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Original Research Article

MVD for trigeminal neuralgia; neuralgia revisited with review of literature

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ABSTRACT

Background: Trigeminal neuralgia (TN) s an uncommon facial pain syndrome. It is commonly characterized by sudden onset of an attack of intense electric shock like facial pain localized along the sensory distribution of the ninth cranial nerve. Although many patients respond to the medical line of treatment but when PTS symptoms become intractable and could not tolerate the medications have to go for surgical decompression. Our study aims to review about micro vascular decompression for trigeminal neuralgia and its outcome along with the review of literature.
Methods: It is a retrospective study conducted on 40 cases who were diagnosed with TN. between the period of July 2009 to July 2015. MVD has been performed on all the patients. Demographic data, clinical presentation, surgical findings, complications and long term follow-up were reviewed.
Results: The median age of onset of TN is 50.4 years divided into five age groups ranging from 24 to 89 years with female preponderance showing 65.5% as compared to males. The distribution of location was in the ratio of right to left is 1.5:1. There was no mortality is seen in the study. 3 patients among them study population had complications 3 patients had facial hyposthesia, 1 patient had V3 paresis and 1 patient had CSF otorrhoea which got settled with drainage LP. All the patients had satisfactory improvement except one patient who had persistent pain.
Conclusions: To conclude micro vascular decompression (MVD) is the surgical procedure of choice for the treatment of medically refractory TN who is fit for surgery. Of all the currently available surgical methods MVD provides the highest rate of long term satisfaction for the patients and offers the lowest rate of pain recurrence.

Keywords: Microvascular decompression, Trigeminal neuralgia, Facial pain, Facial hyposthesia

INTRODUCTION

Trigeminal neuralgia (TN) is a disorder characterized by sudden sharp, shooting, lancinating pain attacks lasting several seconds to several minutes and localized to one or two branches of the trigeminal nerve. These attacks may begin spontaneously or they can be initiated by stimulation of the so-called trigger zones. The classic episodes of shooting pain are interrupted by pain-free intervals with remissions occasionally lasting for years.[1] It is an uncommon craniofacial pain syndrome, representing 0.2–1.3% of facial pain syndromes, with an annual incidence of 0.7 cases per 100,000 habitants per year according to a population-based study.[2-4] In some cases, it is associated with excessive vagal outflow; which leads to bradycardia, hypotension, syncope or cardiac arrest.[5-7]

Dandy elucidated the pathophysiology of trigeminal neuralgia and proposed vascular compression at the root of the trigeminal nerve as the main etiology, causing demyelinization, and ephaptic transmission.[8,9] It is shown that in about 96% of cases of typical TN, vascular compression is to be found in only 3% of people without TN, there is a vascular contact with the trigeminal nerve.[10-12]

The pathological substrate is located in the building of the cranial nerves. In the brain stem the oligodendroglia are responsible for axonal isolation, and outside brain stem is Schwann cells. The transition zone "gaps" is called Redlich-Obersteiner's zone. Thus at this point, there is a transition from oligodendrocytes myelin to Schwann cell myelin. In this area a number of axons have a marginal or no isolation at all. Lack of isolation could leads to a strengthening effect on input (or output). This transmission, promoted by the vascular compression, leads to increased central stimulation, or to increased activity in the central neuron. Sensory neurons of the faces are often hyperexcited when damage occurs in the nerve and this leads to spontaneous generation of electrical impulses at the damaged spot.[13-16] The chronic vascular compression of the trigeminal nerve has focal de-myelinization effect. This leads to dysfunction of local inhibitory interneurons and development of ectopic neuronal pacemakers. The combination of increased input by afferents ectopic pacemakers and the dysfunction of the intersegmental inhibitory neurons lead to hyperactivity of the nucleus of the trigeminal nerve. The result is TN attacks after stimulation of trigger points supplied by trigeminal nerve during washing, tooth brushing, eating, or even by touching the face.

The diagnosis of TN is primarily made on the basis of a careful history of presenting symptoms. The typical "lancinating" pain in one or more of the branches of the trigeminal nerve, a typical course in time, and one or more typical pain triggers are essential for diagnosing TN. In typical TN, on examination no neurological deficit is found in trigeminal nerve distribution. This distinguishes typical from atypical TN; the latter is described as a burning and continuous pain (neuropathy).[17]

There are various treatment options for TN present but all have their limitations, percutaneous techniques include glycerol injections, radiofrequency ablation and balloon compression in or around the Gasserian ganglion.[18] There are also noninvasive methods such as Gamma Knife and drug treatments. Carbamazepine is the gold standard of drug therapy for TN. Finally Jannetta, popularized microvascular decompression (MVD) as a definitive surgical treatment for this pathology.[19-21] MVD series have reported good outcomes in 90–98%, long-term pain improvement have been observed in 64% with a low mortality ranging from 0% to 5.8%.[22]

Currently, the MVD procedure is perceived as the most effective treatment for TN with the most satisfactory outcomes. The purpose of this study was to evaluate the anatomical variations in pathophysiology and role of MVD as a better treatment option in medically refractory cases in this series of cases of Trigeminal neuralgia.

