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Postpartum Emergencies: Headache, Hypertension, Eclampsia, And Cardiomyopathy

It is Friday evening in the ED, and your first patient is a 42-year-old woman whose chief complaint is headache that began at around 9 AM. She is a gravida 3, para 2-0-1-2 with a history of a previous C-section but no other significant past medical history. On further examination, you establish that 6 days earlier she had a C-section delivery with spinal anesthesia for a 36-week pregnancy because of premature rupture of membranes. Otherwise, this pregnancy and previous pregnancies were uneventful, and mother and baby went home on post-op day 4. Her headache has been severe and continuous for the past 6 hours, unrelieved with ibuprofen taken 3 hours earlier. Pain is now 10/10, and she had 2 episodes of vomiting shortly after her arrival in the ED. There are no visual symptoms, fever, chills, or focal weakness. Her triage blood pressure is 170/85. The ED is busy, and your focus is on throughput, but this patient has a concerning differential diagnosis that includes postdural puncture headache, subarachnoid hemorrhage, and venous sinus thrombosis. You are also concerned about the blood pressure, and though it may be elevated because of her pain, you wonder if you should consider postpartum hypertension.

Later in your shift around 4 AM, a 40-year-old woman is brought in by ambulance with a chief complaint of shortness of breath. The nurse calls you to the bedside because the patient has labored respirations. Her shortness of breath has become more severe over the past 3 days, and she is also complaining of cough and orthopnea. She delivered twins by C-section 2 weeks ago, and the pregnancy was complicated by preeclampsia. She has no significant past medical history. Her blood pressure is 180/120 mm

August 2010
Volume 12, Number 8

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CME Objectives

Upon completion of this article, you should be able to:

1. Recognize and treat common postpartum emergencies.
2. Identify the presentation, pathophysiology, and treatment of late postpartum preeclampsia/eclampsia.
3. Know the differential diagnosis, imaging modalities, and treatment for patients with postpartum headache.
4. Recognize the risk factors for postpartum cardiomyopathy and its common presenting signs and symptoms.

Date of original release: August 1, 2010
Date of most recent review: July 10, 2010
Termination date: August 1, 2013
Medium: Print and online
Method of participation: Print or online answer form and evaluation
Prior to beginning this activity, see "Physician CME Information" on page 16.

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Hg, heart rate 136 beats per minute, respiratory rate 32 breaths per minute, temperature 37.4°C (99.32°F), and pulse oximetry 94% with a 100% nonrebreather mask. On examination, she has jugular venous distention, bibasilar rales, an S3 gallop, and trace pedal edema. The patient clearly appears to have acute pulmonary edema. Your question is, why?

Postpartum emergencies often present in the emergency department (ED) and include headache, eclampsia, infection, heart failure, and hypertension. The incidence of these conditions, and of late postpartum eclampsia (LPPE) in particular, appears to be increasing.[1] Although the reasons for this are unknown, they may be related to earlier discharge from the hospital after delivery and increased reliance on the ED for after-hours care. Failure to recognize and treat these conditions can lead to disastrous consequences for the patient, including stroke, permanent brain damage, or death. Eclampsia is a leading cause of pregnancy-related mortality in the postpartum patient, and postpartum headache presents a major diagnostic challenge. This issue of *Emergency Medicine Practice* focuses on the most commonly encountered postpartum emergencies: headache, LPPE, hypertension, and cardiomyopathy.

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Available Online At No Charge To Subscribers

"Current Guidelines For Gastric Decontamination In Toxicologic Emergencies," www.ebmedicine.net/decontamination

Critical Appraisal Of The Literature

A literature search was carried out on articles published between 1980 and 2009 using Ovid MEDLINE®, The Cochrane Collaboration, ACP Journal Club, and PubMed. The search was limited to reports in English involving human subjects. It was performed using the keyword *postpartum* combined with e*clampsia, cerebral venous sinus thrombosis, hypertension, cardiomyopathy, HELLP syndrome, posterior reversible leukoencephalopathy syndrome,* and *headache.* Terms were then exploded to provide the widest search. Articles listed in the bibliographies of other relevant articles were also reviewed.

Well-designed clinical trials, prospective cohort studies, and aggregate studies using meta-analyses were considered the most credible of the published reports. Retrospective studies and case-control studies provided additional support, followed by panel consensus recommendations, case series, and case reports. Neither the American College of Emergency Physicians (ACEP) nor the American Congress of Obstetricians and Gynecologists (ACOG) has published practice guidelines relating to the emergency management of late postpartum complications.

Terminology

Postpartum Hypertension

Blood pressure rises progressively over postpartum days 1 through 5 and peaks on days 3 to 6 after delivery.[2,3] One possible explanation for this pattern is that the total body water and total body sodium that accumulated during pregnancy may become mobilized and shift from the extracellular fluid to the intravascular space.[4] What causes a recurrence of gestational hypertension or de novo postpartum hypertension is not known. The International Society for the Study of Hypertension in Pregnancy defines hypertension as "a diastolic blood pressure of 90 mm Hg or higher on 2 consecutive occasions at least 4 hours apart or a single diastolic blood pressure greater than 110 mm Hg."[5] A systolic blood pressure greater than 140 mm Hg or a diastolic blood pressure greater than 90 mm Hg[6] without proteinuria is also accepted as a definition of hypertension, with readings taken preferably on at least 2 occasions, 6 hours apart.[6]

Preeclampsia

Preeclampsia is defined as systolic blood pressure greater than 140 mm Hg or diastolic blood pressure greater than 90 mm Hg and proteinuria of 0.3 gram or greater in a 24-hour urine specimen. **(See Table 1.)** A random urine protein measurement of 30 mg/dL or 1+ on the dipstick is suggestive, but not diagnostic, of this criterion.[7] Preeclampsia typically occurs after the 20th week of gestation.

Eclampsia

Eclampsia is the occurrence of seizures in association with hypertension and proteinuria. Initially, seizures were thought to be the end result of preeclampsia, but it is now believed that they are just one manifestation of a multisystem disease process in which the blood, brain, lungs, or liver may be involved.[7] Late postpartum eclampsia is defined as eclamptic seizures that occur more than 48 hours after delivery but within 4 weeks post partum.

HELLP Syndrome

The HELLP syndrome includes **H**emolysis with a microangiopathic blood smear, **E**levated **L**iver enzymes (lactate dehydrogenase [LDH] and aspartate aminotransferase [AST]), and a **L**ow **P**latelet count.[8] **(See Table 2.)** Whether this syndrome is a severe form of preeclampsia or is a separate disease entity is a matter of controversy.

Peripartum Cardiomyopathy

Postpartum cardiomyopathy (also called peripartum cardiomyopathy [PPCM] or pregnancy-associated cardiomyopathy) is a syndrome of unknown etiology, with 4 criteria for the diagnosis.[9,10] **(See Table 3.)**

Epidemiology And Pathophysiology

Postpartum Hypertensive Disorders

The true prevalence of postpartum hypertension is not known. However, in a well-designed review of all maternal deaths in the United Kingdom over a 2-year period, hypertensive disorders accounted for 15% of the deaths (20 of 134 cases).[11] Postpartum hypertension may represent the continuation of an antenatal or gestational hypertensive disorder or may occur de novo after delivery. Women with preeclampsia prior to delivery appear to be at increased risk for postpartum hypertension. In a retrospective review of 269 patients, divided between those with gestational hypertension and those with preeclampsia, Ferrazzani et al found that hypertension lasted approximately 2 weeks post partum in preeclamptic women (mean ± SD = 16 ± 9.5 days).[12] Other risk factors for recurrence or continuation of hypertension after delivery were preterm delivery, a history of multiple pregnancies, and elevated levels of uric acid or blood urea nitrogen. Compared with women who had preeclampsia, fewer women with gestational hypertension had postpartum hypertension and their hypertension was of shorter duration (mean ± SD = 6 ± 5.5 days).[12]

Antepartum preeclampsia is a well-recognized clinical entity. There is no clearly defined syndrome of postpartum preeclampsia, although it is mentioned in one retrospective series.[13] There are multiple case reports of patients with postpartum hypertension and proteinuria who go on to have seizures and thus can be considered to have LPPE.

