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Case Report
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TRANSIENT ATRIOVENTRICULAR COMPLETE BLOCK RARELY DETECTED IN A PATIENT WITH COVID-19: A CASE REPORT

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Abstract: Patients infected with the COVID-19 virus may be symptomatic or asymptomatic. Most symptomatic patients present to the hospital with fever, cough, shortness of breath, runny nose, muscle and diarthrosis pain, diarrhea, and loss of taste. In addition, myocardial infarction (MI), myocarditis, stroke, tachyarrhythmias, and pulmonary embolism have also been reported. Although it is rare, bradyarrhythmia can also be seen in this patient group. Cardiopulmonary pathologies were excluded with transthoracic echocardiography, laboratory analyses, thorax computed tomography and cardiac magnetic resonance imaging. In this study, we aimed to present a patient infected with COVID-19, who had no cardiac or pulmonary involvement but had a temporary atrioventricular (AV) complete block.

Keywords: COVID-19, Bradyarrhythmia, High-degree atrioventricular block, SARS-CoV-2, Atrioventricular complete block

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1. Introduction

The virus, which spread all over the world in a short time from Wuhan, China in the second half of 2019, was reported as a disease that usually affects the respiratory system. The most common symptoms include cough, fever, and tiredness, shortness of breath, sore throat, and headache. Although respiratory insufficiency is the primary complication seen in COVID-19, patients are at high risk for multi-system involvement, including acute cardiac injury (Vetter et al., 2020).

There are now well-documented cardiac complications of COVID-19 infection, including myocarditis, heart failure, and acute coronary syndrome (Wang et al., 2020). Although tachycardia is more common, bradycardia is observed, and atrioventricular (AV) complete block may rarely develop (Chinitz et al., 2020).

In this study, we aimed to present a patient who has infected with COVID-19 infection, and who developed a temporary AV-complete block without cardiac and pulmonary involvement and without a history of arrhythmia.

2. Case Report

A 42-year-old male patient, by cautiously keeping his distance, went to the market wearing a protective mask and fainted while shopping, and applied to the emergency department. In his vital signs, oxygen saturation was measured as 98% (on room air), temperature 37.4 °C, and pulse 42 per minute, respiratory rate 20 per minute, blood pressure 126/84 mmHg. The complete block was observed in his electrocardiogram (ECG) (Figure 1).

In the anamnesis, he said that he did not have a history of arrhythmia, did not use any herbal medicines, did not have regular tobacco and alcohol use, did not consume honey, or even applied a special disease-preventing diet at home. In the physical examination of the patient, who had no previous illnesses and did not use medication, no remarkable positive findings were found except hyperemia of the oropharynx. No pathological results were found in the laboratory tests, except for a moderate D-dimer elevation (Table 1).