METHODS

The cases were selected from Department of Neurosurgery of SDM College of Medical Sciences & Hospital and Basveshwar Teaching and General Hospital from July 2009 to July 2015 after taking written approval from Ethical medical board of each hospital. Patients of age 20 years to 70 years, irrespective of gender and Trigeminal neuralgia involving Ophthalmic, mandible and maxillary branches of trigeminal nerve and who were operated with retro mastoid craniotomy and micro vascular decompression were included in the study, patients having TN due to any secondary causes or in association to other neuralgia were excluded. An informed written consent was obtained from patients for participation in this study. The demographic information like age, gender and address were obtained. Routine investigations and radiographs like MRI and P-A face were done preoperatively. Radiographic imaging was carried out to exclude local pathology. Individual symptoms, clinical history, operative findings, and complications were recorded. Descriptive statistics were calculated for variables included demographics (age, gender), site of pain (right or left) and distribution of nerve involvement. Mean and standard deviation was calculated for age. Operative results were assessed by clinical follow up and periodic phone surveys done ranging from 6 months to 60 months with mean of 33 months.

Surgical technique

Exploration of the cerebellopontine angle was performed through a small, keyhole type (about 35 mm diameter) retromastoid craniectomy, with patients in the supine position with the head rotated to the side opposite the neuralgia. The margins of transverse sinus and sigmoid sinuses were exposed and the Dura was opened along the line bisecting their angle. The fifth cranial nerve was exposed through a supracerebellar approach, thus avoiding lateral retraction of the cerebellar hemisphere and traction of the VII-VIII complex of cranial nerves. Neither lumbar CSF drainage nor mannitol was used to avoid anatomical modification before Dural opening. In approaching the trigeminal nerve, care was taken to spare at least two petrous veins. The nerve was cautiously dissected free without unnecessary manipulation. Any compressive arteries were relocated away from the trigeminal nerve and its root entry zone in the pons by the use of small pieces of Teflon. Compressive veins were electro coagulated and divided.

RESULTS

A total number of 40 patients of TN reported at Dept. Of Neuro Surgery SDM College of Medical Sciences and Hospital and Basveshwar Teaching and General Hospital during the study period and were operated for the same with retro mastoid craniectomy and MVD.

The patients were divided into five groups. 2.5% (n=1). Patients were recorded in 1st group (<30 years). 12.5% (n=5) patients were recorded in 2nd group (31-40 years). 32.5% (n=13) patients were recorded in 3rd group (41-50 years). 35% (n=14) patients were recorded in 4th group (51-60 years). 17.5% (n=7) patients were recorded in 5th group (41-50 years). The mean age of patient's was 50.4 years. The most common age group for TN was 40-60 years in our study as shown in.

Age at onset

This ranged between 24 to 89 years, with a mean age of 50.4 years. The peak incidence was in the fifth and sixth decades of life.

Table 1: Age distribution of patients with TN.

Age in years	No of patients (n)	Percentage (%)
<30	1	2.5
31-40	5	12.5
41-50	13	32.5
51-60	14	35
>60	7	17.5

Mean age±SD=50.3±10.3

Gender

Females comprised 62.5 per cent (n=35) of the patients and 37.5% (n=15) were male, representing a ratio of 1.7:1, reflecting an elevated risk for female subjects TN was common in female patients.

Site of involvement

Of the remaining 40 cases, 24 patients (65 per cent) suffered excruciating pain on the right side of the face, and 16 patients on the left side. This gave a site ratio of 1.5:1 confirming a predominance of right side facial affliction. TN affected right side in 65% (n= 24) patients and left side in 35% (n=16) patients. The right to left side ratio is 1.5:1 (Table 2).

Table 2: Side involvement of TN.

Side involvement of TN		
Right	24	65%
Left	16	35%
Total	40	100

Right to left ratio 1.5:1.

Post operatively no anticonvulsants were given to the patients and suture removal was done on post op day 10.There was no mortality seen in the study. All the patients had satisfactory improvement except one patient who had persistent pain. Three patients among the study population had complications three patients had facial hypoesthesia, one patient had V3 paresis and one patient had CSF otorrhoea which got settled with drainage LP.

