

## Medical Science

### To Cite:

Górski J, Górka M, Janowski M, Czyż P, Strzałkowska A. Advances in Neuroregeneration and Multimodal Therapeutic Approaches for Post-Traumatic Trigeminal Neuropathy Following Maxillofacial Fractures: A Comprehensive Review. *Medical Science* 2025; 29: e193ms3689  
doi: <https://doi.org/10.54905/disssi.v29i163.e193ms3689>

### Authors' Affiliation:

<sup>1</sup>Medical University of Lodz, Pomorska 251, 92-213, Lodz, Poland  
<sup>2</sup>Central Teaching Hospital of Medical University of Lodz, Pomorska 251, 92-213, Lodz, Poland  
<sup>3</sup>Szpital Miejski im. Św. Wincentego a Paulo w Gdyni, ul. Wójta Radtkego 18 Gdynia, Poland

### \*Corresponding author:

Jakub Górski,  
Medical University of Lodz, Pomorska 251, 92-213, Lodz, Poland,  
E-mail [jakubgorski26@icloud.com](mailto:jakubgorski26@icloud.com)

### Contact List

Jakub Górski	<a href="mailto:jakubgorski26@icloud.com">jakubgorski26@icloud.com</a>
Marta Górka	<a href="mailto:martagorska@onet.eu">martagorska@onet.eu</a>
Maciej Janowski	<a href="mailto:maciej.janowski96@gmail.com">maciej.janowski96@gmail.com</a>
Paweł Czyż	<a href="mailto:pawel.czyz98@gmail.com">pawel.czyz98@gmail.com</a>
Antonina Strzałkowska	<a href="mailto:antonina.stralkowska99@gmail.com">antonina.stralkowska99@gmail.com</a>

### ORCID List

Jakub Górski 0009-0006-7623-1181  
Marta Górka 0009-0001-0798-5194  
Maciej Janowski 0009-0003-5196-5945  
Paweł Czyż 0009-0005-3351-1610  
Antonina Strzałkowska 0009-0003-0075-0303

### Peer-Review History

Received: 14 July 2025  
Reviewed & Revised: 27/July/2025 to 14/September/2025  
Accepted: 21 September 2025  
Published: 29 September 2025

### Peer-review Method

External peer-review was done through double-blind method.

### Medical Science

pISSN 2321-7359; eISSN 2321-7367



© The Author(s) 2025. Open Access. This article is licensed under a [Creative Commons Attribution License 4.0 \(CC BY 4.0\)](https://creativecommons.org/licenses/by/4.0/), which permits use, sharing, adaptation, distribution and reproduction in any medium or format, as long as you give appropriate credit to the original author(s) and the source, provide a link to the Creative Commons license, and indicate if changes were made. To view a copy of this license, visit <http://creativecommons.org/licenses/by/4.0/>.

# Advances in Neuroregeneration and Multimodal Therapeutic Approaches for Post-Traumatic Trigeminal Neuropathy Following Maxillofacial Fractures: A Comprehensive Review

Jakub Górski<sup>1\*</sup>, Marta Górka<sup>2</sup>, Maciej Janowski<sup>2</sup>, Paweł Czyż<sup>3</sup>, Antonina Strzałkowska<sup>2</sup>

## ABSTRACT

Craniofacial fractures can result in a complication known as Post-traumatic trigeminal neuropathy (PTTN). The symptoms associated with this condition include neuropathic pain and sensory loss. The treatment of PTTN has been dominated by microsurgical interventions and pharmacotherapy. However, these methods are characterized by relatively low effectiveness and rarely restore complete sensation or pain relief. PTTN is related to two distinct pathologies, which are structural nerve damage and aberrant pain processing within the central nervous system. Mesenchymal stem cell therapies enhance healing processes by stimulating axonal growth, remyelination, and inflammation reduction. Biomaterial nerve conductors not only provide structural support, but they also deliver of growth factors and cells, which influence on nerve repair optimisation. Physical modalities (e.g., low-energy laser therapy) have been demonstrated to accelerate regeneration and reduce inflammation. Neuromodulatory techniques are also crucial in treating refractory neuropathic pain, as they act on central pain pathways. Approaches such as peripheral nerve stimulation, repetitive transcranial magnetic stimulation, and percutaneous trigeminal nerve stimulation improve quality of life (when medication alone is insufficient). The future of PTTN treatment lies in a multimodal approach combining early surgery with neuromodulation therapies. These complex strategies aim to achieve nerve reconstruction, lasting pain relief, and full reintegration into daily life after facial trauma.

