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Case Report

Pediatric Guillain-Barré Syndrome triggered by SARS-CoV-2: A case study and implications for post-viral neurological complications

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Abstract

Guillain-Barré Syndrome (GBS) is a significant cause of acute, severe weakness in children, with Acute Inflammatory Demyelinating Polyradiculoneuropathy (AIDP) being the most common subtype. The COVID-19 pandemic, caused by the SARS-CoV-2 virus, has been associated with various neurological manifestations, including GBS. We present the case of a 12-year-old male child who developed GBS after a past COVID-19 infection. Although the patient tested negative for SARS-CoV-2 via RT-PCR at the time of admission, he was positive for COVID-19 antibodies, suggesting a prior infection. The patient was treated with intravenous immunoglobulin (IVIG), with significant clinical improvement. This report highlights the potential association between COVID-19 and GBS in children and underscores the importance of recognizing post-infectious neurological complications.

Keywords: Extrapyramidal sign, Hallervorden-Spatz Disease, Dystonia, Dementia.

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1. Introduction

Guillain-Barré Syndrome (GBS) is an autoimmune condition characterized by acute, progressive, symmetric muscle weakness, often following an infectious illness. Acute Inflammatory Demyelinating Polyradiculoneuropathy (AIDP) is the most common subtype of GBS, primarily affecting the peripheral nervous system. Historically, GBS has been linked to respiratory infections, gastrointestinal illnesses, and immunizations.[1]

Since the outbreak of SARS-CoV-2 in late 2019, numerous neurological complications, including GBS, have been reported in both adults and children. While GBS following COVID-19 infection has been described in adults, pediatric cases remain scarce. This case aims to contribute to the growing body of literature on GBS in children post-COVID-19 infection.[2-4]

2. Case Presentation

A 12-year-old male child was admitted with a 10-day history of progressive lower limb weakness, which eventually led to difficulty walking. The patient had a previous history of fever, cough, and upper respiratory symptoms approximately one month prior to the onset of weakness. He had a known exposure to a confirmed COVID-19 case. Upon admission, the patient was afebrile, with stable vital signs (BP: 110/78 mmHg, HR: 90 bpm, RR: 20 breaths/min, SpO2: 99% on room air). On neurological examination, the patient exhibited reduced muscle power (4/5 in upper limbs, 3/5 in lower limbs), absent deep tendon reflexes in the lower extremities, and abnormal proprioception in the distal lower limbs.

MRI of the spine was normal, and cerebrospinal fluid (CSF) analysis showed albumin cytologic dissociation (protein: 450 mg/dL, 1 nucleated cell/µL, 1 RBC/µL). Nerve conduction studies demonstrated features of a sensory-motor demyelinating polyneuropathy, consistent with AIDP. The patient's SARS-CoV-2 RT-PCR test was negative, but a

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SARS-CoV-2 IgG antibody was detected in his serum, indicating prior exposure to the virus.

Nerve: Median-Lt R-Site: APB

Stim Site	Lat 1 (mS)	Lat 2 (mS)	Amp	Dist. (mm)	CV (m/S)
1.Wrist	18.25	31.50	0.86 mV		
2.Elbow	24.50	38.25	0.66 mV	170	27.20

Nerve: Median-Rt R-Site: APB

Stim Site	Lat 1 (mS)	Lat 2 (mS)	Amp	Dist. (mm)	CV (m/S)
1.Wrist	22.63	40.88	1.27 mV		
2.Elbow	26.75	38.63	0.51 mV	170	41.21

Nerve: Peroneal-Lt R-Site: EDB

Stim Site	Lat 1 (mS)	Lat 2 (mS)	Amp	Dist. (mm)	CV (m/S)
1.Ankle	14.00	34.00	1.54 mV		
2.Blw Fib Head	19.88	36.88	1.26 mV	240	40.85
3.Abv Fib Head	21.63	40.88	0.94 mV	70	40.00

Nerve: Peroneal-Rt R-Site: EDB

Stim Site	Lat 1 (mS)	Lat 2 (mS)	Amp	Dist. (mm)	CV (m/S)
1.Ankle	9.13	25.75	1.79 mV		
2.Blw Fib Head	18.38	34.75	1.28 mV	270	29.19
3.Abv Fib Head	20.75	36.13	0.76 mV	70	29.47

