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Learning the Oral and Cutaneous Signs of Micronutrient Deficiencies

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■ ABSTRACT

Wound healing is a complex process that is influenced by multiple systemic factors, including nutritional status. While nutritional support is commonly recognized as an important aspect of comprehensive wound management, the focus is typically on replacement of macronutrients, specifically calories and protein. Our experience strongly suggests that micronutrients are equally important, that micronutrient deficiencies are common, and that correction of these deficiencies frequently leads to wound healing when incorporated into a comprehensive wound management program. This article provides guidelines for assessment and management of micronutrient deficiencies.

KEY WORDS: illustrations of oral and cutaneous signs of micronutrient deficiency, micronutrients in wound healing, oral and cutaneous signs of micronutrient deficiency

■ Introduction

This article reviews an often-neglected but clinically relevant topic for clinicians involved in wound care: the critical role of micronutrients in healing of chronic wounds. Most wound care clinicians recognize the importance of nutritional assessment and management[1]; however, most nutritional assessment tools and nutritional management guidelines fail to include the diagnosis and management of micronutrient deficiencies. Rather, most assessment guidelines have focused on the visceral and somatic protein status of the patient and identification of protein and calorie deficits. Subsequently, most nutritional interventions have focused on replacement of macronutrients (protein, carbohydrate, and fat) to repair these deficits.[2] Replacement of macronutrient deficits generally requires 25 to 35 nonprotein calories/kg and 0.8 to 1.2 g of protein/kg of body weight per day. Replacement of these macronutrients is an important aspect of nutritional management and is consistently addressed in evidence-based guidelines published by the WOCN Society, as well as the National Pressure Ulcer Advisory Panel and European Pressure Ulcer Advisory Panel. However, we submit that micronutrient replacement is equally important but is not typically addressed in pertinent clinical practice guidelines. Notably, discussion and images of the oral and cutaneous signs of micronutrient deficiencies are not included in these guidelines.

The goal of this article is to make the reader aware of the need to identify and correct deficiencies of micronutrients essential for wound healing. For the purposes of this discussion, the pressure ulcer will be used as an exemplar of the chronic wound. Nutritional compromise is widely recognized both as a risk factor for pressure ulcer development and as a contributing factor to failure to heal.[3-6] However, nutritional deficiencies are sometimes missed or overlooked because many of these patients are chronically ill or of advanced age, and clinicians may misperceive indicators of nutritional deficiencies as typical indicators of aging or illness. In addition, clinicians frequently assume that patients who are eating are not at risk for nutritional compromise, and fail to adequately assess the types and volumes of nutrients being consumed. Fortunately, there are specific oral and cutaneous signs of micronutrient deficiencies that can be incorporated into the physical assessment of any at-risk patient during admission skin assessment and/or wound consultation. To date, no paper has been published with images of the typical signs of micronutrient deficiencies or with recommended supplements for patients exhibiting these signs.

The elderly are at particular risk for both macronutrient and micronutrient deficiencies. This risk may be associated with comorbid conditions affecting appetite or nutrient absorption or changes in the social environment that affect food intake, such as the death of a spouse.

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Another potential contributing factor is economic pressures that limit food choices or result in unintentional food rationing. For example, some individuals reduce their purchase of fresh fruits and vegetables because "they cost too much and/or spoil too quickly." Since fresh fruits and vegetables are the main source of most essential vitamins and minerals, this change in itself places the individual at risk for micronutrient deficiencies. This is particularly true of water-soluble vitamins such as vitamin C, which requires daily intake to prevent rapid depletion that adversely affects metabolic processes and protein synthesis, particularly collagen production.[7]

While most malnourished individuals experience both macronutrient and micronutrient deficiencies, the signs of macronutrient deficiencies occur first and are more easily recognized. They include weight loss and changes in visceral protein laboratory values. These deficiencies, referred to as marasmus and kwashiorkor, are the focus of most nutritional assessment tools and nutritional replacement programs.[8]

However, it is important to realize that micronutrient deficiencies develop concomitantly with the more visible macronutrient deficits and can lead to impaired immune function and compromised wound healing if not corrected. Micronutrients are primarily intracellular, so the cell shrinkage associated with loss of lean body mass also results in deficiencies of these micronutrients. These observations strongly suggest that nutrient loss must be replaced and that nutritional support programs should include attention to micronutrient replacement as well as replacement of calories and protein.[9] Thus, the patient must be routinely examined for oral and cutaneous signs of micronutrient deficiencies.

