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# Association between electrocardiographic features and mortality in COVID-19 patients

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## Abstract

**Background:** Cardiovascular events have been reported in the setting of coronavirus disease-19 (COVID-19). It has been hypothesized that systemic inflammation may aggravate arrhythmias or trigger new-onset conduction abnormalities. However, the specific type and distribution of electrocardiographic disturbances in COVID-19 as well as their influence on mortality remain to be fully characterized.

**Methods:** Electrocardiograms (ECGs) were obtained from 186 COVID-19-positive patients at a large tertiary care hospital in Northern Nevada. The following arrhythmias were identified by cardiologists: sinus bradycardia, sinus tachycardia, atrial fibrillation (A-Fib), atrial flutter, multifocal atrial tachycardia (MAT), premature atrial contraction (PAC), premature ventricular contraction (PVC), atrioventricular block (AVB), and right bundle branch block (RBBB). The mean PR interval, QRS duration, and corrected QT interval were documented. Fisher's exact test was used to compare the ECG features of patients who died during the hospitalization with those who survived. The influence of ECG features on mortality was assessed with multivariable logistic regression analysis.

**Results:** A-Fib, atrial flutter, and ST-segment depression were predictive of mortality. In addition, the mean ventricular rate was higher among patients who died as compared to those who survived. The use of therapeutic anticoagulation was associated with reduced odds of death; however, this association did not reach statistical significance.

**Conclusion:** The underlying pathogenesis of COVID-19-associated arrhythmias remains to be established, but we postulate that systemic inflammation and/or hypoxia may induce potentially lethal conduction abnormalities in affected individuals.

**Abbreviations:** A-Fib, atrial fibrillation; AVB, atrioventricular block; COVID-19, coronavirus disease-19; ECG, electrocardiogram; LBBB, left bundle branch block; MAT, multifocal atrial tachycardia; PAC, premature atrial contraction; PVC, premature ventricular contraction; RBBB, right bundle branch block; SARS-CoV-2, severe acute respiratory syndrome coronavirus 2; SVT, supraventricular tachycardia; TwA, T-wave abnormalities.

Longitudinal studies are warranted to evaluate the risk factors, pathogenesis, and management of COVID-19-associated cardiac arrhythmias.

**KEYWORDS**
A-Fib, atrial fibrillation, cardiac arrhythmias, coronavirus disease-19, COVID-19, electrocardiogram

## 1 | INTRODUCTION

Coronavirus disease-2019 (COVID-19) has affected over fifty million individuals worldwide (Centers for Disease Control & Prevention, 2020). In many countries, including the United States, the number of confirmed infections continues to increase at an alarming rate. Nevertheless, despite a growing body of medical knowledge, much remains to be understood about this emerging disease.

The clinical presentation of COVID-19 is remarkably heterogeneous; although it was initially considered a respiratory illness, multiple organ systems are often affected (Hu et al., 2020). Recent data suggest that cardiac arrhythmias may occur in a significant number of patients diagnosed with COVID-19 (Colon et al., 2020). However, the type, duration, and frequency of COVID-19-associated arrhythmias remains to be established. We aimed to further characterize electrocardiographic abnormalities in patients with COVID-19 and determine which arrhythmias are associated with an increased risk of mortality.

## 2 | MATERIALS AND METHODS

### 2.1 | Population

We evaluated 186 consecutive patients at a 946-bed tertiary care hospital in Northern Nevada between April and June 2020 who had been diagnosed with COVID-19 via reverse transcriptase-polymerase chain reaction (RT-PCR) nasopharyngeal swab testing and had at least one electrocardiogram (ECG) after testing positive. We abstracted cardiologist-confirmed ECG reports and collected data pertaining to patient demographics, comorbidities, medication use, and COVID-19 outcomes during the hospital stay. The study was conducted in accordance with guidelines and regulations of the University of Nevada, Reno School of Medicine.

### 2.2 | Arrhythmia data

At least one ECG was obtained for all patients included in the study. Approximately half of the patients in the cohort (91 [48.9%]) had a baseline ECG for comparison. ECG activity was recorded and classified as follows: normal sinus rhythm, sinus tachycardia, sinus bradycardia, atrial fibrillation (A-Fib), atrial flutter, ST-segment depression, ST-segment elevation, multifocal atrial tachycardia (MAT), premature atrial contraction (PAC), premature ventricular contraction (PVC), atrioventricular block (AVB), left bundle branch block (LBBB), right bundle branch block (RBBB), and T-wave abnormalities (TwA).

