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Review

Trigeminal neuralgia

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Background
Trigeminal neuralgia (TN) is a chronic neuropathic pain disorder characterized by recurrent, severe, unilateral facial pain in the distribution of one or more branches of the trigeminal nerve.

Discussion
Although its prevalence is relatively low, TN remains one of the most debilitating pain syndromes due to its intensity and unpredictable nature. This review aims to summarize current knowledge on the pathophysiology, diagnostic criteria, and management of trigeminal neuralgia, including both pharmacological and surgical options.

Conclusions
Trigeminal neuralgia remains a challenging condition requiring an individualized, multidisciplinary approach.

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Introduction

Trigeminal neuralgia (TN) is a chronic neuropathic syndrome with recurrent, brief, severe and shock like attacks of facial pain. Is unilateral and runs on one or more trigeminal nerve branches (1).

Epidemiology

Prevalence in lifetime is 0.16-0.30%, most cases onset after 50 years and is more frequent in females with a ratio F:M 3:2 (2,3), this increased in prevalence and incidence among women may be possibly attributed to anatomical differences, particularly a smaller posterior fossa, neuroinflammatory responses, including arachnoiditis, variations in gonadal hormone levels and alterations in the functionality of endogenous pain modulation systems (4). About incidence is variable ranging between 2.1 and 27 cases per 100.000 person years and increases with age (4).

Clinical

TN has clinical characteristics and particular signs like trigger factors and refractory periods between paroxysms (5). The frequency and duration of attacks are variable; usually lasts from less than a second up to 2 min (74%) and a minority lasting 2-10 min. Up to 70% occasionally have paroxysms lasting up to 1 hour. The number of attacks is variable even in the same patients and ranges from a few to several hundred daily (5).

The pain is described as stabbing shock like, lancinating or burning; is unilateral in only one nerve branch in 60% of cases, while in 35% two branches commonly V2/3, V2/1. The ophthalmic branch is involved in only 4% and bilateral involvement is rare (1.7-5%) and is often related to secondary causes, such as multiple sclerosis or growing tumors of the cerebellopontine angle (2). The right side is affected more than the left side (60%) (3).

Pain can be spontaneous or provoked by stimulation of triggers points located in the nerve branch or branches involved (2). Around 91-99% of patients report trigger attacks, and these are often considered to be pathognomonic for trigeminal neuralgia (3). The most common triggers are the activation of masticatory muscles, tooth brushing, cold air and touching facial skin; light tactile stimulation is the most potent trigger, while painful and thermal stimulation are usually ineffective. These trigger points are frequently located in the nasal or perioral regions near the midline of the face and include the nasolabial fold, the upper lip, the lateral aspect of the lower lip, the chin, the cheek and the alveolar gingiva. In most people a triggered attack is normally followed by a period of seconds or minutes during which further attacks cannot be provoked, a phenomenon called refractory period (3).

Interestingly, painful attacks usually do not present during sleep and unlike other neuralgias, present a period of complete remission for last weeks or years in up to 63% of patients. Sensory deficits may be found in 30% of cases, however in most cases, they are subtle and detectable only with quantitative sensory testing (2). Adjunctive signs may occur during paroxysm; pain provokes brief muscle spasms of the facial muscles, thus producing the tic. Lacrimation, rhinorrhea, or redness of the face is very rare (1).

Exist two subtypes: purely paroxysmal or concomitant continuous pain, this last phenotype occurs in 14-50%; the importance of the differentiating these two subtypes is underlined by recent evidence suggesting that trigeminal neuralgia with concomitant continuous pain is pathophysiological different and respond less well to treatments compared with the purely paroxysmal form (3) Evolution of the disease over time may add concomitant basal pain in an original paroxysmal picture.

Classification

The TN can be idiopathic, classical and secondary or symptomatic, depending on the underlying cause (Figure 1).