Recommendations for Prevention and Treatment of Pressure Ulcers

In the early 1990s, a panel of experts assembled by the US Department of Health and Human Services developed *the Guideline Technical Report, number 15, Treating Pressure Ulcers, Volume 1*. Chapter 4 addressed nutrition and included recommendations for diagnosis and treatment of both micro- and macronutrient deficiencies (Table 1).[9] In subsequent reports from several pressure ulcer study groups, nutritional support is acknowledged as necessary but specific recommendations are generally limited to protein and calories and do not address micronutrients.[9,10] Nutritional compromise is commonly recognized as a contributing factor to delayed healing, and any delay in healing is costly. The prevalence of pressure ulcer ranges from 11.6% to almost 50% for patients confined to hospitals and nursing homes, of which 30% are stage III and IV wounds.[11,12] Consequently, it would be correct to conclude that the economic impact runs into the billions of dollars annually that affects tens of thousands of people worldwide.[13]

TABLE 1.

Recommended Micronutrient Supplement Dosages[a]

Multivitamin—1 tablet twice daily
Vitamin C—500 mg twice daily
Vitamin D_3—2000 IU twice daily
Zinc sulfate—220 mg twice daily
Fish oil—1 g twice daily
Vitamin B_3—250 mg twice daily (If skin change is consistent with pellagra noted)
Glucosamine/chondroitin 600/400 mg twice daily (If skin changes consistent with ECM depletion and hydration noted)

[a]Recommendations are based on the authors' experiences.

The efficacy of any wound treatment program is dependent on the patient's ability to heal the wound, and this ability is affected significantly by nutritional status. In 1989, a study was completed that focused on nutritional assessment of residents in 2 nursing homes.[3] The evaluation included serum vitamin levels and photographs to document the oral and cutaneous signs of vitamin deficiencies. Marasmus and kwashiorkor were diagnosed by anthropometric measurements as well as serum albumin and total lymphocyte count. The incidence of malnutrition was 32% and all patients with pressure ulcers were found to be severely malnourished. The investigators concluded that a pressure ulcer is a sign of malnutrition.

It is clearly important to thoroughly assess all chronic wound patients for evidence of both macronutrient and micronutrient deficiencies, and to intervene as needed to correct those deficiencies. This article addresses current understanding of the way in which each micronutrient contributes to wound healing, indicators of deficiency, and current recommendations for treatment. Illustrations of common oral and cutaneous indicators of micronutrient deficiencies are provided.

Water-Soluble Vitamins

Vitamin B

B vitamins are needed for various metabolic tasks essential to health.[14] They generally function as coenzymes and cofactors in the cytochrome oxidase pathway, which produces adenosine triphosphate (ATP).[15] Because they are water soluble, they are rapidly excreted in the urine, and deficiencies can develop quickly that negatively affect tissues with high metabolic rates and high cellular turnover rates. Examples include the hematologic system, gastrointestinal tract, and tissues involved in the repair processes such as wound healing. The fact that they are water soluble means that toxicity is typically not an issue; the only exceptions are vitamins B_3, B_6, and B_{12}.[15] Toxicity with vitamins B_3, B_6, and B_{12} is caused by ingestion of megadoses and mediated by an unknown mechanism.

Isolated "single" B vitamin deficiencies do not occur in clinical practice; they can be created only in animals in the laboratory where a diet is prepared excluding the specific B vitamin under study. The one exception is pernicious anemia due to Vitamin B_{12} deficiency, which can occur as a result of altered absorption.

Glossitis is the hallmark of B vitamin deficiencies.[16] Normally, the tongue is covered with villi that give it a velvety appearance; the lingular villi are long enough to hold a small saliva froth causing a slight whitish appearance. The healthy tongue also has a minimal cyanotic or light purplish hue because the tips of the villi are slightly more venous than their arterial bases. Vitamin B deficiencies cause atrophy of the velvety surface, resulting in a reddish-colored tongue with a smooth surface; specific findings are dependent on the predominant B vitamin involved in the deficit. For example, a primary B_2 (riboflavin) deficiency produces a magenta-colored flank steak appearing surface and may also cause cracks at the corners of the mouth, a finding known as angular stomatitis (Figure 1). In contrast, vitamin B_{12} deficiency is characterized by hypertrophic papillae scattered across the villous surface of the tongue.[16]

Vitamin B deficiencies and glossitis are rare in patients on tube feeding, because the recommended daily allowance for B vitamins in tube-feeding formulas is sufficient to prevent deficiencies. However, deficiencies are common in patients on oral feeding and the resulting glossitis is difficult to clear even with the administration of a multivitamin given twice daily in addition to a normal diet. This raises questions regarding the ability of the gut to absorb oral multivitamins, especially for the elderly. Glossitis can be attributed to many causes; however, in our experience, B vitamin deficiency is the most common cause, particularly in the elderly.

