QUALITY IN SPORT
eISSN 2450-3118 · Open Access · Peer-reviewed
apcz.umk.pl/QS Nicolaus Copernicus University in Toruń

MAŁEK, Zuzanna, WAŚKOWSKA, Katarzyna and KWIATKOWSKA, Marta. Diagnosis and Management of Trigeminal Neuralgia, Implications for Athletes: Narrative Review. Quality in Sport. 2026;72:74904. https://doi.org/10.12775/QS.2026.72.74904

ARTICLE TIMELINE
Received: 03.08.2026. Revised: 19.08.2026. Accepted: 07.09.2026. Published: 15.09.2026.

The journal has been awarded 20 points in the parametric evaluation by the Polish Ministry of Higher Education and Science (Annex to the announcement of 05.01.2024, No. 32553). Unique Journal Identifier: 201398. Scientific disciplines assigned: Economics and Finance (Field of Social Sciences); Management and Quality Sciences (Field of Social Sciences).
Punkty Ministerialne z 2019 – aktualny rok 20 punktów. Załącznik do komunikatu Ministra Szkolnictwa Wyższego i Nauki z dnia 05.01.2024 Lp. 32553. Posiada Unikatowy Identyfikator Czasopisma: 201398. Przypisane dyscypliny naukowe: Ekonomia i finanse (Dziedzina nauk społecznych); Nauki o zarządzaniu i jakości (Dziedzina nauk społecznych).

Diagnosis and Management of Trigeminal Neuralgia, Implications for Athletes: Narrative Review

Zuzanna Małek

ORCID: https://orcid.org/0009-0003-2427-3257

zuzia_malek@wp.pl

Institute of Dentistry of the Central Clinical Hospital of the Medical University of Lodz.

ul. Pomorska 251, 92-213 Łódź, Poland

Katarzyna Waśkowska

ORCID: https://orcid.org/0009-0005-4761-0425

k.waskowska534@wp.pl

Institute of Dentistry of the Central Clinical Hospital of the Medical University of Lodz.

ul. Pomorska 251, 92-213 Łódź, Poland

Marta Kwiatkowska
ORCID: https://orcid.org/0009-0008-0198-0115
marta.kwiatek2001@gmail.com
Institute of Dentistry of the Central Clinical Hospital of the Medical University of Lodz.
ul. Pomorska 251, 92-213 Łódź, Poland

Corresponding Author:
zuzia_malek@wp.pl

Abstract

Background:

Trigeminal neuralgia (TN) is a chronic neuropathic pain disorder that causes sudden episodes of intense facial pain. Among athletes, recurrent pain attacks and the adverse effects of treatment may interfere with training, performance, and return to competition.

Aim:

This review analyzes current diagnostic standards, pathophysiological mechanisms, and treatment options for TN. It highlights its impact on physical activity and the particular needs of athletic individuals.

Material and Methods:

A literature search was conducted using electronic databases, including PubMed, with the following keywords: “Trigeminal neuralgia”, “Orofacial Pain”, and “Physical Activity”. The review included randomized controlled trials, meta-analyses, and systematic reviews from 1967 to the present, with most studies published between 2020 and 2025.

Results:

TN is usually diagnosed based on clinical symptoms. Attacks involve sudden, one-sided, brief, and shock-like pain triggered by mild stimuli. MRI is the gold standard for detecting neurovascular compression. As a pharmacological treatment, carbamazepine is the first choice. However, its side effects, such as ataxia and dizziness, can strongly impair athletic performance. Surgical interventions, including microvascular decompression (MVD), have high long-term success rates and may be considered sooner for active people.

Conclusion:

Managing TN requires a multidisciplinary approach that takes into consideration environmental triggers and the impact of medication on the quality of life.

Key words:

Trigeminal Neuralgia, Facial Pain, Microvascular Decompression, Physical Activity.

1. Introduction

Trigeminal neuralgia (TN), historically termed tic douloureux, is a chronic neuropathic pain syndrome characterized by recurrent, unilateral pain attacks. (1,2) Patients describe the pain as shock-like, sharp, or burning. (3,4) The pain is limited to the distribution of branches of the trigeminal nerve. (3,5) TN most commonly affects a single branch but may also involve two or, less frequently, all three branches. (6,7) The pain is extremely severe; it can last from a fraction of a second to a few minutes. (3,7) Due to the extreme intensity of the pain, the condition has earned the colloquial name: "suicide disease". (1,8)

The attacks are usually triggered by objectively mild stimuli. (4) They can be caused by washing the face, eating, grooming, or talking. (9,10) People who exercise outdoors can be affected by attacks triggered by a breeze or cold wind, which can interfere with their training program. (4,9,11) Most pain attacks are followed by a refractory period, during which additional stimulation is unlikely to trigger another attack. (7) Some patients remain

asymptomatic between the TN episodes, but up to half may develop a concomitant persistent background pain. It is described as a dull, aching, or burning sensation. (4,12)

