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Microvascular decompression in treatment of trigeminal neuralgia

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Abstract:Background: The choice of therapeutic modality for treatment of TGN is guided by etiology, pathophysiology, clinical presentation, age, and co morbidities. The atypical trigeminal neuralgic pain requires treatment of the underlying conditions such as tumors, multiple sclerosis (MS), post-herpetic and post-surgical conditions. Oral pharmacotherapy is usually the first line of management. Peripheral Percutaneous Techniques incorporate the use of chemical (alcohol, phenol, and glycerol) or thermal energy (radiofrequency ablation) to produce a lesion in the branches distal to the Gasserian ganglion. Percutaneous Techniques on Gasserian Ganglion destroy the nerve fibers by employing either radiofrequency, chemicals (glycerol) or mechanical force (percutaneous balloon compression. Posterior Fossa Procedures can be invasive or non-invasive. Recurring episodes of severe lancinating pain in one or more divisions of the trigeminal nerve characterize trigeminal neuralgia (TN), a prevalent pain syndrome. One of the main causes of TN in the cerebellopontine angle cistern is neurovascular compression (NVC) in the root entry zone (REZ) of the trigeminal nerve. When other treatments for TN have failed, the surgeons recommend microvascular decompression (MVD). Even in patients who do not exhibit obvious signs of neurovascular compression, MVD has been demonstrated to alleviate pain. On top of that, MVD is widely believed to deliver the best combination of long-term patient satisfaction and little pain recurrence. Along with our personal experiences, we conducted a comprehensive evaluation of the topic.

Keywords: Therapeutic Options, Trigeminal Neuralgia, Microvascular decompression.

Introduction

Vascular compression of the trigeminal nerve at the root entry zone (REZ) causes trigeminal neuralgia (TN), which is marked by paroxysmal shock-like pain that is confined to the innervated area of one or more branches of the trigeminal nerve.1-6 Onset often occurs in people aged 40–60, and very rarely in those younger than 40. This does not apply to cases with symptomatic TN caused by tumors or multiple sclerosis. But TN doesn't show its full clinical face until the occasional pain episodes become chronic, intense, and medication-resistant. Medical antiepileptic medicines are the first line of defense. Surgical intervention is necessary for the permanent alleviation of pain in around 50% of TN patients. Surgical options for treatment

include microvascular decompression (MVD) and percutaneous ablative techniques.in [1]With a high percentage of initial success and few major problems, percutaneous ablative treatments appear to be easy and safe to do. Having said that, the time it takes for pain to subside is often really short—often less than a year—and it might be even shorter with further procedures.Three, eight Dandy initially postulated the possibility of neurovascular compression as the origin of TN in 1934. Around 1962, Gardner offered his support for the concept, and Jannetta later proposed MVD as a surgical method for treating TN. In terms of efficacy and patient satisfaction, the MVD method is now considered the gold standard for TN treatment.

Etiology

What is known as TN according to the International Association for the Study of Pain (IASP) is "a sudden, usually unilateral, severe, brief, stabbing, recurrent episodes of pain in the distribution of one or more branches of the trigeminal nerve." A rate of 3-5 per 100,000 people is recorded.From 11 to 15, Middle-aged and older women are more likely to experience TN.those three numbers As a rule, TN will impact the V2 distribution regardless of whether it involves the V1 or V3 distributions.[18] in Chronic TN sufferers or those recovering from surgery may experience numbness.The year 19

Pathophysiology

Despite extensive discussion, the neurovascular compression (NVC) hypothesis continues to be the most credible explanation for TN's pathogenesis. In this view, the main pathogenic mechanism for TN is vascular compression of the trigeminal nerve just after it leaves the brainstem.the numbers 16 and 20 On the other hand, newer research implies that demyelination of the trigeminal nerve in the REZ is a key pathway in the pathophysiology of aberrant neuronal activity and pain perception.the numbers5,10,13, and 21 The main demyelinating plaque can cause TN in rare instances. Brain stem infarcts, tumors, and other masses of the posterior fossa are among the rarest other possible causes, along with amyloid. Even after considering all of these options, there is still a tiny subset of patients for whom the cause is still a mystery. Focus demyelination, with close apposition of demyelinated axons and absence of intervening glial processes, is observed histologically when an overlaying blood artery compresses the nerve root.in [13] Typical symptoms may be caused by ectopic action potentials that are formed in the sensory root of the nerve. Based on the results of the experiments, this structure is ideal for the ectopic production of spontaneous nerve impulses and their ephaptic transmission to neighboring fibers.The most prevalent cause of TN, according to [13], is when a blood vessel presses on a nerve. The findings presented here support the use of MVD surgery to treat this condition.[22] is a However, this kind of blood vessel loop interaction with the trigeminal nerve is visible in most healthy people, hence some writers continue to doubt the NVC explanation of TN.10 and 23 The NCV idea was called into question when a blinded investigation discovered NVC at the same frequency in TN patients without symptoms. It has been suggested that the sampling error is to blame for the inconsistent finding of focal demyelination.[23] However, TN is usually caused by an abnormal artery or vein pressing on the trigeminal nerve root at its entrance into the pons. This accounts for 80–90% of TN cases and was initially identified as the cause by Janetta.in [13]

