Early Access
Review

Veins and Lymphatics

eISSN 2279-7483
https://www.pagepressjournals.org/index.php/vl/index

Veins and Lymphatics 2026 [online ahead of print]

To cite this article:
Bentivedo A, De Ruggieri G, Floris M, Mastrangeli MR. When sclerotherapy leaves its mark: prevention and management of cutaneous complications. Veins and Lymphatics 2026;15:14912.
doi:10.4081/vl.2026.14912

When sclerotherapy leaves its mark: prevention and management of cutaneous complications

Angela Bentivedo,[1] Giuseppe De Ruggieri,[2] Micol Floris,[3] Maria Rosaria Mastrangeli[4]

[1]General Practice Clinic, ASL 6, Rome;

[2]Department of General Surgery, Policlinico Umberto I Hospital, Rome;

[3]Plastic Surgery Department, Ospedale S. Eugenio Hospital, Rome;

[4]Department of General Surgery, Ospedale S. Eugenio Hospital, Rome, Italy;

Abstract

Sclerotherapy represents a cornerstone in the treatment of chronic venous disease and superficial venous disorders, combining effectiveness, versatility, and a favorable safety profile. Despite these advantages, cutaneous complications remain clinically relevant because of their potential impact on aesthetic outcomes, patient satisfaction, and, in rare cases, tissue integrity. Cutaneous adverse events range from common and usually self-limiting manifestations, such as ecchymosis, hyperpigmentation, and telangiectatic matting, to uncommon but potentially severe complications including skin ulceration and necrosis. This narrative review provides a clinically oriented and critical overview of cutaneous complications associated with sclerotherapy, focusing on pathophysiological mechanisms, patient- and procedure-related risk factors, prevention strategies, and management options. Particular emphasis is placed on practical clinical decision-making, patient counseling, and early recognition of warning signs, with the aim of supporting safe practice and identifying areas in order to support future research.

Key words: cutaneous adverse events; venous interventions; hyperpigmentation disorders; telangiectatic matting; skin necrosis.

Correspondence: Angela Bentivedo, General Practice Clinic, ASL 6, Rome, Italy;
E-mail: dottoressabentivedo@gmail.com

Introduction

Sclerotherapy is a well-established technique for the treatment of telangiectasias, reticular veins, and varicose veins. Contemporary practice includes both liquid and foam formulations, supported by clinical guidelines and large clinical experience.[1-3] Although overall safety is favorable strong, adverse events remain part of the procedure's risk profile. Cutaneous complications are particularly important because they directly affect visible outcomes, often in patients who seek treatment for cosmetic reasons, and because a small subset can result in clinically significant tissue injury.[4,5] Importantly, patient perception of "minor" cutaneous events may be disproportionate to their medical severity; therefore, prevention and counseling are integral components of quality care.[2,4]

Narrative reviews provide an opportunity to integrate evidence with real-world clinical reasoning, especially in fields where randomized trials are limited for rare outcomes. Accordingly, this review summarizes the spectrum of cutaneous complications following sclerotherapy, discusses mechanisms and debated issues, and proposes a pragmatic framework for prevention and management.

Classification of cutaneous complications

Cutaneous complications following sclerotherapy can be classified according to frequency and clinical severity. A concise overview of reported incidence, risk factors, timing, and expected course is provided in Table 1.

Frequent and usually self-limiting events

Frequent cutaneous events include injection-site erythema, localized urticarial wheals, pruritus, transient edema, and ecchymosis/hematoma. These are typically early events

related to mechanical trauma, immediate inflammatory response, or local histamine-mediated phenomena.[2,4] Postsclerotherapy hyperpigmentation and telangiectatic matting are among the most common delayed cosmetic sequelae and remain leading contributors to dissatisfaction after technically successful procedures.[2,4,7]

Uncommon and rare but clinically significant events

Less common complications include persistent inflammation with superficial thrombophlebitis in the treated segment, cutaneous blistering, localized contact dermatitis (often related to compression materials), and infection at needle entry sites. Rare but clinically significant complications include skin ulceration and tissue necrosis, which may result in scarring and prolonged wound care.[2,3] Inadvertent intra-arterial injection is a rare but feared mechanism leading to ischemic injury and extensive necrosis.[6]