Current guidelines for treatment of vitamin B deficiency include a liquid multivitamin administered at the label-recommended dose twice daily. It is our practice to observe for clearing of the glossitis and to increase the multivitamin dose if needed and add a B vitamin twice daily if necessary. Fortunately, toxicity is not an issue (Table 2).

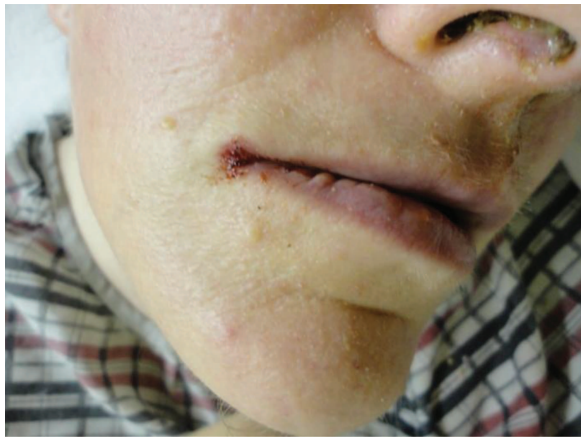


FIGURE 1. Angular stomatitis is a moist crack in the mucosa at the angle of the upper and lower lips. It is a sign of vitamin B_2 deficiency, usually accompanied by a magenta-colored glossitis.

Vitamin B_3 Deficiency

Vitamin B_3 is niacinamide and has a number of names and forms.[17] It is required for the function of more than 50 enzymes. Niacin is also required to convert excess sugar in the diet to fat and to support the production of ATP. In addition, niacin plays a key role in the production of sex hormones. While the body can produce niacin in limited quantities, adequate oral intake is required on a routine basis to prevent as well as repair deficiencies. The classic signs of niacin deficiency are the "three D's: dementia, diarrhea, and dermatitis." In addition, niacin deficiency has been implicated in bullous pemphigoid and granuloma annulare. The dermatitis associated with niacin deficiency is also known as pellagra and is characterized by a "crepe paper appearance" with wrinkles in the skin and flat surfaces in between the wrinkles.[17] The early stage can be compared to an ice pond in the spring, where thinning sheets of ice are broken into irregular small islands floating on water. If the skin is exposed to ultraviolet (UV) light, the "islands" become thick and scaly and the spaces in between turn cherry red (Figure 2). Nursing home patients frequently present with early lesions; however, it may be necessary to gently bunch the skin to accentuate the islands, which are most commonly seen on the extremities. Deficiencies in essential fatty acids (EFAs), in addition to niacin deficiency, can act synergistically to create prominent lesions on the anterior lower extremity (Figure 3; Table 2).

Recommended treatment for niacin deficiency is the administration of vitamin B_3, 250 mg twice daily. Treatment may provide improvement in cognitive function as well as resolution of the cutaneous manifestations.[18]

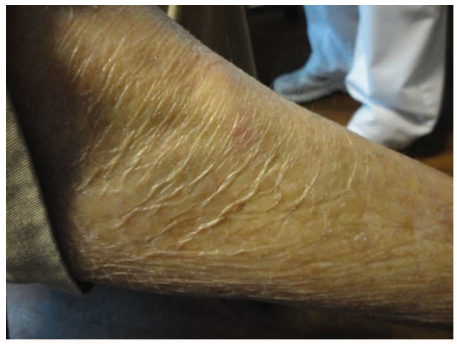
Vitamin C

Vitamin C plays a number of essential roles, including collagen synthesis. Collagen is produced by fibroblasts, via a

TABLE 2.

