

An interesting Case of Delayed Onset Postpartum Eclampsia

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Postpartum eclampsia is defined as eclampsia that occurs after delivery within 48 h. Now-a-days, the incidence of antenatal and intrapartum eclampsia is reduced considerably due to good antenatal care, and preeclampsia diagnosed early and treated well. However, postpartum cases nowadays show a unique pattern occurring too late, which can be nearly missed and difficult to diagnose. Postpartum eclampsia has to be differentiated from other causes such as cerebral malaria, cerebral tumor, cerebral hemorrhage, cerebral venous thrombosis, cerebral aneurysm, brainstem syndrome, posterior reversible encephalopathy syndrome, epilepsy, thrombotic thrombocytopenic purpura, strychnine poisoning, chronic hypertension, chronic renal disease and hemolytic uremic syndrome. Postpartum eclampsia requires prompt treatment. Left untreated can result in seizures and other serious complications. Here, we present a unique and interesting case of atypical delayed onset postpartum eclampsia. Hence that such unique cases should not be missed in the diagnosis and management.

Keywords: Postpartum, Preeclampsia, Pregnancy

INTRODUCTION

Eclampsia occurring after delivery within 48 h is called postpartum eclampsia. Now-a-days a rare existence of late postpartum eclampsia, wherein seizures occurs 3 or 4 days and even 4 weeks postpartum without prodromal symptoms poses a dilemma in diagnosis. Proteinuria may be noted, blood pressure (BP) may be high, blood urea and uric acid levels are elevated soon after seizures, such variant of postpartum eclampsia cause not only difficulty in diagnosis, but also in management.¹ A late onset postpartum eclampsia is always a dilemma in diagnosis and difficult to treat. Lot of studies has to be done apart from the usual clinical history. Investigations like magnetic resonance imaging and computerized tomography may be required to rule out neurological conditions. Late onset postpartum eclampsia with headache and visual disturbances may be noted without proteinuria and hypertension in the antenatal period.² Such cases has to be immediately diagnosed. Its awareness has to be brought to the notice of nursing staff or in house medical officers. This is specially very essential in developing countries where maternal mortality rate is increased due to eclampsia as there are limited resources for investigations and management.³ Sometimes posterior reversible encephalopathy syndrome (PRES), wherein headache, seizures, loss of vision present in postpartum has to be differentiated from late onset postpartum eclampsia. In

PRES white matter changes will be noted in magnetic resonance imaging. A seizure when occurs after 48 h within 30 days of delivery is called late onset postpartum eclampsia.⁴ Magnesium sulfate is the drug of choice. There is increased risk of eclampsia 1-2% and preeclampsia 20-35% in next pregnancy with adverse outcome for both mother and fetus. It is mandatory to diagnose, prevent and manage such cases.⁵

CASE REPORT

A 30-year-old gravida 2, para1 live1 with history of previous lower segment caesarean section, a booked case, came for regular antenatal check-up at VMMC, Karaikal. Patient was normotensive, had pedal edema but no proteinuria. At 38 weeks, she got admitted with labor pain and had a repeat cesarean section for not progressing in labor. During post-operative period her BP was normal, she was conscious and well oriented. She had her suture removal done on the 7th post-operative day, and when she was about to get discharged, she complained of loss of vision, severe headache and threw seizures. There was the proteinuria (Figure 1) and severe hypertension - BP 160/100 mm of Hg (Figure 2). She was treated with magnesium sulfate and labetalol. Her previous BP was normal except for the pedal edema (Figure 3) she had no signs of preeclampsia on admission. She had all investigations done (Table 1). Computed tomography was

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Figure 1: Proteinuria by dipstick method in the patient



Figure 4: Computed tomography scan of postpartum eclampsia with normal findings



Figure 2: Elevated blood pressure during postpartum in the same patient



Figure 3: Bilateral pitting pedal edema in the postpartum period

also done (Figure 4). It is not a classical presentation of postpartum eclampsia since it occurred on the 7th day of the postpartum period. We had a medical opinion to rule out all the other causes of seizures. Hence, she was labeled as a delayed onset postpartum eclampsia. We studied the review of the literature and went through various articles to support our diagnosis.

