Center for Vein Restoration

VENOUS Review

THE OFFICIAL JOURNAL OF CENTER FOR VEIN RESTORATION

Vol. 5, Issue 1

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Relation Between Superficial Vein Reflux and Deep Vein Disease: Clinical Impact of Modern Treatment

by Nicos Labropoulos, PhD, DIC, RVT, Professor of Surgery and Radiology, Stony Brook Universtiy Medical Center. and Antonios P. Gasparis, MD, Associate Professor of Surgery, Director, Stony Brook Vein Center

Chronic venous disease (CVD) is the most prevalent form of vascular disease, affecting about a third of adult Americans. Most patients present with varicose veins but swelling and skin damage are common as well. The signs and symptoms of CVD are result of venous hypertension that develops from reflux, obstruction or a combination of both. In patients with CVD, around 70-80% will have superficial reflux with or without perforator vein incompetence and 10% isolated deep reflux. Combined superficial and deep vein disease has been reported to occur in about 10-20%. Congenital CVD and chronic venous obstruction are rare *(Figure 1)*. While treatment of the isolated superficial venous system in patients with CVD is expected and has been shown to have good outcomes, the role, efficacy and complications of superficial intervention in patients with combined disease has been controversial. In patients with mixed pathology, deep venous obstruction occurs in a small number of limbs, which is either primary or secondary. The majority though, will have underlying reflux which will be segmental and is likely to occur due to volume overload from the recirculating reflux blood in the superficial veins. However, axial vein reflux, which is most often a result of previous thrombosis, also is seen, as about two thirds of patients with proximal vein thrombosis will develop reflux at one year. Patients with a previous episode of thrombosis may have vein segments without or with partial recanalization leading to obstruction or to a combination of reflux and obstruction.

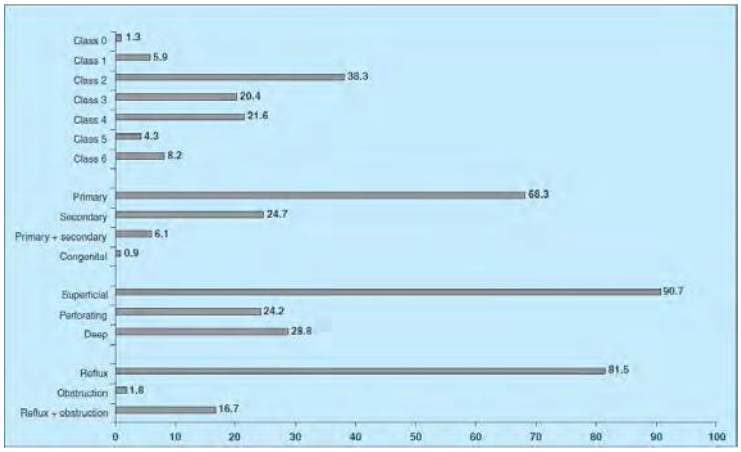

Figure 1. Presentation of 1000 consecutive limbs with CVD according to CEAP classification. In the anatomic classification for simplicity only the overall contribution of each system is shown. This classification has allowed better communication and comparisons in the medical literature as CVD terminology is more specific and has been adopted worldwide.

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ISSN 2159-4767 (Print), ISSN 2159-4775 (Online)

Thrombophilia: Patients Who Clot Too Much

Part 2: Acquired Thrombophilia

by Robert C. Kiser, DO, MSPH

In the last article (Venous Review, Vol. 4, No. 4), we discussed congenital thrombophilias. However, thrombophilia can develop at any time in one's life. Generally this is due to development of autoantibodies against specific factors. This requires both a genetic component (the capacity to manufacture certain autoantibodies in response to specific environmental stressors) and an environmental component (the exposure to a substrate or antigen that prompts the manufacture of the autoantibody). The following is by no means an exhaustive list, but merely the more commonly encountered of the acquired thrombophilias.

Pregnancy and Hormone Treatment

Pregnancy is a state that predisposes to increased coagulation. Risk factors include left leg symptoms, symptoms of swelling, and swelling causing a greater than 1cm difference in calf circumference. Anatomically, the left lower-extremity venous system is mechanically predisposed to compression at the left common iliac vein by the right common iliac artery, which may or may not explain the left-sided predisposition found in one study.[1] Likewise, treatment with estrogen-containing medicines such as oral contraceptive pills increases clotting. The survival advantage for a woman clotting more than usual during and after child birth is the reduction in bleeding during and immediately after child birth.