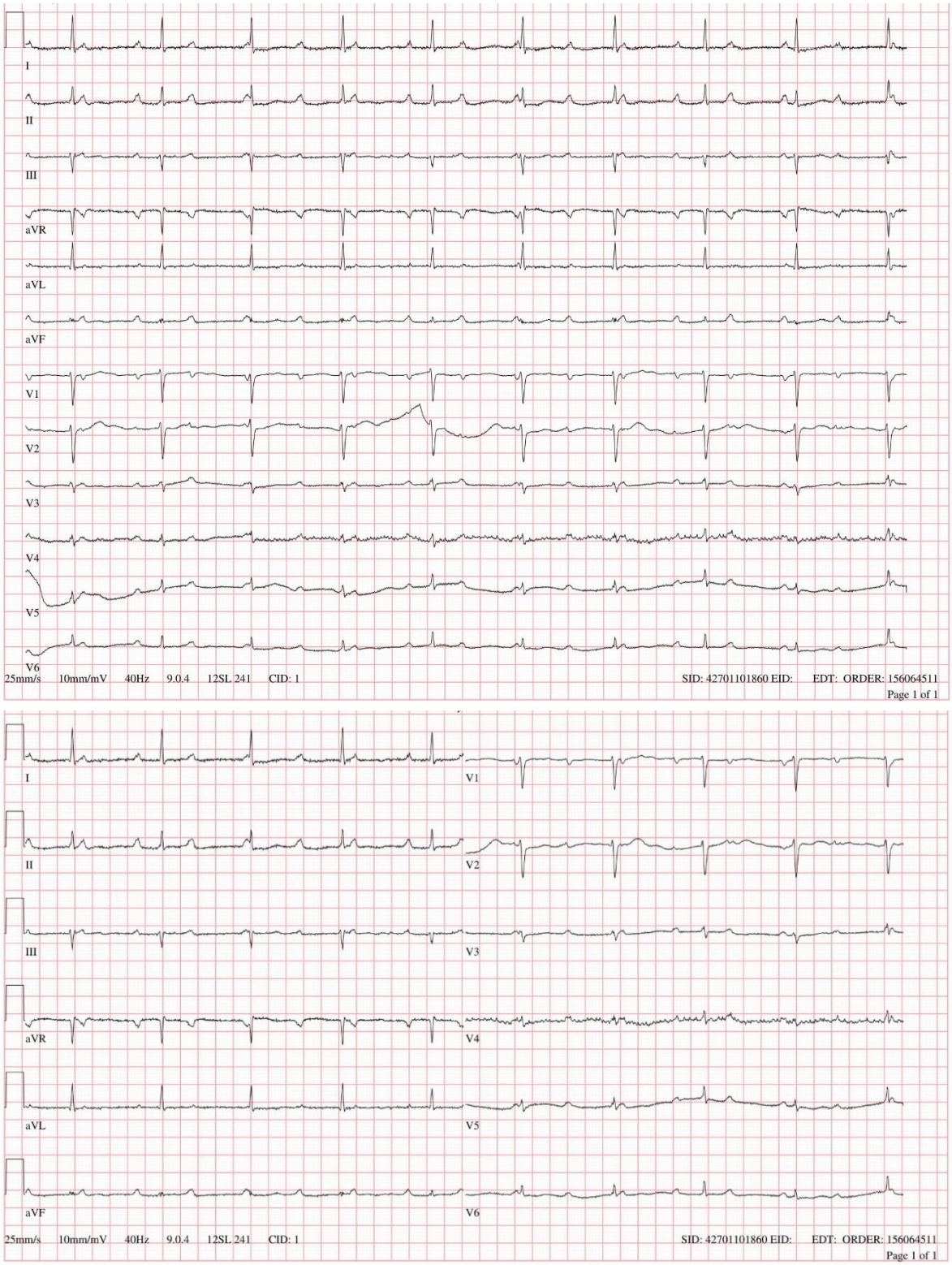


Figure 1. The first electrocardiogram with full block at heart rate beats per minute. Ventilation rate: 58 BPM, QRS duration: 72 ms, QT/QTc: 414/406 ms.

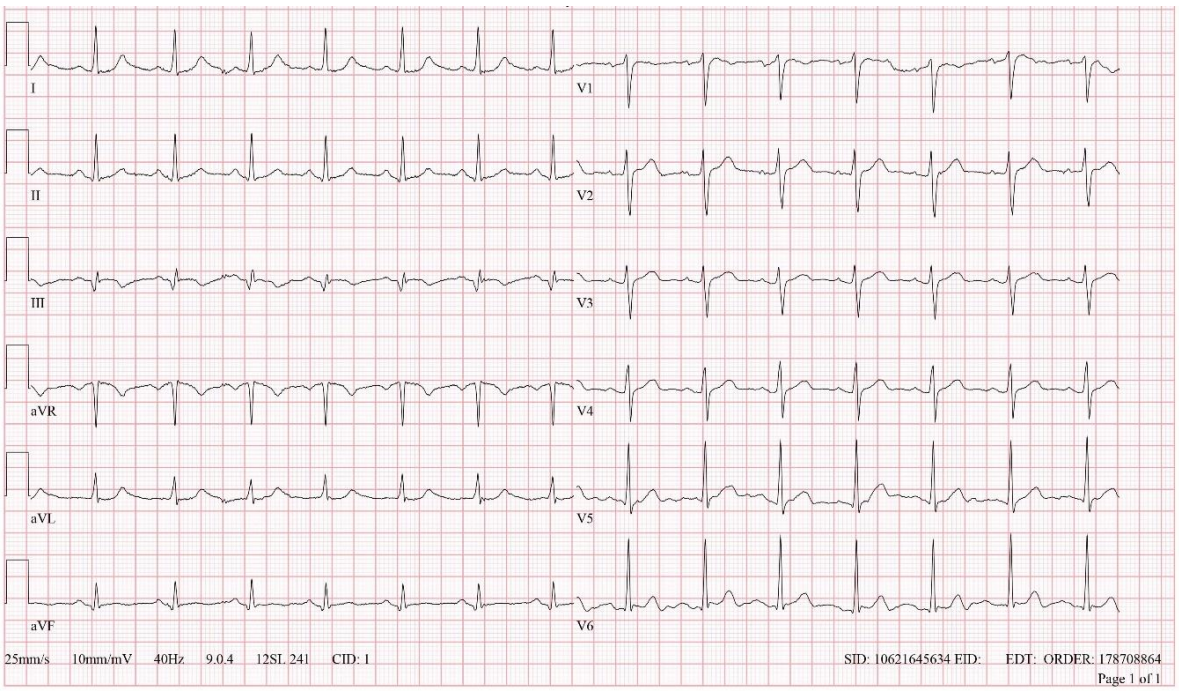
Table 1. Laboratory findings of the patient

