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Coagulation Abnormalities in Preeclampsia: Diagnostic and Prognostic Perspectives- A Perspective

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Abstract

Preeclampsia is a complex hypertensive disorder of pregnancy with significant maternal and perinatal risks. Coagulation abnormalities, often overshadowed by cardiovascular and renal manifestations, play a pivotal role in its pathogenesis and progression. Endothelial dysfunction and systemic inflammation activate the coagulation cascade, leading to platelet consumption, fibrin deposition, and impaired fibrinolysis. Laboratory markers—including platelet count, D-dimer, fibrinogen, and prothrombin time—offer valuable diagnostic insight, while dynamic changes in these parameters can predict disease severity and adverse outcomes. Early identification of coagulation disturbances enables timely intervention, optimizes delivery planning, and reduces complication risk. This perspective underscores the importance of integrating coagulation monitoring into standard preeclampsia management and calls for research into biomarker thresholds and intervention strategies to improve maternal–fetal outcomes.

Keywords: Preeclampsia, Coagulation Abnormalities, D-dimer, Fibrinogen, Prognosis

Introduction

Preeclampsia is a multisystem hypertensive disorder unique to human pregnancy, affecting 2–8% of pregnancies globally and contributing substantially to maternal and perinatal morbidity and mortality. It is characterized by the new onset of hypertension after 20 weeks of gestation, frequently accompanied by proteinuria and, in severe cases, multi-organ dysfunction. Despite advances in antenatal surveillance, preeclampsia remains a leading cause of preterm delivery, maternal death, and adverse neonatal outcomes, particularly in low- and middle-income countries where access to specialized obstetric care is limited [1-2].The pathogenesis of preeclampsia is multifactorial, involving abnormal placentation, exaggerated systemic inflammation, oxidative stress, and profound endothelial dysfunction. While much attention has been given to its cardiovascular and renal manifestations, a significant yet underappreciated aspect of the disease lies in the perturbations of the hemostatic system. These coagulation abnormalities not only reflect the disease's severity but also contribute directly to maternal and fetal complications [3-4].
Normal pregnancy induces a physiologic hypercoagulable state, an adaptive mechanism to minimize blood loss at delivery. This is characterized by increased levels of clotting factors, reduced fibrinolysis, and heightened platelet activation. In preeclampsia, however, this delicate balance is disrupted. Endothelial injury and the release of procoagulant microparticles trigger excessive activation of the coagulation cascade, often resulting in widespread microthrombi formation and consumptive coagulopathy in severe cases [5-6].Coagulation abnormalities in preeclampsia can be detected through a range of laboratory tests. Conventional parameters such as platelet count, prothrombin time (PT), and activated partial thromboplastin time (aPTT) are widely used in clinical practice. More specific markers—including elevated D-dimer levels, altered fibrinogen concentrations, and changes in platelet indices—provide additional insights into disease dynamics. Importantly, these markers can change rapidly, reflecting the evolving pathophysiology and guiding timely clinical decisions [7-8].

While preeclampsia is diagnosed primarily based on blood pressure and proteinuria criteria, incorporating coagulation assessment into the diagnostic process offers several benefits. Subtle changes in platelet count or fibrinogen levels may precede overt clinical deterioration, providing an early warning for impending complications such as HELLP syndrome (Hemolysis, Elevated Liver enzymes, Low Platelets) or disseminated intravascular coagulation (DIC). Such early detection is especially critical in resource-constrained settings, where delays in diagnosis can lead to devastating outcomes [9-10].Beyond their diagnostic value, coagulation parameters hold significant prognostic potential. Persistent thrombocytopenia, progressive hypofibrinogenemia, and disproportionately elevated D-dimer levels have been linked to adverse maternal outcomes including eclampsia, postpartum hemorrhage, and organ failure. From the fetal perspective, coagulation derangements correlate with placental insufficiency, intrauterine growth restriction, and stillbirth. Recognizing these associations can improve risk stratification, optimize delivery timing, and guide the intensity of monitoring [11].

Given the central role of coagulation disturbances in preeclampsia and their underutilization in clinical protocols, there is a pressing need to reframe them as essential diagnostic and prognostic tools rather than ancillary findings. This perspective aims to synthesize current knowledge on coagulation abnormalities in preeclampsia, highlight their diagnostic and prognostic applications, and propose avenues for integrating biomarker-based coagulation monitoring into routine obstetric care. By bridging pathophysiological insights with clinical practice, we can enhance maternal–fetal outcomes and strengthen preeclampsia management strategies [12-14].

