Lower-Extremity Vascular Ulcers

Assessment and Approaches to Management

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KEYWORDS

• Arterial ulcers • Leg ulcers • Lymphedema • Venous hypertension • Venous ulcers • Leg wounds

KEY POINTS

- Chronic lower-extremity skin ulcers are often a complication of disorders of the arteries, veins, and/or lymphatics.
- Wound healing is the culmination of defined physiologic processes that can be negatively impacted by diseases and their treatments.
- Identifying specific causes of poor wound healing permits strategies to compensate for or eliminate obstacles to healing.

INTRODUCTION

Vascular leg ulcers are skin wounds of the lower extremities. They may occur de novo but are often the result of minor trauma that fails to heal normally. Their delayed healing is a consequence of underlying arterial, venous, and/or lymphatic disease. Their prevalence is difficult to determine but is likely in the range of 1% to 1.5%.[1,2] This figure varies depending on the age range being considered, as leg ulcers of all 3 varieties are more common with increasing age. This article will not specifically address leg ulcers associated with dermatologic causes, as these were addressed in a recent *Medical Clinics of North America* issue.[3] Vasculitic conditions are the subject of a separate article in this issue.

Arterial and venous leg ulcers are both caused by a derangement of regional hemodynamics. In arterial wounds, the regional arterial blood pressure is lower than normal at rest. In venous wounds, the regional venous blood pressure is higher than normal when the leg is dependent. However, venous hypertension may be ameliorated by elevating the leg above the heart, or even by activating the calf muscle pump with ambulation. The limb in arterial insufficiency withers while the limb in venous

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insufficiency swells. However, in neither condition does the circulation support normal wound healing.

Lymphatic wounds are included here for 2 reasons. First, the lymphatic system is rightly the third component of the vascular system. Second, derangement of lymphatic function is often concomitant of other vascular leg ulcers, especially venous leg ulcers. The lymphatic system does not convey whole blood, but it does participate in the clearing of fluid, metabolites, immune cells, and debris from the interstitial compartment of the periphery. Similar to the cause of venous leg ulcers, lymphatic dysfunction results from diminished flow away from the legs toward the central circulation, resulting in increased interstitial pressure and the accumulation of interstitial fluid.

ASSESSMENT

Arterial Ulcers

History

The diagnosis of arterial insufficiency is typically ascertained from a history of ischemic pain. Pain in relevant muscle groups with ambulation, or claudication, is evidence of ischemia with exercise. Whereas claudication is ischemia of muscles not skin, and then only with exertion, a lower-extremity skin ulcer that is not healing for lack of arterial perfusion implies resting ischemia. Other painful conditions of the lower extremities (eg, arthritis and neuropathy) need to be distinguished from arterial insufficiency.[4]

The pain associated with arterial insufficiency that impairs wound healing is typically in the most distal parts of the limb, in the feet or toes. It may be exacerbated at night when the feet are elevated in bed and may be relieved by getting the feet back into a dependent position. Such patients may find they sleep better sitting up in a chair than reclining flat. The resultant constant dependent position of the leg may result in lower-extremity swelling that mimics venous or lymphatic disease.

Physical examination

Physical examination of the leg with arterial insufficiency will usually reveal absent pedal pulses and sluggish capillary refill (>3 seconds). Nonetheless, the skin may look adequately perfused or, if the foot is dependent, even hyperemic (a finding known as dependent rubor). However, with elevation of the foot above the heart, pallor may be uncovered. Paradoxically, pedal pulses may be palpable in cases of distal atheroembolization (the blue toe syndrome).

Diagnostic studies

The diagnosis of arterial insufficiency is best confirmed by assessing local tissue perfusion. Imaging studies (duplex ultrasound, computed tomography [CT], MRI) yield anatomic information, but the diagnosis of ischemia is best supported by physiologic data. Ankle and toe pressures are readily measured in clinical vascular laboratories. Ankle pressures are generally more accurate but may be subject to systematic error in cases of noncompressible arteries (medial calcinosis) often found in patients with diabetes mellitus or end-stage renal disease. Toe pressures may be preferred in these patients because the digital vessels tend to remain compliant. Pressures may be indexed by dividing by patients' systemic systolic blood pressure, approximated by brachial systolic occlusion pressure.

