BMJ Best Practice

Superficial vein thrombophlebitis

The right clinical information, right where it's needed

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Summary

- Superficial vein thrombophlebitis (SVT) of the lower limb is most often a complication of varicose veins.
- Not always a benign disease, as concomitant DVT may be present.
- Duplex ultrasonography should be performed on all patients with suspected SVT of the lower limb, as concomitant DVT may be present.
- Underlying prothrombotic conditions should be sought in cases of recurrent SVT and migratory SVT, especially in the absence of varicose veins.
- Anticoagulation to prevent venous thromboembolic complications (DVT or PE) is warranted, especially in patients with SVT near the saphenofemoral junction, or when the superficial thrombus is 5 cm or greater in length.

Definition

Superficial vein thrombophlebitis (SVT) refers to thrombus formation in a superficial vein, and inflammation in the tissue surrounding the vein. It is generally characterised by pain, tenderness, induration, and/or erythema in a superficial vein. There is often a palpable, sometimes nodular, cord with warmth and erythema, which suggests the presence of thrombus. It most often occurs in the saphenous vein and its tributaries of the lower limbs. It can also occur in the veins of the upper limbs or neck, usually due to intravenous cannulation and drug administration. The term SVT is generally reserved for the specific problem of SVT in the lower limbs. This monograph will concentrate on lower-limb SVT.

Epidemiology

Superficial vein thrombophlebitis (SVT) is thought to be a relatively common condition, but its exact incidence remains to be determined, in part due to the lack of studies in a disease thought to be relatively benign and self-limiting. Nonetheless, its incidence is generally thought to be higher than that of DVT, which has an estimated frequency of 1 in 1000 persons a year.[1] [2] [3] Some reports estimate an incidence of anywhere between 3% to 11% of the general population.[4] Most studies report a 4- to 6-fold increased predominance of the condition in women compared with men, possibly because of the increased prevalence of varicose veins in women.[5] [6] [7] [8] [9] [10] The incidence of SVT increases with age and is reported from 0.05 to 0.31 per 1000 persons a year during the third decade to 1.8 to 2.2 per 1000 persons a year in the eighth decade.[11]

Aetiology

Superficial vein thrombophlebitis (SVT) is most often associated with prothrombotic conditions characterised by one or more of the components of the Virchow triad: vessel wall damage (catheterisation, intravenous drugs, sclerotherapy, inflammatory vascular diseases); stasis (varicose veins, immobilisation); and hypercoagulability (oral contraceptive medicines, inherited or acquired thrombophilia).

The aetiological diagnosis depends partly on the location of the SVT. For SVT of the lower limbs, the main cause remains varicose veins. Local trauma to a varicose vein is often a precipitating factor in the development of SVT. Other underlying conditions such as autoimmune diseases (especially Behcet's and Buerger's disease), malignancy, and thrombophilia need to be identified or ruled out as causes in idiopathic, migrant, or recurrent SVT, and in the absence of varicose veins. Extensive SVT on old varicose veins may also be seen with malignancy.

For SVT of the upper limbs and the neck, the main cause consists of iatrogenic interventions such as intravenous catheters or infusion of drugs (chemotherapy, drug addiction). Mondor's disease, which primarily affects superficial veins of the chest wall and breast in women, is idiopathic in most cases but can sometimes be associated with severe exercise or trauma, and, on rare occasions, with breast cancer or thrombophilia.[10]

Pathophysiology

Thrombus formation in the vein is likely to be the result of interplay between the damaged vessel, the coagulation cascade, the fibrinolytic system, platelet activation, and activation of the inflammatory process.[12] However, the specific relation between these processes, particularly the coagulation and

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inflammatory cascades, remains unclear. It is postulated that vessel wall damage, whether due to cannulation of a vein or direct trauma to a varicose vein, triggers a prostaglandin-mediated activation of the inflammatory cascade as well as platelet activation and accumulation.[12] At sites where the endothelium is severely inflamed (e.g., as a result of vessel wall damage or underlying autoimmune disorders), clotting intermediates are activated and accumulate; combined with stasis, this can initiate thrombosis. Histopathological studies of veins with superficial vein thrombophlebitis demonstrate swelling of endothelial cells, leukocyte infiltration of the vein wall, and other changes consistent with inflammation, along with fibrin deposition and thrombus formation.[12]

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Secondary prevention

In women with recurrent SVT, avoidance of OCPs or other hormonal agents should be advised.

Varicose vein surgery may be feasible in patients with recurrent SVT in the setting of varicose veins.

Though the evidence linking long-haul aeroplane travel to SVT is controversial, it is prudent to advise patients with a history of SVT to ambulate regularly during the flight. Prevention with compression stockings and/or prophylactic doses of low molecular weight heparin in very high-risk patients may be useful, although this strategy has not been studied in a randomised clinical trial.

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Case history

Case history #1

A 45-year-old woman presents with a burning pain in her right calf, over an existing varicose vein, for the past 3 days. She has also noticed some reddening around that area extending from just below the knee to halfway down the lateral aspect of the right leg. The varicose vein has been relatively asymptomatic and first appeared after the birth of her first child when she was 32 years old. She has no known medical conditions and does not take any medicines. She has no previous history of DVT. Physical examination reveals a cord-like superficial vein on the outer aspect of her right leg. The overlying skin is erythematous and warmer than the adjacent leg skin. She is afebrile and her heart rate is normal. There is no clinical evidence of DVT.

Other presentations

Atypical presentations of superficial vein thrombophlebitis (SVT) include infected SVT, migratory thrombophlebitis, and Mondor's disease. Infected SVT can present with purulent discharge (suppurative thrombophlebitis) or without purulent discharge (non-suppurative thrombophlebitis). It is usually a complication of a skin infection (such as cellulitis); intravenous cannulation or chemical injection of the vein; or intravenous drug use. Migratory thrombophlebitis is defined by repeated thromboses developing in superficial veins, at varying sites but most commonly in the leg. It is usually associated with malignancy, particularly pancreatic adenocarcinoma. It can also be seen with Behcet's and Buerger's disease. Mondor's disease is a term originally used to describe a thin cord of the lateral thoracic or thoraco-abdominal wall without signs of inflammation (sometimes called 'phlébite fil de fer'). Today it is used to describe thrombophlebitis of a superficial vein in the subcutaneous fat of the breast and anterior chest wall, usually in women, and it may be associated with underlying malignancy. It also includes thrombophlebitis of the dorsal penile vein of the penis, generally caused by trauma or repetitive injury.

Step-by-step diagnostic approach

A diagnosis of superficial vein thrombophlebitis (SVT) is established primarily on the basis of clinical signs: pain, tenderness, induration, warmth, erythema, and/or a palpable cord along the course of a superficial vein. [Fig-1]

However, use of Doppler ultrasonography should be recommended to diagnose concomitant ipsilateral and/or contralateral asymptomatic DVT, to confirm the clinical diagnosis of SVT, and to show thrombus extension and thrombus head location regarding the saphenofemoral and saphenopopliteal junctions. Doppler ultrasonography may also help in the presence of a difficult differential diagnosis that may include cellulitis, erythema nodosum, and lymphangitis.

History

Patients with SVT often report the gradual onset of localised pain followed by the appearance of an area of redness along the course of a superficial vein. Some report constitutional symptoms such as low-grade fever, but high fever and purulent discharge suggest septic (infected) thrombophlebitis. Moreover, septic thrombophlebitis, which most often occurs as a result of bacterial entry from an intravenous catheter,

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may be associated with bacteraemia and severe systemic infection that can lead to shock. Symptoms of SVT typically develop over hours to days and resolve in days to weeks. Pigmentation changes of the skin overlying the SVT are often observed. The cord may remain palpable for several weeks to months after an initial episode of SVT. In a patient with varicose veins, there may be history of local trauma. Other known risk factors of SVT should be elicited on history. Strong risk factors include a history of varicose veins, thrombophilic disorders, autoimmune diseases, sclerotherapy, and intravenous catheterisation, and a previous history of SVT.

