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Trigeminal post-herpetic neuropathy: Epidemiology, pathophysiology, clinical classification, diagnosis, management, and future perspectives—A narrative review

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Abstract
Background: Trigeminal post-herpetic neuropathy (TG-PHN) is a chronic neuropathic pain disorder that may follow reactivation of varicella-zoster virus within the trigeminal sensory system. It is particularly important when the ophthalmic division is involved because persistent facial pain and sensory dysfunction may coexist with ocular morbidity.
Objective: This narrative review summarizes current evidence regarding the epidemiology, risk factors, pathophysiology, clinical manifestations, diagnosis, management, prevention, prognosis, and future research priorities of TG-PHN.
Methods: A narrative review of peer-reviewed literature was undertaken using PubMed/MEDLINE, Embase, Scopus, Web of Science, and Google Scholar. The review emphasized literature published from January 2021 through June 2026, while earlier landmark studies and established classification documents were retained when necessary. Trigeminal-specific evidence was prioritized, with broader post-herpetic neuralgia literature included selectively when clinically applicable.
Results: TG-PHN is characterized by persistent or recurrent neuropathic pain and sensory abnormalities within trigeminal distributions previously affected by herpes zoster. Advanced age, severe acute zoster pain, greater rash severity, prodromal pain, impaired immunity, and selected comorbidities are associated with increased risk of persistent post-herpetic pain. Peripheral nerve injury, neuroinflammation, altered nociceptor excitability, deafferentation, and central sensitization contribute to persistent symptoms. Diagnosis remains primarily clinical. Management is individualized and may include gabapentinoids, tricyclic antidepressants, topical therapies, and selected interventional approaches for refractory disease. Prevention through appropriate management of acute herpes zoster and recombinant zoster vaccination remains important.
Conclusion: TG-PHN is a heterogeneous and potentially disabling neuropathic disorder requiring individualized multimodal management. Trigeminal-specific evidence remains limited, particularly for interventional therapies. Larger prospective studies and well-designed clinical trials are needed to better define disease phenotypes, natural history, treatment response, and long-term outcomes.

Keywords: Trigeminal post-herpetic neuropathy, herpes zoster, post-herpetic neuralgia, Varicella-Zoster Virus, herpes zoster ophthalmicus, facial neuropathic pain, central sensitization, neuroinflammation, gabapentinoids, botulinum toxin, zoster vaccination

Introduction

Abbreviations

Abbreviation	Definition
TG-PHN	Trigeminal Post-Herpetic Neuropathy
PHN	Post-Herpetic Neuralgia
HZ	Herpes Zoster
HZO	Herpes Zoster Ophthalmicus
VZV	Varicella-Zoster Virus
V1	Ophthalmic Division
V2	Maxillary Division
V3	Mandibular Division
QST	Quantitative Sensory Testing
MRI	Magnetic Resonance Imaging
BTX-A	Botulinum Toxin Type A
PRF	Pulsed Radiofrequency
TCA	Tricyclic Antidepressant
ICHD-3	International Classification of Headache Disorders, 3rd Edition
QoL	Quality of Life

Varicella-zoster virus (VZV) is a neurotropic human alphaherpesvirus that establishes lifelong latency within sensory ganglia after primary varicella infection. Reactivation of latent VZV produces herpes zoster (HZ), typically characterized by a painful unilateral vesicular eruption in a sensory distribution. The incidence of HZ increases substantially with advancing age and immunosuppression, reflecting progressive decline in VZV-specific cell-mediated immunity [1, 2].

Cranial involvement is clinically important because VZV may reactivate within the trigeminal ganglion and affect one or more divisions of the trigeminal nerve. The ophthalmic division (V1) is involved most frequently and produces herpes zoster ophthalmicus (HZO), whereas maxillary (V2) and mandibular (V3) involvement may produce neuropathic pain affecting the cheek, oral cavity, dentition, and jaw [2, 3]. Trigeminal disease is particularly important because persistent pain may coexist with sensory dysfunction and, in ophthalmic disease, potentially serious ocular complications [3].

Post-herpetic neuralgia (PHN) is the most recognized chronic pain complication of HZ. Its reported frequency varies according to patient age, study population, follow-up duration, and the definition used, but the risk rises markedly with older age and severe acute zoster [1, 4]. The International Classification of Headache Disorders, third edition (ICHD-3), defines post-herpetic trigeminal neuralgia as unilateral facial pain persisting or recurring for more than 3 months in

the distribution of one or more trigeminal branches affected by HZ [5].

The clinical disorder, however, extends beyond pain alone. Patients may develop burning or aching spontaneous pain, superimposed paroxysms, mechanical allodynia, hyperalgesia, hypoesthesia, pruritus, and other dysesthesias, reflecting injury and dysfunction within the trigeminal sensory system [3, 5]. For this review, the term trigeminal post-herpetic neuropathy (TG-PHN) is used to emphasize the broader neuropathic process while recognizing that "post-herpetic trigeminal neuralgia" remains an established diagnostic term in current classification systems [5, 6].

