

# Guillain-Barré Syndrome in a Pediatric Patient with COVID-19: A Case Report and Review of Literature

Bernadette B. Terencio, MD,<sup>1,2</sup> Rachele F. Patiño, MD<sup>2</sup> and Roland Dominic G. Jamora, MD<sup>3,4</sup>

<sup>1</sup>Neuroscience Department, College of Medicine, San Beda University, Manila, Philippines

<sup>2</sup>Section of Pediatric Neurology, Department of Neurosciences, Asian Brain Institute, Asian Hospital and Medical Center, Muntinlupa, Philippines

<sup>3</sup>Department of Neurosciences, College of Medicine and Philippine General Hospital, University of the Philippines Manila, Philippines

<sup>4</sup>Institute for Neurosciences, St. Luke's Medical Center – Quezon City and Global City, Philippines

## ABSTRACT

The novel coronavirus disease 2019 (COVID-19) has created a global health impact to millions of people. There have been studies of COVID-19 patients manifesting with neurologic symptoms. Although the number of adult COVID-19 infections diagnosed with Guillain-Barré Syndrome (GBS) is increasing, the occurrence of cases in pediatric population remains limited or perhaps underreported. We report a rare case of an asymptomatic COVID-19 infection manifesting as acute progressive ascending polyneuropathy and hyporeflexia in a 16-year-old teen. The diagnosis of COVID-19 infection was confirmed by reverse transcription polymerase chain reaction for SARS-CoV-2 of oropharyngeal and nasopharyngeal swab specimens. Magnetic resonance imaging of the spine revealed abnormal enhancement of the cauda equina, including the dorsal and ventral roots. Electromyography and nerve conduction studies were compatible with an acute inflammatory demyelinating polyneuropathy subtype of GBS. Although lumbar puncture was not done, the clinical findings and electrodiagnostic tests were both consistent with GBS. The patient had improvement of both motor and sensory functions after completing the treatment of intravenous immunoglobulins. Neurologic manifestations of systemic illness especially in children during this time of pandemic warrants scrutiny, as these may mask a potentially dangerous and infectious ongoing COVID infection.

**Keywords:** COVID-19, Guillain-Barré Syndrome, polyneuropathies, pediatrics, SARS-CoV-2

## INTRODUCTION

The coronavirus disease 2019 (COVID-19) outbreak from Wuhan, China in December 2019 has rapidly spread to around 200 countries in the world affecting 190 million people with more than 4 million fatalities as of July 24, 2021.<sup>1</sup> What was initially thought to be a respiratory disease has evolved into a multi-organ disease, affecting even the nervous system.<sup>2</sup> Evidence shows that COVID-19 has a neuro-invasive potential; however, there is still paucity of information regarding its neurologic complications. In a retrospective study, it was reported that about 36.4% of adult patients with COVID-19 manifested with neurologic symptoms involving the central and peripheral nervous systems.<sup>3</sup>

Guillain-Barré syndrome (GBS) is an acute, symmetrical, ascending paralysis that can also cause sensory symptoms. It is associated with antecedent infections, which are thought to activate the immunological response that leads to polyneuropathy.<sup>4</sup> Approximately two-thirds of cases have previously been infected with viruses such as *Campylobacter jejuni*, cytomegalovirus and Epstein-Barr virus. In a systematic review of GBS associated with COVID-19, 69 adult and 4 pediatric cases have been

Corresponding author: Roland Dominic G. Jamora, MD  
Department of Neurosciences  
College of Medicine and Philippine General Hospital  
University of the Philippines Manila  
Taft Avenue, Ermita, Manila 1000, Philippines  
Email: rgjamora@up.edu.ph

reported.<sup>5</sup> The association between COVID-19 and GBS remains unknown and there have been less reports among the pediatric population. After obtaining consent from the patient's parents, we report a pediatric case presenting with acute ascending polyneuropathy associated with an asymptomatic COVID-19 infection. At present, this is the first and only report on pediatric GBS patient with associated COVID-19 infection in the Philippines. We also reviewed the current literature on COVID-19 associated GBS.

## CASE PRESENTATION

A 16-year-old, right-handed teen presented at the emergency department (ED) with a seven-day history of progressive symmetric ascending quadriparesis. Nine days prior to admission, patient complained of bilateral thigh and lower back pain, non-radiating, cramping in character, graded 4/10, which was spontaneously resolved with rest. Seven days prior to admission, there was a sudden onset of weakness on both lower extremities. Six days prior to admission, persistence of the symptoms prompted consult wherein serum sodium and potassium levels, and x-ray of the back and thigh showed unremarkable results. The patient was sent home with vitamins and analgesics. Three days prior to admission, symptoms were now accompanied by difficulty in ambulation. In the interim, the weakness progressed to the upper extremities with associated tingling sensation, which then prompted to seek consult. The patient denied any recent illness including fever, cough, colds, vomiting, diarrhea, shortness of breath, rash or blurring of vision. The patient did not have urinary or fecal incontinence, but she did not have any bowel movement for seven days. There were no known comorbidities, surgeries nor previous hospitalizations. She denied any recent travel or immunizations. The other family members had no history of any recent febrile illness, respiratory or gastrointestinal symptoms. The patient had been interacting with neighbors and had been visiting different households within the community; however, close contact with a confirmed case of COVID-19 infection cannot be established. The patient was also known to be non-compliant with basic health and community quarantine protocols.

