ORIGINAL ARTICLE

Clinicopathological Characteristics of Trigeminal Neuralgia: Insights from a Tertiary Care Center from North India

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Abstract

Introduction: Trigeminal neuralgia (TN) is a severe neurological disorder characterized by intense, electric shock-like facial pain triggered by minimal stimuli. Misdiagnosis often delays effective treatment, significantly impacting quality of life. This study examines TN's diagnosis, prevalence, treatment patterns, and patient impact from a tertiary care perspective with a focus on identifying patterns that may aid in earlier recognition and improved management.

Materials and methods: This retrospective study reviewed the medical records of patients diagnosed with TN per the International Classification of Headache Disorders (ICHD-3 beta) criteria. Data collected included demographics, pain characteristics, treatment details, imaging findings, and previous dental findings.

Results: Sixty patients (mean age: 54 ± 16 years) were analyzed, comprising 31 females (51.7%) and 29 males (48.3%). Unilateral pain was universal, described as electric shock-like in 28 (46.6%), needle prick-like in 18 (30%), and tingling sensation in 7 (11.6%) patients. The V3 branch was affected in 23 (38.3%), and combined V2/V3 in 23 (38.3%). Magnetic resonance imaging (MRI) findings (17 patients) revealed neurovascular compression in 6 cases (35.3%). Medical management, primarily carbamazepine, achieved pain relief in 53 (88.3%), with side effects reported by 7 (11.7%). Before TN diagnosis, 10 patients (16.6%) underwent dental extractions.

Conclusion: TN poses diagnostic challenges that require early identification, MRI use, and a multidisciplinary approach. Enhanced provider education and improved access to diagnostic tools are essential for timely intervention.

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Introduction

The International Association for the Study of Pain (IASP) defines trigeminal neuralgia as a sudden, unilateral, severe, brief, stabbing, recurrent episode of pain in the distribution of one or more branches of the trigeminal nerve. Trigeminal neuralgia (TN) is a rare yet profoundly painful neurological disorder that affects the trigeminal nerve, which supplies sensory input to the face. Often described as one of the most excruciating pain conditions, TN is marked by intense, electric shock-like pains, typically triggered by even light stimuli such as touch, chewing, or speaking.[1–3] These episodes are usually unilateral, last only seconds to minutes, but can be debilitating. In some cases, the pain may persist for hours, leading to significant psychological distress, including anxiety, depression, and even suicidal thoughts.

Misdiagnosis of TN is common, particularly in its early stages, as its symptoms closely resemble those of other conditions, such as dental pain, maxillary sinusitis, temporomandibular joint disorders (TMD), and myofascial pain dysfunction syndrome (MPDS).[4,5] A lack of a multidisciplinary approach in diagnosis can delay appropriate treatment, prolonging suffering and exacerbating psychological distress. Early and accurate diagnosis is crucial for timely and effective intervention.

Treatment for trigeminal neuralgia varies depending on symptom severity and the patient's overall health. Medications, particularly anticonvulsants such as carbamazepine, are the first line of treatment, aiming to stabilize nerve activity and control pain.[6,7] For patients who do not respond adequately to medical management, surgical interventions, such as microvascular decompression or gamma knife radiosurgery, may be employed to address nerve compression or damage.[8–10] While TN itself is not life-threatening, it can severely impact a patient's quality of life, making effective management essential.

This retrospective study evaluates the clinical characteristics, diagnostic approaches, and treatment outcomes of patients diagnosed with TN at a tertiary care center over a 3-year period. By analyzing patient demographics, pain distribution, diagnostic imaging findings, and treatment responses, this study aims to provide valuable insights into the clinical profile of TN and to emphasize the importance of early diagnosis to avoid unnecessary interventions and improve patient outcomes.

