Accelerated Wound Healing: Multidisciplinary Advances in the Care of Venous Leg Ulcers

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Accelerated Wound Healing: Multidisciplinary Advances in the Care of Venous Leg Ulcers

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The etiologies of venous leg ulcers have been well known for millennia, and yet there remains no simple solution to this very common problem. Achieving closure of venous leg ulcers is often a lengthy process that is further complicated by the presence of significant comorbidities. The authors present data on healing venous leg ulcers in a cohort of 231 patients, most of whom had 1 or more complicating factors. Our multidisciplinary and aggressive approach to healing venous leg ulcers is described and has resulted in an average healing time of 29 days, a significantly shorter duration of treatment than the reported average of 6 months.

Introduction

At any given time, approximately 1% of the US population has experienced, or has currently, an active leg ulcer of venous etiology.[1] The incidence of venous leg ulcer (VLU) formation increases with age and is more common in women. In the United States, venous leg ulcers develop in at least 300 per 100,000 of the population.[2,3] The annual cost of treating venous ulcers is more than 1 billion dollars,[4] and that figure does not include the economic impact of associated life style alterations, time lost from work, and frequent hospitalizations. Venous leg ulcers are the end result of severe, chronic venous insufficiency characterized by edema, venous dilatation, leg pain, and stasis dermatitis.

Development of a plan of care for patients with venous leg ulcers must address several concomitant issues including healing of the active ulcer and prevention of ulcer recurrence. Key to achieving these goals is treatment of ulcer-associated infection, prevention of infection development, and stimulation of granulation and epithelial repair. While the reported rate of venous leg ulcer healing averages 6 months,[5] we have developed care protocol modifications that produce average venous leg ulcer healing times of 29 days.

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Patients and Methods

From October, 2001 to February 2005, 231 patients presented to the Lake District Hospital Wound Clinics for treatment of 296 venous leg ulcers. Details of this patient cohort are summarized in Table I.

Before initiating ulcer care, all patients were evaluated for the presence of significant comorbidities, most notably diabetes mellitus, arterial occlusive disease, smoking, the presence of methicillin-resistant *Staphylococcus aureus* (MRSA) infection, and obesity. All ulcers were cultured. Heavy or moderate growth organisms were assessed for antibiotic sensitivity and, when appropriate, patients were referred back to their physicians for antibiotic treatment. Ulcer characteristics, including volume, color and type of exudate, presence of odor, ulcer location, diameter, and depth were documented.

Venous leg ulcer treatment plans were developed for each individual lesion. In all cases utilizing compression bandaging of any type, such bandages were constructed to extend from the metatarsal heads to the knee. In no circumstances were compression bandages localized to the affected limb segment, eg, "gaiter" area, only. When necessary, additional bandaging was extended to include affected toes.

All venous leg ulcers were cleaned thoroughly but gently with full-strength hydrogen peroxide. This solution was delivered as a pressurized stream that, coupled with the foaming action of the catalase reaction, resulted in nontraumatic removal of debris within and around the ulcer. For very heavily crusted, dirty, or infested ulcers, the

Table I. Patient characteristics (n = 231 patients).

Age, years 49 ±19*			
Gender, n (%)	Men 74 (32%)	Women 157 (68%)	
No. of VLUs on admission	1	2	> 2
n (%)	152 (66%)	57 (25%)	22 (9%)
Comorbidities			
Diabetes mellitus	IDDM 27 (12%)	NIDDM 72 (31%)	
PAD (ABI < 0.85), n (%)	127 (55%)		
Active smoking, n (%)	145 (63%)		
Obesity (BMI > 30), n (%)	168 (73%)		
Varicose veins, n (%)	124 (54%)		
Stasis dermatitis, n (%)	154 (67%)		
Lipodermatosclerosis, n (%)	60 (26%)		
Recurrent VLU, n (%)	48 (21%)		
Duration of active ulcer(s)	31 ±19 months (range 2–120 months)		

*Mean ±1 SD.

n = no. of patients, % = percent of cohort, VLUs = venous leg ulcers, PAD = peripheral arterial disease, ABI = ankle/brachial systolic pressure index, IDDM = insulin-dependent diabetes mellitus, NIDDM = noninsulin-dependent diabetes mellitus, BMI = body-mass index.

lesions were first soaked in warm soapy water and gently scrubbed with a foam sponge impregnated with 3% chloroxylemol. This was followed by irrigation with pressurized hydrogen peroxide, as above. Finally, for those patients who were not allergic to iodine, the ulcers were painted with betadine solution before dressing was applied.

