Shahzaib Haider et al



CODEN [USA]: IAJPBB

ISSN: 2349-7750

INDO AMERICAN JOURNAL OF PHARMACEUTICAL SCIENCES

http://doi.org/10.5281/zenodo.3515720

Available online at: <u>http://www.iajps.com</u>

Review Article

DRESSING SELECTION IN CHRONIC WOUND MANAGEMENT: A REVIEW STUDY

¹Dr Shahzaib Haider,²Dr Bushra Sulaiman,³Dr Saira Majeed.

¹MBBS;Nawaz Sharif Medical College,Gujrat., ²MBBS;Khawaja Muhammad Safdar Medical College,Sialkot., ³MBBS;Punjab Medical College,Faisalabad.

Article Received: August 2019	Accepted: September 2019	Published: October 2019
Abstract		

Abstract:

This article discusses the characteristics of an ideal dressing to assist clinicians to select the appropriate dressing choices according to wound. There is no one dressing that is suitable for the management of all types of chronic wounds and few are suited for the treatment of a single wound during all stages of the healing cycle. Wounds should be assessed for necrosis and infection, which need to be addressed prior to selecting an ideal dressing. Moisture-retentive dressings include films, hydrogels, hydrocolloids, foams, alginates, and hydrofibers and are useful in a variety of clinical settings. Antimicrobial-impregnated dressings can be useful in wounds that are superficially infected or are at higher risk for infection. As wounds heal, the ideal dressing type may change, depending on the amount of exudate and depth of the wound; thus success in wound dressing selection hinges on recognition of the changing healing environment.

Corresponding author:

Dr. Shahzaib Haider,

MBBS;Nawaz Sharif Medical College,Gujrat.



Please cite this article in press Shahzaib Haider et al., Dressing Selection In Chronic Wound Management: A Review Study., Indo Am. J. P. Sci, 2019; 06(10).

INTRODUCTION:

Chronic wound is known as a break in the continuity of skin of long duration (6 weeks) or frequent recurrence wound. [1,2] in today's society, chronic wounds represent a major health care burden. Approximately 1% to 2% of individuals will be affected by leg ulceration during their lifetime, and this figure will likely increase as the population ages [3-5]. The associated costs are staggering. A recent article suggests that treatment costs for venous ulcers alone approach \$3 billion, accounting for a substantial portion of the total health care budget⁻⁶ Global wound care expenditures amount to \$13 to \$15 billion annually. [7]

A myriad of factors can delay wound healing. Chronic disease, vascular insufficiency, diabetes, neurologic defects, nutritional deficiencies, advanced age, and local factors such as pressure, infection, and edema can all impair healing. Wound care is a holistic endeavor that requires an accurate identification of the specific entities interfering with wound healing in a particular patient.

Circulation, nutrition, immunity status are required for normal wound healing. The process usually takes 3 to 14 days to complete and has three phases: inflammation, proliferation, and remodeling with wound contraction [8-10] (Fig 1).

Neutrophils and macrophages appear in the wounded area to phagocytize bacteria and debris during in the inflammatory phase. A functioning immune system and adequate supply of growth factors are necessary in this phase of wound healing. In the proliferative phase, fibroblasts produce a collagen matrix, new blood vessels invade the forming granulation tissue, and epidermal cells migrate across the wound surface to close the breach. Protein or vitamin deficiencies may impair normal wound healing through collagen deposition, remodeling, and wound contraction. [10] when any of the components of the wound healing process is compromised, healing may be delayed.

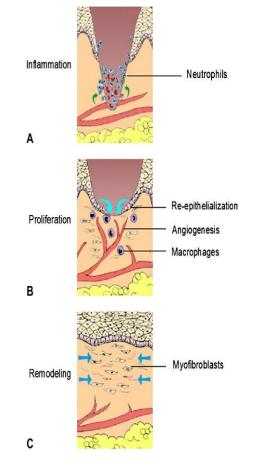


Fig 1. Normal wound healing. Physiologic wound healing occurs in three phases that may overlap in time. [9,10]

Considerations for Dressing Selection:

Before selecting a dressing, it is helpful to consider the underlying cause of tissue damage, tissue perfusion, and bacterial load. Chronic wounds due to venous insufficiency, diabetic foot ulcers, and pressure ulcers are difficult to heal without addressing the underlying tissue edema, poor perfusion, local pressure, immobility, and nutritional deficiencies. Tissue necrosis in wounds also impedes normal granulation tissue formation and requires debridement. Bacteria feed off necrotic tissue, so debridement can also decrease the risk of wound infection. Autolytic debridement occurs naturally through proteolytic enzymes in wound fluid, though that can be pH dependent [11]. If tissue autolysis is not apparent within 72 h, another form of debridement should be considered [12]. These options include surgical, mechanical, enzymatic, and biological methods.

