







## Posterior reversible encephalopathy syndrome in HIV-positive patients: A case report

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### ABSTRACT

Posterior reversible encephalopathy syndrome (PRES) is a clinical and radiological syndrome in which patients present with an acute clinical presentation of seizures, headache, altered mental status, cortical blindness, and classic posterior cerebral edema on imagery. Patients with HIV are at risk for the development of this syndrome, but it is rarely reported. We report a case of a woman HIV-positive who was diagnosed with PRES.

**Keywords:** PRES, encephalopathy syndrome, HIV

## INTRODUCTION

Posterior reversible encephalopathy syndrome (PRES) is a neurotoxic syndrome characterized by headache, vomiting, seizures, cortical blindness, altered mental status, and classic posterior cerebral edema on radiographic imaging [1, 2].

It is diagnosed clinico-radiologically [1]. His mechanism is related to endothelial dysfunction and rupture of the blood-brain barrier and thus extravasation of plasma and macromolecules resulting in vasogenic edema. Its reversibility depends on the underlying cause and the speed of diagnosis and treatment [1]. HIV infection has been associated with PRES syndrome either via endothelial inflammation or immune activation [2, 3]. In this report, we present a case of a HIV-positive patient with PRES syndrome.

## CASE REPORT

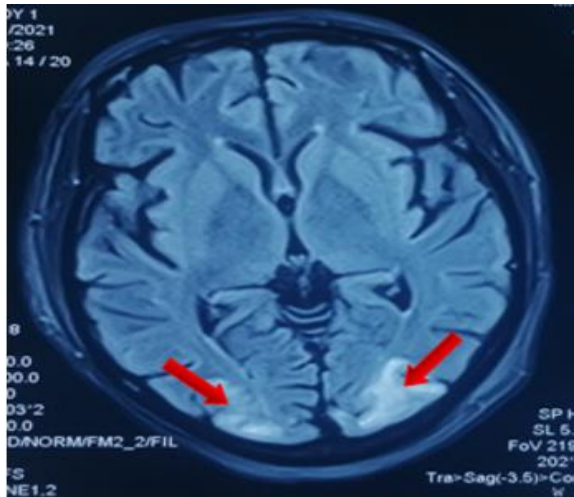
A 36-year-old woman, without medical history, presented to the emergency department with asthenia, vomiting, diarrhea, and dyspnea.

At admission, her Glasgow coma scale was 15/15. Although apyrexial, she was tachycardic (145 beats per minute). Her blood pressure was normal (130/70 mmHg), and her oxygen saturation was 100% on room air.

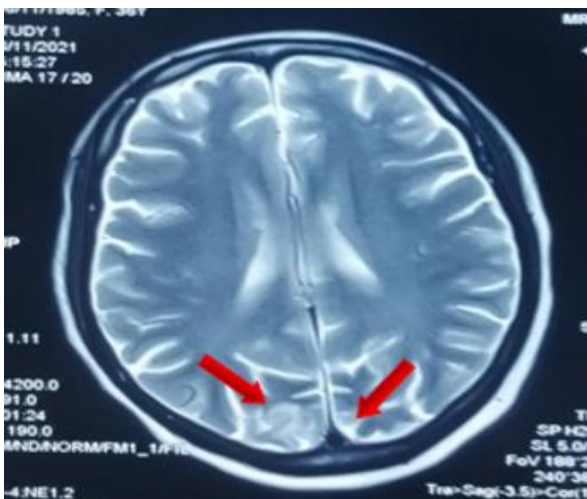
On questioning, she is celibate, but she has behaviors that put her at risk of STDs (unprotected sex). Physical examination revealed conjunctival heat, oral candidiasis, dry skin, skin folds, multiple genital ulcers and, diffuse abdominal pain. The remainder of his physical examination revealed no abnormalities.

Electrocardiography showed a negative T wave in V1 and V2 and, tachycardia. On initial laboratory evaluation, we found: hyperleukocytosis (14,000/mm<sup>3</sup>), anemia (6 g/dl), thrombopenia (63,000/mm<sup>3</sup>), renal failure with both raised creatinine (336 μM/L), and urea (16 mmol/L). A sternal puncture was normal. Cytobacteriological examination of the urine was positive for *Escherichia coli*. The blood cultures revealed the presence of *Salmonella enteritidis*. HIV serology was positive. Computed tomography (CT) of the body showed: a small pleural effusion, small intraperitoneal effusion and, multiple intraperitoneal adenopathies. The patient has been treated with cefotaxime (1g/8h) and metronidazole (500 mg/8h) and she received parental hydration. A few hours after admission, our patient presented severe hypertension up to 220/110 mmHg and lower limb edema followed by two generalized tonic-clonic seizures without loss of consciousness. Blood glucose, calcium, and magnesium levels were normal, but albumin is collapsed. Proteinuria and glucosuria were positive. CT of the brain was normal. Brain MRI showed signal abnormalities in the bilateral occipital and bilateral posterior frontal subcortical white matter. These anomalies are in FLAIR hyper signal, T1 hyposignal with discrete contrast after injection suggestive of PRES syndrome (**Figure 1** and **Figure 2**). The patient had received clonazepam (1 mg) after the seizures and nicardipine intravenously to reduce blood pressure.

