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# Pulmonary embolism in pregnancy and the postpartum period – a literature review

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**Abstract**

**Background:** Pulmonary embolism (PE) is a leading cause of maternal morbidity and mortality worldwide. Pregnancy and the postpartum period increase the risk of venous thromboembolism due to hypercoagulability, venous stasis, and endothelial injury. Diagnosis remains challenging because symptoms often overlap with physiological changes in pregnancy, delaying recognition and increasing the risk of adverse outcomes.

**Aim**: To review current evidence on the epidemiology, risk factors, pathophysiology, clinical presentation, diagnosis, and management of pulmonary embolism during pregnancy.

**Material and methods**: A narrative literature review was conducted using publications from PubMed and Scopus, including original studies, systematic reviews, and meta-analyses on pulmonary embolism during pregnancy and the postpartum period.

**Results:** Pulmonary embolism in pregnancy presents significant diagnostic challenges due to nonspecific symptoms and physiological changes affecting clinical assessment. Pregnancy-adapted diagnostic algorithms combining clinical pre-test probability with D-dimer testing may improve diagnostic accuracy while reducing unnecessary imaging. When imaging is required, computed tomography pulmonary angiography and ventilation–perfusion scanning provide high diagnostic performance with acceptable safety. Management is based on risk stratification. Low-molecular-weight heparin is the first-line treatment for hemodynamically stable patients, whereas reperfusion therapies are reserved for high-risk cases.

**Conclusions:** Pulmonary embolism remains a serious pregnancy complication requiring early diagnosis and prompt treatment. Advances in diagnostic strategies and management have

improved patient care; however, further studies are needed to optimize risk assessment and treatment in this population.

**Key words**: pulmonary embolism, pregnancy, postpartum period, venous thromboembolism, risk factors

## 1. Introduction

Pulmonary embolism (PE) is a serious and potentially life-threatening condition caused by the obstruction of the pulmonary arteries by thromboembolic material, most commonly originating from thrombi in the deep veins of the lower extremities. Together with deep vein thrombosis, it constitutes venous thromboembolism (VTE), which represents one of the leading causes of maternal morbidity and mortality worldwide. The risk of thromboembolic events increases significantly during pregnancy and the postpartum period due to physiological changes in the coagulation system, venous stasis, and vascular endothelial injury [1].

Pregnancy is considered a hypercoagulable state, which is believed to be a protective mechanism against excessive bleeding during childbirth. However, these physiological changes also increase the risk of thrombus formation. The incidence of venous thromboembolism during pregnancy and the puerperium is estimated at approximately 1.2–1.7 cases per 1000 pregnancies, with pulmonary embolism accounting for about 0.3 cases per 1000 pregnancies. The risk of venous thromboembolism increases progressively throughout pregnancy and reaches its highest level in the postpartum period [2–4].

Diagnosis of pulmonary embolism in pregnant women remains a significant clinical challenge. Many symptoms of pulmonary embolism, such as dyspnea, tachycardia, or mild chest discomfort, are nonspecific and may also occur as part of normal physiological changes during pregnancy. Furthermore, D-dimer levels change physiologically during pregnancy, validated risk scores and clinical data are limited, and concerns about fetal exposure to ionizing radiation may complicate the decision-making process regarding diagnostic imaging. These factors may lead to delays in diagnosis and treatment, which can significantly affect maternal and fetal outcomes [5,6].

Early recognition and appropriate management of pulmonary embolism are crucial for reducing complications and mortality. Current clinical practice includes the use of diagnostic algorithms adapted for pregnant patients as well as anticoagulant therapy, with low molecular weight heparin (LMWH) considered the first-line treatment, as it does not cross the placenta and is therefore safe for both the mother and the fetus [1,5,7,8].

The aim of this study was to review the current literature regarding the epidemiology, risk factors, clinical presentation, diagnosis, treatment, and prevention of pulmonary embolism in pregnancy.

## 2. Methods

This paper is a narrative literature review. Research articles available in the PubMed and Scopus databases were analysed. Original studies, review articles, systematic reviews, and meta-analyses concerning pulmonary embolism during pregnancy and the postpartum period were included. The search was conducted using a combination of keywords and Boolean operators, including: “pulmonary embolism”, “pregnancy”, “postpartum period”, “venous thromboembolism”, “risk factors”, “diagnosis”, and “anticoagulation”. Studies published in languages other than English were excluded. No time restriction was applied.

