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Hypertensive disorders of pregnancy: A review and updates for the emergency physician

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ABSTRACT

Hypertensive disorders of pregnancy (HDPs) are a spectrum of disease contributing to immediate and lasting morbidity and mortality in pregnant patients and neonates globally. With decreasing access to obstetric care in the United States, an expected increase in patients will seek care in emergency departments. Consequently, emergency physicians will likely assume a greater share of initial care and resuscitation of patients with HDPs. HDPs represent a spectrum of disease including gestational hypertension; chronic hypertension in pregnancy; pre-eclampsia; superimposed pre-eclampsia; Hemolysis, Elevated Liver enzymes and Low Platelets (HELLP) syndrome; and eclampsia.

This is a review of the current literature and guidelines of HDPs, including updated HDP diagnostic criteria, diagnostic testing, and initial management. Given the growing understanding of lasting cardiovascular effects of HDPs, emergency physicians should carefully consider the long-term impacts of HDPs, even after delivery. Appropriate recognition, treatment, and coordination of care are essential to optimizing care of pregnant patients who present to the emergency department.

1. Introduction

Worldwide, nearly 40% of deaths in pregnant patients are caused by hypertensive disorders of pregnancy (HDPs), with approximately 16% of all deaths in pregnant patients globally attributed directly to HDPs and an additional 23% caused by indirect causes of the disease (including pre-existing hypertension) [1]. HDPs affect 5–10% of all pregnancies, and they are the greatest contributor to pregnant patient morbidity and mortality globally with prevalence increasing over time [2]. In addition to causing morbidity and mortality, there is substantial evidence that HDPs contribute significantly to poor fetal outcomes, including preterm birth and neonatal mortality. Long-lasting health detriments also remain for patients diagnosed with HDPs, even decades after delivery [2-4].

In the United States, increasingly less hospitals are staffed with obstetric specialists. As of 2022, over 40% of U.S. hospitals had no obstetric care, with a discrepancy in coverage between rural locations, where over 50% of hospitals have no obstetric care. In comparison, approximately 35% of rural hospitals are without obstetrics. The trend of limited obstetric care in hospitals—especially in rural areas—suggests that increasingly more patients with HDPs will continue to present to the emergency department directly [5].

In light of limited obstetric services, emergency providers must be ready to recognize, evaluate, and effectively manage patients with HDPs. Timeliness of recognition and treatment is a critical aspect of preventing lasting organ damage, disability, and death. Emergency providers should aim to initiate treatment within 60 min—a goal that has long proven challenging for those caring for patients with HDPs [6] In obstetrics, around half of patients are not initiated on treatment within this time frame. In the emergency department, where HDPs are much less frequently seen, recognition may be even more challenging—leading to significant, life-threatening delays in treatment [6].

As HDPs are a broad spectrum of disorders that can be challenging to recognize and treat, this review serves to examine recent literature and practice guidelines to advise emergency medicine providers on how to approach HDPs.

Noteworthy updates include

- discussion of the role of proteinuria in diagnosing preeclamptic disorders,
- clarification on qualifications for severe preeclampsia,
- when magnesium is warranted, and

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- long-term cardiovascular adverse effects emergency medicine providers should consider when evaluating a patient with a history of HDPs.

This review began in PubMed with the terms (((((((pregnancy) OR (postpartum)) AND (hypertension)) OR (preeclampsia)) OR (HELLP)) OR (eclampsia)) OR (gestational hypertension)) NOT (genetics)) NOT (biomarker) with the search parameters "Human studies, English, Guideline, Meta-Analysis, Multicenter Study, Network Meta-Analysis, Observational Study, Randomized Controlled Trial, Systematic Review". The search was focused on Feb 2021 to Feb 2026. Search results yielded 1441 studies; 153 of these studies made it to the full text screen. A total of 19 articles from the original search were used to create this review, excluding articles not relevant to the emergency physician. An additional 21 articles and practice bulletins were added to the original search.

2. Pathophysiology & risk factors

The pathophysiology of preeclampsia/eclampsia is not fully understood; it is thought to be a complex and multifactorial process. Uteroplacental dysfunction is presumed to be a primary underlying cause, with failure of normal placental development leading to ischemia, oxidative stress, and inflammation. This ultimately results in hypertension and organ damage [7].

There are several risk factors associated with the development of HDPs. These include chronic hypertension, diabetes, obesity, renal disease, history of autoimmune disorders, and history of drug use [8,9]. Patient factors include both younger age (<25 years) and advanced age (>35 years), nulliparity, the use of reproductive therapy, and multigestation [4,9,10]. There is also a notable racial disparity with HDPs. The highest incidence is seen in patients who are Black, followed by those who are Native American and Hawaiian/Pacific Islander [11]. A prior history of gestational hypertension or preeclampsia/eclampsia is also a significant risk factor, to an extent that a history of prior preeclampsia increases the risk for recurrence nearly four-fold in subsequent pregnancies [9,12,13].

3. Spectrum of hypertension in pregnancy: what are the different types of HDPs?

HDPs can be separated into three broad categories: chronic hypertension, gestational hypertension, and preeclampsia/eclampsia. It should be recognized that these disorders exist on an overlapping spectrum. The diagnostic criteria for each of the HDPs as described below are outlined in Table 1.

