SHORT COMMUNICATION

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Review of the current literature regarding cardiac adverse events following COVID-19 vaccination

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INTRODUCTION

The severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), which was responsible for coronavirus disease 2019 (COVID-19) infection, was discovered in Wuhan, China, in December 2019¹. Since then, the disease has spread globally, resulting in a pandemic. Because there is no specific antiviral treatment for COVID-19 disease, vaccination seems to appear the most effective vehicle for controlling the infection. Until now, many vaccines have been developed and approved for immediate use by the health authorities. Two types of messenger RNA (mRNA)based COVID-19 vaccines, namely, BNT162b2 mRNA (Pfizer-BioNTech, NY) and mRNA-1273 (Moderna, Cambridge, MA), have been administered in hundreds of millions of doses since they have received provisional Food and Drug Administration (FDA) approval in the United States in December 2020¹. Janssen Ad26.COV2.S (Johnson and Johnson, New Brunswick, NJ) and The ChAdOx1 [Oxford/AstraZeneca (AZD1222)] were recombinant types of vaccines, in which replication-deficient human adenovirus type 26 vector was used to transfer the virus¹. Although side effects from these vaccines are generally mild and transient, there has been an upsurge of cases with cardiac adverse events reported after COVID-19 vaccination. As a result, the objective of this review was to assess all cardiovascular adverse events reported following COVID-19 immunization, as well as the likely mechanisms behind them.

METHODS

We searched the database of PubMed, Embase, and Cochrane for all possible cardiac adverse events reported after COVID-19 vaccination using the following search inputs until September 13, 2021: "COVID-19 vaccine-induced acute myocarditis," "COVID-19 vaccine-induced acute perimyocarditis," "COVID-19 vaccine-induced acute myocardial infarction," "COVID-19 vaccine-induced ST elevation myocardial infarction," and "COVID-19 vaccine-induced acute coronary syndrome." Only papers written in English were included in this review. Additionally, following a review of the references in the relevant publications, any further papers were collected. Our review was restricted to only cardiac adverse events reported after COVID-19 vaccination. In total, 68 relevant cases were found in the literature. Of them, 61 cases were diagnosed with acute myocarditis (AM), one case with acute perimyocarditis, five cases with acute myocardial infarction (AMI), and one case with Kounis syndrome after COVID-19 vaccination.

Vaccination types

Table 1 describes the vaccine types, symptoms onset, and COVID-19 polymerase chain reaction (PCR) positivity of all published cases. The majority of AM patients who suffered cardiac adverse events after receiving COVID-19 vaccination had previously been immunized with mRNA-based vaccines. [In total = 65 cases, 35 of them with BNT 162b2 (Pfizer) and 30 of them with mRNA-1273 SARS-CoV-2 (Moderna)²⁻¹⁵.] Only three cases had a history of vaccination with adenovirus vector origin [two with Covishield (AZD1222) and one with Janssen Ad.26.COV2.S (Johnson and Johnson)².] Almost all of the AM cases (60/61) were diagnosed following the injection of vaccinations made with mRNA technology, and the majority of them developed

