



TRIGEMINAL NEURALGIA

Durhasri. A.	Undergraduate, Karpaga Vinayaga Institute of Dental Sciences.
Sathiya Priya. C	Undergraduate, Karpaga Vinayaga Institute of Dental Sciences.
Sowmiya. M	Undergraduate, Karpaga Vinayaga Institute of Dental Sciences.
Dr. Sandhiya. M	Postgraduate, Department of Oral Maxillofacial Pathology, Karpaga Vinayaga Institute Of Dental Sciences
Dr. Arunachalan. M	Reader, Department of Oral Maxillofacial Pathology, Karpaga Vinayaga Institute Of Dental Sciences
Dr. Sathish Kumar. M	HOD, Department of Oral Maxillofacial Pathology, Karpaga Vinayaga Institute Of Dental Sciences

ABSTRACT

Trigeminal neuralgia(TN) is a severe unilateral paroxysmal facial pain that causes intense distress and often compromises the Quality of life of the patient. The aetiology and pathophysiology of TN is still not clear. The pain is Severe, acute and is triggered by cutaneous stimuli. Simple stimuli like breeze on the face to daily activities Such as talking, brushing, chewing food, washing the face Can trigger the pain. The treating neurologist should work in a collaborative fashion with the patient and the neurosurgeon to determine when and what type of surgical procedure to try.

KEYWORDS : Trigeminal Neuralgia, botulinum toxin, microvascular decompression, radiofrequency ablation.

INTRODUCTION

Trigeminal neuralgia (TN) is distinguish by touch-elicited, individual short shock-like paroxysmal pain in One or more divisions of the trigeminal nerve.

Trigeminal neuralgia (TN) is a unilateral disorder characterized by brief electric shock-like pains, sudden in onset and termination, limited to the distribution of one Or more divisions of the trigeminal nerve[1,2]. The pain is Severe, acute and is triggered by cutaneous stimuli. Simple stimuli like breeze on the face to daily activities Such as talking, brushing, chewing food, washing the face Can trigger the pain. Pain is episodic with spontaneous healing. The occurrence of trigeminal Neuralgia in the population is 0.07%, Compared to around 2% in Patients with facial pain in general [3,4,5]. Conversely, trigeminal neuralgia (also Known as facial neuralgia) is frequently Mistaken for dental pain, leading to unnecessary diagnostic procedures such as x-rays of the jaw and, in more than a few cases, unnecessary extractions Of teeth [3,6].

Definition

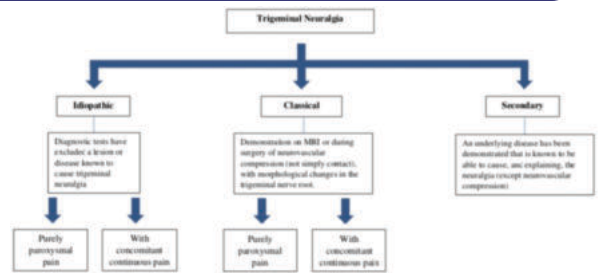
Trigeminal neuralgia (TN) is defined by recurrent unilateral brief electric Shock-like pain that is abrupt in onset and termination. The pain is restricted to one or more of the trigeminal Divisions and is triggered by innocuous sensory stimuli[7].

TN classically presents with “recurrent unilateral brief electric shock like pains, abrupt In onset and termination, limited to the distribution of one or more divisions of the trigeminal nerve and triggered by innocuous stimuli[8,9].

Classification

Trigeminal Neuralgia is divided into classical TN (CTN) and secondary TN (STN).

The International Headache society has divided TN into 3 main categories: classic, secondary/symptomatic, and Idiopathic. Classic TN is associated with neurovascular compression (NVC) of the trigeminal nerve root with associated morphologic changes on MRI. Secondary/ symptomatic trigeminal (STN) neuralgia is associated with an underlying disease. [8,9].



Etiology

Majority of evidence shows the compression of trigeminal nerve root at dorsal root by blood Vessel is the main cause of TGN(10). There are many proofs , first MRI observed in posterior fossa Surgery shows blood vessel contact in T(11)..Second relieving the compression shows pain Relieve in patients(12).Third intra-operative record provides improvement in nerve Status(13).

Fourth, recovering the sensory function. Females are prone to TGN than men(14). It is Seen in older patients. Sometimes, children are also affected. Multiple sclerosis is also associated With TN. TN is the first symptom to see in MS patient. studies shows the Involvement of trigeminal nerve entry in pons is the common features in bot(15).. Tumors like Meningioma is also seen in 2% TN patients(16).

Pathogenesis

The most accepted theory in pathogenecity of TN is demyelination of trigeminal sensory fibers In nerve root(17). The arteries like superior cerebellar artery creates a compression in the root Zone, Sometime veins(17). Due to distribution of fibres ,compress in medial, caudal and cranial Fibres causes V2,V3,V1 symptoms respectively(17). Focal demyelination in vascular indentation is Also seen in histological examination. Arrangement of demyelinated axon in opposite provides Ectopic generation of impulse and ephaptic transmission to adjacent fibres(17). Thus, these Fibres support light, touch, and pain it can explain the paroxysmal pain due to cutaneous Stimuli(17). Sometimes TN is also presented with symptoms like Multiple sclerosis in which the Pathology differs(17).

