Varicose and Telangiectatic Leg Veins

Principles for successful sclerotherapy treatment of varicose and telangiectatic leg veins.

BY MITCHEL P. GOLDMAN, MD

It has been estimated that between 10% and 20% of adults in the US and western Europe have varicose veins, and up to 50% of women by age 50 will have telangiectatic leg veins. Although most patients who present for treatment do so for cosmetic reasons, up to 50% of patients with varicose veins will develop symptoms or adverse sequelae, including superficial thrombophlebitis and leg ulceration. The difference between varicose and telangiectatic leg veins is size. By convention, tortuous veins >4 mm to 5 mm in diameter are referred to as *varicose*, veins between 1 mm and 4 mm in diameter are referred to as *reticular*, and veins <1 mm in diameter are referred to as *telangiectasia*.

PATHOPHYSIOLOGY

The most common predisposing factor for the development of unwanted leg veins is family history. Many other factors may also contribute to the development of varicose veins. Any activity or condition that increases abdominal pressure and impedes the flow of venous blood back to the heart may put strain on the veins, causing them to dilate. This includes standing for long periods of time, lack of exercise, obesity, constipation, wearing high-heel shoes (which requires one to walk using the buttock muscles and not the calf or foot muscles), and wearing tight undergarments or pants (producing a tourniquet effect on the proximal superficial veins). The use of estrogen and/or progesterone hormone supplementation for birth control or postmenopausal symptoms also causes a dilatation of the vein wall. Localized trauma (being hit with a tennis ball or other object) may initiate angiogenesis with the eruption of telangiectasia. Localized trauma may also damage perforating veins, leading to an increase in blood flow from the deep to superficial circulations. Photo damage from the sun or other forms of radiation are also associated with an increased incidence of telangiectasia through degradation of the elastic and collagen network surrounding telangiectasia. Finally, pregnancy puts strain on veins by increasing blood volume, increasing estrogen and progesterone levels, and impeding blood flow through compression of the pelvic venous system. Varicose and/or reticular veins and/or telangiectatic veins appear in one third of patients before age 25 and increase in incidence with age, with 70% of the population having visible cutaneous veins by age 70.

Varicose and telangiectatic veins arise from and may also be a cutaneous marker for an underlying reversal of venous blood flow known as *venous insufficiency*. Regardless of the cause, this reversal of blood flow produces venous hypertension in the lower extremities, which in turn produces an increase in venous diameter that leads to valvular insufficiency. This results in a reversal of blood flow from the deep to the superficial venous system through incompetent communicating veins. The superficial veins respond to increased pressure by dilating to accommodate the increased blood flow. In addition, with movement of the lower limbs, the high venous pressure that normally occurs within the calf muscle pump is transmitted straight to the superficial veins and subcutaneous tissues drained by these communicating veins. When this occurs, the venous pressure in the superficial vascular system increases and telangiectasia appear.

LABORATORY FINDINGS AND DIAGNOSIS

Regardless of the underlying etiologic event leading to venous hypertension, the point of reflux—whether it is through either incompetent perforating veins, feeding reticular veins, or an incompetent saphenofemoral junction—must be treated first. A number of diagnostic tests can be performed to help define the extent of varicose vein disease and to plan treatment. The primary goal is to locate the point of high-pressure reflux. This is easily accomplished with duplex ultrasound scanning, which allows visualization of the abnormal vein and its connections. A complete discussion on the relative merits of each technique is found elsewhere.[1]

TREATMENT

Depending on the cause and extent of disease, surgery—sclerotherapy, laser, radiofrequency ablation, or intense

TABLE 1. ADVANTAGES AND DISADVANTAGES OF SCLEROSING SOLUTIONS

Solution	Pigment	Allergy	Necrosis
Glycerin	–	–	–
STS	+	+	–
POL	+	+	+
HS	+	–	++

STS, sodium tetradecyl sulfate; POL, polidocanol; HS, hypertonic saline.

TABLE 2. RECOMMENDED SOLUTIONS AND CONCENTRATIONS FOR DIFFERENT-SIZED VEINS

<1 mm
72% glycerin; STS .25%; POL foam .5%; HS 11.7%

1 mm to 4 mm
STS foam .25%-.5%; POL foam .5-1%

4 mm to 10 mm
STS foam 1%-3%; POL foam 2-5%

STS, Sodium tetradecyle sulfate; POL, polidocanol; HS, hypertonic saline.

