Journal of Cardiology Cases 24 (2021) 177–181

Contents lists available at ScienceDirect

Journal of Cardiology Cases

journal homepage: www.elsevier.com/locate/jccase

Case Report

Systolic dysfunction and complete heart block as complications of fulminant myocarditis in a recovered COVID-19 patient

Mohammad Hossein Nikoo (MD)[a,d], Reza Mozaffari (MD)[d], Mohammad Reza Hatamnejad (MD, MPH)[c], Mehdi Bazrafshan (MD, MPH)[c], Mohammad Kasaei (MD)[b], Hamed Bazrafshan (MD)[b,d,*]

[a] Associate Professor of Cardiology, Shiraz University of medical science, Non-communicable disease research center, Iran
[b] Associate Professor of Cardiology, Department of Cardiology, Shiraz University of Medical Sciences, Shiraz, Iran
[c] Faculty of Medicine, Shiraz University of Medical Sciences, Shiraz, Iran
[d] Al-Zahra Charity Hospital, Department of Cardiology Medicine, Shiraz University of Medical Sciences, Shiraz, Iran

ARTICLE INFO

Article history:
Received 1 February 2021
Revised 26 March 2021
Accepted 31 March 2021

Keywords:
Different degrees of atrioventricular node block
Reduced ejection fraction
Tachyarrhythmia
Post COVID-19 infection

ABSTRACT

We describe the first case report of fulminant myocarditis and complete heart block which was initially presented by severe systolic dysfunction and tachyarrhythmia, in a patient who recently recovered from covid-19. Continuous close follow-up should be considered for patients infected with COVID-19 after discharge, especially for those with any metabolic and pharmacologic risk factors for the conductive block to recognize these rare complications and reverse CHB early by administering a high dose of corticosteroid or other anti-inflammatory medications.

<**Learning objective:** To illustrate the presentation of COVID-19 fulminant myocarditis by severe systolic dysfunction, tachyarrhythmia, and different degrees of atrioventricular block. To introduce the proper management of fulminant myocarditis in a patient who recently recovered from covid-19.>

Introduction

Myocardial infarction (MI), myocarditis, stroke, tachyarrhythmias, and pulmonary embolism have been reported as complications of coronavirus disease-2019 (COVID-19). However, clinical characteristics and outcomes of bradyarrhythmias in this pandemic is less investigated [1]. Here, we describe a complete heart block secondary to fulminant myocarditis in a patient who has recovered from COVID-2019.

Case presentation

On October 12th, 2020, a non-smoker 38-year-old, Iranian woman, with a previous history of mild COVID-19 infection about 2 months ago as the only underlying disease, referred to our hospital with complaints of malaise, atypical chest pain, nausea, and vomiting for three days before admission. The patient denied taking any medication. On admission, her vital signs showed a blood pressure of 90/60 mmHg, tachycardia with a heart rate of 120 beats per minute (BPM), respiratory rate of 22 breaths per minute, oxygen saturation of 92%, and temperature of 37.1 C. Physical examination revealed engorged jugular veins, bibasal crackles. Heart examination showed S3sound (ventricular gallop) and murmur (systolic, grade II/VI, at aortic valve post). Tenderness or organomegaly was not detectable in the abdomen examination. Physical exam of the extremities indicated symmetrical thready pulses without edema. The telemetry rhythm strip on admission illustrated repetitive sustained ventricular tachycardia (Fig. 1), whereas archived previous electrocardiograms (ECGs) showed normal sinus rhythm. Intravenous amiodarone loading dose (150 mg) was prescribed. While receiving bolus amiodaron, strip rhythm converted to complete heart block (CHB) which was accompanied by episodes of non-sustained ventricular tachycardia (Fig. 1); thus, intravenous amiodarone was discontinued. Transthoracic echocardiography (TTE) showed biventricular dilation and global hypokinesia with left ventricular ejection fraction (EF) of 20–25%. MI was the most proba-

* Corresponding author.
E-mail address: hamedbazrafshan@yahoo.com (H. Bazrafshan).