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	Vaccine types	Presentation after the second dose vaccine (%)	History of COVID-19? (%)	Is the patient COVID-19 polymerase chain reaction positive?	Does the patient have a nucleocapsid antibody?	Time between last vaccination and symptom onset, days
Case series ²	5 BNT162b2 (Pfizer); 1 mRNA-1273 (Moderna); 1 J&J	71	14	6/7 patients were tested, all were negative	4/7 patients tested, all were negative	3 (2–7)
Case series³	5 BNT 162b2 (Pfizer); 3 mRNA1273 (Moderna)	88	25	All were negative	NA	3 (1–4)
Case series⁴	BNT 162b2 (Pfizer)	83	No	All were negative	All were negative	2.5 (1–16)
Case series⁵	2 BNT 162b2 (Pfizer), 2 mRNA1273 (Moderna)	100	No	Negative	NA	2.5 (1–5)
Case series ⁶	7 BNT 162b2 (Pfizer), 16 mRNA1273 (Moderna)	87	13	19/23 were tested, all were negative	NA	2 (1–4)
Case ⁷	BNT62b2 (Pfizer)	100	100	Negative	%100	3
Case ⁸	BNT162b2 (Pfizer)	100	No	Negative	Negative	1
Case ⁹	BNT162b2 (Pfizer)	100	No	Negative	Negative	3
Case ¹⁰	mRNA-1273 (Moderna)	100	No	Negative	NA	4
Case ¹¹	mRNA-1273 (Moderna)	100	No	Negative	Negative	1
Case ¹²	BNT62b2 (Pfizer)	100	No	Negative	Negative	1
Case ¹³	BNT62b2 (Pfizer)	100	No	Negative	Negative	3
Case ¹⁴	mRNA-1273 (Moderna)	100	No	Negative	Negative	1
Case series ¹⁵	BNT62b2 (Pfizer)	100	No	Negative	Negative	12 h–3
Case ¹⁶	BNT62b2 (Pfizer)	No*	No	Negative	Negative	1 h
Case ¹⁷	Covishield (Azd1222)	No*	No	Negative	Negative	2
Case ¹⁸	(Moderna)	No*	No	Negative	Negative	1
Case series ¹⁹	mRNA-1273 (Moderna)	No*	No	Negative	Negative	1–5
Case ²⁰	Azd1222 (Oxford University and AstraZeneca)	No*	No	Negative	Negative	2 h

Table 1. Vaccine types, symptoms onset, and COVID-19 PCR positivity of all published cases.

*Presentation after the first dose vaccine. NA: not applicable.

symptoms 1–3 days after the second dose of immunization $(57/61)^{2-15}$. In contrast to AM cases, the majority of AMI cases (4/5) emerged after the first dose of mRNA-based vaccination was administered¹⁶⁻¹⁹. Only one case of Kounis syndrome had been reported in the literature, and this allergic response occurred 2 h after the first dose of Covishield (AZD1222) vaccination²⁰. Interestingly, the COVID-19 PCR test was negative in all cases.

Baseline clinical characteristics, electrocardiographic findings, and laboratory findings

Table 2 summarizes the baseline characteristics, presenting symptoms, electrocardiography, and laboratory results in all published cases. Patients who were diagnosed with AM were relatively younger and almost all of them were male. By contrast, AMI cases were older. The common complaint in all patients was chest pain. Electrocardiography findings in AM cases ranged from no ischemic changes to ST elevation, PR depression, and nonspecific ST changes²⁻¹⁵. Remarkably, patients who presented with AMI following immunization had ST elevation only in inferior leads¹⁶⁻²⁰. Troponin levels were measured in all patients who developed a cardiac event after vaccination. In all of them, it was reported above the reference range. The data on brain natriuretic peptide (BNP) levels were shared in very few cases^{2,5,10-12,13-15}. On the other hand, C-reactive protein (CRP) levels were elevated in all reported cases. Contrary to COVID-19 infection, lymphopenia was not detected in most patients with post-vaccine cardiac events.

Imaging findings

Table 3 displays the imaging data, in-hospital treatment, and outcomes of all cases. Echocardiography was performed in most cases since it was in the diagnostic algorithm of diseases such as AM and AMI. Left ventricle wall motion defect was observed in all patients with AMI, whereas AM patients had findings in the spectrum from preserved left ventricular ejection fraction (LVEF) without segmental abnormalities to global hypokinesia and low LVEF^{2-6,8-11,13-16}. Cardiac magnetic resonance imaging was performed in almost all AM cases, which demonstrated a subepicardial late gadolinium enhancement and myocardial edema compatible with AM²⁻¹⁵. Although it was considered the gold standard for the diagnosis of AM, the endomyocardial biopsy was not performed on any patients.

