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Electrocardiographic Findings in Coronavirus Disease-19: Insights on Mortality and Underlying Myocardial Processes

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ABSTRACT

Background: Coronavirus disease 2019 (COVID-19) is a respiratory syndrome with high rates of mortality, and there is a need for easily obtainable markers to provide prognostic information. We sought to determine whether the electrocardiogram (ECG) on hospital presentation provides prognostic information, specifically related to death.

Methods and Results: We performed a retrospective cohort study in patients with COVID-19 who had an ECG at or near hospital admission. Clinical characteristics and ECG variables were manually abstracted from the electronic health record and first ECG. Our primary outcome was death.

There were: 756 patients who presented to a large New York City teaching hospital with COVID-19 who underwent an ECG. The mean age was 63.3 ± 16 years, 37% were women, 61% of patients were nonwhite, and 57% had hypertension; 90 (11.9%) died. In a multivariable logistic regression that included age, ECG, and clinical characteristics, the presence of one or more atrial premature contractions (odds ratio [OR] 2.57, 95% confidence interval [CI] 1.23–5.36, P = .01), a right bundle branch block or intraventricular block (OR 2.61, 95% CI 1.32–5.18, P = .002), ischemic T-wave inversion (OR 3.49, 95% CI 1.56–7.80, P = .006), and nonspecific repolarization (OR 2.31, 95% CI 1.27–4.21, P = .006) increased the odds of death. ST elevation was rare (n = 5 [0.7%]).

Conclusions: We found that patients with ECG findings of both left-sided heart disease (atrial premature contractions, intraventricular block, repolarization abnormalities) and right-sided disease (right bundle branch block) have higher odds of death. ST elevation at presentation was rare. (J Cardiac Fail 2020;26:626–632)

Keywords: COVID-19, electrocardiography, myocardial injury, mortality.

Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) is a highly infectious virus that causes coronavirus disease (COVID-19), a respiratory syndrome associated with high rates of critical illness and mortality. As a consequence, there is a need to identify prognostic markers that can aid clinicians in the rapid triage of patients, guide clinical decision making, and inform patients and their families about the anticipated disease trajectory. Electrocardiography (ECG) is a broadly available diagnostic test that can be quickly performed without exposing a large number of personnel to SARS-CoV2. ECG has demonstrated incremental prognostic value in population-based studies and in patients with a variety of underlying cardiovascular conditions, including hypertension, and thus offers a particularly appealing modality during the current pandemic. We thus sought to determine whether findings on the first presenting ECG provide prognostic information, specifically related to death, and subsequently provide insights on myocardial processes underlying a poor prognosis.

Methods

Study Population and Setting

Our retrospective observational cohort study included patients with confirmed COVID-19 who presented to Weill Cornell Medicine/New York-Presbyterian Hospital, a quaternary referral center and 862-bed teaching hospital, from March 3, 2020, through April 9, 2020. All cases of COVID-19 were confirmed by real-time reverse-transcriptase polymerase chain reaction on nasopharyngeal swabs. We included consecutive patients who presented directly to our hospital and underwent an ECG at or near their
initial presentation to the hospital. We excluded patients who had complete ventricular pacing, because these ECGs were otherwise nondiagnostic. Using a standardized protocol and REDCap tool, patient data were manually abstracted from the electronic health record using methods that have been previously described. These data were used to develop a COVID-19 registry database.

**Primary Independent Variable: ECG**

ECGs were personally reviewed and interpreted by 2 electrocardiographers (S.A.M. and P.O., together responsible for the interpretation of >100,000 ECGs per year) who were blinded to the clinical status of the patients. Any disagreement in interpretation between readers was resolved by consensus. No formal testing of between- or within-reader variability of interpretation was performed for this study. Data extracted from each ECG included heart rate, rhythm categorized as normal sinus rhythm or atrial fibrillation/flutter, the presence of atrial premature contractions (APCs), ventricular premature contractions, atrioventricular block, axis deviation, right bundle branch block (RBBB), left bundle branch block, a nonspecific intraventricular conduction block (IVB) (QRS duration of >110 ms), presence of left or right ventricular hypertrophy, an age indeterminate myocardial infarction, the presence of ST segment or T-wave changes (localized ST elevation, localized T-wave inversion, or other nonspecific repolarization abnormalities), and the Bazett-corrected QT interval (in milliseconds).

**Outcomes**

The primary outcome was death occurring through April 23, 2020, ensuring at least 2 weeks of outcome data for all included patients. We ascertained death based on review of discharge summaries and death notes in the electronic health record.

