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Hemodynamic phenotypes in preeclampsia, improving therapeutic strategies: a scoping review

Abstract

Introduction: Preeclampsia (PE) is a multisystem hypertensive disorder classified among the great obstetrical syndromes, with an estimated prevalence of 2–8% worldwide. According to updated definitions (ISSHP/RBEHG), PE is characterized by new-onset hypertension after 20 weeks of gestation associated with proteinuria, maternal organ dysfunction, and/or placental involvement. Recent evidence describes two distinct hemodynamic phenotypes defined by maternal cardiovascular adaptation, each with specific clinical and paraclinical characteristics. Their identification may provide insight into underlying pathophysiology, allow more precise clinical management, and inform individualized selection of antihypertensive treatment. **Objective:** To identify and synthesize the available evidence on maternal hemodynamic profiles and phenotypic classifications in preeclampsia, and to examine their implications for diagnosis and therapeutic management.

Materials and Methods: We conducted a scoping review of literature published between January 1st 1980, and June 30th 2025, in PubMed/MEDLINE, Ebsco, Lilacs, and Embase. Articles and books in English and Spanish were considered using MeSH terms related to hemodynamic profiles, preeclampsia subtypes, maternal cardiovascular adaptation, and great obstetrical syndromes. Reviews and guidelines were also included to integrate evidence and clinical recommendations, with special attention to Latin American contributions.

The protocol for this scoping review was registered in the Open Science Framework (OSF) (DOI: 10.17605/OSF.IO/U5QDJ).

Results and conclusions: A total of 30 articles met inclusion criteria. The evidence supports that phenotypic classification of PE based on maternal hemodynamic adaptation offers a more accurate understanding of its heterogeneous pathophysiology. This approach may improve diagnostic stratification, support individualized antihypertensive selection, and help guide follow-up strategies, although prospective validation is still needed.

Keywords: great obstetrical syndromes, preeclampsia classification, preeclampsia subtype, maternal cardiovascular adaptation, cardiac output, hemodynamics, gestational hypertension.

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Introduction

Preeclampsia (PE) is a pregnancy-specific, multisystem hypertensive disorder defined by the onset of arterial hypertension after 20 weeks of gestation, accompanied by proteinuria, maternal organ dysfunction (such as renal, hepatic, hematologic, or neurological involvement), and/or evidence of placental dysfunction. It is classified among the maternal–placental syndromes and complicates 2–8% of pregnancies worldwide. According to the World Health Organization, PE is the second leading direct cause of maternal death globally, accounting for approximately 14% of maternal mortality. In Latin America and the Caribbean, up to 22% of maternal deaths are attributable to this condition.[1,2]

During normal pregnancy, profound maternal cardiovascular adaptations occur to meet the metabolic demands of both the mother and the developing fetus. These include a 30–50% increase in cardiac output, progressive expansion of plasma volume, and a marked reduction in systemic vascular resistance, all of which ensure adequate uteroplacental perfusion.[1–3] In women who develop preeclampsia, these physiological adaptations are often blunted or dysregulated, resulting in heterogeneous hemodynamic responses.[2] This variability has led to the identification of distinct hemodynamic phenotypes: a hyperdynamic profile, characterized by elevated cardiac output and reduced vascular resistance, and a hypodynamic profile, defined by low cardiac output and increased vascular resistance. Recognition of these phenotypes is clinically relevant, as they may influence both disease severity and response to antihypertensive therapy.[4]

Both early-onset and late-onset preeclampsia have been identified as distinct entities based on maternal hemodynamic patterns. Early-onset PE appears to be associated with abnormal uteroplacental vascular remodeling, characterized by increased total vascular resistance (TVR) and reduced maternal cardiac output (CO). In contrast, late-onset PE is more closely related to maternal constitutional factors such as elevated body mass index (BMI), advanced maternal age, and ethnicity, and is hemodynamically characterized by low vascular resistance and elevated cardiac output.[4]

The classification of PE into early-onset (<34 weeks of gestation) and late-onset (≥34 weeks of gestation) has helped delineate two pathophysiologically distinct entities. Early-onset PE is frequently associated with placental dysfunction, reduced placental volume, fetal growth restriction (FGR), abnormal uterine artery Doppler findings, low birth weight, multiorgan dysfunction, perinatal death, and adverse maternal and neonatal outcomes. In contrast, late-onset PE is more often related to underlying maternal conditions and is typically associated with normal placental morphology, greater placental

volume, normal fetal growth, and normal uterine artery Doppler findings.[3]

Accurate identification of maternal hemodynamic profiles in patients with PE may contribute to a more individualized approach to antihypertensive selection and clinical monitoring, although evidence supporting routine phenotype-guided treatment remains limited.