Anti-phospholipid Antibody Syndrome

Graham Hughes first described Anti-phospholipid antibody syndrome (APLAS) in the British Medical Journal in 1983. The syndrome occurs with the development of antibodies such as lupus anticoagulant and anticardiolipin antibody as well as antibodies to b2-glycoprotein.

People with APLAS may develop venous or arterial thrombosis, and hence are at increased risk of DVT and PE as well as myocardial infarction (MI) and stroke. Additionally, APLAS is associated with an increased risk of miscarriage, especially in the first 12 weeks of pregnancy and in pre-eclampsia. Although the risk of APLAS is increased in those with systemic lupus erythematosus (SLE), most people with APLAS, even those who have the so-called lupus anticoagulant, do not have systemic lupus. APLAS requires two positive tests, at least six weeks apart (some authors say 12 weeks).[2] Tests for APLAS should include:

- Anticardiolipin antibodies (IgG, IgM, and IgA)
- Lupus anticoagulant – a panel of blood clotting tests that may include the dilute Russell Viper venom time (dRVVT), lupus aPTT, mixing studies, and hex-phase phospholipid test or platelet neutralization procedure
- Antibodies to b2-glycoprotein I (IgG, IgM, IgA)

Myeloproliferative Disorders

Myeloproliferative disorders, which can lead to thrombocytosis, are also at increased risk of clotting. Paroxysmal nocturnal haemoglobinuria is a rare disorder that is associated with increased thrombosis.[3]

Inflammatory Bowel Disease

Other diseases are also associated with increased risk of clotting. Inflammatory bowel diseases (ulcerative colitis and Crohn's Disease) are correlated with an increased risk of venous thromboembolism (VTE).[4] The precise mechanism of IBD associated thrombophilia has not been elucidated.[5]

Cancer

It has long been recognized that cancer is associated with an increased risk of VTE, especially lung, kidney, brain, gastrointestinal, female reproductive system, and leukemia and lymphoma.[6] Treatments for cancer, including surgery and chemotherapy, also increase the risk of thrombosis.[7]

Transient Protein C & S Deficiency

Because protein C & S are vitamin K dependent factors, vitamin K antagonists (such as warfarin) create a transient factor C & S deficiency during the first few days of exposure to warfarin. It is for this reason that a second method of anticoagulation (most commonly heparin) should be prescribed for at least the first five days of warfarin treatment, even if the INR is therapeutic before the fifth day. Failure to do so increases the risk of thromboembolic adverse events such as warfarin induced skin necrosis, arterial and venous thrombosis and embolism.[3]

Heparin-induced Thrombosis

Up to 3% of patients given unfractionated heparin, and up to 1% of patients given low molecular-weight heparin (LMWH) will develop heparin-induced thrombosis or HIT. Untreated, the mortality rate is 20% with an additional 10% requiring amputations. Currently there is no reliable, widely available method of predicting who will develop HIT, excepting those patients who have previously developed HIT. Those who have developed HIT in the past should not be rechallenged with heparin or heparinoids. The syndrome tends to develop after approximately 5-10 days of heparinoid exposure and is characterized by the onset or worsening of thromboembolic events despite anticoagulation. Treatment requires not only withdrawing the heparin but also immediately starting a direct fibrin inhibitor such as Argatroban or leiperidine. Consultation with a hematologist is important and useful.[8]

Assessing for Increased Risk

Thrombophilia is a complex syndrome, with diverse etiologies that can be either congenital or acquired. It is also possible for a person to have both congenital and acquired thrombophilias. Regardless of the cause, people with known thrombophilia should be assessed for increased risk when they are undergoing or likely to be exposed to circumstances that may further increase their risk of clotting, this included pregnancy, prolonged immobility, surgery, cancer, chemotherapy, trauma, certain medical diagnoses, and hormone replacement therapy. When any of these factors provoke a thrombotic response in someone without a known thrombophilia, consideration should be given to a thorough work-up for a preexisting thrombophilia, including taking a personal and family history and laboratory tests when appropriate.

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[6] ibid.

