CASE REPORT

Non-hypertension-associated Posterior Reversible Encephalopathy Syndrome in COVID-19

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Abstract

Background: Coronavirus disease-2019 (COVID-19) infection-related neurological events are not uncommon but presenting as posterior reversible encephalopathy syndrome (PRES) without hypertension is a very rare presentation and requires a high index of suspicion.

Case summary: We report a case of a middle-aged female who presented with severe COVID-19 disease with no neurological symptoms. She complained of diminished vision on day 7 of the illness and underwent an MRI brain to rule out an ischemic stroke but the findings were suggestive of PRES. She had no episode of hypertension during the hospital stay. Probably severe COVID-related inflammation was the reason for such a presentation. Conservative management resolved the issue and her symptoms weaned off.

Conclusion: Severe COVID disease can lead to PRES-like symptoms and requires neuroimaging to validate it. Conservative management is the best treatment for such patients.

Keywords: COVID-19 infection, Inflammation, Posterior reversible encephalopathy syndrome.

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Introduction

Posterior reversible encephalopathy syndrome previously known as (reversible posterior leukoencephalopathy syndrome, reversible posterior cerebral edema syndrome, and reversible occipital parietal encephalopathy) is a clinico-radiological syndrome characterized by varied clinical symptoms such as headache, seizures, altered mental status, and visual loss.

The exact pathophysiology is still unknown but hypertension has been always been a common finding in all patients. There are many other drugs and their unknown interaction which can precipitate symptoms leading to PRES. We are discussing a case of coronavirus disease-2019 (COVID-19), a disease patient, developing features of PRES with no event of hypertension.

Case Report

A 52-year-old female was admitted to our intensive care unit (ICU) with hypoxemia due to COVID-19. On admission, she was conscious, oriented, and following verbal commands with no neurological deficit. The heart rate was 102 beats per minute, noninvasive blood pressure 146/72 mm Hg, respiratory rate was >35/minute and she was maintaining saturation (SpO_2) of 88–91% on 15 L oxygen via a non-rebreather mask. She was shifted to high flow nasal oxygen (HFNO) therapy and was initially requiring a fraction of inspired oxygen (FiO_2) at 90% to achieve SpO_2 of 90%. High resolution computed tomography (HRCT) chest was performed which bilateral ground glassing appearance with consolidation with CT severity score of 32/40. She was started on a third-generation cephalosporin, Inj Remdesivir, Tablet Baricitinib, Inj Solu-Medrol, and other supportive therapy including low molecular weight heparin (LMWH). The initial laboratory values are shown in Table 1.

From day 3 onward, her oxygen requirement started reducing with other vital parameters within the normal range. On day 5, the patient complained of abdominal pain which was diagnosed as rectus sheath hematoma on the CT abdomen. Anticoagulation was stopped in view of this finding. Gradually by day 7, her oxygen requirement had reduced to 45% but the patient started complaining of blurred vision and severe headache. On examination, the patient had a perception of light but was not able to see clearly. In view of the thrombogenic potential of COVID and discontinuation of LMWH in our case, MRI brain with stroke protocol was performed. MRI brain showed multiple bilateral frontal, parietal and occipital T2 FLAIR hyperintensities which are more pronounced posteriorly in addition to bilateral cerebellar

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Table 1: Patient's laboratory values at admission

Laboratory parameter	*Results*
Hemoglobin (gm/dL)	11.4
White blood cells (per cubic mm)	7,200
Platelet count (per cubic mm)	221×10^3
Urea (mg/dL)	26
Creatinine (mg/dL)	0.78
Serum sodium (mEq/L)	141.1
Serum potassium (mEq/L)	3.9
Serum bilirubin (mg/dL)	0.9
International normalized ratio	1.11
D-dimer (ng/mL)	3246.6
C-reactive protein (mg/L)	113.2