**Covariates**

Demographics (age, sex, and race) and preexisting comorbid conditions (smoking status, history of immunosuppression, diabetes mellitus, hypertension, chronic obstructive pulmonary disease, end-stage renal disease, coronary artery disease [CAD], chronic heart failure, stroke, and active cancer) were abstracted from the electronic health record. Additionally, the clinical decision to start supplemental oxygen therapy owing to hypoxemia within 3 hours of presentation was abstracted from the respiratory flowsheets.

**Statistical Analysis**

Statistical analysis was performed using SPSS version 25 software (IBM, Inc., Armonk, NY). Data are presented as mean ± standard deviation for continuous variables and proportions for categorical variables. The relationship between clinical and ECG characteristics and death was examined using univariate and multivariable logistic regression analyses. Statistically significant univariate predictors of death were entered into forward selection multivariable models with a P-value of .05 or less required for entry: an age and ECG variables model; age and clinical variables model; and finally, a model incorporating age, clinical, and ECG variables.

**Oversight**

This study was approved by the Weill Cornell Medicine Institutional Review Board, which waived informed consent.

**Results**

**Patient Characteristics**

From March 3, 2020, through April 9, 2020, 945 patients presented to our hospital and were confirmed by PCR to have SARS-CoV-2. Of these patients, 768 underwent an ECG at or near the time of their admission; 12 patients were excluded from analysis for complete ventricular pacing (Fig. 1). Among the remaining 756 patients, the median time between presentation to the ED and initial ECG was less than 1 day and 94.3% had their ECG within 1 day of presentation. Clinical characteristics of the population are shown in Table 1. The mean age was 63.3 ± 16 years, 37% were women, 61% of patients were nonwhite, 37%

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**Figure 1.** Patients presenting with coronavirus disease-19 from March 3, 2020, to April 9, 2020.
obesity, and 29% had diabetes mellitus. Cardiovascular conditions were common: 57% had hypertension, 14% had CAD, 7% had heart failure, and 7% had a prior stroke. More than one-half (55%) required supplemental oxygen within the first 3 hours of presentation.

**Electrocardiographic Findings**

Baseline electrocardiographic characteristics (Table 2) included a mean heart rate of 90 ± 19 bpm and mean Bazett-corrected QT interval of 449 ± 144 ms. The overwhelming majority were in normal sinus rhythm (94.4%); 5.6% of patients had atrial fibrillation/flutter. Atrioventricular block was rare (2.6%): 19 patients (2.5%) had a first-degree block and 1 patient (0.1%) was in sinus rhythm with complete heart block and a junctional escape rhythm. APCs occurred in 7.7% and ventricular premature contractions in 3.4%. A significant proportion (19.3%) had an abnormal axis—13.8% had left axis deviation and 5.5% had a right or right superior axis deviation. Abnormal intraventricular conduction was found in 11.8%, with RBBB in 7.8%, left bundle branch block in 1.5%, and nonspecific IVB in 2.5%. Left ventricular hypertrophy (15.5%) was more common than right ventricular hypertrophy (4.0%) and evidence of a previous Q-wave myocardial infarction was present in 13.9.

Repolarization abnormalities were common (40.2%): 0.7% had localized ST elevation, 10.5% localized T-wave inversion, and 29.0% had nonspecific repolarization abnormalities (Table 2).

**Relationship of Clinical and Electrocardiographic Characteristics to Mortality**

A total of 90 patients (11.9%) died during the follow-up period. The relationship of clinical and ECG variables to all-cause mortality is shown in Tables 3 and 4. In univariate logistic regression analyses, clinical predictors of death included age, race, prior smoking, existing renal or pulmonary disease, active cancer, being immunosuppressed, and greater number of home medications, but did not include sex, obesity, diabetes, home oxygen, use or a history of rheumatic disease. Additionally, cardiovascular comorbidities (CAD, chronic heart failure, hypertension, or prior stroke) were all strong univariate predictors of death. Hypoxemia requiring supplemental oxygen within 3 hours of presentation was also strongly associated with death (OR 3.52, 95% CI 2.07–5.97, P < .001). Notable univariate ECG predictors of death were APCs (OR 3.92, 95% CI 2.13–7.18, P < .001), an abnormal axis (OR 3.37, 95% CI 2.11–5.38, P < .001), a RBBB or IVB (OR 5.17, 95% CI 3.03–8.81, P < .001),

