


CASE REPORT

Open Access

# Concomitant Guillain-Barre syndrome with COVID-19: a case report



Nuvia Mackenzie<sup>1</sup>, Eva Lopez-Coronel<sup>1</sup>, Alberto Dau<sup>1</sup>, Dieb Maloof<sup>1</sup>, Salvador Mattar<sup>1</sup>, Jesus Tapia Garcia<sup>1</sup>, Briyis Fontecha<sup>1</sup>, Cristina M. Lanata<sup>2</sup> and Hernan Felipe Guillen-Burgos<sup>1,3\*</sup> 

## Abstract

**Background:** During the Coronavirus disease 2019 (COVID-19) pandemic, different neurological manifestations have been observed. However, only a few cases of Guillain-Barre syndrome (GBS) and COVID-19 have been reported. Therefore, the aim of this study is to investigate a case of concomitant GBS with COVID-19 in Colombia.

**Case presentation:** A 39-year-old woman was admitted to a teaching hospital in Barranquilla, Colombia with a history of progressive general weakness with lower limb dominance. Previous symptoms such as ageusia, anosmia and intense headache were reported. Upon admission, facial diplegia, quadriparesis with lower extremity predominance and Medical Research Council muscular strength of 2/5 in the lower limbs and 4/5 in the upper limbs were reported. During clinical evolution, due to general areflexia, hypertensive emergency and progressive diaphragmatic weakness, the patient was admitted to an intensive care unit. The cerebrospinal fluid analysis showed protein-cytological dissociation and the GBS diagnosis was confirmed via a nerve conduction and electromyography test. With regard to the symptoms before hospitalisation, SARS-CoV-2 diagnostic testing was performed with positive results in the second test. The patient was managed with supportive care and was discharged after 20 days of hospitalization with clinical improvement.

**Conclusions:** Only a few cases of COVID-19 with GBS have been reported. Different subtypes have been previously identified, such as Miller-Fisher syndrome and dysautonomic GBS with SARS-CoV-2 infection. This study investigated the first confirmed case of COVID-19 with concomitant GBS in Colombia. In patients with GBS, several viral and bacterial pathogens have been found in case-control studies but the factors that induce the immune-mediated destruction of the nerve tissues have not been determined. Further studies are needed to determine the possible association between COVID-19 exposure and GBS.

**Keywords:** COVID-19, SARS-CoV-2, Guillain-Barre syndrome, Case report

\* Correspondence: [hernan.guillen@unisimonbolivar.edu.co](mailto:hernan.guillen@unisimonbolivar.edu.co)

<sup>1</sup>Center for Clinical and Translational Research, La Misericordia Clínica Internacional, Barranquilla, Colombia

<sup>3</sup>Universidad Simón Bolívar, Facultad de Ciencias de la Salud, Barranquilla, Colombia

Full list of author information is available at the end of the article



© The Author(s). 2021 **Open Access** This article is licensed under a Creative Commons Attribution 4.0 International License, which permits use, sharing, adaptation, distribution and reproduction in any medium or format, as long as you give appropriate credit to the original author(s) and the source, provide a link to the Creative Commons licence, and indicate if changes were made. The images or other third party material in this article are included in the article's Creative Commons licence, unless indicated otherwise in a credit line to the material. If material is not included in the article's Creative Commons licence and your intended use is not permitted by statutory regulation or exceeds the permitted use, you will need to obtain permission directly from the copyright holder. To view a copy of this licence, visit <http://creativecommons.org/licenses/by/4.0/>. The Creative Commons Public Domain Dedication waiver (<http://creativecommons.org/publicdomain/zero/1.0/>) applies to the data made available in this article, unless otherwise stated in a credit line to the data.

## Background

A novel coronavirus emerged in Wuhan, China in late December 2019 generating a pandemic disease named coronavirus disease 2019 (COVID-19) caused by the SARS-CoV-2 virus [1]. The rapid spread of the virus from China to Europe, the United States and currently Latin American countries has caused several deaths and cumulative cases in countries with overwhelmed sanitary systems, such as Italy, Spain, the United States and several regions in South America [2]. Moreover, the United States and Brazil were the first and second hotspots the disease with more cases and deaths reported, respectively [3]. In Colombia to date, more than 2.161.462 cumulative cases and more than 56.290 deaths [4] with cluster regions, such as Bogota, D. C, and Barranquilla have been reported. Furthermore, this brief report investigates a case of COVID-19 with the concomitant Guillain Barre syndrome (GBS) variant from the Hospital La Misericordia Clínica Internacional in Barranquilla, Colombia.

