REVIEW ARTICLE

Deep Vein Thrombosis in Special Populations: Pregnancy, COVID-19, and the Elderly

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Abstract

The purpose of this review is to examine the specific epidemiology, risk factors, pathophysiological mechanisms, clinical complexities, and management of deep vein thrombosis (DVT) in three high-risk populations – pregnant women, patients diagnosed with COVID-19, and older adults. A systematic literature review of peer-reviewed meta-analyses, cohort studies, and expert guidelines examining the incidence, diagnosis, prophylaxis, and treatment of DVT in different populations was conducted. A systematic search of the literature was conducted on PubMed, Scopus, and the Cochrane Library based on inclusive keywords. The review incorporates both high-quality meta-analyses and large cohort studies on DVT to contrast incidence, complications, and management strategies across settings and population groups. It is notable that considerable variation in the incidence of DVT occurred between population groups, particularly elevated incidence in intensive care unit COVID-19 patients and pregnant women in the peri-partum period. The elderly population presents unique barriers to diagnosis and treatment due to polypharmacy, complications, and multimorbidity. Existing cumulative data support population-based risk stratification measures and thromboprophylaxis strategies. In this review, the importance of integrated pathways, early risk assessments, and streamlined therapeutic choices are highlighted, as these can help to reduce the burdens of morbidity and mortality attributed to DVT in these vulnerable populations.

Key words: Anticoagulation, COVID-19, deep vein thrombosis, elderly, intensive care unit, pregnancy, risk stratification, special populations, thromboprophylaxis, venous thromboembolism

INTRODUCTION

Overview of deep vein thrombosis (DVT)

DVT is a vascular disorder characterized by the abnormal formation of blood clots (thrombi) in the deep veins, usually in the lower extremities.[1,2] DVT occurs because of the three components of Virchow's triad: venous stasis, hypercoagulability, and injury to the epithelial lining of the blood vessels.[1,2] Venous stasis is reduced blood flow through the veins and usually occurs when patients are immobile or have venous insufficiency. A hypercoagulable state is characterized by an increased tendency for blood to coagulate, either due to inherited factors (e.g., Factor V Leiden mutation), diseases (e.g., cancer), pregnancy, or increased risk associated with the use of medications (e.g., oral contraceptives). Third, injury to the epithelial lining of blood vessels affects the functionality and structure of the vascular epithelium and helps in the adhesion of platelets, leading to thrombus formation.[1,2]

Clinically, DVT has significant acute and chronic long-term complications. The most immediate event is pulmonary embolism (PE), which is highly fatal because the original thrombus can embolize to the pulmonary arteries.[1,3] The most severe chronic sequelae of DVT are post-thrombotic syndrome (PTS) with pain, edema, and venous insufficiency.[1]

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DVT can also result in local symptoms such as limb pain, swelling, and functional mobility limitations, leading to a poor overall quality of life. The Fredericksburg clinic provides examples of high-risk disorders.

There are many risk factors associated with DVT, otherwise unexplained, related to DVT, including but not limited to prolonged immobilization (hospital/bed rest), duration of surgical recovery, malignancy, pregnancy, hereditary thrombophilias, and certain medications.[1] Each of these, through any part(s) of Virchow's triad, ultimately contributes to the development of thrombi and the risk of progression.

DVT in context: A comparison with other types of thrombosis

DVT is characterized by venous thrombus formation due to factors in Virchow's triad.[1] However, other forms of thrombosis, such as arterial thrombosis and cerebral venous sinus thrombosis (CVST), have varied pathophysiologies, risk factor profiles, diagnoses, and treatment complexities. For example, there are major differences in the occurrence of arterial thrombosis. Arterial thrombosis is typically caused by atherosclerosis, rupture of the endothelial plaque, and platelet aggregation due to high shear stress. Hence, venous clotting is fundamentally different from arterial clotting. CVST typically evolves due to infection, hormonal modification, and inflammatory or autoimmune processes.[4]

Risk factors for DVT, including immobility, surgery, cancer, pregnancy, and thrombophilia, are associated with a different thrombotic risk profile.[1] Conversely, identifiable risk factors for other types of thrombotic events have different etiologies. Examples of identifiable risk factors include hormonal therapy, local infection, autoimmune or inflammatory conditions, and mechanical head trauma (the last two are often involved in CVST and arterial thrombosis).[4]

With respect to diagnosis, DVT is diagnosed by ultrasound compression. In patients suspected of having DVT but with low pre-test probability, a negative D-dimer result is useful.[5,6] There is a standard of care for DVT: Anticoagulation, LMWH, unfractionated heparin, or direct oral anticoagulants (DOACs), followed by catheter-directed thrombolysis in the worst cases of DVT.[6] Other types of thrombotic events differ in their diagnostic and treatment strategies. For example, MR venography is used for the diagnosis of CVST, and CVST cases with hemorrhagic infarcts are treated with anticoagulation.[4]

DVT is associated with complications such as PE and PTS, which can result in long-term morbidity and mortality if left untreated.[1] In contrast, complications of arterial thrombosis include stroke and myocardial infarction, whereas CVST may lead to seizures, neurological deficits, and intracranial hypertension.[4]

DVT is a distinct clinical entity governed by Virchow's triad and is characterized by a well-established risk factor profile. It carries significant morbidity due to both acute (e.g., PE) and chronic (e.g., PTS) complications. Compared to other forms of thrombosis, DVT exhibits unique pathophysiological and clinical patterns. A clear understanding of these distinctions is essential for the accurate diagnosis, appropriate treatment, and prevention of complications in both venous and non-venous thrombotic disorders.[5,7]