The impact of TN on the quality of life is profound and multifactorial. (13) The attacks are unpredictable and cause a state of constant fear. (7,14) This leads to severe psychological distress, social withdrawal, and eating avoidance. (7) This is also important in athletes, as it may cause them to give up physical activity. (15) Epidemiological data indicate that TN patients suffer from significantly higher rates of anxiety, depression, and sleep disturbances compared to the general population. (7,10,12) If left untreated, the resulting psychiatric morbidities can complicate future treatment outcomes and, in fact, increase the risk of suicidal ideation. (1,4)

According to the International Classification of Headache Disorders (ICHD-3), TN is etiologically divided into three categories: classical TN (caused by neurovascular compression), secondary TN (resulting from underlying diseases such as multiple sclerosis or tumors), and idiopathic TN (where no clear cause is identified). (5,16) Managing trigeminal neuralgia requires a multidisciplinary approach—involving neurologists, neurosurgeons, dentists, and psychologists. (17,18) While first-line pharmacotherapy often provides initial relief, the pain can return over time. (19,20) Patients often need surgical intervention to permanently restore daily function and psychological well-being. (4,17) Moreover, the pharmacological treatment with carbamazepine or oxcarbazepine often causes systemic side effects including ataxia, dizziness, and somnolence. (9,10,21) These effects can directly affect an athlete's balance, reaction time, and overall performance. (9) In these cases, surgical modalities like microvascular decompression (MVD) are a preferred early consideration to restore function without the burden of sedative medication. (5,8,17)

2. Epidemiology and pathophysiology of trigeminal neuralgia

2.1. Epidemiology

TN is considered the most common form of craniofacial neuropathic pain. (12)

Incidence: The annual incidence is estimated to be between 4 and 29 cases per 100,000 people. (5,17)

Gender: There is a noticeable female predominance, with reported ratios ranging from 1.5:1 up to 3:1.(1,3,22)

Age: The mean age of onset is typically in the fifth to sixth decades of life (53–57 years). (11,17) Onset before age 40 is rare and should be checked for secondary causes such as multiple sclerosis (MS). (23,24)

Laterality: The right side of the face is affected more frequently than the left (approx. 60%). Possibly due to the anatomical narrowness of the foramen rotundum and ovale on that side. (21,24,25)

2.2. Etiology and Classification

The International Classification of Headache Disorders (ICHD-3) divides TN into three etiologic categories: (1,7)

Classical TN: Caused by neurovascular compression (NVC) at the trigeminal root entry zone (REZ), usually by the superior cerebellar artery. (4,7,12)

Secondary TN: Caused by an underlying neurological disease, most commonly multiple sclerosis or cerebellopontine angle tumors. (9,20) MS patients have a 20 times greater risk of developing TN. (10,14)

Idiopathic TN: Diagnosed when no obvious cause is detected. (4,23)

Mechanisms of trigeminal neuralgia

The ignition hypothesis is currently the best-supported explanation for the pathophysiology of TN. (1,19,26) According to this theory, focal demyelination at the root entry zone (REZ) in the transition zone results in axonal hyperexcitability. (8,17) This promotes ephaptic transmission (cross-talk). (5,21,27) This causes innocuous tactile stimuli carried by A-β fibers to activate adjacent A-δ nociceptive fibers, which generate pain. In addition, the expression of voltage-gated sodium channels changes, with upregulation of Nav1.3 and downregulation of Nav1.7. (6,7,28) This additionally increases neuronal excitability and facilitates the generation of ectopic action potentials. Schwann cells also play an important role in both the degeneration and regeneration of the myelin sheath following nerve injury. (22)

Upon injury, such as neurovascular compression, Schwann cells activate. They transition to a repair phenotype. (7,22) Activated SCs no longer maintain myelin and instead begin to degrade and engulf the myelin sheath via phagocytosis. (7,22) This focal demyelination creates short circuits or ephaptic transmission between fibers, which is a primary cause of the sudden pain episodes in TN. (5,9) Moreover, SCs release inflammatory cytokines such as TNF-α and IL-

1β, as well as neurotrophic factors such as BDNF and NGF. They lower the excitability threshold, promoting neuronal hyperactivity and chronic pain hypersensitivity. (5,22,28)

After the initial damage, Schwann cells play a key role in nerve regeneration. Activated SCs proliferate and align into longitudinal columns known as bands of Büngner. These structures create physical channels that provide guidance and structural support for regenerating axons. Additionally, SCs secrete factors such as NGF, BDNF, and GDNF that nourish injured neurons and stimulate their elongation. (22) Once the axon has successfully regrown, Schwann cells wrap around it to form a new myelin sheath. This restores normal impulse conduction and potentially alleviates neuralgic symptoms. (5,22)

3. Diagnosis

3.1. Clinical Symptoms

Diagnosis relies almost entirely on a thorough patient history. (29) The key criteria include:

Paroxysms: Sudden, severe pain attacks. They last from a fraction of a second up to 2 minutes. (14) The pain is often described as shock-like, stabbing, or excruciating. (4,18,30)