## Case presentation

During mid-April 2020, a 39-year-old woman with previous medical history of hypertension controlled with medication, type 2 diabetes mellitus of recent diagnosis controlled with medication, and cholecystectomy (6 years ago) was admitted to a teaching hospital in Barranquilla, Colombia, and presented with symptoms of progressive generalised weakness for 6 days, predominantly involving the lower limbs. Twenty days prior to admission, she had an episode of illness consisting of ageusia, anosmia and intense headache for which she had multiple consultations without improvement of the symptoms. She also complained of intense myalgias and symmetric, ascendant and generalised weakness in the lower limbs, which gradually progressed to inability to walk for which she was referred to our hospital. Upon admission, the patient reported symptoms of headache, malaise, generalised myalgias, cough without expectoration and inability to walk. Her blood pressure on sitting was 167/88 mmHg, heart rate was regular (74 beats per min), respiratory rate was 18 breaths per min (bpm) and SaO<sub>2</sub> was 95% on room air. Based on her neurological examination, she was found to have peripheral facial diplegia, generalised hyporeflexia, generalised quadriparalysis with lower limb predominance, Medical Research Council (MRC) muscular strength of 2/5 in the lower limbs and 4/5 in the upper limbs. She also had left arm paraesthesia. No affection was observed in the bulbar muscles. During the clinical evolution she had generalised flaccid areflexia. She was later shifted to the intensive care unit due to hypertensive emergency (189/112 mmHg), and progressive diaphragmatic weakness with 36 bpm, without breath sounds in auscultation, which could indicate dysautonomic symptoms. Table 1 shows

**Table 1** Laboratory testing upon admission

Laboratory testing upon admission		
Complete blood count		Reference range
Total leucocyte count	12.57 (10 <sup>3</sup> /mm <sup>3</sup> )	4–10.04 (10 <sup>3</sup> /mm <sup>3</sup> )
Neutrophils	79.9%	34–71.1%
Lymphocytes	13%	19.3–51.7%
Monocytes	6.6%	4.7–12.6%
Eosinophils	0.4%	0.7–5.8%
Basophils	0.1%	0.1–1.2%
Haematocrit	35.6%	34.1–44.9%
Haemoglobin	11.2 g/dL	11.2–15.7 (gr/dL)
Platelets	232 (10 <sup>3</sup> uL)	150–450 (10 <sup>3</sup> uL)
<b>Electrolytes</b>		
Sodium	136 mmol/L	135–148 mmol/L
Potassium	4.57 mmol/L	3.3–5.1 mmol/L
Chloride	96.5 mmol/L	98–107 mmol/L
<b>Blood glucose levels</b>		
Glycemia	240 (mg/dL)	70–105 (mg/dL)
<b>Others</b>		
Lactate dehydrogenase	306 (U/L)	140–280 U/L
Ferritin	114.5 ng/mL	15–150 ng/mL
D-dimer	517 ng/mL	< 250 ng/mL
Serum creatinine	0.51 mg/dL	0.5–1.1 mg/dL
Erythrocyte sedimentation rate (ESR)	65	0–25 mm/hour

the results of the initial laboratory tests indicating leukocytosis, neutrophilia (79.90%, with a neutrophil-lymphocyte ratio of 16.27), lactate dehydrogenase and elevated D-Dimer levels. Creatine phosphokinase (CPK) levels were normal. The cerebrospinal fluid analysis revealed protein cytological dissociation (298.3/0.05). Elevated cerebrospinal fluid (CSF) protein is a hallmark of GBS. The chest X-ray and chest CT scan (128-slice SOMATOM, Siemens, GE) were normal. A nerve conduction and electromyography test (Table 2) confirmed the clinical evaluation of the GBS diagnosis. The MRI showed an incipient degenerative cervical disc disease at the C5-C6 level, normal thoracic spine, a Pfirrmann IV degenerative lumbar disc disease with central protrusions and flavum ligamentum hypertrophy at levels L3-L4, L4-L5 and L5-S1, which are not related with the clinical signs and symptoms of the patient.

