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Case Report

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Pres associated with naltrexone implant

Abstract

Posterior reversible encephalopathy syndrome (PRES) has been defined as a neurological syndrome diagnosed with a combination of clinical and neuroimaging findings.
A 31-year-old female patient with no known history of the neurological disease was diagnosed with PRES as a result of physical examination and imaging.
The patient had no other findings other than heroin use and related naltrexone implant. Although it has been reported that PRES is caused by various etiological reasons; no information has been found about whether it will be related to the naltrexone implant used in the treatment of people addicted to heroin.
With this case, we wanted to remind PRES again and state that naltrexone implant, which we have not seen before in the literature, may also be in the differential diagnosis.

Keywords: Emergency service, epileptic seizures, PRES syndrome, naltrexone implant

INTRODUCTION

Posterior reversible encephalopathy syndrome (PRES) was first described by Hinchey in 1996 and is a clinical condition characterized by a combination of clinical and neuroimaging findings, which attracted attention with the reporting of similar cases in the following years (1). Patients may present with headaches, impaired consciousness, seizures, visual abnormalities, nausea, vomiting, and focal neurological deficits (1). In neuroimaging, it is characterized by vasogenic edema involving bilateral cortical/subcortical regions affecting the parietal and occipital regions, followed by the involvement of other regions (1-5). With this case, we wanted to remind the approach to PRES syndrome, which presented with epileptic seizure and bilateral frontoparietal and bilateral occipital involvement.

CASE REPORT

A 26-year-old female patient was admitted to the emergency department with the complaint of a generalized tonic-clonic seizure. The patient, who did not have an epilepsy history before, was loaded with diazepam 0.2 mg/kg intravenous with no response, an additional dose of 0.1 mg/kg intravenous diazepam, and a loading dose of 20 mg/kg intravenous phenytoin and maintenance was given, and the epileptic seizure was stopped. According to the information obtained from the relatives of the patient; the patient was single, living with his family, and had been followed for about 10 years due to heroin use, and she had a history of suboxone drug use since 2012. In addition, it was learned that 2 days ago, an opioid antagonist naltrexone implant was inserted subcutaneously into the abdominal region at the National Drug and Alcohol Treatment Center (AMATEM) and epileptic seizures began to appear following implantation. The patient's blood pressure at admission was 110/60 mmHg, heart rate was 85 beats/minute, and oxygen saturation was measured as normal. Blood biochemical parameters (blood glucose, liver and kidney function, blood electrolytes) were within normal range. No signs of infection were found in the blood. Pathological findings other than increased anion gap

in blood gas Hemoglobin and blood iron levels were within the normal range. In the patient's EEG rare, moderate-high amplitude 5-6 CYC/SEC slow wave discharges were observed to occur as diffuse, paroxysmal, bilateral, and synchronous, and the presence of a mildly active paroxysmal anomaly was detected.

Neurological examination of the patient who was evaluated after the postictal period was moderate-good, eyes open spontaneously; bilateral pupils isochoric, fulfilling simple orders and four extremities spontaneously active and no additional pathological finding was found in her neurological examination.

No infarct or bleeding findings were found in the cranial CT. As cranial MRI findings; T2AG images showed hyperintensity at the level of the bilateral centrum semiovale, left frontal, and both temporoparietooccipital cortical areas (figure 1). At cranial MR venography superior and inferior sagittal sinuses, bilateral transverse and sigmoid sinuses had normal signals and their contours were regular. Bilateral internal cerebral veins, the vein of Galen, sinus recti, and visible cortical veins were found to be patent. The patient was admitted to the neurology service and followed with phenytoin therapy. The general condition of the patient improved. The EEG taken 6 days after admission to neurology service was within normal limits and the patient was discharged with outpatient control. Removal of the naltrexone implant was not considered due to the patient's clinical improvement. It has been reported that undesirable neurological effects may occur with the use of pregabalin and alcohol in naltrexone-implanted patients; there is no information regarding the epileptogenic effects of naltrexone. This case suggests that new studies are needed about the epileptogenic effect of the naltrexone implant.

DISCUSSION

Common etiological factors of PRES syndrome include blood pressure fluctuations, preeclampsia/eclampsia, kidney failure, cytotoxic agents, and autoimmune conditions (6,7). Although cases have been reported in all age groups, it has been observed that it is mostly seen in the young middle-aged group. Pathophysiology of PRES syndrome is thought to be due to endothelial changes and cytokines released from the endothelium, and injuries that develop due to these two mechanisms are thought to be responsible for PRES syndrome. The most accepted theory is the "vasogenic theory" proposing that rapidly developing hypertension damages cerebral autoregulation which cannot be recruited adequately, resulting in deterioration of the blood-brain barrier and secondary vasogenic edema. As a result of the rapid and severe increase in blood pressure, the auto-regulatory response cannot be recruited sufficiently, causing hyperperfusion and extravasation of plasma and macromolecules (7).

PRES symptoms are non-specific, encephalopathy and seizures are the most common symptoms, followed by visual disturbances, headache, and focal neurological deficits (7, 8). Seizures usually occur in the early stages of the disease and are seen in 74-87% of patients (6, 7). Various types of seizures can be seen in these patients. In some cases, status epilepticus may be the presenting symptom of PRES (9). In most cases, seizures are terminated spontaneously or with the use of antiepileptic therapy.

