Advanced Wound Dressings Today

Clinical Insights, Perspectives and Treatment Approaches

Editorial Board

Editor's Welcome

GREETINGS,

Welcome to this special supplement entitled Advanced Wound Dressings Today. We are pleased to make this supplement available to you as we believe the use of advanced wound dressings in today's wound care setting may bring potential for improvement in patient outcomes versus traditional modalities such as gauze and tape.

This issue contains insightful perspectives from our faculty that cover topics such as the use of the CELLUTOME™ Epidermal Harvesting System and the SNAP™ Therapy System as part of a wound management strategy for stalled, chronic wounds, the role of advanced wound dressings across the continuum of care and a discussion on chronic wound biochemistry---just to name a few.

As always, we hope these topics provide practical insight for you as you treat patients with a variety of wounds. We welcome your feedback about this supplement and encourage you to share the content with your peers who may not have received this peer-to-peer educational piece.

Thank you,

Ronald P. Silverman, MD, FACS | Acelity

Senior Vice President and Chief Medical Officer

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Chronic Wound Biochemistry

LEGEND

PDGF	Platelet Derived Growth Factor
IGF-1	Insulin-like Growth Factor 1
EGF	Endothelial Growth Factor
TGF-β	Transforming Growth Factor Beta
TNF- α	Tumor Necrosis Factor Alpha
β-FGF	Fibroblast Growth Factor Beta
IL	Interleukin

By: Robert Snyder, DPM, CWS, MS
Cherison Cuffy, DPM, CWS
Joey K. Ead, MS
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A wound is a disruption of normal anatomic structure and function that is usually inclusive of the skin. Wound healing represents a comprehensive series of physiological and biochemical reactions that create an orderly healing cascade. This process includes four key phases: hemostasis, inflammation, proliferation and remodeling. Wound healing is a dynamic response that progresses along a continuum that should result in the restoration of anatomical and function integrity.

HEMOSTASIS

Any injury extending beyond the boundaries of the epidermis will cause bleeding. This phenomenon activates a series of overlapping events designed to control blood loss, seal the defect, and establish bacterial control. The disruption of blood vessels and exposure of the sub-endothelial environment (collagen) catalyzes platelet aggregation.[1] Furthermore injured cells trigger both extrinsic and intrinsic coagulation pathways. Clot formation protects and seals the disrupted vessels so that blood loss is controlled.

Clot formation serves several key functions including:[1]

- Temporary bacterial barrier
- Interim matrix/scaffold for migrating cells
- Reservoir of growth factors

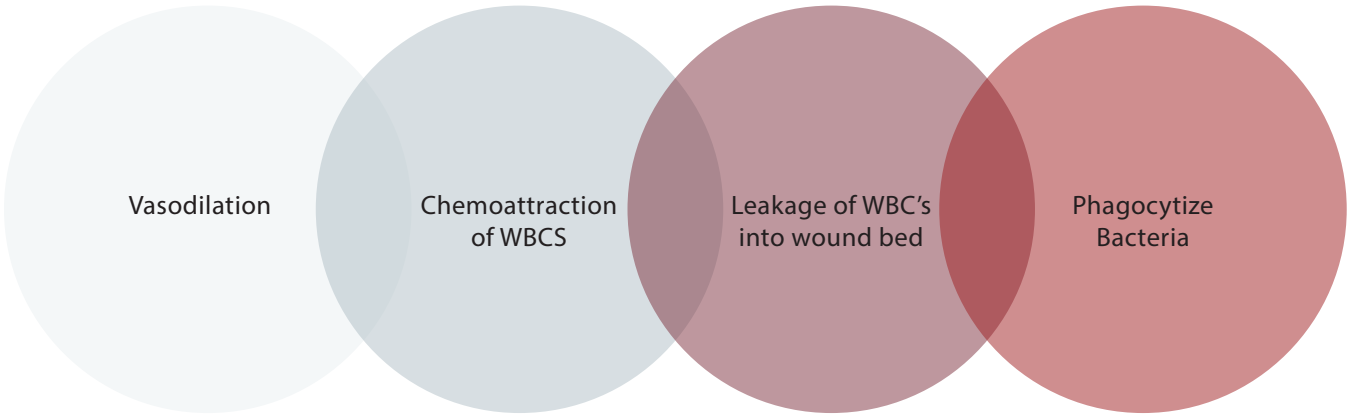
Fibrinolysis is a vital process that occurs after clot formation. Platelet degranulation releases an assortment of energy producing cytokines and growth factors.[1] The inflammatory phase is initiated by several growth factors including PDGF, IGF-I, EGF, TGF-β, and TNF alpha.[1]

INFLAMMATION

Once the hemostatic process is finalized, secretion of pro-inflammatory cytokines and proteases (elastase/collagenase) help remove damaged extracellular matrix (ECM).[2] The ECM gives skin its unique properties of elasticity, tensile strength, and compressibility. In acute wounds the provisional wound matrix (Fibrin; fibronectin) provides a scaffolding to direct cells into the injury and stimulate proliferation, differentiation, and synthesis to form a new ECM.

Before a wound can rebuild its damaged apparatus, breakdown of devitalized tissue and elimination of excess bacteria permeating the wound bed is paramount.[2] Leukocyte migration out of the vessels and into the wound bed is conducted via diapedsis.[1] Teams of white blood cells (WBC) infiltrate the wound environment to help stabilize the bacterial environment and establish a clean wound bed (Figure 1).[2] The principal leukocyte involved during this process is polymorphonuclear cells (PMN) also known as neutrophils. Their primary role is to eliminate deleterious bacteria and foreign debris via phagocytosis. Cell adhesion molecules (CAM) promote the binding mechanism between neutrophils and damaged tissues. Neutrophils release essential growth factors that attract additional leukocyte support. By days 3 to 4 after tissue damage, neutrophils disappear via apoptosis and are replaced by activated macrophages.[1] They continue to phagocytize bacteria and break down devitalized tissue. In addition, important pro-inflammatory cytokines are released from macrophages which include: TFG-beta, B-FGF, TNF-alpha, PDGF, IL-1 and IL-6.[1] It should be noted that lymphocytes are among the last cells to infiltrate the wound bed.[1] They release IL-2 which help recruit fibroblasts. By removing these impediments, the wound healing process can easily transition into the proliferative and rebuilding phases.

Figure 1: Inflammatory Phase[1]



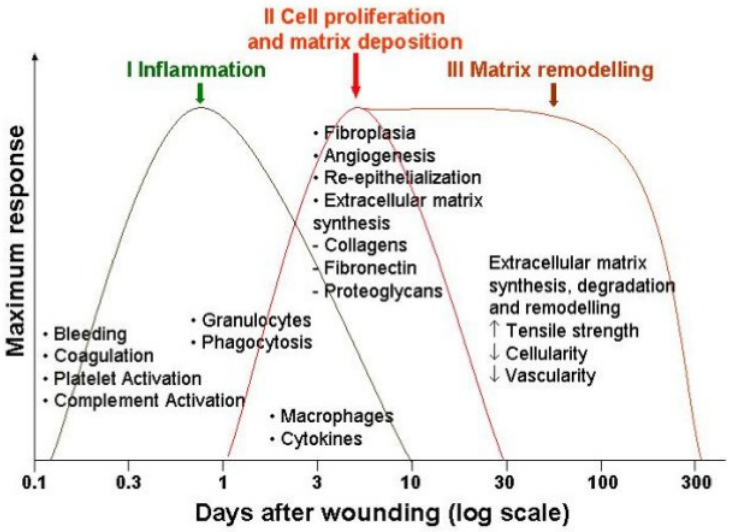
PROLIFERATION

The third essential phase of wound healing is the proliferative phase. Fibroblast propagation dominates this wound-healing segment.[2] PDGF that has been released in the preceding phases stimulate fibroblastic chemo-taxis and collagenase production. Fibroblasts also foster the integration of matrix metalloproteinase's (MMPs), which facilitate the permeation of these cells within the ECM environment. MMPs remove impaired collagen and other structural proteins while fibroblasts establish a healthy ECM network.[1] Several growth factors stimulate the production of new vessels that are generated by intrinsic cells within the wound bed.[1] Vascular endothelial cells, fibroblasts, epidermal cells, and macrophages contribute to angiogenesis by the production of β-FGF, TGF-β and VEGF.[1] The wound surface is covered with new epithelium that is able to restore bacterial barriers and vascular integrity.[2] Keratinocytes proliferate, migrate and differentiate during this phase.[1, 2] Furthermore, proliferative mechanisms continually promote development of granulation tissue, neo-angiogenesis, matrix deposition/collagen synthesis and epithelialization among others.[3]

REMODELING

A hallmark feature of the remodeling phase is the maturation of collagen fibers via crosslinking. This promotes increased tensile strength and fibroblast synthesis (Figure 2).[1] Collagen fibers steadily condense and in conjunction with myofibroblasts, become oriented parallel to the wound bed along lines of stress, resulting in the appearance of striated scar tissue.[1, 2] Contraction of the newly developed ECM could potentially manifest.[2] It should be noted the regenerated tissue is not as tensile and dynamic prior to injury.[2]

Figure 2: Wound Healing Cascade[1]



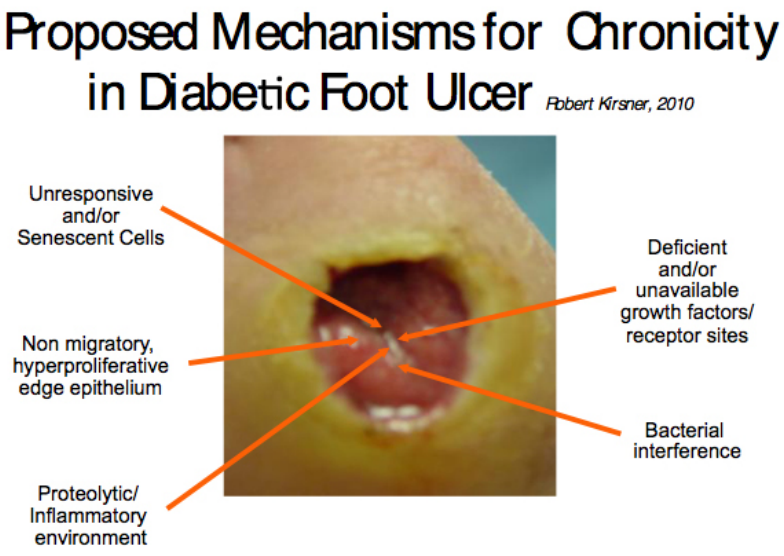
BIOCHEMISTRY OF CHRONIC WOUNDS

Stalled wounds consist of volatile biochemical mechanisms including increased proteases and inflammatory mediators, unresponsive/senescent cells, hyper-proliferative wound edges (neuropathic ulcerations) and bacterial interference (Figure 4). Nonviable tissue deleteriously effects the wound environment by fostering bacterial growth and local tissue hypoxia. In order to combat stagnant wound environments, infection and inflammation need to be mitigated. To end these physiologic obstacles, Robson et al discussed the distinctive factors on how bacterial infections and excess granulation tissue impact wound healing.[4] They noted that the existence of chronic granulation tissue decreased the amount of antibiotics that reached the wound infection, consequently prolonging the healing process.[4] Gardner et al assessed the reliability of clinical tools to evaluate the signs and symptoms of localized infections in chronic wounds.[3] Researchers established the "Clinical Signs and Symptoms Checklist," which centers on primary and secondary signs of infection to be reliable (Figure 3).[3] Clinicians should diagnose infection based on the presence of at least two classic symptoms: inflammation or purulent secretions.[2] Incorporating a structured methodology to monitor and assess wound infections may improve accuracy in identifying this condition.

