



Inpatient Management of Thrombophlebitis: A Collaborative Approach Between Internal Medicine and Surgery with Emphasis on Pharmacologic Therapy

Aditya Pal Singh Lubana¹, Ericka Nadeska Rodgers², Yalemwork M Gethenh³, Emmanuel Adjei-Wiafe⁴, Fátima Merino-Magana⁵, Sunil Kumar Yadav⁶, Shravya Subashini Sivaramakrishnan⁷, Mohammed Adnan Ali Kassis⁸, Yousra Merdjana⁹, Maria Isabel Gomez-Coral^{10*}

¹Government Medical College, Amritsar, India

²Universidad Nacional Autónoma De Honduras, Honduras

³VHC Health, Arlington, VA, USA

⁴University of Ghana Medical School, Accra, Ghana

⁵Universidad Salvadoreña Alberto Masferrer, El Salvador

⁶Institute of Applied Health Sciences, Chittagong, Bangladesh

⁷Gandhi Medical College, Secunderabad, India

⁸Rakmhsu, Rak, UAE

⁹Medical Institute of Tambov State University named after G.R. Derzhavin, USA

¹⁰Universidad del Valle, México

Submission: April 23, 2024; **Published:** May 02, 2025

***Corresponding author:** Maria Isabel Gomez-Coral, Universidad del Valle, México

Abstract

Thrombophlebitis, encompassing both superficial thrombophlebitis (STP) and deep vein thrombosis (DVT), is a significant vascular disorder encountered in inpatient settings, often requiring multidisciplinary management. The clinical presentation varies based on location and severity, ranging from localized tenderness in STP to limb swelling and systemic symptoms in DVT. Duplex ultrasonography remains the gold standard for diagnosis, with laboratory tests aiding in complex or septic cases. Hospitalization is warranted in patients with extensive STP, proximity to deep venous systems, or associated infection. Pharmacological treatment includes anticoagulants such as low-molecular-weight heparin, fondaparinux, and direct oral anticoagulants, which effectively prevent thrombus propagation and recurrence. NSAIDs are used for pain control in superficial disease, though caution is advised when concurrent anticoagulation is needed. Antibiotic therapy is reserved for septic thrombophlebitis or catheter-associated infections, targeting common pathogens like *Staphylococcus aureus*.

The internal medicine department coordinates initial assessment, imaging, and pharmacologic therapy, while surgical consultation is essential in refractory or complex cases, including septic thrombophlebitis near the saphenofemoral junction. Surgical interventions such as venous ligation, thrombectomy, or resection are considered when medical therapy fails. A multidisciplinary approach involving internal medicine, surgery, infectious disease, and hematology ensures individualized care, reduces complications, and supports outpatient transition. Structured patient education and follow-up play a critical role in improving long-term outcomes and preventing recurrence.

Keywords: Thrombophlebitis; Internal medicine approach; Surgical management; Multidisciplinary management of thrombophlebitis

Abbreviations: DVT: Deep Venous Thrombosis; SVT: Superficial Venous Thrombophlebitis; NSAIDs: Non-Steroidal Anti-Inflammatory Drugs; LMWH: Low Molecular Weight Heparin; UFH: Unfractionated Heparin; DOACs: Direct Oral Anticoagulants; STP: Septic Thrombophlebitis; GSV: Great Saphenous Vein; SFJ: Saphenofemoral Junction; VTE: Venous Thromboembolism; PE: Pulmonary Embolism; IV: Intravenous; LMWH: Low Molecular Weight Heparin; IVC: Inferior Vena Cava; CHEST: Chest Journal (guidelines reference); ICU: Intensive Care Unit

Introduction

Thrombophlebitis is an inflammatory reaction of the vein related to a thrombus. It causes blood clot formation and blockage of one or more veins, especially in the legs. Thrombophlebitis can be classified as Superficial Thrombophlebitis (ST) when the vein affected is near the surface of the skin and deep Thrombophlebitis (DT) when the vein affected is deep within a muscle. Both types of Thrombophlebitis can be treated with blood-thinning medications [1]. Superficial Thrombophlebitis (ST) refers to an inflammatory condition affecting the superficial veins, often accompanied by the formation of blood clots. It has long been believed that ST is a benign, self-limiting condition that typically resolves rapidly on its own [2]. This clinical presentation is characterized by discomfort, erythema, induration, perivenous edema, and a reddish cord along the vein tender to the touch. Symptoms of deep vein thrombosis include swelling, tenderness, and pain in the affected leg [3].

