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Research Article

hormonal profile analysis in the Management of preeclampsia among pregnant women in low-resource settings

Rao R^{1*}, Rao P² and Iyer K³

¹Division of Gynecology, Po Research Center for Gynecology, India

²Department of Gynecology, Mackenzie Academy of Obstetrics and Maternal Health, India

³Department of Clinical Obstetrics and Maternal Health, Po Research Center for Gynecology, Pakistan

*Address for Correspondence: Rohan Rao, Division of Gynecology, Po Research Center for Gynecology, India; E-mail: rohan.rao@po.edu

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Abstract

Background: Preeclampsia remains a leading cause of maternal and perinatal death, and its burden falls disproportionately on low-resource settings where laboratory and referral infrastructure are limited [1,2].

Objective: This review synthesizes the evidence on hormonal and angiogenic biomarker profiling, principally soluble fms-like tyrosine kinase-1 (sFlt-1) and placental growth factor (PlGF), and evaluates its role in the diagnosis, risk stratification, and management of preeclampsia among pregnant women in low-resource settings.

Methods: We conducted a narrative synthesis of mechanistic, diagnostic-accuracy, randomized-trial, and multicountry low-resource cohort literature addressing hormonal biomarkers and their integration into preeclampsia management pathways.

Findings: The sFlt-1:PlGF ratio and PlGF alone show strong short-term negative predictive value for ruling out imminent preeclampsia [10,11,12], while risk models built specifically for low-resource use, such as miniPIERS, retain useful discrimination without requiring laboratory biomarker access [15]; aspirin, calcium supplementation, and magnesium sulfate remain universally deliverable interventions acting on related pathophysiological pathways [23,26,22].

Conclusions: Hormonal profile analysis offers genuine diagnostic and prognostic value in preeclampsia, and the most effective low-resource strategy combines targeted biomarker testing where feasible with validated clinical risk tools and universally deliverable pharmacological interventions [16,13].

Keywords: preeclampsia; hormonal profile; sFlt-1; placental growth factor; low-resource settings

1. Introduction

Preeclampsia remains one of the leading direct causes of maternal and perinatal death worldwide, and its burden falls disproportionately on low-resource settings, where laboratory infrastructure, specialist referral pathways, and access to antenatal ultrasound are all limited [1,2]. The disorder arises from a two-stage process in which incomplete trophoblastic remodeling of the uterine spiral arteries leads to relative placental ischemia, driving circulating factors that produce the maternal endothelial dysfunction seen clinically as new-onset hypertension and proteinuria after 20 weeks [3,4]. Circulating hormonal and angiogenic markers, most prominently soluble fms-like tyrosine kinase-1 (sFlt-1) and placental growth factor (PlGF), have moved from research tools into markers with genuine clinical utility for diagnosis, triage, and short-term risk prediction [5,6,7].

Blood pressure and dipstick proteinuria, the two pillars of traditional diagnosis, are nonspecific and vary with measurement technique and observer, a particular problem in busy low-resource clinics staffed by non-specialist personnel [8,9]. The sFlt-1:PlGF ratio and PlGF alone have both been validated as short-term rule-out and rule-in tools, with the PROGNOSIS study establishing a ratio below 38 as carrying a high negative predictive value for the absence of preeclampsia within one week [10], and the PELICAN and Chappell studies establishing analogous performance for PlGF used alone [11,12].

Extending this evidence into low-resource settings is not a simple matter of exporting the same assays and thresholds: point-of-care platforms, cold-chain requirements, cost, and later presentation with more severe disease all differ from the well-resourced cohorts in which the original biomarker studies were performed [13,14]. Risk-prediction tools built for low-resource contexts, such as miniPIERS, deliberately avoid dependence on laboratory hormonal assays in their core

version, though performance improves when biomarker data can be added [15,16]. Models for early-onset disease such as PREP, and screening algorithms combining maternal characteristics with biophysical and biochemical markers such as the Fetal Medicine Foundation competing-risks approach, illustrate the spectrum from purely clinical to biomarker-integrated risk stratification any low-resource strategy must navigate [17,18,19]. This review synthesizes the evidence on hormonal profile analysis, principally sFlt-1, PlGF, and their ratio, alongside adjunct markers such as human chorionic gonadotropin, progesterone, cortisol, and relaxin, and evaluates their role in diagnosis, risk stratification, and management of preeclampsia among pregnant women in low-resource settings.