Materials and Methods

This retrospective study was conducted at our tertiary care center using a convenience sample of patients treated between January 2021 and December 2023. Patients presenting with sharp, shooting, unilateral, lancinating facial pain localized to specific regions, without any identifiable dental etiology, were diagnosed with trigeminal neuralgia based on the criteria outlined in the International Classification of Headache Disorders (ICHD-3 beta) and included in the study.[11,12]

The study was approved by the Institutional Ethical Committee. Patients of both genders and all age groups with the diagnosis of trigeminal neuralgia were included in the study. Exclusion criteria comprised patients with pain attributable to an odontogenic cause or a history of prior surgical intervention. Data collected from medical records included age, gender, presentation of pain, side of pain, duration of pain, division of the trigeminal nerve involved, type of medication, side effects of medications, radiological findings, specialty consulted, and any dental treatment performed.

Results

The study included 60 patients diagnosed with trigeminal neuralgia. The age of patients with trigeminal neuralgia ranged from 24 to 81 years, with a mean age of 54 ± 16 years. A slight female predominance was observed,

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Table 1: Demographic details of trigeminal neuralgia patients

Parameter	*Results*
Age range (years)	24–81
Mean age (mean ± SD)	54 ± 16
Gender (*n*, %)	Male: 29 (48.3%), Female: 31 (51.7%)
Pain presentation (*n*, %)	Unilateral: 60 (100%)
Side of pain (*n*, %)	Right: 35 (58%), Left: 25 (42%)
Duration of pain (months)	Mean: 33 ± 54
Nature of pain (*n*, %)	Electric shock-like: 28 (46.6%) Needle prick-like: 18 (30%) Tingling sensation: 7 (11.6%) Heaviness and light-headedness: 5 (8.3%) Tingling and burning sensation: 2 (3.3%)
Pain severity (NRS score 0–10)	Mean: 6 ± 1
Pre-trigeminal symptoms (*n*, %)	14 (23.3%)
History of wrong dental treatment (*n*, %)	10 (16.6%)

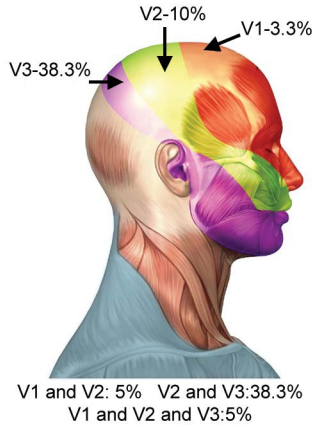

Fig. 1: Distribution of trigeminal neuralgia

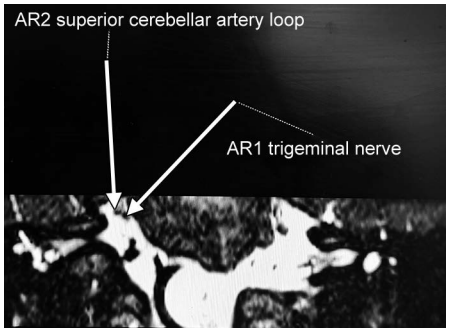

Fig. 2: MRI showing neurovascular conflict between the right anterior inferior cerebellar artery and cisternal segment of the right trigeminal nerve

with 31 (51.7%) of patients being female and 29 (48.3%) males. Pain presentation was unilateral in 100% of cases, with right side involvement in 35 patients (58%) and left side involvement in 25 patients (42%). The pain was described as electric shock in 28 (46.6%) cases, needle prick-like in 18 (30%) cases, tingling sensation in 7 (11.6%) cases, feeling of heaviness and light-headedness in 5 (0.8%) cases, tingling and burning sensation in 2 (0.3%) cases. The mean duration of pain was 33 ± 54 months, and the mean score on the Numeric Rating Scale (NRS) for pain severity was 6 ± 1. Pretrigeminal symptoms, including mild headaches, tingling, heaviness, light-headedness, and burning, were noted in 14 (23.3%) patients. The demographic and characteristic data are detailed in Table 1.

