Posterior reversible encephalopathy syndrome following caesarean section under spinal anaesthesia

INTRODUCTION

Complaints of headache in a patient after spinal anaesthesia usually arouses the suspicion of postdural puncture headache. Similarly, the first diagnosis which comes to our minds if there is development of seizures in a patient following caesarean section will be eclampsia. Though cases of posterior reversible encephalopathy syndrome (PRES) at term are reported,[1] we tend to overlook this increasingly reported neurologic syndrome. It is gaining more and more importance as timely diagnosis and proper treatment results in complete recovery without any permanent neurological sequelae.

CASE REPORT

A 38-year-old primigravida (weight 68 kg, height 154 cm) admitted for safe confinement, at 38 weeks of gestation, was posted for elective caesarean section due to non-progression of labour. The patient had an uneventful antenatal period, with no history suggestive of pregnancy induced hypertension. Physical examination findings, blood and urine reports were within normal limits. The patient was fasting for 8 h and was willing for spinal anaesthesia.

The patient was shifted to theatre and was preloaded with 750 ml of Ringer Lactate solution. Spinal anaesthesia procedure was performed with the patient in left lateral position with a 25 Gauge Quincke needle at L3-L4 interspace. Free cerebrospinal fluid flow was obtained in the first attempt itself and 2 ml of 0.5% hyperbaric bupivacaine solution was injected into the subarachnoid space. The level of sensory block was assessed by pin prick method and the maximum level achieved was at T_4. A female baby was delivered with an APGAR score of 7 at 1 min and 9 at 5 min. The intraoperative as well as the immediate post-operative periods were uneventful.

On first post-operative day (POD), in the ward, she complained of severe occipital headache with radiation to the neck in the upright position which was relieved by lying supine. There was no fever, neck rigidity or vomiting. Blood pressure as well as the blood counts were normal. A diagnosis of post-dural puncture headache was made and she was managed with IV fluids (1000 ml of Ringer lactate/day, in addition to oral fluids), intravenous paracetamol 1 g 8 h and bed rest. Severity of pain came down and the patient was comfortable.

On the third POD she started to complain of severe continuous headache in the occipital region with no relief even in a recumbent posture, with no vomiting or visual disturbances. Soon the patient developed generalized tonic clonic convulsions which were initially managed with midazolam 2 mg intravenously followed by phenytoin 600 mg bolus and 100 mg 8 h administered intravenously. The seizure lasted for <2 min, and the patient became conscious but confused. The vitals remained normal throughout. Finally the patient was shifted for radioimaging and axial fluid attenuated inversion recovery magnetic resonance imaging (MRI). The image showed bilateral symmetric hyperintensities in the occipital cortex [Figure 1]. Higher level image also showed cortical hyperintensities involving frontoparietal cortex of both sides [Figure 2]. These features were suggestive of PRES. The patient was further managed with intravenous magnesium sulphate, as it provides the most favourable outcome when PRES is diagnosed in pregnant patients. Initially, 4 g in 100 ml was given as a bolus infusion over 20 min followed by 2 g/h for 24 h and nimodipine 60 mg 6 h was added orally. Both were continued for 5 days with daily serum magnesium level monitoring for the pathology to revert and later stopped as the patient was asymptomatic with no neurological deficits. Subsequently she was discharged on 8th POD and remained asymptomatic till date.

DISCUSSION

Posterior reversible encephalopathy syndrome is also known as reversible posterior cerebral oedema syndrome, posterior leucoencephalopathy syndrome, hyperperfusion encephalopathy or brain capillary leak syndrome. The patients usually present with headache, seizures and visual loss which is often associated with hypertension. However, hypertension is absent in about one-fourth of patients and even if present, does not usually reach the level of failed autoregulation.[2] Early diagnosis and treatment of PRES will prevent progression to irreversible neurological damage.[3]

It is more common in females and no age group is spared.[4] Predisposing conditions include eclampsia, solid organ transplantation, infections, sepsis, shock, autoimmune disease, and can also occur following cancer chemotherapy.[2] The pathogenesis of PRES remains unclear, but deranged cerebral autoregulation and endothelial dysfunction are believed to play a major role.[5] Neuroimaging is essential for the diagnosis of PRES. Abnormalities are often apparent on computed tomography scans but are best identified by MRI.[6] The common findings observed are symmetrical white matter oedema in the posterior cerebral hemispheres, particularly the parieto-occipital regions, but variations may occur.[7] Atypical imaging features, such as involvement of anterior cerebral regions, deep white matter and brainstem are also frequently seen. Vasoconstriction is common in vascular imaging.

