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Terra incognita: clinically suspected myocarditis in a SARS-CoV-2 positive patient

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A clinically suspected myocarditis in a SARS-CoV-2 positive patient.

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The emerging outbreak of coronavirus disease 2019 infection (COVID-19) was declared a pandemic by the World Health Organization on March 11th, 2020 [1]. As yet, there is scarce data concerning cardiovascular involvement due to COVID-19 [2]. Even though respiratory tract signs mostly characterize the clinical course of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection, cardiac complications, such as myocardial inflammation, may also occur alone [3]. Our case presents a patient whose clinical presentation raised our suspicion for underlying myocarditis.

A 74-year-old male with a recent history of normal left ventricular ejection fraction (LVEF) of 55% was admitted to hospital due to hemodynamically unstable new-onset ventricular tachycardia (VT) of 12 hours duration (Figure S1A, Figure S1B). After unsuccessful self-administration of metoprolol 200 mg orally, arrhythmia had been electrically cardioverted with subsequent symptomatic bradycardia and hypotension. The patient did not present with a fever or respiratory infection symptoms.

The patient had a history of atrial fibrillation, three-time catheter ablation, arterial hypertension, type 2 diabetes, hypothyroidism. As a medical professional, the patient might have been exposed to SARS-CoV-2.

Due to the indicative clinical picture of severely reduced LVEF 25% suggestive of myocardial injury as well as recurrent VT, the patient underwent coronary angiography, which revealed no obstructive coronary artery disease. Due to the third-degree atrioventricular block, a temporary transvenous pacemaker had been inserted.

The following day next episodes of VT occurred. Serum electrolytes and thyroid hormone levels were within normal limits. Overdrive pacing with a rate of 95/min., magnesium substitution, and intravenous amiodarone stabilized the rhythm. On the 3rd day of

hospitalization, the temporary pacemaker was removed, and observation did not reveal any VT recurrence.

Laboratory findings were as follows: higher than three neutrophil-lymphocyte-ratio (NLR) (normal range 1-3), elevated high-sensitivity cardiac troponin T – 72→102 ng/l (normal range: <14 ng/l), dynamically evolving C-reactive protein – 1.2→1.8→94 mg/l (normal range: < 6 mg/l), as well as elevated N-terminal prohormone for brain natriuretic peptide – 2451 ng/l (normal range: <125 ng/l), lactic acid dehydrogenase – 369 U/l (normal range: 80-240 U/l), D-dimers – 1.39 mg/l (normal range: <0.5 mg/l), aspartate aminotransferase – 159 U/l (normal range: <45 U/l) and alanine aminotransferase – 163 U/l (normal range: <35 U/l) while the procalcitonin (normal range: <0.05 ng/ml) was negative. Due to suggestive, especially during the outbreak of COVID-19, laboratory results [2,4] reverse transcription-polymerase chain reaction for SARS-CoV-2 was performed on the 6th day of hospitalization and occurred positive. Consequently, the patient received a single 500 mg dose of azithromycin and a single 75 mg dose of oseltamivir. On the 7th day, he was transferred in good general condition to a hospital for infectious diseases for further treatment. On the 17th day, the patient was still tested positive in a throat swab specimen by RT-PCR and presented no respiratory symptoms.

Cardiac magnetic resonance performed on a 1.5 Tesla magnetic resonance scanner revealed left atrium enlargement and global LV hypokinesia with ejection fraction as much as 20%. The T2-weight sequence did not show myocardial edema (Figure 1, Panels A-C). In contrast, late gadolinium enhancement (LGE) showed a large patchy and linear non-ischemic pattern of fibrosis localized subepicardially and intramurally in basal and mid-cavity segments of the inferior and inferolateral wall and apical segments of the inferior wall (Figure 1, Panels D-G).

As shown in the above case, SARS-CoV-2 may cause, apart from COVID-19-related pneumonia, acute myocardial injury meeting diagnostic criteria for clinically suspected myocarditis [5].