Examination

On physical examination, in a patient with pre-existing varicose veins, there is a tender 'worm-like' mass deep to the skin. The overlying skin is warm and erythematous, and there is often oedema of the surrounding area, without generalised swelling of the whole limb. In a patient without varicose veins, there is often a palpable cord, sometimes nodular, associated with warmth, tenderness, and erythema along the course of a non-varicose superficial vein. Erythema and oedema may extend for some distance into the surrounding tissue, making distinction from cellulitis difficult.

It is also important at this time to examine for the presence of underlying DVT, because clinical signs and symptoms of inflammation often lag behind the extension of the thrombus by several centimetres.[30] The prevalence of concomitant DVT varies widely in the literature from 2.6% to 65%, and, if present, it is thought to be contiguous in 50% to 75%. The thrombus extends by contiguity to the deep venous system through the saphenofemoral or saphenopopliteal junctions, or, less commonly, through the perforating veins.[31] It is important to note that up to 25% of concomitant DVT may not be contiguous with the SVT and may be in the contralateral limb.[32] When the DVT is non-contiguous, the mechanism of DVT occurrence is probably related to a hypercoagulable state.

Concomitant PE can also occur in anywhere between 0.5% to 4% of patients with SVT and symptoms of PE. Hence it is important to elicit history for PE symptoms such as dyspnoea, chest pain, and syncope.

Signs of infected SVT may be present, usually when the SVT is the result of vein instrumentation or cannulation, and can include high fever, frank pus at the site of injection or cannulation, and lymphangitis.

In patients with suspected arterial insufficiency by history and/or physical examination, an assessment of the ankle-brachial pressure index should be performed prior to the prescription of compression stockings.

Doppler ultrasound investigation

More often than not the diagnosis of SVT can be made on history and physical examination. However, clinical examination does not always reveal the true extent of the SVT. Surgical exploration often shows extension of the thrombotic process 5 to 10 cm higher than the level that was clinically suspected.[33] In fact, the practice of systematic Doppler ultrasonography has revealed a large number of DVTs concomitant with SVT (between 5.6% to 36%).[13] [34] [35] [36] [37] [38] This high variability can be explained by differences between studies with regard to the definition of DVT (i.e., proximal or distal, with or without muscular vein involvement). As such, there is emerging consensus that many patients with the clinical diagnosis of SVT in the lower extremities would benefit from bilateral lower-limb Doppler ultrasonography evaluation for confirmation of the diagnosis and exclusion of occult DVT in the ipsilateral and/or contralateral lower limb.

Doppler ultrasonography is particularly important if the patient has other risk factors for DVT (e.g., malignancy, pregnancy) or has oedema of the whole leg, or if the SVT is present above the knee. Several

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studies have shown that the risk of progression to DVT is higher when the SVT is present in the proximal greater saphenous vein or the saphenofemoral junction.[39] [40] [41] Moreover, some factors have been proposed to predict underlying DVT, such as male sex, history of DVT, short interval between onset of symptoms and diagnosis, and severe chronic venous insufficiency.[42] Other factors, such as greater saphenous vein SVT, bilateral SVT, age >60 years of age, and bed rest have been claimed as high-risk factors for underlying DVT but not confirmed.

Patients with an isolated SVT below the knee, associated with varicose vein and with no risk factors for venous thromboembolism (VTE), may not need Doppler ultrasonography evaluation, but this has not been explicitly examined in any trial.

Further investigations

Once Doppler ultrasonography evaluation is completed, further evaluation may be needed. Patients with SVT involving varicose veins and patients with a clear reason for the SVT (e.g., intravenous cannulation, sclerotherapy, oral contraceptive use) are likely to need no further diagnostic work-up, though clinical and Doppler ultrasonography follow-up may still be needed to confirm the absence of thrombus propagation, especially in patients with poor response to conservative therapy or who develop whole leg oedema.

A biopsy should be ordered in SVT cases that are recurrent and/or migratory, and when inflammatory diseases such as polyarteritis nodosa are being considered. This is best accomplished by an elliptical incision transverse to the long axis of the vessel rather than a punch biopsy. Small vessels in the superficial dermis are characteristically spared.[43]

In patients with suspected SVT with concomitant respiratory symptoms or signs of PE (chest pain, dyspnoea, syncope), a ventilation perfusion scan or pulmonary CT angiography to look for concomitant PE should be performed. If both tests are non-conclusive and PE is still suspected, then a conventional pulmonary angiogram is indicated.

Further evaluation for underlying VTE risk factors includes thrombophilia screening and assessment for underlying malignancy. This is indicated in patients with the following:

- SVT in short vein segments not associated with varicose veins
- Extensive saphenous vein thrombophlebitis on Doppler ultrasonography with or without concomitant DVT or PE
- Recurrent SVT
- Idiopathic SVT.

It should be noted, however, that the utility and cost-effectiveness of screening for thrombophilic states in patients with SVT has not been well studied. Thrombophilia screening includes testing for presence of factor V Leiden and prothrombin G202010A genetic variants; deficiencies of protein S, C, and antithrombin III; positive lupus anticoagulant and anticardiolipin antibodies; and hyperhomocysteinaemia.

Risk factors

Strong

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varicose veins

- Varicose veins are the most frequent cause of superficial vein thrombophlebitis (SVT). Up to 80% of patients with SVT of the lower limbs have pre-existing varicose veins with or without chronic venous insufficiency.[6] [10] [13]
- The great saphenous vein is involved in 60% to 80% of cases, and the small saphenous vein is involved in 10% to 20%.[14] SVT is more frequently found in varicose tributaries of the saphenous veins rather than the trunk.[6]
- Venous stasis is thought to be important in the development of SVT in varicose veins.

thrombophilic disorders

- Abnormalities of coagulation are associated with SVT, especially among patients with spontaneous SVT without varicose veins, or involving the greater saphenous vein trunk.
- In the absence of varicose veins, autoimmune disease, and malignancy, the risk of SVT has been shown to be 6-fold higher for the factor V Leiden gene mutation; 4-fold higher for the factor II (prothrombin) G20210A mutation; and 13-fold higher for antithrombin III, protein C, and protein S deficiencies taken together.[15]
- The presence of anticardiolipin antibodies and increased levels of factor VIII have been linked to an increased risk of recurrent SVT.[16] [17]

autoimmune diseases (e.g., Behcet's and Buerger's disease)

- Behcet's disease and Buerger's disease are frequently associated with SVT. The proposed pathogenic mechanism in both disorders involves mainly immune-mediated endothelial cell dysfunction.
- Behcet's disease is an autoimmune vasculitis disorder that is frequently associated with venous vascular complications. Among patients with Behcet's disease, up to 53% of patients will experience an SVT episode.[18] It is usually observed within 5 years of the diagnosis of Behcet's, and rarely does it precede the diagnosis.
- Buerger's disease is a non-atherosclerotic vascular disease also known as thromboangitis obliterans (TAO) and is characterised by segmental vascular inflammation, vaso-occlusive phenomenon, and involvement of small- and medium-sized arteries and veins of the upper and lower extremities.[19] SVT and, more commonly, migratory SVT, are thought to occur in up to 27% to 50% of patients with Buerger's disease.[19]

prior history of SVT

- It is well documented that a previous venous thrombotic episode is an important risk factor for VTE recurrence and that this risk is dependent on patient-specific factors such as the presence of malignancy. With regard to SVT, a prior SVT episode is also likely to be an important predictor of future SVT events, especially in patients with persistent risk factors such as varicose veins. Research has demonstrated that, after a first thrombotic episode of confirmed SVT of the lower extremities in patients with no history of DVT, varicose veins, malignancy, or autoimmune disorders, up to 32% of patients developed DVT at a median elapsed interval of 4 years and 24% had recurrent episodes of SVT.[15]

female sex

- Most studies reveal a preponderance of females (50% to 70%), possibly because of the increased prevalence of varicose veins during pregnancy.[8]

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sclerotherapy

- Sclerotherapy, through the injection of a sclerosant or foam into varicosed veins, provokes direct vessel wall damage, causing transmural wall damage, the subsequent generation of a local thrombus, and eventual transformation of the thrombosed vein into a fibrosis cord. The endpoint of this process is functionally analogous to surgical removal of a vein. As a result, SVT is a normal and expected occurrence following sclerotherapy. However, in rare instances it can extend and lead to post-sclerotherapy thrombophlebitis, which usually occurs within 1 to 2 weeks after the treatment of larger vessels (usually >1 mm).[27]
- The incidence of post-sclerotherapy SVT varies according to technique and type of sclerosant, and has been reported to be as high as 6%.[28]
- SVT and DVT have also been described following endovenous laser ablation of varicose veins.[29]

intravenous catheterisation

- Largely exclusive to upper-limb SVT rather than lower-limb SVT. Nonetheless, SVT can occur as a result of cannulation of superficial veins of the lower limbs and irritation of solutions or drugs delivered through the catheter.

