Guillain-Barre Syndrome Secondary to COVID-19: A case report and short review of other published cases

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ABSTRACT

Background: COVID-19 related Guillain-Barré syndrome has a broad spectrum of presentation. In most reported cases, respiratory symptoms preceded neurological deficits by one to two weeks, suggesting that the clinical course is mostly post-infectious. In this case report, we present a para-infectious case of GBS with COVID-19.

Case presentation: A 37-year-old male patient presented with fever, chills, myalgia, cough, and anosmia. COVID-19 test came positive. He was managed conservatively. On the 7th day of follow-up, he recovered except for a persistent loss of smell and taste. Two weeks after his initial presentation, he reported low back pain and bilateral lower extremity weakness and had a repeat COVID-19 test, which returned positive. His history, physical exam, CSF analysis, nerve conduction, and electromyography test revealed Guillain-Barre Syndrome. We managed GBS with supportive treatment in the hospital, and on follow-up of three months, he recovered fully.

Conclusion: In our case, we report a para-infectious case of GBS with C0VID-19, and we managed this case without intravenous immunoglobulin or plasmapheresis. The decision to treat a COVID-19 related GBS case with a traditional GBS treatment option (intravenous immunoglobulin or plasmapheresis) should be taken in conjunction with co-morbidities and a tailored case by case basis.

Keywords: *COVID-19, Guillain Barre Syndrome, Acute inflammatory demyelinating polyradiculopathy, Anosmia*

Introduction

Guillain Barre Syndrome (GBS) is an acute immune-mediated disease of the peripheral nerves and nerve roots (polyradiculoneuropathy) that is usually elicited by various infections.[1] Classically, GBS presents as progressive, ascending, symmetrical limb weakness, along with areflexia or hyporeflexia and with or without cranial nerve involvement, which can progress over days to several weeks.[1,2] In the majority, an antecedent respiratory or gastrointestinal infection history was found two to four weeks before the onset of the neurological syndrome of GBS.[1] COVID-19 manifestations can range from mild flu-like illness to severe pneumonia or acute respiratory distress syndrome. Neurological, cardiac and thromboembolic complications are widely reported from across the globe.[3,4,5] From a neurological point of interest, SARs-CoV-2 infection can cause encephalopathy, encephalitis, myelitis, meningitis, acute cerebrovascular disease, Guillain Barré Syndrome (GBS), and exacerbation of myasthenia gravis.[6,7] There are very few cases of para-infectious GBS with COVID-19 reported in the literature. Here we report a case of GBS during the active phase of COVID-19 infection.

Case Report

A 37-year-old Hispanic gentleman with a past medical history of diabetes mellitus type-2, asthma, and hypertension initially presented with fever, chills, myalgia, cough, and anosmia. He is a poultry farm worker, and many of his co-workers had come down with similar symptoms. Given the symptoms and outbreak at his work, the nasopharyngeal swab was tested for COVID-19 with CDC Real-Time- Polymerase Chain Reaction primers (RT-PCR) came positive. The patient was managed conservatively. He did not require hospitalization. On the 7th day of follow-up, the patient had recovered, except for the persistence of loss of smell and taste. Two weeks later, to his initial presentation, the patient presented to ER with three days history of low back pain and progressively worsening bilateral lower extremity weakness. In those three days at home, weakness progressed to the point where the patient could not walk without holding support with both hands. Neurology was consulted to evaluate for weakness. Repeat Nasopharyngeal swab tested positive for COVID-19 (RT-PCR). The patient was asymptomatic from a respiratory viewpoint; his oxygen saturation was 99%, he was comfortable on room air. On neurological exam, the patient was awake, alert, and oriented. Strength was MRC 5/5 (Medical Research Council scale) in both upper extremities and MRC 2/5 in both lower extremities distally and MRC 3/5 proximally. The patient was areflexic in the lower extremity, and upper extremity reflexes were diminished at 1+. There was paresthesia involving both lower extremities to pinprick and fine touch in a patchy distribution. Vibratory sense at the first metatarsal head was reduced symmetrically (5 seconds), while normal

