

Nutrition, Physical Assessment, and Wound Healing

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Abstract

The relationship between nutrition and pressure ulcer healing has been a focus of ongoing research and discussion. Commonly implemented nutrition interventions for wound healing include protein and micronutrient supplementation. A variety of biochemical and clinical monitoring tools may be useful in predicting risk for skin breakdown and evaluating the efficacy of nutrition interventions intended to accelerate healing. This article reviews the rationale for routine nutrient supplementation to encourage wound healing, research that substantiates such use, and biochemical monitoring tools that may assist the dietitian in supporting pressure ulcer healing. In addition to traditional elements of a nutrition assessment, the dietitian should consider physical assessment of the pressure ulcer. This article provides the clinician with the basic terminology and characteristics typically used to describe pressure wound condition and healing. Nutrition-focused physical assessment allows the clinician to determine wound condition more accurately, address the wound proactively, identify non-nutritional factors that may influence healing, and implement realistic nutrition interventions.

Introduction

Adequate nutrition plays a fundamental role in the healing of pressure ulcers. The National Pressure Ulcer Advisory Panel includes nutrition as one of the key systemic factors that may influence healing (1). Adequate intake of protein, energy, vitamin C, zinc, and vitamin A frequently forms the basis for nutrition interventions to heal pressure ulcers. A variety of nutrition assessment tools also are employed, including biochemical tests, anthropometric data, and detailed diet history. In 2008, we reviewed a sample of 68 patients at one long-term acute-care hospital (LTAC), who had demonstrated skin breakdown or delayed wound healing, to capture correlations between their wound condition and

nutritional status. The primary goal was to educate clinicians and nursing staff and improve overall patient outcomes. Positive correlations were observed between delayed wound healing and depleted visceral protein stores, inadequate nutritional intake, and suboptimal blood glucose control.

Thorough, accurate nutrition assessment unquestionably requires a multidisciplinary approach. Several key wound characteristics should be reviewed routinely by the dietitian when completing nutrition assessment for individuals who have pressure ulcers. The dietitian who completes a nutrition-focused physical assessment is better prepared to gauge a patient's risk for delayed wound healing, evaluate the effectiveness of current nutrition interventions, and foresee non-nutritional factors that may impede healing. Doing so allows for appropriate use of nutrient supplementation and effective nutrition therapy.

Physical Wound Assessment

Wound care clinicians at the previously mentioned LTAC use the Bates-Jensen Wound Assessment Tool for ongoing measurement of wound status (2). Introduced in 1990, the tool consists of 13 scored parameters that include wound size and stage, necrotic tissue type, wound exudate type, skin color surrounding the wound, and peripheral tissue edema. Scores can range from 13 to 65, with 13 representing tissue regeneration (3). Understanding physical wound characteristics, terminology, and the rationale behind a specific institution's wound assessment tools allows the dietitian to provide comprehensive nutrition assessment for the patient who has a pressure ulcer.

Depth/Stage of Pressure Wound

Depth and stage of the pressure wound are perhaps the most easily recognized measures of wound severity. The updated staging system for pressure ulcers developed by the National Pressure Ulcer Advisory Panel can be found at www.npuap.org/pr2.htm. Stage I and II ulcers generally are considered

the least severe and stage IV ulcers are considered most severe, but a broad range of skin damage or necrosis may be represented within the stage III classification. A stage III wound may appear shallow and superficial or as a full-thickness wound (4,5). The depth of a stage III pressure ulcer varies, depending on its anatomic location. For example, areas with little or no subcutaneous tissue, such as the bridge of the nose, ear, and malleolus, can have shallow stage III pressure ulcers. In contrast, areas with significant adipose tissue can develop extremely deep stage III pressure ulcers (4). For this reason, stage II and III wounds may be misdiagnosed, and consultation with wound care clinicians may be necessary to clarify wound severity.

