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Short Communication

Enhancing neuromuscular recovery after sciatic nerve injury using stem cell therapy: Evidence from a preliminary preclinical study

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Abstract

Sciatic nerve injury results in motor dysfunction and muscle atrophy, with limited effective therapies. Umbilical cord–derived mesenchymal stem cells (UC-MSCs) may promote neuromuscular recovery, but their effects on functional and muscle recovery remain unclear. This study aimed to evaluate the effects of UC-MSC therapy on functional and muscle recovery in an animal model of sciatic nerve injury. An animal experimental study with a post-test-only control group was conducted using adult male Wistar rats. Rats were randomly allocated into three groups: sham operation, saline control with sciatic nerve injury, and UC-MSC treatment after sciatic nerve injury. UC-MSCs were administered at a dose of 1×10^6 cells/kg body weight immediately after nerve injury. Functional recovery was assessed using the extensor postural thrust (EPT) test, and muscle recovery was evaluated using the gastrocnemius muscle index (GMI) post 35 days of observation. Data were analyzed using one-way ANOVA for EPT percentage recovery and Kruskal–Wallis tests for GMI values, followed by post-hoc analysis. Our data indicated there was no significant EPT percentage recovery among the study groups. In contrast, relative gastrocnemius muscle mass was significantly different across groups (p=0.012), with post-hoc analysis demonstrating a significantly higher GMI in the UC-MSC group compared to the saline control group (109.75% vs 81.68%, p=0.003), indicating improved preservation of gastrocnemius muscle mass following UC-MSC therapy. This study highlights that UC-MSC therapy significantly improved gastrocnemius muscle preservation after sciatic nerve injury but did not result in detectable functional motor recovery at the observation time point. These findings suggest that UC-MSCs might exert early structural benefits that may precede functional recovery.

Keywords: Sciatic nerve injury, UC-MSCs, nerve regeneration, extensor postural thrust, gastrocnemius muscle index

Introduction

Peripheral nerve injury (PNI) is a common clinical problem that can result in motor and sensory dysfunction, muscle weakness, and long-term disability [1,2]. Globally, PNI contributes substantially to morbidity, with reported incidence ranging from 13 to 23 per 100,000 population

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per year, and is frequently associated with trauma, orthopedic procedures, and occupational injuries [1]. PNI disrupts signal transmission between the central nervous system and target organs, leading to impaired limb function and reduced quality of life [3]. Although peripheral nerves have an intrinsic capacity to regenerate, the process is slow and often incomplete, especially in severe injuries, resulting in suboptimal functional recovery [4].

The sciatic nerve is widely used as an experimental model for studying PNI, particularly sciatic nerve injury, due to its size, anatomical accessibility, and functional importance [5]. Sciatic nerve injury represents one of the most commonly studied forms of PNI because it produces reproducible motor deficits and secondary muscle atrophy [6]. Injury to the sciatic nerve results in motor deficits and muscle degeneration, making it a suitable model for evaluating regenerative therapies [7]. Despite advances in surgical repair and postoperative rehabilitation for sciatic nerve injury, recovery of motor function and prevention of muscle atrophy remain limited, underscoring the need for adjunctive biological therapies [8].

Mesenchymal stem cells (MSCs) have emerged as a promising therapeutic option for PNI due to their regenerative, anti-inflammatory, and neuroprotective properties [9]. MSCs promote nerve repair primarily through paracrine mechanisms, secreting growth factors and cytokines that support axonal regeneration and preserve muscle integrity [10]. Among MSC sources, umbilical cord–derived MSCs (UC-MSCs) are particularly attractive for clinical translation because they are readily available, non-invasively obtained, genetically stable, and exhibit low immunogenicity [3]. Preclinical studies suggest that UC-MSCs can enhance nerve regeneration and improve functional recovery following sciatic nerve injury [8,11,12]. However, despite growing evidence supporting the regenerative potential of UC-MSCs, data remain limited regarding their simultaneous effects on functional motor recovery and muscle preservation following sciatic nerve injury, particularly when assessed using standardized functional and muscle outcome measures. Therefore, this study aimed to evaluate the effect of UC-MSCs on functional and muscle recovery after sciatic nerve injury in animal models.

Methods

Study design and setting

An experimental study using animal models with a post-test only control group design was conducted at the Animal Research Unit, Faculty of Veterinary Medicine, Universitas Syiah Kuala, Banda Aceh, Indonesia, from October to December 2025. Rats were randomly assigned to three groups: sham operation, sciatic nerve injury treated with saline control, and sciatic nerve injury treated with human UC-MSCs. In the treatment group, UC-MSCs were administered immediately after sciatic nerve injury induction at a dose of 1×10^6 cells/kg body weight, suspended in 1 mL of sterile 0.9% saline and delivered locally at the injury site. Animals were observed for 35 days following sciatic nerve injury and the treatment, functional recovery, and muscle recovery were assessed using the extensor postural thrust (PTS) test and gastrocnemius muscle index (GMI), respectively.

