



Eclampsia Management: Our Experience

Dr Girija Wagh, MD

FICOG Dip in Endoscopy

Professor and Head, OBGYN, Bharati Vidyapeeth University Medical College, Pune, India

Introduction

As a country the biggest challenge faced today by us obstetricians is the maternal mortality and morbidity. Also what we encounter as the most challenging disorder is preeclampsia and eclampsia. As said when we do not know the makers of PIH what can we know about the markers? Truly we donot know why PIH occurs and lifetimes have gone by in bursting this mystery.

Preeclampsia and eclampsia are obstetric diseases, and obstetricians are the group best equipped to diagnose, evaluate and manage them. Today as a clinician however we need to tackle what we have from the experiences gathered and try to deliver the best to our patients. We should not falter there and should try to deliver the best. From making the diagnosis to treating atypical eclampsia, management of preeclampsia involves serious, often unpredictable challenges. In this article, we highlight several challenges that obstetricians face when managing preeclampsia and eclampsia, and offer useful strategies to help minimize morbidity and mortality in both mother and infant.

Although severe preeclampsia represents only a fraction of those amounts, and eclampsia an even lower percentage, they are potentially catastrophic complications of pregnancy and one of the leading causes of maternal death. They also are responsible for a large percentage of infants born prematurely as a result of a worsening maternal or fetal condition.

The National Eclampsia Registry interim statistics reveals that the incidence of hypertensive diseases during pregnancy to be quite high with quite a substantial incidence of eclampsia. These are actually cases reported by the FOGSI members. There can be quite some more which are being treated by peripheral health workers and the incidence can actually be higher. What we know for sure is 1 in 10 pregnancies

are complicated by PIH and therefore we need to have a high index of suspicion.

- By definition Eclampsia is defined as the occurrence of one or more convulsions superimposed on pre-eclampsia.
- Preeclampsia is pregnancy-induced hypertension in association with proteinuria (> 0.3 g in 24 hours) \pm edema and virtually any organ system may be affected

We also know that there are four major types of hypertensive disorders during pregnancy. And we need to classify them. It is important that we do so as that helps in better prognostication and treatment planning.

1. Chronic hypertension
2. Preeclampsia eclampsia syndrome
3. Superimposed preeclampsia
4. Gestational hypertension

Attempts should be made to establish these diagnoses antenatally, intranatally, postnatally and in subsequent pregnancy

Optimum Antenatal Care is a Must

Early and adequate prenatal care cannot me more emphasized ! Although the diagnostic criteria for preeclampsia have been widely established – persistent BP elevation above 140/90mmHg and proteinuria exceeding 300mg over a 24hr collection period- the condition does not always play by the rules. With close monitoring of weight, urine protein, and BP, the clinician can identify and follow the patient and detect a condition much early

Risk Factors for Preeclampsia:

- Chronic hypertension
- Chronic renal disease
- Connective tissue disease

- Current foetal growth restriction
- Gestational hypertension in the current pregnancy
- History of prior preeclampsia
- Insulin dependent diabetes
- Multiple gestation
- Nulliparity
- Obesity
- Thrombophilia.

It is important to diagnose it early:

Early identification of preeclampsia may allow for interventions, including delivery, that will lessen the risk of progression to severe preeclampsia and eclampsia and reduce foetal and maternal morbidity and mortality. It is, therefore, essential for the clinician to ask specifically about signs and symptoms of preeclampsia and to listen carefully to the answers.

Signs and symptoms may sometimes be typical:

- Weight gain
- Increasing edema
- Persistent headache
- Blurred vision
- Malaise
- Nausea
- Epigastric discomfort
- Right upper quadrant discomfort.

Although a number of tests have been proposed to predict who may be at greatest risk for preeclampsia, none have risen to the level that they can be recommended for general population screening.

