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Arrhythmia in a COVID-19 patient: A case report

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ABSTRACT

In this study, we considered an 83-year-old male patient admitted to the Al-Zahra hospital emergency department in Isfahan. He complained of fatigue, weakness, headache, and cough. In addition, he had hallucinations and delusions for two days; but he had no fever and chill. His physical examination showed a blood pressure of 170/100 mm Hg, heart rate of 142 beats per minute (bpm), respiratory rate of 23 pbm, oxygen saturation (in room air) of 83%, and oxygen bag reserve mask of 93%. We realized cardiac involvement during hospitalization, including sinus bradycardia, first-degree atrioventricular (AV) block, recurrent premature ventricular from tricuspid ring, atrial tachycardia (AT) rhythm with variable AV conduction block, increased heart rate with functional bundle branch block, and negative troponin. The patient was treated with medicines to control heart rate and admitted to the cardiac care unit (CCU). Next, the patient was intubated due to a worsening lung condition. Afterward, he was admitted to the intensive care unit (ICU) and died the next day. According to the literature, compromised cardiac vascular is the most common complications in hospitalized patients due to COVID-19 infection and has a higher mortality risk. Cardiac arrhythmias are additionally common clinical manifestations. These arrhythmias seem to be caused by inflammatory responses in the myocardium, electrolyte disorders, and hypoxia. Our patient showed that the COVID-19 virus might induce different types of arrhythmias.

Case Report

Implication for health policy/practice/research/medical education:
COVID-19 infection can disturb the conduction of the cardiac system and increase the risk of various arrhythmias such as atrial tachycardia (AT), various atrioventricular (AV) block, premature ventricular contraction (PVC), premature atrial complex (PAC).
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Introduction

The coronavirus was spread worldwide and influenced the lives of people all over the world during the current outbreak of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) in December 2019 (1). The most significant manifestations of COVID-19 are caused because the virus influences the respiratory system. Moreover, well-documented cardiac complications of COVID-19 are observed in patients with and without previous cardiovascular disease. Cardiac complications include myocarditis, heart failure, and acute coronary syndrome caused by coronary artery thrombosis or plaque rupture related to SARS-CoV-2 (2). Research has shown that arrhythmias are also a complication of this pandemic (3). One study revealed that 16.7% of hospitalized patients and 44.4% of intensive care unit (ICU) patients with COVID-19 suffer from cardiac arrhythmias (4). The inflammatory effects of COVID-19 on the heart are conduction disturbances, myocarditis, pericarditis, and endocarditis. Babapoor-Farrokhran et al showed that arrhythmias previously detected with viral infections are caused by viral myocarditis. In addition, the available evidence suggests that these conditions may happen in patients infected with COVID-19 (3). A retrospective case study in New York City reported that patients had more atrial arrhythmias than ventricular arrhythmias (5). Current data reveal that arrhythmias are more prevalent in intensive care settings (6). The results achieved by previous studies explain these findings and suggest that SARS-CoV-2 infection and secondary heart damage may increase the risk of arrhythmias. The present study describes a man with a COVID-19 infection who

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experienced various arrhythmias during hospitalization.

Case Presentation

In this study, we consider an 83-year-old male with diabetes who has been admitted to the emergency department of Al-Zahra hospital in Isfahan. He complained of fatigue, weakness, headache, and cough. In addition, he had hallucinations and delusions for two days, but no fever and chill. His physical examination showed a blood pressure of 170/100 mm Hg, heart rate of 142 beats per minute (bpm), respiratory rate of 23 pbm, oxygen saturation (in room air) of 83%, and oxygen bag reserve mask of 93%. Blood test of white blood cell showed 8000 per cubic millimeter, lymphocyte count of 5.7%, C-reactive protein of 101 mg/L, TSH = 0.2 mIU/L, K = 5.4 mmol /L, Na = 148 mEq / L, Mg = 2.1 mg/dL, Ca = 7.5 mg / L and Cr 2.8 mg/dL.

