COVID and the Heart–Arrhythmia in COVID-19: A Case Report

Nicy F. Narvas, MD | Emma Y. Gaspar-Trinidad, MD, FPCP, FPCC
ManilaMed (Medical Center Manila), Manila

Abstract

BACKGROUND: A 65-year-old man, Filipino, with comorbidities consulted because of shortness of breath. Chest computed tomography scan showed ground-glass opacities and crazy paving patterns (computed tomography severity score = 29). He required oxygen at 4 L/min, with bradycardia. Cardiac enzymes and transthoracic echocardiogram did not show abnormalities. Serial electrocardiogram and electrolytes showed arrhythmia and electrolyte imbalance.

Accumulated evidence showed that cardiac involvement is common in patients with COVID-19.[1,2] Atrial and ventricular arrhythmia is common even if there is no history of arrhythmia, normal cardiac biomarkers, and transthoracic echocardiogram. Different mechanisms have been proposed for the cause of electrolyte imbalance (see Discussion) that may lead to arrhythmia. Both potassium and calcium were used as a marker of clinical severity and worse prognosis in patients with COVID-19.[3,4]

CONCLUSION: Arrhythmia in COVID-19 patients are more frequent in elderly with comorbidities. Close monitoring and correction of electrolyte imbalance are important to prevent arrhythmia, which can sometimes be fatal.

PRESENTATION OF THE CASE

A 65-year-old man, Filipino, was admitted because of shortness of breath, a week's history of fever (T_{max} of 38.9°C), productive cough, and whitish sputum; examined; and was given Azithromycin. He had hypertension and diabetes and was on maintenance medication. He was oriented, in distress (92% at room air), and had normal vital signs and body mass index.

On auscultation of lungs crackles mid to base

WORKUP AND MANAGEMENT

Figure 1 shows the initial diagnostic examinations performed. The 12-L electrocardiogram showed sinus rhythm, normal QT interval and nonspecific T-wave changes. The chest x-ray showed bilateral pneumonia and/or pulmonary congestion. Figure 2 shows (A) the chest computed tomography (multifocal peripherally distributed ground-glass opacities/crazy paving patterns in both lungs (CO-RADS 5) and (B) the CO-RADS level of suspicion (COVID-19 Reporting and Data System).

A repeat reverse transcriptase–polymerase chain reaction was positive for COVID-19. Table 1 shows electrolyte imbalance while Figure 3 shows the electrocardiogram abnormalities associated with these electrolyte imbalances. Cardiac biomarkers were normal. Transthoracic echocardiogram and cardiac magnetic resonance imaging were done to rule out myocarditis (Figure 4).

DISCUSSION

The current outbreak of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2)–COVID-19 has quickly progressed to a global pandemic. The World Health Organization interim guidelines state that the risk factors for severe disease of COVID-19 include age older than 60 years (increasing age), underlying noncommunicable diseases (diabetes, hypertension, cardiac diseases, chronic lung disease, cerebrovascular diseases, chronic kidney disease, immunosuppression, and cancer).[1]