**Keywords:** Trigeminal Neuropathy, Trauma, Maxillofacial Surgery, Neuroregeneration

## 1. INTRODUCTION

PTTN is also known as post-traumatic trigeminal neuropathic pain (PTNP). It is a condition characterized by persistent pain, paresthesia, and sensory deficits, which affect at least one branch of the trigeminal nerve. These symptoms persist above

three months after surgery in craniofacial region (Ni et al., 2021). The incidence of this condition ranges from approximately 1.5% to 13%. Variations in prevalence rate stem from the diversity of mechanism of injury, anatomical location, and diagnostic criteria – mandibular fractures may cause inferior alveolar nerve damage, while midfacial fractures tend to damage the infraorbital branch, leading to Wallerian degeneration and local ischemic damage (Garcia-Isidoro et al., 2021; Sarica et al., 2022).

The pathophysiology of PTTN involves a series of peripheral and central mechanisms. Damage to the peripheral nervous system can trigger Wallerian degeneration, a process that occurs distally from the site of lesion. The degeneration involves the degradation of myelin, a protective sheath around nerve fibers, by Schwann cells. There is an influx of macrophages, contributing to the inflammatory response. It has been related to raised levels of proinflammatory cytokines (TNF- $\alpha$ , IL-1 $\beta$ , IL-6) released by resident glial and immune cells. These cytokines have been found to sensitize nociceptive fibers and lower activation thresholds (Garcia-Isidoro et al., 2021; Rasche and Tronnier, 2016). Persistent nociceptive input results in central sensitization, which leads to maladaptive neuroplastic changes in trigeminal nucleus caudate and ascending thalamocortical pathways. The clinical symptoms associated with this condition include hyperalgesia, allodynia, and spontaneous pain, despite the occurrence of partial peripheral improvement (Zhang et al., 2021).

Standard treatment includes early microsurgical repair—direct neurorrhaphy or autologous nerve grafting, preferably performed within three months of injury. However, a recent meta-analysis reports complete sensory restoration in fewer than 50 % of cases, while up to one-third of patients continue to experience persistent neuropathic pain despite morphological continuity (Garcia-Isidoro et al., 2021; Sarica et al., 2022). Factors influencing outcome include delay to repair, age, and lesion severity. Pharmacologic first-line agents, such as gabapentinoids (gabapentin, pregabalin) and serotonin norepinephrine reuptake inhibitors (SNRIs), show modest superiority over placebo (number needed to treat ~6–8), yet many patients achieve incomplete pain relief—up to 60 % report residual pain—and adverse effects frequently limit adherence (Garcia-Isidoro et al., 2021; Zhang et al., 2021).

Mesenchymal stem cell-based therapies include autologous adipose-derived MSCs, dental pulp stem cells (DPSCs), and stem cells from human exfoliated deciduous teeth (SHEDs). A human-based study on treating orofacial neuropathic pain with autologous MSCs demonstrated a substantial pain reduction. The VAS score reduced from 7.5 to 4.3 on average after six months in five of nine patients. Furthermore, the authors proclaimed that the treatments were free of serious side effects, suggesting the potential safety and efficacy of this method (Vickers et al., 2014). Preclinical studies have demonstrated that MSC transplantation promotes Schwann-like differentiation, enhances remyelination, and restores electrophysiological function in models of trigeminal nerve damage (Yan et al., 2022).

