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REVIEW ARTICLE

Pre-eclampsia through the eyes of the obstetrician and anesthesiologist

M.M. Siddiqui,[a] J.M. Banayan,[b] J.E. Hofer[b]
[a]*Department of Obstetrics and Gynecology, The University of Chicago, United States*
[b]*Department of Anesthesia and Critical Care, The University of Chicago, United States*

ABSTRACT
Due to the high risk of morbidity and mortality from unrecognized and untreated pre-eclampsia, clinicians should have a high index of suspicion to evaluate, treat and monitor patients presenting with signs concerning for pre-eclampsia. Early blood pressure management and seizure prophylaxis during labor are critical for maternal safety. Intrapartum, special anesthetic considerations should be employed to ensure the safety of the parturient and fetus. Patients who have pre-eclampsia should be aware that they are at high risk for the future development of cardiovascular disease.

Background

Hypertensive disorders of pregnancy (HDP) complicate approximately 5–10% of pregnancies. In the past 20 years, the incidence of HDPs has increased by 25%.[1] These disorders, combined with delayed or inadequate treatment of severe systolic hypertension, continue to be leading causes of maternal death; nearly one woman dies every day in the United States of America (USA) and there are an additional 50–60 000 deaths per year worldwide.[2,3] A vast majority of deaths result from hemorrhagic stroke and the complications of seizures.[4–6]

In addition, for every HDP-related maternal death there are 50–100 near misses.[7–9] Consequently, the goal of clinicians when managing patients with HDP is to prevent maternal mortality and morbidity. This goal is accomplished with aggressive treatment of blood pressures in the extreme of range, seizure prophylaxis with magnesium, timely delivery and vigilant continuation of care in the postpartum period.

Classification of hypertensive disorders of pregnancy

In 2013 the American College of Obstetrics and Gynecology (ACOG) released a comprehensive Task Force Statement on Hypertensive Disorders of Pregnancy.[10] Pre-eclampsia was defined as two blood pressure readings at rest of >140/90 mmHg at least four hours apart (Table 1). Pre-eclampsia with severe features was defined as systolic blood pressure ≥160 mmHg or diastolic blood pressure ≥110 mmHg on at least two occasions four hours apart and/or by laboratory or additional clinical criteria (Table 2). A hypertensive emergency was defined as two severe blood pressure values (which do not need to be consecutive) taken 15–60 min apart. The continuum of gestational hypertension and pre-eclampsia is shown in Fig. 1. The transition from one to the next is dynamic and progressive, and can be recognized with heightened surveillance and frequent re-evaluation of evolving maternal status.

Risk factors

The risk of developing pre-eclampsia is highest if a woman has a personal history of pre-eclampsia in previous pregnancies (seven-fold increase) or a first degree relative with pre-eclampsia (three-fold increase).[11] Other risk factors include multiple gestation, maternal age >40 years, diabetes, obesity and chronic hypertension.[11] Increasing weight gain during pregnancy is also suggested to be a risk factor for pre-eclampsia,[12] with the risk doubling with every 5–7 kg/m^2 increase in body mass index (BMI).[13] Nevertheless, most cases of pre-eclampsia occur in healthy women with no obvious risk factors.[10]

