

# Complications of great and small saphenous vein sclerotherapy: Case analysis and clinical recommendations

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## ABSTRACT

Sclerotherapy is a widely accepted treatment for reticular veins and telangiectasias, but its use in major truncal veins such as the great saphenous vein (VSM) and small saphenous vein (VSP) remains controversial. This paper presents several clinical cases of patients who developed serious complications, including non-healing ulcers and chronic limb edema, following truncal vein sclerotherapy. We discuss the contraindications for sclerotherapy in these large superficial veins, the pathophysiological reasons for complications, and review current best practices for the management of varicose veins, including endovenous thermal ablation and surgical options. Sclerotherapy in major truncal veins remains controversial due to frequent complications and the short-term effect.

**Key words:** Sclerotherapy, Varicose veins, Complications, Ulcers

## INTRODUCTION

Varicose veins represent one of the most common manifestations of chronic venous disease, affecting up to 25–30% of the adult population. To improve the therapeutic possibilities, we must always begin from the base, that is, the etiology and pathophysiology of the disease [1].

Chronic venous insufficiency (CVI) is a condition where the flow in the veins is impaired and venous hypertension occurs. In CVI, several changes occur in the lower extremities, such as edema, trophic changes in the skin, and a feeling of heaviness in the legs. The diagnosis of CVI is based on clinical features according to the Clinical, Etiological, Anatomical, and Pathophysiological (CEAP) classification to improve consistency in the reporting, diagnosis, and management of CVI.

Most systemic diseases impact the process of vascularization, granulation, and finally epithelization of

the ulcer tissue [1]. The primary underlying mechanism for CVI is valvular reflux, other etiologies involved in the disease process are venous stasis, arteriovenous malformation and failure of the calf muscle pump, lifestyle, lower abdominal surgery, pregnancy, obesity, lower extremity injury. There may also be a hereditary component, i.e., genetic disorders such as Klippel–Trénaunay and Parks Weber causing CVI. These etiological factors may lead to chronic endothelial inflammation and other pathophysiological changes [2].

The approach to CVI management involves several different strategies, including conservative therapies (e.g., compression, elevation, and exercise), pharmacologic treatments, and more invasive procedures (sclerotherapy, endovenous laser or radiofrequency ablation, surgical ligation) [2].

## Sclerotherapy

While sclerotherapy is a simple and minimally invasive procedure widely used for treating varicose,

**How to cite this article:** Vasileva M, Spoa D. Complications of great and small saphenous vein sclerotherapy: Case analysis and clinical recommendations. Our Dermatol Online. 2026;17(2):228-233.

**Submission:** 24.10.2025; **Acceptance:** 01.12.2025

**DOI:** 10.7241/ourd.20262.17

telangiectasias, reticular, spider veins, and some hemorrhoids, its application in the great saphenous vein (VSM) and small saphenous vein (VSP) remains controversial.

Injection sclerotherapy induces vascular fibrosis, improves cosmesis, relieves venous insufficiency symptoms, and improves overall vascular health but in small venous blood vessels.

The great saphenous vein and small saphenous vein play a central role in venous return from the lower extremities, and obliteration with a sclerosant carries a risk of significant complications, including recanalization, thrombosis, pigmentation, tissue necrosis, and chronic edema.

The sclerosants commonly employed in current clinical practice may be classified as irritants, osmotic agents, or detergents [3].

Sclerosants damage endothelial cells of the intima and tunica. Irritants and osmotic sclerosants are directly cytotoxic; detergents are less cytotoxic yet disrupt intercellular junctions [3].

In this paper, we present several cases of severe complications following truncal vein sclerotherapy and discuss why this approach should be avoided in favor of evidence-based alternatives.

### **Contraindications and Complications in Sclerotherapy**

There are absolute and relative contraindications to sclerotherapy for varicose veins. Absolute contraindications to sclerotherapy include patients with hypersensitivity to sclerosing agents, acute systemic infection, or acute localized infection at the site of treatment, deep vein thrombosis, peripheral arterial disease, systemic autoimmune connective tissue diseases.