DISCUSSION

Direct comparisons between various series are hindered by different definitions of operative success regarding the recurrence rate of tic pain after MVD that was done using actuarial methods. It is important to ascertain which artery, vertebral or basilar, is compressing the nerve, as the risk of operating in these patients is higher than in patients where the superior cerebellar artery is the trigger.

The theory of vascular compression as the cause of TN is supported by clinical and anatomical evidence.[12,16] In idiopathic TN nerve compression is by a vessel as it exits the medulla oblongata, this theory is supported by the success of MVD in the treatment of compression of trigeminal nerve.[21]

TN usually begins as a relapsing disease with pain-free intervals, which sometimes can last for months or years. These pain-free intervals become shorter until they eventually disappear. With the disease progression, patients may have trouble in talking, eating, face washing, and teeth brushing because of pain caused by these activities. Current treatment usually begins with medications for example, carbamazepine, which fortunately gives an improvement of symptoms. But unfortunately, long-term effect is less effective. It is difficult to continue these drugs because of the many side effects they have, such as hyponatremia.[23] Hence in about half of the patients of TN, surgical treatment becomes necessary.[24]

Average age in our study is 50.4 years with peak incidence in the 5th and 6th decade of life, whereas it is almost same as compared to other similar studies

.Female to male ratio is 1.5:1 in our study. This is similar to other studies with a female to male ratio ranges from 2:1 to 4:3 several similar studies showed.[25,26] Right side of face is afflicted more in TN, In our series also has been found the same results.

Traditionally, a lateral suboccipital approach provides adequate exposure to the trigeminal, facial, and lower cranial nerves. Kawashima et al proposed a transcondylar fossa approach advocating the wide operative view of the cerebellomedullary cistern, smaller retraction of the cerebellum, less risk of cranial nerve injury, and enough space to perform the sling retraction technique, however in our series classical minimally invasive retrosigmoid suboccipital approach has been used wherein PICA, vertebral A and Trigeminal neurovascular complex is adequately exposed by careful dissection of arachanoid and minimal retraction of the cerebellum giving enough space for working without the necessary of removing the jugular tubrcle.[27]

Jannetta, popularized the MVD using a suboccipital craniotomy.[19] After years of experience, the approach was modified according to the surgical goal. In our series slightly modified tailor made retrosigmoid suboccipital craniectomy at the edge of transvers and sigmoid sinuses gives enough exposure and access to the trigeminal, facial and glossopharyngeal neurovascular complex.

In the MVD series, the overall surgical mortality is 1.1%. The rate of long-term pain remission is 84.7% with recurrence in 7%. Transient X cranial nerve dysfunction occurred in 13.2% and permanent deficits in 5.5%.[28] In our study we did not have any mortality except 3 cases developed complication 1 patient had facial hyposthesia, 1 had v3 paresis and one more patient had CSF otorrhoea which got settled with LP drainage.

Rey-Dios and Cohen-Gadol demonstrated in his analyses that the most effective surgical procedure to treat GPN is the MVD.[28] Several studies used rhizotomy as the preferred procedure, but a 3-fold increase in the risk of permanent postoperative vagus dysfunction is objectionable in comparison to MVD.[20,22,27,28] It is also well demonstrated that the rate of pain control is slightly better with rhizotomy (95%) than with MVD (86%). In our series all the patients had satisfactory improvement except one patient who had persistent pain

Other noninvasive treatment options have been described: Percutaneous radiofrequency neurolysis is an alternative in cases who failed medical treatment or in which they cannot undergo intracranial surgery.[29] Gamma Knife radiosurgery is also a potential option to relieve the pain without reported side effects but a high early recurrence risk.[30]

Studies have shown differences between typical and atypical TN, in about 88% of patients with typical TN and 56% of patients with atypical TN and NVC could be demonstrated, compression by a vein is significantly higher in patients with atypical TN than in patients with typical TN

There are a number of factors which are strongly correlated with a good outcome of TN after MVD. These factors include age, gender, preoperative pain longer than 8 years, and the type of compression. Venous compression correlates with a worse outcome.[31]

CONCLUSION

We conclude micro vascular decompression (MVD) is the surgical procedure of choice for the treatment of medically refractory TN who are fit for surgery. MVD has also been shown to provide pain relief even in patients without visible neurovascular compression. MVD addresses the pathology at the root site of lesion and is best available modality being a nerve sparing technique as compared to Rhizolysis and radio frequency ablation. Of all the currently available surgical methods MVD provides the highest rate of long term satisfaction for the patients and offers the lowest rate of pain recurrence.

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