Sclerotherapy – How to Achieve a Spectacular Outcome

Sklerotherapie – Wie man spektakuläre Ergebnisse erzielt

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ZUSAMMENFASSUNG

Bei korrekter Durchführung erzeugt die Sklerotherapie einen bindegewebigen Verschluss, der nicht rekanalisiert werden kann. Entscheidend für den Erfolg ist eine Kontaktzeit zwischen Sklerosierungsmittel und Endothelzellen, die ausreichend ist, um die Vene mit geringstmöglichen Nebenwirkungen hinreichend zu schädigen. Für optimale Ergebnisse der Sklerotherapie ist es erforderlich, den richtigen Patienten und die richtige Vene auszuwählen und dann geeignete Sklerosierungsmittel und Injektionstechniken anzuwenden. Dieser Artikel gibt einen Überblick über die aktuelle Literatur zur Sklerotherapie und umfasst Tipps und Tricks für eine Optimierung der Ergebnisse und Minimierung der Komplikationen.

ABSTRACT

When performed correctly, sclerotherapy results in fibrous occlusion that is not amenable to recanalization. Paramount to success is adequate contact time of a sclerosant to the endothelial cells to cause sufficient damage to the vein while minimizing side effects. To optimize sclerotherapy results, one needs to choose the right patient and the right vein and then use the proper sclerosing agent and injection techniques. This paper will review current literature on sclerotherapy, as well as tips and tricks to optimize outcome and minimize complications.

Introduction

Despite new development in cutaneous lasers for treatment of telangiectasias, sclerotherapy remains the treatment of choice for telangiectasias and reticular veins (Grade 1A recommendation per the European guidelines for sclerotherapy) [1]. In the treatment of incompetent truncal and nontruncal varicosities, foam sclerotherapy offers a safe and cost effective alternative in selected patients to thermal and nonthermal ablation (Grade 1A) [1]. Foam sclerotherapy has a slightly higher rates of recanalization but similar quality of life improvements when compared to surgery and thermal ablation [2].

Sclerotherapy is the injection of liquid or foam substance that injures the endothelial cells lining veins, creating a controlled chemical induced fibrosis. When performed correctly, sclerotherapy produces maximum endothelial damage with minimal thrombus formation, resulting in a fibrous cord that is not amenable to recanalization. If there is insufficient damage, a thrombotic reaction is created that recanalizes over time. Paramount to success is adequate contact time of a sclerosant to the endothelial cells to cause sufficient damage to the vein while minimizing side effects. To optimize sclerotherapy results, one needs to choose the right patient and the right vein and then use the proper sclerosing agent and injection techniques. This paper will review current literature on sclerotherapy, as well as tips and tricks to optimize outcome and minimize complications.

Choosing the Right Patient and the Right Veins

A comprehensive clinical evaluation and preoperative duplex ultrasound examination of the lower extremity is mandatory to exclude underlying venous insufficiency. The most proximal point of reflux should be treated first prior to sclerotherapy of spider and reticular veins in the area affected. Failure to address underlying venous incompetency (saphenofemoral reflux, perforator disease) will result in higher rates of recurrence and hyperpigmentation.

Contraindications for sclerotherapy per the European guidelines for sclerotherapy include known allergies to sclerosants, acute deep vein thrombosis and/or pulmonary embolism, local infection in the area of treatment, severe arterial occlusive disease, and immobility (Grade 1C) [1, 3]. In addition, a patient with a known symptomatic patent foramen ovale should not be treated with foam sclerotherapy. However, it is not necessary to perform a routine preoperative evaluation for patent formane ovale [1].

Relative contraindications include pregnancy, breastfeeding (can consider interrupting breastfeeding for 2–3 days), mild arterial occlusive disease, strong history of multiple allergies, high thromboembolic risk, acute superficial venous thrombosis [1, 3]. Patients with a history of migraines especially following previous foam sclerotherapy should be treated with caution.

It is important to properly consent and set realistic expectations from the onset during the consultation visit. Patient should understand that multiple sessions may be required and should be spaced at least 6 weeks apart to adequately judge the level of improvement from the treatment. The aim is for improvement rather than perfection. If applicable, patients should be informed that foam sclerotherapy is off-labeled but highly effective treatment with higher risk of certain side effects (see complications). Hyperpigmentation is expected post sclerotherapy; most cases resolve within months with the majority disappearing within a year. Finally, photographic documentation pre- and post- procedures are essential to ensure patient satisfaction as patients' recall of preoperative appearance is often poor [4].

