REVIEW ARTICLE

Trigeminal Neuralgia: A Contemporary Narrative Review

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ABSTRACT

Introduction: **Trigeminal neuralgia (TN) is an extremely debilitating condition characterized by sudden, severe and electric shock like recurrent episodes of facial pain.**

Clinical features: The pain frequently occurs unilaterally on the face with right side of the face being commonly affected than the left.

Etiopathogenesis: **Trigeminal neuralgia is often caused by vascular compression of the root of the trigeminal nerve.**

Diagnosis and Investigations: **Diagnosis of TN is usually achieved through classical presentation of symptoms and signs. However, brain imaging is required to make differentiation between sub-types of TN.**

Medical management: **It is treated by either medication or by surgeries. Carbamazepine (CBZ) remains the first line of treatment followed by antiepileptic drug such as gabapentibe, pregabaline and lamotrigine. A derivative of CBZ known as Oxcarbazepine is found to be more efficacious with minimal side effects. Among the newer therapeutic options includes onabotulinumtoxin A and topiramate. Recent advances in pharmacotherapy include vixotrigine, a novel sodium channel blocker and calcitonin gene-related peptide. However, there is limited due to small scale in-vitro studies.**

Surgical management: **Refractory cases of Classical TN or secondary TN cases are managed with surgical approach which includes microvascular decompression, rhizotomy and radiosurgery.**

Rational of the review: **The aim of this review article is to summarize the current advances in diagnosis and treatment.**

KEY WORDS

neuropathic pain, orofacial pain, trigeminal neuralgia, carbamazepine, microvascular compression, monoclonal antibodies

INTRODUCTION

Neuralgia is referred as relentless pain along the course of a nerve, due to injury or irritation of the nerve. TN is a well-recognized disorder characterized by sudden, severe lancinating pain along the course of the nerve. TN is defined as "sudden, recurrent, severe pain in the distribution of one or more of the branches of the fifth cranial nerve"[1)]. Episodes of pain are most frequent in the 2nd and 3rd divisions of the trigeminal nerve, and the right side of the face is most commonly affected. In few individuals, the episodes of the pain are very well characterized, recurring with the similar intensity and in the similar distribution[2)]. Despite the severity of the pain episodes, the majority of TN patients are symptom-free in between attacks and have a normal clinical assessment. An episode is usually only a few seconds long; however numerous episodes may be shown in rapid succession. Episodes can happen at any time during the day and last anywhere from a few days to several months. Pain-free periods, on the other hand, can endure for months or even years.

TN is known to affect both men and women but and increased female predominance was reported. TN is most frequently reported in individuals over fifty years of age[3)].

ETIOLOGY AND PATHOGENESIS

According to the current theory, the vascular compression of the sensory root of the trigeminal nerve near the brainstem leads to the episodes of pain. The vascular compression may lead to focal demyelination and remyelination process, possibly enabled by microvascular ischaemic changes. Most of the TN cases are idiopathic[4)].

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Table 1: The most common pain that might mimic TN

Dental pain	• Dental caries • Pulpitis • Dental abscess • Periodontal disorders • Pericoronitis • Cracked tooth
Neuropathic pain	• Vagoglossopharyngeal neuralgia • Geniculate neuralgia • Postherpetic neuralgia • Posttraumatic pain • Trigeminal neuropathy • Atypical facial pain • Occipital neuralgia
Headache disorders	• Cluster headache • Trigeminal autonomic cephalalgia
Tumours:	• Acoustic neuromas, • Meningiomas
others	• Maxillary sinusitis • Temporo-mandibular joint disorders • Neuralgiform of short onset headache attacks accompanied with cranial autonomic symptoms • Neuralgiform of short onset headache attacks accompanied with conjunctival injection and tearing

In a rat investigation, Vos and colleagues discovered that modest compression of the infraorbital branch of the trigeminal nerve causes a pain condition with facial allodynia and hyperalgesia[5]. Histologic examination of the compressed nerve revealed focal inflammation, significant demyelination-remyelination, and a "neuroma in continuity." **Devor *et al***., analyzed surgical biopsy specimens from TN patients who had presumed vascular compression[6]. The authors noticed axonopathy and loss of axons, demyelination, dysmyelination, residual myelin debris and also an increase in collagen was observed.

