

PREECLAMPSIA: A REVIEW OF CONTEMPORARY CONCEPTS

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ABSTRACT

Preeclampsia is a multisystem disorder specific to pregnancy, characterized by new-onset hypertension after 20 weeks of gestation, associated with proteinuria or signs of maternal organ dysfunction and/or placental dysfunction. The aim of this review article is to provide a comprehensive overview of contemporary knowledge regarding the epidemiology, pathophysiology, risk factors, clinical presentation, diagnostic methods, prevention strategies, and therapeutic approaches in preeclampsia through analysis of current literature in this field. The literature for this review article was identified through searches of the PubMed/MEDLINE, Scopus, and Web of Science databases for the period 2021–2025. Appropriate keywords and MeSH terms related to preeclampsia and biomarkers were used, with particular emphasis on angiogenic and antiangiogenic factors. The analysis included original studies, cohort studies, randomized controlled trials, and systematic reviews. The selected studies were critically analyzed and synthesized to provide a concise and relevant overview of current knowledge on preeclampsia and the use of biomarkers in pregnancy. All healthcare institutions involved in the medical care of pregnant women with hypertensive disorders should implement standardized treatment protocols, regularly review them, and conduct periodic clinical evaluations of outcomes of such management on pregnancy outcomes.

Keywords: *Preeclampsia, hypertensive disorders of pregnancy, placental dysfunction, angiogenic biomarkers, sFlt-1/PlGF ratio, prevention of preeclampsia, management of preeclampsia.*

Abbreviation	Full term / Explanation
PE	Preeclampsia
eclampsia	Eclampsia
HTN	Hypertension
BP	Blood pressure
sFlt-1	Soluble fms-like tyrosine kinase-1
PlGF	Placental growth factor
sEng	Soluble endoglin
VEGF	Vascular endothelial growth factor
HELLP	Hemolysis, Elevated Liver enzymes, Low Platelets
NST	Non-stress test
ART	Assisted reproductive technologies
ROS	Reactive oxygen species
TNF- α	Tumor necrosis factor alpha
IL-6	Interleukin 6
WHO	World Health Organization
Ca	Calcium
MgSO ₄	Magnesium sulfate
Labetalol	Antihypertensive drug
Nifedipine	Antihypertensive drug, calcium channel blocker
Hydralazine	Antihypertensive drug
TGF- β	Transforming growth factor beta
Pl	Placenta
BMI	Body Mass Index
IUGR	Intrauterine Growth Restriction



INTRODUCTION

Preeclampsia is a multisystem disorder specific to pregnancy characterized by new-onset hypertension after 20 weeks of gestation, associated with proteinuria or signs of maternal organ dysfunction and/or placental dysfunction (1–3). Despite significant progress in understanding the underlying mechanisms of this disorder, preeclampsia remains an important cause of maternal and neonatal morbidity and mortality (3, 10).

Globally, hypertensive disorders in pregnancy complicate approximately 5–10% of all pregnancies, while preeclampsia occurs in about 2–8% of pregnant women (1, 20, 30). The incidence of the disease varies significantly between different populations and regions, reflecting differences in genetic predisposition, socioeconomic conditions, access to healthcare, and quality of prenatal care (7, 22).

The clinical manifestations of preeclampsia can range from mild hypertension to severe multisystem disease affecting the kidneys, liver, brain, and cardiovascular system (3, 25). In severe cases, preeclampsia may progress to life-threatening complications such as eclampsia, HELLP syndrome, placental abruption, and acute renal failure (9, 23, 25). These complications significantly increase the risk of maternal and perinatal mortality (9, 23, 30).

Historically, preeclampsia was primarily considered a disease of the maternal cardiovascular system, characterized by hypertension and proteinuria. However, contemporary research has demonstrated that the disorder actually originates from the placenta and involves complex interactions between placental dysfunction, the maternal immune response, and systemic endothelial injury (4, 13, 65, 69).