TG-PHN can substantially impair daily function and quality of life. Facial allodynia may interfere with washing, shaving, eating, speaking, dental care, sleep, and social interaction, while treatment is often complicated by incomplete analgesia and dose-limiting adverse effects, particularly in older adults [3]. Management therefore requires an individualized approach that integrates prevention, appropriate treatment of acute HZ, established neuropathic-pain therapies, and selected interventional strategies for refractory disease.

Despite advances in the understanding and treatment of neuropathic pain, trigeminal-specific evidence remains comparatively limited. Much of the pharmacological evidence is extrapolated from PHN involving mixed anatomical distributions, whereas studies of trigeminal interventions are generally smaller and methodologically heterogeneous [3]. This narrative review synthesizes current evidence on the epidemiology, pathophysiology, clinical classification and manifestations, diagnosis and differential diagnosis, contemporary management, prognosis, prevention, and future directions of TG-PHN, with emphasis on clinically relevant evidence and areas in which further trigeminal-specific research is needed.

Methods

This article was developed as a narrative review of the contemporary literature on trigeminal post-herpetic neuropathy. Relevant peer-reviewed literature was identified through searches of PubMed/MEDLINE, Embase, Scopus, Web of Science, and Google Scholar. The review emphasized publications from January 2021 through June 2026, while earlier landmark studies and established classification documents were retained when they provided foundational evidence not adequately replaced by newer literature. Trigeminal-specific evidence was preferred whenever available, and evidence from post-herpetic neuralgia involving other anatomical distributions was included selectively when it addressed broadly accepted mechanisms or treatments applicable to trigeminal disease.

Search concepts included varicella-zoster virus, herpes zoster, herpes zoster ophthalmicus, trigeminal post-herpetic neuralgia, trigeminal neuropathy, facial neuropathic pain, post-herpetic neuralgia, diagnosis, pharmacological treatment, interventional pain management, neuromodulation, and vaccination. Reference lists of relevant reviews and major studies were also examined for additional eligible publications.

Priority was given to systematic reviews, meta-analyses, randomized controlled trials, observational studies, clinical guidelines, consensus or classification documents, and high-quality reviews directly relevant to TG-PHN. Trigeminal-specific evidence was preferred whenever available. Evidence from PHN involving other anatomical distributions was included selectively when it addressed broadly accepted mechanisms or treatments applicable to trigeminal disease. Publications without clear relevance to TG-PHN, non-peer-reviewed reports, conference abstracts without full publications, duplicate reports, and studies focused exclusively on unrelated facial-pain disorders were not used as principal evidence.

Evidence was synthesized thematically rather than quantitatively. No meta-analysis was performed. Because this is a narrative review, the literature search and study selection were intended to provide a focused critical synthesis rather than the exhaustive reproducibility required of a systematic review. Particular attention was given to the strength, clinical applicability, and trigeminal specificity of the evidence, and emerging or investigational approaches were distinguished from established clinical practice.

Epidemiology and Risk Factors

Herpes zoster is a common consequence of varicella-zoster virus reactivation, with incidence increasing substantially with advancing age and impaired cell-mediated immunity [1, 2]. Although post-herpetic neuralgia (PHN) may follow herpes zoster at any anatomical site, trigeminal involvement is clinically important because of the functional significance of the face and the additional ophthalmic morbidity associated with involvement of the ophthalmic division (V1) [3]. Among trigeminal presentations, herpes zoster ophthalmicus represents the most clinically consequential distribution and is an important setting in which persistent trigeminal neuropathic pain may develop [3].

The frequency of PHN varies considerably across studies because of differences in population age, immune status, vaccination status, follow-up duration, and the definition used for persistent pain [1, 4]. This heterogeneity is particularly relevant to TG-PHN, for which large population-based studies using uniform diagnostic criteria remain limited. Accordingly, precise estimates of the incidence and prevalence of TG-PHN should be interpreted cautiously rather than extrapolated directly from studies of PHN involving all dermatomal distributions [3].

Advanced age is the most consistently recognized risk factor for PHN [1, 4, 7]. Immunosenescence progressively reduces VZV-specific cellular immunity, increasing susceptibility to viral reactivation and persistent neuropathic complications. Contemporary evidence further demonstrates a graded age effect: a recent systematic review and meta-analysis found substantially increased PHN risk among adults aged 60–69 years and an even greater risk among those aged 70 years or older [7].

Characteristics of the acute herpes zoster episode also influence the subsequent risk of persistent neuropathic pain. Severe acute pain and greater rash severity are consistently associated with PHN, while prodromal pain has also been identified as a risk factor in pooled analyses [7, 8]. These associations support the concept that greater acute neural and inflammatory injury may predispose susceptible patients to persistent peripheral and central nociceptive dysfunction.

Comorbid disease and impaired host immunity further modify risk. Diabetes mellitus and malignancy have been associated with an increased likelihood of PHN, while immunosuppression is also recognized as an important clinical risk factor [7, 8]. More recent pooled analyses have

reported associations with chronic kidney disease, chronic obstructive pulmonary disease, and hypertension; however, these findings are less established than the associations with advanced age and severe acute zoster and should be interpreted cautiously [7, 8]. Evidence regarding female sex as an independent predictor remains inconsistent [7].

