



The Practicing Physicians' Guide To Pressure Ulcers in 2008

Rachel Roach, MSN, ANP, GNP, WCC, and Clarisse Dexter, MSN, FNP, GNP, WCC

MS is a 79-year-old woman admitted to the nursing home following a lengthy hospitalization for pneumonia and COPD. Her hospital course was complicated by *Clostridium difficile colitis* and respiratory failure requiring mechanical ventilation. During her stay, she developed a sacral pressure ulcer. While performing your admission examination, you note that the pressure ulcer is 3 cm x 4 cm wide and 1.2 cm deep, with thick, adherent, yellow slough covering the entire wound bed. The walls of the wound are gray, fibrous tissue; there is undermining from 10 to 2 o'clock. There is minimal wound exudate. The peri-wound tissue is macerated.

Pressure ulcers are, in most cases, preventable injuries.¹ This article will guide the clinician in formulating a reasonable, evidence-based plan to heal pressure ulcers.

ASSESSMENT

A comprehensive assessment of the overall health status of the patient and the characteristics of the ulcer are essential, and form the basis for treatment. Aspects to be assessed and documented include:

- Location and size of the ulcer, documented by anatomical part, and measured as length x width x depth in centimeters.
- Describe the wound from the bottom up:
 - A description of the tissue or necrotic debris in the wound bed, noted as slough or eschar, and reporting the percentage of debris versus granulation tissue.
 - Record the characteristics of any exudate present, documenting odor, color and consistency.
 - Note the presence of tunneling, tracts and undermining, using a clock and head-to-toe direction for documentation reference points.
 - Note and remark on the condition of the surrounding skin.

CLASSIFICATION OF PRESSURE ULCERS

The National Pressure Ulcer Advisory Panel (NPUAP) has developed a specific, standardized rating system to "stage" pressure ulcers.²

Stage I is an observable pressure-related alteration of intact skin, as compared with the adjacent skin or opposite area on the body. The ulcer appears as a defined area of persistent redness in lightly pigmented skin, and red, blue or pur-

plish tones in darker skin. This area may be painful, pruritic, and warmer than the surrounding tissue.

Stage II is a partial thickness skin loss involving the epidermis and/or the epidermis. This ulcer appears as an abrasion, blister or a shallow crater.

Stage III is a full thickness loss of the subcutaneous tissue extending to, but not through the underlying fascia. This wound is a deep crater.

Stage IV is also a full-thickness tissue loss, with destruction extending into the muscle, supporting structures or to the bone.

The presence of eschar covering a wound prevents staging. These wounds are documented as "unstageable" until the eschar is removed and the wound bed can be inspected.

Deep tissue injury is the most recent classification of pressure ulcer added by the NPUAP. This type of ulcer often has the appearance of a deep bruise under intact skin and may rapidly progress to a full thickness ulcer.³

TREATMENT

The selection of a treatment for a wound should be based on the needs of the patient, the wound, the caregiver and the clinical setting. The dressing should provide moisture balance in the wound bed, manage exudate, prevent infection, not cause pain to the patient and protect the periphery of the wound from damage. The goals of care of the patient and the cost to the payor should also be considered.

The clinician should become familiar with the different categories of dressings and their composition. Knowledge of the facility or institution's protocol and inventory, along with communication with the wound care team will assist in formulating a comprehensive treatment plan.

Table 1 will assist in selection of an appropriate treatment modality.

Table 1:

Wound Care Matrix	Intact Skin	Stage I	Stage II	Stage III	Stage IV
Exudate	None	None	Light	Dry to Moderate	Heavy
Product Category	Skin Care	Barrier Creams or Transparent Film	Hydrocolloid Dressings	Hydrogels to add moisture or Alginates to absorb	Foams Specialty Absorbent Dressings, or Negative Pressure Wound Therapy

Table 2. Assessment of and Intention to Treat Infection in Chronic Wounds⁶

Bacterial Burden	Contaminated	Colonized	Critically Colonized	Local Infection	Systemic Infection
Wound Clinical Symptoms and Signs	Wound progressing, Host stable	+/- early signs of local infection	No/subtle s/s of infection	Local s/s of infection	Constitutional ss/ of infection
Bacterial culture and sensitivity	No	+/- C&S wound	C&S wound	C&S wound	C&S wound and blood culture
Topical antibiotic	No	+/-	Yes	Yes	Yes
Systemic antibiotic	No	No	+/-	+/-	Yes

