

wound healing perspectives®

A CLINICAL PATHWAY TO SUCCESS

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➔ ATYPICAL WOUNDS

A PUBLICATION OF NATIONAL HEALING CORPORATION®

Equipped to heal patients

Although the types of wounds treated by National Healing's Wound Healing Centers are frequently the result of venous insufficiency, peripheral arterial disease, diabetes mellitus, and chronic obesity, there are a host of atypical wounds that our Wound Healing Centers are best suited to treat.

Atypical wounds are hard to diagnose due to their infrequency and the way they mimic other conditions. Wound Healing Centers collectively see more of these types of wounds than other settings. Wound clinicians have extensive experience in early diagnosis, intervention, and coordination with appropriate specialists to treat these difficult patients.

This issue of *Wound Healing Perspectives* reviews recent literature on *scleroderma* and pyoderma, which are caused by inflammation, and calciphylaxis and leg ulcers from sickle cell disease, which are metabolic and genetic wounds. A future issue will address wounds caused by malignancy or external factors.

We are committed to helping you recognize and heal patients with atypical wounds as well as more common chronic wounds.

Sincerely,



Katy Rowland
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Scleroderma: an overview

According to the Scleroderma Foundation, *scleroderma*, or systemic sclerosis (SSc), is a chronic connective tissue disease generally classified as one of the autoimmune rheumatic diseases. It is characterized by uncontrolled fibrosis and degenerative changes in the skin, blood vessels, synovium, and skeletal muscle.

The word "*scleroderma*" comes from two Greek words: "sclero" meaning hard, and "derma" meaning skin. Hardening of the skin is one of the most visible manifestations of the disease. The disease has been called "progressive systemic sclerosis," but the use of that term has been discouraged since it has been found that *scleroderma* is not necessarily progressive. The disease may take several forms. There is also much variability among patients.

According to the Scleroderma Foundation, approximately 300,000 persons with *scleroderma* in the United States, and approximately three to four times more women than men develop the disease. *Scleroderma* can develop and is found in every age



APPROXIMATELY THREE TO FOUR TIMES MORE WOMEN THAN MEN DEVELOP SCLERODERMA.

group from infants to the elderly, but according to the Scleroderma Foundation, its onset is most frequent between the ages of 25 to 55.

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Diagnostic criteria for scleroderma



American College of Rheumatology Diagnostic Criteria for *scleroderma*

Major criterion:

- Diffuse or proximal sclerosis

Minor criteria:

- Digital pitting scars or loss of substance from the finger pad
- Bibasilar pulmonary fibrosis
- Sclerodactyly (sclerosis affecting only the fingers or toes) ■

While *scleroderma* is rare, it is nonetheless important for caregivers to have a working knowledge of the disease in order to identify the symptoms and to intervene as needed. Early intervention and referral can help improve the patient's quality of life.

Portions of the following overview of *scleroderma* were excerpted from *The Nurse Practitioner's* July 2004 article [Early identification key to *scleroderma* treatment](#).

Scleroderma subtypes

The first *scleroderma* subtype, *limited cutaneous scleroderma*, is also known as CREST syndrome, which stands for calcinosis, Raynaud's phenomenon, esophageal motility disorders, sclerodactyly, and telangiectasia. Two of five of these symptoms must be present to make a diagnosis of CREST. These patients often exhibit a slow disease progression. However, there is a 30% to 40% risk of developing pulmonary hypertension and a 10% risk of developing interstitial lung disease. CREST can be present in patients with autoimmune disorders as well as *scleroderma*.

The second *scleroderma* subtype, *diffuse cutaneous scleroderma*, usually progresses more rapidly and has a 30% to 40% risk of interstitial

lung disease, a 30% risk of serious renal disease, and increased risk of myocardial fibrosis. The proximal limbs, neck, upper chest, back, and abdomen are affected. As thickening worsens, loss of hand function and decreased range of motion occurs secondary to skin tightness as opposed to joint involvement. Pulmonary complications occur in two-thirds of patients with *scleroderma*, making this the leading cause of *scleroderma*-related deaths.

