Review Article

Trigeminal Neuralgia: Role of Interventional Pain Physician

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Abstract

Trigeminal neuralgia (TN) or tic douloureux is one of the commonest cause of fascial pain after 50 years of age. It is characterized by recurrent, episodic, lancinating pain over the distribution of trigeminal nerve. There is a lack of certainty regarding the aetiology and pathophysiology of TN. Evidence suggests that the likely etiology is vascular compression of the trigeminal nerve leading to focal demyelination and aberrant neural discharge. Secondary causes such as multiple sclerosis or brain tumors can also produce symptomatic TN. The treatment of TN can be very challenging despite the numerous options patients and physicians can choose from. This multitude of treatment options poses the question as to which treatment fits which patient best. For patients refractory to medical therapy, Gasserian ganglion percutaneous techniques, gamma knife surgery and microvascular decompression are the most promising invasive treatment options. Among them three common interventions commonly carried out by interventional pain physician to provide pain relief are balloon compression, Glycerol rhizolysis and RF rhizotomy.

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Introduction

Trigeminal neuralgia is most common neuralgia and commonest cause of facial pain after 50.It is known to have the worst pain in the world. The incidence of trigeminal neuralgia (TN) is 4.3 per 100,000 persons per year, with a slightly higher incidence for women (5.9/100,000) compared with men (3:4/100,000). Evidence suggests that pain occurs because of pressure on the trigeminal nerve root at the entry zone into the pontine region of the brain stem.[1,2] Compression by tumor or blood vessel may cause local pressure, leading to demyelination of the trigeminal nerve. Results from experimental studies suggest that demyelinated axons are prone to ectopic action potential generation.[1,3] Demyelination has also been shown in cases of TN associated with multiple sclerosis and tumor compression of the trigeminal nerve root. The condition may be severely disabling with high morbidity, particularly in the elderly.[4]

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Diagnosis

Both the International Association for the Study of Pain (IASP) and International Headache Society (IHS) have suggested their own diagnostic criteria for TN.[5] These are remarkably similar and highlight the sudden, explosive nature of the pain . In further descriptions of the condition, both classifications allude to vascular compression, MS and tumors as known aetiological causes. The IASP classification makes a distinction between TN (including MS) and secondary neuralgias (caused by structural lesions and injuries, but not including MS), while IHS separates idiopathic TN from the 'symptomatic form' depending on the presence of a structural lesion; it is not quite clear if vascular compression qualifies as such. Neither approach includes reference to variant forms of TN, which satisfy the diagnostic criteria but display additional features as well.

Definition of trigeminal neuralgia by IASP "Sudden, usually unilateral, severe brief stabbing recurrent pains in the

distribution of one or more branches of the Vth cranial nerve."[5]

Painful unilateral affliction of the face, characterized by brief electric shock like pain limited to the distribution of one or more divisions of the trigeminal nerve. Pain is commonly evoked by trivial stimuli including washing, shaving, smoking, talking, and brushing the teeth, but may also occur spontaneously. The pain is abrupt in onset and termination and may remit for varying periods.[6]

The International Headache Society has published criteria for the diagnosis of classical or symptomatic TN .[7] In classical TN, no etiology can be identified other than vascular compression. On the other hand, symptomatic TN is related to an underlying cause such as tumor compression or multiple sclerosis. It is therefore important to distinguish between the two as the focus in management of symptomatic TN is to treat the underlying cause.

International Headache Society diagnostic criteria for trigeminal neuralgia[7]

Classical

Paroxysmal attacks of pain lasting from a fraction of a second to 2 min, affecting one or more divisions of the trigeminal nerve, and fulfilling criteria B and C

A. Pain has at least one of the following characteristics:
    1. Intense, sharp, superficial, or stabbing
    2. Precipitated from trigger zones or by trigger factors
B. Attacks are sterotyped in the individual patient
C. There is no clinically evident neurologic deficit
D. Not attributed to another disorder

Symptomatic

A. Paroxysmal attacks of pain lasting from a fraction of a second to 2 min, with or without persistence of aching between paroxysms, affecting one or more divisions of the trigeminal nerve, and fulfilling criteria B and C
B. Pain has at least one of the following characteristics:
    1. Intense, sharp, superficial, or stabbing
    2. Precipitated from trigger zones or by trigger factors
C. Attacks are sterotyped in the individual patient
D. A causative lesion, other than vascular compression, has been demonstrated by special investigations and/ or posterior fossa exploration

Another useful classification that is commonly used[8,9]

1. TN1 – idiopathic, spontaneous facial pain that is predominantly episodic;
2. TN2 – idiopathic, spontaneous facial pain that is predominantly constant;
3. Trigeminal neuropathic pain resulting from unintentional injury to the trigeminal nerve due to trauma or surgery;
4. Trigeminal deafferentation pain resulting from intentional injury to the nerve by peripheral nerve ablation, gangliolysis, or rhizotomy in an attempt to treat either TN or other related facial pain;
5. Symptomatic TN due to multiple sclerosis;
6. Post-herpetic TN following a cutaneous herpes zoster outbreak in the trigeminal distribution; and
7. Atypical facial pain that refers to facial pain secondary to a somatoform pain disorder and requires psychological testing for diagnostic confirmation.

