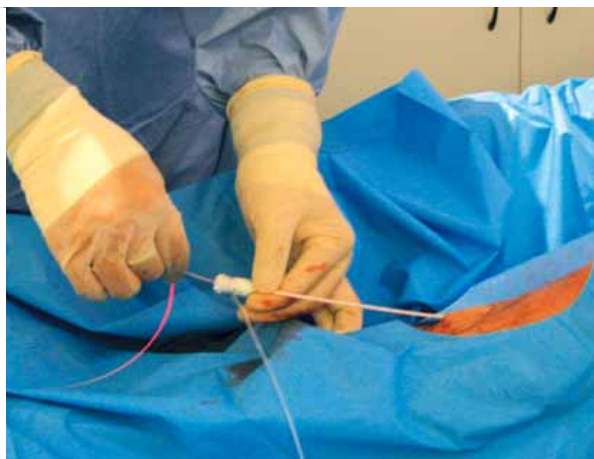


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Superficial venous disorders of the lower limb

Background

VARICOSE veins are the most common superficial venous disorder of the lower limb. Venous malformations can also affect the lower limb, and a number of key syndromes are associated with venous malformation and/or varicose veins (table 1, see next page).

Procedural treatment options for these superficial venous disorders

include surgery, sclerotherapy and laser. This article also provides an update on therapeutic advances in the areas of ultrasound-guided sclerotherapy and endovenous laser ablation of superficial veins.

Venous anatomy

The superficial venous system is a network of veins found within the

subcutaneous layer of the skin. This system drains into a network of deep veins located within the muscular or deep compartments.

The great saphenous vein (medial ankle to groin) and the small saphenous vein (lateral ankle to popliteal fossa) form the major superficial venous systems of the lower limb (figure 1, next page). These veins,

along with their tributaries, form a pattern or network that is subtly different in every individual.

Varicose veins typically arise from the saphenous systems and its tributaries. The superficial venous system can also be secondarily compromised by pressure reflux arising from incompetent deep veins after DVT.

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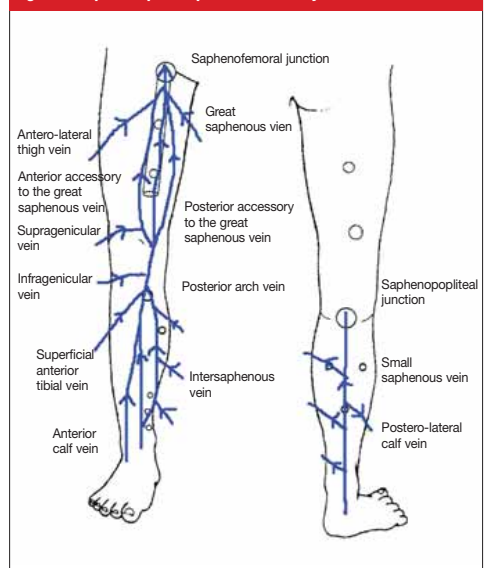
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Table 1: Summary of superficial venous disorders of the lower limb

Varicose veins	Venous malformations	Syndromes (rare)
<ul style="list-style-type: none"> • Common (more so in women than men) • Dilated and tortuous leg veins • Associated with venous congestion and reflux • Duplex studies required • Can lead to skin and vein damage • Compression relieves symptoms • Treat with ultrasound-guided therapy, endovenous ablation or surgery 	<p>Classic venous malformations</p> <ul style="list-style-type: none"> • Most common vascular anomaly • Clinical diagnosis possible • Slow-flow lesion • Present at birth • Can extend into bone and muscles • Responds well to sclerotherapy <p>Arteriovenous malformations</p> <ul style="list-style-type: none"> • Aberrant connection between arterial and venous system • High-flow lesion • Skin feels warm with thrill or bruit • Greater risk of pain and ulceration • Less responsive to treatment <p>Glomovenous malformations</p> <ul style="list-style-type: none"> • Resembles venous malformation • Diagnosis confirmed by biopsy demonstrating glomus cells • May have widespread individual glomangioma lesions • May have autosomal dominant inheritance 	<p>Blue rubber bleb</p> <ul style="list-style-type: none"> • Multiple venous malformation lesions • Blue-purple nodules on skin and gastrointestinal tract • Bleeding complications with GI lesions • Autosomal dominant inheritance • Vascular lesions respond to laser <p>Maffucci</p> <ul style="list-style-type: none"> • Triad of enchondromas, bone deformities, and venous malformations • Prone to fractures • 30% develop chondrosarcomas <p>Klippel-Trenaunay</p> <ul style="list-style-type: none"> • Triad of capillary malformation, varicose veins and limb hypertrophy • Secondary joint and gait problems (unequal limb length) • May have associated deep vein hypoplasia • Varicose veins responds to ultrasound-guided sclerotherapy • Capillary malformation responds to vascular (pulsed dye) laser

Figure 1: Duplex map — superficial venous system of the lower limb.



Varicose veins and chronic venous insufficiency

LOWER-limb varicose veins affect up to 30% of adults and are associated with superficial venous congestion. The term venous congestion is interchangeable with venous incompetence, venous hypertension and venous insufficiency, all indicating elevated intravenous pressure. A subset of superficial venous insufficiency occurs secondary to deep venous insufficiency after DVT.

The prevalence of varicose veins is greater in populations with a more sedentary lifestyle. It is more common in women and may present as early as 10-12 years of age. The severity and extent of varicose veins progressively worsen with time.