Aside from case reports, there are no data on either the incidence or the natural history of postpartum preeclampsia. Data on LPPE, however, are available. For instance, although the overall incidence of eclampsia is decreasing, the relative incidence of LPPE is rising.[1] LPPE now represents about 13% to 16% of all cases of eclampsia.[14-17] Reasons for this change in epidemiology may include early discharge after delivery and hence failure to detect the postpartum rise in blood pressure that peaks 5 to 6 days after delivery.[14,15] In one case series of 54 women, 40% of women with LPPE did not manifest the classic prodromal signs of preeclampsia.[16] On the other hand, in one retrospective chart review of 24 women with LPPE in which data on prodromal symptoms prior to seizure were collected, 22 women had 1 warning sign or symptom and 12 women had 2. Of concern, however, is that only one-third of these women sought medical care for their prodromal complaints prior to their seizure, and 6 out of 7 of this group were discharged without treatment by the ED provider.[17] In a prospective study in the

Table 1. Criteria For The Diagnosis Of Postpartum Preeclampsia

Systolic blood pressure ≥ 140 mm Hg
Or
Diastolic blood pressure ≥ 90 mm Hg
And
Proteinuria ≥ 0.3 g in a 24-hour urine specimen*

* A random urine dipstick reading of 1+ is suggestive but not diagnostic of this criterion.

Table 2. Criteria For The Diagnosis Of HELLP Syndrome[7]

- Microangiopathic hemolytic anemia
- Schistocytes (helmet cells) on peripheral smear
- Platelet count ≤ 100,000/mm^3
- Elevated liver enzymes
- LDH ≥ 600 IU/L, AST ≥ 70 IU/L, bilirubin ≥ 1.2 mg/dL

Abbreviations: AST, aspartate aminotransferase; LDH, lactate dehydrogenase.

Table 3. Criteria For The Diagnosis Of Peripartum Cardiomyopathy[8,9]

- Development of heart failure in the last month of pregnancy or within 5 months of delivery
- Absence of an identifiable cause for the heart failure
- Absence of recognizable heart disease prior to the last month of pregnancy
- Left ventricular systolic dysfunction

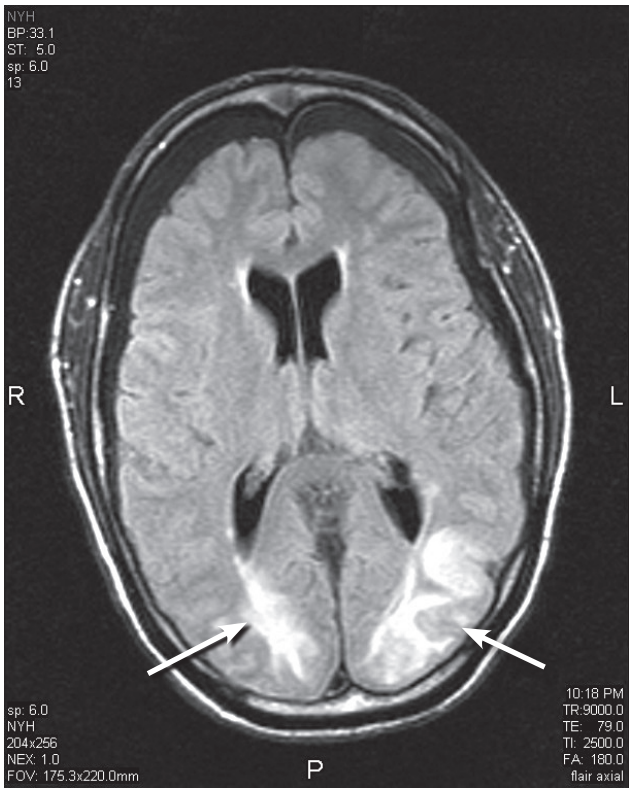
United Kingdom of a cohort of 383 patients, most cases of postpartum eclampsia occurred in the hospital; 38% of these women had no history of hypertension or proteinuria, suggesting that there was no prodrome of preeclampsia and hence the eclamptic seizures could not have been prevented.[18]

The pathophysiology of postpartum hypertension is probably multifactorial. In patients with prenatal chronic hypertension, their postpartum hypertension is a manifestation of their preexisting condition. Atrial natriuretic peptide (ANP) has been observed to rise during the first week post partum,[4] increasing sodium excretion, and is involved with the release of serum hormones that control blood pressure.[19] In one small study, women with de novo postpartum hypertension had significantly lower ANP levels than did normotensive women. This finding has not been confirmed in other studies; hence, ANP levels may be of little help when determining patient management.[20] The use of nonsteroidal anti-inflammatory drugs for postpartum analgesia may also iatrogenically contribute to hypertension by increasing sodium retention.[21]

Many theories have been proposed about the pathophysiology of preeclampsia/eclampsia, including immunologic mechanisms, an enhanced systemic inflammatory state, a relative increase in oxidant activity, increased placental debris in the maternal circulation, the presence of circulating antibodies against angiotensin II receptors, and a prothrombotic state.[22] Whatever the mechanism(s), the placenta is known to play a critical role in the pathophysiology of preeclampsia. Spiral arteries that remain narrow instead of developing into large, tortuous vascular channels can cause placental hypoperfusion.[23,24] Vascular, environmental, immunologic, and genetic factors also appear to be involved.[25] The ischemic placenta is believed to release factors into the maternal bloodstream that alter maternal endothelial cell function. All the clinical features of preeclampsia can be explained as responses to generalized endothelial dysfunction.[26,27]

Imaging studies are also helping to elucidate the pathophysiology of eclampsia. The characteristic magnetic resonance imaging (MRI) finding is symmetric, subcortical/cortical hyperintensity on the flair images, consistent with vasogenic edema.[28] **(See Figure 1 and Diagnostic Studies, page 7.)** Most brain images of women with eclampsia will show vasogenic edema. It has been hypothesized that severe vasogenic edema can lead to ischemia in conjunction with endothelial cell dysfunction and subsequent disruption of vascular integrity.[29] In eclampsia, the degree of brain edema correlates more strongly with markers of endothelial cell dysfunction than it does with the degree of hypertension,[30] supporting the theory that endothelial cells play a role in this disorder. In addition, the cerebral vessels in preeclampsia have lower resistance with elevated perfusion pressure. This places the patient at risk for overperfusion abnormalities and reinforces the so-called "hypertensive encephalopathy" of eclampsia.[31]

Figure 1: MRI Of Posterior Reversible Encephalopathy Syndrome



In this axial flair image, arrows point to bilateral parietal lobe hyperintensity that is characteristic of vasogenic edema in the posterior reversible encephalopathy syndrome (PRES), seen in women with eclampsia.

Note: PRES has been previously known as "reversible posterior leukoencephalopathy syndrome" and is often referred to in literature as such.

The HELLP Syndrome

Although the exact incidence of the HELLP syndrome has not been reported, it is commonly quoted that it develops in approximately 1 to 5 out of every 1000 pregnancies and in 5% to 20% of women with severe preeclampsia/eclampsia.[32] In one series of 437 women diagnosed with the HELLP syndrome, the disease presented postpartum in 30%, usually within 48 hours of delivery but also as late as 7 days post partum. Of these women, 80% had no evidence of preeclampsia before delivery.[32] Although the pathophysiology of the HELLP syndrome may be the same as that for preeclampsia, there is no clear evidence in the literature to confirm this possibility.

