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Hematological Aspects of Pre-eclampsia and Its Complications in West Africa

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Abstract

Pre-eclampsia (PE) and eclampsia remain important causes of maternal and perinatal morbidity in West Africa, but the regional evidence on their hematological features is fragmented. This structured narrative review searched PubMed/MEDLINE, Google Scholar, African Journals Online, medical theses, and the digital repositories of Université Cheikh Anta Diop and Université Assane Seck de Ziguinchor through 20 July 2026. West African original studies were prioritized; international guidelines and high-quality reviews were used for definitions and contextual comparison. Available regional studies are mostly small, single-center, cross-sectional or retrospective. They suggest that thrombocytopenia, anemia, and coagulation abnormalities are more frequent in severe or complicated disease, but reported associations with platelet indices, inflammatory ratios, antiphospholipid antibodies, and inherited thrombophilia are inconsistent. A full blood count with platelet count is central to assessment. Tests for hemolysis and coagulation should be guided by severity, suspected hemolysis, bleeding, placental abruption, or disseminated intravascular coagulation. Current evidence does not support routine D-dimer, platelet-index, inflammatory-ratio, antiphospholipid-antibody or inherited-thrombophilia panels for every patient with PE. Multicenter prospective studies using standard definitions and clinically relevant outcomes are required to identify markers that improve care in West African settings.

Keywords: Pre-eclampsia, Eclampsia, Hematological aspects, Thrombocytopenia, Hemostasis, Antiphospholipid antibodies, Thrombophilia, HELLP syndrome, Maternal–fetal complications, West Africa

Introduction

Pre-eclampsia (PE) is a multisystem pregnancy disorder, and eclampsia is one of its most severe neurological complications. Hypertensive disorders of pregnancy continue to contribute substantially to maternal and perinatal morbidity, particularly where antenatal detection, referral, blood products and critical care are constrained [1–7]. West African reports describe a persistent burden of severe PE, eclampsia, placental abruption and hemolysis, elevated liver enzymes, and low platelet count (HELLP) syndrome, but estimates vary by setting and study design.

Hematological abnormalities are clinically relevant because endothelial injury can activate platelets and coagulation, while severe disease may cause thrombocytopenia, microangiopathic hemolysis and consumption of coagulation factors [8–20]. Nevertheless, a plausible mechanism does not by itself establish the diagnostic or prognostic value of a laboratory marker. Regional studies are heterogeneous, often observational, and sometimes examine related obstetric conditions rather than PE itself.

This review therefore aims to synthesize the hematological evidence on PE and its complications in West Africa, distinguish regional findings from international evidence, appraise the main methodological limitations, and propose a pragmatic laboratory approach for settings with variable resources. The review also identifies priorities for research on antiphospholipid antibodies and inherited thrombophilia.

Methods

This article is a structured narrative review rather than a systematic review. PubMed/MEDLINE, Google Scholar, African Journals Online, medical-thesis sources, and the digital repositories of Université Cheikh Anta Diop and Université Assane Seck de Ziguinchor were searched from database inception to 20 July 2026. The focused hematology synthesis prioritized publications from 2008 onwards, while earlier landmark West African reports and international mechanistic or diagnostic sources were retained when needed.

Search terms combined "pre-eclampsia" or "eclampsia" with "West Africa" or individual country names and the terms "hematology", "platelet", "thrombocytopenia", "coagulation", "hemolysis", "HELLP", "antiphospholipid", "factor V Leiden", "prothrombin G20210A", "protein C", "protein S", "sickle cell disease" and "pregnancy loss". English- and French-language full texts were considered.

Original studies were included when they reported West African data on PE, eclampsia, relevant obstetric complications, or hematological markers. Regional systematic reviews, guidelines and international high-quality reviews were used for context when direct West African evidence was absent. Duplicate reports, studies without extractable outcomes and publications unrelated to pregnancy hypertension or their hematological differential diagnoses were excluded. Country, year, design, sample size, principal findings, and major limitations were extracted. Because populations, definitions, assays and outcomes were too heterogeneous for valid pooling, findings were synthesized narratively and interpreted according to design, sample size, presence of a comparator, consistency, and directness to West African PE.

