Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

American Journal of Emergency Medicine 45 (2021) 154–155

Contents lists available at ScienceDirect

American Journal of Emergency Medicine

journal homepage: www.elsevier.com/locate/ajem

Guillain-Barré syndrome in a patient previously diagnosed with COVID-19

Alexandria C. Defabio, DO [a,b], Thomas R. Scott, BS [a,b], Robert T. Stenberg, MD [a,b], Erin L. Simon, DO [a,b,*]

[a] Cleveland Clinic Akron General, Department of Emergency Medicine, Akron, OH, United States of America
[b] Northeast Ohio Medical University, Rootstown, OH, United States of America

ARTICLE INFO

Article history:
Received 2 July 2020
Received in revised form 28 July 2020
Accepted 29 July 2020

ABSTRACT

As the COVID-19 pandemic continues to progress, the medical community is rapidly trying to identify complications and patterns of disease to improve patient outcomes. In a recent systematic review, it has been reported that isolated cases of Guillain-Barre Syndrome (GBS) have occurred secondary to COVID-19 infection. GBS is defined as a rare, but potentially fatal, immune mediated disease of peripheral nerves and nerve roots that is usually triggered by infections. The incidence of GBS can therefore increase during outbreaks of infectious diseases, as was seen during the Zika virus epidemics in 2013 in French Polynesia and 2015 in Latin America. While several cases of GBS secondary to COVID-19 infection have been reported in Italy, only one case has been reported in the United States (US). The reported case in the US was a 54- year old male. We present a case of GBS secondary to a COVID-19 infection and believe this to be the first documented female case in the US and the second documented case in the US overall. The presented case aims to supplement the existing body of knowledge and to assist clinicians in managing complications of COVID-19.

A 70-year-old female presented to the Emergency Department (ED) for chest pain, shortness of breath, difficulty voiding urine, and numbness in her arms and legs which made walking difficult. She described her chest discomfort as pressure, which did not radiate. The patient had an intrathecal bupivacaine pump implanted to manage reflex sympathetic dystrophy (RSD) and was worried the pump might be malfunctioning. Three months prior to her ED visit the patient had COVID-19 symptoms which included fever, shortness of breath, dry cough and a positive COVID-19 test result. The patient's past medical history was significant for RSD, fibromyalgia, gastroesophageal reflux disease (GERD), hiatal hernia, and asthma. She denied tobacco or alcohol use. Vital signs were blood pressure 133/77 mm/Hg, heart rate 92 beats per minute, temperature 97.5 °F orally, respirations 16 breaths per minute, SpO2 98% on room air. A bladder scan showed 740 mL of urine. Neurologic exam revealed 4 out of 5 strength in the lower extremities bilaterally, 2+ patellar reflexes, and no saddle paresthesia. While the patient's sensation was grossly intact, she reported decreased sensation especially in the lower extremities. The rest of her exam was normal. While in the ED, the patient had stool incontinence, and exam revealed decreased rectal tone. The patient's deep tendon reflexes were reassessed and had decreased to 0 Patellar and 0 Achilles reflexes. The biceps reflex was 1+ bilaterally. The patient's negative inspiratory force (NIF) was normal. The remainder of her physical examination was unremarkable.

Testing in the ED revealed a negative result for COVID-19, minimally increased cerebrospinal fluid (CSF) White Blood Cell (WBC) count (8/cmm), increased CSF glucose value (79 mg/dL), and an increased CSF protein value (127 mg/dL). She had a normal age adjusted D – Dimer value (510 ng/mL) and her Well's Score was low-risk. A rapid meningitis panel was negative. The following labs returned normal results: Myelin Basic Protein CSF, Herpes Simplex CSF, Lyme CSF Antibodies, CSF Venereal Disease Research Laboratory Test (VDRL), West Nile Virus Polymerase Chain Reaction (PCR) CSF, Enterovirus PCR, Cytomegalovirus CSF, Culture CSF, Human Immunodeficiency Virus (HIV) Screen, Heavy Metals Screen, Thyroid-Stimulating Hormone (TSH), Vitamin B12, Angiotensin-Converting Enzyme (ACE)/Angiotensin, C-Reactive Protein, High Sensitive Troponin T, Complete Blood Count (CBC).

Cervical, thoracic, and brain CT scans were unremarkable. X-rays of the chest, abdomen, and thoracic spine were also unremarkable. Neurology, neurosurgery and anesthesia were consulted. Anesthesia determined that the patient's bupivacaine pump was delivering inadequate doses although this was not felt to play a role in her symptoms.

Given the patient's presentation, physical exam, lab values, and imaging the differential diagnosis included local anesthetic systemic toxicity (LAST) syndrome, GBS secondary to COVID-19, and acute ascending demyelinating syndrome secondary to an unknown cause. Her pump was underdelivering medication, so LAST syndrome was ruled out.

