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Exosome Therapy in Diabetic Foot Ulcers

REVIEW ARTICLE

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ABSTRACT

Diabetic foot ulcers are a significant global health problem and are associated with high morbidity and healthcare costs. In recent years, exosomes, small extracellular vesicles that play a role in intercellular communication, have attracted attention as promising therapeutic agents in the field of regenerative medicine. Exosomes can regulate the functions of target cells by carrying biologically active molecules, including proteins, cytokines, growth factors, lipids, DNA, mRNA, and microRNAs. In this review, the biological properties of exosomes, the pathophysiology of diabetic foot ulcers, and the potential therapeutic effects of exosomes in wound healing are discussed. This review suggests that exosomes represent a potent cell-free therapeutic approach that may support wound healing in diabetic foot ulcers by targeting multiple biological mechanisms. Future well-designed clinical studies are needed to clarify the role of exosomes in the treatment of diabetic wounds.

Keywords: Exosomes, diabetic foot ulcer, regenerative therapy, chronic wound, wound healing, extracellular vesicles

INTRODUCTION

Diabetic foot ulcers (DFUs) are among the most serious complications of diabetes mellitus and are associated with high morbidity, an increased risk of amputation, and a substantial economic burden on healthcare systems. With the increasing prevalence of diabetes worldwide, the management of diabetic wounds has become an increasingly important clinical problem. Although current treatment approaches include debridement, infection control, revascularization, and advanced wound care products, wound healing remains inadequate in many patients, and prolonged treatment durations may lead to limb loss.

The impairment of diabetic wound healing is driven by complex and interrelated pathophysiological mechanisms, including hyperglycemia, glycation, chronic inflammation, impaired angiogenesis, cellular dysfunction, and oxidative stress. This multifactorial nature often renders single-target therapeutic approaches insufficient (1).

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In recent years, exosomes, which are carriers of biologically active molecules that regulate intercellular communication, have emerged as a promising therapeutic option in regenerative medicine. As an alternative to cell-based therapies, exosomes have attracted attention due to their lower risks of immunogenicity and tumorigenicity and their translational potential, particularly in the treatment of chronic wounds. This narrative review discusses the biological properties of exosomes, the pathophysiology of diabetic foot ulcers, and the effects of exosome-based therapeutic approaches on wound healing drawing on the current literature.

WHAT ARE EXOSOMES?

Exosomes are nano-sized extracellular vesicles secreted by cells that play an important role in intercellular communication. These structures, typically 30–150 nm in diameter, enable the transfer of information between cells by carrying biological molecules, such as proteins, lipids, mRNA, and microRNA (2).

Exosome production is associated with the endosomal pathway within the cell. The process begins with the formation of early endosomes and continues with their transformation into late endosomes. Multivesicular bodies (MVBs) present within late endosomes form the basis of exosome biogenesis. These vesicles, originating from the endosomal system, are released into the extracellular environment when MVBs fuse with the plasma membrane, releasing the small vesicles they contain, which are then referred to as exosomes. These exosomes are taken up by target cells through endocytosis or membrane fusion (2,3). This mechanism may vary depending on the physiological state of the cell and is regulated by various intracellular and extracellular signals.

Exosomes can be produced by many different cell types. Immune system cells (myeloid cells such as monocytes, macrophages, neutrophils, and dendritic cells, as well as lymphocytes like B and T cells), epithelial cells, mesenchymal stem cells, and tumor cells are the main cell types capable of producing exosomes (4). Exosomes contain biologically active molecules such as proteins, lipids, DNA, mRNA, and microRNA, and they regulate cellular signaling pathways by transferring these molecules to target cells (4,5).

Recent studies have shown that exosomes have significant therapeutic potential in immune regulation, angiogenesis, cell proliferation, regenerative medicine, and tissue repair through their internal content (5). Exosomes derived especially from mesenchymal stem cells have attracted great interest in regenerative medicine and tissue repair studies. This is because these exosomes contain biomolecules with anti-inflammatory and immunomodulatory properties, and they are considered important candidates for cell-free therapeutic approaches, as they retain many advantages of stem cell therapy while posing a lower risk of tumor formation and immune reactions (6).

