Erciyes Med J 2022; 44(1): 109–11 • DOI: 10.14744/etd.2021.39924
CASE REPORT – OPEN ACCESS

Coronavirus Disease-Induced Acute Myocarditis: Two Clinical Presentations with Different Electrocardiographic Findings

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ABSTRACT

Background: In this report, we present two cases of coronavirus disease (COVID-19)-related acute myocarditis (AM) with different clinical presentations and electrocardiographic (ECG) findings.

Case Report: The first case was a 33-year-old male patient who presented with chest pain to the emergency department (ED). The ECG result suggested an acute coronary syndrome (ACS). However, coronary angiography (CAG) results showed normal coronary arteries, and the cardiac magnetic resonance imaging findings were compatible with COVID-19-induced AM. The ECG revealed a normalization of the T-wave negativity in the anterolateral precordial leads. The second case was a 41-year-old female patient who presented with ST-elevation myocardial infarction (STEMI) to the ED. CAG revealed normal coronary arteries, and the reverse-transcription polymerase chain reaction test result for COVID-19 was positive. The patient was diagnosed as having COVID-19-induced AM.

Conclusion: Our cases demonstrate that clinicians should be aware that some patients with COVID-19-induced AM can present with ECG findings mimicking ACS, including STEMI.

Keywords: COVID-19, acute myocarditis, cardiovascular disease, electrocardiography, presentation

Cite this article as:
Çınar T, Çiçek V, Kılıç Ş, Asal S, Selçuk M, Keskin M, et al. Coronavirus Disease-Induced Acute Myocarditis: Two Clinical Presentations with Different Electrocardiographic Findings. Erciyes Med J 2022; 44(1): 109-11.

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Submitted
06.01.2021

Accepted
18.02.2021

Available Online
01.03.2021

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INTRODUCTION

Acute myocarditis (AM) occurs at the early onset of inflammation of the cardiac tissue. It has different causative agents such as autoimmunity and exposure to drugs or toxic substances, of which acute viral infections are the most common (1). The pathophysiology of acute viral infections that lead to AM involves a combination of direct cell damage and cytokine storm due to T lymphocyte-mediated cytotoxicity (2). AM occurs in 1%–5% of patients with acute viral infections (1).

A new type of coronavirus named severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) by the World Health Organization has rapidly spread worldwide, infecting millions of people in a short time. Although coronavirus disease (COVID-19) caused by SARS-CoV-2 mainly manifests as respiratory signs and symptoms, it may also cause cardiac injuries, including acute coronary syndrome (ACS) and AM (3). Hence, in this case series, we aimed to describe two cases of COVID-19-induced AM with different clinical presentations and electrocardiographic findings.

CASE REPORTS

CASE 1 – A 33-year-old male patient was hospitalized in our pandemic inpatient unit 12 days ago because of COVID-19 pneumonia. He was discharged after he tested negative for COVID-19 in a reverse-transcription polymerase chain reaction (RT-PCR) test. He was later admitted to our emergency department (ED) with a 2-day history of chest pain that started a day after discharge from our hospital. His physical examination revealed a blood pressure of 123/78 mmHg and a heart rate of 78 beats per minute. Electrocardiography (ECG) revealed a normal sinus rhythm and symmetrical T-wave negativity in the anterolateral precordial leads (Fig. 1a). The patient was then admitted to the intensive care unit. The control RT-PCR test result was negative. Transthoracic echocardiography (TTE) revealed a normal ejection fraction without left ventricular (LV) wall motion defects. He was treated with medical therapy, including a 300-mg acetylsalicylic acid (ASA), a loading dose of clopidogrel, low-molecular-dose heparin, statin, and beta-blocker treatment. A laboratory analysis revealed an elevated cardiac troponin I (12.000 ng/L; normal range, 0–14 ng/L) and D-dimer levels (1.2 µg/mL; normal range, 0–0.5 µg/mL), along with a normal C-reactive protein (CRP) level (3 mg/L; normal range, 0–5 mg/L). The patient was taken to the catheterization laboratory with a preoperative diagnosis of ACS. However, coronary angiography (CAG) revealed that normal epicardial coronary arteries (Fig. 1b, c). Cardiac magnetic resonance imaging (MRI) was then performed and revealed late gadolinium uptake in the lower inferior and lateral walls of the left ventricle (Fig. 1d,