Clinical Features

The symptoms of TN are quite distinct. There is electric, Lancinating pain on one side of the face, with an abrupt Onset and termination, lasting a few seconds. It can be so Intense that the patient often winces in a tic-like fashion. Cutaneous stimuli may 'trigger' an attack, leaving Some patients unable to chew, drink, shave or brush their Teeth. Some patients will have a brief refractory period Following an attack, during which subsequent cutaneous Stimuli will not trigger an episode.

Potential explanations for this clustering include 'wearing-off' of medications or concentrated periods of facial stimulation Accompanying morning activities. Pain attacks are characteristically absent during sleep. Symptoms often occur in Bouts lasting weeks to months, and initially there are Periods of spontaneous remission. Between attacks, there is Often anxiety regarding the subsequent attack. Other than Subjective unconsciousness, however, there are no other Symptoms in the intervening period [18].

Differential Diagnosis

These Entities can often be distinguished by the quality of pain described; the location of pain (with respect to distributions of trigeminal divisions); stereotypic patterns of pain; the Duration of pain (ranging from brief and paroxysmal to persistent, with attention to Distinguish the summation of multiple overlapping attacks); the frequency of attacks(with Attention to diurnal or seasonal patterns); the onset of symptoms; identified triggers and Associated autonomic Features. Processes to consider, in the appropriate circumstances,Are: dental abnormalities (including dental caries, root abscesses and broken teeth);Temporomandibular joint pain; eye pain (including glaucoma, orbital cellulitis and trauma);Facial trauma and bony fractures; tumour of the facial bones or infiltrating the trigeminal Nerve; giant cell arteritis; Tolosa–Hunt syndrome, trigeminal autonomic cephalgias and Other primary headache syndromes. The pain of herpes zoster tends to be burning in Quality and more constant(although if there are superimposed paroxysms, it may mimic TN). Finally, herpes zoster usually involves the ophthalmic division of the trigeminal nerve Which is uncommonly involved in isolation in typical TN.[19]

Management and Treatment of trigeminal Neuralgia.

Pharmaceutical drugs are used as first line treatment. Surgical and other procedures are done When drugs are contraindicated and pain is unbearable(20).Before starting the procedure liver, Kidney, heart test is done to ensure proper functioning(21)..Most of the TN drugs are Teratogenic in nature(21)..Possibly, ten foods and drug administration are used recently:: Carbamazepine, oxcarbazepine, baclofen, lamotrigine, pimozide, gabapentin ropivacaine, Phenytoin, tizanidine, and botulinum toxin A. In addition, pregabalin, topiramate, levetiracetam, and Vixotrigine, according to various case series and reports, suggest some treatment Response but their use remains off-label(21).

Table 1 Pharmacological management of trigeminal neuralgia (26)

First-line agent Carbamazepine (Tegretol®)†	Start 150 mg daily, increase by 100 mg every 3 days as needed to a total daily dose of 800–1600 mg, divided in 3 doses
Second-line agents Oxcarbazepine (Trileptal®)†	Start 300 mg daily, increase by 300 mg every 3 days as needed to a total daily dose of 1200–1800 mg, divided in 2 doses
Gabapentin (Neurontin®)†	Start 300 mg thrice daily, increase as needed to a total daily dose of 3600 mg, divided in 3 doses. Also commonly used as first-line therapy
Phenytoin (Dilantin®)†	Start 300 mg daily, increase as needed, divided in 2 or 3 doses
Third-line agents (add-on therapy or monotherapy) Lamotrigine (Lamictal®)†	Start 25 mg daily, increase by 25 mg every 7 days as needed to a total daily dose of 200–400 mg, divided in 2 doses
Baclofen (Lioresal®)†	Start 15 mg daily, increase by 5 mg every 3 days as needed to a total daily dose of 60–80 mg, divided in 3 doses

Surgical treatment is done for patients with Pain refractory to a trial at least three Drugs which Include carbamazepine (22). The Medical condition and the age of the patient also Considered Before surgery is advised (23).Side Effects and contraindications of the medication may also Be The reason for considering surgery Studies Measuring quality of life have shown that outcomes Were Best in patients who underwent surgical management And suggested that surgery should Be considered earlier in all patients of TN(24). Recently there is no protocol used to determine The right time for a surgical (25).Various surgical options

Available are ,

1. Peripheral Neurectomy
2. Ablative procedure

Radio frequency ablation

1. Balloon compression
2. Glycerol injection
3. Radio surgery-gamma knife surgery

3.Open procedure

1. Microvascular decompression
2. Trigeminal root section(25).

Prognosis

Remissions and recurrence is characterized by TN. The people have a period of Remission with no pain lasting months or year. TN become more severe and less responsive To treatment overtime, despite increasing pharmacological intervention. Most patients with TN are initially managed medically. Our tertiary referral Centre 50% eventually have a Surgical procedure.[27]

CONCLUSION

Oxcarbazepine and carbamazepine are still the first choice medical treatment, although Many patients experience significant side effects, and those with concomitant continuous Pain respond less. The treating neurologist should work in a collaborative fashion with the patient and the neurosurgeon to determine when and what type of surgical procedure to try[28].

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