Preeclampsia: Current Approaches to Nursing Management

A clinical review of risk factors, diagnostic criteria, and patient care.

ABSTRACT: Preeclampsia, one of four hypertensive disorders of pregnancy, has traditionally been characterized as new-onset hypertension and proteinuria developing after 20 weeks' gestation. It is, however, now understood to be a complex, progressive, multisystem disorder with a highly variable presentation and a number of potentially life-threatening complications. The American College of Obstetricians and Gynecologists Task Force on Hypertension in Pregnancy has refined preeclampsia diagnostic criteria accordingly, and as the disorder's pathogenesis has been more clearly defined, new targets for screening, diagnosis, prevention, and treatment have emerged. This clinical update provides a review of current practice related to preeclampsia risk assessment, prediction, and management. It discusses preeclampsia pathophysiology and points readers to valuable health care resources on the topic.

Keywords: clinical management, nursing care, preeclampsia, pregnancy complication, pregnancy-induced hypertension, risk factors

KW was a healthy 35-year-old woman whose life was forever changed when she developed severe and life-threatening complications during the latter half of her second pregnancy. (This case is real, based largely on the patient's description of her experience of the events. Identifying details have been omitted to protect the patient's privacy.) KW's one previous pregnancy had ended with a first-trimester miscarriage. Her second pregnancy had been unremarkable until 31 weeks' gestation. Throughout this pregnancy, she had received routine prenatal care from her obstetrician as well as intermittent follow-up from a maternal–fetal medicine specialist because of her advanced maternal age.

For the first 30 weeks, KW's pregnancy progressed smoothly. She attended all of her prenatal checkups, maintained a healthy diet and an active lifestyle, and did everything she could to ensure a healthy pregnancy. But at 31 weeks, KW began to show signs and symptoms of preeclampsia.

First, she developed excruciating pain in her upper right side. A trip to the perinatal triage unit revealed that her blood pressure was on the high end of normal, she had protein in her urine, and her platelet count was at the low end of normal. With analgesia, the pain in her right side resolved, and after eight hours of close observation, she was sent home. The focus of KW's care plan became "expectant management," with preparation for delivery at no more than 37 weeks' gestation and intensive maternal and fetal monitoring, the frequency of which was based on findings of maternal and neonatal surveillance. Over the next several weeks, KW's monitoring included