Generalities on Pre-eclampsia and Its Complications

Definitions

Pre-eclampsia

Current International Society for the Study of Hypertension in Pregnancy (ISSHP) recommendations define PE as new-onset hypertension (systolic blood pressure ≥140 mmHg and/or diastolic blood pressure ≥90 mmHg) after 20 weeks of gestation together with proteinuria, maternal organ dysfunction or uteroplacental dysfunction. Proteinuria is therefore not mandatory when other qualifying features are present, and the amount of proteinuria alone does not define severity [18,21,22]. Severe hypertension (≥160/110 mmHg) requires urgent confirmation and management.

Eclampsia

Eclampsia is the occurrence of generalized seizures in a woman with PE when no alternative neurological cause explains them. It may occur antepartum, intrapartum or postpartum [21,23–27].

Complications of pre-eclampsia

Complications reflect progressive maternal organ or uteroplacental dysfunction. The most clinically relevant events are summarized in **Table 1** [28–41].

Table 1. Complications of pre-eclampsia.

Maternal	Fetal and neonatal
Eclampsia; HELLP syndrome; placental abruption; disseminated intravascular coagulation; stroke; pulmonary oedema; acute kidney injury; hepatic hematoma or rupture; maternal death.	Fetal growth restriction; prematurity; low birth weight; fetal compromise; stillbirth; neonatal respiratory and hematological morbidity; perinatal death.

Abbreviations: HELLP: Hemolysis, Elevated Liver Enzymes and Low Platelet Count

Etiology

PE is multifactorial. Established associations include nulliparity, previous or family history of PE, multifetal pregnancy, chronic hypertension, chronic kidney disease, diabetes, obesity, autoimmune disease including antiphospholipid syndrome, and conception using assisted reproductive technology [1,21,22,42–44]. Sickle cell disease is associated with adverse pregnancy outcomes and may increase PE risk, but effect estimates are not uniform across genotypes and settings [45–49]. Biological susceptibility is shared across populations; differences in observed consequences also reflect case ascertainment, access to care and referral delay.

Pathophysiology

The prevailing model links abnormal placentation to placental hypoperfusion, oxidative and inflammatory stress, anti-angiogenic signaling and systemic endothelial dysfunction. This framework explains hypertension, organ dysfunction and activation of platelets and coagulation, but individual pathways vary between early- and late-onset disease [8–16].

Defective trophoblastic invasion

Incomplete remodeling of spiral arteries preserves a high-resistance uteroplacental circulation. Resulting placental stress promotes the release of mediators that affect the maternal vasculature [8,9,13–15].

Table 2. Comparison between developed and African countries regarding the etiology and consequences of pre-eclampsia.

Aspect	Higher-resource settings	Sub-Saharan African settings
Underlying risk factors	Maternal, placental, metabolic, vascular and genetic risk factors.	The same biological risk factors apply; the distribution of chronic hypertension, metabolic disease, haemoglobinopathies and access to prevention varies by setting.
Case detection	Earlier antenatal detection and broader access to laboratory and imaging assessment.	Late booking, incomplete surveillance and referral barriers can increase the proportion of severe presentations.
Consequences	Fetal growth restriction, indicated preterm birth, HELLP syndrome, abruption and later maternal cardiovascular risk.	The same complication spectrum, often with higher case fatality where emergency care, blood products or neonatal support are limited.
Evidence base	Larger cohorts, registries and validated prediction studies are more available.	Predominantly hospital-based observational studies with heterogeneous definitions and limited multicenter validation.

This comparison describes evidence and health-system context; it should not be interpreted as a biological dichotomy between populations. Abbreviation: HELLP, hemolysis, elevated liver enzymes and low platelet count.

Tissue hypoxia and oxidative stress

Intermittent placental perfusion can generate oxidative stress and amplify maternal inflammation. These mechanisms have been associated with endothelial injury and hemostatic activation, although circulating markers are not yet validated for routine diagnosis in West Africa [10–13,50,51].

