DOI: 10.7860/JCDR/2019/39750.12507

Case Report

Obstetrics and Gynaecology Section

Late Onset Atypical Eclampsia: A Case Report

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ABSTRACT

Hypertensive disorders in pregnancy account for substantial maternal and peri-natal morbidity and mortality. Atypical eclampsia presents as a diagnostic dilemma and challenge for the treating obstetrician. The purpose of this case report is to increase awareness of the nonclassic and atypical features of eclampsia so as to avoid complications. Whilst controlling the convulsions by instituting magnesium sulfate therapy, simultaneous search for any organic/metabolic cause for seizure needs to be looked for. Constant vigilance with a high level of suspicion is required for a more positive maternal and foetal outcome.

Keywords: Magnesium sulfate therapy, Postpartum, Seizure

CASE REPORT

A 25-year-old P4L4 referred form one of the community health center with multiple episodes of tonic clonic convulsion, altered consciousness and restlessness since eight hours. There was no history of headache, epigastric pain or blurred vision. She delivered at home 3 weeks back. According to attendant, there was no undue prolongation of labour stages. Baby cried immediately after birth. There was no history of postpartum haemorrhage. There was no history of seizure disorder, other CNS disorder or cardiac disease. There was no prenatal care. Patient received two dose of tetanus toxoid during antenatal period. Previous deliveries were also done at home. No history of swelling in the leg or other premonitory symptoms during antenatal period was observed. Patient received loading dose (Total 14 mg of which 4 mg intravenous infusion and 5 mg intramuscularly in each buttock) of magnesium sufate (Magsulf) at community health center. On examination patient was restless, semiconscious, disoriented, not responding to verbal command. (E2M4V2). There was no sign of meningeal irritation or sensory motor deficit. Patient was afebrile. Her BP was 140/100, pulse was 92/min. Her tongue was bitten and there was oozing from the bitten area. Patellar reflexes and neck rigidity were absent, bilateral plantar were equivocal, Bilateral pinpoint pupil, sluggishly reacting to light. Cardio-respiratory system was within normal limit except added sound on respiration. On per abdominal examination uterus was 14 week size and well contracted. On per vaginal examination internal os was closed and lochia was healthy. Immediate neurology consultation was taken, routine investigation along with magnesium level was sent, CECT brain was advised. Subsequent doses of Magsulf were withheld due to absence of patellar reflex and patient shifted to ICU. Injection leviteracetam 500 mg was started. CECT was normal. Serum electrolyte, blood sugar, LFT, RFT platelet, coagulation profile, Chest X-ray and whole abdominal ultrasonography were within normal limit except sinus tachycardia in ECG and urinary protein was +3, AST and ALT were 134 U/L and 117 U/L. Her magnesium level was 3.6 mh/dL and APTT was 43.7 second. HIV hepatitis B and C and VDRL were negative. Her fundus examination was normal. There was no papilloedema. MRI brain was done which showed post seizure changes. EEG was normal. Patient was discharged on day seven of admission on tablet levitiracetam 500 mg twice a day as per neurologist advice and was asked to review in gynaecology and neurology OPD.

DISCUSSION

Hypertensive disorders of pregnancy contribute significantly to maternal and perinatal mortality in both industrialised as well as developing countries. However, little is known about “atypical preeclampsia-eclampsia syndrome”. Historically, it has been used to describe non-classical forms of hypertensive disorders arising during pregnancy. Eclampsia if occurs before the development of hypertension and/or proteinuria, developing before 20 weeks of pregnancy and 48 hours of postpartum, or inspite of receiving magnesium sulphate it occurs, then it is termed as atypical eclampsia [1]. Trouble with atypical forms of eclampsia is in its unpredictable onset; well timed diagnosis and management are crucial in avoiding morbidity and mortality.