Weak

malignancy

- Though malignancy is highly associated with an increased risk of DVT and PE, the association with SVT is not well known. Based on small retrospective cohort studies, among patients with SVT 10% to 15% may have a diagnosis of malignancy.
- Trousseau's syndrome (migratory superficial vein thrombophlebitis) is rare and is characterised by recurrent and migratory SVT, frequently in unusual sites such as the arm or chest (also called Mondor's disease when involving chest wall veins). It is most often associated with adenocarcinomas, especially pancreatic, lung, gastric, and prostate cancer.
- Malignancy-mediated activation of the coagulation cascade and malignancy-derived procoagulant factors are thought to be important in the development of SVT.

pregnancy

- There is limited information on the association of pregnancy and SVT. A retrospective study of 30,040 pregnant women found an incidence of ultrasonically confirmed SVT of 0.05% with most episodes occurring in the first 48 hours postpartum.[20] Increased age, parity, and hypertension are predisposing risk factors.[12] [19] [20]
- Pregnancy-related changes such as increase in procoagulant factors and reduced fibrinolytic activity may explain the increased risk during pregnancy, particularly in the puerperium. In addition, the pronounced vein dilation (and resulting stasis), particularly in the third trimester, is also a predisposing factor for SVT during pregnancy.

use of oral contraceptives and hormonal replacement therapy

- There is a lack of information on the specific association of OCP and hormone replacement therapy (HRT) on the risk of SVT. Based on several studies that have examined the association between OCP use and venous thromboembolic events (often including SVT), there is a 2- to 6-fold increased relative risk of venous thrombotic events among OCP users and a 2- to 4-fold increased risk among oral HRT users.[21]

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- OCPs that contain third-generation progestins are associated with a greater risk than those that contain second-generation progestins.
- OCP and HRT are associated with exponentially higher venous thromboembolism (VTE) risks when used by women with a thrombophilic condition.
- OCP- and HRT-mediated changes in procoagulant factors and natural anticoagulant proteins are thought to explain the increased risk of thrombosis. In particular, OCPs can produce an acquired activated protein C resistance very similar to that seen with the inherited factor V Leiden mutation.

older age

- Incidence increases with age (0.05-0.31 in 100 per year during third decade to 1.8-2.2 in 100 per year in eighth decade).[11]

history of prior venous thromboembolism (VTE), including DVT and PE

- The risk of SVT in patients with a history of VTE has not been well studied. However, in a study of patients with confirmed first spontaneous VTE and without varicose veins, malignancy, or autoimmune disorders, SVT developed in 7.3% of patients over an average follow-up of 30 months.[16] In addition, it was noted that patients with a first spontaneous VTE and subsequent SVT were at 3-fold increased risk of recurrent VTE compared with patients without SVT.

obesity

- Being overweight is recognised as a weak risk factor for the development of VTE.[22] [23] [24] With regard to SVT, a 2.6-fold increased risk of SVT has been reported in overweight (BMI ≥28 kg/m^2) patients when compared with patients with a BMI <28 kg/m^2, independent of the presence of varicose veins.[25]
- Obesity is a plausible risk factor for SVT because it predisposes to venous stasis and is associated with haemostatic changes.[26]

prolonged immobilisation (e.g., long-haul air travel)

- The role of long-haul flight in the development of SVT is unclear and it is probably associated with only a small percentage of SVTs.

DIAGNOSIS

History & examination factors

Key diagnostic factors

presence of risk factors (common)

- Key risk factors include varicose veins, thrombophilic disorders, autoimmune diseases, and female sex.

previous superficial vein thrombophlebitis (SVT), DVT, or PE (common)

- Commonly reported by patients with SVT.

redness/erythema of overlying skin (common)

- May extend for some distance into the surrounding tissue, making distinction from cellulitis difficult. [Fig-1]

hot/warm overlying skin (common)

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- May extend for some distance into the surrounding tissue, making distinction from cellulitis difficult.

painful/tender over affected vein (common)

- In a patient with pre-existing varicose veins, there is a tender 'worm-like' mass deep to the skin.
- In a patient without varicose veins, a palpable cord, sometimes nodular, may have associated tenderness.

swelling/oedema of surrounding area (common)

- There is often oedema of the surrounding area without generalised swelling of the whole limb.

cord-like mass palpable (common)

- An important sign that can distinguish SVT from other causes of leg swelling and redness. Remains palpable for several weeks to months after initial episode of SVT.

development of symptoms over hours to days (common)

- Symptoms of SVT typically develop over hours to days and resolve in days to weeks.

signs/symptoms of concomitant DVT or PE (common)

- The prevalence of concomitant DVT varies widely in the literature from 2.6% to 65%, and, if present, it is thought to be contiguous in 50% to 75%. Up to 25% of concomitant DVT may not be contiguous with the SVT and may be in the contralateral limb.[32] When the DVT is non-contiguous, the mechanism of DVT occurrence is probably related to a hypercoagulable state. Concomitant PE can also occur in anywhere between 0.5% to 4% of patients with SVT and symptoms of PE. Hence it is important to elicit history for PE symptoms such as dyspnoea, chest pain, and syncope.

Other diagnostic factors

varicose veins (common)

- Present in up to 70% of SVT cases.[13] [35] May be history of local trauma.

recent vein instrumentation (e.g., sclerotherapy) (common)

- SVT following, for example, sclerotherapy is a common and expected complication.

recent vein cannulation and intravenous drug administration (common)

- SVT can occur as a result of cannulation of superficial veins of the upper or lower limbs, and irritation of solutions or drugs delivered through the catheter.

low-grade fever (common)

- May be present in some cases.

pigmentation changes (common)

- Pigmentation changes of the skin overlying the SVT are often observed.

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Diagnostic tests

1st test to order

Test	Result
Doppler ultrasonography • Though the diagnosis of superficial vein thrombophlebitis (SVT) is a clinical one, a Doppler ultrasonography examination of at least the affected leg, and ideally the contralateral leg, should be done in all patients with suspected SVT in order to rule out concomitant DVT and to assess size and location of the thrombus.[44] • Doppler ultrasonography has been shown to be highly sensitive (>95%) and specific (>95%) to diagnose symptomatic and asymptomatic proximal vein DVT.[45] [46] The sensitivity and specificity for SVT is not known. • Ultrasonography may also be used for follow-up to confirm absence of thrombus propagation, especially in patients with poor therapy response.	**lack of compressibility or intraluminal thrombus in the superficial veins**

Other tests to consider

Test	Result
biopsy • Small vessels in the superficial dermis are characteristically spared.[43] • In early lesions, there is a dense inflammatory cell infiltrate mainly composed of neutrophils within the vessel wall. The vein wall appears markedly thickened as a result of exudation of inflammatory cells and oedema. The endothelial cells are swollen. There are thrombi occluding the lumina of the affected veins that eventually undergo recanalisation. • A biopsy should be ordered in SVT cases that are recurrent and/or migratory, and when inflammatory diseases such as polyarteritis nodosum are being considered. This is best accomplished by an elliptical incision transverse to the long axis of the vessel rather than a punch biopsy.	**thrombosis involving large- and medium-sized veins of the upper subcutis and lower dermis**
ventilation perfusion (VQ) scan • In patients with suspected SVT with concomitant respiratory symptoms or signs of PE (chest pain, dyspnoea, syncope), a ventilation perfusion scan or pulmonary CT angiography to look for concomitant PE should be done.	**exclude PE**
pulmonary CT angiography • In patients with suspected SVT with concomitant respiratory symptoms or signs of PE (chest pain, dyspnoea, syncope), a ventilation perfusion scan or pulmonary CT angiography to look for concomitant PE should be done.	**exclude PE**
conventional pulmonary angiography • Indicated if VQ scan or pulmonary CT angiography is non-conclusive and PE is still suspected.	**exclude PE**