Endothelial dysfunction

Systemic endothelial dysfunction alters vascular tone and exposes procoagulant surfaces. Platelet activation and thrombin generation may be accompanied by enhanced consumption, fibrinolytic imbalance and, in severe disease, microangiopathic hemolysis or disseminated intravascular coagulation [10,13,14,19]. The sequence is summarized in **Figure 1**.

Clinical Signs

PE may be asymptomatic and detected during blood-pressure or laboratory surveillance. Warning features include severe hypertension, persistent severe headache, visual symptoms, epigastric or right-upper-quadrant pain, dyspnea, reduced

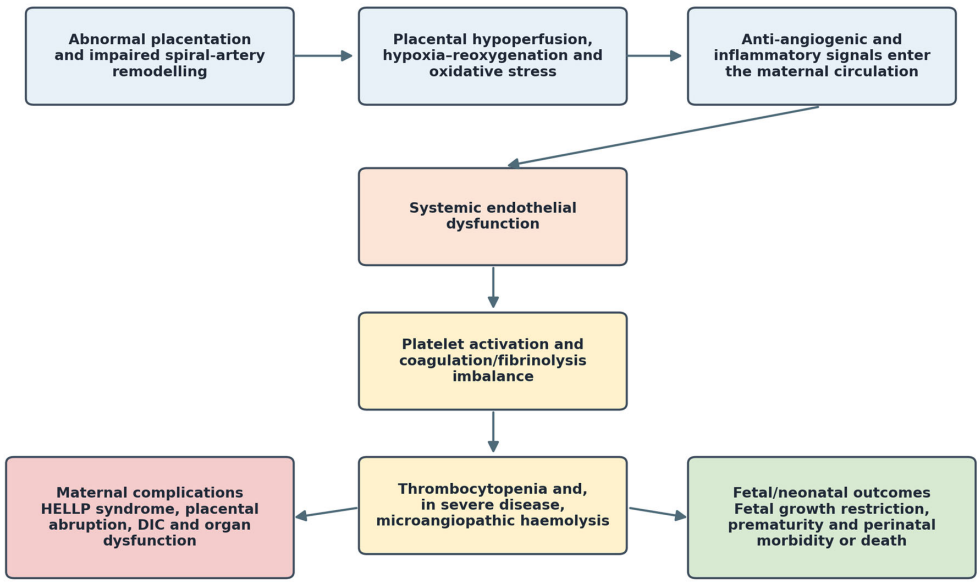


Figure 1. Proposed link between placental dysfunction, endothelial injury, hemostatic abnormalities and maternal–fetal complications. The arrows represent a conceptual pathway and not a validated diagnostic algorithm. Abbreviations: DIC: Disseminated Intravascular Coagulation; HELLP: Hemolysis, Elevated Liver Enzymes and Low Platelet Count.

urine output and signs of fetal compromise. Seizures suggest eclampsia after alternative causes have been considered [17,18,21,22,52]. Clinical severity should be assessed together with maternal laboratory and fetal findings.

Biological Signs

Laboratory assessment evaluates proteinuria and maternal organ involvement. A full blood count (FBC) with platelet count, serum creatinine and liver enzymes are central. Where hemolysis is suspected, lactate dehydrogenase (LDH), bilirubin, haptoglobin where available and a peripheral blood film are useful. Prothrombin time (PT), international normalized ratio, activated partial thromboplastin time (aPTT) and fibrinogen are particularly relevant when bleeding, placental abruption, severe hepatic dysfunction or disseminated intravascular coagulation is suspected [19–22]. Neither proteinuria ≥5 g/24 h nor an isolated D-dimer value should be used as a stand-alone criterion of severe PE.

Consequences

Consequences for the mother

Maternal complications arise from severe hypertension, endothelial injury, microangiopathy, ischemia and hemostatic consumption. Their frequency in West African series is influenced by referral pathways and access to timely obstetric and critical care [28–37].

