Sequelae of Untreated Venous Insufficiency

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ABSTRACT

Untreated venous insufficiency results not only in a gradual loss of cosmesis but also in variety of complications including persistent pain and discomfort, hemorrhage, superficial thrombophlebitis, and progressive skin changes that may ultimately lead to ulceration. In rare instances, chronic soft tissue changes may lead to stiffness of the ankle joint, fixed plantar flexion, and periostitis. This article reviews the variety of complications caused by venous insufficiency.

KEYWORDS: Venous insufficiency, venous, varicose veins complications, venous ulcers

Objectives: Upon completion of this article, the reader should be able to (1) describe the broad range of manifestations of venous insufficiency and (2) outline their progressive nature.

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The etiology of venous disease is multifactorial. Occupations involving working long periods of time standing may result in chronic venous distention and secondary valvular incompetence. In women, vein walls and valves are affected by cyclical changes in progesterone levels. These changes are exacerbated during pregnancy, when blood volume is increased and venous return is restricted by the gravid uterus. With age, degenerative changes and atrophy of the smooth muscle in the vein wall result in susceptibility to dilatation. For all of these reasons, the prevalence of venous disease is higher in industrialized countries¹ and in women and individuals of advanced age.² The incidence of venous disease in the United States is expected to rise as the “baby boom generation” ages.³

Untreated venous insufficiency results not only in a gradual loss of cosmesis but also in variety of complications, the major ones being persistent pain and dis-

comfort, hemorrhage, superficial thrombophlebitis, and progressive skin changes that may ultimately lead to ulceration. (Table 1) Rarely, chronic soft tissue changes may eventually lead to stiffness of the ankle joint, fixed plantar flexion, and periostitis. Each of these categories is considered in detail.

COSMESIS

For many patients, the progressive loss of the cosmetic appearance of their legs is the prime cause for seeking treatment (Fig. 1). Unsightliness is not confined to tortuous dilated veins but includes telangiectasias (“spiders”) and reticular veins. Telangiectasias are more common in women than in men and may develop during pregnancy. The former may have a variety of appearances including stellate, sunburst, and even arborizing patterns (Fig. 2). These intradermal veins may spread gradually

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Table 1 Signs and Symptoms of Varicose Veins

Dyscosmesis—loss of positive body image
Pain and discomfort
Ankle edema
Cramps
Puritis
Superficial thrombophlebitis
Hemorrhage
Eczema
Hyperpigmentation
Lipodermatosclerosis
Atrophie blanche
Ulceration

over the whole of both lower legs, giving them a blue-purplish discoloration (Fig. 3). Women are more concerned with the appearance of their legs than are men, especially if they wish to wear skirts. O'Leary et al⁴ found that 33% of women presented complaining of unsightly legs compared with 8% of men who did so. Increasingly, concerns with physical appearance and "body image" are driving the demand for treatment of varicose veins.

PAIN

Symptoms of venous insufficiency are highly variable. Patients report a spectrum of discomfort ranging from

Figure 2 Cutaneous telangiectasias.

Figure 1 Unsightliness of varicosities is a common cause of self-referral.

Figure 3 Arborizing telangiectasia. These intradermal veins may gradually spread over both lower legs, turning them purplish in color.

fullness or heaviness, dragging or aching, to frank pain. Occasionally, throbbing and itching may occur. The pain is exacerbated by standing, is progressive throughout the day, is typically felt in the muscles in the calf or thigh, and is relieved by rest and limb elevation. It is also frequently relieved by the wearing correctly fitted gradient support hose. Throbbing pain is more typically felt over areas of acute lipodermatosclerosis and superficial thrombophlebitis. Persistent pain related to ulcer formation is not relieved by rest and the surrounding skin is often very tender, although the center of the area may be numb.

Very rarely, a bursting pain is experienced during exercise (venous claudication). This is associated only with severe outflow obstruction. Night cramps are a relatively frequent accompaniment and often occur after a long day of standing without exercise. However, pain that occurs in bed and is sharp or acute is unlikely to be due to varicosities. Discomfort is often unrelated to the size of the varices⁵ and may be linked to gender. Large tortuous varicosities in men may cause only minor discomfort, whereas small spiders may be the cause of major symptoms in women. Furthermore, the patient's perception plays a major role in distinguishing between prominent normal veins and varicosities. It is also worth noting that the varicosities of a patient seen early in the morning and in cool weather may not demonstrate the prominence they have later in the day or when the weather is hotter, or both.