Figure 3: Clinical Signs and Symptoms Checklist (CSSC)[3]

CSSC	DESCRIPTION
Pain	• Pain not detected in ulcer area • Less ulcer pain than in past • Pain remained the same since ulcer development • More ulcer pain than patient had initially
Erythema	Presence of bright or dark red skin or darkening of normal ethnic skin color immediately adjacent to the ulcer opening.
Edema	Assess pitting edema by firmly pressing the skin within 4 cm of ulcer margin with finger, releasing, and waiting 5 seconds to observe indentation.
Heat	Increase in skin temperature of skin adjacent to the ulcer but within 4 cm of the ulcer margin. Assess differences in skin temperature using back of the clinicians hand or wrist.
Purulent exudate	Presence of tan, creamy, yellow or green thick fluid on a dry gauze dressing removed from the ulcer 1 hour after placement
Sanguineous drainage	Presence of blood fluid on dry gauze dressing
Serous exudate	Presence of thin, watery fluid on a dry gauze dressing
Delayed healing of the ulcer	Clinicians report no change or an increase in surface area or volume of the ulcer over the past 4 weeks.
Discoloration of granulation tissue	Granulation tissue that is pale or dull in color
Friable granulation tissue	Granulation tissue bleeding easily when gently manipulated
Pocketing at base of wound	Non-granulating pockets of ulcer tissue surrounded by red granulation tissue
Foul odor	Distinctively unpleasant smell
Wound breakdown	Small open areas in newly formed epithelial tissue (Not injury induced)

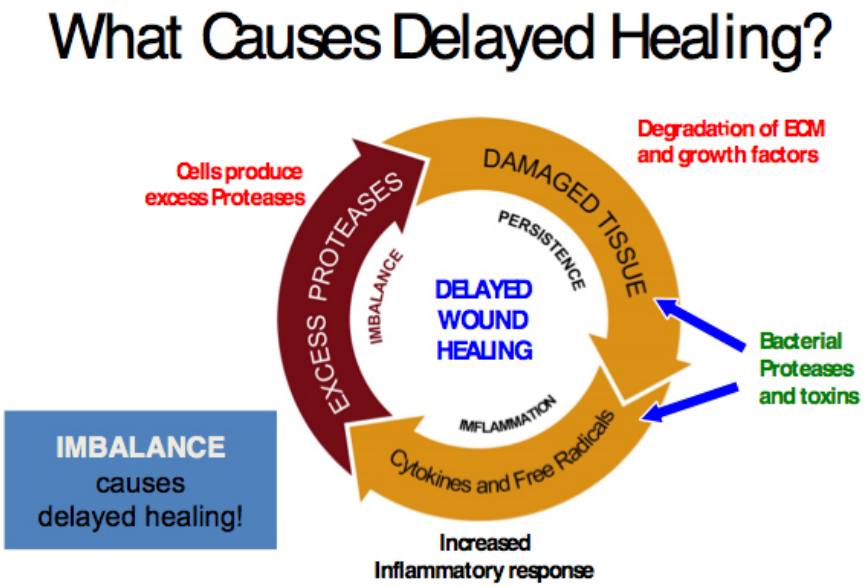
Figure 4: Proposed Mechanism of Chronic Wounds[2, 5]



Falanga V. The Chronic Wound: Impaired Healing and Solutions in the Context of Wound Bed Preparation. *Blood Cells and Diseases*, 2004;32:88-94

Matrix metalloproteinases (MMP's) represent a class of 20 protein-degrading enzymes.[6] It should be noted that proteases are important during the remodeling phase of wound healing by harmonizing ECM formation. Numerous investigations have revealed that high concentrations of serine protease in neutrophils and elevated MMP's are responsible for the degradation of ECM proteins, cytokines, growth factors, and cell surface receptors (Figure 5).[7]

Figure 5: Excess Protease Activity[8, 9]



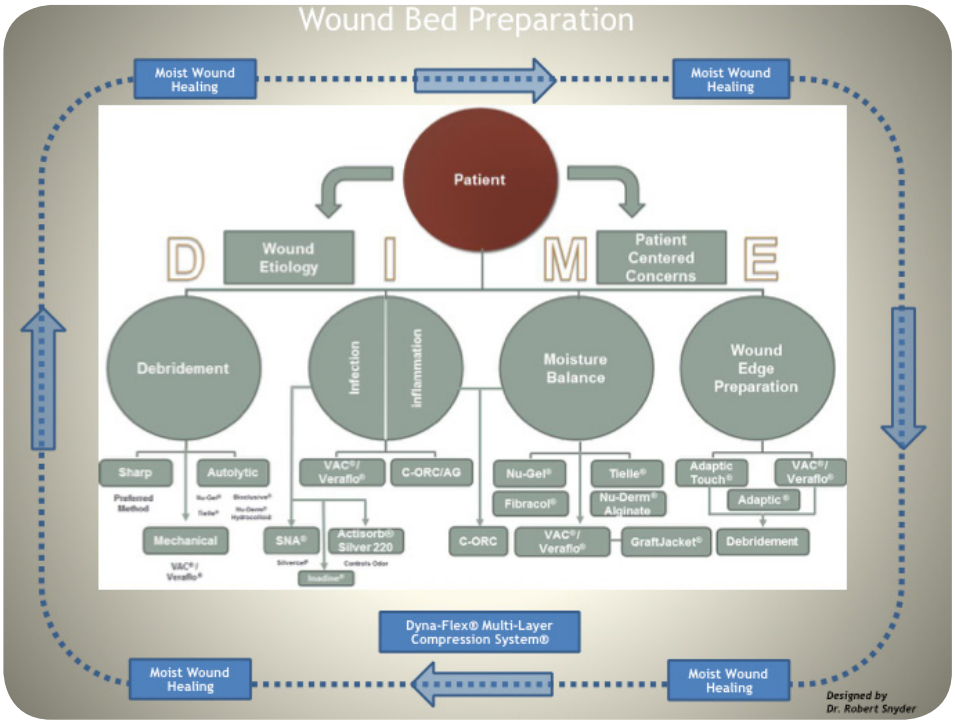
Wysocki AB, Staniano-Coico L, Grinelli F. Wound Fluid from Chronic Leg Ulcers Contains Elevated Levels of Metalloproteinases MMP-2 and MMP-9. *J Invest Dermatol.* 1993;101;64-68. Harris IR, Yee KC, Walters CE, Cunliffe WJ, Kearney JN, Wood EJ, Ingham E. Cytokine and protease levels in healing and non-healing chronic venous leg ulcers. *Exper Dermatol.* 1995;4:342-349.

During normal physiological wound healing processes, there is a burst of protease activity at the start of acute wound healing. However, if increased protease activity is prolonged they have the propensity to degrade the ECM and newly formed tissue.[10] In fact, some studies have discovered that increased quantities of specific MMPs (2, 8, 9) and human neutrophil elastase (HNE) are frequently found in chronic wounds in contrast to acute pathologies.[10] The deleterious effects of these proteases may stimulate the inflammatory response and discharge harmful reactive oxygen species. There are elevated levels of TNF-α and IL-1β. These molecules induce their own synthesis to set up a cycle of non-progressive inflammation, inhibit collagen formation, and decrease the production of protease inhibitors. It should be noted that the bi-directional communication network between cells and their surrounding ECM are compromised in chronic wound environments.[11] This concept is known as dynamic reciprocity (DR), a term first coined by Schultz et al.[11] This interaction has been extensively studied and applied to the physiological process of wound healing. Several therapeutic modalities target this mechanism.[11]

TREATMENT

The "DIME" scheme is an all-encompassing modality for proper wound bed preparation.[12] DIME is a mnemonic that incorporates debridement, infection control, moisture balance, and wound edge preparation (Figure 6).[12]

Figure 6: DIME & Wound Bed Preparation[12]



DIME strategy helps accelerate endogenous healing mechanisms by eliminating factors that obstruct the wound-healing sequence. Debridement treatments are administered to reduce exudate and/or necrotic tissue from the wound. These external modalities include; mechanical, sharp/surgical, enzymatic debridement, and negative-pressure wound therapy (NPWT) among others.[12] Additionally, passive treatments promote the patient's intrinsic healing

mechanisms to help reduce or eliminate impediments to healing.[12] Some passive treatments include hydrogels (hydro-polymer dressings), hydrocolloid dressings, autolytic debridement, antimicrobial or antiseptic dressings, collagen-oxidized regenerated cellulose dressings and non-adherent silicone dressings.[12]

CONCLUSION

Wound management is a specialty that is continuously evolving; wound specialists have a range of treatment options that include biologic skin substitutes, collagen matrices, negative pressure and active dressings among others. It is vital clinicians routinely base their treatment plans on evidence-based research. Additionally, mastery of wound science will lead clinicians to proper treatment pathways.

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Dr. Robert Snyder is Professor and Director of Clinical Research and Fellowship Director in wound care and research at Barry University SPM. He has created and implemented the first post residency Wound Management and Research Fellowship for podiatric medical schools. He is certified in foot and ankle surgery by the American Board of Podiatric Surgery and is also a board certified wound specialist. Dr Snyder is past-president of the Association for the Advancement of Wound Care and past-president of the American Board of Wound Management, the certifying body for Wound Care Specialists. In addition to his doctorate, he holds an MSc in Wound Healing and Tissue Science from Cardiff University. His expertise at Cardiff, Wales, was further acknowledged as he was selected as an Internal Marker for MSc dissertations with an Honorary Title. To constantly expand his knowledge and stay current in all aspects of healthcare, he is completing an MBA in Health Management. Dr. Snyder is a key opinion leader and sought after speaker, lecturing extensively throughout the United States and abroad. He was chosen to develop and teach a wound care course for physicians internationally. Dr. Snyder has published several book chapters and over 150 papers in peer reviewed and trade journals on wound care. He serves on the editorial advisory boards of Ostomy Wound Management, Wounds and Podiatry Management and has recently been appointed as a periodic reviewer for the Lancet and NEJM. He has been a Principal Investigator on more than 50 randomized controlled trials for innovative wound healing modalities and products.