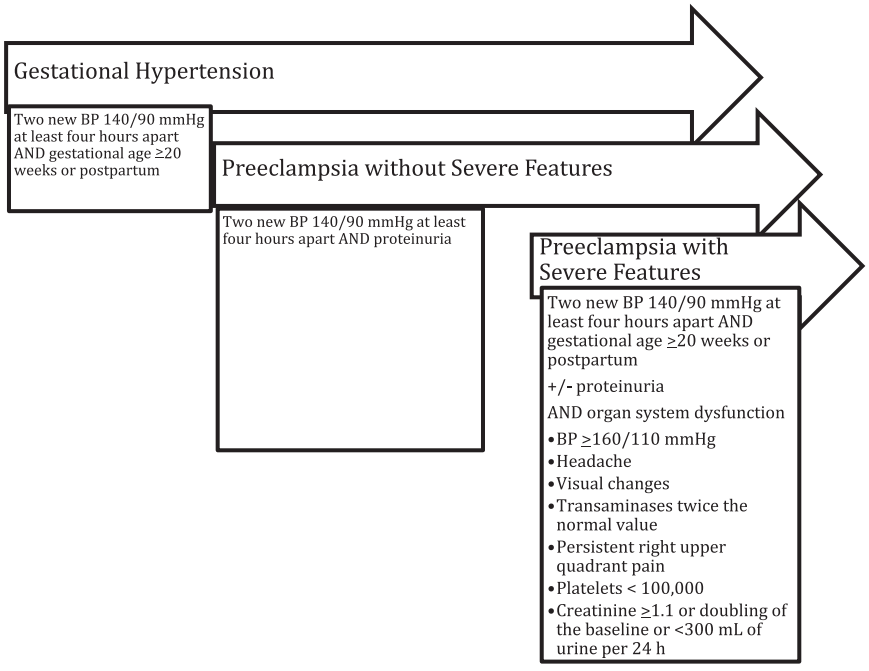
Correspondence to: J.E. Hofer, Department of Anesthesia and Critical Care The University of Chicago, 5841 S. Maryland Ave- MC 4028, Chicago, IL 60637, United States.
E-mail address: jhofer@dacc.uchicago.edu

Table 1 2013 definition of pre-eclampsia adapted from the American College of Obstetricians and Gynecologists' Task Force on Hypertension in Pregnancy[10]

Elevated blood pressures in a pregnant woman with previously normal blood pressure	≥20 weeks' gestation
>140/90 mmHg	On two occasions four hours apart
>160/110 mmHg	Confirm within minutes to determine need for antihypertensive medication
AND	
Proteinuria	
24 hour urine	300 mg of protein
Protein/creatinine ratio	0.3 (each measured as mg/dL)
Urine dipstick	+1

Table 2 Criteria for pre-eclampsia with severe features adapted from the American College of Obstetricians and Gynecologists' Task Force on Hypertension in Pregnancy[10]

Blood pressure at rest, at least four hours apart: systolic >160 mmHg or diastolic >110 mmHg
Renal insufficiency: creatinine ≥1.1 or doubling of the baseline creatinine
Thrombocytopenia: platelet count <100 000
Impaired liver function: persistent right upper quadrant or epigastric pain. Liver enzymes twice the normal value (elevated liver function tests)
Pulmonary edema
New onset cerebral or visual disturbances: headache, scotomata



BP: blood pressure.

Fig. 1 Criteria for Hypertensive Diseases of Pregnancy and the continuum between gestational hypertension to pre-eclampsia with severe features.

Obstetric management of pre-eclampsia

Primary prevention or preventing the occurrence of pre-eclampsia can be initiated with daily aspirin therapy during the late first trimester, for those women with a history of pre-eclampsia with severe features who presented prior to 34 weeks' gestation in a previous pregnancy.[14] A recent randomized control of women at

high risk for early-onset pre-eclampsia demonstrated a 60% decrease in the incidence of pre-eclampsia with the use of prophylactic low-dose aspirin, beginning from 11 to 14 weeks' gestation.[15] Secondary prevention of seizures and reduction in maternal morbidity from pre-eclampsia is achieved with close monitoring. This is used to detect deteriorating maternal status, for example involving the cardiovascular, neurological, pulmonary or renal systems or the development of the hemolysis, elevated liver enzymes and low platelets (HELLP) syndrome.

