Superficial vein thrombosis:
A risk-based guide for general practice

Azeez A Kareem, Ameen Gafoor, Mahmoud Al-Najjar

Background
Superficial vein thrombosis (SVT) is a common presentation in general practice, often appearing as a tender, cord-like swelling along a superficial vein. Although usually self-limiting, SVT may extend into the deep venous system or embolise, increasing the risk of deep vein thrombosis (DVT) or pulmonary embolism. Given its overlap with venous thromboembolism (VTE), general practitioners must recognise and assess higher-risk cases.

Objective
This article outlines a practical, risk-based approach to SVT, drawing on contemporary guidelines to guide investigation, anticoagulation and follow-up.

Discussion
SVT lies on the VTE spectrum, with clinical risk determined by thrombus location, length and proximity to deep veins. Although some cases require only symptomatic care, others benefit from anticoagulation. Duplex ultrasonography is central to diagnosis and monitoring. Rivaroxaban is effective in selected high-risk cases. Special populations, such as pregnant patients or those with renal impairment, require tailored management.

SUPERFICIAL VEIN THROMBOSIS (SVT) is commonly seen by general practitioners (GPs) and is now clearly understood as part of the broader venous thromboembolism (VTE) spectrum, alongside deep vein thrombosis (DVT) and pulmonary embolism (PE).[1,2] Every year in Australia, between 15,000 and 23,000 people develop VTE, with around 52 per 100,000 specifically experiencing a DVT.[1,2]

Clinically, SVT refers to thrombosis and inflammation of a superficial vein, most commonly the great saphenous vein.[2,3] It typically presents with pain, redness and a palpable cord along the vein.[2–4] Although the terms SVT, superficial thrombophlebitis (STP) and 'thrombophlebitis' are still used interchangeably, newer literature has also adopted the abbreviation SVT to specify superficial vein thrombosis and reduce ambiguity in the literature. However, SVT remains the preferred terminology in current guidelines because of its clarity and relevance in modern VTE management.[5–8]

To reduce clinical confusion and improve communication, many traditional eponyms in venous anatomy have been modernised.[5,8] For example, the 'greater' or 'long' saphenous vein is now referred to as the great saphenous vein (GSV), and the 'lesser' or 'short' saphenous vein has been renamed the small saphenous vein (SSV). Similarly, the term 'superficial femoral vein' (SFV) misleadingly implies a superficial structure and has been corrected to simply 'femoral vein' to better reflect its deep venou status.[5,8]

Though SVT has been historically considered a benign condition, contemporary evidence suggests otherwise, particularly when a thrombus is located near deep venous junctions.[2,8] Approximately 20–25% of patients with SVT are found to have concomitant venous thromboembolism (DVT or PE) at diagnosis, including both symptomatic and clinically silent cases.[2,3,8]

In primary care, it is essential to recognise SVT as a clinically relevant thromboembolic disorder rather than a self-limiting complaint. A structured, risk-based approach enables GPs to manage high-risk presentations while avoiding overtreatment in low-risk cases safely.

Risk factors

SVT and DVT share many predisposing factors, reflecting their position along the VTE spectrum. Key clinical risk factors for SVT are summarised in Table 1.

Clinical presentation

Patients usually present with localised pain, erythema, tenderness and a palpable cord along a superficial vein.[2,4] In patients with significant limb swelling, GPs must urgently exclude DVT, particularly when SVT is proximal or located along major superficial veins such as the GSV and SSV.[2]

A first presentation of SVT in patients aged over 40 years, particularly in the absence of known risk factors, should prompt consideration of an underlying malignancy. Additionally, recurring or migratory thromboses – appearing at various anatomical sites over time – may indicate Trousseau's

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Table 1. Risk factors for superficial vein thrombosis (SVT)[4,9]

