The lateral subdermic venous system: *the hidden origin of spider veins of the thigh*

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Spider veins of the lateral thigh are among the commonest reasons women consult a vein practice, and among the likeliest to recur after treatment. Many are fed by a network most anatomy teaching omits: the lateral subdermic venous system, a thin-walled remnant of the embryonic lateral marginal vein that drains through its own perforators to the deep veins, independently of the saphenous trunks. This review covers what the system is, why it behaves as it does, how to recognise and assess the pattern it produces, and what the evidence says about treating it. It also explains why a normal saphenous duplex scan does not exclude a feeder.

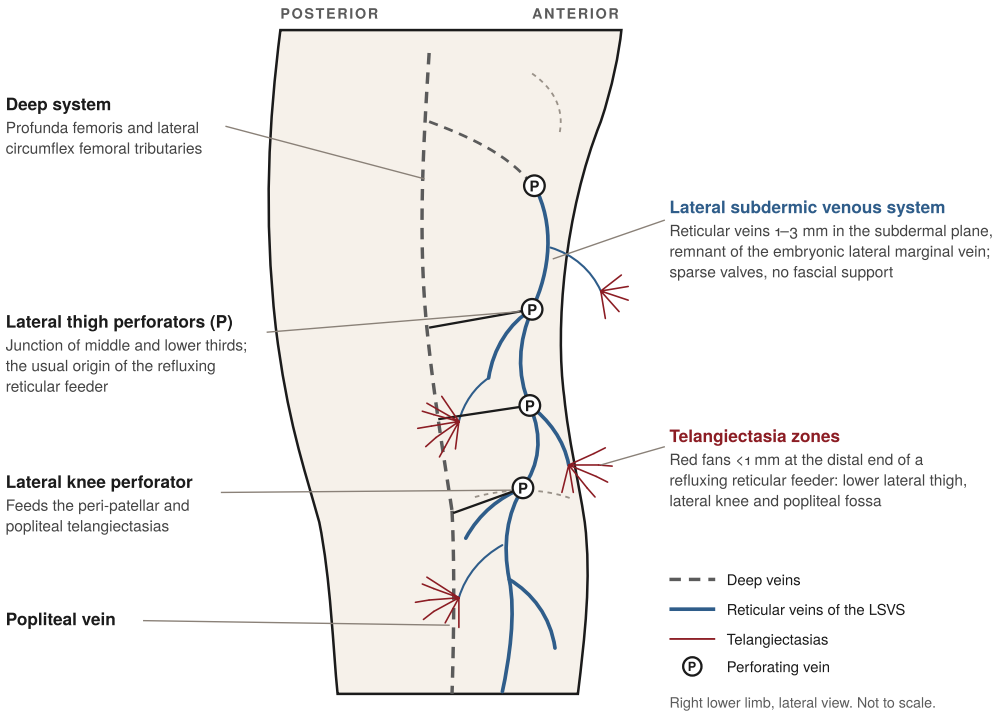


Figure 1. The lateral subdermic venous system. Right lower limb, lateral view; deep veins dashed. The reticular network of the lateral thigh continues over the lateral knee onto the upper calf, and perforators (P) connect it to the deep system at the upper thigh, the junction of the middle and lower thirds, the lower thigh and the lateral knee. Red fans mark the zones where telangiectasias typically cluster.

THE PATIENT WHOSE SPIDER VEINS KEEP COMING BACK

A COMPOSITE CASE, TYPICAL OF REFERRALS TO A VEIN PRACTICE

She is 34, a pharmacist, slim, with two children, and bothered by a cluster of fine purple and red veins on the outer side of her right thigh just above the knee. Looked at closely, the cluster curves: it starts on the posterolateral thigh, sweeps forward around the lateral knee and ends on the anterolateral thigh in an open horseshoe. In the centre of the arc, visible only when she stands in good light, is a faint blue line about 2 mm wide.

The veins appeared during her second pregnancy and did not fade. Two years ago she had 3 sessions of sclerotherapy elsewhere, each directed at the fine red vessels; the result was good for a few months, then the veins returned with a new blush of very fine vessels around the treated area. She was told the cause was hormonal and nothing further could be done. A saphenous duplex scan arranged by her general practitioner was normal.

In the vein clinic the picture differs only in what is looked for. Transillumination shows the blue reticular vein

running from the upper lateral thigh to the knee, with the telangiectatic fans arising from its distal end. On duplex the saphenous trunks are indeed competent, but a 3 mm vein on the lateral thigh refluxes on calf release and dives through the fascia lata towards the profunda femoris. The feeder was never treated; the spider veins were.

The diagnosis is telangiectasias and reticular veins of the **lateral subdermic venous system**, with telangiectatic matting after sclerotherapy directed at the wrong vessel. Her prognosis after correct treatment is good. Her story, with minor variations, is one every vein practitioner has heard, and the reason this review was written.

WHAT IS THE LATERAL SUBDERMIC VENOUS SYSTEM?