Patients may present with joint pain, swelling, and puffiness of the hands, which may occur a year prior to the development of Raynaud's phenomenon. Within five years of *diffuse cutaneous scleroderma* onset, fatigue, weight loss, malaise, and rapidly tightening skin, especially on the fingers, may occur. Patients may not recognize the digit thickening initially but may notice facial changes such as a mask-like appearance, a beak-like nose, and tightening and wrinkling of the skin around the mouth with increased difficulty opening it, also known as *microstomia*. With skin thickening and adherence to the subcutaneous tissue, the skin appears shiny and taut. Fibrosis of the dermis leads to hair loss, dryness, and loss of sweat glands. In addition, the affected person is

susceptible to injury, ulceration, and decreased healing.

Localized scleroderma

Localized *scleroderma* is associated with little or no internal disease. The three types are *localized morphea*, *generalized morphea*, and *linear scleroderma*. *Localized morphea* appears more commonly on the trunk than the extremities. It appears as circumscribed plaques with induration that become smooth and hard, often with hypopigmented centers. One or more lesions are present that soften and leave residual patches. Edema may occur prior to induration by weeks and may or may not be associated with pain and erythema. In contrast, *generalized morphea* involves multiple, large plaques with induration. In *linear scleroderma*, which is more common in children, the skin becomes firm, smooth, and rigid with hypopigmentation and an approximated line with lesions in a unilateral distribution along the arm, leg, or the frontal scalp. These lesions can impair the person's mobility and can affect underlying bones, muscles, and structures, causing contractures and difficulty with activities of daily living.

Pathophysiology

Although the cause of *scleroderma* is unknown,



Scleroderma: an overview *(continued from page 1)*

various theories exist. One theory is that vascular injury to arteries, veins, and capillaries in the skin, heart, lungs, and gastrointestinal tract causes decreased circulation and creates an influx of inflammatory cells. The microvascular bed in the skin and these sites is diminished and results in ischemia. Early lesions show plasma cell lymphocytic infiltrates, capillary destruction, and endothelial cell proliferation.

Another theory for the development of *scleroderma* involves an immune response to a toxin such as silica, epoxy resin, and organic solvents, which results in persistent fibrosis secondary to impaired collagen regulation. Cells, macrophages, endothelial cells, cytokines, and growth factors interact to stimulate fibrosis. Specific solvents and precise conditions surrounding the patient's situation must be further studied.

Diagnosis

Because of the risk of

complications of progressive organ damage and impact on the quality of life, accurate diagnosis and intervention are essential. Clinical and medical histories are the most useful diagnostic tools. A complete history should include characteristics of presenting symptoms, onset, frequency, duration, and symptom progression, as well as personal and family medical history, social history, medications, allergies, diet, activity, and occupational exposures.

The next step involves determining if the patient has *limited cutaneous* or *diffuse cutaneous scleroderma* and the extent of organ involvement. The American College of Rheumatology's diagnostic criteria for scleroderma include having the major criterion of proximal or diffuse sclerosis or at least two of the minor criteria, which are sclerodactyly (sclerosis affecting the fingers or toes only), digital pitting scars or the loss of the

digital pads, and bibasilar pulmonary fibrosis.

Treatment and assessment

Raynaud's phenomenon is treated with cessation of smoking, avoidance of cold exposure, use of warm clothing and gloves, avoidance of vasoconstrictive substances (clonidine, sympathomimetics, cocaine, ergotamines), and various pharmacologic agents aimed at reversing digital vasospasm. The dihydropyridine calcium channel blockers (amlodipine and nifedipine) are first-line agents for the treatment of *scleroderma*-associated Raynaud's phenomenon. Sustained-release preparations are preferred, and the dose should be adjusted to reduce the severity and frequency of attacks. Patients should be monitored for dose-limiting side effects such as worsening of reflux symptoms, lower extremity edema, headache, flushing, and hypotension (Freda et al, 2004). ■

Guidelines of care for scleroderma & sclerodermoid disorders

According to the American Academy of Dermatology Association, patients who see a physician because of *scleroderma* may have a widely heterogeneous group of diseases that can be extremely disabling. Because of the serious nature of SSc and some other syndromes involving *cutaneous sclerosis*, even patients with minimal *morphea* are concerned and need reassurance. The physician's main role is proper diagnosis and general management. It is important for the patient to understand the natural course of the disease to avoid pursuing inappropriate therapy. If systemic disease is suspected, consultation and management with other specialists may be appropriate. ■

Pyoderma gangrenosum

Pyoderma gangrenosum (PG) is an uncommon ulcerative cutaneous condition of uncertain etiology. It is associated with systemic diseases in at least 50% of patients who are affected. **The diagnosis of PG is made by excluding other causes of similar appearing cutaneous ulcerations.** Pathergy, or ulcerations of PG, may occur after trauma or injury to the skin in 30% of patients (Jackson M J, 2003).

