Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

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COVID-19 infection presented as Guillain-Barre Syndrome: Report of two new cases and review of 116 reported cases and case series

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ABSTRACT

Background: /Aims: Corona virus disease 2019 (COVID 19) is a pandemic infectious disease of 2020, which often presents with respiratory and gastrointestinal symptoms. The behavior of the virus and its full clinical picture has not been fully studied yet. Many case reports and case series have been running in order to elaborate different presentations and associations. Pulmonary and gastrointestinal features of COVID-19 infection are well outlined; however, neurological manifestations are less defined.

Case presentation: We report two adult cases of COVID-19 infection presented with acute Guillain-Barre Syndrome (GBS), and a literature review on the causal association between COVID-19 and GBS.

Conclusion: Our two case reports in addition to literature review of 116 published cases may help offer insight into the clinical course of COVID-19 infection. Our two COVID-19 patients presented with neurological manifestations of GBS which were not preceded with any respiratory, gastrointestinal or other systemic infection. This leads us to raise the possibility of establish direct causal association between COVID-19 infection and GBS. Physicians should have high clinical suspicions when encounter GBS patient during the current COVID-19 pandemic and consider co-existence of COVID-19 infection that may warrant SARS-CoV-2 testing, isolation precautions, and specific treatment for Covid-19 infection.

1. Introduction

The novel corona virus (COVID-19) or severe acute respiratory syndrome corona virus 2 (SARS-CoV-2) is a new virus emerged in Wuhan, China, and became a new public health pandemic affecting the whole globe. COVID-19 presents commonly with constitutional symptoms including fever and fatigability, and can progress to affect the lung parenchyma causing serious complication such as pneumonia and sever acute respiratory distress [1]. Extra-pulmonary manifestations of COVID-19 have also been reported including neurological, cardiovascular, hematological, renal, dermatologic and gastrointestinal manifestations. Common neurological symptoms of COVID-19 are headache, dizziness, dysgeusia/ageusia, hyposmia/anosmia, seizure, stroke, and encephalopathy [2]. There have been reported cases of Guillain-Barré Syndrome (GBS) associated with COVID-19 from different parts of the world [[3–50],[51–93]]. Moreover, a number of systematic reviews for some of these cases have also been reported [94–100].

GBS is an autoimmune demyelinating disease that presents with symmetric progressive distal weakness that moves proximally [101]. It is usually preceded by some bacterial and viral infectious agents that may induce autoimmune reaction against myelin mediated via molecular mimicry mechanism [101].

In this report, we describe two COVID-19 infected adult patients presented as acute inflammatory demyelinating polyneuropathy (AIDP) and acute motor sensory axonal neuropathy (AMSAN) sub-types of GBS, respectively. Furthermore, we reviewed 116 published cases and case

Abbreviations: COVID-19, Coronavirus disease 2019; SARS-CoV-2, Severe acute respiratory syndrome coronavirus 2; CT, Computed tomography; GBS, Guillain-Barre Syndrome; MFS, Miller Fisher Syndrome; AIDP, Acute inflammatory demyelinating polyradiculoneuropathy; AMSAN, Acute motor and sensory axonal neuropathy; AMAN, Acute motor axonal neuropathy; BFP, Bifacial weakness with paresthesias.

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series in the literature on the causal association between COVID-19 and GBS to summarize this information for clinicians into a single review.

1.1. Case 1

A 49-year old male patient was admitted to the hospital with symptoms of acute progressive symmetrical ascending weakness. The weakness began at the distal lower extremities 5 days prior to presenting to hospital. Over these preceding days there was gradually worsening of the symptoms. Two days before the symptoms started the patient had experienced sore throat and fatigability. He denied having cough, shortness of breath, diarrhea or gastroenteritis-like symptoms. Upon arrival to the Emergency Department, the patient tested positive for COVID-19 with CT-value of 32.7 by nasopharyngeal swab on reverse transcription polymerase chain reaction (RT-PCR) assay. As the following chest X-ray as well as chest CT-scan were unremarkable, there was no indication to start any antiviral regimen. His past medical history is significant for hypertension, treated by nifedipine.

