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ORIGINAL ARTICLE

MRI imaging of posterior reversible encephalopathy syndrome associated with pregnancy



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KEYWORDS	Abstract Purpose: Our purpose is to characterize MRI, and diffusion-weighted imaging (DWI)
PRES;	findings in pregnant patients who were identified clinically to have PRES. We study the conversion
Posterior reversible encepha-	of reversible vasogenic edema to irreversible cytotoxic edema and predict the progression to infarc-
lopathy syndrome;	tion.
lopathy syndrome; MRI	 Patients and methods: Twenty two pregnant females, aged between 20 and 46 years with gestational age between 20 and 40 weeks of gestation and with neurological manifestations had undergone conventional MRI, diffusion weighted image study, and ADC map. Results: Lesions were mainly affecting the parieto-occipital regions, symmetrical or slightly asymmetrical distribution of the lesions in both cerebral hemispheres was found in most cases. The MRI findings in all the twenty two patients were: abnormal low SI in T1 WI, abnormal high SI on T2 and FLAIR WI. In DWI, hyperintensity with hyperintensity in ADC map was seen in 15 patients, hyperintensity with hypointensity in ADC map in 3 patients.
	<i>Conclusion:</i> The diagnosis of PRES has important therapeutic and prognostic value. The use of diffusion-weighted imaging and ADC maps allows an earlier and clearer differentiation of cytotoxic and vasogenic edema, which can predict the development of infarction.
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1. Introduction

Posterior reversible encephalopathy syndrome (PRES) is a potentially devastating neurologic syndrome characterized by rapidly progressive signs and symptoms, including headache, seizures, consciousness disturbance, and/or visual disturbance (1). Extensive white-matter changes suggestive of posterior cerebral edema are also present. This syndrome was first named reversible posterior leukoencephalopathy syndrome, but it has

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Table 1Clinical presentations of the patients.

Clinical symptoms		No.	%
Headache		20	90.00
Decreased consciousness		17	77.50
Nausea and vomiting		13	60.00
Seizure		11	50.00
Coma		6	30.00
Loss of vision		4	10.00
Chi-square	X^2	29.744	
	P-value	< 0.001	

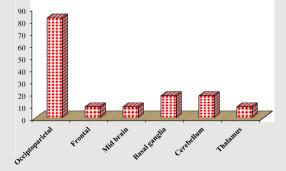
also been known by many other names including PRES, hyper-perfusion encephalopathy, brain capillary leak syndrome, and hypertensive encephalopathy (2). Eclampsia as well as several other pathologic entities which include severe hypercalcemia, thrombocytopenic syndromes, Henoch-Schönlein purpura, hemolytic uremic syndrome, amyloid angiopathy, systemic lupus erythematous, acute glomerulonephritis, various causes of renal failure, acute intermittent porphoria and immunosuppressive medications such as cyclosporine, and various anti neoplastic agents may result in the posterior reversible encephalopathy syndrome (PRES) (3). This syndrome is a variant of hypertensive encephalopathy, both hypertensive encephalopathy and PRES can arise from an acute elevation in blood pressure that overcomes the myogenic vasoconstriction of cerebral arteries and arterioles causing loss of autoregulatory capacity, BBB disruption, and vasogenic edema (4). The difference between hypertensive encephalopathy and PRES is that PRES can develop without a significant elevation in blood pressure (5). It is not entirely known why PRES favors the posterior circulation, but this may arise from a relative lack of sympathetic innervations at the level of the arterioles

Table 2 Classification of patients according to blood pressure.

		Ν	%
Hypertensive		18	81.8
Normotensive		4	18.1
Total		22	100.00
Chi-square	X^2	4.225	
	<i>P</i> -value	0.039	

Table 3The location of PRES.