Diagnostic Criteria

The diagnosis of preeclampsia is based on persistent BP elevation above 140/90 mmHg and proteinuria exceeding 300 mg over a 24-hour collection period. Other criteria have been applied, such as rise in systolic or diastolic BP above baseline and urine dipstick criteria for proteinuria, but BP above 140/90mmHg and proteinuria above 300mg are most frequently used in medical centres. Gestational hypertension and chronic hypertension do sometimes coexist with superimposed preeclampsia, but should not be confused with preeclampsia or lead to management decisions that should apply only to patients with preeclampsia.

Before severe preeclampsia can be diagnosed, the initial criteria for preeclampsia should have been

fulfilled, along with one or more of the findings listed below:

- Persistent blood pressure above 160/110 mmHg
- Proteinuria
- Refractory oliguria (<500cc over 24 hours)
- Renal failure (minimal criterion would be a rise in serum creatinine of 1mg/dl above baseline)
- Persistent right upper quadrant or epigastric pain or both
- Persistent headache
- Scotomata/blurred vision
- Shortness of breath with reduced oxygen saturation or pulmonary edema
- Thrombocytopenia (platelets <100,000 / cu.mm)
- Hemolysis (based on peripheral smear analysis or increased Bilirubin)
- Impaired liver function of unclear etiology
- Eclampsia
- Estimated foetal weight below 5th percentile for gestational age

Prediction

Attempts to predict preeclampsia have met with poor results. Measurement of the ratio of uterine artery systolic to diastolic flow has not been informative in the general healthy population of pregnant women. Nor has uric acid determination been useful; it generally has very poor predictive value and should be interpreted with caution.

When to Hospitalize?

Mild preeclampsia can be managed expectantly until foetal maturity or 37 weeks of gestation. But hospitalization can be offered in the Indian context. This offers an opportunity to investigate the patient properly, monitor the urine output, BP and the fetal condition through USG and Doppler if necessary. Also the patient can be offered dietary advice and the correct categorization after her BP has been monitored round the clock. But any serious presentations such as severe edema, ascites, high BP, severe proteinuria, headache, pain, severe IUGR, convulsions etc demand a hospital care.

Assessment:

Initial evaluation consists of:

- Foetal non stress testing
- Amniotic fluid index

- Serial BP determination
- 24-hour urine collection(if dipstick proteinuria is negative)
- Initial laboratory evaluation comprising of a complete blood count with platelets and aspartate amino transferase (AST), alanine amino transferase (ALT), and creatinine levels and LDH levels

The tests should be directed to assess the maternal conditions as Preeclampsia is a multisystemic disorder. Constant vigilance should be undertaken to prevent eclampsia as far as is possible and to diagnose HELLP early. There is a tendency to prolong the pregnancy as much as possible to be able to achieve salvagibility in the fetus. But one needs to weigh the risk to the mothers system such a prolongation could cause. Also LDH levels above 600 have proved to be a better parameter to guide a clinician regarding the presence of hemolysis. This can help one guide regarding the intervention and rising LDH levels would help this decision. While interpreting renal parameters in Pregnancy one should muster care as these parameters are already reduced in a normal pregnancy due to increased GFR and hemodilution

Additional tests may be ordered as indicated but are of limited value in making management decisions. If foetal and maternal evaluations are reassuring, and if the patient has remained stable, then outpatient management may be considered. In general, if proteinuria exceeds one gram in 24 hours, in-hospital management is recommended, regardless of other parameters.

Controlling Blood Pressure: Why? and How?

Cerebrovascular accident(stroke) is the leading cause of maternal mortality from preeclampsia. Not all cases can be prevented, but one suggested preventive strategies is adequate BP control. Some cases of stroke in the setting of preeclampsia will occur despite systemic BP reading that are not considered to be in a dangerous range. One reason may be an over ride of normal cerebral blood flow auto regulation mechanisms, resulting in increased cerebral blood flow, rising cerebral perfusion pressures, and vessel rupture. Such occurrences may sometimes, but not always, be related to coagulopathy.

When a patient has elevated BP, generally defined as persistent systolic pressures above 160 to 170mmHg and persistent diastolic pressures above 105 to 110 mmHg, antihypertensive therapy is indi-

cated and should be administered in a timely fashion.

