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Deep Vein Thrombosis and Pulmonary Embolism: A Shared Postoperative Concern in Prolonged Immobilised Patients

Dr Prithvansh Malhotra[1], Dr RK Shinde[2], Dr Pooja Batra[3], Dr Muskaan Singhal[4], Dr Jaideep Reddy R[5]

[1]General Surgery, JNMC, Wardha, Maharashtra, India
drprithvanshmalhotra@gmail.com
[2]HOD and Professor, General Surgery, JNMC, Wardha, Maharashtra, India
raju.shinde95@gmail.com
[3]Professor, MS General Surgery, JNMC, Wardha, Maharashtra, India
[4]Obstetrics and Gynaecology, NIMS, Jaipur, Rajasthan, India
singhalmuss.3011@gmail.com
[5]General Surgery, JNMC, Wardha, Maharashtra, India
jaideeepreddy312@gmail.com

ABSTRACT

Venous thromboembolism (VTE), encompassing deep vein thrombosis (DVT) and pulmonary embolism (PE), represents a major preventable cause of morbidity and mortality among postoperative and prolonged immobilized patients. Despite advances in surgical care, anesthesia, and anticoagulant therapy, VTE continues to be a significant global health challenge. Postoperative immobility, endothelial injury, and hypercoagulability—collectively known as Virchow's triad—remain the cornerstone mechanisms driving thrombus formation. Surgical stress, tissue trauma, and prolonged bed rest further exacerbate venous stasis and clot propagation. Clinically, DVT often presents with unilateral leg swelling and pain, while PE manifests with dyspnea, chest pain, or hemodynamic instability, although many cases remain asymptomatic until complications arise. Early diagnosis through clinical scoring systems, D-dimer assays, ultrasonography, and computed tomography pulmonary angiography is crucial to prevent fatal outcomes. Preventive strategies, including pharmacologic prophylaxis with heparins or direct oral anticoagulants, mechanical compression, and early mobilization, have proven highly effective. However, variations in clinical practice, underdiagnosis, and inadequate adherence to prophylactic protocols persist, particularly in resource-limited settings. Future research should emphasize risk-based individualized prevention, development of national guidelines, and incorporation of artificial intelligence for improved prediction and early detection. A multidisciplinary approach integrating clinicians, nurses, and physiotherapists is essential to minimize postoperative VTE burden and improve patient outcomes.

KEYWORDS: Venous thromboembolism, Deep vein thrombosis, Pulmonary embolism, Postoperative immobility, Prophylaxis.

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INTRODUCTION

Venous thromboembolism (VTE) encompasses two interrelated clinical conditions—deep vein thrombosis (DVT) and pulmonary embolism (PE)—that together represent one of the most preventable yet life-threatening postoperative complications worldwide [1]. DVT involves the formation of a thrombus within the deep veins, typically of the lower limbs, whereas PE occurs when a portion of this thrombus dislodges and travels through the venous circulation to lodge in the pulmonary arteries, obstructing blood flow and impairing oxygen exchange [2]. Collectively, these complications are considered a significant global cause of morbidity and mortality, ranking third among cardiovascular disorders after myocardial infarction and stroke [3]. Despite advances in surgical techniques, anesthetic safety, and anticoagulant therapy, VTE continues to pose a serious threat to postoperative patients, particularly those who remain immobilized for prolonged periods following major surgical interventions [4].

Surgical procedures inherently create an environment conducive to thrombosis due to prolonged anesthesia, tissue trauma, and postoperative immobility. During and after surgery, the vascular endothelium may sustain injury, blood flow can become stagnant, and hypercoagulable states may be induced—all components of the pathophysiological concept known as Virchow's triad [5]. These changes collectively promote thrombus formation within the venous system. Once formed, the thrombus can either remain localized in the lower limb veins or dislodge and travel to the lungs, resulting in pulmonary embolism—a potentially fatal event [6].

Prolonged postoperative immobilization further exacerbates venous stasis, slowing the return of blood from the lower extremities and promoting the accumulation of coagulation factors. Reports suggest that a considerable proportion of surgical patients may develop subclinical or symptomatic DVT without adequate prophylaxis, particularly in high-risk populations such as orthopedic or neurosurgical cases [7]. The risk of PE following surgery can reach alarming levels in patients who undergo prolonged procedures or experience delayed mobilization during recovery [8]. In many low-resource settings, including several regions in India, the challenge is compounded by limited diagnostic facilities, reduced awareness among healthcare personnel, and inconsistent implementation of prophylactic protocols [9]. Hence, understanding the shared postoperative mechanisms and risk factors underlying DVT and PE remains vital for effective prevention and management.

The development of DVT and PE is governed by the interplay of venous stasis, endothelial injury, and hypercoagulability [10]. During surgical intervention, vascular injury exposes subendothelial tissue and triggers platelet activation and coagulation cascades. Postoperative immobility contributes to venous stasis by reducing the muscle pumping action that normally propels venous blood back to the heart. Additionally, the surgical stress response stimulates inflammatory mediators and prothrombotic factors, further enhancing coagulability [11].

In immobilized patients, venous pooling in the lower limbs provides an ideal environment for fibrin deposition and thrombus propagation. If the clot remains adherent to the vessel wall, it can cause local pain and swelling; however, once detached, it can travel through the right side of the heart and obstruct pulmonary arteries, leading to PE. The severity of PE depends on the size and number of emboli, with large occlusions potentially causing sudden cardiovascular collapse and death. These processes underscore the continuum between DVT and PE, emphasizing that both represent different stages of the same thromboembolic spectrum [12].

The risk of thromboembolism is particularly high in patients undergoing orthopedic, thoracic, abdominal, and neurosurgical procedures due to extensive tissue manipulation, long operative times, and restricted mobility during recovery. The postoperative phase, especially within the first 10 days after surgery, is considered the most critical window for VTE development. Prolonged immobilization is one of the most significant and modifiable risk factors for VTE [13]. When a patient remains confined to bed after surgery, venous return from the legs decreases, resulting in stasis and hypoxia within the venous endothelium. The lack of muscle contraction impairs the calf-muscle pump mechanism, encouraging thrombus formation. The risk increases proportionally with the duration of immobility and the extent of surgical trauma.

