American Journal of Medical Case Reports, 2021, Vol. 9, No. 4, 249-252
Available online at http://pubs.sciepub.com/ajmcr/9/4/11
Published by Science and Education Publishing
DOI:10.12691/ajmcr-9-4-11

New Onset, Irreversible, Prolonged QT-Interval Requiring Permanent Biventricular Pacemaker in a COVID-19 Patient

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Received January 15, 2021; Revised January 26, 2021; Accepted February 03, 2021

Abstract Various electrocardiographic (EKG) manifestations have been reported in patients with coronavirus disease 2019 (COVID-19). There is growing evidence showing that new onset QT-prolongation is a common EKG finding in COVID-19 patients. In this report, we present a case of a 71-year-old man who was found to have a new onset, irreversible, prolonged QT-interval requiring permanent biventricular pacemaker despite testing negative twice for RT-PCR COVID-19 and correction of all known reversible causes. To date, there are a limited number of reports of irreversible QT-prolongation associated with COVID-19. This case report emphasizes the importance of a physician's clinical judgment in the setting of negative RT-PCR COVID-19 testing. A robust systemic inflammatory state seen in active COVID-19 infection is possibly the key mechanism precipitating the new EKG findings.

Keywords: *QT-prolongation, coronavirus disease 2019, RT-PCR COVID-19, cardiac pacemaker*

Cite This Article: Ahmad Jallad, Andrew V. Doodnauth, Justin Lee, Emmanuel Valery, Stephanie Myers, Dahlia Rizk, and Samy I. McFarlane, "New Onset, Irreversible, Prolonged QT-Interval Requiring Permanent Biventricular Pacemaker in a COVID-19 Patient." *American Journal of Medical Case Reports*, vol. 9, no. 4 (2021): 249-252. doi: 10.12691/ajmcr-9-4-11.

1. Introduction

Coronavirus disease-2019 (COVID-19), caused by severe acute respiratory syndrome coronavirus-2 infection, has resulted in significant morbidity and mortality. As of this writing, > 380,000 people have died as a result of this virus in the United States alone, and New York City has over 500 000 confirmed coronavirus disease 2019 (COVID-19) cases, New York City is considered a COVID-19 epicenter [1]. While this disease predominantly affects the lungs, often leading to pneumonia and acute respiratory distress syndrome, adverse effects on the heart have also been described.

Recent studies have suggested that cardiac conduction disease is becoming more common in critically ill COVID-19 patients. Abnormal electrocardiographic findings are likely multifactorial in COVID-19 patients and may be attributable to acute myocardial injury, hypoxia, metabolic disarray, neurohormonal/catecholaminergic stress, medications and severe systemic inflammation in the setting of viral infection in patients with or without prior CVD [2].

We highlight the importance of COVID-19 and its ability to impact the cardiac conduction system by presenting an interesting case of a 71-year-old male with suspected COVID-19 illness during the peak of the COVID pandemic in Brooklyn, NY. Subsequently, developing a new irreversible prolonged QT interval requiring permanent biventricular pacemaker implant.

2. Case Description

A 71-year-old male with PMHx of HTN, type-2 diabetes and known right bundle branch block, presented to the emergency department with dyspnea and fatigue. Four days prior to presentation the patient had noticed a decreased appetite with associated lower extremity edema. Patient denied recent travel, no sick contacts and no recent changes in medications. Review of systems were pertinent for subjective fever, no chills or cough.

Initial vital signs included temperature of 98.2 F, heart rate of 45 bpm, blood pressure of 169/90 mmHg, respiratory rate of 22 bpm and oxygen saturation of 98% on RA (room air). Physical exam was unremarkable. CXR showed bilateral opacities and initial labs were pertinent

for a potassium 4.6 mmol/L, creatinine of 1.8 mg/dL, magnesium of 2.0 mg/dL, troponin-I of 0.16 ng/mL and brain natriuretic peptide (BNP) of 1028 pg/mL. WBC was 11.09 K/uL, Hgb 13.4 g/dL and platelet count of 190 K/uL. Initial venous blood gas (VBG) showed pH of 7.30, pO2 23.7 mmHg, pCO2 45.6 mmHg, $HCO3^-$ 21.6 mmol/L and lactic acid of 5.7 mmol/L. Patient had mildly elevated initial ferritin level at 299.4 ng/mL and elevated C-reactive protein (CRP) at 41mg/L. Both levels were significantly elevated within 48 hours, 740.0 ng/mL and 277 mg/L respectively. Initial EKG showed sinus rhythm with right bundle branch block (RBBB), second-degree type II atrioventricular block (AVB) and prolonged QT interval (Figure 1), which differed from prior EKG 6 weeks ago, that showed sinus rhythm with RBBB and QTc was 433 ms. Transthoracic echocardiography (TTE) at that same time reported an ejection fraction (EF) of 49 % and CT coronary angiography 10 months prior showed non-obstructive coronary artery disease.