Figure 1. Possible Physiological Changes in Preeclampsia

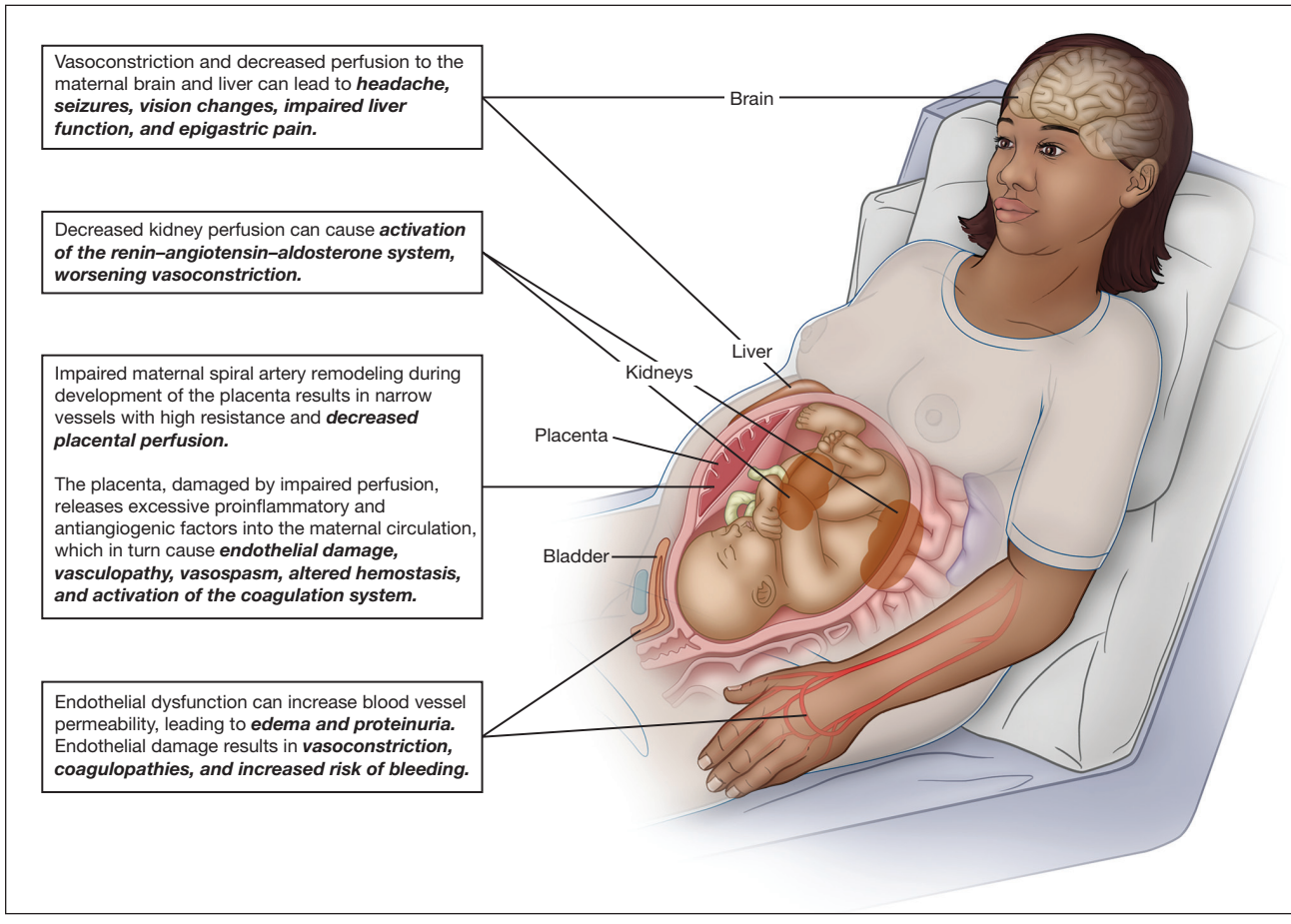

Illustration by Sara Jarret.

- home blood pressure monitoring.
- weekly fetal nonstress tests.
- weekly visits with the maternal–fetal medicine specialist and the obstetrician.
- 24-hour protein measurements.
- weekly blood samples to measure complete blood count, as well as levels of liver enzymes and serum creatinine.
- ultrasound, performed weekly for measurement of amniotic fluid index and every three weeks for fetal growth assessment.

At 35 weeks and three days' gestation, KW developed a persistent headache. Although she knew this was a warning sign of preeclampsia, she believes she was in denial. She wanted to hold onto her pregnancy as long as possible and thought she could put off telling the maternal–fetal medicine specialist until her appointment two days later. Her headache continued, and at her appointment, her blood pressure was 160/110 mmHg. Her physician told her that her pregnancy was over, and she agreed to be admitted for induction.

Because KW's platelet count was extremely low (33,000 per microliter, as she recalls), the physicians were concerned about her bleeding risk. For this reason, they opted to induce labor rather than to proceed immediately to epidural placement and cesarean section. Over a 26-hour labor, however, KW's condition further deteriorated. Although she was receiving a continuous IV infusion of magnesium sulfate for seizure prophylaxis, after 24 hours of labor her vision became gradually blurry, she started seeing spots, and eventually, she saw nothing but "a white blur." Soon after her loss of vision, she had a seizure, indicating that her condition had advanced to eclampsia (pregnancy-related seizures). This life-threatening deterioration led to an emergency cesarean delivery, multiple platelet transfusions, an ICU stay, and—

owing to his prematurity—a neonatal ICU stay for her newborn son for monitoring of lung function and bilirubin levels.

While KW was recovering from her cesarean section, her vision returned. Remarkably, just 36 hours after giving birth to a healthy baby boy, KW was able to walk to the neonatal ICU to see and breastfeed her son for the first time. A little less than a week after delivery, KW and her son were discharged from the hospital. KW recovered from the acute symptoms of preeclampsia. Following delivery, however, she developed chronic hypertension requiring antihypertensive therapy, and one year after the birth of her son, she experienced a second seizure. Although she was diagnosed with a mild seizure disorder, she has been seizure free for two years. She understands that a history of preeclampsia puts her at elevated risk for cardiovascular disease, necessitating annual cardiovascular screening.

