Treating Superficial Venous Thrombophlebitis

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Abstract
Superficial venous thrombophlebitis (SVT) is characterized as a localized inflammatory condition of the venous vessels underlying the skin. It arises from thrombosis of a superficial vein, and clinical presentation usually involves pain, erythema, and tenderness at the sites of inflammation. Although the condition is usually self-limited and not serious or fatal, symptomatic superficial thrombophlebitis can be debilitating, limit movement and certain capabilities, or progress to involve the deep venous system and cause pulmonary embolism. SVT is typically associated with venous valvular insufficiency, pregnancy, infection, and prothrombotic conditions, including malignancy. Currently, medical therapies comprising bedrest, elastic stockings, compression bandages, nonsteroidal anti-inflammatory drugs, and low molecular weight heparins are used to reduce the extension of inflammation and recurrence of thrombotic events in patients experiencing SVT. In patients refractory to conservative measures, surgical interventions such as phlebectomy, sclerotherapy, saphenous junction ligation, or saphenous vein stripping are potential treatments. (JWCCN 2008;6:760–765)

Superficial venous thrombophlebitis (SVT) is a localized venous disorder, defined as an inflammatory reaction with potential subsequent thrombus formation within a superficial vein underlying the skin. It is important for clinicians to distinguish between SVT and deep venous thrombosis (DVT) because they have differing treatment algorithms and long-term sequelae, although these conditions can occur simultaneously and often individually predispose the patient to the other condition.

As the name implies, DVT in the lower extremities refers to the deep venous vessels, including the iliac, common femoral, superficial femoral (now recommended to be referred to as the femoral vein to avoid confusion in the nomenclature), popliteal, and tibial veins. DVTs typically are asymptomatic, except for swelling, and usually do not present with inflammation. SVT in the lower extremity predominantly affects the saphenous veins, and is more often symptomatic (tenderness, pain, or erythema) and occurs mostly on the superficial venous vessels visible on the skin surface.

Although SVT has an incidence of 125,000 cases per year in the United States, the exact incidence of spontaneous SVT is unknown and probably underreported. There is a fourfold increased incidence from the third to eighth decade in men, and a preponderance among women in their fifth decade to 70%. Mean age of patients with SVT is typically 60 years, but because SVT often resolves independently without intervention or physician treatment, the condition is likely underrecognized and therefore undertreated. This article reviews the etiology, diagnosis, and pharmacologic and surgical treatment strategies of SVT.

Etiology
SVT can occur in essentially all parts of the body but is seen mostly in the lower extremities and external jugular veins. The anatomic distribution of SVT in the lower extremities is mostly found in the great saphenous vein (60%–80%) and small saphenous vein (10%–20%), and is bilateral in 5% to 10% of cases. The pathophysiology of SVT is believed to be associated with dysfunction leading to Virchow’s triad: 1) intimal damage (trauma, infection, inflammation, etc.), 2) venous stasis, and 3) a state of hypercoagulability. Anatomic factors, including underlying venous reflux, contribute to the development of SVT. In 70% of cases of SVT, superficial venous reflux is identified on duplex.1
Many clinical factors predispose individuals to SVT (Table 1), including conditions of increased risk for thrombosis, namely pregnancy, hormone replacement therapy, catheter-related infections, venous stasis, obesity, history of previous thromboembolism, and drug abuse. During pregnancy, the incidence of SVT increases by as much as 48-fold. The incidence is higher for women in pregnancy and the postpartum period, with a range of 0.68 to 12 per 1000 deliveries. Strenuous exercise or trauma can sometimes be implicated as the cause of SVT, with placement of an intravenous catheter the most common iatrogenic cause in patients with cancer.

In many cases of SVT, however, no inciting event can be identified. Considering certain clinical scenarios is still helpful to understand the most common clinical presentations in patients with SVT. Traumatic thrombophlebitis occurs typically after direct injury, often to an extremity. Echymosis is often present, indicating some amount of extravasation that has occurred because of vein injury. Palpation of a tender cord along the course of the affected vein is often seen in hospitalized patients with extended placements of intravenous catheters. Chemotherapeutic infusions further increase the likelihood of SVT, especially when constituted into hypo- or hypertonic solutions. The thrombotic vein segment can remain a palpable cord or mass for weeks after infusion cessation or catheter removal.

