REVIEW

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Understanding Trigeminal Neuralgia: A Comprehensive Review of Symtoms, Diagnosis, and Management

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ABSTRACT

Trigeminal neuralgia (tic douloureux) is a rare and debilitating facial pain condition affecting one or more branches of the trigeminal nerve. It is frequently misdiagnosed as a dental issue or temporomandibular disorder due to overlapping symptoms, delaying appropriate treatment. Diagnosis is primarily clinical, often requiring the expertise of a neurologist to distinguish it from other causes of facial pain. Imaging studies such as MRI or CT scans are not diagnostic but play a crucial role in ruling out secondary causes and identifying pathological changes in the affected nerve root. Although not life-threatening, trigeminal neuralgia significantly impacts the quality of life, causing severe, recurrent pain episodes that can be challenging to manage. Timely and accurate diagnosis, along with the implementation of effective therapeutic strategies, is essential to alleviate symptoms and improve patient outcomes. This review highlights the clinical presentation, diagnostic approach, and current treatment modalities for trigeminal neuralgia, emphasizing the importance of a multidisciplinary approach to care.

Keywords: Cranial nerve, facial pain, trigeminal neuralgia, trigeminal nerve.
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INTRODUCTION

Trigeminal neuralgia (TN) is a severe orofacial pain disorder characterized by sudden, paroxysmal episodes of sharp, stabbing pain along one or more branches of the trigeminal nerve. Despite its historical recognition dating back to the seventeenth century, TN remains one of the most challenging conditions to diagnose and treat.[1,2] It significantly impacts quality of life, leading to severe pain, disability, and substantial healthcare burdens. Misdiagnosis is common, with TN frequently mistaken for dental conditions, migraines, or temporomandibular disorders, resulting in unnecessary and invasive dental procedures.[3] The lack of specific biomarkers or diagnostic tests compounds these challenges, leaving clinicians reliant on patient-reported symptoms and clinical evaluations. Imaging, such as MRI, can aid in identifying neurovascular compression or ruling out secondary causes but is not definitive for diagnosis.[1,2,4]

Understanding and managing TN requires a multidisciplinary approach tailored to the individual. The pathophysiology involves complex interactions of neurovascular, genetic, and anatomical factors, with neurovascular compression and focal demyelination often playing critical roles. First-line treatment typically involves anticonvulsant medications like carbamazepine or oxcarbazepine, while surgical options, such as microvascular decompression, are considered for refractory cases. Advances in imaging and pain research have revealed structural and functional brain abnormalities, offering new insights into TN's mechanisms. This review focuses on the clinical features, diagnostic challenges, and treatment options for TN, emphasizing the importance of distinguishing it from other orofacial pain disorders to ensure timely and effective care.[1–4]

ANATOMY OF TRIGEMINAL NERVE

The trigeminal nerve (TGN), or fifth cranial nerve, plays a crucial role in the development of TN, a condition characterized by severe, paroxysmal facial pain. Emerging from the lateral mid-pons, the TGN consists of sensory and motor components that branch into three divisions: the ophthalmic (V1), maxillary (V2), and mandibular (V3) nerves.[5] While the motor root controls muscles involved in mastication and other facial movements, the sensory root innervates critical areas of the face, including the eyelids, sinuses, teeth, and skin. The sensory branches of the TGN are particularly relevant in TN, where misfiring of these pathways leads to the intense pain characteristic of the condition.[5,6] The somatotopic organization of the nerve, where specific fibers correspond to particular facial areas, plays a key role in the distribution of pain in TN, often correlating with the site of neurovascular compression. This compression, commonly at the trigeminal root entry zone (TREZ), causes irritation and demyelination of the nerve, leading to the hyperexcitability of nerve fibers. Anatomical variations in the trigeminal ganglion and nerve branches, including

their diameter, branching patterns, and susceptibility to compression, can influence the presentation and severity of TGN symptoms. The trigeminal ganglion's role in neuropeptide regulation, particularly calcitonin gene-related peptide (CGRP), is central to the pain mechanisms in TGN, as these peptides facilitate vasodilation and contribute to pain propagation.[5–7]

