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Review Article

Hypertension in pregnancy: effects on maternal and fetal health and long-term outcomes

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ABSTRACT

Hypertensive disorders of pregnancy (HDP) are heterogeneous maternal-placental syndromes and continue to be a major cause of maternal and perinatal morbidity and mortality worldwide. Their clinical spectrum ranges from gestational and chronic hypertension to preeclampsia, eclampsia and HELLP syndrome with markedly different biological mechanisms and outcomes. This narrative review critically discusses the current knowledge on the pathophysiology, classification, diagnosis, complications, and long-term outcomes of HDP. Current evidence supports a multifactorial model whereby abnormal placentation and impaired spiral artery remodelling interact with maternal cardiovascular, metabolic, immune, genetic and environmental susceptibility to produce endothelial dysfunction and clinical disease. Imbalances in angiogenesis, in particular sFlt-1 and PlGF changes, have improved our understanding of disease biology and have provided clinically useful tools to assess risk in suspected preeclampsia. However, biomarker performance is dependent on gestational age, disease phenotype, population and clinical setting, which limits their universal applicability. Severe HDP can lead to eclampsia, HELLP syndrome, renal and hepatic dysfunction, stroke, placental insufficiency, foetal growth restriction, stillbirth, and medically indicated preterm birth. Emerging evidence links HDP with subsequent maternal cardiovascular and renal disease and potential long-term cardiometabolic effects in exposed offspring. Importantly, the evidence available indicates that no single biomarker or molecular pathway is sufficient to represent the heterogeneity of HDP. Therefore, future strategies should focus on integrating clinical features with angiogenic, placental, cell-free nucleic acid, metabolomic and proteomic markers, possibly supported by artificial intelligence.

Keywords: Hypertensive disorders of pregnancy, Preeclampsia, Gestational hypertension, Eclampsia, Diagnostic biomarkers, Maternal health

INTRODUCTION

Hypertensive disorders of pregnancy (HDP) are one of the most important complications in modern obstetrics and are a major cause of maternal, foetal and neonatal morbidity and mortality worldwide. These conditions occur in approximately 2-8% of pregnancies and are a major component of preventable maternal morbidity and mortality, especially in low- and middle-income countries where late diagnosis, inadequate antenatal monitoring and limited access to specialised obstetric care remain significant healthcare problems.[1] The HDP group includes chronic hypertension, gestational hypertension, preeclampsia, superimposed preeclampsia on chronic hypertension, eclampsia and HELLP syndrome. While these disorders share the clinical feature of elevated maternal blood pressure, the underlying biology, clinical presentation, disease trajectory and maternal-fetal consequences differ substantially.

We believe that this heterogeneity matters clinically, as the increase in blood pressure alone is not a good proxy for the biological severity or prognosis of HDP. So, the time of disease onset, markers of placental dysfunction, maternal cardiovascular and metabolic susceptibility, and maternal or foetal complications should be analysed together in the evaluation of disease severity and risk. Among these conditions, preeclampsia is particularly complex as it is a multisystem disorder characterised by placental dysfunction, diffuse endothelial dysfunction, inflammatory activation, and variable involvement of maternal organs beyond hypertension alone. In the last 20 years, significant progress in our understanding of HDP has demonstrated that abnormal placental development is a key initiating mechanism in many instances.[2]

Impairment of trophoblastic invasion and remodelling of the maternal spiral arteries can contribute to placental hypoperfusion, ischaemia and oxidative stress. These processes promote the release of anti-angiogenic factors, inflammatory mediators, extracellular vesicles and oxidative products in the maternal circulation, which collectively lead to systemic endothelial dysfunction and vascular injury.[3] Nevertheless, the data reviewed in this manuscript suggest that placental dysfunction alone cannot fully account for the dramatic clinical heterogeneity of HDP. Instead, the maternal response to placental stress appears to be determined by the maternal baseline cardiovascular, metabolic, inflammatory and immunologic status.

