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LETTER TO THE EDITORS

Onset of Covid-19 with impaired consciousness and ataxia: a case report

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COVID-19 is an infectious disease caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) [1]. To date, the major concerns about the SARS-CoV2 infection are pulmonary complications [2]; however, neurological manifestations, as dizziness, headache, taste and smell impairment are frequent [3, 4]; cerebrovascular diseases, impaired consciousness, muscle injury, polyradiculoneuropathy and epilepsy have also been reported [3, 5]. Moreover, the presence of SARS-CoV-2 RNA had been identified in cerebrospinal fluid in a patient with encephalitis [6]. Neurological manifestations mostly occur early in the illness and may be the presenting features of COVID-19 [3–6]. Several mechanisms have been proposed to explain the neuro-invasive potential of the virus, especially its affinity for ACE2 and previous demonstration of neuro-invasivity in other coronaviruses; indirect mechanism might contribute to the neurological damage as well [7].

Here, we present a case of COVID-19 occurring together with neurological symptoms. The patient is a 73-year-old Caucasian male, in treatment for hypertension (Atenolol/Chlortalidone) and type 2 diabetes (Metformin), without previous neurological history, cognitively intact and professionally active. On the 23rd of February 2020, the patients gradually developed asthenia, gait ataxia, confusion and drowsiness; after 3 days lipothymia and urinary incontinence occurred. The following day, due to the worsening of drowsiness and confusion the patient was admitted to the Emergency Room (ER); at admission, body temperature was 38.6 °C, oxygen saturation was 98% in air; the neurological examination found confusion, balance impairment and gait ataxia; no meningeal signs were present. The EEG (awake, closed eyes) showed a reactive, unstable and symmetrical background alpha activity in posterior regions; the main features of the record were sporadic, low-voltage, focal polymorph delta elements in the anterior-frontal left cortex and sporadic spikes without clear epileptic correlate in the frontotemporal lobe, predominantly on the left; hyperventilation and intermittent photic stimulation did not modify the activity. Brain CT scan was negative for acute or chronic disease, chest X-ray and abdominal US were negative; in blood tests, C-reactive protein (CRP) was elevated (66 mg/L), white blood cells count (5.39 10^9/l), neutrophils count (2.87 10^9/l), creatine kinase, lactate dehydrogenase, procalcitonin were normal; urinary stick was mildly positive for protein (1 +). After the administration of paracetamol and the resolution of fever, the patient was discharged with a diagnosis of urinary infection, ceftriaxone was prescribed. In the following days, although the patients did not report hyperthermia, asthenia and drowsiness progressively worsened the patient being in soporous state for most of the day after 2 days and dyspnea appeared; for these reasons, the patient was readmitted to the ER (6 days after the previous discharge), lipothymia or orthostatic hypotension was not reported as the patient was bedridden. Temperature was normal (36.6 °C), blood gas analysis showed hypoxemia, chest X-ray showed bilateral pneumonia, blood tests showed increased CRP (141 mg/L) and fibrinogen (800 mg/dL); lymphocyte count, CK, LDH, procalcitonin were normal. The patient was tested positive for SARS-CoV-2 and admitted to the infectious disease department, for 3 weeks. He was treated with lopinavir/ritonavir, chloroquine, steroids and levofloxacin and recovered from both respiratory and neurological symptoms except for severe asthenia, which is still present more than 6 weeks from onset.

A limitation of this case is the absence of SARS-CoV-2 testing on the first admission. Moreover, as the respiratory symptoms developed a week after the onset of neurological symptoms, it is relevant to question whether the neurological manifestations were independent on SARS-CoV-2 and the patient contracted the infection in the ER or in the following days; however, we are not aware of any contacts

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occurred in the ER nor outside. As in other reported cases of neurological manifestations in COVID-19, it is not easy to determine whether the neurological symptoms have been directly caused by SARS-CoV-2 or whether they were epiphenomena of the systemic infection; moreover, in first instance, they could have been caused by a possible urinary infection. Nevertheless, contrarily to hyperthermia, neurological symptoms did not improve with antibiotic therapy, but as respiratory ones, they worsened and only resolved with antiviral therapy.

We report this case to highlight that COVID-19 might present with neurological symptoms in absence of other clinical manifestations and to stress the importance especially during the epidemic period of considering SARS-CoV-2 infection as a differential diagnosis in patient presenting with neurological symptoms, to avoid delayed diagnosis or misdiagnosis and to limit transmission [3].

Compliance with ethical standards

Conflicts of interest The authors declare that they have no conflict of interest.

Ethical standards This article does not contain any studies with human subjects or animals performed by any of the authors.

Informed consent Informed consent is not required as this article contains no picture or video of a recognizable patient.

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