Trigger Zones: The trigeminal neuralgia episodes are typically triggered by light stimulation. Patients often report that talking, washing the face, or a breeze can cause pain. (9,24,31)

Refractory Period: After each attack, there is a brief interval in which the affected nerve becomes temporarily less responsive to trigger stimuli. (7,30,32) This happens because the high-frequency pathological repetitive neuronal discharges eventually cease, leading to a temporary phase of neuronal inactivity. (4,12)

Phenotypes: Purely paroxysmal pain (TN1) or TN with concomitant continuous background pain (TN2). (3,21) The TN2 pain is often described as aching or burning. (7,19) The presence of continuous background pain in TN2 suggests central sensitization. (7,32) This means that the central nociceptive system becomes hyperexcitable. (5,27) It leads to amplified signal transmission within the brainstem and supraspinal modulating circuits. (7) A central mechanism involving denervation supersensitivity in TN2 is associated with progressive nerve root atrophy. (5) Patients with this type of trigeminal neuralgia are less responsive to pharmacological therapy. They should be treated surgically to prevent further disease progression. (4,10)

3.2. Imaging and Differential Diagnosis

Magnetic Resonance Imaging (MRI) is performed to detect or exclude secondary causes. (25)

MRI can detect:

Identifiable diseases causing nerve damage, such as Multiple Sclerosis (demyelinating plaques), tumors in the cerebellopontine angle, and vascular malformations such as aneurysms or AVMs. (13,33)

Neurovascular Compression (NVC): Direct contact between the trigeminal nerve and a blood vessel, most commonly the superior cerebellar artery (SCA). (3,31)

Morphological Nerve Changes: Objective signs of injury from compression, including nerve atrophy (volume loss), indentation, dislocation, distortion, or flattening. (4,13,17)

Inflammatory Conditions: Uncommon pathologies like trigeminal lipomatosis or radiculitis following infection or vaccination. (1,34)

Advanced Microstructural Changes: Using Diffusion Tensor Imaging (DTI), MRI can detect demyelinating damage and localized edema within the nerve root. (7,26)

Global Brain Impact: Chronic TN can lead to gray matter volume reduction in areas like the somatosensory cortex and thalamus. (3,28)

High-resolution sequences such as CISS (Constructive Interference in Steady State) and TOF (Time-of-Flight) are essential for visualizing neurovascular contact and morphological changes. (23,24,34)

Differential diagnoses for trigeminal neuralgia include Temporomandibular Disorders (TMD), persistent idiopathic facial pain, and SUNCT syndrome. (1,29)

4. Treatment Strategies

Successful treatment of trigeminal neuralgia (TN) is achieved by a multidisciplinary approach. It involves neurologists, neurosurgeons, dentists, and psychologists. (9) Psychological support is necessary for patients to cope with the physical pain and emotional burden. (7,23)

4.1. Pharmacological Treatment

Pharmacotherapy is the first defense against pain in TN. It's meant to stabilize hyperexcited neuronal membranes and reduce ectopic discharges. (6,20)

First-line Therapy: Carbamazepine (200–1200 mg/day) and Oxcarbazepine (600–1800 mg/day) remain the gold standard for initial medical treatment. (7,17) Carbamazepine is the only drug for TN approved by the FDA. (4) It offers initial relief in nearly 90% of patients. However, its effectiveness decreases over time, with an approximately 50% success rate. (14,21,23) It has a lot of side effects like ataxia, dizziness, somnolence, hyponatremia, as well as a rare risk of aplastic anemia. (3,17,30) Oxcarbazepine is a structural analog that is often better tolerated. (7,9) It has a lower risk of drug interaction. However, it carries a higher risk of hyponatremia. (2,15) The side effects of both these medications limit their utilization in elderly patients, cardiological patients, and athletes. (3,25,35)

Adjuvant and Second-line Therapies: If first-line agents are ineffective or poorly tolerated, medications such as Lamotrigine, Gabapentin, Pregabalin, and Baclofen may be substituted or added. (5,6,26)

Lamotrigine acts as a sodium channel blocker but requires slow titration over several weeks to avoid severe skin reactions, such as Stevens-Johnson syndrome. (8,9) The slow onset makes it unsuitable for acute use.

Baclofen is particularly useful in patients with multiple sclerosis who experience spasticity. (6,14) Adverse effects are common and occur in up to 75% of patients. They include muscle weakness, nausea, sedation, confusion, and hallucinations. (6,12) To avoid severe withdrawal symptoms, baclofen should always be discontinued gradually. (9)

Botulinum toxin type A is an adjunctive treatment. (12,25,26) Administered via subcutaneous injections, it provides a local effect of pain relief for up to three months. (20) It interacts with the SNARE complex (specifically SNAP-25) to block the fusion of synaptic vesicles. (3,6) Side effects are typically local and transient, resulting from temporary involvement of adjacent motor nerve fibers. These include facial asymmetry, drooling, and mild weakness of the masticatory muscles. (5)

