


Review Article

Diagnostic, therapeutic and preventive medical interventions in superficial thrombophlebitis

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Abstract

Superficial thrombophlebitis belongs to the group of the most common vascular disorders today. It is known as a benign, generally self-limiting inflammatory disorder that rapidly resolves and relieves symptoms. However, as it is a frequently underdiagnosed condition, rapid progression of the disease to the deep venous circulation or recurrent symptoms without prior treatment is possible. Clinically, it manifests with localized pain, erythema, local warmth, and induration along the affected venous tract. Unlike deep vein thrombosis (DVT), it rarely produces significant edema. Diagnosis is primarily clinical, although Doppler ultrasound has been documented as an important diagnostic tool in these cases to confirm the extent of the thrombus and thus rule out complications. Treatment includes pharmacological, non-pharmacological, and surgical measures. Local methods include nonsteroidal anti-inflammatory drugs (NSAIDs), compression with elastic stockings, and early mobilization, as well as anticoagulation in some cases or surgical resection, as explained below. It is generally considered a disease with a good prognosis with the implementation of appropriate and timely measures.

Key words

Superficial thrombophlebitis, Doppler, Anticoagulation, Fondaparinux, Compression therapy, Topical heparin.

Introduction

Superficial thrombophlebitis belongs to the most common vascular disorders currently; it is known as a benign inflammatory disorder, generally self-limited, consisting of rapid resolution and relief of symptomatology. However, over time, as the pathophysiological process of the disease has become known, a high probability of developing thromboembolic complications that may compromise patients' lives has been documented.

Thromboembolic disease has a natural history loaded with diverse risk factors and symptomatology that this research aims to present, in order to obtain a focus on the importance of early treatment, since it has been demonstrated that when there is involvement of axial veins such as the saphenous vein, accessory saphenous vein, and small saphenous vein, propagation of the thrombus to the deep venous system may occur.

The passage of the thrombus to the deep venous system leads to health complications, some severe such as deep venous circulation thrombosis (DVT) and others fatal such as pulmonary embolism. Based on various studies, it is concluded that many patients diagnosed with deep venous thrombosis may have concomitant superficial thrombophlebitis, which generates a clinical underdiagnosis of this disease.

The underdiagnosis of superficial thrombophlebitis is the main reason for conducting this research, since if it is possible to demonstrate the diagnostic, therapeutic, and preventive effectiveness of superficial phlebothrombosis, it is possible to avoid the previously mentioned complications, which may be severe or fulminant in many cases.

Methodology

The research approach is qualitative, since data have been collected without numerical measurements to provide answers to the research question; the data were gathered through the

evaluation of scientific articles and informational sources such as books and websites. The population of this research takes into account the totality of articles documenting medical, diagnostic, therapeutic, and preventive interventions for superficial thrombophlebitis.

The project sample consists of a total of 15 scientific articles that meet the previously selected inclusion and exclusion criteria, such as publication years from 2019 to July 2025, in English and Spanish languages, and free-access articles. For this purpose, the PubMed, EBSCO, and Dialnet databases were used.

Concept of Superficial Thrombophlebitis

The term "phlebitis" individually represents the concept of inflammation within a vein, and on the other hand, "thrombosis" indicates the presence of a clot or thrombus inside the vein; therefore, the term superficial phlebitis denotes the presence of pain and inflammation in a vein without the presence of a thrombus [1].

The author Manuel Frías Vargas defines superficial thrombophlebitis, also called superficial venous thrombosis (SVT), as an inflammatory process at the venous level with confirmed thrombosis of the veins, in this case the superficial type, at any anatomical location. By using the term SVT, multiple synonyms of this pathology are encompassed, among them: thrombophlebitis, varicophlebitis, periphlebitis, and phlebothrombosis [1].

Etiology and Pathogenesis of Superficial Thrombophlebitis

Venous thrombotic pathology in any of its presentations, superficial or deep, is composed of the classic Virchow's triad described in 1856, consisting of three mechanisms explained below [2]:

Blood stasis: it is defined as the stagnation or decrease of blood flow due to compression of the vessel or the presence of an obstacle in the circulatory pathway. This factor occurs when there is immobility of a limb or the entire body,

for example, with prolonged bed rest, immobilization due to fractures or major surgeries, among others.