SVT frequently is found in patients with varicose veins and has been implicated in the progression to DVT. Varicose veins arise from saphenous vein reflux and insufficiency, and if thrombosis occurs within the affected segment, the length of the saphenous vein can be extended. If the thrombosis is extensive, it can affect the deep system from communications throughout the leg or at the saphenofemoral junction. The common etiologic mechanism of stasis in SVT and DVT contributes to this association, and certainly the association of varicosities and SVT highlights this relationship. SVT in this clinical scenario presents as a tender, hard nodule surrounded by erythema overlying a previously noted varicosity. Bleeding can also occur in this region if the inflammatory reaction extends through the vein wall and skin, and typically occurs at ankle level.

SVT can present as a more serious infection, and septic phlebitis is almost always associated with intravenous catheterization. Aerobic, anaerobic, and mixed infections are seen with organisms such as Staphylococcus aureus, Pseudomonas, Klebsiella, Peptostreptococcus, Propionibacterium, Bacteroides fragilis, Prevotella, and Fusobacterium all being implicated. Unexplained fevers in the extremes of ages, such as elderly patients and neonates, should suggest the possibility of supplicative SVT.

Migratory thrombophlebitis deserves special consideration because of its association with malignancy. This was first reported by Trousseau in 1856 and is particularly prevalent in patients with adenocarcinoma of the tail of the pancreas. Migratory SVT is also noted in vasculitides, such as polyanarteritis nodosa, erythema nodosum, erythema induratum, Buerger's disease, and Behçet's syndrome. When located on the breast, SVT is known as Mondor's disease, and on rare occasions is discovered with an associated breast cancer, particularly when the episode of SVT is recalcitrant or recurrent. Mondor's disease can occur after breast surgery, with the use of birth control pills, and with hypercoagulable disorders, such as protein C deficiency and anticardiolipin antibodies.

Although it may seem intuitive that cancer patients should have a high rate of SVT because of all the aforementioned etiologic associations, compared with DVT few data examine the association. Suspicion for an underlying malignancy or hypercoagulability should be considered if an episode of SVT is recurrent, difficult to treat, migratory, aggressive, or without evidence of underlying reflux. Increased tissue factor production as in Trousseau's syndrome in adenocarcinomas is highly associated with superficial thrombophlebitis. Genetic thrombophilic and hypercoagulable states (e.g., factor V Leiden, antithrombin deficiency, and anticardiolipin antibodies are also implicated.

Table 1 Risk Factors Associated With Superficial Thrombophlebitis

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<th>Risk Factors</th>
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<tr>
<td>Pregnancy</td>
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<td>Oral contraceptives</td>
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<td>Prothrombotic conditions (i.e., factor V Leiden,</td>
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<td>antithrombin deficiency)</td>
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<td>Central venous catheterization–associated infection</td>
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<td>Drug-induced thrombophlebitis</td>
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<td>Venous stasis, immobility</td>
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<tr>
<td>Obesity</td>
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<td>Varicose veins</td>
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<tr>
<td>Malignancy</td>
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antithrombin III deficiency) also have increased risk for developing superficial thrombophlebitis. Martinelli et al.\textsuperscript{7} reported that among 63 patients diagnosed with SVT of the lower extremities as a first thrombotic episode, prevalence of several thrombophilic and hypercoagulable states was higher than in the control population. Furthermore, in a study of 45 patients, de Godoy et al.\textsuperscript{14} showed that those with 2 or more episodes of lower extremity SVT were more likely to have antibodies directed toward antidiolipin than controls.

**Diagnosis**

A positive diagnosis of SVT is made mostly on clinical grounds alone. Symptomatic presentation occurs over hours to days and often spontaneously resolves within days to weeks of initial presentation. Patients will present with tenderness, pain, and erythema, which courses along the affected vein. A nodular mass may often be palpated, indicating the presence of a thrombus. This palpable nodularity sometimes persists from a few weeks to months after the episode of superficial thrombophlebitis resolves.\textsuperscript{1} Later in the course of SVT, as the induration subsides, the erythema gives way to a ruddy or bruised color.

Constitutional symptoms may also be present, including low-grade fevers or muscle aching. Infection rarely complicates SVT, and therefore antibiotics are not indicated unless septic or supplicative thrombophlebitis is suspected. These patients will have higher-grade fevers, pus expressed from the wound, or an abscess identified. The differential diagnosis for SVT includes cellulitis, lymphangitis, panniculitis, erythema nodosum, and insect bites.

