Foundations

Etiology and Anatomic Distribution of Venous Disease in Patients with Venous Ulcers

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ABSTRACT **Leg ulcers due to chronic venous disease affect 0.5 to 1% of Western populations. Reflux and/or obstruction of the lower extremity veins are the underlying pathophysiological mechanisms. The superficial venous system is the most common site of venous insufficiency in patients with chronic venous disease; however, the incidence of deep venous insufficiency is increased in patients with venous ulcers. Skin damage is most often associated with multisystem incompetence, and it is rare with deep venous reflux alone. Only 30 to 50% of limbs with ulceration have a documented episode of deep vein thrombosis. Recent developments in the diagnosis of venous disease have allowed more accurate identification of the malfunctioning venous segments and a more rational application of surgical treatment.**

Keywords **Chronic venous disease, deep vein thrombosis, ambulatory venous pressure**

Lower extremity ulceration represents the most advanced stage of chronic venous disease (CVD). Venous hypertension secondary to venous outflow obstruction, reflux, or both is the main cause for it.

For many years, the study of CVD suffered from the lack of a sufficiently organized classification system, which made comparison between reports and treatment results difficult. Moreover, different investigators used a variety of diagnostic procedures, invasive and noninvasive, to study the distribution and

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pathophysiology of venous disease. The lack of standardized techniques in these procedures further added to the confusion. Duplex ultrasound examination of the lower extremity veins was introduced in recent years and significantly improved our understanding of the etiology of CVD. This noninvasive test allowed a more accurate identification of the malfunctioning vein segments and has shown that reflux is the predominant cause of ulceration with a significant contribution of the superficial system. These observations contributed to the development of a more rational therapeutic approach.

In 1994, an international committee on CVD[1] revised the previously existing classification system and suggested that limbs with CVD should be classified according to clinical signs (C), etiology (E), anatomic distribution (A), and pathophysiological condition (P). This system (CEAP) was designed to provide the additional details necessary to accurately compare limbs in treatment trials.

CLINICAL PRESENTATION OF CVD

Seven clinical classes (0–6) were described in the CEAP classification. Limbs in higher categories have more severe signs of CVD and may have some or all of the findings defining a less severe clinical category. Each limb is further characterized as symptomatic (S) or asymptomatic (A).[1] The most common symptoms seen with telangiectases and reticular and varicose veins include lower extremity aching, pain, skin irritation, itching, heaviness, and burning sensation.

The prevalence of lower extremity ulceration secondary to CVD in European and Western populations is estimated to be 0.5 to 1%.[2,3] In a cross-sectional population study of 382 patients with active leg ulcers, Nelzen et al.[4] reported that venous insufficiency was the dominating etiologic factor in 54%, and the overall incidence of venous disease in this selected population was 72%. This percentage is significantly different from previous reports in the middle of the 20th century that quoted venous etiology in 84 to 97% of patients with leg ulcers.[5–7] It is very likely that the prevalence of CVD as a cause of ulceration has been underestimated in this study[4] for the following reasons. Only four sites of the leg veins were examined for reflux and obstruction, using a bidirectional Doppler. It has been shown, however, that saphenous reflux can exist in the absence of saphenofemoral or saphenopopliteal incompetence.[8,9] Also, arterial disease was defined as an ankle to brachial index (ABI) of <0.9 without necessarily this being the cause of the ulcer because many of the limbs had an ABI of >0.5. Similar results have been reported in other studies that suggest a change in the etiologic spectrum toward arterial and mixed ulcers most likely due to the ongoing aging of the population.[10,11] However, the following limitations exist in these studies. Clinical examination was used to evaluate CVD in one study,[11] whereas in the other,[10] the sample size was small and patients with foot ulcers only were excluded.