Labetalol, nifedipine have been used effectively in such acute settings, when administered parenterally (except nifedipine, which can be given orally and should never be given sublingually) and when given in proper dosage.

Pharmacotherapy of acute hypertension:

Table 1. Pharmacotherapy of acute hypertension.

Drug	Dosage	Directions
Labetalol	10-20 mgIV push	Repeat every 10-20mins, doubling the dosage each time until a maximum total cumulative dosage of 300mg has been given.
Nifedipine	10 mg	Repeat in 20mins for four doses (maximum 40mg); then give 10-20 mg orally (never sublingually) every 4-6h to achieve a stable BP of 140-150/90-100 mmHg.

Preventing Seizures

Magnesium sulfate is the drug of choice to prevent both initial and recurrent eclamptic seizures. Two large clinical trials ended any doubts about its efficacy, demonstrating its superiority over both phenytoin and diazepam in the settings of preeclampsia and eclampsia.

Magnesium sulfate is best administered intravenously(iv) via continuous infusion pump. An initial bolus of 4-6gm is given over 15-30mins; this amount does not need be adjusted to the patient's level of renal function. A continuous infusion of magnesium sulfate is usually initiated at a rate of 2gm/hour. It is this infusion dosage that may need to be altered, based on the patients urine output and renal function.

Evidence of magnesium toxicity includes:

- Loss of deep tendon reflexes
- Respiratory depression
- Blurred vision
- Cardiotoxicity

Each of these toxicities can occur at ostensible therapeutic levels of serum magnesium, so there can be no substitute for the regular (atleast every 2hours) clinical assessment of the patient who is receiving a continuous infusion of magnesium sulfate.

There is no debate about the utility of magnesium sulfate in severe preeclampsia, but when it comes to intrapartum management of mild preeclampsia or cases in which preeclampsia first manifests in the post partum period, data are not so clear. This debate will not be resolved to anyone's satisfaction in the course of this article. Historically, the practice has been to use magnesium in these circumstances, but the pendulum has begun to shift based on the few arguments:

- Eclampsia is a rare event (about 1 case for every 300 to 1000 deliveries)
- Most cases occur outside of the hospital
- Some women experience seizures before preeclampsia has been diagnosed
- Some patients experience seizures while taking magnesium sulfate.

One might argue that number of potentially preventable cases of eclampsia is lower-perhaps in the range of one in every 3000 to 10,000 deliveries-and that this low rate does not justify routine use of the drug.

Regardless of one's position on this debate, there is broad consensus that regular careful clinical assessment of the patient who has preeclampsia is essential to minimize the morbidity and mortality. This disease can progress from mild to severe rapidly. Only thorough regular careful assessment can a physician observe this change soon enough to alter management as necessary.

Treatment of Magnesium Toxicity:

10% calcium gluconate (1g) is administered IV to reverse the effects of suspected magnesium toxicity.

In addition, because magnesium freely crosses the placenta, it is recommended that a new born resuscitation team be present at all deliveries during the mother was receiving magnesium sulfate because neonatal respiratory and cardiac depression have been reported in this setting.

Delivering The Patient:

Preeclampsia, severe preeclampsia, and eclampsia present a dilemma for the managing clinician: subject her to the rigors of labor, or to the heightened risk of cesarean delivery? Overall, a properly vaginal delivery is less hemodynamically stressful than cesarean delivery for the mother. To accomplish vaginal delivery, it is necessary to provide optimal anesthesia and analgesia.

Risks of Regional Anesthesia:

Women who have preeclampsia are volume depleted. As such, they are prone to hypertension after administration of regional anesthesia if the block sets up too rapidly. For this reason, epidural anesthesia or some of the newer combined techniques offer optimal analgesia by allowing for slower implementation of the regional block.