Table 2: Trigger factors for trigeminal neuralgia

Trigger factors	*Frequency (N)*	*Percentage (%)*
Face washing and brushing	5	8.3%
Chewing	2	3.3%
Shaving	1	1.7%
Speaking	1	1.7%
Exposure to cold wind	1	1.7%
Spontaneous	50	83.3%

Pain distribution among the various branches of the trigeminal nerve was as follows: V1: 2 (3.3%), V2: 6 (10%), V3: 23 (38.3%), V1 and V2: 3 (5%), V2 and V3: 23(38.3%), and V1, V2 and V3: 3 (5%). A previous history of inappropriate dental extraction was reported in 10 (16.6%) cases (Fig. 1).

Spontaneous pain occurred in 50 (83.3%) patients. Trigger factors for pain included: chewing—2 (3.3%), speaking—1 (1.7%), exposure to cold wind—1 (1.7%), face washing and brushing—5 (8.3%), and shaving—1 (1.7%). The trigger factors for trigeminal neuralgia are given in Table 2.

During their first visit, 27 (45%) patients consulted the dental department, 7 (11.6%) visited the pain clinic, 3 (5%) the ENT department, 21 (35%) the neurology department, and 1 patient each (1.6%) attended the neurosurgery and general medicine departments. Following these initial consultations, 56 patients were referred to other departments for further evaluation. Of these, 15 (26.7%) were referred to the dental department, 27 (48.2%) to the pain clinic, 9 (16%) to the neurology department, and 5 (8.9%) to the neurosurgery department. After their second visit, 16 patients required additional referrals: 3 (18.7%) to the dental department, 7 (43%) to the pain clinic, 4 (25%) to neurology, and 2 (12.5%) to neurosurgery. The details of various specialties visited by the patients were provided in Table 3.

Magnetic resonance imaging data were available for only 17 patients. Among the 17 who had a magnetic resonance imaging (MRI), 11 showed normal findings, and 6 had evidence of neurovascular compression (Fig. 2).

Medical management was employed in 59 (98.3%) cases, while radiofrequency ablation (RFA) was utilized in one (1.7%). Treatment involved a single medication in 27 (45%) cases and multiple medications in 33 (55%). Among patients prescribed a single drug, 22 were on carbamazepine,2 on oxcarbazepine, and 3 on pregabalin. For combination therapies, carbamazepine and pregabalin were prescribed to 26 patients, oxcarbazepine and pregabalin to 4 patients, and carbamazepine with baclofen to 3 patients. Pain relief was achieved in 53 (88.3%) patients receiving medical management, while 7 (11.7%) did not experience relief. Side effects from medications were reported in 7 (11.7%) cases, including dizziness in 4 patients, a skin rash in 1, and mild itching with dizziness in 2. The management and diagnosis details of trigeminal neuralgia were provided in Table 4.

Discussion

Trigeminal neuralgia is a debilitating craniofacial pain condition characterized by recurrent, sudden-onset, electric shock-like pain along one or more branches of the trigeminal nerve. The condition is not only challenging to diagnose but also significantly impacts patients' quality of life due to its severity and unpredictability.[13] Although its exact etiology remains uncertain, neurovascular compression, demyelination, or both are believed to contribute to the pathophysiology.[14] This study provides valuable insights into the clinical characteristics, diagnostic approaches, and treatment outcomes of TN in a tertiary care setting.

In our study, the mean age of onset for TN was 54 ± 16 years. Katusic et al.[15] reported a median onset age of 67 years, with a range from 24 to 93 years, while Maarbjerg et al. found an average onset age

Table 3: Specialties visited by trigeminal neuralgia patients

Specialty	*1st visit (n, %)*	*2nd visit (n, %)*	*3rd visit (n, %)*
Dental	27 (45%)	15 (26.7%)	3 (18.7%)
Pain clinic	7 (11.6%)	27 (48.2%)	7 (43%)
ENT	3 (5%)	–	–
Neurology	21 (35%)	9 (16%)	4 (25%)
Neurosurgery	1 (1.6%)	5 (8.9%)	2 (12.5%)
General Medicine	1 (1.6%)	–	–
Total patients	**60**	**56**	**16**

