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Trigeminal Neuralgia - Impact on Daily Activities and Sports, Treatment Options That Enable a Pain-Free Life

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Summary

OBJECTIVE

This review aims to examine the multifactorial nature of Trigeminal Neuralgia (TN), a neuropathic disorder characterized by intense, unilateral facial pain, and to evaluate the epidemiology, pathomechanisms, classification, clinical presentation, diagnostic protocols, and current treatment options for TN. Additionally, the review emphasizes the need for comprehensive treatment strategies that address both physiological and psychological aspects to improve patient outcomes.

MATERIALS AND METHODS

A systematic literature search was conducted across PubMed and Scopus databases, focusing on publications related to "trigeminal neuralgia," "TN pathophysiology," "TN treatments," and "TN diagnosis." Inclusion criteria required studies that discussed TN's etiology, clinical characteristics, and therapeutic approaches, resulting in a selection of articles that cover both pharmacological and surgical treatments, as well as recent advances in TN management.

RESULTS

The review highlights that TN has a prevalence of 0.16%–0.3%, predominantly affecting women, with an increased incidence among individuals over 50 years of age. TN etiology includes neurovascular compression, demyelinating conditions like multiple sclerosis, and familial genetic predispositions. Classification systems, such as the 2018 ICHD-3, provide diagnostic criteria and categorize TN into classical, secondary, and idiopathic types. First-line treatments involve antiepileptic medications such as carbamazepine and oxcarbazepine, with second-line options including lamotrigine and baclofen. For refractory cases, microvascular decompression and percutaneous techniques are recommended, with options like gamma knife radiosurgery (GKRS) and botulinum toxin injections showing promise. Despite available treatments, TN's recurrent nature underscores the need for personalized, multidisciplinary management to address both physiological and mental health impacts.

KEYWORDS: trigeminal neuralgia, trigeminal neuralgia treatment, trigeminal neuralgia pathophysiology, trigeminal neuralgia lidocaine

1. Introduction

Trigeminal Neuralgia (TN), clinically referred to as tic douloureux, is a neuropathic pain disorder that specifically impacts the branches of the trigeminal nerve (cranial nerve V). This condition is distinguished by recurrent episodes of intense, unilateral facial pain, typically described as sharp, electric shock-like sensations that begin and end abruptly. These pain episodes are often short in duration but are severe enough to profoundly affect the individual's quality of life. Due to its chronic and unpredictable nature, TN frequently hinders patients' ability to perform fundamental tasks, including mastication, deglutition, and verbal communication, leading to considerable limitations in daily functioning.

In addition to the physical impairments, epidemiological research has highlighted the significant psychological burden associated with TN. Patients with this disorder commonly experience increased levels of anxiety and depression, which are believed to arise from the persistent fear of pain recurrence and the social limitations imposed by the condition. The chronicity of TN symptoms, combined with their impact on basic activities, contributes to a cumulative mental health burden, underscoring the need for comprehensive treatment approaches that address both the physical and psychological dimensions of this debilitating neuropathic disorder. [1,2]

2. Epidemiology

The lifetime prevalence of Trigeminal Neuralgia (TN) is estimated at approximately 0.16% to 0.3%, with an annual incidence of 4 to 29 cases per 100,000 person-years [1]. Females are more frequently affected by TN than males, with a prevalence ratio of 3:2, indicating that about 60% of TN cases occur in women [1]. The risk of TN increases with advancing age, particularly in the fifth and sixth decades, with the typical age of onset ranging from 53 to 57 years [1,2]. Familial classical trigeminal neuralgia (FCTN), which accounts for roughly 2% of TN cases, shows a strong female predominance, with an average onset age of 62.9 years (±13.93 years). Interestingly, a trend toward earlier onset has been observed in successive generations. The

maxillary branch (V2) of the trigeminal nerve is most commonly implicated in TN symptoms [3].

3. Pathomechanism

The etiology of trigeminal neuralgia is multifactorial.[4] It is primarily caused by damage to the trigeminal nerve (V) and the development of abnormal bioelectric discharges within its nerve cells.[6]

3.1 Trigeminal Nerve Root Compression

Injury to the trigeminal nerve is often linked to neurovascular compression (NVC), typically occurring where blood vessels in the prepontine cistern interact with the nerve root near Meckel's cave [4]. This compression, which can lead to nerve degeneration, may also result from tumors such as meningiomas and vestibular schwannomas, as well as aneurysms, arteriovenous malformations, and cystic growths [5]. These compressive forces induce myelin erosion through inflammatory mechanisms, impacting ion channel function by reducing Nav1.7 expression and increasing Nav1.3 regulation. Such alterations heighten neuronal excitability, exacerbating nociceptive sensitivity associated with TN [4].

