

Original article

Posterior Reversible Encephalopathy Syndrome, postoperatively in an Emergency Caesarean section

Dr Deepika Shukla, Dr Aartee Potnis , Dr Ezhil Bharthi S , Dr Sai Durga krishna Kiran K

Name of the Institute/college: Dept of Anaesthesia , D Y Patil University School of Medicine, Navi Mumbai

Corresponding author : Dr Aartee Potnis

Abstract:

Posterior reversible encephalopathy syndrome(PRES) or reversible posterior leukoencephalopathy syndrome(RPLS) is a rare clinical and neuroradiological entity. A 22 year old Primigravida with 40 weeks gestation was taken up for emergency caesarean section for fetal distress due to non-progress of labour. She had fever and wet cough with past history of febrile convulsions, for which she took medications till 6 yrs of age. On general examination, pulse rate 100/min, BP 140/90mmHg and bilateral pedal oedema was present. Systemic examination was normal. Investigations showed Hb of 10.8 gm/dl with low Platelet count of 76,000/uL. Dengue and Malarial Antigen Test were Negative. Urine test, Liver function tests and PT/INR were sent. Posterior reversible encephalopathy syndrome (PRES) presents with a variety of neurological features which, although devastating, are potentially reversible on prompt recognition and institution of appropriate treatment, but clinicians often fail to suspect it. Delayed diagnosis is frequent and can lead to long-term neurological disability.

Keywords : Posterior reversible encephalopathy syndrome

Introduction :

Posterior reversible encephalopathy syndrome (PRES) or reversible posterior leukoencephalopathy syndrome(RPLS) is a rare clinical and neuroradiological entity first described in 1996 by Hinchey et al(1). This is characterized by headache, vomiting, confusion, seizures, visual abnormalities, motor signs and parieto-occipital white matter changes on neuroimaging(2). Importantly, these changes appear to be completely reversible if the underlying cause is treated early in the clinical course. Common clinical associations for PRES include hypertensive encephalopathy, renal failure, autoimmune disorders and treatment with

immunosuppressant or cytotoxic medications(3). We report a case of reversible encephalopathy syndrome, occurring in the postpartum period, managed successfully.

Case summary :

A 22 year old Primigravida with 40 weeks gestation was taken up for emergency caesarean section for fetal distress due to non-progress of labour. She had fever and wet cough with past history of febrile convulsions, for which she took medications till 6 yrs of age. On general examination, pulse rate 100/min, BP 140/90mmHg and bilateral pedal oedema was present. Systemic examination was normal. Investigations showed Hb of 10.8 gm/dl with

low Platelet count of 76,000/uL. Dengue and Malarial Antigen Test were Negative. Urine test, Liver function tests and PT/INR were sent.

In view of low platelet counts and pending blood reports, General anaesthesia with endotracheal intubation was done using rapid sequence induction technique with Thiopentone and succinylcholine. Patient was maintained on air: O₂ with Isoflurane and Inj.Atracurium. A female baby was extracted, with APGAR of 8/10 at 1min and 10/10 at 5min. Analgesia was achieved with fentanyl 100mcg. Intraoperatively, vitals showed tachycardia upto 100/min along with increased BP as high as 160/95 mmHg. Isoflorane increased and propofol infusion started @50mcg/kg/min to deepen the anaesthesia plane. Edema developed over the eyelids. Urine output was 75ml over one hour with 700ml estimated blood loss. Rest intraoperative period was uneventful. Inj.Hydrocortisone 100 mg and Inj.Dexamethasone 8mg i.v was given half hour before extubation.

Before reversal, oral suctioning under direct laryngoscopy revealed active bleeding from right posterior tonsillar pillar, trauma during laryngoscopy suspected. Throat packing done, bleeding stopped. The patient was reversed with myopyrollate after confirming eye opening, head lift and hand grip. Immediately after extubation, patient went into larygospasm with drop in SpO₂ to 90% and no air entry on auscultation. Inj.Thiopentone 200mg i.v. and Inj.Succinylscholine 75 mg i.v. were given. Direct laryngoscopy revealed airway edema, 6.0mm cuffed ETT inserted and patient shifted to ICU for elective ventilation under

hydrocortisone, antibiotic and nebulization cover to relieve edema.