Peripartum Cardiomyopathy

The incidence of peripartum cardiomyopathy (PPCM) varies, depending on the geographical area

being studied. In the United States, the incidence is 1:15,000 deliveries, whereas in South Africa the incidence is 1:1000, and in Nigeria it is 1:100.[33,34] These widely differing statistics may reflect variations in the definition of the disease or cultural and dietary practices that may affect a woman's volume status and postpartum hypertension.[35,36] The etiology of PPCM is unknown, but postulated mechanisms include inflammatory cytokines,[37] myocarditis,[37,38] an abnormal immune response,[5,39] familial disease,[40] and gestational hypertension.[41] Risk factors for PPCM are listed in **Table 4**.

Differential Diagnosis

Headache can be a premonitory symptom of LPPE or other potentially serious problems, although there are also many benign explanations for headaches in the postpartum period, including the stress of motherhood, anxiety, sleep deprivation, hormonal changes, and increased caloric needs for lactation coupled with irregular meals. In a retrospective review of 95 women with severe postpartum headache, 39% were diagnosed with tension headache, 24% with preeclampsia/eclampsia, 16% with postdural puncture headache, 11% with migraine, and the remaining 10% with serious conditions such as intracranial hemorrhage, a mass, or cerebral venous sinus thrombosis.[49] When the headache is accompanied by hypertension and proteinuria, the working diagnosis should be preeclampsia until proved otherwise. However, diagnosis can be difficult if the patient has a recent history of spinal or epidural anesthesia at delivery, has a prenatal history of hypertension or headache, or has had a rise in catecholamines secondary to pain and stress. Case reports of eclampsia accompanied by postdural puncture headache,[50] intracranial hemorrhage, and cerebral venous sinus thrombosis misdiagnosed as preeclampsia[51,52] exemplify this challenge.

Cerebral venous sinus thrombosis is a relatively rare type of cerebrovascular accident due to thrombosis of the cerebral veins or dural sinus. Headache is the main presenting symptom early in its course. Pregnancy and the puerperium constitute risk factors, presumably owing to the hypercoagulable state of pregnancy and/or an inherited genetic thrombophilia.[53,54] After delivery, women are susceptible to the same headache-inducing conditions that are present in the general population, such as embolic stroke, carotid or vertebral artery dissection, infections, vasculitis, or tumor. **Table 5** includes a list of conditions to consider in the patient with postpartum headache.

Table 4. Risk Factors For Development Of Peripartum Cardiomypathy[42-48]

- Maternal age above 30
- African descent
- Multiple fetuses
- Preeclampsia, eclampsia, or postpartum hypertension
- Maternal cocaine abuse
- Long-term tocolytic therapy
- Selenium deficiency

Table 5. Differential Diagnosis Of Postpartum Headache[49-52]

Diagnosis	Signs and Symptoms
Central nervous system infection	Fever, leukocytosis, meningismus, altered mental status
Subarachnoid or intracerebral hemorrhage	Thunderclap onset, meningismus; may have focal neurologic findings
Vasculitis	Hemolysis, abnormal renal and liver function tests
Cerebral venous sinus thrombosis	Symptoms usually progressive; may have focal neurologic findings and/or signs of intracranial hypertension
Carotid or vertebral artery dissection	Isolated Horner's syndrome; other neurologic findings
Acute ischemic stroke	Acute onset of focal neurologic findings
Preeclampsia	Elevated blood pressure, ± proteinuria, visual disturbances, nausea, abdominal pain
Metabolic abnormality	Abnormal chemistry profile
Acute glaucoma	Injected eye, iritis, hazy cornea, blurry vision, nausea
Pseudotumor cerebri	Visual complaints, papilledema
Tumor	Pain on awakening, may be worse with Valsalva, abnormal mental status or neurologic exam
Postdural puncture or post spinal anesthesia headache	Epidural anesthesia at delivery, exacerbated by standing
Trigeminal neuralgia	Brief, lancinating pain in distribution of the trigeminal nerve branches
Sinusitis	Nasal congestion, sinus tenderness
Migraine	History of migraines; symptoms typical of usual migraine
Tension	Pressing or tight sensation; bilateral, mild to moderate; worsens as the day progresses

The most common presenting complaint in patients with PPCM is dyspnea. Other symptoms are those typically seen in heart failure, but early in its course PPCM may be manifest only as cough, generalized fatigue, chest discomfort, abdominal pain, and/or pedal edema. The differential diagnosis includes upper or lower respiratory infection, pulmonary embolism, myocardial ischemia, and postpartum fluid overload.

Prehospital Care

Prehospital care post partum is largely supportive, with treatment aimed at the underlying symptoms, which might include altered mental status, possible cerebrovascular accident, seizure, acute pulmonary edema, dyspnea, or chest pain. Management of seizures in the postpartum patient should be according to standard seizure protocol: check the ABCs, provide intravenous (IV) access, measure blood glucose, and administer IV benzodiazepines. Magnesium sulfate is available in some regions for prehospital treatment of patients with eclampsia who are pregnant. Magnesium is also used to treat seizures in patients with LPPE. Its use by prehospital caregivers should only be undertaken with online medical control, since the diagnosis of eclampsia in the postpartum patient may not be as straightforward as it is ante partum. Patients should be transported to the nearest appropriate facility; in the case of LPPE, that would ideally be one with the capability for rapid neuroimaging.

Emergency Department Evaluation

A complete set of vital signs should be obtained for all patients who present to the ED post partum. Since eclampsia can present up to a month after delivery and with only minimally elevated blood pressure, careful and repeated blood pressure assessment is important if the initial blood pressure at triage is elevated. As noted earlier, patients with LPPE can often have few or no symptoms yet rapidly progress to seizures.[14,16,18] Therefore, patients with even mildly elevated blood pressure or headache should be triaged as emergent.

The patient who presents post partum will often have a variety of signs and symptoms, many referable to her postpartum state and others to her presenting problem. Although the history should focus on the chief complaint, a review of systems is helpful because preeclampsia, the HELLP syndrome, and cardiomyopathy often have a plethora of associated symptoms. Essential aspects of the history include information on any complications of the most recent pregnancy, labor, or delivery; the type of anesthesia used; the postpartum course; and any previous pregnancies and pregnancy-related conditions. Careful attention should be paid to getting an accurate blood pressure reading and reassessing the blood pressure at regular intervals. A check of deep tendon reflexes is also useful, since patients with preeclampsia typically have hyperreflexia, with the degree often correlating with the severity of central nervous system hyperirritability.

Headache

As for any patient with headache, the postpartum patient requires a careful history and physical examination. Important questions are as follows:

1. Is this a new headache?
2. Is it worse than the patient's typical headache?
3. Is it sufficient in degree or symptoms to be of concern to the patient?
4. What medications were taken to relieve the headache, and when?
5. Was the onset sudden or gradual or associated with exertion or position?
6. Were there any accompanying changes in mental status, vision, or motor function?
7. Does the patient have fever, neck pain, or signs of an infectious disease?

The physical examination should include evaluations for meningismus and papilledema, as well as a careful neurologic assessment of mood, mental status, reflexes, and the presence or absence of localizing signs. Patients with accompanying hypertension, as defined previously, should be considered to have preeclampsia and should be managed as outlined in the Treatment section **(see page 10)**.