Antihypertensive management

Untreated or undertreated systolic hypertension is most commonly implicated in strokes and deaths from pre-eclampsia.[5] Approximately 60% of deaths may involve failure of women to recognize danger signs or symptoms and a delayed or inadequate response by healthcare providers.[16] Pregnant women who have a stroke have higher rates of death during hospitalization and experience complications such as dependence on mechanical ventilation, pneumonia, seizure and prolonged hospital stay.[17] Because of disruption of cerebral autoregulation in pre-eclampsia, pre-eclamptic women are at a higher risk for intracranial bleeding at much lower blood pressures (>155 mmHg).[5,18] Whereas overall both stroke prevalence and mortality among older adults in the USA has declined in the past decade, the rate of pregnancy-related stroke has increased by 61.5%.[17]

If severe hypertension (>160/110 mmHg) is encountered, a repeat measurement should be obtained every five minutes for 15 min and the physician informed. If severe blood pressures persist for 15 min or more, treatment should begin promptly, ideally within 60 min of the second elevated value. If two severe blood pressures are obtained within 15 min, treatment may be started. Treatment with first-line agents should be initiated within 30–60 min of confirmed severe hypertension in order to decrease the risk of maternal stroke.[5,19–21]

First-line therapies include intravenous labetalol or hydralazine and oral short-acting nifedipine.[21] A Cochrane Review comparing the use of antihypertensive agents found comparable efficacy for a variety of agents, with acceptable choices including labetalol, nifedipine, and hydralazine.[22] Oral nifedipine has been found to be as efficacious as intravenous labetalol in the treatment of severe hypertension.[23]

The objective of treatment is to achieve blood pressures in the range of 140–150/90–100 mmHg while avoiding an overcorrection of blood pressure that compromises uteroplacental blood flow.[10] If elevated blood pressure is resistant to first line therapies, the ACOG 2017 Committee Opinion offers recommendations for second-line interventions such as continuous infusions of nicardipine or esmolol.[24] In extreme emergencies intravenous sodium nitroprusside may be used briefly, but cautiously, because of concerns about increasing maternal intracranial pressure and the potential for cyanide and thiocyanate toxicity in the mother and fetus. If medication infusions are required to manage blood pressure, the Committee Opinion recommends consultation with a specialist in Anesthesiology, Maternal-Fetal Medicine or Critical Care to guide implementation and management of second-line therapy. Both the ACOG and the Safe Motherhood Initiative have created a Hypertensive Emergency Checklist, outlining antihypertensive treatment with oral short-acting nifedipine or intravenous labetalol or hydralazine, which can be easily adapted to any clinical setting. The recommended dosing protocols (Table 3) do not require cardiac monitoring. Resources including safety bundles, checklists and patient information are available at https://www.acog.org/Womens-Health/pre-eclampsia-and-Hypertension-in-Pregnancy.[25]

Management of eclampsia

The incidence of eclampsia is reported to be 1/2000–1/3500 pregnancies, with more than 90% of cases occurring after 28 weeks' gestation.[26–28] In the developed world there is an increasing incidence of eclampsia at 48 h after delivery.[27]

In the USA 20% of maternal deaths are attributed to HDPs, with half the deaths resulting from eclampsia. The common underlying insult is acute severe hypertension causing vasogenic edema and leading to encephalopathy and seizures. Brain imaging shows vasogenic edema in most women with eclampsia.[29,30] The most commonly reported symptom preceding an eclamptic seizure is headache.[27]

Between 59% and 75% of preeclamptic patients report one of the following symptoms: persistent occipital or frontal headache, blurred vision, photophobia, right upper quadrant/epigastric pain or altered mental status.[27] Eclamptic seizures are associated with high rates of maternal morbidity from placental abruption, disseminated intravascular coagulation, acute renal failure, pulmonary edema, aspiration pneumonia and cardiopulmonary arrest.[26,28]

The principles of management of eclamptic seizures are to prevent recurrent seizures, prevent maternal injury and aspiration, support cardiopulmonary function and avoid urgent delivery for transient nonreassuring fetal status. Magnesium sulfate is the most effective agent for preventing eclampsia and recurrent eclamptic seizures.[31,32]

The Cochrane database reports the rate of seizures with magnesium to be 9.4% vs 23.1% with the use of diazepam or phenytoin. A 4 g loading dose is given over 15–20 min, followed by a 2 g/h infusion. Ten percent of patients will have a second seizure while on magnesium so an additional bolus of 2 g over 3–5 min can be