Venous Ulcers

History

Venous leg ulcers are the most common and account for at least two-thirds of vascular leg ulcers.[5] Ambulatory venous hypertension may be the result of damage to lower-

extremity veins from trauma, surgery, and/or venous thrombosis, so a history should be obtained to elucidate these possibilities. A family history of venous thromboembolic disease may suggest an underlying genetic hypercoagulable condition. Venous hypertension may also occur de novo, as a result of venous valvular reflux, although there is often a family history of venous disease, suggesting a genetic cause. A family history of venous varicosities, leg swelling, hyperpigmentation at the ankles, and/or leg ulcers should be sought.

Physical examination

In the presence of profuse venous varicosities, the diagnosis of venous hypertension may seem obvious. However, ambulatory venous hypertension of a degree that impairs wound healing usually implies substantial pathologic condition in the deep venous system, which need not correlate with the presence of superficial varices. Hemosiderin deposition leading to hyperpigmentation around the ankle is characteristic of chronic venous hypertension. There may be scarring from previously healed leg ulcers around the ankles, and there may be sclerotic changes of the subcutaneous tissues of the ankles even in the absence of overt scars, referred to as lipodermatosclerosis or, in its most severe form, atrophie blanche.[6]

Edema of the subcutaneous tissues is characteristic of venous hypertension and is another important physical finding. The edema of venous hypertension typically spares the feet, because venous insufficiency leading to leg ulcers is usually driven by deep vein pathologic condition, and the venous drainage of the feet is predominantly into the superficial veins. Measurements of calf and ankle circumference permit quantification of the severity of edema and facilitate documenting patients' progress and response to treatment. Of course, if leg swelling and prominent veins are of recent onset, acute deep vein thrombosis may need to be excluded.

Obesity contributes to ambulatory venous hypertension. Negative intrathoracic pressure with inspiration helps draw venous blood up the inferior vena cava, but positive intrabdominal pressure resists venous blood flow into the inferior vena cava. Hence, as obesity increases, so do lower-extremity ambulatory venous pressures.[7]

Lower-extremity venous return is also driven by contraction of calf muscles, which compress the capacitance veins in the deep compartments of the leg. However, anything that compromises the function of the calf muscle pump (eg, paraplegia, stroke, neuropathy, ankle fusion) will exacerbate ambulatory venous hypertension and promote venous leg ulcers. Conversely, active contraction of the calf muscles, especially when the leg is dependent, will facilitate venous return and reduce venous blood pressures.

Diagnostic studies

Ambulatory venous hypertension is usually a clinical diagnosis. Both outflow obstruction and venous valvular reflux may contribute to venous hypertension, but despite ultrasound imaging, CT venogram, MR venogram, and contrast venography, imaging techniques do not correlate well with the severity of the clinical disease. Invasive measurement of pressures is possible, of course, but has been limited to research applications. Ambulatory venous plethysmography is an intriguing diagnostic modality but is not widely available in clinical practice.[8]

Lymphatic Ulcers

History

Lymphedema is diagnosed by a history of lower-extremity swelling. Swelling from primary lymphedema typically precedes any history of trauma, surgery, or infection. Its onset may be in childhood (typically adolescence) or as an adult. There may be a family history of lymphedema, suggesting a genetic contribution, but this is not essential.

Lymphedema may also develop secondary to events that may damage or compromise lymphatic drainage of the legs. Surgical procedures to remove groin and/or pelvic lymph nodes for cancer are the most obvious example. Similarly, radiation therapy to the groin or pelvic lymph nodes may cause sclerosis of lymphatic channels and result in lymphedema. Trauma, infection, or surgery of the lower extremity can damage lymphatic structure and function, resulting in chronic lymphedema. The accumulation and stasis of interstitial fluid in lymphedema predispose to cellulitis. Recurrent bouts of cellulitis can, in turn, further damage lower-extremity lymphatics, leading to a vicious cycle of increasing severity. The most common cause of acquired lymphedema worldwide is filariasis, but this is rare in North America.

