OPEN ACCESS Clinical Images and Medical Case Reports

ISSN 2766-7820

Case Report

Open Access, Volume 4

Case report: Delayed onset of Guillain-Barré syndrome following SARS-CoV-2 infection

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Received: Feb 01, 2023 Accepted: Feb 23, 2023 Published: Mar 02, 2023 Archived: www.jcimcr.org Copyright: © Hirani R (2023).

DOI: www.doi.org/10.52768/2766-7820/2308

Abstract

To date, little is known about the rare complications of SARS-CoV-2 infection. Here, we report a case of delayed onset of Guillain-Barre Syndrome (GBS) after such infection in a 40-year-old who was presented to the emergency department with fever and shortness of breath with no previous chronic condition. On a following visit, he was presented with bilateral lower extremity weakness. A final diagnosis of GBS was made after spinal fluid analysis. Medical providers should be aware of the potential for delayed presentation of Guillain Barré Syndrome on the scale of months following SARS-CoV-2 infection.

Keywords: Guillain-Barré syndrome; Coronavirus; COVID-19; SARS-CoV-2; Neuropathy.

Introduction

The outbreak of novel coronavirus (SARS-CoV-2) began in Wuhan, China in December 2019 and has spread to many countries, with the first documented case in the United States occurring in January 2020 [1]. While the virus is most notable for its respiratory effects, many neurologic manifestations have been reported [2]. Guillain-Barré syndrome (GBS) is a demyelinating disease that arises secondary to infection, with typical clinical manifestations of progressive, ascending symmetrical limb paralysis with areflexia occurring usually within days of infection. At the time of this report, a small but increasing number of cases of GBS have been reported in association with the SARS-CoV-2 infection, all with onset of symptoms within days to weeks [3-5]. Here, we present the case of a 40-year-old man who developed GBS over two months post-COVID-19 infection.

Case report

A 40-year-old man with no chronic medical conditions presented to the emergency department on 4/1/2020 with fever,

shortness of breath, and hypoxia. SARS-CoV-2 nasopharyngeal test was positive. His hospitalization was complicated by acute respiratory distress syndrome (ARDS) with chest roentgenogram demonstrating bilateral infiltrates and severe pneumonia requiring mechanical ventilation. He was treated with hydroxychloroguine, azithromycin, cefepime and tocilizumab. He was extubated on 4/13/20 and discharged on 4/30/20.

He returned to the emergency department on 6/16/2020 complaining of bilateral lower extremity weakness, left leg numbness, and left-hand paresthesia which developed over the prior week. He was afebrile with blood pressure 150/97, pulse 95 beats per minute, respiration rate 18, and oxygen saturation 97% on room air. Neurological examination revealed normal mental status, bilateral lower motor neuron facial weakness with Bell's phenomenon, 4/5 motor strength of upper extremities and 3/5 of lower extremities. Deep tendon reflexes (DTRs) were absent throughout and sensation was intact throughout. Laboratory examination including complete blood count, comprehensive metabolic panel, and urinalysis were within normal

Citation: Miller JA, Hirani R, More JM, Ober D, Etienne M. Case report: Delayed onset of Guillain-Barré syndrome following SARS-CoV-2 infection. J Clin Images Med Case Rep. 2023; 4(3): 2308.

limits. SARS-CoV-2 nasopharyngeal test was negative. Computed tomography of the head and MRI of brain, cervical and thoracic spine had no acute findings.

Over the next two days, he developed severe bifacial weakness and worsening weakness of all extremities, lower worse than upper extremities. He also had diminished sensation to light touch in his distal lower extremities. Spinal fluid analysis showed protein of 291 mg/dL and white count of 5/cu mm. A diagnosis of GBS was made and he was treated with intravenous immunoglobulin (IVIG) 400 mg/kg/day for a duration of five days. Ten days after initiation of IVIG treatment, his upper extremity motor function was documented at 5/5 and lower extremity 4+/5. DTRs remained absent at patella and Achilles but were 2/4 at brachioradialis.

Discussion

There are currently over 600 million confirmed cases of SARS-CoV-2 worldwide, which have resulted in over six million deaths [2]. The most commonly reported symptoms include fever, cough and difficulty breathing. However, neurologic symptoms of this disease are beginning to be elucidated [3,4]. Neurologic manifestations associated with other beta coronaviruses (such as SARS and MERS) include polyneuropathy, myopathy, stroke and GBS [7]. While there have been a handful of case reports on neurologic sequelae associated with COVID-19, post-viral complications require further investigation [5].

In this report, we discuss a 40-year-old man with a history of COVID-19 complicated by ARDS who developed GBS over two months after initial infection with SARS-CoV-2. In addition to being one of just a handful of GBS cases reported following CO-VID-19, this case is unique in the interval (~9 weeks) between infection and GBS development, as well as the presentation of bilateral facial palsy. Other cases of GBS post-COVID-19 have had intervals of 5-21 days between infection and the onset of GBS symptoms, while a few have presented para-infectiously [4-6]. Reviewing other case reports, about 50% of patients who developed GBS had severe respiratory failure requiring intubation. Most of the GBS cases post-COVID-19 have had the typical symptoms of GBS. However, one patient exhibited symptoms of dysautonomia, which is present in roughly 20% of GBS cases [9]. It is possible that the severity of our patient's COVID-19 course (month-long hospitalization requiring mechanical ventilation) could have influenced his unique GBS time course and presentation. Future studies are needed to clarify the mechanisms by which GBS develops after COVID-19 infection.

The association of post-infectious GBS and SARS-CoV-2 is becoming clearer. As such, neurologists and clinicians should be aware of this clinical presentation, especially given that the interval between infection and onset of GBS has varied in the literature currently available. Further investigation of neurologic sequelae is needed as more epidemiologic data becomes available.

Declarations

Conflict of interest: The authors have no conflicts of interest to declare

Financial disclosures: This case report was not funded.

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