International Journal for Multidisciplinary Research (IJFMR)
E-ISSN: 2582-2160 ● Website: www.ijfmr.com ● Email: editor@ijfmr.com

Deep Vein Thrombosis

Dr. Anjum Abbasi[1], Mr. Devendra[2], Ms. Ritu[3]

[1]P.hD, MSN, BSN Assistant Professor Galgotias University School of Nursing
[2,3]Assistant Professor, Galgotias University School of Nursing

Abstract
Deep vein thrombosis (DVT) is a major preventable cause of morbidity and mortality worldwide. Venous thromboembolism (VTE), which includes DVT and pulmonary embolism (PE), affects an estimated 1 per 1,000 people and contributes to 60,000–100,000 deaths annually. Normal blood physiology hinges on a delicate balance between pro- and anti-coagulant factors. Virchow's Triad distills the multitude of risk factors for DVT into three basic elements favoring thrombus formation: venous stasis, vascular injury, and hypercoagulability. Clinical, biochemical, and radiological tests are used to increase the sensitivity and specificity for diagnosing DVT. Anticoagulation therapy is essential for the treatment of DVT. With few exceptions, the standard therapy for DVT has been vitamin K-antagonists (VKAs) such as warfarin with heparin or fractionated heparin bridging. More recently, a number of large-scale clinical trials have validated the use of direct oral anticoagulants (DOACs) in place of warfarin in select cases. In this review, we summarize the pathogenesis, diagnosis, and medical management of DVT, with particular emphasis on anticoagulation therapy and the role of DOACs in the current treatment algorithm.

Keywords: Deep vein thrombosis (DVT), vitamin K-antagonists (VKAs), warfarin, direct oral anticoagulants (DOACs), anticoagulants

Introduction:
Deep vein thrombosis (DVT), a subset of venous thromboembolism (VTE), is a major preventable cause of morbidity and mortality worldwide. The incidence of VTE is estimated to be 1 per 1,000 people annually (1,2), with DVT accounting for approximately two-thirds of these events (3). Pulmonary embolism (PE), a dreaded complication of DVT, occurs in up to one-third of cases and is the primary contributor to mortality (4). Much of the morbidity of DVT results from the development of post-thrombotic syndrome, which occurs in up to 50% of patients within 2 years of DVT and encompasses a number of symptoms including leg pain, swelling, and in severe cases, venous ulcers (5,6). Anticoagulation is the mainstay of therapy for DVT, with the goal of preventing progression to PE and recurrence of thrombosis. The 30-day mortality rate exceeds 3% in patients with DVT who are not anticoagulated, and this mortality risk increases 10-fold in patients who develop PE (7). The advent of direct oral anticoagulants (DOACs) has generated a need to compare these newer agents with the more conventional vitamin K-antagonists (VKAs) for the treatment of DVT. Several recent clinical trials have addressed this question and demonstrated a similar safety and efficacy profile between the two drug classes. With more therapeutic options, clinicians are now better able to incorporate disease- and patient-specific considerations into the medical management of DVT.

Goals:

Explain the results of the patient's assessment, the evaluation procedure, and any recommended laboratory testing for a patient who may have deep vein thrombosis.

Provide an overview of deep vein thrombosis management and treatment options, including precautions.

Describe the assessment and treatment of deep vein thrombosis, as well as the role that interprofessional team members play in working together to improve patient outcomes and deliver well-coordinated care.

Etiology Hazard Elements

The risk factors listed below are thought to be the root causes of deep vein thrombosis:

Reduced blood flow: bed rest, general anesthesia, surgery, stroke, extended flights, and immobility[8].[9]

Elevated venous pressure can result from a tumor, pregnancy, stenosis, or congenital defect that increases outflow resistance, or from mechanical compression or functional impairment that reduces vein flow.[10]

Trauma, surgery, peripherally implanted venous catheters, prior DVT, and intravenous drug usage are examples of mechanical vein injuries.[11][12][11]

Increased blood viscosity due to dehydration, thrombocytosis, and polycythaemia rubra vera[13]

Thrombosis may be caused by anatomic differences in the venous architecture.

