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REVIEW ARTICLE

Guillain-Barre syndrome during COVID-19 pandemic: an overview of the reports

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Abstract

Similar to severe acute respiratory syndrome coronavirus (SARS-CoV) and Middle East respiratory syndrome coronavirus (MERS-CoV), the coronavirus disease 2019 (COVID-19) has neurological symptoms. COVID-19 patients have such clinical symptoms as headache, vomiting, nausea, dizziness, myalgia, anosmia, ageusia, and disorder of consciousness. These symptoms confirm that the nervous system is involved in the COVID-19 infection. Guillain-Barré syndrome (GBS) is a heterogeneous disorder which often follows a viral infection. According to the assessment case reports from the beginning of the COVID-19 infection so far, it is possible that GBS is linked to the COVID-19 infection. It seems that paying attention to the neurological effects of COVID-19 is necessary.

Keywords Guillain-Barré syndrome · SARS · MERS · COVID-19

Introduction

The first unexplained pneumonia cases occurred in Wuhan, China, and quickly spread to other countries [1]. It was later revealed that these unexplained pneumonia cases had been caused by a new coronavirus. It has been stated that the symptoms of this new coronavirus infection are very similar to those of SARS-CoV which spread in 2003 [2]. Both act on the same receptor, namely the angiotensin-converting enzyme 2. This virus is called SARS-CoV-2 and has been called by the WHO as the coronavirus disease 2019 (COVID-19) [3, 4]. COVID-19, like SARS-CoV and MERS-CoV, affects the nervous system. The neurological manifestations of the COVID-19 infection are due to its effects on the central nervous system (CNS) (headache, dizziness, consciousness disorder, acute brain disease, seizures, and ataxia), [5] peripheral nervous system (PNS) (anosmia, ageusia, visual impairment, nerve pain), and skeletal muscles [6].

Guillain-Barré syndrome (GBS) is an autoimmune disorder related to the peripheral nervous system. The clinical characteristics of GBS are the progressive weakness of the limbs and reduction in or loss of tendon reflexes (hyporeflexia and areflexia, respectively). In this disorder, protein concentrations in the cerebrospinal fluid (CSF) increase, while the white cell count is normal [7, 8]. GBS is usually caused by a viral or bacterial infection. In response to the antigen, the immune system is activated and the nerve roots and peripheral nerves are injured because of the structural similarity of this antigen to axons and myelin [9]. The symptoms peak within 4 weeks and the patients should be monitored because 20% to 30% of them will need mechanical ventilation [10, 11]. The goal of the current study was to review the available information on the reports of GBS associated with the COVID-19 infection.

Literature search strategy

In this study, a literature search was done on SCOPUS, PubMed, Embase, Cochrane database, Google Scholar, and Ovid according to preferred reporting items for GBS related to COVID-19 infection. The used keywords were "SARS-CoV," "MERS-CoV," "COVID-19," "SARS-CoV2," "neurology," "nervous system," "neurological manifestations," "Guillain-Barré syndrome."

Guillain-Barré syndrome in SARS and MERS

An epidemic of severe acute respiratory syndrome (SARS) arose from SARS-CoV in Asia in February 2003 [12].

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SARS was characterized by myalgia, fever, and other systemic symptoms from which the patient usually recovered after a few days [13]. Neurological manifestations were observed in 30% of the patients [14]. MERS-CoV was first reported in Saudi Arabia in June 2012 [15]. Previous reports have stated that CNS may be one of the target organs of the MERS-CoV infection and should thus be considered in the patient [16]. There are some reports about the effects of SARS and MERS on the nervous system and the development of GBS.

