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Review Article

Trigeminal neuralgia: recent approach in classification, diagnosis and management

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ABSTRACT

Pain and fear of pain continue to be the commonest and strongest motivation for the people to seek facial pain treatment. Pain is a personal experience of the sufferer that cannot be shared and wholly belongs to the sufferer. Trigeminal neuralgia (TN) is a notable facial pain disorder resulting in periodic severe pain that produces one of the most severe kinds of pain known to mankind. Treatment of this debilitating condition may be varied, ranging from medical to surgical interventions. However antiepileptic drugs are commonly used for its treatment. This article brings out the recent approaches in diagnosis and treatment of trigeminal neuralgia.

Keywords: Botulinum toxin, Drug treatment, Microvascular decompression, Pregabalin, Surgical treatment, Trigeminal neuralgia

INTRODUCTION

The International Association for the Study of Pain (IASP) has defined pain as "an unpleasant sensory and emotional experience associated with actual or potential tissue damage".[1] Pain and fear of pain continue to be the commonest and strongest motivation for the people to seek facial pain treatment. Pain is a personal experience of the sufferer that cannot be shared and wholly belongs to the sufferer. Trigeminal neuralgia (TN) is a notable facial pain disorder characterized by sudden, severe, brief, stabbing or lancinating recurrent episodes of pain in the distribution of one or more branches of the trigeminal nerve.[2] It is one of the classical neuropathic pain condition that have been known for centuries. The first known description of trigeminal neuralgia or a similar condition was written in second century AD by Aretaeus of Cappadocia, a contempary of Galen, making reference to a pain in which 'spasm and distortion of the countenance take place.[3]

The first full account of trigeminal neuralgia was published in 1773 when John Forthergill presented a paper to the Medical Society Of London. He described the typical features of the condition in detail, including paroxysms of unilateral facial pain, evoked by eating or speaking or touch, starting and ending abruptly and associated with anxiety.[3]

The trigeminal nerve which is commonly affected in this disease is the largest cranial nerve and is of mixed type nerve that provides sensory innervations to the face and mucous membranes of the oral and nasal cavities and motor innervations to the muscles of mastication. This nerve has three divisions namely ophthalmic, maxillary, and mandibular nerves. (Figure 1) The sensory portion of the trigeminal supplies touch, pain, temperature to the face. The motor division of the nerve are distributed in mandibular nerve and supplies the muscles of mastication, masseter, temporal, pterygoid, mylohyoid, and digastric muscle.[4]

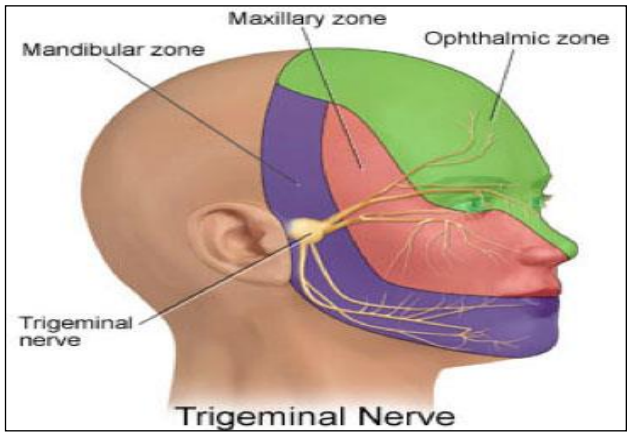

Figure 1: Trigeminal nerve.

The prevalence of trigeminal neuralgia in the general population is 0.015% and overall incidence ranges from 12.6 to 27 per 100,000/year which increases with the advancing age. Middle aged and elderly persons are primarily affected, higher incidence is seen in women with 5.9 cases per 100,000 in females as compared with men with 3.5 cases per 100,000 in males.[5,6] Etiologic theories of Trigeminal Neuralgia.[7] First theory is diseases-related, second theory is due to direct trauma to the nerve and the third theory suggests the polyetiological origin of the disease. In reality, for most patients with TN, there is no identifiable cause.

Compression of the trigeminal nerve root caused by

- Intracranial vascular abnormalities e.g. aberrant loop of the superior cerebellar artery, aneurysm of the intrapetrous portion on the internal carotid artery.
- Intracranial tumours
- Petrous ridge (Bacillar compression).
- Foreign object
- Bone lesion such as osteoma.