### Table 1. Baseline Clinical Characteristics of 756 Patients Hospitalized With Coronavirus Disease-19 With 12-Lead Electrocardiograms, Overall and by Survival Status

| Variable                        | All (N = 756) | Survived (n = 666) | Died (n = 90) | P Value   |
|---------------------------------|--------------|--------------------|--------------|-----------|
| Age (years)                     |              |                    |              | <.001     |
| Mean ± SD                       | 63.3 ± 16.0  | 61.1 ± 15.3        | 79.3 ± 11.8  |           |
| Median                          | 64.0         | 62.0               | 79.6         |           |
| Interquartile range             | 51.9–74.6    | 50.6–72.2          | 72.0–88.4    |           |
| Female sex                      |              |                    |              | .546      |
| Race                            |              |                    |              | <.001     |
| White                           | 296 (39.2%)  | 243 (36.5%)        | 53 (58.9%)   |           |
| Black                           | 89 (11.8%)   | 83 (12.5%)         | 6 (6.7%)     |           |
| Asian                           | 56 (7.4%)    | 47 (7.1%)          | 9 (10.0%)    |           |
| Other                           | 162 (21.4%)  | 154 (23.1%)        | 8 (8.9%)     |           |
| Not specified                   | 153 (20.2%)  | 139 (20.9%)        | 14 (15.6%)   | .001      |
| Smoker                          |              |                    |              |           |
| Never smoker                    | 554 (73.3%)  | 499 (74.9%)        | 55 (61.1%)   |           |
| Active smoker                   | 29 (3.8%)    | 28 (4.2%)          | 1 (1.1%)     |           |
| Former smoker                   | 173 (22.9%)  | 139 (20.9%)        | 34 (37.7%)   |           |
| Obesity (n = 716)               | 267 (37.3%)  | 241 (38.3%)        | 26 (30.2%)   | .185      |
| Coronary artery disease         | 109 (14.4%)  | 80 (12.0%)         | 29 (32.2%)   | <.001     |
| Heart failure                   | 55 (7.3%)    | 39 (5.9%)          | 16 (17.8%)   | <.001     |
| Stroke                          | 55 (7.3%)    | 38 (5.7%)          | 17 (18.9%)   | <.001     |
| Diabetes                        | 222 (29.4%)  | 194 (29.1%)        | 28 (31.1%)   | .792      |
| Hypertension                    | 427 (56.5%)  | 360 (54.1%)        | 67 (74.4%)   | <.001     |
| Pulmonary disease               | 142 (18.8%)  | 116 (17.4%)        | 26 (28.9%)   | .013      |
| Home oxygen use                 | 155 (20.5%)  | 135 (20.3%)        | 20 (22.2%)   | .771      |
| Renal disease                   | 72 (9.5%)    | 58 (8.7%)          | 14 (15.6%)   | .059      |
| Rheumatic disease               | 46 (6.1%)    | 41 (6.2%)          | 5 (5.6%)     | 1.000     |
| Active cancer                   | 42 (5.6%)    | 29 (4.4%)          | 13 (14.4%)   | <.001     |
| Immunosuppressed                | 25 (3.3%)    | 15 (2.3%)          | 10 (11.1%)   | <.001     |
| Hypoxemia within 3 hours        | 414 (54.8%)  | 343 (51.5%)        | 71 (78.9%)   | <.001     |
| Home medication number > median | 323 (42.7%)  | 264 (39.6%)        | 59 (66.3%)   | <.001     |

SD, standard deviation.

Hypoxemia within 3 hours defined by need for supplemental oxygen. Obesity if body mass index > 30 kg/m² or ≥ 27.5 kg/m² in Asians.
localized T-wave inversion (OR 2.65, 95% CI 1.50–4.70, \( P < .001 \)), and nonspecific repolarization abnormality (OR 1.86, 95% CI 1.18–2.92, \( P = .007 \)). Additional ECG findings associated with mortality were left and right ventricular hypertrophy and previous Q-wave myocardial infarction, but not heart rate or Bazett-corrected QT interval. Notably, localized ST elevation on presentation was a rare event \((n = 5)\) limiting any precise estimate of its association with death in our cohort.