### 2.3 | Statistical analysis

The frequency distribution of ECG rhythms before and after COVID-19 diagnosis was compared using Fisher's exact test of independence. Wilcoxon signed-rank test was used to assess the difference between baseline and post-infection ECG measurements. The Wilcoxon rank-sum test was used to compare ECG changes between survivors and non-survivors. We used the Z-test to assess the equality of proportions of observations among categories of comparison. A multivariable logistic regression model was constructed to assess the influence of ECG features on mortality. All tests were performed as 2-tailed and statistical significance levels set at a $p < .05$ in all applied analysis. Statistical analysis was performed using Stata (StataCorp Release 16).

## 3 | RESULTS

### 3.1 | Population

The median age was 60 years (range: 18–95 years). Patients younger than 61 years were disproportionately affected as compared to older patients based on known population demographics (98 [52.7%] vs. 88 [47.3%], respectively). Males were slightly overrepresented as compared to females (99 [53.2%] vs. 87 [46.8%], respectively). 154 patients were discharged, and 32 patients died during the hospitalization (82.7% vs. 17.2%, respectively). Troponin T levels were available in 140 patients. Baseline patient characteristics—including comorbidities and medications—and in-hospital events are further detailed in Table 1.

### 3.2 | Arrhythmia data

Most patients (152 [81.7%]) exhibited normal sinus rhythm on ECG. TwA represented the most common arrhythmia and were observed in 72 patients (38.7%). Among patients with TwA, 27 (14.5%) had normal troponin T levels and 45 (24.2%) had troponin levels greater than 19 ng/ml (our local upper limit of normal). There was a significantly increased rate of TwA among all patients with a troponin T

**TABLE 1** Baseline patient characteristics, in-hospital events, and post-infection ECG features

| Baseline patient characteristics | Alive ($n$ = 154, 82.8%) | Dead ($n$ = 32, 17.2%) | Total ($n$ = 186, 100%) | $p$-value | Cramér's V |
|---|---|---|---|---|---|
| Female | 72 (46.8) | 15 (46.9) | 87 (46.8) | .57 | |
| 61 years and above | 65 (42.2) | 23 (71.9) | 88 (47.3) | <.01 | 0.22[a] |
| Tobacco use | 34 (22.1) | 10 (31.3) | 44 (23.7) | .19 | |
| Alcohol use | 39 (25.3) | 5 (15.6) | 44 (23.7) | .17 | |
| Coronary artery disease | 4 (2.6) | 2 (6.3) | 6 (3.2) | .28 | |
| Heart failure | 12 (7.8) | 6 (18.8) | 18 (9.7) | .06 | |
| Hypertension | 60 (39) | 20 (62.5) | 80 (43.1) | .01 | 0.18[a] |
| A-Fib-Flutter | 6 (8.6) | 3 (14.3) | 9 (9.9) | .34 | |
| Stroke | 13 (8.4) | 3 (9.4) | 16 (8.6) | .54 | |
| COPD | 9 (5.4) | 0 | 9 (4.8) | .18 | |
| Asthma | 7 (4.6) | 1 (3.1) | 8 (4.3) | .59 | |
| Diabetes | 56 (36.4) | 13 (40.6) | 69 (37.1) | .39 | |
| Chronic kidney disease | 11 (7.1) | 7 (21.9) | 18 (9.7) | .02 | 0.19[a] |
| Obesity | 40 (26) | 5 (15.6) | 45 (24.2) | .15 | |
| Sleep apnea | 13 (8.4) | 1 (3.1) | 14 (7.5) | .27 | |
| Beta blockers | 31 (55.4) | 8 (50) | 39 (54.2) | .46 | |
| ACEI/ARB | 23 (41.8) | 7 (43.8) | 30 (42.3) | .56 | |
| Diltiazem | 4 (10.3) | 0 | 4 (7.7) | .31 | |
| Amlodipine | 15 (33.3) | 2 (15.4) | 17 (29.3) | .18 | |
| QT prolonging medications | 88 (57.1) | 19 (59.4) | 107 (57.5) | .49 | |
| In-hospital events | | | | | |
| Hypoxia | 91 (59.1) | 25 (78.1) | 116 (62.4) | .03 | 0.15[a] |
| Sepsis | 29 (18.8) | 14 (43.8) | 43 (23.1) | <.01 | 0.22[a] |
| Extended hospital stay (>11 days) | 39 (25.3) | 14 (43.8) | 53 (28.5) | .03 | 0.15[a] |
| ICU stay | 28 (18.2) | 24 (75) | 52 (28) | <.001 | 0.48[a] |
| Elevated troponin T (>19 ng/ml) | 32 (27.8) | 21 (84) | 53 (37.9) | <.001 | 0.44[a] |
| Anticoagulation | 75 (48.7) | 14 (43.8) | 89 (47.9) | .38 | |
| Post-infection ECG features | | | | | |
| Sinus rhythm | 127 (82.5) | 25 (78.1) | 152 (81.7) | .36 | |
| Sinus bradycardia | 13 (8.4) | 1 (3.1) | 14 (7.5) | .27 | |
| Sinus tachycardia | 44 (28.6) | 12 (37.5) | 56 (30.1) | .21 | |
| Atrial fibrillation-flutter[b] | 12 (7.8) | 12 (37.5) | 24 (12.9) | <.001 | 0.33[a] |
| SVT | 1 (0.7) | 2 (6.3) | 3 (1.6) | .08 | |
| PAC | 6 (3.9 | 5 (15.6) | 11 (5.9) | .02 | 0.19[a] |
| PVC | 7 (4.6) | 3 (9.4) | 10 (5.4) | .24 | |
| A-V Block | 14 (9.1) | 8 (25) | 22 (11.8) | .02 | 0.19[a] |
| LBBB | 2 (1.3) | 1 (3.1) | 3 (1.6) | .43 | |
| RBBB | 9 (5.8) | 5 15.6) | 14 (7.5) | .07 | |
| T-wave abnormalities | 54 (35.1 | 18 (56.3) | 72 (38.7) | .02 | 0.16[a] |
| ST elevation | 8 (5.2) | 7 (21.9) | 15 (8.1) | <.01 | 0.23[a] |
| ST depression | 7 (4.5) | 9 (28.1) | 16 (8.6) | <.001 | 0.32[a] |