3.1. Chronic hypertension

Individuals who are pregnant who have pre-existing hypertension or hypertension that is diagnosed within the first 20 weeks have chronic hypertension in pregnancy (CHTN). A diagnosis within those first 20 weeks suggests that the hypertension existed pre-pregnancy [14]. The diagnosis can be made with blood pressures of ≥140 mmHg systolic and/or ≥ 90 mmHg diastolic, with two or more measurements taken at least four hours apart.

Affecting around 2% of pregnancies, CHTN co-exists with pregnancy, as opposed to resulting from pregnancy itself, like other HDPs [15]. CHTN has increased in prevalence over the past several decades—a trend that has been attributed to both increasing age of pregnant individuals, as well as increasing prevalence of obesity and metabolic syndrome. This increase is also likely related to an improvement in detection. Mirroring the broader pattern of diseases on the HDP spectrum, CHTN disproportionately affects Black patients, affecting as many as 5% of pregnancies within that racial group. CHTN also disproportionately affects Native American and Hawaiian/Pacific Islander origin patients as well [11].

Table 1
Diagnosing hypertensive disorders of pregnancy.

Chronic Hypertension	Gestational Hypertension	Preeclampsia without Severe Features	Preeclampsia with Severe Features	HELLP	Eclampsia
HTN diagnosed <20 weeks gestation or pre-pregnancy *with* Elevated Blood Pressure Systolic BP ≥140 *or* Diastolic BP ≥90 *Without proteinuria or signs of end organ damage*	HTN diagnosed ≥20 weeks gestation *with* Elevated Blood Pressure Systolic BP ≥140 *or* Diastolic BP ≥ 90 *Without proteinuria or signs of end organ damage*	≥20 weeks gestation to 6 weeks postpartum *with* Elevated Blood Pressure Systolic BP ≥140 *or* Diastolic BP ≥ 90 *plus* Proteinuria • Protein to creatinine ratio of ≥0.3 • 2+ protein • ≥0.3 g protein/24 h *Without signs of end organ damage*	≥20 weeks gestation to 6 weeks postpartum *with* Severe Range Blood Pressure Systolic BP ≥ 160 *or* Diastolic BP ≥110 *or* Elevated Blood Pressure Systolic BP ≥ 140 *or* Diastolic BP ≥ 90 *plus* Signs of end organ damage • Platelets <100,000 • LFTs >2× upper limit of normal • Creatinine >1.1 or 2× baseline • Pulmonary edema • Headache, vision changes, mental status changes • Severe right upper quadrant or epigastric pain	≥20 weeks gestation to 6 weeks postpartum *with or without* Elevated Blood Pressure Systolic BP ≥140 *or* Diastolic BP ≥ 90 *plus* Criteria for HELLP • LDH >600 IU/L • AST and ALT >2 times the upper limit of normal • Platelets <100,000 × 10^9/L	≥20 weeks gestation to 6 weeks postpartum *with or without* Elevated Blood Pressure Systolic BP ≥140 *or* Diastolic BP ≥ 90 *plus* New onset seizure
Management					
Outpatient management; discontinue antihypertensives contraindicated in pregnancy	Outpatient management	Can consider outpatient management with obstetric consultation and close follow up versus inpatient management, delivery (>37 weeks gestation)	Urgent blood pressure management; seizure prophylaxis; urgent delivery	Urgent blood pressure management; seizure prophylaxis; urgent delivery	Urgent blood pressure management; seizure prophylaxis; urgent delivery

CHTN has a well-established association with adverse outcomes in both pregnant patients and neonates. This includes an overall five-fold increase in maternal mortality and five-fold risk for preeclampsia [15].

Patients with CHTN are also at a significantly increased risk of the following:

- developing HELLP (hemolysis, elevated liver enzymes, and low platelets) syndrome
- needing a cesarean section
- developing postpartum hemorrhage
- experiencing renal failure
- undergoing a cerebrovascular accident
- experiencing pulmonary edema [11,15]

Adverse neonatal outcomes associated with chronic hypertension include stillbirth, preterm delivery, and small for gestational age (SGA) neonates [15].

3.2. Gestational hypertension

Gestational hypertension (HTN) is hypertension that occurs during pregnancy and is thought to be a result of placental and cardiovascular changes that occur during pregnancy. Gestational HTN affects around 7% of pregnancies [16].

Gestational hypertension is diagnosed in individuals *without* preexisting chronic hypertension who are ≥20 weeks in gestation with an elevated blood pressure of ≥140 mmHg systolic and/or ≥ 90 mmHg diastolic noted on at least two occasions greater than 4 h apart. Notably, patients with a diagnosis of gestational HTN cannot have proteinuria, any signs of end-organ damage, or severely elevated blood pressures (systolic blood pressure ≥ 160 mmHg or diastolic blood pressures ≥110 mmHg), as this would indicate preeclampsia. Gestational HTN is expected to resolve by 12 weeks postpartum.