Shortly upon presentation, the patient developed acute respiratory failure, followed by profound bradycardia and cardiac arrest. Cardiopulmonary resuscitation was initiated and return of spontaneous circulation was achieved after 2 minutes of chest compression and administration of 1mg of IV epinephrine. Patient was intubated and a transvenous pacemaker was placed. Differential diagnosis included, but was not limited to acute hypoxic respiratory failure due to COVID-19 (given pandemic state in Brooklyn, NY) and progression of AV conduction abnormalities causing hemodynamic and respiratory collapse. The patient was admitted to the medical intensive care units (MICU) for further management.

On the following day, once the pacing rate was decreased to 40 bpm, we were able to appreciate a high degree AV block with an uncorrected QT interval at > 700ms (Figure 2). Bedside TTE was performed and showed an EF 45% and global hypokinesis with minor regional wall motion abnormalities. Given persistence of conduction abnormalities and significant new QT prolongation without readily identifiable reversible causes a decision was made to proceed urgently with biventricular pacemaker implantation despite repeat COVID-19 PCR results x2 being reported as negative (Figure 3).

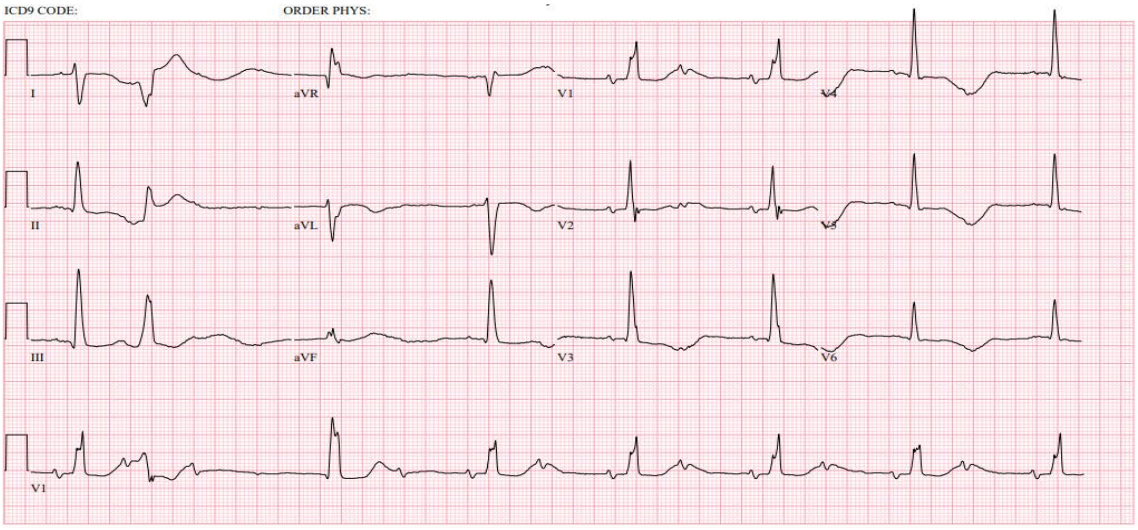

Figure 1. Initial EKG showing right bundle branch block and second degree type II AV block with 2:1 AV conduction with occasional premature ventricular complexes. Note massively prolonged QT interval irrespective of the correction methods.