Table 3 First line antihypertensive medications that do not require cardiac monitoring when treating acute-onset severe hypertension, in pregnant and postpartum patients. Table adapted from the Committee Opinion No. 767 Summary: Emergent Therapy for Acute-Onset, Severe Hypertension During Pregnancy and the Postpartum Period[24]

Labetalol	Hydralazine	Immediate release nifedipine
Intravenous (IV)	Intravenous	Oral
Dose every 10 min	Dose every 20 min	Dose every 20 min
Escalating dose 20 mg→40 mg→80 mg →80 mg→80 mg	5–10 mg dose	Escalating dose 10 mg→20 mg→20 mg
Maximum dose 300 mg IV in 24 h	Maximum dose 30 mg IV in 24 h	If blood pressure goal not achieved, administer labetalol

administered in such instances. The use of magnesium for eclamptic seizure prophylaxis is also recommended for women with severe features.[10] While administration for pre-eclampsia without severe features is not recommended, frequent clinical evaluation to determine if severe features have developed should guide the initiation of magnesium therapy.[10] Despite the potential risk of exacerbating uterine atony, magnesium should be continued intra-operatively during cesarean delivery because (i) the half-life of magnesium is approximately five hours and discontinuation is unlikely to have an immediate effect on uterine tone, and (ii) stopping magnesium infusion may lead to subtherapeutic levels in the postpartum period, increasing the risk of an eclamptic seizure.[33,34]

In the presence of acute kidney injury from pre-eclampsia and also magnesium therapy for seizure prophylaxis, assessment of serial deep tendon reflexes and serum magnesium levels is necessary to monitor for magnesium toxicity, given that magnesium is renally excreted. Infrequently, toxic magnesium levels require intensive care unit (ICU) admission and dialysis for magnesium clearance.[35]

Recommendations for delivery

As a general guideline, delivery is recommended at 34 weeks' gestation for pre-eclampsia with severe features; at 37 weeks' gestation in the absence of severe features; and urgently in the setting of eclampsia. Nevertheless, deteriorating maternal or fetal status may necessitate preparing for delivery at earlier gestations. Although the majority of women with pre-eclampsia will have successful vaginal deliveries, there is a higher likelihood of cesarean delivery in this population regardless of parity or gestational age.[36]

Preterm pre-eclamptic women were more likely to have a cesarean delivery compared to non-preeclamptic women, whether nulliparous (29% vs 7%) or multiparous (18% vs 8%).[36] In addition to higher cesarean delivery rates, women with pre-eclampsia who had prolonged labor greater than 24 hours, followed by a cesarean delivery, were found to have a 10-fold increased risk of adverse outcomes compared to women having a planned cesarean or vaginal delivery.[37] However, there is a lack of robust evidence from randomized controlled trials to assist clinicians in making a recommendation for planned cesarean delivery versus vaginal delivery for patients with pre-eclampsia with severe features.[38]

Role of echocardiography and ultrasound imaging in management

Echocardiography appears to be a useful diagnostic tool in managing patients with HDP. Interpretation, however, should consider the structural anatomical changes observed during pregnancy. For example, a mild four-chamber dilation, an increase in left ventricular wall thickness and an increase in left and right ventricular mass are typical echocardiographic findings in normal pregnancy. These changes are attributed to the increased stroke volume and cardiac output seen in pregnancy.[39] Other normal echocardiographic findings seen in pregnancy, with or without pre-eclampsia, include mild valvular regurgitation in the mitral, tricuspid, and pulmonic valves.[39] Transient pericardial effusions occur in up to 40% of pregnant women but these resolve in the postpartum period.[40] However, aortic valve insufficiency is not a normal finding and warrants further workup.[41] Because of the elevated afterload in pre-eclampsia, all the normal changes of pregnancy may be more pronounced, especially in preterm pre-eclampsia.[42] Generally, women with HDP do not have diminished left ventricular ejection fraction but may exhibit subclinical myocardial dysfunction, as defined by worsening longitudinal, circumferential and radial strain.[43] In addition, preeclamptic patients are at risk for diastolic dysfunction.[44]

Echocardiography may even be a useful tool to predict the development of preterm pre-eclampsia. Women with placental insufficiency and impaired left ventricular function are more likely to develop pre-eclampsia than those without.[42] However, more work is required to evaluate the predictive utility of echocardiography.