Multiple sclerosis

Others

- Trauma
- Viral infection (post-herpetic neuralgia)
- Ratner's jawbone cavities
- Infiltrative disorders of the trigeminal nerve root or gasserian ganglion
- Trigeminal ganglion is not normal but show unique pathological changes such as degenerative hypermyelination
- Familial occurrence as reported in Charcot Marie-Tooth disease
- Sarcoidosis and Lyme disease neuropathy

Although substantial advances have been made in the understanding the pathophysiology of TN over the past 2 decades but still many basic question and problems remained unanswered such as:

- There is no objective test for Trigeminal neuralgia
- Trigeminal neuralgia patients have no clinical sensory abnormality
- Trigeminal nerve compression does not produce trigeminal neuralgia in animals

Classification and diagnostic criteria

Trigeminal neuralgia can be classified based on etiology or symptoms

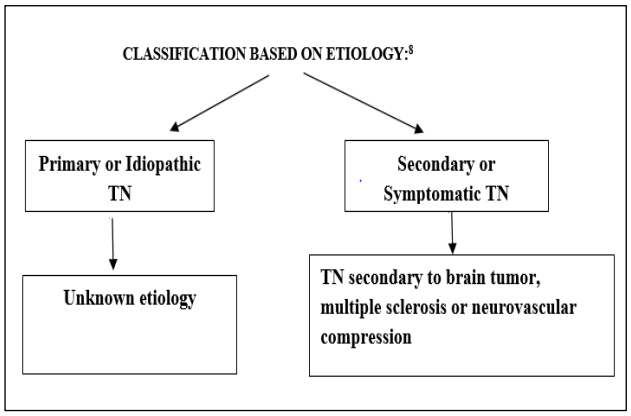

Figure 2(a): Classification based on etiology.

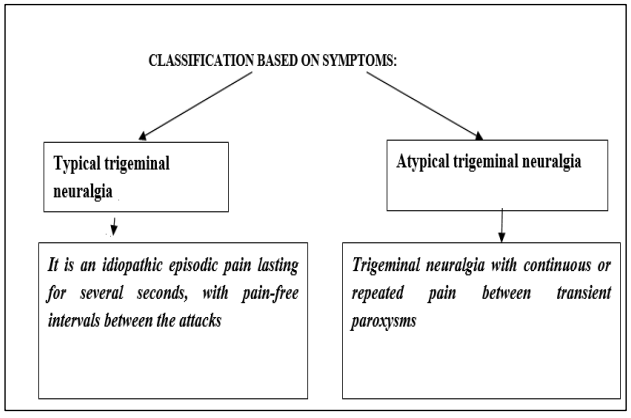

Figure 2(b): Classification based on symptoms.

Although the hallmark findings of TN were known for centuries, White and Sweet made a significant contribution

by putting forward more accurate diagnostic criteria for TN. This criterion rapidly gained popular acceptance with clinicians and scientists, and the 'Sweet criteria'' are still commonly used worldwide to define the diagnosis of TN.

Sweet diagnostic criteria for TN[9]

- The pain is paroxysmal.
- The pain may be provoked by light touch to the trigger zones.
- The pain is confined to the trigeminal distribution.
- The pain is unilateral.
- The clinical sensory examination is normal.

The current International Headache Society criteria (IHS) and International Classification of Headache Disorders II (ICHD-II) establishes trigeminal neuralgia as a clinical diagnosis under the classification of cranial neuralgias and central causes of facial pain (ICHD-II diagnostic code 13). The diagnosis of TN (diagnostic code 13.1) replaces the earlier term ''Tic douloureux''. ICHD-II further subdivides trigeminal neuralgia into classic TN and symptomatic TN.