The mechanism of phlebitis is believed to result from multifactor effects on venous walls, including chemical irritation, bacterial pollution, and machinery traumas. Thrombophlebitis is also affected by the personal factors of patients, including being inactive for a prolonged period, having varicose veins, family history tendency of blood clots formation, pregnancy, hormone replacement therapy, previous episodes of Thrombophlebitis, stroke, overweight or obese, cancer, Smoke, trauma, immune function deficiency, diabetes, malignancy, and high haemoglobin level [4-6]. Thrombophlebitis occurs almost equally in women and men, though males are slightly more likely to suffer it.

There is evidence in the literature suggesting that STP is not always benign. It may precipitate or be associated with deep venous thrombosis (DVT), and it could cause pulmonary embolism (PE). Some studies have shown that the risk of DVT or PE is increased four to sixfold in patients with SVT [3]. Some studies showed that 6 to 36 % of patients with SVT have concomitant DVT, and 2 to 13 % have PE, with 33% of asymptomatic PE [3]. Difference studies reported concomitant DVT or symptomatic PE on the first presentation to be 25 to 30 %, and 5 to 7 % of those patients had symptomatic PE [1,3]. Two mechanisms explain the association between SVT and DVT or PE. The first one is the migration of SVT toward the deep venous system via the saphenofemoral junction, the sapheno-popliteal junction, or a perforating vein, and the second one is the hypercoagulability state that may explain the coexistence of two types of thrombosis [3]. Superficial Thrombophlebitis could manifest as atypical patterns with significant clinical implications; such presentation is migratory Thrombophlebitis, known as Trousseau syndrome. In 5% to 15% of cases, migratory STP can be the initial indicator of an occult malignancy, especially those involving the body and tail of the pancreas [7-10,11]. Recognizing such presentations is crucial, as what may appear to be a minor vascular event could signal a serious underlying pathology.

This review aims to highlight current best practices in the inpatient management of Thrombophlebitis, focusing on the synergistic role of Internal Medicine and Surgical teams. The increasing complexity of hospitalized patients demands a coordinated, evidence-based approach, especially in choosing pharmacologic interventions and determining the need for surgical consultation or intervention. Internal Medicine typically oversees the initial assessment, diagnostic evaluation, and pharmacologic therapy, while Surgical teams may be consulted for complications such as abscess formation or progression despite treatment. Radiology contributes through imaging studies to confirm diagnosis and monitor progression, and Pharmacy supports safe anticoagulant use. This collaborative approach ensures comprehensive care, timely intervention, and improved patient outcomes [8,9].

Pathophysiology and Classification

Thrombophlebitis refers to the inflammation of a vein associated with thrombus formation and is underpinned by the elements of Virchow's triad: endothelial injury, venous stasis, and hypercoagulability. These three factors interact to promote thrombogenesis. Endothelial injury may result from direct trauma, surgical manipulation, or the insertion of intravenous (IV) catheters, all of which disrupt the normally anti-thrombotic inner surface of the vein [12]. Venous stasis can occur in settings of immobility, congestive heart failure, or prolonged hospitalization, reducing the shear stress that typically inhibits clot formation. Hypercoagulability, whether inherited (e.g., Factor V Leiden mutation) or acquired (e.g., malignancy, hormonal therapy), further predisposes to thrombosis by shifting the haemostatic balance toward coagulation [13].

Thrombophlebitis is broadly classified into superficial Thrombophlebitis (STP) and deep vein thrombosis (DVT), depending on the location of the affected veins. STP usually affects superficial veins, such as the great or small saphenous veins, and while traditionally considered benign, STP may extend into deep venous systems or be associated with underlying systemic diseases [14]. DVT involves the deeper venous structures, particularly in the lower extremities, and carries a significantly higher risk for pulmonary embolism (PE) and chronic venous insufficiency if untreated [15].