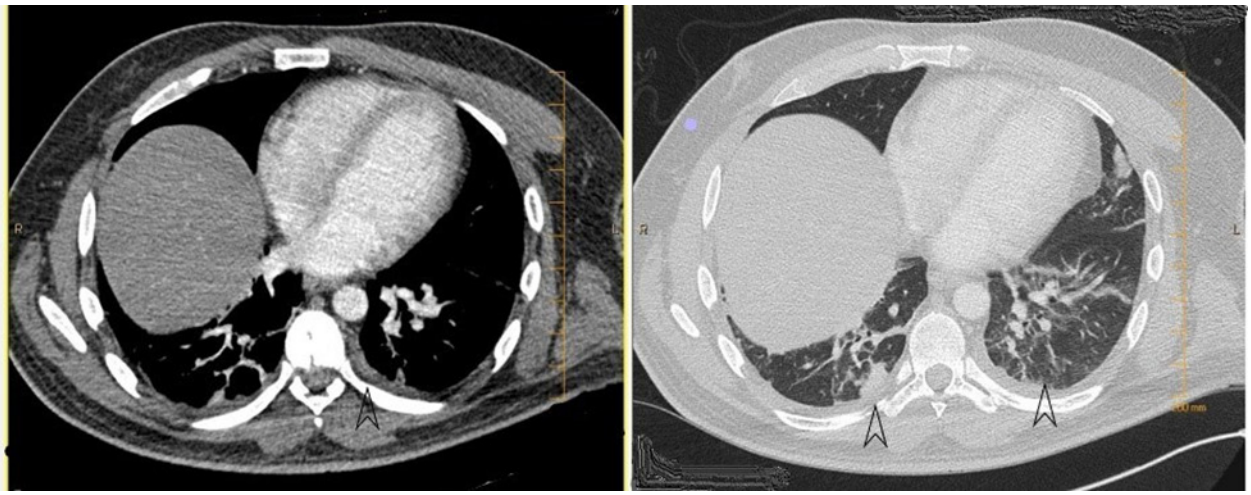

Figure 1: CT chest showing consolidation of lung bases

in both upper limbs at the base of the thumb (more than 14 seconds); there was no joint position loss sensation in upper or lower limbs. His rectal tone and perianal sensation were intact, and plantar reflexes were mute. There was no extraocular weakness, facial palsy, hypophonia, orthopnea, or respiratory distress. His NIF (negative inspiratory force) was -65 cm of water (normal). Other cranial nerve examination was normal. Based on history, exam findings, Guillain-Barré syndrome was suspected. We recommended MRI (with and without contrast) of the cervical, thoracic, and lumbar spine, which was unremarkable. His CT chest showed ground-glass opacity in bilateral lung bases at day 19 of his initial symptoms (Figure 1). Blood work showed; ESR 67 mm/hour (normal value 0-22 mm/hour), CRP 15.16 mg/dL (normal value 0.00-0.50 mg/dL), D-dimer 5.40 mcg/ml (normal value <0.50), Fibrinogen 560 mg/dl (normal value <50 mg/dl) LDH 797 units/L (normal value 140-280 U/L), CK 31 units/L (normal value 22-198 U/L), HbA1c 13.4% (normal value 4%-5.6%). CSF study showed protein of 107 mg/dl (normal value 8-43 mg/dl), 0 WBC, glucose of 166 mg/dl (normal value 50-80 mg/100 mL or greater than 2/3 of blood sugar). CSF analysis for Epstein Barr virus, Cytomegalovirus, Varicella, Herpes simplex type I & II were negative. We performed NCV-EMG (Nerve Conduction Velocity- Electromyography) to confirm Guillain-Barré syndrome and look for a loss pattern, either axonal or demyelination. Nerve conduction study summarized in *Table 1*. Sensory nerve conduction studies of the left radial and both sural nerves showed low evoked response amplitude and slow conduction velocity. Motor nerve conduction studies of the left median and left peroneal nerves showed normal distal motor latency, normal evoked response amplitude, and slow conduction velocity. The left tibial nerve shows normal distal motor latency, low evoked response amplitude, and slow conduction velocity in demyelinating range (laboratory criteria for tibial nerve: CMAP amplitude more than 3.2 milli-volts and conduction velocity less than 32 meter/second). There were no temporal dispersions. The minimum F wave latencies obtained were prolonged on the left median, peroneal and tibial nerves. Needle electromyography (though limited) showed reduced recruitment in tibialis anterior, peroneus tertius, and vastus medialis with neurogenic motor unit potentials in peroneus tertius. Based on the history of rapidly progressive lower extremity weakness, albumin-cytological dissociation on the CSF study, and electrodiagnostic findings, the patient was diagnosed with a acute inflammatory demyelinating polyradiculopathy (AIDP). Based on electrodiagnostic criteria for AIDP, published by Al-Shekhlee et