The association between infected central venous catheters and septic SVT is important when treating cancer patients. The incidence of septic thrombophlebitis increases when indwelling peripheral venous catheters remain for more than 2 to 3 days.\textsuperscript{11} Many advocate routine replacement of adult peripheral catheters every 72 to 96 hours.\textsuperscript{12} Moreover, bacterial infection may originate from normal flora of the skin, migrating along the catheter to the vascular space. Sources for the contamination include intravenous fluids or hematogenous spread from distant sites.\textsuperscript{6} Postsurgical patients presenting with SVT often present secondary to infectious causes at the site of the postoperative wound.

Although SVT is diagnosed mostly on clinical grounds, radiographic confirmatory studies for SVT are usually indicated and consist of a duplex ultrasound of the affected region. Because the clinical signs and symptoms of inflammation can often lag behind the extension of thrombus by several inches, a duplex study can determine the extent of thrombus in the superficial vein and also provide critical information about possible extension into the deep system. This assessment is obviously more important in cases of close proximity to the saphenofemoral or saphenopopliteal junctions, so that an additional diagnosis of DVT can be made and long-term therapies changed. Systemic lung scanning has identified perfusion defects in up to 33% of patients with SVT, again highlighting the link among SVT, DVT, and pulmonary embolism.\textsuperscript{13} The risk for DVT in patients presenting with SVT is reported to be wide (0.75%–40%) and likely reflects the heterogeneous nature of the patients who develop SVT. Lutter et al.\textsuperscript{14} reported 12% of 186 patients with SVT of the above-knee greater saphenous vein to have deep venous extension on duplex. A review by Sullivan et al.\textsuperscript{15} of 6 series of above-knee SVTs noted extension of SVT into the deep system in 3.4% of patients treated surgically and 2.2% treated medically. Future research is necessary to help elucidate which patients are at high risk for SVT and should be studied more carefully, whether through initial duplex or at follow-up during therapy. Idiopathic or spontaneous SVT should probably be screened with duplex for associated underlying DVT.

**Treatment**

Treatment of SVT depends on the clinical presentation and extent and severity of symptoms (Table 2). In the absence of deep vein thrombosis, SVT has no emergent or severe associations. The mainstay of treatment centers on use of nonsteroidal anti-inflammatory drugs (NSAIDs) and elastic stocking compression therapy to alleviate the inflammatory reaction, which typically lasts 7 to 21 days. Thrombectomy (and phlebectomy) is performed infrequently and reserved for patients with SVT uncontrolled by pharmacotherapy or those with severe infection.

**Pharmacotherapy**

**NSAIDs:** NSAIDs show significant efficacy in reducing superficial thrombophlebitis, progression of inflammation, and recurrence of SVT by two thirds.
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Table 2 Treatment Options for Superficial Venous Thrombophlebitis*

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<tr>
<th>Medication</th>
<th>Interventions</th>
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<tr>
<td>Low molecular weight heparin</td>
<td>Surgery: saphenofemoral venous ligation</td>
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<td></td>
<td>Surgery: saphenofemoral venous stripping</td>
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<tr>
<td>Nonsteroidal anti-inflammatory drugs</td>
<td>Surgery: thrombectomy</td>
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<tr>
<td></td>
<td>Conventional sclerotherapy</td>
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<td></td>
<td>Foam sclerotherapy</td>
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*Various treatment options are available for treatment of symptoms associated with superficial thrombophlebitis. Medication is generally efficacious in alleviating the symptoms and in aiding with the resolution of the acute condition. Various procedures are also available for patients presenting with a set of symptoms that cannot be ameliorated with the use of nonsteroidal anti-inflammatory drugs or low molecular weight heparins.