The patient was hospitalized due to SARS-CoV-2 infection symptoms. He was treated with clindamycin 600 mg/q8h/intravascular, dexamethasone intravenous (IV) injection 8 mg/ once a day/IV, levothyroxine 100 micrograms/once a day/oral (previous thyroidectomy), famotidine 40 mg/once a day/oral, atorvastatin 20 mg/once a day/oral, heparin sodium 5000 IU/ml three time a day subcutaneously, prazosin 1 mg/8 h, and neutral protamine Hagedorn insulin. The diagnosis was confirmed by chest computed tomography (CT) and polymerase chain reaction test (PCR) for COVID-19 (Figure 1).

The electrocardiogram (ECG) (Figure 2) showed normal sinus rhythm (NSR) with an extended PR interval. Additionally, in the afternoon, he had an atrial tachycardia (AT) rhythm with variable atrioventricular (AV) block (Figure 3). His blood pressure was 130/80 mm Hg. The patient was admitted to the CCU, and metoprolol tartrate 25 mg q12h was administered after cardiac counseling. On echocardiography, the left ventricular function was normal, and there was no pericardial effusion or heart valve disease. Afterward, the right ventricular function was maintained.

Frequent PVC from tricuspid ring with compensatory phase were seen in second day of hospitalization (Figure 4). on the third day of hospitalization, upon hospitalization, 1 mg of intravenous atropine was administered due to sinus bradycardia. The result was the early coupling of premature atrial contractions (PACs) (Figure 5). The, metoprolol was stopped. The patient was intubated due to worsening lung conditions and admitted to the ICU. In the ICU, the ECG indicated an AT rhythm with variable AV conduction block (Figure 6). The next day; ECG showed normal sinus rythm with frequent early coupling PACs with functional bundle branch block with compensatory phase (Figure 7). Unfortunately, the patient died due to worsening his lung condition. The last rhythm was asystole.

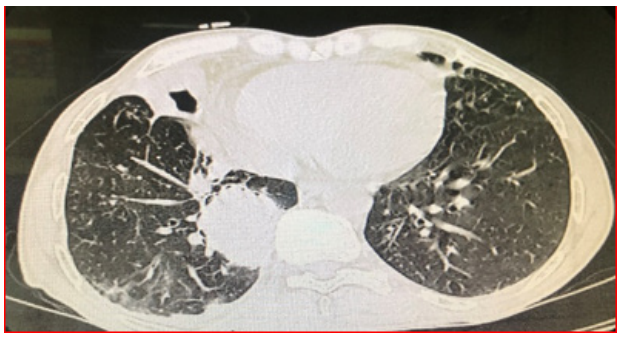

Figure 1. Chest computed tomography (CT).

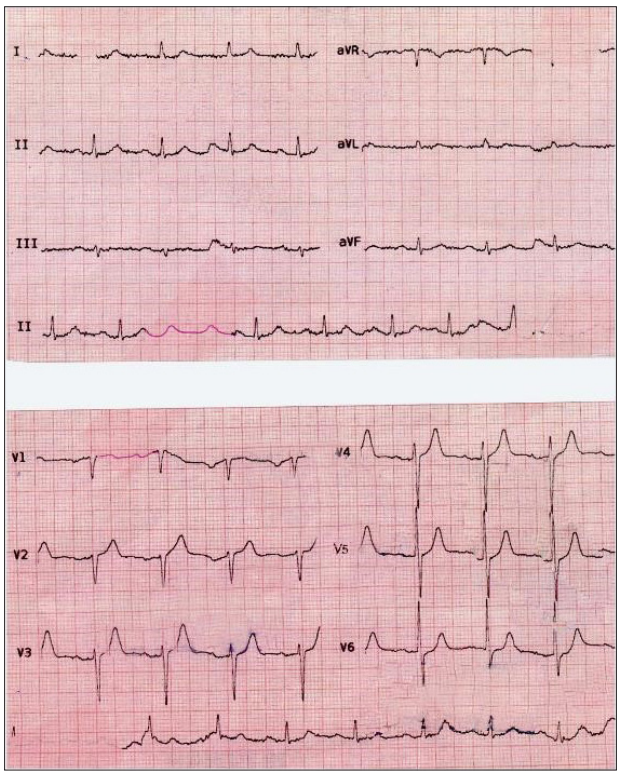

Figure 2. The electrocardiogram (ECG) on the first day of hospitalization.

