

2020

RESPONSE: From Cardiology to COVID The Impact of a Pandemic on Fellowship Training

J. T. Kuvin

Zucker School of Medicine at Hofstra/Northwell

Follow this and additional works at: <https://academicworks.medicine.hofstra.edu/publications>



Part of the [Cardiology Commons](#)

Recommended Citation

Kuvin JT. RESPONSE: From Cardiology to COVID The Impact of a Pandemic on Fellowship Training. . 2020 Jan 01; 76(7):Article 6703 [p.]. Available from: <https://academicworks.medicine.hofstra.edu/publications/6703>. Free full text article.

This Article is brought to you for free and open access by Donald and Barbara Zucker School of Medicine Academic Works. It has been accepted for inclusion in Journal Articles by an authorized administrator of Donald and Barbara Zucker School of Medicine Academic Works. For more information, please contact academicworks@hofstra.edu.

Rhythm, conduction, and ST elevation with COVID-19: Myocarditis or myocardial infarction?



Amtul Mansoor, MD, David Chang, MD, Raman Mitra, MD, PhD, FHRS

From the Division of Electrophysiology, Department of Cardiology, North Shore University Hospital, Northwell Health, Manhasset, New York.

Introduction

Coronavirus disease 2019 (COVID-19) caused by the coronavirus 2 (SARS-CoV-2) predominantly affects the respiratory system but may also result in cardiac complications, including myocarditis.¹ SARS-CoV-2 binds to the angiotensin-converting enzyme-2 receptor expressed on the surface of alveolar and cardiac cells, which may account for the direct cardiac involvement in COVID-19.² Myocarditis is an inflammatory disease caused by infectious and noninfectious etiologies.³ Fulminant myocarditis is the most severe type of myocarditis and is predominantly caused by viral infections. It is characterized by a sudden and severe inflammation of myocardium resulting in cardiogenic shock, ventricular tachyarrhythmias, or bradyarrhythmias, and can be fatal.³ We describe a unique case in a critically ill patient with COVID-19, revealing rapid electrocardiographic changes and death within 24 hours despite aggressive treatment. The patient started with a normal baseline electrocardiogram (ECG), which progressed to marked PR prolongation with striking ST changes to a stable accelerated idioventricular rhythm arising from the left ventricle, and finally to multiorgan system failure and pulseless electrical activity. The differentiation of acute myocardial infarction from fulminant myocarditis based on the ECG, clinical course, and laboratory abnormalities is discussed.

Case report

A 72-year-old woman with hypertension presented with 2 weeks of myalgias and upper respiratory symptoms. Vital signs in the emergency department revealed a temperature of 101°F, heart rate of 137 beats per minute (bpm), respiratory rate of 24 breaths per minute, and oxygen saturation of 60% on room air. She remained hypoxic and was intubated and

KEY TEACHING POINTS

- It is important to recognize the cardiovascular complications among COVID-19 patients, including ischemia and conduction disease.
- It is important to differentiate between fulminant myocarditis and myocardial infarction.
- Owing to risk of exposure to COVID-19, one must weigh the benefits and risks of pursuing an invasive strategy to diagnose and treat cardiac manifestations late in the course of advanced disease.

transferred to the intensive care unit. The 12-lead ECG on admission was normal aside from sinus tachycardia (Figure 1A). Chest radiography revealed bilateral diffuse airspace opacities representing acute respiratory distress syndrome. A point-of-care echocardiogram revealed mildly reduced left ventricular dysfunction. The patient had a leukocytosis of 24,000/ μ L with lymphopenia, erythrocyte sedimentation rate of 82 mm/h, C-reactive protein of 27 mg/dL, D-dimer of 3455 ng/mL, ferritin of 928 ng/mL, and an elevated serum pro-brain natriuretic peptide of 4639 pg/mL with lactate of 4.2 mmol/L. A nasopharyngeal swab was positive for SARS-CoV-2 polymerase chain reaction assay. She was empirically started on vancomycin, meropenem, chloroquine, and azithromycin. Owing to oliguria, intravenous diuretic therapy was initiated, with improved urine output despite worsening creatinine level. Hemodynamics were stable and lactate normalized.

