Case Report

A complex case of posterior reversible encephalopathy syndrome after combined spinal epidural of preeclampsia parturient: A case report

ABSTRACT

Posterior reversible encephalopathy syndrome (PRES) is a disorder characterized by vasogenic edema affecting the posterior brain region. We report a case of PRES in a 36-year-old woman with preeclampsia who underwent an emergency cesarean section with spinal anesthesia. After surgery, she developed right leg weakness, headache, and seizures. Imaging showed white matter edema consistent with PRES. The exact cause of PRES is unclear, but elevated blood pressure and endothelial dysfunction are implicated. Tight blood pressure control in PRES is crucial for management, and prompt recognition and treatment are essential for favorable outcomes.

Key words: Posterior reversible encephalopathy syndrome, pre-eclampsia, spinal anesthesia

Introduction

Posterior reversible encephalopathy syndrome (PRES) is a clinical and radiological disorder characterized by vasogenic edema primarily affecting the posterior occipital and parietal lobes of the brain.[1] It presents with symptoms including headache, seizures, altered mental status, and visual impairment.[2] This report highlights the diagnostic and management challenges of a patient with a complex presentation of symptoms and PRES following cesarean delivery.

Case Report

A 36-year-old woman with a twin pregnancy at 35 + 2 weeks underwent an emergency cesarean section due to pre-eclampsia, which presented with high blood pressure (BP) (165/92 mmHg) and proteinuria (3+).

In the operating room, her initial vital signs were recorded as follows: BP of 167/101 mmHg, heart rate (HR) of 72 beats per minute, oxygen saturation (SpO_2) of 99%. After positioning the patient laterally, an 18-gauge Tuohy needle (Combined Spinal/Epidural Minipack with Lock, Portex®, USA) was inserted at the lumbar spine level 3-4. After confirming the loss of resistance with air, clear cerebrospinal fluid was obtained on the first attempt with a 27-gauge pencil point needle. Subsequently, 9 mg of 0.5% bupivacaine was slowly injected to achieve a T-4 sensory level block, and a 4 cm epidural catheter was inserted into the epidural space, directed cephalad.

Access this article online	
Website: https://journals.lww.com/sjan	Quick Response Code
DOI: 10.4103/sja.sja_646_23	

How to cite this article: Heo MH, Choi HY, Lee K, Kim JY. A complex case of posterior reversible encephalopathy syndrome after combined spinal epidural of preeclampsia parturient: A case report. Saudi J Anaesth 2024;18:134-6.

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Submitted: 24-Jul-2023, **Accepted:** 24-Jul-2023, **Published:** 02-Jan-2024

After the start of the surgery, the patient complained of mild upper abdominal pain, and an additional dose of 75 mg 0.75% ropivacaine was administered through the epidural catheter. The babies were delivered without complications, and the surgery proceeded smoothly.

On postoperative day (POD) 1, the patient complained of chest discomfort [Table 1]. Her vital signs were measured slightly high BP (150/70 mmHg). The laboratory tests and imaging results showed no abnormalities except for D-dimer levels of 5.08 mcg/ml (normal <0.5 mcg/ml), and an NT-pro BNP level of 319.3 pg/ml (normal <97.3 pg/ml). After administering IV furosemide 10 mg, the chest discomfort resolved.

On POD 2, the patient developed right leg weakness with a motor grade of 1/5 observed in right hip flexion and a headache (Numeric Rating Scale, NRS, 5/10). The headache worsened with positional changes, and post-dural puncture headache (PDPH) was suspected. A computed tomography scan of the lumbosacral spine was performed to evaluate the possibility of an epidural hematoma causing the right leg weakness, which revealed the presence of an air bubble (16.2 × 3.9 mm) in the epidural space [Figure 1a and b]. However, no epidural hematoma was found and it was decided to manage the symptoms with acetaminophen and hydration instead of performing an epidural blood patch (EBP).

However, on POD3, the patient's BP increased (160/100 mmHg), and the headache worsened to a thunderclap-like pattern (NRS 7/10) accompanied by visual disturbances, and then, she developed a generalized tonic-clonic seizure for one minute. A magnetic resonance imaging (MRI) of the brain was performed, which showed areas of hyperintensity in the posterior parietal sections, indicating white matter edema consistent with PRES [Figure 2a and b]. Another seizure occurred six hours later, and the patient was treated with supplemental oxygen and intravenous lorazepam (2 mg). An electroencephalogram showed cerebral dysfunction but no epileptiform discharges, and she was referred to the neurology department.

On POD 4, her BP was controlled, resulting in a gradual improvement of headache and right leg weakness. A follow-up brain MRI on POD 16 showed a complete resolution of PRES findings. The patient was discharged the following day without any sequelae.

Discussion

Our patient underwent a cesarean section due to pre-eclampsia with spinal anesthesia and subsequently presented with a combination of symptoms, leading to a diagnosis of PRES.