The term comes from the Argentinian surgeon Alfonso Albanese, who in 1969 described a "lateral subdermic varicose vein system of the legs" and its surgical treatment (the paper is often cited to *Vasa*; it appeared in *Vascular Surgery*).[1] Later authors credit Albanese with describing its multiple perforator connections to the deep veins of the thigh and its ability to become varicose without saphenous insufficiency.[2]

The 2002 international consensus on venous nomenclature adopted the name **lateral venous system** (*systema venosum laterale membri inferioris*), extending "on the lateral thigh and leg" and representing "the remnant of the embryonic vena marginalis lateralis", citing Albanese.[3] The UIP duplex consensus repeats the definition and adds the eponym "Albanese system",[4] and Oğuzkurt lists "lateral subdermic (venous) system" as a formally obsolete synonym, noting that an abnormally developed lateral venous system is seen in Klippel–Trénaunay syndrome.[5] This review uses "lateral subdermic venous system" because practitioners still do.

Anatomically the system is an epifascial, subdermal network of reticular veins of roughly 1 to 3 mm on the lateral thigh and knee, continuing onto the upper calf. Simka and colleagues describe its calf component draining upward and its thigh component downward, with blood leaving through perforators at the lateral knee.[6] The 2002 consensus records that the anterior and posterior thigh circumflex veins may originate from it, its connection to the saphenous territories, and that lateral leg perforators connect it to the fibular veins;[3] the UIP document names lateral thigh, posterolateral thigh (former Hach) and lateral knee perforators.[4]

The perforators give the system its clinical character. Labropoulos and colleagues, examining 2820 limbs with chronic venous disease, found incompetent posterolateral thigh perforators in 24 (0.85%); all pierced the fascia lata 12 to 25 cm above the popliteal crease and joined tributaries of the deep femoral vein, the femoral vein or both, and the authors assigned them to "the lateral thigh venous system, which is undeveloped in most people". Isolated reflux in them never exceeded C3.[7] In the same group's series of non-saphenous reflux, 36 of 42 refluxing thigh tributaries connected to perforators joining the deep femoral, femoral or muscular veins.[8] The system has its own outflow to the deep veins; when a perforator becomes incompetent, the network above it fills under pressure while the saphenous trunks remain normal.

One source of confusion should be cleared away. The "lateral accessory saphenous vein" of older surgical literature is an obsolete name for the anterior thigh circumflex vein, a saphenofemoral tributary and a well-known cause of groin recurrence.[5] It is not the lateral subdermic venous system of the mid and lower thigh, although the two may communicate.

NOT TO BE CONFUSED WITH: THE PERSISTENT LATERAL MARGINAL VEIN OF KLIPPEL–TRÉNAUNAY SYNDROME

The lateral subdermic venous system and the persistent lateral marginal vein (the "vein of Servelle") share an embryonic origin and nothing else, and conflating them is dangerous. The persistent lateral marginal vein is a single large, valveless embryonic trunk running from the foot or lateral leg up the lateral thigh. Lee describes it as a failure of the second embryonic transition: the fibular vein persists as the marginal vein, which "is always valveless and can cause severe reflux, chronic venous hypertension/stasis" and thrombo-embolic events, and which in Klippel–Trénaunay syndrome may be the only major drainage of a limb with underdeveloped deep veins.[9] Oduber and colleagues found one in 12 of 70 patients with the syndrome (17.1%).[10] The lateral subdermic venous system, by contrast, is a network of 1 to 3 mm reticular veins appearing in adolescence or adult life in an otherwise normal limb.

The rule: a large lateral vein in a limb with a capillary malformation, limb-length discrepancy or soft-tissue overgrowth is a persistent marginal vein until proven otherwise, and requires full assessment of the deep venous system before any sclerotherapy is contemplated, because it may be the limb's main outflow. The practice's approach is set out in the Klippel–Trénaunay syndrome review.

EMBRYOLOGY: WHY THE REMNANT BEHAVES AS IT DOES

Lower-limb venous development is usually described in three stages, adapted from Lewis's observations in rabbit embryos. In the first (about weeks 5 to 6) the limb bud is vascularised by the primitive fibular vein, which gives a distal extension, the primitive lateral marginal vein along the caudal border of the bud, and a proximal extension, the primitive sciatic vein; every vein is valveless at this stage. In the second (weeks 7 to 8) the fibular vein becomes the small saphenous vein, and in the third, complete by about week 12, the femoral vein becomes the main outflow and the primitive posterior tibial vein becomes the great saphenous vein, carried medially by limb rotation.[6,9,11] The lateral marginal vein regresses once the fibu-

lar vein becomes dominant; in the adult a thin lateral marginal network remains, connected to the fibular veins by fibular perforators,[11] as the 2002 consensus describes.[3]

Three features of this history explain the system's behaviour. It is a **regression remnant**, not a purpose-built conduit, and its walls lack the muscular media of the saphenous trunks. It belongs to the stage in which all limb veins were **valveless**; whatever valves it acquires are few and poorly supported. And it lies **subdermally and epifascially**, outside the saphenous compartment, with no fascial envelope to limit dilatation. With direct perforator connections to a high-pressure deep system, it is an obvious candidate for early dilatation. It should be said plainly that the remnant hypothesis rests on homology of position, stated as "represents" or "probably represents" in every nomenclature document; no fetal dissection study tracing the thigh component was identified.[3,4,6]

HOW COMMON ARE SPIDER VEINS, AND IN WHOM?

