International Journal of Reproduction, Contraception, Obstetrics and Gynecology
Awowole IO et al. Int J Reprod Contracept Obstet Gynecol. 2026 May;15(5):1799-1802
www.ijrcog.org
pISSN 2320-1770 | eISSN 2320-1789

DOI: https://dx.doi.org/10.18203/2320-1770.ijrcog20261289

Case Report

Posterior reversible encephalopathy syndrome presenting as refractory postpartum seizures in a resource-constrained setting: a diagnostic dilemma and maternal near-miss from Nigeria

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Received: 12 March 2026
Accepted: 09 April 2026

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ABSTRACT

Posterior reversible encephalopathy syndrome (PRES) is an uncommon complication of pregnancy, whose rarity may lead to delayed diagnosis adverse outcomes, especially when seizures persist despite standard anti-seizure regime for eclampsia. A 25-year-old primiparous with sickle cell haemoglobinopathy was admitted about 48 hours into the puerperium with generalized tonic-clonic seizures, severe hypertension and altered mental state. She was managed for eclampsia and suspected sepsis with magnesium sulphate, antihypertensives and antibiotic therapy, with transient improvement. She however deteriorated rapidly and lapsed into unconsciousness, prompting further evaluation. After a period of delay due to financial difficulties, computerized tomography scan revealed widespread bilateral vasogenic edema that was characteristic of PRES. Following the initiation of corticosteroid therapy, the seizures ceased and she achieved full neurological recovery without any deficit. This case depicts the diagnostic dilemma of persistent pregnancy-associated seizures, especially in resource-constrained settings. Heightened suspicion, early imaging and multidisciplinary vigilance are recommended for such patients.

Keywords: Postpartum eclampsia, Posterior reversible encephalopathy syndrome, Seizure, Vasogenic edema, Low-and-middle-income-countries

INTRODUCTION

Convulsion in a pregnant woman, up to six weeks after delivery is an obstetric and a neurological emergency due to the associated feto-maternal morbidity and mortality.[1] Preeclampsia, which complicates about 2-8% of all pregnancies, is the commonest predisposing factor; it is therefore reasonable that eclampsia is almost exclusively the first differential diagnosis of convulsion in pregnancy and the puerperium. Other differential diagnoses include bacterial and tuberculous meningitis, encephalitis, intracranial tumors, cerebral venous sinus thrombosis, haemorrhagic/ischaemic cerebrovascular accidents and posterior reversible encephalopathy syndrome. The symptoms, including headaches, hypertension and visual disturbances overlap in these conditions. The Magpie trial confirmed the superiority of magnesium sulphate as the anticonvulsant of choice for primary and secondary seizure prophylaxis, without inebriating the fetus.[2]

However, when convulsions persist despite optimal dosing of magnesium sulphate, evaluation for other aetiologies are immediately indicated.

In low-and middle-income countries where access to diagnostic imaging is limited and the quality of care is significantly restricted by financial constraints, managing a patient with convulsion in the immediate postpartum period could be daunting.

We hereby present the diagnostic dilemma encountered, in the management of a patient with a near-miss due to repeated episodes of convulsion in the immediate postpartum period, following delivery in a peripheral hospital in Nigeria.

CASE REPORT

A 25-year old P1+0, 1 Alive with background sickle cell haemoglobinopathy presented to the obstetric emergency unit of the Obafemi Awolowo University Teaching Hospitals Complex, Nigeria with repeated generalized tonic-clonic seizures and altered consciousness. The symptoms started about 48 hours after an emergency caesarean section that was done on account of intrapartum fetal distress. The estimated blood loss at surgery was about 500 ml, for which she had 2 units of blood transfused. Initial evaluation revealed a young woman with a Glasgow coma score (GCS) of 8/15, febrile (temperature 38 degrees Celsius), and an admission blood pressure of 210/120 mmHg.

Empirical treatment for postpartum eclampsia and puerperal sepsis was initiated with intravenous Magnesium sulphate, Labetalol, Cefuroxime and Metronidazole. Full blood count revealed leucocytosis with neutrophilia. Her renal and liver function tests were within normative references. Following the treatment, her clinical condition improved initially, with her GCS increasing to 14/15. She however, deteriorated rapidly, to a GCS of 3/15. She also developed left-sided proptosis and anisocoria. This prompted consideration of other diagnoses, including bacterial meningitis, posterior reversible encephalopathy syndrome (PRES) and cerebral venous thrombosis.

Following a 5-day delay due to financial constraint, a brain computed tomography (CT) scan was eventually done, which revealed extensive bilateral symmetrical vasogenic edema that diffusely affected cerebral and cerebellar hemispheres (Figure 1).