[7] Caprini, J Thrombotic Risk Assessment: A Hybrid Approach. http://www.venousdisease.com/Publications/JACaprini-HybridApproach3-10-05.pdf

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Relation Between Superficial Vein Reflux and Deep Vein Disease: Clinical Impact of Modern Treatment

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Segmental deep-vein reflux due to superficial vein incompetence is most often seen at the saphenofemoral (SFJ) and saphenopopliteal junctions (SPJ). It also can be due to reflux in the gastrocnemius

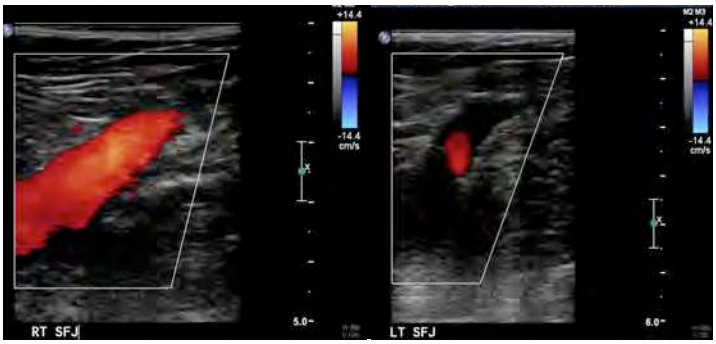

Figure 2. Bilateral SFJ reflux in a male patient who presented with long standing varicose veins, swelling and mild skin discoloration. He had no history of thrombosis and after detail imaging with ultrasound no signs of previous thrombosis were found. There is reflux in the right proximal common femoral vein in continuity with the SFJ. The common femoral vein distal to the SFJ is normal. As it is discussed in the text such deep vein reflux is abolished after eliminated the GSV reflux. On the left side there is reflux in the SFJ without affecting the common femoral vein.

veins (almost always the medial) and perforator veins (Figure 2). The reflux in the perforator veins is not isolated but it occurs only in the presence of reflux in superficial veins that are connected with the affected perforators. Longstanding reflux in the superficial and perforator veins may lead to reflux development in the deep veins that are connected with the affected perforators. This is the same phenomenon as in the case of SFJ, SPJ and gastrocnemial veins. Deep vein reflux which is induced by the superficial vein incompetence is easily eliminated in >95% of patients after treating the superficial veins. This has been demonstrated in different studies and it does concur with our experience as well. It has to be noted that many patients may have deep vein reflux due to previous

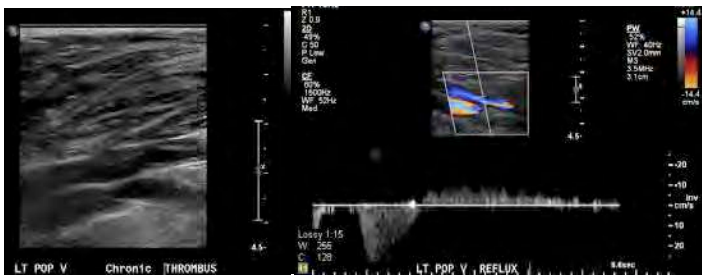

Figure 3. Axial deep vein reflux from the common femoral to the calf veins in a male patient who presented with skin damage after recurrent episodes of deep vein thrombosis in the left lower extremity. Skin damage is seen more often in patients with recurrent episodes of thrombosis and in those with both reflux and obstruction as in the current patient. The image on the left panel shows the chronic intraluminal changes in the popliteal vein with prominent wall thickening and intraluminal fibrous tissue. The image on the right demonstrates significant reflux on the Doppler waveform.

thrombosis. In such occasions the deep vein reflux may improve in some cases after elimination of the superficial reflux, but it does not disappear. The latter is more prevalent in the presence of axial reflux, such as when there is reflux in continuity from the common femoral vein to popliteal or more distal (Figure 3).

