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Short Communication

The challenge of diagnosing Guillain–Barre syndrome in patients with COVID-19 in the intensive care unit

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A B S T R A C T

Various neurological complications have been described in COVID-19 patients, especially Guillain–Barre syndrome (GBS). The underlying mechanisms on the association between SARS-CoV-2 infection and GBS remain unclear, but several hypotheses have been proposed. It seems that post-SARS-CoV-2 GBS shares many characteristics with classic post-infectious GBS; however, it may occur in sedated and intubated patients hospitalized in the intensive care unit for SARS-CoV-2 acute respiratory distress syndrome, which presents challenges in the diagnosis and treatment of GBS. In this study, we describe three cases of post-SARS-CoV-2 GBS that were hospitalized in the intensive care unit.

1. Introduction

Since December 2019, the COVID-19 pandemic quickly spread all over the world, causing an increase in the number of hospitalizations and deaths due to acute respiratory distress syndrome (ARDS) (Hasan et al., 2020). However, as we have learned more about SARS-CoV-2, it has become clear that COVID-19 is responsible for a multitude of systemic complications, including neurological complications. Various studies have described a temporal relationship between COVID-19 and Guillain–Barre syndrome (GBS) (Meppiel et al., 2021; Shehata et al., 2021), but no causal relationship has been proven till date. GBS is an acute inflammatory demyelinating polyradiculoneuropathy (AIDP) that includes other acute inflammatory polyradiculoneuropathies, such as Miller Fisher, and often occurs post infection (Sejvar et al., 2011). The relationship between GBS and Campylobacter jejuni, Zika virus, or influenza virus has been previously described (Loshaj-Shala et al., 2018; Leonhard et al., 2021; Grisanti et al., 2021). The diagnosis of post-COVID-19 GBS could be difficult, especially when patients are hospitalized in the intensive care unit (ICU), sedated, and intubated (Finsterer and Scorza, 2021a) or suffering from critical illness polyneuropathy (CIP) (Finsterer and Scorza, 2021b). To better describe GBS in the ICU, we analyzed the characteristics of three patients infected by SARS-CoV-2 and diagnosed with GBS in the ICU in our center.

2. Material and methods

Between March 1, 2020, and May 31, 2021, we identified all COVID-19 patients in the ICU in our French tertiary care center with a diagnosis of GBS that met the Brighton criteria. We started collecting data in March 2020 at the beginning of the pandemic in France. We collected data from clinical examinations (motor and sensitive testing, deep tendon reflexes, and cranial nerve examination), routine blood chemistry analyses, cerebrospinal fluid (CSF) analyses (including cell count, protein, glucose, standard bacterial culture, and search of specific anti-
ganglioside antibodies (the tested antibodies were anti-GM1, anti-GM2, anti-GM3, anti-GD1a, anti-GD2, anti-GD3, anti-GT1a, anti-GT1b, anti-GQ1b, and anti-GM4)), electromyography (EMG) with measurements for motor and sensory nerve conduction, and brain imaging. The criteria by Hadden et al. (1998) were used to define demyelination on EMG. Thus, the primary demyelinating form was defined as a motor conduction velocity inferior to 90% of the lower limit of normal (LLN), a distal motor latency superior to 110% of the upper limit of normal (ULN), and a compound muscle action potential amplitude after proximal stimulation/comound muscle action potential amplitude after distal stimulation (dCMAP) ratio inferior to 0.5, dCMAP superior or equal to 20% of the LLN, and a F-response latency inferior to 120% of the ULN.

We collected the data during the patients’ follow-ups at weeks 4, 8, and 12. The study was approved by the Ethical Committee of Medicine Odontology and Pharmacy Faculties and Hospitals (University Hospital of Strasbourg) N◦ CE-2020-32.

