CASE REPORT

POST PARTUM CONVULSION: PRES OR ECLAMPSIA

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ABSTRACT

Introduction: Postpartum convulsion is still a challenging problem due to its numerous aetiologies. Posterior reversible encephalopathy syndrome (PRES) is one of them. It happens to be a diagnostic and therapeutic challenge when it founds in eclampsia and pre-eclampsia patients.

Case report: Here we are presenting a case report of PRES in a 20 year old primigravida with severe pre-eclampsia with 36 weeks of pregnancy, and sharing our experience in diagnosing and treatment of this rare entity.

Conclusion: Convulsion developing in post-partum period is not always eclampsia. Surgeons and physicians should be aware of this entity. With encouraging result of the presented case, we propose early clinicoradiological diagnosis with contrast MRI and prompt management in the form of timely control of blood pressure and seizures can prevent devastating complications and permanent neurologic damage.

Keywords: Eclampsia, MRI, PRES.

INTRODUCTION

Postpartum convulsion is a difficult entity to manage. Its various etiological factors, like eclampsia, hysteria, puerperal cerebral thrombosis, cerebral malaria, meningitis, encephalitis, intracranial tumour, epilepsy, Posterior reversible encephalopathy syndrome (PRES) etc. makes it more challenging(1). PRES is a new entity. Various cases of PRES developed during the peripartum period have been reported(1-9).

It happens to be a diagnostic and therapeutic challenge when it develops in eclampsia and pre-eclampsia patients (10). We are reporting one such case of PRES in a patient with severe pre-eclampsia.

CASE REPORT

A 20 years old prim gravida with 9 months of amenorrhoea, referred to Index medical college Indore (India), with chief complaints of headache, blurring of vision and epigastric pain. On examination, the patient had increased blood pressure (190/110 mmHg), anaemia (Hb-7 gm %) with pallor of moderate degree and grade II pedal oedema. Rest of the findings were within normal limits. Systemic examination reveals no abnormality in respiratory, cardiovascular and central nervous system. The per abdomen examination showed 36 weeks relaxed gravid uterus with singleton foetus in cephalic presentation in non-engaged position with foetal heart rate of 142/min. Per vaginal examination reveals closed and unaffected posterior placed cervix, with vertex presentation at -4 station, and adequate pelvis. Her investigations showed haemoglobin 7.8 gm%, platelet 2.89 lakh per cumm, urine albumin 3 plus and rest of the investigations (liver function test, renal function test, coagulation profile) were within normal range.

Management was started with stat dose of intravenous 20 mg Labetalol followed by 40 mg to control the blood pressure. As her blood pressure comes to 150/100 mmHg, Tablet of Labetalol 200mg TDS started for maintenance. Magnesium Sulphate by Pritchard method was given to prevent convulsion, she was induced with Dinoprostone gel and observed for progress of labour. Due to foetal distress and non-progress of labour Emergency LSCS was done which delivered 2.7 kg male child, with an APGAR score of 7 in one minute and 9 in 5 minutes. Intraoperative and postoperative period was uneventful. Postoperatively her blood pressure remained in the range of 140/90 to 150/100 mmHg, Tablet Labetalol 100 mg three times daily was stared with intramuscular Magnesium Sulphate till 24 hours after delivery. She received one unit whole blood in postoperative period in view of low haemoglobin. On 4th postoperative day, she started complaining of headache and blurring of vision and all of a sudden she develops generalized tonic clinic seizure. Her blood pressure was found to be 200/130 mmHg. Immediately she was shifted to ICU, where IV line access, oral airway insertion, oxygen inhalation and urinary catheterization done. Within 15 min duration she again had three episodes of generalized tonic clinic seizures at an interval of 4-5 min, each lasting for 1-2 minutes. Intravenous Midazolam and 600 mg of Phenytoin (loading dose) was given immediately, and maintained with 100 mg of phenytoin IV 8 hourly. Injection Kepra (Levetiracetam) 500mg 12 hrly, Tab Labetalol 200mg tds and Tab Depin were also started.
Once patient is relatively stabilized, MRI was done which shows focal areas of hyper intensity in left posterior parietal and occipital lobe, involving left basal ganglia and right caudate nucleus in T2 weighted image; highly suggestive of posterior reversible encephalopathy syndrome (PRES).

She remained in the confused state for next twelve hours, Next morning she was fully conscious and well oriented to time place and person. Her blood pressure comes in the range of 140/90 to 150/100 mmHg. Similar treatment continued for two more days. On 8th day, urinary catheter and abdominal stitches were removed and oral drugs regimen started as Tab Eptoin 300mg HS in tapering mode, Tab Kepra 500mg bid, Tab Labetalol 100mg tds and oral antibiotics, and on 10th day she was discharged. On follow up visit of 15th day she was all right, her BP was 120/80 mmHg, urine albumin nil and repeat MRI shows normal findings compare to previous one.

DISCUSSION
PRES is a clinicoradiological entity described by Hinchey et al (9). Clinical and radiological features include headache, encephalopathy, seizures, cortical visual changes and parieto-occipital white matter oedema visualized on imaging modalities (11).

The mechanism of development of PRES is not clearly understood but physiological accumulation of fluids during pregnancy may increase the endothelial permeability of cerebral vasculature which gets deteriorated further by increased blood pressure resulting in vasogenic oedema (12). This is reversible when cause is controlled, so control of blood pressure is of vital importance in PRES. Diagnosis of PRES is a challenge for any practitioner as its clinical presentation is nonspecific. Imaging modalities is crucial for diagnosis. Cerebral MRI (FLAIR MRI) has been considered the most appropriate tool in diagnosing PRES and demonstrating brain changes (4,9). Characteristic findings are bilateral symmetrical vasogenic oedema in territories of the posterior cerebral circulation, posterior parietal, temporal and occipital lobes (2, 4).

Most commonly there is vasogenic oedema within the occipital and parietal regions (~95% of cases), perhaps relating to the posterior cerebral artery supply. The oedema is usually symmetrical. Despite being termed posterior, PRES can be found in a non-posterior distribution, mainly in watershed areas, including within the frontal, inferior temporal, cerebellar and brainstem regions (13). Similar to our case, which is reversible and has only vasogenic oedema, which was confirmed by later normal MRI. However in some severe forms, the progressive dysfunction of the cerebrovascular regulation mechanisms can cause damage to the blood-brain barrier: in these cases, MRI with gadolinium injection shows enhancement on T1-weighted images (14). Equally, a cytotoxic oedema may appear, which would show a decreased diffusion coefficient, as in the case described by Benziada- Boudour et al (15).

18 cases of PRES have been reported and 3 cases out of these manifested as post-partum eclampsia and one case was of normotensive with no abnormality (7). There is a dictum that patient with postpartum convulsion should be diagnosed as eclampsia until proven otherwise. MRI of brain is usually normal in eclampsia patients. Only on radiological basis diagnosis of PRES can be made. But whatever is the diagnosis, control of blood pressure is of vital importance in management.

CONCLUSION
Convulsion developing in post-partum period is not always eclampsia. Surgeons and
physicians should be aware of this entity. With encouraging result of the presented case, we propose early clinico-radiological diagnosis with contrast MRI and prompt management in the form of timely control of blood pressure and seizures can prevent devastating complications and permanent neurologic damage. Moreover this case presentation generates a scope for multi-centric met analytical study for evaluation of other causes and development of an extensive management protocol for post-partum convulsion disorder.

REFERENCES