

## CASE REPORT

# Myocarditis following AstraZeneca (an adenovirus vector vaccine) COVID-19 vaccination: A case report

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## Abstract

Coronavirus disease-19 (COVID-19) vaccines are massively administered globally and some adverse events, such as myocarditis, are being reported. Most of the reported cases of post-vaccination myocarditis have occurred following mRNA vaccinations. However, there have also been recent reports of myocarditis following adenovirus vector vaccinations. We present a case of a 32-year-old female patient who developed myocarditis following the administration of the first dose of the AstraZeneca vaccine. The patient developed inappropriate exertional tachycardia and exertional dyspnea from Day 3 and was diagnosed with myocarditis by subsequent echocardiography about 3 months later. We are unable to confirm a direct association between myocarditis and AstraZeneca vaccination. However, we would like to increase awareness regarding the possibility of developing myocarditis following AstraZeneca vaccination.

## KEYWORDS

AstraZeneca-associated myocarditis, COVID-19 vaccination-associated myocarditis, mRNA COVID-19 vaccines, myocarditis, viral vector vaccines

## 1 | INTRODUCTION

The severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2), also known as coronavirus disease (COVID-19), mostly affects the lungs but may also involve other organs such as the heart. There have been reports of cardiovascular complications following COVID-19 such as myocarditis, venous thromboembolism, acute myocardial infarction, heart failure, and arrhythmias.<sup>1-4</sup> Various types of COVID-19 vaccines have been developed. The two main COVID-19 vaccines are either mRNA-based or adenovirus vector vaccines. Following the widespread administration of COVID-19 vaccines around the world,

some adverse events such as myocarditis are being reported. Most of the reported cases of post-vaccination myocarditis have been reported following mRNA vaccines (Pfizer/BioNTech and Moderna). However, there have been recent reports of myocarditis after adenovirus vector vaccines (AstraZeneca and Janssen).<sup>5-9</sup>

Most individuals that develop myopericarditis following COVID-19 vaccination present with various signs and symptoms such as chest pain, tachycardia, palpitation, hypotension, edema, clinical features of decreased cardiac output, shortness of breath, fatigue, fever, nausea, and vomiting. The workup of these patients usually includes testing for inflammatory and cardiac biomarkers,

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serologic SARS-CoV-2 antibodies level, electrocardiogram (ECG), echocardiogram, and cardiac magnetic resonance imaging (CMR). Most cases of myopericarditis following COVID-19 vaccinations show clinical recovery with or without treatment, and it seems to be a temporal and self-limited condition.<sup>6,10,11</sup>

In this study, we present the second reported case of myocarditis following AstraZeneca vaccination in a 32-year-old female patient following the first dose. The patient complained of inappropriate exertional tachycardia and dyspnea, fatigue, and palpitation for about 3 months but did not have any other symptoms such as chest pain or elevated troponin levels. The results of her initial work-ups were normal. However, subsequent echocardiography about 3 months later demonstrated myocarditis with subnormal left ventricular (LV) function.

## 2 | CASE PRESENTATION

A previously healthy 32-year-old Persian female presented to our cardiology clinic with an approximately 3-month history of inappropriate exertional tachycardia and dyspnea following the first dose of AstraZeneca vaccination. She had not experienced any other symptoms such as chest pain, cough, or viral infection symptoms. Her past medical history was unremarkable except for an episode of vasovagal syncope about 10 months earlier. The patient denied using any medication or having any exposure to COVID-19 patients or prior SARS-CoV-2 infection. In addition, there was no history of allergy or any adverse reactions to prior vaccinations.

