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Case Report

Posterior reversible encephalopathy syndrome (PRES) in a 29 weeks pregnancy: A case report

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Abstract

Posterior Reversible Encephalopathy Syndrome also known as PRES is combination of acute neurologic clinical manifestation including alteration in consciousness, seizure, headache, visual abnormalities, nausea/vomiting and focal neurological signs associated with neuroimaging findings consistent with PRES. Focal neurological signs consist of symptoms or signs due to damage or the dysfunction of a specific anatomic site in the central nervous system. PRES, has diverse etiology, common precipitants are considered acute elevations of blood pressure, renal decompensation, fluid retention, and treatment with immunosuppressive drugs. Its clinical presentation has wide variation. Its reversibility depends on the timing of diagnosis and management. In this case report we present a case of a 37 year old primi gravida with 29 weeks 3 days gestation who suffered with severe pre eclampsia rapidly progressing to eclampsia and further complicated with PRES in peripartum period, she was well managed with quick diagnosis and management.

Keywords: Posterior Reversible Encephalopathy Syndrome, pre eclampsia, eclampsia, peripartum period.

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1. Introduction

In 1996, Hinchey et al first described Posterior reversible encephalopathy syndrome (PRES) or reversible posterior leukoencephalopathy syndrome (RPLS) [1]. It can be related to multiple conditions like high blood pressure, immunosuppressed state, autoimmune disorders, thrombotic thrombocytopenic purpura, HIV syndrome, acute intermittent porphyria, blood transfusion and electrolyte disturbances [2,3]. Clinically, it presents as an acute neurological condition with seizures (74% of patients cases) both focal and generalized, and even status epilepticus, encephalopathy, visual symptoms (blurred vision, hemianopia, or cortical blindness) and headache. Focal deficits are rare, except in which are due to hypertensive urgency, hypertension arterial, and if present, it is moderate or normal in 20% of cases.[4] On CT or MR imaging studies, the cortical and subcortical edema is often widespread but predominates in the parietal and occipital regions, likely leading to suggest the posterior cerebral involvement [5.6]. If promptly diagnosed and properly treated, PRES can be reversed. However, if the diagnosis is delayed or incorrect diagnosis is made it may lead to irreversible damage [7].

2. Case Report

A 37 year old primi gravida with 29 weeks 3 days gestation was hospitalized with history of pregnancy induced hypertension diagnosed 3 days back. Patient had history of PCOD diagnosed 15years back, hypothyroidism diagnosed during this pregnancy. She had no significant family history. She had conceived this pregnancy through *in vitro* fertilization,

On admission physical examination revealed that she was fully alert and oriented. She had a temperature of 38.1°C, blood pressure 130/90 mm Hg, pulse rate 90/min and respiratory rate 16/min. Respiratory and cardiovascular examination was within normal limits. Abdominal examination, uterus was at 28-30 weeks, relaxed and fetal heart sounds were present.

On the 3rd day of hospitalization, she had severe abdominal pain and her blood pressure increased to 210/110mmhg which did not respond much to medications, she was shifted to intensive care unit.

On evaluation her liver functions were found to be raised, her platelet counts had dropped, and, it was considered to develop HELLP syndrome and decision was taken to perform LSCS under coverage of platelet transfusion on 4th day of hospitalization.

Before shifting to LSCS she developed GTCS, and she was urgently shifted to OT and caesarean was performed under GA, a preterm female baby of 835gm was delivered and shifted to NICU, patient was extubated on table and shifted back to intensive care unit.

Again in ICU, patient had 2 episodes of seizures which were managed with antiepileptics. Shortly after resolution of seizures, while the patient was regaining consciousness, she started to report bilateral diminution of vision. She was found to have an elevated BP of 180/100 mm Hg; rests of the vital signs were within normal limits. Ocular examination revealed diminution of vision of bilateral eyes. Pupils were normally reactive to light and fundus examination was unremarkable. Rest of the cranial nerve examination was unremarkable. Power was 5/5 across all major joints and sensory function was intact all over the body. Cerebellar signs were intact and there was no evidence of meningeal signs such as nuchal rigidity or Kernig's/Brudzinski's sign. Plantars were down going bilaterally.