Hypercoagulability: it is defined as a homeostatic mechanism that generates clot formation and involves an increase in disorders that raise the concentration or activation of coagulation factors, among them hereditary or acquired deficiencies of plasma proteins that normally function to inhibit thrombus development such as antithrombin III, protein C and S, factor V Leiden, and mutations in the thrombin gene.

Endothelial injury: as its name indicates, it refers to vascular damage; most frequently it occurs as a consequence of trauma or due to a surgical intervention. However, its appearance is also possible due to infection or inflammation at the level of the vascular wall. In the case of superficial thrombophlebitis, this is why the placement of intravenous catheters is one of the main causes of vascular injury.

Following this blood triad, the thrombosis process results in the recruitment of activated platelets that initiate the release of microparticles, which generate proinflammatory mediators that bind to neutrophils, and through stimulation these cells secrete nuclear material, generating extracellular networks of a prothrombotic type.

Classification of Superficial Thrombophlebitis

Superficial thrombophlebitis can be located in the upper and lower extremities, as well as in other anatomical sites such as the abdominal wall. However, it was previously indicated that the majority of cases occur in the lower limbs compared to other anatomical sites [1].

Chemical superficial thrombophlebitis: Specifically, the significant risk of chemical phlebothrombosis is present when the pH and osmolarity of intravenous solutions or medications differ from blood levels. For this reason, it has been demonstrated that hypertonic

solutions with osmolarities greater than 450 mOsm/L or substances with a pH lower than 5 generate the highest number of phlebitis cases associated with superficial thrombophlebitis in the upper limbs [3].

Mechanical superficial thrombophlebitis: This subtype of superficial vascular pathology is classified among the most frequent. It is generated as a result of trauma or injury to the venous wall, classically due to one component of Virchow's triad: endothelial injury. Unlike deep thrombophlebitis, superficial types are found in 80–90% of cases in the upper limbs as a consequence of intravenously positioned central or peripheral catheters, with greater involvement of the cephalic and basilic veins [4].

Mechanical phlebothrombosis may occur due to any traumatic mechanism affecting the venous wall; however, most cases occur because of intravenous catheters. The materials used to manufacture peripheral venous catheters have different sizes and physicochemical properties that influence the manifestation of thrombophlebitis [3].

Septic superficial thrombophlebitis: Also known as bacterial or suppurative thrombophlebitis, this subtype of vascular pathology presents with the typical clinical picture of superficial venous thrombosis plus clinical manifestations such as high fever, leukocytosis, chills, purulent discharge, among others, suggesting an infection concomitant with the initial inflammatory process. It may be suspected when erythema extends beyond the margin of the affected vein [4].

Intravenous cannulas are not the only cause, since this type of thrombophlebitis may also manifest in the presence of skin lesions such as ulcers or open dermal wounds.

Migratory superficial thrombophlebitis: Also known as thrombophlebitis migrans, it is a subtype of vascular phlebothrombosis characterized by recurrent episodes of

thrombosis affecting superficial venous circulation, especially in the lower limbs, abdomen, and trunk. It manifests through the appearance of nodules with a linear distribution along the affected extremity, accompanied by an indurated cord that is easily palpable [5].

Mondor's disease: Described in 1939 as thrombosis of the thoracoepigastric vein, it is characterized as a superficial thrombophlebitis localized precisely in that vein, in the thoracoabdominal wall, with greater involvement in females at a 9:1 ratio. Clinically, it manifests as a suddenly appearing palpable indurated subcutaneous cord on the thoracic wall [5].

Risk factors for superficial thrombophlebitis

This condition has predisposing factors based on vascular alterations that favor thrombus formation due to coagulation abnormalities, including variations that lead to decreased venous flow, impaired fibrinolysis, and endothelial dysfunction. As previously mentioned, this group of circulatory disorders can be explained by Rudolf Virchow's triad [6].

The main risk factors for superficial phlebothrombosis are briefly described below:

Varicose veins: These represent one of the principal predisposing factors for superficial venous thrombosis (SVT), accounting for 62–90% of cases [7].

They are defined as tortuous superficial veins of the lower extremities caused by venous dilation. As a combination of progressive venous reflux and venous hypertension, they are considered a common sign of chronic venous insufficiency. The great saphenous vein and its tributaries are most commonly affected; however, the small saphenous vein may also be involved.

Chronic venous disease: This is a severe manifestation of venous hypertension, generally affecting the lower extremities. It results from inadequate coaptation of venous valve leaflets

due to thickening and scarring or from dilated veins with reduced functional capacity.

