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Guillain Barre vs. Miastenia Gravis as an Atypical Presentation of SARS COV 2 By Molecular Mimic: Purpose of a Case

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Abstract

Introduction: Guillain-Barré syndrome (GBS) is an acute polyradiculoneuropathy due to an autoimmune process, which is triggered in most cases by a viral or bacterial infection, its incidence is 1.75/100,000 inhabitants worldwide. It is a very rare pathology and even less frequent are the cases in young women, so we carry out a review of its epidemiology, clinical manifestations, diagnostic criteria and management. Objective: In this work the case of a young adult woman with GBS is presented.

Description of the case: This is a 22-year-old female patient, who was admitted for 10 days at the ESE Hospital La Divina Misericordia with a picture of approximately 1 hour of evolution consisting of dysphagia, odynophagia, asthenia, episodes of dizziness, adynamia, accompanied by a decrease in muscle strength in the upper limbs, also associated with loss of tongue sensitivity.

Conclusions: GBS in young women is a rare variant. In most reports the course of the episodes is unpredictable.

Keywords: Guillain barré syndrome; Myasthenia gravis; Sars-CoV-2; Molecular mimicry

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Introduction

Every day, more neurological manifestations related to SARS-CoV-2 infection are being registered, among which we highlight Guillain Barré syndrome and Myasthenia Gravis.

Guillain-Barré syndrome (GBS) consists of an autoimmune polyneuropathy, with heterogeneous clinical variants, in most cases it presents as a monophasic paralysis preceded by a viral or bacterial infection, such as influenza, HIV, herpes virus and now by SARS-CoV-2; It is a disease that presents with acute paralysis secondary to inflammation of peripheral nerves and nerve roots, clinically manifested as paresthesia, numbness and progressive weakness of the extremities until walking is impossible. It can affect the muscles of the face, swallowing and ventilation [1].

On the other hand, Myasthenia Gravis (MG) is an autoimmune disease that occurs through the post-synaptic blockage of the myoneural plaque given by autoantibodies that bind to Acetylcholine Receptors (HACR) or to molecules of the postsynaptic membrane, which that generates fatigue and localized or generalized muscle weakness, predominantly proximal and with a fluctuating course. Muscle weakness is characterized in that it can become very severe, causing dysphagia and compromising the respiratory muscles, reaching a state in which the patient will require mechanical ventilation and endotracheal intubation [2].

The relationship between these autoimmune diseases and SARS-CoV-2, the trigger for the Covid 19 pandemic, which we remember is the seventh coronavirus isolated and characterized by being capable of causing infections in humans, which encodes 4 structural proteins: protein S (spike protein), protein E (envelope), protein M (membrane) and protein N (nucleocapsid). The N protein is inside the virion associated with the viral RNA, and the other four proteins are associated with the viral envelope. Protein S assembles into homotrimers, forming structures that protrude from the virus envelope. Protein S contains the binding domain to the cell receptor and is therefore the determining protein of the tropism of the virus and is also the protein that has the fusion activity of the viral membrane with the cell and thus allows the release of the viral genome inside the cell to be infected, it would be explained through the process of molecular mimicry, which is defined as the similarity in the antigenic determinants of two specific molecules. Being able to happen that a microorganism presents a molecular mimicry with a molecule of an immunocompetent host. In this case, the antibodies produced against the microorganism would react with the host molecule, causing an autoimmune disease [3].

This would be supported, in the investigations carried out, in which they affirm that molecular mimicry could explain some autoimmune events observed in Covid 19 disease, according to the authors, there are 33 different 8 mer/9 mer peptides, that are identical between SARS-CoV-2 and the reference proteome,

among human antigens that SARS-CoV-2 mimic, there are four human helicases involved (MCM8, DNA2, MOV10L1 and ZNFX1) [4].

Additionally, it is known that the pathophysiological mechanism of the entry of the virus into the cell, through the viral protein Spike, not only occurs through the receptor for the angiotensin-converting enzyme-2, but also uses glycoproteins and gangliosides that contain sialic acid on cell surfaces. As the SARS-CoV-2 spike protein interacts with the GalNAc residue of GM1 and ganglioside dimers to anchor to cell surface gangliosides, the likelihood of cross-reactivity between ganglioside-bearing epitopes is predicted Spike protein and peripheral nerve glycolipids at this level is elevated [5].

As far as we know, around 50 cases of GBS associated with SARS-CoV-2 have been described in the world, 2 of these cases having been reported in Colombia, now with regard to their relationship with MG, the spectrum is narrower, since most reports are based on cases of patients with a previous diagnosis of MG, with very few reporting cases of MG debut in relation to the new coronavirus [5].