Women who have preeclampsia, especially severe preeclampsia, are usually candidates for regional analgesia and anesthesia. Some requisites for regional anesthesia under these conditions include the following:

- The patient can tolerate preblock hydration
- She has adequate IV access
- There is reproducible means of determining BP
- The patient has a normal coagulation profile. (a normal platelet count with normal transaminase should be sufficient to confirm this; women who have preeclampsia are not at increased risk of having altered prothrombin time, partial thromboplastin time, or fibrinogen levels, provided there are no other mitigating clinician circumstances).
- The anesthesiology team is skilled in the administration of regional anesthesia.

If Eclampsia Occurs:

Do not proceed to emergent cesarean section. Rather, stabilize the mother, protect her from injury during the seizure, protect her airway, and allow the seizure to take its course.

Begin magnesium at once. If it was being infused before the seizure, consider giving an additional 2g bolus over several minutes. As the mother stabilizes, the foetal heart rate will recover and she can be reassessed to determine optimal timing and route of delivery.

In case of fetal compromise cesarean section may be a better choice than a vaginal delivery

Practice AMTSL

Whether a vaginal delivery or a CS active management with oxytocin should be practiced to prevent PPH. It is safe to use Oxytocin 5 U bolus equally diluted over 2-3 minutes or Prostaglandin injections. PG can be used also as misoprostol sublingually or transvaginally. due to hemoconcentration even average loss may not be well tolerated by these patients. Also hypotension, use of magnesium sulphate and

endothelial dysfunction can contribute to more blood loss which may not be well compensated and tolerated by these patients. All attempts to reduce blood loss should be undertaken. The fluids used should be liberal but judicious recommendation is 80 ml/kg/hr as over infusion can cause pulmonary edema very fast in these women.

Post Delivery Management

It involves close vigilance for eclampsia, PPH, HELLP, Pulmonary edema and thromboembolic complications. Delivery of the baby is the treatment but the 72 hours post delivery are an important period when hemodynamic transition is occurring in the mother which need close observation and early detection of eclampsia. The NER data has shown a high index of postpartum eclampsia and it has to be remembered that such an occurrence leading to morbidity has to be avoided by all means

Preventing Complications

Preeclampsia/eclampsia produces multiple systemic derangements that can involve a diversity of organ systems including hematologic, hepatic, renal, and cardiovascular systems as well as the central nervous system. The severity of these derangements often correlates with maternal medical (eg, pre-existing renal or vascular pathology) or obstetric factors (eg, multifetal gestations or molar pregnancy). Systemic derangements associated with eclampsia can include the following:

- Cardiovascular
 - Generalized vasospasm
 - Increased peripheral vascular resistance
 - Increased left ventricular stroke work index
 - Decreased central venous pressure
 - Decreased pulmonary wedge pressure
- Hematologic
 - Decreased plasma volume
 - Increased blood viscosity
 - Hemoconcentration
 - Coagulopathy
- Renal
 - Decreased glomerular filtration rate
 - Decreased renal plasma flow
 - Decreased uric acid clearance
- Hepatic
 - Periportal necrosis
 - Hepatocellular damage
 - Sub capsular hematoma

- Central nervous system
 - Cerebral over perfusion due to loss of autoregulation
 - Cerebral edema
 - Cerebral hemorrhage

Eclampsia and preeclampsia account for approximately 63,000 maternal deaths worldwide annually.¹ In developed countries, the maternal death rate has been reported as 0-1.8%. A CDC study found an overall preeclampsia-eclampsia case-fatality rate of 6.4 per 10,000 cases at delivery with a rate twice as high for black women compared with white women. This same study found an increased risk of death among women older than 30 years and those with no prenatal care. The highest risk for maternal death was found in pregnancies at 28 weeks' gestation or less. The maternal mortality rate is as high as 14% in developing countries.² The perinatal mortality rate from eclampsia in recent reviews in the United States and Great Britain ranges from 5.6-11.8%.^{3, 4}

Maternal complications of eclampsia may include permanent CNS damage from recurrent seizures or intracranial bleeds, disseminated intravascular coagulopathy, renal insufficiency, pulmonary edema, and cardiopulmonary arrest. A majority of maternal deaths associated with eclampsia have concurrent HELLP syndrome.⁴ Causes of neonatal death include prematurity, placental infarcts, intrauterine growth retardation, abruptio placentae, and fetal hypoxia. Complications As many as 56% of patients with eclampsia may have transient deficits, including cortical blindness. Studies have failed to demonstrate evidence of persisting neurologic deficits after uncomplicated eclamptic seizures during the follow-up period.³ Maternal, as well as fetal, death can be a consequence of eclampsia and its complications.