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Test	Result
assessment for malignancy • This is indicated in patients with SVT in short vein segments not associated with varicose veins; extensive saphenous vein thrombophlebitis on Doppler ultrasonography, with or without concomitant DVT or PE; or recurrent or idiopathic SVT.	**malignancy**
thrombophilia screening • Screening for thrombophilic disorders may be useful in patients with SVT in short vein segments not associated with varicose veins; in patients with extensive saphenous vein thrombophlebitis on Doppler ultrasonography, with or without concomitant DVT or PE; in patients with recurrent SVT; and in patients with idiopathic SVT. However, the utility and cost-effectiveness of screening for thrombophilic states in patients with SVT has not been well studied.	**presence of factor V Leiden and prothrombin G202010A genetic variants; deficiencies of protein S, C, and antithrombin III; positive lupus anticoagulant and anticardiolipin antibodies; hyperhomocysteinaemia**

Differential diagnosis

Condition	Differentiating signs / symptoms	Differentiating tests
Deep vein thrombosis	• Acute onset of swelling, warmth, redness, and pain that is more diffuse and can involve the whole leg. Palpable cord is not common along the course of affected vein unless there is concomitant superficial vein thrombophlebitis (SVT).	• Doppler ultrasound: thrombus in the deep venous system will confirm diagnosis.
Cellulitis	• Erythema is usually localised and confluent and associated with tenderness and generalised swelling. There may be associated lymphangitis or tender lymphadenopathy, which is not characteristic of SVT. A palpable cord is not present with cellulitis. Systemic findings of fever, chill, and myalgias are more common with cellulitis.	• Doppler ultrasonography: compressible veins, absence of superficial vein thrombus.
Lymphoedema	• Usually chronic rather than acute non-pitting oedema of the extremity and involving the digits. Oedema does not resolve or improve with recumbency.	• Doppler ultrasonography: compressible veins, absence of superficial vein thrombus; B-mode imaging shows subcutaneous oedema.

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Condition	Differentiating signs / symptoms	Differentiating tests
Chronic venous insufficiency	• Usually chronic leg swelling with associated ectatic veins, varicose veins, and skin changes that may include hyperpigmentation, stasis dermatitis, panniculitis, lipodermatosclerosis, and venous ulcers.	• Diagnosis usually made on clinical examination. • Confirmation on Doppler ultrasonography: can show valvular incompetence and chronic venous obstruction.
Erythema nodosum	• Most common form of panniculitis, usually consisting of raised painful bilateral tender lesions that are frequently located over both shins. There is no palpable cord.	• Skin biopsy: septal panniculitis.
Cutaneous polyarteritis nodosa	• A form of vasculitis consisting of nodules of a bright red to blue colour that follow the course of arteries. Usually bilateral and become confluent to form painful subcutaneous plaques.	• Skin biopsy: necrotising vasculitis of the arteries.
Insect stings or bites	• Swelling and redness usually extend over a large area and do not follow the course of a vein. There may be associated pruritus.	• Diagnosis usually made on clinical examination. • Doppler ultrasonography: negative for superficial vein thrombus.
Tendonitis	• Usually pain, mild swelling, and warmth in the area of a tendon (Achilles' heel, patellar). Pain increases with movement of affected joint, and a tendon friction rub may be palpable. Pain worse during and after activity, and the tendon and joint area can become stiff as the swelling impinges on the movement of the joint.	• Ultrasound: thickened, blurred tendon, and possible hypo-echoic foci within tendon. • MRI: tendon injury.
Lymphangitis	• Erythema, warmth, and tenderness along the course of a lymphatic vessel from source of infection (e.g., cellulitis) to regional lymph nodes. Lymph nodes usually palpable.	• Doppler ultrasonography: compressible veins, absence of superficial vein thrombus.

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Step-by-step treatment approach

Superficial vein thrombophlebitis (SVT) has traditionally been considered a relatively benign condition, with conservative management recommended, mainly focusing on relief of local symptoms. However, since the recognition of the important association between SVT and venous thromboembolism (VTE; includes DVT and PE), antithrombotic treatments aimed at preventing thrombus extension and recurrence such as unfractionated heparin (UFH), low molecular weight heparin (LMWH), fondaparinux, and oral anticoagulants have been proposed. Several studies report that, in patients with SVT, especially SVT of the main trunk of the saphenous vein, 6% to 44% of cases are associated with DVT, 20% to 33% are associated with asymptomatic PE, and 2% to 13% are associated with symptomatic PE.[5] [13] [34] [36] [38] [42] [47] [48] [49] Moreover, a few retrospective studies report an increased risk of subsequent VTE complications in patients with untreated SVT (1.7% to 26.9% risk of VTE within 3 months).[7] [38] [47] [50] Therefore, there is growing consensus that the therapeutic approach should aim not only to resolve or improve local symptoms but also to prevent SVT extension and thromboembolic complications.

Histopathological studies of veins with SVT demonstrate changes consistent with inflammation, along with fibrin deposition and thrombus formation.[12] As a result, the mainstay of SVT treatment has traditionally included non-steroidal anti-inflammatory drugs (NSAIDs), although NSAIDs should not be given in combination with anticoagulants due to increased risk of bleeding. For cases with a high risk of thrombus progression into the deep venous system and embolisation, anticoagulants and surgical treatments have been employed to target the coagulation cascade and, in the case of surgical treatment, to interfere with thrombus development.[51]

General approach

Though there is no consensus on how best to approach the treatment of SVT, anticoagulants are increasingly being considered the most effective at treating symptoms and preventing SVT extension, SVT recurrence, and VTE complications. Agents aimed at relieving local symptoms, such as compression stockings and topical treatments, have more of an ancillary role in the treatment of SVT as they have little impact on preventing SVT extension and thromboembolic complications. One guideline has recommended prophylactic anticoagulation for all patients with a superficial thrombus ≥5 cm in length, or within 3 to 5 cm of the saphenofemoral junction.[44] Additionally, an individualised approach is suggested, in which patients with at least one risk factor for VTE may benefit from prophylactic anticoagulation regardless of the length of the thrombus or its proximity to the saphenofemoral junction. This decision should be made through risk-benefit analysis as well as through shared decision making, with respect to patient preference. If the decision is made not to treat a patient with at least one risk factor for VTE, repeat duplex scan 7 to 10 days later is recommended.[44]

NSAIDs alone may be an alternative to LMWH or fondaparinux as they have been shown in a small number of studies to prevent SVT extension when compared with placebo, and to have a similar reduction in the incidence of thromboembolic events as LMWH.[47] [50] [52] However, they should not be prescribed in patients at higher risk of thromboembolism (e.g., extensive SVT with involvement above the knee, particularly if within 2 cm of the saphenofemoral junction; thrombus ≥5 cm in length; severe SVT symptoms; involvement of the greater saphenous vein; history of venous thrombosis or SVT; active cancer) and should not be given in combination with anticoagulants due to increased risk of bleeding.

Surgical interventions with the aim of relieving local symptoms and preventing SVT extension and thromboembolic complications have not been well studied. Saphenofemoral disconnection is considered

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for patients with contraindications to anticoagulant treatment (active bleeding, severe thrombocytopenia) who are at risk of thromboembolic disease due to the location of the thrombus.

Superficial phlebitis with infection, such as phlebitis originating at an intravenous catheter site, is referred to as septic thrombophlebitis. This clinical entity requires therapeutic approaches that are different from those applicable to sterile phlebitis (or SVT). If there is a suspicion of septic (infected) phlebitis or suppurative phlebitis (phlebitis in the setting of bacteraemia), then antibiotics should be considered. Empiric therapy for peripheral vein suppurative thrombophlebitis should include an agent with activity against staphylococci plus an agent with activity against *Enterobacteriaceae* . Antibiotics should be tailored accordingly to culture and sensitivity data when available.