Choosing the Right Sclerosant

Appropriate choice of sclerosant is paramount to maximizing effectiveness while minimizing side effects. Several variables exist with any sclerosant choice, including the type of agent (osmotic, irritant, and detergent based), concentration, volume, and method of preparation (liquid or gas). To minimize side effect, the principle of the least should be used – using the lowest concentration possible with minimal amount of volume and the lowest injection pressure.

Types of Sclerosant

Sclerosants can be classified into three main categories: hyperosmotic agents, chemical irritants, and detergents.

Hyperosmotic agents such as hypertonic saline and dextrose cause cellular dehydration of endothelial cells and red blood cells. The advantage of hypertonic saline is its lack of allergenicity and low cost. While it was widely used prior to the availability of detergent based solution, its use is less favored due to the increased risk of ulcerations from extravasation and hyperpigmentation and the intense burning sensation upon injection from its irritant effect on nerve endings [3].

► Table 1 Recommended Sclerosant Concentration based on Vein Diameter

vein diameter	recommended STS concentration	recommended POL concentration
< 0.5 mm	0.1%	0.25%
0.5–2 mm	0.2 %	0.5%
2–4 mm	0.5 %	1 %
4–6 mm	0.5 % Foam	1 % Foam

Chemical irritant such as chromated glycerin and polyiodinated iodine are caustic agents on the vein walls. Glycerin is commonly used for fine telangiectasia due to its low risk of skin necrosis, pigmentation, and risk for matting. It is typically used as a 72 % solution compounded 2:1 with 1 % lidocaine with epinephrine to reduce stinging and to cause local vasoconstriction to increase its effects[3].

Detergent based sclerosants, the most widely used are polidocanol (POL) and sodium tetradecyl sulphate (STS) due to their long-term safety and efficacy profiles. Detergent sclerosants cause endothelial damage by protein theft denaturing of the cell surface lipids. They are the most commonly used agents for sclerotherapy worldwide due to their low side effect profile. The ability to create a foam agent due to the natural property of detergent also makes it more versatile as a sclerosant agent for a wider range of vessel size. Cochrane review concluded no significant differences in outcome or complication rates between STS and POL, in part due to limited available high guality studies comparing sclerosants [5, 6]. POL has an added benefit that it is less painful than other sclerosants with its local anesthetic effects [5]. As these sclerosants are commercially available worldwide, compounded sclerosants are not recommended. Several studies have reported inconsistencies in concentrations and presence of contaminants in compounded sclerosants [3, 7].

Concentration

One should match the sclerosant concentration to the caliber of the vessel, with increasing concentration used for larger veins. The key to sclerotherapy is to use the minimum effective concentration that would cause optimal fibrosis without complication. Too low a concentration would result in inadequate endothelial damage and subsequent thrombus formation rather than fibrosis; too high a concentration would cause iatrogenic reaction such as hyperpigmentation and potential ulceration.

Kobayashi et al, showed that 0.1 % STS and 0.3 % POL produced equivalent effect on cell death within 15 seconds [8]. In clinical practice, STS is approximately 2–3 × stronger than POL for the same concentration [9, 10]. For telangiectasia measuring <0.5 mm, a low concentration such as STS 0.1% or POL 0.25% is usually sufficient. For telangiectasia 0.5–2 mm in size, STS 0.2% or POL 0.5% might be required. Reticular veins measuring 2–4 mm in size should be treated with POL 0.1% liquid or 0.5% foam (**► Table 1**).

Volume

The volume of solution required to produce sclerosis is directly related to the vessel size. Larger caliber veins need more volume than smaller caliber veins. Approximately 0.2-0.5 cc for spider veins and 0.5-1 cc for reticular veins is usually sufficient; the idea is to inject until you see a 1-2 cm area of blanching in the site of injection or sooner if you stop seeing the solution flow. Avoid getting carried away by injecting more volume than necessary as an attempt to treat a greater area with a single injection. Both STS and POL are inactivated by plasma proteins in blood [11], thus limiting the distal effect of the sclerosant from the point of injection. Thus, one need to inject at multiple site along a vein cluster to introduce fresh sclerosant to the endothelial cells. To maximize detergent sclerosant effects, one can use an empty vein technique by leg elevation, create a foam sclerosant to displace the blood, or inject saline first to dilute then blood followed by the sclerosant.

Total dose of POL should not exceed 2 mg/kg/d (i. e. POL 1% = 14 mL) and STS should be limited to no more than 4 mL of 3% solution per session [1].