Gestational HTN is a significant risk factor for the development of subsequent adverse events and development of other HDPs, including preeclampsia and eclampsia. In fact, many experts prefer to think of gestational HTN and preeclampsia as a spectrum rather than distinct categories, given the frequency in which gestational HTN develops into preeclampsia, as well as the overlapping nature of these disease processes [8]. Patients with gestational HTN diagnosed before 32 weeks are even more likely to develop preeclampsia than those who develop gestational HTN later in pregnancy, with an estimated 50% of those with early gestational HTN developing preeclampsia [8].

3.3. Preeclampsia/preeclampsia with severe features

Preeclampsia is a disorder of pregnancy associated with new-onset hypertension occurring after 20 weeks of gestation and up to 6 weeks postpartum, with accompanying organ dysfunction. [8] Preeclampsia impacts approximately 5–7% of pregnancies worldwide and leads to complications for both the pregnant patient and the fetus/neonate [17].

The diagnostic criteria for preeclampsia are as follows [8]:

Elevated Blood Pressure

- Systolic blood pressure ≥ 140 mmHg or diastolic blood pressure ≥ 90 mmHg on two occasions at least 4 h apart.
  Systolic blood pressure ≥ 160 mmHg or Diastolic blood pressure ≥ 110 mmHg confirmed over a short time interval.
  **Blood pressures elevated to this extent qualify patients for the diagnosis of preeclampsia with severe features, with no further diagnostic criteria needed.*
  AND

Proteinuria and/or Signs of End Organ Damage

- Proteinuria
  - o 24 h urine collection with 300 mg or more protein noted;
  - o Urine protein/creatinine ratio of 0.3 mg/dL or more; or
  - o Urine dipstick result of 2+
    OR
- Signs of end organ damage:

**If present, diagnostic of preeclampsia with severe features.*

- o Thrombocytopenia (<100,000 × 10^9/L);
- o Renal insufficiency (serum creatinine >1.1 mg/dL, or a doubling of serum creatinine in absence of other kidney disease);
- o Impaired liver function (liver transaminases > twice normal concentration) or severe right upper quadrant/epigastric pain;
- o Pulmonary edema; or
- o New-onset headache and/or visual disturbances

Historically, the diagnosis of preeclampsia required the presence of proteinuria; however, updated diagnostic criteria do not require proteinuria in the presence of other signs of end-organ damage as listed above. Presence of any of these signs of end-organ damage also push the diagnosis from preeclampsia to preeclampsia with severe features; additionally, if a patient has elevated blood pressures in the severe range, they meet diagnostic criteria for pre-eclampsia with severe features, regardless of the presence of proteinuria or other biomarkers.

Emergency medicine physicians must maintain a high index of suspicion for preeclampsia in any pregnant patient or those of childbearing age who could be pregnant. This includes patients who are newly postpartum—a status that patients may not disclose unless prompted. Although the vast majority of the time, elevated blood pressures in non-pregnant individuals do not require emergent work up or treatment, any blood pressure in a pregnant or newly postpartum patient above 140/90 mmHg should prompt additional work up. Elevated readings in these patient populations should not simply be attributed to external factors like pain or chronic hypertension without further investigation. Additionally, since proteinuria is no longer required for the diagnosis, emergency physicians should not be falsely reassured by an absence of proteinuria without evaluating for other signs of end-organ damage.

Complications of preeclampsia include pulmonary edema, coagulopathy/disseminated intravascular coagulopathy (DIC), myocardial infarction, stroke, acute respiratory distress syndrome, renal failure, and multi-organ dysfunction. Early-onset preeclampsia (onset at <34 weeks) tends to be more severe and associated with fetal growth restriction, abnormal placentation, and systemic endothelial dysfunction.

3.4. HELLP syndrome

HELLP syndrome is a serious complication on the spectrum of severe preeclampsia and carries a high risk of morbidity and mortality. HELLP syndrome is a thrombotic microangiopathy leading to microthrombi, platelet dysfunction and consumption that leads to liver ischemia and thus its characteristic diagnostic findings.

The following criteria are used to make the diagnosis of HELLP syndrome [8]:

- Lactate dehydrogenase (LDH) >600 IU/L;
- Aspartate aminotransferase (AST) and alanine aminotransferase (ALT) > 2 times the upper limit of normal; or
- Platelet count <100,000 × 10^9/L.

Patients with HELLP syndrome can present with right upper quadrant or epigastric pain, general malaise, and nausea with vomiting. A high level of suspicion should be maintained for patients with preeclampsia who meet the above criterion. As with preeclampsia, HELLP syndrome can occur in the postpartum period, with around 1/3 of cases occurring *after* delivery. The absence of elevated blood pressure should not rule this out; up to 15% of those with HELLP syndrome lack elevated blood pressure or proteinuria [8]. HELLP syndrome and other thrombotic microangiopathies (such as thrombotic thrombocytopenic

purpura, hemolytic uremic syndrome, and antiphospholipid syndrome) all involve microangiopathic hemolytic anemia (MAHA). These diseases can overlap in clinical presentation/laboratory abnormalities. Emergency physicians must keep other thrombotic microangiopathies on the differential, since patients may still experience these serious diseases during pregnancy [18].