The aim of this review was to conduct a scoping review providing a novel perspective on preeclampsia from a hemodynamic standpoint. Beyond the traditional early- and late-onset classification, this article seeks to offer conceptual tools to support a targeted and individualized clinical approach, grounded in an understanding of the underlying pathophysiology and distinct disease phenotypes.

Materials and methods

Based on the research question—*What is known about maternal hemodynamic phenotypes in preeclampsia, and how might these profiles guide antihypertensive and fluid management strategies?*—this study was conducted using a scoping review methodology in accordance with the PRISMA-ScR guidelines (Preferred Reporting Items for Systematic Reviews and Meta-Analyses extension for Scoping Reviews). The objective was to identify and synthesize the available literature on hemodynamic profiles in preeclampsia, focusing on key concepts such as definitions, hemodynamic states, phenotypic classifications, and clinical implications.

Eligibility Criteria

Eligibility criteria were defined according to the PCC (Population–Concept–Context) framework:

a) **Population:** Pregnant women diagnosed with preeclampsia.

b) **Concept:** Maternal hemodynamic profiles, cardiovascular function, and phenotypic classification of preeclampsia assessed using imaging, Doppler, or other hemodynamic monitoring techniques.

c) **Context:** Clinical and research settings evaluating diagnostic approaches or therapeutic implications of hemodynamic assessment in preeclampsia.

The review included observational studies, clinical trials, systematic reviews, and narrative reviews evaluating hemodynamic profiles in preeclampsia. Articles published in English or Spanish between January 1, 1980, and June 30, 2025, were considered. Full-text availability was required.

Case reports, editorials, letters to the editor, conference abstracts, and studies not addressing maternal hemodynamic assessment in preeclampsia were excluded.

Information sources and search strategy

A comprehensive literature search was conducted in PubMed/MEDLINE, EBSCO, LILACS, and Embase, covering studies published between January 1, 1980, and June 30, 2025.

The search strategy combined controlled vocabulary (MeSH and Emtree terms) with free-text terms related to preeclampsia and maternal hemodynamics, adapted to each database's indexing system.

The search included the following terms:

("Preeclampsia"[MeSH] OR preeclampsia OR "pregnancy-induced hypertension")

AND

("Hemodynamics"[MeSH] OR hemodynamics OR "cardiac output" OR "systemic vascular resistance" OR "arterial stiffness")

AND

(phenotype OR phenotypes OR subtype OR subtypes OR profile OR profiles)

No language restrictions were applied during the search. Reference lists of included studies and relevant reviews were manually screened to identify additional eligible publications.

The final search was conducted on June 30, 2025.

Study selection

The selection process was conducted manually by two independent reviewers. Titles and abstracts were initially screened for relevance, followed by full-text assessment of potentially eligible articles. Discrepancies were resolved through discussion and consensus with a third reviewer. No specific software (e.g., Rayyan) was used.

In addition to original studies reporting hemodynamic parameters and PE phenotypes, guidelines and review articles providing conceptual frameworks or clinical recommendations were also included. For review articles, only the synthesized evidence was considered, and individual studies were not double-counted. To enhance clarity, guidelines and reviews are presented separately from primary studies.

Data extraction focused on maternal hemodynamic assessment (e.g., cardiac output, vascular resistance, Doppler indices, echocardiography, and impedance cardiography) and phenotype classification (e.g., hypodynamic vs. hyperdynamic, early- vs. late-onset PE). When such data were unavailable, studies were retained for their relevance and marked as "NA" in the summary tables.

Data extraction and analysis

A standardized data extraction template was developed in Excel, including article type, study design, population characteristics, hemodynamic assessment methods, and main findings. A descriptive analysis was performed, accompanied by a narrative synthesis of key results.

Quality assessment of the studies

Although risk-of-bias assessment is not mandatory in scoping reviews, a critical appraisal of included studies was conducted using the Newcastle–Ottawa Scale, according to the respective observational study design. Overall, the included studies demonstrated moderate to high methodological quality, supporting the robustness of the available evidence. The most common limitations involved group comparability and completeness of outcome assessment; however, these did not preclude inclusion, as the primary objective was to map existing evidence on preeclampsia phenotypes.

Results

The PRISMA flow diagram (Figure 1) illustrates the study selection process. For consistency in terminology, early-onset preeclampsia was defined as diagnosis before 34 weeks of gestation, and late-onset preeclampsia as diagnosis at or after 34 weeks. A qualitative synthesis is presented in Table 1, detailing the main findings and study characteristics.