Table 1
Patients’ characteristics.

| Patient | 1 | 2 | 3 |
|---------|---|---|---|
| Age     | 53 y | 68 y | 78 y |
| Sex     | Male | Female | Female |
| Comorbidity | BPPV, asthma | Localized scleroderma (morpha) | Atrial fibrillation, hypothyroidism, depressive disorder, herniated disc |
| Early symptoms of COVID-19 | Fever, myalgia, abdominal pain, emesis | Asthenia, cough, dyspnea | Asthenia, cough |
| COVID-19 diagnosis | Positive RT-PCR COVID on nasopharyngeal swab | Positive RT-PCR COVID on nasopharyngeal swab | Positive RT-PCR COVID on nasopharyngeal swab |
| Time from COVID-19 symptom onset to hospitalization | 19 days | 10 days | 6 days |
| Mechanical ventilation | Yes | Yes | Yes |
| Severity of COVID-19 | Mild illness (non-respiratory symptoms) | Critical illness | Critical illness |
| Time from intensive care unit admission to GBS diagnosis | 3 days | 22 days | 18 days |
| GBS subtypes | AIDP | Miller Fisher syndrome with involvement of the peripheral nerves | AIDP |
| Neurological symptoms | Painful tetraparesis | External ophthalmoplegia | Flaccid tetraparesis |
| Neurological examination | Areflexia in lower limbs | Flaccid tetraparesis | Facial diplegia |
| Hyporeflexia in upper limbs | Diffuse areflexia | Diffuse areflexia |
| Findings on CSF analysis | High protein level (1.7 g/L), 2 cells/mm$^3$ | Normal protein level (0.23 g/L), 2 cells/mm$^3$ | High protein level (3.31 g/L), 2 cells/mm$^3$ |
| Anti-ganglioside antibodies (IgG) | anti-GD3 IgG | Negative | Negative |
| (IgG) | anti-GT1a IgG | Not done | Leukoencephalopathy; cortical/subcortical brain atrophy |
| (IgG) | anti-GQ1b IgG | Increased distal motor latency in right ulnar nerve | Decreased motor conduction velocity in left tibial and left ulnar nerves and increased distal motor latency in left and right ulnar nerves and left and right tibial nerves |
| Brain MRI | Not done | Normal protein level (0.23 g/L), 2 cells/mm$^3$ | Normal protein level (0.23 g/L), 2 cells/mm$^3$ |
| Electromyography | Decreased motor conduction velocity in left tibial nerve, increased distal motor latency in median and right tibial nerves, increased F-response latency in right tibial nerve, and decreased sensitive conduction velocity in left ulnar nerve | Increased distal motor latency in right ulnar nerve and no F-response latency in right ulnar nerve | Normal protein level (0.23 g/L), 2 cells/mm$^3$ |
| Neurotoxic drugs before diagnosis | No | Hydroxychloroquine | No |
| Treatment of GBS | No | Atazanavir/ritonavir | Atazanavir/ritonavir |
| Specific COVID-19 therapies | Immunoglobulins (two courses) | Immunoglobulin (one course) | Immunoglobulins (two courses) |
| Differential diagnosis | | | |
| Campylobacter jejuni | Serology negative | No data available | Serology negative |
| EBV | anti-EBNA IgG negative | anti-EBNA IgG negative | anti-EBNA IgG negative |
| | anti-VCA IgM negative | anti-VCA IgG negative | anti-VCA IgM negative |
| CMV | IgM positive | No data available | IgM positive |
| | IgG positive | IgG negative | IgM negative |
| HBV | Anti-Hbs positive | No data available | Anti-Hbs positive |
| | Ag Hbs and Anti-Hbc negative | Ag Hbs negative | |
| HCV | Anti-VHC negative | No data available | Anti-VHC negative |
| HEV | anti-VHE IgM negative | No data available | anti-VHE IgM negative |
| HIV | Serology negative | No data available | Serology negative |
| | Complete recovery | Weakness in lower limbs, balance disorder |
| Clinical outcome | Sensory impairment of sole, balance disorder | 20% of the LLN, and a F-response latency inferior to 120% of the ULN. | |
| Length of stay in intensive care unit | 41 days | 41 days | 73 days |
| Length of stay in hospital | 97 days | 79 days | 156 days |