The patient experienced mild fever, myalgia, and chills on days one and two after vaccination which resolved with ibuprofen use. On day three, she developed dyspnea at rest, exertional dyspnea, and inappropriate exertional tachycardia. Her respiratory rate (RR) at rest was around 19–20 but she had not experienced tachycardia at rest. Following a consultation with an Internist, laboratory tests were obtained to rule out pulmonary embolism. The tests showed a D-dimer level of 266 ng/ml, fibrinogen level of 430 mg/L, and normal platelet levels. A diagnosis of airway hyperresponsiveness was established and symptomatic treatment with montelukast and cetirizine was initiated. However, her symptoms did not improve and another laboratory evaluation on Day 10 demonstrated a D-dimer level of 800 ng/ml, normal platelets levels, fibrinogen level of 330 mg/dl, positive (+) C-reactive protein (CRP), and erythrocyte sedimentation rate (ESR) level of 24 (the first hour). In addition, the polymerase chain reaction test for COVID-19 was negative, and the chest X-ray (CXR) and ECG results were normal. The patient then visited a pulmonologist and was prescribed budesonide/

formoterol inhaler and prednisone (50 mg/daily), chlorphenamine, and rivaroxaban (10 mg/daily) for 10 days. Dyspnea at rest improved slightly but exertional dyspnea and inappropriate exertional tachycardia worsened and her pulse rate (PR) would increase up to 150–160 during regular activity. Therefore, on Day 22, the patient was admitted to the hospital for further evaluation.

Upon admission, PR was around 130–140 as the patient had walked a couple of minutes to reach the emergency department and RR at rest was 19. The first ECG showed sinus tachycardia, but subsequent ECGs were normal. Laboratory tests showed a D-dimer of 1800, normal troponin levels (12 and 10 (6-h later) ng/L), a hemoglobin level of 13.2 g/dl, and normal thyroid tests. CXR, Doppler sonography of lower extremities, and chest CT-angiography were normal. Echocardiography was normal (LV ejection fraction (LVEF) by Simpson's mode was 65%). Spirometry test showed FEV1 of 102% of the predicted value and FEV1/FVC of 0.83 of the predicted value (Table 1). During hospital admission, the patient only received enoxaparin (prophylactic dose) and was discharged with metoprolol and fluticasone nasal spray.

After discharge from the hospital and upon a visit to another pulmonologist for a second opinion, she was diagnosed with airway hyperresponsiveness and was prescribed tiotropium inhalation spray and rivaroxaban 10 mg/daily (due to the increase in her D-dimer level) which was extended for another month. The patient was also referred to a psychiatrist to rule out anxiety as exertional dyspnea, especially while talking, was worsened, and therefore, was prescribed sertraline and chlordiazepoxide.

On a follow-up visit after a month for a consultation to whether receive the second dose of vaccination, RR at rest had decreased to around 15–16 but exertional dyspnea and inappropriate exertional tachycardia had remained. Therefore, due to these persistent symptoms, the patient was referred to a cardiologist for a reevaluation with echocardiography to rule out an underlying cardiac cause such as pulmonary hypertension (PH). Echocardiography demonstrated no PH (SPAP = 20 mmHg). In addition, the size of the LV was normal with preserved systolic function (LVEF by Simpson's mode was 50%). The impression was that based on subnormal LV function and mildly increased LV wall thickness, recent myocarditis may have been the cause of symptoms. Consequently, carvedilol 6.25 mg/BD and rivaroxaban 10 mg/daily were prescribed. Following the patient's visit to our clinic for a second opinion, carvedilol and rivaroxaban were discontinued, but captopril 12.5 mg/BD and bisoprolol 2.5 mg/daily were prescribed for 3 months. Due to the prolonged time between the development of the symptoms and diagnosis, CMR was not recommended. The patient was also recommended to follow-up after 3 months of optimal medical therapy with

**TABLE 1** Results of the patient's imaging, electrocardiograms, echocardiography, and spirometry tests during admission to the hospital