Global incidence and burden of DVT in general and in special populations

DVT is the formation of blood clots in the deep veins, typically in the legs. It is a significant global health concern because it can lead to serious complications, such as PE, where the clots travel to the lungs, and can be fatal. It is important to understand the global incidence and burden of DVT in both the general and specific high-risk populations to formulate effective prevention and management plans.[8]

In general, venous thromboembolism (VTE), including DVT and PE, occurs in approximately 1–2 out of 1000 people each year globally. This translates to hundreds of thousands of cases annually in larger countries, such as the United States.[9] Population studies from Western Europe, North America, Australia, and parts of Latin America generally indicate an annual incidence of 0.75–2.69 cases/1000, with a dramatic increase in older adults (70+ years, with an incidence between 2 and 7/1000).[9-11] In some Asian populations, such as Chinese and Korean ethnicities, the incidence may appear lower; however, the absolute burden may be perceived as substantial because of demographic aging.[10,12] VTE appears to constitute a substantial global disease burden, representing a major contributor to the global disease burden and among the leading causes of lost disability-adjusted life years, especially among hospitalized individuals across low-, middle-, and high-income countries.[10,12]

Specific risks and burdens of DVT exist among special populations. For example, women who take combined oral contraceptives (COCs) with certain progestogens (i.e., cyproterone acetate, desogestrel, drospirenone, or gestodene) have a statistically modestly increased risk of VTE compared to women who take a COC with levonorgestrel.[13] This is an example of how contraceptive choice can influence the risk of thrombosis.

Natural disasters, especially earthquakes, have been linked to the incidence of DVT in survivors. The contributory factors of risk are immobilization, dehydration, stress, and secondary injuries, with a pooled DVT rate of approximately 9%, which is substantially higher than that in the general population.[14] Overall, this indicates an increased and important need for targeted screening and prophylaxis among disaster-impacted

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populations, where undetected asymptomatic DVT can progress to fatality.

In Africa, the epidemiology of VTE is not well defined but is potentially significant, particularly in surgical cases and during the pregnant and postpartum periods. The reported DVT prevalence in post-operative patients has ranged from 2.4% to 9.6% in recent literature, which was used to estimate that the incidence of DVT in pregnant/postpartum women could average between 380 and 448 cases/100,000 births. Medical patients receive PE through risk stratification and risk-reduction protocols, with mortality rates between 40% and 69.5% due to investigation and prophylaxis gaps preventing adequate healthcare access. Importantly, at least one-quarter of at-risk patients in Africa were not offered appropriate thromboprophylaxis.[15] This data point is indicative of the considerable unmet need in a resource-limited context.

The DVT burden has implications for healthcare systems, with significant costs associated with screening practices and treatment. For example, in endovenous ablation procedures for varicose veins, approximately ~0.7% of patients will have DVT post-procedure; however, for routine duplex ultrasound screening, the costs are very high relative to the number of DVT detected, which raises questions about cost-effectiveness and clinical value.[10]

Patients with chronic kidney disease (CKD) represent another special population with an elevated cardiovascular risk, including the risk of venous thrombosis. CKD is a global health issue affecting 15–20% of adults worldwide and is associated with multiple cardiovascular outcomes from overlapping risk factors and an increase in or emergence of inflammatory and coagulation-based pathophysiological mechanisms.[16] While the literature is poor at quantifying the degree of DVT in CKD populations, the increased DVT risk requires careful assessment and multidisciplinary management of patients with CKD.

Chinese epidemiological data showed an overall increase in hospitalization for VTE rates from 3.2 to 17.5/100,000 population between 2007 and 2016, whereas in-hospital mortality for VTE decreased from 4.7% to 2.1%. Older men specifically showed very high hospitalization rates, and consistent with other data, the northern regions reported the highest in-hospital mortality rates. While the overall rates are lower than average hospitalizations in Western countries, these data suggest that there is an increasing burden of VTE in this region and highlight the importance of regional viral epidemiology.[17]

The COVID-19 pandemic and vaccination programs have demonstrated the relative rarity of specific thrombotic events, such as portal vein thrombosis and other thromboses involving the liver, all of which occurred in only a small fraction of the vaccinations provided worldwide, which outweighed the risks of vaccination. DVT is a serious global health issue that occurs at different incidence rates attributable to age, ethnicity, region, and other risk factors, such as hormonal therapy, surgery, pregnancy, chronic illness, and immobilizing acute events such as natural disasters. The burden of disease is great, as it causes morbidity and mortality and costs healthcare dollars and resources that would be better directed elsewhere. Special populations require specific forms of risk assessment, prevention, and management to provide a robust response to reduce DVT and its adverse outcomes on a global scale.[11,14,18]

Objectives of the review

This review aims to:

- The epidemiology, pathophysiology, and clinical presentation of DVT in pregnancy, COVID-19, and the elderly were critically explored.
- Delineate population-specific risk factors and diagnostic approaches that account for physiological and pathological differences.
- Evaluate current treatment strategies and clinical guidelines, highlighting the adaptations necessary for each special population.
- The long-term outcomes and complications unique to these cohorts, including recurrence risk and PTS, should be discussed.
- Identify research gaps and propose areas for future investigation to improve DVT care in high-risk populations.