Disease presentation, severity and progression are therefore likely to reflect the relationship between placental abnormalities and maternal susceptibility including genetic predisposition, pre-existing cardiovascular disease, obesity, diabetes mellitus, chronic kidney disease, immune dysregulation and environmental exposures. This interaction may explain the observation that women with apparently similar degrees of hypertension may have vastly different maternal and foetal outcomes. Clinically, this endorses a move to a more integrated model that takes into account maternal phenotype, placental function, gestational age at onset and evidence of end-organ involvement, rather than an understanding of HDP primarily as a blood pressure-defined disorder. Recent advances in molecular medicine have improved our understanding of HDP and have generated potential tools for earlier risk assessment.[3]

Biomarkers, including the soluble fms-like tyrosine kinase-1/placental growth factor (sFlt-1/PlGF) ratio, circulating cell-free DNA, metabolomic signatures, proteomic profiles and artificial intelligence-assisted prediction models, have emerged as promising approaches for identifying women at increased risk or for diagnostic stratification before or around the onset of clinical disease.[2] However, there is no evidence yet to justify the uniform application of these approaches in all clinical settings. Differences in population characteristics, gestational age, disease phenotype, assay performance, availability and cost may influence their diagnostic or predictive value. Thus, in our opinion, the major challenge is no longer only the identification of potential biomarkers, but the determination of which combinations of clinical, biochemical and placental markers provide sufficiently reproducible and clinically meaningful information to change patient management. [3] This distinction is particularly relevant to translation into routine obstetric practice.

The effects of HDP reach well beyond pregnancy, too. There is increasing epidemiological evidence that women with a history of hypertensive pregnancy are at increased subsequent risk of chronic hypertension, ischemic heart disease, heart failure, stroke, chronic kidney disease and metabolic disorders. Similarly, offspring exposed to hypertensive pregnancies would be more prone to develop cardiovascular, hypertensive, metabolic and neurodevelopmental disorders later in life. These findings suggest that HDP may be both a complication of pregnancy and an important early-life marker of future maternal and offspring health.[4]

We believe that this long-term perspective adds weight to the argument for considering pregnancy as an opportunity to identify women and families at increased risk of lifetime cardiometabolic risk rather than limiting management to the immediate peripartum period. HDP have been extensively reviewed. Most existing reviews summarize individual areas of evidence without fully integrating advances in molecular biology, biomarker research, disease phenotyping, and long-term cardiovascular outcomes. The growing body of evidence has significantly changed the current understanding of HDP pathogenesis, diagnosis and risk stratification. A better synthesis should therefore not only inform on new findings, but also discuss their clinical significance, limitations and potential translation to other populations and healthcare systems.[4]

The literature is growing but there are important gaps in our knowledge on how to reliably predict disease onset, the mechanisms that lead from early placental dysfunction to maternal clinical disease, effective prevention strategies and the long-term outcome for both mother and offspring. Future advances in molecular profiling and precision medicine need to be validated further before they can be incorporated into clinical practice with ease.

The present review critically appraises the current evidence of the epidemiology, pathophysiology, classification, diagnosis, maternal and fetal complications and long-term outcomes of HDP. Recent diagnostic and biomarker-based approaches are particularly emphasized, in light of their current limitations and translational challenges.

The review also highlights unmet clinical needs and gaps in current research and sets out future directions that may improve early risk stratification, enable more personalized

management and ultimately improve short and long-term maternal and neonatal outcomes.

PHYSIOLOGICAL AND MOLECULAR CHANGES IN HYPERTENSIVE PREGNANCIES

HDP should be considered as heterogeneous syndromes that result from an interaction between abnormal placentation and maternal cardiovascular, metabolic, immunological and vascular susceptibility. While hypertension is the hallmark clinical feature, it is a downstream manifestation of a disease process that may begin far earlier in gestation. Importantly, the biology of women with HDP is heterogeneous. For instance, early-onset preeclampsia is more strongly related to placental dysfunction, while late-onset disease may be more influenced by maternal cardiometabolic and vascular factors. Such heterogeneity has implications for the interpretation of biomarkers and the development of strategies for prediction and prevention.[5]