HELLP syndrome

HELLP syndrome combines hemolysis, elevated liver enzymes and a low platelet count. Diagnostic thresholds vary, but a platelet count below 100×10^9/L is commonly used for complete HELLP syndrome. Trends in platelets, LDH, bilirubin, aminotransferases, and the blood film are more informative than a single isolated value [33,53,54].

Subcapsular liver hematoma

This rare complication should be considered when severe right-upper-quadrant or epigastric pain accompanies PE or HELLP syndrome. Urgent imaging and multidisciplinary management are required [33,34].

Subcapsular liver hematoma

Capsular rupture is an obstetric emergency associated with intra-abdominal bleeding and hemodynamic instability. It may require massive-transfusion support and surgical or interventional-radiology expertise [33,34].

Hepatic infarction

Hepatic infarction is uncommon and is usually reported with severe PE or HELLP syndrome. Diagnosis relies on compatible symptoms, liver-test abnormalities and imaging [33,34].

Acute kidney injury

Acute kidney injury may accompany severe PE, placental abruption, HELLP syndrome, hemorrhage or pre-existing renal disease. Serial creatinine and urine-output assessment are more useful than proteinuria quantity alone [20,21,35].

Pulmonary edema

Pulmonary edema reflects endothelial permeability, altered oncotic pressure, cardiac dysfunction and sometimes excessive fluid administration. It is a severe feature requiring urgent management [21,33,36].

Abruptio placentae (retroplacental hematoma)

Placental abruption can cause substantial thrombin generation, falling fibrinogen, thrombocytopenia and disseminated intravascular coagulation. Senegalese reports document its prognostic importance, but detailed serial coagulation data remain limited [37,38,55–57].

Consequences for the fetus

Acute fetal distress

Uteroplacental insufficiency may produce acute or chronic fetal compromise. The consequences include fetal growth restriction, indicated preterm birth and fetal death [16,39–41].

Prematurity

Prematurity is birth before 37 completed weeks. In PE, it may follow spontaneous disease or medically indicated delivery for maternal or fetal safety [39–41,55,58–60].

Intrauterine growth restriction (IUGR)

The contemporary term fetal growth restriction (FGR) describes failure to reach growth potential. In PE it usually reflects placental insufficiency and is associated with prematurity, low birth weight and perinatal morbidity [40,41].

Intrauterine fetal death (IUFD)

Severe placental insufficiency, abruption or acute maternal deterioration may lead to fetal death. Definitions vary by gestational-age and birth-weight threshold, which complicates comparison between studies [40,41,58,61].

Consequences for the newborn

Most neonatal consequences are mediated by placental insufficiency, growth restriction and prematurity rather than by PE-specific congenital anomalies [39–41,58].

Table 3. Consequences of pre-eclampsia in the newborn.

Domain	Commonly reported consequences
Growth, respiratory and metabolic	Fetal growth restriction, low birth weight, prematurity, respiratory morbidity, hypoglycemia and temperature instability.
Hematological, neurological and survival	Thrombocytopenia, neutropenia or anemia may occur; severe prematurity or fetal compromise can require neonatal care and is associated with perinatal mortality.

Long-term consequences

A history of PE is associated with later chronic hypertension, cardiovascular disease, stroke, kidney disease and metabolic risk. Postpartum counselling and long-term primary-care follow-up are therefore important [21,22,61,62]. Children born after pregnancies complicated by PE may also have consequences related to prematurity or growth restriction, but regional longitudinal data are sparse.

State of Knowledge in West Africa on Pre-eclampsia

Socio-demographic profiles

West African studies frequently report primigravidity, limited antenatal booking and extremes of maternal age among women presenting with PE or eclampsia [30,43,55,56,63–65]. Age findings are not uniform: some hospital-based reports emphasize younger women, whereas the 15-year Maiduguri series reported that 55.2% of eclampsia cases were older than 35 years [65]. A sub-Saharan African meta-analysis found associations with primigravidity and several clinical risk factors, but pooled estimates were heterogeneous [42]. These patterns should not be interpreted causally because referral, parity distribution and case definitions differ across studies.