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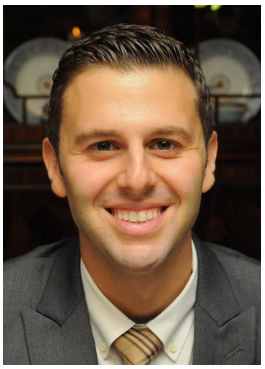
Dr. Cherison Cuffy is a graduate of Temple University School of Podiatric Medicine. He then completed a three-year podiatric medicine and surgery residency at Northwest Medical Center in Margate Florida. Dr. Cuffy is currently faculty at Barry University School of Podiatry as an assistant professor. He now participates in the clinical education of the podiatry students along with a focus on clinical research at the Paul and Margaret Brand Research Center.



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Advanced Wound Dressings in the Continuum of Care

Note: As with any case study, the results and outcomes should not be interpreted as a guarantee or warranty of similar results. Individual results may vary, depending on the patient's circumstances and condition.

By: Katherine Lincoln, DO, FAAFP

Chronic wounds are challenging for all wound care providers. Knowledge of wound care science has evolved significantly in the last 20 years. Now, providers of wound care have so many choices of materials and products, each with its own specific wound specific properties. As a patient travels through the wound treatment journey, the wound care provider is able to move through a continuum with the patient.[2] As the wound closes and changes, so should the products used to treat the wound.

In this article, two case reports will be reviewed, demonstrating the importance of choosing a product to address the state of the wound over a time period encompassing multiple weeks.

Case 1

This 42 year old male with diabetes and diabetic neuropathy presents to the outpatient wound care center (WCC) with his right great toe on the plantar surface. His medical history is also significant for coronary artery disease and hypertension. The wound has been present for 5 months and the patient has completed a course of TMP/SMX (trimethoprim/sulfamethoxazole) from another provider without wound resolution. The wound measures 4cm x 2cm x 0.4cm at the initial assessment at the WCC. (Figure 1-A)

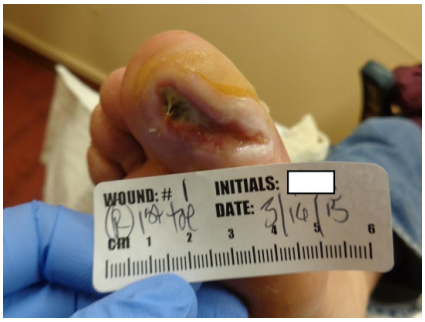

Figure 1-A

With the patient's medical history in mind, an ankle-brachial index (ABI) was done in the office and showed a 0.9 ratio bilateral, suggesting reasonable arterial flow to this toe wound. After vascular assessment, the necrotic tissue was addressed. Some of the thick adherent, yellow fibrin was removed with scissor and forceps. The wound base could not clearly be seen and a topical collagenase ointment was chosen for topical debridement. Topical collagenase was added to the wound daily during this period. A total contact cast is the gold standard for a diabetic toe ulcer, but this patient refused application.[1] A medical equipment shoe was chosen for off-loading, although this is neither ideal nor the preferred approach.

The wound was cultured at the initial visit and was found to have no pathologic bacteria after 7 days. Diabetic foot wounds often develop infections so high clinical suspicion of infection should be maintained throughout the wound care journey.

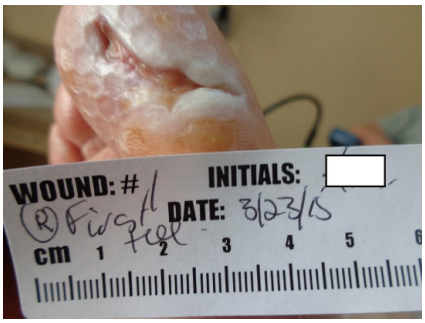

Figure 1-B

Figure 1-B shows the wound at one week into treatment at the WCC. Topical debridement was used daily for 1 week and one can note that the fibrin burden as well as the overall size of the wound has been significantly reduced. A curette was used to attempt removal of the remaining necrotic material. As there was still an adherent layer of fibrin covering the wound after debridement, topical collagenase was used daily for an additional week. This ointment is covered with a foam dressing and the patient is compliant with a medical equipment shoe.

The week 3 image (Figure 1-C) reflects significant progression of this wound toward closure. There is a significant reduction of fibrin and the wound base shows improved granulation tissue. Curetting this wound revealed a red, beefy, granular base. A sterile, freeze dried mix of 44% oxidized regenerated cellulose (ORC), 55% collagen, and 1% silver-ORC was applied to the base.[3] This combination product was chosen to maintain a physiologically moist microenvironment, leading to optimized wound healing. In addition, this product is chosen for its antimicrobial properties as diabetic foot wounds are at risk for polymicrobial colonization which can progress to frank infection.[6]

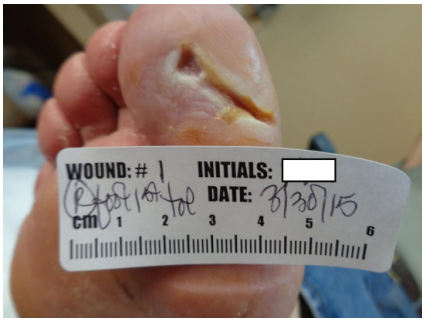

Figure 1-C

(Figure 1-D) This image shows a healing wound at week 6. The wound was able to overcome adversity, including off-loading in a secondary choice device due to patient demand. The matrix was able to provide a local microenvironment conducive to wound closure. At this discharge visit, the patient was extensively educated about the risk of diabetic foot ulcers, risk of amputation, and need for proper foot wear to avoid future ulcerations. Long term control of blood sugars has been emphasized. At discharge, the patient agrees to diabetic shoes as a preventative device.

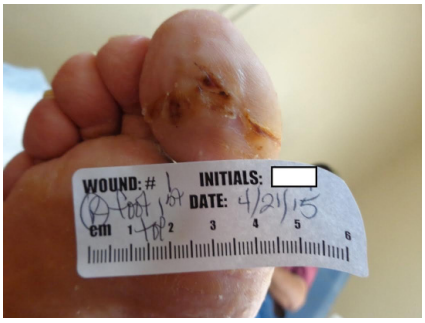

Figure 1-D

Case 2

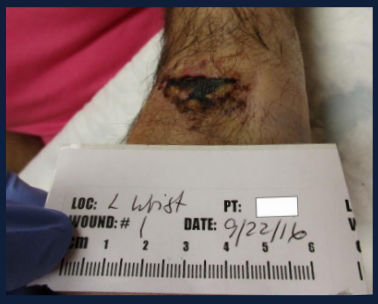

Figure 1-B

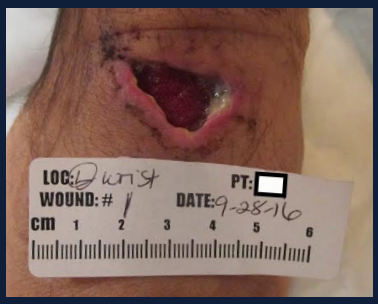

Figure 2-B

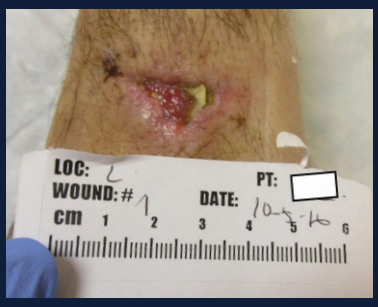

Figure 3-B

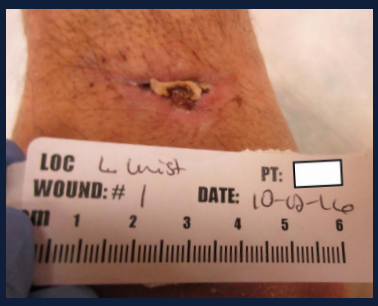

Figure 4-B

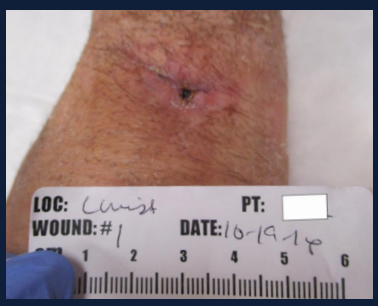

Figure 5-B

This 36 year old female with rheumatoid arthritis taking methotrexate presents with a surgical non-healing wound. Her other medical conditions include polycystic ovary syndrome (PCOS), hypothyroidism, and hypertension. The patient had left wrist surgery to remove a rheumatoid nodule, her sutures were removed at post-op day 10 and the wound was not closed. Sutures were replaced for 2 weeks and when they were removed, the wound continued to have a significant deficit in closure. The patient's first visit to the WCC was at 4 weeks post-op revealing a non-healed surgical wound on her left wrist (Figure 2-A).

On presentation, the wound measured 0.6cm x 2.4cm x 0.8cm. When a portion of eschar was removed with scissor and forceps, a significant tissue defect was noted. Post-debridement the wound measured 1.0 cm x 2.5 cm x 0.9 cm with tendon exposed.[4] After debridement, a wound culture was obtained from the base of the wound. Packing gauze was saturated with half strength hypochlorite solution (Dakins solution) and changed daily. It was chosen as a topical antiseptic while the culture was pending.

(Figure 2-B) At the one week interval, the wound base can be clearly seen. A selective debridement was performed in the office to remove dead tissue and slough, depicted in the photograph as yellow material. The base of the wound was prepared with a curette to remove biofilm and create a fresh surface. A freeze dried mix of 44% oxidized regenerated cellulose (ORC), 55% collagen, and 1% silver-ORC was applied to the base of the wound and wound was covered in a foam border dressing.[5] An additional matrix was added three times a week. Light compression was applied with a tubular compressive dressing. The culture from presentation was reviewed and showed no pathologic growth.

(Figure 3-B) At week 3 of care, this image shows a marked reduction in wound volume and improved quality of granulation tissue. The yellow necrotic tissue was removed with a curette and the wound was irrigated with saline. Additional ORC/collagen/silver was placed in the wound base and the wound was covered with a foam border dressing. Light compression was maintained.

(Figure 4-B) At week 4 of care, a good wound closure trajectory is seen in this photograph. ORC/collagen/silver can be seen stuck to the wound surface. The wound was cleaned and a curette was used to remove all non-viable tissue. The base of the wound was at skin level and red/pink granulation was seen. ORC/collagen/silver was reapplied and compression resumed. No signs of infection or inflammation were observed and no significant discharge from the wound was noted. Additionally, there was no limitation in the range-of-motion of the joint. The patient continues to take daily methotrexate for rheumatoid arthritis.