Most of the available evidence for treatment of SVT pertains to thrombosis in the lower extremities. A recent Cochrane review on upper-extremity SVT caused by intravenous catheterisation has concluded that the evidence for treatment of this condition is scarce and low in quality.[53] There are insufficient data to assess the safety and efficacy of topical treatments, systemic anticoagulation, or NSAIDs for upper-extremity SVT.[53]

Anticoagulation with LMWH or UFH

The 2012 American College of Chest Physicians (ACCP) guidelines on anti-thrombotic therapy for VTE disease[54] suggest prophylactic or intermediate doses of LMWH, or prophylactic doses of fondaparinux for 45 days;[51] 1[A]Evidence there is also some weak evidence for intermediate doses of UFH for 45 days for the treatment of SVT.[54] Intermediate doses are larger than prophylactic doses, and smaller than treatment doses. This ACCP guideline was updated in 2016, but the update does not cover prevention of VTE in people with SVT.[55] Other guidelines have indicated that, unless contraindicated, fondaparinux may be preferred to LMWH or UFH.[44] As an alternative to UFH or LMWH, warfarin can be overlapped with 4 days of UFH or LMWH and continued for 45 days. A repeat Doppler ultrasonography 7 to 10 days following start of anti-coagulant therapy may be required to assess for SVT extension, especially in cases of proximal greater saphenous vein SVT treated with intermediate or prophylactic doses of LMWH, prophylactic doses of fondaparinux, or intermediate doses of UFH. If there is extension, then anti-coagulation with warfarin overlapped with 4 days of full-dose (weight-adjusted) LMWH or UFH for at least 3 months is recommended.[54]

In cases where the thrombus is located at <2 cm from the saphenofemoral junction, then warfarin (target INR 2.4; range 2.0-3.0) overlapped with 4 days of treatment-dose (weight-adjusted) LMWH or UFH for at least 3 months may be considered. In patients with SVT and concomitant DVT or PE, first-line therapy includes treatment doses of LMWH, UFH, or fondaparinux followed by warfarin to target INR 2.5. Oral anticoagulants, such as rivaroxaban, dabigatran, or apixaban, can also be used first-line in these patients.

It should be noted that, before the practitioner prescribes anticoagulants, he or she should do a thorough assessment of bleeding risk. In certain cases of heparin therapy, surveillance for heparin-induced thrombocytopenia may be necessary.

Surgical intervention: saphenofemoral junction disconnection (e.g., ligation)

The 2012 ACCP guidelines recommend medical treatment with anticoagulants over surgical treatment.[54] This recommendation is based on a few studies that compared surgical therapy with anticoagulation and showed similar rates of SVT progression but higher rates of complications, such as wound infections, with surgical therapy.[7] However, in cases where the thrombus is located at <2 cm from

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the saphenofemoral junction, or where the thrombus is free-floating in the common femoral vein and there are contraindications to anticoagulation (active bleeding, severe thrombocytopenia), then ligation with or without thrombectomy can be considered. Once there is no longer a contraindication to anticoagulation, LMWH or UFH should be started or resumed in order to prevent thromboembolic complications.

Non-steroidal anti-inflammatory drugs (NSAIDs)

Oral NSAIDs, usually in combination with elastic bandages or compression stockings, can be considered as first-line therapy for SVT that involves tributaries of varicose veins, and in cases where the affected saphenous vein is short in length and away from the saphenofemoral junction. Oral NSAIDs, when compared with placebo, have been shown to help with local symptoms and to prevent SVT extension or recurrence.[47] [56] However, contraindications to NSAIDs (such as peptic ulcer disease) should be considered before they are prescribed, and there should be a follow-up, either clinical or with Doppler ultrasonography, to determine whether there is extension. In addition, NSAIDs should not be given together with, or adjunctive to, systemic anticoagulation therapy as this combination may increase the risk of bleeding. Topical anti-inflammatory agents have been used and may have some effect in alleviation of pain and local inflammatory signs, especially in small SVT of varicose veins, but their use is controversial.[48] [50] [57] [58] [59]

Compression therapy: elastic bandages and compression stockings

Compression therapy is important in the resolution of the SVT and helps relieve local symptoms such as swelling and pain. Compression therapy aims to control venous reflux and peripheral oedema by either active or passive options.

- Inelastic bandages counteract the increase in muscle volume resulting from muscle contraction (i.e., exerts passive compression). At rest the bandages deliver little or no pressure.
- Compression stockings or long stretch bandages provide an active pressure on the limb both at rest and during muscle contraction.

Bandages in the acute phase of SVT can provide relief from symptoms such as itchiness, pain, and swelling, and compression stockings or long stretch bandages can help with resolution of the SVT and prevent chronic swelling. It should be noted that compression therapy has not been shown to prevent SVT extension or thromboembolic complications. A wide range of ready-to-wear stockings is commercially available, enabling a perfect fit in the vast majority of cases. Stockings may also be made to measure. The major limitations to compression therapy are usually poor patient compliance and, in the elderly, difficulty in applying.

An important contraindication to compression therapy includes a systolic arterial pressure at the ankle <80 mmHg or an ankle-brachial pressure index (ABPI) <0.5. An ABPI of 0.5 to 0.8 indicates that arterial disease may be present and that compression may further compromise arterial blood supply. Hence compression stockings should generally be avoided in these cases. Other contraindications to the use of stockings include acute dermatitis, open wounds, and phlegmasia cerulea dolens. Caution is advised in patients with diabetes, neuropathy, skin sensitivities or allergies, and signs of infection.

Local heat and leg elevation

Although there is no evidence for its therapeutic efficacy, local heat application and leg elevation can be used as an ancillary treatment of SVT. Patients should be encouraged to ambulate and to elevate the affected leg when resting. However, these strategies have not been shown to prevent thromboembolic

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complications. They are presumed to be reasonable and inexpensive therapies to recommend to all patients for symptom relief, but they have not been studied in clinical trials.

Topical treatments

A number of randomised trials have evaluated the use of topical therapies in the treatment of SVT, more specifically upper-extremity thrombophlebitis from catheterisation. Several studies have reported significant improvements in signs and symptoms (pain, oedema, erythema) with the use of therapies such as topical heparinoid compared to placebo or no intervention. The level of evidence on such therapies continues to be low given the small sample sizes and questionable methodological quality of these trials.[53] There are currently insufficient data to assess the safety and efficacy of topical treatments in SVT.[53]

Varicose vein surgery

After the acute treatment of SVT of a varicose vein, and in the case of repeated episodes, referral for varicose surgery (ligation and stripping of the affected veins) may be useful. Guidelines for varicose surgery should be followed.

Treatment details overview

Consult your local pharmaceutical database for comprehensive drug information including contraindications, drug interactions, and alternative dosing. (see Disclaimer)

Presumptive (summary)		
Patient group	**Tx line**	**Treatment**
extension into the femoral vein or popliteal vein, or concomitant DVT or PE	**1st**	**management according to venous thromboembolic protocol**

Acute (summary)		
Patient group	**Tx line**	**Treatment**
anticoagulation not contraindicated	**1st**	**anticoagulation**
anticoagulation not contraindicated	**adjunct**	**supportive therapy**
anticoagulation contraindicated	**1st**	**saphenofemoral disconnection**
anticoagulation contraindicated	**adjunct**	**supportive therapy**
anticoagulation contraindicated	**2nd**	**non-steroidal anti-inflammatory drugs (NSAIDs)**

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Acute (summary)

anticoagulation contraindicated	adjunct	supportive therapy
SVT: <5 cm in length on localised venous ectasia (varicophlebitis), not within 3-5 cm of the saphenofemoral junction, no risk factors for VTE	1st	non-steroidal anti-inflammatory drugs (NSAIDs)
	adjunct	supportive therapy
SVT: <5 cm in length on localised venous ectasia (varicophlebitis), not within 3-5 cm of the saphenofemoral junction, at least one risk factor for VTE	1st	assessment of risk-benefit of treatment options with patient
	adjunct	supportive therapy

Ongoing (summary)