Test	Result
Imaging	CXR: <ul style="list-style-type: none"> <li>• Normal</li> <li>• No acute lung abnormality</li> </ul> Multi-slice CT-angiography of pulmonary arteries with contrast (PTE Protocol): <ul style="list-style-type: none"> <li>• No pulmonary embolism</li> <li>• No COVID-19 lung involvement</li> </ul> Doppler Sonography of bilateral lower extremities: <ul style="list-style-type: none"> <li>• No DVT</li> </ul>
ECG	<ul style="list-style-type: none"> <li>• Sinus tachycardia (the first ECG)</li> <li>• Subsequent ECGs = normal</li> </ul>
Echocardiography	<ul style="list-style-type: none"> <li>• LVEF by Simpson's mode of 65%</li> <li>• Normal LV</li> <li>• Hyperechoic and collapsed IVC</li> <li>• Normal RA, RV</li> <li>• Normal rhythm</li> <li>• Mild TR</li> <li>• Normal SPAP</li> <li>• Collapsed IVC</li> <li>• No clot</li> <li>• No pericardial effusion</li> <li>• No LVH</li> <li>• No valvar heart disease</li> <li>• No pulmonary hypertension</li> </ul>
Spirometry	<ul style="list-style-type: none"> <li>• FEV1 = 103% of the predicted value</li> <li>• FEV1/FVC = 0.83 of the predicted value</li> </ul>

Abbreviations: CT, computed tomography; CXR, chest x-ray; DVT, deep vein thrombosis; ECG, electrocardiogram; IVC, inferior vena cava; LA, left atrium; LVEF, left ventricular ejection fraction; LVH, left ventricle hypertrophy; MR, mitral regurgitation; MVP, mitral valve prolapse; PAP, pulmonary artery pressure; PTE, pulmonary thromboembolism; RA, right atrium; RV, right ventricle; TR, tricuspid regurgitation.

another echocardiography and was discouraged from receiving the second dose of AstraZeneca vaccination. The details of the patient's workup and laboratory results are shown in [Tables 1 and 2](#).

### 3 | DISCUSSION

Myocarditis is the inflammation of the cardiac muscle that is caused by an underlying event resulting in an inflammatory cellular infiltration that leads to myocardium injury. Viral infection is the most common etiology of myocarditis. However, any other factors such as bacterial infections and drug-induced hypersensitivity reactions are also involved.<sup>12,13</sup> Some of the viruses associated with myocarditis include coxsackievirus B, adenovirus, parvovirus B19, Epstein-Barr virus, and coronaviruses that cause SARS (severe acute respiratory syndrome), MERS (Middle East respiratory syndrome), and COVID-19. The possible underlying mechanisms of COVID-19-associated myocarditis include direct invasion of myocardial cells by the SARS-CoV-2 virus or COVID-19-induced cytokine

storm.<sup>2,12,13</sup> There have been reports of myocarditis following some vaccinations such as eosinophilic myocarditis following conjugate meningococcal C and hepatitis B vaccines as well as myopericarditis following smallpox, influenza, diphtheria, and tetanus vaccines.<sup>14</sup>

Following the COVID-19 pandemic, various types of COVID-19 vaccines have been developed and the two main types are mRNA-based or adenovirus vector vaccines. Both mRNA and adenovirus vector vaccines express the SARS-CoV-2 spike protein without replicating. The SARS-CoV-2 spike protein is involved in the attachment to the host cells and viral entry and triggers an effective immune response.<sup>15</sup> The most commonly administered mRNA-based COVID-19 vaccines are Pfizer/BioNTech (BNT162b2) and Moderna (mRNA-1273) and the most commonly administered adenovirus vector vaccines are AstraZeneca (ChAdOx1 nCoV-19 or AZD1222) and Janssen (Ad26.COV2. S).<sup>15</sup> Following the widespread administration of COVID-19 vaccinations around the world, some adverse events such as myocarditis are being reported. Most of the reported cases of post-vaccination myocarditis are following mRNA