Varicose veins are frequently under-diagnosed, in part due to the perception that the condition is trivial and of cosmetic concern only. However, in the extreme, severe venous disease can lead to thromboembolic complications and malignant changes (squamous cell carcinoma arising from chronic venous ulcers) that can be potentially fatal.

Assessment

Clinical features

A careful history and examination will help define the clinical problem and allow an assessment of the risk of current and potential complications.

The history should note any leg symptoms along with any aggravating and relieving factors. Varicose veins present clinically as dilated, tortuous veins (figure 2), the valves of these veins being functionally incompetent, with resultant reflux or reverse flow. The reverse flow leads to venous congestion that can cause leg heaviness, aching, burning,

Figure 2: Lower limb varicose veins.



Figure 3: Vulvar varicosities.



Table 2: Clinical features of chronic venous congestion

Symptoms	Signs	
	Vein	Skin
None	Venular flares	Dermatitis
Aching	Varicosities	Haemosiderin staining
Heaviness	Phlebitis	Oedema
Throbbing	Thrombosis	Acute lipodermatosclerosis
Burning	Haemorrhage	Chronic lipodermatosclerosis
Tightness		Cellulitis
Cramps		Poor wound healing
Itching		Atrophie blanche (pre-ulcer)
Swelling		Scarring
Tiredness		Ulceration
Dermatitis		Malignant transformation (squamous cell carcinoma)

swelling and cramps that are exacerbated by prolonged standing and relieved by rest and leg elevation (table 2).

Leg discomfort from venous insufficiency is improved by walking, compression and leg elevation. This is the opposite pattern to that for arterial insufficiency, where walking, compression and leg elevation worsen symptoms.

Enquire about family history, risk factors (eg, standing occupations, multiple pregnancies, previous DVT), history of previous vein

treatment and any relevant medications such as oral contraception that may increase the risk of post-sclerotherapy DVT.

It should be noted that lower-limb symptoms do not correlate well with the degree of venous insufficiency. Additionally, some patients may not realise the degree of discomfort they have been putting up with until after successful treatment of their varicose veins.

Chronic venous congestion can also be associated with secondary complica-

tions such as thrombophlebitis and skin ulceration that have significant impacts on patient health and their quality of life (see Complications of varicose veins and chronic venous insufficiency, page 34).

However, most patients requesting treatment are still primarily concerned about the cosmetic appearance of the veins. Patients are often surprised to learn that there may be progressive damage from chronic lower-limb venous hypertension, even in the absence of pain. A minority will seek help solely because of their concern for potential complications such as leg ulcers, based on their observation of affected relatives.

Physical examination should focus on the veins and skin of the entire lower limb (groin to foot). The arterial pulses should be palpated to exclude coexisting arterial insufficiency. If indicated on history (pain with sexual intercourse, vulvar congestion) examination of the vulva and perineum for

varicosities may also be necessary (figure 3).

Doppler and duplex studies have largely replaced the art of vein palpation and the various manoeuvres used commonly in the past to assess venous reflux. At the very least, note the location of any bulging veins and look carefully for any skin changes indicating chronic venous insufficiency (table 2).

Investigation

Duplex ultrasound studies are the most useful tool to map the venous system of the leg, and the means by which incompetent veins are identified, measured and documented. Duplex scans can visually identify venous reflux or incompetence even when the veins are not clinically obvious. In most centres this has replaced the more invasive and labour-intensive venography for routine evaluation of venous disease of the lower limb. A portable handheld audio Doppler unit is a useful screening tool for the presence of venous reflux before undertaking formal confirmatory duplex studies if necessary.

Treatment

Treatment of incompetent leg veins can enhance quality of life by improving the cosmetic appearance of the varicose veins, relieving symptoms of venous congestion and reversing complications arising from chronic venous congestion. Early intervention will reduce the risk of ongoing vein- and skin-related damage secondary to chronic venous congestion.

Common procedures for the treatment of varicose veins include:

- Surgical ligation of the

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saphenofemoral junction with vein stripping.

- Endovenous thermal ablation (using laser or radiofrequency energy).
- Ambulatory phlebectomy.
- Sclerotherapy.

Surgical saphenofemoral junction ligation and saphenectomy

Traditional saphenofemoral junction ligation and saphenectomy ('vein stripping') involves incision at the groin crease followed by ligation and division of the truncal veins and its tributaries. A vein-stripping wire is inserted through the truncal vein from the groin down towards the knee where it exits through the distal incision. The threading wire has an attachment to the entry point of the vein at the groin and will invaginate and extract the vein as the wire is passed through the vessel, which exits the vein at a second incision point at knee level.

Endovenous thermal ablation

In recent years, endovenous thermal ablation techniques have become a popular alternative to the surgical vein-stripping procedure. Endovenous thermal ablation uses either radiofrequency or laser energies delivered via a catheter inside the vein (figure 4). Radiofrequency devices appeared on the market first, but over time there has been a proliferation of various laser devices with a range of wavelengths (810nm, 980nm, 1064nm, 1320nm through to 1500nm).

Endovenous thermal ablation involves first passing either the radiofrequency catheter or fibre-optic laser wire into the incompetent vein segment (usually from the knee to the groin). Next, tumescent anaesthetic (large-volume dilute anaesthetic) is infiltrated around the target vein (within its fascial sheath), causing the tissues at this location to become swollen and firm and to collapse the vein so that it is in close contact with the device wire. The tumescent anaesthetic also forms a protective fluid buffer between the treated vein and the surrounding tissue. Finally, the device is activated to heat- ablate the treated vein segment.

