Original Research Article

Clinico-radiological spectrum of obstetric patients with posterior reversible encephalopathy syndrome in a tertiary care hospital

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ABSTRACT

Background: Posterior reversible encephalopathy syndrome (PRES) is a disorder that is not uncommon in pregnancy induced hypertension. We have studied the clinico radiological profile of such patients to understand history, symptomatology and neuro imaging findings of this entity.

Methods: The present study included 20 patients of PRES among the inpatients of a tertiary care hospital in south India from January to March 2020.

Results: Mean age 25.0 years. Most common symptoms included seizures and headache followed by vomiting, visual disturbances.9 patients (45%) had parieto-occipital signal changes on neuro imaging. Magnetic imaging resonance (MRI) (n=20) revealed involvement of atypical sites viz. frontal (30%), temporal (20%), cerebellum (20%), basal ganglia (20%), deep white matter (30%); and brainstem (10%).Diffusion restriction was seen in 40% patients.

Conclusions: Atypical MRI presentations of PRES are common and there is a need to consider a strong possibility for the diagnosis of PRES.

Keywords: Posterior reversible encephalopathy syndrome, Eclampsia, Hypertension

INTRODUCTION

Posterior reversible encephalopathy syndrome is a condition that predominantly affects the cerebral white matter. Oedematous lesions particularly involve the posterior parietal and occipital lobes, and may spread to basal ganglia, brain stem, and cerebellum.\(^1\) This rapidly evolving neurological condition is clinically characterised by headache, nausea and vomiting, seizures, visual disturbances, altered sensorium, and occasionally focal neurological deficit.\(^2\) Posterior leukoencephalopathy syndrome is often associated with an abrupt increase in blood pressure and is usually seen in patients with eclampsia, renal disease, and hypertensive encephalopathy. It is also seen in the patients treated with cytotoxic and immunosuppressive drugs. The neuroimaging findings on magnetic resonance imaging (MRI) of brain often reveals areas of vasogenic edema as hypointense areas on the T1-weighted MR images and hyperintense areas on the T2-weighted/fluid-attenuated inversion recovery (FLAIR) MR sequences, with lack of diffusion restriction.\(^3,4\) Early recognition of this condition is of paramount importance because prompt control of blood pressure or withdrawal of immunosuppressive agents will cause reversal of the syndrome. Delay in the diagnosis and treatment can result in permanent damage to affected brain tissues.

Objectives of the study

To study etiological, clinical, and radiological profile of PRES in Obstetric patients in South India. To identify
atypical patterns of presentation of PRES and promote early diagnosis and treatment for a favourable outcome.

**METHODS**

The current cross sectional observational was conducted at Govt. Rajaji Hospital, Madurai from January 2020 to March 2020. During this period 20 antenatal patients with PRES were identified from the inpatient department of obstetrics and gynaecology and enrolled in the study. The study qualified ethical standards and written informed consent was obtained from all the patients.

Cases diagnosed as PRES were included in the study after ruling out exclusion criteria. Clinical details were collected from all cases.

The inclusion criteria for the study being- age >18 years, variable combination of clinical manifestations suggestive of PRES: seizure activity, consciousness impairment, headaches, visual abnormalities, nausea/ vomiting, and focal neurological signs, brain imaging consistent with diagnosis of PRES: two neuro physicians and an experienced neuroradiologist should have a consensus that MRI signal abnormalities are consistent with PRES and patients willing to give written informed consent.

Care was taken to exclude conditions which may mimic PRES, including but not limited to encephalitis, acute disseminated encephalomyelitis, central nervous system vasculitis, cerebral venous sinus thrombosis, mitochondrial myopathy, encephalopathy, lactic acidosis, and stroke-like episode, and cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy, through relevant clinical and laboratory data.

Once enrolled, detailed history was obtained and thorough general physical, systemic and neurological examination was carried out. All the relevant clinical and radiological data were noted on a predesigned proforma. Pregnancy Induced Hypertension was diagnosed on the basis of American College of Obstetrics and Gynaecology guidelines. All the patients underwent detailed laboratory testing, MRI brain imaging as well as assessment by neurologist.

Imaging studies were reviewed by an experienced radiologist. All patients underwent MRI imaging with T2 weighted, T2 FLAIR, T1-weighted, and diffusion-weighted imaging sequences. Additional sequences like contrast-enhanced T1-weighted and time-of-flight intracranial MR angiography, were done whenever deemed necessary.