Pressure

The key is to use minimum injection pressure and slow pace. Too much pressure may lead to vessel rupture and extravasation resulting in higher rates of hyperpigmentation. Worse, high pressure can result in sclerosant traveling into arterioles which can result in ulcerations. Too little pressure will result in an inadequate amount of solution to travel the length of the affected vessel leading to additional insertion sites. Ideally a 3 or 5-cc syringe is best for treating telangiectasia. The larger piston in a 3- or 5 cc syringe results in lower pressure when depressing the plunger versus a 1-cc syringe which delivers high pressure [12].

Foam

While liquid sclerosant is preferred for treating telangiectasis; foam sclerosant is superior in treating larger varicose veins. Foam sclerotherapy is nearly 3–4 times as effective as its liquid counterpart. The ESAF study, which was a randomized, prospective controlled multicenter trial, showed greater efficacy with POL foam in eliminating reflux in the GSV (69%) vs liquid POL (27%) with greater patient satisfaction (82% with foam vs 58% with liquid).

Efficacy and safety of foam is well established. Meta-analysis of 73 studies with over 11,000 patients demonstrated a medial rate of venous occlusion of 85 %, using both STS and POL based sclerosants with more than 90 % of subjects reporting symptomatic improvements [13]. A review of four randomized controlled trials in six publication comparing US guided foam sclerotherapy with endothermal ablation by Davies et al, showed higher closure rate with endothermal techniques (83–95 %) compared to US guided foam sclerotherapy (54–83 %). However, patient reported outcomes such as Venous Clinical Severity Score, Chronic Venous Insufficiency Quality of Life, etc) showed significant improvements regardless of the modality [14].

Several factors contribute to foam's superior results compare to its liquid counterpart. Foam is a highly viscous solution that completely displaces blood within the vein lumen whereas liquid sclerosant mixes with blood and is inactivated. The generation of miniscule air bubbles also increases surface area for contact with the endothelial cells. Foam sclerosant also produces more vasospasm than liquid sclerosants. All of these effects result in longer contact time for foam, maximizing endothelial cell injury and allowing for lower total volume and concentration to be used compare to liquid sclerosant.

Attempts with foam sclerotherapy have been reported as early as 1940 s, but early techniques were unable to produce a uniformly effective foam. Mainstream acceptance of foam began with Cabrera in 1990 s, and further refined by Tessari and later Frullini with the double syringe system (DSS), making foam production simple, inexpensive and most importantly easily reproducible. The Tessari method uses a three-way stopcock while Frullini uses a two-way connector; in both methods, an adaptor between two syringes is used to create a turbulent flow to generate the foam.

The quality, stability and efficacy of foam is influenced by several variables including the specific sclerosant and concentration used to create the foam, method of preparation, the gas-to-sclerosant ratio, and the type of gas mixture used. The ideal foam should be small microbubbles with dense consistency; this allows for localized efficacy while limiting the duration of the bubbles and potential for distal effect [15, 16]. Both STS and POL are widely use as the sclerosant of choice for creating foam. Syringes with low silicone content should be used as the silicone lubricant reduces foam lamellae and its stability [16, 17]. No difference in foam stability was found when comparing various two vs threeway connectors [18]. However, three-way connector does allow for partial closure which narrows the caliber between the two syringe to create a longer-lasting foam [16]. The use of a 5 µm in-line filter for creating foam, while not necessary to prevent bacterial contamination [19], improves foam stability [20]. Tessari et al noted that smaller needles (27 and 30 gauge) may produce a less stable foam and advocates the use of 25 gauge for injection [16]. Subsequent study noted variability in foam stability between needle size (83 sec for 25 gauge; 70 sec for 27 gauge, 67 sec for 30 gauge), though this is unlikely to affect its clinical effectiveness [20]. Standard liquid- to-gas ratio is 1:4, as this produces a dense and stable foam [16].

The stability (half-life) of foam varies depending on the type of gas used (room air, sterile air, CO2 vs other mixture of gas). Room air which contains 72% nitrogen, is readily available, creates a stable foam (1–2 min) [20], but does not dissolve quickly in blood due to its nitrogen content. CO2 is more soluble in blood but creates a less stable foam (25 sec) [20], that must be injected immediately [16]. The use of a physiologic gas with the addition of O2 creates a foam that has a slightly longer half-life and readily diffusible in blood. Beckitt et al, in their prospectively maintained database, noted statistically significant lower incidence of hyperpigmentation (7.2% vs 3.3%) and occlusion rate (91% vs 83%) with the use of physiological gas of 70 % CO2 and 30 % O2 vs room air. The authors propose that the higher solubility of the CO2 may allow for greater endothelial damage. They found no differences in neurologic events between the two gases, but this may be partially due to the low overall incidence (0.7 %) [21]. European consensus guideline recommends the use of air for generating foam (Grade 1A) or mixture of CO2 and O2 (Grade 2B) [1].