2. Methods

This is a narrative synthesis of the published literature on hormonal and angiogenic biomarker profiling in preeclampsia, with particular attention to feasibility, diagnostic performance, and management implications in low- and middle-income settings. We drew on landmark cohort studies establishing the pathophysiological rationale for angiogenic markers [6,5], prospective diagnostic accuracy and rule-out studies of the sFlt-1:PlGF ratio and PlGF alone [20,10,11,12], randomized trials and systematic reviews of pharmacological management including magnesium sulfate, low-dose aspirin, and calcium supplementation [21,22,23,24,25,26,27], and multicountry risk-prediction and epidemiological studies conducted in low-resource obstetric populations [15,16,14,13].

Studies were selected for topical relevance, favoring randomized trials, large prospective cohorts, and systematic reviews, supplemented by mechanistic and biomarker-discovery studies establishing the physiological basis for the markers discussed [3,4,28,29,30]. International consensus guidance from FIGO, ACOG, and WHO was reviewed to contextualize

how biomarker findings have been translated into practice recommendations applicable to low-resource care [31,32,33,34]. As this is a narrative rather than a systematic review, no formal risk-of-bias scoring or meta-analytic pooling was performed; findings are organized thematically around the diagnostic, predictive, and therapeutic-monitoring roles hormonal profiling can serve in resource-constrained maternity care.

3. Results

Angiogenic and placental hormone markers in preeclampsia pathophysiology

The circulating hormonal signature of preeclampsia centers on an antiangiogenic shift, driven principally by excess placental secretion of sFlt-1, a soluble decoy receptor that binds and neutralizes vascular endothelial growth factor and PlGF, together with a fall in free PlGF [6]. Levine and colleagues showed that sFlt-1 rises and PlGF falls beginning approximately five weeks before clinical onset, with the divergence most pronounced in early-onset and severe disease [5]. Soluble endoglin, a coreceptor for transforming growth factor-beta, rises in parallel and adds independent predictive information, particularly for preterm and severe phenotypes [7]. Together these findings established angiogenic hormone profiling as a window onto placental dysfunction that precedes overt disease by weeks [3,30].

Beyond the core angiogenic axis, adjunct hormonal markers reflect the same underlying placental pathology. Reduced first-trimester pregnancy-associated plasma protein-A and abnormal human chorionic gonadotropin trajectories have long been associated with preeclampsia risk, and metabolomic profiling has identified early-pregnancy biomarker panels that improve discrimination when combined with angiogenic markers [28]. Relaxin, a placental and corpus luteal hormone with vasodilatory actions, is paradoxically reported as elevated in some cohorts alongside reduced renal responsiveness, suggesting an insufficient counter-regulatory response

[29]. Cortisol and other stress-axis hormones show altered diurnal patterns in preeclamptic pregnancies, consistent with the systemic inflammatory and endothelial activation of the syndrome [35]. Standardized case definitions from international consensus groups have been essential to making findings across these heterogeneous marker cohorts comparable, given that preeclampsia is a clinically heterogeneous syndrome rather than one disease entity [36,37].

Table 1: Principal hormonal and angiogenic markers evaluated in preeclampsia and their reported clinical application [6,5,7,20,29].		
Marker	Direction of change	Reported clinical application
sFlt-1	Increased	Rises weeks before clinical onset; core component of the sFlt-1:PlGF ratio
PlGF	Decreased	Short-term rule-out of preeclampsia-related delivery; stand-alone triage test
sFlt-1:PlGF ratio	Increased	Ratio <=38 carries high negative predictive value at one week
Soluble endoglin	Increased	Adds independent predictive value, especially for early-onset/severe disease
Relaxin	Increased (paradoxical)	Marker of insufficient compensatory vasodilatory response

Diagnostic and short-term predictive performance of the sFlt-1:PlGF ratio

The sFlt-1:PlGF ratio has moved furthest toward clinical adoption of any hormonal biomarker combination in preeclampsia. Verlohren and colleagues validated an automated, standardized assay platform for the ratio and proposed diagnostic cutoffs discriminating preeclampsia from other hypertensive and non-hypertensive complications with high accuracy [20]. The PROGNOSIS study established that a ratio of 38 or below carried a negative predictive value exceeding 99% for preeclampsia absence within one week, giving clinicians a validated tool to rule out imminent disease and safely avoid unnecessary hospitalization [10]. Chappell and colleagues, and subsequently the larger PARROT trial, showed that PlGF testing alone similarly discriminates women who will develop preeclampsia requiring delivery within 14 days,

and that revealing results to clinicians shortens time to correct diagnosis and reduces adverse maternal outcomes [11,12].

