Superficial vein Thrombophlebitis

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Superficial thrombophlebitis is a **common inflammatory-thrombotic disorder** in which a thrombus develops in a vein located near the surface of the skin **(Markovic et al., 1997)**

Most superficial veins that develop thrombosis also have phlebitis, in contrast to deep venous thrombosis (DVT), a sometimes asymptomatic condition in which phlebitis may be absent

Although superficial thrombophlebitis usually occurs in the lower extremities **(saphenous vein of the leg)**, it also has been described in the penis and the breast **(Mondor disease) (Nazir and Khan.2010)**

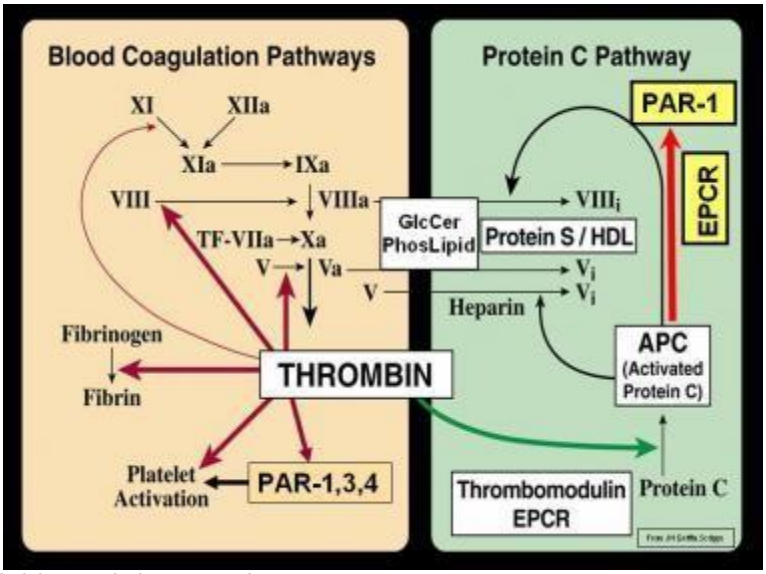
It can also develop anywhere that medical interventions occur, as in the arm or neck (external jugular vein) when intravenous (IV) catheters are used. **(Kagel and Rayan.2004)**

Thrombosis of superficial veins has long been regarded as a benign disorder and a selflimiting, however, it can be complicated by deep vein thrombosis **(DVT)** and even pulmonary embolism **(PE).**

65.6% of patients who presented with great saphenous vein thrombosis were found to have associated DVT **(Ascher et al., 2003)** .

Pathophysiology

Vascular endothelial injury as triggering agent result in thrombus formation. This injury initiates an inflammatory response that results in immediate platelet adhesion at the injury site. Further platelet aggregation is mediated by thromboxane **A2 (TxA2)** and by **thrombin**.



Fig(1)coagulation cascade

Platelet aggregation due to TxA2 is inhibited irreversibly by aspirin and reversibly by other nonsteroidal anti-inflammatory drugs (NSAIDs); thrombin-mediated platelet aggregation, on the other hand, is not affected by NSAIDs, including aspirin.

Contributing Risk factors include,

Varicose veins, Obesity, Age older than 60 , Cigarette smoking ,IV drug abus, Hypercoagulable states **(Franchini et al.,2006)**

Defects responsible for hypercoagulability Table(1)

Activated protein C resistance (factor V Leiden) Protein C deficiency Antithrombin deficiency Hyperhomocysteinemia Prothrombin 20210A allele Dysplasminogenemia High plasminogen activator inhibitor Dysfibrinogenemia Elevated factor VIII Antiphospholipid syndrome	Thrombocythemia Dysproteinemia Heparin-induced thrombocytopenia Estrogens Birth control pills Hormone replacement therapy Malignancy Pregnancy Bed rest Surgey .Trauma

Disorder	*Gene frequency*	*Cause of hypercoagulability*
APC resistance	3.6%-6.0%	10%-64%
Protein S deficiency	0.50%	1.4%-7.5%
Protein C deficiency	0.33%	1.4%-8.6%
Antithrombin deficiency	0.10%	0.5%-4.9%
Prothrombin 20210A	0.7%-6.0%	5.0%-7.1%

Table (2) Frequency of Inherited Defects and Hypercoagulability (Ames etal., 1998)

Systemic lupus erythematosus, Acquired immunodeficiency syndrome (AIDS) - Lupus anticoagulant, Drug-induced lupus anticoagulant, Behçet disease, Blood type A, hemolytic anemia **(Whitlatch and Ortel.2008)**

Phlebitis occurs in diseases associated with vasculitis, such as polyarteritis nodosa (periarteritis nodosa) and Buerger disease (thromboangitis obliterans) in which there was phlebitis in **8 of 19** patients, and in **43%** of the **255** patients of buerger disease**. (Shionoya.1990)**

Pregnancy, throughout and after 6 weeks of delivery, due to increase in most procoagulant factors and a reduction in fibrinolytic activity occur. Plasma fibrinogen levels gradually increase after the third month of pregnancy, reaching

double the normal state. In the second half of pregnancy, levels of factors VII, VIII, IX, and X also increase **(Pomp et al., 2008)**

Also those who carry the **factor V Leiden** or **prothrombin C-20210-a gene**, because they already have a predisposition to clotting, which would also be exacerbated by pregnancy.