Pathophysiology of cutaneous complications

The principal mechanisms linking sclerotherapy to cutaneous sequelae are summarized in Figure 1. In brief, sclerosants induce endothelial injury and a controlled inflammatory response. When these effects extend beyond the target lumen - due to erythrocyte extravasation, persistent intraluminal thrombus, high local concentration, extravasation, vasospasm, or arterial occlusion - cutaneous complications may arise.[2,4]

Ecchymosis, hematoma, and early inflammatory reactions

Ecchymosis and localized hematoma reflect venipuncture trauma and perivenous leakage of blood. Although usually self-limiting, extensive bruising can delay perceived recovery and may coexist with post-inflammatory hyperpigmentation. Immediate localized wheals

or pruritus can occur as a nonspecific inflammatory response or mild hypersensitivity. These events are generally managed conservatively and are best prevented by gentle technique and appropriate post-procedural compression.[4]

Postsclerotherapy hyperpigmentation

Postsclerotherapy hyperpigmentation typically presents as linear or macular brown discoloration overlying the treated vein. Histology and clinical series support hemosiderin deposition as a major contributor, driven by erythrocyte extravasation and macrophage processing of iron.[8] Prospective follow-up suggests that most cases fade over months, with persistence beyond one year in a minority.[6] Importantly, retained intravascular coagulum is thought to intensify local inflammation and increase iron deposition, providing a rationale for early clot evacuation when clinically appropriate.[4]

Telangiectatic matting

Telangiectatic matting consists of fine (<0.2 mm) erythematous vessels that develop weeks after treatment, often in a network pattern adjacent to treated veins. A large retrospective analysis reported an overall incidence around 16% in clinical practice, although rates vary by population and technique.[7] Proposed mechanisms include reactive angiogenesis, microcirculatory changes, and treatment of only the visible telangiectasias without addressing feeder reticular veins or underlying reflux.[2,7,8] Individual susceptibility - potentially influenced by hormonal factors - appears to modulate risk and persistence.

Superficial thrombophlebitis and retained coagulum

A tender cord or localized inflammation along a treated vein can reflect superficial thrombophlebitis or retained coagulum within the sclerosed segment. Besides discomfort, retained coagulum is clinically relevant because it may be associated with prolonged inflammation and hyperpigmentation.[2]

Ulceration and necrosis

Tissue necrosis after sclerotherapy is rare but may be disfiguring. Contemporary analyses highlight that round necrosis is often associated with extravasation, whereas stellate (star-like) necrosis more commonly reflects ischemic injury secondary to arterial/arteriolar compromise. Mechanisms include sclerosant extravasation, high-pressure injection, veno-arteriolar reflex vasospasm, and inadvertent intra-arterial injection.[2] Early warning signs include disproportionate pain, burning, and immediate blanching or livedoid change at the injection site.

Risk factors

Patient-related risk factors

Patient-related factors associated with an increased probability of cutaneous sequelae include fair skin phototype, tendency to post-inflammatory pigment alteration, obesity, and extensive telangiectatic disease. Underlying venous reflux or untreated reticular feeder veins may predispose to matting and recurrence of telangiectasias.[2,7,8]

Procedure-related risk factors

Procedure-related risk factors include use of higher-than-necessary sclerosant concentration or volume, injection under excessive pressure (especially in small

intradermal vessels), inadequate visualization of the target lumen, and insufficient post-procedural compression.[2,4] For severe ischemic events, risk is increased in anatomically vulnerable regions with potential arteriole proximity, and when patient feedback is limited or pain signals are ignored.[2]

Prevention strategies

Prevention remains the cornerstone of managing cutaneous complications. The European Guidelines for sclerotherapy provide practical recommendations on indications, concentrations, technique, and side effects for polidocanol and sodium tetradecyl sulfate.[2,3] Core preventive principles include: (I) selecting the lowest effective sclerosant dose and concentration for the target diameter; (II) using slow, low-pressure injection; (III) maintaining good visualization and avoiding intradermal injection when not intended; (IV) stopping immediately if severe pain, burning, or blanching occurs; (V) applying appropriate compression and post-procedural instructions; and (VI) considering early evacuation of retained intravascular coagulum to reduce inflammation and pigmentation risk.[2]

For telangiectatic matting, prevention is strongly linked to a hemodynamic approach: identifying and treating feeder reticular veins and, when clinically indicated, addressing underlying reflux before or in staged fashion, rather than overtreating superficial telangiectasias in isolation.[2,7,8]

Management of cutaneous complications

A pragmatic summary of prevention and management strategies is provided in Table 2. Management should be individualized according to severity, timing, patient preference, and local expertise (Table 3).

