Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

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Prognostic value of right ventricular strain pattern on ECG in COVID-19 patients

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ABSTRACT

Objective: COVID-19 spread worldwide, causing severe morbidity and mortality and this process still continues. The aim of this study to investigate the prognostic value of right ventricular (RV) strain in patients with COVID-19.

Methods: Consecutive adult patients admitted to the emergency room for COVID-19 between 1 and 30 April were included in this study. ECG was performed on hospital admission and was evaluated as blind. RV strain was defined as in the presence of one or more of the following ECG findings: complete or incomplete right ventricular branch block (RBBB), negative T wave in V1-V4 and presence of S1Q3T3. The main outcome measure was death during hospitalization. The relationship of variables to the main outcome was evaluated by multivariable Cox regression analysis.

Results: A total of 324 patients with COVID-19 were included in the study; majority of patients were male (187, 58%) and mean age was 64.2 ± 14.1. Ninety-five patients (29%) had right ventricular strain according to ECG and 66 patients (20%) had died. After a multivariable survival analysis, presence of RV strain on ECG (OR: 4.385, 95% CI: 2.226–8.638, $p < 0.001$), high-sensitivity troponin I (hs-TnI), d-dimer and age were independent predictors of mortality.

Conclusion: Presence of right ventricular strain pattern on ECG is associated with in hospital mortality in patients with COVID-19.

1. Introduction

The new coronavirus (SARS-CoV-2), which emerged towards the end of 2019, turned into a pandemic and caused serious effects on health and economy systems all over the world [1,2]. While some cases lead to asymptomatic or mild symptoms, in some cases the infection may progress to the lower respiratory tract, resulting in pneumonia, acute respiratory distress syndrome (ARDS) and, in the last stage, multiple organ failure, resulting in death [3,4]. The most important cause of mortality in COVID-19 patients is lung involvement. The virus infects cells, especially by connecting to the mainly expressed angiotensin converting enzyme-2 (ACE-2) receptor in lung tissue [5,6]. It is thought that COVID-19 infection harms many organs with direct and/or excessive immune response [4]. Many studies have reported that cardiac involvement is a sign of poor prognosis [3,4,7]. In a study examining right ventricular (RV strain) by echocardiography in COVID-19 patients, strain values were shown to be associated with mortality [8].

Electrocardiography (ECG) is a bedside diagnosis and fast diagnostic method. Due to the arrhythmic effects of drugs used in treatment in patients diagnosed with COVID-19, ECG is used as part of the baseline assessment [9]. Some ECG parameters, such as the right ventricular strain pattern, have been associated with obstruction of the pulmonary circulation and overload of the right ventricular pressure [10-12]. Prognostic significance of right ventricular strain on ECG has been demonstrated in patients with pulmonary embolism [11,13]. To our knowledge no

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previous studies investigated the prognostic significance of RV strain detected in ECG in patients with COVID-19.

The aim of this study is to evaluate the relationship between RV strain pattern and mortality in ECG in COVID-19 patients.

2. Methods

For this single-center study, we retrospectively identified all consecutively hospitalized patients between April 1 and April 30, 2020 diagnosed with COVID-19 by real time reverse transcription-polymerase chain reaction (RT-PCR). Patients with known cardiomyopathy, pulmonary hypertension, history of heart failure, history of severe valvular disease, a prior documented episode of pulmonary embolism, and a history of severe chronic obstructive pulmonary disease (COPD) were excluded. Based on these criteria, 61 patients were excluded from the study and thus a total of 324 patients were included in the analysis. Demographic characteristics of all patients such as gender, age, smoking, known hyperlipidemia, hypertension, diabetes, were recorded from medical records. This research presented no more than minimal risk to participants, thus informed consents were not obtained from individuals.

Laboratory findings; creatinine, sodium, potassium, glucose, high-sensitivity troponin I (hs-TnI), d-dimer, hemoglobin, white blood cells (WBC), procalcitonin and C-reactive protein (CRP) levels were recorded, which were analyzed from the blood samples collected from the patient on admission. The study complies with the Declaration of Helsinki, and the trial protocol was approved by the local ethics committee and Ministry of Health.