A further distinction exists between aseptic and septic Thrombophilia. Aseptic Thrombophilia is typically sterile, developing secondary to trauma or chemical irritation, while septic Thrombophilia occurs when infection complicates thrombus formation, commonly in the context of indwelling IV catheters or injection drug use. Septic cases often involve pathogens like *Staphylococcus aureus* and may lead to systemic complications such as bacteraemia or metastatic abscess formation [16]. Clinically relevant associations include the use of peripheral and central venous catheters, which are frequent

sources of endothelial injury and thrombosis in hospitalized patients. Additionally, malignancies—especially pancreatic, gastric, and lung cancers—are strongly linked to thrombotic events, a phenomenon described in Trousseau's syndrome [17]. Recognizing these associations is essential for risk stratification and targeted management.

Clinical Presentation and Diagnosis

The clinical presentation of Thrombophlebitis varies based on the anatomical site and severity of the thrombotic process. In cases of superficial Thrombophlebitis, patients typically report localized pain, tenderness, and swelling along the course of the affected vein. On examination, a firm, palpable cord may be present, often accompanied by erythema and warmth of the overlying skin [18]. In contrast, DVT may present more subtly or with more diffuse symptoms such as unilateral limb swelling, deep aching pain, and occasionally, low-grade fever. In some cases, DVT may be entirely asymptomatic and discovered incidentally.

Duplex ultrasonography remains the diagnostic modality of choice for both STP and DVT. It provides a non-invasive and highly sensitive method to assess venous compressibility, blood flow, and thrombus presence [19]. In cases of suspected DVT, a lack of compressibility in the vein is a hallmark finding. For STP, ultrasound can confirm superficial clot extension and evaluate for possible propagation into deep veins, a critical concern in high-risk patients [20].

Laboratory investigations can support the diagnosis, particularly in equivocal cases. D-dimer, a fibrin degradation product, is elevated in many thrombotic conditions, including Thrombophlebitis, but lacks specificity. Inflammatory markers such as C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR) may be elevated in septic or extensive Thrombophlebitis [21]. Blood cultures are essential when infection is suspected, particularly in patients with fever, catheter-associated phlebitis, or other systemic signs.

The differential diagnosis of Thrombophlebitis includes conditions such as cellulitis, which also presents with erythema and tenderness but lacks the characteristic cord-like venous thickening. Lymphangitis may mimic the linear erythema seen in superficial Thrombophlebitis but typically follows lymphatic drainage patterns and is associated with more systemic symptoms [22]. Differentiating from DVT is also crucial, particularly when symptoms overlap. A thorough history, physical examination, and appropriate imaging are vital for accurate diagnosis and timely intervention.

Inpatient Management Overview

Inpatient management of superficial Thrombophlebitis (STP) and related complications requires careful consideration of specific indications for hospitalization, clear goals of care, and

coordinated multidisciplinary team roles [23,24]. Hospitalization is warranted for patients with extensive STP, particularly when involving the great saphenous vein or occurring within 3 cm of the saphenofemoral junction, due to the elevated risk of progression to deep vein thrombosis (DVT) or pulmonary embolism (PE) [25]. Additionally, cases complicated by infection, such as septic Thrombophlebitis, necessitate admission for systemic antibiotic therapy and close monitoring to prevent sepsis or metastatic complications like endocarditis [23].

The primary goals of inpatient care include symptom control through analgesics and anti-inflammatory agents, preventing thrombosis extension using anticoagulants like low-molecular-weight heparin (LMWH) or fondaparinux, and managing infections with targeted antimicrobial therapy [24]. Internal medicine physicians typically lead the care team, overseeing medical management and coordinating diagnostics, such as duplex ultrasonography, to assess thrombus extent.

Surgical consultation is reserved for select cases, such as refractory septic Thrombophlebitis requiring venous excision or extensive varicosities needing phlebectomy, ensuring a tailored approach to complex presentations [25]. This multidisciplinary strategy optimizes outcomes by addressing the disease's thrombotic and infectious components.