On physical examination his vital signs showed he was afebrile, the blood pressure was 130/90 mmHg, and his heart rate was 77 beats per minute, saturating 98% oxygen on room air with a respiratory rate of 18 breaths per minute and normal body mass index. On general inspection he was conscious, alert and in no apparent distress. Chest and cardiovascular examination was unremarkable. Neurological examination revealed intact cranial nerves; muscle examination showed 3/5 power in distal lower limbs and 4/5 power in proximal muscles. In the distal upper extremities the power was 4/5. The deep tendon reflexes were absent bilaterally with reduced sensation to pain and light-touch in the distal lower and upper limbs. There were no meningeal signs elicited. Cortical functions were preserved. Laboratory investigations showed a white blood count (WBC) of 14.5 × 103/u (normal, 4 10) with neutrophils 55.5% and lymphocytes 32.3%, and Hemoglobin of 16.1 gm/dL (normal, 13–17). The C-reactive protein (CRP), kidney and liver parameters were within normal ranges. Serum ferritin of 652 ug/L (normal 30–490), Interleukin-6 (IL-6) of 4 pg/ml (normal <5.9) and D-dimer of 0.46 mg/L (normal 0–0.49). Viral serology including HIV tests were negative. A head CT-scan showed no acute intracranial pathology, mass-effect or midline shift. Lumbar puncture was performed and cerebrospinal fluid (CSF) analysis showed protein-cellular dissociation with high protein level of 1.04 gm/L (normal 0.15–0.45) and normal WBCs of 1/μL (normal 0–5). Chest X-ray was unremarkable. Bilateral tibial motor nerve conduction study (NCS) showed dispersed motor action potentials with slow conduction velocity (CV) mainly from left side and decreased compound motor action potential (CMAPs) amplitude along with absent F waves and H reflexes. Bilateral peritoneal motor NCS showed slow CVs mainly from left side with minimal reduced proximal CMAPs amplitude with absent left F wave and present from right side. Right median motor NCS showed reduced CMAP amplitude in comparison with left side, mild slow CV and mild prolonged F wave latency. Bilateral ulnar motor NCS showed normal Distal Latency (DMLs), normal CVs but slightly reduced proximal CMAPs and prolonged F waves. Bilateral sural and superficial peroneal nerves were normal. The findings are consistent with acute inflammatory demyelinating polyneuropathy (AIDP). The patient was treated with intravenous immunoglobulin (0.4 g per kg body weight per day) for 5 days. Infectious disease team consulted and, as per our hospital protocol for COVID-19 infection, agreed not to start COVID specific treatment since the patient was asymptomatic with CT value > 30 of COVID-19 RT-PCR. Patient showed significant improvement in his power with complete recovery after 6 weeks of follow up.

1.2. Case 2

A 43-year old male not known to have any past medical history, presented with acute onset of progressive ascending weakness over the last 7 days prior to admission. The weakness was associated with paresthesia involving the feet and hands. Patient denied having sensory loss, bulbar symptoms, visual disturbance or incardination. Patient mentioned having sore throat 3 days before the start of symptoms improving with over the counter gurgling solution. Patient denied having fever, cough, shortness of breath, diarrhea or gastroenteritis-like symptoms. Upon presentation to the hospital patient was afebrile and vitally stable. Patient had no cervical lymphadenopathy. Chest and cardiovascular examination was unremarkable. Neurological examination showed power of 4/5 in the all 4 limbs, generalized areflexia, planters downgoing. He had no cranial nerves involvement, sensory deficit or coordination impairment. Laboratory investigation upon admission showed WBC of 9.6 × 103/u (normal 4–10 × 10^3/u) with lymphopenia of 0.9 × 103/u (normal 1–3x103/u). Kidney and liver parameters were unremarkable. CRP was 13.4 mg/L (normal 0–5), ferritin, procalcitonin, D-dimer as well as IL-6 levels were within normal limits. CSF analysis showed protein-cellular dissociation with high protein level of 0.73 gm/L (normal 0.15–0.45) and normal WBCs of 2/μL (normal 0–5). Chest X ray was unremarkable. HIV test was negative. NCS of right median nerve showed undetectable sensory response, prolongation of distal motor latency, significant reduction of motor conduction velocity and undetectable F waves. Left median nerve showed prolongation of DML, preserved motor conduction velocity with some reduction in amplitude with dispersion from elbow stimulation, and delayed F waves. Left median sensory showed delayed latencies and mildly reduced sensory conduction velocity. Right ulnar showed preserved DML, sensory response, amplitude and CV but F wave was undetectable. Left ulnar showed similar findings. Lower limbs sensory responses were normal. Lower limbs motor studies showed reduced left tibial amplitude with dispersion and prolonged DML. Right tibial motor study was unremarkable. Peroneal study showed prolongation of DML and reduction in CV. The findings were consistent with acute motor and sensory polyneuropathy (AMSAN). On second day of admission, the COVID-19 RT-PCR assay obtained by nasopharyngeal swab and the patient tested positive with CT-value of 32. Treatment of the patient was initiated with intravenous immunoglobulin (0.4 g per kg body weight per day) for total 5 days. Infectious disease team consulted and, as per our hospital protocol for COVID-19 infection, agreed not to start COVID treatment since the patient was asymptomatic with CT value > 30 of COVID-19 RTPCR. Patient showed significant improvement in his power and discharged with complete recovery.

