Complex partial seizure with postictal aggression as a presentation of atypical eclampsia: a case report

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ABSTRACT

Background: Complex partial seizure with sudden onset in near term pregnancy can be challenging to the attending clinician and it is essential to proceed with a presumptive diagnosis of eclampsia, especially in resource poor settings. Unlike generalized tonic clonic seizures, which are more common in eclampsia, complex partial seizures may not jeopardize the fetus with the same severity. However the associated postictal aggression manifested in self-inflicted trauma poses an imminent risk onto the fetus.

Case presentation: A nineteen year old gravida two para one living zero woman presented with seizure at thirty six weeks and three days of gestation in a district hospital. The blood pressure recordings during her entire pregnancy were normal and proteinuria was not present. Her previous pregnancy had a similar course, with self-inflicted trauma to her abdomen in a postictal state and resulted in loss of fetus. She did not have any seizures in between then and the current admission. While being managed in the labor ward, the family physicians witnessed complex partial seizure with postictal aggression. The delivery was expedited with caesarean section and she had no further seizure activity.

Conclusion: Complex partial seizures with postictal aggression in pregnancy are rare but can be an atypical presentation of eclampsia and poses a diagnostic dilemma to the clinician.

Keywords: Complex partial seizure, postictal aggression, atypical eclampsia, district hospital.

BACKGROUND

Eclampsia complicates around 1 in 2000 pregnancies in developed countries and the incidence increases in developing countries, ranging from 1 per 100 to 1 per 1700 pregnancies. Maternal mortality is around 1 in 50 in women with eclampsia and perinatal deaths are found to be 1 in 14 pregnancies with eclampsia¹-². The risk of recurrence is 1.4% in subsequent pregnancies³. Most cases of eclampsia present in the third trimester or immediately postpartum. It is not necessary for preeclampsia to precede eclampsia. Eclampsia can also present without accompanying hypertension or proteinuria¹. Generalized tonic clonic seizures has been considered the prototype of eclampsia. Unexplained seizures occurring without prior symptoms and signs of preeclampsia, before 20 weeks of gestation or after 48 to 72 hours postpartum is termed atypical
eclampsia and constitutes about 10% of eclamptic cases\textsuperscript{4}. Over the last three decades, several cases of atypical eclampsia have been reported, and only one case with partial seizure in eclampsia has been documented\textsuperscript{6}. This rarity is further superseded by episodes of postictal aggression. Postictal aggression is a manifestation in postictal state characterized by episodes of physical or verbal aggression and has been described with cases of epilepsy\textsuperscript{7}. Complex partial seizures are not common in eclampsia and are assumed not to harm the fetus like generalized seizures do\textsuperscript{5}. However the risk to the fetus in the case of maternal complex partial seizures is further increased with self inflicted aggression in postictal state.

**CASE PRESENTATION**

A nineteen year old gravida two para one living zero woman was referred to us for sudden onset seizure at thirty six weeks and three days of gestation. Three days following her antenatal visit at thirty-six weeks, she suddenly started having abnormal movement of her right lower limb, which lasted for five minutes and thereafter she fell unconscious. She was alone at home lying unconscious when she was noticed by her mother in law. The total duration of unconsciousness could not be ascertained. She was rushed to a nearby district hospital, which took three hours, where she was immediately given a loading dose of magnesium sulphate as per Pritchard regimen and was referred. She was receiving regular antenatal care following national protocol. Since she had neonatal death of her first child, she was advised for monthly follow up and she had completed five antenatal visits in this pregnancy. The recordings of her blood pressure and weight gain were normal during these visits. On arrival, she was conscious and oriented. Her temperature and blood pressure were normal with minimal edema on both her lower limbs. Her central nervous system examination was normal. She was evaluated for eclampsia, which included complete blood counts, renal function tests, liver function tests, serum uric acid, urine routine microscopy and fetal scan. Her normal laboratory investigations compelled us to further consider her diagnosis and management. Her fetal scan was also normal.