On the morning of day 6, worsening hypoxemia developed, associated with hypotension (76/57 mm Hg) requiring norepinephrine, phenylephrine, and vasopressin. A high-sensitivity troponin T (hsTt) was elevated at 118 ng/L. Marked ECG rhythm, conduction, and ST-segment changes occurred. Preceding this event, no significant changes in inflammatory laboratory values were present except for a marked increase in immature granulocytes. Key laboratory values are graphed in Figure 2. Bedside point-of-care echocardiography demonstrated mildly decreased left ventricular

KEYWORDS Coronavirus; COVID-19; Fulminant; Myocardial infarction; Myocarditis

(Heart Rhythm Case Reports 2020;6:671–675)

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors. The authors have no conflict to disclose. **Address reprint requests and correspondence:** Dr Amtul Mansoor, Division of Electrophysiology, Department of Cardiology, North Shore University Hospital, 300 Community Dr, Manhasset, NY 11030. E-mail address: amansoor@northwell.edu.

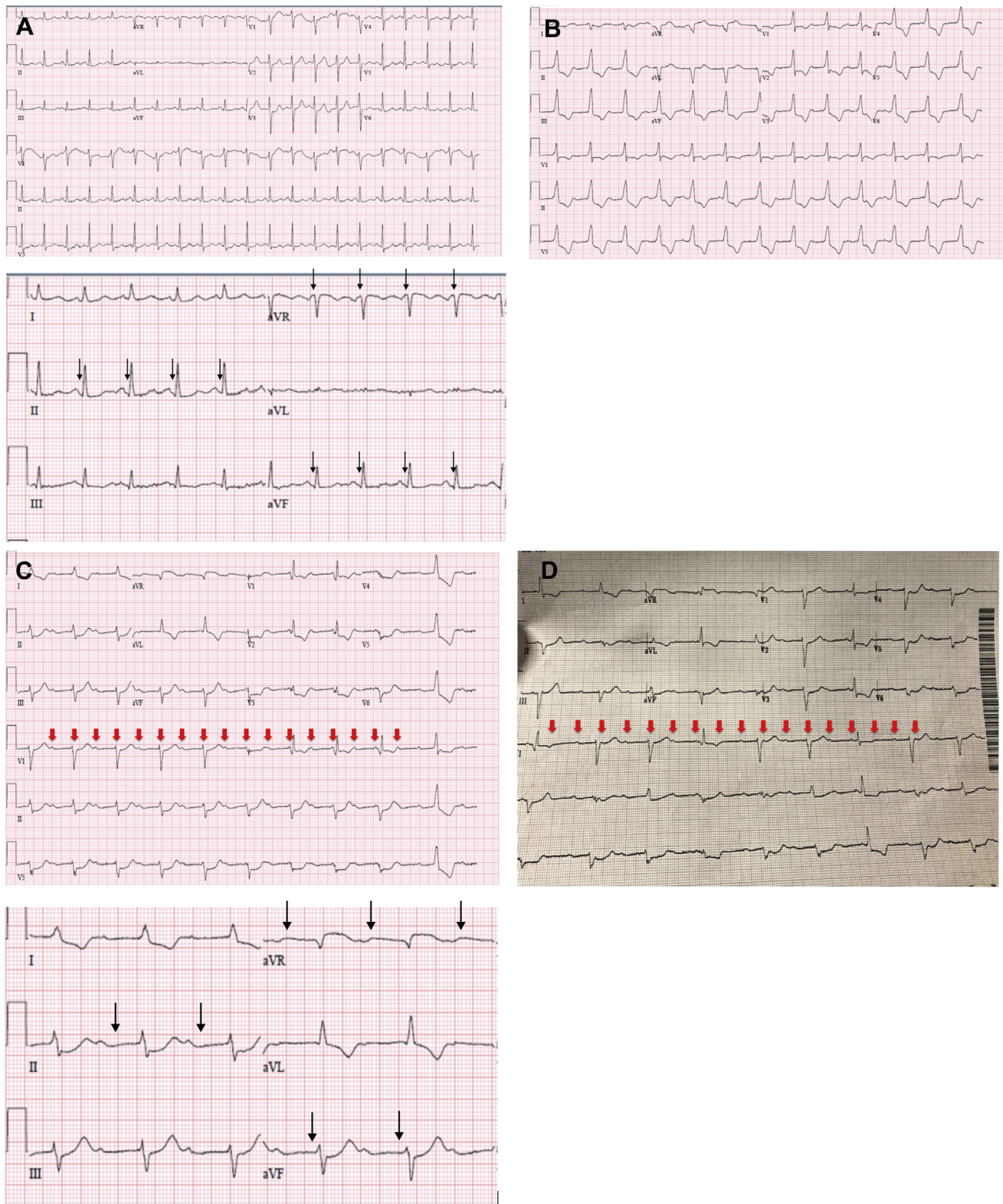


