CASE REPORT

Temporal evolution of electrocardiographic anomalies observed in a young woman after COVID-19 vaccine

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Electrocardiographic alterations of ventricular repolarization have already been reported in cases of myocarditis following coronavirus disease-19 (COVID-19) vaccine, but it is not clear how long they persist. A 21-year-old female presented with chest discomfort and diffuse myalgia a week after receiving the first dose of a messenger RNA (mRNA) COVID-19 vaccine. The 12 leads standard electrocardiogram unveiled negative T waves in anterior and inferior leads, while her troponin-I values resulted in the upper limit. A mild form of post-COVID-19 vaccine myocarditis was diagnosed since the echocardiogram excluded major systolic alterations and pericardial effusions. The patient refused hospitalization, but luckily, she remained hemodynamically stable, presenting a quick clinical response to oral non-steroidal anti-inflammatory therapy. However, the electrocardiographic abnormalities required weeks for resolving. In our case, the later normalization of electrocardiographic anomalies was not associated with an ominous clinical course.

Key words: COVID-19 vaccines, electrocardiography, myocarditis

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INTRODUCTION

Coronavirus disease-19 (COVID-19) pandemic represents one of the major challenges for the health systems globally.[1] Indeed, COVID-19 has exerted a negative impact on worldwide populations in terms of mortality and morbidity, configuring it as a disastrous event on a planetary scale.[2]

The availability of vaccines against severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2) infection has provided a fundamental preventive tool for contrast COVID-19, consenting to save around 140,000 lives only in the United States by the year 2021.[3,4] Currently, the COVID-19 vaccines approved by the World Health Organization include messenger RNA (mRNA) and viral vector vaccines.[5]

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COVID-19 vaccines can rarely cause severe adverse reactions, comprising myopericarditis, which may provoke electrocardiographic changes in the ventricular repolarization.[6]

However, how long these electrocardiographic alterations persist and the prognostic significance of their duration is unclear.

CASE REPORT

A 21-year-old young woman underwent a cardiological examination as an outpatient at our institute for chest discomfort, diffuse myalgia, and asthenia. These symptoms had been onset for 1 week, starting 2 days after she received the first dose of a COVID-19 mRNA vaccine. The patient's medical history was silent, except for reported claustrophobia. She was not taking any medication. On presentation, her blood pressure was

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110/70 mmHg, and her oxygen saturation in room gas was 98%.

The patient was normal weight (body mass index: 23 kg/m^2). The cardiovascular, thoracic, abdominal, and neurological examinations were normal.

The electrocardiogram (ECG) showed sinus tachycardia with a heart rate of 102/min and pronounced ventricular repolarization anomalies [Figure 1a]. Even if the echocardiogram excluded contractility anomalies and pericardial effusion [Figure 1b], the clinical case was suggestive of a mild myocarditis induced by the COVID-19 vaccine, according to Bozkurt criteria, for the presence of typical symptoms and ECG alterations.[7]

Hence, hospitalization was proposed to the patient to ensure a strict clinical monitoring, but she refused the hospital admission. She also refused to undergo a cardiac magnetic resonance imaging (MRI) with contrast for claustrophobia. However, she accepted to perform other laboratory and instrumental examinations as outpatient.

Table 1 reports the values of analytes of interest measured from the patient's blood samples. Of note, C-reactive protein was slightly increased, whereas high sensibility troponin-I was at the upper limits and decreased 24 h later. The nasopharyngeal swab for RNA SARS-Cov-2 and the antibody titers for other infectious pathogens (adenovirus, enterovirus, herpes virus, human immunodeficiency virus, hepatitis C virus, mycoplasma, and streptococcus) were also negative.

The patient underwent even a 24-h Holter ECG, which did not identify any major arrhythmias.

Anti-inflammatory oral therapy (ibuprofen 600 mg daily) was prescribed for the treatment of patient's symptoms, which resolved for 10 days. A month later, the onset of symptoms, an ECG documented a reduction of the electrocardiographic abnormalities [Figure 1c]. 2 months

Table 1: Laboratory tests

Analyte	Result	Reference range
Hs-TnI (ng/L) on presentation	11.8	2.3–12
Hs-TnI (ng/L) after 24 h	9.3	2.3–12
NT-proBNP (pg/mL)	64	Up to 125
Cr (mg/dL)	0.69	0.5–1
BUN (mg/dL)	18	16–25
Na (mEq/L)	139	135–150
K (mEq/L)	4.1	3.5–5.3
Ca (mg/dL)	10.5	8.5–11
Mg (mg/dL)	2.3	1.5–2.5
AST (Unit/L)	19	Up to 40
ALT (Unit/L)	13	Up to 40
CK (Unit/L)	56	Up to 200
LDH (Unit/L)	154	Up to 247
Glycemia (mg/dL)	84	60–110
WBC (per mcrL)	7990	4000–9000
HB (g/dL)	13	12.5–15
CRP (mg/L)	8.4	Up to 8
TSH (mcrU/mL)	3.163	0.340–5.600
ANA (positivity, dilution)	Negative	Positive ≥1:80

Hs-TnI=high-sensitive troponin I; NT-proBNP=N-terminal prohormone of brain natriuretic peptide; Cr=Creatinine; BUN=Blood urea nitrogen; Na=Sodium; K=Potassium; Ca=Calcium; Mg=Magnesium; AST=Aspartate aminotransferase; ALT=Alanine aminotransferase; CK=Creatine kinase; LDH=Lactate dehydrogenase; WBC=White blood cells count; HB=Hemoglobin; CRP=C-reactive protein; TSH=Thyroid-stimulating hormone; ANA=Antinuclear antibody