Acute Pain Management: In-hospital management of severe paroxysms may require intravenous (IV) rescue therapy. This route of administration is preferred during a crisis when medications cannot be taken orally. (1,33)

Phenytoin (15 mg/kg) and Fosphenytoin can provide rapid relief but require cardiac and blood pressure monitoring due to a risk of hypotension and asystole. (11,23)

IV Lidocaine (1.5–5 mg/kg) is another rescue option that modulates both peripheral and central pain mechanisms. Its side effects include dizziness or potential arrhythmias. (12,33)

Complementary and Investigational Options: Electroacupuncture combined with low-dose carbamazepine has demonstrated a synergistic analgesic effect. It offers an alternative for patients unable to tolerate high pharmacological dosages. (3,17,35)

New selective Nav1.7 blockers, such as Vixotrigine, are currently in phase 3 trials and may offer better tolerability by targeting nociceptive afferents without central nervous system depression. (10,28)

4.2. Surgical Treatment

Surgical intervention is especially useful in patients who don't respond to pharmacological treatment or suffer from its side effects. (1,21) It has a greater success rate; however, invasive treatment always carries risks. The following section describes the most commonly performed surgical procedures. (4,5)

Microvascular Decompression (MVD): It is the treatment of choice for classical TN caused by neurovascular compression. (3,36) This non-ablative procedure is performed by suboccipital craniotomy. (6,37) After identifying the offending vessel, the practitioner separates it from the nerve root entry zone using a prosthesis such as a Teflon sponge. (7,25) Most commonly, the superior cerebellar artery is the one to compress the trigeminal nerve. (4,8,31) MVD offers the highest long-term success rate. 62–89% of patients remain pain-free five years after the procedure. (5,9,20) Potential complications include a small risk of mortality (0.3%), hearing loss (1.8–5%), and cerebrospinal fluid leaks (1.6–4%). (4,5,38)

Neuroablation: This technique involves controlled damage to the trigeminal nerve to interrupt pain transmission. (5,39) It is preferred for elderly or frail patients who cannot undergo major surgery. (6,17)

- Percutaneous Techniques: These include radiofrequency thermocoagulation (thermal damage), balloon compression (mechanical damage), and glycerol rhizolysis (chemical damage) of the Gasserian ganglion. (6,7,17) They provide immediate relief. (14) Unfortunately, they are associated with more recurrent cases and sensory loss or facial numbness. (8,9,24)

- Stereotactic Radiosurgery: This is a non-invasive, ablative procedure. It includes the use of the Gamma Knife or CyberKnife. (7,17,21) It focuses ionizing radiation on the trigeminal root entry zone. (9) Unlike other surgical interventions, the pain relief is delayed by weeks or up to six months. (4,8) It also has a higher recurrence rate compared to MVD. (9,23)

Internal Neurolysis (Nerve Combing): This procedure is used when no neurovascular compression is identified intraoperatively. (7,9) The nerve fascicles are divided longitudinally to interrupt any abnormal signaling. (9) While effective, this procedure carries a significant risk of facial hypoaesthesia and anesthesia dolorosa. (5)

Tab. 1 Comparison of Treatment Modalities Effectiveness (3–5,7,20,23,26,38)

Treatment	Initial effectiveness	5-year pain-free	Key notes
Carbamazepine	70–90% initial response	50%	~50% require additional treatment/surgery within 5–10 years
Oxcarbazepine	Comparable to CBZ	—	Better tolerability, fewer interactions
MVD	80–96%	62–89% (~70% at 10 years)	Gold standard for classical TN
Radiofrequency	High immediate relief	26–82%	Higher recurrence than MVD
Balloon compression	High	55–80%	Preferred in frail/elderly
Gamma Knife	Delayed onset	30–66%	Weeks–months to effect
Glycerol rhizolysis	Moderate	19–58%	Highest recurrence

5. Impact on Quality of Life with Special Consideration of Athletes

TN dramatically impairs daily functioning and social interactions. The patients live in a constant fear of pain. (5,6) This leads to severe psychosocial stress. Literature describes suicidal thoughts and attempts in TN patients. (4,7,18)

The trigeminal neuralgia episodes are excruciating; the pain is described as the worst a person can endure. (8,11) Even treated patients can experience recurrent attacks, which cause a profound mental health impact. (5,19) TN patients present a significantly higher risk of developing comorbid psychiatric conditions, including anxiety, depression, and sleep disorders. (9,18,35) Research estimates that TN patients have a three times higher risk of anxiety and depression compared to the general population. Approximately 35.7% of patients suffer from mild to severe depression, and half experience anxiety symptoms. (9,18) The prevalence and severity of mental health disorders correlate with both the strength of pain and duration of the main disease. (4) However, the difference between the suicidal rates in different TN phenotypes has been described. Whereas the anxiety level is high for both TN types, the risk of suicidal ideation is significantly higher in patients with TN Type 2. (18)

