Guillain-Barré syndrome associated with SARS-CoV-2 infection. Comments after 16 published cases☆

Síndrome de Guillain-Barré tras infección por SARS-CoV-2. Comentarios tras la publicación de 16 nuevos casos

Dear Editor:

It was with great interest that we read the article by Velayos-Galán et al.[1] We would like to report a similar case of Guillain-Barré syndrome (GBS) that manifested after bilateral pneumonia due to SARS-CoV-2 infection, and analyse the data published on this entity.

On 4 May 2020, a 70-year-old man presented subacute weakness in all 4 limbs, which worsened over the following 5 days. Three weeks earlier, he had presented bilateral pneumonia due to SARS-CoV-2 infection: a chest CT scan showed ground-glass opacities in both lungs, and PCR results were positive for SARS-CoV-2 in nasopharyngeal and oropharyngeal swabs. The patient was treated with oxygen therapy, hydroxychloroquine, azithromycin, ceftriaxone, and dexamethasone. The patient had no relevant medical history. His body temperature was 36.5°C and baseline oxygen saturation was 99%. Pulmonary auscultation revealed no alterations. A neurological examination revealed asymmetrical weakness (Medical Research Council grade 4/5 in the right hand; 4+/5 in the left hand; 4/5 in the left leg, and 3+/5 in the right leg) and areflexia in the legs and feet. Two days after admission, symptoms worsened. Muscle strength was 4/5 in the arms and hands and 3/5 in the legs and feet. Light touch and pin prick distal sensitivity was decreased in distal regions.

Laboratory analysis at admission revealed lymphocytopaenia (0.52×10^3 cells/L; normal range: $1.1\text{-}3.2 \times 10^3$ cells/L) and thrombocytopaenia (113×10^3 cells/L; normal range: $125\text{-}300 \times 10^3$ cells/L). CSF analysis showed normal cell count (0×10^3 cells/L; normal range: $0\text{-}8 \times 10^3$ cells/L) and a slightly increased protein level (49 mg/dL; normal range: 8-43 mg/dL). Nerve conduction studies performed on day 6 revealed delayed distal latencies and absence of F waves in the early phase, in the context of mixed-type (axonal and demyelinating) acute motor polyneuropathy of moderate and symmetrical intensity in all 4 limbs, with associated axonal sensory involvement, loss of motor units, and signs of neurogenic involvement of the muscles analysed without acute denervation (Table 1). The patient was diagnosed with GBS and started treatment on high-dose intravenous immunoglobulins (0.4 g/kg/day for 5 days) 8 hours after admission; symptoms improved on day 3 of treatment.

Upon discharge, 14 days after admission, he only presented mild weakness in the interosseous muscles of the hands (4+/5) and dorsiflexor muscles of both feet (4+/5), as well as generalised areflexia. The PCR results for SARS-CoV-2 at discharge were negative.

To our knowledge, 3 cases of concomitant GBS and SARS-CoV-2 infection with parainfectious profiles[1–3] and 13 cases of GBS subsequent to SARS-CoV-2 infection[4–12] have been reported to date (Table 2). The favourable progression of most of the patients who presented GBS after the infection is noteworthy.

In our patient, the gradual progression of neurological symptoms resembles that observed with postinfectious aetiologies. Therefore, we suspect an association between acute polyneuropathy and SARS-CoV-2 infection. This association is

Table 1 Motor and sensory nerve conduction findings.

Motor nerve conduction study	Distal latency (ms)		Amplitude (mV)		Conduction velocity (m/s)		F-wave latency (ms)	
		(at +10 days)		(at +10 days)		(at +10 days)		(at +10 days)
Left median nerve	5.2	7.8	4	2.7	37.1	38.9		
Right median nerve	5.6	7.8	4.4	2.9	45.2	40.4		
Right ulnar nerve	4.7	7.7	3.2	0.86	44.8	43.6		
Left peroneal nerve	4.8	5.8	2.5	2.3	34.7	34.5		
Right peroneal nerve	4.5	6.7	3.2	1.6	31.1	35.8		
Left tibial nerve	5.7	5.4	1.6	1.4	35.7	37.9	67.9	69.8
Right tibial nerve	6.2	6.9	1.1	1.3	34.2	37.1	69.8	68.1

Sensory nerve conduction study	Amplitude (μV)		Conduction velocity (m/s)	
		(at +10 days)		(at +10 days)
Left median nerve		2.4		46.9
Right median nerve	4.4	6.2	47.3	49.7
Right ulnar nerve	8.3	Absent	50	Absent
Left sural nerve	4.2	2.9	46.5	45.1
Right sural nerve	4.6	3.9	48.5	48.5

☆ Please cite this article as: Guijarro-Castro C, Rosón-González M, Abreu A, García-Arratibel A, Ochoa-Mulas M. Síndrome de Guillain-Barré tras infección por SARS-CoV-2. Comentarios tras la publicación de 16 nuevos casos. Neurología. 2020;35:412—415.

