ORIGINAL ARTICLE

MAGNETIC RESONANCE IMAGING FEATURES OF POSTERIOR REVERSIBLE ENCEPHALOPATHY SYNDROME (PRES)- A HOSPITAL BASED STUDY

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PJR October - December 2016; 26(4): 312-316

Introduction

Posterior reversible encephalopathy syndrome (PRES) is a neurotoxic syndrome and a neuroradiological entity. Patients suffering from this syndrome present with headache, altered mental status, seizures and visual disturbances etc.[1] There are a number of risk conditions for this syndrome including pre eclampsia/ eclampsia, severe hypertension, acute or chronic renal failure, chemotherapy, sepsis and bone marrow or organ transplantation.[2-5]

The clinical presentations of this syndrome may be non-specific but MRI features are very distinctive. The term PRES itself is a misnomer as the lesions are not limited to the occipital lobes but can also involve the parietal and temporal lobes, basal ganglia, brain stem, deep white matter and cerebellum.[6] PRES lesions may show a diverse pattern of distribution but usually having distinct symmetrical or near asymmetrical pattern. The typical manifestations are focal areas of cortical/ subcortical and white matter vasogenic edema which manifest as areas of hyperintensity on T2W and FLAIR sequences.[7,8] These changes are reversible if promptly diagnosed and treated leading to clinical improvement. Although these areas of vasogenic edema may also be seen in computerized tomography (CT) but MRI is the investigation of choice with T2W, FLAIR, DW and ADC sequences.[9,10]

The purpose of this study is to identify the distinct MR imaging features and lesion distribution pattern of this reversible syndrome in the southwest region of Pakistan and to compare it with other national and international studies.

Materials and Methods

The study has been done in a tertiary care hospital and all imaging was done on a 1.5 T Phillips Achieva MRI scanner. Patients were enrolled and followed up from 1st December 2014 to 29th February 2016 (Fifteen months).

The clinical symptoms / signs of patients like headache, altered mental status, neck rigidity, visual disturbances and seizures were recorded. The associated risk factors for this syndrome like preeclampsia/ eclampsia, renal failure, sepsis, severe hypertension and bone marrow or organ transplantation etc. were also noted.

Inclusion criteria included patients with any clinical neurological presentation who underwent MRI and the findings were supportive of the diagnosis of PRES. All patients whowere clinically suspected of having PRES but with no confirmatory features on MRI were excluded from the study. Patients, in which follow up could not be done and patients suffering from claustrophobia or any contraindication to MR examination were also excluded from the study.

The MRI sequences obtained were sagittal and coronal T1W, T2W, FLAIR, DWI and ADC mapping. Each study was analyzed by two consultant radiologists in a double blind interpretation. No contrast enhanced imaging was done. The expected demonstration of focal areas of cortical or subcortical or white

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Submitted 2 April 2016, Accepted 31 May 2016

matter vasogenic edema with symmetrical or near symmetrical distribution was considered diagnostic of PRES. Patients having asymmetrical or unilateral involvement suggestive of PRES were also included. The target finding was analyzed and dominant distribution patterns were categorized into respective groups by mutual consensus of radiologists. The patients were all followed up clinically with 6 patients also undergoing follow up MRI examinations.

Results

We included twenty six patients in our study who met the clinical and radiological inclusion criteria. Patient ages ranged from 23 to 65 years with mean age being 35.9 years. Among them, 19 were female and seven were male and the male to female ratio was 1: 2.7. Among these patients, the noted risk conditions for posterior reversible encephalopathy syndrome were preeclampsia/ eclampsia (50%), severe hypertension (23%), renal failure/ uremia (15%), septicemia (8%) and one patient with renal transplant (4%) (Fig.1). The main clinical presentation were altered mental status (35%), headache (31%), seizures (23%) and visual disturbances (11%) (Fig. 2).