Neuroradiological Evaluation

Neurovascular compression was detected in 28.5% of the 288 individuals studied by Bondt et al. using MRI and MRA for trigeminal impairments, while normal neuroradiological findings were observed in 60.2% of the patients.[24] However, the MRI resolution is crucial for detecting these anomalies. The ability to distinguish between venous and arterial compression at a better resolution could aid in the prediction of TN recurrence after MVD.In [20], But, in some TN cases, NCV might not be discovered during surgery. The percentage of cases where NCV was not present during surgery ranged from 4% to 89%, with 7.5% being the norm.In [20], Symptomatic TN caused by multiple sclerosis plaque is quite rare. The intrapontine course of the trigeminal pathways must not have demyelinating plaque or hyperintensity signal in T2-weighted images for MVD to be performed. In spite of an intrapontine trigeminal lesion, Ferroli et al. reported a successful MVD in a patient who did not have multiple sclerosis.[25]

Treatment Options

The choice of therapeutic modality for treatment of TGN is guided by etiology, pathophysiology, clinical presentation, age, and co morbidities. The atypical trigeminal neuralgic pain requires treatment of the underlying conditions such as tumors, multiple sclerosis (MS), post-herpetic and post-surgical conditions. Oral pharmacotherapy is usually the first line of management [1-5].

Patients refractory to medical treatment (not relieved by an adequate trial of at least three drugs in sufficient dosage) are the candidates for next step of management i.e. surgical intervention. These may be invasive [microvascular decompression (MVD) of trigeminal nerve], minimally invasive [percutaneous rhizotomy] or non-invasive modalities [stereotactic radiosurgery by gamma knife or cybersurgery] depending upon the nature of procedure performed.

Another way of classification of the management strategy is based on the site of intervention, as below.

- **Peripheral Percutaneous Techniques** incorporate the use of chemical (alcohol, phenol, and glycerol) or thermal energy (radiofrequency ablation) to produce a lesion in the branches distal to the Gasserian ganglion.
- **Percutaneous Techniques on Gasserian Ganglion** destroy the nerve fibers by employing either radiofrequency, chemicals (glycerol) or mechanical force (percutaneous balloon compression).
- **Posterior Fossa Procedures** can be invasive or non-invasive.

(a) Invasive Procedures:

- **Microvascular Decompression (MVD):** It is the procedure of choice for classic TGN with neurovascular compression (NVC). open surgical technique involves the correction of NVC by separation of vessel and nerve, and keeping them apart. The reported success rate of MVD is as high as 83.5% (80–89%) with a severe complication rate of 0.1% [6].
- **Partial Sensory Rhizotomy (PSR):** In those patients without NVC, destructive procedures such as PSR may be considered as an alternative. However, PSR is rarely performed these days.
- **Internal Neurolysis:** where nerve fibers are separated longitudinally causing lesser damage as compared to PSR, thus resulting in lesser sensory deficits [7].
- **Cryotherapy:** involves frozen application at particular branch of the nerve after it has been surgically exposed [80].

(b) Non-invasive Procedure:

- **Stereotactic Radiosurgery (SRS):** ablative non-invasive procedure by means of gamma knife (GK) therapy where irradiation of nerve causes an electric block of pain transmission. The drawback of GK is onset of pain relief which may take several weeks to 2–3 months. If performed as a primary treatment, it has a better prognosis rather than when attempted after surgical intervention [7]. However, it can be done as a repeat procedure even if it fails during first time, but with a lower success rate.
- **Low-level Laser therapy:** uses a single wave length light which alters cell and tissue function. In many studies, it has been found to decrease intensity and frequency of pain without any side effects [82].