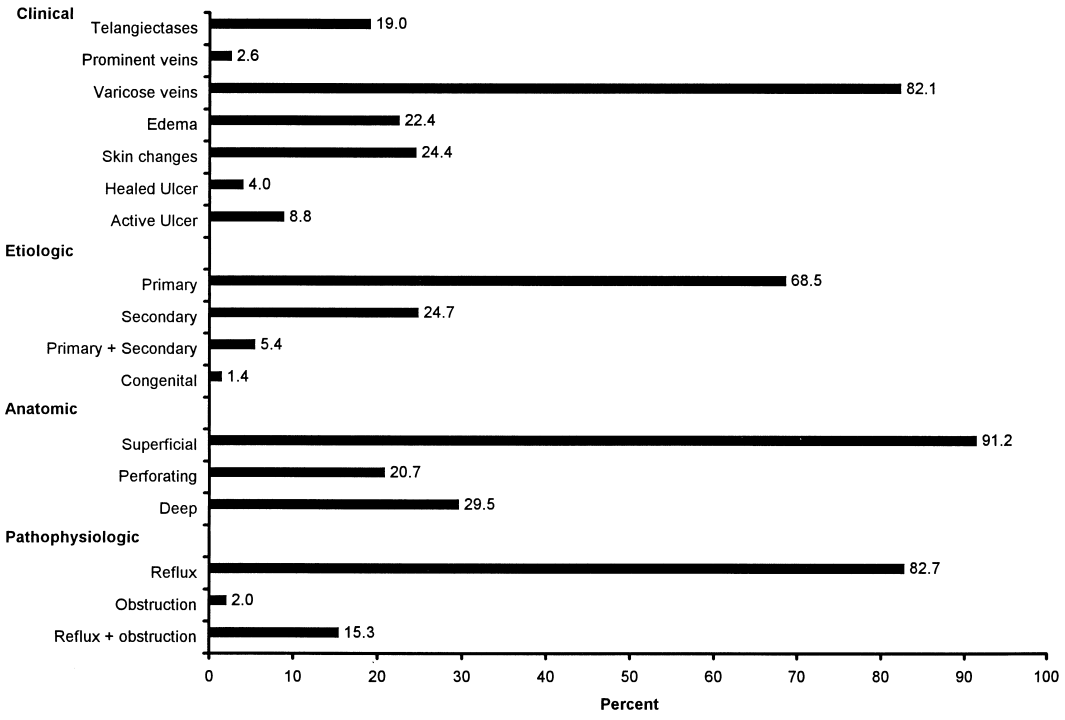

Fig. 1 Combined data from the two main studies using the CEAP classification system on consecutive patients.[12,13]

Kistner et al.[12] applied the CEAP classification in 102 legs of 70 patients with CVD. They found 14 legs (13.7%) with ulceration ($C_{5–6}$). In a similar study of 250 limbs in 182 patients, Labropoulos[13] reported that 5.2% had healed ulcers (C_5) and 7.2% had active ulcers (C_6). The combined results of these studies are presented in Figure 1.

ETIOLOGY

According to CEAP classification the etiology of CVD may be congenital (C), primary (P), or secondary (S).[1]

Congenital anomalies of the lower extremity veins are responsible for a very small number of leg ulcers and are rarely diagnosed. These abnormalities are present at birth but may be recognized later. Primary venous dysfunction denotes CVD of undetermined etiology. CVD may also be secondary to an acquired condition such as deep vein thrombosis (DVT) or trauma.

In the study mentioned above, Labropoulos[13] reported that 0.8% of the limbs had congenital abnormalities, 64% had primary CVD, and 27.6% secondary. In 7.6% primary venous insufficiency existed in some vein segments

and secondary in others. These were usually patients with previously diagnosed varicose veins who developed DVT and subsequent deep venous reflux during their follow up period. The same author studied 94 patients with 112 ulcerated limbs and found that 39% of the affected limbs had DVT and 3% had superficial thrombophlebitis in the past.[14] Kistner et al.[12] reported congenital etiology in 3% of the limbs, primary in 79%, and secondary in 18% (all postthrombotic). No patients with both primary and secondary CVD were reported in this study. Venous ulcers were found in 8 of 18 limbs (44%) with secondary CVD (7 were C_6 and 1 was C_5). Of the remaining 10 limbs, 7 were C_4 and 3 were C_3. Only 7% of limbs with primary CVD had ulcers. Other reports[15–19] also show that in limbs with ulcers the incidence of previous DVT is higher compared with limbs with clinically less severe CVD. These data clearly suggest that primary disease is significantly more common than secondary, but patients with secondary disease develop more advanced clinical signs.