Table 2 Patient profiles.

Patient	Symptom onset	Symptoms	CSF	EMG	Treatment	Progress	Authors
43-year-old man	Respiratory infection and diarrhoea 10 days before	Progressive tetraparesis, distal sensory alteration. Bilateral facial palsy, dysphagia.	Not reported	Demyelinating polyradiculoneuritis	Ig for 5 days. Dolquine®, lopinavir, ritonavir. Corticosteroids	Favourable	Velayos-Galán et al.[1]
70-year-old woman	14 days before onset of respiratory symptoms (fever and cough)	Hand and foot paraesthesia. Gait alterations. Respiratory failure	Albuminocytologic dissociation. PCR for SARS-CoV-2 not available	Demyelinating, sensorimotor polyradiculoneuritis	Ig 0.4 g/kg/day for 5 days	Respiratory failure, no subsequent progression is specified.	Alberti et al.[2]
61-year-old woman	8 days before respiratory symptoms (dry cough and fever)	Weakness and fatigue	Albuminocytologic dissociation. PCR not conducted	Demyelinating neuropathy	Arbidol, lopinavir, ritonavir	Favourable; full recovery	Zhao et al.[3]
54-year-old woman	15 days before onset of anosmia and ageusia	Proximal symmetric paraparesis, sensory alterations in the limbs. Dysphagia	Albuminocytologic dissociation. PCR test not available	Demyelinating polyneuropathy	Ig 0.4 g/kg/day for 5 days	Initial exacerbation. Almost complete recovery	Scheidl et al.[5]
70-year-old woman	3 days after respiratory symptoms (dry cough)	Quadriplegia and sensory alterations	Albuminocytologic dissociation. Negative PCR results	Sensorimotor axonal neuropathy	Ig 2 g/kg for 5 days. Dolquine®. Azithromycin	No improvement after treatment	Otmani et al.[6]
70-year-old man	10 days after acute respiratory syndrome	Paraparesis, distal allodynia, difficulties in bladder voiding, and constipation	Albuminocytologic dissociation. Negative PCR results	Demyelinating, sensorimotor polyneuropathy	Ig 0.4 g/kg/day for 5 days	Rapid recovery	Coen et al.[7]
71-year-old man	7 days after onset of fever	Paraesthesia at limb extremities followed by flaccid tetraparesis. Moderate dyspnoea and low back pain	High protein levels and mild pleocytosis. Negative PCR results	Acute sensory and motor polyradiculoneuritis with predominant demyelinating features	Ig 0.4 g/kg/day for 5 days, ritonavir + lopinavir, hydroxychloroquine	Respiratory failure, death	Alberti et al.[2]

Table 2 (*Continued*)