Ambulatory phlebectomy

Non-truncal veins such as incompetent tributaries and tortuous varicosities can be treated surgically with ambulatory phlebectomy. Ambulatory phlebectomy removes short segments of veins through small incisions using special vein-removing hooks (stab-avulsion technique).

Tortuous veins are not suitable for endovenous ther-

Figure 4: Endovenous laser ablation (proximal great saphenous vein).



Figure 5: Ultrasound-guided sclerotherapy (foam sclerosant).



Modern-day sclerotherapy can effectively treat venous disorders ranging from bulging varicose veins to selected vascular malformations.

mal ablation methods because they will not readily permit passage of the device catheter or wire. In practice, many of these smaller tributaries can be simply and effectively treated with ultrasound-guided sclerotherapy.

Sclerotherapy

Sclerotherapy can be used to treat varicose veins effectively and safely, either as a stand-alone procedure or in combination with other modalities. Increasingly, ultrasound-guided sclerotherapy (UGS) is recognised as an acceptable first-line treatment for varicose veins (figure 5).

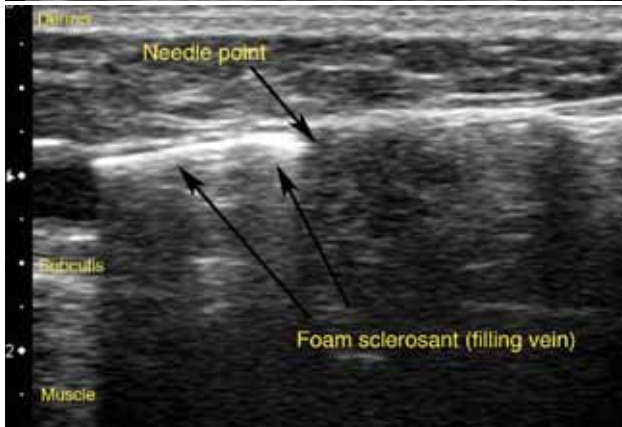
Even if surgical vein stripping or endovenous device ablation is undertaken, residual venectasias and telangiectasias can only be definitively treated with sclerotherapy. Sclerotherapy is also a reasonable alternative to repeat surgery in the event of postsurgical varicose vein recurrences.

Sclerotherapy is the non-surgical technique of injecting veins with sclerosing agents (sclerosants) to chemically ablate them. Modern-day sclerotherapy can effectively treat venous disorders ranging from cosmetic telangiectasias and bulging varicose veins to selected vascular malformations. The different types of sclerotherapy are described below.

Figure 6: Direct vision sclerotherapy (liquid sclerosant).



Figure 7: Ultrasound-guided sclerotherapy (needle insertion and foam injection).



Direct vision sclerotherapy

This procedure involves injecting visible veins with sclerosants to disrupt the endothelium that would otherwise lead to vein sclerosis or fibrosis (figure 6). The treated vein shrivels and is gradually absorbed by the body.

Commonly used sclerosants include hypertonic saline, sodium tetradecyl sulfate and polidocanol. In addition, 80-100% ethanol is used for sclerotherapy of vascular malformations (see later section). The limitation of direct vision sclerotherapy is its inability to adequately treat incompetent leg veins that are not easily visualised clinically.

Ultrasound-guided sclerotherapy

UGS relies on real-time duplex imaging to guide the needle into incompetent veins that are not clearly visible by direct vision (figures 5 and 7). Up to one-third of seemingly surface cosmetic leg veins may have 'hidden' incompetent or refluxing veins that would have been missed without the benefit of Doppler or duplex ultrasound imaging. The duplex ultrasound can precisely locate the abnormal or incompetent veins as well as adjacent structures such as deep veins and arteries, ensuring precise

localisation of the target vein and minimising accidental injection outside the vein.

Foam sclerotherapy

Certain sclerosants such as sodium tetradecyl sulfate and polidocanol are amenable to foaming, as they have detergent-like properties. Foam UGS has increased the efficacy of large-vessel sclerotherapy without compromising safety. In recent years foam UGS has become an accepted first-line treatment for primary varicose veins and is being increasingly used for treatment of vascular malformations (figure 5).

It has been demonstrated that detectable amounts of foam micro-bubbles can reach the heart, lungs and brain during leg vein treatment. There is no evidence that this has long-term detrimental effects in most patients. Transient effects such as headaches, visual disturbances and coughing may occur.

Rarely, strokes following foam sclerotherapy have been reported, presumably due to air embolism from a patent foramen ovale. To minimise excessive air embolism the total foam volume per treatment session should be closely monitored.

The leg veins are injected with sclerosants of suitable

Figure 8: Matting (red patches) and staining (brown spots and streaks).



concentration, depending on the vessel diameter. One or both lower limbs can be treated at the same time provided the sclerosants do not exceed safe recommended levels.

Liquid sclerosants are mainly used with direct vision sclerotherapy whereas foam sclerosants are routinely used with UGS. In practice, a combination of foam and liquid sclerosants are commonly used on the same patient.

What happens after sclerotherapy?

After treatment, compression (20-30mmHg) is required for 1-3 weeks, depending on the size of the treated veins. The patient is required to walk for about half an hour a day (wearing the stocking) during the treatment period. Walking serves to decrease superficial venous pressure in the legs and helps reduce post-treatment discomfort, trapped blood and DVT risk.