At the time of examination at the ED, the patient arrived conscious, alert, oriented and not in cardiorespiratory distress. The blood pressure was 140/90 mmHg, heart rate at 90 beats per minute, respiratory rate at 18 breaths per minute, oxygen saturation at 99% at room air, and temperature at 36.6°C. She weighed 47 kg with a height of 150 cm and body mass index of 20.8, which was appropriate for her age. The systemic physical examination was normal. Digital rectal examination was not done. The cranial nerves were intact. The motor examination revealed hypotonia, weakness on all extremities with a motor strength of 3/5 for both proximal and distal upper extremities and 2/5 for both proximal and distal lower extremities. There was generalized

hyporeflexia and no extensor toe sign. Sensory examination revealed decreased light touch sensation in the right hand. There were no meningeal signs. The patient was wheelchair-bound, giving a GBS disability scale score of 4.

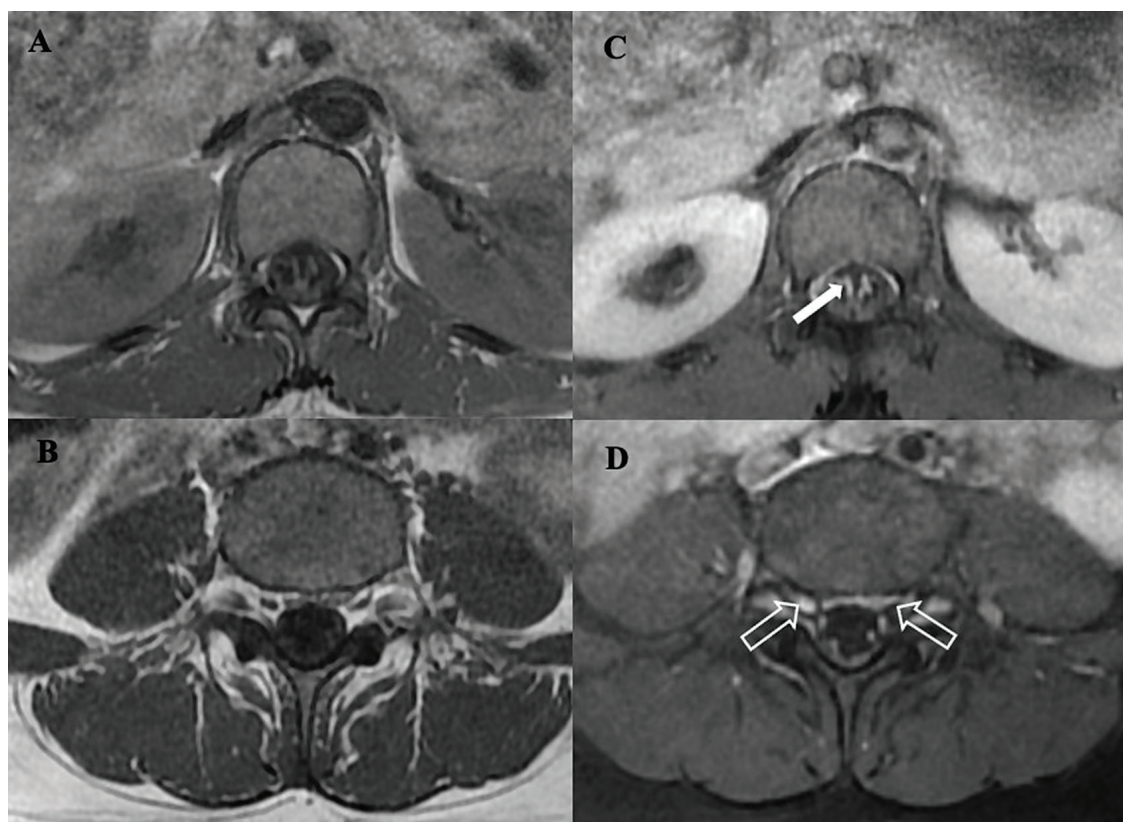
Initial blood test results such as complete blood count and blood chemistries were all normal. Given the current pandemic, COVID-19 infection and GBS were considered. The nasopharyngeal and oropharyngeal swab reverse transcription polymerase chain reaction (RT-PCR) test for SARS-CoV-2 was positive. The electrocardiogram and chest X-ray were normal. A lumbar tap was not done as the family did not consent. The thoraco-lumbosacral magnetic resonance imaging (MRI) revealed contrast enhancement of the cauda equina including the dorsal and ventral roots and thickening and contrast enhancement of the exiting nerves in the thoracic and lumbar spine (Figure 1). The patient developed urinary incontinence and constipation on the 9<sup>th</sup> day of illness.