High-estrogen oral contraceptives, a woman may increase her risk of thrombosis by a factor of 3-12 times, Newer low-dose oral contraceptives are associated with a much lower risk of thrombophlebitis **(Rosendaal et al.,2001).**

Thrombophlebitis in **varicose vein** may follow trauma to a varix, or without an antecedent cause. Bleeding may occur as the reaction extends through the vein wall. it is observed in varicose veins surrounding venous stasis ulcers.

Infection-related thrombophlebitis following operations or after injection treatments, trauma, or exposure to radiation therapy **(Mermel et al., 2009)**

In case of hypercoagulability in association with malignancies, with the classic example being Trousseau syndrome—a thrombotic event occurring prior to an occult malignancy, The pathophysiology of malignancy-related thrombosis is poorly understood, but tissue factor, tumor-associated cysteine proteinase, circulating mucin molecules, and tumor hypoxemia have all been implicated as causative factors ***(Varki, 2007)***

Patient complain of tenderness, induration, pain and/or erythema along the course of a superficial vein usually establish a clinical diagnosis, especially in patients with known risk factors ***(Fernandez et al., 2010).***

On physical examination, the skin over the affected vein exhibits erythema, warmth, swelling, and tenderness. Later in the disease, as induration subsides, erythema gives way to a ruddy or bruised color ***(Karwowski et al., 2013)***

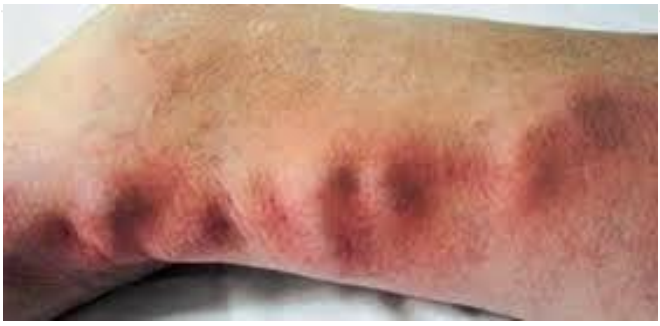


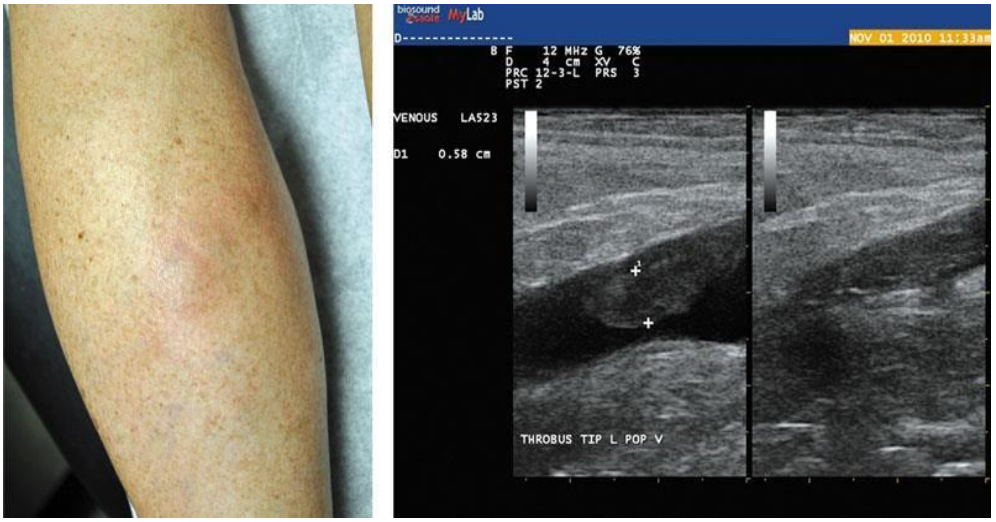
fig (2) superficial thrombophlebitis of the great saphenous vein

In addition, there is a palpable, sometimes nodular cord, due to thrombus within the affected vein. Persistence of this cord when the extremity is raised suggests the presence of thrombus **(Fernandez et al.,2010)**