Surgical debridement includes excision of eschars and removing devitalized tissue and necrotic debris, all of which can interfere with wound healing. A highly pressurized water jet system has also been developed for debridement as an alternative to traditional surgical techniques [13]. Mechanical debridement techniques include traditional salinemoistened gauze dressings, with removal of any hardened fibrinous exudate as the dressing dries. Enzymatic debridement uses chemical agents that can dissolve collagens and necrotic tissue. Collagenase (Santyl ointment, Healthpoint) is derived from a bacterium, Clostridium histolyticum, and works to digest collagen in dry ulcers when applied daily. This topical agent has been shown to improve endothelial cell and keratinocyte migration in animal studies [14].

MediHoney (Derma Sciences) is another alternative for debridement with a topical agent [15]. Biologic debridement with maggots is an ancient technique that has been performed for centuries, and recently resurged with the development of sterile maggots bred under aseptic conditions (Medical MaggotsTM; Monarch Labs). Maggots actually prefer to feed on necrotic tissue over viable tissue and secrete a proteolytic enzyme that liquefies dead tissue. They also secrete antimicrobial peptides (defensins) [15]. Enzymatic and biologic debridement may be preferred for patients who are poor surgical candidates or have lower extremity wounds that may heal poorly with invasive intervention. Assessment for infection prior to dressing selection is critical, although bacterial colonization does not necessarily mean infection. Low levels of bacteria can actually facilitate healing through production of proteolytic enzymes.

Bacteria encased in extracellular substances can form biofilms, which can contribute to chronic inflammation and failure of wounds to heal. Higher levels of bacteria, or a critical concentration of bacteria, tip the scales towards infection. The transition from colonization to infection can be inferred from progressive wound deterioration, breakdown of tissue, purulent exudate, warmth, erythema, increased pain, and increased swelling. The pathogens in infected wounds also change over time. Gram-positive and normal skin flora are found in acute wounds. Chronic wounds then become colonized by Gram-negative bacteria. Even later, deeper wounds harbor anaerobic flora. Wounds of several months duration can have on average four to five different pathogens [12].

Superficially infected wounds may be amenable to topical antimicrobials, dressings impregnated with antimicrobials, or cleansing with antiseptics such as polyhexanide, chlorhexidine, and triclosan. Deeper wounds that are infected often require debridement prior to dressing and systemic antibiotics if systemic infection is suspected.

Basic Dressing Options:

When underlying causes of tissue damage, tissue perfusion, and bacterial load have been carefully considered and addressed, a wound dressing will be most functional. Currently, there are a myriad of different dressings available. When selecting a dressing, one categorizes the wound based on standard characteristics: is the wound shallow or deep? Is there significant exudate? Dressing absorptive capacity should ideally match the wound's exudate generation and depth. Deeper wounds may require dressings that are available in filler form that can be lightly packed into any dead space. Additional considerations include if the patient can realistically take care of his or her wound. Dressings that are difficult to apply or require frequent changing may not be ideal for a patient who has no ancillary support. All dressings should protect wounds from further trauma or contamination. The ideal wound dressing should facilitate collagen synthesis and epithelial regeneration by removing deterrents in wound healing, including bacteria, exudate, external trauma, and other barriers (Table 1).

Table 1 Ideal wound dressing properties

General characteristics Easy to apply and maintain Aesthetically pleasing Cost permissive Easily stored Non-allergenic Facilitate healing Maintain moist environment Minimize trauma or maceration to wound edges Retention of heat Facilitates gas exchange Minimize risk of infection Debride necrotic tissue Absorb exudate Minimize external contamination

Moisture-Retentive Dressings:

As is commensurate with the data supporting their moisture-retentive dressings use, the have transformed the landscape of options for topical wound care. Understanding how to apply the different types of these dressings to clinical scenarios is an important skill for any wound practitioner. The moisture-retentive dressing options include films, hydrogels, hydrocolloids, foams, alginates, and hydrofibers.

Dressing	Table 2 Wound dressings and examples Clinical Applications	Example
		Zhampie
Moisture retentive		
Film	finor split-thickness skin graft donor sites Minor abrasions Intravenous access sites Occlusion for topical medication to improve absorption econdary dressings for hydrogels, foams, alginates First-degree burns Prevention of skin breakdown Stage 1 pressure ulcer	Bioclusive (Systagenix) Blisterfilm TM (The Kendall Co) Carrafilm TM (Carrington Laboratories) Kendall TM Polyskin TM II (Covidie)) Mepore Film (Molnlycke Health Care) Omniderm (Omidron Scientific Ltd) Opsite TM (Smith & Nephew) Tegaderm TM (3 M) Transeal (DeRoyal)
Hydrogel	Dry venous or arterial ulcers Calciphylaxis Coumadin necrosis Painful, non-exudative wounds	2nd skin (Spenco Medical, Ltd) Carrasyn (Carrington Laboratories Clearsite (ConMed Corporation) Elasto-Gel TM (SW Technologies) FlexiGel TM (Smith & Nephew) Hypergel (Molnlycke Health Care Kendall TM Curafil TM (Covidien) Kendall TM Curagel TM (Covidien) Normlgel (Molnlycke Health Care Nu-gel (Systagenix) Tegagel TM (3 M) Transigel TM (Smith & Nephew) Vigilon (C.R. Bard)