## 3. Epidemiology

Venous thromboembolism is one of the leading causes of maternal morbidity and mortality in developed countries. Pregnancy and the postpartum period are associated with a significantly increased risk of thromboembolic events. Epidemiological data indicate that thromboembolic events occur in approximately 1.2–1.7 cases per 1000 pregnancies, with pulmonary embolism representing around 0.3 cases per 1000 pregnancies, corresponding to an approximately 5–10-fold higher risk of VTE compared with non-pregnant women [2–4,9]. Although deep vein thrombosis is more frequently diagnosed, PE represents the most severe and potentially life-threatening manifestation of VTE. PE remains one of the leading direct causes of maternal death in developed countries, accounting for approximately 5.7–9.2% of all maternal deaths [10,11]. The case-fatality rate among pregnant women diagnosed with PE is about 3%, significantly higher than that for non-pregnant women [12]. Population-based studies indicate

that the mortality rate related to pregnancy-associated pulmonary embolism is approximately 1.02 deaths per 100,000 live births [10].

The risk of venous thromboembolism, and consequently pulmonary embolism, is not evenly distributed throughout pregnancy and the postpartum period. The incidence of VTE increases slightly during the first trimester, rises more substantially in the third trimester, and reaches its peak in the first three weeks after delivery [13,14]. Approximately half of all pregnancy-associated VTE events occur in the postpartum period [15]. Although the risk of VTE remains elevated for up to 12 weeks postpartum, it is highest during the first six weeks after delivery. During this period, the risk of VTE has been reported to be approximately 21.5- to 84-fold higher than baseline levels observed in non-pregnant, non-postpartum women [16,17]. Long-term epidemiological observations indicate that while the overall incidence of VTE during pregnancy has remained relatively stable over time, the incidence of pulmonary embolism in the postpartum period declined by more than half [18]. This trend suggests improvements in the prevention and management of thromboembolic complications during the postpartum period.

## 4. Pathophysiology of venous thromboembolism in pregnancy

Pregnancy-associated pulmonary embolism develops as a consequence of physiological changes in pregnancy that reflect the components of Virchow's triad: hypercoagulability, venous stasis, and endothelial injury. These pregnancy-related adaptations are essential to limit hemorrhage during childbirth; however, they simultaneously increase the tendency for thrombus formation and the risk of embolization into the pulmonary circulation [19].

### Hypercoagulability

Pregnancy is associated with a pronounced shift toward a prothrombotic state driven by hormonal influences, particularly placental estrogens, which enhance hepatic synthesis of multiple coagulation factors. As gestation progresses, levels of several clotting factors rise substantially, with reported increases ranging from approximately 20% to 1000% for factors VII, VIII, IX, X, and XII. Fibrinogen concentrations also increase markedly, rising approximately two- to threefold during pregnancy. Additionally, von Willebrand factor levels may increase up to fourfold [20].

At the same time, anticoagulant mechanisms are attenuated. Free protein S levels decrease significantly, by up to 55%, and although antithrombin and protein C concentrations remain relatively stable, the efficiency of the protein C–dependent pathway is reduced due to alterations in thrombomodulin and protein C receptor levels, resulting in acquired activated protein C resistance [20–22].

Fibrinolysis is concurrently suppressed. Levels of plasminogen activator inhibitor-1 increase by approximately 3- to 4-fold during pregnancy, while PAI-2, which is negligible before pregnancy, reaches high concentrations at delivery [21]. These changes are accompanied by increased thrombin generation, enhanced platelet aggregation, and localized intravascular coagulation within placental vessels. As a result, D-dimer levels increase progressively throughout pregnancy [20–22].

**Venous stasis**

Venous stasis represents a key mechanism contributing to thrombus formation during pregnancy. Mechanical compression of the inferior vena cava and the left common iliac vein by the gravid uterus reduces venous return from the lower extremities and pelvic veins. In addition, progesterone-induced relaxation of the vascular wall leads to decreased venous tone, increased venous capacitance, and venous pooling. These physiological and anatomical changes promote blood stasis and increase the risk of deep vein thrombosis [1,19].