Telangiectasias of the legs are so common that the Edinburgh Vein Study investigators suggested they "may be considered normal". In that random sample of 1566 adults aged 18 to 64 years, telangiectasias were present on the right leg in 84%: 79% of men and 88% of women, 92% grade 1. Fewer than 1% of people with grade 2 to 3 trunk varices were free of telangiectasia, yet 51% of those with grade 2 to 3 telangiectasia had no clinical varicose veins, and telangiectasia alone did not cause symptoms.[12] In the baseline paper the age-adjusted sex difference for grade 1 hyphenweb varices was not significant, and trunk varices were commoner in men (Table 1).[13] The Bonn Vein Study of 3072 adults found C1 disease in 59.1%: 58.4% of men and 59.5% of women.[14,15] The San Diego Population Study of 2211 ethnically diverse adults reported higher odds of spider veins in women (odds ratio 5.4) and of varicose veins (2.2), but lower odds of trophic changes and deep functional disease.[16] The Vein Consult Program, a survey of 91 545 general-practice attendees in more than 20 countries, found C1 to C3 disease commoner in women in every country and no sex difference in C4 to C6.[17]

Table 1. Prevalence of telangiectasias and reticular veins (CEAP C1) by sex in population studies

Study	Population	Men	Women	Note
Edinburgh Vein Study, 2008[12]	1566 adults, 18–64 yr, random sample	79%	88%	Any telangiectasia, right leg; 92% grade 1
Edinburgh Vein Study, 1999[13]	Same cohort, age-adjusted	79.2% (grade 1); 5.9% (grade 2)	84.4% (grade 1); 9.2% (grade 2)	Hyphenweb varices; only the grade 2 difference significant
Bonn Vein Study[14,15]	3072 adults, 18–79 yr	58.4%	59.5%	CEAP C1; 59.1% overall
San Diego Population Study[16]	2211 adults, multi-ethnic	Women vs men: odds ratio 5.4 for spider veins		Sex-specific percentages not given in the abstract
Vein Consult Program[17]	91 545 general-practice attendees, 20+ countries	C1–C3 more common in women in every country		Any chronic venous disorder in 83.6%

Two conclusions follow, and the first is often missed. **Having telangiectasias is not a female trait.** In Edinburgh four in five men had them; in Bonn the sex difference in C1 prevalence was one percentage point. The female excess is real but concentrated in the more extensive, higher-grade lesions and, as San Diego shows, in the spider pattern specifically. What is strongly female is **presentation and treatment-seeking**: in the Bonn sample, 31.0% of women had previously had treatment of some kind for their veins, as compared with 12.7% of men.[15] A vein practice therefore sees a sample weighted towards women, and should not infer from its waiting room that the anatomy is sex-specific.

The second conclusion concerns site. Edinburgh found the commonest sites of telangiectasia to be the postero-medial thigh, popliteal fossa and upper third of the calf, not the lateral thigh.[12] The lateral thigh is therefore not "the commonest site" for spider veins, whatever practice-based texts imply; it is a **distinct, recognisable subgroup** with its own anatomy and feeder, and the one most likely to be treated badly when the feeder is ignored. Simka's statement that the lateral plexus "is often the first site where varicose veins develop, especially in women" is a clinical observation, not a population finding.[6]

Three further features matter to the woman in the consulting room. The **natural history** is slow deterioration: at the 13-year Edinburgh follow-up, telangiectasia worsened at 1.6% per year, most often from grade 1 to grade 2, predicted by female sex (odds ratio 1.87), age over 55 years (1.68), family history (1.60) and superficial reflux.[18] **Heredity** is strong: in Cornu-Thenard's 134 families, as quoted by Boisseau, the risk of varicose veins was 90% when both parents were affected and, with one affected parent, 25% for sons and 62% for daughters,[19] and in a German cohort of 4033 nuclear families the heritability of chronic venous disease (C2 and above) was 17.3%.[20] The **hormonal relationship** has its own section below.

PATHOPHYSIOLOGY: THE FEEDER-VEIN CONCEPT

The single most useful idea in the management of leg telangiectasias is that most are not primary lesions but the visible end of a pressure gradient that begins in a larger vein. The evidence comes largely from Doppler and duplex studies of the early 1990s, much of which reaches the modern reader through Goldman's textbook; it is cited alongside the primary papers here because their abstracts could not be checked for this review.

Weiss and Weiss examined 700 patients with Doppler and, as summarised by Goldman, found audible reflux in 88% of those whose telangiectasias were linked to a feeding reticular vein of the lateral subdermic venous system; treating the reticular veins first improved efficacy and reduced hyperpigmentation.[21,22] Somjen found closely situated incompetent reticular veins in 89% of telangiectasias on duplex.[21,23] Raymond-Martimbeau and Dupuis, in 884 telangiectasia zones in 525 patients, found 71.2% connected directly to reticular veins, 12.6% to the superficial system and 8.8% to the deep system.[21,24] Mariani reported that when the point of reflux was treated first, 95% of 109 patients had enduring resolution at 3 years.[21,25]

How often is the saphenous system involved? Thibault and colleagues found superficial incompetence in 23% of spider-vein patients without visible varicosities, and in 37% of clinically "normal" legs of patients with one abnormal leg.[21,26] Engelhorn and colleagues found saphenous reflux, mostly great saphenous, in 46% of 269 limbs of women with telangiectasias.[27] Kern's systematic review puts saphenous reflux in C1 patients at 28 to 46%,[2,28] so in more than half the saphenous trunks are competent and the feeder, if there is one, lies elsewhere.

