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Review Article

A Comprehensive Review of Deep Vein Thrombosis: Risk Factors, Diagnosis, and Treatment

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Abstract:

Background: Deep vein thrombosis (DVT) is a serious vascular condition causing blood clots in deep veins, mainly in the lower limbs, with risks of pulmonary embolism, post-thrombotic syndrome, and chronic venous insufficiency. Despite advances in diagnosis and treatment, it remains a global health concern. This review explores its etiology, risk factors, symptoms, diagnosis, and management strategies.

Methods: A systematic review was conducted using PubMed, NIH, CDC, and other medical databases, analyzing peer-reviewed articles, clinical guidelines, and case studies on DVT.

Results and Discussion: DVT results from endothelial damage, venous stasis, and hypercoagulability, increasing the risk of pulmonary embolism. Key risk factors include age, immobility, surgery, cancer, pregnancy, obesity, smoking, and genetic predisposition. Diagnosis involves duplex ultrasonography, D-dimer testing, and CT pulmonary angiography, with the Wells Criteria aiding risk assessment. Treatment includes anticoagulants (LMWH, DOACs, warfarin), with thrombolytics and IVC filters for severe cases. Prevention focuses on mechanical compression, early ambulation, and lifestyle changes to reduce recurrence and improve outcomes.

Conclusion: DVT remains a significant health concern due to its potential complications, including life-threatening pulmonary embolism. Advances in diagnostic imaging and anticoagulant therapy have improved patient outcomes, but challenges remain in optimizing long-term management and recurrence prevention. Future research should focus on personalized treatment approaches, enhanced biomarker identification, and improved prophylactic strategies. A multidisciplinary approach involving early detection, appropriate intervention, and long-term risk management is essential for improving patient prognosis.

Keywords: Deep vein thrombosis, pulmonary embolism, thrombolysis,

Introduction

The development of thrombosis in the deep veins of the lower limbs or pelvis is known as deep vein thrombosis (DVT). Damage to the vessel endothelium results in slow blood flow, which encourages the production of blood clots and decreases venous blood flow. In extreme situations, this can lead to pulmonary embolism (PE) when the thrombi travel through the vasculature from the deep veins to the lungs early detection of DVT and appropriate anticoagulant therapy are crucial from a clinical standpoint since PE can be lethal in some situations. However, early diagnosis is clinically

hard since DVT can be asymptomatic and its clinical signs are nonspecific [1].

Epidemiology

The wide prevalence of pulmonary emboli along with deep vein thrombosis remains undetectable during life until physician discovery through autopsies. Studies usually downplay incidence and prevalence numbers. Research indicates DVT occurs in one of every thousand individuals while DVT develops in eighty people from every hundred thousand each year. Venous thrombosis affects over 200,000 people in the US annually and among these cases, pulmonary embolism develops in 50,000 patients

Age: Children are seldom affected by deep vein thrombosis, and the risk rises with age, with most cases happening in those over 40.

Gender: There is no unanimity regarding whether there is a sex bias in the prevalence of DVT.

Ethnicity: Based on data from the United States, African Americans and white people are more likely to have DVT and to experience complications than Hispanic and Asian populations.

Associated diseases: The most common conditions seen in hospitals are cancer, congestive heart failure, obstructive airway disease, and surgical patients. [2]

History

The earliest recorded mention of DVT has been traced in the Ayurvedic book Sushruta Samhita, which was written between 600 and 900 BC. The first case or Western reference to DVT has been attributed to a French text published in 1271 that documented DVT symptoms in a 20-year-old man's leg.

Rudolf Virchow, a German physician and pathologist, published his research findings in 1856 following the introduction of foreign substances into dogs' jugular veins, which then progressed to the pulmonary arteries. Virchow concentrated on addressing the implications of these foreign substances triggering pulmonary emboli. He mentioned three elements that are now known to be endothelial damage, stasis, and hypercoagulability [3].