In addition to point-of-care focused echocardiography, clinicians are incorporating ultrasound in a multitude of other assessments to guide medical management. A recent review article[45] provides a broad

description of the increased role of ultrasound in obstetric anesthesia, including its use for assessing; A. the airway (to identify women who could benefit from early labor analgesia and avoidance of general anesthesia if there is an anticipated difficult airway); B. gastric volume (to identify who is at greater risk of aspiration); C. intracranial hypertension (to detect changes in the optic nerve sheath diameter that occurs in pre-eclampsia); D. vascular access, E. neuraxial and transversus abdominis plane blocks; and (potentially most importantly) F. pulmonary edema[46,47] (to guide fluid management). In a small prospective cohort study, lung ultrasound detected interstitial edema in parturients with pre-eclampsia and the presence of B-lines was associated with increased left ventricular end-diastolic pressures.[46] This is important as interstitial edema has been recognized as a "silent step" preceding alveolar edema.[48] Early detection of this interstitial fluid, seen as B-lines on ultrasound imaging, can promote conservative fluid management and prevent morbidity.

Immediate and long-term morbidity

Despite the normalization of blood pressure postpartum, women with HDP are still at risk of adverse outcomes immediately after delivery. These include hemorrhagic stroke, eclamptic seizures and peripartum cardiomyopathy.[49–51] The time from hospital discharge until the first six-week postpartum visit is critical. Immediately after delivery, women may experience silent hypertension secondary to fluid shifts and altered cerebral autoregulation and this may lead to hemorrhagic stroke, especially within the first 10 days after hospital discharge.[49] In some instances, a re-admission in the postpartum period because of a pregnancy-associated stroke may be the first time a patient is diagnosed with HDP. Therefore, extreme vigilance is required during evaluation of all postpartum women returning to the emergency room with complaints. Women with HDP have transient lowering of their blood pressures for 48 h after delivery, followed by an increase at three to six days postpartum.[10,52–54] Currently ACOG recommends careful surveillance of blood pressure until the third postpartum day and again between seven and 10 days post delivery.[10] Patient and family education about postpartum pre-eclampsia and identification of danger signs/symptoms is emphasized when a patient is discharged home.

Another complication that may occur immediately after delivery is an eclamptic seizure. Although the majority of cases of eclampsia occur intrapartum or within 48 h of delivery, one-fourth of all eclamptic seizures occur later, some as late as six weeks after delivery.[50] This underscores the need to inform patients about their risk that pre-eclampsia might progress and/or an eclamptic seizure occur after discharge.

Though most women with HDP have normal blood pressure after delivery, a small proportion remain at an increased risk of developing cardiovascular disease later in life.[55,56] It is hypothesized that HDP is a marker, rather than a cause, for developing cardiovascular disease.[57] Interestingly, the factors that predispose a woman to develop severe placental vascular disease are the same factors that predispose her to develop coronary artery disease and myocardial dysfunction prematurely.[58]

Type II diabetes mellitus and hypertension are two of the most strongly linked future cardiovascular morbidities in women with HDP.[59] Women with a history of HDP are at a five-times higher risk of developing hypertension later in life than those without.[60] Similarly, women with history of pre-eclampsia have double the risk of developing ischemic heart disease, stroke and venous thrombo-embolic events five to 15 years after pregnancy, compared to women with uncomplicated pregnancies.[61] Consequently, the American Heart Association and the European Society of Cardiology now include HDPs as a risk factor for cardiovascular disease in women.[62]

Women with pre-eclampsia are approximately 80% more likely to suffer an ischemic stroke later in life[57] and those who experience eclampsia may be at increased risk for developing a seizure disorder.[63] Compared with women with uncomplicated pregnancies, women with a history of pre-eclampsia demonstrate an altered metabolic phenotype years after delivery, having higher BMI, blood pressure, non-fasting glucose levels, low-density lipoproteins and lower high-density lipoproteins.[64]