A diagnosis of PRES was considered whenever typical imaging findings were seen as described by Bartynski and Boardmann: dominant parieto-occipital (PO) pattern, holohemispheric watershed pattern, dominant superior frontal sulcus pattern, asymmetrical or partial expression (A/P) of the three primary patterns.

The imaging findings were also described according to their site, i.e., frontal, parietal, occipital, temporal, deep white matter, basal ganglia/thalami, brainstem, and cerebellum as well imaging characteristics.

Data was processed using Microsoft Excel and analysed using statistics of frequency percentage mean and standard deviation. Continuous parametric variables, for example, age, blood pressure, pulse rate, complete biochemistry, complete hemogram, clinical symptoms with duration, etc., were analyzed by applying analysis of variance test, whereas skewed variables were analyzed using Mann–Whitney U test.

**RESULTS**

**Age and parity**

Mean age was 25.0 (range 20–30 years). Maximum patients were in the 3rd decade of age. Thus, PRES is a disorder of young adults. 15 of the study patients were primigravida, one was 3rd gravida and 4 were 2nd gravida. 18 (90%) were antenatal cases while 2 (10%) were postnatal.

**Clinical features**

In the current study, the most common presentation was seizures and headache followed by vomiting, visual disturbances. 3 (15%) patients had altered sensorium. Headache persisted for >2 weeks among 9 (45%) individuals. All patients had seizures and received antiepileptic drugs in addition to Pritchard regimen. All patients improved spontaneously with meticulous control of blood pressure. Hypertension was noted in all patients. Mean systolic blood pressure (SBP) and diastolic blood pressure (DBP) pressures were 162 mm Hg and 96 mm Hg whereas peak SBP and DBP were 190 mmHg and 110 mmHg, respectively. The clinical feature results are summarized in Table 1.

**Radiological features**

4 patients had MRI changes in the parieto regions similar to the changes which are classically stated in the literature. The MRI findings are summarized in Table 2. The most striking feature noted in our study group was the involvement of atypical sites on MRI i.e. involvement of areas beyond the classically described posterior circulation territory. Atypical sites of signal changes included frontal (35%), temporal (15%), cerebellum (10%), basal ganglia (15%), deep white matter (15%), and brainstem (5%).

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Table 1: Clinical feature summary in PRES.

| Age | Sex | Headache | Seizures | Vomiting | Visual disturbance | Altered sensorium | Systemic disease | Etiology       |
|-----|-----|----------|----------|----------|-------------------|------------------|-----------------|----------------|
| 25  | F   | +        | +        | -        | -                 | -                | AP eclampsia    | PIH            |
| 26  | F   | +        | +        | -        | -                 | +                | PP eclampsia,SLE| PIH, SLE       |
| 30  | F   | +        | +        | -        | -                 | -                | AP eclampsia    | PIH            |
| 23  | F   | +        | +        | -        | -                 | -                | AP eclampsia    | PIH            |
| 25  | F   | +        | +        | -        | -                 | -                | AP eclampsia    | PIH            |
| 21  | F   | +        | +        | -        | -                 | -                | AP eclampsia    | PIH            |
| 20  | F   | +        | +        | -        | +                 | -                | AP eclampsia    | PIH            |
| 28  | F   | +        | +        | -        | -                 | -                | PP eclampsia    | PIH            |
| 30  | F   | +        | +        | -        | -                 | -                | AP eclampsia    | PIH            |
| 20  | F   | +        | -        | -        | -                 | -                | RHD,AP eclampsia| PIH            |
| 22  | F   | +        | -        | -        | -                 | -                | AP eclampsia    | PIH            |
| 25  | F   | +        | +        | -        | -                 | +                | AP eclampsia    | PIH            |
| 26  | F   | +        | -        | -        | +                 | -                | Imminent eclampsia| PIH        |
| 28  | F   | +        | +        | -        | -                 | -                | AP eclampsia    | PIH            |
| 27  | F   | +        | +        | -        | -                 | -                | AP eclampsia    | PIH            |
| 28  | F   | +        | +        | -        | -                 | -                | AP eclampsia    | PIH            |
| 26  | F   | +        | +        | -        | -                 | -                | AP eclampsia    | PIH            |
| 24  | F   | +        | +        | -        | -                 | -                | AP eclampsia    | PIH            |

Table 2: MRI changes in PRES.