DVT IN PREGNANCY

Epidemiological data on DVT during pregnancy and postpartum

DVT refers to the development of blood clots within the deep veins. DVT usually occurs in the veins of the legs. Pregnant women and women after childbirth have a higher risk of detecting DVT than non-pregnant women due to changes in physiology that increase clotting, reduced mobility, and compression of the vascular system of the body by a growing uterus. Understanding the prevalence (occurrence at a point in time) and incidence (occurrence of new cases over time) of DVT is important for its prevention and management.[15]

Epidemiological studies have shown that pregnancy and the postpartum period are periods of increased VTE risk, which includes DVT and PE. The pooled incidence of pregnancy-associated VTE ranges from approximately 1.2–1.4/1000 pregnancies (DVT: Approximately 1.1/1000 pregnancies; PE: Approximately 0.3/1000 pregnancies).[19,20] Importantly, over half of these events occurred after childbirth, stressing the postpartum period as one of the critical times to monitor.[19]

Importantly, the risk is not equal during pregnancy or the postpartum period. The greatest incidence of postpartum VTE occurs during the first 3 weeks after childbirth, and then decreases at subsequent time points; however, the risk remains elevated until 12 weeks after childbirth.[21] Certain obstetric risk factors (e.g., cesarean delivery, preeclampsia, hemorrhage, and postpartum infection) also increase the risk of postpartum DVT and VTE.[21] For instance, the odds of receiving VTE prophylaxis are doubled for cesarean delivery because of the recognized risk factors for DVT and VTE.[22]

The incidence of DVT during pregnancy and postpartum differs depending on the geographical region and some characteristics of the studied populations. In African populations, the incidence of DVT in pregnant and postpartum women has been reported to be between 380–448/100,000 births/year, demonstrating a large burden in these settings, which can be hypothetically increased by the fact that prophylaxis is commonly limited.[15] Likewise, studies in Egyptian hospitals have shown that an overall VTE incidence of 0.55/1000 maternities is noted, with postpartum women accounting for an increased risk compared to pregnant women.[22]

Many underlying conditions and risk factors can significantly contribute to the risk of developing DVT during pregnancy. Thrombophilia, defined as inherited and acquired disorders that can increase an individual's tendency to clot, is an important factor noted in a significant number of women who develop pregnancy-associated thrombosis, particularly in Caucasian populations with genetically determined mutations, such as factor V Leiden and prothrombin gene G20210A.[23] Similarly, both obesity and heightened incidences of elevated BMI correlate with an increased risk of DVT in terms of incidence rates and determining whether prophylaxis should be delivered,[22,24] In terms of the metabolic events seen with preeclampsia, the hypertensive disorder of pregnancy has been associated with a VTE incidence higher than a 40–50% increased risk during pregnancy, the postpartum period, and in some cases, well into the period beyond puerperium, indicating a persisting prothrombotic state.[25]

Although cerebral venous thrombosis (CVT) is a rare form of venous thrombosis affecting the brain venous sinuses, it has also increased incidence rates in pregnancy and puerperium, especially in women with a history of CVT or thrombophilia. However, CVT has a much lower incidence than DVT and is treated differently in terms of clinical presentation and management.[26-28]

Even with the recognition of this risk, the management of VTE prophylaxis in pregnancy differs considerably worldwide. For example, in Africa, up to 25% of patients at risk may not receive prophylaxis, contributing to significant morbidity and mortality in both mothers and infants.[15] In contrast, developed countries may have better guidelines, but they too cannot portray VTE in the postpartum period as only one-third of patients (readmitted for postpartum VTE) will return to the same hospital; thus, some quality metrics may not identify the postpartum VTE event.[29] Epidemiological data suggest that VTE in the antepartum period (while pregnant) and postpartum (within approximately 6 weeks after delivery) occurs in mothers in approximately 1 and 1.4/1000 pregnancies, respectively, and therefore is clinically relevant, but does not occur frequently enough to evaluate severity and how the complications may contribute to maternal morbidity or mortality. The puerperium phase, including the postpartum period, represents the highest time frame of risk, approximately 3 weeks after delivery, due to increased risk factors such as cesarean delivery, preeclampsia, obesity, and thrombophilia. Regional differences in the incidence and use of prophylaxis indicate the need to improve awareness and protective strategies globally. There is a basic need for healthcare providers to understand these patterns of epidemiology to identify at-risk women and implement prophylaxis in a timely manner, to relieve the burden of pregnancy-associated DVT.[20]

Pathophysiological changes: Hormonal, hemodynamic, and coagulation-related

DVT refers to the presence of blood clots in the deeper veins, particularly in the legs. It is important to understand the hormonal, hemodynamic, and coagulation factors that lead to DVT to understand how it develops individually.

Hormonal factors

Hormones can significantly affect the development of DVT. A factor of interest is that hormone-containing therapies, such as COCs and hormone replacement therapy, increase the chance of clots by changing the balance of clotting factors in the blood. Hormonal treatments increase procoagulant factors while decreasing natural anticoagulant levels. This makes the patient hypercoagulable.

For example, estrogen increases the production of clotting proteins, such as fibrinogen and factors VII, VIII, and X, which promote coagulation and clotting. The effects of hormone use depend on the administration route (oral versus transdermal) and different hormone combinations. The use of testosterone therapy in men is also of interest. Initially, testosterone therapy was presumed to increase the risk of VTE. However, no definitive association has demonstrated that testosterone therapy actually increases the risk of DVT, as the most recent evidence has demonstrated that there is no statistically significant association with DVT.[30] Hormonal changes during pregnancy and the postpartum period have been shown to increase DVT, as this condition appears to affect reproductive physiology.[31] During pregnancy, these changes increase the factors that lead to DVT, and the pathophysiological changes in venous blood flow, and both anatomical and physiological changes lead to a higher risk of DVT.