The biochemical properties of exosomes include specific molecular markers that distinguish them from other extracellular vesicles. Proteins from the tetraspanin family, such as CD9, CD63, and CD81, are among the characteristic surface markers of exosomes, and proteins such as Alix, TSG101, and flotillin are also frequently used biomarkers for exosome identification (7). The lipid composition of exosomes is also highly distinctive; they possess a membrane rich in lipids such as cholesterol, sphingomyelin, and ceramide. This structure enables exosomes to remain stable in the extracellular environment.

From a functional perspective, exosomes are considered important biological tools that facilitate intercellular communication. The RNA and proteins they carry can influence gene expression and signaling pathways in target cells. Through these properties, exosomes play roles in many physiological and pathological processes, including immune regulation, tissue regeneration, synaptic communication, and cancer development (2). Additionally, their presence in biological fluids such as blood, urine, and saliva has made them potential biomarkers for the early disease detection.

PATHOPHYSIOLOGY OF DIABETIC FOOT ULCERS

Diabetic wounds, particularly diabetic foot ulcers, are among the most important complications of diabetes mellitus and are classified as chronic wounds. Normal wound healing is a complex, orderly biological process comprising three main phases: inflammation, proliferation, and remodeling. However, in diabetic patients, metabolic changes due to hyperglycemia, microvascular damage, diabetic neuropathy, and cellular dysfunction disrupt the normal progression of this process, leading to delayed wound healing (8,9). The main mechanisms responsible for delayed healing in diabetic wounds include chronic inflammation, impaired angiogenesis, cellular dysfunction, and oxidative stress.

Chronic Inflammation

In normal wound healing, the inflammatory phase is short-lived and facilitates the removal of damaged tissue and the initiation of the healing process. However, in diabetic wounds, inflammation persists, resulting in a chronic inflammatory

environment. This condition leads to changes in macrophage phenotype distribution, causing pro-inflammatory M1 macrophages to become dominant. Impaired transition from M1 to anti-inflammatory M2 macrophages disrupts inflammation resolution and tissue repair processes. The increased secretion of cytokines such as tumor necrosis factor alpha (TNF-α) and interleukin-6 (IL-6) by M1 macrophages sustains the inflammatory response and delays wound healing (10,11). Additionally, chronic inflammation increases levels of matrix metalloproteinases (MMPs) and leads to the degradation of the extracellular matrix, thereby negatively affecting the formation of granulation tissue (12).

Impaired Angiogenesis

The formation of new blood vessels, or angiogenesis, is critical in wound healing for the delivery of oxygen and nutrients to the tissue. In diabetic patients, endothelial dysfunction caused by hyperglycemia reduces the efficiency of the angiogenesis process. High glucose levels impair endothelial cell functions and lead to structural changes in the vessel wall. Furthermore, decreased expression of vascular endothelial growth factor (VEGF) results in insufficient formation of new blood vessels (13). This condition leads to hypoxia in the wound area and delays the development of granulation tissue (13,14).

Cellular Dysfunction

Significant abnormalities are also observed at the cellular level in diabetic wounds. Fibroblasts, keratinocytes, and endothelial cells are the main cellular components of wound healing. However, the hyperglycemic environment and the chronic inflammatory microenvironment negatively affect the proliferation, migration, and differentiation capacities of these cells (15). Reduced fibroblast activity leads to insufficient collagen synthesis and delayed formation of granulation tissue. In addition, decreased migration of keratinocytes also slows the epithelialization process (10,11). As a result, re-epithelialization of the wound surface becomes difficult, and the wound may become chronic.

Oxidative Stress

Oxidative stress is another important factor in the pathogenesis of diabetic wounds. As a result of hyperglycemia, the production of reactive oxygen species (ROS) increases in cells. Elevated ROS levels disrupt cellular functions by causing lipid peroxidation in cell membranes, protein damage, and DNA damage, and may also impair immune cell function, thereby slowing wound healing (16,17). Moreover, oxidative stress further delays the wound healing process by enhancing the inflammatory response (16). The insufficiency of antioxidant defense mechanisms also contributes to the exacerbation of this process.

