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Guillain-Barré syndrome in a COVID-19 patient

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Abstract

Coronavirus type 2 is a β-coronavirus whose infection is characterized by a predominantly respiratory clinical picture. However, neurological symptoms are garnering great interest related to pulmonary infection and direct viral invasion of the central nervous system, with a possible association between Guillain-Barré syndrome and SARS-CoV-2 infection. This report describes this relationship in a 44-year-old female patient with classical Guillain-Barré syndrome signs and symptoms on admission, and respiratory signs and symptoms six days prior to the onset of neurological symptoms. There were positive SARS-CoV IgG and IgM blood tests and an epidemiological link of direct contact with people infected with SARS-CoV-2. She required ICU care due to the risk of respiratory failure, along with immunoglobulin treatment, but did not need mechanical ventilation; she improved and was discharged. One month later she consulted again and was thought to have had a Guillain-Barré relapse. She was hospitalized and treated until she progressed and her symptoms resolved. **(Acta Med Colomb 2022; 47. DOI: https://doi.org/10.36104/amc.2022.2204).**

Keywords: *SARS-CoV-2, COVID-19, Guillain-Barré, neuropathies, demyelinating diseases.*

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Introduction

Guillain-Barré syndrome (GBS) is a neurological disorder classified as a mixed axonal and demyelinating acute polyneuropathy. It occurs mainly in childhood, but it may debut at any age. It is caused by immunological phenomena which destroy the myelin sheath of peripheral nerves and may be secondary to infectious, toxic, or biochemical agents, or in the context of tumors (1).

It is known as a demyelinating disease because the main damage is in the myelin of peripheral nerves, which causes paresis, muscle weakness and even bilateral ascending paralysis. If the nerve damage reaches the diaphragmatic nerves, the patient may need invasive ventilatory support. Guillain-Barré syndrome has no cure, only symptomatic treatment and ventilatory support, if needed (2).

SARS-CoV-2 infection is especially virulent in people with chronic diseases and those who are immunosuppressed. Although respiratory signs and symptoms predominate in COVID-19, studies have been reported which mention neurological manifestations secondary to this infection affecting at least 36% of patients, in addition to vascular and kidney disorders (2). The most common neurological finding is anosmia. Nervous system disorders are more common in serious infections than in mild infections (3).

Various studies have shown that angiotensin-converting enzyme 2 (ACE2) acts as a functional receptor of SARS-CoV in human tissues. Due to the sequential similarity of the S proteins in SARS-CoV and SARS-CoV-2, it was predicted that SARS-CoV-2 also uses ACE2 as a functional receptor (3, 4, 5). Viral docking to ACE2 in the blood-brain barrier, facilitating its entrance to the central nervous system, as well as the possible existence of retrograde, transcribriform and hematogenous dissemination routes, are proposed as possible mechanisms through which SARS-CoV-2 can cause neurological damage (6, 7).

Different healthcare centers have reported several patients admitted with GBS who were actively COVID-19 positive or had had the disease, leading to a proposed association between both diseases. Some authors state that COVID-19 related GBS causes more acute onset symptoms (8). Different mechanisms through which the virus produces acute arreflexia have been proposed: antibodies produced against viral glycoproteins could also interact against native neuronal surface proteins (9).

In light of this clinical case, we reinforce the hypothesis of an association between GBS and SARS-CoV-2 infection, as has already been documented by other authors (10-12).

Case presentation

This was a 44-year-old female patient with no significant medical history. She had experienced 20 days of respiratory symptoms together with a mild, oppressive, nonradiating holocranial headache, not improved at all by taking NSAIDs, and accompanied by asthenia, adynamia, arthralgias, myalgias, anosmia, dysgeusia and general malaise. The patient reported that after six days with the previously described symptoms, she began to show lower extremity paresthesias, dysesthesias and hyperalgesia along with decreased muscle strength in the upper extremities.

On December 30, 2021, she took a SARS-CoV-2 antigen test which was positive for IgM and IgG. A chest HRCT was also taken as a diagnostic aid (Figure 1), showing a peripherally distributed ground-glass appearance in all four quadrants with upper left predominance and involvement of 25% of the lung parenchyma.

The patient isolated at home for approximately five days, during which she self-medicated with acetaminophen with codeine for symptom management, with no immediate improvement.

On January 7, 2021, she was admitted to the emergency room at Hospital Departamental de Nariño with anosmia, dysgeusia, and symmetric loss of strength in the extremities associated with paresthesias, dysesthesias and a 10/10 holocranial headache. On physical exam she had normal deep tendon reflexes (DTR: ++ /++++). The patient had lab test results taken on January 5 (1) which were notable for leukocytosis, neutrophilia and lymphopenia. She was hospitalized for inpatient treatment in the COVID area.

Due to the patient's neurological symptoms and atypical presentation, an electromyography was ordered, finding moderate symmetrical proximal sensory-motor and distal myelinic polyradiculoneuropathy, with abnormal nerve conduction and prolonged distal latency, indicating a distal myelinic lesion of the left median motor and orthodromic, sural and tibial nerves. With these results, the patient was diagnosed with atypical GBS. A CSF sample was also taken, with no abnormalities found.

Table 1. *Lab tests on the patient's admission and readmission.*

Lab test	Admission (7/01/21)	Readmission (14/02/21)
Leukocytes (x10^3/uL)	17,960	5,900
Hemoglobin (g/dL)	15.2	14.8
Hematocrit (%)	45.3	44.2
Platelets (x10^3/uL)	396	229
Neutrophils (%)	79	54
Lynphocytes (%)	18	38
Blood glucose (mg/dL)	74	NR
Creatinine (mg/dL)	1.1	0.89
LDH (U/L)	208	NR
CRP (g/dL)	<6	0.3
Potassium (mmol/dL)	4.76	NR
Sodium (mmol/dL)	138	NR
TSH (lU/mL)	1.93	NR
CPK	23	NR

LDH: lactate dehydrogenase, PCR: C-reactive protein, TSH: thyroid stimulating hormone, CPK: creatine phosphokinase.

