Posterior Reversible Encephalopathy Syndrome (PRES) and Low Serum Magnesium in Cesarean Delivery: Cause or Coincidence?

Sonam Patel, Priyanka Dwivedi*, Ankita Kabi, Vijeta Bajpai and Gaurav K Chowdhary

Department of Anesthesiology, Pain Medicine and Critical Care, All India Institute of Medical Sciences, Gorakhpur, India

Received: 14 March 2024

Accepted: 6 May 2024

*Corresponding author: drpriyad1111@gmail.com

DOI 10.5001/omj.2027.01

Abstract

Posterior reversible encephalopathy syndrome (PRES) is a transient neurological condition that typically occurs during pregnancies complicated with hypertensive disorders such as preeclampsia or eclampsia. It is characterized by nonspecific symptoms, including seizures, headaches, altered consciousness, and focal neurological deficits. While preeclampsia and eclampsia are common causes of PRES, other causes include hypertensive encephalopathy, renal failure, immunosuppressant therapy, thrombotic thrombocytopenic purpura, systemic lupus erythematosus (SLE), and acute intermittent porphyria. We present an unusual case involving a 29-year-old parturient at 37 weeks of gestation who experienced sudden headache, irritability, tachypnea, and seizures during caesarean delivery and the early postpartum stage. Brain magnetic resonance imaging (MRI) revealed subtle flair hyperintensity in the posterior occipital region and left cerebellar hemispheres with adjacent sulcal effacement, indicative of PRES. The patient responded to magnesium sulfate therapy and was discharged after five days of post-delivery hospitalization.

Keywords: Posterior Reversible Encephalopathy Syndrome; Magnesium; Pregnancy; Vasogenic Edema; India

Introduction

Posterior reversible encephalopathy syndrome (PRES), also known as reversible posterior leukoencephalopathy syndrome (RPLS), is a distinctive cerebrovascular disease characterized by a set of clinical symptoms and radiological features. Pathophysiological mechanisms underlying PRES are still incompletely understood.

Magnetic Resonance Imaging (MRI) is the gold standard for diagnosing PRES. The MRI typically exhibits vasogenic edema in subcortical white matter, accompanied by cytotoxic oedema in certain cases. PRES is frequently associated with several medical conditions, such as pre-eclampsia, eclampsia, hypertension, post-transplant immunosuppression, cancer chemotherapy, and various autoimmune disorders.[1,2] Lesions attributed to PRES primarily manifest in parietal and occipital lobes (up to 50% of cases), superior frontal sulcus (up to 27%), and both anterior and posterior watershed zones (up to 29%). Less frequently reported are involvement of deep white matter, basal ganglia, thalami, brainstem, and pons (up to 13%).[3]

Clinical presentations of PRES include neurological disturbances such as impaired consciousness, headaches, seizure episodes, focal neurological signs, and gastrointestinal symptoms such as nausea and vomiting. Notably, PRES is generally reversible upon removal of the underlying cause.

Recent reports have associated hypomagnesemia with the acute phase of PRES, independent of its etiology.[4] Magnesium, an essential trace element, helps stabilize blood pressure and exhibits neuroprotective effects by reducing inflammation and maintaining the integrity of blood-brain barrier. Magnesium sulfate, a conventional treatment for conditions such as pre-eclampsia and eclampsia, highlights its relevance in neuroprotection. Here,

we present a unique case of clinically and radiologically diagnosed PRES, notable for its occurrence without a prior history of hypertension, preeclampsia, eclampsia, or other common triggers of PRES.

Case Report

A 29-year-old woman, gravida 2, para 1, with one previous lower segment caesarean section (LSCS), presented for an emergency LSCS due to non-reassuring fetal status. She had no known comorbidities. Her preoperative lab results and baseline vitals were within normal limits—heart rate: 92 beats/min; blood pressure: 110/78 mmHg, respiratory Rate: 18 breaths per minute, oxygen saturation (SPO_2): 100% at room air.

A subarachnoid block (SAB) was administered using a 25G Quincke's needle using 0.5% heavy bupivacaine (2 ml) at L3-L4 level. The surgery was started after an adequate level of anesthesia (T6) was achieved. Ten minutes into the surgery, the patient developed hypotension (90/50 mmHg) which was managed with intravenous (IV) fluid and a single bolus dose of 6 mg IV mephentermine. Another episode of hypotension (86/50 mmHg) occurred after ten more minutes, with bradycardia (50 beats/min) which was managed with a bolus dose of 0.6 mg IV of atropine and 6 mg IV of mephentermine.