Kern adds a mechanism directly relevant to the lateral thigh. Most telangiectasias arise within a territory of reflux, but in some clusters the feeding reticular vein lies *distal* to the telangiectasias, against the gravitational gradient, so that reflux cannot explain them. Kern calls this the "maritime pine" (*pin maritime*) pattern and attributes it to dynamic systolic venous hypertension transmitted upward through incompetent distal perforators during calf contraction, "and not by reflux".[2,28] The lateral knee, where the system's perforators are concentrated, is exactly where such a pattern would be expected, and clusters fanning upward from the knee are a familiar sight.

Perforators matter in treatment failure: Schuller-Petrović and colleagues identified a feeder vein linked to a perforating vessel in 14 of 26 patients whose telangiectasias had resisted sclerotherapy.[29] Labropoulos's 2001 series defines the broader population of non-saphenous reflux: present in 84 of 835 limbs (10%); 67 of 72 patients (93%) women; thigh tributaries 50% of limbs, pelvic sources 34%, sciatic nerve veins 10% and popliteal fossa veins 8%; and more pregnancies on average (3.2) than in women with saphenous reflux (2.2).[8]

Put together, the evidence supports a simple working model. An incompetent lateral thigh or knee perforator transmits deep venous pressure into a reticular vein of the lateral subdermic venous system, which dilates and refluxes along the thigh; at its distal end, where it curves around the lateral knee, the pressure reaches the subpapillary venular plexus and telangiectasias form in fans and arcs. The saphenous trunks need not be involved at all, which is why the general practitioner's duplex report in the composite case was both correct and irrelevant.

WHY WOMEN?

It is tempting to answer "hormones" and move on, but some of the evidence points the other way. Venous tissue does express sex-hormone receptors: Perrot-Applanat and colleagues found progesterone receptor in about 90% of varicose saphenous vein samples from 5 men and 25 women, with oestrogen receptor absent or barely detectable in 25 of 30, and concluded that saphenous veins of both sexes are direct targets of progesterone;[30] Serra and colleagues found oestrogen receptor expression higher in the veins of 40 patients with chronic venous disease than in controls, rising with severity.[31]

Here is the paradox. When Sadick and Niedt biopsied leg telangiectasias themselves, from patients with high-oestrogen and high-progesterone risk factors, they found no oestrogen or progesterone receptor in any patient,[32] and a second study by the same group on post-sclerotherapy matting was also negative. Assay insensitivity or an indirect action of oestrogen have been offered as explanations; Kern concludes that "the role of oestrogen and progestogen is unclear" for telangiectasias.[2,21] That is the defensible position: the receptor data support a hormonal effect on the larger veins, including the feeders, and are silent or negative on the telangiectasias themselves.

The clinical associations are nonetheless consistent. Perrot-Applanat's own rationale was that varicose disease worsens during sex-hormone therapy, in the luteal phase and in pregnancy.[30] Goldman's chapter, drawing on older surveys, states that about 70% of women develop telangiectasias during pregnancy, most resolving within weeks of delivery, and that one third of patients first noticed their veins during pregnancy and 18% on oral contraceptives.[21] The practice's page on varicose veins in pregnancy covers the mechanisms. Oestrogen is also an established risk factor for matting after sclerotherapy: in Davis and Duffy's retrospective analysis, as cited by Goldman, 29% of women who developed matting had been on systemic oestrogen or become pregnant, against 19% of those who did not; family history and obesity were the other risk factors.[21,33] Heredity contributes independently: Cornu-Thenard's daughter-versus-son figures (62% versus 25%) suggest a sex-related pattern of inheritance, and Goldman cites surveys reporting a family history of leg veins in 43 to 90% of telangiectasia patients.[19,21]

What is *not* established should also be stated. No controlled study links gynoid fat distribution to the lateral thigh pattern, although the association is often asserted; no primary data were found on puberty, hormone replace-

ment therapy or the menopause in relation to the lateral pattern; and no trial has tested whether stopping oestrogen-containing contraception before sclerotherapy reduces matting, so that advice is expert opinion.

RECOGNISING THE PATTERN, AND WHAT ELSE IT MIGHT BE

The lateral pattern has a characteristic distribution: reticular veins and telangiectasias on the lateral and anterolateral thigh, often in the lower third, continuing over the lateral knee and sometimes onto the upper calf and popliteal fossa (Figure 1). The telangiectasias are arborising or "cartwheel" fans arising from a visible or palpable blue reticular vein; Goldman notes that on the lateral thigh they often connect to "the lateral network described by Albanese".[21] In the author's experience the lower lateral thigh telangiectasias frequently form a **horseshoe**: an open arc following the distal end of the feeder as it curves around the lateral thigh and knee, with the feeder entering the arc from above (Figure 2). The shape matters because the feeder is often faint, and the arc points to where it will be found.

FIGURE 2 · PHOTOGRAPH TO BE SUPPLIED

Figure 2. The horseshoe pattern. Telangiectasias of the lower lateral thigh in an open arc around the lateral knee, with the blue reticular feeder running into the arc from the upper thigh. The spider veins were treated twice elsewhere without the feeder; the matting medial to the arc appeared after the second treatment.

