Journal of Bangladesh College of Physicians and Surgeons
Vol. 35, No. 1, January 2017

Posterior Reversible Encephalopathy Syndrome (PRES)

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Summary::

Posterior Reversible Encephalopathy Syndrome is a rare and acute neuro-radiological entity, characterized by several symptoms of different aetiologies. The main etiologies of PRES are HTN, Pre-eclampsia, Eclampsia, Immunosuppressive therapy etc, but there are many unexplained causes too. Here we report a case of post-partum woman who developed PRES in post-operative period, who was complicated by the postdural puncture headache following spinal anesthesia- a rare and uncommon etiology.

(J Bangladesh Coll Phys Surg 2017; 35: 43-45)

Introduction:

Posterior Reversible Encephalopathy Syndrome (PRES) is a proposed cliniconeuroradiological entity, characterized by several symptoms of different etiology. It predominantly affects the posterior circulation territory and clinical hallmarks are headache, confusion, altered mental status, seizures, visual disturbance and rarely other neurological signs[1]. PRES is seen, not only in relation to pre-eclamsia, but in a variety of disease or condition[3]. Postdural puncture headache is a well described complication of spinal anesthesia in caesarian delivery. Although PRES has not been associated with postdural puncture headache after spinal anesthesia in post partum women[4].

This is a case report of post partum women of an uneventful pregnancy who developed PRES after spinal anaesthesia which was complicated by the postdural puncture headache.

Case Report:

A 20 years old primipara underwent caesarean delivery under spinal anesthesia at her 40th week of gestation because of failure to progress. Her medical history and laboratory examination were unremarkable and pregnancy was uneventful throughout the antenatal period.

After an initial I/V preload of Lactate Ringer's solution (1000 ml), Spinal block was performed, using a midline approach. On the first attempt, clear cerebrospinal fluid was obtained. The patient delivered a healthy baby. The mother's vital signs were normal in per- operative and post operative period.

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Received: 18 May 2016 **Accepted:** 6 Nov. 2016

The patient was well until two days of post surgery. On the third day she developed severe postdural headache over bi frontal area, particularly in erect posture, which was relieved by recumbent position. The diagnosis of post dural puncture headache was made. The headache improved after two days of supportive therapy which includes aggressive intravenous hydration, bed rest without pillow and oral analgesics. On 5th postoperative day the patient developed a severe throbbing headache over the occipital area which was no longer postural. On the same day, she developed convulsion which was tonic-clonic in nature with aura. There were 2-3 episodes of convulsion at an interval of 1-2 minute within 25-30 minutes. During this seizure she was unconscious, B/P was 120/80mmHg, pulse 100/m, and urine albumin was nil. Her hematological and biochemical marker were within normal limit. She was nursed in ICU with Anticonvulsive (Mgso4) for 24 hrs, parenteral antibiotic and sedative. Routine hematological, biochemical test as well as chest X- ray, ECG were done and found normal. Diagnostic lumbar puncture revealed clear CSF fluid without any inflammatory cell. The patient's condition was stabilized after 48 hours of ICU nursing. Afterward CT scan and MRI of brain were performed and revealed edema of the bilateral Parieto occipital lobes with density changes and signal alteration respectively, impression was PRES.

During the period of post convulsion hospitalization, there was no episode of marked hypertension, urine albumin was nil and biochemical marker were absolutely normal. After diagnosis patient was treated by multidisciplinary approach by medical specialists and obstetricians. Treatment regimen was initial intravenous nalepsin followed by Injection Mannitol, parentral steroid and later on oral phenytoin were added.

Considering the stability and general condition of the patient gradually medications were withdrawn. She was