Pharmacological Management

Anticoagulation

Anticoagulation is the cornerstone of therapy for deep venous thrombosis (DVT), especially in cases involving proximal DVT or distal DVT with risk factors such as elevated D-dimer levels, thrombus extension, or underlying malignancy, as recommended by the CHEST guidelines [26]. The primary goal is to prevent thrombus progression, recurrence, and pulmonary embolism. Available anticoagulants include unfractionated heparin (UFH), fondaparinux, low-molecular-weight heparin (LMWH), and direct oral anticoagulants (DOACs). Initial treatment typically begins with LMWH or fondaparinux, followed by warfarin. However, DOACs such as rivaroxaban and apixaban are increasingly preferred due to their ease of use and favourable safety profiles. LMWH has been shown to be as effective as UFH for the treatment of DVT and offers the advantage of once-daily dosing, facilitating outpatient care [27].

Fondaparinux has demonstrated similar efficacy to rivaroxaban in managing superficial thrombophlebitis, while rivaroxaban is also associated with lower costs and fewer adverse events [28]. Apixaban, another DOAC, is effective in treating venous thromboembolism (VTE) and has a low rate of unplanned discontinuation [29]. In postoperative VTE, a four-week anticoagulation course is generally sufficient. In patients with unprovoked DVT, a three-month course is standard, while those with persistent risk factors may benefit from extended therapy

beyond six months [30,31]. Treatment durations shorter than three months are associated with increased recurrence risk [32].

Anti-inflammatory agents

The pharmacological management of thrombophlebitis focuses on alleviating pain, reducing inflammation, and preventing complications such as septic thrombophlebitis or thrombus propagation. Non-steroidal anti-inflammatory drugs (NSAIDs), administered over several weeks, remain the first-line agents for symptom control [33–35]. No individual NSAID has demonstrated superior efficacy over others in managing pain associated with thrombophlebitis [36,37]. Commonly used agents include ibuprofen, diclofenac, and ketoprofen. Limited evidence suggests that topical diclofenac may provide pain relief comparable to its oral formulation [36,38]. However, NSAID use is not without risk. The potential for gastrointestinal and bleeding complications limits their use, particularly in patients who also require anticoagulation [39]. This includes those with superficial venous thrombophlebitis (SVT) located near the deep venous system—within 3 to 5 cm—especially when involving the great or small saphenous veins, extensive SVT (>5 cm), or progressive SVT associated with significant symptoms. In such cases, non-pharmacological measures such as limb elevation and the application of ice over the affected veins can provide symptomatic relief [36–39].

Antibiotic therapy

Antibiotics are not routinely indicated in the treatment of thrombophlebitis. Their use is reserved for cases with clinical signs of infection, such as fever, purulent drainage, erythema, and a palpable cord at the site of inflammation—features suggestive of septic thrombophlebitis or cellulitis. *Staphylococcus aureus* is the most commonly implicated pathogen, followed by *Streptococcus* species and *Enterobacteriaceae*. In burn patients, infections may be polymicrobial, necessitating broad-spectrum empiric coverage. Ultimately, antimicrobial therapy should be tailored based on culture and sensitivity results when available. Intravenous antibiotic therapy administered for up to four weeks has shown effectiveness in managing thrombophlebitis-related infections [39,40]. Early identification of septic involvement and timely initiation of targeted therapy are essential to minimize complications.

Role of the Internal Medicine Department

The Internal Medicine department plays a central role in diagnosing and managing Thrombophlebitis, beginning with the initial evaluation, where internists assess symptoms like pain, swelling, redness, and warmth in the affected area. Clinical judgment and tools like the Wells score help distinguish Thrombophlebitis from other conditions such as cellulitis, DVT, or lymphedema [41]. Internists are also responsible for identifying and addressing risk factors that contribute to Thrombophlebitis.

Common factors include immobility, cancer, recent surgery, and the use of central venous catheters [42]. Managing these proactively—such as encouraging early mobilization or coordinating with oncology—can help prevent progression or recurrence.

Another important role is the initiation and adjustment of medications. Internal medicine teams typically start anticoagulants like low molecular weight heparin or direct oral anticoagulants, carefully considering the patient's kidney function, bleeding risk, and other comorbidities [43]. Anti-inflammatory medications may be used in select cases, especially in superficial Thrombophlebitis [44]. Internists also coordinate investigations like duplex ultrasounds and lab work and, if needed, arrange consultations with surgery, hematology, or infectious disease. Their holistic approach ensures that all necessary diagnostics and specialist input are organized in a timely way [45].