The ACOG Task Force on Hypertension in Pregnancy recommends assessment of blood pressure, lipids, fasting blood glucose and BMI in women with recurrent pre-eclampsia or pre-eclampsia who have given birth prior to 37 weeks' gestation, but advises clinicians to use their own judgment and balance the value of such interventions with cost and patient inconvenience.[10] Women diagnosed with a HDP and being discharged from hospital after delivery should have a plan for cardiovascular follow-up in order to combat the known risk of cardiovascular disease in the future.[65]

Anesthetic management of pre-eclampsia

If the patient is admitted to a labor room and the obstetric team is committed to delivering the patient, it is prudent for the anesthesia professional to have an early discussion with the parturient about her options for labor analgesia and, arguably, to recommend early neuraxial analgesia. Due to airway concerns in pregnancy which may be exaggerated in pre-eclampsia, the presence of a functional neuraxial catheter for labor analgesia offers the possibility of avoiding general

anesthesia and airway instrumentation should the need arise due to emergency cesarean delivery.

The risk of a 'failed airway' in obstetrics is estimated to be 1:300, which is significantly higher than the risk in the general surgical population.[66] The peripheral edema of pre-eclampsia can manifest as laryngeal edema, making the risk of failing to secure the airway even higher. In obese non-pregnant patients with a BMI >40 kg/m^2, it has been demonstrated that, following pre-oxygenation with eight vital capacity breaths over one minute, the time to desaturate to 92% following rapid sequence induction and apnea is approximately 160 s.[67] In an obese pregnant patient, an accelerated time to desaturation is expected because of the cephalad shift of the diaphragm from the gravid uterus reducing the functional residual capacity, combined with the metabolic needs of the fetus. For these reasons, the anesthesia professional may consider recommending an early neuraxial catheter that could be utilized for both labor analgesia and surgical anesthesia.

Neuraxial anesthesia is safe for pre-eclamptic women undergoing cesarean delivery.[68–71] There were once concerns that focused on the sympathectomy from neuraxial anesthesia causing abrupt hypotension and subsequent fetal compromise but multiple studies have demonstrated the safety of neuraxial anesthesia even when pre-eclampsia with severe features is present.[68–71] It appears that women with pre-eclampsia with severe features actually experience less hypotension after spinal anesthesia than do healthy parturients.[69]

When a neuraxial anesthetic is not used for cesarean delivery, whether due to patient choice, lack of time or the presence of coagulopathy, general anesthesia presents a challenge. At the time of intubation, a major concern is the acute increase in blood pressure that can precipitate intracranial hemorrhage associated with direct laryngoscopy. To prevent such morbidity, placing a pre-induction arterial cannula for continuous blood pressure monitoring, and the empiric addition of drugs to the rapid sequence induction to mitigate the hypertensive response to laryngoscopy, should be considered.[72]

Strategies to prevent peri-induction hypertension include use of medications that are easy to prepare, have fast onset but short duration, minimal transfer to the neonate and few maternal side effects.[72] Recommended intravenous medications include esmolol 1–2 mg/kg, nitroglycerin 1.5–2.5 μg/kg and remifentanil 0.5–1 μg/kg. Despite the intravenous nitroglycerin dosing being substantial, hemodynamic side effects are minimal.[73]

Intravascular volume management in patients with HDP is typically restrictive. In normal pregnancy, central venous pressures (CVP) are unchanged due to increased vasculature capacitance accommodating the increase in circulating volume. A recent Guytonian model of hypertension challenges the use of CVP to accurately evaluate volume status. In this model, mean systemic filling pressures (Pms) were evaluated, instead of CVP, in patients with normal pregnancy versus pre-eclamptic pregnancy.[74] In both pregnant states the Pms was elevated, with an exaggerated elevation in pre-eclampsia (29% higher than in normal pregnancy). This elevated Pms supports restrictive fluid management in HDP since the baseline volume status is already increased, although the ideal fluid strategy remains unknown. In a systematic review of randomized fluid management strategies evaluating crystalloid versus colloid and the outcomes of pulmonary edema, perinatal mortality, preterm birth and cesarean delivery, the authors found insufficient data to make a final recommendation.[75] Ideally, fluid management should be individualized based on markers of end-organ perfusion (including urine output, mental status, pH and lactate) and/or point-of-care bedside ultrasound examinations. Lung ultrasonography to detect suspected pulmonary edema and transthoracic echocardiography (rapid obstetric screening echocardiography known as the ROSE scan) to evaluate ventricular volume status can help guide fluid therapy.[76–78]