Classic TN (13.1.1)

It is the most common idiopathic form of disorder. It is defined as "a unilateral disorder characterized by brief electric shock-like pains, abrupt in onset and termination, 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/or brushing the teeth (trigger factors) and frequently occurs spontaneously. Small areas in the nasolabial fold and/or chin may be particularly susceptible to the precipitation of pain (trigger areas). The pain usually remits for variable periods." The ICHD-II specific diagnostic criteria for "classic TN" can be listed as follows:[10]

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

B. Pain has at least one of the following characteristics:
1. Intense, sharp, superficial or stabbing component
2. It is precipitated from activation of trigger areas or by triggering factors
   - Attacks are stereotyped in the individual patient
   - There is no clinically evident neurological deficit
   - Not attributed to another disorder

Symptomatic TN (13.1.2)

It has the same key features as that of TN but results from another disease process (such as multiple sclerosis or a cerebellopontine angle tumor). It is defined by IHS as 'pain indistinguishable from classic TN but caused by a demonstrable structural lesion other than vascular compression.''

The ICHD-II diagnostic criteria for symptomatic TN are as follows:

A. Paroxysmal attacks of pain lasting from a fraction of a second to two minutes, with or without persistence of aching between paroxysms, affecting one or more divisions of the trigeminal nerve and fulfilling next two criteria B and C

B. Pain has at least one of the following characteristics:

- Intense, sharp, superficial or stabbing type.
- Precipitated from trigger areas or by trigger factors
- Attacks are stereotyped in the individual patient.
- Causative lesion, other than vascular compression, has been demonstrated by special investigations and/or posterior fossa exploration.

Table 1: International classification of headache disorders 3 beta diagnostic criteria.[11]

Classical trigeminal neuralgia (CTN) ICHD-3 beta diagnostic for CTN	PTN (Painful trigeminal neuropathy) ICHD -3 beta diagnostic criteria for PTN
A. At least 3 attacks of unilateral facial pain fulfilling criteria B and C	A) Unilateral head and/or facial pain persisting or recurring for 3 and fulfilling criteria C
B. Occurring in one or more divisions of the trigeminal nerve with no radiation beyond the trigeminal distribution.	B) Presence or history of herpetic lesions along distribution of TN, identifiable traumatic events, diagnosis of MS, a presence of space occupying lesion, or another disorder capable of causing PTN.
C. Pain has at least 3 of the following 4 characteristics: • Recurring in paroxysmal attacks lasting from a fraction of a second to 2 minutes. • Severe in intensity • Electric shock like shooting, stabbing, or sharp in quality • Precipitated by innocuous stimuli to the affected side of the face	C) Evidence of causation of PTN demonstrable
D) No clinically evident neurologic deficit	D) Not better accounted for by another ICHD-3 diagnosis
E) Not better accounted for by another ICHD-3 diagnosis	

Table 2: Differential Diagnosis of TN.[15]

Diagnosis	Features that differentiate from TN
Cluster headache	Longer lasting pain, orbital or supraorbital
Dental pain	Localized, related to biting or hot or cold foods
Giant cell arteritis	Persistent pain, temporal, jaw claudication
Glossopharyngeal neuralgia	Pain in tongue, mouth, throat
Migraine	Longer lasting pain, photophobia, phonophobia
Multiple sclerosis	Eye symptoms, other neurologic symptoms
Trigeminal neuropathy	Persistent pain, associated with sensory loss
Postherpetic neuralgia	Continuous pain, tingling, history of zoster.
Intracranial tumor	May have other neurologic symptoms and sign
Otitis media	Pain localized to ear
TM joint syndrome	Jaw abnormality, tenderness at jaw
SUNCT (shorter lasting, unilateral neuralgiform, conjunctival injection and tearing information)	Ocular or periocular autonomic symptoms

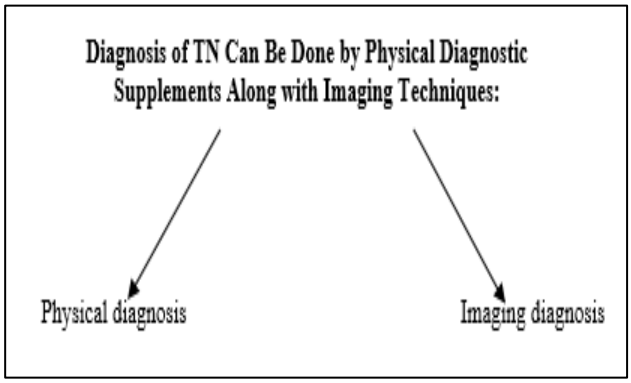

Figure 3: How diagnosis of TN can be done?