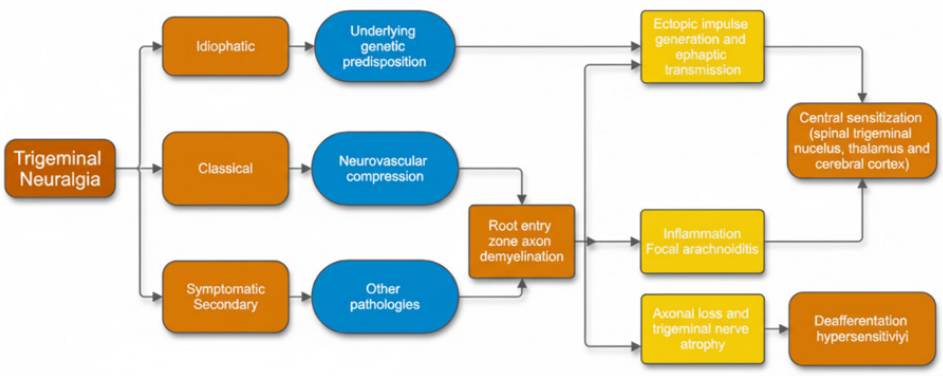

Figure 1. Classification and pathophysiology (Modified 4.)

The idiopathic type, accounting for approximately 10% of cases, is diagnosed when no apparent cause can be found. The classical type is the most common and accounts for 75% of cases, is diagnosed when there is trigeminal neurovascular compression with morphological changes ipsilateral to the side of the pain, demonstrated either on magnetic resonance imaging with appropriate trigeminal sequences or during surgery. Simple trigeminal contact without morphological changes (distortion, indentation, atrophy) is not sufficient to underpin such a diagnosis as this is a common neuroimaging finding in healthy people.

Finally, the secondary type, accounting 15% of cases, is attributable to an identifiable underlying neurological disease (except trigeminal neurovascular compression that is known to cause the neuralgia, such as cerebellopontine angle tumor, arteriovenous malformation and multiple sclerosis) (3).

Pathophysiology

The trigeminal nerve provides sensory innervation of the face and other structures in the head. It has three primary branches-the ophthalmic, maxillary and mandibular nerves- all plays a part in the transmission of facial sensations (4). A definite mechanism underlying remain unclear, yet evidence suggest that its development requires multiple factors influenced by genetic predisposition, particularly mutations in genes regulating membrane excitability, may predispose to a hyperexcitable neuronal state. Systematic reviews of patients with trigeminal neuralgia identified that various genes may have a role in the development of neuralgia, including SCN3A, TRAK1, NTRK1, GABRG1, SLC6A4, MAOA and MPZ; however, the implications of these findings for familiar cases still require further investigation. Particularly, mutation in ion channels, such as sodium and calcium channels, as well as gene polymorphisms affecting serotonin transporters and monoamine oxidase type A, have been highlighted, suggesting their possible role in neuronal excitability and plasticity in trigeminal neuralgia (4).

Anatomical changes, such as compression from a vascular loops or lesions at the trigeminal root entry zone can lead to focal demyelination, axonal loss and inflammation. These changes contribute to increased excitability of demyelinated nociceptive trigeminal axons, resulting in the generation of spontaneous ectopic impulses and ephaptic transmission between nociceptive and non-nociceptive fibers (4).

Ephaptic transmission between large myelinated, non-nociceptive afferents and nociceptive afferents may explain how innocuous stimuli can trigger painful paroxysms. That the most frequent trigger zones are perioral could be explained by the large number of afferents innervating this area (4).

Paroxysmal burst of ectopic activity arising from large diameter, non-nociceptive afferents may induce a secondary central dysfunction-repeated, abnormally high frequency activity in tactile afferents projecting to wide dynamic range neurons in the spinal trigeminal nucleus may increase their excitability and induce a persistent derangement that provokes high frequency signals from same neurons and thus pain. The perioral location of the trigger point would in this case be explained by the large representation of the perioral region in the spinal trigeminal nucleus, which makes it the most likely source of paroxysmal activity. In addition, trigger zones and pain sensation may be dissociated, a phenomenon related to cross-excitation between somatosensory afferents (4). To conclude, the primary cause of trigeminal neuralgia must necessarily affect the peripheral afferents, but the pathophysiological mechanism may or not secondarily involved the brainstem neurons (1).

