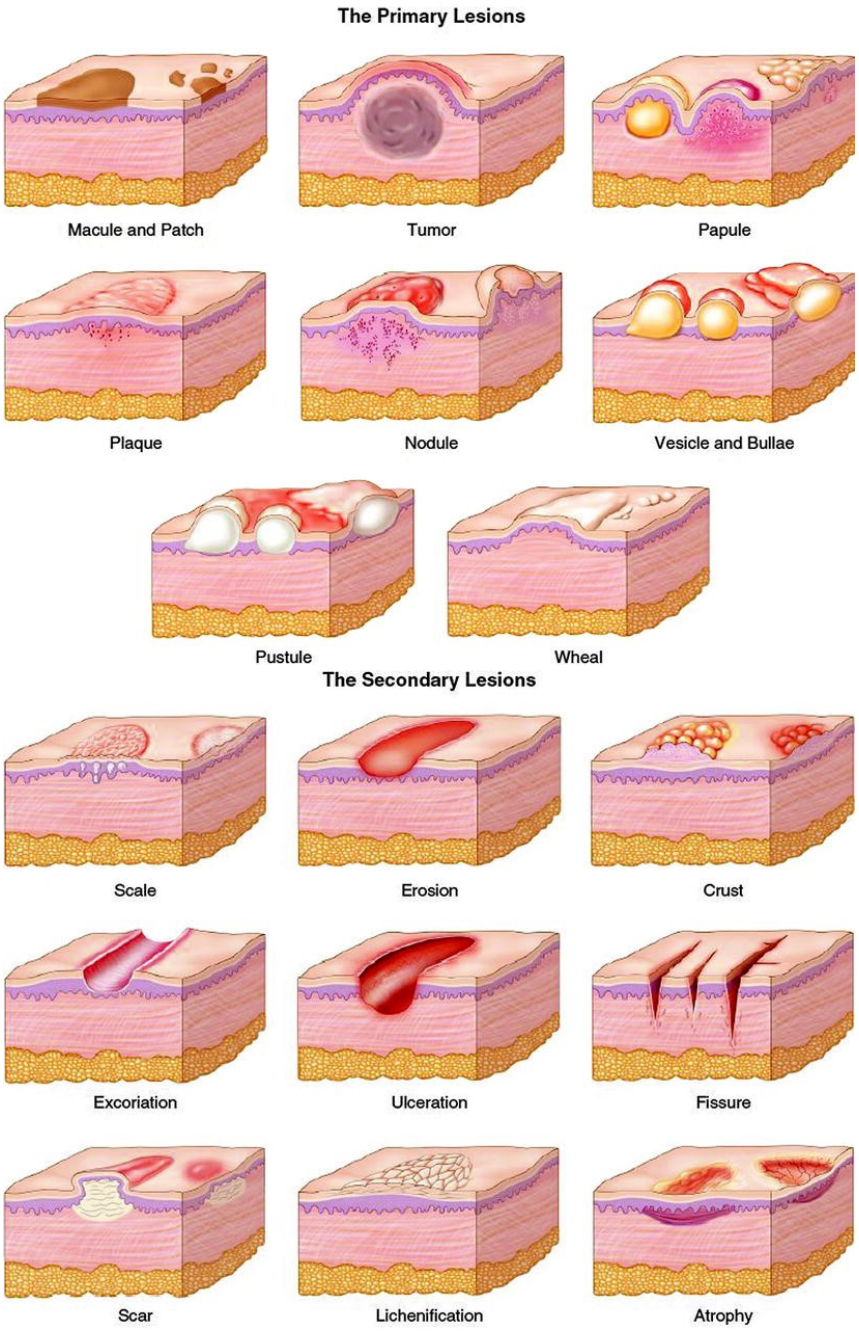
ProCare Training Manual
Chapter 10
Commonly Misdiagnosed and Recalcitrant Wounds

Occasionally the recalcitrant or problematic wound is referred to the wound healing center after months or even years of standard treatment from the patient's primary care physician. Some of these wounds are often misdiagnosed as other types of chronic wounds leading to a delay in the appropriate treatment which in turn delays healing. Some of the more common recalcitrant wounds are those directly related to radiation injury, spider bites, malignancies, vasculitis and Pyoderma Gangrenosum. There are numerous other disease specific complications that delay wound healing, but they are too numerous to expand on during this training session. You are encouraged to research the less common wound etiologies and become familiar with their presentations.

To have a better understanding of the presentation of some of the following diseases, it is important to be able to differentiate between the common symptoms. Below is a listing of some of the more common skin eruptions and the layers of skin that are involved. (Figure 10-A)

Figure 10-A



Malignancies

Chronic non-healing wounds that have shown no improvement in approximately 2-3 months should be looked at for malignancy. Malignancies and wounds are related in two ways: there can be malignant degeneration of a chronic wound into cancer or, malignancy can present as a chronic wound. Malignancy found within a chronic wound is rare, but should be considered if wounds are refractory to healing. The common signs and symptoms are:

- History of repeated trauma
- Wounds with no obvious etiology
- Wounds at unusual locations
- Asymmetric wounds
- Exuberant granulation tissue (hypergranulation)
- Rolled out edges (instead of rolled under)
- Fungating growth
- Purple-red color around the ulcer
- Ulcer in center of pigmented lesion (suspect melanoma)
- Wounds secondary to burns, trauma, radiotherapy, and diabetes are at a higher risk for malignant degeneration.

Generally speaking, malignant wounds will not heal until the malignancy is amenable to treatment with anticancer therapy. For basal or sqaumous cell cancer, complete excision is the treatment of choice.