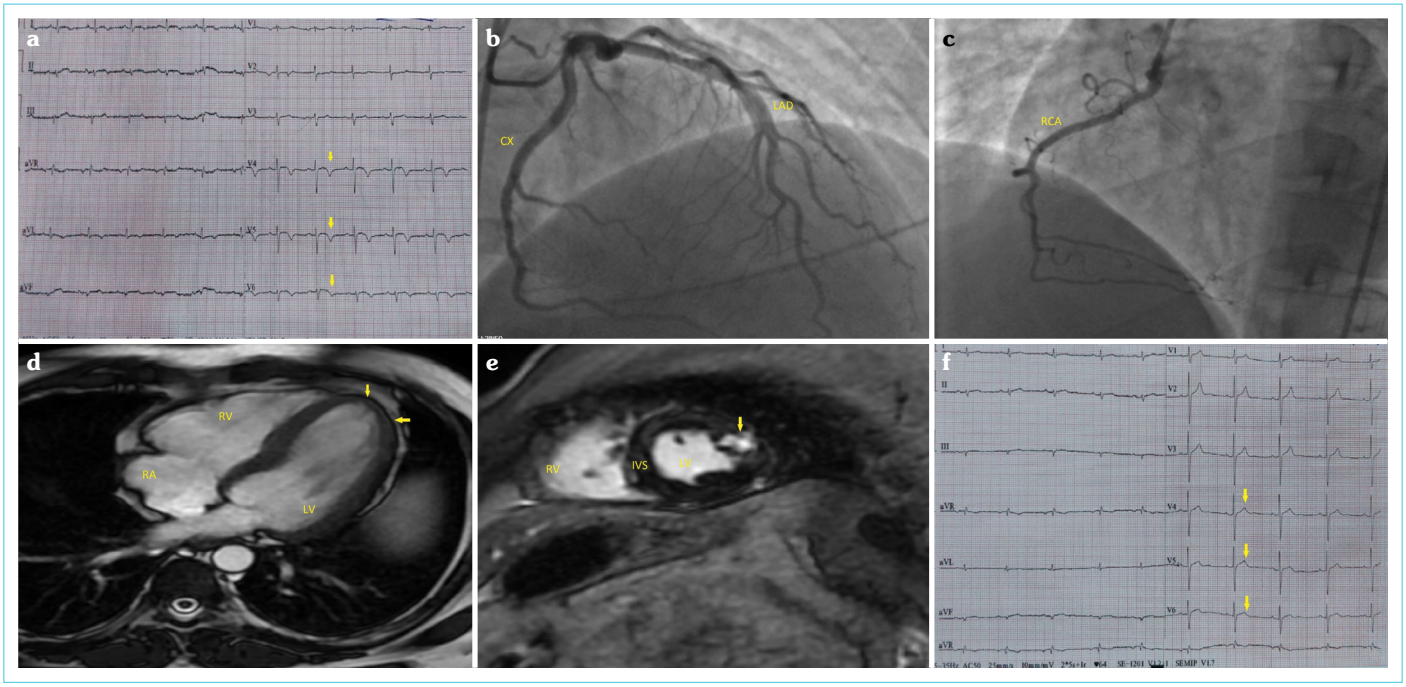

Figure 1. (a) Electrocardiogram of the patient obtained in the emergency department (arrows). (b, c) Coronary angiogram showing normal epicardial coronary arteries. (d, e) Cardiac magnetic resonance image showing late gadolinium uptake in the apical, apicolateral, and mid-lateral walls of the left ventricle in the long-axis 4-chamber apical and short-axis 2-chamber views (arrows). (f) Control electrocardiogram of the patient before discharge from the hospital (arrows)

LAD: Left anterior descending artery; CX: Circumflex artery; RCA: Right coronary artery; LA: Left atrium; LV: Left ventricle; RA: Right atrium; RV: Right ventricle; IVS: Interventricular septum

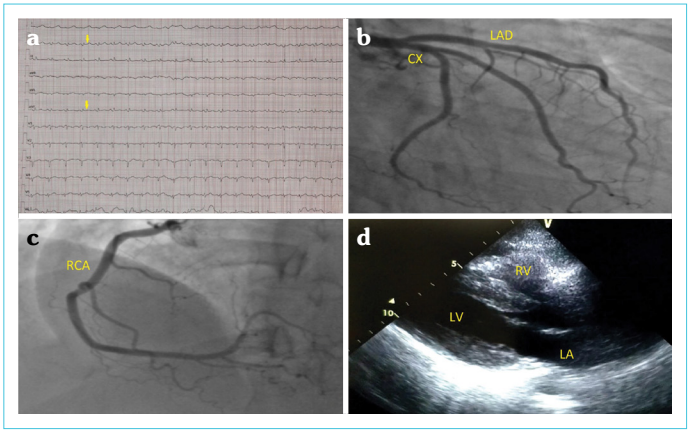

Figure 2. (a) Electrocardiogram of the patient (arrows). (b, c) Coronary angiogram showing normal epicardial coronary arteries. (d) Control transthoracic echocardiogram showing a normal left ventricle ejection fraction

e). Control ECG revealed normalization of the T-wave negativity in the anterolateral precordial leads (Fig. 1f). The hospital course of the patient was uneventful, and he was discharged with outpatient clinic follow-up and ASA and beta-blocker therapies.