Main etiologies

Regional studies identify the same broad risk domains reported internationally: chronic hypertension, kidney disease, diabetes, obesity, sickle cell disease, multifetal pregnancy, and previous or family history of PE [42–44,65–73]. However, most data are observational and support association rather than individual causal attribution.

Sickle cell disease and pre-eclampsia

Sickle cell disease is prevalent in parts of West Africa and pregnancy is high risk. International and African syntheses have associated sickle cell disease—particularly severe genotypes—with PE, anemia, thrombosis, preterm birth, and fetal growth restriction [45–49]. The multinational meta-analysis included 21 studies and a very large comparator population, but most women were enrolled in high-income settings; its effect estimates therefore require local validation. A Ghanaian prospective study of 150 women with PE linked hemoglobin variants and oxidative-stress measures with outcomes but lacked a normotensive comparator [51].

Metabolic disorders and pre-eclampsia

Obesity and pre-existing or gestational diabetes have been associated with PE through vascular, inflammatory and metabolic pathways [59,67–69]. African meta-analyses show substantial variation in gestational-diabetes prevalence and diagnostic criteria. Consequently, regional associations should be interpreted with attention to screening practices and confounding rather than presented as proof of a single mechanism.

Hypertension and pre-eclampsia

Chronic hypertension is an established risk factor for superimposed PE and adverse pregnancy outcome [43,44,70,71]. It must be distinguished from the new-onset hypertension that forms part of the PE definition. Studies that describe "hypertension" without separating pre-existing disease from the diagnostic blood-pressure rise of PE risk circular interpretation.

Idiopathic pre-eclampsia

Because PE is a syndrome with multiple pathways, the absence of a recognized risk factor does not establish a distinct idiopathic subtype. Regional studies rarely apply biomarker-defined phenotypes, and unmeasured chronic hypertension, kidney disease or metabolic risk may remain. Future work should use standard early- versus late-onset and maternal–placental phenotypes [21,44,71–73].

Multiple pregnancies and the incidence of pre-eclampsia

Multifetal pregnancy is associated with a higher PE risk, probably in part because of greater placental mass. The association appears in regional and international studies, although West African estimates are imprecise and may be influenced by referral patterns [21,42,63].

Family history of pre-eclampsia

A family or personal history of PE has been associated with recurrence or future disease [42,44,65,72]. This supports a familial component but does not yet identify a clinically useful genetic screening panel for West African populations.

Spontaneous and/ or recurrent miscarriages and pre-eclampsia

Recurrent pregnancy loss and severe placenta-mediated complications may raise clinical suspicion of antiphospholipid syndrome (APS). In Northern Nigeria, Abdullahi et al. found antiphospholipid antibodies (aPL) in 14.1% of 85 women with recurrent fetal loss and 4.7% of 85 controls [74]. This was not a PE cohort, and the finding should not be extrapolated to universal aPL screening in PE. International evidence supports targeted evaluation based on clinical history [75,76].

Hematological Aspects in West Africa (2008–2025)

The most reproducible hematological abnormality in severe PE is a falling platelet count. Anemia may coexist because of nutritional deficiency, hemoglobinopathy, hemorrhage or microangiopathic hemolysis; its mechanism must therefore be established rather than attributed automatically to PE. Nigerian studies have reported differences in platelet count or indices, but results are not consistent across cohorts [50,77,78]. A 2023 international meta-analysis confirmed lower platelet counts in PE on average, while also showing heterogeneity that limits the use of a single threshold as a diagnostic test [79].

HELLP syndrome

Direct West African data describing complete serial hematological profiles in HELLP syndrome are scarce. The international syndrome definition and management literature supports assessment of platelets, blood-film evidence of microangiopathy, LDH, bilirubin, and aminotransferases [33,53,54,80]. A low platelet count alone is not specific, and alternative diagnoses such as gestational thrombocytopenia, immune thrombocytopenia, thrombotic microangiopathy, infection and acute fatty liver of pregnancy may require consideration.

