

## LETTER TO THE EDITOR

# SARS-CoV-2 infection in two pediatric patients with immune cytopenias: A single institution experience during the pandemic

To the Editor:

In pediatrics, viral infections precede approximately 60% of cases of immune thrombocytopenia (ITP)<sup>1</sup> and 15% of cases of autoimmune hemolytic anemia (AIHA).<sup>2</sup> Here, we describe two previously healthy children, each diagnosed with an autoimmune cytopenia associated temporarily with SARS-CoV-2 viral infection, one with acute ITP and one with AIHA.

First, a 16-year-old male presented with rash and mouth sores. His parents both reported flu-like symptoms 3-4 weeks prior for which they home-quarantined. The patient's vital signs were normal, with exam notable for diffuse petechiae throughout skin and wet purpura of oral mucosa. Laboratory studies demonstrated an isolated low platelet count of 45 000/ $\mu$ L. The patient was diagnosed with acute ITP and discharged home with a scheduled follow up, as current guidelines recommend observation only in patients with platelet count  $\geq$ 10 000/ $\mu$ L, no symptoms, or only minor mucocutaneous bleeding.<sup>3</sup> The following day, his petechiae and purpura worsened with platelet count measuring 4000/ $\mu$ L. Given possible exposure, he underwent COVID-19 serology testing, which returned positive. Hospital guidelines prevented SARS-CoV-2 polymerase chain reaction (PCR) testing. He began corticosteroid treatment for ITP with clinical improvement, and platelet count increased to 73 000/ $\mu$ L 1 week later.

Second, a 14-year-old female presented with fever, headache, and fatigue with myalgia, vomiting, and abdominal pain. With normal vital signs and an unremarkable physical exam, an abdominal ultrasound demonstrated mild splenomegaly. Laboratory results included: hemoglobin 4.0 g/dL; reticulocyte count 17.7%; haptoglobin  $<$ 10 g/dL; and lactate dehydrogenase (LDH) 367 U/L with a positive direct antiglobulin test for anti-IgG and anti-C3bC3d. Antibody identification detected a mixed cold agglutinin and a warm autoantibody, suggesting a mixed-type AIHA. An initial SARS-CoV-2 PCR by nasopharyngeal swab was negative. She was transfused warmed, packed red blood cells and started on corticosteroids. On discharge, hemoglobin levels gradually increased to 9.7 g/dL, then declined to 7.5 g/dL with labs showing the presence of an interfering substance, suggestive of an autoantibody leading to persistent red cell destruction. She started on weekly rituximab. During treatment, she disclosed a household member had been symptomatic and COVID-19 positive several weeks prior. The patient underwent repeat SARS-CoV-2 testing with a positive result by PCR without COVID-19-associated symptoms. Following a 4-week course of rituximab, hemoglobin improved to 10.8 g/dL, with normalization of LDH and reticulocyte count.

A few case reports of adults describe autoimmune cytopenias secondary to SARS-CoV-2 infections, including a report of seven patients with AIHA,<sup>4</sup> and one with ITP.<sup>5</sup> Until now, no cases of SARS-CoV-2-induced autoimmune cytopenias have been reported in pediatric patients.

In our cases, neither patient exhibited symptoms of acute infection with SARS-CoV-2 prior to or at the time of presentation. While we know the immune dysregulation by this virus can cause severe phenotype, such as the recently described multisystem inflammatory syndrome in children (MIS-C), there may be other less severe phenotypes, such as autoimmune cytopenias, which may eventually fall under the spectrum of MIS-C but have not yet been defined.<sup>6</sup> This suggests utility in COVID-19 testing in pediatric patients who present with autoimmune cytopenias during this pandemic.

## CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

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