Other contributing risk factors include advanced age, obesity, malignancy, dehydration, hormonal therapy, smoking, preexisting cardiovascular disease, prior history of VTE, and inherited thrombophilias. The use of certain medications, including oral contraceptives or hormone replacement therapy, may further predispose individuals to hypercoagulable states [14]. In surgical settings, additional risks arise from procedures involving prolonged anesthesia or mechanical ventilation. Orthopedic surgeries such as hip or knee replacements are among the most common contexts for postoperative DVT, while abdominal and thoracic surgeries also present high risks due to restricted postoperative mobility and the physiological stress response to surgery. Immobilized patients in critical care units, those with fractures requiring casting, and those with neurological deficits also demonstrate elevated susceptibility. Even when pharmacological prophylaxis is employed, incomplete adherence or delayed initiation can allow thrombus formation to occur.

The clinical manifestations of DVT are often subtle and nonspecific. Common symptoms include leg swelling, pain, warmth, tenderness, and erythema, but these findings may be absent in up to half of affected individuals [15]. Consequently, many cases remain undetected until complications such as pulmonary embolism occur. PE typically presents with acute shortness of breath, pleuritic chest pain, tachycardia, cough, or hemoptysis, but these symptoms can mimic other postoperative complications such as pneumonia or cardiac dysfunction. Diagnosing DVT and PE in postoperative patients is particularly challenging because physiological postoperative changes can obscure clinical findings. Diagnostic evaluation usually begins with a combination of clinical scoring systems, such as the Wells or Geneva criteria, followed by objective testing. Duplex ultrasonography is the standard noninvasive method for detecting DVT in the lower limbs, while computed tomography pulmonary angiography remains the gold standard for diagnosing PE. D-dimer testing can be helpful in excluding VTE in low-risk patients, but postoperative elevation of D-dimer due to surgical trauma limits its specificity. These diagnostic complexities highlight the importance of preventive measures and risk assessment before surgery to minimize postoperative VTE events.

The aim of this paper is to comprehensively review the pathophysiology, risk factors, clinical manifestations, diagnostic approaches, and preventive strategies related to deep vein thrombosis and pulmonary embolism as shared postoperative concerns in prolonged immobilized patients. It seeks to emphasize the interconnected nature of these conditions, underline their significance in surgical and immobilized populations, and highlight the urgent need for standardized prophylactic protocols and increased clinical vigilance, particularly in the Indian healthcare context. By synthesizing available evidence, this paper aims to support improved awareness, prevention, and management strategies to reduce morbidity and mortality associated with postoperative venous thromboembolism.

OVERVIEW OF VENOUS THROMBOEMBOLISM (VTE)

Venous thromboembolism (VTE) is a collective term used to describe two closely related clinical entities—deep vein thrombosis (DVT) and pulmonary embolism (PE). These conditions represent a continuum of the same pathological process involving abnormal coagulation and obstruction of venous blood flow. VTE is recognized as one of the leading causes of preventable morbidity and mortality in hospitalized and postoperative patients worldwide [16]. Although both conditions are manifestations of the same disease spectrum, DVT refers to the formation of a blood clot within the deep veins, typically in the lower limbs, while PE occurs when part of this thrombus dislodges and travels to the pulmonary arteries, obstructing blood flow and gas exchange.

The clinical significance of VTE lies in its silent progression and potentially fatal consequences. Many cases of DVT remain asymptomatic until they evolve into pulmonary embolism, which can cause sudden collapse or death if untreated [17]. Epidemiological studies suggest that VTE is the third most common cardiovascular disorder after myocardial infarction and stroke. It contributes substantially to global morbidity, prolonged hospital stay, and healthcare costs. The incidence of DVT among surgical patients is particularly high, ranging between 10% and 40% depending on the type of surgery, duration of anesthesia, and postoperative immobility [18]. In certain high-risk groups such as orthopedic, oncologic, or neurosurgical patients,

the incidence may rise to 60% in the absence of adequate prophylaxis.

Venous thromboembolism is a major cause of preventable hospital death. It often develops in postoperative or immobilized patients due to the interplay of multiple physiological disturbances. Despite being largely preventable through early mobilization and appropriate prophylaxis, the disease continues to claim thousands of lives each year due to late diagnosis and inadequate preventive measures. Mortality associated with untreated PE can reach up to 30%, but with timely anticoagulation, it drops below 10% [19]. Chronic complications such as post-thrombotic syndrome and chronic thromboembolic pulmonary hypertension further highlight the long-term burden of the disease.

The burden of VTE is not confined to high-income countries. In low- and middle-income settings, including parts of Asia and Africa, underdiagnosis and lack of awareness have led to underestimation of true incidence. Hospital audits have shown that a significant proportion of postoperative deaths labeled as cardiac or respiratory failure may, in fact, be undetected pulmonary embolism. This reinforces the need for vigilant postoperative monitoring and prophylactic measures to reduce preventable mortality [20].

DVT and PE are not distinct conditions but represent different stages of the same disease continuum. The thrombus typically originates in the deep veins of the lower limbs, particularly in the calf or thigh. Prolonged stasis, vascular injury, or hypercoagulability contribute to clot formation. When a portion of the thrombus breaks away, it travels through the venous system into the right heart chambers and then into the pulmonary arteries, causing obstruction. The severity of PE depends on the size and number of emboli and the underlying cardiopulmonary reserve of the patient [21]. Once pulmonary arteries are occluded, there is a sudden increase in pulmonary vascular resistance, which may lead to right ventricular failure, hypoxemia, and, in severe cases, cardiac arrest. Smaller emboli may cause localized infarction, pleuritic pain, and hemoptysis. Thus, the prevention of DVT automatically reduces the risk of PE, underscoring the concept of VTE as a shared pathophysiological entity rather than two separate conditions.