It is especially important for emergency physicians to differentiate between HELLP syndrome and thrombotic thrombocytopenic purpura (TTP)-Hemolytic Uremic Syndrome (HUS) as the management of these diseases differs. For TTP-HUS, the acute treatment involves plasmapheresis with steroids, followed by immunosuppressive agents. In contrast, the management of HELLP Syndrome involves antihypertensives and emergent delivery of the neonate. The most useful way to differentiate between HELLP Syndrome and TTP-HUS is lab testing for ADAMTS13 enzyme deficiency, a marker associated with TTP-HUS. Unfortunately, pregnancy can precipitate a decrease in ADAMTS13 activity, making TTP-HUS possible in this time frame as well. When patients have persistent hemolysis and worsening renal failure, often to the point of requiring dialysis or complement inhibition, physicians should consider a diagnosis of pregnancy-associated atypical hemolytic uremic syndrome (aHUS) [19].

Patients with HELLP syndrome experience high morbidity and mortality rates, with a mortality rate around 1–2%. A high proportion of those with HELLP syndrome subsequently develop co-morbid conditions such as disseminated intravascular coagulation (DIC), renal failure, placental abruption, and subcapsular hematoma/hepatic rupture [20].

3.5. Eclampsia

Eclampsia is a severe, life-threatening, end-stage condition within the spectrum of HDPs. It is characterized by new-onset tonic-clonic, focal, or multifocal seizures in the absence of other causative conditions. Like preeclampsia, eclampsia can occur in pregnancy as well as the postpartum.

The underlying pathophysiology of eclampsia is not fully known; however, it is thought to involve an alteration to normal cerebral blood flow autoregulation and disruption of the blood brain barrier. This disruption leads to cerebral edema, hemorrhage, ischemia, and necrosis. Although eclampsia characteristically involves elevated blood pressures, as many as 25% of those with eclampsia do not have elevated blood pressure and those with postpartum eclampsia are less likely to have severely elevated blood pressures than those who are antepartum, making the diagnosis even more challenging [21].

Eclampsia does not always follow a linear progression from preeclampsia, and many patients present atypically. Warning signs can often include persistent headache, visual disturbances, epigastric pain, and altered mental status [21]. However, we cannot rely on these preceding symptoms as a minority of patients can have abrupt onset eclampsia without warning signs. Additionally, 20–38% of women with eclampsia do not demonstrate classic signs of preeclampsia (such as hypertension or proteinuria) prior to having a seizure [22].

A severe and closely-related and occasionally overlapping clinical entity to preeclampsia/eclampsia is posterior reversible encephalopathy syndrome (PRES). Patients with PRES typically present with headaches, visual disturbances, acute mental status changes, and seizures. The diagnosis of PRES requires MRI findings which typically include reversible, symmetrical, posterior subcortical vasogenic edema [23].

Keeping a high index of suspicion in those of childbearing age with new onset seizures is critical; a new seizure in a pregnant patient or less than 6 weeks postpartum patient is eclampsia until proven otherwise. Most eclamptic seizures occur in the antepartum period, but approximately 20–30% can occur in the postpartum period [21]. In very rare cases, eclampsia can occur prior to 20 weeks gestation, often with molar pregnancy or severe underlying autoimmune/inflammatory conditions [21]. Similar to other HDPs, eclampsia that develops early, at <32 weeks gestation, is associated with worse outcomes than development later in pregnancy [21] Eclampsia places patients at high risk of hypoxia, trauma, aspiration, pneumonia, residual neurologic damage, or death.

3.6. Superimposed preeclampsia and overlapping features

HDPs are known as a spectrum due to the high amount of overlap in presentations. Anywhere from 20 to 50% of patients with CHTN will go on to develop preeclampsia (sometimes referred to as superimposed preeclampsia). Patients who are Black, obese, and those with higher baseline blood pressures being more likely to progress [14]. Superimposed preeclampsia can be especially challenging to diagnose. Physicians should note that 10% of patients with chronic hypertension can have proteinuria with no other co-occurring conditions; the presence of proteinuria does not definitively indicate superimposed preeclampsia [24]. Accordingly, there are no exact proteinuria or blood pressure levels for superimposed preeclampsia, though sudden increases in proteinuria or blood pressure should prompt additional workup and potential consultation with obstetrics [14,24]. When patients with CHTN begin to experience symptoms indicating the potential for preeclampsia, clinical judgment is paramount. Any signs of end-organ damage (signs and symptoms as described above) are an indication of superimposed preeclampsia with severe features and treatment should be initiated urgently.

3.7. ED presentation and recognition

Based on a recent retrospective review of approximately one million pregnancies, the most common way patients with suspected preeclampsia present is with hypertension, which occurs around 48% of the time in this population [17]. About 20% of these patients who are suspected to have preeclampsia due to elevated blood pressures ended up actually having preeclampsia, making hypertension the most predictive sign. Elevated blood pressures in a pregnant patient should always warrant an investigation and further testing for preeclampsia.