Clinical features

Patient usually report acute episodes of sharp, shooting, stabbing pain that last anywhere from a few seconds to several minutes and pain feel like electric shocks. Pain is seen experienced along the course of the trigeminal nerve, over the region of the forehead, eyes, lips, gingiva, teeth, jaw, and cheek. Talking, brushing one's teeth, touching one's face, chewing, or swallowing can all cause pain[7].

The mandibular division, followed by the maxillary division, is the most usually affected of the three divisions of the trigeminal nerve. The ophthalmic branch is rarely involved, and discomfort in the area of the scalp can be perceived as originating from the nose, eyes, or head[8-10].

Copp and LeBlanc reported a case of trigeminal neuralgia affecting the ophthalmic branch inwhich the patient reported with headaches around the left orbital region[11]. Dufour reported a case of trigeminal neuralgia in an old man who presented with stabbing pain in eye[12].

TN does not trigger pain directly in the eye itself. It can, however, trigger sudden, sporadic, shock-like pain around the eye if the ophthalmic branch is affected[9]. The right side of the face is more frequently affected than the left[9]. In severe cases, pain episodes might occur hundreds of times every day. Between attacks, some individuals may go months or years without experiencing any symptoms. Affected individuals rarely have pain surges when sleeping. Patients endure sudden pain attacks prompted by non-painful stimuli such as touching their faces, chewing, speaking, or brushing their teeth[13].

DIAGNOSIS

TN is basically diagnosed by its typical clinical presentation. The identification of TN rests on the clinician's skill to identify a characteristic signs and symptoms which describe the disorder[14]. Diagnostic local anesthetic blocks given at the trigger points and along the distribution of the nerves should eliminate all paroxysms of pain. This inhibitory test also helps to differentiate atypical facial pain and other forms of trigeminal neuralgia and neuritis.The fact that particular activities provoke the discomfort is a vital clue to the diagnosis of TN[15].

Differential diagnosis of trigeminal neuralgia[16-20]

Other causes of orofacial pain and headaches must be distinguished from TN. Table 1 summarizes the most common pain that might mimic TN.

Investigations

TN is diagnosed based on the typical clinical presentation and history. It is very essential to carry out investigations to rule out symptomatic TN. A large number of patients have symptomatic TN as a result of another disease process; any patient with TN symptoms should have diagnostic brain imaging scans as part of their first examination. A comprehensive neurological examination would aid in the detection of any neurological deficits, areas of involvement, and trigger zones[17,21].

Magnetic resonance imaging (MRI) of the brain is considered the gold-standard to exclude secondary causes of TN. High-resolution MRI may be helpful in identifying vascular compression. If magnetic resonance imaging (MRI) is not an option, a computed tomography (CT) scan of the head and a CT cerebral angiography can be performed instead. Neuroimaging is also useful for subdividing the symptoms into idiopathic and classical TN, so that classical TN patients can be evaluated for trigeminal microvascular decompression when necessary[15,17].

MANAGEMENT

The treatments options for trigeminal neuralgia include medical management and surgical management.

MEDICAL MANAGEMENT

Medical management is the first line of treatment in most of cases with TN. Medical treatment is frequently adequate and effective, with surgical intervention only being considered if pharmacologic treatment option fails. For the vast majority of patients, medical therapy is appropriate treatment.

Carbamazepine: Carbamazepine (CBZ) is the drug of choice for TN. It is an anticonvulsant medication that inhibits synaptic transmission in the trigeminal nucleus by blocking sodium channels that are reliant on usage[22]. Carbamazepine is usually taken twice a day in doses of 100 to 1200 mg. The high incidence of adverse effects, such as ataxia, nausea, and vomiting, drowsiness, and vertigo, bone marrow depression, prolonged leukopenia, and aplastic anaemia, arethe major limiting factor in its use. Therefore, Patients on carbamazepine should have their liver function, CBC and serum electrolytes test done before starting and at 1, 3, and 6 months after treatment[23].