al.,[8] we categorized our patient electro-diagnostically as a suggestive case of AIDP. However, H reflexes were not checked. Also, we suspect the patient had an underlying chronic sensorimotor axonal neuropathy likely from long-standing poorly controlled type-II diabetes mellitus. Throughout the hospital course, he was managed conservatively and did not require additional respiratory support. He was getting physiotherapy while in hospital and after discharge. He was discharged upon the improvement of motor strength. At six weeks of follow-up, he was able to walk with a cane; his sense of taste and smell had returned as well. At three months of telehealth follow-up, he was walking without any support. The patient's clinical syndrome of acute flaccid paralysis and demyelination on electrodiagnostic studies was not secondary to uncontrolled diabetes, as the patient improved in a relatively shorter duration, unlike diabetic neuropathy. Follow-up EMG-NCV was not planned as the patient was clinically improved.

Discussion

Guillain-Barré syndrome can occur secondary to an autoimmune response to a bacterial or viral epitope that leads to the formation of antibodies that cross-react with

Table 1: Nerve Conduction Study-Electromyography

Sensory Nerve Conduction Study				
Nerve/Site	Segment	Peak ms.	Amp. uV	Vel m/s
Left Radial- Forearm	Snuffbox	2.7 (<2.9)	7.0 (>15)	46.3
Left Sural- Calf	Lateral Ankle	5.2 (<4.4)	1.6 (>6)	34.1
Right Sural- calf	Lateral Ankle	4.6 (<4.4)	1.6 (>6)	36.5
Motor Nerve conduction study				
Nerve/Site	Recording site	Latency ms	Amp. mV	Vel m/s
Left Median- Wrist	APB	4.1 (<4.4)	4.8 (>4)	
Left Median- Elbow	APB	9.6	4.4	45 (>49)
Left Peroneal- Ankle	EDB	4.8 (<6.5)	4.6 (>2.5)	
Left Peroneal- Fibular head	EDB	14.4	3.8	33.4 (>44)
Left Peroneal- Popliteal fossa	EDB	16.9	3.5	37.3 (>44)
Left Tibial- Ankle	AH	4.5 (<5.8)	6.9 (>4)	
Left Tibial- Popliteal fossa	AH	18.2	3.7	30.7
F wave				
Recording site	Value. mS			
Left- Median	32.6 (<31)			
Left- Paroneal	60.5 (<57)			
Left Tibial	59.1 (<58)			

ganglioside, i.e., molecular mimicry.[20] In earlier epidemics, MERS (Middle Eastern respiratory syndrome) and Zika virus were reported to cause Guillain-Barré syndrome.[21] *Table 2* describes the clinical courses of reported COVID-19 related GBS cases. The spectrum of presentation is broad; outcomes are variable with different interventions. In most cases, respiratory symptoms preceded neurological deficits by one to two weeks, suggesting the clinical course is mostly post-infectious, like other commonly known GBS triggers. Abolmali M. et al. reported three cases of COVID-19-related GBS which the author describes as para-infectious as all three patients developed neurological manifestations during the active phase of COVID-19.[22] None of these three cases were reported to get tested for SARs-CoV-2 PCR in CSF.