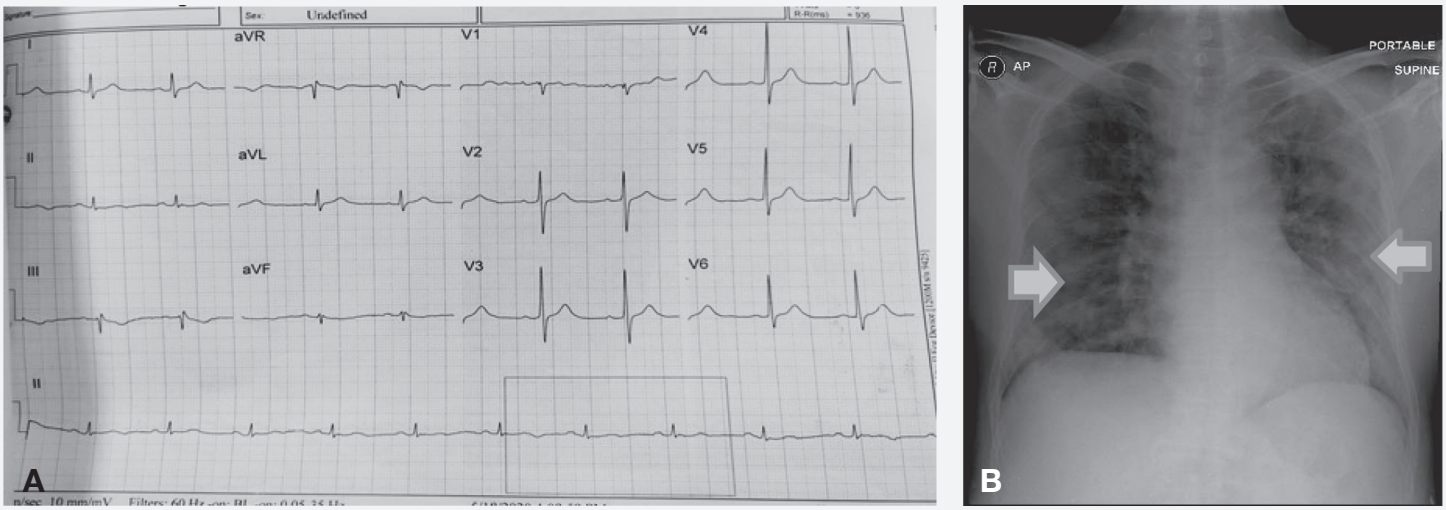
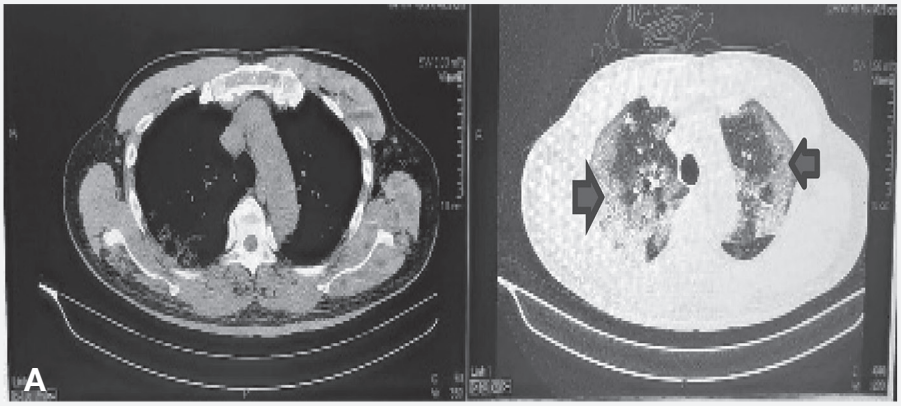


Figure 1. Diagnostic examination: (A) Sinus rhythm, normal QT interval, nonspecific T-wave changes; (B) Bilateral pneumonia and/or pulmonary congestion



CO-RADS*

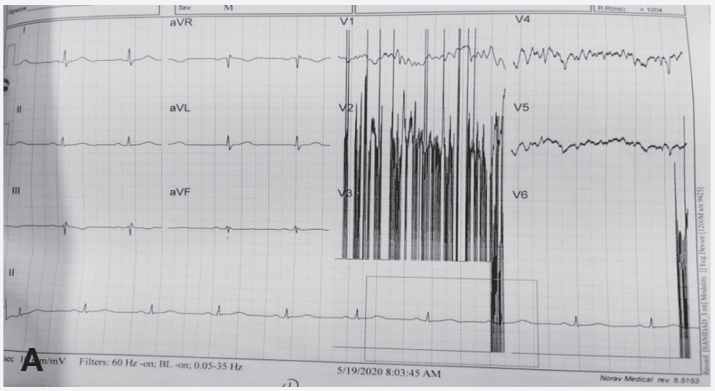
Level of suspicion COVID-19 infection

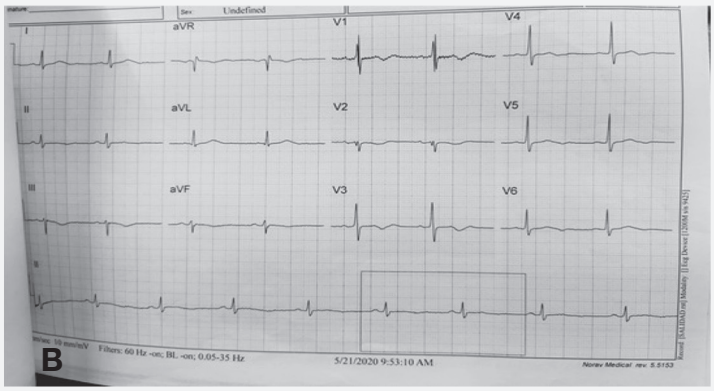
		CT findings
CO-RADS 1	No	normal or non-infectious abnormalities
CO-RADS 2	Low	abnormalities consistent with infections other than COVID-19
CO-RADS 3	Indeterminate	unclear whether COVID-19 is present
CO-RADS 4	High	abnormalities suspicious for COVID-19
CO-RADS 5	Very high	typical COVID-19
CO-RADS 6	PCR +	