There are two variants of PG. The first is the classic ulceration usually observed on the legs. The second, a more superfi-

cial variant known as atypical PG, occurs primarily on the hands.

It is unknown what causes PG, but according to Jackson (2003), dysregulation of the immune system is believed to be involved. PG occurs in the US in about 1 in every 100,000 people each year. As with many diseases, PG seems to occur more predominantly among people in the 4th and 5th decades of life.

According to Jackson (2003), PG patients usually describe the initial lesion as a bite-like reaction, with a small, red

papule or pustule changing into a larger ulcerative lesion.

Typically, pain is the predominant complaint among patients, accompanied by malaise and arthralgias. Systemic illnesses are present in 50% of patients with PG.

No specific therapy has been found to be uniformly effective for patients with PG. In patients with an associated underlying disease, the effective therapy of the associated condition may be associated with a control of the cutaneous process as well (Jackson 2003). ■

Characteristics of peristomal pyoderma gangrenosum

Clinical description:

Painful pink-purple papules develop into ulcers with violaceous undermined borders; multiple fistulous tracts; may cause leakage of stoma bag; inflammation necrosis will be present.

Histopathology:

Neutrophilic collections with granulation tissue; mixed dermal inflammatory infiltrate (neutrophils, lymphocytes, histiocytes); biopsy findings not conclusive for pyoderma.

Associated

Systemic Diseases:

Inflammatory bowel disease, abdominal malignancies, monoclonal gammopathy, connective tissue disease. ■

SOURCE: MANAGINE PERISTOMAL PYODERMA GANGRENOSUM. ADVANCES IN SKIN & WOUND CARE, (14)6, 326-327.

Managing peristomal pyoderma gangrenosum and a pouching system

Cairns BA, et al (1994) reported that pyoderma gangrenosum (PG) is a debilitating skin disease most often associated with inflammatory bowel disease and is a reportedly rare cause of peristomal ulceration. The lesions of PG rapidly evolve from small, erythematous pustules to deep, painful, pyogenic ulcers within hours to days of onset. Although the behavior and the appearance of the lesions of peristomal PG are diagnostic, a lack of familiarity with PG often leads to misdiagnosis and inappropriate therapy. Once the wound has been

accurately diagnosed, however, there are appropriate and effective methods for managing the wound and the pouching system. According to Thomas C (2001), the following four steps should be followed for effective management:

- First, cleanse the wound with normal saline solution. Then apply the prescribed anti-inflammatory cream to the wound base. Next, apply stomahesive powder to the exposed wound to create a dry surface for pouch adhesion.
- Second, apply a two-inch Eakin wafer over

the stomahesive powder and wound after customizing the center of the ring to the size of the stoma. Apply stomahesive paste around the stoma to prevent leakage.

- Third, apply the wafer over the Eakin ring and stomahesive paste after customizing the center of the ring to the size of the stoma.
- Fourth, snap the drainable pouch in place. Apply the clip to the bottom of the pouch. Empty every four hours or as needed. ■

Calciphylaxis: Diagnosis and treatment

Calciphylaxis is a rare, often fatal condition, characterized by progressive cutaneous necrosis that frequently occurs in patients with end-stage renal disease. Many eliciting factors have been suggested as its cause, but the most commonly linked phenomenon is the development of secondary hyperparathyroidism. This secondary hyperparathyroidism leads to an elevated calcium-phosphate product and development

of penis, and *proximal*, which can occur on the trunk, abdomen, buttocks, and proximal extremities (Trent et al, 2001). Patients presenting with distal lesions have a mortality rate of 42%. Patients with proximal lesions have a mortality rate of 72%.

The ulcers of *calciphylaxis* are usually bilateral and symmetric and may be fairly deep, extending into muscle. According to

calcium-phosphate product, an elevated intact parathyroid hormone level, radiographic evidence, and confirmatory histology would substantiate the diagnosis of *calciphylaxis*. (Trent et al, 2001).

