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Clinical Letter

Guillain-Barré Syndrome After COVID-19 Vaccination in an Adolescent

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Guillain-Barré syndrome (GBS) has been associated with SARS-CoV-2 infection in adults and children,[1] and it has been noted as an adverse effect with the Janssen COVID-19 vaccine in adults.[2] There have been no cases reported of children developing GBS after COVID-19 vaccination, to our knowledge.

We describe a child who developed GBS within one month of the administration of the second dose of the Pfizer-BioNTech COVID-19 vaccine. Our patient is a 14-year-old male who received the second dose of the Pfizer-BioNTech COVID-19 vaccine on June 11, 2021. He had previously never been diagnosed with COVID-19. On July 3, he experienced left lower extremity swelling up to the knee after a probable bee sting to the bottom of his left second toe, which resolved within two weeks without treatment. On July 11, he reported subjective facial weakness and subjective tongue swelling, for which he received oral prednisone at an urgent care facility. Owing to increasing facial weakness, he was evaluated in the emergency department on July 14, and he underwent diagnostic testing for common causes of facial palsy. No other weakness was reported on examination at that time. He was admitted on July 19 for progressive facial and limb weakness with areflexia. COVID-19 antigen testing was negative upon admission. On examination, he had significant difficulty ambulating, bilateral facial weakness worse on the left, and 4+/5 strength throughout the left hemibody but preserved strength on the right side. Over the next few days, he became quadriparetic and was unable to ambulate independently. Breathing was never impaired.

This child's diagnosis of GBS was confirmed through clinical presentation; cerebrospinal fluid showing 4 white blood cells and 165 mg/dL protein, indicating cytoalbuminocytologic dissociation; and electrodiagnostic studies demonstrating a severe, generalized polyradiculoneuropathy, with demyelinating features indicating the acute inflammatory demyelinating polyradiculoneuropathy variant of GBS. Although this patient did not have any sensory losses, both acute inflammatory demyelinating polyradiculoneuropathy and the acute motor axonal neuropathy variants can cause pure motor symptoms.[3] Additional cerebrospinal fluid and serum testing excluded alternative etiologies, including negative Lyme antibodies, Lyme polymerase chain reaction, and ganglioside antibodies.

Our patient was treated with 2 g/kg IVIg over 3 days. Beginning on the third day of his IVIg course, he demonstrated marked improvement in his facial and limb weakness. Before discharge, he was able to ambulate with assistance and went home with outpatient physical therapy seven days after admission. At his follow-up appointment in mid-August, he reported full resolution

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