Electrodiagnostic tests such as nerve conduction studies (NCS) and electromyography (EMG) was done ten days after onset of lower extremity weakness. The NCS revealed absence of compound muscle action potential of the motor nerves in both upper and lower extremities (Table 1A, Figure 2A-C). The median sensory nerve response was absent (Figure 2D). The sural and ulnar sensory nerve responses have normal latencies, sensory nerve action potential amplitudes, and conduction velocities (Figure 2E-G). The F-waves were absent on both upper and lower extremities (Figure 2F-I). On needle EMG examination (Table 1B), there was normal resting activity but with absence of spontaneous activity was seen in the first dorsalis interosseous and vastus lateralis muscles. The tibialis anterior and deltoid

**Table 1A.** Motor and sensory nerve conduction studies

	Amplitude (n ≥ 6)	Latencies (n < 3)	Conduction velocity (m/sec) (n ≤ 50)
<b>Motor nerve conduction</b>			
<b>Right median nerve</b>			
Wrist	NR	NR	NR
Elbow	NR	NR	NR
<b>Right ulnar nerve</b>			
Wrist	NR	NR	NR
Elbow	NR	NR	NR
<b>Right tibial nerve</b>			
Ankle	NR	NR	NR
Popliteal	NR	NR	NR
<b>Sensory nerve conduction</b>			
<b>Right median nerve</b>			
Wrist	NR	NR	NR
<b>Right ulnar nerve</b>			
Wrist	35.5µV	1.6	83
<b>Right sural nerve</b>			
Calf	55.6µV	1.6	88
<b>Left sural nerve</b>			
Calf	55.2µV	2.0	70

NR: no response



**Figure 1.** Lumbar magnetic resonance imaging (MRI). (A and B) Pre-contrast axial T1-weighted MRI demonstrated no abnormality of the cauda equina and nerve roots. (C and D) Post-contrast axial T1-weighted MRI showed contrast enhancement of the cauda equina (solid arrows) and nerve roots (open arrows).

**Table 1B.** Electromyography examination

Muscle	Rest activity				Voluntary activity			
	Insertional Activity	Fibrillation	Positive sharp waves	Fasciculation	Duration	Amplitude	Polyphasic	Recruitment
<i>Deltoid</i>	Normal	0	0	0	Normal	0	+1	Reduced
<i>Right 1<sup>st</sup> dorsal interosseus</i>	Normal	0	0	0	No activity	0	0	No activity
<i>Right vastus lateralis</i>	Normal	0	0	0	No activity	0	0	No activity
<i>Right tibialis anterior</i>	Normal	0	0	0	Normal	0	+1	Reduced
<i>Right paraspinal muscle T10-T11</i>	Normal	0	0	0	Normal	Full	0	Full

showed single motor unit potentials. The electrodiagnostic testing done showed evidence of diffuse sensory and motor demyelinating polyneuropathy, which is compatible with acute inflammatory demyelinating polyneuropathy (AIDP) variant of GBS.

Intravenous immunoglobulin (IVIg) infusion at 1g/kg/day was given for two days with no untoward adverse reactions. Vitamin D 2,000IU/day and zinc sulfate 20mg elemental zinc twice a day were started as nutritional support for children with COVID-19 infection. After IVIg infusion, there was a significant improvement of the motor strength (4/5 for both upper extremities and 3/5 for both

