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Hypertensive Disorders of Pregnancy: Risk Stratification, Prevention and Evidence-Based Management

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Abstract

Hypertensive disorders of pregnancy remain a major cause of maternal and perinatal morbidity and mortality and include chronic hypertension, gestational hypertension, pre-eclampsia, and pre-eclampsia superimposed on chronic hypertension. This narrative review synthesizes evidence available through 30 June 2021 regarding classification, pathophysiology, prediction, prevention, acute treatment, timing of delivery, and post-partum follow-up. Contemporary diagnostic frameworks emphasize new-onset hypertension after 20 weeks of gestation and recognize that pre-eclampsia can be diagnosed in the absence of proteinuria when specific maternal organ dysfunction or uteroplacental dysfunction is present. Abnormal placentation, endothelial injury, and an imbalance between pro-angiogenic and anti-angiogenic factors provide a biologically plausible model linking placental disease to the maternal syndrome. First-trimester multimarker screening can identify a substantial proportion of pregnancies destined to develop preterm pre-eclampsia, although availability and implementation vary. Low-dose aspirin for appropriately selected high-risk women and calcium supplementation in populations with low dietary calcium intake are the most consistently supported preventive strategies. Severe hypertension requires prompt treatment, magnesium sulfate remains the standard anticonvulsant for eclampsia prevention and treatment, and definitive management of pre-eclampsia ultimately requires delivery, with timing individualized by gestational age, disease severity, and maternal-fetal status. Pregnancy complicated by hypertension should also be regarded as a marker of future cardiovascular risk, making structured post-partum surveillance clinically important.

Keywords: pre-eclampsia; gestational hypertension; chronic hypertension; pregnancy; aspirin; magnesium sulfate; maternal cardiovascular risk

1. Introduction

Hypertension in pregnancy spans a spectrum from chronic hypertension that predates conception to a placenta-driven multisystem syndrome that may evolve rapidly. Pre-eclampsia complicates an estimated 2-8% of pregnancies globally and, together with other hypertensive disorders, contributes substantially to severe maternal morbidity, medically indicated preterm birth, fetal growth restriction, placental abruption, stroke, and maternal death [1-5]. Although mortality is concentrated in settings with delayed diagnosis or limited access to emergency obstetric care, hypertensive disorders remain an important cause of preventable complications in all health systems.

The clinical challenge is twofold. First, the syndrome is heterogeneous: some patients develop predominantly maternal hypertension and laboratory abnormalities, whereas others manifest severe placental insufficiency, fetal growth restriction, or early-onset disease. Second, the trajectory can change within hours. Contemporary guidance therefore prioritizes standardized blood-pressure measurement, recognition of end-organ manifestations, repeated maternal laboratory evaluation, and fetal surveillance rather than reliance on proteinuria alone [1,2].

This review summarizes major evidence available by the end of June 2021, with emphasis on practical risk stratification, prevention, management, and long-term implications.

2. Methods

A focused narrative review was undertaken using PubMed/MEDLINE-indexed literature and authoritative professional guidance. The search emphasized the terms pre-eclampsia, hypertensive disorders of pregnancy, gestational hypertension, chronic hypertension, aspirin, calcium, magnesium sulfate, angiogenic factors, uterine artery Doppler, antihypertensive treatment, and timing of delivery. Priority was given to major practice guidelines, randomized

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controlled trials, systematic reviews/meta-analyses, and influential mechanistic or cohort studies published on or before 30 June 2021. Publications after the cutoff were not used in the synthesis.

3. Classification and Diagnostic Framework

Most contemporary frameworks classify hypertensive disease in pregnancy as chronic hypertension, gestational hypertension, pre-eclampsia, or pre-eclampsia superimposed on chronic hypertension [1,2]. Chronic hypertension is present before pregnancy, before 20 weeks of gestation, or persists beyond the expected post-partum resolution period. Gestational hypertension develops after 20 weeks without diagnostic features of pre-eclampsia. Pre-eclampsia is characterized by hypertension after 20 weeks together with proteinuria and/or specific maternal organ dysfunction; severe-range blood pressure itself represents a high-risk feature requiring urgent assessment [1,2].

The diagnostic shift away from mandatory proteinuria is clinically important. Thrombocytopenia, renal insufficiency, impaired liver function, pulmonary edema, persistent neurologic symptoms, or other evidence of significant organ involvement may establish the diagnosis in a hypertensive pregnant patient even when urinary protein is absent [1]. Uteroplacental dysfunction, including fetal growth restriction, also contributes to risk assessment in international frameworks [2].

Table 1. Practical classification of hypertensive disorders of pregnancy [1,2].