The presence of necrotic tissue is a significant barrier to wound healing and, therefore, often is removed by debridement (5). Enzymatic, surgical, mechanical, or autolytic debridement all serve the same goal of removing nonliving necrotic or black eschar tissue to allow living tissue to heal (5). However, necrotic tissue may benefit the wound by creating a stable barrier to infection. For example, necrotic tissue that forms on the heels of feet generally is considered "stable eschar," is highly protective against infection, and should not be removed by debridement (6). In some cases, the progression toward wound necrosis may be foreseen. Generally, as wound tissue becomes necrotic, it initially is light yellow and nonadherent, advancing to loosely adherent yellow slough before finally transitioning to black eschar (2). A wound base that is covered by slough or necrotic tissue (eschar) generally is deemed "unstageable" (6). The depth and stage of the wound is difficult to determine accurately until sufficient amounts of slough/eschar are removed and the base of the wound is exposed (6).

A vacuum-assisted closure device may be used to treat chronic nonhealing or dehiscent wounds (7). The device employs vacuum suction to close the wound. A vacuum-

assisted closure device is contraindicated in several instances, including in the presence of osteomyelitis; malignancy; and hard, black eschar (7).

Wound Edge Assessment

Understanding the condition of the wound edge aids in clarifying wound severity. "Undermining" describes skin surrounding the wound that has become detached from the underlying fascia (5). It appears as overhanging skin edges at the wound margin, with the pressure ulcer having a larger area at its base than at the skin surface. Undermining typically is caused by bacterial infection and commonly is associated with osteomyelitis (8). When undermining extends through the tissue and perhaps connects to another wound, tunneling has occurred. Undermining and tunneling may delay wound healing by interrupting blood circulation and oxygen perfusion to the wound site (9).

Wound Exudate

The type and amount of wound exudate is an extremely useful characteristic for the dietitian to assess. A high-output draining wound may result in significant loss of fluid, protein, and zinc (10–12). Foul-smelling, purulent wound drainage is suggestive of infection (5). Infectious complications occur frequently in stage IV pressure ulcers (13), and the risk of wound dehiscence is increased with infection (14). Infection delays wound healing by impairing collagen formation (9).

Other Considerations

Edema and induration (firmness due to localized inflammation and trapped fluid, often caused by edema) surrounding a wound may indicate infection (15). Although the exact mechanism by which chronic edema delays wound healing is unknown, it appears to interfere with normal circulation and may impede the flow of normal inflammatory cells to the wound site (16). Removal of cell metabolites and debris also may be interrupted by the presence of edema (16).

The appearance of granulation tissue and the extent of epithelial tissue also should be assessed. In general, bright, beefy-red granulation tissue at the wound site is a

healthy indication of good circulation (15). Alternatively, pale pink granulation tissue may be a physical sign of decreased circulation to the wound site that suggests the potential for delayed healing (17). The extent to which a wound is covered with epithelial tissue indicates how well the wound is healing. A wound that is 100% epithelialized indicates intact skin surface (6).

Relying solely on assessment of wound size as an indicator of wound healing or decline is not sufficient. For example, pressure wounds often become larger following debridement (5), in which case the clinician may falsely assume that the wound has worsened. Therefore, to assess wound healing accurately, the dietitian must be aware of whether a wound has been debrided.

Various extraneous factors may contribute to the development of new wounds and affect healing of existing wounds. Skin exposed to excessive moisture, often from urine, feces, or perspiration, is more likely to ulcerate than dry skin (18). Conversely, skin with too little moisture is more likely to ulcerate than healthy skin. Heat, friction, and irritation from wrinkled bedsheets also may contribute to skin breakdown (5). Certain medications, including glucocorticoids, immunosuppressive agents, and anticoagulants, may interfere with wound healing (10,15). For example, corticosteroids can delay healing by impairing collagen synthesis and inhibiting fibroblast proliferation to the wound site (9).

Obese individuals are more prone to wound infection and impaired wound healing because adipose tissue is poorly vascularized, resulting in decreased blood flow to the wound and impaired delivery of nutrients (19). Obese patients also have a higher incidence of wound dehiscence because surgery can be technically more difficult and there is a higher risk of hematoma formation (19).

Physical Assessment and Nutrient Supplementation

Vitamin C

Vitamin C is essential for collagen formation and, thus, critical for wound healing (20).