**Endothelial injury**

Endothelial injury constitutes the third component of Virchow's triad in pregnancy. Vascular damage occurs during implantation, endovascular remodeling of uterine spiral arteries, and placental separation. In addition, increased circulating cytokines and growth factors during pregnancy may contribute to endothelial dysfunction and vascular injury [23].

Endothelial injury may also result from local or systemic inflammation and hypoxemia, leading to downregulation of anticoagulant pathways and upregulation of prothrombotic mediators such as tissue factor, selectins, and von Willebrand factor, which promote leukocyte and platelet adhesion. Increased blood volume and vessel diameter during pregnancy may further enhance vascular stress, contributing to endothelial injury, particularly in the pelvic and lower limb veins [23,24].

### From deep vein thrombosis to pulmonary embolism

Most pregnancy-associated pulmonary emboli originate from thrombi formed in the proximal veins of the lower extremities or pelvic circulation. In contrast to the general population, where the majority of deep vein thromboses occur in the calf veins, pregnancy is associated with a predominance of proximal thrombosis. Approximately 62% of cases involve the iliofemoral veins, 17% the iliac veins, and only a small proportion occur in the calf veins. Left-sided thrombosis is more common, and proximal involvement of the femoral or iliac veins accounts for more than 60% of cases. This distribution is associated with an increased risk of embolization and subsequent pulmonary embolism [1,25].

## 5. Risk factors

Pulmonary embolism in pregnancy and the postpartum period is a multifactorial condition influenced by both physiological changes of pregnancy and additional maternal risk factors. As it most commonly arises from thrombi originating in the deep veins of the lower extremities or pelvic circulation, its risk factors largely overlap with those predisposing to VTE. Approximately half of women with pregnancy-associated PE present with at least one identifiable risk factor, and the coexistence of multiple factors further increases the overall risk. Importantly, many of these risk factors appear to confer a greater relative risk in pregnant women compared with the general population [22].

### Major pre-existing risk factors

One of the most important risk factors for pulmonary embolism during pregnancy and the postpartum period is a prior history of thromboembolic events. Compared with women without previous events, those with a history of venous thromboembolism are at increased risk of recurrent deep vein thrombosis and pulmonary embolism, with the risk during subsequent pregnancies estimated to be three- to fourfold higher than outside pregnancy [26,27]. This risk is further amplified by the prothrombotic state of pregnancy itself, as reflected by an approximately 3.5-fold higher rate of recurrent venous thromboembolism compared with nonpregnant women [23]. A history of superficial vein thrombosis has also been identified as an independent risk factor for thromboembolic events during pregnancy and the postpartum period [28].

Another important group of risk factors comprises inherited thrombophilias, including deficiencies of antithrombin, protein C, and protein S, as well as factor V Leiden and prothrombin gene mutations [29,30]. The highest risks are observed in women homozygous for factor V Leiden or the prothrombin G20210A variant, whereas more common forms, such as heterozygosity for these mutations, are associated with lower, although still increased, risk [27].

Other maternal characteristics further contribute to the risk of pulmonary embolism, including advanced maternal age, obesity, chronic comorbidities, parity ≥3, racial and ethnic background, and smoking [30–32]. Advanced maternal age, particularly ≥40 years, is associated with an increased risk, especially in the postpartum period [33]. Obesity appears to be more strongly associated with pulmonary embolism than with deep vein thrombosis [34]. Racial and ethnic differences have also been observed, with a higher susceptibility to pulmonary embolism reported among African American women compared with White women [23].

**Pregnancy- and delivery-related risk factors**

In addition to pre-existing maternal characteristics, several factors related to pregnancy and delivery further increase the risk of pulmonary embolism.

Pregnancy complications play an important role in thromboembolic risk. Preeclampsia, particularly when occurring preterm, has been independently associated with an increased risk of thromboembolic events. This association persists beyond pregnancy and the puerperium, suggesting a sustained prothrombotic tendency [35,36]. Pregnancy-associated hypertension has also been identified as a significant risk factor. Furthermore, assisted reproductive techniques, including in vitro fertilization, are associated with an increased likelihood of thromboembolic complications [32,33].