After sclerotherapy, telangiectasias and venectasias will clear within 2-3 months, while varicose veins may take up to six months or more to fully resolve. The two most common unwanted side effects are matting and staining (figure 8).

Matting is the development of very fine compensatory vessels over the treated vein as a result of the body's attempt to replace the vein that is being destroyed. Staining results from excessive trapped blood (iron in haemoglobin) within the treated vein that can stain the skin a brownish colour. Most cases of matting and staining are temporary and will resolve with time (3-6 months).

Mild degrees of phlebitis (during convalescence) may occur, especially when treating larger and more severe varicose veins. Rare complications include:

- DVT.
- Prolonged ankle oedema.
- Skin ulceration.
- Allergies.

DVTs are exceedingly uncommon due to the ambulatory nature of the procedure.

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Complications of varicose veins and chronic venous insufficiency

CHRONIC venous hypertension can lead to stasis changes affecting both the skin and the venous system (table 2, page 30). A common management trap is to needlessly delay treating varicose veins until the onset of complications. Even when varicose veins are asymptomatic, potential skin and vein-related complications may still occur secondary to the chronic venous hypertension invariably associated with varicose veins.

Vein-related complications include:

- Venular flares.
- Varicosities.
- Phlebitis.
- Haemorrhage.
- Thrombosis.

Skin-related complications include:

- Stasis dermatitis.
- Haemosiderin staining (iron staining from red blood cell extravasation).
- Oedema.
- Acute lipodermatosclerosis (painful inflammation of fat).
- Chronic lipodermatosclerosis (end-stage fibrosis of fat).
- Cellulitis.
- Poor wound healing.
- Ulceration.
- Scarring.
- Skin cancers arising from chronic venous ulcers.

Superficial thrombophlebitis

Chronic venous insufficiency directly contributes to the formation of varicosities by distorting and expanding the vein. The congestion and relative stasis within affected veins predispose to clots and inflammation (thrombophlebitis).

Superficial thrombophlebitis (STP) typically presents as a segment of varicose vein that has become inflamed, tender and firm (figure 9). The affected leg becomes painful and swollen. Inflammation of the overlying skin may be mistaken for cellulitis.

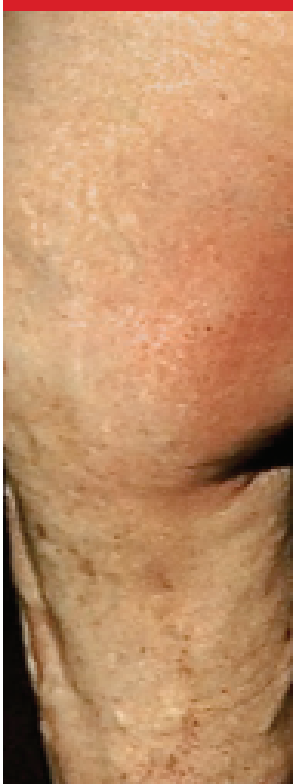
The risk of STP is increased with high-dose oral contraceptive pills but the risk with low-dose oral contraceptives has not been quantified. Individuals with thrombophilia (eg, factor V Leiden mutation, protein C and S deficiency, prothrombin gene mutation) are also at increased risk of developing STP. Other risk factors for varicose vein STP include obesity, immobility and smoking.

STP is managed by:

- Compression of the lower limb with graduated stockings.
- 'Active rest'.
- Use of NSAIDs.

A 30-40mmHg graduated compression stocking (up to the thigh) will help support the leg and has been shown

Figure 9: Superficial thrombophlebitis (thigh).



to have intrinsic fibrinolytic activity.

Rest and using a warm compress over the STP can also alleviate some discomfort. However, patients should be encouraged to maintain their mobility to limit clot progression.

Interestingly, studies show that oral NSAIDs are equivalent to low-molecular-weight heparin in DVT prevention in the setting of STP. The anti-inflammatory actions of NSAIDs reduce the release of inflammatory mediators that can lead to further clotting. They can be prescribed as ibuprofen 400mg every 4-6 hours (maximum 3.2g/day).

Below-knee STP without extension into deep veins carries little risk of thromboembolic problems and the patient can be reassured accordingly.

Although STP can be diagnosed clinically, a duplex scan is nevertheless recommended to exclude any thrombotic extension into the deep veins, especially above-knee STP. Extension of thrombosis into the deep veins constitutes DVT and should be treated according to standard DVT management protocols.

Varicose vein rupture

Apart from STP, varicose veins may rupture and bleed, either with trauma or spontaneously (even in bed), which is often alarming for the patient. Varicose vein haemorrhage typically presents in older patients in whom there is associated skin fragility from ageing and coexisting varicose-vein-related complications such as skin ulcers and erosions.

The bleeding vein should be compressed and banded to obtain haemostasis. If the bleeding does not settle

Figure 10: A: Varicose dermatitis before sclerotherapy. B: After sclerotherapy.



with compression, urgent foam USG to the affected vein is indicated.

Varicose dermatitis

This presents as itchy patches of dermatitis on the leg that are generally associated with more severe forms of venous congestion and varicose veins (figure 10A). Although topical steroids can temporarily relieve the itch, relapse invariably occurs unless the underlying venous congestion is adequately managed. The eczema can occasionally 'spread' to other areas of the body and become generalised.

Varicose dermatitis may coexist with other secondary skin manifestations such as haemosiderin staining, lipodermatosclerosis and ulcers. Varicose dermatitis often becomes excoriated through scratching, and this may serve as a portal of entry for secondary bacterial infections, which can complicate existing erosions and ulcers.