Patient group	Tx line	Treatment
recurrent superficial vein thrombophlebitis (SVT) with extensive varicose veins	1st	varicose vein surgery ± prophylactic LMWH

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Treatment options

Presumptive

Patient group	Tx line	Treatment
extension into the femoral vein or popliteal vein, or concomitant DVT or PE	**1st**	**management according to venous thromboembolic protocol** » Must be treated according to local venous thromboembolism (VTE) practice guidelines.[54] [55] » Initial treatment doses of low molecular weight heparin (LMWH), unfractionated heparin (UFH), or fondaparinux followed by warfarin to target INR 2.5. Rivaroxaban, dabigatran, or apixaban can also be used for initial treatment. » Unfractionated heparin is the preferred anticoagulant in patients with renal impairment (i.e., creatinine clearance <30 mL/minute). » The newer anticoagulants (dabigatran, rivaroxaban, and apixaban) are considered suitable alternatives. However, there are no clinical data on their use in the management of SVT. Support for their use in SVT is extrapolated from their use in the management of deep vein thrombosis, and they are used in a similar way to warfarin. Dabigatran requires lead-in therapy with a parenteral anticoagulant such as UFH or a LMWH for 5 to 10 days before starting therapy, whereas rivaroxaban and apixaban can be initiated as monotherapy with no need for lead-in therapy. » Rivaroxaban, dabigatran, and apixaban are not recommended in patients with hepatic or severe renal impairment. » Rivaroxaban, dabigatran, and apixaban have a rapid onset of action, do not undergo any interactions with food, and are short-acting; however, they do undergo drug interactions and have limited reversibility at this time. » Consult specialist or local protocols for guidance on dose. **Primary options** » enoxaparin **-or-** » dalteparin **-or-** » tinzaparin

TREATMENT

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Presumptive

Patient group	Tx line	Treatment
		-or- » fondaparinux **-or-** » heparin **--AND--** » warfarin **OR** **Primary options** » rivaroxaban **OR** **Primary options** » apixaban **OR** **Primary options** » dabigatran

Acute

Patient group	Tx line	Treatment
anticoagulation not contraindicated	**1st**	**anticoagulation** » The 2012 ACCP guideline on antithrombotic therapy for VTE disease recommends prophylactic or intermediate doses of low molecular weight heparin (LMWH), prophylactic doses of fondaparinux, or intermediate doses of unfractionated heparin (UFH) for at least 45 days.[54] 1[A]Evidence A 2016 update of this guideline has been published; however, it does not cover SVT.[55] Intermediate doses are larger than prophylactic doses, and smaller than treatment doses. » Other guidelines have indicated that, unless contraindicated, fondaparinux may be preferred to LMWH or UFH.[44] » As an alternative to UFH or LMWH, warfarin can be overlapped with 4 days of UFH or LMWH and continued for 45 days. LMWH and fondaparinux, when compared with placebo, have been shown to be associated with a decreased risk of SVT extension and recurrence, while effectively controlling local symptoms.[51] Quality of the evidence for comparison of

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Acute

Patient group	Tx line	Treatment

fondaparinux with LMWH is low because there is no direct comparison in patients with SVT.[49]

» In cases where the SVT is located within 2 cm of the saphenofemoral junction, treatment doses of LMWH overlapped with warfarin can be considered. Warfarin dose is adjusted according to INR. Once INR is at least 2 for two days, LMWH can be discontinued.

» The newer anticoagulants (dabigatran, rivaroxaban, and apixaban) are considered suitable alternatives. However, there are no clinical data on their use in the management of SVT. Support for their use in SVT is extrapolated from their use in the management of deep vein thrombosis, and they are used in a similar way to warfarin. Dabigatran requires lead-in therapy with a parenteral anticoagulant such as UFH or a LMWH for 5 to 10 days before starting therapy, whereas rivaroxaban and apixaban can be initiated as monotherapy with no need for lead-in therapy.

» Consult specialist or local protocols for guidance on dose.

Primary options

» fondaparinux

OR

Primary options

» enoxaparin

OR

Primary options

» dalteparin

OR

Primary options

» tinzaparin

OR

Primary options

» heparin

OR

Secondary options

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Acute

Patient group	Tx line	Treatment
		» enoxaparin **-or-** » dalteparin **-or-** » tinzaparin **-or-** » heparin **--AND--** » warfarin **OR** **Secondary options** » rivaroxaban **OR** **Secondary options** » apixaban **OR** **Secondary options** » dabigatran
anticoagulation not contraindicated	**adjunct**	**supportive therapy** » Compression stockings are usually prescribed for 10 to 14 days. Short or long stretch elastic bandages in the acute phase to relieve pain, followed by compression stockings (class I to II: 15-23 mmHg) to help in the resolution of superficial vein thrombophlebitis (SVT), are recommended. They are put on in the morning before getting up and removed in the evening when going to bed. Compression stockings should not be used where the systolic arterial pressure at the ankle is <80 mmHg or the ankle-brachial pressure index (ABPI) is <0.5. » Local heat and leg elevation may help alleviate acute symptoms.
anticoagulation contraindicated	**1st**	**saphenofemoral disconnection** » Where heparin is contraindicated, ligation with or without thrombectomy may be indicated, where local expertise is available. » Even in patients in whom therapeutic anticoagulation for treatment of SVT is contraindicated, prophylactic low molecular weight heparin (LMWH) is nevertheless recommended postoperatively for at least 7 to 10 days to reduce postoperative venous

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Acute

Patient group	Tx line	Treatment
		thromboembolism (VTE). Once there is no longer a contraindication to therapeutic anticoagulation, therapeutic LMWH or unfractionated heparin should be started or resumed, to reduce VTE complications. **Primary options** » enoxaparin: prophylaxis: 40 mg subcutaneously once daily **OR** **Primary options** » dalteparin: prophylaxis: 5000 units subcutaneously once daily **OR** **Primary options** » tinzaparin: prophylaxis: 3500 anti-Xa units subcutaneously once daily
anticoagulation contraindicated	**adjunct**	**supportive therapy** » Compression stockings are usually prescribed for 10 to 14 days. Short or long stretch elastic bandages in the acute phase to relieve pain, followed by compression stockings (class I to II: 15-23 mmHg) to help in the resolution of superficial vein thrombophlebitis (SVT), are recommended. They are put on in the morning before getting up and removed in the evening when going to bed. » Compression stockings should not be used where the systolic arterial pressure at the ankle is <80 mmHg or the ankle-brachial pressure index (ABPI) is <0.5. » Local heat and leg elevation may help alleviate acute symptoms.
anticoagulation contraindicated	**2nd**	**non-steroidal anti-inflammatory drugs (NSAIDs)** » Oral NSAIDs can be used as an alternative to anticoagulants, especially if the superficial vein thrombophlebitis (SVT) is short in length and far removed from the saphenofemoral junction. NSAIDs can alleviate symptoms and may prevent SVT extension and recurrence.[50] [56] However, there are not adequate data to support a decreased incidence of venous thromboembolism with NSAIDs.