Higher Chance of Coagulation

Genetic deficiencies: factor V Leiden mutation, antithrombin III deficiency, and anticoagulation proteins C and S[14][15][16]

Acquired: Nephrotic syndrome, burns, oral estrogens, smoking, hypertension, diabetes, vasculitis, myocardial infarction, heart failure, cancer, sepsis, and systemic lupus erythematosus and lupus anticoagulant

Constitutional Elements

The known causes of DVT and VTE include obesity, pregnancy, being older than 60, surgery, critical care hospitalization, dehydration, and malignancy.[18][19][20][21]

Two factors contribute to the association between obesity and a hypercoagulability status: 1. elevated fibrinogen levels that may even double the normal value, and 2. slowed venous circulation flow, particularly in the lower extremities and the infra diaphragmatic.[20] Both factors, which are linked to abnormalities in a number of coagulation factors, increase the risk of thromboembolic events, thrombophlebitis, and venous thrombosis. They also increase the risk of pulmonary thromboembolisms (PE), which are the main cause of death for obese people.

It is possible to classify potential risk factors for deep vein thrombosis using the temporary, persistent, or unprovoked criteria. Consequently, the following are temporary risk factors: 1. general anesthesia surgery; 2. hospital stay; 3. cesarean section; 4. hormone replacement therapy; 5. pregnancy and the postpartum phase; and 6. lower extremity injuries resulting in limited mobility lasting more than 72 hours.[4]

Persistent risk factors include certain medical disorders and active malignancies that raise the chance of venous thromboembolism. Among the medical problems that predispose people include inflammatory bowel disease and systemic lupus erythematosus.

Unprovoked venous thromboembolism should be the term used to describe any additional etiological risk factors that are not classified into either transitory or persistent groupings. (VTE)[4][1] For example, a recent cohort study examining the relationship between blood lipid levels and lower extremity deep vein thrombosis (LEDVT) with 500 participants showed that increased levels of total cholesterol, high-density lipoprotein (HDL-C), and apolipoprotein A1 (ApoA1) were linked to a lower risk of LEDVT. Nonetheless, a higher risk of LEDVT was linked to higher triglyceride (TG) levels.[1]

Access: Epidemiology

Prevalence and incidence: Pulmonary emboli and deep vein thrombosis are frequent, frequently "silent" conditions that are not recognized or discovered until after death. As a result, it is common to underestimate the incidence and prevalence. It is estimated that 80 instances of DVT occur annually.cases per 100,000 people, with an incidence of one case per 1000 people for DVT in the lower leg.More than 200,000 **Age:** The risk of deep vein thrombosis rises with age, with the over-40 age group having the highest incidence.

Gender: Whether or not there is a sex bias in the occurrence of DVT is a topic of debate.

Ethnicity: Research from the United States indicates that African Americans and Whites had a higher incidence of DVT and a higher risk of complications when compared to Hispanics and Asians.

Associated diseases: Patients having surgery, congestive heart failure, obstructive airway disease, and cancer are the most often occurring associated ailments in hospitals.

Pathophysiology.

Thrombosis is a defense mechanism that closes off injured blood arteries and stops blood loss. Thrombosis is neutralized or stabilized by fibrinolysis. The causes of venous thrombosis are often complex, with each patient's contribution to the Virchow triad being varied, yet they always lead to an early thrombus contact with the endothelium.

This promotes venous thrombosis by inducing leukocyte adherence to the endothelium and stimulating the generation of local cytokines. Thrombus propagation is contingent upon the relative equilibrium between the coagulation and thrombolytic pathways. The most prevalent location for DVT is in the lower limb below the knee. It begins in low-flow locations behind venous valve pockets, like the soleal sinuses.

It has been suggested that DVT and atherosclerosis (AS) may be related.[1] AS could be brought on by the endothelial dysfunction that is a part of the pathophysiological mechanism of DVT. Consequently, patients with DVT are expected to have a higher chance of developing AS later on.[1] .

It is well known that relying just on clinical signs and symptoms to diagnose DVT is incredibly incorrect. DVT symptoms and indicators are typically non-specific. They could be confused with and misdiagnosed as other illnesses affecting the lower extremities. Thus, cellulitis, lymphedema, and superficial vein thrombosis should be ruled out.. A sensitivity of 75% to 91% and a specificity of 3% to 87% characterize the former index, while a sensitivity of up to 97% and a specificity of up to 88% characterize the latter.[29] Neither one of the symptoms or indicators, by itself or in combination, is sensitive enough or specific enough to correctly identify or rule out thrombosis.

Patients with a score of 0 to 1 have a low clinical probability; patients with a score of two or more have a high clinical probability.

If a patient receives a score of two or higher, they should have a D-dimer test performed if the results of

the proximal leg vein ultrasonography scan are not positive within four hours. A 24-hour dosage of a parenteral anticoagulant should be administered in the interval and a D-dimer test should be performed if imaging cannot be completed in four hours. Within 24 hours following the request, an ultrasound scan of the proximal leg vein should be performed.

All patients should have a proximal leg vein ultrasound scan performed six to eight days after a negative result, provided the D-dimer test was positive.