DEFINITION

Trigeminal neuralgia, historically known as "tic douloureux de la face," was first described in the mid-eighteenth century and is characterized by sudden, sharp, electric shock-like pain in the face. It has an incidence of about 5 cases per 100,000 people and is defined by the International Headache Society as a disorder marked by recurrent, unilateral, brief, electric shock-like pains, triggered by innocuous stimuli, and limited to the distribution of one or more branches of the trigeminal nerve. Trigeminal neuralgia may either arise without an obvious cause, termed "idiopathic," or result from an underlying pathology, which is referred to as "secondary" neuralgia. When the condition is caused by vascular compression of the trigeminal root, it is classified as "classical" or primary trigeminal neuralgia. The term "tic douloureux" was coined in 1756 by Nicholas Andre, while John Fothergill in 1773 provided a detailed report, calling it "Fothergill's disease," and described its characteristic paroxysmal pain triggered by activities like talking, eating, or light touch. In 1853, Trousseau referred to it as "epileptiform neuralgia." The condition has evolved in understanding, with the International Classification of Headache Disorders (ICHD-3Beta) defining trigeminal neuralgia as sudden, brief, electric shock-like pain, often unilateral and triggered by non-harmful stimuli, emphasizing the condition's consistent presentation across centuries.[8,9]

ETIOLOGY

The etiology of trigeminal neuralgia is classified into idiopathic and symptomatic forms. Idiopathic trigeminal neuralgia, where the cause remains unclear, is most commonly attributed to vascular compression from blood vessels near the trigeminal nerve root. In 80-90% of idiopathic cases, compression is caused by adjacent arteries or veins, with the superior cerebellar artery being the main contributor in 75-80% of cases. Continuous compression leads to morphological changes, including damage to the myelin sheath and demyelination, which increases the nerve's sensitivity to stimuli, causing intense, paroxysmal pain. Symptomatic trigeminal neuralgia occurs when an underlying condition compresses or irritates the trigeminal nerve. Conditions such as multiple sclerosis, where demyelination of the trigeminal nerve nucleus leads to neuralgia, as well as tumors like meningiomas or acoustic neuromas, and vascular malformations, can all contribute to nerve compression. Additional factors such as space-occupying lesions, skull abnormalities, and trauma, particularly fractures in the zygomaticomaxillary region, can also cause compression. Multiple sclerosis is a known risk factor for symptomatic trigeminal neuralgia, contributing to 2-4% of cases, while degenerative conditions like atherosclerosis and hypertension may also play a role in its development by affecting vascular changes that impact the nerve.[7,10,11]

EPIDEMIOLOGY

Trigeminal neuralgia (TN) is a chronic pain condition affecting the fifth cranial nerve, leading to sudden, severe facial pain. Globally, the prevalence of TN ranges from 0.01% to 0.3%, with women being more frequently affected than men, exhibiting a prevalence ratio between 1.0–1.7:1. The condition predominantly occurs in individuals over 40 years of age, with approximately 90% of cases in this age group, and is rare in children. In younger individuals, TN is often associated with multiple sclerosis. The prevalence of multiple sclerosis in the general population is estimated to be between 1% and 6.3%. Regarding the affected side, the right side is more commonly involved, accounting for about 60% of cases, while the left side is affected in 39%, and bilateral involvement occurs in 1%. In terms of the specific branches of the trigeminal nerve affected, the maxillary (V2) and mandibular (V3) branches are most commonly involved, accounting for approximately 42% of cases, followed by V2 alone (20%), V3 alone (17%), V1 and V2 (14%), all three branches (5%), and V1 alone (2%). Bilateral TN is often associated with a family history of the condition.[4,7,8]