In a forward-selection multivariable logistic regression analysis that included age and statistically significant univariate clinical predictors of mortality from Table 3, we found that CAD, stroke, pulmonary disease, immunosuppressed status, and hypoxemia within 3 hours of arrival increased the odds of death. In a model including age and electrocardiographic variables, APCs, RBBB or IVB, localized T-wave inversion, and nonspecific repolarization abnormality increased the odds of death. Finally, in a multivariable model incorporating age, clinical characteristics, and ECG variables, CAD, an immunosuppressed state, and hypoxemia were the clinical characteristics that remained associated with death. ECG variables that increased the odds of death remained APCs (OR 2.57, 95% CI 1.23–5.36, \( P = .01 \)), a RBBB or IVB (OR 2.61, 95% CI 1.32–5.18, \( P = .002 \)), localized T-wave inversion (OR 3.49, 95% CI 1.56–7.80, \( P = .002 \)), and nonspecific repolarization abnormality (OR 2.31, 95% CI 1.27–4.21, \( P = .006 \), Table 4).

### Discussion

This analysis of more than 750 patients with ECGs is the largest study of noninvasive diagnostic testing in patients with confirmed COVID-19 to date. Our findings revealed that APCs, RBBB/IVB, localized T-wave inversion, and nonspecific repolarization abnormalities were associated with an increased odds of death after accounting for age and other important clinical characteristics. ST segment elevation, previously described as an important complication of this disease,\(^9\) was rare on presentation. Our findings underscore the potential for ECGs to serve as a valuable tool to inform prognosis, even before many patients develop respiratory failure requiring invasive mechanical ventilation; and provide insights on myocardial processes at play.

APCs occurred in 7.7% of patients, and were associated with a 2.57-fold increased odds of death. Historically, the presence of APCs have correlated with increased left ventricular filling pressures, especially after acute myocardial infarction. Whether the presence of APCs in this clinical scenario is indicative of elevated filling pressures and/or increased myocardial stiffness is not known.\(^10\)–\(^12\) However, cytokine hypersecretion, a common finding in COVID-19, has also been linked to transient cardiac systolic and diastolic dysfunction.\(^13\) Additionally, our observation of T-wave inversions on ECG and the presence of CAD as risk factors for death, coupled with recent data showing that troponin elevations are common,\(^14\),\(^15\) suggest a potential role for subendocardial ischemia as well. Future studies examining the complex interplay between SARS-CoV-2 infection and cardiovascular perturbations like elevated filling pressures, left ventricular stiffness, and myocardial ischemia represent an important and fruitful area for future investigation.

### Table 2. Baseline Electrocardiographic Characteristics of 756 Patients Hospitalized With Coronavirus Disease-19 With 12-Lead Electrocardiograms, Overall and by Survival Status

| Variable                                      | All \((n = 756)\) | Survived \((n = 666)\) | Died \((n = 90)\) | \( P \) Value |
|-----------------------------------------------|------------------|------------------------|------------------|--------------|
| Heart rate (bpm)                              | 90 ± 18          | 90 ± 18                | 89 ± 24          | .553         |
| Bazett-corrected QT interval (ms)              | 449 ± 144        | 448 ± 153              | 459 ± 42         | .506         |
| Atrial fibrillation or flutter                | 42 (5.6%)        | 32 (4.8%)              | 10 (11.1%)       | .027         |
| Atrial premature contractions                 | 58 (7.7%)        | 40 (6.0%)              | 18 (20.0%)       | <.001        |
| Ventricular premature contractions            | 26 (3.4%)        | 20 (3.0%)              | 6 (6.7%)         | .138         |
| Atrioventricular block                        |                  |                        |                  | (.025)       |
| None                                          | 736 (97.4%)      | 652 (97.9%)            | 84 (93.3%)       |              |
| First degree                                   | 19 (2.5%)        | 14 (2.1%)              | 5 (5.6%)         |              |
| Third degree                                   | 1 (0.1%)         | 0 (0%)                 | 1 (1.1%)         |              |
| Abnormal axis                                  | 146 (19.3%)      | 110 (16.5%)            | 36 (40.0%)       | <.001        |
| Intraventricular conduction block             |                  |                        |                  | (.001)       |
| None                                          | 667 (88.2%)      | 605 (90.8%)            | 62 (68.9%)       |              |
| Right bundle branch block                     | 59 (7.8%)        | 40 (6.0%)              | 19 (21.1%)       |              |
| Left bundle branch block                      | 11 (1.5%)        | 10 (1.5%)              | 1 (1.1%)         |              |
| Nonspecific intraventricular block            | 19 (2.5%)        | 11 (1.7%)              | 8 (8.9%)         |              |
| Left ventricular hypertrophy                  | 117 (15.5%)      | 95 (14.3%)             | 22 (24.4%)       | (.019)       |
| Right ventricular hypertrophy                 | 30 (4.0%)        | 22 (3.3%)              | 8 (8.9%)         | .024         |
| Myocardial infarction age undetermined        | 105 (13.9%)      | 82 (12.3%)             | 23 (25.6%)       | .001         |
| Localized ST elevation (injury)               | 5 (0.7%)         | 4 (0.6%)               | 1 (1.1%)         | 1.000        |
| Localized T-wave inversion (ischemia)         | 80 (10.5%)       | 61 (9.2%)              | 19 (21.1%)       | .001         |
| Nonspecific repolarization abnormality        | 219 (29.0%)      | 182 (27.3%)            | 37 (41.1%)       | .010         |

