Sclerotherapy for Telangiectasia: The Impact of Small Changes in Vessel Diameter Upon Treatment Outcomes

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Because lower extremity telangiectasia is often considered to be nothing more than a cosmetic nuisance, almost no attempts have been made to identify clinical features which reproducibly affect treatment outcomes. Long-term observations suggest that very small changes in vessel diameter (which are easily assessed using magnification and a measuring device specifically designed for that purpose) along with the awareness of the long-term impacts of previous treatments are particularly useful for predicting treatment outcomes. Telangiectatic etiologic heterogeneity, associated comorbidities, and several other poorly characterized variables are also discussed.

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LOWER EXTREMITY TELANGIECTASIA: A CLOSER LOOK

Although simplistically defined as veins no larger than 1 mm in diameter, a definition which deceptively implies homogeneity, telangiectasia is in fact an extremely heterogeneous group. Telangiectasia differ from one another in terms of clinical appearance, response to sclerotherapy, etiology, and highly unpredictable sensitivity to sclerosants. The type and timing of complications are also quite variable. Telangiectasia which occur in large numbers around the ankles (corona phlebectasia) often represent a recognizable biomarker for venous hypertension and reflux. Although true recurrences following adequate treatment are uncommon, the dynamics of posttreatment proliferation and involution (vascular remodeling), persistence of improvement, and the long-term impact of previous treatments and venous hypertension and reflux are also extremely variable. They vary both from one person to another as well as between different vessels of the same size on the same person. This baffling array of disparate outcomes following "identical treatments," for veins that "look exactly alike," has provoked both an aura of therapeutic nihilism and a number of treatment strategies which are thought to improve treatment outcomes. These include the injection of reticular veins, vigorous compression, and doppler and duplex investigations to determine the presence of venous hypertension or reflux. Current dogma suggests that reflux and venous hypertension are the root "cause" of telangiectasia. My experience suggests this is not always true.

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MEASURING TELANGIECTASIA: SMALL CHANGES IN SIZE, BIG CHANGES IN RESPONSE TO TREATMENT

Although a multiplicity of variables may affect treatment outcomes, careful pretreatment measurement of telangiectasia in 0.1-mm increments using magnification and a ruler specifically designed for that purpose (available at www.drdavidmduffy.com) demonstrate the existence of distinct *intrinsic* patterns of treatment response associated with miniscule changes in vessel diameter which are often too small to be appreciated with the naked eye. No matter how telangiectasia are treated, 3 patterns (fast, slow, and resistant) emerge which can often be predicted solely on the basis of very small changes in vessel size and in certain cases the performance of previous treatments or the presence of reflux or venous hypertension. The very smallest vessels (under 0.1–0.3 mm in diameter) usually respond gradually to repeated treatments or are resistant. Posttreatment pigmentation or palpable thrombi are extremely rare. Larger telangiectasia, reticular veins, and varicose veins often respond abruptly. This type of response is often followed by hemosiderotic hyperpigmentation and palpable thrombi. The occurrence of these complications and the rapidity of treatment results can sometimes be modified by increasing or decreasing sclerosant concentrations or injecting reticular veins and the use of compression. Resistance can occur following the treatment of vessels of any size.

TELANGIECTATIC RESISTANCE

In the case of telangiectasia, resistance to sclerotherapy should be viewed in several contexts. Certain individuals with or without treatment are insensitive to ordinarily used concentrations of sclerosants. In patients undergoing treatment for the first time this can sometimes be overcome by increasing sclerosant concentrations or repeated treatments. A second type of resistance involving telangiectasia under 0.3 mm in size commonly occurs in patients who have received previous treatments. Its occurrence may involve the disabling of apoptosis or excessive angiogenesis. This type of vessel has been called matting, neovascularization, and microtelangiectatic flaring. It can be intuitively viewed as a form of aberrant collateral circulation. For this type of vessel, increasing the concentrations of sclerosants is counterproductive.