	Age, gender	Presenting symptoms	Diagnosis	Electrocardiographic (ECG) findings	Lab findings
Case series ²	24 (19–30), all cases were male	Chest pain was present in all cases, 42% had nonspecific symptoms	AM	4 patients had ST elevations, 1 patient had nonspecific ST/T changes	Lymphopenia: – CRP: elevated in 71% Troponin: elevated in all cases BNP: elevated in 50%
Case series ³	29 (21–56), all cases were male	Chest pain was present in all cases, 63% had nonspecific symptoms	AM	6 patients had ST elevation, 1 patient had peaked T waves, one patient had normal ECG	Lymphopenia: – CRP: elevated in 88% Troponin: elevated in all cases
Case series ⁴	22 (16–45), all cases were male	Chest pain was present in all cases, 33% had nonspecific symptoms	AM	All cases had ST elevations	Lymphopenia: – CRP: elevated in all cases Troponin: elevated in all cases
Case series⁵	30 (23–70), 75% of cases were male	Chest pain was present in all cases, 33% had nonspecific symptoms	AM	All cases had ST elevation, two cases had PR depression	Lymphopenia: – CRP: elevated in all cases Troponin: elevated in all cases BNP: elevated in 50%
Case series ⁶	25 (20–51), all cases were male	Chest pain was present in all cases	AM	19/23 cases had ST elevations, T-wave inversions, and nonspecific ST changes	Lymphopenia: – CRP: NA Troponin: elevated in all cases
Case ⁷	56, Male	Chest pain	AM	ST elevation	Lymphopenia: – CRP: elevated Troponin: elevated

Table 2. Baseline characteristics, presenting symptoms, electrocardiography, and laboratory findings of all published cases.

Table 2. Continuation.

	Age,	Presenting	Diagnosis	Electrocardiographic	Lab
	gender	symptoms		(ECG) findings	findings
Case ⁸	39, Male	Chest pain, myalgia, fatigue, fever	AM	ST elevation	Lymphopenia: – CRP: NA Troponin: elevated
Case ⁹	30, Male	Chest pain, myalgia, fatigue, fever	AM	ST elevation	Lymphopenia: – CRP: elevated Troponin: elevated
Case ¹⁰	24, Male	Chest pain, myalgia, fatigue, fever	AM	No ischemic changes	Lymphopenia: — CRP: elevated Troponin: elevated BNP: normal
Case ¹¹	52, Male	Chest pain, myalgia, fatigue, fever	AM	Incomplete right bundle branch block and left axis deviation	Lymphopenia: – CRP: elevated Troponin: elevated BNP: elevated
Case ¹²	66, Male	Chest pain, myalgia, fatigue, fever	AM	ST elevation	Lymphopenia: — CRP: NA Troponin: elevated
Case ¹³	24, Male	Chest pain, myalgia, fatigue, fever	AM	No ischemic findings	Lymphopenia: – CRP: elevated Troponin: elevated BNP: elevated
Case ¹⁴	34, Male	Chest pain, myalgia, fatigue, fever	AM	Lateral PR depression and ST elevation mirrored in aVR with PR elevation and ST depression	Lymphopenia: – CRP: elevated Troponin: elevated BNP: elevated
Case series ¹⁵	15 and 22, all cases were male	Chest pain, myalgia, fatigue, fever	AM and acute myopericarditis	J-point elevation in the lateral leads with slightly widened QRS complexes and no ischemic findings	Lymphopenia: – CRP: elevated Troponin: elevated BNP: elevated
Case ¹⁶	86, Male	Chest pain, myalgia, fatigue, fever	STEMI	ST elevation inferior wall	Lymphopenia: – CRP: NA Troponin: NA BNP: NA
Case ¹⁷	63, Male	Chest pain, myalgia, fatigue, fever	STEMI	ST elevation in leads II and III and aVF	Lymphopenia: NA CRP: NA Troponin: elevated
Case ¹⁸	96, Female	Chest pain, myalgia, fatigue, fever	STEMI	ST-segment elevation in the anterior and inferior leads	Lymphopenia: NA CRP: NA Troponin: elevated
Case series ¹⁹	42 and 68, 50% of cases were male	Chest pain	STEMI and AMI	ST elevation in leads II and III and aVF	Lymphopenia: NA CRP: NA Troponin: elevated
Case ²⁰	62, Female	Chest pain	Kounis syndrome	ST elevation in inferior leads (II, III, and AVF) and reciprocal ST segment depression in lead I and AVL	Lymphopenia: NA CRP: NA Troponin: elevated

AM: acute myocarditis; CRP: C-reactive protein; BNP: brain natriuretic peptide; STEMI: ST elevation myocardial infarction; AMI: acute myocardial infarction; NA: not applicable.

