

# Common pregnancy complication...complicated

By Sarah Henkel, LP, NR-EMTP, MS, and Rich Henkel, LP, NR-EMTP, BBA



*iStockphoto*

## Introduction

Preeclampsia occurs in about eight percent of pregnancies and is sometimes referred to as pregnancy-induced hypertension or toxemia of pregnancy. It is part of a spectrum of blood-pressure disorders that can affect pregnant women along with chronic and gestational hypertension. Considered to be on the more severe side of the spectrum, it can lead to eclampsia, which is the second leading cause of maternal death in the United States.

Paramedic and EMT students are often taught that the cure for both eclampsia and preeclampsia is delivery of the infant. While that is often true, the symptoms of preeclampsia can continue or even develop up to four weeks postpartum. It is estimated that between 14 and 33 percent of eclampsia cases actually manifest in this manner. Recognizing the signs and symptoms of preeclampsia in both the prenatal and postpartum setting and providing appropriate treatment is vital to decreasing the likelihood the symptoms will progress to eclampsia.

## Pathophysiology and risk factors

In the prehospital setting, preeclampsia

is defined as hypertension (pre-existing or gestational onset after 20 weeks) with diastolic blood pressure  $\geq 90$  mmHg and one or more of the following adverse conditions:

- Maternal symptoms: persistent, new or unusual headache, visual disturbances, persistent abdominal or right upper quadrant pain, severe nausea or vomiting, chest pain or dyspnea
- Maternal signs of end-organ dysfunction: eclampsia, severe hypertension, pulmonary edema or suspected placental abruption

In the hospital setting, patients may also present with proteinuria (protein in the urine), abnormal lab tests results or fetal morbidity.

Preeclampsia most typically manifests some time after the 20th week of pregnancy and resolves with delivery; however, it can develop postpartum. Preeclampsia is also called toxemia of pregnancy because it was once believed that a toxin produced by the mother in response to a foreign protein from the fetus was responsible for the symptoms. Although this is now known to *not* be the cause, the underlying causes of preeclampsia are still unknown despite a

significant amount of research. Essentially, the pathology responsible for the elevation of blood pressure is spasm of the blood vessels. Normally, the blood vessels of a pregnant woman have a diminished response to the effects of substances such as norepinephrine. In pregnancy-induced hypertension, resistance to vasospasm is somehow compromised. Thus, unrestricted vasospasm causes the blood pressure to increase. This affects how much blood can get to different parts of the body. Research has shown that in women with preeclampsia, blood flow to the kidneys, brain, liver, retina and placenta are decreased.

While no theories have been proven, researchers theorize that blood vessels, brain or nervous system factors, diet and genes may all play a role in the development of preeclampsia. For purposes of assessment and management, preeclampsia-eclampsia syndrome can be classified according to three stages: mild preeclampsia, severe preeclampsia and eclampsia.

### ***Mild preeclampsia***

This condition is characterized by a blood pressure reading of at least 140 mmHg systolic, or a systolic blood pressure 30 mmHg or diastolic blood pressure 15 mmHg diastolic above the patient's pre-pregnancy level. The blood pressure readings are taken on two occasions six hours apart, with special attention to the diastolic pressure, which reflects peripheral vasospasm. Pathologic changes in the kidneys produce proteinuria, oliguria (low output of urine) and edema. The kidneys have a diminished capacity to filter urine, lowering urine output and causing additional fluid retention, edema and weight gain.

### ***Severe preeclampsia***

Preeclampsia becomes severe when systolic blood pressure exceeds 160 mmHg or the diastolic pressure exceeds 110 mmHg. To help establish the diagnosis, two blood pressure readings are taken six hours apart after the woman has been on bed rest. Other symptoms are a marked increase in proteinuria, decreased urinary output, visual disturbances and marked hyperreflexia (overactive reflexes).

### ***Eclampsia***

If the patient's condition continues to deteriorate and edema worsens, she becomes eclamptic. Eclampsia is an acute, life-threatening complication of pregnancy. Eclampsia is characterized by tonic-clonic seizures, usually in patients with preeclampsia. Eclampsia includes any seizures and coma that occur during pregnancy but are not due to preexisting or organic brain disorders. Cerebral edema caused by preeclampsia predisposes the patient to the convulsions and coma. Signs and symptoms that may signal progression to eclampsia include: elevated body temperature, sudden rise in blood pressure, gastrointestinal symptoms and severe headache, blurred vision and other signs of increased central nervous system irritability.