Venous stasis	• Varicose veins (present in up to 80% of SVT cases) • Immobility (eg recent surgery, prolonged travel, bed rest) • Obesity
Vessel wall injury	• Recent intravenous cannulation or central line placement • Local trauma or lower limb/pelvic surgery
Hypercoagulable states	• Active malignancy • Hormonal therapy (eg oral contraceptives, hormone replacement therapy) • Pregnancy (highest risk early postpartum) • Inherited thrombophilia (eg Factor V Leiden, protein C/S deficiency)
History of thrombosis	• Personal history of venous thromboembolism (SVT, deep vein thrombosis or pulmonary embolism) • Family history of venous thromboembolism
Other contributory factors	• Age >60 years • Systemic inflammatory or autoimmune disease (eg lupus, Behçet's disease) • Smoking

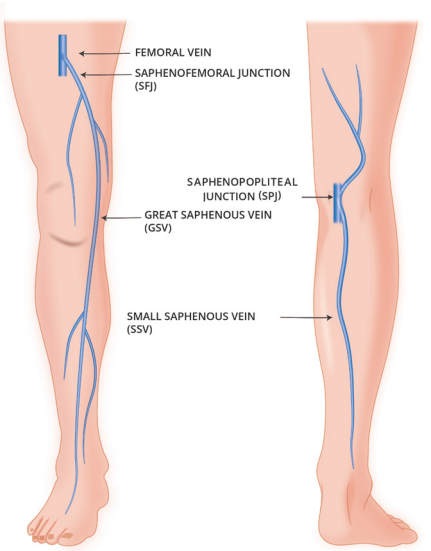


Figure 1. Anatomical illustration of the lower limb superficial venous system. Key labelled structures include the great saphenous vein (GSV), small saphenous vein (SSV), femoral vein, saphenofemoral junction (SFJ) and saphenopopliteal junction (SPJ). This layout highlights common sites of superficial vein thrombosis and their proximity to the deep venous system.

syndrome, a characteristic paraneoplastic manifestation of migratory thrombophlebitis, and warrant further investigation.[2]

Although SVT is often diagnosed clinically, it might mimic other conditions such as cellulitis, lymphangitis or vasculitis. Duplex ultrasonography is recommended in such cases, especially when the symptoms are proximal or atypical, or when VTE risk factors are a concern.[9]

Upper limb involvement may occur, particularly after recent intravenous cannulation or central venous catheter use. These cases are typically isolated and short, and anticoagulation is usually not required unless the thrombus is extensive, is symptomatic beyond the cannulated segment or occurs in patients with elevated thrombotic risk. Supportive measures such as warm compresses, nonsteroidal anti-inflammatory drugs (NSAIDs) and catheter removal are typically sufficient.[3]

Approach

Anatomy

As depicted in Figure 1, the main veins in the lower limb superficial venous system are the GSV and SSV. These are superficial veins that drain into the deep venous system via the saphenofemoral junction (SFJ) and saphenopopliteal junction (SPJ), respectively. These anatomical junction sites are clinically significant, as a thrombus located within 3 cm of these junctions is considered high risk for extension into the deep venous system and subsequent PE.[5,8]

Perforating veins connect the superficial and deep venous systems, typically allowing blood to drain from superficial veins into deep veins. This unidirectional flow is maintained by valves within the perforators that prevent retrograde movement.[2] However, if these valves become incompetent or if superficial venous pressure increases (eg from varicose veins), blood may reflux from the deep system back into superficial veins. This reversed flow contributes to venous stasis and endothelial irritation, promoting thrombus formation within the superficial system. Once formed, the thrombus may then propagate in the normal anatomic direction – from superficial to deep – mainly when located near junctions such as the SFJ or SPJ, thereby increasing the risk of DVT or PE.[2]

Diagnostic evaluation

Although clinical judgement is key, duplex ultrasonography is the definitive test for diagnosing SVT. It provides information on thrombus length and proximity to major deep vein junctions (eg SFJ, SPJ).[4,10] Duplex ultrasonography is also essential in confirming or excluding associated DVT. It is recommended in most patients with suspected SVT, particularly if symptoms are proximal or atypical, or when risk factors for VTE exist. For isolated distal SVT confined to varicose veins without risk factors, a clinical diagnosis may suffice. D-dimer testing has no role in SVT diagnosis.[3]