https://doi.org/10.1016/j.jccase.2021.03.009
1878-5409/

ble cause to justify complete heart block and severely decreased EF. Therefore, the patient was brought to the catheterization laboratory. Coronary angiography revealed normal epicardial coronary arteries (Supplemental Figure 1), and also temporary pacemaker (TPM) was implanted simultaneously. Fulminant myocarditis was supposed as the main differential diagnosis after MI exclusion. Cardiac magnetic resonance (CMR) imaging and cardiac biopsy were not applicable according to unstable patient conditions. Laboratory analysis strengthened our hypothesis for multiorgan failure including myocarditis (higher than the normal range for troponin I, creatine kinase-MB, liver function tests, blood urea nitrogen, creatinine, and elevated inflammatory markers with C-reactive protein (see Table 1 for results, reference range)). Immunoglobulin G, immunoglobulin M, and polymerase chain reaction (PCR) against potential infectious causes of myocarditis including Cytomegalovirus, Epstein-Barr virus, Human immunodeficiency virus, Herpes simplex virus I/II, Parvovirus, Coxsackie, Adenovirus, and Enterovirus were not detected. A previous history of covid-19 (although there was no evidence of involvement in a real-time PCR, and high-resolution computed tomography at that time (Supplemental Figure 2)), in addition to the absence of other common causes for myocarditis, suggested that chronic residual inflammation after respiratory recovery of COVID-19 might cause fulminant myocarditis resulting in complete heart block and severe systolic dysfunction. Immunomodulatory therapy (high dose intravenous dexamethasone 8 mg 3 times daily) and other standard heart failure therapies were prescribed. Therapeutic anticoagulation was initiated with respect to the evidence of hypercoagulability and concern for related thrombotic complications. The patient responded well to the high-dose dexamethasone therapy with a rapid decrease in her laboratory markers and rhythm recovery from CHB to the first degree atrioventricular block (AVB) with left bundle branch block (LBBB), and normal sinus rhythm without block, respectively, within 4 days (Fig. 1). TPM was removed on the 5th day of hospitalization and the patient was discharged after stabilizing on the 7th day of hospitalization. After discharging the patient, CMR imaging was performed with a result of normal ventricles size, EF of 52%, and 47.4% for left and right ventricles, respectively, and evidence