Physical diagnosis

TN is a clinical diagnosed and the patient's history is very critical in its evaluation. Diagnosis is made on the basis of characteristic of pain in the trigeminal nerve distribution. Careful interview and physical examination are needed for the diagnosis. Approximately 5% of patients with TN can present with brain tumor. When a patient is diagnosed with TN, MRI is recommended to exclude tumor, cyst or multiple sclerosis (MS). Yokono et al reported a case of hysterical conversion manifesting as TN because some time it is impossible to distinguish psychosomatic disorder from TN.[12]

Imaging diagnosis

MRI-brain reveals tumors or MS with great precision and neurovascular compression with a fair degree of precision. In patient of TN, imaging showed a close relationship between the region of neuralgic manifestation and the site of trigeminal nerve compression.[13] As progress in diagnostic imaging continues, the accuracy of the imaging diagnosis has increased. However, MRI-imaging does not differentiate a symptomatic nerve with neurovascular compression from an asymptomatic nerve with neurovascular compression.[14] Furthermore MRI cannot differentiate TN patients with neurovascular compression whose symptoms will be improved after microvascular decompression (MVD) from those who will remain symptomatic after MVD. Neither diagnostic imaging nor electrophysiologic testing can definitively determine a diagnosis of TN.

Medical management

Regardless of the classification system used to categorize TN, the first-line treatment is always medical therapy. Of the drugs currently used to treat TN, all of them were originally developed for other indications, and most of them were anticonvulsant drugs. Additionally, only a handful of these drugs have been investigated in small randomized controlled trials for the treatment of TN and many of these trials have methodological flaws and were outdated.

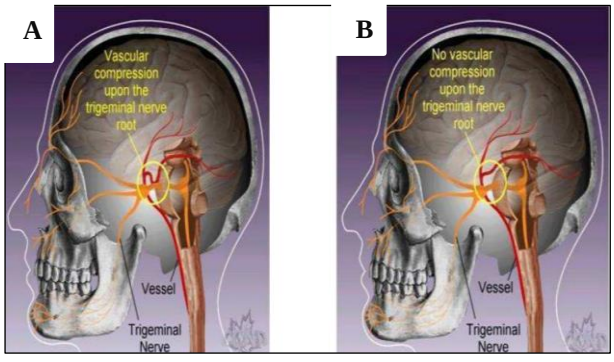

Figure 4: Picture showing trigeminal nerve compression in TN. (A): Patient with vascular compression around the trigeminal nerve root, (B) Patient without vascular compression around the trigeminal nerve root.

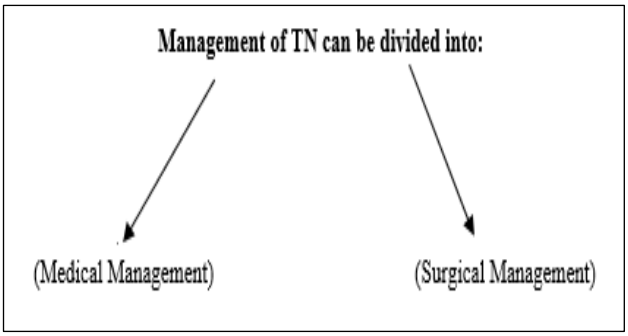

Figure 5: Management of TN.

Medical management

Regardless of the classification system used to categorize TN, the first-line treatment is always medical therapy. Of the drugs currently used to treat TN, all of them were originally developed for other indications, and most of them were anticonvulsant drugs. Additionally, only a handful of these drugs have been investigated in small randomized controlled trials for the treatment of TN and many of these trials have methodological flaws and were outdated. Out of all drugs carbamazepine is the drug of choice and is the most studied medication for the treatment of TN. Other drugs includes oxcarbazepine, lamotrigine, gabapentin, pregablin, topiramate, phenytoin, baclofen, tizanidine, pimozide, sumatriptan, tocainide and proparacaine. However conventional analgesics such as codeine, nonsteroidal anti-inflammatory drugs are not widely used in trigeminal neuralgia.[16]

