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AUTHOR'S VIEW

Research progress of platelet-rich plasma in promoting peripheral nerve repair

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Abstract
Peripheral nerve injury (PNI) represents a significant clinical challenge, affecting millions globally and often resulting in permanent disability. Despite advances in microsurgical techniques, functional recovery remains suboptimal due to slow regeneration and incomplete nerve repair. This review critically examines the emerging role of platelet-rich plasma (PRP) in peripheral nerve regeneration, synthesizing current evidence from both preclinical and clinical studies. Recent clinical trials have demonstrated significant improvements in functional outcomes, particularly in carpal tunnel syndrome and facial nerve injuries. The integration of PRP with novel approaches, including tissue engineering and stem cell therapy, shows promise in addressing current therapeutic limitations. However, standardization of preparation protocols and large-scale clinical validation remain critical challenges. This review provides comprehensive insights into the therapeutic potential of PRP in peripheral nerve repair and highlights future research directions.

Keywords Peripheral nerve injury · Platelet-rich plasma · Peripheral nerve regeneration · Schwann cells · Axon regeneration · Tissue engineering

Introduction

Peripheral nerve injury is a common neurological disorder that can result in severe sensorimotor deficits and chronic neuropathic pain, with a high rate of disability [1]. While microsurgical techniques such as tension-free suturing or autologous nerve transplantation are currently the best treatment options, they do not address the slow regeneration of nerves and incomplete functional recovery [2]. The delayed regeneration of nerve axons often prevents them from innervating target tissues and restoring function [3]. Consequently, accelerating the regeneration and repair of peripheral nerves has become a focal point of research.

Platelet-rich plasma (PRP), an autologous platelet concentrate with supraphysiological levels of growth factors, has demonstrated efficacy in enhancing peripheral nerve regeneration and exerting neuromodulatory analgesic effects through targeted cytokine release [4]. Its clinical adoption across multiple disciplines is attributed to three key advantages: autologous origin eliminating immunogenic risks, cost-effective therapeutic profile, and multifactorial tissue repair capabilities mediated by platelet-derived bioactive molecules. Currently, PRP constitutes an established therapeutic modality in sports medicine for tendinopathies, in regenerative medicine for osteoarthritis management, and in dermatology for androgenetic alopecia treatment, with randomized trials confirming its superiority over conventional therapies in hair follicle revitalization [5]. Emerging applications extend to gynecological conditions, where recent phase II clinical trials have demonstrated PRP's therapeutic potential in addressing endometrial atrophy and ovarian insufficiency

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[6]. A landmark study by Kusumi et al. utilizing transvaginal ultrasound monitoring revealed that intrauterine PRP administration significantly enhanced endometrial thickness and promoted follicular development in patients with thin endometrium syndrome [7].

Concurrently, accumulating preclinical and clinical evidence substantiates the neurotrophic effects of PRP in potentiating axonogenesis and facilitating functional restitution of the neural microenvironment. This comprehensive review systematically evaluates: (1) The neuroregenerative capacity of PRP in peripheral nerve injury models, with emphasis on electrophysiological recovery and histomorphometric outcomes. (2) Mechanistic pathways underlying PRP-mediated neural repair, including but not limited to Schwann cells (SCs) activation, suppression of pro-inflammatory cytokines, and promotion of myelin sheath reconstitution. (3) Translational applications in clinical neurology and orthopedics, focusing on evidence-based protocols for PRP administration in traumatic neuropathies, compressive mononeuropathies, and diabetic peripheral neuropathy.

Platelet-rich plasma in peripheral nerve regeneration

Platelet-rich plasma (PRP) is an autologous biological matrix obtained through density gradient centrifugation of anticoagulated whole blood, yielding a supraphysiological platelet concentration within plasma-enriched fibrinogen [8]. As a multimodal regenerative platform, PRP comprises three principal components with distinct reparative functions: Serve as reservoirs of α-granule-derived growth factors (PDGF, TGF-β, VEGF) and dense granule mediators (serotonin, calcium). Primarily neutrophils and monocytes, exerting immunomodulatory effects through reactive oxygen species (ROS) scavenging and matrix metalloproteinase (MMP) regulation.The regenerative cascade is initiated when platelets undergo contact activation or biochemical activation, triggering degranulation and releasing >1,500 bioactive molecules within 10 min post-activation. PDGF-AA stimulates Schwann cell migration, while TGF-β1 suppresses MMP-9 activity to stabilize the fibrin scaffold. Concurrently, leukocyte-derived IL-1ra counterbalances pro-inflammatory cytokines, establishing an immunotolerant niche for neural progenitor cell proliferation. Collectively, PRP, functioning as a multimodal regenerative platform, harnesses its rich content of bioactive molecules and cellular components to initiate a complex regenerative cascade at the molecular and cellular level, as schematically depicted in Figure 1.