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Table 1 Summary of the 30 studies included in the review, detailing study design, country, objectives, methodology, and key findings. The table highlights the heterogeneity of approaches, with observational studies, systematic and narrative reviews, meta-analyses, and guidelines or expert recommendations. NA: not available/not applicable. For review articles and guidelines, "NA" indicates that no primary hemodynamic assessment or phenotype classification was reported, but the study was included for its conceptual or clinical relevance

Author	Country	Study design	Hemodynamic assessment	Phenotype(s)	Main findings
ACOG Practice Bulletin No. 222.[1]	USA	Guideline / Practice Bulletin	NA	NA	Clinical guidance on diagnosis and management of gestational hypertension and preeclampsia
Say et al.,[2]	Global/WHO	Systematic Analysis	NA	NA	Global causes of maternal death; contextualizes PE burden
Raymond & Peterson,[3]	USA	Critical Review	NA	Early vs. Late onset PE	Differences between early- and late-onset preeclampsia summarized
Valensise et al.,[4]	Italy	Observational	Echocardiography+ Doppler	Early vs. Late PE	Two distinct maternal hemodynamic states identified
Di Pasquo et al.,[5]	Italy (Multicenter)	Prospective Multicenter	Hemodynamic-guided therapy	Hyperdynamic/ Hypodynamic	Tailored therapy based on maternal hemodynamics showed effectiveness
Chinali et al.,[6]	Multinational (LIFE study)	Prospective	Echocardiography (diastolic indices)	NA	Diastolic indices predicted outcomes during antihypertensive treatment
Ling et al.,[7]	UK	Longitudinal Cohort	Echocardiography	Various	Progressive maternal cardiac dysfunction documented
Gyselaers,[8]	Belgium	Narrative Review	Conceptual framework	Conceptual	Proposed hemodynamic pathways of gestational hypertension and PE
Vonck et al.,[9]	Belgium	Observational	Hepatic Doppler Hemodynamics	NA	Association between hepatic hemodynamics and fetal growth
Bijl et al.,[10] (IWGNH)	International	Methodological Recommendations	Cardiac output measurement methods	NA	Recommendations for cardiac output measurement in pregnancy
Foo et al.,[11]	UK	Prospective Cohort	Arterial function / CV assessment	NA	Pre-pregnancy CV function linked to later PE/FGR
Sibai,[12]	USA	Editorial/ Commentary	NA	Classification/ Prediction	Highlights role of maternal and uteroplacental hemodynamics
Masini et al.,[13]	Italy/UK	Review / Proposal	Multiple	Hyperdynamic vs. Hypodynamic	Argues for phenotype-based treatment strategies
Longo,[14]	USA	Hypothesis / Review	NA	NA	Endocrinologic control hypothesis for maternal blood volume and CO
Meah et al.,[15]	UK	Meta-analysis	Multiple	NA	Quantifies changes in cardiac output during pregnancy
Valensise et al.,[16]	Italy	Longitudinal Observational	Echocardiography+ Uteroplacental Doppler	NA	Links maternal cardiac function with uteroplacental resistance
Foo et al.,[17] (IWGNH)	International	Recommendations	Arterial function assessment	NA	Consensus recommendations for arterial assessment
Mahendru et al.,[18]	UK	Longitudinal	Arterial stiffness/ Hemodynamics	NA	Tracks maternal cardiovascular function across timeline
Yagel et al.,[19] (AJOG MFM)	Israel	Expert Review	Integrated cardio-placental model	Type I / Type II	Proposes classification of preeclampsia
Buddeberg et al.,[20]	UK	Observational (term PE)	Echocardiography	Cardiac maladaptation	Demonstrates cardiac maladaptation in term PE

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Table 1 Continued...

Verlohren et al.,[21]	Multicenter Europe	Diagnostic Study	sFlt-1/PlGF ratio	NA	Defines biomarker cutoffs for PE diagnosis
Burwick & Rodriguez,[22]	USA	Review	Angiogenic biomarkers	NA	Review of biomarkers in preeclampsia
Perry et al.,[23]	UK	Cross-sectional	NICOM (non-invasive CO monitor)	NA	Device-specific reference ranges
Rana et al.,[24]	USA	Review	Pathophysiology overview	NA	Comprehensive PE pathophysiology review
Miranda et al.,[25]	Colombia	Observational	Doppler echocardiography	Severe phenotype	Hemodynamic profile linked to severe PE
Easterling et al.,[26]	USA	Longitudinal	Hemodynamics (incl. echo)	NA	Differences in normal vs. PE pregnancies
Yagel et al.,[27] (UOG)	Israel	Expert Opinion / Review	Integrated model	Type I / Type II	Redefines PE classification
Bartsch et al.,[28]	Canada	Systematic Review & Meta-analysis	NA	Risk stratification	Identifies early pregnancy risk factors
Lain & Roberts,[29]	USA	Review	NA	Pathogenesis/ Management	Overview of PE pathogenesis and management
Dimopoulou et al.,[30]	UK	Long-term Cohort	CV assessment post-HDP	NA	Long-term cardiovascular outcomes after HDP