Figure 1 **A:** The patient's initial 12-lead electrocardiogram (ECG) in the emergency department. ECG upon admission to the emergency department demonstrating sinus tachycardia with heart rate of 123 beats per minute (bpm) and QTc 460 ms. Black arrows highlight the PR elevation present in aVR and PR depression in leads II and aVF, findings most specific to myopericarditis. **B:** ECG on March 17, 2020, 7:57 AM. Accelerated idioventricular rhythm at 105 bpm from the anterobasal left ventricle, which may arise from the left anterior fascicle, with underlying sinus or atrial tachycardia at 150 bpm and atrioventricular (AV) dissociation. QTc is 622 ms. **C:** ECG on March 17, 2020, 8:11 AM. ECG demonstrating sinus or atrial tachycardia with 2:1 AV block and left anterior hemiblock with development of an incomplete right bundle branch block halfway through the tracing (V₁ rhythm strip) followed by higher-grade block and a ventricular premature beat. Note the ST elevation in aVR and ST depression in leads 2 and V₄–V₆ with loss of precordial R waves. Red arrows denote the P waves. The blocked P waves highlights the PR elevation in aVR and PR depression in II and aVF (black arrows). QTc is 452 ms. **D:** ECG on March 17, 2020, 8:18 AM. ECG demonstrating continued sinus or atrial tachycardia with 2:1 conduction and ischemic ST changes and fusion beats. QTc is 472 ms.

