

## Case Report

# Cortical blindness associated with posterior reversible encephalopathy syndrome in an eclamptic woman: a case report

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**Abstract:** We reported a case of a 30-year-old multipara who was admitted at 37 weeks of gestation for induction of labour due to preeclampsia. After postpartum, the patient developed severe hypertension, eclamptic fits, confusion and cortical blindness. The patient was treated in the intensive care unit with antihypertensive drugs, calcium-channel antagonists, anticonvulsants and anti-edematous agents. Nineteen days later, there was a complete clinical improvement, except for a left homonymous hemianopia which remained. A head MRI showed that most of the lesions in the bilateral subcortical areas of the parietal, temporal and frontal lobes observed in previous images had disappeared, with the exception of the left occipital lobe lesions, which were significantly reduced. A neurological follow-up at 18 months found no permanent neurological abnormalities, except for the left homonymous hemianopia. Diagnosis of cortical blindness associated with posterior reversible encephalopathy syndrome is based on the presence of clinical features of acute neurologic compromise, abnormal neuroimaging, and nearly complete reversibility of findings after timely treatment. Further research should focus on the development of more effective therapeutic strategies for this condition.

**Keywords:** Posterior reversible encephalopathy syndrome, cortical blindness, eclamptic

## Introduction

Posterior reversible encephalopathy syndrome (PRES) is a clinical-radiological entity which was first reported by Hinchey *et al.* [1] as reversible changes to the central nervous system (CNS) on brain nuclear magnetic resonance imaging (MRI) [2]. Prior to 2000, this condition had various designations, including reversible posterior leukoencephalopathy syndrome, reversible posterior cerebral oedema syndrome, and reversible occipital parietal encephalopathy [3]. Among common neurological complications in eclampsia, cortical blindness is the most dreaded and may be accompanied by PRES. Despite the increasing number of reports that have aroused awareness among clinicians, the pathophysiology of PRES remains largely unknown [4].

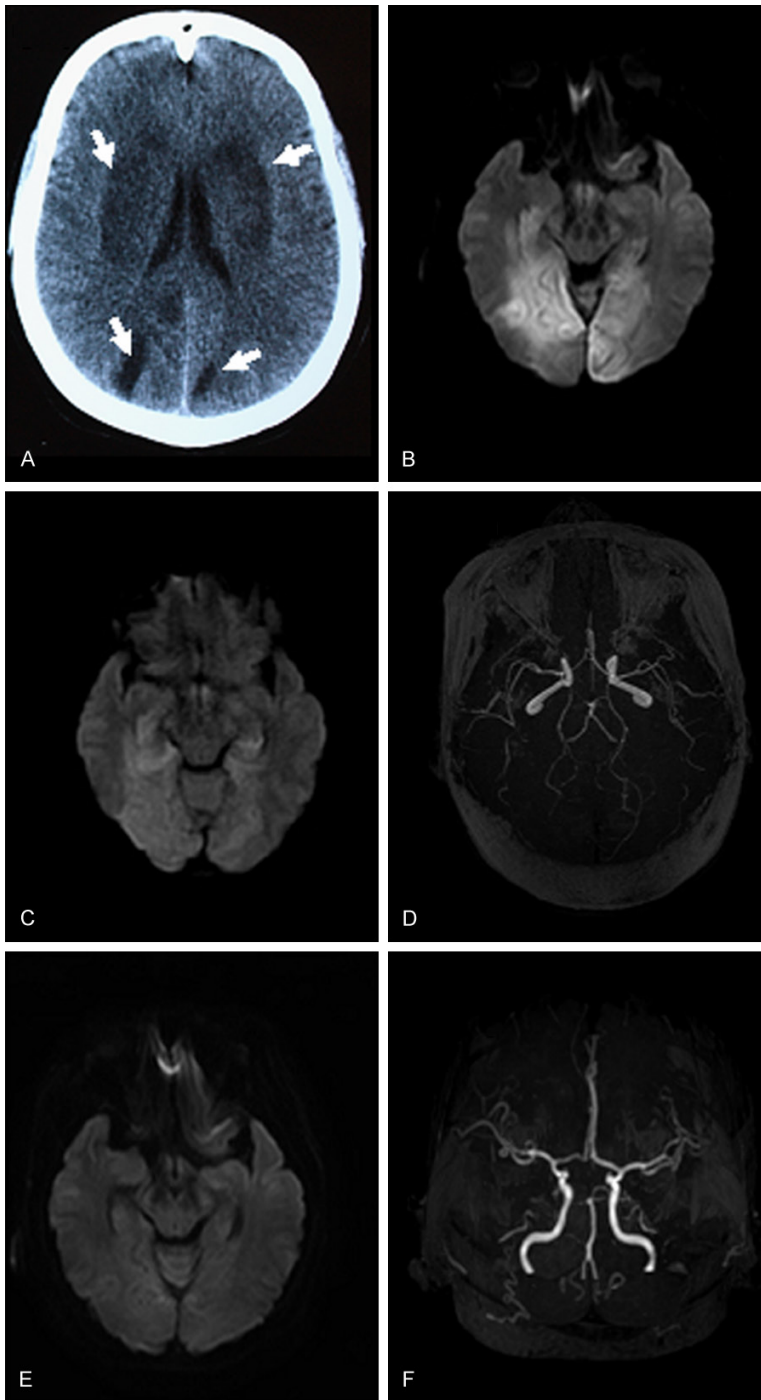
Here, we report a case of cortical blindness in a pregnant woman with eclampsia complicated