Aim

The aim of this perspective article is to explore the role of coagulation abnormalities in preeclampsia, highlighting their diagnostic and prognostic significance, and to discuss how monitoring these disturbances can inform risk stratification, clinical management, and maternal-fetal outcomes.

Coagulation Pathophysiology in Preeclampsia

Preeclampsia represents a pathological exaggeration of the normal hypercoagulable state of pregnancy, driven by a complex interplay between endothelial injury, systemic inflammation, platelet activation, and dysregulated fibrinolysis. The initiating event is thought to be abnormal placentation, which results in impaired spiral artery remodeling and reduced uteroplacental perfusion. This ischemic environment promotes oxidative stress and the release of antiangiogenic factors such as soluble fms-like tyrosine kinase-1 (sFlt-1), which directly injure the maternal vascular endothelium(Table 1) [15-17].

Table 1: Coagulation Pathophysiology in Preeclampsia

Pathophysiological Feature	Mechanism	Clinical Implications
Hypercoagulability	Elevated procoagulant factors (factor VIII, fibrinogen, von Willebrand factor) and increased platelet activation.	Promotes placental microthrombosis, fetal growth restriction, and placental insufficiency.
Platelet Dysfunction and Thrombocytopenia	Accelerated platelet consumption and destruction, particularly in severe preeclampsia and HELLP syndrome.	Indicates disease severity; guides monitoring and transfusion strategies.
Altered Coagulation Cascade	Dysregulation of intrinsic and extrinsic pathways; prolonged PT and aPTT in advanced cases.	Risk of disseminated intravascular coagulation (DIC); informs clinical decision-making.
Endothelial Dysfunction and Microthrombosis	Endothelial activation increases tissue factor expression, microparticle release, and adhesion molecule upregulation.	Leads to multi-organ involvement (kidney, liver, brain) and correlates with maternal morbidity.
Imbalance in Fibrinolysis	Altered plasminogen activation and increased D-dimer levels.	Reflects ongoing coagulation and fibrinolysis; assists in risk stratification and prognosis.
Tumor/Placental Microvascular Injury	Microthrombosis in uteroplacental circulation due to coagulation abnormalities.	Contributes to fetal compromise, intrauterine growth restriction, and preterm birth.

Endothelial Dysfunction and Coagulation Activation

Damaged endothelial cells lose their antithrombotic properties and express higher levels of tissue factor, triggering the extrinsic coagulation pathway. This leads to the generation of thrombin, a potent platelet activator, which further amplifies coagulation through positive feedback loops. Concurrently, von Willebrand factor release from injured endothelium promotes platelet adhesion and aggregation, fostering microthrombus formation within the placental and systemic microvasculature [18-20].

Platelet Consumption and Microangiopathy

Sustained platelet activation results in accelerated consumption, reflected as progressive

thrombocytopenia in laboratory assessments. Morphological changes in platelets, such as increased mean platelet volume (MPV), signal heightened reactivity. The formation of widespread microthrombi contributes to microangiopathic hemolysis, a feature shared with HELLP syndrome and thrombotic microangiopathies, further exacerbating tissue ischemia [21-22].

Altered Fibrin Turnover and Fibrinolysis Suppression

Preeclampsia is marked by excessive fibrin deposition in small vessels, driven by elevated fibrinogen levels early in the disease course. Paradoxically, as disease severity increases, fibrinogen levels may decline due to consumptive coagulopathy. The fibrinolytic system is simultaneously suppressed by elevated levels of plasminogen activator inhibitor-1 (PAI-1), which impairs clot breakdown and promotes a prothrombotic state. D-dimer, a degradation product of cross-linked fibrin, becomes disproportionately elevated, reflecting both increased clot formation and attempted fibrinolysis [23].

Disseminated Intravascular Coagulation in Severe Cases

In its most severe form, preeclampsia can progress to overt disseminated intravascular coagulation (DIC), characterized by uncontrolled thrombin generation, widespread fibrin deposition, and secondary hyperfibrinolysis. Clinically, this manifests as bleeding, oozing from puncture sites, and multi-organ failure. DIC in preeclampsia is often precipitated by catastrophic complications such as placental abruption or intrauterine fetal demise [24].

Integration with Clinical Outcomes

These pathophysiological processes underpin the high incidence of maternal complications—including stroke, hepatic rupture, and acute kidney injury—as well as fetal complications such as intrauterine growth restriction and stillbirth. Understanding these mechanisms reinforces the rationale for routine coagulation assessment in preeclampsia, not merely as a supportive test but as a core component of monitoring disease trajectory and anticipating complications [25-26].

Diagnostic Relevance of Coagulation Markers

Coagulation abnormalities in preeclampsia provide valuable diagnostic insight, complementing the traditional clinical criteria of hypertension and proteinuria. While these hemostatic changes have historically been considered secondary findings, accumulating evidence supports their integration into early disease detection and severity assessment.

