

Case report

Guillain-Barré syndrome associated with COVID-19 infection: a case from the UK

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SUMMARY

Originating from Wuhan, China, COVID-19 has rapidly spread worldwide. Neurological manifestations are more commonly associated with severe COVID-19 infection. Guillain-Barré syndrome (GBS) is a rare immune-mediated postinfectious neuropathy. It has been reported as a possible rare complication of COVID-19. We report a case of GBS associated with COVID-19 in the UK.

BACKGROUND

Severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2) is typically associated with respiratory disease and fever. Originating from Wuhan, China, COVID-19 has rapidly spread worldwide. COVID-19 commonly presents with respiratory symptoms with mild clinical features in the majority of patients. The neurological manifestations of COVID-19 include headache, stroke, seizures, encephalitis, hypogeusia and neuralgia.¹⁻³

Guillain-Barré syndrome (GBS) is a rare immune-mediated postinfectious neuropathy typically leading to ascending weakness. It has been reported as a possible rare sequela of COVID-19,⁴⁻⁶ though the first reported case in Wuhan suggested a parainfectious presentation.⁷ We report a case of GBS associated with mild COVID-19 infection in the UK.

CASE PRESENTATION

A 49-year-old man of South Asian descent with no medical history, apart from sinusitis, presented with difficulty in mobilising. There was no recent travel history. He reported a 3-week history of shortness of breath, headache and cough. He had completed two courses of antibiotics in view of his background of recurrent sinusitis. On initial presentation to the emergency department, he reported worsening cough and distal lower limb paraesthesia during the previous week, resulting in difficulty mobilising, requiring the use of a frame. He was apyrexial on presentation with a mild tachycardia of 109, normal oxygen saturation of 99% on room air and haemodynamically stable. He had an elevated lactate on venous blood gas (3.3 mmol/L) and a mildly elevated C-reactive protein (20 mg/L). His white cell count, sodium, potassium and renal function and liver function were normal. His oropharyngeal swabs tested positive for SARS-CoV-2 on RT-PCR. His family members were self-isolating and had no symptoms of COVID-19. He had a mildly elevated creatine kinase (CK) of 589 IU/L,

which led to an initial diagnosis of viral myositis and was discharged.

The patient represented 3 days later with worsening lower limb paraesthesia and unable to mobilise due to ascending lower limb weakness. His repeat CK was normal (151 IU/L). Four days later, he had developed facial diplegia, weakness of Medical Research Council (MRC) Muscle Scale 1/5 in his lower limbs, 3/5 proximal upper limbs and 2/5 distal upper limbs. He had distal reduced sensation to pinprick and vibration sense and was areflexic. There was no meningism, spinal cord sensory level, bladder or bowel involvement or dysautonomia. He developed dysaesthesia in his lower limbs which responded well to neuropathic medication, pregabalin.

INVESTIGATIONS

His cerebrospinal fluid (CSF) showed cytoalbuminologic dissociation (protein >1.25 g/L, white cell count 1×10^6 /L). His CSF was negative for SARS-CoV-2. He continued to test positive for SARS-CoV-2 on oropharyngeal swabs for 4 weeks after initial presentation. His neurophysiology studies showed absent sensory response of the median nerve (table 1A) and severe slowing of motor responses of the median and ulnar nerve with reduced amplitude (table 1B). He had severe neurogenic firing with normal motor units, without spontaneous activity, on electromyography (table 1C), indicating a demyelinating polyneuropathy. Meeting all domains of the Brighton criteria,⁸ a diagnosis of GBS was confirmed.

TREATMENT

During the initial week of admission, the patient was monitored in intensive care and required a nasogastric tube for feeding due to swallowing difficulties, without respiratory dysfunction. He received intravenous immunoglobulin (IVIG) 0.4 g/kg daily for 5 days and his symptoms stabilised, avoiding the requirement of intubation.

