Case Report
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Posterior Reversible Encephalopathy Case In Emergency Department

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Abstract

Posterior reversible encephalopathy syndrome (PRES) is a clinical condition with neurological symptoms such as headache, seizure, nausea-vomiting, and visual impairment. The most common cause is hypertension. In magnetic resonance imaging (MRI), hyperintensity due to vasogenic edema is observed in the posterior cerebral areas. In this case, we aimed to present a PRES case with newly diagnosed hypertension.

Key words: Emergency service, intensive care, posterior reversible encephalopathy syndrome, vasogenic brain edema

Introduction

Posterior reversible encephalopathy syndrome (PRES) was first described in 1996 by Hinchey et al.[1]. According to this definition; Patients have imaging findings with edema in the posterior cerebral white matter accompanying symptoms such as headache, nausea, vomiting, altered state of consciousness, seizures, and visual impairment[1,2]. Clinical and imaging findings are generally reversible[3]. Occipital and parietal lobe involvement is frequently seen on imaging[4]. Its etiology includes hypertension, renal failure, eclampsia, vasculitis, use of immunosuppressive and immunomodulatory drugs, and chemotherapeutic drugs[3-5]. Treatment; It may be possible by eliminating the etiological cause[6]. Our aim in this case; To discuss the possibility of PRES in a patient who comes to the emergency department (ED) with various neurological symptoms in the light of the literature.

Case

A 28-year-old male patient was brought to the emergency room with complaints of seizure and loss of consciousness. The glasgow coma scale score of the patient was 13, and he was not stable and looked like toxic. The patient's relatives had a history of having a headache for 1 month. He was experienced syncope and had tonic clonic contractions for three minutes on the road today. The patient had no history of any illness, and he did not use any medication. On admission he had a blood pressure of 207/134 mmHG, pulse 145 / min, respiratory rate 34 / min, and fever 38.1 C in his vital signs. On physical examination, he was confused. Neurological examination revealed no lateralizes deficits. He had not neck stiffness and kerning brudznski signs were negative. Other systemic examinations did not reveal any pathology.

After inserting a bladder catheter was to the patient, macroscopic hematuria was observed. During the observation of the patient in the ED, he had another seizure; iv diazem stopped the seizure activity. In the laboratory findings of the patient, urea 128 mg / dL (18-55 mg / dL), creatinine 4.26 mg / dL (0.50-1.40 mg / dL), uric acid 16.5 mg / dL (35, -7.2 mg) / dL), white blood cell 17.4 10 ^ 9 / L (4-10.5 10 ^ 9 / L), hemoglobin 14.9g / dL (13.5-18 g / dL), platelet 310 10 ^ 9 / L (150-450 10 ^ 9 / L), blood gas ph 7.42 (7.35-7.45), PCO2 32mmhg (35-48mmhg), lactate 1.5mmol / L (0.9-1.7mmol / L) was seen. No acute pathology was found in the brain computed tomography (CT) of the patient in the ED.

The patient underwent lumbar puncture (LP) due to fever and mental status change. Protein, glucose and cell counts of the cerebrospinal fluid (CSF) were within the normal limits. The PCR examination of the viral panel was negative. The patient's urine was examined for the purpose of any drug or sustance misuse and no drug was detected.Hyperintensity in T2 AG and FLAIR sequences are observed in cortical-subcortical and periventricular areas in the right cerebellar hemisphere and both occipitoparietal regions In brain MRI. After ıv Gadolinium infusion, slightly expansile pathological signal changes with indistinct borders and no significant enhancement were observed (Figure 1).

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The patient was admitted to the emergency critical intensive care unit with the diagnosis of posterior reversible encephalopathy with current findings. Intravenous nicardipine infusion was initiated for blood pressure regulation and midazolam infusion for sedation.The patient was followed up with intravenous treatment for 3 days, and after blood pressure regulation was achieved, sedation was discontinued and oral antihypertensive treatment was started. In the renal ultrasonography performed during the intensive care follow-up, grade 2 increase in renal parenchyma was observed and 10 g proteinuria was detected in 24-hour urine. Control brain MRI lesions taken during follow-up were observed to regress (Picture 2). During the intensive care follow-up, the patient who did not have any seizures and blood pressure was regulated. The patient was transferred to the nephrology clinic for evaluation nephropathies.