Factors such as vascular compression or pathologies such as tumors, arteriovenous malformations and arachnoid or epidermoid cysts in the cerebellopontine area can lead to areas of focal demyelination in the root entry zone. Compression of the trigeminal nerve root (classical type) can lead to myelin erosion and disintegration due to inflammation, especially at the nerve indentation area. These changes result in pathological demyelination and remyelination, a common feature in peripheral nerve compression cases. Not all patients with neurovascular compression develop neuralgias whilst some patients with neuralgias have no identifiable etiology. This difference indicates that neurovascular compression is not the sole causative factor (4).

The nerve root entry zone is particularly susceptible to demyelination as a response to injury or compression owing to the transition from peripheral myelin (Schwann cell myelinated sheath) to central myelin (oligodendroglia generated myelin sheath). Demyelination induces dysregulation of voltage-gated ion channels, predominantly sodium channels, which in turn leads to their upregulation, thereby resulting in neuronal hyperexcitability, and ectopic discharges (4).

Diagnosis

Since TN is a clinical diagnosis, a detailed history and examination should be performed.

Brain MRI is crucial to exclude secondary causes of TN and identify neurovascular compression, using high-resolution 3D cisternae fast imaging, "FIESTA", T1 gadolinium, and MRI angiography. These techniques improve every year and should distinguish neurovascular compression or distortion of arteries or veins over the nerve, from simple vessel contact, which is quite common in this area.

If MRI is not possible, the second alternative is a brain CT scan with cerebral angiography, focusing more accurately on the posterior fossa.

Electrophysiological recording of the trigeminal reflex (more specific) and evoked potentials are complementary and may help to reveal sensory deficits.

Differential diagnosis

Pain in the face and the V1 territory are the main areas to challenge for the correct diagnosis.

Table 1. Differential diagnosis and clinical characteristics

Cause	Characteristic
Dental Causes	These are the first to consider, and having a reliable dental consultant available is recommended. Common causes include caries, pulpitis, dental fractures, pericoronitis, and alveolar osteitis. Night pain predominance is common in these cases
Sinus Causes Maxillary sinusitis	Dull sustained pain, worsening by movements of the head
Salivary Glands	Salivary gland stones
Temporomandibular Disorders	Trigger point on joints, tendons, myofascial pain
Other Neuralgias: Glossopharyngeal neuralgia Nervus intermedius neuralgia Post-herpetic neuralgia	Pain when swallowing, shouting, placing more focus in the throat, pillar of the pharynx Location more "otic", auriculotemporal Accompanied by allodynia, burning, itching, plus basal pain, not responding to sodium channel blockers
Painful trigeminal neuropathies	Continuous without remissions
Post-traumatic trigeminal neuropathy	Trauma, surgery, or dental procedures, constant pain, sensory loss, are common
Hemifacial Spasm	VII nerve compression, but sometimes is accompanied by trigeminal neuralgia
Burning mouth syndrome	Inside the mouth, not following V branches, aching, burning, sometimes accompanied by movement disorder as masticatory diskinesia
Trigeminal Autonomic Cephalalgias TACs	SUNCT/SUNA Cluster Headache Paroxysmal Hemicrania

SUNCT/SUNA and TN: Special considerations

SUNCT/SUNA and TN are considered different disorders by IHCD 3.

