

Acute cortical blindness caused by pre-eclampsia in the antepartum; posterior reversible encephalopathy syndrome (PRES)

Yacong Wang¹, Qinying Cao², Lihong Zhang³,
Su'e Zhang², Liang Shi⁴, Ou Sha⁵

1. Department of Ophthalmology, The Fourth Hospital of Shijiazhuang, Shijiazhuang 050011, Hebei, China
2. Department of Obstetrics, The Fourth Hospital of Shijiazhuang, Shijiazhuang 050011, Hebei, China
3. Department of Neurology, The Second Hospital of Hebei Medical University, Shijiazhuang 050000, Hebei, China
4. Department of Radiology, The First Hospital of Shijiazhuang, Shijiazhuang 050011, Hebei, China
5. School of Medicine, Shenzhen University Science Health Centre, Guangdong 518060, China

DOI: <http://dx.doi.org/10.4314/ahs.v15i2.51>

We present a case report of a patient presenting posterior reversible encephalopathy syndrome (PRES), a rare acute neurological condition associated with pre-eclampsia. A possible common aetiology and successful clinical management approach is reported.

PRES is a rare neurological syndrome first described in 1996¹. Its clinical manifestation varies and is nonspecific¹. Most PRES patients suffer from headaches, consciousness disorders, seizures, vomiting, mental status changes and visual abnormalities¹. Its typical imaging finding is characterized by a bilateral posterior cerebral vasogenic edema, especially in the parietal lobe and occipital lobe^{1,2}. Pre-eclampsia or eclampsia is the most

common cause of PRES³. Most PRES cases associated with pre-eclampsia/eclampsia are postpartum and intrapartum, rather than antepartum⁴. Visual abnormalities in PRES commonly include blurred vision, hemianopia, visual neglect, etc¹. Reversible cortical blindness is a very rare symptom⁵.

A 23-year-old woman (gravida 2, para 1) at week 37⁺³ of gestation was admitted to the Department of Obstetrics of the Fourth Hospital of Shijiazhuang, China, with bilateral visual loss accompanied by headache, nausea and vomiting with unknown reasons for the past 4 hours. The first and second trimester had been uneventful with no history of hypertension and epilepsy before or during pregnancy. There was no other significant history.

On admission, all vital signs were normal except her blood pressure which was 175/107 mmHg. An obstetric examination confirmed a fetus in a head-down position. A color doppler ultrasound showed a single live fetus with intrauterine growth retardation and oligohydramnios, and a laboratory test showed proteinuria (3+). A diagnosis of severe pre-eclampsia was made. Continuous infusion of magnesium sulphate at a dose of 0.5g/h was immediately administered to the patient to relieve vasospasm, and nifedipine and labetalol were administered orally to control blood pressure.

As a result of the reported bilateral visual loss, fundoscopic examination was performed by an ophthalmologist and slight narrowing of the retinal artery was detected. Cortical blindness was suspected and a cranial MRI examination with diffusion-weighted imaging (DWI) was performed with a 1.5 T scanner (Achieva, Philips Healthcare, Best, The Netherlands). An 8-chan-

Corresponding authors:

Lihong Zhang

Department of Neurology

The Second Hospital of Hebei

Medical University

Shijiazhuang 050000

Hebei, China

Email: lihong.zhang@yahoo.com

Tel: +86-311-66007336

Fax: 311-66007448

Ou Sha

Department of Medicine

School of Medicine

Shenzhen University Health Science Centre

Shenzhen 518060

Guangdong, China

Email: shaou@szu.edu.cn

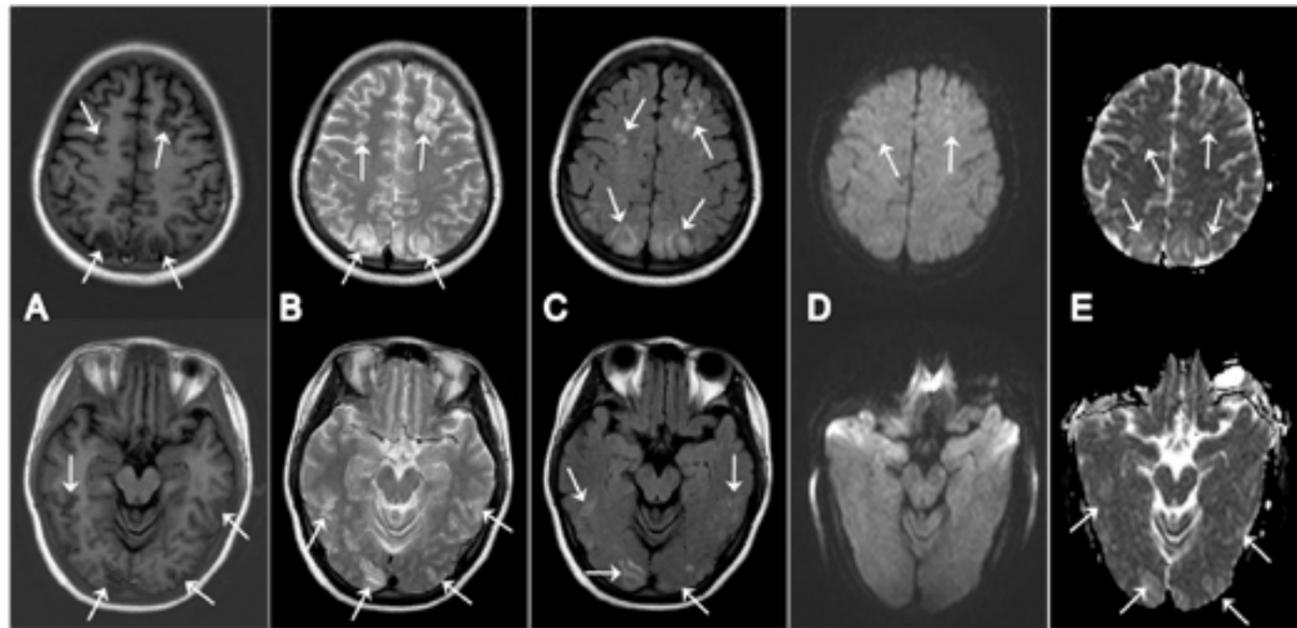
Tel: +86-755-8667 1918

Fax: +86-755-8667 1906

nelled head-neck coil was used as signal transmitter and receiver. The image showed low signal intensity in the bilateral frontal lobes, parietal lobes, temporal lobes and occipital lobes in T1-weighted images (T1WI). In contrast, hyperintense signals were found in T2-weighted

images (T2WI) and fluid-attenuated inversion recovery (FLAIR). Isointensity or slightly increased signals were found through DWI. Hyperintense signals showed apparent diffusion coefficient (ADC) maps (Fig. 1), suggesting a diagnosis of PRES.

Fig. 1 Axial MR imaging in PRES patient with pre-eclampsia. A, T1WI shows roughly symmetric low signal intensity located in the bilateral frontal lobes, parietal lobes, temporal lobes and occipital lobes (arrows); B and C, T2WI and FLAIR sequence, respectively, both of which show hyperintense signals in the same positions indicated by arrows; D, DWI shows slightly increased signals in the frontal lobes and isointense signals in the other lobes (arrows); E, ADC maps reveals hyperintensities in similar regions (arrows), which are consistent with the images in vasogenic edema.



After controlling the blood pressure, the patient underwent a caesarean section 6 hours after admission to the hospital, and delivered a 1450 gram baby. The subsequent pathological examination showed inflammatory cell infiltration in the chorion of the placenta. Some of the chorion stroma had become marginally fibrotic, some showing fibrous necrosis. Inflammatory cell infiltration was also found in the fetal membrane. Three days after surgery, the patient's vision recovered, her headache disappeared, and blood pressure dropped to 130/61 mmHg. Eight days later, her wound healed and

she was discharged from the hospital. A final diagnosis of PRES with severe pre-eclampsia was confirmed.

The pathophysiological mechanism of PRES caused by pre-eclampsia is still unclear. One hypothesis is that high blood pressure causes cerebrovascular autoregulation failure, resulting in hyperperfusion. Another is that hypoperfusion is caused by vasospasm or vasoconstriction⁶. Vasogenic edema in brain tissue occurs in both hypotheses⁶. Consequently, there is endothelial damage, at least in part, at the blood-brain barrier⁷.

The cause of pre-eclampsia is unclear, but is known to be associated with endothelial damage in the placenta^{8,9}. In this case, necrosis and phagocyte infiltration were found in the placenta. The diffuse vascular endothelial activation and injury, consequent vasoconstriction and an increase in endothelial permeability, may induce the variable signs of pre-eclampsia, including vasogenic edema of brain tissue^{8,9}. If vasogenic edema occurs in the visual cortex of the patient, it may induce cortical blindness.

As PRES improved and resolved after delivery of the baby and the placenta, it is suggested that the culprit may be in the placenta^{3,10}. The endothelial damage noted in the placenta may be paralleled in the endothelial damage at the blood-brain barrier with a possible common aetiology^{8,9}.