| Location of changes                                                                 | Diffusion                      | MR angiography                  |
|------------------------------------------------------------------------------------|--------------------------------|---------------------------------|
| Frontal, occipital                                                                  | Restricted in centrum semiovale| Normal                          |
| Bilateral parieto occipital regions                                                 | Normal                          | Normal                          |
| Bilateral posterior temporal and occipital regions                                  | Normal                          | Normal                          |
| Bilateral posterior temporal, occipital cerebellar hemisphere and brainstem regions | Normal                          | Normal                          |
| Bilateral cerebral subcortical region, bilateralcapsuloganglionic regions, bilateral cerebellar hemispheres | Restricted in bilateral cerebellar hemispheres | Normal                          |
| Bilateral parieto occipital regions                                                 | Normal                          | Normal                          |
| Bilateral parieto occipital regions, right temporal lobe                             | Restricted in bilateral cerebellar hemispheres | Normal                          |
| Bilateral parietal regions                                                           | Normal                          | Normal                          |
| Bilateral parietal regions, frontal lobe, left external capsule and right caudate nucleus | Restricted | Normal                          |
| Bilateral frontal, right centrum semiovale and corona radiata                      | Restricted in bilateral cerebellar hemispheres | Normal                          |
| Bilateral frontal, parietal and occipital regions                                   | Restricted in bilateral cerebellar hemispheres | Normal                          |
| Bilateral posterior parietal regions                                                | Normal                          | Hypoplastic right vertebral artery |
| Bilateral parieto occipital regions, bilateral high frontal regions                 | Minimal diffusion restriction    | Normal                          |
| Bilateral parieto occipital regions and bilateral cerebellar hemispheres            | Minimal diffusion restriction    | Normal                          |
| Left fronto parietal region                                                         | Normal                          | Irregular and beaded appearance of bilateral ICA and MCA |
| Bilateral parietal regions and left capsuloganglionic regions                       | Normal                          | Normal                          |
| Bilateral parieto occipital regions                                                 | Normal                          | Normal                          |

Continued.
Diffusion restriction in posterior reversible encephalopathy syndrome:

A total 7 (35%) of the 20 patients had restricted diffusion on MR imaging. The sites included occipital and parietal regions, frontal lobe, temporal lobe, centrum semiovale, corona radiata and cerebellar hemispheres. One patient also showed vasospasm in bilateral internal carotid and middle cerebral arteries.

| Location of changes                      | Diffusion     | MR angiography |
|------------------------------------------|---------------|----------------|
| Bilateral frontal and occipital regions  | Normal        | Normal         |
| Bilateral parieto occipital regions      | Normal        | Normal         |
| Bilateral parieto occipital regions      | Normal        | Normal         |

DISCUSSION

In the current study, we observed 20 patients of PRES with complete clinical, ophthalmological and MRI data at presentation to delineate its clinico-radiological profile in a comprehensive manner.

Demographic profile and clinical features

In our study, PRES predominantly affects antepartum female population. This is in concurrence with the study by Cho who reported that PRES is associated with the pregnancy, in the peripartum period and presented with seizures of generalized tonic-clonic type, headache and visual disturbances. Our study is also comparable with Pedraza et al who reported that PRES is most commonly associated with hypertension, pre-eclampsia-eclampsia, and HELLP syndrome.

Etiology of PRES

In the current study, all patients had pregnancy induced hypertension of which 18 (90%) had antepartum eclampsia and 2 (10%) had postpartum eclampsia. One among the postpartum eclamptic had Systemic Lupus Erythematosus, and one among the antepartum eclampsia had rheumatic heart disease additionally.
In the present study the most common clinical presentation was seizures and headache which is similar to that of previous studies. However, visual symptoms were reported less commonly by our patients.

**Neuroimaging findings**

The most common location in the neuroimaging in this study was the parieto-occipital region similar to previous studies. Among the atypical locations were frontal lobe, temporal lobe, basal ganglia and cerebellum. These findings are comparable with other studies.

In addition to involvement of atypical sites, we also noted presence of diffusion restriction and vasoconstriction on MRI, features which are considered atypical for PRES.

**Limitations**

The present study was a cross sectional observational study hence we were unable to assess outcome and follow up. The sample size was small, hence exact prevalence PRES could not be ascertained. The patients enrolled in the study might not represent general population.

**CONCLUSION**

The present study delineates the clinico-radiological profile of PRES. Atypical MRI presentations are common and we need to rule out PRES in appropriate clinical settings. Early diagnosis and prompt control of blood pressure paves way for a favourable outcome.

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**Ethical approval:** The study was approved by the Institutional Ethics Committee

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