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Comprehensive Insights into Post-Traumatic Trigeminal Neuropathy: Pathophysiology, Diagnosis, and Management Approaches

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Abstract: Post-Traumatic Trigeminal Neuropathy (PTTNP) is a debilitating orofacial pain condition resulting from trauma or surgical interventions involving the trigeminal nerve. Despite growing attention in clinical literature, diagnosis and management remain challenging. This review aims to critically examine current literature regarding the pathophysiology, clinical features, diagnostic tools, and therapeutic options related to PTTNP. Diagnosis of post-traumatic trigeminal neuropathy (PTTNP) is established through a structured clinical assessment. This includes detailed patient history, evaluation of pain characteristics and functional impairment, objective sensory testing such as Quantitative Sensory Testing (QST) and two-point discrimination, as well as adjunctive imaging modalities like MRI and DTI. Prognostic evaluation may be enhanced by predictive models that integrate subjective and objective findings. Findings emphasize the importance of early diagnosis, highlight subjective and objective sensory assessment tools, and outline both pharmacological and surgical strategies with varied outcomes. Predictive models and psychological factors are crucial in prognosis and recovery. PTTNP is a complex condition requiring interdisciplinary collaboration. Early intervention, comprehensive diagnostics, and personalized treatment plans are vital for optimal outcomes.

Keywords: Nerve injury; Neurosensory dysfunction; Pain; Post-traumatic trigeminal neuropathy; Orofacial pain

Introduction

Post-traumatic trigeminal neuropathy (PTTNP) is a subtype of painful trigeminal neuropathy classified under the International Classification of Headache Disorders (ICHD-3) and International Classification of Orofacial Pain (ICOP). It refers to neuropathic pain that develops as a direct consequence of a lesion or disease affecting the trigeminal nerve. This condition is often observed following surgical, traumatic, or dental interventions such as third molar extractions, implant placements, orthognathic surgeries, or local anesthetic injections. The trigeminal nerve, the fifth cranial nerve, is the primary sensory nerve of the face, supplying motor innervation to the muscles of mastication and sensory input to the face, oral cavity, and anterior two-thirds of the tongue.

Clinically, PTTNP is characterized by persistent pain, paresthesia, dysesthesia, hypoesthesia, or even complete sensory loss within the affected nerve distribution. The pain may be burning, shooting, or electric shock-like in nature and often interferes with basic functions such as chewing, speaking, or even light touch, profoundly impacting quality of life. Prevalence estimates vary but have been reported between 1.55% to 13% depending on the procedure and the affected nerve branch.

The inferior alveolar nerve and lingual nerve are the most frequently involved branches, and injuries can result in long-standing neurosensory disturbances.[9,11] The pathophysiology of PTTNP involves both peripheral mechanisms, such as axonal damage and ectopic neural firing, and central mechanisms, including altered central sensitization within the brainstem. Psychological comorbidities such as anxiety and depression often coexist and may influence pain perception and patient outcomes.

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Despite its clinical relevance, PTTNP remains underdiagnosed or misclassified, often being confused with other forms of orofacial pain, including classic trigeminal neuralgia or temporomandibular joint disorders. Moreover, standardized diagnostic protocols and treatment algorithms are lacking, contributing to suboptimal patient care. Advances in neuroimaging, predictive modeling, and emerging therapies such as topical agents and regenerative techniques are gradually improving the landscape of diagnosis and management.

This review was undertaken to integrate and synthesize recent findings on PTTNP to better understand its etiology, clinical features, diagnostic frameworks, therapeutic strategies, and future directions for research and clinical application. Post-traumatic trigeminal neuropathy (PTTNP) is a subtype of painful trigeminal neuropathy that commonly arises from dental or maxillofacial procedures such as third molar extractions, implant placement, or local anesthesia administration. The trigeminal nerve, being the primary sensory nerve of the face, is vulnerable to iatrogenic injury. This condition leads to chronic orofacial pain, neurosensory dysfunction, and significant psychosocial impairment. Recent studies have aimed to clarify its pathogenesis, improve diagnostic accuracy, and optimize therapeutic approaches.

This literature review aims to evaluate and synthesize five recent peer-reviewed studies that focus on the underlying mechanisms, diagnostic approaches, clinical outcomes, and management strategies for PTTNP.