Generally, post-infectious cases are caused by an autoimmune reaction to the infectious agent, which cross-react with neural antigen. In such cases, neuropathy develops several days to weeks later after the initial infection. On the other hand, following COVID-19 illness, if hyperimmune response ensues, the appearance of neuropathic symptoms earlier is logical. The para-infectious course of neuropathy develops relatively faster within a few days due to infectious agents, like neuroborreliosis, neuro brucellosis, and West Nile Virus-related acute flaccid paralysis, etc. None of the reported GBS cases were found to have COVID-19 RT-PCR positive on CSF. COVID-19 cases may remain clinically silent at the beginning or asymptomatic throughout.[23] Some can develop aggressive diseases relatively later than others. We hypothesize the timeline of initial respiratory symptoms in relation to onset of the neurological syndrome might not accurately indicate (rather underestimate) the window period between actual infection and onset of neurological syndrome in some cases. The incubation period also varies from few days to two weeks. While suffering from asymptomatic or mild symptomatic COVID-19 infection, the patient can still trigger an immune response. We could not conclude any correlation between the severity of COVID-19 disease and Guillain Barre syndrome. Relatively milder to severe, both patterns of cases were reported to cause GBS.

In our case, the patient presented after two weeks of COVID-19 diagnosis; his respiratory symptoms had resolved by then. But we still could find ground-glass

opacity on chest CT. Among the eighteen cases described (including ours), eight cases were diagnosed as acute inflammatory demyelinating polyneuropathy (ours was a paraparetic variant), five cases as acute motor-sensory axonal neuropathy (AMSAN), two cases as Miller Fisher variant, two cases as facial diplegic variant, one case as acute motor axonal neuropathy (AMAN), and one case as polyneuritis cranialis variant. One case was reported as a severe autonomic failure, and one with dysautonomia. CSF protein was high in eleven cases. Four cases had normal CSF protein. COVID-19 RT-PCR in CSF was negative in all tested cases. We did not test CSF for COVID-19 (RT-PCR) in our case, as it's not a reliable marker for neurological injury. COVID-19 related neurological issues seem to be caused by indirect mechanisms.[24] Thirteen cases were treated with IVIG, one case with IVIG and plasmapheresis, two cases (including our case) were not treated with IVIG or plasmapheresis. We did not choose to treat our case with IVIG, as COVID-19 illness is widely reported to cause thromboembolic events,[25] and thrombotic events are a known side effect of IVIG (likely related to hyper-viscosity).[26,27] Plasmapheresis was not chosen, because the patient was thrombocytopenic with platelet count of 23,000/microliter (normal range 150,000-450,000/microliter), and anemic, hemoglobin of 6.9 gm/dL (normal 13.5-17.5 gm/dL). Plasmapheresis can deplete coagulation factors and antithrombin III, leading to bleeding complications.[28] Our patient did not have any signs of impending respiratory failure, nor did his weakness progress further. Two of the total eighteen cases who did not get IVIG or plasmapheresis had a good recovery. Our patient also had fair motor recovery at six weeks, and at three months, was walking without support. Of the eight cases treated with IVIG, five had a good recovery, two did not improve, and one progressed to neuromuscular respiratory failure.

Overall, we believe GBS related to COVID-19 disease can present with a large spectrum of neurologic syndrome. Neuromuscular respiratory failure, severe dysautonomia secondary to GBS would make hospital courses in ICU even more complicated. Early recognition of GBS manifestation and early decision on plasmapheresis and intravenous immunoglobulin is mandatory. At the same time, co-morbidities, clinical course, and the severity of the clinical situation should be gazed meticulously before deciding on a treatment plan.

