Content available at: https://www.ipinnovative.com/open-access-journals

Journal of Oral Medicine, Oral Surgery, Oral Pathology and Oral Radiology

Journal homepage: www.joooo.org

Review Article

Management of trigeminal neuralgia from past to present: A review

Shaikh Amjad[1,*], Mubasshir Ahmed Shaikh[2], Shahnawaz Mulani[3], Revati V Deshmukh[4]

[1]*Dept. of Dentistry, JIIUS, Indian Institute of Medical Sciences and Research, Jalna, Maharashtra, India*
[2]*Dept. of Orthodontics, ACPM Dental College, Dhule, Maharashtra, India*
[3]*Dept. of Prosthodontics, Aditya Dental College, Beed, Maharashtra, India*
[4]*Dept. of Oral Pathology and Microbiology, CSMSS Dental College and Hospital, Aurangabad, Maharashtra, India*

ARTICLE INFO

Article history:
Received 20-02-2022
Accepted 12-07-2022
Available online 17-09-2022

Keywords:
Trigeminal neuralgia
Carbamazepine

ABSTRACT

Orofacial pain in dental clinic are common and diagnosis of trigeminal neualgia required skill and special training here is review article that describes management of trigeminal details in past and present.

1. Historical Aspects

Description regarding of trigeminal neuralgia can be find more than 300 years back. Aretaeus of Cappadocia were the first to describe migraine, he characterised a headache as one that causes "spasms and facial deformities." The phrase tic douloureux was originated by Nicholaus Andre (1756). In a presentation titled "On a Painful Affliction of the Face," which he awarded to the medical society of London in 1773, John Fothergill was the first to provide a detailed and comprehensive description of this illness. The trigeminal neuralgia was extensively covered by Osler in his 1912 book, The principles and practice of medicine.

Cushing (1900) described a method for treating trigeminal neuralgia that involved entire ablation of the gasserian ganglion.

2. Definition of TGN

The most severe and painful condition known to humanity is trigeminal neuralgia, often known as tic douloureux. The discomfort is most commonly felt in the lower face and jaw, although it can also affect the area around the nose and above the eye. The stimulation of the trigeminal nerve, which gives branches to the forehead, face, and lower jaw, causes this acute, shooting, electric shock-like pain. It usually only affects one side of the face. The pain can be triggered by an action like touch, brushing the teeth, wind or by eating. It may begin with mild and short symptoms but if left untreated can progressively become more severe.

Though trigeminal neuralgia cannot always be treated, there are therapies that can help to alleviate the debilitating pain. Anticonvulsive medicines are usually the first line of treatment. Those who have lost their ability to respond to medicine Surgery can be a viable and successful alternative.

3. The Trigeminal Nerve

It is one of the cranial nerves which is mixed type, carrying both sensory and motor components. It is providing the sensation to the face. One trigeminal nerve runs to the right and other to the left side of the head. Each of these nerves has three branches. "Trigeminal" means three derived from the Latin word "tria," and "geminus," means twin. The

* *Corresponding author*.
E-mail address: drshaikhamjad@gmail.com (S. Amjad).

https://doi.org/10.18231/j.jooo.2022.024

trigeminal nerve splits into three branches after it exits the brain and travels through the skull, distribution for sensation throughout the face:

1. Ophthalmic Nerve (V1): The first branch of the ophthalmic nerve regulates sensation in the eye, upper eyelid, and forehead.
2. The second branch, the Maxillary Nerve (V2), controls feeling in the lower eyelid, cheek, nostril, upper lip, and upper gum.
3. Mandibular Nerve (V3): The third branch of the mandibular nerve controls sensations in the jaw, lower lip, lower gum, and some chewing muscles.

4. Diagnosis

Characteristic history and neurologic examination are generally sufficient to diagnose trigeminal neuralgia, hence any laboratory test, radiography or electrophysiologic study are routinely not indicated.

Strict criteria for TN as per the International Headache Society (IHS) are given below: [1]

1. A – Pain attacks lasting a fraction of a second to two minutes, affecting one or more divisions of the trigeminal nerve and meeting criteria B and C.
2. B – At least one of the following traits characterises pain: (1) acute, sharp, superficial, or stabbing; or (2) triggered by trigger elements or trigger locations.
3. C – Not linked to another condition.
4. D – Attacks stereotyped in the individual patient
5. E – No clinically obvious neurologic deficiency.