Parameters	Results	Reference ranges
Glucose (mg/dL)	101	74-106
Renal Functional Tests		
BUN (mg/dL)	22	8-20
Creatinine (mg/dL)	1.02	0.81-1.44
Na (mmol/L)	137	136-146
K (mmol/L)	4.4	3.5-5.1
Liver Metabolism		
ALT (U/L)	40	0-35
AST (U/L)	52	10-50
Total bilirubin (mg/dL)	0.39	0.3-1.2
Direct bilirubin (mg/dL)	0.05	0-0.2
CRP (mg/L)	10.8	0-5
Total Blood Counts		
WBC ($10^3/mm^3$)	8.9	4-10.5
HBG (g/dL)	14.2	13.7-17
HTC (%)	46.1	42-52
PLT ($10^3/mm^3$)	248	150-450
NEU ($10^3/mm^3$)	5.4	1.82-7.42
LYM ($10^3/mm^3$)	2.4	0.85-3
MON ($10^3/mm^3$)	0.5	0.19-0.77
Troponin T (ng\L)	4	0-14
Procalcitonin (ng/ml)	0.15	0-2
NT-Pro B-Type Natriuretic Peptide (pq/mL)	75	<900
Fibrinogen (mg/dL)	252	200-500
Iron (µg/L)	168	60-180
Ferritin (µg/L)	70	11-306
TIBC (µg/L)	302	250-450
Thyroid Function Tests		
Free T3	2.76	2.5-3.9
Free T4	0.99	0.61-1.12
TSH	1.47	0.34-5.86
Calcium (mg/dL)	9.2	8.8-10.6
Blood Gas Parameters		
pH	7.39	7.35-7.45
$PaCO_2$ (mm Hg)	40	35-45
PaO_2 (mm Hg)	90	80-100
HCO_3 (mmol/L)	24	22-26
Lactate (mmol/L)	0.8	0.5-2

BUN= blood urea nitrogen, ALT= alanine aminotransferase, AST= aspartate aminotransferase, CRP= C reactive protein, WBC= white blood cells, HBG= hemoglobin, HTC= hematocrit, PLT= platelets, LYM= lymphocyte, NEU= neutrophil, MON= monocyte, aPTT= activated partial prothrombin time, PT= prothrombin time, INR= international normalized ratio, TIBC= total iron binding capacity, ABG= arterial blood gases, TSH= thyroid stimulating hormone

In his digital rectal examination, the colic stool was observed, and gastrointestinal bleeding was eliminated. In his serial ECGs, AV complete block continued, and control cardiac biomarkers remained negative. In addition, no inotropes and pacemakers were planned, as the patient did not show an unstable clinical picture during the follow-up. No pathological finding was detected in the thorax computed tomography. Cranial BT and diffusion MRI results were found to be normal, and intracranial pathologies were eliminated. Systolic and diastolic functions, pulmonary artery diameter, and right chambers of the heart were found to be normal in echocardiography, and massive and sub-massive pulmonary embolism was excluded. Pericardial effusion was not detected. When COVID-19 was detected in the nasopharyngeal PCR test, he was hospitalized and monitored in a room reserved for COVID-19 patients. Except for a mild subfebrile fever and a moderated d-dimer elevation, troponin and pro-BNP values were observed to be normal during his hospitalization. Pulmonary artery diameter and pericardial echogenicity were normal in the control echocardiography, and no effusion was observed. Cardiac magnetic resonance result was normal. On his ECG, the patient, whose treatment was completed with favipiravir, saline, and enoxaparin, was observed to be in sinus rhythm

approximately 22 hours after the first application (Figure 2). When the nasopharyngeal PCR result obtained from the patient, who remained stable during his hospitalization, was found to be negative, the patient was discharged. In the ECG taken one month later, it was observed that the sinus rhythm maintained. During this period, he did not have an active complaint.



