Case Report

Posterior Reversible Encephalopathy Syndrome in the Emergency Service

M Yigit, E Yigit[1], KA Turkdogan[2]

Department of Emergency Medicine, Haseki Training and Research Hospital, University of Health Sciences, [1]Department of Emergency Medicine, Sisli Hamidiye Etfal Training and Research Hospital, University of Health Sciences, Istanbul, [2]Department of Emergency Medicine, Adnan Menderes University, Aydin, Turkey

Date of Acceptance:
11-Jun-2017

ABSTRACT

Posterior reversible encephalopathy syndrome (PRES) is an entity characterized by headache, altered mental status, seizures, visual disturbances, and focal neurological signs. The most common etiologies of PRES are hypertension and renal failure, and the most frequent pathophysiology is hyperperfusion. PRES is generally symmetrical, often in the occipital and parietal lobes, and is typically characterized by vasogenic edema in the subcortical white matter. This study involves a 38-year-old female patient who had hypertension, used immunosuppressive drugs and was also found to have nephropathy. After 3 months of treatment for PRES, the patient's symptoms had declined.

KEYWORDS: *Emergency service, hyperperfusion, posterior reversible encephalopathy, vasogenic edema*

INTRODUCTION

The clinical radiological situation known as posterior reversible encephalopathy syndrome (PRES) or reversible posterior leukoencephalopathy syndrome was first defined by Hinchey *et al*. in 1996. The syndrome is characterized by headache, altered mental status, seizures, visual disturbances, and focal neurological signs. Neuroradiological imaging is usually decimetric and is typically characterized by vasogenic edema in the subcortical white matter in the occipital and parietal lobes.[1,2] Although systemic hypertension is the most common cause of PRES, systemic infections, preeclampsia/eclampsia, malignancies, chemotherapy, and immunosuppression are also considered in establishing the etiological factors of autoimmune diseases (systemic lupus erythematosus and Wegener's granulomatosis).[3-6] Treatment is based on eliminating the causes for the occurrence of the disease.[1] Our aim is to show that PRES should be considered in patients who have a variety of neurological symptoms but do not present lateralized evidence and also to provide discussion accompanied by the literature.

CASE REPORT

A 38-year-old female patient was brought to the emergency room by her relatives because she had fainted in the bathroom. After fainting, the patient had had contractions in her arms and hands and had become drowsy. Her medical history revealed that she had undergone a total thyroidectomy 3 months earlier and had been using L-thyroxine. She had been taking 12 mg/day of prednisolone for 1 month following a diagnosis of nephropathy in the presence of protein-losing. Additionally, her blood pressure measurements had been high in the week before she was brought to the emergency room. The patient had never fainted before this. Throughout the physical examination, the patient was conscious and was open, cooperative, and oriented. Her blood pressure was 179/105 mmHg, pulse rate was 92 beats per minute, respiratory rate was 14 breaths per minute, body temperature was 37.1°C, oxygen saturation by pulse oximetry was 95% while breathing room air, and peripheral capillary blood glucose was 114 mg/dL. The patient's white blood cell count was 9.16 x 103/μL, and her laboratory values also revealed the

Address for correspondence: Dr. M Yigit, Department of Emergency Medicine, Haseki Training and Research Hospital, University of Health Sciences, Millet Street, 34096, Fatih, Istanbul, Turkey.
E-mail: yigitacil@gmail.com

Access this article online

Quick Response Code:

Website: www.njcponline.com

DOI: 10.4103/njcp.njcp_120_16

How to cite this article: Yigit M, Yigit E, Turkdogan KA. Posterior Reversible Encephalopathy Syndrome in the Emergency Service. Niger J Clin Pract 2018;21:248-50.

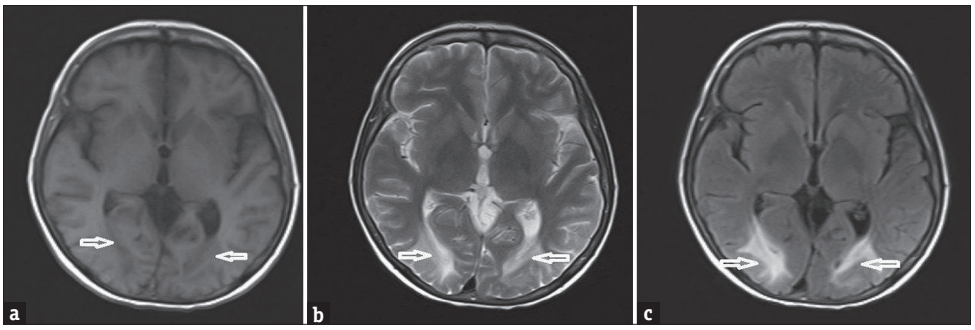

Figure 1: In both cerebellar hemispheres, in the bilateral occipital lobes, and in the supratentorial space, frontal and parietal white matter localization of hypointense T1 (a), T2 (b) and fluid attenuation inversion recovery (c) hyperintense multiple signal changes were observed through magnetic resonance imaging