Dressing strategies were, again, individualized to address the characteristics of specific wounds, with the overall goal of channeling exudative elements away from the wound bed. Toward this end, heavily exudative venous leg ulcers were packed and covered with multiple layers of calcium alginate felt that was supported with several layers of sterile gauze. An appropriately compressive, overlying bandage system, utilizing short-stretch or nonstretch materials, was constructed such that no bandage materials, other than sterile cotton gauze, were in direct skin contact.

Those patients whose VLUs were complicated by the presence of severe stasis dermatitis and/or lipodermatosclerosis had a paste bandage Unna's Boot (Unna's Boot Dressing. Patient Technology, Inc, Atlanta, GA 30360, USA) applied over the ulcer dressing and extending from the metatarsal heads to the knee. This compression bandage was reinforced with sterile cotton gauze and overlying short stretch materials.

The degree of compression achieved was controlled by continuous monitoring of digital artery pressure pulse waveforms when present. In the absence of digital artery waveforms, intermittent monitoring of forefoot or ankle waveforms was performed. A handheld, 8 or 9 MHz continuous-wave Doppler instrument was also used to insonate digital or pedal arteries before, during, and after bandaging. In all cases, developed bandage compression did not exceed the measured, distal artery perfusion pressure.

Frequency of dressing/bandage system changes was dictated by the amount of exudate put forth by the venous leg ulcer. Heavily exudative wounds initially required up to 6 layers of alginate felt and daily dressing changes. This schedule was quickly revised to provide fewer dressing changes as healing progressed. During the final healing phases, whenever possible, patients were fitted for appropriate compression garments and were instructed in a local care regimen. Discharge from clinic care occurred when the ulcer was completely healed. At that time the patient was strongly encouraged to continue use of the provided compression devices and to seek physician consultation at the first sign of ulcer recurrence.

Nutritional evaluation of venous leg ulcer patients revealed a nearly universal, inadequate consumption of high-quality dietary protein, vitamins C, E, and B complex and minerals including zinc and selenium. Appropriate supplementation was often necessary to promote healing. In diabetic patients, close monitoring and control of serum glucose to levels consistently < 120 mg/dL was essential to the healing process. For many patients, the establishment and maintenance of good personal and environmental hygiene practices, as well as avoidance of leg trauma, were necessary to prevent venous leg ulcer recurrence.

Venous leg ulcers that were infected with methicillin- and/or vancomycin-resistant *Staphylococcus aureus* (MRSA/VRSA) or very resistant strains of *E. coli*, *Pseudomonas* or *Proteus* required very aggressive treatment aimed at reducing the density of flora and inhibiting further growth. We found that pressurized irrigation of such lesions with a 0.05% solution of sodium hypochlorite in normal saline and use of antimicrobial dressing materials, including calcium alginate felt coated with ionic silver (Acticoat Absorbent Antimicrobial Dressing. Smith and Nephew, Inc, Largo, FL, USA) and cotton gauze impregnated with 0.2% polyhexamethylene biguanide (Kendall Kerlix A.M.D. Antimicrobial Super Sponges. Tyco Healthcare Group LP, Mansfield, MA, USA), were essential to defeating the infection. All wound care, regardless of flora status, was performed using contact isolation protocols.

Healing of all venous leg ulcers was greatly supported by exposing the ulcers, for daily, 4-hour periods, to 100% oxygen under pressure while the patient was also administered 100% humidified oxygen by mask. Small, shallow venous ulcers required only 2–3 consecutive days of oxygen treatment. Large, deep lesions, overtly infected lesions, VLUs in diabetic patients, patients with peripheral arterial disease (PAD) and/or smokers, and any ulcers infected with MRSA, VRSA, or other highly resistant organisms were administered a series of 5–7 consecutive daily oxygen treatments.

To provide oxygen under pressure to venous leg ulcers, any overlying dressing/bandage was first removed and the lesion was cleaned with hydrogen peroxide. A 4-inch-wide strip of medical-grade, closed-cell, soft foam was placed around the leg at a point proximal to the lesion. A clear plastic bag was placed over the leg and foot and secured over the foam cuff by another, overlying foam cuff secured circumferentially with tape.

Oxygen tubing was inserted into the bag, via a small cut, and secured with tape. A small puncture was made in the bag to act as a pressure vent and prevent bursting. Oxygen inflow to the bag was set to 10–15 L/m. Concomitant, humidified oxygen was administered by mask at a rate of 10 L/m.

Depending on the type of bag used, the integrity of the seal around the limb, and the size of the vent cut into the bag, 2.5–3.0 atmospheres of pressure were present in the bag when maximally inflated. Following completion of the oxygen treatment, appropriate wound toilet, dressing, and bandaging were performed.

