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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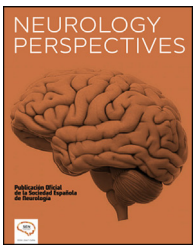

ORIGINAL ARTICLE

Guillain-Barré syndrome associated with SARS-CoV-2 infection: A case series from 4 Colombian cities during the pandemic

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Abstract SARS-CoV-2 infection has been associated with multiple neurological manifestations. One such manifestation, which has been described since the early stages of the COVID-19 pandemic and is relevant for current neurological practice, is Guillain-Barré syndrome (GBS). The literature describes neurotoxic mechanisms of the virus itself and the possible pathways by which it may affect the peripheral nerves in experimental studies; however, we still lack information on the mechanisms causing the immune response that gives rise to GBS in the context of SARS-CoV-2 infection. Colombia is one of the Latin American countries worst affected by the pandemic, with the third-highest number of cases in the region; thus, it is essential to recognise GBS, as this potential postinfectious complication may severely compromise the patient's functional status in the absence of timely diagnosis and treatment. We present a series

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of 12 cases of GBS associated with SARS-CoV-2 infection from hospitals in 4 different Colombian cities and describe the clinical presentation, laboratory and electrophysiological study findings, and treatment.

PALABRAS CLAVE
Guillain-Barré;
Coronavirus;
SARS-CoV-2;
Debilidad;
Electrodiagnóstico;
Polineuropatía aguda;
Inmunoglobulina;
Recambio plasmático

Síndrome de Guillain Barré y Sars Cov2: serie de casos en el primer año de pandemia

Resumen En el año 2020 se declaro la pandemia ocasionada por la infección por el virus SARSCoV-2, virus de la familia del coronavirus, adoptándose el nombre de COVID-19 a la enfermedad 1. En Bogotá, Colombia, se confirmó el primer caso de COVID-19 el 6 de marzo de 2020 (2). Los principales síntomas reportados en la infección por SARSCoV-2 son fiebre (43.8% en la admisión y 88.7% durante la hospitalización) y tos (67.8%) (3). Otros síntomas encontrados son fatiga (38.1%), producción de esputo (33.7%) y cefalea (13.6%). Los principales signos neurológicos reportados en los pacientes con infección severa por SARS-Cov-2 son agitación (69%), compromiso en tracto corticoespinal (67%) y delirium (65%) (4). Las principales complicaciones neurológicas descritas asociadas a Covid 19 son: anosmia, disgeusia, encefalopatia, Síndrome de Guillain Barre, complicaciones cerebrovasculares y daño en musculo esquelético (5–8).
En el presente articulo se presenta una serie de casos de pacientes con síndrome de Guillain-Barré asociado a infección por SARS-CoV-2. Se recolectaron casos de diferentes instituciones medicas de Colombia.

Introduction

The COVID-19 pandemic associated with the novel coronavirus SARS-CoV-2 was declared in 2020.[1] The first confirmed case of COVID-19 in Bogota (Colombia) was recorded on 6 March 2020.[2] The main symptoms reported are fever (43.8% of cases at admission and 88.7% during hospitalisation) and cough (67.8%).[3] Other symptoms include fatigue (38.1%), sputum production (33.7%), and headache (13.6%). The main neurological signs reported in patients with severe COVID-19 are agitation (69%), corticospinal tract involvement (67%), and delirium (65%).[4] The main neurological complications associated with the disease are anosmia, dysgeusia, encephalopathy, Guillain-Barré syndrome (GBS), cerebrovascular complications, and skeletal muscle damage.[5–8]

We present a series of cases of GBS associated with SARS-CoV-2 infection. Cases were gathered from several Colombian hospitals.

Methods

We conducted a descriptive case series study between 2020 and 2021, gathering data from the following hospitals: Fundación Cardioinfantil (Bogota), Fundación Santafe de Bogotá (Bogota), Clínica Medilaser (Neiva), Clínica San Luis (Bucaramanga), and Clínica Ospedale (Manizales). The following inclusion criteria were established: age older than 18 years; clinical and electrophysiological diagnosis of acute neuropathy according to the Asbury criteria; symptoms suggestive of SARS-CoV-2 infection between 1 week and 2 months prior to or coinciding with acute neurological symptoms; or documented positive SARS-CoV-2 test result (antigen, polymerase chain reaction [PCR], or FilmArray respiratory panel) at the time of neuropathy or within a month before onset of weakness.

Results

Table 1 presents the main clinical and laboratory characteristics of patients with GBS associated with SARS-CoV-2 infection.

