

CASE REPORT

Inherited Thrombophilia as a Cause of Post-Partum Massive Pulmonary Embolism: A Case Report

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ABSTRACT

Introduction: Pulmonary embolism (PE) is a life-threatening manifestation of venous thromboembolism (VTE) and remains one of the leading causes of maternal morbidity and mortality worldwide. The post-partum period is associated with a heightened hypercoagulable state, which may be further amplified by inherited thrombophilia such as protein S deficiency. Early recognition is challenging due to the non-specific clinical presentation and the overlap of symptoms with physiological post-partum changes.

Case Report: We report the case of a 28-year-old female who presented to the emergency department in an unconscious state following sudden collapse, 15 days after an uncomplicated lower segment caesarean section. The patient had a preceding history of unilateral leg pain. Initial resuscitation was successful, and subsequent investigations revealed massive pulmonary embolism with right ventricular strain. Laboratory evaluation demonstrated markedly elevated D-dimer levels and significantly reduced free protein S levels, consistent with protein S deficiency. The patient was managed with systemic thrombolysis followed by intensive care support and long-term anticoagulation.

Conclusion: Pulmonary embolism should be considered in all post-partum patients presenting with sudden collapse or unexplained hemodynamic instability. Early diagnosis, prompt thrombolytic therapy in appropriate cases, and evaluation for underlying thrombophilia are essential to reduce mortality and guide long-term management strategies.

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KEYWORDS:

• Pulmonary Embolism • Protein S Deficiency • Post-Partum • Venous Thromboembolism • Thrombolysis • Emergency Medicine

Key Message:

- Pulmonary embolism is a catastrophic yet potentially reversible cause of sudden collapse in post-partum patients.
- In young individuals with massive PE, inherited thrombophilia such as protein S deficiency should be actively investigated, as identification significantly influences acute management, duration of anticoagulation, and future pregnancy counselling.

INTRODUCTION

Pulmonary embolism (PE) is a potentially fatal manifestation of venous thromboembolism (VTE) and remains a major cause of sudden cardiovascular collapse in both medical and obstetric populations. Despite advances in diagnostic imaging and therapeutic strategies, PE continues to be associated with significant morbidity and mortality, particularly when presentation is acute and hemodynamic compromise is present. In the obstetric population, pulmonary embolism remains one of the leading causes of maternal mortality worldwide, accounting for a substantial proportion of pregnancy-related deaths, especially during the post-partum period.¹

Massive pulmonary embolism, characterized by sustained hypotension, shock, or cardiac arrest, represents the most severe form of the disease and is associated with high early mortality. Right ventricular failure resulting from acute pressure overload plays a central role in the pathophysiology and prognostication of massive PE. Early identification and prompt initiation of reperfusion therapy, including systemic thrombolysis, are crucial to improving outcomes in these patients. However, the decision to administer thrombolytic therapy in post-partum patients is complex due to concerns regarding bleeding risk, necessitating careful risk-benefit assessment.

Pregnancy and the puerperium are well-recognized hypercoagulable states

Physiological adaptations during pregnancy lead to profound changes in the haemostatic system, including increased levels of pro-coagulant factors such as fibrinogen and clotting factors VII, VIII, IX, and X, along with reduced fibrinolytic activity. These changes serve a

protective role in minimizing haemorrhage during delivery; however, they simultaneously increase the risk of thromboembolic events. Venous stasis caused by compression of the inferior vena cava and pelvic veins by the gravid uterus, endothelial injury during vaginal delivery or surgical intervention, and post-partum immobility further compound this risk. The hypercoagulable state persists into the post-partum period, with the first six weeks after delivery representing the period of highest thromboembolic risk.²

The incidence of pregnancy-associated venous thromboembolism is estimated to be approximately 1–2 per 1000 pregnancies, with the risk of pulmonary embolism being significantly higher in the post-partum period compared to the antepartum period. Caesarean section is an independent and well-established risk factor, conferring a two-to four-fold increase in the risk of VTE compared to vaginal delivery. Additional risk factors include advanced maternal age, obesity, prolonged immobilization, postpartum haemorrhage, infection, and inherited or acquired thrombophilia.