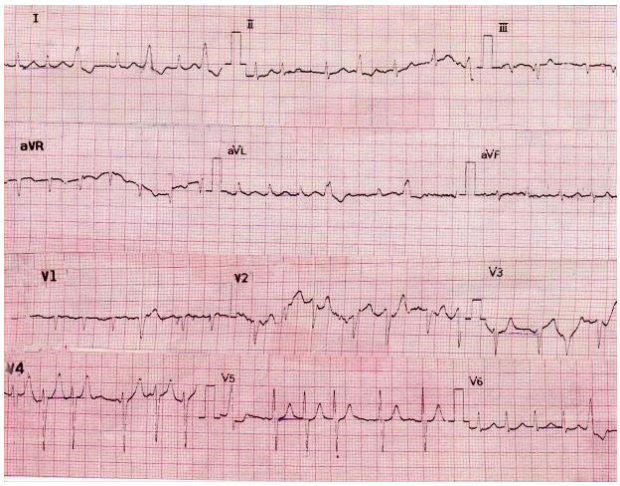

Figure 3. Atrial tachycardia (AT) rhythm with variable atrioventricular (AV) block.

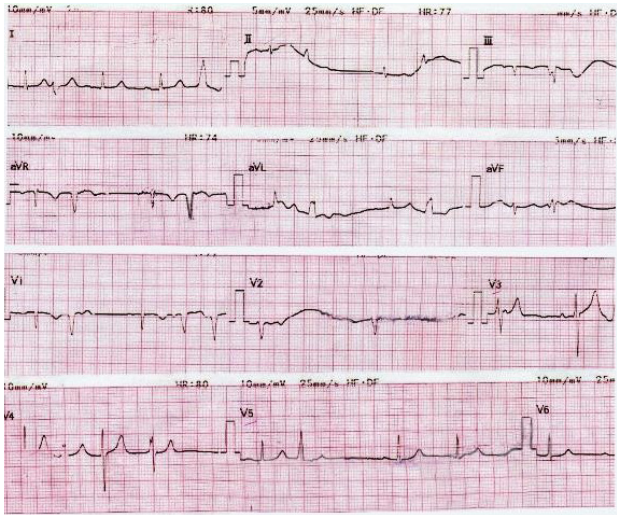

Figure 4. Frequent PVC from tricuspid ring with compensatory phase.

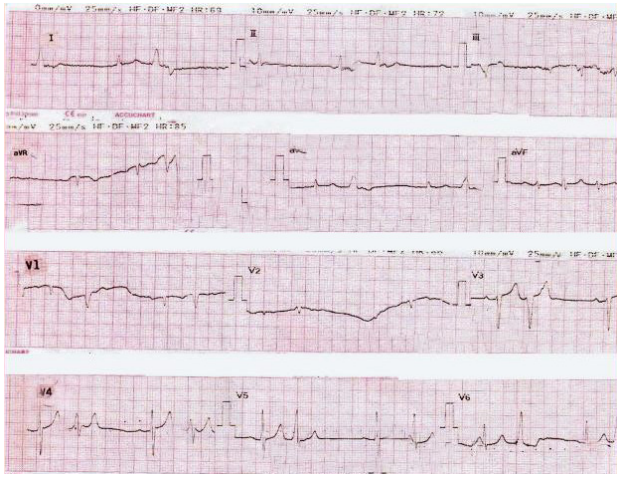

Figure 5. Early coupling of premature atrial contractions (PACs).