Oxycarbazine: Oxcarbazepine is a derivative ofCBZ. It is rapidly converted into its pharmacologically active metabolite. Oxcarbazepine is a suitable alternate medication to carbamazepine. Better tolerability, predictable metabolism, and fewer interactions with other medications can advantage over carbamazepine[24].

Gabapentin: an anticonvulsant drug. It is usually used in the treatment of epilepsy or migraines. It is also advised in the treatment of TN. Adverse effects of this drug are dizziness and drowsiness which go away by itself[25].

Phenytoin (Dilantin): It works in the same way to carbamazepine, but it may be less effective. Gingival hyperplasia, hirsutism, sedation, and folate insufficiency are some of the side effects. When carbamazepine monotherapy wears off, as it often does after a year or more, it may provide treatment as an add-on medicine[26].

Baclofen: It is a muscle relaxant that can be prescribed, alone or in combination with carbamazepine / oxcarbazepine. 10 mg/d is considered sufficient, if needed, it can be increased to 60-80 mg/day[27].

Botulinum toxin type A: Botulinum toxin when used with other systemic drugs may reduce pain intensity. Only Botulinum toxin type A should be considered for the medium-term management of TN due to delayed onset of action. The recommended dose is 25-195 units /12 weeks. Adverse effects includes transient bruising at injection site,

facial asymmetry, drooling and difficulty in chewing[28].

Novel / additional pharmacotherapy

Calcitonin gene-related peptide (CGRP) receptor antagonist- These are neuropeptides seen contributing in inflammatory and nociceptive pathway at both peripheral and central level. They includes monoclonal antibodies such as erenumab and fremanezumab which are targeted against the CGRP receptor and thus assist in manipulating nociceptive response[29]. They have been successfully tried and got FDA approval for migraine[30]. With the reports calming raised level of these neuropeptides in cerebrospinal fluid and plasma of TN, the research of their receptor antagonist in the treatment of TN have considered. Recently, in a case series by Parascandolo E *et al*., concluded therapeutic benefits of Erenumab in refractory cases of TN[31].

Injections for temporary relief

It causes neurolysis of the nerve and hence provides relief temporarily. Injection may lead to fibrosis of the area and surgery in the area offibrosis becomes difficult[32].

Alcohol block[33,34]

About 0.5~1.5 ml of 80~100% alcohol is injected in the whole branch & smaller peripheral nerve branches. Both External approach and intraoral method can be used to block the various nerve, depending on their involvement.

Gasserian ganglion injection

In this technique about 2 to 4 ml of novocaine is injected into the peripheral nerve trunk repeatedly for 6 to 7 times for 6 weeks. Indications for injections include, debilitating patient, as a palliative treatment in cancer patient and where nerve avulsion is contraindicated. The disadvantages include, relief of pain for shorter duration since the nerve regeneration is fast and the injection may lead to fibrosis of that area.

SURGICAL TREATMENT

As patients experience breakthrough pain, the medications used to treat TN often lose their effectiveness over time. Surgery is a viable and successful alternative for patients who have failed to respond to medicinal therapy[35]. All trigeminal neuralgia patients should have an MRI before considering surgery, with the posterior fossa receiving special consideration. Other causes of trigeminal nerve compression, such as mass lesions, big arteries, or other vascular abnormalities, should be ruled out by imaging[36].

Types of surgical procedures are either ablative procedure that destroys the nerve ornon-ablative that preserve the nerve.

Ablative procedures

These include, alcohol injections, percutaneous procedures on the gasserian ganglion (Chemical method using gylcerol, thermal method using radiofrequency thermocoagulation and mechanically using balloon compression in Meckel's cave), neurectomy, cryotherapy and peripheral acupuncture[35,36].

Microvascular decompression and medullary tractotomy Glycerol Rhizotomy

Injection of 0.1 - 0.2 ml of glycerol into the Meckel's cave, through a percutaneous needle placement is carried out in this technique.There has been a high success rate with low morbidity reported. The recurrence rate of this operation is rather high[37].