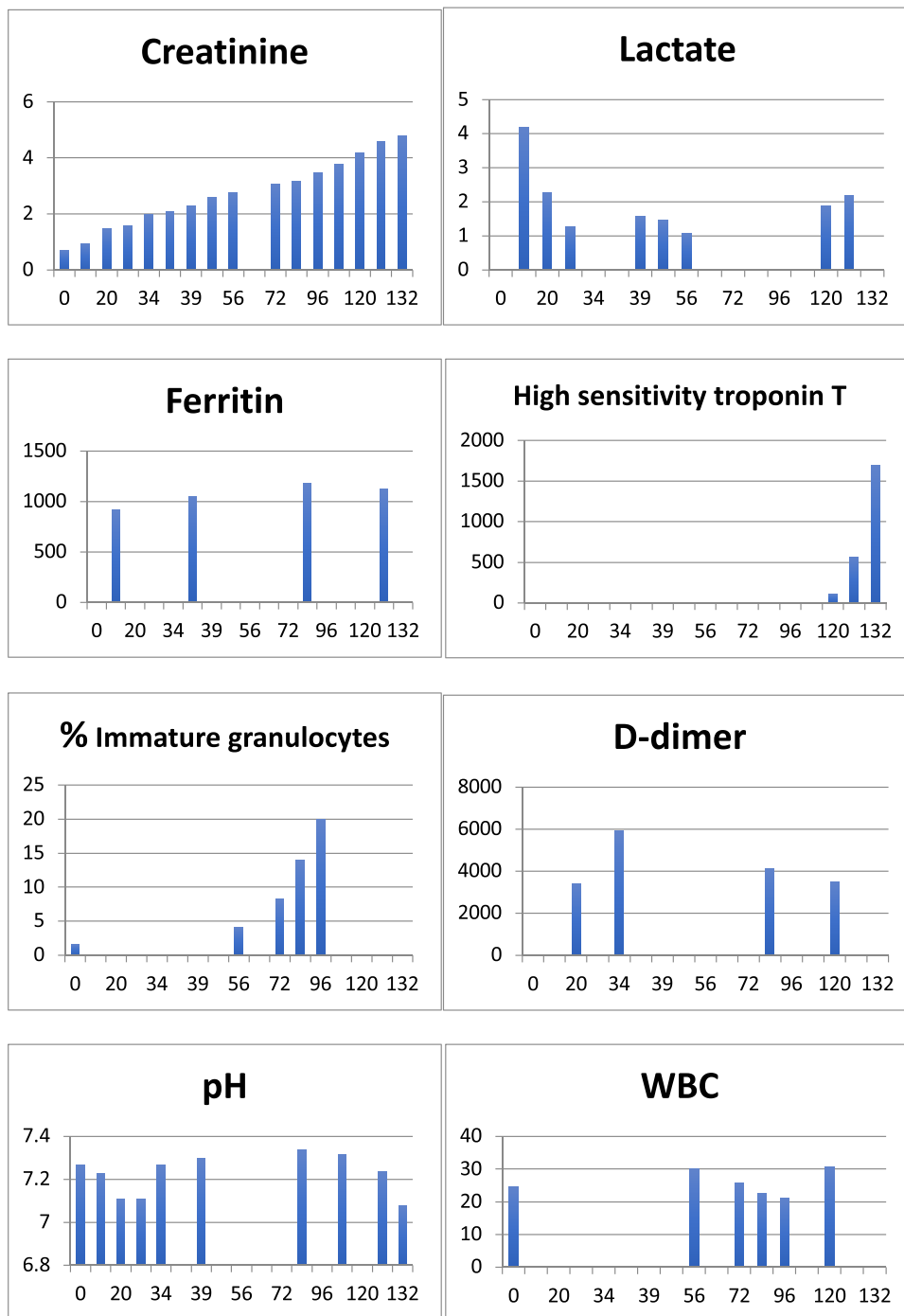


Figure 2 Trends of patient's creatinine, high-sensitivity troponin T, ferritin, D-dimer, pH, lactate, white blood cells (WBC), and % immature granulocytes plotted against hours into hospitalization.

function but no significant segmental wall motion abnormalities, mild mitral regurgitation, mildly enlarged right ventricle with normal right ventricular function, no tricuspid regurgitation, and no pericardial effusion. The serial ECGs are shown in Figure 1 (B–D). Figure 1B shows an accelerated idioventricular rhythm (AIVR) at 106 bpm with atrioventricular dissociation and underlying sinus vs atrial tachycardia at 150 bpm. The AIVR had a morphology compatible with origin from the anterobasal left ventricle. Within a few

minutes, the AIVR resolved and the underlying rhythm of sinus or atrial tachycardia with 2:1 conduction and a new left axis deviation and left anterior hemiblock is evident (Figure 1C). ST elevation in aVR and marked ST segment depressions in V₅ and V₆ with loss of precordial R waves are noted. Halfway through this tracing, a right bundle branch block (RBBB) also develops. At first glance, the tracing appears to be sinus rhythm with marked PR prolongation but the 2:1 conduction is visible at the end of the tracing (arrows

showing P waves) and a single ventricular beat of the same morphology as the AIVR. These ST ECG changes are often seen with a left main lesion.⁴ Alternatively, the AIVR has morphology compatible with left anterior fascicular ventricular tachycardia and the absence of capture beats with resolution into 2:1 conduction, consistent with development of heart block. A subsequent ECG with similar ST changes is shown in [Figure 1D](#) without RBBB and with occasional ventricular ectopy.