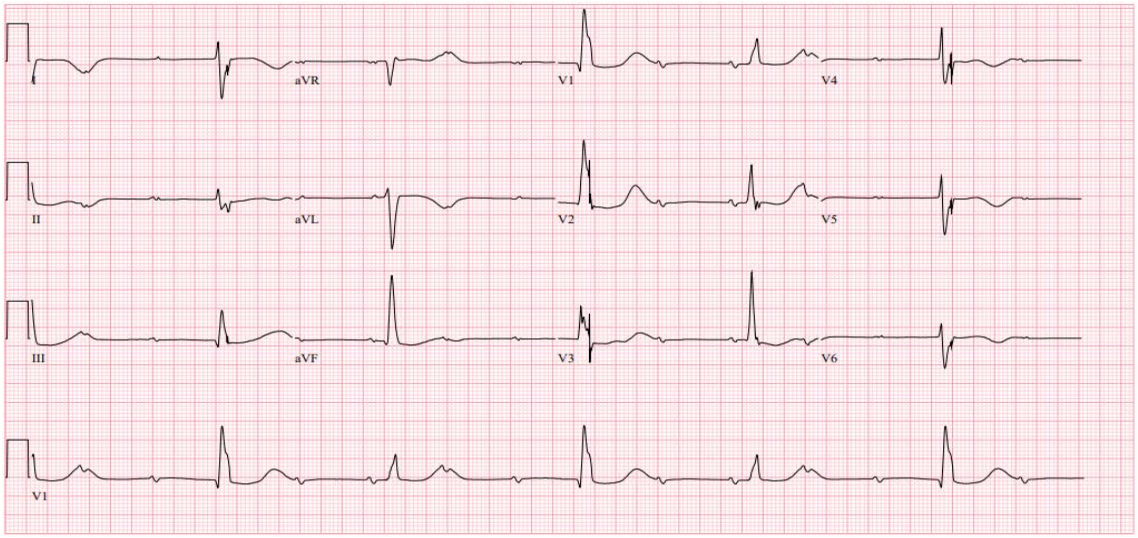

Figure 2. Repeat EKG (8 hr later) after transvenous pacemaker setting was changed to pacing rate at 40bpm. While off pacing, the patient re-demonstrated sinus rhythm with a high grade AV block. Again, significant QT prolongation is noted. (QT 732ms/QTc 558ms).

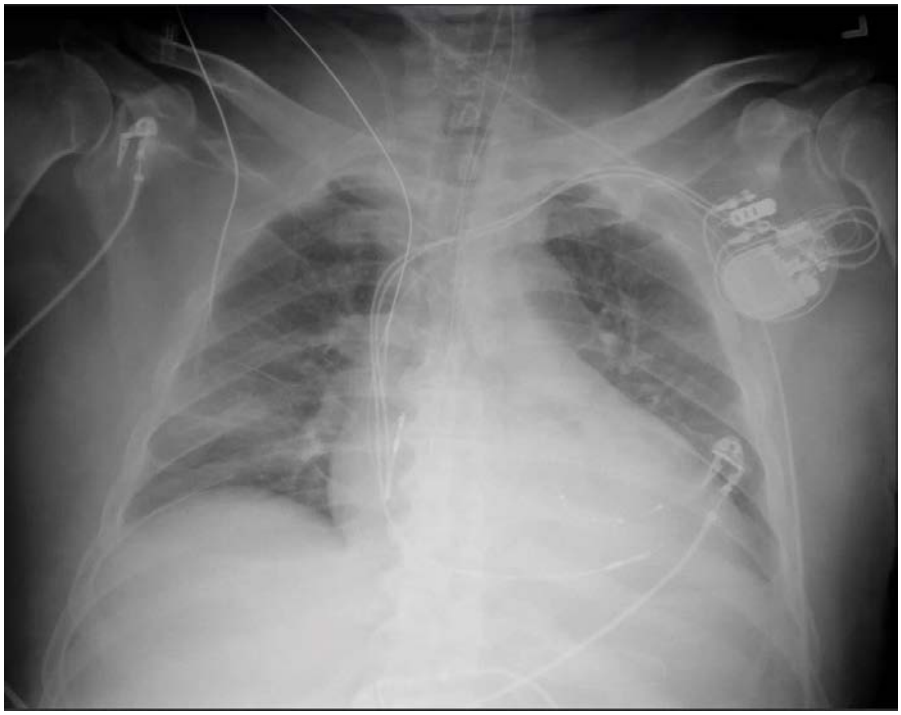

Figure 3. Chest X-ray finding after placement of biventricular pacemaker.

Patient was extubated on the day following pacemaker implantation, remained hemodynamically stable and was downgraded to the internal medicine service. He completed a course of doxycycline for presumed community acquired pneumonia and safely discharged with outpatient cardiology follow up.

3. Discussion

Patient's presentation with subjective fever, respiratory failure, mildly elevated ferritin and CRP, and infiltrative lung process is difficult not to be interpreted as potential COVID-19 infection as nearly all admissions to our hospital are. The most common clinical features reported at the onset of illness include fever (99%), fatigue (70%), dry cough (59%), anorexia (40%), myalgias (35%), dyspnea (31%), and sputum production (27 %) [3]. RT-PCR COVID 19 was sent twice and was all negative, as well as the expanded respiratory viral panel. Available data suggest, RT-PCR having a sensitivity of 60% in COVID-19 patients, while CT of the chest is 97 % sensitive, when compared to the PCR tests as a reference. However, the specificity of the CT scan was only 25 percent [4]. Early reports have suggested a high number of false-negative tests from upper respiratory specimens. Hence, if initial testing is negative, but the clinical suspicion for COVID-19 remains high and determining the presence of infection is important for management or infection control, it is recommended that the test be repeated. Furthermore, the WHO also recommends testing lower respiratory tract specimens, if possible [5,6].

