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Case Report

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PRES syndrome in the practice of a neurologist: a clinical case

Abstract

Posterior reversible encephalopathy syndrome (PRES) is a neurotoxic condition that occurs secondarily in response to the inability of the posterior circulation system to respond to acute changes in blood pressure. Hyperperfusion leads to a violation of the blood-brain barrier, resulting in the development of vasogenic edema, more often in the parietal-occipital region.[1]

This article presents a clinical case of a young patient with the PRES syndrome, after taking large amounts of narcotic and alcoholic substances, and clinically manifested by convulsions and spastic hypertension in all extremities, which led to restriction of the patient's movement. The peculiarity is the persistence of clinical manifestations and the lack of visibleeffects from treatment.

Therefore, given the importance of changes in brain matter and the versatility of clinical manifestations, a clinical case of PRES syndrome in a patient is presented below to improve the awareness of practicing neurologists. The peculiarity of this case is the severity of clinical manifestations and an unfavorable outcome, which is relatively rare in this syndrome.

Keywords: neurotoxic, encephalopathy, vasogenic edema, hypertensive encephalopathym neuroimaging

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Introduction

Posterior reversible encephalopathy syndrome (PRES) is a neurological disorder characterized by a range of neurologicalsigns and symptoms and distinctive neuroimaging findings reflecting vasogenic edema.[2] PRES syndrome is also known by the terms acute hypertensive encephalopathy or posterior reversible leukoencephalopathy. The term is inaccurate because, in this syndrome, changes can spread beyond the posterior parts of the brain.[3–5] In addition, although in most cases, with adequate treatment, most of the changes completely regress, in some patients the condition may progress leading to permanent damage to the brain parenchyma with residual neurological deficiency.[6]

Clinical manifestations of posterior reversible encephalopathy syndrome include headache, seizures, encephalopathy, and visual impairment.[7] In most cases, the syndrome of posterior reversible encephalopathy is manifested by vasogenic edema in the parietal and occipital lobes (observed in ~ 98% of cases), which may be due to a violation in the posterior circulationsystem.[8]

There are several theories about the pathophysiology of the development of PRES syndrome in the literature. The first theory suggests a rapid increase in blood pressure up to a hypertensive crisis, which was observed in most patients at the beginning of the disease.[2] According to this hypothesis, an increase in blood pressure above the upper limit of autoregulation leads to brain hyperperfusion, which can cause increased permeability of vascular walls and vasogenic edema.[6] Increased cerebral perfusion pressure contributes to additional dysfunction of the blood-brain barrier.[3,8]

The second theory is that the syndrome is triggered by endothelial dysfunction caused by circulating endogenous or exogenous toxins.[3] Confirming this hypothesis, PRES syndrome is often observed in patients with preeclampsia, sepsis, or during treatment with immunosuppressants or cytotoxic drugs.[9] A common factor in these diverse conditions is the presence of endogenous (preeclampsia, sepsis) or exogenous (chemotherapy, immunosuppressants) toxins that cause endothelial dysfunction.[10] A variation of the 'toxic/immunogenic' theory is that the trigger is the excessive release of pro-inflammatory cytokines, leading to activation of the endothelium, release of vasoactive agents, increased vascular permeability, and edema formation. This mechanism is considered a key feature causing PRES syndrome in patients withautoimmune disorders or sepsis.[10]

Case presentation

Patient R, 43 years old, was admitted to the Department of Neurology of the Central Clinical Hospital on 05/17/2023 with the following complaints: stiffness in movements, painful muscle spasms, movement restrictions in all limbs due to muscle tension, more pronounced in the left arm, in the right leg. From the anamnesis of the disease, it is known that thepatient has been ill for 2 weeks, on 05/11/2023, he took narcotic drugs and alcohol in large quantities, and by the evening he became ill, for the first time there were single seizures with loss of consciousness lasting up to 2-3 minutes, accompanied by tongue biting, after the end of the attack, the patient noted the pronounced difficulty in movement, he stopped moving due to muscle tension. An ambulance was called, examined by a doctor, an increase in blood pressure (BP) to 210/100 mmHg was recorded. (previously, there was no increase in blood pressure), he was taken to the hospitalof a multidisciplinary hospital in Almaty and received inpatient treatment from 05/11/2023 to 05/17/2023 in the toxicology department with a diagnosis: Severe poisoning with narcotic substances, opiates. Secondary toxic (opioid, alcoholic) leukoencephalopathy. Spastic tetraparesis. Toxic hepatopathy. There is no improvement in the dynamics of thecondition, the patient does not move independently due to pronounced rigidity in the limbs. In addition to the anamnesis (according to the mother): the patient has been using a narcotic substance (the name is not known) in large doses intravenously since May 6 of this year. The patient also used heroin, and tramadol in large doses for a long time. He alsoconsumed alcoholic beverages during the week before the illness.

Upon admission, the patient's condition is severe, the skin is pale in color and moist, and there is increased sweating. Hemodynamics is stable. Blood pressure during treatment in the neurology department ranges between 120- 130/80mmHg, pulse rates average 74-88 beats per minute. On palpation, the abdomen is soft and painless. Liver along the edge of the costal arch.

