrez, MD, Miguel Angel Lopez-Gonzalez, MD, Jose Luis Soto-Hernandez MD (eds).Surgical tratment of Trigeminal Neuralgia without vascular compression: 20 years of experience. Mexico City 14269, Mexico, 2005.
2. Elias WF, Burchiel KJ. Trigeminal Neuralgia and other craniofacial pain syndromes: an overview. Semin Neurosurg 2004;15:59-69.
3. Ishikawa M, Nishi S, Aoki T, et al. Operative findings in cases of trigeminal neuralgia without vascular compression: proposal of a different mechanism. J Clin Neurosci 2002;9:200-4.
4. Jannetta PJ. Outcome after microvascular decompression for typical trigeminal neuralgia, hemifacial spasm, tinnitus, disabling positional vertigo, and glosspharyngeal neuralgia. Clin neurosurg: 1996;26:159-62

5. Lee SH, Levy EI, scarrow AM, et al. Recurrent trigeminal neuralgia attributable to veins after microvascular decompression. Neurosurgery 2000;46:356-62.
6. Lovely TJ, Jannetta PJ. Microvascular decompression for trigeminal neuralgia. Surgical technique and long term results. Neurosurg Clin N Am 1997;8:11-29.
7. Rath SA, Klein HJ, Richter HP. Findings and long-term results of subsequent operations after failed microvascular decompression for trigeminal neuralgia, Neurosurgery 1996;39:933-40.
8. Revuelta R, Soto-Hernandez JL, Vales LO, et al. cerebellopontine angle cysticercus and concurrent vascular compression in a case of trigeminal neuralgia. Clin Neurol Neurosurg 2003;106: 19-22.
9. Urgosik D, Liscak R, Novotny Jr.J, et al. Treatment of essential trigeminal neuralgia with gamma knife surgery. J Neurosurg (Suppl) 2005;102:20-33.
10. Zakrzewsha JM, Lopez BC, Kim SE, et al. patient reports of satisfaction after microvasculr decompression and partial sensory rhizotomy for trigeminal neuralgia. Neurosurgry 2005;56:1304-12.
11. Guevara N, Deveze A, Buza V, Laffont B, Magnan J. Microvascular decompression of cochlear nerve for tinnitus incapacity: pre-surgical data, surgical analyses and long-term follow-up of 15 patients.Eur Arch Otorhinolaryngol. 2007 Oct 2; [Epub ahead of print].