2. Discussion

GBS is an acute monophasic paralyzing illness, usually provoked by a preceding infection. It occurs world-wide and all age groups are affected [101]. Several mechanisms have been proposed in pathogenesis of GBS [102]. One proposed mechanism for GBS is that an antecedent infection evokes an immune response, which in turn cross-reacts with peripheral nerve components because of the sharing of cross-reactive epitopes (molecular mimicry). The end result is an acute polyneuropathy. This immune response can be directed towards the myelin or the axon of peripheral nerve [103]. Second proposed mechanism is immune reactions directed against epitopes in Schwann cell surface membrane or myelin can cause AIDP. Both the cellular and humoral immune responses participate in the process. Invasion by activated T-cells is followed by macrophage-mediated demyelination with evidence of complement and immunoglobulin deposition on myelin and Schwann cells. Third proposed mechanism is immune reactions against epitopes contained in the axonal membrane cause the acute axonal forms of GBS: AMAN and AMSAN [102]. The pathophysiology of these variants is better understood than that of AIDP [104]. Other subtypes of GBS include Miller Fisher syndrome (MFS) and Bickerstaff encephalitis that associated with anti-GQ1b antibodies and considered as overlapping variants of the anti-GQ1b antibody syndrome [105].

Herein, we described two cases of acute progressive symmetric ascending neuropathy in concomitant with COVID-19 infection with diagnosis of AIDP and AMSAN sub-types of GBS, respectively. The time

period from symptoms and signs of acute viral infections to GBS clinical features onset is typically several days to a few weeks. Although it may not be typical, a short duration between febrile illness onset to symptom worsening has been reported [87,96]. Our two patients presented with neurological manifestations of GBS which were not preceded with any respiratory, gastrointestinal or other systemic infection related to COVID-19. This may suggest presence of direct causal association between GBS and COVID-19 infection. The clinical presentation and work up findings suggest to a possible etiological association with COVID-19 infection, in line with emerged data of the literature review.

Guillain-Barré syndrome (GBS) occurs with an approximate incidence of 0.16–3 cases per 100,000 annually in the general population [106], however, an accurate estimation of the incidence of GBS in COVID-19 patients is unknown, as a potential association remains uncertain. After thorough literature review, we found a substantial number of reported cases and case series of Covid-19 infection presented with GBS. Table 1 summarizes demographic, type of relationship with Covid-19, clinical and GBS sub-types, response to specific GBS treatment, and outcome of different GBS cases who presented in concurrent with Covid 19 infection [[3–50],[51–93]]. The findings may suggest that such association seems to be not uncommon condition with similar clinical and neurophysiological study patterns. In addition to our two reported cases, a total of 116 case reports of Covid-19 patients (aged 7–94 years) diagnosed with GBS in 23 different countries were included in this review. The mean age at onset in patients with GBS generally overlapped that of Covid-19 individuals [107]. Nevertheless, GBS cases with Covid-19 in pediatric and adolescent age group have been increasingly described [24,25,36,46,62], suggesting that, with the prolongation of Covid-19 pandemic spreading, a wider age range might be affected.

Although both genders were affected, male predominant (67%, 79/118) was observed in this cohort, reflecting the gender epidemiology of Covid-19. In this regard, males show a worse Covid-19 prognosis (64.3%) compared to the females, likely due to a commonly shorter life expectancy or to higher circulating Angiotensin-Converting-Enzyme 2 levels, the cellular receptor for SARS-CoV-2, in males compared to females [96].

GBS is typically a post-infectious condition; approximately two-thirds of GBS cases give a history of an antecedent respiratory tract or gastrointestinal infection [108]. Infections are thought to trigger the immune response that leads to acute polyneuropathy. Examples of infections that can trigger GBS include campylobacter, HIV and less commonly hepatitis A, B, C, and E, herpes simplex virus, herpes zoster virus, chikungunya virus, and the bacteria Haemophilus influenzae, *Escherichia coli*, and Mycoplasma pneumonia [106]. Although most of reported cases in this cohort developed neurological symptoms of GBS with a typical latency after Covid-19 infection, our two patients in addition to few other cases [7,62,66,67,90,91] appeared to have a para-infectious profile, as described in infection with Zika virus [109]. The immense production of cytokines in SARS-CoV-2 may contribute to the intensification of the autoimmune process underlying GBS [110].