Traditional advice has been against saphenous ablation in the presence of deep venous obstruction. This was felt because of the thought that secondary saphenous varices result from deep venous obstruction and function as collaterals. Therefore, it was feared that obstructive disease may be made functionally more severe by removing the saphenous vein that may be functioning as important collateral. When testing venous outflow function in patients with previous deep vein thrombosis, Labropoulos et al. demonstrated that only 9.6% of limbs had their venous outflow reduced by occlusion of the superficial veins. The deep collaterals seem to be more important than the superficial venous system in bypassing the obstruction. Raju et al.,when comparing patients with and without deep obstruction who underwent saphenectomy, found similar outcomes in the two groups with no clinical deterioration in those with obstruction. In addition, the risk of DVT following saphenous ablation does not seem to be increased in patients with previous thrombotic events as shown by Puggioni et al. Therefore, the saphenous vein plays an insignificant role as a collateral pathway in patients with deep venous obstruction and can be safely treated to correct underlying hemodynamic pathology.

In patients with advanced CVD (C4-6) and superficial reflux, interrogation of the deep venous system for proximal obstruction, even in the absence of previous DVT, is warranted. Marston et al. found that as many as 30% of patients with chronic venous insufficiency have iliac vein obstruction on CT scan. When obstruction is in the iliac veins, consideration for its significance and intervention should be considered. The presence of such combined disease (superficial reflux and iliac vein obstruction) may warrant treatment of both levels of disease, as it is impossible to identify the pivotal diseased segment that contributes to the clinical presentation. In fact, we are presently unable to quantify segmental reflux or obstruction or describe how they interact. It is, therefore, reasonable to treat the superficial reflux and the proximal obstruction with iliac vein stenting. This can be done in a staged or combined fashion. Neglen et al. reported combined saphenous ablation and iliac vein stenting in 99 limbs with significantly improved hemodynamic parameters, improved clinical symptoms (pain and swelling) and significant improvement in all quality-of-life categories after treatment. This was achieved with good 4-year patency (>90%) and low complication rate.

Patients with mixed superficial and deep pathology and significant clinical symptoms (especially C4-6) should be offered not only treatment of their superficial system but also evaluation and therapy of any underlying iliac vein obstruction with excellent clinical outcomes. When evaluating the superficial veins in patients with deep vein obstruction it is necessary to demonstrate significant reflux in the superficial veins prior to intervening. This is very important as superficial veins can be dilated in order to compensate for the deep vein obstruction. In such patients the diameter change should not be compared with the studies on patients with primary superficial vein reflux. Therefore, superficial veins with large diameter should not be removed unless there is significant reflux that may contribute to the patients' signs and symptoms. Patients with deep vein obstruction are evaluated in the supine position. However, when these patients are tested for reflux, this must be done in the standing position. This is paramount as reflux should be evaluated in the standing position in all patients. We overemphasized this for the patients with venous obstruction as many centers still examine reflux in the supine position in such patients.

Continued on Page 4

New CMEs Announced

Venous insufficiency, the often undiagnosed medical condition behind varicose veins and spider veins (telangliectasia) affects a broad range of people – statistically as much as 10 to 20 percent of the population, including patients in your practice. Common causes include heredity, age, sex, weight, history of DVT, pregnancy, inactivity and prolonged sitting or standing. Certain groups, such as expectant moms, have extra difficulty – studies show that varicose veins get worse with each subsequent pregnancy.

To help physicians like you address this increasingly prevalent condition, we're expanding our offering of CME courses on venous insufficiency. In these informative and relevant 3-hour sessions, our physician presenters will describe causes, symptoms, diagnosis and treatment of venous insufficiency. Participants will learn about the staging/classification of venous insufficiency, how duplex ultrasonography is used to evaluate patients and how modern, outpatient treatments including radiofrequency and laser ablation, and foam and cosmetic sclerotherapy can make addressing varicose and spider veins quick and relatively painless so your patients can return to normal activity with short recovery times.

CME course offerings for April and May are listed here. To learn more or to request a CME in a region we serve, please contact Brent Matherly at 443-370-3830 or 301-860-0930 and at brent.matherly@centerforvein.com.