The pivotal role of the placenta in this pathophysiology. Coordinated trophoblast invasion and remodelling of maternal spiral arteries is required for normal placental development. In early pregnancy, the vessels are progressively remodelled by trophoblast-mediated changes from relatively narrow, high-resistance arteries to dilated, low-resistance conduits capable of sustaining adequate uteroplacental perfusion. In preeclampsia and some other forms of HDP, this remodelling is incomplete and leads to a persistently high resistance uteroplacental circulation. However, abnormal spiral artery remodelling alone does not fully explain the clinical phenotype as placentation abnormalities can also be present in pregnancies without clinical preeclampsia. Thus, placental pathology must interact with maternal susceptibility to be able to produce clinically overt disease.[6]

Intermittent hypoxia-reoxygenation, oxidative stress and cellular stress in placenta may result from insufficient placental perfusion. These perturbations seem to activate multiple stress-response pathways, such as inflammatory signalling and abnormal placental factor release into the maternal circulation, instead of just causing a chronic placental hypoxia. Syncytiotrophoblast-derived extracellular vesicles, inflammatory mediators, cell-free nucleic acids and anti-angiogenic proteins may then contribute to systemic endothelial activation. Thus, the maternal vascular endothelium is a key interface between placental pathology and the clinical manifestations of disease. Hypertension, proteinuria and end-organ involvement can be produced by increased vascular permeability, altered vascular tone, platelet activation and inflammatory activation in combination.

Disruption of angiogenic signalling is one of the molecular pathways that has drawn much clinical and translational interest in HDP. The placenta produces more soluble fms-like tyrosine kinase-1 (sFlt-1) which binds the proangiogenic molecules vascular endothelial growth factor (VEGF) and placental growth factor (PlGF) and reduces their availability. This shift to an anti-angiogenic state is associated with endothelial dysfunction and is more pronounced in preeclampsia. The sFlt-1/PlGF ratio has thus become a clinically useful tool for the assessment of women with suspected pre-eclampsia, particularly in the setting of an uncertain diagnosis. However, its role must be understood as part of broader clinical assessment and not as evidence of one causal pathway.[6,7]

Variations in gestational age, disease phenotype, assay platforms and population characteristics may affect biomarker concentrations and their clinical performance. Another explanation for variable presentation of HDP is importance of maternal cardiovascular phenotype. Obesity, insulin resistance, dyslipidaemia, chronic hypertension, diabetes, renal disease and pre-existing vascular dysfunction may all reduce maternal ability to adapt to the hemodynamic requirements of pregnancy. Pregnancy can thus be considered a physiologic cardiovascular stress test; women with limited cardiovascular reserve may develop hypertension or endothelial dysfunction when the demands of pregnancy exceed their adaptive capacity. This idea also helps explain the increased risk of hypertension and cardiovascular disease later in life in women with a history of HDP.

Thus, HDP may not only be a pregnancy specific complication but also an early clinical marker of underlying maternal cardiometabolic vulnerability. The contribution of the immune system also supports the notion of HDP as a multisystem disorder. Abnormal maternal immune tolerance at the maternal-fetal interface, altered regulatory T cell activity, complement activation, and an imbalance between pro- and anti-inflammatory pathways have all been implicated. The evidence, however, does not point to a single immune mechanism that applies to all patients. Instead, dysregulation of immunity seems to interact with placental stress and susceptibility of the maternal vasculature, which may exacerbate endothelial injury and systemic inflammation. This distinction is important as it alters the interpretation of inflammation from being a primary cause of HDP, to potentially being both a contributor and consequence of placental stress and maternal vascular susceptibility, potentially amplifying endothelial injury and systemic inflammation.[8,9]

