

Posterior Reversible Encephalopathy Syndrome in Covid 19 Disease: A Rare Case

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Abstract

Covid-19 disease can have many neurological complications, posterior reversible encephalopathy being one of them. A 42-year-old female patient was admitted to the emergency department on the 4th day of PCR test positivity with decreased consciousness. Patient with a Glasgow coma score of 6 (E1M3V2). The patient's vital signs were stable and there was no abnormality in his laboratory. No pathology was found in the brain tomography to explain the loss of consciousness. In MRI evaluation, the patient was diagnosed with Posterior reversible encephalopathy syndrome (PRES) according to radiological and clinical information. The standard Covid -19 treatment added to the antiedema treatment was applied to the patient. Brain MRI was repeated four days later and previous MRI findings showed regression. The patient was discharged to the ward with stable neurological status on the 22nd day of hospitalization. PRES is a rare but serious complication in Covid-19. Especially in the Covid-19 intensive care unit, intubation and sedation suppress the clinical picture, and the difficulty in getting patients on magnetic resonance imaging can complicate the diagnosis. Therefore, PRES should be kept in mind in the presence of neurological symptoms such as encephalitis, meningitis, encephalopathy and cerebrovascular disease.

Keywords: Covid-19, posterior reversible encephalopathy syndrome, intensive care unit, neurology

Introduction

Approximately 250 million cases and 5 million deaths due to the Covid -19 disease have been reported due to the SARS-CoV-2 (Severe Acute Respiratory Syndrome Coronavirus 2) virus, which emerged in 2019 in Wuhan, China (1). Most of the Covid 19 patients are asymptomatic and the most common symptoms are fever, cough, shortness of breath, myalgia and headache. The most common serious manifestation of the disease is pneumonia. In severe cases, Severe Acute Respiratory Syndrome is the most important complication (2). In addition to these symptoms, neurological symptoms were also observed in more than 30% of the patients. These neurological symptoms are myalgia, dizziness, headache, hyposmia, hypogeusia, polyneuropathy, encephalitis and encephalopathy (4). Although the pathophysiology of the damage caused by the SARS-CoV-2 virus has not yet been resolved, theories exist. One of them is that the virus can enter the brain via the transcriptional route or by using ACE-2 receptors in endothelial and glial tissues, as in other central nervous system pathogens (3). Cerebral edema, which can be seen in Covid 19 patients, also supports this theory.

Posterior reversible encephalopathy syndrome (PRES), a condition that can develop due to eclampsia-preeclampsia,

hypertension, drug intoxications and many metabolic diseases, was first described by Hinchey et al. in 1996. The syndrome may progress with clinical findings in the form of seizures, headache, nausea, vomiting, mental status changes, visual loss, and focal motor loss (4). The pathophysiology of PRES is unknown. Two possible mechanisms are thought to be hypertension and endothelial dysfunction. As a result, the blood-brain barrier deteriorates and vasogenic cerebral edema develops (5). In the diagnosis of PRES, nonspecific changes can be seen in computerized brain tomography (CT), while specific findings are seen in brain magnetic resonance imaging (MRI). Therefore, MRI rather than CT is recommended for diagnosis because characteristic findings of cerebral edema with bilateral and symmetrical involvement in the occipital and parietal regions are easily demonstrated by MRI (5). The fact that this syndrome is reversible reveals the importance of rapid diagnosis and initiation of treatment. In the treatment of PRES, it is essential to control blood pressure and seizures, as well as to adjust the dose if cytotoxicity has developed due to drug use and to treat the known metabolic disease.

In this case report, we aimed to review the posterior reversible encephalopathy syndrome (PRES), which is a consequence of the endothelial dysfunction associated with COVID-19.

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Received: 12.11.2021 • **Accepted:** 14.12.2021

DOI: 10.33706/jemcr.1022869

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Available online at www.jemcr.com

Cite this article as: Taskin O, Yilmaz A, Soylu VG, Demir U, Kurada G. Posterior reversible encephalopathy syndrome in covid 19 disease: a rare case. Journal of Emergency Medicine Case Reports. 2022;13(2): 43-46

