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A Case Report of Three Guillain-Barré Syndrome during SARS-CoV-2 Pandemic: Promising Outcome in a Patient with History of Multiple Sclerosis

Amir Ali Ghahremani[1], Amir Bigdeli[2], Davoud Salarbashi[3,4], Pouyan Shakouri[5], Sepideh Elyasi[6*]

[1]Department of Neurology, School of Medicine, North Khorasan University of Medical Sciences, Bojnord, Iran.

[2]Department of Nephrology, School of Medicine, North Khorasan University of Medical Sciences, Bojnord, Iran.

[3]Nanomedicine Research Center, School of Medicine, Gonabad University of Medical Sciences, Gonabad University of Medical Sciences, Gonabad, Iran.

[4]Department of Nutrition and Food Sciences, School of Medicine, Gonabad University of Medical Sciences, Gonabad, Iran.

[5]Student Research Committee, School of Medicine, North Khorasan University of Medical Sciences, Bojnord, Iran.

[6]Department of Clinical Pharmacy, School of Pharmacy, Mashhad University of Medical Sciences, Mashhad, Iran.

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ABSTRACT

Neurologic complications in patients with coronavirus disease 19 (COVID-19) are common particularly in hospitalized patients. Guillain–Barré syndrome (GBS) is a rare complication of COVID-19. Most patients with GBS and COVID-19 presented with progressive, ascending limb weakness evolving over one to four days. There are some reported cases of GBS, mostly in form of case reports or case series, and also a comprehensive review on this topic. However, none of these cases experienced multiple sclerosis (MS) as an underlying disease. So, in this manuscript we reported three cases of COVID-19 induced GBS; one of them with history of MS and promising outcome after receiving five sessions of plasmapheresis and corticosteroid therapy.

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Introduction

The pandemic of novel coronavirus infection emerged on 2019 December in Wuhan, China and soon spread worldwide, as severe acute respiratory coronavirus 2 (SARS-COV-2). Its associated disease, named coronavirus disease 19 (COVID-19). The first defined symptoms of infection were related to respiratory system, but as the time passed extra respiratory involvements became progressively evident. At first it proposed that the COVID-19 spares the nervous system but thereafter the neurologic complications in both central and peripheral nervous system were identified (1,2). CNS involvement ranges from encephalopathy to ischemic stroke (3). Peripheral nervous system is also affected by COVID-19, and several cases of acute polyneuropathy and had recently reported (1). The classic form is an immune-mediated acute-onset demyelinating polyradiculoneuropathy (acute inflammatory demyelinating polyneuropathy) classically presenting with ascending weakness, loss of deep tendon reflexes, sensory deficits and autonomic disorder. Diagnosis of Guillain–Barré syndrome (GB) is made based on clinical presentation, and electrophysiological, and cerebrospinal fluid (CSF) examinations (classically albuminocytological dissociation) (4). Here, the authors report three cases of an acute and severe peripheral polyneuropathy associated with SARS-COV-2 infection; one of them is the first reported case in patient with a history of multiple sclerosis.

Case presentation

A 66-years- old male patient was admitted to the emergency department, with symptoms of low grade fever, cough, dyspnea and weakness. He experienced these symptoms from 12 days before admission that had worsened since 3 days prior to hospitalization in March 2020. No past medical and drug history was reported by the patient. On

*Corresponding Author: Dr Sepideh Elyasi
Address: Department of Clinical Pharmacy, School of Pharmacy, Mashhad University of Medical Sciences, Mashhad, Iran. Tel: +98 5131801588.
Email: ElyasiS@mums.ac.ir