(Figure 5-B) This patient is discharged at week 5 of care from the WCC with a healed surgical wound on the left wrist, after the small fragment of material was removed.

While chronic wounds are difficult to heal, advanced wound care products and dressings give providers the tools to heal these chronic wounds. As these cases demonstrate, products used to treat chronic wounds can be changed to adapt to the situation.

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Patient data and photos courtesy of Katie Lincoln, DO, FAAFP; Texas, USA

NOTE: Specific indications, contraindications, warnings, precautions and safety information may exist for Systagenix products. Please consult with a healthcare professional and product instructions for use prior to application. Rx only.

PRA000310-R0-US, EN (03/17)

Wound Bed Preparation for Epidermal Grafting

NOTE: As with any case study, the results and outcomes should not be interpreted as a guarantee or warranty of similar results. Individual results may vary depending on the patient's circumstances and condition.

By: Mark R. Shaw, DO, FACEP, FAPWCA

Today, after more than 400 epidermal grafts performed at our wound center, this procedure has become the treatment of choice in our center for the final closure of wounds and ulcers when indicated. With a success rate above 90%* we have learned a few things that help to make that a reality. Essential to that outcome is wound bed preparation.

Wound bed preparation is defined as "the management of the wound to accelerate endogenous healing or to facilitate the effectiveness of other therapeutic measures." There are multiple factors that will ultimately affect the outcome of wound healing. Each one of these has to be appropriately addressed and managed in order to develop a wound bed that is adequately prepared for epidermal grafting. Not to ignore anyone of these elements; it has been our experience that there are 4 primary factors that have demonstrated the highest incidence for graft success. They are:

1. Recognition of adequate blood flow
2. Development of an appropriate granulation wound bed
3. Infection control
4. Chronic inflammation management

Primary attention to these along with other exogenous factors will go a long way to achieving the ultimate result of closure.

All patients need appropriate vascular screening to assure that adequate oxygen at the tissue level is present. Patients with lower extremity wounds should have initial ankle brachial index evaluations for this purpose regardless of the type of wound being managed. If results indicate an increased probability of vascular insufficiency arterial duplex ultrasonography is performed and the patient sent for vascular consultation. If indicated, revascularization is performed prior to grafting. In our experience arterial ulcerations have the highest incidence of graft failure. Recognition and treatment therefore becomes essential in this class of wound.

* Success rate defined as complete closure in 12 or less weeks

An adequate granulation bed is the foundation needed for epidermal graft placement. It must be void of eschar, thick fibrinous exudate such as slough and necrotic tissue. Sequential sharp surgical debridements are paramount in meeting this end. This is performed on a weekly basis until only granulation tissue is present. Keep in mind that gentle management of tissues during this process will maintain and enhance neovascularization, the literal lifeblood of the graft. If the patient is unable to tolerate this form of debridement or an existing coagulopathy prohibits it, enzymatic debridement is a viable alternative. Ideally, all tendon or bone should be covered. Improving the wound depth is required to obtain a reasonable cosmetic outcome. Control of wound depth in many cases can be best managed with NPWT. Ultimately developing a depth of 2 mm or less prior to graft application will limit the deformity created by healing over an untoward cavitation.

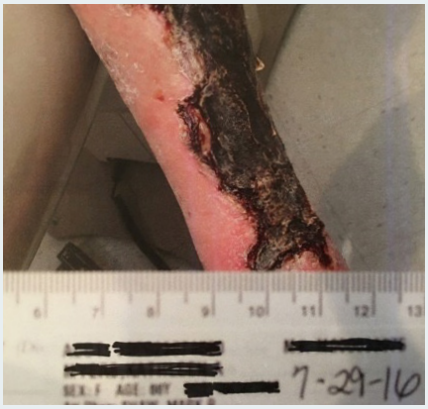
Infection control requires early recognition and diagnosis. Obtaining a proper wound culture is the first step in this process. A deep tissue source following debridement is critical in evaluating the wound for an acute infection. Targeted antibiotic therapy can then proceed. The eradication of an active infection will reduce the significant potential for graft failure. We recommend a minimum of 2 weeks of treatment prior to grafting. Obviously, this can be longer when IV therapy or when multi organism infection control is required.

When a wound has developed a state of persistent inflammation a graft failure is almost assured. When a potential wound for grafting demonstrates this stalled status (no change in status over 2-4 weeks), one such cause can be increased activity of the inflammatory cells. We now use collagen with ORC as a primary dressing for 2 weeks prior to grafting in all patients. We believe that this helps to promote the progression of the wound from the inflammatory to the proliferative stage of wound healing which is more receptive to receiving an epidermal graft.

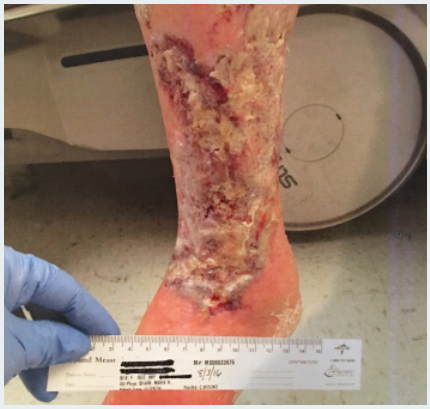
In order to help elucidate this process of wound bed preparation for epidermal grafting the following two clinical cases give a very good representation of affective outcomes when the factors that affect wound healing are controlled and appropriate interventions utilized.

CASE 1

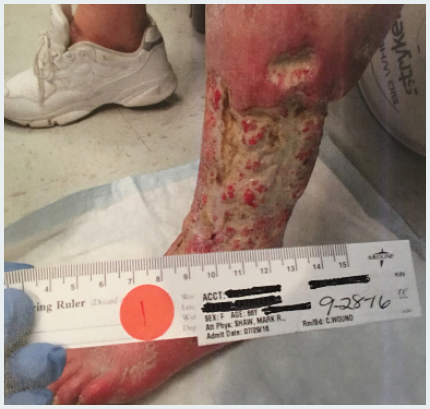
This case involves a 66 F with extensive tissue loss of the left lower extremity due to arterial ulcerations in the face of significant PVD. This patient has multiple co-morbid conditions including CAD, hypertension and a prior failed fem-pop bypass graft. She is still under our care having just received her first two staged epidermal grafts. Her treatment in the clinic has encompassed 6 months of therapy including surgical debridement, collagenase dressing, NWPT, IV and oral antibiotic therapy and endovascular revascularization.



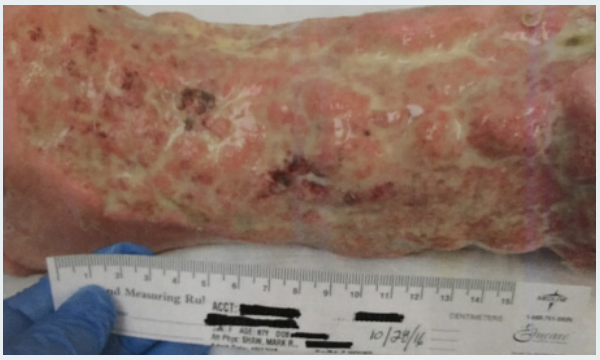
Initial Presentation



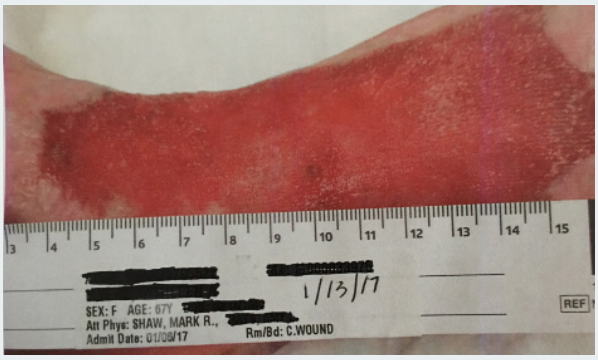
1 Week Post Debridement/
Collagenase Dressings



9 Weeks Post Debridement/
Hydrofiber With Silver Dressings



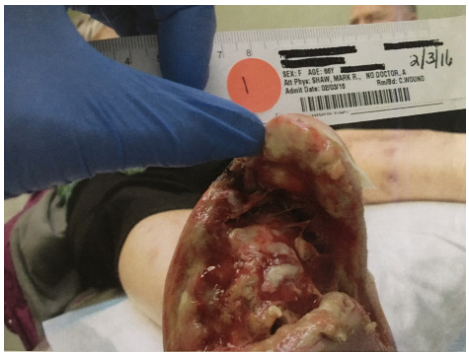
NPWT Initiated (13 Week Application)



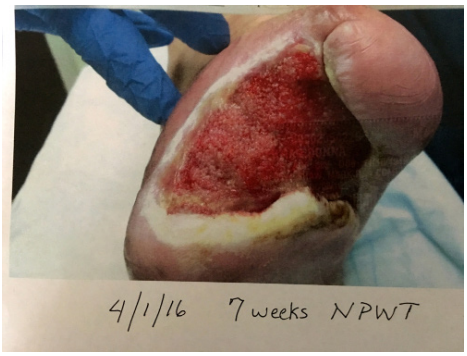
Epidermal Grafting Application Day

CASE 2

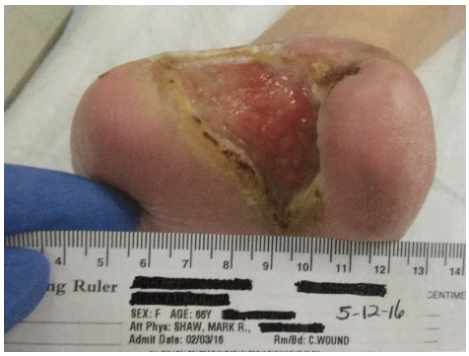
This case involves a 67 F who underwent a trans-metatarsal amputation of her left foot and additional surgical debridement for osteomyelitis and necrotizing fasciitis/gas gangrene. This patient has significant co-morbid conditions that consist of diabetic neuropathy, PVD, hypertension, hypothyroid disease and nicotine addiction. Her treatment in the clinic included surgical debridement, NWPT (13 weeks), HBO (60 treatments), IV and oral antibiotic therapy and epidermal grafting to closure.



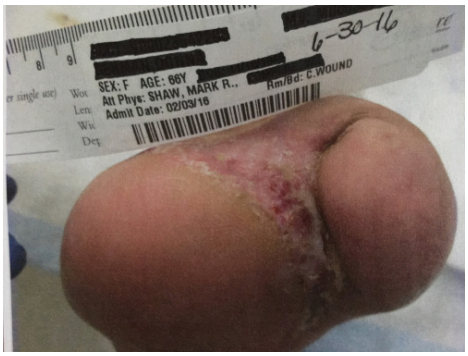
Initial Presentation



Post Debridement/7 Weeks Npwt



13 Weeks Npwt/Epidermal Graft Application



Wound Closure 7 Weeks Post Epidermal Graft

CONCLUSIONS

In our opinion, epidermal grafting, in its current state, is becoming more and more recognizable as the standard of practice for the final closure procedure of any wound or ulcer. Its ease of application, patient safety, patient convenience and excellent closure results will continue to help it grow in acceptance.

