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# Post-Dural Puncture Posterior Reversible Encephalopathy Syndrome

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## ABSTRACT

We report the case of a young patient with 36 weeks pregnancy, and an acute respiratory infection with severe bronchospasm, who developed an occipital headache and neck pain on the third day of inadvertent dural puncture during placement of combined epidural spinal anaesthesia for caesarian section. It was diagnosed as post-dural puncture headache until generalised tonic clonic seizures occurred the next day raising the suspicion of postpartum eclampsia or meningitis. Posterior reversible encephalopathy syndrome was diagnosed on MRI of the brain which showed features of reversible ischemia in the posterior region of the brain. With anticonvulsant therapy and antibiotics there was complete resolution of neurological symptoms. We highlight the importance of high index of suspicion of this reversible encephalopathy in obstetric cases with intentional or inadvertent dural puncture, with headache similar to post-dural puncture headache, and the essential role of neuroradiology in confirmation of the diagnosis, as placement of an epidural blood patch would be highly detrimental in these cases.

**Key words:** *Postdural puncture headache. Postpartum seizures. Cerebral autoregulation. Encephalopathy. Reversible ischemia. Respiratory tract infection.*

## INTRODUCTION

First described in 1996 posterior reversible encephalopathy syndrome (PRES) is a transient clinico-neuroradiological syndrome characterized by symptoms of neurological disease such as headache, altered mental status, seizures and visual loss in a variety of non-neurological clinical entities.<sup>1</sup> The most common association is with hypertension, immunosuppressed states, obstetric patients, infection, sepsis and shock.<sup>2-4</sup> In obstetric patients it may be associated with uncomplicated pregnancy, complications of pregnancy, and intentional or unintentional dural puncture. Most cases reported in obstetrics were in the third trimester of pregnancy. If unrecognized and inappropriately treated it can lead to permanent neurological damage or death. The cornerstone of successful management is a high index of suspicion, early radiological confirmation of diagnosis and appropriate therapy. This precisely is the aim behind the reporting of this case.

## CASE REPORT

We report the case of a young previously healthy multiparous patient with 36 weeks pregnancy who was referred to our centre for progressively increasing dyspnoea, orthopnoea and dry cough due to an acute

respiratory tract infection for the last 4 days. The patient reported decreased fetal movements. Her previous pregnancies were uneventful. She had diffuse generalised bilateral ronchi on chest auscultation. Her laboratory tests showed a raised white cell count of 27,000/cmm and C reactive protein of 3.6 mg/dl. In the arterial blood gas analysis, the pH was 7.49, PaCO<sub>2</sub> was 29.60 mmHg, PaO<sub>2</sub> was 62.50 mmHg, HCO<sub>3</sub> was 27 mEq/L, Base Excess was 0.2 and the SaO<sub>2</sub> was 93%, indicative of hypoxemia and respiratory alkalosis. A limited echocardiogram to exclude peripartum cardiomyopathy reported preserved left ventricular systolic function with a visually estimated ejection fraction of 55%, normal valves and chambers. The rest of the laboratory tests and chest X-ray were unremarkable. A diagnosis of severe respiratory tract infection with reactive airways was made and aggressive therapy with antibiotics, bronchodilators and steroids started. An abdominal ultrasound showed a single, viable fetus with oligohydramnios. After 24 hours of therapy there was some resolution of bronchospasm but arterial hypoxemia remained unresolved. The decision to proceed with caesarian section and tubal ligation was taken in view of severe maternal illness. Pre-operatively the patient was hemodynamically stable and afebrile. Arterial oxygen saturation was 94% on 4 litres of oxygen by face mask. In the pre-operative arterial blood gas analysis the pH was 7.53, PaCO<sub>2</sub> was 24.6 mmHg, PaO<sub>2</sub> was 58.2 mmHg, HCO<sub>3</sub> was 21 mEq/L, Base Excess was 0.2 and the SaO<sub>2</sub> was 93%. Regional anaesthesia was planned with the patient's consent, primarily to avoid airway manipulation, opioids and non-steroid anti-inflammatory drugs intraoperatively and postoperatively.

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Intravenous lactated Ringer's infusion was started. ECG and arterial oxygen saturation were monitored per routine. An arterial line was secured through left radial artery for invasive arterial pressure monitoring and blood gas analysis. She was positioned for combined spinal epidural placement in the lateral decubitus position, in which she was comfortable and cooperative. After all aseptic precautions CSE placement was attempted at L3-4 interspace. Inadvertent dural puncture occurred during epidural needle insertion. 1.5 ml of 0.5% bupivacaine with 25 µg fentanyl was given intrathecally through the puncture and patient positioned for surgery. The urinary bladder was catheterized.