When referring patients for ultrasonography, GPs should request reports that include confirmation of SVT (whether acute or chronic), the vein involved, the length of the thrombus and its distance from the SFJ and SPJ. Additional essential details include the presence or absence of DVT (whether contiguous or separate), evidence of propagation, varicose vein involvement, compressibility findings and whether the thrombus correlates with the patient's symptoms. If ultrasonography indicates that the vein does not compress,

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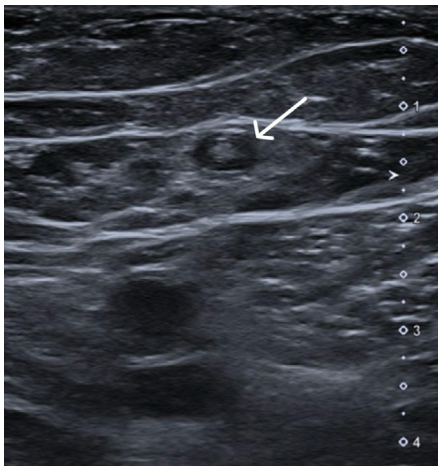


Figure 2. Duplex ultrasound of the anterior accessory saphenous vein showing superficial vein thrombosis. The affected segment appears non-compressible with internal echoes consistent with thrombus and increased echogenicity.

as shown in Figure 2, this suggests the presence of a thrombus. These elements are critical for accurate risk stratification and guide whether conservative management, prophylactic anticoagulation or full therapeutic treatment is appropriate.[10]

Risk stratification

SVT is stratified by thrombus characteristics and patient risk profile:

- Low risk: Thrombus <5 cm in length, >3 cm from deep vein junctions (SFJ/SPJ), no additional risk factors (eg a 3-cm thrombus in mid GSV, no malignancy or thrombophilia).[7,8,11]
- Intermediate risk: Thrombus ≥5 cm in length or within 3–5 cm of a deep venous junction, no systemic risk factors (eg a 6-cm thrombus located 4 cm from SPJ in a healthy patient).[8]
- High risk: Thrombus ≤3 cm from a deep venous junction, or evidence of propagation, recurrent SVT, thrombophilia or malignancy (eg a 4-cm thrombus located 2 cm from SFJ in a patient with known thrombophilia).[6,8,12]

Patients presenting with SVT must be assessed to stratify risk and tailor management according to the diagnostic guidelines outlined in Figure 3.

Management

The management of SVT draws on existing VTE treatment protocols, with dosing adapted to thrombus characteristics and risk level. Care should be guided by clearly defined risk stratification criteria (Table 2).

Low-risk SVT

Patients presenting with low-risk SVT (<5 cm thrombus length and >3 cm from deep venous junctions) typically require symptomatic care without anticoagulation.[13] Graduated compression stockings may also be considered for symptomatic relief, particularly in patients with varicose veins, provided there are no contraindications such as severe peripheral arterial disease.[2] If symptoms persist or worsen following conservative management, repeat clinical examination and follow-up duplex ultrasonography within 2–3 days are recommended (Figure 3).