Success in accomplishing an effective outcome is directly linked to an adherence of definable and replicable practice techniques. Wound selection, management of both endogenous and exogenous factors and appropriate epidermal grafting application with ongoing wound management are all final elements needed to complete this task.

As demonstrated, effective wound bed preparation enables even the most complicated wounds to close. Focus on having adequate blood flow, wound bed granulation, infection control and management of chronic inflammation satisfy a valuable link in the chain of success for wound closure with epidermal grafting.

MARK R. SHAW, DO, FACEP, FAPWCA

Dr. Shaw graduated from Albright College in Reading, Pa. with a bachelor of science and a medical degree from Des Moines University in Des Moines, Iowa. He is board certified and a fellow in emergency medicine, He is also a fellow of the American Board of Wound Healing.

Today, Dr. Shaw is a medical director and full time clinician at the Mount Nittany Center for Wound Care in State College, Pa. with extensive experience in epidermal grafting having performed over 400 procedures. He currently functions as a consultant, national/regional speaker and advisory board member for the Acelity corporation.

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Use of the CELLUTOME™ Epidermal Harvesting System and the SNAP™ Therapy System as Part of a Wound Management Strategy for Stalled, Chronic Wounds

This clinical case is based upon the clinical experience of Animesh Bhatia. Results may not be typical and individual results may vary. Users should read and understand all Instructions for Use, including safety information, prior to application of the product. The images contained in this case study are courtesy of Animesh Bhatia.

By: Animesh Bhatia, DPM, CWS
Columbus Podiatry and Surgery, Inc, Columbus, OH

INTRODUCTION

Traditionally, split-thickness skin grafts have been used for wound closure. This grafting option requires surgery, creates a second wound at the donor site, and can have complications (eg, graft rejection, graft contraction, or infection).[1, 2] Various grafting techniques have evolved over time, leading to the development of epidermal grafting as a viable alternative to traditional skin grafting procedures in challenging wounds that require only the epidermal layer.[3-6] Epidermal grafting differs from traditional grafting methods as it can be performed in an office or outpatient setting without the use of a surgeon, operating room, or anesthesia. Following grafting, bolsters are typically used to secure grafts in place over the wound. Options for bolsters range from secondary dressings and self-adhesive wraps to negative pressure wound therapy (NPWT).

I report on my experience with epidermal grafts harvested using the CELLUTOME™ Epidermal Harvesting System, followed by use of the SNAP™ System as a bolster, as part of my wound management strategy for stalled, chronic wounds.

METHODS

Prior to epidermal grafting, all wounds underwent wound bed preparation techniques using sharp debridement, collagenase ointment, collagen dressings, or PROMOGRAN PRISMA™ Matrix (Systagenix, an ACELITY Company, Gargrave, UK). After the wound beds showed healthy granulation tissue, epidermal grafting was performed.

Donor sites (thigh) were prepared for epidermal graft harvesting using hair removal and an isopropyl alcohol wash. The CELLUTOME™ System vacuum head and harvester were securely attached to the donor site. Negative pressure (-400mmHg to -500mmHg) and warmth (37°C to 41°C) were applied for 35-45 minutes. After epidermal microdomes were formed, the vacuum head was removed, the microdomes were harvested onto an ADAPTIC TOUCH™ Non-Adhering Silicone Dressing (Systagenix, an ACELITY Company), and the dressing with grafts attached was immediately placed over the wound and left in place for 7 days.

The epidermal grafts were bolstered using the SNAP™ System, a lightweight, portable, mechanically powered negative pressure system that provides -125mmHg of negative pressure. The SNAP™ Advanced Dressing was placed over the ADAPTIC TOUCH™ dressing and connected to the SNAP™ 125mmHg Therapy Cartridge. The SNAP™ System was then secured to the patient's extremity using the SNAP™ Therapy Strap. Dressing changes were performed per the manufacturer's instructions. In some cases, wounds required further debridements and/or use of collagen dressings as well as additional epidermal graft applications. Wounds were monitored weekly during either SNAP™ Advanced Dressing changes or re-application of collagen dressings and were considered healed when fully re-epithelialized.

CASE STUDIES

The following cases highlight the use of epidermal grafts bolstered with SNAP™ System. The patients were 3 females and 1 male with an average age of 79 years (range: 69-85 years) who had a pressure ulcer (n=1), venous leg ulcer (n=1), or traumatic wound (n=2).

CASE 1

The patient was an 85-year-old female who presented to the clinic with a 30-day-old stage 3 pressure ulcer on the right heel measuring 1.2cm x 1.8cm x 0.1cm (Figure 1A). Medical history included peripheral vascular disease (PVD), hypertension, hyperthyroidism, neuropathy, chronic kidney disease, gastroesophageal reflux disease, osteoarthritis, osteoporosis, coronary artery disease, cataracts, cardiomyopathy, and ischemic polymyelgia rheumatica. After the patient was in an Unna boot for 1 week, the wound received epidermal grafts, followed by SNAP™ System therapy, which was used as a bolster. One week later, a PROMOGRAN PRISMA™ Matrix dressing was placed over the wound. Two weeks later, a small portion of the wound with necrotic tissue was debrided using a curette. Following debridement, the wound was covered with a PROMOGRAN PRISMA™ Matrix dressing and patient was placed in an Unna boot. One week later the PROMOGRAN PRISMA™ Matrix dressing was re-applied. The next week, at 2 months after presentation, the wound was fully closed with no complications (Figure 1B).

CASE 2

The patient was a 69 year-old male who presented with a venous leg ulcer of the left medial shin measuring 1.7cm x 1.0cm x 0.4cm, which had been present for 120 days (Figure 2A). Medical history included tobacco use, hypertension, hyperlipidemia, coronary artery disease, and Vitamin D deficiency. Upon presentation, the wound showed signs of hypergranulation. Silver nitrate and a hydrogel sheet were applied to the wound. After 14 days, the wound still showed signs of hypergranulation and treatment was changed to Promogran Prisma™ Matrix dressings and off-loading using an Unna boot for 4 weeks. However, the wound remained open, and extensive debridement was performed to prepare the wound for epidermal grafting. One week later, epidermal grafts were applied (Figure 2B), followed by use of SNAP™ System as a bolster. The wound showed signs of re-epithelialization 7 days post grafting (Figure 2C), and SNAP™ Therapy was continued for an additional 5 weeks. Although wound appearance improved, the wound size was increasing; therefore, SNAP™ System therapy was discontinued (per patient request), and the wound received collagen dressings for the next 2 weeks. The wound was then debrided in preparation for a second application of epidermal grafts, but dermatitis developed around the wound area. Therefore, treatment was changed to sodium chloride impregnated gauze dressings (Mesalt® Sodium Chloride Impregnated Gauze, Mölnlycke Health Care, Gothenburg, Sweden) for the wound and a steroid cream for the dermatitis. By the 3-month follow-up, the dermatitis had resolved, and the wound was fully closed without complications (Figure 2D).

CASE 3

The patient was an 81 year-old female who presented with a traumatic wound of the right lower leg measuring 5.5cm x 4.7cm. Medical history included coronary heart disease, hypertension, atrial fibrillation, asthma, congestive heart failure, valve disease, osteoarthritis, shortness of breath, and swelling of feet and ankles. The patient had been treated at another facility with oral antibiotics and SILVERCEL™ Antimicrobial Alginate Dressing (Systagenix, an ACELITY Company). Upon presentation to my clinic, treatment was changed to collagenase ointment with wet/dry dressings daily. After 6 weeks, the wound underwent sharp debridement using a curette. One week later, epidermal grafts were applied to the wound (Figure 3A and 3B), and SNAP™ System therapy was used as a bolster. Wound re-epithelization was observed 3 weeks post grafting (Figure 3C). Six weeks later, the wound received a second application of epidermal grafts (Figure 3D) bolstered with SNAP™ System therapy and the SNAP™ Advanced Dressing. The SNAP™ Therapy system and dressing were changed once a week. The wound was fully closed without complications 5 weeks following the second epidermal graft application (Figure 3E).

DISCUSSION

The cases presented here were complex with each patient having multiple comorbidities that contributed to stalled, chronic wound healing. In these patients, debridement, collagenase ointments, and collagen dressings were first used to prepare the wound bed for epidermal graft application, which was followed by the use of SNAP™ System as a bolster. Additional graft applications as well as debridement and collagen dressings were sometimes necessary, and together, these different advanced wound therapies led to eventual closure of all wounds.

With the commercially available CELLUTOME™ System, the harvesting procedure is minimally invasive and can be performed in an office setting without any anesthesia. Several case series using this system to harvest epidermal grafts have been published with positive wound healing outcomes for a majority of patients with complex wounds.[3-8] In my recently published case series using epidermal grafts in patients with multiple comorbidities and chronic wounds, 82.4% (28/34) of the wounds showed complete healing.[9]

NPWT was used as a bolster over epidermal grafts in the 3 patients. NPWT is indicated for a variety of wounds and has been used as a bolster for skin grafts with positive results.[10, 11] The availability of a lightweight, mechanical NPWT system, such as SNAP™ System, has provided healthcare professionals the opportunity to use NPWT when the traditional powered NPWT systems may not be appropriate or available. SNAP™ System was used as the bolster in these patients, and all wounds remained closed at follow up.

In these patients, the combination of wound bed preparation, epidermal grafting, and SNAP™ System proved to be successful wound management tools. More studies are needed to determine the clinical and economic feasibility of this treatment regimen; however, these early experiences are promising.

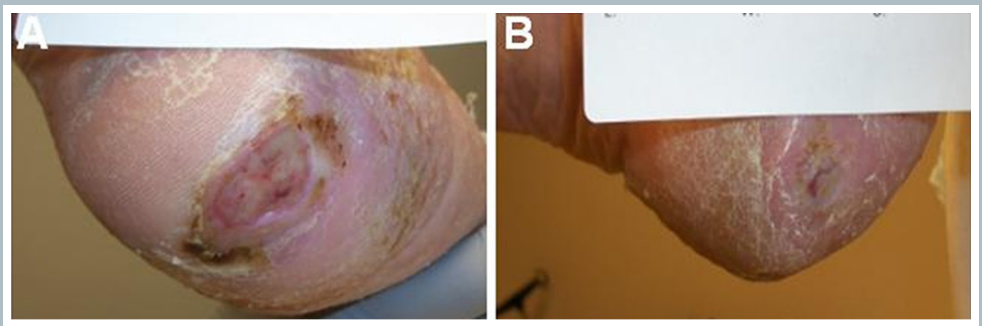

Figure 1. Stage 3 pressure ulcer on right heel. Wound at presentation (A) and wound fully closed at 2-month follow-up visit (B).