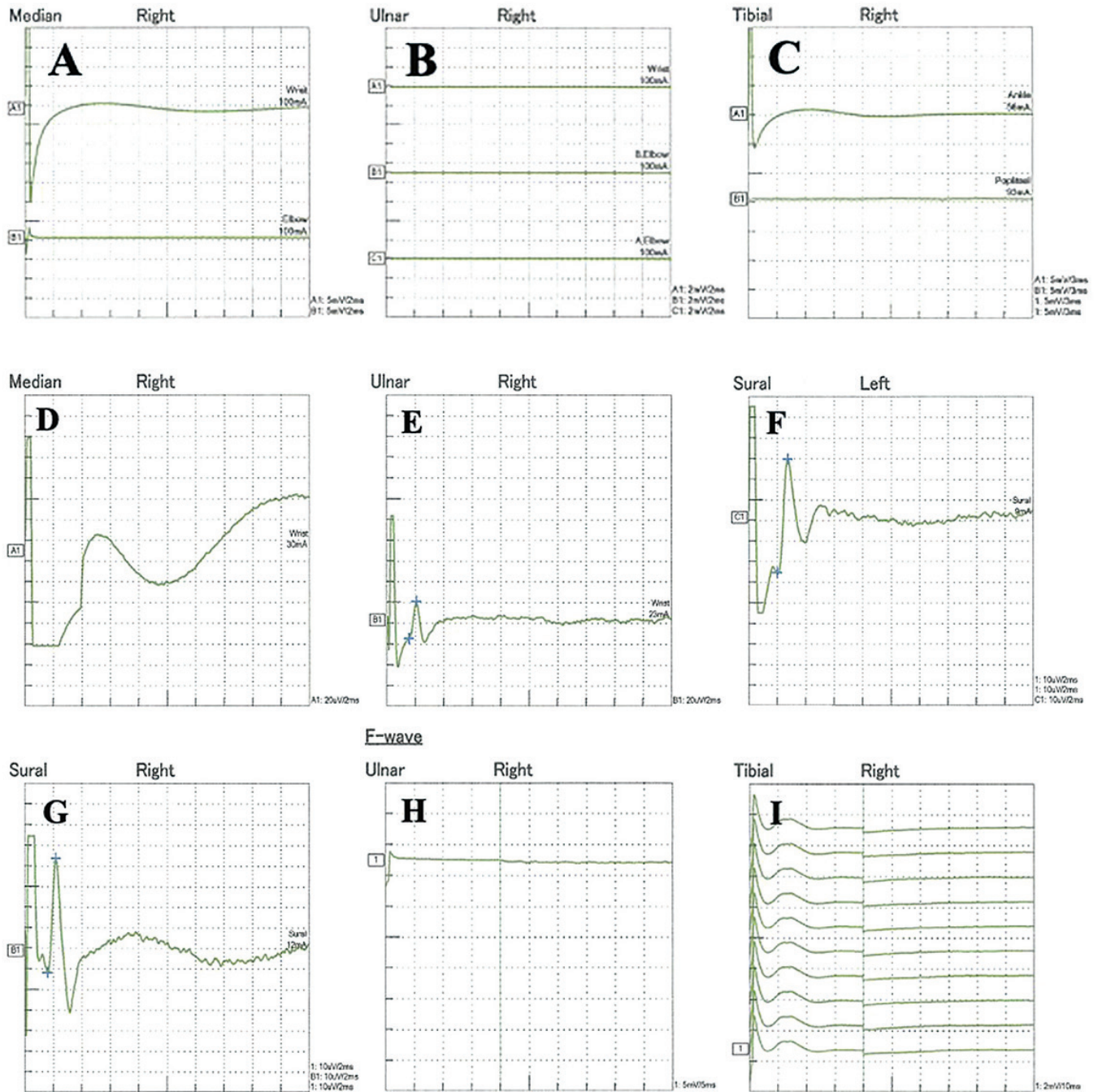
lower extremities). The deep tendon reflexes on the upper extremities was +2, but still hyporeflexive (+1) on both lower extremities. She regained the ability to sit with assistance and move her extremities independently, but still with noted heaviness. The patient's motor strength was reported to have clinical improvement with a GBS disability score of 3 (able to walk 10 meters across an open space with help) on discharge five days after IVIg infusion. Physiotherapy was continued at home. Follow-up was delayed and only done through teleconsultation. Two months after discharge, she had a steppage gait but was able to walk independently. There was no difficulty or limitation in daily activities.

**Table 2.** Summary of clinical findings, results of diagnostic investigations, and outcome in 8 pediatric GBS associated with COVID-19