While there is no evidence based limit on maximum volume of scleroant foam per session, the European Consensus meeting recommended a maximum of 10 mL (Grade 2B), in part out of concern for rare neurologic and pulmonary events, especially in patients with known patent foramen ovale [1].

My Sclerotherapy Technique

Patient Positioning

I prefer to have patients lying sideways, with one leg draped over the other (**> Fig. 1**). This is a comfortable and stable supine position for most patents and allow for visualization of the lateral and posterior aspect of one leg and medial and anterior aspect of the other. Once treatment is done on this side, the patient simply turns to the other side allowing for full treatment of the legs without having the patient in an uncomfortable prone position.

Sclerotherapy tray

My sclerotherapy tray consists of pre-drawn syringes of liquid sclerosant clearly labeled and separated into their own basin (POL 0.25% and 0.5% or STS 0.1% and 0.2%). When the plunger is completely pulled back and the syringe is filled to 3 mL, one has minimal dexterity and control. Thus, I prefer to draw only 2 mL of sclerosant agent in each 3-mL syringe. Cotton balls and a basin with alcohol are used for cleanse the skin. Extra 3-mL syringes, 30G needles, 25G butterfly needle, and three-way stopcock are set aside for foam sclerotherapy (**> Fig. 2**).

Approach to Telangiectasia and Reticular veins

Transillumination devices and duplex ultrasound should be used to visualize reticular and perforator veins; these often accompany telangiectasia in a significant percentage of cases [22]. Comprehensive treatment should be directed at both the telangiectasia and the underlying varices. I treat the reticular veins first, followed by the venulectasias and then final the associated telangiectatic web [23].

The skin is first cleansed with isopropyl alcohol on cotton balls. Placing a small drop of sclerosant on the skin before injection can improve visibility by reducing light scatter. Alternatively, one can use special devices with polarizing light to help visualize the veins. Using a 30–32G needles on a 3-cc syringe, the needle should be bent approximately 30–45° angle to facilitate a near parallel approach of the vein. The nondominant hand should be used to stretch the skin taut. Slow injection with low volume is key to reduce the risk of extravasation, skin necrosis, matting and hyperpigmentation. One should see the flow of sclerosant temporarily displacing the blood in the telangiectasia. I usually inject between 0.2–0.5cc to create a 1–2 cm diameter of clearing.

When treating reticular veins, I will occasionally aspirate and look for slight venous return at the needle hub to ensure proper placement before injecting. Note that one should not allow too much blood to mix with the sclerosant in the syringe as this will



Fig. 1 My preference for patient positioning – this allows for visualization of the lateral and posterior aspect of one leg and medial and anterior aspect of the other.



▶ Fig. 2 My sclerotherapy tray setup. Pre-drawn 2 mL sclerosant in each 3-mL syringes, clearly labeled with concentration and separated in their own trays. Silver basin with isopropyl alcohol and cotton balls for cleansing the skin. An empty basin for discarded sharps. Extra syringes, three way stopcock and needles for foam sclerotherapy.

inactivate the sclerosant [11]. If there is resistance to flow or if a bleb forms at the site of injection, stop immediately and find a nearby branching vessel to inject.

Foam Sclerotherapy

I create foam using two 3-cc syringes, one syringe is filled with 0.5 ml of liquid sclerosant and the second with 2 cc of room air to create an optimal ratio of 1:4 of liquid to gas. While there are several choices for gas, I primarily use room air because it is readily available and unlikely to cause any major side effects with low volume (less than 10 mL). Luer Lock connection is preferred to minimize accidental syringe pop-off due to the pressure when created foam sclerosant. I prefer to use a three-way stopcock, rotated 45° from the fully opened position to create a narrower caliber opening, which creates a denser longer lasting foam. The sclerosant and gas is agitated back and forth between the two syringes approximately 10-20 times to produce a smooth consistent microfoam. As the foam tends to degrade within 1–2 minutes, it should be mixed immediately before injecting using either a butterfly needle or direct puncture in 0.5-2 ml aliquots. When treating reticular and small varicose veins, I prefer to do direct puncture with a 30G needle, either with direct visualization or ultrasound guidance when the vessel is not visible. While literature has suggested that small needles may disrupt fine microbubbles, this is unlikely to be of consequence when treating small caliber vessels [20].