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Hemodynamic changes

The dynamics of blood movement have a marked effect on the development of DVT. The condition known as venous stasis, that is, slowed blood flow, also occurs in the deep veins of the legs, where venous blood stagnates, and clotting can occur. The enlarging uterus of a pregnant woman compresses the pelvic veins and reduces venous return, resulting in venous stasis.[31] Immobilization, either through bed rest or hospitalization, slows venous blood flow with the same results, increasing the risk of developing DVT.[32] Obesity can alter hemodynamics by increasing intra-abdominal pressure, which can restrict venous blood return and promote stasis.[33] Hemodynamic factors combine with endothelial injury and hypercoagulability to meet the three factors identified by Virchow's classic description of thrombus formation.

Coagulation-related pathophysiology

Thrombosis, including DVT, arises from multiple coagulation pathways, immune responses, and endothelial components. Upon endothelial injury or insufficiency, inflammation activates various coagulation pathways that lead to the formation of a fibrin clot. Furthermore, the classic triad has been augmented due to recent understanding of immunothrombosis and the interactions of immune cells, such as neutrophils, that release extracellular traps that encourage clotting.[34] The relevance of this in disease states, such as COVID-19-induced hyper-inflammation and systemic microthrombosis, has been recognized. There is also an inherited or acquired predisposition to hypercoagulability and, therefore, DVT due to defects in (e.g., Factor V Leiden mutation, antiphospholipid syndrome).[34] Cancer and certain chemotherapy treatments further activate coagulation and impair natural anticoagulation, increasing the risk of VTE.[34] Obesity-associated inflammation and oxidative stress promote endothelial dysfunction and coagulation activation, with more prothrombotic factors involved.[33]

Clinical and epidemiological context

The risk of DVT recurrence depends on the cause of the bout of DVT. Women at risk for estrogen-related VTE have a low risk after completing anticoagulation therapy; therefore, many of these women can be safely treated in the short term.[35] Patients with cardiovascular risk or those who have had previous VTEs have a higher risk of recurrence and complications.[36] Prolonged anticoagulation reduces symptomatic VTE among high-risk hospitalized patients but simultaneously increases the bleeding risk, indicating a fine balance between preventing thrombosis and avoiding hemorrhage.[32]

Summary

Overall, hormonal aspects, such as estrogen levels and changes during pregnancy, lead to the stimulation of coagulation factors and inhibition of anticoagulants, which promotes clotting. Hemodynamically stagnant blood flow occurs during immobilization, pregnancy, and/or obesity, which leads to a greater chance of thrombosis. Coagulation abnormalities reflect perturbations in the complex components of coagulation cascades, alterations in immune responses, and the state of endothelial vessels, while the likelihood of which factors will provoke an individual's reactivity depends on genetic susceptibility and acquired vulnerability. Awareness of these interrelated pathways allows for the initiation of several effective management and preventative approaches for DVT.[37,38]

Diagnostic challenges (safety of imaging modalities; interpretation of D-dimer)

Diagnosis of DVT is complex. D-dimer assays are not sufficiently specific, and imaging has limitations. Compression ultrasonography remains the initial imaging modality because of its safety and accessibility; however, its sensitivity decreases in specific populations and sites. Computed tomography (CT) and magnetic resonance imaging (MRI) are the preferred imaging modalities because they provide better visualization, but they are associated with radiation exposure, contrast exposure, and logistical issues. The application of D-dimer levels must include clinical pre-test probability for VTE and age-adjusted levels to reduce false-positive results and unnecessary imaging. Even with improvements in DVT diagnosis, obtaining a definitive diagnosis in at-risk populations, such as the elderly, pregnant patients, or patients with COVID-19, continues to present limitations, and a safeguard for diagnosis must be decided based on the balancing act of safety, accuracy, and resources for the diagnostic algorithm.[39]

The literature review has shown that the algorithm for diagnosing DVT is dependent on the application of the integrated approach. This integrated approach assesses the clinical probability, D-dimer assay, and imaging assays. Le Gal and Righini (2015)[40] state that current diagnostic algorithms use a sequential and non-invasive approach, with the first step being clinical risk assessment, followed by D-dimer measurement in patients identified as low-to-moderate risk who can be excluded for VTE. Only patients with a positive D-dimer result or with a high clinical suspicion would then undergo imaging. This approach has been proven safe and cost-effective; however, the authors acknowledge the ongoing debates about overdiagnosis and inadequate management of some subgroups. D-dimer interpretation was scrutinized by Innocenti *et al.*,[41] who discussed the test's high sensitivity and poor specificity. Their narrative review covers a broad cohort from an emergency department setting and makes it clear that elevated D-dimer levels are not specific to thrombosis and can occur in numerous non-thrombotic conditions (i.e., infection, inflammation, pregnancy, and malignancy), creating significant issues for prognostication. They recommend D-dimer testing only in patients with a low-to-moderate pre-test probability for DVT, in which case

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Table 1: Prevention and risk stratification during prenatal care[49,50]