Environmental exposures may add another layer of risk. Epidemiological evidence is mounting that exposure to fine particulate matter (PM2.5) and other forms of air pollution are associated with gestational hypertension and preeclampsia. Possible mechanisms include oxidative stress, systemic inflammation, endothelial dysfunction and changes in placental vascular function. Chronic psychosocial stress, poor nutrition and socioeconomic disadvantage may similarly influence risk thru interrelated metabolic, inflammatory and behavioural pathways. However, associations of environmental exposures with HDP are susceptible to residual confounding and variation

in exposure assessment. These factors should now be regarded as potential modifiers of disease susceptibility rather than established independent molecular causes.[10]

A major implication of the current evidence is that HDP cannot be fully explained by placental ischemia, angiogenic imbalance or endothelial dysfunction alone. These mechanisms seem to be interrelated parts of a larger disease network. Placental abnormalities may initiate or enhance circulating signals, while maternal cardiovascular and metabolic attributes determine the degree to which these signals translate to clinical disease. This interaction may be further modulated by genetic and epigenetic factors, immune response and environmental exposures. Therefore, the resulting phenotype can vary greatly between women, even with apparently similar clinical diagnoses.

More promising may be an approach combining maternal characteristics with angiogenic biomarkers, placental and cell free nucleic acids, metabolomic and proteomic signatures and longitudinal cardiovascular measurements. Integration of these multidimensional data may be possible using artificial intelligence and machine-learning approaches, but their clinical utility will depend on external validation, interpretability, reproducibility and demonstration of improved patient outcomes rather than prediction accuracy alone.[11]

CLASSIFICATION OF HYPERTENSION IN PREGNANCY (RECENT EVIDENCE-BASED UPDATE)

HDP are a heterogeneous group of conditions with different timing, underlying mechanisms, clinical severity, and maternal–fetal consequences. The present classification encompasses chronic hypertension, gestational hypertension, preeclampsia, and chronic hypertension with superimposed preeclampsia.

Eclampsia and HELLP syndrome are severe hypertensive disease; they are not entirely different pathophysiological entities. This distinction is clinically relevant as the risk associated with HDP is not only related to the degree of blood-pressure elevation but also to the presence of placental dysfunction and maternal organ involvement.[10]

Chronic hypertension

Chronic hypertension is defined as hypertension diagnosed prior to pregnancy, before 20 weeks of gestation, or continuing beyond the postpartum period. It reflects pre-existing maternal vascular or cardiometabolic disease and is increasingly encountered with increasing maternal age, obesity and metabolic disorders. Chronic hypertension per se increases risk of pregnancy, but superimposed preeclampsia markedly increases risk of placental abruption, fetal growth restriction, preterm birth and maternal organ dysfunction.

Therefore, it is clinically more useful to identify women with chronic hypertension who will subsequently develop placental disease than to treat all women with chronic hypertension as if they are equally at risk.[12-14]

Gestational hypertension

Gestational hypertension is defined as new-onset blood pressure of ≥140/90 mmHg after 20 weeks of gestation in the absence of proteinuria or other evidence of maternal organ dysfunction. It is often regarded as a milder phenotype than preeclampsia; but this should not lead to complacency. Women with gestational hypertension may develop preeclampsia, especially if the hypertension develops early or is associated with other maternal or placental risk factors.[14,15]The biological basis of gestational hypertension is heterogeneous as well. Contributory factors may include maternal cardiovascular reserve, pre-pregnancy blood pressure, obesity and metabolic status and the normal haemodynamic adaptation to pregnancy. Thus, in some women, gestational hypertension may be largely maternal vascular phenotype or an early manifestation of placental disease.[16-18]

Preeclampsia

Preeclampsia is defined as new onset hypertension after 20 weeks of gestation with proteinuria and/or maternal organ dysfunction which may involve the kidneys, liver, haematological system, nervous system or uteroplacental system. It should be regarded as a clinical syndrome with multiple biological pathways rather than as a single disease mechanism. The relative contribution of abnormal placentation, impaired spiral artery remodelling, angiogenic imbalance, endothelial dysfunction, inflammation, and maternal cardiovascular susceptibility differ among patients.[19,20] Early-onset disease is more commonly associated with severe placental dysfunction and foetal growth restriction. Maternal metabolic and cardiovascular vulnerability may be more important in the pathogenesis of late-onset disease. However, these phenotypes overlap and the distinction should not be taken as absolute. Emerging metabolomic, proteomic and cell-free nucleic acid studies suggest that molecular abnormalities may precede clinical disease.[21]