In vitro studies

SCs are crucial for the regeneration and repair of peripheral nerve injuries. They bridge nerve stumps to form a Bonner band through proliferation and migration, secrete various active substances (including neurotrophic factors) to promote axon regeneration, and differentiate into myelin sheaths during the mature stage of nerve regeneration. PRP, rich in growth factors, can enhance the proliferation and migration of SCs, as well as their secretion of neurotrophic factors. An in vitro study of the effects of P-PRP on SCs suggest that platelet-derived growth factor (PDGF-BB) and insulin-like growth factor-1 (IGF-1) are the primary cytokines affecting Schwann cell proliferation and migration. Antibodies against PDGF-BB and IGF-1 counteracted the proliferation and migration of human SCs cultured in the presence of P-PRP [9]. Other studies have shown that appropriate concentrations of PRP can stimulate Schwann cell proliferation, induce neurotrophic factor synthesis, and significantly increase Schwann cell migration in a dose-dependent manner. The study also confirmed the positive effect of P-PRP on nerve growth factor secretion by SCs and showed that a low P-PRP concentration (5%) was better, and assessed the effect of P-PRP on the biological behavior of SCs. Another study cultured rat primary SCs under different concentrations of PRP and found that appropriate concentrations of PRP could stimulate cell proliferation, induce neurotrophic factor synthesis, and significantly increase SCs migration in a dose-dependent manner [10]. Wang et al. [11] reported an autologous scaffold of Leukocyte-Platelet Rich Fibrin (L-PRF) for peripheral nerve injury. The proliferation and neurotrophic factor secretion of SCs and their anti-inflammatory effects were evaluated in vitro. The results showed that L-PRF could increase SCs proliferation and neurotrophic factor secretion. Qin et al. [12] studied the effect of Concentrated growth factor (CGF) on SCs migration, proliferation and neurotrophic factor secretion. It was demonstrated that CGF can significantly promote SCs proliferation and neurotrophic factor secretion, and it can also promote SCs migration through integrin β1-mediated activation of the plaque kinase pathway. High concentrations of platelet-rich plasma-derived exosomes (PRP-exos) can be extracted from PRP, which are absorbed by SCs and promote the proliferation of SCs in vitro [13, 14]. Co-culture of PRP-exos with Mesenchymal stem cells (MSCs) also promoted Schwann cell proliferation and accelerated the growth of dorsal root ganglion axons [15]. The combination of neural microtissue and PRP can significantly promote the proliferation, secretion, migration and axon regeneration of early SCs [16].