Hypoxemia within 3 hours defined by need for supplemental oxygen. Obesity if body mass index \( \geq 30 \text{ kg/m}^2 \) or \( \geq 27.5 \text{ kg/m}^2 \) in Asians.
Table 3. Univariate Logistic Regression Prediction of Mortality in 756 Patients Hospitalized With COVID-19 With 12-Lead Electrocardiograms

| Variable                          | χ²   | P value | Odds Ratio | 95% CI |
|-----------------------------------|------|---------|------------|--------|
| **Clinical characteristics**      |      |         |            |        |
| Age (per 10 years)                | 81.7 | <.001   | 2.83       | 2.24–3.47 |
| Male sex                          | 0.5  | .471    | 1.19       | 0.75–1.89 |
| Race (relative to white)          | 20.1 | <.001   |            |        |
| Black                             | 6.0  | .014    | 0.33       | 0.14–0.80 |
| Asian                             | 0.1  | .741    | 0.88       | 0.41–1.90 |
| Other                             | 13.3 | <.001   | 0.24       | 0.11–0.52 |
| Not specified                     | 5.9  | .015    | 0.46       | 0.25–0.86 |
| Smoker                            | 13.5 | .001    |            |        |
| Active                            | 1.2  | .281    | 0.33       | 0.04–2.47 |
| Former                            | 11.6 | .001    | 2.26       | 1.42–3.61 |
| Obesity (n = 716)                 | 2.1  | .151    | 0.70       | 0.43–1.14 |
| **Coronary artery disease**       | 23.9 | <.001   | 3.48       | 2.11–5.74 |
| **Heart failure**                 | 15.0 | <.001   | 3.48       | 1.85–6.53 |
| **Stroke**                        | 18.1 | <.001   | 3.85       | 2.07–7.16 |
| **Diabetes**                      | 0.2  | .698    | 1.10       | 0.68–1.77 |
| Hypertension                      | 12.8 | <.001   | 2.48       | 1.51–4.07 |
| **Pulmonary disease**             | 6.7  | .010    | 1.93       | 1.17–3.17 |
| **Home oxygen use**               | 0.2  | .667    | 1.12       | 0.66–1.91 |
| **Renal disease**                 | 4.2  | .041    | 1.93       | 1.03–3.63 |
| **Rheumatic disease**             | 0.1  | .823    | 0.90       | 0.35–2.23 |
| **Active cancer**                 | 13.6 | <.001   | 3.71       | 1.85–7.44 |
| **Immunosuppressed**              | 15.8 | <.001   | 5.43       | 2.36–12.48 |
| **Hypoxia within 3 hours**        | 21.8 | <.001   | 3.52       | 2.07–5.97 |
| **Home medication number > median** | 21.3 | <.001   | 3.00       | 1.88–4.77 |
| Electrocardiographic characteristics |     |         |            |        |
| Heart rate (bpm)                  | 0.5  | .464    | 1.00       | 0.98–1.01 |
| Bazett-corrected QT interval (ms) | 0.4  | .533    | 1.00       | 0.99–1.01 |
| Atrial fibrillation or flutter     | 5.7  | .017    | 2.48       | 1.17–5.23 |
| Atrial premature contractions      | 19.4 | <.001   | 3.92       | 2.13–7.18 |
| Ventricular premature contractions | 3.0  | .081    | 2.31       | 0.90–5.91 |
| Atrioventricular block (any)      | 2.6  | .161    | 2.77       | 0.97–7.90 |
| Abnormal axis                     | 25.8 | <.001   | 3.37       | 2.11–5.38 |
| RBBB or IVB                       | 36.4 | <.001   | 5.17       | 3.03–8.81 |
| Left ventricular hypertrophy      | 6.1  | .013    | 1.95       | 1.15–3.30 |
| Right ventricular hypertrophy     | 6.0  | .014    | 2.86       | 1.23–6.62 |
| Myocardial infarction age undetermined | 11.1 | .001   | 2.45       | 1.44–4.14 |
| Localized ST elevation (injury)    | 0.3  | .581    | 1.86       | 0.21–16.82 |
| Localized T-wave inversion (ischemia) | 11.2 | .001   | 2.65       | 1.50–4.70 |
| Nonspecific repolarization abnormality | 7.2  | .007    | 1.86       | 1.18–2.92 |