Case Report

A 42-year-old female patient was admitted to the emergency department on the 4th day of PCR test positivity with regression in consciousness. It was learned from her family that the patient did not have any additional disease, was not vaccinated against Covid-19 and did not use the favipiravir given for treatment. The patient, whose glasgow coma score was calculated as 6 (E1M3V2), was intubated and received mechanical ventilator support. Her heart rate was 103 / min rhythmic, arterial pressure was 123/67 mmHg, SpO₂ was 98 and fever was 37.8 ° C . Muscle strength was 2-3/5 on the upper left, 1-2/5 on the lower left, and Babinski was positive on the left, and nuchal rigidity could not be evaluated as optimal due to agitation. While the patient's thorax tomography had a ground-glass image compatible with bilateral covid, there was no pathology to explain the loss of consciousness in the brain tomography. The patient who underwent bedside echo did not have any cardiac etiology. Patient admitted to covid intensive care unit . Laboratory findings are; ph:7.39, pCO₂: 21.1 mmHg, pO₂:158 mmHg, lactate:1.9 mmol/L, Na:145 mEq/L, glucose: 247mg/dL, urea: 56mg/dL, creatinine: 0.87 mg/dL, alt :18U/L, ast 23 U/L, hs troponin: 5 ng/L, ldh: 498 U/L, ferritin: 480 ng/ml, hs troponin: 5 ng/L, sedimentation: 56, CRP: 75.4 mg/L, wbc: 30.82 10³μ/L, hgb:15.9 g/dL, hct:49.4 %, plt:480 10³ μ/L, lymph: 2.22 10³ μ/L, INR:1.32, and D-Dimer: 1.94mg/L.

Lumbar puncture was performed on the patient for the differential diagnosis of meningitis and encephalitis. Sample and simultaneous biochemistry were studied for cerebrospinal fluid culture and cell count. The patient was administered favipiravir, meropenem and other supportive

treatments. Brain magnetic resonance imaging was performed at the 20th hour of hospitalization of the patient who had no growth in the cerebrospinal fluid culture, no leukocytes in the cell count, and no abnormality in biochemical parameters. In MRI evaluation, it was observed that there was an increase in subcortical and cortical intensity in the bilateral posterior region and occipital area in the left hippocampus posterior and temporal lobe level (Figure 1). The patient was diagnosed with Posterior reversible encephalopathy syndrome (PRES) based on radiological and clinical information. The patient was administered 3 mg/h midazolam infusion and antiedema treatment. Brain MRI was repeated four days later, and previous MRI findings showed regression (Fig. 2). Midazolam was discontinued. The patient was awakened and cooperation was established. The patient was extubated on the 15th day of hospitalization and respiratory physiotherapy was started. The patient's covid treatment was continued and completed simultaneously. She was discharged to the service in a neurologically stable condition on the 22nd day of her admission to the care.

Discussion

PRES is characterized by headache, confusion, seizures, blurred vision (with normal pupillary reflex and fundus examination and intermittent hallucinations) and often accompanied by hypertension (6). No seizure was observed in our patient, and bilateral light reflex was found to be positive and the fundus examination was normal in her

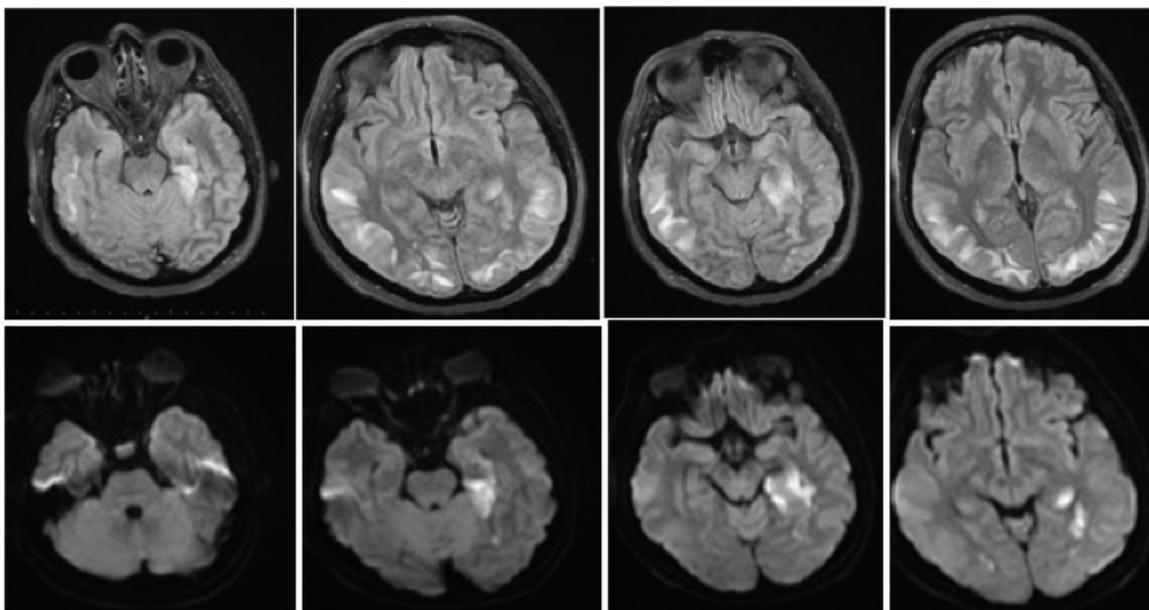


Figure 1. Patient's first Brain MRI Findings.