Table 4: Diagnosis and management of trigeminal neuralgia

Management modality	*N (%)*
Medical management	59 (98.3%)
Radiofrequency ablation (RFA)	1 (1.7%)
Medication details	*N (%)*
Single drug therapy	27 (45%)
Combination drug therapy	33 (55%)
Pain relief with medication	53 (88.3%)
Side effects with medication	7 (11.7%)
MRI findings	*N (%)*
Performed MRI	17 (28.3%)
Refused or not available	43 (71.7%)
Normal MRI findings	11/17 (64.7%)
Neurovascular compression	6/17 (35.3%)

of 52.9 years.[16] Wettervik et al.[17] highlighted that the incidence of TN increases significantly with age, rising from 0.1 cases per 100,000 person-years in individuals aged 0–19 to 23.1 per 100,000 person-years in those aged 80 and older. Brameli et al.[18] observed that TN is extremely rare in children and adolescents, comprising only 0.5% of cases (5 out of 1,040 patients) in their outpatient department, with a mean age of 15.1 ± 3.0 years among these younger patients. These findings underscore that TN is exceptionally uncommon in pediatric and adolescent populations, and its onset is most frequently observed between the ages of 50 and 60.

Our study noted a slight female predominance, with 51.7% of patients being female (31 out of 60) and 48.3% male (29 out of 60). This contrasts with the findings of Katusic et al., who reported a more significant gender disparity, with 73% of cases occurring in females and only 27% in males.[15] Similarly, Maarbjerg et al. observed that 60% of TN patients were female compared to 40% male.[16] Wettervik et al. also reported a higher incidence of TN in females, with rates of 7.3 per 100,000 person-years compared to 3.7 per 100,000 person-years in males.[17] These findings collectively highlight the tendency for a female predominance in TN, though the degree of disparity varies across studies.

Trigeminal neuralgia is classified into idiopathic, classical, and secondary TN. Idiopathic TN has no identifiable cause or neurovascular compression, with nerve dysfunction suspected. Classical TN is the most common type, caused by vascular compression (usually the superior cerebellar artery), leading to nerve demyelination and hyperexcitability. Secondary TN results from underlying conditions like multiple sclerosis, tumors, or vascular malformations, often presenting with additional neurological deficits. Evaluation of the cerebellopontine angle (CPA) is crucial in patients with TN to rule out secondary causes such as tumors, vascular malformations, or multiple sclerosis. MRI with high-resolution sequences is the gold standard for detecting neurovascular compression or structural abnormalities affecting the trigeminal nerve at the CPA.

In our study, all patients experienced unilateral pain, consistent with findings by Katusic et al.,[15] who reported that only one out of 75 patients presented with bilateral TN, underscoring the rarity of bilateral involvement. Bilateral cases are typically associated with secondary TN, often linked to underlying conditions such as multiple sclerosis or intracranial tumors. Ortega et al. described an uncommon case of bilateral TN in a woman in her 50s, attributed to a bilaterally duplicated superior cerebellar artery, as revealed by MRI.[19] Wettervik et al. classified TN as classical in 19% of cases, secondary in 13%, and idiopathic in 67%.[17] These findings reinforce the understanding that TN predominantly manifests unilaterally, with bilateral occurrences being rare and usually indicative of secondary etiologies.

In our study, the maxillary branch (V2) and the combined maxillary and mandibular branches (V2, V3) of the trigeminal nerve were the most frequently involved. Similarly, Katusic et al.[15] reported that the maxillary division was the most commonly affected (35%), followed by the mandibular division (29%), with 19% of cases involving both. The ophthalmic division (V1) was the least affected, accounting for only 4% of cases, while involvement of all three divisions together was extremely rare (1%). Wettervik et al.[17] reported similar findings, with the ophthalmic branch alone involved in 3% of cases, the maxillary branch alone in 34%, and the mandibular branch alone in 22%. Combinations included ophthalmic and maxillary in 11%, maxillary and mandibular in 25%, and ophthalmic and mandibular in 5% of cases.