Following the administration of atropine the patient developed tachycardia (130 beats/min) and raised blood pressure (160/100 mmHg) along with severe headache. About 40 minutes post spinal anesthesia blockade (SAB) and subsequent delivery, she developed generalized tonic-clonic seizure (GTCS), which was immediately controlled by a bolus dose of IV midazolam 1 mg. Another GTCS episode occurred 15 minutes later (55 minutes post SAB), managed with IV midazolam 1mg and seizure prophylaxis via a loading dose of IV phenytoin 1 g in 100 mL normal saline over 30 minutes. Her blood pressure and heart rate remained elevated for approximately 30 minutes after the dose of atropine. She developed post-ictal confusion and irritability but maintained her respiration without airway management or mechanical ventilation. She delivered a healthy female infant with Apgar scores of 6 and 7 at 3 and 5 minutes, respectively.

After 75 minutes, she was shifted to postoperative area on completion of surgery. The effect of spinal anesthesia lasted for 96 minutes (sensory block regression to T10). During her postoperative period, she had two more episodes of GTCS within 12 hours of delivery, accompanied by post-ictal confusion, headache, nausea, and tachypnoea, managed with IV lorazepam 4 mg. However, her vital signs remained within normal limits in the post-operative ward.

Arterial blood gas (ABG) analysis after the second seizure episode revealed respiratory acidosis (pH: 7.30; pO_2: 433 mmHg; pCO_2: 54.1 mmHg; HCO_3: 26.9 mmol/L; Na^+: 138 mmol/L; K^+: 4.0 mmol/L; Ca^{2+}:1.02 mmol/L). Postoperatively all laboratory results were within normal limits except low serum magnesium (1.45 mg/dL), which was replaced by a loading dose of 4 g IV magnesium sulfate over 30 minutes followed by maintenance of 2 g/hour for 24 hours postpartum. During the 24-hour observation period, she remained seizure-free, and her magnesium levels normalized to 1.84 mg/dL. Consequently, she was shifted to the obstetrical ward for recovery.

Notably, her previous pregnancy had been uneventful, and during her current gestation she had made regular antenatal visits. Additionally, there was no significant family history, addiction, or known substance allergies.

Electroencephalography (EEG) yielded normal results. Magnetic resonance imaging (MRI) of the brain revealed hyperintensity in the posterior occipital region with adjacent sulcal effacement, consistent with a diagnosis of PRES. [Figure 1].

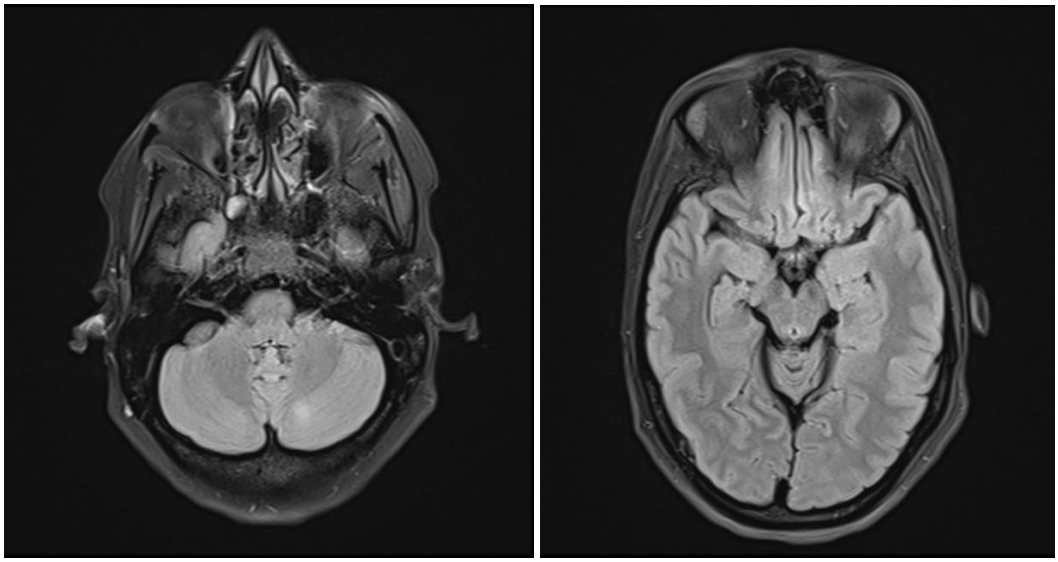

Figure 1: Magnetic resonance images (MRI) of the brain showing hyper-intensity in the posterior occipital region with adjacent sulcal effacement.