In the presentation of our case, we raised the SARS-CoV-2 infection as a possible triggering event of MG or GB in the patient, based on the theory of molecular mimicry as a possible cause of the condition, in light of this case in clinical practice, we reinforce the hypothesis of the association between Guillain-Barré/ Myasthenia Gravis syndrome and SARS-CoV-2 virus infection, as has already been documented by other authors.

Case Report

A 21-year-old woman from Talaigua Nuevo, Bolívar municipality, Republic of Colombia, who is admitted to the emergency service due to a sudden onset clinical picture of approximately 1 hour of evolution consisting of dysphagia, odynophagia, asthenia, episodes of dizziness, adynamia, accompanied by decreased muscle strength in upper limbs, also associated with loss of tongue sensitivity; Additionally, the patient states that the only symptom that he presented in the last days was an occasional dry cough with a predominance of the day.

Physical examination revealed 3/5 muscle strength in all limbs, Achilles and Patellar hyporeflexia, accompanied by difficulty in changing position from supine to sitting position. On physical examination at the pulmonary level, slight decrease in vesicular murmur in the right base, without added noises.

Patient denied recent infectious or pathological antecedents, having as the only antecedent hypertensive disorder in pregnancy type Severe Preeclampsia.

The diagnostic possibility of acute inflammatory polyneuropathy versus mysthenic crisis as Debut was raised. However, given the respiratory symptoms and the clinical findings, it was not ruled out that the condition was related to Sars-cov-2 infection.

Due to this, hemogram was requested that did not show alterations in the red series or platelet line. With a slight increase in the leukocyte line (13,000) with neutrophils in (80.5%) Positive antigen for Sars-cov-2, which despite its scarce relationship between Covid-19 and pathologies of neuromuscular origin, became the main causal suspicion of symptoms clinical.

He also presented a positive PCR (203 mg/dl) Ionogram without hydroelectrolyte. alterations, thus ruling out hydroelectrolytic disorder as a cause of muscle weakness.

However, the patient persisted with bulbar involvement, with sudden onset of palpebral ptosis and diplopia, dysarthria and respiratory deterioration evidenced by respiratory distress with oxygen saturation below the reference ranges. Hemodynamic instability, manifested by the appearance of sustained supraventricular tachycardia with deterioration

So we proceed to perform orotracheal intubation and electrical cardioversion. pharmacological with satisfactory reversal and stabilization; Later, CPK, VDRL, HIV, Hepatitis B extension paraclinics, lumbar puncture with cytochemicals and culture of common germs of the cerebrospinal fluid and chest CT scan are requested.

Electromyography + neuroconduction velocity + H reflex + F wave of the 4 limbs was indicated, which revealed acute neurogenic involvement of a moderate degree, without current denervatory activity, with topography of lesion in the peripheral motor nerve trunk of 4 limbs, constituting a Axonomielinic polyneuropathy predominantly pure motor axonal with poor compensation for axonal collaterals. Findings suggestive of a pure motor axonal variant of Guillain-Barré Syndrome.

The chest tomography report shows multiple confluent opacities with a ground-glass pattern of parenchymal condensation, without mediastinal masses or adenopathies, typical findings of infection by COVID-19, CORADS 5.

Due to the above and given the results of the multiple studies carried out, immunomodulatory management with immunoglobulin 0.4/gr/k/day was started for 5 days and a request for a new Neuroconduction study after the start of treatment.

Patient who receives the first dose of immunomodulatory treatment, with respiratory deterioration present despite orotracheal intubation and management established, enters cardiorespiratory arrest who begins a sequence under the context of a non-shockable rhythm together with the application of 5 ampoules of adrenaline, not showing evidence of a favorable or successful response, leading the patient to death **(Table 1).**

Neuroconduction study

Neuroconduction of 4 limbs (upper and lower limbs).

Bilateral radio motor neuroconduction with preserved right velocity, with preserved latency and decreased amplitude and adequate morphology.

Bilateral ulnar motor neuroconduction with decreased right velocity, with preserved latencies and decreased amplitude, and adequate morphology.

Bilateral fibula motor neuroconduction with decreased left velocity, with prolonged left latency, decreased amplitudes, and adequate morphology.

Table 1 Paraclinicals.