Physical examination

Leg swelling may be symmetric, in which case systemic causes of lower-extremity swelling (cardiac, hepatic, or renal disease) may need to be explored. Asymmetric leg swelling, if acute, may of course warrant excluding deep vein thrombosis. Regional lymphadenopathy should also be evaluated and may be the first sign of underlying malignancy.

Lower-extremity swelling in lymphedema may include prominent involvement of the feet and even the toes (Stemmer sign). It is important to determine whether the swelling diminishes with leg elevation, typically overnight in bed. The Starling forces that drive the production of interstitial fluid in the subcutaneous tissues of the legs are substantially reduced if the feet are at the level of or above the heart. Therefore, if leg swelling does not diminish with elevation and the skin does not "pit" with digital compression, a diagnosis of lipedema should be considered. Lipedema also spares the foot, unlike lymphedema.

Diagnostic studies

The diagnosis of lymphedema is entirely clinical. Neither radiologic imaging nor physiologic assessment in the vascular laboratory is of help. Lymphangiograms were performed decades ago, when they were still used in the diagnosis and management of malignancies, but they are now rarely used in clinical practice. However, as surgeons continue to explore techniques for lymphatic reconstruction, methods of assessing lymphatic structure and function are being developed rapidly.[9]

Wound Assessment

History

Regardless of cause, wounds require specific assessment (**Box 1**). A history of how the wound started, how long it has been present, and how it has progressed or changed over time will provide clues to both the causes of the skin injury and the reason or reasons it is not healing. Also important is how the wound has been cared for. How has it been cleansed? What topical or systemic agents have been used to facilitate wound healing? How has it been dressed? How has it impacted the patient's activity or limited the patient? How painful is the wound, and how has the patient dealt with the pain? A history of fever or exudate from the wound will also guide management.

A complete medical and surgical history is important (**Table 1**). Special attention should be paid to conditions that may be associated with compromised wound healing (eg, diabetes mellitus, steroid use, immunosuppression in association with transplantation, cancer treatment, autoimmune diseases). In wounds of the lower extremities, conditions that may be associated with lower-extremity edema (eg, heart failure, renal failure, liver failure) or the use of diuretics (eg, hypertension) will be of interest. Also, a history of lower-extremity trauma and/or surgery may yield important

Box 1
Wound history

- How did the wound start?
- How long has it been there?
- How has it changed over time?
- How is it being cleansed?
- What topical agents have been applied?
- What systemic agents have been administered to aid healing?
- What dressings have been used, and how often are they changed?
- How much pain is associated with the wound?
- How much exudate is there from the wound?

clues, especially if there is prosthetic material retained in the affected limb. Although nutritional deficiencies are uncommon in North America, an inquiry into dietary habits, and sometimes a formal nutritional assessment, may be helpful.

Physical examination

Examination of wounds should include a detailed description of the location and size, including depth (**Box 2**). Color, texture (whether the skin is intact, partially preserved, or absent and its condition), the tissues/planes visible (subcutaneous fat, fascia, muscle, periosteum, bone, and so forth), any exudate, its character, and an estimate of its amount (scant, moderate, copious), and odor of wounds are all important. The peri–wound skin should be included in the examination (**Box 3**). Skin that is dry, cracked, hypertrophic, callused, macerated, indurated, or erythematous will have implications for management. The wound should be palpated, and tenderness, or conversely, the presence of neuropathy, should be assessed. Finally, if there is fluctuance on palpation, there may be an indication for drainage of fluid, blood, or pus.

A diagnosis of invasive infection, cellulitis, or systemic sepsis is generally made on clinical grounds. Pronounced erythema of the peri–wound skin, swelling, and/or induration of the wound and peri–wound tissues, increased warmth of the periwound, and increased pain and tenderness of the wound are hallmarks of locally invasive infection. Systemic signs of fever, tachycardia, or hypotension suggest systemic sepsis.