Basal Cell Carcinoma

Basal cell carcinoma is a malignancy arising from epidermal basal cells. Typically, basal cell carcinomas present as a wound, outgrow their blood supply and erodes and subsequently forms an ulcer.

Squamous Cell Carcinoma

Unlike basal carcinomas, which have a very low metastatic potential, a squamous cell carcinoma can metastasize and grow very rapidly. The development of squamous cell carcinoma has been reported in chronic wounds secondary to burns, trauma, radiotherapy, diabetes, and a sinus tract draining from chronic osteomyelitis. It also appears that squamous cell carcinomas appear more frequently than basal cell carcinomas within a venous ulcer.

Kaposi's Sarcoma

Kaposi's Sarcoma is highly associated with HIV and herpes simplex infection. It is mainly associated with AIDS and reported to present as an ulcer. Treatment includes observation, surgical excision, radiation therapy, intralesion chemotherapy, systemic chemotherapy or using a combination of chemotherapies.

Cutaneous Lymphomas

Cutaneous lymphomas may present as various types of skin lesions and rarely as an ulcer. Ulcerative cutaneous lymphomas are associated with poor prognosis and are usually seen in the severely immunocompromised patient.

Fungating Wounds

Fungating tumors or wounds are a devastating complication resulting from complicated underlying pathology such as metastasis. It is an ulcerating malignant skin lesion that is open and most usually draining a significant amount. The wound may be a result of a primary cancer, a metastasis to the skin from a local tumor, or a tumor at a distant site. The lesion presents as a rapidly growing fungus and/or takes on a "cauliflower" like appearance that also may ulcerate and form shallow craters. Associated sinus tracts and fistula formation often create very malodorous wounds. These types of fungating wounds are most commonly associated with breast cancer but may also originate from cancer of the head, neck, kidney, lung, ovary, colon and penis. Lymphoma, leukemia and melanoma can also produce Fungating skin lesion.

Treatment of the Fungating wound is focused on three key points:

- Wound pain management
- Odor control
- Control of exudate

These wounds are typically very painful, malodorous and drain copiously. Dressing selection should include a highly absorbent, odor controlling dressing (with a charcoal base) and should be non-adherent. The less the patient has to change the dressing, the better it is for the patient and family. The goal of wound treatment for the fungating lesion is often a palliative, comfort level approach, rather than an aggressive wound healing approach. Remember, unless the tumor responds to anticancer treatment, the wound will not heal.

Infectious Factors

Many skin rashes or ulcers are indicative of an infectious process and can occur around wounds or be misinterpreted as a result of pressure, shear friction, or chemical irritation. Infections can be categorized according to infecting organism: fungus, bacterial, viral or arthropod. A few of these will be discussed.

Fungus

Candidiasis represents an epidermal infection with Candida (Yeast). The primary lesion with candidiasis is a pustule. Secondary lesions are papules which result from abraded pustules and plaque. Erythema and maceration are common with candidiasis. Lesions are typically beefy red, with satellite erythematous papules and pustules. When located in the skin folds, solid plaques of moist red lesions are commonly seen. Satellite lesions (outside the advancing edge of Candida) and pruritus (itching) are an important diagnostic feature of Candida. Often, candidiasis is misinterpreted as contact dermatitis. The distinctive difference between these two infections is that with candidiasis pustules and satellite lesions are evident and with contact dermatitis, the distribution of the rash is limited to the area that is in contact with the irritant.

Risk factors that increase the likelihood of infection with candidiasis organisms include the presence of a moist warm environment (such as skin folds), a hot humid external environment, tight underclothes, diabetes and prolonged antibiotic therapy. Diabetes predisposes the patient to develop yeast because the associated increase in the amount of glucose in the saliva, sweat and urine prevents bacteria from inhibiting yeast growth. Antibiotics predispose a patient to develop candidiasis by removing the competing organisms, which increase yeast growth.

Treatment of infections with yeast include removing as many of the predisposing factors such as tight clothing, antibiotics, hyperglycemia, etc… as possible. Powders and absorptive dressings should be used in skin folds to absorb moisture. Antifungal creams, ointments and powders are available over the counter or by prescription. Typically, yeast infections are treated for 10 days. When using antifungal creams and ointments, use sparingly as excessive product can increase the warmth and moisture level of the skin fold. Powders are preferred for excessively moist skin folds.

Bacterial

Folliculitis, impetigo and necrotizing fasciitis are frequently seen secondary infections that the wound care nurse should be familiar with. Many bacterial infections are caused by *Staphylococcus aureus* which is consistent with normal skin flora. The overgrowth of *Staphylococcus aureus* is commonly preceded by bacterial infections.

Folliculitis is a bacterial infection that involves the hair follicle. The primary lesion is a pustule that is pierced by a hair, secondary lesions are crusts and erythema. Folliculitis may be limited to the superficial hair follicle or progress deeper into the follicle. Folliculits is commonly a secondary effect of an adhesive bandage over a hairy body part, but can also be found on the scalp, extremities and skin folds.

Predisposing factors of folliculitis include diabetes, malnutrition, obesity, immunodeficiency and chronic staphylococcal infections. Standard treatment includes the use of warm compresses 3-4 times per day for mild cases. Adhesives should be avoided and the hair should be clipped rather than shaved. Oral antibiotics are often recommended for deep folliculitis.