Inherited thrombophilia plays a critical role in the pathogenesis of venous thromboembolism, particularly in young patients presenting with unprovoked or severe thrombotic events. Among these, protein S deficiency is a rare but clinically significant disorder. Protein S is a vitamin K-dependent plasma glycoprotein that functions as an essential cofactor for activated protein C in the regulation of coagulation. By facilitating the inactivation of clotting factors Va and VIIIa, protein S plays a pivotal role in maintaining haemostatic balance. Deficiency of protein S leads to impaired anticoagulant function,

resulting in a prothrombotic state and increased susceptibility to venous thrombosis.³

Protein S deficiency may be inherited or acquired and is classified into three types based on levels of total protein S, free protein S, and functional activity. Inherited protein S deficiency is most commonly autosomal dominant and may remain clinically silent until exposure to additional prothrombotic triggers such as pregnancy, surgery, trauma, or prolonged immobilization. The true prevalence of protein S deficiency in the general population is estimated to be between 0.03% and 0.13%, though its contribution to thromboembolic events is likely underrecognized due to limited routine screening and diagnostic challenges during acute illness or pregnancy.

Diagnosis: The diagnosis of pulmonary embolism in the post-partum period is often challenging due to the overlap of symptoms with common physiological changes of pregnancy and puerperium. Dyspnoea, tachycardia, lower limb discomfort, and fatigue may be misattributed to post-operative pain, anaemia, or deconditioning. Additionally, clinical prediction scores commonly used in the general population have limited validation in pregnant and post-partum patients. As a result, a high index of clinical suspicion is essential, particularly in patients presenting with sudden collapse, unexplained hypoxia, or hemodynamic instability.

Recognition of underlying thrombophilia in patients presenting with life-threatening pulmonary embolism has important implications beyond the acute management phase. Identification of conditions such as protein S deficiency influences the duration of anticoagulation therapy, guides counselling regarding future pregnancies, and informs strategies for thromboprophylaxis during subsequent high-risk periods. Despite this, routine screening for inherited thrombophilia is not universally recommended and is often reserved for selected patients with unprovoked, recurrent, or unusually severe thromboembolic events.

This case report describes a young post-partum female who presented with sudden cardiovascular collapse due to massive pulmonary embolism secondary to protein S deficiency. The case highlights the diagnostic challenges, the importance of

rapid resuscitation and thrombolytic therapy, and the need for heightened awareness of inherited thrombophilia in the post-partum population. Through this report, we aim to emphasize the critical role of early recognition and comprehensive evaluation in improving outcomes for this potentially fatal yet treatable condition.

CASE REPORT

A 28-year-old female was brought to the emergency department in an unconscious state following a sudden collapse at home. She was 15 days post an uncomplicated lower segment caesarean section performed for obstetric indications. According to her family members, the patient had complained of right-sided lower limb pain for four days prior to presentation, which acutely worsened approximately two hours before collapse. There was no history of chest pain, dyspnoea, fever, seizures, trauma, or syncope prior to the event. She had no known comorbidities, was a non-smoker, and had no prior history of thromboembolic disease.

On arrival: The patient was unresponsive with a Glasgow Coma Scale score of E1V1M1.

Central pulses not palpable.

Blood pressure: Not recordable

No spontaneous respiration noted

Oxygen saturation: could not be measured in view of cold extremities.

Cardiopulmonary resuscitation was initiated immediately as per Advanced Cardiac Life Support (ACLS) protocol. Return of spontaneous circulation was achieved, following which the patient was endotracheally intubated and mechanically ventilated. Intravenous fluids and vasopressor support were initiated to stabilize her hemodynamic status.

Following initial stabilization, a focused clinical assessment was performed.

Given the sudden cardiovascular collapse in the post-partum period and the antecedent history of unilateral leg pain, pulmonary embolism was considered the leading diagnosis.

Differential diagnoses included amniotic fluid embolism, acute coronary syndrome, intracranial catastrophe, and septic shock.

Electrocardiography demonstrated sinus tachycardia with an S1Q3T3 pattern. Bedside two-dimensional echocardiography revealed a

dilated right ventricle with preserved left ventricular systolic function and no regional wall motion abnormalities, suggestive of acute right heart strain.

