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Sequential Regenerative Activation of Cavernosal Tissue using Low-intensity Extracorporeal Shockwave Therapy and Systemic Exosome Administration in Neurogenic Erectile Dysfunction

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Abstract

Introduction: Neurogenic erectile dysfunction (NED) results from injury to the cavernous nerves and is characterized by impaired nitrergic signaling, tissue hypoxia, endothelial dysfunction, and progressive fibrotic remodeling of the corpora cavernosa. Current treatments primarily address symptoms rather than underlying regenerative mechanisms.

Objective: The objective of the study is to propose a mechanistically guided regenerative therapeutic model combining low-intensity extracorporeal shockwave therapy (Li-ESWT) with systemic administration of mesenchymal stem cell-derived exosomes to promote neural repair and restore the cavernosal neurovascular microenvironment.

Materials and Methods: A conceptual therapeutic protocol is described based on the biological interaction between shockwave-induced mechanotransduction and chemotactic recruitment of regenerative extracellular vesicles through activation of the stromal-derived factor-1 (SDF-1)/C-X-C chemokine receptor type 4 (CXCR4) axis.

Results: Li-ESWT induces transient upregulation of angiogenic and chemotactic mediators including vascular endothelial growth factor, endothelial nitric oxide synthase, and SDF-1, generating a temporary regenerative microenvironment. Systemically administered exosomes expressing CXCR4 receptors may preferentially migrate toward tissues with increased SDF-1 expression, facilitating targeted delivery of regenerative signaling molecules capable of promoting neural regeneration, angiogenesis, and antifibrotic modulation.

Conclusion: Sequential activation of tissue mechanotransduction followed by systemic exosome administration may represent a biologically guided regenerative strategy for NED. This approach constitutes the conceptual basis for the PROSIN protocol, a multimodal regenerative therapy designed to restore penile neurovascular function.

Keywords: Neurogenic erectile dysfunction, Extracorporeal shockwave therapy, Exosomes, Regenerative medicine, Stromal-derived factor-1 C-X-C chemokine receptor type 4 axis, Cavernous nerve regeneration

Introduction

Neurogenic erectile dysfunction (NED) is a frequent consequence of cavernous nerve injury, particularly following radical prostatectomy or in the setting of long-standing metabolic disease such as diabetes mellitus [1,2].

Damage to the cavernous nerves disrupts nitrergic signaling pathways that regulate cavernosal smooth muscle relaxation, resulting in impaired penile hemodynamics and loss of spontaneous nocturnal erections [3,4]. Absence of physiologic oxygenation cycles leads to tissue hypoxia, oxidative stress, and progressive fibrotic remodeling of cavernosal and perineural structures [5-8].

Experimental and clinical studies have demonstrated that fibrosis may develop within 6–12 months after nerve injury associated with radical prostatectomy, while in poorly controlled diabetes, similar structural changes may develop over longer periods [8,9].

Conventional treatments for erectile dysfunction – including phosphodiesterase type-5 inhibitors [10,11], intracavernosal injections [12], and vacuum devices [13] primarily target symptomatic improvement of penile hemodynamics. However, these strategies do not directly address the underlying pathophysiological processes responsible for neural degeneration and fibrotic tissue remodeling.

In recent years, regenerative medicine approaches have emerged as promising strategies aimed at restoring tissue structure and function rather than merely improving erectile performance [14]. Among these, low-intensity extracorporeal shockwave therapy (Li-ESWT) [15-18] and extracellular vesicle-based therapies have gained increasing scientific attention [19-21].