5.1.Unique Challenges for Athletes

Because of its extreme and unpredictable character, trigeminal neuralgia is specifically challenging for people with an active lifestyle. (4,7) The key problem for athletes is that pain episodes can be triggered by routine elements of training. (15,25)

The most common triggers connected to physical activity are:

Movement: Rapid head movement can cause the pain episode. This involves talking or heavy breathing. (10,30)

Environmental Factors: Things such as air breeze, changes in temperature, or humidity can trigger the pain. This is especially important for outdoor activities such as jogging or cycling. (11,40)

Hydration: Maintaining hydration during exercise can be challenging, as drinking cold fluids may stimulate intraoral trigger zones and provoke pain attacks. Although it is challenging for all patients with trigeminal neuralgia, athletes during training are exposed to multiple stimuli that may increase susceptibility to pain attacks. (7,20)

Treatment in active individuals

Standard pharmacological treatment with antiepileptic drugs is associated with adverse effects that can impair sports performance. (4,10)

Ataxia, dizziness: These are the most common side effects. (4,14) They can significantly increase the risk of falls and injury by impairing balance and reaction time. They also affect performance in sports requiring precision. (9,10)

Somnolence: Carbamazepine can cause low concentration and reaction time. (5,9)

Hyponatremia: Both carbamazepine and oxycarbamazepine can cause hyponatremia. It is especially important in athletes, as they also lose electrolytes in sweat. (4,12,17)

Because of the side effects of the pharmacological treatment, surgical intervention is often considered early in active individuals. (14) The exception is botulinum toxin type A. It is administered subcutaneously or submucosally and does not cause systemic side effects, such as affecting the body's overall function or performance. (4,20) The effects are local, and the pain relief lasts for approximately three months. (5,6)

However, microvascular decompression is still considered the gold standard for TN treatment. It targets the cause of the pain rather than reducing the symptoms. (20,23,39) When nerve compression is dealt with, many patients are able to completely discontinue pharmacological treatment. (5,41)

When it comes to Electroacupuncture, it can be useful in inoperable cases. (7) It is an adjuvant therapy that allows dosage reduction. This can significantly increase tolerance of the treatment by reducing side effects. (35)

6. Summary and Conclusions

Trigeminal neuralgia (TN) is a chronic neuropathic pain syndrome. (1) It is characterized by sudden, unilateral, and brief episodes of pain. (3,5) Patients describe the pain as electric, shock-like, or stabbing with great force. (18,24) It is limited to the area of one or more branches of the fifth cranial nerve. (4,23) The condition predominantly affects individuals over the age of 50, with a slight female predominance. (3,11) It is etiologically classified into classical TN caused by neurovascular compression, secondary TN resulting from underlying diseases such as multiple sclerosis or tumors, and idiopathic TN where no cause is identified. (7,9) Diagnosis

relies almost entirely on a thorough clinical history to identify pathognomonic features. (9,17,25) The pain is caused by stimulating trigger zones with refractory periods following episodes. (27,32) Magnetic resonance imaging (MRI) is essential to exclude structural causes and evaluate morphological changes, such as nerve root atrophy at the entry zone to the brainstem. (1,13) Effective management of TN requires a multidisciplinary approach. (3,9,21) The extreme intensity of the pain is linked to a significant risk of anxiety, depression, and suicidal ideation. (6,18,39) It is especially noticeable in the TN Type 2 phenotype characterized by concomitant continuous background pain. (7,18) Pharmacotherapy with sodium channel blockers, specifically carbamazepine and oxcarbazepine, is the first-line standard. (4,17,42) Its long-term utility is, however, often limited by reduced efficacy and adverse effects like ataxia, dizziness, and hyponatremia. (20,35) These are particularly concerning for physically active patients and athletes. (6,9,35) Microvascular decompression (MVD) remains the surgical gold standard for classical TN, especially in recurrent cases. (8,23) MVD offers durable relief and pain-free rates of 62–89% at five years by directly addressing the cause of the pain. (3,8) In elderly or medically frail patients, less invasive neuroablative procedures such as stereotactic radiosurgery or balloon compression serve as alternatives. (10,17) Unfortunately, they carry higher recurrence rates and the risk of permanent sensory deficits. (6,23) MVD demonstrates superiority over chronic pharmacotherapy for long-term pain control. (15,20) Moreover, specific requirements of athletes for maintaining motor coordination and metabolic safety indicate the need for early consideration of surgery or local botulinum toxin type A. (7,21,26) These modalities help avoid debilitating systemic drug effects. Ultimately, clinical outcomes and prognosis are highly dependent on the pain phenotype, as TN Type 2 patients represent a more therapeutically challenging group. They tend to require more aggressive and comprehensive neurological and psychological support. (4,5,18)

Disclosure

Supplementary Materials:

Not applicable.

Author's Contribution:

Conceptualization: Zuzanna Małek

Methodology: Aleksandra Wyciślok, Zuzanna Małek, Olgierd Czapiński, Katarzyna Waśkowska, Weronika Kubiak, Julia Kalsztein, Marta Kwiatkowska, Sonia Krzykawska

Software: Not applicable.