Impetigo is most commonly seen in children with the initial onset being a pustule with little or no erythema. Lesions quickly form a yellow-tan crust. When the crust is disrupted, a superficial glistening base is seen. Ulcerations are not present because the infection is superficial and barely extends below the stratum corneum. Impetigo can occur anywhere but is most commonly seen on the face. It is important to distinguish impetigo from streptococcal infections and herpes simplex. Streptococcal infections are deeper, extending through the epidermis so that an ulcer is seen when the crust is removed and often these ulcers are surrounded by erythema. Herpes simplex begins with grouped, clear vesicles that are uniform in size, and it recurs a the same site. Treatment of impetigo includes topical or systemic antibiotics, depending on the severity.

Necrotizing Fasciitis is a rare subcutaneous tissue infection that spreads rapidly along the superficial fascial plane. The overall mortality rate from this infection is 47%; early diagnosis (within 4 days of onset) decreases the mortality rate to around 12%. Necrotizing fasciitis is characterized by widespread necrosis of the fascia and deep subcutaneous tissue with thrombosis of nutrient vessels and sloughing of overlying tissue. It usually occurs in the extremities after a minor operation or injury. Initial signs are pain, swelling at the site of the wound, chills, fever, toxemia, and rapidly spreading painful cellulitis. The skin appears normal over the cellulitis, but as the infectious process compromises blood supply, the skin becomes erythematous, edematous, and reddish-purple to patchy blue gray. Bullae form within 3-5 days which progresses to necrosis of the skin, sloughing and frank cutaneous gangrene. Diagnosis between cellulitis and necrotizing fasciitis is determined when probing the wounded area. In cellulitis, there is no easily obtainable tract along the fascial planes as there is with necrotizing fasciitis. Treatment consists of prompt,

extensive, and aggressive surgical debridement to remove all nonviable fascia and tissue. The wound must then be monitored closely for further progression of sloughing, possibly requiring additional debridements. Aggressive antibiotic support and hyperbaric oxygen treatments should be started as soon as possible.

Fournier's Gangrene is a form of necrotizing fasciitis that initially affects the male genitalia. Its differentiation from necrotizing fasciitis is only by anatomical location. Diagnosis and treatment are the same as with necrotizing fasciitis.

Viral

Stress and illness often trigger herpes simplex virus (HSV) and varicella-zoster virus (VZV). They are highly contagious infections and required prompt treatment to prevent the spread of the infection to others.

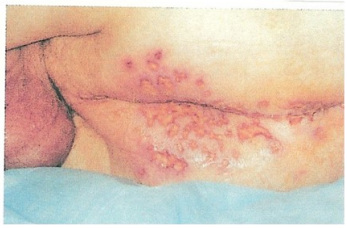
Herpes Simplex Virus infections of the epidermis can be spread when a susceptible noninfected person comes into direct contact (mucous membrane or broken skin) with a person shedding the virus. Viral shedding can occur even in the absence of symptoms, which is when most infections are transmitted. Therefore, HSV should be considered a chronic contagious process and all people should be treated as potentially contagious. Historically HSV has been classified into two classes. HSVI usually refers to oral herpes and HSVII as genital herpes. Ultimately, HSV lesions are not limited to the lips and genital areas and may occur anywhere on the skin.

HSV infections have two phases. During the primary infection phase, the virus becomes established in a nerve ganglion. Uniform grouped vesicles develop on an erythematous base, which is a key indicator of HSV. The vesicles contain large numbers of infective viral particles. Vesicles soon become pustules that erode, drain, and crust over. Primary lesions usually last 2-6 weeks and heal without scarring. As the lesion heals the virus enters the skin nerve endings and ascends through the peripheral nerves to the dorsal root ganglia where it remains in a latent stage. During times of stress, illness, fatigue, fever, compromised immune system or trauma (to name a few), a reactivation of the virus occurs triggering a recurrence of symptoms. The reactivated virus presents as vesicles on an erythematous base or ulcers. Crusts cover the eruptions within 2-4 days and are shed in approximately 8 days.

Antiviral medications are effective in treating HSV infections and are available in topical, oral and intravenous preparations. Early initiation of oral acyclovir for genital herpes decreases healing time, viral shedding and duration of pain. When caring for the wounds, it is often best to keep the ulcers dry. Although, refrigerated hydrogel sheets and Burrow's solution (aluminum acetate) soaks are soothing to the very painful ulcers. During the period of viral shedding, it is imperative that precautions are taken to prevent transmission of the virus.

Varicella-Zoster Virus (VZV) is the combination of varicella (chickenpox) and herpes zoster (shingles). It is transmitted by direct contact with vesicle fluid or airborne droplets from the infected individual. Patients with herpes zoster are less contagious than those with varicella. Herpes zoster is an infection within the epidermis that occurs along one or two adjacent dermatome distributions.

Figure 10-B and 10-C herpes zoster various distributions along the buttocks and perianal area



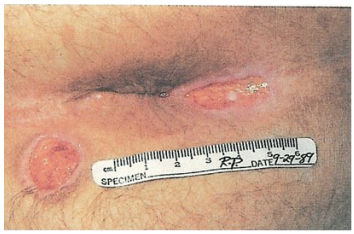
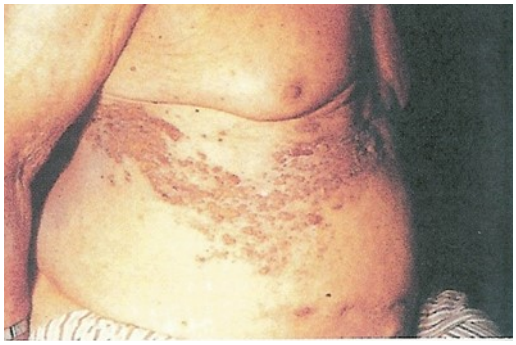


Figure 10-D Shingles along the torso



Eruptions result from a reactivation of the VZV that entered the cutaneous nerves and has remained dormant in the dorsal root ganglia after a bout with chickenpox. Reactivation can occur as a result of immunosuppression, fatigue, and emotional trauma and occurs in 15% of people.

