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Atypical Eclampsia - Must - Know Obstetric Complication

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Abstract

Atypical eclampsia is occurrence of Eclampsia before 20 weeks, after 48 hours postpartum or in absence of typical signs of hypertension and/or proteinuria. Such atypical and non-classic features pose a challenge to the treating obstetrician. We present here such a case of atypical eclampsia who presented with convulsion and proteinuria alone. She was rightly diagnosed and promptly treated.

Keywords*: Atypical Eclampsia; Convulsion; Proteinuria; Magnesium Sulphate Therapy.*

Key message: *Aypical Eclampsia Diagnosis and Management is a Challenge to the Obstetrician.*

Introduction

Eclampsia is defined as the development of convulsions and/or unexplained coma during pregnancy or postpartum in patients with signs and symptoms of pre-eclampsia after 20 weeks pre partum and before 48 hours postpartum [1]. Recent data suggest that in some women, pre-eclampsia and even eclampsia may develop in the absence of hypertension or proteinuria. Occurrence of Eclampsia before 20 weeks, after 48 hours postpartum or in absence of typical signs of hypertension and/or proteinuria is termed as atypical Eclampsia [2]. Atypical eclampsia constitutes about 8% of eclamptic cases [3]. Problems with the atypical forms are their unpredictable onset and thus the difficulty in making a timely diagnosis to initiate management, which is critical in avoiding complications. We present a case of atypical eclampsia and discuss the challenges we faced, with an overview of the literature.

Case History

A 42 years old $G_{10}P_9L_9$ presented to our emergency department with the complaint of fits off and on since 12 hours. This was the first pregnancy from her third marriage. She had reported first at the nearest Primary Health Centre (PHC) with the history of fits at 30 weeks of gestation. Over the PHC referral letter her recorded Blood Pressure (BP) was 80/60mm of Hg with unconsciousness. There is no documentation of administration of Magnesium sulphate or antihypertensive therapy to the patient. She reported in our emergency department in an unconscious state and also had one episode of generalized tonic clonic convulsion. On examination, she was unconscious not responding to painful stimuli, had mild pallor, no icterus or pedal edema. Her BP was 130/90mm of Hg, pulse-112/min. and respiratory rate was 20/min. Respiratory and cardiovascular system examination were within normal limits, but with SpO_2 of 94%. On obstetric examination, fundal height was 32 wks with fetal heart rate (FHR) of 145 beats/min and on per vaginal examination, it was closed os with long posterior cervix. Patient was immediately shifted to Intensive Care Unit and put on ventilator. All relevant investigations were done. On bed side urine examination, proteinuria was found ++. With the provisional diagnosis of atypical eclampsia, Magnesium sulphate anticonvulsant therapy was started. Bed side ultrasonography showed 30 weeks pregnancy with FHR-140/min and mildly decreased liquor. Her Hb -12.3 gm %, TLC-20,000/CUMM, DLC- N82L15M2E1, SGOT 95IU/L, SGPT-50IU/L, Total Protien -5.4gm/

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dl, albumin -2.5gm/l, globulin-2.9gm/dl, serum bilirubin -0.6 mg /dl,serum and magnesium 1.3mg/dl, Serum K+ 4.5 mmol/l, Na+145mmol/l, coagulation profile was within normal limit. Blood urea and serum creatinine were within normal limit which excludes renal causes of proteinuria. But serum uric acid was found high at 7.6mg/dl. Since, bishop score was unfavouarble, decision for termination of pregnancy by caesarean section was taken. A male baby of birth weight 1.5 kg with average gestational age of 32 weeks was delivered. Intra operative period was quite uneventful and patient was shifted back to ICU and baby put in Neonatal Intensive Care Unit. As recommended, Magnesium sulphate therapy was continued for 24 hour of delivery. Post operative period was uneventful with no more fits. She gradually started to respond to painful stimuli, opened her eyes and responded to verbal commands. She was then weaned of ventilator and her urine albumin also became negative. On 7th day her liver enzymes returned to normal limits. Caesarean wound was found healthy and suture removal done. During her 10 days stay in the hospital her BP was never high, also she was not on any antihypertensive medication. Unfortunately the baby expired due to extreme prematurity and Hyaline Membrane Disease. Patient was finally discharged on 10th post operative day and she had also come for follow-up once with absolutely no complication.

Discussion

Eclampsia remains a leading cause of maternal and perinatal mortality and morbidity. 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. So, in our case, a thorough neurological and cardio logical examination and relevant investigations were done to rule out all other differential diagnoses of convulsion in pregnancy. Primigravida are at higher risk of convulsions and antepartum convulsions are more dangerous than those beginning after delivery [4]. In women, the partner-specific protective effect of insemination in pre-eclampsia might be explained by induction of immunological hypo-responsiveness conferring tolerance to histocompatibility antigens present in the ejaculate and shared by the conceptus [5]. Even in our case, though she was $G_{10}P_9L_{9,}$ new paternity might be a contributing factor.

In our case, apart from convulsion that responded to Magnesium sulphate therapy, patient had protineuria, elevated liver enzymes and high uric acid level. Suspicious findings may include marginally elevated BP or liver enzymes, fetal distress, blurred vision, and headache. The initial diagnosis should be atypical pre-eclampsia and eclampsia when suspected and a management plan should be started immediately for atypical forms, rather than searching for a rare disease in a differential diagnosis [1]. Atypical eclampsia constitutes about 8% of eclamptic cases [3]. Problems with the atypical forms are their unpredictable onset and thus the difficulty in making a timely diagnosis to initiate management, which is critical in avoiding complications. Prenatal follow-up must be very cautious paying attention to any markers such as intermittent hypertension, functional symptoms or appearance of proteinuria [6].

Conclusion

In conclusion, the absence of hypertension or proteinuria should not preclude the diagnosis of preeclampsia/eclampsia. Even minor clues of marginally elevated BP or trace proteinuria, may be critical for appropriate, timely management. Obstetricians should be aware of atypical presentations, maintain a high level of suspicion, and be ready to for prompt management.

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