Prior to ultrasound guided injection of a perforator vein or a vein that is not clinically visible, I always check for the presence of arteries near the injection site. The needle should be clearly visible on the ultrasound during the initial puncture to confirm its location in the vein lumen. When treating larger veins, I prefer to elevate the leg slightly to reduce its size and use 25G butterfly needle for access. This allows me to gain access first and then affix the newly created foam syringe on the other end of the tubing without disturbing the placement of the needle. Intravenous position is confirmed by aspiration of blood at the hub of the needle. Foam only lasts approximately 60 sec, and altered foam with liquid and large bubbles should not be used.

When treating a perforator veins with foam sclerotherapy, it is safer to inject the varicose veins connected to the perforator vein rather than the perforator vein directly. The ultrasound should follow the progression of the foam during treatment and injection should stop when foam is visualized entering the perforator vein.

Postsclerotherapy Care

Compression regimen recommendation varies greatly among established guidelines and individual practitioners [24]. There are few studies on this subject, with one showing greatest efficacy and least hyperpigmentation with three weeks of stockings [25] while another showed no cosmetic improvement between 8 hours vs 6-weeks of compression [26]. As per the European guidelines, I prefer to place patients in Class 2 (23–32 mmHg) stockings for 1–3 weeks [1]. I advise my patients to walk after treatment but avoid heavy impact activities (such as aerobic exercise) for one week.

Minimizing Complications

Complications following sclerotherapy are generally predictable and manageable. Pigmentation and matting are the most common side effects and patients should be counseled to expect these changes after their treatment. Small skin ulcerations (less than 5 mm) are rare. Neurosensorial complications, chest tightness, and dry cough have been reported but their incidence is likely less than 0.01% [1]. Fortunately, there have only been isolated cases of severe adverse effects such as large area of skin necrosis, pulmonary embolism, and anaphylaxis [27]. When performed properly, sclerotherapy has a low incidence of complications.

Hyperpigmentation and Microthrombus

Pigmentation from sclerotherapy occurs in about 10–30% of patients and is primary due to hemosiderin staining from red blood cell extravasation or trapped blood (intravascular microthrombus) in a sclerosed vessel rather than post inflammatory hyperpigmentation from melanocytic alteration [28]. While we often attribute hyperpigmentation to overtreatment (such as injection too high volume or concentration), undertreating a vein can also result in inadequate endothelial damage and subsequent thrombus formation rather than fibrosis.

Proper apposition of the vein wall post treatment is essential to allow of fibrosis rather than thrombosis. This can be accomplished preoperatively by leg elevation to reduce the size and empty the vein. Post-procedural compression stocking is recommended to minimize side effects such as thrombophlebitis and pigmentation (Grade 2B) [29]. Even with meticulous technique, intravascular microthrombi can occur and drainage with a needle or small incision 2–4 weeks post treatment is recommended to reduce pigmentation [1, 3].

Matting

Telangiectatic matting, in which new fine capillary networks develop in the area of a sclerosed vein, can occur in 15–24% of cases [3]. This neo-angiogenesis is often due to inadequate treatment of an undiagnosed underlying venous incompetence. In some cases, matting is a result of excessive inflammation due to high concentration or volume used; hence one should always match the strength and volume of sclerosant to the vessel size. Transillumination to look for underlying reticular veins and an ultrasound to evaluate for perforator reflux should be performed in the area of the matting. Proximal reflux should be treated first with subseguent gentle sclerosant with lower concentration and volume. For patients who are prone to matting, one can consider the use of glycerin as a sclerosant or cutaneous laser devices for these fine vessels [3]. Finally, if matting persist despite treatment, it should be left alone. The majority of telangiectatic matting will resolve; a tincture of time is often all that is necessary.

Skin necrosis

Cutaneous necrosis following sclerotherapy have a reported incidence between 0.2%-1.2%. Extravasation of hypertonic saline is commonly associated with skin necrosis. However, extravasation alone is unlikely the cause of skin ulcerations with detergent based sclerosants, when using recommended sclerosant concentrations and low volumes [12]. Subcutaneous injections of liquid and foamed POL to mimic extravasation did not result in cutaneous necrosis with volume less than 0.5 mL [30]. Rather, inadvertent passage of sclerosant into small arterioles is likely the cause of most skin necrosis. This can occur when injection pressure exceeds capacity of the vein, causing venous-capillary reflux into the surrounding arterioles [31].