The placenta plays a central role in the pathogenesis of the disease through the release of various inflammatory mediators, oxidative stress molecules, and antiangiogenic factors into the maternal circulation (13, 65). Recent advances in molecular biology and obstetric research have significantly improved the understanding of the mechanisms leading to the development of preeclampsia (13, 66).

A particularly important discovery is the identification of angiogenic biomarkers, such as soluble fms-like tyrosine kinase-1 (sFlt-1) and placental growth factor (PlGF), which have provided new insights into the pathophysiology of the disease and opened possibilities for earlier diagnosis and prediction (5, 28, 60).

Despite these advances, the exact etiology of preeclampsia is still not fully elucidated. Today, this disorder is considered a multifactorial syndrome resulting from the interaction of genetic, immunological, vascular, and environmental factors (6, 66). This complexity makes the prevention and treatment of preeclampsia particularly challenging.

OBJECTIVE

The aim of this review article is to summarize contemporary scientific knowledge on the epidemiology, pathophysiology, risk factors, diagnostic approaches, prevention strategies, and therapeutic options in preeclampsia, based on the analysis of publications from recent years.

LITERATURE REVIEW METHODOLOGY

This study was designed as a narrative literature review with the aim of providing a comprehensive overview of current knowledge on preeclampsia. A literature search was conducted in the PubMed/MEDLINE, Scopus, and Web of Science databases covering the period from January 2021 to May 2025. The search strategy included combinations of the following keywords and MeSH terms: preeclampsia, hypertensive disorders of pregnancy, placental dysfunction, angiogenic biomarkers, sFlt-1, placental growth factor, sFlt-1/PlGF ratio, prevention of preeclampsia and management of preeclampsia. The review included original research articles, prospective and retrospective cohort studies, randomized controlled trials, systematic reviews, meta-analyses, and relevant international professional society guidelines. Studies were excluded if full-text versions were unavailable, if they were not published in English, or if their content was not directly related to the epidemiology, pathophysiology, diagnosis, or management of preeclampsia (33, 34). The objective of this study was not to provide a quantitative synthesis of evidence but rather to critically review and integrate the most relevant contemporary findings in this field.

EPIDEMIOLOGY

Preeclampsia represents one of the most significant complications of pregnancy and continues to be a major global public health problem. According to leading epidemiological studies, hypertensive disorders in pregnancy affect approximately 10% of all pregnancies worldwide, while preeclampsia occurs in 2–8% of pregnancies (2, 20).

The incidence of preeclampsia varies significantly across different regions of the world. A higher prevalence has been observed in low- and middle-income countries, where limited access to prenatal healthcare and underdeveloped healthcare infrastructure contribute to delayed diagnosis and greater disease severity (7, 23). In contrast, in developed countries, a gradual decline in maternal mortality associated with preeclampsia has been observed due to improvements in prenatal screening, early detection, and advances in obstetric medicine (2, 22).

Several demographic and clinical factors are associated with an increased risk of developing preeclampsia. One of the most important risk factors is nulliparity, as women experiencing their first pregnancy have a significantly higher risk compared to multiparous women (8, 21). Advanced maternal age, particularly above 35 years, has also been identified as a significant risk factor (21, 55, 56).



Maternal obesity represents another important factor contributing to the development of preeclampsia. Obesity is associated with chronic low-grade inflammation, insulin resistance, and endothelial dysfunction, all of which significantly contribute to the development of hypertensive disorders in pregnancy (9, 24, 55).

In addition to maternal factors, certain pregnancy-related conditions increase the likelihood of developing preeclampsia. These include multiple pregnancy, pregnancies achieved through assisted reproductive technologies, and molar pregnancy (10, 25). Multiple pregnancy is associated with a larger placental mass and higher levels of circulating angiogenic factors, which may contribute to the development of placental dysfunction (25).

Preeclampsia also has significant consequences for fetal health. The disorder is often associated with intrauterine growth restriction, preterm birth, and low birth weight (11). Placental insufficiency resulting from impaired uteroplacental blood flow may compromise the supply of oxygen and nutrients to the fetus, leading to adverse neonatal outcomes (11, 22).