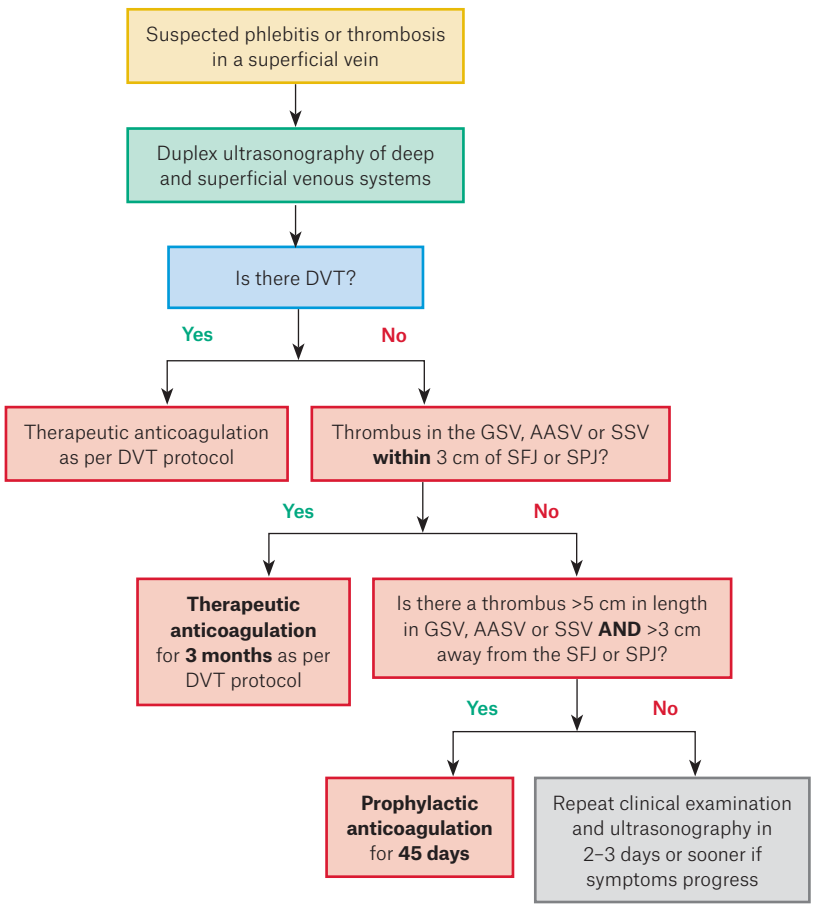


Figure 3. Risk-stratified diagnostic and treatment algorithm for SVT. Initial duplex ultrasonography assessment determines the presence of DVT or proximity of superficial thrombus to the SFJ or SPJ. Treatment decisions are based on thrombus length, location and associated risk factors. Management pathways include therapeutic anticoagulation for high-risk SVT (within 3 cm of deep junctions or with DVT), prophylactic anticoagulation for intermediate-risk cases, and surveillance for low-risk SVT.

AASV, anterior accessory saphenous vein; DVT, deep vein thrombosis; GSV, great saphenous vein; SFJ, saphenofemoral junction; SPJ, saphenopopliteal junction; SSV, small saphenous vein; SVT, superficial vein thrombosis.

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Intermediate-risk SVT

Intermediate-risk cases, defined as thrombi ≥5 cm in length but ≥3 cm from deep veins, require prophylactic-dose anticoagulation for 45 days. Fondaparinux 2.5 mg once daily has the strongest trial evidence (CALISTO)[14] and is listed on the Pharmaceutical Benefits Scheme (PBS) in Australia; however, in general practice, prophylactic-dose rivaroxaban 10 mg once daily or enoxaparin 40–80 mg once daily are more commonly prescribed.[8,12] Rivaroxaban has also been shown to be non-inferior to fondaparinux in the SURPRISE trial.[15] Enoxaparin remains an acceptable option in practice,[8] though its evidence is supported mainly by smaller and older trials.[12,16]

High-risk SVT

High-risk cases, identified by thrombus proximity (within 3 cm of deep veins), recurrent disease or significant clinical risk factors (eg previous VTE, thrombophilia), require therapeutic-dose anticoagulation for 3 months. Options include full-dose low molecular weight heparin (LMWH), rivaroxaban or apixaban. The regimen for apixaban is 10 mg twice daily for 7 days, then 5 mg twice daily;[8,16] the regimen for rivaroxaban is 15 mg twice daily for 21 days, then 20 mg once daily.[8,13,16] Full-dose LMWH (eg enoxaparin 1 mg/kg twice daily or 1.5 mg/kg once daily) remains an alternative. Recent data from the COBRRA trial suggest an increased risk of bleeding with rivaroxaban when compared with apixaban in acute VTE; however, these findings are extrapolated from VTE data and have not altered SVT-specific recommendations but may influence therapeutic choice.[17]

