Ulcers and Wound Healing of Venous Stasis Ulcers

by Robert C. Kiser, DO, MSPH

Human skin is messy. Epidermis, from microscopic cells to macroscopic flakes are constantly being shed and replenished from lower layers. Furthermore, if epidermis is injured by trauma it must replenish itself to provide the protective, semi-permeable barrier against the environment that it maintains. This process requires a dynamic balance between building up of skin and shedding or breaking down skin. If the building up of skin is too exuberant, conditions such as psoriasis and Ichthyosis occur in which the skin becomes thick and scaly. When skin does not replenish and heal fast enough, or when conditions favor breakdown of skin more than growth of new skin, ulcers develop.

Types of Ulcers: Mechanical Pressure

Ulcers may be caused by many different factors, or several factors acting in concert. Pressure ulcers, decubitus ulcers, or “bed sores” occur when constant pressure and/or shear forces are exerted on tissue, usually overlying a bony prominence, over a prolonged period of time. Curiously, although decubitus ulcers have been known of for thousands of years, the exact pathophysiology has not been elucidated. It is believed that the mechanical forces change interstitial pressures and pressure gradients, reduce capillary exchange, and create an environment in which tissue necrosis is favored over tissue healing.

Malignancy

Skin cancer can manifest as erosive non-healing ulcers.

Systemic Diseases

Numerous systemic diseases are associated with cutaneous ulcers, including diabetes, renal disease, lupus and inflammatory bowel diseases.

Ulcers of Venous Insufficiency or Venous Stasis Ulcers

Venous stasis ulcers will be the topic of the rest of this article. Ulcers are the end-stage of venous insufficiency. The region most commonly affected is the “gaiter region” – the area just above the ankle, most commonly the medial, but sometimes the lateral malleolus. Venous insufficiency occurs when the valves within the veins no longer function properly. Valves within veins assist venous blood to go up the leg against gravity. When these valves no longer function properly, blood flows back down the lower extremity and creates a gravity-dependent,

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An inflammatory reaction is triggered by the sclerosant with the intention of secondary to superficial venous incompetence. A needle is inserted into the larger-caliber varicose veins, as well as for chronic venous insufficiency. STS or polidocanol to 1.5-2.0 ml of air. The accepted protocol is to produce foam. The sclerosant is mixed with air by oscillating the two between a three-way valve to prepare foam. The sclerosant may be used for ultrasound-guided sclerotherapy. Tessari used disposable syringes and a three-way valve to prepare foam. The sclerosant is mixed with air by oscillating the two between the two syringes (20 cycles). This method can be used to produce larger quantities of foam from a smaller amount of sclerosant and is stable for 2 minutes after mixing stops. Sclerosants used to prepare foam include sodium tetradecyl sulphate (STS) 1–3% and polidocanol 0.5–3%. Although polidocanol is about half as potent as STS, both may cause ulceration if injected into soft tissues. 1% Polidocanol is typically mixed with air to create foam suitable for treating superficial varicose veins, and 0.5% Polidocanol is typically mixed with air to create foam suitable for treating superficial reticular veins. When mixed 1:3 or 1:4 with air, low concentrations of polidocanol (0.5%) produce better-performing foam. A higher concentration of polidocanol or STS is recommended for larger tributary and perforator vessels. STS can also be used to treat superficial varicose veins (1% solution) and reticular veins (0.2% solution) when mixed with air to produce foam. The accepted protocol is to use 0.5 ml of STS or polidocanol to 1.5-2.0 ml of air. Foam sclerotherapy is suitable for the treatment of any type of superficial venous incompetence and should be considered for all categories of superficial venous disease ranging from telangiectases and reticular veins to larger-caliber varicose veins, as well as for chronic venous insufficiency secondary to superficial venous incompetence. A needle is inserted into the affected vein or veins and is followed by the injection of sclerosant foam. An inflammatory reaction is triggered by the sclerosant with the intention of producing vein occlusion. Duplex ultrasound imaging has increasingly been incorporated into the procedure in order to locate the incompetent superficial vein to be cannulated, to guide cannulation, and to monitor the injection and flow of foam. This also minimizes the risk of foam diffusion to the deep venous system. More than one injection may be given during the same session in an attempt to ensure that all of the target veins have been completely filled. It is common to apply compression following foam sclerotherapy although scientific evidence to support this practice is not available and the duration for which it should be applied has not been established. Some authors use bandages and others use stockings alone. The aim of post-procedure compression is used to flatten the vein and avoid retained blood, while increasing the flow of blood up the leg. Many believe it is also essential to reducing the risk for thrombophlebitis. As with earlier practices in sclerotherapy, immediate ambulation and return to work are encouraged. There is little need for time away from work. The most generally accepted contraindications for foam sclerotherapy are similar to those for conventional liquid sclerotherapy, such as allergy to the sclerosant, history of acute superficial or deep vein thrombosis, or hypercoagulopathies. Potential adverse events associated with foam sclerotherapy include allergy to a sclerosant, thrombophlebitis (most common), staining of the skin and a palpable, fibrosed vein (often seen in thin patients with larger-caliber superficial vessels). Other risks include potential nerve damage, cutaneous necrosis, ulceration and matting in cases of sclerosant extravasation. There are concerns that the sclerosant can potentially enter the deep veins and induce venous thromboembolism; the risk is higher in obese patients where post-procedure wrapping is less effective and post-procedure activity is less likely. Theoretically, in people with a patent foramen ovale (PFO), the sclerosant may enter the arterial circulation and induce an ischemic stroke or retinal artery occlusion. Also, if foam is inadvertently injected directly into the artery, loss of limb is possible. Transient visual disturbances and precipitation of migraine headaches are reported following foam sclerotherapy. The mechanism is uncertain but is felt to be vasospastic. Coughing is also a rare but reported side-effect; however, it is usually transient. The recurrence rate following this treatment as compared to surgery has yet to be firmly established. Ultrasound imaging studies suggest that 75 – 90% of veins treated in this way remain occluded after 3 years although more than one treatment may be required to achieve these results. Foam sclerotherapy offers an adjunct to surgical intervention for patients with tortuous tributaries, superficial varices, small vascular networks which are not amenable to current surgical techniques, and reflux in vessels where health insurance companies will not approve surgical ablation techniques. Foam sclerotherapy is FDA approved and can be conducted on an outpatient basis often with no anesthesia required. This technique promises to be a useful addition to the methods currently employed for managing superficial venous incompetence. 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gradient pressure within the venous system. This leads to elevated pressures within the veins, all the way back to the capillary networks where oxygen from arterial blood is exchanged with tissues, and deoxygenated venous blood full of metabolic byproducts of cellular respiration is collected. These “dammed-back” capillaries undergo changes such as fibrin cuffing, which further diminish nutrient exchange. The befouled environment leads to changes in the surrounding tissues including the deposition of collagen in the skin and adipose tissue.