VTE frequently arises in patients exposed to certain predisposing conditions. Major surgery is one of the strongest risk factors, as it induces all three elements of Virchow's triad—venous stasis, endothelial injury, and hypercoagulability. Long operative times, tissue trauma, and anesthesia contribute to reduced venous return, while the postoperative inflammatory response increases the levels of procoagulant proteins. In addition, immobilization during recovery further worsens venous stasis. Certain surgeries carry particularly high risk. Orthopedic procedures such as hip and knee arthroplasties, pelvic surgeries, and neurosurgical operations are well-documented examples. Patients undergoing cancer surgery are also vulnerable due to malignancy-induced hypercoagulability. Even minor procedures can provoke thrombus formation if prolonged immobilization follows. Prolonged bed rest, mechanical ventilation, plaster immobilization, or limb fractures can all lead to venous stasis and thrombosis [22]. Postoperative immobility plays a central role in the pathogenesis of DVT and PE. When skeletal muscles remain inactive, the calf muscle pump mechanism that normally assists venous return is compromised. Venous blood becomes static, allowing platelets and fibrin to accumulate along the vessel wall. The longer a patient remains immobile, the greater the risk of thrombus formation. In high-risk postoperative cases, even short periods of immobility may initiate clot development, particularly when other risk factors—such as dehydration or infection—are present [23].

Pathophysiology and Mechanistic Insights

Venous thromboembolism (VTE) is a complex pathophysiological process that encompasses both deep vein thrombosis (DVT) and pulmonary embolism (PE). It develops through an intricate interplay between vascular injury, blood flow disturbances, and changes in the coagulation system. The classic explanation for this process is provided by Virchow's Triad, which describes three major predisposing factors: venous stasis, endothelial injury, and hypercoagulability [24]. Each of these components contributes to the initiation and propagation of venous thrombi, particularly in patients undergoing surgery or experiencing prolonged postoperative immobilization. Understanding the mechanistic basis of thrombosis is crucial for identifying vulnerable individuals, guiding preventive strategies, and improving therapeutic interventions. Venous stasis is considered the most significant and modifiable factor in postoperative thrombus formation. Under normal physiological conditions, contraction of the calf muscles promotes venous return to the heart through the deep venous system. However, surgical procedures requiring anesthesia and postoperative immobilization interrupt this muscular activity, leading to stagnation of blood flow in the lower limbs. Reduced venous shear stress encourages the accumulation of platelets and red blood cells along the vessel wall, creating a favorable environment for clot formation [25].

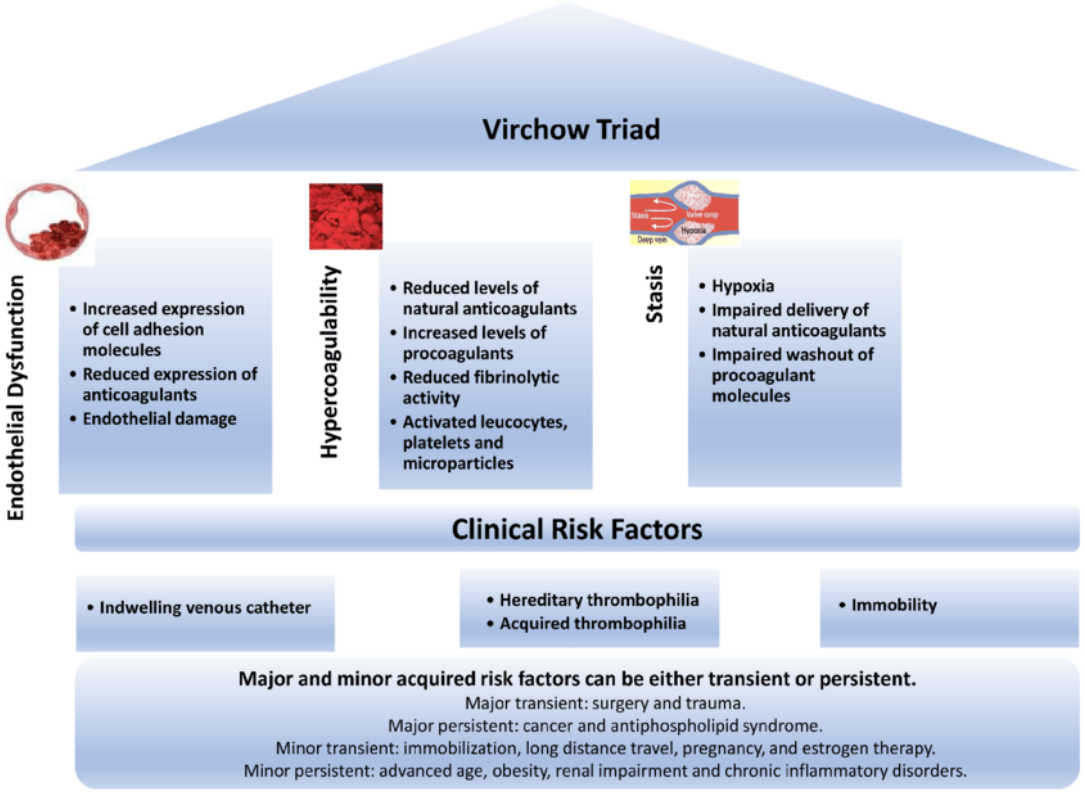

Figure 1: Pathogenesis of venous thromboembolism and risk factors [25]

