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Guillain-Barre Syndrome in a Child Infected with COVID-19

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Abstract

Guillain-Barre syndrome (GBS) characterizes a monophasic ascending, symmetrical paralysis, with areflexia, progressing over days to weeks. Normally, as a post-infectious autoimmune procedure, it leads to destroying myelin. Scattered reports exist regarding the concurrent evidence of COVID-19 infection and adults with possible GBS. However, few former cases were reported in children. Here in, we report an 11 years-old boy with GBS concurrent with COVID-19 infection.

Keywords: Guillain-Barre Syndrome, COVID-19, Children

1. Introduction

Guillain-Barre syndrome (GBS) is the most prevalent reason for acute severe weakness in children. Moreover, acute inflammatory demyelinating polyradiculoneuropathy (AIDP) is the most prevalent subgroup in the western world (1). Normally, AIDP is an autoimmune post-infectious process resulted from molecular mimicry of infectious agents to peripheral nerves. It leads to the destruction and inflammation of myelin. In most cases, a former infection can be recognized. Minor respiratory illness is the most prevalent infectious trigger; however, other viral infections, immunization, and gastrointestinal illnesses can be the proceeding process (2).

Meanwhile, from the beginning of the pandemic, it has been reported that COVID-19 can be presented with various symptoms, such as neurological ones presenting in over one-third of adult patients (3). The most prevalent neurological presentations include anosmia and ageusia; however, encephalitis, encephalopathy, acute disseminated encephalomyelitis, stroke, neuro-inflammatory, and autoimmune diseases have also been reported (4-6). Few reports exist on the concurrent evidence of COVID-19 infection (7, 8). Several reasons have been proposed to explain the milder symptoms of COVID-19 in children than in adults, including variations in children's and adults' immune systems and differences in the expression of ACE2 as a virus attachment receptor (9). Our case was one of the few cases in the pediatric age group.

2. Case Presentation

The patient was a formerly healthy 11-year old boy presenting to the emergency department with progressive weakness from the previous couple of days that started as bilateral lower extremity weakness. It proceeded to walk inability and paralysis. Over a few days, the weakness worsened and ascended to the upper extremity accompanying by disturbed speech and mild dysarthria. At first, he had urinary retention but no fecal or urinary incontinence. He had a head trauma three weeks ago without any complication. He denied any current illnesses within the previous two months such as upper respiratory infection, fever, shortness of breath, cough, rash, emesis, or diarrhea. Other family members had no past or current febrile or respiratory illness.

On physical examination, the patient was not feverish with a heart rate of 96 beats per minute, blood pressure of 120/80 mmHg, oxygen saturation of 98% in room air, and respiratory rate of 26 breaths per minute. He seemed to be attentive, conscious, and well-oriented without any sign of cranial nerve abnormalities, except for dysarthria. Initial neurological evaluation revealed symmetrical weakness influencing lower limb muscle groups with decreased motor power (1/5), hypotonia, and absent ankle and knee reflexes. In upper extremities, muscle strength was 2/5, and reflexes were absent, too. Sensation to light touch was intact, but the proprioception of the distal lower extremities was decreased.

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The patient rapidly developed poor gag reflex and nasal speech, so he was transferred to the PICU in favor of near respiratory failure. At the PICU, he was evaluated with a suspected diagnosis of GBS. The CBC showed WBC of 5400/mL, with absolute neutrophils of 3510/mL, Hb of 12 mg/mL, and Plt of 303000/mL, while hepatic function test, electrolytes, ferritin, and fibrinogen were normal. The CRP and ESR were 3+ and 63 mm/h, respectively. Blood, stool, and urine cultures, and urine toxicology were negative. Lumbar puncture was also performed that revealed albuminocytologic dissociation with RBCs of 0/mL, nucleated cells of 4/mL, and proteins of 122 mg/dL. Gram staining and culture were negative. The CSF real-time polymerase chain reaction of SARS-CoV-2 was negative. But, nucleic acid amplification of SARS-CoV-2 on nasopharyngeal specimens was positive. Besides, the SARS-CoV-2 IgG antibody of 0.8 was weakly positive in his serum.