Patient	Symptom onset	Symptoms	CSF	EMG	Treatment	Progress	Authors
76-year-old woman	8 days after onset of respiratory symptoms (fever and cough)	Low back pain radiating to both thighs, predominantly proximal progressive tetraparesis, distal-onset paraesthesia	Not performed	Not performed	None administered due to fast progression	Dysphagia, dyspnoea requiring mechanical ventilation, followed by death	Marta-Enguita et al.[8]
54-year-old man	10 days after dry cough and fever. 2 days after diarrhoea.	Hypoaesthesia and weakness in the lower limbs	Not conducted due to typical symptoms	Not conducted due to typical symptoms	Ig 0.4 g/kg/day for 5 days. Hydroxychloroquine	Progressive improvement	Virani et al.[9]
64-year-old man	11 days after onset of fever and cough	Distal paraesthesia in feet and hands followed by flaccid tetraparesis	Albuminocytologic dissociation	Demyelinating polyneuropathy	Ig 0.4 g/kg/day	Respiratory failure requiring mechanical ventilation	Camdessanche et al.[10]
65-year-old man	15 days after respiratory symptoms (cough, fever, and dyspnoea)	Acute ascending quadriparesis progressing to bilateral facial paresis and quadriplegia	Patient did not consent to the procedure	Axonal sensorimotor polyneuropathy	Hydroxychloroquine, lopinavir + ritonavir, azithromycin. Ig 0.4 g/kg/day for 5 days	Unknown	Sedaghat et al.[11]
Unknown	7 days after onset of fever, cough, and ageusia	Flaccid tetraparesis, facial diplegia. Paraesthesia in the upper limbs. Respiratory failure	Albuminocytologic dissociation. Negative PCR results	Unknown	2 cycles of Ig 0.4 g/kg/day for 5 days	Persistent weakness of the lower limbs, paraplegia, and dysphagia	Toscano et al.[12]
Unknown	10 days after onset of fever and pharyngitis	Facial diplegia. Paraesthesia in the lower limbs and ataxia	Albuminocytologic dissociation. Negative PCR results	Unknown	Ig 0.4 g/kg/day for 5 days	Improved ataxia and facial diplegia	Toscano et al.[12]
Unknown	10 days after onset of fever and cough	Flaccid tetraparesis and facial weakness. Respiratory failure	Albuminocytologic dissociation. Negative PCR results	Unknown	2 cycles of Ig 0.4 g/kg/day for 5 days	Progression to flaccid quadriplegia and respiratory failure	Toscano et al.[12]
Unknown	5 days after onset of cough and hyposmia	Flaccid tetraparesis and ataxia	Normal. Negative PCR results	Unknown	Ig 0.4 g/kg/day for 5 days	Mildly improved tetraparesis, inability to stand at 1 month	Toscano et al.[12]
Unknown	7 days after onset of cough, ageusia, and anosmia	Flaccid quadriplegia. Facial weakness. Respiratory failure	Slightly elevated protein level. Negative PCR results	Unknown	Ig 0.4 g/kg/day for 5 days and plasmapheresis	Unknown	Toscano et al.[12]

CSF: cerebrospinal fluid; EMG: electromyography; Ig: immunoglobulins; PCR: polymerase chain reaction.

supported by the fact that the patient observed home isolation for 21 days before neurological symptom onset, as well as the negative results for antiganglioside antibodies. However, the postinfectious onset, acute clinical course, and typical neurophysiological findings of GBS (mixed-type polyneuropathy of motor and sensory fibres), together with the absence of history of autoimmune, neoplastic, or neurological disease, suggest postinfectious aetiology. A significant limitation is the lack of availability of SARS-CoV-2 serology tests and CSF PCR tests at our centre.

While our case is suggestive of a possible association between GBS and SARS-CoV-2 infection, further case reports with epidemiological data are needed to demonstrate a causal relationship. This case also underscores the need to consider possible neurological symptoms of SARS-CoV-2 infection.

The authors agree that there is a need for careful observation of neurological complications of SARS-CoV-2 infection.

The Spanish Society of Neurology is currently conducting a national observational study on neurological presentations and manifestations of COVID-19.

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https://doi.org/10.1016/j.nrleng.2020.06.002
2173-5808/

Emergency implementation of a teleneurology service at the neuromuscular diseases unit of Hospital Regional de Málaga during the SARS-CoV-2 pandemic☆

Implantación emergente de un servicio de Teleneurología en la Unidad de Neuromuscular del Hospital Regional de Málaga durante la pandemia por SARS-CoV-2

Dear Editor:

Teleneurology was initially developed to provide care for complex, acute disorders in patients living in remote places, as was the case with telestroke.[1] However, in recent years its use has been expanded to treat other neurological diseases, and teleneurology has been gradually incorporated into routine outpatient follow-up.[2] Benefits include a reduction in time and travel costs for patients, improved access from remote areas, and perceived satisfaction among professionals and patients and their family members. Its limitations include the loss of traditional in-person relationships, the inability to perform a complete neurological examination, and neurologists' concerns about a possible loss of diagnostic accuracy.[3] We may distinguish 3 means of communication between neurologists and patients in teleneurology: telephone, audiovisual systems, and written consultation.[4] We propose the abbreviations t-consultation for telephone consultations, v-consultation for video consultations, and e-consultation[5] for written consultations.

Since the publication in Spain of Royal Decree 463/2020,[6] declaring the state of alarm that transferred management of the healthcare crisis caused by the SARS-CoV-2 coronavirus

☆ Please cite this article as: Romero-Imbroda J, Reyes-Garrido V, Ciano-Petersen NL, Serrano-Castro PJ. Implantación emergente de un servicio de Teleneurología en la Unidad de Neuromuscular del Hospital Regional de Málaga durante la pandemia por SARS-CoV-2. Neurología. 2020;35:415—417.