Platelet Count and Indices

Thrombocytopenia is the most consistent hematologic abnormality in preeclampsia and can occur before overt clinical deterioration. A platelet count below 150×10^9/L raises suspicion for disease progression, while a rapid decline, even within the normal range, may signal impending HELLP syndrome or disseminated intravascular coagulation (DIC). Platelet indices such as mean platelet volume (MPV) and platelet distribution width (PDW) offer additional clues, with elevated values suggesting heightened platelet activation and turnover [27].

Fibrinogen Levels

In healthy pregnancy, fibrinogen concentrations rise significantly as part of the physiologic hypercoagulable state. In preeclampsia, fibrinogen may initially be elevated but declines with increasing disease severity, reflecting consumption in the formation of widespread microthrombi. A precipitous drop in fibrinogen should prompt urgent evaluation for consumptive coagulopathy or placental abruption [28].

D-dimer

D-dimer, a fibrin degradation product, naturally increases during normal pregnancy but rises

disproportionately in preeclampsia. This exaggerated elevation indicates excessive fibrin formation and breakdown due to widespread microvascular thrombosis. While elevated D-dimer alone is not specific for preeclampsia, its use in combination with other parameters enhances diagnostic precision and helps differentiate severe from non-severe disease forms [29].

Prothrombin Time (PT) and Activated Partial Thromboplastin Time (aPTT)

Prolongation of PT and aPTT is uncommon in early preeclampsia but, when present, usually indicates advanced disease or DIC. These parameters are particularly useful for confirming coagulopathy in patients with clinical bleeding or abnormal platelet and fibrinogen levels [30].

Emerging Biomarkers

Novel coagulation-related biomarkers, including thrombin–antithrombin complexes, soluble thrombomodulin, and tissue factor pathway inhibitor, are under investigation for their potential to detect subclinical coagulation activation. Incorporating these into future diagnostic algorithms could allow even earlier detection of at-risk pregnancies [31].

Integrated Diagnostic Utility

When interpreted collectively, coagulation markers can identify early pathological changes, stratify disease severity, and inform the timing of delivery. Their diagnostic role extends beyond confirming suspected cases—they offer a dynamic picture of disease progression, which is particularly valuable in resource-limited settings where access to advanced imaging or continuous monitoring is limited [32].

Prognostic Implications

Coagulation abnormalities in preeclampsia are not merely laboratory curiosities—they carry substantial prognostic significance for both maternal and fetal outcomes. The dynamic trends of these parameters often provide more predictive value than isolated measurements, enabling clinicians to anticipate complications and tailor management strategies [33].

Maternal Prognosis

Progressive thrombocytopenia is one of the most reliable indicators of disease worsening and is closely associated with HELLP syndrome, disseminated intravascular coagulation (DIC), and severe postpartum hemorrhage. A rapid decline in platelet count, even from within the normal range, signals heightened coagulation activation and microangiopathy. Similarly, falling fibrinogen levels, especially below 3 g/L, suggest consumptive coagulopathy and increased hemorrhagic risk during delivery. Disproportionately elevated D-dimer levels may reflect widespread microvascular thrombosis, correlating with complications such as stroke, acute kidney injury, and hepatic rupture [34-35].

Fetal Prognosis

From the fetal perspective, coagulation derangements mirror placental vascular pathology. Elevated D-dimer and reduced fibrinogen levels often parallel impaired uteroplacental perfusion, which contributes to intrauterine growth restriction (IUGR), oligohydramnios, and stillbirth. Platelet and fibrinogen trends may serve as indirect markers of placental function, helping guide decisions on timing of delivery to minimize hypoxic injury [36-37].

Risk Stratification and Clinical Decision-Making

Serial measurement of coagulation markers enhances risk stratification. For instance, a patient with stable blood pressure but progressively worsening coagulation parameters may require expedited delivery, even in the absence of overt clinical deterioration. Conversely, stable coagulation profiles can support conservative management in well-controlled cases, potentially

prolonging gestation and improving neonatal outcomes [38].

Predicting Postpartum Complications

The prognostic utility of coagulation markers extends into the postpartum period, where women with persistently abnormal profiles remain at elevated risk of thromboembolic events and delayed hemorrhage. Monitoring these markers after delivery can facilitate early detection of ongoing coagulopathy and prompt initiation of anticoagulation or transfusion support when indicated [39].

