Research Article

Late Postpartum Eclampsia with Posterior Reversible Encephalopathy Syndrome

Smita S Aundhekar (Patne)[1], Roohi Shaikh[2*]

[1]Associate professor, OBGY Department, L.N. Medical College and Research Centre, Bhopal, Madhya Pradesh, INDIA.
[2]Assistant Professor, OBGY Department, IIMSR, Warudi, Badnapur, Jalna, Maharashtra, INDIA.
Email: psmita390@gmail.com, dr.roohi.shaikh@gmail.com

Abstract

Posterior reversible encephalopathy syndrome (PRES) is a recently described clinicoradiologic entity that is associated with several medical conditions like hypertensive encephalopathy and eclampsia. It presents with rapid onset of symptoms including headache, seizures, altered consciousness and visual disturbances. We present a case of 35 years old primigravida with unremarkable antenatal period, who developed convulsions in the postpartum period following cesarean delivery. The magnetic resonance imaging brain revealed vasogenic edema suggestive of PRES. She was managed with supportive treatment in intensive care unit. She recovered completely without neurological sequelae and discharged on 8th post-operative day. This case report highlights the importance of awareness, prompt diagnosis and treatment to improve the outcome in this potentially life-threatening but reversible condition.

Keywords: Posterior reversible encephalopathy syndrome, postpartum seizures, eclampsia, caesarean section, vasogenic oedema.

[*]**Address for Correspondence:**
Dr. Roohi Shaikh, Labour Colony, Aurangabad, Maharashtra, INDIA.
Email: dr.roohi.shaikh@gmail.com
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INTRODUCTION

Posterior reversible encephalopathy syndrome(PRES) is a rare clinico-neuroradiological syndrome associated with various clinical conditions, presenting with headache, confusion, seizures, cortical visual disturbances or blindness and parieto-occipital white matter edema visualized on neuroimaging modalities. PRES is also known as Reversible Posterior Leukoencephalopathy Syndrome [RPLS] or reversible posterior cerebral oedema syndrome, hyperperfusion encephalopathy or brain capillary leak syndrome. PRES has been accompanied by a number of medical conditions such as hypertensive encephalopathy, pre-eclampsia, eclampsia, acute or chronic renal diseases, hemolytic uremic syndrome, use of cytotoxic and immunosuppressive drugs, blood transfusion and electrolyte disturbances. Pre-eclampsia and eclampsia are among the most common causes of PRES. Prompt recognition and treatment are crucial to avoid the permanent damage leading to sequelae and even mortality.

CASE REPORT

A 35 year old primigravida with 38 weeks of gestation was admitted with labour pains. She had no complaints of headache or visual abnormalities. Her blood pressure was 130/80 mm of Hg with pulse rate of 108/minute. There was no past history of hypertension, vision abnormalities, seizures or any other pathologies. Complete blood count revealed a hemoglobin level of 7 g/dl and platelet count of 1,62,000/mm^3. Urinalysis revealed no proteinuria. *(Renal and hepatic function tests as well as electrocardiogram was within normal limits). Emergency caesarean section was done for fetal distress with cephalopelvic disproportion. The intraoperative period was uneventful. She received 3 units of blood transfusion in the postoperative period. On the 5th post-operative day, she complained of severe headache, followed by one episode of tonic-clonic convulsion and the BP recorded at that moment was high (150/100 mm of Hg). She was promptly treated with magnesium sulphate 4g in 100 ml

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saline infused over 15 minutes. In the postictal phase, her pulse rate was 114 beats/min, BP 150/90 mm of Hg and oxygen saturation of 100% with oxygen supplement. A neurological examination carried out after the convulsion episode was essentially normal without any sensory or motor deficit and fundoscopy was normal. Routine investigation showed hemoglobin of 9 g/dl, total leucocyte count of 17.3 x 10^3/ml, platelets of 120 x 10^3 /ml, normal renal and liver function tests and clotting parameters. She was subsequently shifted to intensive care unit (ICU) for further management. In the ICU, her treatment consisted of Cap Depin 10mg tds with continuous BP monitoring and Injection Magnesium Sulphate 2gm intramuscular every 3 hourly for 24 hours. Her magnetic resonance imaging (MRI) on day 6 revealed bilateral hyperintensity in the fronto-parietal, occipital cortical and subcortical white matter. These findings were consistent with PRES. Sodium phenytoin 100 mg iv tds was started. She was closely monitored with all the necessary ICU protocols including periodic neurological assessment and biochemical profile. She had an uneventful hospital stay and discharged on day 10 without any neurological sequelae. A follow-up MRI after 4 weeks showed complete resolution of the high signal foci previously noted.

DISCUSSION

Posterior reversible encephalopathy syndrome is a clinicoradiological entity described by Hinchey et.al. in 1996. It presents with neurologic signs and symptoms such as headache, altered consciousness, seizures, visual loss and often associated with abrupt increase in blood pressure. The typical MRI findings in T_2-weighted and FLAIR sequences show hyperintense foci located bilaterally at the grey-white junctions, involving subcortical white matter of most parts of brain although it was earlier believed to affect the posterior parietal and occipital lobes mainly. Global incidence of PRES is unknown. It has been reported in patients aged 4-90 years, although the most cases occur in age group of 39-47 years with a marked female predominance. Mechanical ventilation is required in 35-40% of patients with PRES for 3-7 days. Recurrence are reported to be occurring in 6% cases. Permanent neurologic damage is due to ischemia (10-23%) and hemorrhage (5-17%). Death is reported in 15% cases. The pathophysiology of PRES remains controversial suggesting two main hypothesis. Uncontrolled hypertension with failed autoregulation leads to hyperperfusion and cerebral vessel damage resulting in interstitial extravasation of protein and fluid causing vasogenic edema remains a popular consideration. The other theory alludes to systemic inflammatory state causing endothelial dysfunction leading to cerebral hypoperfusion which is then responsible for the disruption of the blood-brain barrier resulting in vasogenic edema. Appropriate reduction of BP may prevent progression from vasogenic to cytotoxic edema resulting in cerebral infarction and permanent neurologic deficit. The association of PRES with toxemia of pregnancy is well-established. PRES can occur within several weeks after delivery and the clinical presentation is often confusing. Our patient delivered by emergency cesarean section was completely healthy and symptom-free during antenatal period. No clinical trials have evaluated the management of PRES, but rapid withdrawal of the trigger appears to hasten recovery and avoid complications. For example, aggressive BP management, withdrawal of offending drug or delivery in eclampsia. Antiepileptic drugs should be used to treat seizures and ventilatory support should be instituted in generalized status epilepticus and to protect the airway in obtunded patients. In our patient, the seizure episode was managed effectively with magnesium sulfate and phenytoin and BP was controlled with antihypertensive drug. Prompt diagnosis and multidisciplinary management resulted in favourable outcome.

CONCLUSION

A clinical report of parturient with PRES after delivery is presented. It emphasizes the need for early diagnosis and prompt treatment to avoid any short and long term neurological sequelae including death in a reversible condition like PRES.

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