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A Case of Recurrent Atypical Eclampsia with Recurrent Pregnancy Loss and Management in Present Pregnancy.

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ABSTRACT

An eclampsia occurring in absence of hypertension and/or proteinuria, before 20weeks, after 48hours postpartum and/or without prior signs and symptoms of preeclampsia is termed as atypical eclampsia. Here is a case report of 30yrs old female who is gravid5, para3, dead3, ectopic pregnancy1 with previous 2 intra-uterine death and 1 still birth with previous 2 consecutive intra-partum eclampsia with no signs and symptoms of preeclampsia. In present pregnancy patient was normotensive but had mild proteinuria at 36weeks. An atypical eclampsia can be in any unpredictable form. This case report purpose is to discuss differentials in atypical eclampsia and increase its awareness so as for early diagnosis and management thereby preventing fetal and maternal complications.

Keywords: eclampsia, atypical eclampsia, intra-partum eclampsia, recurrent pregnancy loss.

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INTRODUCTION

The word eclampsia is from greek term for lightening. The first known description eclampsia was by Hippocrates in the 5th century BCE [1]. Typical eclampsia affects about 1.4% of deliveries whereas atypical eclampsia constitutes about 8% of eclamptic cases [2]. Intra-partum eclampsia constitutes for about 15-20%(3). Eclampsia occurring without prior signs and symptoms of preeclampsia is termed as atypical eclampsia. As we all know that pre-eclampsia is a manifestation of placental bed deficiency, where eclampsia is in the extreme end of maternal complication and intrauterine fetal demise at the extreme end of fetal complications. Eclampsia is a multisystem disorder and an obstetrical emergency where quick and timely decision is required. The mainstay of treatment is to control the convulsive episode and delivery of the baby.

CASE REPORT

A 30 years old female booked at us from first trimester who is gravid5, para3, dead3, ectopic pregnancy1 with previous 2 intra-uterine death and 1 still birth with previous 2 consecutive intra-partum eclampsia with no signs and symptoms of preeclampsia. LMP-20/7/14, EDD-27/4/15 with gestational age of 36weeks as per LMP admitted with mild proteinuria for safe confinement. Her obstetrical history was, first pregnancy- spontaneous conception, she was unbooked and hence no antenatal records were available, perceives fetal movements well till the delivery time and delivered a girl term baby which was a still birth. Second pregnancy-spontaneous conception after 1year of previous pregnancy, she was normotensive and had no proteinuria throughout pregnancy, two days prior to EDD patient entered into labour and was admitted in hospital. Patient was diagnosed to have intrauterine fetal demise and labour accelerated. Patient developed intra-partum eclampsia which was appropriately managed and patient delivered a dead born girl baby. Third pregnancy- was spontaneous conception after 1 year of previous pregnancy, with similar history where two days prior to EDD patient entered into labour and was admitted in hospital. Patient was diagnosed to have intra-uterine fetal demise and labour accelerated. Patient developed intra-partum eclampsia which was appropriately managed and delivered a dead male baby. Fourth pregnancy was a chronic ectopic pregnancy for which she underwent laparotomy with salpingectomy. Fifth pregnancy (present one) was after 5months of previous pregnancy. First trimester was uneventful. In second trimester patient was diagnosed as gestational diabetes mellitus and was on meal plan (FBS-94, PPBS 126). Neurology opinion was obtained and gave differential diagnosis of 1)pregnancy induced hypertension 2)seizure complicating pregnancy, hence patient was prophylactically started on tablet tor-leva (Levetiracetam) 500mg BD and tablet ecospirin 75mg till 35weeks of gestation. Third trimester was uneventful. Patients remained normotensive throughout her pregnancy. All laboratory investigations were done which include haemoglobin, platelets, RFT, HIV, HbsAg, VDRL, urine routine analysis, ABO blood group compatibility were within normal limits. Urine analysis showed no proteinuria till 36weeks of gestation. Patient was admitted with proteinuria(1+) but normal blood pressure and no other signs and symptoms of preeclampsia. One course of steroids was given, ultrasound Doppler study showed normal flow with adequate liquor amnii. Elective caesarean section was planned at the completion of 37weeks of gestation in view of precious pregnancy and intra-uterine fetal demise after 38weeks of gestation in previous pregnancies. Neurology review was obtained and adviced Inj. Torleva 1gm in 100ml NS over 20 mins followed by 500gm IV BD for 2days prior to planning for LSCS. Patient was strictly monitored for blood pressure and fetal movements. At 37weeks of gestation patient developed labour pain. In view of G5P3D3E1, with recurrent pregnancy loss and 2 recurrent intra-partum eclampsia in labour patient was taken up for emergency LSCS under spinal anesthesia. Intra-operatively blood pressure was normal and an alive term male baby delivered weighing 2.4 kg with APGAR score of 8/10,9/10. Post operatively patient was monitored closely and was uneventful. Patient was discharged after 1week.Patient postnatal checkup until 6 weeks were normal.

DISCUSSION

Eclampsia is defined as convulsive episode occurring in women in antenatal or postpartum period. In developing countries it constitutes for about 1 in 100 to 1 in 1700cases[4].There is loss of cerebral vascular auto-regulation which leads to either over dilatation or intense vasospasm of cerebral arterioles. In auto-regulatory response to severe hypertension cerebral vasoconstriction occur leading to ischemia cyto-toxic edema and infarction. Another theory is when the cerebral vascular auto-regulation fails it causes dilatation of vessels resulting in hyper-perfusion and vasogenic edema [5]. The classic teaching that eclampsia is the end point of a disease process, starting sub-clinically and proceeding to mild pre-eclampsia and then severe pre-eclampsia implies that hypertension and proteinuria should precede the onset of eclampsia. In contrast to this

paradigm, eclampsia can potentially be encountered at the beginning of the disease process before hypertension and proteinuria develops [6]. In eclampsia the first organ to involve are arteries and kidney thereby presenting with hypertension and proteinuria and then other organs are involved. Whereas in atypical eclampsia there is a cerebral involvement first leading to eclapmsia, followed by involvement of kidneys and placenta which can lead to fetal anoxia, cerebral haemorrhages, intra-uterine fetal demise or prematurity. The most common causes for maternal death are intracranial bleeding and acute renal failure secondary to abruption placenta. The treatment mainly includes control of convulsions, control of hypertension and delivery of fetus. Hence instead of relying completely on the presence of hypertension and proteinuria, management plan should be initiated immediately in atypical cases. As in our case, even a minor sign such as mild proteinuria should not be overlooked and which could be an alarming sign in the absence of hypertension and severe proteinuria. Sibai et al. reported that 56 of 179 cases of eclampsia were potentially unavoidable, despite proper antenatal care and management. Of the 56 patients, 24 convulsed, despite a recent prenatal visit with no previous hypertension, proteinuria, or symptoms suggestive of the forthcoming eclampsia . The extension of this series to 254 eclamptic cases found that 80 patients (32%) did not have edema, 58 (23%) had minimal or no hypertension, and 49 (19%) did not have proteinuria at the time of convulsions [7-8]. These findings show the challenges in the early recognition and management of atypical presentations and hence the treating obstetricians should be aware of atypical cases, maintain a high level of suspicion, and take immediate steps.

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