The Importance of Patient Transition When Managing Ulcerations Related to Venous Disease

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Twenty thousand newly diagnosed venous leg ulcers (VLUs) occur every year[1] with 0.5%[2] of the adult population in the U.S. having an active open wound daily. Patients will often present with a VLU to a provider who does not specialize in wound management. A lack of understanding related to the complexities of wound management in this patient population may lead to suboptimal outcomes.

The toll of VLUs is costly to the patient due to altered or loss of employment coupled with medical costs, strained family relationships, reduced socialization, and sequelae often related to pain management that includes constipation, falls, altered sleep patterns, and mentation changes.[3]

Costs for the management of VLUs can range from $150 million[4] to $3 billion[5] annually in the U.S. Individual episode expenditures vary by setting, though the literature is sparse in the skilled nursing facility/short term rehabilitation arena. It is no surprise that VLUs associated with wound infection add to this (**Table 1**). Also of note, patients with congestive heart failure are more likely to have a VLU-associated wound infection.[4]

A typical transitional scenario involves an acute hospitalization for a venous ulceration, complicated by cellulitis. The patient is usually managed by the hospitalist and local dressings are applied. The patient is then either discharged to a short-term rehabilitation setting or managed by a home health agency.

The use of compression, the mainstay of treatment, is not well established. While a doppler ultrasound to rule out deep vein thrombosis is often done in this setting, it is rare to see vascular studies using an Ankle-Brachial Index, that can help determine the amount of appropriate compression the patient will need to assist in their wound healing.

Once the patient transitions to the new setting, a new provider assumes care. In the home setting, the primary care provider (PCP) is typically managing the patient with the home health care agency performing active wound care. If the patient is also followed at a local wound center, the home health nurse must take these recommendations, send them to the PCP who then approves, modifies, or rejects the recommendations. Each scenario is heterogeneous and may delay care as compression is not often initiated at the time of hospital discharge. Organizational formularies may prevent optimal compression or topical wound management choices. Additionally, provider skill, staff turnover, and a variety of payor reimbursement requirements add layers of complexity for patients with multiple comorbidities. The scenario is similar for patients transitioning from a short-term rehabilitation facility setting to a home health setting with a significant impact on the patient's care plan. In my clinical experience, the key to transitioning lies in understanding disease etiology, the transition setting strengths and weaknesses, and patient engagement.

Setting	Infection (n=9)	No Infection (n=69)	P
Total costs	$27,408 ± $10,859	$11,088 ± $9,343	<.0001
Inpatient costs	$9,492 ± $8,328	$255 ± $1,438	<.0001
Outpatient wound center costs	$7,961 ± $9,575	$6,176 ± $8,397	.56
Home Health costs	$9,956 ± $7,650	$4,657 ± $45,486	.01

Table 1. VLU Cost by Setting and Presence or Absence of Infection; Adapted from: Melikian, et al.[4]

UNDERSTANDING DISEASE ETIOLOGY

Venous disease severity is categorized by the CEAP (Clinical Manifestations, Etiology, Anatomic Distributions and Pathophysiology) system (**Table 2**) and clinical practice guidelines for management outlined by the Society of Vascular Surgery® and the American Venous Forum, which provide a structure to manage patients.

CEAP Classification	Clinical Manifestations
C0	No visible signs of venous disease
C1	Telangiectasias or reticular veins
C2	Varicosities ≥3 mm
C3	Edema
C4a	Skin pigmentation changes
C4b	Lipodermatosclerosis
C5	Healed venous ulceration
C6	Active venous ulcer

Table 2. CEAP Classifications of Venous Disease[1]

In humans, venous return is accomplished by a system of superficial veins connected to the deep veins by perforator veins. Through obstructive or reflexive mechanisms, weakness develops in the vein wall that results in venous disease. Varicosities typically form in the greater and lesser saphenous veins. Obstruction of the iliac veins or inferior vena cava can result in extensive varicose veins.[5]

Venous hypertension, venous valvular incompetence, and structural changes in the vein wall with associated inflammation are the primary pathophysiological mechanisms causing venous disease. Venous reflux disease is caused by venous valvular incompetence, venous outflow obstruction, or calf-muscle pump failure. Valvular incompetence occurs in either or both the superficial or deep venous system and results in venous hypertension below the affected valve. In patients with perforator vein incompetence, high pressure generated in the deep veins during calf muscle contraction is transmitted to the superficial system. Valvular incompetence results from structural changes of the valve leaflets. A combination of microvascular and macrovascular events culminates into visible ulcerations, though the precise pathophysiological process has not been specifically elucidated.[5]