When the valves are unable to prevent venous reflux toward the feet, the lower limbs develop venous hypertension, generating increased hydrostatic pressure in the subcutaneous veins and surrounding tissues. This predisposes patients to both superficial and deep venous thrombosis (**Table – 1**).

Venous excision and ablation: Superficial phlebothrombosis frequently occurs following open surgical ablation, radiofrequency ablation, chemical adhesive ablation, sclerotherapy, or endovenous ablation procedures. After open venous excision, the residual venous segment may undergo spasm and thrombosis, and the thrombus may propagate retrogradely into a patent vein.

The residual segment can develop inflammation, thrombosis, and luminal fibrosis, presenting clinically with pain and swelling. However, in some patients with SVT secondary to endovenous procedures, the thrombotic process may remain confined to the treated segment if the energy delivered during the procedure was adequate [8].

Deep venous thrombosis and prior superficial thrombophlebitis: Although varicose veins are most commonly affected in this vascular pathology, involvement of otherwise healthy veins has been reported in 5–10% of cases. In the absence of varicose veins, lower-extremity SVT may serve as a potential marker of hypercoagulability or malignancy. Patients with a history of SVT have an increased risk of recurrence, as well as a higher likelihood of developing deep venous thrombosis (DVT) and pulmonary embolism.

Use of intravenous catheters: This is classified as one of the main mechanical etiologies of SVT in both upper and lower extremities, resulting from a combination of endothelial injury and venous stasis.

Table – 1: Compression Levels in Chronic Venous Disease.

Class	Pressure	Level of Support	Indication
I	Mild	Light	Asymptomatic individuals as needed for comfort
II	Moderate	Moderate	Mild varicosities, tired/aching legs, mild ankle, leg, or foot swelling
III	Firm	Strong	Moderate to severe varicosities, moderate swelling, phlebitis, post-venous ablation
IV	Extra firm	Maximum	Severe varicosities, severe swelling, management of active ulcers, post-DVT, post-surgery, lymphedema

Source: Scovell, S. (2023). Superficial vein thrombosis and phlebitis of the lower extremity veins.

Additional risk factors predisposing to both superficial and deep thrombophlebitis include obesity, inherited or acquired thrombophilias, pregnancy, oral contraceptive use and estrogen therapy, prolonged immobilization, major surgery, vascular trauma, among others.

Clinical presentation of superficial thrombophlebitis

This vascular condition presents clinical findings related to venous inflammation and thrombosis. Due to phlebitis, tributary veins located just beneath the dermis - often associated with varicosities - may be easily visualized. In contrast, vascular changes related to venous thrombosis are not always visible, as the superficial axial veins are typically located 1.5 cm or more below the skin surface [9].

The clinical manifestations of superficial venous thrombosis (SVT) are classified into complicated and uncomplicated presentations:

Complicated SVT

This presentation is characterized by recurrent involvement of the same vein, possible concomitant infection, and a close association with pulmonary embolism or deep venous thrombosis.

Dermal hyperpigmentation is a frequent physical finding. It may appear after an initial episode of SVT or following recurrence. This sign results from thrombus degradation and subsequent

hemosiderin deposition within the vascular tissues of the thrombosed veins.

Uncomplicated SVT

Most symptomatic SVT cases are classified as uncomplicated. They present with typical localized manifestations, including tenderness over the affected area, palpable induration, pain, and varying degrees of edema, erythema, and localized warmth along the course of the superficial vein - generally in association with a tributary varicosity. Pruritus may occasionally be present.

Low-grade fever may occur in some cases of SVT. However, if the fever is high, septic thrombophlebitis should be considered, particularly if located at a site of infection, or the possibility of a severe complication of superficial phlebotrombosis should be evaluated.

Diagnostic Medical Interventions

Physical examination and clinical manifestations are usually sufficient for the clinical diagnosis of superficial thrombophlebitis, especially in patients with predisposing risk factors. However, the requirement to perform a duplex ultrasound or any additional evaluation with laboratory studies or others depends on the clinical scenario, including the need to rule out suspicion of superficial thrombophlebitis coexisting with deep venous thrombosis [10].