Some case reports have confirmed that patients with SARS-CoV and MERS-CoV develop acute polyneuropathy [17–21]. It has been stated that 21 to 25 days after the SARS-CoV infection, weakness in the limbs begins with the involvement of the peripheral nerves. Neurological studies have also shown that the compound muscle action potential (CMAP) amplitudes are temporarily reduced. Electromyography has shown acute denervation and polyphagia in patients with SARS-CoV [22]. According to a case report, 21 days after the onset of SARS-CoV, three patients showed peripheral nerve disorders and experienced neuropathy and myopathy [19]. An acute polyneuropathy which is a common form of GBS has been observed in patients the MERS-CoV infection [20]. Similarly, a 28-year-old patient with MERS-CoV had weakness in the legs and was unable to walk. In this case, the neurological findings were consistent with the diagnosis of GBS [19]. GBS was also reported in a child with beta coronavirus (HCV-OC43) infection with such symptoms as unilateral peripheral palsy [22, 23]. At the time, electrophysiological studies on follow-up examinations were recommended to achieve a good prognosis.

Guillain-Barré syndrome in COVID-19

The most common microorganisms that are involved in the development of axonal and demyelinating subtypes of GBS include Epstein–Barr virus, *Campylobacter jejuni*, cytomegalovirus, influenza A virus, *Haemophilus influenza*, and *Mycoplasma pneumoniae*. Previously discovered types of coronavirus (SARS-CoV and MERS) and Zika virus have been associated with GBS as well [24]. The mechanism of the GBS incidence is based on molecular mimicry and anti-ganglioside antibodies after an infection in genetically predisposed patients [25]. These antibodies show the highest association with certain forms of GBS [26, 27]. A possible mechanism is an autoimmune reaction in which the antibodies on the pathogen, which are similar to the protein structures of the peripheral nerve components, lead to the damage of the nervous system [9]. This likeness has been termed "molecular mimicry" which is defined as the theoretical possibility that sequence similarities between foreign and self-peptides are enough to lead to the cross-activation of autoreactive B cell or T cell by pathogen-derived peptides [25].

As of Aug 2020, several case reports of GBS have been published in connection with the COVID-19 infection. These studies suggest a link between COVID-19 and GBS. At the time of writing this paper, approximately 31 cases of GBS associated with COVID-19 infection have been reported. This study showed that, on average, 11.92 ± 6.20 days after COVID-19 infection, the neurological symptoms of GBS begin. The minimum time for this event was 3 days and the maximum time was 24 days. It is noteworthy that in two cases, GBS incidence coincided with COVID-19 infection (Table 1). The age of five patients was not mentioned in these reports. However, the mean age of the other patients was 57.26 ± 15.82, with the youngest patient being 5 years old and the oldest patient being 84 years old. There were 17 men and 14 women in the reported cases (Table 1). The diagnosis of GBS is based on clinical features, cerebrospinal fluid (CSF) testing, and nerve conduction studies [28]. The results of the current study showed that the more common clinical features that led the patients to refer to medical centers included paresthesia in the feet and hands, symmetric weakness in the lower limbs, facial palsy, acute proximal tetraparesis, gait difficulties, and root-type pain in all four limbs (Table 1). Of the total number of reported cases, 5 patients required hospitalization in the ICU (Table 1). Anti-ganglioside antibodies were positive in 5 patients and negative in 26 patients. The CSF protein levels were not reported in 3 patients, were normal in 4 patients, and increased in 24 patients. In addition, the cell count in the CSF was normal in all patients (Table 1). The major subtypes of GBS include acute inflammatory demyelinating polyneuropathy (AIDP), acute motor axonal neuropathy (AMAN), acute motor sensory axonal neuropathy (AMSAN), and Miller Fisher syndrome (MFS) [29]. In the current study, the different subtypes of GBS were found in relation to COVID-19 infection. However, most reports referred to AIDP (Table 2). Intravenous immunoglobulin therapy was performed for all patients.

Table 1 The patient information with GBS related to COVID-19 infection

		mean ± SD
Age		57.26 ± 54.83
Gender (%)	Male	17 (54.83)
	Female	
Times the onset of GBS after infection		11.92 ± 6.20
Protein level in CSF (%)	Normal	4 (12.90)
	Not reported	3 (9.67)
	Evaluated	24 (77.43)
Anti-gangliosides antibodies (%)	Positive	5 (16.12)
	Negative	26 (83.88)
Admitted in ICU (%)		5 (16.12)