KW's case is presented here to set the stage for a clinical update on preeclampsia–eclampsia, one of four hypertensive disorders of pregnancy (see *The Hypertensive Disorders of Pregnancy*[1]). KW had developed a severe form of preeclampsia, which was complicated by the syndrome known as HELLP (in which the patient exhibits hemolysis, elevated liver enzymes, and a low platelet count) and eventually progressed to eclampsia. This article provides nurses with current information related to diagnosis, pathophysiology, risk factors, prediction, prevention, and management of preeclampsia. It further suggests preeclampsia resources for use by nurses, pregnant women, and preeclampsia survivors.

PREECLAMPSIA REDEFINED

Although preeclampsia has traditionally been defined by the presence of new-onset hypertension and proteinuria after 20 weeks' gestation, the American College of Obstetricians and Gynecologists (ACOG) Task Force on Hypertension in Pregnancy emphasizes that it is a complex, progressive, multisystem disorder of pregnancy that can present in different forms, with some women experiencing unremitting headaches or severe right upper quadrant pain and others experiencing no symptoms before prenatal visits reveal they have elevated blood pressure and protein in their urine. The ACOG Task Force has refined the diagnostic criteria accordingly (see Table 1[1]). Based on evidence that proteinuria is neither a robust predictor nor the sole predictor of adverse outcomes, it was removed both as an indicator of severe preeclampsia and as a necessary criterion for preeclampsia diagnosis.[1-3] Women with either proteinuric or nonproteinuric preeclampsia are at risk for adverse outcomes and require increased maternal and fetal surveillance.[4,5] In 2000, the National High Blood Pressure Education Program of the National Heart, Lung, and Blood Institute had removed edema from the diagnostic criteria because it is common in women with normal pregnancies; in addition, it removed systolic blood pressure increases of 30 mmHg or diastolic blood pressure increases of 15 mmHg in women with blood pressure below 140/90 mmHg, because such women are not more likely to have adverse outcomes.[6]

Recommended terminology. The ACOG Task Force has also recommended changes in the terminology used to describe the severity of preeclampsia. They suggest that the term "preeclampsia without severe features" be used instead of "mild preeclampsia."[1] In addition to the multisystem findings listed in Table 1, either of the following would be considered a severe feature of preeclampsia[1]:

- systolic blood pressure at or above 160 mmHg or diastolic blood pressure at or above 110 mmHg

The Hypertensive Disorders of Pregnancy

The four hypertensive disorders of pregnancy are classified as follows[1]:

- chronic hypertension—hypertension of any cause that predates pregnancy or develops before 20 weeks' gestation
- gestational hypertension—new-onset hypertension that develops in a previously normotensive woman after 20 weeks' gestation in the absence of proteinuria
- preeclampsia–eclampsia—new-onset hypertension that develops in a previously normotensive woman after 20 weeks' gestation and is accompanied either by new-onset proteinuria or, in the absence of proteinuria, by signs of multisystem involvement, such as thrombocytopenia
  - preeclampsia is said to progress to eclampsia if convulsions occur
  - preeclampsia–eclampsia may be accompanied by the following group of laboratory findings: hemolysis, elevated liver enzymes, and a low platelet count, known as the HELLP syndrome, which is often considered a subtype of preeclampsia
- chronic hypertension with superimposed preeclampsia—preeclampsia that develops in a woman with chronic hypertension

Table 1. Diagnostic Criteria for Preeclampsia

Blood pressure	• Greater than or equal to 140 mm Hg systolic or greater than or equal to 90 mm Hg diastolic on two occasions at least 4 hours apart after 20 weeks of gestation in a woman with a previously normal blood pressure • Greater than or equal to 160 mm Hg systolic or greater than or equal to 110 mm Hg diastolic, hypertension can be confirmed within a short interval (minutes) to facilitate timely antihypertensive therapy
and	
Proteinuria	• Greater than or equal to 300 mg per 24-hour urine collection (or this amount extrapolated from a timed collection) or • Protein/creatinine ratio greater than or equal to 0.3[a] • Dipstick reading of 1+ (used only if other quantitative methods not available)
Or in the absence of proteinuria, new-onset hypertension with the new onset of any of the following:	
Thrombocytopenia	• Platelet count less than 100,000/microliter
Renal insufficiency	• Serum creatinine concentrations greater than 1.1 mg/dL or a doubling of the serum creatinine concentration in the absence of other renal disease
Impaired liver function	• Elevated blood concentrations of liver transaminases to twice normal concentration
Pulmonary edema	
Cerebral or visual symptoms	

