Review Article

Microvascular decompression as a surgical management for trigeminal neuralgia: A critical review of the literature

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Abstract

Trigeminal neuralgia (TN) is a common pain syndrome and is characterized by recurrent episodes of intense lancinating pain in one or more divisions of the trigeminal nerve. Neurovascular compression (NVC) has been considered as the main cause of TN in the root entry zone (REZ) of the trigeminal nerve in the cerebellopontine angle cistern. Microvascular decompression (MVD) is the surgical procedure of choice for the treatment of medically refractory TN. MVD has also been shown to provide pain relief even in patients without visible neurovascular compression. Additionally, it has been accepted that MVD can provide the highest rate of long-term patient satisfaction with the lowest rate of pain recurrence. We did, systematic review of the subject and also our own experiences.

Key words: *Microvascular decompression, neurovascular compression, trigeminal neuralgia*

Introduction

Trigeminal neuralgia (TN) is characterized by paroxysmal shock-like pain localized to the innervated area of one or more branches of trigeminal nerve and is due to vascular compression of the trigeminal nerve at the root entry zone (REZ).[1-6] The age of onset is usually between 40–60 years and seldom in patients younger than 40 years of age. The exception being symptomatic TN due to multiple sclerosis or tumors. Nevertheless, TN does not present a full-blown clinical picture until before the brief episodes of pain become severe or frequent and unresponsive to medication.[7,8] The initial treatment of choice is medical, antiepileptic drugs (AEDs). About a half of patients with TN eventually require surgical intervention for the definitive relief of pain.[9] The surgical treatment modalities include percutaneous ablative procedures and microvascular decompression (MVD).[10] Percutaneous ablative procedures seem to be safe and simple, with fewer serious complications and a high initial success rate. However, the duration of pain-relief is usually for a shorter period, mostly for less than a year and with repeated interventions the period of pain relief may be even shorter.[3,8] In 1934, Dandy hypothesized for the first time that neurovascular compression may be the cause of TN. Gardner supported the hypothesis around 1962, later Jannetta suggested MVD as a surgical procedure for the treatment of TN. Currently, the MVD procedure is perceived as the most effective treatment for TN with the most satisfactory outcomes.

Etiology

The International Association for the Study of Pain (IASP) defines TN as "a sudden, usually unilateral, severe, brief, stabbing, recurrent episodes of pain in the distribution of one or more branches of the trigeminal nerve." The incidence is 3–5 per 100,000.[11-15] TN occurs in middle and old ages and it is more frequent in females.[7,16,17] In general, TN does not involve the V1 and V3 distributions without also affecting the V2 distribution.[18] Numbness may develop in patients with chronic TN or as a sequel of surgical treatment.[19]

Pathophysiology

Though there has been much debate over the exact pathophysiology of TN, the neurovascular compression (NVC) theory remains the most plausible explanation. This theory proposes vascular compression of the trigeminal nerve at the point where the nerve leaves the brainstem as the primary pathogenic mechanism for TN.[16,20] More recent and emerging evidence, however, suggests that demyelination of the trigeminal nerve in the REZ plays an important role in the pathophysiology of abnormal neuronal activity and consequent pain perception.[5,10,13,21] In a few cases, TN may be due to the primary demyelinating plaque. Other rare causes include: Infarcts of the brain stem, tumors and other masses of posterior fossa, and rarely amyloid. Once all of these possibilities are ruled out, there remains a small proportion of patients in whom the etiology remains unknown. Histological studies have revealed that compression of the nerve root by an overlying blood vessel results in focal demyelination, with close apposition of demyelinated axons and absence of intervening glial processes.[13] Ectopic action potentials thus generated in the sensory root of the nerve may result in the typical symptoms. Experimental studies have indicated that this anatomical arrangement favors the ectopic generation of spontaneous nerve impulses and their ephaptic conduction to adjacent fibers.[13] Compression of the nerve by an impinging blood vessel is the most common cause of TN. These findings form the rationale for MVD surgery as the treatment for this disorder.[22] On the other hand, some authors still question the NVC theory of TN as such contact of blood vessel loop with trigeminal nerve can be demonstrated in a great majority of the healthy persons.[10,23] In a blinded study, NVC was found with similar frequency on the asymptomatic side in patients with TN, thus questioning the NCV theory. Focal demyelination is not a consistent finding, the observation which has been attributed to the sampling error.[23] Nevertheless, the most common cause of TN is focal compression of the trigeminal nerve root, close to its point of entry into the pons, by aberrant artery or vein. This was first recognized as the cause of TN by Janetta and accounts for 80–90% of cases of TN.[13]