Finally, ongoing follow-up and monitoring of anticoagulation are key parts of their job. This includes adjusting doses, managing side effects, and watching for complications like bleeding or heparin-induced thrombocytopenia [43]. They help ensure treatment is safe and effective by keeping a close watch. The internal medicine team is the backbone of care, working with other specialties to ensure a well-rounded and patient-centred approach.

Role of the Surgery Department

Although most venous pathologies can be resolved with medical treatment, patients could develop complications requiring invasive procedures. John Hunter performed the first ligation of the femoral vein for suppurative Thrombophlebitis in 1874 [46,47]. Since then, surgical indications and techniques have been improved to prevent and treat complications. The role of the Surgery Department in managing these cases is the comprehensive evaluation of patients and the timely assessment of indications for a surgical procedure.

One condition that may require surgical evaluation is septic Thrombophlebitis (STP). STP etiology can be divided into two categories: First, venous infections due to a break in the skin that introduces pathogen organisms, which are associated with an intravenous catheter or other devices in the upper trunk and veins of the limbs. Second, venous infections in which septic Thrombophlebitis occur by dissemination from adjacent infections such as Lemierre syndrome, pyelphlebitis, septic Thrombophlebitis of the dural sinuses, and septic Thrombophlebitis of the pelvic veins [48–50].

A particular concern in lower limb cases is the involvement of the great saphenous vein (GSV) close to the saphenofemoral junction (SFJ) due to its direct connection to the deep venous system through the femoral or perforating veins and its significant risk of thrombus extension, septic emboli, and pulmonary embolism. Those anatomical and clinical features are clear

indications for early surgical consultation [51-53]. In 2005, Nasr concluded that surgical saphenous ligation with compression is superior to compression alone in preventing superficial thrombophlebitis propagation and thromboembolism [51].

The progression of septic Thrombophlebitis despite appropriate medical management with an adequate anticoagulation regimen and antimicrobial therapy, is a clear signal of resistant organisms or mechanical propagation of thrombus. Infected veins should be surgically explored within 24–48 hours if prompt relief is not achieved by removing the tainted catheter or device and delivering appropriate antibiotic therapy. Surgical intervention in this setting may include incision and drainage of an existing abscess, percutaneous mechanical thrombectomy, venous ligation, and resection of a thrombosed venous segment [51-54].

Surgical thrombectomy can safely remove the source of embolization and may enhance the effectiveness of antibiotic therapy by removing most of the infected material, leaving the infected vein wall in close contact with the antibiotic. Recent reports show that endovascular treatment may also be a helpful addition in managing catheter-related infected thrombosis [55]. The Surgery Department's role in treating complex or recurrent cases is to get involved in the multidisciplinary management of those patients, maintaining joint follow-up with the Hematology and Internal Medicine departments. Finally, considering the opinion of the other specialists, a surgeon will determine when a patient meets the criteria for referral to Vascular Surgery management.

Multidisciplinary Coordination in the Inpatient Setting

As Thrombophlebitis requires multiple specialties to oversee, communicate, and manage patients, Interdisciplinary rounds have been shown to be a core part and effective means of good patient health care. Not only do they improve outcomes, but they also decrease readmission rates [56,57]. Individualized treatment plans for each case are necessary to achieve an optimal outcome. Effective communication and shared decision-making on whether to pursue medical or surgical treatment are always warranted [58-60]. The primary team should also keep in mind that Infectious disease may be required in cases where Suppurative or septic Thrombophlebitis is suspected. Hematology may also be consulted in cases where a hypercoagulable state is assumed to be the culprit [61].

Outpatient follow-up and patient education are key components in improving health outcomes. Studies have shown that structured education programs can enhance medication adherence [62]. In addition, a study published in Pharmacotherapy found that patients who received education and telephone follow-up post-discharge were more likely to engage with healthcare providers [63]. Targeted education programs improve educational

knowledge and confidence and break barriers between providers and patients [64,65]. Current studies have already built an evidence-based foundation for outpatient thrombophlebitis, and outpatient management should always be part of the treatment plan.