In immobilized patients, especially after orthopedic, abdominal, or neurosurgical procedures, blood pooling occurs in the deep veins of the calf and thigh. Prolonged periods of bed rest or limb immobilization in casts exacerbate this effect. The stasis allows activated clotting factors to remain in contact with the endothelium longer than normal, increasing the likelihood of thrombin generation and fibrin deposition. Postoperative hypovolemia and dehydration, common after major surgery, further increase blood viscosity, thereby intensifying stasis-induced thrombogenesis. Even short periods of immobility, such as during long surgical procedures or extended anesthesia, can initiate microthrombus formation that may later propagate [26]. Endothelial injury constitutes the second major element of Virchow's triad. The vascular endothelium normally maintains an antithrombotic surface by releasing nitric oxide, prostacyclin, and tissue plasminogen activator. However, during surgery, direct mechanical trauma, tissue handling, and the use of surgical instruments can disrupt this delicate balance, exposing subendothelial collagen and tissue factor to circulating blood. These events trigger the adhesion of platelets to the injured endothelium and activate the coagulation cascade [27]. Endothelial cells subjected to oxidative stress or inflammatory mediators released during surgical trauma express adhesion molecules that recruit leukocytes and platelets. This inflammatory activation not only promotes thrombosis but also perpetuates vascular dysfunction. Central venous catheters, prosthetic implants, and orthopedic hardware may also contribute to localized endothelial irritation, enhancing the risk of thrombus formation at specific sites. In major surgeries, systemic inflammatory responses and cytokine release further augment the prothrombotic environment by increasing fibrinogen levels and impairing natural anticoagulant pathways such as antithrombin III and protein C. The third component of Virchow's triad, hypercoagulability, represents a state in which the blood's ability to clot is abnormally heightened. This may be due to inherited factors (e.g., deficiencies in protein C, protein S, or antithrombin) or acquired conditions such as malignancy, sepsis, pregnancy, and surgery. In the postoperative setting, surgical stress triggers a cascade of hormonal and inflammatory changes that collectively favor coagulation [28].

Tissue injury during surgery releases large amounts of tissue factor, a potent initiator of the extrinsic coagulation pathway. Simultaneously, levels of procoagulant proteins such as fibrinogen, factor VIII, and von Willebrand factor rise sharply, while fibrinolytic activity declines. This imbalance results in increased thrombin generation and impaired clot resolution. Furthermore, anesthesia and the use of certain medications such as estrogen-based drugs, corticosteroids, and antifibrinolytic agents can enhance coagulability. In cancer patients, tumor cells release procoagulant substances and inflammatory cytokines that directly activate the coagulation cascade. This explains why malignancy-related surgeries carry a higher risk of VTE compared to benign operations. Postoperative infection, dehydration, and reduced mobility further amplify hypercoagulability, creating a systemic prothrombotic environment that may persist for weeks after surgery. Once the triad conditions are present, the process of thrombus formation begins. Initial platelet adhesion to the damaged endothelium is followed by activation and aggregation, leading to the release of adenosine diphosphate (ADP), thromboxane A_2, and other prothrombotic mediators. The coagulation cascade is subsequently activated, generating thrombin, which converts fibrinogen into fibrin, stabilizing the clot. The resulting fibrin mesh traps red blood cells and additional platelets, forming a thrombus that can grow both proximally and distally within the vein. In DVT, thrombi often originate in the valve cusps of the deep veins, where blood flow is naturally slower. As the thrombus enlarges, it may partially or completely occlude the vein, impairing venous return and causing local symptoms such as swelling and pain [29]. If a portion of the thrombus detaches, it becomes an embolus that can travel through the venous system to the right atrium,

then to the right ventricle, and finally lodge in the pulmonary arteries, resulting in PE. The risk of embolization depends on thrombus size, adherence to the vessel wall, and blood flow dynamics. Large emboli can cause sudden hemodynamic collapse, while smaller ones may result in segmental or subsegmental infarctions. Pulmonary embolism represents the most severe consequence of DVT. Once an embolus reaches the pulmonary arteries, it obstructs blood flow to portions of the lung, leading to ventilation-perfusion mismatch, hypoxemia, and increased pulmonary vascular resistance [30]. The right ventricle is forced to work harder to overcome this resistance, and in massive embolism, acute right heart failure may ensue. In addition to mechanical obstruction, the release of vasoactive substances such as serotonin and thromboxane further exacerbates pulmonary vasoconstriction. Smaller emboli may cause pulmonary infarction, pleuritic chest pain, and hemoptysis. Chronic or recurrent emboli can lead to chronic thromboembolic pulmonary hypertension (CTEPH), a condition that severely impairs long-term cardiopulmonary function. The severity of PE depends on the extent of vascular obstruction and the patient's underlying cardiopulmonary reserve. Rapid diagnosis and intervention are therefore critical to prevent irreversible complications.

Epidemiology and Global Burden

Venous thromboembolism (VTE), encompassing deep vein thrombosis (DVT) and pulmonary embolism (PE), has emerged as a major global health concern due to its significant contribution to postoperative morbidity and mortality. Despite being a preventable condition, it continues to affect millions of individuals each year and remains one of the leading causes of in-hospital deaths worldwide [31]. The epidemiology of VTE is influenced by a complex interplay of demographic, clinical, and environmental factors, including age, type of surgery, duration of immobility, comorbid conditions, and the use of prophylactic measures. Although advancements in medical and surgical care have improved outcomes, the burden of VTE persists globally, particularly in regions with limited access to diagnostic tools and preventive care.

The global incidence of VTE varies widely across populations and healthcare settings. Estimates suggest that the annual incidence of DVT ranges between one and two cases per 1,000 adults, while PE occurs in approximately 60 to 70 per 100,000 individuals per year [32]. However, these figures are likely underestimates due to the large proportion of asymptomatic or undiagnosed cases. In hospitalized and postoperative patients, the risk of VTE is considerably higher, with incidence rates ranging from 10% to 40% depending on the type of surgery, patient profile, and preventive strategies used. In orthopedic procedures such as total hip or knee replacement, where prolonged immobility and tissue trauma are common, the incidence may exceed 50% in the absence of prophylaxis. Neurosurgical, oncologic, and major abdominal surgeries are also associated with elevated risks, primarily due to extended operative times and postoperative immobilization [33].

Epidemiological studies consistently show that VTE incidence increases with age, peaking in individuals over 60 years. This age-related rise can be attributed to declining mobility, endothelial dysfunction, and the accumulation of comorbidities such as obesity, malignancy, and cardiovascular disease. Women are at slightly higher risk during pregnancy and the postpartum period due to hormonal changes that promote a hypercoagulable state. However, men tend to have higher recurrence rates following a first VTE episode, indicating potential differences in vascular biology or hormonal influence [34]. Moreover, ethnic and geographic variations are evident. While Western populations report relatively high rates of VTE, studies in Asian populations, including India, have reported lower apparent incidences. This difference may reflect underdiagnosis rather than true epidemiological disparity, as awareness and screening remain limited in many parts of Asia.