Category	Risk factors	Risk stratification tools	Preventive measures	Clinical considerations
Low risk	• Age <35 • BMI <25 • No personal/family history of VTE • No thrombophilia • Normal pregnancy	None typically required	• Encourage ambulation • Hydration • Routine antenatal care	No pharmacologic prophylaxis indicated
Moderate risk	• Age >35 • Obesity (BMI ≥30) • Multiparity • Assisted reproductive technology • Prolonged immobility (bed rest >3 days)	RCOG VTE Risk Assessment Tool	• Early mobilization • Consider mechanical prophylaxis (graduated compression stockings)	Monitor for symptoms throughout pregnancy and postpartum
High risk	• Personal or strong family history of VTE • Known thrombophilia (e.g., Factor V Leiden, Protein C/S deficiency) • Antiphospholipid syndrome • Previous unprovoked VTE	Modified Caprini or RCOG scoring, Thrombophilia testing if indicated	• Pharmacologic prophylaxis with LMWH antepartum and/or postpartum	Monitor anti-Xa levels if renal impairment or obesity is present. Avoid Vitamin K antagonists during pregnancy
Postpartum considerations	• Cesarean section • Postpartum hemorrhage • Preeclampsia/eclampsia	RCOG and ACOG guidelines for postpartum thromboprophylaxis	• LMWH for ≥10 days postpartum in moderate/high-risk women	The highest DVT risk is in the first 6 weeks postpartum

LMWH: Low molecular weight heparin, BMI: Body mass index, VTE: Venous thromboembolism, RCOG: Royal College of Obstetricians and Gynecologists, ACOG: American College of Obstetricians and Gynecologists, DVT: Deep vein thrombosis

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Table 2: Incidence of VTE in ICU versus non-ICU COVID-19 patients

Study	Design	Sample size	ICU VTE incidence (%)	Non-ICU VTE incidence (%)	Key findings
Sridharan *et al.*[51]	Meta-analysis (11 studies)	N/A	17.2%	Not specified	Therapeutic anticoagulation reduced VTE odds (OR=0.33)
Porfidia *et al.*[52]	Meta-analysis (30 studies)	3487	24% (95% PI 5–66%)	9% (95% PI 0–94%)	ICU patients had significantly higher VTE rates
Kollias *et al.*[53]	Meta-analysis (47 studies)	6459	PE: 32%; DVT: 27%	Not stratified	Positive correlation between ICU admission and VTE prevalence
Fontana *et al.*[54]	Review (11 studies)	1369	24.7–53.8% (screened); up to 35.3% (non-screened)	4.4–8.2% (median~6%)	ICU screening increases detected VTE rate
Mazzaccaro *et al.*[55]	Meta-analysis (69 studies)	106,838	60.8–85.4%	Not stratified	Median pooled incidence 16.7%; highest in ICU
Mai *et al.*[56]	Comparative cohort analysis	N/A	RR 3.10 (vs. ICU non-COVID controls)	RR 0.95 (vs. non-ICU controls)	COVID-19 increases ICU VTE risk significantly
Sharif-Kashani *et al.*[57]	Cohort study	N/A	22.7%	8%	Clear ICU versus non-ICU difference
Consensus/ Narrative reviews[58-63]	Expert reviews	N/A	>20% to>50% (screened)	<10%	Consistent trend of higher VTE in ICU COVID-19 patients

Pooled estimate summary. Weighted average VTE incidence (from stratified studies): (i) ICU patients: ~30.5% (95% CI: 25.0–36.0%). (ii) Non-ICU patients: ~8.5% (95% CI: 6.0–11.0%). (iii) Relative risk (ICU vs. non-ICU): RR ≈ 3.59 (95% CI approx. 2.8–4.6). VTE: Venous thromboembolism, ICU: Intensive care unit, DVT: Deep vein thrombosis

a negative D-dimer test safely rules out DVT, effectively lowering unnecessary imaging. Notably, the authors discuss younger evidence supporting age-adjusted D-dimer cutoffs, particularly for older age groups, as noted by Robert-Ebadi

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Table 3: Anticoagulation protocols: Inpatient and post-discharge (heparin, DOACs, monitoring)[48,50,64-67]

Population	Setting	Preferred anticoagulants	Dosing and duration	Monitoring considerations	Special notes
Pregnancy	Inpatient	LMWH (e.g., enoxaparin)	Weight-based dosing (e.g., 1 mg/kg BID or 1.5 mg/kg daily); continue ≥6 weeks postpartum	Anti-Xa monitoring in renal impairment, obesity, or high risk	DOACs are contraindicated in pregnancy and breastfeeding; warfarin is avoided antepartum
	Post-discharge	LMWH	Continue until ≥6 weeks postpartum (minimum total of 3–6 months treatment)	Anti-Xa if needed	Monitor adherence and signs of bleeding
COVID-19	Inpatient	LMWH or UFH (Unfractionated Heparin)	Prophylactic or intermediate dose, depending on risk stratification; escalation in ICU settings	aPTT for UFH; routine CBC and D-dimer	Heparins are preferred due to lower interaction with the inflammatory state; avoid DOACs in unstable patients
	Post-discharge	DOACs (e.g., rivaroxaban 10 mg/day for 30–45 days) or LMWH	Continue 2–6 weeks post-discharge based on thrombotic risk	No routine lab monitoring for DOACs; renal function check	Consider drug-drug interactions (e.g., antivirals); assess bleeding risk
Elderly	Inpatient	LMWH or UFH	Adjust dose for renal impairment and low body weight	Anti-Xa or aPTT as applicable; renal function monitoring	Higher bleeding risk; fall risk assessment crucial
	Post-discharge	DOACs (e.g., apixaban, rivaroxaban) or warfarin	Minimum 3 months; longer in recurrent VTE	INR for warfarin; renal and liver function for DOACs	DOACs preferred if renal function is adequate; warfarin if cost/access is an issue