Neuroradiological Evaluation

Bondt *et al*., evaluated 288 patients with magnetic resonance imaging (MRI) and magnetic resonance angiography (MRA) for trigeminal deficits and found that 28.5% of patients had neurovascular compression and 60.2% of patients had normal neuroradiological findings.[24] But the detection of these abnormalities is highly dependent on the MRI resolution. Using a higher resolution, venous or arterial compression can be differentiated, which may help in predicting the recurrence of TN following MVD.[20] However, NCV may not be found at surgery in all the cases of TN. The reported rates of absence of NCV at operation varied from 4% to 89%, with an average of 7.5%.[20] Multiple sclerosis plaque is rare cause of symptomatic TN. Presence of demyelinating plaque, hyperintensity signal in T2-weighted images, along the intrapontine course of the trigeminal pathways is a contraindication for MVD. Ferroli *et al*., reported successful MVD in a patient without multiple sclerosis, despite an intrapontine trigeminal lesion.[25]

Treatment Options

Many drugs have been tried in TN. Carbamazepine is the first line of treatment. About 70% of patient with TN will have good initial pain control with carbamazepine.[26] Most of the remaining 30%, of patients will have initial satisfactory response to other pain controlling medications.[27] Unfortunately, with conventional medical therapies, the mean time of recurrence of symptoms is approximately one year.[28] Other issues associated with drug therapy include: Drug tolerance, warring of drug effect, and serious acute and chronic drug adverse events. The next options for symptomatic relief for patient refractory to medical therapy are invasive and surgical procedures.[12]

Various invasive therapeutic methods, including injection of ethyl alcohol or butyl alcohol into the ganglion, glycerol injection into the trigeminal cistern, peripheral nerve divisions, as well as radiofrequency thermocoagulation of the preganglionic fiber, have been performed for pain relief.[9,29,30] Currently, percutaneous radiofrequency thermocoagulation, stereotactic radiosurgery, and percutaneous microballoon compression (PMC) are the common ablative therapies for TN.[15,29,31] It seems that percutaneous radiofrequency thermocoagulation therapy is more efficacious in pain relieving, but is associated with a higher complication rate compared with stereotactic radiosurgery.[29] However, the percutaneous trigeminal ganglion approach could be dangerous to a certain extent. For example, it can cause intracranial hemorrhage, carotid injury, carotid-cavernous fistula, and other cranial nerve injuries.[32,33] Encouragingly, neuronavigation can eliminate most of the complications associated with traditional fluoroscopy guided foramen oval haunting and improves the success rate of trigeminal nerve targeting.[34] Although a 75% success rate for treating TN by radiofrequency was reported 14 years after the creation of gasserian lesions, other studies have found that only about 20% of patients remained free of pain 6–7 years after this procedure.[9] Regarding results of PMC, Skirving and colleagues reported a recurrence rate of 19.2% within five years and 31.9% over the entire follow-up period.[35] Overall, disadvantages of these procedures include injury to the trigeminal nerve, ganglion or root,

resulting in neurological deficits such as numbness with extended sensory loss, corneal hypesthesia, and deafferentiating pain, which is essentially untreatable.