Recent studies have also highlighted long-term health consequences of preeclampsia for both the mother and the child. Women who have experienced preeclampsia have an increased risk of developing cardiovascular diseases, hypertension, and metabolic syndrome later in life (12, 49–51). Similarly, children born from preeclamptic pregnancies may have an increased risk of developing cardiovascular and metabolic disorders in adulthood (52–54).

These findings underscore the importance of early detection, prevention, and adequate management of preeclampsia in order to prevent both short-term and long-term adverse health outcomes for both mother and child.

PATHOPHYSIOLOGY OF PREECLAMPSIA

The pathophysiology of preeclampsia is complex and involves a series of interconnected processes that begin in early pregnancy and progress throughout gestation. Although the precise mechanisms are not yet fully understood, current evidence supports the concept that preeclampsia arises as a consequence of abnormal placental development followed by systemic endothelial dysfunction in the mother (13, 64, 65).

The most widely accepted explanation of disease development is the two-stage model of preeclampsia, which includes abnormal placentation and a subsequent systemic maternal response (15).

Abnormal Placentation

During normal pregnancy, cytotrophoblast cells invade the spiral arteries of the uterus and transform them into large, low-resistance vessels capable of providing adequate blood flow to the placenta. This physiological process of vascular

remodeling ensures optimal delivery of oxygen and nutrients to the fetus (14, 67).

In preeclampsia, trophoblast invasion into the spiral arteries is incomplete and insufficient. As a result, the spiral arteries remain narrow and retain their high-resistance structure, significantly reducing uteroplacental blood flow (13, 15, 68).

This abnormal vascular remodeling leads to placental hypoperfusion and intermittent ischemia (15). Placental ischemia triggers oxidative stress and the release of numerous inflammatory mediators and antiangiogenic factors into the maternal circulation (12, 22). These substances play a key role in the development of systemic endothelial dysfunction responsible for the clinical manifestations of the disease (64).

Imbalance of Angiogenic Factors

One of the most significant advances in understanding the pathophysiology of preeclampsia is the identification of an imbalance in angiogenic factors as a key pathogenic mechanism. The placenta normally produces a range of angiogenic factors that regulate blood vessel development and maintain endothelial integrity (16, 28).

The two most important molecules in this process are vascular endothelial growth factor (VEGF) and placental growth factor (PlGF), which promote angiogenesis and vascular stability (28).

In preeclampsia, the placenta releases increased amounts of antiangiogenic factors, such as soluble fms-like tyrosine kinase-1 (sFlt-1) (17, 28). sFlt-1 binds circulating VEGF and PlGF, preventing their interaction with endothelial receptors. This process leads to reduced angiogenic signaling, endothelial dysfunction, and diffuse vasoconstriction (17, 28, 65).

Numerous studies have shown that elevated levels of sFlt-1 and decreased levels of PlGF are strongly associated with the development and severity of preeclampsia (5, 28, 59). These findings have enabled the development of diagnostic tests based on the sFlt-1/PlGF ratio, which are now widely used in clinical practice (5, 59, 60).

Endothelial Dysfunction

Endothelial dysfunction is considered the central pathophysiological event responsible for the clinical manifestations of preeclampsia. The imbalance of angiogenic factors disrupts normal endothelial function and leads to increased vascular permeability, vasoconstriction, and activation of the coagulation system (11, 19, 64).

These vascular changes contribute to the development of hypertension and damage to multiple organ systems, including the kidneys, liver, brain, and cardiovascular system (11, 19).



Endothelial injury also leads to platelet activation and microvascular thrombosis, further exacerbating tissue ischemia (19).

Immunological and Inflammatory Mechanisms

Pregnancy requires complex immunological adaptation between the maternal immune system and the semi-allogeneic fetus. In normal pregnancy, maternal immune tolerance enables proper placental development and fetal growth (20, 62).

In preeclampsia, this immunological tolerance appears to be disrupted. Abnormal interactions between maternal immune cells and trophoblast cells may impair placental implantation and contribute to placental dysfunction (62, 63).