Epidemiology

The point prevalence of venous stasis ulceration western nations is estimated to be from 0.02 to 1 percent.1 Risk factors include age, obesity, history of leg injury and history of venous thromboembolism (VTE). Venous stasis ulcers were responsible for 2 million work days lost in 2002.2

Symptoms and Presentation

Venous stasis ulcers present with areas of poorly healing skin wounds, generally of the medial malleolus, red-based or exudative, with local skin necrosis and irregular borders. Frequently the surrounding tissue has other signs of venous insufficiency, such as hyperpigmentation, pitting or woody edema, excess collagen deposition and fibrosis. The patient will frequently complain of factors that limit quality of life, including pain, odor, exudation, spontaneous bleeding and alteration of lifestyle (such as no longer being able to swim or wear shorts). Without proper treatment, venous stasis ulcers can take many months to heal. Patients may suffer with their ulcers for months or even many years without bringing the condition to the attention of their physician. Often there is some provoking trauma such as a scrape or bump, and barring such trauma patients will often complain that that they have been bitten by a “spider.” Frequently the patient self-diagnoses the ulcer as a “brown-recluse bite,” even in areas that brown recluse spiders are extremely uncommon. Like proteolytic spider bites, ulcers take a long time to heal and heal with scarring. Unlike such bites, ulcers tend to recur after healing if appropriate measures are not taken to treat the underlying venous insufficiency causing the ulcer.

Conservative Treatment

The mainstay of treatment for venous stasis ulcers is compression. Compression can be provided by dressings, single or multilayered bandaging, graduated compression stockings, or Velcro-strap devices. Compression may be elastic or inelastic. The most common form of single-layer bandage is the zinc-paste bandage created by Paul Gerson Unna (1850–1929). Unna’s boots have numerous formulations, but the principle components are a cotton bandage impregnated with zinc paste, glycerin and sometimes calamine. Gelatin and other chemicals sometimes are used to emulsify and spread the zinc paste. The application of the boot requires hands-on training to perform effectively. Principles include starting at the metatarsal bases and wrapping to the tibial tubercle and applying with sufficient firmness in an even, contour-forming pressure to avoid gauging the skin. The boot is left in place for 7-10 days and then changed—although earlier changes can be necessary for very exudative wounds or if there is sufficient edema that the bandage becomes loose. Unna’s boot has many advantages, including being an inelastic compression that provides compression during ambulation, but is non-compressive when the patient is recumbent and not ambulating.

