Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

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Covid-19 and Guillain-Barré syndrome: More than a coincidence!

A 70-year-old woman, receiving 7.5 mg prednisone as a maintenance therapy for rheumatoid arthritis (RA), presented with a rapidly, bilateral weakness and tingling sensation in all four extremities resulting in a total functional disability within 48 hours. The patient denied any sphincter disturbances, dyspnea or swallowing difficulties. She first received a diagnosis of RA exacerbation but no improvement was seen after corticosteroids increase. At admission to our Neurology department, at the tenth day of symptom's onset (April 13), neurological examination showed quadriplegia, hypotonia, areflexia and bilateral positive Lasègue sign. Cranial nerves were intact. Temperature, lung and cardiac auscultation were, also normal. On April 1st, three days prior to the ongoing symptom's onset, the patient presented an episode of dry cough without dyspnea or fever, spontaneously resolving within 48 hours. Initial blood tests showed no abnormality, except for a lymphocytopenia (520/ml, normal: 1500–5000). A nerve conduction study (NCS), on day 10, revealed a marked reduction or absence of electrical potentials in both motor and sensory nerves in all four limbs, with little or no abnormalities in conduction velocities and latencies. The needle electromyography (EMG) found diffuse and abundant fibrillation potentials at rest. These findings were consistent with an Acute Motor and Sensory Axonal Neuropathy (AMSAN) subtype of Guillain-Barré syndrome (GBS). CSF analysis showed increased protein level at 1 g per liter (normal range: 0,2–0,4) with normal white blood cell count. Chest CT (day 10) revealed ground-glass opacities in the left lung (Fig. 1). SARS-CoV-2 on RT-PCR assay was positive at oropharyngeal swab (day 10), negative in CSF. The patient was treated with intravenous immunoglobulin (2 g/kg for 5 days) and a combination of Hydroxychloroquine (600 mg per day) and Azithromycine (500 mg at the first day, then 250 mg per day). No significant neurological improvement is seen after one week of treatment.

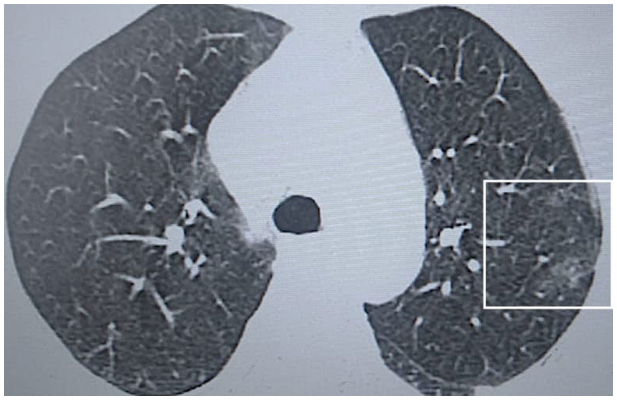

Fig. 1 – Chest computer tomography revealed a ground-glass opacities in the upper lobe of the left lung.

The Covid-19 infection hides many secrets that are yet to be revealed and little is known about its neurological manifestations. Here, we describe a case of a patient with mild respiratory symptoms linked to a COVID-19 infection, followed by a rapidly evolving quadriplegia arguing for a SARS-Cov-2-induced GBS. A negative PCR analysis in the CSF supports a post infectious, dysimmune mechanism.

Zhao et al. [1] reported the case of a 61-year-old man who presented with an Acute Inflammatory Demyelinating Polyneuropathy (AIDP) subtype of GBS, associated with SARS-Cov-2 infection. Being the first reported case, the authors questioned the cause-effect relationship between both events, since respiratory symptoms appeared after GBS's onset. After this first case, we found three other reports published to date. Camdessanche et al. [2] described a case of AIDP GBS subtype in a 64-year-old man, while the case of Sedaghat and Karim [3] resembled ours, an AMSAN form. Toscano et al. [4] reported a series of five patients from three Italian hospitals. Their findings were consistent with an axonal variant in three patients and with demyelinating process in two patients.

We add to the literature another case of GBS related to a Covid-19 infection. All these cases argues that SARS-Cov-2 virus could be a triggering factor of GBS. Since mild respiratory symptoms were noted in our patient, we suggest that all newly diagnosed Guillain-Barré cases should be tested for a Covid-19 infection in the current pandemic, even if they lack respiratory complaints. This would probably result in larger series and would help clarify the spectrum of this neurological condition.

Disclosure of interest

The authors declare that they have no competing interest.

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Acute meningoencephalitis in a patient with COVID-19

A 69-year-old man with a 7-day history of fever and cough, was admitted to the University Hospital of Guadeloupe (French indies) for confusion and severe headache. He had no medical history and reported having traveled to the Middle-East (cruise ship) with his wife 15 days before hospitalization. He did not report insect bites. A week after returning home, he presented fever, myalgia, cough, anosmia, ageusia, cervical pain, stiff neck, and diarrhea. At admission, he reported a worsening of his condition for 24 hours with painful and stiff neck and headache, confusion, walking disability with falls, and dyspnea. Noteworthy, his wife had also had isolated cough and ageusia for the past 10 days.

On examination, the patient was febrile (38.5 °C) with diffuse headache, neck stiffness, altered consciousness (Glasgow Coma Scale 14), confusion, swallowing disorders, and right-sided hemiparesis. Respiratory rate was increased (36/min), pulse rate was 95/min, blood pressure 160/89 mmHg, and oxygen saturation was 91% in ambient air.

Laboratory analyses showed increased C-reactive protein at 95 mg/L and creatine kinase level (655 U/L), raised transaminases (aspartate aminotransferase = 85 U/L, alanine aminotransferase = 94 U/L) and lactate dehydrogenase (442 U/L). Arterial partial pressure of oxygen was decreased at 64 mmHg with normal arterial partial pressure of carbon dioxide and pH. Chest computed tomographic scan was highly suggestive of COVID-19 (Fig. 1). Cerebrospinal fluid was purely lymphocytic (37×10^6/L) with no red blood cells, an increased protein level at 84 mg/dL and normal glucose level. Brain MRI with gadolinium was normal. Electroencephalogram showed a bilateral slowed activity without seizures. The detection of SARS-CoV-2 by specific real-time reverse PCR (RT-PCR) was negative in nasopharyngeal swab and cerebrospinal fluid (CSF) on days 2 and 4 after admission but was positive in bronchoalveolar lavage on day 5. Using a similar biological tool, the search for *Influenza* virus was negative. RT-PCR for varicella-zoster virus, herpes simplex virus (HSV), and enterovirus in CSF were all negative. Other tests for endemic infections in our area were performed, but all negative.

The patient received nasal oxygen therapy. Acyclovir infusions were performed for 3 days and stopped when RT-PCR for HSV was found negative. We started hydroxychloroquine sulfate 200 mg, three times per day, and azithromycin 250 mg daily for 7 days. On hospital day 4, his neurological condition improved with normal consciousness and abatement of swallowing disorders. At discharged on day 10, mild neuropsychiatric features were still present with an alteration of executive functions. Montreal Cognitive Assessment was decreased to 26/30.

We describe meningoencephalitis one week after the onset of COVID-19 based on the combination of altered mental consciousness, fever, focal neurological defects, and cerebrospinal fluid abnormalities. We suspect the meningoencephalitis was related to COVID-19, possibly through a direct infectious mechanism although the virus was not detected in CSF [1].