Etio-pathogenesis of Complications: The failure of cytotrophoblastic epithelial-to-endothelial transformation and a subsequent lack of adhesion molecules, integrins, and cadherins results in a damaged placenta which in turn secretes the antiangiogenic factors, sFlt1 and endoglin into the maternal circulation. These factors lead to impaired VEGF/PlGF and TGF- β signaling, resulting in systemic endothelial cell dysfunction mediated by a variety of factors. Endothelial dysfunction, in turn, results in the systemic manifestations of preeclampsia. Endothelial dysfunction, vasoconstriction and platelet aggregation and edema result all over the body with various manifestations which can conglomerate into complications

Maternal Complications: Maternal complications are those related to the effect of severe preeclampsia on multiple organ systems, together with those associated with medical complications during pregnancy and the course of labor.

1. Eclampsia: The term comes from the Greek word for lightning. While the risk of death from complications of eclampsia is relatively high in the developing world ranging from about 14 to 22% – the risk is much lower in developed nations. Compared to women without seizures, eclamptic women had significantly higher rates of headache, visual changes, epigastric pain, and nausea and vomiting. HELLP syndrome, disseminated intravascular coagulation, acute renal failure, neurological complications, and acute respiratory distress syndrome. More than half of patients need cesarean delivery. Early neonatal mortality rate is also high.

2. HELLP Syndrome: One of the most severe forms of preeclampsia it occurs in 4 percent to 12 percent of the women who have preeclampsia. HELLP stands for: hemolysis, elevated liver enzymes, and lowered platelets. The criteria of the syndrome are debated but include hemolysis as evidenced by an abnormal peripheral blood smear; platelet count of less than $100 \times 10^9/L$; and serum AST value of greater than 70 U/L, serum lactate dehydrogenase value of greater than 600 U/L, or total bilirubin value of greater than 1.2 mg/dL. Clinically the diagnosis of HELLP syndrome may be challenging because patients may present with vague symptoms including nausea, vomiting, headache, malaise, or viral-like symptoms and is often mistaken for the flu acidity or gallbladder problems.

3. Antepartum hemorrhage: Abruptio placenta plus preeclampsia is a serious condition with a high risk for maternal death. It is important to remember that many patients with abruptio placentae have underlying preeclampsia because signs of shock may be present even with a normal blood pressure. These patients are hemodynamically very unstable. Although initially they also require active resuscitation, they quickly become fluid overloaded, resulting in pulmonary edema. Renal complications, such as acute tubular necrosis, commonly occur.

4. Hepatic complications: Subcapsular hematoma and hepatic rupture are very unusual catastrophic complication of preeclampsia/eclampsia. The reported incidence of this condition varies from 1 in 40,000 to 1 in 2, 50,000 deliveries. Infarction with vascular disruption leads to intrahepatic hemorrhage and parenchymal destruction. Ultrasound scan is the

quickest means of diagnosis although computerized tomography is more sensitive. The management of contained hematoma is to support the patient, with surgery reserved for those who are hemodynamically unstable or documented expansion of hematoma. Surgical management includes stitching of the lesions, packing, argon laser ablation, use of gelatin sponge, hepatic artery ligation, embolization of hepatic artery, use of recombinant factor VII A and liver transplantation.

5. Renal complications: Typically in preeclampsia, renal blood flow and GFR fall with a decreased urate clearance and increased calcium reabsorption leading to hyperuricemia and hypocalciuria. Histopathologically, there is swelling of the glomerular endothelial cells, referred to as glomeruloendotheliosis. Pre-renal oliguria may develop to acute tubular necrosis, most often with a good prognosis. Acute cortical necrosis is rare and has poor prognosis. Renal failure is relatively unusual even with severe cases, unless there is significant bleeding or hemodynamic instability or marked DIC. The renal and extrarenal abnormalities typically resolve spontaneously within the first two weeks postpartum.

