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Report of a patient with neurological symptoms as the sole manifestation of SARS-CoV-2 infection☆

Paciente con clínica neurológica como única manifestación de infección por SARS-CoV-2

Dear Editor:

The typical symptoms associated with SARS-CoV-2 infection have been described extensively in the literature[1,2]; however, the advance of the epidemic is revealing a broader spectrum of clinical manifestations. While neurological symptoms are common over the course of SARS-CoV-2 infection,[3] they have not yet been well defined. We present the case of a 30-year-old woman with no relevant medical or surgical history who presented neurological symptoms as the sole manifestation of SARS-CoV-2 infection.

The patient consulted due to a sensation of instability and loss of balance of 48 hours' progression. Symptoms were accompanied by nausea and vomiting and significantly worsened when she stood; she was unable to walk unassisted. Three weeks earlier, she had presented anosmia and ageusia, lasting 10 days. She reported no headache, hypoacusia, tinnitus, sensation of fullness in the ear, fever, cough, dyspnoea, or diarrhoea. She had not used any drugs or ototoxic medications, or suffered recent head trauma.

Haemodynamic and respiratory vital signs were normal. Higher cognitive functions were intact. Physical examination revealed persistent, purely horizontal, right-beating nystagmus, which was more pronounced with rightward gaze and accompanied by oscillopsia. The head impulse test could not be evaluated due to the appearance of saccades associated with the nystagmus. The alternate occlusion test revealed no deviation. The patient presented positive Romberg sign, falling backward, and was unable to walk in tandem. No auricular lesions were observed. No other relevant findings were observed in the neurological or physical examination.

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PCR results were positive for SARS-CoV-2, and blood analysis revealed mild lymphocytopaenia (1000 cells/mm^3), D-dimer level of 2270 ng/mL, fibrinogen level of 326 mg/dL, LDH level of 235 U/L, and C-reactive protein level of 1.2 mg/L.

In the light of the atypical nystagmus detected in the HiNTS (Head-Impulse, Nystagmus, Test-of-Skew) plus exam and the inability to evaluate the head impulse test, and given the hypercoagulable state described in cases of COVID-19,[4] we performed a contrast-enhanced brain MRI study to rule out central structural lesions as a cause of vestibular dysfunction. A chest CT angiography was also performed to rule out pulmonary embolism. Neither study revealed any abnormalities.

Having ruled out other possible aetiologies, we established a diagnosis of acute vestibular dysfunction probably due to SARS-CoV-2 infection.

Clinical and laboratory parameters progressed favourably with symptomatic treatment (antiemetic drugs and vestibular sedatives), hydroxychloroquine, and low-molecular-weight heparin.

Our patient's neurological symptoms are compatible with those described in the only case series published to date on neurological manifestations of COVID-19.[3] In that study, the central nervous system was most frequently affected, with dizziness being the most common symptom. The main peripheral nervous system symptoms were anosmia and ageusia, which have already been reported in past studies.[5,6] According to Mao et al.,[3] onset of neurological symptoms occurs early in the course of the disease. Anosmia and ageusia may have been these early symptoms in our patient.

However, our patient is younger than the mean age of patients with neurological manifestations in the study by Mao et al.[3] Also, respiratory tract and gastrointestinal symptoms of varying intensity seem to be a constant feature in patients with neurological symptoms and SARS-CoV-2 infection[3]; our patient presented none of the typical symptoms,[1,2] with neurological symptoms being the sole manifestation of the infection.

The pathogenic mechanisms leading to neurological involvement in patients with SARS-CoV-2 infection are not well defined, but may resemble those of such other coronaviruses as SARS-CoV and MERS-CoV, invading the central nervous system via the haematogenous or retrograde neuronal pathways, which would explain the peripheral nervous system involvement.[3,7] It has also been suggested that the virus may spread locally across the ethmoid cribriform plate.[8] The clinical course observed in our patient suggests that the virus may have accessed the nervous system directly by crossing the olfactory epithelium, as she first presented anosmia and subsequently vestibular symptoms; this is compatible with the hypothesis proposed by Matías-Guiu et al.[8] and Butowt et al.[7]

We should be aware that neurological symptoms may present as the sole manifestation of SARS-CoV-2 infection. It is essential to ensure early diagnosis of the infection in patients with acute neurological symptoms in the context of an epidemic in order to take actions to prevent the transmission of the disease; nonetheless, we should continue to follow the usual approach to diagnosis and treatment.

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