Innocuous tactile stimuli can provoke pain in trigeminal branches, especially V1 in TN, with the presence of autonomic symptoms that are common in SUNCT/SUNA (lacrimation, conjunctival injection). SUNCT/SUNA are described as having spontaneous stabs of pain with no refractory period between attacks, but some descriptions report cutaneous triggers in SUNCT (6). This can overlap in phenotype, suggesting some similarities or supporting a common etiological/pathophysiological basis, especially MRI findings that show neurovascular compression and resolution by surgery in some cases of SUNCT/SUNA (7). A series of 161 patients in an open-label study showed efficacy of sodium channel blockers, indicating another overlap between TN and SUNCT/SUNA (8).

Table 1. Differential diagnosis and clinical characteristics

	TN	SUNCT/SUNA	Cluster Headache (CH)
N Events	**1/100**	20/600	1/8
Autonomic	**++ (more in V1)**	++++	++++
Pain	**Stabs + basal pain**	Stabs++	Excruciating, long
Duration	**Seconds to 2 min**	1/600	
Location	**V2 > V3 > V1**	V1 > V2 > V3	Periorbital/ temporal/face

Multiple Sclerosis

Patients with MS have a 20-fold increased risk of developing TN (9). Around 1.9% to 4.9% of patients with MS suffer from this neuropathic pain condition, without differences between relapsing-remitting, secondary, and primary progressive forms. Conversely, MS is detected in 2% to 14% of patients with TN (10). The clinical picture is similar to typical TN, with attacks and possibly concomitant dull pain. Response to sodium channel blockers is similar, and neurovascular compression is also probable, complicating decisions about surgical management

Other secondary conditions

Lesions involving the cavernous sinus may produce trigeminal dull or aching pain. Extrasellar tumors can affect V1, including meningiomas, giant carotid aneurysms, and Tolosa-Hunt Syndrome. Pain is long-lasting, and other cranial nerve deficits are frequent: VI, III, IV nerve palsies, and hypoesthesia of the V territory are common.

Treatment

Pharmacological treatment remains the first-line management strategy for rescue therapy in exacerbations and prophylaxis. However, other alternatives may be considered depending on the clinical scenario. Drugs are grouped into first-line, second-line, and others. Monotherapy is preferred, although one-third of patients require combination therapy. After three unsuccessful drug trials, surgical treatments emerge as an opportunity when pain is refractory to drug treatment or in cases where adverse effects or tolerance limit it. These include percutaneous procedures, vascular decompression, and other techniques (11–13).

Pharmacological Management

Pharmacological therapy for trigeminal neuralgia includes two types of treatment: acute or rescue, and chronic or preventive (11).

Treatment of acute pain

Intravenous infusions of drugs such as fosphenytoin or phenytoin, lidocaine, and lacosamide may be effective in acute trigeminal neuralgia (12). In one study, complete pain relief was achieved in 78% of patients receiving lacosamide and 72% of patients receiving phenytoin, with no significant differences between groups (12,13). Opioids are ineffective for acute trigeminal neuralgia and should not be used (12).

Long-term preventive treatment

Anticonvulsants are the most effective drugs. Carbamazepine is the first-line agent in the treatment of classic trigeminal neuralgia (14–16). However, its prolonged use is associated with adverse effects (15).

Oxcarbazepine is comparable to carbamazepine in reducing the symptoms of classic trigeminal neuralgia and is associated with fewer side effects (12,16). Oxcarbazepine can be considered a first-line medication for secondary trigeminal neuralgia (15).

In patients with persistent symptoms or intolerance to carbamazepine and oxcarbazepine, second-line treatment may include baclofen, lamotrigine, gabapentin, and botulinum toxin type A, either as adjuvants or as monotherapy (12,17).

Baclofen may be effective in trigeminal neuralgia secondary to multiple sclerosis. A low-level study of baclofen and carbamazepine concluded that baclofen was slightly superior to carbamazepine alone, but the combination of both drugs was significantly superior (15).

Lamotrigine can be used in the management of secondary trigeminal neuralgia; however, it is important to note that it may aggravate the symptoms of multiple sclerosis. The level of evidence for this drug is low (15).