Declarations

1. Ethics approval and consent to participate:
This study is approved by the University of Missouri I.R.B.

2. Ethics Statement:
Written consent was obtained from the patient before submission for publication.

4. Competing Interest:
The authors have no conflict of interest to report.

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Biswajit Banik (BB) and Mukaish Kumar (MK) wrote the manuscript of the article. Raghav Govindarajan (RG), BB, and MK were involved in designing concepts, literature search and involved in drafting the article. All authors contributed to the manuscript, and RG approved the final version of the manuscript.

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Table 2: Summary of COVID-19 associated GBS cases

Author	Pertinent clinical information and timeline of neurological and COVID symptoms.	Investigations	Electrodiagnostics	Diagnosis	Treatment	Outcome
(Zhao, Shen et al. 2020)[9]	61-year female, presented with fatigue and acute weakness of both lower extremities progressive to upper extremities. On exam, symmetric Lower extremity strength 3/5 (MRC scale) and upper extremity strength 4/5 (MRC scale), areflexia in both legs and feet, also distal decreased sensation to pin prick and light touch. Patient initially presented with neurological symptoms, on 8th day of presentation with neurological symptoms developed dry cough, fever. CT showed ground glass opacity. Oropharyngeal swab tested positive for COVID.	-CSF protein: 124 mg/dl. (Day 4) -CSF WBC-Normal	Delayed distal latencies Absent F waves in early course.	AIDP	-IVIG -Ropinavir -Arbidol -Ritonavir	Total recovery at day 30 with normal reflexes.
(Padroni, Mastrangelo et al. 2020)[10]	70-year old female, presented with acute onset of asthenia, hand-feet paresthesia, gait difficulty. On exam, B/L UE and LE extremity strength 4/5 (MRC scale), absent deep tendon reflexes, preserved light touch and pinprick sensation. Patient had developed fever (38.5 C) and dry cough 24 days prior to her presentation with neurological symptoms. She tested positive on nasopharyngeal swab (RT-PCR) on 2nd day of respiratory symptoms.	-CSF protein 48 mg/dl. (Day 4) -WBC: $1x10^6$/L, normal (0-0.8 x 10^6/L)	Conduction block, Temporal dispersion, Soleus H reflex absent.	AIDP	-IVIG	Patient intubated on day 5.
(Ottaviani, Boso et al. 2020)[11]	66-year female, presented with 3 days of walking difficulty and fatigue progressing to paraparesis. On exam, paraparetic, distal UE strength 4/5 (MRC scale), diffuse areflexia, no clear sensory deficit. Patient had mild fever and cough 10 days prior neurological symptoms. Initial nasopharyngeal swab was negative for COVID, repeat swab positive. CT chest showed ground glass appearance.	-CSF protein 180 mg/dl. -WBC 0. -Ganglioside antibody negative. -CSF SARS CoV-2 not detected.	Absent F wave. Diffuse prolong distal motor latencies. Reduced distal CMAP, slight reduction in conduction velocity.	AIDP with axonal loss.	IVIG Lopinavir Ritonavir Hydroxychloroquine	Patient worsened. Needed Mechanical intubation, developed unilateral facial palsy
(Assini, Benedetti et al. 2020)[12]	55-year male, initially developed anosmia, ageusia; hospitalized for COVID-19 (Oropharyngeal swab positive) related respiratory issues. On 3rd day pt. required intubation for respiratory failure secondary to COVID pneumonia. Later, on day 20, developed Neurological syndrome of eyelid ptosis, dysphagia and dysphonia. On exam, bilateral masseter weakness, tongue protrusion deficit due to CN XII palsy, deficit of soft palate elevation due to CN X palsy, diffuse hyporeflexia, no extremity weakness. Respiratory symptoms onset at least 20 days prior to onset of neurological symptoms.	CSF protein: Normal. Oligoclonal band both in CSF and serum with increased IgG/Albumin ratio (233)	Symmetric demyelination with sural sparing pattern. Repetitive nerve stimulation negative. Anti-ganglioside antibody negative.	AIDP with Miller fisher overlap	-IVIG -Idrossichlorochine -Arbidol -Ritonavir -Lopinavir	Very rapid clinical recovery in swallowing, speech, tongue motility and strength, ptosis.

