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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Letter to the editor

COVID-19 may induce Guillain–Barré syndrome

A 64-year-old man without medical history was admitted to our hospital after he fell and hurt his left shoulder leading to a tear of the rotator cuff. He had fever and cough for two days. In the context of COVID-19 pandemic, SARS-CoV-2 RT-PCR on nasopharyngeal swab was performed and positive. Clinical presentation was moderate with high grade fever for three days requiring oxygen 2–3 L/min through nasal cannula for five days. He received paracetamol, preventing thromboembolism by low molecular weight heparin and lopinavir/ritonavir 400/100 mg twice a day for ten days. Thoracic CT scan showed only 10–25% of ground glass opacities.

Eleven days after the symptom onset, while he did not need oxygen anymore having had no fever for five days, the patient complained of paresthesia in feet and hands. In three days, he installed a flaccid severe tetraparesia. MRC strength evaluation was 2/5 in the legs, 2/5 the arms, 3/5 in the forearms and 4/5 in the hands. Tendon reflexes were abolished in the four limbs. The 128 Hz tuning fork test was negative in the lower limbs and lightly felt in the upper limbs. Facial muscles were normal. The patient complained swallowing disturbance with a risk of suffocation as liquids took the wrong path. The patient was admitted in ICU and mechanically ventilated because of respiratory insufficiency. An intravenous immunoglobulin treatment (0,4 g/kg per day during 5 days) was initiated.

Electrodiagnostic tests five days after neurological symptom onset showed a demyelinating pattern in accordance with Guillain–Barré syndrome (GBS) criteria (Table 1) [1]. On needle examination, no rest activity was observed and during muscle contraction, only one single motor unit was recorded with a firing rate up to 25 Hz in the right tibialis anterior, the right vastus lateralis, the left first interosseus and the left deltoideus muscles.

On CSF analysis, protein level was 1.66 g per liter and cell count normal. Anti-gangliosides antibodies were absent in the serum. Biological tests were not in favor of a recent infection with Campylobacter jejuni, Mycoplasma pneumoniae, Salmonella enterica, CMV, EBV, HSV1 & 2, VZV, Influenza virus A & B, VIH, and hepatitis E.

COVID-19 pandemic is a worldwide disaster. Pulmonary disorder and respiratory insufficiency are the main problems linked to SARS-CoV-2 infection, which explains difficulties in ICU to treat numerous patients [2]. Recently, Zhao et al. questioned the link between COVID-19 and GBS [3]. Our case is the first GBS with a chronology undoubtedly in favor of a complication of COVID-19 infection. This must be known by clinicians as GBS may lead to ICU admission and needs to be differentiated from a possible ICU-acquired weakness after ICU treatments.

Table 1 – Motor nerve conduction study.

Nerve	Distal Latency (ms)	Velocity (m/s)	Amplitude (mV)	Conduction Block (%)	F mini Latency (ms)
Median R					
Wrist-APB	3.69 (N < 4)		5.9 (N > 4)		**38.7** (N < 30)
Elbow-wrist		**42.9** (N > 45)	4.8	−7.3	
Ulnar R					
Wrist-ADM	3.08 (N < 3.6)		5.9 (N > 4)		**37.5** (N < 32)
Below elbow-wrist		**43.4** (N > 45)	**3.9**	**−36.2**	
Below elbow-above elbow		**40.6**	**2.5**	−21.6	
Above elbox-axilla		54.2	**2.3**	−9.2	
Axilla-Erb		52.8	**0.14**	**−85.1**	
Ulnar L					
Wrist-ADM	3.54 (N < 3.6)		5.0 (N > 4)		**38.7** (N < 32)
Below elbow-wrist		**44** (N > 45)	4.3	−19.3	
Below elbow-above elbow		53	4	−10.9	
Above elbow-axilla		61.9	**3.8**	−4.9	
Axilla-Erb		45.8	**0.71**	**−79.5**	
Fibular R					
Ankle-EDB	**7.48** (N < 5)		**1.15** (N > 2)		**No F** (N < 52)
Below fibula-ankle		**26.7** (N > 40)	**0.8**	−29.3	
Above fibula-below fibula		**37.5**	**0.76**	−12.2	
Fibular L					
Ankle-EDB	**5.16** (N < 5)		**1.21** (N > 2)		**No F** (N < 52)
Below fibula-ankle		**27.3** (N > 40)	**0.69**	−14	
Above fibula-below fibula		**32.4**	**0.5**	−18.6	
Tibial R					
Malleolus-FHB	**8.91** (N < 6)		**1.2** (N > 4)		**No F** (N < 55)
Knee-malleolus		**27.7** (N > 40)	**0.79**	−21.4	
Tibial L					
Malleolus-FHB	**8.43** (N < 6)		**1.46** (N > 4)		**No F** (N < 55)
Knee-malleolus		**30.5** (N > 40)	**0.69**	**−61.1**	

ADM: abductor digiti minimi; APB: abductor pollicis brevis; EDB: extensor digitorum brevis; FHB: flexor hallucis brevis; L: left; N: normal; R: right; Bold: abnormal result according to our laboratory normal values in parenthesis.

Disclosure of interest

The authors declare that they have no competing interest.

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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.

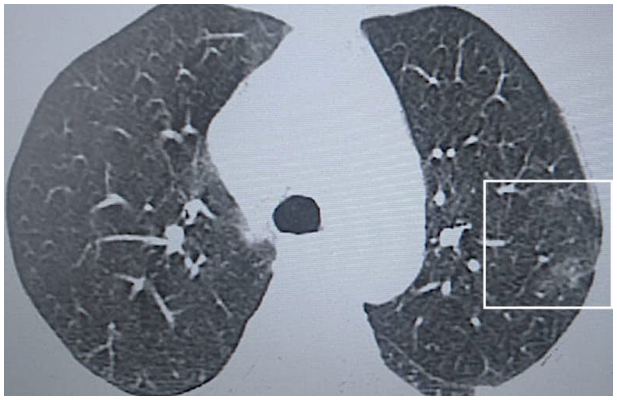

Fig. 1 – Chest computer tomography revealed a ground-glass opacities in the upper lobe of the left lung.

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.