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Guillain-barrel syndrome-sequelae in COVID-19 positive children.

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Abstract

Introduction: Guillain-Barrel Syndrome is an important cause of severe, acute weakness in children. Acute Inflammatory Demyelinating Polyradiculoneuropathy (AIDP) is the most common subtype. History of past infection was noticed in the majority of cases. In Wuhan China in late 2019, severe acute respiratory syndrome corona virus-2 (SARS-CoV-2) originated and rapidly spread around the world.

Case: We had 12 years old male child who was admitted with history sudden onset of lower limb weakness. He had history of contact with COVID-19 patient. Clinical and Laboratory finding suggesting of Guillain-Barré Syndrome.

Discussion: This case is the first reported case of a child with GBS with past history acute SARS-CoV-2 infection one month back in our set up. GBS in a child has been reported associated with other forms of corona virus. The patient came with obvious symptomatology of GBS with symmetric ascending weakness with loss of reflexes.

Conclusion: The relation between COVID-19 and GBS was demonstrated previously in case reports of adults with a different variety of GBS, like demyelinating, axonal, and Miller-Fisher in connection with COVID-19. Typical post infectious presentations have been seen in our report after one month.

Keywords: Guillain-barrel syndrome, COVID-19, SARS-CoV-2, COVID-19 positive children.

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Introduction

Guillain-Barre Syndrome is an important cause of severe, acute weakness in children, and Acute Inflammatory Demyelinating Polyradiculoneuropathy (AIDP) is the most common subtype [1]. GBS is characterized by a monophasic, ascending, and symmetrical paralysis that progresses over days to weeks and is associated with areflexia. AIDP is a post- infectious autoimmune process to peripheral nerves which causes inflammation and destruction of myelin. History of past infection was noticed in the majority of cases. Respiratory illness are the most common infectious triggers, but gastrointestinal illnesses, other viruses and immunizations have also been related with GBS [2].

In Wuhan China in late 2019, severe acute respiratory syndrome corona virus-2 (SARS-CoV-2) originated and rapidly spread around the world. It brought out a pandemic of novel corona virus disease 2019 (COVID-19). The majority of paediatric disease is asymptomatic. The common symptoms of COVID-19 are fever, malaise, and respiratory symptoms, which may range from mild cough to severe pneumonia. It presents occasionally with gastrointestinal symptoms [3]. However, COVID-19 can present with variety of other symptoms which includes neurological symptoms in adult patients [4]. Anosmia is the most common neurological presentations among COVID-19 patients. However, others include encephalopathy, encephalitis, stroke, acute-disseminated encephalomyelitis, as well as neuro-inflammatory auto-immune diseases [5].

There are scattered reports of adults with possible GBS and concurrent evidence of COVID-19. There are very few previously reported cases on GBS in children with evidence of COVID-19 [6-10].

Case Presentation

A 9 years old male presented in outpatient department with progressive weakness in both lower limbs and difficulty in walking since 10 days. He had history of frequent fall onto his buttocks before presentation. He complained pain in both lower limbs since 8 days. Over the next couple of days, he started experiencing bilateral lower extremity weakness that progressed and which leads to inability to walk. He was evaluated at a local family physician where lumbar spine and right ankle x-rays were done which were normal. Over the next few days, his weakness worsened as he began to develop upper extremity weakness. The patient was transferred to a tertiary care children's hospital for further evaluation.

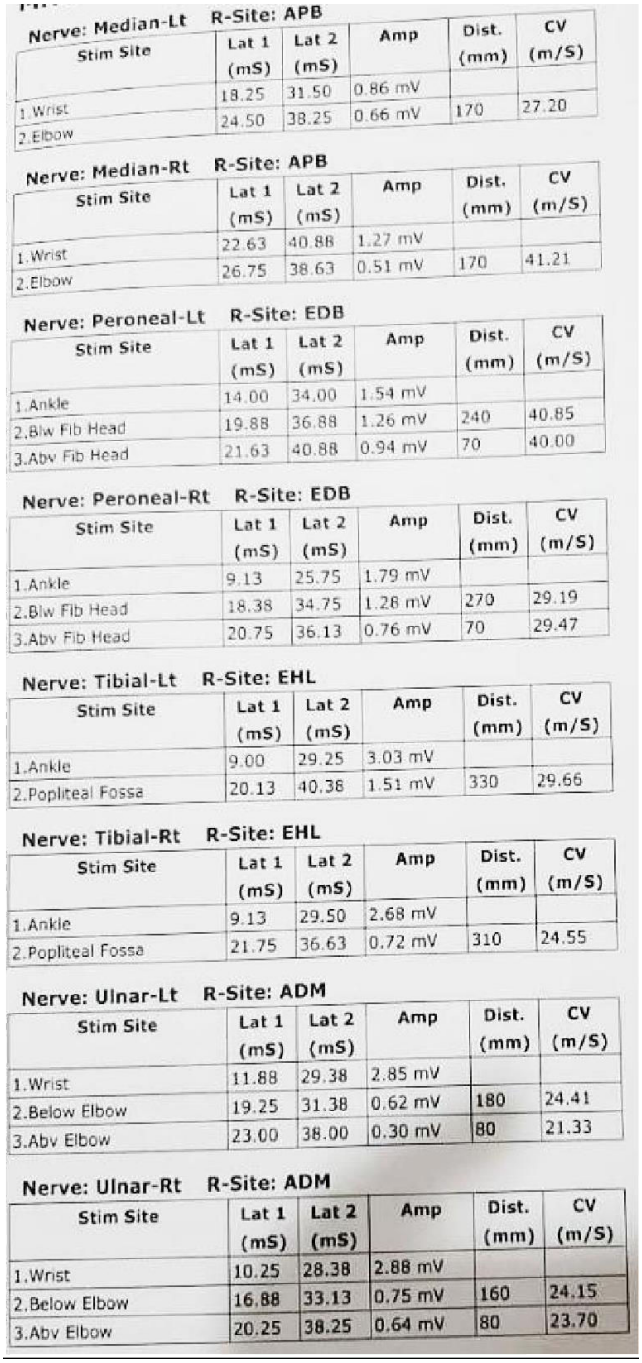
The parents gave history of illness 1 month back; including fever, upper respiratory infection, and cough. The patient's SARS-CoV-2 nucleic acid amplification was positive. The patient did not have urinary or faecal incontinence. He passed stool once in 3 days and there was no difficulty voiding urine.