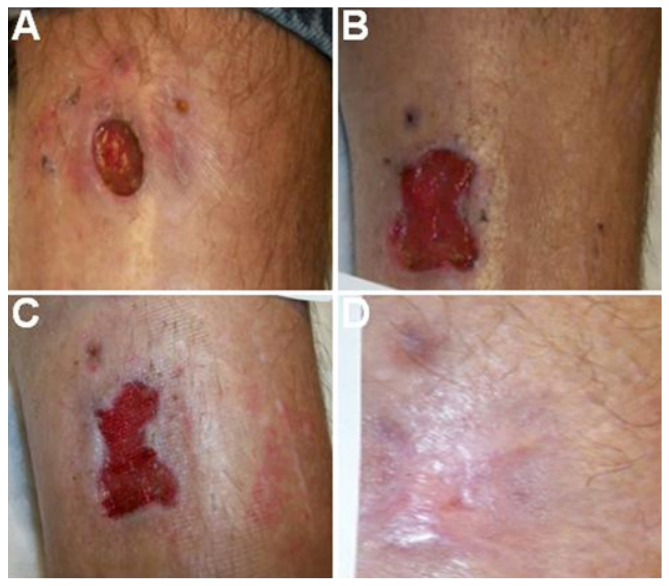

Figure 2. Venous leg ulcer of the left medial shin. Wound at presentation (A), wound on day of epidermal grafting (B), wound at 7 days post grafting (C), and wound fully closed at 3-month follow up (D).

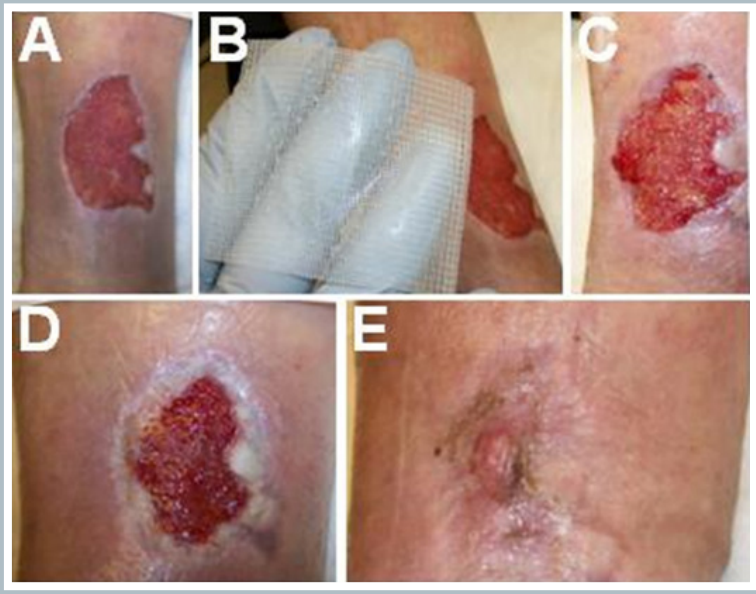

Figure 3. Traumatic wound of the right lower leg. Wound prior to epidermal graft application (A), application of epidermal grafts (D), and wound fully closed (E).

NOTE: Specific indications, contraindications, warnings, precautions and safety information may exist for all KCI and Systagenix products and therapies. Please consult a clinician and product instructions for use prior to application. Rx only.

ANIMESH (ANDY) BHATIA, DPM, CWS

Dr. Bhatia serves as Vice President on the board of trustees of the Ohio Foot and Ankle Medical Association. He is also the Medicare Carrier Advisory Rep for Ohio. He serves on the BOT of the American Academy of Podiatric Practice Management (AAPPM), and was a Governor appointed physician member of the Ohio State Board of Orthotics, Prosthetics and Pedorthics. He is a board certified wound specialist, and a fellow of the ASPS and AAPPM. He also serves as Chairman of the APMA Coding Committee. He has lectured nationally and internationally on the topics of wound care, practice management and coding. Dr. Bhatia has been in private practice for eighteen years in Columbus, Ohio, and serves as Medical Director of a multi-specialty private practice group focused on podiatry and wound care. He is also the Assistant Medical Director of the Wound Clinic at Fairfield Medical Center in Lancaster, Ohio.

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Approach to Using Silver Dressings for Chronic Wound Care

By: Jayesh B. Shah, MD, UHM(ABPM), CWSP, FAPWCA, FCCWS, FUHM, FACP, FACHM

INTRODUCTION

Management of infection and biofilm are necessary for wound bed preparation.[1] Silver has a broad spectrum of antimicrobial activity including activity against MRSA and VRE.[2] Although use of silver has been described in history since the ancient times,[3] it really became more popular after 1970's when silver was used in treatment of burns using either 0.5% silver nitrate solution or topical cream like silver sulfadiazine.[4] Recently, there is a newer class of silver dressings available in a variety of forms like transparent dressings, gauze, island dressings, foams, and absorptive filler. Silver dressings are also available with various combinations like collagen, hydrofiber, alginates, foam, honey and hydrogels.[4] These dressings are designed to provide the antimicrobial activity of topical silver in a more convenient application.[4] These silver dressings differ considerably in the amount of silver ions and their physical and chemical properties.[2, 4]

MECHANISM OF ACTION

Silver ions bind to the bacterial wall at multiple sites, causing membrane damage and cytoplasm leakage, which makes some silver dressings bactericidal.[4] Silver ions also bind to the cell proteins, causing cell death and destroying cell DNA.[4]

NEWER SILVER DRESSINGS AND EVIDENCE

Newer silver dressings have silver in form of complexes of silver salts or nanocrystalline silver metal. It may be found as element Ag or ion Ag^{+}.[4, 5] Sibald et al studied the use of nanocrystalline silver primary dressing on chronic venous stasis ulcer patients who failed multilayer compression therapy. Study results found that silver dressings reduces bacterial counts, increases lymphocyte count and improves healing rates[4, 6] in a statistically significant way. Castellano et al. compared eight silver containing dressings and concluded that dressings with higher concentration of silver ions may be more appropriate for wounds that contain 105 organisms.[4, 7] Similarly, Gago et al compared healing of venous ulcers with silver dressing and concluded that patients who had higher concentration of silver in dressing showed reduced healing time and quicker resolution of infection.[4, 8] While Parson's et al found that there was no correlation between antibacterial effect and silver content of dressings.[2]

CASE STUDY

In a recent case, a 60 year old female who works as a cashier and stands on her feet all the time presented to my office. The patient had a history of hypertension, a history of venous stasis and a history of ulcers in the past which were treated with compression therapy. The patient was not wearing stockings and she developed ulcer on left ankle which started from rubbing of shoes. She initially presented with a blister which progressively got worse. So she was sent to the wound care center for a complete comprehensive evaluation. Upon deeper examination, the patient had normal arterial doppler with triphasic blood flow.

On admission we started the patient on calcium alginate with silver and conducted debridement. Her ABI Right measured 1.02 with the left being non compressible. So, we obtained a wound culture, started the patient on Cefdinir 300mg 1 tab PO BID X10 days for one week. Ultimately, the wound got worse so we then started the patient on Dakin's solution 0.25% daily for 1 week (see Figure 1).

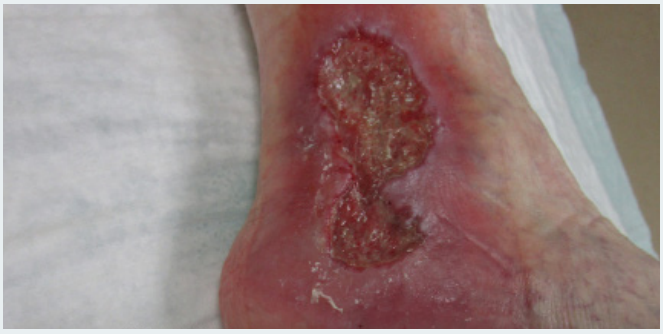

Figure 1: A 6.6 cm X 2.7 cm X 0.4 cm wound

At the two week mark, we started the patient on silver powder mixed with iodine-based gel with a compression dressing 3 times per week.

At the one month mark, the wound was still worse with a wound size of 6.8cm x 3 cm x 0.3cm with 100% soft necrotic tissue and purulent drainage. The patient was started on 0.25% Dakin's solution with daily dressing changes. In addition the patient was admitted to hospital where a culture was run for multidrug resistant pseudomonas and the patient was also started on IV Merrem® for four weeks (see Figure 3).

At the two month mark, the patient was discharged from a rehab facility after 4 weeks of IV Merrem®. The wound size was now smaller at 5cm x 1.5cm

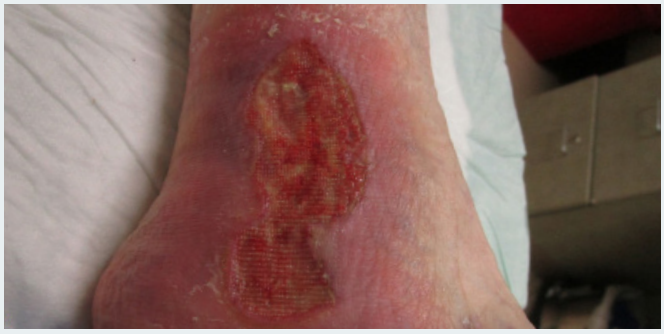

Figure 2: Wound has increased in size to 6.9 cm X 2.6cm X 0.2cm with continued purulent drainage.