Abruptio placentae (retroplacental hematoma)

Senegalese studies document the clinical burden of placental abruption but provide limited serial information on fibrinogen and D-dimer [37,55,56]. In suspected abruption, platelet count, PT, aPTT, and fibrinogen should be interpreted together with bleeding, hemodynamic status, and trends. Normally early result does not exclude evolving consumption.

Coagulation profiles

Physiological pregnancy is hypercoagulable, while uncomplicated PE may have normal routine coagulation times. Nigerian data from Ekun et al. showed longer PT and aPTT with lower platelet counts in 49 women with PE than in 50 controls [50], but the small cross-sectional design and possible differences in disease severity limit generalization. Routine coagulation screening in every uncomplicated case has uncertain yield; testing is most valuable when disease is severe, bleeding is present, abruption or liver dysfunction is suspected, or an invasive procedure is planned [13,19–22].

The place of antiphospholipid antibodies (aPL) in the occurrence of pre-eclampsia and its complications in West Africa

The laboratory aPL panel comprises lupus anticoagulant, anticardiolipin antibodies, and anti-β2-glycoprotein I antibodies. APS requires compatible clinical features and persistent laboratory positivity; the 2023 American College of Rheumatology/European Alliance of Associations for Rheumatology (ACR/EULAR) criteria are classification criteria and should not replace clinical judgment [76]. Direct West African PE evidence is very limited. The Nigerian recurrent fetal-loss study [74] and international meta-analysis of placenta-mediated complications [75] support further study but not testing every woman with PE. Testing is more defensible when the history suggests APS, such as thrombosis, recurrent pregnancy morbidity or selected severe early placenta-mediated complications, with specialist interpretation and repeat confirmation when required.

Thrombotic disease and pre-eclampsia in West Africa

Pregnancy, PE and immobility can increase venous thromboembolism risk, but this does not establish inherited thrombophilia as a cause of PE [12,14,81–84]. In Nigeria, Okoye et al. found no association between protein C or protein S deficiency and PE in a cross-sectional comparison of 90 cases and 90 controls [85]. In Ghana, Ababio et al. reported an association between factor V Leiden and PE in 81 women [86], but the small exploratory sample and absence of independent regional replication preclude routine population screening. Factor V Leiden, prothrombin G20210A, protein C, protein S and antithrombin testing should be reserved for clinically selected patients after consideration of timing, anticoagulation, and pregnancy-related changes.

Critical synthesis of regional evidence

The studies summarized in **Table 4** do not support a single regional hematological signature. Adeyemo et al. and Ekun et al. reported platelet or coagulation differences [50,77], whereas Nwagha et al. found no significant difference in platelet count or indices and inadequate specificity for inflammatory ratios [78]. Okoye et al. did not confirm an association with protein C or protein S deficiency [85], and the factor V Leiden signal reported by Ababio et al. remains unreplicated [86]. The aPL study by Abdullahi et al. addressed recurrent fetal loss rather than PE [74]. These inconsistencies likely reflect small samples,

Table 4. Selected West African studies and contextual comparative syntheses on pre-eclampsia, eclampsia and hematological markers.