Hydrocolloid	Leg stasis ulcers Arterial ulcers Pressure ulcers Diabetic ulcers Partial-thickness burns Donor sites Skin abrasions Superficial acute wounds	Duoderm (ConvaTec) Comfeel (Coloplast) Cutinova (Smith & Nephew) Hydrocol II (UDL Laboratories) NuDerm (Systagenix) Replicare (Smith & Nephew) Tegasorb TM (3 M)
Foam	Wounds over bony prominences Mildly exudative wounds Donor sites	Allevyn (Smith & Nephew) Aquacel Foam (ConvaTec) Biatain (Coloplast) Biopatch (Johnson & Johnson Medical) Flexzan (UDL Laboratories) Kendall TM Curafoam TM (Covidien)) Kendall TM Hydrasorb (Covidien) Lyofoam (Molnlycke Health Care) Mepilex (Molnlycke Health Care) Polymem (Ferris Corp)

	Clinical Applications	Example
Dressing		
Alginate	eep and exudative pressure ulcers, pyoderma gangrenosum, diabetic wounds Bleeding wounds Donor sites	Algisite TM (Smith & Nephew) Algosteril (Systagenix) Kendall TM Curasorb TM (Covidien) Kalginate (DeRoyal) Kaltostat (ConvaTec) Melgisorb (Molnlycke Health Care) SeaSorb (Coloplast) Sorbsan (UDL Laboratories)
	eep and exudative pressure ulcers, pyoderma gangrenosum, diabetic wounds Traumatic wounds Partial-thickness burns	Aquacel (ConvaTec)
Hydrofiber Impregnated		
Silver	Superficially infected wounds	Acticoat TM (Smith & Nephew) Actisorb Silver 220 (Systagenix) Aquacel Ag (ConvaTec) Askina Calgitrol Ag (B. Braun) Silvercel (Systagenix) Silverlon (Cura Surgical)
Iodine	Superficially infected wounds	Inadine (Systagenix) Iodoflex TM (Smith & Nephew) Iodosorb TM (Smith & Nephew)

Shahzaib Haider et al

Honey Tissue an sincered	Superficial and partial-thickness burns	Medihoney (Derma Sciences)
Tissue engineered Epidermal grafts (autografts) Dermal replacement grafts	* Extensive deep dermal or full-thickness burns	Epicel (Genzyme Biosurgery)
Xenogeneic	Partial- and full-thickness wounds Vascular ulcers Pressure ulcers Surgical wounds Severe burns and burn scars (Integra TM)	OASIS Wound Matrix (Cook Biotech) EZ Derm (Molnlycke Health Care) Integra TM (Integra NeuroSciences) Biobrane TM (UDL Laboratories)
Allogeneic	Full-thickness diabetic ulcers, wounds related to dystrophic epidermolysis bullosa (Dermagraft)	Alloderm (LifeCell) GraftJacket TM (Wright Medical))ermagraft (Shire Regenerative Medicine)
Composite grafts (epidermal ? dermal)	Venous ulcers and full-thickness diabetic foot ulcers (Apligraf)	Apligraf (Organogenesis) OrCel TM (Ortec International)

Antimicrobial Dressings (Silver, Honey, Iodine)

Wounds that are superficially infected may benefit from dressings impregnated with antimicrobials. These dressings can kill bacteria on the wound surface or within the dressing for up to 7 days.

Ancient Romans used silver nitrate in wounds. Silver foil dressings were used for their antibacterial properties in the mid-1800s until World War II increased demand for surgical gauze dressings. Silver is considered a broadspectrum antimicrobial that can be used in superficially infected wounds. Silver particulates can be impregnated into hydrogels, alginates, foams, and even compression garments as well as other topical wound agents.

Iodine is also considered a broad-spectrum antimicrobial. Iodine can be used in two different forms, available as either a gel or sheet form. Povidone-iodine is an antiseptic that is impregnated into gauze.

Medical-grade honey can also promote autolytic debridement. Animal models have demonstrated accelerated wound healing with honey-treated wounds compared with conventional dressings. However, a more recent Cochrane review concluded that there is inconclusive evidence to fully support the use of honey in wound healing.