Conclusion

Effective inpatient management of thrombophlebitis demands a multidisciplinary, evidence-based approach. Timely diagnosis, risk stratification, pharmacologic therapy, and selective surgical intervention-guided by internal medicine-are key to optimizing outcomes and preventing complications. Coordinated care and structured follow-up remain central to long-term success.

References

1. Lee AY, Levine MN (2003) Venous thromboembolism and cancer: risks and outcomes. *Circulation* 107(23 Suppl 1): I17-I21.
2. Trousseau A (1865) Phlegmasia alba dolens. In: *Clinique Médicale de l'Hôtel-Dieu de Paris*. (2nd edn). London: The New Sydenham Society 3: 654-712.
3. Di Nisio M, Wickers IM, Middeldorp S (2018) Treatment for superficial thrombophlebitis of the leg. *Cochrane Database Syst Rev* 2(2): CD004982.
4. Decousus H, Quéré I, Presles E (2010) Superficial venous thrombosis and venous thromboembolism: a large, prospective epidemiologic study. *Ann Intern Med* 152(4): 218-224.
5. Nasr WR, Comerota AJ, Meuse MA (2015) Diagnosis and management of superficial venous thrombosis. *J Vasc Surg Venous Lymphat Disord* 3(1): 113-117.
6. Zurcher RM, Kucher N (2021) Superficial vein thrombosis: current evidence and practical guidance. *Vasc Med* 26(1): 41-49.
7. Kalodiki E, Stvrtnová V, Allegra C (2005) Superficial thrombophlebitis: guidelines of the European Venous Forum. *Int Angiol* 24(1): 1-18.
8. Lattimer CR, Kalodiki E, Azzam M (2014) Clinical significance of superficial venous thrombosis. *Phlebology* 29(1 Suppl): 33-40.
9. Elders A, Sedhom R, Saunders C (2015) Superficial vein thrombosis in primary care: a survey of diagnosis and management. *Br J Gen Pract* 65(630): e802-e807.
10. Van Langevelde K, Sramek A, Rosendaal FR (2011) Venous thrombosis: understanding the pathogenesis, risk factors and long-term follow-up. *Br J Haematol* 152(6): 818-827.
11. Kröger K, Moerchel C, Ose C (2015) Superficial vein thrombosis: risk factors and clinical significance. *Clin Appl Thromb Hemost* 21(5): 468-472.
12. Verlato F, Zucchetto P, Prandoni P (1999) An unexpectedly high rate of pulmonary embolism in patients with superficial thrombophlebitis of the thigh. *J Vasc Surg* 30(6): 1113-1115.
13. Di Nisio M, Wickers IM, Middeldorp S (2007) Treatment for superficial thrombophlebitis of the leg. *Cochrane Database Syst Rev* 2: CD004982.
14. Decousus H, Prandoni P, Mismetti P (2010) Fondaparinux for the treatment of superficial-vein thrombosis in the legs. *N Engl J Med* 363(13): 1222-1232.
15. Gillet JL, Perrin MR, Hiltbrand B (2001) Superficial venous thrombosis: a frequent and benign disease? A prospective study in 85 patients. *J Mal Vasc* 26(1): 16-22.