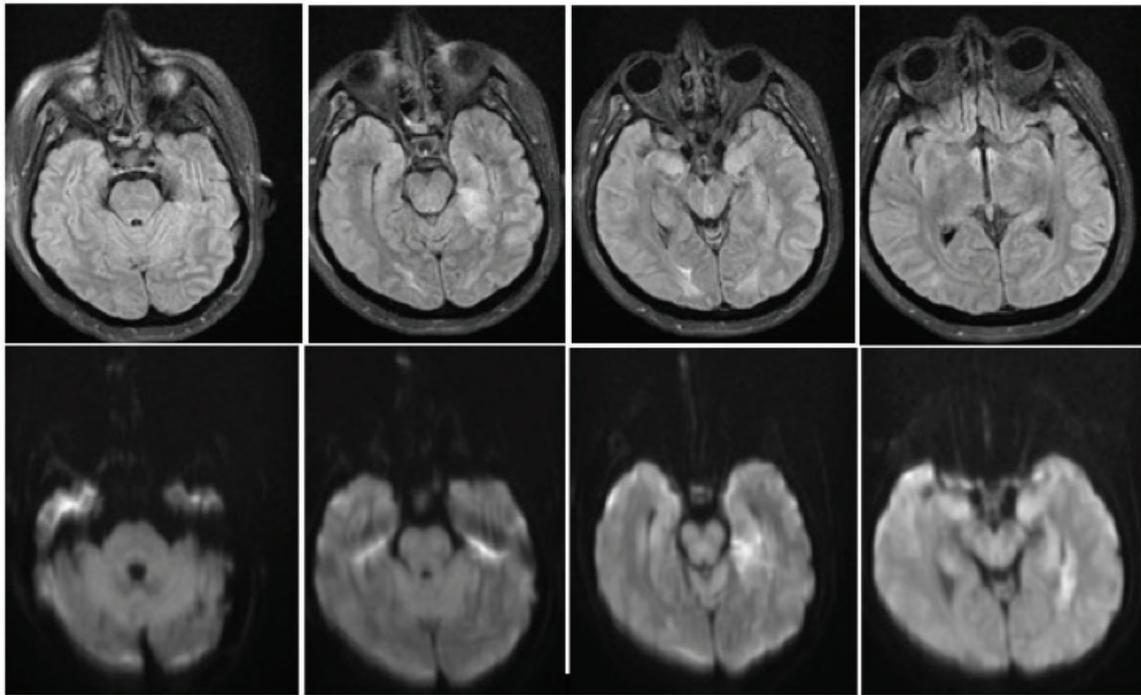


Figure 1. Patient's first Brain MRI Findings.

neurological examination. In severe covid 19 disease, PRES may develop due to comorbidities, inflammatory response and endothelial dysfunction. Although the pathophysiology of this syndrome has not been fully resolved, PRES is mostly characterized by impaired cerebral blood barrier autoregulation after severe hypertension or as a result of severe infection or vasotoxicity. Most of these patients have either an autoimmune condition or an immune disorder (7). While the most common cause was hypertension, our patient had normal blood pressure. Endothelial dysfunction in Covid 19 disease may be associated with PRES and other vascular complications. In recent studies, this hypothesis was supported by detecting microcirculation and vascular glycocalyx changes in covid 19 patients (8). The absence of hypertension and severe inflammation in our patient also supports this theory. Due to severe infection, the immune system is activated and cytokine production is stimulated. As a result, especially tumor necrosis factor α and vascular endothelial growth factor levels may increase. This increases the blood-brain barrier permeability, leading to the development of cerebral edema. This situation has also been described in viral infections such as sepsis, influenza A and parainfluenza (9). A similar complication may develop in PRES associated with Covid 19. Infection values were also high in our patient.

PRES cases have also been reported during Tocilizumab (IL-6 Antibody) treatment, which is used for cytokine storm in Covid 19 disease (10). Tocilizumab was not used in the treatment of our patient. Renal failure is also an important

risk factor for PRES, and our patient's kidney function tests were normal (8).

PRES can be seen in patients with covid 19 due to many potential triggers. Especially uncontrolled hypertension, excessive cytokine production, endothelial dysfunction, renal failure and immunomodulatory treatments may cause the development of PRES.

Conclusion

In conclusion, PRES is a rare but serious complication in covid 19. Especially in the covid 19 intensive care unit, intubation and sedation suppresses the clinical picture and the difficulty of taking the patients to magnetic resonance imaging can make the diagnosis difficult. Therefore, PRES should be kept in mind in the presence of neurological symptoms, such as encephalitis, meningitis, encephalopathy and cerebrovascular disease. It should be known that early diagnosis and treatment are of vital importance and that recovery without sequelae is possible.

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