

CASE REPORT**Posterior Reversible Encephalopathy Syndrome: Three Ethiopian Hypertensive Patients Presented with Recurrent Seizure: Case Series and Literature Review**Zewde Yared^{1*}, Yifru Yared¹**OPEN ACCESS**

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ABSTRACT

BACKGROUND: *Posterior Reversible Encephalopathy Syndrome (PRES) is a potentially reversible neurological disorder of acute to subacute onset characterized by headache, nausea and vomiting, visual disturbance, seizure and altered mental status. Neuroimaging findings are characteristic, which allow early diagnosis in the appropriate clinical setting and enable to institute appropriate therapy timely.*

CASE PRESENTATION: *We report 3 adult patients with a history of hypertension presented with recurrent episode of seizure and altered mentation. While all the 3 patients were preliminarily diagnosed with Ischemic stroke, they were subsequently diagnosed with posterior reversible encephalopathy syndrome after neuroimaging revealed the typical features of the syndrome. They were started on antihypertensive and anticonvulsant drugs. On follow-up examination after 3-4 weeks, the patients showed marked clinical and neuro-imaging improvements.*

CONCLUSION: *Posterior reversible encephalopathy syndrome is a rare condition. The presenting clinical symptoms are non-specific and may mimic other neurological disorders. Therefore, early recognition of classic radiographic features is vital to the diagnosis. Timely diagnosis and treatment of this syndrome is important as the treatment outcome is mostly favorable.*

KEYWORDS: *Posterior Reversible Encephalopathy Syndrome, Stroke, Hypertension, Seizure*

INTRODUCTION

Posterior Reversible Encephalopathy Syndrome (PRES) is a rare neurological syndrome characterized by non-specific symptoms like headache, visual disturbance, seizures and altered mentation (1). Characteristic neuroimaging findings suggestive of vasogenic edema are non-enhancing, subcortical white matter abnormalities often seen bilaterally and predominantly in posterior cerebral hemispheres (2). In this report, we present three cases of PRES admitted to Tikur Anbessa Specialized Hospital over a year-long time period. All the three patients had uncontrolled hypertension and presented with new onset seizure and altered mentation.

CASE 1

A 17-year-old male, with a history of nephrotic syndrome and chronic hypertension for 4 years on prednisolone 10 mg and Enalapril 5 mg daily presented with recurrent attacks of generalized tonic clonic seizure and loss of consciousness for two hours. His blood pressure (BP) on admission was 180/100 mmHg and funduscopy revealed bilateral papilledema with blot hemorrhage on the right eye. The rest of neurologic and systemic examinations were normal.

He was started with anticonvulsant (diazepam and phenytoin), and antihypertensive (Nifedipine) was added. After stabilization, brain Magnetic Resonance Imaging (MRI) scan was taken; and it revealed T2 weighted hyperintense lesions in subcortical white matter involving the bilateral occipital, parietal, and high posterior frontal lobes without contrast enhancement, mass effect or associated diffusion restriction [Figure 1(A)]. Clinically, he recovered gradually, and follow-up non-contrast head computerized tomography (CT) scan taken after 3 weeks showed complete resolution of previously noted abnormal signals [Figure 1(B)]. The diagnosis of PRES was made, and he was discharged in a stable condition.

CASE 2

A 52-years-old female, known hypertensive patient for 2 years naive to treatment presented with sudden onset global headache and blurred vision followed by loss of consciousness and generalized tonic-clonic seizure at home.

At admission, her BP was 210/100mmHg, and antihypertensive drug was started. Her neurologic and general examination was normal. Brain MRI revealed symmetrical, T2-weighted hyperintense signals in bilateral occipital, parietal and posterior frontal lobes, without contrast enhancement [Figure 2(A& B)].

Tests for other causes of stroke were negative including Magnetic Resonance Venography (MRV). Her clinical condition improved and follow-up brain MRI after 4 weeks showed complete resolution of all white matter lesions [Figure 2(B)]. All these changes after optimal blood pressure control were consistent with PRES, and she was discharged in stable condition.