with PRES after delivery, in which vision was not completely restored at an 18-month follow-up after delivery.

## Case report

A 30-year-old multipara was admitted to a community hospital at 37 weeks of gestation with a diagnosis of preeclampsia where she underwent emergency caesarean section under general anaesthesia. At 4 h postpartum, she endured two seizures and loss of consciousness. Blood pressure was 168/108 mmHg. The patient received administration of magnesium sulphate and the subsequent head computed tomography (CT) revealed no evidence of acute haemorrhage, but showed low attenuation (hypodense) signals in symmetrical bilateral areas of the parietal lobe, occipital lobe, and basal ganglia (**Figure 1A**). At 10 h postpartum, the patient was intubated and transferred by ambulance to the intensive care unit of our hos-

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**Figure 1.** CT scan obtained on 4 hours after postpartum (A). FLAIR image obtained on postpartum day 5 (B), shows hyperintense regions in the occipital, parietal, temporal, frontal lobes, corpus callosum, basal ganglia and thalamus. FLAIR image obtained on postpartum day 7 (C) shows the enhanced hyperintense regions. MRA of the circle of wills (D) shows multifocal cerebral artery stenosis. FLAIR image (E) from MRI of the same patient obtained on postpartum 19th day shows near complete resolution of the lesions, but the persistence of high signal on left occipital lobe. MRA obtained on postpartum 19th day after injection of nimodipine (F) shows nearly complete resolution.

was 4, while her pupils were 5 mm in diameter, dilated, and unresponsive to light. Grade 3 peripheral oedema of the lower extremities was observed. Her body temperature was 37.3°C, blood pressure was 168/108 mmHg, and pulse rate was 108 beats per minute. She was intubated and her oxygen saturation was 98% under assisted breathing by mechanical ventilation (SIMV: f, 12/min;  $\text{FiO}_2$ , 60; TV, 360 mL; I:E = 1:2).

This patient received continuous intravenous magnesium sulphate administration for the first 24 h. At the same time, the patient was administered labetalol and nicardipine infusions to maintain her blood pressure at 144-168/88-120 mmHg. This patient also received other medication such as furosemide, glycerine and aminoflilina as an anti-oedema therapy. During this therapeutic process, negative fluid balance was maintained. Five days later, the patient regained consciousness and the severe peripheral oedema disappeared while her body weight decreased from 68.2 to 63.3 kg. The patient did not have a seizure after presenting at our institute and she continued to gradually recover; however, her vision was found to be impaired. On examination, she was only able to perceive light and dark visual stimuli, but unable to distinguish between colours or identify objects even though her pupils became reactive to light and the pupil diameters decreased to 4 mm. Other neuro-ophthalmological parameters were normal. Subsequent MRI revealed lesions with increased

pital. On examination, she was unconscious and her score on the reaction level scale (RLS)

intensity on fluid attenuated inversion recovery (FLAIR) sequences that were distributed in the

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bilateral subcortical areas of the occipital, parietal, temporal and frontal lobes, as well as the corpus callosum, basal ganglia and thalamus (**Figure 1B**).