The most common cause of convulsions developing in association with hypertension and/or proteinuria during pregnancy or immediately postpartum is eclampsia. However some other medical conditions can also cause convulsions during pregnancy. The main differentials of eclampsia are cerebrovascular accident like haemorrhage, ruptured aneurysm or malformation, arterial embolism or thrombosis, cerebral venous thrombosis, hypoxic ischemic encephalopathy, hypertensive encephalopathy, seizure disorders, previously undiagnosed brain tumour, metastatic gestational trophoblastic disease, metabolic disorder like hypoglycaemia, hyponatremia, reversible posterior leukoencephalopathy syndrome, Thrombophilia (catastrophic antiphospholipid syndrome), Thrombotic thrombocytopenic purpura, Postdural puncture syndrome and Cerebral vasculits [2].

Differential diagnoses are primarily important in the presence of focal neurologic deficits, prolonged coma, or atypical eclampsia. These differentials need to be ruled out on cardiac and neurological evaluation in the patient.

Patients can have convulsions in absence of hypertension and proteinuria representing cerebral (seizures) involvement of the disease process prior to arterial (hypertension) and renal (proteinuria) involvement. Suspicious findings may include marginally elevated BP or liver enzymes, fetal distress, blurred vision, and headache. Our patient was having high blood pressure (140/100) her urine albumin was 3+ and her AST and ALT were 134 U/L and 117 U/L. Her EEG, CT and MRI scan were normal. There was no abnormal pathology. Our diagnosis was based on clinical presentation, biochemical parameter and excluding other causes by performing various appropriate investigations. Late postpartum eclampsia is

defined as the development of signs and symptoms of eclampsia for the first time at >48 hours but <4 weeks after delivery [3,4]. This case comes under this category. Graves JC et al., presented a case of eclampsia late in the postpartum period (10 days) and the equally rare onset of eclampsia without prior evidence of preeclampsia during her pregnancy [5]. In such patients, magnesium sulfate therapy should be initiated without delay while other possible causes as mentioned being ruled out [6,7]. Also, if the condition does not respond to such therapy or the patient continues to have seizures despite magnesium sulfate therapy or continues to have cerebral symptoms, then neuro-imaging with MRI and angiography, should be performed to rule out the presence of other cerebral disease [7]. The classic finding in eclampsia is posterior reversible encephalopathy syndrome. Another differential diagnosis is spontaneous reversible vasculopathy syndrome or cerebral angiopathy in the presence of unexplained blindness or other neurologic deficits [8]. This can be diagnosed by magnetic resonance angiograph or traditional cerebralangiography. One more such case of atypical eclampsia was presented on day four postpartum. She was not having prior features of preeclampsia except that she had mild headache and nausea before convulsion [9]. Albayarak M et al., also presented four various presentation of atypical preeclampsia and eclampsia [10].

Sibai BM et al., in their study reported that 31.2% of eclampsia was not avoidable inspite of proper antenatal management [11]. In one of the study in 1992 from United Kingdom 38% of the patient had eclamptic convulsions before the onset of proteinuria and hypertension and 75% of these developed in the hospital [12]. These findings replicate that many times early recognition and management is difficult in atypical presentations.

While managing these patients of atypical eclampsia following factors like clinical presentation, laboratory, and imaging findings, the time of onset in relation to gestational age, and delivery should be taken into consideration.

Shin JE et al., in their retrospective study compared the outcomes after typical and atypical ecampsia and concluded that perinatal outcomes were better in atypical cases, however, maternal outcomes were similar. Therefore, and more attention should be focused on atypical eclampsia [13].

CONCLUSION

Hypertensive diseases in pregnancy possess a serious threat to maternal and fetal life. Atypical eclampsia further complicates the case scenario because of delay in diagnosis and management. Obstetricians should be aware of atypical presentations, maintain a high level of suspicion, irrespective of gestational age at the time of onset or the number of days after delivery, and be ready to take immediate steps. Awareness regarding this clinical entity and prompt identification can certainly improve the feto-maternal prognosis. Alternately, one must be careful to avoid mis-diagnosis or over-diagnosis of other conditions, that might lead to unnecessary intervention.

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FINANCIAL OR OTHER COMPETING INTERESTS: None.

Date of Submission: **Sep 26, 2018**
Date of Peer Review: **Nov 15, 2018**
Date of Acceptance: **Nov 26, 2018**
Date of Publishing: **Jan 01, 2019**