LARGE VESSEL RESISTANCE

Another type of resistance[1] occurs when larger vessels, both telangiectasia and other types of vessels, do not respond. Increasing sclerosant concentrations may prove effective for treating larger vessels; treatment of underlying venous hypertension and reflux can prove effective for smaller ones.

Vessel Size: The Effects of Biomass

When treating tiny veins, a 0.1-mm change in diameter may substantially increase or decrease vessel biomass and alter expected treatment outcomes. This effect may be analogous to treatment response alterations associated with microscopic changes in tumor thickness. Treatment outcomes following treatment of telangiectasia can vary dramatically with small changes in their diameter. These small changes are associated with abrupt versus slow outcomes as well as the occurrence of pigmentation and palpable thrombi. In previously treated patients, the increase in resistance is confined generally with vessels under 0.3 mm in size. Larger vessels occurring or remaining after previous treatments are rarely resistant. The treatment of larger telangiectasia (over 0.5 mm in diameter) is often followed by pigmentation. Vessels of this size may often require only one treatment.

SHORTCOMINGS

Although precise measurements and a careful patient history can provide an effective way of predicting specific *patterns* of treatment response, they are not foolproof prognosticators of treatment outcomes, optimal concentrations, or type of sclerosants. There is no perfect way to establish treatment protocols for any particular patient or single vein. The inability of phlebologists to establish uniform treatment protocols reflects poorly understood differences between patients and the impossibility of standardizing individual treatment techniques.

Shortcomings notwithstanding, careful measurement of telangiectasia is a small first step in unraveling the eccentricities of treatment outcomes, which may become clearer when the true identity and importance of factors other than vessel size and previous treatments are recognized, prioritized, and codified.

LIMITATIONS AND TRADE-OFFS

Comparing Varicose Veins to Telangiectasia

Attempts to apply principles derived from the treatment of varicose veins to the treatment of telangiectasia are fundamentally flawed. Chief among these is the notion that as vessels become larger in diameter, sclerosant concentrations must be increased. Unfortunately, telangiectasia do not always respond to treatment like tiny reticular or varicose veins in which resistance can often be overcome by increasing sclerosant concentrations. Dilutional considerations do not apply to eyelash-sized, highly resistant microtelangiectasia (matting, neovascularization). These types of telangiectasia probably represent the effects of vascular remodeling which occurs following previous treatments and can persist for years. The development of this type of vasculature is *promoted* by the use of higher

sclerosant concentrations. Posttreatment telangiectasia in a certain size range is routinely unaffected or worsened by the use of sclerosants capable of destroying large refluxing varicose veins. Even more perversely, as telangiectasia becomes slightly larger and protuberant (a finding often noted in older patients), it is much more fragile than very small telangiectasia and may respond abruptly to lower concentrations of sclerosants. Reticular veins may also be more fragile than very small telangiectasia and prone to immediate destruction associated with thrombi and pigmentation, even when mild or dilute sclerosants which are ineffective for smaller telangiectasia are employed. The upshot, different types of telangiectasia have to be treated on an individual basis. The margin between effective treatment and increased complications is relatively narrow.

TELANGIECTASIA BY THE NUMBERS

0.1–0.2 mm in Diameter

Previously untreated (virgin) microtelangiectasia in this size range are rarely resistant. They typically respond gradually over several months, a process which is rarely accelerated when employing higher sclerosant concentrations. The use of higher concentrations *does* promote neovascularization and subsequent resistance. Vessels of this size rarely develop thrombi or hyperpigmentation even when employing more potent sclerosants (Figure 1). Two to 3 treatments are often the rule, and sometimes more when large numbers of telangiectasia must be treated. The full effects of each treatment develops gradually and is maximal at 4 to 6 weeks; accordingly, treatments are routinely carried out at that interval. Patients must be warned that the full benefits of these treatments may not be obvious for several months. Extremely dilute sclerosants can sometimes produce excellent results more slowly, with less risk of neovascularization but more treatment failures. Compression, and the injection of reticular veins, and the elimination of venous reflux, may be quite effective in some patients, in others they may not. There is no particular treatment method that guarantees better or longer lasting results.[1]