[a] Each measured as mg/dL.
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on two occasions at least four hours apart in a patient on bedrest, unless antihypertensive therapy had been initiated previously
- severe and persistent right upper quadrant or epigastric pain that does not resolve with medication and is not explained by alternative diagnoses

FREQUENCY, MORBIDITY, AND MORTALITY

In the United States, preeclampsia complicates 2.3% to 3.8% of pregnancies.[7,8] Globally, it's estimated to affect 4.6% of pregnancies,[7] though frequency varies by region, being reportedly as low 0.2% in Vietnam and as high as 7.67% in Nicaragua.[9]

Despite the relatively low percentage of pregnancies affected by preeclampsia, it remains a leading cause of maternal morbidity and mortality. Hypertensive disorders are responsible for roughly 14% of pregnancy-related maternal deaths worldwide[10] and about 7.4% of such deaths in the United States[11]—and preeclampsia is the most common hypertensive disorder of pregnancy.[1] Preeclampsia can affect virtually every organ system, putting the mother at risk for a host of morbidities, including disseminated intravascular coagulation and respiratory, cardiovascular, cerebrovascular, liver, kidney, uterine, and neurologic dysfunction.[9,12-14]

For infants exposed to preeclampsia in utero, preeclampsia-related morbidity is associated primarily with prematurity, though these infants tend to have an unfavorable cardiometabolic profile (that is, elevated blood pressure, a high body mass index [BMI], or signs of altered cardiac function and structure) in adolescence or adulthood.[15-18] Despite the significant health burden that preeclampsia places on women and their infants, no test can reliably predict preeclampsia and no known practices can prevent its occurrence. Clinical management of preeclampsia is, therefore, symptom based, and the only known cure for preeclampsia is delivery.

PATHOPHYSIOLOGY OF PREECLAMPSIA

Depending on time of onset during gestation, preeclampsia may represent more than a single condition or include disease subtypes with different causes and clinical presentations. Current evidence suggests that the pathogenesis of preeclampsia proceeds in stages.[19] The early stages occur during pregnancy weeks 8 through 18 and involve altered placental development that reduces blood flow between the maternal and fetal circulations; no clinical signs or symptoms are associated with these early stages. Signs and symptoms of preeclampsia do not appear until the second half of pregnancy, though they are secondary to the impaired maternal perfusion of the placenta that occurs in the early weeks of pregnancy.[19]

Table 2. Preeclampsia Risk Factors[1, 20-22]

Pregnancy-Related Risk Factors	Preexisting Risk Factors
• Nulliparity • Personal or family history of preeclampsia • Multifetal gestation • In vitro fertilization	• Advanced maternal age (> 40 years) • Preexisting medical conditions: o Chronic hypertension o Chronic renal disease o Thrombophilia o Type 1 or 2 diabetes mellitus o Autoimmune disease (for example, systemic lupus erythematosus) o Antiphospholipid antibodies o Obesity

Role of placenta in maternal–fetal circulation. The placenta provides the primary source of sustenance to the growing fetus throughout pregnancy, delivering necessary nutrients and oxygen while removing waste. Two factors are critical in this exchange process: delivery of maternal circulation to the placenta and adequate placental surface area for an exchange of substances between the maternal and fetal circulations. The maternal circulation delivers essential substances for fetal sustenance through the spiral arteries to the placenta. Maternal blood bathes the chorionic villi that house the fetal vessels, transferring substances across the villi into the fetal veins, where they are circulated throughout the fetal system. Fetal waste is returned to the fetal arteries, housed in the chorionic villi, transferred to the maternal blood bathing the villi in the placenta, and removed by the maternal circulation through the spiral veins.

