Chronic venous disease and venous leg ulcers: An evidence-based update

Teresa J. Kelechi, PhD, RN, CWCN, FAAN, Jan J. Johnson, MSN, RN, ANP-BC, CWOCN, and Stephanie Yates, MSN, RN, ANP-BC, CWOCN

Chronic venous disease (CVD) is a complex chronic vascular condition with multifaceted primary and secondary etiologies leading to structural and functional changes in veins and valves and blood flow of the lower legs. As a consequence, a spectrum of clinical manifestations arise, ranging from symptoms of mild leg heaviness and achiness to debilitating pain, and signs of skin changes, such as eczema and hemosiderosis, to nonhealing, heavily draining venous leg ulcers (VLUs). Triggers such as trauma to the skin are responsible for a large majority of VLU recurrences. Diagnostic testing for venous reflux includes ultrasound imaging; unfortunately, there are no diagnostic tests to predict VLUs. The hallmark of treatment of both CVD and VLUs is compression. Leg elevation, exercise, and wound management with dressings and advanced healing technologies that provide an environment conducive to healing should focus on reducing pain, necrotic debris, drainage, and odor, as well as preventing infection. VLUs that become chronic without evidence of healing over a 4-week period respond best to multidisciplinary wound experts within a framework of patient-centered care. Nurses are in key positions to provide early recognition of the signs and symptoms as well as initiate prompt diagnostic and promote early treatment to offset the progression of the disease and improve quality of life. (J Vasc Nurs 2015;33:36-46)

Chronic venous disease (CVD) is a vascular condition characterized by a myriad of symptoms ranging from lower leg swelling and skin redness to venous leg ulcers (VLUs). The disease can become chronic and progressively debilitating. Complaints of leg achiness, pain, and heaviness are common. Affecting millions of individuals worldwide, CVD remains underdiagnosed, misunderstood, and disregarded as posing significant health threats.[1] The purpose of this evidence-based article is to increase awareness of the signs and symptoms, diagnostic tests, and classification criteria of CVD, as well as VLU treatment, including primary and secondary prevention strategies. Early recognition and prompt treatment can alleviate and/or prevent the physical, functional, and psychological complications of this chronic vascular disorder.

SIGNIFICANCE

Decades of investigation into CVD etiology and risk factors and treatments for VLUs[2,3] have resulted in numerous consensus statements and evidence-based guidelines[3–5]; however, the overall management remains suboptimal.[6] Unfortunately, individuals with CVD and VLUs experience negative functional and psychological consequences, which lead to low physical activity,[7] increased risk for other comorbid conditions (such as obesity,[8] anxiety and depression,[9] social isolation[10]), and poor quality of life.[11] The economic burden is enormous. In 2008, the National Institute of General Medicine Sciences reported that between $20 and $25 billion was spent for ulcer care, not accounting for costs associated with lost wages and human suffering. As the population ages, the incidence of new VLUs is expected to far surpass the 600,000 VLUs that develop each year in the United States.[12] Similarly, the prevalence rate for VLUs that affect 6.5 million individuals is expected to increase requiring more intensive screening and treatment to prevent ulceration.[1]

From the College of Nursing, Medical University of South Carolina, Charleston, South Carolina; Wound Management Center, Duke University Medical Center, Durham, North Carolina; Duke Cancer Center, Duke University Medical Center, Durham, North Carolina.

Corresponding author: Teresa J. Kelechi, PhD, RN, CWCN, FAAN, College of Nursing, Medical University of South Carolina, 99 Jonathan Lucas Street, MSC 160, Charleston, SC 29425-1600. Tel.: +1 843-792-4602, 843-810-3157(cell); fax: +1 843-792-2104. (E-mail: kelechtj@musc.edu).

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PATHOPHYSIOLOGY

The most common etiologic factors associated with CVD include hypertension of the venous system of the lower legs, incompetent perforator veins that connect the deep and superficial veins, weak valves leading to reflux or retrograde blood flow, and failure of the calf muscle pump to move deoxygenated blood from the venous system.[13] Veins can be injured as a result of inflammation, trauma, venous thromboembolism, surgery, and comorbid conditions, such as obesity. These factors lead to vein damage, venous insufficiency, and/or CVD.[14–16]