CME Courses & Speaking Engagements

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Falls Church, VA 22046

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kristin.rekucki@centerforvein.com
(703) 626-1906

Maryland

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RSVP by April 10
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Laine.haley@centerforvein.com
(410) 991-8074

Glen Burnie
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RSVP by April 17
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heather.buck@centerforvein.com
(443) 534-8578

May

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Thursday, May 3
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Edgewood, MD 21040

RSVP by April 25
Christina Pierelli, Physician Liaison
christina.pierelli@centerforvein.com
(800) 349-5347

Silver Spring
Monday, May 21
Tavira Restaurant
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Chevy Chase, MD 20815

RSVP by May 11
Kathy Rewa, Physician Liaison
kathy.rewa@centerforvein.com
(800) 349-5347

District of Columbia

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Thursday, May 17
De Carlos Restaurant
4822 Yuma Street NW
Washington, DC 20016

RSVP by May 9
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andrea.perez@centerforvein.com
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[Top Row-L to R] Luis A. Dibos, MD, FACS, Thomas C. Militano, MD, FACS, Khanh Nguyen, DO, Frank Sbrocco, MD, J. Andrew Skiendzielewski, DO, Arvind Narasimhan, MD. [Bottom Row-L to R] Rory C. Byrne, MD, Jerrilyn M. Jutton, MD, FACS, Sanjiv Lakhanpal, MD, FACS, Shekeeb Suan, MD, FACS, Stephan Corriveau, MD. [Inset] Jaime F. Marquez, MD, FACS, PA. (Not shown: Eddie Fernandez, MD, Paul Johnson, MD, Robert C. Kiser, DO, MSPH, Sean K. Stewart, MS, MD; Patricia Fedorchak, MS, CRNP; Kathleen Petro, MD.)

New Centers Announced

We're proud to announce the recent opening of three new Center for Vein Restoration locations, bringing the number to 17 clinics serving patients coping with vein disease in Maryland, Virginia, Washington, DC and Michigan. Meanwhile, additional clinic locations will be announced later in 2012. Details of our newest centers are below.

- ***Germantown, Maryland***

The Germantown Center is located at 19785 Crystal Rock Dr., Suite 310. Our team can be contacted by phone at (301) 515-7203.

Woodbridge, Virginia

Fairfax, Virginia

- ***Fairfax, Virginia***

Our Center in Fairfax is located at 8316 Arlington Blvd. Suite 610B. Our team can be contacted by phone at (703) 289-1122.

- ***Woodbridge, Virginia***

The Woodbridge Center is located at 2200 Opitz Blvd, Suite 320. Our team can be contacted by phone at (703) 490-8585.

Detailed information on all local Centers can be found on our web site, www.centerforvein.com. Patients can be referred easily via our "Doctors Referral" button on the Web site home page or by phoning our one-stop call center at 800-FIX-LEGS.

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Woodbridge, VA 22191
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Fax: (703) 490-8484

Michigan

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Portage, MI 49024
Ph: (269) 323-8000
Fax: (269) 323-8003

From The Editor

VENOUS Review
The Official Journal of Center for Vein Restoration

Editor-in-Chief, President & CEO,
Center for Vein Restoration
Sanjiv Lakhanpal, MD, FACS

Editor,
Robert C. Kiser, DO, MSPH

As spring approaches we often think of the future, and of re-committing to the important things in life, be they family, career or the health. At Center for Vein Restoration, we're excited to announce that we're more committed than ever to providing the best possible care for patients in as many communities as possible. And we're excited to announce progress in several areas.

First, we're proud to have grown to 17 locations with the recent opening of Germantown, Md., Fairfax/Merrifield, Va. and Woodbridge, Va. Centers. And, we'll be announcing more locations in the coming months.

Second, we're pleased to offer more CME courses to our local physicians on the topic of venous insufficiency. We have several CMEs planned in March and April in Michigan, Maryland and Virginia. We invite you to join us for one of our upcoming 3-hour sessions to learn about diagnosis and treatment of venous insufficiency – which affects 10-20 percent of the population – ranging from conservative measures to advanced, outpatient modalities like radio frequency and laser ablation, and both ultrasound-guided foam sclerotherapy and cosmetic sclerotherapy.

And finally, we're proud to be reaching another milestone as we enter the fifth year of publication of Venous Review. As you'll doubtless read, we're continuously working to provide a variety of relevant and informative articles on many aspects of vein health. We hope you enjoy these articles and that you find them useful in your own practice.

On behalf of everyone at Center for Vein Restoration, thank you for your continued interest and we wish you a happy, healthy and fruitful spring.

Regards,

Robert C. Kiser, DO, MSPH
Editor

Visit our website: www.centerforvein.com

Center for Vein Restoration

12200 Annapolis Road, Suite 225
Glenn Dale, MD 20769