6. Hematological Complications: Thrombocytopenia, Disseminated intravascular coagulation and associated bleeding disorders including massive PPH.

7. Cardiovascular dysfunction: Untreated preeclamptic women almost always have low filling pressures and a hyperdynamic circulation. Hence cardiac failure can occur. Some cases of peripartum cardiomyopathy may be associated with preeclampsia.

8. Respiratory dysfunction

A. Pulmonary edema: The main cause of maternal mortality in severe preeclampsia is now pulmonary edema. Incidence is 3-6% in severe preeclampsia. Patients with preeclampsia are usually volume depleted, and pulmonary edema most commonly occurs in the early postpartum period and is often associated with aggressive fluid replacement. Other predisposing factors include: a. Reduced albumin concentration and myocardial dysfunction contribute to edema formation. b. Reduced Colloid Osmotic Pressure (COP) c. Increased capillary permeability probably due to endothelial damage

B. Adult respiratory distress syndrome: appears to have become more common, it is not known whether this is a consequence of modern methods of respiratory support rather than of the disease itself.

9. Neurological complications

Headache: The most common neurologic symptom in preeclampsia/eclampsia is headache. Headache occurs in about in 75% women with seizures (eclampsia) and always precedes the seizure. Headache can be bitemporal, frontal, occipital, or diffuse.

Seizures: Convulsions are the other most common feature of this syndrome. Convulsions are usually generalized tonic-clonic in nature.

Visual disturbances: The most common symptom is blurring of vision. The visual disturbances are ominous and may indicate impending seizure. Blindness in women with eclampsia is rare and can be due to involvement of the occipital cortex or retina. Retinal detachment may cause altered vision although it is usually one sided and seldom causes total visual loss. Most women with varying degree of amaurosis are found to have radiographic evidence of extensive occipital lobe hypodensities. Blindness persists from 24 hrs to 72 hrs, it subsequently resolves completely.

Coma: Coma is a dreaded complication in eclampsia. Coma may be a result of intracerebral hemorrhage that, at times, may dissect into the ventricular system or over the surface of the brain, creating a massive subarachnoid hemorrhage. Investigations include head CT, which involve exposure to ionizing radiation and MRI which is safer in pregnancy.

Cerebrovascular accident: The association between eclampsia and cerebral hemorrhage has been recognized since 1881, and this is reported to be the most common cause of death in patients with eclampsia

10. Gastrointestinal Complications: Ischemia associated with pre-eclampsia cannot only damage the liver but also the pancreas and gallbladder.

11. Musculoskeletal complications:

Dislocation of shoulder is a very rare complication of eclampsia. Other reported musculo-skeletal complication of eclampsia include simultaneous bilateral central dislocation of hip

12. Maternal death: Events associated with maternal deaths include cerebral hemorrhage (45%), cardiopulmonary arrest (40%), disseminated intravascular coagulopathy (39%), adult respiratory distress syndrome (28%), renal failure (28%), sepsis (23%), hepatic hemorrhage (20%), and hypoxic ischemic encephalopathy (16%).

Fetal Complications: Severe preeclampsia is associated with different degrees of fetal injury and

include: a. Intrauterine growth retardation b. Premature delivery: c. Fetal distress/fetal demise

Managing complications: Managing complications of eclampsia needs a multidisciplinary approach and an institutional care. Eclampsia is unfortunately known to afflict the population which is many a times far away from such settings and therefore are deprived from such care. It therefore is important to train the first contact medical personnel catering to deliveries to be able to identify the possibilities of these complications well ahead of their occurrence. Abnormal blood pressure reading should be the first important alarm bell which mandates the patient to be fully investigated. The first approach to a pregnant patient with PIH is to classify the hypertension and the second step is to assess the possibility of a complication. Deterioration of these patients is very swift and therefore proper evaluation is important. There always has to be a proper evaluation of the systemic parameters through the various laboratory evaluations. In country like India where anemia and infections which cause thrombocytopenia are so rampant that Platelet count itself cannot suffice. We have observed that lactose dehydrogenase can help as a good marker for identifying the deterioration in the hemolysis and help guide a clinician as a decision making tool in situations of dilemma.