LMWH: Low molecular weight heparin, DOAC: Direct oral anticoagulant, UFH: Unfractionated heparin, aPTT: activated partial thromboplastin time, INR: International Normalized Ratio, CBC: Complete blood count

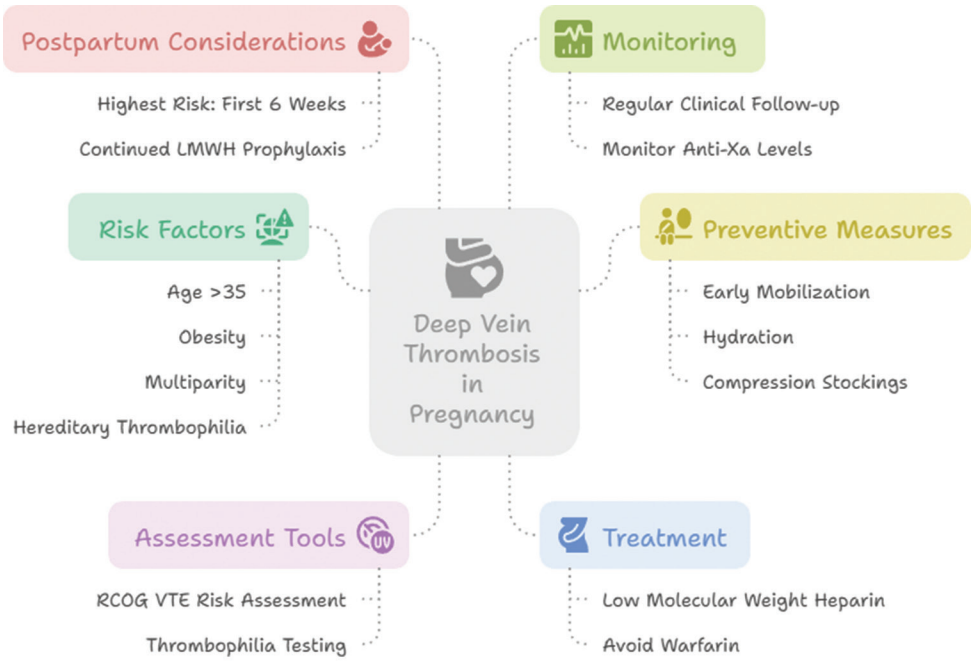

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Figure 1: Deep vein thrombosis in pregnancy: Risks and management

and Righini,[42] who found that age-adjusted cutoffs provide improved specificity without loss of sensitivity and can potentially reduce imaging by up to 30% for this population. Imaging modalities also have their own limitations. Huisman

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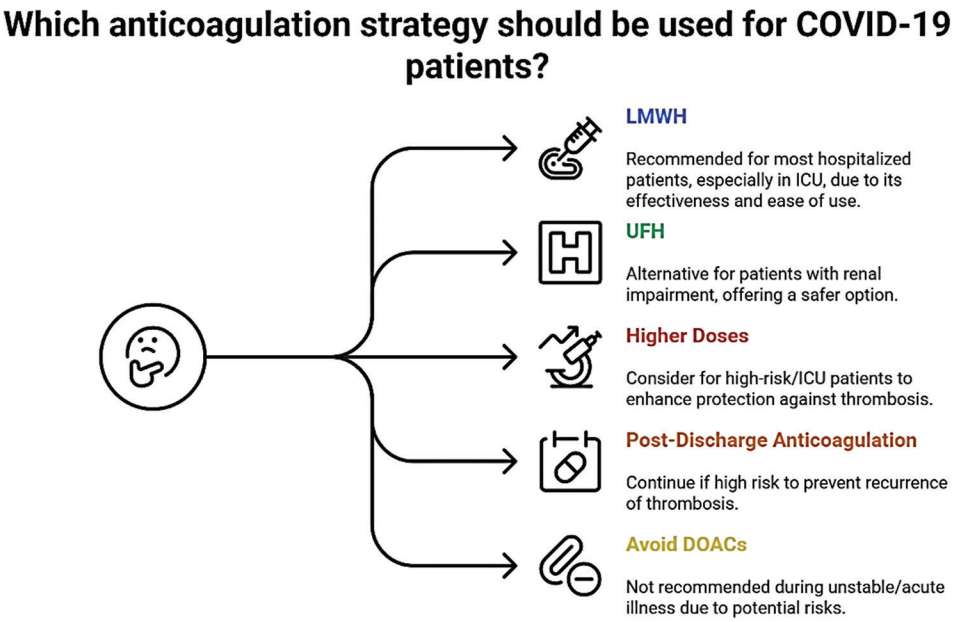
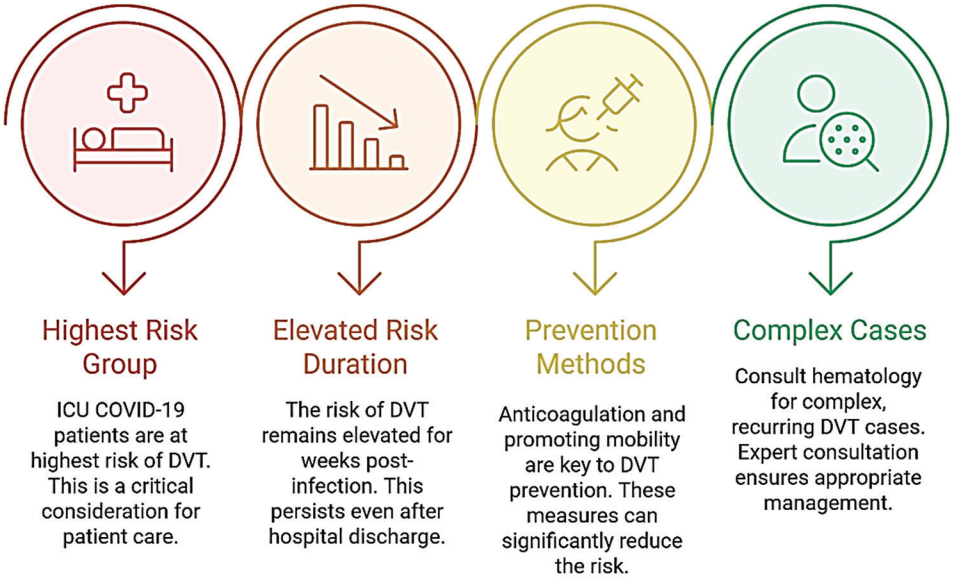