Surgery was uneventful except for one episode of bronchospasm after injection of syntocinon which was managed with ipratropium nebulisation. After delivery of the baby, propofol infusion was started for sedation at the rate of 25-30 µg/kg/minute. At the end of surgery the patient was explained the need for epidural catheter placement for pain relief which was placed successfully at L2-3 interspace above the dural puncture site. Epidural infusion of marcaine 0.1% + fentanyl 2 µg/ml was started at the rate of 8-10 ml/hour. Epidural catheter was removed on postoperative day two and oral paracetamol started for analgesia. On postoperative day three, she developed neck pain and headache in the occipital region, which was worsened by sitting-up. A presumptive diagnosis of post-dural puncture headache was made and conservative therapy started. As headache was not relieved by the next day an epidural blood patch was scheduled. During transfer to the operating room the patient had a generalized tonic clonic seizure, which was controlled with intravenous midazolam 3 mg, followed by intravenous phenytoin loading with 15 mg/kg. An intravenous infusion of magnesium sulphate 4 gm followed by 1 gm hourly was also started on suspicion of postpartum eclampsia and a neurological consultation was requested to rule out meningitis. The white cell count was 21,000/cmm on the day of the seizure. Encephalopathy was not suspected.

On neurological examination the reflexes were brisk and Babinski's sign was present on both sides. There was pain on neck flexion but no nuchal rigidity was noted. An MRI followed by an LP for CSF analysis was scheduled. The MRI reported hyperintense signals bilaterally in parietal and occipital lobes in gyriform pattern not showing diffusion restriction, which could represent reversible ischemic changes. Diffuse meningeal enhancement was noted which was most likely due to dural puncture (Figure 1). The rest of the brain was normal.

Posterior reversible encephalopathy syndrome was diagnosed in consideration of the radiological evidence and clinical context. CSF analysis was omitted and treatment continued with phenytoin, vancomycin, levoflox and meropenem. Headache and neck pain

resolved completely within 24 hours and no further seizures occurred. Although subjectively the patient was comfortable complete resolution of bronchospasm occurred on postoperative day six. The white cell count prior to discharge was 16,000/cmm. The patient was discharged on postoperative day eight with advice to continue antibiotics and bronchodilators, and taper steroids till follow-up in two weeks time. She was lost to follow-up.

## DISCUSSION

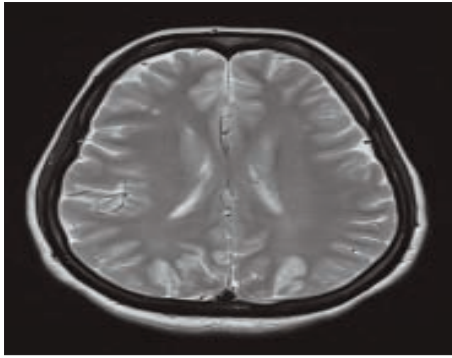
Posterior reversible encephalopathy syndrome (PRES) is a clinico-neuro-radiologic entity with signs and symptoms of encephalopathy in many diverse, primarily non-neurological conditions. Hypertension, immunosuppression, infection, sepsis, shock,<sup>1-4</sup> uncomplicated and complicated pregnancies,<sup>5-7</sup> intentional or inadvertent dural puncture,<sup>8-10</sup> and drugs like cyclosporin, erythropoietin, cisplatin and long-term steroids may cause PRES. Hemolytic uremic syndrome, hepatorenal syndrome, acute intermittent porphyria, HIV, blood transfusion, post-carotid endarterectomy and thrombotic thrombocytopenic purpura are also risks for this encephalopathy. There is a definite female preponderance of cases.

The symptoms are of acute onset and progress over hours to days. Our patient was 36 weeks pregnant, with severe respiratory tract infection, who had an inadvertent dural puncture during epidural placement. The onset of occipital headache and neck pain on postoperative day three was assumed to be a post-dural puncture headache; occurrence of tonic clonic seizures raised a suspicion of late post-partum eclampsia or meningitis. We did not suspect a reversible encephalopathy.