Location/year	First author [ref.]	Design and sample	Main findings	Key limitations/interpretation
Senegal, 2021	Coly MN [56]	Mixed retrospective/ prospective hospital study; n=74	Exploratory associations between ABO group and eclampsia or placental abruption; anemia and thrombocytopenia were not associated with ABO group.	Small single-center sample; mixed design; multiple comparisons; limited adjustment.
Nigeria, 2022	Adeyemo MA [77]	Case–control; n=80 (40 PE, 40 controls)	Platelet count/indices and erythrocyte sedimentation rate differed between groups; authors proposed usefulness in assessment.	Small cohort; single center; cross-sectional sampling; diagnostic performance not validated.
Nigeria, 2016	Abdullahi ZG [74]	Analytical cross-sectional; n=170 (85 recurrent fetal loss, 85 controls)	aPL prevalence was 14.1% in recurrent fetal loss and 4.7% in controls.	Not a PE cohort; older testing framework; persistence and PE-specific value not established.
Nigeria, 2023	Geidam AD [65]	15-year retrospective review; n=761 eclampsia cases	Primigravidity and lack of antenatal booking were common; substantial maternal and perinatal morbidity.	Retrospective records; referral bias; limited detailed hematological-marker data.
Nigeria, 2017	Okoye HC [85]	Cross-sectional comparison; n=180 (90 PE, 90 controls)	Protein C measures did not differ materially; free protein S was higher in PE; deficiencies were not associated with PE.	Single time point; physiological pregnancy changes affect protein S; no causal inference.
Nigeria, 2018	Ekun OA [50]	Comparative cross-sectional; n=99 (49 PE, 50 controls)	Lower platelets and antioxidant markers and longer PT/aPTT were reported in PE.	Small sample; severity mix and confounding; assays not linked to prospective outcomes.
Ghana, 2019	Ababio GK [86]	Prospective follow-up; n=81	Factor V Leiden polymorphism was reported in association with PE and selected outcomes.	Exploratory small sample; high reported allele frequency requires independent genotyping replication.
Ghana, 2023	Abdul G [51]	Multicenter prospective study; n=150 women with PE	Hemoglobin variants and oxidative-stress biomarkers were associated with selected adverse outcomes.	No normotensive comparator; purposive enrolment; associations need external validation.
Nigeria, 2025	Nwagha TU [78]	Comparative cross-sectional; n=90 (45 PE, 45 controls)	RDW and NLR were higher and PLR lower in PE; platelet count/indices did not differ. NLR AUC was 0.82 but specificity was 22.2%.	Small sample; cross-sectional; low specificity; authors did not support stand-alone diagnostic use.
Sub-Saharan Africa, 2020 (context)	Meazaw MW [42]	Systematic review/meta-analysis; 51 studies, 25,789 women	Synthesized demographic, obstetric and medical factors associated with PE/eclampsia.	Mostly observational; high heterogeneity; English-only; only 16 studies entered meta-analysis.
Multinational, 2016 (context)	Boafor T [49]	Systematic review/meta-analysis; 21 studies, 26,349 women with sickle cell disease	Sickle cell disease was associated with increased PE and other adverse pregnancy outcomes.	Most participants came from high-income settings; genotype and study-quality heterogeneity limit directness.

Abbreviations: ABO: ABO Blood-group System; aPL: Antiphospholipid Antibodies; aPTT: Activated Partial Thromboplastin Time; AUC: Area Under the Receiver-operating Characteristic Curve; NLR: Neutrophil-to-Lymphocyte Ratio; PE: Pre-Eclampsia; PLR: Platelet-to-Lymphocyte Ratio; PT: Prothrombin Time; RDW: Red-Cell Distribution Width

different phenotypes and gestational ages, single time-point measurements, assay variation and incomplete control of confounding.

Accordingly, platelet count and tests directed at hemolysis or consumptive coagulopathy have clear clinical roles, while platelet indices, inflammatory ratios, D-dimer, aPL and inherited-thrombophilia panels remain context-specific or investigational for PE risk stratification. No included West African study demonstrated that routine use of an expanded biomarker panel improves maternal or neonatal outcomes.

Practical laboratory guidance in West African settings

Laboratory requests should answer a clinical question, use serial trends when disease is evolving, and be adapted to local turnaround time. **Table 5** separates core assessment from targeted or research tests [21,22,76,78,79].

Conclusion

PE and its complications remain major maternal and perinatal problems in West Africa. Available evidence suggests that platelet count, evidence of hemolysis and coagulation abnormalities are most clinically relevant when disease is severe or complicated. However, the regional literature is dominated by small observational studies and does not establish routine diagnostic or prognostic value for platelet indices, inflammatory ratios, D-dimer, aPL or inherited-thrombophilia panels in all women with PE. A pragmatic approach should prioritize an FBC with platelet count and organ-function tests, then add hemolysis and coagulation studies according to the clinical presentation. Expanded testing should be targeted and interpreted with obstetric and hematology expertise.