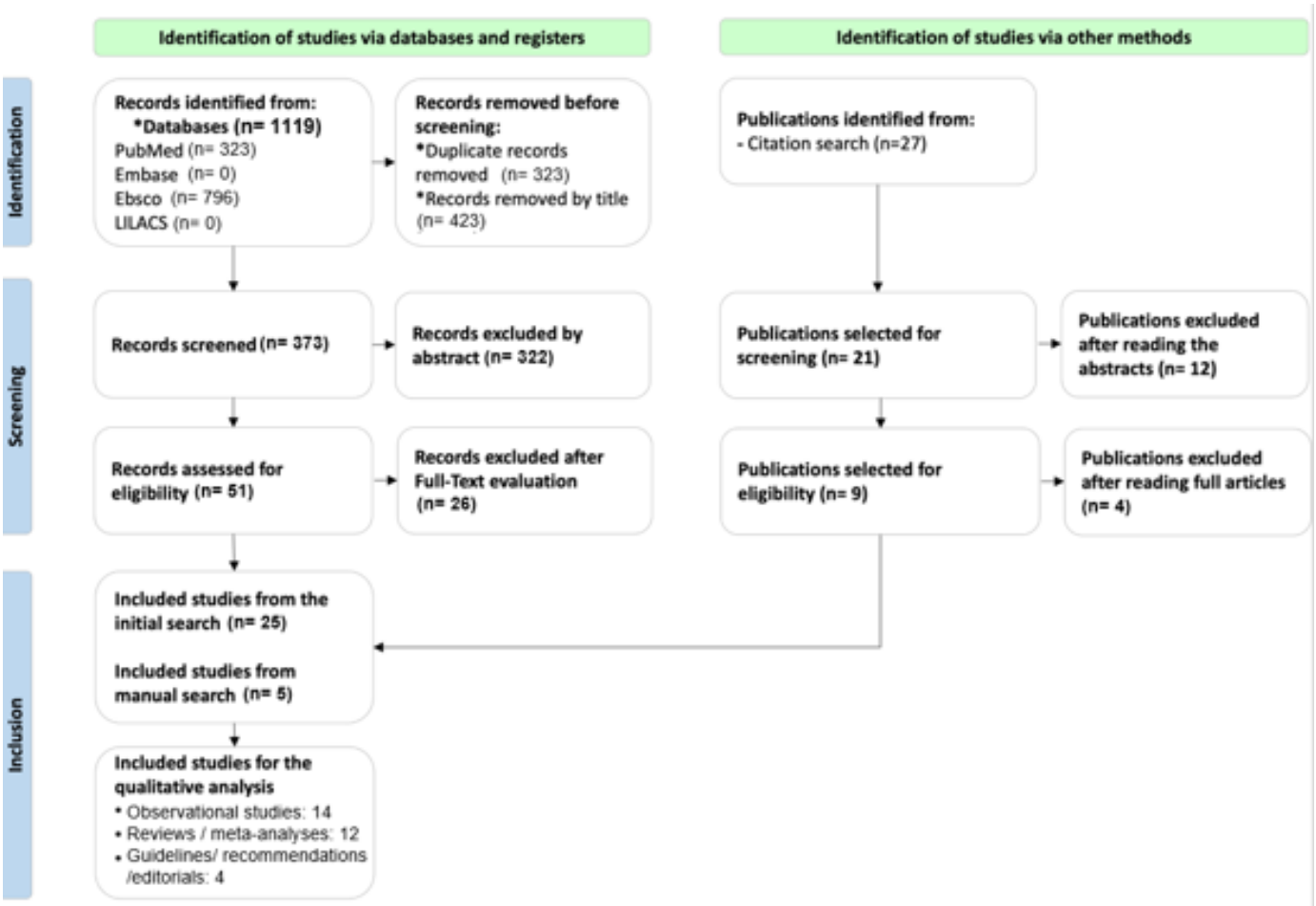

Figure 1 PRISMA flow diagram of the study selection process.