The radiological features were consistent with posterior reversible encephalopathy syndrome and treatment with intravenous dexamethasone was instituted, alongside the previous treatment. The convulsions abated and she regained full consciousness within 48 hours of instituting the steroid therapy, without any obvious neurological sequelae.

She had 4 units of blood transfused to correct low haematocrit and was subsequently discharged for outpatient follow up at the postnatal and neurology clinics.

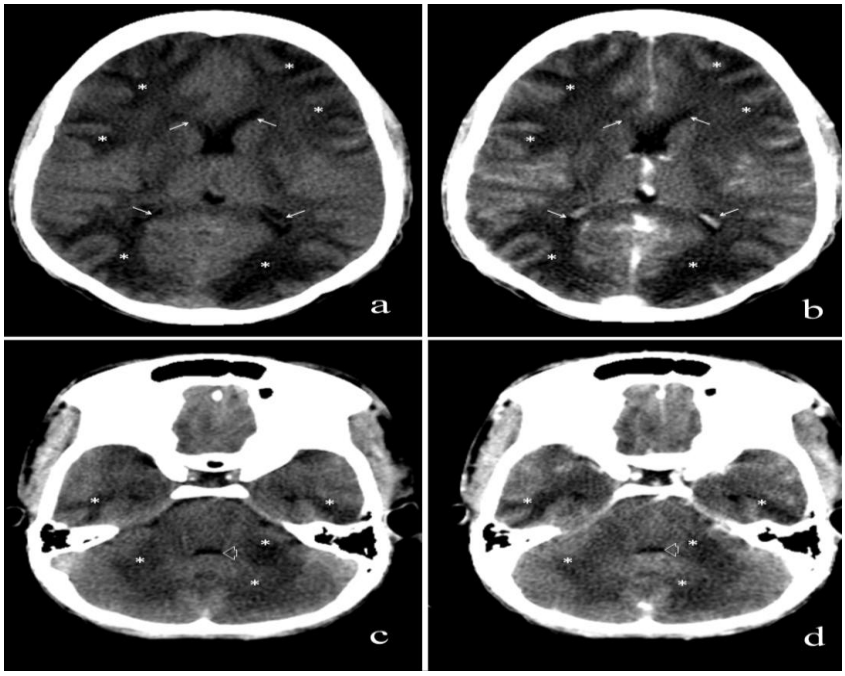

Figure 1: Pre-contrast and post-contrast axial brain CT scan images at the (a and b) ganglionic, and (c and d) pontine levels showing extensive bilateral symmetrical areas of primarily subcortical white matter hypodensity involving the cerebrum and cerebellum on both sides (*) with no demonstrable enhancement post intravenous contrast. Mild narrowing of the lateral ventricles (thin arrows) and fourth ventricle (thick arrows) is also noted. These findings are consistent with diffuse vasogenic edema with no associated parenchymal haemorrhage.

DISCUSSION

Posterior reversible encephalopathy syndrome is a rarely recognized subacute neurological disorder that is characterized by headache, visual impairments, convulsions, and loss of consciousness. It is reported to occur in about 2-3 per 100,000 patients, with a male to female ratio of at about 2:1. The peak age of incidence is in the 13-20 years' age range, with more predilection for the black ethnicity.[3] The incidence of PRES is, however, often under-reported as the commonly reported symptoms of headache, visual disturbance, convulsions and loss of consciousness are non-specific to the disorder. Given the cost and restricted access to quality neurological images in LMICs, patients with PRES are further likely to suffer delayed diagnosis or be outrightly undiagnosed, as reported in the index case, with adverse consequences.

The aetiopathology of PRES hinges on vasogenic oedema, which is most often related to hypertension in about 80% of radiologically confirmed cases. In women, preeclampsia and eclampsia, leading to increased vascular permeability when blood pressure exceeds the autoregulatory limit are the most common predisposing factors to PRES.[3] Other risk factors include acute and chronic renal impairments, immune-mediated systemic disorders that trigger the generation of blood-brain barrier modulatory cytokine and autoantibodies, diselectrolytaemia, sepsis, traumatic spine injuries, intoxications, cytotoxic agents and angiogenic inhibitors.[4] Background haemoglobinopathy as reported in the index patient, together with preeclampsia further aggravated the patients' predisposition towards malignant PRES, which is defined as GCS ≤7 with progressive clinical deterioration despite treatment.[5] This is seen as significant cerebellar and supratentorial oedema with mass effect in affected patients.[3]

The confirmatory diagnosis of PRES following an initial clinical suspicion is radiological. The associated vasogenic oedema are most prominent bilaterally, though asymmetrical in the subcortical, parietal and occipital lobes in 90% of affected patients on magnetic resonance imaging.[6] Contrast enhanced CT scan also reveals similar patterns, though with less sensitivity compared with MRI. Unilateral affectations have been reported but this is rarely seen. Other areas of the brain such as the superior frontal gyrus, temporal lobe and the brainstem may also be affected, though less frequently.[7] The additional benefits of cross-sectional imaging, however, include the exclusion of other possible causes of repeated convulsions such as intracranial haemorrhage, which was suspected in the index patient due to severe hypertension with refractory response to initial antihypertensive therapy. In other instances, intracranial bleeding may occur as a complication of PRES, rather than as the aetiology of clinical symptoms.[8] However, there was no evidence of intracranial bleeding in the index patient. Superior sagittal sinus thrombosis was also considered, but the classic findings of hyperdense sinus and empty delta signs were absent on the cranial CT scan.