In the 1960s, techniques for ultrasonography-based DVT observation were developed. In the 1970s and 1980s, impedance plethysmography was frequently used for diagnosis; however, ultrasound emerged as the favored diagnostic technique, especially when the usefulness of probe compression was shown in 1986. However, contrast venography and impedance plethysmography were still considered widespread in the mid-1990s.

The 20th century witnessed the beginning of several pharmacological treatments for DVT, including subcutaneous injections of LMWH in 1982, and oral anticoagulants in the 1940s. The idea that venous thrombosis and vascular inflammation are connected was first put out in 1974. The cornerstone of pharmaceutical therapy for around 50 years was a months-long warfarin (Coumadin) regimen. Direct oral anticoagulants (DOACs) were developed in order to circumvent the requirement for injections for heparin and heparin-like medications and the blood monitoring needed for warfarin. DOACs, such as dabigatran (Pradaxa), apixaban (Eliquis), and rivaroxaban (Xarelto), entered the market in the late 2000s and early 2010s. A "furious battle" between the three manufacturers of these medications "for the prescription pads of doctors" was reported by the New York Times [3].

Methodology

This review on deep vein thrombosis (DVT) was conducted through a comprehensive analysis of existing literature, including peer-reviewed journal articles, clinical studies, and authoritative medical sources. Relevant information was gathered from databases such as PubMed, the CDC, and NIH, focusing on the pathophysiology, risk factors, diagnostic techniques, and treatment strategies for DVT. Studies were selected based on their relevance, credibility, and recent advancements in the field.

The methodology also included an evaluation of the latest diagnostic imaging methods, anticoagulant therapies, and preventive measures. The findings were synthesized to present a structured overview of the disease, its complications, and ongoing research aimed at improving patient outcomes.

Discussion

Pathophysiology

Venous thrombosis typically manifests in regions where the hemodynamics are diminished or mechanically modified, such as the recesses adjacent to the valves within the deep venous system of the lower extremities. While venous valves promote the efficient circulation of blood, they concurrently may serve as loci for hypoxic conditions and venous stasis. Numerous postmortem studies have substantiated the propensity for venous thrombi to form in the sinuses adjacent to venous valves. A rise in hematocrit is concomitant with a reduction in oxygen tension as blood flow diminishes. The resultant hypercoagulable microenvironment may lead to the downregulation of particular antithrombotic proteins that are primarily expressed on venous valves, including thrombomodulin and endothelial protein C receptor (EPCR). Hypoxic conditions incite the synthesis of various procoagulant factors while concurrently diminishing critical anticoagulant proteins. Among these, P-selectin functions as an adhesion molecule that recruits immune cells containing tissue factor to the endothelial surface. Essentially, a venous thrombus is comprised of two distinct constituents: an external fibrin clot enriched with erythrocytes and an internal platelet-rich white thrombus that is characterized by the so-called lines of Zhan. The outer framework, constituted of fibrin and extracellular DNA complexed with histone proteins, may significantly influence the thrombus's susceptibility to thrombolytic therapy and tissue plasminogen activator (TPA). The likelihood of thrombus formation escalates with the procoagulant to anticoagulant ratio. The proportion of endothelial cell surface area to blood volume partially dictates the concentration of these proteins. Procoagulant factors are predominantly favored in large arterial systems, which exhibit a lower ratio of cell surface area to blood volume. Prothrombin, von Willebrand factor, factor VIII, and factor VII are believed to play a pivotal role in shifting the balance towards coagulation [4, 5].