B Vitamin Adverse Effects

Vitamin	Harmful Effects
B_1—Thiamine	No known toxicity from oral intake
B_2—Riboflavin	No evidence of toxicity
B_3—Niacin	Intake of 3000 mg/day of nicotinamide and 1500 mg/day of nicotinic acid
B_5—Pantothenic acid	No known toxicity
B_6—Pyridoxine	Intake of more than 1000 mg/day
B_7—Biotin	No known toxicity
B_9—Folic acid	Masks B_{12} deficiency
B_{12}—Cyanocobalamin	Acne-like rash



(A)

(B)

FIGURE 2. (A) Pellagra is a vitamin B_3 deficiency. Early signs are a crepe paper appearance to the skin, as illustrated here. Thin islands of epidermis are separated by rivulets. (B) Advanced pellagra is the typical alligator skin noted in sun-exposed areas of niacin-deficient individuals. In the past, farmers working with an open collar developed these legions on their superior chest/inferior neck.

complex process involving frequent repetition of the amino acids proline and lysine along a linear structure. The microscopic structure of collagen has the appearance of twine with cords wrapped around each other and connected by cross-links. These cross-links are produced by hydroxylation of proline and lysine to produce hydroxyproline and hydroxylysine, and vitamin C is essential for this hydroxylation process.[19] Vitamin C plays a number of other essential roles involving the oral, ophthalmic, musculoskeletal, cardiac, and gastrointestinal systems; deficiencies can cause pathologic manifestations and, on occasion, death. A discussion of vitamin C's other roles is beyond the scope of this article.

Vitamin C is water soluble, so it is not stored in the tissues.[19] If the levels of vitamin C are insufficient to support the daily needs for collagen synthesis, the result is a progressive collagen deficiency. On the other hand, any intake in excess of the minimum daily requirement of 60 mg/day will be excreted in the urine.

Vitamin C deficiency, also known as scurvy, can occur in either acute or chronic form. The clinical presentation of acute scurvy has been described as the "4 H's" (hemorrhage, hyperkeratosis, hypochondriasis, and hematologic abnormalities); however, this form is rarely seen in pressure ulcer patients.[20] The chronic form of scurvy is characterized by a total body collagen deficit,[21] which causes skin and capillary fragility; the clinical presentation is purpura, skin tears, and pressure ulcers (Figures 4 and 5).

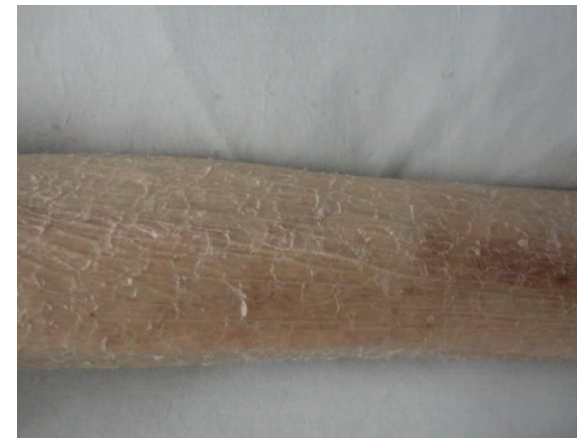


FIGURE 3. Essential fatty acid deficiency presents as a large "snowflake" exfoliated condition of the epidermis. It typically starts with the anterior area of the lower extremities. It can progress to involve the entire body. These "snowflakes" can be released by gentle rubbing and gathered off the surface of the mattress.

Early indicators of vitamin C deficiency include atrophy of the dermis, which is most easily observed on the dorsum of the hand, the forearms, and the temple area.[22] Specifically the clinician should assess the patient for purpura and for loss of mechanical "push back" or elastic resistance against external pressure. We have observed that severe collagen deficiency due to scurvy can cause a condition described as "plastic wrap" skin, where the dermis is so atrophic that the tendrils of the extensor tendons and the venous plexus can be easily seen beneath a

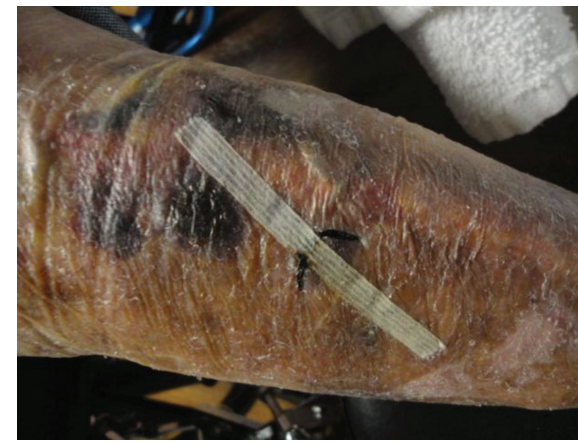


FIGURE 4. Profound chronic scurvy leaves the skin susceptible to a typical triangular skin tear. This may be a sign of scurvy and not a condition in itself; the patient should be assessed for other indicators of vitamin C deficiency and treatment should be initiated.