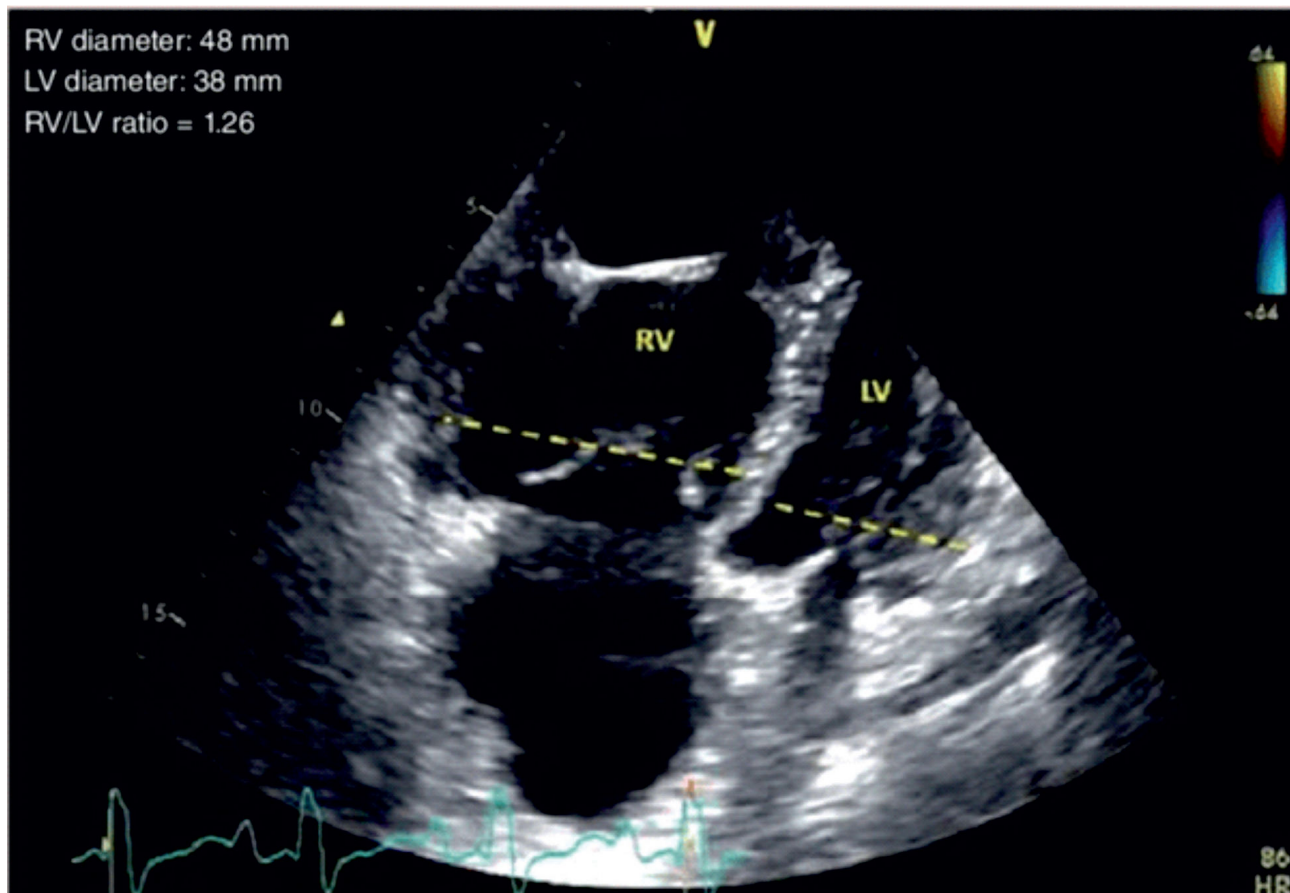


Figure 1: Two-dimensional transthoracic echocardiography showing right ventricular dilation with features of acute right ventricular pressure overload, consistent with massive pulmonary embolism

Arterial blood gas analysis showed metabolic acidosis with hypoxemia.

Once the patient was hemodynamically stabilized, computed tomography pulmonary angiography (CTPA) was performed, which demonstrated acute pulmonary thromboembolism involving the main pulmonary arteries.

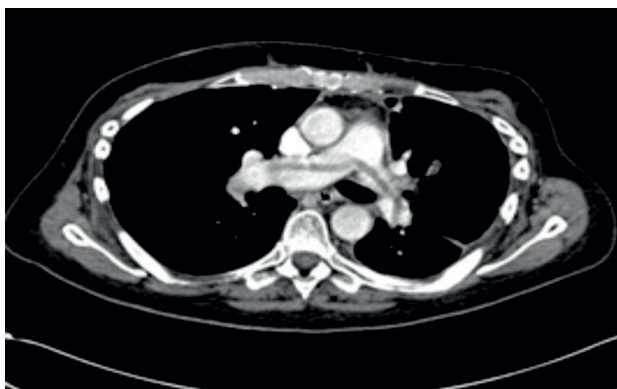


Figure 2: Computed tomography pulmonary angiography demonstrating intraluminal filling defects within the pulmonary arteries, confirming the diagnosis of acute pulmonary embolism

Based on the clinical presentation and imaging findings, a diagnosis of massive pulmonary embolism was established.