3. Investigations

Laboratory findings were significant for an elevated white cell count of 17 000, haemoglobin 13, platelets 49000. Urinalysis was remarkable for more than 3gm protein. LFTs were raised with SGOT 892 and SGPT 645.serum creatinine also increased to 1.2. PT, PTT, INR were within normal limits. T2-weighted and fluid-attenuated inversion recovery images of brain MRI showed bilateral posterior parietooccipital hyper densities in the cortex and subcortical white matter consistent with posterior reversible leukoencephalopathy syndrome (Figure 1, Figure 2)

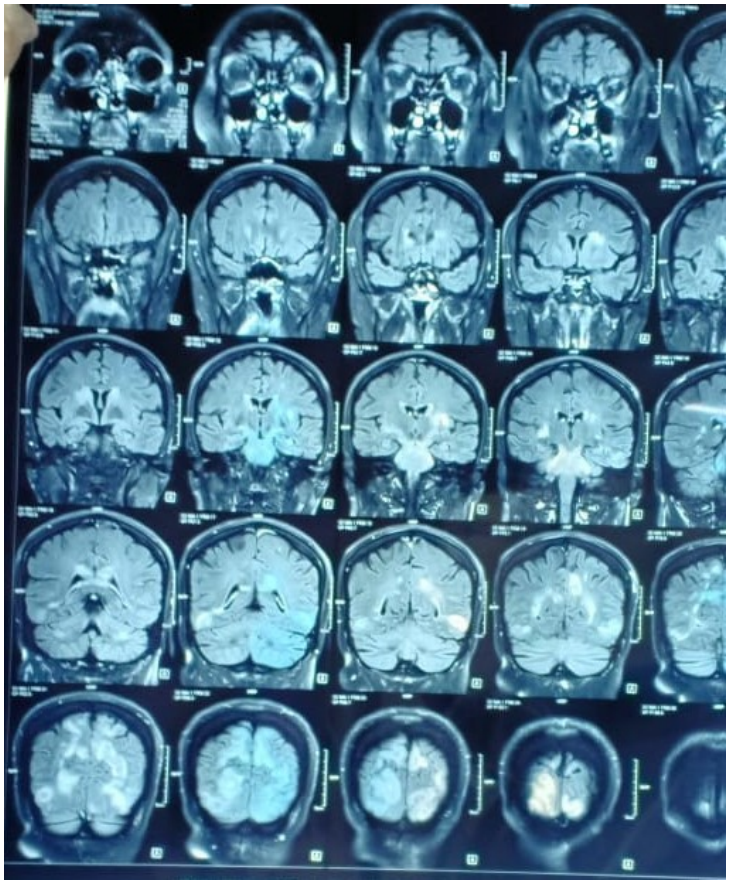

Figure 1: Images of brain MRI

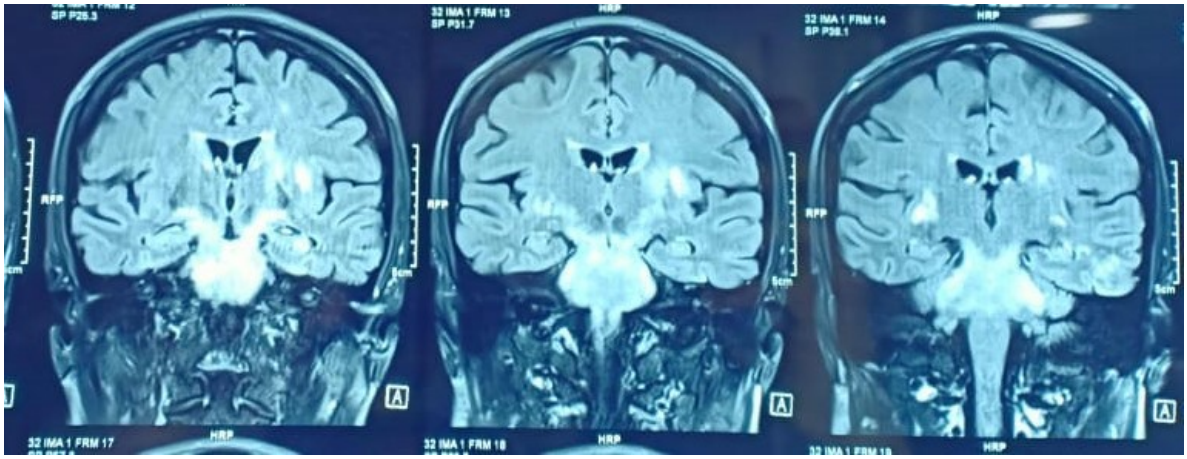

Figure 2: Images of brain MRI

4. Treatment

First with the impression of HELLP syndrome, the patient was transferred to the intensive care unit. Labetalol infusion was started at a rate of 1 mg/min with close monitoring of blood pressure. Meanwhile Magnesium sulphate was given in a loading dose of 6 g intravenously over 15 min followed by a maintenance dose of 2 g/h with monitoring of respiratory rate, urinary output and patellar reflex. After delivery of baby, to manage seizures, injection phenytoin and levitrecetam was administered, patient's blood pressure slowly came down and later antihypertensives were not needed to be given. Her urine output improved. Her serum creatinine, platelet count and LFTS gradually came back to normal over 2 days after delivery. She was continuously monitored for haemodynamic stability. After MRI result consistent with PRES, magnesium and phenytoin were discontinued.