Check: Katarzyna Waśkowska

Formal analysis: Marta Kwiatkowska, Zuzanna Małek

Investigation: Katarzyna Waśkowska, Zuzanna Małek

Resources: Marta Kwiatkowska

Data curation: Zuzanna Małek, Katarzyna Waśkowska

Writing-rough preparation: Zuzanna Małek, Marta Kwiatkowska

Writing review and editing: Zuzanna Małek, Marta Kwiatkowska, Katarzyna Waśkowska

Visualization: Katarzyna Waśkowska

Supervision: Zuzanna Małek

Project administration: Marta Kwiatkowska

Receiving funding: Not applicable.

All authors have read and agreed with the published version of the manuscript.

Funding statement:

This research has not received any external funding.

Institutional Review Board Statement:

Not applicable.

Informed Consent Statement:

Not applicable.

Data Availability Statement:

Not applicable.

Acknowledgments:

Not applicable.

Conflict of Interest Statement:

The authors deny any conflict of interest.

Declaration of generative AI and AI-assisted technologies in the writing process:

In preparing this work, the authors used ChatGPT (OpenAI) for the purpose of grammar correction and improving writing quality only. After using this tool, the authors have reviewed and edited the content as needed and accept full responsibility for the substantive content of the publication.

References:

1. Alwardian M, Chrysikos D, Samolis A, Papachristou A, Spartalis E, Piagkou M, et al. Trigeminal Neuralgia and Potential Correlations with Anatomical Variations of the Trigeminal Nerve. Acta Medica Acad. sierpnia 2021;50(2):292–9. doi:10.5644/ama2006-124.344 PubMed PMID: 34847681.

2. Brito AJ. Nevralgia do trigémio. Acta Médica Port. 1999;12(4–6):187–93. doi:10.20344/amp.2144

3. Alzeeralhouseini A, Moisak G, Labzina E, Rzaev J. Trigeminal neuralgia caused by venous compression: a comprehensive literature review. J Med Life. maja 2024;17(5):462–70. doi:10.25122/jml-2024-0040 PubMed PMID: 39144685; PubMed Central PMCID: PMC11320616.

4. Araya EI, Claudino RF, Piovesan EJ, Chichorro JG. Trigeminal Neuralgia: Basic and Clinical Aspects. Curr Neuropharmacol. lutego 2020;18(2):109–19. doi:10.2174/1570159X17666191010094350 PubMed PMID: 31608834; PubMed Central PMCID: PMC7324879.

5. Lambru G, Zakrzewska J, Matharu M. Trigeminal neuralgia: a practical guide. Pract Neurol. października 2021;21(5):392–402. doi:10.1136/practneurol-2020-002782 PubMed PMID: 34108244; PubMed Central PMCID: PMC8461413.

6. Gambeta E, Chichorro JG, Zamponi GW. Trigeminal neuralgia: An overview from pathophysiology to pharmacological treatments. Mol Pain. 2020;16:1744806920901890. doi:10.1177/1744806920901890 PubMed PMID: 31908187; PubMed Central PMCID: PMC6985973.

7. Chen Q, Yi DI, Perez JNJ, Liu M, Chang SD, Barad MJ, et al. The Molecular Basis and Pathophysiology of Trigeminal Neuralgia. Int J Mol Sci. 25 marca 2022;23(7):3604. doi:10.3390/ijms23073604 PubMed PMID: 35408959; PubMed Central PMCID: PMC8998776.

8. Bennetto L, Patel NK, Fuller G. Trigeminal neuralgia and its management. BMJ. 27 stycznia 2007;334(7586):201–5. doi:10.1136/bmj.39085.614792.BE PubMed PMID: 17255614; PubMed Central PMCID: PMC1782012.

9. Chong MS, Bahra A, Zakrzewska JM. Guidelines for the management of trigeminal neuralgia. Cleve Clin J Med. 1 czerwca 2023;90(6):355–62. doi:10.3949/ccjm.90a.22052 PubMed PMID: 37263669.

10. Di Stefano G, Maarbjerg S, Truini A. Trigeminal neuralgia secondary to multiple sclerosis: from the clinical picture to the treatment options. J Headache Pain. 19 lutego 2019;20(1):20. doi:10.1186/s10194-019-0969-0 PubMed PMID: 30782116; PubMed Central PMCID: PMC6734488.

11. Sveinsson OA, Arkink EB, Ulfarsson E, Thors B. [Trigeminal neuralgia - Overview]. Laeknabladid. lutego 2025;111(2):62–7. doi:10.17992/lbl.2025.02.825 PubMed PMID: 39878591.

12. Chow A, Haider I, Athanaselos A, Patel M. In-Hospital Management of Acute Trigeminal Neuralgia Pain Crises. Clin Med Res. grudnia 2024;22(4):215–21. doi:10.3121/cmr.2024.1945 PubMed PMID: 39993829; PubMed Central PMCID: PMC11849969.