Unfortunately, we could not perform serum multiplex PCR, including other infectious agents, due to limited resources. Electro-diagnostic testing was performed that demonstrated acute and symmetric motor polyneuropathy, mainly of axonal type in all limbs with moderate severity, while lower limbs involvement was more than upper limbs involvement, compatible with motor neuropathology.

Intravenous immunoglobulin (IVIG) started on the second day of admission. His exam revealed improvement over the following several days with upper extremity strength of 4/5 and lower extremity strength of 3/5. Physical therapy was also initiated. On the follow-up visit 15 days later, he continued to reveal slow improvement. He had recovered bilateral plantar flexion and dorsiflexion, the capability of independently sitting and walking on parallel bars with some help. Nasopharyngeal SARS-CoV-2-PCR was negative on the seventh day of admission.

3. Discussion

To our knowledge, this is the first report of a child with GBS concurrent with SARS-CoV-2 infection in Iran. The association between GBS and COVID-19, as a neurologic manifestation, has been reported in adults with various GBS variants such as demyelinating and axonal type, and also in association with other coronaviruses in children (6, 10). The exact mechanisms and nature of such phenomena require further investigations, but typical post-infectious processes have been postulated in the

literature (11-15). As SARS-CoV-2 and other coronaviruses, including MERS and SARS, have specifically neurotropic characteristics, they can be accompanied by diseases of the peripheral and central nervous system (16). Thus, molecular mimicry to peripheral nerves potentially can destroy myelin and inflammation through immunologic responses and present as GBS symptoms. The post-infectious mechanism for GBS is best manifested in the preceding infection setting with influenza, C. jejuni, and Zika virus, where symptoms initiate approximately one to two weeks after acute infection (17, 18).

This phenomenon was suspected in our patient due to the detected levels of SARS-CoV-2 IgG. Meanwhile, concurrent positive nasopharyngeal PCR can be the remnant of the previous infection, as well. Although clinical findings are invaluable, in many cases, they can provide more valuable supportive data than serological tests (19).

In summary, there is increasing evidence of the spectrum of neurological diseases associated with COVID-19, and our case is regarded to be one of the first pediatric patients representing the probable association between SARS-CoV-2 infection and GBS, which can be added to the growing evidence.

Footnotes

Authors' Contribution: Study concept and design: Romina Azadkiya; Analysis and interpretation of data: Romina Azadkiya; Drafting of the manuscript: Ghazal Shariatpanahi and Romina Azadkiya. Review and editing: Sedigheh Rafiei Tabatabaei, Ghazal Shariatpanahi, Romina Azadkiya, Parvaneh karimzadeh, and Abdollah Karimi. All authors read and approved the final manuscript.

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References

1. Asbury AK. New concepts of Guillain-Barre syndrome. *J Child Neurol*. 2000;**15**(3):183–91. doi: 10.1177/088307380001500308. [PubMed: 10757475].
2. Donofrio PD. Guillain-Barre Syndrome. *Continuum (Minneap Minn)*. 2017;**23**(5, Peripheral Nerve and Motor Neuron Disorders):1295–309. doi: 10.1212/CON.0000000000000513. [PubMed: 28968363].
3. 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):683–90. doi: 10.1001/jamaneurol.2020.1127. [PubMed: 32275288]. [PubMed Central: PMC7149362].
4. Koralnik IJ, Tyler KL. COVID-19: A global threat to the nervous system. *Ann Neurol*. 2020;**88**(1):1–11. doi: 10.1002/ana.25807. [PubMed: 32506549]. [PubMed Central: PMC7300753].
5. Vonck K, Garrez I, De Herdt V, Hemelsoet D, Laureys G, Raedt R, et al. Neurological manifestations and neuro-invasive mechanisms of the severe acute respiratory syndrome coronavirus type 2. *Eur J*

