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IMAGES IN CLINICAL NEUROLOGY

Post-COVID-19 rhombencephalitis

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Herein, we reported a case of a 69-year-old female with a history of hypertension, migraine, and Goodpasture syndrome. The patient exhibited sudden-onset symptoms indicative of rhombencephalitis, accompanied by magnetic resonance imaging (MRI) findings, after a recent COVID-19 (coronavirus disease 2019) infection. The patient presented in February 2022 with severe occipital headache, accompanied by nausea and vomiting, distinct from the patient's usual migraine attacks. The following day, the patient experienced episodes of vertigo and visual obscurations after waking up. The patient had a history of acute kidney injury treated with corticosteroids and hemodialysis in April 2020. Following a COVID-19 infection three weeks earlier, the patient developed brain lesions suggestive of rhombencephalitis. Noncontrast brain MRI in February 2022 revealed hyperintensities in FLAIR (fluid-attenuated inversion recovery)-weighted sections in both internal capsules, midbrain, pons, and brachium pontis, as well as in both cerebellar hemispheres and the superior part of the medulla oblongata (Figures 1a-c), consistent with pathological findings indicative of rhombencephalitis. The patient's cerebrospinal fluid (CSF) analysis revealed 43/mm^3 lymphocytes, 12/mm^3 polymorphonuclear cells, an elevated total protein level of 120 mg/dL (normal: 15-45 mg/dL), and an elevated immunoglobulin (Ig) G index of 0.91 (normal: 0.23-0.64). The pattern of the oligoclonal bands was type 3. Despite extensive testing using multiplex polymerase chain reaction assays for a spectrum of viral and bacterial pathogens, including severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), all results returned negative. In contrast, specific antibodies against SARS-CoV-2 were identified in both the CSF and serum, with the CSF/serum SARS-CoV-2 antibody index reaching 4.1, suggesting active intrathecal synthesis of antibodies.

The patient was admitted to the hospital and discharged after a two-week period. Given the diagnosis of Goodpasture syndrome, consultations were sought from the Departments of Nephrology and Rheumatology, and comprehensive testing for relevant vasculitis markers was conducted. However, in this particular case, the preclinical serum biochemistry, the patient's medical history, and the profile of CSF biomarkers collectively argued against the diagnosis of reversible encephalopathy syndrome.

The patient received treatment with methylprednisolone for five days, resulting in clinical improvement. A CSF examination conducted one week after treatment with high-dose methylprednisolone revealed a reduction in lymphocytes to 7/mm^3 and polymorphonuclear cells to 2/mm^3. The total protein level was moderately elevated at 54.5 mg/dL, and the IgG index decreased to 0.7. The only notable findings were the persistent presence of SARS-CoV-2 antibodies in both the CSF and serum. No other specific abnormalities were detected through the investigations. The positive response to methylprednisolone treatment, coupled with the complete resolution observed in the MRI at the one-year follow-up, corroborated the diagnosis (Figures 1d-f).