In Indonesia, a study conducted in 2022 at the Indonesian National Brain Center Hospital examined 100 patients with TN. The study found that only one diagnosis was made by a general practitioner, and none of the patients were immediately referred to a neurosurgeon following their diagnosis. The average duration from onset to referral was 4.7 years. Additionally, 25.5% of patients experienced drug-related side effects due to prolonged medication. Only 50% of patients were covered by the universal health coverage system, with some patients incurring significant out-of-pocket expenses for treatment. The study highlighted the need for early referral to neurosurgeons to improve management and outcomes for TN patients in Indonesia.[12]

CLASSIFICATION

Trigeminal neuralgia (TN) is a disorder characterized by recurrent, sudden, and severe unilateral facial pain, often described as electric shock-like sensations, limited to the distribution of one or more divisions of the trigeminal nerve. The International Classification of Headache Disorders, Third Edition (ICHD-3), classifies TN into several categories:[7,13]

- Classical Trigeminal Neuralgia: This form may present as purely paroxysmal or with concomitant continuous pain. It is often attributed to vascular compression of the trigeminal nerve or its root.
- Secondary Trigeminal Neuralgia: This type is associated with underlying conditions such as multiple sclerosis, space-occupying lesions, or other causes.
- Idiopathic Trigeminal Neuralgia: In this form, no specific cause is identified. It may present as purely paroxysmal or with concomitant continuous pain.

Additionally, the ICHD-3 includes classifications for painful trigeminal neuropathy, which refers to facial pain in the distribution of one or more branches

of the trigeminal nerve caused by another disorder and indicative of neural damage.[13]

PATHOPHYSIOLOGY

Trigeminal neuralgia (TN) is primarily caused by the compression of the trigeminal nerve, leading to abnormal firing of nerve fibers and hyperexcitability of the nerve root. The most common cause of TN is vascular compression, where blood vessels, typically the superior cerebellar artery, press on the trigeminal nerve near its root entry zone (TREZ). This compression leads to demyelination of the nerve fibers, which disrupts normal conduction and triggers the pain characteristic of TN, often in response to innocuous stimuli. In cases of secondary TN, such as those associated with multiple sclerosis, the demyelination occurs within the central nervous system, particularly affecting the trigeminal nerve nucleus, leading to similar hyperexcitability. The pain experienced in TN is typically paroxysmal and electric shock-like, confined to the area innervated by one or more divisions of the trigeminal nerve. Additionally, structural changes such as vascular malformations, tumors, and other space-occupying lesions can also lead to TN by compressing the trigeminal nerve. These pathophysiological processes result in altered sensory processing, central sensitization, and an exaggerated pain response, contributing to the recurrent and severe episodes of pain associated with TN. Recent studies have highlighted the importance of neuroplastic changes and altered pain modulation mechanisms in the central nervous system in the development and persistence of TN symptoms.[1,10,14]

DIAGNOSIS

Trigeminal neuralgia (TN) is primarily diagnosed through clinical evaluation, although additional diagnostic steps are necessary when the etiology is unclear. The following diagnostic guidelines are based on international standards, including the *International Classification of Headache Disorders, 3rd edition* (ICHD-3) by the International Headache Society:[8,10,11,13,15–19]

Clinical History and Symptom Assessment

Pain Characteristics

TN is marked by sudden, severe, stabbing (electric shock-like) pain, occurring in the distribution of the trigeminal nerve (V1, V2, or V3 branches). The pain is typically unilateral, brief (lasting seconds to minutes), and recurrent, often triggered by light touch, speaking, chewing, or exposure to wind.[13]

Trigger Zones

Identify areas of skin or mucosa that provoke attacks upon minimal touch, often found around the nose, mouth, or jaw. Trigger zones are significant clinical indicators of TN.[13,15]

Attack Frequency and Pattern

Pain typically occurs episodically with periods of remission, although some cases may involve persistent pain.[7,13,15]