To minimize the risk, one should inject low volume under minimal pressure while visualizing the needle tip and flow of the solution into the vessel. Avoid injection directly over the bony prominence of the medial and lateral malleolus as these areas are prone to ulceration. Instead one can usually find an alternative access point in the adjacent skin. A skin bleb is consistent with extravascular injection and one should stop injecting immediately. The development of a porcelain white blanching and pain may indicate sclerosant entering the arterioles. Recommended treatments include massage, injection of normal saline or hyaluronidase, and topical nitroglycerine [3]. Fortunately, most cases of skin necrosis result in small 2–5 mm ulceration that often heal with minimal sequelae. Nevertheless, they can be rather painful and protracted; local wound care with vaseline/bandage or hydrocolloid dressing provides a moist wound environment for healing.

True intra-arterial injections can result in extensive tissue necrosis and is an emergency. Ultrasound guidance should be used to identify neighboring arteries when injecting a vein that is not clinically visible or palpable [1]. The presence of severe pain upon injection should prompt immediate cessation and evaluation of possible intra-artierial injection. Catheter-directed anticoagulation and thrombolysis, as well as systemic anticoagulation and steroids have been recommended as treatment [1].

Neurological Events

The prevalence of neurosensorial complications (visual disturbances such as blurred vision, double vision, or scotoma with or without associated paraesthesia and dysphasic speech) following sclerotherapy ranges from 0.09% to 2% in large case series of more than 500 patients and from 0% to 4.5% in prospective randomized controlled trials [32]. These symptoms can occur with any kind of sclerotherapy, though more commonly associated with foam than liquid sclerotherapy sessions [33]. It has been reported with the use of either POL or STS.

In the past, authors attributed these neurosensorial complication to paradoxical microbubble embolism through a patent foramen ovale or intrapulmonary shunts. Echocardiography and transcranial doppler studies during foam sclerotherapy showed that bubbles common reach the right heart and cerebral circulation; their presence did not correlate with visual disturbances. Recent evidence has shown that these transient neurological events are likely migraines with aura rather than transient ischemic cerebrovascular events. When endothelial cells are damaged during sclerotherapy, there is local release of multiple inflammatory mediated factors, including endothelin-1, which travels systemically to the brain resulting in vasospasm [34] or cortical spreading depression in susceptible patients [32, 35]. Patients with a history of migraine with aura or known patent foramen ovale may be at higher risk of visual disturbances and should be properly informed prior to treatment. Routine screening for patent formane ovale prior to foam sclerotherapy is not necessary [1].

Almost all reported cases of neurosensorial complications are during treatment of truncal vessels using large volumes of foam [36]. One should limit total foam volume to less than 10 mL per session as recommended by the European consensus guideline and consider multiple smaller volume injections rather than one bolus [1]. Some authors have advocated specific maneuvers such as manual compression of the saphenofemoral junction at the time of the injection [37], leg elevation before and after treatment, immobility post sclerotherapy, use of CO2-O2 gas and filters to generate microfoam; none of these maneuvers prevented bubble emboli to the heart [38]. Morrison noted lower incidence of visual disturbance with high volume injections (25–27 mL) when using CO2 foam rather than room air foam (8.2% vs 3.1%) [39]. Recent studies showed no difference in neurologic disturbances in low volume injections (less than 10 mL) between physiological gas (CO2-O2) vs air [21].

Summary

Selecting the ideal patient and veins along with proper counseling and preoperative planning are paramount to effective sclerotherapy. One should match the concentration and type of sclerosant to the caliber of the vessel. The lowest concentration, pressure and volume should be used to maximize outcome and minimize side effects. Successful sclerotherapy requires meticulous technique. When performed properly, sclerotherapy produces excellent results that is visually satisfying for both the practitioner and the patient.

Conflict of Interest

The author has received lecture fees from Kreussler. Consultant for Merz Aesthetics.