Management involves standard general measures for dermatitis such as short showers, moisturising and application of short-term, potent topical steroids. Treat any secondary infection with systemic antibiotics. A compression stocking is strongly recommended, and arrangements should be made for definitive treatment of the underlying varicose veins (figure 10B).

Venous pigmentation (haemosiderin staining)

This is one of the more common skin manifestations of chronic venous congestion (figure 11). The brownish skin discoloration results from haemosiderin (iron) staining of the skin secondary to asymptomatic extravasation of iron-con-

taining red blood cells into the subcutaneous tissue.

Haemosiderin staining can be persistent and is often a great source of embarrassment to patients. Colour reversal is slow (years) and frequently incomplete even after adequate correction of the underlying venous hypertension. Attempts to lighten the discolouration with skin bleaching and lasers are generally unsuccessful.

Lipodermatosclerosis

The progression towards lipodermatosclerosis (LDS) indicates ongoing reactive inflammation and scarring of the skin secondary to chronic venous insufficiency. The injury occurs within the subcutis, where the varicose veins are located. This is an ominous sign, heralding the onset of irreversible skin changes that puts the patient at risk of delayed wound healing and subsequent leg ulcers.

LDS occurs when there is fibrotic replacement of subcutaneous adipose tissue. In the initial stages (acute LDS), there may be significant inflammation in which the skin becomes painful and red (figure 12), and is often mistaken for cellulitis. It is not uncommon to find patients with LDS on multiple courses of antibiotics (without improvement) for presumed infection.

With time the skin becomes increasingly 'woody' from fibrous replacement of the subcutaneous tissue (figure 13). The inverted-champagne-bottle leg is characteristic of late stage (chronic) LDS.

The onset of LDS should prompt urgent treatment of the underlying varicose veins and venous hypertension, which serves to prevent further damage to the skin. Specific therapy for established

Figure 11: Venous pigmentation (haemosiderin staining).



Figure 13: Chronic lipodermatosclerosis.



LDS is difficult, as the fibrotic skin changes are usually permanent. High-frequency (3MHz) therapeutic ultrasound, similar to that used for sport injuries, can help soften the affected skin when used 2-3 times a week for a period of 6-8 weeks.

Venous ulcers

Venous ulcers result from end-stage chronic venous hypertension, when the skin finally breaks down (figure 14). It is invariably associated with other features of chronic venous hypertension, such as varicose dermatitis, haemosiderin staining and LDS. The ulcers are typically located over the medial ankle, with a variable degree of pain. Atrophie blanche is a pre-ulceration stage in which the skin becomes scarred and atrophic.

The ulcer needs to be swabbed if secondary infection is suspected, but the results must be correlated clinically, as light growths of

Figure 12: Acute lipodermatosclerosis.



Figure 14: Venous ulcer.



contaminant or opportunistic micro-organisms do not necessarily imply infection. A secondary infection commonly gives rise to an increase in pain, along with a purulent exudate. Rarely, longstanding ulcers can lead to malignant transformation into squamous cell carcinoma, which can potentially metastasise.

The initial management is compression (eg, four-layer compression bandages). Always check the ankle-brachial pressure index for peripheral arterial vascular disease, as mixed arterio-venous ulcers can occur. As soon as the venous ulcer is stable or improving, varicose vein treatment should be started as a definitive strategy to actively heal the ulcer and prevent relapses. Some ulcers will not heal unless the underlying varicose veins are adequately treated. Atypical-looking ulcers need to be biopsied to exclude malignancy.

Venous malformation

Classic venous malformation

THIS is the most common type of vascular malformation (1-4% prevalence). The malformation is present at birth and grows in proportion to the rest of body (figure 15). Venous malformations (VMs) are at times confused with haemangiomas, which are readily distinguishable by their later onset (not present at birth) and their biphasic pattern of an initial rapid growth phase followed by involution. Both conditions can affect skin and underlying structures such as muscle and bone of the affected limb.

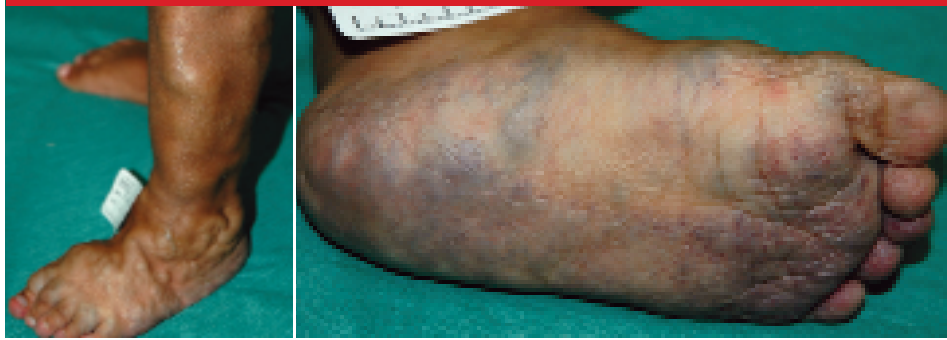
Common presenting symptoms for lower-limb VMs include lesion growth, disfigurement and pain. Limb enlargement can occur, especially with large venous malformations. VMs occurring on the plantar foot can be painful with weight bearing. The malformation can also extend into bones and muscles and may compress adjacent structures. Haemorrhage into knee joints can lead to joint degeneration.