The eclamptic seizure is typically characterized by four stages. In the *stage of invasion*, facial twitching can often be observed around the mouth. In the *stage of contraction*, tonic contractions render the body rigid; this stage may last about 15 to 20 seconds. The next stage is the *stage of convulsion*, when involuntary and forceful muscular movements occur; the tongue may be bitten and foam may appear at the mouth. The patient usually stops breathing and becomes cyanotic. This stage can last about one minute. The final stage is *coma*. When the patient awakens, she is unlikely to remember the event. In some rare cases, there are no convulsions and the patient progresses directly into a coma. Maternal mortality from eclampsia is high. The cause of death can be cerebral hemorrhage, circulatory collapse or renal failure. Infant mortality is also high due to hypoxia or development of acidosis in the fetus.

While the exact etiology of preeclampsia and eclampsia are unknown, there are several factors that appear to increase the risk for developing the disease. These are listed in **Figure 1**. Of these, women younger than 20 years of age and experiencing their first pregnancy seem to be at the highest risk.

**Figure 1**

#### **Risk factors for preeclampsia and eclampsia**

- Previous preeclampsia
- Pre-existing hypertension or diastolic BP  $\geq$  90 mmHg
- Pre-existing renal disease, proteinuria or diabetes
- First pregnancy or multiple pregnancies
- Obesity
- Family history of preeclampsia (mother or sister)
- Age  $\leq$  20 or  $\geq$  40 years
- Interpregnancy interval  $\geq$  10 years
- African American descent

## Clinical presentation

While the definition of preeclampsia is fairly straightforward, patients may present in a variety of ways. Since you will have no way of knowing if a patient has protein in her urine, you will have to rely on elevated blood pressure and the patient's symptoms. However, you will notice in **Figure 2** that you cannot count on hypertension as a sign in all patients.

<b>Figure 2</b>	
<i>Clinical presentation of preeclampsia/eclampsia and prevalence of symptoms at diagnosis</i> (adapted from tables in Ginzburg and Wolff, and Yancey et al)	
<b>Presentation</b>	<b>% of cases</b>
Headache	83
Hyperactive reflexes	64
Hypertension	86
Proteinuria	60
Edema (typically moderate pitting edema in the feet)	67
Visual changes	38
Epigastric pain/nausea/vomiting	15

### **Headache**

Headache can be mild or severe. It may be located anywhere on the skull, but is most commonly in the frontal lobe and may be described in a variety of ways, including throbbing, stabbing or sharp. The headache may be accompanied by changes in mental status or any of the other symptoms listed in the table above, or it may be the sole presenting symptom.

### **Hyperactive reflexes**

With preeclampsia, a woman's reflexes become unusually active. Increasing blood pressure will lead to increasing hyperreflexia, until uncontrollable seizures eventually result. Testing for this change is difficult in the field setting; in a clinic setting an overactive patellar response is a good indicator.

### **Visual changes**

Visual problems may occur because high

blood pressure stresses the retina, pushing it forward. In extreme cases, this can lead to retinal detachment and possibly blindness. Patients will typically complain of blurred or spotty vision or sensitivity to light.

## Treatment

In the prehospital setting, the treatment for preeclampsia begins with identifying that preeclampsia is the possible cause of the signs or symptoms. In the postpartum patient, that might not be your first thought, so obtaining a detailed patient history is imperative. Ask any woman of child-bearing age whether she is currently pregnant or has recently delivered a baby. If she is postpartum, find out details about her pregnancy, just as you would if you were assessing her prenatally. Based on the risk factors identified earlier, be sure to ask at least the following questions:

- How many times has she been pregnant (gravidity) and how many children does she have (parity)?
- Does she have other medical history?
- Have any women in her immediate family ever been diagnosed with eclampsia or preeclampsia?
- If she is postpartum:
  - Were there any complications during the pregnancy or following delivery?
  - Was her child born vaginally or by cesarean?
  - How many days ago was her child born?

Determining if the patient has a history of migraine headaches or if an epidural was administered during her delivery are also important findings. Both migraines and side effects from an epidural can cause headache, visual disturbances and nausea and vomiting, making them difficult to distinguish from preeclampsia. Both may even cause elevated blood pressure, depending on the patient's tolerance for pain.

During your patient history, consider the alternatives diagnoses listed in **Figure 3**. Ask questions about recent trauma, perform a blood glucose analysis to rule out hypoglycemia and perform any other diagnostics you have available to narrow down your diagnosis to

probable preeclampsia.