Four other patterns must be distinguished.

The pelvic-origin pattern. Veins of pelvic origin reach the thigh through pudendal, inguinal, gluteal and obturator escape points and are distributed over the vulva, perineum, groin and anterior upper thigh, medial thigh, and posterior and posteromedial thigh and buttock. They occur in up to 16.5% of women with varicose veins and 25.6% of those with recurrence, are strongly parity-related, and fewer than 10% of affected women have pelvic symptoms. The key duplex clue is a competent great saphenous terminal valve with reflux beginning below the external pudendal confluence.[34] The posteromedial pattern is thus pelvic or saphenous until proven otherwise, whereas the lateral pattern is perforator-fed; distribution and duplex, not the appearance of the telangiectasias, separate them. The practice's page on vulval varicose veins describes the pelvic pattern for patients.

The medial pattern. Telangiectasias of the medial thigh and knee should raise suspicion of great saphenous reflux, and ankle clusters reflect great (medial) or small (lateral) saphenous incompetence; these are the patterns to which the Edinburgh and Engelhorn data on saphenous reflux apply most directly.[12,27]

Klippel–Trénaunay syndrome, discussed in the box above, is the one differential in which misdiagnosis can harm: a large lateral vein since childhood, a capillary malformation or limb asymmetry changes the consultation entirely.

Corona phlebectatica is a different lesion in a different place: fan-shaped intradermal veins at the ankle and foot, classified since 2020 as C4c because it marks advanced venous disease, belonging with the skin changes discussed in the lipodermatosclerosis review. The 2020 CEAP update left C1 itself unchanged as "telangiectasias or reticular veins", conventionally below 1 mm and 1 to 3 mm respectively.[35] The practice's patient-facing description is on the spider veins page; whether these veins are cosmetic or medical is addressed in a separate article.

ASSESSMENT: WHAT THE GUIDELINES SAY, AND WHAT THE PRACTICE DOES

Guidelines disagree on investigating C1 disease more than on almost anything else in venous care.

GUIDELINE POSITIONS ON ASSESSMENT BEFORE C1 TREATMENT

European guidelines for sclerotherapy (2014). History, clinical examination and duplex ultrasound before treatment (GRADE 1C), with the proviso that for C1 veins continuous-wave Doppler "may be sufficient". Treat from proximal to distal leakage points and from larger to smaller veins.[36]

ESVS (2022). For patients with reticular veins or telangiectasias, duplex ultrasound should be performed before treatment to look for associated incompetent veins (Class I, Level C). For reticular veins, sclerotherapy is the recommended first choice (Class I, Level A); transcutaneous laser for telangiectasias carries a Class IIa, Level B recommendation.[37]

SVS/AVF/AVLS Part II (2024). In asymptomatic C1 patients, duplex ultrasound should not be performed routinely, because testing could result in unnecessary saphenous ablation (consensus statement); the rationale adds that asymptomatic C1 veins should not be treated to prevent progression. In symptomatic C1 (bleeding, severe pain or burning) duplex may be performed; "saphenous ablation for C1 disease without bleeding is rarely required". The supporting text nonetheless relays Somjen's finding that incompetent reticular veins, present in 80 to 90% of cases, should be treated with the telangiectasias.[38]

The divergence is real: ESVS recommends duplex for every C1 patient being treated, while the North American guideline discourages routine duplex in asymptomatic C1 for fear of overtreating the saphenous trunks. Both agree that the **reticular feeder must be found and treated**; the

North American concern is about what a scan might lead to, not whether feeders exist.

The practice's rule is that **every patient has a duplex scan before any treatment**, including patients with spider veins alone: 28 to 46% of C1 patients have saphenous reflux that would otherwise be missed,[28] the lateral perforator can often be imaged, and the pelvic-origin pattern cannot be distinguished without a scan. The guard against overtreatment is clinical judgement, not the absence of information: a refluxing saphenous trunk found incidentally in an asymptomatic woman with a lateral thigh cluster does not need ablation, it needs to be noted, discussed and followed.

Duplex has limits at the small end: lateral knee perforators are small and difficult to detect,[4] and 1 to 3 mm reticular feeders are at the edge of what a standard probe resolves. Bedside tools fill the gap.

Transillumination. A side-lit transilluminator such as the Veinlite, pressed against the skin in a dimmed room, shows the reticular veins and their connections to the spider clusters as dark lines on a glowing background (Figure 3); Goldman records that it shows "exactly the same pattern" as Doppler.[21] In Moreira's phleboscopy series, reticular veins draining complex telangiectasias, invisible on inspection, were found in 55 of 171 limbs (32.2%), and such veins left in place "can account for poor cosmetic results".[39]

FIGURE 3 · PHOTOGRAPH TO BE SUPPLIED

Figure 3. Transillumination of a reticular feeder. Veinlite side-transillumination of the lateral thigh: the feeder, barely visible to the naked eye, appears as a dark line running into the telangiectatic cluster. The entry point for feeder sclerotherapy is marked.