Our analysis showed 64 patients (60%) were diagnosed with AIDP, 18 (17.1%) with AMSAN, 11 (10.5%) with AMAN, nine (8.6%) with MFS, and four (3.8%) with bifacial weakness with paresthesias (BFP). In 12 patients (11%) the subtype of GBS was not specified. The distribution of GBS electrophysiological variants in this cohort of Covid 19-associated GBS manifests prevalently with AIDP followed with AMSAN and AMAN, consistent with classic GBS [87,94]. The distribution of clinical subtypes of GBS confirming the predominance of the sensorimotor variant compared to MFS and other rare subtypes.

A total of 89 patients received intravenous immunoglobulins (IVIg), 10 patients received plasmapheresis, and eight patients received combination of both therapeutic modalities. Combination of IVIg and Methylprednisolone was used in four patients and in two of Spanish patients received only prednisolone, one of them had poor outcome [16,44]. In five patients no specific treatment was provided. One patient with MFS remained without specific therapy and recovered spontaneously [28], and Likewise two other patients with AIDP [14,30]. A total of 16 patients required artificial ventilation.

Although it was difficult to distinguish respiratory failure and the need for artificial ventilation due to GBS from that due to COVID-19-related lung disease, in most reported patients respiratory insufficiency was attributed to the GBS and not to the COVID-19 infection. The outcome was poor in 35 patients and seven died during hospitalization.

The strength of the present study is the inclusion of high number of published GBS associated-Covid-19 cases or case series (118 cases). Moreover, it includes cases reported during Covid-19 highest spreading in the first wave as well as second wave of the Covid 19 pandemic, in which different strains of Covid-19 have been identified. However, our study is limited by the possibility of occurrence of selection bias, given that most reported cases or case series have been described in Europe, Canada and USA (81 out of 118). Another potential weakness of the present cohort is the different subtypes of GBS presented, along with current thinking that GBS refers to a heterogenous collection of conditions, with different pathogeneses. A further limitation is lack of screening which exclude concomitant infection by other viral or bacterial organisms known to be associated with GBS in some of the published cases. In fact, in two case reports, GBS occurred after coinfection by Campylobacter jejuni and Covid-19 [74,89]. However, due to the temporal association we believe this possibility is unlikely.

3. Conclusion

Our two case reports in addition to literature review of 116 published cases may help offer insight into the clinical course of COVID-19 infection. We shared our experience with two COVID-19 patients who presented with neurological manifestations of GBS which were not preceded with any respiratory, gastrointestinal or other systemic infection. This leads us to raise the possibility of establish direct causal association between COVID-19 infection and GBS. However, future reports and extensive epidemiological studies are needed to ascertain the nature of this relationship. Moreover, physicians should have high clinical suspicions when encounter GBS patient during the current COVID-19 pandemic and consider co-existence of COVID-19 infection that may warrant SARS-CoV-2 testing, isolation precautions, and specific treatment for Covid-19 infection.

Ethical approval

The institutional approval for this study was obtained from the Medical Research Center of Hamad Medical Corporation, Qatar (MRC-04-21-291).

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Conflicts of interest

The authors declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Author contribution

Contributors MO, MAA, AA and A-NE were all involved in the patients care. A-NE viewed the importance of the cases and formed the research team. MO and MAA obtained consent from the patients. MO, MAA, AA, and A-NE wrote the initial manuscript. A-NE reviewed the manuscript and updated the final version. All authors critically reviewed the initial and the final draft of the manuscript and approved it for submission.

Table 1
Summary of demographic, clinical, main treatment and outcome of reported cases and case series to date on Guillain-Barré syndrome (GBS) in COVID-19 patients.

