DOI: 10.1002/pbc.28443

LETTER TO THE EDITOR



COVID-19 in a child with severe aplastic anemia

Since the beginning of the COVID-19 outbreak, there has been a paucity of data on the course of the disease in children with primary or acquired immunodeficiency.¹⁻⁶ In this report, we describe the novel case of a child with aplastic anemia affected with COVID-19.

Revised: 11 May 2020

A 14-year-old boy, who has been followed with a diagnosis of severe aplastic anemia at our hematology department, presented with fever, sore throat, and epistaxis for 2 days. Since he had a fully matched sibling donor and was planned to undergo hematopoietic stem cell transplantation, immunosuppressive treatment had not been administered. At his previous admission, his hemoglobin was 7.7 g/dL, platelet count was 9×10^9 /L, and white blood cell count was 1.34×10^9 /L with absolute neutrophil, lymphocyte, and monocyte counts of 0.17×10^9 /L, 1.02×10^9 /L, and 0.10×10^9 /L, respectively. He had had a history of exposure with a person suspected to have COVID-19. At admission, his body temperature was 38.5°C and hyperemia of the oropharynx was noted. He had no tachypnea or dyspnea; no rales or rhonchi were heard on auscultation of the lungs. Oxygen saturation was normal (>95%) in room air. Complete blood count revealed white blood cell count of 1.70×10^{9} /L, absolute neutrophil count of 0.19×10^{9} /L, absolute lymphocyte count of 1.40×10^9 /L, absolute monocyte count of 0.06×10^{9} /L, hemoglobin 8.3 g/dL, and platelet count 12×10^{9} /L. C-reactive protein level was 0.00221 g/L. The anteroposterior chest radiograph revealed bilateral minimal paracardiac infiltration. Highresolution computed tomography of the chest revealed no abnormality including infiltrate, consolidation, or ground-glass opacities. Febrile neutropenia treatment with cefepime was started and platelet suspension was transfused. The test for severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) by quantitative real-time reverse transcription polymerase chain reaction (QRT-PCR) from combined nasal and oropharyngeal swab specimens was positive. His body temperature was normalized on the second day of hospitalization. His condition never worsened and oxygen saturation never decreased. Regular erythrocyte and platelet transfusion program were continued. No microorganism was isolated in peripheral blood cultures. Following twice-negative repeat QRT-PCR testing, the patient was discharged on the 15th day of hospitalization in good condition.

Cases of COVID-19 in age <20 years comprise around 2% of the infected population and mortality in this age group is very rare.¹⁻³ There is an inverse relationship with the age of the child and severity of the disease,³ the reasons for which are still not established. Unlike older age and comorbidities including diabetes, hypertension, obesity, and smoking, there is a paucity of data on the impact of immunodeficiency states as a risk factor on the prognosis of COVID-19.⁷ Filocamo et al reported that children treated with immunosuppressive drugs

for rheumatologic disorders do not have an increased risk of respiratory or life-threatening complications of COVID-19 compared with the general population.⁸ In a systematic review, 110 immunosuppressed patients, including a few children with immunodeficiency or posttransplantation, were reported to have an overall better outcome when compared with other comorbidities.⁹ We speculate that the immunocompromised state might be a favorable factor for better prognosis by limiting the inflammatory reaction, which is commonly associated with the severity of disease. Such a speculation is contrary to the general opinion that immunocompromised state increases the risk of infection, but, indeed, is paradoxically protective.^{7,9,10}

In our patient, we speculate that both younger age and the immunocompromised state are the two contributing factors for the very mild disease. However, further reports with larger sample sizes are warranted to delineate the impact of immunocompromised states on the prognosis of children affected with COVID-19.

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