CASE 3

A 55-year-old female, known bronchial asthma and hypertension patient on follow-up at our hospital, was admitted for acute exacerbation of asthma and took Intravenous hydrocortisone. On the next day, her respiratory symptom improved, but later that day, she had sever headache, blurred vision followed by generalized tonic-clonic seizure and loss of consciousness. By the time, her BP was 150/100mmHg, and antihypertensive drug was titrated and loaded with Phenytoin.

Her brain MRI revealed bilateral, symmetrical T2 weighted hyperintensities in the subcortical white matter and overlying gray matter of posterior parietal and occipital lobes with out contrast enhancement [Figure 3 (A)]. On follow-up of brain MRI after 3 weeks, all previous abnormalities resolved completely [Figure 3(B)]. All the above changes were suggestive of PRES, and she was discharged improved.

DISCUSSION

The first case of PRES was described in 1996 by Hinchey et al (1). Recently, it is increasingly recognized due to more ready availability of MRI (3).

The causes of PRES are diverse, but the common precipitants are acute elevation of blood pressure, renal failure and treatment with immunosuppressive drugs (1,3). Our study revealed a similar etiology with more than one risk factor identified in two of our participants, demonstrating how PRES might be multifactorial.

PRES has been reported in almost all age groups but most frequently in young-or-middle-aged adults with female predominance, likely resulting from difference in vascular reactivity to various hormones (4). Similarly, our study found more females with PRES.

The most common clinical features are headache, vomiting, visual disturbance ranging from blurred vision to cortical blindness, and altered mentation including confusion, somnolence and coma. The onset is usually acute to subacute, and seizures are common initial symptoms which may begin focally but usually become generalized. The tendon reflexes are often brisk, and some patients have focal neurologic

deficits depending on the location of the lesion (1).

The pathogenesis of PRES is poorly understood, but it is postulated that impaired autoregulation of cerebral blood vessels, leading to cerebral hyperperfusion and blood-brain barrier dysfunction causing vascular leakage and edema mainly in the posterior circulation where the sympathetic innervation is reduced. Another mechanism is direct toxic effect on the endothelium by circulating endogenous (preeclampsia, sepsis) or exogenous (chemotherapy, immunosuppressive agents) substances (3).

The most common neuroimaging abnormality in PRES include edema without infarction involving the subcortical white matter in the posterior cerebral hemispheres, especially the bilateral parietal and occipital regions. The calcarine and paramedian occipital lobe structures are usually spared; a fact that distinguishes PRES from bilateral posterior cerebral artery infarction (1). This lesion appears as areas of hypoattenuation on CT scan, but MRI is the imaging modality of choice, which reveals T1-weighted hypointense and T2-weighted hyperintense areas, and iso- or-hypointense areas on diffusion weighted imaging (DWI) that partially or completely resolve on follow-up imaging. Involvement of other brain areas, such as the frontal lobes, brain stem, cerebellum and basal ganglia, have been reported in PRES patients (2).

Both clinical and imaging findings are non-specific. Therefore, the diagnosis of PRES is often made after excluding other mimickers like posterior circulation stroke, cerebral venous thrombosis, and leucoariosis. In the appropriate clinical scenario, a characteristic lesion on neuroimaging which showed a rapid clinical and imaging resolution with appropriate treatment is the hallmark of PRES (1,3).

Although reversible by definition, secondary complications, such as ischemic infarction or intracranial hemorrhage, have been reported. The prognosis of PRES is favorable. Most patients recover completely in few days with early recognition and prompt treatment of the underlying cause. Neuroimaging findings may persist for weeks, although most imaging findings have been reported to resolve within eight days to seventeen months after the first abnormality (1).

In conclusion, this case series highlights the need to consider PRES in the differential diagnosis of patients presented with acute neurological events and high blood pressure. To the best of our knowledge, this is the first case series on PRES reported from Ethiopia. Awareness of the characteristic neuro-imaging features of PRES is essential to avoid misdiagnosis and delayed treatment, since the clinical features are non-specific but the treatment outcome is mostly favorable.