The father is a daily wage worker and works outside the home. The patient had history of playing outside home and visited to a local mall 3 weeks prior to presentation, during which he wore a mask. Patient belonged to containment zone for COVID-19.

At the time of admission, the patient was afebrile with blood pressure 110/78 mmHg, heart rate of 90 beats per minute, respiratory rate of 20 breaths per minute, and oxygen saturation of 99% on room air. He appeared anxious but was alert and oriented. On central nervous examination, he was able to do conversation and able to speak a sentence with 5-6 words at a time. Cranial nerves were intact. Overall muscle power was reduced to 4/5 in the upper limbs and 3/5 in the lower extremities. Deep tendon reflexes were absent in lower extremities bilaterally. Sensation was intact to light touch, but proprioception of the distal lower extremities was abnormal. Single breath count test was.

An MRI of the spine was done and it was normal. A lumbar puncture was also performed. The cerebrospinal fluid demonstrated albuminocytologic dissociation with 1 nucleated cell/cumm, 1 RBCs/cumm, and protein of 450 mg/dl. Gram stain, culture was negative.

Electro diagnostic testing including F waves was performed 10 days after symptom onset. It demonstrated nearly absent CMAP (compound muscle action potential) amplitude in both median nerves. Markedly reduce CMAP in both peroneal nerves. Both ulnar and tibial nerves shows increase latency with reduce velocity. Conduction block was seen in left tibial and right ulnar nerve. Sensory nerve conduction showed absent SNAP (sensory nerve action potential) amplitudes in all sampled upper and lower limb nerves. F waves was absent in both peroneal and median nerve. H reflex was absent bilaterally. (Figures 1-3). These findings were compatible with a sensory motor demyelinating polyneuropathy.