Headache occurs in about 23% of patients with suspected preeclampsia, making it the most common symptom, but only around 10% of these patients are ultimately diagnosed with preeclampsia [17]. Similarly, around 10% of patients who have presenting symptoms of visual changes develop preeclampsia, but this is a much rarer symptom making up only 2% of complaints. Edema is also less common, seen in 8% of patients, though it is the most predictive *symptom* of preeclampsia with 11% of those patients progressing to preeclampsia. Abdominal pain and nausea are present 8% and 12% of the time, respectively, and about 7–9% of those patients go on to develop preeclampsia. It is important to note that no one sign is anywhere near 100% predictive of preeclampsia, and emergency physicians should consider preeclampsia in all patients who are pregnant or recently post-partum. Additionally, although elevated blood pressure is a key feature, cases can present atypically, and those with non-elevated blood pressures can still ultimately be diagnosed with preeclampsia. Physicians should consider preeclampsia as a diagnosis for patients presenting with any of these symptoms.

A diagnostic algorithm and initial emergency department workup for patients with potential HDP diagnoses is outlined in Fig. 1 and Table 2 respectively.

4. How do we treat HDPs?

4.1. Acute management – blood pressure

First-line agents for the management of hypertension in pregnancy include labetalol (IV), hydralazine (IV), and nifedipine (PO) [10]. Severe-range hypertension (≥160/110 mmHg) requires urgent initiation of treatment within 30–60 min to quickly reduce the blood pressure. Anti-hypertensives should be rapidly up titrated until blood

pressures are below the severe range. If there is sufficient control with IV medication, patients can be transitioned to PO medication [8]. In the presence of pulmonary edema, IV nitroglycerin is also an option for management of severe pregnancy-related hypertension [2].

Blood pressures should be closely monitored. A suggested frequency of monitoring is every 10 min from initial treatment until one hour after goal blood pressure is achieved, followed by measurements every 15 min for the next hour and every 30 min the following hour. After meeting this threshold, blood pressure monitoring can be spaced to every four hours if well controlled [14]. Table 3 outlines antihypertensive medications to use in acute blood pressure management.

4.2. Acute management – seizure prophylaxis and treatment

Patients with eclampsia and preeclampsia with severe features should be treated with magnesium sulfate as it can stop seizures and reduce the risk of disease progression [8]. The mechanism of action of magnesium is primarily believed to be related to calcium antagonism and its function as an NMDA blocker [9]. There is variability in the

Table 2
Initial ED workup.

Test	Clinical Relevance
Urine studies Urine dipstick/urinalysis Urine protein/urine creatinine	Evaluate for proteinuria
CBC	Evaluate for hemolysis, thrombocytopenia
CMP	Evaluate for renal dysfunction, liver dysfunction
PT/PTT/INR	Evaluate for coagulopathy
LDH	Evaluate for hemolysis
Uric Acid	Marker of disease severity, renal dysfunction
Chest x-ray*	Evaluate for pulmonary edema
RUQ Ultrasound*	Evaluate liver dysfunction, subcapsular hematoma, hepatic rupture
Fetal assessment	Evaluate for signs of fetal distress, gestational age
Obstetric consultation	

* If clinically indicated.