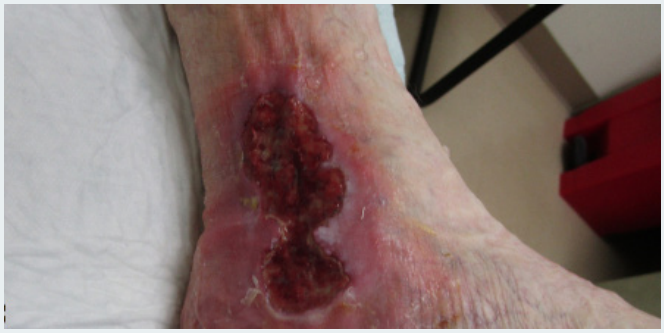

Figure 3: Wound at the one month mark with 100% soft necrotic tissue

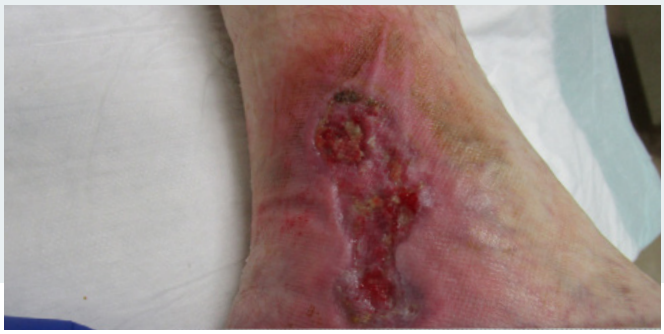

Figure 4: Wound at 8 week mark

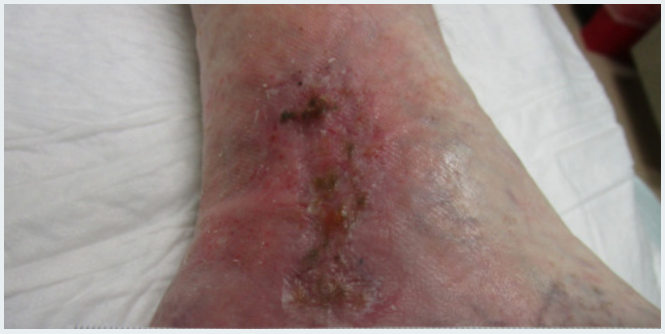

Figure 5: Wound at ~12 week mark

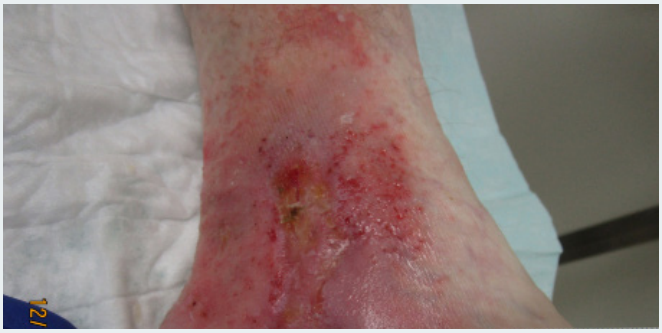

Figure 6: Wound at ~ 3.5 month mark

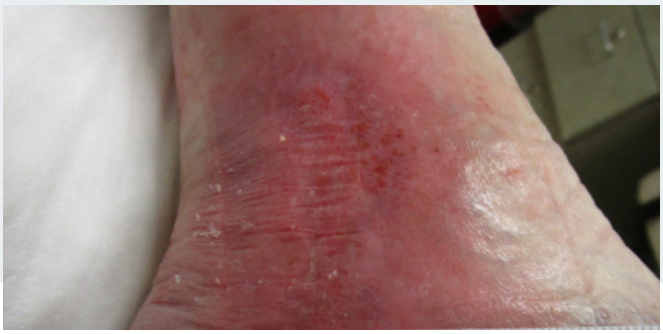

Figure 7: Wound at discharge (~4.5 month mark)

x 0.2cm with 30% of the wound area having commenced epithelization. In addition, there was 50% granulation tissue and minimal slough. We then started the patient on Collagen Silver Dressing and a compression bandage (see Figure 4).

At the approximate three month mark since presentation, we continued the patient on Collagen-Silver and a compression bandage. The wound continued to epithelialize (~ 80%) and the wound size had now diminished to 1cm x 0.3cm x 0.1cm (see Figure 5)

At the three and a half month mark, the wound has achieved significant epithelialization. The patient was started on amylactin lotion and continued use of a compression bandage. The wound size is now measured at 0.8cm X 0.3cm X 0.1cm with 80 -90% epithealialization (see Figure 6). About a week after this visit, the patient's wound had epithelialized 100% and now measured 0.1cm x 0.1cm x 0.1cm and was managed with amylactin lotion and a compression bandage.

After a few more weeks of treatment and follow up, the patient was discharged from our care with a class II compression stocking (see Figure 7).

CONCLUSION

Choice of wound dressing should be based on the wound type and clinical parameters such as exudate handling effects and presence of biofilm or critical colonization.[4] Dry wounds should receive moist dressings and wet wounds should receive absorbent dressings. Based on clinical studies, general recommendation can be made that dressings with higher silver can be used in refractory wound with recurrent biofilm and in clinically infected wounds.[4, 7] Clinically, 10 minute 0.25% acetic acid or 0.25% Dakin's solution soaks are recommended to treat biofilms prior to the application of silver based dressings. Dressing with lower concentration of silver or combination of collagen and silver could be considered in wounds that are stalling or suspected of being colonized or critically colonized.[4, 7, 8] Studies are not able to recommend specific length of time for use of silver dressings but it is prudent to not use silver dressings for more than 9 -12 weeks. Long term use of silver may be associated with systemic toxicity though Sibald et al found that there was only a slight increase in blood silver concentration indicating that nanocrystalline silver dressings are not systemically toxic[6] while silver sulfadiazine does contain fast release silver which was easily absorbed by tissues and was associated with systemic toxicity.[4, 9]

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Understanding Topical Wound Solutions

By: Rose Raizman, RN-EC, MSc, MNur, CETN(C), SRH

Currently the most accepted paradigm of successful wound care is "DIME" or "TIME"[1] where clinicians are called upon to first address tissue viability and debride devitalized tissues from the wound (D or T in the acronym), eliminate infection (I), address moisture (M) balance in the wound, and promote epithelial growth by ensuring that edges (E) of the wound are contracting. Striving for cost efficiency and optimal patient outcomes, clinicians often try to find a therapy that would address all domains simultaneously. Unfortunately, only one modality cannot, often times, address all these needs, thus a combination of therapies is required. For example, the wound bed would be cleansed by an appropriate solution, followed by debridement, and a suitable wound cover dressing, or combination of such dressings chosen.

To help clinicians in this important and challenging task, the wound care industry is developing dressings and technologies. However, the fast pace of these innovations has become a challenge for wound care clinicians in terms of staying current and being aware of interactions between therapies or dressings that could potentially deactivate one another. Antiseptic preparations are those that non-selectively reduce flora on the surface of the skin or wound bed. Some have been found to be cytotoxic to fibroblast keratinocytes and leukocytes in vitro, but in vivo no such correlation has been confirmed and they have been rendered to be safe, yet controversies about their usage still exist.[2, 3] In depth chemical analysis or effectiveness of these therapies and cleansers is beyond the scope of this paper.

This paper reviews the role of cleansing solutions in wound care and some possible interactions leading towards inactivation of key components of advanced wound dressings. The most common choice of cleansers include: sodium chloride, water, iodine, chlorhexidine, hypochlorous acid-based cleansers and commercially available cleansers that contain surfactants. The active ingredients within dressing components are varied and numerous but common elements are silver, iodine, polyhexamethylene biguanide (PHMB), gentian violet/methylene blue, and pDADMAC. The predominant concern from a clinical perspective is that the components within the cleanser and the active wound dressing ingredient could deactivate one another's clinical benefits and detract from the healing process.

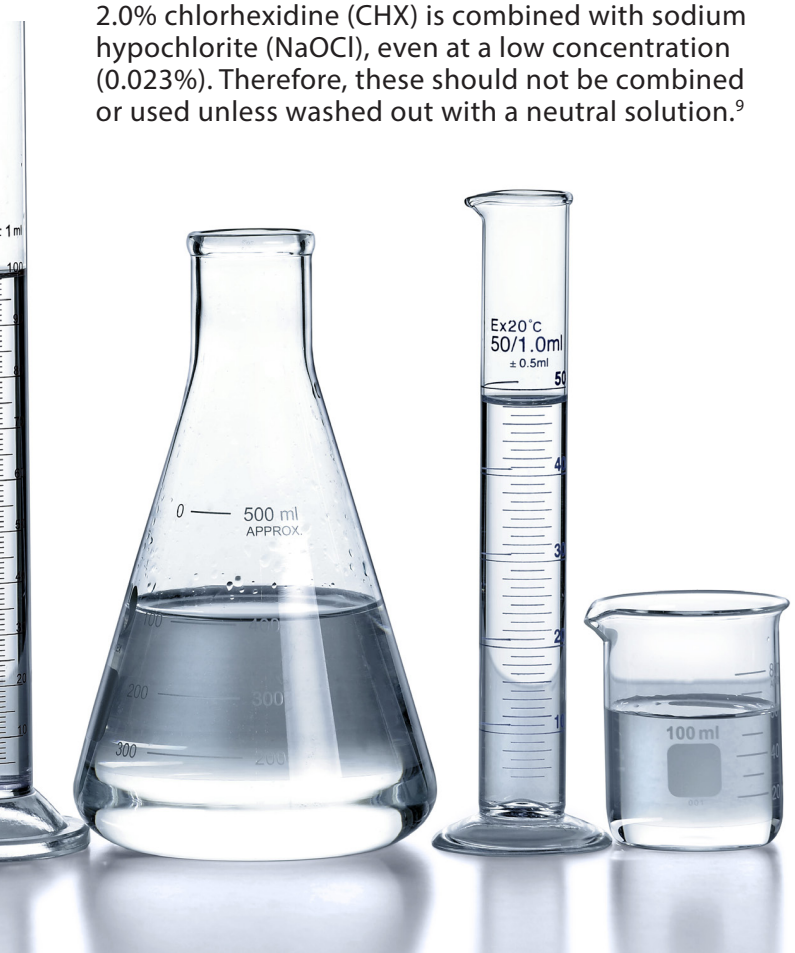
Let's review the possible interactions among the antiseptic solutions and wound dressings with active ingredients:

BIGUANIDES

CHLORHEXIDINE GLUCONATE was formulated from a series of bisbiguanides processing which has a marked bacteriocidal action against a wide range of microorganisms.[4] Chlorhexidine gluconate is believed to have anti-inflammatory effects on the tissue. Lower concentrations (e.g. 0.02%) are recommended for use in wound irrigation.[5] Its possible mode of action is combining with the cell surface, disorganizing permeability barriers and coagulating the cytoplasmic content of the cells.[6]

Activity of chlorhexidine is pH dependent and is reduced in the presence of organic matter, blood or pus.[7] Chlorhexidine is believed to be fairly compatible with most available dressings, although caution should be employed when combining silver nitrate and chlorhexidine preparations. Even a 0.5% of silver nitrate when combined with 0.02% chlorhexidine gluconate solution leads to precipitation of chlorhexidine nitrate.[8]

Another possible interaction leading to perchloric acid (PCA) precipitate is with hypochlorous acid. An immediate reaction can be observed when 2.0% chlorhexidine (CHX) is combined with sodium hypochlorite (NaOCl), even at a low concentration (0.023%). Therefore, these should not be combined or used unless washed out with a neutral solution.[9]



POLYHEXAMETHYLENE BIGUANIDE (PHMB) is a small cationic polymer that is used as an antiseptic and in a variety of advanced wound care products as it appears to be highly histocompatible and non-cytotoxic.[4] It transfers from the surface of the bacteria to the cytoplasm, especially when pH is between 5-6, and leads to bacterial death due to release of lipopolysaccharides and potassium ion efflux.[10]

