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Acute myopericarditis in a patient with mild SARS-CoV2 respiratory infection

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Summary title:

Acute myopericarditis in COVID-19.

Key words: SARS-CoV-2 • COVID-19 • Acute myopericarditis • Myocardial injury

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Abstract:

Herein is presented a case of a 71 year-old woman with mild SARS-CoV2 respiratory infection who experienced acute myopericarditis diagnosed by clinical, biological, ECG data and cardiac magnetic resonance imaging. The presented case highlights the risk of cardiac involvement, even in the absence of severe respiratory COVID-19 infection. The mechanisms involved in acute myocardial injury in SARS-CoV-2 infection are not well known and requires further studies to precise whether it is related to direct myocardial damage by the virus or to a systemic condition.

Brief Summary :

A 71 year-old woman with mild SARS-CoV2 respiratory infection experienced an acute myopericarditis diagnosed by clinical and cardiac magnetic resonance imaging data. As particular features she had LV regional wall motion abnormalities and persistence of myocardial edema more than 3 weeks after the onset of the symptoms.

A 71-year-old woman with a previous history of breast cancer treated with surgery, chemotherapy, radiotherapy and hormonotherapy and no previous cardiovascular events or echocardiography abnormalities at oncological follow-up presented to the emergency department with a 2-week history of flu-like symptoms, mild fever (38°C), chest pain and mild decrease of blood oxygen saturation (SpO₂ 91%). She was hemodynamically stable, and had no clinical signs of heart failure. Electrocardiogram (ECG) revealed diffuse inverted T waves and elongated QT up to 700 ms (*panel A*). She had mild persistent elevations in high sensitivity troponin T (hs-Tn T), BNP, C-reactive protein, and fibrinogen levels (60 ng/l, 474 ng/l, 9 mg/l, 5.5 g/l respectively). Pulmonary computed tomography (CT) displayed mild bilateral peripheral lower pulmonary lobe ground-glass opacities (*panel B*). A nasopharyngeal

swab was performed, and was positive for SARS-CoV-2 on real time reverse transcriptase-polymerase chain reaction assay. She had neither concomitant infections nor auto-immune disease. The patient was admitted to a specialized COVID-19 unit in our institution. Echocardiography showed infero-septal and infero-apical left ventricle (LV) wall hypokinesia, left ventricle ejection fraction (LVEF) 56% and a moderate pericardial effusion. There was no evidence of coronary disease on coronary angiography. Cardiac magnetic resonance imaging (CMR) at day 9, showed recovery of LV wall motion, normal LVEF (61%) and persistence of a mild pericardial effusion (*Panels C,D*). Short inversion time inversion recovery (STIR) sequences and T2 map, showed elevated myocardial T2 value up to 60 ms (N=50 ms) suggestive of myocardial edema in the basal inferior LV wall (*Panel C*). Furthermore, myocardial T1 mapping showed focal increased native myocardial T1 up to 1350 ms (N=1200 ms, CMR 3T) and increased extracellular volume (ECV =0.39%). Cardiac contrast-enhanced MRI showed multiple areas of inferior sub-epicardial and mid-wall late gadolinium enhancement (LGE) based on visual analysis. Although LGE volume quantification was not done, LGE intensity was more than 4 standard deviations higher than reference myocardium (*Panels E,F*). Based on clinical, biological, ECG and CMR Lake Louise Criteria and T1, T2 mapping the diagnosis was consistent with acute myopericarditis. The initial follow-up during hospitalization showed resolution of chest pain but persistence of ECG abnormalities without any specific treatment. Echocardiography follow-up on day 21, showed disappearance of pericardial effusion, no pericardial thickening and normal LV wall motion.

Discussion

Myocarditis can be classified on the basis of etiology, clinico-pathological and clinical criteria. Acute myocarditis is a type of myocardial injury that could be of infectious, autoimmune, or toxic origins (1). Acute myocardial injury diagnosis based on elevation of

troponin and ECG abnormalities has been reported in patients with COVID-19 (2). An increased level of hs-Tn in 7.2% of overall COVID-19 patients and 22% in those required ICU care has been reported (3). There are few reports of acute myocarditis in COVID-19 patients undergoing CMR (3,4), and only one showed myocarditis without symptoms and signs of interstitial pneumonia (4). Herein, we report acute myopericarditis diagnosed using CMR in a mild respiratory COVID-19 infection. Consistent with the literature (3), the present case showed LV regional wall motion abnormalities, which is not a common feature of other forms of non COVID-19 myocarditis. Another particular feature in this case is that the degree of myocardial edema was quite intense as it was still present on CMR obtained more than 3 weeks after symptom onset. Significant interstitial edema within the myocardium has been reported in a case of acute myocarditis in a COVID-19 patient undergoing endomyocardial biopsy (3). Viral induced myocarditis results from the interaction of the virus with the host's immune system. The viral non-COVID-19 myocarditis most commonly is initiated by the introduction of the virus or reactivation of a dormant virus. The viral proliferation in cardiomyocytes can cause direct injury, however the most important myocardial injury results from the activation of the innate and acquired immunity after viral invasion (1). The pathogenesis of acute myocardial injury in SARS-CoV-2 infection is still poorly known. One potential mechanism is direct myocardial involvement mediated via host protein angiotensin converting enzyme 2 (ACE2), as it is now known that the SARS-CoV-2 virus uses ACE2 as a co-receptor for entry into the lung and brain cells (5). It is unclear if cardiac involvement in SARS-CoV-2 viral infection could be related to direct damage of cardiomyocytes via this process. In non-COVID-19 viral myocarditis the earliest response to the viral infection is the activation of the innate immune system, which induces inflammatory responses via toll-like receptors (TLRs) present on the all cells and particularly in the cardiovascular system. The activation of innate system induces the expansion of T and B lymphocytes (1). The COVID-19 associated

endomyocardial biopsy showed diffuse CD3+ T lymphocytic inflammatory infiltrates with huge interstitial edema without the presence of SARS-Cov-2 within the myocardium (3).

Severe hypoxia in respiratory COVID-19 infection induces oxidative stress, and myocardial injury can occur as a consequence of increased myocardial oxygen demand. This mechanism of myocardial injury is unlike in the present case as there were neither hemodynamic modifications nor severe hypoxia.

The presented case highlights the risk of cardiac involvement, even in the absence of severe respiratory COVID-19 infection. The mechanisms involved in acute myocardial injury in SARS-CoV-2 are still not well known and requires further studies.

Teaching points:

- The diagnosis of acute myopericarditis in COVID-19 can be challenging.
- Cardiac involvement even in the absence of severe respiratory COVID-19 infection.
- The mechanisms involved in myocardial injury in SARS-CoV-2 are still not well known.

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Figure legends:

ECG, pulmonary CT, and cardiac MRI data in a 71-year-old woman, presenting with mild SARS-CoV2 respiratory infection and acute myopericarditis. **(A)** ECG: diffuse inverted T waves and elongated QT. **(B)** Pulmonary CT showing mild peripheral right lower pulmonary lobe ground-glass opacities (black arrow). **(C)** Cardiac MRI short axis T2-map showing inferior myocardial edema (white arrow). **(D)** Vertical long axis (Steady-state free precession (SSFP) MRI sequence) showing pericardial effusion (asterix). Vertical long axis **(E)** and short-axis **(F)** Phase-Sensitive Inversion-Recovery (PSIR) sequence shows multiple areas of subepicardial and mid-wall late gadolinium enhancement (LGE) in the inferior-basal LV wall (white arrows) and in the inferior-apical wall (arrowhead).