Neurol. 2020;**27**(8):1578–87. doi: 10.1111/ene.14329. [PubMed: 32416028]. [PubMed Central: PMC7276727].
6. Ellul MA, Benjamin L, Singh B, Lant S, Michael BD, Easton A, et al. Neurological associations of COVID-19. *The Lancet Neurology*. 2020;**19**(9):767–83. doi: 10.1016/s1474-4422(20)30221-0.
7. Alberti P, Beretta S, Piatti M, Karantzoulis A, Piatti ML, Santoro P, et al. Guillain-Barre syndrome related to COVID-19 infection. *Neurol Neuroimmunol Neuroinflamm*. 2020;**7**(4). doi: 10.1212/NXI.0000000000000741. [PubMed: 32350026]. [PubMed Central: PMC7217652].
8. Ottaviani D, Boso F, Tranquillini E, Gapeni I, Pedrotti G, Cozzio S, et al. Early Guillain-Barre syndrome in coronavirus disease 2019 (COVID-19): a case report from an Italian COVID-hospital. *Neurol Sci*. 2020;**41**(6):1351–4. doi: 10.1007/s10072-020-04449-8. [PubMed: 32399950]. [PubMed Central: PMC7216127].
9. Ohadian Moghadam S, Afshar D. A review on coronavirus disease 2019 (COVID-19) in pediatric patients. *Arch Pediatr Infect Dis*. 2020;**8**(3). doi: 10.5812/pedinfect.104225.
10. Turgay C, Emine T, Ozlem K, Muhammet SP, Haydar AT. A rare cause of acute flaccid paralysis: Human coronaviruses. *J Pediatr Neurosci*. 2015;**10**(3):280–1. doi: 10.4103/1817-1745.165716. [PubMed: 26557177]. [PubMed Central: PMC4611905].
11. Toscano G, Palmerini F, Ravaglia S, Ruiz L, Invernizzi P, Cuzzoni MG, et al. Guillain-Barre syndrome associated with SARS-CoV-2. *N Engl J Med*. 2020;**382**(26):2574–6. doi: 10.1056/NEJMc2009191. [PubMed: 32302082]. [PubMed Central: PMC7182017].
12. Centers for Disease Control and Prevention. *Multisystem inflammatory syndrome in children(MIS-C) associated with coronavirus disease 2019(COVID-19)*. CDC; 2020. Available from: https://emergencycdcgov/han/2020/han00432.
13. Corman VM, Landt O, Kaiser M, Molenkamp R, Meijer A, Chu DK, et al. Detection of 2019 novel coronavirus (2019-nCoV) by real-time RT-PCR. *Euro Surveill*. 2020;**25**(3). doi: 10.2807/1560-7917.ES.2020.25.3.2000045. [PubMed: 31992387]. [PubMed Central: PMC6988269].
14. Sedaghat Z, Karimi N. Guillain Barre syndrome associated with COVID-19 infection: A case report. *J Clin Neurosci*. 2020;**76**:233–5. doi: 10.1016/j.jocn.2020.04.062. [PubMed: 32312628]. [PubMed Central: PMC7158817].
15. Zhao H, Shen D, Zhou H, Liu J, Chen S. Guillain-Barre syndrome associated with SARS-CoV-2 infection: causality or coincidence? *Lancet Neurol*. 2020;**19**(5):383–4. doi: 10.1016/S1474-4422(20)30109-5. [PubMed: 32246917]. [PubMed Central: PMC7176927].
16. 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):552–5. doi: 10.1002/jmv.25728. [PubMed: 32104915]. [PubMed Central: PMC7228394].
17. Cao-Lormeau VM, Blake A, Mons S, Lastere S, Roche C, Vanhomwegen J, et al. Guillain-Barre Syndrome outbreak associated with Zika virus infection in French Polynesia: a case-control study. *Lancet*. 2016;**387**(10027):1531–9. doi: 10.1016/S0140-6736(16)00562-6. [PubMed: 26948433]. [PubMed Central: PMC5444521].
18. Rees JH, Soudain SE, Gregson NA, Hughes RA. Campylobacter jejuni infection and Guillain-Barre syndrome. *N Engl J Med*. 1995;**333**(21):1374–9. doi: 10.1056/NEJM199511233332102. [PubMed: 7477117].
19. Shirvani A, Azimi L, Mansour Ghanaie R, Alebouyeh M, Fallah F, Rafiei Tabatabaei S, et al. Utility of available methods for diagnosing SARS-CoV-2 in clinical samples. *Arch Pediatr Infect Dis*. 2020;**8**(3). en. e103677. doi: 10.5812/pedinfect.103677.