ANATOMIC DISTRIBUTION

Based on the anatomic classification in CEAP, the vessels involved should be described as superficial, deep, or perforating.[1] Any combination of these systems may be affected. The studies by Kistner et al.[12] and Labropoulos[13] show that the superficial veins are affected in 90% of limbs with CVD, the deep venous system only in 30%, and the perforator veins in 20% (Fig. 1). Of the 14 limbs with ulcers in the first study,[12] 4 had reflux limited to the superficial and perforator veins, 2 to the superficial and deep veins, 1 to the deep veins only, and in the remaining 7 all 3 venous systems had reflux.

For many years it was a common belief that venous ulcers resulted from reflux and/or obstruction of the deep venous system secondary to an episode of DVT. The introduction of color flow duplex scanning in the last decade has allowed a more detailed study of the anatomic distribution and pathophysiology of venous incompetence. Several recent studies[4,8,12,14,15,19–21] have shown that reflux confined to the superficial veins alone is responsible for 17 to 54% of venous ulcers. Overall, reflux in the superficial veins is seen in 79 to 93% of limbs with ulceration.[12,14,15,19,22,23]

The prevalence of isolated deep vein reflux in ulcerated limbs ranges between 2.1 and 15%.[8,12,19,23] It should be noted however that, in the study that showed high prevalence,[8] the perforating veins were not examined; therefore, this percentage was clearly overestimated. In both studies that used CEAP classification[12,13] isolated deep venous reflux was seen in 6% of limbs, whereas overall involvement of the deep venous system is found in 29 to 50%.[12,13,22,23] The prevalence of deep venous reflux in patients with venous ulcers (C_6) is higher, ranging between 50 and 70% in various reports.[12,14,15,19,24]

The hemodynamic significance of incompetent perforating veins remains a controversial issue. Some investigators reported that incompetent perforators

do not contribute to venous hypertension,[25–27] whereas others suggest that they are important.[19,22,28–30] In most studies, reflux in the perforator veins only was seen in less than 3% of limbs with CVD.[12–15,21] Although the role of these veins in the development of signs and symptoms remains unclear, their number and size increases with worsening of CVD.[19,31–34]

Most patients with ulcers (52–70%) have incompetence in more than one system,[14,15,19,21] and reflux in all three venous systems is seen in 16 to 50%.[12,14,15,19,21,23] Interestingly, in a small percentage (4–6.3%) of limbs with apparent venous ulcers, no incompetence or obstruction is detected in any of the venous systems.[14,19]

In a study of 34 limbs with 43 ulcers,[15] examination of the local veins (veins passing through the ulcer or within 2 cm from its periphery) revealed reflux in 86% of ulcerated areas. When the pattern of reflux in the local veins (superficial vs deep) was compared with that of the axial veins, it was found to be similar in limbs that had reflux limited to either the superficial or the deep venous system (with or without associated perforator incompetence). This finding explains the very good results of surgical treatment targeting the affected venous system in patients with this type of axial vein reflux. Conversely, when reflux was seen in both the superficial and deep venous systems, the pattern of reflux in the local veins was not predictable, indicating that examination of the local veins in the ulcer area may be necessary prior to applying a treatment plan. Reflux in the perforating veins at the ulcerated area was seen in only 28% of ulcers. Perforator reflux at that level was always associated with superficial and/or deep vein incompetence.

Regarding the distribution of the disease, it should be mentioned that saphenous reflux often occurs in the absence of saphenofemoral and saphenopopliteal junction incompetence.[9,35,36] This may in part explain why recent studies have found that junction ligation without stripping results more often in residual/recurrent varicosities.[37–39]

PATHOPHYSIOLOGY

The main mechanisms responsible for the symptoms and signs of CVD according to CEAP are reflux, obstruction, or a combination of the two.[1] In the two main reports of patients with CVD who were classified using the CEAP system,[12,13] obstruction without reflux was seen only in 2% of limbs and reflux with obstruction in 12 to 17%. Combined reflux and obstruction was more often seen in limbs belonging to classes C_4 to C_6.[13] This is not surprising because a recent prospective study demonstrated that a combination of reflux and obstruction had worst prognosis for developing skin damage compared with reflux or obstruction alone (odds ratio 3.5, 95% CI 1.4–8.6).[40] In a study of patients with venous outflow obstruction,[41] the incidence of limb ulceration was 10%, whereas 40% of patients belonged to clinical classes C_4 to C_6. Their symptoms, however, cannot be attributed to

venous outflow obstruction exclusively because in all these patients some degree of reflux was detected. The hemodynamic changes in the venous system were more significant with more proximal obstruction and poor collateralization.