Location of abnormal SI		No.	%
Occipto-parietal		18	81.82
Frontal		2	9.09
Mid brain		2	9.09
Basal ganglia		4	18.18
Cerebellum		4	18.18
Thalamus		2	9.09
Chi-square	X^2	18.500	
-	<i>P</i> -value	0.0024	



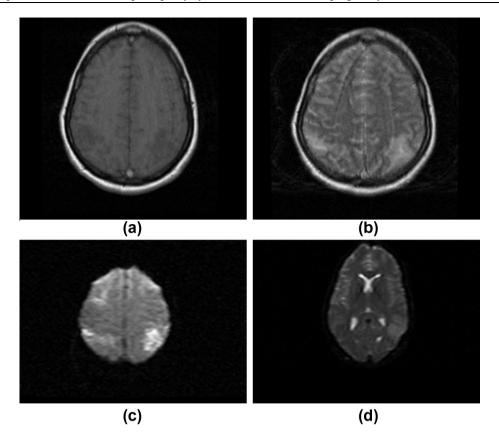


Fig. 1 (a) T1 WI showing bilateral and symmetrical occiopitoparietal low SI. (b) T2 WI showing bilateral and symmetrical occiopitoparietal high SI. (c and d) DWI and ADC map showing free diffusion with high SI in DWI due to T2 shine through and high SI in ADC map denoting vasogenic edema follow up after 2 weeks revealed complete resolution.

supplied by the vertebrobasilar system compared with the anterior circulation; this innervation presumably protects the brain from marked increases in intravascular pressure, such as with severe hypertension. PRES is not an entirely posterior phenomenon, but rather appears in a gradient-like fashion from posterior to anterior, presumably reflecting the gradient of sympathetic innervations (6).

2. Patients and methods

The study protocol was approved by the local Ethics Committee, and informed consent was obtained from all patients.

This prospective study was done between February 2012 and June 2013 with 22 women of reproductive age with their age ranged between 20 and 46 years, between 20 and 40 weeks of gestation and with neurological manifestations, 18 patients (81.8%) presented with elevated blood pressure above 140/90 mmHg and 4 patients (18.1%) were normotensive. Twenty patients (90%), complained of headache, 11 patients (50%) of seizures, decreased consciousness in 17 patients (77%), nausea and vomiting in 13 patients (59%), coma in 6 patients (27%) and loss of vision in 4 patients (18%), Table 1. All patients underwent complete neurological examination. MRI examination was done for all patients using GE signa-1.5 Tesla LX apparatus.

All the twenty two patients were examined by; axial T1 weighted (TR/TE: 450/10 ms), T2 weighted (TR/TE:3881/120 ms), and FLAIR (TR/TE/TI:6000/110/2000 ms). DW

imaging was done using an echo planar imaging (EPI) sequence (TR/TE: 5381/81 ms); slice thickness: 5 mm and the apparent diffusion coefficient (ADC) map were done, 3D time of flight magnetic resonance angiography (3D–TOF–MRA) (TR/TE: 40/3.5) was done to ensure the vascular normality in PRES.

3. Results

According to the hypertensive status 18 patients (81.8%) were hypertensive and 4 patients (18.1%) were normotensive, (table 2).

PRES lesions were typically located in the territories of the posterior circulation, mainly in the parieto-occipital in 18 patients (81.82%), and posterior temporal region with white matter predominance, frontal region in 2 patients (9.09%), midbrain in 2 patients (9.09%), basal ganglia in 4 patients (18.18%), cerebellum in 4 patients (18.18%), and in the thalamus in 2 patients (9.09%) (Table 3). Symmetrical or slightly asymmetrical distribution of the lesions in both cerebral hemispheres was found in most cases, except in one patient who had a lesion mainly over the right cerebral hemisphere. The MRI findings in all the twenty two patients were: abnormal low SI in T1 WI, abnormal high SI on T2 and FLAIR WI.