The patient was stabilized after six days: blood pressure and renal failure were controlled, and lower limb edema was reduced with furosemide (20mg/8h). However, fluid diarrhea has persisted. CD 4 cell count was 16 cells/μl and HIV viral load was 258,000 copies/L. Malaria, cryptococcal antigen, syphilis, hepatitis B and C and toxoplasmosis screening were negative. Search for BK in sputum and urine was negative. CMV PCR in blood was positive. Abdominal CT showed pancolitis. We started foscavir (6g/12h) with a loading dose, antiretroviral therapy (tenofovir, emtricitabine, and efavirenz), and



**Figure 1.** FLAIR MRI brain showing bilateral occipital hyperintensities



**Figure 2.** MRI brain showing enhancement in the occipital lobe after injection of gadolinium

chemotherapy prophylaxis (cotrimoxazole). One week after, we noted an improvement of diarrhea but a reappearance of edema, worsening of his renal function, installation of hypokalemia rebellious to potassium supplementation without potassium bladder leakage, and visual disorders. A few hours after, she presented hemodynamic and respiratory instability requiring noradrenaline and high flow oxygen therapy and then the onset of recurrent generalized tonic-clonic seizures. Twenty-four hours later, the patient presented a cardiorespiratory arrest not recovered by resuscitation.

## DISCUSSION

Clinically, PRES syndrome's symptoms are on a wide spectrum of neurotoxicities such as altered vision, headache, confusion, seizures and, mental alteration. We cannot know exactly the etiology of PRES, which is likely to be multifactorial. PRES has been identified in multiple conditions such as immunosuppressive therapy, sepsis, autoimmune conditions like lupus erythematosus [1]. Recently, PRES has been reported in a case of SARS-Cov2 infection [4]. PRES may be associated with different pathologies, the most frequent are marked

arterial hypertension, acute renal failure, fluid retention and hyperkalemia. The association of arterial hypertension, renal failure, extracellular volume expansion and electrolyte imbalances may cause interstitial brain edema by increasing the permeability of capillaries and cytotoxic edema by direct injury of the brain parenchyma [5]. Our patient had a combination of associated conditions including an immunosuppression, acute hypertension, renal failure, systemic infection, and seizures, except hyperkalemia. On the contrary, she had severe hypokalemia, this can be explained by digestive losses by diarrhea. PRES is likely more common than currently reported among HIV patients [6]. Through advanced immune deficiency, our patient was more vulnerable to PRES due to chronic endothelial inflammation. Most cases reported were in patients with advanced HIV infection and CD4 cell count <200 cells/ $\mu$ L [7]. In a review of fourteen case reports of HIV-positive suffering from PRES, it was found that all patients had low CD4 cells count ranging from four to 287 cells/ $\mu$ L and many patients had either not been on antiretroviral therapy (ART) or had started only recently [8]. ART can also be associated with PRES manifestations like hypertension and brain edema by endothelial dysfunction directly or by way of metabolic derangements [9]. Weiss et al. reported a case of an HIV-positive patient who presented an immune reconstitution syndrome (IRIS) after the initiation of ART. Ten days after, this patient had PRES manifestations [2]. Through IRIS has not been yet described as a risk factor for PRES, it shares the characteristic systemic aberrant T-cell activation seen in other predisposing conditions [2].

MRI was essential in the diagnosis of PRES as well as the radiological improvement after blood pressure control. Findings in MRI imaging reveal hyperintense signaling on T2-weighted and fluid-attenuated inversion recovery (FLAIR) images and hypointense signaling in T1-weighted images, indicative of cerebral edema [8]. The regions most commonly affected are the parietal and occipital lobes bilaterally [5].

## CONCLUSION

Our observation underlines the importance of monitoring blood pressure and being vigilant for signs and symptoms of hypertension-related complications in HIV-infected patients especially in the advanced stage of the disease. So, high clinical suspicion is required to provide rapid treatment.

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