

The management of venous stasis ulcers

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Case history

A Caucasian male in his fifties with lower extremity edema and large bilateral stasis ulcers that had been present intermittently for the past 30 years and continuously for the past six years. He had been hospitalized on numerous occasions and had failed six skin grafts and a growth factor clinical trial prior to this presentation. He denied comorbid factors except for two pack per day smoking and obesity. He stated that he had never received compression therapy of any type. On examination, lower extremity circumference measurements were right calf 53 cm, left calf 65 cm, right ankle 27.5 cm, left ankle 32 cm, right mid foot 28.5 cm, left mid foot 31 cm. Ulcer dimensions were 13.5 cm x 11cm x 6cm over the left lateral malleolus, 32 cm x 24cm x 5 cm over the left medial malleolus, and 5.5 cm x 3.2 cm x 4 cm over the right lateral malleolus. Pulses below the knee were Dopplerable bilaterally. Surface swab cultures were positive for staphylococcus aureus and pseudomonas aeruginosa for which his referring physician treated him with ofloxacin and clindamycin. Tissue biopsy for culture and histology were done in the center. These cultures were negative, and systemic antibiotics were discontinued. A KOH prep revealed the presence of hyphae, which was treated by topical applications of an antifungal agent. Multiple biopsies showed no evidence of malignant transformation. Plain radiographs showed no evidence of osteomyelitis.



Introduction

The prevalence of lower extremity venous disease in our population is greater than that of peripheral arterial occlusive disease, although the latter receives more attention. An estimated 35 percent of the adult population has lower extremity venous abnormalities and one-fifth of these individuals will acquire one or more venous ulcers in their lifetime. Up to 600,000 individuals in the United States have a venous ulcer at any given time, and the annual incidence is 0.35% of the adult population. As many as 2 million working days are lost each year to venous ulcers with up to one-third of these patients limited in their ability to work outside the home and nearly 13% retiring prematurely as a result of the disease.

Pathophysiology



There are both macro and microvascular responses which occur in the presence of chronic venous insufficiency. At the macrovascular level there is failure of the “calf muscle pump” which can result in reflux with valvular incompetence. Valvular incompetence of the deep perforating veins is present in the majority of patients who present with ulceration. Microvascular changes include increased capillary permeability, decreased capillary density, extravasation of red blood cells causing hemosiderin deposits and leading to lipodermatosclerosis, and white blood cell trapping with associated inflammation. Over time chronic lymphedema develops. A variety of factors contribute to the development of ulceration including concomitant peripheral arterial occlusive disease (10-25%), rheumatoid or other collagen vascular disease (10-25%), and diabetes mellitus (5-12%). The typical clinical course of these ulcers is one of alternating periods of ulceration and temporary healing.

Diagnosis

The typical venous stasis ulcer presents with an irregular shape and well defined borders, usually surrounded by fibrosed skin and often by erythema. The distribution of these ulcers has been classically described as in the malleolar (“gaiter”) area. There is almost always clinically identifiable edema and often associated lipodermatosclerosis. Secondary bacterial overgrowth is common, and cellulitis is a frequent complicating event. The differential diagnosis includes arterial insufficiency ulcers, primary bacterial infection, primary dermatological conditions, primary or metastatic neoplasms (or malignant transformation

within a chronic venous stasis ulcer), and vasculitic processes all of which may also coexist with a stasis ulcer.

Specific diagnostic assessment includes the following:

1. Arterial assessment
 - Hand held Doppler for ankle and digital pressures, ABI
 - Arterial Doppler studies with abnormal pulse exam or history of claudication, etc
 - TcPO₂ study
2. Venous assessment
 - Venous Doppler (may be required for reimbursement for pneumatic compression pumps)
 - Venous plethysmography or duplex scanning
 - Venography (candidates for surgical venous reconstruction)
3. Lymphatic studies for the evaluation of unilateral swelling with unclear etiology (generally not required).
4. Biopsy of suspicious lesions or those which fail to respond to appropriate compression therapy.
5. Radiographs of deep, chronic ulcers to rule out underlying osteomyelitis.
6. Screening laboratory studies directed at identifying concomitant disease suggested by the history.
7. Cultures.



Treatment

The most important principle in the management of venous stasis ulcers is establishing an adequate regimen of edema control. A variety of options are available and are summarized in Table 1. For most patients, the primary means of edema control is a modified Unna's boot or one of the commercially available dry, prepackaged compression wraps. This compression wrap is usually initially changed twice weekly then extended to once each week once tolerance and response to compression therapy has been established. In cases with marked edema, compression pumping can be used initially over the compression boot on an intermittent basis to more rapidly achieve edema control. Compression must be at least 20-30 mmHg at the ankle decreasing towards the knee and must be sustained. However, in patients with evidence of peripheral arterial occlusive disease, compression may need to be limited further; in severe cases compression is contraindicated. Ulcer care involves debridement limited to only obviously non-viable tissue, treatment of invasive soft tissue infection with systemic antibiotics, control of secretions, prevention of desiccation of healing tissue, and control of maceration of the surrounding skin. Typical ulcer contact dressings include absorbent calcium alginate or foam dressings during the highly exudative phase and hydrocolloid occlusive dressings once there is less exudate. Both of these dressing types can be applied under an Unna's boot or dry compression wrap.