Treatment types for *calciphylaxis* include both medical and surgical therapies. Medical therapies consist of phosphate binders, low-phosphate diet,

HYPERBARIC OXYGEN AND CIMETIDINE HAVE BEEN EFFECTIVE TREATMENTS FOR CALCIPHYLAXIS IN CERTAIN CASES (TRENT ET AL, 2001).

of vascular, cutaneous, and subcutaneous calcification, resulting in tissue death (Trent et al, 2001).

Calciphylaxis is most often seen following the start of dialysis—approximately 1% of patients with chronic renal failure present with the disorder, while 4.1% of patients receiving hemodialysis are affected (Kang et al, 2000 and Budisavjevic et al, 1996). There is an estimated 5-year survival rate of less than 50% in patients who develop *calciphylaxis*. Sepsis from infected skin wounds is a major cause of morbidity and mortality for these patients (Trent et al, 2001).

Two clinical variants of *calciphylaxis* exist: *distal*, which appears on the posterolateral calves, fingers, toes, and glans

of penis, and *proximal*, which can occur on the trunk, abdomen, buttocks, and proximal extremities (Trent et al, 2001). Patients presenting with distal lesions have a mortality rate of 42%. Patients with proximal lesions have a mortality rate of 72%.

Gilson et al (1999), vesicles often appear at the periphery of the ulcers. Trent et al. reported that because of the loss of the cutaneous barrier and underlying immune deficiency secondary to chronic renal failure and/or diabetes mellitus, patients with *calciphylaxis* are predisposed to infection, subsequent sepsis, and death.

Diagnosis of *calciphylaxis* can usually be made based on the patient's clinical condition. For example, *calciphylaxis* should be suspected for a woman with a history of renal failure secondary to diabetes mellitus who develops necrotic plaques and ulcers. A laboratory evaluation that shows the presence of elevated calcium and/or phosphate level, or an elevated

reduced calcium in the dialysate, antibiotics, and diphosphonates.

Hyperbaric oxygen and cimetidine have been used in certain cases with good results. Anticoagulation has been helpful in patients with protein C and protein S deficiencies (Trent et al, 2001).

Surgical treatment can include wound debridement, amputation, renal transplantation, and parathyroidectomy. ■

Risk factors for calciphylaxis

- Female
- Alkalinization of dialysis
- Immunosuppression
- Protein C deficiency
- Protein S deficiency
- Diabetes mellitus
- Hyperlipidemia
- Antithrombin III deficiency
- Smoking
- Elevated calcium-phosphate product ■

SOURCE: CALCIPHYLAXIS: DIAGNOSIS AND TREATMENT. *ADVANCES IN SKIN & WOUND CARE*, (14)6, 310.

Leg ulcers in sickle cell disease

Treatment options for sickle cell ulcers



Sickle cell disease (SCD) is an inherited blood disorder that causes the bone marrow to produce red blood cells with defective hemoglobin, hemoglobin S (sickled hemoglobin). Leg ulcers are the most common cutaneous manifestation of SCD. These ulcers are characterized by an indolent, intractable course, typically healing up to 16 times slower than venous ulcers. A patient who experiences his or her first sickle cell ulcer is likely to ulcerate again: approximately 97% of healed sickle cell ulcers will recur in less than 1 year. Due to the recalcitrant nature of these ulcers, patients may experience significant disfigurement, social isolation, and loss of income (Trent et al, 2004).

antithrombin II deficiency; possessing certain leukocyte antigens (HLA); having thrombocytosis, and living in certain geographic areas (Trent et al, 2004). History of a sickle cell leg ulcer carries a 23-times increased risk of developing future ulcerations; having one active ulcer carries a 146-fold increased risk (Eckman JR 1996). Prognosis improves in these patients, however, with the presence of sickle/beta-thalassemia and sickle C hemoglobin (Koshy et al, 1989).

The pathogenesis of leg ulcers in SCD is unclear, but one theory is that vascular obstruction occurs due to rigid and inflexible sickled red blood cells becoming lodged within smaller blood vessels, followed by tissue

endothelium, slow blood flow, and additional occlusion of the vessels. Furthermore, granulocytes interact with the sickled cells, leading to the release of injurious cytokines, which cause added tissue injury (Trent et al, 2004).