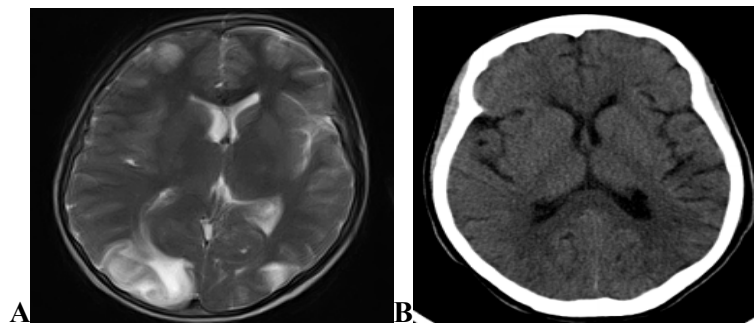


Figure 1: A) Axial T2-Weighted image shows a hyperintense signal in deep and subcortical white matter in high frontal, parietal and bilateral occipital regions B) Follow-up Non contrast brain CT showed complete resolution of previously noted abnormal signals.

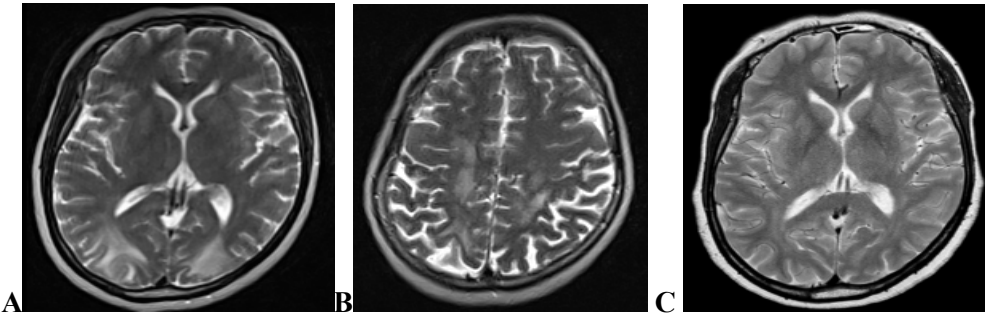


Figure 2: A and B) T2-weighted axial images shows hyperintense signals in subcortical white matter of bilateral occipital, parietal, and high posterior frontal lobes. B) Follow-up axial T2-weighted image shows complete resolution of all previously noted signal abnormalities.

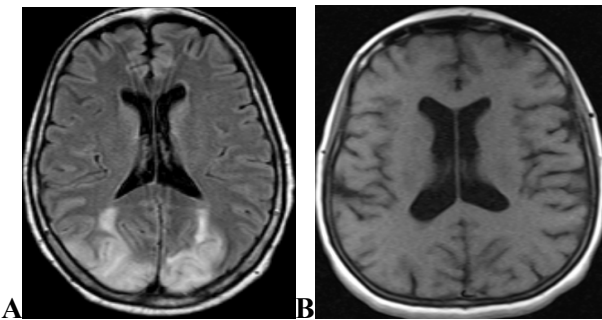


Figure 3: A) Axial FLAIR image shows hyperintense signal in subcortical white matter and overlying gray matter of bilateral posterior parietal and occipital lobes B) Follow-up axial FLAIR image shows complete resolution of bilateral parietal and occipital signal abnormalities.

REFERENCES

1. Hinchey J, Chaves C, Appignani B, et al. A reversible posterior leukoencephalopathy syndrome. *N Engl J Med*, 1996; 334(8): 494–500.
2. Mukherjee P, McKinstry RC. Reversible posterior leukoencephalopathy syndrome: Evaluation with diffusion-tensor MR imaging. *Radiology*, 2001;219:756–65.
3. Javed MA, Sial MS, Lingawi S, Alfi A, Lubbad E. Etiology of posterior reversible encephalopathy syndrome(PRES). *Pak J Med Sci*, 2005;21:149–54.
4. Hausser RA, Lacey DM, Knight MR. Hypertensive encephalopathy: magnetic resonance imaging demonstration of reversible cortical and white matter lesions. *Arch Neurol*, 1988;45(10):1078–1083.