LOOK-ALIKE (0.1–0.2 mm) TELANGIECTASIA

Vascular Remodeling/Microtelangiectasia

There are 2 distinct types of very tiny (0.1–0.2 mm in diameter) telangiectasia, which although they look alike respond quite differently to treatment. The difference between the two is the occurrence of previous treatment. Telangiectasia 0.1 to 0.2 mm in diameter occurring in previously *untreated* patients usually responds gradually. Resistance is quite rare. Telangiectasia of the same size which remains or occurs after previous treatments can be extremely resistant to any type of treatment within a certain time frame. The term *matting* was coined to describe this type of telangiectasia.[2] The emergence

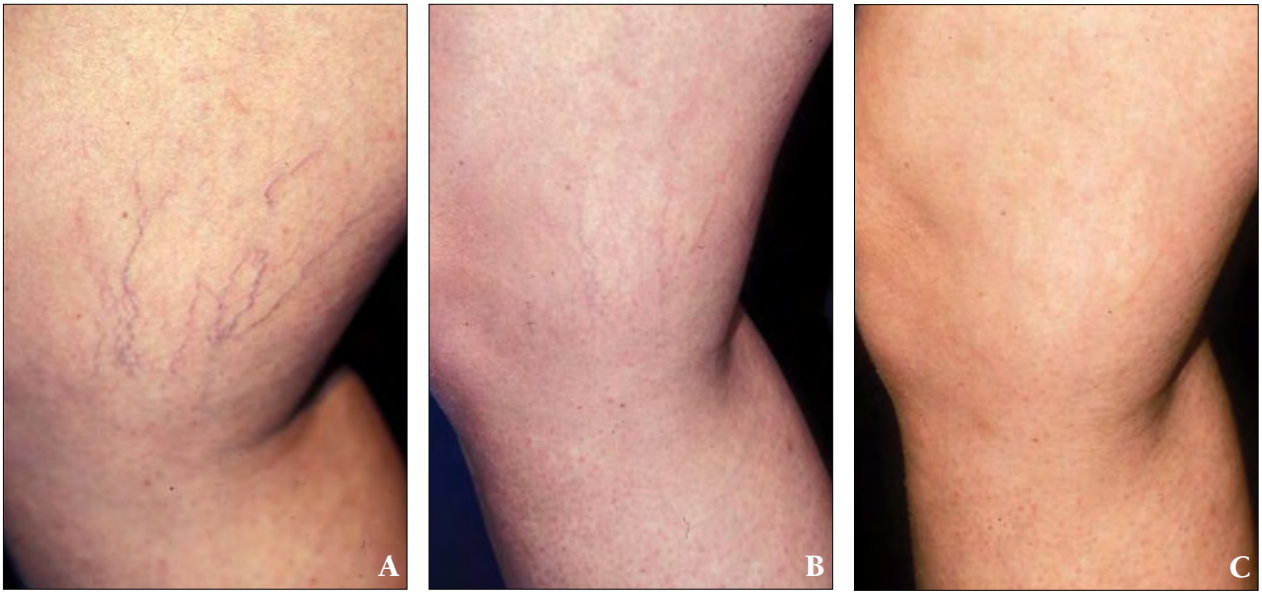

Figure 1. Pretreatment appearance of previously untreated telangiectasia 0.1 to 0.5 mm in diameter (A). Three months after 2 treatments there was complete disappearance of the larger vessels with substantial fading than the smaller ones (B). Ten months after the second treatment there was almost complete resolution (C). The inner knees are an area subject to matting and should be treated cautiously with low concentrations of sclerosants. In this case, 0.75% polidocanol was employed.