EDEMA

Ankle edema associated with uncomplicated varicose veins is usually mild and becomes more noticeable as the day progresses. Edema associated with deep system insufficiency is more severe and may be persistent. All patients with severe edema should be evaluated for deep system incompetence. In addition to concerns for cosmesis, it may interfere with the fitting of footwear. Venous edema must be distinguished from lymphedema. The former is pitting in nature, whereas the latter does not pit and is often described as "brawny edema" (Fig. 4).

Figure 4 Pitting edema.

Figure 5 Superficial thrombophlebitis.

SUPERFICIAL THROMBOPHLEBITIS

Thrombophlebitis is a common complication of varicose veins, perhaps because their prominence makes them more susceptible to local trauma. It is thought that this seldom occurs spontaneously and an episode of minor trauma or a period of bed rest is often associated. When superficial thrombophlebitis occurs in a normal vein, it is often associated with deep venous thrombosis. It may also be caused by occult malignancy (Trousseau phenomenon). Thrombophlebitis arises as a tender, hot, thickened area along the course of a varicose vein (Fig. 5). It is often extremely painful, and the patient may have fever and malaise. Long saphenous vein thrombophlebitis has the potential to propagate beyond the saphenofemoral junction into the common femoral vein, resulting in iliofemoral thrombosis and a subsequent risk of pulmonary embolism.

HEMORRHAGE

Bleeding from large varicosities typically follows local trauma; however, bleeding may occasionally occur spontaneously (Fig. 6). Elderly patients with thin-walled veins are at increased risk. Patients may be unaware of the venous rupture and first notice the problem when they feel blood running down the leg or even after feeling faint. Patients who faint while sitting down must be made supine to lower the venous pressure. The bleeding

Figure 6 Hemorrhage.

may be profuse and even life threatening,⁶ although it is easily managed with simple compression and leg elevation. Application of tourniquets that are not tight enough to occlude arterial inflow but enhance venous congestion may increase rather than reduce the rate of hemorrhage.

SKIN CHANGES

Although most patients are acutely aware of skin changes associated with venous disease, occasionally, patients with varicose veins may fail to notice minor skin changes in the lower leg because they are so slow to develop. On the other hand, some patients may be anxious and seek evaluation because of the fear of deterioration in their condition. O'Leary et al found that this was a reason for referral in 14% of patients.⁴

Whereas most patients with isolated and uncomplicated long saphenous incompetence do not develop ulceration, there is a group without evidence of incompetent communicating veins who do.⁷ Between 40% and 50% of patients with venous ulcers have objective evidence of post-thrombotic damage⁸ (Fig. 7).

Pigmentation

Prolonged venous hypertension resulting in venous dilatation and passage of red blood cells through the endothelium into the interstitium results in breakdown and conversion of hemoglobin to hemosiderin. This

Figure 7 Possible mechanism of ulceration in chronic venous insufficiency. (Adapted from Falanga V. Chronic wounds: pathophysiologic and experimental considerations. *Prog Dermatol* 1992;26:1-8.)

Figure 8 Pigmentation from hemosiderin deposition.

remains as a brown pigment stain on the skin. It is typically located on the lower medial third of the lower leg but can involve the entire gaiter distribution. With time, the pigmentation darkens and may eventually appear almost black (Fig. 8).

Dermatitis

With time, chronic inflammatory changes resulting in fibrin and hemosiderin deposition with local edema can result in venous dermatitis or varicose eczema. This can be either dry and scaly or vesicular and weeping. With progressive loss of epithelium, venous ulceration may develop spontaneously. In most cases, however, ulcers begin as the result of minor local trauma. Contact dermatitis can be seen in response to medications or even the rubbing of elastic bandages.