Women with preeclampsia exhibit elevated levels of pro-inflammatory cytokines, including tumor necrosis factor alpha (TNF- α) and interleukin-6 (IL-6). These inflammatory mediators further exacerbate endothelial damage and contribute to the systemic inflammatory response (62, 63).

Oxidative Stress

Oxidative stress represents another important component in the pathogenesis of preeclampsia. Placental ischemia leads to increased production of reactive oxygen species (ROS), which can damage cellular structures and further impair endothelial function (12).

Excessive oxidative stress contributes to lipid peroxidation, inflammation, and apoptosis in placental tissue (12). These processes further worsen placental dysfunction and sustain the vicious cycle of systemic inflammation in the mother.

RISK FACTORS

Preeclampsia is a multifactorial disease influenced by various maternal, genetic, and environmental factors. Identification of women at increased risk is crucial for timely prevention and adequate prenatal monitoring (21, 24).

Maternal Risk Factors

Several maternal conditions significantly increase the likelihood of developing preeclampsia. One of the strongest predictors is a history of preeclampsia in a previous pregnancy, which substantially increases the risk of recurrence (23).

Chronic hypertension represents another important risk factor (41). Women with pre-existing hypertension have a higher likelihood of developing superimposed preeclampsia due to already present endothelial dysfunction (21, 23, 55).

Metabolic disorders such as diabetes mellitus and obesity are also strongly associated with the development of hypertensive disorders in pregnancy (24). Obesity contributes to systemic inflammation, insulin resistance, and endothelial in-

jury, all of which play a significant role in the pathogenesis of the disease (24, 55).

Other maternal risk factors include chronic kidney disease, autoimmune diseases such as systemic lupus erythematosus, antiphospholipid syndrome, and maternal age above 35 years (21).

Pregnancy-Related Risk Factors

Certain conditions related to pregnancy itself also increase the risk of preeclampsia. Multiple pregnancies are associated with a larger placental mass and increased production of placental hormones and angiogenic factors, which may contribute to disease development (69, 70).

Pregnancies achieved through assisted reproductive technologies (ART) are also associated with an increased risk (71, 72). This may be due to altered placental development or underlying causes of infertility in the mother (10, 25).

Genetic Factors

Genetic predisposition plays a significant role in the development of preeclampsia. Studies have shown that women with a family history of preeclampsia have an increased risk of developing this disorder (21).

Several genetic polymorphisms related to angiogenesis, immune regulation, and oxidative stress have been identified as potential contributors to the pathogenesis of preeclampsia (13, 65). However, the exact genetic mechanisms remain the subject of intensive research.

CLINICAL MANIFESTATIONS

The clinical presentation of preeclampsia may vary considerably—from mild hypertension to severe multisystem disease. Symptoms usually appear after the 20th week of pregnancy, although in rare cases they may occur earlier.

The most characteristic clinical sign is newly developed hypertension. Blood pressure values $\geq 140/90$ mmHg, measured on two separate occasions, are considered a diagnostic criterion for hypertensive disorders in pregnancy (26, 41, 42).

Proteinuria is another classic feature of the disease, most commonly defined as the presence of 300 mg or more of protein in a 24-hour urine sample (26). However, contemporary clinical guidelines recognize that preeclampsia may occur even in the absence of significant proteinuria if other signs of organ dysfunction are present (27, 42).

The most common symptoms reported by patients include: severe headache, visual disturbances, epigastric or right upper quadrant abdominal pain, nausea and vomiting, and sudden swelling of the face and hands. These symptoms may indicate a severe form of the disease and require urgent medical evaluation (25, 27).



DIAGNOSTIC CRITERIA

Modern diagnostic criteria for preeclampsia have evolved significantly over the past decades. Current guidelines emphasize that preeclampsia is a multisystem disorder rather than a condition defined solely by hypertension and proteinuria (27, 42, 61).

According to contemporary clinical guidelines, the diagnosis of preeclampsia can be established in the presence of hypertension after 20 weeks of gestation, along with at least one of the following findings: proteinuria, thrombocytopenia, renal insufficiency, elevated liver enzymes, pulmonary edema, or new-onset cerebral or visual disturbances (27, 42).