AQ3 **Figure 2:** Anticoagulation strategy for COVID-19



AQ3 **Figure 3:** Deep vein thrombosis risk factors and prevention

and Klok[43,44] provided an in-depth and thorough overview of diagnostic imaging for VTE, reporting that compression ultrasonography remains the gold standard for diagnosing DVT because of its safety and availability at the bedside. Nonetheless, sensitivity declines for pelvic and iliac vein thromboses and in morbidly obese patients. Operator dependency remains a problem. CT venography and MRI have better visualization, but there are even more chemical agents that are associated with radiation and nephrotoxicity concerns, as noted by Wadajkar *et al.*[45] The authors also mention contemporary imaging advances regarding new methods and imaging with developed novel nanotechnology, which, while still investigational, should provide greater patient specificity and reduced invasiveness.

Wrenn and Kabrhel[46] have explained further that there are challenges in the emergency department despite clinical decision support systems. Directive pathways involve complications. These complications are even more complicated given a clinical state with a high D-dimer, such as pregnancy or systemic inflammation. The authors reviewed contemporary cohorts, which indicated that D-dimer alone is inadequate in these situations without confirmation by imaging. The authors express the need to weigh the risks of radiation and nephropathy from contrast against underdiagnosing and all potential morbidity therein.

The COVID-19 pandemic added further complications, as Zanza *et al.*[47] reported that systemic inflammation and

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endothelial dysfunction resulted in frequent elevations in D-dimer, which complicated its diagnostic role. Access to imaging studies may also be hampered by infection control protocols, adding further compounding factors to trauma evaluation by intensivists. The authors recommended a lowered threshold for imaging studies within the critical care unit but acknowledged a lack of high-quality evidence supporting optimal diagnostic pathways in this subgroup.

Szymanski *et al.*[8] presented a more recent review associated with hospitalist practice written by authors with clinical knowledge, emphasizing the need for comprehensive risk stratification utilizing clinical, laboratory, and imaging data. They suggested that DOACs alter treatment pathways; however, limitations remain, primarily due to diagnostic considerations. Their review supports the appropriate utilization of validated clinical decision rules and age-adjusted D-dimer cutoff values to guide imaging utilization.

Baloira Villar and Ruiz Iturriaga[39] addressed only PE; however, they emphasized the importance of DVT as a precursor diagnosis and used clinical probability estimation with imaging and laboratory testing to guide management.

The diagnostic framework for DVT is grounded in a foundational framework, but the literature does not provide evidence to support special populations and emerging technologies. Compression ultrasonography is the safest first-line imaging modality; however, it has limitations that require cautious interpretation. CT and MRI have limitations when used in vulnerable populations, including the elderly and pregnant individuals. Although D-dimer testing is helpful, it requires a level of sophistication for interpretation. Age-adjusted cutoffs are designed to increase specificity; however, they are not as specific in stating that D-dimer is negative in patients with inflammatory states, malignancy, pregnancy, or COVID-19. The current evidence base for DVT diagnosis is limited predominantly to observational studies and expert opinions, with few trials of randomized controlled design studying the safety or accuracy of DVT diagnosis in special populations. Emerging technologies to enhance imaging and nanotechnology-aided diagnostics have potential but will require more research and evidence to establish their validity. The diagnostic evaluation of DVT must always be individualized through the use of validated clinical decision rules, judicious interpretation of D-dimer, and safe and understanding the appropriate imaging modalities and patient level of risk.[41,43,46]

Management: Preferred anticoagulation (LMWH), timing considerations, and fetal safety

DVT is the aggregation of blood clots that form in the deep veins (often in the legs). DVT is managed differently during pregnancy than in non-pregnant patients because managing DVT also means managing pregnancy. Therefore, treatment options must protect the individual while also considering the developing fetus. DVT is mainly treated with anticoagulation therapy, which can prevent the clot from growing or new clots from forming. Low-molecular-weight heparin (LMWH) is often the preferred anticoagulant in pregnant patients, as LMWH has a strong safety profile and effectiveness. LMWH does not cross the placenta, thereby minimizing fetal exposure and associated risks of bleeding or birth defects that are sometimes seen with other anticoagulants, such as warfarin.[48] In general, the timing of anticoagulation therapy is important. When DVT is diagnosed, therapeutic doses of LMWH should be started immediately to prevent clot extension and PE (a very serious and sometimes fatal complication). Prophylactic doses of LMWH are recommended for women with a history of unprovoked or hormone-associated VTE to prevent recurrence during pregnancy, and standard postpartum prophylaxis is recommended for all women with a history of VTE since the risk remains elevated even after delivery.[49] The coordination of delivery, particularly cesarean section, requires special management. Anticoagulation must be stopped or modified to reduce bleeding during delivery or surgery, but quickly restarted to reduce the risk of newly forming clots. Balancing these management decisions often relies on multidisciplinary decisions among obstetricians, hematologists, anesthesiologists, and radiologists.[48,49]

