

COVID-19 and Posterior Reversible Encephalopathy Syndrome

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The novel severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) caused an epidemic in December 2019 in Wuhan, China, which became a pandemic (as designated by the WHO), creating a current health emergency.¹ A preliminary report warned that SARS-CoV-2 had neuroinvasive potential because some infected patients had neurologic symptoms such as headache, nausea, and vomiting.² Several subsequent reports have described the emergence of various neurologic disorders in the evolution of SARS-CoV-2 infectious processes. In this article, we report the case of a patient with coronavirus disease 2019 (COVID-19) who presented with posterior reversible encephalopathy syndrome (PRES), diagnosed on clinical, laboratory, and imaging bases.

PRACTICAL IMPLICATIONS

Consider posterior reversible encephalopathy syndrome in the differential diagnosis of patients with COVID-19 with sudden neurologic symptoms, mainly seizures.

Case

A 67-year-old female patient underwent emergent left carotid endarterectomy due to sudden obstruction of the artery treated previously with stenting and angioplasty. The surgery was successful. In the postoperative period, however, the patient required 10 seconds assisted cardiopulmonary resuscitation, with reversal without any medical or neurologic sequelae. CT of the brain (figure, A) and CT angiography of the brain and neck showed no change and confirmed the patency of the operated artery. After this complication, the patient had a favorable evolution and was discharged 2 days later, asymptomatic.

Four days after discharge, family members found the patient at home with tonic-clonic seizure and loss of consciousness. On admission, she was disoriented and agitated, had difficulty following verbal commands, and mobilizing the extremities, and the pupils were equal, round, and reactive. Her blood pressure was 150/88 mm Hg (similar to baseline), respiratory rate was 24 breaths/min, and oxygen saturation level (SaO₂) was 83%. Pulmonary auscultation revealed diffuse bronchi.

Brain CT on admission showed areas of bilateral parieto-occipital hypodensity, suggestive of PRES, with no sign of hemorrhage (figure, B and C). Owing to the patient's low SaO₂ and in the context of the COVID-19 pandemic, chest CT was performed and showed ground-glass opacities in both lungs (figure, D). A nasal-swab RT-PCR test was positive for SARS-CoV-2. The laboratory findings demonstrated leukopenia. The basic metabolic panel, hepatic enzymes, and CSF analysis were without abnormalities.

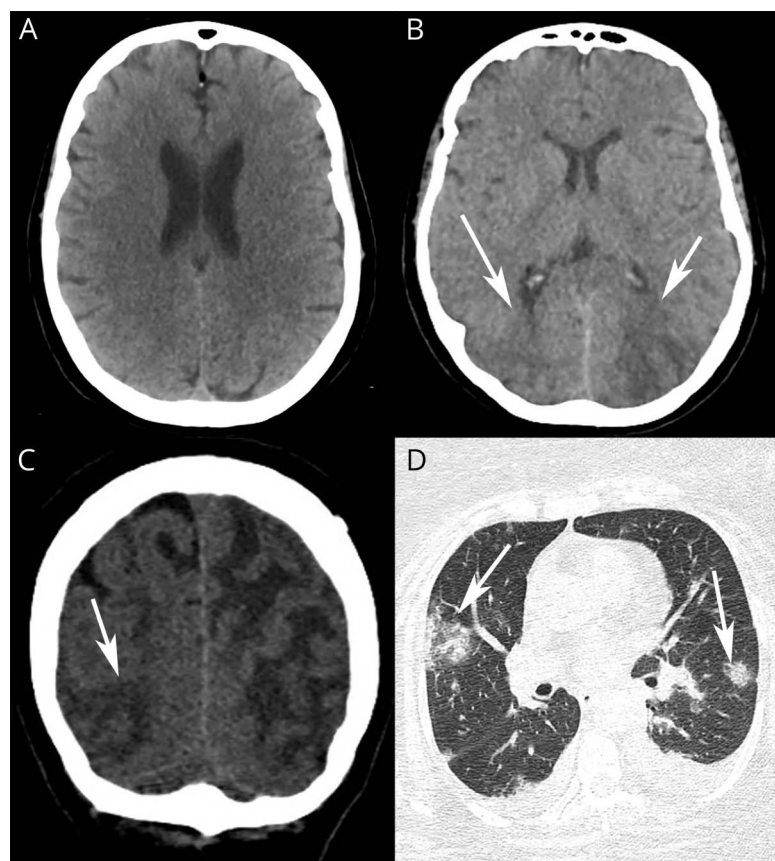
The patient evolved with full neurologic recovery, but her pulmonary and inflammatory conditions worsened, and she died 1 week later.

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(A) Brain CT performed 7 days before admission showed no abnormality. It was performed after a postsurgical cardiopulmonary resuscitation (promptly reversed and with full recovery). The purpose is to show previously normal parieto-occipital regions. (B, C) Brain CT performed on readmission showed bilateral asymmetrical parieto-occipital hypodense lesions (arrows). (D) Chest CT showed round peripheral ground-glass opacities in both lungs (arrows), consistent with findings typically reported in COVID-19 pneumonitis.

Discussion

Based on clinical, laboratory, and imaging findings, the diagnosis of PRES was proposed, mainly because of the full neurologic recovery and CSF normal, excluding meningoencephalitis and ischemic stroke, the 2 most common conditions.

PRES is characterized by acute onset of neurologic symptoms, vasogenic edema on neuroimaging (hypodense lesions on CT, especially in the parieto-occipital white matter, and hyperintense regions on T2 and fluid-attenuated inversion recovery MRI), and reversibility of clinical and/or radiologic findings.^{3,4} It is associated mainly with abrupt and severe hypertension, which occurs in eclampsia and acute renal failure. It also occurs in patients treated with immunosuppressive or cytotoxic agents (e.g., cyclosporine and tacrolimus),³ those with connective tissue diseases (e.g., systemic lupus erythematosus),⁵ and those with conditions such as thrombotic thrombocytopenic purpura.³ Some cases of PRES occur in the absence of hypertension because the pathogenesis of this syndrome is multifactorial.³⁻⁶ The primary mechanism is the loss of cerebral vascular endothelial cell regulation.

PRES has been associated with an inflammatory state and hypercoagulability, such as in sepsis of various origins.⁷ In the case reported here, the patient was infected with SARS-CoV-2 and presented with a condition compatible with PRES, in clinical and imaging terms, with no other evident justification for the syndrome in her clinical history. Health care providers should be aware that patients with COVID-19 can present with acute neurologic symptoms, and PRES should be considered as a possibility.

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Fabio Noro, MD, PhD	Federal University of Rio de Janeiro	Conception and design of the study, analysis, and interpretation of data, acquisition of data, drafting the article, and final approval of the version to be submitted
Fernando de Mendonça Cardoso, MD, PhD	Rede Dor-São Luiz, Rio de Janeiro	Analysis and interpretation of data
Edson Marchiori, MD, PhD	Federal University of Rio de Janeiro	Conception and design of the study and final approval of the version to be submitted

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