Category	Core concept	Clinical priority
Chronic hypertension	Hypertension predating pregnancy or recognized before 20 weeks; may persist post-partum	Baseline renal/protein assessment; superimposed pre-eclampsia risk
Gestational hypertension	New hypertension after 20 weeks without pre-eclampsia features	Serial symptoms, labs, fetal growth and progression monitoring
Pre-eclampsia	Hypertension after 20 weeks with proteinuria and/or maternal organ dysfunction	Severity assessment, fetal surveillance, delivery planning
Superimposed pre-eclampsia	New pre-eclampsia features in a patient with chronic hypertension	Higher maternal-fetal risk; distinguish from worsening chronic hypertension

4. Pathophysiology and Risk Prediction

Pre-eclampsia is best understood as a syndrome arising from abnormal placental development interacting with maternal cardiovascular, metabolic, immunologic, and genetic susceptibility. In early-onset disease, impaired remodeling of the spiral arteries can lead to high-resistance uteroplacental flow, placental ischemia-reperfusion injury, oxidative stress, and release of circulating factors that induce widespread endothelial dysfunction [4,5].

The angiogenic model has provided a major mechanistic advance. Prospective studies showed that circulating soluble fms-like tyrosine kinase-1 (sFlt-1) rises and placental growth factor (PlGF) falls before clinical pre-eclampsia, while soluble endoglin is also increased in severe disease [9,10]. These observations support the concept that an anti-angiogenic state contributes to hypertension, glomerular endothelial injury, hepatic dysfunction, neurologic manifestations, and other maternal features.

Prediction based on a single risk factor or single laboratory test is limited. Multivariable first-trimester approaches combine maternal characteristics with mean arterial pressure, uterine artery pulsatility index, and placental biomarkers such as PlGF. In a large prospective dataset, a combined algorithm predicted a high proportion of early and preterm pre-eclampsia at a 10% screen-positive rate, with lower performance for term disease [7]. Uterine artery Doppler alone has moderate predictive ability and performs better for severe or early disease than for the broad population [8].

5. Prevention in Women at Increased Risk

Low-dose aspirin is the best studied pharmacologic preventive intervention. Individual-patient meta-analysis demonstrated a modest reduction in pre-eclampsia with antiplatelet therapy [12], while meta-analytic data suggested greater benefit when aspirin is started early in pregnancy [13]. The ASPRE randomized trial used 150 mg nightly from

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11-14 weeks until 36 weeks in women identified as high risk by a multimarker algorithm and reported a lower incidence of preterm pre-eclampsia than placebo [11]. Dose and eligibility recommendations vary across organizations, but the evidence available by 2021 consistently favored early initiation in appropriately selected high-risk patients rather than universal treatment.

Calcium supplementation has a different evidence base. Trials and systematic reviews indicate that supplementation reduces hypertensive disorders most clearly in populations with low baseline calcium intake and in women at elevated risk [14]. The World Health Organization therefore recommended calcium supplementation during pregnancy in settings where dietary calcium intake is low, and also supported low-dose aspirin for women at high risk [15].

Preventive care also includes optimization of chronic hypertension and pre-existing diabetes, smoking avoidance, appropriate weight and activity counseling, and early antenatal engagement. These measures are not substitutes for aspirin or calcium when indicated, but they address modifiable contributors to overall obstetric and cardiovascular risk.

6. Clinical Management

6.1 Initial assessment and surveillance

New hypertension after 20 weeks warrants confirmation with appropriate cuff size and technique, symptom review, assessment for severe-range values, complete blood count with platelets, serum creatinine, liver enzymes, and evaluation for proteinuria. Fetal assessment is guided by gestational age and severity and commonly includes growth assessment, amniotic fluid evaluation, and antenatal surveillance when expectant management is chosen [1,2].

Warning symptoms such as persistent severe headache, visual disturbance, epigastric or right-upper-quadrant pain, dyspnea, or altered mental status require urgent evaluation. Likewise, fetal growth restriction, abnormal fetal testing, placental abruption, or persistent severe hypertension can shift management toward delivery.

6.2 Antihypertensive therapy

Acute severe hypertension should be treated promptly to reduce maternal risk, particularly hemorrhagic stroke. Commonly recommended agents include intravenous labetalol, intravenous hydralazine, or immediate-release oral nifedipine, selected according to contraindications, clinician experience, and local availability [1,15]. For ongoing control of non-severe hypertension, labetalol, extended-release nifedipine, and methyldopa have long obstetric experience. Angiotensin-converting enzyme inhibitors and angiotensin receptor blockers are generally avoided during pregnancy because of fetal toxicity. The CHIPS randomized trial showed that tighter blood-pressure control reduced severe maternal hypertension without worsening the primary perinatal outcome, helping to frame the balance between maternal protection and concerns about excessive blood-pressure lowering [17].