Table 1
Medical/surgical history regarding vascular leg ulcer healing

• Conditions associated with compromised healing	• Diabetes mellitus • Autoimmune disease • Transplantation • Inflammatory conditions • Malignancy
• Conditions associated with leg swelling and/or diuretic use	• Heart failure • Hepatic disease • Hypertension • Lower-extremity cellulitis, surgery, or trauma • Renal insufficiency • Venous thromboembolism
• Dermatologic diagnoses	
• Diet/nutrition	

Box 2
Vascular leg ulcer physical characteristics

- Location
- Size (including depth)
- Color
- Texture (granular/slough/necrotic)
- Tissues exposed at base (subcutaneous fat, fascia, muscle, periosteum, bone)
- Exudate (character and quantity)
- Undermining
- Odor

Classification systems have been devised for wounds. A common classification designed for pressure injuries but used widely in clinical practice identifies 4 degrees of severity based on the level of wound penetration from the skin: (1) Damage to intact skin; (2) Partial-thickness skin loss; (3) Full-thickness skin penetration into subcutaneous fat; (4) Penetration to fascia, muscle, tendon, or bone[10] (**Box 4**).

A specific classification system has been developed for wounds in arterial insufficiency and is endorsed by the Society for Vascular Surgery known as the Wound, Ischemia, foot Infection (WIfI) risk stratification[11] (**Table 2**). This system incorporates a description of the degree of tissue injury, the physiologic severity of ischemia, and the presence or extent of infection. The WIfI score on initial presentation has been shown to correlate with the risk for leg amputation.[12]

Diagnostic studies

Initial wound assessment is usually limited to history and physical examination. Photographs, ideally with the inclusion of a ruler to allow calibration for measurements, are a useful adjunct to wound documentation and can facilitate monitoring progress. Further diagnostic studies may be directed at elucidating underlying vascular pathophysiology, as outlined above.

Microbiological cultures are rarely helpful and, therefore, are used selectively. Typically, if a clinical diagnosis of infection is made, empiric antibiotics will be indicated. Cultures may be confirmatory or allow focusing of antimicrobial therapy.

Wound biopsy is not typically helpful for the diagnosis of vascular leg ulcers per se, although it may be indicated to exclude other diagnoses. Biopsy may be indicated if a nonvascular cause is suspected, such as for an inflammatory vasculitis or dermatopathy.

Box 3
Periwound skin physical characteristics

- Moist/dry
- Intact/cracked/excoriated
- Hot/warm/cool
- Hypertrophic/callused/macerated/thin
- Indurated/edematous
- Erythematous/cyanotic/bruised

Box 4
Pressure injury staging system

- Stage 1: Intact skin with nonblanchable erythema
- Stage 2: Partial-thickness loss of skin with exposed, intact dermis
- Stage 3: Full-thickness loss of skin with extension into the subcutaneous adipose
- Stage 4: Full-thickness loss of skin with extension to fascia, muscle, tendon, ligament, cartilage, and/or bone

(Adapted from[10] with permission.)

In these instances, biopsy at the ulcer's edge, including some peri–wound skin, is usually the most helpful. Biopsy is also indicated if malignancy is suspected, in which case careful documentation of the biopsy site is mandatory to ensure its inclusion in subsequent extirpation, if indicated.

MANAGEMENT

Fundamentals

Wound healing is the conjunction of myriad metabolic and physiologic mechanisms inherent to human biology. There is no method or technique to make a wound heal. Optimal management permits wound healing to proceed unimpeded. Wound management is directed at identifying obstacles to normal wound healing and mitigating or overcoming those obstacles, thereby enabling wound healing.

The onset of a wound is the initial injury of the skin and/or subcutaneous tissues. Injury sets in motion the mechanisms to heal the wound. After hemostasis, the inflammatory phase is first. In simplest terms, the goal of the inflammatory phase of wound healing is to prepare the wound bed by removing debris and nonviable tissue and controlling bacterial contamination to prevent pathologic infection. Macrophages and polymorphonuclear leukocytes are prominent in the inflammatory phase. The inflammatory phase normally peaks in 24 hours and lasts about 3 days before starting to wane.[13]

Table 2
Wound, ischemia, foot infection classification system

Wound	0: No ulcer, no gangrene 1: Shallow ulcer (may have phalangeal exposure), no gangrene 2: Deep ulcer (exposed bone), gangrenous toe or toes 3: Deep ulcer, gangrene proximal to toes			
Ischemia		ABI	Ankle Pressure (mm Hg)	Toe Pressure (mm Hg)
	0	≥0.8	>100	≥60
	1	0.6–0.79	70–100	40–59
	2	0.4–0.59	50–70	30–39
	3	≤0.39	<50	<30
Foot infection	0: No infection 1: Local infection involving only skin & subcutaneous tissue 2: Local infection involving deep structures (abscess, osteomyelitis, septic arthritis, and similar) 3: Local infection with systemic signs			

(Adapted from[11] with permission.)