There is currently no gold standard diagnostic test. Brain imaging is the cornerstone of confirming the diagnosis of PRES. Although vasogenic edema can be visualized on non-contrast computed tomography (CT) in some patients, brain MRI, especially T2-weighted and fluid-attenuated inversion recovery (FLAIR) sequences is much more sensitive compared to tomography imaging (10). We could not detect any pathology in the cranial CT of our patient, and the diagnosis of PRES was made with cranial MRI T2-weighted imaging.

PRES was initially defined as a reversible benign entity with a good outcome, mortality was observed in 19% of the patients, and varying degrees of functional impairment were reported in 44% of the patients (11,12). Some outcomes that require long-term care include epilepsy and motor deficits. Severe cases require aggressive supportive care in the intensive care unit (ICU). Despite a widespread myth about its benign course and reversible nature of disease both clinically and radiologically, permanent brain damage, severe functional disorders, and mortality have been reported (6, 11,12).

Suboxone has a half-life of approximately 30 hours and is long-acting due to its slow dissociation from the receptor (13). Since our patient did not have regular suboxone use, a naltrexone implant was considered, suggesting that discontinuation or initiation of suboxone had no effect on seizures. Although seizures have been reported very rarely due to the use of suboxone in the literature, no information has been found about pressing due to suboxone and naltrexone implants.

Management is primarily supportive and intravenous anticonvulsants if needed; first-line diazepam, second-line fosphenytoin, and phenobarbital are recommended. In resistant cases, propofol, pentobarbital, midazolam are recommended.

In addition, continuous EEG monitoring is recommended in these patients. No findings were found in the control EEG of our patient, who was followed up in line with these clinically recommended approaches.

CONCLUSION

Although it is defined as a benign clinical condition, PRES syndrome can also lead to serious conditions that can result in permanent brain damage. Patients may apply to the emergency department with status epilepticus or other epileptic forms. There is no information that PRES Syndrome, which has been reported to develop due to some underlying risk factors, may be due to naltrexone implant. For this reason, more detailed clinical studies are needed.

Patient informed consent
Informed consent was obtained from family of the patient prior writing this case report.

Conflict of interests
The authors declare that there is no conflict of interest in the study.

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REFERENCES

1. Hinchey J, Chaves C, Appignani B, Breen J, et al. A reversible posterior leukoencephalopathy syndrome. N Engl J Med. 1996;334:494–500.
2. Schwartz RB, Jones KM, Kalina P, Bajakian RL, et al. . Hypertensive encephalopathy: findings on CT, MR imaging, and SPECT imaging in 14 cases. AJR Am J Roentgenol. 1992;159:379–83.
3. Schwartz RB, Bravo SM, Klufas RA, Hsu L, et al. Cyclosporine neurotoxicity and its relationship to hypertensive encephalopathy: CT and MR findings in 16 cases. AJR Am J Roentgenol. 1995;165:627–31.
4. Bartynski WS, Grabb BC, Zeigler Z, Lin L, et al. Watershed imaging features and clinical vascular injury in cyclosporin A neurotoxicity. J Comput Assist Tomogr. 1997;21:872–80.
5. Bartynski WS, Zeigler Z, Spearman MP, Lin L, et al. Etiology of cortical and white matter lesions in cyclosporin-A and FK-506 neurotoxicity. AJNR Am J Neuroradiol. 2001;22:1901–14.
6. Lee VH, Wijdicks EF, Manno EM, Rabinstein AA. Clinical spectrum of reversible posterior leukoencephalopathy syndrome. Arch Neurol. 2008;65:205–10.
7. Fugate JE, Claassen DO, Cloft HJ, Kallmes DF, et al. Posterior reversible encephalopathy syndrome: associated clinical and radiologic findings. Mayo Clin Proc. 2010;85:427–32.
8. Liman TG, Bohner G, Heuschmann PU, Scheel M, et al. Clinical and radiological differences in posterior reversible encephalopathy syndrome between patients with preeclampsia-eclampsia and other predisposing diseases. Eur J Neurol. 2012;19:935–43.
9. Kozak OS, Wijdicks EF, Manno EM, Miley JT, et al. Status epilepticus as initial manifestation of posterior reversible encephalopathy syndrome. Neurology. 2007;69:894–7.
10. Bartynski WS, Boardman JF. Distinct imaging patterns and lesion distribution in posterior reversible encephalopathy syndrome. AJNR Am J Neuroradiol. 2007;28:1320–7.
11. Legriel S, Schraub O, Azoulay E, Hantson P, et al. . Determinants of recovery from severe posterior reversible encephalopathy syndrome. PLoS ONE. 2012;7:e44534.
12. Alhilali LM, Reynolds AR, Fakhran S. A multi-disciplinary model of risk factors for fatal outcome in posterior reversible encephalopathy syndrome. J Neurol Sci. 2014;347:59–65.
13. L Amass 1, W K Bickel, S T Higgins, G J Badger. Alternate-day dosing during buprenorphine treatment of opioid dependence. Life Sci. 1994;54:1215-28.