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

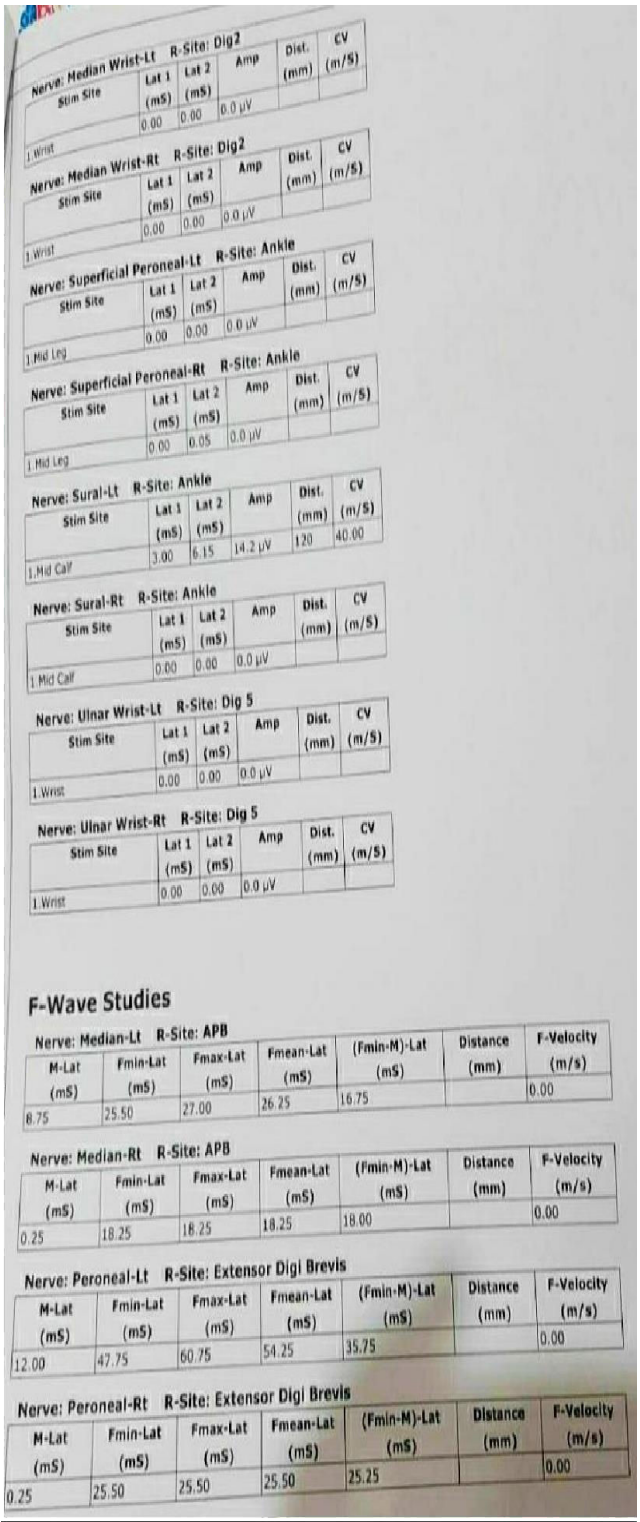
Nerve: Ulnar-Lt R-Site: ADM

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.*



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

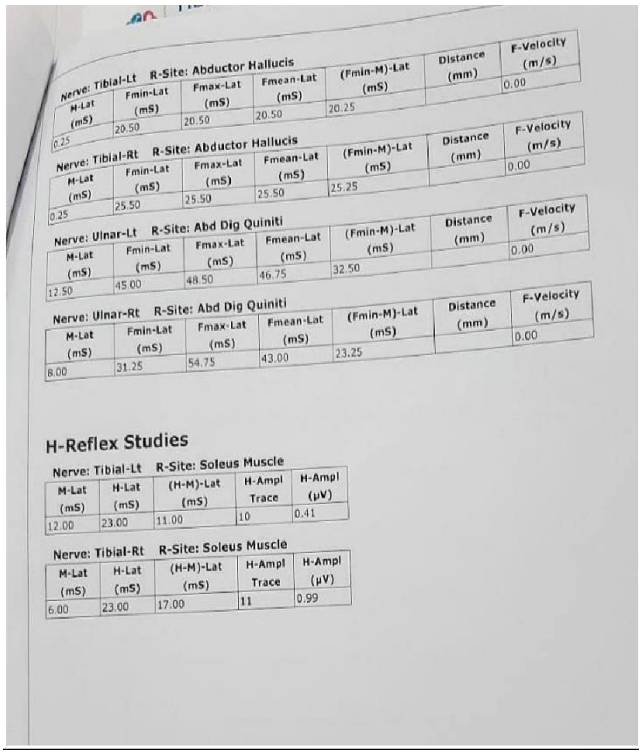
Nerve: Peroneal-Lt R-Site: Extensor Digi Brevis

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.*



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*

A diagnosis of GBS, AIDP form, was made. Patient underwent infectious evaluation for cause of the GBS. Blood, urine, and stool cultures were negative. SARS-CoV-2 IgG antibody was detected in his serum. Further work up showed WBC 14,200/ cumm. Treatment was initiated with Intravenous Immuno Globulin (IVIG) on day two of hospitalization. He was given a total of 2 g/kg of IVIG over 48 hours. His exam demonstrated improvement over the next several days following IVIG with 4/5 upper extremity power and 4/5 lower extremities bilaterally. Physical therapy was initiated. At the time of publication, three weeks following IVIG, he continues to demonstrate slow improvement. He is able to sit and walk independently. He was tested SARS-CoV-2 which came negative on admission. Informed consent was taken from parents of the patient for reporting the case.

Discussion

Case is the first reported case of a child with GBS with past history acute SARS-CoV-2 infection one month back in our set up. GBS in a child has been reported associated with other forms of corona virus. The patient came with obvious symptomatology of GBS with symmetric ascending weakness with loss of reflexes. The workup subsequently was consistent with GBS, AIDP form. The CSF showed raised protein without pleocytosis, there was enhancement of the posterior nerve roots in the cauda equina on MRI, and Electrophysiological findings (EMG) demonstrated a demyelinating process.

The relation between COVID-19 and GBS was demonstrated previously in case reports of adults with a different variety of GBS, like demyelinating, axonal, and Miller-Fisher in connection with COVID-19 [10-12]. Typical post infectious

presentations have been seen in our report after one month [13-17].

Conclusion

SARS-CoV-2 and other corona viruses, SARS and MERS specifically, have been shown to have neurotropic nature and it causes diseases of the central and peripheral nervous system. The absence of SARS-CoV-2 in the CSF is constant reports as per past publications. The real course and physiology of these reasons have yet to be determined.

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