A proximal leg vein ultrasound scan should be performed on the patient within four hours if the D-dimer test is positive but the patient does not score two on the DVT Wells score. If this cannot be done, the patient should receive an interim 24-hour dosage of a parenteral anticoagulant.Within 24 hours following the request, an ultrasound scan of the proximal leg vein should be performed.

When a patient presents with a suspected DVT, clinical decision guidelines such the Wells Criteria and the Pulmonary Embolism Rule-Out Criteria (PERC) should be used. A key consideration when choosing a diagnosis and course of treatment is risk stratification. Patients who fit the PERC criteria might not require additional testing, while those who don't fit the criteria and have a low probability according to the Wells criteria might be candidates for D-dimer rule-out. It is advisable to use the test cautiously, maybe adjusting the cut-off values for senior individuals.

Diagnostic ultrasound, vascular studies, CT venograms, and point-of-care ultrasound (POCUS) are among the imaging modalities that can be used to assess for DVT. The following describes the POCUS exam.

When 24-hour ultrasonography is not accessible, the emergency provider's quick diagnosis or rule-out might speed essential therapy and shorten the patient's stay. There is proof that emergency physicians are capable of conducting a two-point compression exam at the popliteal and femoral veins, which have the highest likelihood of detecting a deep vein thrombosis (DVT). Recent research, however, indicates that a two-region strategy using repeated compression testing by doctors may considerably increase diagnostic sensitivity without appreciably lengthening the diagnostic process.

The point-of-care ultrasound exam is most helpful in patients with high and low pre-test likelihood, and it should be used in conjunction with other clinical decision guidelines.

Apply 20 to 30 degrees of reverse Trendelenburg while the patient is supine in the frog-leg position in order to enhance venous distention. The high-frequency linear transducer (5 to 10 MHz) should be positioned at the inguinal ligament's anatomical location in the transverse plane. One can see the common femoral vein just distal to the inguinal ligament. Directly press against the vein. There isn't a DVT present when the vein completely collapses. Proceed a short distance along the femoral vein to the point where the deep femoral vein and greater saphenous vein diverge from the common femoral vein.A proximal DVT is ruled out by complete compression of all venous structures at these levels.

Then go on to the popliteal area. In the popliteal fossa, position the high-frequency transducer transversely after flexing the knee and laterally rotating the leg. Usually, the popliteal vein is located directly in front of the popliteal artery. Once more, apply a compressive force and check for total compression. Complete the two-region method by compressing the areas immediately proximal and distal to the popliteal fossa.

D-dimer test could be sufficient as an alternative.

Standard laboratory tests ought to be sent as well to assess renal function, blood count, and coagulation status.

Management / Treatment

The goals of DVT treatment are to lower morbidity, avoid or reduce the risk of pulmonary embolism, and prevent or minimize the development of post-thrombotic syndrome.

Anticoagulation is the mainstay of treatment. According to NICE guidelines, only patients with pulmonary emboli and proximal DVT—not distal DVT—should be treated. The advantages and disadvantages of anticoagulation must be considered for each patient.[36][37][38]

The primary approach to treating DVT should be based on the underlying cause of the condition, which is as follows:

When treating cancer-associated thromboembolism, low molecular weight heparin and factor Xa inhibitors, such as rivaroxaban, are the recommended anticoagulants.[39] However, the greater levels of anticoagulation should be taken into account in the following situations: 1. newly discovered cancer; 2. severe VTE conditions; and 3. unfavorable side effects from cancer treatments, such as nausea.[40]

When once-daily oral medication is the recommended course of action, the following choices are feasible: 1. rivaroxaban; 2. edoxaban; 3. antagonist of vitamin K (VKA)

When liver illness is present, low-molecular-weight heparin should be used to treat DVT.[41] When INR levels are elevated, direct oral anticoagulants (DOACs) should not be used.

LMWH is the recommended anticoagulation therapy during pregnancy because most anticoagulants have the ability to pass the placenta.

Furthermore, the instructions that follow describe how long treatment must last.

Fondaparinux or low-molecular-weight heparin for five days or until the INR is more than two for twenty-four hours (unfractionated heparin for individuals with elevated risk of bleeding and renal failure) antagonists of vitamin K for three months

Low-molecular-weight heparin anticoagulation for six months should be considered for cancer patients.

If the patient has spontaneous DVT, vitamin K antagonists should be considered after three months.

The FDA and NICE recently approved rivaroxaban, an oral factor Xa inhibitor that is appealing because it does not require routine INR monitoring.