In our study, 83.3% of patients experienced spontaneous pain without identifiable trigger factors. Among those with triggers, the most common were washing the face, brushing teeth, and chewing. Di Stefano et al.[20] reported that the most frequent triggers included touching the face (79%), talking (54%), chewing (44%), and brushing teeth (31%). Koh et al.[21] identified weather-related triggers in 20% of patients, with strong winds affecting 8%, cold temperatures 7%, and cold winds 5%. Additionally, 25% of patients had food-related triggers, with 17% reporting hard or tough food, 8% citing hot or cold food, 7% mentioning spicy food, and 3% identifying sweet food as a trigger.

Trigeminal neuralgia must be distinguished from several conditions with overlapping symptoms. Temporomandibular joint disorders (TMD) present with dull, aching jaw pain aggravated by movement, unlike TN's sudden, electric shock-like pain. Glossopharyngeal neuralgia (GPN) causes sharp pain in the throat and ear, triggered by swallowing, differentiating it from TN's facial involvement. Postherpetic neuralgia (PHN) is a persistent burning pain following herpes zoster, whereas TN pain is episodic and trigger-dependent. Myofascial pain dysfunction syndrome (MPDS) is linked to muscle stiffness and stress, lacking TN's paroxysmal nature. Cluster headaches cause unilateral eye pain with autonomic symptoms, whereas TN does not. Atypical facial pain is constant and nonlocalized, unlike TN's well-defined nerve distribution. Sinusitis leads to dull facial pain with congestion, while TN remains sharp and stimulus-induced. Dental pain due to infections or abscesses is persistent and relieved by dental interventions, differing from TN's transient attacks. Proper differentiation through

clinical examination and imaging is crucial to prevent misdiagnosis and unnecessary treatments.[4,22,23]

Trigeminal neuralgia is frequently misdiagnosed in its early stages, as its symptoms often closely resemble those of other conditions, such as dental pain, maxillary sinusitis, TMD, MPDS.[24–26] Misdiagnosis often leads to unnecessary dental procedures, including root canal treatments (RCT) or tooth extractions. In our study, among the patients visited dental department, 16.6% had undergone dental interventions prior to receiving an accurate diagnosis of TN.

Supporting these findings, Von Eckardstein et al.[27] reported that 82% of patients sought dental care after their initial pain episode, yet TN was correctly identified in only 4.7% of cases during the first visit. Of these patients, 31% underwent tooth extractions, and another 31% received RCTs. Similarly, Ayele et al.[28] found that 41% of TN patients had a history of tooth extraction on the affected side prior to diagnosis. Their study also revealed a significant correlation between higher doses of carbamazepine and a history of tooth extraction, with extraction patients requiring an average dose of 736 ± 478.6 mg compared to 661.1 ± 360.4 mg in those without extractions. Agarwal et al.[29] and Tripathi et al.[30] also highlighted the prevalence of unnecessary dental procedures, reporting that more than 80% and 41.8% of TN patients, respectively, had undergone at least one such intervention before being accurately diagnosed. These findings emphasize the critical need for early and precise diagnosis to prevent prolonged pain, unnecessary treatments, and the associated emotional and financial burdens. Increased awareness among healthcare providers, especially dentists, about the diagnostic nuances of TN is essential to mitigate misdiagnosis and improve patient outcomes.

The study revealed that patients with TN are often referred to multiple departments before receiving a definitive diagnosis and treatment. Among the participants, 55 required a second consultation, and 16 underwent a third before an accurate diagnosis was made. To minimize delays and reduce the inconvenience of repeated visits, it is crucial to educate healthcare professionals about TN. Establishing a multidisciplinary pain management team can facilitate early and precise diagnosis, improving patient outcomes and overall care efficiency.