In view of massive pulmonary embolism with hemodynamic instability, systemic thrombolytic therapy with intravenous alteplase was administered after careful assessment of bleeding risk. The patient was subsequently transferred to the intensive care unit for further management. She was maintained on mechanical ventilation, anticoagulation therapy, vasopressor support, and close hemodynamic monitoring.

Initial laboratory investigations revealed a markedly **elevated D-dimer level of 67,774 ng/ml** (reference range: 0–243 ng/ml).

Given the patient's young age and the severity of the presentation, an evaluation for underlying thrombophilia was initiated. Coagulation and thrombophilia workup revealed a **significantly reduced free protein**

S level of 13.6 (reference range: 74–146), while protein C functional assay was within normal limits. These findings were consistent with protein S deficiency.

Over the next 48 hours, the patient demonstrated gradual clinical improvement with stabilization of vital parameters and decreasing vasopressor requirements. She was successfully weaned off ventilatory support and extubated once her neurological status improved. Anticoagulation therapy was continued, and multidisciplinary care involving emergency medicine, critical care, cardiology, and haematology teams was provided.

Following systemic thrombolysis and initial stabilization in the intensive care unit, the patient was continued on therapeutic anticoagulation with parenteral heparin. After adequate overlap, oral anticoagulation with **warfarin** was initiated, with careful INR monitoring. Given the presence of inherited protein S deficiency and the life-threatening presentation of massive pulmonary embolism, a decision was made to continue **long-term anticoagulation with warfarin**, targeting an INR of 2.0–3.0. The patient was counselled regarding the need for regular INR monitoring, drug–diet interactions, and adherence to therapy.

Table 1: Timeline of events

Timeline	Clinical Event
Post-operative day 11	Onset of right-sided lower limb pain
Post-operative day 15	Sudden collapse at home; brought to emergency department
Emergency department arrival	Unconscious, hypotensive, tachycardic; CPR initiated
Immediate management	ROSC achieved; intubation and mechanical ventilation
Initial evaluation	Elevated D-dimer; ECG with S1Q3T3; echocardiography showing RV dilation
Diagnostic imaging	CT pulmonary angiography confirming massive pulmonary embolism
Etiological evaluation	Thrombophilia workup revealing protein S deficiency
Definitive treatment	Systemic thrombolysis with intravenous alteplase
ICU course	Hemodynamic stabilization; ventilatory and vasopressor support
Clinical improvement	Extubation and transition to anticoagulation therapy
Discharge	Stable condition; advised lifelong anticoagulation and follow-up

The patient was discharged in a stable condition on oral warfarin with instructions for regular INR monitoring and outpatient follow-up; however, further clinical follow-up could not be obtained as the patient was lost to follow-up.

DISCUSSION

This case illustrates several clinically important aspects of pulmonary embolism management in the post-partum period, particularly when complicated by an underlying inherited thrombophilia. The patient's presentation with sudden cardiovascular collapse underscores the unpredictable and catastrophic nature of massive pulmonary embolism, emphasizing the need for rapid recognition and decisive intervention in the emergency setting.

One of the key diagnostic challenges highlighted by this case is the nonspecific nature of early symptoms preceding collapse. The

patient had reported unilateral lower limb pain for several days prior to presentation, which is a well-recognized prodromal symptom of deep vein thrombosis. However, such symptoms are frequently overlooked or attributed to benign musculoskeletal causes in post-operative or post-partum patients. This case reinforces the importance of maintaining a high index of suspicion for venous thromboembolism when lower limb symptoms occur in high-risk clinical contexts, even in young patients without known comorbidities.^{1,2}

The role of bedside investigations in the early identification of massive pulmonary embolism is also demonstrated. Electrocardiographic findings such as sinus tachycardia and the S1Q3T3 pattern, although neither sensitive nor specific, can provide supportive evidence when interpreted in the appropriate clinical setting. Bedside transthoracic echocardiography was particularly valuable in this case, revealing right ventricular dilation

and pressure overload. Echocardiographic evidence of acute right heart strain is a critical marker of disease severity and has important prognostic implications, guiding the urgency of reperfusion therapy.⁵

Systemic thrombolysis remains the treatment of choice for massive pulmonary embolism associated with hemodynamic instability. In post-partum patients, the decision to administer thrombolytic therapy can be challenging due to concerns regarding haemorrhagic complications. However, current international guidelines clearly support thrombolysis in life-threatening pulmonary embolism after individualized risk-benefit assessment.⁶ In the present case, prompt administration of intravenous alteplase resulted in rapid hemodynamic stabilization, supporting its role as a life-saving intervention despite the inherent bleeding risks associated with the post-partum state.