Once you have established that preeclampsia is a likely cause of her symptoms, focus on supporting the ABCs. Administer supplemental oxygen via nasal cannula, non-rebreather or BVM as appropriate. Be prepared to intubate or administer another advanced airway such as the Combitube or King Airway per your local protocol if the patient's condition progresses to eclampsia. Documenting changes in blood pressure will be important, especially during long transports. Obtain and document an accurate blood pressure reading to use as a baseline. Thereafter, monitor and record vital signs every five minutes. Make sure to obtain blood pressure readings from the same arm. Once the ABCs have been addressed, focus on minimizing stimulation to the patient. Talk in a low voice, avoid sounding panicked and dim the lights as much as possible. Do not transport with lights and sirens unless absolutely necessary. Transport the patient in the left lateral recumbent position or in the position of comfort, and be prepared for her condition to deteriorate.

Advanced care includes starting a large bore IV with normal saline TKO and cardiac monitoring. If the prenatal or postpartum patient begins to seize, magnesium sulfate is the drug of choice. It will likely stop the seizure and prevent additional seizures from occurring. Although controlled clinical trials support the effectiveness of magnesium

sulfate in preventing and treating eclamptic seizures, questions still exist as to its safety. There are concerns regarding the possibility of magnesium toxicity, which can ultimately lead to cardiac arrest. In addition, there are reports that in some patients, eclamptic seizures do not cease even with elevated levels of magnesium sulfate, suggesting that magnesium sulfate is not effective in treating all cases of eclampsia. Even still, magnesium sulfate appears to be the most often used first line drug in treating eclamptic seizures.

Just as the actual causes of preeclampsia and eclampsia are unknown, the mechanism of action for magnesium sulfate remains unclear. Several possible mechanisms of action have been proposed, including acting as a vasodilator, with actions either peripherally or in the cerebral circulation to relieve vasoconstriction, protecting the blood-brain barrier to decrease cerebral edema formation, and acting as a central anticonvulsant.

The typical dose of magnesium sulfate is four grams administered over three minutes, but refer to your local protocols regarding administration. While magnesium sulfate is being administered, the patient must be monitored frequently to assess the respiratory rate. If the respiratory rate becomes too depressed leading you to suspect magnesium overdose, calcium gluconate can be used to counteract the effects. If your system does not carry magnesium sulfate, diazepam or midazolam can also be used as anticonvulsants; however, if the patient is currently pregnant, these medications pose a higher risk to the fetus and they may not be as effective in stopping the seizures.

Depending on your local protocols, other therapies for eclampsia may include hypotensive drugs such as labetalol or hydralazine to reduce blood pressure, or sedatives such as phenobarbital to manage central nervous system irritability.

### Case studies

Let's consider the following patient. A previously healthy 37-year-old woman (gravida 3, parity 1) had an unremarkable pregnancy until the 28th week. She was diagnosed at that time with gestational diabetes

**Figure 3**

*Differential diagnosis for postpartum seizure*

(adapted from table in Ginzburg and Wolff)

- Postpartum eclampsia
- Epilepsy
- Hypoglycemia
- Drug or alcohol induced withdrawal or poisoning
- Head trauma and intercranial hemorrhage
- Brain tumor or abscess
- CVA
- Meningitis

that was subsequently controlled by diet. Blood pressure readings were normal throughout the pregnancy and she had no medical conditions. At 35 weeks, she developed mild pitting edema in her ankles. At 36 weeks she vaginally delivered a healthy baby. The patient was discharged one day post delivery.

On postpartum day five, the patient presented to the emergency department with a one day history of a gradual onset throbbing headache, photophobia and three episodes of vomiting. Her blood pressure was 205/105 mmHg; all other vitals were unremarkable. Two hours and ten minutes later, she had a generalized seizure lasting two minutes. The seizure was terminated with diazepam and she was given medication to lower her blood pressure. Two hours after the initial seizure, the patient reported having no headache and her mental state was clear. Her blood pressure was 104/49 mmHg. Minutes later she had a second seizure, this one terminated with lorazepam. She was given a magnesium sulfate drip and transferred to intensive care. She remained in intensive care for two days, and then was discharged home on blood pressure medication.

While this patient presented to the ED, she could have easily called 9-1-1. If she had called 9-1-1 and you responded, what clues were there that this patient might be showing symptoms of preeclampsia or be at risk for developing eclampsia? The clues were:

- Pitting edema during pregnancy
- Gestational diabetes during pregnancy
- Five days postpartum
- Headache, photophobia and vomiting
- Significantly elevated BP

So let's consider a scenario where the patient does choose to access 9-1-1. Your ALS ambulance is called to a private residence for an unconscious pregnant woman. On arrival, you find a 34-year-old woman who is 28 weeks pregnant, conscious but confused. She is complaining of a headache and blurry vision. The husband reported hearing loud snoring while he and his wife were sleeping, and he was unable to wake her. There have been no complications to the pregnancy and she has no

medical problems.