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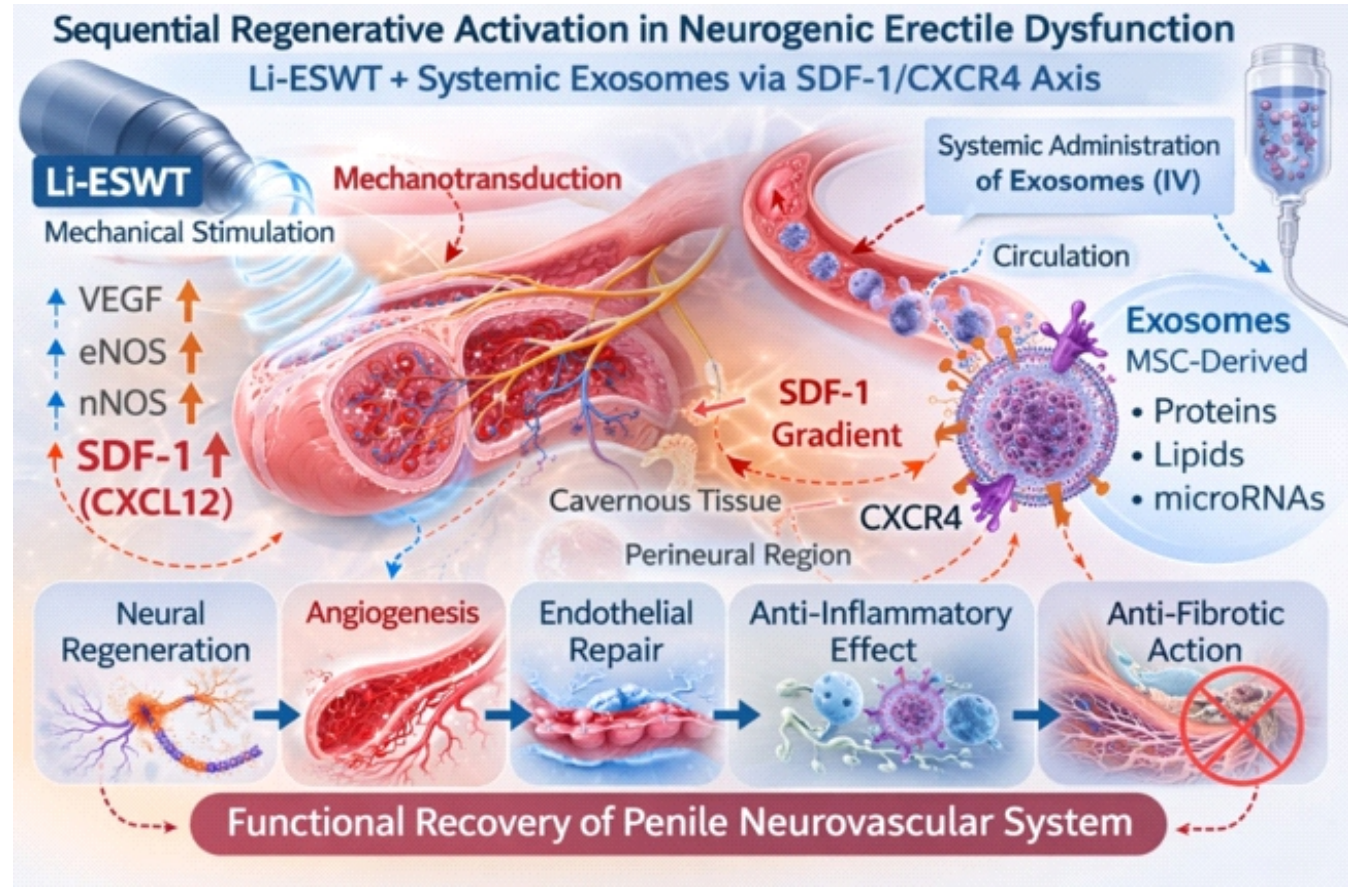

Figure 1: Sequential regenerative activation in neurogenic erectile dysfunction.

This study proposes a mechanistically integrated regenerative therapeutic model combining Li-ESWT-induced mechanotransduction with systemic administration of mesenchymal stem cell-derived exosomes, designed to enhance targeted regenerative signaling within cavernosal tissues.

Pathophysiological Rationale

The therapeutic objective of this report is not only to improve erection but also to restore the cavernous neurovascular microenvironment and promote neural regeneration before stable fibrosis occurs.

The proposed treatment involves combining Li-ESWT with systemic administration of uncharged exosomes, based on two complementary mechanisms:

1. Local activation of regenerative processes through mechanotransduction
2. Biological targeting (homing) of circulating exosomes to the treated tissue.

Therapeutic Biological Mechanisms

Mechanotransduction induced by shockwave therapy

Li-ESWT has been shown to activate cellular mechanotransduction pathways capable of inducing angiogenesis, endothelial repair, and neural regeneration [15-18,22-25]. Mechanical stimulation of tissues by acoustic shockwaves triggers a cascade of molecular responses that includes the release of angiogenic and neurotrophic factors [22,23], such as:

- Vascular endothelial growth factor (VEGF)
- Endothelial nitric oxide synthase (eNOS)
- Neuronal nitric oxide synthase
- Stromal-derived factor-1 (SDF-1)

These mediators promote endothelial activation, increased microvascular permeability, and recruitment of regenerative cells [22,23].