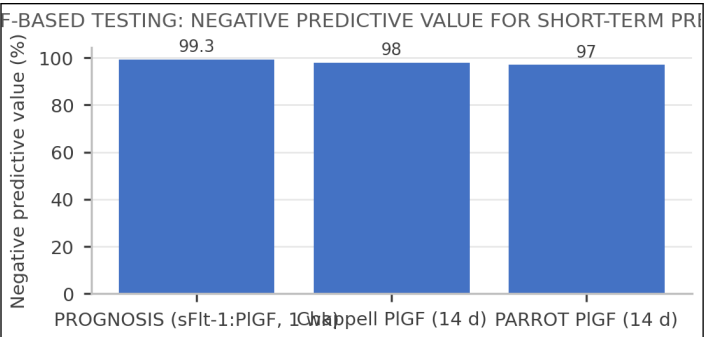


Figure 1: Reported negative predictive value of PIGF-based testing for ruling out preeclampsia-related delivery within a short-term window, drawn from the PROGNOSIS sFlt-1:PIGF validation cohort and the Chappell and PARROT PIGF-alone studies [10,11,12].

These findings have direct relevance to low-resource management, because a validated rule-out test reduces unnecessary referral, transport, and inpatient admission. Uptake has nonetheless been slower than the diagnostic evidence would predict, reflecting cost, platform availability, and the absence of local validation cohorts [13,14]. The miniPIERS and fullPIERS risk models illustrate the tension directly: fullPIERS incorporates laboratory and, where available, biomarker data to predict adverse maternal outcomes within 48 hours with strong discrimination, while miniPIERS was deliberately built to perform adequately using only clinical variables, so risk stratification remains possible where hormonal assays cannot be obtained at first contact [16,15].

Hormonal profiling and therapeutic decision-making

Hormonal and angiogenic biomarker results increasingly inform not only diagnosis but the timing and intensity of management. Elevated sFlt-1:PIGF ratios correlate with disease severity and interval to delivery, giving clinicians a quantitative complement to blood pressure trend and symptom review when deciding surveillance intensity, corticosteroid timing, and delivery threshold [10,34]. This is distinct from, but complementary to, established pharmacological interventions that act indirectly on the

same pathways. Low-dose aspirin, initiated before 16 weeks in high-risk women, produces a substantial reduction in preterm preeclampsia, attributed in part to improved trophoblastic invasion and moderation of the antiangiogenic shift [23,25,27,24]. Calcium supplementation, of particular relevance where dietary intake is inadequate, reduces hypertensive-disorder risk, plausibly through vascular smooth muscle and renin-angiotensin-aldosterone effects intersecting the same endothelial pathways [26].

Table 2: Management interventions relevant to low-resource settings and their mechanistic relationship to the hormonal/angiogenic profile [23,26,21,22,38].

Intervention	Setting relevance	Mechanistic link to hormonal profile
Low-dose aspirin from <16 weeks	Universally deliverable	Improves trophoblastic invasion; moderates antiangiogenic shift
Calcium supplementation	Targets low dietary intake	Affects vascular tone/renin-angiotensin axis intersecting endothelial pathways
Magnesium sulfate	Cornerstone for severe disease/eclampsia	Neuroprotective; acts independent of angiogenic markers
Individualized BP control (CHIPS)	Applicable without biomarker access	Complements, does not replace, biomarker-guided surveillance

Magnesium sulfate remains the cornerstone of eclampsia prevention once severe disease is established, and the Magpie trial and Cochrane synthesis confirmed its efficacy in reducing eclampsia and probable maternal death across well-resourced and low-resource sites, independent of hormonal profiling [21,22]. The CHIPS trial's demonstration that less-tight blood pressure control does not worsen perinatal outcomes further supports individualized management informed by the whole clinical and biochemical picture [38]. Table 1 summarizes the principal hormonal and angiogenic markers and their clinical application, and Table 2 summarizes management interventions relevant to low-resource settings and their mechanistic link to the hormonal profile.