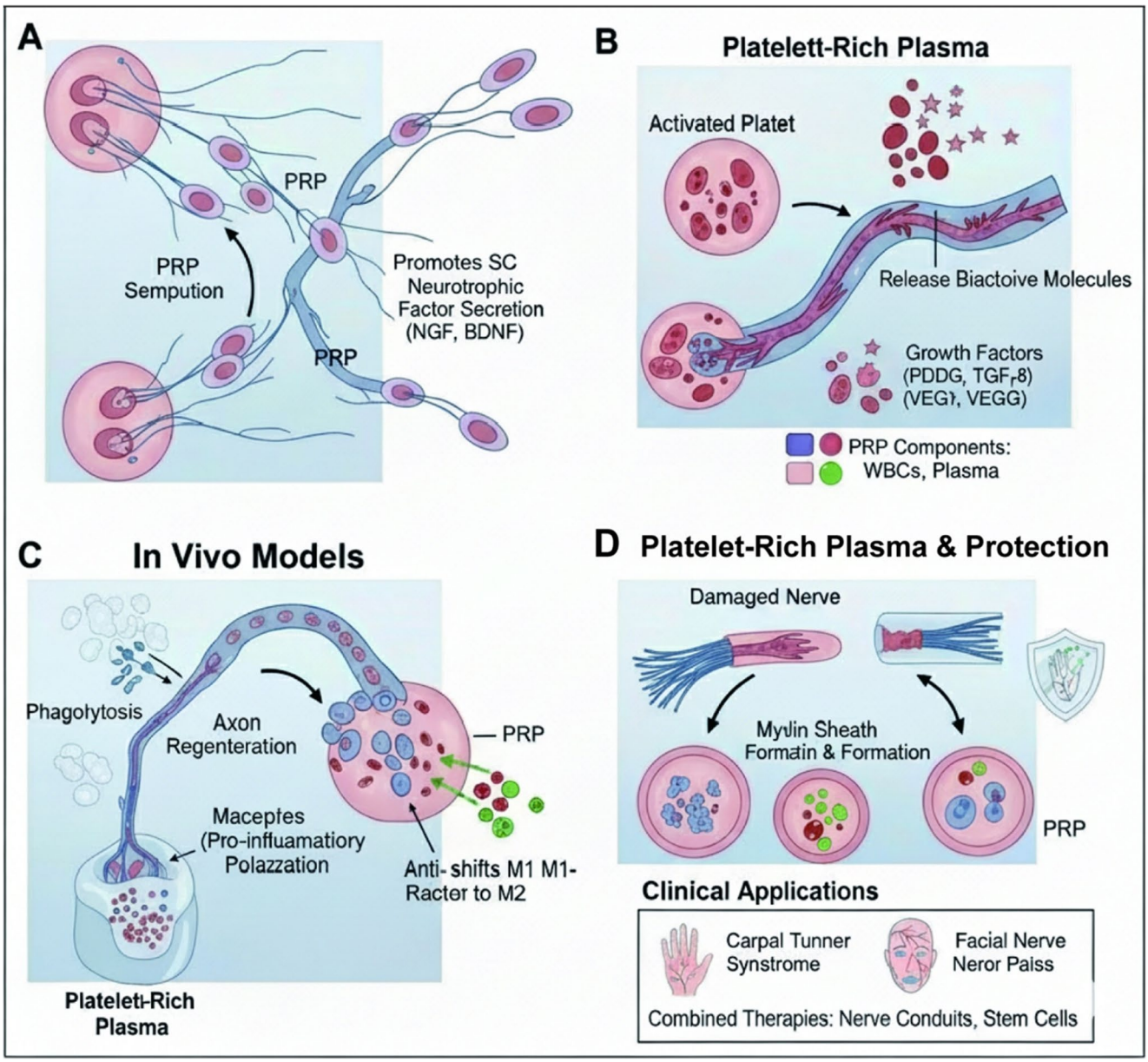

Fig. 1 Schematic diagram illustrating the therapeutic effects and mechanisms of platelet-rich plasma (PRP) in peripheral nerve repair. (**A**) *In vitro* models show PRP promotes Schwann cell proliferation and migration. (**B**) *In vivo* model illustrating PRP's role in the nerve gap. (**C**) PRP enhances axon regeneration and nerve fiber density in a repair model, and (**D**) mediates the transition of macrophages from the pro-inflammatory M1 phenotype to the anti-inflammatory M2 phenotype, reducing inflammation and promoting tissue remodeling

Regulation of inflammatory cells by platelet-rich plasma

The repair of peripheral nerve injuries involves various inflammatory cells, with macrophages playing a pivotal role. Macrophages exhibit two phenotypes: M1 and M2 [17]. M1 macrophages are primarily involved in antigen presentation, while M2 macrophages secrete anti-inflammatory cytokines, suppress immune responses, and play a key role in tissue repair. Early nerve damage is associated with M1 macrophages, which phagocytize myelin and other cellular debris during distal nerve disintegration, removing substances that hinder nerve regeneration and creating a favorable environment [18]. In the later stages of nerve repair, M2 macrophages dominate, promoting fibrosis and tissue regeneration [19]. The transition of macrophages from a pro-inflammatory phenotype (M1) to an anti-inflammatory phenotype (M2) is critical for axon regeneration regulation, and a slow transition from M1 to M2 and prolonged inflammation can impede nerve regeneration [20, 21].

