Case Report
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Central Pontine Myelinolysis as a Result of Ideally Corrected Hyponatremia in a Post-Covid Patient

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Abstract

Central pontine myelinosis (CPM) has a broad prognostic spectrum ranging from asymptomatic cases to mortality. CPM is a non-inflammatory neurological condition with various clinical features and onset patterns. In this case report, we aim to present the first case of central pontine myelinolysis because of an optimally corrected hyponatremia in a post-COVID-19 patient our knowledge while assessing the clinical and radiological findings. This case showed us that hyponatremia developed in a patient without comorbidity in the days following the COVID-19 infection and that ODS might occur later, despite the slow recovery of hyponatremia. For patients with symptomatic recovery from hospitalized COVİD-19 disease, care should be taken in terms of fluid and electrolyte disorders that may develop later.

Keywords: Hyponatremia, central pontine myelinolysis, COVID-19

Introduction

Hundreds of case reports about osmotic demyelination syndrome have been published since its first report in 1959. The term was coined because it not only indicates the pathologic character of the disease but also points to the specific anatomical location. Central pontine myelinolysis (CPM) is an essential subset of a group of disorders called osmotic demyelination syndrome (ODS), which involves damage in different parts of the brain, mainly in the pontine white matter pathways[1]. Hyponatremia causes a decrease in the serum tonicity which makes it easier for water to move from the extracellular field toward brain cells. This will cause water to move towards the extracellular area, and brain cells will undergo apoptosis. This phenomenon is known as osmotic demyelination syndrome (ODS).

Post covid syndrome is diagnosed if the symptoms and signs after COVİD-19 infection last longer than twelve weeks and other reasons to explain this situation are excluded[2]. Hyponatremia is one of the most common electrolyte disturbances in the acute phase of COVİD-19 infection. Hyponatremia can be seen both at the time of diagnosis and during treatment. In the literature, the presence of hyponatremia has been reported at a rate of 30% in COVİD-19 patients[3].

In this case report, we aim to present the first case of central pontine myelinolysis because of an optimally corrected hyponatremia in a post-COVID-19 patient our knowledge while assessing the clinical and radiological findings.

Case Report

A 54-year-old male patient presented with complaints of increasing speech and walking difficulties for one month. He did not have any chronic disease in his history. He had a history of hospitalization due to COVİD-19 infection 38 days before his admission. It was learned that complaints of muscle pain, weakness, difficulty in walking, seizures, speech disorder and, agitation continued approximately ten days after discharge. The serum sodium level at discharge was 136 mmol/l, decreasing to 122 mmol/l within ten days. During this period, the patient had applied to psychiatry and internal medicine outpatient clinics many times. Finally, the patient was hospitalized in the same clinic for hyponatremia and treated for four days. No pathology was detected in cranial computed tomography (CT) or magnetic resonance (MR) imaging. According to the neurologist, electroencephalography results were typical. Looking back, serum sodium decreased to 122 mEq/L in electrolyte follow-ups and increased to 136 mEq/L in the following four days.

When she applied to the emergency department, her blood pressure was: 161/86 mmHg, heart rate: 90 beats/min, respiratory rate: was 20/min, SpO2: 97, and body

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temperature was 36.6. His Glasgow coma scale (GCS) score was 15, he was in a normal state of consciousness, and he was oriented and cooperative but agitated. His neuromuscular examination revealed heavy dysarthria. He had ptosis in the right eye. He had bilateral intentional tremors during his cerebellar examination. He had truncal ataxia and hyperreflexia of deep tendons. His finger-to-nose test was mildly impaired in the right limb. No pathology was detected in other system examinations. In the laboratory values of the patient, leukocyte 12.9 10^3/ul, hemoglobin 13.6 g/dl, hematocrit 40.6%, platelet 548 10^3/ul, C-reactive protein: 1.21 mg/l, INR: 1.23, blood urea 59 mg/dL, glucose 110 mg/dL, sodium 139 mEq/L, potassium 4.08 mEq/L, aspartate aminotransferase 24 U/L, alanine aminotransferase 41 U/L, , troponin-I 2.3 ng/L were found. The patient's electrocardiogram was normal.

In the non-contrast brain CT scan done in our emergency department, there was a hypodensity in the pons. His cranial diffusion MR scan results were concordant with central pontine myelinolysis. The patient was administered intravenous steroids and then hospitalized in the neurology ward. In our case, pontine demyelination was detected after the examinations, as the complaints did not resolve approximately one month after the COVİD-19 infection.