Gabapentin may be superior to carbamazepine in terms of efficacy and safety in patients with primary trigeminal neuralgia, but the evidence is insufficient (15,18,19).

A study with high-grade evidence reported that ropivacaine (blockade) combined with gabapentin (oral administration) reduced pain and improved quality of life in patients with trigeminal neuralgia. The combination of ropivacaine and carbamazepine was also found to reduce the side effects and limitations of carbamazepine (15).

Intradermal or submucosal administration of botulinum toxin type A has demonstrated effective analgesia in patients with trigeminal neuralgia and could be used as therapy (15,20). A systematic review concluded that botulinum toxin type A is an effective medication and has fewer adverse effects than carbamazepine and oxcarbazepine, and can therefore be recommended as

an important therapeutic option (19,21).

Second- and third-generation anticonvulsant medications, such as topiramate, eslicarbazepine, retigabine, and lacosamide, are promising, but their evidence base is still lacking (11). There is also insufficient evidence on the efficacy of drugs such as levetiracetam, phenytoin, and pregabalin in trigeminal neuralgia; however, one study suggests that pregabalin may be effective in combination with carbamazepine in refractory cases (15). Vixotrigine, a selective sodium channel blocker, is currently undergoing Phase 3 research (13).

Regarding calcitonin gene-related peptide antagonists (gepants, monoclonal antibodies), their efficacy in the treatment of trigeminal neuralgia has not been demonstrated. The role of cannabinoids has not been established, despite their positive results in other types of neuropathic pain.

Additionally, there are studies on the use of sumatriptan and intranasal lidocaine, which indicate significant improvement in analgesia in patients with trigeminal neuralgia (TN), however, these were low-grade (22).

Other drugs of interest include lasmiditan, pimozide, and tizanidine, but there is no solid research supporting their use (11). On the other hand, drugs such as proparacaine, dextromethorphan, and tocainide are considered inappropriate for the treatment of trigeminal neuralgia (15).

Concomitant Continuous Pain

In cases where TN coexists with ongoing pain mediated by other pathophysiological mechanisms, monotherapy with sodium channel blockers is not sufficient, so the addition of other types of drugs is usually required for pain control. Calcium channel blockers and antidepressants are effective in the treatment of neuropathic pain from various causes; however, they have not been fully studied in patients with trigeminal neuralgia (22).

Table 3. Summarizes the main current lines of preventive pharmacological treatment for trigeminal neuralgia

TN	First Line	Second Line (monotherapy or adjuvant)	Other Adjuvants
Idiopathic	Carbamazepine Oxcarbazepine	Baclofen Lamotrigine Botulinum toxin type A	Gabapentin Ropivacaine Pregabalin Topiramate Antidepressants and calcium channel blockers (Continuous concomitant pain)
Classical			
Secondary	First-line drug therapy + Treatment of the cause		

Interventional treatment

Criteria for considering

Interventional treatment is indicated when, after using at least two different drugs at adequate doses and for a sufficient duration (one of them carbamazepine or oxcarbazepine), symptom relief is insufficient or the adverse effects are unacceptable. In this context, the patient is referred to neurosurgery for continued management and planning of preventive surgical options. If there is vascular compression of the trigeminal nerve and there are no contraindications, microvascular decompression (MVD) is considered the first line of surgery. If there is no significant compression, neuroablative procedures or stereotactic radiosurgery are considered.

Factors associated with better outcome: single arterial compression and age >60 years; venous or multiple compression predict worse outcome (23).

Current Interventional Options

1. Surgical Microvascular Decompression (MVD)
2. Radiofrequency thermolesion
3. Percutaneous Balloon Microcompression
4. Stereotactic Radiosurgery (Gamma Knife)
5. Percutaneous Glycerol Rhizolysis

Surgical Microvascular Decompression

Objective: To relieve vascular pressure on the trigeminal nerve by retrosigmoid craniotomy, interposing a Teflon seal between the vessel and the nerve. Rare complications: infection, hearing loss, facial hypoesthesia, cerebrospinal fluid (CSF) leak, cerebrovascular accident (CVA), hemorrhage. Results: Immediate relief in 80–90% of patients; 10-year recurrence rate in 15–25% (24). It is the preferred procedure in patients with evident vascular compression due to its sustained efficacy and low complication rate (25).