CI, confidence interval; IVB, intraventricular conduction block; RBBB, right bundle branch block.

Hypoxemia within 3 hours defined by need for supplemental oxygen.

Obesity if body mass index ≥30 kg/m² or ≥27.5 kg/m² in Asians.

Non-specific repolarization abnormalities were also associated with death, an observation that has been made in the general population. However, the striking aspect of our observation is that it predicted death in a very short time.

Period—during hospitalization. Meanwhile, myocardial injury as defined by localized ST segment elevation was rare, occurring in just 5 patients, only 1 of whom died. ST elevation has been described in the setting of COVID-19; however, our data suggest that this is not a common finding on hospital presentation and suggest that other findings, which may otherwise be overlooked, merit greater attention up front by clinicians caring for patients with COVID-19.

RBBB/IVB was additionally associated with death, and may reflect a higher incidence of early right ventricular (RV) dysfunction in this population. Given the high rate of respiratory failure in a significant proportion of infected individuals, cardiac dysfunction secondary to increased afterload on the right ventricle (referred to as acute cor pulmonale) is not unexpected. Although ECG findings are insensitive for the detection of acute cor pulmonale, RBBB has been attributed to acute RV overload and distention in multiple studies of acute pulmonary embolism, with a higher frequency noted in cases with larger clot burdens.
mortality in patients with COVID-19, independent of all other echocardiographic parameters. To our knowledge, the present study is the first to show that RBBB at the time of presentation, often before the development of acute respiratory failure, is associated with worse survival. Importantly, the mechanisms of pulmonary vascular disease in COVID-19 are incompletely understood and may include early vascular endothelial injury, microcirculatory thrombi, and pulmonary venous thromboembolism. Therefore, the ECG may provide early evidence for an “RV at risk,” even before the onset of severe hypoxemia, pulmonary vasoconstriction, and overt clinical deterioration.

Our study has several strengths. First, clinical data and outcomes were manually abstracted from the electronic health record using a process that has previously shown high reliability. Comorbid conditions manually abstracted from different sections of a medical chart (and often include data from free-text sections of the chart) may be more comprehensive than automated data abstractions from the electronic health record which rely on structured fields and/or billing codes. Second, all ECGs were interpreted by experienced electrocardiographers, which has several advantages, including improved accuracy over automated interpretation. Our study also has several limitations. First, because the course of this disease is ongoing in our institution, data on death for all patients are inherently incomplete, which may bias the results. Of note, all patients had at least 2 weeks of outcome data. Additionally, we had incomplete laboratory data for the majority of patients included in our study. Markers of myocardial injury (namely, troponin and B-type natriuretic peptide) were obtained clinically at the discretion of the clinician caring for the patient, and often well after the initial presentation to the hospital, which is when these ECGs were obtained. Owing to concerns about confounding by indication, we did not include laboratory data in this analysis. Additionally, given that we did not have prior ECGs in all patients, we did not compare the presentation ECG with prior ECGs for the purpose of new findings, and this factor should be studied further. We did not formally assess the reliability of the 2 ECG readers. However, these readers review more than 100,000 ECGs per year at this institution and have historically been concordant in their interpretation. Finally, given the large number of variables analyzed, it is possible that some of the associations reported occurred by chance. However, our findings are consistent with emerging data that myocardial processes are an important prognostic indicator in COVID-19.

In conclusion, our data show that the ECG may be a useful prognostic tool in COVID-19, even among patients who are yet to require invasive mechanical ventilation. Our findings highlight that patients with ECG findings of both left sided heart disease (APCs, IVB, repolarization abnormalities) and right-sided disease (RBBB) have a higher odds of death. Localized ST segment elevation at presentation was rare.

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Supplementary materials
Supplementary material associated with this article can be found, in the online version, at doi:10.1016/j.cardfail.2020.06.005.

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