Magnetic resonance imaging plays a crucial role in the assessment and diagnosis of TN. MRI is used to visualize the anatomical structures of the brain, particularly the nerve roots and surrounding vascular structures, to identify potential causes of nerve compression, such as blood vessels pressing against the nerve or tumors. High-resolution MRI techniques, such as 3D T2-weighted imaging and magnetic resonance angiography (MRA), provide detailed views of the neurovascular anatomy, aiding in distinguishing classical TN from secondary causes such as multiple sclerosis or structural lesions.[31] Early and accurate imaging with MRI assists in tailoring appropriate treatment strategies, whether medical or surgical, and in assessing the potential benefits of microvascular decompression surgery. However, in our study, only 28.3% of patients underwent MRI after recommendation. This low uptake could be attributed to financial constraints or a lack of awareness among patients, highlighting the need for better access to diagnostic imaging and education on its importance in managing TN effectively.

Carbamazepine remains the first line of treatment for TN and was prescribed for 55 patients in this study. While effective in alleviating neuralgic pain, carbamazepine is associated with hypersensitivity reactions in approximately 10% of patients, with varying severity and frequency.[32] In our study, adverse effects such as dizziness, itching, and rashes were reported in 11.7% of patients taking carbamazepine. Alternative medications, including oxcarbazepine, pregabalin, and gabapentin, were also used and did not result in any adverse reactions. Notably, 55% of the study population required more than one medication for effective pain management.

Surgical approaches are considered for TN cases refractory to medical management. Microvascular decompression (MVD) is the preferred surgical option for classical TN, relieving nerve compression by repositioning or cushioning the offending vessel. Percutaneous procedures like radiofrequency rhizotomy, balloon compression, or glycerol injection offer pain relief by disrupting pain pathways in the trigeminal nerve. Stereotactic radiosurgery (gamma knife or cyber knife) is a noninvasive alternative, using focused radiation to target the trigeminal root entry zone. The choice of procedure depends on patient factors, severity, and imaging findings.

Trigeminal neuralgia presents significant diagnostic and therapeutic challenges, frequently being misdiagnosed as dental pain, resulting in unnecessary procedures. Prompt diagnosis and increased awareness among healthcare providers, especially dentists, are essential to mitigate these errors. A multidisciplinary approach involving neurologists, dentists, maxillofacial surgeons, and radiologists is vital to creating patient-centered care plans and reducing diagnostic delays. MRI is instrumental in detecting neurovascular compression or secondary causes of TN, and its broader accessibility is critical for accurate diagnosis and effective management. Addressing barriers to advanced diagnostic tools, particularly in rural areas, is imperative to ensure equitable healthcare delivery and improve patient outcomes.

Strengths and Limitations

To the best of the authors' knowledge, this is the first study to describe the demographic and clinical characteristics of and the impact of medical management on patients with trigeminal neuralgia presenting to a tertiary care institute catering mainly to rural and semi-urban populations in North India. This study had some limitations that warrant consideration. The relatively small sample size may limit the generalizability of our findings. Additionally, the study population primarily comprised patients from suburban and rural areas, potentially introducing a socioeconomic bias that could affect healthcare access and treatment choices. Furthermore, MRI use was low among our participants, primarily due to financial constraints and limited awareness, limiting our ability to assess the full spectrum of neurovascular causes of trigeminal neuralgia. Finally, the retrospective nature of the study may have introduced recall and reporting biases. Future research should focus on multicentric prospective studies with large sample sizes to address these limitations and provide more robust insights.

Conclusion

Trigeminal neuralgia remains a debilitating condition with a significant impact on patients' quality of life. Our study highlights the importance of early, accurate diagnosis to prevent unnecessary dental procedures and emphasizes the key role of imaging, especially MRI, in identifying neurovascular and secondary causes. Although carbamazepine remains a cornerstone in medical management, awareness of potential hypersensitivity reactions and the need for genetic screening is essential, especially in diverse populations. Enhanced education for healthcare providers and improved access to diagnostic resources are necessary to optimize care and outcomes for TN patients. Future research with larger, more diverse cohorts and improved access to comprehensive diagnostic and genetic testing is warranted to strengthen the understanding of this complex disorder.

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