Laboratory investigations play an important role in confirming the diagnosis and assessing disease severity. Typical laboratory findings in preeclampsia may include elevated liver enzymes, decreased platelet count, and impaired renal function (25, 27).

Ultrasound examination is also essential for assessing fetal growth and placental function. Doppler ultrasound of uterine and umbilical arteries can provide valuable information about uteroplacental blood flow and fetal condition (28, 57).

Early detection of preeclampsia is crucial for preventing serious maternal and fetal complications (61). Therefore, regular prenatal monitoring and risk assessment remain fundamental components of obstetric care (16, 42).

BIOMARKERS AND CONTEMPORARY DIAGNOSTIC APPROACHES

Early detection of preeclampsia represents one of the most important goals of modern obstetric research. Traditional diagnostic criteria based on hypertension and proteinuria often identify the disease only after the onset of clinical symptoms. Therefore, considerable attention has been directed toward identifying reliable biomarkers that could enable earlier diagnosis and more precise risk assessment (5, 58).

Angiogenic Biomarkers

Among the most extensively studied biomarkers in preeclampsia are angiogenic and antiangiogenic factors produced by the placenta. The balance between these molecules plays a crucial role in maintaining normal endothelial function during pregnancy (16, 28).

The two most important proangiogenic factors are vascular endothelial growth factor (VEGF) and placental growth factor (PlGF), which promote angiogenesis and maintain vascular endothelial integrity (28).

In women who develop preeclampsia, circulating levels of these proangiogenic factors are significantly reduced (5, 28, 29).

VEGF and PlGF

Vascular endothelial growth factor (VEGF) and placental growth factor (PlGF) belong to the VEGF ligand family and play a key role in regulating angiogenesis, vasculogenesis, and endothelial function in both physiological and pathological conditions (28, 29).

VEGF represents a group of glycoproteins (the most well-known being VEGF-A) that act as potent mitogens specific to endothelial cells. Its effects are mediated through binding to tyrosine kinase receptors VEGFR-1 (Flt-1) and VEGFR-2 (KDR/Flk-1) (28).

Its biological effects include stimulation of endothelial cell proliferation and migration, induction of angiogenesis and vascular permeability, and inhibition of endothelial cell apoptosis (28, 29). VEGF is crucial during embryonic development as well as in adult tissue repair processes. Its expression is strongly regulated by hypoxia via HIF-1 (30).

PlGF is a member of the VEGF family that primarily binds to VEGFR-1 (Flt-1), unlike VEGF-A, which has a higher affinity for VEGFR-2. Its biological functions include modulation of angiogenesis, particularly under pathological conditions, synergistic interaction with VEGF, and involvement in inflammatory processes (28, 31). PlGF is physiologically most highly expressed in the placenta during pregnancy (31).

In normal pregnancy, there is a balance between proangiogenic and antiangiogenic factors, such as sFlt-1 (28, 29). Disruption of this balance leads to endothelial dysfunction and the development of preeclampsia (5, 28, 59).

sFlt-1 and sEng

Soluble fms-like tyrosine kinase-1 (sFlt-1) and soluble endoglin (sEng) are key antiangiogenic factors (17). sFlt-1 is a circulating form of the VEGF receptor that binds VEGF and PlGF, preventing their biological activity (67). This mechanism leads to inhibition of angiogenesis and endothelial dysfunction (17). sEng acts by inhibiting TGF- β signaling and reducing nitric oxide synthesis, thereby contributing to vasoconstriction (35).

Their synergistic action leads to pronounced endothelial dysfunction and more severe forms of the disease (34, 35, 36).

sFlt-1/PlGF Ratio

One of the most clinically useful diagnostic markers is the sFlt-1/PlGF ratio. Numerous studies have demonstrated that this ratio has high predictive value for the development of preeclampsia (5, 59, 60).

A ratio lower than 38 is generally considered indicative that preeclampsia is unlikely to develop in the short term, whereas significantly elevated values indicate an increased risk of disease progression (59). This biomarker is particu-



larly useful in the differential diagnosis between preeclampsia and other hypertensive disorders in pregnancy (60).