Patients who have had spinal or epidural anesthesia for delivery may have a headache due to a slow leak of cerebrospinal fluid (CSF) from the dural puncture site, which leads to a contraction of the subarachnoid space. It is commonly taught that pain from a CSF leak (which is constant, dull, and throbbing) usually occurs within 48 hours of the procedure and is exacerbated by upright posture and relieved with supine posture, although there is no hard evidence to support this claim. Associated symptoms include nausea, vomiting, tinnitus, and visual disturbances. It is usually not relieved by analgesics and may last for 2 to 14 days without treatment.[55]

Preeclampsia, Eclampsia, And The HELLP Syndrome

Elevated blood pressure remains the hallmark of preeclampsia, but many patients will also have premonitory symptoms. **(See Table 6.)** It is important to inquire regarding the presence or absence of these symptoms, which include headache, confusion, visual disturbances, nausea, vomiting, and epigastric pain. Although the HELLP syndrome usually manifests within 48 hours of delivery, its presentation can be delayed, and many of these patients will appear seriously ill.[56] Signs to look for on physical examina-

tion include altered mental status, focal neurologic deficits, reduced visual acuity, hyperreflexia, right upper quadrant or diffuse abdominal tenderness, peripheral edema, and purpuric rash.

Peripartum Cardiomyopathy

Patients with PPCM typically complain of fatigue, cough, orthopnea, dyspnea worsened by exertion, paroxysmal nocturnal dyspnea, and pedal edema. Symptoms are usually progressive. On physical examination, these patients may have either an elevated or a low blood pressure, tachycardia, rales, an S3 gallop, distended jugular veins, and peripheral edema.

Diagnostic Studies

Headache

For the patient whose headache, on the basis of signs and symptoms, clearly appears to be a postdural puncture headache and whose blood pressure is normal, further diagnostic testing may not be indicated. In other patients with headache who are not hypertensive, further diagnostic testing will depend on the differential diagnosis. Patients with typical migraine or tension headaches also do not require additional testing. However, patients with lateralizing neurologic signs, altered mental status, fever, or meningismus will require CT scanning, a complete blood count, a metabolic profile, and cultures (if toxicity is suspected). Lumbar puncture is indicated if meningitis or encephalitis is a possibility or if subarachnoid hemorrhage is a consideration and the CT proves to be nondiagnostic. Opening CSF pressures should be measured, since elevated pressures are diagnostic of pseudotumor cerebri. If cerebral venous sinus thrombosis is suspected, MRI and MR venography are the imaging modalities of choice.[57,58] **(See Figure 2.)**

Preeclampsia, Eclampsia, And The HELLP Syndrome

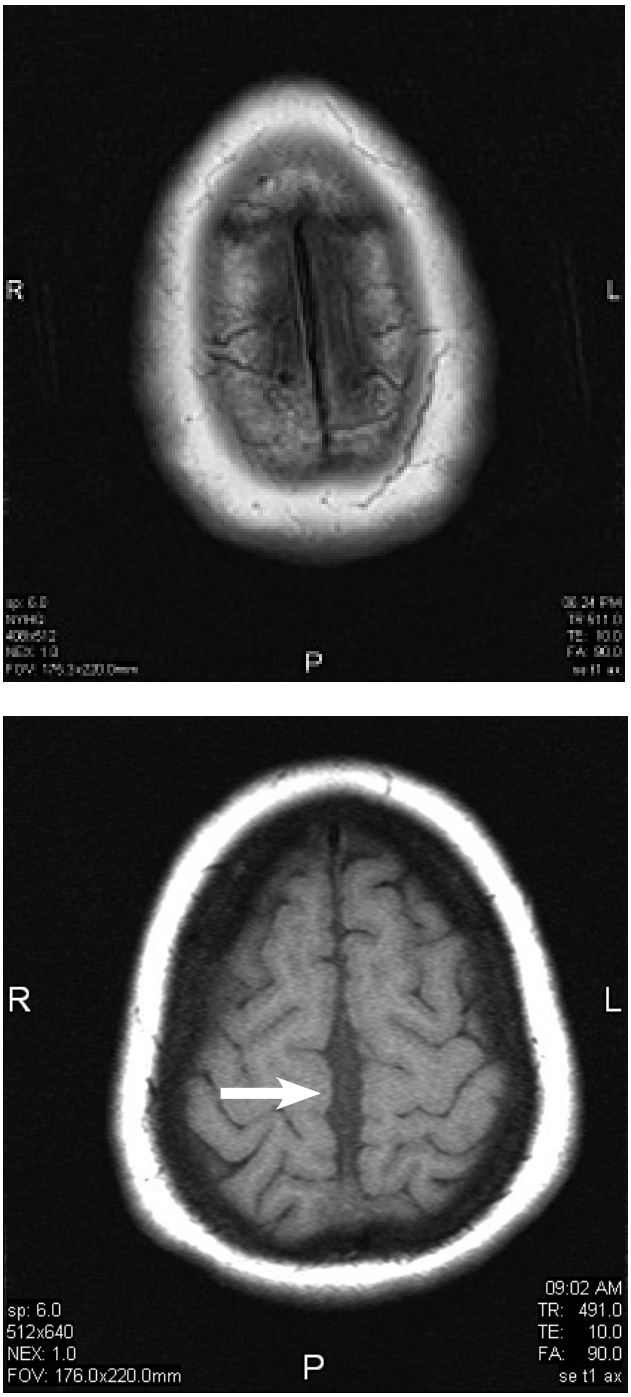
Since the diagnosis of antepartum preeclampsia is traditionally based on an elevated blood pressure and a level of at least 300 mg of protein in a 24-hour urine sample, the results of a urine dipstick test can be used as a preliminary indication of the presence or absence of proteinuria, ie, 1+ or greater on the dipstick roughly correlates to 300 mg in 24 hours.[4]

Table 6. Common Chief Complaints In Postpartum Preeclampsia[14]

- Headache
- Nausea
- Vomiting
- Visual disturbances
- Right upper quadrant or epigastric pain
- Malaise

