Abstract: Multisystem inflammatory syndrome (MIS) in children is a severe illness characterized by fever, laboratory evidence of inflammation, and multisystem organ dysfunction resulting from severe acute respiratory syndrome coronavirus 2 infection in a patient younger than 21 years. We present the case of a 39-year-old man with evidence of prior COVID-19 who seemed to meet all non-age-related criteria for MIS in children as well as criteria for the working definition of MIS in adults, and who improved after treatment with aspirin, corticosteroids, and intravenous immunoglobulin. Clinicians should be aware of this new inflammatory illness, not only in children but potentially also in adults with antecedent or concurrent COVID-19.

Key Words: SARS-CoV-2, COVID, coronavirus, inflammation, MIS, case report, multisystem inflammatory syndrome

Infect Dis Clin Pract 2021;29: e174–e176

Multisystem inflammatory syndrome (MIS) has previously been described in children during or after COVID-191,2 but has not been well characterized in adults, with very few cases reported globally to date.3–5 This report describes an adult with a severe illness characterized by fever, laboratory evidence of inflammation, and multisystem organ dysfunction without severe respiratory involvement in the context of COVID-19 who improved with aspirin, corticosteroids, and intravenous immunoglobulin.

CASE PRESENTATION

A 39-year-old African American man with diabetes mellitus type 2 presented to the emergency department in July 2020 with 3 days of fever, fatigue, myalgias, sore throat, dyspnea, vomiting, and diaphoresis. He reported that 2 of his neighbors with whom he had contact were diagnosed with COVID-19 in the 10 days before presentation.

At the time of presentation to the emergency department, the patient appeared ill but was not in distress. He was febrile (temperature, 39.4°C), tachycardic (pulse, 127 beats per minute), and tachypneic (respiratory rate, 27 breaths per minute). His systolic blood pressure was 134 mm Hg, and his diastolic blood pressure was 95 mm Hg. His peripheral capillary oxygen saturation was 98%. He exhibited dry mucus membranes with no pharyngeal erythema or exudates and mild cervical lymphadenopathy. His lungs were clear to auscultation. His abdomen was nontender and nondistended. He had a confluent, blanching rash on his chest and back. Pertinent laboratory studies on presentation are presented in Table 1. Of note, his C-reactive protein level was 366 mg/L, and his ferritin level was 1907 ng/mL. Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) was not detected by polymerase chain reaction (PCR) on nasopharyngeal swab using the DiaSorin LIAISON platform. A chest radiograph demonstrated no infiltrates. Computed tomography angiography of the chest showed no pulmonary embolism and mild bibasilar atelectasis. The patient's glucose was 403 (hemoglobin Alc was 9.2%), but the venous blood gas (pH 7.44 and Pco2 35 mm Hg) and serum beta hydroxybutyrate (0.46 mmol/L) were not consistent with diabetic ketoacidosis. Vancomycin and cefepime were administered, and he was admitted to the intensive care unit for persistent tachycardia. SARS-CoV-2 was not detected by PCR on a second nasopharyngeal swab using the platform developed by the Centers for Disease Control and Prevention.6

On hospital day 2, the patient developed a petechial rash on his left forearm. Doxycycline was administered for possible tickborne illness. All other antimicrobials were discontinued when blood cultures remained without growth at 48 hours. The PCR test for respiratory pathogens from a nasal swab and for Clostridioides difficile from a stool sample were both negative. Rocky Mountain spotted fever antibody, Ehrlichia/Anaplasma PCR, cytomegalovirus PCR, Epstein-Barr virus PCR, urine Legionella antigen, anti-nuclear antibody, and rheumatoid factor were negative.

On hospital day 4, the patient experienced new confusion. Magnetic resonance imaging (MRI) of the brain showed scattered cerebral white matter T2/flair hyperintensities potentially consistent with vasculitis or chronic microvascular changes. He experienced episodes of regular narrow complex tachycardia with heart rate of 130 to 140 beats per minute, for which he was administered metoprolol. An electrocardiogram demonstrated diffuse ST segment elevations. His troponin I increased to 1.43 ng/mL and decreased thereafter. Transthoracic echocardiogram was normal, and cardiac MRI showed increased T1 signal, suggestive of myocardial edema or inflammation. Given his elevated troponin and diffuse ST segment elevations, this study supported a clinical diagnosis of myopericarditis. SARS-CoV-2 was not detected by PCR on a third nasopharyngeal swab using the Centers for Disease Control and Prevention platform.

At this time, his immunoglobulin G (IgG) to SARS-CoV-2 was found to be positive using the Abbott Architect SARS-CoV-2 IgG assay, which has demonstrated a specificity of 99.9%.7 Because his systemic inflammation, myopericarditis, hepatitis/hyperbilirubinemia, erythematous rash on trunk and arms, and possible neurologic involvement with alteration in mental status resembled the manifestations of the MIS in children (MIS-C), he was administered 5 mg/kg (325 mg) of
## DISCUSSION

Multisystem inflammatory syndrome in children is a recently described febrile hyperinflammatory illness associated with antecedent or concurrent COVID-19. Criteria to establish a diagnosis of MIS-C include serious illness requiring hospitalization, fever (temperature, >38.0°C) or subjective fever lasting 24 hours or more, laboratory evidence of inflammation, multisystem organ involvement, laboratory-confirmed SARS-CoV-2 infection (PCR or IgG) or a known link to a person with COVID-19 in the preceding 48 hours, and he was discharged home after a total of 10 days.

### ACKNOWLEDGMENTS

The authors would like to thank the patient.

### REFERENCES

1. Levin M. Childhood multisystem inflammatory syndrome—a new challenge in the pandemic. N Engl J Med. 2020;383(4):393–395.
2. Feldstein LR, Rose EB, Horwitz SM, et al. Multisystem inflammatory syndrome in U.S. children and adolescents. N Engl J Med. 2020;383(4):334–346.
3. Shaigany S, Gnirke M, Guttmann A, et al. An adult with Kawasaki-like multisystem inflammatory syndrome associated with COVID-19. *Lancet*. 2020;396(10246):e8–e10.

4. Sokolovsky S, Soni P, Hoffman T, et al. COVID-19 associated Kawasaki-like multisystem inflammatory disease in an adult. *Am J Emerg Med*. 2020;39:253.e1–253.e2.

5. Morris SB, Schwartz NG, Patel P, et al. Case series of multisystem inflammatory syndrome in adults associated with SARS-CoV-2 infection—United Kingdom and United States, March-August 2020. *MMWR Morb Mortal Wkly Rep*. 2020;69:1450–1456.

6. Centers for Disease Control and Prevention. CDC’s Diagnostic Test for COVID-19 Only and Supplies. Available at: https://www.cdc.gov/coronavirus/2019-ncov/lab/virus-requests.html. Published 2020. Accessed October 30, 2020.

7. Bryan A, Pepper G, Wener MH, et al. Performance characteristics of the Abbott Architect SARS-CoV-2 IgG assay and seroprevalence in Boise, Idaho. *J Clin Microbiol*. 2020;58(8):e00941-20.

8. Puntmann VO, Carej ML, Wieters I, et al. Outcomes of cardiovascular magnetic resonance imaging in patients recently recovered from coronavirus disease 2019 (COVID-19). *JAMA Cardiol*. 2020;5:1265–1273.