It is imperative that clinicians, including obstetricians, ophthalmologists and neurologists are familiar with variable features and factors of PRES and its association with blindness. A funduscopy examination is essential to diagnose a patient with pre-eclampsia who complains of ophthalmic symptoms⁷. If cerebral oedema is suspected, an MRI must also be performed. In PRES, the MRI image shows symmetrical or bilateral vasogenic edema in the parietal and occipital lobe. Vasogenic edema is also commonly found in the frontal lobe, temporal lobe and cerebellar hemisphere^{1,2}. In the brain edema area, an MRI image shows hypointense or isointense signals in T1WI and hyperintense signals in T2WI and FLAIR sequence^{2,11}. The combination of DWI and ADC maps can separate vasogenic edema from cytotoxic edema, which is critical in the diagnosis of PRES¹¹. The hypointense or isointense signals in DWI and hyperintense signals in ADC images indicate the area of vasogenic edema. The signals in DWI sometimes increase slightly due to T2 "shine-through" effect in vasogenic edema. In contrast, the hyperintense signals in DWI and hypointense signals in ADC images indicate the area of cytotoxic edema. Therefore, DWI and ADC maps can diagnose PRES and early stage cerebral infarction easily, enabling prompt diagnosis, critical to guide therapy and informed prognosis of the patient with PRES¹².

The key to treat pre-eclampsia and PRES include control of blood pressure, prevention of seizures, and termination of pregnancy¹³. Antihypertensive treatment should be given when the patient's systolic pressure is consistently higher than 160 mmHg or diastolic blood

pressure is 105-110 mmHg or above. Common anti-hypertensive drugs include labetalol, hydralazine and short-acting oral nifedipine. The preferred medicine to prevent seizures is magnesium sulphate to relieve vascular spasm. As soon as blood pressure is stabilised, the pregnancy should be terminated immediately¹³.

Conflicts of interest:

There is no conflict to disclosure.

Acknowledgement

Grant support from the National natural science foundation of China (No. 81171154), Collaborative innovation program of Shenzhen (GJHS20120621153317134) and Science & technology support program of Hebei Province (No. 122777142) is gratefully acknowledged. The authors thank Ms Taraneh Afnan-Holmes of Shenzhen University for her native English revision of this manuscript.

References

- Hinchey J, Chaves C, Appignani B, Breen J, Pao L, Wang A, et al. A reversible posterior leukoencephalopathy syndrome. *New England Journal of Medicine* 1996; 334(8): 494-500.
- Hugonnet E, Da ID, Boby H, Claise B, Petitcolin V, Lannareix V, et al. Posterior reversible encephalopathy syndrome (PRES): Features on CT and MR imaging. *Diagnostic and Interventional Imaging*. 2013; 94: 45-52.
- Liman TG, Bohner G, Heuschmann PU, Endres M, Siebert E. The clinical and radiological spectrum of posterior reversible encephalopathy syndrome: The retrospective Berlin PRES study. *Journal Neurology*. 2012; 259(1): 155-164.
- Hegde HV, Patil PB, Rameshkumar R, Sunita TH, Bhat MT, Desai RM, et al. A rare case of antepartum posterior reversible encephalopathy syndrome. *Anaesthesia and Intensive Care*. 2011; 39 (3): 499-502.
- Llovera I, Roit Z, Johnson A, Sherman L. Cortical blindness, a rare complication of pre-eclampsia. *The Journal of Emergency Medicine*. 2005; 29(3): 295-297.
- Bartynski WS. Posterior reversible encephalopathy syndrome, part 2: Controversies surrounding pathophysiology of vasogenic edema. *AJNR American Journal of Neuroradiology*. 2008; 29(6): 1043-1049.
- Pula JH and Eggenberger E. Posterior reversible encephalopathy syndrome. *Current Opinion in Ophthalmology*. 2008; 19(6): 479-484.
- Redman CW and Sargent IL. Latest advances in understanding preeclampsia. *Science*. 2005; 308(5728): 1592-1594.

9. Noris M, Perico N, Remuzzi G. Mechanisms of disease: Pre-eclampsia. *Nature Clinical Practice Nephrology*. 2005; 1(2): 98-114.
10. Servillo G, Striano P, Striano S, Tortora F, Boccella P, De RE, et al. Posterior reversible encephalopathy syndrome (PRES) in critically ill obstetric patients. *Intensive Care Medicine*. 2003; 29(12): 2323-2326.
11. Doelken M, Lanz S, Rennert J, Alibek S, Richter G, Doerfler A. Differentiation of cytotoxic and vasogenic edema in a patient with reversible posterior leukoencephalopathy syndrome using diffusion-weighted MRI. *Diagnostic and Interventional Radiology*. 2007;13(3): 125-128.
12. Nasr R, Golar M, Berger J. Posterior reversible encephalopathy syndrome in a woman with pre-eclampsia. *Journal of Obstetrics Gynaecology*. 2010. 30(7): 730-732.
13. Sibai B, Dekker G, Kupfermanc M. Pre-eclampsia. *Lancet*. 2005. 365(9461): 785-799.