In DWI, hyperintensity was seen with no restriction and hyperintensity in ADC map due to T2 shine through phenomenon in 15 patients (68.18%), (Figs. 1 and 2), hyperintensity in

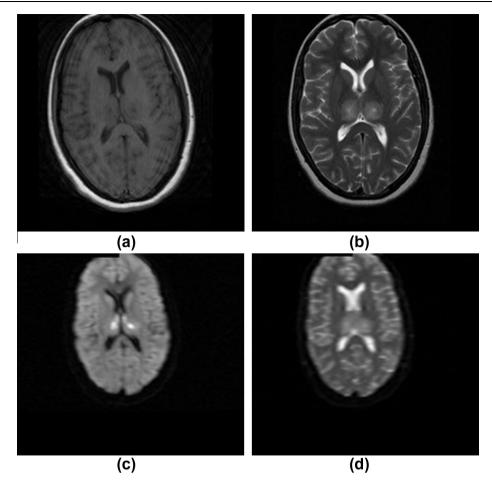


Fig. 2 (a) T1 WI showing low SI in both thalami. (b) T2 WI showing high SI in both thalami. (c and d) DWI and ADC map showing free diffusion with high SI in DWI due to T2 shine through and high SI in ADC map denoting vasogenic edema.

Table 4The diffusion findings.				
Chi-square	DWI	ADC map	No.	%
_	Hyperintense	Hyperintense	15	68.18
-	Hyperintense	Hypointense	4	18.1
	Normotensive	Hypointense	3	13.63
X^2	3.273	0.074	_	-
P-value	1.455	0.227	-	-

DWI with hypointensity in ADC map due to infarction was seen in 4 patients (18.1%), (Fig. 3), and normotensive in DWI with hyperintensity in ADC map was seen in 3 patients (13.63%), (Table 4).

Follow up of 5 cases by MRI and diffusion study showed complete resolution with disappearance of hyperintensity.

4. Discussion

Pregnancy can precipitate new neurological diseases as a result of the alteration in physiology that accompanies the pregnant state, the pregnant patient presenting with neurological problems poses both diagnostic and therapeutic challenges, often forcing the clinician to rely on neuroimaging as part of the work up (7). In our study 18 patients were hypertensive and 4 patients were normotensive but all had the clinical and radiological manifestations of PRES, as in Miza (5) and Hosley and McCullough (8) study in which they found that PRES can develop without a significant elevation of blood pressure. Also in Chou et al (1) study markedly elevated blood pressure was noted in most patients at initial presentation, they have observed that some patients have only mildly elevated or even normal blood pressure. Patients with PRES often have non localizing neurologic symptoms and signs, such as headache, seizure, consciousness disturbance, and/or visual dysfunction. Seizure may be the first neurologic symptom, headache and abnormalities of visual function such as blurred vision or transient blindness are also frequent complaints (1). In our study the most common clinical manifestation was headache which was present in twenty patients followed by decreased consciousness in 17 patients, and seizures in 11 patients. The clinical findings are often nonspecific, so the diagnosis may be difficult to establish. This was in agreement with Brewer et al. (2) in which the headache was present in 87% of cases and visual disturbance in 34%.

The most common MRI abnormality in PRES is brain edema, mainly in the white matter in the posterior portions of the cerebral hemisphere in the parieto-occipital region. The edema may extend to the adjoining gray matter (9). In our study, the most common affected site was the parieto-occipital

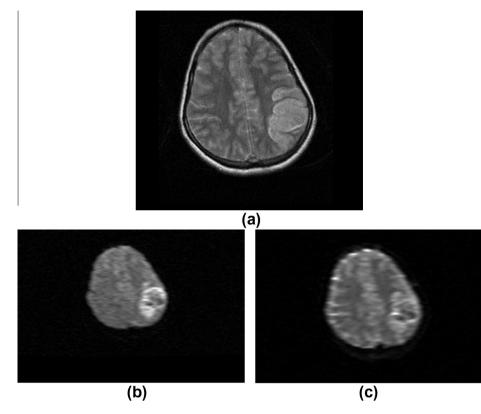


Fig. 3 (a) T2 WI showing area of high signal intensity seen in the left occipital region. (b) Coronal FLAIR showing area of high signal intensity in the left occipital region. (c) DWI showing area of restricted diffusion (high SI) in the left occipital region. (d) Left occipital low SI in ADC map denoting infarction.

region-consistent with the findings of others (2) – which was present in 18 patients (81.82%) Fig. 1. The other regions of the cerebral hemispheres affected were the frontal region (9.09%), midbrain (9.09%), basal ganglia (18.18%), cerebellum (18.18%), and thalamus in (9.09%) Fig. 2.