Ecchymosis, hematoma, and early inflammatory reactions

Most early reactions are managed conservatively with reassurance and symptomatic care. Compression and avoidance of local trauma are usually sufficient. When localized contact dermatitis is suspected, clinicians should consider sensitivity to adhesives or compression materials and adjust accordingly.

Postsclerotherapy hyperpigmentation

First-line management is observation and counseling, emphasizing that gradual fading over months is expected in most patients.[6] When clinically appropriate, evacuation of retained coagulum by needle aspiration or expression through a small incision has been proposed to reduce inflammation and potentially shorten pigmentation persistence.[2] For persistent pigment beyond a typical recovery window, selected patients may be candidates for laser or light-based modalities, acknowledging heterogeneity in protocols and outcomes.

Telangiectatic matting

Initial management is conservative, as spontaneous improvement is common. Persistent matting should prompt reassessment for untreated feeder veins or reflux. Additional targeted treatment of feeder reticular veins may be more effective than repeated aggressive treatment of the fine matting vessels themselves.[2,7,8] Adjunctive vascular laser therapy can be considered when appropriate expertise and device access are available.

Superficial thrombophlebitis and retained coagulum

Painful or inflamed treated segments should be evaluated to distinguish expected post-sclerotherapy changes from superficial thrombophlebitis or extensive retained coagulum. In addition to symptom control, addressing retained coagulum may reduce prolonged inflammation and post-treatment pigment.[2]

Suspected extravasation and ischemic injury: immediate actions

When extravasation or ischemia is suspected during injection, immediate cessation is mandatory. The injection site should be assessed for blanching, livedoid change, severe pain, and evolving purpura. Early intervention strategies described in the literature include dilution and dispersion approaches, with hyaluronidase showing protective effects in experimental models of extravasation necrosis.[9] For suspected intra-arterial injection, case series emphasize rapid recognition and urgent escalation, as outcomes depend on timely restoration of perfusion and mitigation of thrombosis/vasospasm.[5] Recent guidance and expert proposals underline the need for a structured emergency plan in practices performing sclerotherapy.[10]

Ulceration and necrosis: wound care and follow-up

Once ulceration or necrosis is established, management centers on wound care principles, pain control, infection prevention, and close follow-up. Severe cases may require multidisciplinary input. Case reports and literature review of atypical ulcers after foam sclerotherapy illustrate prolonged healing and residual scarring, reinforcing the importance of early recognition and aggressive wound management when needed.[3]

Patient counseling and informed consent

Given that many patients undergo sclerotherapy for cosmetic indications, counseling must explicitly address the probability of transient bruising, the possibility of delayed hyperpigmentation and matting, and the rare risk of ulcer or necrosis. Counseling should include expected timelines, variability of recovery, and the availability of management options. Transparent communication aligns expectations and reduces dissatisfaction and medico-legal risk.[2,4]

Future perspectives

Several knowledge gaps persist. High-quality prospective data on predictors of persistent hyperpigmentation and matting, standardized definitions of clinically meaningful persistence, and comparative effectiveness of interventions are needed. For necrosis and intra-arterial injection, multicenter registries and consensus-driven emergency algorithms may improve preparedness and outcomes.[2,10]

Conclusions

Cutaneous complications are an inherent aspect of sclerotherapy. Most are mild and self-limiting, but their aesthetic impact can be substantial. A careful hemodynamic assessment, meticulous technique, proactive prevention, early recognition of warning signs, and structured management pathways remain fundamental to safe practice.

References

1. Rabe E, Pannier F. Sclerotherapy in the treatment of varicose veins. Dtsch Arztebl Int 2020;117:303-10.

2. De Maeseneer MG, Kakkos SK, Aherne T. Editor's Choice - European Society for Vascular Surgery (ESVS) 2022 Clinical Practice Guidelines on the Management of Chronic Venous Disease of the Lower Limbs. Eur J Vasc Endovasc Surg 2022;63:184-267.

3. Wong M, Parsi K, Myers K, Sclerotherapy of lower limb veins: Indications, contraindications and treatment strategies to prevent complications - A consensus document of the International Union of Phlebology-2023. Phlebology 2023;38:205-58.