Research and Prognostic Models

Emerging studies suggest that integrating coagulation markers into multivariable prognostic models—alongside blood pressure trends, renal function, and angiogenic markers—may yield more accurate predictions of adverse outcomes than traditional clinical criteria alone. Such models could transform risk-based triage, especially in high-burden, low-resource regions [40].

Translational Perspective

Ultimately, the prognostic value of coagulation parameters underscores their role as dynamic biomarkers that reflect the evolving pathophysiology of preeclampsia. Their integration into standard monitoring protocols has the potential to shift the focus from reactive to proactive management, thereby improving survival and reducing morbidity for both mother and child [41-43].

Clinical Integration and Future Directions

The recognition of coagulation abnormalities as both diagnostic and prognostic tools in preeclampsia opens the door for a more comprehensive, biomarker-driven approach to management. However, translating this understanding into routine clinical practice requires strategic integration into existing antenatal care protocols [44].

Incorporating Coagulation Testing into Standard Care

Currently, coagulation testing in preeclampsia is often reserved for cases with severe disease or suspected HELLP syndrome. A more proactive strategy would involve baseline coagulation profiling at diagnosis, followed by serial measurements in moderate to high-risk patients. Key parameters—platelet count, fibrinogen, D-dimer, and prothrombin time—could be prioritized for their accessibility, cost-effectiveness, and proven clinical relevance. In resource-rich settings, expanded testing for advanced biomarkers such as thrombin–antithrombin complexes or soluble thrombomodulin could further refine early detection [45-47].

Point-of-Care Testing (POCT) for Wider Access

The development and deployment of point-of-care devices for rapid coagulation assessment could revolutionize preeclampsia management in resource-limited settings. Portable systems capable of measuring platelet count, PT, and D-dimer within minutes would enable timely decision-making in rural clinics and emergency obstetric units [48-50].

Integration into Risk Stratification Models

Combining coagulation parameters with clinical indicators, angiogenic biomarkers, and ultrasound findings could yield robust risk stratification models. Such tools would allow obstetric teams to personalize surveillance intensity, optimize timing of delivery, and allocate resources effectively. Integration into electronic medical record (EMR) systems could facilitate automated alerts when critical laboratory thresholds are crossed [51-52].

Therapeutic Implications and Trials

The consistent association between coagulation abnormalities and poor outcomes raises the question of whether targeted interventions—such as low-dose heparin or antiplatelet agents—could

modify disease progression in select patients. Carefully designed clinical trials are needed to evaluate the safety and efficacy of such therapies, particularly in the context of balancing hemorrhagic and thrombotic risks during pregnancy [53].

Postpartum Surveillance

Given that hypercoagulability can persist beyond delivery, postpartum coagulation monitoring may identify women at elevated risk for venous thromboembolism or delayed hemorrhage. This extended surveillance could be particularly important for those with severe antepartum coagulopathy or persistent hypertension [54].

Research Priorities

Future research should focus on defining evidence-based cutoff values for key coagulation markers in pregnancy, validating composite scoring systems, and assessing the cost-effectiveness of routine coagulation monitoring in diverse healthcare settings. Longitudinal studies exploring the interplay between coagulation profiles and maternal–fetal outcomes will be crucial for refining management algorithms [55].

Towards a Precision Obstetrics Approach

By embedding coagulation monitoring into a broader precision obstetrics framework, clinicians can shift from reactive to anticipatory care. Such an approach not only addresses the immediate risks of preeclampsia but also contributes to long-term cardiovascular and hematologic health for mothers, aligning with the goals of sustainable maternal care systems worldwide [55].

Conclusion

Coagulation abnormalities in preeclampsia are more than secondary laboratory findings—they are central to the disease's pathophysiology, clinical presentation, and outcomes. Changes in platelet count, fibrinogen levels, D-dimer, and coagulation times provide critical diagnostic clues and serve as dynamic predictors of maternal and fetal risk. Integrating these parameters into routine antenatal assessment can enhance early detection, refine risk stratification, and guide timely intervention, ultimately improving survival and reducing morbidity. Future strategies should focus on standardizing monitoring protocols, developing point-of-care testing solutions, and exploring targeted therapeutic interventions within well-designed clinical trials. Recognizing coagulation profiles as essential components of preeclampsia management moves obstetric care toward a more proactive, precision-based model, offering tangible benefits for both mothers and their infants.

Conflicts of Interest

The author declares no conflict of interest

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Abbreviations

aPTT – Activated Partial Thromboplastin Time
DIC – Disseminated Intravascular Coagulation
HELLP – Hemolysis, Elevated Liver Enzymes, Low Platelets
PlGF – Placental Growth Factor
PT – Prothrombin Time
sFlt-1 – Soluble Fms-Like Tyrosine Kinase-1
TEG – Thromboelastography

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