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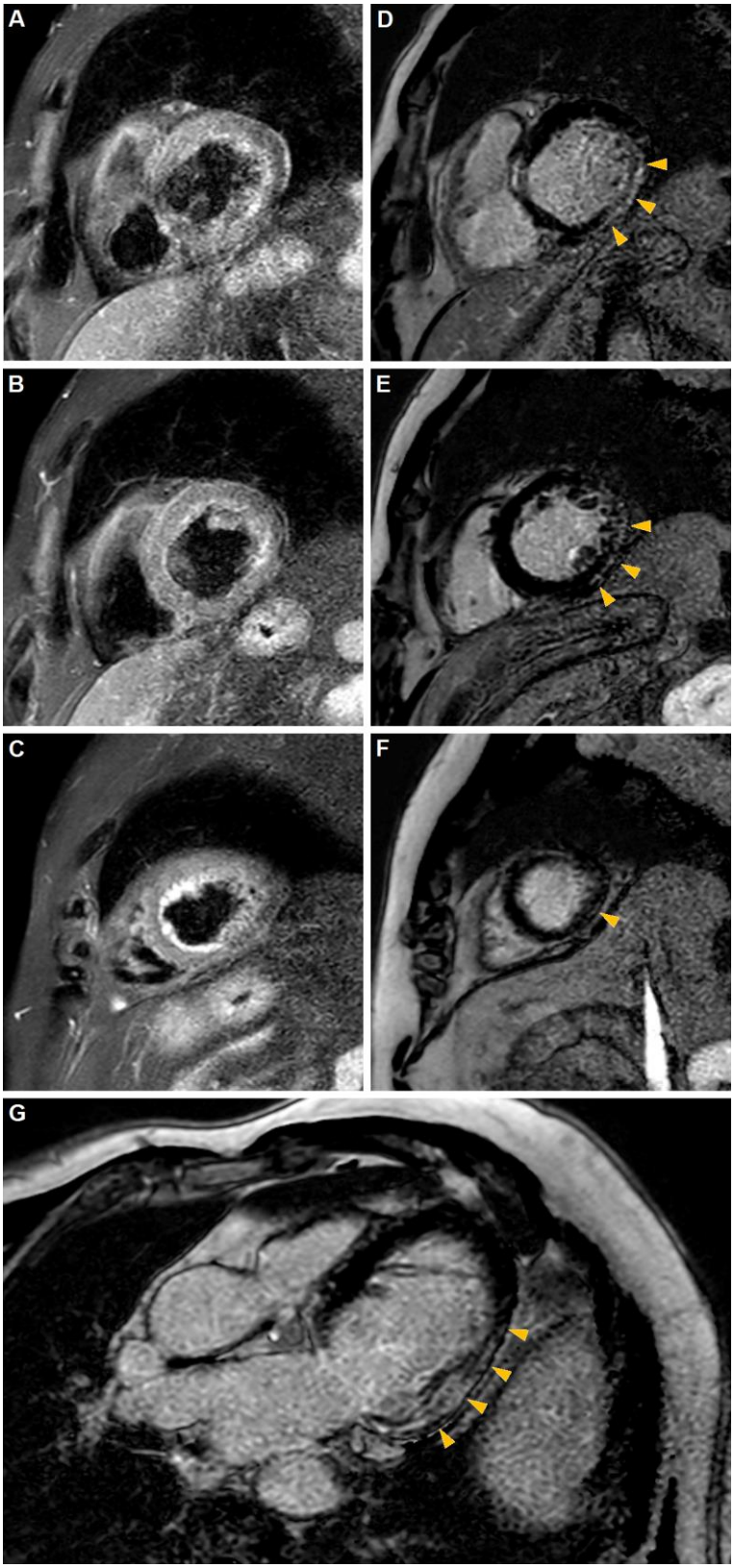

Figure 1. A-C. The T2-weight sequence in a short-axis view. D-E. Late gadolinium enhancement in a short-axis view. G. Late gadolinium enhancement in four-chamber