In conclusion, interconnected mechanisms such as chronic inflammation, impaired angiogenesis, cellular dysfunction, and oxidative stress significantly delay the wound healing process in diabetic wounds. In chronic wounds developing on this pathophysiological basis, the effectiveness of conventional treatment methods such as infection control, wound care, debridement, revascularization, offloading, and wound dressings remains limited. Healing rates reported with conventional treatments are approximately 24%-30% within 12-20 weeks, indicating that these approaches are still insufficient (18). These prolonged healing times highlight the challenges of managing chronic wounds. Therefore, regenerative products and alternative therapeutic approaches have emerged as actively investigated treatment options in the management of DFUs (19,20). Taken together, the complex pathophysiology of chronic wounds and the limitations of current treatments underscore the need for more effective, innovative therapeutic strategies.

MECHANISMS OF ACTION OF EXOSOMES IN CHRONIC WOUND AND DIABETIC WOUND HEALING

Exosomes are among the biological agents that have attracted attention in the field of regenerative medicine for the treatment of chronic wounds, especially diabetic foot ulcers. The therapeutic effects of these vesicles largely stem from their ability to deliver biomolecules that regulate intercellular communication to target tissues. In particular, exosomes derived from mesenchymal stem cells can accelerate the healing process by modulating various cellular mechanisms involved in different stages of wound healing (21).

The prolongation of the inflammatory phase in chronic wounds is a major factor in delayed healing. The anti-inflammatory effects of exosomes occur through the regulation of macrophage polarization. Various studies have shown that stem cell-derived exosomes promote the transition from the pro-inflammatory M1 macrophage phenotype to the anti-inflammatory and regenerative M2 macrophage phenotype (22). MicroRNAs and cytokine-regulating molecules carried by exosomes play an important role in this process. An increase in M2 macrophages facilitates the suppression of inflammation and the transition of the wound environment to the proliferative phase (23).

One of the important effects of exosomes in wound healing is the regulation of fibroblast activity. Fibroblasts play a central

role in the formation of granulation tissue and the synthesis of the extracellular matrix. However, in chronic wounds, fibroblasts exhibit reduced proliferative and migratory capacity and impaired functional activity. Exosomes can stimulate the production of collagen types I and III by increasing fibroblast proliferation. In addition, they support cellular organization in the wound bed by enhancing fibroblast migration. A significant portion of these effects has been shown to be associated with the activation of the PI3K/Akt and extracellular signal-regulated kinase (ERK) signaling pathways (24).

Angiogenesis is one of the most critical steps in chronic wound healing and is often impaired in diabetic foot ulcers. The effects of exosomes on endothelial cells promote the formation of new blood vessels, thereby accelerating wound healing. In particular, microRNAs contained in mesenchymal stem cell-derived exosomes have been shown to activate VEGF and other angiogenic signaling pathways. These mechanisms contribute to the reformation of the microvascular network by increasing endothelial cell proliferation, migration, and tube formation (25).

Another important effect of exosomes is the regulation of keratinocyte functions. Keratinocyte proliferation and migration are the main determinants of the epithelialization process. It has been shown that exosomes increase keratinocyte proliferation and accelerate the reformation of the epithelial layer by activating various cellular signaling pathways in epidermal cells. This effect may contribute to the correction of delayed epithelialization, especially in diabetic wounds (21).

In the diabetic wound environment, increased oxidative stress exacerbates cellular damage and negatively affects the healing process. The antioxidant mechanisms of exosomes also contribute to wound healing. Studies have shown that exosomes can reduce the formation of ROS and strengthen antioxidant defense systems. In this way, cellular stress is reduced, and a more favorable microenvironment for tissue regeneration is established (26,27).

Exosomes also regulate extracellular matrix remodeling. In chronic wounds, excessive activity of matrix MMPs can lead to tissue degradation. Exosomes have been shown to support matrix stability by regulating the balance between MMPs and their tissue inhibitors (TIMPs). This regulation facilitates the formation of granulation tissue while also potentially limiting the uncontrolled development of scar tissue (28,29).