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A total of 1119 records were identified across PubMed/MEDLINE, Ebsco, Lilacs, and Embase. After removal of duplicates and title review, 373 records remained for screening. Following abstract review, 51 articles were excluded. The full text of 51 papers was assessed, of which 26 were excluded due to the following reasons: studies not involving pregnant women, absence of hemodynamic data, review articles or editorials, and duplicates. Finally, 30 studies were included in the qualitative synthesis

The included studies reported maternal cardiovascular and hemodynamic findings associated with preeclampsia, including changes in cardiac output, total peripheral vascular resistance, cardiac remodeling, and angiogenic markers. The main findings of the reviewed studies are summarized below.

Perry H. et al.[23], in a prospective cohort study of 411 healthy pregnant women with singleton gestations, evaluated changes in stroke volume, cardiac output (CO), TVR throughout pregnancy using a noninvasive bioreactance-based device. The study demonstrated a progressive increase in CO until approximately 35 weeks, followed by a decline toward term. TVR decreased until around 36 weeks and then increased again as gestation approached 40 weeks. Stroke volume showed a linear increase throughout gestation. Maternal characteristics associated with increased CO included body weight, height, and age ($p < 0.05$). Ethnicity was significantly associated with stroke volume ($p = 0.001$), but not with CO or TVR.[23]

Hypertensive disorders of pregnancy have been associated with abnormal cardiac adaptation, as demonstrated in the prospective longitudinal study of maternal hemodynamics by Ling HZ et al[7]. In this study, pathological cardiac remodeling and altered hemodynamic profiles were observed in patients with hypertension and preeclampsia (PE). From 20 weeks of gestation onward, the hemodynamic profile in hypertensive patients shifted from hyperdynamic to hypodynamic, whereas in those with PE there was a continuous decrease in cardiac output (CO) and an increase in TVR.[7]

A study conducted in the obstetric high-dependency unit at Hospital Universitario Infantil de San José in Bogotá, Colombia, found that 67% of patients with severe PE had abnormal echocardiographic findings, with a higher frequency in early-onset PE. The most common abnormalities were pulmonary hypertension (49%), left ventricular hypertrophy (27.5%), and diastolic dysfunction (13.7%).[31]

Buddeberg BS et al[20] demonstrated impaired cardiac adaptation to increased workload in pregnancy complicated by PE in a case-control study of 30 women at term. Conventional echocardiography revealed left ventricular diastolic dysfunction with an increased E/e' ratio in the PE group ($p = 0.001$) compared with normotensive controls, as well as a significant reduction in global left ventricular strain ($p < 0.001$), affecting both the epicardium ($p < 0.001$) and endocardium ($p < 0.001$), and a reduced longitudinal strain rate ($p < 0.001$). These findings are associated with an increased risk of long-term cardiovascular disease.

Dimopoulou S et al[30] conducted a longitudinal analysis demonstrating that women with a history of hypertensive disorders of pregnancy developed mild cardiovascular abnormalities. These included increased left ventricular systolic pressure (elevated mitral transvalvular flow; $p = 0.004$), altered diastolic function indices (increased E/e' ratio; $p < 0.001$), and structural changes such as increased relative wall thickness ($p < 0.001$), persisting at least two years postpartum. This analysis included a detailed cardiovascular assessment in mid-pregnancy among 112 women who subsequently developed preeclampsia or gestational hypertension. Persistent changes during follow-up also included an increased peak systolic velocity ratio in the ophthalmic artery, reflecting elevated peripheral vascular resistance.

In a study by Herbert Valensise et al[4], maternal echocardiography and uterine artery Doppler were performed in 1,345 normotensive nulliparous women at 24 weeks of gestation. On follow-up, 2.3% developed late-onset PE and 5.5% developed early-onset PE. Bilateral uterine artery notching was observed in 15.6% and 60% of these groups, respectively ($p < 0.05$). Cardiac output was significantly higher in late-onset PE compared with early-onset PE (8.96 ± 1.83 L/min vs. 4.49 ± 1.09 L/min; $p < 0.001$), along with lower TVR (739 ± 244 vs. $1,605 \pm 248$ dyn·s·cm^{-5}).[4]

Among functional cardiac changes, late-onset PE was associated with an increased left ventricular mass index and prolonged mitral deceleration time (DTE), a prognostic marker of left ventricular diastolic dysfunction,[6] whereas early-onset PE was characterized by prolonged isovolumetric relaxation time and increased relative wall thickness.

Sibai BM et al[12] emphasized the role of inadequate or exaggerated cardiovascular adaptation before 20 weeks of gestation, which is associated with complications such as gestational hypertension, PE, and FGR. Early-onset PE involves placental ischemic lesions, elevated TVR, and reduced CO, whereas late-onset PE is not associated with these findings and is more commonly linked to maternal factors such as elevated body mass index.