VMs are low-flow lesions and episodic thrombosis may occur. They can get worse with trauma, incomplete surgical excision and hormonal changes. They can also recur after treatment, so patients need to be prepared for repeat procedures over time.

VMs can usually be diagnosed clinically. Ancillary imaging tools such as X-ray, ultrasound, MRI and CT scans can help delineate the extent of the malformation and any associated abnormalities. Duplex ultrasound is a quick non-invasive method of investigating the malformation and can also be used to guide sclerotherapy of the VM. X-rays can show phleboliths that are characteristic of VMs and can also demonstrate any bone involvement by the VM. MR and CT angiography/venography can non-invasively show the extent of the lesion in exquisite detail.

Treatment of VMs generally involves sclerotherapy (80-100% ethanol for larger

Figure 15: Venous malformation lower limb.



lesions; 1-3% sodium tetradecyl sulfate for smaller lesions) and compression. Lasers (1064nm Nd:YAG) may shrink some of the smaller and more superficial lesions.

Surgery can be used to treat small isolated lesions but is not as useful for lesions with deeper involvement and for those close to vital neurovascular structures. Surgery can be attempted after pre-treatment with sclerotherapy to shrink the lesion to a more manageable size.

In general, the outcome and prognosis of a VM is related to its location, size, type (diffuse versus circumscribed), and whether there is any involvement with vital neurovascular structures. A multidisciplinary team of dermatologist, paediatrician, radiologist, plastic surgeon and vascular surgeon is necessary for complicated VM patients.

Arteriovenous malformation

Arteriovenous malformations and VMs are similar except the AVM has a defining aberrant (direct) communication between the arterial and venous systems. AVMs are high-flow lesions whereby the affected skin feels warmer and may exhibit a thrill on palpation or a bruit on auscultation.

It is important to differentiate between VMs and AVMs because the latter are more commonly complicated by pain, ulceration and progressive destruction that may require surgical intervention (figure 16). Congestive heart failure can be a consequence of large AVMs.

In general, treatment is

Figure 16: Arteriovenous malformation with ulceration.



Figure 17: A: Glomovenous malformation before sclerotherapy and Nd:YAG laser. B: After sclerotherapy and Nd:YAG laser.



more difficult for AVMs because of intrinsic arterial connections that can lead, for example, to tissue or organ ischaemia with attempted sclerotherapy and embolisation. Sclerotherapy (ethanol 80-100%) along with conservative embolisation with coils and glues (cyanoacrylate) can temporarily shrink lesions, making them more amenable to surgical resection (with less intraoperative bleeding).

Procedural intervention is aimed at improving quality of life and controlling complications such as pain, ulceration and bone hypertrophy

or lysis. The overall prognosis is generally favourable although long-term monitoring for post-treatment recurrence is necessary.

Glomovenous malformation

Glomovenous malformations (GVMs) present as slightly raised blue-green lesions that closely resemble VMs in presentation (figure 17). The diagnosis can only be confirmed on a skin biopsy in which the diagnostic glomus cells (immature smooth muscle cells) are seen lining the vascular channels of the

venous malformation.

GVMs result from mutations in the glomulin gene, and may have an autosomal dominant inheritance. Although always present at birth, GVMs usually become clinically apparent only later in life. Patients with a solitary plaque of GVM may also have a widespread eruption of individual glomangiomas.

A glomus tumour, on the other hand, is an unrelated condition in which a tight collection of glomus cells is found under and around the nail. Treatment options for GVMs and VMs are essentially similar.

Imaging of vascular malformations

Ultrasound, CT and MRI or magnetic resonance angiography, and to a lesser extent X-rays, are all used for imaging of vascular malformations.

MRI offers the most information and can usually reliably assess the extent of the lesion as well as determine whether the lesion is high or low flow. If the lesion has high-flow characteristic this would be more consistent with an AVM rather than a VM. Low-flow lesions are either VM or lymphatic malformations. Contrast enhancement can further distinguish between lymphatic and venous malformation, whereby VM shows positive enhancement due to uptake of contrast media.

Ultrasound has the advantage of portability and easy access. It is useful for triage, assessment and treatment of vascular lesions. Ultrasound is also an excellent way to track lesion progression and response to treatment. Duplex, colour Doppler and Doppler spectral analysis are ultrasonic tools that help differentiate arterial from venous flow.

For high-flow lesions such as AVM and arteriovenous fistula, angiography is still considered the 'gold standard' and a prerequisite for interventional therapy, including surgical resection. Angiography also serves to guide sclerotherapy and embolisation of malformations.

Further reading

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Online resources

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- Emedicine: www.emedicine.medscape.com:
 - Weiss R, Field CF. Varicose veins and spider veins. (Updated 7 July 2010.)
 - Speer AL, et al. Arteriovenous malformations. (Updated 9 August 2010.)
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 - Konez O. Vascular anomalies. (Updated 25 February 2010.)

Varicose veins: key facts

- Dilated, tortuous veins affecting the lower limb
- Results from either faulty venous valves or weakness in the vein wall
- More common in women than men
- Hereditary and sedentary lifestyle factors contributory
- Often of cosmetic concern (may be asymptomatic) for patients
- Patients may complain of symptoms of venous congestion
- Progressively worsen with age and can lead to skin- and vein-related complications
- Duplex mapping required to assess severity and extent of underlying venous disease
- Both surgical and non-surgical treatment options exist and should be considered
- Skin complications will not resolve unless the underlying venous hypertension is managed

Author's case study

A 42-YEAR-old man presents with a persistently itchy rash on the left leg over the past four years (figure 10A). He has been diagnosed with localised dermatitis and prescribed topical steroids, which temporarily relieve the itch but do not clear the rash. He is also concerned about the aching in the left leg, particularly with prolonged standing.