physical examination, the temperature was 37.8°C with blood pressure 135/90 mm/Hg, heart rate 98 beats/minute, respiratory rate 19/minute, and oxygen saturation of 82% on room air and the patient was conscious. He was diagnosed with COVID-19 after examining oropharyngeal sampling, and chest computer tomography (CT). Lung CT showed diffused consolidations and ground-glass opacities in both lungs. Reverse transcription-polymerase chain reaction (RT-PCR) for COVID-19 was positive and the patient was treated with hydroxychloroquine, ceftriaxone, azithromycin and dexamethasone. At second day of admission the arterial O2 saturation reduced to 79% and he transferred to intensive care unit. Remdisivir and interferon beta were administered to him. On fifth day of admission, he had tingling sensation in his legs and acute progressive symmetric ascending quadriparesis. He had no urinary and fecal incontinence. His thoracic and lumbosacral MRI was normal. His blood pressure reached 181/80 mmHg. The muscle strength examination showed weakness in four limbs with a Medical Research Council (MRC) scale of 4/5 in proximal, 4/5 in distal of the upper extremities and 2/5 in proximal, 3/5 in distal of the lower extremities. Deep tendon reflexes were absent generally. There was a reduction in the vibration and fine touch sensation distal to the ankle joints and also bifacial nerve palsy (House–Brackmann grade 3). He had no spine sensory level. Meningeal irritation signs and upper motor neuron disorder signs were negative. The laboratory examination results were follows: serum glucose 132 mg/dL; creatinine 1.2 mg/dL; alanine aminotransferase 95 IU/L; aspartate aminotransferase 115 IU/L; sodium 136 mmol/L; potassium 4.2 mmol/L; white blood cell count 25,500 cells per microliter (neutrophils = 93%; lymphocytes = 4%); Erythrocyte sedimentation rate 70 mm/hour, C-reactive protein 108 mg/L, and hemoglobin 14.3 g/ dL. The neurophysiological study was performed on day 6. Electro diagnostic parameters demonstrated decreased amplitude at compound muscle action potential and no response at sensory nerve action potential and absent F-responses. Electromyography showed decreased recruitment. These findings are consistent with acute axonal sensorimotor polyradiculoneuropathy (AMSAN). Plasmapheresis was ordered for the patient and ceftriaxone was replaced by meropenem. On day 8, despite abovementioned measures, after an unsuccessful CPR patient died.

The second case was a 62-years- old woman who admitted to the emergency department in August 2020, with the complaint of lower extremities muscular pain and weakness from 20 day ago. The symptoms began with paresthesia and progressed to generalized weakness. She also complained of recent constipation and weight loss. She had a splenectomy surgery five years ago because of the ITP. No other past medical and drug history was reported by the patient. She was addicted to inhaled opium. On physical examination, the temperature was 37.2°C with blood pressure 130/84 mm/Hg, heart rate 87 beats/minute, respiratory rate 19/minute, and oxygen saturation of 97% on room air and the patient was conscious. Cranial nerve examination was normal. The muscle strength examination showed weakness in four limbs with a Medical Research Council (MRC) scale of 3/5 in proximal, 4/5 in distal of the upper extremities and 4/5 in proximal, 4/5 in distal of the lower extremities. Deep tendon reflexes were reduced. The laboratory examination results were follows: serum glucose 149 mg/dL; blood urea nitrogen: 28 mg/dL; creatinine 0.9 mg/dL; alanine aminotransferase 24 IU/L; aspartate aminotransferase 45 IU/L; sodium 143 mEq/L; potassium 3.7 mEq/L; magnesium 2.4 mg/dL, calcium 9.6 mg/dL, white blood cell count 9100 cells per microliter (neutrophils = 59%); Platelet count 121000 cells per microliter, Erythrocyte sedimentation rate 11 mm/hour, C-reactive protein positive, and hemoglobin 14.5 g/dL. She was admitted in general surgery ward. On third day she transferred to neurology ward and electromyography (EMG) and nerve conduction velocity (NCV) performed for her. Absence of all sensory study and F responses with sural sparing and low amplitude of motor nerves were reported. Electro diagnostic finding was consistent with axonal sensorimotor polyradiculoneuropathy.

So, on fourth day of admission prednisolone tablet 50 mg/d and enoxaparin 60 mg SC administered to the patient. The platelet count reduced to 20000/µL. On 5th day, and intravenous Immunoglobulin (IVIG) introduced for her with daily dose of 250 mg and vial hydrocortisone 100 mg daily. The platelet count reached 8000/µL and BP was 180/110. On sixth day of admission the COVID-19 PCR reported positive with SaO2 of 97% without supplemental oxygen and respiratory rate of 19/minute. So she transferred to the COVID-19 ward. Kaletra® (lopinavir/ritonavir) with dose of two tablets per day were administered to her. After 9 day of hospitalization, she was discharged with normal force and without significant weakness, SaO2=94%, RR=18/min and platelet count=231000/µL.