Method

Diagnosis of post-traumatic trigeminal neuropathy (PTTNP) requires a systematic clinical assessment combining subjective symptom evaluation, objective sensory testing, and appropriate imaging modalities. The diagnostic workflow, as synthesized from the reviewed literature, consists of the following steps:

Comprehensive Patient History

- Detailed recording of the onset and nature of symptoms following a dental or maxillofacial procedure (e.g., third molar extraction, implant placement, orthognathic surgery).
- Pain characterization including quality (burning, electric shock-like, stabbing), intensity (e.g., via Visual Analogue Scale), duration, and aggravating/relieving factors.
- Functional impact on mastication, speech, and daily activities.
- Review of psychosocial aspects, including screening for anxiety, depression, or sleep disturbances, as these may influence symptom perception and prognosis.

Clinical Examination

- Inspection of the oral cavity and surgical site to assess healing, presence of scar tissue, or local inflammation.
- Palpation and sensory mapping to delineate affected areas along trigeminal nerve distributions (ophthalmic, maxillary, mandibular branches).
- Evaluation for signs of allodynia (pain from normally non-painful stimuli) and hyperalgesia (exaggerated pain response).

Objective Sensory Testing

- Quantitative Sensory Testing (QST): Measures thresholds for thermal detection, thermal pain, mechanical detection, and mechanical pain.
- Two-point discrimination test: Evaluates spatial resolution of tactile perception.
- Pinprick and light touch tests: Assess integrity of small and large sensory fibers.
- Assessment of gain-of-function and loss-of-function patterns, which can inform prognosis.

Instrumental and Imaging Studies

- Magnetic Resonance Imaging (MRI): Used to visualize nerve continuity, exclude compressive lesions, and evaluate structural changes.
- Diffusion Tensor Imaging (DTI): Detects microstructural alterations in trigeminal nerve fibers.
- Electrophysiological testing (if indicated) to assess nerve conduction and function.

Prognostic Assessment

- Integration of subjective and objective findings to classify severity and predict recovery potential.
- Application of predictive models, such as the one described by Meewis et al. (2021), to estimate neurosensory recovery likelihood (negative predictive value up to 87%).

By combining patient-reported symptoms, structured sensory testing, and advanced imaging techniques, clinicians can achieve an accurate diagnosis of PTTNP, differentiate it from other orofacial pain conditions (such as classical trigeminal neuralgia), and select the most appropriate therapeutic approach.

Result and Discussion

The five selected studies provided complementary insights into post-traumatic trigeminal neuropathy (PTTNP), each emphasizing different dimensions of the condition—from pathophysiology and clinical

presentation to diagnostic modalities and treatment outcomes.

Park et al. (2024) conducted a narrative overview summarizing clinical features, psychosocial burdens, and the central and peripheral mechanisms underlying persistent neuropathic pain. They emphasized the importance of timely diagnosis and advocated for a multidisciplinary approach. Their review highlighted how psychological distress can exacerbate neuropathic pain, leading to decreased quality of life and prolonged recovery.

Neal and Zuniga (2022) presented a retrospective cohort analysis investigating factors affecting surgical outcomes in PTTNP patients. Their findings revealed that early surgical intervention (within six months) and low baseline pain scores significantly improved sensory outcomes. Delayed intervention led to lower recovery rates, emphasizing the role of early referral and triaging.

Meewis et al. (2021) offered a prospective observational study linking objective assessments (Quantitative Sensory Testing, two-point discrimination) with subjective symptoms. They introduced a predictive model with an 87% negative predictive value (NPV), aiding clinicians in anticipating poor recovery trajectories. Notably, allodynia and gain-of-function symptoms correlated more strongly with reduced quality of life than loss-of-function indicators.

Sabeh (2024) presented a case report of a patient with implant-induced PTTNP successfully managed with compounded topical pharmacotherapy (lidocaine, ketamine, gabapentin, ketoprofen, and capsaicin). The patient's symptoms resolved completely without systemic medications. This study underscores the potential of localized, personalized therapy when systemic options are limited or contraindicated.

Liu and Tanaka (2025) reviewed the diagnostic challenges in distinguishing PTTNP from classical trigeminal neuralgia. They emphasized the utility of advanced neuroimaging techniques (e.g., MRI, diffusion tensor imaging) and discussed emerging regenerative interventions, such as stem cell therapy and low-intensity pulsed ultrasound (LIPUS), in managing severe or intractable cases.