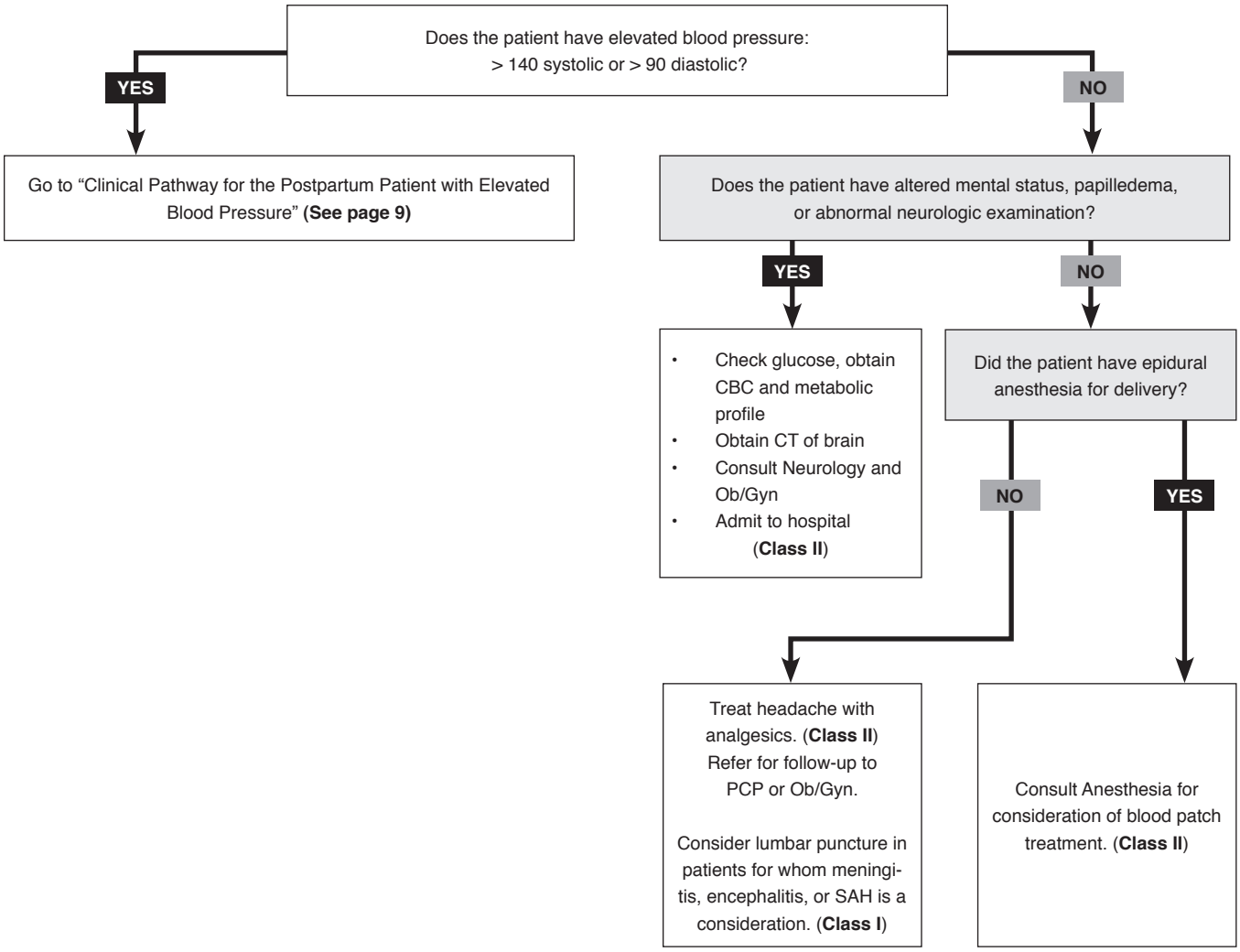
However, the usefulness of a urine protein determination in the postpartum patient is not clear, since the absence of proteinuria in a random specimen does not rule out preeclampsia. In 2 previously cited studies from Great Britain, of 383 women with postpartum eclampsia, 38% had neither proteinuria nor

Figure 2. MRI Of Normal Brain And MRI Of Brain Showing Sagittal Sinus Thrombosis



Top image: A normal brain MRI T1 axial image.
Bottom image: Sagittal sinus thrombosis, with arrow pointing to loss of normal flow void in the sagittal sinus.

Clinical Pathway For The Management Of The Postpartum Patient With Headache



Abbreviations: CBC, complete blood count; CT, computed tomography; Ob/Gyn, obstetrician/gynecologist; PCP, primary care provider; SAH, subarachnoid hemorrhage.

Class Of Evidence Definitions

Each action in the clinical pathways section of *Emergency Medicine Practice* receives a score based on the following definitions.

Class I
- Always acceptable, safe
- Definitely useful
- Proven in both efficacy and effectiveness

Level of Evidence:
- One or more large prospective studies are present (with rare exceptions)
- High-quality meta-analyses
- Study results consistently positive and compelling

Class II
- Safe, acceptable
- Probably useful

Level of Evidence:
- Generally higher levels of evidence
- Non-randomized or retrospective studies: historic, cohort, or case control studies
- Less robust RCTs
- Results consistently positive

Class III
- May be acceptable
- Possibly useful
- Considered optional or alternative treatments

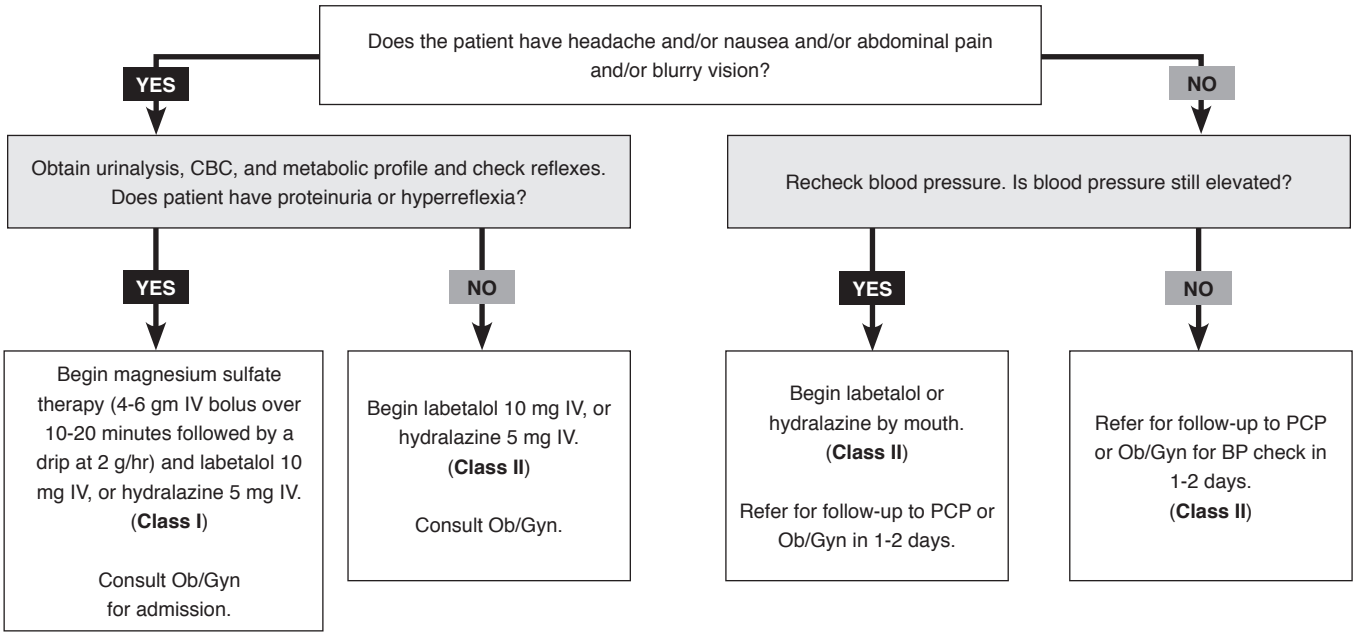
Level of Evidence:
- Generally lower or intermediate levels of evidence
- Case series, animal studies, consensus panels
- Occasionally positive results

Indeterminate
- Continuing area of research
- No recommendations until further research

Level of Evidence:
- Evidence not available
- Higher studies in progress
- Results inconsistent, contradictory
- Results not compelling

Significantly modified from: The Emergency Cardiovascular Care Committees of the American Heart Association and representatives from the resuscitation councils of ILCOR: How to Develop Evidence-Based Guidelines for Emergency Cardiac Care: Quality of Evidence and Classes of Recommendations; also: Anonymous. Guidelines for cardiopulmonary resuscitation and emergency cardiac care. Emergency Cardiac Care Committee and Subcommittees, American Heart Association. Part IX. Ensuring effectiveness of community-wide emergency cardiac care. *JAMA*. 1992;268(16):2289-2295.

Clinical Pathway For The Management Of The Postpartum Patient With Elevated Blood Pressure (> 140 Systolic Or > 90 Diastolic)



See Class of Evidence descriptions on page 8.

Abbreviations: BP, blood pressure; CBC, complete blood count; IV, intravenous; Ob/Gyn, obstetrician/gynecologist; PCP, primary care provider.

hypertension,[3,18] and of 67 women with antepartum preeclampsia and postpartum hypertension, 55% did not have proteinuria.[3] Furthermore, the presence of protein may also be a false-positive result, since it may be difficult to get a true clean-catch urine specimen after a recent vaginal delivery.

