

mechanism would actually increase the severity of COVID-19.

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## Isolated syncope as a form of presentation of COVID-19 infection<sup>☆</sup>

### Síncope aislado como forma de presentación de infección por COVID-19

Dear Editor:

SARS-CoV-2 infection has become a severe public health problem; presentation of the infection ranges from mild



or even asymptomatic forms to severe pneumonia or acute respiratory distress syndrome, which can be fatal.<sup>1–6</sup> Early identification of the infection is essential to ensuring proper isolation and clinical monitoring of these patients and their households. Most patients do present symptoms, with the most frequent being fever (72.3% of cases), respiratory symptoms, digestive symptoms, and neurological symptoms.<sup>6</sup> We describe an exceptional case of a patient without known cardiac disease who presented syncope as the only manifestation of SARS-CoV-2 infection.

The patient was a 78-year-old man, a former smoker, with history of arterial hypertension, dyslipidaemia, hyperuricaemia, chronic bronchitis, and pulmonary fibrosis due to asbestos exposure at work. He had no known heart conditions and denied having presented such symptoms as dyspnoea, angina, or palpitations. The patient was receiving long-term treatment with atorvastatin, enalapril, omepra-

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zole, zolpidem, levosulpiride, allopurinol, and inhaled glycopyrronium bromide. He visited the emergency department after a sudden, self-limited episode of loss of consciousness, with no prodromic or simultaneous symptoms. During the episode, he presented no anomalous body movements or automatism. He fully recovered within 2 minutes. The patient had not presented fever, altered thermal sensation, or respiratory or digestive symptoms. In the evaluation conducted at the emergency department, his arterial blood pressure was 108/68 mm Hg and baseline oxygen saturation was 97%. An electrocardiography study revealed no alterations. An emergency blood analysis returned no relevant findings, with the exception of a glomerular filtration rate of 37.5 mL/min and a D-dimer level of 5869 units. These results led us to request a CT angiography study of the pulmonary arteries, which revealed no signs of pulmonary embolism but did show alveolar infiltrate in the left upper lobe parenchyma, compatible with pneumonia, as well as pleural thickening and calcification; no changes were observed with respect to prior examinations, and the alterations were attributed to the pre-existing lung condition. An oropharyngeal swab returned positive PCR results for SARS-CoV-2, and the patient was admitted to hospital.

During hospitalisation, treatment was started with lopinavir/ritonavir, hydroxychloroquine, ceftriaxone, and corticosteroid boluses, as well as prophylactic doses of low-molecular weight heparin. Clinical progression was favourable: the patient remained afebrile and presented no new symptoms or further episodes of loss of consciousness. Follow-up chest radiographies showed a gradual resolution of the pulmonary infiltrate. Renal function markers also improved, and the patient was discharged a week after admission.

We present the first case of SARS-CoV-2 infection presenting with syncope as the only symptom. The literature includes very few cases of syncope as a form of presentation of COVID-19 and no cases of syncope in isolation: all the cases previously reported were in patients also presenting the characteristic symptoms of infection.<sup>1,3,7</sup>

Approximately 1% of emergency department visits are motivated by syncope.<sup>8</sup> It has numerous possible causes, and it is essential to detect potentially severe underlying diseases. The condition is classified into 3 main groups: neurally mediated, orthostatic, and cardiac.<sup>8,9</sup>

In patients with SARS-CoV-2 infection and syncope, one of the main causes to rule out is pulmonary embolism, which has been detected in up to 20% of the autopsy studies performed.<sup>5,10</sup> In cases of low or moderate suspicion of pulmonary embolism, D-dimer level should be quantified, given its high sensitivity (> 90%); however, it has poor specificity (40%-60%), and other diseases, including SARS-CoV-2 infection itself, may also cause elevated D-dimer levels.<sup>2,11</sup> In our patient, syncope did not coincide with a postural change, and he reported no previous orthostatic symptoms; however, long-term antihypertensive treatment may have favoured syncope due to a haemodynamic alteration. Other cases have been reported of syncope in the context of SARS-CoV-2 infection, although all the reported cases were in patients with pre-existing heart diseases; to the contrary, our patient did not have history of heart disease and no compatible signs were observed in the studies performed.<sup>1,3,7</sup> In patients receiving treatment for COVID-19, it is important

