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Myocarditis after BNT162b2 vaccination in a healthy male

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ABSTRACT

Myocarditis following mRNA COVID-19 vaccination has recently been reported to health authorities in the United States and other countries. Cases predominately occur in young adult males within four days following the second dose of either the Moderna (mRNA-1273) or Pfizer-BioNTech (BNT162b2) vaccines. Although the number of cases reported have been small in comparison with the large number of people vaccinated, myocarditis may be a rare adverse reaction to the COVID-19 vaccination that is now only becoming apparent due to the widespread use of the vaccine. In this article, we present a case of a 20-year-old male with no prior medical history who presented to the emergency department (ED) with chest pain. He had received the BNT162b2 vaccine two days prior to his presentation to the ED. The patient had an elevated troponin at 89 ng/L which increased on repeat examination. His electrocardiogram showed diffuse concave ST segment elevations and a later MRI confirmed the diagnosis of myocarditis. Based on these findings, the patient was diagnosed with myocarditis. The patient had a previous infection with SARS-CoV-2 approximately two months prior to the onset of his symptoms, but since he had fully recovered before the time of his presentation to the ED, it is unlikely that the infection caused the myocarditis. To our knowledge, this is the first published case of myocarditis following BNT162b3 vaccination.

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A 20-year-old male with no prior medical history presented to the emergency department (ED) with a chief complaint of midsternal chest pain that radiated to the left side. The pain started in the morning and remained while resting. The patient also complained of mild shortness of breath secondary to pain. Upon examination, pain worsened with sitting and improved while lying flat. The patient had received his second dose of the Pfizer-BioNTech (BNT 162b2) vaccination two days prior to the onset of chest pain. The patient denied history of venous thromboembolism or family history of cardiovascular disease. The patient had a history of tobacco use. Approximately two months prior to ED presentation, the patient tested positive for SARS-CoV-2 and recovered with no sequelae.

In the ED his initial troponin was 89 ng/L and increased to a maximum of 108 ng/L. The patient tested negative for SARS CoV-2 by PCR. Vital signs revealed blood pressure 121/54 mm/Hg, heart rate 113 beats per minute, temperature 98.4 °F orally, respirations 20 breaths per minute, SpO₂ 100% on room air. The patient's electrocardiogram showed diffuse concave ST segment elevations with PR depressions. (Fig. 1). Myocarditis was suspected and bedside ultrasound revealed a small pericardial effusion without evidence of tamponade, which supported the diagnosis. The patient was subsequently given a dose of colchicine 0.6 mg and then admitted to the hospital for further evaluation.

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Inpatient echocardiogram showed left ventricular ejection fraction (LVEF) 59% with no other abnormalities. The patient underwent left heart catheterization which was unremarkable. Cardiac computed tomography showed a coronary artery calcium score of zero and no pathology. Cardiac magnetic resonance imaging was positive for myocarditis. After his chest pain resolved, the patient was discharged with colchicine 0.6 mg for three months, metoprolol XL 12.5 mg daily for three months, and ibuprofen 600 mg three times daily for two weeks.

Myocarditis is most often caused by a viral infection; however, other causes include idiopathic, autoimmune, and hypersensitivity [1]. When there is an infectious etiology, patients typically present with flu-like symptoms in addition to chest pain [1]. Myocarditis is a potential complication of infection with COVID-19. Cardiac complications were first documented in Wuhan, China where 22–31% of COVID-19 patients admitted to the ICU developed myocarditis and 7% of COVID-19 deaths were secondary to myocarditis [2,3]. Pathologic mechanisms proposed for development of myocarditis following infection with COVID-19 include direct invasion of the myocardium, cytokine storm, or other hyperimmune activation that promotes inflammation [2].

Myocarditis has also been reported following live viral vaccinations, most notably the smallpox vaccine. From the early 1950s until 2003, six cases of cardiac complications following smallpox vaccination were reported in the United States [4]. A campaign to vaccinate U.S. military personnel against smallpox with the DryVax vaccine between 2002 and 2003 resulted in 67 cases of myocarditis or pericarditis out of the 540,824 personnel vaccinated [5]. Due to concern for cardiac