International Headache Society (IHS) for symptomatic TN differ slightly from the strict criteria and incorporate the following: [1]

1. A – Paroxysmal attacks of pain lasting from a fraction of a second to 2 minutes, with or without persistence of aching between paroxysms, affecting 1 or more divisions of the trigeminal nerve and fulfilling criteria B and C.
2. B – Pain has at least 1 of the following characteristics: (1) intense, sharp, superficial or stabbing; or (2) precipitated from trigger areas or by trigger factors.
3. C – Attacks stereotyped in the individual patient.
4. D – A causative lesion, other than vascular compression, demonstrated by special investigations and/or posterior fossa exploration.

If therapy with carbamazepine is started, complete blood count and liver function tests are required. The serum sodium level should be measured after therapy as carbamazepine can cause hyponatremia.

5. Differential Diagnosis

1. Classic trigeminal neuralgia encompasses neuralgia that is idiopathic or caused by compression of the trigeminal nerve from a neighbouring blood vessel, according to the International Headache Society (IHS). According to studies, around 80% of TN patients are classic. [2]
2. Other anatomical problems, such as tumours and demyelination from multiple sclerosis, might cause secondary trigeminal neuralgia (MS). [3]

The diagnosis of TN begins with a thorough examination of the patient's medical history, as well as the pain's features and pattern of distribution. Patients typically describe sudden, intense face discomfort that lasts a few seconds and occurs several times during the day. These assaults can happen up to a hundred times every day and can last for weeks or months. [4] While the time and triggers of these episodes appear to be unpredictably variable, patients rarely report discomfort that keeps them awake at night.

The diagnosis of TN necessitates a high level of clinical suspicion due to the wide range of concomitant symptoms. It's important to keep track of any patterns associated with the fifth cranial nerve, as well as exacerbating and/or alleviating variables. Sensations, electric shock, burning, and scintillation should all be considered while evaluating the pain quality. Physically, allodynia, hyperalgesia, and triggers are common pain symptoms associated with TN. To assess cranial nerve abnormalities such as hearing loss, sensory alterations, or facial nerve paresis, a complete neurologic examination is required. [5]

Magnetic resonance angiography (MRA) is the imaging technique to demarcate cranial blood vessels; indentations in the blood vessels surrounding the trigeminal root indicate that blood vessels are encroaching upon the nerve. [5]

6. Etiology of TGN

The most likely multifactorial disorder is trigeminal neuralgia (TN).

The majority of trigeminal neuralgia have idiopathic causes, but Pathophysiology discusses how compression of the trigeminal roots by tumour masses or vascular anomalies may result in comparable discomfort. In one investigation, the superior cerebellar artery (81 percent) was shown to be the compressing vessel in 64% of instances, while venous compression was found in 36% of cases. [6]

Trigeminal neuralgia is divided into two categories: classic and symptomatic. The most common type is idiopathic and involves situations where the nerve and a normal artery (superior cerebellar artery or trigeminal artery) are in close proximity.

There are numerous causes of symptomatic variants. Trigeminal neuralgia symptoms may be caused by persistent meningitis, aneurysms, tumours, or other lesions that irritate

the trigeminal nerve roots. Lesions in the pons at the root entrance zone of the trigeminal fiber's, a region of demyelination from multiple sclerosis (uncommon), and an aberrant vascular course of the superior cerebellar artery have all been observed to induce pain that is comparable to trigeminal neuralgia.

Tumor-related causes of trigeminal neuralgia are acoustic neurinoma, chordoma at the level of the clivus, pontine glioma or glioblastoma,[7] metastases, epidermoid and lymphoma. Paraneoplastic etiologies may be the cause of Trigeminal neuralgia.

Vascular causes include arteriovenous malformation, pontine infarct or aneurysm.

Inflammatory causes are multiple sclerosis (common), sarcoidosis, and Lyme disease neuropathy.

Infrequently, adjacent dental fillings composed of dissimilar metals may trigger attacks,[8] and one atypical case followed tongue piercing. A case of trigeminal neuralgia was reported in a patient with spontaneous intracranial hypotension; both conditions resolved following surgical treatment.[9]

7. Pathophysiology

Trigeminal neuralgia (TN pathophysiology)'s is still debatable, therefore either the central or peripheral nervous system, or both, may be to blame. Due to its sensory role, the trigeminal nerve (cranial nerve V) can generate pain. 85 percent of the time, there are no anatomical lesions, although many scientists concur that the aetiology of the idiopathic variant requires vascular compression at the trigeminal nerve entry into the pons. The focal demyelination of the trigeminal nerve is caused by this compression. The cause is initially labelled as idiopathic and then as classic trigeminal neuralgia.