Biomaterial-based neural conduits are designed using biodegradable polymers such as PLGA, PCL, and chitosan. These conduits provide structural guidance and regulated delivery of neurotrophic factors, which facilitate axon regeneration. Tissue engineering reviews underscore the significance of scaffold porosity, fiber alignment, mechanical compliance, and degradation rate in optimizing Schwann cell alignment and axonal outgrowth through neural gaps (Zhai and Wang, 2024; Xu et al., 2003).

Low-level laser therapy has shown promise in enhancing nerve regeneration. A systematic review of peripheral nerve regeneration studies concluded that LLLT (wavelengths 600–850 nm) consistently accelerates axonal regrowth, improves myelination, reduces inflammation, enhances electrophysiological recovery, and upregulates neurotrophic factors such as NGF (Nerve growth factor) and BDNF (Brain-derived neurotrophic factor), with both animal and early clinical evidence supporting functional benefit (Zhai and Wang, 2024).

A meta-analysis of implantable trigeminal nerve stimulation (TNS) in patients with trigeminal neuropathic pain revealed a 61.3% response rate and an average pain reduction of 2.36 points on VAS. A comparison of the results obtained from peripheral branch stimulation and ganglion or root stimulation revealed that the former yielded superior results (Ni et al., 2021). Simultaneously, MCS has been evaluated in 36 patients with refractory trigeminal neuropathic pain, achieving significant long-term pain reduction in ~72 % during test stimulation and sustained efficacy in most subjects at 5.6-year follow-up (Rasche and Tronnier, 2016). Systematic reviews of noninvasive brain stimulation (repetitive transcranial magnetic stimulation - rTMS and transcranial direct current stimulation - tDCS) in patients with neuropathic pain demonstrate high analgesic response rates (~97% with rTMS, ~81% with tDCS). Its potential utility in treating refractory orofacial pain syndromes is underscored by these findings (Zhang et al., 2021; Henssen et al., 2020).

The above-mentioned evolving therapeutic approaches reflect a shift toward multimodal treatment paradigms. The combination of microsurgical repair with complementary neuroregenerative therapies (stem cells, biomaterial scaffolds, photobiomodulation) and neuromodulation has been shown to enhance both structural nerve repair and symptomatic relief in a synergistic manner.

The objective of this study is to ascertain the most opportune timing, the most suitable patient selection, the most effective biomarker stratification, and the combination of interventions that will optimize long-term sensory recovery and pain-free function in the treatment of PTTN.

