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RESEARCH ARTICLE

STUDY OF PRES IN POST-PARTUM PERIOD IN A TERTIARY CARE CENTRE – A RETROSPECTIVE STUDY

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ABSTRACT

Posterior reversible encephalopathy syndrome (PRES) is a cliniconeuroradiological entity presenting with headache, confusion, visual disturbances or blindness, and seizures. Parieto-occipital white matter changes due to vasogenic oedema can be observed on imaging modalities. It rarely occurs without seizures and usually occurs after delivery. We are reporting 9 post-partum patients with and without history of preeclampsia complicated by PRES with seizures and other clinical features at the postpartum period. Clinical improvement with complete resolution without any complications was observed except for one mortality which was admitted with advanced stage. Posterior reversible encephalopathy syndrome is reversible when early diagnosis is established and appropriate treatment is started without delay.

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INTRODUCTION

Reversible posterior encephalopathy syndrome is a clinicoradiologic entity characterized by headache, altered mental status, seizures, and visual loss and is associated with white matter vasogenic edema predominantly affecting the posterior occipital and parietal lobes of the brain. Most cases of PRES occur with hypertension or immunosuppression, but it can occur with many diverse clinical entities. The pathophysiology of PRES is related to disordered cerebral autoregulation. Two pathophysiologic mechanisms have been proposed regarding cerebral autoregulation—cerebral vasospasm, which results in cytotoxic edema, and vasodilatation, which results in vasogenic edema. The pathophysiology of PRES also implicates endothelial dysfunction, especially in cases without severe hypertension, such as pre-eclampsia or cytotoxic therapies. The most characteristic imaging pattern in PRES is the presence of edema involving the white matter of the posterior portions of both cerebral hemispheres, especially the parieto-occipital regions, in a relatively symmetric pattern that spares the calcarine and paramedian parts of the occipital lobes

MATERIALS AND METHODS

Study design - Retrospective Study

Study period - 1 year (May 2014 – May 2015)

Study place - KIMS Hospital and Research Centre, Bangalore

Subjects - Women who presented to Emergency department and already admitted in obstetrics ward for various others reasons (like ceaserean section, FTVD, post partum sterilisation) were included in our study. Diagnosis of PRES was reached by clinical features and MRI brain findings.

RESULTS

We found 9 cases of PRES, the diagnosis of which is established by clinical features and radiologically by MRI. Neuroimaging is essential for the diagnosis of PRES and the radiological abnormalities encountered in PRES are best demonstrated by magnetic resonance imaging (MRI). MRI shows symmetrical white matter edema in the posterior cerebellar hemispheres that particularly involve the parietooccipital regions bilaterally (Hinchey *et al*., 1996). T2-weightedMRI shows areas of hyperintense signal and is thought to capture the images with the best quality, but fluid attenuated inversion recovery (FLAIR) sequences may improve detection of corticalsubcortical areas of injury and help distinguish vasogenic edema from cytotoxic edema. More severe radiological findings are more likely to be observed with more severe clinical pictures (Fugate *et al*.,). The details of the cases are discussed below with the suitable tables.

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Table 1. Age distribution

Age	No. of Patients	Percentage
19– 25 yrs	2	22.2
25 – 30 yrs	5	55.6
30 – 35 yrs	2	22.2

Out of the 9 patients, 55.5% of women were between the age group of 25-30years and 22.2% were between 19-25yrs and 30-35yrs.

Table 2. Parity of the patients

Parity	No. of .Patients	Percentage
Para 1	3	33.3
Para 2	5	55.6
Para 3	0	0
Para 4	1	11.1

Out of 9 patients, primipara was 33.3%, para-2 were 55.5% and para-4 were 11.1%

Table 3. Etiology of pres

Etiology	No. of Patients	Percentage
Hypertensive Disorder of pregnancy	6	66.7
1)Pre-eclampsia	4	
2)Eclampsia	1	
3)HELLP syndrome	1	
Dehydration	1	11.1
Electrolyte imbalance (Hyperkalemia with hyponatremia)	1	11.1
Idiopathic	1	11.1

Out of 9 patients, most common etiology being hypertensive disorder of pregnancy corresponding to 66.7%, which consisted of pre-eclampsia, eclampsia and HELLP syndrome followed by dehydration, electrolyte imbalance and idiopathic which is 11.1% each.