Surgical management

Presently, surgical treatment options for TN are generally explored only when patients are refractory to medical management. A patient is said to be refractory when he/she cannot bear the adverse effects of the medication and experience breakthrough pain or cannot take the medications because patients are medically unfit receiving polypharmacy for multiple ailments. However, there is current evidence from cohort data implying that earlier surgical treatment may increase patient satisfaction and improve long-term outcomes.[17]

Unfortunately, there are only few randomized controlled trials that compare the outcomes of the numerous surgical procedures for TN, thus complicating the decision regarding which procedure to opt. The surgical procedures are commonly divided into two main categories namely ablative procedures, which destroy the trigeminal nerve and nonabrasive procedures, which preserve the nerve function. Ablative procedure includes peripheral neurectomy, cryotherapy, alcohol block, glycerol injections, percutaneous balloon compression, glycerol rhizotomy, radiofrequency thermocoagulation and stereotactic radiosurgery while nonablative techniques includes microvascular decompression and Botulinum toxin type-A injection.[18]

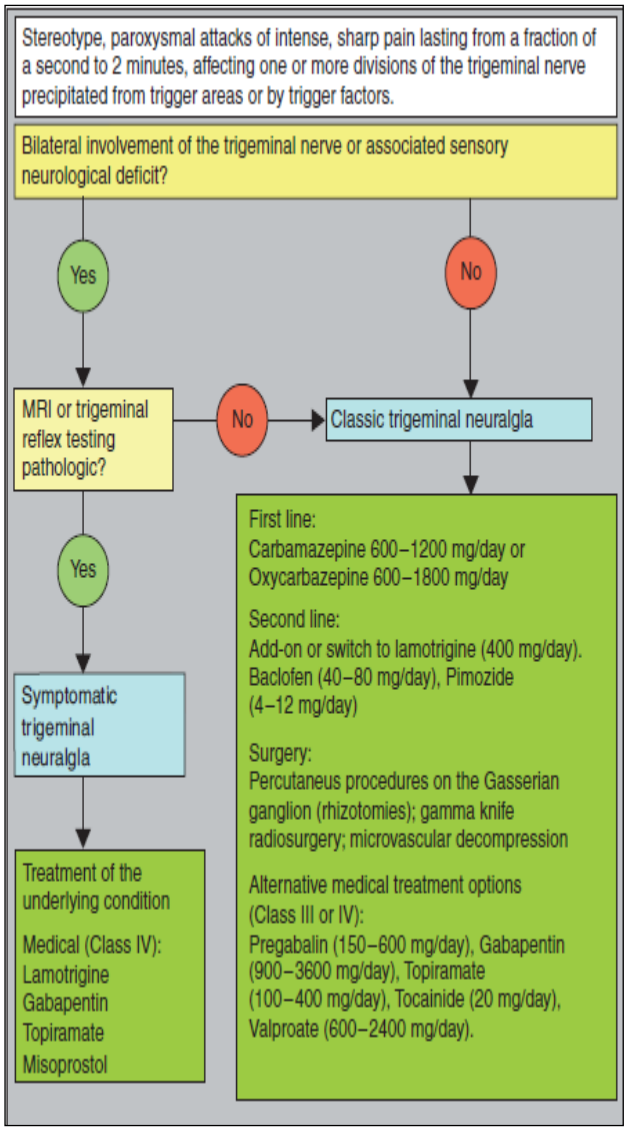

Figure 6: Showing algorithm for management of TN.

Algorithm for Management of TN[19]

Drug Therapy of TN

Carbamazepine

It is an iminostilbene derivative, chemically related to the tricyclic antidepressants. The effect of carbamazepine on pain suppression is probably mediated via central and peripheral mechanisms. It is a sodium channel blocker and act by promoting the inactivated state of voltage activated sodium ion channels.[20] Carbamazepine is the drug of choice and most studied medication for treatment of TN.