Discussion

PRES as a clinical and radiological syndrome is first described by, Hinchey et al in 1996. The pathophysiology of the diesease is not fully understood. The diesease is characterized with focal neurological symptoms such as headache, nausea, vomiting, confusion, seizures, and visual impairment[5]. Our patient is also admitted to headache, seizure and confusion.

Hypertension is The most common cause of PRES[2]. Autoimmune diseases, kidney failure, immunosuppressive treatments, electrolyte disturbances, sepsis, and pregnancy may also cause PRES[2]. Although our patient had no hypertension in his past medical history, his blood pressure values were measured high.

Imaging is essential in diagnosis. Computed tomography of the brain often detects neuroradiographic anomalies, but the best imaging method is magnetic resonance imaging (MRI). On MRI, hyperintensity in T2W and flair sequences, characterized by bilateral, asymmetric, vasogenic edema, typically involving the posterior cerebral hemisphere, especially parietooccipital area, are observed[6]. The lesions are rarely seen in the cerebellum, thalamus, brain stem and spinal cord[6,9]. In our case, lesions were observed in the right cerebellum and parietooccipital region.

The most widely accepted theory in pathophysiology is central hyperperfusion. In normal cerebral vascular autoregulation, arterioles respond to vasoconstriction, dilatation, and vasoactive substances (such as nitric oxide, thrombox-

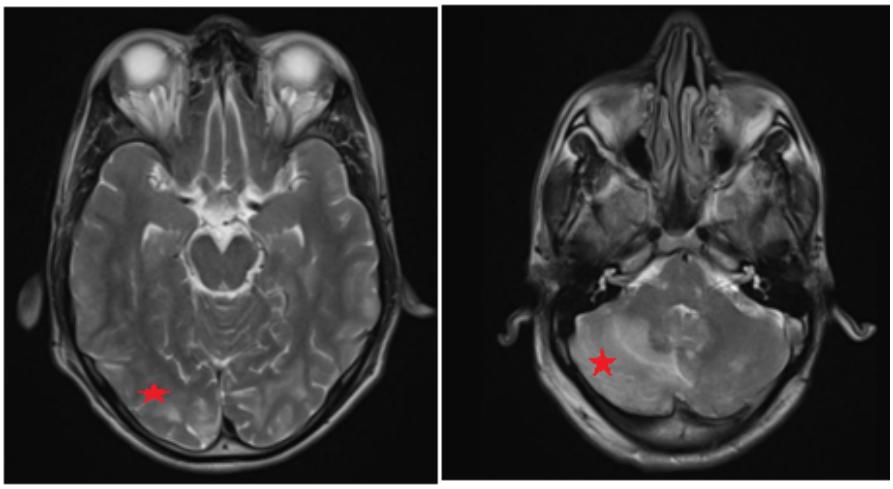

Figure 1: Hyperintensity in T2 Flair sequence on brain MRI.