In neurological status: The level of consciousness is clear. On the Glasgow coma scale - 15 points. Answers the questions correctly. The contact is accessible, the commands are executed. Speech is not impaired. The memory is not impaired. Meningeal signs are negative. No pathology was detected on the cranial nerves. Pupils D = S, eye slits D = S, photoreactions are alive. Movement of the eyeballs in full, no nystagmus, no diplopia. Muscle strength in the extremities is moderately reduced, equal to 4.0 points on both sides. Muscle tone is diffusely increased by spastic type on both sides.D=S. Tendon reflexes D=S, hyperreflexia on both sides. Pathological signs: Babinski sign (+) on both sides. Sensitivity is preserved. He does not perform coordination tests due to hypertension. He was not tested in the Romberg pose. Urination was through a catheter.

In laboratory studies: leukocytosis was noted in the general blood test (14.0 10^9/μL), and in the general urine analysis and biochemical analyses, the indicators were within the normal range.

From the instrumental examination: EEG dated 05/15/2023: no pathological activity was detected.(all the patterns of MR studies will be reflected in the report).

MRI of the brain with contrast on 05/13/2023: The MR pattern is more typical for posterior recurrent encephalopathy syndrome, probably toxic origin (Figure 1).

MRI of cervical level from 05/13/2023: disco-osteophyte complexes in segments C5-C6, C6-C7. spinal cord without features. MRI of thoracic level from 05/13/2023: There are no pathological changes in the thoracic spine (Figure 2).

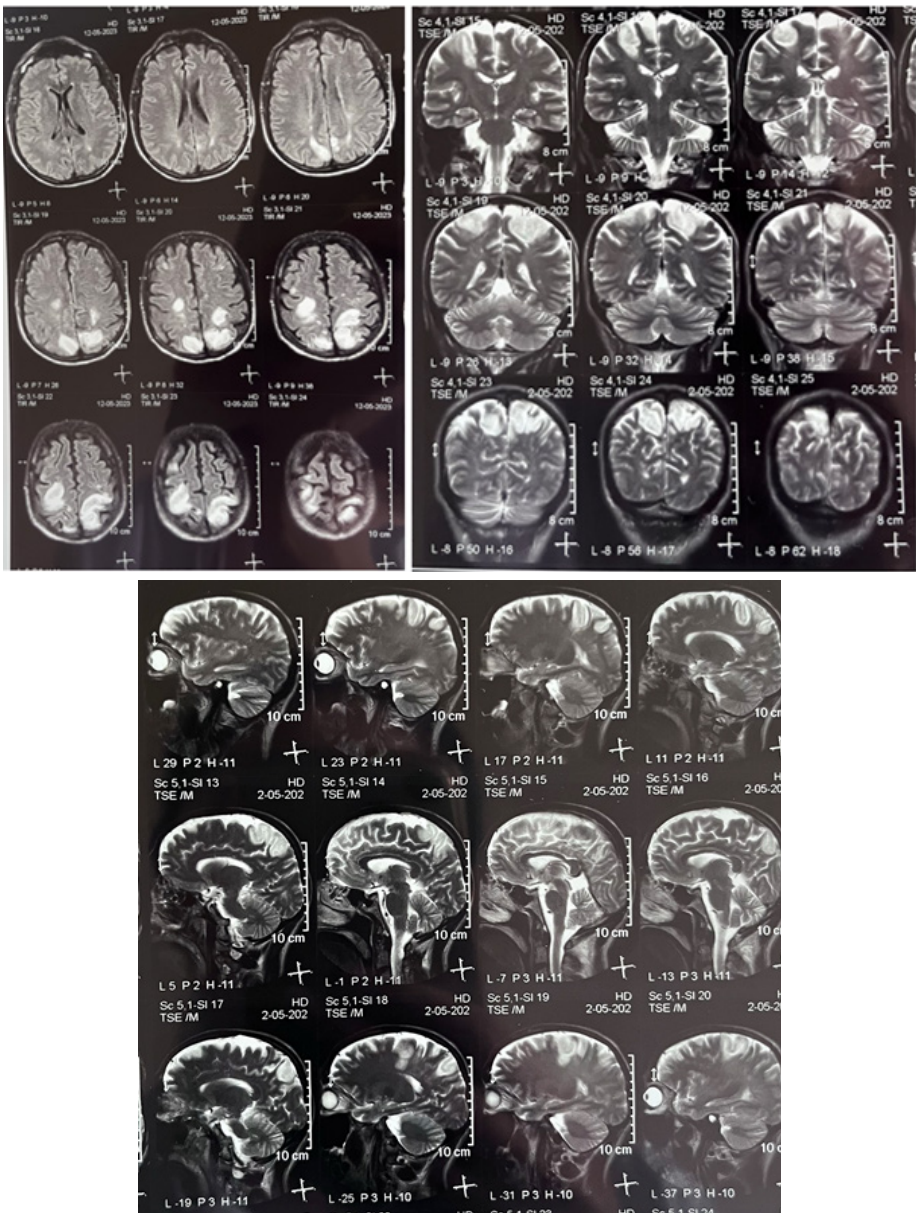

Figure 1 MRI of the brain.