The third case was a 42 years' woman with 10 years' history of multiple sclerosis (MS), who was under treatment with interferon and she was admitted with complaint of fever, weakness and myalgia from 5days ago. She was agitated and her vital signs were as follows: temperature 39°C with blood pressure 110/70 mm/Hg, heart rate 76 beats/minute, respiratory rate 12/minute, and oxygen saturation of 96% on room air and the patient was conscious. The laboratory examination results were follows: blood urea nitrogen: 167 mg/dL; creatinine 4.7 mg/dL; alanine aminotransferase 108 IU/L; aspartate aminotransferase 164 IU/L; total bilirubin 4.8 mg/dL, direct bilirubin 3.67 mg/dL, sodium 139 mEq/L; potassium 3.7 mEq/L, calcium 6 mg/dL, albumin 3.3 g/dL, LDH=1077 U/L, white blood cell count 9700 cells per microliter (neutrophils = 90%, lymphocyte=8%); Platelet

count 242000 cells per microliter, Erythrocyte sedimentation rate & C-reactive protein positive. Viral markers (anti-HCV, Hbc Ab total, HAV IgM, HTLV Ab and HIV), direct and indirect coombs and wrights and 2-ME were negative. The RT-PCR for COVID-19 was also performed which was negative. Abdominal and pelvic ultrasonography just showed increased echogenicity of the kidney parenchyma and slightly free abdominal fluid. No thrombosis was detected based on color Doppler sonography in hepatic veins. Diagnosis of acute kidney injury (AKI) due to rhabdomyolysis was proposed for patient and she was treated with fluid therapy and some doses of parenteral corticosteroid. She also received broad-spectrum antibiotic because of the fever. Her creatinine and urea reduced significantly after one week (creatinine 1.6 mg/dL, Urea 106 mg/dL), but liver enzymes and bilirubin had no meaningful change (AST 243 U/L, ALT 196 U/L, total/direct bilirubin 4.47/3.2 mg/dL) and patient had leukocytosis with left shift (26700 cells per microliter and Neutrophils 80% & lymphocytes 11%). RT-PCR for COVID-19 was done again because of high suspicious, which was positive but lung CT did not show any specific finding in consistent with COVID-19. She still complained of generalized weakness with lower extremities predominance and paraparesis. The muscle strength examination showed weakness in four limbs with a Medical Research Council (MRC) scale of 4/5 in proximal, 4/5 in distal of the upper extremities and 2/5 in proximal, 2/5 in distal of the lower extremities. The plantar reflex was normal (flexor). With doubt of polyneuropathy, EMG-NCV performed for her. The electrophysiological findings were consistent with an asymmetric sensorimotor polyneuropathy which is length dependent. After two weeks of treatment in COVID-19 ward and fever resolution, she still had quadriparesis and radicular pain. The second EMG-NCV reported a symmetric axonal sensorimotor polyradiculoneuropathy with no change in extremities forces in comparison with two weeks ago but foot drop was noted in right leg. Lumbar puncture was performed and increased protein but no WBC was reported. She completed a total of five sessions of plasma exchange every other day using 20% albumin and saline in a 60%/40% proportion with diagnosis of Guillain-Barré syndrome. After 4 sessions, right lower limb distal force increased significantly and upper limb force reached 5/5. After 10 days from the end of the plasma exchange sessions she was able to walk with use of walker and after 1 month without any help. Her laboratory also returned to normal values (urea=11 mg/dL, Creatinine=0.6 mg/dL, calcium 8.8 mg/dL, and normal liver function tests). She had full muscle force after 3-month follow-up. The summery of three cases' information is reported in Table 1 and the electrophysiological data are summarized in Table 1.

Table 1. Clinical characteristics and laboratory findings of three patients with Guillain-Barré syndrome after COVID-19.

	Case 1	Case 2	Case 3
Age/gender	66/Male	62/Female	42/Female
Comorbidities	None	Idiopathic thrombocytopenic purpura	Multiple sclerosis
Symptoms of COVID-19	Low grade fever, cough, dyspnea and weakness	Weakness	Fever, weakness and myalgia
Method of COVID-19 Dx	Lung CT, RT-PCR	RT-PCR	RT-PCR/ clear Lung CT
Neurological signs & symptoms	Tingling sensation in his legs and acute progressive symmetric ascending quadriparesis, weakness in in four limbs	Began with paresthesia and progressed to generalized weakness	Generalized weakness with lower extremities predominance and paraparesis
Time of neurological symptoms onset (days)	17	No other symptoms before neurological ones	12
Urinary and fecal incontinence	No	No	No
CSF analysis	No LP	No LP	Increased protein but no WBC
Serum studies	WBC 25500 cells/mm3; Lymphocytes 4% & neutrophils 93%, Normal kidney function; rise of transaminase levels	WBC 9100 cells/mm^3; (neutrophils = 59%); Platelet count 121000 cells/mm^3; ESR 11 mm/hour, CRP+;; normal kidney & liver function	WBC 9700 cells/mm^3 (neutrophils = 90%, lymphocyte=8%); Platelet count 242000 cells/mm3; rise of Scr (4.7 mg/dL) and abnormal liver function tests; calcium 6 mg/dL, albumin 3.3 g/dL; LDH=1077 U/L
MRI results	Thoracic and lumbosacral: NL	-	-
Treatment	Plasmapheresis (1 session) + steroid	4 cycle of IVIG (day 5-8) + steroid	Plasmapheresis (5 sessions) + steroid
Clinical outcome	Death (after 8d)	Discharge after 9d with normal force and without significant weakness (normal walking after 3m follow-up)	After 10 days from the end of the plasma exchange sessions she was able to walk with use of walker and after 1 month without any help