For TG-PHN specifically, ophthalmic involvement deserves particular attention. V1 disease may be accompanied by ocular inflammation and corneal sensory abnormalities in addition to persistent neuropathic pain, increasing the potential clinical burden of trigeminal zoster [3]. Nevertheless, the available literature remains substantially stronger for PHN overall than for individual trigeminal divisions, and risk estimates derived from general PHN populations should not automatically be considered trigeminal-specific.

The epidemiology of TG-PHN is also increasingly influenced by prevention. Because persistent post-herpetic neuropathic pain occurs downstream of VZV reactivation, prevention of herpes zoster through vaccination has the potential to reduce the burden of both HZ and its chronic pain complications. The preventive role of recombinant zoster vaccination is therefore discussed separately in the management and prevention section of this review.

Overall, TG-PHN should be regarded as a clinically important subset of post-herpetic neuropathic pain in which advanced age, severe acute zoster manifestations, impaired immunity, and selected comorbidities contribute to risk [1, 3, 4, 7, 8]. The relative scarcity of large trigeminal-specific epidemiological studies remains an important evidence gap and limits precise estimates of incidence, natural history, and division-specific risk.

Pathophysiology

The pathophysiology of TG-PHN begins with reactivation of latent VZV within the trigeminal ganglion. Viral replication and the accompanying inflammatory response produce injury to sensory neurons and their peripheral and central projections. The resulting neuropathic state reflects a combination of neuronal injury, neuroinflammation, altered nociceptor excitability, peripheral sensitization, and changes in central pain processing [3].

Trigeminal Ganglion and Peripheral Nerve Injury

During trigeminal herpes zoster, VZV reactivation within ganglionic neurons produces inflammation and neuronal damage that may extend along affected trigeminal fibers. Degeneration and loss of sensory fibers can produce hypoesthesia or numbness, whereas surviving or injured afferents may become hyperexcitable and generate spontaneous or exaggerated pain signals [3]. This combination helps explain why patients may experience sensory loss and severe pain within the same trigeminal distribution.

Peripheral inflammation further alters nociceptive signaling. Cytokines and other inflammatory mediators released during VZV reactivation can increase the excitability of sensory neurons and contribute to persistent nociceptor sensitization [3]. Changes involving transient receptor potential channels, purinergic receptors, voltage-gated ion channels, and neuropeptides such as calcitonin gene-related peptide and substance P have also been implicated in the amplification of peripheral pain signaling [3].

Peripheral and Central Sensitization

Persistent activity in damaged trigeminal afferents can increase the responsiveness of second-order neurons within the trigeminal brainstem sensory complex. This process contributes to central sensitization, in which normally innocuous sensory input may be perceived as painful and painful stimuli may evoke exaggerated responses [3]. Clinically, these changes are expressed as mechanical allodynia, hyperalgesia, spontaneous burning pain, and superimposed paroxysmal pain.

Altered inhibitory control may further sustain the neuropathic state. Changes in excitatory and inhibitory neurotransmission within the trigeminal brainstem pathways, thalamus, and higher pain-processing regions can amplify nociceptive transmission even after the acute cutaneous infection has resolved [3]. These central changes help explain why persistent pain may continue in the absence of active skin lesions.

Deafferentation and Sensory Loss

Structural damage to trigeminal sensory fibers may also result in partial deafferentation. Loss of normal sensory input can coexist with abnormal spontaneous activity from injured neurons and altered processing within central nociceptive pathways [3]. Consequently, TG-PHN may present with apparently contrasting findings, including numbness accompanied by burning pain, allodynia within areas of reduced sensation, or intermittent electric-like exacerbations superimposed on continuous discomfort.

Neuroimmune Mechanisms

Neuroimmune interactions contribute to both the acute inflammatory response and subsequent alterations in pain processing. Activation of immune cells and glial elements can promote the release of inflammatory mediators that modify neuronal excitability and synaptic transmission [3] Although these mechanisms provide potential targets for future therapy, many molecular pathways identified in experimental models have not yet translated into established TG-PHN-specific treatments.

Integrated Mechanism of Persistent Pain

TG-PHN is therefore best understood as the consequence of interacting peripheral and central mechanisms rather than a single pathological process. VZV-induced trigeminal ganglion injury initiates inflammation and sensory-neuron damage, peripheral sensitization increases nociceptive signaling, deafferentation alters normal sensory input, and central sensitization amplifies or sustains pain transmission [3] The relative contribution of these mechanisms varies among patients and contributes to the heterogeneous clinical presentation of TG-PHN.

This pathophysiological heterogeneity also explains why no single treatment is universally effective. Patients dominated by spontaneous neuropathic pain, mechanical allodynia, sensory loss, or mixed sensory abnormalities may respond differently to pharmacological, topical, and interventional therapies. Understanding these mechanisms therefore provides a useful clinical framework for the manifestations and treatment approaches discussed in subsequent sections.

