

Guillain Barre Syndrome & Covid-19: A Case Report

Mansour A^{1,2}, Ettouki O^{1,2}, Simou M^{1,2}, Nour M^{1,2}, Musoni L^{1,2}, Elkhaouri I^{1,2}, Darif A^{1,2}, Raid M^{1,2}, Hamza G^{1,2}, Ezzouine H^{1,2} and Charra B^{1,2*}

¹Department of Medical intensive care unit, Ibn Rochd University Hospital of Casablanca, Hassan II University of Casablanca, Morocco

²Faculty of Medicine and pharmacy, Hassan II University of Casablanca, Morocco

*Corresponding author:

Boubaker Charra,
Department of Medical Intensive Care Unit,
Ibn Rochd University Hospital of Casablanca,
Faculty of Medicine and pharmacy,
Hassan II University of Casablanca,
Morocco, E-mail: boubaker.ch68@gmail.com

Received: 20 Dec 2020

Accepted: 05 Jan 2021

Published: 09 Jan 2021

Copyright:

©2021 Charra B et al., This is an open access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and build upon your work non-commercially.

Citation:

Charra B. Guillain Barre Syndrome & Covid-19: A Case Report. *Ann Clin Med Case Rep.* 2021; V5(7): 1-3.

1. Abstract

Besides respiratory symptoms, coronavirus disease 2019 (COVID-19), like the severe acute respiratory syndrome coronavirus (SARS-CoV) and Middle East respiratory syndrome coronavirus (MERS-CoV), has neurological signs. Symptoms like myalgia, headaches, dizziness, anosmia, ageusia and disorder of consciousness confirms that the nervous system is involved in COVID-19 infection. Guillain barre syndrome (GBS) is a neurological disorder that usually follows a viral infection, it is possible that COVID-19 infection and GBS are closely related. In this case report, we try to elucidate the relation between SARS-CoV-2 and GBS.

2. Introduction

The first unexplained pneumonia cases occurred in Wuhan, China, and quickly spread to other countries [1], It was later revealed that these unexplained pneumonia cases had been caused by a new coronavirus. It has been stated that the symptoms of this new coronavirus infection are very similar to those of SARS-CoV which spread in 2003 [2].

COVID-19 patients have such clinical symptoms as headache, vomiting, nausea, dizziness, myalgia, anosmia, ageusia, and disorder of consciousness. These symptoms confirm that the nervous system is involved in the COVID-19 infection.

We present a case of a 72-year-old man who was initially diagnosed with COVID-19 pneumonia due to symptoms of fever and cough. 12 days later, he developed symmetric ascending quadriparesis and paresthesia. The diagnosis of Guillain barre syndrome (GBS) was made through cerebrospinal fluid analysis and he was successfully treated with intravenous immunoglobulin administration.

3. Case report

A 72-year-old man, without any history of medical pathologies, was admitted in the emergency unit for respiratory distress: symptoms were my-

algia, cough, headaches and fever. His vitals showed a respiratory rate at 25cycles per minute and blood saturation was 92% on room air, under the current epidemiological situation a chest CT scan showed ground glass opacities compatible with SARS-CoV-19 (Figure1) infection which was confirmed with nasopharyngeal secretions PCR.

The patient was then transferred to our unit for further care. Medical interview of the patient revealed Influenza illness and extreme asthenia 12 days before his admission without any digestive signs, medical examination showed a patient with no loss of consciousness or changes in mental status a Glasgow coma score of 15/15, systolic blood pressure= 112mmhg, diastolic blood pressure= 78mmhg, heart rate= 79bpm, Oxygen saturation= 95% with 5l of oxygen and a respiratory rate= 26cycles per minute. Neurological examination showed: abolished tendon reflexes in the four limbs, swallowing disturbance, diffuse areflexia, medical research council (MRC) strength evaluation was 1/5 in the legs, 2/5 in the arms, 3/5 in the forearms and 4/5 in the hands, Sensation to light touch and pinprick was decreased distally in addition of a decreased vibration sense in the lower limbs.