Neurological Examination

A focused neurological examination is essential to check for other neurological signs, especially in atypical cases (e.g., bilateral symptoms, young onset). Assessment of reflexes, motor function, and sensory examination of the trigeminal nerve branches is necessary.[7]

Imaging

MRI with Contrast

According to the American Academy of Neurology (AAN) and the European Federation of Neurological Societies (EFNS) guidelines, MRI is not routinely required for diagnosing typical trigeminal neuralgia (TN). In cases where the symptoms are characteristic—such as unilateral, sharp, and electric shock-like pain triggered by innocuous stimuli—diagnosis can generally be made clinically without imaging. However, MRI should be used when atypical features are present, such as bilateral symptoms, onset under 40 years of age, or neurological abnormalities, as well as when secondary causes like multiple sclerosis or space-occupying lesions are suspected. Contrast-enhanced MRI is preferred in these cases to assess for vascular compression or structural abnormalities. In the absence of these atypical features, routine imaging is not necessary. CT scans are rarely indicated, but may be considered if MRI is unavailable or in cases with suspected trauma or structural abnormalities, especially in older patients.[14,15,18,19]

CT-Scan

CT scans are rarely indicated in the diagnosis of trigeminal neuralgia (TN) and are typically used when MRI is unavailable or when there is suspicion of structural abnormalities such as trauma. While MRI is the preferred imaging modality for assessing nerve root compression or secondary causes like multiple sclerosis, CT may be used in emergency situations, such as when there is concern about trauma leading to fractures in the zygomaticomaxillary region or other craniofacial fractures. However, CT scans are not as effective as MRI in evaluating soft tissue structures like the trigeminal nerve root and vascular compression. Therefore, while CT may help rule out structural causes like tumors or fractures, it is not useful for directly assessing the vascular compression or demyelination of the trigeminal nerve, which are key to diagnosing TN. As such, CT is typically considered a secondary option when MRI is contraindicated or unavailable.[14,15,18,19]

Electrophysiological Testing

Electrophysiological tests, such as somatosensory evoked potentials (SSEPs) or trigeminal reflex testing, may be used in suspected cases of multiple sclerosis or other demyelinating conditions to assess nerve function.[13]

DIFFERENTIAL DIAGNOSIS

Differential diagnosis of trigeminal neuralgia (TN) is essential to distinguish it from other conditions that present with facial pain, as the management strategies differ significantly. Common conditions that mimic TN include neuralgias, neuropathies, and headache disorders with overlapping clinical features, as summarized in the **Table 2**.

TREATMENT

The management of trigeminal neuralgia (TN) aims to reduce pain and improve the patient's quality of life, and treatment

Table 1. Diagnostic criteria for TN based on the International Classification of Headache Disorders (ICHD-3)[13]

Criteria	Typical Trigeminal Neuralgia	Atypical Trigeminal Neuralgia
Pain characteritics	Sudden, severe, stabbing (electric shock-like) pain	Pain with unusual characteristics such as constant pain or more diffuse pain
Pain location	Unilateral, in the distribution of one or more branches of the trigeminal nerve (V1, V2, V3)	Usually unilateral but can also be bilateral in rare cases
Pain duration	Brief (seconds to minutes), recurrent, with remissions	May have more persistent pain or a different duration
Triggering factors	Triggered by innocuous stimuli (light touch, chewing, speaking, wind, etc.)	Pain may be triggered by other factors or not triggerable
Episodes of pain	At least 3 episodes of electric shock-like pain	Episodes may be atypical or involve prolonged or continuous pain
Age of onset	Typically occurs after the age of 40	Atypical TN may occur in younger patients, often with MS (Multiple Sclerosis)
Associated features	No associated neurological signs (unless secondary TN)	May have associated features such as bilateral symptoms, neurological deficits, or evidence of demyelination
Diagnosis confirmation	Based on the above criteria and exclusion of secondary causes	Requires further diagnostic workup to rule out secondary causes, such as MRI to assess demyelination or structural abnormalities
Imaging	MRI (preferably) for ruling out secondary causes	MRI is necessary to assess for multiple sclerosis, tumors, or vascular compression