Duplex ultrasound, also known as Doppler, is an economical and noninvasive method whose purpose is to verify the location of perforating

veins and thoroughly evaluate the superficial axial veins in order to exclude DVT; therefore, it is the method of choice for the diagnosis of SVT. In any finding of thrombosis, the exact location and length of the thrombus must be documented in detail, since the need to treat superficial venous thrombosis is based on whether the thrombus length is greater than or equal to 5 cm [1].

When clinical symptoms extend along an axial vein with significant edema, the patient should undergo Doppler ultrasound because the risk factors shared with deep venous thrombosis are frequent.

Patients with SVT located less than or equal to 5 cm from the deep venous system may present a high prevalence of concomitant DVT. Likewise, the risk of pulmonary embolism increases when superficial thrombophlebitis occurs in the cephalic saphenous vein [10]. Doppler ultrasound should be requested in SVT cases longer than 5 cm that affect multiple veins or in superficial venous thrombophlebitis occurring in healthy veins without varicosities.

Doppler Ultrasound Findings

Ultrasound allows visualization of the venous system; SVT is documented with thickening of the venous wall accompanied by periluminal or subcutaneous edema. On some occasions, the presence of an intraluminal thrombus can be identified or the extension of the thrombus from the superficial veins to the deep venous circulation can be visualized through the confluences of the saphenofemoral junction, saphenopopliteal junction, or through the perforating veins [10].

Noninvasive visual examination allows the actual extent of the thrombus and its progression to be revealed, as well as the concomitance of DVT, whether ipsilateral or bilateral.

Superficial phlebothrombosis manifests from a retrograde flow extension of the thrombus from the deep veins to the superficial veins; therefore,

on some occasions it is not possible to determine whether the superficial venous system was affected prior to the deep system [10].

Additional Diagnostic Evaluations

Additional tests are reserved for complicated superficial thrombophlebitis. In patients in whom anticoagulant therapy is required, baseline coagulation studies and a D-dimer should be requested at the start of treatment and repeated at 45 days; if the D-dimer remains elevated, this indicates that the thrombosis has not resolved. Regarding patients with septic thrombophlebitis, a complete blood count and blood cultures should be requested in order to guide the necessary antibiotic therapy [10].

Medical Interventions in Treatment

Medical / Non-pharmacological Treatment

- **Warm or cold compresses:** Placement of these on the affected segment and elevation of the extremity with thrombophlebitis have been shown to be effective for symptom relief and to contribute to the inflammatory process [11].
- **Early ambulation:** The patient is instructed to ambulate constantly, since immobilization favors venous stasis in the vascular process [4].
- **Compression therapy:** Compression stockings or compression band therapy should be prescribed to all patients without contraindications to their use, such as peripheral arterial disease, and should cover the entire area of venous tract involvement. Although it has been shown that compression therapy alone is not as effective, it has been documented that it provides additional benefit when combined with anticoagulant or anti-inflammatory treatment. Annex I documents the amount of pressure required in association with the indication for compression therapy; the recommended compression in the

treatment of SVT is class II, 20–30 mmHg [12].

Pharmacological Treatment

- **Antibiotic therapy:** This is reserved only for patients with septic superficial thrombophlebitis, which, being an intravascular abscess, additionally requires anticoagulation treatment for thrombus removal. This empirical treatment is based on vancomycin 15 mg/kg intravenously (IV) every 12 hours plus ceftriaxone 1 g IV every 24 hours; in the presence of positive cultures, a duration of 10 days is provided [13].
- **Nonsteroidal anti-inflammatory drugs (NSAIDs):** Pain management is an important point to address due to venous inflammation. NSAIDs are a good option in cases of superficial thrombophlebitis; however, the severity of each individual must be adequately classified because these may increase the risk of bleeding, and therefore should be avoided in patients who require anticoagulation. It has been demonstrated that oral NSAID therapy has been sufficient to control pain, even to reduce the risk of recurrence and the extension of the localized thrombus [13].
- **Topical therapy:** With the use of topical anti-inflammatory agents such as diclofenac gel or some heparinoids, relief of local symptoms and acceleration of complete resolution in thrombophlebitis of tributary veins have been documented [14].
- **Anticoagulation:** Normally, SVT resolves through the measures previously described; however, the use of anticoagulants may indicate sustained symptom relief, as well as prevent the risk of pulmonary thromboembolism and minimize the risk of recurrence of the vascular process. For this reason, patients with superficial thrombophlebitis have been classified

according to the location and extent of the thrombus, as well as the individually determined severity range in each case [13].