Feasibility of hormonal profiling in low-resource obstetric settings

Multicountry studies conducted in low-resource settings highlight both the promise and the constraints of hormonal profile analysis. The miniPIERS study, across sites in Africa, Asia, and Latin America, found that adverse maternal outcomes could be predicted using low-cost clinical variables alone with reasonable discrimination, improving further when basic laboratory data were incorporated, without requiring specialized angiogenic assays [15]. A secondary analysis of the CRADLE trial across ten low- and middle-resource regions documented substantial variation in eclampsia incidence and case fatality, underscoring that delayed recognition, rather than biomarker unavailability alone, drives preventable severe outcomes, and that low-cost interventions such as accurate blood pressure devices retain outsized value [14,8]. Figure 1 illustrates reported negative predictive value of PlGF-based testing for ruling out preeclampsia-related delivery across key validation cohorts, and Figure 2 illustrates the relative reduction in preterm preeclampsia with low-dose aspirin by timing of initiation [23,25,27,24]. A randomized interventional study of point-of-care angiogenic testing alongside standard care further suggests that even intermittent biomarker access can improve recognition of women progressing to preeclampsia or eclampsia [39].

4. Discussion

The accumulated evidence supports a layered role for hormonal profile analysis that is achievable, in graded fashion, even where full angiogenic testing is not universally available. At the mechanistic level, the sFlt-1/PlGF antiangiogenic shift, together with adjunct changes in relaxin, cortisol, and placental hormone trajectories, explains why preeclampsia behaves as a systemic endothelial disorder rather than a localized hypertensive event, and this clarity has enabled the diagnostic and short-term predictive tools now entering guideline recommendations [6,5,3,31]. Where resources

allow at least intermittent PlGF or sFlt-1:PlGF testing, the evidence for using these results to rule out imminent preeclampsia or identify women needing urgent surveillance is strong enough to be standard of care in several national guidelines [10,12,32].

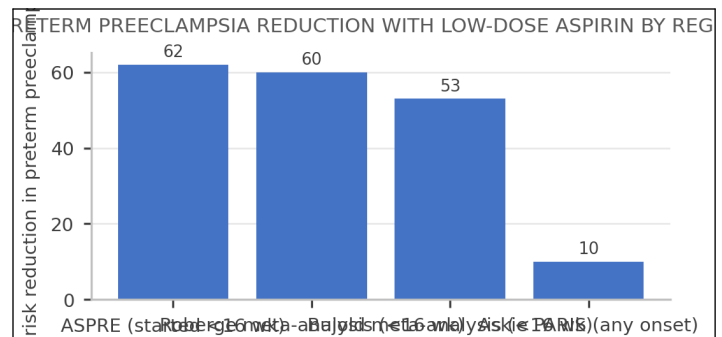


Figure 2: Relative reduction in preterm preeclampsia with low-dose aspirin in high-risk women by trial and timing of initiation, drawn from the ASPRE trial and subsequent meta-analyses [23,25,27,24].

For settings where such testing remains unavailable, the pragmatic response is to combine what hormonal profiling has taught about disease trajectory with clinical risk models validated for low-resource use, such as miniPIERS, alongside universally implementable interventions: accurate blood pressure measurement, calcium supplementation where dietary intake is low, risk-based aspirin prophylaxis from early pregnancy, and magnesium sulfate for severe disease and eclampsia prevention [15,26,23,21,22]. WHO and FIGO guidance both frame biomarker testing as an adjunct that strengthens, rather than replaces, this core package, supported by the finding that clinical-variable-only models retain useful discrimination without hormonal data [33,31,15].

Local validation of sFlt-1:PlGF and PlGF thresholds in low-resource populations, who differ in nutritional status and gestational age at presentation from the largely European and North American cohorts underlying current cutoffs, remains limited, and cost and platform availability are the dominant barriers to scale-up rather than any deficiency in diagnostic performance [13,14]. Point-of-care hormonal assay platforms suitable for

primary-level facilities represent the most consequential unmet need, since the value of hormonal profiling is greatest at first contact, where triage decisions including referral are made [16,8].

5. Conclusion

Hormonal profile analysis, anchored by the sFlt-1:PIGF ratio and PIGF alone, together with adjunct markers reflecting placental and endothelial dysfunction, has transformed the diagnostic and short-term predictive management of preeclampsia and offers genuine, evidence-based value when integrated into care pathways in low-resource settings [5,10,11]. Because full biomarker access remains inconsistent across low-resource health systems, the strongest available strategy combines targeted hormonal testing where feasible with clinical risk-prediction tools built for resource-limited use and with universally deliverable interventions, including calcium supplementation, risk-based aspirin prophylaxis, and magnesium sulfate, that address the same underlying pathophysiology even in the absence of a laboratory result [15,26,23,22]. Expanding affordable, point-of-care hormonal testing, alongside continued clinical-model refinement and health-system strengthening, is the priority for translating three decades of angiogenic biomarker research into measurable reductions in preeclampsia-related maternal and perinatal mortality among pregnant women in low-resource settings [1,13].

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