4. Bossart S, Ramelet AA, Seyed Jafari SM. Revealing the origin of postsclerotherapy hyperpigmentation: identification of melanin and hemosiderin as causative pigments in a histopathological study. Phlebology 2026;:217-23.

5. Gloviczki P, Lawrence PF, Wasan SM. The 2023 Society for Vascular Surgery, American Venous Forum, and American Vein and Lymphatic Society clinical practice guidelines for the management of varicose veins of the lower extremities. Part II: Endorsed by the Society of Interventional Radiology and the Society for Vascular Medicine. J Vasc Surg Venous Lymphat Disord 2024;101670.

6. Kang M, Yang A, Lee BB. Skin necrosis following sclerotherapy. Part 1: Differential diagnosis and classification. Phlebology 2022;37:683-92.

7. Kang M, Yang A, Lee BB. Skin necrosis following sclerotherapy. Part 2: Risk minimisation and management strategies. Phlebology 2022;37:693-701.

8. Rabe E, Pannier F. Indications, contraindications and performance: European Guidelines for Sclerotherapy in Chronic Venous Disorders. Phlebology 2014;29:26-33.

9. Rai KM, Rao KS, Maudar KK. Accidental intra-arterial drug injection. Med J Armed Forces India 2017;53:137-9.

10. Parsi K, De Maeseneer M, van Rij M, et al. Guidelines for management of actual or suspected inadvertent intra-arterial injection of sclerosants. Phlebology 2024;39:683-719.

Contributions: all the authors made a substantive intellectual contribution. All the authors have read and approved the final version of the manuscript and agreed to be held accountable for all aspects of the work.

Conflict of interest: the authors declare no potential conflict of interest.

Funding: none.

Ethics approval and consent to participate: not applicable.

Informed consent: not applicable.

Patients' consent for publication: not applicable.

Availability of data and materials: all data generated or analyzed during this study are included in this published article.

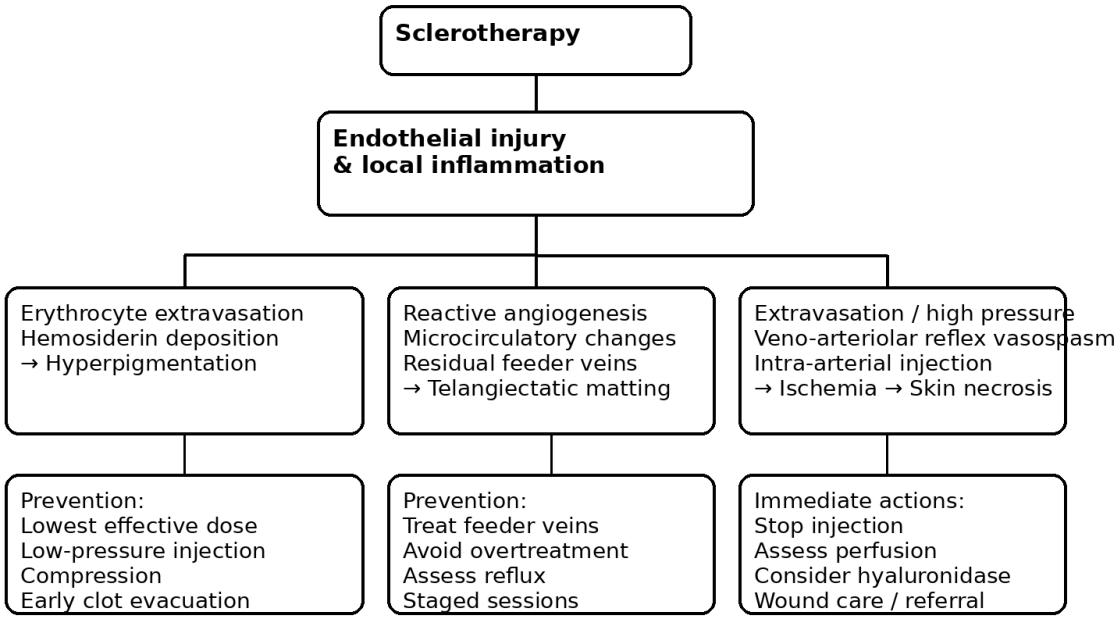

Figure 1. Pathophysiological mechanisms of cutaneous complications following sclerotherapy.