On about the third day following wounding, the proliferative phase begins. Collagen production and angiogenesis accelerate. Fibroblasts are prominent, although there appears to be a vital role for multipotential or stem cells, recruited from local tissues and/or bone marrow. The traditional marker of the progress of the proliferative phase has been the tensile strength of the wound (reflecting the production and organization of collagen). In normal wound healing, wound tensile strength is only 5% of the preinjury value after 1 week, 50% after 1 month, and 80% by 2 months.[13]

The third phase is wound maturation. During maturation, there is little change in tensile strength, but the volume of the wound, which now comprises scar tissue, diminishes. The scar will become flatter, softer, and paler. This is often associated with an improvement in cosmetic appearance, but the maturation process takes at least 6 months and often as much as a year. Nonetheless, even the fully healed wound/scar remains an active lesion indefinitely with increased collagen turnover. Hence, the finding that scars that have been healed for years spontaneously dehisce in scurvy (vitamin C deficiency).

On this background, then, it is important that a wound be clean and free of debris and foreign bodies. Gentle cleansing and copious lavage are useful. Harsh chemicals (hydrogen peroxide, isopropyl alcohol, iodine, sodium hypochlorite, and so forth) have little place. Mechanical debridement may be indicated.

The open wound is contaminated. Sterility is not an achievable goal outside of, perhaps, a formal operating room. Topical antimicrobials are touted for their ability to kill or suppress the growth of microorganisms. However, one might question whether the real issue is not what organisms they discourage, but rather which ones they allow, because those are the ones that will populate a wound following their application.

On the other hand, maintaining a moist environment is essential. Desiccation kills tissues at the wound surface, creating new, dead debris that requires removal for wound healing to progress. Topical ointments and foam or gel dressings are available to promote a moist, physiologically balanced wound environment while absorbing wound exudate and suppressing bacterial and/or fungal overgrowth. Periodic changing of these dressings will permit wound cleansing as well as renewal of a wound environment that will permit healing.[14]

An intact blister, with clear fluid and without excessive pain and/or erythema of adjacent tissues, may provide a suitable moist environment for a short time. However, it can easily become a nidus of infection and should be debrided before it does. Similarly, a firm, solidly adherent scab or eschar may be an effective barrier to protect the underlying tissues from desiccation or bacterial overgrowth while healing gets started but will require monitoring. Evidence of separation suggests the barrier is no longer effective, at which point the nonviable material should be carefully removed.[14]

Arterial Ulcers

The primary intervention for lower-extremity wounds that have a significant ischemic component is to restore adequate arterial flow. Both endovascular and open surgical techniques may be useful. Although moderate peripheral arterial disease (Ankle Brachial Index [ABI] >0.5) will often not interfere with healing, conditions that may compromise the microvasculature (eg, diabetes mellitus, radiation) often mandate the best arterial flow possible. When healing is thwarted by microvascular disease (radiation injury, diabetes mellitus) despite adequate arterial inflow, hyperbaric oxygen therapy may be a useful adjunct.

Care should be taken to avoid unduly compressive or constricting dressings in patients with arterial disease. Arterial blood flow is driven by blood pressure.

Compressive or constricting dressings may diminish blood flow, resulting in ischemia of the wound.

Although the removal of frankly dead or infected tissue is appropriate, debridement of ischemic wounds should be judicious. Specifically, debriding back to healthy, bleeding tissues should be avoided because in the face of ischemia the bleeding tissues that are injured will not heal and further tissue necrosis may ensue.