Discussion

The association between infection and GBS is extensively documented. The most frequently reported infectious agents associated with the syndrome are *Campylobacter jejuni*, cytomegalovirus, or *Mycoplasma pneumoniae*, accounting for up to two-thirds of cases.[9] Although this association was not described in the SARS-CoV-1 outbreak, it has been frequently reported in patients with COVID-19.[10] In the recent Zika virus disease outbreak in Colombia, nearly 70 cases of GBS were reported in patients with reverse transcription PCR-confirmed Zika virus infection.[11]

Peripheral nerve damage in COVID-19 has been reported in the current pandemic, with the first case reported in April

Table 1 Patients with Guillain-Barré syndrome and SARS-CoV-2 infection. Clinical, electrophysiological, and laboratory characteristics.

Patient	Sex	Age (years)	Previous respiratory/ gastrointestinal symptoms	Time to onset of neurological symptoms (days)	Pneumonia	Neurological signs/symptoms	Diagnosis of SARS-CoV-2 infection	CSF	EMG + NCS	Level of certainty (Brighton level)	Treatment	Modified Rankin Scale score at discharge (0 to 6)
1	M	55	Cough, odynophagia, diarrhoea	30	No	Sudden-onset dysarthria, facial diplegia	11/04/20 positive PCR	Proteins: 210 mg/dL; leukocytes: 0	Severe acute axonal neuropathy of the left and right facial nerves with involvement of the frontal, zygomatic, buccal, and marginal mandibular branches	1	None	1
2	M	73	Diarrhoea	7	No	Loss of muscle strength in all 4 limbs with inability to walk, loss of mobility Areflexia	28/09/20 positive PCR	Proteins: 55 mg/dL; leukocytes: 2.5 cells/μL (100% mononuclear)	Acute motor axonal polyradiculoneuropathy with minimal signs of acute denervation	1	IV immunoglobulins	3
3	M	54	Fever, diarrhoea	5	No	Neck and lumbar pain, upper and subsequently lower limb weakness, myalgia	01/10/20 positive FilmArray respiratory panel	Proteins: 67.4 mg/dL; leukocytes: 0	Demyelinating sensorimotor polyneuropathy, intrinsic muscle fibre disease	2	IV immunoglobulins	4
4	M	51	None	NA	No	Intense lumbar pain irradiating to the posterior aspect of both legs with progressive lower limb weakness and subsequent paraesthesia of the hands	21/10/20 positive PCR	Proteins: 149 mg/dL; leukocytes: 0	Demyelinating polyradiculoneuropathy with predominant lower limb involvement	2	Plasma exchange	3
5	M	62	Ageusia	1	No	Predominantly occipital holocranial pressing headache,	26/10/20 negative SARS-CoV-2 antigen test	ND	Predominantly axonal sensorimotor polyneuropathy	3	IV immunoglobulins	3

Patient	Sex	Age (years)	Previous respiratory/ gastrointestinal symptoms	Time to onset of neurological symptoms (days)	Pneumonia	Neurological signs/symptoms	Diagnosis of SARS-CoV-2 infection	CSF	EMG + NCS	Level of certainty (Brighton level)	Treatment	Modified Rankin Scale score at discharge (0 to 6)
						bilateral labial commissure deviation, right-sided hemiparesis, dysarthria, dysphagia, areflexia, glove-and-stocking paraesthesia	03/11/20 positive PCR					
6	W	57	Cough, dyspnoea, anosmia	20	Yes	Impaired level of consciousness, flaccid quadriparesis, areflexia	12/12/20 positive PCR	Proteins: 52.8 mg/dL; leukocytes: 0	20/01/21 acute demyelinating sensorimotor polyneuropathy	2	Plasma exchange	5
7	W	61	Yes	15	No	Progressive ascending lower limb weakness with generalised hypoaesthesia, fall from standing height	10/01/21 positive PCR	Proteins: 147 mg/dL; glucose: 175 mg/dL (central: 271 mg/dL); leukocytes: 2 cells/μL	Symmetrical motor axonal polyneuropathy	1	Plasma exchange	3
8	W	71	None (close contact)	NA	No	Paraesthesia, burning pain (proximal and subsequently distal in lower limbs, peripheral facial nerve territory)	20/02/21 inconclusive PCR; 22/02/ 21 positive PCR	Proteins: 230 mg/dL; leukocytes: 2.5 cells/μL	Predominantly demyelinating polyneuropathy with symmetrical motor involvement	1	Plasma exchange	3
9	M	57	Fever, diarrhoea	8	No	Lower limb paraesthesia, inability to walk, urinary	22/10/20 positive PCR	Proteins: 288 mg/dL; Leukocytes: 0		2	Plasma exchange	3

Table 1 (*continuación*)