Following trigeminal nerve (V) trauma, Schwann cells (SC) become activated and release neurotrophic agents, including nerve growth factor (NGF) and brain-derived neurotrophic factor (BDNF), which may further influence TN pathophysiology [6]. Additionally, patients with TN show elevated levels of inflammatory mediators in cerebrospinal fluid, such as pro-inflammatory cytokines, members of the TNF superfamily, chemokines, and growth factors, which are thought to contribute to TN's pathological development [7].

3.2 Demyelinating Diseases

Individuals with multiple sclerosis (MS) are at a significantly elevated risk of developing trigeminal neuralgia (TN), primarily due to the demyelination of nerve fibers associated with the disease. The likelihood of TN occurrence in MS patients is estimated to be around 20 times higher than in the general population [4, 38].

3.3 Familial Classical Trigeminal Neuralgia (FCTN)

Familial classical trigeminal neuralgia (FCTN) is presumed to follow an autosomal dominant inheritance pattern, with genetic anticipation observed in successive generations. Contributing factors in FCTN may include hereditary anatomical variations at the skull base, a family history of tortuous blood vessels, and genetic mutations affecting calcium channel genes, all of which are thought to play a role in FCTN pathogenesis [3].

4. Classification

The most recent classification of Trigeminal Neuralgia (TN) was introduced in 2018 by the International Headache Society (IHS) and the International Association for the Study of Pain (IASP) [2, 4]. According to the third edition of the International Classification of Headache Disorders (ICHD-3), diagnostic criteria for TN were established to help differentiate between various forms and underlying causes of the disorder [1, 9].

Table 1: Diagnostic Criteria for Trigeminal Neuralgia (TN)

Criterion	Description
A	Recurrent unilateral facial pain in one or more trigeminal nerve branches, confined to the affected area, meeting criteria B and C.
B	*Pain Characteristics:*
	1. Lasts from a fraction of a second to 2 minutes.
	2. Severe intensity.
	3. Sharp, electric shock-like, or stabbing pain.
C	Triggered by non-painful stimuli in the affected area of the trigeminal nerve.
D	Not better explained by another diagnosis per ICHD-3 criteria.

Sformatowana tabela

The revised classification system for Trigeminal Neuralgia (TN) includes three etiological categories.

Table 2: Etiological Categories of Trigeminal Neuralgia (TN)

Type	Description
Classical TN	Resulting from neurovascular compression (NVC) with morphological changes in the trigeminal nerve root.
Secondary TN	Caused by other pathological conditions, such as:
	- Tumors in the cerebellopontine angle.
	- Multiple Sclerosis (MS).
	- Brain tumors.
Idiopathic TN	Absence of NVC or NVC without morphological changes in the trigeminal nerve root, as confirmed by MRI and electrophysiological studies.

[8,15]

Sformatowana tabela

Table 3: Phenotypes of Trigeminal Neuralgia (TN)

Phenotype	Description
TN Purely Paroxysmal	Characterized solely by paroxysmal (sudden, brief) pain episodes.
TN with Coexisting Continuous Pain (CCP)	Includes both paroxysmal pain and a continuous dull, burning, or aching pain.

Sformatowana tabela

5. Clinical Symptoms

Trigeminal neuralgia (TN) pain predominantly affects regions supplied by the trigeminal nerve's second and third branches, with involvement of the first branch being rare, observed in less than 5% of cases [5]. The pain typically presents unilaterally, more frequently on the right side of the face, while bilateral pain appears in 1.7% to 5% of cases [1].

Initially, TN symptoms manifest as brief, infrequent episodes, but over time, these pain attacks tend to increase in frequency and intensity. The pain, often referred to as "paroxysms," is sudden, severe, and short-lived, typically lasting only a few seconds. Patients commonly describe the sensation as electric shock-like, stabbing, or burning in nature [10, 11]. These episodes are interspersed with refractory periods, whose durations vary among individuals [1, 2]. In severe cases, pain attacks may recur several times daily, with minimal refractory time in between.

A hallmark of TN is that pain can be triggered by benign sensory stimuli, including daily actions such as light touch, speaking, chewing, toothbrushing, or exposure to cold wind on the face [11]. Additionally, TN may involve autonomic symptoms, including unilateral tearing, pupil constriction, eyelid drooping, sweating, and nasal congestion, which often coincide with pain episodes [5].