Relative contraindications to sclerotherapy include pregnancy, lactation, impaired mobility, asthma, deep venous insufficiency, and thrombophilia.

Large varicose veins are also a contraindication to sclerotherapy due to the increased risk of recanalization, complications, and treatment failure.

In patients in whom sclerotherapy is contraindicated, other treatment options should be considered.

Complications of sclerotherapy can be systemic and local. Local complications occur at the injection site and include pain, erythema, edema, ulceration, pruritus, and telangiectatic changes. Approximately 30% of patients may experience skin hyperpigmentation or discoloration from hemosiderin deposition 6 to 8 weeks after sclerotherapy [3].

Systemic side effects of sclerotherapy include chest tightness, transient ischemic attacks, dizziness, visual problems, and headaches. These side effects are more common in patients who have had a problem with injecting the sclerosant into the wrong vein. A systematic review found no significant differences in complication rates or optimal outcomes between foam and liquid sclerotherapy [3].

The patient must always be advised that there is a risk of skin necrosis or cosmetic complications such as pigmentation and telangiectasias, all patients who are on oral contraceptives and other exogenous estrogens, tetracyclines as well as those receiving medications for psychiatric diagnoses may increase the risk of side effects or compromise optimal treatment outcomes [4].

Sclerotherapy of VSM/VSP is a poor choice because of the high recurrence rate. A foam or liquid sclerosant often fails to permanently occlude long truncal veins. Also, there is a risk of deep vein extension due to saphenofemoral or saphenopopliteal junction reflux. The sclerosant may enter the deep system. Tissue necrosis and ulceration are also important complications due to accidental extravasation or reflux into tributaries causing skin breakdown. A very common complication is chronic limb edema because of loss of major superficial venous drainage that may worsen venous hypertension if collaterals are inadequate.

Because VSP is close to the sural nerve, it can be damaged. Cosmetic complications are the most common but less dangerous; the legs are with pigmentation and matting.

### **CASE REPORT 1**

A 68-year-old female patient came for an examination due to an ulcer on the right foot after sclerosis eight months previously (Fig. 1). She had been treated several times with bandages, antibiotics, but without improvement. She gave information that pronounced swelling, redness and wounds on the right lower leg occurred after intervention st. post sclerosatio VSM



**Figure 1:** Lower leg eroded with ulceration and impetiginization.

lat. dex, st. post sclerosatio v. Parva lat. dex 8 months ago. The patient is DM type 2 on insulin, HBB, CVI ischemicum regio parietalis lat. syn., Dihemiparesis pp lat.dex., HTA. She was regularly followed by the endocrinologist. *Pseudomonas aeruginosa* was isolated from a wound swab. On clinical examination, the patient was hardly mobile, with pronounced swelling of the right lower leg, erythema. On the heel, there was ulceration with a granulated bottom and scars. Above the malleolus, the entire lower third of the lower leg was eroded.

A Doppler echo was performed, which registered the presence of atherosclerotic plaques in all arteries and deep venous insufficiency in the right leg. Laboratory analyses showed leukocytosis, elevated D dimers, and high CRP.

The patient had pronounced lymphedema and compression therapy was completely contraindicated, we hospitalized her and she was placed on a diuretic, a broad-spectrum antibiotic and regular hydrocolloid dressings, and advised to elevate her leg. Unfortunately, the patient suffered another ischemic stroke, after which she fell into a coma for several days and died.

## CASE REPORT 2

A 68-year-old male patient presented to the doctor due to the recurrence of a venous ulcer on the right foot (Fig. 2). He reported that he had had recurrent venous ulcers and lymphedema for the past three years. The first swelling and appearance of the ulcer occurred



**Figure 2:** Stasis dermatitis, ulceration, and scars from past ulcerations.

shortly after sclerosing the right great saphenous vein three years previously. On clinical examination, varicose veins were visible on the left limb. On the right limb, the middle and lower third of the lower leg was stasis dermatitis and, in places, the beginning of lipodermatosclerosis and ulceration.