Tissue-Engineered Biologic Dressings:

Tissue-engineered biologic dressings are created to simulate natural scaffolding and matrices that are

formed during wound healing. The advancement of technology has allowed the development of cultured keratinocytes and fibroblasts to be incorporated into polymers to form biomaterials that function to replace tissue rather than solely facilitate wound healing. These tissue-engineered dressings essentially mimic autologous skin grafts but are advantageous through bypassing the creation of painful donor sites. Pinch grafting has been used as a substitute for splitthickness skin grafts as well but also requires donor site harvesting.

Epidermal Replacements is the only tissueengineered graft that requires a donor-site skin biopsy from the patient. A 1-cm sample of skin can grow enough epidermal autograft to cover most of the entire body.

Dermal Replacements Dermal replacements can be xenogeneic or allogeneic. These dressings are typically composed of collagen and additional extracellular matrices components including fibroblasts, glycosaminoglycans, and growth factors. Xenogenic grafts are typically made from porcine or bovine collagen. E-Z Derm (Molnlycke Health Care) and BiobraneTM (UDL Laboratories) are acellular matrices derived from porcine-derived collagen.

CONCLUSIONS:

Wound care has advanced significantly in the last century, providing practitioners with tools to treat each wound based on its unique properties. Wounds should be assessed for necrosis and infection prior to selecting an ideal dressing. Familiarity with the types of moisture-retentive dressings allows the practitioner to select the dressing that addresses the level of drainage and depth of the wound. For refractory wounds that do not respond to moisture-retentive dressings, tissue-engineered grafts have become a viable option in the past few decades, especially those that have been approved for burns, venous ulcers, and diabetic ulcers. In addition, the adjunctive antimicrobial dressing options continue to expand, providing practitioners with new tools for keeping infection at bay.

REFERENCES:

- Fowler E. Chronic wounds: an overview. In: Krasner D, editor. Chronic wound care: a clinical source book for healthcare professionals. King of Prussia, PA: Health Management Publications, Inc; 1990. pp. 12-8.
- Singh A, Halder S, Menon GR, Chumber S, Misra MC, Sharma LK, et al. Meta-analysis of randomized controlled trials on hydrocolloid occlusive dressing versus conventional gauze dressing in the healing of chronic wounds. Asian J Surg 2004;27:326-32.
- 3. Rees RS, Hirshberg JA. Wound care centers: costs, care, and strategies. Adv Wound Care 1999;12:4-7.
- 4. Callam M. Prevalence of chronic leg ulceration and severe chronic venous disease in Western countries. Phlebology 1992;7:S6-S12.
- Nelzen O, Bergqvist D, Linghagen A. The prevalence of chronic lower-limb ulceration has been underestimated: results of a validated population questionnaire. Br J Surg 1996;83:255-8.
- Bergan JJ, Schmid-Scho"nbein GW, Coleridge Smith PD, Nicolaides AN, Boisseau MR, Eklof B. Mechanisms of disease: chronic venous disease. N Engl J Med 2006;355:488-98.
- Walmsley S. Advances in wound management: executive summary. In: Clinica reports. London: PJB Publications, Ltd; 2002.
- 8. Goldman R. Growth factors and chronic wound healing: past, present, and future. Adv Skin Wound Care 2004;17:24-35.
- Baum CL, Arpey CJ. Normal cutaneous wound healing: clinical correlation with cellular and molecular events. Dermatol Surg 2005;31:674-86.
- 10. Singer AJ, Clark RA. Cutaneous wound healing. N Engl J Med 1999;341:738-46.
- Greener B, Hughes AA, Bannister NP, Douglass J. Proteases and pH in chronic wounds. J Wound Care. 2005;14(2):59–61.
- 12. Schultz GS, Sibbald RG, Falanga V, Ayello EA,

Dowsett C, Harding K, et al. Wound bed preparation: a systematic approach to wound management. Wound Repair Regen. 2003;11(Suppl 1):S1–28.

- Granick M, Boykin J, Gamelli R, Schultz G, Tenenhaus M. Toward a common language: surgical wound bed preparation and debridement. Wound Repair Regen. 2006;14(Suppl 1):S1–10. doi:10.1111/j.1743-6109.2005.00096.x.
- Demidova-Rice TN, Geevarghese A, Herman IM. Bioactive peptides derived from vascular endothelial cell extracellular matrices promote microvascular morphogenesis and wound healing in vitro. Wound Repair Regen. 2011;19(1):59– 70. doi:10. 1111/j.1524-475X.2010.00642.x
- 15. Derma Sciences I. http://www.dermasciences.com/products/ advanced-wound-care/medihoney.