Electrocardiogram abnormalities and prognosis in COVID-19

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Background: COVID-19 is a major pandemic with potential cardiovascular complications. Few studies have focused on electrocardiogram (ECG) modifications in COVID-19 patients.

Method and results: We reviewed from our database all patients referred to our hospital for COVID-19 between January 1st, 2020, and December 31st, 2020. 669 patients were included and 98 patients died from COVID-19 (14.6%). We systematically analyzed ECG at admission and during hospitalization if available. ECG was abnormal at admission in 478 patients (71.4%) and was more frequently abnormal in patients who did not survive (88.8% vs. 68.5%, p < 0.001). The most common ECG abnormalities associated with death were left anterior fascicular block (39.8% vs. 20.0% among alive patients, p < 0.001), left and right bundle branch blocks (p = 0.002 and p = 0.02, respectively), STQ pattern (14.3% vs. 6.0%, p = 0.006). In multivariate analysis, at admission, the presence of left bundle branch block remained statistically related to death (OR = 3.82, 95% confidence interval (CI): 1.52–9.28, p < 0.01), as well as STQ pattern (OR = 3.17, 95% CI: 1.38–7.03, p < 0.01) and repolarization abnormalities (OR = 2.41, 95% CI: 1.40–4.14, p < 0.01).

On ECG performed during hospitalization, the occurrence of new repolarization abnormality was significantly related to death (OR = 2.72, 95% CI: 1.14–6.54, p = 0.02), as well as a new STQ pattern (OR = 13.23, 95% CI: 1.49–286.56, p = 0.03) and new supraventricular arrhythmia (OR = 3.8, 95% CI: 1.11–13.35, p = 0.03).
Coronavirus Disease 2019 (COVID-19) is a main pandemic infection that has hit the world with multiple waves (1). Its evolution through the years ahead remains uncertain (2), because of the emergence of new variants (3, 4), vaccination campaigns and innovative treatments (5).

Previous cardiovascular comorbidities seem to worsen the prognosis of the infection (6), but COVID-19 may cause several cardiovascular complications via different mechanisms (7). Systemic inflammation can destabilize vascular plaque, while viral illness increases cytokine activity, increasing cardiac demand, like influenza (8). Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) may also cause direct damage to the heart utilizing ACE2 receptors located within cardiac tissue (9). This infection is thereby associated with venous thromboembolic events (10–14), myocarditis (7, 15, 16), arrhythmias (14) and increased risk of acute myocardial infarction (17–20) and possible coronary tropism of the virus in thrombi (21, 22). All these complications may induce electrocardiographic abnormalities. Electrocardiogram (ECG) is a simple and broadly available exam which can be rapidly performed without exposing a large number of staff to the virus. Systematic standard ECG may be a useful screening tool for cardiovascular complications in patients presenting with COVID-19.

There are few published studies of ECG modifications related to COVID-19 (23–28). A retrospective cohort study of 756 patients comparing ECG abnormalities showed that both left- and right-sided heart disease in patients with COVID-19 have higher odds of death (26), but the confounding factors are not well-described.

A narrative review shows that up to 90% of critically ill patients have at least one ECG abnormality, including supraventricular tachycardia or ST modification, mainly related to cytokine storm, hypoxic injury, electrolyte abnormalities, plaque rupture, coronary spasm, microthrombi, or direct endothelial or myocardial injury (27). The objective of this study was to describe ECG abnormalities during COVID-19 and their impact on prognosis.

**Methods**

We reviewed from our database all patients referred to our hospital for COVID-19 between January 1st, 2020 and December 31st, 2020. All the cases of COVID-19 were proved by SARS-CoV-2 RT-PCR on nasopharyngeal swabs. We included all hospitalized patients [including in intensive care unit (ICU)] with an available ECG. Patients with ventricular pacing were excluded.

