

Why Treat Superficial Venous Disease?

Focusing only on the heart and arteries isn't enough.

BY JAMES D. JOYE, DO, FACC

Why treat superficial venous disease? I would turn this question around for my interventional cardiologist (IC) colleagues and instead ask, "Why NOT treat superficial venous disease?" Granted, most of the training we ICs received focused on the heart and arterial systems and dealt minimally, if at all, with the venous system. Yet the heart, arteries, and veins comprise a system, and as experts on cardiovascular disease, we need to understand that system and treat it when it becomes dysfunctional. Especially for ICs already treating peripheral arterial disease, treating venous disease completes their practice's offerings.

EVOLUTION OF PRACTICE

Caring for patients with venous disease really represents another step in the evolution of interventional cardiology. Until about 2000, most ICs focused primarily on the heart and did little to treat peripheral arterial problems. As awareness of peripheral arterial disease grew, many ICs began treating that condition. Now that we have a better awareness and understanding of the impact of chronic venous insufficiency (CVI), I believe it is time that we add the treatment of CVI to our practices.

In more practical terms, in my experience, many (often the majority) of our patients with cardiac and/or arterial disease also present with venous disease. In my opinion, we owe it to them to provide complete care. Many of these patients can be readily identified by visual inspection of their legs or by simply asking them questions about venous disease symptoms during their visit. Also, you can make sure that the venous disease symptoms are included on the patient ques-

tionnaire. Finally, educational brochures and posters about venous disease can be present in the waiting and examination rooms.

I should add that my "case" for treating venous disease comes with the benefit of hindsight. I have been practicing interventional cardiology since 1995, performing both cardiology and endovascular procedures, including advanced work in limb salvage, claudication, and wound care. I did not start treating venous disease until 2007. Before that, I believed that many physicians who treated vein problems focused on the cosmetic rather than the clinical aspects and were pursuing it more for financial reasons than for patient care. Besides, I had my hands full just treating cardiac and arterial disease, so I would refer out patients with advanced venous disease.

Over time, however, I was increasingly asked to help in cases with large vessel issues (e.g., central vein stenosis, May-Thurner syndrome, and deep vein thrombosis). I also found that, in some patients, arterial treatments did not resolve some problems, such as lingering lower extremity ulcers. Some patients had advanced venous disease, presenting with profound edema, chronic dermatologic changes, recurrent ulcers, and other typical symptoms. I realized I had been missing half of the circulatory picture, the venous system. To provide the care these patients needed, I decided to learn about the venous system and venous disease.

IDENTIFYING VENOUS DISEASE

I realized that there was much about venous disease I did not know, and I believe this probably holds true for many ICs today. First, CVI is fairly common: an estimated 25% of adults in the United States have varicose

veins, and 6% have more advanced chronic venous disease.¹ Risk factors for venous disease include older age, female sex, obesity, family history, prolonged standing (e.g., in an occupation), multiple pregnancies, and frequent heavy lifting.^{2,3}

CVI develops when venous pressure is increased and the return of blood is impaired. This can be due to incompetence of valves in the axial deep, superficial, or perforator veins; venous obstruction; or both. Muscle pump dysfunction in the lower legs can also worsen venous return. Compromised venous return produces venous hypertension, particularly with standing or ambulation. Over time, venous hypertension may result in dermal changes with hyperpigmentation, subcutaneous tissue fibrosis, and possible ulceration.⁴

In my discussions about the development of venous microangiopathy over the last several years, I have encountered several hypotheses about its progression. The “fibrin cuff” hypothesis suggests that fibrin-containing fluid accumulates in the pericapillary space, increasing the diffusion barrier, inhibiting tissue repair, and maintaining inflammation. The “growth factor trapping” hypothesis postulates that fibrin and other macromolecules trap growth factor, preventing it from stimulating healing. In the “white blood cell trapping” hypothesis, white blood cells trapped in the capillaries or postcapillary venules release inflammatory mediators and proteolytic enzymes, resulting in endothelial damage that may increase permeability or impede flow, leading to occlusion. Regardless of the true underlying basic science, the reality is that chronically overpressured superficial veins will dilate, become varicose, and hydrostatically deliver an unhealthy amount of extravascular fluid to the lower extremities.

