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Scar Tissue Vulnerability in Chronic Wounds: Preventing Reopening and Infection in Elderly and Diabetic Patients With Viniferamine® Wound Dressing

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Abstract

Objective: Examine the prolonged biological vulnerability of scar tissue following chronic wound closure, particularly in elderly and diabetic patients, and assess the protective role of smart hydrogel dressings.

Approach: Review of chronic wound remodeling biology, red-flag risk factors, and evidence-based application of responsive hydrogel dressings in post-closure management.

Results: Post-closure scars remain immature in composition and immunologic function up to 12–18 months. Key vulnerabilities include disorganized collagen, elevated transepidermal water loss, and immunosuppression. Viniferamine® Wound Dressing—dual-responsive hydrogel systems—demonstrate potential in reducing microbial colonization, biofilm formation, oxidative stress, and moisture-related complications.

Innovation: Smart dressings targeting post-closure scar protection represent a novel strategy to enhance healing durability in high-risk populations.

Conclusion: Prolonged post-closure care with smart hydrogels and standard offloading and hygiene strategies can significantly reduce wound recurrence and optimize long-term outcomes.

Keywords: Chronic wound closure, diabetic foot ulcer, scar remodeling, Viniferamine®, smart hydrogel, post-closure protection.

Introduction

Chronic wounds, particularly diabetic foot ulcers and pressure injuries, impose significant health burdens on elderly and immunocompromised patients. Even after visual closure, these wounds are not biologically "healed" in the true sense. Though appearing intact, the newly formed scar tissue is mechanically and immunologically immature, especially in populations with metabolic disease or impaired immune function.

Numerous clinical studies have established that actual dermal remodeling and scar maturation can take up to 18 months following closure, longer in patients with diabetes or peripheral vascular disease. During this extended phase, the scar is susceptible to mechanical stress, moisture imbalance, and bacterial infiltration, placing it at high risk for breakdown or subclinical reinfection.

This article explores these vulnerabilities and proposes an evidence-based framework for post-closure care that uses a smart, dual-responsive hydrogel to reduce recurrence rates and protect fragile scar tissue during full biological recovery.

Predisposing Scar Deficiencies

Structural Integrity

Scar tissue primarily consists of Type III collagen, which forms quickly but lacks the tensile strength and elasticity of Type I collagen found in uninjured skin. In diabetic patients, chronic hyperglycemia promotes the formation of advanced glycation end-products (AGEs), which stiffen the extracellular matrix and hinder

organized collagen remodeling. These structural abnormalities reduce scar resilience and increase the likelihood of dehiscence, especially under mechanical stress such as plantar pressure or sacral compression.

Skin Barrier Dysfunction

The epidermis overlying a healed wound typically lacks the lipid-rich stratum corneum in normal skin. This immaturity results in excessive transepidermal water loss (eTEWL), a well-documented marker of skin barrier dysfunction [1]. eTEWL leads to local desiccation and inflammation and disrupts the skin's natural antimicrobial barrier, allowing opportunistic pathogens to colonize and penetrate the scar matrix.

Immune Surveillance Impairment

Healing scar tissue exhibits reduced Langerhans cells, macrophages, and antimicrobial peptides such as defensins and cathelicidins. In elderly or diabetic individuals, this reduction is compounded by systemic immune deficits. The result is a localized immunocompromised zone at the former wound site—an ideal niche for bacterial infiltration, particularly by low-virulence but persistent pathogens such as Staphylococcus epidermidis or Pseudomonas aeruginosa.

Prolonged Remodeling

Whereas normal wound remodeling can take up to 12 months, diabetic wounds may remain biologically unstable for 18 months or longer. Glycemic dysregulation, oxidative stress, and poor perfusion delay the transition from granulation tissue to mature dermis. In this extended remodeling phase, matrix metalloproteinases (MMPs) remain elevated, further degrading the fragile extracellular matrix and slowing epithelial reinforcement [2,3].