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Acute

Patient group	Tx line	Treatment
		» NSAIDs should be considered in those patients in whom there are contraindications to LMWH or UFH. » NSAIDs may be favoured over LMWH in cases where the SVT involves small tributaries of varicose veins, because the risk of extension and recurrence is low. » Duration of NSAID treatment usually consists of a 6- to 10-day trial. » Repeat Doppler ultrasonography is recommended at 7 to 10 days to assess for SVT extension. » Ibuprofen and diclofenac have less risk of gastrointestinal adverse effects compared with other NSAIDs. » NSAIDs should not be given together with, or adjunctive to, systemic anticoagulation therapy as this combination may increase the risk of bleeding. **Primary options** » ibuprofen: 400 mg orally four times daily **OR** **Primary options** » diclofenac: 50 mg orally (immediate-release) two or three times daily **OR** **Primary options** » naproxen: 500 mg orally once or twice daily **OR** **Primary options** » piroxicam: 10 mg orally once or twice daily **OR** **Primary options** » indometacin: 50 mg orally two or three times daily
anticoagulation contraindicated	**adjunct**	**supportive therapy** » Compression stockings are usually prescribed for 10 to 14 days. Short or long stretch elastic

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Acute

Patient group	Tx line	Treatment
		bandages in the acute phase to relieve pain, followed by compression stockings (class I to II: 15-23 mmHg) to help in the resolution of superficial vein thrombophlebitis (SVT), are recommended. They are put on in the morning before getting up and removed in the evening when going to bed. » Compression stockings should not be used where the systolic arterial pressure at the ankle is <80 mmHg or the ankle-brachial pressure index (ABPI) is <0.5. » Local heat and leg elevation may help alleviate acute symptoms.
SVT: <5 cm in length on localised venous ectasia (varicophlebitis), not within 3-5 cm of the saphenofemoral junction, no risk factors for VTE	**1st**	**non-steroidal anti-inflammatory drugs (NSAIDs)** » It is feasible to treat less-extensive SVT with oral and/or topical NSAIDs. Because the risk of extension and recurrence is thought to be highest with SVT of the saphenous veins rather than of small varicose veins, NSAIDs may be favoured over low molecular weight heparin (LMWH) or fondaparinux in cases where the SVT involves small tributaries of varicose veins. » Duration of NSAID treatment usually consists of a 6- to 10-day trial. » Repeat Doppler ultrasonography is recommended at 7 to 10 days to assess for SVT extension and/or progression. » Selective COX-2 inhibitors are not generally recommended because there may be an increased risk of thrombotic events compared with placebo and some NSAIDs. The thrombotic risk, however, is likely to be minimal when treating for a short period of time (6-12 days for SVT). » NSAIDs should not be given together with, or adjunctive to, systemic anticoagulation therapy as this combination may increase the risk of bleeding. **Primary options** » ibuprofen: 400 mg orally four times daily **OR** **Primary options**

TREATMENT

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Acute

Patient group	Tx line	Treatment
		» diclofenac: 50 mg orally (immediate-release) two or three times daily **OR** **Primary options** » naproxen: 500 mg orally once or twice daily **OR** **Primary options** » piroxicam: 10 mg orally once or twice daily **OR** **Primary options** » indometacin: 50 mg orally two or three times daily
	adjunct	**supportive therapy** » Compression stockings are usually prescribed for 10 to 14 days. Short or long stretch elastic bandages in the acute phase to relieve pain, followed by compression stockings (class I to II: 15-23 mmHg) to help in the resolution of SVT, are recommended. They are put on in the morning before getting up and removed in the evening when going to bed. » Compression stockings should not be used where the systolic arterial pressure at the ankle is <80 mmHg or the ankle-brachial pressure index (ABPI) is <0.5. » Local heat and leg elevation may help alleviate acute symptoms. » There are currently insufficient data to assess the safety and efficacy of topical treatments in SVT. » There is low-quality evidence that treatments such as topical heparinoid may help alleviate acute symptoms of catheter-induced upper-extremity SVT.[53] **Primary options** » heparinoid topical: (0.3%) apply to the affected area(s) up to four times daily
SVT: <5 cm in length on localised venous ectasia (varicophlebitis),	**1st**	**assessment of risk-benefit of treatment options with patient**

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Acute

Patient group	Tx line	Treatment
not within 3-5 cm of the saphenofemoral junction, at least one risk factor for VTE		» One guideline indicates that in patients with one or more VTE risk factors, there may be a benefit from treatment with prophylactic anticoagulation with LMWH or fondaparinux.[44] In such patients, a treatment decision can be made based on individualised risk-benefit analysis as well as patient preference. » An alternative choice is treatment with NSAIDs. Selective COX-2 inhibitors are not generally recommended because there may be an increased risk of thrombotic events compared with placebo and some NSAIDs. However, the thrombotic risk is likely to be minimal when treating for a short period of time (6-12 days for SVT). NSAIDs should not be given together with, or adjunctive to, systemic anticoagulation therapy as this combination may increase the risk of bleeding. **Primary options** » enoxaparin: prophylaxis: 40 mg subcutaneously once daily **OR** **Primary options** » dalteparin: prophylaxis: 5000 units subcutaneously once daily **OR** **Primary options** » fondaparinux: prophylaxis: 2.5 mg subcutaneously once daily **OR** **Primary options** » ibuprofen: 400 mg orally four times daily **OR** **Primary options** » diclofenac: 50 mg orally (immediate-release) two or three times daily **OR** **Primary options** » naproxen: 500 mg orally once or twice daily **OR**

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Acute

Patient group	Tx line	Treatment
		Primary options » piroxicam: 10 mg orally once or twice daily **OR** **Primary options** » indometacin: 50 mg orally two or three times daily
	adjunct	**supportive therapy** » Compression stockings are usually prescribed for 10 to 14 days. Short or long stretch elastic bandages in the acute phase to relieve pain, followed by compression stockings (class I to II: 15-23 mmHg) to help in the resolution of SVT, are recommended. They are put on in the morning before getting up and removed in the evening when going to bed. » Compression stockings should not be used where the systolic arterial pressure at the ankle is <80 mmHg or the ankle-brachial pressure index (ABPI) is <0.5. » Local heat and leg elevation may help alleviate acute symptoms.

Ongoing

Patient group	Tx line	Treatment
recurrent superficial vein thrombophlebitis (SVT) with extensive varicose veins	**1st**	**varicose vein surgery ± prophylactic LMWH** » In cases of repeated episodes of SVT in the setting of extensive varicose veins, referral can be considered for varicose vein surgery, such as varicose vein stripping or sclerotherapy. » Varicose vein surgical procedures should be done only after the acute SVT episode to avoid thromboembolic complications induced by such procedures. » Prophylactic low molecular weight heparin therapy should be considered at the time of surgery. **Primary options**

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Ongoing

Patient group	Tx line	Treatment
		» enoxaparin: prophylaxis: 40 mg subcutaneously once daily **OR** **Primary options** » dalteparin: prophylaxis: 5000 units subcutaneously once daily **OR** **Primary options** » tinzaparin: prophylaxis: 3500 anti-Xa units subcutaneously once daily

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Recommendations

Monitoring

Patients with uncomplicated superficial vein thrombophlebitis (SVT) should be followed-up until the SVT resolves and for at least 3 months following treatment to assess for complications such as SVT recurrence or subsequent venous thromboembolism (VTE). Patients with concomitant DVT or PE should be followed until treatment is finished. Treatment duration will no doubt vary according to the risk of DVT or PE recurrence following cessation of anticoagulant treatment. An episode of SVT with DVT provoked by a transient risk factor such as pregnancy requires only 3 months of anticoagulation, given the low risk of recurrence in the year following stoppage of anticoagulation, whereas malignancy-induced SVT with DVT will be likely to require indefinite anticoagulation due to the high risk of recurrence (up to 30% in the year following termination of anticoagulation).

Patients with recurrent SVT should be assessed for varicose veins or underlying prothrombotic or inflammatory conditions.

Patient instructions

Patients with SVT should be advised to continue with normal activities and to keep affected leg raised when sitting or resting. They should be advised to seek medical attention or come back for follow-up if there is no improvement or if there is worsening of leg symptoms. This may suggest extension into the deep venous system. Similarly, new symptoms of dyspnoea or chest pain should prompt immediate medical attention to rule out PE.

Patients should be educated and instructed to seek medical attention with regard to potential complications related to anticoagulant and non-steroidal anti-inflammatory drug (NSAID) therapy, or if there are symptoms or signs of infected thrombophlebitis (e.g., fever, purulent discharge).

Women with SVT secondary to use of oral contraceptives or other hormonal agents should generally be advised to avoid these medicines in the future, though the risk of recurrence with these medicines depends on age and other factors such as smoking.

Patients with SVT in the setting of varicose veins should be informed that there is a high likelihood of recurrence and that surgical intervention for varicose therapy may be warranted with repeated SVT episodes.