Clinically, herpes zoster consists of a unilateral vesicular rash along one or two dermatomes. Vesicles develop in clusters and vary in size. Over the next few days, the vesicles fill with purulent fluid followed by rupture and crusting. In debilitated or the immunocompromised patients, secondary infection may follow the eruptions leading to necrosis. Treatment of VZV is very similar to that of HSV with antivirals and Burrow's soaks to act as an

astringent on the lesions. Lesions should NOT be dressed with an occlusive dressing as this will delay healing.

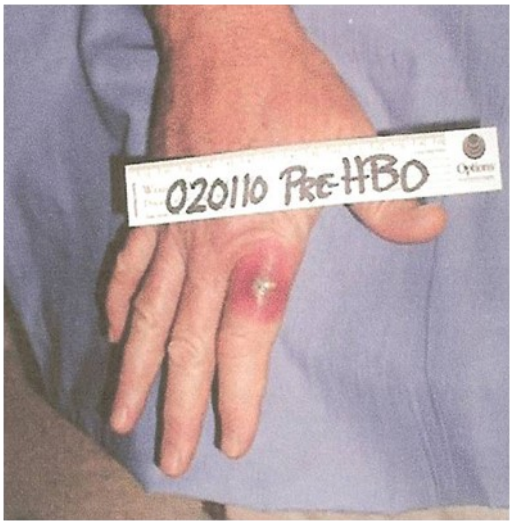
Arthropod

Brown Recluse Spider bites initially present with swelling and erythema. Because of the release of a necrolytic toxin, purpura and subsequent extensive necrosis follows. The brown recluse spider (fiddleback) is nocturnal and not known to be aggressive. These spiders will only bite when trapped or pressed against a fixed object such as skin and clothes (especially in folds). The most common body location for the brown recluse spider bites is on the extremities but can also be found on the buttocks or genitalia.

There are commonly two stages of spider bites seen with the brown recluse spider. The first is the superficial stage in which some initial discomfort is felt a few hours after the bite and then there is usually some erythema, possible minimal blister formation and pruritus. The area affected is rather small, about 1-2 cm. A small thin eschar may form, falling off in a week or two, leaving no scar. The second stage is the more publicized and less common of bites. This is a more severe wound and is thought to be the result of greater volume or deeper envenomation from the brown recluse spider. The patient may develop a generalized punctuate rash, fever, nausea and vomiting. Pain is not felt at the time of the bite but appears several hours later and can vary from a persistent, moderately severe pain to a throbbing pain. A hemorrhagic blister forms in the bite area and a "bulls eye ring" forms around the bite site over the next several hours and measures several centimeters in diameter. The center of the ring is blue, the middle ring is white and the outer ring is red. Over the next few days, the blister and the cyanotic area become necrotic and gangrenous and can extend to areas away from the center. The gangrenous area often forms an eschar that frequently extends to the muscular fascia if left untreated.

Treatment regimens are varied. You may have heard reports of the use of stun guns (high velocity electrical current) in the immediate treatment of brown recluse spider bites. Other treatment regimens include ice packs, antibiotics, pain control, tetanus toxoid, and nonocclusive dressings (to facilitate debridement). Hyperbaric oxygen treatments are also recommended for treatment of severe spider bites, especially those that have required surgical debridements, grafts and/or flaps. Patients should be observed for systemic reactions which could occur 2-3 days after the bite. Systemic reactions may include renal failure or coagulation disorders such as DIC (disseminated intravascular coagulopathy).

Figure 10-E Brown recluse spider bite

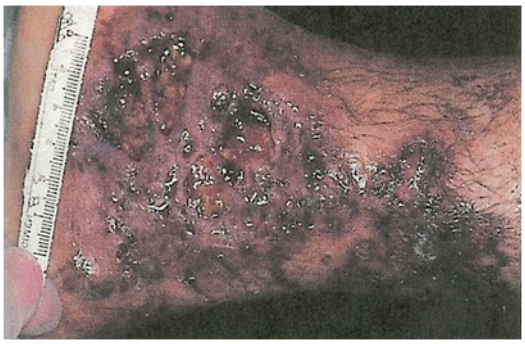


Vasculitides or Connective Tissue Disorders

Inflammatory ulcers including the various manifestations of pyoderma granulosum, rheumatoid arthritis, lupus, scleroderma and vasculitis and must be carefully assessed to avoid being misdiagnosed as a venous or arterial ulcer.

Pyoderma Granulosum (PG) is a chronic neutrophilic inflammatory disease that can cause painful ulcerative lesions. PG is usually associated with immune reactions and underlying systemic disorders, 40-50% of PG cases are idiopathic. When PG does accompany a systemic disease, it does not necessarily parallel the underlying disease. Some of the systemic diseases associated with pyoderma granulosum are: rheumatoid arthritis, inflammatory bowel disease, chronic active hepatitis, myeloma and ankylosing spondylitis. The pathophysiologic mechanism or pyoderma granulosum is unknown. By history, the presence of numerous polymorphonuclear leukocytes creates a dense infiltrate of the dermis that can extend from the superficial dermis to the subcutaneous tissues.