The vein wall weakening, and subsequent dilatation contributes to disease progression. On the microvascular side, histological studies have demonstrated disrupted smooth muscle cell and elastin arrangement, overproduction of collagen Type I with concomitant reduction of collagen Type III and abnormal deposition of fibroblast and transforming growth factors β within the vein wall. Overproduction of collagen Type I decreased synthesis of collagen Type III, and disruption of the arrangement of smooth muscle cells and elastin fibers have been observed in histological studies of varicose venous segments.[2] Increased levels of tissue inhibitors of matrix metalloproteinases observed in varicose vein specimens may favor the deposition of extracellular matrix material in the vein wall. Increased levels of transforming growth factor β1 and fibroblast growth factor β have also been observed in the walls of varicose veins and may contribute to structural degradation.[6]

When dilatation occurs, the stretched vein leaks fibrinogen, growth factors and white blood cells into the surrounding interstitial space, subcutaneous tissue, and dermis. Pathophysiological hypotheses suggest that the formation of fibrin cuffs surround the vessels leading to the loss of oxygen and nutrient diffusion, an inflammatory cascade is initiated with the trapping of leukocytes in the area, and growth factors imperative for healing are absent, as they are physically bound within the exudate contents that have escaped from the dilated vein.[7] Loss of the calf muscle pump contributes to venous engorgement, as calf muscle dysfunction affects venous function. Absent or reduced calf muscle strength alters venous return hemodynamics causing blood to pool in the lower extremity and adding to capillary leak and subsequent edema.[8]

As in the management of any wound, eliminating the underlying cause is critical in achieving successful outcomes. Determining the root cause – obstruction, reflux, or both - is of utmost importance along with evaluation of the calf muscle pump. Risk factors (**Table 3**), modifiable and non-modifiable, combined with the physical signs commonly associated with venous insufficiency (**Table 4**) are first determined followed by diagnostic testing (**Table 5**).

Modifiable Risk Factors	Non-Modifiable Risk Factors
Obesity	History of Deep Vein Thrombosis
Smoking	Pregnancy
Sedentary lifestyle	Female Sex
Constipation	Factor V Leiden Mutation
Hypertension	Advancing Age
Compliance with compression	Family history of venous disease
Medications associated with lower extremity edema as a side -effect	History of trauma to lower extremity

Table 3. Risk Factors Associated with Venous Disease; courtesy of Catherine T. Milne, MSN, APRN, ANP/ACNS-BC, CWOCN-AP

CLINICAL FINDINGS ASSOCIATED WITH ADVANCING VENOUS DISEASE

- Hemosiderin staining of leg
- Edema – often "woody" in nature
- Atrophie blanche
- Ulceration
- Stasis dermatitis

Table 4. Common Clinical Findings with Venous Insufficiency; courtesy of Catherine T. Milne, MSN, APRN, ANP/ACNS-BC, CWOCN-AP

Duplex/Reflux Studies
Plethysmography
Venography
Intravascular ultrasound
CT
MRI

Table 5. Advanced Diagnostic Testing Employed to Determine Obstructive and Reflux Venous Disease; CT= Computerized tomography; MRI= magnetic resonance imaging; courtesy of Catherine T. Milne, MSN, APRN, ANP/ACNS-BC, CWOCN-AP

While waiting for test results, a simple vascular exam with an ankle-brachial index and toe pressure, if available, can assist to determine the amount of compression applied at the first visit. It is important to note that even in a patient with established arterial disease, edema management must be addressed, as edema alone will impair arterial function. Mild compression in this subset of patients is recommended.[9] Referring the patient to a skilled vascular surgeon who can correct the underlying obstructive or reflexive disorder should be considered. The wound specialist should manage the highly exudative and often inflamed wound coordinating care with direct caregivers in the setting that the patient spends most of their time in order to optimize outcomes.