Authors	Age/ Sex	Country	COVID-19 clinical presentation	GBS clinical presentation		GBS diagnostics	GBS subtype	Treatment	Days of hospital stay	Outcome
				Onset	Disease course					
<i>Akçay, et al.</i> <sup>6</sup>	6/M	Turkey	Fever; contact with a COVID-19 positive 1 week prior to onset of symptoms	Symmetric ascending paralysis	Ascending bilateral extremity flaccid weakness, areflexia, neck flexor and extensor muscle weakness, severe respiratory muscle weakness	Electrophysiology CSF analysis: albumin-cytologic dissociation Spinal MRI: enhancement of cauda equina and nerve roots	Acute motor axonal neuropathy	Plasma exchange (10 sessions) 5% albumin replacement (4 sessions) Methylprednisolone (30 mg/kg/day) for 5 days IVIg 2 g/kg/day (2 cycles, repeated 14 days after)	60 days	Weakness of neck flexor and extensor muscles, still with weakness on both upper and lower extremities; on home-ventilation
<i>Curtis, et al.</i> <sup>7</sup>	8/M	Indiana, USA	Cough, Increased secretions with poor clearance	Bilateral lower extremity weakness and difficulty ambulation	Ascending weakness with involvement of upper extremities, dyspnea, difficulty voiding	Electrophysiology CSF analysis: albumin-cytologic dissociation Spinal MRI: enhancement of the posterior nerve roots from the T11 level through the cauda equina	Acute inflammatory demyelinating polyneuropathy	IVIg 1 g/kg/day (2 days) Mechanical invasive ventilation	Not mentioned	Slow improvement of weakness on both upper and lower extremities; extubated 3 days after IVIg
<i>Tabatabaei, et al.</i> <sup>8</sup>	11/M	Iran	Asymptomatic	Bilateral lower extremity weakness	Ascending weakness with involvement of upper extremities, disturbed speech and mild dysarthria, areflexia, poor gag reflex and nasal speech	Electrophysiology CSF analysis: albumin-cytologic dissociation, negative for SARS-CoV-2 Spinal MRI: not done	Acute motor axonal neuropathy	IVIg (dose not mentioned)	Not mentioned	Improvement of weakness on both upper and lower extremities
<i>Khalifa et al.</i> <sup>9</sup>	11/M	Kingdom of Saudi Arabia	Acute upper respiratory tract infection, low-grade fever, dry cough	Gait ataxia, tingling sensation in the lower extremities	Symmetrical lower extremities weakness and paresthesia, hypotonia, ankle and knee areflexia, impaired pain and light touch sensation on lower extremities with impaired proprioception, morbilliform skin rash on the palmar aspect of both hands	Electrophysiology CSF analysis: albumin-cytologic dissociation Cranial MRI: normal Spinal MRI: enhancement of the cauda equina nerve roots	Acute inflammatory demyelinating polyneuropathy	IVIg 1 g/kg/day (2 days)	15 days	Improved power on lower extremities, balanced gait, decreased numbness and normal proprioception, electrophysiological improvement
<i>Manji et al.</i> <sup>10</sup>	12/M	Tanzania	Low grade fever and dry cough	Lower back pain, lower extremities weakness	Rapidly progressive ascending quadriparesis, areflexia, bilateral facial paresis, altered level of consciousness and signs of shock	Electrophysiology: not done CSF analysis: not done MRI: not done	-	IVIg 400 mg/kg/day (5 days) Mechanical invasive ventilation	6 days	Improvement of upper extremities weakness, improvement of lower extremities power and tone; rapidly decompensated after self-extubation and died
<i>Paybast et al.</i> <sup>11</sup>	14/F	Iran	Symptoms of upper respiratory tract infection	Mild weakness on lower extremities	Ascending quadriparesis, hypoactive deep tendon reflexes in upper extremities and absent in lower extremities, decreased light touch, position and vibration sensation in all distal extremities up to ankle and elbow joints, gait ataxia	Electrophysiology: not done CSF analysis: albumin-cytologic dissociation MRI: not done	-	IVIg 20 g daily (5 days)	Not mentioned	Recovery of symptoms except for generalized hyporeflexia and decreased light touch sensation
<i>Frank et al.</i> <sup>12</sup>	15/M	Brazil	Fever, intense sweating, frontal headache, retro-orbital pain	Pain and weakness in the lower extremities	Progressive symmetrical upper and lower extremities weakness, areflexia, no sensory loss, normal plantar response	Electrophysiology Two CSF analysis (2 weeks apart), both showing normal cell count and CSF biochemistry Cranial and spinal MRI: normal	Acute motor axonal neuropathy	IVIg 400 mg/kg/day (5 days)	15 days	Minimal improvement of upper and lower extremity weakness
<i>Meshref, et al.</i> <sup>13</sup>	18/F	Egypt	Asymptomatic; contact with a COVID-19 positive 1 week prior to onset of symptoms	Difficulty ambulation with bilateral weakness on lower extremities	Ascending quadriparesis, difficulty swallowing, nasal tone of speech with facial diplegia	Electrophysiology CSF analysis: refused Cranial and Cervical MRI: normal Spinal MRI: not done	Acute motor axonal neuropathy	IVIg 400 mg/kg/day (5 days); prophylactic anticoagulant	Not mentioned	Improvement in weakness, difficulty of swallowing and nasal tone of speech

IVIg, Intravenous immunoglobulin; CSF, Cerebrospinal fluid; MRI, Magnetic resonance imaging; GBS, Guillain-Barré Syndrome





**Figure 2.** Electromyography and nerve conduction studies of the patient. (A to C) Motor responses of the right median, ulnar and tibial were absent. (D) The median sensory nerve responses were absent. (E to G) The ulnar and sural nerve responses have normal sensory nerve action potential amplitudes and conduction velocities. (F to I) The F-wave latencies were absent in the sural, ulnar and tibial nerves.