Clinical Classification and Manifestations

TG-PHN is characterized by persistent or recurrent unilateral facial pain within one or more trigeminal nerve

distributions previously affected by herpes zoster. Under ICHD-3 and the International Classification of Orofacial Pain, post-herpetic trigeminal neuralgia is distinguished from acute painful trigeminal neuropathy by persistence or recurrence of pain for more than 3 months after the herpes zoster infection [5, 6].

Clinical Course

Trigeminal pain associated with herpes zoster evolves across the acute and chronic phases of infection. During the acute phase, pain may precede or accompany the characteristic vesicular eruption and is usually confined to the affected trigeminal distribution. In some patients, neuropathic symptoms persist after resolution of the cutaneous lesions and evolve into established TG-PHN [3, 5].

Pain may be continuous or intermittent and is commonly described as burning, aching, stabbing, lancinating, or piercing. Pruritus can be prominent and may coexist with pain. Paresthesia and dysesthesia are also common, and some patients experience brief paroxysmal exacerbations superimposed on continuous background discomfort [3, 5].

Sensory Abnormalities

Sensory dysfunction is an important clinical feature of TG-PHN. Patients may demonstrate hypoesthesia, numbness, mechanical allodynia, hyperalgesia, or increased sensitivity to thermal or punctate stimuli within the affected trigeminal territory [3, 5]. The coexistence of sensory loss and pain reflects the underlying neuropathic nature of the disorder.

Mechanical allodynia may be particularly disabling because normally innocuous activities such as touching the face, washing, shaving, applying cosmetics, exposure to clothing or bedding, eating, and exposure to wind may provoke pain. Sensory findings vary among patients, and not every individual demonstrates the same combination of positive and negative sensory abnormalities [3].

Trigeminal Distribution

The ophthalmic division (V1) is the trigeminal branch most commonly affected in TG-PHN [3, 5]. Pain and sensory abnormalities may involve the forehead, scalp, upper eyelid, and periocular region. Patients with previous herpes zoster ophthalmicus may also have persistent ocular symptoms or complications requiring ophthalmological follow-up.

Maxillary division (V2) involvement produces symptoms involving the cheek, lateral nose, upper lip, upper dentition, and maxillary region. Mandibular division (V3) involvement may affect the lower face, lower lip, mandibular dentition, jaw, and portions of the oral cavity [3]. More than one trigeminal division may occasionally be involved.

Functional and Psychosocial Impact

Persistent trigeminal neuropathic pain can interfere substantially with daily activities. Depending on the affected distribution and severity, patients may experience difficulty with eating, speaking, facial hygiene, shaving, dental care, sleep, and other routine activities [3]. Persistent pain and sleep disruption may also contribute to fatigue, reduced social participation, anxiety, depressive symptoms, and impaired quality of life.

The clinical presentation of TG-PHN is therefore heterogeneous. Some patients predominantly experience continuous burning pain, whereas others have marked allodynia, sensory loss, pruritus, dysesthesia, or intermittent paroxysmal pain. Recognition of these different manifestations is important because TG-PHN may otherwise be confused with classical trigeminal neuralgia, dental disease, temporomandibular disorders, persistent idiopathic facial pain, or other causes of craniofacial pain.

Distinction from Classical Trigeminal Neuralgia

TG-PHN and classical trigeminal neuralgia are clinically distinct disorders. Classical trigeminal neuralgia is characterized predominantly by recurrent, brief, electric shock-like facial pain precipitated by innocuous stimuli within the trigeminal distribution. In painful trigeminal neuropathy, including TG-PHN, pain is more commonly continuous or near-continuous, often accompanied by broader areas of allodynia and clinically detectable sensory abnormalities [5].

The preceding history of herpes zoster in the corresponding trigeminal distribution, together with persistent neuropathic pain and associated sensory changes, therefore provides the principal clinical context for distinguishing TG-PHN from classical trigeminal neuralgia [3, 5].

Diagnosis and Differential Diagnosis

The diagnosis of TG-PHN is primarily clinical and is based on a compatible history of herpes zoster involving the trigeminal nerve followed by persistent or recurrent neuropathic pain in the corresponding trigeminal distribution. According to ICHD-3 criteria, post-herpetic trigeminal neuralgia is characterized by unilateral facial pain persisting or recurring for more than 3 months in the distribution of one or more trigeminal nerve branches previously affected by herpes zoster [5]. The temporal relationship between the preceding zoster episode, anatomical distribution of pain, and associated sensory abnormalities is central to the diagnosis [3, 5].

Clinical Evaluation

A detailed history should establish the location and characteristics of the preceding herpes zoster eruption, the trigeminal division involved, onset and duration of pain, pain quality, provoking factors, associated pruritus or dysesthesia, previous treatments, and the effect of symptoms on daily activities. Pain may be continuous or intermittent and may coexist with mechanical allodynia, hyperalgesia, hypoesthesia, numbness, or other sensory abnormalities [3].

Neurological examination should include comparison of facial sensation across the ophthalmic, maxillary, and mandibular divisions of the trigeminal nerve. Light touch, pinprick, and temperature sensation may identify areas of sensory loss or gain. Mechanical allodynia can be assessed using gentle non-painful stimulation. Corneal sensation and ocular findings require particular attention in patients with previous ophthalmic zoster, especially when persistent ocular symptoms are present [3].