Pregabalin (PGB)

It is an anticonvulsant drug used for treatment of neuropathic pain and as an adjunct therapy for partial seizures with or without secondary generalization in adults. Pregabalin is available in 25, 50, 75, 100, 150, 200,

225, and 300 mg capsules. The recommended dose is titrated according to response and tolerability ranging from 150 mg/day to 300 mg/day after 3 to 7 days and then to 600 mg per day after an additional 7 days. It is widely used to treat all kinds of neuropathic pain. It is a GABA analogue structurally related to gabapentin, thus modifing the synaptic or non-synaptic release of GABA. The drug binds to the alpha 2 delta (α2δ) subunit of the voltage gated calcium channels(VGCC) in the brain and spinal cord causing decreased presynaptic calcium entry leading to decreased synaptic release of glutamate.[21]

Oxcarbazepine

It is primarily anticonvulsant drug and a keto derivative of carbamazepine. It has a similar mechanism of action to carbamazepine but not metabolised by the liver cytochrome enzymes. Therefore, it has fewer side effects and does not interact with other medications as compared to carbamazepine.[22] Oxcarbazepine is started at 150 mg per day and can be increased by 150 mg every 3 days until pain relief is achieved. The maintenance dose ranges from 750 to 1800 mg per day with a maximum dose of 2400 mg per day.

Lamotrigine

It belong to 2nd generation antiepileptic drug. It alters the release of glutamate and aspartate via both sodium and calcium channel modulation. It has been shown to be an effective treatment for both classical and symptomatic TN secondary to multiple sclerosis in small case series but only as an add-on therapy to sub effective therapy in one small randomized control trial.[23] Lamotrigine is started in dose of 25 mg per day and slowly increased by 25 mg per week until pain relief is achieved. The maintenance dose is 100-400 mg per day with a maximum dose of 400 mg per day.

Topiramate

It is a new generation antiepileptic drug acts by inhibiting voltage-gated sodium channels and enhancing GABA levels. It was shown to be more effective than carbamazepine in the treatment of TN in a meta-analysis carried out in China after 2 months of treatment.[24] Topiramate is started in dose of 25 mg per day and increased by 25 mg per week until pain relief is achieved. The maintenance dose is between 50 and 100 mg per day with a maximum daily dose of 400 mg.

Phenytoin

It is a 1st line antiepileptic drug acts by modulating voltage-dependent sodium and calcium channels and was the first prevalent medication used in the treatment of TN. However, because of its side effects profile and the advent of newer medications, it is seldom used for the chronic management of TN. Phenytoin is typically started at 100 mg per day and dose can be increased upto 100 to 200 mg three times a day until pain relief is achieved Fosphenytoin. It a phosphate ester prodrug of phenytoin with better parenteral tolerance. It has also been shown to be an effective treatment for patients presenting with severe pain that is refractory to oral medications

Gabapentin

Gabapentin is a new generation AED. It is a gamma-aminobutyric acid (GABA) analogue, that has been shown to be effective in the treatment of both classical and symptomatic TN in case reports.[25] It is started in dose of 300 mg per day and dose can be increased by 300 mg every 3-7 days until pain relief is achieved. The maintenance dose is commonly 900 mg and the maximum daily dose is 2400 mg.

Baclofen

It is centrally acting muscle-relaxant. It- is a GABA derivative that in both the racemic and L-forms, has been reported to be effective for the treatment of TN, either given as a monotherapy or in combination with other medications.[26] It is started in dose of 5 to 10 mg per day and increased by 5 mg every 3 days until pain relief is achieved. The maintenance dose ranges from 50 to 75 mg daily and is typically divided into 3 or 4 doses per day.

Tizanidine

It is a centrally acting muscle relaxant and a central alpha-2 adrenergic receptor agonist. It was first considered as a viable treatment for TN in a double-blind study comparing it to carbamazepine.[27] The tizanidine patients were individually titrated to pain relief with the maintenance dose ranging from 6 to 18 mg per day.

Pimozide

It is a 1st generation typical antipsychotic drug and a dopamine receptor antagonist. The average daily dose ranged from 4 to 12 mg and all of the patients treated with pimozide were relieved of their neuropathic pain as compared to only 56% of the carbamazepine treated patients.[28]

Sumatriptan

It is drug primarily used to treat migraine and acts as a vascular serotonin 5-HT1B/1D receptor agonist producing cerebral vasoconstriction. Because mechanical compression of the trigeminal root by an artery is thought to play a significant role in the cause of classical TN, it is speculated that sumatriptan should work in a majority of patients by inhibiting vasodilation near the irritated nerve root and thereby preventing neurogenic inflammation.[29]

In 2 randomised controlled trials, (RCT) it was found that 3 mg of sumatriptan injected subcutaneously was effective in treating both idiopathic and medical refractory TN.