Laboratory findings include a white cell count of 3820 /mm³, lymphocytes= 1413 /mm³, blood platelets= 154 000 hemoglobin= 13.4 g/dL, elevated erythrocyte sedimentation rate of 25mm the 1st hour and 56mm 2nd hour, C-reactive protein= 26.9 mg/L.

Echocardiography was remarkable for a decreased left ventricular fraction of ejection estimated at 30% and global hypokinesia of the cardiac muscle.

CSF analysis revealed cytoalbuminologic dissociation with 115 mg/dL of proteins and 0 WBC.

Human immunodeficiency virus (HIV), Hepatitis virus B (HVB), Hepatitis virus C (HVC) serologies returned negative, SARS-CoV-2 serology

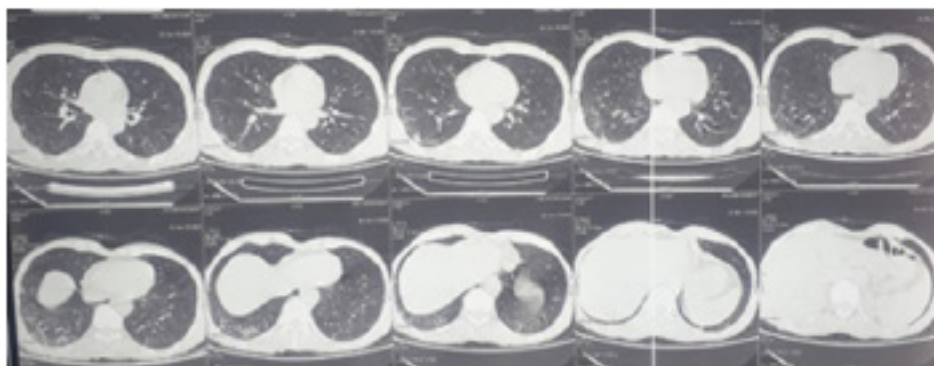


Figure 1: chest CT scan showing ground glass opacifications consistent of SARS-CoV-2 infection.

showed positive IgG and negative IgM antibodies and multiplex assay returned positive of SARS-CoV-2 and negative for 16 other viruses including adenovirus, parainfluenza virus 1, parainfluenza virus 2, parainfluenza virus 3, parainfluenza virus 4, respiratory syncytial virus, human rhinovirus/enterovirus and other coronaviruses.

The patient was diagnosed with GBS and started on intravenous immunoglobulin (IVIG) 0.4g/Kg/day for 5 days, associated with SARS-CoV-2 preconized treatment in Morocco: azithromycin 500mg three times daily for 7 days, Enoxaparin sodium 6000 IU twice a day, aspirin 100mg once daily, B6 B12 C D vitamins were also introduced (hydroxychloroquine wasn't introduced because of bad cardiac evaluation). After 8 days of therapy, the patient improved, recovered from COVID-19, his vitals were an oxygen saturation of 98% at room air, respiratory rate= 21cycles per minute, tendon reflexes in the four limbs were positive, swallowing reflex was present, MRC strength evaluation was 5/5 in the legs, 4/5 in the arms, 4/5 in the forearms and 5/5 in the hands and sensation was improved.

4. Discussion

SARS-CoV-2 frequently afflicts the respiratory system and gastrointestinal tracts. It shares its identity with other human coronaviruses including SARS-CoV and Middle East respiratory syndrome coronavirus. In this group of viruses, the respiratory system is commonly affected but they have also shown the involvement of the nervous system [3].

The case series by Mao et al in Wuhan, China, was one of the first studies that showed neurologic manifestations in patients with COVID-19. They concluded that patients with more severe COVID-19 illness were more likely to have neurologic symptoms. In contrast, our patient's respiratory status was relatively stable [4].

Increasing reports of neurologic manifestations of COVID-19 are emerging, but only a few cases of GBS associated with this virus have been established. GBS is an immune-mediated response, likely from a recent infection, where the immune system attacks the peripheral nerves due to a molecular mimicry phenomenon. This has preceded two-thirds of the times by an upper respiratory infection or gastroenteritis.

The studies showed that most patients with GBS due to the COVID-19 infection were elderly men. The studies have shown that most patients with GBS are mostly elderly men [5].