Radiofrequency thermocoagulation

This consists of selective thermal radiofrequency ablation of the affected trigeminal nerve divisions.

Results: immediate pain relief in 79–97%; recurrence in 11% at the first year and up to 42% at 5 years.

Complications: decreased corneal reflex, masseter weakness, dysesthesias, keratitis.

Percutaneous balloon microcompression

Percutaneous technique under general anesthesia that compresses the Gasserian ganglion with a balloon, producing ischemic damage. Particularly useful for pain in the V1 territory. Some comparisons suggest similar

efficacy to radiofrequency but with less dysesthesia.

Stereotactic Radiosurgery (Gamma Knife)

Noninvasive method consisting of the administration of a single dose of radiation to the affected nerve. Advantages: no mortality, low rate of sensory disturbance. Initial results: 60–70% pain reduction. Most common complication: appearance of facial sensory symptoms due to partial nerve injury (26).

Percutaneous glycerol rhizotomy

Chemical neurolysis using glycerol injection into the trigeminal cistern under fluoroscopic guidance.

In a large series of 3,370 patients, Xu et al. (27) reported pain relief in 73% of patients after one injection, with an overall success rate of 99.5% after four injections. The pain recurrence rate throughout the study was 33% at 5 years.

Complications: hyperesthesia, hypoalgesia, facial dysesthesia, ocular complications.

Other treatments and adjuvants

- Acupuncture: Very low evidence; may reduce pain and adverse events compared to carbamazepine, but is not formally recommended (28).

- Botulinum toxin type A: Reviews show significant reduction in pain intensity and frequency, with an effect lasting at least 3 months; weak recommendation (29).

- Low-level laser therapy: Clinical reports and animal models show analgesia without serious side effects; mechanism by modulation of inflammatory mediators and increased endorphins (28).

General recommendations

MVD is preferred over ablative procedures in classic trigeminal neuralgia when there is no evidence of neurovascular compression.

Ablative treatments are preferred if there is no neurovascular compression. Furthermore, the patient should be individualized according to age, comorbidities, vascular anatomy, and patient preferences (19,30).

Table 4. Interventional Options: Summary

Option	Procedure	Complications	Effectiveness
Surgical Microvascular decompression	Relieves pressure from a pulsating vessel on the trigeminal nerve	Infections, hearing loss, CSF leak, stroke (rare)	Immediate pain relief in 80-90%; recurrence in 15-25% at 10 years
Radiofrequency thermolesion	Selective radiofrequency thermal ablation of the affected trigeminal nerve divisions.	Decreased corneal reflex, masseter weakness, dysesthesia, keratitis.	High percentage of immediate pain relief.
Stereotactic radiosurgery	Irradiates a section of the nerve at high doses	Facial sensory symptoms.	Pain reduction by 60-70%
Balloon microcompression	Compress and injure the nerve with a small balloon	Temporary masticatory weakness, facial numbness.	Similar to other percutaneous techniques.
Rhizolysis with glycerol	Inject glycerol into the trigeminal cistern	Facial hypoalgesia, dysesthesia.	Initial relief 70-90%, recurrence in 20-40%.

Other options to consider in patients refractory to pharmacological or surgical treatment, although only available in case reports or case series, include topical lidocaine on the oral mucosa, lidocaine patches, intranasal lidocaine, sumatriptan (oral or subcutaneous), or local terminal branch blocks in affected areas. When neuralgia recurs after a surgical procedure, repeating the procedure or using another technique is considered. There are series of up to three interventions, mainly with radiofrequency lesions or microvascular decompression (31,32).

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