

Case Report

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# A Case of Posterior Reversible Encephalopathy Syndrome (PRES) with Chronic Renal Failure

Ibrahim Guney<sup>1\*</sup>, Vefa Oner<sup>2</sup>, Lutfullah Altintepe<sup>1</sup>, Ali Karagoz<sup>1</sup> and Raziye Yazici<sup>1</sup>

<sup>1</sup>Nephrology Department, Konya Research and Training Hospital, Konya, Turkey

<sup>2</sup>Radiology Department, Konya Research and Training Hospital, Konya, Turkey

## Abstract

**Objective:** The posterior reversible encephalopathy syndrome (PRES) is a clinical and radiological entity.

**Methods:** We present a patient with predialysis CRF and PRES.

**Patient:** A 16-year-old man with predialysis CRF admitted with headache and confusion to emergency clinic. Cranial and diffusion magnetic resonance imaging (MRI) were studied and PRES signs were seen in the bilateral parieto-occipital region.

**Results:** All PRES signs of the patient improved at the 3<sup>rd</sup> month.

**Conclusion:** PRES in the patients with CRF was known as uncommon condition. But PRES should be brought to mind, if there is an uremic and/or hypertensive encephalopathy.

**Keywords:** Hypertension; Posterior reversible encephalopathy syndrome; Renal failure

## Introduction

The posterior reversible encephalopathy syndrome (PRES) is characterized by headache, seizures, altered mental status and visual disturbances. It is a clinical and radiological entity and typically causes the reversible changes in the posterior circulation system of the brain [1]. The most common causes of PRES are hypertensive encephalopathy, eclampsia-preeclampsia, drug intoxications (immunosuppressive and cytotoxic drugs) and chronic renal failure (CRF) with hypertension, collagen vascular disease, thrombotic thrombocytopenic purpura, human immunodeficiency virus (HIV) infection, acute intermittent porphyria, and organ transplantation [2-4]. We present a patient with predialysis CRF and PRES.

## Case Report

A 16-year-old man with predialysis CRF admitted with headache, nausea, vomiting, confusion and fatigue to emergency clinic. He admitted to nephrology clinic for high creatinemia (4.8 mg/dl) and malign hypertension (blood pressure is 230/110 mmHg). Tonic-clonic Seizures involving his arms and legs were observed. He did not have findings of meningeal irritation. His visual acuity was decreased, he could perceive hand movements. His pupils isochoric, and direct and indirect light reflexes were normal. Motor and sensory examinations were normal. Deep tendon reflexes were equal and normoactive bilaterally, and Babinski sign was absent bilaterally.

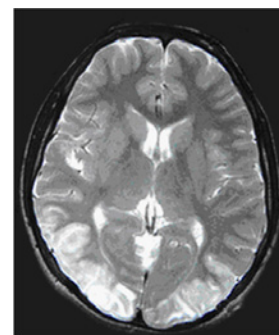
His laboratory tests on admission were as follows: hemoglobin, 12 g/dl; white blood cells, 11.2 K/ $\mu$ L; platelets, 155 K/ $\mu$ L; urea, 133 mg/dl; creatinine 4.8 mg/dl; Na, 135 meq/L; K, 6.8 meq/L; Ca, 8.2 mg/dl; albumin, 3.5 mg/dl; SGOT, 12 U/L; SGPT, 11 U/L;

Cranial and diffusion magnetic resonance imaging (MRI) were studied and PRES signs (hyperintense lesions were observed on T2A and FLAIR sequence) were seen in the bilateral parieto-occipital region (Figure 1). After the anti-hypertensive, anti-edema and anti-epileptic treatments, all signs of the patient improved at the 3<sup>rd</sup> day. In addition, all PRES signs of the patient also improved at the 3<sup>rd</sup> month (Figure 2).

## Discussion

The posterior reversible encephalopathy syndrome (PRES) is characterized by headache, seizures, altered mental status and visual disturbances. It is a clinical and radiological entity and typically causes the reversible changes in the posterior circulation system of the brain, because the posterior cerebral arterial circulation has a lower level of sympathetic innervation [1].

Two possible mechanisms have been proposed in the pathophysiology of PRES. The first is vasospasm due to acutely increased blood pressure: it has been suggested that vasospasm contributes to ischemia and



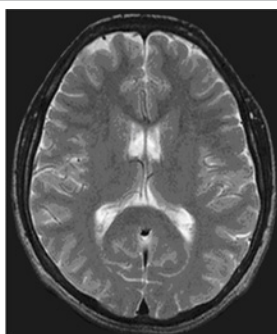
**Figure 1:** Axial T2 MR image show symmetrically located, bilateral hyper intense lesions in the parietooccipital regions affecting the cortex and sub cortical white matter.

**\*Corresponding author:** Ibrahim Guney, Nephrology Department, Konya Research and Training Hospital, Konya, Turkey, Tel: 0090-3323236709; E-mail: [driguney@hotmail.com](mailto:driguney@hotmail.com)

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**Figure 2:** After the treatment (3<sup>rd</sup> month), all of lesions completely disappeared in axial T2 inversion.

cytotoxic edema at regions of the arterial border zone [5]. The second, more recent hypothesis is supported by diffusion images suggesting that dilatation develops in cerebral arterioles due to autoregulatory failure. Cerebral autoregulation keep blood flow constant, and protect the brain during changes in blood pressure; but, sudden and severe increases in blood pressure can impair autoregulation, and this impairment can lead to arteriolar vasodilatation and endothelial dysfunction. In conclusion, plasma and red blood cells migrate to the extravascular space from the intravascular space, and vasogenic edema occurs [6].

The most common causes of PRES are hypertensive encephalopathy, eclampsia-preeclampsia, drug intoxications (immunosuppressive and cytotoxic drugs) and chronic renal failure (CRF) with hypertension, collagen vascular disease, thrombotic thrombocytopenic purpura, human immunodeficiency virus (HIV) infection, acute intermittent porphyria, and organ transplantation [2-4].

PRES can be diagnosed with cranial MR-images. On MR-images, bilateral symmetrical edema in the parieto-occipital region, supplied

by the posterior cerebral circulation, is hyperintense on T2-weighted and fluid-attenuated inversion recovery (FLAIR) sequences, and hypointense on T1-weighted sequences [4].

In the treatment of PRES, regulation of blood pressure and seizures are important. Mean arterial pressure should be reduced 20-25% within the first 1-2 hours. Rapid decrease in blood pressure should be avoided due to the risk of hypoperfusion and consequent cerebral infarction.

PRES mostly is a benign and reversible condition, especially when the causative factor like hypertension can be eliminated. Although PRES can be diagnosed with MR-images, suspicion must be raised by the clinician. Both should be familiar with this underdiagnosed, clinically frightening syndrome to avoid persistent deficits.

As a conclusion, PRES in the patients with CRF was known as uncommon condition. But PRES should be brought to mind, if there is an uremic and/or hypertensive encephalopathy.

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