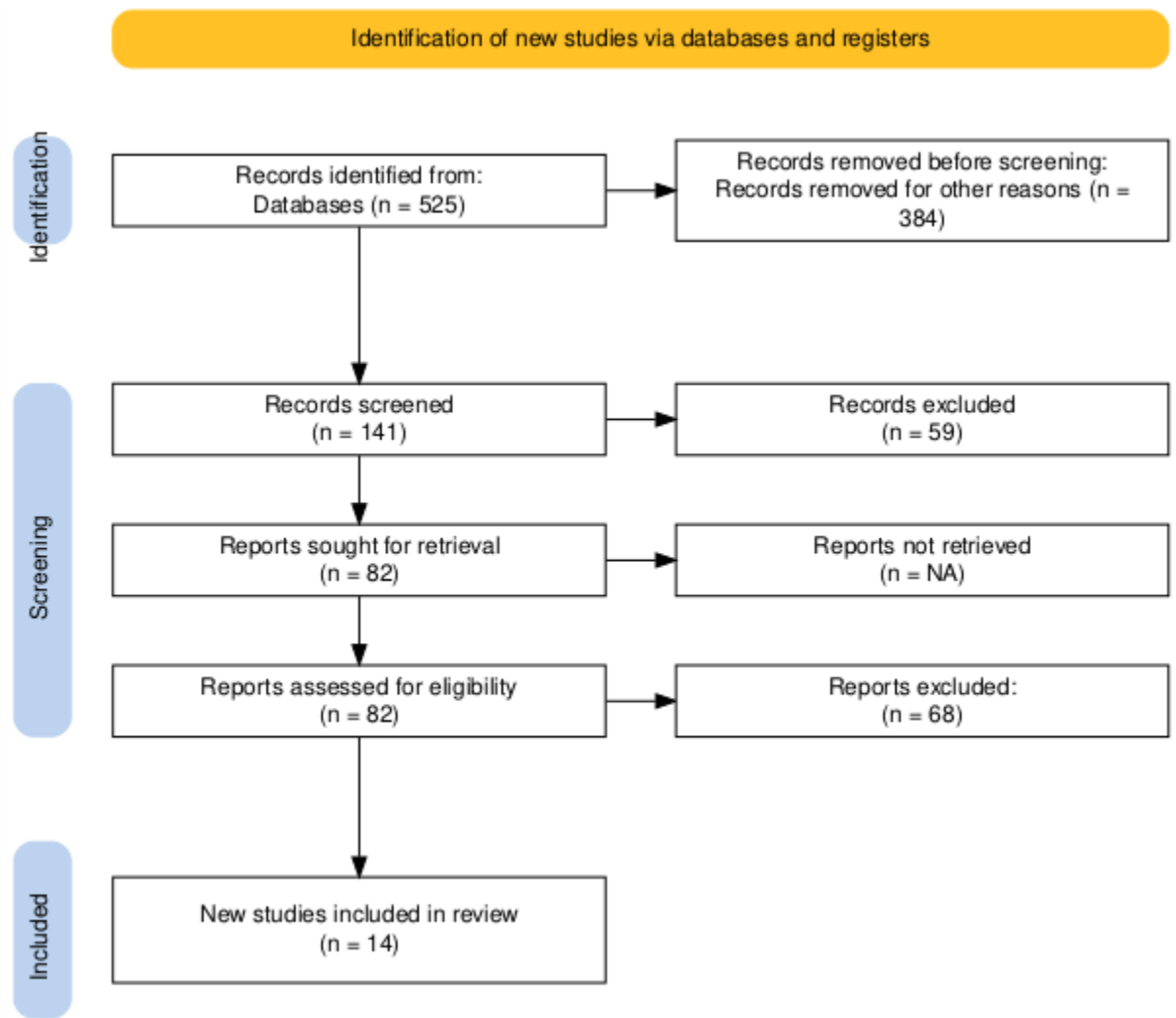


Figure 1. PRISMA diagram

## 2. REVIEW METHODS

### Search Strategy

The topic of this review was investigated by examining related literature contained in major databases, which include PubMed, Embase, and Scopus. All the presented data originate from research conducted in the last 25 years, up to June 2025. The screening is compatible with the PRISMA 2020 guidelines (Figure 1).

### Inclusion & Exclusion Criteria

Studies included systematic reviews, meta-analyses, or other analyses that evaluate interventions for nerve repair and pain management in patients with post-traumatic trigeminal neuropathy or other peripheral neuropathies. These studies may include human and animal models. In addition, randomized controlled trials or controlled cohort studies in humans with a minimum sample size of ten participants were included, as well as rigorously designed preclinical animal studies reporting quantitative outcomes related to nerve regeneration.

Excluded from the analysis were case reports, non-peer-reviewed articles, editorials, studies lacking full-text access, and studies focused exclusively on central neuropathic conditions.

### Study Selection & Data Extraction

Two reviewers independently evaluated the titles, abstracts, and full texts of the documents and resolved differences through consensus. The data extracted encompassed a wide range of information, including the study design, the population or model utilized, the intervention details, the comparator, the duration of the study, the outcomes (e.g., VAS pain scores, nerve conduction velocity, histologic regeneration), the follow-up, and the adverse events.