16. Weill A, Ricordeau P, Fournier JP (2012) Venous thromboembolism recurrence after hospitalization for superficial vein thrombosis: a nationwide study. *J Thromb Haemost* 10(5): 858-864.
17. Nicolaidis AN (2000) Investigation of chronic venous insufficiency: a consensus statement. *Circulation* 102(20): E126-E163.
18. Comerota AJ (2006) Thrombophlebitis: modern diagnosis and treatment. *Curr Treat Options Cardiovasc Med* 8(2): 117-123.
19. Hach-Wunderle V, Bauersachs R, Gerlach H (2012) Diagnosis and treatment of superficial vein thrombosis. *Dtsch Arztebl Int* 109(50): 839-846.
20. Mahler F, Bachmann F (2010) Management of thrombophlebitis and superficial vein thrombosis. *Vasa* 39(1): 35-42.
21. Kearon C, Akl EA, Ornelas J (2016) Antithrombotic therapy for VTE disease: CHEST guideline and expert panel report. *Chest* 149(2): 315-352.
22. Lemos DW, Posacioglu H, O'Donnell TF (2000) The natural history of iliofemoral deep venous thrombosis treated with anticoagulation. *J Vasc Surg* 32(6): 130-137.
23. Lurie F, Kistner RL, Eklof B (2003) Mechanisms of venous valve closure and role of the valve in circulation: a new concept. *J Vasc Surg* 38(5): 955-961.
24. Schobersberger W, Tockner B, Haselbacher M (2006) Superficial thrombophlebitis: pathophysiology and therapy. *Wien Med Wochenschr* 156(13-14): 376-382.
25. Jorgensen JO (1982) Surgical treatment of superficial thrombophlebitis. *Br J Surg* 69(12):744-746.
26. Morrison N, Neuhardt DL (2010) Update on the management of superficial thrombophlebitis. *Perspect Vasc Surg Endovasc Ther* 22(3): 176-182.
27. Comerota AJ (2009) Treatment of superficial venous thrombosis: is anticoagulation necessary? *Perspect Vasc Surg Endovasc Ther* 21(3): 154-157.
28. Kesieme E, Kesieme C, Jebbin N (2011) Deep vein thrombosis: a clinical review. *J Blood Med* 2: 59-69.
29. O'Donnell TF, Iafrati MD (2002) Superficial venous thrombophlebitis: clinical relevance and potential complications. *Vasc Med* 7(3): 209-213.
30. Ginsberg JS (1996) Management of venous thromboembolism. *N Engl J Med* 335(24): 1816-1828.
31. Labropoulos N, Giannoukas AD (2005) Where do superficial thromboses end? *Phlebology* 20(1): 2-3.
32. Prandoni P (2014) Superficial vein thrombosis: more needs to be done. *Lancet Haematol* 1: e8-e9.
33. Blättler W, Partsch H (2003) Leg compression and ambulation in thrombophlebitis and deep vein thrombosis. *Vasa* 32(2): 73-78.
34. Jawien A, Grzela T, Ochwat A (2003) Prevalence of chronic venous insufficiency in men and women in Poland: multicenter cross-sectional study in 40,095 patients. *Phlebology* 18(3): 110-122.
35. Ricci S, Georgiev M (2012) The use of low-molecular-weight heparins in superficial vein thrombosis. *Curr Vasc Pharmacol* 10(6): 781-785.
36. Verhamme P, Zuily S, Diehm C (2020) Management of superficial vein thrombosis in primary care: results from an international Delphi consensus. *J Thromb Thrombolysis* 50(2): 318-326.
37. Kakkos SK, Gohel M, Baekgaard N (2022) Editor's choice-European Society for Vascular Surgery (ESVS) 2022 clinical practice guidelines on the management of chronic venous disease of the lower limbs. *Eur J Vasc Endovasc Surg* 63(2): 184-267.
38. Wittens C, Davies AH, Bækgaard N (2015) Editor's choice-management of chronic venous disease: clinical practice guidelines of the European Society for Vascular Surgery (ESVS). *Eur J Vasc Endovasc Surg* 49(6): 678-737.
39. Musani MH, Matta F, Yaekoub AY (2010) Venous thromboembolism and cancer: current trends and future prospects. *Clin Chest Med* 31(4): 841-856.
40. White RH (2003) The epidemiology of venous thromboembolism. *Circulation* 107: 14-18.
41. Haenen L, Ten Cate-Hoek AJ (2016) Superficial vein thrombosis: a common and benign disease? *Neth J Med* 74(10): 423-431.
42. Jorgensen JO (1988) Surgical intervention for superficial vein thrombosis. *Phlebology* 3(2): 73-75.
43. Caprini JA (2012) Superficial venous thrombophlebitis: treat or ignore? *Thromb Haemost* 108(2): 226-228.
44. Weitz JI, Eikelboom JW, Samama MM (2012) New antithrombotic drugs: antithrombotic therapy and prevention of thrombosis. *Chest* 141(2 Suppl): e120S-e151S.
45. Ascher E, Hingorani A, Markevich N (2001) Predictive factors for thromboembolic complications in lower extremity superficial venous thrombophlebitis. *Ann Vasc Surg* 15(1): 83-89.
46. Eriksson H, Almgren B, Ekberg O (1999) Ultrasonographic findings and clinical outcome in patients with superficial thrombophlebitis. *Eur J Ultrasound* 9(1): 37-43.
47. Samama MM, Gerotziafas GT (2013) Comparative pharmacology of new oral anticoagulants. *Drugs* 73(10): 1043-1053.
48. Erdman WA, Meissner MH (2005) Thrombophlebitis: surgical and nonsurgical treatment options. *Semin Intervent Radiol* 22(3): 238-243.
49. Mlekusch W, Sabeti S, Haumer M (2002) Low molecular weight heparin in the treatment of superficial vein thrombosis. *J Vasc Surg* 36(4): 835-839.
50. Di Nisio M, Wichers IM, Middeldorp S (2007) Treatment for superficial thrombophlebitis of the leg. *Cochrane Database Syst Rev* 2: CD004982.
51. Decousus H, Prandoni P, Mismetti P (2010) Fondaparinux for the treatment of superficial-vein thrombosis in the legs. *N Engl J Med* 363(13): 1222-1232.
52. Gillet JL, Perrin MR, Hiltbrand B (2001) Superficial venous thrombosis: a frequent and benign disease? A prospective study in 85 patients. *J Mal Vasc* 26(1): 16-22.
53. Hach-Wunderle V, Bauersachs R, Gerlach H (2012) Diagnosis and treatment of superficial vein thrombosis. *Dtsch Arztebl Int* 109(50): 839-846.
54. O'Donnell TF (2012) Superficial venous thrombosis: current treatment and prevention strategies. *Vasc Med* 17(1 Suppl): S9-S14.
55. Kröger K, Moerchel C, Ose C (2015) Clinical significance of superficial vein thrombosis. *Clin Appl Thromb Hemost* 21(5): 468-472.
56. Brosi P, Dick F, Do DD (2014) Predictive factors for concurrent deep vein thrombosis and pulmonary embolism in patients with superficial vein thrombosis. *J Vasc Surg Venous Lymphat Disord* 2(1): 27-33.