Below is the classification of patients according to the risk of developing venous thromboembolism (VTE) from SVT [10]:

Low risk of venous thromboembolism (VTE):

This group includes patients with isolated SVT or uncomplicated SVT; therefore, they do not require anticoagulation treatment. Only repetition of duplex ultrasound is recommended in order to identify those who present thrombus extension and who subsequently require anticoagulant drugs.

Within this category are: patients with superficial thrombophlebitis far from the saphenofemoral junction or the saphenopopliteal junction; SVT in the great saphenous vein either below the knee or above the knee; focal superficial phlebothrombosis affecting only one axial vein, that is, with an extension less than or equal to 5 cm; and patients who do not have predisposing risk factors for VTE.

Intermediate risk of VTE: This category is reserved for patients with uncomplicated axial SVT but with a significant risk for thromboembolism; therefore, prophylactic anticoagulation is recommended. At this level are patients with proximal thrombophlebitis located 3–5 cm from the deep venous circulation, predominantly in the great or small saphenous vein.

Patients with superficial phlebothrombosis with greater extension than the affected vein segment, that is, greater than or equal to 5 cm and not limited to local involvement, and finally those with SVT with extension associated with symptomatology.

High Risk of VTE: Patients with a significant alarm level require therapeutic anticoagulation. This category includes patients with superficial

vein thrombosis (SVT) located within 3 cm of the deep venous system, especially in the great and small saphenous veins; patients with recurrent superficial thrombophlebitis after completion of anticoagulant therapy; those with SVT that extends despite prophylactic anticoagulation; and patients with superficial thrombophlebitis who have VTE risk factors.

Predisposing Factors for High-Risk VTE include age over 65 years, male sex, previous thromboembolism or prior superficial thrombophlebitis, cancer, autoimmune disease, and damage to non-varicose veins. These patients should receive prolonged treatment with either prophylactic doses or therapeutic anticoagulation.

Pharmacological Treatment should be administered according to four main groups [13]:

1. **SVT \leq 3 cm from the sapheno-femoral or sapheno-popliteal junction:** Therapeutic doses of low molecular weight heparin (LMWH).
2. **SVT $>$ 3 cm from the sapheno-femoral or sapheno-popliteal junction and $>$ 5 cm in length:** Therapeutic doses of Fondaparinux 2.5 mg/day subcutaneously, Rivaroxaban 10 mg/day orally, or intermediate doses of LMWH (e.g., Enoxaparin 40–80 mg/day subcutaneously).
3. **SVT $>$ 3 cm from the sapheno-femoral or sapheno-popliteal junction and $<$ 5 cm in length without risk factors:** Oral NSAIDs, such as Ibuprofen 400 mg every 8 hours or Naproxen 500 mg every 12 hours, with reassessment in approximately 7–10 days.
4. **SVT $>$ 3 cm from the sapheno-femoral or sapheno-popliteal junction and $<$ 5 cm in length with severe symptoms or associated risk factors:** LMWH at prophylactic or intermediate doses, prophylactic Fondaparinux, or Rivaroxaban 10 mg/day orally for 45 days.

Surgical Treatment [15]

Surgical repair is reserved for patients with indications for pharmacological anticoagulation but with contraindications to such therapy. The procedure involves ligation of the saphenous vein. This is defined as excision of the great saphenous vein (GSV) via an oblique incision in the inguinal region at the sapheno-femoral junction with the common femoral vein. Ligation of the tributary veins prevents superficial venous flow, eliminating recurrent reflux and varicosity formation.

Although this technique has largely been replaced by surgical ablation, in SVT there is a relative contraindication for this procedure due to the higher risk of deep vein thrombosis (DVT) or thromboembolism.

Main medical complications of superficial thrombophlebitis

Deep Venous Thrombosis (DVT)

As discussed throughout this project, thrombophlebitis primarily affects the superficial venous circulation. However, this condition may become complicated if the thrombotic process extends into the deep venous system, resulting in thrombus formation within the deep circulation. These thrombi most frequently occur in the legs and thighs, and less commonly in the upper extremities [6].

Superficial venous thrombosis (SVT) and deep venous thrombosis (DVT) share most risk factors. Therefore, in the majority of clinical cases, a Doppler ultrasound study should be performed to determine the depth of venous involvement and the extent of thrombus propagation in order to guide appropriate treatment [15].