## 3. RESULTS & DISCUSSION

### Prevalence and Clinical Features of PTTN

Typically, PTTN presents with extensive craniofacial trauma and surgical intervention. The prevalence rate ranges from 1.5% to 13%, this variability seems to be linked with the type of the injury, the diagnostic criteria used, and the nerve branch involved. PTTN most often affects women at the age between 40 to 60 years old. The inferior alveolar (mandibular) and infraorbital branches are the most frequently affected. A range of sensory symptoms manifests, including tingling, numbness, dysesthesia, burning, electric shock-like pain, hyperalgesia, and sensory deficits. Due to the fact that trigeminal neuralgia's symptoms are overlapping with those of PTTN, the diagnostic of these two separate conditions is challenging in clinical practice (Park et al., 2024).

### Peripheral and Central Pathophysiology

The pathophysiology of PTTN is the result of peripheral nerve damage (e.g., tearing, compression, or ischemia), which automatically triggers Wallerian degeneration, Schwann cell dedifferentiation, macrophage clearance, and elevated levels of proinflammatory cytokines (e.g., TNF- $\alpha$ , IL-1 $\beta$ , IL-6). These processes have been shown to sensitize nociceptors and impede the process of regrowth at the site of the lesion. The phenomenon of persistent peripheral stimulation leads to a state of central sensitization, which manifests in the trigeminal nucleus caudate and ascending thalamocortical networks.

### Pharmacologic Treatment Efficacy

Systematic reviews in neuropathic pain analogues—including traumatic trigeminal neuropathy—indicate that first-line agents such as gabapentinoids (gabapentin, pregabalin) and SNRIs yield modest efficacy. However, efficacy is often partial—many patients report residual pain in up to 60% of cases—and tolerability issues limit adherence (Park et al., 2024). Evidence specific to PTTN is scarce, and high-quality RCTs in this patient population are notably lacking.

### Microsurgical Nerve Repair Outcomes

Despite the direct evidence in PTTN is limited, broader peripheral nerve repair studies show that microsurgical neuroorrhaphy or autologous nerve grafts can restore morphological continuity and improve sensation in a substantial portion (e.g., ~70%) of patients, on condition that they are performed early (<3–6 months). Yet, persistent neuropathic pain is common despite structural repair. Factors such as surgical delay, lesion severity, patient age, preoperative pain, and psychological co morbidities significantly influence outcome (Park et al., 2024). Meta-analyses of peripheral nerve repair emphasize the limited symptom resolution with surgery alone, and the importance of timing and patient selection.

### Stem Cell–Based Neuroregeneration

A 2021 systematic review of mesenchymal stem cell (MSC) therapy in peripheral nerve injury summarizes promising preclinical and early human data: MSCs (adipose-derived, dental pulp, or deciduous tooth derived) are valued for neurotrophic and anti-inflammatory/paracrine effects, low immunogenicity, and potential to differentiate into Schwann-like cells (Lavorato et al., 2021; Nectow et al., 2012). In animal models, MSCs promote axonal outgrowth, remyelination, and functional recovery. Human clinical studies remain preliminary—few small cohorts report reductions in neuropathic pain (e.g., VAS drop), but robust RCTs in PTTN are absent. It is a consensus that MSC therapy holds promise as a promising adjunct. Yet, there is a call for standardization of protocols and for the conduct of controlled trials to assess its efficacy and safety (Lavorato et al., 2021; Sasso et al., 2020).

Biomaterial Nerve Guidance Conduits (NGCs)

Over the past decade, biomaterial science has advanced NGC design through both natural (collagen, chitosan, silk, fibrin) and synthetic polymers (PLGA, PCL, PLA, polypyrrole blends) (Jiang et al., 2020). These conduits provide structural guidance, directional topology (via micro/nanofibers), controlled neurotrophic factor release, and favorable mechanical properties matching nerve compliance. Studies in rats and larger animal models demonstrated that conduits with angled luminal fillers, anisotropic topographies, or embedded gradient delivery of NGF/BDNF outperform hollow conduits in bridging defects up to ~20–30 mm with partial functional recovery. However, no current conduit matches the autograft gold standard across all defect sizes. Conduits incorporating MSCs and neurotrophic gradients hold particular translational promise (Nectow et al., 2012).