VLUs are the most common type of lower extremity ulcer, accounting for >70% of all types of ulcers, including those with an arterial or neuropathic/diabetic etiology, or a combination of etiologies.[17] Approximately 50% of VLUs are chronic, failing to proceed through an orderly reparative process for timely healing of the anatomic or functional injury.[18] Chronic VLUs are open for 4–6 weeks or more,[5] and have a tendency to become infected, malodorous, and painful. Several theories have been established

about the causes of VLUs including the development of fibrin cuffs, leukocyte entrapment, and microangiopathy.[19] Also, recent studies of pathogenic associations in VLUs have demonstrated an overabundance of activated neutrophils secreting high levels of proteases,[20] such as metalloproteinase-9, that kill growth factors.[21] These aberrancies contribute to a chronic inflammatory state that disturbs the microcirculatory blood flow and inhibits healing.[22] Several risk factors for developing CVD and VLUs have been identified (Table 1).[23,24]

CLINICAL PRESENTATION

The initial presentation of CVD varies widely. Common symptoms of CVD include heaviness or aching of the legs, especially at the end of the day; calf, thigh, or buttocks pain; swelling around the lower part of the legs; dry skin; a feeling of tightness in the lower limb; skin irritation; and itching.[25] If pain is present, it is typically reported as worsening with prolonged dependency, improving with elevation, and may be severe enough to limit ambulation.[26]

VLUs are confined generally to the lower aspect of the leg at the gaiter region, located between the malleolus and calf muscle. The ulcers tend to be large, have irregular borders, variable exudate, pain, and odor, and may present with or without fibrin, which is a yellowish material embedded between small red granulation buds of healing tissue.[27]

Classification systems

When physical signs of CVD are present, the most commonly used classification system is the CEAP classification: C (clinical presentation), E (etiologic factors), A (anatomical location), and P (pathologic process; Table 2).[28,29] Other venous classification systems such as the Charring Cross Venous Ulcer Questionnaire and the Venous Clinical Severity Score, assess risk, quality of life, function, treatment outcomes, and severity of the disease.[11] There is no classification system for VLUs or consensus on the best approach to staging them (eg, full vs partial thickness).

ASSESSMENT AND DIAGNOSTICS

The diagnosis of VLU is primarily made based on an assessment of the patient's history and the clinical examination. The assessment should determine if there is a family history of CVD and/or past or present thromboembolic conditions, and calf muscle pump dysfunction, which cause increased resistance to venous return and damage to the valves.[4] Ask the patient about a history of healed or current ulcerations, noting the location, character, duration, treatment, number of recurrences, and possible "triggers." Approximately 74% of VLUs begin with a specific trigger such as cellulitis, penetrating injury/trauma, contact dermatitis, rapid onset of leg edema, burns, dry skin with itching/scratching, and insect bites.[30] Factors identified with poor healing outcomes include having a longstanding ulcer, infection, longer topical and systemic antibiotic use, and poor adherence to compression therapy.[31] Assessment and documentation of VLUs should include measurements of the size (length, width, and depth), location on the leg, condition of the wound edges (eg, rolled down in appearance), characteristics of the wound bed (ie, slough, hypergranulation), presence of exudate, odor, and pain, and the condition of the periwound skin.[3,4]

TABLE 1

RISK FACTORS

Nonmodifiable	*Modifiable*
Older age	Physical inactivity
Diabetes	Higher body mass index
Hypertension	
Joint/skeletal disease of the legs (eg, rheumatoid arthritis)	
History of superficial and deep venous thromboembolism	
Family history of venous leg ulcers	
Deep or perforator vein reflux, deep vein obstruction, or combination of both	

TABLE 2

CLINICAL CLASSIFICATION OF CHRONIC LOWER EXTREMITY VENOUS DISEASE*

Class	*Clinical signs*
0	No visible or palpable signs of venous disease
1	Teleangiectases, reticular veins, malleolar flare
2	Varicose veins, distinguished from reticular veins by a diameter of ≥3 mm
3	Edema without skin changes
4	Skin changes ascribed to venous disease 4a – hyperpigmentation 4b – venous eczema 4c – lipodermatosclerosis 4d – atrophie blanche
5	Skin changes (as defined in class 4) in conjunction with healed ulceration
6	Skin changes (as defined in class 4) in conjunction with active ulceration

*Adapted with permission from Wound, Ostomy and Continence Nurses Society, 2015.

Because patients with CVD may have concomitant arterial disease, hand-held Doppler ultrasonography verifies the presence of an audible signal when edema makes palpation of the pulse difficult. Assessment in the clinical setting should include an