(Assini, Benedetti et al. 2020)[12]	60-year male, hospitalized for COVID-19 respiratory symptoms, requiring tracheostomy and assisted ventilation; at day 20 of hospitalization, developed acute weakness in lower limb with distal distribution and foot drop on right side. Gastroparesis, paralytic ileus, loss of BP control noticed. On exam, deep tendon reflexes absent, distal weakness of all four limbs noticed. Respiratory symptoms onset 20 days prior (or more) to onset of neurological symptoms.	CSF protein: Normal. Oligoclonal band in CSF and serum with increased ratio. CSF IgG/albumin 170.	Severe sensory-motor axonal polyneuropathy with relative sparing of conduction velocity. Loss of amplitude in sensory and motor axon potential.	AMSAN- with severe autonomic neuropathy	-IVIG -Hydroxychloroquine -Tocilizumab	Symptomology improved except gastroparesis. Paralytic ileus improved. Hyporeflexia and foot drop persisted.
(Toscano, Palmerini et al. 2020)[13]	77-year female, presented with paresthesia in limbs and hands, over next 36 hours became flaccid areflexic, tetraparetic. Later during IVIG developed dysphagia, tongue weakness. COVID-19 symptoms 7 days prior to onset of neurological symptoms.	Day 2: CSF- Normal Day 10: CSF Protein: 101 mg/dl. (Normal 23-43 mg/dl). CSF WBC: 4/mm^3 Spine MRI- Caudal nerve root enhancement	Axonal loss Sural sparing pattern	AMSAN	-IVIG, two cycles	Poor outcome after 2 cycle of IVIG. Persistence of severe UE weakness, LE paraplegia Dysphagia
(Toscano, Palmerini et al. 2020)[13]	23-year male, presented to ER, with facial weakness that progressed to total LMN type facial paresis in 2 days. Also had loss of taste, lower limb paresthesia, generalized areflexia, sensory ataxia. COVID-19 related respiratory symptoms 10 days prior to presentation.	Day 3: Protein 123 mg/dl, no cells.	-Axonal sensory motor changes involving both limb, sural sparing pattern. -Decreased facial nerve CMAP amplitude. -MRI head- Facial nerve enhancement bilateral	AMSAN with facial diplegia	-IVIG	Improved ataxia, paresthesia, mild improvement of facial weakness
(Toscano, Palmerini et al. 2020)[13]	55-year male, presented with neck pain, paresthesia in four limbs and lower limb weakness. In two days, he was flaccid areflexic tertraparetic. COVID-19 related symptoms 10 days prior to onset of neurological symptoms.	Day 3, CSF Protein: 193 mg/dl, no cells. CSF: negative PCR for SARs-CoV-2	Severe axonal neuropathy MRI spine: Enhancement of caudal nerve root	AMAN	-IVIG- 2 cycles	Poor outcome; Neuromuscular respiratory failure, facial diplegia
(Toscano, Palmerini et al. 2020)[13]	76-year male, presented with lumbar pain, lower extremity weakness and loss of smell. In four days become flaccid areflexic tetraparetic. COVID-19 related symptoms 5 days prior to onset of neurological symptoms.	CSF protein: Normal, no cell. CSF: negative PCR for SARs-CoV-2	No electrodiagnostic data.	AIDP	-IVIG	Some motor improvement, unable to stand after 1 month