CSF: Cerebro spinal fluid, CT:Computed tomography, CRP: C reactive protein, Dx: Diagnosis, ESR: Erythrocyte sedimentation rate, IVIG: Intravenous immunoglubin, LP:lumbar puncture, MRI: Magnetic resonance imaging, RT-PCR: Reverse transcription polymerase chain reaction, Scr: Serum creatinine

Discussion

Guillain–Barré syndrome is an autoimmune process that affects peripheral nerves, and commonly manifests as demyelinating neuropathy with ascending paresthesia and weakness (5). The precise etiology and pathophysiology of GB is not well defined, but it usually is preceded by a respiratory or gastrointestinal infection. Campylobacter jejuni, Epstein–Barr virus, cytomegalovirus, and Zika virus are the most reported microbes causing GB (6). However, various other viral infections like Coronaviridae family viruses could also be in association with GB syndrome. During 2012 outbreak of Middle East respiratory syndrome (MERS) caused by MERS coronavirus (CoV) that shares many similar features with SARS-COV-2 a few cases of Guillain–Barré syndrome had been reported (7).

Considering these knowledge about previous viral epidemics we expected increasing number of reports on involvement of peripheral nervous system especially peripheral neuropathy in COVID-19 pandemics. The first case of Guillain-Barré syndrome associated with SARS-CoV-2 infection has perhaps been reported by Zhao et al. who reported the GBS in a 61 years old woman which later diagnosed as COVID-19 (8). Following this case other cases have been reported. It seems that abnormal immune response to infections triggered the peripheral nerve damage, in some cases intermediated by the production of autoreactive antibodies (anti-ganglioside antibodies) (4). Actually, two probable mechanisms are involved in COVID-19 peripheral (PNS) and central nervous system (CNS) damage: 1) high tendency of the virus to PNS and CNS resulting in hematogenous (infection of endothelial cells or leucocytes) or trans-neuronal (via olfactory tract or other cranial nerves) COVID-19 dissemination to these systems; It is responsible for the most common neurological COVID-19 symptoms (e.g., hypogeusia, hyposmia, headache, vertigo, and dizziness), and 2) abnormal immune-mediated response causing secondary neurological involvement which can lead to severe complications during or after the course of the illness, either dysimmune (e.g., myelitis, encephalitis, GBS) or induced by cytokine overproduction (hypercoagulable state and cerebrovascular events) (4).

In this case report we presented three patients who experienced GB after PCR confirmed COVID-19 infection. A systematic review by Abu-Rumeileh et al., summarized 73 cases of GBS in COVID-19 patients published in 52 articles. The mean age of patients was 55 years and most of them were men (4). The mean age of these three cases also was 56.67 years which is close to Abu-Rmeileh report. The mean age of patients with GBS mainly overlapped that of classic COVID-19 subject. However, the sex distribution of these three patients was different from Abu-Rumeileh et al., report (66.7% female) (4). They mentioned than 68.5% of reported GB cases were male. This finding may also reflect the gender epidemiology of SARS-CoV-2 resulting from higher circulating Angiotensin-Converting-Enzyme 2 (ACE2) levels -the cellular receptor for SARS-CoV-2- in men (9). Higher prevalence of Zika virus–GB in male is also in consistent with COVID-19-GB (10).

After Italy with 20 reported cases, Iran is at second place with 10 GB cases (without considering these 3 ones). Variable comorbidities were reported in these 73 cases with no prevalence of a particular disease (4). Multiple sclerosis had been reported in none of them. However, one of our cases who had complete recovery from GB had MS as past medical history and the other one had the history of ITP. The autoimmune nature of these past medical histories may be important as a triggering factor. In previous reported cases, other kind of autoimmune diseases like rheumatoid arthritis and psoriasis expressed in some limited cases (4).