Anticoagulation decisions may become complicated during emergencies. For example, if a pregnant woman presents with extensive DVT involving the inferior vena cava, decision-making and care become more complex. Rapid anticoagulation with intravenous unfractionated heparin may be warranted because of its shorter half-life and reversibility at the time of delivery. In some cases, mechanical means or interventions may be considered, such as (for example) thrombectomy or vena cava filters as options before delivery; however, these may pose their own contraindications and risks, thus are outside the realm of this chapter; evaluation and consideration of these options on an as-needed basis, based on local expertise, is recommended.[48]

In conclusion, low molecular weight heparin remains the gold standard anticoagulant for DVT in pregnancy due to its therapeutic efficiency and safety in the fetal environment. Therapeutic anticoagulation can be initiated as soon as feasible after diagnosis, and prophylactic anticoagulation can be considered based on individualized risk factors and the assessment of potential thrombosis after delivery. Evaluation of delivery must carefully balance the risks of bleeding and clotting and may necessitate the partnership of various healthcare providers to optimize outcomes for both the mother and the fetus.[48,49]

DVT IN COVID-19

The incidence of VTE in COVID-19 patients is markedly higher in ICU settings than in non-ICU hospitalized patients. Synthesizing high-quality meta-analyses and large cohort

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studies yielded a pooled VTE incidence of approximately 30.5% in ICU patients versus 8.5% in non-ICU patients, corresponding to a relative risk of approximately 3.6. This substantial increase reflects the severity of illness, heightened inflammatory and prothrombotic states, and possibly more aggressive diagnostic surveillance in the ICU settings. The data further suggest that ICU COVID-19 patients have a 3- to 4-fold higher risk of VTE than their non-ICU counterparts.

These findings underscore the critical need for vigilant thromboprophylaxis and individualized anticoagulation strategies in ICU patients with COVID-19 infection. While therapeutic anticoagulation may reduce VTE incidence, the balance with bleeding risk requires careful management. Overall, the evidence robustly supports that ICU admission is a strong independent risk factor for VTE in patients with COVID-19.

DVT IN ELDERLY PATIENTS: RISKS, MANAGEMENT, AND PROPHYLAXIS

The incidence of DVT is markedly age-dependent, with a sharp increase in prevalence after the age of 45 years, and slightly higher rates are observed in men than in women in advanced age groups.[68,69] The risk profile of elderly individuals is multifactorial and involves both exogenous and endogenous elements. Common predisposing factors include hospitalization, surgical intervention, trauma, immobility, hormone therapy, and malignancy. Inherited and acquired thrombophilias, along with comorbidities such as obesity and cancer, further increase this risk.[70]

The management of DVT in elderly patients typically involves systemic anticoagulation, with certain clinical scenarios warranting interventional strategies.[71] A retrospective analysis of individuals aged >60 years who underwent total knee arthroplasty identified preoperative hematocrit levels, anesthesia modality, and diabetic status as independent predictors of post-operative DVT.[72] However, despite established treatment protocols, management of this population remains challenging.

Older adults often present with non-specific clinical symptoms and are prone to multiple comorbidities, polypharmacy, and frailty-related complications, which can obscure diagnosis and complicate anticoagulant management.[71] Moreover, arterial hypertension, a common condition in elderly patients, adds therapeutic complexity, especially in the context of limited high-quality evidence supporting individualized interventions in frail geriatric patients.[72]

Prophylactic strategies have shown promise in reducing DVT-related morbidity and mortality. Early identification of modifiable risk factors and prompt initiation of thromboprophylaxis are central to preventive care.[73,74] A multifactorial intervention implemented in a post-acute care setting demonstrated a significant reduction in DVT incidence among elderly patients.[74] Furthermore, the application of a standardized DVT protocol, introduced via a Knowledge Translation Committee, enhanced prophylaxis uptake in hospitalized older adults, emphasizing the importance of system-level changes in addressing this under-recognized and preventable condition.[73]

CONCLUSION

This review discusses the clinical and pathophysiological features of DVT in three high-risk populations: Pregnant women, individuals with COVID-19, and the elderly. Each group has unique risk profiles and requires individualized management strategies. Pregnancy induces a physiological hypercoagulable state, increasing DVT risk, especially in the third trimester and postpartum period. COVID-19 amplifies DVT risk in ICU patients due to systemic inflammation, cytokine storm, endothelial injury, and immobility. Elderly patients benefit from early risk stratification and prophylactic interventions. Adherence to thromboprophylaxis guidelines during pregnancy remains variable, with opportunities for improved risk-based tailoring. Limitations of this review include potential heterogeneity across source studies, lack of patient-level data, and evolving guidelines. Future directions should focus on developing integrated risk prediction models, clarifying optimal anticoagulant regimens, and improving adherence to thromboprophylaxis protocols.

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