Authors	Country	No. Of cases	Age (yrs)/ gender	GBS diagnosis Method	GBS subtype	GBS-Covid-19 temporal relation	GBS Treatment	Outcome
Aasafara J et al.	Morocco	1	36/F	Clinical/CSF/ NCS	BFP	Post-infectious	IVIg (5 days)	Favorable
Abbaslou MA et al.	Iran	1	55/F	Clinical/CSF/ NCS	AMSAN	Post-infectious	IVIg (duration NR)	Died
Abolmaali M et al.	Iran	3	88/F	Clinical/CSF/ NCS	AMSAN	Para-infectious	PE (6 sessions)	Favorable
			47/M		AMSAN	Post-infectious	PE (2 sessions)	Died
			58/M	Clinical/CSF/ NCS Clinical/CSF/ NCS	AMSAN	Post-infectious	PE (sessions number NR)	Died
Abrams RMC et al.	USA	1	67/F	Clinical/CSF	NR	Post-infectious	PE (5 sessions)	Poor
Agosti E et al.	Italy	1	68/M	Clinical/CSF/ NCS	AIDP	Post-infectious	IVIg (5 days)	Favorable
Alberti P et al.	Italy	1	71/M	Clinical/CSF/ NCS	AIDP	Post-infectious	IVIg (5 days)	Died
Ansari B et al.	Iran	1	59/F	Clinical/NCS	AIDP	Post-infectious	PE (sessions number NR)	Favorable
Arnaud S et al.	France	1	64/M	Clinical/CSF/ NCS	AIDP	Post-infectious	IVIg (5 days)	Favorable
Assini A et al.	Italy	2	55/M	Clinical/CSF/ NCS	MFS	Post-infectious	IVIg (5 days)	Favorable
			60/M	Clinical/CSF/ NCS	AMSAN	Post-infectious	IVIg (5 days)	Favorable
Atakla HG et al.	Guinea	1	41/M	Clinical/CSF/ NCS	AIDP	Post-infectious	IVIg (5 days)	Favorable
Bigaut K et al.	France	2	48/M	Clinical/CSF/ NCS	AIDP	Post-infectious	IVIg (5 days)	Favorable
			70/F	Clinical/CSF/ NCS	AIDP	Post-infectious	IVIg (5 days)	Favorable
Bracaglia M et al.	Italy	1	66/F	Clinical/NCS	AMSAN	Para-infectious	IVIg (5 days)	Favorable
Börü ÜT. Et al.	Turkey	1	35/M	Clinical/CSF/ NCS	AIDP	Post-infectious	No specific treatment	Favorable
Bueso T et al.	USA	1	60/F	Clinical/CSF	NR	Post-infectious	IVIg (5 days)	Favorable
Caamaño DSJ et al.	Spain	1	61/M	Clinical/CSF/ MRI	BFP	Post-infectious	Oral prednisolone	Favorable
Camdessanche JP et al.	France	1	64/M	Clinical/CSF/ NCS	AIDP	Post-infectious	IVIg (5 days)	Poor
Chakraborty N et al.	India	1	75/M	Clinical/CSF/ NCS	AMAN	Para-infectious	IVIg (5 days)	Favorable (ICU)