to consider the possibility of long QT syndrome, given the synergistic risk of drugs used to treat the disease.<sup>12</sup> We should also consider epilepsy in the differential diagnosis of all patients presenting loss of consciousness. Seizures have been described in patients with no history of epilepsy, mainly in patients with encephalopathy or encephalitis due to SARS-CoV-2 infection.<sup>13,14</sup>

Further studies are needed to clarify the aetiopathogenesis of syncope in patients with COVID-19; in patients presenting isolated syncope, we should take appropriate precautions due to the possibility of underlying SARS-CoV-2 infection.

## Conflicts of interest

The authors have no conflicts of interest to declare.

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## Impact of the COVID-19 pandemic on a cohort of ALS patients in Catalonia<sup>☆</sup>



### Impacto de la pandemia de COVID-19 en una cohorte de pacientes con ELA en Cataluña

Dear Editor:

SARS-CoV-2, the virus responsible for the current pandemic, is associated with high mortality rates, especially in elderly patients and those with chronic diseases.<sup>1</sup> Respiratory complications constitute the most common cause of death due to amyotrophic lateral sclerosis (ALS)<sup>2</sup>; management at multidisciplinary ALS units is known to increase the life expectancy of these patients.<sup>3</sup> Therefore, it seems reasonable to suggest that patients with ALS are at increased risk from COVID-19 and that they constitute a vulnerable population in view of the potential consequences of lockdown on their daily care. However, no epidemiological data are currently available to confirm this hypothesis.

### Methods

We created a survey to gather data on the impact of lockdown on the care provided to patients with ALS. The Miquel Valls Foundation (the main care platform for patients with ALS in Catalonia) used e-mail and mobile messaging to distribute the survey among patients with ALS and their caregivers. All respondents were  $\geq 18$  years old, had been diagnosed with ALS according to the revised El Escorial criteria,<sup>4</sup> and gave informed consent to participate. The study was approved by the local ethics committee.

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## Results

We collected data on 57 patients, although the survey was sent to a total of 169 patients with ALS. The questions referred to the period of lockdown following the declaration of a state of alarm in Spain (14 March to 21 June 2020). During this period, mobility was restricted and hospital care was largely focused on managing patients with COVID-19. According to our data, 19 patients with ALS died due to the natural course of the disease during lockdown. Previous studies estimate the number of deaths of these patients in Catalonia during a 3-month period at 29.<sup>5</sup> Although this is a rough estimate, it seems to suggest that mortality did not increase during lockdown.

Table 1 presents the clinical and epidemiological characteristics of our sample. Although only a small percentage of all patients receiving the survey actually completed it, they represent a cohort of patients with severe ALS and dependence: median ALSFRS-R score was 22 ( $Q_1$ – $Q_3$ , 15–31), most were on stages 3 (26.31%) and

**Table 1** Demographic and clinical characteristics of our sample of patients with amyotrophic lateral sclerosis.

	Patients with ALS (N = 57)
<b>Clinical data</b>	
Age in years, median ( $Q_1$ – $Q_3$ )	60 (52–68)
Sex	
Women	25 (43.85%)
Men	32 (56.14%)
Form of onset	
Spinal	42 (73.68%)
Bulbar	15 (26.32%)
ALSFRS-R, median ( $Q_1$ – $Q_3$ )	22 (15–31)
King's ALS clinical staging system	
1	1 (1.75%)
2	8 (14.03%)
3	15 (26.31%)
4a	5 (8.77%)
4b	28 (49.12%)

ALS: amyotrophic lateral sclerosis; ALSFRS-R: Revised Amyotrophic Lateral Sclerosis Functional Rating Scale.