Table 1. Cutaneous complications after sclerotherapy: incidence, risk factors, and clinical course.

Complication	**Reported incidence**	**Main risk factors**	**Typical onset**	**Natural course**
Ecchymosis/hematoma	Common	Trauma, anticoagulation, fragile vessels	Immediate	Resolves in days-weeks
Early inflammatory wheals/pruritus	Common	Local inflammatory response, mild hypersensitivity	Immediate	Self-limited
Hyperpigmentation	~10-30% (varies)	Higher concentration, larger veins, retained coagulum	Weeks	Fades over months; minority >1 year
Telangiectatic matting	~5-20% (variable)	Hormonal factors, feeder veins, residual reflux	Weeks	Often improves; may persist
Superficial thrombophlebitis/retained coagulum	Uncommon	Larger segments, incomplete emptying	Days-weeks	May prolong inflammation/pigment
Contact dermatitis (compression/adhesives)	Uncommon	Allergy/irritation to materials	Days	Resolves after removal/therapy
Ulceration	<1%	Extravasation, high-pressure injection	Immediate-days	Variable; may scar
Skin necrosis	Rare	Ischemia, intra-arterial injection, vasospasm	Immediate	Risk of scarring; prolonged care

Sources (PubMed): Weiss RA 1990 (PMID: 2398199); Georgiev M 1990 (PMID: 2362024); Davis LT, Duffy DM 1990 (PMID: 1691217); Gillet JL et al. 2014 (PMID: 24843083); Kang M et al. 2022 Part 1 (PMID: 35503729); Mwipatayi BP et al. 2016 (PMID: 27376773); Zimmet SE 1993 (PMID: 8349902); Zimmet SE 1996 (PMID: 8556261).

Table 2. Prevention and management of cutaneous complications following sclerotherapy.

Complication	Prevention strategies	First-line management	Second-line / escalation
Ecchymosis/hematoma	Gentle technique; small needles; compression	Reassurance; compression; symptomatic care	Evaluate for coagulopathy/medications if severe
Early wheals/pruritus	Avoid overtreatment; monitor response	Observation; symptomatic care	Consider allergy evaluation if recurrent
Hyperpigmentation	Lowest effective dose; avoid high pressure; compression; consider early clot evacuation	Observation/counseling	Selected cases: clot evacuation; laser/light modalities
Telangiectatic matting	Treat feeder veins; assess reflux; staged sessions	Observation; reassess hemodynamics	Target feeder veins; vascular laser in selected cases
Thrombophlebitis/retained coagulum	Appropriate technique; compression; follow-up	Clinical assessment; symptom control	Consider clot evacuation if symptomatic/persistent
Contact dermatitis	Use hypoallergenic materials; patient history	Remove irritant; topical therapy	Dermatology referral if severe
Suspected extravasation/ischemia	Low-pressure injection; stop if pain/blanching	Immediate cessation; assess perfusion	Urgent escalation; consider hyaluronidase per protocols; wound/vascular consult
Ulceration/necrosis	Prevention as above; emergency plan	Wound care; infection prevention; close follow-up	Multidisciplinary care; surgical consult if extensive

Sources (PubMed): Rabe E et al. 2014 guideline (PMID: 23559590); Gillet JL et al. 2014 (PMID: 24843083); Kang M et al. 2022 Part 2 (PMID: 36113125); Zimmet SE 1996 (PMID: 8556261); Mwipatayi BP et al. 2016 (PMID: 27376773).

Table 3. Red flags and immediate actions. Early recognition of warning signs during or immediately after sclerotherapy is critical to limit tissue injury. The following red flags should prompt immediate interruption of the procedure and escalation of care.

Clinical warning sign	**Recommended immediate action**
Severe or disproportionate pain during injection	Stop injection immediately; assess skin perfusion and patient symptoms
Immediate skin blanching or livedoid discoloration	Stop injection; suspect ischemia; avoid further sclerosant; urgent assessment
Rapid swelling or intense burning at injection site	Suspect extravasation; stop injection; consider dilution/dispersion measures
Dusky discoloration developing within minutes to hours	Monitor closely; initiate early wound/vascular consultation
Signs suggestive of intra-arterial injection	Emergency escalation; vascular consultation; initiate institutional protocol