Nerve: Tibial-Lt R-Site: EHL

Stim Site	Lat 1 (mS)	Lat 2 (mS)	Amp	Dist. (mm)	CV (m/S)
1.Ankle	9.00	29.25	3.03 mV		
2.Popliteal Fossa	20.13	40.38	1.51 mV	330	29.66

Nerve: Tibial-Rt R-Site: EHL

Stim Site	Lat 1 (mS)	Lat 2 (mS)	Amp	Dist. (mm)	CV (m/S)
1.Ankle	9.13	29.50	2.68 mV		
2.Popliteal Fossa	21.75	36.63	0.72 mV	310	24.55

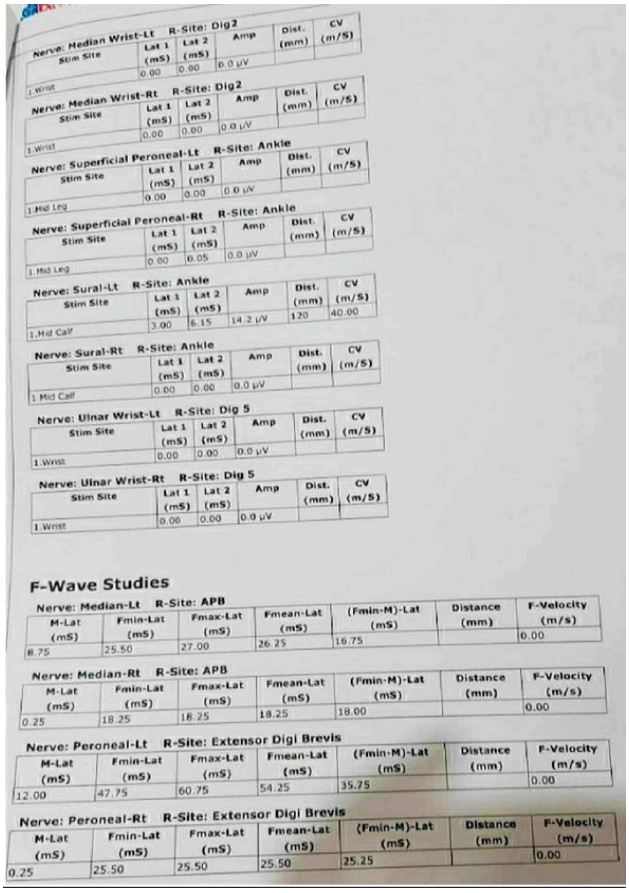
Nerve: Ulnar-Lt R-Site: ADM

Stim Site	Lat 1 (mS)	Lat 2 (mS)	Amp	Dist. (mm)	CV (m/S)
1.Wrist	11.88	29.38	2.85 mV		
2.Below Elbow	19.25	31.38	0.62 mV	180	24.41
3.Abv Elbow	23.00	38.00	0.30 mV	80	21.33

Nerve: Ulnar-Rt R-Site: ADM

Stim Site	Lat 1 (mS)	Lat 2 (mS)	Amp	Dist. (mm)	CV (m/S)
1.Wrist	10.25	28.38	2.88 mV		
2.Below Elbow	16.88	33.13	0.75 mV	160	24.15
3.Abv Elbow	20.25	38.25	0.64 mV	80	23.70

Figure 1: Nerve conduction studies and EMG examination of the upper and lower limbs.



Nerve: Median Wrist-Lt R-Site: Dig2

Stim Site	Lat 1 (mS)	Lat 2 (mS)	Amp	Dist. (mm)	CV (m/S)
1.Wrist	0.00	0.00	0.0 µV		

Nerve: Median Wrist-Rt R-Site: Dig2

Stim Site	Lat 1 (mS)	Lat 2 (mS)	Amp	Dist. (mm)	CV (m/S)
1.Wrist	0.00	0.00	0.0 µV		

Nerve: Superficial Peroneal-Lt R-Site: Ankle

Stim Site	Lat 1 (mS)	Lat 2 (mS)	Amp	Dist. (mm)	CV (m/S)
1.Mid Leg	0.00	0.00	0.0 µV		