6.3 Seizure prevention and treatment

Magnesium sulfate remains the treatment of choice for eclampsia and is widely recommended for seizure prophylaxis in pre-eclampsia with severe features. The Magpie Trial randomized more than 10,000 women with pre-eclampsia and found that magnesium sulfate approximately halved the risk of eclampsia compared with placebo, establishing the evidence base for modern practice [16]. Clinical use requires attention to respiratory status, reflexes, urine output, and renal function because magnesium is renally cleared.

6.4 Timing of delivery

Delivery is the definitive treatment for pre-eclampsia because the placenta is central to disease pathogenesis. Timing depends on gestational age, severity, maternal stability, fetal condition, and the resources available for neonatal care. At term, randomized evidence from HYPITAT supported induction rather than prolonged expectant management for gestational hypertension or mild pre-eclampsia after 36 weeks, reducing poor maternal outcomes without an increase in cesarean delivery [18].

For late preterm pre-eclampsia, the PHOENIX trial found that planned earlier delivery from 34 to less than 37 weeks reduced maternal morbidity/severe hypertension but increased neonatal-unit admission, illustrating the trade-off between maternal benefit and short-term neonatal consequences [19]. Before these gestations, carefully selected stable patients

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may be managed expectantly in an appropriately resourced setting, but maternal or fetal deterioration requires delivery irrespective of gestational age [1,2].

Table 2. Selected evidence-based management priorities available by June 2021.

Clinical situation	Evidence-based priority	Key sources
Severe-range blood pressure	Prompt antihypertensive treatment and evaluation for severe features	[1,15,17]
Pre-eclampsia with severe features	Magnesium sulfate when indicated; maternal-fetal stabilization; delivery planning	[1,16]
Gestational hypertension / pre-eclampsia at term	Delivery is generally favored rather than prolonged expectant management	[1,18]
Late preterm pre-eclampsia	Balance maternal benefit of planned delivery against neonatal prematurity burden	[19]
Low dietary calcium setting / high-risk patient	Consider evidence-based prevention with calcium and/or low-dose aspirin as appropriate	[11-15]

7. Post-partum Care and Long-Term Cardiovascular Health

Hypertension and complications can persist or first become apparent after delivery. Early post-partum blood-pressure surveillance, symptom education, medication adjustment, and assessment for persistent hypertension are therefore essential. Breastfeeding compatibility should be considered when selecting treatment.

A hypertensive pregnancy is also a cardiovascular risk marker rather than an isolated obstetric event. Large cohort data and systematic evidence available before July 2021 demonstrated higher subsequent risks of chronic hypertension and cardiovascular disease among women with prior pre-eclampsia or other hypertensive disorders [20,21]. The association is strongest after recurrent, severe, or preterm disease. Post-partum transition to primary care should include counseling regarding blood pressure, body weight, smoking, glucose and lipid risk factors, and the importance of long-term cardiovascular prevention.

8. Discussion

The evidence base available by mid-2021 supports a shift from a narrow, proteinuria-centered concept of pre-eclampsia toward a dynamic multisystem disorder requiring repeated risk assessment. Major advances include recognition of angiogenic dysfunction, improved first-trimester risk models, evidence for early aspirin in selected high-risk women, strong randomized evidence for magnesium sulfate, and clearer delivery strategies at term and late preterm gestations.

Important uncertainties remained. Risk-prediction algorithms were not uniformly accessible, optimal aspirin dose varied among guidelines, and the best blood-pressure target for different phenotypes was still debated. Equally important, much of the preventable global burden reflected system-level delays in diagnosis, referral, antihypertensive treatment, seizure prophylaxis, blood products, and timely delivery rather than lack of scientific knowledge. Therefore, protocolized care and reliable escalation pathways are as important as sophisticated biomarkers.

For clinicians, the practical principle is to combine standardized diagnosis with anticipation: identify baseline risk early, institute validated prevention when indicated, recognize progression before irreversible organ injury, stabilize severe hypertension and neurologic risk, and plan delivery in a gestational-age-specific manner.

9. Conclusion

Hypertensive disorders of pregnancy require early risk recognition, precise classification, rapid treatment of severe hypertension, appropriate use of magnesium sulfate, and gestational-age-specific delivery planning. Evidence available through June 2021 supports low-dose aspirin for selected high-risk women and calcium supplementation in low-intake populations, while multimarker screening offers a promising route to targeted prevention. Because pre-eclampsia also

predicts later cardiovascular disease, care should extend beyond delivery into structured post-partum and long-term risk reduction.

Declarations

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Data availability: No new dataset was generated for this review.

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