## DISCUSSION

GBS is an acute, immune-mediated peripheral polyradiculoneuropathy generally manifests as a symmetric motor paralysis with or without involvement of the sensory and autonomic nerves.<sup>4</sup> An overall worldwide incidence rate of GBS has been reported to be 1–2 per 100,000 per year in children under 15 years old.<sup>14</sup> Neurologic symptoms typically arises a few days to four weeks after bacterial or viral infections including influenza, *Campylobacter jejuni*, *Haemophilus influenzae*, *Mycoplasma pneumoniae*, Epstein-Barr virus, cytomegalovirus, and more recently, Zika virus.<sup>4</sup>

The exact mechanism of GBS in relation to COVID-19 has not yet been established. However, molecular mimicry and subsequent antibody cross reactivity between structural components of both pathogens and myelin sheath of peripheral nerves is the most plausible proposed hypothesis. This immune response can be directed towards the myelin or the axon of peripheral nerve leading to weakness on the extremities which progresses upward.<sup>15</sup>

To date, adult cases of GBS associated with COVID-19 are seemingly increasing. A systematic review showed that patients with COVID-19 related GBS have the same manifestations with the classic post-infectious GBS and possibly with similar immune-mediated pathogenic mechanisms.<sup>3</sup> We identified only eight studies of COVID-19 related GBS in children who initially presented with acute progressive weakness.<sup>6-13</sup> Four cases presented with upper respiratory tract symptoms preceding GBS onset.<sup>7,9-11</sup> On the other hand, two cases manifested with fever prior to the onset of weakness<sup>6,13</sup> (Table 2). Two patients, including our patient were asymptomatic.<sup>8,13</sup> In Uganda, a study revealed that patients with SARS-CoV-2 infection can experience GBS with or without COVID-19 symptoms.<sup>16</sup>

The interval between the onset of symptoms of COVID-19 and the first manifestation of GBS ranges from one to four weeks.<sup>3</sup> In some cases, the onset of neurologic symptoms begin during the course of viral infection; thus, GBS is considered to be a para-infectious sequelae.<sup>6,7,17</sup> The pulmonary epithelial cell binding of severe acute respiratory syndrome coronavirus (SARS-CoV-2) induces a systemic inflammatory response that produces elevated cytokine levels, stimulate glial cells and hence create a pro-inflammatory state in the central nervous system. The expression of angiotensin-converting enzyme 2 (ACE2) on endothelial cells of the blood brain barrier allows viral invasion into the central nervous system.<sup>18</sup> The coronavirus mainly invades the respiratory epithelial cells of the lungs through an interaction between the viral plasma glycoprotein (protein S) and the ACE2 present on the host cell. This ACE2 is not only found in respiratory systems but also acts on the cardiovascular, renal, gastrointestinal, and cerebral systems.<sup>2,18</sup>

The incidence of SARS-CoV-2 infections is lower in children compared to adults. There is growing evidence that many COVID-19 infections are asymptomatic. More than

20% of the patients with COVID-19 were asymptomatic, majority of whom belong to the younger age group.<sup>19</sup> Another review reported that pediatric SARS-CoV-2 infections were asymptomatic and estimated to be between 16–45%.<sup>20,21</sup> It has been linked to the mild immune response and low level of ACE2 of children, resulting to absence of any clinical manifestations.<sup>19</sup> There is also limited research suggesting that pediatric patients play a part in transmission of the disease.<sup>22-24</sup> The viral load and infectivity of an asymptomatic carrier is close to that of symptomatic patients, which shows the potential transmission of asymptomatic patients.<sup>25,26</sup> Considering the high transmission rate in the Philippines, it is most likely that our patient was an asymptomatic carrier.<sup>1</sup>

The immunopathogenesis, clinical course, and therapeutic response differ between these two main categories of GBS – acute inflammatory demyelinating polyneuropathy (AIDP) and acute motor axonal neuropathy (AMAN). In our study, four out of eight patients had EMG-NCS results compatible with AMAN, two with AIDP and two did not perform any testing. All reported pediatric patients with GBS had a favorable response to treatment with IVIg except for one. This patient had rapidly progressive ascending quadriparesis, areflexia, bilateral paresis, altered level of consciousness and signs of shock for which a mechanical ventilation was warranted. Unfortunately, there was no electrophysiologic testing done due to rapid onset of clinical presentation. IVIg was given and resulted in improvement in muscle tone; however, this deteriorated after self-extubation. The severity of the disease is correlated with the outcome. According to Abu et al., patients with clinical history and/or radiologic findings of COVID-19 pneumonia are likely had to have poor outcome or no improvement.<sup>5</sup>

The primary mode of treatment for GBS is IVIg or therapeutic plasma exchange.<sup>27</sup> In children, IVIg is more preferred than plasma exchange due to greater convenience and availability.<sup>28</sup> The standard dose for GBS therapy in adult and children is 2g/kg divided in 2 to 5 days. A randomized study by Korinthenberg et al. revealed that treatment given with 1g/kg for two days had no significant difference in the effectiveness as compared to 400mg/kg for five days. Although, early and greater risk of relapse have occurred more with the two-day course than with the five-day course.<sup>29</sup> Our patient had motor improvement after completion of the two-day regimen of IVIG. Nevertheless, complete recovery still warrants regular physical therapy and exercise.