Table 1: Investigations done after seizure episode

Laboratory investigations	Results
Platelet count	2,50,000 cells/mm ³
RBS	98 mg/dl
Blood urea	26 mg/dl
Serum creatinine	0.7 mg/dl
Serum uric acid	9 mg/dl
Total protein	6.5 g/dl
Albumin	4 g/dl
Globulin	2.5 g/dl
Total bilirubin	0.8 mg/dl
Direct bilirubin	0.3 mg/dl
Indirect bilirubin	0.5 mg/dl
ALT	20 U/L
AST	20 U/L
ALP	50 U/ml
LDH	120 U/L
Prothrombin time	10 s
Partial thromboplastin time	22 s
INR	1 IU
Sodium	140 mEq/L
Potassium	4 mEq/L
Bicarbonate	26 mEq/L
Chloride	100 mEq/L
Magnesium	1.2 mEq/L
urine routine	
Albumin	2+
Sugar	Nil
Pus cells	1-2/HPF
Epithelial cells	2-3/HPF

RBS: Random blood sugar, ALT: Alanine transaminase, AST: Aspartate aminotransferase, ALP: Alkaline phosphatase, LDH: Lactate dehydrogenase, INR: International normalized ratio

DISCUSSION

Nowadays, the incidence of antenatal and intrapartum eclampsia is reduced considerably due to good antenatal checkup and preeclampsia diagnosed promptly and treated

well. But postpartum cases nowadays show a unique pattern occurring too late which can be nearly missed and difficult to diagnose. Hence can result in cerebral hemorrhage and maternal mortality.⁶ The classical definition of postpartum eclampsia should occur within 48 h of delivery. Nowadays, we find cases which occur very late. Further, typical symptomology like proteinuria and hypertension which are considered the classical sign of eclampsia are no longer present initially, which further cause confusion in diagnosis.⁷ In spite of the difficulty in diagnosis prompt treatment with magnesium sulfate and labetalol resolves the disease, if one is aware of the late onset of postpartum eclampsia.⁸ The incidence of preeclampsia and eclampsia is 6-8% in all pregnancies. In the case of postpartum eclampsia convulsions occur within 48 h. When it occurs more than 48 h up to 4 weeks of delivery, it is called late postpartum eclampsia.⁹ Eclampsia should be diagnosed in any postpartum lady who develops hypertension, proteinuria and seizures and it is treatable with magnesium sulfate. The differential diagnosis includes epilepsy, cerebral venous thrombosis, cerebral hemorrhage and any space occupying lesions in the brain. Sometimes vertical blindness caused by posterior leukoencephalopathy, which is also treatable with magnesium therapy. Magnesium sulfate can prevent as well as treat eclampsia.¹⁰ Such cases that occur late cause a dilemma in diagnosis.¹¹ In the case of delayed postpartum eclampsia not only the diagnosis, but also the treatment is disputed. In a study states that serum levels of anti angiogenic and angiogenic proteins were measured, which helped in diagnosis.¹²

CONCLUSION

The complications in eclampsia are acute renal failure, acute liver failure and respiratory complications like aspiration pneumonia and acute pulmonary edema. The mortality in eclampsia is due to intracranial hemorrhage. Treatment of eclampsia should be initiated as early as possible in order to prevent complications. In modern obstetrics, the time limit for postpartum eclampsia has to be altered as it occurs even after 48 h. It is essential to educate the public and medical

staff regarding the late occurrence of postpartum eclampsia. The awareness is very essential in order to prevent the morbidity and mortality. Although there is a dilemma in diagnosis, it is treatable.

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