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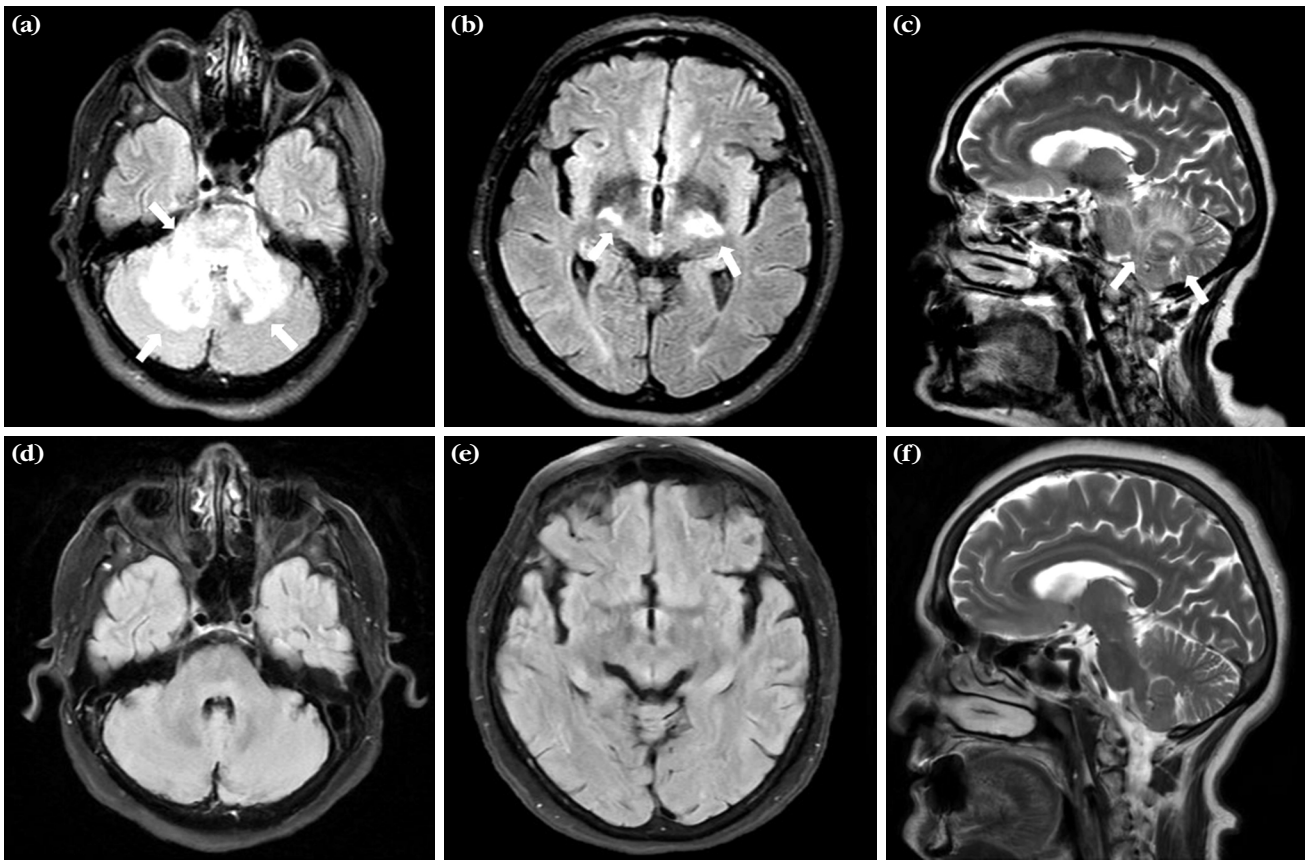

Figure 1. Brain MRI scans of the patient. **(a)** Axial FLAIR MRI at the level of the cerebellum and brainstem demonstrates bilateral hyperintensities within the cerebellar hemispheres and pons. **(b)** Axial FLAIR MRI at the level of the superior mesencephalon and thalamus reveals bilateral hyperintense signals within both internal capsules extending into the brainstem. **(c)** Sagittal T2-weighted MRI shows hyperintensity in the dorsal pons and midbrain extending into the thalamus and cerebellum. **(d-f)** Follow-up axial FLAIR and T2-weighted MRI one year after treatment shows complete radiological recovery.

MRI: Magnetic resonance imaging; FLAIR: Fluid-attenuated inversion recovery.

This case highlights the potential neurological complication of COVID-19, particularly the development of rhombencephalitis in susceptible individuals.[1,2] In our case, as in other autoimmune and infectious encephalitis cases, CSF findings indicated blood-brain barrier disruption. While the brain MRI was initially conducted without contrast, the alterations in the CSF infer the possibility of contrast enhancement. This assertion is corroborated by analogous cases documented in the literature, where contrast enhancement is a frequent finding. Similar to our case, early recognition and management are crucial for optimizing outcomes in such cases.

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