Figure 10-F Pyoderma Granulosum



Common characteristics of all PG lesions are that the wound margins are ragged, elevated, and violaceous (purple in color). Ulcers are exudative and extremely tender, the wound edges are often undermined. A band of erythema may extend from the wound edge, which defines the directions in which the ulcer will extend. Healing may occur along one edge of the wound while enlargement of wound occurs on the opposite edge. Ulcers heal very slowly and leave atrophic, irregular scars.

The most common sites for PG are the lower extremities, although the buttocks, abdomen, face and hands can also be affected. A diagnostic characteristic of PG is a phenomenon known as pathergy, the abnormal exaggerated inflammatory response to noxious stimuli. These lesions will often be reported to have developed after a benign bump against furniture and debridement of these wounds can often increase the wound volume. PG is difficult to diagnose and is thought of

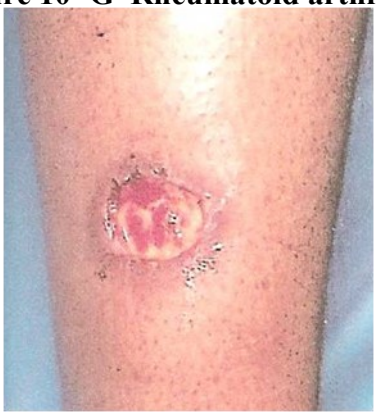
as a disease of exclusion. A thorough history and physical, including an investigation of underlying diseases, as well as wound biopsy and laboratory analysis all must be obtained to diagnose PG.

Several treatment regimens have been used to manage PG with no one particular treatment emerging as clearly effective. PG is not an infectious disease process, nor is it a disease to be managed by debridement. Systemic corticosteroids are often needed and lead to reliable results. Unfortunately, their use is limited by their significant side effects. Topical wound management should address the needs of the wound (protection, absorption or hydration). Topical vitamin A has also been shown to decrease the anti-inflammatory effects of steroid therapy. Because of the intense pain that accompanies PG, nonadhesive dressings are preferred. Debridement should only be delivered with great caution for the tendency of pathergy and is often accomplished via autolytic debridement.

Rheumatoid Arthritis (RA) is characterized by inflammation involving the membranous lining of the joint. This inflammation causes pain, stiffness, warmth, redness and swelling. RA is an autoimmune disorder signified by the invasion of inflammatory cells in the joint space causing destruction of the synovial lining. RA leg ulcers are multifactorial with vasculitis and venous insufficiency predominating. Limited ankle movement contributes to poor calf-muscle pump function and places the RA patient at increased risk for lower extremity ulcer formation.

The ulcers typically present as palpable purpura and echymosis that may progress to ulceration. The ulcers are usually shallow with well demarcated edges which are very painful and slow to heal. Due to the venous insufficiency often associated with RA, compression therapy may be indicated. Pinch grafting (local skin grafting performed by a surgeon) has been noted to be a good alternative to conservative treatment for minor leg ulcers which decreases pain and healing time. Bioengineered skin grafts or wound products such as Oasis®, Apligraf®, Dermagraft®, are also an option to provide a biological dressing to decrease pain.

Figure 10- G Rheumatoid arthritis

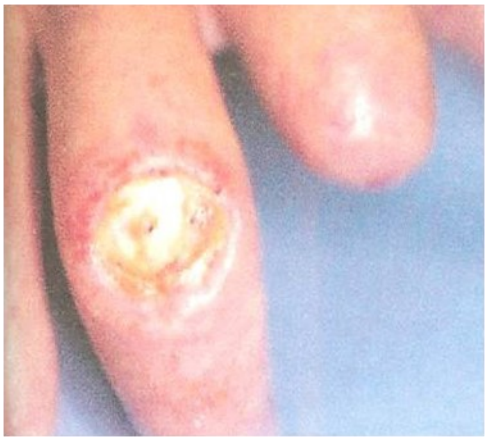


Scleroderma which is also known as systemic sclerosis, is a chronic autoimmune connective tissue disease classified as a rheumatic disease. It is characterized by uncontrolled fibrosis and degenerative changes in the skin, blood vessels, synovium, and skeletal muscles. The word "scleroderma" comes from two Greek words. "Sclero" meaning hard and "derma" meaning skin. Hardening of the skin is one of the most visible signs of this disease. In general 3-4 times more women develop scleroderma than men. Scleroderma can also present at any age.

Patients may present with joint pain, swelling, and puffiness of the hands, which can occur up to a year prior to the development of Raynaud's phenomenon (inability to tolerate cool temperatures). Other symptoms noted in later progression are esophageal motility disorders, fatigue, weight loss, malaise, and rapidly tightening skin especially on the fingers and toes. Facial changes include a "mask-like" appearance, a beak-like nose, and tightening and wrinkling of the skin around the mouth, with increased difficulty opening it. As the skin thickens, it adheres to the subcutaneous tissue causing the skin to appear shiny and taut. Fibrosis of the dermis leads to hair loss, dryness, and loss of sweat glands.

Because of the risk of complications of progressive organ damage and impact on the quality of life, accurate diagnosis and intervention are essential. Raynaud's phenomenon is treated with the cessation of smoking, avoidance of cold exposure, use of warm clothing and gloves and the avoidance of vasoconstrictive substances. The dihydropyridine calcium channel blockers (amlodipine and nifedipine) are first line agents for the treatment of scleroderma associated Raynaud's phenomenon. Topical treatment of scleroderma wounds should address the wound's need for protection, pain, moisture or absorption. Digital ulcers are difficult to reepithialize and are usually refractory to healing. Occlusive dressings may provide pain relief. Topical vitamin A has also been used with success to counteract the localized effects of systemic steroids in the wound. Aggressive sharp debridement is generally discouraged due to the delayed healing responses seen. Hyperbaric oxygen therapy has seen some improvement with the treatment of scleroderma due to angiogenesis (formation of new blood vessels), but is not a definitive cure for the chronic autoimmune disorder.