After five days of post-surgical recovery, the patient was discharged home. Though another MRI after three weeks was advised, she did not consent to it. She was followed up telephonically for four months.

Discussion

PRES should be considered in pregnant women presenting with sudden-onset headaches, seizures, vomiting, and altered mental status.[3] Our patient had a normal antenatal history, but experienced increased blood pressure and subsequent headache and seizure during surgery following administration of mephentermine and atropine. Differential diagnosis included 'Hemolysis, Elevated Liver Enzymes, and Low Platelet Count' (HELLP) Syndrome, preeclampsia or eclampsia, cerebrovascular event, local anesthetic systemic toxicity (LAST), and PRES. No signs of eclampsia or preeclampsia were present.[5] HELLP syndrome was ruled out due to normal blood results and no history of hypertension. LAST was considered unlikely due to small anesthetic doses and absence of early symptoms. Fundus examination and cranial nerve assessments did not reveal abnormalities suggestive of cerebrovascular events.

The most popular theory for the pathophysiology of PRES is that rapidly developing hypertension may lead to hyperperfusion and disruption of the blood–brain barrier leading to vasogenic edema, mainly of the white matter.[6] Presence of bilateral and extensive edema is suggestive of PRES. In vasogenic edema, MRI reveals hyperintense T2 and Fluid-attenuated inversion recovery (FLAIR) signals without restricted diffusion. In contrast, cytotoxic cerebral edema involves intact blood-brain barrier, with MRI indicating diffusion restriction.[7] Magnesium plays a crucial role in blood pressure regulation. Blood pressure fluctuations and hypomagnesemia might have contributed to PRES in our patient.[4,6] However there has been a case report of magnesium toxicity leading to PRES in a preeclampsia patient.[8]

Prognosis in PRES cases are generally favorable, contingent upon the timely and adequate management of the underlying condition. Most (70%–90%) PRES patients recover within two to eight days.[9] Adverse outcomes such as cerebral hemorrhage, ischemia, irreversible neurological deficits, and mortality have been reported in 8%–17% of cases.

Conclusion

This case offers two significant learning points. First, it underscores that PRES can manifest in pregnancy even in the absence of conventional risk factors. This expands the spectrum of potential causes for PRES, emphasizing the need for vigilance in pregnant patients presenting with relevant symptoms. Second, conducting routine serum magnesium assessments in all pregnant women could aid in preventing PRES associated with magnesium deficiency.

Disclosure

The authors declare no conflicts of interest. Informed consent was obtained from the patient.

References

1. Fischer M, Schmutzhard E. Posterior reversible encephalopathy syndrome. J Neurol 2017 Aug;264(8):1608-1616.

2. Sulaiman W, Husin NM, Baba MS. Posterior reversible encephalopathy in undiagnosed systemic lupus erythematous: a case report. Oman Med J 2011 Nov;26(6):e026.

3. Zhang Y, Liang B, Zhao C, Zhou Y, Yan C. Posterior reversible encephalopathy in a pregnant woman without preeclampsia or eclampsia: a case report. Medicine (Baltimore) 2022 Sep;101(36):e30519.

4. Chardain A, Mesnage V, Alamowitch S, Bourdain F, Crozier S, Lenglet T, et al. Posterior reversible encephalopathy syndrome (PRES) and hypomagnesemia: a frequent association? Rev Neurol (Paris) 2016;172(6-7):384-388.

5. Mai H, Liang Z, Chen Z, Liu Z, Xu Y, Chen X, et al. MRI characteristics of brain edema in preeclampsia/eclampsia patients with posterior reversible encephalopathy syndrome. BMC Pregnancy Childbirth 2021 Oct;21(1):669.

6. Rabinstein AA, Mandrekar J, Merrell R, Kozak OS, Durosaro O, Fugate JE. Blood pressure fluctuations in posterior reversible encephalopathy syndrome. J Stroke Cerebrovasc Dis 2012 May;21(4):254-258.

7. Schaefer PW, Buonanno FS, Gonzalez RG, Schwamm LH. Diffusion-weighted imaging discriminates between cytotoxic and vasogenic edema in a patient with eclampsia. Stroke 1997 May;28(5):1082-1085.

8. Lee AT, Cordova JC, Jamplis RP, Pomicter GR. Posterior reversible encephalopathy syndrome and eclampsia in the setting of magnesium toxicity: a case report. A A Pract 2023 Nov;17(11):e01726.

9. Hobson EV, Craven I, Blank SC. Posterior reversible encephalopathy syndrome: a truly treatable neurologic illness. Perit Dial Int 2012;32(6):590-594.