Although the exact cause of PRES, in this case, is not known, several possibilities can be considered. Firstly, as a pre-eclamptic patient, the patient had a slightly elevated

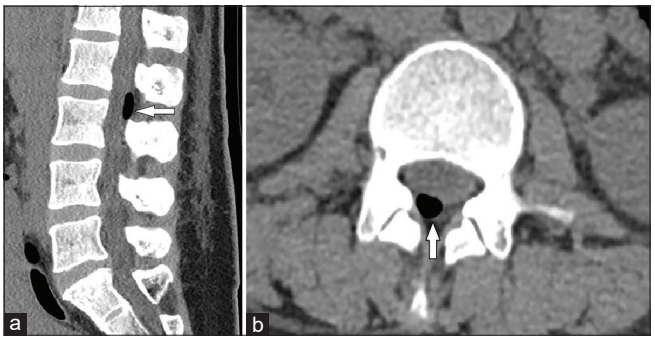

Figure 1: The air bubble (16.2 × 3.9 mm, white arrow) in L2-3 level epidural space on Lumbosacral spine Computed tomography on postoperative day 2. (a) Sagittal view, (b) Axial view

Table 1: Postoperative patient's symptoms, signs and examinations

POD	BP	Symptom & Signs	Lab (normal range)/CT or MRI/EEG
1	150/70 mmHg	Chest discomfort	CK-MB: 1.1 ng/ml (<4.0 ng/ml) Troponin I: 5.7 pg/ml (<15.6 pg/ml) NT-proBNP: 319.3 pg/ml (<97.3 pg/ml) D-dimer: 5.08 mcg/ml (<0.5 mcg/ml)
2	150/80 mmHg	Right leg weakness (Grade 1 in right hip flexion) Headache (NRS 5/10)	Lumbosacral spine CT : Air bubble in the epidural space (16.2×3.9 mm)
3	160/100 mmHg	Headache (Thunderclap-like pattern) (NRS 7/10) Visual disturbance Generalized tonic-clonic seizures two times	Brain MRI : Hyperintensity in the posterior parietal sections, indicating white matter edema EEG : Cerebral dysfunction but no epileptiform discharge.
16	120/70 mmHg	No problematic symptoms or signs	Brain MRI : Complete resolution of PRES findings EEG : Normal finding

POD: Postoperative day, BP: blood pressure, CT: computed tomography, MRI: Magnetic resonance imaging, EEG: Electroencephalogram, NRS: Numeric Rating Scale, PRES: posterior reversible encephalopathy syndrome

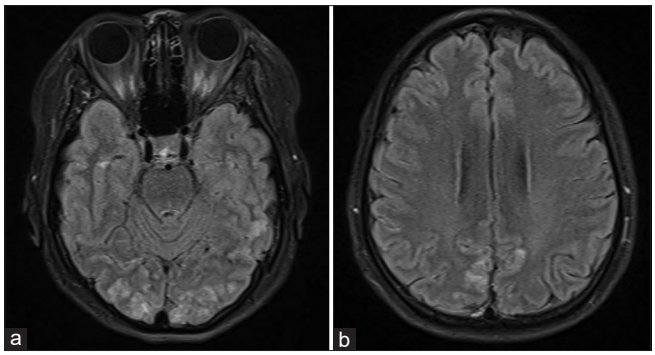

Figure 2: T2W1/T2 FLAIR MRI showing bilateral occipital lobe (a) and posterior cingulate gyrus (b) hyperintensities, which are nearly symmetrical, consistent with Posterior reversible encephalopathy syndrome

BP with a systolic reading of 150 mmHg even after the surgery. The persistent elevation of BP could have triggered hyperperfusion, leading to the worsening of vasogenic edema.[2] In cases where pregnancy and pre-eclampsia lead to endothelial dysfunction and a breakdown of autoregulation, there is a possibility that the patient is susceptible to PRES even without severely elevated BP.[3] Second, there is a possibility that PRES was triggered by CSF leakage resulting from PDPH. The decrease in CSF volume can lead to the collapse of ventricles, and this traction effect can induce cerebral vasospasm, thereby increasing the likelihood of PRES occurrence.[4] In addition, persistent CSF leakage results in increased blood flow to the leptomeningeal vessels, according to the Monro-Kellie doctrine, which aims to compensate for the loss of CSF volume.[5] If there was CSF leakage due to PDPH, it is possible that it acted as a contributing factor to exacerbate PRES.

In our patient, the mix of several symptoms made the initial diagnosis of PRES challenging. In a typical PRES patient, the diagnosis is usually based on symptoms such as dull, diffuse, gradual onset, thunderclap and non-postural headache, visual disturbances, and seizures, followed by an MRI. However, in our patient's case, severe right leg weakness (motor grade 1) and postural headaches occurred before the common PRES symptoms manifested. These symptoms raised the suspicion of conditions such as PDPH epidural hematoma or epidural air rather than PRES. As a result, PRES was not initially considered. Furthermore, the headache was mild (NRS 3) and there was concern that performing EBP might worsen right leg weakness. Therefore, hydration and medication were chosen as treatment options instead of EBP. However, it was possible that hydration might have increased the likelihood of PRES.[4] The diagnosis of PRES was delayed because our patient's symptoms of limb weakness and headache were not typical of PRES. If the possibility of PRES had been predicted sooner, the diagnosis and treatment could have been made before the seizure occurred.[3,6]

In conclusion, in patients with pre-eclampsia, the early diagnosis of PRES is essential to prevent neurological symptoms associated with PRES and tight BP control is important for treatment.[7] Neurological sequelae and serious complications may occur, emphasizing the need for careful management, especially in high-risk patients, even though most cases have a reversible course.[2]

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given her consent for her images and other clinical information to be reported in the journal. The patient understands that her name and initials will not be published and due efforts will be made to conceal identity, but anonymity cannot be guaranteed.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

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