These results are promising as they could lead to better early risk stratification, but most are investigational and need external validation before routine clinical use. Similarly, long-term observational data have indicated an association between preeclampsia and increased maternal cardiovascular and cerebrovascular risk later in life. This lends support to the concept of preeclampsia as an important early marker of future maternal vascular disease rather than an event limited to pregnancy.[22,23]

Superimposed preeclampsia on chronic hypertension

Superimposed preeclampsia occurs in women with chronic hypertension who develop new proteinuria, worsening

hypertension with evidence of maternal organ dysfunction, or other features consistent with preeclampsia after 20 weeks. It results from the interaction between pre-existing maternal vascular disease and pregnancy-induced placental dysfunction and is associated with a particularly high maternal and fetal risk.[24]

Women with superimposed preeclampsia are at increased risk of severe maternal complications, medically indicated preterm birth, fetal growth restriction, and placental complications compared with women with chronic hypertension alone. Biomarkers like PlGF and the sFlt-1/PlGF ratio may help differentiate between superimposed preeclampsia and worsening chronic hypertension, particularly when baseline proteinuria or renal disease complicates standard clinical assessment. Biomarkers should be used, however, to complement rather than replace clinical evaluation.[25]

Individual assessment of maternal blood pressure, symptoms, laboratory abnormalities, fetal growth and placental function is required by management. Management is based on antihypertensive treatment, magnesium sulphate when indicated, corticosteroids when preterm delivery is anticipated and appropriate timing of delivery. For established preeclampsia the treatment of choice is delivery, with the timing a balance between maternal safety and fetal maturity.[26,27]

Eclampsia and HELLP syndrome

Eclampsia and HELLP syndrome are severe complications of HDP and are linked to significant maternal and perinatal morbidity. Eclampsia is the occurrence of new-onset generalized seizures in a woman with preeclampsia or pregnancy-related hypertension after other neurologic causes have been excluded.[28,29]HELLP syndrome (haemolysis, elevated liver enzymes, and thrombocytopenia) is a severe manifestation of systemic endothelial and microvascular damage closely associated with preeclampsia. Recent evidence indicates that these conditions are overlapping clinical phenotypes within the HDP spectrum rather than completely distinct disorders.[30,31]However, not all patients with preeclampsia/eclampsia will develop HELLP and the underlying biological mechanisms and clinical trajectories may be different. This differentiation is important because early recognition of severe features may allow for early intervention.

This difference is important because early recognition of severe features may be possible before irreversible maternal or fetal complications occur and thus timely intervention can be instituted. A recent systematic review and meta-analysis estimated the global prevalence of eclampsia and HELLP syndrome to be around 0.43% and 0.39% of pregnancies, respectively, with considerable geographical variation. These differences may reflect variation in maternal risk profiles, diagnostic practices, referral systems, and access to antenatal and emergency obstetric care rather than true differences in disease biology alone. Clinical studies further show the severity of HELLP syndrome. A multicentre study in Indonesia of 1,808 women with preeclampsia showed 12.1% developed HELLP. Women with HELLP had increased risk of acute kidney injury, eclampsia, preterm birth, fetal death, neonatal sepsis and maternal and neonatal mortality.[32,33]

The ongoing burden of eclampsia and HELLP, especially in resource-poor settings, suggests that successful management of HDP is about more than blood-pressure control. Early recognition of severe disease, standard diagnostic criteria, timely referral and coordinated maternal-fetal management are essential to reduce adverse outcomes. Severe maternal complications may require a medically indicated preterm delivery, with a rise in neonatal morbidity. Furthermore, recent evidence suggests that in utero exposure to HDP may have effects on the cardiovascular, metabolic and neurodevelopmental health of the offspring later in life. So HDP should not be looked at only as an immediate obstetrical complication, but also as a condition with possible sequelae after the delivery time. Integrated maternal and fetal surveillance with appropriate postpartum and long-term follow-up may therefore be critical for immediate and long-term disease burden reduction.[29,31]