Table 2. Risk stratification and management principles for SVT[8,12-16]

Risk category	Management
Low-risk SVT: thrombus <5 cm in length, >3 cm from deep vein junctions (SFJ/SPJ), no additional risk factors	Anticoagulation not required • Topical/systemic nonsteroidal anti-inflammatory drugs (ibuprofen or diclofenac) for 7–14 days • VTE prophylaxis: limb elevation, compression stockings, ambulation, warm/cool compresses
Intermediate-risk SVT: thrombus ≥5 cm or within 3–5 cm of a deep venous junction, no systemic risk factors	Prophylactic anticoagulation per VTE treatment guidelines: 45 days • Fondaparinux 2.5 mg once daily for 45 days • Enoxaparin[A] 40–80 mg once daily for 45 days, **or** • Rivaroxaban[B] 10 mg once daily for 45 days, **or** • Apixaban[C] 2.5 mg twice daily for 45 days Follow-up: Reassess thrombus progression and ongoing VTE risk
High-risk SVT: thrombus ≤3 cm from a deep venous junction, or evidence of propagation, recurrent SVT, thrombophilia or malignancy	Therapeutic anticoagulation per VTE treatment guidelines: 3 months • Enoxaparin[A] 1 mg/kg twice daily or 1.5 mg/kg once daily, **or** • Rivaroxaban[B] 15 mg twice daily for 21 days, then 20 mg once daily, **or** • Apixaban[C] 10 mg twice daily for 7 days, then 5 mg twice daily Close follow-up: Monitor thrombus propagation or resolution with serial duplex ultrasonography

[A] Enoxaparin: For prophylaxis, 40–80 mg once daily. For therapeutic anticoagulation, 1 mg/kg twice daily or 1.5 mg/kg once daily, with twice-daily dosing preferred in patients at higher bleeding risk (eg advanced age, active cancer or extremes of weight ≥150 kg).

[B] Avoid rivaroxaban if creatinine clearance <30 mL/min; seek specialist input.

[C] Apixaban has not been directly trialled in SVT; use extrapolated from VTE data. The COBRRA trial[17] demonstrated lower bleeding risk with apixaban compared with rivaroxaban in acute VTE. Consider where rivaroxaban is less suitable (eg renal impairment, higher bleeding risk).

SFJ, saphenofemoral junction; SPJ, saphenopopliteal junction; SVT, superficial vein thrombosis; VTE, venous thromboembolism.

Non-GP specialist referral (eg vascular surgery, haematology) may be required in complex cases, including:

- recurrent or propagating SVT despite anticoagulation[6,16]
- thrombus within 3 cm of a deep vein not resolving on treatment[6,16]
- SVT associated with active malignancy[3,4,5]
- suspicion of inherited or acquired thrombophilia[3,6]
- atypical anatomical location or unclear duplex ultrasonography findings[4,6]
- requirement for anticoagulation beyond 45 days (intermediate risk) or 3 months (high risk).[16]

Special populations

Pregnancy

Direct oral anticoagulants (DOACs) are contraindicated during pregnancy.[1] In low-risk cases, prophylactic or intermediate-dose LMWH (eg enoxaparin 40–80 mg daily) may be used for 1–6 weeks.[1] The choice of dose and duration depends on thrombus length, severity of symptoms and gestational age, with non-GP specialist input recommended for a thrombus ≥5 cm in length, for bilateral symptomatic SVT or if symptoms persist despite treatment.[1] For patients not receiving anticoagulants, serial duplex ultrasonography every 7–10 days is recommended.[12,16]

Malignancy

Patients with active cancer should be given anticoagulation therapy for a minimum of 45 days. Extension beyond 45 days should be considered on the basis of the patient's risk profile and persistence of symptoms.[3,16]

Renal impairment

Rivaroxaban should be avoided in patients with estimated glomerular filtration rate (eGFR) <30 mL/min.[3,16] Enoxaparin may be used with dose adjustment based on renal function and bleeding risk.[3,16] Non-GP specialist input is recommended when managing anticoagulation in patients with severe renal impairment.