Infection of ischemic wounds is especially problematic. As already pointed out, bacterial colonization is common and of little clinical consequence. Correspondingly, antimicrobial therapy absent of clinical infection is of no utility. On the other hand, invasive infection is a risk of any open wound. Invasive infection warrants systemic antibiotics. However, in ischemic wounds, systemic antibiotics are unlikely to reach the wound sufficiently to be effective. Furthermore, even if the antibiotics could somehow be "forced" into the region of infection, absent of an influx of leukocytes and their ability to provide a vigorous oxidative attack on invading microbes, infection may still prevail.[15] Consequently, surgical consultation should be sought for infected ischemic wounds. Appropriate interventions may include drainage of abscess, debridement of nonviable tissues, and revascularization to alleviate the ischemia.

Venous Ulcers

The mainstay in the management of venous leg ulcers is compression therapy.[16] Compression is applied from the midfoot to the proximal calf, but short of the knee. Compression should be greater than 40 mm Hg.[17] Compression may be elastic or inelastic and may take the form of rolled dressings, stockings, or adjustable inelastic garments.[18]

High-grade compressive dressings require expertise to apply safely and effectively. They should be changed at least weekly, and more often if there is copious drainage from wounds. Healing often takes several months. Expedient healing is the goal, but on a week-by-week basis, it is sufficient to see progress with wounds granulating, becoming shallower, and closing in from the periphery.

Maintaining a clean ulcer bed is intuitive. Gentle cleansing with mild soap and water is fundamental. However, debridement and even surgical curettage may be used, although the benefits have not been well documented in the literature. The twin objectives are to remove slough and bioburden in order to achieve a clean ulcer bed, as well as to convert a stalled, chronic wound to an acute, healing wound by inciting a degree of inflammation (the first phase of wound healing). Debridement of venous leg ulcers is recommended by both the Society for Vascular Surgery and the American Venous Forum.[19]

Because both venous leg ulcers and peripheral arterial disease are more common with advancing age, it is not unusual to see patients in whom both are considerations. Moderate compression, greater than 40 mm Hg, may be applied if ankle pressure is greater than 60 mm Hg, toe pressure is greater than 30 mm Hg, and ABI is greater than 0.6. However, close monitoring of the patient is prudent, and compression should be abandoned if there is marked pain with compressive bandaging or if ulceration deteriorates. If arterial perfusion pressures are not adequate or compression is not tolerated, consideration should be given to arterial revascularization before using compression.[20]

Good skin hygiene is also fundamental to venous leg ulcer healing. A hypertrophic response of epidermal thickening and excessive dryness is common and is referred to as stasis eczema. This, in turn, can be associated with pruritis, which can result in scratching and excoriation, leading to additional skin injury. The use of hypoallergenic, nondrying soap for cleansing and liberal application of topical emollients will improve

the skin texture, reduce epidermal hypertrophy, and ameliorate pruritis. Pharmacologic agents have also been identified to facilitate venous ulcer healing. They include flavonoids,[21] sulodexide,[22] and pentoxifylline,[23] but the evidence is of limited quality, and the improvement seen is modest.

Lymphatic Ulcers

The management of lymphatic ulcers parallels that of venous ulcers. In clinical practice, the two often overlap with coexistent pathologic condition of both vascular systems. The difference between them is that there are almost always more than sufficient veins for venous return, they simply are not working adequately when the leg is dependent. In severe lymphedema, there are often insufficient lymphatic channels to clear the accumulated interstitial fluid, so the emphasis is on limiting its accumulation in the first place.[24]

Elastic garments and stockings can be used for lymphedema management, but because the goal is to manipulate the Starling forces that drive interstitial fluid production, the pressures needed can be problematic. Inelastic garments that are applied when swelling has ebbed and that will not permit expansion of the subcutaneous compartment are often more easily used, better tolerated, and consequently more effective.[25]

Lymphedema pumps consist of leggings with multiple circumferential pneumatic compartments that are inflated sequentially to high pressures. These can also be crucial to the management of lymphedema. Although the pumps are only therapeutic for the hour or two a day that patients spend in them, their ability to achieve an acute reduction of subcutaneous interstitial fluid in the legs then permits the timely application of the garments described above that can maintain control of interstitial fluid accumulation.