IDENTIFYING STRENGTHS AND WEAKNESSES IN THE TRANSITION SETTING

Direct communication with caregivers helps clarify patient goals, identify care issues, and allows for providers to expediently alter the plan of care as needed to optimize outcomes. Common barriers include a lack of knowledge in the application of multilayered compression, the substitution of a multilayered compression wrap to a zinc-based impregnated gauze wrap (Unna Boot), knowledge about wound exudate and periwound skin management, and an underappreciation of bioburden and the role of inflammation in wound healing. Identifying key caregivers and presenting information in a collaborative manner can help promote knowledge transfer thereby allowing the wound provider to learn about organizational barriers in the transitioned setting that may prevent optimal outcomes. Ongoing and frequent conversations with caregivers improve team dynamics and often serve as a "safety huddle".[10]

CASE STUDY #1:

An 89-year old female in a skilled nursing facility with a positive family history for venous disease, long-standing bilateral leg edema with hemosiderin deposition and stasis dermatitis, sustained a traumatic wound when transferring to her wheelchair (**Figures 1-3**). Multiple therapies were attempted but wound healing ultimately failed. The in-house nurse practitioner asked for a wound consultation.

Case Description

Day 1 – An ankle Brachial index of 1.4 indicated calcified vessels and +2 edema was also noted. The wound bed was pink and clean but not granulating. The patient's drainage was serous in nature in mild to moderate amounts. The staff reported that the patient was often seen scratching her legs. The patient's Power of Attorney for Healthcare Decisions (POA) refused diagnostic testing due to the patient's severe dementia and combative behavior. After wound cleansing, a mid-potency topical steroid was applied to the periwound skin to reduce stasis dermatitis and its associated symptoms. A primary dressing of 3M™ Promogran Prisma™ Collagen Matrix with ORC and Silver was applied and secured with a secondary dressing (3M™ Tegaderm™ Silicone Foam Border Dressing) followed by elastic tubular compression to reduce edema. Promogran Prisma Matrix was reapplied 3 times a week along with a new Tegaderm Silicone Foam Border Dressing.

Day 14 – The edema and wound size was reduced, and the nursing staff were able to obtain and apply a topical steroid, Promogran Prisma Matrix, Tegaderm Silicone Foam Border Dressing, and elastic tubular compression without difficulty. Nursing assistants reported markedly reduced pruritus.

Day 28 – Wound closure is achieved. A physical therapist who was added to the team suggested obtaining an adjustable compression wrap to facilitate compliance with compression application. The nursing home administrator approved the purchase.

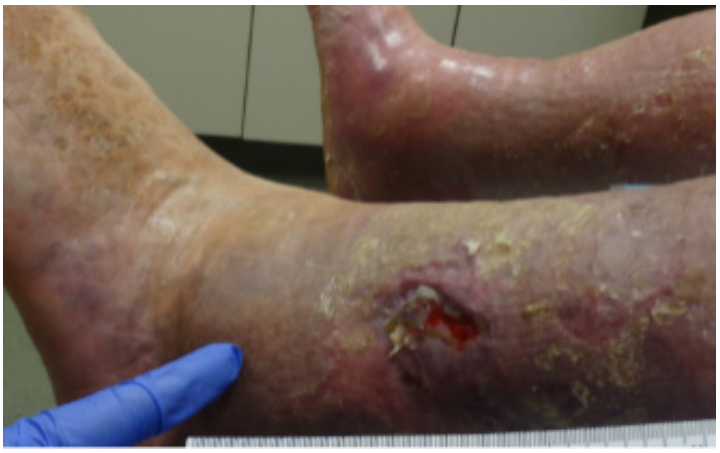

Figure 1. Traumatic wound at presentation; patient photo courtesy of Catherine T. Milne,MSN, APRN, ANP/ACNS-BC, CWOCN-AP

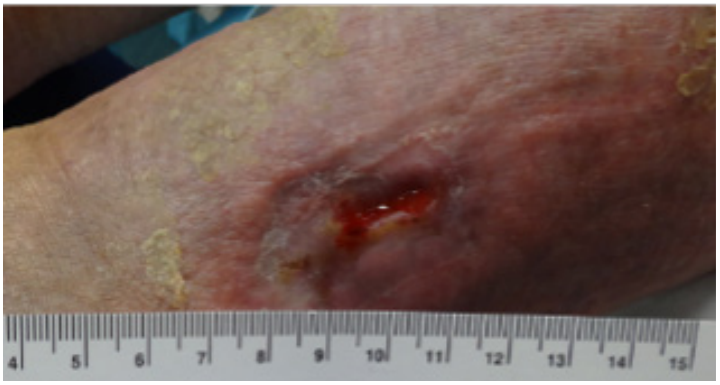

Figure 2. Wound after 14 days of wound management with 3M™ Promogran Prisma™ Collagen Matrix with ORC and Silver, 3M™ Tegaderm™ Silicone Foam Border Dressing, and elastic tubular compression; patient photo courtesy of Catherine T. Milne, MSN, APRN, ANP/ACNS-BC, CWOCN-AP