Abbreviations: ACEI, angiotensin-converting enzyme inhibitor; ARB, angiotensin II receptor blocker; AV block, atrioventricular block; COPD, chronic obstructive pulmonary disease; LBBB, left bundle branch block; PAC, premature atrial contraction; PVC, premature ventricular contraction; RBBB, right bundle branch block; SVT, supraventricular tachycardia.

[a]Denotes statistically significant associations with mortality. The strength of associations is described using Cramer's V, with a higher V denoting a stronger association.

[b]Atrial fibrillation-flutter includes patients with atrial fibrillation and/or atrial flutter.

level greater than 19 ng/ml ($p = .02$). However, there was no significant difference in mortality in comparing TwA patients with and without troponin T elevation.

Sinus tachycardia was also identified in 56 patients (30.1%). Other ECG changes that were present with increased frequency among COVID-19 patients as compared to the general population included A-Fib, ST-segment depression, ST-segment elevation, and PAC, which were identified in 21 (11.3%), 16 (8.6%), 15 (8.1%), and 11 (5.9%) patients, respectively (Khurshid et al., 2018). A-Fib and atrial flutter, when considered as a single category, was identified in 24 individuals (12.9%). The incidence of ST-segment depression in our sample was 8.6%. There was a significant association between troponin T level and the presence of ST-segment depression ($p < .01$). However, serum troponin T levels were not significantly different between patients with ST-segment depression who died and those who survived. Figure 1 illustrates the frequency distribution of cardiac rhythms before and after COVID-19 infection.

The majority of patients (182 [68.8%]) had normal ventricular rates. Bradycardia and tachycardia occurred in 4 (2.2%) and 54 (29%) patients, respectively. Tachycardia was significantly more common among those who died (median ventricular rate was 99 beats/min [range: 68–174] vs. 88 beats/min [range: 49–146], respectively; $p < 0.001$). Figure 2 summarizes the baseline and post-infection ventricular rates among survivors and non-survivors.

A total of 91 patients had baseline ECGs for comparison. New-onset arrhythmias occurred with the following frequency: TwA—31 patients (34.1%); A-Fib—9 patients (9.9%); atrial flutter—1 patient (1.1%); A-Fib/atrial flutter—10 patients (11.0%); ST-segment depression—7 patients (7.7%); ST-segment elevation—5 patients (5.5%); PACs—4 patients (4.4%); and sinus tachycardia—7 patients (7.7%). Q waves were not present in patients with ST depression. The distribution of ECG measurements before and after COVID-19 diagnosis is further detailed in Figure 3. The median baseline PR interval was 162 milliseconds (msec; range: 112–247); this was not significantly different from the post-infection PR interval of 156 msec (86–219). There was no significant difference in QRS duration before and after infection (99 [68–170] msec vs. 88 [62–184] msec). The post-infection corrected QT interval did not differ from the baseline value (443 [336–570] msec vs. 445 [383–521] msec).