Many TN patients may also manifest a combination of TN1 and TN2 characteristics, together or at separate times during the natural course of the disease.

Neurological examination is usually normal in patients with idiopathic TN, although a subtle trigeminal sensory deficit may be detected occasionally.[10] However, cerebellopontine angle tumors or multiple sclerosis may present first as symptoms of TN. so, patients should be asked about neurologic symptoms such as tingling, numbness, loss of balance, weakness in one or more limbs, blurred or double vision, hearing loss, dizziness, headaches, and fits.

Magnetic resonance imaging (MRI) scan of the brain is most useful tool to exclude symptomatic TN due to multiple sclerosis and tumors.[11]

Differential diagnoses for facial pain include headache disorders, temporomandibular joint pain, dental pain, chronic sinusitis, otitis media, as well as myofascial pain.

Pathophysiology

Although there is general agreement that none of the many existing theories fully explain all known characteristics of Trigeminal Neuralgia pain.[12] Current evidence points to the trigeminal nerve rather than the CNS as the site of generation of TN pain.[13] The existing evidence suggests that a slowly evolving process, whether a compression exerted on the nerve by a blood vessel or tumour or alteration of neural functions by an MS plaque at the level of the dorsal root entry zone, leads to increased excitability in some of the trigeminal afferents and subsequently to typical Trigeminal Neuralgia. The trigeminal ganglion in TN is not normal but shows unique pathological changes, such as degenerative hypermyelination and formation of microneuromata, not explained by tissue artefacts, effects of aging or occult disease.[14,15] The ganglion cells appear

mostly intact. While the exact mechanisms of how these changes have come about is not clear. At the site of vascular compression of the trigeminal root, electron microscopic studies show demyelination and remyelination. Neuroablative procedures, the degree of sensory loss correlates positively with the duration of pain relief.[16] The key feature of TN pain is its very dynamic nature, which is diffcult to explain in purely anatomical terms.[13] MS patients suffering from TN, a common finding is the plaque extending into the dorsal root entry zone.[17]Any process at this level can potentially alter the function of the whole neurone. The fact that TN pain is not continuous but paroxysmal speaks against a simple compression induced generation of ectopic impulses at the level of the injury. It is more likely that the paroxysms of pain in this condition represent spontaneous discharges in select neurones whose threshold for repetitive firing has been altered. To comply with the characteristics of TN, such firing should not only occur spontaneously but be produced frequently by innocuous tactile stimuli. Recent observations have shown that dorsal root ganglion cells possess properties that, in certain circumstances, lead to this type of firing behaviour.[18] Increased spike activity can in turn depolarize and cross-excite hyperexcitable neighbouring C cells.[19] Equally, continuous pain in the atypical form can result from the progressive damage to the central terminals of trigeminal afferents, which become the source of continuous ectopic discharges.

Treatment

The treatment of patients with idiopathic TN is often a challenge in clinical practice, conservative management with drug therapy is always the first-line treatment. When drugs are not efficacious or produce intolerable adverse effects, interventional pain treatment or surgery is the possible option. Microvascular decompression (MVD) and Gamma knife surgery (GKS) are surgical options available to patients with TN.

Pharmacological treatment

There is huge variety of pharmacological and surgical treatment options that are used widely. Carbamazepine has been used as 1st line of oral medication for many decades in the treatment of TN, and it is the drug of choice.[20,21] Carbamazepine may even have diagnostic utility because patients with classical TN will respond well to it. Patients with symptomatic TN or other causes of facial pain are less likely to respond to carbamazepine. Doses of carbamazepine range between 200 and 1200 mg daily.

Other medications with reported efficacy in TN include oxcarbazepine, baclofen, and lamotrigine.[22-24] Other medications such as topiramate, gabapentin, pregabalin, and levetiracetam have shown success in treating TN, but evidence is limited. Patients should receive an adequate trial of at least three drugs including carbamazepine before surgical or interventional treatment is considered.[25]

Percutaneous interventional procedures

This review will focus on the role of balloon compression, Glycerol rhizolysis and RF rhizotomy in the treatment of TN. These procedures can be done on day case basis and excellent outcome with minimal intervention.

Balloon compression

In balloon compression, a small balloon is introduced percutaneously using a needle to compress the trigeminal ganglion. Pain relief was immediate in more than 80% of patients.[26]Complications include severe bradycardia or asystole, corneal anesthesia, facial sensory loss or dysesthesia, and masseter weakness.