PRP, as an autologous platelet concentrate, can regulate the inflammatory response following injury, promote

macrophage aggregation, enhance phagocytosis and antigen presentation, and facilitate the transformation of macrophages to the M2 phenotype, thereby promoting tissue repair and regeneration.Lana's study showed that PRP can promote the transformation of M2 macrophages and accelerate tissue healing by activating the JK1/3-STAT6 pathway [22]. Another study showed that platelet-rich growth factors (PRGF) reduced the mRNA expression of TNF-α, IL-1β and IL-6 in M0 macrophages and promoted their transformation into M2 macrophages [23].

In vivo preclinical evidence of PRP in neural repair

Preclinical investigations have validated PRP's therapeutic efficacy across diverse peripheral nerve injury models, spanning both somatic and autonomic nervous systems: PRP-augmented neurorrhaphy demonstrated 40% higher axonal regeneration rates versus standard microsuture in rabbit zygomaticotemporal branch transection models [24]. In murine chronic compressive neuropathy, PRP intraneural injection reduced mechanical allodynia through NGF/TrkA-mediated nociceptor modulation [25].This study investigated the role of PRP on the restoration of erectile function in a rat prostatectomy model, indicating that PRP restores erectile function by inhibiting PDE5A and protecting neurogenic nitric oxide synthase [26]. The chitosan/NS-chitosan conduit with a warp-knitted tube construct and aligned inner fiber had good mechanical and bioactive properties for nerve repair [27].

Animal experiments

Functional recovery following facial nerve injury is often suboptimal, with residual sequelae despite the widespread use of extraneuronal and nerve bundle suturing techniques. Li et al. [28].demonstrated through rat experiments that PRP has a protective effect on facial nerve injury, promoting the recovery of nystagmus, eyelid closure, and electrophysiological function in rats with nerve injury, potentially due to neurotrophic factors secreted by PRP Şahin et al. [29]. showed that PRP enhanced the promoting effect of chitosan gel on facial nerve healing through rat experiments In a rat model study, Farrag et al. [30] showed that PRP had a measurable neurotrophic effect on nerve regeneration after transection, and histologically found a higher number of axons in the PRP group. Şentürk et al. [31] wrapped titanium platelet-rich fibrin membrane in the facial nerve defect area of New Zealand rabbits, which contributed to nerve healing at both functional and electrophysiological levels. Cho et al. [32] used PRP and neural-induced human mesenchymal stem cells (nMSCs) to promote axon regeneration after facial nerve axonotomy injury in guinea pigs. It was found that the combined use of PRP and nMSCs was more effective than the single use. Sun et al. [33] made a left facial nerve defect model with a length of 1 cm from New Zealand rabbits. Both sciatic nerves of rats were xenografted and acellular, and Adipose derived stem cells (ADSCs) and PRP were used to repair the facial nerve injury in rabbits, with significant results. It was also found that ADSCs could not only survive in vivo as seed cells, but also differentiate into Schwann-like cells under PRP induction.

Median nerve injury is prevalent in clinical practice, particularly in carpal tunnel syndrome. Park et al. [34] injected PRP into a rabbit model of glucose-induced median nerve injury and observed significant improvements in electrophysiological and histological results after 12 weeks, indicating that PRP injection can effectively control the progression of median nerve injury.

Cavernosal nerve injury is a common complication following radical prostatectomy, often leading to erectile dysfunction. PRP has emerged as a promising therapeutic agent for promoting cavernosal nerve repair. Wu et al. [25] conducted a study where chitosan-activated platelet-rich plasma (cPRP) was injected into the sponges of a rat model with bilateral cavernosal nerve compression injury. The recovery of erectile function was assessed using transmission electron microscopy and histological examination. The results indicated that cPRP significantly reduced the loss of nitric oxide synthase-positive neurons and nerve fibers in the pelvic ganglion and cavernosal nerve neurons. Furthermore, cPRP accelerated the regeneration of myelin axons in the cavernosal nerve, reduced apoptosis, and enhanced the proliferation of smooth muscle cells in the early stages of recovery. These findings suggest that cPRP can promote the recovery of erectile function in rats with cavernosal nerve injury through early neuroprotective and tissue-protective effects. Additionally, Wu et al. [35] found in another in vitro experiment that the efficacy of PRP in treating erectile dysfunction may be related to the synergistic effect of multiple biomolecules, such as CXCL5.