pulsed light, or a combination of all techniques is—necessary. Surgical ligation and stripping procedures are essentially a procedure of the past. For varicose veins >1 cm in diameter and for patients with an incompetent great saphenous vein or small saphenous vein, sclerotherapy is usually ineffective except in the most expert of hands. Very strong sclerosing solutions are necessary to destroy the vein, and a phlebitic reaction usually occurs, resulting in a thrombus cord formation and overlying pigmentation of the skin. The procedure usually needs to be repeated two to four times, but even with initial closure of the vein, recurrence usually occurs within 5 years from persistent high-pressure reflux. I recommend endoluminal laser closure. This technique is performed under local tumescent anesthesia with the patient immediately ambulatory after the procedure. In our experience, it causes minimal—if any—pain, and patients can resume all normal activities within 24 hours. We recommend the 1320-nm endoluminal laser, although lasers of other wavelengths and radiofrequencies are also used.[2]

For veins 4 mm to 10 mm in diameter, ambulatory phlebectomy is my treatment of choice. Veins of this size are removed through 2-mm to 3-mm incisions without the chance of recurrence because they are on the surgical tray and not within the patient; there is also a decreased risk of adverse sequelae.[3] A beneficial effect of ambulatory phlebectomy is the harvesting of the veins that are essentially collagen tubes. These can be transferred to areas that need filling, such as the nasolabial groves and lips, saving the patient the cost of using temporary or artificial filling substances.[4]

Sclerotherapy is recommended for veins <4 mm in diameter. For veins <1 mm in diameter that are persistent after sclerotherapy and phlebectomy, laser or intense pulsed light treatment may also be used.[1-5]

SCLEROTHERAPY

Mechanism of Action

Sclerotherapy is defined as the introduction of a foreign substance into the lumen of a vessel, causing thrombosis and subsequent fibrosis. Extensive reviews of the mechanism of action for specific sclerosants appear elsewhere.[1] In short, detergent sclerosants, such as sodium morrhuate, sodium tetradecyl sulfate, ethanolamine oleate, and polidocanol, produce endothelial damage through interference with the cell surface lipids. Hypertonic saline and hypertonic glucose solutions produce dehydration of endothelial cells through osmosis, resulting in endothelial destruction. Chemical irritants or caustic agents, such as glycerin and polyiodinated iodine, produce direct destruction of the endothelial cells. The advantages and disadvantages of each solution are listed in Table 1. The recommended concentration of sclerosing solution for various types and diameters of sclerosing solutions is found in Table 2. In the US, Sotradecol (Bioniche Life Sciences Inc., Belleville, Ontario, Canada, distributed by AngioDynamics, Queensbury, NY) is the only FDA-approved sclerosing agent. Hypertonic saline and glycerin solutions are also widely used for veins <1 mm in diameter. It is illegal to import other sclerosing solutions from foreign countries, and studies have shown no benefit of foreign sclerosing solutions over Sotradecol. I also do not advise using compounded detergent sclerosing solutions because they have been found to contain a variety of contaminants that could pose both short-term and long-term health problems.[6]

Injection Technique

Sclerotherapy should progress from the largest to smallest vessels. The injected quantity of solution should be enough to fill the vessel and displace intravascular blood. When you stop seeing the solution flowing, you should stop the injection because this means that the solution is flowing into the deeper venous system. To avoid the risk of initiating new telangiectasias from forming around the edge of the treated area, the minimal effective amount of sclerosing concentration should be used. This will minimize the resulting inflammation. It is important to remember that sclerotherapy is a controlled thrombophlebitic reaction. The entire venous system of each leg is treated at one time to avoid leaving areas of refluxing blood flow from causing recanalization of the treated vessel or extravasation of red blood cells from the damaged vessel, which leads to hyperpigmentation.

TABLE 3. COMPLICATIONS AND ADVERSE SEQUELAE OF SCLEROTHERAPY

- Hyperpigmentation
- Temporary swelling
- Telangiectatic matting
- Pain with injection
- Localized urticaria
- Tape compression blister
- Tape compression folliculitis
- Recurrence
- Vasovagal reflex
- Localized hirsutism
- Cutaneous necrosis
- Allergic reaction
- Superficial thrombophlebitis
- Arterial injection
- Pulmonary embolism and DVT
- Nerve damage
- Migraine headaches
- Scotoma

I recommend using a foamed sclerosing solution for all veins >1 mm in diameter. Foaming a sclerosing solution can only be done with a detergent sclerosing solution, such as Sotradecol. I use 1 mL of solution to 4 mL of air. Foaming a sclerosing solution increases its effective sclerosing power by two while decreasing its adverse effect profile by four because the solution is diluted fourfold by air. There are few adverse effects from foaming a sclerosing solution, and certainly the use of foam does not add to any other risk of sclerotherapy. It has been stated with very little support that the air in foam has some risks. Proper evaluation of the literature demonstrates that air-generated foam has very little, if any, additional risk than the use of a liquid solution. A complete discussion on foam sclerotherapy is found elsewhere.[1,7,8]