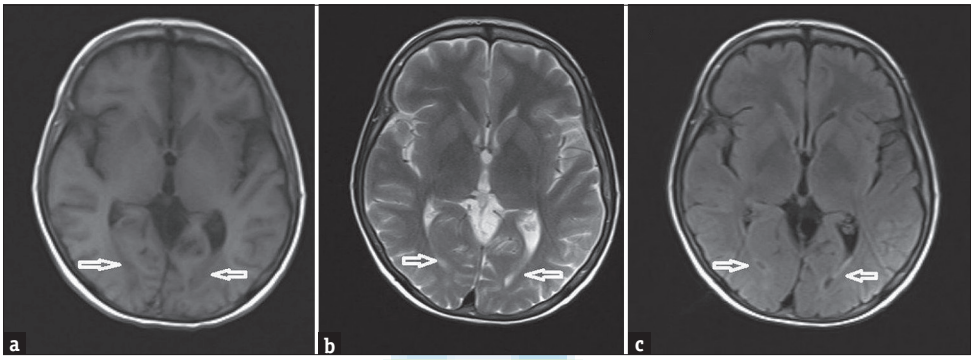

Figure 2: (a-c) At the end of 3 months of cranial magnetic resonance imaging monitoring, the findings indicating posterior reversible encephalopathy syndrome had decreased

following: hemoglobin of 9.3 g/dL, hematocrit of 29.2 %, platelet of 358 K/mL, glucose of 74 mg/dL, urea of 87 mg/dL, creatinine of 2 mg/dL, total calcium of 7.6 mg/dL, sodium of 136 mmol/L, potassium of 3.3 mmol/L, total protein of 4.2 g/dL, albumin of 1.8 g/dL, thyroid-stimulating hormone of 10.26 mlU/L, free triiodothyronine of 0.67 pmol/L and free thyroxine of 6.95 pmol/L. Brain computed tomography showed vasogenic edema and reduced density in the white matter, which is consistent with what is seen in the bilateral parieto-occipital lobes of patients with PRES. Cranial magnetic resonance imaging (MRI) revealed that in both cerebellar hemispheres, in the bilateral occipital lobes, and in the supratentorial space, there was frontal and parietal white matter localization of hypointense T1, T2 and fluid attenuation inversion recovery (FLAIR) hyperintense multiple signal changes [Figure 1a-c].

Based on these findings, the patient was considered to have PRES and was hospitalized. Antihypertensive therapy was initiated during the hospitalization, and the patient's L-thyroxine medication dose was adjusted. After obtaining the renal biopsy results, the patient was given Endoxan as a supportive treatment following the diagnosis of IgA nephropathy. The patient showed no neurological symptoms during her 12 days in critical care and was discharged for outpatient follow-up. She remained stable throughout the 3-month follow-up, which included cranial MRI monitoring, and at the end of this time, the findings indicating PRES had decreased [Figure 2a-c].

Discussion

PRES is a clinico-radiological situation that was identified early in 1996 and is accompanied by focal neurological symptoms such as headache, nausea, vomiting, confusion, and visual disturbances and for which the pathophysiological mechanism is still not fully understood.[7] Although the most common etiologies are hypertension and renal failure, autoimmune diseases, immune deficiency, pregnancy, immunosuppressive drug use, sepsis, and infection of the central nervous system are also considered to be causes.[1,5,8] In the pathophysiology, the most common hyperperfusion theories are accepted. Therefore, it is important to try to keep the cerebral blood flow through the blood–brain barrier constant. The arteriolar system plays a role through the effects of the sympathetic system. In response to the increased systemic blood flow, the brain blood flow is kept stable through contraction of the arterioles with the sympathetic system effect. However, when the blood pressure exceeds a certain value, synthesis arterioles can no longer contract and become dilated. As a result, fluid, erythrocytes, and macromolecules seep into the brain parenchyma. Because

of the way in which the brain cortex is organized, even though it shows resistance to the edema, the blood–brain barrier is easily damaged and the edema can spread to subcortical areas. This explains why the sympathetic system is weaker in terms of the posterior circulation and the effect is more pronounced in this region.[9]

In our case, when we considered the etiological factors, we found hypertension, the use of immunosuppressive drugs, and nephropathy, all of which can be seen as underlying causes of PRES. In a study conducted by Demirtaş *et al*., patients with PRES were divided into three pathophysiological groups based on the features of their cranial imaging.[10] The first group included patients with lesions located in the occipital lobe and in whom hypertension-induced phenomena were involved. In the second group, the patients had normal blood pressure and their endothelial damage was in the pathophysiology although their lesions were in similar regions to those of the first group. The third group included patients with normal blood pressure, the presence of symmetrical basal ganglia involvement that had affected the thalamic, cerebrum, and brain stem, and the prominent presence of endothelial damage due to metabolic causes. In our case, there were lesions in the parieto-occipital lobes, and the patient had had a long-standing history of high blood pressure. Based on these findings, our patient can be categorized as being in the first group. Cranial MRI was used for diagnosis. The presence of hyperintense lesions in the cortical and subcortical areas of the posterior cerebral regions is characteristic in FLAIR and T2 images of patients with PRES. It is not possible to differentiate between cytotoxic and vasogenic edema on T2-weighted images, but this distinction can be detected by diffusion-weighted apparent diffusion coefficient (ADC) images. Therefore, ADC mapping must be used when PRES is being considered in a patient's diagnosis.[11,12] In our case diffusion-weighted images, the T2 and FLAIR images were used for the diagnostic phase. Treatment included regulation of the patient's blood pressure, treatment for the underlying etiological factors, and control of seizures. Full recovery was achieved by restoring autoregulation. Our patient was discharged with complete neurological recovery using treatment that was aimed at the cause as well as through supportive treatment.

Conclusion

Consequently, PRES may be encountered in the emergency department, with its pathophysiological mechanisms and clinical presentation. If physicians working in emergency rooms know about the clinical and radiological findings, diagnosis, and treatment of these patients and are able to provide patient management, this might reduce the complications that can develop because of the loss of critical time.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

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