Sample size and selection

Adult male Wistar rats (*Rattus norvegicus*), aged 3–4 months and weighing 200–300 grams, were obtained from the Animal Research Unit, Faculty of Veterinary Medicine, Universitas Syiah Kuala, Banda Aceh, Indonesia. The minimum sample size was calculated using Federer's formula for three experimental groups, yielding a minimum of nine rats per group. To account for potential dropouts, one additional rat per group was included. Animals were selected using simple random sampling from a homogeneous population to ensure unbiased allocation. Only healthy rats without limb abnormalities were included, and any animal losing more than 10% of body weight during acclimatization or dying during the study was considered a drop-out.

Animal housing and acclimatization

Rats were housed in standard laboratory cages under controlled room temperature with a 12-hour light/dark cycle. Two to three rats were kept per cage and provided with 20 grams of pellet feed daily and *ad libitum* water. Environmental conditions, including temperature and hygiene,

were monitored throughout the study. Prior to experimental procedures, the rats underwent a seven-day acclimatization period to allow adaptation to the housing environment. During this period, body weight and general health were assessed daily to identify any animals meeting drop-out criteria.

Human umbilical cord–derived mesenchymal stem cell (UC-MSC) preparation

Human mesenchymal stem cells were obtained from Wharton's jelly of umbilical cords, provided by Prodia Stem Cell (ProSTEM), Jakarta, a certified private cord blood bank in Indonesia. ProSTEM operates in compliance with the Indonesian Ministry of Health Regulation No. 48 of 2012 on the Implementation of Stem Cell Banks from Umbilical Cord and maintains facilities certified under Good Manufacturing Practice, following international standards established by the Association of Blood Banks (AABB) and the Foundation for the Accreditation of Cellular Therapy–NetCord (FACT–NetCord). Prior to administration, UC-MSCs were suspended in sterile saline (0.9% NaCl) at a target dose of 1×10^6 cells/kg body weight. Given that the animals weighed 200–300 g, each rat received approximately $2–3\times10^5$ UC-MSCs in an injection volume of 0.2–0.3 mL, with the volume adjusted proportionally to individual body weight to ensure accurate dosing.

Nerve injury animal model and treatment

After the acclimatization period, rats were randomly assigned to three groups: sham operation, saline control with sciatic nerve injury, and UC-MSC treatment after nerve injury. Rats were anesthetized via intramuscular injection of ketamine (90 mg/kg) and xylazine (5 mg/kg). The gluteal region was shaved and prepared aseptically with povidone iodine. A minor incision exposed the sciatic nerve from the sciatic notch to the popliteal branching. Axonotmesis was induced in the right hind limb using a straight mosquito artery forceps for 60 seconds at approximately 1.5 cm from the foramen ischiadicus, and the injury site was marked with a 3/0 silk suture. Sham-operated rats underwent the same exposure without nerve injury.

Immediately after injury, rats in the saline group received 0.2–0.3 mL of sterile saline locally at the injury site according to body weight, while rats in the UC-MSC group received the prepared UC-MSC suspension. The injection site was carefully exposed to form a confined space around the injured nerve to prevent leakage of the suspension. All surgical procedures were performed by the same operator using consistent instruments to minimize inter-subject variation. Following treatment, rats were placed on a heating pad at 37°C until initial recovery was observed, then returned to their cages with easy access to soft food and water. Animals were observed for 35 days following sciatic nerve injury, and functional and muscle assessments were performed on day 36.

Motor functional assessment

Following a 35-day post-injury and treatment observation period, motor function was assessed using the EPT test on day 36. Each rat was gently held in a vertical position with the torso supported, and the hind paw was placed on a calibrated digital scale to measure the force generated during reflexive extension. The procedure was performed for both the injured limb (experimental EPT) and the contralateral limb (normal EPT). Motor deficit was calculated as the percentage reduction in force of the injured limb relative to the contralateral limb, using the formula: Motor deficit (%) = ((Force of contralateral limb – Force of injured limb) ÷ Force of contralateral limb) × 100. The force measurements were recorded in grams, while the reported EPT outcome is expressed as a percentage. Negative values may occur when the injured limb generates slightly greater force than the contralateral limb, reflecting inter-individual variability rather than measurement error. This assessment provided a quantitative measure of functional recovery following sciatic nerve injury and subsequent treatment.