Patient	Sex	Age (years)	Previous respiratory/ gastrointestinal symptoms	Time to onset of neurological symptoms (days)	Pneumonia	Neurological signs/symptoms	Diagnosis of SARS-CoV-2 infection	CSF	EMG + NCS	Level of certainty (Brighton level)	Treatment	Modified Rankin Scale score at discharge (0 to 6)
						incontinence, paraesthesia of the hands, areflexic flaccid paraparesis, glove-and-stocking hypoaesthesia, sensory ataxia						
10	M	43	Dysphagia, fever	15	No	Limb paraesthesia, facial diplegia	18/07/20 positive PCR	ND	Predominantly demyelinating acute sensorimotor polyneuropathy with an axonal component, mild severity, with more marked involvement of the cranial nerves. Given the clinical context, these findings suggest acute inflammatory demyelinating polyneuropathy	2	None	2
11	W	42	Fever, joint pain	8	No	Facial diplegia Paraesthesia in all 4 limbs Readmission: difficulty walking	20/07/21 positive PCR	26/07/21 CSF: proteins: 67 mg/dL; glucose: 59 mg/dL; leukocytes: 0 28/08/21 proteins: 220 mg/dL; glucose: 57 mg/dL;	Acute demyelinating sensorimotor polyneuropathy (segmental demyelination) with a secondary axonal component, mild severity, with more marked involvement of the facial nerves. Findings suggest acute inflammatory	1	IV immunoglobulins	3

Patient	Sex	Age (years)	Previous respiratory/ gastrointestinal symptoms	Time to onset of neurological symptoms (days)	Pneumonia	Neurological signs/symptoms	Diagnosis of SARS-CoV-2 infection	CSF	EMG + NCS	Level of certainty (Brighton level)	Treatment	Modified Rankin Scale score at discharge (0 to 6)
12	M	38	Cough, fever, dysphagia	45	No	Dysphagia Weakness in the lower limbs Unable to walk	15/05/21 positive PCR	leukocytes: 1 cell/μL ND	demyelinating polyneuropathy Acute motor axonal polyneuropathy with a demyelinating component, moderate severity	2	IV immunoglobulins	2

CSF: cerebrospinal fluid; EMG + NCS: electromyography with nerve conduction study (including F wave and H reflex); IV: intravenous; M: man; NA: not applicable; ND: no data; PCR: polymerase chain reaction; W: woman.

2020 in Wuhan (China) in a 61-year-old woman who presented acute lower limb weakness, areflexia, progressing within 1 day, with paraclinical findings of albuminocytologic dissociation, absence of F waves, and increased latency in the nerve conduction study. These symptoms preceded the respiratory symptoms.[7] Italian researchers reported a similar case in a 71-year-old patient, who developed acute weakness several days before the onset of infectious symptoms; this suggests that GBS may develop in parallel with SARS-CoV-2 infection.[9]

A systematic review of 39 studies, including a total of 50 patients with GBS manifesting after onset of COVID-19, found that the most common form was acute inflammatory demyelinating polyneuropathy (66%); the axonal variant was not infrequent, occurring in 34% of the population studied. In the majority of patients (66%), diagnostic certainty was level 1 according to the Brighton criteria. CSF protein levels were slightly more elevated in patients with axonal variants. Patients were treated with intravenous immunoglobulins and plasma exchange; neither treatment was found to be superior in terms of functional outcomes.[12]

The peripheral neurotropic mechanisms under study in relation to SARS-CoV-2 include a subset of human nociceptors expressing the *MRGPRD* and *CALCA* genes, which also express *ACE2* mRNA; the high affinity of SARS-CoV-2 for the ACE2 receptor would explain some painful phenomena in patients with this infection.[13]

We must also consider the role of the so-called "cytokine storm," an exaggerated immune response triggered by SARS-CoV-2 and characterised by increased production of multiple inflammatory factors, which mediates tissue damage in patients with COVID-19.[7] The immune mediators whose expression is increased in response to the infection include IL-1β, IL-2, IL-6, IL-7, IL-10, G-CSF, CXCL10, MCP-1, MIP-1α, and TNFα, with IL-1β, IL-6, CXCL10, and TNFα presenting the greatest capacity to cause tissue damage in various organs due to their proinflammatory properties. IL-1β and IL-6 have been associated with neurotoxicity and may cause endothelial dysfunction.[9]

A total of 54 lineages of SARS-CoV-2 are in circulation in Colombia. Of these, high frequency of variants B.1, B.1.111, and B.1.420 has been reported in 29 departments.[14] Over 13 different mutations of variant B.1.111 have been described. The first case of the Brazilian P.1 variant was recorded in March 2021 in Bogota; no further cases have been recorded.[15] Circulation of the British B.1.1.7 variant has also been documented.[16]