TABLE 2 Patient's laboratory results

Laboratory tests	Day 3	Day 10	On admission		
			1st day (Day 22)	2nd day (Day 23)	3rd day (Day 23)
WBC, $\mu\text{l}$	3420 (low)	6100	8800	11,600 (high)	9000
RBC, $10^6/\mu\text{l}$	4.70	4.54	4.93	4.26	4.49
Hb, g/dl	13	12.5	13.2	12 (low)	11.8 (low)
HCT, %	38.4	38.1	39.8%	34.1 (low)	35.9
Neutrophil, %	44 (low)	–	82.3 (high)	58.4	49.8 (low)
Lymph, %	45.6	–	14.6 (low)	33	37.9 (low)
Eosinophil, %	0.2 (low)	–	–	–	–
Platelets, $10^3/\mu\text{l}$	180	229	241	217	207
D-Dimer, ng/ml	266 (high)	800 (high)	1800 (high)	–	–
PTT (patient time), s	25	41	28	–	–
PT (patient plasma), s	12.5	13.9 (high)	9.60 (low)	–	–
INR (ratio)	1	1.1	0.98 (low)	–	–
Fibrinogen, mg/L	430 (high)	330	–	–	–
ESR (1 h)	–	24 (high)	10	–	–
CRP	–	Positive (+)	1 mg/L	–	–
PCR for COVID-19	–	Negative	–	–	–
Troponin, ng/L	–	–	• 12 • 10 (6-h later)	–	–
BS, mg/dl	–	–	131	96	86 (FBS)
ALT, U/L	–	–	16	–	–
ALP, U/L	–	–	246	–	–
AST, U/L	–	–	21	–	–
Bili (D), mg/dl	–	–	0.2	–	–
Bili (T), mg/dl	–	–	0.5	–	–
BUN, mg/dl	–	–	8 (low)	–	9
Creatinine, mg/dl	–	–	0.9	–	0.8
Ca, mg/dl	–	–	9.6	–	–
Ph, mg/dl	–	–	2 (low)	–	–
Mg, mg/dl	–	–	21	–	–
Alb, g/dl	–	–	4.6	–	4
K, mEq/L	–	–	4.6	–	138
Na, mEq/L	–	–	140	–	–
CPK, U/L	–	–	46	–	–
LDH, U/L	–	–	535	–	224
TSH, $\mu\text{g}/\text{dl}$	–	–	1.09	–	–
T3, ng/dl	–	–	120	–	–
T4, $\mu\text{g}/\text{dl}$	–	–	9.5	–	–
VBG					
V_PH	–	–	7.44 (high)	7.39	7.39
V_PCO <sub>2</sub> , mmHg	–	–	25.9 (low)	47.5 (high)	44 (high)
V_BE, mmol/L	–	–	4.5	3.1	1.7
V_BE ecf, mmol/L	–	–	5.7	3.4	1.7
V_BB, mmol/L	–	–	41.7 (low)	49.4	48.4

TABLE 2 (Continued)

Laboratory tests	Day 3	Day 10	On admission		
			1st day (Day 22)	2nd day (Day 23)	3rd day (Day 23)
V_HCO <sub>3</sub> , mmol/L	-	-	17.3 (low)	28 (high)	26.1 (high)
V_PO <sub>2</sub> , mmHg	-	-	52.8 (high)	60.4 (high)	48.8 (high)
V_PO <sub>2</sub> Sat %	-	-	88.1 (high)	90.7 (high)	84.1 (high)
V_temp C	-	-	37.0	37.0	37.0

Abbreviations: ALP, alkaline phosphatase; ALT, alanine transaminase; AST, aspartate transaminase; Billi, bilirubin; BS, blood sugar; BUN, blood urea nitrogen; CPK, creatine phosphokinase; CRP, C-reactive protein; D, direct; ESR, erythrocyte sedimentation rate; FBS, fasting blood sugar; HCT hematocrit; INR, international normalized ratio; LDH, lactic dehydrogenase; LVH, left ventricle hypertrophy; PCR, polymerase chain reaction; PT, prothrombin time; PTT, partial thromboplastin time; RBC, red blood cell; T, total; TSH, thyroid stimulating hormone; VBG, venous blood gas; WBC, white blood cell.