Symptoms of venous disease include varicose veins; restless legs; heaviness and fatigue; leg or ankle swelling; pain, aching, or cramping; burning or itching of the skin; skin changes; and ulcers on the skin (e.g., on the lower legs). Venous disease is not merely a cosmetic problem; it can be a serious medical condition. Patients with CVI may experience pain, debilitating symptoms, and a reduced quality of life.⁴⁻⁶ In some severe cases, CVI can even require limb amputation.⁵ However, CVI need not be severe to affect quality of life. A study of current and retired employees of the University of California, San Diego, showed that even modest venous disease caused significant pain, reduced individuals’ perception of health, and impaired physical functioning, including at work and in daily activities.¹ Arterial and venous claudication present differently. Venous claudication presents itself as

daily prolonged discomfort and disability.⁴

Obviously, it is important that we treat severe CVI (e.g., CEAP class 4–6), but also selectively treating CEAP class 2 to class 3 patients can improve quality of life. Patients with more moderate CVI may usually feel fine, but by the end of the day, their legs can feel full, heavy, and fatigued. As a result, they are at risk for becoming increasingly sedentary, setting up a path to poor health outcomes over time. Thus, treating even milder forms of CVI can not only prevent progression to more severe disease but can also have a positive impact on overall health.⁴ The opportunity to effectively treat these patients with CVI requires that ICs become educated on the topic, aware of the natural history, and committed to a treatment strategy.

For ICs involved in treating patients with critical limb ischemia (CLI), it is important to know that many patients with CLI have mixed arterial and venous disease. For example, an IC I know well had been treating a patient with arterial disease and recurrent CLI for some time. Despite some very skilled arterial reconstruction, close surveillance, and wound care, the patient’s wound simply would not heal. He referred the patient to me for a second opinion, and within minutes of talking and examining her, it was clearly obvious that her medial malleolar distribution, moist ulcer was perpetuated by CVI. Her problem was not arterial insufficiency, and additional endovascular intervention was not what she needed. A simple venous reflux ultrasound confirmed my suspicion, and subsequent endovenous ablation ultimately led to wound healing. I am embarrassed to admit that earlier in my career, I made the same mistake on a handful of my CLI patients. It was not until I made the commitment to become educated about CVI that I was better able to more comprehensively care for my patients with lower extremity disease.

VENOUS EDUCATION

Resources are increasingly available for those seeking to learn more about venous disease and how to incorporate its treatment into their practice. In addition to satellite programs at most major vascular and endovascular meetings, dedicated vein meetings are now starting to emerge. Dedicated 2- to 3-day seminars are also available for intensive study of CVI, device-specific training, and the ultrasound skills that are necessary. In my opinion, the challenge for the IC really lies in devoting the time and energy to learn the venous system and how it behaves and misbehaves. Compared to most of

(Continued on page 18)

(Continued from page 5)

the interventional procedural work that we perform, the technical aspects of endovenous ablation are not difficult to learn or apply. Many of the catheter and wire skills used for coronary and endovascular procedures readily translate to endovenous procedural work. The reverse is also true, as I feel that the unique skill sets learned to perform venous procedures has made me better on the arterial side of my practice. Endovenous procedures intimately require a dedication to transcutaneous, hand-held ultrasound to identify pathology, gain access, and guide therapy. Now that I am comfortable with ultrasound guidance, and in the interest of patient safety, I encourage the routine use of ultrasound for all vascular access.

CONCLUSION

Given that CVI is a widely prevalent condition among cardiovascular patients, ICs can determine the amount of time they want to devote to it in their practice. In my case, treatment of CVI and its sequelae comprises approximately 5% of my consultative and procedural work. For others, it may be significantly more. I find this work to be professionally satisfying on many levels. I perform radiofrequency ablation (my preference) in my office and must confess that after a long week of intense cardiac and vascular interventions, I look forward to “lead-free” Fridays! ■

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