The patient was deemed too high risk for emergent coronary angiography. She was on subcutaneous heparin. Intravenous heparin was not given owing to worsening anemia. On vasopressors, her rhythm remained in the AIVR at 110 bpm. The possibilities of acute myocarditis or pulmonary embolus were not excluded by the limited bedside echocardiogram, though Takotsubo cardiomyopathy was less likely. Owing to concern for COVID-19-related myocarditis, the patient was started on pulse-dose steroids and intravenous immunoglobulin therapy, despite which she became hypotensive, with further elevation of lactate to 13.6 mmol/L and troponin to 1696 ng/L and pH reduced to 7.06. She died within 12 hours from the onset of ECG changes with pulseless electrical activity.

Discussion

This case illustrates unique rhythm and ST changes suggesting either late myocardial infarction or myocarditis after COVID-19. Initial presentation was weakness and hypoxia, with no signs of cardiac or renal disease. On the sixth day, an acute event occurred with sudden change in ECG rhythm with evidence for diffuse ischemia, which could be associated with left main disease, resulting in pulseless electrical activity and death. Whether this was due to hypercoagulability-related coronary thrombosis, ruptured plaque in the setting of systemic inflammation owing to cytokine storm, or destabilized coronary artery plaque is unclear.⁵ The concurrent conduction abnormalities may be due to diffuse ischemia, though an early fulminant myocarditis could present in a similar manner. Both would account for a global hypokinesia on echocardiography, while ST elevation in aVR with diffuse ST depression in the inferolateral leads is associated with left main disease.

Fulminant myocarditis is an acute, diffuse cardiac inflammation leading to cardiogenic shock, ventricular arrhythmias, and multiorgan failure.³ It can display a wide range of ECG changes, as seen in our patient, none of which are specific to its diagnosis. These include the presence of PR-segment depression in the precordial and limb leads, which may demonstrate atrial repolarization irregularities and reciprocal ST elevation in aVR ([Figure 1A](#)). Patients with fulminant myocarditis can also have presence of pathologic Q waves and intraventricular conduction delay or bundle branch block. Our patient did not have ECG changes with Q waves present but did have wide QRS, which progressed to RBBB ([Figure 1C](#)). This can occur owing to myocardial damage affecting the electrical conduction system and has been associ-

ated with a poorer prognosis, especially in fulminant myocarditis. This can lead to bradyarrhythmias or tachyarrhythmias.⁶ In this case, our patient developed AIVR with atrioventricular dissociation with low probability of survival. Our patient received treatment with chloroquine and azithromycin, medications known to increase the corrected QT interval. Recent studies have shown that in patients hospitalized with COVID-19 that received chloroquine/hydroxychloroquine and azithromycin, the treatment did result in an increase in the corrected QT interval but, despite this increase, did not result in arrhythmogenic death or torsades de pointes.⁷ It is less likely that these medications may have caused the arrhythmias, as the progression of these arrhythmias was more characteristic of fulminant myocarditis.

Coronary thrombosis and ST-elevation myocardial infarction (STEMI) has been described in COVID-19 patients. Shi and colleagues⁸ showed higher in-hospital mortality rate (42/82, 51.2%) in COVID-19 patients with evidence of myocardial injury based on elevated hsTt compared to those without myocardial injury (15/335, 4.5%). In addition, a higher degree of hsTt elevation correlated with a higher mortality rate.

The timing of ECG changes and coronary intervention may also be a useful prognostic parameter. Bangalore and colleagues⁹ reported on 18 COVID-19 patients with STEMI, and although not specifically mentioned in their paper, their data showed that none of the 8 patients with STEMI occurring 24 hours or more after admission survived with or without coronary angiography, while only 5 out of 10 patients with STEMI on presentation survived. A significant number of the STEMI patients had only nonobstructive lesions. Additionally, Stefanini and colleagues¹⁰ reported that of the patients that underwent urgent angiography, 40% did not have obstructive coronary artery disease. It was unable to be determined if the clinical presentation was due to type 2 myocardial infarction, or myocarditis, or cytokine storm.