Patients should be examined 2 weeks after injection so that any area of thrombosis (representing trapped blood and always called a *coagulum* to the patient) can be evacuated early. Evacuation of the coagulum will minimize the appearance of hyperpigmentation and speed resorption of the destroyed blood vessel. The treated area should not be re-treated sooner than 6 to 8 weeks after injection to allow for resolution of inflammation between treatments. The patient is instructed to walk immediately after the injection session to help prevent deep vein thrombosis and constrict the superficial and perforating veins. Calf muscle movement produces a rapid blood flow in the deep venous system that dilutes out any sclerosant that may have migrated to this area. Following injection of all varicose or telangiectatic veins, the treated veins are compressed to minimize significant thrombosis.

Posttreatment Compression

Postsclerotherapy compression, initially described by Sigg[9] and Orbach[10] in the 1950s and Fegan[11] in the 1960s, is perhaps the most important advance in sclerotherapy for varicose veins since the introduction of relatively safe synthetic sclerosing solutions (eg, sodium tetradecyl sul-

fate) in the 1940s. Primarily, compression eliminates a thrombophlebitic reaction and substitutes a sclerophlebitis with the production of a firm fibrous cord. Compression serves at least five purposes. First, compression, if adequate, may result in direct apposition of the treated vein walls to produce a more effective fibrosis. Therefore, sclerosing solutions of lesser strength may be successfully used. Second, compressing the treated vessel will decrease the extent of thrombus formation that inevitably occurs with the use of all sclerosing solutions, thus decreasing the risk for recanalization of the treated vessel. Third, a decrease in the extent of thrombus formation may also decrease the incidence of postsclerotherapy pigmentation. Fourth, the limitation of thrombosis and phlebitic reactions may prevent the appearance of angiogenesis/telangiectatic matting. Finally, the function of the calf muscle pump is improved by the physiologic effect of a graduated compression stocking. By externally supporting untreated large veins, compression stockings will narrow vein diameter, restoring competency to its valvular function, which decreases retrograde blood flow. External pressure will also retard the reflux of blood from incompetent perforating veins into the superficial veins. In short, graduated compression sclerotherapy is now the standard practice for the treatment of varicose and telangiectatic leg veins.

In the majority of cases, telangiectatic spider veins are in direct connection to underlying varicose veins, either directly or through tributaries. Therefore, like varicose veins, treatment should first be directed to plugging the leaking high-pressure outflow at its point of origin. An appropriate analogy is to think of spider veins as the fingers and of the feeding varicose vein as the arm. Treatment should first be directed to the feeding arm and, only if necessary, directly to the spider fingers. There are a number of advantages to this systematic approach to sclerotherapy. When performed in this manner, the spider veins often disappear without their direct treatment, thus limiting the number of injections into the patient. Another advantage is that the larger feeding vein is both easier to cannulate and less likely to rupture with injection of the sclerosant, thus minimizing the extent of extravasated red blood cells and sclerosing solution. Compression of leg veins with a graduated compression stocking even <1 mm in diameter has been shown to minimize the development of postsclerotherapy hyperpigmentation, cutaneous necrosis, and telangiectatic matting.[12]

Microsclerotherapy

Microsclerotherapy of spider veins is performed using a standard technique. The patient is placed in a supine position. Gravitational dilatation of telangiectasias is unnecessary. A 2 power loupe for magnification is helpful to aid visualization. The goal of microsclerotherapy is to cannulate the vessel so that the sclerosing solution will be deposited within and not outside the vessel wall. A 30-gauge needle is sufficient. For vessels <1 mm in diameter, I have found that a 72% glycerin solution mixed 2:1 with 1% lidocaine with epinephrine is best. With this sclerosing solution, there is virtually no risk of ulceration, pigmentation, and telangiectatic matting. In addition, resolution of the veins appears better than with a detergent solution.[13]

COURSE AND PROGNOSIS

Unfortunately, as with any therapeutic technique, sclerotherapy carries with it a number of potential adverse sequelae and complications (Table 3). However, these complications are quite rare. Fairly common adverse sequelae include temporary perivascular cutaneous pigmentation and a temporary flare of new perivascular telangiectasias. Relatively rare complications include localized cutaneous necrosis, thrombophlebitis of the injected vessel, arterial injection with resultant distal necrosis, and pulmonary emboli. These complications and ways to minimize them are discussed in detail elsewhere.[1] ■

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