Chan JL et al.	Canada	1	58/M	Clinical/CSF/ NCS	AIDP	Post-infectious	IVIg (5 days)	Favorable
Chan M et al.	USA	2	68/M	Clinical/CSF/ MRI	AIDP	Post-infectious	PE (5 sessions)	Favorable
			84/M	Clinical/CSF	NR	Post-infectious	IVIG & PE (5 sessions)	Poor
Coen M et al.	Switzerland	1	70/M	Clinical/CSF/ NCS	AIDP	Post-infectious	IVIG (5 days)	Favorable
Colonna S et al.	Italy	1	62/M	Clinical/CSF/ NCS	AIDP	Post-infectious	IVIG (5 days)	Poor
Curtis M et al.	USA	1	8/M	Clinical/CSF/ MRI/NCS	AIDP	Post-infectious	IVIG (2 days)	Poor
Das KY et al.	India	1	7/M	Clinical/CSF/ NCS	NR	Post-infectious	IVIg (duration NR)	Favorable
Defabio AC et al.	USA	1	70/F	Clinical/CSF	NR	Post-infectious	IVIg (duration NR)	Favorable
Diez-Porras L et al.	Spain	1	54/M	Clinical/CSF/ NCS	AIDP	Post-infectious	IVIg (1 day)	Poor (ICU)
Dinkin M et al.	USA	2	36/M	Clinical/MRI	MFS	Post-infectious	IVIG (3 days)	Favorable
			71/F	Clinical/MRI	MFS	Post-infectious	None- specific	Favorable
Dufour C et al.	USA	1	36/F	Clinical/CSF	NR	Post-infectious	IVIG (5 days)	Favorable
Ebrahimzadeh SA et al.	Iran	2	46/M	Clinical/CSF/ NCS	AIDP	Post-infectious	No specific therapy	Favorable
			65/M	Clinical/CSF/ NCS	AIDP	Post-infectious	IVIg (duration NR)	Favorable
Elkouly et al.	USA	1	75/M	Clinical/CSF	NR	Post-infectious	IVIG (5 days), IV steroids	Favorable
El-Otmani H et al.	Morocco	1	70/F	Clinical/CSF/ NCS	AMSAN	Post-infectious	IVIG (5 days)	Poor
Esteban M et al.	Spain	1	55/M	Clinical/NCS	AIDP	Post-infectious	IVIG (5 days)	Poor
Farzia MA et al.	Iran	1	41/M	Clinical/NCS	AIDP	Post-infectious	IVIG (5 days)	Favorable
Fernández-Domínguez et al.	Spain	1	74/M	Clinical/CSF	MFS	Post-infectious	IVIG (5 days)	Favorable
Frank CHM et al.	Brazil	1	15/M	Clinical/CSF/ NCS	AMAN	Post-infectious	IVIG (5 days)	Favorable
Gale A et al.	UK	1	58/M	Clinical/CSF/ NCS	AIDP	Post-infectious	IVIG (5 days)	Poor
Galán AV et al.	Spain	1	43/M	Clinical/NCS	AIDP	Post-infectious	IVIG (5 days)	Favorable