Diagnostic Investigations

Routine laboratory testing or neuroimaging is generally unnecessary when the clinical history and examination are characteristic of TG-PHN [3]. Additional investigation should be guided by atypical features or diagnostic uncertainty.

Magnetic resonance imaging may be considered when pain has an unusual distribution, neurological deficits extend

beyond the affected trigeminal territory, symptoms are progressive, multiple cranial nerves are involved, or another structural neurological disorder is suspected. Quantitative sensory testing and neurophysiological studies may provide additional characterization of trigeminal sensory dysfunction in selected patients, although these investigations are not routinely required to establish the diagnosis.

Virological testing is usually unnecessary after a well-documented episode of trigeminal herpes zoster. Polymerase chain reaction or other VZV-specific investigations may become relevant when VZV reactivation is suspected without the characteristic rash, when neurological complications are present, or when the diagnosis remains uncertain.

Differential Diagnosis

Classical trigeminal neuralgia is an important differential diagnosis. It typically produces recurrent, brief, electric shock-like attacks within one or more trigeminal divisions and may be triggered by innocuous facial stimulation. TG-PHN more commonly produces persistent or near-continuous neuropathic pain accompanied by sensory abnormalities and a history of herpes zoster affecting the same trigeminal territory [5].

Painful post-traumatic trigeminal neuropathy should be considered when facial pain and sensory disturbance follow dental procedures, facial trauma, maxillofacial surgery, or other identifiable trigeminal nerve injury. The temporal relationship to trauma rather than herpes zoster usually distinguishes this condition from TG-PHN [6].

Dental and oral disorders may mimic V2 or V3 TG-PHN. Pulpal disease, periodontal infection, cracked teeth, temporomandibular disorders, and other odontogenic conditions should therefore be considered when pain predominantly involves the maxillary or mandibular region. Dental examination and appropriate imaging may be required when the clinical findings suggest an odontogenic source.

Persistent idiopathic facial pain is generally characterized by persistent facial or oral pain without a preceding neurological lesion that adequately explains the symptoms. Its distribution may be poorly localized and does not necessarily follow the anatomical boundaries of the trigeminal nerve [6].

Other differential diagnoses include migraine and other primary headache disorders, temporomandibular disorders, sinus disease, giant cell arteritis in an appropriate clinical setting, neoplastic or inflammatory trigeminal neuropathies, multiple sclerosis, and other structural lesions affecting the trigeminal pathway. The presence of progressive sensory loss, motor abnormalities, bilateral symptoms, multiple cranial neuropathies, systemic manifestations, or other neurological deficits should prompt evaluation for an alternative or additional diagnosis.

In patients with previous herpes zoster ophthalmicus, persistent or recurrent ocular pain should not automatically be attributed to TG-PHN. Keratitis, uveitis, elevated intraocular pressure, neurotrophic keratopathy, and other ocular complications may coexist with or mimic neuropathic pain and warrant ophthalmological assessment when clinically suspected [3].

Overall, TG-PHN remains principally a clinical diagnosis established by the characteristic relationship between previous trigeminal herpes zoster, persistent neuropathic pain in the same anatomical distribution, and compatible sensory abnormalities [3, 5]. Additional investigations are reserved primarily for atypical presentations, suspected complications, or situations in which an alternative cause of facial pain must be excluded.

Management

Management of TG-PHN is directed toward reducing neuropathic pain, improving sleep and daily function, minimizing treatment-related adverse effects, and preserving quality of life. Because no single therapy is consistently effective, treatment is individualized according to pain severity and phenotype, trigeminal distribution, patient age, comorbidities, previous treatment response, and medication tolerability [3].

Pharmacological Management

Gabapentin and pregabalin are commonly used first-line systemic therapies for PHN and are also used in TG-PHN [3, 9]. Both act through the α2δ subunit of voltage-gated calcium channels and reduce excitatory neurotransmitter release. Dizziness, somnolence, peripheral edema, and gait instability may limit treatment, particularly in older adults, and dosage adjustment is required in renal impairment.

Tricyclic antidepressants, particularly amitriptyline and nortriptyline, are established treatments for neuropathic pain and may be considered in selected patients with TG-PHN [9]. Their use may be limited by sedation, anticholinergic effects, orthostatic hypotension, and cardiac adverse effects, especially in older adults. Treatment should therefore be individualized according to comorbidity and tolerability.

When monotherapy provides inadequate relief, combination treatment using agents with complementary mechanisms may be considered.[3] Dose escalation should be balanced against adverse effects, and treatment goals should include improvement in function and sleep in addition to reduction in pain intensity.

Topical Therapy

Topical lidocaine provides a treatment option for localized peripheral neuropathic pain and may be useful when allodynia is confined to a relatively small facial region [3, 9]. Its limited systemic absorption can be advantageous in older patients or those receiving multiple medications. Application near the eye or mucosal surfaces requires appropriate caution.

High-concentration capsaicin is another established topical treatment for PHN and has also been used in trigeminal disease.[3] Treatment produces prolonged nociceptor desensitization but may cause substantial local burning and irritation during application. Facial use requires particular care because of proximity to the eyes and other sensitive structures.