of small numbers of very small telangiectasia following sclerotherapy or lasers is common (Figure 2). The emergence of neovascular vessels probably reflects the underlying inability of certain patients to control vessel growth after trauma. They are also an excellent example of clinically visible angiogenesis in response to a known stimulus. This process may have implications of growth and metastasis of angiogenesis-dependent soft tissue tumors. The occurrence of this type of vasculature is the number one cause of dissatisfaction following small vessel sclerotherapy.[1] Neovascularization commonly involves the inner and outer thighs within 25 cm of the knees and is associated with obesity, use of female hormones, telangiectasia that has been present for many years, and a profusion of telangiectasia during pregnancy.[3] With passage of time, this type of vessel may resolve spontaneously or become responsive to sclerotherapy or other modalities. The use of more concentrated sclerosants is contraindicated. A number of publications suggest that patients with venous hypertension or those who have not received compression are more liable to have this problem.

RETICULAR VEINS AND VENOUS HYPERTENSION: RELATIONSHIP TO RESISTANT TELANGIECTASIA

Failure to treat reticular veins is often cited as an important contributory cause of resistance when treating telangiectasia (Figure 3). A recent article[4] discussed the complex processes associated with new vessel growth. In this paper, it was noted that vascular endothelial growth factor and other secreted factors promote the remodeling of collateral blood vessels involving a number of extremely complex molecular interactions. These include a hypoxia-inducible factor 1 (HIF-1), ubiquitin, and the "activation of hundreds of targeted genes." It is hard to imagine that the simple process of treating reticular veins, use of compression, or elimination of venous hypertension will have long-term impacts upon results following the treatment of telangiectasia. Resistant telangiectasia may occur despite any type of treatment manipulation and in the absence of venous disease. Seventy-five percent of the patients treated in this office between 1978 and 2010 who developed these vessels did not have any underlying venous disease as established by duplex studies (unpublished data). It has been speculated that the disabling of apoptotic processes by following previous treatments may be the root cause of this type of resistance.[2] Patients who develop resistant telangiectasia should be told to treat slowly (2–3 times yearly) with low concentrations of sclerosants. A history of multiple previous treatments, venous hypertension or venous reflux, or the existence of known risk factors mandates careful patient counseling regarding the possibility of resistance.