To meet the needs of the fetus as it grows, the maternal uterine spiral arteries must undergo a transformation that allows them to perfuse the placenta effectively throughout gestation. Normally, transformation of the maternal spiral arteries is established during placental development in the early weeks of pregnancy.[19] In preeclampsia, however, this transformation is impaired, resulting in reduced placental perfusion that triggers an inflammatory response, resulting in the release of proinflammatory cytokines and creating an imbalance of proangiogenic and antiangiogenic factors.

In the second half of pregnancy, this inflammatory process produces the classic signs and symptoms that form the basis for the preeclampsia diagnosis.[19, 20] The endothelial lining of blood vessels throughout the maternal circulation is damaged, causing systemic vasoconstriction and, in turn, hypertension.[20] The damaged endothelium increases the likelihood of clot formation at the sites of endothelial damage. Products of coagulation are consumed in the formation of clots, reducing their availability in the maternal circulation and increasing the risk of bleeding. Increased vascular permeability causes fluid to move from the maternal capillaries into the interstitial space, producing edema, hemoconcentration, and reduced blood flow. Edema and vasoconstriction impair liver function, decreasing the availability of albumin and key coagulation products. Reduced renal blood flow activates the renin–angiotensin–aldosterone system, worsening vasoconstriction. Increased permeability of the renal glomerular capillaries allows protein, primarily albumin, to pass into the glomerular filtrate and be excreted in urine. Throughout the maternal system, endothelial dysfunction and reduced perfusion increase blood pressure and cause symptoms such as headache, visual disturbances, and epigastric pain.

PREECLAMPSIA RISK FACTORS

A number of preexisting and pregnancy-related risk factors have been associated with preeclampsia (see Table 2[1, 20-22]). A recent meta-analysis evaluated the relationship between preeclampsia and 14 clinical factors that can be assessed at or before 16 weeks' gestation: personal history of preeclampsia, prior placental abruption, prior fetal growth restriction, prior stillbirth, nulliparity, advanced maternal age (variously defined as over 35 and over 40), overweight (BMI above 25 kg/m^2) or obesity (BMI above 30 kg/m^2), chronic hypertension, prepregnancy diabetes mellitus (type 1 or type 2), chronic kidney disease, systemic lupus erythematosus, antiphospholipid antibody syndrome, assisted reproduction, and multifetal pregnancy.[21] Other than prior fetal growth restriction, all clinical factors were significantly associated with an increased risk of preeclampsia. Factors with the highest pooled unadjusted relative risks for preeclampsia were a personal history of preeclampsia and chronic hypertension.[21]

Although we are currently unable to predict preeclampsia, information related to these risk factors should be collected at a woman's first prenatal visit. This information can assist health care providers in identifying women who may be at elevated risk for preeclampsia and may benefit from increased surveillance or preventive measures. For example, women with a history of early-onset preeclampsia that resulted in delivery at less than 34 weeks' gestation or preeclampsia in more than one prior pregnancy may benefit from daily low-dose aspirin therapy initiated late in the first trimester.[1] The absence of risk factors, however, does not guarantee that an otherwise healthy woman will not develop preeclampsia during or immediately following her pregnancy. In fact, most cases of preeclampsia are diagnosed in healthy nulliparous women.[1] For this reason, it is

of utmost importance that all women be monitored routinely for preeclampsia throughout pregnancy and in the postpartum period. Additionally, pregnant women should be informed about preeclampsia warning signs so they can be active participants in their own health care.

EMERGING APPROACHES TO PREECLAMPSIA

The care of women with preeclampsia is significantly limited by the lack of cost-effective screening tests that can reliably identify risks early in pregnancy and interventions that can effectively prevent or treat all variations of preeclampsia, which differ in presentation, onset, and severity.[20] However, as preeclampsia pathogenesis is better defined, targets for screening, diagnosis, prevention, and treatment are emerging.