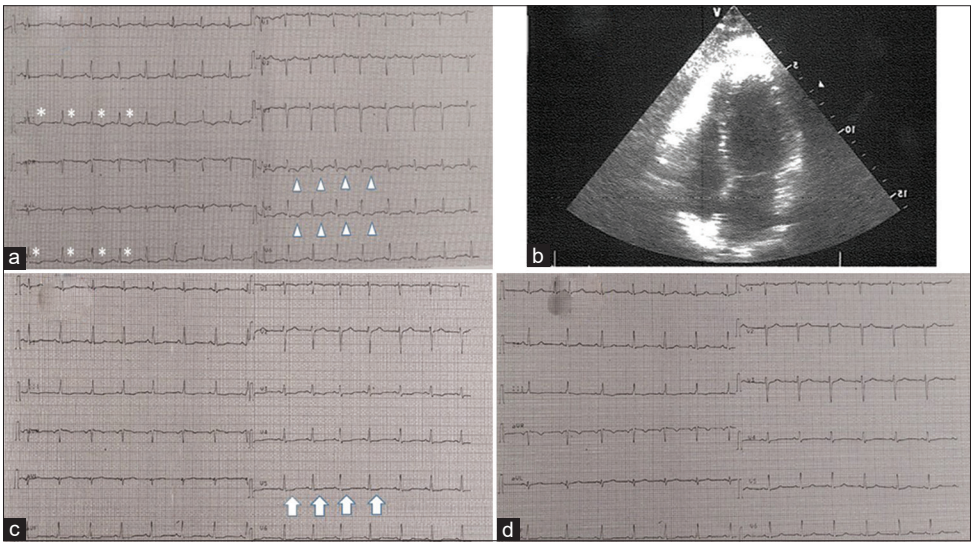

Figure 1: Electrocardiographic and echocardiographic features at presentation and on follow-up. (a) Electrocardiogram at presentation: sinus tachycardia with ST-segment depression and negative T waves in V4-V5 leads (white arrowheads). Negative T waves are also evident In DII, DIII, and aVF leads (white asterisks). (b) Echocardiographic four-chamber systolic views at presentation: the dimension of heart chambers are normal, absence of pericardial effusion. (c) Electrocardiogram at 1 month: low voltage T waves are present in V4-V5. Minimal J point depression is observable in V5 lead (white arrows). T waves in DII, DIII, and aVF leads are biphasic. (d) Electrocardiogram at 2 months: significant recovery phase anomalies are no longer visible. Biphasic T waves persist only in DIII lead

later, a further ECG documented the resolution of ECG abnormalities [Figure 1d]. Then, the patient underwent a maximal exercise test, which resulted in negative for myocardial ischemia.

DISCUSSION

The present clinical case confirms that electrocardiography, i.e. a widely available and not invasive technique, allows the early identification of cardiac electrical abnormalities associated with myocarditis after COVID-19 vaccination. Electrocardiography significantly helps the diagnosis since cardiac necrosis markers can result negative, whether they are measured several days after symptoms' onset. Certainly, cardiac MRI is a key diagnostic technique for myocarditis, but it is relatively expansive and not always available. Moreover, it is limited by patient-related factors, including claustrophobia.[8]

Electrocardiographic abnormalities suggestive of myocarditis after the COVID-19 vaccine have already been described, but no case report, to our knowledge, has outlined the temporal evolution of the electrocardiographic changes.[9]

A recent systematic review showed that electrocardiographic alterations were present in about two-thirds of patients with myocarditis after the mRNA COVID-19 vaccine, consisting, especially of ST-segment abnormalities.[10]

Although the occurrence of myocarditis is more frequent in young men after the second dose of mRNA COVID-19 vaccine, our case shows that this diagnosis must be considered even in other situations (e.g. young women undergoing the first dose of mRNA COVID-19 vaccine).

In addition, our case highlights that the electrocardiographic abnormalities from COVID-19 vaccine myocarditis may take a long time to resolve, even in mild cases. At present, the prognostic significance of a later resolution of electrocardiographic abnormalities observed after COVID-19 vaccines is unknown. The present case suggests that a longer duration of ECG abnormalities is not associated with a worse course.

The pathogenetic mechanism underlying COVID-19 postvaccine myocardial damage is not yet understood, but it has been hypothesized that it could involve autoimmune phenomena.[11] However, the risk of myopericarditis after the COVID-19 mRNA vaccine is so low that it should not discourage vaccination.[12]

Surely, the identification of abnormalities of the ventricular repolarization after COVID-19 vaccination needs the investigation of other pathologic conditions in differential diagnosis, including anemia, ionic imbalances, thyroid dysfunction, ischemic heart disease, and myopericarditis of other etiologies.

In conclusion, the present report describes a single case of subtle myocarditis induced by the COVID-19 vaccine in a young woman. Therefore, it could not be representative of the clinical course of patients of diverse ages and gender. However, it importantly demonstrates that electrocardiography, used in conjunction with other diagnostic examinations, significantly helps clinicians in the assessment and follow-up of patients with COVID-19 vaccine-related myocarditis.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given her consent for her images and other clinical information to be reported in the journal. The patient understands that her name and initials will not be published and due efforts will be made to conceal her identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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