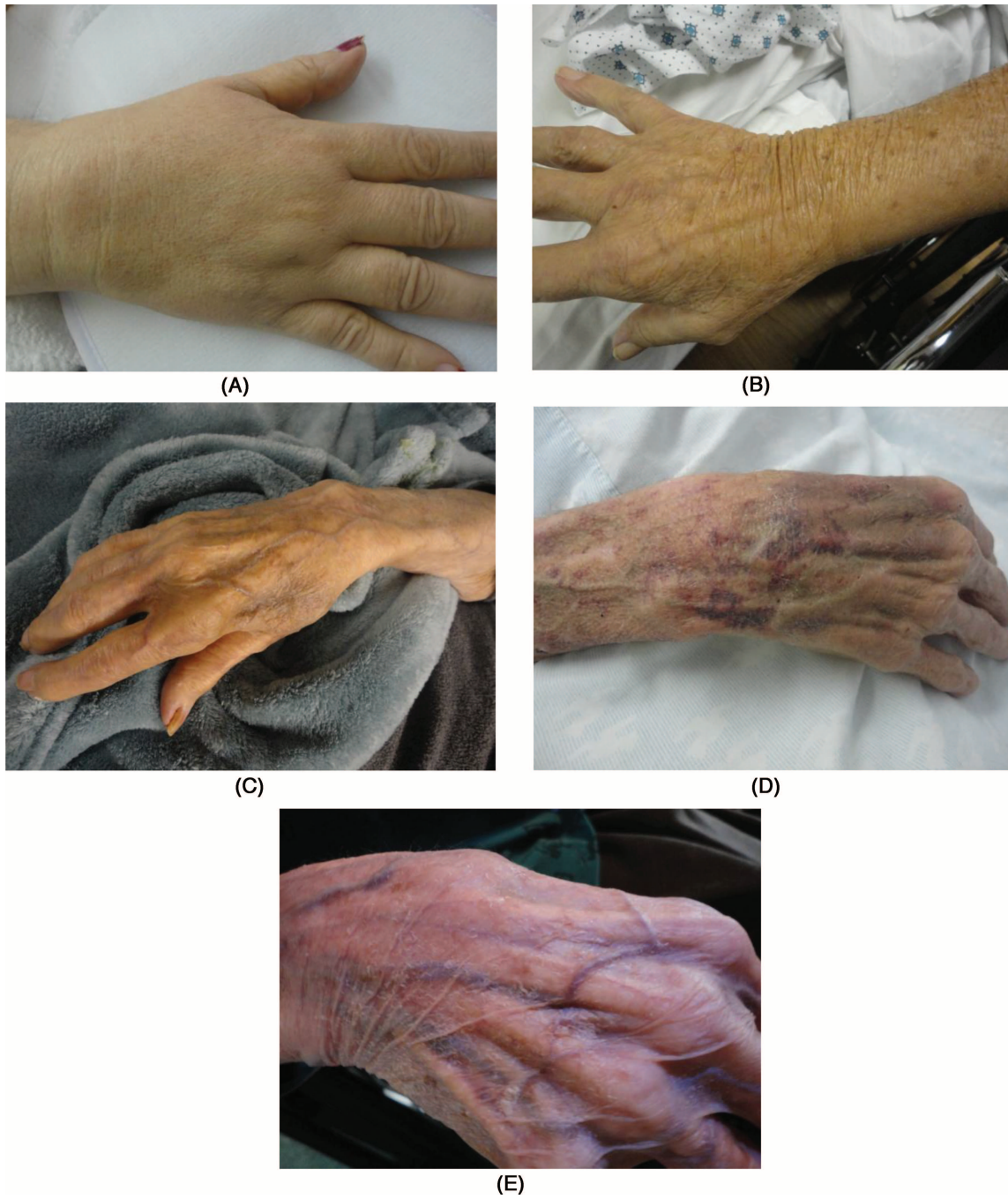


FIGURE 5. Chronic scurvy can be staged by observing the thickness of the dermis and its transparency. Early stages prevent direct observation of the tendrils of extensor tendons and the venous plexus over the tendon sheath. The skin becomes progressively transparent with stage IV, featuring a slightly opaque, transparent epidermis revealing an unobstructed view of the deeper anatomy. (A) Normal dermis; (B) +1; (C) +2; (D) +3; and (E) +4.