**Photobiomodulation (Low Level Laser Therapy)**

Studies of LLLT in peripheral nerve crush models (e.g., sciatic nerve in rodents) consistently demonstrate beneficial effects, including improved morphology, electrophysiology, vascular proliferation, improved collagen synthesis, reduced inflammation, and upregulation of growth factors – NGF and BDNF. Wavelengths of 660 nm and 830 nm, power settings of 30–40 mW, and energy densities of 4–10 J/cm<sup>2</sup> are most commonly effective. Positive outcomes of the experiments are reported almost consistently (17 out of 19), regardless of the heterogeneity in the protocols used. Systematic data in orofacial nerve models is lacking; nonetheless, given translational parallels, LLLT may augment regeneration when combined with other modalities (Sasso et al., 2020).

**Neuromodulation Techniques**

Neuromodulation approaches include non-invasive options (transcutaneous trigeminal nerve stimulation, TENS, rTMS, tDCS) and invasive options (peripheral nerve stimulation, motor cortex stimulation). Although specific RCTs in PTTN are very limited, case reports and extrapolation from other neuropathic pain contexts highlight potential analgesic benefit via modulation of central sensitization pathways. Implantable PNS (Peripheral nerve stimulation) in trigeminal neuropathic pain in broader contexts achieved response rates of ~60% in pooled series; MCS demonstrated sustained long-term control (~70% responder rate at 5+ years follow up) in small cohorts. Noninvasive TNS lacks high-quality evidence in PTTN, yet analogies from depression/PTSD neuromodulation suggest symptomatic modulation is plausible (Sasso et al., 2020). The summary is given in Table 1.

**Table 1.** Comparison of Therapeutic Approaches for Post-Traumatic Trigeminal Neuropathy

<b>Pharmacologic Treatment</b>	Widely available; first-line; modest efficacy	- incomplete pain relief - often side effects
<b>Surgical Repair</b>	Best if performed early (<6 months)	- persistent pain (most often) - outcomes depending on timing and injury severity
<b>Stem Cell Therapy (MSCs)</b>	Anti-inflammatory; low immunogenicity	- lacks human RTCs
<b>Nerve Conduits (NGCs)</b>	Support axon growth; customizable	- minor effectiveness compared to autografts in significant defects
<b>Photobiomodulation (LLLT)</b>	Improves regeneration; noninvasive	- limited clinical evidence in the trigeminal nerves
<b>Neuromodulation</b>	Modulates central pain; effective in some chronic cases (~60–70%)	- limited clinical evidence in the trigeminal nerves

**Limitations and Future Directions**

Several limitations affect current proof about PTTN. High-quality RTCs are scarce, particularly for neuromodulation and MSC-based therapies. Study designs differ significantly in terms of definitions, outcome measures, and follow-up duration. There's a translational gap between animal models e.g., sciatic nerve crush and the trigeminal nerve's specific structure and function. Standard ways to identify patients and predict their response are missing, hindering personalized treatments. Also, different LLLT methods and conduit designs cause varied results in studies.

RCTs comparing early microsurgery with surgery combined with MSC adjunctive therapy or photobiomodulation are essential. A focus should be placed on exploring non-invasive treatment options such as transcutaneous trigeminal nerve stimulation or rTMS.

Priority should be given to translating advanced NGCs characterized by built-in growth gradients, anisotropic architecture, and controlled release of NGF, BDNF, or MSCs into early-phase clinical trials targeting trigeminal nerve gaps. Systematic evaluation of combination protocols, such as the combination of conduit connection with MSCs, LLLT, and neuromodulation, could facilitate the study of potential additive or synergistic effects. A unified framework for diagnosis and treatment outcomes is also essential. This should include PRISMA-compliant registries, GRADE-based evidence hierarchies, and core outcome sets for sensory and pain outcomes. Those actions ought to enable harmonization of research and clinical practice.

#### 4. CONCLUSION

Standard surgical and pharmacological treatment of PTTN often fails, leading to persistent neuropathic pain and incomplete sensory recovery. It is clear that attention is now focused not only on symptom relief but also on regenerative methods and combinations of therapies to restore nerve function and improve treatment outcomes.