In postoperative patients, the burden of VTE is closely linked to the nature and duration of surgery. Procedures involving the lower limbs, pelvis, or abdomen are particularly high-risk because they interfere with venous return and may damage vascular structures. Major trauma, spinal cord injury, and long-term bed rest are also well-established contributors. The use of general anesthesia, blood transfusions, and postoperative dehydration further compound the risk. Patients in intensive care units or those requiring prolonged mechanical ventilation represent another vulnerable group. Without adequate prophylactic measures, the risk of DVT in such patients can be as high as 60%, and approximately one in five may experience a silent or symptomatic pulmonary embolism [35].

The mortality associated with VTE remains a global concern. Pulmonary embolism is recognized as the third most frequent cause of cardiovascular death, following myocardial infarction and stroke. Acute PE may lead to sudden circulatory collapse, while recurrent or submassive emboli can cause chronic pulmonary hypertension and long-term disability. Even when nonfatal, VTE contributes to substantial morbidity, often resulting in chronic complications such as post-thrombotic syndrome characterized by pain, swelling, and venous ulcers. These complications affect quality of life and increase healthcare costs due to prolonged treatment and rehabilitation needs. Globally, it is estimated that VTE contributes to more than half a million deaths annually, most of which occur in hospital or shortly after discharge [36]. Alarmingly, a large proportion of these deaths are preventable through appropriate prophylaxis, early mobilization, and timely diagnosis.

Regional differences in healthcare infrastructure significantly influence the detection and management of VTE. In high-income countries, the availability of advanced imaging techniques such as Doppler ultrasonography and computed tomography pulmonary angiography allows for early diagnosis and intervention. In contrast, low- and middle-income countries face substantial challenges due to limited access to diagnostic resources and trained personnel. As a result, many cases remain undiagnosed or are detected only when complications arise. Furthermore, lack of awareness among healthcare professionals and patients contributes to inadequate use of prophylactic measures. In several developing regions, including parts of South Asia, prophylactic anticoagulation is often reserved for high-risk surgeries, leaving moderate-risk patients unprotected. This selective approach may explain the continued occurrence of preventable postoperative VTE cases despite existing medical knowledge [37].

Risk Factors Associated with Prolonged Immobilization

Prolonged immobilization is one of the most significant and well-documented risk factors contributing to the development of venous thromboembolism (VTE), encompassing both deep vein thrombosis (DVT) and pulmonary embolism (PE). In postoperative patients, immobility plays a central role in disturbing normal venous hemodynamics, triggering clot formation, and predisposing individuals to potentially fatal complications. The postoperative state creates a unique physiological environment characterized by venous stasis, endothelial dysfunction, and a hypercoagulable state—all of which synergize under the conditions of immobility to promote thrombogenesis [38]. The risk increases with the duration and severity of immobility, as well as with the presence of additional comorbid or surgical factors. Understanding these risk determinants is vital for effective prevention, early detection, and management of VTE in immobilized surgical and medical patients.

In the normal physiological state, contraction of the leg muscles facilitates venous return to the heart through the calf muscle pump mechanism. When a patient becomes immobilized, this muscle pump activity is significantly reduced or absent, leading to venous stasis, which is the first and most critical step in thrombogenesis. Immobilization following surgery, trauma, or medical illness allows pooling of blood in the lower extremities, increasing the local concentration of clotting factors and promoting platelet aggregation [39]. Venous stasis also leads to localized hypoxia within the vessel wall, which in turn triggers endothelial activation and expression of procoagulant proteins. The longer the immobility persists, the more likely it is that small, asymptomatic clots will form and propagate into larger, clinically significant thrombi. Prolonged bed rest is a particularly important risk factor among postoperative and critically ill patients. After major surgeries, especially orthopedic, abdominal, pelvic, or neurological operations, patients often remain confined to bed for extended periods due to pain, surgical site protection, or general weakness. This lack of movement results in sluggish venous circulation, particularly in the deep veins of the lower limbs. Studies have demonstrated that even a few days of strict bed rest can significantly increase the likelihood of DVT formation [40]. In patients with fractures or those requiring limb immobilization through plaster casting, the risk is even higher due to both mechanical obstruction of venous return and reduced mobility. Immobilized limbs with restricted movement exhibit reduced venous flow velocity, which promotes clot formation along the vein walls.

Another major contributor to thrombosis in immobilized individuals is the duration of surgery and anesthesia. Longer operative times are directly correlated with a higher incidence of postoperative DVT because anesthesia induces systemic vasodilation and muscle relaxation, further reducing venous return. When this is combined with intraoperative blood loss, dehydration, and tissue manipulation, the risk of postoperative thrombosis increases markedly. Prolonged anesthesia not only leads to intraoperative stasis but also delays postoperative recovery and ambulation, extending the period of immobility [41]. This is particularly relevant in elderly patients or those with comorbidities such as diabetes, obesity, or heart failure, who often experience slower postoperative mobilization. Orthopedic surgery is a well-known example where immobility plays a decisive role in the development of thromboembolic complications. Procedures involving the hip, knee, or pelvis carry the highest risk due to both mechanical and physiological factors. Following total joint replacement or fracture fixation, patients are often required to restrict limb movement to facilitate healing, resulting in prolonged periods of inactivity. Venous stasis combined with local tissue trauma creates an ideal environment for thrombus formation. Moreover, orthopedic patients frequently exhibit increased blood viscosity and elevated fibrinogen levels postoperatively, further amplifying the risk. Similarly, patients undergoing spinal surgeries or those with spinal cord injuries experience paralysis of lower limb muscles, which eliminates the muscle pump mechanism entirely and significantly raises the likelihood of thrombosis [42].

