Pericardial effusion secondary to COVID-19 infection

Selman Gokalp1, Erman Çilsal2, Bekir Yukcu1, Canan Yolcu3, Gulsen Akkoc4 and Alper Guzeltas1

1Department of Pediatric Cardiology, University of Health Sciences, Istanbul Mehmet Akif Ersoy Thoracic and Cardiovascular Surgery Training and Research Hospital, Istanbul, Turkey; 2Department of Pediatric Cardiology, Adana Numune Training and Research Hospital, Yuregir, Adana, Turkey; 3Department of Pediatric Cardiology, University of Health Sciences, Istanbul Haseki Education and Research Hospital, Istanbul, Turkey and 4Department of Pediatric Infectious Diseases, University of Health Sciences, Istanbul Haseki Education and Research Hospital, Istanbul, Turkey

Dear Editor,

We thank Mungmunpuntipantip and Wiwanitkit for their interest in our manuscript “A case of a very large haemorrhagic pericardial effusion in an adolescent patient with COVID-19 infection.”. Authors claim that pericardial effusion in patients with COVID-19 is typically not a haemorrhagic effusion. Our knowledge about COVID-19 infection grows each day; we learn different clinical entities related to this infection. As shown by many other case reports published either previously or later than our manuscript, pericardial effusion secondary to COVID-19 infection might be either haemorrhagic1,2 or serohaemorrhagic in nature.3-6 Our patient’s platelet count was 352 × 10^9/L, coagulation studies revealed prothrombin time 14.4 s, activated partial thromboplastin time 22.3 s, an international normalised ratio of 1.23. Fibrinogen and D-dimer were 440 mg/dl and 1.81 μg/ml, respectively. If the patient has an underlying haemostatic disorder, he probably had an associated haemorrhage. Also, developing hemopericardium without any other bleeding problems would be an unexpected clinical finding. Since the patient had no mucocutaneous bleeding or other reasons to suspect a haemostasis disorder, we did not need a complete laboratory workup.

We appreciated their acknowledgement of tuberculosis as a possible cause of the pericardial effusion. Our manuscript has already mentioned that the patient’s BCG scar was positive, and the tuberculin skin test was negative. As a developing country, we consider tuberculosis infection in every patient with pneumonia or pleural effusion. The patients’ pericardial fluid tested negative for acid-fast bacilli smear, and culture for tuberculosis was negative.

The diagnosis of myopericarditis is usually based on laboratory tests confirming or excluding possible aetiologies and infections. In our case, after excluding all other possible causes and confirmatory laboratory tests for COVID-19, it should be logical to accept the pericardial effusion secondary to COVID-19 infection rather than a coincidence.

Acknowledgements. None.

Financial support. This research received no specific grant from any funding agency, commercial, or not-for-profit sectors.

Conflicts of interest. None.

Ethical approval. All procedures performed in this case were in accordance with the ethical standards of the institutional and national research committee and with the 1975 Helsinki declaration and its later amendments or comparable ethical standards.

Informed consent. Informed consent was obtained from the patient and the patients’ parents.

References