Study limitations

This is a narrative review and does not claim the exhaustiveness of a PRISMA-conformant systematic review. Scopus and Web of Science were not searched, and grey-literature retrieval may be incomplete. No formal risk-of-bias instrument or quantitative meta-analysis was applied. Regional studies used heterogeneous PE definitions, laboratory methods, sampling times and outcomes; many were small, single-center and cross-sectional or retrospective. Some contextual evidence came from sub-Saharan African or international studies and may not be directly transferable to West Africa. These limitations reduce certainty and require cautious interpretation.

Table 5. Pragmatic use of hematological tests in pre-eclampsia.

Clinical situation	Suggested tests	Established or plausible value	Important cautions
All women with suspected or diagnosed PE	FBC with platelet count; creatinine; AST/ALT; proteinuria assessment.	Detects thrombocytopenia and maternal organ dysfunction; establish a baseline for trends.	Normal single results do not exclude deterioration. Interpret anemia by cause.
Suspected HELLP syndrome or hemolysis	Repeat FBC/platelets; blood film; LDH; bilirubin; haptoglobin where available; AST/ALT.	Assesses platelet consumption and microangiopathic hemolysis.	No single test is diagnostic. Consider thrombotic microangiopathy and other differentials.
Bleeding, abruption, severe liver disease, shock or planned urgent procedure	PT/international normalized ratio; aPTT; fibrinogen; FBC; blood group and compatibility testing.	Assesses evolving consumption and supports transfusion planning.	Use serial results and clinical bleeding. Early coagulation tests may still be normal.
Possible APS based on thrombosis or qualifying pregnancy morbidity	Lupus anticoagulant; anticardiolipin IgG/IgM; anti-β2-glycoprotein I IgG/IgM.	Supports APS evaluation when the clinical history is compatible.	Not a universal PE screen. Persistent positivity generally requires confirmation ≥12 weeks apart.
Personal venous thromboembolism or strong family history	Specialist-directed factor V Leiden, prothrombin G20210A, antithrombin, protein C and protein S testing.	May inform selected thrombosis evaluation.	Not routine for PE alone; pregnancy, acute thrombosis and anticoagulation affect results.
Exploratory risk stratification	D-dimer, mean platelet volume and other platelet indices, RDW, NLR and PLR.	Research signals exist in small studies.	Insufficient regional validation, specificity or outcome benefit for routine PE diagnosis.

Abbreviations: aPTT: Activated Partial Thromboplastin Time; APS: Antiphospholipid Syndrome; AST: Aspartate Aminotransferase; ALT: Alanine Aminotransferase; FBC: Full Blood Count; HELLP: Hemolysis, Elevated Liver Enzymes and Low Platelet Count; Ig: Immunoglobulin; LDH: Lactate Dehydrogenase; NLR: Neutrophil-to-Lymphocyte Ratio; PE: Pre-Eclampsia; PLR: Platelet-to-Lymphocyte Ratio; PT: Prothrombin Time; RDW: Red-cell Distribution Width

Perspectives and next steps

Future research should use multicenter prospective designs, standard ISSHP phenotypes, appropriate normotensive and disease-control groups, serial sampling and pre-specified maternal and neonatal outcomes. Diagnostic-accuracy, incremental-value, and cost-effectiveness analyses are needed before new markers enter routine care. A planned doctoral program could evaluate aPL—lupus anticoagulant, anticardiolipin, and anti-β2-glycoprotein I antibodies—together with factor V Leiden and prothrombin G20210A in carefully selected women with severe PE, eclampsia or recurrent pregnancy morbidity. Repeating aPL testing, quality-controlled genotyping, and transparent handling of ancestry, anticoagulation, and gestational age would be essential. The aim should be a validated, affordable clinical strategy rather than a broad unselected panel.

Declarations

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this article.

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Author contributions

Mame Ngoné Coly: Conceptualization, literature review, Writing – original draft, Writing – review and editing. Macoura Gadji: Supervision, Writing – review and editing. Demba Macalou, Mamadou Wagué Guèye and Sérigne Modou Kane Guèye: Validation, Writing – review and editing. All authors read and approved the final version.

Data availability

No new datasets were generated or analyzed during the current review.

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