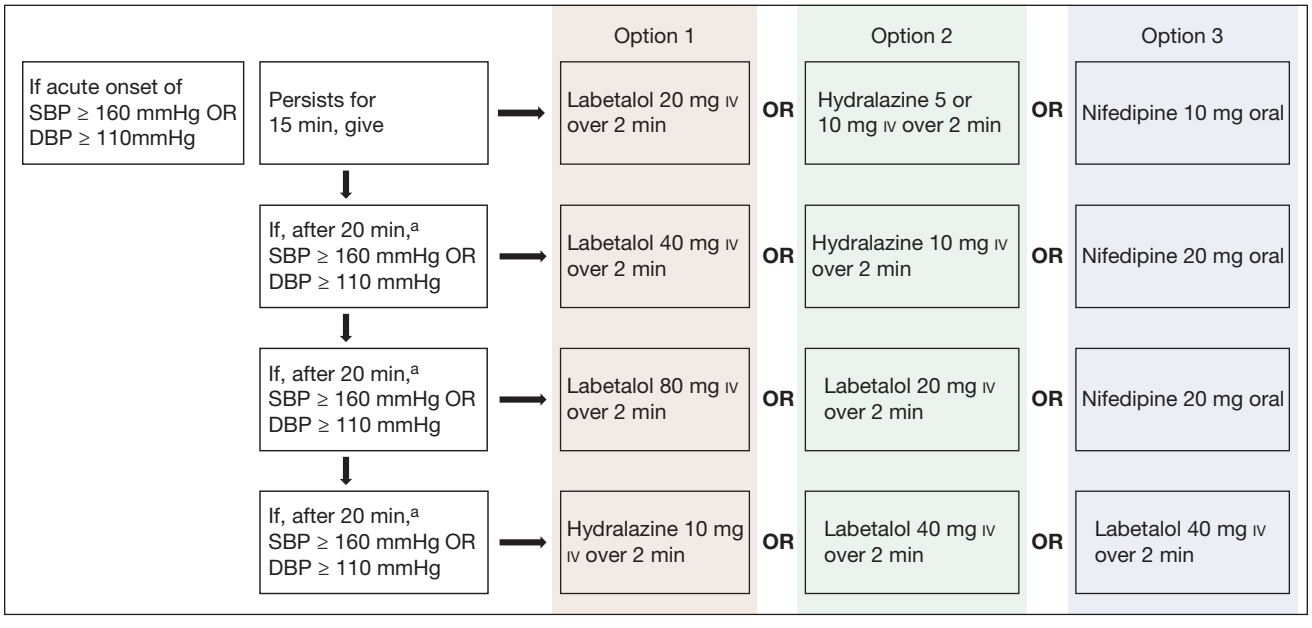
Preeclampsia screening. Ideally, care of pregnant women should include selective and sensitive screening tests that identify preeclampsia risk factors before the development of overt, later-stage maternal signs and symptoms indicative of widespread pathology. Emerging screening tests are focusing on pathologies that occur in the early stages of pregnancy and are related to biomarkers in maternal blood, as well as to placental development and perfusion.[23, 24]

Biomarkers in maternal blood. Genetic alterations associated with angiogenic balance[25] and inflammation[26] provide support for continued investigation into placental pathology and biomarkers for risk detection. Certain proteins found to be overexpressed in first- and second-trimester blood samples drawn from women who later developed preeclampsia have been identified as potential preeclampsia biomarkers.[27] When used alone, these proteins have little value for accurately predicting preeclampsia, but they may be helpful in clinical practice when used in conjunction with other tests.[28] Although single biomarkers show promise, the greatest potential for early identification of preeclampsia—in the first or second trimester, before the onset of signs and symptoms—lies in combining biomarkers and clinical parameters.[29]

Placental perfusion. The ability to measure functional placental response and substances released in response to reduced placental perfusion may provide a means to diagnose preeclampsia before the onset of signs and symptoms in later pregnancy. Decreased placental perfusion is a common finding in women diagnosed with preeclampsia and can be measured using uterine artery Doppler velocimetry.[30] However, the value of measuring placental perfusion in early pregnancy may be limited,[31] even when coupled with maternal preeclampsia biomarkers, which are not necessarily universal across all preeclampsia phenotypes.[20]

Immune factors are also a target of investigation in preeclampsia. It is well established that maternal immune tolerance is necessary to allow the genetically distinct fetus to grow and develop without rejection, and there is evidence of increased risk of preeclampsia when the interval between pregnancy and first vaginal

Figure 2. First-Line Pharmacologic Management of Acute Hypertension in Pregnancy and the Postpartum Period[41]



DBP = diastolic blood pressure; SBP = systolic blood pressure.