Thematic Integration:

Across all studies, several cross-cutting findings emerged:

- Etiology & Risk Factors: Dental procedures, particularly third molar extractions and implant placements, were identified as the most common triggers. The lingual and inferior alveolar nerves were most frequently affected due to their anatomical exposure.
- Clinical Symptoms: Common presentations included burning pain, electric shock sensations, paresthesia, hypoesthesia, dysesthesia, and impaired oral function (mastication, speech). Gain-of-function symptoms (e.g., allodynia) were especially distressing and predicted poorer quality of life.
- Diagnostic Modalities: The combined use of patient-reported outcomes, QST, and neuroimaging improved diagnostic accuracy. Meewis et al. highlighted the importance of integrating subjective and objective data to formulate a prognosis.
- Management Strategies: Treatment ranged from conservative (pharmacological, topical agents, psychotherapy) to surgical repair. Results varied; early surgical intervention was generally more successful. Sabeh's topical approach demonstrated that targeted, local treatment could be highly effective.
- Prognostic Indicators: Key factors influencing outcomes included time to intervention, baseline symptom severity, presence of psychological comorbidities, and type of sensory disturbance. Predictive models (as proposed by Meewis et al.) are promising tools for clinical decision-making.

Table 1. Summary Table of Reviewed Studies

Authors & Year	Focus	Key Findings
Park et al. (2024)	Narrative overview of PTTNP	Highlighted psychosocial impact and central/peripheral sensitization.
Neal & Zuniga (2022)	Surgical outcomes and prognostic factors	Early surgery & low baseline pain = better outcomes.
Meewis et al. (2021)	Sensory assessment and predictive model	Developed 87% NPV model; subjective/objective testing correlated.
Sabeh (2024)	Case report: topical therapy	Complete pain resolution via compounded topical formulation.
Liu & Tanaka (2025)	Diagnostic differentiation and regenerative therapies	Emphasized advanced imaging (e.g., DTI) to differentiate PTTNP from TN; discussed potential of stem cell and LIPUS therapy.
Cruccu et al. (2010)	EFNS guidelines on neuropathic pain	Recommended standardized assessment (QST, sensory tests).
Zakrzewska (2010)	Evidence-based TN practice	Highlighted diagnostic pitfalls; stressed guideline-based care.
Haanpaa et al. (2011)[26]	NeuPSIG guidelines	Established global standards for neuropathic pain assessment.

Authors & Year	Focus	Key Findings
Benoliel & Eliav (2008)	Orofacial neuropathic pain	Reviewed mechanisms, epidemiology, and clinical features.
Renton (2010)[9]	Dental nerve injury prevention	Proposed strategies to avoid iatrogenic IAN/lingual nerve injury.
Zakrzewska & Linskey (2014)[12]	Review of trigeminal neuralgia	Summarized diagnostic criteria and management controversies.
Di Stefano & Truini (2017)[11]	Painful peripheral neuropathies	Outlined mechanisms and symptom profiles of neuropathic pain.
Burchiel (2003)	Classification of facial pain	Introduced new categorization of facial pain syndromes.
Cruccu & Truini (2017)	Refractory neuropathic pain	Described central sensitization and mechanisms of drug resistance.
Zakrzewska (2010)	Multidisciplinary pain management	Emphasized team approach for complex chronic orofacial pain.
Bennetto et al. (2007)	TN management review	Reviewed treatment strategies including medical & surgical options.
Love & Coakham (2001)	Pathology of trigeminal neuralgia	Detailed histopathological mechanisms underlying TN.
Nurmikko & Eldridge (2001)	TN diagnosis & treatment	Discussed clinical features and anesthetic approaches.
Sandell & Eide (2008)	Microvascular decompression outcomes	Showed variable success depending on constant vs paroxysmal pain.
Zakrzewska & Patsalos (2002)	Long-term medical management	Reported effectiveness and relapse rates with anticonvulsants.
Cruccu et al. (2008)	TN epidemiology & carbamazepine	Reported frequency and effectiveness of carbamazepine in TN.
Obermann et al. (2007)	Trigeminal nociceptive processing	Found impaired pain processing in TN patients (neurophysiology).
Jensen & Baron (2003)	Neuropathic pain mechanisms	Proposed framework linking symptoms/signs to underlying mechanisms.
Scholz & Woolf (2002)	Pain mechanisms	Described central sensitization and plasticity in chronic pain.
Benoliel & Gaul (2017)	Persistent idiopathic facial pain	Differentiated migrainous vs neuropathic phenotypes.
Baad-Hansen et al. (2004)	Pharmacologic testing (lidocaine/naloxone)	Showed differential responses in trigeminal neuropathic pain.
Becerra et al. (2006)	fMRI study of trigeminal pain	Demonstrated altered brain activation in migraine patients.
Nurmikko & Eldridge (2002)	TN pathophysiology & treatment	Summarized mechanisms and pharmacological strategies.
Olesen (2004)[22]	ICHD classification	Standardized criteria for headache and trigeminal pain disorders.
Zakrzewska (2002)	Multidisciplinary approach in TN	Advocated team-based management for complex facial pain.