Prothrombin not only facilitates the generation of thrombin but also attenuates the anticoagulant functionalities of activated protein C, which in turn obstructs a fundamental anticoagulant pathway. Three such mechanisms encompass the heparin-antithrombin pathway, the tissue factor inhibitor pathway, and the protein C anticoagulant pathway (which comprises protein C, protein S, thrombomodulin, and potentially EPCR). An elevated propensity for thrombus formation is associated with anomalies in these pathways. The role of the tissue factor inhibitor pathway in human physiology remains inadequately elucidated. Furthermore, certain hereditary polymorphisms elevate the concentrations of von Willebrand factor, prothrombin, factor VII, VIII, and IX, thereby predisposing the organism to thrombus development. Activated factor Via demonstrates resistance to the inhibitory influence of protein C in the context of factor V Leiden, which impacts up to 5% of Caucasian populations and augments the likelihood of thrombosis by a factor of seven. Additional contributors to the risk of clot formation include advancing age, obesity, the use of oral contraceptives, and malignancy. Venous stasis may arise from the compressive effects exerted by neoplasms on the venous structures. Additionally, it induces the release of procoagulants, such as tissue factor, from membrane particles, which further promotes thrombosis. Obesity and the consumption of oral contraceptives represent two distinct risk factors for thrombotic events. These factors synergistically elevate the risk of thrombosis [4, 5].

Risk factors

Both provoked and unprovoked deep vein thromboses (DVTs) are feasible occurrences.

While unprovoked thromboembolic events may indicate an elevated inclination towards coagulation, provoked thromboembolic events can be associated with recognized risk factors, the majority of which are temporally constrained. A significant proportion of DVTs identified in the emergency department (ED) are unprovoked, and they exhibit a greater likelihood of recurrence compared to provoked instances: 15% versus 5% over the subsequent 12 months.

Up to 80% of patients diagnosed with DVTs possess at least one, and oftentimes multiple, identifiable risk factor, these risk factors can be broadly classified as either hereditary or acquired[6].

Until they receive a formal diagnosis for their initial venous thromboembolism (VTE), individuals with hereditary thrombophilias are often unaware of their medical condition. Notably, despite the increased susceptibility of this demographic to VTE occurrences compared to the general population, their likelihood of recurrence mirrors that of individuals with unprovoked deep vein thromboses (DVTs). The prevalence of spontaneous thromboembolic events may be attributed to the undiagnosed nature of thrombophilias in many cases.

In comparison to Caucasians, African-Americans exhibit a prevalence of venous thromboembolism events (VTEs) that is 30% to 100% higher. Males demonstrate a greater propensity for experiencing recurrent deep vein thromboses (DVTs); nevertheless, there exists no discernible racial bias in the incidence of DVTs. The probability of developing DVTs increases with advancing age, partially attributable to the heightened occurrence of medical comorbidities and additional risk factors associated with DVTs among the elderly population. Both obesity and tobacco use have been correlated with an elevated risk for the development of DVT.

Acquired

DVTs can develop because of many risk factors, and over 50% of individuals with DVTs had multiple acquired risk factors. Moreover, depending on the underlying genetic risk, having it in addition to a significant medical condition or acquired risk factor raises the chance of DVT by an odds ratio of up to more than 80 [6].

Surgeries, Trauma, and Immobilization

Major orthopedic and neurovascular procedures, in specific, have been linked to a markedly increased risk of DVTs and PEs, particularly in patients who also have other risk factors including advanced age, a history of DVTs, or a medical condition. Extended periods of surgery and immobility following surgery are also linked to a higher incidence of DVTs. Because of immobility and anatomical risk, major and small trauma both significantly increases the risk of DVTs.

Depending on the technique, 5–11% of surgically caused DVTs return within 4 years. Long-term immobilization from air or land travel doubles or quadruples the risk of DVTs. DVT risk is also increased by immobilization brought on by other illnesses, such as stroke-related hemiplegic.

Prior Thromboembolism

Patients with spontaneous DVTs and those with hereditary or permanent risk factors are particularly at high risk for recurrence if they have a history of previous thromboembolic events. Both a history of PE and a history of DVT increase the likelihood of recurrent DVT [6].