Apart from surgical causes, medical conditions leading to immobilization are also major risk factors for VTE. Patients suffering from acute stroke, paralysis, severe infections, or advanced malignancies often remain bedridden for extended periods. In stroke patients, hemiplegia results in decreased mobility on one side of the body, predisposing the affected limb to venous stasis. In cancer patients, immobility is compounded by malignancy-related hypercoagulability, creating a dual mechanism for thrombus formation. Furthermore, prolonged mechanical ventilation in intensive care units (ICUs) contributes to immobility-related VTE, especially when sedation and neuromuscular blockade are used. ICU patients often present multiple concurrent risk factors—immobility, dehydration, infection, and central venous catheterization—that collectively create a high-risk environment for thrombosis [43]. The risk factors associated with prolonged immobilization are multifactorial and interrelated, involving a combination of patient-related, surgical, and situational variables [44]. The absence of physical activity disrupts venous circulation, endothelial function, and coagulation balance, promoting clot formation. Factors such as surgery duration, obesity, age, dehydration, comorbid illness, and hormonal influences further amplify this risk. Prolonged bed rest, limb casting, mechanical ventilation, or paralysis are among the most significant contributors. Recognition of these risk factors allows healthcare professionals to adopt individualized prophylactic strategies, including early mobilization, mechanical compression, hydration management, and pharmacological anticoagulation.

Clinical Manifestations and Diagnostic Approaches

The clinical manifestations of venous thromboembolism (VTE), which includes deep vein thrombosis (DVT) and pulmonary embolism (PE), are often subtle, nonspecific, and variable, making diagnosis challenging in postoperative and immobilized patients. The recognition of symptoms, combined with the use of appropriate diagnostic tools, is essential for early detection and effective management. In many cases, especially in surgical patients, VTE may develop silently, and the first clinical indication may be a life-threatening pulmonary embolism [45]. The clinical presentation largely depends on the site, size, and extent of the thrombus, as well as the patient's physiological response and underlying comorbidities. In deep vein thrombosis, the most common manifestations involve the lower extremities. Patients typically present with unilateral leg swelling, pain, tenderness, warmth, and erythema, usually affecting the calf or thigh region. The discomfort is often exacerbated by standing or walking, and pitting edema may be present due to impaired venous drainage. However, these findings are not specific to DVT, as similar symptoms may occur in cellulitis, muscle injury, or postoperative inflammation [46]. In some cases, DVT may remain

asymptomatic until complications arise. This silent nature of the disease contributes to underdiagnosis and underscores the importance of routine clinical vigilance, especially in postoperative patients who remain immobilized for extended periods.

In cases where the thrombus propagates or dislodges, it can travel to the pulmonary circulation and result in a pulmonary embolism, which is the most severe manifestation of VTE. The classic symptoms of PE include sudden onset of shortness of breath, pleuritic chest pain, cough, hemoptysis, tachycardia, and hypoxia [47]. In massive PE, patients may experience syncope, hypotension, cyanosis, or sudden cardiac arrest due to right ventricular failure. Smaller emboli may cause milder symptoms such as low-grade fever, pleuritic pain, or transient dyspnea. Unfortunately, these symptoms are often nonspecific and may mimic other postoperative complications such as pneumonia, myocardial infarction, or heart failure, leading to diagnostic delays and increased mortality [48]. Accurate diagnosis of VTE requires a combination of clinical assessment, laboratory testing, and imaging studies. The initial step involves evaluating clinical probability using standardized scoring systems such as the Wells score or Geneva score, which stratify patients into low, moderate, or high-risk categories based on symptoms, risk factors, and clinical findings [49]. These scoring systems guide further diagnostic testing and help avoid unnecessary imaging in low-risk patients. In patients with suspected DVT, duplex ultrasonography is the preferred noninvasive diagnostic tool. It allows real-time visualization of venous flow and thrombus formation with high sensitivity and specificity. Compression ultrasonography remains the first-line investigation, as failure of the vein to collapse under probe pressure indicates thrombus presence [50].

When ultrasonography results are inconclusive or proximal extension of the thrombus is suspected, venography or magnetic resonance venography may be used for confirmation. Laboratory tests such as D-dimer assays are valuable in excluding VTE in low-risk patients, as a normal D-dimer level effectively rules out active thrombosis. However, in postoperative patients, D-dimer levels are often elevated due to surgical trauma and tissue inflammation, limiting the specificity of this test [51]. For suspected pulmonary embolism, computed tomography pulmonary angiography (CTPA) is the gold standard for diagnosis. It provides detailed visualization of emboli within the pulmonary arteries and can assess the extent of obstruction. In patients who cannot undergo CTPA due to renal dysfunction or contrast allergy, ventilation-perfusion (V/Q) scans offer an alternative imaging modality. Adjunctive diagnostic methods such as echocardiography may reveal right ventricular strain or dilation in cases of massive PE, supporting the diagnosis in hemodynamically unstable patients. Electrocardiography and arterial blood gas analysis may provide indirect evidence, such as tachycardia, right axis deviation, or hypoxemia, but they lack specificity. Postoperative monitoring protocols often include regular limb examinations and early investigation of unexplained hypoxia, tachycardia, or chest discomfort to ensure timely detection of VTE [52].

Preventive and Prophylactic Strategies

The prevention of venous thromboembolism (VTE), including deep vein thrombosis (DVT) and pulmonary embolism (PE), is a critical aspect of postoperative care and management in immobilized patients. Given that VTE is largely preventable, the adoption of evidence-based prophylactic strategies can significantly reduce morbidity and mortality rates. Prevention focuses on identifying high-risk patients, minimizing predisposing factors, and implementing both mechanical and pharmacological interventions tailored to individual needs. The choice of prophylactic method depends on patient characteristics, type of surgery, duration of immobilization, and bleeding risk [53]. Early risk assessment remains the cornerstone of effective VTE prevention. All surgical and immobilized patients should undergo a standardized evaluation using validated scoring systems such as the Caprini or Padua risk assessment models, which categorize individuals into low, moderate, high, or very high-risk groups. These models consider factors like age, obesity, malignancy, prior history of VTE, prolonged immobility, and type of surgery. Based on the risk category, clinicians can implement appropriate preventive measures, ensuring that both overuse and underuse of prophylaxis are avoided. Regular reassessment during hospitalization is essential, as a patient's risk profile may change postoperatively due to complications, infection, or extended immobility [54].