PHMB (as in Kerlix™ AMD, Covidien LP, Mansfield, MA) deactivates sodium hypochlorite and enzymatic debriders.[1] In contrast, pDADMAC (e.g. Bioguard® Dressings, Quick-Med Technologies, Inc., Gainsville, FL) are large cationic polymers that are safe with enzymatic debriders (e.g. Santyl® Oinment, Smith & Nephew, Inc., Memphis, TN).[11]

POVIDONE IODINE (PVP-I) is a chemical complex composed of molecular iodine and polyvinylpyrrolidone (povidone), commonly used for wound irrigation. Povidone iodine has been Food and Drug Administration (FDA) approved for the short-term treatment of superficial and acute wounds.[12] Iodine impregnated dressings as well as cadexomer iodine are commonly used topical treatments. Note that iodine is contraindicated in patients with thyroid disease after treatment with radioiodine, patients with Duhring disease, iodine allergy sensitivity, perturbation of neonatal thyroid hormone, and when extensive body areas are exposed due to risks of systemic toxicity.[13] Some authors suggest that slow replacement of neutral lipids in elderly can increase exposure to iodine from povidone iodine in the elderly.[14] Iodine reacts with hydrogen peroxide, silver, taurolidine (used for catheter related bloodstream infections), mercury, and proteins, rendering them ineffective.[6, 15]

HYPOCHLOROUS ACID (HOCL) is highly active against all bacterial, viral, and fungal human pathogens and can kill spore-forming and non-spore bacteria in a relatively short period of time. Its intended use is for cleansing (or deep cleaning), irrigating, and debriding acute and chronic wounds.[2] Hypochlorous acid leads to cell death by the oxidation of sulfhydryl enzymes and amino acids, ring chlorination of amino acids, loss of intracellular contents, decreased uptake of nutrients, and inhibition of protein synthesis, decreased oxygen uptake, oxidation of respiratory components, decreased adenosine triphosphate production, breaks in DNA, and depressed DNA synthesis.[16]

The original Dakin's solution (pH 9 to 10) is known to be cytotoxic to healthy cells and granulating tissue, and as such its use is not recommended for periods longer than 7-10 days[16] and limited to use in wounds requiring extensive debridement. However, recently HOCL solution has regained popularity and new manufacturing processes have allowed for a change in pH ranges (3.5-5.5 etc), oxidation-reduction potential (ORP), HOCL concentrations and thus the overall safety profile of these cleansers has improved.[17]

SILVER is widely used as an active component in a vast variety of dressings (e.g. cleansers, alginates, gels/ointments, foams, collagen, and ORC/collagen preparations). Silver ions are believed to act by binding to the DNA helix and blocking transcription.[4] While some companies explicitly recommend the use of Sterile Water to cleanse the wound to prevent interaction between Sodium and Argentum Ions, others leave it up to the clinicians to choose.

It is important to note that bodily fluids have a physiologic concentration of sodium and thus some neutralization will happen regardless of an irrigation solution. However, unbound iodine molecules can neutralize Silver Ions more readily and therefore clinicians should refrain from combining Silver and Iodine based products. If Iodine irrigation is still required, rinsing the wound afterwards with a neutral solution such as water or saline is recommended.

Both Silver and Iodine inactivate growth factor therapies and are also advised not to be used with enzymatic debridement.[1, 11]

GENTIAN VIOLET AND METHYLENE BLUE (GV/MB) antibacterial dressings consisting of organic antimicrobial dyes have been used for many years in the clinical setting with minimal toxicity to humans. Both GV and MB dyes are basic with a positive charge, thus showing differential activity toward gram-negative versus gram-positive bacteria.[18] Gentian Violet and Methylene Blue are contraindicated in 3rd degree burns.[19] No specific stated interactions of these dressings with other agents available on the market are reported in the literature or noted by manufacturers in their product monographs or package inserts.

Let's look at a few examples:

CASE 1

A 74 year-old male presented with an infected diabetic wound ulcer on the left foot. During baseline assessment, the wound measured 6 cm x 7 cm x 3 cm and was 100% covered with slough. A probable bone involvement was suspected. The standard care for a person with diabetes who has developed a wound should involve a multidisciplinary team approach (infectious disease, surgeon, chiropodist or podiatrist, dietitian, endocrinologist, etc.) along with initial sharp debridement and initiation of systemic antibiotics. The wound care regimen prescribed consisted of using negative pressure (V.A.C. VERAFLO™ Dressing Kit - Medium 2 foam dressings [17 x 15 x 1.8cm]) with one quarter Dakins solution (Negative Pressure Wound Therapy with instillation and dwell time (NPWTi-d) (V.A.C. VERAFLO™ Therapy, KCI, San Antonio, TX). At this stage reducing bioburden while removing necrotic tissue was of great importance. Since the wound was completely covered by slough, there were no concerns of cytotoxicity when an irrigation solution was chosen. It should also be noted that systemic absorption was highly improbable at that point. Quarter strength Dakins solution was chosen in this particular case. It is very important to remember that hypochlorous acid, chlorhexidine and PHMB should not be used together in any combination to prevent deactivation of active components.

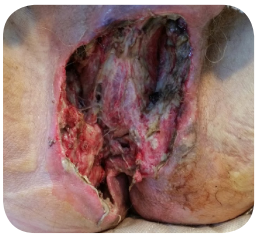
The following week, the wound had 80% of granulation tissue growth with exposed bone—which was assumed to be osteomyelitis but was not confirmed by x-ray--- and moderate exudate. A change in the wound care regimen needed to be established to address bioburden, possible cytotoxicity, and moisture control. Peer-reviewed literature using an in vivo model supports the approach to cleanse the wound with povidone iodine or chlorhexidine. However, caution should be exercised that silver dressings should not be used concomitantly with an iodine-based cleanser due to possible interactions. Therefore, if a decision is made to apply a silver-based foam or ORC Collagen with silver, the wound care choice should be a chlorhexidine-based cleanser.

At follow up appointments, the wound progressed towards proliferation and epithelialization and thus the subsequent cleansers of choice were saline or tap water.[20] Although both solutions are non-cytotoxic and can be used as wound irrigation solutions, one should always keep in mind that saline preparation should not be left open for longer than 24 hours to prevent contamination.

CASE 2

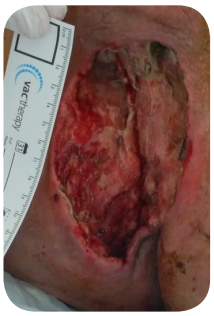
A 69-year-old male presented with perineal Fournier's Gangrene and an abdominal abscess. The patient was started on intravenous antibiotics.

DAY 0



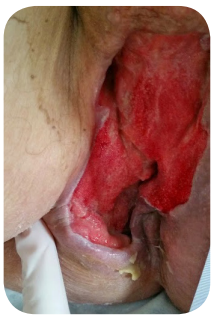
The wound had 100% necrotic tissue so the goals of therapy were wound bed preparation by removal of necrotic tissue and infectious materials and granulation tissue formation. The cleanser of choice at this stage should address the opportunity to apply a continuous cleansing solution for ongoing irrigation. Current research shows no clear advantage of any particular irrigation solution.[21, 22] The choice for this wound was Negative Pressure Wound Therapy with instillation and dwell time (NPWTi-d) (V.A.C. VERAFLO™ Therapy, KCI, San Antonio, TX) with Hypochlorous Acid 1:20. NPWTi-d settings included a 10 minute dwell time and 4 hours of continuous negative pressure at -125 mmHg. Dressings were changed 3 times per week.

DAY 2



The wound presented with mixed necrotic and granulation tissue. Conservative sharp wound debridement involving the removal of necrotic and devitalized tissue at the bedside with scissors was commenced. The goals remained the same (removal of necrotic tissue and infectious materials as well as granulation tissue formation). Dressing changes were conducted as per protocol and were changed 3 times per week.

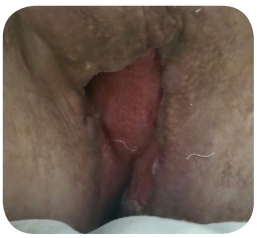
DAY 13



The wound reached 100% granulation with moderate discharge and a wound depth of 1 cm.

The main goals at this point were to address an edge effect and maintain moisture control. Therefore NPWTi-d was discontinued; however, we continued the use of NPWT without irrigation to promote further granulation tissue, ensure exudate was managed (to promote moisture control and create the optimal healing environment). Supportive cleanser to manage bioburden was Povidone –iodine Solution.

DAY 22



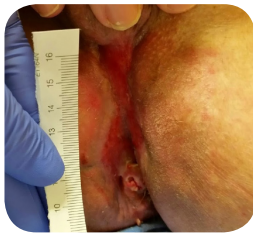
The wound bed reached 100% granulation with minimal discharge. The edges were level with the wound bed and NPWT was discontinued.

As the edge effect and moisture control were the final goals, one more challenge still existed insofar as the wound had a close proximity to the anus. Antimicrobial control was also a consideration to enhance optimal healing at this final stage.

A supportive cleanser of choice should be non-cytotoxic and compatible with the dressing that has been selected. In order to maintain the edge effect, the dressing of choice can be a collagen or silver based collagen.

The cleanser that was chosen in this particular case was chlorhexidine 2%. ORC Collagen with silver was applied as a topical treatment, and a foam dressing was used to maintain moisture balance and changed every 2-3 days. It should be noted that Povidone Iodine was discontinued due to possible interactions with silver dressings.

DAY 45



The wound was close to being fully epithelialized. At this stage, moisture balance and edge effect were most important. The supportive cleanser was non-toxic (such as NaCl 0.9% or tap water) and an absorptive dressing such as self-adherent foam for less frequent dressing changes (2-3 days) or a gauze dressing if dressing changes are frequent (e.g. after each bowel movement or a trip to the washroom) may be used. In this particular case foam was chosen as per patient preference.

CONCLUSION

Wound care practitioners should engage in comprehensive approaches that involve both patients and multidisciplinary team members. Both 'art and science' are required while deciding on optimal care plans. Clinicians should remember that the composition of dressings and cleansers can contain either ionic or salt components of the metals and therefore there are potential chemical interactions that can deactivate active components.

It should be remembered that metal ions can interact with each other or other substrates. For example, refrain from combining silver based dressings with iodine based dressings. In addition, manufacturer's instructions should be consulted for the choice of preferred cleansing agents.

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Rose Raizman's nursing career spans more than 20 years in a variety of nursing positions. Currently she is applying her passion for helping people to obtain and maintain optimal level of health as a wound care NP at community hospital in Ontario, Canada. Rose received Master of Medicine from Hebrew University in 2001 in Israel and Master of Nursing combined with Primary Health Nurse Practitionner in 2015 in Canada. She holds CNA certification in Enterostomal Therapy and her primary areas of expertise are ostomy, wound and continence care in all care settings. She has presented and published her work nationally and internationally.

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