Table 2 Characteristics of patients with GBS related to COVID-19 infection

Patient no.	GBS symptoms	Nerve studies
1 [30]	• Paresthesia in feet and hands	• A flaccid severe tetraparesia • Medical Research Council (MRC) strength evaluation was 2/5 in the legs, 2/5 the arms, 3/5 in the forearms and 4/5 in the hands • Tendon reflexes were abolished in the four limbs • Swallowing disturbance
2 [31]	• Asthenia, and myalgia in legs • Paraesthesia, hypoesthesia, and distal weakness in the lower limbs • Midthigh and tip of the fingers associated with ataxia • Right peripheral facial palsy	• Decreased light touch from midthigh to feet and the tip of the fingers • Decreased vibration sense in the lower limbs • Symmetric weakness for dorsiflexion • Extension of the toes (MRC score = 3/5) and flexion of the thigh (MRC = 4/5) • Areflexia in the forelimbs apart from the left biceps reflex
3 [31]	• acute proximal tetraparesis and distal forelimb and perioral paraesthesia.	• proximal lower limb weakness (MRC 2/5), distal weakness (MRC 4/5), and diffuse areflexia • A typical demyelinating pattern with a conduction block in the left median nerve • temporal dispersion, upper limb increased motor distal latencies • Diffuse decreased motor and sensory conduction velocities • Lower than 38 m/s in 9 nerves of 10 tested, and neurogenic pattern on EMG • Left peripheral facial palsy
4 [32]	• Symmetric weakness in lower limbs, leading to falls and paraplegia	• Weakness in the upper limbs (MRC 4/5) and diffuse areflexia, but no clear sensory deficits • F-waves with diffuse prolonged distal motor latencies and reduced distal compound muscle action potential amplitudes with a slight reduction of conduction velocities, thus suggesting a mixed pattern of demyelination and axonal damage • No sensory nerve action potential was registered
5 [33]	• Paresthesia at limb extremities • Distal weakness rapidly evolving to a severe • Flaccid tetraparesis	• Symmetric limb weakness (MRC score 3/5 at upper limbs and 2/5 at lower limbs) • Hypoesthesia at the 4 limbs • Absent deep tendon reflexes • Severe paresthesia in both hands and feet • Absence of both the sural nerve sensory nerve action potential (SAP) and the tibial nerve compound muscle action potential (CMAP)
6 [34]	• Asthenia and paresthesia at feet and hands • Gait difficulties	• Symmetric distal upper and lower limbs weakness • Loss of deep tendon reflexes • Preserved light touch and pinprick sensation
7 [35]	• Bilateral lower limb pain • Irritability • Difficulty walking • Loss of balance • Unilateral peripheral facial and bulbar palsy	• Left facial droop • Effacement of left nasolabial fold while smiling • Inability to close the left eye • Raise the left eyebrow or frown • All consistent with Bell's palsy • The position sensation was decreased • Deep tendon reflexes were absent • Dysmetria and appendicular ataxia were present in all four limbs
8 [36]	• Weakness in both legs and severe fatigue	• Symmetric weakness (MRC grade 4/5) • Areflexia in both legs and feet • Sensation to light touch and pinprick was decreased distally
9 [37]	• Numbness and weakness of his lower extremities	• (MRC: 2/5 strength in his lower extremities with 3/5 in his upper extremities) • Absence of deep tendon reflexes • Ascending paralysis with supporting physical exam findings
10 [38]	• Gait disturbance • Weakness in hip flexors • Paresthesias of his hands and feet	• Proprioceptive sense at the toes • Bilateral facial weakness • Dysphagia • Dysarthria • Neck flexion weakness and inability to ambulate
11 [38]	• Paresthesia of his hands and feet • Gait disturbance	• Initial examination revealed 3/5 shoulder shrug, 4−/5 hip and neck flexion • Diminished vibration and proprioception at the toes • Reflexes were 1+ in the arms and absent in the legs • Unable to stand or ambulate independently

Table 2 (continued)