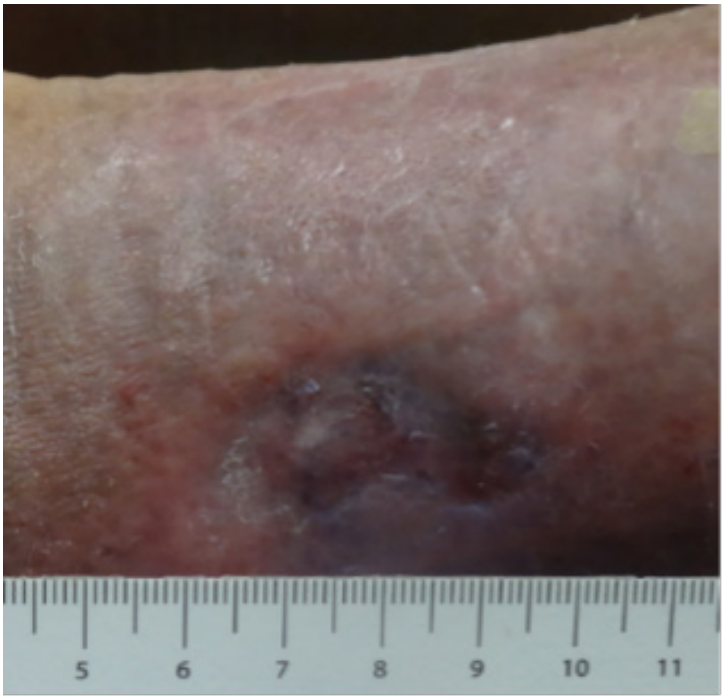

Figure 3. Wound closed after 28 days of wound management with 3M™ Promogran Prisma™ Collagen Matrix with ORC and Silver, 3M™ Tegaderm™ Silicone Foam Border Dressing, and elastic tubular compression; the patient was transitioned to an adjustable compression wrap to manage edema; patient photo courtesy of Catherine T. Milne, MSN, APRN, ANP/ACNS-BC, CWOCN-AP

PATIENT ENGAGEMENT

VLUs are associated with recurrence rates of 60%-70% over a ten-year period[11] making it critical that providers actively engage the patient in the management of their disease at the initial visit. Explaining the pathophysiology, and treatment strategies while communicating that the chronicity of the disease is manageable over the course of the patient's lifetime is imperative. Assessing the patient's ability for engagement is the first step and is well outlined in the literature.[12] While patients vary in their ability to participate in the care of their VLU, this should not result in those caring for the patient to discontinue encouragement and education about ongoing disease management. By asking patients to articulate perceived barriers in disease management, and specifically addressing these issues, one can nurture the patient's involvement in their own care.

CASE STUDY #2:

A 69-year-old morbidly obese male with a 10-month history of venous disease presented at our facility (**Figures 4-6**). The patient had been managed by his primary care provider (PCP) in his home with the assistance of a home health nurse, who changed his dressings three times per week with a calcium alginate and gauze wrap. No compression was used. The patient reported his biggest problem was that he wet his socks and bed sheets and could not wear shoes. The patient also reported that he sat in his kitchen chair all day and did not elevate his legs because he was concerned that he might ruin his furniture. An ankle brachial index of 0.99 with triphasic wave forms was noted. The patient did not wish to pursue diagnostic testing due to stigma associated with his symptomology.

Case Description

Day 1 — Local wound care with wound cleansing, mechanical debridement, periwound skin protectant (3M™ Cavilon™ Advanced Skin Protectant) was applied followed by application of 3M™ Kerramax Care™ Super-Absorbent Dressing and 3M™ Coban™ 2 Two-Layer Compression System. Dressings were changed every other day for one week then every 3 days until day 32. Communication with the home health nurse caring for the patient and his PCP led to a change in the orders for the home health agency. As compression therapy was utilized, the home health agency changed the nursing staff for the patient to one with proficiency in application of a multilayer compression wrap.

Day 14 — Reduced edema and drainage was noted along with overall wound improvement. Early patient engagement was seen as the patient agreed to try to elevate his legs. The situation was discussed with the home health nurse who reported that the patient's couch and recliner chair was covered with magazines and other materials, preventing him from elevating effectively. The home health nurse agreed to contact the patient's niece to clear debris from furniture and made a "contract" with patient for leg elevation.