Myocardial infarction may be associated with severe inflammation and ventricular dysfunction noted in fulminant myocarditis, as seen in patients with ST elevation and elevated hsTt, as well as increased leukocytosis, inflammatory markers, and pro-brain natriuretic peptide.¹¹ Our patient was started on a course of immunoglobulin and corticosteroids for possible fulminant myocarditis; however, no clinical improvement occurred. Given the late ischemic and rhythm changes in the hospital course, her prognosis was poor. The only laboratory value that portended the event was the increasing percentage of immature granulocytes. There have been isolated case reports of this in COVID-19 patients, but none that preceded an acute cardiac event.¹² A better understanding of the effect of SARS-CoV-2 on the heart would have been possible with an autopsy, but the family did not wish to pursue this.

Conclusion

COVID-19 patients may present with myocardial injury in the form of ECG changes, wall motion abnormalities on the echocardiogram, and laboratory abnormalities. Distinguishing a myocardial infarction vs fulminant myocarditis

may be difficult. The high comorbidity of these patients will often prevent aggressive management. The prognostic significance of immature granulocytes needs further study.

References

1. Madjid M, Safavi-Naeini P, Solomon SD, Vardeny O. Potential effects of coronaviruses on the cardiovascular system: a review [published online ahead of print March 27, 2020]. *JAMA Cardiol*. <https://doi.org/10.1001/jamacardio.2020.1286>.
2. Inciardi RM, Lupi L, Zaccone G, et al. Cardiac involvement in a patient with coronavirus disease 2019 (COVID-19). *JAMA Cardiol* 2020;5:1–6.
3. Chen C, Zhou Y, Wang DW. SARS-CoV-2: a potential novel etiology of fulminant myocarditis. *Herz* 2020;1–3.
4. Kurisu S, Inoue I, Kawagoe T, et al. Electrocardiographic features in patients with acute myocardial infarction associated with left main coronary artery occlusion. *Heart* 2004;90:1059–1060.
5. Driggin E, Madhavan MV, Bikdeli B, et al. Cardiovascular considerations for patients, health care workers, and health systems during the COVID-19 pandemic. *J Am Coll Cardiol* 2020;75:2352–2371.
6. Buttà C, Zappia L, Laterra G, Roberto M. Diagnostic and prognostic role of electrocardiogram in acute myocarditis: A comprehensive review. *Ann Noninvasive Electrocardiol* 2020;25. <https://doi.org/10.1111/anec.12726>.
7. Saleh M, Gabriels J, Chang D, et al. The effect of chloroquine, hydroxychloroquine and azithromycin on the corrected QT interval in patients with SARS-CoV-2 infection. *Circ Arrhythm Electrophysiol* 2020;13:e008662.
8. Shi S, Qin M, Shen B, et al. Association of cardiac injury with mortality in hospitalized patients with COVID-19 in Wuhan, China. *JAMA Cardiol* 2020;5:802–810.
9. Bangalore S, Sharma A, Slotwiner A, et al. ST-segment elevation in patients with Covid-19 – a case series. *N Engl J Med* 2020;382:2478–2480.
10. Stefanini GG, Montorfano M, Trabattoni D, et al. ST-elevation myocardial infarction in patients with COVID-19: clinical and angiographic outcomes. *Circulation* 2020;141:2113–2116.
11. Zheng Y, Ma Y, Zhang J, et al. COVID-19 and the cardiovascular system. *Nat Rev Cardiol* 2020;17:259–260.
12. Mitra A, Dwyer DM, Schivo M, et al. Leukoerythroblastic reaction in a patient with COVID-19 infection. *Am J Hematol* 2020;95:999–1000.