Delivery-related factors are particularly important, as the risk of pulmonary embolism is highest in the postpartum period. Cesarean section is one of the most significant contributors, with the risk of venous thromboembolism approximately twofold higher compared with vaginal delivery [37,38].

Additional postpartum factors include infection and immobilization, both of which further contribute to thrombus formation. Severe postpartum hemorrhage and the need for blood

transfusion represent closely related risk factors. Transfusion may reflect the severity of hemorrhage rather than act as an independent factor; however, both conditions have been associated with an increased risk of thromboembolic events. The use of coagulation products in the management of severe hemorrhage may further contribute to thrombosis [32].

## 6. Diagnosis

### Clinical presentation

Diagnosing pulmonary embolism in pregnancy remains particularly challenging due to the nonspecific nature of clinical presentation and the overlap with physiological changes of pregnancy. Symptoms such as dyspnea, tachycardia, chest pain, and lower limb discomfort are common in both pregnant women with and without pulmonary embolism, which limits the diagnostic value of clinical assessment alone. Although several clinical prediction tools, including the Wells and revised Geneva scores, are widely used in the general population, their performance in pregnancy is suboptimal and they are not considered reliable as standalone decision-making tools [39].

To overcome the limitations of conventional diagnostic tools, pregnancy-specific diagnostic approaches have been developed. One such tool is the pregnancy-adapted Geneva (PAG) score, introduced in 2021 to estimate the pre-test probability of pulmonary embolism in pregnant women and support early clinical decision-making. The score is based on a set of clinical parameters, including (1) age ≥ 40 years, (2) lower limb surgery or fracture in the last month, (3) previous deep vein thrombosis or PE, (4) unilateral lower limb pain, (5) hemoptysis, (6) tenderness of the lower limb and unilateral edema, and (7) heart rate > 110 bpm (Table 1). It enables stratification of patients into different probability categories and has shown good ability to distinguish between risk levels, although further prospective validation in independent populations is still needed [40].

Table 1. The Pregnancy-Adapted Geneva score (modified from Robert-Ebadi et al. [40])

**Clinical variables and assigned points**

| Clinical feature | Points |
|---|---|

**Clinical variables and assigned points**

| | |
|---|---|
| Age ≥ 40 years | 1 |
| Recent lower limb surgery or fracture (within 1 month) | 2 |
| Previous DVT or PE | 3 |
| Unilateral lower limb pain | 3 |
| Hemoptysis | 2 |
| Unilateral lower limb tenderness with edema | 4 |
| Heart rate > 110 bpm | 5 |
| **Maximum total score** | **20** |

| Score | Clinical probability | PE prevalence |
|---|---|---|
| 0-1 | Low | 1.0–4.9% |
| 2-7 | Intermediate | 6.9–18.9% |
| ≥7 | High | 35.5–82.2% |

## D-dimer levels and clinical pre-test probability

The interpretation of D-dimer levels in pregnancy is particularly challenging due to physiological changes in the hemostatic system. D-dimer concentrations increase progressively throughout gestation and reach their highest values in the third trimester, reflecting pregnancy-related activation of coagulation. According to available studies, D-dimer levels exceed the conventional cut-off value (500 µg/L) in up to 99% of women in the third trimester. Consequently, pregnancy-specific reference ranges have been proposed to account for these physiological changes, with reported values of 169–1202 µg/L in the first trimester, 393–3258 µg/L in the second trimester, and 551–3333 µg/L in the third trimester [41]. In addition, alternative trimester-adjusted cut-off values have been suggested in other studies. For example, Kovac et al. proposed lower diagnostic thresholds of 286 ng/mL, 457 ng/mL, and 644 ng/mL

for the first, second, and third trimester, respectively, as potential criteria for excluding pulmonary embolism in pregnant patients [42].