Atypical TGN is characterized by constant neuropathic type of pain secondary to trauma, surgery, post-herpes or MS. In these cases, pain is often resistant to standard therapies. Percutaneous techniques and SRS are the preferred modalities of treatment. Neuromodulation procedures alter the nerve activity through targeted delivery of a stimulus either electrical or chemical to trigeminal nerve, peripheral nerves, motor cortex or spinal cord. and have been described in various case series but evidence is not strong enough to incorporate them in routine treatment [7]. These procedures may be classified as:

(a) Invasive Procedures:

- Transcutaneous Electrical Pulsed Stimulation Of Trigeminal Nerve/ Peripheral Nerve Stimulation (Supraorbital, infraorbital, occipital)
- Gasserian Ganglion Pulsed Stimulation
- Motor Cortex Stimulation
- Deep Brain Stimulation

- Spinal Cord Stimulation
- Sphenopalatine Ganglion Blockade

(b) Non-invasive Procedures

- Transcranial Magnetic Stimulation
- Transcutaneous Electrical Nerve Stimulation (TENS)

Pharmacotherapy of Trigeminal Neuralgia:

First-Line Pharmacotherapy:

Carbamazepine and oxcarbazepine are the first-line treatment options for TN and offer meaningful initial pain control in almost 90% of patients[51] although this may not be sustained in the long term. The benefit of these drugs is offset by adverse effects, which lead to withdrawal in up to 40% of patients.[84] Carbamazepine is known for its metabolic interaction with other medications, which can be problematic in elderly people with comorbidities. Oxcarbazepine causes fewer side effects and has lower potential for drug interactions than carbamazepine, though it is more likely to cause excessive central nervous system depression or dose-related hyponatraemia. The tolerability of both these drugs is gender related; women are significantly less tolerant.

The individual response to both drugs varies considerably, hence if one is not effective, then the other one can be tried. If changing over from carbamazepine to oxcarbazepine, then 200 mg of carbamazepine is equipotent to 300 mg of oxcarbazepine. It is important to be aware that the modified-release (retard) version of carbamazepine available is best used when patients have stabilised.

Liquid versions of both drugs are useful when patients find it hard to swallow due to pain severity. While these drugs are effective for control of the paroxysmal pain, their effect on the concomitant continuous pain is usually limited.

Contraindications to using these agents include cardiac conduction problems and allergic reactions. There is a high degree of cross-reactivity Between the aromatic antiseizure medications (carbamazepine, oxcarbazepine, phenytoin, phenobarbital). Carbamazepine and oxcarbazepine do not generally require regular monitoring of serum drug concentrations; in most patients, the drug doses can be titrated or tapered by clinically considering the balance between the efficacy and adverse effects. However, we advocate regular monitoring of renal, calcium and liver function tests. Patients may develop hyponatraemia and a cholestatic picture on liver function testing which, while not usually of clinical concern, need careful monitoring to ensure that they do not progressively worsen. Older women are already at increased risk of osteoporosis and this needs to be monitored in long-term use.

The HLA-B*1502 allele is highly associated with the outcome of carbamazepine-induced Stevens-Johnson syndrome and toxic epidermal necrolysis. This association has been found mostly in the Han Chinese, but not in Caucasian patients. Hence, all Han Chinese patients should be tested for this allele before starting carbamazepine.

Second-Line Pharmacotherapy:

Lamotrigine has been reported to be helpful as an add-on therapy in a small randomised cross-over trial.[8] Lamotrigine can be used in patients who cannot tolerate carbamazepine and oxcarbazepine. It is generally associated with fewer side effects than carbamazepine and oxcarbazepine. The dose of lamotrigine should be escalated slowly as the incidence of lamotrigine-induced rash is well recognised to be dose dependent. About 10% of people taking lamotrigine develop cutaneous reactions. However life-threatening conditions, like Stevens-Johnson syndrome, can rarely occur.