All reported GB cases (n=72) except two were symptomatic for COVID-19 with various severity. Fever (73.6%, 53/72) and cough (72.2%, 52/72) were the most common reported manifestation (4). In our three reported cases, two patients were symptomatic and had fever and cough as mentioned above but one patient did not have any other complains. However, the RT-PCR for COVID-19 was positive in all of them. Six patients out of 73 reported cases had negative results in Abu-Rumeileh et al., report (4).

Just in four patients in Abu-Rumeileh et al., review (8, 11-13), GB presented before COVID-19 symptoms [median delay time of GB: 14, min 2–max 33 days], which is in consistent with our findings. Similar to these reported cases, common clinical manifestations at onset included sensory symptoms (72.2%, 52/72) alone or in combination with paraparesis or tetraparesis (65.2%, 47/72). Moreover, all cases but one (14) showed lower limbs or generalized areflexia, like our cases which had reduced or absent DTR. From the clinical point of view, most examined patients presented with a classic sensorimotor variant (70.0%, 51/73) (4) as we found in these three cases. In the evaluated population, 81.8% of patients completed electro-physiological criteria for acute inflammatory demyelinating polyneuropathy (AIDP) (45/55), 12.7% (7/55) for AMSAN, and 5.4% (3/55) for acute motor axonal neuropathy (AMAN) subtypes. Electrophysiological subtype was not definite in 18 patients due to the lack of enough information (4). Three reported cases in this article all had diagnosis of AMSAN.

The diagnosis of GBS was recognized based on clinical, CSF, and electrophysiological findings in 44/73 (60.3%) patients, which is the highest level of diagnostic certainty. In other cases, the diagnosis defined based on clinical, and electrophysiological data in 18/73 (24.7%) cases, clinical, and CSF data in 8/73 (11.0%), and only clinical findings in 3/73 (4.1%) patients (4). In these three cases just one patient diagnosed based on clinical, CSF, and electrophysiological

findings and for the other two patients we just had clinical and electrophysiological information because of the fast progressing nature of disorder in them. The results of CSF analysis are similar to typical neurochemical findings in non-COVID-19 GB and elevated CSF protein and pleocytosis was the main finding in most patients (4). We also found a high protein level and a near normal WBC count in the third case that underwent LP. Besides, the mostly normal cell count, and the absence of SARS-CoV-2 RNA in all tested CSF samples, makes a direct invasion from SARS-CoV-2 into the nerve roots with intrathecal viral replication less probable (4).

Seventy reported cases were treated with intravenous immunoglobulin (IVIG). Plasma exchange and steroid therapy were performed in ten (four of them received also IVIG) and two patients, respectively. In two patients, no therapy was given (4). In our three reported cases all of them received steroid, one of them IVIG and two patients' plasma exchange. The latency period between COVID-19 symptoms and GB clinical improvement after IVIG therapy, can support important role of post-infectious immune-mediated mechanism in COVID-19 induced GB (4). For confirmation of this hypothesis, blood inflammatory markers (e.g., CRP, IL-6, TNF-α, IL-1, etc.) level measurement in COVID-19 patients experiencing GBS is recommended.

Mechanical or non-invasive ventilation was implemented in 21.4% (15/70) and 7.1% (5/70) patients due to worsening of GB or COVID-19, respectively. Between these three cases, just the first case was intubated and unfortunately died less than 24 hours after connection to mechanical ventilation. Most of reported cases [72.1% (49/68)] demonstrated clinical improvement with partial or complete remission, 10.3% (7/68) cases showed no improvement, 11.8% (8/68) still required critical care treatment, and 5.8% (4/68) died (4). Between these three patients that we reported, one of them died, and the other two patients had near to complete remission after 3 months' follow-up. It is especially interesting in the patient with previous history of MS. She completely recovered after 5 sessions of plasma exchange and her liver and kidney function are also normalized. The second case with history of ITP recovered after IVIG therapy.

It is worth to mentioned that, patients with no improvement or poor outcome in Abu-Rumeileh et al., review (n=19) showed a slightly higher but not significant frequency of clinical history and/or a radiological picture of COVID-19 pneumonia (14/19, 73.7%) compared to those with a favorable prognosis (29/48, 60.4%, p = 0.541) (4) which is in line with our finding in these three cases. The only patient out of three cases, who died, had involvement in lung CT. Moreover, the earlier group of patients was significantly older (mean 62.7±17.8 years, p=0.011) (4). The case that died in our center also was older than the other two patients (age 66 y).

In conclusion, GB is an infrequent but probable neurological complication of COVID-19. Considering high infectivity of COVID-19 infection in pandemic era it is recommended to assess all patients with GB presentation for SARS-CoV2 infection as a triggering factor.

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