Results

Table II lists healing times for venous leg ulcers of varying sizes, and Table III correlates healing times for venous leg ulcers occurring in patients categorized by the presence of 1 or more comorbidities. As expected, small, shallow VLUs healed rapidly while the large, deep VLUs required extended healing times.

The number of days required to achieve complete healing was extended in all VLU patients, regardless of ulcer size, in whom were present more than one of the listed comorbidities. The greatest impediments to rapid healing of VLUs

Table II. Correlation of venous leg ulcer size with time to healed.

No. of Patients	VLU Dimensions, cm Maximum Diameter/Depth	No. of Days to Healed
21	< 1/< 1	2–5
37	1–2/< 1	6–20
34	2–4/1–2	10–40
121	4–7/> 2	14–52
18	> 7/> 2	17–120

VLU = venous leg ulcer.

Table III. Correlation of comorbidities present with time to healed.

No. of Patients	Comorbidities Present	Days to Healed
36	DM + smoking	19–46
96	DM + obesity	12–28
17	DM + PAD	23–100
79	DM + PAD + smoking	51–120
12	DM + MRSA	5–26
88	Obesity + stasis dermatitis	4–22
52	Obesity + stasis dermatitis + smoking	20–34

DM = diabetes mellitus, PAD = peripheral artery disease (ABI < 0.85), MRSA = methicillin resistant *Staphylococcus aureus.*

were the presence of diabetes mellitus, smoking, and peripheral artery disease. Those patients who had all 3 of the comorbidities also had the longest healing times. Obese patients and those with varicose veins, stasis dermatitis, lipodermatosclerosis, and/or resistant ulcer infections did not have significantly extended healing times unless these conditions were complicated by the presence of diabetes, smoking, and/or peripheral artery disease. The length of time an ulcer had been present before treatment and a history of recurrent venous stasis ulceration appeared to have little or no impact on time to achieve complete healing.

The specific effects of individual elements of the treatment protocols cannot be determined from these data since all appropriate treatment strategies were aggressively employed in every case.

Discussion

All venous leg ulcers in all 231 patients were healed completely. Healing times were lowest (2–5 days) in patients with small ulcers who were < 50 years old and who were also nonsmokers and did not have diabetes mellitus. Extended healing times (> 1 week) correlated well with larger ulcer sizes and the presence of 2 or more comorbidities, eg, diabetes mellitus plus significant peripheral artery disease (ABI < 0.80) and/or persistent smoking. The patients who required the longest healing times were those for whom compression bandaging was contraindicated,[6] ie, diabetic patients with severe arterial occlusive disease (ABI < 0.70). However, with aggressive management, all of the patients in this last designation (n = 12) were healed within 120 days.

Continuous monitoring of digital artery pressure pulse waveforms or Doppler signals is of great utility in controlling the degree of compression achieved during bandaging. Whether compression bandages were constructed with paste dressings (Unna's Boot), short stretch, or nonstretch materials, they always extended from the metatarsal heads to the knee and were always tightest at the ankle level. Use of materials that were at least 4" wide prevented development of constricting bands in the bandages. Even bandages constructed to achieve little or no compression still extended from the metatarsal heads to the knee in order to support the superficial venous system.

Venous leg ulcers that are frankly infected require very close monitoring and frequent dressing changes to prevent tissue maceration. Often, such patients have already undergone care elsewhere and the infecting agent is iatrogenic and widely resistant to antibiotics.[7] In these circumstances, and barring known drug allergies, the patient is referred for antibiotic treatment while continuing to undergo appropriate wound care efforts. When such ulcer infections occur in the diabetic patient, rapid identification of the responsible organism(s) and institution of antibiosis are essential to control the wound and prevent further tissue destruction.[8]

The addition of ulcer treatment via exposure to oxygen-enriched environments has proven to be a powerful adjunct to VLU healing.[9] Provision of 100% oxygen via mask to the patient at the same time that pressurized oxygen was administered to the lesion has shown itself to be superior to local treatment alone. Appropriate nutritional amendment further accelerates the healing process.[10] Whenever possible, we encourage our patients with VLU to resume normal physical activity and to ambulate freely. Persistent calf muscle pump action and the increased limb perfusion caused by exercise can only improve the ulcer environment and predispose to healing.

Finally, rapid healing of VLUs has resulted in considerable cost reduction, both in terms of the direct cost of treatment as well as the reduction of the number of days lost from work and the number of associated hospital admissions. The value of the improved quality of life for the VLU patient is incalculable.

From this experience we conclude that an aggressive, multidisciplinary approach to venous leg ulcer care results in accelerated healing of these lesions, even in the presence of significant and severe comorbidities and challenges.

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