13. Zhao W, Yin C, Ma L, Ding M, Kong W, Wang Y. Predictive value of MRI for identifying symptomatic neurovascular compressions in classical trigeminal neuralgia: a PRISMA-compliant meta-analysis. BMC Neurol. 29 listopada 2024;24(1):466. doi:10.1186/s12883-024-03977-6 PubMed PMID: 39614218; PubMed Central PMCID: PMC11606272.

14. Zakrzewska JM, Linskey ME. Trigeminal Neuralgia. Am Fam Physician. 15 lipca 2016;94(2):133–5.

15. Zakrzewska JM. Differential diagnosis of facial pain and guidelines for management. Br J Anaesth. lipca 2013;111(1):95–104. doi:10.1093/bja/aet125 PubMed PMID: 23794651.

16. Zhao L, Hou Y, Wang J. Knowledge, attitudes, and practices among patients with combined dentition defect and non-functional impacted teeth toward tooth autotransplantation. BMC Oral Health. 4 lipca 2024;24(1):761. doi:10.1186/s12903-024-04545-7 PubMed PMID: 38965503; PubMed Central PMCID: PMC11225189.

17. Snel BJ, Cohen SP, Erdine S, Day MR, Van Zundert J, Vissers K, et al. 13. Trigeminal Neuralgia. Pain Pract Off J World Inst Pain. czerwca 2025;25(5):e70051. doi:10.1111/papr.70051 PubMed PMID: 40384548; PubMed Central PMCID: PMC12086744.

18. Martinelli R, Vannuccini S, Burattini B, D'Alessandris QG, D'Ercole M, Izzo A, et al. Psychological assessment in patients affected by trigeminal neuralgia. A systematic review. Neurosurg Rev. 13 maja 2025;48(1):414. doi:10.1007/s10143-025-03556-4 PubMed PMID: 40355578; PubMed Central PMCID: PMC12069416.

19. Eide PK. Familial occurrence of classical and idiopathic trigeminal neuralgia. J Neurol Sci. 15 marca 2022;434:120101. doi:10.1016/j.jns.2021.120101 PubMed PMID: 34954619.

20. Gerwin R. Chronic Facial Pain: Trigeminal Neuralgia, Persistent Idiopathic Facial Pain, and Myofascial Pain Syndrome—An Evidence-Based Narrative Review and Etiological

Hypothesis. Int J Environ Res Public Health. października 2020;17(19):7012. doi:10.3390/ijerph17197012 PubMed PMID: 32992770; PubMed Central PMCID: PMC7579138.

21. Khan M, Nishi SE, Hassan SN, Islam MdA, Gan SH. Trigeminal Neuralgia, Glossopharyngeal Neuralgia, and Myofascial Pain Dysfunction Syndrome: An Update. Pain Res Manag. 2017;2017:7438326. doi:10.1155/2017/7438326 PubMed PMID: 28827979; PubMed Central PMCID: PMC5554565.

22. Liao JY, Zhou TH, Chen BK, Liu ZX. Schwann cells and trigeminal neuralgia. Mol Pain. 2020;16:1744806920963809. doi:10.1177/1744806920963809 PubMed PMID: 33054604; PubMed Central PMCID: PMC7786407.

23. Kolakowski L, Pohl H, Stieglitz L, De Vere-Tyndall A, Soyka MB, Räber-Jäggy P, et al. Interdisciplinary strategies for diagnosis and treatment of trigeminal neuralgia. Swiss Med Wkly. 24 lipca 2024;154:3460. doi:10.57187/s.3460 PubMed PMID: 39137345.

24. Krafft RM. Trigeminal Neuralgia. Am Fam Physician. 1 maja 2008;77(9):1291–6.

25. Villegas Díaz D, Guerrero Alvarado G, López Medina A, Gómez Clavel JF, García Muñoz A. Trigeminal neuralgia: therapeutic strategies to restore quality of life. J Oral Facial Pain Headache. 38(3):32–7. doi:10.22514/jofph.2024.024 PubMed PMID: 39800569; PubMed Central PMCID: PMC11810647.

26. Mousavi SH, Lindsey JW, Westlund KN, Alles SRA. Trigeminal Neuralgia as a Primary Demyelinating Disease: Potential Multimodal Evidence and Remaining Controversies. J Pain. lutego 2024;25(2):302–11. doi:10.1016/j.jpain.2023.08.012 PubMed PMID: 37643657.

27. De Stefano G, Litewczuk D, Mollica C, Di Pietro G, Galosi E, Leone C, et al. Sex differences in trigeminal neuralgia: a focus on radiological and clinical characteristics. Neurol Sci Off J Ital Neurol Soc Ital Soc Clin Neurophysiol. grudnia 2023;44(12):4465–72. doi:10.1007/s10072-023-06923-5 PubMed PMID: 37436558; PubMed Central PMCID: PMC10641090.