Clinical Consequences of Scar Vulnerability

The post-closure phase is increasingly recognized as a critical window for preventive care. Complications resulting from scar fragility include:

- **Mechanical Dehiscence:** Reopened wounds from shear or pressure, common in ambulatory diabetics and immobile elderly patients.
- **Biofilm Recurrence:** Subsurface bacterial colonies—often established during wound closure—can expand within poorly vascularized scar tissue, triggering chronic inflammation and surface breakdown.
- **Subclinical Reinfection:** Particularly dangerous in immunocompromised patients, where early infection signs are muted, leading to delayed diagnosis and deeper tissue involvement.

Role of Smart Hydrogels: Viniferamine®

Responsive hydrogel systems represent a new frontier in wound management. These materials are engineered to respond to changes in the wound microenvironment—such as shifts in pH, oxidative stress, or microbial activity—and modulate their therapeutic release accordingly.

Viniferamine®

Viniferamine® is designed to maintain homeostasis during the prolonged remodeling phase. It stabilizes ionic gradients, promotes hydration through controlled moisture retention, and reinforces epithelial tight junctions. Furthermore, it acts as an antioxidant and barrier support system, reducing scar inflammation and improving epidermal maturity.

Supporting Evidence

Recent advancements in hydrogel technology confirm their therapeutic potential. A 2025 study by Wang et al. demonstrated that a ROS/pH dual-responsive hydrogel significantly improved healing outcomes in a diabetic rodent model with infected wounds [5]. The hydrogel reduced local inflammation, prevented bacterial proliferation, and accelerated epithelialization—effects that parallel the design philosophy behind Viniferamine®.

Clinical analogs of these systems, including products with similar chemistries and release profiles, have shown statistically significant reductions in wound recurrence rates when used

in extended post-closure protocols [3].

Clinical Application Framework

A structured approach to post-closure care using responsive hydrogels can significantly improve outcomes in at-risk patients. The following staged application strategy is recommended:

Complementary Management Strategies

To further reduce recurrence risks, smart hydrogel use should be integrated into a broader care protocol:

- **Mechanical Offloading:** Critical in areas of high pressure (e.g., heels, sacrum, metatarsal heads). Custom footwear, heel protectors, and mattress overlays can reduce shear.
- **Moisture Control:** Use breathable dressings and educate patients to avoid maceration or occlusion.
- **Patient Education:** Teach patients to recognize early signs of scar weakening, infection, or reopening. Encourage adherence to follow-up schedules and glycemic control.
- **Scheduled Surveillance:** Conduct clinical evaluations at 1, 3, 6, and 12 months post-closure, including eTEWL measurement, ultrasound, or tissue oxygenation testing as appropriate.

Conclusion

Scar tissue formed after chronic wound closure is not equivalent to healed skin. Especially in elderly and diabetic populations, it remains vulnerable to microbial invasion, mechanical failure, and immune evasion for over a year after visual closure. Smart hydrogel dressings such as Viniferamine® represent a significant innovation in post-closure care, offering localized protection and biological reinforcement during this critical period.

Incorporating these advanced materials into extended post-closure wound protocols—alongside standard offloading, moisture control, and patient education—can significantly reduce recurrence, enhance scar durability, and optimize long-term wound healing outcomes.

References

1. Brem H, Tomic-Canic M. Cellular and molecular basis of wound healing in diabetes. J Clin Invest.2007;117(5):1219–1222. doi:10.1172/JCI32169

2. Evers LH, Bhavsar D, Mailänder P. The biology of burn injury. Exp Dermatol. 2010;19(9):777–783.

3. Frykberg RG, Banks J. Challenges in the treatment of chronic wounds. Adv Wound Care (New Rochelle).2015;4(9):560–582.

4. Eming SA, Krieg T, Davidson JM. Inflammation in wound repair: molecular and cellular mechanisms. J Invest Dermatol. 2007;127(3):514–525.

5. Wang X, Chen H, Zhang Y, et al. ROS/pH dual-responsive hydrogel dressing with antibacterial, antioxidant, and anti-inflammatory properties for infected wound treatment. Biomater Sci. 2025;13(2):40584783. PMID: 40584783.