The prevalence of DVT increases with worsening of CVD.[24] A documented episode of DVT was found in 33 to 50%[8,12,14–19,22] of patients with ulceration, which is much higher prevalence than any other CVD class.[24] This prevalence is probably underestimated because many thrombi remain undetected. Some of these thrombi may resolve without leaving any evidence of luminal damage other than reflux. In patients with DVT the determinants for clinical severity include the extent of reflux, presence of persistent popliteal obstruction, and rate of recanalization.[42] It has been demonstrated that rethrombosis occurs in 24.6% (95% CI, 19.6–29.7%) of patients at 5 years and in 30.3% (95% CI, 23.6–37.0%) at 8 years.[43] The development of ipsilateral recurrent DVT had a significant relation with the risk for postthrombotic symptoms (hazard ratio, 6.4; 95% CI, 3.1–13.3).[43]

The pathophysiological mechanism responsible for venous ulceration is venous hypertension. It has been shown[44,45] that venous hypertension leads to increased capillary permeability to fibrinogen and extravasation of red blood cells through widened intercellular gap junctions. This results in deposition of extracellular matrix around the capillaries that consists of fibrin, collagen types I and III, tenascin, laminin, and fibronectin. It has been suggested that this pericapillary cuffing acts as a diffusion barrier for oxygen and perhaps other nutrients, causing hypoxia of the overlying dermis, which, therefore, becomes very susceptible even to minor trauma.[44] There are, however, no convincing data to support this hypothesis.[46] In fact, Michel a few years later demonstrated in a theoretical model based on the Krogh-Erlang equation that it is very unlikely for the pericapillary cuffs to act as diffusion barriers to oxygen.[47] More recently it has been shown that the the microvascular changes in the skin of limbs with CVD are characterized by activated endothelium and inflammatory cells in the perivascular space.[48] There is some evidence to support endothelial injury form leukocytes that are being attached to the cutaneous microvessels. It has been hypothesized that such a repeated injury over a long period may be responsible for the development of local skin damage.[48]

Nicolaides et al.[49] measured the ambulatory venous pressure (AVP) in patients with CVD and found that, when it was less than 30 mmHg, the incidence of leg ulceration is zero. This incidence increased linearly with AVP to reach 100% when the AVP was greater than 90 mmHg. Poor calf muscle pump function has also been associated with CVD severity.[50] Christopoulos et al.[50] showed that patients with ulceration had worst ejection fraction compared with patients with varicose veins only. However, when patients were matched for age and duration of disease in another study, the amount of reflux was found to be most significant for the severity of CVD.[23]

CONCLUSION

For many years the management of lower extremity ulcers due to CVD was rather empirical because the pathophysiology and anatomic distribution of the disease were in many cases unclear. Duplex ultrasound has proved that the superficial venous system is involved in over 90% of ulcerated limbs[12,14,15] and represents an easily accessible target for surgical intervention. When venous ulceration is due to superficial and perforator incompetence, surgical treatment may heal up to 90% of the ulcers with very good medium- to long-term results.[51,52] According to recent studies about 30 to 50% of patients with ulcers belong to this category.[8,14,15,19,20] However, very often more than one venous system is incompetent in patients with venous ulcers, and the contribution of each system in the clinical picture may be difficult to determine. When the deep veins are involved surgery of the superficial veins has worst results with very high recurrence rates at 5 years.[52] The modified Linton procedure in patients with refractory ulcers and deep vein reflux had a 22% recurrence rate at 4 years.[53] In patients with deep venous reflux and/or obstruction additional procedures that can improve the underlying abnormality may be required. Few studies have shown encouraging results in such patients.[54–57] Most recently Gloviczki et al., from the North American Study Group on endoscopic subfascial perforator ligation found that correction of superficial and perforator vein reflux in the absence of deep venous obstruction predicted ulcer healing.[29] Postthrombotic limbs had a higher recurrence rate compared with limbs with primary venous reflux at 2 years (46 vs 20%, $p < 0.05$). Therefore, a detailed study of the affected extremity that will identify and provide functional information about all malfunctioning venous segments is of outmost importance when planning the treatment of these patients.