Sufficient amounts of vitamin C stabilize the collagen structure by increasing tensile strength and maintaining elasticity of connective tissues (20). As noted previously, the presence of wound infection may be evident by purulent, odorous wound exudate. Infection may suggest the need to increase vitamin C intake to assist the body's ability to resist infection (21–23). Improved resistance to infection from microbial pathogens has been demonstrated with adequate vitamin C intake (23,24). An eightfold increase in wound dehiscence has been reported in patients who had depleted concentrations of vitamin C compared with individuals who had adequate concentrations (25). Vitamin C supplementation in nondeficient patients has not been shown to accelerate wound healing (26,27).

Vitamin C concentrations can be decreased significantly in critically ill patients and persons who have diabetes, cancer, and liver disease (21,28,29). Acute stress, smoking, burns, and infection can increase requirements for vitamin C (29). Vitamin C deficiency may be unrecognized for prolonged periods of time. Teixeira (30) reported that 88% of elderly hospitalized patients had vitamin C deficiencies, with the lowest concentrations observed among patients admitted from nursing homes. Among the 68 LTAC patients we evaluated who demonstrated skin breakdown or delayed wound healing, 67% did not have vitamin C supplementation ordered by their physicians. Given the potential for malnutrition in the chronically ill LTAC patient, vitamin C deficiency may have existed in these 68 individuals. Its low risk of toxicity and observed ability to support wound healing suggests consideration of vitamin C supplementation in the presence of skin breakdown or delayed healing.

The recommended dose of vitamin C for wound healing ranges from 500 to 1,000 mg/day (10,31,32). Patients who have stage III or IV pressure ulcers may benefit from supplementation of 500 mg twice daily for 14 days (31,33). Ongoing use generally is not considered harmful, but the clinician must use discretion when recommending a vitamin C supplement (31,33). The risk is low,

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but toxicity may occur in patients who are at risk for oxalate stone formation (20,31,33). To maximize wound healing but minimize soft-tissue damage in patients with renal failure, research suggests a maximum recommended intake of 100 mg/day (34–36). Most renal-specific multivitamins contain approximately 60 to 100 mg of vitamin C.

Zinc

Zinc plays an essential role in proper immune function, collagen synthesis, and wound healing (11,37,38). When assessing wound exudate, the clinician should be aware that zinc deficiency can occur as a result of excessive wound drainage (11,39). In the presence of a wound, the body redistributes zinc such that wound concentrations increase while normal skin concentrations decrease (40). Zinc deficiency may delay pressure ulcer healing by minimizing wound tensile strength and inhibiting wound closure (37,41). Zinc is a cofactor for the synthesis of protein and is transported by albumin (11,40). Zinc deficiency has been shown to correlate positively with depleted albumin, prealbumin, and transferrin (41). Therefore, zinc supplementation may improve healing by replenishing visceral protein stores (41). Bates and McClain (41) found that serum albumin, prealbumin, and transferrin were depleted in patients who were zinc-deficient. Concentrations of all three visceral proteins improved when zinc was administered as the sole therapeutic intervention, with the most rapid response observed in prealbumin (41).

If zinc deficiency is *suspected*, such as in the presence of substantial wound exudate, delayed wound healing, hair loss, long-term corticosteroid therapy, advanced age, or poor nutritional intake, a maximum 14-day dose of 220 mg zinc sulfate (equivalent to 50 mg elemental zinc) may correct the deficiency (10,11,37,42). However, providing supplemental zinc to the nondeficient patient has *not* been shown to accelerate healing (11,42–44).

Even modestly increased intake, close to the Recommended Dietary Intake of 11 mg/day for adult males and 8 mg/day for adult females, may interfere with copper

absorption (11,45). Decreased copper absorption may delay wound healing by suppressing the immune system, increasing susceptibility to wound infection, and reducing oxygen transport to cells through inhibition of iron absorption (11,44). Copper deficiency also is problematic for wound healing because copper is a cofactor for collagen synthesis and collagen cross-linking (11). A study by Fischer and associates (44) found that supplementation of zinc in a moderately high dose (50 mg/day of elemental zinc) for 6 weeks resulted in decreased serum copper status.

