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ATYPICAL ECLAMPSIA WITH POSTERIOR (PROGRESSIVE) REVERSIBLE ENCEPHALOPATHY SYNDROME

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Abstract : Atypical eclampsia refers to the occurrence of eclampsia with signs and symptoms of preeclampsia before 20 weeks of gestation, after 48hours postpartum or in the absence of signs and symptoms of preeclampsia. Posterior (progressive) reversible encephalopathy syndrome (PRES) is one of the complication of preeclampsia and eclampsia, where cerebral auto regulation is lost, leading on to cerebral hyperperfusion and vasogenic edema involving mainly the parieto occipital cortex. The important concern regarding atypical eclampsia is its unpredictable onset, if undiagnosed, is the leading causing of maternal and perinatal morbidity and mortality. Anticipation, timely diagnosis and management is mandatory. Here is one such case report which presented as ATYPICAL ECLAMPSIA with POSTERIOR (PROGRESSIVE) REVERSIBLE ENCEPHALOPATHY SYNDROME.

Keyword : Atypical eclampsia, Eclampsia, Preeclampsia, PRES, MRI Brain

INTRODUCTION

Eclampsia is defined as development of convulsions during pregnancy or within 48 hours postpartum in patients with signs and symptoms of preeclampsia. Atypical Eclampsia is a non classical form of hypertensive disorder, where seizures occurs in the absence of signs and symptoms of preeclampsia, Eclampsia occurring before 20 weeks of gestation, or after 48 hours postpartum in the presence of signs and symptoms of preeclampsia.

The incidence is 8%. Since the onset is unpredictable and there may be delay in the diagnosis, there is an increased rate of maternal and perinatal morbidity and mortality. PRES is one of the complications of severe preeclampsia, where cerebral autoregulation is lost leading on to cerebral hyperperfusion and vasogenic edema involving mainly the parieto occipital cortex.

CASE REPORT

20 years old primi gravid with LMP on 22/11/2011 and EDD on 29/08/2012, was admitted in the labour ward, a week before EDD with the complaints of labour pains for safe confinement. She was a booked case and her antenatal period was uneventful. As per her antenatal records, her BP was normal throughout pregnancy and there was no evidence of proteinuria. O/E Patient was afebrile, no pallor, no icterus, no pedal edema. Vitals: PR: 86/min, B.P: 110/80mm/Hg. CVS – S1S2+ , RS – NVBS + , CNS – No Focal neurological deficit. P/A Uterus was term, not acting, head unengaged, FH-148/min. P/V Cervix uneffaced, posteriorly placed, firm in consistency, Os closed, Head at -3 station felt through fornices, Pelvis gynaecoid. No show or draining PV. Her lab parameters were normal. Since she was not in labour, she was transferred to the antenatal ward. Next day early morning, she was brought to the labour ward with the complaints of blurring of vision. No history of trauma/head injury, no h/o fever, headache, vomiting, epigastric pain. O/E Patient was conscious, oriented, afebrile, no pallor, no pedal edema. PR: 86/min, BP: 180/110 mm/Hg, RR: 16/min.CVS: S1S2+, RS: NVBS, CNS: No neck stiffness, PERL, DTR- Normal, No Focal neurological deficit. P/A Uterus term, Not acting, Not tense, Not tender, Head unengaged, FH-146/min. P/V Cervix uneffaced, posteriorly placed, firm in consistency, Os closed, Head at -3 station felt through fornices, Pelvis gynaecoid, no show or draining PV. While examining, the patient developed generalised tonic clonic convulsions and the patient was stabilised with ABC's of resuscitation. Bladder catheterised, Mgso4 loading dose was given immediately and antihypertensives (IV Labetolol) given to control hypertension. Lab investigations were taken. After stabilising, the patient was taken up for Emergency LSCS as the Modified Bishop score was 0/12 and delivered a live male baby (birth weight-2.8 kg) with Apgar of 7/10. Post operatively, Mgso4 maintenance dose was continued by monitoring the urinary output, Respiratory rate and Knee jerk and anti hypertensives continued to control blood pressure. Neurophysician and ophthalmologist's opinion were obtained. Antiedema measure with 10% Mannitol 100ml twice daily was started as per Neurophysician opinion. CT and MRI Brain taken, whichrevealed PRES. Optic fundus was found to be normal. With antiedema measures, Mgso4 regimen and anti hypertensives she regained her vision after 12 hours, blood pressure was kept under control and patient became stable after 24 hours without any residual neurological deficit.

INVESTIGATIONS

Urine albumin-nil. Hb-11gm/dl, Platelet count-1.9 lakhs/cumm

Blood urea-24mg/dl, Sr creatinine- 0.9mg/dl Blood sugar-94mg/dl LDH - 503 mg/dl LFT: Sr Bilirubin- 0.8mg/dl, SGOT-35 IU/L, SGPT-38IU/L, total proteins-6.8g/dl, Sr Albumin-3.8g/dl Na+ 142meq/l K+ 3.1meq/l Coagulation profile was normal. CT scan - **Posterior reversible encephalopathy syndrome** MRI Brain T2 FLAIR– Evidence of vasogenic edema involving the parieto occipital type 3 pattern (PRES)

MRI Brain showing cerebral edema

DISCUSSION

In classical hypertensive disorders of pregnancy, the disease process usually starts with Hypertension, progressing to preeclampsia and eclampsia. In this case, patient was a booked case, even the day before the episode, her vitals and laboratory parameters were normal. She developed, imminent symptoms and eclampsia instantly, which was diagnosed and managed promptly with anticonvulsants (Mgso4 regimen), anti hypertensives (Labetolol) and anti edema (Mannitol) measures and the patient recovered without any neurological deficit. Hence early diagnosis and management of atypical eclampsia prevents maternal and perinatal morbidity and mortality.

PRES - Posterior (Progressive) reversible encephalopathy syndrome

Synonyms – Reversible posterior leukoencephalopathy syndrome, reversible posterior cerebral edema syndrome, reversible occipito parietal encephalopathy. PRES is a clinico radiological entity that was first described by Hinchey et al in 1996. Acute changes in blood pressure leads to failure of auto regulation of cerebral circulation mainly involving the posterior circulation and causes cerebral hyperperfusion and vasogenic edema. The preponderance of posterior circulation is because of poor sympathetic innervation. Clinical manifestations are impairment of consciousness (13%), seizure activity (92%), headache, nausea and vomiting (26%), visual abnormalities in spite of normal fundoscopic examination (26%), focal neurological deficit (3%). Severe manifestations are coma and status epilepticus. Frequency of PRES associated with acute hypertension is 67%. CT Brain – diagnostic, **MRI (FLAIR) - Both diagnostic and prognostic-** increases the ability to identify subtle areas of edema. Radiological patterns identified are holohemispheric watershed pattern (23%), superior frontal sulcus pattern (27%) and dominant parieto-occipital region (22%). Differential diagnosis: Posterior cerebral artery territory infarct, venous thrombosis, vasculitis, encephalitis and demyelinating disorders.

CONCLUSION:

To conclude, absence of Hypertension or proteinuria should not preclude diagnosing eclampsia. Eclampsia may present as atypical forms. The occurrence of PRES should also be kept in mind. Hence, the obstetricians should be aware of the atypical presentation of eclampsia and manage promptly to reduce the maternal and perinatal morbidity and mortality.

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