The third consideration is providing an environment to the wound bed that provides moisture to promote healing and controls exudate. Lastly, optimize the repair process by providing the patient with nutritional support, vitamin supplementation, adequate hydration and by avoiding exposure to cold (vasoconstriction reduces blood flow to the wound). It is also important to provide an appropriate support surface, such as a low

WOUND BED PREPARATION

Wound bed preparation provides a conceptual approach to treatment decisions. Wound bed preparation is defined as “the global management of the wound to accelerate endogenous healing or to facilitate the effectiveness of other therapeutic measures.”⁴ The first step is removal of dead tissue and contaminants in a timely manner.⁵ This cleaning of the wound bed can be accomplished by bedside sharp debridement, autolysis (synthetic dressings cover a wound and allow devitalized tissue to self-digest from enzymes normally present in wound fluid), mechanical (wet to dry dressings), chemical removal (collagenase enzymatic debridement), and whirlpool irrigation of the wound. Wounds should be cleansed with low toxicity solutions, preferably normal saline. Topical antiseptics (such as Dakin’s solution, Domeboro’s solution, Betadine and Acetic Acid solutions) should be reserved for wounds that are not expected to heal or those in which the local bacterial burden is of greater concern than the stimulation of healing.

The second consideration is to control the bacteria in the wound. It is important to differentiate between contamination and the colonization of bacteria in a wound. On the continuum of bacterial burden, contamination is the presence of bacteria on the wound surface. Colonization reflects the presence of replicating bacteria that is not yet causing injury to the host. Critical colonization occurs when the presence of bacteria (or bio-burden) delays or stops healing, but without signs or symptoms of infection. When pain, erythema, warmth, purulent discharge, odor or new breakdown is present, local infection must be suspected. Systemic infection is marked by symptoms which extend beyond the borders of the wound. Symptoms may include erythema, induration, fever, and leukocytosis. These symptoms indicate the need for treatment with a systemic antibiotic.

The swab cultures of contaminated or colonized wounds are of limited diagnostic value. If a wound culture is indicated, a tissue biopsy is preferred.

air loss or air fluidized mattress for pressure relief. Adjunct therapies should also be considered. This may include negative pressure wound therapy, physical therapy, growth factor modalities and referral to plastic surgery when indicated.

SUMMARY

Let us now revisit the patient MS. The clinician performed sharp debridement of the devitalized tissue with a scalpel. The remaining slough was removed using enzyme therapy - Santyl Collagenase, for example. The debridement revealed a stage III ulcer. The wound was irrigated daily with normal saline and lightly packed with a hydrogel-impregnated dressing (to provide moisture). The macerated skin surrounding the wound was protected from excessive moisture using a topical material, Skin Prep, for example. A nutritionist was consulted, who advised a multivitamin and a protein supplement. Wound closure was achieved in a timely fashion.

REFERENCES

1. Niezgoda, JA, Mendez-Eastman S. The effective management of pressure ulcers. *Advances Skin Wound Care* 2006; 19(1) Supplement 1: 3-15.
2. <http://www.npuap.org>
3. <http://www.npuap.org/DOCS/DTL.doc>
4. Falanga V. Wound bed preparation and the role of enzymes. *Wounds* 2002; 14:47-57.
5. Ayello EA, Cuddigan JE. Conquer chronic wounds with wound bed preparation. *The Nurse Practitioner* 2004; 29: 8-25.
6. Sibbald RG, Williamson D, et al., Preparing the wound bed-debridement, bacterial balance, and moisture balance. *Ostomy Wound Manag* 2000; 46: 25.

Disclosure of Financial Interests

The authors have no financial interests to disclose.

9SOW-RI-GERIATRICS-12009

THE ANALYSES UPON WHICH THIS PUBLICATION IS BASED were performed under Contract Number 500-02-RI02, funded by the Centers for Medicare & Medicaid Services, an agency of the U.S. Department of Health and Human Services. The content of this publication does not necessarily reflect the views or policies of the Department of Health and Human Services, nor does mention of trade names, commercial products, or organizations imply endorsement by the U.S. Government. The author assumes full responsibility for the accuracy and completeness of the ideas presented.