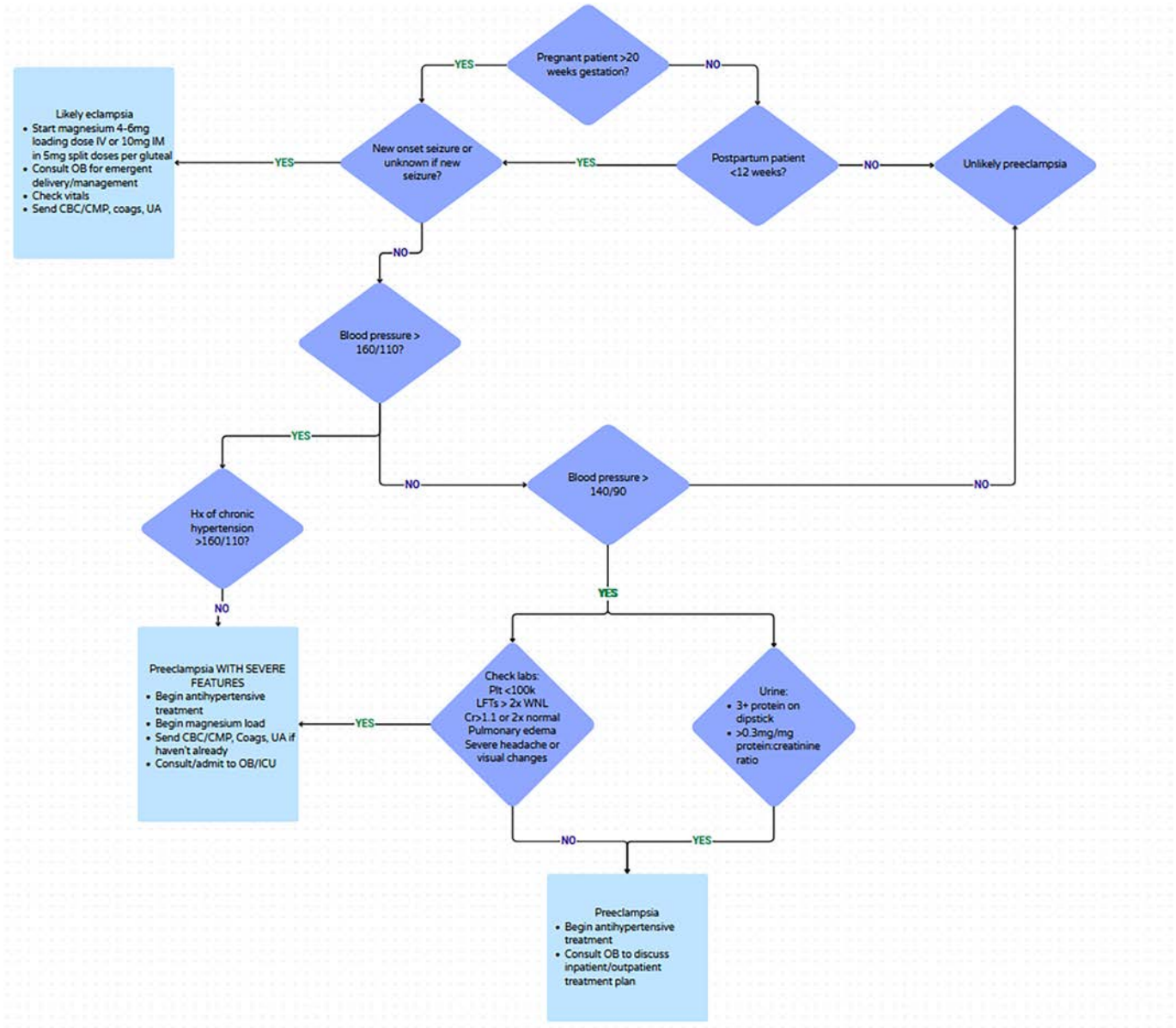

Fig. 1. Initial ED algorithm.

literature regarding the therapeutic ranges of magnesium, but it is recommended to start with a 4-6 g loading dose over 20–30 min, followed by 1–2 g/h for at least 24 h (or until delivery.) [8] Magnesium should not be given at rates >2 g/h as there is data showing worsened neonatal mortality. If IV access is not available, magnesium can be given intramuscularly (IM) as a dose of 10 g total (5 g given in each buttock.) Additional doses of 5 g can be given every 4 h [8].

Risks associated with magnesium administration include loss of deep tendon reflexes, respiratory depression, flaccid paralysis, hypotension, bradycardia and ultimately cardiac arrest if untreated. These adverse effects occur with increasing magnesium levels, with loss of deep tendon reflexes occurring prior to the other adverse effects. Deep tendon reflexes are therefore a useful indication of early magnesium toxicity. Providers should conduct magnesium checks with an evaluation of deep tendon reflexes. To prevent interrater discrepancy, the same provider should conduct these checks as long as possible, during patient care handoffs, both providers should perform checks together to ensure consistency.) To prevent magnesium toxicity from advancing, the first step is to stop magnesium administration and continue hourly checks until deep tendon reflexes normalize. If a patient shows worsening magnesium toxicity, such as respiratory depression or cardiac effects, the patient should be given calcium gluconate (through a peripheral IV) or calcium chloride (through a central IV) [9].

For recurrent seizures, adding additional antiepileptics such as benzodiazepines is appropriate. Keep in mind that these increase the risk of aspiration and apnea, so a patient's airway status should be monitored closely [8].

The most recent American College of Obstetricians and Gynecologists (ACOG) guidelines provide no consensus regarding magnesium for seizure prevention in those with gestational hypertension or preeclampsia without severe features because there is very limited data that shows benefit from magnesium in these populations [8]. In their recent guidelines, ACOG referenced two randomized trials from 1997 and 2003 totaling 357 patients. These studies showed that, for patients with preeclampsia without severe features, there was no significant difference in progression to eclampsia amongst those who received magnesium versus placebo.

The reviewers of this article were unable to find any additional randomized controlled trials addressing the potential benefits of administering magnesium to a broader range of patients with HDPs. Because of this, there is then concern that the potential adverse effects of magnesium administration may outweigh the benefits. In those with preeclampsia without severe features who are <37 weeks of gestation, administration of magnesium may be variable and based upon local practice patterns, hospital guidelines, or local resources. Therefore, our recommendation is to follow local practices or to create hospital guidelines in conjunction with available obstetric experts.

4.3. Delivery

For patients with preeclampsia or eclampsia, definitive treatment involves delivery of the fetus. This decision must be made by the obstetric and maternal fetal medicine team, particularly in cases of preterm delivery. Close observation without immediate delivery may occur in patients without severe features who are <37 weeks of gestation. For those with severe features, eclampsia, or signs of perinatal decompensation, urgent delivery is often recommended [2]. In patients with HDPs at <34 weeks gestational age and at risk of birth within the next 7 days, antenatal steroids are recommended to reduce the risk of neonatal morbidity and mortality, respiratory distress, and intraventricular hemorrhage [25].

Patients with HELLP syndrome or thrombocytopenia should be considered for higher platelet transfusion goals if planned for delivery (>20,000 $\times 10^9$/L) or cesarean section (>50,000 $\times 10^9$/L).