Figüre 2. Normal ECG. Ventilation rate: 86 BPM, PR interval: 162 ms, QRS duration: 100 ms, QT/QTc: 352/421 ms

3. Results and Discussion

While many COVID-19 patients survive the infection with mild symptoms without the need for hospitalization, some may show multisystemic involvement in addition to heart diseases. It may cause myocardial injury, myocarditis, pericarditis, and sometimes arrhythmias (Zhou et al., 2020).

COVID-19 disease with cardiac involvement is considered to be a high mortality risk. Troponin value, which increases especially with d-dimer and pro-BNP, is an important indicator of the deterioration in the patients' conditions (Liu et al., 2020).

Except for the moderate height of d-dimer in the patient, there was no high pathological laboratory finding. The absence of pathological findings in echocardiography and lab values may explain the stable course of the patient's clinic. In our patient, the development of isolated AV complete block without pulmonary and cardiac involvement may have a specific relation with COVID-19.

Viruses holding the heart usually settle in the myocardium and can exacerbate conduction system disorders and develop AV block. In our case, pro-BNP and troponin and trans echocardiography were found to be normal, and this suggests that the virus might follow a different path. The virus usually affects heart cells in different ways but causes arrhythmia rather by affecting calcium channels (Y. Wang et al., 2020).

Transient AV complete block development may have been caused by SARS-CoV-2 virus affecting calcium channels. One of the possible mechanisms of complete heart block in COVID-19 patients involves a high systemic inflammatory load and cytokine storm that causes ischemic damage to myocardial cells (Huang et al., 2020). Another possible mechanism involves a direct viral injury to the myocardium and the manifestation of the conduction system itself as a temporary complete heart block (Madjid et al., 2020). In fact, ACE2, the host receptor for SARS-CoV-2, is widely expressed not only in the lungs but also in the cardiovascular system, and therefore, signaling pathways associated with ACE2 could explain the immediate detrimental effect of the novel coronavirus on myocardial disease. Regardless of the overall inflammatory load in the tissue, viral myocarditis can also disrupt the cardiac conduction system, causing local inflammation (Madjid et al., 2020). Nevertheless, there was no sign of myocarditis in the patient. In addition, cytokine storm and high inflammatory load did not develop. Transient AV block development without evidence of systemic infection may be due to the more specific atrioventricular node effect of COVID-19. What's more, COVID-19 patients have a higher risk of developing acute kidney failure (Ronco and Reis, 2020), it can cause major electrolyte disturbances and hyperkaliemia, promoting AV block. Renal failure was not documented in the patient.

In the case of hypoxemia, it can promote cell death of cardiomyocytes and induce arrhythmia by disrupting the function of ion channels, leading to prolongation of cardiac action potential and/or changes in repolarization (Garrott et al., 2017). Since our patient did not develop

hypoxia, this may not be regarded as a possible cause for the development of arrhythmia.

Since our patient reported that he did not have a history of arrhythmia, did not use any herbal medicines, did not have regular tobacco and alcohol use, did not consume honey, or even applied a special disease-preventing diet at home, an external cause resulting from nutrition or lifestyle was not considered to cause complete AV block.

In the related literature, there are some cases reporting the development of complete AV block in the COVID-19 infected patients without any significant cardiac comorbidity or underlying conduction disease (Kir et al., 2020). In line with the findings of these case reports, although our patient had no known history of cardiac disease or arrhythmia, a complete AV block developed.

A medical approach with atropine or isoprenaline is recommended according to ESS in patients with unstable Av complete block. If the AV block does not improve after the patient has recovered, the pacemaker should be implanted.

In our patient, a temporary av block developed, which is rarely seen in patients infected with COVID-19. Pacemaker implantation was not planned as the related findings of the patient were constantly stable during follow-up.

In conclusion, more research is needed to better characterize the prevalence, type, and predictive factors of arrhythmias in patients infected with COVID-19 and to provide guidance for close cardiac monitoring during their hospital stay and after their discharge.