Microvascular Decompression

In the earlier years, Jannetta strongly supported the hypothesis of microvascular compression and perfected and popularized the MVD operation for the treatment of TN.[36] Thereafter, Barker *et al*., emphasized MVD by demonstrating a 70% cure rate in a 10-year follow-up study.[9] However, 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.[9] Furthermore, 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.[37,38]

Some studies have shown differences between typical and atypical TN, in about 88% of patients with typical TN and 56% of patients with atypical TN a NVC could be demonstrated, compression by a vein is significantly higher in patients with atypical TN than in patients with typical TN. Pain relief by MVD is higher (~80%) in patients with typical TN than in patients with atypical TN (~56%). Probably this may be the most important prognostic factor in MVD.[38] In these patients, the type of vascular compression may play a role in the recurrence of TN symptoms following MVD. The presence of venous compression is a sign of a poor prognosis.[20]

MVD, as a surgical approach, may offer certain definitive benefits. MVD caries a very low risk of anesthesia dolorosa or paresthetic complaints.[39,40] Facial numbness is an occasional sequel of both radiofrequency thermal rhizotomy and glycerol rhizotomy, but it correlates well with pain relief.[9] Whereas facial numbness is rare following MVD.[31] It is perhaps fair to state that all surgical interventions appear to have a similar short-term efficacy, approximately 80%, but the long-term effects of MVD appear to be superior.[41] Since the benefits of MVD are long lasting and the procedure being more invasive, we believe that MVD is more appropriate for younger patients, whereas ablative therapies are usually preferable in patients older than 70 years.[31,42-44]

Complications of Microvascular Decompression

The complications following MVD include cerebrospinal fluid (CSF) leak, cerebellar injury, hearing loss, and facial palsy. CSF leak, the most frequent complication after MVD surgery, may be associated with intracranial infections such as meningitis.[45,46] The incidence of CSF leak following MVD is in the range of 0.9-12%.[46]

Trigemino-cardiac reflex (TCR) is an important complication during anesthesia and may occur during stimulation of any of the sensory branches of the trigeminal nerve.[47] It is characterized by bradycardia, arterial hypotension, apnea, and gastric hypermotility. TCR is defined as a drop in mean arterial blood pressure (MAP) and heart rate (HR) of more than 20% of the baseline values before the stimulus and coinciding with the manipulation of the trigeminal nerve.[47-49] In a retrospective study or 28 patientss, the incidence of TCR during MVD was 18%. Their HR fell 46% and their MABP 57% during operative procedures near the trigeminal nerve as compared with levels immediately before the stimulus. After cessation of manipulation, HR and MABP returned to levels before the stimulus.[49] Thus, it is important for both neurosurgeons and anesthesiologists to take all the precautions to prevent this serious complication during MVD.

Reasons for Recurrences Due to MVD

Factors that have been shown to be significant predict recurrence include: Symptoms lasting for more than eight years, compression of the trigeminal REZ by a vein rather than an artery, and the lack of immediate postoperative pain relief. Female gender also seems to have a higher rate of recurrence.[9] Jannetta noted significant vascular compression in 96% of patients with TN and in 12% of them it was due to a vein.[36] However, the reasons for the high recurrences in patients with venous compression are not clear. Re-exploration is often the strategy in patients with recurrence of symptoms. Interestingly, the rate of negative exploration is quite variable, 18–28.5% depending on the experience of the surgeon.[36] In the retrospective cohort study of MVD by Kalkanis and colleagues the morbidity rates were significantly lower at high-volume hospitals and with high-volume surgeons. The mortality rate was 0.3%, and the rate of discharge other than to home was 3.8%. One or more common complications were seen in 3% of patients.[31]

Lack of immediate postoperative pain relief indicates poor long-term outcomes following MVD.[9] In the study by Barker and colleagues, of the 1182 patients who had MVD, 132 required second operation, 10% of them within 30 days of first operation. Veins or small arteries were found compressing the nerve.[9] Neurological complications like facial numbness was found more frequently after the second operation. The chance of postoperative pain relief was less following the second MVD surgery.[9,19] Minimizing trauma by careful handling of the trigeminal nerve during the operation seems to be the essential component to maintain normal trigeminal nerve function and also to reduce the risk of recurrence.[19] Various studies have shown, 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.[50,51]

Conclusion

MVD is an effective surgical treatment for patients with TN refractory to medical treatment. The indications for alternative surgical methods include: (1) severe associated medical co-morbidities contraindicating general anesthesia; (2) persistence of symptoms even after adequate decompression; (3) technical difficulties in safe repositioning of the vessel; and (4) no demonstrable blood vessel compression.

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