Baseline characteristics of the population were systematically recorded: demographic characteristics (sex, body mass index, age), habits (smoking status), medical background (diabetes, hypertension, dyslipidemia, ischemic heart disease, history of familial cardiovascular disease, heart failure, history of supraventricular arrhythmia, history of venous thromboembolic disease, known pulmonary hypertension, peripheral arterial disease, history of stroke, chronic obstructive pulmonary disease or asthma, chronic lung failure, i.e., requiring home oxygen therapy, chronic kidney disease, defined as glomerular filtration rate< 60 ml/kg/m², active cancer or immunosuppression), initial presentation (including time from symptoms onset), and hospitalization duration. We assessed several biomarkers at admission: leukocytes, C-reactive protein, hemoglobin, D-dimers, potassium, creatinine and troponin. We also recorded the main drugs that were administered to the patient (including heparin, oral anticoagulant drugs, antibiotics, immunotherapy, catecholamine, steroids) and major events that occurred during hospitalization, including pulmonary embolism, intensive care admission, extracorporeal life support, type of ventilation (non-invasive oxygen therapy, high-flow oxygen therapy, continuous positive airway pressure, mechanical ventilation).

Electrocardiograms at admission were systematically analyzed. We also assessed potential occurrence of new ECG abnormalities in patients with ECG performed during hospitalization. In case of multiple ECGs, we considered the first ECG done during hospitalization. The ECG interpretation was performed by two independent physicians blinded to clinical status and outcome. Any disagreement in interpretation between readers was resolved by consensus. The following ECG parameters were systematically assessed: (1) heart rate, (2) sinus rhythm status, (3) atrial and ventricular arrhythmias, (4) PR duration (in milliseconds), (5) atrioventricular block, (6) low voltage (defined as low amplitude of QRS complexes
Flowchart of the study.

Results

We included 697 patients hospitalized with confirmed COVID-19. Of these patients, 28 were excluded because of pacing (Figure 1), and 669 patients were included in the primary analysis. Baseline characteristics of the global population and according to vital status are presented in Table 1. One hundred and seventy-four patients (26.0%) were admitted to ICU. Ninety-eight patients (14.6%) died from COVID-19 during hospitalization. Mean age was 69.1 ± 17.2 years and was significantly older in deceased patients (80.9 ± 11.1 years vs. 67.1 ± 17.3 years, p < 0.001). Deceased patients more frequently had hypertension (67.3% vs. 45%, p < 0.001) and dyspnea (73.5% vs. 52.7%, p < 0.001). Mean leukocyte count was 8.2 ± 4.3 G/L and was statistically higher in the non-survival group (p = 0.001), as were CRP level (p < 0.001), D-dimers (p = 0.01), serum creatinine (p < 0.001), troponin (p = 0.03) and NT pro
TABLE 1 Baseline characteristics of the study population presenting with COVID-19.

| Clinical characteristics                  | Alive, n = 571 (85.4%) | Dead, n = 98 (14.6%) | P value |
|-------------------------------------------|-------------------------|----------------------|---------|
| Male                                      | 330 (57.8%)             | 62 (63.3%)           | 0.365   |
| Body mass index, kg/m²                    | 26 ± 5.6                | 25.9 ± 6.8           | 0.845   |
| Age, years                                | 67.1 ± 17.3             | 80.9 ± 11.1          | <0.001  |
| Smoker                                    | 44 (7.7%)               | 7 (7.1%)             | 0.990   |
| Diabetes mellitus                         | 105 (18.4%)             | 26 (26.5%)           | 0.082   |
| Hypertension                              | 257 (45%)               | 66 (67.3%)           | <0.001  |
| Dyslipidemia                              | 152 (26.6%)             | 37 (37.8%)           | 0.032   |
| History of familial cardiovascular disease | 6 (1.1%)                | 1 (1%)               | 0.610   |
| Coronary artery disease                   | 61 (10.7%)              | 20 (20.4%)           | 0.011   |
| Heart failure                             | 16 (2.8%)               | 11 (11.2%)           | <0.001  |
| Supraventricular arrhythmia               | 63 (11%)                | 20 (20.4%)           | 0.015   |
| History of pulmonary embolism or thrombosis | 31 (5.4%)              | 9 (9.2%)             | 0.223   |
| Pulmonary hypertension                     | 1 (0.2%)                | 0 (0%)               | 0.317   |
| Peripheral arterial disease               | 21 (3.7%)               | 12 (12.2%)           | <0.001  |
| History of stroke                         | 31 (5.4%)               | 14 (14.3%)           | 0.116   |
| Chronic obstructive pulmonary disease      | 77 (13.5%)              | 14 (14.3%)           | 0.957   |
| Chronic lung failure                      | 24 (4.2%)               | 3 (3.1%)             | 0.800   |
| Active cancer                             | 24 (4.2%)               | 17 (17.3%)           | <0.001  |
| Chronic kidney disease                    | 35 (6.1%)               | 14 (14.3%)           | 0.008   |