Figure 10-H Scleroderma



Lupus is also a chronic autoimmune disorder that is characterized by exacerbation and remission cycles. Lupus affects multiple organs, including skin, serosal surfaces, CNS, kidneys and the red blood cells. Circulating immune complexes and autoantibodies cause tissue damage and organ dysfunction. It is not known exactly what triggers an exacerbation, but it is theorized that it is influenced by environmental changes,

the patient's immune response and hormones. Common symptoms include the typical butterfly rash over cheeks (fascial erythema over cheeks and nose), fatigue, weight loss, fever and malaise. Potential manifestations include seizures, hemiparesis, pericarditis, pleuritis, renal failure, nausea, vomiting, abdominal pain, and arthralgias.

The incidence of lower leg ulceration in patients with Lupus is 2-8%. These ulcers can be extremely painful and are typically found over the pretibial surface and malleolar area. They are characterized by well defined margins with a purulent wound bed and varying amounts of granulation tissue. The periwound of these ulcers is often erythematous. Treatment is often challenging. Again, address the needs of the wound bed (moisture and protection), keeping in mind that these wounds are very painful, and aggressive sharp debridement may be less beneficial than autolytic or enzymatic debridement.

Vasculitis is simply the inflammation of blood vessels. Vasculitis plays a crucial role in many of the collagen vascular disorders with a key pathologic feature being inflammation and necrosis of blood vessels. Vessels of any type can be affected, so any organ or system may be involved. Vasculitic ulcers that result in skin lesions are usually a sign of a complex process and may indicate a systemic disorder. Small, medium or large vessels can be affected. Vasculitides is a broad term that refers to systemic disorders that result in vasculitis. Specific diseases include rheumatoid arthritis, systemic lupus, polyarteritis nordosa, hypersensitivity vasculitis, Wegener's granulomatosis, Sjogren's syndrome, cryoglobulinemic vasculitis, and dermatomyositis.

The general signs and symptoms of vasculitis are fever, myalgias, arthralgias, and malaise. Patients sometimes describe a vague flu-like illness. Peripheral neuropathy may also be present. The cutaneous effects of vasculitis depend on the varying levels of disease and the organs affected. Lesions can range from erythematous macules and/or nodules to hemorrhagic vesicles and palpable purpura to necrotic lesions and ulcerations. They are most frequently found on the back, hands, and buttocks, inside of forearms, lower extremities and extensor surfaces of joints. Treatment is aimed at controlling the underlying disease process. Steroids and immunosuppressive drugs are often prescribed. Local wound care includes debridement and maintaining an appropriately balanced wound care bed for healing (moisture, warmth, protection). Vitamin A ointment has been shown to counteract the anti-inflammatory effects of the steroid therapy.

Other Unusual Wound Conditions

The Irradiated Wound

Approximately 1.3 million new cases of invasive cancer are diagnosed each year. Roughly one half of these patients will receive radiation therapy as treatment for their cancer. Five percent of the patients receiving radiation therapy will develop long-term radiation effects. Radiation causes injury to the soft tissue and bones which then leads to hypovascular, hypocellular and hypoxic tissue. This causes the tissue to breakdown and in turn causes wounds that are difficult to heal. Unfortunately, as time passes, the damaged tissue worsens. This can lead to wounding anywhere from immediately after treatment up to a reported 20 years after treatment, known as late effects of radiation.

While radiation is effective in the destruction of cancerous cells, it is non-selective. Rapidly proliferating tissues such as intestinal mucosa, bone marrow, and skin are more susceptible to radiation. In skin, the rapidly dividing cells (keratinocytes, hair follicles, and sebaceous glands) are more sensitive to radiation. Skin is particularly vulnerable to the effects of radiation due to its state of continuous cellular renewal. The areas at highest risk are those in the treatment field, skin folds and bony prominences. Patients receiving combination therapy are also at increased risk because the concomitant use of chemotherapy may sensitize the basal cells to radiation.

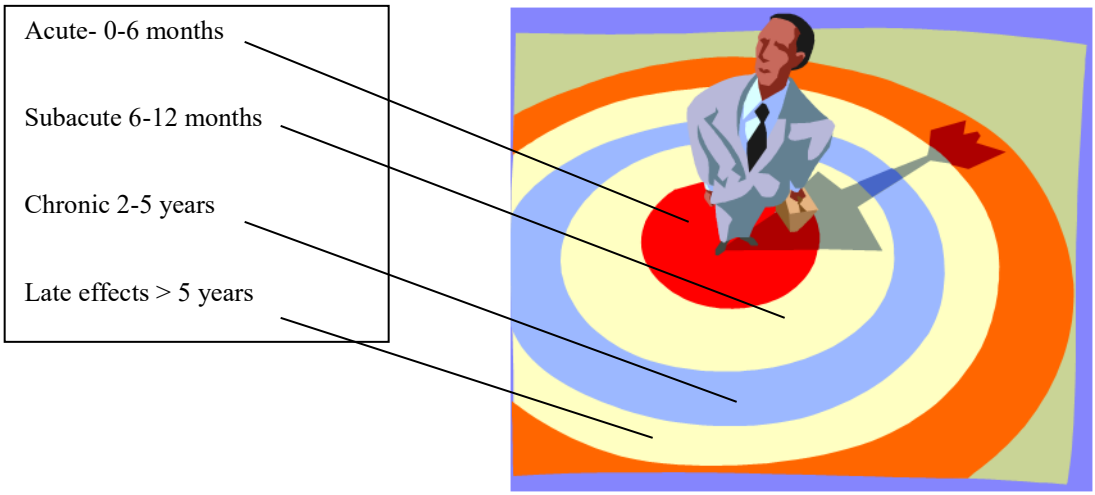
Radiation side effects are generally divided into two categories. First, there are those that happen during or just after the treatment, called acute reactions. Second, there are those that occur months or even years after the treatment called chronic complications or late effects of radiation. Osteoradionecrosis is radiation damage to the bone, soft tissue radionecrosis is radiation damage to the muscle, skin or internal organs.