OUTCOME AND FOLLOW-UP

The patient's motor symptoms began to improve within the first week of IVIG administration. Four weeks later, the patient no longer had facial diplegia and his motor strength had improved to 4/5 proximally, 3/5 distally and lower limbs 3/5. Eleven weeks later, after receiving neurorehabilitation and optimisation of neuropathic medications, the patient was able to mobilise unassisted.



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Table 1 Neurophysiology results

(A) Nerve	Latency (ms)	Amplitude (mV)	Velocity (m/s)
Right median	Not excitable (≤ 3.5)	Not excitable (≥ 20)	Not excitable (≥ 50)
Right ulnar	2.29 (≤ 3.1)	0.51 (≥ 17)	41.5 (≥ 50)
Right radial	1.98 (≤ 2.9)	27.5 (≥ 15)	50.5 (≥ 50)
Right sural	3.07 (≤ 4.4)	14.3 (≥ 6)	45.6 (≥ 40)

(A) Sensory nerve conduction studies (NCS).

(B) Nerve	Latency (ms)	Amplitude (mV)	Velocity (m/s)
Right median	24.48 (≤ 4.4)	2.3 (≥ 4.0)	28.1 (≥ 49)
Right ulnar	10.52 (≤ 3.3)	3.9 (≥ 6.0)	37.3 (≥ 49)
Right tibial	25.52 (≤ 5.8)	0.4 (≥ 4.0)	21.5 (≥ 41)

(B) Motor NCS.

(C) Muscle	Spontaneous activity			Motor units				Recruitment	
	Insertion	Fib	PSW	Fasc	Firing	Amp	Dur		Poly.
Right vastus lateralis	None	None	None	None	3+	Norm	Norm	None	Discrete
Interpretation	Can be prolonged in denervation	Few fibs and PSW can be normal but profuse in complete denervation		Can be present in normal muscle and denervation	Rapid firing in denervation	Can be normal in acute nerve lesions	Can be normal in acute nerve lesions	None	Full interference in Healthy muscle Discrete recruitment in denervation as more motor units drop out

(C) Needle electromyography. Reference range in brackets.
Amp, amplitude; Dur, duration; Fasc, fasciculations; Fib, fibrillation; Norm, normal; Poly, polyphasic; PSW, positive sharp waves.

Patient's perspective

A week or so prior to the lockdown, I came down with sinusitis which for me is a yearly occurrence. Nonetheless, I followed the governments suggested self-isolation protocol by remaining in my room upstairs for the couple of weeks period as my brother and father are highly vulnerable to catching infections due to their health conditions.

Towards the end of my isolation, my legs and arms began to become stiff and painful to move, making an everyday task such as walking difficult. As a person who enjoys weekly sessions of badminton and gym workouts, this sudden inability to move my limbs came as an utter shock as I never imagined something like this happening to myself, as I feel that I am quite fit and active.

On the 8th of April I called an ambulance in the morning and proceeded to lug myself downstairs slowly in excruciating pain, after which I was taken to A&E for a check. After my examination I was discharged with a bit of treatment. When being collected from the hospital by a family member, I was unable to stand up from the wheelchair I was in and get into the car. Two security guards had to haul me in.

On arrival home, I got out of the car with a Zimmer frame and only meters away from the door, my legs lost all strength and gave way causing me to collapse in the driveway. I spent a few hours with the help of the family trying to get up and into the house but I was not able to get up for myself and as a result my family could not lift my dead weight. As a result of this, an ambulance was called out and took me to A&E for me to only be sent home again, with no explanation to the loss of strength and mobility of my limbs.

As my mobility condition worsened, I had no choice but to sleep and live downstairs in the living room which is adjacent to my brother's room and along with my father who also sleeps downstairs. This was a big concern for me, as I still had signs of an infection. Throughout the next 2 days my mobility further worsened, meaning I was unable to sit up from lying in bed, my ability to grasp objects in my hand had lessened, I was not able to stand up from a sitting position and many more.

These all increased the difficulty of performing normal tasks especially going to the toilet, which had to be done with the help of my family yet again. My family were forced to lift me out of bed and wrestle me onto a commode before I could proceed to be taken to the toilet. Where the process had to be reversed to get onto the toilet seat. Once completed, as before, the strenuous task of getting back to bed had to be achieved.