transparent epidermis. Damage to the dermis in areas exposed to UV light can be differentiated from age-related dermal thinning on light microscopy. Skin damaged by UV light presents with solar elastosis in damaged areas, whereas age-related damage is characterized by thinning of the dermis. Chronic scurvy may also contribute to pressure ulcer development, because the abnormally fragile blood vessels clot or collapse at pressures well below those necessary to create a stage I lesion in normally nourished patients. It is true that pressure on the skin over a bony prominence is required to create a pressure ulcer, but we believe that such wounds occur earlier in patients suffering from a collagen deficit due to a vitamin C deficiency. In our experience, stage III and IV pressure wounds are typically preceded by chronic vitamin C deficiency. We have further observed that chronic vitamin C deficiency

causes an accumulating deterioration of total body collagen. The physical signs are most easily observed as thinning of the dermis, increased capillary fragility resulting in purpuric lesions, and skin tears.

Treatment of vitamin C deficiency should be initiated as soon as manifestations are first observed; we advocate aggressive replacement to prevent skin breakdown. Although a formal study has not been done to measure the impact of aggressive supplementation on the incidence of skin breakdown, we have had a small number of at-risk patients who were placed on our "cutaneous support supplement profile" and we believe that it has been beneficial. We have also observed rapid healing of skin tears in response to vitamin C replacement. However, controlled studies need to be done to objectively demonstrate these outcomes.

For the patient with chronic scurvy and a wound, high doses of vitamin C are rarely needed; we recommend an initial dose of 500 mg given twice daily. It remains controversial whether vitamin C should be administered in a tablet or liquid form; we believe that liquid supplements provide better absorption. Nevertheless, additional research needs to be done to identify factors affecting absorption, such as age, genetic makeup, form of supplement (tablet vs liquid), and different esters of vitamin C.

Fat-Soluble Vitamins

Vitamins A, E, and K play a role in skin and bone metabolism.[23] Vitamin D_3 has multiple functions and will be addressed separately. Deficiencies of fat-soluble vitamins are less common than water-soluble forms because these vitamins are stored and are not as rapidly depleted. The only potential physical indicator of vitamin A, E, and K deficiency is a reddish, scaly, pruritic skin lesion[24]; in many cases, there are no physical findings of deficiency. There is no evidence at present that any megadoses of fat-soluble vitamins are needed or beneficial. Thus, the present practice is to give the RDA-recommended dose once daily.

Vitamin D_3 has captured the interest of nutritionists over the past decade. It is now recognized that vitamin D has multiple functions over and above its role in calcium absorption and bone health, including enhanced physical strength and immunocompetence and a protective role against a number of cancers.[25,26] In addition, Vitamin D_3 can activate more than 2000 genes,[27] and genome control of cell proliferation and differentiation is critical to wound healing. Calcitriol is a phospholipid that crosses the cell membrane and enters the nucleus, where it initiates gene expression that affects immune function as well as cell differentiation.[28,29] For example, after wounding, vitamin D increases the expression of genes in the keratinocyte that code for antimicrobial receptors and the antimicrobial peptide cathelicidin. This molecule assists in the eradication of infectious microbes that are ever present on the surface of open wounds.[30-33]

Vitamin D can be conceptualized as a hormone rather than as a vitamin because it is synthesized in the skin with exposure to sunlight in addition to being consumed in the diet.[33] Pressure ulcer patients' multiple morbidities typically preclude adequate exposure to sun light; thus, adequate dietary intake is essential for these patients. Since the final step in the conversion of vitamin D_2 to vitamin D_3, calcitriol, is carried out in the kidney, renal health is essential to maintenance of adequate vitamin D_3 levels.[28,29,33,34]

We recommend that serum vitamin D_3 levels be checked at the time of initial assessment and monthly thereafter; normal levels are 32 to 100 ng/mL. If the serum level is subnormal, we recommend administering 2000 IU of vitamin D_3 by mouth twice daily. Once the levels normalize, we typically reduce the dose to 2000 IU daily.