The patient was managed with supportive care; administered with enoxaparin (40 mg SC per day), losartan (50 mg BID), meperidine IV for muscle pain, hydroxychloroquine (400 mg twice first day and later on 200 mg BID per 10 days) and dexamethasone (8 mg every 8 h during 3 days); and received standard care. She did not require

**Table 2** Electromyography test

Nerve conduction study	Stimulation point	Record point	Distal latency (ms)	Amplitude (mV)	Velocity (m/s)
<b>Motor NCS</b>					
Right tibial nerve	Ankle-popliteal fossa	ABD halluc is brevis	14.1	3.2	19.9
Right common peroneal nerve	Ankle-head of fibula	EXT digitorum brevis	13.7	3.0	19.4
Left tibial nerve	Ankle-popliteal fossa	ABD halluc is brevis	14.7	3.4	19.7
Right median nerve	Wrist-elbow	ABD pollicis brevis	26.3	4.2	23.8
Left median nerve	Wrist-elbow	ABD pollicis brevis	24.2	4.5	23.4
<b>Sensitive NCS</b>					
Right sural nerve	Ankle	Calf	14.9	3.2	–

supplemental oxygen or ventilation, and no alterations were observed in the chest images. She also received plasmapheresis for 5 days, one session per day. After three sessions, the patient had neurological improvement, (MRC) muscular strength of 3/5 in the lower limbs, and improved respiratory rate (20 bpm). During the hospitalization, the patient underwent two nasal swab tests for SARS-CoV-2. The first RT-PCR test (Charité/Berlin Protocol) for SARS-CoV-2 was negative, whereas the second RT-PCR test (Charité/Berlin Protocol) for SARS-CoV-2 was positive. She was discharged after 20 days of hospitalisation with improvement of neurological status (MRC of 4/5 in the lower limbs) and respiratory symptoms.

### Discussion and conclusions

Neurological outcomes in patients with COVID-19 have been reported [5]. The neurological manifestations range from headache, dizziness, confusion, to in a few cases, more severe conditions, such as encephalopathy, acute disseminated encephalomyelitis and GBS, among others [6].

GBS is a neuroinflammatory disease with a global incidence of 1–2 per 100,000 person-years [7]. GBS is the most common cause of acute flaccid paralysis and its diagnosis includes patient history, neurological examination, electrophysiological test and CSF analysis [7]. Several viral and bacterial pathogens have been found in GBS patients in case-control studies, but the factors that induce the immune-mediated destruction of the nerve tissues have not been determined [8]. Moreover, the association between previous exposition to pathogens (e.g. cytomegalovirus, Epstein-Barr virus, *Mycoplasma pneumoniae*, Zika virus, *Campylobacter jejuni*. and novel coronavirus SARS-CoV-2) and GBS outcome has been reported [9–13].

This study reports the first case of concomitant GBS with COVID-19 with a travel history from Wuhan [14], followed by other cases in countries with a high incidence of SARS-CoV-2, such as Italy [13], Iran [15] and Spain [16]. Other subtypes of GBS have been identified with SARS-CoV-2 infection such as Miller-Fisher

syndrome (MLS) [17] and dysautonomic GBS [18]. Although a possible hypothesis of GBS caused by COVID-19 has been proposed, only fewer cases have been reported [19]. Nevertheless, in patients with GBS and COVID-19, it not possible to identify SARS-CoV-2 virus via CSF analysis. A retrospective systematic screening of CSF samples from COVID-19 patients, showed negative findings [20].

We speculate that polyneuropathies, such as GBS, could be identified in COVID-19 patients, but more studies are needed to explore the neurological outcomes of these kinds of patients. It is not clear whether the infection preceded the GBS, or whether the infection is concomitant or considered a casualty in this group of patients. Because GBS is rare and the pandemic continues, the information contained in this report is very important for future research. More studies are needed to determine if COVID-19 has a direct association with GBS, such as its previously reported association with the Zika virus (ZIKV) [12]. During the last outbreak of the ZIKV in Colombia, the researchers reported virological evidence of ZIKV in patients with GBS in Colombia [12].