4.4. Treatment of chronic and gestational hypertension

4.4.1. ASA prophylaxis

Both ACOG and the Society for Maternal-Fetal Medicine (SMFM) recommend low-dose aspirin (81 mg/day) prophylaxis for pregnant individuals at high risk for pre-eclampsia. There is debate amongst other experts (such as the World Health Organization and the Royal College of Obstetricians and Gynecologists) regarding optimal dosing, with many suggesting doses >100 mg of aspirin daily to be most effective at reducing preeclampsia risk; more randomized control trials are needed for widespread consensus [26].

Aspirin prophylaxis should be initiated between 12 and 28 weeks of gestation and continued until delivery [27]. Early initiation (<16 weeks of gestation) is associated with the greatest risk reduction [27]. Risk factors should be discussed with patients by their OB/GYN. Low-dose aspirin may be especially recommended for those with only one moderate risk-factor, including those at risk due to socioeconomic status or racial factors. Daily low-dose aspirin is considered safe and has a low likelihood of patient or fetal complications [27].

4.4.2. Outpatient management

CHTN and gestational HTN are typically managed in the outpatient setting. There is some controversy regarding pharmacologic treatment of blood pressures between 140 and 160 mmHg systolic and 90–110 mmHg diastolic in the outpatient setting. Expert consensus is mixed on whether the benefit of lower blood pressure goals outweighs the risk of potential adverse fetal effects of more aggressive treatment. Emerging data, however, demonstrates decreased adverse pregnancy outcomes and overall benefit to treatment of non-severe hypertension in pregnancy [15,28]. Although the current ACOG recommendations do not specify any recommended pharmacologic treatment of blood pressures <160/110 without signs of preeclampsia, several other notable organizations (including SMFM, AHA/ACC 2025 guidelines, and the International Society for the Study of Hypertension in Pregnancy) support more aggressive blood pressure goals, incorporating pharmacologic treatment of sustained blood pressures above 140/90 mmHg [29-31].

Many common first-line antihypertensives (such as ACE inhibitors, angiotensin II receptor blockers, mineralocorticoid receptor antagonists, and direct renin inhibitors) are contraindicated in pregnancy. Appropriate first-line antihypertensive medications that are recommended

Table 3
Anti-hypertensive medications used for acute blood pressure management in preeclampsia [22,25].

Drug	Dose	Onset	Complications
Labetalol	10–20 mg IV, then 20–80 mg every 30 min to max of 300 mg total	1–2 min	Bradycardia, heart block, bronchospasm, decompensation of heart failure ***Contraindications:*** *Asthma, pre-existing myocardial disease, decompensated cardiac function, heart block, bradycardia*
Hydralazine	5 mg IV, then 5–10 mg IV every 20–40 min to max of 20 mg total	10–20 min	Higher doses may be associated with maternal hypotension, headaches, reflex tachycardia, and abnormal fetal heart tracings
Nifedipine (immediate release)	10–20 mg orally, repeated in 20 min if needed; can repeat 10–20 mg every 2–6 h; max daily dose 180 mg	5–10 min	Can lead to reflex tachycardia, headaches, peripheral edema, flushing

during pregnancy for non-severe hypertension include nifedipine XL and labetalol. Another reasonable alternative is methyldopa [2]. Those with known HTN who are newly pregnant should be counseled on antihypertensive use, including avoidance of teratogenic antihypertensives and obstetric follow up for further guidance.

Home blood pressure monitoring in those with HDPs may be an effective strategy to decrease complications when compared to office monitoring, with reports showing a lesser risk for induction of labor, postpartum readmission, and lowered birth weight [28,32]. Patients with HDP in the emergency department who are planned for discharge may benefit from a home blood pressure cuff and counseling regarding blood pressure monitoring. This is particularly true for those with barriers to reliable outpatient follow up.

4.5. Disposition

To determine the safest disposition for pregnant patients with HDPs, the emergency physician must factor in patient condition and stability, presence of active labor, and availability of obstetric and neonatal services. As the number of hospitals without obstetric care in-house decline, evaluation for the potential necessity of transfer becomes increasingly relevant.

The Emergency Medical Treatment and Labor Act (EMTALA) requires that a medical screening examination occur including evaluation for emergency medical conditions or active labor and stabilization prior to transfer. Patients who are <20 weeks gestation can likely be evaluated with usual emergency department care [33]. Patients who are >20 weeks of gestation should be evaluated for signs of active labor and any emergency medical condition. Should the patient be in active labor, the risks of transfer likely outweigh the benefits and delivery should occur prior to transfer. If they are not in labor and have signs and symptoms of HDPs, such as

- elevated blood pressure ≥ 140/90 mmHg
- symptoms of pre-eclampsia: headache, blurry vision, chest pain, dyspnea, abdominal pain, new lower extremity edema, or
- symptoms of eclampsia including seizure and altered mental status,

Further evaluation and treatment should be done in conjunction with available obstetric services. Those with preeclampsia with severe features, eclampsia, and HELLP syndrome will need either admission or transfer to an appropriate level of obstetric care. Those who have end-organ damage, hypertensive emergencies, or progression to eclampsia/seizures should be monitored in an intensive care setting with obstetrics capabilities, in the event that emergent delivery or high-risk fetal monitoring are needed [33].