The little unmyelinated and sparsely myelinated primary afferent fibres that are intended for nociception are the typical site of neuropathic pain. The actual pain mechanisms are changed. Action potentials leap from one fibre to the next as a result of microanatomic small and big fibre damage in the nerve, which is essentially demyelination.[10] This damage is frequently seen in the root entry zone (REZ).[11] A factor is the absence of inhibitory inputs from big myelinated nerve fibres. Sensory inputs are amplified by a reentry process. One more central mechanism (eg, delay between stimulation and pain, refractory period). An attack may be started.[12]

8. Investigations

A trigeminal nerve tumour or MS can be found using MRI. Any compression brought on by a blood artery can be seen with a high-resolution three-dimensional MRI. Modern scanning methods can determine the level of compression and whether a vessel is pressing on a nerve.[13] These scans typically do not reveal vein compression. TN is often diagnosed based on the patient's descriptions of their symptoms, the patient's medical history, and a clinical examination.[14] Clinician rely on symptoms and past experiences to diagnose TN because there are no conclusive diagnostic tests. The location, kind, and triggering elements of the pain, including acute, fast, and shock-like pain, are used by Clinician to make their diagnosis. To more accurately locate the pain, physical and neurological exams must be performed.[15]

9. Treatment

9.1. Nonsurgical procedures

There are numerous efficient pain-relieving techniques, including medication. Based on how well a patient responds to the medication, dosages are gradually increased.[16]

The most typical treatment prescribed by Clinician to treat TN is the anticonvulsant drug carbamazepine. Most patients have pain control in the early phases. A Clinician has reason to question whether TN is present when a patient does not experience relief from this medicine. As time goes on, carbamazepine's effectiveness declines. Dizziness, double vision, drowsiness, and nausea are examples of potential adverse effects.[17]

The anticonvulsant medicine gabapentin, which is most frequently prescribed to treat migraines or epilepsy, can also be used to treat TN. Dizziness and/or drowsiness are two minor side effects of this medication that go gone on their own.[18]

The first line of treatment has more recently been oxcarbazepine, a relatively contemporary drug. Because of its identical structure and fewer side effects, it is preferable to carbamazepine. Dizziness and double vision are potential adverse effects.[19]

Other medical includes baclofen, amitriptyline, nortriptyline, pregabalin, phenytoin, valproic acid clonazepam, sodium valproate, lamotrigine, topiramate, phenytoin and opioids.

Apart from their side effects there are certain drawbacks to these medications.[20] Some patients may require higher doses to alleviate the pain, and simultaneously the side effects become more severe at higher doses. Anticonvulsant drugs can lose their effectiveness over time which requires increased in the drug doses leading to adverse drug reactions. In patients with a history of bone marrow suppression, chronic kidney and liver diseases these medications can have more toxic effects. These patients require blood monitoring to make sure their safety.[21]

9.2. Surgery

If medication doesn't work, surgery can be necessary to control the discomfort. There are two methods for performing surgery: 1) Open head surgery 2) lesioning

techniques

Patients who have pressure on the trigeminal nerve caused by a neighbouring blood artery, as identified by a brain MRI, have open surgery. The fundamental issue causing the TN can be corrected with this procedure. Contrarily, lesioning techniques involve actions that might harm the trigeminal nerve.[22] The symptoms of a lesion may not last long and in some cases, they may cause facial numbness.

9.3. Wide operation

The trigeminal nerve root is exposed during the microvascular decompression operation, and the blood vessels that are compressing the trigeminal nerve root are gently moved away from the location of compression. Decompression may lessen signs and enable the trigeminal nerve to return to its regular, pain-free state.[23] Although this operation is the most successful, it is also the most invasive because a craniotomy is necessary to open the skull. Face numbness, facial paralysis, diminished hearing, double vision, stroke, or death are risks connected with this procedure.