Euthanasia and tissue collection

After the observation period, rats were euthanized under adequate sedation using cervical dislocation. The procedure was performed carefully to ensure rapid and humane termination. Gastrocnemius muscles from both the injured and contralateral limbs were excised carefully, and surrounding connective tissue was removed to prevent measurement bias. Muscle samples were immediately processed for subsequent weighing.

Muscle recovery analysis

The gastrocnemius muscles were weighed using an analytical balance with a precision of 0.001 g. Muscle recovery was assessed by calculating the GMI as the weight of the injured muscle divided by the weight of the contralateral muscle, multiplied by 100. The resulting value was expressed as a percentage, providing an objective measure of muscle recovery and atrophy after sciatic nerve injury and treatment intervention.

Statistical analysis

Normality of continuous variables was assessed using the Shapiro–Wilk test, and homogeneity of variances was evaluated using Levene's test. EPT data, which were normally distributed and homogeneous, were analyzed using one-way analysis of variance (ANOVA). When significant differences were detected, post-hoc Least Significant Difference (LSD) tests were applied. GMI data, which were not normally distributed, were analyzed using the Kruskal–Wallis test, followed by post-hoc Mann-Whitney U tests. Statistical significance was set at $p<0.05$. All data were processed and analyzed using IBM SPSS Statistics version 23 (IBM Corp., Armonk, NY, USA).

Results

Effect of UC-MSCs on motor function

One animal in the saline group did not complete the study because it died during the observation period. The motor function among three experimental groups: sham operation, saline control with sciatic nerve injury, and UC-MSC treatment after nerve injury, was assessed, and the results of the EPT test are summarized in **Table 1**. Rats in the sham group showed relatively stable hindlimb function, with a mean EPT of −0.82%±33.34%. Saline-treated rats had greater variability in EPT (14.65%±34.47%), reflecting heterogeneous motor responses after nerve injury. UC-MSC–treated rats had a mean EPT of −17.90%±39.02%, suggesting a trend toward improved postural extension compared to saline controls. Comparison across groups showed no significant differences in EPT (**Table 1**).

Table 1. Comparison of motor function across experimental groups assessed using the extensor postural thrust (EPT) test

Assessment	Study groups			*p*-value
	Sham surgery (n=10)	Saline treatment (n=9)	UC-MSC treatment (n=10)	
Extensor postural thrust (%), mean±SD	−0.82±33.34	14.65±34.47	−17.90±39.02	0.160[a]

UC-MSC: Umbilical cord–derived mesenchymal stem cells
[a] Analyzed using the ANOVA test

Effect of UC-MSCs on muscle structure recovery

For gastrocnemius muscle mass, the sham group had a median GMI of 89.66%, the saline group 81.68%, and the UC-MSC group 109.75%, indicating a potential protective effect of UC-MSC therapy (**Table 2**). Comparison across groups showed no significant differences in EPT, whereas GMI differed significantly ($p=0.012$), prompting pairwise post-hoc analysis (**Table 2**).

Table 2. Comparison of muscle recovery across experimental groups assessed using the gastrocnemius muscle index (GMI)

Assessment	Study groups			*p*-value
	Sham surgery (n=10)	Saline treatment (n=9)	UC-MSC treatment (n=10)	
Gastrocnemius muscle index (%), median (min–max)	89.66 (71.63–120.63)	81.68 (61.22–104.56)	109.75 (82.81–143.88)	0.012[a]

UC-MSC: Umbilical cord–derived mesenchymal stem cells
[a] Analyzed using the Kruskal–Wallis test

Post-hoc pairwise comparisons were performed for the GMI using Mann–Whitney U tests following a significant Kruskal–Wallis test (**Table 3**). After adjustment for multiple comparisons, GMI did not differ significantly between the sham operation and saline control groups, nor

between the sham operation and UC-MSC treatment groups. In contrast, the UC-MSC group had a significantly higher GMI compared with the saline control group (p=0.003) (**Table 3**), suggesting a protective effect of UC-MSC therapy on gastrocnemius muscle mass following sciatic nerve injury.

Table 3. Post-hoc pairwise comparisons for gastrocnemius muscle index (GMI) between study groups

Study group comparison	Mann–Whitney U	Z value	Adjusted p-value [a]
Sham vs saline	24.0	−1.715	0.950
Sham vs UC-MSC	31.0	−1.436	0.165
Saline vs UC-MSC	10.0	−2.858	0.003*

UC-MSC: Umbilical cord–derived mesenchymal stem cells
* Statistically significant at p=0.05

Discussion

This study demonstrated that UC-MSC administration following sciatic nerve injury significantly improved gastrocnemius muscle preservation, as reflected by higher GMI values compared with saline-treated controls, while no statistically significant differences were observed in motor function assessed by EPT. These findings indicate that UC-MSC therapy exerted a measurable structural benefit on denervated muscle but did not result in detectable functional motor improvement within the observation period.