28. Mannerak MA, Lashkarivand A, Eide PK. Trigeminal neuralgia and genetics: A systematic review. Mol Pain. 2021;17:17448069211016139.

doi:10.1177/17448069211016139 PubMed PMID: 34000891; PubMed Central PMCID: PMC8135221.

29. Slettebø H. Is this really trigeminal neuralgia? Diagnostic re-evaluation of patients referred for neurosurgery. Scand J Pain. 1 października 2021;21(4):788–93. doi:10.1515/sjpain-2021-0045

30. Henderson WR. Trigeminal neuralgia: the pain and its treatment. Br Med J. 7 stycznia 1967;1(5531):7–15. doi:10.1136/bmj.1.5531.7 PubMed PMID: 5334334; PubMed Central PMCID: PMC1840726.

31. Truini A, Galeotti F, Cruccu G. New insight into trigeminal neuralgia. J Headache Pain. września 2005;6(4):237–9. doi:10.1007/s10194-005-0195-9 PubMed PMID: 16362674; PubMed Central PMCID: PMC3452002.

32. Kumar S, Rastogi S, Kumar S, Mahendra P, Bansal M, Chandra L. Pain in trigeminal neuralgia: neurophysiology and measurement: a comprehensive review. J Med Life. 15 grudnia 2013;6(4):383–8. PubMed PMID: 24701256; PubMed Central PMCID: PMC3973876.

33. Mohamed MW, Irem-Oko F, Sheikh A, Phillips N, Mckinlay J, Anderson I. Lidocaine infusion for the treatment of intractable trigeminal neuralgia: retrospective case series and systematic review. Acta Neurochir (Wien). 29 września 2025;167(1):259. doi:10.1007/s00701-025-06672-8 PubMed PMID: 41021111; PubMed Central PMCID: PMC12479685.

34. Sehgal A, Kumar J, Singh I, Gopal A. Trigeminal Lipomatosis: A Rare Cause of Intractable Neuralgia. J Radiol Case Rep. sierpnia 2023;17(8):49–56. doi:10.3941/jrcr.v17i8.4709 PubMed PMID: 38090637; PubMed Central PMCID: PMC10713231.

35. Li R, Sun J, Luo K, Luo N, Sun R, Gao F, et al. Electroacupuncture and carbamazepine for patients with trigeminal neuralgia: a randomized, controlled, 2 × 2 factorial trial. J Neurol. sierpnia 2024;271(8):5122–36. doi:10.1007/s00415-024-12433-x PubMed PMID: 38816482; PubMed Central PMCID: PMC11319385.

36. Brinzeu A, Sindou M. Retrogasserian trigeminal radiofrequency-thermorhizotmoy for trigeminal neuralgia. Acta Neurochir (Wien). 10 maja 2024;166(1):209. doi:10.1007/s00701-024-06074-2 PubMed PMID: 38727725; PubMed Central PMCID: PMC11087336.

37. Sakakura K, Akutsu H, Yamamoto T, Masuda Y, Ishikawa E, Matsumura A. Trigeminal neuralgia in a patient with Marfan syndrome: case report. Neurol Med Chir (Tokyo). 2015;55(1):101–5. doi:10.2176/nmc.cr.2013-0072 PubMed PMID: 24390183; PubMed Central PMCID: PMC4533392.

38. Molina-Olier O, Marsiglia-Pérez D, Alvis-Miranda H. Surgical treatment of trigeminal neuralgia in adults. Cir Cir. 2022;90(4):548–55. doi:10.24875/CIRU.20000772 PubMed PMID: 35944472.

39. Wang B, Zhang L, Yu Y. Treatment of redo-microvascular decompression or internal neurolysis plus microvascular decompression for recurrent trigeminal neuralgia: a review of long-term effectiveness and safety. J Int Med Res. marca 2022;50(3):3000605221080721. doi:10.1177/03000605221080721 PubMed PMID: 35249412; PubMed Central PMCID: PMC8905060.

40. Cuenca-Zaldívar JN, Corral Del Villar C, García Torres S, Araujo Zamora R, Gragera Peña P, Padrós-Augé J, et al. Impact of climatic factors on trigeminal neuralgia and facial neuropathy in primary care during a fourteen-year time-series study. Sci Rep. 22 kwietnia 2026;16(1):18619. doi:10.1038/s41598-026-49760-0 PubMed PMID: 42020495; PubMed Central PMCID: PMC13270028.

41. Yoshizaki W, Fujikawa Y, Torikoshi S, Katayama T, Iwasaki K, Toda H. Effects of microvascular decompression on quality-of-life in trigeminal neuralgia patients aged 70 years and older. Surg Neurol Int. 3 lutego 2023;14:41. doi:10.25259/SNI_997_2022 PubMed PMID: 36895226; PubMed Central PMCID: PMC9990813.

42. Kc E, Islam J, Park YS. Trigeminal ganglion itself can be a viable target to manage trigeminal neuralgia. J Headache Pain. 24 listopada 2022;23(1):150. doi:10.1186/s10194-022-01512-x PubMed PMID: 36424545; PubMed Central PMCID: PMC9686102.