The CEAP classification system was created to offer guidelines for reporting venous disease to improve precision and allow for more accurate comparisons between different reports. Use of this classification provides organized, detailed information about the key elements of venous abnormalities in each patient and clarifies the interrelationships among the clinical manifestations, etiology, anatomic distribution, and pathophysiology of CVD. Therefore, it will facilitate the development of more uniform treatment plans in the different categories of disease, thus improving the outcome of this common problem.

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Expert Commentary

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Although the clinical history and physical examination are important first steps in the evaluation and management of venous disease, experience has shown these can be refined with utilization of vascular laboratory data. Noninvasive tests such as duplex evaluation for the presence or absence of deep venous thrombosis, assessment of valvular reflux, as well as air and photo plethysmography add a great deal to the specific diagnosis and selection of treatment options. It is important to identify sites of reflux and obstruction prior to surgical treatment of chronic venous insufficiency.[1] To that end, knowledge of the patterns of chronic venous insufficiency encountered in patients with chronic venous insufficiency is clearly desirable. The review of etiology and anatomic distribution of venous disease in patients with venous ulcers by Drs. Labropoulos and Tassiopoulus is therefore important both from a research and clinical perspective. One particular strong point of this paper is that it incorporates a standardized classification of venous disease, allowing for more meaningful comparison of studies designed to evaluate results of specific interventions and thus strengthening and clarifying the data underlying their potential clinical application.

To describe newer data on the etiology and anatomic distribution of venous disease in patients with venous ulcers, Labropoulos and Tassiopoulos reviewed several articles addressing venous disease by etiology and anatomic distribution according to the CEAP classification system.[2] This system, which categorizes venous disease by clinical (C), etiologic (E), anatomic (A), and pathophysiologic (P) features, is a major step forward to accurate and reproducible classification of chronic venous insufficiency. The review focuses primarily on two studies, one by Kistner[3] and the other by Labropoulos,[4] which both describe chronic venous insufficiency with the new CEAP system. The article is quite specific and contains many interesting details concerning the etiology and distribution of abnormalities in chronic venous insufficiency. It is somewhat surprising to note isolated deep venous insufficiency was seen in only 6% of limbs with venous ulcers,[5] and that isolated superficial incompetence in some studies was seen in up to 54% of limbs with venous ulcers. Labropoulos and Tassiopoulos therefore suggest that the distribution of venous disease according to the CEAP classification system indicates that a significant portion of patients with venous ulceration could and should be treated with surgical intervention on the superficial venous system. The authors also note only 30–40% of patients with chronic venous insufficiency

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have a prior history of deep venous thrombosis,[5] and therefore contend congenital or acquired venous wall weakness may be more prevalent an etiology of chronic venous insufficiency than has been previously recognized.

Overall, Labropoulos' and Tassiopoulos' review is well organized and of appropriate breadth. The information presented provides important data for the clinician to consider when evaluating a patient with venous ulcer disease and when considering various treatment options. Perhaps as more outcome data are obtained for patients evaluated with newer Duplex technology and classified under the CEAP system, indications for and outcomes of operative intervention in chronic venous insufficiency may be clarified by real data.

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The Last Word

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Patients with venous ulceration have more venous sites involved compared to the rest of chronic venous insufficiency classes. Our paper and the commentary emphasize the use of duplex scanning to delineate the distribution and extent of venous reflux and obstruction. The addition of plethysmographic testing aids the overall hemodynamic evaluation and provides objective evidence for lower limb function improvement after an intervention. The use of a standardized classification allows meaningful comparisons among different studies. However, today there are very few studies that have used the CEAP system, and therefore our knowledge on venous ulceration is limited. From the current studies it appears that the majority of limbs with venous ulceration have reflux only. Obstruction or deep venous reflux only is uncommon. These findings suggest that most limbs are amenable to surgical treatment. Prospective studies in a large number of patients will increase our understanding on pathophysiology of venous ulceration and on the effects of different interventions on healing, recurrence, and ulcer-free length of time.

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