Follow-up was planned at 6 months, with duplex surveillance at 3 and 6 months. Patients treated with stockings alone and those undergoing simple ligation had a higher rate of thrombus extension on follow-up duplex. Regarding development of venous thromboembolism, a trend favored patients receiving anticoagulation. No side effects of treatment (major bleeding, HIT, postsurgical complications) or deaths were reported. Regarding cost (direct and indirect), warfarin, unfractionated heparin, and elastic compression therapies were associated with the lowest cost, whereas the LMWH group incurred the highest cost. The use of LMWH for 4 weeks in patients with proximal great saphenous vein SVT received a grade 2B recommendation from the 7th American College of Chest Physicians (ACCP) Conference on Antithrombotic Therapy.18

Surgical Therapy

For patients with symptomatic veins and substantial venous incompetence, surgery is the optimal treatment. Saphenofemoral ligation with stripping of the long saphenous vein and phlebectomies is the preferred procedure, after which the patient requires no further intervention.19 LMWH and surgical saphenofemoral disconnection both showed efficacy in treating symptomatic superficial thrombophlebitis; however, surgery was associated with statistically insignificant greater reduction of extension or recurrence of inflammation.20

Surgical procedures paired with ancillary treatment also seem useful in treating symptomatic SVT. Thrombectomy plus elastic stockings improves clinical symptoms and reduces the recurrence of venous thrombotic events compared with compression bandage alone.17 Moreover, venous ligation plus elastic stockings was associated with an insignificant reduction in thrombotic events, but reduced recurrence of superficial thrombophlebitis and its inflammatory extension.

Sclerotherapy

Conventional sclerotherapy involves injecting a sclerosing agent (e.g., sodium tetradecyl or polidocanol) into varicosities, followed by a period of compression bandaging or elastic stockings.21 The main risk for sclerotherapy is local necrosis and scarring. Unfortunately, conventional sclerotherapy is effective only for small varicose veins and is short-acting when saphenofemoral reflux is present. Foam sclerotherapy involves adding air to the sclerosing agent to rapidly treat superficial thrombophlebitis. A recent randomized, controlled study evaluated the efficacy and safety of foam sclerotherapy versus surgical saphenofemoral ligation in patients with symptomatic veins and substantial venous incompetence.
trial found that foam treatment had short-term advantages compared with conventional surgery, although this practice is still in its infancy. 22

**Therapeutic Recommendations**

The treatment algorithms for SVT depend highly on the clinical presentation. For highly localized and mildly tender superficial thrombophlebitis involving a varicose vein cluster that is well away from the main saphenous trunk, treatment with an NSAID and elastic support suffices. No need for immobilization exists, and patients can expect recovery in the short-term. For patients with more prominent localized varicosities or if symptoms persist, phlebectomy and removal of the affected segment can hasten recovery.

More severe cases of SVT not involving the deep system, which present with more aggressive symptoms of severe pain, erythema, and diffuse brawny induration, are treated initially with bedrest, elevation of the affected extremity, and application of warm compresses. As ambulation becomes possible after some improvement in the pain, elastic stockings are used. If infection is a component, then antibiotic therapy is indicated and surgical removal of the affected vein segment is considered. If the proximal greater saphenous vein is involved, then a short course of a LMWH can improve recovery time and potentially decrease long-term thromboembolic extension or deep system involvement.

An important aspect to the medical treatments described is follow-up surveillance with duplex. When cessation of symptom improvement or evidence of ascending superficial thrombophlebitis occurs during medical therapy, surgical therapy is considered. Operative intervention involves ligation of the saphenofemoral junction with or without stripping of the greater saphenous vein (for those with venous insufficiency), and provides good long-term recovery from symptoms and prevention of future thromboembolic consequences. For patients who are more immobile or at high risk for surgery, long-term anticoagulation with LMWH is a safe alternative. Again, any evidence of extension into the deep system during follow-up converts the algorithm to one for treating a DVT, with its typical recommendation for at least 6 months of anticoagulation. A very individualized treatment course should be considered for all patients with SVT because of the heterogeneous nature of presentation, symptomatology, and response to medical and surgical therapy.

**Conclusions**

In the absence of obvious infection, SVT treatment should be symptomatic relief with NSAIDs to limit the extension of inflammation to the affected vein. LMWHs can be administered to prevent thromboembolic complications in patients with proximal saphenous vein involvement. However, the population at high-risk and optimal duration of LMWH therapy remain to be elucidated. Surgery for SVT should be reserved acutely for infection and chronically for extension of the clot near the saphenofemoral junction, and for those who are unreliable for anticoagulation, have undergone failed anticoagulation treatment, or experience symptomatic reflux in the superficial vein after resolution of the acute thrombotic event.

**References**

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