4. Conclusion

Although tachycardia is reported to be more common in patients infected with COVID-19, bradycardia may also be seen, though encountered in a rare ratio. Although they do not have a history of cardiac pathology and cardiopulmonary involvement and do not use av nodal blocking agents, patients infected with COVID-19 may develop temporary AV complete block. If there is no decompensation during the illness and the patients have a stable course, it is necessary to wait for the spontaneous recovery of the AV complete block instead of being aggressive and invasive.

Author Contributions

All authors had equal contribution and reviewed and approved the manuscript.

Conflict of Interest

The author declared that there is no conflict of interest.

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Ethical Approval/Informed Consent

All procedures in this study were in accordance with the ethical standards as laid down in the 1964 Declaration of Helsinki and its later amendments. The patient gave her informed consent to the anonymous publication of data for scientific purposes. This study has been notified to the local Ethics Committee.

References

Chinitz JS, Goyal R, Harding M, Veseli G, Gruberg L, Jadonath R, Maccaro P, Gandotra P, Ong L, Epstein LM. 2020. Bradyarrhythmias in patients with COVID-19: Marker of poor prognosis? Pacing Clin Electrophysiol, 43(10): 1199–1204.

Garrott K, Kuzmiak-Glancy S, Wengrowski A, Zhang H, Rogers J, Kay MW. 2017. K (ATP) channel inhibition blunts electromechanical decline during hypoxia in left ventricular working rabbit hearts. J Physiol, 595(12): 3799–3813. https://doi.org/10.1113/JP273873.

Huang C, Wang Y, Li X, Ren L, Zhao J, Hu Y, Zhang L, Fan G, Xu J, Gu X, Cheng Z, Yu T, Xia J, Wei Y, Wu W, Xie X, Yin W, Li H, Liu M, Cao B. 2020. Clinical features of patients infected with 2019 novel coronavirus in Wuhan, China. Lancet, 395(10223): 497–506. https://doi.org/10.1016/S0140-6736(20)30183-5.

Kir D, Mohan C, Sancassani R. 2020. Heart break: An unusual cardiac manifestation of COVID-19. JACC, 2(9): 1252–1255. https://doi.org/10.1016/j.jaccas.2020.04.026.

Liu PP, Blet A, Smyth D, Li H. 2020. The Science Underlying COVID-19: Implications for the Cardiovascular System. Circulation, 142(1): 68–78. https://doi.org/10.1161/CIRCULATIONAHA.120.047549.

Madjid M, Safavi-Naeini P, Solomon SD, Vardeny O. 2020. Potential Effects of Coronaviruses on the Cardiovascular System: A Review. JAMA Cardiol, 5(7): 831–840. https://doi.org/10.1001/jamacardio.2020.1286.

Ronco C, Reis T. 2020. Kidney involvement in COVID-19 and rationale for extracorporeal therapies. Nature Reviews. Nephrol, 16(6): 308–310. https://doi.org/10.1038/s41581-020-0284-7.

Vetter P, Vu DL, L'Huillier AG, Schibler M, Kaiser L, Jacquerioz F. 2020. Clinical features of covid-19. BMJ, 369: 1470. https://doi.org/10.1136/bmj.m1470.

Wang D, Hu B, Hu C, Zhu F, Liu X, Zhang J, Wang B, Xiang H, Cheng Z, Xiong Y, Zhao Y, Li Y, Wang X, Peng Z. 2020. Clinical characteristics of 138 hospitalized patients with 2019 novel coronavirus-infected pneumonia in Wuhan, China. JAMA, 323(11): 1061–1069. https://doi.org/10.1001/jama.2020.1585.

Wang Y, Wang Z, Tse G, Zhang L, Wan EY, Guo Y, Lip GYH, Li G, Lu Z, Liu T. 2020. Cardiac arrhythmias in patients with COVID-19. J Arrhythmia, 36(5): 827–836. https://doi.org/10.1002/joa3.12405.

Zhou P, Yang XL, Wang XG, Hu B, Zhang L, Zhang W, Si HR, Zhu Y, Li B, Huang CL, Chen HD, Chen J, Luo Y, Guo H, Jiang RD, Liu MQ, Chen Y, Shen XR, Wang X, Shi ZL. 2020. A pneumonia outbreak associated with a new coronavirus of probable bat origin. Nature, 579(7798): 270–273. https://doi.org/10.1038/s41586-020-2012-7.