Patients who have gestational hypertension, chronic hypertension, or preeclampsia without severe features (<37 weeks of gestation) and no sign of fetal distress can likely be managed in an outpatient setting with weekly lab tests, fetal monitoring, and monthly ultrasounds for fetal growth [8]. Emergency physicians should make these plans with obstetric consultation, and these patients should have good follow up and strict return precautions. Patients who will have difficulty with outpatient follow-up should be managed inpatient under obstetric care. Discharged patients should be counseled to return to the emergency department should they develop concerning symptoms including headache, difficulty breathing, visual changes, abdominal pain, leg swelling, and decreased urination.

5. Post pregnancy risks after preeclampsia/eclampsia

HDPs are associated with lasting effects for the pregnant individual, including increased risk for hypertension, coronary artery disease, strokes (ischemic and hemorrhagic), heart failure, renal disease, and venous thromboembolism. Although many of the pathologic changes that occur during HDPs resolve with delivery, patients with a history of HDP continue to have lasting risk for many cardiovascular diseases. This may be due to persistent endothelial dysfunction, cardiac remodeling, or previously underappreciated underlying cardiovascular risk. Often, the degree of lasting effects is associated with HDP disease severity.

Many patients with HDPs return to normotensive blood pressures during the postpartum period; however, there is a well-documented increase in risk for subsequent development of chronic hypertension. Patients with a history of HDP have a four-times higher risk for developing CHTN [34].

In those with a history of preeclampsia, there is an estimated two- to eight-fold increased risk of subsequently developing coronary artery disease, including a disproportionally higher risk of microvascular disease [7,9]. Cardiac dysfunction and remodeling, left ventricular hypertrophy, and an increased risk of arrhythmias and implantable cardioverter-defibrillator placement is commonly seen in these patients as well [35,36]. Left ventricular hypertrophy affects around 80% of those with HDP nearly a decade following their delivery [35]. Notably, the degree to which there is cardiac remodeling and dysfunction following pregnancy is directly associated with degree of severity of HDP [36].

HDPs are an established risk factor for acute stroke, both during pregnancy and postpartum, with hemorrhagic stroke being a significant cause of death in those with HDPs [37]. There is also data to suggest a long-term increased risk in venous thromboembolism (VTE) in those with a history of preeclampsia as well [38].

6. Neonatal outcomes

Potential fetal risks associated with HDPs include fetal growth restriction (estimated fetal weight < 10th percentile for gestational age) and preterm birth due to uteroplacental perfusion abnormalities [2,40]. Patients with HDPs should have access to higher level obstetric units and neonatal units due to the increased risk of complications [9].

Preeclampsia is the leading cause of medically-indicated preterm birth in the United States, with these infants accounting for 6% of all preterm births. This puts these infants at risk for known complications of prematurity, such as respiratory distress syndrome and retinopathy of prematurity.

The risk of intrauterine fetal demise increases in the setting of preeclampsia, particularly when severe; the estimated stillbirth rate of severe preeclampsia is 21 per 1000 [39]. This increased risk of stillbirth also extends to future pregnancies for patients with preeclampsia, with higher odds ratios for stillbirth in subsequent pregnancies found when deliveries occur earlier on (ie: 20–27 weeks) versus later on (ie; 32–36 weeks.) [40]

The decreased uteroplacental blood flow in preeclampsia is a significant risk factor for intrauterine growth restriction (IUGR). Preeclampsia is the most common cause of IUGR in the non-anomalous infant, and this risk of IUGR correlates to increased perinatal mortality. Additional risks of preeclampsia on the neonate include neonatal thrombocytopenia and neutropenia, although the mechanism for these has not been fully elucidated [39]. Neurodevelopmental outcomes for neonates of preeclamptic mothers are variable. More studies are needed to determine the impact. Studies have also shown that infants exposed to preeclampsia are at increased risk of diabetes and cardiovascular morbidity in adulthood. Therefore, these alterations of the intrauterine environment can have long-lasting effects, even years later.

7. Conclusions

HDPs encompass a broad spectrum of presentations, ranging from stable disease states managed in the outpatient setting to significant end-organ damage and acute illness requiring emergent stabilization. Early recognition with a high threshold of suspicion for patients at risk is vital for appropriately diagnosing and managing these patients. Treatment will depend on where on the HDP spectrum patients land. Management will generally involve aggressive BP management and

magnesium for preeclampsia with severe features, HELLP, and eclampsia, and potential delivery with obstetrics. Even after treatment and acute resolution of HDPs, emergency providers should be aware of the long-term effects of HDPs and view preeclampsia and eclampsia as risk factors for later disease, including cardiovascular disease, stroke, and heart failure.

Author contribution

All authors conducted comprehensive review, drafted, reviewed and approved of final manuscript.

CRediT authorship contribution statement

Lucille Martin: Writing – review & editing, Writing – original draft, Conceptualization. **Michele Callahan:** Writing – review & editing, Writing – original draft, Conceptualization. **Jennifer Wang:** Writing – review & editing, Writing – original draft, Conceptualization.

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