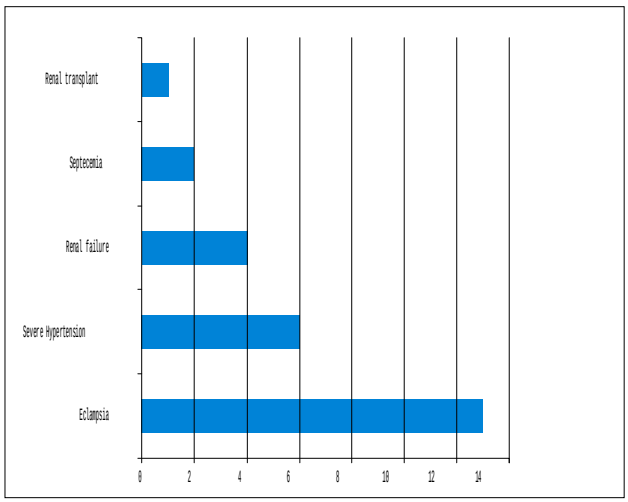

Figure 1: Associated risk conditions of patient (n=26)

The target findings of focal cortical/ sub cortical and white matter vasogenic edema were noted and dominant distribution pattern was recorded in respective groups. The lesions were occipito-parietal in fifteen patients (58%),temporo-occipital in five patients (19%),

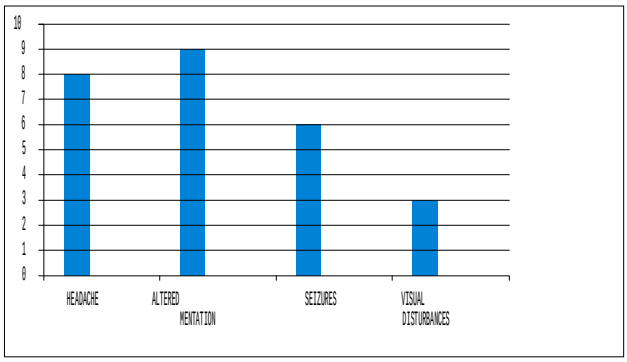

Figure 2: Clinical presentation of patients (n=26)

frontal in four patients (15%) and in the basal ganglia/ deep white matter including external or internal capsules in two patients (8%) (Fig. 3).

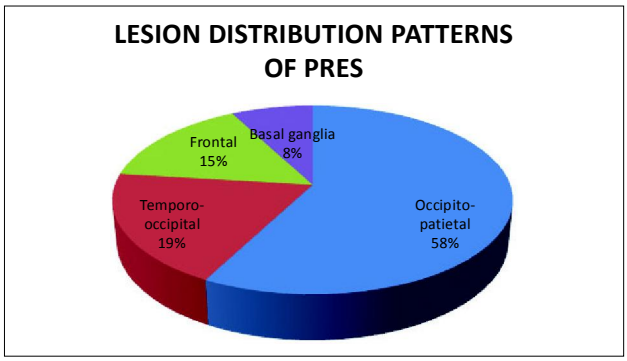

Figure 3: Lesion Distribution pattern in study patients (n=26)

In twenty four (92%) patients lesions were bilateral either symmetrical or nearly symmetrical and in two (8%) patients lesions were unilateral. No cerebellar involvement was noted in any patient in our study. Small hemorrhages were noted in lesions in two of our patients. Restricted diffusion was noted in one lesion in one of our patient. Follow up MRI were done around four weeks in six patients (23%) which showed complete or near complete regression of lesions. Clinical follow was done in twenty patients (77%) which showed clinical recovery on subsequent follow up visits. None of our patients had any contraindication to MRI examination like intracerebral clips or pace-maker etc).

Discussion

This syndrome is a misnomer as it does not affect only posterior part of brain but other areas like frontal/ temporal lobes, basal ganglia, brain stem, deep white

matter and cerebellum.[6] Other names for this are reversible posterior encephalopathy syndrome and hypertensive encephalopathy. PRES is a neurotoxic state. This is a reversible condition if timely diagnosed and treated but delay in diagnosis may lead to permanent neurological deficits and even death.[11]

It is considered that PRES occurs either due to failure of cerebral blood flow autoregulation or endothelial dysfunction in cerebral vessels. Failure of autoregulation occurs due to acute change in blood pressure leading to hyperperfusion and endothelial damage so causing vasogenic edema.[12,13] When endothelial dysfunction is the cause, it leads to vasoconstriction and ischemia causing vasogenic edema.

There are several conditions that can act as a triggering factor for PRES including preeclampsia, eclampsia, renal failure, acute glomerulonephritis, thrombocytopenic purpura, SLE, scleroderma, sepsis, bone marrow or organ transplantation etc. In our study we found eclampsia as commonest risk factor followed by severe hypertension. PRES usually presents with headache, altered mental status, seizures and visual disturbances etc. In our study altered mental status was the most common presentation of this syndrome followed by headache.