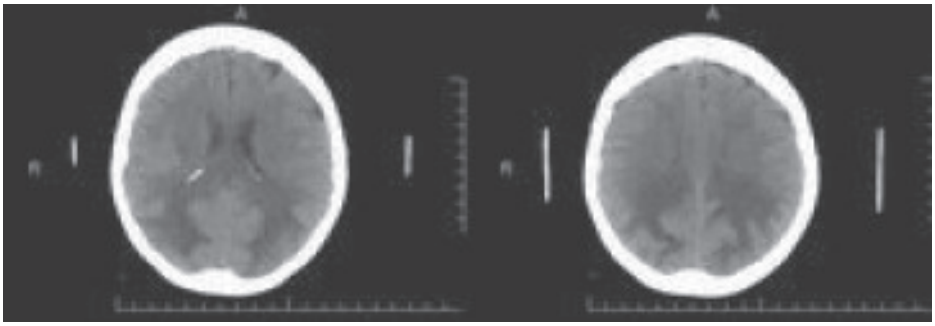

Fig.-1: *CT Scan- Diffuce hypodensity in both parieto occipital region.*

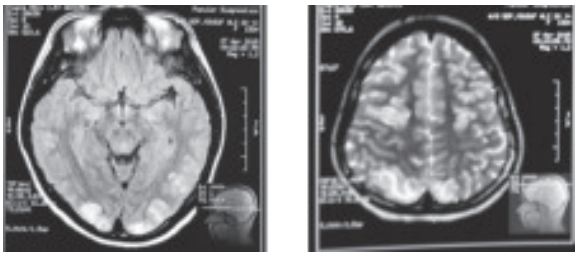

Fig.-2: *MRI- Hyper intense signal in both parieto occipital region in T2 and flare image.*

discharged from hospital at 12th post operative day with the advice for follow -up after 02 weeks. After 02 weeks MRI of brain was done which showed an almost complete resolution of the previous lesion.

Discussion.

PRES was first introduced into clinical practice in 1996[2]. Pathogenesis of PRES is thought to be multi-factorial, yet two different theories are dominating and still being debated[3]. The hyper perfusion theory also called the "vasogenic theory" and the hypo perfusion/ ischaemic theory also called the "cytotoxic theory"[3]. However, the pathogenesis is attributed to a failure of cerebral auto regulation that is probably facilitated in posterior brain region due to sparse sympathetic innervations of the vertebrobasilar vascular system[2].

Clinical symptoms, as well as neuroimaging finding in this patient are compatible with PRES. The patient presented with severe headache over the frontal followed by occipital area, decreased awareness and confusion followed by convulsion. These occurred 2 days after an episode of typical postdural puncture headache following spinal anesthesia for cesarean delivery. CT scan finding was bi lateral patchy hypodensities involving the cortex and subcortical white matter in parieto-occipital lobe. The MR imaging revealed cytotoxic edema of the cerebrum involving bilateral parieto-occipital lobes; MR angiography was unremarkable. These finding indicate that the lesion of PRES of this patient was associated with ischemia-induced cytotoxic edema.

Although the exact mechanism for PRES remains unknown, the temporal association of encephalopathy with diffuse cerebral vasospasm after an episode of postdural puncture headache may have been causative in this patient[4] .Persistent leakage of CSF through the dural opening left by the lumbar puncture needle can result in sagging of the brain and traction on nerves and meningeal vessels. Presumably, the diminished volume of CSF in the cerebral ventricle may collapse the ventricle. This traction might cause mechanical stimulation on the arterial wall and in theory, could induce vasospasm. Clearly, more clinical data are needed to prove this putative mechanism of collapsed ventricle-induced vasospasm[4]. In the current patient, diffuse cerebral artery vasospasm possibly was due to the traction of this vessel by anatomic brain displacement, could It provides an explanation for the development of PRES after spinal anesthesia .Furthermore, lack of evidence for systemic vasculopathy argues against an immune-mediated vacuities in our patient. In addition due error in the CNS, blood pressure auto regulation, and lack of sympathetic innervations of vessels emanating from basilar and vertebral arteries , blood flow in the CNS will increase. this cause elevated capillary filtration pressure and damage the capillary wall, eventually leading to increase blood brain barrier permeability and the consequence of cerebral oedema. All these observations suggest that diffuse cerebral vasospasm after an episode of post dural puncture

headache following spinal anesthesia could have caused PRES in this patient.

PRES has been reported to be reversible; although, irreversible brain damage can sometimes occur due to late recognition or incorrect treatment [2, 4]. Importantly, institution of early treatment, leads to symptom resolution without neurologic deficit, as was seen in this patient. Therefore, aggressive treatment for cerebral vasospasm is essential.

Several clinical studies have also shown that intravascular magnesium sulfate safely relieved maternal cerebral vasospasm[4]. In this case, all syndromes subsided after treatment with magnesium sulphate, Manitol, steroid, with sedative and other supportive treatment. The abnormal findings on neuro imaging had almost reversed after 15 days.

We describe the successful management of a postpartum woman with cerebral vasospasm-induced PRES associated with postdural puncture headache following spinal anesthesia as another condition underlying PRES. This management could be successful only with good intensive care support, proper monitoring and a team work combined with obstetrician and medical specialist.

Conclusion:

The cause of PRES is certainly multifactorial. The syndrome should be promptly recognized since it is reversible. The key to diagnosis is certainly the image, but clinicians must raise suspicion when there is a case with atypical features.

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