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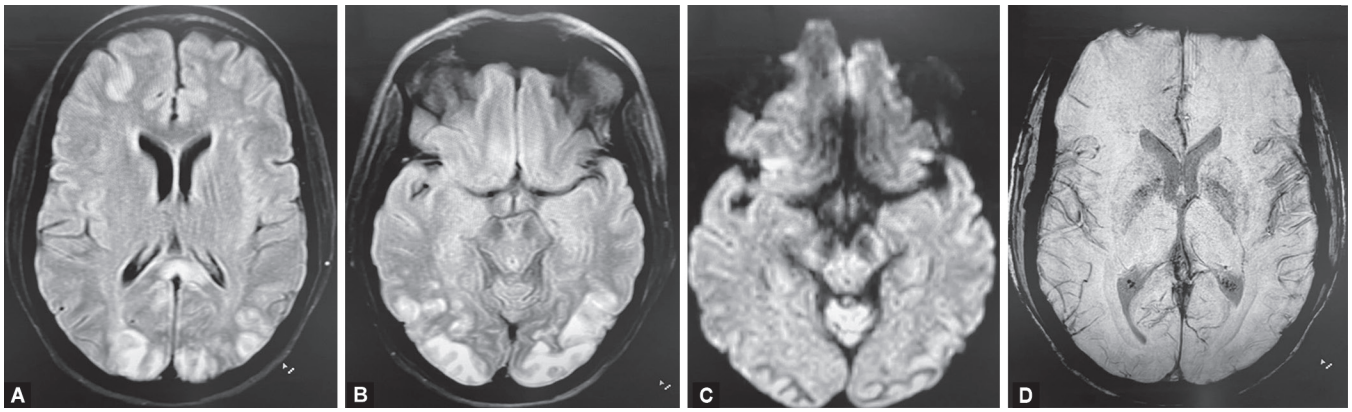

Figs 1A to D: MRI findings in PRES

hemispheres involvement compatible with PRES diagnosis (Fig. 1). MRI brain and neck angiography was done to rule out any possibility of acute posterior circulation stroke. Another differential diagnosis like cerebral hemorrhage was also ruled out. The patient was managed conservatively, and she started to have an improvement in vision over the next 4 to 5 days. The patient was eventually weaned off oxygen and was discharged in stable condition on day 15.

Discussion

COVID-19 patients have presented with varied neurological symptoms like new onset ischemic stroke, headache, or seizures.[1,2] PRES generally presents with rapid symptoms of headache, visual disturbances, and seizures.[3] It is mostly associated with episodes of hypertension before the symptoms present.[2] The exact pathophysiological mechanism of PRES is still unclear but the few proposed mechanisms are: (i) Severe hypertension overshoots the limits of autoregulation which results in vasogenic cerebral edema,[4] (ii) Cerebral vasoconstriction causing subsequent infarcts in the brain, (iii) Endothelial damage with blood–brain barrier disruption further leading to fluid and protein transudation in the brain.[5] PRES has also been associated with systemic inflammatory conditions like sepsis, transplantation, autoimmune states, and eclampsia.[6]

Patients with chronic kidney disease or acute kidney injury are at higher risk for PRES as the incidence of hypertension is higher in these patients. Presentation of PRES in absence of hypertension is rare with no proper pathophysiological mechanism known. All the previously proposed theories had hypertension as a baseline presentation.

The incidence of PRES in COVID has been very rare and the proposed mechanism has been a hyperimmune response to the COVID-19 viral infection resulting in endothelial wall injury and causing vasogenic edema.[7] As COVID-19 attaches to angiotensin-converting enzyme (ACE) 2 receptors in lung and brain parenchyma, it probably induces changes in the renin-angiotensin aldosterone system (RAAS) and causes blood pressure fluctuations and disrupts the blood-brain barrier.[5,8] This could act as a trigger for PRES in COVID patients.

Ours is probably the first case report of PRES without any incident of hypertension in COVID. Our patient had a hyperimmune response with features of cytokine storm and probably this was the triggering factor for PRES. As there is no specific treatment for PRES, the patient improved as COVID disease improved which all the more reinforces the fact PRES can present in severe COVID disease without hypertension.

Conclusion

Posterior reversible encephalopathy syndrome can present in varied inflammatory conditions and the presence of hypertension is common but not necessary. Supportive therapy and treatment of the cause are key to management.

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