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RESEARCH ARTICLE

POSTERIOR REVERSIBLE ENCEPHALOPATHY SYNDROME IN PREGNANCY

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ABSTRACT

Posterior reversible encephalopathy Syndrome (PRES), is a reversible syndrome of headache altered sensorium, seizures, loss of vision and often associated with hypertension. Preeclampsia and eclampsia are among the most common causes of PRES. Early recognition and treatment of this syndrome is important to prevent permanent neurological sequelae. Neuroimaging shows, bilateral symmetrical subcortical edema in occipitoparietal region. Treatment is primarily supportive and involves correction of underlying cause.

INTRODUCTION

Posterior reversible encephalopathy Syndrome (PRES), a neuroradiological entity was first described by Hinchey in 1996 [1]. It is a syndrome of headache, altered sensorium, seizures, loss of vision and mostly associated with hypertension. It can occur in patients who are taking immuno suppresants and chemotherapeutic agents. Also seen in patients with systemic lupus erythemetosus, sepsis and eclampsia.[2] Diagnosis can be confirmed by Magnetic resonance imaging (MRI). It shows bilateral, symmetrical odema in the parieto occipital region. Brainstem and cerebellum are also involved. Usually the condition is reversible, but early recognition and treatment are important to prevent neurological deficit. The clinical features, diagnostic methods and management of PRES have been discussed here.

MATERIALS AND METHODS

It is a retrospective study conducted in pregnant patients who had been diagnosed to have PRES in our hospital from January 2015 to January 2017. Seven patients had been diagnosed to have PRES after taking CT and MRI. The maternal and neonatal outcome had also been studied.

RESULTS

Of the seven patients, five had eclampsia and emergency caesarean section was done under general anesthesia. Except one case, all cases were nearing term as shown in table 1. These patients were previously diagnosed, as cases of pregnancy induced hypertension and were on antihypertensives.

They had headache, altered sensorium and generalized seizures as shown in table 2. One patient had blurred vision, which improved later. Two patients had nausea and vomiting. All patients were treated with antihypertensives and Magnesium sulfate. Other essential symptomatic measures were taken. All of them were ventilated for 3-4 days and extubated.

Table 1 Time of Manifestation

Gravida	Presentation at Weeks of Gestation	Blood pressure at time of admission mm/hg
Primi	36	170/100
Second	37	160/100
Second	38	180/110
Primi	31	180/100
Second	37	160/100
Second	5th POD	140/90
Primi	6th POD	130/80

Table 2 Clinical Manifestation

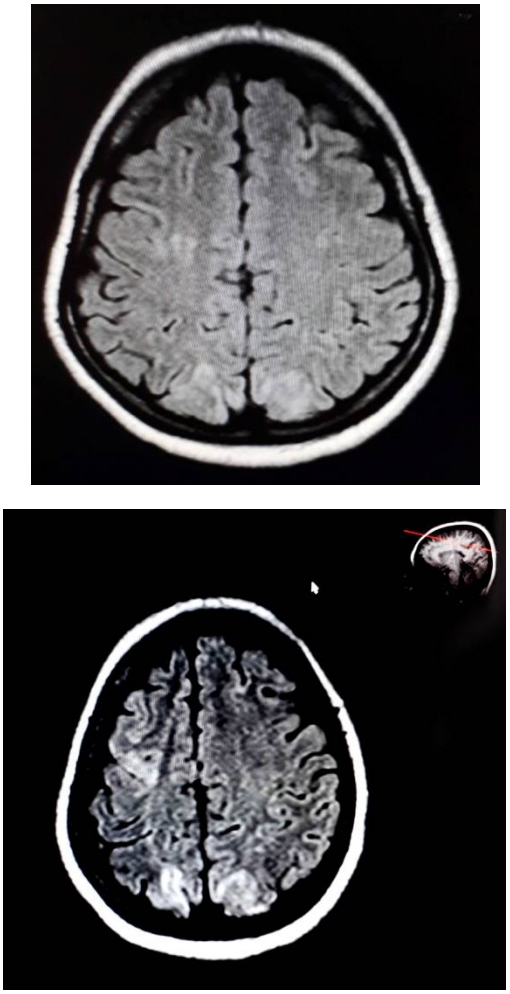
Symptoms	No (7)
Headache	7
Loss of Consciousness	5
Blurred vision	1
Nausea /vomiting	2
Convulsion	7

Other two patients were normotensive and caesarean section was done under spinal anesthesia. Five to six days after surgery they developed headache, photophobia, altered sensorium and status epilepticus. They were intubated and ventilated for one week and supportive treatment was given. In all patients, minimal alteration in renal function and liver function was noted. Hemogram were near normal.