Other treatment modalities have been utilized including sclerotherapy for superficial and perforating veins, valvuloplasty, venous transpositions and other surgical reconstruction, pharmacological intervention, topical human platelet derived growth factor, cellular and/or tissue based products, and hyperbaric oxygen treatment. In our experience, the single most important treatment modality for stasis ulcers is external limb compression of some type. Hyperbaric oxygen treatment is rarely indicated but is used when reversible hypoxia remains after achieving adequate local edema control in support of skin grafting. When used, adjunctive hyperbaric oxygen treatment must be accompanied by continued aggressive edema control.



Case history

Following admission to the hospital the Caucasian male was placed at bed rest with leg elevation. A venous duplex scan showed no evidence of deep vein thrombosis (DVT), and an arterial Doppler study showed no evidence of significant peripheral vascular disease (PVD). Every other day Unna's boot applications were begun with a calcium alginate and foam contact dressing. After three days, he was discharged to outpatient care with twice weekly boot changes. At four weeks the ulcer on the right leg was healed and the

boot replaced by a stocking. A gradient sequential compression pump was acquired and used to provide additional compression with pumping at 60mmHg for one hour BID. He continued with twice weekly Unna's boot changes to his left leg with excellent edema control and 60% reduction in wound surface area by eight weeks when his care was transferred to another more convenient facility. It was anticipated that he would have complete resolution of both wounds by 12 weeks.



Clinical outcomes

When an accurate diagnosis is made and an appropriate level of edema control is consistently achieved, initial healing of stasis ulcers can be expected in 90 percent of patients. The treatment duration will be dependent upon the extent of local edema, the size of the wound(s), and the presence of co-morbid factors. Common causes for treatment failure include inadequate edema control, unrecognized systemic or local disease, underlying osteomyelitis or invasive soft tissue infection (bacterial or fungal), or unrecognized local malignancy. Recurrence of ulceration is most frequently the result of failure to maintain consistent edema control following initial healing. Stasis ulcer patients require lifelong monitoring and reinforcement of edema control compliance. Venous stasis ulcer management following the general principles presented here provides both successful and cost effective outcomes.

Table 1

Device	Pros	Cons
Compression bandages	<ul style="list-style-type: none"> ▪ Some are removable for hygiene ▪ Inexpensive ▪ Patient or care giver can apply if appropriately trained 	<ul style="list-style-type: none"> ▪ Can unravel ▪ Care must be taken to apply evenly and in a graduated fashion
Stockings	<ul style="list-style-type: none"> ▪ Can be removed at night and for hygiene ▪ Most appropriate for maintenance phase ▪ Compression reduced as edema resolved ▪ Elasticity value reduces over time ▪ Best fitted after edema resolves 	<ul style="list-style-type: none"> ▪ Can be difficult to don ▪ Must be fitted by trained personnel
Boot system	<ul style="list-style-type: none"> ▪ Particularly appropriate if patient unable to apply elastic bandages ▪ Bulky in appearance ▪ Must be applied by trained personnel 	<ul style="list-style-type: none"> ▪ Cannot be removed at night or for hygiene
Legging orthosis	<ul style="list-style-type: none"> ▪ Can be removed at night for hygiene ▪ Easier to do than compression stockings or elastic bandages 	<ul style="list-style-type: none"> ▪ Must be custom fitted by trained personnel ▪ Bulky in appearance
Pneumatic pump	<ul style="list-style-type: none"> ▪ Quickly reduces edema ▪ Effective when edema is severe and/or refractory 	<ul style="list-style-type: none"> ▪ Expensive ▪ Requires special equipment ▪ Requires periods of immobilization

References

1. Burton CS. Venous Ulcers. The American Journal of Surgery 1994;167(1A) 37S-41S.
2. Goldman MP, Fronek A. Consensus paper on venous leg ulcer. J Dermatol Surg Oncol 1992; 18:592-602.
3. Kerstein MD, Gahtan V. Outcomes of venous ulcer care: Results of a longitudinal study. Ostomy Wound Management 2000; 40(6):22-29.
4. Langemo DK. Prospective multicenter study of managing lower extremity venous ulcers. Advances in Wound Care 1996; 9(3):12.
5. Macdonald JM. Wound healing and lymphedema: A new look at an old problem. Ostomy Wound Management 2001; 47(4):52-57.
6. Margolis DJ. Management of unusual causes of ulcers of lower extremities. J WOCN 1995; 22:89-94.
7. Moffatt CJ, O'Hare L. Venous leg ulceration: Treatment by high compression bandaging. Ostomy/Wound Management 1995; 41(4):16-25.

8. Sibbald RG, Torrance GW, Walker V, Attard C, MacNeil P. Cost-effectiveness of Apligraf in the treatment of venous leg ulcers. *Ostomy Wound Management* 2000; 40(8).
9. Criqui MH, Denenberg JO, Bergan J, Langer RD, Fronck A. Risk factors for chronic venous disease: the San Diego Population Study. *J Vasc Surg.* 2007 Aug;46(2):331-7.
10. Simka M, Majewski E. The social and economic burden of venous leg ulcers. *Am Clin Dermatol* 2003;4:574-81.
11. Association for the Advancement of Wound care (AAWC). AAWC Venous Ulcer Guidelines. 2010. <http://www.aawconline.org/professional-resources/resources/>. Accessed June 26, 2013.
12. Vascular Disease Foundation. Vascular Disease Statistics. Updated May 4, 2012. vascular-disease.org/education-prevention/knowledge-is-power/vascular-disease-statistics/. Accessed June 26, 2013.
13. McGuckin M, Kerstein M. Venous ulcers and family physicians. *Adv Skin Wound Care* 1998;11:344-6