Initial symptoms

| Cough                                      | 275 (48.2%)             | 45 (45.9%)           | 0.763   |
| Dyspnea                                   | 301 (52.7%)             | 72 (73.5%)           | <0.001  |
| Chest pain                                | 60 (10.5%)              | 3 (3.1%)             | 0.032   |
| Syncope or faintness                      | 92 (16.1%)              | 14 (14.3%)           | 0.758   |
| Asthenia                                   | 238 (41.7%)             | 30 (30.6%)           | 0.051   |
| Anosmia or ageusia                        | 54 (9.5%)               | 4 (4.1%)             | 0.121   |
| Gastrointestinal symptoms                 | 174 (30.5%)             | 17 (17.3%)           | 0.011   |
| Time from symptom onset to hospitalization | 7 [4–10]               | 6 [3–10]             | 0.057   |

Laboratory data

| Leukocytes (G/L)                          | 8.0 ± 4.1               | 9.6 ± 5.2            | 0.001   |
| CRP (mg/L)                                | 77 [33–151]             | 127 [66–205]         | <0.001  |
| Hemoglobin (g/dL)                         | 13.3 ± 1.9              | 12.9 ± 2.2           | 0.022   |
| D-dimers (ng/mL)                          | 792 [702–1,320]         | 1,127 [806–1,958]    | 0.011   |

(Continued)

TABLE 1 (Continued)

| Alive, n = 571 (85.4%) | Dead, n = 98 (14.6%) | P value |
|------------------------|----------------------|---------|
| Serum potassium (mmol/L) | 4.2 ± 0.5            | 4.2 ± 0.5 | 0.656   |
| Serum creatinine (µmol/L) | 75 [73–77]          | 96 [90–106] | <0.001  |
| Cardiac troponin I at admission (µg/L) | [0.022–0.109]      | [0.034–0.455] | <0.001  |
| NT pro BNP (ng/L)      | 526 [150–1,895]      | 1,810 [632–6,433] | <0.001  |

Management

| Intensive care unit admission             | 132 (23.1)            | 42 (42.9)          | <0.001  |
| Oxygen therapy                           | 409 (71.6)            | 95 (96.9)          | <0.001  |
| High-flow nasal oxygen therapy           | 77 (13.5)             | 24 (24.5)          | 0.008   |
| Continuous positive airway pressure      | 7 (1.2)               | 4 (4.1)            | 0.104   |
| Mechanical ventilation                   | 30 (5.3)              | 25 (25.5)          | <0.001  |
| Hospitalization duration                 | 9 [5–17]              | 10 [5–17]          | 0.504   |

All results are presented as mean (%), mean ± SD, or median (25th to 75th percentiles).

BNP (p < 0.001). The median time from symptoms onset to admission was 7 days, without any statistical difference between the two groups (p = 0.057). Pulmonary embolism was diagnosed in 37 patients (5.5%), heart failure in 45 patients (6.7%), acute coronary syndrome in 10 patients (1.5%) and pericarditis in 2 patients (0.3%). No myocarditis was observed.

ECG characteristics at admission

The ECG characteristics at admission are presented in Table 2. All patients had an ECG at admission, and 169 patients (25.3%) had another ECG during hospitalization. An abnormal ECG at admission was observed in 478 patients (71.4%) and was more frequently present in patients who did not survive (88.8 vs. 68.5%, p < 0.001). An abnormal ECG was also more frequently present in patients admitted in ICU than in non-ICU patients (79.3 vs. 68.7%, p < 0.001). The most common abnormality associated with death was left anterior fascicular block (39.8 vs. 20.0%, p < 0.001). Left and right bundle branch blocks were also statistically associated with death (10.2 vs. 3.0%, p = 0.002 and 12.2 vs. 5.3%, p = 0.02, respectively), as were S1Q3 pattern (14.3 vs. 6.0%, p = 0.006) and non-specific repolarization abnormalities (30.6 vs. 14.4%, p < 0.001). Mean corrected QT interval was 439 ± 31 ms (446 ± 36 ms among non-survival patients vs. 435 ± 29 ms among patients who survived, p = 0.006).