Tocainide

It is a class 1b antiarrhythmic drug and a sodium channel blocker. It is a derivative of lidocaine. It was tested in a double blind crossover study with carbamazepine in 12 patients with TN. The daily dose of tocainide was 60 mg per kg and it was found to be as effective as carbamazepine in the treatment of TN.[30]

Proparacaine Hydrochloride (HCL) (0.5%) eye drops

Proparacaine HCL (0.5%) eye drops acts by unknown mechanism but probably it act by inhibiting sodium ion fluxes. There have been numerous publications reporting successful treatment of trigeminal neuralgia with proparacaine-HCL eye drops.

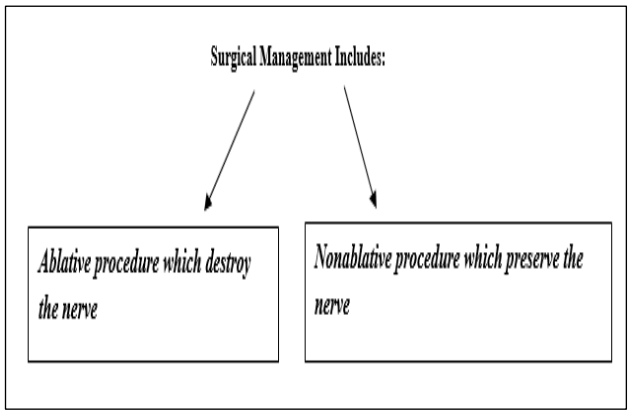

Figure 7: Showing surgical management of TN.

Ablative procedure

Peripheral neurectomy (pn)

It is an oldest surgical technique that dates back to the eighteenth century and is still used today, mainly by maxillofacial surgeons. The concept behind PN is that, by splitting or avulsing the peripheral branch of the trigeminal nerve, the surgeon can achieve precise and long-lasting pain relief. A PN can be performed on the following branches of the trigeminal nerve namely the supraorbital, supratrochlear, intratracheal and the lacrimal nerve branches of the ophthalmic division (V1) or on the infraorbital nerve of the maxillary division (V2) or on the lingual, inferior alveolar, and mental nerves of the mandibular division (V3) The PN is started by first injecting the accessible terminal suspected trigeminal nerve branch with local anaesthetic agent. Overall, PN is a safe procedure without any major complications reported and can be repeated for pain recurrence.[31]

Cryotherapy

Peripheral cryotherapy is a technique that was first explained by Lloyd in 1976.[32] It is a simple and repeatable procedure during which the affected peripheral branch is frozen by direct application of a fine cryoprobe. To begin the procedure, a local injection of an anaesthetic agent is used to determine which nerve to be treated with the cryotherapy. There are numerous variations in the techniques used to perform cryotherapy on the affected nerve. Generally, the affected peripheral nerve is surgically exposed and then frozen with a fine cryoprobe under local anesthesia with or without general anesthesia, with the cryoprobe set at 120º C. This therapy is safe and has shown varying degree of success regarding pain relief.

Alcohol block

It is a destructive technique that requires a compliant and conscious patient because alcohol is directly injected into the nerve. The patient is initially given local anesthesia to determine the correct region or nerve that needs to be injected. Then the patient is injected with 0.5 to 1.5 mL of 80-100% alcohol directly into the affected nerve. The alcohol injections are very painful, so a few drops of local anaesthetic can be injected initially to reduce the burning sensation of alcohol. This procedure is commonly done from an external approach, but some maxillofacial surgeons may use an intraoral approach. The resulting fibrosis from the alcohol block makes it extremely difficult to repeat this procedure.[33] Duration of pain relief following alcohol block varies from less than 2 to 13 months.

Glycerol injection

Glycerol is a very viscous substance and is very difficult to administer and painful for the patient. A glycerol injection is given in the same manner as an alcohol block. Initially, local anaesthetic is used to locate the correct nerve for injection. Then 1.0 to 1.5 mL of sterile glycerol is injected through a large bore needle.[34] It was found that patients who had undergone previous surgery for TN had a mean recurrence of 2 months after glycerol injection as compared to 7.6 months in patients without prior surgery. Glycerol injections provides another option that is minimally invasive with a low incidence of morbidity that could be useful as an alternative surgical treatment of TN.