Echo Doppler of the lower extremities:

Arterial flow - regular Doppler signal at all levels of AFS, AP, ATP, ATA

Venous flow - left - deep veins flowing and compressible, insufficient especially v. popliteal

Left - SFU - not visible, VSM was not visible to the upper third of the thigh, in the middle third of the thigh, a perforator was present. VSM was dilated and branched, especially around the knee joint with numerous varicosities. No acute thrombotic process was observed. Organized lymph nodes were observed on the left inguinal.

Right arterial triphasic regular Doppler signal at the level of AFS, AP. Due to pronounced swelling of the lower leg and foot, ATP and ATA signals cannot be obtained. Deep veins on the right were compressible and insufficient. Superficial veins - VSM was observed in the upper third of the upper leg, then it was occluded, but numerous insufficient varicose veins observed in the upper leg much more pronounced at the level of the knee joint and lower leg where several perforators and several varicosities that were non-compressible as part of the PTS. VSM appeared in the lower third of the lower leg, dilated and insufficient. At the level of the entire lower leg and ankle, pronounced edema

was observed. VSP dilated with numerous varicosities and a perforator in the middle third. In the right inguinal region, an enormously enlarged lymph node was present. The patient was placed on hydrocolloid dressings and compression therapy was given: oral anticoagulant therapy rivaroxaban 20 mg continuously for three months and troxerutin 300 mg twice daily for three months. After complete healing of the ulcer, which healed with the formation of a scar, the patient was referred to regular lymphatic drainage.

A recommendation was given after a three-month break to take troxerutin 300 mg twice a day for another three months, vitamin C 1000 mg continuously, in combination with lymphatic massage and compression therapy. In this way, the patient has not opened a new venous ulcer for a year, although the problem with lymphedema remains.

### CASE REPORT 3

A 74-year-old man presented for examination due to a persistent ulcer for more than four months after sclerosing of the saphenous vein magna and saphenous vein parva on the right leg (Fig. 3). After the treatment, the condition worsened with swelling, hyperpigmentation and the beginning of ulceration. The patient was admitted to the same institution for several months for glutathione infusion therapy and surgery without success. At the time of the examination, extensive ulcerations were present on the right lower leg with dimensions of 20 x 7 cm, larger and the size of a child's hand, two smaller posteriorly. There were ulcers with zones of necrosis, fibrin plaques, in places deeper and



**Figure 3:** Ulcer with dimensions of 20 x 7 cm, zones of necrosis, fibrin plaques.

with an unpleasant odor. An echo Doppler examination of the right extremity was performed. Arterial flow - AFS with two-phase extended Doppler signal with discrete atherosclerotic changes at the beginning and with larger occlusive changes in the middle third of the thigh. ATP so monophasen continuous Doppler signal. Venous flow - superficial veins were completely non-compressible. Dlaboki vein insufficiency. There was an expressed island along the entire length. The entire right foot was swollen and fluctuant, during which an incision was made, from which purulent contents were drained. During the drainage, a rubber drain came out from the inside that was placed three months ago during a bandage and never removed. An X-ray of the foot was done.

The X-ray of the right foot showed osteopenia of the depicted skeleton of the right foot with advanced arthritic changes with asymmetric and reduced distal and proximal interphalangeal joint spaces. There was subchondral sclerosis of the joint surfaces and luxation of the proximal phalanx of the V finger.

The patient had myasthenia gravis and was on chronic therapy with Decortin 25 mg, tbl. Ciclosporin 50 mg 2x1, tbl. pyridostigmine 60 mg. The patient was admitted for a swab from the wound. *Staphylococcus aureus* was isolated and antibiotic therapy was prescribed according to the antibiogram ceftriaxone. Wound debridement was performed and daily dressings with a spray containing stable ozonides with vitamin E acetate, tea tree leaf oil *Melaleuca alternifolia*, thioctic acid were started. After a month and a half, we began doing PRF once a week on the patient, and the results were visible after the first week. The patient was not a candidate for wound closure with his own skin due to the dimensions of the wound and the peripheral arterial occlusive disease. The patient also received vitamin C 1000 mg, vitamin E 400 IU, cilostazol 100 mg 2x1, and acetylsalicylic acid 100 mg 1x1. The patient is still using these medications, took trypsin, chymotrypsin, and serrapeptase for a month. The patient showed significant improvement, although the healing process was long. After only a month there, there was a significant reduction in the dimensions of the wounds. After two months, the wound was already at skin level. The swelling and pain in the foot resolved in two weeks.