6. Investigations and Diagnostics

According to the European Academy of Neurology (EAN) guidelines, Trigeminal Neuralgia (TN) should be categorized as either primary (classical or idiopathic) or secondary. For accurate diagnosis, MRI (magnetic resonance imaging) is essential, utilizing a combination of three high-resolution sequences: 3D T2-weighted, 3D TOF-MRA, and 3D T1-Gad. This approach facilitates the exclusion of secondary causes, the detection of neurovascular compression (NVC), and the evaluation of any morphological changes in the nerve resulting from vascular contact. When MRI is contraindicated, a CT scan with contrast can be an alternative imaging modality [8].

7. Treatment

First-line treatment for TN includes the medications carbamazepine (CBZ) and oxcarbazepine (OXC). If these agents lead to intolerable side effects or do not adequately control symptoms, second-line options, such as lamotrigine, baclofen, gabapentin, and pregabalin, are recommended. These may be administered as monotherapy or in combination with CBZ or OXC [8, 12].

For cases resistant to medication or where side effects are unmanageable, surgical intervention may be considered. Microvascular decompression (MVD) is the preferred surgical approach for patients with identified neurovascular compression. However, if MRI shows no neurovascular conflict, neuroablative techniques may be more appropriate. Given the

complexity of TN, optimal treatment requires a multidisciplinary approach involving healthcare professionals such as physicians, surgeons, nurses, and psychologists, aiming to address both the physiological and psychological needs of patients affected by this condition [8].

Table 4: First-Line Medications [12, 13, 14, 15]

Category	Carbamazepine (CBZ)	Oxcarbazepine (OXC)
Mechanism of Action	Antiepileptic agent; inhibits voltage-gated sodium channels to stabilize hyperexcitable neuronal membranes [15].	Similar mechanism to CBZ; frequency-dependent inhibition of sodium channels, stabilizing nerve excitability [15].
Usual Dose Range	800–1,200 mg daily [13].	1,200–1,800 mg daily [13].
Side Effects	Vertigo, somnolence, nausea, dizziness, ataxia, diplopia, hepatotoxicity [13].	Dizziness, ataxia, nausea, hepatotoxicity [13]; calmness, dizziness, blurred vision, unstable lethargy, diplopia [14].
Cautions	Myelosuppression, allergic skin rash, hyponatremia; contraindicated in patients on MAO inhibitors or with AV conduction block [12].	Cardiac insufficiency, AV conduction block, contraindicated for patients on acetylcholine receptor inhibitors [12].
Comparison	CBZ and OXC show similar efficacy in reducing pain episodes, but OXC has greater tolerability and lower interaction potential [15]. OXC is preferable as first-line treatment in secondary TN, with fewer adverse effects than CBZ. For patients with MS, monitoring for disease progression is recommended [14].	

Table 5: Second-Line Medications [12, 13, 14, 15, 16]

Second-Line Therapy	Lamotrigine (LTG)	Baclofen
Mechanism of Action	Acts on voltage-gated sodium channels, stabilizing neuronal membranes and inhibiting excitatory neurotransmitter release [15].	GABA B receptor agonist that reduces excitatory neurotransmitter release [15].
Dosage Regimen	200 mg twice daily [13].	40–80 mg daily [13].
Side Effects	Headache, rash, fatigue, dizziness, ataxia, nausea [12].	Nausea, somnolence, fatigue, gastrointestinal issues [12].
Caution	Risk of skin rash [12].	Use with caution in patients with cerebrovascular disease, reduced lung function, psychiatric conditions, Parkinson's disease, or epilepsy [12].
Additional Information	LTG is effective in secondary TN but may worsen symptoms in some multiple sclerosis (MS) patients [14].	Baclofen can be added if CBZ or OXC alone are insufficient. Also used in secondary TN with MS, aiding in spasticity reduction [13, 14].

8. Botulinum Toxin Treatment

Botulinum toxin A (BTX-A), a neurotoxin derived from *Clostridium botulinum*, blocks acetylcholine release at neuromuscular junctions, thereby reducing muscle contractions [17]. When administered intradermally or submucosally, BTX-A has shown analgesic effects in trigeminal neuralgia (TN) patients [8]. Adverse effects may include facial asymmetry, localized weakness, hematoma, edema, pain, and itching at the injection site [18]. Research indicates that repeated BTX-A injections are as effective as single doses, helping to reduce both pain intensity and frequency of TN attacks in both primary and secondary cases [19, 20].

9. Acute Management of Trigeminal Neuralgia Episodes

Lidocaine (LMP) is widely used to manage acute TN exacerbations and can be administered as a nasal aerosol, orally, as a patch, or via infusion. The aerosol application provides fast relief, especially when applied to the mucosal surface at a dose of 10 mg (two sprays) in the affected nostril [21]. Orally, lidocaine should remain in the mouth briefly before expelling to achieve effective local anesthesia [13]. In cases with facial trigger points, a 5% lidocaine patch is highly beneficial [22]. For patients unresponsive to other treatments, intravenous infusion of lidocaine at 5 mg/kg over 60 minutes can provide significant relief under ECG monitoring in an outpatient setting [23]. Additionally, lidocaine with corticosteroids is used for trigeminal nerve blocks, providing short- to medium-term relief [24].