Supplementary Table 1 provides univariate analysis for identifying variables at admission associated with death. In multivariate analysis (Table 3), the presence of left bundle...
TABLE 2 ECG characteristics in patients presenting with COVID-19.

| Feature                              | Alive, n = 571 (85.4%) | Dead, n = 98 (14.6%) | P-value |
|--------------------------------------|-------------------------|----------------------|---------|
| Abnormal ECG                         | 391 (68.5%)             | 87 (88.8%)           | <0.001  |
| Supraventricular arrhythmia          | 52 (9.1%)               | 14 (14.3%)           | 0.162   |
| PR duration, ms                      | 157 ± 29                | 168 ± 41             | 0.004   |
| Atroventricular block, first degree  | 42 (8.1%)               | 16 (19.3%)           | 0.003   |
| Atroventricular block, second degree | 0 (0%)                  | 0 (0%)               | -       |
| Abnormal ECG                          | 51 (9.9%)               | 9 (9.2%)             | 0.912   |
| Q wave                               | 55 (9.6%)               | 10 (10.2%)           | 0.994   |
| S1Q3 pattern                         | 34 (6.0%)               | 14 (14.3%)           | 0.006   |
| QRS duration, ms                     | 51 (8.9%)               | 23 (23.5%)           | <0.001  |
| Complete right bundle branch block    | 31 (5.4%)               | 12 (12.2%)           | 0.020   |
| Incomplete right bundle branch block  | 53 (9.3%)               | 7 (7.1%)             | 0.622   |
| Left bundle branch block             | 17 (3.0%)               | 10 (10.2%)           | 0.002   |
| Abnormal QRS axis                    | 125 (21.9%)             | 46 (46.9%)           | <0.001  |
| QRS axis, degrees                    | 16 ± 48                 | −6 ± 52              | <0.001  |
| Left anterior fascicular block       | 114 (20.0%)             | 39 (39.8%)           | <0.001  |
| QRS fragmentation                    | 5 (0.9%)                | 2 (2.0%)             | 0.61    |
| Right ventricular hypertrophy        | 3 (0.5%)                | 1 (1.0%)             | 0.903   |
| Left ventricular hypertrophy         | 7 (1.2%)                | 2 (2.0%)             | 0.863   |
| Negative T waves                     | 74 (13.0%)              | 19 (19.4%)           | 0.123   |
| Non-specific repolarization abnormalities | 82 (14.4%)            | 30 (30.6%)           | <0.001  |

**TABLE 3** Multivariate analysis for identifying variables at admission independently associated with death.

| Variables                        | OR    | 95% CI          | P-value |
|----------------------------------|-------|-----------------|---------|
| Model 1                          |       |                 |         |
| Age (per 10 years)               | 1.98  | 1.62–2.46       | <0.001  |
| Oxygen saturation (<92 vs. ≥92%) | 0.54  | 0.32–0.90       | 0.01    |
| Dyspnea                          | 3.25  | 1.92–5.66       | <0.001  |
| Active cancer                     | 5.94  | 2.73–12.96      | <0.001  |
| Abnormal ECG at admission         | 2.28  | 1.15–4.92       | 0.02    |
| Model 2                          |       |                 |         |
| Age (per 10 years)               | 1.81  | 1.46–2.28       | <0.001  |
| Oxygen saturation (<92 vs. ≥92%) | 0.47  | 0.28–0.81       | <0.01   |
| ECG at admission                  |       |                 |         |
| S1Q3 pattern                      | 3.17  | 1.38–7.03       | <0.01   |
| Left bundle branch block          | 3.82  | 1.32–9.28       | <0.01   |
| Repolarization abnormalities      | 2.41  | 1.40–4.14       | <0.01   |

EKG, electrocardiogram; CL, confidence interval.

Characteristics of ECG performed after admission

The characteristics of ECG performed after admission in 169 patients are presented in Table 4. An abnormal ECG was observed in 135 patients (79.9%) and 72 (42.9%) had a modified ECG as compared to the ECG at admission. The most frequent new ECG abnormality was T wave inversion (20 patients, 11.8%). Thirteen patients (7.7%) had a new supraventricular arrhythmia. Four patients (1.8%) presented a new S1Q3 pattern. Among the 169 patients who had a new ECG during hospitalization, 13 had a combination of hydroxychloroquine (HCQ) and azithromycin (AZT) regimen. The mean QTc interval was 443 ± 24 in patients under HCQ/AZT combination regimen vs. 437 ± 32 in patients without such treatment (p = 0.29). No patient in the HCQ/AZT group had a new prolonged QTc nor a QTc >500 ms. In multivariate analysis (Table 5), the occurrence of the following ECG parameters was associated with death: new repolarization abnormality (OR = 2.72, 95% CI: 1.14–6.54, p = 0.02), new S1Q3 pattern (OR = 13.23, 95% CI: 1.49–286.56, p = 0.03) and new-onset supraventricular arrhythmia (OR = 3.8, 95% CI: 1.11–13.35, p = 0.03).

