



Case Report

Myocarditis After SARS-CoV-2 Vaccination: A Vaccine-Induced Reaction?

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ABSTRACT

Vaccination plays an important role in the fight against SARS-CoV-2 to minimize the spread of coronavirus disease 2019 (COVID-19) and its life-threatening complications. Myocarditis has been reported as a possible and rare adverse consequence of different vaccines, and its clinical presentation can range from influenza-like symptoms to acute heart failure. We report a case of a 30-year-old man who presented progressive dyspnea and constrictive retrosternal pain after receiving SARS-CoV-2 vaccine. Cardiac magnetic resonance and laboratory data revealed typical findings of acute myopericarditis.

RÉSUMÉ

La vaccination joue un rôle important dans la lutte contre le SARS-CoV-2 afin de minimiser la propagation de la maladie à coronavirus 2019 (COVID-19) et ses complications potentiellement mortelles. La myocardite a été signalée comme une conséquence potentielle, indésirable et rare, de différents vaccins, et sa présentation clinique peut aller de symptômes de type grippal à une insuffisance cardiaque aiguë. Nous rapportons le cas d'un homme de 30 ans qui a présenté une dyspnée progressive et une douleur rétrosternale constrictive après avoir reçu le vaccin contre le SARS-CoV-2. La résonance magnétique cardiaque et les données de laboratoire ont révélé des résultats typiques d'une myopéricardite aiguë.

A 30-year-old man presented at the emergency department complaining dyspnea, constrictive retrosternal pain, nausea, and profuse sweating. Of note, the patient had suffered from fever (38.8°C) and arthralgia 72 hours earlier when he received his second dose of SARS-CoV-2 vaccine (mRNA BNT162b2), which was injected 21 days after the first dose.

The patient tested negative at nasopharyngeal swab testing for SARS-CoV-2, as required before hospital admission.

Anamnesis was negative for cardiovascular or metabolic disorders and recent infectious diseases.

At physical examination he was afebrile, with moderate tachycardia (heart rate 93 beats/min) and normal blood pressure (115/58 mm Hg). At auscultation, neither lung alterations nor heart murmurs were identified; oxygen saturation was of 99% on room air.

Laboratory data revealed elevated cardiac troponin I (12,564.80 pg/mL; normal < 34.2 pg/mL), creatine kinase-MB (53.8 ng/mL; normal 0-5.2), lactate dehydrogenase (228

U/L; normal 125-220), activated partial thromboplastin time (75.2 seconds; normal 20-40), and C-reactive protein (39.6 mg/L; normal 0-5). White blood cells were $10.4 \times 10^3/\mu\text{L}$ (normal 4.0-10.0), with mild eosinophilia ($0.9 \times 10^3/\mu\text{L}$, normal $0.0-0.5 \times 10^3$). Serum levels of cardiac troponin I, creatine kinase-MB, and C-reactive protein during the first 72 hours from hospital admission are shown in Figure 1.

Electrocardiography (ECG) showed subtle ST-segment elevation suggestive of potential myocardial injury or pericarditis in V2-V4 and nonspecific T-wave changes in V5 and V6. Transthoracic echocardiography revealed preserved ejection fraction, mild pericardial effusion, and segmental wall motion abnormality of the apical portion of interventricular septum. No coronary artery disease was found at coronary angiography. Cardiac magnetic resonance imaging (MRI), performed 72 hours after hospital admission, revealed good systolic function and increased myocardial and pericardial signal intensity on T2-weighted short tau inversion recovery sequences (T2 ratio 2.1; normal < 2). T1-weighted phase-sensitive inversion recovery sequences, performed 15 minutes after intravenous injection of gadolinium, showed subepicardial enhancement of the myocardium, suggesting a provisional diagnosis of myopericarditis (Fig. 2).

Extensive infectious and rheumatologic workup was unremarkable. Virus serology did not show IgM antibodies nor 4-

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See page 1667 for disclosure information.