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Letter to the Editor

How to clarify the mechanism of multi-systemic inflammatory syndrome related to COVID-19

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To the editor:

Carrasco-Molina and colleagues presented two patients who developed multi-systemic inflammatory syndrome in adults (MIS-A), a recently-proposed entity associated with coronavirus disease 2019, which was successfully managed by immunosuppressant-incorporated medical therapy [1]. Several concerns have been raised.

In case 1, the authors performed an endomyocardial biopsy [1]. They probably performed concomitant right heart catheterization to measure hemodynamics. Such data should more clarify the mechanism of MIS-A. Of note, pulmonary artery pulsatility index might clarify the involvement of right heart during MIS-A. Also, did the authors measure right heart function such as tricuspid annular plane excursion by transthoracic echocardiography? Given the pathophysiological mechanism of MIS-A, bilateral hearts would be involved.

Based on the concept of cardio-renal-anemia syndrome, heart failure often accompanies renal impairment and anemia [2]. Given the nature of systemic inflammation, multi-organ failure would have developed in their patients. Did the authors measure urine protein, serum creatinine, hemoglobin, and erythropoietin in their cases?

The American College of Rheumatology guidelines for MIS in children recommend anticoagulation therapy in patients with moderate to severe left ventricular dysfunction [3]. Although detailed therapeutic strategy for MIS-A remains unestablished, did the authors consider anticoagulation therapy for their patients to prevent thromboembolic complications?

Declaration of competing interest

None.

References

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Author's reply

We truly appreciate the interest in our article. The letter from Drs Ishikawa and Imamura demonstrates how much we have yet to understand about multisystemic inflammatory syndrome in adults (MIS-A) related to coronavirus disease 2019 (COVID-19). The question about performing a right heart catheterization to measure hemodynamics is interesting to understand this disease. In case 1, a right ventricular biopsy was performed on the 7th day after admission. Right ventricular systolic intracardiac pressure was 25 mm Hg and right end-diastolic pressure was 3 mm Hg. In case 1, echocardiogram showed severe left ventricular dysfunction [left ventricular ejection fraction (LVEF) 32.8 %] with mildly dilated right ventricle and preserved systolic function, with tricuspid annular plane systolic excursion (TAPSE) of 2.16 cm and baseline diameter of 4.7 cm. Case 2 presented severely depressed left ventricular systolic function (LVEF 28 %) with global hypokinesia, with normal sized right ventricle with moderately depressed function and TAPSE of 1.5 cm.

The two cases presented in our article had obvious signs of systemic inflammation. Attached are analytical data of hemoglobin, serum creatinine, and inflammatory parameters of the two cases (Tables 1 and 2). Erythropoietin was not measured. As can be seen in the data presented, it is worth highlighting the mild anemia in both cases which could be explained by inflammatory status, no significant increase in serum creatinine level was evident but clinical signs of acute heart failure were present and treatment for this condition had to be established.

Anticoagulation in these patients is a controversial topic. The American College of Rheumatology guidelines for MIS in children refer that patients with MIS and documented thrombosis or an LVEF <35 % should receive therapeutic anticoagulation with enoxaparin [1]. However, the 2021 European Society of Cardiology Guidelines for the diagnosis and treatment of acute and chronic heart failure suggest thromboprophylaxis in acute heart failure in absence of other pathology in which anticoagulation was indicated [2] and indications for anticoagulation in patients with myocarditis include standard general indications such as evidence of systemic embolism or presence of acute left ventricular thrombus [3]. For all these reasons, the two cases we have presented received anticoagulation at prophylactic doses. However, there are no studies on therapeutic anticoagulation in specific cases of MIS-A related to COVID-19, so more studies are needed.

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