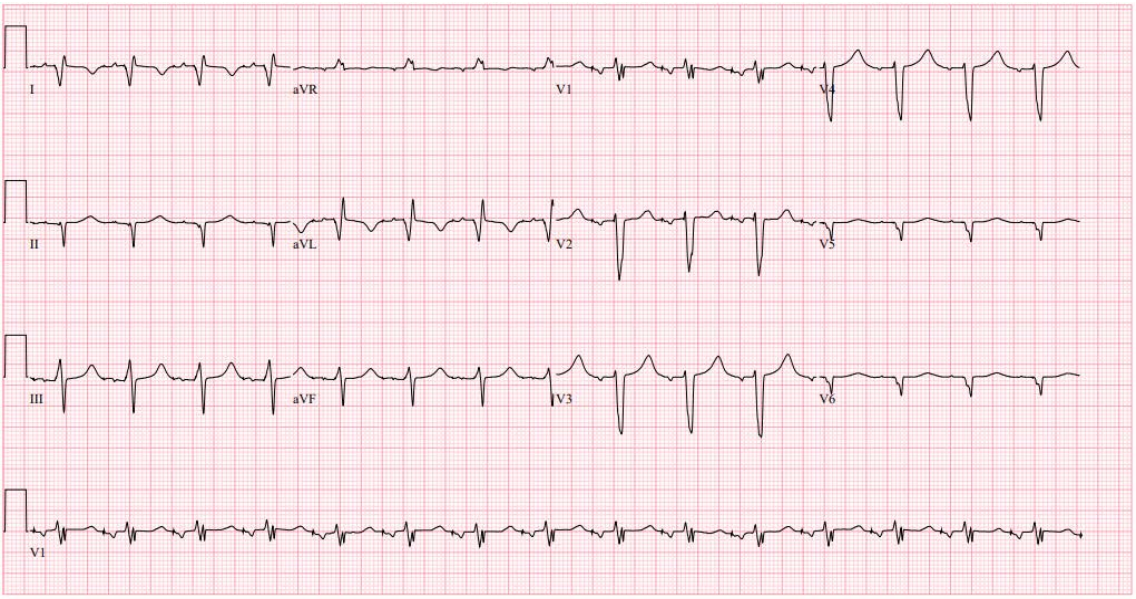

Figure 4. EKG after placement of biventricular pacemaker. Note the notably shortened QT interval.

Despite all the clues leading to the diagnosis of COVID-19 infection, this patient had a relatively common presentation of advanced conduction disease. Presence of 2:1 AVB and RBBB inevitably indicates presence of an infra-Hisian disease and necessitates pacemaker implantation [7]. Presence of massive QT prolongation is somewhat perplexing. We could not identify any reasons for QT prolongation. Neither medications nor electrolyte abnormalities that could explain it were present. We do not believe that profound myocardial ischemia was responsible for it either, since we have not seen ST segment deviation nor biochemical evidence for ischemia. Most likely reason, in our opinion, for the QT prolongation was residual effect for resuscitative efforts, combined with pre existing cardiomyopathy and a robust systemic inflammatory state. Given inherent fickleness of temporary pacing we made a decision to urgently proceed with pacemaker implantation and planning for setting the lower rate of the device to 90 bpm to afford shortening of the QT interval [8,9] (Figure 4).

The exact mechanism for the QT prolongation is uncertain, but in our opinion is due to a severe inflammatory state, resulting in cardiomyopathy and conduction deficits involving the atrioventricular node, His bundle, and Purkinje system.

4. Conclusion

In conclusion, we report an elderly male with suspected COVID-19 infection who developed worsening pathologic and excessive prolongation of QTc from baseline requiring biventricular pacemaker. Although the development of severe, irreversible QT-prolongation is multifactorial in etiology, we hypothesize that a robust systemic inflammatory response associated with COVID-19 is likely the predominant mechanism. Our case demonstrates the importance of a physician's mindful multifaceted approach to a critically ill patient irrespective of their COVID-19 test results.

Learning Objectives

1. Despite the dramatic influx of patients with COVID-19 infection, patients with "routine cardiac issues" will still present to the hospital.
2. Patients with left ventricular systolic dysfunction that require ventricular pacing benefit from resynchronization therapy [10].
3. Increasing pacing rate helps shorten the QT interval [8,9].

Acknowledgements

This work is supported, in part, by the efforts of Dr. Moro O. Salifu MD, MPH, MBA, MACP, Professor and Chairman of Medicine through NIMHD Grant number S21MD012474.

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