Day 32 — Both the home health nurse and the patient report increased frequency of elevation of legs. The wound drainage significantly decreased, and the wound continued to reduce in size. The Kerramax Care Dressing was changed to Promogran Prisma Matrix and Tegaderm Silicone Foam Border Dressing. Dressings were reapplied or changed 3 times a week. Compression using the Coban 2 Compression System was continued. The patient agreed to vascular surgeon referral for testing to determine correctable venous structural issues.

Day 48 — The patient began venous ablation procedures with the vascular surgeon. Transition compression garments were ordered after discussion with the vascular surgeon to determine amount of compression anticipated to continue healing and prevent recurrence.

Day 56 — The wound resolved, and wound care was discontinued. The patient continued to use the Coban 2 Compression System while ablation procedures continued.

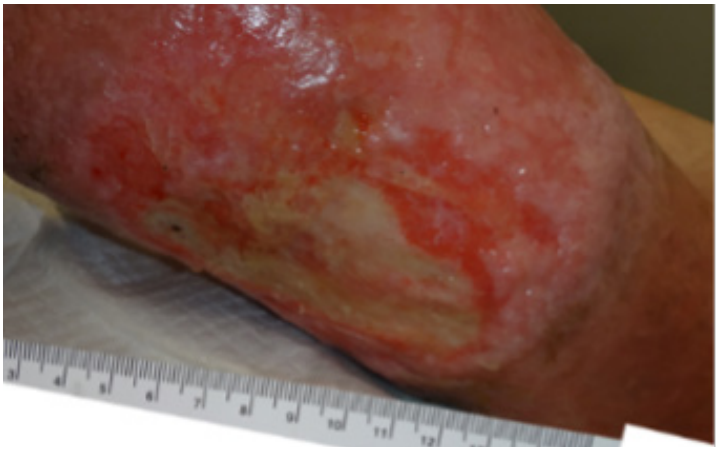

Figure 4. Wound at presentation; patient photo courtesy of Catherine T. Milne, MSN, APRN, ANP/ACNS-BC, CWOCN-AP

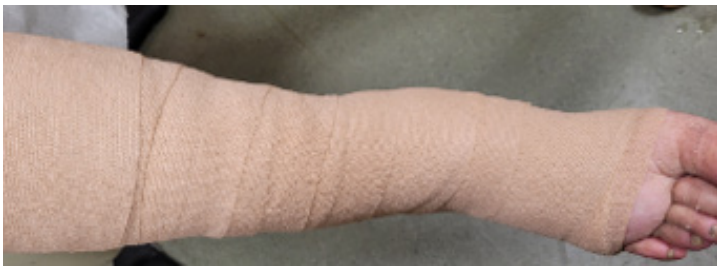

Figure 5. Application of 3M™ Coban™ 2 Two-Layer Compression System; patient photo courtesy of Catherine T. Milne, MSN, APRN, ANP/ACNS-BC, CWOCN-AP

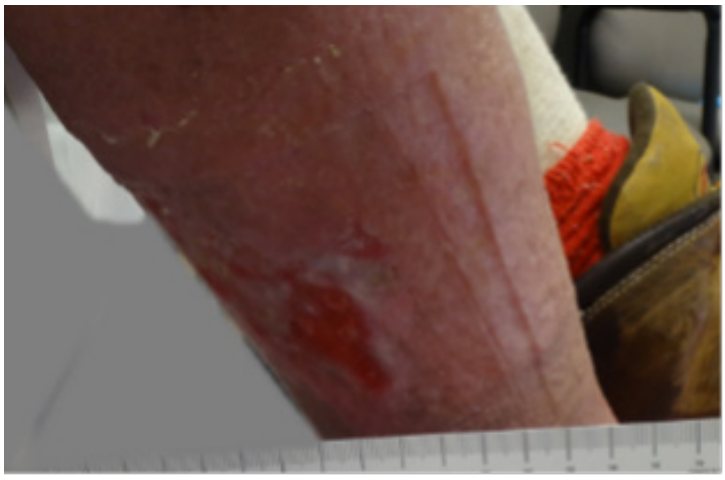

Figure 6. Reduced wound size after 32 days of wound management; patient photo courtesy of Catherine T. Milne, MSN, APRN, ANP/ACNS-BC, CWOCN-AP

SUMMARY

VLUs are a challenging wound management issue. Correcting the pathophysiological abnormality, when possible, in combination with the use of an evidence-based local wound management and compression strategy, as well as an appreciation of the strengths and constraints of the management setting can help optimize patient outcomes.

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