Malignancy

Hypercoagulability is associated with neoplastic diseases. The thrombogenic capacity of an oncological patient is influenced by an array of factors. In general, the risk escalates with the dimensions of the tumor and the degree of cellular differentiation. Furthermore, the likelihood of thromboembolic incidents is exacerbated by the administration of certain chemotherapeutic agents, the utilization of

central venous catheters, and the necessity for surgical intervention in cancer cases. In the context of central venous catheters, the incidence of venous thrombotic events may ascend to 12%.

A pronounced risk of venous thromboembolism (VTE) is correlated with hematological malignancies (such as myeloproliferative neoplasms like leukemia and myelomas) as well as solid-organ tumors (including lung, pancreatic, colorectal, renal, and prostate cancers). The highest risk is particularly associated with multiple myeloma, acute leukemia, and metastatic cancers. An elevated thromboembolic propensity is also linked to various malignancies, including lymphoma, ovarian, gastric, renal, adenocarcinoma, glioblastoma, and metastatic melanoma, in addition to pancreatic cancer.

Clinically significant VTE occurs in approximately 10% of cases involving advanced breast cancer or breast cancer that has been subjected to chemotherapy. The induction phase of chemotherapy in malignant conditions is characterized by the highest risk of thrombosis, especially when agents such as fluorouracil, tamoxifen, or L-asparaginase are administered. The use of adjunctive erythropoiesis-stimulating agents (EPO) in chemotherapy elevates the risk irrespective of the tumor stage. Additionally, the administration of thalidomide or lenalidomide in the treatment of multiple myeloma has been identified as another contributory risk factor. Although the majority of malignancy-related VTEs are connected with recognized malignancies, thromboembolism can sometimes occur before malignancy is diagnosed. According to a Danish research, 78% of malignancies were identified before to the incident [6].

Pregnancy

The obstruction of the inferior vena cava by the uterus, coupled with a hypercoagulable state, renders pregnancy a significant risk factor for the development of deep vein thrombosis (DVT). Women who have undergone multiple pregnancies, as well as those in the postpartum period, exhibit the highest susceptibility to this condition. This risk is further exacerbated by the presence of additional contributing factors, such as tobacco use, diabetes mellitus, hypertension, obesity, hereditary thrombophilias, antiphospholipid antibodies, and advanced maternal age. Pregnant women are anticipated to experience an incidence of venous thromboembolism (VTE) that is age-adjusted to be 5 to 50 times higher than that of their non-pregnant counterparts.

Antiphospholipid Antibody Syndrome (APLS)

An elevated risk of arterial and venous thrombosis affecting any organ system is linked to the presence of antiphospholipid antibodies (APLA). The most prevalent and recurring thrombotic complication of APLS is DVTs. In one research, 14% of individuals with recurrent VTEs had APLAs.

Chronic Medical Conditions

The occurrence of deep vein thromboses has been directly linked to multiple health conditions

Cardiac: Dyslipidemia, hypertension, atherosclerosis, and heart failure

Renal: Microalbuminuria, nephrotic syndrome, chronic kidney disease, and renal transplant

The three blood-related health conditions associated with VTE development are hyperhomocysteinemia together with paroxysmal nocturnal hemoglobinuria and polycythemia vera.

Respiratory conditions: obstructive sleep apnea and asthma

Endocrine: diabetes mellitus, polycystic ovary syndrome

Iatrogenic

The use of birth control pills represents the foremost medication which increases DVT risk factors specifically among young female users. Postmenopausal women who take hormone

replacement medication show increased chances of developing deep vein thrombosis. The use of Heparin (heparin-induced thrombocytopenia) as well as testosterone and tamoxifen and glucocorticoids (especially systemic) and antidepressants has been associated with higher DVT risk to patients. The injection of medication through intravenous routes into lower extremities results in local stress on femoral veins along with irritation hence leading to DVT occurrence.