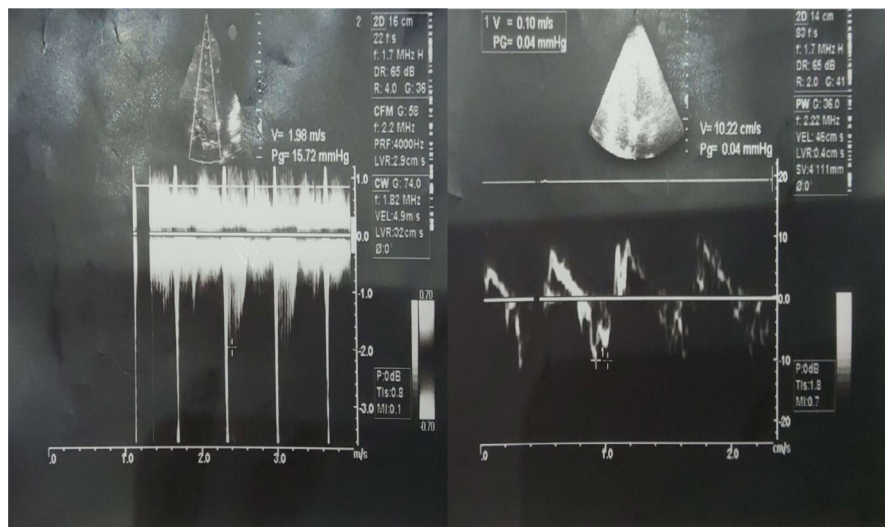
COVID-19 vaccinations and the majority of the patients are young males. However, there have been recent reports of myocarditis following adenovirus vector COVID-19 vaccines as well.<sup>5-10</sup>

Myocarditis after mRNA COVID-19 vaccination usually occurs following the second dose and mostly affects young healthy males which usually require hospitalization. The clinical features of these patients include chest pain, fever, dyspnea, cough, and headache mostly within 3 days. Laboratory results may include elevated troponin and/or CRP and mild elevations of B-type natriuretic peptide (BNP) or N-terminal pro-BNP (NT-pro-BNP) levels. Polymerase chain reaction tests of the patients were negative for COVID-19 and most patients had spike antibody levels suggestive of an effective immune response. The ECG abnormalities include ST elevations, diffuse ST changes, peaked T-wave, junctional rhythm, non-specific ST changes, and ST depression. However, some patients had normal ECG. Echocardiography has demonstrated heterogeneous results. For example, it is reported that echocardiogram abnormality was seen in 40% of the patients and LVEF <50% in a small percentage of patients. However, it has also been reported that LV function was seen in 66% and 34% of the patients who were ≥30 years of age and young adults <30 years of age, respectively. There was mild dysfunction in patients aged >18 years and LV function was usually normal in children ≤18 years. Pericardial effusion may also be found on echocardiography. CMR of the patients showed late gadolinium enhancement (LGE) and myocardial edema which are suggestive of myocarditis. Although in the early stages of post-mRNA vaccination myocarditis, localized or generalized myocardial edema without LGE or other clinical characteristics may be the only evidence of myocarditis. Almost all patients had clinical improvement and recovery with or without treatment and were discharged mostly within a week.<sup>6,10,11</sup>

There have been several suggestions for the pathogenesis and underlying mechanisms of developing

myocarditis after mRNA-based COVID-19 vaccines. For example, some young individuals may develop an increased antibody response. Cytokine expression in the myocardium which is induced by cross-reactive anti-idiotypic antibodies and abnormal apoptosis may be involved in the inflammation of the myocardium and pericardium as well. In addition, other factors such as an innate inflammatory response or a molecular mimicry (between the virus spike protein and a cardiac protein) may have possible effects. On the contrary, young individuals generally have higher seropositivity for SARS-CoV-2. Therefore, the RNA in the mRNA vaccines may have the potential to trigger the immune response through cytokines by activating the pre-existing immune cells that have auto-reactivity.<sup>6</sup>

