Different Clinical and Imaging Faces of Posterior reversible leukoencephalopathy syndrome (PRES)

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ABSTRACT

Posterior reversible leukoencephalopathy syndrome (PRES) is classically characterized by headache, confusion, seizure, visual loss, and imaging findings of bilateral cortical and subcortical vasogenic edema in the posterior circulation area. We present three cases of PRES with different clinical and/or imaging features. They were associated with pregnancy. First case was a typical PRES, second case’s lesions were predominantly in the anterior circulation area of the brain, and third case was hemorrhagic PRES. Different clinical and/or radiological features can be seen in cases with PRES.

Key words: PRES features, hemorrhagic PRES, eclampsia

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INTRODUCTION

In 1996, Hinchey et al described a clinico-radiological entity which was called reversible posterior leukoencephalopathy syndrome. This syndrome is characterized by visual loss, seizures, altered mental functioning and changes in subcortical white matter of the parietal-temporal-occipital regions of the brain. These clinical signs and abnormalities on imaging are associated with sudden elevations in systemic blood pressure and immunosuppressive therapy and always reversible (1). PRES associated with different diseases have been reported in recent years (2-4). Here we presented 3 cases of PRES who had different clinical and/or imaging findings.

CASE 1

A 23 year-old, 32 weekly pregnant was referred to our emergency department because of sudden vision loss and headache. Her blood pressure was 160/90 mmHg. Neurologic examination demonstrated bilateral cortical blindness with limit of vision to perception of light only. Pupils were equal and reacting to light and examination of fundi was unremarkable. One positive proteinuria of her urine analyze was determined. Initially, pre-eclampsia and eclampsia in the patient was not thought by gynecology and obstetrics physicians. Ophthalmological examination was normal except that blindness. Bilaterally doubtful hypodense appearance in occipital regions was seen on the brain CT. The patient was admitted to our neurology department. Hyperintensities in bilaterally occipital regions were seen on brain FLAIR weighted images on the second day (Figure 1). In addition she had a generalized tonic-clonic seizure and three positive proteinuria in the second day of admission. Cesarean section (C/S) was promptly performed because of eclampsia. A follow-up visit at 4 weeks revealed complete neurologic recovery with the vision of 20/25 bilaterally. Latest MRI showed almost resolution of the lesions.

CASE 2

A 22 year-old, pregnant was referred to our emergency department because of eclampsia. C/S was promptly performed. The patient was consulted due to agitation and confusion after a day of C/S. There were agitation, confusion and Babinski’s sign on the right on the neurologic examination. Her brain MRI relieved bilaterally high signal intensities in the subcortical white matter areas of the frontal and parietal lobes (Figure 2). Repeat brain MRI after eleven days revealed dramatic improvement with almost complete resolution of high signal intensities.

CASE 3

A 26 year-old, pregnant was referred to our emergency department because of eclampsia. C/S was promptly performed. After three days she discharged but six days later she was handed to our hospital again with seizure. There was homonymous hemianopia on her neurologic examination. Other neurologic and fundoscopic examinations were unremarkable. Phenytoine was started for her seizures. A heterogeneous hyperdense lesion (consistent with a cerebral hemorrhage) in the left parietooccipital region on her brain CT (Figure 3-a), and subcortical hyperintense lesion on the right parietal lobe were seen on the coronal sections of brain FLAIR weighted MRI (Figure 3-b). Complete resolution of the cerebral lesions were seen on control brain FLAIR MRI on day 21 (Figure 3-c).

DISCUSSION

PRES classically have headache, altered mental status, seizures, stupor and visual disturbances and changes in the subcortical white matter of the temporoparietooccipital lobes that are shown up in the neuroimages, and most common causes of PRES are hypertensive encephalopathy, eclampsia, immunosuppressive drugs, and uremic encephalopathy (1). We discussed three cases with PRES; they have different clinical and radiological features in each other.

According to her clinical and radiological features we can say that our first patient is a typical PRES case. Our second patient is also typical PRES as clinically but her brain lesion was in the anterior circulation area of brain. In contrast to original description of this syndrome, lesions can be seen in anterior circulation area of brain in some cases with PRES (5,6).

The third patient was also clinically PRES, but her radiological findings were not typical for it. She was initially diagnosed as intracerebral hemorrhage according to her brain CT feature (Figure 3a). But one day later, her brain MRI showed a hyperintense appearance in the
right parietal region on the FLAIR images in addition to the hemorrhagic lesion (Figure 3b, arrow). Any vascular abnormalities under the hemorrhagic lesion were not seen on brain MRI and CT-angiographic and venographic examinations. Hemorrhagic lesions or intracerebral hemorrhage in PRES are seen very rarely (7). The hyperintense lesion in parietal lobe on FLAIR MRI (Figure 3b, arrow) made us to think that the patient was a PRES. FLAIR MRI is especially the best way to determine PRES lesions (8).

In conclusion, according to these cases, different clinical and/or radiological features can be seen in cases with PRES.

REFERENCES

2. Becker K. Hypertensive encephalopathy, eclampsia, and
reversible posterior leucoencephalopathy. Neurocrit Care 2006;12:30-45