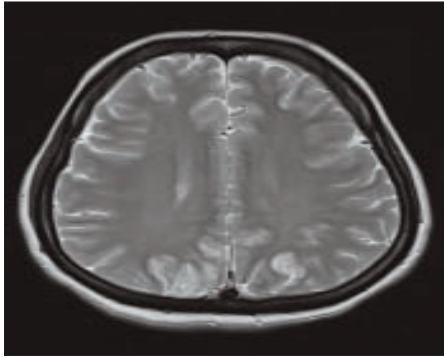
Magnetic resonance imaging (MRI) is considered the Gold standard for diagnosing PRES. Diffusion weighted MRI can detect white matter edema early (Figure 1f), and reliably differentiates between vasogenic and cytotoxic edema.<sup>2,6</sup> Since most of the lesions in PRES are in the cortical and sub-cortical areas, adjacent bright CSF may make it difficult to identify these areas on T2 images (Figures 1a,b). Fluid attenuated inversion recovery (FLAIR) is a technique used to suppress adjacent CSF on T2-weighted MR images (Figures 1c,d). This makes lesions in sub-cortical and cortical areas more prominent, identifying such lesions in 95% cases.<sup>1-6</sup> Diagnosis in this case was established by radiologists and neurologists upon the distinctive MRI pattern of reversible ischemia in the posterior region of the brain (Figures 1 a-f).

Disruption of autoregulation and blood brain barrier are focal to the pathophysiology of brain edema in PRES.<sup>2,6,8</sup> The posterior circulation may be more

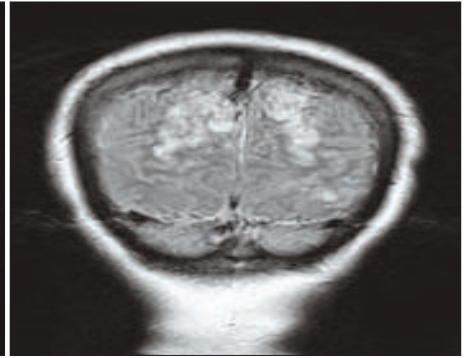
**Figure 1 (a-f):** These are the regions of reversible ischemia.



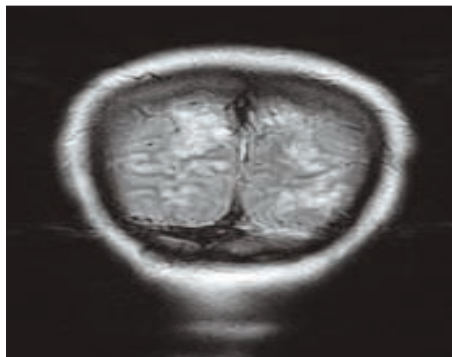
**Figure 1a:** T2-weighted MRI scan of the brain showing hyperintense signals in the occipital region (Note a small area of abnormal signal intensity in the right frontal region).



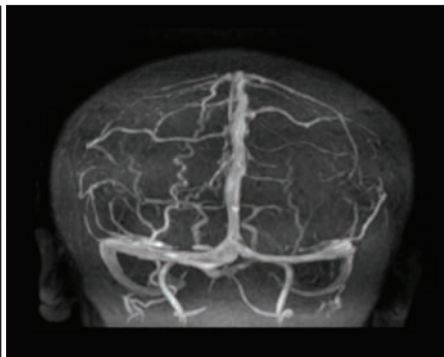
**Figure 1b:** T2-weighted MRI scan of the brain showing hyperintense signals in the Occipital region.



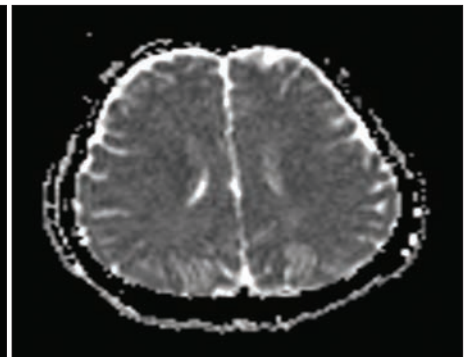
**Figure 1c:** Fluid attenuated inversion recovery (FLAIR) MRI scan of the brain showing hyperintense signals in the occipital region.



**Figure 1d:** Fluid attenuated inversion recovery (FLAIR) MRI scan of the brain showing hyperintense signals in the occipital region.



**Figure 1e:** Magnetic venous angiogram (MRV) was normal.



**Figure 1f:** Diffusion weighted MRI was normal.

vulnerable to disruption of autoregulation because of the lower sympathetic innervation compared to the internal carotid artery territory.<sup>2</sup>

Most patients recover completely, with resolution of CT and MRI changes, as was the case in this patient. The awareness of this reversible encephalopathy among anaesthesiologists, obstetricians and other clinicians is essential to avoid missing the diagnosis, and initiating correct therapy.

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