Table 2. The differential diagnosis for TN.[13,20–22]

Condition	Key Features	Differentiating Factors from TN
Glossopharyngeal Neuralgia	Sudden, sharp, electric shock-like pain in the distribution of the glossopharyngeal nerve (throat, tongue, ear).	Pain occurs in the throat, base of tongue, or ear, triggered by swallowing or speaking, not facial triggers.
Trigeminal Neuropathy Post-Trauma	Persistent or recurrent facial pain following trauma or surgery involving the trigeminal nerve.	Associated with a history of trauma, numbness, or sensory changes in the affected area.
Persistent Idiopathic Facial Pain (PIFP)	Constant, dull, or aching facial pain, not following the distribution of a specific nerve.	Pain is not paroxysmal or electric shock-like and does not follow the trigeminal nerve branches.
Trigeminal Neuropathy Caused by Acute Herpes Zoster	Pain and burning in the trigeminal distribution, often with a rash or vesicles in the affected area.	Presence of vesicular rash and postherpetic neuralgia symptoms in the same region.
Short-lasting Unilateral Neuralgiform Headache with Autonomic Symptoms (SUNA)	Sharp, stabbing pain lasting seconds to minutes with autonomic symptoms (e.g., lacrimation, rhinorrhea).	Similar to TN, but always includes autonomic symptoms like nasal congestion, tearing, or rhinorrhea.
Short-lasting Unilateral Neuralgiform Headache with Conjunctival Injection and Tearing (SUNCT)	Unilateral pain with conjunctival injection and tearing, episodes lasting seconds to minutes.	Prominent conjunctival injection and tearing distinguish SUNCT from TN.
Cluster Headache	Severe unilateral headache with autonomic symptoms (lacrimation, ptosis, nasal congestion), lasting 15-180 minutes.	Pain lasts longer than TN episodes and is associated with a "cluster" of attacks over days to weeks.

strategies are guided by the nature of TN (classical, secondary, or idiopathic). The most recent guidelines emphasize a multimodal approach, including pharmacological therapy, interventional techniques, and surgical options, depending on disease severity and response to initial treatments.[7,13,23–25]

Pharmacological Therapy (First-Line Treatment)

Carbamazepine and oxcarbazepine remain the first-line drugs for classical TN, supported by guidelines from the European Academy of Neurology (EAN) and American Academy of Neurology (AAN). Both have demonstrated robust efficacy in controlling paroxysmal pain episodes. Alternative anticonvulsants like gabapentin, pregabalin, or lamotrigine can be considered in cases of drug intolerance or partial response. Baclofen may be added as adjunctive therapy in refractory cases.[23–25]

Interventional Procedures (Second-Line Treatment)

For patients who do not respond to or cannot tolerate medications, minimally invasive techniques are recommended: [23–25]

- Microvascular Decompression (MVD): The most effective surgical option for classical TN caused by neurovascular compression. It aims to relieve pressure on the trigeminal nerve.
- Radiofrequency Thermocoagulation (RFT): Used for patients with medically refractory TN, particularly in older patients or those unsuitable for open surgery.
- Stereotactic Radiosurgery (Gamma Knife Surgery): Non-invasive and effective for pain relief in TN, with reduced recovery time.