CASE 2 – A 41-year-old female patient was admitted to the ED because of typical chest pain since 2 hours before. At admission, her blood pressure was 81/47 mmHg; heart rate, 115 beats per minute; and O2 saturation, 93% on room air. ECG revealed ST elevation in the inferior leads (Fig. 2a). Bedside TTE revealed an LV ejection fraction of 40% and global LV hypokinesia that was more prominent in the inferior and inferolateral walls. Before CAG, a 300-mg ASA and a loading dose of clopidogrel were administered. The patient was taken to the catheterization laboratory for primary percutaneous coronary intervention. However, CAG revealed normal epicardial coronary arteries (Fig. 2b, c). The patient was transferred to the coronary intensive care unit with a preoperative diagnosis of AM. Laboratory examination revealed elevated cardiac troponin I (4.000 ng/L; normal range, 0–14 ng/L) and CRP levels (13 mg/L; normal range, 0–5 mg/L) along with a normal D-dimer level (0.1 µg/mL; normal range, 0–0.5 µg/mL). Although the lymphocyte levels were normal, the RT-PCR test results for COVID-19 were positive. The patient was therefore transferred to an isolated intensive care unit. During the isolated intensive care unit follow-up, inotrope was administered to increase the patient's blood pressure, as she was hypotensive. In addition, the COVID-19 treatment protocol (hydroxyl chloroquine and favipiravir) and low-molecular-dose heparin were administered. After 2 days of inotrope treatment, her blood pressure normalized. Control TTE revealed a normal LV ejection fraction (Fig. 2d). Cardiac MRI was recommended to confirm the diagnosis of AM; however, the patient did not accept the medical advice. She was discharged on the 10th hospitalization day with medical therapy, including ASA and beta-blockers.

DISCUSSION

In contrast to our cases, most reported cases have shown that patients diagnosed as having COVID-19-related AM at the time of initial admission had moderate to severe illness, which was mostly suggestive of respiratory failure (4). In this case series, we present two cases of COVID-19-induced AM with completely differ-

ent clinical presentations. In the first case, the patient developed COVID-19-induced AM after testing negative for COVID-19 in the RT-PCR test. Almost all the published cases were diagnosed within the first week after the initial symptoms of COVID-19 (4). Hence, we think that our patients might be among the first ones to develop AM after the completion of treatment for COVID-19 pneumonia. In addition, this finding supports the hypothesis that prolonged and ongoing systemic inflammation can lead to intense myocardial inflammation that result in COVID-19-induced AM. We found that the ECG of the first patient was also suggestive of ACS due to the symmetrical T-wave negativity in the anterolateral precordial leads, despite the normal epicardial coronary arteries.

In the second case, the clinical presentation is clinically important because the patient was diagnosed initially as having ST-elevation myocardial infarction (STEMI) and later as having COVID-19-induced AM (5, 6). Inciardi et al. (7) recently reported a case of COVID-19-induced AM with dyspnea, elevated cardiac troponin levels, and LV segmental wall abnormalities. They noted a clinical improvement in the patient in the first week after the initiation of inotropic support therapy. Our second case had a similar clinical course in terms of cardiac function improvement with inotropic support. However, the distinguishing feature in our case was that the presenting complaint was chest pain and ST elevation was observed on ECG.

Currently, no specific treatment has been established for COVID-19-induced AM, as the data available so far are limited (8, 9). The treatment should be based on whether LV systolic dysfunction was present or not and the identification of the etiology of AM (8, 9). Hydroxychloroquine and antiviral (favipiravir) therapies should be administered to patients with COVID-19-induced AM. Moreover, because both arterial and venous thromboses are common in COVID-19 cases, anticoagulation should be given as in our cases. The current literature reports that patients diagnosed as having acute fulminant myocarditis and cytokine syndrome, most likely resulting from SARS-CoV-2 infection, can be treated with tocilizumab, intravenous immunoglobulin, and high-dose corticosteroids even though results regarding this issue are conflicting (4, 8, 9). Lastly, patients with acute fulminant myocarditis due to COVID-19 who are resistant to medical therapy can be treated with extracorporeal membrane oxygenation support (10).

CONCLUSION

In conclusion, we think that clinicians should be aware that some patients with COVID-19-induced AM can present with ECG findings mimicking ACS, including STEMI.

Informed Consent: Written informed consent was obtained from patients who participated in this study.

Peer-review: Externally peer-reviewed.

Author Contributions: Concept – TÇ, VÇ, ŞK; Design – TÇ, MS, MK; Supervision – NK, ALO; Resource – VÇ, ŞK, SA; Materials – TÇ, VÇ, MS; Data Collection and/or Processing – VÇ, ŞK, SA; Analysis and/or Interpretation – TÇ, MK; Literature Search – TÇ, VÇ; Writing – TÇ; Critical Reviews – NK, ALO.

Conflict of Interest: The authors have no conflict of interest to declare.

Financial Disclosure: The authors declared that this study has received no financial support.

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