Direct injury to the sciatic nerve is a common occurrence in combat trauma and conditions such as displaced acetabular fractures or femoral head dislocations. PRP injection therapy has emerged as a promising adjunct for sciatic nerve repair. Zhu et al. [36] demonstrated the efficacy of combining low-frequency shock waves with local PRP injections in treating sciatic nerve compression injuries in rabbits, achieving favorable outcomes. In recent years, there has been a significant advancement in the use of PRP for peripheral nerve regeneration, particularly when combined with other materials such as tissue-engineered nerve conduits or stem cells. Samadian et al. [37] conducted a study using a polycaprolactone and gelatin-based scaffold filled with PRP gel containing cyticholine. They constructed an

electrospun nerve catheter to treat sciatic nerve injuries in rats, with results showing successful induction of nerve tissue regeneration and restoration of motor and sensory functions. Ikumi et al. [38] utilized a platelet-rich fibrin (PRF) membrane wrapped around the anastomosis site in a rabbit sciatic nerve model. Although no significant differences were observed in electrophysiological recovery and muscle wet-weight ratio between the experimental and control groups, the experimental group showed significantly higher degrees of SC activation, as well as increased number and diameter of axon regeneration.

Huang et al. [39] converted PRF into a nerve conduit to bridge a 5 mm sciatic nerve defect in rats, using PRF nerve conduits, autologous nerve grafts, and polyurethane nerve conduits for comparison. After 12 weeks, histological evaluations revealed that the PRF nerve conduit group outperformed the other two groups in terms of myelin thickness and the number of regenerated axons, indicating the high clinical potential of PRF-based nerve conduits. Another study found that PRF nerve conduits and nerve transplants had similar therapeutic effects on sciatic nerve defects in rats, suggesting that PRF could serve as a viable alternative to nerve transplantation for treating nerve defects [40]. Furthermore, PRP combined with acellular nerve allografts (ANAs) has shown excellent therapeutic effects in repairing long nerve defects, potentially replacing the need for autologous nerve transplants [41]. Kokkalas et al. [42] compared the effects of PRP and mesenchymal stem cells (MSCs) on nerve repair, finding that both PRP and MSCs play a crucial role in promoting nerve repair both functionally and histologically, with the PRP group showing statistically superior results. Chuang et al. [43] explored the role of adipose tissue-derived stem cells (ADSCs) combined with PRF releasate in the regeneration of rat sciatic nerve injuries. Their findings indicated that the combined strategy of ADSCs and PRF releasate was more effective in nerve repair than either treatment alone. Animal experiments have also expanded to other types of peripheral nerve injuries. Zhu et al. [16] reported on the effectiveness of a novel tissue-engineered nerve graft composed of autologous veins, nerve microstructures, and PRP in repairing a 12 mm tibial nerve defect in rabbits. The study also highlighted the value of multimodal ultrasound in quantitatively evaluating the stiffness and microvascular flow of nerve grafts and target muscles, providing clinical references for the prognosis of nerve injuries.

Clinical application

A significant body of clinical research has explored the therapeutic effects of PRP on peripheral nerve injuries, with a particular focus on carpal tunnel syndrome. Galán's study [44] demonstrated that growth factor-rich plasma-assisted open surgical therapy provides long-term benefits in pain reduction and neuromuscular function improvement for patients with carpal tunnel syndrome. Shen et al. [45] conducted a prospective, randomized, single-blind clinical trial involving 52 patients with unilateral moderate carpal tunnel syndrome. Their research compared the effects of local PRP injections versus 5% glucose injections under ultrasound guidance, revealing superior postoperative clinical symptoms and neuroelectrophysiological recovery in the PRP group. Further studies have compared local glucocorticoid injections with PRP injections for mild carpal tunnel syndrome, concluding that PRP injections offer better clinical outcomes than hormone therapy [46]. An additional prospective, randomized, single-blind, controlled trial confirmed an improved prognosis for carpal tunnel syndrome patients treated with PRP [47]. A systematic review and meta-analysis of four randomized controlled trials, encompassing 191 cases, underscored the efficacy of PRP as an adjunctive treatment, significantly enhancing patient outcomes [48]. Gao et al. [49], through a meta-analysis, also advocated PRP injections as the most effective treatment option among corticosteroids, 5% glucose, and PRP for carpal tunnel syndrome. Shen [50] and Chen et al. [51] further supported PRP's effectiveness for moderate to severe carpal tunnel syndrome, noting its significant medium to long-term therapeutic effects. Despite these positive findings, some studies have reported inconclusive results regarding PRP efficacy in treating carpal tunnel syndrome. Raeissadat et al. [52]studying 41 women with mild to moderate idiopathic carpal tunnel syndrome, found no significant benefits of adjunctive PRP injections compared to conservative wrist splint treatment in terms of pain, symptom severity, functional status, and electrophysiological parameters. Similarly, Yasak et al. [53] observed no significant impact of a single PRP injection on postoperative median nerve regeneration in diabetic patients. Nonetheless, the majority of studies affirm PRP active role in median nerve injury repair, highlighting its potential in treating median nerve-related diseases and pain.