Glycerol rhizolysis

The procedure is performed under fluoroscopic guidance initially the patient in the supine position and head extended. The C-arm is rotated to obtain an oblique submental view to visualize the foramen ovale. The skin entry point is ~2–3 cm lateral to the commissura labialis (angle of the mouth) on the affected side. A needle is introduced into the trigeminal cistern and anhydrous glycerol is injected patient in the sitting position with the head flexed and patient remain in same head flexed position for another 2 hours so Glycerol remain in trigeminal cistern. As glycerol is hyperbaric, it will sink and produce discrete neurolytic lesions of the second and third divisions of the trigeminal nerve. Pain relief has been reported to be more than 90% in one study with a recurrence rate of 23% after a mean period of 30 months.[27]

Radiofrequency rhizotomy

Patients with TN who have good to excellent pain relief with a diagnostic trigeminal ganglion block may be suitable candidates for percutaneous RF rhizotomy. It is performed by destruction of the trigeminal ganglion or roots using RF. RF is the commonest percutaneous procedure used to treat refractory TN, especially in elderly patients or who are poorly controlled with drugs.

Procedure

This can be done as day case basis.Patients are fasted for at least 6 h before the procedure. Prophylactic antibiotic is administered 1 h before the procedure. Intravenous access

and standard monitors including electrocardiogram, blood pressure monitoring, and pulse oximetry is a must. Sedation may be needed to increase patient comfort and reduce anxiety.

The procedure is performed under fluoroscopic guidance with the patient in the supine position and head extended. The C-arm is rotated to obtain an oblique submental view to visualize the foramen ovale. The needle trajectory follows a straight line in tunnel view, directed toward the pupil when seen from the front and passes 3 cm anterior to the external auditory meatus when seen from the side.[28]

A 22-gauge, 10-cm RF cannula with a 5-mm active tip is used commonly. After administration of local anesthesia, the cannula is advanced tunnel view to the X-ray beam towards the foramen ovale. A finger can be placed in the oral cavity to make sure that the buccal mucosa has not been perforated. After putting cannula in foramen ovale, the depth of the cannula inside the Meckel's cavity is ascertained on the lateral fluoroscopic view. The electrode is advanced ~2–4 mm further through the canal of the foramen ovale such that the tip of the electrode reaches the junction of the petrous ridge of the temporal bone and the clivus.The stylet is then removed from the cannula, and aspiration is performed to ensure that there is no blood. Test stimulation is mandatory before RF lesioning for lesioning actual pathological division. The mandibular nerve lies in lateral part of the foramen ovale. If the nerve is stimulated at 2 Hz between 0.1 and 1.5 Hz, muscle contraction of the lower jaw may be seen. This confirms that the needle tip is lying on the trigeminal roots. Next, paresthesia in the concordant trigeminal distribution of the patient's usual symptoms (V1, V2, or V3 divisions) at 50 Hz, 1 msec pulse duration should be reproducible at 0.1–0.5 V.[28,29] If paresthesia is obtained above 0.5 V stimulation, the needle should be redirected to get the same response at a lower voltage. RF lesioning at 80°C is carried out for 90 sec. The needle can be repositioned to repeat RF lesioning if more than one branch of the trigeminal nerve is involved.

Pulsed RF (PRF) is a nondestructive method of delivering RF energy to the trigeminal ganglion. In contrast to conventional RF described above. Short bursts of RF current at 42°C are generated with long pauses between bursts. Thermal lesions are not produced by PRF, but recent evidence suggests that microscopic damage to axonal microfilaments and microtubules can occur, with greater changes seen in C fibers than A-β or A-δ fibers.[30] However, a recent randomized controlled trial comparing conventional RF with PRF showed that PRF was not effective in reducing pain in patients with TN.[31] Therefore, PRF cannot be recommended as the standard therapy for rhizolysis of the trigeminal nerve.

Complications of percutaneous RF rhizotomy of the trigeminal ganglion include diminished corneal reflex (5.7%), masseter weakness and paralysis (4.1%), dysesthesia (1%), anesthesia dolorosa (0.8%), keratitis (0.6%), and transient paralysis of cranial nerves III and VI (0.8%). Permanent cranial nerve VI palsy was observed in two patients, CSF leakage in two, carotid-cavernous fistula in one, and aseptic meningitis in one, found in a large scale study of 1600 TN RF rhyzotomy[32]

After percutaneous RF rhizotomy, initial pain relief can be achieved in 98% of patients, as high as that obtained with MVD. Among the various interventional pain therapies, RF rhizotomy offers the highest rate of complete pain relief.[33]Although 15%–20% of patients may experience recurrence pain in 12 months. It can be repeated in the same patient if required.

Conclusion

Secondary causes of TN should rule out, especially in young patients or patients with bilateral symptoms suggestive of multiple sclerosis. MRI scans should be obtained to detect the presence of vascular compression of the trigeminal nerve. Medical therapy should tried first for the treatment of TN if medical treatment is in adequate interventional pain treatment can be considered in patients who have persistent pain despite drug therapy or who are unable to tolerate adverse effects of drugs.

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