Mesenchymal stem cells (MSCs) appear to be a promising solution. They promote axonal regeneration, rebuild myelin sheaths and modulate the local immune system. MSCs differentiate into Schwann cell-like cells, which support functional nerve regeneration. Biomaterials known as nerve guidance conduits (NGCs) support this process by providing a scaffold for axon growth. NGCs also enable the controlled release of neurotrophic factors such as NGF and BDNF. Integrating those methods or using specially designed conduits with growth factor gradients has been demonstrated to improve regenerative outcomes.

Low-level laser therapy (LLLT) is another effective method of supporting regeneration. LLLT by stimulating neurotrophic signals and improving microcirculation causes inflammation reduction. Techniques such as peripheral or transcutaneous nerve stimulation and non-invasive brain stimulation techniques (e.g., rTMS, tDCS) address negative changes in the central nervous system. Future treatment should combine these regenerative and neuromodulatory methods into integrated and standardized treatment protocols. These will permanently restore sensation and nerve function.

#### List of abbreviations

AMSTAR 2 - A MeaSurement Tool to Assess systematic Reviews 2

BDNF - Brain-derived neurotrophic factor

DPSC - Dental pulp stem cells

GRADE - Grading of Recommendations Assessment, Development and Evaluation

LLLT - Low-level laser therapy

MCS - Motor cortex stimulation

MSC - Mesenchymal stem cell

NGF - Nerve growth factor

NGCs - Nerve guidance conduits

PCL - Polycaprolactone

PLA - Polylactic acid

PLGA - Poly(lactic-co-glycolic acid)

PNS - Peripheral nerve stimulation

PRISMA - Preferred Reporting Items for Systematic Reviews and Meta-Analyses

PTNP - Post-traumatic trigeminal neuropathic pain

PTTN - Post-traumatic trigeminal neuropathy

RCT - Randomized controlled trial

rTMS - Repetitive transcranial magnetic stimulation

SHED - Stem cells from human exfoliated deciduous teeth

SNRIs - Serotonin-norepinephrine reuptake inhibitors

TENS - Transcutaneous electrical nerve stimulation

TNS - Transcutaneous nerve stimulation

tDCS - Transcranial direct current stimulation

VAS - Visual analogue scale



**Acknowledgments**

The authors have no acknowledgments to disclose.

**Author contributions**

Project management, literature selection – Jakub Górski, Marta Górską

Conclusions and recommendations – Jakub Górski, Maciej Janowski

Writing – Jakub Górski, Paweł Czyż, Antonina Strzałkowska

Synthesis of results – Maciej Janowski, Marta Górską

Language correction – Antonina Strzałkowska, Paweł Czyż

Data analysis – Jakub Górski, Marta Górską

All authors contributed in the preparation of the final manuscript.

**Informed consent**

Not applicable.

**Ethical approval**

Not applicable.

**Funding**

This study has not received any external funding.

**Conflict of interest**

The authors declare that there is no conflict of interest.

**Data and materials availability**

All data associated with this study are present in the paper.

