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POST PARTUM POSTERIOR REVERSABLE ENCEPHALOPATHY SYNDROME

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ABSTRACT

Posterior reversible encephalopathy syndrome (PRES) is an acute neurotoxic syndrome that is characterized by a spectrum neurological and radiological feature from various risk factors.Delay in the diagnosis and treatment may result in death or in irreversible neurological sequelae. Common neurological symptoms includes headache, impairment in level of consciousness, seizures, visual disturbances, and focal neurological deficits. Common triggering factors include blood pressure fluctuations, renal failure, eclampsia, exposure to immunosuppressive or cytotoxic agents and autoimmune disorders.The classic radiographic findings include bilateral subcortical vasogenic edema predominantly affecting the parieto-occipital regions but atypical features include involvement of other regions, cortical involvement, restricted diffusion, hemorrhage, contrast enhancement. It is the first case of PRES without underlying disease. We report a case of postpartum eclampsia presented 8 days after delivery, which is the latest onset ever described. Wesuggest The presence of prodromal symptoms should be thoroughly investigated, even in the absence of antecedent pre-eclampsia

Key words - postpartum hemorrhage, intracerebral hemorrhage, Posterior reversible encephalopathy syndrome

Introduction

Posterior reversible encephalopathy syndrome (PRES) is a well-recognized entity characterized by a combination of clinical and neuroimaging findings. We report a case of postpartum eclampsia presenting 8 days after delivery, which is the latest onset ever

described. Earliest onset in 48 hours is most common [1]. , early treatment initiation with magnesium sulphate and antihypertensive medication prevents severe complications and reduces mortality. . It is caused by a wide variety of causes ultimately leading to a vasogenic cerebral oedema of occipital and parietal lobes of the brain.Common triggering factors of PRES include blood pressure fluctuations, preeclampsia/eclampsia, renal failure, cytotoxic agents, and autoimmune conditions We present here a young woman with headache, generalised tonic–clonic seizures and visual disturbances in a late postpartum stage.

Case Report

A 28 year old P2L2A1, post-operative case of Lscs P2L2A1 was referred on POD 8 withC/o headache, blurring of vision ,giddiness since 2 days C/o seizures one episode in the morning at home. The patient developed one episode of witnessed generalized tonic–clonic seizure in the casualty. After stabilizing the patient shifted to ICU, while the patient was regaining consciousness, she started to report headache and a bilateral loss of vision. She **was** found to have an elevated BP of 180/100 mmHg; rest of the vital signs were within normal limits. On per abdomen examination uterus involuting well, wound was healthy, on L/E Lochia was healthy. An ocular examination revealed a diminution of vision of bilateral eyes to perception of hand movement. Rest of the cranial nerve examination was unremarkable. Her antenatal period was uneventful. She had no history of raised BP, headache or blurring of vision, seizures during pregnancy. Patient was diagnosed with GDM and was managed medically during pregnancy. Patient was taken up for emergency LSCS I/V/O failed induction. Intra operative and immediate post-partum period was uneventful. Patient had no history of chronic hypertension, PIH, epilepsy.

Laboratory findings were significant for an elevated white cell count of 17000, haemoglobin 10.8 gm%, Prothrombin time was Elevated, APTT, INR - With in normal limits. Urine analysis was remarkable for 3+protein.Serum electrolytes, LDH & CRP, Renal function test,

and Liver function test were within normal limits. T2-weighted and fluid attenuated inversion recovery images of brain MRI showed hyperintensities in bilateral high frontoparietal lobes and occipital lobes and also in bilateral cerebellum hemispheres which were suggestive of posterior reversible encephalopathy syndrome.

No evidence of dural venous sinus thrombosis

No acute infarct,no intracranial bleed or space occupying lesions

No features of raised intra cranial pressure

MRV shows no significant abnormality

Treatment

The patient was shifted to the intensive care unit. Patient was stabilized and managed with anti convulsants and anti hypertensives20% Magnesium sulfate was started with zuspan regimen and Lorazepam 3mg Iv stat dose Tablet Labetalol 100mg stat dose and twice a day was given

DISCUSSION

Eclampsia is a poorly understood multisystem complication of pregnancy that substantially contributes to maternal morbidity and mortality. The typical clinical picture is of generalised tonic clonic seizures during the third trimester, labour, or early puerperium in women who already have hypertension, proteinuria, and oedema. The previously controversial existence of a delayed postpartum variant of eclampsia is now acknowledged by most experts. Convulsions with initial presentation more than 48 hours but less than four weeks after delivery are commonly referred to as late postpartum eclampsia [4]. This patient presented with convulsions and elevated BP on the postpartum day 8 the magnetic resonance scan showing hyperintense posterior cerebral encephalopathy. Postpartum eclampsia can present with a variety of clinical and neurological symptoms and signs.Lubrasky6 and Chames5 reported that

44% and 79% of their respective patients with late onset postpartum eclampsia had not been identified as having pre-eclampsia before seizure onset [5]. They reported that severe and persistent headache, visual symptoms, epigastric or right upper quadrant pain, and hypertension can present as prodromal symptoms before the onset of eclampsia.4–6 . Patient presented with same symptoms .Eclampsia should be considered in any postpartum woman who develops with any of these prodromal symptoms. Further indicators include convulsions up to four weeks after delivery, hypertension, or proteinuria. This is important as eclampsia is amenable to treat with magnesium sulphate. The differential diagnosis includes epilepsy, cerebral venous thrombosis, intracerebralhaemorrhage, phaeochromocytoma, space occupying lesions, and metabolic disorders.Neuroimaging, especially magnetic resonance imaging, shows micro-ischaemic injury patterns in parieto-occipital lobes.10 This form of posterior leukoencephalopathy syndrome can cause cortical blindness, which may be reversible with control of hypertension and magnesium sulphate therapy. Lesions ona magnetic resonance scan cannot predict whether damage leading to cortical blindness is permanent or likely to be reversible [6].

Magnesium sulphate remains the drug of choice for preventing and treating eclamptic seizures. If eclampsia is not treated at appropriate time, grave complications such as intracerebralhaemorrhage and death can occur. Late onset postpartum eclampsia can occur in normotensive uncomplicated postpartum women as well as in women with pre-eclampsia [7]. The presence of prodromal symptoms should be thoroughly investigated, even in the absence of antecedent pre-eclampsia. We suggest that such patients seen in accident and emergency units within 14 days of delivery should be assessed by an experienced obstetrician.

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