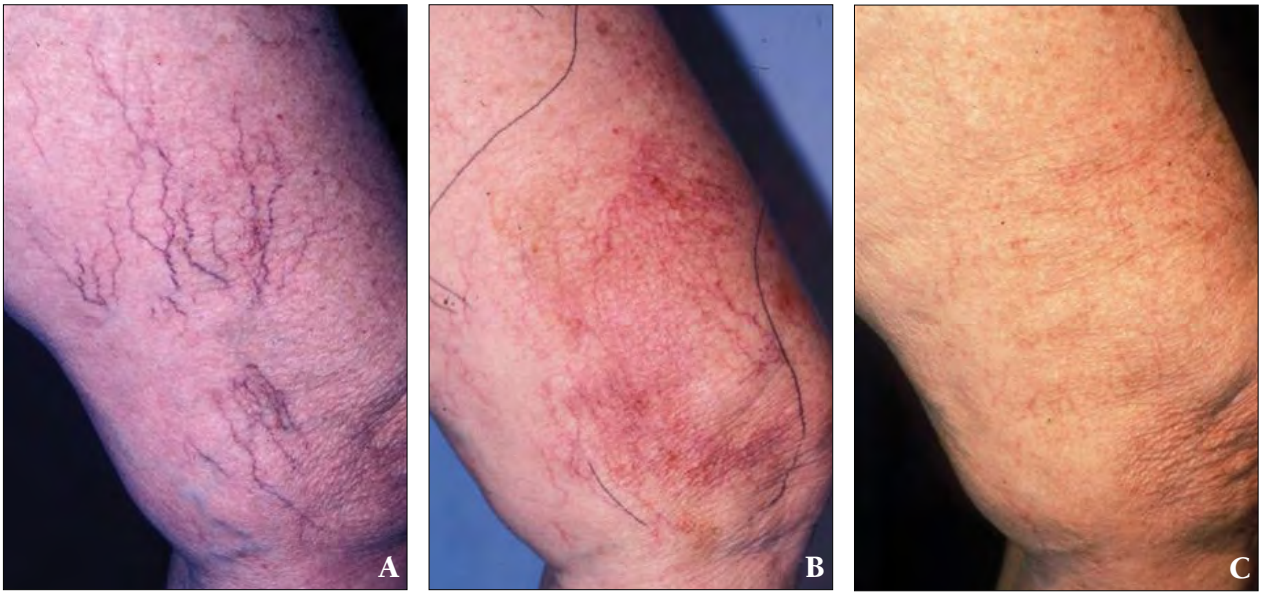

Figure 2. This pretreatment photograph reveals telangiectasia between 0.4 and 0.9 mm on an obese 68-year-old woman who was being treated with estrogen (A). There was no evidence of reflux on duplex exam. Although expected pigmentation did not occur following treatment with 0.5% polidocanol, matting was noted within 2 weeks of treatment. This photograph taken 1 year after treatment revealed extensive matting which did not respond to treatments carried out using different concentrations of polidocanol (B). Two years after treatment spontaneous resolution was observed (C). No treatments were carried out during that period.

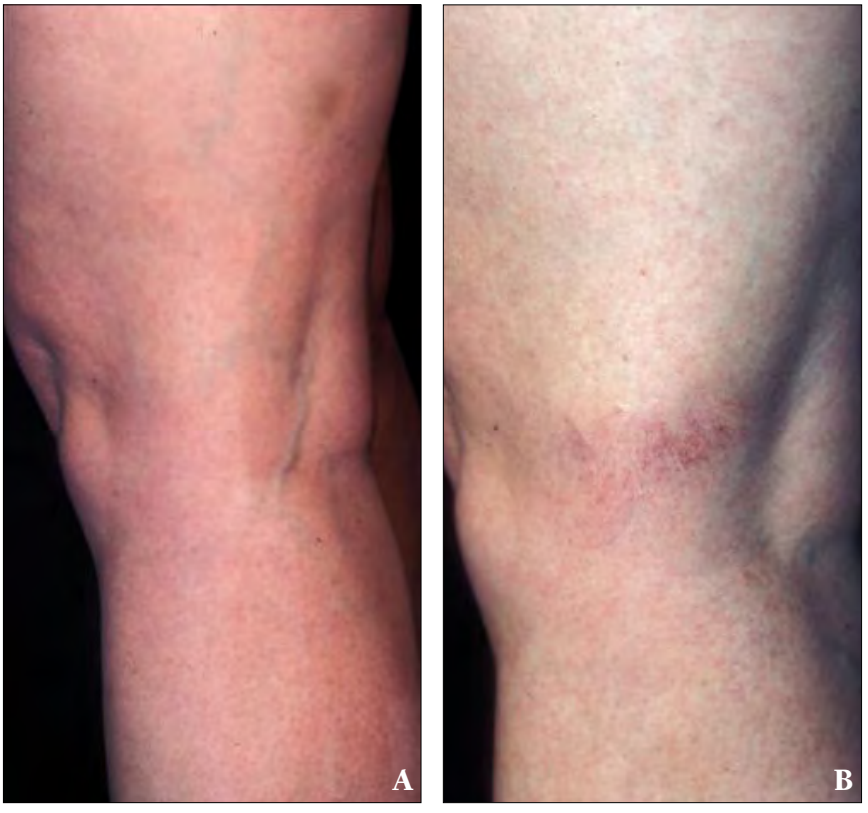

Figure 3. This pretreatment photograph reveals a 2.5-mm reticular vein occurring in a 35-year-old woman without evidence of venous hypertension/reflux (A). Class I compression hosiery was employed for 2 weeks. No thromboses occurred. Note the absence of any telangiectasia. One month following treatment with 1% polidocanol she developed matting which was unresponsive to further treatments (B), finally resolving 1 year from the time of her first treatment. It is unusual to read about this complication particularly in view of the fact that injection of reticular veins is supposed to minimize this complication.