### 3.3 | Mortality data

A total of 32 patients (17.2%) died during the hospitalization. A-Fib, atrial flutter, and ST-segment depression were predictive of mortality (Table 2). In addition, the mean ventricular rate was higher among patients who died as compared to those who survived (Figure 2). The use of therapeutic anticoagulation was associated with reduced odds of death; however, this association did not reach statistical significance. No other statistically significant associations between ECG changes and mortality were identified.

## 4 | DISCUSSION

COVID-19 is a novel infection caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). Initial reports from Wuhan, China—the epicenter of the pandemic—described COVID-19 as a predominantly respiratory illness. However, emerging data indicate that COVID-19 may present with multisystem inflammation affecting the brain, gastrointestinal tract, skin, and/or heart (Hu et al., 2020).

Cardiac conduction abnormalities have recently been added to the list of COVID-19 sequelae. Indeed, heart palpitations are reported as part of the presenting symptomatology in nearly 10% of affected individuals (Angeli et al., 2020). Furthermore, one recent investigation revealed that 16.7% of patients who were hospitalized with COVID-19 exhibited a cardiac arrhythmia on ECG; this increased to nearly 50% among those managed in the intensive care unit (Wang et al., 2019). However, there is a paucity of data describing the type, frequency, and clinical implications of COVID-19 associated arrhythmias. Our analysis contributes to the growing body of medical knowledge pertaining to the effects of COVID-19 on the cardiac conduction system.

Most patients in our sample demonstrated normal sinus rhythm on ECG. Sinus tachycardia was recorded in approximately one-third of patients. However, this is a normal, non-specific physiologic response to increased cardiac demand and frequently occurs in the setting of infection. Sinus bradycardia was also present in a small number of individuals. Pathologic arrhythmias that were recorded included A-Fib, atrial flutter, SVT, MAT, PAC, PVC, AVB, RBBB,