In ICU, she was maintained on Atracurium and Midazolam infusion. Her B.P was 170/100 with HR of 90/min. Inj. Labetalol started. All Investigations were sent. 6hours postpartum, patient had one episode of convulsion for which Inj.Phenytoin and Inj.Mannitol were started. Urine showed proteinuria. Liver and renal function tests, coagulation profile were normal. CT scan findings showed signs of PRES syndrome. She was extubated as her sensorium, vital and ventilatory parameters improved on the third postoperative day. Rest of her hospital stay was uneventful till she was discharged.

Discussion:

The reported incidence of PRES is around 0.01%. At our teaching hospital, we have about 7000 deliveries per year. Most cases of PRES are associated with hypertensive disorders, particularly those occurring during pregnancy. In most obstetrics cases found in the literature, PRES usually develops only after delivery, as has been reported in our case. The onset is usually subacute but may be heralded by a seizure. Seizures are common at the onset of neurologic symptoms but can also develop later. The main point is that early accurate diagnosis and treatment of PRES may prevent brain damage, hemorrhage and infarction.^[7] Early neuroimaging is therefore indicated. Multiple theories have been proposed on the pathophysiology of PRES, the most accepted being the vasogenic oedema. Cerebral autoregulation maintains a constant blood flow to the brain despite alterations in the systemic pressures. Once this mechanism gets disrupted,

increased perfusion pressure is sufficient to overcome the blood–brain barrier, allowing extravasation of fluid, macromolecules and even red blood cells. So, PRES represents vasogenic rather than cytotoxic oedema in the majority of cases.

Clinical improvement always follows the treatment of elevated blood pressure and withdrawal of offending agents. Magnesium therapy should be initiated as soon as eclampsia or PRES in pregnancy is suspected, as it treats both seizures and hypertension.. Even mild fluctuations in blood pressure during or after anaesthesia, or changes in serum electrolytes, notably magnesium, may be sufficient to precipitate PRES in susceptible patients.[8] Early treatment usually results in complete reversal of the deficits over few days to several weeks.

Neuroimaging CT shows oedema as bilateral symmetrical hypodensities involving the white matter typically in the parieto-occipital regions. This is explained by better autoregulation of the anterior circulation due to better sympathetic innervations as compared to the posterior

circulation. MRI shows high signal intensity on T₂-weighted and fluid attenuated inversion recovery (FLAIR) sequences. FLAIR provides T₂-weighted images while suppressing the signal from CSF by use of an inversion pulse. FLAIR frequently allows detection of what otherwise would be subtle findings on conventional spin-echo T₂-weighted images. Reversibility of PRES may be clinically or radiologically incomplete, the condition may be complicated by ischaemic or haemorrhagic stroke, and may lead to a chronic seizure disorder or death. In this case postoperative CT helped us to diagnose PRES. Follow-up neuroimaging was not considered in view of rapid clinical recovery of the patient.

Conclusion:

Posterior reversible encephalopathy syndrome (PRES) presents with a variety of neurological features which, although devastating, are potentially reversible on prompt recognition and institution of appropriate treatment, but clinicians often fail to suspect it. Delayed diagnosis is frequent and can lead to long-term neurological disability.

Références

1. Hinchey J, Chaves C, Appignani B, Breen J, Pao L, Wang A, et al. A reversible posteriorleukoencephalopathy syndrome. *Engl J Med.* 1996;334:494–500. [PubMed]
2. Bartynski WS, Boardman JF. Distinct imaging patterns and lesion distribution in posterior reversible encephalopathy syndrome. *AJNR Am J Neuroradiol.* 2007;28:1320–7. [PubMed]
3. Long TR, Hein BD, Brown MJ, Rydberg CH, Wass CT. Posterior reversible encephalopathy syndrome during pregnancy: Seizures in a previously healthy parturient. *J Clin Anesth* 2007;19:145-8. †
4. Casey SO, Sampaio RC, Michel E, Truwit CL. Posterior reversible encephalopathy syndrome: Utility of fluid-attenuated inversion recovery MR imaging in the detection of cortical and subcortical lesions. *Am J Neuroradiol* 2000;21:1199-206.