[a] In the case of labetalol, if BP remains elevated after 10 minutes, continue to the next step.

Note: If BP threshold is reached, continue close BP monitoring. If BP remains elevated, seek consultation for additional antihypertensive treatment.

Preeclampsia Resources

Foundations and Organizations

- Preeclampsia Foundation: www.preeclampsia.org
- March of Dimes: www.marchofdimes.org
- American Congress of Obstetricians and Gynecologists: www.acog.org
- Association of Women's Health, Obstetric and Neonatal Nurses: http://www.awhonn.org

Reports and Books

- Royal College of Obstetricians and Gynaecologists and the Royal College of Midwives. *Hypertension in pregnancy: the management of hypertensive disorders during pregnancy*. London: 2010 Aug. www.ncbi.nlm.nih.gov/books/NBK62652/pdf/Bookshelf_NBK62652.pdf
- American College of Obstetricians and Gynecologists Task Force on Hypertension in Pregnancy. *Hypertension in pregnancy*. Washington, DC; 2013. www.acog.org/Resources-And-Publications/Task-Force-and-Work-Group-Reports/Hypertension-in-Pregnancy
- Larsen LW. *Zuzu's Petals: A True Story of Second Chances*. Boulder, CO: In the Telling Press; 2011.
- Woodwell WH, Jr. *Coming to Term: A Father's Story of Birth, Loss, and Survival*. Jackson, MS: University Press of Mississippi; 2001.

exposure to paternal semen is short, as when the father is a new sexual partner, or the woman has been using a barrier contraceptive with an established sexual partner until shortly before becoming pregnant.[32] The association between preeclampsia and inflammation point to proinflammatory cytokines, such as C-reactive protein[33] and interleukin-6,[34] as potential biomarkers of disease.

Potential preeclampsia prophylaxis. Because oxidative stress as a consequence of poor placental perfusion has been implicated in preeclampsia, it's been suggested that antioxidants, such as lutein, may play a role in preeclampsia prevention.[35] Two 2015 Cochrane reviews, however, found no benefit to routine antioxidant supplementation with vitamins C or E for this purpose, and the ACOG Task Force does not recommend routine use of either to prevent preeclampsia.[1, 36, 37]

For women at high risk for preeclampsia, use of low-dose aspirin started at or before 16 weeks' gestation is believed to target placental pathology by balancing the endothelial products prostacyclin, a vasodilator, and thromboxane, a vasoconstrictor that induces platelet aggregation.[38, 39] The ACOG Task Force recommends that the use of aspirin for preeclampsia prevention be limited to women with a history of early-onset preeclampsia that required delivery before 34 weeks' gestation or preeclampsia in more than one prior pregnancy.[1]

NURSING MANAGEMENT OF PREECLAMPSIA

The absence of definitive strategies to prevent preeclampsia limits nurses' ability to provide anticipatory guidance and teach patients evidence-based approaches for reducing preeclampsia risk. Nurses can, however, encourage all women planning pregnancy to work toward achieving a healthy body weight and consume a healthy diet replete with recommended nutrients. Current guidance is to limit foods with added sugars and those that are high in fat and to eat a variety of fruits, grains, vegetables, low-fat or fat-free dairy, and proteins, avoiding such sources of mercury as shark, swordfish, mackerel, and tilefish, and limiting the consumption of another source, tuna, to less than six ounces per week.

Current screening approaches include preeclampsia risk identification through the collection of demographic information and comprehensive personal and family history. Risks related to a short time interval between exposure to paternal semen and pregnancy can be assessed through a review of sexual history that includes duration of the sexual relationship and use of a barrier birth control method. A personal or family history of preeclampsia may provide an opportunity to discuss potential screening and diagnostic strategies.

Warning signs of preeclampsia can occur during the second half of pregnancy and in the postpartum period. These include severe headache, right upper quadrant epigastric pain, nausea, visual changes (such as loss of visual fields or seeing spots), difficulty breathing, and swelling in areas such as the face or hands.[40] Nurses play a key role in teaching pregnant women about these subtle, subjective warning signs.

