## CASE STUDY

# Guillain-Barré syndrome after SARS-CoV-2 infection

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#### Introduction

Since the first reports in December 2019 in Wuhan, China, severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) has rapidly developed into a pandemic associated with substantial morbidity and mortality.

Guillain–Barré syndrome (GBS) is an acute immune-mediated polyradiculoneuropathy that may be triggered by various bacterial and viral infections. Reports on possible neurological manifestations of SARS-CoV-2 are still scarce. We report a case of GBS after an infection with SARS-CoV-2.

#### **Case presentation**

A 50-year-old male with no relevant medical history presented with 4 days of progressive bilateral facial weakness, paresthesia of distal extremities and an unsteady gait. Four weeks earlier he had experienced an episode of dry cough lasting several days without fever or other symptoms of infection.

Neurological examination showed facial diplegia, normal eye movements, mild symmetric proximal muscle weakness and impaired propriocepsis in the legs. The patient had an ataxic gait and tendon reflexes were absent. Routine blood examination showed no abnormalities. Routine analysis of cerebrospinal fluid showed a normal cell count and total protein level. Polymerase chain reaction for SARS-CoV-2 in the cerebrospinal fluid was negative. Fecal polymerase chain reaction and serum immunoglobulin (Ig)M and IgG for SARS-CoV-2 were all positive. Anti-GQ1b was negative. Serological tests for Borrelia burgdorferi, syphilis, Campylobacter jejuni, cytomegalovirus, hepatitis E, Mycoplasma pneumoniae and Epstein-Barr virus were all negative. Magnetic resonance imaging of the cerebrum was normal.

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Electromyography showed a sensorimotor, predominantly demyelinating polyradiculoneuropathy.

Due to progression of limb weakness and inability to walk, intravenous Ig (2 g/kg in 5 days) was initiated on day 7 of hospitalization and recovery started within days. On day 14 the patient was discharged with a mild proximal weakness in the lower extremities and facial diplegia.

#### Discussion

We describe a case of GBS 4 weeks after a SARS-CoV-2 infection. To our knowledge, this is one of the first cases of a GBS subtype after SARS-CoV-2 infection.

Guillain–Barré syndrome is considered a postinfectious disorder in which the infection may evoke an immune response to peripheral nerve antigens, via 'molecular mimicry' and other mechanisms, resulting in demyelination and/or axonal damage [1]. About two-thirds of GBS cases report an antecedent infection, particularly gastrointestinal tract and respiratory infections [1].

Previous studies report a peak in the incidence of GBS during epidemics of Zika and influenza viruses [2,3]. GBS has also been described after infection with other coronaviruses [4]. Zhao *et al.*[5] recently reported a patient with GBS developing symptoms of SARS-CoV-2 7 days after onset of neurological symptoms. This sequence of events may argue against a post-infectious pathogenesis, although the incubation period of the infection in this case is unknown [5]. Other patients have been described who developed GBS 3–10 days after the first symptoms of SARS-CoV-2 infection, like the current case, which is more in line with the typical sequence of a post-infectious immune-mediated disorder [6,7

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Our patient reported an episode of dry cough 3– 4 weeks before admission, which is a classical presentation of a mild SARS-CoV-2 infection. Positive IgM and IgG serology for SARS-CoV-2 confirmed the diagnosis. The latency period of 3–4 weeks after

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infection and negative SARS-CoV-2 polymerase chain reaction in cerebrospinal fluid militates against a direct infection of the nervous system and supports a post-infectious immune-mediated pathogenesis [1,2].

It is remarkable that all published cases show different clinical features, suggesting a heterogeneous immunological response. Tested anti-ganglioside antibodies were negative in the current and most previously reported cases, but these antibodies are usually absent in GBS preceded by viral infections [1].

In the absence of the required studies to establish if SARS-CoV-2 can trigger GBS, clinicians should be aware that the current SARS-CoV-2 pandemic may lead to more cases of GBS. Diagnosis may be hampered by the seemingly heterogeneous presentation. Optimal treatment for these patients is yet to be determined, although our patient showed a rather fast and good response after treatment with intravenous Ig.

## **Disclosure of conflicts of interest**

The authors declare no financial or other conflicts of interest relevant to the article.

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