References

- Rabe E, Breu FX, Cavezzi A et al. European guidelines for sclerotherapy in chronic venous disorders. Phlebology 2014; 29: 338–354. doi:10.1177/ 0268355513483280
- [2] Rasmussen LH, Lawaetz M, Bjoern L et al. Randomized clinical trial comparing endovenous laser ablation, radiofrequency ablation, foam sclerotherapy and surgical stripping for great saphenous varicose veins. Br J Surg 2011; 98: 1079–1087. doi:10.1002/bjs.7555
- [3] Weiss MA, Hsu JT, Neuhaus I et al. Consensus for sclerotherapy. Dermatol Surg 2014; 40: 1309–1318. doi:10.1097/DSS.00000000000225
- [4] Santiago FR, Piscoya M, Chi YW. Change in perception of sclerotherapy results after exposure to pre-post intervention photographs. Phlebology 2018; 33: 282–287. doi:10.1177/0268355517736178
- [5] Schwartz L, Maxwell H. Sclerotherapy for lower limb telangiectasias. Cochrane Database Syst Rev 2011. doi:10.1002/14651858.CD008826.pub2
- [6] Tisi PV, Beverley C, Rees A. Injection sclerotherapy for varicose veins. Cochrane Database Syst Rev 2006. doi:10.1002/14651858.CD001732.pub2
- [7] Mann MW. Sclerotherapy: it is back and better. Clin Plast Surg 2011; 38: 475–487, vii. doi:10.1016/j.cps.2011.02.006

- [8] Kobayashi S, Crooks S, Eckmann DM. Dose- and time-dependent liquid sclerosant effects on endothelial cell death. Dermatol Surg 2006; 32: 1444–1452. doi:10.1111/j.1524-4725.2006.32350.x
- [9] Goldman MP. Treatment of varicose and telangiectatic leg veins: doubleblind prospective comparative trial between aethoxyskerol and sotradecol. Dermatol Surg 2002; 28: 52–55. doi:10.1046/j.1524-4725.2002.01190.x
- [10] Bush R, Bush P. Evaluation of sodium tetradecyl sulfate and polidocanol as sclerosants for leg telangiectasia based on histological evaluation with clinical correlation. Phlebology 2017; 32: 496–500. doi:10.1177/ 0268355516673768
- [11] Parsi K, Exner T, Connor DE et al. The lytic effects of detergent sclerosants on erythrocytes, platelets, endothelial cells and microparticles are attenuated by albumin and other plasma components in vitro. Eur J Vasc Endovasc Surg 2008; 36: 216–223. doi:10.1016/j.ejvs.2008.03.001
- [12] Goldman MP BJ, Guex JJ. Sclerotherapy Treatment of Varicose and Telangiectatic Leg Veins. Fourth Edition. Aufl Mosby Elsevier Inc; 2007
- [13] Rathbun S, Norris A, Stoner J. Efficacy and safety of endovenous foam sclerotherapy: meta-analysis for treatment of venous disorders.
 Phlebology 2012; 27: 105–117. doi:10.1258/phleb.2011.011111
- [14] Davies HO, Popplewell M, Darvall K et al. A review of randomised controlled trials comparing ultrasound-guided foam sclerotherapy with endothermal ablation for the treatment of great saphenous varicose veins. Phlebology 2016; 31: 234–240. doi:10.1177/0268355515595194
- [15] Carugo D, Ankrett DN, Zhao X et al. Benefits of polidocanol endovenous microfoam (Varithena®) compared with physician-compounded foams. Phlebology 2016; 31: 283–295. doi:10.1177/0268355515589063
- [16] Cavezzi A, Tessari L. Foam sclerotherapy techniques: different gases and methods of preparation, catheter versus direct injection. Phlebology 2009; 24: 247–251. doi:10.1258/phleb.2009.009061
- [17] Lai SW, Goldman MP. Does the relative silicone content of different syringes affect the stability of foam in sclerotherapy? J Drugs Dermatol 2008; 7: 399–400
- [18] Rao J, Goldman MP. Stability of foam in sclerotherapy: differences between sodium tetradecyl sulfate and polidocanol and the type of connector used in the double-syringe system technique. Dermatol Surg 2005; 31: 19–22. doi:10.1111/j.1524-4725.2005.31008
- [19] de Roos KP, Groen L, Leenders AC. Foam sclerotherapy: investigating the need for sterile air. Dermatol Surg 2011; 37: 1119–1124. doi:10.1111/ j.1524-4725.2011.02044.x
- [20] McMaster S. Sodium tetradecyl sulphate foam stability prior to injection: factors affecting liquid reformation. Phlebology 2011; 26: 222–226. doi:10.1258/phleb.2010.010025
- [21] Beckitt T, Elstone A, Ashley S. Air versus physiological gas for ultrasound guided foam sclerotherapy treatment of varicose veins. Eur J Vasc Endovasc Surg 2011; 42: 115–119. doi:10.1016/j.ejvs.2011.04.005
- [22] Weiss RA, Weiss MA. Doppler ultrasound findings in reticular veins of the thigh subdermic lateral venous system and implications for sclerotherapy. J Dermatol Surg Oncol 1993; 19: 947–951. doi:10.1111/ j.1524-4725.1993.tb00983.x
- [23] Mann MW. Sclerotherapy: it is back and better. Clin Plast Surg 2011; 38: 475–487, vii. doi:10.1016/j.cps.2011.02.006
- [24] Tripey V, Monsallier JM, Morello R et al. French sclerotherapy and compression: Practice patterns. Phlebology 2015; 30: 632–640. doi:10.1177/0268355514554024