Symmetrical or slightly asymmetrical distribution of the lesions in both cerebral hemispheres was found in 21 cases, only one patient had a lesion mainly over the right cerebral hemisphere.

Conventional MRI shows decreased signal intensity in T1WI, increased SI in T2WI and FLAIR WI in all cases as in Zidan and Hindawy (6) study.

In diffusion study hyperintensity in DWI with hyperintensity in ADC map in 15 patients (68.18%) was explained to be due to vasogenic edema (T2 shine through phenomenon and not due to true restriction of diffusion), and hyperintensity in DWI with hypointensity in ADC map was seen in 4 patients (18.1%) denoting the presence of cytotoxic edema and development of ischemic infarction with irreversible tissue damage as a common complication of PRES (Fig. 3). These results were in agreement with those of Chou et al. (1), Zidan and Hindawy (6), Covarrubias et al. (10), Watanbe et al. (11) and Koch et al. (12). In three cases (13.63%), the diffusion was normotensive with hyperintensity in ADC map, this occurs due to severe vasogenic edema, in Covarrubias et al. (10) and Watanbe et al. (11) study they noticed that the cytotoxic edema developed immediately adjacent to the area with elevated ADC value.

5. Conclusion

The diagnosis of PRES has important therapeutic and prognostic value. The use of diffusion-weighted imaging and ADC maps allows an earlier and clearer differentiation of cytotoxic and vasogenic edema, which can predict the development of infarction.

Conflict of interest

We have no conflict of interest to declare.

References

- (1) Chou MC, Lai PH, Yeh LR, Li JY, Yuan MK, Liang HL, et al. Posterior reversible encephalopathy syndrome:MRI & DWI in 12 cases. Kaohsiung J Med Sci 2004;20(8):381–8.
- (2) Brewer J, Owens MY, Wallace K, Reeves AA, Morris R, Khan M, LaMarca B, Martin JN. Posterior reversible encephalopathy syndrome in 46 of 47 patients with eclompsia. Am J Obst Gynecol 2013;208(6):468.
- (3) McKinney A, Short J, Truwit CL. Posterior reversible encephalopathy syndrome: incidence of atypical regions of involvement and imaging findings. AJR 2007;189:904–12.
- (4) Marilyn J. Cerebral circulation in pregnancy and eclampsia. Hypertens Am Heart Assoc 2007;50:14–24.
- (5) Miza A. Posterior reversible encephalopathy a variant of hypertensive encephalopathy. J Clin Neurosci 2006:590–5.

- (6) Zidan D, Hendawy S. Acute neurologic disorders during pregnancy and peurperium: a challenging MRI issue. Egypt J Radiol Nucl Med. 2007;38(3):1085–96.
- (7) Brass S, Copen W. Neurological disorders in pregnancy from a neuroimaging prospective. Semin Neurol 2011;31(4):361–73.
- (8) Hosley CM, McCullough LD. Acute neuroradiological issues in pregnancy and the peripartum. Neurospitalist 2011;1(12):104–16.
- (9) Ducros A. Reversible cerebral vasoconstriction syndrome. Lancet Neurol 2012;2(10):906–17.
- (10) Covarrubias DJ, Luetmer PH, Campeau NG. Posterior reversible encephalopathy syndrome:prognostic utility of quantitative dif-

fusion-weighted MR images. AJNR Am J Neuroradiol 2002;23:1038–48.

- (11) Watanabe Y, Mitomo M, Tukodo Y, Yoshida K, Choi S, Hosoki T, et al. Eclamptic encephalopathy: MRI, including diffusion weighted images. Neuroradiology 2003;44:981–5.
- (12) Koch S, Rabinstein A, Falcone S, Forteza A. Diffusion-weighted imaging shows cytotoxic and vasogenic edema in eclampsia. Am J Neuroradiol 2001;22:1068–70.