Near-infrared vein viewers project an image of the subdermal veins onto the skin but have no comparative accuracy data against duplex for feeder identification, and handheld continuous-wave Doppler confirms that a visible reticular vein refluxes but cannot image it; the European guideline's proviso that Doppler "may be sufficient" for C1 reflects its historical place rather than current practice.[36]

TREATMENT

The feeder-first principle

Every source that addresses the question gives the same instruction: treat the reticular feeder before the telangiectasias. In Goldman's "arm and fingers" analogy, inject the arm (the feeder) first and the fingers (the spider veins) often fade without direct treatment; the larger feeder is easier to cannulate, and pigmentation, matting and recurrence are at least theoretically reduced.[21] The supporting data are Weiss and Weiss's and Mariani's results above, second-hand as they are,[21,22,25] and the European guideline formalises the sequence as proximal to distal and larger to smaller.[36] No randomised trial has compared feeder-first with telangiectasia-only treatment,[40] so the principle rests on haemodynamic logic and consistent observational experience.

Agents and concentrations

The European guidelines for sclerotherapy give the following concentrations for the two agents licensed in most countries.[36]

Table 2. Sclerosant concentrations for C1 veins in the European guidelines for sclerotherapy (2014)

Target	Polidocanol, liquid	Sodium tetradecyl sulphate, liquid	Polidocanol, foam	Sodium tetradecyl sulphate, foam
Telangiectasias	0.25–0.5%	0.1–0.2%	Up to 0.5% (GRADE 1B)	Up to 0.25% (GRADE 2C)
Reticular veins	0.5–1%	Up to 0.5%	Up to 0.5% (GRADE 2C)	Up to 0.5% (GRADE 2C)

Volume per injection point: telangiectasias up to 0.2 mL; reticular veins up to 0.5 mL. Maximum polidocanol 2 mg/kg per day; maximum sodium tetradecyl sulphate 4 mL of 3% or 10 mL of lower concentrations per session; foam not more than 10 mL per session in routine use.[36]

The head-to-head trials are small and consistent: the licensed agents work about equally well and differ mainly in pain and side effects. In the EASI study of 316 patients, 26-week satisfaction was 88% with polidocanol, 63% with sodium tetradecyl sulphate 1% and 11% with isotonic saline.[41] The Cochrane review of 35 randomised trials and 3632 participants pooled these and others: any sclerosant beat placebo but produced more hyperpigmentation (risk ratio 11.88) and matting (4.06); polidocanol matched other agents in resolution, pigmentation and matting and was less painful; sodium tetradecyl sulphate produced more hyperpigmentation (risk ratio 1.71) and matting (2.10); hypertonic saline produced less pigmentation but far more pain. No trial reported recurrence.[40]

Liquid or foam?

For C1 veins the European guideline makes liquid sclerotherapy the method of choice (GRADE 1A) and foam an alternative (2B), with comparable results if a low concentration of a more liquid foam is used.[36] Uncu randomised 100 women to foam or liquid polidocanol and found complete disappearance in 84% and 72%, a non-significant difference.[42] Kern found more matting and microthrombi with polidocanol 0.25% foam than liquid,[43] and Cochrane's pooling of two studies found no difference in resolution but possibly more matting with foam (risk ratio 6.12, wide confidence interval, low certainty).[40] Goldman

reserves foam for reticular and varicose veins and avoids it below 1 mm.[21] The practical synthesis, and the practice's approach, is foam for the reticular feeder and liquid for the telangiectasias; the patient pages on sclerotherapy for spider veins and foam sclerotherapy describe both.

Compression after C1 sclerotherapy

The evidence is divided. Kern's randomised trial of 100 women found that 23 to 32 mm Hg stockings for 3 weeks after a single liquid session gave significantly better vessel disappearance ($p \approx 0.026$),[44] and in Weiss's trial of 40 patients 3 weeks of 20 to 30 mm Hg compression outperformed 1 week, 3 days and none.[45] Against these, Bayer found that 1 week of 18 to 20 mm Hg stockings made no difference in 50 patients,[46] and the Cochrane pooled analysis of two studies was null,[40] partly because its outcome definition differed from Kern's. The European guideline says compression "may be applied" (GRADE 2C) and that 3 weeks of 23 to 32 mm Hg stockings after telangiectasia treatment improve results (2B).[36] A reasonable reading is that compression probably helps a little, that 3 weeks beats 3 days, and that for small isolated clusters the benefit may not justify the inconvenience.

Transcutaneous laser

Laser treats leg telangiectasias less well than facial ones, for anatomical reasons: leg vessels are deeper, with thicker adventitia and basement membrane, span a wider range of size, depth and flow, and sit under a higher hydrostatic pressure that may reduce photothermal coagulation.[47] Melanin competition reduces efficacy in darker skin. Most relevant here, laser cannot treat the feeder: it addresses the fingers and leaves the arm, which is why Parlar's group notes that sclerotherapy "can also treat feeder veins".[48]

The comparative trials show comparable clearance. In Parlar's split-leg trial of 56 women, polidocanol 0.5% foam and long-pulsed Nd:YAG gave the same blinded photographic clearance ($p = 0.84$); sclerotherapy worked faster and laser was more painful ($p = 0.003$).[48] Ianosi's split-leg trial of 285 women found Nd:YAG superior to polidocanol 0.5% below 1 mm (95% versus 53% good or very good) but not above 1 mm, with burns in 14 legs; patients with superficial reflux, which is to say the feeders, were excluded.[49] Cochrane pooled five trials: no difference in resolution or matting, less hyperpigmentation with laser (risk ratio 0.57), inconsistent findings on pain.[40] The one setting in which laser clearly adds value is in combination: Moreno-Moraga randomised 517 legs to polidocanol microfoam with or without Nd:YAG and reported 3-year clearance of 89 to 95% with the combination against 15 to 18% with microfoam alone,[50] figures not reproduced elsewhere.