Nerve: Superficial Peroneal-Rt R-Site: Ankle

Stim Site	Lat 1 (mS)	Lat 2 (mS)	Amp	Dist. (mm)	CV (m/S)
1.Mid Leg	0.00	0.05	0.0 µV		

Nerve: Sural-Lt R-Site: Ankle

Stim Site	Lat 1 (mS)	Lat 2 (mS)	Amp	Dist. (mm)	CV (m/S)
1.Mid Calf	3.00	6.15	14.2 µV	120	40.00

Nerve: Sural-Rt R-Site: Ankle

Stim Site	Lat 1 (mS)	Lat 2 (mS)	Amp	Dist. (mm)	CV (m/S)
1.Mid Calf	0.00	0.00	0.0 µV		

Nerve: Ulnar Wrist-Lt R-Site: Dig 5

Stim Site	Lat 1 (mS)	Lat 2 (mS)	Amp	Dist. (mm)	CV (m/S)
1.Wrist	0.00	0.00	0.0 µV		

Nerve: Ulnar Wrist-Rt R-Site: Dig 5

Stim Site	Lat 1 (mS)	Lat 2 (mS)	Amp	Dist. (mm)	CV (m/S)
1.Wrist	0.00	0.00	0.0 µV		

F-Wave Studies

Nerve: Median-Lt R-Site: APB

M-Lat (mS)	Fmin-Lat (mS)	Fmax-Lat (mS)	Fmean-Lat (mS)	(Fmin-M)-Lat (mS)	Distance (mm)	F-Velocity (m/s)
8.75	25.50	27.00	26.25	16.75		0.00

Nerve: Median-Rt R-Site: APB

M-Lat (mS)	Fmin-Lat (mS)	Fmax-Lat (mS)	Fmean-Lat (mS)	(Fmin-M)-Lat (mS)	Distance (mm)	F-Velocity (m/s)
0.25	18.25	18.25	18.25	18.00		0.00

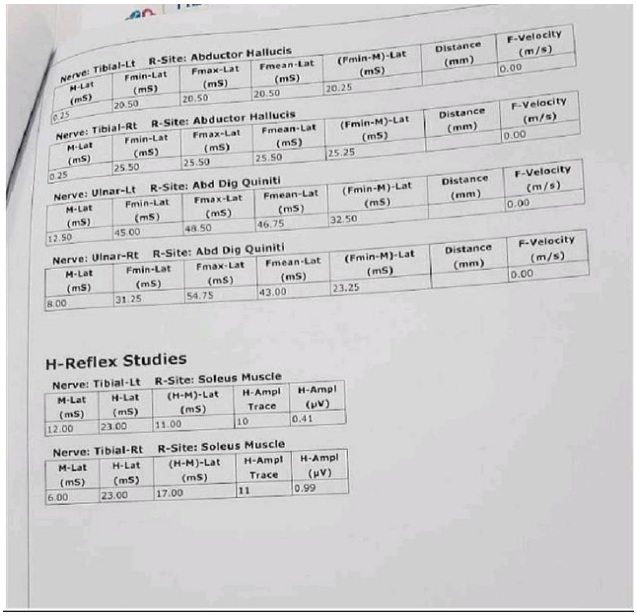
Nerve: Peroneal-Lt R-Site: Extensor Digi Brevis

M-Lat (mS)	Fmin-Lat (mS)	Fmax-Lat (mS)	Fmean-Lat (mS)	(Fmin-M)-Lat (mS)	Distance (mm)	F-Velocity (m/s)
12.00	47.75	60.75	54.25	35.75		0.00

Nerve: Peroneal-Rt R-Site: Extensor Digi Brevis

M-Lat (mS)	Fmin-Lat (mS)	Fmax-Lat (mS)	Fmean-Lat (mS)	(Fmin-M)-Lat (mS)	Distance (mm)	F-Velocity (m/s)
0.25	25.50	25.50	25.50	25.25		0.00

Figure 2: Nerve conduction studies and EMG examination showing absent sensory nerve action potentials (SNAPs) and reduced compound muscle action potentials (CMAPs).