A QUICK TEST FOR POSSIBLE RESISTANCE

Rapid refill of very tiny vessels following removal of digital pressure may be an indication of potential resistance.

Telangiectasia 0.3 mm in Diameter (Predictable Improvement)

Good news begins at 0.3 mm (the diameter of a #30 needle), particularly when treating patients who have small numbers of telangiectasia, have never been treated, and have no evidence of venous hypertension or reflux. Vessels of this size usually require several treatments and fade slowly but predictably over several months without pigmentation, thrombi, or progressive resistance. The reason that vessels of this size are singled out has to do with the fact that unlike smaller telangiectasia, vessels 0.3 mm in diameter are usually not rendered more resistant by previous treatments. As with 0.1- and 0.2-mm telangiectasia, pigment and palpable thrombi rarely occur.

Telangiectasia 0.4–0.5 mm in Diameter (Transitional Telangiectasia)

When treating telangiectasia 0.4 to 0.5 mm in diameter, slow and fast pattern overlap can occur. Vessels in this size range can respond abruptly or slowly to treatment. They appear to be more sensitive than smaller telangiectasia to changes in sclerosant concentrations and the effects of compression. Approximately 25% of telangiectasia measuring 0.4 mm in diameter and about 50% of those measuring 0.5 mm in diameter will respond rapidly. The effects of employing low or high concentrations in vessels this size are not always predictable. Both high and low concentrations can also result in extremely abrupt destruction associated with palpable thrombi and pigmentation or slow results. As with varicose veins, complete vessel destruction and the presence of thrombi can be detected within hours of injections. In addition, injection of reticular veins which dilutes sclerosant concentration and vigorous compression may not reduce the incidence of these complications. As an aside, thin walled reticular veins 1 to 2 mm in diameter will sometimes be destroyed suddenly using very dilute sclerosant concentrations which are inadequate to affect targeted telangiectasia.

Telangiectasia 0.6–0.9 mm in Diameter

Vessels in this size range are often observed in patients older than 60 years and have been present for many years. They are often purple or blue-green in color and can be extremely fragile and thin-walled. Protuberance of this type of vessel suggests extreme fragility. Clusters of protuberant and tortuous vessels in this size range as

noted earlier which involve the ankles (corona phlebactatica, phlebectasia) suggest long-term venous hypertension and reflux. Their presence should prompt doppler and/or duplex studies. Although treatment of telangiectasia in the presence of severe venous reflux often produces only transient results, occasionally despite the presence of reflux, improvements are long lasting. Typically vessels in this size range only require 1 treatment using very low concentrations of sclerosants. Their treatment is almost always associated with subsequent thrombi and pigmentation. The need for only 1 treatment is sometimes attributed to technique sophistication but is in fact related to the size of the vessel treated and its fragility.[5] Occasionally (particularly when using very low sclerosant concentrations) a gradual fading pattern occurs which may reflect apoptotic processes. Compression and the injection of reticular veins can sometimes reduce pigmentation and palpable thrombi when treating vessels whose walls are robust enough not to disintegrate following treatment. Bruising, thrombi, and pigmentation often make treated vessels of this size look worse before they look better. Patients should be reassured that the presence of thromboses is not dangerous; it is actually a good sign that the vein will not need repeated treatments. They also need to know that pigmentation is temporary. For patients with this type of vein, follow-up is usually carried out 1 week after treatment. At that time incision and drainage of thrombi may be useful to minimize hyperpigmentation. Photographs of posttreatment results seen in patients with similar veins are particularly reassuring for patients.

SCLEROSANTS

There is no perfect sclerosant. All can be associated with both minor and sometimes major complications. As a general rule, milder sclerosants at lower volumes are associated with more treatment failures and fewer serious complications. Certain patients are more susceptible to serious complications. These include people with thrombotic disorders; protein S and protein C deficiencies; and those who are obese, inactive, and have a history of migraine headaches with aura, suggestive of patent foramen ovale. Certain anatomical sites are associated with a higher incidence of complications. An understanding of the differences in response on the basis of the locations treated is particularly important when treating the retromalleolar areas which are prone to tissue necrosis, even when low concentrations of sclerosants are employed.