SDF-1 is a key chemokine involved in tissue regeneration and stem cell recruitment [26-28]. Its receptor, CXCR4, is expressed on a variety of regenerative cell populations, including mesenchymal stem cells and extracellular vesicles derived from them [29].

Upregulation of SDF-1 within injured tissues generates a chemotactic gradient that attracts CXCR4-expressing cells and vesicles [26-29]. This biological process, commonly referred to as "homing" [28,30,31], plays an important role in tissue repair by directing regenerative elements toward sites of injury. In this way, the tissue treated with Li-ESWT becomes a biological activator of the tissue microenvironment and a site of regenerative recruitment, favoring the uptake and local concentration of exosomes administered systemically, thus implementing a regenerative therapy guided by chemotactic gradients. Activation of the SDF-1/CXCR4 axis following Li-ESWT may therefore transform the treated cavernosal tissue into a biological recruitment site for regenerative vesicles and cells.

Therapeutic potential of exosomes

Exosomes are nanosized extracellular vesicles released by cells that function as mediators of intercellular communication [31-34]. Mesenchymal stem cell-derived exosomes contain a complex cargo of proteins, lipids, and regulatory microRNAs capable of modulating multiple regenerative pathways [33].

The mechanism of action of exosomes is multiple [31-35]:

- Promote axonal regeneration,
- Stimulate angiogenesis,
- Reduce apoptosis in smooth muscle cells,
- Modulate inflammatory responses,
- Inhibit fibrotic signaling pathways.

Preclinical and clinical studies have reported promising results, not only in erectile dysfunction [36,37] but also in other pathologies [38-40].

Proposed Treatment Protocol

Li-ESWT application: Mechanical stimulation of the corpora cavernosa

Shock waves are applied to the corpora cavernosa, crura, and perineal region to activate mechanotransduction responses that stimulate angiogenesis, endothelial repair, and neural regeneration.

Energy density: 0.15 mJ/mm2

Number of pulses per session: 2000 shocks.

Anatomical distribution:

- 500 distal penile shaft.
- 500 proximal penile shaft.
- 500 cavernosal crura.
- 500 perineal region.

Total number of sessions: 12 consecutive sessions.

Intravenous exosome administration

Infusion within the homing window, ideally 2 h after the Li-ESWT session (between 30 min and 4 h maximum).

Four intravenous infusions of exosomes are administered after the completion of the 1st, 6th, 9th, and 12th Li-ESWT sessions. This synchronizes the systemic delivery of exosomes with the tissue's regenerative activation windows.

Exosome dosage

This protocol proposes the administration of four doses of 100 billion exosomes per intravenous infusion. This is because, due to prior uptake in the lungs, liver, and spleen, only 5–10% (5–10 billion) will reach the target tissues.

The exosomes to be used in this proposed protocol would be unmodified exosomes (without additional microRNA loading) derived from mesenchymal stem cells obtained from periumbilical subcutaneous fat. These extracellular vesicles contain proteins, lipids, and microRNAs capable of modulating regenerative processes relevant to NED.

Circulating CXCR4-bearing exosomes interact with the SDF-1 gradient generated in the treated tissue, favoring their relative accumulation in the cavernous and pudendal territories.

Once internalized by cells of the cavernous tissue or the perineural environment, the exosomes can activate intracellular pathways related to neuronal survival and axonal growth, including PI3K-Akt, MAPK, and neurotrophic factors such as brain-derived neurotrophic factor, nerve growth factor, and glial cell-derived neurotrophic factor.

Integrated synergistic protocol (ISP)

Simultaneous implementation of an ISP including tadalafil, pentoxifylline, pirfenidone, curcumin, resveratrol, and melatonin, aimed at creating an oxygenated tissue microenvironment and interrupting the fibrogenic cascade, to reprogram gene expression in affected tissues from a profibrotic and pro-inflammatory state toward an antifibrotic and functional phenotype.

Treatment Rationale

The integration of Li-ESWT and exosome therapy represents a biologically coherent regenerative strategy that may address several key mechanisms involved in NED, including neural degeneration, endothelial dysfunction, inflammation, and fibrosis.

The objective of the therapeutic sequence is to synchronize the local release of regenerative signals induced by Li-ESWT-mediated SDF-1 expression with the systemic presence of CXCR4-bearing exosomes, thereby achieving targeted therapy directed toward injured tissues, even at sites that are not clinically detectable (Fig. 1).