On the following day, due to a fall, another ambulance had been called in which claims of me potentially having COVID-19 had been made. And yet again was forced in a self-isolation. Despite this I was still unable to perform tasks, so my family had to come to my aid with gloves and masks on.

On the early morning hours of the 11th of April, I awoke with extreme discomfort, weakness and a complete loss of energy. I rang the 111-advice service in which the verbal examination indicated that I required hospital support. And the 111 workers requested an ambulance to be sent out. In the wait of the ambulance I began to lose consciousness, fortunately my family were able to keep me aware. The ambulance arrived only moments after. In which it took four personnel to assist me in getting out of bed and onto the wheelchair.

I was examined for the third time and was diagnosed with the Coronavirus. Doctors claimed that the virus had attacked my nervous system and had damaged it. This became a concern

Continued

Patient's perspective Continued

for the doctors, as prospects that the nerves in my respiratory system could also be attacked, reducing my own ability to breathe. As a precaution I was transferred into the ICU.

Over the coming weeks numerous tests and treatments were administered to help cure me. With recent news that my condition is noted as new and unseen. I have written this passage depicting my struggles.

I am still in hospital and am in the process of a recovery, even though it may be a slow one. This experience has been extremely frightening with the constant apprehension that my life might never be the same again. Among the fight it has also been an extremely painful and disconcerting process as I have never experienced any illness first-hand in my entire life.

My utmost gratitude goes towards those who have helped me and continue to help me in my recovery of the illness especially all the workers of the NHS.

DISCUSSION

The first reported case of COVID-19-associated GBS was reported from Wuhan as a suspected parainfectious disease, as the patient developed COVID-19 symptoms 7 days after the onset of GBS symptoms.⁷ Here, we have presented a patient from the UK who developed GBS as a likely postinfectious complication 3 weeks after the initial onset of infectious symptoms. Our case supports increasing numbers of reports of COVID-19-associated autoimmune neuropathies,^{5 6} including the Miller-Fisher variant of GBS.⁹ As with reports from Italy, our patient was highly responsive to immunoglobulin therapy,¹⁰ emphasising the importance of early recognition and treatment.

Toscano *et al* report five cases of COVID-19 who developed GBS 5 to 10 days post infection; all five cases had negative PCR for SARS-CoV-2 in CSF. Our patient remained positive for SARS-CoV-2 for approximately 7 weeks post onset of initial COVID-19 symptoms, supporting previous reports of RT-PCR remaining positive in patients who have fully recovered from COVID-19, which raises questions about the duration of viral shedding.¹¹ Neurological manifestations of COVID-19 is often associated with severe acute respiratory distress syndrome but our patient presented with neurological sequelae following mild COVID-19 symptoms. Despite neurological sequelae, RNA may not be detected in CSF,^{4 12} as in

Learning points

- ▶ Neurological complications are more commonly associated with severe COVID-19 symptoms but postinfectious neuropathies can occur even following mild COVID-19 infections.
- ▶ Guillain-Barré syndrome (GBS) is a rare but serious complication of severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2) infection and should not be overlooked as viral-associated myositis. Early reports suggest a positive response to intravenous immunoglobulin therapy.
- ▶ SARS-CoV-2 PCR may be negative in cerebrospinal fluid but SARS-CoV-2 PCR from oropharyngeal swabs can remain positive for several weeks after initial COVID-19 symptoms.
- ▶ Early recognition of COVID-19-associated GBS is vital to initiate immunoglobulin therapy.

our case; though there has been a reported case of encephalitis with SARS-CoV-2 detected in CSF but not nasopharyngeal swabs.¹² GBS has severe consequences and early recognition is vital to monitor for loss of ambulation and initiation of immunoglobulin treatment. Patients presenting with paresthesia and difficulty in mobilising after COVID-19 symptoms should not be overlooked as viral-associated myositis. GBS should be considered as a potential rare but serious complication weeks after initial COVID-19 infection.

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