# Cardiac arrest in COVID-19 myocarditis: a case report

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**Introduction**: Coronavirus disease 2019 (COVID-19) was first described in China, in patients with flu-like symptoms in December 2019[1]. This family of viruses is known for its cardiotropism[2]. Arrhythmia is possible clinical manifestation in COVID-19 patients and several cases of COVID-19 myocarditis have been reported, some as a cause of death[3].

**Case report**: We present a case of a 37-years old, previously healthy, female patient who was admitted to COVID-19 Intensive care unit (ICU) at University Hospital Centre Zagreb after out of hospital cardiac arrest and successful resuscitation. She manifested episodes of chest pain and palpitations during two months prior to cardiac arrest. Initial laboratory findings showed elevated levels of high-sensitive troponin I and NT-proBNP, significant hypokalemia and normal values of C-reactive protein. Additional urgent work-up (pulmonary CT angiography and brain CT scan) showed no significant pathology and Sars-Cov-2 PCR RNA test came positive, without respiratory involvement. Due to ECG changes and ultrasound finding of reduced left ventricular ejection fraction (LVEF 25-30%) with anteroseptal and apical akinesia and inferior hypokinesia, urgent coronary angiography was performed, there were no signs of coronary artery disease, and the suspected diagnosis was Takotsubo cardiomyopathy or myocarditis. Soon after admission heart failure therapy was introduced, and follow-up echocardiography showed improvement in LVEF (40-45%). Patient was given no specific antiviral treatment nor corticosteroid therapy. Additional work-up regarding serology for cardiotropic viruses came negative, and IgG antibodies for Covid-19 showed borderline result. Cardiac magnetic resonance imaging (MRI) performed 18 days after initial event described recovered left ventricular ejection fraction (LVEF 53%), with mild hypokinesia, oedema and mid-wall late gadolinium enhancement in apical 2/3 of anterior, anteroseptal and anterolateral wall, with pattern characteristic for myocarditis (**Figure 1**)[4]. At follow-up, one month after discharge, patient is completely recovered, without signs of heart failure or arrhythmias, with preserved LVEF and normal NT-proNBP levels.

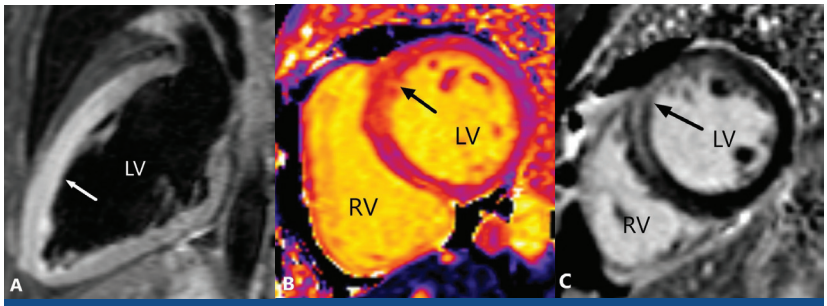


**FIGURE 1. Cardiac magnetic resonance imaging (MRI) findings consistent with typical characteristics of myocarditis; A) MRI (2-chamber view) shows a high intensity area of the left ventricular wall on T2-weighted short-tau inversion recovery STIR, consistent with myocardial oedema in apical 2/3 of the anterior, anteroseptal and anterolateral wall (arrow); B) MRI native T1 mapping (SAX; short axis view) shows significantly increased native myocardial T1 values - mean value 1267 ms in comparison with normal myocardium mean value of 1025 ms in the same area (arrow), where native myocardial T2 values were also increased - mean value 70 ms in comparison with normal myocardium mean value of 53 ms (image not shown); C) MRI (SAX view) shows post-contrast enhancement on LGE of mid-wall distribution in the same area (arrow).**

LV- left ventricle, RV- right ventricle.

**Conclusion**: This case once again highlights cardiac complications of SARS-CoV-2 infection, without respiratory involvement. Also, it shows good prognosis without specific antiviral treatment, emphasizing importance of early introduction of heart failure therapy.

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## LITERATURE

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