Complications

Complications	Timeframe	Likelihood
septic (or suppurative) superficial vein thrombophlebitis	**short term**	**low**

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FOLLOW UP

Complications	Timeframe	Likelihood
Though septic complications are possible, they usually occur in upper extremity SVT due to intravenous catheterisation and/or infusions. In the presence of signs of infection (e.g., fever, purulent discharge, lymphangetic streak), antibiotic treatment is indicated. Empiric antibiotic therapy for peripheral vein suppurative thrombophlebitis should include an agent with activity against staphylococci plus an agent with activity against *Enterobacteriaceae* . Antibiotics should be tailored accordingly to culture and sensitivity data when available. If purulent discharge persists or an abscess is present, then surgical intervention may be needed.		
anticoagulant-associated bleeding	**short term**	**low**
The risk of anticoagulant bleeding varies according to type of anticoagulant, dose of anticoagulant, and patient risk factors. In addition, the definition of major and minor bleeding is not standard across studies and the reported incidence of bleeding in the literature varies. In general, unfractionated heparin (UFH) carries a higher risk of bleeding than low molecular weight heparin (LMWH), and the rate of major haemorrhage with UFH ranges from 0% to 7%. As a result, a thorough assessment of risk factors for bleeding (e.g., active peptic ulcer disease, thrombocytopenia, liver disease, other coagulopathy) must be done prior to any decision to prescribe anticoagulants. The management of heparin-associated bleeding depends upon the location and severity of bleeding, but usually necessitates prompt removal of heparin and may include urgent hospitalisation for red blood cell transfusions and administration of an antidote in the case of UFH.		
heparin-induced thrombocytopenia (HIT)	**short term**	**low**
HIT is a rare but serious complication of patients who are receiving or who have recently received heparin. It is usually manifested by a 50% platelet count drop 5 to 10 days after starting a course of heparin. It is more commonly associated with UFH than LMWH. Thrombosis is an important complication of HIT. Routine platelet count monitoring for HIT is recommended for patients treated with therapeutic doses of UFH and may be appropriate for patients treated with LMWH. An urgent platelet count should be done if symptomatic thrombosis develops in a patient with SVT receiving heparin. If HIT is suspected, referral to a haematologist or thrombosis specialist is recommended.		
NSAID-associated renal failure	**short term**	**low**
NSAIDs can lead to salt and fluid retention and hypertension due to altered renal haemodynamics. They can even cause renal impairment, especially in combination with other nephrotoxic agents. An assessment of concomitant medicines and renal function should be done before prescribing NSAIDs for SVT.		
NSAID-associated gastropathy	**short term**	**low**

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Complications	Timeframe	Likelihood
Common gastrointestinal adverse drug reactions associated with NSAID use include dyspepsia and gastric ulceration and bleeding. Around 10% to 20% of patients experience dyspepsia with NSAIDs. The effects are dose-dependent, and the risk of ulceration increases with duration of therapy and with higher doses. There are also some differences in the propensity of individual agents to cause gastrointestinal adverse effects. Indometacin and piroxicam have the highest prevalence of gastric adverse effects, and ibuprofen and diclofenac appear to have the lowest. It is prudent to use the lowest effective dose for the shortest period of time. Concomitant use of acid-suppressing medicines may prevent or reduce gastrointestinal adverse effects (e.g., proton-pump inhibitors or misoprostol).		
recurrent SVT	**variable**	**medium**
The risk of SVT recurrence ranges between 1.6% to 12.2% in treated patients and 3.3% to 36.7% in untreated patients.[60] The risk varies according to the persistence of underlying risk factors such as varicose veins, malignancy, previous VTE, and/or family history of VTE.[61]		
venous thromboembolism (VTE)	**variable**	**low**
In patients with superficial vein thrombophlebitis (SVT) treated with anticoagulation or non-steroidal anti-inflammatory drugs (NSAIDs), the risk for subsequent DVT or PE is about 0% to 5.5%.[60] A few studies report an incidence of subsequent VTE of 1.7% to 26.9% for patients who are not treated pharmacologically or surgically during a follow-up of 3 months.[7] [38] [47] [50] The risk of subsequent DVT or PE is dependent on factors such as a prior history of VTE.		

Prognosis

Uncomplicated superficial vein thrombophlebitis (SVT) is generally considered to be a benign and self-limiting condition. Not much is known about its natural course and prognosis except that symptoms generally subside in 1 to 2 weeks, though hardness of the vein may persist for longer. If, however, there is concomitant venous thromboembolism (VTE), such as DVT or PE, then the course may be more prolonged and anticoagulation may be required for more than 3 months. In addition, complications of DVT or PE include VTE recurrence, as well as chronic venous insufficiency and post-thrombotic syndrome in the case of DVT.

In patients with varicose veins or other risk factors such as malignancy or Behcet's disease, risk of SVT recurrence is not negligible. Depending on the presence or absence of underlying risk factors such as varicose veins, malignancy, previous VTE, and/or family history of VTE, the risk of SVT recurrence ranges anywhere between 1.6% to 12.2% in treated patients and 3.3% to 36.7% in untreated patients.[60] [61] Moreover, there is a risk for subsequent DVT or PE in about 0% to 5.5% of patients with SVT treated with anticoagulation or non-steroidal anti-inflammatory drugs (NSAID).[60] In the case of recurrent SVT in the setting of varicose veins, surgical interventions such as sclerotherapy can be considered to decrease the risk of recurrence.

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Diagnostic guidelines

Europe

Superficial thrombophlebitis of the lower limb: practical recommendations for diagnosis and treatment

Published by: Thrombosis Guidelines Group of the Belgian Society on Thrombosis and Haemostasis; Belgian Working Group on Angiology **Last published:** 2005

Summary: Duplex ultrasonography is recommended in all cases to assess extent of superficial vein thrombophlebitis (SVT) and any associated DVT. Underlying predisposing factors should be sought in patients without varicose veins.

Treatment guidelines

Europe

Superficial thrombophlebitis of the lower limb: practical recommendations for diagnosis and treatment

Published by: Thrombosis Guidelines Group of the Belgian Society on Thrombosis and Haemostasis; Belgian Working Group on Angiology **Last published:** 2005

Summary: Most patients require only local treatment and do not need anticoagulation. Mobilisation with elastic compression should be done immediately in all cases. Extensive superficial vein thrombophlebitis (SVT) involving the main trunk of the great saphenous vein or the small saphenous vein should be treated with surgery or anticoagulation.

North America

Antithrombotic therapy for VTE disease: CHEST guideline and expert panel report

Published by: American College of Chest Physicians **Last published:** 2016

Summary: Updates the recommendations on 12 of the topics that were in the 9th edition of these guidelines, and addresses 3 new topics. Unlike the earlier 2012 version, this guideline does not specifically cover SVT.

Guidelines for management of superficial vein thrombosis

Published by: University of Washington School of Medicine, Anticoagulation Services **Last published:** 2015

Summary: Recommendations for the management of SVT, presented in a flow diagram format.

Antithrombotic therapy for VTE disease

Published by: American College of Chest Physicians **Last published:** 2012

Summary: Suggests prophylactic doses of low molecular weight heparin (LMWH) or fondaparinux for 45 days rather than no anticoagulation.

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Evidence scores

1. Symptom improvement: there is good-quality evidence in the form of a trial of 3002 patients with superficial vein thrombophlebitis (SVT) and without concomitant DVT or PE to show that fondaparinux 2.5 mg daily for 45 days versus placebo is effective in the treatment of patients with acute SVT (relative risk reduction 85%, 95% CI: 74-92).[49] The primary outcome was a composite of death, symptomatic PE, symptomatic DVT, or symptomatic extension to the saphenofemoral junction, or symptomatic recurrence of SVT at day 47. There was no difference in bleeding between the 2 groups. **Evidence level A:** Systematic reviews (SRs) or randomized controlled trials (RCTs) of >200 participants.

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Key articles

- Di Nisio M, Wichers IM, Middeldorp S. Treatment for superficial thrombophlebitis of the leg. Cochrane Database Syst Rev. 2013;(4):CD004982. Full text Abstract
- Kearon C, Akl EA, Comerota AJ, et al. Antithrombotic therapy for VTE disease: antithrombotic therapy and prevention of thrombosis. 9th ed. American College of Chest Physicians evidence-based clinical practice guidelines. Chest. 2012;141(suppl 2):e419S-e494S. Full text Abstract

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Images

Figure 1: Greater saphenous vein superficial vein thrombophlebitis

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