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Correspondence

Guillain-Barré syndrome in the early postpartum period following COVID-19 infection


There are a growing number of reports of neurological sequelae from coronavirus disease-2019 (COVID-19) caused by the severe acute respiratory syndrome-CoV-2 (SARS-CoV-2) virus.^{1,2} We present a 30-year-old woman who developed Guillain-Barré syndrome (GBS) in the early postpartum period after recovering from severe COVID-19.

The patient was referred to our emergency department with an intra-uterine fetal death at 34 weeks' gestation. She was a known type-2 diabetic. At presentation, she was confused, had a heart rate of 130 bpm, blood pressure of 130/80 mmHg, respiratory rate of 50 breaths/min and peripheral oxygen saturation (SpO₂) of 75% in room air. She had a history of fever and cough for seven days before admission, and had developed shortness of breath and confusion one day before admission. Initial laboratory findings confirmed the presence of diabetic ketoacidosis. She was treated for presumed COVID-19 and initially managed with oxygen therapy and non-invasive ventilation but rapidly deteriorated, requiring intubation and ventilation. The decision was made to proceed to caesarean delivery. Postoperatively, the patient was transferred to the intensive care unit for continued respiratory support and COVID-19 was confirmed by a reverse-transcriptase polymerase chain reaction (RT-PCR) from a nasopharyngeal swab. Chest computed tomography revealed bilateral lung infiltrates affecting 75% of the lung parenchyma and consistent with COVID-19. The patient received methylprednisolone, hydroxychloroquine, ceftriaxone and a therapeutic dose of enoxaparin. Her condition gradually improved, and on day five after delivery she was extubated. By day nine, she was ambulant with an SpO₂ of 94% in room air.

The patient then developed a circumoral vesicular lesion that was attributed to herpes zoster infection (based on clinical assessment by the attending dermatologist) and was treated with oral and topical aciclovir. On day 10 a repeat nasopharyngeal swab was negative for SARS-CoV-2 and by the end of the same day, the patient had developed bilateral lower limb weakness which progressed to tetraparesis over the next three days. Neurological examination revealed flaccid tetraparesis, absent deep tendon reflexes and no bulbar involvement. Cerebrospinal fluid analysis showed a mildly elevated protein level (total protein 60 g/L, albumin 36 g/L) with mild pleocytosis (white cell count 10 000/μL). Five days following the onset of muscle weakness, the patient could not expectorate sputum, her SpO₂ fell to 85% and she was intubated and ventilated. Electromyography examination and nerve conduction studies of the upper and lower limbs showed early signs of demyelinating sensorimotor polyneuropathy (GBS). The patient received 1 g methylprednisolone and plasmapheresis

was scheduled. However, she became hemodynamically unstable and a norepinephrine infusion was started. Despite supportive respiratory and cardiovascular treatment, the patient progressively deteriorated and died one day later.

Guillain-Barré syndrome is usually preceded by an infection that triggers an abnormal autoimmune response affecting the peripheral nerves and their spinal roots.³ There are a few cases now described of GBS in patients with COVID-19.^{2,4,5} In the present case, the onset of neurologic symptoms was relatively delayed (17 days) compared with previous reports and was after the patient had a negative RT-PCR for SARS-CoV-2. This late presentation might have been due to the increased risk of autoimmune disease in the postpartum period⁶ and the case is in keeping with the post-infection presentation of GBS observed with other viral infections.³ Guillain-Barré syndrome has been reported following herpes zoster infection, with the interval between the onset of skin rash and weakness ranging between 3 and 42 days.⁷ In our patient the interval was only one day so we believe that SARS-CoV-2 was the more likely trigger.

Guillain-Barré syndrome is a rare but serious post-infective neuropathy with a reported mortality of 3–13%.⁸ In our patient the progressive respiratory muscle weakness and inability to cough led to retention of secretions and pneumonia. The patient had suffered from a rapid deterioration from severe sepsis. Although GBS has been associated with COVID-19 previously, this case highlights the possibility of developing GBS despite a negative RT-PCR. Patients should be instructed to seek medical advice urgently if they develop neurological symptoms after recovery from COVID-19.

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