Nerve: Tibial-Lt R-Site: Abductor Hallucis

M-Lat (mS)	Fmin-Lat (mS)	Fmax-Lat (mS)	Fmean-Lat (mS)	(Fmin-M)-Lat (mS)	Distance (mm)	F-Velocity (m/s)
0.25	20.50	20.50	20.50	20.25		0.00

Nerve: Tibial-Rt R-Site: Abductor Hallucis

M-Lat (mS)	Fmin-Lat (mS)	Fmax-Lat (mS)	Fmean-Lat (mS)	(Fmin-M)-Lat (mS)	Distance (mm)	F-Velocity (m/s)
0.25	25.50	25.50	25.50	25.25		0.00

Nerve: Ulnar-Lt R-Site: Abd Dig Quiniti

M-Lat (mS)	Fmin-Lat (mS)	Fmax-Lat (mS)	Fmean-Lat (mS)	(Fmin-M)-Lat (mS)	Distance (mm)	F-Velocity (m/s)
12.50	45.00	48.50	46.75	32.50		0.00

Nerve: Ulnar-Rt R-Site: Abd Dig Quiniti

M-Lat (mS)	Fmin-Lat (mS)	Fmax-Lat (mS)	Fmean-Lat (mS)	(Fmin-M)-Lat (mS)	Distance (mm)	F-Velocity (m/s)
8.00	31.25	54.75	43.00	23.25		0.00

H-Reflex Studies

Nerve: Tibial-Lt R-Site: Soleus Muscle

M-Lat (mS)	H-Lat (mS)	(H-M)-Lat (mS)	H-Ampl Trace	H-Ampl (µV)
12.00	23.00	11.00	10	0.41

Nerve: Tibial-Rt R-Site: Soleus Muscle

M-Lat (mS)	H-Lat (mS)	(H-M)-Lat (mS)	H-Ampl Trace	H-Ampl (µV)
6.00	23.00	17.00	11	0.99

Figure 3: Nerve conduction studies and EMG examination demonstrating increased latency and reduced velocity in motor nerves.

A diagnosis of GBS, AIDP form, was made. The patient was treated with intravenous immunoglobulin (IVIG) therapy (2 g/kg over 48 hours). Over the next few days, there was gradual improvement in muscle strength (4/5 in both upper and lower limbs). Physical therapy was initiated, and at the time of follow-up three weeks later, the patient showed continued recovery, with the ability to sit and walk independently.

3. Discussion

This case represents the first documented pediatric GBS case in our setting associated with a past SARS-CoV-2 infection. GBS in children has been linked to several viral infections, including coronaviruses such as SARS-CoV and MERS-CoV. Recent reports also suggest an association between COVID-19 and various forms of GBS, including demyelinating, axonal, and Miller-Fisher syndrome.[4,5]

Our patient presented with the classic features of GBS, including symmetric ascending weakness and areflexia. Electrophysiological findings were consistent with AIDP, and CSF analysis demonstrated the characteristic albumin cytologic dissociation. The temporal relationship between the patient's previous COVID-19 symptoms and the onset of GBS strongly supports a post-infectious autoimmune process triggered by the virus.

While SARS-CoV-2 has been shown to have neurotropic properties, its exact role in the pathogenesis of GBS remains unclear. Studies on the neuro-invasive potential of SARS-CoV-2 have primarily focused on its effects on the central nervous system, with few cases investigating its impact on the peripheral nervous system. The absence of SARS-CoV-2

in the CSF in our case aligns with previous findings that suggest the virus may trigger immune-mediated peripheral nerve damage without direct viral invasion.[6]

This case emphasizes the importance of considering GBS in the differential diagnosis of pediatric patients with progressive neurological deficits following a COVID-19 infection. It also highlights the need for further studies to understand the underlying mechanisms linking COVID-19 and GBS.[7,8]

4. Conclusion

GBS following COVID-19 infection, although rare, is a potential neurological complication that should be recognized in pediatric patients. Our case demonstrates the need for heightened awareness of post-viral autoimmune neurological disorders in children, particularly in the context of the ongoing COVID-19 pandemic. Timely diagnosis and treatment with IVIG can lead to significant recovery, as seen in this patient.

5. Human Subjects

Consent was obtained or waived by all participants in this study.

6. Conflicts of Interest

None.

7. Payment/Services Info

All authors have declared that no financial support was received from any organization for the submitted work.

8. Financial Relationships

All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work.

9. Other Relationships

All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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