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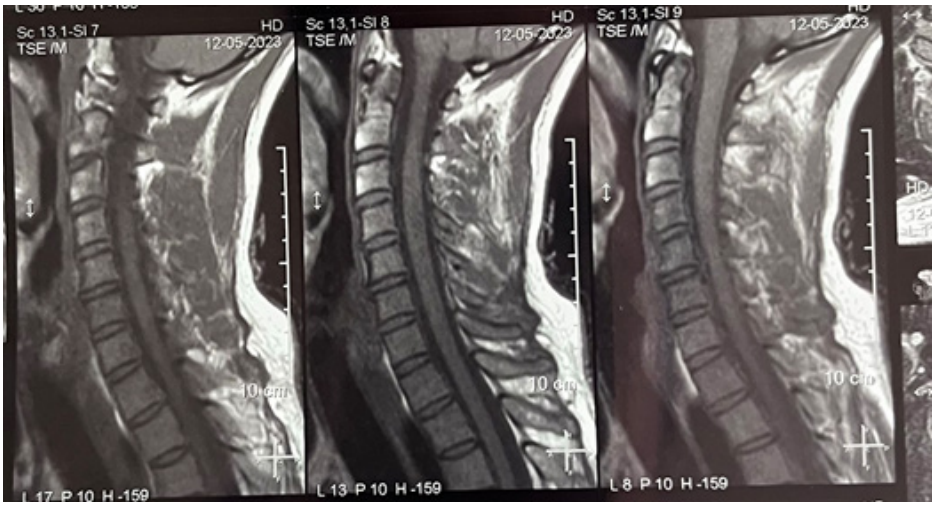

Figure 2 MRI of the spine.

Symptomatic treatment was carried out: decongestant therapy (L-lysine, magnesium sulfate) anticonvulsants (carbamazepine), B vitamins. Antihypertensive therapy was not carried out, since there was no increase in blood pressureabove 130/80 mmrt in the department. In dynamics, on the 10th day of hospitalization, the patient's condition improved with minimal treatment effect: painful muscle spasms decreased moderately, there was no increase in blood pressure (within 120-130/80 mmHg), and further, convulsive seizures were not observed in the patient in the department. Spastichypertension in the extremities persists, the patient does not move independently, and increased sweating also persists. The emotional background of the patient is calm, the criticism has been reduced, and there is a lack of interest in the further prognosis of the course of the disease. The patient notes a sleep disturbance, due to moderate pain in the extremities more at night. Periodically, the patient refuses to meet with relatives, and they refuse to consult a psychiatrist further.

The patient was discharged from the hospital on the 10th day of hospitalization, the increased muscle tone of the extremities persists, the patient does not move independently and cannot serve himself, movements in the extremities aresharply limited, while painful spasms decreased during the daytime, persist only at night.

Discussion

There is no accurate data on the prevalence of this disease in the literature. The syndrome occurs in a wide age range. Neurological disorders tend to regress within a few weeks.[11] However, there are descriptions of cases where the recoverywas delayed for up to 1 year or more. In our case, the clinical manifestations persisted for more than 10 days. Unfortunately, in our case, further monitoring of the patient was impossible, after discharge from the hospital, contact with the patient and the patient's family was lost, as a result, the patient's condition remains unclear at the moment. I would also like to add about the competence of the patient and the patient's relatives. There remains a big question aboutthe patient's compliance with all points of the recommendations, starting from changing the daily routine, diet and habitsand ending with taking medications. This issue is especially relevant in patients with cognitive impairment (from mild to severe) and reduced criticism. It is also necessary to take into account the level of education of relatives, who in most cases do not recognize evidence-based medicine and are more inclined to choose alternative medicine. Such circumstances make it difficult to further monitor patients and the course of the disease with scientific interest.

Conclusion

The prognosis of the PRESS syndrome is mainly determined by the underlying pathology that caused these changes in the central nervous system. However, neurological complications persist in some cases and may require long-term treatment. To date, no specific prognostic factors have been identified. The severity of lesions in MRI imaging may be an important parameter determining the long-term prognosis.

Many aspects of PRES syndrome concerning the pathophysiology and treatment remain unclear today. The data on cerebral perfusion in patients is heterogeneous since both cases of hyperperfusion and a decrease in central nervous system perfusion were observed. Although there is agreement on the elimination of the etiological factor caused by cytotoxic drugs, further treatment with immunosuppressants or chemotherapy remains a difficult issue, which is usually solved ona case-by-case basis. In future research, from my point of view, several of the following questions should be considered: Is it necessary to permanently cancel medications that cause symptoms of PRES syndrome? If not, what is the optimal duration of the treatment break? Are patients at risk of relapse of PRES syndrome? Is there a linear correlation between clinical symptoms and the dose of a cytotoxic substance?

Acknowledgments

None.

Conflicts of interest

The authors declare no conflicts of interest.

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