The treatment of PRES is targeted towards treating the underlying factors, which in this case included magnesium sulphate and antihypertensive therapy. She was monitored closely to avoid fluctuations in blood pressure, which could be counter-productive.[9] While high-dose or prolonged steroids can be a predisposing factor for PRES, initiation of steroid therapy for the vasogenic edema as part of a broader management therapy is associated with recovery and resolution of symptoms in a systematic review, especially in patients with underlying inflammatory or autoimmune diseases.[10] Prognosis is reassuring following recovery from PRES. Some patients may however have acute dysfunctions post recovery, while a few others may require long term anticonvulsant therapies for repeat seizures.[8]

CONCLUSION

This case depicts the clinical challenges that are often encountered in the management of patients with pregnancy-associated seizures, especially in resource-constrained settings like Nigeria. The repeated convulsions despite magnesium sulphate therapy on the background of eclampsia, sickle cell haemoglobinopathy and delayed access to high quality neuroimaging created a triplex diagnostic dilemma involving posterior reversible encephalopathy syndrome, cerebral venous sinus thrombosis and haemorrhagic stroke. A high index of suspicion amongst Obstetricians, Radiologists and Neurologists is critical for early recognition of PRES. Timely neuroimaging remains pivotal in establishing the diagnosis, and excluding other life-threatening intracranial pathologies. This case further emphasizes the disproportionate burden borne by women in LMICs, where financial and infrastructural constraints may delay diagnosis, yet where favourable outcomes are still achievable with vigilant clinical reassessment and judicious intervention.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

REFERENCES

1. Lucia M, Viviana M, Alba C, Giulia D, Carlo DR, Porpora MG, et al. Neurological Complications in Pregnancy and the Puerperium: Methodology for a Clinical Diagnosis. J Clin Med. 2023;12(8):2994.
2. Magpie Trial Follow-Up Study Collaborative Group. The Magpie Trial: a randomised trial comparing magnesium sulphate with placebo for pre-eclampsia. Outcome for women at 2 years. Br J Obstet Gynaecol. 2007;114(3):300-9.
3. Otite FO, Patel SD, Anikpezie N, Hoffman H, Beutler T, Akano EO, et al. Demographic Disparities in the Incidence, Clinical Characteristics, and Outcome of

Posterior Reversible Encephalopathy Syndrome in the United States. Neurology. 2023;101(15):e1554-9.
4. Fugate JE, Rabinstein AA. Posterior reversible encephalopathy syndrome: clinical and radiological manifestations, pathophysiology, and outstanding questions. Lancet Neurol. 2015;14(9):914-25.
5. Ando Y, Ono Y, Sano A, Fujita N, Ono S. Posterior Reversible Encephalopathy Syndrome: A Review of the Literature. Int Med. 2022;61(2):135-41.
6. 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(8):494-500.
7. Bartynski WS. Posterior Reversible Encephalopathy Syndrome, Part 1: Fundamental Imaging and Clinical Features. Am J Neuroradiol. 2008;29(6):1036-42.
8. Fugate JE, Hawkes MA, Rabinstein AA. Posterior reversible encephalopathy syndrome: evolving insights in diagnosis, management, and outcomes. Lancet Neurol. 2025;24(9):789-800.
9. Brickman AM, Reitz C, Luchsinger JA, Manly JJ, Schupf N, Muraskin J, et al. Long-Term Blood Pressure Fluctuation and Cerebrovascular Disease in an Elderly Cohort. Arch Neurol. 2010;67:564-9.
10. Srichawla BS, Kaur T, Singh H. Corticosteroids in posterior reversible encephalopathy syndrome: Friend or foe? A systematic review. World J Clin Cases. 2025;13(12):98768.

Cite this article as: Awowole IO, Aderibigbe A, Eke U, Isah-Raji A, Adeniyi O, Komolafe M. Posterior reversible encephalopathy syndrome presenting as refractory postpartum seizures in a resource-constrained setting: a diagnostic dilemma and maternal near-miss from Nigeria. Int J Reprod Contracept Obstet Gynecol 2026;15:1799-802.