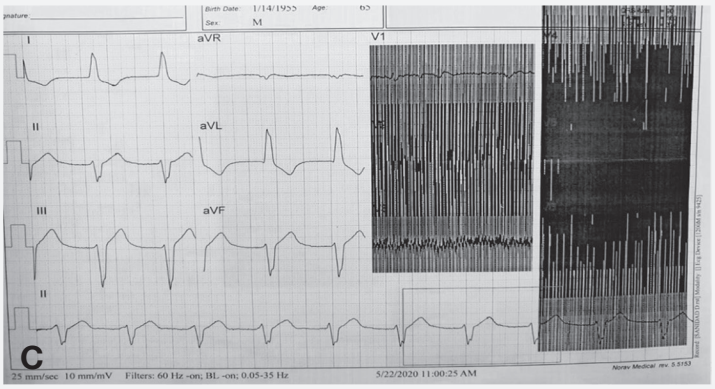
B

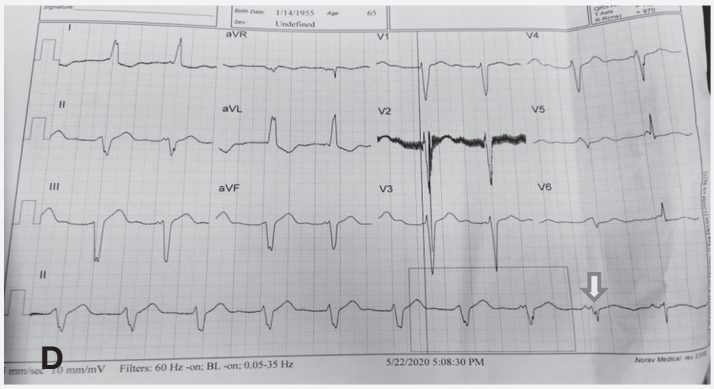
FIGURE 2. A, Chest computed tomography: multifocal peripherally distributed ground-glass opacities/crazy paving patterns in both lungs (CO-RADS 5); B, CO-RADS level of suspicion. CO-RADS, COVID-19 Reporting and Data System

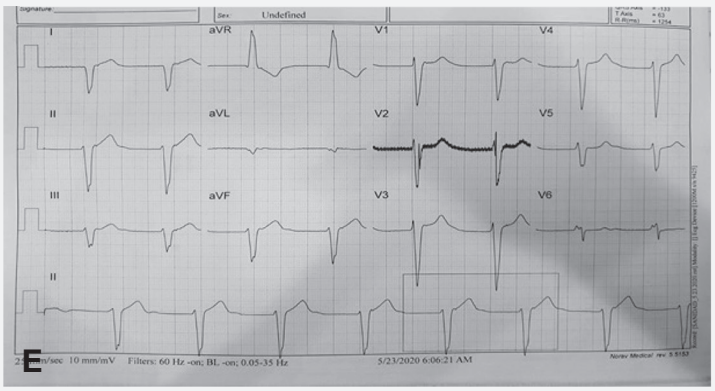
Figure 3. Serial electrocardiogram monitoring: (A) Sinus bradycardia, prolonged QTc, (B) Post tocilizumab and hydroxychloroquine: sinus bradycardia, prolonged QTc; hydroxychloroquine was stopped, (C) Idioventricular rhythm, left axis deviation normal QTc, (D) Slow ventricular tachycardia (arrow, fusion beat), left axis deviation, prolonged QTc, (E) Idioventricular rhythm, extreme axis deviation, prolonged QTc, (F) Sinus bradycardia, normal QTc, (G) Sinus rhythm, normal QTc