In the postpartum period, a common presenting complaint is headache. If the patient had a neuraxial anesthetic the possibility of a post-dural puncture headache should be in the differential diagnosis, along with other causes including but not limited to migraine, tension headache, cerebral venous thrombosis, intracranial bleed, postpartum pre-eclampsia and posterior reversible encephalopathy syndrome (PRES).[79] If the headache is accompanied by hypertension, an evaluation for pre-eclampsia including laboratory studies should be pursued. The patient's blood pressure can be controlled using the agents previously discussed and anti-seizure prophylaxis with magnesium may be indicated.[21,62] If the diagnosis is unclear, a computerized tomography scan to search for intracranial pathology or vasogenic cerebral edema resulting in PRES should be considered.

Due to the high risk of morbidity from unrecognized and untreated pre-eclampsia, clinicians should have a high index of suspicion to evaluate, treat and monitor women presenting with signs consistent with pre-eclampsia. The ideal location to manage patients with peripartum pre-eclampsia is difficult to decide. In 2016 the ACOG published a practice bulletin on critical care in pregnancy to help guide healthcare providers.[80] If there is need for endotracheal intubation or hemodynamic compromise requiring vasopressor support, an intensive care unit admission is appropriate. However, this decision is dependent on the level of care at an institutional level. Escalated care for patients with less severe peripartum pre-eclampsia may be appropriate on the labor and delivery ward, where clinicians and nurses are familiar with the peripartum physiology of preg-

nancy, knowledgeable about the National Partnership for Maternal Safety Consensus Bundle on severe hypertension and can implement algorithms to manage pre-eclampsia.[81]

A standardized approach to treating HDPs, combined with administration of magnesium sulfate, has been demonstrated to significantly decrease the incidence of eclampsia (from 42.6% to 0.62%) and severe maternal morbidity (from 16.7% to 2.4%).[82] Regardless of where the patient is ultimately managed, the implementation of therapy should not be delayed while transport to an critical care unit is being arranged. Clinicians and nurses on the labor and delivery floor need to know how to mobilize resources locally and initiate treatment of pre-eclampsia promptly. Early consultation with an intensivist may be prudent so that the patient can be quickly transitioned to be the primary provider if blood pressure and end-organ perfusion marker goals (urine output, mental status, lactate levels) are not met on the labor and delivery unit.

Summary

Despite knowledge about pre-eclampsia, its incidence is increasing, and patients continue to experience severe complications such as hemorrhagic stroke, eclamptic seizures and peripartum cardiomyopathy. The 2013 ACOG Task Force Statement on Hypertensive Disorders of Pregnancy provides clinicians with guidelines to aid diagnosing pre-eclampsia,[10] while the 2019 Committee Opinion No. 767 gives treatment algorithms for first- and second-line antihypertensive medications, as well as magnesium therapy, to guide treatment of acute-onset severe hypertension during pregnancy and postpartum.[24] Although the early diagnosis and aggressive treatment of severe hypertension is central to the effective management of pre-eclampsia and the prevention of immediate complications, women who experience HDPs continue to be at risk for long-term sequelae including cardiovascular disease, seizure disorders and metabolic syndrome.

During the peripartum period, optimal outcomes for the mother and fetus may be achieved from a multidisciplinary approach including an obstetrician, anesthesiologist, intensivist, nurse/midwife and neonatologist. Expertise from these disciplines can guide decision-making about the timing and mode of delivery, maternal and fetal monitoring and medical management. Close adherence to ACOG guidelines and physician vigilance with respect to surveillance and implementation of treatment algorithms should help optimize outcomes.

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