2.1. Electrocardiographic evaluation

A 12-lead admission ECG was obtained on hospital admission from all patients before any treatment was started. All ECGs (filter range 0.5 Hz to 150 Hz, AC filter 60 Hz, 25 mm/s, 10 mm/mV) were analyzed by 2 independent cardiologist according to the modified Minnesota criteria, who were blinded to the study design and clinical data. ST depressions, at least 1 mm or more horizontal and/or downsloping ST depression (in DI, DII, aVL, aVF, V1-V6 derivations) seen after point J was considered. Right ventricular branch block (RBBB), defined as broad QRS > 120 ms, rSR' pattern in V1–3 ('M-shaped' QRS complex), wide, slurred S wave in the lateral leads (I, aVL, V5–6). Acute right ventricular strain was defined when at least one of the following patterns was found on ECG: [1] presence of S1Q3T3; [2] presence of complete or incomplete RBBB; [3] T wave inversions in the precordial leads (V1-V3) [14-16]. S1Q3T3 subtypes are S1Q3, S1rSr'3 and S1S2S3. $T_{NEG}V_2$ defined as T wave inversion in V2 or V3. Clockwise rotation of the QRS vector in the precordial leads (CLOCKROT) defined as R = S in V4, V5 or V6. The criteria for the diagnosis of Qr in V1 were the presence of a prominent Q wave of ≥0.2 mV and a ventricular depolarisation <120 ms. $ST_{POS}V_1$ was defined as ST elevation in V1 ≥ 0.1 mV.

According to the World Health Organization (WHO) interim guidance, the diagnosis of COVID-19 is based on real-time RT-PCR test. SARS-CoV-2 RNA was detected by real-time RT-PCR method in the Public Health Microbiology Reference Laboratory of the Ministry of Health. Acute cardiac injury was defined as hs-TnI serum levels above the 99th percentile upper reference limit [17]. Systemic inflammatory response syndrome (SIRS) score is the systemic response of the organism to a variety of severe clinical situations. SIRS is defined by two or more of the following conditions: body temperature > 38 °C or < 36 °C; heart rate > 90 beats per minute; respiratory rate > 20 breaths per minute or PaCO2 < 32 mmHg; and WBC count >12,000/cu mm, < 4000/cu mm, or > 10% immature (band) forms [18]. The primary end point was a composite of death for any cause during in-hospital stay. Patients were compared according to the presence of RV strain.

2.2. Statistical analysis

All statistical tests were conducted using the Statistical Package for the Social Sciences 19.0 for Windows (SPSS Inc., Chicago, IL, USA). The Kolmogorov-Smirnov test was used to analyze normality of the data. Continuous data are expressed as mean ± SD, and categorical data are expressed as percentages. Fisher Exactor/and Chi-square test were used to assess differences in categorical variables between groups. Student's *t*-test or Mann Whitney *U* test was used to compare unpaired samples as needed. The univariate effects of type of age, gender, hypertension (HT), diabetes mellitus (DM), creatinine, d-dimer, CRP, procalcitonin, hs-TnI, RV strain, heart rate, ST depression, T wave inversion and ST elevation in V1 on ECG in COVID-19 patients was investigated using the log rank test. The possible factors identified with univariate analyses were further entered into the Cox regression analysis, with backward selection, to determine independent predictors of mortality. The proportional hazards assumption and model fit was assessed by means of residual (Schoenfeld and Martingale) analysis. Cumulative survival curves were derived according to the Kaplan-Meier method and differences between curves were analyzed on log-rank statistics. Significance was assumed at a 2-sided $p < 0.05$.

3. Results

Sixty-six of 324 COVID-19 patients included in the study are the non-survivor group and their clinical and demographic characteristics are shown in Table 1. When patients are grouped as the non-survivor group and the survivor group; while there was no statistically significant difference between the groups in terms of gender, the average

Table 1
Demographic and clinical characteristics of patients non-survivor and survivor patients.