The most dominant pattern of distribution is occipito-parietal,[7,9] but there can be involvement of diverse areas of the cerebrum, cerebellum or brainstem. What ever the distribution it is important to diagnose this condition promptly as the condition is reversible if its treatment is instituted early. Delay may lead to permanent neurological damage and death.

PRES lesions can also be depicted on computerized tomography (CT) as focal hypodense areas but CT may be normal in early cases.[2] MRI is the investigation of choice. The vasogenic edema in PRES can be well differentiated from other conditions like infarctions / hemorrhages on different sequences of MRI.[13] This ability of MRI in differentiating vasogenic edema from other neurological pathologies is vital in early diagnosis of PRES. MRI presentation of PRES is usually characteristic and is so life saving.

On MRI, PRES lesions are hypointense on T1W and hyperintense on T2W / FLAIR sequences and show facilitated diffusion on DWI and ADC[7,9,11] (Fig. 4 to 7). These lesions are typically symmetrical or near symmetrical and unilateral lesions are rare. In our study symmetrical or near symmetrical lesions were seen in 92% and the dominant pattern was occipito-parietal.

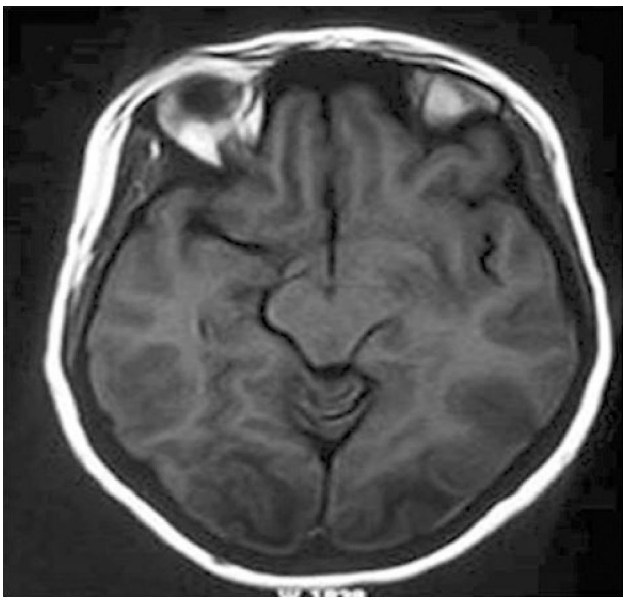

Figure 4: T1weighted MR images showing hypointense PRES lesions in temporo-occipital regions .

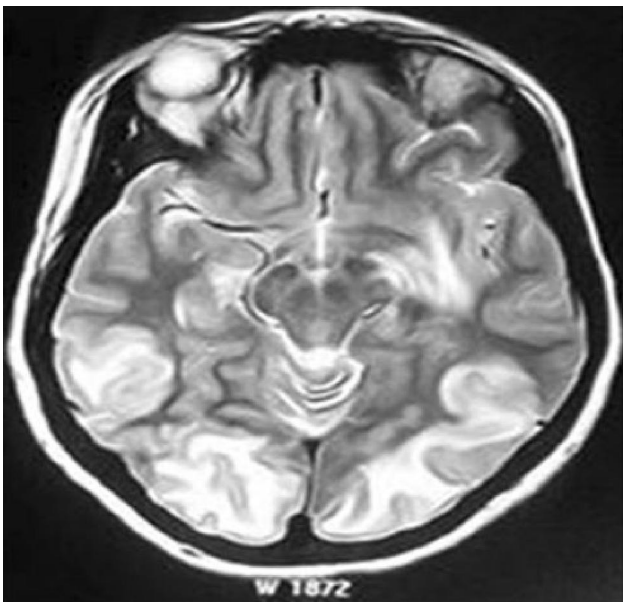

Figure 5: T2 weighted MR image showing hyperintense focal lesions of similar patient in Fig 4.