Novel Biomarkers

In addition to angiogenic factors, contemporary research is investigating other potential biomarkers for early detection of preeclampsia. These include inflammatory cytokines, markers of oxidative stress, microRNAs, and placental extracellular vesicles (61). Advances in molecular biology and genomics may further improve the ability to identify women at high risk (13).

Critical Appraisal of Biomarkers

Despite significant advances, several limitations remain regarding the clinical application of biomarkers in preeclampsia.

Studies differ considerably with respect to:

- inclusion criteria;
- gestational age at assessment;
- laboratory methodologies;
- ethnic composition of the studied populations.

In addition, optimal cutoff values for many biomarkers have not been universally established across different populations.

Although the sFlt-1/PIGF ratio is currently the most extensively validated biomarker, its use should be considered part of an integrated clinical approach rather than a substitute for comprehensive clinical evaluation of the pregnant woman.

Contemporary risk assessment models increasingly combine clinical risk factors, Doppler ultrasound parameters, and biochemical biomarkers, thereby achieving greater diagnostic accuracy than any individual parameter used in isolation.

THERAPY

Currently, there is no specific therapy that can cure preeclampsia prior to delivery, and therefore delivery remains the only definitive treatment (42–44). Management is primarily symptomatic and focused on controlling blood pressure, preventing seizures, and maintaining optimal fetal oxygenation (16, 42).

Medications used include antihypertensive agents (labetalol, nifedipine) and magnesium sulfate for the prevention of eclampsia (44, 45).

In severe cases, such as preeclampsia with severe features or fetal compromise, urgent delivery is indicated regardless of gestational age (45–47).

PREVENTION OF PREECLAMPSIA

Preventive strategies for preeclampsia are primarily aimed at identifying women at increased risk and implementing early interventions that can reduce the incidence and severity of the disease (48).

Risk Assessment in Early Pregnancy

Early prenatal screening plays a crucial role in identifying women at increased risk of developing preeclampsia. Risk assessment typically includes analysis of maternal medical history, clinical characteristics, and laboratory parameters.

Women considered high-risk include those with a history of preeclampsia, chronic hypertension, diabetes mellitus, autoimmune diseases, chronic kidney disease, and multiple pregnancy.

Early identification of these factors enables timely implementation of preventive measures and closer monitoring throughout pregnancy (2, 38).

Low-Dose Aspirin Prophylaxis

The use of low-dose aspirin has become the most commonly recommended preventive therapy for preeclampsia. Contemporary clinical guidelines recommend administration of 75–150 mg of aspirin daily, starting between the 12th and 16th week of pregnancy, in women at high risk (36–38).

Aspirin is believed to improve placental blood flow through inhibition of platelet aggregation and increased production of prostacyclin, a vasodilatory mediator.

Large randomized clinical trials have demonstrated that early administration of aspirin significantly reduces the risk of preeclampsia, particularly the early-onset form of the disease.

Calcium Supplementation

Calcium supplementation has also shown a protective effect in populations with low dietary calcium intake (19, 38–40).

Calcium plays an important role in vascular smooth muscle function and may contribute to blood pressure regulation during pregnancy. The World Health Organization (WHO) recommends daily calcium supplementation in pregnant women in regions where dietary calcium intake is insufficient.

The Role of Vitamin D in Preeclampsia Prevention

In recent years, vitamin D has attracted considerable attention due to its potential role in the regulation of immune responses, angiogenesis, and placentation. Numerous observational studies have demonstrated an association between low serum concentrations of 25-hydroxyvitamin D



[25(OH)D] and an increased risk of developing preeclampsia (73, 74).

Proposed mechanisms include:

- impaired immune tolerance;
- endothelial dysfunction;
- disrupted angiogenesis;
- activation of the renin–angiotensin system.