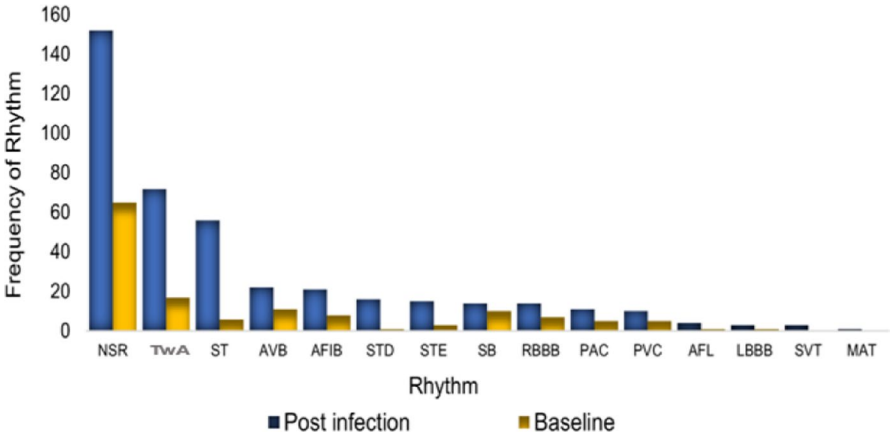


**FIGURE 1** Distribution of ECG rhythms before and after COVID-19 infection

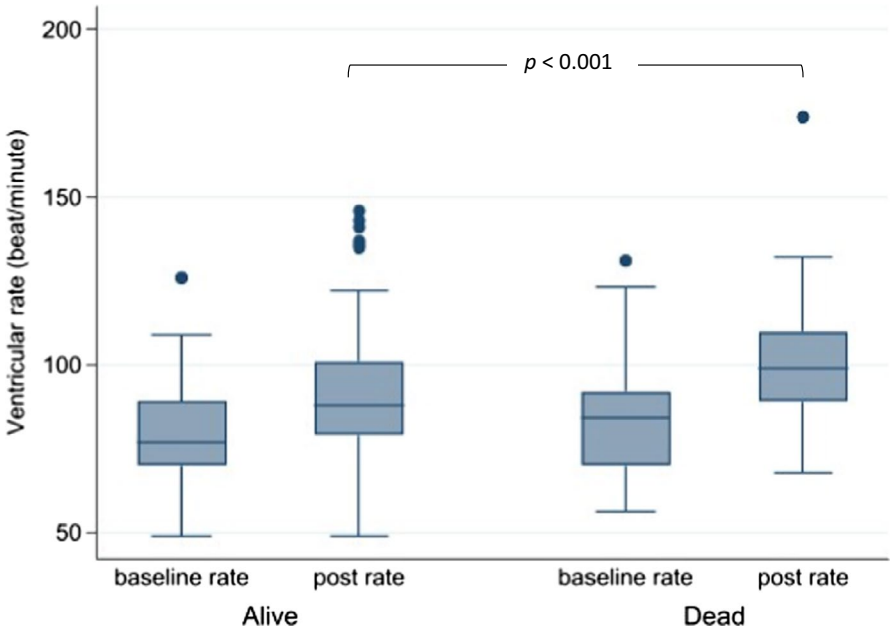


**FIGURE 2** Box plot comparing pre- and post-infection ventricular rates among survivors and non-survivors

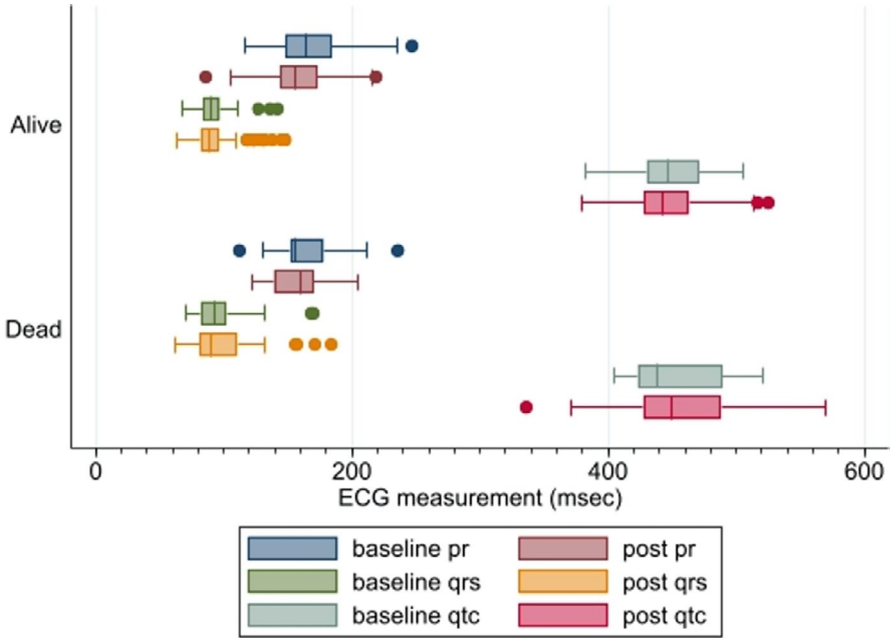


**FIGURE 3** Box plot comparing pre- and post-infection ECG measurements among survivors and non-survivors. AF, atrial flutter; A-FIB, atrial fibrillation; AVB, A-V block (1° and Mobitz type II); LBBB, left bundle branch block; MAT, multifocal atrial tachycardia; NSR, normal sinus rhythm; PAC, premature atrial contraction; PVC, premature ventricular contraction; RBBB, right bundle branch block; SB, sinus bradycardia; ST, sinus tachycardia; STD, ST-segment depression; STE, ST-segment elevation; SVT, supraventricular tachycardia; TwA, T-wave abnormalities

T-wave abnormalities, and ST-segment elevation. The presence of A-Fib, atrial flutter, or ST-segment depression was predictive of mortality.

COVID-19-associated A-Fib was recently described by Colon et al., who observed that 10.4% of patients admitted with COVID-19 developed new-onset A-Fib during their hospitalization (Colon et al., 2020). Other atrial tachyarrhythmias that were recorded included atrial flutter and atrial tachycardia. Notably, all patients with atrial tachyarrhythmias required management in the intensive care unit, and 26.3% died due to COVID-19-related complications. The authors noted that individuals who developed atrial tachyarrhythmias tended to be older and had higher levels of C-reactive protein and D-dimer as compared to their counterparts who remained in normal sinus rhythm. It is therefore conceivable that the characteristic multisystem inflammatory response of COVID-19 is implicated in the pathogenesis of cardiac conduction disease. Indeed, C-reactive protein, D-dimer, interleukin-6, and other inflammatory molecules have been associated with an increased risk of A-Fib (Chung et al., 2001). The underlying mechanism remains to be established, but it has been hypothesized that the release of inflammatory cytokines triggers a neurohormonal cascade that causes atrial hypocontractility via disruption of calcium-dependent signaling and induces the production of A-Fib-promoting fibrous tissue (Harada et al., 2015).