Essential Fatty Acid Deficits

All cell membranes have a double wall made of proteins. Between the outer and inner layers is a space that is occupied by EFAs omega 3 and omega 6. Limited evidence suggests that EFAs may play a role in wound healing, but further study is needed. Physiologic stress causes conversion of the EFAs to prostaglandins, leukotrienes, and thromboxanes. If the primary EFA is omega 3, prostaglandin E1, leukotriene 5 series, and prostanoid TxA3 are produced and the end result is reduced inflammation, vasodilation, and anticoagulation.[35] In contrast, if the primary EFA is omega 6, prostaglandin E 2, leukotriene 4 series, and the prostanoid TxA2 are produced and the end result is increased inflammation, vasoconstriction, and enhanced platelet aggregation.

Since these fatty acids cannot be synthesized by humans, other mammals, fish, or birds, they must be obtained through the diet. Essential fatty acids are produced by photosynthesis in plants, and these plants are in the food chain of both mammals and fish. The ratio of omega 3 to omega 6 is directly related to their percentage in the diet.[36] For example, a diet high in red meat increases the levels of omega 6 whereas a diet high in fish will increase the levels of omega 3. This is because algae present in water produce omega 3 whereas land-based plants produce omega 6; 40% of corn oil is omega 6.[35]

Essential fatty acid deficiencies lead to grossly visible flaking of the skin, which is usually first seen on the lower extremity along the anterior shin. Rubbing the epidermis will release dry skin cells that resemble large snowflakes as they settle on the sheets.[37]

The recommended treatment for EFA deficiencies is administration of concentrated fish oil, specifically a 1000-mg gel capsule twice daily. We recommend cautious selection of the specific product in order to avoid products contaminated with mercury and pesticides. The best source is the North Sea, because this area is not frequented by agribusiness, which uses pesticides, or by manufacturing, which uses mercury. Alternatively, fish caught 25 km offshore are

safe to eat. The clinician should also ensure that the fish oil has no fishy odor or after taste, because these are indicators that the oil is rancid and should be exchanged or discarded. Finally, oxidized fish oil is not a good choice because it must be metabolized and detoxified and this process depletes antioxidants and other substrates.[38]

Zinc

Zinc is the most abundant trace mineral in the human body. It plays a pivotal role in approximately 300 enzyme reactions and its role in wound healing has been well-established.[39,40] For example, matrix metalloproteinases are essential to autolytic debridement of necrotic tissue and establishment of a clean wound bed, and all matrix metalloproteinases are zinc-dependent. Moreover, the term *zinc* is the "metallo" in *metall*oproteinase. In addition, zinc is a powerful antioxidant. Both zinc and vitamin C appear to be important micronutrients for wound healing.

Cutaneous manifestations of zinc deficiency are limited. Patients with zinc deficiency caused by a genetic malabsorption syndrome develop a condition called acrodermatitis enteropathica, which is manifest by an erythematous flaky rash along the lateral eye brow, nasolabial fold, and chin (Figure 6). However, these findings are not common in pressure ulcer patients.

A Zinc Tally Test is a functional test that is considered the most accurate test for measuring zinc deficiency. The individual is asked to retain 2 mL of a 0.1% solution of zinc sulfate in his or her mouth for 1 minute. Individuals who are not zinc deficient typically report an unpleasant metallic taste within about 30 seconds. We recommend substitution of over-the-counter remedies containing zinc for the 0.1% solution. This test has been shown to be equivalent to the standard zinc taste test. A normal serum zinc level is 60 to 130 μg/dL.[21,41]

For patients with chronic zinc deficiencies, we recommend treatment with oral zinc sulfate 220 mg administered twice daily. When serum levels reach 100 to 150 μg/mL, the dose should be reduced to 220 mg/day. While establishment of normal serum levels can be accomplished in a short period of time, structural defects related to the deficiency may persist for some time. For example, in patients with severe osteoporosis, establishment of a normal vitamin D level does not mean that the bony structures have normalized. Similarly, taste abnormalities associated with zinc deficiency may persist despite successful treatment.

The authors have noted that despite current recommendations to limit supplementation to 6 weeks or less, oral administration of zinc sulfate has not been associated with anemia. Nevertheless, patients receiving long-term zinc replacement should be monitored for anemia and for copper levels, since high levels of zinc may interfere with copper absorption. In our experience, long-term replacement is safe and unlikely to cause problems with copper absorption or iron-carrying capacity or anemia.