COMMONLY EMPLOYED SCLEROSANTS

Several sclerosants are either US Food and Drug Administration (FDA) approved or legal to use off-label for the treatment of telangiectasia.[6] All can cause unwanted thromboses, destruction of nontargeted vessels, minor complications such as hyperpigmentation and neovascularization, as well as more serious problems including superficial and deep thrombophlebitis and with extreme rarity pulmonary emboli and tissue necrosis. Some are associated with serious allergic reactions. Detergent sclerosants polidocanol (POL) and sodium sotradecol sulfate have the advantage of FDA approval specifically for treating lower extremity veins, but both are capable of serious to sometimes fatal allergic reactions, although this event is extremely rare.[6] Both agents are extremely versatile and comfortable to use providing effective sclerosis of vessels of all sizes by adjusting their concentration. It is worth noting that POL is only approved in 2 concentrations, 0.5% and 1%. The use of other concentrations created extemporaneously for specific purposes is not illegal but is considered to be off-label. Sodium sotradecol sulfate is 2 to 3 times more potent than POL and has been observed to produce concentration dependent extravasation tissue necrosis at a 1% concentration. 0.5% Sodium sotradecol sulfate did not produce tissue necrosis upon direct injection.[1] Polidocanol when injected into the author's forearm at a 3% concentration did not produce this complication.[1] The injection of a much milder concentration of POL (0.25%) into telangiectasia located around the medial malleolus (an anatomical site prone to this complication) has produced tissue necrosis.[1] It is worth remembering that all sclerosants, no matter how dilute, may produce tissue necrosis through multiple mechanisms. Manufacturers recommend that detergent sclerosants should not be employed in people with polyallergic diatheses. Polidocanol, which began life as a local anesthetic is the more comfortable of the 2 and the only sclerosant, which has ever undergone rigorous testing before FDA approval.[6] For the treatment of telangiectasia, POL 0.25% to 0.75% is commonly employed. Sodium sotradecol sulfate 0.1% to 0.3% is also effective. 72% Glycerin, with or without epinephrine, although viscous and slightly uncomfortable to use, produces effects similar to 0.5% POL. Glycerin is inexpensive, rarely causes serious complications when small volumes are employed, and will not produce tissue necrosis when injected directly into the skin.[1] Some experienced phlebologists regard it as the treatment of choice for telangiectasia.[7] It is also legal to use off-label. Hypertonic saline (HS), FDA approved as an abortifacient, is also inexpensive and legal to use off-label. The use of this painful and relatively weak sclerosant is associated with an unacceptably high incidence of technique-related extravasation tissue necrosis. Tissue damage following its use may be the leading cause of malpractice actions following sclerotherapy in the United States.[7] The widespread use of this

agent was encouraged by an inordinate fear of allergies following the use of detergent sclerosants and its low cost. In good hands, it works well. Hypertonic saline has only one virtue, its absolute lack of allergies when it is not combined with other agents. It is employed at either 11.7% or 23.4% depending on the type of telangiectasia treated. Occasionally larger veins will respond to it as well on an individual basis. Syringes containing HS must be carefully marked. Colored gummed stickers are applied to the plunger of the syringe to identify it. Syringes containing HS must be kept separate from other sclerosants in a closed marked container. Bottles of unused HS must also kept separately.

FOAMED SCLEROSANTS

The ability of detergent sclerosants to be foamed following agitation makes them particularly effective for the treatment of large refluxing veins. Foamed sclerosants are approximately 2 to 3 times more potent than their liquid equivalents probably on the basis of mechanical displacement of venous blood resulting in prolonged unobstructed contact with endothelium. Foamed sclerosants typically are associated with an increase in the type of complications associated with increased potency. These include pigmentation, neovascularization, migrainoid attacks, and the destruction of nontargeted veins at an unexpected distance from the injections. They have not proved more efficacious than lower concentrations of liquid sclerosants when treating telangiectasia. Some experienced phlebologists[7] employ dilute foams to treat reticular veins as part of the process of treating associated telangiectasia. In general their use is contraindicated for the treatment of telangiectasia.