The literature shows there is variability in the presentation of COVID-19 and GBS. Our case had a typical course of viral symptoms preceding GBS findings. However, two other case reports identified concurrent respiratory

and neurologic symptoms [6,7].

Besides, the duration from onset of viral illness to neurologic manifestations have ranged from 5 to 24 days [8].

One of the most common neurological symptoms of GBS is acute muscle weakness. The pattern of muscle weakness may be helpful in the diagnosis of GBS. Weakness in the limbs and acute flaccid quadriplegia were observed in most GBS case reports after the diagnosis of COVID-19. Furthermore, demyelinating polyneuropathy was commonly observed in most of these reports. Some of the COVID-19-related GBS patients had the axonal variants of GBS [9].

In most of the patients in other case reports, similar to what happens in GBS, the protein levels in the CSF were elevated and the cell counts were normal [10].

IVIG is preferred over plasmapheresis for treating GBS due to fewer side effects. However, thrombotic events occur in 1–16.9%. All of the reported COVID-19 cases with GBS, including our case, received IVIG, and none of them reported thrombotic events.

5. Conclusion

There are several theories on how the virus attacks the nervous system. Studies postulate that the virus can infect a peripheral neuron, use an active retrograde transport mechanism across the synapse onto the cell body and reach the brain [11]. Other proposed mechanisms include direct damage through angiotensin converting enzyme-2ACE2 receptors, cytokine-related injury and hypoxia-related sequela [12].

Finally, more cases with epidemiological data should be studied and future investigations should be carried out in this regard. Due to the possible association of GBS and COVID-19, it is recommended that the patients be followed up by physicians with respect to neurological manifestations.

References

1. Li YC, Bai WZ, Hashikawa T. The neuroinvasive potential of SARS-CoV2 may play a role in the respiratory failure of COVID-19 patients. *J Med Virol.* 2020; 92(6): 552-555.
2. Zhu N, Zhang D, Wang W, Li X, Yang B, Song J, et al. A novel coronavirus from patients with pneumonia in China, 2019. *N Engl J Med.* 2020; 382(8): 727-733.
3. Montalvan V, Lee J, Bueso T, Toledo JD, Rivas K. Neurological manifestations of COVID-19 and other coronavirus infections: a systematic review. *Clin Neurol Neurosurg.* 2020; 194: 105921-7.

4. Mao L, Jin H, Wang M, Hu Y, Chen S, He Q, et al. Neurologic manifestations of hospitalized patients with coronavirus disease 2019 in Wuhan, China. *JAMA Neurol.* 2020; 77(6): 683-690.
5. Sejvar JJ, Baughman AL, Wise M, Morgan OW. Population incidence of Guillain-Barré syndrome: a systematic review and meta-analysis. *Neuroepidemiology.* 2011 ; 36(2): 123-133.
6. Alberti P, Beretta S, Piatti M, Karantzoulis A, Piatti ML, Santoro P, et al. Guillain-Barré syndrome related to COVID-19 infection. *Neurol Neuroimmunol & Neuroinflamm.* 2020; 7: e741.
7. Virani A, Rabold E, Hanson T, Haag A, Elrufay R, Cheema T, et al. Guillain-Barré syndrome associated with SARS- CoV-2 infection. *IDCases.* 2020; 20: e00771.
8. Camdessanche JP, Morel J, Pozzetto B, Paul S, Tholance Y, Nevers EB. COVID-19 may induce Guillain-Barré syndrome. *RevNeurol.* 2020; 176(6): 516-518.
9. Alzaidi MA, Nouri KA. Guillain-Barre syndrome. Pattern of muscle weakness. *Neurosciences (Riyadh).* 2002; 7(3): 176-8
10. Dimachkie MM, Barohn RJ. Guillain-Barré syndrome and variants. *Neurol Clin.* 2013; 31(2): 491-510.
11. Baig AM, Khaleeq A, Ali U, Syeda H. Evidence of the COVID-19 virus targeting the CNS: tissue distribution, Host-Virus interaction, and proposed neurotropic mechanisms. *ACS Chem Neurosci.* 2020; 11: 995-8.
12. Bridwell R, Long B, Gottlieb M. Neurologic complications of COVID-19. *Am J Emerg Med.* 2020; 38(7): 1549.e3-1549.e7