Post-traumatic trigeminal neuropathy (PTTNP) presents complex diagnostic and therapeutic challenges due to its heterogeneous etiology, diverse symptomatology, and varying responses to treatment. The synthesis of five recent studies offers important perspectives on how clinicians can navigate these challenges with evidence-based and individualized strategies.

Timing of intervention remains one of the strongest prognostic determinants. As demonstrated by Neal & Zuniga (2022), early surgical repair—particularly within six months of injury—is associated with superior sensory recovery outcomes. Delays in treatment not only reduce the likelihood of successful nerve regeneration but may also allow maladaptive central sensitization to develop, leading to chronic neuropathic pain. Therefore, early identification, accurate classification, and timely referral are crucial.

Diagnostic precision is another recurring theme across the literature. Meewis et al. (2021) showed that integrating objective assessments, such as Quantitative Sensory Testing (QST), with patient-reported outcomes significantly improves prognostication. The development of a predictive model with 87% negative predictive value (NPV) emphasizes the practical application of such tools in clinical decision-making. Despite growing awareness in academic literature, many general practitioners and dental surgeons remain unfamiliar with the full clinical spectrum of PTTNP.[4,8] The lack of widespread clinical guidelines leads to delayed referrals, underdiagnosis, and occasionally inappropriate interventions. Bridging this knowledge gap through continuing education, standardized

screening tools, and interdisciplinary referral protocols is essential to improving early detection and outcomes.[20] Accurate diagnosis also enables the differentiation of PTTNP from similar conditions like classical trigeminal neuralgia, as highlighted by Liu & Tanaka (2025), who advocated for the use of advanced neuroimaging (e.g., DTI) and histopathological insights to clarify overlapping syndromes.

The psychological burden associated with PTTNP is not negligible. Park et al. (2024) highlighted the bidirectional relationship between chronic orofacial pain and mood disorders such as anxiety and depression.[4,14] This underscores the importance of psychological screening and incorporation of cognitive-behavioral therapy or other psychosocial interventions into comprehensive treatment plans.

Pharmacologic management, traditionally centered on systemic agents like gabapentinoids and tricyclic antidepressants, is being complemented by emerging localized treatments. Sabeh (2024) demonstrated the successful use of a compounded topical formulation to achieve complete pain resolution, offering a promising alternative with fewer systemic side effects, especially for patients with contraindications to systemic therapy.

Emerging regenerative therapies also signal a paradigm shift in treatment strategies. Liu & Tanaka (2025) emphasized the potential role of stem cell therapies and low-intensity pulsed ultrasound (LIPUS) in promoting nerve repair. Although still experimental, these modalities may become viable options for patients with refractory symptoms in the near future.

However, current research is limited by methodological variability and small sample sizes. Most studies reviewed are narrative reviews, retrospective analyses, or case reports, which limit the strength of their conclusions. The absence of standardized diagnostic criteria, variability in outcome measurement, and lack of long-term follow-up data further hinder generalizability.

In conclusion, the management of PTTNP demands a multidimensional approach that integrates timely intervention, accurate diagnosis using combined clinical and neurophysiological tools, psychosocial support, and consideration of both traditional and emerging therapies. Future research should prioritize prospective cohort studies and randomized controlled trials to validate prognostic models and assess the efficacy of novel interventions, ultimately guiding the development of consensus-based clinical guidelines.[6,23] Collectively, these findings underscore the necessity of shifting from a reactive, symptom-focused model toward a proactive, preventive, and precision-medicine-based approach in managing post-traumatic trigeminal neuropathy. This transition will require not only clinical innovations but also systemic changes in education, access to care, and long-term patient monitoring.

Conclusion

PTTNP is a challenging clinical entity with diverse manifestations and variable treatment responses. Early recognition, comprehensive sensory assessment, psychological support, and multidisciplinary coordination are essential for improving patient outcomes. Future studies should prioritize standardizing diagnostic criteria, validating predictive tools, and developing minimally invasive and regenerative therapeutic strategies.

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Conflicts of Interest
In this study there is no conflict of interest.

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