	Echocardiographic findings (%)	Cardiac MRI findings	Median hospitalization, days (range)	In-hospital treatment (%)
Case series ²	Abnormal in 57 [mild hypokinesia in 3, 1 reduced LVEF, one mild LV enlargement], normal in 43.	All cases had LGE, one with wall motion abnormality, three with myocardial edema in T2	3 (2–4)	43 with NSAIDs, 43 with colchicine, 43 with famotidine, 14 with steroids
Case series ³	All cases had motion abnormality with regional or generalized hypokinesia	All cases had LGE, six with myocardial edema	All cases were reported as stable	38 with NSAIDs, 25 with colchicine, 13 with steroids
Case series ⁴	2/6 with hypokinetic segments but preserved LVEF, 4/6 had normal LVEF	All cases had mild subepicardial edema and LGE	6 (4–8)	100 with NSAIDs and colchicine
Case series⁵	One patient with LVEF 40, the others had normal LVEF	All cases had LGE, increased T1 and T2 intensity	3 (2–4)	50 with NSAIDs, 75 with colchicine, 25 with steroid
Case series ⁶	4/23 cases had LVEF <50	All cases had subepicardial LGE or focal myocardial edema	NA	NA
Case ⁷	NA	LGE and myocardial edema in T2 imaging	7	NA
Case ⁸	Normal LVEF	Subepicardial enhancement	6	Anti-inflammatory medications
Case ⁹	Abnormal wall motion abnormality and mild pericardial effusion	Subepicardial LGE of the myocardium	7	Beta-blocker, acetylsalicylic acid, steroid
Case ¹⁰	Normal LVEF	Patchy mid-myocardial and epicardial LGE with edema	NA	Beta-blocker
Case ¹¹	No wall motion abnormalities, LVEF was preserved	Mild myocardial and subepicardial linear and nodular LGE and mild hypokinesia	4	ACE inhibitor, beta- blocker
Case ¹²	Reduced LVEF of 44	Edema on T2 sequences and subepicardial enhancement in the lateral mediastinal region	NA	NA
Case ¹³	Normal LVEF	Subepicardial enhancement involving the lateral wall	NA	NA
Case ¹⁴	Reduced LVEF of 43 without pericardial effusion	Subepicardial LGE in the anterolateral and inferolateral segments, as well as patchy myocardial edema on T2	5	High-dose aspirin, colchicine, ACE inhibitor, beta-blocker
Case series ¹⁵	Normal LVEF	NA	2	Aspirin, NSAIDs, and colchicine
Case ¹⁶	NA	NA	Not survived	Balloon angioplasty and glycoprotein IIb/ Illa receptor inhibitor (eptifibatide)

Table 3. Imaging findings, in-hospital treatment, and outcomes of all published cases.

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Table	3.	Contin	uation.
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	Echocardiographic findings (%)	Cardiac MRI findings	Median hospitalization, days (range)	In-hospital treatment (%)
Case ¹⁷	Inferior wall hypokinesia with LVEF of 50	NA	5	Thrombolysed with 1.5 million IU streptokinase, and anti-platelets and anti- anginal drugs
Case ¹⁸	Anterior and inferior wall hypokinesia with LVEF of 35 %	NA	3	Heparin
Case series ¹⁹	LVEF of 50% and hypokinesia of the anterolateral and inferolateral walls and LVEF of 60%, with hypokinetic inferior and inferolateral walls	NA	2–7	PCI
Case ²⁰	Inferior wall motion abnormality and preserved LVEF	NA	3	PCI

MRI: magnetic resonance imaging; LVEF: left ventricle ejection fraction; LGE: late gadolinium enhancement; NA: not applicable; NSAIDs: nonsteroidal anti-inflammatory drugs; PCI: percutaneous coronary intervention.