(Toscano, Palmerini et al. 2020)[13]	61-year male, presented with difficulty in climbing stairs, lower limb paresthesia, over one day pt. unable to stand. On exam, generalized areflexic, paraparetic. On 2nd day of IVIG developed tetraparesis, dysphagia, and facial weakness. On 3rd day, Neuromuscular respiratory failure. COVID-19 related symptoms 7 days prior onset of neurological symptoms .	Day 3: -Protein 40 mg/dl. -CSF WBC: 3/mm^3 CSF: negative PCR for SARs-CoV-2	Day 4: conduction block, demyelination.	AIDP	-IVIG -Plasmapheresis	Poor outcome: Developed bacterial pneumonia during IVIG, delayed Plasmapheresis
(Sedaghat and Karimi 2020)[14]	65-year male, presented with 5 days of acute progressive ascending quadriparesis. On exam, pt. was quadriplegic, facial diplegic, areflexic. Sensory loss to vibration and fine touch. COVID-19 related symptoms roughly 10 days prior to onset of neurological symptoms.	No CSF study available.	Decreased amplitude of CMAP, no sensory nerve action potential.	AMSAN	-IVIG -Hydroxychorloquine -Ritonavir, Lopinavir -Azithormycin	Unknown.
(Caamano and Beato 2020)[15]	61-year male, presented with liquid dipping on his right facial commissure which progressed to bifacial weakness. On exam, facial nerve palsy, absent blink reflex, good motor strength. COVID -19 symptoms 10 days prior to onset of neurological symptoms.	CSF protein: 44 mg/dl CSF WBC: 0. CSF: negative PCR for SARs-CoV-2	Electrodiagnostic not done	Facial diplegic variant	-Prednisone for two weeks.	No improvement.
(Gutierrez-Ortiz, Mendez et al. 2020)[16]	50-year male, presented with vertical diplopia, paresthesia around mouth. On exam, deep tendon reflex absent, planter flexor, right eye hypertropia, R eye intranuclear ophthalmoplegia, L eye nystagmus on L gaze. COVID-19 respiratory symptoms, along with ageusia, anosmia started 5 days prior to neurological symptoms.	CSF protein: 80 mg/dl GD1b-IgG positive CSF: negative PCR for SARs-CoV-2	Electrodiagnostic not done	Miller fisher variant	-IVIG	Ataxia and cranial nerve weakness improved, except ageusia and anosmia.
(Guierrez- Ortiz, Mendez et al. 2020)[16]	39-year male, presented with acute onset of diplopia. On exam, pt. had esotropia, severe abduction deficit in both eyes, fixation nystagmus, upper gaze more impaired, bilateral abducens palsy, absent deep tendon reflexes. Good motor strength, no sensory loss, no ataxia. COVID-19 related symptoms onset 3 days prior to onset of neurological symptoms.	CSF protein: 62 mg/dl CSF: negative PCR for SARs-CoV-2	Electrodiagnostic not done	Polyneuritis cranialis Varitant	No treatment, sent home.	Two weeks later complete recovery.

(Otmani, Moutawakil et al. 2020)[17]	70-year female, presented with progressive bilateral weakness and tingling resulting in total functional disability within 48 hours. On exam, by day 10, quadriplegic, hypotonic, areflexic. COVID-19 symptoms onset 13 days prior to onset of neurological symptoms.	CSF protein: 100mg/dL CSF: negative PCR for SARs-CoV-2	Marked reduction or absent of electrical potential in both sensory and motor nerve, little or no abnormality in conduction velocity.	AMSAN	IVIG Azithromycin Hydroxychloroquine	No improvement after one week.
(Camdessanche, Morel, et al.2020)[18]	64-year male, presented with paresthesia of hand and feet, over next 3 days developed flaccid tetraparesis. On exam, patient was MRC 2/5 in the legs, 2/5 arms, 3/5 in forearm, 4/5 in hands. Deep tendon reflexes absent, loss of vibration in lower limbs. COVID-19 symptoms onset 11 days prior to onset of neurological symptoms	CSF: Protein 166mg/dl.	Demyelination.	AIDP	-Lopinavir -Ritonavir	Not known
(Brooks, Megan 2020) [19]	54-year male, presented with ascending limb weakness and numbness. On exam deficit was quadriparesis and areflexia, burning dysesthesia, mild opthalmoparesis. COVID-19 related symptoms onset 2 weeks earlier to onset of neurological symptoms.	CSF study: Not reported	Demyelination	AIDP Dysautonomia	-IVIG	Was intubated for short period Motor strength outcome not known.