The goal for zinc supplementation should be to replenish the body's zinc concentrations in the presence of known or suspected deficiency without providing excessive amounts. Among the observed LTAC patients with nosocomial or declining pre-existing wounds, 74% had not been prescribed a zinc supplement by the physician. The potential for malnutrition in the chronically ill LTAC patient and the presence of nonhealing pressure ulcers or new skin breakdown suggest the potential presence of zinc deficiency in these patients. Therefore, in such cases, a maximum 14-day dose of 220 mg elemental zinc may be warranted. However, because zinc appears to have no known benefit for the nondeficient patient and may have deleterious effects, zinc supplementation should be recommended with caution on a case by case basis.

Vitamin A

Vitamin A plays an integral role in cell division, cell differentiation, and immune system function (46). Adequate concentrations of vitamin A appear to influence the body's therapeutic inflammatory response to wounds (37). Although the mechanism by which this occurs is not understood entirely, vitamin A clearly plays an important role in wound healing. Vitamin A is believed to augment the inflammatory response by enhancing lysosomal membrane lability, increasing macrophage influx, and stimulating collagen synthesis (37). In vitro studies have shown increased collagen synthesis of fibroblast cell cultures in the presence of vitamin A (37).

Depletion of vitamin A can result in impaired wound healing and susceptibility to infection. Vitamin A deficiency is rare because large amounts are stored in the liver (10). If a deficiency is suspected, the recommended dose is 700 to 3,000 IU (with the higher range for males) (39). Megadoses of vitamin A, up to 25,000 IU/day, occasionally are administered to patients receiving steroid therapy (20) because high doses of corticosteroids can deplete hepatic stores of vitamin A (37). Vitamin A has been shown to reverse the corticosteroid-induced adverse effect of inhibited cutaneous and fascial wound healing (47). Such mega doses of vitamin A appear to diminish the number of infectious complications (48). Doses of 25,000 IU/day (more than five times the Dietary Reference Intake) have been used without any significant adverse effects (36). Decreased serum concentrations of vitamin A, retinol-binding protein, retinyl esters, and beta-carotene have been noted after burns, fractures, and elective surgery (37). Vitamin A also has corrected nonsteroid-induced, postoperative immune depression (47). Patients receiving mega doses of vitamin A should be monitored closely for wound improvement. The course of treatment generally is limited to 7 to 12 days (19). Prolonged supplementation does not appear to improve wound healing, and excessive vitamin A intake can be toxic (37). Vitamin A toxicity may contribute to hypercalcemia in patients undergoing hemodialysis, and excessive use of vitamin A shortly before and during pregnancy may increase the risk of birth defects (49,50). Vitamin A intake should not exceed 5,000 IU/day for patients during hemodialysis and 3,300 IU/day during pregnancy (49,50). Multivitamin preparations containing vitamin A should be used with caution.

Protein

Protein malnutrition delays wound healing by impairing collagen synthesis and deposition and decreasing wound strength (37). Protein malnutrition also diminishes immune function, which increases the potential for wound infection and, thus, delayed healing (37). Prolonged inadequate protein intake may promote tissue edema, which delays

healing by slowing oxygen diffusion from capillaries to cell membranes. The presence of a significant pressure wound alone induces a hypermetabolic and catabolic state, thereby increasing nutrient needs (40). Inadequate protein intake in the presence of a wound promotes loss of lean body mass. Because the wound is the preferred site for use of available nutrients, the body initially draws upon lean muscle mass to provide the amino acids necessary for wound healing (40). A 15% or greater loss of lean body mass is associated with impaired wound healing; a 30% or greater loss is associated with development of new wounds (40).

Significant protein losses can occur from draining wounds or fistulae, the extent of which is difficult to quantify (12). Unless exogenous protein losses are suspected to be significant, protein intake of 1.5 to 2 g/kg generally is adequate (19). However, some studies have shown that as much as 100 g/day of protein may be lost through wound exudate, which varies according to several factors, including the size and depth of the wound (51,52). Protein modulators may be necessary to provide adequate protein for wound healing. The Protein Digestibility Corrected Amino Acids Score (PDCAAS), established by the Food and Agriculture Organization/World Health Organization, evaluates quality and digestibility of a protein (such as egg, casein, and whey) against a standard reference (53). Consideration of the PDCAAS may provide useful guidance when selecting from the variety of available liquid and protein modulators.