5. Recovery and follow up

The patient's vision improved after about 6 h of the onset of symptoms. She was observed in the intensive care unit for 4 days. She continued to be symptom free and was later transferred to a regular floor. The patient continued to improve clinically and later on discharged.

6. Discussion

Eclampsia and pre-eclampsia are found to be commonly in association with PRES during pregnancy and the postpartum period. Pre-eclampsia can get complicated by HELLP syndrome and in about one-third of cases it occurs in the immediate post-partum period. Early diagnosis and appropriate treatment can reduce incidence of poor clinical outcomes [8]. Frequently the most common symptoms are usually generalized, tonic–clonic seizures and often precede other manifestations [9]. Visual abnormalities due to the involvement of the occipital lobe range from cortical blindness, homonymous hemianopia, blurred vision, visual neglect and visual hallucinations [10]. Encephalopathy caused confusion, lethargic behaviour with slowed motor responses or deep stupor [11]

In this case report, we set out a case of pregnancy induced hypertension whose clinical features were abdominal pain, diminution of vision in both eyes and seizures. She rapidly progressed from preeclampsia to severe preeclampsia to eclampsia which further got complicated by clinical and neuro radiological findings consistent with PRES.

The pathophysiology of PRES is still a controversial concept. There are two hypotheses regarding the same. Normally cerebral autoregulation with the help of neurologic and myogenic mechanism maintain a constant perfusion to the brain.[12] The older hypothesis states that hypertension leads to autoregulatory vasoconstriction which further leads to hypoperfusion resulting in ischemia and cerebral edema [5]. While according to the newer hypothesis sudden increase in blood pressure overcomes the autoregulatory capability of brain vasculature leading to abrupt dilatation of cerebral vasculature with resultant hypoperfusion. It further leads to breakdown of blood brain barrier with focal transudation of fluid into the interstitium and petechial hemorrhage, which is detected as vasogenic edema which predominates in white matter of parieto-occipital region [1]. Though on neuroimaging of PRES, most common abnormality is edema in white matter of parieto-occipital region. In a recent study, involving 136 patients, 92% had parieto-occiptal regions involved but involvement of other brain regions such as temporal lobe, brain stem, cerebellum, basal ganglia and frontal region has also been reported [13,14]. Hence awareness of the variation helps in recognising PRES. In the present case, MRI showed bilateral posterior parieto-occipital hyper densities in the cortex and subcortical white matter consistent with posterior reversible leukoencephalopathy syndrome. (Figure 1, Figure 2)

Signs and symptoms of PRES depend not only on area of brain involved but also on extent or severity of brain involvement.

Cases of PRES in peripartum period with different symptomatology have been reported in literature. Strianoetal [15] reported two patients with PRES in postpartum period who had eclampsia and chronic epilepsy which developed as a sequel.[15] In another paper, two patients were described who experience PRES in late postpartum period without classic pre eclamptic sign but with impairment of consciousness and epileptic seizures.[16] PRES should be considered in differential diagnosis of post partum seizures.[17]. In our case, headache and seizures occurred associated with cortical blindness which later on got reversed with prompt treatment. There was no limb weakness. Therefore the case is important as it illustrates atypical combination of symptoms of PRES.

Many authors support that hypertensive encephalopathy and eclampsia share similar pathophysiologic mechanisms [18-20]. The imaging findings and clinical features of post partum eclampsia are identical to those of hypertensive encephalopathy. The pathologic process is also characterised by cerebral edema and petechial haemorrhages, especially in the parieto-occipital and occipital lobes. The spectrum of cerebral lesion in eclampsia seen in MRI varies from irreversible areas of vasogenic edema to those that may progress to cytotoxic edema and infraction in up to one fourth of females. In the present case symptoms heralded as headache progressing to seizures later on progressing swiftly to reversible cortical blindness.

The widespread use of MRI technology has made PRES familiar to many clinicians. In this case diagnosis was made immediately and treatment was initiated without delay. On examination, one month after delivery, no sequelae remained.

7. Conclusion

In conclusion, this paper reports a severe pre eclampsia case complicated with PRES in peripartum period of a pre eclamptic female in whom appropriate control of blood pressure and quick management of sudden increase in blood pressure led to complete clinical recovery.

The clinician should be acquainted with PRES in the peripartum period of a pre eclamptic female with neurological manifestation, as immediate reversal will avoid perpetual brain injury with absolute resolution of clinical and imaging findings.

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