In-hospital treatment and outcomes

No case of acute fulminant myocarditis was reported after COVID-19 vaccination. Most reported AM cases were hospitalized for 3–5 days on average, and all of them were discharged uneventfully^{2-5,7-9,11,14,15}. In the treatment of AM, high-dose aspirin, colchicine, beta-blockers, and steroids were most preferred^{1-5,12,14}. In addition to anti-ischemic and anti-aggregant therapy, the primary percutaneous coronary intervention was performed in patients presenting with AMI¹⁶⁻²⁰. All AMI cases were discharged uneventfully, except the 86-year-old male patient who did not survive during the in-hospital course¹⁶.

DISCUSSION

AM is generally regarded as an uncommon adverse effect following vaccination. According to reports, the majority of previously documented post-vaccine AM cases were subclinical and were discovered by routine pre- and post-vaccine troponin level assessments²¹. However, in our review, all the cases documented following COVID-19 immunization were symptomatic. This implies that asymptomatic individuals might not be identified, and as a result, cardiac events following immunization might be significantly greater than predicted.

Although the causes of AM due to COVID-19 vaccinations are not well understood, several potential pathophysiological explanations have been proposed. It has been considered that in some people with genetic vulnerability, the immunological response to mRNA-based COVID-19 vaccines may be uncontrollable, resulting in the activation of an abnormal innate and acquired immune response²². Also, both dendritic cells and Toll-like receptor-expressing cells subjected to mRNA may still be able to produce cytokines in certain people, albeit this may be significantly decreased when exposed to mRNA with nucleoside alterations as opposed to unmodi-fied RNA²². As a result, the immune system may recognize the mRNA as an antigen, leading to hyperactivation of the inflammatory and immunologic pathways, which may have a role in the occurrence of AM in certain people as part of a systemic response²².

During vaccination, an allergic reaction may develop, which can be classified as a vaccine-related adverse effect. It is always difficult to determine whether a response is caused by the vaccination or by other causes. Adjuvants are usually included in the vaccines to enhance stability, solubility, and absorption, which can result in IgE-mediated anaphylactic responses following immunization. This might be one explanation for AMI following the COVID-19 vaccination. The fact that all published AMI cases had their complaints started within a short time after the initial dosage of vaccination supports this hypothesis. Another potential AMI cause, as proposed by Warkentin et al., is vaccine-induced prothrombotic immune thrombocytopenia, which is similar to heparin-induced thrombocytopenia and leads to thrombotic manifestation²³.

Future perspective

The number of documented cases supports the "very uncommon" interpretation of vaccine-related cardiac side effects despite the fact that hundreds of millions of COVID-19 vaccinations have been administered globally. It was also clearly demonstrated that the majority of the patients with cardiac adverse events demonstrated full recovery in terms of both symptoms and imaging. Moreover, it must be highlighted that since there has been no causative link between COVID-19 vaccinations and cardiac events, the effectiveness of the COVID-19 vaccination far exceeds some possible drawbacks. Consequently, more research on AM, AMI, and other cardiac events before and after COVID-19 vaccination will enrich the literature about the long-term effects of the vaccination and determining the incidence rate.

AUTHORS' CONTRIBUTIONS

T**Ç**: Conceptualization, Formal Analysis, Writing – original draft, Writing – review & editing. **MIH**: Conceptualization, Formal Analysis, Writing – review & editing. **ALO**: Supervision, Formal Analysis, Writing – review & editing. **VÇ**: Data curation, Funding acquisition, Resources, Writing – review & editing. **MS**: Data curation, Funding acquisition, Resources, Writing – review & editing. **SY**: Funding acquisition, Resources, Writing – review & editing.

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ERRATUM

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Where it reads: Mert İlker Hayıroçlu

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