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Case Report

Characteristic Electrocardiographic Manifestations in Patients With COVID-19

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ABSTRACT

Cardiac involvement has been reported in patients with COVID-19, which may be reflected by electrocardiographic (ECG) changes. Two COVID-19 cases in our report exhibited different ECG manifestations as the disease caused deterioration. The first case presented temporary SIQIIITIII morphology followed by reversible nearly complete atrioventricular block, and the second demonstrated ST-segment elevation accompanied by multifocal ventricular tachycardia. The underlying mechanisms of these ECG abnormalities in the severe stage of COVID-19 may be attributed to hypoxia and inflammatory damage incurred by the virus.

Case 1

A 66-year-old woman with no remarkable medical history was admitted to the hospital with diagnosed COVID-19. The symptom of dyspnea and pulmonary imaging (Supplemental Fig. S1A) developed and progressively worsened in the following 30 days of hospitalization. Finally, trachea intubation and vein-to-vein extracorporeal membrane oxygenation (VV-ECMO) were used to maintain optimal PaO₂. The ECMO was withdrawn 5 days later when the patient became stabilized.

Case 2

A 70-year-old man was admitted to the hospital with a diagnosis of COVID-19. He had a history of hypertension and type 2 diabetes. Despite therapy, lesions in both lungs...
Figure 1. Electrocardiography series of patient 1: (A) Sinus rhythm with first-degree atrioventricular block (AVB); (B) sinus tachycardia, first AVB with SITIIIQIII; (C) Mobitz type 1 second-degree AVB and atrioventricular junctional escape beat; (D) high-grade AVB or nearly complete AVB with junctional escape rhythm; (E) first-degree AVB and recovery of SITIIIQIII.
Figure 2. Electrocardiography series of patient 2: (A) Sinus tachycardia with incomplete right bundle branch block; (B) slightly elevated ST segment; (C) ventricular tachycardia; (D) ventricular tachycardia and ventricular fusion; (E) remarkable ST-segment elevation in the form of triangular QRS-ST-T waveform.
increased (Supplemental Figure S1B) and hypoxemia worsened. On the 14th day of hospitalization, trachea intubation was required to maintain optimal oxygenation. The patient’s first ECG recording showed basic rhythm of sinus tachycardia with an incomplete right bundle branch block (Fig. 2A). On the 34th day of hospitalization, the patient developed severe hypoxia and VV-ECMO was undertaken. However, the patient’s hypoxemia did not significantly improve and severe hypotension ensued. Artery blood gas showed a critically low PaO2 level of 57.3 mm Hg and lactic acidosis (10.8 mmol/L).

The day after VV-ECMO incubation, the patient’s ECG demonstrated ST-segment elevations in the inferior and precordial leads (Fig. 2B) and the amplitude of ST elevation gradually increased to form a triangular QRS-ST-T waveform (Fig. 2E). During the evolution of ST elevation, 2 episodes of multifocal ventricular tachycardia developed (Fig. 2, C and D). Lidocaine was administered and sinus rhythm was restored. Simultaneous blood chemical tests showed positive cardiac troponin I, elevated creatine kinase of 900.9 U/L (normal range 10-190 U/L), and a significant increase of N-terminal pro-B-type natriuretic peptide up to 24,245 pg/mL (normal range < 900 pg/mL). The echocardiogram revealed diffuse hypokinesis, especially in the anterior and inferior walls. The patient died within 24 hours of the occurrence of ventricular tachycardia and ST-segment elevation.

Discussion

It is reported that acute cardiac injury is not uncommon in patients with COVID-19.1,2 The percentage of COVID-19 patients with myocardial injury has been reported variously at 12%3 and 7.2%,2 and to be much higher in critically ill patients. As seen in the present report, abnormal ECG changes were recorded during the critical condition of these 2 cases. There were several possible mechanisms. First, angiotensin-converting enzyme 2 (ACE2) has been identified as a functional receptor for coronaviruses,3 which is highly expressed in the heart and lungs. Therefore, ACE2-related signalling pathways might have played a role in cardiac injury. Second, hypoxemia caused by COVID-19 may cause damage to myocardial cells. Third, systemic inflammatory response and immune system disorders may be important factors.4

The ECG changes may reflect different cardiac injuries with diverse manifestations. In the first patient, the temporary occurrence of SIQIIIITIII and subsequent transient, nearly complete AVB may reflect transient pulmonary artery hypertension secondary to trachea secretory obstruction, which may cause extensive small pulmonary artery compression. Acute pulmonary embolism should be ruled out, although the reversibility of STIIQIIII in a short time made it unlikely. Another potential mechanism that may have induced this reversible complete AVB is local inflammation of the myocardium.3

The development of ST-segment elevation and multifocal ventricular tachycardia in the second patient may have several explanations. The presence of multiple coronary heart disease risk factors and the elevation of myocardial biomarkers made ST-segment-elevation myocardial infarction (MI) the first consideration. But the extensive ST-segment elevation in the inferior leads and V1-V4 could not be explained by a single coronary artery occlusion. The most plausible explanation is type 2 MI secondary to severe hypoxia and hypotension, considering the patient’s critical clinical state. However, the characteristic triangular QRS-ST-T waveform in localized leads (inferior and V1-V4) could not be explained by global hypotension and hypoxia. We therefore speculate that this ECG change might have resulted from acute myocarditis that was induced by SARS-CoV-2 infection.

Conclusion

Dynamic ECG change is the hallmark of cardiac injury, which usually signifies a critical status in patients with COVID-19.

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Disclosures

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Supplementary Material

To access the supplementary material accompanying this article, visit the online version of the Canadian Journal of Cardiology at www.onlinecjc.ca and at https://doi.org/10.1016/j.cjca.2020.03.028.