In summary, this study reported the first case of concomitant GBS with COVID-19 in Colombia, with favourable outcomes. As the pandemic continues, we would be able to determine whether this potential association could be attributed to the increase in GBS cases.

### Abbreviation

BID: Twice a day, *bis in die*; COVID-19: Coronavirus disease 2019; CPK: Creatine phosphokinase; CSF: Cerebrospinal serum fluid; GBS: Guillain-Barre syndrome; LDH: Lactate dehydrogenase; MRC: Medical Research Council; RT-PCR: Reverse transcription-polymerase chain reaction; SARS-CoV-2: Severe acute respiratory syndrome coronavirus 2; SC: Subcutaneous; ZIKV: Zika virus

### Acknowledgments

Not applicable.

### Authors' contributions

Design of work: N. M, EL-C, A. D, D. M, S. M, J.T.G, B. F and C.M.L H.F.G-B. Acquisition and analysis: N. M, EL-C, B. F and H.F.G-B. Interpretation of data: N. M, H.F.G-B. Writing and submission: N. M, C.M.L and H.F.G-B. All authors have read and approved the manuscript.

### Funding

The author(s) received no specific funding for this work.

### Availability of data and materials

All data are included in the electronic medical record of the patient. The datasets generated and/or analysed during the current study are not publicly available due to our policy statement of sharing clinical data only on request but are available from the corresponding author on reasonable request.

### Declarations

#### Ethics approval and consent to participate

This work was conducted according to the Helsinki Declaration, Belmont Report, International Ethical Guideline for Biomedical Research Involving Human Subjects and Council of International Organizations of Medical Sciences. Written informed consent was obtained from the patient to participate in this case report with anonymised data.

#### Consent for publication

Written informed consent was obtained from the patient for publication as a case-report with anonymised data.

#### Competing interests

The authors declare no conflict of interest.

#### Author details

<sup>1</sup>Center for Clinical and Translational Research, La Misericordia Clínica Internacional, Barranquilla, Colombia. <sup>2</sup>Rosalind Russell/Ephraim P Engleman Rheumatology Research Center, University of California, San Francisco, California, USA. <sup>3</sup>Universidad Simón Bolívar, Facultad de Ciencias de la Salud, Barranquilla, Colombia.