9.4. Procedures for lesioning

TN can be treated by percutaneous radiofrequency rhizotomy employing electrocoagulation (heat). By eliminating the affected area of the nerve and reducing the pain signal to the brain, it can reduce nerve pain. The trigeminal nerve is reached by inserting the hollow needle through the cheek. Some of the problematic nerve fibres are destroyed by a heating current that is transmitted through an electrode.[24] Balloon compression done intravenously utilises a needle that is inserted into the trigeminal nerve through the cheek. The neurosurgeon inserts a balloon into the trigeminal nerve using a catheter. The painful nerve fibres are located where the balloon is inflated. After compressing the nerve fibres that cause pain, it is removed.[25]

9.5. Rhizotomy with percutaneous glycerol

Glycerol is administered during this procedure through a needle into the region where the nerve splits into its three major branches. The goal is to selectively harm the nerve in order to prevent the brain from receiving pain signals.[26]

The trigeminal nerve root is the target of stereotactic radiosurgery using the Gamma Knife, Cyberknife, and Linear Accelerator (LINAC) to administer a single dosage of highly concentrated ionising radiation. Since it is noninvasive, open surgery's various dangers and problems are also avoided. Radiation exposure causes a gradual nerve lesion to build over time, which eventually prevents pain signals from reaching the brain.[27]

Surgery or lesioning techniques should always be carefully weighed against their potential hazards. Although a significant portion of TN patients experience pain reduction following this treatment, it is not a foolproof solution for everyone.

9.6. Neuromodulation

Another surgical procedure that can be instituted for TN patients includes placement of one or more electrodes in the soft tissue near the nerves, under the skull on the covering of the brain and sometimes deeper into the brain, to carry electrical stimulation to the part of the brain responsible for sensation of the face. In peripheral nerve stimulation, the leads are kept under the skin on branches of the trigeminal nerve. In motor cortex stimulation (MCS), the area which supplies the face is stimulated.[28] In deep brain stimulation (DBS), parts that innervate the sensation pathways to the face may be stimulated.

Patients should follow-up with their primary treating clinician and health care providers regularly throughout their treatment

Patients undergoing neuromodulation surgery are required to attend the clinic periodically in the year following the procedure.[29] Any changes to the stimulation settings are monitored during these visits, along with the patient's post-operative recovery. Regular follow-up visits with the clinician are necessary to ensure that the treatment is more accurate and effective.[30] Following any kind of neurostimulation surgery, patients will meet with a device representative who will work with their surgeons to modify the device's settings and parameters as necessary.[31]

10. Conclusion

The relative effectiveness of various treatments, including side effects and recurrence rates, must therefore be understood by the physician. Even if the patient responds well to medical therapy, our philosophy is to compassionately consider requests for early surgical treatment.

11. Source of Funding

None.

12. Conflict of Interest

None.

References

1. The International Classification of Headache Disorders: 2nd edition. *Cephalalgia*. 2004;24(Suppl 1):9–160.
2. Cruccu G, Gronseth G, Alksne J, Argoff C, Brainin M, Burchiel K, et al. AAN-EFNS guidelines on trigeminal neuralgia management. *Eur J Neurol*. 2008;15(10):1013–28.
3. Devor M, Wood I, Sharav Y, Zakrzewska JM. Trigeminal neuralgia during sleep. *Pain Pract*. 2008;8(4):263–8.