Masini G et al[13] described early-onset PE as being associated with low cardiac output (<5 L/min), high vascular resistance (>1,400 dyn·s·cm^{-5}), and intravascular volume depletion, whereas late-onset PE typically presents with high cardiac output (>8 L/min), normal or low vascular resistance (<900 dyn·s·cm^{-5}), and intravascular volume overload.

Yagel et al[19] proposed an integrated model incorporating maternal, placental, and fetal factors in the development of PE, identifying two subtypes: Type I PE, which tends to present earlier and is characterized by placental dysfunction or poor perfusion, shallow trophoblast invasion, elevated soluble fms-like tyrosine kinase-1 (sFlt-1), low placental growth factor (PlGF), high TVR, and low CO; and Type II PE, which occurs later in gestation and is associated with maternal cardiovascular intolerance, a moderately dysfunctional placenta, and inadequate perfusion. In this subtype, the sFlt-1/PlGF ratio may be normal or only mildly altered, TVR is low, and CO is high.[19]

Differences in the clinical presentation of PE phenotypes are also reflected in angiogenic imbalance involving two major placenta-derived biomarkers used to predict severe disease. Verlohren et al[21] established sFlt-1/PlGF ratio cut-off values for the diagnosis of early- and late-onset PE, identifying a threshold of 33 with 95% sensitivity and 94% specificity for early-onset PE, and a cut-off of 85 with 99.5% specificity. For late-onset PE, a threshold of 110 was established to maintain specificity.[21,22]

Figure 2 provides a summary of the specific risk factors associated with each preeclampsia phenotype and their corresponding perinatal outcomes.