Table 4. Clinical features

Clinical features	No. of Patients	Percentage
Convulsions	9	100
Headache	8	88.9
Transient loss of vision	6	66.7
Behavioural changes	2	22.2
Hypertension	6	66.7

Patient presented with clinical features included headache in 88.9% patients, hypertension in 66.7%, visual disturbances like transient loss of vision in 66.7% and behavioural changes in 22.2% and convulsions in 100% patients. Even though one or two clinical features are predominant, overlapping of features found in many cases.

Table 5. Laboratory findings

Abnormal findings	No. of patients	Percentage
Anaemia	4	44.4
Thrombocytopenia	5	55.6
Altered RFT	3	33.3
Altered LFT	1	11.1
Hyperkalemia with hyponatremia	1	11.1

Abnormal laboratory findings were found in 7 patients out of 9 patients. In two patients dehydration was predominant and no abnormality found in one patient labelled as idiopathic. In other 7 patients many findings were overlapping which included anaemia in 44.4%, thrombocytopenia in 55.6%, abnormal renal parameters in 33.3%, abnormal liver function test in 11.1% and hyperkalemia with hyponatremia in 11.1%.

Table 6. Ventilator support

Ventilator	No. of Patients	Patients
Required	1	11.1
Not required	8	88.9

Out of 9 patients, one patient required ventilator who was referred from peripheral health care center with severe pre-eclampsia and HELLP syndrome, with post-operative day-3 who had undergone cesaerean section in view of impending eclampsia and fetal distress. And there was one mortality out of 9 patients who had come with convulsions and HELLP syndrome who was put on ventilator support as described above.

DISCUSSION

PRES is a recently described clinicoradiologic syndrome that is associated with several medical conditions, including hypertensive encephalopathy and eclampsia. It has been described as clinical findings of headache, visual changes, altered mental status, and seizures (Hinchey *et al*., 1996) in conjunction with radiologic findings of posterior cerebral white matter edema. (Finocchi *et al*., 2005; Lamy *et al*., 2004) Most evident on T2-weighted MRI images, the lesions are hyperintense and located at the gray-white junction, and most often involve the parieto-occipital regions bilaterally. Less frequently the lesions involve the frontal, temporal, and cerebellar regions bilaterally. (Hinchey *et al*., 1996) More severe radiologic findings have been associated with more severe clinical findings in PRES. (Schwartz *et al*., 2000) It is unknown, however, whether imaging before seizure onset would reveal evidence of PRES.

Conclusion

PRES is usually reversible with appropriate treatment. (Schwartz *et al*., 2000; Hinchey *et al*., 1996; Finocchi *et al*., 2005; Striano *et al*., 2005; Schaefer *et al*., 1997) However, it is important to recognize and treat the etiology responsible for PRES, as PRES has been shown to progress from reversible vasogenic edema to irreversible ischemic damage if appropriate treatment is not promptly initiated. Ischemic damage can cause irreversible neurologic sequelae, such as epilepsy, as well as death. (Schaefer *et al*., 1997; Striano *et al*., 2005; Schaefer *et al*., 2003) The reversibility of PRES is due to its underlying pathophysiology, which has been attributed to failure of cerebral autoregulation and endothelial dysfunction. The leading pathophysiologic hypothesis for PRES involves a breakdown of brain vascular autoregulation due to an increase in blood pressure above the patient's baseline level. It is believed that the posterior brain is at greater risk for autoregulation breakdown because it is less extensively innervated, rendering it less able to adjust to blood pressure fluctuations. (Hinchey *et al*., 1996; Finocchi *et al*., 2005) The failure of autoregulation results in vasogenic edema (Hinchey *et al*., 1996; Finocchi *et al*., 2005; Schwartz *et al*., 2000). The presence of endothelial dysfunction decreases the thresholdblood pressure at which vasogenic edema occurs (Hinchey *et al*., 1996; Finocchi *et al*., 2005). For this reason,

vasogenic edema may occur with mildly elevated or normal blood pressure. Blood pressure in eclamptic patients varies, with 20% to 54% of patients having severe hypertension (systolic blood pressure [SBP] > 160 mm Hg or diastolic blood pressure [DBP] > 110 mm Hg), 30% to 60% having mild hypertension (SBP 140–160 mm Hg or DBP 90–110 mm Hg), and 16% having no hypertension (Hinchey *et al*., 1996). Otherwise the patient is given supportive treatment with antiepileptics, anticoagulants if needed, adequate hydration and ventilator support if required. The prognosis of PRES is usually benign. Regardless of etiology, hypertension is a feature in the vast majority of PRES patients. Most investigators believe that hypertensive encephalopathy and preeclampsia share similar mechanisms. Clinical improvement always follows the treatment of elevated blood pressure and other underlying cause.

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