Inherited Risk Factors

Factor V Leiden mutation and prothrombin gene mutation are the most common inherited hypercoagulable disorders, contributing to nearly 50% of hereditary thrombophilic conditions that elevate DVT risk. Deficiencies in Protein C and Protein S often co-exist with Factor V Leiden mutation, and individuals may present with multiple hereditary thrombophilic disorders. Additionally, acquired risk factors can compound the risk in patients with hereditary thrombophilia. The likelihood of developing DVT significantly increases when multiple inherited thrombophilic conditions or a combination of acquired and genetic risk factors are present. Identified hereditary thrombophilic disorders include [6].

- Dysfibrinogenemia
- Factor XII deficiency
- Hyperhomocysteinemia
- Non-O blood group
- Protein S deficiency
- Antithrombin deficiency
- Factor V Leiden mutation
- Prothrombin gene mutation
- Protein C deficiency

Risk Factor Stratification

And the Wells Criteria functions as a well-known clinical instrument to evaluate DVT risk in patients while performing risk assessment instead of diagnostic confirmation. The tool provides direction on what diagnostic tests such as D-dimer testing or ultrasound Doppler imaging should be performed depending on which risk factors exist. A single point is assigned for each present factor among active cancer therapy or recent treatment, prolonged bed rest, major surgery within the past three months, swollen calfs or visible superficial veins or diffuse swelling of the lower limbs, deep vein tenderness or pitting edema or leg paralysis or immobilization and DVT prior to diagnosis. A different diagnosis that justifies deduction of two points. The scoring algorithm divides patient groups into three categories with low risk for patients scoring zero (5% probability) and medium risk for those with one to two points (17% risk) in addition to high risk for patients scoring three or above (risk range from 17% to 53%). Negative D-dimer results are sufficient to eliminate DVT in patients classified as low risk and single negative results are acceptable for medium-risk patients. Ultrasonography with Doppler is mandatory in high-risk patients yet additional scans might be necessary for verification. Negative results from both D-dimer testing and Doppler examinations eliminate the possibility of DVT across all groupings even when medical urgency determines patients need priority care [6, 7].

Diagnosis

The following tests are performed for DVT:

- **Duplex ultrasonography:** is an imaging technique that examines the veins' blood flow using sound waves. It is able to identify blood clots or obstructions in the deep veins. It is the typical imaging test used to identify DVT [8, 9].
- **D-dimer:** A blood chemical produced when a clot breaks up is measured by a D-dimer blood test. A negative D-dimer test indicates that the patient most likely does not have a blood clot.
- **Contrast venography:** A unique kind of X-ray, a major vein in the foot or ankle is injected with contrast material (dye) to allow the physician to view the deep veins in the leg and hip. Although it is the most reliable test for identifying blood clots, it is an intrusive process, meaning that medical professionals must enter the body with

devices. As a result, duplex ultrasonography has essentially superseded this test, which is now only utilized in specific individuals.

- **Imaging tests:** CT, MRI.

The following tests are performed for Pulmonary Embolism:

- **CTPA:** A unique kind of X-ray exam called computed tomographic pulmonary angiography(CTPA) involves injecting a contrast agent (dye) into a vein. Images of the lungs' blood arteries can be obtained using this examination. It is the typical imaging test used to identify PE.
- **The ventilation-perfusion (V/Q):** This scan is a specialized test that employs a radioactive material to show the areas of the lungs receiving blood flow (perfusion scan) and oxygen (ventilation scan) in order to determine whether there are any areas of the lungs where ventilation and perfusion differ. For instance, the V/Q scan may reveal normal oxygen levels but poor blood flow to the areas of the lungs that are serviced by the occluded blood vessels if there are clots in some of the blood arteries in the lungs. When CTPA is unavailable or should not be performed due to potential risks to the patient, this test is utilized.
- **Pulmonary angiography:** This a form of X-ray exam that involves inserting a large catheter (a long, thin hollow tube) into a big vein (often in the groin) and into the arteries of the lung, followed by the injection of contrast material (dye) through the catheter. It is the most reliable test for diagnosing PE and delivers pictures of the lung's blood vessels. It is only used on specific individuals, though, because it is an intrusive test.
- **MRI:** A magnetic field and radio waves are used in magnetic resonance imaging (MRI) to create pictures of the lung. However, this test is often only performed on specific individuals, such as pregnant women or those in whom the use of contrast material may be hazardous [8, 9].