The cases presented in this article were recorded between the second and third waves of the pandemic. The cities with the largest numbers of patients are Bogota, Medellín, and Barranquilla. The second phase of vaccination is currently being completed in Colombia.[17] At least 28 910 000 cases of SARS-CoV-2 infection and 920 000 deaths due to COVID-19 have been recorded in Latin America; during the third wave, Colombia was the country with the third-highest infection rate in the region, after Brazil and Argentina.[14]

The mean age of the patients in our series was 55.3 years, with the youngest being 38 years old. We also observed differences between patients in the time of onset of muscle weakness, which ranged from 1 to 45 days after onset of infectious symptoms, with several presenting the neurological disorder 1–2 weeks after the respiratory symptoms. Two

patients did not present respiratory symptoms prior to GBS, with this syndrome being the initial manifestation of SARS-CoV-2 infection, which was subsequently detected in the aetiological study. One patient presented respiratory symptoms for 2 weeks, and was vaccinated a week after resolution of these symptoms.

Regarding the symptoms of GBS, 8 patients initially presented with progressive ascending lower limb weakness associated with sensory symptoms. One patient presented headache and right-sided hemiparesis with areflexia. Four patients presented peripheral facial nerve involvement, with facial diplegia in 3 and peripheral facial nerve palsy in 1. The upper limbs were affected at onset in only 1 patient, with another presenting impaired level of consciousness (coma); this, alongside the other findings of areflexia and weakness, may be considered a possible case of Bickerstaff encephalitis. Two patients presented rapid progression with an Erasmus GBS Respiratory Insufficiency Score (EGRIS) of 5, requiring ventilatory support. Dysautonomic symptoms were described in 1 patient. Though presentation was heterogeneous, ascending weakness was the predominant form, with diagnostic certainty of level 1 or 2 in the majority of cases. Antiganglioside antibody determination was not performed in any case; diagnostic certainty was established based on clinical picture and complementary test results. Only 2 patients presented pneumonia secondary to SARS-CoV-2 infection. The majority of patients presented mild respiratory or gastrointestinal symptoms, demonstrating that the clinical severity of COVID-19 was not directly correlated with the risk of developing acute neuropathy.

Regarding laboratory tests, the diagnosis of SARS-CoV-2 infection was confirmed by PCR or antigen tests in all patients. Cerebrospinal fluid (CSF) analysis data are available for 9 of the 12 patients included, with most showing albuminocytologic dissociation, small to large increases in protein levels, and normal cell counts. No patient presented pleocytosis or presence of the virus in the CSF.

Electrophysiological data (electromyography plus nerve conduction study, including H reflex and F wave) are available for 10 patients. Symmetrical axonal sensorimotor polyneuropathy was the most frequent electrophysiological finding, followed by acute motor demyelination; this contrasts with results reported in the literature, in which acute inflammatory demyelinating polyneuropathy is the most common form, in spite of an axonal and demyelinating component.

In terms of treatment, 5 patients were treated with sequential intermittent plasma exchange, and 5 received intravenous immunoglobulins, with no patient presenting adverse reactions; most patients presented clinical improvements and were discharged to continue with outpatient rehabilitation. No patient died. Two patients with mild GBS only required rehabilitation. It is noteworthy that the patient who was vaccinated was initially admitted with mild symptoms but subsequently presented clinical progression and an increase in CSF protein levels. Two of the 12 patients did not require treatment with intravenous immunoglobulins or plasma exchange due to mild forms of GBS, with facial diplegia and no gait impairment.

Regarding functional outcomes, most patients presented moderate to severe disability at discharge.

Conclusion

Among the neurological manifestations of COVID-19, we must be alert to acute weakness syndromes, and seek to promptly recognise and detect GBS. We must also be aware that the presentation of SARS-CoV-2–associated GBS can be highly heterogeneous, and the syndrome may even be the initial manifestation of the viral infection. In our series, the severity of respiratory symptoms was not correlated with the risk of presenting acute neuropathy, with most patients presenting mild respiratory symptoms. Albuminocytologic dissociation was observed in the majority of cases, in a comparable percentage of cases of axonal and demyelinating forms of the syndrome, and the response to plasma exchange was adequate.

Further research is needed to better understand the molecular mechanisms involved in SARS-CoV-2–associated acute neuropathy. However, in the light of our findings and the emerging status of this novel virus, routine testing for SARS-CoV-2 should be performed in the aetiological study of acute flaccid paralysis.

Ethical considerations

We declare that we followed our centre's protocols regarding the publication of patient data, and that all patients gave informed consent to the publication of their cases.

Conflicts of interest

The authors have no conflicts of interest to declare.

Declaration of interests

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.neurop.2022.06.004.

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