BPPV, benign paroxysmal positional vertigo; GBS, Guillain–Barre syndrome; CSF, cerebral spinal fluid; MRI, magnetic resonance imaging; RT-PCR, reverse transcriptase-polymerase chain reaction; AIDP, acute inflammatory demyelinating polyneuropathy; EBV, Epstein-Barr virus; CMV, Cytomegalovirus; HBV, Hepatitis B virus; HCV: Hepatitis C virus; HEV, Hepatitis E virus; HIV: Human immunodeficiency virus; M. pneumoniae: M. pneumoniae.
peripheral nerves. Their demographic characteristics, clinical data, and GBS and one had a Miller Fisher syndrome with involvement of the M. pneumoniae
positive, except anti-CMV IgM and anti-VCA IgM in one patient and anti-
plasma pneumoniae
test results for other infections that could trigger GBS ( [38x343]
raparesia and no damage was observed on chest tomography. Lumbar
did not present any respiratory symptoms before the beginning of tet-
muscular deficiency and respiratory failure caused by GBS. This patient
validation of Brighton criteria
must be consistent with the diagnosis of Guillain-Barré syndrome. Level 1 is the
highest level of diagnostic certainty, level 4 is the lowest level of diagnostic
From Fokke et al., 2014, “Diagnosis of Guillain-Barré syndrome and

diagnosis of GBS was in the ICU for the three patients. In addition,
anti-ganglioside antibodies were found in one patient. Serological

*If CSF is not collected or results not available, nerve electrophysiology results
must be consistent with the diagnosis of Guillain-Barré syndrome. Level 1 is the
highest level of diagnostic certainty, level 4 is the lowest level of diagnostic
certainty. From Fokke et al., 2014, “Diagnosis of Guillain-Barré syndrome and
validation of Brighton criteria”.

3. Results

Among the 294 adult patients with confirmed SARS-CoV-2 infection
hospitalized in the ICU in our center between March 1, 2020, and May
31, 2021, three were diagnosed with GBS (1.02%). Two had classical
GBS and one had a Miller Fisher syndrome with involvement of the
peripheral nerves. Their demographic characteristics, clinical data, and
results of investigations are shown in Table 1. The mean age of the three
patients was 66 years (range, 53–78 years). None of them had other
neurological comorbidities. All patients required invasive mechanical
ventilation, of which two required it due to respiratory failure caused by
ARDS and they did not present any neurological symptoms at the
beginning of respiratory failure when they were still conscious. One of
the patients required invasive mechanical ventilation due to neuromuscular
deficiency and respiratory failure caused by GBS. This patient
did not present any respiratory symptoms before the beginning of tet-
raparesia and no damage was observed on chest tomography. Lumbar
puncture and EMG were performed in all patients, and the results were
compatible with those of GBS (Table 1). EMG was performed 2 days after
the onset of neurological symptoms in patient 1 and approximately 15
days after the onset of neurological symptoms in patients 2 and 3. Hence,
a diagnosis of GBS was made in the ICU for the three patients. In addition,
anti-ganglioside antibodies were found in one patient. Serological
test results for other infections that could trigger GBS (C. jejuni, Myco-
plasma pneumoniae, EBV, CMV, HBV, HCV, HEV, and HIV) were nega-
tive, except anti-CMV IgM and anti-VCA IgM in one patient and anti-
P. pneumoniae IgG in one patient. The serology data are missing for one
patient. There was no clinical argument for these infections. For this
study, we arrived at a diagnosis of GBS when the Brighton criteria were
fulfilled (Fokke et al., 2014; Shahrizaila et al., 2021) (Table 2). One
patient had neurological symptoms before sedation, which presented 20
days after the first COVID-19 symptoms when the patient was not yet
hospitalized. Two patients were sedated before the first neurological
symptoms appeared; the delay of neurological symptom onset could not be
precisely defined (approximately 14 days after the onset of COVID-19
symptoms). All patients were sedated with sufentanil and midazolam for
5 to 10 days. All patients had a certain or highly probable GBS diagnosis
following the Brighton criteria (level 1 or 2). All patients were treated by
intravenous immunoglobulin (IVIG; 400 mg/kg/day) for 5 consecutive
days, and two of them received two courses of IVIG treatment with the
same protocol due to the severity of the neurologic impairment. The three
patients had a favorable outcome complete recovery (n = 1) or
with negligible to moderate sequela (n = 2), albeit with a slow recovery,
as evidenced by the length of stay in the ICU (mean, 51.7 days; range,
41–73 days).