Treatment

Anticoagulants and, in rare instances, thrombolytics are prescribed to nearly all patients with deep vein thrombosis (DVT). Deep vein thrombosis can be effectively managed with a variety of anticoagulants. Among the non-pharmacologic therapies are inferior vena cava filters and surgery.

Medications for DVT

1. **Anticoaggulants**

Low molecular weight heparins (LMWHs)

There exists a practice of choosing low molecular weight heparin (enoxaparin, dalteparin, or tinzaparin) as one of the initial treatment options because these drugs can provide outpatient care. The same protective effects against PE-related death together with thrombus growth and DVT recurrence are achieved by LMWHs at levels similar to unfractionated heparin (UFH). Rate of coagulation factor proteases inhibition by LMWHs functions similarly to UFH through their velocity-enhanced antithrombin mechanism yet they inactivate coagulation factors Xa and IIa to a lower degree. LMWHs demonstrate antithrombin-mediated anti-inflammatory actions which support clot formation while decreasing inflammatory conditions and resulting symptoms [10].

Unfractionated heparin (UFH)

External outpatient care is possible through the administration of low molecular weight heparins which include medications such as enoxaparin, dalteparin, or tinzaparin. Both unfractionated heparin and low molecular weight heparins show identical effectiveness in preventing mortal PE conditions together with thrombus extension and DVT recurrence. The anticoagulation properties of LMWHs function by speeding up antithrombin-mediated inhibition until it inactivates coagulation factors Xa and IIa at a subdued degree. The anti-inflammatory properties of LMWHs emerge

from their interactions with antithrombin to facilitate blood clot development while symptoms simultaneously decrease [10].

Factor Xa inhibitors

The treatment regimen with apixaban or rivaroxaban should start as single-agent therapy immediately following diagnosis. These drugs can be given at any time without interfering with each other to replace injectable heparin treatment. Rivaroxaban therapy starts with twice-daily 15 mg dosing for three weeks followed by daily 20 mg dosing which extends from three to six months. Doctors prescribe Apixaban in two daily 10 mg doses for seven days followed by three to six months on two daily 5 mg doses [10].

Vitamin K antagonists (warfarin)

Warfarin is suitable for DVT management of non-pregnant patients and this medication applies to patients with severe kidney failure too. Warfarin assumes its position as the second option for medical treatment of Venous Thromboembolism that develops alongside cancer conditions. Doctors can start warfarin (5–10 mg) directly since it takes heparin five days to develop its maximal therapeutic benefits. Clinicians must prescribe smaller amounts of warfarin to elderly patients and also those who have liver disease. The therapeutic aim during warfarin treatment requires patients to sustain international normalized ratio (INR) at a range from 2.0 to 3.0 while INR testing takes place weekly for the first one to two months and monthly afterward. The healthcare provider adjusts warfarin dosages from 0.5 to 3 mg to maintain an INR value within this target band. Warfarin-treated patients should receive information about medication interactions with both OTC remedies and food substances during treatment [10].

2. **Thrombolytic agents**

Thrombolytic drugs, including streptokinase, alteplase, and tenecteplase, can dissolve clots and may be more effective than heparin alone in select DVT patients, though they carry a higher risk of bleeding. Clinical studies indicate that catheter-directed thrombolytic therapy does not significantly reduce the risk of post-thrombotic syndrome compared to standard anticoagulation, making careful patient selection essential. Thrombolytic therapy may be beneficial for younger patients (under 60) with severe iliofemoral DVT who have no bleeding risk factors. It should also be strongly considered in cases of limb ischemia, such as phlegmasia cerulea dolens. For patients with massive pulmonary embolism (PE) associated with cardiogenic shock, respiratory failure, or systemic hypotension (systolic BP < 90 mm Hg), thrombolytic therapy should be evaluated. However, it is generally not recommended for most cases of submassive PE unless the patient's condition worsens despite anticoagulant treatment. Routine use is discouraged in individuals with right ventricular dysfunction and submassive PE [10].