The acute side effects usually occur 2-3 weeks after beginning therapy or when completing therapy. Acute radiation effects are cumulative, therefore, the greatest reactions occur toward the end of therapy. However, side effects are usually self-limiting, and most subside 1 to 3 months after therapy has ended.

The chronic complications or late effects, unfortunately, do not get better with time, but often worsen with time. Almost all late effects of radiation are a result of scarring and narrowing of the blood vessels within the area which has received the treatment. Think of the late effects of radiation as a bull's eye. The inner most circle is the area of acute effects of radiation and over time the area increases in a circular fashion of widening destruction.

Side effects of radiation can occur anywhere from 2 weeks post treatment until as many as 20 years later. Landthaler reported that most irradiated skin ulcerations erupted on the average at 8 years, 7 months. It has also been reported that patients that received chemotherapy along with radiation therapy were at greater risk for complications. Patients with pre-existing comorbidities such as diabetes and high blood pressure as well as smokers where at an increased risk for complications as well.

The irradiated skin progresses through stages of injury depending on the level or timing of cellular injury. Typically the area progresses through erythema to dry desquamation and then moist desquamation. These irradiated wounds typically present with ill defined, jagged edges with necrotic slough (non-viable tissue) in the base of the wound. Skin that has been irradiated heals with thinner, smoother epithelium. Loss of hair follicles and sebaceous glands, elasticity and decreased healing capacities provide an increased susceptibility to trauma and infection. These wounds are also very painful and should be dressed with the appropriate dressing and or treatment plan to minimize the patient's pain.



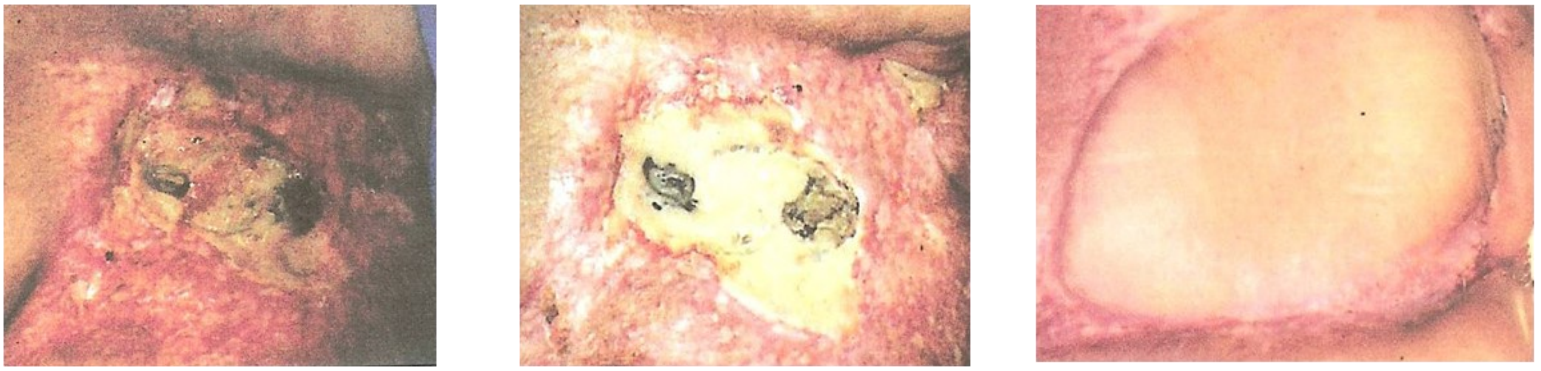


Figure 10-I (left) Radiation necrosis of chest wall several years after mastectomy and radiation treatments. Dry desquamation (middle) same wound 3 weeks after HBO treatments (moist desquamation) preparing patient for myocutaneous flap. (Right) post flap and 6 weeks HBO

Radiation injuries are not limited to the skin or bones. Organs are often involved as well as the surrounding soft tissues. Below is a table of the most common radiation injuries and their symptoms.

Table 10-J

Diagnostic Name	Symptoms of Radiation Injury
Osteoradionecrosis	Dental caries requiring extraction, non-healing wound in oral cavity (usually from dental extractions), non-healing wound under jaw.
Radiation Cystitis	Urinary frequency, urgency, nocturia, gross hematuria, pain
Radiation Proctitis	Diarrhea, rectal bleeding, tenesmus (painful urge to urinate or defecate), abdominal pain, constipation, pain

Think of the bull's eye diagram above. An injury starts in the center and works its way outward over time. Unfortunately, some patients will be more susceptible to radiation than others for unknown reasons. The only proven treatment for the cessation of radiation injury is hyperbaric oxygen treatments. As stated above, radiation injury is related to the narrowing and necrosis of blood vessels, leading to cellular death. When hyperbaric oxygen (HBO) therapy is added to the treatment regimen, angiogenesis (formation of news blood vessels) occur and increase the