Her past history revealed that around thirty-six weeks during her first pregnancy, she had sudden onset of similar myoclonic seizure involving her right lower limb which lasted for around one and half hours followed by unconsciousness. She stated that witnesses reported she had abnormal movements involving all four limbs, with rigidity, frothing of mouth, tongue bite and loss of bowel and bladder control. She was rushed to the district hospital in her unconscious state where she was managed for eclampsia with magnesium sulphate. In the hospital, as she started to gain consciousness, she suddenly became violent and started banging her own abdomen with her fist. She confirmed that she did not remember those events. She underwent caesarian section and delivered a baby with poor Apgar score. The baby could not be resuscitated and was declared dead at thirty minutes of life. She failed to comply with contraception advised by the clinician thereafter and became pregnant for the second time at six months postpartum. She denied any seizure activity prior to her first pregnancy and in-between the period after delivery of her first child until this latest hospital stay. There are no such episodes in her other family members as well. She did not have any complaints of repeated headache, vomiting, visual disturbances, weakness or tingling of limbs, inappropriate behavior, hallucinations, persecutory delusions, or head trauma in the past. Her family history was normal.

Our differential diagnosis included epilepsy and dissociative disorder and we admitted her for observation. We continued her on magnesium sulphate, as we could not totally rule out eclampsia. We started regular fetal heart rate monitoring. Magnesium sulphate was stopped on the second day
after completing 24 hours as no further seizure episode was witnessed. On her third day of admission, she started having myoclonic seizure of right lower limb for five to six minutes followed by unconsciousness for around two to three minutes and suddenly she emerged with aggression previously described, banging her abdomen with fist, and biting as we tried to restrain her. She was sedated with intravenous midazolam. Fetal scan was repeated at bedside and was normal. Almost two hours later as she regained consciousness, we interviewed her and she denied remembering anything after few minutes of focal seizure. An hour later the scene repeated itself and we had her sedated again. Since her seizure frequency had increased along with postictal aggression, we decided to proceed caesarian section and she delivered a healthy newborn with normal Apgar scores.

She was kept for observation in hospital for ten days. No further seizure activity was witnessed. Her laboratory investigation repeated postpartum did not reveal any abnormality. She was also followed on telephone four weeks postpartum and we found her normal without seizure activity.

**DISCUSSION**

In both of the consecutive pregnancies of this patient, there were focal seizures associated with postictal aggression, which is quiet unusual in eclampsia and compelled us to think of an alternative diagnosis and put us in a dilemma as whether to continue magnesium sulphate till delivery. There have been very few case reports on complex partial seizure in pregnancy and these cases were secondary to tuberculoma⁹, neurocysticercosis¹⁰ and venous thrombosis¹¹. None of them have reported postictal aggression. Complex partial seizures causes uterine hyperactivity, which may result in fetal bradycardia⁶. During her first pregnancy, as her focal seizure underwent secondary generalization, her fetus might have had suffered in utero hypoxia resulting in low Apgar and fetal death. Despite the occurrence of complex partial seizure in her second pregnancy, the fetal outcome was satisfactory.

Postictal state is a known phenomenon in seizure. Nevertheless postictal aggression as in this case is rarely observed. Punching oneself and biting behavior during postictal state have been studied. Postictal aggression was reported more in males and was associated with front temporal origin. Someone entering the space near the patient was stated as the triggering factor⁷. These episodes of aggression further jeopardize the viability of fetus in utero by direct trauma to the abdomen, increasing the risk of abruption or direct fetus injury.

Availability of computerized tomographic scan in our district hospital might have helped to rule out organic pathology. Nevertheless, absence of repeated headache, visual disturbances, focal neurological deficits, typical time of onset and seizure free interval prior to and in-between her two pregnancies together imply a diagnosis of eclampsia.

**CONCLUSION**

Eclampsia can manifest in absence of any heralding symptoms and signs and may not be accompanied by hypertension or proteinuria. Complex partial seizures, though rare, can occur with eclampsia. It is necessary to consider other possible diagnoses including epilepsy when seizures in pregnancy fail to meet the criteria of eclampsia. However, our case was consistent with atypical eclampsia. Postictal aggression is an unusual phenomenon in postictal period and has not been reported in eclampsia yet, but has been described in epilepsy and appears possible with seizures. Timely recognition of diagnosis, prompt intervention with magnesium sulphate and delivery could improve fetal outcomes in atypical eclampsia.

PKBA was responsible for managing this case & drafted the manuscript. BG and SKD were responsible for managing this case and was involved in revision of the manuscript. LW participated in literature reviews for this case report and provided feedback on the manuscript. All authors have read the final manuscript and approved it.
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REFERENCES