Pulmonary Embolism (PE)

Although superficial thrombophlebitis has historically been considered a relatively benign thromboembolic condition, progression into the deep venous system via DVT may lead to an acute and potentially life-threatening

complication such as pulmonary embolism (PE) [16].

In this condition, embolic material - most commonly a thrombus originating from the deep venous circulation - obstructs one or more pulmonary arteries. This results in impaired pulmonary blood flow and increased right ventricular pressure due to elevated pulmonary vascular resistance [16].

Postphlebotic Syndrome

Also known as post-thrombotic syndrome, this condition typically develops as a consequence of long-standing venous hypertension combined with thrombotic obstruction that produces valvular reflux. In addition, persistent inflammation and vascular wall remodeling contribute to its development in conditions such as superficial and deep thrombophlebitis and chronic venous insufficiency [17].

Prevention of Superficial Thrombophlebitis [11]

Preventive measures for this condition are based on the need to improve circulation. Key recommendations include regular mobilization of the lower limbs through exercise or physical activity and avoidance of prolonged immobility to prevent thrombus formation within the venous system.

Compression therapy is beneficial for individuals undertaking long journeys with limited lower limb movement or those who spend prolonged periods standing. It is also recommended for patients with varicose veins, with the pressure level adjusted according to the extent of venous involvement [18].

For superficial thrombophlebitis caused by intravenous cannulas, the primary recommendation is to rotate catheter placement every 3 days, position catheters parallel to the vein, and administer intravenous saline solutions prior to infusing hypertonic substances [12].

Conclusions

Regarding diagnostic medical interventions, the primary recommendation is the use of Doppler ultrasound as the preferred method for diagnosing superficial thrombophlebitis, primarily to assess thrombus location and extent. This method is considered effective, low-cost, and comprehensive. Established criteria also exist for requesting complementary imaging studies, such as Duplex ultrasound.

It is crucial to integrate complementary diagnostic tests, such as D-dimer assays, alongside Doppler ultrasound for a more precise diagnosis of superficial thrombophlebitis. Although D-dimer is not specific for this condition, its use as a comparative tool during recovery can enhance patient monitoring, track disease progression, and guide treatment adjustments.

Accurate therapeutic determination for each case of superficial thrombophlebitis requires confirmation by Doppler ultrasound, as the condition demands targeted treatment to prevent progression to deep vein thrombosis (DVT) or pulmonary embolism (PE).

All patients with superficial thrombophlebitis, regardless of thrombus risk classification or extent, should receive nonsteroidal anti-inflammatory drugs (NSAIDs) for 7–14 days to relieve pain and inflammation. Elevation of the affected limb and early ambulation are recommended to promote venous return and prevent stasis.

Patients may also benefit from Class II compression therapy (20–30 mmHg) as an adjunct to recovery. In cases of septic superficial thrombophlebitis, empirical antibiotic therapy is indicated.

Therapeutic anticoagulation is indicated when SVT is ≤ 3 cm from the venous junction, or > 3 cm from the sapheno-femoral or sapheno-popliteal junction with thrombus extension > 5

cm, or >3 cm from the junction with <5 cm extension in the presence of risk factors or severe symptoms.

The preferred anticoagulant is Fondaparinux at therapeutic doses of 2.5 mg/day subcutaneously. Rivaroxaban (20 mg/day orally) is also an option for stable patients, both administered for 45 days. (Martinez Segura)

Combined therapy with elastic bandaging, anticoagulation, and anti-inflammatory medications is considered the complete treatment of choice. Timely and appropriately selected therapy can halt and reverse thrombotic progression.

Preventive interventions include topical heparin prior to intravenous catheter insertion, an effective, low-cost, and easy-to-apply method, making it an ideal prophylactic strategy for superficial thrombophlebitis. Its use reduces discomfort from frequent catheter changes, hospital stay, and overall costs.

When comparing topical heparinoids to topical diclofenac, both are effective in preventing superficial thrombophlebitis; however, topical heparinoids demonstrate superior results on phlebitis scoring scales, while topical NSAIDs remain an excellent alternative when anticoagulants are contraindicated.

Compression therapy as a preventive measure provides gradual limb compression, enhancing tissue perfusion, reducing venous pressure and valvular reflux, minimizing edema, and improving microcirculation.

Ethical Considerations

This study is a systematic review of previously published literature. Therefore, ethical approval from an institutional review board was not required.

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