Treatment Tips and Protocols

Limiting the number of treatments in any given timeframe while employing the lowest concentration and volume of sclerosant that is effective minimizes all types of complications (Table). Milder sclerosants such as glycerin and POL are most useful for fragile telangiectasia. The use of support hose and the treatment of all types of vasculature is often beneficial. It is particularly useful for symptomatic patients, those with any degree of reflux, or those who must be sitting or standing many hours daily. Travelers with extensive telangiectasia and varicose veins are advised to wear the hosiery while flying, ambulate while in the plane, minimize caffeine and alcohol, and drink copious amounts of water to maintain hydration. Ideally patients should not fly for at least 2 weeks after treatment if the flight is over 2 hours. In the event that a long flight is essential, patients will be asked to take aspirin as tolerated. Thrombotic complications following sclerotherapy can occur even when small volumes and concentrations of sclerosants are used and they can occur up to 3 or 4 weeks after sclerotherapy. They are worsened by dehydration and inactivity. Patients who can tolerate hosiery often experience less discomfort (heaviness in the legs, aching, and swelling). Many women report these benefits when they wear hosiery during their menstrual cycles.

The Downside to Compression Hosiery—Compression hosiery which is graduated exerts the bulk of its pressure on the feet and ankles. This may compromise the arterial circulation in patients who have lower extremity arterial insufficiency, and is particularly risky in patients with diabetes and associated neuropathy. Patients with anatomical foot disorders such as bunions may suffer unacceptable pressure on bony prominences. Discomfort wearing compression hosiery has led some of my patients to cut the toes out of their hosiery to make them more comfortable and less constrictive. Washing the hosiery before use for

Treatment Protocols

Previously untreated telangiectasia 0.1–0.3 mm in diameter	Treat every 4–6 weeks. Limit this to 3–4 treatments followed by a 3-month break.
Telangiectasia over 0.3–0.5 mm in diameter	Treat every 6 weeks. Limit this to 3–4 treatments followed by a 3-month break.
Telangiectasia over 0.6 mm in diameter	Treat every 1–2 weeks. Use of compression hosiery and injection of reticular veins may be beneficial to reduce pigmentation and thrombi. Incision and drainage of thrombi may reduce pigmentation. The use of hosiery may be beneficial.
Resistant telangiectasia 0.1–0.2 mm in diameter (matting, neovascularization)	Treat every 3–6 months. Test for the presence of venous hypertension/reflux. Warn patients in advance. The presence of very rapid refill noted after applying digital pressure to telangiectasia suggests both venous reflux and the possibility of resistance. I use ½ of a petri dish pressed upon the vessels while the patient watches to see how quickly the vessels refill.

the first time along with the use of rubber gloves while donning them helps insure patient compliance (they are easier to put on).

CONCLUSION

Subtyping telangiectasia into specific groups on the basis of very small changes in diameter, the occurrence of previous treatments, and the presence or absence of venous hypertension or reflux is a very useful way of predicting treatment outcomes and the occurrence of specific types of complications. These are only 2 of the variables which affect treatment outcomes. A large number of interactive variables affect treatment outcomes. A partial list includes the number of treated vessels, their location, the thickness of their walls, the presence or absence of venous hypertension, estrogen therapy, obesity, number of pregnancies, etc. A description of these variables and their impact on treatment outcomes will appear in forthcoming articles. An understanding of the impact of the variables discussed here should be viewed as a first step in gaining a wider understanding of the processes which underlie successful and unsuccessful treatment outcomes. As more knowledge accrues, precise protocols, realistic expectations, personalized treatments, and the ability to predict and sometimes avoid certain types of complications will unquestionably emerge.

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