You apply high flow oxygen via a non-rebreather mask due to the patient's confusion, and measure vital signs. Her pulse is 110, respirations 22, heart and lung sounds are normal, and blood pressure is 130/90 mmHg. You start a large bore IV of normal saline TKO, place her on the cardiac monitor (no abnormalities noted) and use a calm and reassuring voice as you attempt to gather additional patient history. You place the patient on your stretcher in the left lateral recumbent position, load her into your ambulance and dim the lights, then begin the 25 minute transport to the hospital without lights and sirens. Ten minutes into the transport, you again measure vital signs. Her pulse is 100, respirations 20, and blood pressure 148/94 mmHg. Moments later, the patient begins twitching around her mouth, and then develops a generalized tonic-clonic seizure accompanied by vomiting. You tell your partner to expedite the transport by turning on the lights and sirens as you begin reassessing the ABCs. Because she is already lying on her left side, vomit drains out the side of her mouth and you assist with your suction unit. Your system does not carry magnesium sulfate, so you administer the anticonvulsant you have available, diazepam 5 mg IV. This stops the seizure. Eight minutes later the patient has a second seizure for which you administer another 5 mg of diazepam. The second seizure does not stop. Having no other anticonvulsants available, you carefully monitor the patient's airway and transfer care of the still seizing patient to the staff in the ED.

## Conclusion

Preeclampsia and eclampsia can present either pre- or postpartum, and pose significant risks to both mother and fetus. The diagnosis in the postpartum patient can be easy to miss, especially if a detailed patient history is not conducted. To differentiate these conditions from others, a detailed patient history is vital.

Eclampsia is a true, life-threatening emergency. Keeping the possibility of postpartum preeclampsia or eclampsia in mind when responding to female patients of child bearing age will help keep you from overlooking this possibility and prevent

the treatment from being delayed. Accurate assessment and rapid treatment is the key to successfully caring for these patients.

**This article is provided for education only. Always consult with your medical director and follow your local protocols in making treatment decisions.**

## References

<http://medical-dictionary.thefreedictionary.com/preeclampsia>

[http://bodyandhealth.canada.com/channel\\_condition\\_info\\_details.asp?channel\\_id=2049&disease\\_id=107&relation\\_id=37908](http://bodyandhealth.canada.com/channel_condition_info_details.asp?channel_id=2049&disease_id=107&relation_id=37908)

Bushnell, Cheryl and Monique Chireau. (2011). Preeclampsia and stroke: Risks during and after pregnancy. *Stroke Research and Treatment, 2011* [electronic version]. Accessed April 21, 2012, from <http://www.hindawi.com/journals/srt/2011/858134/>.

Caroline, Nancy L. (2008). *Emergency Care in the Streets* (6th ed., pp. 35.4-35.11). Sudbury: Jones and Bartlett.

Chiandiramani, Manju et al. (2007, Sept/Oct). Modern management of postpartum hypertension. *Trends in Urology, Gynecology and Sexual Health* [electronic version]. Retrieved April 21, 2012, from [www.tugsh.com](http://www.tugsh.com).

Euser, Anna and Marilyn Cipolla. (2009, Feb.) Magnesium Sulfate for the Treatment of Eclampsia. *Stroke, 40*, 1169-1175 [electronic version]. Retrieved April 27, 2012 from <http://stroke.ahajournals.org/content/40/4/1169.full>.

Ginzburg, Val and Bryan Wolff. (2009, Feb). Headache and Seizure on postpartum day 5: late postpartum eclampsia. *Canadian Medical Association Journal, 180 (4)* 425-428.

Lim, Kee-Hak. (2011, Nov). Preeclampsia. *Medscape Reference*. [electronic version]. Retrieved April 21, 2012, from <http://emedicine.medscape.com/article/1476919-overview>.

Veltkamp, R. et al. (2000, August). Late onset postpartum eclampsia without pre-eclamptic prodromi: clinical and neuroradiological presentation in two patients. *Journal of Neurology, Neurosurgery and Psychiatry, 69*, 824-827.

Yancey, Lynne, et al. (2011). Postpartum preeclampsia: Emergency department presentation and management. *The Journal of Emergency Medicine, 40 (4)*, 380-384.

## About the authors

*Sarah Henkel holds a master's degree in safety and emergency management and is a Texas-licensed and nationally registered paramedic. She has been working in the field of EMS for nine years. Sarah currently works for the Arlington Fire Department in the role of special event emergency planner. Rich Henkel holds a bachelor of business administration and is a Texas-licensed and nationally registered paramedic. He has been working in the field of prehospital care for more than 15 years and is the program director for the EMERG EMT program.*