Glutamine, the most abundant amino acid in the body, accounts for 60% of the intracellular amino acid pool (36,40). Glutamine may benefit wound healing by stimulating the immune response, promoting anabolism, providing fuel for rapidly dividing epithelial cells during healing, and acting as an antioxidant (36,40). Glutamine supplementation has been shown to increase survival in individuals who have major burns (40), but its impact on healing of pressure ulcers has yet to be substantiated.

Arginine is another amino acid commonly

associated with pressure ulcer healing (9,37). Arginine is a precursor of proline in collagen and stimulates release of anabolic hormones, such as insulin-like growth factors and human growth factors, that promote wound healing (20,40). High doses of arginine have been shown to increase tissue collagen content (40), but the benefit of arginine supplementation on chronic or nonhealing wounds is not well defined (9,40).

Energy

Adequate energy intake is required for synthesis of collagen and nitrogen, anabolism, and wound healing (54). Energy requirements for individuals who have stage III or IV pressure ulcers are approximately 35 to 40 kcal/kg/day of body weight (20). However, clinical judgement should be used to adjust for energy needs on an individual basis, with consideration of multiple factors, including wound severity, patient age, and the presence of obesity (54).

Additional Considerations

Use of biochemical parameters is an important component of a thorough nutrition assessment. Commonly assessed serum proteins include albumin, prealbumin, and transferrin. Depleted visceral protein concentrations may indicate malnutrition and are associated with increased risk for pressure ulcers (55,56). Because albumin has a long half-life, it should not be used to assess daily changes in nutritional status (37), although it may be helpful in classifying chronic undernutrition (39). The shorter half-life of prealbumin may make it more useful in evaluating nutritional status in acute settings as well as progress in refeeding (20,39). Among the LTAC patients who had worsening or nosocomial wounds, 67% had below-normal prealbumin concentrations. A prealbumin value of less than 15 mg/dL is suggestive of depletion (<30 mg/dL for individuals with chronic renal failure) (57). Decreased prealbumin in the presence of poor wound healing and new skin breakdown may warrant increased protein and/or energy intake. Of note, though, prealbumin, albumin, and transferrin are negative acute-phase reactants (20,39,57). Therefore, depleted concentrations may result from decreased hepatic synthesis in the presence of inflammation (58) or liver

disease, infection, stress, and sepsis (20,39). Retinol-binding protein is considered a sensitive indicator of malnutrition, although it is difficult to measure and not typically used in clinical practice (57).

Transferrin is a relatively inaccurate marker for assessing nutritional status due to its relationship with iron (39). However, along with percent saturation values, transferrin can help distinguish an inability to use iron stores due to iron-deficiency anemia (55). Anemia is considered a major risk factor for pressure ulcers (56), making hemoglobin and hematocrit important to assess.

Hyperglycemia can be a barrier in wound healing for a number of reasons. Increased blood glucose concentrations can impair blood flow to the wound, thus reducing adequate oxygen and nutrients needed for wound healing (59). In addition, decreased immunity associated with hyperglycemia and diabetes contributes to poor wound healing (59,60). Chemotaxis (the process by which white cells are brought to the infection site) and phagocytosis (the ingestion of bacteria by white cells) are processes necessary for wound healing and controlling infections. Both can be compromised in the presence of elevated blood glucose (37). Hyperglycemia is believed to interfere with the transport of ascorbic acid into fibroblasts, which may, in turn, delay wound healing (37). Finally, a lack of or inability to use insulin impairs metabolism of carbohydrates, fats, and proteins, all of which play crucial roles in wound healing (59). Maintaining blood glucose control, therefore, is critical to achieve optimal wound healing.