CT brain showed hypodense lesion predominantly involving white matter of parietal and occipital areas and few had cerebellar odema.

Magnetic resonance imaging (MRI) showed bilateral symmetrical subcortical oedema in the parieto occipital region. Cerebral oedema had also been noted in external water shed zone and superior frontal sulcus region.

MRI as shown in fig.



MRI with T2 – FLAIR – weighted images showing bilateral hyperintense lesions in parietal region and external water shed zone

PRES affected primi and second gravid equally. They had headache, nausea, vomiting, photophobia, altered sensorium and generalized seizures. There T2 weighted MRI images show high signal indicating odema in the parieto occipital region. All the cases needed intensive care unit support for 10 to 15 days. No mortality had been reported. Of the seven babies, four were admitted in neonatal care unit. One baby was extremely premature and needed support for long time.

DISCUSSION

Pre eclampsia patients are prone to develop PRES, because of sudden increase in blood pressure. In literature 7-20% of patients with PRES were reported to have Pre eclampsia and eclampsia. Onset of PRES occurred from 28 weeks of gestation to 13 days after delivery.[3]

Exact pathogenesis is not known, however two hypothesis have been postulated. 1. The hyperperfusion theory and 2. The cytotoxic theory.

Of the two, the most accepted being the hyperperfusion theory. According to this theory, cerebral autoregulation is altered when there is sudden rise in blood pressure. Lack of sympathetic innervation of posterior vessels compared to Anterior,[4] results in elevated capillary filteration pressures. It increases the blood brain barrier permeability and leads to cerebral odema.

PRES can also occur in normotensive patients, which explains the cytotoxic theory. Activation of immune system leads to release of various proinflammatory mediators from the endothelial cells. It results in vasoconstriction and hypoperfusion, which leads to cerebral odema [6].

Clinical Presentation

Pre eclampsia and eclampsia are diagnosed based on clinical and laboratory criteria, whereas PRES is diagnosed radiographically.

Presentation varies from headache, nausea, seizures and often associated with hypertension.[7] About 70-80% of patients have moderate to severe hypertension and in 20-30% of cases, blood pressure is normal or minimally elevated[8]. Bartynski[2] *et al* reported seizures (70%) as the most common presentation. Incidence of focal neurological deficits is low.

Histopathology in PRES

Activated astrocytes, scattered macrophages have been noted without inflammation. Evidence of demyelination, neuronal anoxic damage and laminar necrosis have been seen with ischemia [9].

Magnetic resonance imaging (MRI)

Magnetic resonance imaging (MRI) is the investigation of choice for PRES. It typically shows bilateral symmetrical subcortical oedema in occipito parietal region. However oedema can occur in post frontal area, brainstem, basal ganglia and cerebellum[1]. Fluid attenuated inversion recovery images help in better visualization of cortical and subcortical odema. Bartynsky had described the image patterns of MRI as follows[2],

1. Holo hemispheric watershed lesion (22.8%)
2. Superior frontal sulcus pattern (27.2%)
3. Dominant parieto- occipital pattern (22.1%)
4. Partial or asymmetric expression of primary patterns (28%)

Advanced Imaging

Catheter angiography shows vasoconstriction and vasodilation of vessels as string of beads[10]. Diffusion weighted MRI is the study of choice in PRES to differentiate between vasogenic and cytotoxic oedema[11,12,13]. Apparent diffusion coefficient mapping is of great utility in differentiation of the two.

Management

In eclampsia with PRES, the definitive treatment consists of immediate delivery [14] (i.e. Induction/ Caesarean section) and

control of blood pressure. To reduce mean arterial pressure, an initial reduction of not more than 20-25% within the first 2 hours is advisable, as rapid reduction worsens the cerebral perfusion[15,16]. Magnesium sulfate is the drug of choice in eclampsia with proven decreased maternal mortality and improved infant outcome [17]. Continuous hemodynamic, cardiac, respiratory and neurological monitoring is required. Other symptomatic treatment is essential.

Complications

Cerebral infarction[3] is the earliest sign of irreversibility in PRES. Although rare, cerebral hemorrhage[18], and herniation[19] can also occur. A case of obstructive hydrocephalus[20] after PRES had also been reported.

CONCLUSION

PRES is a diagnostic challenge, due to non specific clinical presentation, especially during pregnancy. It can also occur even after child birth. Early recognition and resolution of underlying cause is the keystone of management. Futher studies are needed to identify the factors of adverse prognostic significance and to develop neuro protective strategies.

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