* Corresponding author at: Department of Emergency Medicine, Cleveland Clinic Akron General, 1 Akron General Avenue, Akron, OH 44307, United States of America.
E-mail address: SimonE@ccf.org (E.L. Simon).

https://doi.org/10.1016/j.ajem.2020.07.074
0735-6757/

The patient was started on intravenous acyclovir; however, this was discontinued when it was determined that the patient was negative for both herpes simplex virus (HSV) and varicella zoster virus (VZV). The patient was admitted to the inpatient unit with neurology consultation and administered five rounds of intravenous immunoglobulin therapy (IVIG). At the time of discharge her lower extremity strength was 5/5 and her sensation had largely returned. Given that the patient responded to IVIG therapy, a diagnosis of GBS was presumed secondary to a previous COVID-19 infection. Literature suggests the novel coronavirus may have neurotrophic and neuroinvasive characteristics [1]. The mechanism of GBS in patients infected with COVID-19 has not yet been determined. Nine cases of GBS in patients with a history of COVID-19 have been recently reported in countries outside the United States. Of these, four patients exhibited the demyelinating form of GBS in both Germany and France. Symptoms of GBS in those patients occurred 1 to 3 weeks after the onset of COVID-19 symptoms [2,3]. All patients had fever and respiratory symptoms 5 to 10 days before the onset of neurological symptoms. The electrodiagnostic findings were consistent with an axonal variant of GBS in four of nine patients. Four other cases found the demyelinating subtype and in one patient, the pathophysiology was not clear [4-8].

COVID-19 stimulates inflammatory cells and produces various inflammatory cytokines and as a result, it creates immune-mediated processes [9]. GBS is an acute monophasic paralyzing illness that is recognized as a heterogenous syndrome with several variant forms [10]. It is thought that COVID-19 could evoke an immune response, which would in turn generate a humoral or T-cell independent antibody response [9,11]. The antibodies produced could then bind to structurally identical glycans present on nerve gangliosides, resulting in acute polyneuropathy with the immune response directed towards the myelin or axon of peripheral nerves [2]. Peripheral nerve remyelination is a natural physiological response; however, axonal regeneration is slow and can be irreversible if widespread [11]. It is important for emergency physicians to understand that GBS can occur weeks to months after COVID-19 infection, and that prompt recognition and treatment improves outcomes.

Prior presentations

None.

Funding sources/disclosures

None.

Author contribution statement

AD conceived the case report. AD and RS contributed to the medical management of the patient in the emergency department. TS and ELS drafted the manuscript, and all authors contributed substantially to its revision. AD takes responsibility for the paper as a whole.

Declaration of Competing Interest

None.

References

[1] Sahin AR. 2019 novel coronavirus (COVID-19) outbreak: a review of the current literature. Eurasian J Med Oncol. 2020. https://doi.org/10.14744/ejmo.2020.12220 Published online.
[2] Scheidl E, Canseco DD, Hadji-Naumov A, Bereznai B. Guillain-Barré syndrome during SARS-CoV-2 pandemic: a case report and review of recent literature. J Peripher Nerv Syst. 2020;25(2):204–7. https://doi.org/10.1111/jns.12382.
[3] Bigaut K, Mallaret M, Baloglu S, et al. Guillain-Barré syndrome related to SARS-CoV-2 infection. Neurol Neuroimmunol Neuroinflam. 2020;7(5):e785. https://doi.org/10.1212/NXI.0000000000000785.
[4] Toscano G, Palmerini F, Ravaglia S, 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.
[5] Sedaghat Z, Karimi N. Guillain Barre syndrome associated with COVID-19 infection: a case report. J Clin Neurosci Off J Neurosurg Soc Australas. 2020;76:233–5. https://doi.org/10.1016/j.jocn.2020.04.062.
[6] Camdessanche J-P, Morel J, Pozzetto B, Paul S, Tholance Y, Botelho-Nevers E. COVID-19 may induce Guillain–Barré syndrome. Rev Neurol (Paris). 2020;176(6):516–8. https://doi.org/10.1016/j.neurol.2020.04.003.
[7] Padroni M, Mastrangelo V, Asioli GM, et al. Guillain-Barré syndrome following COVID-19: new infection, old complication? J Neurol. 2020;267(7):1877–9. https://doi.org/10.1007/s00415-020-09849-6.
[8] Virani A, Rabold E, Hanson T, et al. Guillain-Barré Syndrome associated with SARS-CoV-2 infection. IDCases. 2020;20:e00771. https://doi.org/10.1016/j.idcr.2020.e00771.
[9] Huang C, Wang Y, Li X, 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.
[10] Leonhard SE, Mandarakas MR, Gondim FAA, et al. Diagnosis and management of Guillain–Barré syndrome in ten steps. Nat Rev Neurol. 2019;15(11):671–83. https://doi.org/10.1038/s41582-019-0250-9.
[11] Willison HJ, Jacobs BC, van Doorn PA. Guillain-Barré syndrome. Lancet. 2016;388(10045):717–27. https://doi.org/10.1016/S0140-6736(16)00339-1.