Hydration plays many roles in the wound healing process. Adequate fluid is essential to hydrate the wound site, aid in oxygen perfusion, and transport materials to and from the site (20). Fluid also acts as a solvent for vitamins, minerals, and amino acids (20). Hydration status can be assessed through blood urea nitrogen (BUN), BUN/creatinine ratio, serum osmolality, and urine specific gravity; elevated values may suggest hypovolemia (20,57). A disproportionate

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rise in BUN compared with creatinine is suggestive of dehydration (57). Urine specific gravity and osmolality are considered nonspecific when assessed in isolation, but they reflect the kidney's ability to concentrate or dilute urine appropriately, potentially making them helpful in assessing hydration status (57).

Hypocholesterolemia (<160 g/dL), along with poor appetite and weight loss, often is considered a potential nutritional risk factor (20,56). Stress, severe infection, and cytokine-induced inflammatory states decrease serum cholesterol (20). Other laboratory values that should be assessed include total lymphocyte count and C-reactive protein. Elevated values for each can indicate infection that, if not managed correctly, can delay wound healing (37).

Conclusion

The integral relationship between nutrition and pressure ulcers underscores the critical role of the dietitian in providing effective nutrition interventions to support wound healing. Supplementation of vitamin C, vitamin A, and zinc is a common practice for treatment of severe pressure ulcers. Further research is needed to clarify the potential benefits of these and other nutrition supplements, including glutamine, arginine, and the various forms of protein modulars, in accelerating healing. Monitoring of biochemical data provides evidence of an individual's nutritional status and progress toward achieving nutrition goals. Assessment of visceral proteins, such as prealbumin, albumin, and transferrin, may be useful but should not be considered definitive indicators of nutritional status. The potential ability of hyperglycemia and inadequate hydration to delay wound healing make glucose and hydration monitoring key components of nutrition assessment for individuals with pressure ulcers.

Our observational review of 68 LTAC patients with worsening pre-existing or newly developing wounds provided insight into the opportunities for dietitians to affect wound healing with nutrition intervention. Among the patients we observed, positive correlations were noted between skin breakdown and low prealbumin and elevated serum glucose

Figure. Nutrition Assessment for Wound Healing Checklist

Patient: _____

Nosocomial Wound: _____

Declining Wounds: _____

Stage of Wounds: _____

Exudate Amount: _____ Scant _____ Moderate _____ Large

Tunneling/Undermining: _____ Present _____ Not Present

Induration/Edema: _____ Present _____ Not Present

Necrotic Tissue: _____ % **Epithelialization:** _____ %

Granulation: _____ % **Color:** _____

Prealbumin (or other recent visceral protein level): _____

Intake

PO: _____ Appetite?: _____

Supplement/Protein: _____

TF: _____

Average TF Infusion Rate: _____ % Infused

PN: _____ g protein Appropriate? _____

Blood Glucose

Recent Accuchecks: _____

Average Accuchecks (past week): _____

On prednisone? _____

Insulin regimen? _____

Vitamins and Mineral Supplementation? (Check if ordered)

Multivitamin _____ Zinc _____

Vitamin C _____ Vitamin A _____

Hydration Status

BUN/Creat: _____ Water flushes: _____

Sodium: _____ IV fluids: _____

values, inadequate nutritional intake, and absence of vitamin/ mineral supplementation. These observations provided the framework for recommendations later made to the LTAC's Medical Executive Committee that included educational efforts to increase multi-disciplinary awareness of the relationship between nutrition and wound healing,

reassessment of routine micronutrient supplementation for patients with severe pressure wounds, and the acquisition of high-quality protein supplements.

Finally, the incorporation of wound assessment in nutrition-focused physical assessment can enhance the dietitian's ability to support wound

healing. Understanding of the institution's wound assessment tool (Figure) and the key terminology and physical wound characteristics is essential to providing optimal nutrition care to individuals with pressure ulcers. Such understanding allows for more accurate assessment of wound severity, the identification of non-nutritional factors that influence healing and increase an individual's risk for skin breakdown, and overall enhancement of the quality of care provided by the dietitian. Ongoing collaboration between the dietitian and wound care professional is an indispensable element of providing quality care to patients with pressure ulcers.

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