The goals in the management of PRES include (i) the removal or reduction of the causative drug, (ii) management of blood pressure, if hypertension present, (iii) control of seizures and (iv) in pregnant

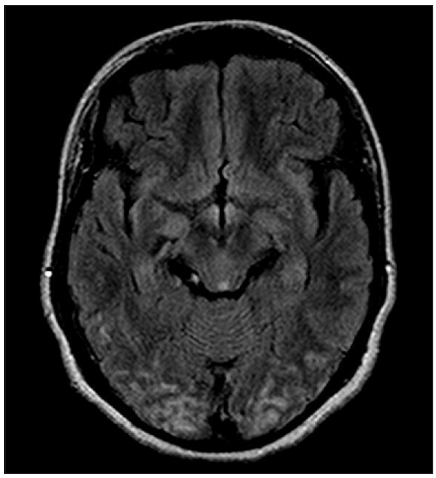

Figure 1: Axial fluid attenuated inversion recovery magnetic resonance imaging showing bilateral symmetric hyperintensities in the occipital cortex

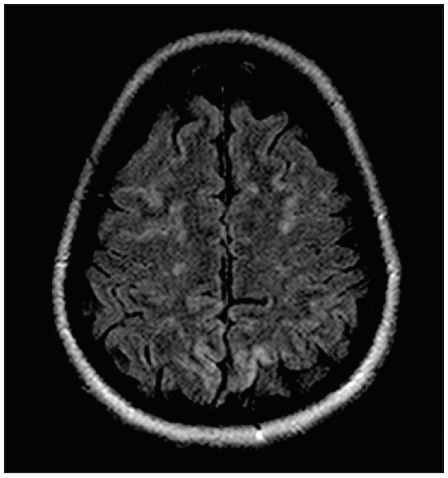

Figure 2: Axial fluid attenuated inversion recovery magnetic resonance imaging at a higher level showing cortical hyperintensities involving both fronto parietal cortex

patients termination of pregnancy by caesarean section, if symptoms are refractory.[8] In addition, maintenance of airway and arterial oxygenation, sufficient hydration, correction of hypoglycaemia, electrolyte disturbances, or coagulopathy should be taken care of simultaneously.

Intravenous nicardipine (5–15 mg/h) and labetalol (2–3 mg/min) are considered first-line medications for control of hypertension in PRES.[8] Hydralazine and diazoxide may also be used. However, nitroglycerine should not be used as it aggravates cerebral oedema.

Status epilepticus or other kinds of epileptogenic activity require immediate treatment to prevent permanent neuronal damage or death. Lorazepam is administered intravenously in boluses of 2 mg given slowly over 2 to 5 min, up to a maximum dose of 10 mg. This is followed by phenytoin (15–18 mg/kg initially) if seizure persists after 20 min. In resistant cases midazolam, barbiturates, or propofol can be used.[9] However in pregnant women with preeclampsia, magnesium sulphate is effective in prevention and management of seizures.[10]

Other differential diagnoses of convulsions in the peripartum period include intracerebral haemorrhage, thrombotic phenomena, head injury, meningitis, encephalitis, first onset of epilepsy, first manifestation of intracranial tumours or neurocysticercosis. A high index of clinical suspicion in the appropriate situations will help to recognize PRES.

CONCLUSION

The possibility of PRES should be kept in mind as a cause of eclampsia, since timely management of this otherwise reversible condition will prevent permanent neurological damage.

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REFERENCES

1. Achar SK, Shetty N, Joseph TT. Posterior reversible encephalopathy syndrome at term pregnancy. Indian J Anaesth 2011;55:399-401.
2. Bartynski WS. Posterior reversible encephalopathy syndrome, part 1: Fundamental imaging and clinical features. AJNR Am J Neuroradiol 2008;29:1036-42.
3. Stott VL, Hurrell MA, Anderson TJ. Reversible posterior leukoencephalopathy syndrome: A misnomer reviewed. Intern Med J 2005;35:83-90.
4. Alehan F, Erol I, Agildere AM, Ozcay F, Baskin E, Cengiz N, *et al.* Posterior leukoencephalopathy syndrome in children and adolescents. J Child Neurol 2007;22:406-13.
5. Bartynski WS. Posterior reversible encephalopathy syndrome, part 2: Controversies surrounding pathophysiology of vasogenic edema. AJNR Am J Neuroradiol 2008;29:1043-9.
6. Oppenheim C, Logak M, Dormont D, Lehéricy S, Manaï R, Samson Y, *et al.* Diagnosis of acute ischaemic stroke with fluid-attenuated inversion recovery and diffusion-weighted sequences. Neuroradiology 2000;42:602-7.
7. Fugate JE, Claassen DO, Cloft HJ, Kallmes DF, Kozak OS, Rabinstein AA. Posterior reversible encephalopathy syndrome: Associated clinical and radiologic findings. Mayo Clin Proc 2010;85:427-32.
8. Servillo G, Bifulco F, De Robertis E, Piazza O, Striano P,

Tortora F, *et al*. Posterior reversible encephalopathy syndrome in intensive care medicine. Intensive Care Med 2007;33:230-6.
9. Knake S, Hamer HM, Rosenow F. Status epilepticus: A critical review. Epilepsy Behav 2009;15:10-4.
10. Euser AG, Cipolla MJ. Magnesium sulfate for the treatment of eclampsia: A brief review. Stroke 2009;40:1169-75.

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