Clinically, there is evidence of dermatitis of the left lateral leg that is associated

with dilated veins and telangiectasias underlying the rash. What is your provisional diagnosis? How would you investigate and manage this patient?

Provisional diagnosis

Varicose dermatitis with symptoms of venous congestion (aching leg).

Investigation

Duplex imaging is essential to confirm the superficial venous disease and to pro-

vide a venous map of the leg that is a necessary template for treatment (sclerotherapy). A bacterial swab may be clinically indicated for infected stasis dermatitis or ulcers but was not clinically indicated for this patient. It is not necessary to biopsy varicose dermatitis.

Management

The patient had a duplex ultrasound scan, which showed reflux in the super-

ficial anterior tibial vein and the anterolateral calf vein, corresponding to the area of recalcitrant dermatitis.

Topical steroids offer only temporary relief in the presence of ongoing venous congestion. External compression with stockings during waking hours is helpful but needs to be ongoing, making this difficult for most patients to adhere to.

For definitive treatment, this patient received ultrasound-guided injections of

foam sclerosants (sodium tetradecyl sulfate 0.5%) to the incompetent tributaries underlying the dermatitis. Compression stockings were applied for a period of three weeks.

Within a month post-procedure, the patient reported significant improvements in the chronic venous congestion symptoms and marked resolution of the dermatitis that has been treatment-resistant for more than four years (figure 10B).

cont'd next page

GP's contribution



DR MICHELLE CROCKET
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Case study

WENDY is now in her mid 30s and had developed varicose veins in her teenage years, which were treated when she was about 17. As she was not a patient of the practice at the time of the surgery, no information was available from the treating surgeon. However, the procedure was most likely stripping of the great saphenous veins bilaterally.

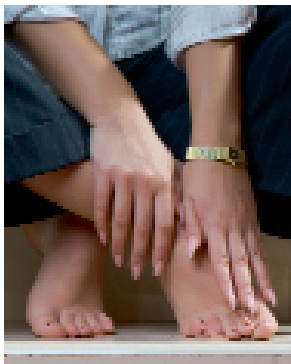
After the procedure she developed bilateral lower leg oedema that had persisted. This was both cosmetically unacceptable and uncomfortable. She also experienced recurrent episodes of cellulitis that required intramuscular penicillin injections followed by oral penicillin. These episodes occurred about 3-4 times a year.

Her distress was further exacerbated when a subsequent surgeon told her she should not have had the procedure.

Questions for the author

How common is this complication?

Persistent lower leg/ankle oedema is uncommonly encountered after varicose vein stripping or sclerotherapy. The cause for this is essentially unknown. It is possible that unintended disruption of the draining lymphatics could result in lymphoedema that may be responsible for some cases of leg swelling. Another cause of persistent leg swelling post-procedure is iatrogenic DVT resulting in deep venous incompetence and congestion. Any form of chronic limb oedema may predispose to skin infections such as cellulitis. However it has to be pointed out that the differential diagnoses for cellulitis in the setting of varicose veins and venous congestion include superficial thrombophlebitis and acute lipodermatosclerosis, as discussed in the article.



Could this have been predicted?

No. It is difficult to predict a patient's susceptibility to persistent leg swelling, after a vein procedure, as the aetiology is not precisely known. A positive family history of leg swelling from lymphoedema may point towards an increased likelihood of developing this problem post-treatment (anecdotal evidence).

Could this have been prevented?

Most post-treatment leg swelling has an unknown cause and therefore cannot be prevented. Patients with inherited 'weak' lower limb lymphatics may present with lymphoedema for the first time either spontaneously in adult-

hood or triggered by procedures such as leg vein stripping or sclerotherapy, and this would be difficult to prevent in the context of necessary varicose vein treatment. General precautions against DVT should be routinely undertaken. Early and regular mobilisation and avoiding long-distance travel post-procedure can reduce the risk of post-thrombotic damage to deep veins that can result in persistent lower limb oedema.

General questions for the author

GPs see many patients with varicose veins that appear to be 'minor' without any skin changes or other complications. Is symptomatic treatment with compression sufficient for these patients, and at what stage should they be referred for assessment by a vascular surgeon?

Compression is an effective measure against the symptoms and complications of varicose veins and venous congestion. Compression stockings can certainly keep the patient out of trouble when they are worn regularly. Many patients have difficulty adhering to a regular

compression schedule especially in warmer weather. In the long term, it would be beneficial for these patients to be formally assessed by a vein specialist for definitive treatment to correct any existing vein problems and prevent potential complications.

You have mentioned that NSAIDs are useful in DVT prevention in the presence of superficial thrombophlebitis. Is there any evidence for their use in DVT prevention in other settings such as travel? Is there evidence for use of mild compression stockings in DVT prevention for travel?

NSAIDs do not offer DVT prophylaxis in long-distance travel or other situations of reduced mobility. There are several studies highlighting the benefits of compression stockings in preventing travel-related DVT, and is especially recommended for those suffering from varicose veins.

Is sclerotherapy uncomfortable?

Patients tolerate the procedure remarkably well considering that the number of injections per session range from

15 to 30, or even more in patients with extensive leg telangiectasias. Fine-gauge needles (25-gauge or 30-gauge) are used, which are mostly perceived as a pricking or scratching sensation. No anaesthetic is required for the procedure.