Patient no.	GBS symptoms	Nerve studies
12 [39]	• Bilateral weakness and tingling • Sensation in all four extremities	• quadriplegia, hypotonia, areflexia, and bilateral positive Lasègue sign • reduction or absence of electrical potentials in both motor and sensory nerves in all four limbs
13 [40]	• Bilateral facial nerve palsy	• No other neurological findings at examination
14 [41]	• Paraparesis • Distal allodynia • Difficulties in voiding and constipation	• Bilateral lower limb flaccid paresis • Absent deep tendon reflexes of the upper and lower limb • Idiomuscular response to percussion of the muscle *tibialis anterior* • Indifferent plantar reflexes • No sensory deficit
15 [42]	• Pain and numbness in distal lower and upper • Extremities progressive weakness in legs	• A mild peripheral facial • Nerve palsy on the right side • Muscles' forces were 4/5 in distal and proximal lower extremities according to the MRC grading • The upper extremities showed no weakness • Deep tendon reflexes were absent in all four limbs • Demyelinating polyneuropathy
16 [42]	• Ascending lower and upper • Extremities weakness • Paresthesia	• Decreased forces, MRC grade of 2/5 at proximal and 3/5 at distal lower limbs, and 4/5 in both arms • Deep tendon reflexes were absent in both legs and decreased to 1+ in the upper extremities
17 [43]	• Symmetric paresthesias • Ascending appendicular weakness	• Mental status and cranial nerves were normal • Strength was 4/5 neck flexion, 3/5 proximal upper and lower extremities bilaterally • Tendon reflexes were absent • Sensation to light touch was diminished to wrists and • knees bilaterally
18 [44]	• Symmetric ascending quadriparesis	• weakness in four limbs with a MRC scale of 2/5 in proximal, 3/5 in distal of the upper extremities and 1/5 in proximal, 2/5 in distal of the lower extremities • Deep tendon reflexes were absent • Reduction in the vibration and fine touch sensation distal to the ankle joints and also bifacial nerve palsy • No spine sensory level • AMSAN form
19 [45]	• Unsteadiness and paraesthesia in both hands • Bilateral facial nerve palsy, oropharyngeal weakness, and severe proximal tetraparesis	• Mild proximal tetraparesis 4/5 on the Medical Research Council (MRC) scale with global areflexia • Touch, pinprick and proprioception were normal
20 [46]	• Root-type pain in all four limbs • Weakness in his lower limbs • Inferior bilateral facial paresis • Paraparesis	• Left external rectus muscle with horizontal diplopia • Global areflexia • Acute demyelinating polyneuropathy • Denervation • Paresis of the left external rectus muscle with horizontal diplopia when looking to the left
21 [47]	• Perioral paresthesias, but no facial weakness • Ataxic gait	• Flexor bilaterally • Neuro-ophthalmologic examination revealed visual acuity of 20/b25 in both eyes • Right internuclear ophthalmoparesis and right fascicular oculomotor palsy
22 [47]	• Acute onset of diplopia	• Visual acuity of 20/25 in both eyes • Severe abduction deficits in both eyes, and fixation nystagmus, with the upper gaze more impaired, all consistent with bilateral abducens palsy • Deep tendon reflexes were absent
23 [48]	• Proximally pronounced • Moderate symmetric paraparesis	• Progressive proximally pronounced paraparesis, areflexia, and sensory loss with tingling of all extremities
24 [49]	• Inferior bilateral facial paresis • Paraparesis	• Arreflexia
25 [50]	• Progressive weakness • Numbness of the lower extremities	• Mild dysarthria due to jaw weakness and bilateral, predominantly lower limb weakness, with 4−/5 strengths in knee and ankle flexor and extensor

Table 2 (continued)

Patient no.	GBS symptoms	Nerve studies
		muscles, and 4−/5 in the left and 4+/5 in right hip flexor muscles by the Medical Research Council (MRC) scale • Tendon reflexes were absent
26 [51]		• Flaccid areflexic tetraplegia evolving to facial weakness, upper limb paresthesia and respiratory failure
27 [51]		• Facial diplegia and generalized areflexia evolving to lower limb paresthesia with ataxia
28 [51]		• Flaccid tetraparesis and facial weakness evolving to areflexia and respiratory failure
29 [51]		• Flaccid areflexic tetraparesis and ataxia
30 [51]		• Facial weakness, flaccid areflexic paraplegia, and respiratory failure
31 [52]	• Acute progressive paresthesia of distal lower extremities evolving to the upper limbs leading to quardiparesthesia	• Facial paralysis and mildly dysarthric speech • Deep tendon reflexes were generally absent • Acute demyelinating polyneuropathy