Other factors have been implicated in ulcer formation in SCD. One example is trauma, which is believed to cause sickle cell ulcers by stimulating the sickling of red blood cells and causing the previously discussed ischemia. Anemia may also cause ischemia and tissue necrosis. Further, venous incompetence may contribute to the development of sickle cell ulcers.

Common sites for sickle cell ulcers include the

A PATIENT WHO EXPERIENCES HIS OR HER FIRST SICKLE CELL ULCER IS LIKELY TO ULCERATE AGAIN: APPROXIMATELY 97% OF HEALED SICKLE CELL ULCERS WILL RECUR IN LESS THAN 1 YEAR. (TRENT ET AL, 2004).

Khouri et al (1991) reported that the incidence of leg ulcers in patients with SCD ranges from 25.7% to 75%. Risk factors for the development of ulcers include being older than 20 years; being male; having a lower level of fetal hemoglobin and a hemoglobin level less than 6 g/dL; having

necrosis as a result of ischemia.

In addition, injury to red blood cells leads to the upregulation of integrins, which act as adhesion molecules on endothelial cells. This upregulation promotes platelet aggregation, adherence of sickled cells due to en-

anterior tibial area, dorsum of the foot, Achilles tendon area, and ankles, with the medial malleolus being more affected than the lateral malleolus. Sickle cell ulcers classically appear as round, punched-out ulcers with raised margins, deep bases, and necrotic slough. Surrounding

Topical treatments

- Triple antibiotic ointment
- Arginine-glycine-aspartate

Dressings

- Unna's boot
- DuoDERM
- Solcoseryl
- Collastat
- Omiderm ■

SOURCE: LEG ULCERS IN SICKLE CELL DISEASE. *ADVANCES IN SKIN & WOUND CARE*, (17)8, 414.

Leg ulcers in sickle cell disease (continued from page 6)

brown hyper-pigmentation and scaling may also be present. Multiple ulcers or scarring from healed ulcers may be found in the vicinity. Patients usually complain of extreme tenderness or pain at the site of the ulcer. This is because sickled cells lead to tissue death, which is painful. In essence, sickle cell ulcers represent a type of ischemic ulcer (Trent et al, 2004).

A variety of dressings have been used to heal sickle cell ulcers. The use of Unna's boots, zinc-oxide impregnated dressings, controlled edema in patients with sickle cell

debridement and the use of myocutaneous flaps and split-thickness skin grafts. Debridement with dressings or debriding ointments enables the removal of necrotic slough (Eckman et al, 1996). Autologous split-thickness skin grafts or pinch grafts are advocated for the use of recalcitrant ulcers. Most grafts fail, however, because of the inherent circulatory difficulties and vascular insufficiency in the areas of these ulcers. To increase the chance of skin survival after transplantation, the use of myocutaneous flaps is encouraged because they carry their own blood

placement to promote flap survival (Trent et al, 2004).

According to Trent et al, (2004), Gordon and Bui reported a patient with long-standing sickle cell ulcers who healed within 6 weeks with the use of Apligraf®, a bilayered skin equivalent manufactured from neonatal foreskin keratinocytes and fibroblasts. The patient experienced no recurrence after 33 weeks. Others have reported less success using cultured autologous epidermal grafts. Other treatment options are listed in the columns on page six and seven. ■

LEG ULCERS ARE THE MOST COMMON CUTANEOUS MANIFESTATIONS OF SICKLE CELL DISEASE. (TRENT ET AL, 2004)

ulcers and led to subsequently faster healing (Trent et al, 2004). Surgical interventions for sickle cell ulcers include

supply to the area of ulceration. Some authors have used anticoagulation in conjunction with myocutaneous flap

Treatment options for sickle cell ulcers

(continued)

Surgical interventions

- Debridement
- Split-thickness skin grafts
- Myocutaneous flaps
- Apligraf®

Systemic medications

- Zinc sulfate
- Pentoxifylline
- Antithrombin
- Hydroxyurea
- L-carnitine
- Arginine butyrate
- Transfusions
- Erythropoietin ■

SOURCE: LEG ULCERS IN SICKLE CELL DISEASE. *ADVANCES IN SKIN & WOUND CARE*, (17)8, 414.