There have been two reports of myocarditis following Janssen vaccination in two young healthy males. They had developed chest pains on Days 2 and 5, respectively, and were hemodynamically stable on admission. The first patient had elevated troponin I level and negative SARS-CoV-2 antibody. His ECG showed normal sinus rhythm with ST-elevation, but the results of his CXR and coronary angiography were unremarkable. The other patient had elevated levels of high-sensitive cardiac troponin-T (hsTropT) and CRP, and normal ECG. In addition, the echocardiography of the first patient revealed LVEF of 51%, LV end-diastolic (LVED) internal dimension of 4.8 cm, intraventricular septal diastolic thickness (2D) of 1.0 cm, mild global hypokinesis, and normal diastolic function. His CMR showed an LVEF of 50% with no wall motion abnormalities. There was mild sub-epicardial LGE in the LV walls without pericardial thickening or enhancement, or edema. This patient was treated with β-blocker, ACE inhibitor, aspirin, and clopidogrel which were discontinued on his discharge 2 days later. CMR of the second patient showed a small area of myocarditis in the LV with a 2% scar. However, the LV systolic function was normal without hypokinesis. This patient received symptomatic treatments which lead to his discharge following clinical improvement.<sup>5,8</sup>



**FIGURE 1** Patient's echocardiography showing: normal LV size with preserved systolic function (left ventricle ejection fraction (LVEF) by Simpson's mode of 50%); normal diastolic function, no LV hypertrophy, normal valves but trivial tricuspid regurgitation; no pulmonary hypertension (SPAP = 20 mmHg); normal sizes and functions of the left and right atrium and right ventricle. Based on subnormal LV function and mild increased LV wall thickness, recent myocarditis was suggested as the cause of the symptoms

Furthermore, Ujueta et al. reported a 62-year-old female patient who had developed progressive myalgia, weakness, and fatigue about 4 days after receiving the Janssen vaccine. She was hemodynamically unstable on admission and her laboratory tests showed elevated troponin, lactic acid, NT-pro-BNP, and leukocytosis. SARS-CoV-2 test was negative, and CXR was unremarkable. ECG showed sinus tachycardia with T-wave inversions and enlargement of the right atrium. Echocardiography showed severe biventricular cardiomyopathy with an LVEF of 29% and slight pericardial effusion. Cardiac angiography did not show any obstructive disease. She was immediately administered maximal doses of vasopressin, phenylephrine, and epinephrine but developed cardiac arrest and expired a few hours later. Lymphohistiocytic myocarditis with sparse eosinophils was detected on autopsy.<sup>9</sup>

Chamling et al. reported the first case of myocarditis following AstraZeneca vaccination. The patient was a 68-year-old female with a history of coronary artery disease who developed acute chest pain radiating to the left shoulder within the first day following her first dose of vaccination. Her laboratory tests were normal for inflammatory parameters but there were increased levels of cardiac enzymes including hsTropT, creatine kinase (CK), and CK-myocardial band (CK-MB). ECG, echocardiography, and coronary angiography were unremarkable. CMR reported non-ischemic myocardial damage and active inflammation suggestive of autoimmune myocarditis associated with her vaccination. The authors suggested that COVID-19 vaccines may cause an increased immune response in some individuals that may cause autoimmune myocarditis.<sup>7</sup>

In the present case, ECGs, troponin levels, and echocardiography were normal, and there was no history of chest pain. However, subsequent echocardiography

3 months later revealed myocarditis with subnormal LV function, mild increased LV wall thickness, and an LVEF by Simpson's mode of 50% (Figure 1). Therefore, the patient was prescribed a  $\beta$ -blocker and an ACE inhibitor and was discouraged from receiving the second dose of the AstraZeneca vaccine. This patient did not have prior viral symptoms, infections, or exposures to patients with COVID-19 or other viral infections or other etiologies of myocarditis before and after receiving the first dose of her vaccination. It seems that there is a temporal association between the development of myocarditis and AstraZeneca vaccination in this case. We are unable to show any evidence of a direct link between myocarditis and AstraZeneca vaccination. However, we would like to increase awareness regarding the possibility of developing myocarditis following AstraZeneca vaccination.