Interventional Management

Interventional treatment may be considered when pain remains disabling despite appropriately selected conservative therapy. Procedures described for TG-PHN include peripheral trigeminal nerve blocks, botulinum toxin injections, pulsed radiofrequency, and peripheral nerve stimulation [3] The choice of intervention depends on the affected trigeminal division, pain distribution, previous treatment response, procedural risk, and local expertise.

Peripheral nerve blocks targeting the supraorbital, infraorbital, auriculotemporal, or mental nerves may provide temporary pain relief in selected patients [3] Deeper interventions involving the maxillary or mandibular divisions or the Gasserian ganglion have also been described. These procedures should be performed by clinicians familiar with trigeminal anatomy and image-guided pain interventions when appropriate.

Botulinum toxin type A has demonstrated analgesic benefit in studies of PHN and may be considered for localized refractory neuropathic pain.[3] Injection technique, dose, and treatment interval vary among studies, and trigeminal-specific evidence remains more limited than the broader PHN literature.

Pulsed radiofrequency has been investigated at peripheral trigeminal branches and the Gasserian ganglion. Unlike destructive continuous radiofrequency techniques, pulsed radiofrequency is intended to modulate nociceptive signaling while limiting thermal neural injury. It may be considered in selected patients with refractory trigeminal zoster-related pain, although treatment protocols and long-term outcomes remain variable [3].

Peripheral nerve stimulation and other neuromodulatory approaches have also been used for medically refractory TG-PHN [3]. These techniques are generally reserved for carefully selected patients after failure of less invasive therapies because the available evidence consists largely of small clinical series and specialized-center experience.

Multimodal Management

TG-PHN frequently requires a multimodal approach combining pharmacological, topical, and, when necessary, interventional treatment [3]. Management should also address sleep disturbance, mood symptoms, functional limitations, and the consequences of persistent facial allodynia. Patients with previous ophthalmic zoster and ongoing ocular symptoms require appropriate ophthalmological assessment rather than assuming that all persistent periocular pain represents neuropathic pain alone.

Treatment response should be reassessed periodically, with ineffective medications discontinued and unnecessary polypharmacy avoided. In older adults, particular attention should be given to sedation, cognitive effects, falls, renal function, drug interactions, and anticholinergic burden.

Overall, management should progress from appropriately selected conservative therapies toward targeted interventions when pain remains refractory. The therapeutic objective is not necessarily complete elimination of pain but meaningful improvement in pain burden, function, sleep, and quality of life [3, 9].

Prevention

Prevention of TG-PHN is closely linked to prevention and appropriate early management of herpes zoster. Because persistent trigeminal neuropathic pain develops following VZV reactivation and neural injury, strategies that reduce the occurrence or severity of herpes zoster may also reduce the subsequent burden of PHN [10].

Early Management of Herpes Zoster

Antiviral therapy is recommended during acute herpes zoster to limit viral replication, accelerate lesion resolution, and reduce the severity and duration of acute symptoms. Acyclovir, valacyclovir, and famciclovir are commonly used, with treatment ideally initiated within 72 hours of rash onset [10]. Treatment may still be appropriate beyond this period when new lesions continue to develop or when patients have ophthalmic, neurological, or immunocompromising conditions.

Early recognition is particularly important in trigeminal zoster involving the ophthalmic division because of the potential for ocular complications. Patients with suspected ocular involvement require prompt antiviral treatment and ophthalmological assessment when indicated [3, 10].

Although antiviral therapy is important in the management of acute herpes zoster, it does not reliably eliminate the subsequent risk of PHN. Prevention of chronic neuropathic pain therefore depends not only on treatment of the acute episode but also on primary prevention of VZV reactivation [10].

Recombinant Zoster Vaccination

Recombinant zoster vaccine (RZV) is the principal preventive strategy against herpes zoster and its complications. Vaccination substantially reduces the occurrence of herpes zoster and provides protection against PHN [10, 11]. Because TG-PHN develops as a consequence of trigeminal VZV reactivation, reduction in herpes zoster incidence is expected to reduce the burden of trigeminal post-herpetic pain.

RZV is administered as a two-dose vaccine and is recommended for older adults as well as adults at increased risk of herpes zoster because of immunodeficiency or immunosuppression [11]. Its non-live formulation permits use in populations for whom a live zoster vaccine may be inappropriate.

Vaccination remains relevant in individuals with a previous episode of herpes zoster because prior infection does not guarantee protection against recurrence. However, RZV is preventive rather than therapeutic and is not a treatment for established TG-PHN.

Overall, vaccination represents the most effective population-level approach to reducing the burden of herpes zoster and its chronic neuropathic complications, while prompt recognition and treatment of acute trigeminal zoster remain important components of individual patient care.[10, 11].

Prognosis and Clinical Outcomes

The clinical course of TG-PHN is variable. Pain may gradually diminish as neural injury and inflammation stabilize, particularly during the first year after herpes zoster; however, persistent symptoms can continue for several years in a subset of patients [3]. The duration and severity of symptoms vary considerably among individuals, and complete resolution cannot be predicted reliably at initial presentation.