Received: 24 July 2020 Accepted: 18 March 2021

Published online: 23 March 2021

### References

- Huang C, Wang Y, Li X, Ren L, Zhao J, Hu Y, et al. Clinical features of patients infected with 2019 novel coronavirus in Wuhan, China. *Lancet*. 2020;395(10223):497–506. [https://doi.org/10.1016/S0140-6736\(20\)30183-5](https://doi.org/10.1016/S0140-6736(20)30183-5).
- Burki T. COVID-19 in Latin America. *Lancet Infect Dis*. 2020;20(5):547–8. [https://doi.org/10.1016/S1473-3099\(20\)30303-0](https://doi.org/10.1016/S1473-3099(20)30303-0).
- Dong E, Du H, Gardner L. An interactive web-based dashboard to track COVID-19 in real time. *Lancet Infect Dis*. 2020;20(5):533–4.
- Ministerio de Salud y Protección Social. Tablero de Reporte de Casos. Coronavirus (COVID-19). Consultated (February 8th, 2021) in: <https://covid19.minsalud.gov.co/>.
- Roy D, Ghosh R, Dubey S, Dubey MJ, Benito-León J, Kanti RB. Neurological and neuropsychiatric impacts of COVID-19 pandemic. *Can J Neurol Sci*. 2021;48(1):9–24. <https://doi.org/10.1017/cjn.2020.173>.
- Ellul MA, Benjamin L, Singh B, Lant S, Michael BD, Easton A, et al. Neurological associations of COVID-19. *Lancet Neurol*. 2020;19(9):767–83. [https://doi.org/10.1016/S1474-4422\(20\)30221-0](https://doi.org/10.1016/S1474-4422(20)30221-0).
- Sejvar JJ, Baughman AL, Wise M, Morgan OW. Population incidence of Guillain-Barré syndrome: A systematic review and meta-analysis. *Neuroepidemiology*. 2011;36(2):123–33. <https://doi.org/10.1159/000324710>.
- Hardy TA, Blum S, McCombe PA, Reddel SW. Guillain-Barré syndrome: modern theories of etiology. *Curr Allergy Asthma Rep*. 2011;11(3):197–204. <https://doi.org/10.1007/s11882-011-0190-y>.
- Orlikowski D, Porcher R, Sivadon-Tardy V, Quincampoix JC, Raphaël JC, Durand MC, et al. Guillain-Barre syndrome following primary cytomegalovirus infection: A prospective cohort study. *Clin Infect Dis*. 2011; 52(7):837–44. <https://doi.org/10.1093/cid/cir074>.
- An JY, Yoon B, Kim JS, Song IU, Lee KS, Kim YI, et al. Guillain-Barré syndrome with optic neuritis and a focal lesion in the central white matter following Epstein-Barr virus infection. *Intern Med*. 2008;47(17):1539–42. <https://doi.org/10.2169/internalmedicine.47.1224>.
- Meyer Sauter PM, Roodbol J, Hackenberg A, de Wit MC, Vink C, Berger C, et al. Severe childhood Guillain-Barré syndrome associated with mycoplasma pneumoniae infection: a case series. *J Peripher Nerv Syst*. 2015; 20(2):72–8. <https://doi.org/10.1111/jns.12121>.
- Parra B, Lizarazo J, Jiménez-Arango JA, Zea-Vera AF, González-Manrique G, Vargas J, et al. Guillain-Barré syndrome associated with Zika virus infection in Colombia. *N Engl J Med*. 2016;375(16):1513–23. <https://doi.org/10.1056/NEJMoa1605564>.
- Toscano G, Palmerini F, Ravaglia S, Ruiz L, Invernizzi P, Cuzzoni MG, et al. Guillain-Barré syndrome associated with SARS-CoV-2. *N Engl J Med*. 2020; 382(26):2574–6. <https://doi.org/10.1056/NEJMc2009191>.
- Zhao H, Shen D, Zhou H, Liu J, Chen S. Guillain-Barré syndrome associated with SARS-CoV-2 infection: causality or coincidence? *Lancet Neurol*. 2020; 19(5):383–4. [https://doi.org/10.1016/S1474-4422\(20\)30109-5](https://doi.org/10.1016/S1474-4422(20)30109-5).
- Sedaghat Z, Karimi N. Guillain Barre syndrome associated with COVID-19 infection: a case report. *J Clin Neurosci*. 2020;76:233–5.
- Romero-Sánchez CM, Díaz-Maroto I, Fernández-Díaz E, Sánchez-Larsen Á, Layos-Romero A, García-García J, et al. Neurologic manifestations in hospitalized patients with COVID-19: the ALBACOV registry. *Neurology*. 2020;95(8):e1060–70.
- Gutiérrez-Ortiz C, Méndez-Guerrero A, Rodrigo-Rey S, San Pedro-Murillo E, Bermejo-Guerrero L, Gordo-Mañas R, et al. Miller fisher syndrome and polyneuritis cranialis in COVID-19. *Neurology*. 2020;95(5):e601–5.
- Ghosh R, Roy D, Sengupta S, Benito-León J. Autonomic dysfunction heralding acute motor axonal neuropathy in COVID-19. *J Neuro-Oncol*. 2020;26(6):964–6.
- Finsterer J, Scorza FA, Ghosh R. COVID-19 polyradiculitis in 24 patients without SARS-CoV-2 in the cerebro-spinal fluid. *J Med Virol*. 2020;93(1):66–8. <https://doi.org/10.1002/jmv.26121>.
- Billaud G, et al. Systematic SARS-CoV-2 screening in cerebrospinal fluid during the COVID-19 pandemic. *Lancet Microbe*. 2020;1(4):e149.

### Publisher's Note

Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.

Ready to submit your research? Choose BMC and benefit from:

- fast, convenient online submission
- thorough peer review by experienced researchers in your field
- rapid publication on acceptance
- support for research data, including large and complex data types
- gold Open Access which fosters wider collaboration and increased citations
- maximum visibility for your research: over 100M website views per year

At BMC, research is always in progress.

Learn more [biomedcentral.com/submissions](https://biomedcentral.com/submissions)