**Discussion**

In the present study, we evaluated the ECG characteristics of patients hospitalized with COVID-19. The main results are: (1) in a large cohort of COVID-19 patients, an abnormal ECG was observed in 478 patients (71.4%) and was more frequently described in patients who did not survive; (2) left bundle branch block, S1Q3 pattern and repolarization abnormalities at admission were associated with death; (3) new repolarization abnormality, new S1Q3 pattern and new-onset supraventricular arrhythmia during hospitalization were associated with death.

It is now well-known that ECG is modified by systemic inflammation, which may be consecutive to COVID infection...
or any of its complications (6, 8). In previous studies, an abnormal ECG was observed in between 47 and 93% of COVID-19 patients (23, 24, 26–28). In our study, 71.4% of patients presented with ECG abnormalities: 68.7% in patients admitted in medical wards and 79.3% in ICU patients. To our knowledge, no study reported on the proportion of ECG abnormalities in non-ICU COVID-19 patients. Even when these last patients presented with less severe COVID-19 involvement, most of them had ECG abnormalities and physicians should be aware that ECG should be systematically performed for a prognostic assessment. The observed differences between studies may be explained by different study populations, with a high prevalence of ECG abnormalities in the ICU. About one quarter of patients were admitted in ICU in the present study, which included all consecutive patients hospitalized in our university hospital during 2020.

In our study, CRP was not associated with ECG abnormality \( (p = 0.96) \), but was associated with new-onset supraventricular arrhythmia \[ \text{mean CRP level was} \ 163.4 \text{ mg/L in the group of patients with new-onset supraventricular arrhythmia vs.} \ 107.2 \text{ mg/L in the group of patients without new-onset supraventricular arrhythmia} \ (p = 0.04) \]. This is consistent with previous studies, which identified systemic inflammation caused by COVID-19 as a key factor in arrhythmogenesis (14, 30).

In the present study, new-onset supraventricular arrhythmia was associated with death in multivariate analysis which is consistent with previous studies (24, 26, 31). In our study, no significant difference in QTc duration was found between patients with HCQ/AZT combination therapy and patients without treatment. This is in contradiction with the study by Bernardini et al. (32); where the HCQ/AZT combination therapy caused a significantly increase of QT interval compared to HCQ alone or no treatment group. This may be due to the fact that in our institution, we prescribed HCQ/AZT combination regimen during a limited period (04/06/2020 to 04/16/2020) in a limited number of patients \( (n = 13) \) and ECG was not systematically performed. Other ECG abnormalities that we describe in our study are consistent with the literature (26–28, 33). Interestingly, we found that left bundle branch block, S1Q3 pattern and repolarization abnormalities were independently associated with death in cases of COVID-19.

ECG abnormalities may be associated with death by many mechanisms. Systemic inflammation can increase cardiac demand, and destabilize vascular plaque, resulting in myocardial infarction (17–20), and thereby cause T wave inversion or non-specific repolarization abnormalities, which can worsen the prognosis (8). In a postmortem study (21), the most common pathological cause of myocyte necrosis in patients with COVID-19 infection was microthrombi. Indeed, only 3 patients in this study had a ST-elevation myocardial infarction, supporting the hypothesis of intra-coronal microthrombi rather than macrothrombus. Another mechanism of death

