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Case Report

Atypical Eclampsia

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Abstract

Eclampsia occurring without prior signs and symptoms of preeclampsia is called atypical eclampsia. We present a case of 28 year old primigravida who developed intrapartum seizures. There was no prior clinical or biochemical evidence of preeclampsia. We have discussed the differentials of intrapartum seizures and a review of literature on atypical eclampsia.

Introduction

Eclampsia is defined as the development of convulsions and/or unexplained coma during pregnancy or postpartum in patients with signs and symptoms of preeclampsia after 20 weeks prepartum and before 48 hours postpartum.[1] Occurrence of Eclampsia before 20 weeks, after 48 hours postpartum or in absence of typical signs of hypertension and/or protienurea is termed as atypical Eclampsia.[2] Correct and timely diagnosis and management of these cases is challenging for obstetricians. We here present a case of intrapartum eclampsia without prior history of preeclampsia.

Case Report

A 28 year old Primigravida was booked at 8 weeks of pregnancy. Her antenatal course was unremarkable and she remained normotensive throughout this period. However, her family history was strongly positive for hypertension as both her parents were hypertensive. All booking laboratory investigations including haemoglobin, platelets, Rubella IgG, Hepatitis BsAg, Anti HCV antibody, ABO blood group compatibility were within normal limits. Urine analysis showed no protienurea during antenatal visits. She did not complain of any headache, visual changes, or epigastric pain before the delivery.

She presented at 38 weeks with spontaneous onset of labour, which was augmented with amniotomy and oxytocin at five centimeter dilatation of cervix. Her first stage of labour lasted for 07 hours and was uneventful. During the second stage she suddenly developed tonic clonic seizures which lasted for one minute accompanied with frothing of mouth, up rolling of eyes and cyanosis. Her vitals were as follows: Blood pressure 70/30 mmHg, Pulse 98 beats per minute, there were no respiratory effort and oxygen saturation dropped to 76%. Cardiopulmonary resuscitation was performed and patient was intubated. Crash Caesarean was performed within 4 minutes of intubation. Intraoperatively her blood pressure went up to 160/110 mmHg. Patient delivered a female baby girl with a good APGAR SCORE. Baby weighed 2.09 kg. No congenital abnormality was present. During her stay, neurology and cardiology opinion was sought. Neurological exam and EEG were normal. ECG and Echocardiography were unremarkable. Cardiology consult ruled out any cardiac cause. Laboratory workup including complete blood count, serum electrolytes, renal functions test, liver functions test and coagulation profile was within normal limits. Random blood sugar was 89 mg/dl. Uric acid 5.7 mg/dl. Serum D.Dimer was 1.47.Urine Detailed Report showed: No protein, haemoglobin: +3, nitrite absent, RBC>20/hpf, WBC Nil, Epithelial cells Nil. Arterial Blood Gases: pH: 7.32, PCO2 27.2 mmHg, P O2 65 mmHg, HCO3 14 mmol/dl, Oxygen Saturation 99.7 % (Room air). Her tracheal aspirate showed normal respiratory epithelial cells. Post operatively she remained normotensive for the first twenty four hours. Later on her blood pressure started rising gradually with consistently high diastolic pressures of 105-110mmHg. Atenolol 100 mg per day was started that stablized her blood pressure. She was discharged on third post operative day. Her postnatal follow ups in clinic were unremarkable.

Discussion

Pre-eclampsia is a multisystem disorder. It usually warns about occurrence of eclampsia beforehand. However, hypertension is only one of the signs and is not always present in pre-eclamptic patients developing eclampsia.[3] It is difficult to predict which organ system will predominantly be involved. Eclampsia, a rare but serious complication of preeclampsia, becomes more problematic when it develops without prior preeclamptic signs and symptoms.[4] In our case, patient remained free of signs and symptoms throughout antenatal period but became hypertensive intraoperatively and in the postoperative period.

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 given in the Table.[1] These diagnoses are

Table: Differential Diagnosis of Eclampsia.[1]

DIFERENTIAL DIAGNOSIS OF ECLAMPSIA
■ Cerebrovascular accidents
● Haemorrhage
● Ruptured aneurysm or malformation
● Arterial embolism or thrombosis
● Cerebral venous thrombosis
● Hypoxic ischemic encephalopathy
● Angiomas
■ Hypertensive encephalopathy
■ Seizure disorder
■ Previously undiagnosed brain tumours
■ Metastatic gestational trophoblastic disease
■ Metabolic disease
● Hypoglycemia, hyponatremia
■ Reversible posterior leukoencephalopathy syndrome
■ Thrombophilia
■ Postdural puncture syndrome
■ Cerebral vasculitis

particularly important in the presence of focal neurologic deficits or prolonged coma. These differentials were ruled out on cardiac and neurological evaluation in our patient.

Another important cause for Intrapartum seizures/sudden maternal collapse is amniotic fluid embolism which carries a very high maternal mortality rate of 13-90%.[5] To rule out amniotic fluid embolism in our case, tracheal aspirate was analyzed which showed absence of foetal squamous epithelial cells. In the absence of any other cause but the persistent hypertension, patient was labeled as having an atypical eclampsia.

Maternal collapse, due to whatever reason, remains a feared obstetric complication. Its initial management is a challenge for those most likely to be immediately present - obstetricians and midwives. Decision to perform a perimortem Caesarean section in this situation depends on preceding condition, timing of cardiac arrest and initial resuscitation effort. Shorter the duration between onset of cardiac arrest and commencement of cardiopulmonary resuscitation and the shorter time taken to deliver the foetus once cardiopulmonary resuscitation is in progress, the more likely is that a surviving foetus will be neurologically intact. Survival is best (70%) if Caesarean is performed within five minuets of start of cardiopulmonary resuscitation.[6] Early delivery not only saves the baby but also improves venous return to heart of the mother due to decompression of gravid uterus occluding inferior vena cava. In our case, Caesarean section was performed within four minutes of start of cardiopulmonary resuscitation and baby was delivered alive with good APGAR score.

Prompt and appropriate intervention is critical to maximize the survival possibilities for the mother and baby in case of sudden maternal collapse. It is of utmost importance that the people who are involved in maternal care during delivery should be prepared for these unusual circumstances. They should be familiar with basic life support, use of adjuvant airways and attempting defibrillation within three minutes of collapse. Decision to perform a perimortem Caesarean should be taken accordingly without any delay. Staff must be trained on annual basis for obstetrics emergenscies and regular drills should be performed to improve the survival.[5]

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