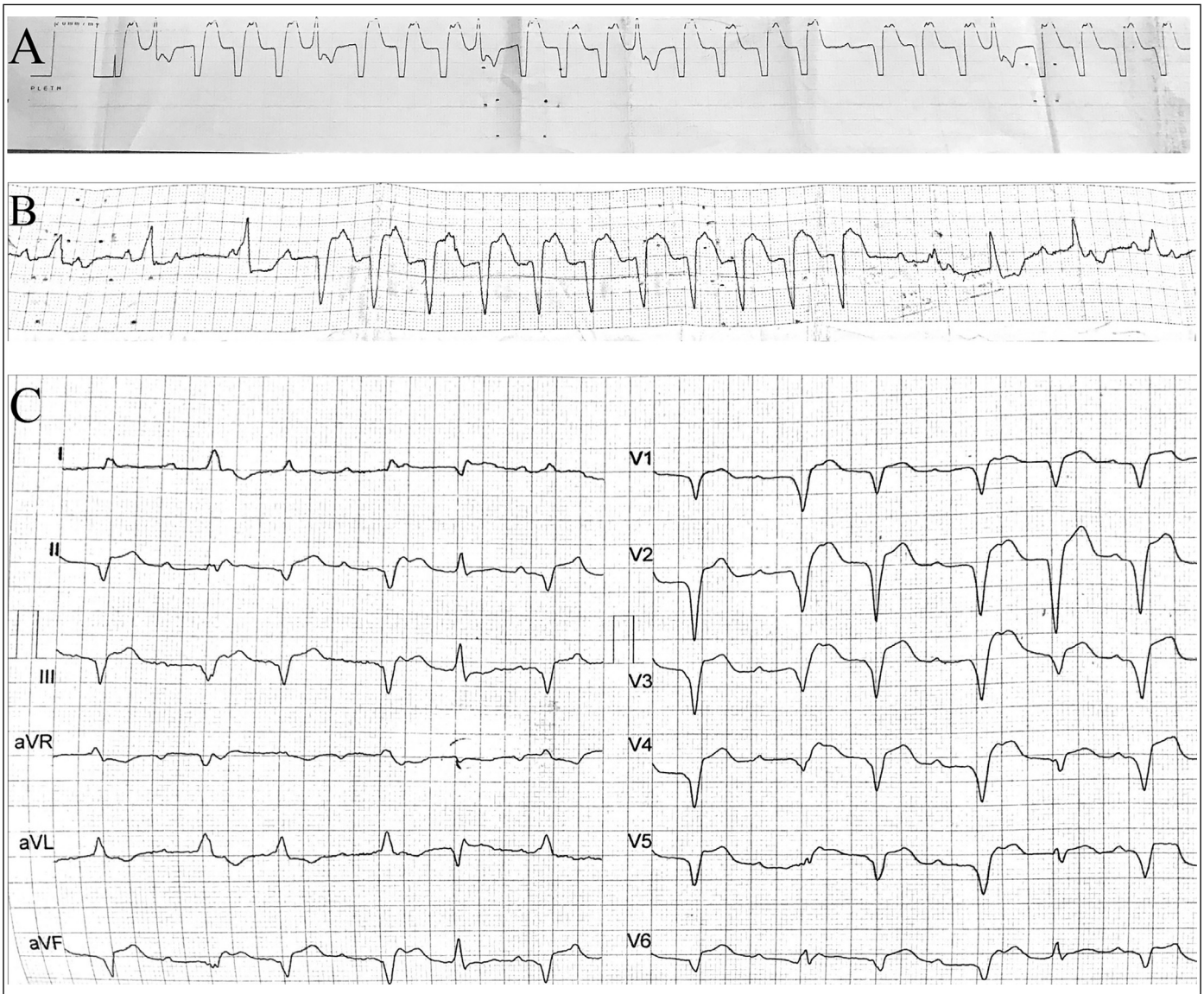

Fig. 1. Rhythm series of the patient (A) Repetitive sustained ventricular tachycardia (B) Complete heart block accompanied by non-sustained ventricular tachycardia (C) Complete heart block with two different escape rhythm and one of the escape focus could be explained as parasystole mechanism (D) The first degree AVB with LBBB (E) The first normal sinus rhythm after CHB.

of diffuse myocardial inflammation of the left ventricular myocardium with no regional wall motion abnormality (Supplemental Figure 3). In the follow-up visit to the outpatient clinic about 2 months later, normal sinus rhythm on ECG and normal echocardiography findings were observed. The university ethics committee approved the study protocol. The patient gave written informed consent.

Discussion

Despite pulmonary manifestations, evidence about cardiac involvement has been growing. In a study from Wuhan, cardiac involvement was concluded by increased troponin levels in about 7% of patients and was associated with worse outcomes [2]. Despite our previous knowledge about myocarditis caused by the viral infection [3], subsequent mechanisms were hypothesized to describe myocarditis caused by COVID-19. Direct viral attachment to