Treatment for GBS was begun, administering Gammaraas immunoglobulin (normal human immunoglobulin) at 0.4 g/kg/day for five days, pregabalin tablets 75 mg po every 12 hours and 25 mg quetiapine tablets, one per day, for pain.

On January 12, 2021, five days after admission, the patient was transferred to the ICU due to the risk of acute

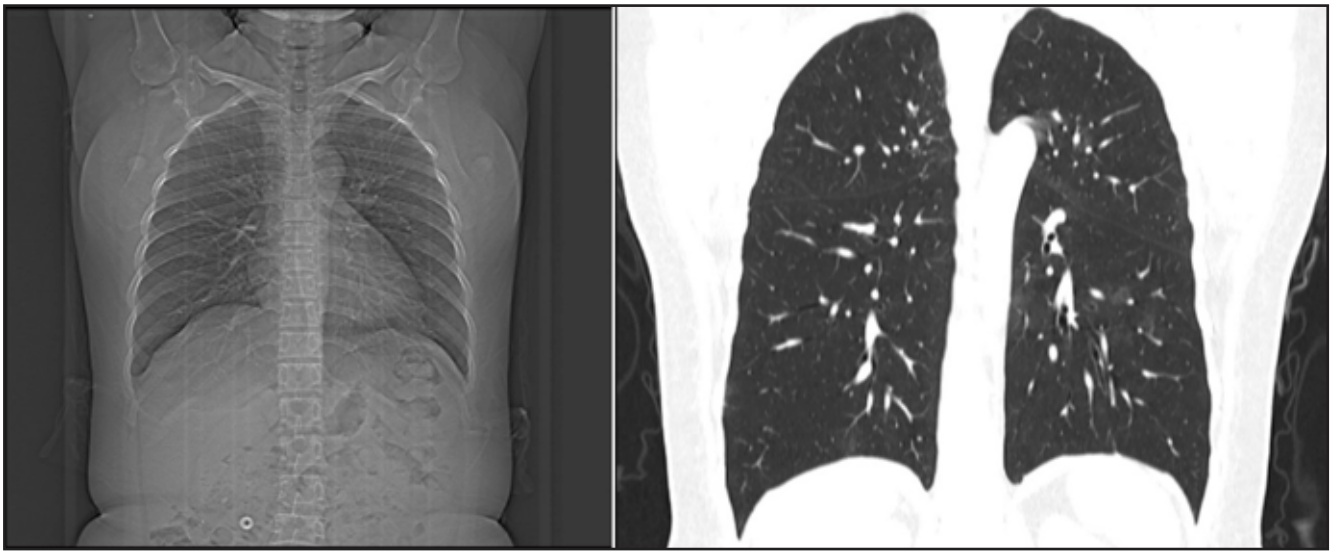

Figure 1. *Chest HRCT with a peripheral ground-glass pattern in all four quadrants, predominantly the upper left.*

respiratory failure. She was monitored in the ICU for three days and the immunoglobulin treatment was continued until January 14, when she was transferred to the internal medicine department where she continued with pain treatment and completed the immunoglobulin treatment. She was discharged on January 15, 2021.

On February 14, 2021, approximately one month after discharge, the patient consulted again with a five-day history of a holocranial headache radiating to the eyes, associated with facial paresthesia, blurred vision, asthenia, adynamia, myalgias and weakness in the extremities. On physical exam, the patient had stabbing paresthesias and general dysesthesia, along with diminished symmetrical ++++/+++++ and distal +++/+++++ muscle strength, with no meningeal signs.

The neurology service considered this to be a GBS relapse and the patient was hospitalized for treatment once again with immunoglobulin 0.4 g/kg per day for five days, plus cyanocobalamin, dexamethasone 8 mg IV every 12 hours, diphenydramine 50 mg po and pregabalin. This treatment began on February 15 and ended on February 19 when the patient was discharged, as she progressed satisfactorily and showed symptom resolution.

Discussion

Different cases of patients with COVID-19 associated GBS have been reported to date. However, a great deal of information regarding this association and its implications is still lacking. The purpose of this report is to analyze the available evidence on this topic in the adult population, presenting a case compatible with both illnesses and their joint presentation. Some differences between this case and the other studies analyzed can be seen, notably differences in a greater severity of the disease in COVID-19 associated GBS cases.

According to the literature, there has been an abnormal increase in patients admitted for GBS, showing a much older age prevalence, with a mean of 60 years, unlike past cases of this syndrome with a mean of 40 years (13-15). This case study still falls within the age group that had the greatest frequency of GBS prior to its association with COVID-19. More causal studies (RCTs) will be needed to provide more accurate results and conclusions.

In most cases, the symptoms prior to developing this syndrome were ageusia and hyposmia, and COVID-19 respiratory symptoms, like pneumonia, were more severe (16).

The time of onset of the first neurological signs and symptoms has been found to range from five to 21 days after the COVID-19 symptoms (17-19), which coincides with the case we are discussing.

The studies analyzed are case reports or case series with small samples and, therefore, the analysis categorizes them as having a high risk of bias. However, despite this limitation, it is important to emphasize that a tendency is being shown towards an association between both illnesses with this new presentation of symptoms.

More studies are needed with higher-level evidence designs and more representative samples in order to carry out conclusive analyses on this topic.

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