Older age, severe acute zoster pain, greater rash severity, prodromal pain, and ophthalmic involvement have been associated with an increased likelihood of developing persistent post-herpetic pain [7, 8]. These factors are useful for recognizing patients who may require closer follow-up after acute trigeminal herpes zoster.

Clinical outcome is not determined solely by pain intensity. Persistent allodynia, sensory loss, dysesthesia, pruritus, and sleep disturbance may remain clinically important even when overall pain severity improves [3]. Facial involvement can also affect eating, grooming, speaking, dental care, and

other routine activities, contributing to prolonged functional impairment.

Treatment response is similarly heterogeneous. Some patients achieve satisfactory symptom control with pharmacological or topical therapy, whereas others require combination treatment or selected interventional approaches [3]. In longstanding disease, treatment is therefore directed toward meaningful reduction in pain and sensory symptoms together with improvement in sleep, physical function, and quality of life rather than assuming that complete pain elimination will always be achievable.

Patients with previous herpes zoster ophthalmicus may require continued ophthalmological follow-up when ocular complications or persistent ocular symptoms are present. Likewise, new or progressive neurological findings during follow-up should prompt reassessment rather than being attributed automatically to established TG-PHN.

Overall, TG-PHN has a variable prognosis ranging from substantial improvement to persistent neuropathic symptoms requiring long-term management [3]. Early recognition of patients at greater risk for persistent pain, appropriate treatment of the acute zoster episode, vaccination, and individualized management of established neuropathic pain remain central to reducing its long-term clinical burden.

Discussion

TG-PHN represents a distinctive form of post-herpetic neuropathic pain because VZV reactivation occurs within the trigeminal sensory system and may affect facial, oral, and ophthalmic territories. Although it shares fundamental mechanisms with PHN elsewhere in the body, its anatomical distribution creates particular clinical challenges related to facial allodynia, sensory dysfunction, ocular involvement, and interference with routine activities such as eating, grooming, speaking, and sleep [3].

The clinical manifestations of TG-PHN reflect the combined effects of peripheral nerve injury and altered central pain processing. VZV-associated injury within the trigeminal ganglion and peripheral sensory fibers can produce both positive sensory phenomena, including burning pain, allodynia, hyperalgesia, and dysesthesia, and negative findings such as hypoesthesia or numbness [3]. This mixture of symptoms distinguishes TG-PHN from classical trigeminal neuralgia and reinforces its characterization as a painful trigeminal neuropathy rather than simply a paroxysmal facial pain syndrome [5, 6].

Diagnosis remains predominantly clinical. A history of trigeminal herpes zoster followed by persistent neuropathic pain and sensory abnormalities in the corresponding distribution is usually sufficient to establish the diagnosis [3, 5]. Additional investigations are most useful when the presentation is atypical, when another craniofacial pain disorder is suspected, or when progressive neurological or ophthalmic findings raise concern for an alternative or additional disorder.

Management remains challenging because trigeminal-specific therapeutic evidence is limited. Gabapentinoids, tricyclic antidepressants, and topical therapies form the basis of conservative treatment, while selected patients with persistent disabling symptoms may benefit from interventional approaches [3, 9]. Nerve blocks, botulinum toxin, pulsed radiofrequency, and peripheral nerve stimulation have been described in refractory disease, but the evidence supporting these approaches is less extensive than that supporting established pharmacological treatment [3]. Treatment selection should therefore remain individualized rather than following an inflexible procedural sequence.

The heterogeneity of TG-PHN is also clinically important. Patients differ in the relative prominence of continuous pain, paroxysmal exacerbations, allodynia, sensory loss, pruritus, and functional impairment [3]. These differences may partly explain the variable response to treatment and support a multimodal approach based on the predominant clinical phenotype, comorbidities, medication tolerance, and previous therapeutic response.

Prevention remains particularly important because treatment of established PHN is frequently incomplete. Prompt recognition and treatment of acute trigeminal herpes zoster are essential components of care, particularly in ophthalmic disease, while recombinant zoster vaccination provides the principal strategy for reducing herpes zoster and its subsequent complications [10, 11].

Despite growing interest in TG-PHN, important evidence gaps remain. Much of current management continues to be extrapolated from studies of PHN involving non-trigeminal dermatomes, and many trigeminal interventional studies involve relatively small patient populations [3]. Further prospective studies focused specifically on trigeminal disease are needed to clarify clinical phenotypes, identify predictors of persistent pain and treatment response, and determine which patients are most likely to benefit from specific pharmacological or interventional strategies.

Overall, TG-PHN should be approached as a heterogeneous neuropathic disorder requiring attention to pain, sensory dysfunction, functional impairment, and associated ophthalmic complications. Current evidence supports individualized multimodal management, while prevention and development of more trigeminal-specific therapeutic evidence remain important priorities [3].

Limitations

Several limitations should be considered when interpreting this narrative review. First, the literature specifically addressing trigeminal post-herpetic neuropathy remains smaller than the broader evidence base for post-herpetic neuralgia. Consequently, some aspects of treatment and prognosis are informed by studies that included PHN affecting multiple anatomical distributions rather than exclusively the trigeminal nerve.