DIAGNOSIS

Correct diagnosis of HDP is essential for identifying women at risk of maternal and fetal complications and determining the need for timely intervention. Diagnosis relies mainly on standardized measurement of blood pressure, assessment of proteinuria and assessment of maternal organ function, and clinical assessment. Biomarkers and imaging provide additional information when appropriate. Hypertension is defined as systolic blood pressure (SBP)≥140 mmHg or diastolic blood pressure (DBP) ≥90 mmHg, confirmed in repeated measurement after 20 weeks of gestation. Severe-range hypertension (SBP ≥160 mmHg or DBP ≥110 mmHg) is considered an emergency and treated as such.[35]

Gestational hypertension is newly diagnosed hypertension without proteinuria or another maternal organ dysfunction. Preeclampsia is diagnosed when hypertension is accompanied by proteinuria and/or maternal organ dysfunction involving the renal, hepatic, haematological, neurological, or other systems.[35,36]

Superimposed preeclampsia is the appearance of features of preeclampsia in women with chronic hypertension at the time of pregnancy. Eclampsia is defined as the occurrence of new-onset seizures in the absence of another identifiable cause, whereas HELLP syndrome is defined as haemolysis, elevated liver enzymes, and thrombocytopenia.[37-39] Laboratory investigations like complete blood count, platelet count, liver and renal function tests, LDH and assessment of urinary protein are

of particular importance in the identification of severe disease and organ involvement.

Biomarkers, like the sFlt-1/PlGF ratio, can enhance risk stratification in women with suspected preeclampsia, especially when clinical findings are inconclusive. Uterine artery Doppler may add additional information about placental perfusion but its diagnostic utility depends on gestational age and clinical setting.[40,41] Importantly these should supplement and not replace clinical assessment. Persistent headache, visual changes, epigastric or right upper-quadrant pain, and decreased fetal movements should be evaluated immediately. Diagnosis should generally be viewed as a dynamic process of repeated assessment of mother and fetus, not one measurement of blood pressure or laboratory result. Early recognition of severe features allows appropriate surveillance and timely intervention that may prevent progression to eclampsia, HELLP syndrome, fetal growth restriction and adverse perinatal outcomes.[35,42]

MATERNAL AND FETAL COMPLICATIONS

HDP can affect multiple maternal and fetal organ systems and outcomes are largely determined by severity, gestational age at onset, duration of disease and maternal comorbidities. The major pathways are placental insufficiency, maternal endothelial dysfunction and impaired uteroplacental perfusion; Of note, the consequences of HDP are not limited to the time of delivery, with a growing number of studies documenting long-term health consequences for both mothers and their children.[43,44]

Maternal complications

Maternal complications include severe hypertension, preeclampsia, eclampsia, HELLP syndrome, acute kidney injury, pulmonary edema, hepatic dysfunction, stroke, disseminated intravascular coagulation and multi-organ failure. The risk is especially high with early onset and severe disease. HELLP syndrome is a severe phenotype associated with increased maternal morbidity including renal failure, hepatic complications, haemorrhage, admission to intensive care unit and maternal death.

Clinicians care about when a disease starts. Early-onset preeclampsia, particularly when it occurs before 34 weeks of gestation, is associated generally with increased maternal and fetal morbidity compared with late-onset disease, although late-onset disease can also lead to serious complications.[36] Beside pregnancy, HDP is increasingly being recognized as a marker of future maternal health. Women with a history of HDP are at risk for chronic hypertension, CVD, stroke, heart failure, and chronic kidney disease. Thus, pregnancy represents a window of opportunity to identify women who may benefit from long-term cardiovascular risk assessment and preventive care.