Despite these findings, D-dimer testing alone is insufficient to exclude pulmonary embolism in pregnancy [43]. Therefore, the diagnostic value of D-dimer is significantly enhanced when interpreted in conjunction with clinical pre-test probability. Evidence from recent studies supports the use of integrated diagnostic algorithms, such as the pregnancy-adapted YEARS model, which combines selected clinical criteria with D-dimer testing to guide further management. This approach relies on a limited set of key clinical features, including signs of deep vein thrombosis, hemoptysis, and the clinician's assessment of pulmonary embolism as the most likely diagnosis. Within this framework, D-dimer thresholds are adapted according to clinical findings: a cut-off of 500 µg/L is applied in patients with at least one of the YEARS criteria, whereas a higher threshold of 1000 µg/L may be used when none of these features are present. This strategy enables safe exclusion of pulmonary embolism in a subset of pregnant women without the need for imaging, thereby reducing unnecessary radiation exposure. Although its diagnostic efficiency is lower in the third trimester, it still allows a meaningful proportion of patients to avoid computed tomography pulmonary angiography [44].

Additional evidence comes from the study by Kearon et al., which showed that pulmonary embolism may be excluded without further diagnostic testing in patients with low clinical pre-test probability and D-dimer levels below 1000 ng/mL, as well as in those with intermediate probability when D-dimer levels remain below 500 ng/mL [45]. Collectively, these findings support the concept that adjusting D-dimer interpretation to clinical probability represents a safe and effective strategy in the diagnostic evaluation of suspected pulmonary embolism during pregnancy.

**Imaging**

When pulmonary embolism cannot be excluded based on clinical assessment and laboratory testing, further imaging is required. Compression ultrasonography of the lower limbs is recommended as an initial step in the presence of symptoms suggestive of deep vein thrombosis, although its diagnostic yield is limited in asymptomatic patients and in cases of isolated pelvic thrombosis [46]. If chest imaging is necessary, both computed tomography pulmonary angiography (CTPA) and ventilation–perfusion (V/Q) scanning demonstrate high diagnostic accuracy and comparable safety profiles, with fetal radiation exposure remaining

well below harmful thresholds [47]. While V/Q scanning may be preferred in patients with a normal chest radiograph, CTPA is more widely available and allows assessment of alternative diagnoses. Importantly, the risk associated with imaging is considered lower than the risk of a missed diagnosis [48].

### Additional diagnostic tools

A range of additional methods may provide supportive information in selected clinical scenarios. One of these is electrocardiography, in which sinus tachycardia represents the most common finding. Other features, such as the S1Q3T3 pattern, P pulmonale, and right axis deviation, may also be observed, although they occur less frequently. Right bundle branch block, atrial arrhythmias, and clockwise rotation have also been reported; however, these findings are nonspecific and may be similarly present in control populations [49].

Echocardiography is widely available and can be used as a bedside tool in the emergency setting to support the diagnosis, particularly in patients with suspected high-risk pulmonary embolism, where it may facilitate prompt initiation of treatment [50]. Emerging diagnostic modalities, including magnetic resonance imaging and lung ultrasound, show potential value; however, their routine use remains limited due to insufficient evidence and restricted availability [51,52].

Overall, contemporary diagnostic strategies based on pregnancy-specific clinical assessment, D-dimer testing, and the selective use of imaging modalities enable safe and effective diagnosis of pulmonary embolism in pregnancy while minimizing unnecessary radiation exposure.

## 7. Treatment

Pulmonary embolism in pregnancy requires prompt and carefully tailored management, balancing maternal benefit against potential fetal risk. The overall principles of treatment are similar to those applied in the general population; however, therapeutic decisions must account for pregnancy-specific physiological changes, altered pharmacokinetics, and concerns related to fetal safety. Importantly, general principles of risk stratification used in nonpregnant patients can also be applied in pregnant women, allowing classification into low-, intermediate-, and high-risk pulmonary embolism to guide treatment decisions, although their use requires careful clinical interpretation in the context of pregnancy [31].

## Anticoagulant Therapy

In hemodynamically stable patients, anticoagulation is recommended as first-line therapy. Both low molecular weight heparin (LMWH) and unfractionated heparin (UFH) are considered safe during pregnancy and breastfeeding, as they do not cross the placental barrier or pass into breast milk. Subcutaneous administration of LMWH is the preferred therapeutic approach due to its more predictable pharmacokinetic profile and anticoagulant effect compared with UFH [53]. The standard dosing regimen for enoxaparin is 1 mg/kg administered subcutaneously every 12 hours, due to limited evidence supporting once-daily dosing in pregnancy [54,55]. Current guidelines do not recommend routine monitoring of anti-factor Xa levels in pregnant women with normal renal function receiving LMWH [54]. The use of novel oral anticoagulants is contraindicated in pregnancy, as they are able to cross the placenta, potentially affecting the fetal coagulation system and exerting teratogenic effects [31].

