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Canadian Journal of Cardiology ■ (2020) 1.e1−1.e4 www.onlinecjc.ca

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.

RÉSUMÉ

Des cas d'atteinte cardiaque se manifestant par des changements décelables à l'électrocardiogramme (ECG) ont été rapportés chez certains patients atteints de la COVID-19. Nous exposons les cas de deux patients atteints de la COVID-19 dont les ECG révèlent une détérioration de la fonction cardiaque causée par la maladie. Dans le premier cas, le patient a présenté temporairement un aspect SIQIIITIII suivi d'un bloc auriculo-ventriculaire quasi complet réversible, tandis que dans le deuxième cas, le patient a présenté une élévation du segment ST accompagnée d'une tachycardie ventriculaire multifocale. Ces anomalies électroencéphalographiques chez des patients gravement atteints de la COVID-19 pourraient s'expliquer par l'hypoxie et les lésions inflammatoires provoquées par le 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 PaO_2. The ECMO was withdrawn 5 days later when the patient became stabilized.

The patient's ECG revealed dynamic changes when her clinical state was unstable. The baseline ECG showed sinus rhythm with a first-degree atrioventricular block (AVB) (Fig. 1A). On the day of the trachea intubation, the ECG recording showed sinus tachycardia with SITIIIQIII morphology (Fig. 1B). Simultaneous echocardiography revealed an enlarged right atrium and right ventricle accompanied by severe tricuspid regurgitation, which could result from the elevated pulmonary artery pressure. Mobitz type I second-degree AVB and atrioventricular junctional escape beat were recorded (Fig. 1C) the same day. On the following day, temporary nearly complete AVB (or high-grade AVB) developed (Fig. 1D). Soon after, the ECG showed a recovery to first-degree AVB, and the SITIIIQIII disappeared (Fig 1E).

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

Received for publication March 18, 2020. Accepted March 22, 2020.

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See page 1.e4 for disclosure information.

https://doi.org/10.1016/j.cjca.2020.03.028

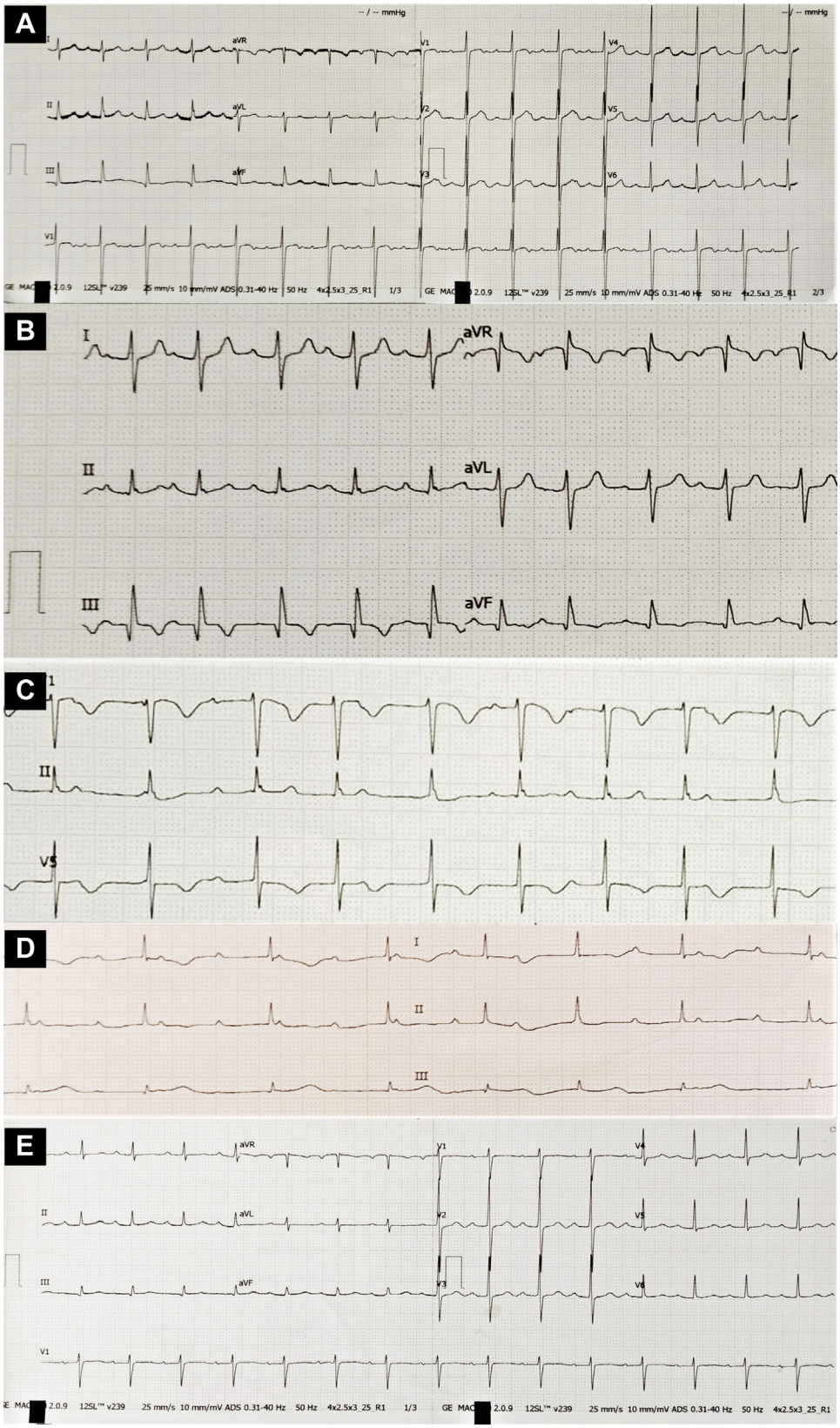

Figure 1. Electrocardiography series of patient 1: (**A**) Sinus rhythm with first-degree atrioventricular block (AVB); (**B**) sinus tachycardia, first AVB with SITIIIQIII; (**C**) Mobitz type 1 second-degree AVB and atrioventricular junctional escape beat; (**D**) high-grade AVB or nearly complete AVB with junctional escape rhythm; (**E**) first-degree AVB and recovery of SITIIIQIII.