## 4 | CONCLUSION

In conclusion, although there have been reports of a potential association between myocarditis and COVID-19 vaccines, the exact association is still unclear and requires further investigations. With the massive administration of various types of COVID-19 vaccines, more cases of post-vaccination myocarditis will be probably reported which will help to further understand this potential association.

## CONFLICT OF INTEREST

The authors declare that they have no conflict of interest.

## AUTHOR CONTRIBUTIONS

All authors passed four criteria for authorship contribution based on recommendations of the International Committee of Medical Journal Editors. All authors had equal contributions to this study.


## ETHICAL APPROVAL

The authors state that they have followed the principles outlined in the Declaration of Helsinki for all human or animal experimental investigations. Written informed consent was obtained from the patient.

## CONSENT

Written informed consent was obtained from the patient to publish this report in accordance with the journal's patient consent policy.

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## REFERENCES

1. Azevedo RB, Botelho BG, Hollanda JVG, et al. Covid-19 and the cardiovascular system: a comprehensive review. *J Hum Hypertens*. 2021;35(1):4-11.
2. Rezkalla SH, Kloner RA. Viral myocarditis: 1917–2020: from the Influenza A to the COVID-19 pandemics. *Trends Cardiovasc Med*. 2021;31(3):163-169.
3. Daneshfar M, Dadashzadeh N, Ahmadpour M, et al. Lessons of mortality following COVID-19 epidemic in the United States especially in the geriatrics. *J Nephroarmacol*. 2021;10(1):e06.
4. Sadighpour T, Mubarak M, Sabaeifard P, Saeifar S, Kenari F. COVID-19 and renal involvement; evolving role of thromboinflammation, vascular and glomerular disease in the pathogenesis. *J Nephropathol*. 2021;10(3):e23.
5. Sulemankhil I, Abdelrahman M, Negi SI. Temporal association between the COVID-19 Ad26.COVS vaccine and acute myocarditis: a case report and literature review. *Cardiovasc Revasc Med*. 2021;S1553-8389(21)00578-9.
6. Das BB, Moskowitz WB, Taylor MB, Palmer A. Myocarditis and pericarditis following mRNA COVID-19 vaccination: what do we know so far? *Children (Basel)*. 2021;8(7):607.
7. Chamling B, Vehof V, Drakos S, et al. Occurrence of acute infarct-like myocarditis following COVID-19 vaccination: just an accidental co-incidence or rather vaccination-associated autoimmune myocarditis? *Clin Res Cardiol*. 2021;110(11):1850-1854.
8. Rosner CM, Genovese L, Tehrani BN, et al. Myocarditis temporally associated with COVID-19 vaccination. *Circulation*. 2021;144(6):502-505.
9. Ujueta F, Azimi R, Lozier MR, Poppiti R, Ciment A. Lymphohistocytic myocarditis after Ad26.COVS viral vector COVID-19 vaccination. *Int J Cardiol Heart Vasc*. 2021;36:100869.
10. Bozkurt B, Kamat I, Hotez PJ. Myocarditis with COVID-19 mRNA vaccines. *Circulation*. 2021;144(6):471-484.
11. Sinagra G, Merlo M, Porcari A. Exploring the possible link between myocarditis and mRNA COVID-19 vaccines. *Eur J Intern Med*. 2021;92:28-30.
12. Cooper LT Jr. Myocarditis. *NEngl J Med*. 2009;360(15):1526-1538.
13. Van Linthout S, Tschöpe C. Viral myocarditis: a prime example for endomyocardial biopsy-guided diagnosis and therapy. *Curr Opin Cardiol*. 2018;33(3):325-333.
14. Su JR, McNeil MM, Welsh KJ, et al. Myopericarditis after vaccination, vaccine adverse event reporting system (VAERS), 1990–2018. *Vaccine*. 2021;39(5):839-845.
15. Rijkers GT, Weterings N, Obregon-Henao A, et al. Antigen presentation of mRNA-based and virus-vectored SARS-CoV-2 vaccines. *Vaccines (Basel)*. 2021;9(8):848.

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