Magnetic resonance angiography (MRA) shows diffuse or focal vasoconstrictions or vessel pruning in PRES[14] whereas MRV remains unremarkable. MR spectroscopy will show reduction of the N-acetylasparatate peak, choline peak and N-acetylasparatatecreatinine ratio in the involved areas.[15]

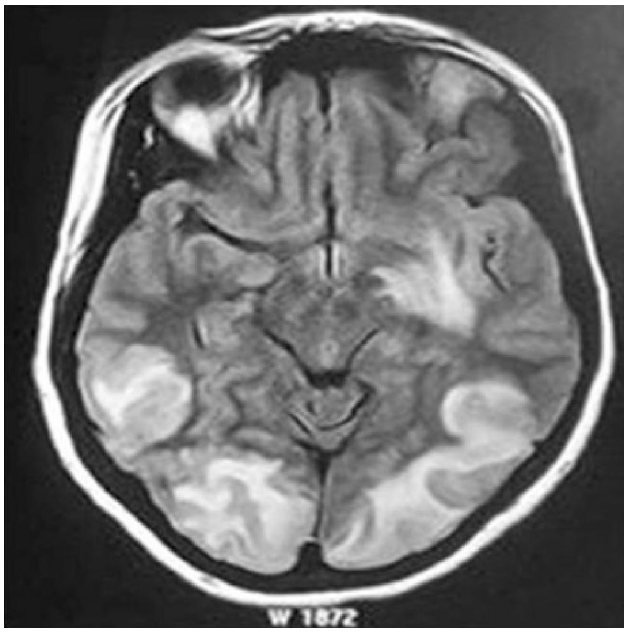

Figure 6: MR FLAIR image showing focal hyperintese PRES lesions in similar patient of Fig 4.

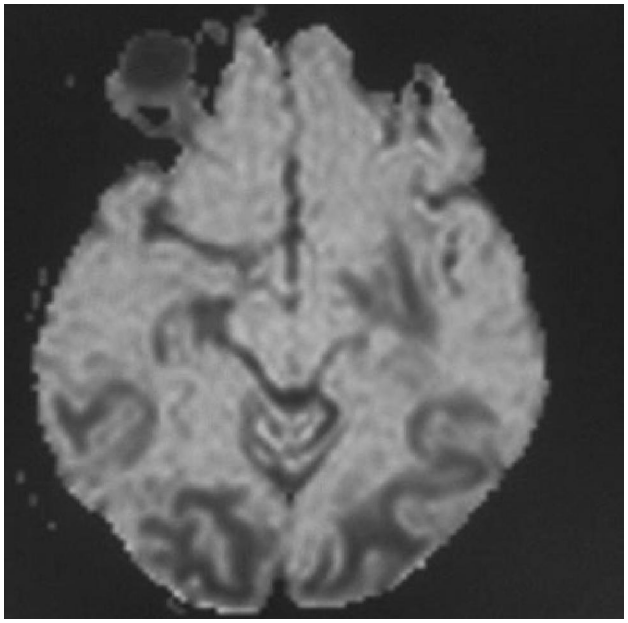

Figure 7: DW image showing facilitated diffusion of similar lesions in Fig 4.

A study was done by W.S. Bartynski and J.F. Boardman in Presbyterian university hospital Pittsburg, PA.They analyzed MR imaging of 114 patients of PRES. They found PRES lesions in occipito-parietal region in 98% cases, in frontal lobes 68% cases, in inferior temporal lobes in 40%, in cerebellum in 30%, in basal ganglia in 14%, in brain stem in 13% and in deep white matter in 18%. They found partial and asymmetrical lesions in 28%.[9] Our study was consonant with their in the occipito-parietal distributions but in contrast we observed no cerebellar or brainstem lesions. Asymmetrical and partial distribution of lesions is also rare in our study as in above mentioned study. M. McKinney and colleagues did a study in University of Minnesota Medical Center, Minneapolis, USA and concluded that most of PRES lesions were in parietooccipital region (98.7%) in their study followed by posterior frontal, temporal, cerebellum, brain stem and basal ganglia.[6]

Another study was done by Noha Mohammad Abdel Maboud Ibrahim and Manal E badawy in Tanta university Hospital, Tanta Egypt. The study was done in 22 pregnant patients suffering from PRES. They also noted maximum PRES lesions in occipito-parietal regions in 81.8%.[16] This is in agreement with our study. They found cerebellar lesions in 18% which is contrary to our study. A local study was done by Rohana Naqi and Muhammad Azeemuddin in Dow university of Health sciences karachi. They found maximum lesions in occipito-parietal region in 41.6% followed by deep white matter lesions in 25%, and frontal, temporal, cerebellar and basal ganglia lesions in 8.3% each.[17]

When we compare our study with all above mentioned studies, we noted one common thing that occipito-parietal pattern of PRES lesions is the most common pattern in all studies. So, we can say that this typical pattern is similar in incidence all over the world. We did not find any cerebellar lesions in our study patients and basal ganglia lesions in only two patients. It is possible that basal ganglia and infratentorial distribution of PRES lesions is less frequent in this part of world as compared to rest of the world. We found imaging and clinical improvement in all our study patients which suggests that it is true that PRES is reversible condition if early diagnosed and promptly treated. We also found the fact that MRI has the vital role in its early diagnosis.