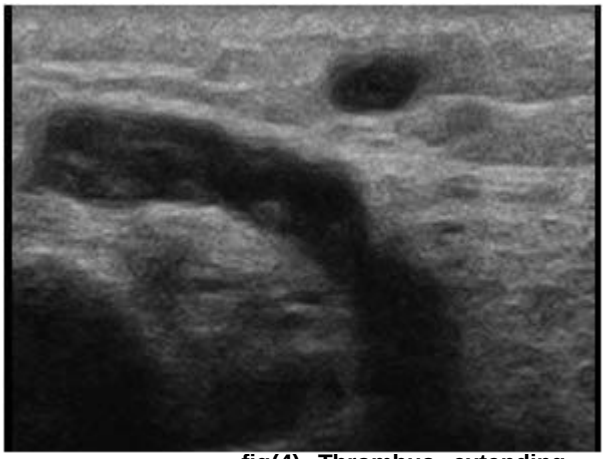
Patient characteristics and **predisposing factors** for thrombophlebitis nearly mirror those for DVT; thrombophlebitis is a risk factor for the development of DVT, and vice versa.

Lower extremity superficial phlebitis is associated with conditions that increase the risk of thrombosis, including abnormalities of coagulation or fibrinolysis, endothelial dysfunction, infection, venous stasis, intravenous therapy and intravenous drug abuse which are components of **virchows triad (Fernandez et al., 2012)**

Duplex ultrasound identifies the presence, location and extent of venous thrombosis, also may identify the pathology that may be a source of the patient's complaints.



Fig(3)



fig(4) Thrombus extending in the proliferating vein.

Ultrasound is indicated if superficial phlebitis involves or extends into the proximal onethird of the medial thigh otherwise clinical diagnosis will be enough **(Fernandez et al., 2010).**

Current treatment options are aimed at resolving symptoms, preventing recurrence and preventing extension to the deep venous system, for potentiality of thromboembolism.

Treatment with **compression stockings** should be offered to patients with **lower extremity superficial phlebitis**, if not contraindicated (e.g., peripheral artery disease).

Gradient compression stockings are adjunctive therapy that is both benign and effective. They provide a gradient of compression that is highest at the toes (at least **30-40 mm Hg)** and gradually decreases to the level of the thigh. Which reduces capacitive venous volume by approximately **70%** and increases the measured velocity of blood flow in the deep veins by a factor of **5 or more**.

Gradient compression hose also have been shown to increase local and regional intrinsic **fibrinolytic activity**. Patients may find them helpful for reducing swelling and pain once the acute inflammation subsides.

Non-steroidal anti-inflammatory drugs (NSAID) are effective in relieving the pain associated with venous inflammation and were found in a randomized trial to significantly decrease extension and/or the recurrence of superficial vein thrombosis. **(Arch Intern Med. 2003)**

Also (NSAIDs) and low-molecular-weight heparin (LMWH) are known to be the first options **(Di Nisio et al., 2007)**

Anticoagulation for patients with lower extremity superficial thrombophlebitis at increased risk for thromboembolism (affected venous segment of ≥5 cm, in proximity to deep venous system, positive medical risk factors **(Kearon et al., 2008)**

Treatment with **fondaparinux** (inhibitor factor X, long acting, once daily) reduces the risk of subsequent venous thromboembolism **(Di Nisio et al., 2012)**

Also when compared with placebo **fondaparinux** found to be a good option in those patients as regard rate of recurrence and reducing symptoms and extension of SVT. **(Di Nisio et al., 2013)**

In a small, randomized trial of 60 patients with great saphenous vein thrombosis, **Lozano et al** compared treatment using LMWH with surgical saphenous ligation, Patients in the LMWH group experienced no episodes of **DVT** or **PE** but had a **10%** incidence of recurrent superficial venous thrombosis.

Among the patients treated surgically, two pulmonary emboli **(6.7%)** were found, and one episode of recurrent superficial venous thrombosis **(3.3%)** occurred **(Lozano et al., 2003)**

Surgical intervention is reserved for extension of the clot to within 1 cm of the saphenofemoral junction or in patients unfit for anticoagulation, or failure of anticoagulation and patients with intense pain **(Karwowski et al., 2013)**

Surgical therapy with ligation of saphenofemoral junction or stripping of thrombosed superficial veins appears to be associated higher rates of venous thromboembolism compared with treatment with anticoagulants **(Belcaro et al., 1999)**

The thrombophlebitic vein may be excised in Patients who demonstrate signs and symptoms of septic thrombophlebitis require urgent **venous excision** to control the septic focus with removal of the infected thrombosed vein **(Quenet et al., 2003).**

Complications recorded include superficial vein thrombosis extension to the deep vein system and/or recurrence of SVT.

Supportive thrombophlebitis is suspected when erythema extends significantly beyond the margin of the vein and is associated with significant fever. If

suspected, antibiotic treatment, surgical drainage and potentially vein excision are indicated (**Davidovic et al., 1990)**

Venous thromboembolism can occur with superficial vein thrombosis. Estimates of the percentage of patients with SVT who also have **DVT** vary between **6% and 53%,** and symptomatic **pulmonary embolism** has been reported in **0% to 10%** of patients **wi**th SVT. **(Decousus et al., 2010)**

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