Laser retains a role for sclerosant allergy, needle phobia, failed sclerotherapy and sub-millimetre matting vessels too fine to cannulate (North American guideline, Grade 2B),[38] and the ESVS guideline gives it a Class IIa recommendation for telangiectasias.[37] The author's position, and the practice's, is not to offer transcutaneous laser for leg spider veins: for the lateral thigh pattern the target is the feeder, which laser cannot reach, and for the telangiectasias themselves sclerotherapy is as effective in the pooled evidence, less painful and cheaper. Practitioners who offer laser for sub-millimetre vessels once the feeder is closed are acting within the evidence. Phlebectomy has no place in C1 disease, and the evidence for radiofrequency thermocoagulation of fine vessels is anecdotal; Cochrane found no randomised trial of either.[40]

Complications

Table 3. Complications of sclerotherapy for telangiectasias and reticular veins

Complication	Frequency	Course and comment
Hyperpigmentation	10–30% transient; permanent in about 1–2%, up to 10% in some series[21,51]	Appears at 4–6 weeks; about 70% resolves by 6 months and most by 1 year. Risk factors: concentration, vessel diameter 0.6–1.2 mm, dark skin, untreated proximal reflux, below-knee site, high ferritin.[21,52] Persistence beyond a year should prompt a search for an untreated feeder
Telangiectatic matting	About 15–20%[51]	Fine red vessels below 0.2 mm appearing 3–6 weeks after treatment; most resolves over 3–12 months. Patient risk factors: oestrogen, pregnancy, obesity, family history; procedure risk factors: untreated feeder, fast injection, large volume, high concentration. High-risk sites: anterior and anterolateral thigh, medial knee[21,33,52]
Cutaneous necrosis and ulceration	Rare (0.01–<0.1%) with liquid; very rare with foam[36]	Extravasation, excessive concentration or inadvertent arteriolar injection
Visual disturbance	Very rare with liquid; uncommon (0.1–<1%) with foam[36]	Transient, migraine-aura-like; three cases after polidocanol 0.25% foam in Kern's trial[43]
Deep-vein thrombosis	Distal: rare (liquid), uncommon (foam); proximal: very rare[36]	Encourage walking after treatment
Allergy and anaphylaxis	Isolated cases[36]	Resuscitation equipment should be available wherever sclerotherapy is performed
Intra-arterial injection	Extremely rare[36]	Identify arteries before ultrasound-guided puncture; if suspected, catheter-directed treatment and early systemic steroids

Two points in the table bear on the lateral thigh: the anterolateral thigh is a high-risk site for matting, and an untreated feeder is a procedure-related risk factor for both matting and persistent pigmentation. The composite patient's matting and recurrence were predictable conse-

quences of treating the telangiectasias without their source.

A TREATMENT SEQUENCE FOR THE LATERAL THIGH PATTERN

1. **Duplex the whole limb.** Record saphenous competence, pelvic-origin reflux, and lateral thigh and knee perforators where seen.
2. **Map the feeder.** With the patient standing in a dimmed room, trace the reticular vein by transillumination to its proximal origin and mark it.
3. **Treat the feeder first** with foam sclerotherapy, injected at its most accessible point and followed visually along its length.
4. **Wait.** Review at 4 to 6 weeks; many telangiectasias fade once the pressure source is removed. Evacuate coagula.
5. **Treat residual telangiectasias with liquid microsclerotherapy**, usually 2 to 4 sessions about a month apart.
6. **Compression** when extensive veins have been treated; optional for small clusters.
7. **Judge the final result at 3 to 6 months**, not before, with photographs at baseline and each review.
8. **If matting or pigmentation persists beyond a year**, rescan for the feeder that was missed.

IN THE AUTHOR'S PRACTICE

In the author's practice every patient, including those with spider veins alone, has a full lower-limb venous duplex scan before any treatment is planned. Feeders are mapped with Veinlite transillumination in a dimmed room with the patient standing. Reticular feeders of the lateral subdermic venous system are treated with sodium tetradecyl sulphate foam at 0.5 to 1.5%, and the telangiectasias with liquid microsclerotherapy using sodium tetradecyl sulphate at 0.3 to 0.5%, usually in 2 to 4 sessions about a month apart. Compression is used when extensive veins have been treated and is otherwise not insisted on; transcutaneous laser is not offered for leg spider veins. The final result is judged at 3 to 6 months, and patients are told this at the first consultation. The author's observation that the lower lateral thigh cluster frequently forms a horseshoe following the distal curve of the feeder is, as far as he is aware, undescribed in the literature, and is offered as clinical observation rather than audited data.