In a series of 437 women with the HELLP syndrome[32] and in a comprehensive review,[56] Sibai et al noted that 40% to 90% of patients have right upper quadrant or epigastric pain, 86% to 100% have proteinuria, and 82% to 88% have hypertension, although either or both of the latter 2 findings may be absent. Patients may be seriously ill at presentation (or shortly thereafter) as a result of disseminated intravascular coagulation, acute renal failure, pulmonary edema, subcapsular liver hematoma, or retinal detachment.[32,56] Therefore, blood tests should include a complete blood count and a peripheral blood smear to check for hemolysis, anemia, and thrombocytopenia; an assessment of electrolytes; and renal and liver function tests. Although it is generally recommended that a baseline magnesium level be obtained if the patient has already had seizures and/or if it is anticipated that magnesium therapy will be instituted, there is no evidence to support this recommendation. Lastly, a uric acid level and renal function tests may be of some prognostic value.[12,59,60] Urinalysis should be performed and prothrombin time and partial thromboplastin time measured. If the latter times are prolonged and fibrinogen levels are decreased, disseminated intravascular coagulation should be considered, and the patient should be typed and cross-matched for transfusion with packed red cells and fresh frozen plasma.

In the patient with altered mental status or seizures, the possibilities of hypoglycemia, exposure to toxins, and substance abuse should be ruled out. A noncontrast CT scan of the head may detect hemorrhage, edema, and mass lesions; an MRI may be preferred in select cases, such as suspected transient ischemic attacks, posterior circulation lesions, cerebral venous sinus thrombosis, or PRES.[61] **(See Figure 1, page 4.)**

Posterior reversible encephalopathy syndrome is a clinical and radiologic syndrome that includes headache, confusion, visual changes, and seizures and is accompanied by diagnostic white matter abnormalities seen on MRI. It is a rare—but potentially lethal—complication of eclampsia. Although PRES can be seen in a number of other conditions such as hypertensive encephalopathy, renal disease, or in patients receiving immunosuppressive therapy, the pathologic findings on MRI are a result of the vasogenic edema and resulting increased intracerebral pressure that are seen in all of these conditions.[28] Some investigators suggest that PRES should be considered an indicator of eclampsia even when other features of the latter disorder (such as hyper-

tension and proteinuria) are not present.[62] Hence, an MRI may be of value in the diagnosis, treatment, and prognosis of the postpartum ED patient who presents with seizures or with headache and mildly elevated blood pressure; however, this approach has not been studied.

In patients with the HELLP syndrome, a chest x-ray is indicated if they have signs or symptoms of pulmonary congestion. Hepatic hematoma is a known complication of HELLP, and in patients with right upper quadrant pain, CT imaging is more dependable than ultrasound in detecting the characteristic lesions.[63]

Peripartum Cardiomyopathy

The diagnostic evaluation in patients with PPCM is similar to that for congestive heart failure. Necessary tests include a complete blood count; serum chemistries; cardiac, liver, and renal function studies; a chest x-ray; and an electrocardiogram (ECG). Cardiac enzymes may be elevated in the presence of severe heart failure due to PPCM, but significant elevations may also indicate other possible causes for the patient's symptoms, such as a preceding ischemic event or viral myocarditis.

The ECG in PPCM was first described by Demakis et al in a prospective cohort of 8 patients in 1971.[9] It usually shows sinus tachycardia or occasionally atrial fibrillation, along with nonspecific T-wave abnormalities and left ventricular hypertrophy. Q waves are sometimes present in the anterior precordium, and PR and QRS intervals may be prolonged.[9,64] An echocardiogram may be helpful to assess the extent of left ventricular dysfunction and contractility in patients who present with advanced heart failure without a history of preexisting cardiac disease. There is no evidence to justify obtaining an echocardiogram while the patient is in the ED, although most authors on the subject recommend it as part of the overall evaluation and to rule out other possible causes of heart failure, such as previously undiagnosed valvular abnormalities.

Treatment

Headache

Although there are no studies to evaluate its efficacy, the recommended therapy for postdural puncture headache is bed rest and analgesics while awaiting the restoration of CSF volume; however, the former is often impossible for the new mother and the latter may be ineffective. By the time the patient gets to the ED, the definitive treatment to be considered is an epidural blood patch, which has been reported to be successful in 96% to 98% of patients with moderate-to-severe prolonged spinal headache.[65] The procedure is a relatively simple one and involves the injection of 10 to 20 mL of autologous blood into the epidural space, at or near the site of the previous puncture. Many anesthesiologists consider postdural puncture headache to be a complication of anesthesia about which they should be notified. If the patient presents to the same hospital where her delivery took place, the anesthesiology group is usually more than willing to do a follow-up consultation and to provide treatment. Although the mechanism that accounts for the success of the blood patch is not completely understood, it is hypothesized that the patch tamponades the leak, preventing persistent leakage while permitting restoration of normal CSF volume.

Treatment for migraine and tension headaches is the same as it is for all other patients. Acetaminophen, ibuprofen, caffeine, β-blockers, calcium-channel blockers, prednisone, and most triptans are usually safe for the breastfeeding mother; opioids may cause drowsiness in the breastfed infant. Prochlorperazine is acceptable for short-term or one-time use.[66] Because information about butalbital, ergotamine, and metoclopramide is insufficient, these agents should probably be avoided. Aspirin should also be avoided because of the theoretical risk of causing Reye's syndrome in the infant.[66]

Postpartum Hypertension

Management can be problematic in the case of the postpartum patient who has an elevated blood pressure but does not appear to be preeclamptic (ie, no signs or symptoms of headache, visual disturbances, nausea, vomiting, epigastric pain, or proteinuria) and is otherwise asymptomatic. As with other adult patients with asymptomatic hypertension in the ED, clinicians should attempt to answer the same questions regarding the potential for harm; ie, the significance of an isolated elevated blood pressure in the ED at a single point in time, whether the blood pressure reflects hypertension or something else, whether there is any need for immediate treatment, and whether the potential for benefit with treatment is greater than the risk. (See the June 2010 issue of *Emergency Medicine Practice*, "Hypertension In The Emergency Department: Treat Now, Later, Or Not At All.") The ACEP clinical policy on asymptomatic hypertension in the ED offers management recommendations,[67] but there is no evidence that more aggressive treatment of hypertension in the postpartum patient may prevent LPPE. A 2007 Cochrane Review that included 24 trials involving 2949 women on antihypertensive drug therapy in pregnancy concluded that treatment did not reduce their risk for preeclampsia.[68] However, the significant number of patients with LPPE who had no symptoms or diagnosis of preeclampsia prior to their seizure[16,18] suggests that the more liberal prescribing of antihypertensive medication in this group may be indicated. Nevertheless, according to a Cochrane Review on the prevention and treatment of post-

partum hypertension that included 6 trials and 315 women, "there are no reliable data to guide management of women who are hypertensive post partum or who are at increased risk of becoming so." The reviewers recommend that if a clinician believes that the hypertension warrants treatment, the drug(s) used should be based on his or her familiarity with it.[69] The oral antihypertensive drugs commonly used post partum are listed in **Table 7**.

Preeclampsia And Eclampsia

The definitive treatment for the patient with antepartum preeclampsia or eclampsia is delivery, which is obviously not a consideration post partum. The published literature offers no guidance on the management of the postpartum patient suspected to have preeclampsia, so treatment is based on practice patterns used to treat pregnant patients with preeclampsia for whom delivery is not an option because of fetal prematurity. Therapy in these patients is directed toward control of blood pressure and prevention of progression to eclampsia.