Baclofen can help in TN especially in people with multiple sclerosis who may be using the drug for spasticity. It also shows synergistic actions with CBZ and therefore patients showing inadequate symptomatic benefit can be tried on a combination of the two drugs. However, an RCT demonstrating such efficacy is presently lacking.[8]

Acute treatment for severe exacerbation:

Severe exacerbation during which there is a marked increase in the frequency and intensity of pain, resulting in an inability to eat or drink and may require admission to hospital for rehydration, short-term pain management and long-term optimisation of preventive treatments. Though opioids are frequently used, they are generally ineffective and should be avoided. Topical lidocaine or local anaesthetic injections into the trigger zones can provide transient relief.[97] Intravenous infusions of fosphenytoin (15 mg/kg over 30 min) and lidocaine (5 mg/kg over 60 min) under cardiac monitoring can be highly effective but should be administered by specialized teams with expertise in their use.

Surgical Treatment of Trigeminal Neuralgia:

MVD is the first choice of surgical treatment in patients with classical trigeminal neuralgia, while RFT (ablation treatment) should be the preferred choice when an MRI does not show any vascular contact. RFT is also used as an alternative option when a patient is thought unable to tolerate MVD (9). In clinical practice, compared with the invasive technique of MVD, RFT is minimally invasive. RFT has advantages and limitations in terms of its efficacy and complications. Although its side effects may not be permanent, they cannot be entirely excluded. Repeated puncture may cause unnecessary damage, although precise cannulation may reduce the incidence of complications. Inaccurate positioning is the major reason for puncture failure and is considered a significant cause of pain recurrence and complications (9).

Tennessee has tried several medications. The initial course of treatment is with carbamazepine. Initial pain management with carbamazepine is good for about 70% of TN patients.[26] Other pain-controlling drugs will initially provide a good response for the majority of the remaining 30% of patients.[27] Regrettably, symptoms typically return around a year after undergoing standard medical treatments.[28] in pharmacological resistance, pharmacological effect warring, and severe acute and chronic side effects are additional concerns with drug therapy. When medical treatments fail to alleviate symptoms, invasive and surgical procedures become the next line of defense.[12]

Injecting ethyl or butyl alcohol into the ganglion, injecting glycerol into the trigeminal cistern, dividing peripheral nerves, and radiofrequency thermocoagulation of the preganglionic fiber are all examples of invasive therapeutic procedures that have been used to alleviate pain.Numbers 9, 29, and 30 At present, the most prevalent ablative treatments for TN include stereotactic radiosurgery, percutaneous microballoon compression (PMC), and percutaneous radiofrequency thermocoagulation.References [15, 29, 31]; In terms of pain relief, percutaneous radiofrequency thermocoagulation therapy appears to be more effective than stereotactic radiosurgery; nonetheless, it is linked to a higher incidence of complications.[29] But there's a chance that the percutaneous trigeminal ganglion method is risky. A few examples of the kinds of cranial nerve injuries it might produce are carotid injury, cerebral hemorrhage, carotid-cavernous fistula, and others.references 32 and 33 Upbeat news: neuronavigation boosts the success rate of trigeminal nerve targeting and gets rid of most problems with conventional fluoroscopy-guided foramen oval haunting.on page 34 While one study indicated a 75% success rate for radiofrequency TN treatment 14 years after gasserian lesions were created, another study indicated that only approximately 20% of patients were pain-free 6-7 years after the procedure.[9] Skirving et al. found a recurrence rate of 19.2% within 5 years and 31.9% across the full follow-up period when it came to PMC findings.[35] in On the whole, these treatments can cause neurological deficits like numbness with extended sensory loss, ocular hypesthesia, and deafferentiating pain—essentially untreatable—if the trigeminal nerve, ganglion, or root is injured.

Microvascular Decompression

Jannetta first offered significant evidence in favor of the microvascular compression theory and was instrumental in honing and popularizing the MVD procedure for the treatment of TN.[36] Then, in a follow-up research that lasted 10 years, Barker et al. highlighted MVD by showing a cure rate of 70%.[9] The actuarial recurrence rate of tic pain following MVD is defined differently between series, which makes direct comparisons between them difficult.[9] Additionally, it is crucial to determine if the nerve is being