Radiofrequency thermocoagulation (RFT)

RFT is most frequently performed at the level of trigeminal ganglion and sensory root. About 60 to 70 degrees when applied to the peripheral nerves leading to selective destruction of small nerve fibers thus eliminating abnormal pain. Sensory loss is the disadvantage of this procedure. Serious but unusual complications of RFT are cranial nerve palsies meningitis, carotid-cavernous fistula and abscess[38].

In this technique a 22 gague insulated needle except at its tip is passed medial to the mandibular ramus and through the foramen ovale, to rest at the ventral aspect of the trigeminal ganglion in the meckle's cavity. After fluoroscopic checking of the location the patient still awake is given a mild electrical stimulus through the needle tip to elicit paresthesia and determine whether the needle is in proper position. When this is confirmed GA or deep sedation given and one or two lesions of 60-70 degrees are made for 30 second each[39].

Balloon compression

Compression of the ganglion appeared to have similar results to root decompression procedures in many trials. A guide needle is placed into the foramen ovale and a fogarty catheter is advanced until its tip lies in Meckel's cave under fluoroscopic supervision. The balloon is gently inflated with 0.5-1.0 ml of contrast dye until it occupies the cave and ensures appropriate compression for 1-6 minutes[40].

Peripheral neurectomy

This procedure is based on the concept of splitting or avulsing the peripheral branches of the trigeminal nerve, to achieve accurate and long-term pain relief. First the involved branch of the nerve should be confirmed by local anesthetic test. Then the surgery should be carried out. Neurectomy or nerve avulsion provides relief for only 6-12 months. Both intraoral and extraoral approaches can be used for this procedure[41]. Areas where neurectomy carried out are, mental nerve, infra orbital nerve, lingual or buccal nerve and inferior alveolar nerve.

Cryotherapy

Peripheral cryotherapy was first described by Lloyd in the year 1976. It is a simple surgical technique in which the affected peripheral branches of Trigeminal nerve are exposed and frozen by direct application of a cryoprobe with a tip temperature from -50 to -70 degrees .The effects last less than 6-12 months. This is a safest procedure and has shown different degrees of pain reduction success[42].

Microvascular decompression (MVD)

The most common surgical technique for TN is microvascular decompression. MVD is frequently conducted on young, healthy people who only have pain in the ophthalmic division. The MVD is based on the theory that veins close to the trigeminal nerve root compress it, causing aberrant nerve stimulation to occur[43]. Under general anaesthesia, this operation is conducted by incising the skin behind the ear and executing a 3-cm craniotomy. The trigeminal nerve is revealed once the dura is retracted, and it is identifiable by an artery loop squeezing the nerve as it reaches the pons. After that, Teflon felt is used to cushion the problematic vascular anatomy. Dizziness, temporary facial palsy, cerebrospinal fluid leaks, meningitis, cerebellar stroke, and hearing loss are all serious consequences that can occur in 1-5 percent of cases. MVD is known to provide the maximum rate of long-term patient satisfaction with the minimum rate of pain recurrence[44,45].

Gamma Knife surgery (Brain stereotactic radiosurgery)

Gamma knife surgery has recently been proven as an effective treatment for trigeminal neuralgia. Lars Laskell developed gamma knife surgery as an alternative to surgery. In this procedure, a dose of focused radiation is directed to the root of trigeminal nerve. In this process radiation damages the trigeminal nerve and reduces or eliminates pain. It is a painfree procedure and it can be performed without anesthesia. About 70% of patients experience significant pain relief within weeks. The relief may last for several years, but in few cases the nerve may recover and restart transmitting pain[46].

The success of radiosurgery demands a very accurate stereotactic system because the target is small. In a recent study by Regis *et al*., the control rate was up to 83%, with a complication rate of 6% facial anesthesia and 4% hyperesthesia was noted[47].

CONCLUSION

TN is a very painful disorder characterized by sudden, severe lancinating pain along the course of the nerve. The diagnosis and manage-

ment of TN is a challenging process and multidisciplinary team approach is required.Early and prompt diagnosis is very important to frame a treatment plan. Medical management with carbamazepine/oxycarbamazepine is the first line of treatment before considering ablative procedures. As treatment options become more invasive, the outcome improves, but the adverse -effects increases. Therefore, it is important to customize the treatment plan according to the patient's conditions.

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