Mechanical prophylaxis methods are particularly valuable in patients with contraindications to anticoagulant therapy, such as those with a high risk of bleeding. These methods work by enhancing venous return, reducing stasis, and promoting endothelial function. The most commonly used devices include graduated compression stockings (GCS) and intermittent pneumatic compression (IPC) devices. GCS apply graded pressure, highest at the ankle and decreasing proximally, facilitating upward venous flow. IPC devices cyclically inflate and deflate around the legs, mimicking natural muscle contractions to prevent venous stasis. When used correctly, mechanical methods are effective, especially when combined with early mobilization. However, their efficacy is dependent on proper sizing, adherence, and continuous use. Patients with peripheral arterial disease or skin ulcers require special consideration before applying compression devices to prevent complications [55]. Pharmacological prophylaxis remains the mainstay for moderate- to high-risk patients, particularly following major surgical procedures or prolonged immobilization. The most widely used agents include low molecular weight heparin (LMWH), unfractionated heparin (UFH), and direct oral anticoagulants (DOACs). LMWH is preferred in most cases due to its predictable pharmacokinetics, once-daily dosing, and lower risk of heparin-induced thrombocytopenia. It acts by inhibiting factor Xa and thrombin, thereby preventing clot propagation. UFH is typically reserved for patients with renal impairment, as it has a shorter half-life and can be easily reversed if bleeding occurs. DOACs such as rivaroxaban and apixaban have gained increasing acceptance for postoperative thromboprophylaxis, especially in orthopedic surgeries, due to their oral administration and fixed dosing. However, their use must be guided by renal and hepatic function assessments to ensure safety [56].

The timing and duration of pharmacologic prophylaxis are crucial for optimal effectiveness. In most surgical patients, anticoagulant prophylaxis should begin 6 to 12 hours postoperatively once hemostasis is secured and continue for at least 7 to 10 days. In high-risk cases, such as orthopedic or oncologic surgeries, extended prophylaxis for up to 4 to 6 weeks may be necessary, as the risk of thrombosis persists well beyond discharge. Combination therapy, utilizing both pharmacological and mechanical methods, is recommended in very high-risk individuals to achieve synergistic protection against VTE [57]. Early mobilization is

a simple yet highly effective preventive measure. Encouraging patients to ambulate as soon as medically feasible restores the calf muscle pump function, promotes venous return, and reduces stasis. Even passive leg exercises, ankle dorsiflexion, and range-of-motion activities can significantly decrease thrombotic risk in patients unable to ambulate independently. Nurses and physiotherapists play an essential role in implementing mobilization protocols and ensuring adherence. Maintaining adequate hydration and avoiding unnecessary fluid restrictions also support hemodynamic stability and reduce blood viscosity, further minimizing the risk of thrombosis [58]. For patients at particularly high risk or with contraindications to anticoagulants, inferior vena cava (IVC) filters may be considered as a temporary mechanical barrier to prevent embolization of thrombi from the lower limbs to the lungs. However, their use should be restricted to specific cases, as they do not prevent clot formation and may increase the risk of long-term venous stasis if left in place. Regular follow-up and removal once the acute risk period has passed are essential to prevent complications associated with prolonged filter placement [59].

Management and Therapeutic Interventions

The management and therapeutic interventions for venous thromboembolism (VTE), including deep vein thrombosis (DVT) and pulmonary embolism (PE), are centered on preventing clot extension, reducing the risk of recurrence, alleviating symptoms, and minimizing long-term complications. Early diagnosis and prompt initiation of treatment are critical, as delays can lead to severe consequences such as hemodynamic instability, pulmonary hypertension, or death. The therapeutic approach is determined by the clinical severity, patient comorbidities, contraindications to anticoagulation, and the presence of risk factors such as immobilization or recent surgery [60]. The mainstay of treatment for VTE is anticoagulation therapy, which prevents further thrombus formation and facilitates the body's natural fibrinolytic mechanisms to dissolve existing clots. The first-line agents include unfractionated heparin (UFH), low molecular weight heparin (LMWH), and direct oral anticoagulants (DOACs). UFH acts rapidly by inhibiting thrombin and factor Xa and is commonly used in hospital settings for initial therapy, especially in patients with renal impairment or those at high risk of bleeding. It is administered intravenously and requires close monitoring of the activated partial thromboplastin time (aPTT) to maintain therapeutic levels. LMWH is preferred for its more predictable pharmacokinetics, longer half-life, and subcutaneous administration, eliminating the need for frequent laboratory monitoring. It is widely used in both inpatient and outpatient settings and has been shown to reduce recurrence and mortality in acute VTE [61].

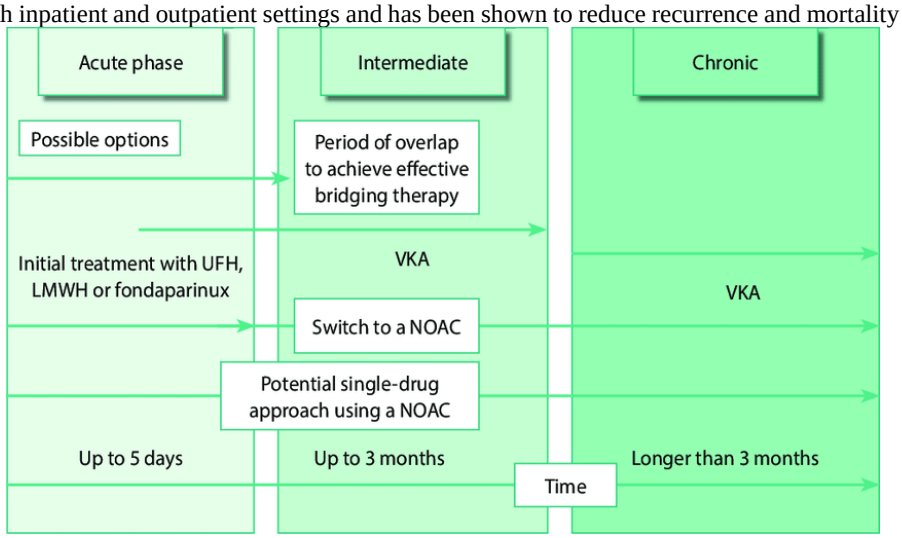

Figure 2: Treatment phases in the management of venous thromboembolism [61]