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Table 1 (*continued*)

Authors	Country	No. Of cases	Age (yrs)/ gender	GBS diagnosis Method	GBS subtype	GBS-Covid-19 temporal relation	GBS Treatment	Outcome
Gigli GL et al.	Italy	1	53/M	Clinical/CSF/ NCS	AIDP	Post-infectious	IVIG (5 days)	Favorable
Guijarro-Castro C	Spain	1	70/M	Clinical/CSF/ NCS	AMSAN	Post-infectious	IVIG (5 days)	Favorable
Gutiérrez-Ortiz C et al.	Spain	1	50/M	Clinical/CSF	MFS	Post-infectious	IVIG (5 days)	Favorable
Helbok R et al.	Austria	1	68/M	Clinical/CSF/ MRI/NCS	AIDP	Post-infectious	IVIg (duration NR)/PE (4 sessions)	Poor (ICU)
Hirayama T et al.	Japan	1	54/F	Clinical/CSF/ NCS	AIDP	Post-infectious	IVIg (duration NR)	Favorable
Hutchins et al.	USA	1	21/M	Clinical/CSF	BFP	Post-infectious	NR	NR
Julio-Coamano DS et al.	Spain	1	61/M	Clinical/CSF	NR	Post-infectious	Oral Prednisolone	Poor
Khaja M et al.	USA	1	44/M	Clinical/CSF/ MRI	BFP	Para infectious	IVIG (5 days)	Favorable
Khalifa M et al.	Saudi Arabia	1	11/M	Clinical/CSF/ NCS	AIDP	Post-infectious	IVIG (5 days)	Favorable
Klinic D et al.	Netherlands	1	50/M	Clinical/CSF/ NCS	AIDP	Post infectious	IVIg (5 days)	Favorable
Korem S et al.	USA	1	58/F	Clinical/CSF	NR	Post infectious	IVIg (4 days)	Favorable
Kushwaha S et al.	India	1	65/M	Clinical/NCS	AIDP	Post infectious	IVIg (5 days)	NR
Lampe A et al.	Germany	1	65/M	Clinical/CSF/ NCS	AIDP	Post infectious	IVIg (duration NR)	Favorable
Lantos JE et al.	USA	1	36/M	Clinical/MRI	MFS	Post-infectious	IVIg (NR)	Favorable
Lascano AM et al.	Switzerland	3	52/F	Clinical/MRI/ CSF/NCS	AIDP	Post-infectious	IVIg (2 days)	Favorable
			63/F		AIDP	Post-infectious	IVIg (10 days)	Favorable
			61/F	Clinical/CSF/ NCS Clinical/MRI/ CSF/NCS	AIDP	Post-infectious	IVIg (2 days)	Favorable
Mackenzie N et al.	Colombia	1	39/F	Clinical/CSF/ NCS	AIDP	Post-infectious	PE (5 sessions)	Favorable
Manji HK et al.	Tanzania	1	12/M	Clinical	NR	Post-infectious	IVIg (5 days)	Died
Manganotti P et al.	Italy	5	72/M,	Clinical/CSF/ NCS	AIDP (n = 3),	Post-infectious	IVIg (5 days)	Favorable
			72/M,			Post-infectious	IVIg (5 days)	Favorable
			49/F,	Clinical/CSF/ NCS	AMAN (n = 2)	Post-infectious	IVIg (5 days)	Favorable
			94/M			Post-infectious	Methylprednisolone (5 days)	Favorable
			76/M	Clinical/CSF/ NCS Clinical/CSF/ NCS Clinical/CSF/ NCS		Post-infectious	IVIg (5 days)	Favorable
Manganotti P et al.	Italy	1	50/F	Clinical/CSF/ MRI	MFS	Post-infectious	IVIg (5 days)	Favorable
Marta-Enguita J et al.	Spain	1	76/F	Clinical	NR	Post-infectious	None- specific	Died
Masuccio FG et al.	Italy	1	70/F	Clinical/CSF/ MRI/NCS	AMAN	Post-infectious	PE (duration NR) & IVIg (1 day)	Poor
McDonnel EP et al.	USA	1	54/M	Clinical/CSF	AIDP	Post-infectious	IVIg (5 days)	Poor
Meshref M et al.	Saudi Arabia	1	18/F	Clinical/MRI/ NCS	AMAN	Post-infectious	IVIg (5 days)	Favorable
Mostel Z et al.	USA	1	69/F	Clinical/CSF/ NCS	AIDP	Post-infectious	IVIg (5 days)	Poor
Mozhdehipanah H et al. (Paybast S et al.)[a]	Iran	4	38/M	Clinical/CSF/ NCS	AIDP	Post-infectious	PE (5 sessions)	Favorable
			14/F		NR	Post-infectious	IVIg (5 days)	Favorable
			44/F	Clinical/CSF	AMSAN	Post-infectious	IVIg (3 days)	Died
			66/F	Clinical/CSF/ NCS Clinical/CSF/ NCS	AIDP	Post-infectious	IVIg (5 days)	Poor
Naddaf E et al.	USA	1	58/F	Clinical/CSF/ MRI/NCS	AMSAN	Post-infectious	PE (5 sessions)	Favorable
Nanda S et al.	India	4	55/F	Clinical/CSF/ NCS	AMAN	Post-infectious	IVIg (5 days)	Favorable
			72/M		AIDP	Post-infectious	IVIg (5 days)	Favorable
			55/M	Clinical/CSF/ NCS Clinical/CSF/ NCS Clinical/CSF/ NCS	AMSAN	Post-infectious	IVIg (5 days)	Favorable
			49/M		AMAN	Post-infectious	IVIg (5 days)	Favorable
Oguz-Akarsu E et al.	Turkey	1	53/F	Clinical/CSF/ MRI/NCS	AIDP	Para-infectious	PE (5 sessions)	Favorable
Ottaviani D et al.	Italy	1	66/F	Clinical/CSF/ NCS	AIDP	Post-infectious	IVIg (5 days)	Poor (ICU)
Padroni M et al.	Italy	1	70/F	Clinical/CSF/ NCS	AIDP	Post-infectious	IVIg (5 days)	Poor (ICU)
Paterson RW et al.	UK	3						