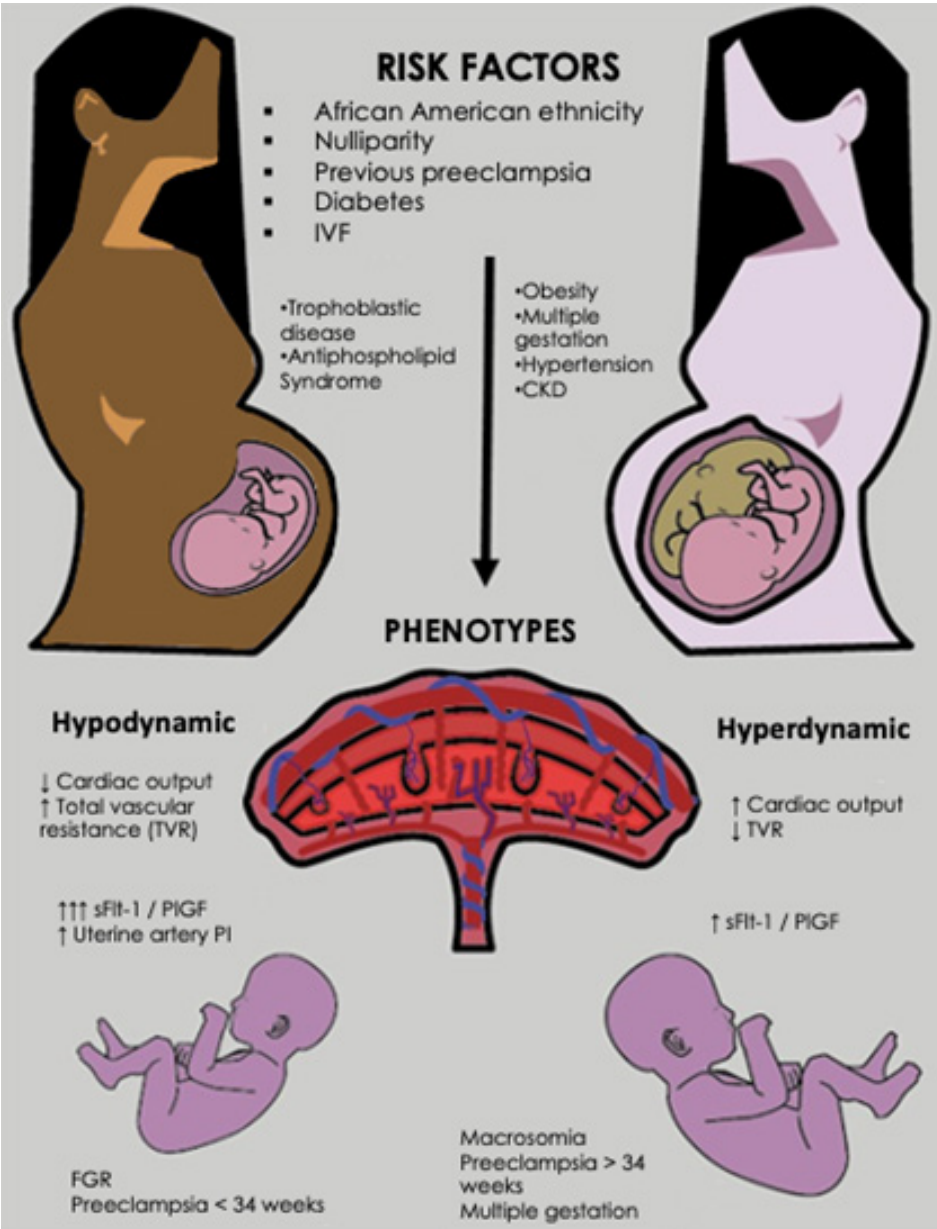

Figure 2 Risk factors and perinatal outcomes associated with preeclampsia phenotypes.

Abbreviations: FGR = Fetal Growth Restriction, sFlt-1 = Soluble fms-like tyrosine kinase-1, PlGF = Placental Growth Factor, PI = Pulsatility Index, TVR = Total Vascular Resistance, IVF = In Vitro Fertilization, CKD = Chronic Kidney Disease.

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Table 2 summarizes the main clinical, hemodynamic, and pathophysiological characteristics of the identified preeclampsia phenotypes.

Table 2 Preeclampsia phenotypes

Category	Hypodynamic	Hyperdynamic
Hemodynamic profile	↓ Cardiac Output	↑↑↑ Cardiac Output
	↑↑↑ Total Vascular Resistance (TVR)	↓ TVR
Cardiac characteristics	- ↓ Ventricular wall thickness and diameters	- Ventricular enlargement and hypertrophy
	- Inadequate filling	- Excessive filling
	- Pressure overload state	- No pressure overload
	- Concentric hypertrophy	
	- Increased total body water	
Placental alteration	- Placental dysfunction	- Placental dysfunction
	- Poor perfusion	- Poor perfusion
	↑ Uterine artery Doppler PI	
Angiogenic markers	↑ sFlt-1	Normal or slightly ↑ sFlt-1
	↓ PlGF	↓ PlGF
Risk factors	- African American ethnicity	- Obesity RR 2.8 (95% CI 2.6–3.1)*
	- Nulliparity RR 2.1 (95% CI 1.9–2.4)*	- Multiple gestation RR 2.9 (95% CI 2.6–3.1)*
	- Previous preeclampsia RR 8.4 (95% CI 7.1–9.9)*	- Chronic hypertension RR 5.1 (95% CI 4.0–6.5)*
	- Pre-gestational diabetes RR 3.7 (95% CI 3.1–4.3)*	- Chronic kidney disease RR 1.8 (95% CI 1.5–2.1)*
	- Assisted reproductive technologies RR 1.8 (95% CI 1.6–2.1)*	
	- SLE RR 2.5 (95% CI 1.0–6.3)*	
	- Maternal age >35 years RR 1.2 (95% CI 1.1–1.3)*	
	- Thromboplastic disease	
	- APS RR 2.8 (95% CI 1.8–4.3)*	
Clinical impact	Early-onset preeclampsia (<34 weeks)	Late-onset preeclampsia (>34 weeks)
	↑ Fetal Growth Restriction (FGR)	Appropriate birthweight or macrosomia

Abbreviations: TVR = Total Vascular Resistance; sFlt-1 = Soluble fms-like tyrosine kinase-1; PlGF = Placental Growth Factor; RR = Relative Risk; SLE = Systemic Lupus Erythematosus; APS = Antiphospholipid Syndrome; HTN = Hypertension; CKD = Chronic Kidney Disease; FGR = Fetal Growth Restriction.

Bartsch E, Medcalf KE, Park AL, Ray JG; High Risk of Pre-eclampsia Identification Group. Clinical risk factors for pre-eclampsia determined in early pregnancy: systematic review and meta-analysis of large cohort studies. BMJ. 2016 Apr 19;353:i1753.[28]

Miranda et al[25] highlighted hemodynamic monitoring as a useful tool for identifying more severe phenotypes, irrespective of blood pressure levels. In an observational study of 30 patients with late-onset preeclampsia, the phenotype characterized by elevated total vascular resistance showed a trend toward higher BNP (brain natriuretic peptide) levels, longer hospital stays, and neonates with significantly lower birth weight percentiles.