Surgical considerations

Surgical options such as vein ligation or ablation are typically delayed ≥3 months after the acute phase, during which appropriate anticoagulation (eg enoxaparin) is used to

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manage thrombotic risk. This is particularly relevant in recurrent cases associated with varicosities.[16]

Complications

SVT may progress to DVT or PE, particularly in high-risk cases.[4,18] Patients with a thrombus up to 3 cm from the SFJ should be treated for DVT, given that 14–70% progress to DVT.[2] Other recognised complications, such as suppurative thrombophlebitis and recurrent thrombophlebitis, are possible and further emphasise the importance of early risk assessment and appropriate treatment without delay.[4,18] Suppurative SVT typically arises in association with intravenous cannulation in the upper limbs; if there is local or systemic evidence of infection, antibiotics should be considered alongside supportive measures.[14] The probability of recurrent SVT is dependent on risk factors (active cancer, pregnancy), which should be reflected in the dosage and duration of the anticoagulation regimen.[3]

Follow-up and patient education

All patients with SVT should be counselled on the signs of DVT and PE, including unilateral leg swelling, chest pain and dyspnoea. Follow-up duplex ultrasonography within 7–10 days is recommended in intermediate- and high-risk cases, or if symptoms progress, to monitor for propagation.[3] Patients should be counselled on the importance of follow-up adherence. Preventive strategies such as compression stockings and consideration of varicose vein treatment should be discussed.[19,20] Additionally, patients should be informed that SVT carries an ongoing risk of recurrent thrombosis and progression to VTE,[18,21,22] and be counselled on the importance of medication adherence and the benefits and risks associated with anticoagulation therapy.

Conclusion

SVT is a clinically relevant thrombotic condition within the VTE spectrum. Timely recognition, targeted use of duplex ultrasonography, and risk-based management allow GPs to optimise care, balancing safety with avoidance of unnecessary anticoagulation.

Key points

- SVT is part of the VTE spectrum and can progress to DVT or PE.
- Duplex ultrasonography is recommended for proximal, atypical or high-risk SVT presentations.
- Low-risk SVT (<5 cm, >3 cm from deep veins, no risk factors) is typically managed conservatively; anticoagulation is reserved for intermediate- and high-risk cases.
- Apixaban may be considered when rivaroxaban is unsuitable (eg renal impairment, higher bleeding risk), though direct trial data in SVT are limited.
- Non-GP specialist referral is recommended for recurrent SVT, thrombus near deep veins, thrombophilia or associated malignancy.

Authors

Azeez A Kareem, James Cook University, Cairns, Qld

Ameen Gafoor, James Cook University, Townsville, Qld

Mahmoud Al-Najjar, BSc (Med), MBBS (UNSW), EMAD (ACEM), FRACGP, FARGP, FRACS (Vascular), Consultant Vascular and Endovascular Surgeon & Trauma Surgeon, Australian Vascular, Perth, Mandurah, Bunbury, Albany and Geraldton, WA; Clinical Senior Lecturer, Rural Clinical School of Western Australia, The University of Western Australia, Perth, WA; Co-opted Member, Rural Surgery Section Committee, Royal Australasian College of Surgeons; Elected Member, WA State Committee, Royal Australasian College of Surgeons; Appointed Member, WA State Trauma Subcommittee, Royal Australasian College of Surgeons; Member, Australasian College for Emergency Medicine

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Correspondence to:
info@australianvascular.com.au

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correspondence **ajgp@racgp.org.au**

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