## Reperfusion therapy

Mortality in acute pulmonary embolism is primarily related to the development of obstructive shock and ensuing hemodynamic instability. Reperfusion therapies are therefore designed to promptly eliminate the obstructing thrombus, leading to restoration of pulmonary blood flow and stabilization of the patient's circulatory status.

Systemic thrombolysis, administered intravenously, is recommended as first-line therapy exclusively in hemodynamically unstable patients with high-risk pulmonary embolism [56]. Due to its more favorable bleeding profile, alteplase is currently the most commonly used fibrinolytic agent. Available data suggest that fibrinolytic drugs do not cross the placenta in clinically significant amounts at therapeutic doses, and animal studies have not demonstrated teratogenic effects [57].

Systemic thrombolysis is associated with favorable maternal outcomes, with reported survival rates of approximately 92%. However, treatment carries a substantial risk of complications, including cardiac arrest (17.6%) and major maternal hemorrhage (28.4%) [58]. Despite these risks, the high likelihood of rapid clinical deterioration and death in untreated high-risk pulmonary embolism supports the use of thrombolysis in life-threatening cases during pregnancy [59].

Catheter-Directed Therapy (CDT) represents a potential alternative to systemic thrombolysis. These techniques have been proposed as a means of achieving thrombus resolution with a lower risk of bleeding and may therefore be particularly relevant in the peripartum period, when hemorrhagic risk is highest. CDT is generally considered in high-risk patients in whom thrombolysis or adequately dosed anticoagulation has failed or is contraindicated [56]. Reported rates of major bleeding, including intracranial hemorrhage, are approximately 18% [60]. Although early data on the safety and efficacy of catheter-based approaches are encouraging, robust evidence on both short- and long-term outcomes remains limited, and their routine use cannot yet be recommended [61]. Treatment decisions should therefore be individualized and based on expert multidisciplinary assessment.

Surgical embolectomy using cardiopulmonary bypass may be considered in cases of hemodynamic instability or when anticoagulation and thrombolytic therapy are ineffective or contraindicated [62]. The procedure involves removal of thrombi from the main pulmonary arteries via surgical access. Reported outcomes include hemodynamic improvement and/or return of spontaneous circulation in approximately 93.8% of patients, with maternal survival rates ranging from 84% to 86.1% [58].

## 8. Conclusions

Pulmonary embolism remains a significant cause of maternal morbidity and mortality, with pregnancy and the postpartum period representing a state of increased thromboembolic risk due to physiological changes affecting all components of Virchow's triad. The overlap between symptoms of pulmonary embolism and normal pregnancy-related changes continues to pose a major diagnostic challenge, often complicating timely recognition of the condition.

Recent advances in diagnostic strategies, including pregnancy-adapted clinical algorithms and the use of D-dimer in combination with clinical pre-test probability, have improved the safety and efficiency of the diagnostic process, allowing for a reduction in unnecessary imaging while maintaining a low risk of missed diagnoses. Imaging modalities such as CTPA and V/Q scanning remain essential when pulmonary embolism cannot be excluded by non-invasive methods, with acceptable safety profiles for both the mother and the fetus.

Management of pulmonary embolism in pregnancy requires an individualized approach that balances maternal benefit with fetal safety. Anticoagulation with low molecular weight heparin remains the cornerstone of treatment in hemodynamically stable patients, while reperfusion therapies, including systemic thrombolysis, catheter-directed techniques, and surgical embolectomy, are reserved for high-risk cases with hemodynamic instability.

Despite growing evidence and improvements in clinical practice, several aspects of pulmonary embolism in pregnancy remain insufficiently studied, particularly regarding optimal risk stratification, long-term outcomes, and the role of emerging diagnostic and therapeutic modalities.

In conclusion, early recognition, appropriate risk assessment, and timely initiation of treatment are essential to improving maternal outcomes. Continued research and the development of pregnancy-specific clinical guidelines are necessary to further optimize the management of pulmonary embolism in this unique patient population.

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