# New fever and acute heart failure weeks after COVID-19 – red flags for multisystem inflammatory syndrome in adults

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**Introduction**: A life-threatening hyperinflammatory condition occurring several weeks after primary infection with SARS-CoV-2 that can include severe acute heart failure has been reported in children in early 2020. Later on, a condition with similar characteristics has also been reported in adults.[1,2]

**Case report**: 26-year-old male patient with obesity and arterial hypertension presented to the emergency department with 3 days history of fever, chills, dyspnea, exercise intolerance, headache, dry cough, nasal discharge and diarrhea approximately six weeks after he has been diagnosed with mild COVID-19. Initial blood tests showed markedly elevated laboratory inflammatory markers. CT scan showed enlarged lymph nodes in the neck and small areas of residual ground-glass opacities in the lungs. Pulmonary thromboembolism was ruled out. He was admitted to the infectious disease clinic and was started on cloxacillin, ceftriaxone, antipyretics and appropriate rehydration. On the third day of hospital treatment, the patient complained of chest and neck pain and severe dyspnea. His general condition deteriorated rapidly, and he was transferred to the intensive care unit due to the development of cardiogenic shock. Inotropic and vasopressor support was initiated. Echocardiography showed mildly dilated left ventricle with severe impairment of global systolic function (LVEF 20 %). High-sensitivity troponin I and NT-proBNP were markedly elevated with a peak concentration of 792 and 18200 ng/l, respectively. Therefore, the patient was transferred to the tertiary center coronary care unit (CCU). Pulse steroid therapy and intravenous immunoglobulin were immediately initiated, while heart failure therapy was gradually introduced in the following days. Seven days after admission to CCU, multiparametric cardiac magnetic resonance imaging (MRI) revealed preserved LVEF with diffusely prolonged myocardial T1 relaxation time and T2 relaxation time, confirming myocardial injury and edema, respectively. There was no late gadolinium enhancement (**Figure 1**). Updated Lake-Louise criteria for myocarditis have been fulfilled.[3] The following criteria for multisystem inflammatory syndrome in adults (MIS-A) were met: age over 21 years, a positive test result for previous SARS-CoV-2 infection, documented fever >38.0°C for ≥24 hours, laboratory evidence of inflammation, involvement of cardiovascular and gastrointestinal system, and severe illness requiring hospitalization.

**Conclusion**: This case indicates that there is a vast diversity of clinical presentation and underlying mechanisms of COVID-19 and post-COVID-19 syndromes with myocardial injury. Recently described MIS-A is a rare but potentially life-threatening complication of previous SARS-CoV-2 infection. Clinicians should be aware of this syndrome in order to recognize it on time and start with appropriate treatment.

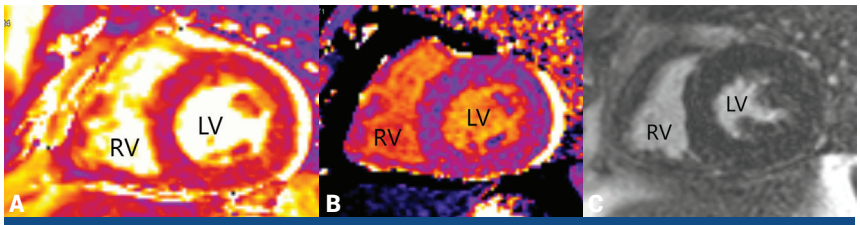


**FIGURE 1. Cardiac magnetic resonance imaging (MRI) findings; Multiparametric cardiac MRI revealed preserved left ventricular ejection fraction with diffusely prolonged myocardial T1 relaxation time (1058 ms, normal values 954-1042 ms, A) and T2 relaxation time (49,0 ms, normal values <47,5 ms, B), confirming myocardial injury and edema, respectively. There was no late gadolinium enhancement (C). There was left ventricular concentric symmetric myocardial hypertrophy with myocardial thickness of 15 mm.**

LV- left ventricle, RV- right ventricle.

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## LITERATURE

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