Radiofrequency thermocoagulation

Percutaneous radiofrequency thermocoagulation (RFT) was first developed in 1913 by Rethi. RFT was used to target the trigeminal rootlets for pain control in TN by Sweet et al, in 1974. This procedure is generally done with induction anesthesia that allows for intraoperative awakening because patient cooperation is critical during the stimulation phase to ensure the correct placement and localization of the RFT lesion. RFT is a destructive procedure with some authors stating that the pain recurrence rate negatively correlates with the degree of sensory loss. The drawback of this procedure is sensory loss. Serious but rare complications of RFT include abscess, cranial nerve palsies, blindness, meningitis and carotid-cavernous fistula.[35]

Nonablative procedure

Microvascular Decompression (MVD)

Jannetta in 1967 first described a trial, where trigeminal nerve root was compressed at the pons by an aberrant loop of artery or vein as a possible source for TN and reported performing the first MVD. Currently, most neurologists and neurosurgeons would agree that microvascular compression is the primary source of most classical TN and based on numerous studies, MVD is an effective treatment. Similar to PSR, MVD is done under general anesthesia and intraoperative monitoring of brain stem auditory evoked responses and somatosensory evoked potentials was done. The trigeminal nerve root was reached and sufficiently exposed through a posterior fossa craniotomy using a microscope. Once the trigeminal root was exposed, the offending vessel was easy located. The most common offending vessel was the superior cerebellar artery. Once the nerve was completely decompressed, the site was closed, and the patient remained hospitalized for observation for at least a few days. The complications of MVD included partial or complete hearing loss, sensory deficits, facial palsies, cerebellar injury, cerebrospinal fluid leak, meningitis and even death. The cause of mortality was due to brain stem infarction. Few patients suffered with ipsilateral hearing loss. MVD has been shown in numerous studies to be the most effective long-term treatment for TN.[36]

Botulinum toxin type A injection

Botulinum toxin type A (BTX-A) has been evaluated in prospective studies and some randomized placebo controlled studies and was found to be effective in the treatment of TN.[37] The mechanism by which BTX-A acts in neuropathic pain has recently been elucidated. It has been shown in TN, there is release of several pain-related neurotransmitters including substance P, calcitonin gene-related peptide and glutamate in brain. BTX-A directly inhibit peripheral sensation and indirectly affect central sensation by inhibiting the release of inflammatory neurotransmitters. The reported complications of BTX-A included transient edema, ptosis, transient dysesthesia, transient trouble with chewing and most commonly mild facial asymmetry.

DISSCUSSION

Trigeminal neuralgia (TN) which is also known as Fothergill disease or Tic douloureux disease is a form of neuropathic pain characterized by the occurrence of abrupt pain which is generally one-sided, severe, brief, sharp and recurrent in the distribution area of one or several branches of the Vth nerve whereas neuropathic pain is defined as "pain arising as a direct consequence of a lesion or disease affecting the somatosensory nervous system. The first-line of treatment is always medical therapy. Of the drugs currently used to treat trigeminal neuralgia, most of them are anticonvulsants. Additionally, only a handful of these drugs have been investigated in small randomized control trials for the treatment of trigeminal neuralgia and many of these trials have methodological flaws and are outdated.[38-40]

CONCLUSION

The treatment of TN is a challenge both for neurologists and neurosurgeons. The lack of a full comprehension of the complex pathogenesis at the basis of TN remains a key factor explaining the results that are not always satisfying with the medical therapy. Progress has been made in the recent years both for the pathogenesis and surgical treatment due to implementation of neuroradiological techniques. With considerable amount of information about trigeminal neuralgia readily available, many patients and their relatives are keen to consider surgery as first line of treatment, in anticipation of premature cure. It is therefore imperative for the doctor to have an understanding of relative efficacy of various treatments including complication and recurrence rates. As long as there is no evidence based medicine to guide treatment choice the doctor will have to combine theory with clinical experience in as balanced a way as possible.

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