Multilayer bandages may include layers of sponge-foam, cotton-wool, crepe, elastic-stretch bandages, and Coban-type self-adherent bandages. The advantages of the multilayer bandages include the ability to absorb moisture and exudate, the increased circumference the wadding or foam layer provides allows for decreased point pressure while maintaining adequate circumferential pressure to the limb, the ability to apply granulation favoring or antimicrobial dressings directly to the wound. The disadvantages of the multilayer dressings include cost and the bulk of the dressing.

Debridement

Another aspect of wound care that is important is debridement. Debridement of necrotic debris should be provided to encourage growth of granulation tissue. Debridement can be mechanical (sharp or blunt) or chemical. Several products are available for chemical debridement, including balms of Peru-castor oil mixes (such asGranules) and collagenase (Santyl). One of the most effective and safe chemical debridement creams, in the author’s opinion and experience, was papain-urea cream (Accuzyme). Unfortunately, the Food and Drug Administration took Accuzyme off the market several years ago.3

Antimicrobials

Antibiotics are seldom useful in the treatment of venous stasis ulcers unless signs of infection are present. These signs include increase in pain beyond the baseline, increasing erythema of the skin surrounding the ulcer, and lymphangitis, a rapid increase in the size of the ulcer.4 Antibiotics are frequently overprescribed for venous stasis ulcers on the basis of necrosis and exudate. Unfortunately, the necrosis of ulcerating skin is generally not due to bacteria, but due to autolysis of the skin cells, and therefore antimicrobials do not improve the situation, and may increase the potential for super-infection with resistant organisms.5

Phlebological Treatment

The underlying cause of venous stasis ulceration is venous stasis. Using modern techniques such as radiofrequency ablation, endovenous laser ablation, and ultrasound-guided sclerotherapy the root cause of the imbalance in healing and breaking down skin tissue can be corrected, and balance restored. By addressing and correcting the increased pressure in the superficial system, but ablating refluxing axial veins or perforating veins, the phlebologist not only can help to heal the venous stasis ulcer, but can prevent chronic recurrence. Older techniques such as vein stripping and ligation and the Linton procedure for perforator closure were sometimes avoided while the ulcer was open, as these techniques utilized incisions through or near already compromised skin. With more modern, minimally invasive techniques, the points of high superficial venous pressure can be addressed with 2mm or smaller incisions, and the balance of healing versus lysis of skin restored. By addressing the underlying cause of the ulcer, the phlebologist, working with the primary physician and/or wound healing center, can help to provide long-term wound healing for the venous stasis ulcer.

References:

Q: “What will my patient’s insurance cover?”

A: Insurance coverage for varicose vein treatment varies depending on your patient’s insurance provider and your patient’s specific policy. In general, most insurance providers separate vein treatments into two categories, Medically Necessary Procedures and Cosmetic Procedures. Larger veins that are symptomatic (pain, aching, burning, itching and/or swelling along with additional criteria set by your patient’s insurance) are usually considered medically necessary and are covered by your patient’s insurance. After documentation of venous insufficiency (the valves in your patient’s legs are not properly functioning), which is determined by an ultrasound, most insurance providers consider in-office procedures to be medically necessary. That includes radiofrequency or laser ablation, ultrasound-guided foam sclerotherapy, and ambulatory phlebectomy.

Appearance-related procedures, however, are usually not covered by insurance. For example sclerotherapy for small, non-symptomatic spider veins (telangiectasia) are not considered medically necessary and, therefore, patients would have to pay out of pocket.

At Center for Vein Restoration, we know that the insurance process can sometimes be very confusing to patients. Our Patient Services team will submit your patient’s claim and acquire pre-certification for your patient’s procedure, when deemed necessary by your patient’s insurance provider. However, it is best to call your patient’s insurance company to find out the details of his or her policy.

Meet Our New Physicians

We’re proud to announce the addition of three new physicians to our team: Richard Nguyen, MD; Vinay Satwah, DO, FACOI; and Khoa Q. Tran, MD:

Richard Nguyen, MD is board certified in emergency medicine. He also is an attending physician, CEP in the Department of Emergency Medicine of the Greater Baltimore Medical Center in Towson, MD. He is a former attending physician, SSEP, Department of Emergency Medicine, Holy Cross Hospital, Silver Spring, MD and was a resident in the Department of Emergency Medicine at the University of Maryland Medical System in Baltimore, MD.