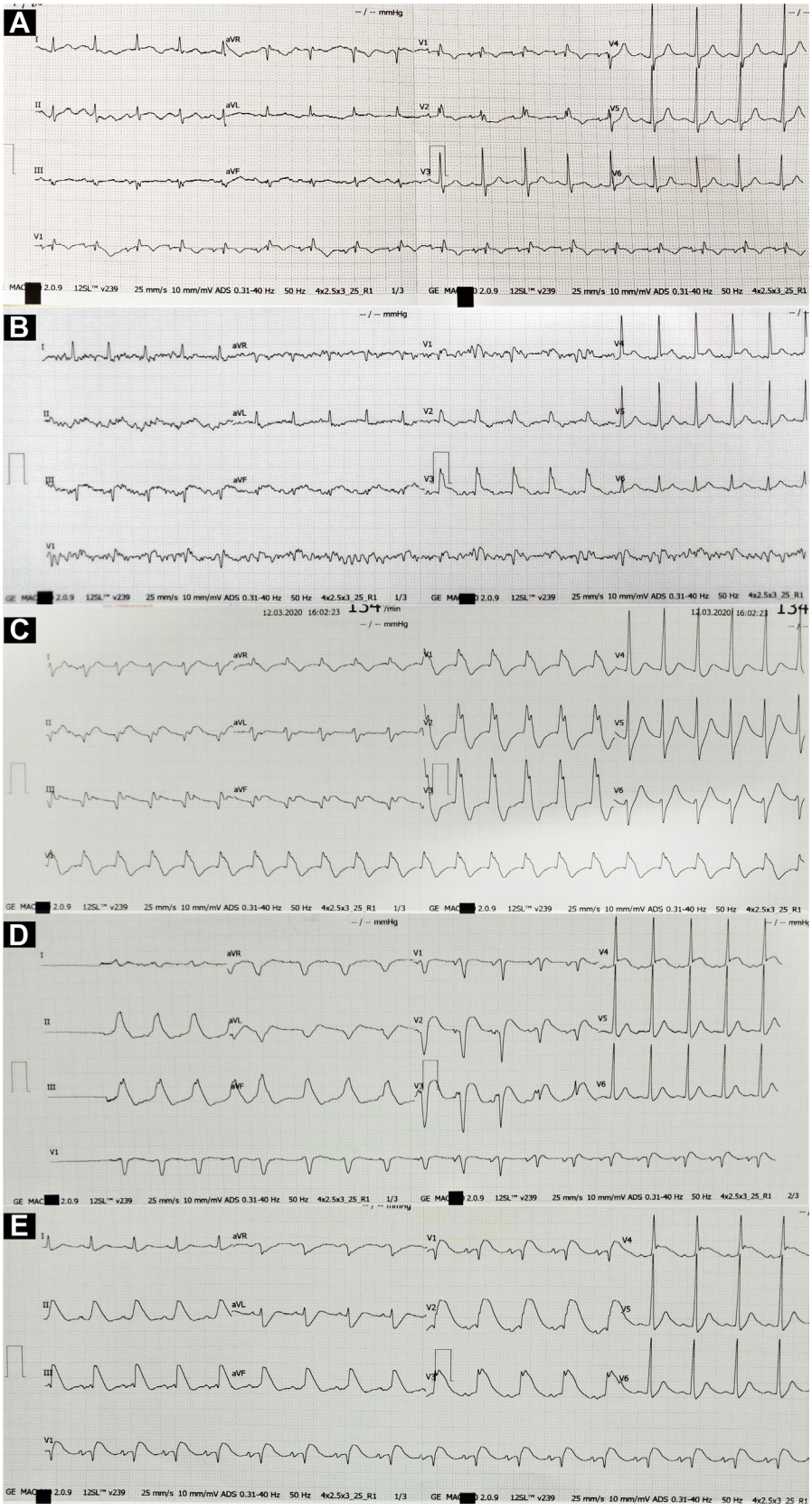

Figure 2. Electrocardiography series of patient 2: (**A**) Sinus tachycardia with incomplete right bundle branch block; (**B**) slightly elevated ST segment; (**C**) ventricular tachycardia; (**D**) ventricular tachycardia and ventricular fusion; (**E**) remarkable ST-segment elevation in the form of 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 oxygenation did not significantly improve and severe hypotension ensued. Artery blood gas showed a critically low PaO_2 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 creatine kinase MB of 72.6 U/L (normal range 0-24 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%[1] 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 S1QIIITIII and subsequent transient, nearly complete AVB may reflect transient pulmonary artery hypertension secondary to trachea secretive obstruction, which may cause extensive small pulmonary artery compression. Acute pulmonary embolism should be ruled out, although the reversibility of SITIIIQIII in a short time made it unlikely. Another potential mechanism that may have induced this reversible complete AVB is local inflammation of the myocardium.[5]

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.

Acknowledgements

The authors are grateful to Dr Shuhui Wang Lorkowski and Dr C.M. Lorkowski for their linguistic assistance during the preparation of this manuscript.

Funding Sources

This study was supported by the Emergency Project of Prevention and Control for COVID-19 of Central South University (502701002).

Disclosures

The authors have no conflicts of interest to disclose.

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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.