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Unusual Presentation of COVID-19 Disease Leading to Respiratory Distress and Coma

Julie Paradis[1], Hendrik Kevers[1], Soufiane Chebli[1], Serge Blum[2], Pascal Reper[3,4]

[1] Internal Medicine, CHR Haute Senne, Soignies, Belgium

[2] Neurology, CHR Haute Senne, Soignies, Belgium

[3] Critical Care, CHR Haute Senne, Soignies, Belgium

[4] Critical Care department, CHU UCL Namur, Yvoir, Belgium

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ABSTRACT

Multiple neurological complications including Guillain-Barré syndrome (GBS) have been associated with COVID-19. We describe a case of GBS related to SARS-CoV-2 infection with an unusual presentation beginning with mobilization problems at home without previous classic respiratory or general manifestations. Asymptomatic infection with COVID-19 can lead to critical situations with respiratory insufficiency because of neurological complications such as GBS.

LEARNING POINTS

- Asymptomatic infection with COVID-19 can result in critical situations with respiratory insufficiency and need for mechanical ventilation because of neurological complications such as Guillain-Barré syndrome (GBS).
- All patients presenting with GBS should be tested for SARS-CoV-2.
- Neurological complications including neuromuscular impairment can be part of respiratory failure secondary to COVID-19 infection.

KEYWORDS

Coma, COVID-19, Guillain-Barré syndrome, respiratory failure

BACKGROUND

Hospital staff have dealt with the COVID-19 pandemic for the past year. The most frequent SARS-CoV-19 symptoms are respiratory with a wide spectrum ranging from cough to acute respiratory failure. However, neurological symptoms associated with COVID-19 infection have also been described [1]. Recently, there have been multiple reports of Guillain-Barré syndrome (GBS) associated with COVID-19 infection. Most cases of COVID-19-related GBS present with acute onset of areflexic quadriparesis following COVID-19 diagnosis. However, some important differences exist [2].

We describe a case of GBS related to SARS-CoV-2 infection with an unusual presentation beginning with mobilization problems at home without previous classic respiratory or general manifestations.

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CASE DESCRIPTION

A 71-year-old woman presented to the emergency room because of weakness in her lower limbs and paraesthesia in the hands and feet leading to difficulties walking and to falling in the last 48 hours. She had no relevant medical history, but had hypertension and hypothyroidism. Ten days before she presented, she had experienced a mild sore throat without fever. On admission, neurological examination showed areflexia in the lower limbs without any autonomic disorder or facial asymmetry. She had no respiratory symptoms, no fever and no cough. The chest and cerebral CT scans were normal. Cerebrospinal fluid (CSF) showed a typical albumin-cytological dissociation suggestive of GBS. On admission, the patient was tested for SARS-CoV-2 with a positive PCR nasopharyngeal swab. Blood serology was also positive for SARS-CoV-2, suggesting it was not a recent infection. No SARS-CoV-2 RNA was found in the CSF; antiganglioside antibodies in CSF were also negative. A few weeks previously family members had been diagnosed with COVID-19 infection with fever and typical gastrointestinal and upper respiratory symptoms.

Two days after admission the patient developed swallowing and speech disorders. The limb weakness evolved to flaccid symmetrical motor paralysis and sensory impairment associated with severe hypertension. She was admitted in the ICU because of impaired awareness and coma. She required immediate intubation for respiratory support because of severe hypercapnia and respiratory acidosis ($PaCO_2$ 134.9, pH 7.019, PaO_2 89 mmHg, face mask with FiO_2 60%). She received intravenous immunoglobins (IVIG) with little neurological improvement and so a second IVIG infusion was administered. The patient remained in the ICU for 6 weeks because of respiratory infection and weaning difficulties. A percutaneous tracheostomy was performed 5 weeks after admission. Electromyography showed major impairment of neuromuscular function with severe demyelinating polyneuropathy. The patient is currently recovering and receiving intensive rehabilitation.

DISCUSSION

Neurological complications associated with SARS-CoV-2 infection are being increasingly described. COVID-19 infection can cause multiple neurological problems such as headache, anosmia, ageusia, stroke and ischaemic cerebral complications [3, 4]. We know little about the neurotropism of this virus or the underlying mechanisms. Neurological manifestations of other beta coronaviruses (SARS and MERS) have been described and include polyneuropathy, myopathy, stroke and also GBS [1, 4, 5].

GBS is an acute immune-mediated demyelinating polyradiculopathy. Nerve damage is caused by an aberrant immune response to infection. Viruses and bacteria are both implicated in the pathogenesis of GBS. Most cases of GBS occur within 1–4 weeks of infection. Some pathogens such as *Campylobacter jejuni*, *Mycoplasma pneumoniae*, Herpes virus, Zika virus, CMV or EBV are more likely to cause GBS [3]. The SARS-CoV-2 virus attaches to the angiotensin-converting enzyme 2 receptor present in the cell membrane of numerous human organs, including lung, kidney, liver, nervous system and skeletal muscle. The pathogenesis of GBS in COVID-19 patients has not yet been investigated.

COVID-19 stimulates inflammatory cells to produce inflammatory cytokines and as a result, triggers immune-mediated processes. All GBS patients described in the literature were treated with IVIG with some receiving a second course of IVIG and others plasma exchange.

The interval between the onset of COVID-19 symptoms and the first symptoms of GBS ranged from 5 to 10 days in all published patients. Only a few COVID-19 patients with GBS had dysautonomic features. In all these patients, a real-time PCR assay of CSF was negative for SARS-CoV-2. Compound muscle action potential amplitudes on electrophysiological studies were low but could be obtained, and some patients had prolonged motor distal latencies. On electromyography, fibrillation potentials could also be detected in COVID-19 patients. The findings were generally consistent with an axonal variant of GBS in some patients and with a demyelinating process in others.

We describe GBS in one patient with COVID-19 without initial manifestations such as fever or respiratory symptoms. Typical COVID-19 symptoms were observed in her family members who had positive PCR tests. Our patient consulted for mobilization problems followed by rapid respiratory deterioration associated with coma and respiratory acidosis. She regularly presented symptoms of hypertension suggesting dysautonomic problems. Time to onset of GBS also seems short, as observed in other COVID-19 patients with GBS [2].

During this pandemic, severe respiratory insufficiency in stable or mildly symptomatic patients has been observed. Could this sudden respiratory deterioration be partially explained by neurological damage? Thromboembolic complications like pulmonary embolisms and oxygenation impairment leading to acute respiratory distress are major causes of respiratory insufficiency in COVID-19 patients, but neurological effects could also be responsible for acute respiratory deterioration and coma [5].

Prolonged weaning from artificial respiratory support is associated with pulmonary lesions and complications secondary to infection. Neuromuscular and neurological impairment can also have significant effects. Clinicians need to be aware of such neurological complications even if the previous infection is asymptomatic or mildly symptomatic. GBS with COVID-19 should be distinguished from critical illness neuropathy and myopathy, which appear later in the course of critical illness [2, 3].

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CONCLUSION

Asymptomatic infection with COVID-19 can result in critical situations with respiratory insufficiency requiring mechanical ventilation because of neurological complications such as GBS. All patients presenting with GBS syndrome should be tested for COVID-19.

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