Vinay Satwah, DO, FACOI is Board certified in internal medicine and general cardiology. He is a former hospitalist with St. Peter’s University Hospital in New Brunswick, NJ, and a former clinical instructor at Drexel University School of Medicine. He is a member of the American College of Phlebology, American Osteopathic Association, and the American College of Cardiology, and serves as a cardiology fellows’ liaison for the American Heart Association.

Khoa Q. Tran, MD is Board certified in internal medicine and general cardiology. His is a former hospitalist, with INOVA Loudoun Hospital, Leesburg, VA. He also was formerly an academic hospitalist and teaching faculty member in the Department of Internal Medicine at Lankenau Hospital in Wynnewood, PA. He is a member of the Society of Hospital Medicine, the American College of Physicians, and the American Society for Laser Medicine & Surgery.

New CMEs Announced

We are pleased to announce more CME sessions for summer 2012 on venous insufficiency. Each course is valued at 3 CME credits. Details are below; to learn more or to request a CME in a region we serve, please contact Brent Matherly at 443-370-3830 or 301-860-0930 and at brent.matherly@centerforvein.com.

CME Courses & Speaking Engagements

June

Maryland

Easton
Thursday, June 14
Easton, MD 21601
RSVP by June 7
Nina Firmani, Physician Liaison
Nina.firmani@centerforvein.com
(443) 534-1584

Glen Burnie
Thursday, June 21
Ellicott City, MD 21043
RSVP by June 14
Chelsea Mahoney, Physician Liaison
Chelsea.mahoney@centerforvein.com
(410) 310-1934

Virginia

Greenbelt
Thursday, June 27
Pane e Vino
9020 Lorton Station Boulevard
Lorton, VA 22079
RSVP by June 17
Daniel Morgan, Physician Liaison
Daniel.morgan@centerforvein.com
(703) 909-3535

Our CMEs will be on a summer break, but will resume in September. Visit www.centerforvein.com and go to the Physician’s Corner for the latest CME information.

Would you like us to offer a CME course near you? Please contact Brent Matherly at 443-370-3830 or 301-860-0930 and at brent.matherly@centerforvein.com.
New Centers Open in DC & VA

We’re excited to announce the addition of two new locations. The new centers bring to 19 the number of clinics serving patients in Maryland, Northern Virginia, the District of Columbia and Western Michigan.

Details of our newest centers are below.

• **District of Columbia**
  Now our second location in the District of Columbia, our newly opened clinic is located at 3301 New Mexico Ave. NW. You can reach our team there at (800) 349-5347.

• **Leesburg, Virginia**
  Our Leesburg, Virginia clinic is located at 44035 Riverside Parkway, Suite 400. You can reach our team there at (800) 349-5347.

**Leesburg, Virginia**
Detailed information on all local Centers can be found on our web site, www.centerforvein.com. Patients can be referred easily via our “Doctors Referral” button on the Web site home page or by phoning our one-stop call center at 800-FIX-LEGS.
Summer for many people is synonymous with vacation—getting away from it all to enjoy family and friends backyard barbecues and time at the beach. But as your patients may tell you, many people forego travel—or even socializing at home—because of their painful or embarrassing varicose and spider veins.

Summer also happens to be the name of an Olympic swimmer—Summer Sanders—who recently started campaigning for more awareness of varicose vein treatment options. Her life story—including rising to the peak of athletic performance—helps dispel myths of about venous insufficiency, such as the one that says only sedentary or old people end up with this condition. The opposite is true; despite lifestyle factors that are contributors, the greatest factor still is heredity.

We are dedicated to helping patients like yours not only regain optimal venous function but also recapture the quality of life that’s been eluding them, whether they’re retirees, moms, or elite athletes. And we’re proud to announce that we’re able to treat even more patients with the opening of our 18th and 19th clinics in Leesburg, Virginia and Washington, D.C.

We’re also pleased to continue to offer our CME courses on venous insufficiency for you and your colleagues; summer dates are listed in this newsletter. We hope you can join us for one of these 3-hour sessions, which each carry 3 CME credits.

On behalf of everyone at Center for Vein Restoration, thank you for your continued interest and we wish you a happy and healthy summer.

Regards,
Robert C. Kiser, DO, MSPH
Editor