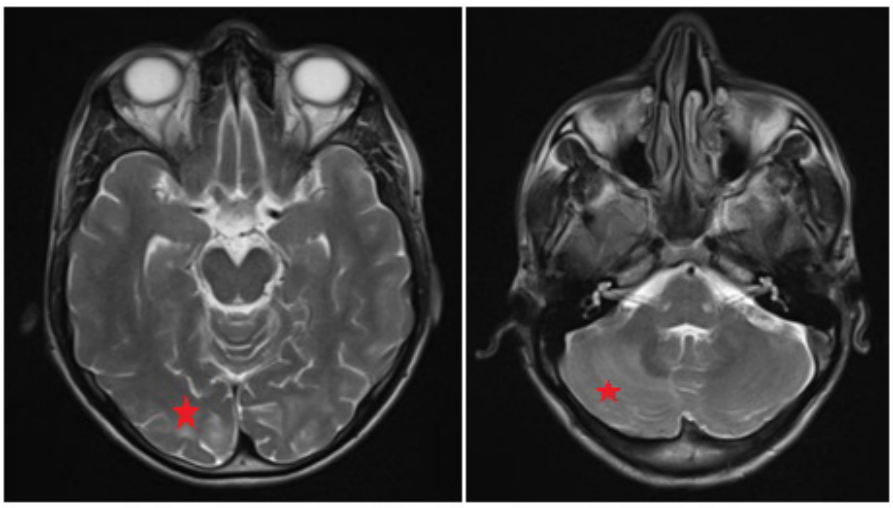

Figure 2: Reduced hyperintensity in the T2 Flair sequence in brain MRI compared to figure 1.

ane A2, endothelin-1) secreted from the endothelium against systemic blood pressure changes[6]. Therefore, some literature suggest that hypertension alone is not a sufficient reason for PRES[6-8]. Sudden blood pressure changes or direct damage of cytotoxic agents to the endothelium cause blood-brain barrier disruption, vascular leakage and brain edema[6]. In our case, the patient's initial blood pressure values were 207 / 134mmHg in our ED.

Angiographic studies revealed that while hypertensive crisis occur in pres, posterior system arteries became vasospastic[9]. In many studies, fibrinoid necrosis, interstitial edema and petechial microhemorrhages were observed in the arteriole walls, but no infarction finding was found [9,10]. sudden changes in blood pressure is accepted to be responsible for the pathogenesis of this syndrome, the there are some cases reported without severe hypertension[10].

PRES lesions are mostly located in parietooccipital area (70%) fed by the posterior cerebral circulation[4,6]. İnvolvement of the cerebellum, brainstem, basal ganglia, thalamus, spinal cord, and corpus callosum has also rarely reported. In our case, hyperintense lesions were observed bilaterally in the parietooccipital and right cerebellum.

Imaging and clinical signs are not specific for PRES diagnosis.

The diagnosis is made by excluding possible etiologies and evaluating risk factors (hypertension, renal insufficiency, immunosuppressor therapy ... etc)[5].

The possible etiologies in the differential diagnosis list of PRES may listed as bilateral posterior lobe infarctions, cerebral venous thrombosis, herpes virus and other encephalitis, cerebral vasculitic involvement, mitochondrial encephalopathy, hypertensive encephalopathy, hypoglycemia, and hyponatremia[2].

These diagnoses were also considered in the differential diagnosis list in our patient, and this work-up was made by the help of neurological examination, laboratory tests, central imaging (BBT and MRI) and LP.

The patient was diagnosed with PRES because the MRI examination of the patient had typical features for PRES.

Quick diagnosis and prompt treatment are very important in evaluation of PRES. Symptoms are often reversible. Correcting the underlying cause of the disease is the mainstay of the teratment. Regardless of the etiology, hypertension occurs in the majority of cases, and blood pressure regulation allows the patient to recover dramatically. The use of easily titrated parenteral antihypertensives such as nicardipine and labetol is recommended in the treatment[5]. Seizures are frequently observed in PRES patients. The choice of anticonvulsant agent should be made, taking into account the patient's kidney functions, the need for sedation and his/her accompanying diseases[5]. In our patient, nicardipine was used for blood pressure regulation and midazolom was used as an anticonvulsant and sedative. It has been reported that clinical and radiological findings usually disappear between 1 and 4 weeks in PRES[10]. In our patient, after blood pressure regulation was achieved, the symptoms has resolved and hyperintense images decreased in the MRI investigation one week later.

Conclusion

PRES should be considered in the differential diagnosis of hypertensive patients presenting with acute neurological deficits in the ED. Early diagnosis and treatment is crucial and the recovery of patients without sequelae depends on this approach.

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