Selected bibliography

Author unknown, (1996). Guidelines of care for scleroderma and sclerodermoid disorders. *Journal of American Academy of Dermatology*, 35, 609-14. • Hausteil UF. (2002). Systemic sclerosis - scleroderma. *Dermatology Online Journal*, 8(1), 3. • Buyon JP, Giesser BS, Nelson JL. (2000). Deciphering autoimmune disease in women. *Contemporary OB/GYN*, 8, 76-86. • Eckman JR (1996). Leg ulcers in sickle cell disease. *Hematol Oncol Clin North Am*, (10): 1333-44. • Freda B.J, Chatterjee S. (2004). Systemic scleroderma. Retrieved May 11, 2005, from <http://www.clevelandclinicmeded.com/diseasemanagement/rheumatology/scleroderma/scleroderma.htm>. • Gordon S, Bui A. (2003). Human skin equivalent in the treatment of chronic leg ulcers in sickle cell disease patients. *Journal of the American Podiatric Medical Association*, 93: 240-1. • Gunduz OH, Fertig N, Lucas M, et al. (2001). Systemic sclerosis with renal crisis and pulmonary hypertension. *Arthritis Rheumatology*, 4 (7), 1663-1666. • Jancic B. (2002). Advances make scleroderma manageable. *Skin & Allergy News*, 33(3):4. • Joslin N. (2004). Early identification key to scleroderma treatment. *The Nurse Practitioner*, 29(7), 24-39. • Khouri RK, Upton J. (1991). Bilateral lower limb salvage with free flaps in a patient with sickle cell ulcers. *Annals of Plastic Surgery*, 27:574-6. • Koshy M, Entsuah R, Koranda A, et al. (1989). Leg ulcers in patients with sickle cell disease. *Blood* 74:1403-8. • Nietert PJ, Silverstein MD, Silver RM. (2001). Hospital admissions, length of stay, charges, and in-hospital death among patients with systemic sclerosis. *Journal of Rheumatology*, 28(9), 2031-2037. • Russ V, White B. (2002). Systemic sclerosis: multiple therapies control morbidity; early assessment of internal organ involvement is needed for timely treatment. *The J Musculo Med*, 19(3), 110-121. • Trent J T, Kirsner R S. (2001). Calciphylaxis: Diagnosis and treatment. *Advances in Skin & Wound Care* (14)6, 309-312. • Trent J T, Kirsner R S. (2004). Leg Ulcers in Sickle Cell Disease. *Advances in Skin & Wound Care* (17)8, 410-16.

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Working with a Wound Healing Center: Surgical debridement



Surgical intervention in the treatment of chronic wounds often involves debridement. Dead tissue provides a medium for infections, initiates an inflammatory response, places a phagocytic demand on the wound, and retards wound healing. Necrotic or devitalized tissue also may mask underlying fluid collections or abscesses. Therefore, debridement of nonviable tissue is crucial to optimal wound healing, which can be impaired unless all necrotic tissue, exudates, and metabolic wastes have been

removed from the wound. (Sieggreen, 1997).

Debridement is only appropriate, however, when nonviable tissue is present, or if a patient's wound is filled with a soft, fibrinous, yellow-green tissue that will harden into eschar if left exposed to air (Sieggreen, 1997). Debridement is not appropriate for ulcers caused by pyoderma, as it causes ulcers to spread.

A qualified clinician with expertise in wound care should be consulted prior to implementing any type

of debridement treatment. With its dedicated, skilled physicians, Wound Healing Centers are uniquely equipped to perform every type of debridement. In addition, Wound Healing Centers offer follow-up education for primary care physicians and their patients to prevent recurrence. The knowledge gained by tracking outcomes consistently is applied to each treatment plan to speed healing and improve each patient's quality of life. ■

CONTACT YOUR LOCAL WOUND HEALING CENTER IF YOUR PATIENTS SHOW SIGNS OF ATYPICAL WOUNDS

- Patients who have failed traditional wound care for more than 30 days.
- Venous insufficiency patients that have not healed with traditional wound care.
- Patients who have not responded to venous therapy treatments.
- Patients who experience hard, sclerotic tissue that does not respond to usual scar management.
- Patients who experience Crohn's disease or stomach ulcers and now have wounds.
- Patients who suffer from sickle cell disease and now have wounds.
- Patients who have been on dialysis
- Patients who have a colostomy with trouble around the stoma.
- Patients who complain of severe extremity digit pain with temperature changes.



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