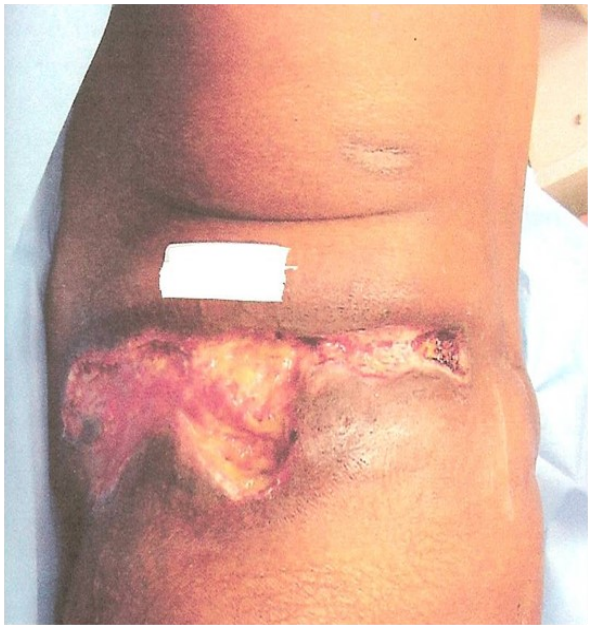
evidence that HBO therapy initiates or promotes cancer. Therefore, when HBO therapy is added to the treatment regimen of the patient suffering from radiation side effects, the "bull's eye" effect (rings of injury) can be halted or delayed enough to allow appropriate healing.

Sickle cell disease is an inherited blood disorder that causes the bone marrow to produce red blood cells with defective hemoglobin, hemoglobin S (sickled hemoglobin). These defective red blood cells clog the capillaries and prevent normal flow to the tissues, which can lead to local hypoxia, thrombosis and ischemia. Sickle cell patients are prone to ulceration and the ulcerations can become chronic. These ulcers are painful, debilitating, difficult to treat and very likely to recur. 97% of healed sickle cell ulcers will recur within 1 year. It is not uncommon for the sickle cell ulcer to become infected or heavily colonized with bacteria. Sickle cell leg ulcers have been noted to heal 16 times slower than venous ulcers. Trauma and anemia have also been associated with an exacerbation of sickle cell disorder.

Sickle cell ulcers are commonly found on the anterior tibial area, dorsum of the foot, Achilles tendon area, and ankles, with the medial malleolus being effected more often than the lateral malleolus. Sickle cell ulcers appear as round "punched out" ulcers with raised margins, deep basis and necrotic slough. The periwound may be brown, hyper -pigmented and scaling. Ulcer formation may be a single ulcer or clusters of ulcers that are very painful. Essentially sickle cell ulcers are the direct result of ischemia secondary to the malformed sickle shaped rigid cells becoming lodged in the microvasculature. Treatment includes the control of edema with compression dressings and/or bed rest. Systemic management of the disease process is crucial as well as debridement, prevention of infection, protection from trauma, providing a moist wound environment and pain management. The use of split thickness skin grafts have been used but, myocutaneous flaps are encouraged because they carry their own blood supply. Another common treatment method is the use of Apligraf® (a bilayered skin equivalent manufactured from neonatal foreskin, keratinocytes and fibroblasts).

Calciphylaxis is a rare, often fatal condition characterized by progressive cutaneous necrosis that frequently occurs in patients with end stage renal disease. Many factors have been suggested as its cause, but the most commonly linked phenomenon is the development of secondary hyperparathyroidism which leads to elevated calcium-phosphate product and development of vascular, cutaneous, and subcutaneous calcification, resulting in tissue death. Calciphylaxis is most often seen following the start of dialysis. There is an estimated 5 year survival rate of less than 50% in patients who develop calciphylaxis. Sepsis from infected wounds is a major cause of mortality and morbidity for these patients.

Figure 10-K Calciphylaxis on torso



There are two clinical variants of calciphylaxis: distal, which appears on the posterolateral calves, fingers, toes and glans penis; and proximal which can occur on the trunk, abdomen, buttocks and proximal extremities. Patients with distal calciphylaxis have a mortality rate of 42% and patients with proximal lesions have mortality rate of 72%. The wounds are characterized by indurated, painful necrotic lesions with a violaceous (purple-red) discoloration. Initially patients develop painful, mottled skin lesions that progress to subcutaneous nodules and ulcerations that eventually become gangrenous. The subsequent infection and gangrene contributes to the high mortality rate in these patients. A distinctive finding with calciphylaxis is that peripheral pulses are intact because blood flow distal to the necrosis or deeper than the necrosis remains intact.

Treatment for calciphylaxis includes both medical and surgical therapies. Medical management includes the use of phosphate binders, low phosphate diets, reduced calcium in the dialysate, antibiotics and diphosphonates. Prompt recognition of the disease and normalization of abnormal calcium and phosphorus levels are crucial. Hyperbaric oxygen and cimetidine have been used with good results. Surgical treatment can include wound debridement, amputation, renal transplantation, and parathyroidectomy.

Topical wound management should address specific wound needs: fill dead space, provide a physiologic environment and absorb exudates. Aggressive debridement is often necessary to reduce the risk for wound infection.

There are numerous disease processes that contribute to delayed wound healing. Couple this with a misdiagnosed wound and you have lost even more ground on the road to healing. Therefore, it is important to obtain a decisive diagnosis as soon as possible along with treating the patient holistically. By doing this, you will greatly improve your outcomes.