The absence of significant differences in EPT among groups suggests that global motor recovery following peripheral nerve injury may require a longer time course to become functionally apparent [13]. EPT reflects integrated neuromuscular performance that depends on successful axonal regeneration, reinnervation of target muscles, and coordinated motor unit recruitment [14]. Variability in EPT values across injured animals further highlights the heterogeneous nature of functional recovery after sciatic nerve damage, which is influenced by injury severity and individual regenerative capacity.

In contrast, the significant improvement in GMI observed in UC-MSC–treated animals indicates that structural muscle changes are more responsive to early therapeutic intervention. Denervation-induced muscle atrophy is a well-recognized consequence of sciatic nerve injury, and GMI serves as a sensitive indicator of muscle mass preservation [15,16]. The higher GMI values in the UC-MSC group compared with saline controls suggest that UC-MSC therapy mitigated muscle wasting following nerve injury.

Mechanistically, the beneficial effects of UC-MSCs on muscle preservation are likely mediated through paracrine signaling rather than direct differentiation into muscle or neural cells [13, 15, 17]. UC-MSCs secrete a broad spectrum of bioactive molecules, including vascular endothelial growth factor (VEGF), insulin-like growth factor-1 (IGF-1), hepatocyte growth factor (HGF), and anti-inflammatory cytokines, which collectively support myofiber survival and reduce denervation-induced catabolism [18,19]. These factors have been shown to suppress ubiquitin-proteasome-mediated protein degradation pathways and attenuate oxidative stress, both of which contribute to muscle atrophy following loss of neural input [20,21].

In addition, UC-MSC–derived paracrine factors may indirectly enhance the muscle microenvironment by modulating local inflammation and promoting angiogenesis [22]. Improved microvascular perfusion and reduced inflammatory infiltration can preserve metabolic support to denervated muscle fibers, thereby slowing the progression of atrophy during the early post-injury phase [23]. Post-hoc analysis confirmed that the difference in GMI between UC-MSC and saline groups remained statistically significant after correction, supporting the robustness of this finding.

Taken together, the divergent outcomes between EPT and GMI underscore the multidimensional nature of recovery following peripheral nerve injury. Structural preservation of muscle appears to precede measurable improvements in motor function, suggesting that UC-MSC therapy may provide an early regenerative advantage that could facilitate later functional recovery [24,25]. This dissociation between structural and functional outcomes is consistent with

experimental models showing that muscle integrity can be maintained despite incomplete or delayed reinnervation [26].

Some limitations of this study should be acknowledged. First, the observation period was relatively short and may not have been sufficient to detect delayed functional recovery following peripheral nerve injury. Second, the assessment was limited to functional and muscle outcomes, without direct evaluation of nerve regeneration. Finally, molecular mechanisms underlying the observed muscle preservation were not examined. Future studies with longer follow-up and additional outcome measures are needed to better characterize the long-term effects of UC-MSC therapy.

Conclusion

UC-MSC therapy significantly preserved gastrocnemius muscle mass following sciatic nerve injury, whereas motor function assessed by EPT did not differ significantly among groups. These findings suggest that UC-MSCs confer early structural protection against denervation-induced muscle atrophy, which may precede measurable functional recovery. Further studies with longer follow-up periods that incorporate direct assessments of nerve regeneration, electrophysiological outcomes, and molecular mechanisms are needed to clarify the temporal relationship between structural preservation and functional recovery and to better define the therapeutic potential of UC-MSCs in peripheral nerve injury.

Ethics approval

The study protocol was reviewed and approved by the Ethics Committee of the Faculty of Veterinary Medicine, Universitas Syiah Kuala, Banda Aceh, Indonesia (Approval No. 439/KEPH/IX/2025).

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Competing interests

All the authors declare that there are no conflicts of interest.

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Underlying data

Derived data supporting the findings of this study are available from the corresponding author on request.

Declaration of artificial intelligence use

This study utilized artificial intelligence (AI) tools and methods to support manuscript preparation in three main areas: language enhancement, content summarization, and technical writing assistance. AI language model, including ChatGPT was employed to improve grammar, sentence structure, and readability. The authors critically reviewed and revised all AI-generated outputs to ensure accuracy, coherence, and alignment with the study's objectives. The final decisions, interpretations, and manuscript content reflect the authors' independent judgment and intellectual contributions.

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