

Postpartum Eclampsia Without Antenatal Proteinuria and Hypertension- A Case Report

Banu BİNGÖL, Aydan BİRİ, Yeter DİNGİL, Tuncay NAS, Özdemir HİMMETOĞLU

Ankara-Turkey

Postpartum eclampsia without the classical pre-eclamptic signs edema, proteinuria and hypertension is a rarely noticed complication of pregnancy. We report of a woman with postpartum eclampsia on 10th hour who had no signs of hypertension and proteinuria during antenatal period. The patient was followed from the beginning of her pregnancy in our clinic. Her arterial pressure never exceeded 120/70 and no proteinuria was reported. There was no complication during the labor and delivery. On the postpartum 10th hour her arterial pressure became 180/120 and she had a seizure. There are only a few reports in literature like our case and all are reported to be as postpartum late eclampsia cases. (*Gynecol Obstet Reprod Med* 2005; 11:68-69)

Key Words: Postpartum eclampsia, Proteinuria, Hypertension

Postpartum eclampsia is a rare, frightening and tragic complication of hypertensive pregnancies, usually developing within 48 hours of delivery,¹ but this period is also accepted to be beyond 48 hours up to 4th week postpartum called as late postpartum eclampsia.² Eclampsia is defined as the new onset of convulsions during pregnancy or postpartum, unrelated to other cerebral or pathologic conditions in a woman with preeclampsia. The reported rate of eclampsia in the Western World is 1 in 2000 to 1 in 3000 deliveries. The reported maternal mortality rates from eclampsia range from 0.5% in some centers in the United States to as high as 14% in Mexico.³ According to Miles et al preeclampsia recurred in 38.5% of subsequent gestations, it appears that this syndrome may be a predisposing factor for preeclampsia.⁴

Based on human autopsy data; radiologic investigations and animal studies, eclampsia is believed to result from explosive vazospasm, endothelial dysfunction and cytotoxic edema of cerebral cortex. In a study of Katz et al eclampsia is found to be a progression from severe preeclampsia. In 32 of 53 cases (60%) seizures were the first signs of preeclampsia.⁵

Case Report

We present a 23 year old primigravid woman whose antenatal follow-up began first at 11th gestational week. In all of her visits her arterial pressure was reported to be within normal limits. Ultrasonographic evaluation was performed 12th, 16th, 25th and 35th weeks and all were extremely normal. She did not have a history of hypertension before pregnancy, no proteinuria was reported.

Gazi University Faculty Of Medicine Department Of Gynecology And Obstetrics

Address of Correspondence Banu Bingöl

Güven Şeker Sitesi

E-Blok, No: 14

Çiğdem, Ankara

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The patient admitted to the Obstetrics Service at 40th weeks of gestation with uterin contractions. She gave birth to a baby weighting 2700 grams. The patient was normotensive prepartum and postpartum until 10th hour. She had sudden onset of severe headache with blood pressure of 180/120 mmHg and a generalized tonic clonic seizure soon followed. She was treated with diazepam and phenytoin.

Immediately intravenous magnesium was begun. Emergent head CT scan and transcranial Doppler was normal. Her spontaneous diuresis was more than 100 ml per hour. Biochemical results showed HELLP syndrome (aspartate aminotransferase: 74 IU/L, lactate dehydrogenase 1526 IU/L, platelet count: 78000). Her urine was taken by using a foley catheter and showed 2 (+) proteinuria with dipstick analysis. After the seizure, she was alert and oriented with mild neck stiffness but no focal neurological signs. MRI findings were consistent with minimal hyperintensities on T2 weighted images in the cortex and white matter. She received intravenous magnesium sulphate seizure prophylaxis during postpartum 24 hours consisting of a 4 gr. loading dose and a 2 gr. per hour maintenance dose. This dose was continued until the patient fulfilled clinical criteria for discontinuation of seizure prophylaxis. These criteria included: absence of persistent headache, visual changes and epigastric pain, greater than 50% of the hourly postpartum blood pressures less than 150 mmHg systolic and less than 100 mmHg diastolic (including the hour immediately before medication discontinuation); no indication for acute antihypertensive therapy within the preceeding 2 hours (systolic blood pressure greater than or equal to 160 mmHg or diastolic blood pressure greater than or equal to 110 mmHg and spontaneous diuresis of more than 100 ml per hour for 2 hours or more consecutively. Vital signs and urinary output were assessed hourly during the recovery unit. Vital signs were assessed every 4 hours until the time of hospital discharge. She did not experience a following seizure and her arterial pressure became within normal limits in 24 hours and proteinuria disappeared after four days. On the postpartum 5th day her MRI scan was extremely normal. EEG (electroencephalography) was performed.

med on postpartum 2nd week and 4th week both of which showed no pathological changes.

Discussion

Up to one third of cases of eclampsia occurs in the postpartum period. Often there is evidence of preeclampsia which alerts the physician to be prepared for the possibility of seizures.⁶ Although Lubarsky et al⁷ reported that 44% of their patients had not been identified as pre-eclamptic before seizure onset, they did not comment on whether pre-eclamptic signs were truly absent or were not identified due to insufficient caretaker attention towards pre-eclampsia during the postpartum period. There was no preeclamptic onset in our patient.

Eclampsia is accepted to be caused by ischemia from cerebral underperfusion resulting from this vasoconstriction, although some cases of eclampsia may be caused by hypertensive encephalopathy from cerebral overperfusion. Recent studies showed the presence of characteristic MRI findings in eclampsia which may partially represent vasogenic oedema.⁸ Conversely however the potential absence of these findings may lead to dismissal of the diagnosis. Our patient showed these characteristic multiple minimal hyperintensities on T2 weighted images in the cortex and white matter but we can not be able to find these characteristic MRI findings in all our eclamptic cases. A follow-up MRI shows complete resolution of all cortical lesions.

We conclude that postpartum eclampsia can manifest without a preceding preeclamptic phase. Although MRI is an important diagnostic test in atypical or severe eclampsia, it may fail to show characteristic findings early after onset of eclamptic seizures.

The significant reduction in the incidence of eclampsia through the years can be attributed to prenatal care; early detection of signs and symptoms of preeclampsia and prophylactic use of magnesium sulphate. Unfortunately such efforts at the education of parents and health care providers have not been continued into the postpartum period and seizures may begin without any prodromal signs of preeclampsia.

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