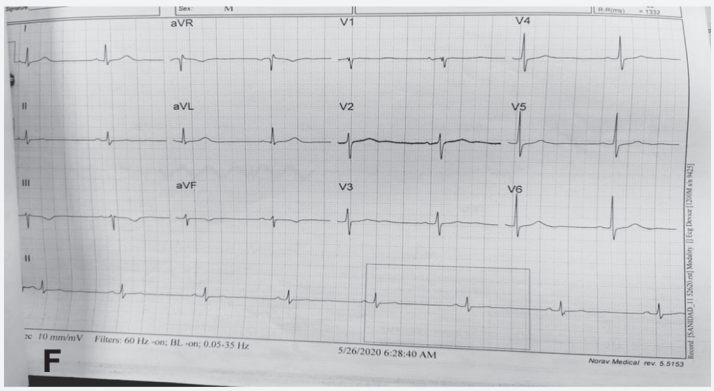


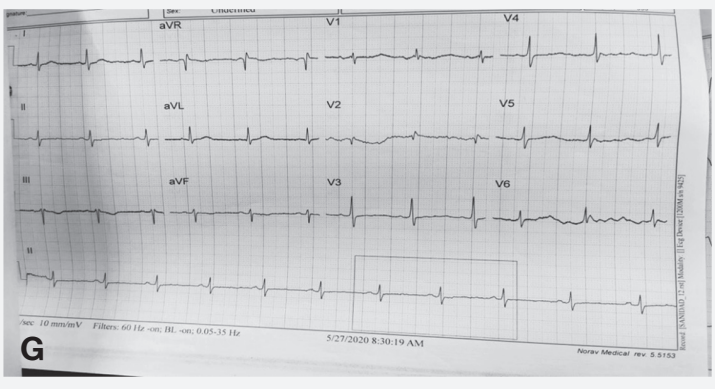












The prevalence of arrhythmias and conduction system disease, and cardiovascular disease, in general, in patients with COVID-19 varies. Hypoxia and electrolyte abnormalities were both known to contribute to the development of acute arrhythmias. These have been frequently reported in the acute phase of severe COVID-19 illness. The proposed mechanisms for arrhythmogenicity in viral infections are through the interplay between host factors and viral characteristics, which include altered intercellular coupling, interstitial edema, and cardiac fibrosis that lead to abnormal conduction in addition to abnormal Ca^{2+} handling and down-regulation of K^{+} channels

Table 1. Monitoring of Serum Electrolytes and Urine Creatinine/Potassium

Electrocardiogram	Serum				Urine			
	Potassium, mmol/L	Ionized Calcium, mmol/L	Magnesium, mmol/L	Chloride, mmol/L	Sodium, mmol/L	Creatinine, mg/dL	Urine Potassium, mmol/L	Fractional Excretion of Potassium, %
Sinus rhythm	4.12				140			
Sinus bradycardia, prolonged QTc (0.47 s)	3.04	1.06	2.01	104.6		18.9	10.7	16.84
Idioventricular rhythm, QTc 0.41 s	3.03	1.19	2.03		138	81.5	37.1	10.25
	4.42							
Sinus bradycardia, normal QTc (0.43 s)	3.11	1.18	2.22			45.8	19.2	6.43
	4.35							
Sinus rhythm, normal QTc (0.40 s)	3.83							