Lipodermatosclerosis

Progressive fibrosis of the skin and subcutaneous tissues induced by chronic venous hypertension is referred to as lipodermatosclerosis but is sometimes called fat necrosis,

Figure 9 Lipodermatosclerosis is noted in the margins of the ulcer. Constriction of the gaiter distribution is also noted, causing bottle deformity of the leg.

folliculitis, or chronic cellulitis. It may occur acutely or in a chronic condition. The acute form eventually progresses to become chronic, but the chronic variety can develop spontaneously.

Acute lipodermatosclerosis is painful and disabling. It typically appears as a thickened raised red-brown area in the skin of the lower leg. In addition to pain and tenderness, there is a constant sensation of heat. Chronic lipodermatosclerosis is marked by stiff and shiny skin that is fixed, hard, and indurated, contracting the subcutaneous tissues. Progressive contraction of the skin and subcutaneous tissues results in shrinking of the gaiter area and, accentuated by any edema in the calf above, gives the leg a stick-like or inverted bottle shape (Fig. 9).

Atrophie Blanche

Skin necrosis with replacement by scar tissue but without ulceration or sloughing results in small areas or patches on skin that are usually gray-white in color and usually only a few millimeters in size. These lesions, known as atrophie blanche, are associated with depression on the skin surface, are colored with thin transparent epithelium, and are sometimes surrounded by a halo of fine dilated venules. Coalescence of multiple areas may form

Figure 10 Atrophie blanche. White scars appear in a larger area of chronic skin changes.

a large scar, may break down spontaneously, or may, following trauma, form ulcers (Fig. 10).

Ulceration

All the previously described skin changes are preulcerous conditions. If these progressive changes are not reversed, impairment of tissue nutrition and oxygenation progresses to slow tissue death. If there is a supervening injury, ulceration may progress rapidly.

When ulceration begins, there is partial skin loss in an already abnormal area. In the absence of healing, remaining layers of skin necrose to produce full-thickness skin loss. Necrosis extends into the subcutaneous fat, superficial fascia, deep fascia, muscles, and even the periosteum. Although peripheral nerve endings are directly involved in the inflammatory process and may give rise to local pain, in large infected ulcers pain may be a minor feature.

Ulcers that are not aggressively treated and become secondarily infected may progress to expose tendons and periosteum. Very rarely, this may result in a need for amputation.

Although ulcers are rarely life threatening and systemic septicemia almost never occurs, they cause much morbidity from pain, discomfort, and fluid discharge. They may result in secondary amyloid disease

and can also cause anemia and hypoproteinemia. Rarely, they may undergo malignant change (Marjolin's ulcer).

In addition to causing great individual distress, ulcers have serious economic and psychological effects and are a considerable drain on medical resources. It is estimated that between 300,000 and 400,000 patients suffer from venous ulcers in North America.⁹ Various estimates of the incidence of ulceration in the United Kingdom and other European countries range between 0.25% and 0.5% of the population.¹⁰

OTHER COMPLICATIONS

Ankle Joint Stiffness

Progressive subcutaneous scarring occasionally extends into the subcutaneous tissue around the ankle joint, restricting ankle movement, reducing calf pump efficiency, and exacerbating the venous hypertension. Fibrous ankylosis may, eventually, fix the ankle joint with scar tissue.

Figure 11 Periosteal new bone formation underneath an area of recurrent ulceration.

Fixed Plantar Flexion

Chronic pain of acute lipodermatosclerosis or ulcer may result in abnormal weight bearing and eventually ankle stiffening and shortening of the Achilles tendon.

Periostitis

Long-standing inflammation in soft tissues may induce hyperemia in the underlying periosteum, which can then produce new subperiosteal bone. It is most often a coincidental finding on a plain radiograph (Fig. 11).

CONCLUSION

The sequelae of untreated varicose veins range from mental distress related to unsightliness through occupation-limiting symptoms of pain and discomfort in the limb and the potential for chronic ulceration and associated soft tissue changes. The latter present a lifelong challenge.

All patients presenting with a complaint of varicose veins require detailed and thoughtful evaluation, thorough diagnostic evaluation, and carefully considered intervention when appropriate. Many may require sustained long-term follow-up.

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