**REFERENCES**

1. Garcia-Isidoro S, Castellanos-Sanchez VO, Iglesias-Lopez E, Perpiña-Martinez S. Invasive and Non-Invasive Electrical Neuromodulation in Trigeminal Nerve Neuralgia: A Systematic Review and Meta-Analysis. *Curr Neuropharmacol* 2021;19(3):320–33. doi: 10.2174/1570159X18666200729091314
2. Henssen D, Kurt E, van Walsum AMVC, Kozicz T, van Dongen R, Bartels R. Motor cortex stimulation in chronic neuropathic orofacial pain syndromes: a systematic review and meta-analysis. *Sci Rep* 2020;10(1):7195. doi: 10.1038/s41598-020-64177-z
3. Jiang H, Qian Y, Fan C, Ouyang Y. Polymeric Guide Conduits for Peripheral Nerve Tissue Engineering. *Front Bioeng Biotechnol* 2020;8:582646. doi: 10.3389/fbioe.2020.582646
4. Lavorato A, Raimondo S, Boido M, Muratori L, Durante G, Cofano F, Vincitorio F, Petrone S, Titolo P, Tartara F, Vercelli A, Garbossa D. Mesenchymal Stem Cell Treatment Perspectives in Peripheral Nerve Regeneration: Systematic Review. *Int J Mol Sci* 2021;22(2):572. doi: 10.3390/ijms22020572
5. Nectow AR, Marra KG, Kaplan DL. Biomaterials for the development of peripheral nerve guidance conduits. *Tissue Eng Part B Rev* 2012;18(1):40–50. doi: 10.1089/ten.teb.2011.0240
6. Ni Y, Yang L, Han R, Guo G, Huang S, Weng L, Wang X, Li Z, Huang D, Hu R, Zhou H. Implantable Peripheral Nerve Stimulation for Trigeminal Neuropathic Pain: A Systematic Review and Meta-Analysis. *Neuromodulation J Int Neuromodulation Soc* 2021;24(6):983–91. doi: 10.1111/ner.13421
7. Park HJ, Ahn JM, Ryu JW. Post-Traumatic Trigeminal Neuropathic Pain: A Narrative Review of Understanding, Management, and Prognosis. *Biomedicines* 2024;12(9):2058. doi: 10.3390/biomedicines12092058
8. Rasche D, Tronnier VM. Clinical Significance of Invasive Motor Cortex Stimulation for Trigeminal Facial Neuropathic Pain Syndromes. *Neurosurgery* 2016;79(5):655–66. doi: 10.1227/neu.0000000000001353
9. Sarica C, Iorio-Morin C, Aguirre-Padilla DH, Paff M, Villeneuve SA, Vetkas A, Yamamoto K, Samuel N, Milano V, Loh A, Santyr B, Zemma A, Lozano A, Hodaie M. Clinical

- outcomes and complications of peripheral nerve field stimulation in the management of refractory trigeminal pain: a systematic review and meta-analysis. *J Neurosurg* 2022;137(5):1387–95. doi: 10.3171/2021.12.JNS212869
10. Sasso LL, de Souza LG, Girasol CE, Marcolino AM, de Jesus Guirro RR, Barbosa RI. Photobiomodulation in Sciatic Nerve Crush Injuries in Rodents: A Systematic Review of the Literature and Perspectives for Clinical Treatment. *J Lasers Med Sci* 2020;11(3):332–44. doi: 10.34172/jlms.2020.54
11. Vickers ER, Karsten E, Flood J, Lilischkis R. A preliminary report on stem cell therapy for neuropathic pain in humans. *J Pain Res* 2014;7:255–63. doi: 10.2147/JPR.S63361
12. Xu X, Yee WC, Hwang PYK, Yu H, Wan ACA, Gao S, Boon K, Mao H, Leong K, Wang Shu. Peripheral nerve regeneration with sustained release of poly (phosphoester) microencapsulated nerve growth factor within nerve guide conduits. *Biomaterials* 2003;24(13):2405–12. doi: 10.1016/S0142-9612(03)00109-1
13. Yan X, Liu Y, Yu S, Huang D, Hu R. Repair Effects of Bone Marrow Mesenchymal Stem Cells on Demyelination of Trigeminal Ganglion in Rats with Trigeminal Neuralgia. *J Pain Res* 2022;15:613–22. doi: 10.2147/JPR.S347907
14. Zhai X, Wang Y. Physical modulation and peripheral nerve regeneration: a literature review. *Cell Regen* 2024;13(1):32. doi: 10.1186/s13619-024-00215-9
15. Zhang KL, Yuan H, Wu FF, Pu XY, Liu BZ, Li Z, Li K, Liu H, Yang Y, Wang Y Analgesic Effect of Noninvasive Brain Stimulation for Neuropathic Pain Patients: A Systematic Review. *Pain Ther* 2021;10(1):315–32. doi: 10.1007/s40122-021-00252-1