| TABLE 4 New ECG abnormalities observed during hospitalization, \( n = 169 \). |
|-----------------|-----------------|-----------------|-----------------|
| Alive, \( n = 135 \) | Dead, \( n = 34 \) | \( P \) value |
|-----------------|-----------------|-----------------|
| Abnormal ECG | 101 (74.8%) | 34 (100%) | 0.002 |
| Modified ECG | 50 (37.3%) | 22 (64.7%) | 0.007 |
| New supraventricular arrhythmia | 6 (4.4%) | 7 (20.6%) | 0.005 |
| New atrioventricular block | 2 (1.5%) | 0 (0%) | 1.000 |
| New low voltage | 2 (1.5%) | 1 (2.9%) | 0.493 |
| New Q wave | 2 (1.5%) | 2 (5.9%) | 0.181 |
| New S1Q3 pattern | 1 (0.7%) | 3 (8.8%) | 0.026 |
| New QRS > 120 ms | 1 (0.7%) | 2 (5.9%) | 0.103 |
| New right bundle branch block | 0 (0%) | 2 (5.9%) | 0.040 |
| New incomplete right bundle branch block | 2 (1.5%) | 1 (2.9%) | 0.493 |
| New left bundle branch block | 0 (0%) | 0 (0%) | NA |
| New non-specific bundle block | 0 (0%) | 0 (0%) | NA |
| New abnormal QRS axis | 5 (3.7%) | 2 (5.9%) | 0.629 |
| New left anterior fascicular block | 3 (2.2%) | 2 (5.9%) | 0.264 |
| New right ventricular hypertrophy | 1 (0.7%) | 0 (0%) | 1.000 |
| New left ventricular hypertrophy | 0 (0%) | 0 (0%) | NA |
| New negative T wave | 9 (6.7%) | 11 (32.4%) | <0.001 |
| New non-specific repolarization abnormalities | 10 (7.4%) | 11 (32.4%) | 0.696 |
| New prolonged QTc | 13 (9.6%) | 6 (17.6%) | 0.308 |
| New premature atrial complexes | 5 (3.7%) | 4 (11.8%) | 0.082 |
| New premature ventricular complexes | 3 (2.2%) | 3 (8.8%) | 0.097 |

ECG, electrocardiogram; QTc, corrected QT interval. All results are presented as value (%).

| TABLE 5 Summary of multivariate analysis for identifying variables independently associated with death among patients who had a second ECG during hospitalization, \( n = 169 \). |
|-----------------|-----------------|-----------------|-----------------|
| Variables | OR 95% CI | \( P \) value |
|-----------------|-----------------|-----------------|
| Model A | | |
| Age (per 10 years) | 1.76 | 1.26–2.55 | 0.002 |
| Modified ECG (as compared to baseline) | 3.56 | 1.60–8.34 | 0.002 |
| Model B | | |
| Age (per 10 years) | 1.81 | 1.41–2.29 | <0.001 |
| New negative T wave | 2.72 | 1.14–6.54 | 0.02 |
| New S1Q3 pattern | 13.23 | 1.49–286.56 | 0.03 |
| New supraventricular arrhythmia | 3.80 | 1.11–13.35 | 0.03 |

Legend:
- ECG: electrocardiogram
- CI: confidence interval
- OR: odds ratio
- Model A: adjusted for age (per 10 years), and modified ECG.
- Model B: adjusted for age (per 10 years), new negative T wave, new S1Q3 pattern, and new supraventricular arrhythmia.

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during COVID-19 is pulmonary embolism. It is now well-established that COVID-19 significantly increases the risk of pulmonary embolism (11, 34). In our study, SIQ3 pattern was statistically associated with death. This SIQ3 pattern is not pathognomonic for pulmonary embolism, but the occurrence of this ECG parameter may be helpful for the risk stratification of COVID-19 patients.

Potential limitations of the present study merit consideration. First, we recruited patients presenting with COVID-19 from the first wave and we did not study the ECG characteristics associated with new variants. Even if the prognosis of COVID-19 caused by these new variants is better as compared to the first wave (4), the most recent waves were accompanied by great contagiousness and many hospitalizations (3, 4). Second, in our study, ECG was not systematically repeated during hospitalization, and we analyzed at least two ECG during hospitalization in 169 selected patients, leading to a potential bias of interpretation. However, experts blinded to clinical data and outcome interpreted the ECG, and the occurrence of new ECG abnormalities was associated with death and was consistent with the results of ECG at admission. Finally, the low number of patients presenting with new SIQ3 pattern during hospitalization does not allow to definitively conclude on the prognostic impact of SIQ3 pattern.

Conclusion

The presence of abnormal ECG during COVID-19 is frequent. Several ECG abnormalities such as left bundle branch block, SIQ3 pattern, repolarization abnormalities and supraventricular arrhythmia are associated with death as well as the occurrence of ECG abnormalities during hospitalization. Clinicians should be aware of the usefulness of ECG for risk stratification during COVID-19.

Data availability statement

The raw data supporting the conclusions of this article will be made available by the authors, without undue reservation.

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