compressed by a vertebral or basilar artery; operating on patients with basilar compression is associated with a higher risk than operating on patients with superior cerebellar artery compression.References [37,38]
Around 88% of patients with typical TN and 56% of patients with atypical TN have revealed variations between the two types of TN, according to several research. Compression by a vein is substantially greater in individuals with atypical TN compared to patients with normal TN, and an NVC could be shown. Approximately 80% of patients with conventional TN experience pain reduction from MVD, compared to 56% of individuals with atypical TN. Among MVD prognostic factors, this one might be the most crucial.Recurrence of TN symptoms after MVD may depend on the kind of vascular compression in these patients [38]. A bad prognosis is indicated by the presence of venous compression.In [20],
MVD has the potential to provide certain clear advantages as a surgical method. Anesthesia dolorosa and paresthetic symptoms are quite uncommon with MVD caries.39 and 40 Although facial numbness is a rare side effect of glycerol rhizotomy and radiofrequency thermal rhizotomy, it does go hand-in-hand with reduced discomfort.[9] On the other hand, MVD rarely causes facial pain.[31] in Although the short-term effectiveness of all surgical procedures seems to be around 80%, MVD seems to have better long-term effects.[41] The If a patient is less than 70 years old, ablative therapies are typically the way to go, although MVD is better for younger people due to its longer-lasting advantages and more invasive nature.(31, 42, 43, 44)
Mini-Vascular Decompression Risks
Cerebellar damage, hearing loss, facial palsy, cerebrospinal fluid (CSF) leak, and MVD are all potential consequences. Intracranial infections like meningitis may be linked to cerebrospinal fluid leak, the most common consequence following MVD surgery.(45, 46) Between 0.9% and 12% of patients experience a cerebrospinal fluid leak after MVD.[46]
Stimulating any of the trigeminal nerve's sensory branches might trigger the crucial anesthetic complication known as the trigemino-cardiac reflex (TCR).[47] Gastric hypermotility, bradycardia, apnea, and arterial hypotension are the hallmarks of this condition. When the trigeminal nerve is stimulated and the patient's mean arterial blood pressure (MAP) and heart rate (HR) fall by more than 20% from their pre-stimulus values, this is called transcutaneous cardiac resistance (TCR).48,49, 47 There was an 18% incidence of TCR during MVD in a retrospective analysis of 28 patients. Compared to levels just before the stimulus, their heart rates decreased by 46% and their mean arterial blood pressure by 57% during surgical procedures close to the trigeminal nerve. Both HR and MABP went back to their pre-stimulus levels as the manipulation stopped.in the text. Therefore, neurosurgeons and anesthesiologists must take every measure to avoid this potentially fatal problem during MVD.

Reasons for Recurrences Due to MVD

Recurrence is more likely to occur if the following factors are present: symptoms persist for longer than eight years, the trigeminal REZ is compressed by a vein instead of an artery, and there is no rapid relief from pain after the operation. It appears that the recurrence rate is higher among females as well.[9] Significant vascular compression was observed in 96% of TN patients by Jannetta, with veins accounting for 12% of these cases.[36] It is unclear, however, why venous compression patients have such a high recurrence rate. When patients experience a return of their symptoms, re-exploration is frequently the next step. Depending on the surgeon's level of skill, the rate of negative exploration can range from 18.5% to 28.5%, which is extremely varied.[36] Hospitals and surgeons with high patient throughput had much lower morbidity rates in the MVD retrospective cohort research conducted by Kalkanis and colleagues. There was a 0.3% mortality rate and a 3.8% rate of discharge to a location other than home. Just 3% of participants experienced any of the usual side effects.[31] in
Poor long-term results after MVD are indicated by the absence of early postoperative pain alleviation.Barker et al. found that out of 1,178 patients who underwent MVD, 132 needed a second procedure, with 10% of those patients requiring the second procedure within 30 days of the first. Compressing the nerve were veins or tiny arteries.After the second procedure, neurological problems, such as a lack of sensation in the face,

were more commonly observed. After the second MVD procedure, the likelihood of experiencing postoperative pain reduction decreased.9 and 19 Reducing the likelihood of recurrence and maintaining normal trigeminal nerve function appears to depend on minimizing stress to the nerve during surgery.The year 19 Among the several surgical options available today, MVD has the best track record for patient satisfaction and the fewest cases of pain recurrence, according to a number of studies.References [50,51]

Conclusion

When medical treatments for TN fail, MVD can be a lifesaving surgical option. First, if there are serious comorbidities that make general anesthesia unsafe; second, if symptoms continue after sufficient decompression; third, if there are technical challenges to safely reposition the vessel; and fourth, if there is no visible compression of the blood vessel, then alternative surgical methods should be considered.

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