DOACs, such as rivaroxaban, apixaban, edoxaban, and dabigatran, are increasingly used for both the initial and long-term treatment of VTE. These agents selectively inhibit factor Xa or thrombin, providing consistent anticoagulation without the need for regular monitoring. Their oral administration, rapid onset of action, and fixed dosing make them particularly convenient for patients transitioning from hospital to home care. However, they must be used cautiously in patients with renal or hepatic impairment, as accumulation can increase bleeding risk. For individuals unable to tolerate DOACs, vitamin K antagonists (VKAs), such as warfarin, remain an effective alternative. Warfarin therapy requires close monitoring through the international normalized ratio (INR), aiming for a therapeutic range between 2.0 and 3.0. Since VKAs have a delayed onset, they are often initiated concurrently with heparin until therapeutic INR levels are achieved [62]. The duration of anticoagulant therapy depends on the underlying cause and risk profile of the patient. In cases of VTE provoked by a transient risk factor such as surgery or immobilization, treatment is typically continued for a minimum of three months. For unprovoked VTE or patients with persistent risk factors, extended or indefinite therapy may be recommended to prevent recurrence. Regular follow-up is essential to reassess bleeding risk and adjust therapy accordingly. For patients at high risk of recurrence or with recurrent VTE despite adequate anticoagulation, long-term prophylaxis may be necessary, using lower maintenance doses of anticoagulants.

In patients with massive or life-threatening pulmonary embolism, where there is evidence of hemodynamic instability or right ventricular failure, thrombolytic therapy may be indicated. Thrombolytic agents such as alteplase, streptokinase, or urokinase dissolve the thrombus by activating the fibrinolytic system. The primary goal of thrombolysis is to rapidly restore pulmonary perfusion, reduce right ventricular strain, and prevent cardiogenic shock. However, thrombolysis carries a significant risk of bleeding, particularly intracranial hemorrhage, and is therefore reserved for carefully selected patients. For those in whom systemic thrombolysis is contraindicated, catheter-directed thrombolysis (CDT) or mechanical thrombectomy may be considered, offering localized clot dissolution with reduced systemic exposure to thrombolytic drugs [63]. Inferior vena cava (IVC) filters are

mechanical devices placed in the inferior vena cava to prevent emboli from reaching the lungs. These are typically used when anticoagulation therapy is contraindicated, such as in patients with active bleeding, recent surgery with high hemorrhagic risk, or allergy to anticoagulants. Temporary or retrievable filters are preferred to minimize long-term complications such as filter thrombosis or migration. Once the contraindication to anticoagulation resolves, filters should be removed promptly to prevent chronic venous obstruction. However, IVC filters are not substitutes for anticoagulants and should only be considered as adjunctive therapy or for short-term use in specific high-risk cases [64].

Table 1: Research Gaps, Challenges, and Future Directions in Venous Thromboembolism (VTE) Management

S. No.	Identified Research Gap / Challenge	Description / Future Direction	Ref.
1	Underdiagnosis and Underreporting	Many VTE cases, especially in postoperative and immobilized patients, remain undetected due to nonspecific symptoms and limited diagnostic access. Need for large-scale population-based studies to assess true incidence and outcomes.	[65]
2	Lack of Standardized National Guidelines	Absence of uniform national protocols and inconsistent adherence to prophylaxis practices across institutions. Emphasis on developing region-specific, cost-effective preventive strategies suited to local healthcare systems.	[66]
3	Limited Diagnostic Infrastructure	Deficiency of Doppler ultrasound and advanced imaging facilities in smaller and rural hospitals delays timely diagnosis. Expansion of portable and point-of-care diagnostic tools is required to improve detection rates.	[67]
4	Long-Term Safety of New Anticoagulants	Limited evidence on the safety, efficacy, and cost-effectiveness of direct oral anticoagulants (DOACs) in elderly, oncologic, or renally impaired patients. Further multicentric trials are needed to establish optimal use.	[68]
5	Personalized Medicine Approaches	Integration of genetic, biochemical, and clinical risk factors for individualized thrombosis prediction. AI-based predictive models could enhance perioperative risk assessment and treatment decisions.	[69]
6	Lack of Multidisciplinary Collaboration	Insufficient collaboration among physicians, surgeons, nurses, and physiotherapists in implementing VTE prevention protocols. Continuous medical education and team-based strategies are recommended.	[70]

CONCLUSION

Venous thromboembolism remains one of the most significant and preventable postoperative complications, particularly in immobilized patients recovering from major surgery. Despite the availability of effective preventive and therapeutic strategies, many cases continue to go undiagnosed or inadequately managed due to limited awareness, lack of standardized protocols, and resource constraints. The interplay of immobility, endothelial injury, and hypercoagulability underscores the complexity of VTE pathophysiology, demanding comprehensive perioperative care. Early mobilization, accurate risk assessment, and consistent use of pharmacological and mechanical prophylaxis are critical in preventing thrombotic events. Strengthening multidisciplinary collaboration and promoting continuous medical education are vital to ensure adherence to evidence-based guidelines. Future directions must focus on developing cost-effective diagnostic tools, refining personalized prophylactic regimens, and leveraging emerging technologies such as artificial intelligence for early risk prediction. By bridging research gaps and improving clinical implementation, healthcare systems can significantly reduce VTE-related morbidity and mortality, thereby enhancing the quality and safety of postoperative recovery.

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