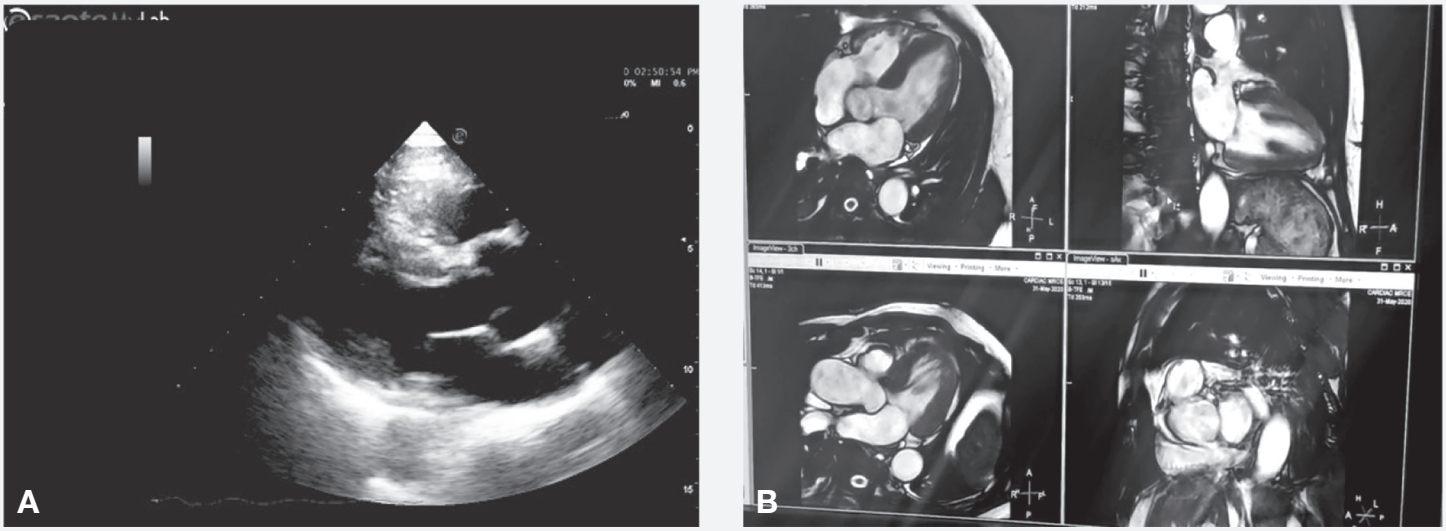


Figure 4. (A) Transthoracic echocardiogram: concentric left ventricular remodeling, normal overall left and right ventricular systolic function, (B) Cardiac magnetic resonance imaging: basal to midseptal left ventricular hypertrophy. Normal biventricular systolic function with no regional wall motion abnormalities. No evidence of myocardial edema, infarction, or fibrosis

that result in repolarization abnormalities and action potential conduction abnormalities.[3]

Electrolyte disturbances were reported in 7.2% of 416 hospitalized patients with COVID-19 infection.[5] These were attributed to COVID-19–associated diarrhea or to renal injury. In renal injury, the balance of fluid and electrolytes can be altered that leads to retention of phosphorus and a reduction in the levels of calcium in the blood. Hypokalemia may contribute to myocardial dysfunction, ventricular arrhythmia, and respiratory muscle dysfunction. Other mechanism of arrhythmia in COVID-19 is a viral myocarditis that can cause cardiac conduction system disease, resulting in sinoatrial node and atrioventricular node dysfunction. One of the most common arrhythmias in relation to COVID-19 is sinus bradycardia.[3] In some reports, bradycardia and intermittent high-degree atrioventricular block were seen in a patient with COVID-19 infection who had normal echocardiography and cardiac biomarkers.[5] Other reports found that in hospitalized patients, 7% had ventricular tachyarrhythmias during hospitalization. Malignant arrhythmias, including ventricular tachycardia/ ventricular fibrillation, were more common in patients with

elevated troponin T levels compared with patients with normal troponin T levels (11.5% vs 5.2%).[5] There are also reports of ventricular arrhythmias and torsade de pointes due to QT-prolonging medications, especially azithromycin and hydroxychloroquine.[5]

Hypocalcemia is a common laboratory abnormality in viral infection and pneumonia.[4] Cappellini et al[6] described that in viral infections Ca^{2+} is essential for virus structure formation, entry, gene expression, virion maturation, and release.[6] Most patients in the study by Liu et al[4] were elderly, with poor nutritional status, which showed chronic malnutrition will lead to vitamin D deficiency that results in hypocalcemia. In addition, hypoxia of tissue and organ induces cell membrane damage, resulting in calcium influx. Finally, the proinflammatory cytokines in COVID-19 patients inhibit parathyroid hormone secretion and impair response to parathyroid hormone, resulting in imbalance of calcium.[4] Given the high similarity between SARS-CoV-2 and SARS-CoV genomes, it is likely that the mechanisms seen in SARS-CoV could be the same for SARS-CoV-2.[6]

CONCLUSION

Arrhythmia in COVID-19 patients are more frequent in elderly with comorbidities such as in our patient, who developed arrhythmia associated with electrolyte imbalance probably due to the viral infection itself causing myocarditis, conduction system disease, gastrointestinal losses, and renal injury. Close monitoring and correction of hypokalemia and hypocalcemia are important as it was reported to be markers of clinical severity and worse prognosis.

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