Treatment for Secondary TN

Addressing the underlying cause is crucial (e.g., multiple sclerosis or tumor resection in cases of space-occupying lesions). Pharmacological therapy may be less effective, and surgical or interventional approaches are often required. [23–25]

Emerging Therapies and Personalized Approaches

Recent studies highlight botulinum toxin injections as an adjunct or alternative in refractory TN cases. Neuromodulation techniques, such as percutaneous electrical nerve stimulation (PENS) or trigeminal nerve stimulation (TNS), are being explored. Precision medicine approaches, including genetic profiling, may guide future TN treatment based on individual patient characteristics. [23–25]

PROGNOSIS

The prognosis for trigeminal neuralgia (TN) varies based on the underlying cause, timely diagnosis, and treatment approach. Idiopathic or classical TN typically responds well to first-line medications like carbamazepine or oxcarbazepine, though the effectiveness may decline over time due to tolerance or side effects. For refractory cases, surgical interventions such as microvascular decompression (MVD) have high success rates, providing long-term relief for 80%-90% of patients, with recurrence rates of 10%-30% within 5-10 years. Secondary TN, caused by conditions like multiple sclerosis (MS) or tumors, depends largely on managing the underlying condition. MS-related TN can be more resistant to standard treatments, often requiring a multidisciplinary approach, while space-occupying lesions may improve with surgical management. Emerging therapies, such as stereotactic radiosurgery and neurostimulation, offer potential for refractory cases, though their long-term outcomes are still under study. Prognosis is influenced by factors like age, duration of symptoms, and comorbidities. Early diagnosis and appropriate treatment significantly improve the quality of life, but TN remains a chronic condition requiring ongoing management and monitoring.[7,13,22]

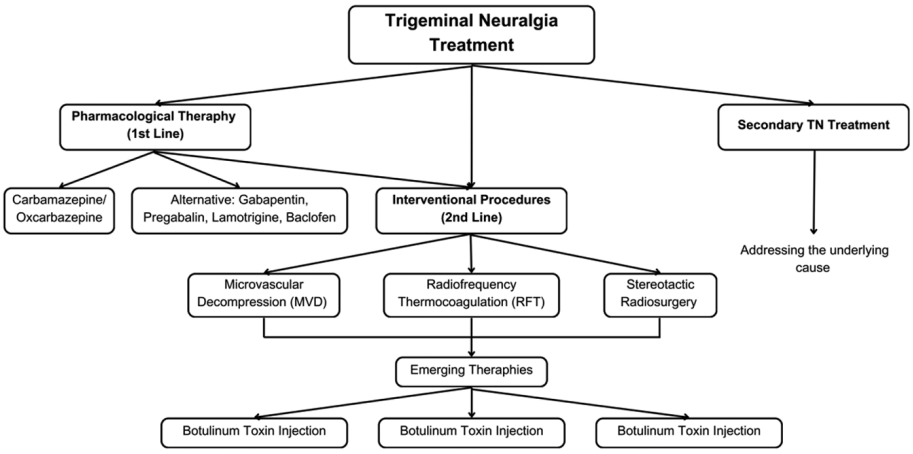

Figure 1. Flowchart treatment options for TN.

CONCLUSION

Trigeminal neuralgia (TN) is a severe, recurrent facial pain disorder that significantly affects quality of life. Its etiology is classified as idiopathic, often linked to vascular compression, or secondary to conditions like multiple sclerosis or tumors. TN primarily affects individuals over 40 years old, with a higher prevalence in women, and is commonly unilateral. Diagnosis is based on clinical evaluation, supported by imaging such as MRI or CT to exclude secondary causes, while differential diagnoses include glossopharyngeal neuralgia and idiopathic facial pain. Treatment begins with pharmacological options like carbamazepine and oxcarbazepine, while refractory cases may require surgical interventions such as microvascular decompression or stereotactic radiosurgery. Emerging therapies, including neurostimulation, offer additional options for complex cases. Early diagnosis and evidence-based management are crucial for improving outcomes, while ongoing research is vital to developing novel therapeutic strategies.

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Conflict of Interest

The authors declare no conflicts of interest related to this study, including any financial or personal relationships that could influence the objectivity of the study, interpretation of the results, or manuscript writing.

Author Contribution

Andrea Valentino responsible for the concept, design, and approved the final version for submission to this journal. Farah Mardhiyah contributed to data analysis, manuscript preparation, execution of the study.

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