Accurate blood pressure measurement is crucial. Ask the patient to sit comfortably—with her back supported, her feet flat on the floor, and her arm at heart level. Select the proper cuff size based on the patient's arm circumference, and measure blood pressure on the same arm each time.

Changes in body weight may indicate fluid imbalance associated with generalized edema. A weight gain of more than three to five pounds in one week, reduced urine output, or the presence of edema, including pulmonary edema, suggest preeclampsia-associated fluid imbalance, especially during the second half of pregnancy.

Preeclampsia diagnosis and surveillance. Blood and urine tests may provide objective evidence of preeclampsia during pregnancy and in the postpartum period. Upon diagnosis of preeclampsia, maternal and fetal surveillance is initiated to determine progression and severity. Maternal assessment includes evaluation of subjective symptoms, serial blood pressure measurement, physical assessment, and laboratory analyses to guide intervention. Fetal surveillance includes serial nonstress testing to evaluate fetal oxygenation, ultrasound measurement of amniotic fluid volume as a proxy for fetal–placental perfusion, and estimation of fetal growth and gestational age. The nonstress test may be accompanied by a biophysical profile, scored based on ultrasound measurements of fetal breathing, body movements, muscle tone, and amniotic fluid level. Umbilical artery Doppler velocimetry, which measures blood flow in the umbilical cord or between the uterus and the placenta, may be indicated as a follow-up if there are concerns related to reduced placental perfusion.

Determining optimal timing of delivery. Gestational age at diagnosis and severity of preeclampsia are major factors in determining optimal timing of delivery, which is the only way to reverse preeclampsia that occurs during pregnancy. The ACOG Task Force recommends delivery for women with preeclampsia who are at or beyond 37 weeks' gestation or who are between 34 and 37 weeks' gestation and have preeclampsia with severe features.[1] In women between 20 and 34 weeks' gestation, preeclampsia with severe features is ideally managed in a facility with adequate maternal and neonatal intensive care resources. Because of the risk of preterm birth, care includes corticosteroid administration to enhance fetal lung maturity.

Pharmacologic treatment for severe sustained hypertension in pregnancy and the postpartum period is instituted when systolic blood pressure rises to or above 160 mmHg or when diastolic blood pressure rises to or above 110 mmHg. The goal is to stabilize blood pressure at 140–150/90–100 mmHg (see Figure 2[41]). If blood pressure is reduced to below established goals, perfusion to maternal organs and the fetus may be insufficient. Nursing management includes assessment of maternal response to antihypertensive therapy.

Magnesium sulfate may be used for seizure prophylaxis and control in women who have preeclampsia with severe features, or eclampsia.[1] Typical administration includes a loading dose of 4 to 6 g IV infused over a period of 15 to 20 minutes, followed by a maintenance dose of 1 to 3 g IV per hour. Nursing management includes assessment for magnesium toxicity, evidenced by loss of consciousness, absent deep tendon reflex activity, and a respiratory rate below 12 breaths per minute. As magnesium sulfate is excreted by the kidneys, urine output below 30 mL per hour increases the risk of toxicity.[42] Fluid replacement should be judicious, even with oliguria, as preeclampsia predisposes women to fluid imbalance.

Long-term risk of cardiovascular disease. Since preeclampsia is an established risk factor for cardiovascular disease, nurses should reinforce with patients the importance of long-term follow-up. The ACOG Task Force recommends that women with a history of either preeclampsia and preterm delivery or recurrent preeclampsia undergo yearly assessments that measure blood pressure, lipid levels, fasting blood glucose levels, and BMI.[1]

The ACOG Task Force recommends delivery for women with preeclampsia who are at or beyond 37 weeks' gestation.

CONCLUSION

KW's case highlights the progressive and multisystemic nature of preeclampsia. Although clinical management of preeclampsia continues to be symptom based, emerging approaches for the prediction and prevention of preeclampsia are continuously being evaluated for their potential use in evidence-based clinical care. In the interim, nurses continue to play a major role in the care and education of women with and at risk for preeclampsia. For helpful information to assist nurses, pregnant women, and preeclampsia survivors, see *Preeclampsia Resources*. ▼

For 150 additional continuing nursing education activities related to maternal–child topics, go to www.nursingcenter.com/ce.

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