Discussion

Hyponatremia is discussed as the most common electrolyte disorder in COVID-19 infection, as well as a poor prognostic factor. Inflammation, increased IL-6 secretion, and other inflammatory cytokines can be shown as the cause of hyponatremia developing during Covid-19 infection. Our case was treated by being hospitalized due to COVİD-19 infection. Days after she was discharged from the hospital, complaints of speech disorder, gait disturbance, and seizures emerged. Later, hyponatremia was detected. For patients with symptomatic recovery from hospitalized COVİD-19 infection, care should be taken in terms of fluid and electrolyte disorders that may develop later. A study mentions that COVID coinfection could also be a risk factor for the manifestation of extrapontine myelinolysis[4]. CPM was also reported to be not as rare as formerly known and to be responsible for most of the neurologic damage in patients with chronic hyponatremia[5]. The exact mechanism leading to osmotic demyelination syndrome is not fully understood, and there is no specific treatment. After the diagnosis, the treatment is supportive, and the main goal is to prevent complications. Ayus et al.[6] reported that a rapid correction of hyponatremia into mild hyponatremia is not dangerous and they recommend the serum sodium level be adjusted so it would not exceed 126 mM/l in the first 48 hours of treatment. They reported that a slow correction of hyponatremia (slower than 0.6 Mm/l) has higher mortality than a rapid correction of hyponatremia (faster than 2 mM/l). McKee et al.[7] showed that in the pathogenesis of CPM, rather than a fast or excessive correction of hyponatremia, a very high serum hyperosmolarity and chronicity of hyponatremia are also important factors. The American Expert Panel Recommendations endorse a serum sodium (Na) correction limit of 10–12 mEq/L in any 24-hour and 18 mEq/L in any 48-hour period for patients at average risk of ODS, and eight mEq/L in any 24-hour period for patients at high risk of ODS[8].

Conclusion

This case showed us that hyponatremia developed in a patient without comorbidity in the days following the COVID-19 infection, and that ODS may occur later, despite the slow recovery of hyponatremia. For patients with symptomatic recovery from hospitalized COVİD-19 infection, care should be taken in terms of fluid and electrolyte disorders that may develop later.

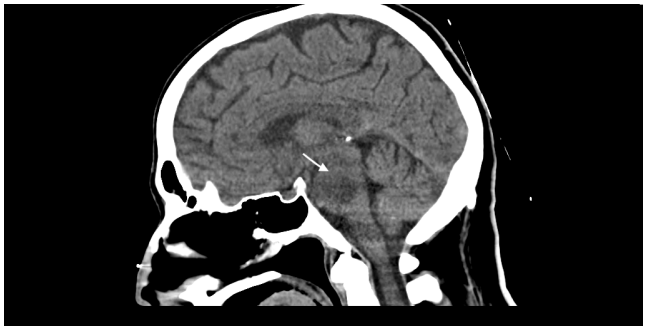

Figure 1: Hypodense, non-enhancing lesion in sagittal sequence, CT image of CPM (white arrow)

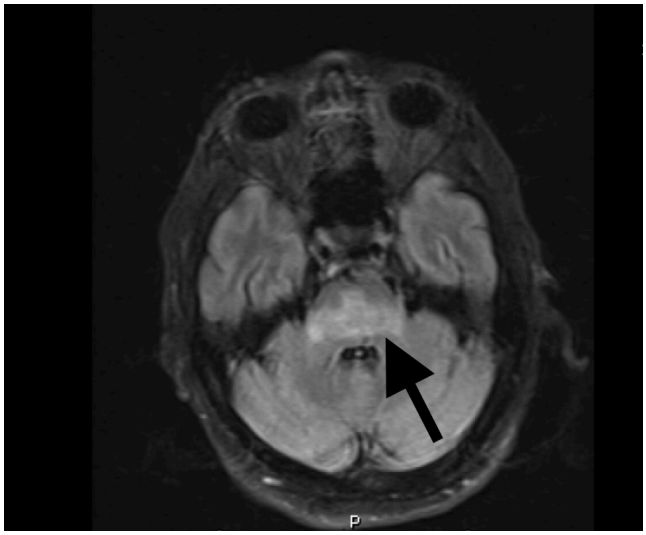

Figure 2: Hyperintense lesions in FLAIR MRI (white arrow)

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