Current Views on the Management of Incompetent Perforator Veins

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Chronic venous disease (CVD) is prevalent worldwide. Many patients suffer from signs and symptoms of CVD. The impact of superficial and deep vein disease is well established. However, the role of perforator veins is controversial (1-3). There are many randomized trials showing the effect of treatment on superficial veins mostly by ablation techniques, phlebectomies and sclerotherapy. There is an increasing body of evidence for treating deep vein obstruction but a definitive trial (CTRACT) is on its way (4). The purpose of this paper was to define the impact of perforator veins in patients with CVD.

STRUCTURAL AND FUNCTIONAL CONSIDERATIONS

The perforator veins pierce the deep fascia and connect the superficial veins with the deep axial and muscular veins. They may often have more than one connection with the deep veins. Most of the perforator veins have at least one valve located closer to the deep veins (5). Although some calf perforator veins allow inward (towards the deep veins) and outward (towards the superficial veins) flow most of the blood volume is directed inwards (2). There are about 150 perforator veins in each human lower extremity (60 in the thigh, 55 in the leg and 28 in the foot) as shown by van Limborgh in 1961 using micro injection techniques (6,7). About 30 of them may become incompetent and may contribute into the venous hypertension (2). Most of the incompetent perforator veins are located in the calf. A perforator vein is accompanied by a perforating artery which is smaller in diameter than the vein and a nerve which is thinner than the artery.

It is believed that perforator veins are the cause of venous reflux and recurrent venous disease. This information is based on cross-sectional studies where function was examined at one time point without knowing the sequence of events. The volume in the perforating veins is small and reflux has been defined as outward flow of >350 ms but for simplicity has been accepted as >0.5 s similar to the superficial veins (8). However with time in patients having reflux more blood is going through the perforators to be drained in the deep veins. These perforator veins due to the higher volume of blood dilate over time to accommodate the need and at some point may become incompetent (9). Perforator veins develop also reflux by vein wall disease extension from the affected superficial veins that are connected to the perforators as demonstrated in a longitudinal study (9). In primary venous disease reflux in the perforator veins alone with normal superficial veins connecting with them has never been demonstrated. In all occasions when the perforator veins are incompetent the superficial veins that connect with them are also have reflux. This has important clinical implications.

Another issue has been the relevance of the perforator veins as cause of recurrent varicose veins. Most studies showing an association with this they have not provide a clear evidence. Even in the REVAS study which was prospective an association was demonstrated with recurrence but it was not demonstrated to be the cause (10). In REVAS and other studies incompetent perforators persist due to treatment failure or develop reflux as venous disease progresses (1,10,11). In all papers published before and after treatment never has been demonstrated that reflux started in a perforator and then extended in the superficial veins.

CLINICAL IMPACT

The size and number of incompetent perforators increases with venous disease severity (2). Such veins are more common
in patients with skin damage and particularly in those with ulcers and are most often found in the lower calf and ankle regions (2,12). The guidelines are against treating perforating veins in patients with CEAP classes 0 to 3 (13-15). They suggest treatment of pathological perforator veins which are those having ≥0.5 s reflux duration and >3.5 mm in diameter and are found at or near the ulcer which is difficult to heal or when it recurs (12). The evidence for treating such perforators is weak, while there are no papers supporting treatment in patients with CEAP class 4. Nevertheless, still many practitioners treat perforating veins even in the absence of skin damage.

A systematic review of 20 studies (1RCT and 19 case series) having 1140 treated limbs indicated that SEPS with or without saphenous ablation lead to an 88% chance for ulcer healing and 13% chance for recurrence at short term (16). They suggested that RCTs should be performed to demonstrate the effect of treating the perforating veins. Subsequently two RCTs were performed and none showed a clear benefit from treating the perforating veins in patients with venous ulcers (17,18). The first one was mostly designed to address conservative versus surgical treatment and added the SEPS in 51 limbs who had superficial surgery and in 40 limbs with previous superficial surgery (17). They suggested in the secondary end points benefit on ulcer free period but that is highly questionable as many things were not controlled and unequal comparisons were made. In the second RCT there was no short-term clinical benefit from adding SEPS to saphenous surgery in patients with venous ulcers and incompetent perforators, despite SEPS reducing the number of perforators at 1 year (18).

Thermal ablation of the perforator veins has an occlusion rate ranging from 60% to 80% (19). Closure rate may improve by retreating failures. Ultrasound guided foam sclerotherapy may have lesser closure rate compare to thermal ablation but it is easier to perform and also treat the varicose veins at and near the ulcer area. There may be some benefit in treating perforating veins in patients with venous ulcers as described above. However, prospective studies having enough power and good follow-up are needed.

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