The patient's clinical presentation, together with the EMG-NCS findings were diagnostic of GBS. The lack of concurrent COVID-19 surveillance data makes it challenging to estimate the incidence of GBS related with COVID-19.

## CONCLUSION

We present a case of GBS that occurred in a pediatric patient with asymptomatic COVID-19. A high index of suspicion for GBS and COVID-19 infection is warranted

for early diagnosis and treatment. This case report highlights the possibility of COVID-19-related GBS in the pediatric population.

### Statement of Authorship

BBT: Conceptualization, data curation, formal analysis, interpretation of data, writing-original draft, writing-review, and editing. RFP: Conceptualization, data curation, formal analysis, interpretation of data, writing-original draft, writing-review, and editing. RDGJ: Conceptualization, data curation, formal analysis, interpretation of data, writing-original draft, writing-review, and editing.

All authors approved the final version submitted.

### Author Disclosure

All authors declared no conflicts of interest.

### Funding Source

The study has no funding support.

### REFERENCES

- World Health Organization. WHO Coronavirus Disease (COVID-19) Dashboard [Internet]. 2021.[cited 2021 July 24]. Available from: <https://covid19.who.int>
- Collantes ME, Espiritu AI, Sy MC, Anlacan VM, Jamora RD. Neurologic manifestations in COVID-19 infection: a systematic review and meta-analysis. *Can J Neurol Sci.* 2021; 48(1):66-76. doi: 10.1017/cjn.2020.146
- 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
- Yuki N, Hartung HP. Guillain-Barré syndrome. *N Engl J Med.* 2012; 366(24):2294-304. doi: 10.1056/NEJMra1114525
- Abu-Rumeileh S, Abdelhak A, Foschi M, Tumani H, Otto M. Guillain-Barré syndrome spectrum associated with COVID-19: an up-to-date systematic review of 73 cases. *J Neurol.* 2021; 268(4):1133-70. doi: 10.1007/s00415-020-10124-x
- Akçay N, Menentoğlu ME, Bektaş G, Şevketoğlu E. Axonal Guillain-Barré syndrome associated with SARS-CoV-2 infection in a child. *J Med Virol.* 2021; 93(9):5599-602. doi: 10.1002/jmv.27018
- Curtis M, Bhumbra S, Felker MV, Jordan BL, Kim J, Weber M, et al. Guillain-Barré syndrome in a child with COVID-19 infection. *Pediatrics.* 2021; 147(4):e2020015115. doi: 10.1542/peds.2020-015115 2.
- Tabatabaei SR, Shariatpanahi G, Azadkiya R, Karimzadeh P, Karimi A. Guillain-Barré syndrome in a child infected with COVID-19. *Arch Pediatr Infect Dis.* 2021;e113564. doi: 10.5812/pedinfect.113564
- Khalifa M, Zakaria F, Ragab Y, Saad A, Bamaga A, Emad Y, et al. Guillain-Barré syndrome associated with severe acute respiratory syndrome coronavirus 2 detection and coronavirus disease 2019 in a child. *J Pediatric Infect Dis Soc.* 2020; 9(4):510-3. doi: 10.1093/jpids/piaa086
- Manji HK, George U, Mkopi NP, Manji KP. Guillain-Barré syndrome associated with COVID-19 infection. *Pan Afr Med J.* 2020;35 (Suppl2):118. doi: 10.11604/pamj.suppl.2020.35.2.25003
- Paybast S, Gorji R, Mavandadi S. Guillain-Barré syndrome as a neurological complication of novel COVID-19 infection: a case report and review of the literature. *Neurologist.* 2020; 25(4):101-3. doi: 10.1097/NRL.0000000000000291
- Frank CH, Almeida TV, Marques EA, de Sousa Monteiro Q, Feitoza PV, Borba MG, et al. Guillain-Barré Syndrome associated with SARS-CoV-2 infection in a pediatric patient. *J Trop Pediatr.* 2020 July 12: fmaa044. doi: 10.1093/tropej/fmaa044
- Meshref M, Alshammari H, Khairat SM, Khan R, Khan I. Guillain-Barré syndrome associated with COVID-19 infection: a case report with review of literature. *Cureus.* 2021; 13(2):e13096. doi: 10.7759/cureus.13096