On post-admission day 7, the consciousness level of this patient worsened, resulting in subsequent coma. Repeat urgent MRI with magnetic resonance angiography (MRA) and venography revealed severe posterior parenchymal changes on FLAIR sequences (**Figure 1C**), with multifocal stenosis and dilatation of the intracranial arteries (**Figure 1D**). A magnetic resonance venogram showed no evidence of cerebral venous sinus thrombosis. Nimodipine was administered as an anticonvulsant therapy to relieve vasospasm. Afterward, she gradually recovered and regained consciousness the following day. On post-admission day 19, her vision improved and she was able to count fingers and identify colours. Only a left homonymous hemianopia remained. A head MRI at this time showed that most of the lesions in the bilateral subcortical areas of the parietal, temporal and frontal lobes observed in previous images had disappeared, with the exception of the left occipital lobe lesions, which were significantly reduced (**Figure 1E**). Notably and importantly, MRA revealed complete resolution of vasoconstriction (**Figure 1F**), which had been seen previously within the territories of cerebral arteries.

A neurological follow-up at 18 months found no permanent neurological abnormalities except the left homonymous hemianopia. However, we were unable to perform corresponding MRI for further comparisons of the left homonymous hemianopia due to financial constraints.

### Discussion

PRES is a rare but severe pathological condition of the CNS associated with a variety of pathological conditions [4]. Even though the main obstetric precipitating factors involved in PRES are preeclampsia and eclampsia, the characteristics and proportions of eclamptic patients at risk for PRES remains unclear [5].

In the present case, eclampsia played a crucial role in the pathogenesis of PRES, which is characterized by headache, altered mental status, seizure attacks and visual disturbances [6]. Ocular involvement may occur in as many as

50% of patients with preeclampsia/eclampsia [7]. The exact pathogenesis of cortical blindness in preeclampsia/eclampsia remains largely unknown [5, 8, 9].

Cortical blindness is an uncommon complication of preeclampsia and eclampsia that is usually characterized by sudden bilateral visual loss with normal pupillary responses and fundi changes [10].

PRES has diverse patterns of expression, which sometimes can make diagnosis difficult. When suspected clinically, the diagnosis is generally confirmed neuroradiologically either by CT or MRI, and the abnormalities are often apparent on CT scan, but are best depicted by MRI [2, 4]. On imaging, PRES is characterized by abnormalities in the white and grey matter, predominantly affecting the posterior regions [2].

In this case, an eclamptic woman was diagnosed with PRES and cortical blindness based on the presence of clinical features and abnormal neuroimaging findings. In addition, rapidly progressive brain oedema and dynamic arterial changes strongly suggested a diagnosis of postpartum cerebral angiopathy or, more specifically, reversible cerebral vasoconstriction syndrome (RCVS) [11].

PRES associated with vasospasm, as revealed by MRA, has been reported in patients with eclampsia and preeclampsia [9]. Imaging and clinical features consistent with PRES are common in RCVS patients, strongly suggesting the existence of a clinical and pathophysiologic continuum between these two pathological conditions [9, 11].

Identification of PRES and blindness can be helpful for the timely management of these complications. The management of PRES should focus on prompt, continuous reduction of blood pressure or withdrawal of the causative substances [4]. The prognosis of PRES is usually good, with complete to near-complete resolution of clinical symptoms within 3 months. However, there remains a risk of permanent neurological deficits or even death [4, 8].

Cortical blindness is a rare but disturbing complication of PRES in eclamptic pregnant women. In our patient, vision was not completely restored at 18 months after delivery by cae-

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sarean section and treatment with antihypertensive drugs, calcium-channel antagonists, anticonvulsants and anti-oedema measures. Further intensive research should focus on uncovering the precise mechanisms underlying different complications of PRES in order to develop more effective therapeutic strategies.

### Disclosure of conflict of interest

None.

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