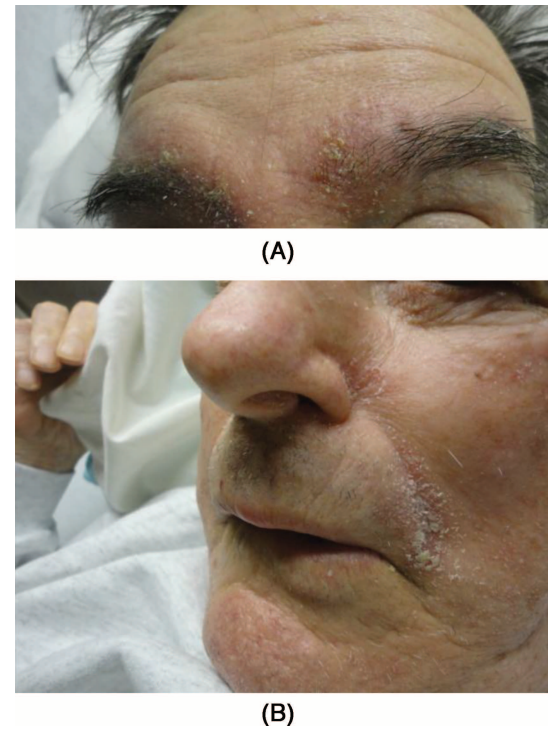


FIGURE 6. Zinc rash is a seborrheic-like reddish, flakey condition best seen along the lateral eyebrow (A) and the nasal labial folds (B).

Glucosamine/Chondroitin

Glucosamine and chondroitin sulfate are key nutrients in the formation and maintenance of granulation tissue, also known as the extra cellular matrix (ECM); they are commercially advertised for joint health.[42] Glycosaminoglycans (GAG) are one component of granulation tissue and are also known as GAG molecules; specific elements of GAG molecules are chondroitin sulfate, heparin sulfate, and keratin. Glycosaminoglycan molecules attract sodium and water, which contribute to the somewhat spongy characteristic of granulation tissue; GAG molecules also serve to provide adhesion for the various elements that make up the ECM (elastin, fibronectin, laminin, and collagen). Elements within the ECM also secrete and maintain some of the growth factors required for wound healing.[42,43] There are no diagnostic studies for GAG deficiency, although some clinicians presume a GAG deficiency when severe vitamin C deficiencies are present as discussed earlier in this article. Prolonged tenting of the skin in a well-hydrated patient may suggest GAG deficiency, but studies are required to substantiate this hypothesis (Figure 7). Glucosamine supplements are available without prescription. Many contain a combination of glucosamine/chondroitin and methylsulfonylmethane, which is an excellent source of sulfur, a major component of ECM. We recommend a common over-the-counter dose of approximately 600 mg of glucosamine, 400 mg of chondroitin, and 250 mg of methylsulfonylmethane given twice daily.

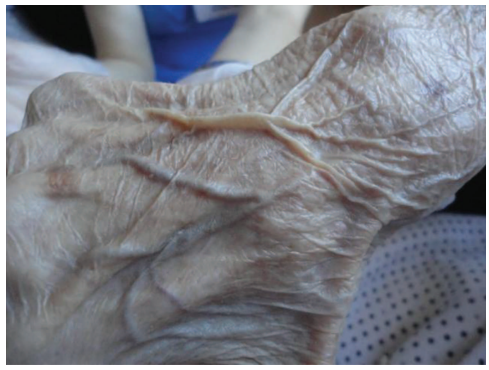


FIGURE 7. A deficiency in glucosamine and other intracellular constituents responsible for local hydration results in prolong tenting of the skin despite normal hydration on physical examination.

■ Discussion

Thousands of years ago, accurate observations made by Egyptians and Greeks set standards in wound care, which have stood the test of time. These include the following: "With good nutrition a clean wound will probably heal," which is attributed to Hippocrates c. 400 bc. Yet despite increasing evidence that micronutrients play an important role in wound healing, little emphasis has been given to the diagnosis and treatment of micronutrient deficiencies. All vitamins and trace minerals play some role in health and the recovery of wellness. Regarding wound healing, there are essential micronutrients that, if not available, will retard recovery. Fortunately, the most important deficiencies result in oral and cutaneous changes that are observable on physical examination.

■ Conclusion

Current evidence indicates that micronutrients play an important role in wound healing, and that nutritional management should include attention to micronutrient deficiencies. Since many deficiencies present initially as cutaneous changes, bedside assessment of the wound patient should include inspection for indicators of these deficiencies. When deficiencies are observed, serum studies should be done, if available, to confirm the deficiency and treatment promptly initiated.

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