- Momen AA, Shakurnia A. The epidemiology of Guillain-Barré syndrome in children under 15 years old in southwest Iran. *Biomed Hub.* 2017; 2(3):1-8. doi: 10.1159/000480693
- Kajumba MM, Kolls BJ, Koltai DC, Kaddumukasa M, Kaddumukasa M, Laskowitz DT. COVID-19-associated Guillain-Barré Syndrome: atypical para-infectious profile, symptom overlap, and increased risk of severe neurological complications. *SN Compr Clin Med.* 2020 Nov 21:1-13. doi: 10.1007/s42399-020-00646-w
- Shahrizaila N, Yuki N. Guillain-barré syndrome animal model: the first proof of molecular mimicry in human autoimmune disorder. *J Biomed Biotechnol.* 2011;2011: 829129. doi: 10.1155/2011/829129
- Paliwal VK, Garg RK, Gupta A, Tejan N. Neuromuscular presentations in patients with COVID-19. *Neurol Sci.* 2020; 41(11):3039-56. doi: 10.1007/s10072-020-04708-8
- Pajo AT, Espiritu AI, Apor AD, Jamora RD. Neuropathologic findings of patients with COVID-19: a systematic review. *Neurol Sci.* 2021; 42(4):1255-66. doi: 10.1007/s10072-021-05068-7
- Gao Z, Xu Y, Sun C, Wang X, Guo Y, Qiu S, et al. A systematic review of asymptomatic infections with COVID-19. *J Microbiol Immunol Infect.* 2021; 54(1):12-16. doi: 10.1016/j.jmii.2020.05.001
- Assaker R, Colas AE, Julien-Marsollier F, Bruneau B, Marsac L, Greff B, et al. Presenting symptoms of COVID-19 in children: a meta-analysis of published studies. *Br J Anaesth.* 2020; 125(3):e330-e332. doi: 10.1016/j.bja.2020.05.026
- Poline J, Gaschignard J, Leblanc C, Madhi F, Foucaud E, Nattes E, et al. Systematic SARS-CoV-2 screening at hospital admission in children: a French prospective multicenter study. *Clin Infect Dis.* 2021; 72(12):2215-7. doi: 10.1093/cid/ciaa1044
- Qiu H, Wu J, Hong L, Luo Y, Song Q, Chen. Clinical and epidemiological features of 36 children with coronavirus disease 2019 (COVID-19) in Zhejiang, China: an observational cohort study. *Lancet Infect Dis.* 2020; 20(6):689-96. doi: 10.1016/S1473-3099(20)30198-5
- Lopez AS, Hill M, Antezano J, Vilven D, Rutner T, Bogdanow, et al. Transmission dynamics of COVID-19 outbreaks associated with child care facilities - Salt Lake City, Utah, April-July 2020. *MMWR Morb Mortal Wkly Rep.* 2020; 69(37):1319-23. doi: 10.15585/mmwr.mm6937e3
- Kelvin AA, Halperin S. COVID-19 in children: the link in the transmission chain. *Lancet Infect Dis.* 2020; 20(6):633-4. doi: 10.1016/S1473-3099(20)30236-X
- Zou L, Ruan F, Huang M, Liang L, Huang H, Hong Z, et al. SARS-CoV-2 viral load in upper respiratory specimens of infected patients. *N Engl J Med.* 2020; 382(12):1177-9. doi: 10.1056/NEJMc2001737
- Zhao H, Lu X, Deng Y, Tang Y, Lu J. COVID-19: asymptomatic carrier transmission is an underestimated problem. *Epidemiol Infect.* 2020;148:e116. doi: 10.1017/S0950268820001235
- Patwa HS, Chaudhry V, Katzberg H, Rae-Grant AD, So YT. Evidence-based guideline: intravenous immunoglobulin in the treatment of neuromuscular disorders: report of the Therapeutics and Technology Assessment Subcommittee of the American Academy of Neurology. *Neurology.* 2012; 78(13):1009-15. doi: 10.1212/WNL.0b013e31824de293
- van Doorn PA, Kuitwaard K, Walgaard C, van Koningsveld R, Ruts L, Jacobs BC. IVIG treatment and prognosis in Guillain-Barré syndrome. *J Clin Immunol.* 2010; 30(Suppl 1):S74-S78. doi: 10.1007/s10875-010-9407-4
- Korinthenberg R, Schessl J, Kirschner J, Mönning JS. Intravenously administered immunoglobulin in the treatment of childhood Guillain-Barré syndrome: a randomized trial. *Pediatrics.* 2005; 116(1):8-14. doi: 10.1542/peds.2004-1324