Cleansing, debriding, and dressing lymphatic ulcers mirror the care of venous ulcers. Similarly, in lymphedema, the adjacent skin is subject to many of the same stresses as in ambulatory venous hypertension, so a clinical picture of stasis dermatitis is common. The use of hypoallergenic, nondrying soap for cleansing and liberal application of topical emollients will similarly improve and protect the skin.

PREVENTION OF RECURRENCE

The prevention of recurrent arterial ulcers is the maintenance of adequate arterial perfusion. This mandates the management of risk factors for atherosclerotic peripheral arterial occlusive disease. If the patient has undergone revascularization, by either endovascular techniques or open surgical intervention, then the maintenance of patency of those interventions, including both pharmacologic therapy and surveillance imaging, is crucial. However, the elaboration of these is beyond the scope of this article.

The recurrence rate of venous ulcers is high. Prevention of recurrence is similar to treatment, in that compression is key. However, compression is also unpopular, and once the ulcers are healed, patients' motivation can wane. Higher pressure is more effective in preventing recurrence, but lower pressure is better tolerated.[26,27] However, the lowest recurrence rates for venous leg ulcers correlate with the highest compliance in wearing compression garments, perhaps more than the degree of compression.[28]

The cause of venous leg ulcers is venous hypertension, often demonstrably the result of venous valvular reflux in the affected leg. Although deep venous reflux may be the primary driver, there is often a substantial component of superficial venous reflux. Studies have demonstrated lower recurrence rates following ablation of incompetent superficial veins, even in the presence of coexisting deep venous reflux.[29]

Furthermore, there may be an advantage to addressing superficial venous reflux to hasten the healing of extant venous ulcers.[30] Similarly, iliac and/or inferior vena caval obstruction (May-Thurner syndrome) may be alleviated by endovascular stenting with a reduction in venous leg ulcer recurrence.[31]

FAILURE TO HEAL

Everything in this article so far has focused on healing vascular leg ulcers. However, wound care practitioners recognize that some wounds do not heal—ever. However, this should be a designation that is only assigned after exhaustive attempts to heal the wound, and even then, the designation is only tentative. However, before deciding that a vascular leg ulcer will not heal, there are some things to consider.

Is the ulcer not healing, or is one ulcer healing yet another is appearing? The leg is never healed, but each individual ulcer may heal. This suggests an underlying skin disorder that is driving the development of new ulcers.

Is it fundamentally a skin disorder not caused by a vascular cause? Pyoderma gangrenosum, calciphylaxis, and skin cancers are examples of skin conditions that may be recalcitrant. Dermatologic conditions can coexist with arterial, venous, and/or lymphatic disease. In the event of a nonhealing leg ulcer, consultation with dermatology may help identify and curtail a cause, thereby permitting durable healing of the leg.

Is the patient manipulating the wound so as to prevent healing? The compressive dressings used for venous and lymphatic ulcers are often effective in preventing patients from getting access to their wounds, thereby allowing them to heal. Nonetheless, just as orthopedists are familiar with patients sliding objects inside their casts, patients may find a way to thwart the protection afforded by the compressive dressings. They may do it inadvertently, not realizing that they are undermining efforts to heal their ulcers, or they may do it for secondary gain.

Finally, if healing seems to evade all efforts, the best that may be achieved is stability and maintenance of the wound. Efforts should be directed at keeping the wound clean, keeping the wound free of infection, and minimizing its negative impact on the patient's quality of life. Chronic wounds are a burden, but effective wound care can help sustain the individual who must bear such a burden.

CLINICS CARE POINTS

- It is important to determine both the etiology of a leg ulcer and the factors that are impeding its healing.
- Cleansing a chronic wound to minimize devitalized tissue and reduce bioburden supports healing.
- Both desiccation and maceration of wounds slow healing. Wound dressings should be selected to maintain a moist environment but to avoid both desiccation and maceration.
- Venous leg ulcers are maintained by venous hypertension. Reduction of venous hypertension and compressive support are essential to achieve healing.
- Wounds complicated by ischemia require referral for revascularization.

DISCLOSURE

The author has nothing to disclose.

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