In our study, there was a significant increase in mortality among patients with A-Fib or atrial flutter as compared to those without either arrhythmia. Causation could not be assessed due to the retrospective study design. It is plausible that the relationship between A-Fib/atrial flutter and mortality in COVID-19 patients is unrelated

**TABLE 2** Multivariable logistic regression of death

| Variable | aOR | 95% Confidence Interval | *p*-value |
|---|---|---|---|
| Aged 61 and above | 2.81 | 1.05–7.53 | .04 |
| ST depression | 5.47 | 1.17–25.59 | .03 |
| A-Fib or A-Flutter | 4.79 | 1.63–14.11 | .004 |
| PAC | 3.09 | 0.73–13.11 | .13 |
| Sinus tachycardia | 2.64 | 0.71–9.82 | .15 |
| ST elevation | 1.89 | 0.35–10.22 | .46 |
| T-wave abnormalities | 1.23 | 0.45–3.39 | .69 |
| Anticoagulation | 0.56 | 0.22–1.43 | .22 |

Abbreviations: A-Fib, atrial fibrillation; A-Flutter, atrial flutter; PAC, Premature atrial contraction.

to any specific properties of SARS-CoV-2 and simply represents a manifestation of severe disease; it has been established that individuals with sepsis or septic shock have a markedly increased risk of developing new-onset A-Fib (Meierhenrich et al., 2010). Among the comorbid conditions known to impact the risk for embolic events in patients with A-Fib, only hypertension showed a significant association with death in our sample.

Considering the risks of hemodynamic instability and thromboembolism, prompt management of A-Fib/atrial flutter is vital; this may involve antiarrhythmic drugs and/or electrical cardioversion as well as anticoagulation. In addition, in the setting of COVID-19, corticosteroids should be considered for attenuation of the underlying inflammatory response. Indeed, recent data indicate that administration of dexamethasone reduces mortality in hospitalized COVID-19 patients (Ledford, 2020). Further studies are needed to investigate the effect of corticosteroids on arrhythmia burden in patients with COVID-19.

In addition to A-Fib, ST-segment depression was predictive of mortality in our cohort of COVID-19 patients. ST-segment depression is a non-specific indicator of myocardial ischemia and is commonly observed in myocarditis, hypoxia, and acute posterior myocardial infarction. Non-cardiac causes of ST-segment depression include pulmonary embolism, pneumothorax, and sepsis. The underlying cause of ST-segment depression in COVID-19 is variable. Myocarditis represents a common and potentially lethal disease manifestation (Hu et al., 2020). COVID-19-associated acute myocardial infarction has also been reported (Hu et al., 2020). There are myriad potential causes of myocardial ischemia in the setting of overwhelming infection. We postulate that ST-segment depression represents a surrogate marker for severe systemic inflammation, which is thought to be a hallmark of COVID-19.

### 4.1 | Limits of the study

The aim of the study was to evaluate ECG manifestations of COVID-19 and determine which, if any, were associated with an increased risk of mortality.

The study had some limitations inherent to its design. First, it was retrospective in nature and therefore causality could not be assessed. Second, baseline ECGs were not available in all patients, and it is possible that some ECG abnormalities were present prior to the diagnosis of COVID-19; this limited the sample size used in the analysis. Third, treatment data were not collected, and some pharmacologic agents used for the management of COVID-19 may have affected ECG recordings. Despite these limitations, we believe our analysis reveals important findings pertaining to COVID-19-related cardiac conduction disease. As the COVID-19 pandemic continues to accelerate, further investigation into the mechanisms of pathogenesis and optimal management of infection-associated arrhythmias has the potential to improve patient outcomes worldwide.

## ETHICS APPROVAL STATEMENT

The study protocol and implementation were deemed exempt from full review by the Institutional Review Board of the University of Nevada, Reno School of Medicine. The study was conducted in accordance with institutional regulations and guidelines.

## CONFLICT OF INTEREST

The authors declare that they have no conflicts of interest or relationships with industry.

## DATA AVAILABILITY STATEMENT

Data are available from the authors upon reasonable request.

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