Fetal complications

Fetal complications are mainly due to placental dysfunction and impaired uteroplacental perfusion, while neonatal morbidity is also affected by the gestational age at delivery. Common complications are fetal growth restriction, low birth weight, oligohydramnios, abnormal Doppler findings, preterm birth, stillbirth and increased perinatal mortality. These risks are especially high in early-onset preeclampsia and severe disease. Preterm delivery is a significant cause of neonatal morbidity and is often medically indicated when the continued course of pregnancy poses greater risk to the mother or fetus than delivery. Consequently, neonates may develop respiratory distress, intraventricular haemorrhage, feeding difficulties, sepsis, and increased NICU care requirements. It is, therefore, important to differentiate between complications directly attributable to HDP and related complications of prematurity in interpreting neonatal outcomes.[34,36]

Emerging longitudinal evidence also suggests an association between in utero exposure to HDP and elevated blood pressure, poor cardio-metabolic profiles and cardiovascular risk in later life, although the extent of the associations varies across studies and may be confounded by shared maternal and familial factors. Therefore, in our opinion, the clinical impact of HDP should not be evaluated only on the basis of short-term maternal or neonatal outcomes. The combination of severe maternal disease, medically indicated preterm birth, placental insufficiency and potential long-term offspring effects highlights the need for integrated maternal-fetal surveillance and appropriate postpartum follow-up. Early identification and tailored management may mitigate acute morbidity but also represent an opportunity to address the longer-term health sequelae of HDP.[37]

LONG TERM OUTCOMES

HDP are not only complications of pregnancy but also early markers of future maternal and offspring health risks. Women who have had gestational hypertension or pre-eclampsia are at increased risk of developing chronic hypertension, cardiovascular disease, heart failure, stroke, renal disease and adverse metabolic profiles subsequently. The risk appears higher for women with early-onset, severe or recurrent preeclampsia, although these associations may partly reflect cardiometabolic susceptibility present before the preeclampsia. Thus, HDP may provide an important opportunity for early identification of women who may benefit from cardiovascular and renal risk assessment after pregnancy. There is emerging evidence on longer-term neurological outcomes, but associations with migraine, epilepsy and sleep disorders are less well established than cardiovascular outcomes.[34]

The potential consequences for offspring are also increasingly being recognized. Exposure to HDP in utero

might be linked to higher blood pressure and less favourable cardiometabolic profiles in childhood and adolescence. Some studies have also reported associations with neurodevelopmental and cognitive outcomes, although findings are heterogeneous and may be influenced by prematurity, fetal growth restriction, maternal characteristics and other confounding factors. The balance of evidence supports HDP as a marker of long-term maternal cardiovascular risk in our view, whereas evidence for specific neurological and offspring outcomes is an evolving area of research. Therefore, postpartum transition to long-term preventive care should include monitoring blood pressure, assessment of cardiovascular and metabolic risk factors, and appropriate renal evaluation.[36]

CONCLUSION

HDP is a heterogeneous maternal-placental disorder where abnormal placentation, maternal vascular susceptibility, angiogenic imbalance, inflammation, and endothelial dysfunction interact to cause clinical disease. Early detection and appropriate management are crucial because severe phenotypes such as eclampsia and HELLP syndrome can rapidly develop maternal and foetal complications. The current diagnostic approaches are mainly based on blood pressure, clinical assessment and laboratory evaluation. The advances in angiogenic biomarkers and other molecular markers may improve risk stratification and identification of women likely to develop severe disease. But these approaches need to be further validated in diverse populations before they are widely implemented. In our opinion, the clinical importance of HDP goes beyond pregnancy itself. Thus, effective management should shift from a pregnancy-limited approach to an integrated maternal-fetal and life-course strategy that combines early diagnosis, individualised surveillance, timely delivery when indicated, and postpartum cardiovascular risk reduction. Further prospective studies are required to elucidate which biomarkers and biological pathways can optimally predict disease progression and long-term outcomes in mothers and their offspring.

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