However, although epidemiological data suggest a possible association, the results of randomized controlled trials remain inconsistent. Some studies have demonstrated a reduced risk of preeclampsia among women with marked vitamin D deficiency, whereas others have failed to confirm a statistically significant preventive effect of supplementation. Methodological differences among studies further complicate the interpretation of the available evidence. These differences include:

- varying vitamin D doses;
- differences in the timing of supplementation initiation;
- different definitions of vitamin D deficiency;
- heterogeneity of the study populations.

Therefore, the current level of evidence is insufficient to recommend routine vitamin D supplementation solely for the prevention of preeclampsia. Nevertheless, maintaining an adequate vitamin D status remains important for the overall health of the pregnant woman and the normal development of the fetus. Additional large-scale multicenter randomized studies are needed to more precisely define the potential role of vitamin D in the prevention of preeclampsia (75).

Lifestyle Modifications

Lifestyle modifications may also contribute to reducing the risk of preeclampsia. These include maintaining a healthy body weight prior to pregnancy, regular physical activity, and adequate prenatal care.

Although these measures alone cannot completely prevent the disease, they contribute to improved maternal cardiovascular health and more favorable pregnancy outcomes.

COMPLICATIONS OF PREECLAMPSIA

Preeclampsia can lead to serious complications for both the mother and the fetus.

Maternal complications include eclampsia, HELLP syndrome (Hemolysis, Elevated Liver enzymes, Low Platelets), acute renal failure, pulmonary edema, and disseminated intravascular coagulation (25, 27, 42).

Fetal complications include intrauterine growth restriction, preterm birth, hypoxia, and perinatal death (16, 27).

Early diagnosis and timely intervention significantly reduce the risk of these outcomes.

SCIENTIFIC AND CLINICAL SIGNIFICANCE

Research on preeclampsia has important implications not only for obstetrics but also for broader fields of cardiovascular and metabolic medicine.

Preeclampsia is increasingly recognized as an early indicator of long-term cardiovascular risk in women. Women who have experienced preeclampsia during pregnancy have a significantly increased risk of developing hypertension, ischemic heart disease, stroke, and metabolic syndrome later in life (18, 23, 53, 54).

These findings highlight the importance of long-term cardiovascular follow-up and preventive strategies in women with a history of hypertensive disorders in pregnancy.

Furthermore, studying the mechanisms of preeclampsia provides important insights into vascular biology, endothelial dysfunction, and inflammatory processes, which are also involved in numerous other cardiovascular diseases.

FUTURE PERSPECTIVES

Future research in the field of preeclampsia will likely focus on improving early prediction of the disease and developing targeted therapeutic approaches.

New technologies, including genomic analyses, proteomics, and predictive models based on contemporary scientific knowledge, may significantly improve the identification of women at high risk of developing preeclampsia already in early pregnancy.

Another promising area of research involves the development of novel pharmacological therapies aimed at correcting angiogenic imbalance and improving placental function.

Although delivery is currently the only definitive treatment, future therapeutic strategies may enable prolongation of pregnancy and improvement of neonatal outcomes.

CONCLUSION

All healthcare institutions involved in the medical care of pregnant women with hypertensive disorders should implement standardized treatment protocols, regularly review them, and conduct periodic clinical evaluations of the outcomes of such management on pregnancy outcomes.

Preeclampsia remains one of the most important complications of pregnancy and a significant cause of maternal and perinatal morbidity and mortality worldwide.

The disease is characterized by complex pathophysiological mechanisms that include abnormal placentation, imbalance of angiogenic factors, endothelial dysfunction, inflammation, and oxidative stress.



Contemporary research in molecular biology and obstetrics has significantly improved the understanding of the disease and enabled the development of new diagnostic methods, particularly angiogenic biomarkers such as the sFlt-1/PlGF ratio.

Preventive strategies, including the use of low-dose aspirin and calcium supplementation, have demonstrated significant benefits in women at high risk.

However, early detection and careful monitoring remain essential for preventing severe complications.

Further research into the molecular mechanisms of preeclampsia, as well as the development of innovative diagnostic and therapeutic approaches, will be crucial for improving outcomes for mothers and newborns in the future.

CONFLICT OF INTEREST STATEMENT

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