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Table 1 (*continued*)

Authors	Country	No. Of cases	Age (yrs)/ gender	GBS diagnosis Method	GBS subtype	GBS-Covid-19 temporal relation	GBS Treatment	Outcome
			52/M	Clinical/CSF/	AIDP	Para-infectious,	IVIg (5 days) (n = 3),	Poor (ICU)
			20/M	MRI/NCS	AIDP	Post-infectious	Methylprednisolone (5 days) (n =	Favorable
			63/M	Clinical/CSF/ MRI/NCS	AIDP	Post-infectious	2)	Favorable
				Clinical/CSF/ MRI/NCS				
Pelea T et al.	Germany	1	56/F	Clinical/CSF/ NCS	AMAN	Post-infectious	IVIg (5 days), PE (7 sessions)	Poor (ICU)
Pfefferkorn T et al.	Germany	1	51/M	Clinical/CSF/ MRI/NCS	AIDP	Post-infectious	IVIg (5 day) then PE (14 session)	Poor (MV)
Raahimi MM et al.	UK	1	46/M	Clinical/CSF/ MRI/NCS	AIDP	Post-infectious	IVIg (5 days)	Favorable
Rajdev K et al.	USA	1	36/M	Clinical/CSF/ MRI/NCS	AIDP	Post-infectious	IVIg (5 days) & PE (5 sessions)	Favorable
Rana S et al. et al.	USA	1	54/M	Clinical/NCS	AIDP	Post-infectious	IVIg (5 days)	Poor
Reyes-Bueno JA et al.	Spain	1	51/F	Clinical/CSF/ NCS	MFS	Post-infectious	IVIg (5 days)	Favorable
Riva N et al.	Italy	1	60/M	Clinical/CSF/ NCS	AIDP	Post-infectious	IVIg (5 days)	Favorable
Sancho-Saldana A et al.	Spain	1	56/F	Clinical/CSF/ NCS	AIDP	Post-infectious	IVIg (5 days)	Poor
Scheidl E et al.	Germany	1	54/F	Clinical/CSF/ NCS	AIDP	Post-infectious	IVIg (5 days)	Favorable
Sedaghat Z. (Karimi N & Sedaghat Z)[b]	Iran	1	65/M	Clinical/NCS	AMSAN	Post-infectious	IVIg (5 days)	Favorable
Senel M et al.	Germany	1	61/M	Clinical/CSF	MFS	Post-infectious	IVIg (5 days)	Favorable
Sharma R et al.	Qatar	1	25/M	Clinical/CSF/ MRI/NCS	AMAN	Post-infectious	IVIg (5 days)	Poor (LR)
Sidig A et al.	Sudan	1	65/M	Clinical/CSF/ NCS	AIDP	Post-infectious	IVIg (duration NR)	Favorable
SU XW et al.	USA	1	72/M	Clinical/CSF/ NCS	AIDP	Post-infectious	IVIg (4 days)	Poor (ICU)
Tekin AB et al.	Turkey	1	34/F	Clinical/CSF/ NCS	AMSAN	Post-infectious	IVIg (5 days)	Favorable
Tiet MY et al.	UK	1	49/M	Clinical/CSF/ NCS	AIDP	Post-infectious	IVIg (5 days)	Favorable
Toscano G et al.	Italy	5	77/F	Clinical/CSF/	AMSAN	Post-infectious	IVIg (n = 5)	Favorable (n
			23/M	NCS	AMSAN	Post-infectious	IVIg second course (n = 2)	= 1)
			76/M	Clinical/CSF/	AIDP	Post-infectious	PE (n = 1)	Poor (ICU)
			61/M	NCS	AIDP	Post-infectious		(n = 2)
			55/M	Clinical/CSF/ NCS	AMAN	Post-infectious		Poor (n = 2)
				Clinical/CSF/ NCS				
				Clinical/CSF/ NCS				
VelayosGalan A et al.	Spain	1	43/M	Clinical/EMG-NCS	AIDP	Post-infectious	IVIg (5 days)	Favorable
Virani A et al.	USA	1	54/M	Clinical/MRI	NR	Post-infectious	IVIg (5 days)	Favorable
Webb S et al.	UK	1	57/M	Clinical/CSF/ NCS	AIDP	Post-infectious	IVIg (5 days)	Favorable
Zhao H et al.	China	1	61/F	Clinical/CSF/ NCS	AIDP	Para-infectious	IVIg (duration was NR)	Favorable
Zito A et al.	Italy	1	57/M	Clinical/CSF/ NCS	AMSAN	Post-infectious	IVIg (5 days)	Favorable
Zubair AS et al.	USA	2	32/M	Clinical/CSF/	AMSAN	Post-infectious	IVIg (duration NR)	Favorable
			61/M	NCS	AMSAN	Post-infectious	IVIg (duration NR)	Favorable
				Clinical/CSF/ NCS				
Present cases	Qatar	2	49/M,	Clinical/CSF/	AIDP	Para-infectious	IVIg (5 days)	Favorable
			43/M	NCS	AMSAN	Para-infectious	IVIg (5 days)	Favorable
				Clinical/CSF/ NCS				

M male*; F* female*; AIDP* acute inflammatory demyelinating polyradiculoneuropathy; *AMSAN* acute motor and sensory axonal neuropathy; *AMAN*, acute motor axonal neuropathy; *BFP* - Bifacial weakness with paresthesias; *MFS* Miller Fisher syndrome; *COVID-19* coronavirus disease 2019; *GBS* Guillain-Barre syndrome; *PE* plasma exchange; *NR* not reported; *CSF* cerebrospinal fluid; *NCS* nerve conduction study; *ICU* intensive care unit; *IV* intravenous; *MRI* magnetic resonance imaging; *MV* mechanical ventilation; *LR* late recovery; *Poor* Intensive care unit admission, incomplete recovery or death.

[a] Two cases of these 4 case series have also been published by Paybast S et al. in another report (reference number 63).

[b] A follow up on the outcome of this patient has published in reference number [80].

Informed consent

Informed consent was obtained from the patients for their anonymized information to be published in this article.

Declaration of competing interest

All authors have been contributed in this study and declare no conflicts of interest related to the manuscript content.

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