NATURAL HISTORY, RECURRENCE AND EXPECTATIONS

Telangiectasias progress slowly: in the Edinburgh cohort deterioration ran at 1.6% per year over 13 years, almost all from grade 1 to grade 2, and superficial reflux predicted it (odds ratios 1.89 to 2.34 by segment, 3.97 with more than three refluxing segments).[18] None of the Cochrane trials reported recurrence or time to resolution,[40] so the long-term figures are observational; Mariani's 95% 3-year durability after feeder-first treatment is the most encouraging and the most in need of independent confirmation.[21,25] Clinical experience, including the author's, is that treated clusters stay clear once the feeder is closed, and that "recurrence" is more often new telangiectasias from a new or untreated feeder than reopening of treated vessels.

Patients should therefore be told that treatment is a course, not a session; that the result is judged at 3 to 6 months; that pigmentation and matting are common, usually temporary, and more likely with oestrogen, pregnancy, obesity or a family history; and that the condition is one of maintenance, since the predisposition that produced the first cluster will probably produce others over the decades, particularly with further pregnancies, so that an occasional maintenance session is part of the natural history, not a failure of treatment. Pregnancy is a relative contraindication to sclerotherapy and breastfeeding should be interrupted for 2 to 3 days after a session;[36] it is usually sensible to defer treatment until some months after delivery, when pregnancy-related telangiectasias have had the chance to regress.

THE SOUTH AFRICAN CONTEXT

No South African guideline, consensus statement or society position on the treatment of telangiectasias and reticular veins was found; practice here follows the European and North American documents cited above.

Funding is the more immediate issue. Medical schemes generally regard C1 sclerotherapy as cosmetic: Polmed's general exclusions list "minor superficial varicose veins treatment with sclerotherapy" among excluded cosmetic procedures where no pathological necessity is proven, and Medihelp's MedVital exclusion lists name sclerotherapy and cosmetic procedures. Neither mentions spider veins by name, rules vary by scheme and plan, and patients should check their benefits. The practice's position is that C1 treatment is self-funded unless a scheme indicates otherwise, and patients are told so before the first session (see the fees and medical aid page); the preceding duplex scan is a diagnostic investigation, considered separately. The distinction matters clinically as well as financially: when a C1 patient's scan shows symptomatic saphenous reflux, the saphenous disease is a medical condition in its own right while the telangiectasias remain cosmetic, and keeping the two conversations separate avoids both undertreating the saphenous disease and appearing to use it to fund the cosmetic work.

AREAS OF UNCERTAINTY

- **Is the lateral subdermic venous system really the remnant of the lateral marginal vein?** Every nomenclature document says so, by homology of position; no fetal dissection study was identified.
- **How common is the lateral pattern?** No population data on site exist beyond Edinburgh's three commonest sites, none of them the lateral thigh.
- **Does feeder-first treatment improve outcomes?** No randomised trial has tested it; the evidence is observa-

tional and partly second-hand.

- **Should every C1 patient have a duplex scan?** ESVS says yes; the North American guideline says not routinely if asymptomatic.
- **What is the role of hormones in the telangiectasias themselves?** The vessels were receptor-negative in both studies that looked; the associations may act on the feeders.
- **Does compression after C1 sclerotherapy help?** Two trials say yes, one no, and the pooled analysis is null.
- **What are the recurrence rates?** No randomised trial has reported them.
- **Is the horseshoe pattern a reproducible sign?** It is one practitioner's observation, not yet validated elsewhere.

KEY CLINICAL POINTS

1. **The lateral subdermic venous system is a real and distinct structure**: an epifascial reticular network of the lateral thigh and knee with its own perforators to the deep veins.
2. **It can reflux with normal saphenous trunks**; a normal saphenous duplex report does not exclude a feeder.
3. **Telangiectasias are near-universal in both sexes.** What is female is the extent, presentation and treatment-seeking, not the anatomy.
4. **The lateral thigh is not the commonest site for spider veins** but a recognisable subgroup with its own feeder, and the one most often treated badly.
5. **Most leg telangiectasias have a feeding reticular vein** (70 to 90% in the duplex series); 28 to 46% of C1 patients also have saphenous reflux.
6. **Find the feeder before injecting anything**: transillumination shows the reticular-to-spider connection; duplex shows the perforator and excludes saphenous and pelvic sources.
7. **Treat the feeder first, with foam; the telangiectasias second, with liquid.** Recurrence and matting after telangiectasia-only treatment usually mean an untreated feeder.
8. **Laser cannot treat the feeder.** Its clearance is comparable to sclerotherapy, with more pain and cost; its role is for sclerosant allergy, needle phobia and sub-millimetre matting.
9. **A large lateral vein in a limb with a capillary malformation or overgrowth is a persistent marginal vein**, not a lateral subdermic feeder, and needs deep-system assessment before any sclerotherapy.
10. **Set expectations**: 2 to 4 sessions, result at 3 to 6 months, pigmentation and matting common and usually transient, maintenance rather than cure.

A BEDSIDE RULE

Spider veins on the lateral thigh + a normal saphenous scan + recurrence after previous sclerotherapy = look for the lateral reticular feeder, and treat it first.

Figures. Figure 1 was drawn for this article. Figures 2 and 3 are clinical photographs from Vein Centres South Africa, published with the patients' written consent and with identifying features removed.

Disclosure. The author performs sclerotherapy and other venous procedures in private practice and has no financial relationship with any manufacturer of sclerosants, transillumination devices or lasers. He acts as an unpaid proctor for Medtronic in endovenous radiofrequency ablation; no fees or expenses are received. This article is for medical education and does not replace individual clinical assessment.

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