4. Discussion

We report three cases of GBS in the ICU that were diagnosed ac-
cording to the clinical, biological, and electrophysical criteria during
their hospitalization for COVID-19. The pathophysiology of GBS post-
SARS-CoV-2 infection remains unclear and debated. Some studies have
suggested the possibility of an inflammatory response targeting the
nervous system, demonstrated by an increased level of pro-
inflammatory markers in CSF such as IL-6, IL-8, or TNF-α (Gigli et al.,
2020; Manganiotti et al., 2021). Araújo et al. have proposed the mechan-
amism of direct viral toxicity on the nervous system, highlighting the
positivity of the reverse transcriptase-polymerase chain reaction COVID
in the CSF of one patient (Araújo et al., 2021). Other authors have
considered molecular mimicry (Siracusa et al., 2021), already largely
described with other post-infectious GBS like C. jejuni, which have been
linked with the detection of anti-ganglioside antibodies in the CSF, as we
found in one patient (Lozsha-Shala et al., 2018). Finally, some authors
have suggested the eventuality of a genetic predisposition by alleles of
human leukocyte antigen (HLA) (Gigli et al., 2020). Our three patients
were over 50 years old; however, younger cases have been described in
the literature, including pediatric cases (Curris et al., 2021). Two pa-
tients had a critical COVID-19 illness (respiratory failure requiring me-
chanical ventilation), and one patient had a mild COVID-19 illness (non-
respiratory symptoms without oxygen supplementation). However, the
relationship between COVID-19 severity and GBS is unclear. Gigli et al.
described a paucisymptomatic patient infected by SARS-CoV-2 who
developed GBS (Gigli et al., 2020). Some studies have suggested a higher
frequency of the demyelinating form of GBS (AIDP) when it occurs after
COVID-19 (Paladidinou et al., 2021; Sheikh et al., 2021). In our study,
two patients had a AIDP and one patient had a Miller Fisher syndrome
with involvement of the peripheral nerves. Our study only considered
sedated and intubated patients hospitalized in the ICU, which made it
harder to confirm the diagnosis of GBS since the patients cannot express
any muscular weaknesses or sensory impairments. However, in one
patient, the first GBS symptoms started before ICU admission, which is
why lumbar puncture has to be done as soon as possible when a patient
presents with neurological symptoms. In two patients, the first GBS
symptoms started in the ICU after they were sedated and treated with
specific COVID-19 therapy. As it was not possible to perform motor
examinations for these patients, the main argument to consider
regarding the diagnosis of GBS was the difficulty in weaning from me-
chanical ventilation and the absence of clinical response to the modifi-
cation of the respiratory rate imposed with the ventilator. Given the
inability to perform a complete neurological examination on a sedated
patient, it is essential to consider and eliminate all differential diagnosis
prior to diagnosing GBS (Finsterer and Scorzà, 2021b). The first condi-
tion to rule out is CIP, which can be clinically and electromyographically
difficult to distinguish from GBS, especially with the axonal variant
(Finsterer and Scorzà, 2021b; Zhang et al., 2014). We excluded CIP due
to the delay between ICU admission and diagnosis, which was consid-
teed too short for patient 1 (3 days), clinical examination (external
ophthalmoplegia for patient 2, which is not compatible with CIP), CSF
analyses (cytoalbuminologic dissociation for two patients and presence
of anti-ganglioside antibodies for patient 1) and electrophysiological
data. CIP usually occurs due to axonal damage (Laconi, 2013), and all
three patients had the primary demyelinating form according to Hadden’s
criteria. Second, we excluded all toxic neuropathy, which is a
classical differential diagnosis of GBS and can be frequently found in
patients under polycamidation in the ICU. Only one patient was
administered neurotoxic drugs before the onset of the first neurological
symptom, but the exposure time to this drug seems too short to be
responsible for neuropathy (7 days of treatment with hydroxy-
chloroquine and 5 days of treatment with atazanavir/ritonavir). One
potential explanation for a causal relationship between COVID-19 and
GBS is the negativity of the infection’s serologies that can trigger GBS.
One patient had positive anti-VCA IgM but negative EBV PCR, which