There are now a number of 'vein clinics' staffed by non-vascular surgeons that treat varicose veins. Can you make any comment on these, such as when it may be appropriate for patient to be treated at these clinics?

Over the past decade, varicose vein treatment methods have expanded to include ultrasound-guided sclerotherapy, endovenous laser treatments and other non-surgical alternatives. Consequently, the range of healthcare providers has broadened to include interventional radiologists, dermatologists and other specialist and non-specialist practitioners. The Australasian College of Phlebology is a useful contact point for appropriately trained doctors from various disciplines interested in managing venous disorders such as varicose veins.



How to Treat Quiz

Superficial venous disorders of the lower limb — 27 May 2011

INSTRUCTIONS

Complete this quiz online and fill in the GP evaluation form to earn 2 CPD or PDP points. We no longer accept quizzes by post or fax.

The mark required to obtain points is 80%. Please note that some questions have more than one correct answer.

ONLINE ONLY

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1. Which TWO statements are correct?

- a) Varicose veins may be due to incompetence of valves within the affected superficial veins
- b) Varicose veins may be due to incompetence of valves between superficial and deep leg veins
- c) Deep venous thrombosis cannot cause superficial venous hypertension
- d) Venous insufficiency is associated with reduced venous pressure

2. Which TWO statements are correct?

- a) In varicose veins there is increased venous blood flow towards the heart
- b) Leg discomfort from venous insufficiency is made worse by walking, compression and leg elevation
- c) Risk factors for venous insufficiency include standing occupations, multiple pregnancies, and previous DVT
- d) Venous and arterial insufficiency can coexist

3. Which TWO statements are correct?

- a) Symptoms correlate well with the degree of venous insufficiency
- b) Phlebitis, thrombosis and haemorrhage can occur with chronic venous congestion
- c) Duplex ultrasound maps the venous system of the leg, and identifies and measures incompetent valves and venous reflux

d) Treatment of incompetent leg veins cannot reverse complications arising from chronic venous congestion

4. Which TWO statements are correct?

- a) Treatment of incompetent leg veins can relieve symptoms of venous congestion
- b) Vein stripping involves ligation of the saphenofemoral junction and removal of the saphenous vein
- c) Endovenous thermal ablation of veins is only indicated for patients for whom vein surgery is contraindicated
- d) Tortuous veins are well suited to endovenous thermal ablation methods

5. Which TWO statements are correct?

- a) Non-truncal veins such as incompetent tributaries and tortuous varicosities can be treated surgically with ambulatory phlebectomy
- b) Sclerotherapy can be used as primary therapy to treat varicose veins
- c) Sclerotherapy cannot be used for varicose vein recurrence previously treated with surgery
- d) Residual venectasias and telangiectasias following vein stripping or endovenous vein ablation is usually not responsive to sclerotherapy

6. Which TWO statements are correct?

- a) Sclerosants disrupt the endothelium, resulting in a sclerotic or fibrotic vein that is gradually absorbed by the body
- b) Surface 'cosmetic' leg veins rarely have occult incompetent or refluxing veins
- c) Duplex ultrasound can precisely locate deep veins and arteries, as well as superficial veins
- d) Foam sclerotherapy is contraindicated for large veins

7. Which TWO statements are correct?

- a) The risk of air embolisation is minimised by strictly limiting the amount of foam sclerosant used per treatment
- b) Walking should be avoided after sclerotherapy
- c) Telangiectasias and varicose veins usually resolve within one week after sclerotherapy
- d) Most cases of matting and staining will resolve over 3-6 months

8. Which TWO statements are correct?

- a) Skin and vein complications only occur if varicose veins are symptomatic
- b) Skin complications of chronic venous hypertension include stasis dermatitis, cellulitis and ulceration
- c) Tenderness and redness associated with

superficial thrombophlebitis is usually due to infection

d) Treatment of superficial thrombophlebitis includes compression of the lower limb with graduated stockings, and regular walking

9. Which TWO statements are correct?

- a) Heparin then warfarin are first-line pharmacotherapy for superficial thrombophlebitis
- b) Below-knee superficial thrombophlebitis is associated with a high risk of DVT
- c) Bleeding from varicose vein rupture which does not settle with compression should be treated with foam sclerotherapy
- d) Management of stasis dermatitis should include treatment of the underlying chronic venous hypertension

10. Which TWO statements are correct?

- a) The presence of lipodermatosclerosis is an indication for urgent treatment of the underlying venous hypertension
- b) The skin inflammation of lipodermatosclerosis is usually due to infection
- c) Secondary infection of chronic venous ulcers is associated with purulent exudate and increased pain
- d) Compression bandaging is contraindicated for chronic venous ulcers

CPD QUIZ UPDATE

The RACGP requires that a brief GP evaluation form be completed with every quiz to obtain category 2 CPD or PDP points for the 2011-13 triennium. You can complete this online along with the quiz at www.australiandoctor.com.au. Because this is a requirement, we are no longer able to accept the quiz by post or fax. However, we have included the quiz questions here for those who like to prepare the answers before completing the quiz online.

NEXT WEEK The next How to Treat investigates thyroid disorders in pregnancy. The author is **Clinical Associate Professor Aidan McElduff**, senior staff specialist in endocrinology, Royal North Shore Hospital; clinical associate professor, discipline of medicine, University of Sydney; and endocrinologist, Northern Sydney Endocrine Centre, St Leonards, NSW.

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