Change from heparin to fondaparinux, which is not linked to heparin-induced thrombocytopenia, if the platelet count falls to less than 75,000.

Thrombolysis: The following conditions can be treated with thrombolytic medication:

Indications of iliofemoral DVT symptoms lasting fewer than 14 days

good state of function

a minimum of one year left to live

minimal chance of bleeding

Because cerebral bleeding might occur from the use of thrombolytic treatment, patient selection must be done carefully. Endovascular procedures such as mechanical thrombectomy, stenting, and catheter-directed extraction have been attempted recently, with varying degrees of success.

Compression stockings: If there are no contraindications, below-knee graduated compression stockings with an ankle pressure greater than 23 mm Hg for two years

Vena cava inferior filters: When anticoagulation is not recommended or when emboli are happening even with appropriate anticoagulation

Patients with clinically active malignancy would benefit from thromboprophylaxis due to the increased risk of thrombosis. Further evidence that thromboprophylaxis reduced the risk of VTE in cancer patients receiving chemotherapy or surgery was provided by a meta-analysis examining 33 trials and 11,972

patients. There was no discernible increase in the incidence of serious bleeding.[7]

According to the most recent National Comprehensive Cancer Network (NCCN) guidelines, hospitalized cancer patients should receive anticoagulation as thromboprophylaxis using unfractionated heparin or low molecular weight heparin. When a pan atient has indwelling neuraxial catheter, is suffering active bleeding, has thrombocytopenia (platelet count < 50,000/MCL), or shows signs of hemorrhagic coagulopathy, mechanical prophylaxis should be utilized instead of anticoagulant medication. Severe arterial insufficiency and acute deep vein thrombosis are contraindications to mechanical prophylaxis.

Regarding outpatient VTE prophylaxis, surgical pelvic or abdominal oncology patients would benefit from continuing VTE prophylaxis up to four weeks post-operation. The use of aspirin or anticoagulation therapy for patients with multiple myeloma on immunomodulatory medications is recommended based on risk stratification with the IMPEDE VTE score.[9][10] For patients with solid cancers on chemotherapy and high Khorana score, prophylactic anticoagulation with direct oral anticoagulation or low molecular weight heparin showed a decrease in the incidence of pulmonary embolism

The treatment of choice for cancer-related thrombosis when it comes to anticoagulation is still low molecular weight heparin (LMWH).[12] One mg/kg every 12 hours or once daily for patients with a creatinine clearance of less than 30 mL/minute is the recommended dosage for LMWH. Refrain from administering LMWH to dialysis patients. Direct oral anticoagulants such as warfarin, fondaparinux, apixaban, rivaroxaban, and edoxaban are among the other therapeutic possibilities. Researchers discovered that apixaban was equally effective as LMWH in treating cancer-associated VTE in the Caravaggio study, with no discernible increase in bleeding risk.[13]

Patients with acute deep vein thrombosis or pulmonary emboli that pose a risk to life or limb may benefit from thrombolytic therapy, however there are certain contraindications, including brain tumors or metastases, ongoing bleeding, and a history of intracranial hemorrhage.

Provoked: As a result of acquired conditions (disease, immobilization, trauma, oral contraceptives, surgery, etc.).

Difficulties

The two main DVT complications are as follows:

Heart emboli (paradoxical emboli in the event of an atrial-septal defect)

Syndrome after thrombosis

bleeding resulting from anticoagulant use

Visit:

Care Following Surgery and Rehabilitation

Pharmacological and mechanical arms are two types of thromboprophylaxis techniques used for hospitalized patients who may be at risk of deep vein thrombosis. When it comes to the pharmaceutical side of things, one should take into account unfractionated and low-molecular-weight heparins, novel oral anticoagulants (NOACs) such dabigatran, rivaroxaban, and direct thrombin binders like dabigatran, as well as aspirin or warfarin.[20][48][49]

Note that in the extended treatment scenarios, NOACs are an effective treatment for thromboprophylaxis of VTE or VTE-related mortality, according to the results of the network meta-analysis. The bleeding risk consequence is different, though.