Discussion

The neurological manifestations in COVID-19 have attracted a lot of attention [53]. Since the onset of the COVID-19 pandemic, there have been reports of the possible link between GBS and the COVID-19 infection [17]. In this review, based on the case reports of the involvement of the nervous system following the COVID-19 infection, a conclusion was made about the possible association between this infection and GBS. Given the importance of COVID-19, further studies are needed to understand the effects of this infection on the nervous system. In particular, the study of the mechanism of GBS following the coronavirus attack is important.

The development of neurological symptoms in a time interval after infection is the classical phenotype of GBS. In most cases, the onset of the neurological symptoms related to GBS was about 1 to 4 weeks after the diagnosis of COVID-19. However, in 2 cases, the symptoms coincided with the diagnosis. In this regard, Zhao et al. [36] proposed a so-called "parainfectious" profile pattern when GBS occurs at the time of infection.

The current study showed that most patients with GBS due to the COVID-19 infection were elderly men. The studies have shown that most patients with GBS are mostly elderly men [10]. The reported incidence of GBS ranges from 1 to 2 cases per 100,000 adults and from 0.4 to 1.4 cases per 100,000 children per year [10, 54–56]. So far, only one child has been diagnosed with GBS following a coronavirus infection [35]. The clinical characteristics of nonelderly adult patients with GBS are distinct from those of the elderly [57]. However, since only one child has been diagnosed with GBS associated with COVID-19, no statement can be made in this regard at this time.

One of the most common neurological symptoms of GBS is acute muscle weakness. The pattern of muscle weakness may be helpful in the diagnosis of GBS [58]. Weakness in the limbs and acute flaccid quadriparesis were observed in most GBS case reports after the diagnosis of COVID-19. Furthermore, demyelinating polyneuropathy was commonly observed in most of these reports. Some of the COVID-19-related GBS patients had the axonal variants of GBS.

In most of the patients in this study, similar to what happens in GBS [59], the protein levels in the CSF were elevated, the cell counts were normal, and the anti-ganglioside antibodies were absent.

Many infectious agents have been associated with GBS including the Epstein–Barr virus, cytomegalovirus, *Campylobacter jejuni*, human immunodeficiency virus (HIV), and ZiKa virus. In the current study, there were some case reports of GBS related to the COVID-19 infections. However, in two case reports, the clinical characteristics did not fully support the typical post-infectious pattern of GBS and rather resembled a form of acute paralysis that has already been associated with the ZiKa virus [32, 60, 61].

After the diagnosis of COVID-19, some GBS cases were admitted to the ICU [30, 31, 33, 43]. Therefore, the correct and timely diagnosis of GBS associated with COVID-19 can prevent its progress.

Coronaviruses are thought to cause GBS in certain patients either directly through neuroinvasive capacity (ACE2 receptors on neuronal tissues) or indirectly through the response of the immune system (inflammatory mechanism) [36, 53]. The data indicate that SARS-CoV-2 is able to cause an immune reaction with an increased level of interleukin-6 (IL-6) which stimulates the inflammatory cascade and damages tissues. Therefore, inflammatory factors may play an important role

in the organ dysfunctions of patients with COVID-19 infection [44, 62–64]. These immunological processes are probably responsible for most of the neurological manifestations.

Conclusion

According to the case reports from around the world, it is possible that GBS is linked to the COVID-19 infection. However, more cases with epidemiological data should be studied and future investigations should be carried out in this regard. Due to the possible association of GBS and COVID-19, it is recommended that the patients be followed up by physicians with respect to neurological manifestations. Finally, it is suggested that research on the relationship between COVID-19 and the nervous system be not limited to the current period so that if we encounter a new type of this virus in the future, the necessary measures could be taken.

Compliance with ethical standards

Conflict of interest The author declares no conflicts of interest.

Ethical approval Not applicable.

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