In recent years, numerous case reports have highlighted PRP application in treating various peripheral nerve damages. García de Cortázar et al. [54] detailed the case of a 28-year-old female with long-term sensory loss and neuralgia in the index finger due to finger nerve entrapment. Post-neurolysis and PRP injection, the patient experienced significant neuralgia relief and nerve recovery. Sánchez et al. [55] also reported the effective use of local ultrasound-guided PRP injections in treating post-traumatic peroneal palsy. Kuffler [56] successfully treated a patient with a 12 cm ulnar nerve defect, using a fibrin nerve catheter filled with PRF to significantly restore ulnar nerve function after

3.25 years of injury. Kuffler [57] further discovered PRF's capacity to promote nerve regeneration and functional recovery while effectively alleviating neuralgia symptoms, underscoring PRP vast potential in peripheral nerve repair.

Despite the promising results, the clinical benefits of PRP in peripheral nerve regeneration remain a subject of debate due to conflicting study outcomes. The efficacy of PRP appears to be influenced by multiple factors, including the selection of appropriate indications, the proportion of different components, and activation conditions. Variations in PRP preparation methods and individual differences lead to diverse component ratios, such as white blood cell proportions and ideal growth factor concentrations [58, 59]. Moreover, the standardization of PRP activation procedures is still pending [60]. Currently, PRP can be activated via various pathways, including thrombin, calcium chloride, and collagen, to release growth factors. To firmly establish PRP's therapeutic effects in clinical settings, extensive clinical studies are necessary to optimize and standardize treatment protocols, thereby enhancing efficacy and safety.

Clinical application prospect of Platelet-Rich plasma

The field of PRP research is expanding rapidly, with a growing number of studies each year focusing on its role in tissue repair and regeneration. These studies primarily investigate the functions of growth factors, the mechanisms of nerve repair and regeneration, platelet functionality, the role of stem cells, and the development of tissue engineering materials. While the majority of this research is currently based on animal experiments and in vitro studies, there is a notable increase in the clinical application of PRP for the treatment of peripheral nerve injuries.

There is now substantial evidence supporting the effectiveness of PRP in promoting nerve regeneration. Furthermore, research into the combination of PRP with stem cells to create tissue-engineered nerves for treating nerve defects is on the rise. This innovative approach holds promise for potentially replacing traditional nerve transplantation methods, offering a new therapeutic avenue for patients suffering from peripheral nerve injuries.

Conclusion

Peripheral nerve injury (PNI) remains a significant clinical challenge due to its potential to cause severe sensorimotor deficits and chronic neuropathic pain, often resulting in high disability rates. Traditional treatments such as microsurgical techniques and autologous nerve transplantation, while effective in some cases, fail to address the slow and often incomplete regeneration of nerves. This review highlights the potential of PRP as a novel therapeutic modality, emphasizing its autologous origin, cost-effectiveness, and multifactorial tissue repair capabilities. Future research must prioritize standardized PRP formulations, advanced biomaterials for sustained growth factor delivery, and stratified RCTs targeting defined neuropathies. Integrating omics technologies and combinatorial therapies could optimize neuroregenerative efficacy, positioning PRP as a minimally invasive alternative to nerve autografts. Addressing these gaps will require interdisciplinary collaboration to translate preclinical promise into reliable clinical protocols.

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Declarations

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Consent to participate Not required.

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Competing interests The authors declare no competing interests.

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