4. Goldstein LB, Genese F. Trigeminal Neuralgia: A Closer Look at This Enigmatic and Debilitating Disease. *Pract Pain Manag*. 2012;12(8):34–8.
5. Merrill R. Trigeminal Neuralgia. RFE316 Lecture 4. Los Angeles, CA: UCLA School of Dentistry; 2018.
6. Anderson VC, Berryhill PC, Sandquist MA, Ciaverella DP, Nesbit GM, Burchiel KJ. High-resolution three-dimensional magnetic resonance angiography and three-dimensional spoiled gradient-recalled imaging in the evaluation of neurovascular compression in patients with trigeminal neuralgia: a double-blind pilot study. *Neurosurgery*. 2006;58(4):666–73.
7. Hess B, Oberndorfer S, Urbanits S, Lahrmann H, Horvath-Mechtler B, Grisold W. Trigeminal neuralgia in two patients with glioblastoma. *Headache*. 2005;45(9):1267–70.
8. Cheshire WP. The shocking tooth about trigeminal neuralgia. *N Engl J Med*. 2000;342(26):2003.
9. Cheshire WP, Wharen RE. Trigeminal neuralgia in a patient with spontaneous intracranial hypotension. *Headache*. 2009;49(5):770–3.
10. Burchiel KJ. Abnormal impulse generation in focally demyelinated trigeminal roots. *J Neurosurg*. 1980;53(5):674–83.
11. Devor M, Amir R, Rappaport ZH. Pathophysiology of trigeminal neuralgia: the ignition hypothesis. *Clin J Pain*. 2002;18(1):4–13.
12. Cruccu G, Biasiotta A, Galeotti F, Iannetti GD, Truini A, Gronseth G. Diagnostic accuracy of trigeminal reflex testing in trigeminal neuralgia. *Neurology*. 2006;66(1):139–41.
13. Eller JL, Raslan AM, Burchiel KJ. Trigeminal neuralgia: definition and classification. *Neurosurg Focus*. 2005;18:E3.
14. Blom S. Trigeminal neuralgia: its treatment with a new anticonvulsant drug (G-32883). *Lancet*. 1962;1(7234):839–40.
15. Dalessio DJ. Trigeminal neuralgia. A practical approach to treatment. *Drugs*. 1924;24(3):248–55.
16. Campbell FG, Graham JG, Zilkha KJ. Clinical trial of carbazepine (tegretol) in trigeminal neuralgia. *J Neurol Neurosurg Psychiatry*. 1966;29(3):265–7.
17. Rockliff BW, Davis EH. Controlled sequential trials of carbamazepine in trigeminal neuralgia. *Arch Neurol*. 1966;15(2):129–36.
18. Beydoun A. Safety and efficacy of oxcarbazepine: results of randomized, double-blind trials. *Pharmacotherapy*. 1920;8(2):152–8.
19. Sist T, Filadora V, Miner M, Lema M. Gabapentin for idiopathic trigeminal neuralgia: report of two cases. *Neurology*. 1997;48(5):1467.
20. Khan OA. Gabapentin relieves trigeminal neuralgia in multiple sclerosis patients. *Neurology*. 1951;51(2):611–4.
21. Solaro C, Lunardi GL, Capello E, Inglese M, Uccelli MM, Uccelli A, et al. An open-label trial of gabapentin treatment of paroxysmal symptoms in multiple sclerosis patients. *Neurology*. 1998;51(2):609–11.
22. Chogtu B, Bairy KL, Smitha D, Dhar S, Himabindu P. Comparison of the efficacy of carbamazepine, gabapentin and lamotrigine for neuropathic pain in rats. *Indian J Pharmacol*. 2011;43(5):596–8.
23. Carrazana EJ, Schachter SC. Alternative uses of lamotrigine and gabapentin in the treatment of trigeminal neuralgia. *Neurology*. 1998;50(4):1192.
24. Lunardi G, Leandri M, Albano C, Cultrera S, Fracassi M, Rubino V, et al. Clinical effectiveness of lamotrigine and plasma levels in essential and symptomatic trigeminal neuralgia. *Neurology*. 1997;48(6):1714–7.
25. Zakrzewska JM, Chaudhry Z, Nurmikko TJ, Patton DW, Mullens LE. Lamotrigine (lamictal) in refractory trigeminal neuralgia: results from a double-blind placebo controlled crossover trial. *Pain*. 1997;73(2):223–30.
26. Loeser JD. The management of tic douloureux. *Pain*. 1977;3(2):155–62.
27. Braham J. Pain in the face. *Br Med J*. 1968;3(5613):316.
28. He L, Wu B, Zhou M. Non-antiepileptic drugs for trigeminal neuralgia. *Cochrane Database Syst Rev*. 2006;3:CD004029.
29. Baker KA, Taylor JW, Lilly GE. Treatment of trigeminal neuralgia: use of baclofen in combination with carbamazepine. *Clin Pharm*. 1985;4(1):93–6.
30. Fromm GH, Terrence CF, Chattha AS. Baclofen in the treatment of trigeminal neuralgia: double-blind study and long-term follow-up. *Ann Neurol*. 1984;15(3):240–4.
31. Parekh S, Shah K, Kotdawalla H. Baclofen in carbamazepine resistant trigeminal neuralgia - a double-blind clinical trial. *Cephalalgia*. 1989;9(Suppl 10):392–3.

Author biography

Shaikh Amjad, Associate Professor https://orcid.org/0000-0002-0309-081X

Mubasshir Ahmed Shaikh, Associate Professor

Shahnawaz Mulani, Associate Professor

Revati V Deshmukh, Senior Lecturer

Cite this article: Amjad S, Shaikh MA, Mulani S, Deshmukh RV. Management of trigeminal neuralgia from past to present: A review. *J Oral Med, Oral Surg, Oral Pathol, Oral Radiol* 2022;8(3):110-114.