10. Invasive Interventions

Invasive options for TN management include microvascular decompression (MVD), various percutaneous techniques (radiofrequency thermocoagulation, balloon compression, and chemical neurectomy), radiosurgery, and stereotactic radiosurgery.

10.1 Microvascular Decompression (MVD)

Considered the gold standard for classical TN treatment, MVD involves a suboccipital craniotomy to relieve neurovascular compression (NVC) [25]. Higher success rates have been observed in male patients, though complications such as permanent hearing loss, persistent hypesthesia, lasting ataxia, and stroke may occur [27]. Approximately 10% of cases experience recurrence, with risk factors including long-standing TN, older age, and atypical symptoms [39].

10.2 Percutaneous Procedures

Percutaneous techniques target the Gasserian ganglion through approaches like thermal injury, chemical rhizotomy, or mechanical compression. These methods are indicated when pharmacological treatments fail or are intolerable [26]. Although initially successful, percutaneous techniques often have a higher recurrence rate than MVD [34].

10.2A Radiofrequency Thermocoagulation (RFT)

Performed through the foramen ovale, RFT is often suitable for patients unfit for MVD. Immediate pain relief occurs in 85–97% of classical or idiopathic TN cases, with about 80% achieving relief lasting over a year. However, complications can include facial numbness, corneal reflex loss, and masseter weakness [28].

10.2B Glycerol Rhizotomy (PGR)

PGR, performed under CT guidance, is effective for TN cases associated with multiple sclerosis and treatment resistance. Potential side effects include sensory disruptions, masticatory weakness, and corneal desensitization [29-31].

10.2C Balloon Compression (PBC) of the Gasserian Ganglion (GG)

PBC provides rapid pain relief in about 80–90% of cases and is a low-risk option suitable for elderly patients. Common side effects include facial numbness, masticatory weakness, and corneal inflammation, though it is not recommended for patients with atypical facial pain or postherpetic neuralgia [32, 33].

10.3 Gamma Knife Radiosurgery (GKRS)

GKRS is an option for patients with contraindications for surgery due to age or comorbidities. It is used for TN linked to multiple sclerosis or herpes zoster. Hypesthesia is the most frequent complication, and pain recurrence is more likely compared to MVD. In cases of initial relief followed by recurrence, a second GKRS procedure may be considered [34, 35].

10.4 Stereotactic Radiosurgery (SRS)

SRS offers a non-invasive alternative for patients with complex comorbidities or when previous surgeries were ineffective. It is particularly beneficial for TN cases related to multiple sclerosis when standard medications fail to control symptoms adequately [36].

Conclusion

Trigeminal Neuralgia (TN) represents a significant challenge in clinical management due to its severe impact on daily life, psychological well-being, and functional ability. This review highlights the multifactorial nature of TN and the importance of a comprehensive approach that addresses both the physiological and psychological dimensions of this condition. Although various treatment options, including first-line pharmacological agents, second-line therapies, and invasive interventions, have shown efficacy, no single approach guarantees long-term relief due to TN's recurrent nature. The study underscores the value of individualized treatment strategies, multidisciplinary care, and ongoing research into advanced therapeutic options, which together may enhance pain management and quality of life for TN patients.

Discussion

This review highlights the complex, multifactorial nature of Trigeminal Neuralgia (TN) and its substantial impact on daily life, sports, and mental health, as well as the challenges in managing this chronic condition. However, a critical limitation of this review lies in its reliance on previously published research without contributing new clinical or experimental data. While this approach offers a comprehensive summary of available literature, it restricts the scope of analysis to secondary sources, leaving gaps in understanding about individualized patient responses and the effectiveness of combined therapies. Additionally, the review does not delve into novel or experimental therapies, which may hold potential for more effective pain management and fewer side effects than current options. Future studies that examine innovative treatments and patient-specific factors could provide deeper insights into optimizing TN management.

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Conceptualization- EKC, AP, JW

Methodology- EKC, CEJ, MG and JŚ;

Software- EHM and KB;

Check- ISW, AP, KK

Formal analysis- ISW, JW

Investigation- EKC

Resources- EKC, KK

Data curation- EJ, MG and JŚ;

Writing - rough preparation-EKC, EJ

Writing - review and editing- ISW, JW

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