An important aspect of this case is the identification of protein S deficiency as the underlying etiological factor. While pregnancy and caesarean section are recognized transient risk factors for venous thromboembolism, the severity of presentation in a young patient prompted evaluation for an inherited thrombophilia. Protein S deficiency is known to significantly increase the risk of venous thrombosis, particularly when combined with additional prothrombotic triggers.^{3,4} Identifying this condition has crucial implications for long-term management, recurrence prevention, and counselling regarding future pregnancies.

Long-term anticoagulation strategy in patients with inherited thrombophilia must be individualized. Given the life-threatening nature of the thromboembolic event and the presence of protein S deficiency, long-term anticoagulation was considered appropriate in this patient. Transition from parenteral anticoagulation to oral warfarin with appropriate overlap was undertaken, with a planned target INR of 2.0–3.0. Vitamin K antagonists remain an acceptable option for long-term anticoagulation outside pregnancy, particularly in resource-limited settings and where close INR monitoring is feasible.⁷

Counselling regarding future risk modification is an essential component of care in such patients. Women with protein S deficiency require multidisciplinary follow-up and thromboprophylaxis planning during

future pregnancies and other high-risk situations. Low molecular weight heparin is preferred during pregnancy and the immediate post-partum period, while avoidance of oestrogen-containing contraceptives and early mobilization during hospitalizations should be emphasized.

A limitation of this case is the inability to assess long-term outcomes, as the patient was subsequently lost to follow-up. This reflects a common challenge in real-world clinical practice, particularly in acute care settings, and highlights the importance of patient education and structured follow-up pathways for individuals diagnosed with inherited thrombophilia.

Overall, this case reinforces the need for early clinical suspicion, judicious use of bedside diagnostic tools, timely reperfusion therapy, and etiological evaluation in young patients presenting with massive pulmonary embolism. Recognition of underlying thrombophilia not only influences acute management but also plays a pivotal role in preventing recurrence and guiding long-term care.

CONCLUSION

Pulmonary embolism in the post-partum period represents a medical emergency with potentially catastrophic outcomes if not recognized and treated promptly. This case highlights the importance of maintaining a high index of suspicion for pulmonary embolism in post-partum patients presenting with sudden cardiovascular collapse or unexplained hemodynamic instability, even in the absence of classic respiratory symptoms. Early recognition, rapid resuscitation, and timely diagnostic evaluation remain critical determinants of survival.

The present case underscores the clinical value of bedside investigations such as electrocardiography and transthoracic echocardiography in the emergency setting. While these modalities are not diagnostic, they provide crucial information regarding disease severity and right ventricular involvement, thereby guiding urgent therapeutic decisions. Confirmation with CT pulmonary angiography remains essential to establish the diagnosis and direct definitive management.

Systemic thrombolysis continues to be

the cornerstone of treatment for massive pulmonary embolism associated with hemodynamic compromise. Although the post-partum state is associated with an increased risk of bleeding, this case reinforces that life-threatening pulmonary embolism constitutes a clear indication for thrombolytic therapy following careful risk-benefit assessment. The favourable short-term outcome observed after thrombolysis in this patient supports its role as a life-saving intervention.

Identification of protein S deficiency as the underlying etiological factor had significant implications for long-term management. Inherited thrombophilia should be actively considered in young patients presenting with severe or unprovoked thromboembolic events, as recognition influences decisions regarding the duration of anticoagulation, counselling for future pregnancies, and thromboprophylaxis during high-risk periods. Current obstetric guidelines emphasize the importance of risk stratification and appropriate anticoagulation strategies during pregnancy and the puerperium in women with thrombophilia.⁸ Transition to long-term oral anticoagulation with warfarin was appropriate in this case given the severity of presentation and the presence of an inherited prothrombotic condition.

Although long-term outcomes could not be assessed due to the patient being lost to follow-up, this limitation reflects a

common challenge in real-world acute care practice. Overall, this case emphasizes the need for heightened clinical awareness, multidisciplinary management, and structured follow-up pathways to reduce morbidity and mortality associated with post-partum pulmonary embolism.

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