### DISCUSSION

Sclerosis of the great saphenous vein and the lesser saphenous vein carries greater risks than benefits.

Given the duration of the effect of sclerosing, and the magnitude of the side effects and complications, according to our experience and practice, the treatment of saphenous veins with sclerosing is risky. In order to perform sclerosing of the saphenous veins, a good assessment is required in order to minimize the risk of complications that the patient then faces for a long period of time and some, unfortunately, for life. According to numerous studies, foam sclerotherapy has a low complication rate, low cost, and acceptable total occlusion rate and reproducibility [5]. However, our practice has shown that complications are quite common, although all of them are present in patients who are probably not followed up to the end or are not well investigated for existing comorbidities. According to research, thrombophilia may also be considered a contraindication for sclerotherapy, after a large group of patients who were studied retrogradely and faced thromboembolism shortly after the performed sclerosing of the saphenous veins; however, most had thrombophilia diagnosed after the incident [6].

Skin necrosis as a complication is described both after the perivascular injection of high-percentage sclerosant and, in rare cases, after correct intravascular injection of the sclerosant at low concentrations [7].

To reduce the risk of skin necrosis, the injection of large volumes at any injection point should be avoided. The sclerosant should be injected at the lowest possible pressure [6].

Extensive necrosis may occur following inadvertent intra-arterial injection; the risk of this complication may be reduced by the use of ultrasound to identify blood vessels. If severe pain occurs during the injection of the sclerosant, the procedure should be stopped immediately. If intra-arterial injection is suspected, anticoagulant therapy should be administered. Prompt administration of systemic corticosteroids may help reduce the inflammatory reaction.

Sometimes, there are malformations of the blood vessels, and then complications are almost inevitable, which is why a thorough duplex ultrasound is recommended, and sometimes additional examinations are needed to clarify the anatomical and hemodynamic condition.

Other transient general or local reactions that may occur after sclerotherapy include chest tightness, vasovagal syncope, metallic taste in the mouth or

nausea, thrombus, hematoma, ecchymosis or pain at the injection site, and the appearance of local swelling, induration and erythema [7].

Deep venous insufficiency undiagnosed before sclerotherapy will lead to the appearance of lymphedema, a condition that is permanent, and the patient will face swelling in the extremities for life, all of which will lead to secondary wound opening, impetigo of cracked skin, immobility, and reduced quality of life.

It is important that, before sclerotherapy, a diagnosis shall be obtained, including medical history and clinical and duplex ultrasound examinations, and that a good assessment be made.

## CONCLUSION

Before sclerotherapy, patients should be informed about possible complications and side effects. Each patient should be aware of alternative treatment methods with their advantages and disadvantages, as well as details about the entire sclerotherapy procedure. Each possible complication should be explained, as well as common side effects.

Regarding the expected outcome of sclerotherapy, patients should be informed that more frequent controls or re-treatment may be necessary.

Foam sclerotherapy is more effective than liquid sclerotherapy for subcutaneous varicose veins.

Ultrasound-guided foam sclerotherapy may avoid the need for intra-arterial injection.

Sclerotherapy of the great and small saphenous veins carries an unacceptably high risk of complications compared to modern alternatives. Based on clinical evidence and the cases presented, truncal vein sclerotherapy should not be considered a primary treatment option. Endovenous ablation techniques remain the gold standard, with proven efficacy, durability, and safety.

## Consent

The examination of the patient was conducted according to the principles of the Declaration of Helsinki.

The authors certify that they have obtained all appropriate patient consent forms, in which the patients gave their consent for images and other clinical information to be included in the journal. The

patients understand that their names and initials will not be published and due effort will be made to conceal their identity, but that anonymity cannot be guaranteed.

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**Source of Support:** This article has no funding source.  
**Conflict of Interest:** The authors have no conflict of interest to declare.