RT-PCR-SARS-Cov 2	Positive
Blood count	Hemoglobin 14.4 g/dl Leukocytes: 12,600 Neutrophils: 80% Lymphocytes 15,400 Platelets 308,000
PCR:	203 mg/dl
VSG	20.0 mm/h
Procalcitonin	0.02 ng/ml
CK-CPK:	572 U/L
TPT	25.5 Seg
TP	21.7
Sodium	142.8 mmol/l
Potassium	4.09 mmol/l
Chlorine	110 mmol/l
BUN	11.8 mg/dl
Creatinine	0.9 mg/dl
Hepatitis C	Negative
Hepatitis B	Non-reactive
VIH	Negative
V.D.R.L	Non-reactive
Vitamin B12	448 pg/ml
Blood cultures	Negative at 12 hours
	Negative at l 24 hours
	Negative at 48 hours
	Negative at 72 hours
	Negative at 5 days

Table 2 Requested laboratories.

4 limb F waves	H reflexes of 2 upper extremities	H reflex of 2 lower limbs:
F waves through bilateral median nerve shows prolonged left latencies and absent right	H reflex through the bilateral median nerve on the absent flexor radial muscle	Triceps suralis stimulation was performed through the absent bilateral posterior tibial nerve.
F waves through the bilateral posterior tibial nerve show absence of responses.		

Bilateral posterior tibial motor neuroconduction with decreased left velocity, with preserved latencies, decreased left amplitude, and adequate morphology.

Sensitive

Sensory neuroconduction of the bilateral median nerve with preserved velocity, with adequate latencies, amplitude and morphology.

Sensory neuroconduction of the bilateral ulnar nerve with preserved velocity, with adequate latencies, amplitude and morphology.

Sensory neuroconduction of the bilateral sural nerve with preserved velocity, with adequate latencies, amplitude and morphology **(Table 2).**

Electromyography conclusion

The study reveals an acute neurogenic compromise of a moderate degree without current denervation activity, with topography of a lesion in the trunk of the peripheral motor nerve of 4 extremities, constituting an axonomyelinic polyneuropathy of pure motor axonal predominance with poor compensation for axonal collaterals. Findings suggestive of a pure motor axonal variant of Guillain Barre Syndrome.

Discussion

There are different infectious microorganisms including cytomegalovirus, Epstein-Barr virus, human immunodeficiency virus, Zika virus, among others, such as the MERS-CoV coronavirus, which have been related as triggers of guillain barre syndrome and myasthenia gravis, although all these were not ruled out in our patients, we collected solid evidence that demonstrated an association with SARS-CoV-2 infection, with which different reports have already postulated a post-infectious causal relationship [6].

In the reports of patients with a diagnosis of SARS-CoV-2 infection, it was evidenced that the patients were infected prior to the onset of guillain barre syndrome and myasthenia gravis and that this infection was probably also the cause of the respiratory symptoms present in them, In accordance with the vast majority of reported cases, our patient did not present evidence of SARS-CoV-2 infection in CSF samples, which removed the possibility of direct damage to the virus as a pathogenic mechanism and supported, together with the temporal profile of the symptoms a post-infectious mechanism probably autoimmune through the process of molecular mimicry [7,8].

Toscano et al. described 5 patients with guillain barré syndrome after the onset of the disease caused by the SARS-CoV-2 virus, in Italy, the symptoms appeared on day 5 and 10 of the disease. The CSF real-time polymerase chain reaction (PCR) test was negative for SARS-CoV-2. The patients were treated with intravenous immunoglobulin and only one also received plasmapheresis [9].

Restivo DA et al. state that the symptoms of myasthenia gravis appeared, in all cases, between 5 and 7 days after the onset of fever; the time that elapsed until the appearance of symptoms of myasthenia gravis coincides with the period referred to for other neurological disorders, associated with infections in patients with COVID-19, myasthenia gravis could be due to different mechanisms. Among those that stand out most is the fact that antibodies against SARS-CoV-2 proteins can cross-react with subunits of acetylcholine receptors, since the virus has antigenic determinants similar to the components of the neuromuscular junction, which again reinforces the aforementioned molecular mimic mechanism [10].

In conclusion, our report shows a rare case of guillain barre syndrome vs myasthenia gravis and SARS-CoV-2 infection, in which its characteristics such as the temporal relationship between symptoms, the absence of evidence of the presence of the virus in CSF and therefore direct damage to the nervous system, , support the hypothesis of a probable post-infectious immunological relationship that would be explained through the molecular mimicry process, in addition, according to what was observed in our case, the possibility is exposed, that even when

patients with SARS-CoV-2 infection, have been asymptomatic or have presented mild respiratory symptoms, they could develop post-infectious complications such as guillain barre syndrome and myasthenia gravis, However, both observations, although increasingly accepted, should continue to be evaluated in subsequent studies that include a larger number of patients, which is a limitation of this report.

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