Table 2
Brighton criteria.

| Diagnostic criteria | Level of diagnostic certainty | 1 | 2 | 3 | 4 |
|---------------------|-------------------------------|---|---|---|---|
| Bilateral and flaccid weakness of limbs | + | + | + | ± |
| Decreased or absent deep tendon reflexes in weak limbs | + | + | + | ± |
| Monophasic course and time between onset-nadir 12 h to 28 days | ± | ± | ± | ± |
| CSF count cell –50/µl | ± | ± | ± | ± |
| CSF protein concentration > normal value | ± | ± | ± | ± |
| NCS findings consistent with one of the subtypes of GBS | + | ± | ± | ± |
| Absence of alternative diagnosis for weakness | + | + | + | + |

+, present; −, absent; ±, present or absent; GBS, Guillain-Barre syndrome; NCS, nerve conduction studies.

*If CSF is not collected or results not available, nerve electrophysiology results must be consistent with the diagnosis of Guillain-Barré syndrome.
excludes primary EBV infection. This patient had doubtful anti-CMV IgM (0.7) (negativity threshold inferior to 0.7; positivity threshold superior to 1). Since CMV serology was performed on the second day of IVIg, we could not interpret the IgG results. It is likely that the positivity of CMV IgM reflects viral reactivation or polyclonal stimulation, especially since the patient did not present any clinical argument for CMV infection. Patient 3 had positive anti-M. pneumoniae IgG but negative IgM, which evoked an old infection.

The Brighton criteria were developed and validated by studies (Fokke et al., 2014) to help clinicians standardize the diagnosis of GBS. GBS is a therapeutic urgency, particularly when patients are hospitalized in the ICU, for whom the rehabilitation is often longer and more challenging (Dhar et al., 2008) due to prolonged bed rest and intensive care complications being added to GBS. In our study, the mean duration of ICU stay was 51.7 days (range, 41–73 days), and the duration of hospitalization was 97, 79, and 156 days for patients 1, 2, and 3, respectively, which reflects the severity of clinical presentations and the latency of rehabilitation. During the COVID-19 crisis, free hospital beds are rare and diagnosis should be rapid to reduce the duration of ICU stay. Two patients received two courses of IVIg. There is no consensus about the efficacy of the second 5-day course of IVIg for patients with severe neurological impairment. Although some studies have reported significant improvement after the second course (e.g., Hadden et al., 1998), recent studies have refuted this result and showed no significant difference between first and second courses of IVIg (Walgaard et al., 2021).

Our study has several limitations. The first is due to its retrospective and monocentric character. Second, the use of the Brighton criteria as a diagnostic method in our study was questionable because the clinical criteria evaluation was only performed after the initiation of specific treatment of GBS by IVIg and neurological examination was performed on sedated patients, which could introduce a potential bias in the study. Regardless, our study has provided a new viewpoint about post-COVID-19 GBS patients and highlights the necessity to consider GBS in the ICU and the difficulties related to arriving at this diagnosis. Diagnosing GBS in the ICU remains difficult, especially when patients are sedated. Thus, clinicians should aim to diagnose GBS before ventilation weaning becomes difficult for COVID-19 patients. In addition, lumbar puncture should be performed as soon as possible and EMG should be performed to reduce the delay before beginning specific treatment. To help clinicians manage post-COVID-19 GBS, we propose the algorithm presented in Fig. 1.

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### Declaration of Competing Interest

None.

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**Fig. 1.** Algorithm proposal for the management of a clinical suspicion of Guillain-Barre syndrome in SARS-CoV-2 patients under mechanical ventilation.
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