- [25] Weiss RA, Sadick NS, Goldman MP et al. Post-sclerotherapy compression: controlled comparative study of duration of compression and its effects on clinical outcome. Dermatol Surg 1999; 25: 105–108. doi:10.1046/j.1524-4725.1999.08180.x
- [26] Raj TB, Makin GS. A random controlled trial of two forms of compression bandaging in outpatient sclerotherapy of varicose veins. J Surg Res 1981; 31: 440–445. doi:10.1016/0022-4804(81)90084-6
- [27] Guex JJ. Complications of sclerotherapy: an update. Dermatol Surg 2010; 36 (Suppl. 2): 1056–1063. doi:10.1111/j.1524-4725.2009.01409.x
- [28] Goldman MP, Kaplan RP, Duffy DM. Postsclerotherapy hyperpigmentation: a histologic evaluation. J Dermatol Surg Oncol 1987; 13: 547–550. doi:10.1111/j.1524-4725.1987.tb00940.x
- [29] Rabe E, Partsch H, Hafner J et al. Indications for medical compression stockings in venous and lymphatic disorders: An evidence-based consensus statement. Phlebology 2018; 33: 163–184. doi:10.1177/ 0268355516689631
- [30] Schuller-Petrović S, Pavlović MD, Neuhold N et al. Subcutaneous injection of liquid and foamed polidocanol: extravasation is not responsible for skin necrosis during reticular and spider vein sclerotherapy. J Eur Acad Dermatol Venereol 2011; 25: 983–986. doi:10.1111/ j.1468-3083.2010.03873.x
- [31] Miyake RK, King JT, Kikuchi R et al. Role of injection pressure, flow and sclerosant viscosity in causing cutaneous ulceration during sclerotherapy. Phlebology 2012; 27: 383–389. doi:10.1258/phleb.2011.011076
- [32] Willenberg T, Smith PC, Shepherd A et al. Visual disturbance following sclerotherapy for varicose veins, reticular veins and telangiectasias: a systematic literature review. Phlebology 2013; 28: 123–131. doi:10.1258/phleb.2012.012051
- [33] Guex JJ, Allaert FA, Gillet JL et al. Immediate and midterm complications of sclerotherapy: report of a prospective multicenter registry of 12,173 sclerotherapy sessions. Dermatol Surg 2005; 31: 123–128; discussion 128. doi:10.1111/j.1524-4725.2005.31030
- [34] Frullini A, Barsotti MC, Santoni T et al. Significant endothelin release in patients treated with foam sclerotherapy. Dermatol Surg 2012; 38: 741– 747. doi:10.1111/j.1524-4725.2012.02390.x
- [35] Gillet JL. Neurological complications of foam sclerotherapy: fears and reality. Phlebology 2011; 26: 277–279. doi:10.1258/phleb.2011.011e04
- [36] Guex JJ. Complications and side-effects of foam sclerotherapy. Phlebology 2009; 24: 270–274. doi:10.1258/phleb.2009.009049
- [37] Hill D, Hamilton R, Fung T. Assessment of techniques to reduce sclerosant foam migration during ultrasound-guided sclerotherapy of the great saphenous vein. J Vasc Surg 2008; 48: 934–939. doi:10.1016/ j.jvs.2008.05.077
- [38] Parsi K. Venous gas embolism during foam sclerotherapy of saphenous veins despite recommended treatment modifications. Phlebology 2011; 26: 140–147. doi:10.1258/phleb.2010.010030
- [39] Morrison N, Neuhardt DL, Rogers CR et al. Comparisons of side effects using air and carbon dioxide foam for endovenous chemical ablation. J Vasc Surg 2008; 47: 830–836. doi:10.1016/j.jvs.2007.11.020