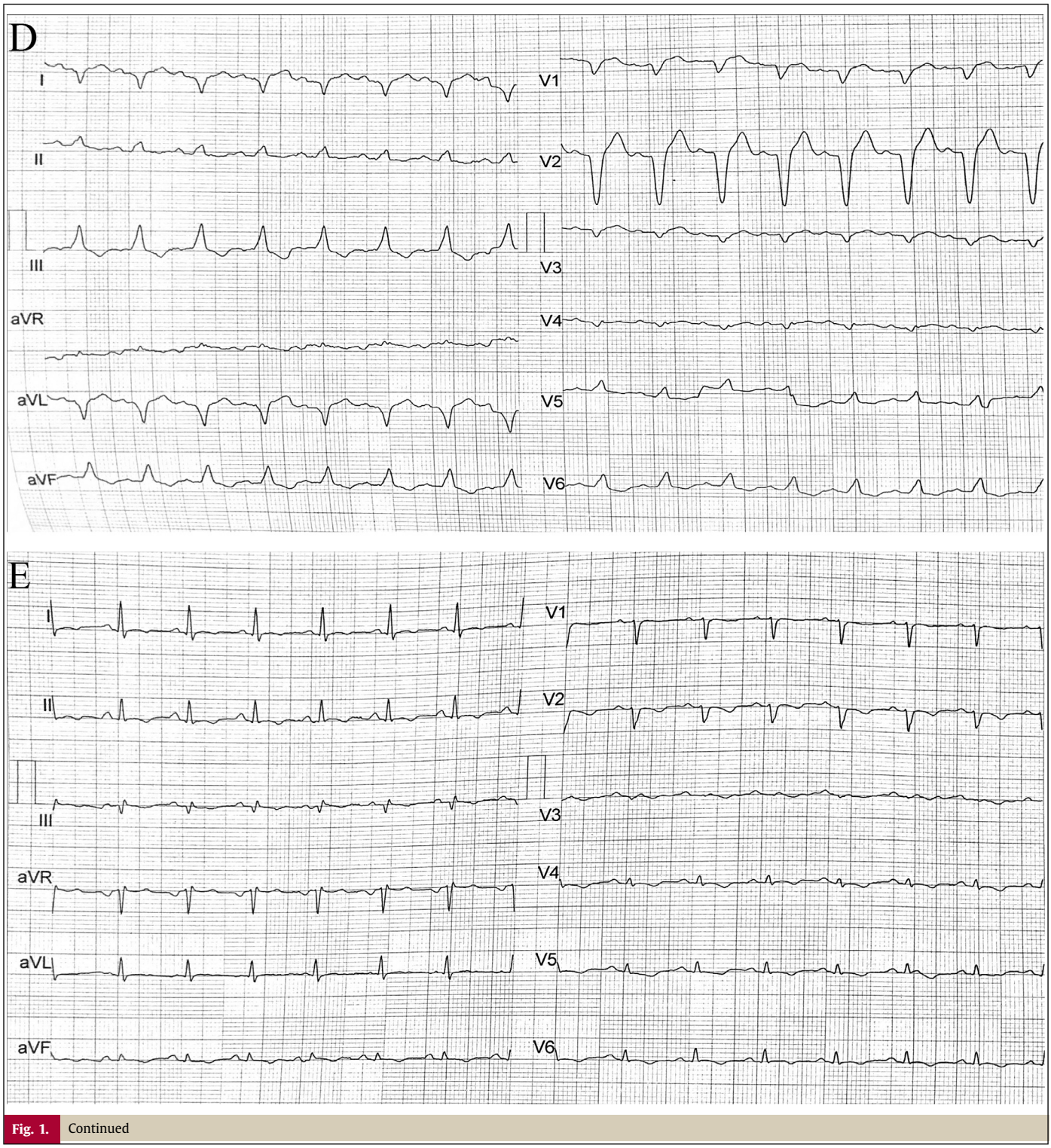

Fig. 1. Continued

Table 1
Laboratory analysis.

Laboratory parameters	Result	Normal range
Troponin I (Mic gr/L)	10.32 *	Less than 0.9
White cell blood count (10^3/μL)	7.4	4–10
Hemoglobin (g/dL)	13.2	12–16 for women
Platelet count (10^3/μL)	164	150–450
Prothrombin time (sec)	17.2 *	12–14
Partial thrombin time (sec)	33	25–40
INR (Index)	1.63 *	0.9–1
C-reactive protein (mg/L)	23 *	Less than 6
ESR (mm/hr)	4	0–29 for women
Fasting blood sugar (mg/dL)	196 *	Less than 99
Sodium (mEq/dL)	131 **	136–145
Potassium (mEq/dL)	4.2	3.5–5.5
Calcium (mg/dl)	8.2 **	8.6–10.3
Blood urea nitrogen (mg/dL)	35 *	8–20
Creatinine (mg/dL)	1.1 *	0.6–1 for women
SGOT (mg/dL)	96 *	0–40
SGPT (IU/L)	77 *	1–40
Alkaline phosphatase (mg/dL)	119	80–306
Albumin (mg/dL)	4.4	3.5–5.4
Total bilirubin (mg/dL)	0.72	0.1–1.2
Direct bilirubin (mg/dL)	0.27	0.1–0.3
Amylase (U/L)	23	Less than 100
Lipase (IU/L)	21	Up to 60
Triglyceride (mg/dL)	134	50–150
Cholesterol (mg/dL)	182	Less than 200
HDL–C (mg/dL)	58	30–80
LDL-C (mg/dL)	93	Less than 150
Uric acid (mg/dL)	5.9	3.6–8.2
CK-MB (IU/L)	83 *	Less than 24

All samples were collected on admission.
* shows higher than the reference interval.
** shows lower than the reference interval.
INR, international normalized ratio; ESR, erythrocyte sedimentation rate; SGOT, serum glutamic-oxaloacetic transaminase; SGPT, serum glutamic-pyruvic transaminase; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; CK-MB, creatine kinase-MB.

Abbreviations list

MI	myocardial infarction
COVID-19	coronavirus disease-2019
BPM	beats per minute
ECG	electrocardiogram
CHB	complete heart block
TTE	transthoracic echocardiography
EF	ejection fraction
TPM	temporary pacemaker
CMR	cardiac magnetic resonance
PCR	polymerase chain reaction
ACE2	angiotensin-converting enzyme 2
AVB	atrioventricular block
LBBB	left bundle branch block