57. Labropoulos N, Giannoukas AD, Delis K (2005) Secondary thrombus extension from superficial to deep veins: prospective study with duplex scanning. *J Vasc Surg* 41(4): 684-690.
58. Rosendaal FR (1999) Venous thrombosis: a multicausal disease. *Lancet* 353(9159): 1167-1173.
59. Bounameaux H (2002) Superficial vein thrombosis: is it clinically relevant? *Curr Opin Hematol* 9(5): 403-407.
60. Michiels JJ, Gadisseur A, Van der Planken M (2004) Evidence-based thromboprophylaxis and treatment in superficial vein thrombosis. *Clin Appl Thromb Hemost* 10(1): 1-6.
61. Goldenberg NA, Manco-Johnson MJ (2008) Pediatric thrombophilia. *Hematology Am Soc Hematol Educ Program*, pp: 387-396.
62. Margaglione M, Grandone E (1999) Population genetics of factor V Leiden: evolutionary implications. *Semin Thromb Hemost* 25(4): 367-373.
63. Flinterman LE, Van Hylckama Vlieg A, Rosendaal FR (2009) Recurrent venous thrombosis and inherited thrombophilia: a prospective cohort study. *J Thromb Haemost* 7(4): 496-502.
64. Baglin T (2005) Venous thromboembolism in cancer: treatment and prevention. *BMJ* 331(7510): 1100-1101.
65. Khorana AA (2012) Cancer-associated thrombosis: updates and controversies. *Hematology Am Soc Hematol Educ Program* 2012: 626-630.



This work is licensed under Creative Commons Attribution 4.0 License
DOI: [10.19080/JPCR.2025.11.555804](https://doi.org/10.19080/JPCR.2025.11.555804)

Your next submission with Juniper Publishers will reach you the below assets

- Quality Editorial service
- Swift Peer Review
- Reprints availability
- E-prints Service
- Manuscript Podcast for convenient understanding
- Global attainment for your research
- Manuscript accessibility in different formats
(Pdf, E-pub, Full Text, Audio)
- Unceasing customer service

Track the below URL for one-step submission
<https://juniperpublishers.com/online-submission.php>