Second, the available TG-PHN literature is heterogeneous with respect to patient selection, duration of symptoms, affected trigeminal divisions, outcome measures, treatment protocols, and length of follow-up. This variability is particularly evident in studies evaluating interventional procedures and limits direct comparison among treatment approaches.

Third, evidence for several interventional and neuromodulatory therapies is derived predominantly from small observational studies, case series, and selected patient populations. Although these approaches may be useful in refractory disease, their comparative effectiveness and long-term durability remain incompletely defined.

Finally, this article is a narrative review rather than a systematic review or meta-analysis. Study selection and evidence synthesis were therefore designed to provide a focused clinical overview rather than an exhaustive quantitative assessment of all available literature.

These limitations highlight the need for larger prospective studies using standardized definitions, clinically meaningful outcome measures, and trigeminal-specific patient populations.

Future Directions

Future research in TG-PHN should focus on improving disease-specific evidence rather than continuing to extrapolate predominantly from studies of post-herpetic neuralgia involving other anatomical distributions. Large prospective studies using standardized diagnostic criteria and clearly defined trigeminal distributions are needed to characterize the natural history of TG-PHN and identify factors associated with persistent pain, sensory dysfunction, and treatment response [3].

A major priority is improved clinical phenotyping. TG-PHN encompasses patients with different combinations of continuous pain, paroxysmal pain, mechanical allodynia, hyperalgesia, sensory loss, dysesthesia, and pruritus [3]. Future studies should determine whether these clinical patterns reflect distinct underlying mechanisms and whether they can be used to guide individualized treatment selection.

Trigeminal-specific clinical trials are also needed to establish the comparative effectiveness of pharmacological and interventional therapies. Although conventional neuropathic-pain medications remain central to treatment, evidence for procedures such as botulinum toxin injection, pulsed radiofrequency, peripheral nerve blocks, and neuromodulation remains less extensive [3]. Well-designed prospective trials with standardized pain, functional, quality-of-life, and safety outcomes would help define the appropriate role and timing of these interventions.

Greater attention should also be given to prevention and early identification of patients at risk for persistent neuropathic pain. Future studies may clarify whether clinical characteristics during acute trigeminal herpes zoster can reliably identify individuals who would benefit from closer follow-up or earlier multimodal pain management [7, 8]. Continued evaluation of herpes zoster vaccination in older and immunocompromised populations will also remain important as vaccination patterns alter the epidemiological burden of HZ and its complications [11].

Advances in sensory testing, neuroimaging, neurophysiology, and molecular research may further improve understanding of the peripheral and central mechanisms responsible for persistent trigeminal pain. At present, however, these approaches remain primarily research tools rather than established methods for routine diagnosis or treatment selection in TG-PHN [3].

Ultimately, progress in TG-PHN will depend on integrating better clinical phenotyping with trigeminal-specific therapeutic studies and long-term outcome assessment. Such research may allow management to move from largely empirical treatment toward more individualized approaches based on the predominant clinical and neurobiological characteristics of each patient.

Conclusion

Trigeminal post-herpetic neuropathy is a persistent neuropathic pain disorder that may follow reactivation of varicella-zoster virus within the trigeminal sensory system. Although it shares important features with post-herpetic neuralgia affecting other anatomical regions, trigeminal involvement presents distinctive clinical challenges because of its facial distribution, frequent ophthalmic involvement, sensory abnormalities, and potential effects on vision-related care, eating, grooming, sleep, and quality of life [3].

The disorder results from interacting peripheral and central mechanisms, including trigeminal ganglion and sensory-fiber injury, neuroinflammation, altered neuronal excitability, deafferentation, and central sensitization [3]. Diagnosis remains predominantly clinical and is supported by a history of trigeminal herpes zoster followed by persistent or recurrent neuropathic pain and sensory abnormalities within the corresponding trigeminal distribution [3, 5, 6].

Management should be individualized and may include systemic neuropathic-pain medications, topical therapies, and selected interventional approaches for patients with refractory symptoms [3, 9]. Treatment goals should extend beyond reduction in pain intensity to improvement in sleep, daily function, and overall quality of life. Prevention remains an important component of disease control, particularly through recombinant zoster vaccination and appropriate management of acute herpes zoster [10, 11].

Despite advances in the understanding of neuropathic pain, important gaps remain in TG-PHN-specific evidence. Larger prospective studies and well-designed clinical trials are needed to better define its natural history, characterize clinically meaningful phenotypes, and establish the comparative effectiveness of available therapies [3]. Improved trigeminal-specific evidence may ultimately support more individualized and mechanism-informed management of this challenging chronic pain disorder.

Ethics Statement

This article is a narrative review of previously published literature and did not involve the recruitment of human participants, collection of identifiable patient information, or use of animals. Therefore, institutional review board or ethics committee approval and informed consent were not required.

Conflict of Interest

The authors declare no conflicts of interest related to this work.

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Author Contributions

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Data Availability Statement

No new datasets were generated or analyzed for this narrative review. All information discussed in this article was derived from previously published literature.

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