Brain damage due to intracranial hemorrhage or ischemia may result in permanent neurologic damage and is the most common cause of death in women with eclampsia.[70,71] In a case series of 28 pregnant women with eclampsia and stroke, 15 of whom died, the mean systolic blood pressure was 175 mm Hg and the mean diastolic blood pressure was 98 mm Hg. Diastolic blood pressure was above 110 mm Hg in only 3 women and was above 105 mm Hg in only 5, suggesting that their strokes were not directly related to the absolute elevations in blood pressure.[72] Although it is not clear whether elevated blood pressure is a cause or an effect of the pathophysiology of stroke in eclampsia, practice patterns and prudence suggest that—just as for the antepartum patient—the postpartum patient will benefit from careful, ongoing monitoring of blood pressure and lowering of blood pressures to 130 to 150 mm Hg systolic and 80 to 100 mm Hg diastolic.

Intravenous labetalol in an initial dose of 20 mg followed at 10-minute intervals by doses of 20 to 80 mg, to a total cumulative dose of 300 mg, is usually effective. Instead of intermittent therapy, an IV infusion of 1 to 2 mg/min may be used after the first dose. Hydralazine may also be used in a dose of 5 mg by slow IV push over 1 to 2 minutes; a repeat bolus of 5 to 10 mg can be given every 20 minutes to a total dose of 30 mg. There are insufficient data to allow reliable conclusions about the comparative effects of labetalol and hydralazine.[68]

One goal of therapy in the patient with postpartum preeclampsia is to prevent progression to eclampsia. Magnesium sulfate has been shown to be effective in this regard, reducing the risk of eclampsia by 50% compared with placebo.[73,74] Seizures in eclampsia are usually of short duration and self-limited. In well-designed prospective studies, treatment with magnesium has also been shown to be more effective and safer in preventing further seizures in pregnant patients than either diazepam or phenytoin.[75,76] Magnesium sulfate is given at a loading dose of 4 to 6 g IV over 15 minutes followed by 2 to 3 g IV per hour. Patients should be observed to detect any loss of reflexes and respiratory depression, both of which are signs of hypermagnesemia. If seizures recur at therapeutic doses of magnesium, other anticonvulsant drugs can be administered. At that point, consideration should also be given to other possible causes of seizures, such as intracranial hemorrhage or metabolic abnormalities. **Table 8 (see page 12)** provides a summary of pharmacologic therapies for postpartum preeclampsia or to prevent or treat eclampsia.

As for more invasive approaches to severe LPPE, in a case report of refractory seizures, 2 patients had rapid recovery after uterine curettage.[77] In another case report, plasma exchange reportedly resulted in a full recovery.[78] Cerebral angioplasty improved outcome in 1 patient with neurologic deficits in one reported case.[79]

The HELLP Syndrome

When diagnosis of the HELLP syndrome is confirmed by pathognomonic laboratory abnormalities, efforts should be directed, as in eclampsia, toward controlling blood pressure and preventing seizures. Platelet transfusion may be indicated when counts are less than 20,000 cells/µL or if there is evidence of bleeding. Although dexamethasone was previously thought to enhance recovery, this drug has not been shown to be effective in large randomized trials.[80,81] Evidence of abdominal distention or increasing abdominal girth is suggestive of a ruptured hepatic hematoma. Treatment should be aimed at maintaining adequate intravascular volume. If the patient is

Table 7. Oral Antihypertensive Therapy For Postpartum Hypertension In Breastfeeding Mothers[83,84]

Drug	Comments
Labetalol or propranolol	Preferred for initial therapy; not concentrated in breast milk
Sustained-release nifedipine or verapamil	May be used as a second choice if β-blockers are contraindicated
Angiotensin-converting enzyme (ACE) inhibitors and angiotensin-receptor blockers	Avoid during neonatal period; may be used thereafter
Thiazide diuretlcs	May reduce volume of breast milk; doses should be less than 50 mg per day

hemodynamically stable, percutaneous embolization of the hepatic artery can be done[82]; if not, operative management should be considered.

Peripartum Cardiomyopathy

Treatment for PPCM is similar to that for other types of acute heart failure, including oxygen, diuretics, vasodilators, and arrhythmia management.

Disposition

The disposition of the postpartum patient in the ED depends on the suspected diagnosis. Patients with tension or migraine headache can be prescribed analgesics and followed as outpatients by their primary care providers, with obstetric follow-up as scheduled. Patients with postdural puncture headache will usually get complete relief if given a blood patch and should require no further follow-up or treatment. In all cases, careful instructions should be given about returning to the ED if symptoms persist or recur. Patients with simple postpartum hypertension may be followed by their primary care provider, with similar instructions to return to the ED if symptoms such as headache, nausea, vomiting, or visual disturbances develop.

When the headache is due to infection, cerebrovascular accident, or a space-occupying lesion, the patient should be referred for a neurologic and/or neurosurgical consultation and admitted to the hospital, usually to the intensive care unit.

The decision to admit patients with mild symptoms and mildly elevated blood pressure is usually based on the judgment of the clinician. Since obstetricians have more experience in this regard, their input may be valuable. For those patients who are discharged, careful instructions to return immediately to the ED for new or worsening symptoms, and close, early follow-up are necessary. Because postpartum patients can progress rapidly from having absent or mild symptoms to the onset of seizures and the HELLP syndrome, an extended period of observation may be warranted if the decision on whether or not to admit a patient to the hospital is not clear-cut. Patients with more severe symptoms of preeclampsia, eclampsia, or the HELLP syndrome will require an obstetric consultation and admission to the hospital, usually to an intensive care unit, for careful monitoring and observation for seizures, neurologic changes, and end-organ failure.

Patients with PPCM whose symptomatology is mild and who have no arrhythmias may be managed with outpatient therapy and close follow-up by a cardiologist. Those who are more symptomatic will require admission to the hospital for diuresis, optimization of ventricular function, and a diagnostic work-up.

Table 8. Pharmacologic Therapy Of Postpartum Preeclampsia And Prevention/ Treatment Of Eclampsia

For blood pressure management:

- Hydralazine 5 mg IV initially, 5-10 mg IV every 20 minutes
- Labetalol 10-20 mg IV initial dose; then as needed, additional doses every 10 minutes that are double the prior dose up to 80 mg as a single dose and a maximum total dose of 300 mg (eg, 20, 40, 80, 80 or 10, 20, 40, 80, 80)

For seizure prophylaxis/treatment:

- Magnesium sulfate 4-6 g IV over 15 minutes or 2-3 g IV per hour*

For persistent seizures**:

- Diazepam 0.1-0.3 mg/kg IV push, to maximum cumulative dose of 20 mg
- Lorazepam 0.02-0.03 mg/kg IV push, to maximum cumulative dose of 0.1 mg/kg
- Phenytoin 20 mg/kg IV with 50 mg/min infusion, or fosphenytoin 20 PE/kg at 150 PE/min***

* Monitor for loss of reflexes or respiratory depression.

** Consider other cause of seizure.

*** Monitor for hypotension and/or cardiac arrhythmias.

Abbreviations: IV, intravenously; PE, phenytoin sodium equivalent units.

Summary

Common postpartum emergencies include hypertension, headache, preeclampsia and/or eclampsia, and infections. An increasing percentage of all cases of eclampsia now occur post partum. The HELLP syndrome and peripartum cardiomyopathy are rarer postpartum complications. Headache is a common symptom that either can be premonitory of late postpartum eclampsia or can be a reflection of another benign disorder or of a serious disease process such as subarachnoid hemorrhage and cerebral venous sinus or arterial thrombosis. Patients with postpartum preeclampsia may present with mild symptoms and mildly elevated blood pressure, or they may rapidly progress to eclampsia or even present with seizures with no premonitory symptoms. Consultation with specialists in obstetrics, anesthesia, neurology, or cardiology is often indicated, and admission may be necessary. A careful history and physical examination, knowledge of the differential diagnoses, early treatment, and careful follow-up will optimize outcomes.