angiotensin-converting enzyme 2 (ACE2) receptor for internalization leads to down-regulation of ACE 2 on cardiac pericytes; thus, angiotensin 2 converting will be decreased and the level of angiotensin 2 will rise, which is assumed as proinflammatory substrate. Cytokine storm in addition to direct endothelial invasion causes myocardial involvement and myocarditis [4]. Conductive tissue as a part of the myocardium will be involved either. Different degrees of atrioventricular node block including CHB are obtained from inflammation and edema in conductive tissue. Decreasing oxygen supply as a result of respiratory involvement, and hypercoagulable states come from plaque destabilization, which are the other suggested mechanisms [5]. Elevated inflammatory markers and treatment of atrioventricular block with anti-inflammatory therapy indicate that inflammation can be assumed as the main cause. However, the exact mechanism of CHB is not well established. In addition to the active phase of COVID-19, myocarditis can be derived in the recovery phase. Huang et al. [6] found cardiac involvement in a proportion of 58% of patients who have recovered from COVID-19. Puntmann et al. [7]. investigated a cohort of German patients who recently recovered from COVID-19 infection, in their study, CMR revealed cardiac involvement in 78 patients (78%), and ongoing myocardial inflammation in 60 patients (60%). There are few case reports of patients with COVID-19 who developed CHB [8–10]; nevertheless, our case has different novel aspects. This is the first report of fulminant myocarditis and CHB which was initially presented by severe systolic dysfunction and tachyarrhythmia, in a patient who had recovered from covid-19. Also, most of them had an underlying inflammatory disease that was reactivated by COVID-19 and triggered the cytokine storm [8–10]; however, our subject did not have any underlying comorbidity.

Conclusion

Although myocarditis is common among the recovered patients, CHB and fulminant myocarditis are the rare manifestations of post-covid-19 complications. Continuous close follow-up should be considered for patients infected with COVID-19 after discharge, especially for those with any metabolic and pharmacologic risk factors for the conductive block; we need to recognize these rare complications and reverse CHB early by administering a high dose of corticosteroid or other anti-inflammatory medications. However, further studies are recommended to be conducted for better management of fulminant myocarditis in a patient who has recovered from COVID-19.

Conflict of interest

The authors confirm that there are no known conflicts of interest associated with this publication and there has been no significant financial support for this work that could have influenced its outcome.

Acknowledgement

The authors would like to thank the participant and her families, Shiraz University of Medical Sciences, Shiraz, Iran, and also Center for Development of Clinical Research of Nemazee Hospital, Dr. Nasrin Shokrpour for editorial assistance.

Supplementary materials

Supplementary material associated with this article can be found, in the online version, at doi:10.1016/j.jccase.2021.03.009.

References

[1] Fried JA, Ramasubbu K, Bhatt R, Topkara VK, Clerkin KJ, Horn E, et al. The Variety of Cardiovascular Presentations of COVID-19. Circulation 2020;141(23):1930–6.
[2] Wang D, Hu B, Hu C, Zhu F, Liu X, Zhang J, et al. Clinical characteristics of 138 hospitalized patients with 2019 novel coronavirus–infected pneumonia in Wuhan, China. JAMA 2020;323(11):1061–9.
[3] Fung G., Luo H., Qiu Y., Yang D., McManus B. Myocarditis. Circ Res . 2016;118(3):496–514.
[4] Liu PP, Blet A, Smyth D, Li H. The science underlying COVID-19: implications for the cardiovascular system. Circulation 2020;142(1):68–78.
[5] Babapoor-Farrokhran S, Gill D, Walker J, Rasekhi RT, Bozorgnia B, Amanullah A. Myocardial injury and COVID-19: possible mechanisms. Life Sci 2020;253:117723.
[6] Huang L, Zhao P, Tang D, Zhu T, Han R, Zhan C, et al. Cardiac involvement in patients recovered from COVID-2019 identified using magnetic resonance imaging. JACC Cardiovasc Imaging 2020;13(11):2330–9.

[7] Puntmann VO, Carerj ML, Wieters I, Fahim M, Arendt C, Hoffmann J, et al. Outcomes of cardiovascular magnetic resonance imaging in patients recently recovered from coronavirus disease 2019 (COVID-19). JAMA Cardiol 2020;5(11):1265–73.
[8] Haddadin FI, Mahdawi TE, Hattar L, Beydoun H, Fram F, Homoud M. A case of complete heart block in a COVID-19 infected patient. J Cardiol Cases 2021;23(1):27–30.
[9] Gupta MD, Qamar A, Mp G, Safal S, Batra V, Basia D, et al. Bradyarrhythmias in patients with COVID-19: a case series. Indian Pacing Electrophysiol J 2020;20(5):211–12.
[10] El-Assaad I, Hood-Pishchany MI, Kheir J, Mistry K, Dixit A, Halyabar O, et al. Complete heart block, severe ventricular dysfunction, and myocardial inflammation in a child with COVID-19 infection. JACC Case Rep 2020;2(9):1351–5.