Discussion

Preeclampsia (PE) is a multisystem disorder unique to pregnancy, characterized by new-onset hypertension after 20 weeks of gestation. It is considered one of the major obstetric syndromes and represents the second leading direct cause of maternal mortality worldwide.[1,2] Among the proposed etiologies, maternal systemic endothelial inflammation plays a central role, predisposing to oxidative stress at the level of the syncytiotrophoblast and leading to abnormal spiral artery remodeling and atherosis. This process results in increased expression of antiangiogenic factors, such as soluble fms-like tyrosine kinase-1 (sFlt-1), and reduced levels of placental growth factor (PlGF).[26]

Among the pathological alterations observed in PE, impaired maternal cardiac adaptation plays a key role in determining cardiovascular patterns. In early-onset PE, reduced ventricular wall thickness and chamber dimensions, together with apparent inadequate filling, pressure overload, and concentric hypertrophy, have been described. In contrast, patients with a hyperdynamic hemodynamic profile exhibit enlarged and hypertrophied ventricles in a state of volume overload without significant pressure overload.[4]

This synthesis of the evidence integrates the available literature on hemodynamic profiles in PE, identifying two main phenotypes as distinct pathophysiological entities. These phenotypes correspond to the cardiovascular changes observed in early- and late-onset PE, with a predominance of increased total peripheral vascular resistance in the former and elevated cardiac output in the latter.[11] In this review, early-onset preeclampsia was consistently defined as diagnosis before 34 weeks of gestation (<34 weeks), and late-onset preeclampsia as diagnosis at or after 34 weeks of gestation (≥34 weeks), to ensure comparability across studies. Each phenotype develops within a

characteristic clinical context and is associated with specific risk factors across different populations.

Women with early-onset, hypodynamic PE are at increased risk of fetal growth restriction, abnormal uterine artery Doppler findings with elevated resistance from the first trimester, and a greater imbalance in placental angiogenic factors. These features contrast with the hyperdynamic phenotype observed in patients who develop PE closer to term, in whom risk factors such as obesity, multiple gestation, chronic kidney disease, and chronic hypertension are more prevalent.[19] These abnormal hemodynamic patterns, described since the 1980s, have contributed to the development of a conceptual framework focused on understanding the cardiovascular alterations in patients affected by this condition.[26]

From a clinical perspective, the identification of hemodynamic phenotypes in preeclampsia offers a promising conceptual framework for individualized management. In theory, patients with a hypodynamic profile—characterized by low cardiac output and elevated total peripheral vascular resistance—could preferentially respond to antihypertensive agents with predominant vasodilatory effects, whereas women with a hyperdynamic phenotype—marked by increased cardiac output—might respond differently to agents that reduce heart rate and cardiac output, such as beta-blockers. However, these therapeutic considerations remain largely hypothesis-generating and are primarily supported by physiological rationale and observational data rather than by randomized controlled trials.

Several noninvasive techniques, including maternal echocardiography and thoracic electrical bioimpedance, enable assessment of maternal hemodynamic parameters and may facilitate earlier phenotype identification in specialized clinical settings.[10] The information obtained from these assessments may facilitate earlier phenotype identification and could support future individualized management strategies tailored to predominant hemodynamic patterns.

Similarly, serial hemodynamic assessment (serial assessment of blood pressure, heart rate, cardiac output, vascular resistance) combined with fetal Doppler evaluation may provide additional information for maternal–fetal surveillance and risk stratification. Nevertheless, its routine clinical implementation remains investigational, and standardized monitoring protocols have not yet been established.

The main limitation of this study, inherent to its scoping review design, is the inability to draw definitive conclusions from the available data. As such, it does not provide specific clinical recommendations or standardized management guidelines. Nevertheless, it highlights the need for future research focused on prenatal monitoring of cardiovascular parameters and their correlation with maternal, fetal, and neonatal outcomes. Such efforts may contribute to the development of more robust evidence for surveillance strategies and hemodynamically informed antihypertensive management. Importantly, the current evidence base is largely derived from small observational studies, and randomized controlled trials evaluating phenotype-based therapeutic strategies in preeclampsia remain scarce.

Conclusion

In conclusion, recognition of distinct hemodynamic phenotypes in preeclampsia provides a relevant framework for understanding disease heterogeneity and may ultimately contribute to improved risk stratification and more tailored management approaches, pending prospective validation. Future research should focus on prospective studies validating standardized hemodynamic assessment tools during pregnancy and evaluating whether hemodynamically guided management improves maternal, fetal, and neonatal outcomes. Additionally, interventional studies are needed to determine the effectiveness of phenotype-based therapeutic strategies and to inform future evidence-based recommendations for clinical practice.

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Conflicts of interest

Authors has no conflicts of interest to declare.

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