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CASO CLÍNICO

Guillain-Barré Syndrome Underlying SARS-CoV-2 or Just a Confounding Factor?

Síndrome de Guillain-Barré Subjacente ao SARS-CoV-2 ou Simplesmente um Fator Confundidor?

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ABSTRACT

Guillain-Barré syndrome was frequently associated with a viral infection. Molecular mimicry is one of the mechanisms through which viruses can be involved in the etiology of this inflammatory demyelinating neuropathy. We describe a case of a 58-year-old man with a diagnosis of SARS-CoV-2 infection who developed clinical symptoms which are coincident with Guillain-Barré syndrome. SARS-CoV-2 could have been the trigger and even though the causative connection remains unclear, there is a probable association between SARS-CoV-2 and GBS. However, more studies are needed to support this cause-effect relationship.

RESUMO

A síndrome de Guillain-Barré é frequentemente associada a uma infeção viral. O mimetismo molecular é um dos mecanismos pelos quais os vírus podem estar envolvidos na etiologia desta neuropatia inflamatória desmielinizante. Descrevemos um caso de um homem de 58 anos com diagnóstico de infeção por SARS-CoV-2 que desenvolveu sintomas clínicos compatíveis com a síndrome de Guillain-Barré. O SARS-CoV-2 pode ter sido o *trigger* responsável e, embora a relação causal permaneça incerta, existe uma provável associação entre o SARS-CoV-2 e o GBS. No entanto, mais estudos são necessários para apoiar a relação causa-efeito.

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INTRODUCTION

The most common symptoms of SARS-CoV-2 infection are fever, cough, and shortness of breath and many other manifestations/complications can occur.[1] We present a case report in which a SARS-CoV-2 infected patient developed progressive and ascending weakness consistent with Guillain-Barré syndrome (GBS). This entity is typically a demyelinating neuropathy in which progressive weakness begins distally and ascends involving respiratory muscles and requiring mechanical ventilation in severe cases.[2] Although there is a gap of evidence about the type of complications associated with COVID-19, GBS has been identified in the context of SARS-CoV-2 infection.

CASE REPORT

A 58-year-old man with arterial hypertension, type 2 diabetes mellitus and obstructive sleep apnea, with a diagnosis of SARS-CoV-2 infection in the previous week presented to the emergency department complaining about weakness of the lower extremities and numbness of the arms since the day before.

He also complained of diarrhoea and anorexia which had started at the same time as respiratory symptoms, without fever or other symptoms.

On admission, he had no fever and he was hemodynamically stable. He had a normal PaO_2/FiO_2 ratio and a peripheral saturation of 97% on room air. Neurologic examination confirmed a flaccid and asymmetric tetraparesia, with absent deep tendon reflexes.

Laboratory findings showed an inflammatory pattern with a C-reactive protein of 7.97 mg/dL and an Interleukin 6 of

1172 pg/mL; chest X-ray showed an interstitial infiltrate and chest computed tomography (CT) with bilateral ground glass opacities and peripheral areas of lung consolidation.

The patient was initially admitted to a general COVID-19 ward.

Serology test results for Influenza virus, Epstein-Barr virus, cytomegalovirus, herpes simplex virus, varicella zoster virus, human immunodeficiency virus and hepatitis B and C and syphilis were all negative. In addition, the patient had negative blood cultures and no Campylobacter bacteria were found in his stool.

Lumbar puncture (LP) revealed a mild albumin-cytological dissociation. Cerebrospinal fluid (CSF) gram and culture and SARS-CoV-2 rtPCR were negative.

Intravenous immune globulin therapy (0.4 g/kg/day) was started and maintained for an 8-day course.

In the first day of hospitalization, the patient developed bulbar dysfunction with dyspnea, dysphagia and dysphonia, associated with dysautonomia with urinary retention. In addition, he developed respiratory failure requiring increasing oxygen supplementation. A trial of non-invasive ventilation was started with no improvement.

On the second day of hospitalization, he was transferred to our intensive care unit (ICU) and was intubated because of his significant neurological and respiratory worsening. Intubation was initially complicated due to hypotension in the context of dysautonomia, with the need for vasopressor support. It was decided not to introduce hydroxychloroquine because of its nonnegligible neuromyotoxicity.

Control mode ventilation was started assuring a protective lung ventilation, with tidal volumes of 6 mL/kg. Despite trials of assisted pressure ventilation, weaning was difficult and he underwent surgical tracheostomy 15 days after intubation.

Through ICU stay, respiratory and physical rehabilitation were part of the treatment plan to prevent atelectasis and muscle wasting.

Twenty days after hospital admission, the patient was discharged to an internal medicine ward with no need for supplementary oxygen therapy and progressive neurological improvement.

DISCUSSION

A viral infection is frequently identified in the days/weeks preceding GBS diagnosis, and molecular mimicry is one of the mechanisms through which viruses can be involved in the etiology of this inflammatory demyelinating neuropathy. In this case, SARS-CoV-2 could have been the trigger, even though SARS-COV2 rt-PCR assay in the CSF was negative.[3]

Critical illness neuropathy and myopathy should also be excluded. However, these entities usually appear later in the course of critical illness, do not show an upward clinical evolution, are not accompanied with dysautonomia and are not presented at the admission.[2]

Endotracheal intubation in GBS patients is frequently complicated due dysautonomia which can cause hypotension during airway manipulation as we observed in this patient.[4]

Although few cases have been reported about the association between neurological signs and SARS-CoV-2 infection, we should consider potential neurological symptoms as first symptoms or a complication of the infection and be aware of this possibility.[2,5] On the other hand, respiratory failure in severe GBS patients often requires prolonged respiratory support and ICU care. Even though the causative connection remains unclear, it is likely that there is an association between SARS-CoV-2 and GBS. However, more studies are needed to understand the cause-effect relationship.[1,5]

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