Case Conclusions

On repeat measurement of her blood pressure, your patient with the headache had a reading of 155/90 mm Hg. You prescribed prochlorperazine for her headache and nausea and IV saline, and you ordered a CBC, metabolic

profile, uric acid level, coagulation panel, and urinalysis. The bedside urine dipstick result for protein was 1+. You consulted obstetrics and anesthesia. The obstetrician evaluated the patient and called your attention to her hyperreflexia. The patient also seemed lethargic, which you concluded may be due to the prochlorperazine but may also be due to preeclampsia. You and the obstetrician decided to begin magnesium sulfate with a bolus of 4 g over 15 minutes followed by an infusion of 2 g per hour. The anesthesiologist arrived and concluded that this was not a postdural puncture headache, and your patient was moved to the ICU. You found out the following week that she had done well, with no progression to eclampsia and rapid resolution of her symptoms, and that she was discharged home 2 days later.

As for your patient with dyspnea, her ECG showed atrial fibrillation with a rapid ventricular response and no evidence of an acute myocardial infarction. She rapidly responded to nitrates, diuretics, and BiPAP, with a reduced respiratory rate and improved oxygenation. Her pretreatment chest x-ray showed significant biventricular enlargement and interstitial edema. When the cardiologist came in to consult, he informed you that he suspected postpartum cardiomyopathy, so the patient was transferred to the CCU.

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Evidence-based medicine requires a critical appraisal of the literature based upon study methodology and a number of subjects. Not all references are equally robust. The findings of a large, prospective, randomized, and blinded trial should carry more weight than a case report.

To help the reader judge the strength of each reference, pertinent information about the study, such as the type of study and the number of patients in the study, will be included in bold type following the reference, where available. In addition, the most informative references cited in this paper, as determined by the authors, are noted by an asterisk (*) next to the number of the reference.

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Risk Management Pearls For Postpartum Emergencies

1. Late postpartum eclampsia can rapidly develop with only one or no premonitory signs and symptoms of elevated blood pressure, proteinuria, or headache.
2. Preeclampsia should be considered in postpartum patients with mildly elevated blood pressure and/or proteinuria.
3. Symptoms of preeclampsia post partum include headache, nausea, vomiting, visual disturbances, abdominal pain, and malaise.
4. Patients with postpartum preeclampsia can progress very rapidly from relatively mild symptoms and an elevated blood pressure to seizures.
5. Even mild abnormalities on CBC or liver or renal function tests post partum may be indicative of the HELLP syndrome.
6. Although a urine dipstick result of 1+ for protein roughly correlates with 300 mg of protein in a 24-hour urine sample (a diagnostic criterion for preeclampsia), a negative dipstick result is not sufficiently sensitive to rule out preeclampsia.
7. Headache post partum can be from a number of serious neurovascular abnormalities, including preeclampsia, subarachnoid hemorrhage, stroke, or cerebral venous sinus thrombosis.
8. Peripartum cardiomyopathy can often be mistaken for mild upper respiratory congestion but can quickly deteriorate into fulminant congestive heart failure.
9. Dyspnea in the postpartum patient may be indicative of postpartum cardiomyopathy, pulmonary embolism, or preeclampsia.
10. Magnesium sulfate is the drug of choice for the prevention and treatment of postpartum eclamptic seizures.

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1. Patients with LPPE usually have elevated blood pressures and proteinuria prior to the manifestation of seizures.
   a. True
   b. False

2. All of the following are risk factors for PPCM EXCEPT:
   a. African descent
   b. Age less than 20
   c. Maternal cocaine abuse
   d. Preeclampsia

3. **Which of the following statements about the HELLP syndrome is true?**
   a. 30% of cases are seen post partum.
   b. Dexamethasone has been shown to reduce the degree of hemolysis.
   c. In the setting of hypotension and abdominal pain, the most likely etiology is disseminated intravascular coagulation.
   d. Platelet transfusion is rarely necessary.

4. **Which of the following statements about PPCM is true?**
   a. It does not respond to the usual treatments for congestive heart failure.
   b. It is primarily a manifestation of diastolic dysfunction.
   c. It occurs during the last month of pregnancy or during the 5 months post partum.
   d. It primarily occurs in women with known underlying heart disease prior to pregnancy.

5. **Which of the following statements regarding neuroimaging is true?**
   a. A CT scan of the head should be done to rule out other causes of intracranial disease.
   b. Imaging is not necessary, since results on brain CT are negative.
   c. MRI has prognostic value in determining which patients will have permanent neurologic deficits.
   d. MRI is necessary to make the diagnosis of LPPE.

6. **The optimal treatment of seizures in patients with LPPE is:**
   a. Diazepam bolus repeated as necessary
   b. Lorazepam bolus followed by magnesium loading dose and maintenance drip
   c. Magnesium sulfate loading dose followed by a maintenance drip
   d. Magnesium sulfate loading dose followed by phenytoin maintenance

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Date of Original Release: August 1, 2010. Date of most recent review: July 10, 2010. Termination date: August 1, 2013.

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Target Audience: This enduring material is designed for emergency medicine physicians, physician assistants, nurse practitioners, and residents.

Goals & Objectives: Upon completion of this article, you should be able to: (1) demonstrate medical decision-making based on the strongest clinical evidence; (2) cost-effectively diagnose and treat the most critical ED presentations; and (3) describe the most common medicolegal pitfalls for each topic covered.

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Postpartum Emergencies: Headache, Hypertension, Eclampsia, And Cardiomyopathy

Sixsmith D. August 2010, Volume 12; Number 8

This issue of Emergency Medicine Practice *focuses on the most commonly encountered postpartum emergencies: headache, LPPE, hypertension, and cardiomyopathy. For a more detailed discussion of this topic, including figures and tables, clinical pathways, and other considerations not noted here, please see the complete issue on the EB Medicine website at www.ebmedicine.net/topics.*

Key Points	Comments
Peripartum cardiomyopathy (PPCM) is a rare form of heart failure that occurs in the last month of pregnancy or within 5 months of delivery.	PPCM should be considered in the differential diagnosis of the postpartum patient with new onset of dyspnea.[8,9]
Late postpartum eclampsia (LPPE) now comprises 13% to 16% of all cases of eclampsia.	LPPE should be suspected in symptomatic women with elevated blood pressure and/or proteinuria.[14-16]
Patients with preeclampsia or LPPE may have none or only one of the symptoms of headache, nausea, visual disturbances, and abdominal pain.	There should be a high index of suspicion of preeclampsia for any postpartum patient with symptoms, elevated blood pressure, or proteinuria.[14,18]
The HELLP syndrome includes **H**emolysis with a microangiopathic blood smear, **E**levated **L**iver enzymes (lactate dehydrogenase [LDH] and aspartate aminotransferase [AST]), and a **L**ow **P**latelet count, and occurs late post partum.	Complete blood count, renal, and liver function tests are part of the laboratory evaluation of the postpartum patient.[32]
Headache is a common postpartum complaint with a broad differential that includes several serious conditions.	Preeclampsia and vascular emergencies, though rare, should be in the differential diagnosis.[49]
Blood patch relieves the symptoms of postdural puncture or spinal headache.	Blood patch is a successful treatment in 96% to 98% of patients with postdural puncture or spinal headache.[62]
Treatment of uncomplicated postpartum hypertension should be based on the physician's judgment and familiarity with the antihypertensive medication.	While stroke is a significant cause of perinatal mortality, there are no reliable data on the efficacy of drug therapy in reducing risk.[65]
Magnesium is the recommended treatment for the postpartum preeclampsia and LPPE.	Although no studies have specifically been done using magnesium in LPPE, it is commonly used for treatment post partum.[67,68]

See reverse side for reference citations.

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These references are excerpted from the original manuscript. For additional references and information on this topic, see the full text article at ebmedicine.net.

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