In recent years, various bioengineering approaches have been developed to enhance the therapeutic effects of exosomes. In particular, hydrogel- or biomaterial-based carrier systems can increase their biological activity by allowing exosomes to remain longer at the wound site. These systems enable the controlled release of exosomes and provide continuous biological stimulation during wound healing. Experimental studies have demonstrated that exosome-loaded biomaterials can significantly enhance granulation tissue formation and angiogenesis in diabetic wound models (30).

Considering these biological and molecular properties, exosomes are thought to exert significant therapeutic effects in the diabetic wound environment, particularly where cellular dysfunction and chronic inflammation are predominant, through multiple mechanisms including the regulation of inflammation, fibroblast activation, angiogenesis, epithelialization, and the reduction of oxidative stress. These multifaceted effects make exosomes a promising biological therapeutic candidate for the treatment of chronic wounds and diabetic foot ulcers. Therefore, a better understanding of the pathophysiology of DFUs is of critical importance for the development of exosome-based therapeutic strategies (31). However, for this potential to be translated into clinical practice, it is essential to standardize production methods, establish optimal dosing and application protocols, and conduct large-scale clinical studies.

WHICH EXOSOMES IN CHRONIC WOUNDS AND DIABETIC FOOT ULCERS?

The current literature indicates that exosomes derived from mesenchymal stem cells (MSCs) have significant, multifaceted therapeutic effects in the wound-healing process. MSC-derived exosomes exert their effects through mechanisms such as regulating inflammation, stimulating angiogenesis, enhancing fibroblast proliferation and migration, and supporting extracellular matrix remodeling. These effects contribute to the restoration of impaired healing processes in chronic wounds, especially those associated with diabetes (25).

In particular, exosomes derived from umbilical cord mesenchymal stem cells (UC-MSCs) have been reported to show pronounced positive effects on wound healing due to their strong anti-inflammatory properties and high biological activity. These exosomes have been shown to suppress pro-inflammatory cytokines, promote macrophage polarization toward the M2 phenotype, and support tissue regeneration by enhancing angiogenesis (32). In addition, experimental studies have demonstrated that UC-MSC-derived exosomes accelerate the epithelialization process and increase granulation tissue formation (33).
Furthermore, exosomes derived from adipose tissue

mesenchymal stem cells (AD-MSCs) offer important advantages for clinical applications due to their high yield and the possibility of being obtained through minimally invasive methods. AD-MSC exosomes have similarly been shown to enhance angiogenesis, support fibroblast proliferation, and reduce oxidative stress. With these properties, they are considered a promising biological therapeutic option in terms of both efficacy and accessibility (27).

FUTURE PERSPECTIVES

Exosome-based therapies are receiving increasing attention in the field of regenerative medicine. In the future, it may be possible to develop exosomes enriched with specific microRNAs or growth factors through exosome engineering. Additionally, combining exosomes with biomaterials and hydrogel systems may allow them to remain longer at the wound site. Such strategies may significantly enhance the therapeutic efficacy of exosome-based treatments.

CHALLENGES IN CLINICAL APPLICATION

Exosomes are versatile biological agents capable of simultaneously influencing different phases of wound healing. They exert their effects through various mechanisms, including the regulation of inflammation, enhancement of cellular proliferation, promotion of angiogenesis, and reduction of oxidative stress. These multifaceted mechanisms make exosomes a promising therapeutic option, particularly in chronic wounds with complex pathophysiology.

However, several important challenges remain in translating exosome-based therapies into clinical practice. These challenges include the lack of standardized isolation methods, the absence of clearly defined dosage and application protocols, and the limited availability of long-term safety data. Furthermore, variability in the biological properties of exosomes derived from different cellular sources may lead to heterogeneity in therapeutic outcomes. Therefore, larger randomized controlled studies are needed for exosome-based therapies to be integrated into clinical practice.

CONCLUSION

Exosomes have emerged as a promising therapeutic option in the treatment of DFUs due to their multifaceted biological effects that support wound healing. By regulating inflammation, enhancing angiogenesis, promoting cellular proliferation, and reducing oxidative stress, exosomes demonstrate significant potential in the management of chronic wounds. Nevertheless, further randomized controlled studies and the establishment of standardized production and application processes are required to enable their successful translation into clinical practice.

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