As a result, it was said that apixaban had the best profile. The comparison was released using aspirin, warfarin, and endpoints of INR 2.0 to 3.0 for each of the NOACs.[49][50][51]

The following actions in the mechanical prophylactic arm ought to be assessed:

Compression stockings with gradations
Devices for intermittent pneumatic compression
pumps for veinous feet
gadgets for electrical stimulation
Nevertheless, mechanical devices must to be used in addition to pharmaceutical prophylaxis.[20]
Navigate to: Patient Education and Deterrence
Accusation
Put on compression undergarments.
Give up smoking.
Improving the Results of Healthcare Teams
Deep vein thromboses (DVTs) can happen in practically any kind of medical specialization and in a variety of circumstances. If DVT is not diagnosed, it can lead to a potentially catastrophic pulmonary embolus. DVTs can lead to longer hospital stays and medication treatments that can run anywhere from three to nine months, which raises the overall cost of healthcare. Therefore, an interdisciplinary healthcare team made up of physicians (MDs, DOs, NPs, and PAs), specialists, nurses, physical therapists, vascular technicians, and pharmacists is ideally suited for diagnosing and managing it.
The prevention of DVT is the main concern. When it comes to teaching patients about DVT prevention, nurses and pharmacists play a crucial role alongside clinicians.
Healthcare professionals should abide by the DVT prophylaxis and treatment policies of each hospital. The pharmacist should be knowledgeable about the current anticoagulants and their indications if a DVT has formed.
Once DVT is identified, the patient is treated for three to six months with an anticoagulant, and hematology nurses or pharmacists are required to monitor the INR during this time. These patients also require bleeding control monitoring.
The interprofessional team must communicate openly in order to safely treat DVT and reduce the drug's morbidity.

Results

Every year in the US alone, pulmonary emboli claim the lives of almost 300,000 individuals. In spite of numerous standards and training for healthcare personnel, DVT prevention is frequently neglected. The majority of patients have preventable DVT, and it is the responsibility of healthcare professionals to be informed about this condition. If a person has a DVT and survives, post-thrombotic phlebitis is a chronic consequence that cannot be cured.[53][54] For this reason, ongoing management of these patients requires open communication and interdisciplinary care coordination.

References

1. R. C. Auer et al., “Use of helical CT is associated with an increased incidence of postoperative pulmonary emboli in cancer patients with no change in the number of fatal pulmonary emboli,” J. Am. Coll. Surg., vol. 208, no. 5, pp. 871–878, May 2009
2. G. Le Gal et al., “Risk for recurrent venous thromboembolism in patients with subsegmental pulmonary embolism managed without anticoagulation: a multicenter prospective cohort study,” Ann. Intern. Med., vol. 175, no. 1, pp. 29–35, Jan. 2022.
3. A. Bariteau, L. K. Stewart, T. W. Emmett, and J. A. Kline, “Systematic review and Meta-analysis of

outcomes of patients with subsegmental pulmonary embolism with and without anticoagulation treatment," Acad. Emerg. Med., vol. 25, no. 7, pp. 828–835, Jul. 2018

4. S. M. Stevens et al., "Executive summary: antithrombotic therapy for VTE disease: second update of the CHEST guideline and expert panel report," Chest, vol. 160, no. 6, pp. 2247–2259, Dec. 2021,
5. S. Schulman and C. Kearon, "Definition of major bleeding in clinical investigations of antihemostatic medicinal products in non-surgical patients," J. Thromb. Haemost., vol. 3, no. 4, pp. 692–694, Apr. 2005,
6. S. Kaatz, D. Ahmad, A. C. Spyropoulos, and S. Schulman, "Definition of clinically relevant non-major bleeding in studies of anticoagulants in atrial fibrillation and venous thromboembolic disease in non-surgical patients: communication from the SSC of the ISTH," J. Thromb. Haemost., vol. 13, no. 11, pp. 2119–2126, Nov. 2015
7. P. Prandoni et al., "Recurrent venous thromboembolism and bleeding complications during anticoagulant treatment in patients with cancer and venous thrombosis," Blood, vol. 100, no. 10, pp. 3484–3488, Nov. 2002,
8. A. T. Cohen, A. Katholing, S. Rietbrock, L. Bamber, and C. Martinez, "Epidemiology of first and recurrent venous thromboembolism in patients with active cancer. A population-based cohort study," Thromb. Haemost., vol. 117, no. 1, pp. 57–65, Jan. 2017
9. Yan, R. Kieser, C. C. Wu, W. Qiao, and C. M. Rojas-Hernandez, "Clinical factors and outcomes of subsegmental pulmonary embolism in cancer patients," Blood Adv., vol. 5, no. 4, pp. 1050–1058, Feb. 2021,
10. N. Kraaijpoel et al., "Treatment and long-term clinical outcomes of incidental pulmonary embolism in patients with Cancer: an international prospective cohort study," J. Clin. Oncol., vol. 37, no. 20, pp. 1713–1720, Jul. 2019, H. Robert-Ebadi and M. Righini, "The 2019 ESC guidelines on pulmonary embolism: some further insights," Eur. J. Intern. Med., vol. 77, pp. 6–8, Jul. 2020