Conclusion

PRES is a clinical dilemma and shows diversity in its lesion distribution on imaging but having distinctive MR imaging pattern of symmetrical or near symmetri-

cal distribution of focal vasogenic edema in brain, puts MRI as a choice investigation in diagnosing this potentially reversible condition. So, MRI of all clinically suspected cases should be done for prompt diagnosis and management to save patients from permanent neurological deficit and death.

References

1. Lee VH, Wijdicks EF, Manno EM, Rabinstein AA. Arch. Clinical spectrum of reversible posterior leukoencephalopathy syndrome. Neurol. 2008 Feb; **65(2):** 205-10.

2. Roth C, Ferbert A. The posterior reversible encephalopathy syndrome: what's certain, what's new? PractNeurol 2011; **11:** 136-44.

3. Hinchey J, Chaves C, Appignani B, Breen J, Pao L, Wang A, et al. A reversible posterior leukoencephalopathy syndrome. N Engl J Med 1996; **334:** 494-500.

4. Fugate JE, Claassen DO, Cloft HJ, Kallmes DF, Kozak OS, Rabinstein AA. Posterior reversible encephalopathy syndrome: associated clnical and radiologic findings. Mayo ClinProc 2010; **85:** 427-32.

5. Jorge S, Lopes JA, De Almeida E, Martins Prata M. Posterior reversible encephalopathy syndrome (PRES) and chronic kidney disease [Spanish]. Nefrologia 2007; **27(5):** 650.

6. Mc Kinney AM, Short J, Truwit CL, McKinney ZJ, Kozak OS, SantaCruz KS, et al. Posterior reversible encephalopathy syndrome: incidence of atypical regions of involvement and imaging findings. AJR 2007; **189:** 904-12.

7. Bartynski WS. Posterior reversible encephalopathy syndrome, part 1: fundamental imaging and clinical features. AJNR Am J Neuroradiol 2008; **29:** 1036-42.

8. Zidan D, Hendawy S. Acute neurologic disorders during pregnancy and peurperium: a challenging MRI issue. Egypt J RadiolNucl Med. 2007; **38(3):** 1085-96.

9. Bartynski WS, Boardman JF. Distinct imaging patterns and lesion distribution in posterior reversible encephalopathy syndrome. AJNR 2007; **28:** 1320-7

10. Covarrubias DJ, Luetmer PH, Campeau NG. Posterior reversible encephalopathy syndrome: prognostic utility of quantitative diffusion-weighted MR images. AJNR Am J Neuroradiol 2002; **23:** 1038-48.

11. Lamy C, Oppenheim C, Meder JF, Mas JL. Neuroimaging in posterior reversible encephalopathy syndrome. J Neuroimaging. 2004; **14:** 89-96.

12. Schwartz RB, Mulkern RV, Gudbjartsson H, Jolesz F. Diffusion-weighted MR imaging in hypertensive encephalopathy: Clues to pathogenesis. Am J Neuroradiol. 1998; **19:** 859-862.

13. Mukherjee P, McKinstry RC. Reversible posterior leukoencephalopathy syndrome: Evaluation with diffusion-tensor MR imaging. Radiology. 2001; **219:** 756-65.

14. Bartynski WS, Boardman JF, Zeigler ZR, Shadduck RK, Lister J. Posterior reversible encephalopathy syndrome in infection, sepsis, and shock. AJNR AmJ Neuroradiol 2006; **27:** 2179-90.

15. Eichler FS, Wang P, Wityk RJ, Beauchamp NJ Jr, Barker PB. Diffuse metabolic abnormalities in reversible posterior leukoencephalopathy syndrome. AJNR Am J Neuroradiol 2002; **23:** 833-7.

16. Noha Mohammad Abdel Maboud Ibrahim and Manal E badawy. MRI imaging of posterior reversible encephalopathy syndrome associated with pregnancy. The Egyptian Journal of Radiology and Nuclear Medicine 2014; **45:** 505-10.

17. Rohana Naqi and Muhammad Azeemuddin. Posterior reversible encephalopathy syndrome. J Pak Med Assoc 2012: **62(7):** 657-60.