While both Li-ESWT and extracellular vesicle therapies have independently demonstrated promising results in preclinical and clinical studies, their combined application within a coordinated regenerative framework has not yet been extensively explored.

Most LI-ESWT protocols published in the literature use weekly application schedules. However, from a tissue mechanobiology perspective, the regenerative response induced by Li-ESWT exhibits relatively short kinetics, characterized by a transient activation of molecular cascades that include the release of VEGF, eNOS, SDF-1, and various neurotrophic factors [22-25].

Several experimental studies have demonstrated that shockwave-induced mechanotransduction generates a temporary increase in angiogenic and chemotactic signals that peak within the first few hours after the mechanical stimulus, subsequently declining progressively. In this context, applying stimuli at excessively long intervals could allow the biological activation to decrease before the regenerative stimulus can be reinforced [22,23]. For this reason, a repeated stimulation scheme with short intervals, such as the three-session-per-week protocol, could promote a biological summation of mechanotransduction, maintaining the angiogenic, neurotrophic, and chemotactic pathways induced by Li-ESWT more sustained.

The serial stimulation model allows for the maintenance, over several weeks, of a tissue microenvironment characterized by the repeated expression of regenerative mediators such as VEGF and SDF-1. This potentially increases the recruitment of circulating reparative elements, including extracellular vesicles and systemically administered exosomes, which, by expressing CXCR4 receptors, will migrate to the injured sites where SDF-1 is located. Thus, the intensive protocol of 12 sessions distributed over 4 weeks could amplify the regenerative homing phenomenon, generating multiple biological windows for tissue uptake of exosomes and optimizing the interaction between mechanotherapy (Li-ESWT) and biological therapy (extracellular vesicles).

In this context, the therapeutic strategy is not based on an isolated

stimulus, but on cumulative regenerative stimulation, intended to keep the tissue repair microenvironment active throughout the therapeutic cycle.

Limitations

Several limitations must be acknowledged regarding the conceptual therapeutic model proposed in this report. First, the synergistic interaction between Li-ESWT and systemic administration of mesenchymal stem cell-derived exosomes remains largely hypothetical and is primarily supported by indirect experimental evidence. Although both modalities have independently demonstrated regenerative potential in preclinical and early clinical studies, robust data evaluating their combined use in erectile dysfunction are currently lacking.

Second, the biodistribution of intravenously administered exosomes represents an important biological constraint. A significant proportion of circulating extracellular vesicles may be sequestered in organs such as the lungs, liver, and spleen, potentially limiting the fraction of vesicles reaching the target cavernosal tissue. While the proposed chemotactic recruitment mechanism mediated by the SDF-1/CXCR4 axis may theoretically enhance tissue targeting, the magnitude of this effect in vivo remains uncertain.

Third, the optimal timing between Li-ESWT stimulation and exosome administration has not yet been established. The proposed interval of approximately 2 h is based on the transient kinetics of mechanotransduction-induced signaling molecules reported in experimental studies; however, the precise temporal dynamics of SDF-1 expression and its capacity to recruit circulating vesicles in human cavernosal tissue require further investigation.

Conclusion and Perspectives

The present study proposes a therapeutic model based on sequential regenerative activation of cavernosal tissue. In this model, Li-ESWT acts as an initial stimulus capable of activating cellular mechanotransduction processes and promoting local release of regenerative chemokines, particularly SDF-1.

This phenomenon generates a homing window mediated by the SDF-1/CXCR4 axis, which may facilitate tissue uptake of systemically administered exosomes.

Within this therapeutic approach, the use of unmodified mesenchymal exosomes represents the initial strategy, leveraging their intrinsic capacity to modulate neural regeneration, angiogenesis, and inflammatory regulation.

Future optimization through exosome enrichment with specific neuroregenerative microRNAs – such as miR-133b-3p, miR-21-5p, miR-124-3p, or miR-126-3p – using molecular engineering techniques is proposed as a potential evolution of this therapeutic model to enhance peripheral neural regeneration mechanisms.

The model proposed here provides a conceptual basis for future translational research and clinical trials investigating the synergistic interaction between mechanical stimulation and biologically guided regenerative therapies.

Declaration of patient consent: The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given the consent for his/ her images and other clinical information to be reported in the journal. The patient understands that his/ her names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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