B. **Inferior vena cava (IVC) filter**

An inferior vena cava (IVC) filter is placed just below the renal veins via catheterization of the internal jugular or femoral vein, with some filters designed for temporary use until anticoagulation contraindications resolve. IVC filters can help prevent pulmonary embolism (PE) in patients with lower extremity DVT who cannot receive anticoagulants or those experiencing recurrent DVT despite adequate anticoagulation. While they reduce the risk of acute embolic events, they may increase the likelihood of recurrent DVT and long-term complications, such as the formation of venous collaterals that allow emboli to bypass the filter. Additionally, filters can become clogged or dislodged, leading to complications like venous congestion, lower body ischemia, or acute kidney injury. In cases of filter dislodgement, removal via angiographic or surgical intervention may be necessary. Despite their widespread use, the effectiveness of IVC filters in preventing PE remains uncertain, and they should be removed whenever possible to minimize risks [11].

C. **Surgery**

Surgery is rarely required but may be necessary in severe cases to prevent limb-threatening gangrene. When phlegmasia alba dolens or phlegmasia cerulea dolens does not respond to thrombolytic therapy, surgical interventions such as thrombectomy, fasciotomy, or both may be needed to restore blood flow and prevent tissue loss.

Prevention

- In order to avoid pulmonary embolism, which might result in major problems, deep vein thrombosis must be prevented.
- In order to avoid deep vein thrombosis, some surgical patients may be prescribed medications, such as anticoagulants. Patients who have previously had a clot should adhere to their doctor's advice.
- Moving the lower leg helps prevent deep vein thrombosis brought on by extended sitting or recline. Knee flexion, or flexion, may be beneficial.

Additional preventive actions might consist of:

- Following surgery or sickness, getting up and moving as soon as possible is important because exercise promotes blood circulation, which helps prevent clots from developing.
- During certain surgeries, a pneumatic compression device—which resembles a specially fitting sleeve—is applied to the legs to assist keep the blood flowing.
- Elastic stockings to encourage circulation and lessen edema
- See your doctor for a diagnosis and course of treatment.

Preventing deep vein thrombosis in hospital inpatients

Fondaparinux may provide additional options for DVT prevention, though the effectiveness of aspirin remains uncertain. Physicians should follow established guidelines to ensure proper prophylaxis against DVT, particularly for hospitalized patients, to reduce the risk of both fatal and non-fatal pulmonary embolism and post-thrombotic complications. For individuals at low risk, early ambulation is essential, while mechanical prophylactic measures, such as intermittent pneumatic compression devices and graded compression stockings, can offer additional protection. Patients at higher risk should be assessed for anticoagulation therapy using low molecular weight heparin, unfractionated heparin, or vitamin K antagonists, unless contraindicated [12].

Conclusion

Deep vein thrombosis (DVT) remains a significant health concern, impacting both patients and healthcare systems. Early detection and timely intervention are crucial in preventing complications such as chronic venous insufficiency, post-thrombotic syndrome, and pulmonary embolism. Advances in diagnostic imaging, biomarkers, anticoagulant therapies, and mechanical treatments have improved DVT management, yet challenges persist in preventing recurrence, identifying high-risk individuals, and optimizing the duration of anticoagulant therapy. Continued research is essential to enhance understanding of DVT pathophysiology and develop more targeted, effective treatment strategies. Ultimately, a multidisciplinary approach—focusing on early prevention, accurate diagnosis, and personalized treatment—is key to improving patient outcomes.

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