Pathophysiology of chronic venous insufficiency

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PATHOPHYSIOLOGY OF CHRONIC VENOUS INSUFFICIENCY

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Chronic venous insufficiency (CVI) is the term used to describe more advanced forms of long standing venous dysfunction of the limbs, associated with edema (CEAP Class 3), skin changes(Class 4), healed or active venous ulcerations (Class 5,6). While varicose veins affect more than 20% (25% of women, and 15% of men) of the United States population, CVI was estimated to occur in up to 2% (0.5 to 3%) of the population in Western countries, a prevalence comparable to that of diabetes.

Age-adjusted Incidence of Chronic Venous Insufficiency
Olmsted County, Minnesota, 1966 to 1990

The estimated incidence of venous stasis syndrome in the US is approximately 150,000 new cases per year; approximately 25% of these are due to post-thrombotic syndrome (PTS). PTS represents the long-term sequelae of deep venous thrombosis (DVT). The exact prevalence of PTS is unknown, but it is proportional to the prevalence of DVT in a certain population. While the incidence of DVT is between 1.0 and 1.6 per 1000 persons per year, prospective studies have shown that PTS developed after DVT in 17% of the limbs at 1 year, 23% at 2 years, 28% at 5 years, and in 29% at 8 years..
ANATOMY AND PHYSIOLOGY

Recent changes in terminology of leg veins and discovery of the saphenous sub-compartment warrant a brief discussion. The venous drainage of the lower extremity is accomplished through superficial and deep venous systems, with perforating veins connecting the two. All larger veins are equipped with unidirectional bicuspid valves which direct flow toward the deep system and cranially. The physiologically dominant deep system consists of 3 sets of tibial veins that merge in the upper calf to form the popliteal vein. The popliteal vein becomes the femoral vein at the adductor hiatus, which joins the deep femoral vein draining blood from the thigh muscles to form the common femoral vein in the femoral triangle.

Calf muscle contraction provides the main pumping force that propels blood towards the heart. The superficial venous system consists of the great saphenous vein (GSV) and the small saphenous vein (SSV) and their tributaries. The GSV originates at the medial end of the dorsal venous arch of the foot, ascends anterior to the medial malleolus, along the medial aspect of the calf and thigh to join the common femoral vein in the femoral triangle. The SSV arises at the lateral end of the dorsal venous arch, ascends posterior to the lateral malleolus, up the posterior aspect of the calf, to drain into
the popliteal vein in the popliteal fossa.

The GSV lies deep to the saphenous fascia, while their tributaries lie relatively unsupported, superficial to the saphenous fascia, thereby accounting for early varicose transformation of clusters of tributary veins.
Perforating veins connect the superficial to the deep venous system, either directly to the main axial veins (direct perforators) or indirectly to muscular tributaries or soleal venous sinuses (indirect perforators). The term “communicating” veins refers to interconnecting veins within the same system. While flow through perforators of the foot is usually from deep to superficial, in normal limbs unidirectional flow in calf and thigh perforators, from the great and small saphenous systems towards the deep veins, is assured by venous valves.

In the mid and distal calf the most important direct medial perforators do not originate directly from the great saphenous vein. This observation is extremely important as stripping of the GSV will not affect flow through incompetent medial calf perforators. The most significant are the posterior tibial perforators, termed also the Cockett perforators, which connect the posterior arch vein (Leonardo’s vein) to the paired posterior tibial veins. Three groups of Cockett perforators have been identified. The Cockett I perforator is located posterior to the medial malleolus and may be difficult to reach endoscopically. The Cockett II and III perforators are located 7-9 cm and 10-12 cm above the lower border of the medial malleolus, respectively.
Anatomic dissections by Mozes et al. demonstrated that 63% of medial perforators can be identified in the superficial posterior compartment. The remaining Cockett II, III and paratibial perforators lie within the deep posterior compartment or can be found within the intermuscular septum separating the deep and superficial compartments. Therefore, in order to interrupt all clinically relevant perforating veins a paratibial fasciotomy and exploration of the deep compartment must be performed.

PATHOPHYSIOLOGY

The most important risk factor for varicosity is a family history of varicose veins. There is evidence now that varicosity is linked to an autosomal dominant gene with variable penetrance. Additional risk factors include female gender, pregnancy, advanced age, a profession with prolonged standing at work, obesity and diet leading to constipation. There is also increasing evidence that at the cellular level, in the wall of varicose veins, there is conversion of smooth muscle cells from a contractile phenotype to a secretory phenotype.

While the prevalence of varicose veins is higher in the elderly, there are sufficient data to support that varicose veins may stay asymptomatic for lifetime. The incidence of complications, however, such as thrombophlebitis, deep vein thrombosis, bleeding, swelling and skin changes, including ulcerations, increase with age and depend greatly on involvement of the deep system.

The relationship between CVI and ambulatory venous hypertension has been known for decades. Studies have confirmed that ambulatory venous pressure has not only diagnostic but also prognostic significance in CVI. High ambulatory venous pressure may be due to primary valvular incompetence (PVI) in superficial, deep and/or perforator veins, or it may be the result of a previous deep venous thrombosis (DVT). Valvular incompetence is believed to occur secondary to weakness and dilatation of the vein wall preventing adequate coaptation of the venous valve cusp. Reflux of blood across incompetent valves in the great, and, less frequently in the small saphenous vein, results in increased pressure in the superficial venous system. Valves in deep and perforator veins may similarly be primarily incompetent, allowing blood to reflux down the deep veins, and outwards from the
deep to the superficial veins during calf muscle contraction. Deep venous valvular incompetence may also be secondary to DVT, valve destruction occurring from fibrotic organization of thrombus attached to valve leaflets. Post-thrombotic syndrome (PT), may result from valvular incompetence or partial deep venous obstruction due to incomplete recanalization of DVT. Deep venous incompetence (DVI) is initially compensated by the calf muscle pump, but eventually results in secondary incompetence of valves in perforating veins, and transmission of pressure from the deep to the superficial veins. Advanced venous dysfunction is associated with ambulatory venous hypertension and is characterized by induration and inflammation of the skin and subcutaneous tissues, microvascular stasis, accumulation and activation of leukocytes in the perimalleolar area, and release of necrotizing lysosomal enzymes, ultimately leading to tissue destruction, chronic ulceration and difficulties in healing. These events cause severe discomfort and lead to considerable disability.

Selected references


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