Table 2. Eden's criteria for risk assessment: can help guide the clinician to assess risks inpatients of eclampsia.

-
- Long interval bet the onset of fit and commencement of treatment
 - Antepartum eclampsia with long delivery interval
 - Fits >10
 - Coma in between the fits
 - Temp> 102 ° F :
 - PR 120/min
 - BP > 200 mmHg : SBP
 - Oliguria < 400 ml/24 hrs
 - Proteinuria > 5g/24 hrs
 - Non response to treatment
 - Jaundice
-

Prognosis: About 25% of women with eclampsia have hypertension in subsequent pregnancies. 5% of patients with hypertension develop severe preeclampsia. About 2% of women with eclampsia develop eclampsia with future pregnancies. Multiparous women with eclampsia have a higher risk for development of essential hypertension and a higher mortality rate in subsequent pregnancies as compared with primiparous women

Patient Education: The patient should be advised and educated on the course of the disease and any residual problems. The patient should be educated on the importance of adequate prenatal care in subsequent pregnancies. If the patient has preexisting hypertension, she should have good control prior to conception and throughout pregnancy. Her case should be followed for recognition and treatment of preeclampsia.

Miscellaneous Medicolegal Pitfalls: The mode of delivery should be based on obstetric indications, with the understanding that vaginal delivery is preferable from a maternal standpoint. When emergent cesarean delivery is indicated, substantiating the absence of coagulopathy prior to the procedure is important. Fetal bradycardia is common following an eclamptic seizure and usually resolves within 10 minutes. Consider placental abruption if uterine hyperactivity remains and fetal bradycardia persists. Cervical examination should not be overlooked. The delivery mode may be largely dependent on the cervical status. Fluid management is critical in patients with eclampsia. Avoid the use of multiple agents, unless necessary, to abate eclamptic seizures. Ruling out eclampsia in an obstetrical patient who has been involved in an unexplained trauma is important.

Special Concerns Do not overlook other neurologic causes, particularly if the seizure occurs more than 24 hours after delivery. When preeclampsia occurs in the early second trimester (ie, 14-20 weeks'

gestation), the diagnosis of hydatiform mole or choriocarcinoma should be considered. Eclampsia always should be considered in a pregnant patient with a seizure episode. A pregnant patient who has been involved in an unexplained trauma (such as a single-vehicle auto accident) and has exhibited seizure activity should be evaluated for eclampsia.

Post Partum Care

Every patient of preeclampsia should be monitored closely for 6 weeks with proper advice regarding the use of antihypertensives and should report at regular intervals. They should be guided and encouraged to use contraception at least for a period of 2-3 years. The preferred method would be an IUCD. They should be counseled regarding the importance of prenatal checkup counseling and the necessary care periconceptionally in the subsequent pregnancy.

References

1. Vigil-De Gracia P. Maternal deaths due to eclampsia and HELLP syndrome. *Int J Gynaecol Obstet.* Feb 2009;104(2):90-4. [Medline]
2. Sibai BM. Diagnosis, prevention, and management of eclampsia. *Obstet Gynecol.* Feb 2005;105(2):402-10. [Medline].
3. Sibai BM, Sarinoglu C, Mercer BM. Eclampsia. VII. Pregnancy outcome after eclampsia and long-term prognosis. *Am J Obstet Gynecol.* Jun 1992;166(6 Pt 1):1757-61; discussion 1761-3. [Medline].
4. Douglas KA, Redman CW. Eclampsia in the United Kingdom. *BMJ.* Nov 26 1994;309(6966):1395-400. [Medline]