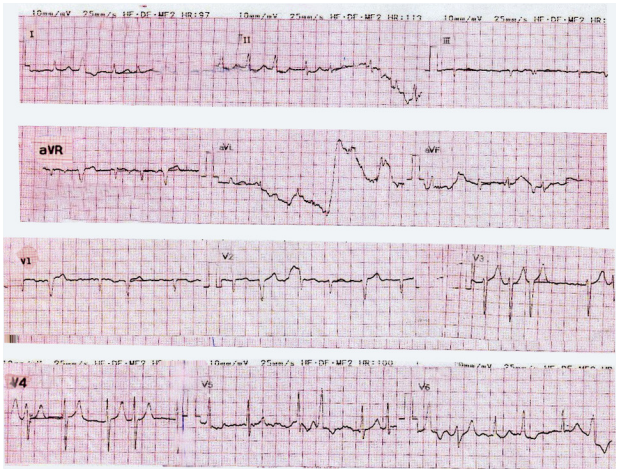

Figure 6. The electrocardiogram (ECG) indicated an atrial tachycardia (AT) rhythm with variable AV conduction block.

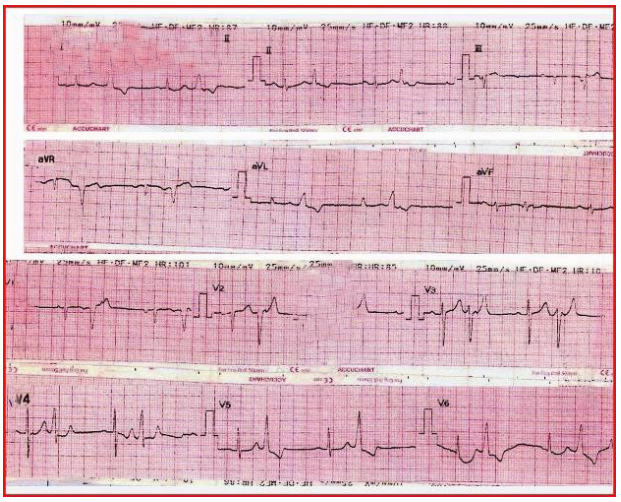

Figure 7. Normal sinus rhythm (NSR) with bigeminal early coupling PACs.

Discussion

Currently, cardiovascular compromise is a common complication in hospitalized patients with COVID-19 infection, leading to higher mortality risk (7). In this respect, 16% of cases experience arrhythmia (8). In a recent series comprising nearly 148 patients, almost 10% reported palpitations (9). In a recent study of similar size, arrhythmias were reported in approximately one-sixth of patients and often occurred in the ICU subgroup, nearly half of which were influenced (4). Despite these studies, the features of these arrhythmias have not yet been published or described. The development of potentially deadly arrhythmias, especially in increased cardiac biomarkers, must serve as a differential diagnosis (10). Arrhythmias in COVID-19 patients are complex and multifactorial and may be caused by metabolic disorders such as hypoxia, acidosis, vascular volume imbalance, neuro-hormonal, drug side effects, and catecholamines stress (11). Sepsis is characterized by a systemic milieu involving inflammatory cytokines and autonomic dysfunction (12).

Additionally, severe infection lowers the sympathetic nervous system (SNS). In addition, there is a relationship between SNS activity and supraventricular tachyarrhythmia (13). In this regard, tachycardia is an independent prognosticator of mortality in patients with sepsis (14). The mechanisms suggested for this arrhythmogenesis include SNS-induced calcium entry into cardiac myocytes as well as a spontaneous release of calcium from the sarcoplasmic reticulum (15).

Our patient is of scientific value because, despite negative troponin, standard electrolyte and normal left ventricular function had different types of arrhythmia. These findings show that cardiac damage in COVID-19 can occur in the conduction system without myocardial inflammation and injury.

Conclusion

COVID-19 infection can disturb the conduction system without myocardial inflammation and injury.

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Authors' contribution

Conceptualization: MM.
Methodology: AA.
Validation: FT.
Formal analysis: ET.
Investigation: MD.
Resources: MP.
Data curation: MS.
Writing–original draft preparation: MM.
Writing–review and editing: FT.
Visualization: FT.
Supervision: AA, MM.

Conflicts of interest

The authors declare that they have no competing interests.

Ethical issues

This case report was conducted according to the World Medical Association Declaration of Helsinki. The patient gave consent to publish as a case report. Besides, the authors have entirely observed ethical issues (including plagiarism, data fabrication, and double publication).

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