Characteristic	Non-survivor ($n = 66$)	Survivor ($n = 258$)	*p*
Age (years)	67.9 ± 13.3	60.4 ± 14.9	<0.001
Male gender, n(%)	40 (60%)	147 (57%)	0.594
Body mass index (kg/m^2)	27.2 ± 6.2	26.8 ± 5.4	0.554
Chronic medical illness – n (%)			
Hypertension	37 (56%)	129 (50%)	0.379
Diabetes mellitus	18 (27%)	66 (25%)	0.780
Hyperlipidemia	12 (18%)	37 (14%)	0.437
Smoking	33 (50%)	115 (44%)	0.430
Vital signs and symptoms on admission			
Body temperature, °C	37. 7 ± 0.9	37.1 ± 0.7	<0.001
Respiration rate, breaths/min	25.2 ± 5.1	20. 2 ± 1.6	<0.001
Systolic blood pressure (mmHg)	113.5 ± 19.5	126. 4 ± 16.5	<0.001
VCough	48 (73)	123 (48)	<0.001
Shortness of breath	51 (77)	56 (22)	<0.001
Laboratory findings on admission hospital			
Hemoglobin(g/dl)	11.3 ± 2.2	12.2 ± 1.9	0.003
White blood cell (10^3/μl)	9 (6–12)	6 (4–9)	<0.001
Creatinine (mg/dl)	1.1 (0.7–1.7)	0.8 (0.6–1.0)	<0.001
Sodium (mmol/L)	135.1 ± 4.7	137.6 ± 4.4	<0.001
Potassium (mmol/L)	4.5 ± 0.7	4.0 ± 0.5	<0.001
Glucose (mg/dL)	157.8 ± 72.2	131.6 ± 60.2	0.003
C-reactive protein (mg/L)	117 (32–152)	46 (15–111)	<0.001
Procalcitonin (ng/mL)	0.2 (0.1–1.0)	0.1 (0.1–0.3)	<0.001
hs-troponin I (pg/ml)	49 (14–389)	7 (4–20)	<0.001
D-dimer (ng/mL)	560 (131–1015)	92 (4–155)	<0.001
Complications and clinical outcome			
SIRS score	2.1 ± 1.1	0.8 ± 0.7	<0.001
Hospital length of stay, days	10 (7–14)	11 (7–15)	0.556
Hospitalization rate of ICU, n(%)	56 (85%)	41 (15%)	<0.001
Discharged, n(%)	–	197 (76%)	
Mechanical ventilation, n(%)	56 (85%)	5 (2%)	<0.001
Oxygen requirements, n(%)	10 (15%)	213 (82%)	<0.001

Abbreviations: hs-troponin I, high-sensitive troponin I; SIRS, Systemic Inflammatory Response Syndrome; ICU, intensive care unit.

Table 2
Electrocardiographic characteristics of COVID-19 patients.

	Non-survivor ($n = 66$)	Survivor ($n = 258$)	p
Heart rate, per minute	96.8 ± 20.7	90.1 ± 17.6	0.008
HR >100 per minute, n(%)	29 (43%)	70 (27%)	0.008
RV strain, n(%)	36 (54%)	59 (22%)	<0.001
RBBB, n(%)	10 (15%)	16 (6%)	0.017
ST depression (>1 mm), n (%)	31 (47%)	63 (24%)	<0.001
T wave inversion, n(%)	31 (47%)	73 (28%)	0.004
Incomplete RBBB, n(%)	14 (21%)	21 (8%)	0.002
S1Q3T3 and subtypes, n(%)	17 (25%)	33 (12%)	0.009
T_{neg} in V2, n(%)	13 (19%)	29 (11%)	0.063
T_{neg} in D2D3AVF, n(%)	12 (18%)	39 (15%)	0.542
P pulmonale, n(%)	6 (9%)	11 (4%)	0.117
CLOCKROT, n(%)	21 (31%)	45 (17%)	0.010
R dominans in V1, n(%)	6 (9%)	10 (4%)	0.080
QR in V1, n(%)	8 (12%)	17 (6%)	0.133
$ST_{POS}V_1$, n(%)	9 (13%)	12 (4%)	0.008
LBBB, n(%)	2 (3%)	7 (2%)	0.889

Abbreviations: HR, heart rate; RV, right ventricular; RBBB, right bundle branch block; T_{neg}, T wave inversion; CLOCKROT, Clockwise rotation of the QRS vector in the precordial leads; ST_{POS}, ST elevation; LBBB, left bundle branch block.

age was higher than the non-survivor group. Forty of 66 patients the non-survivor group were male (60%), while 147 of 258 patients the survivor group were male (57%). While the average age of patients the non-survivor group was 67.9 ± 13.3, the average age of patients the survivor group was 60.4 ± 14.9 and there was a statistically significant difference ($p < 0.001$). Body temperature and respiration rates were significantly higher in the non-survivor group ($p < 0.001$). Systolic blood pressure was lower in the non-survivor group ($p < 0.001$). SIRS score differed significantly between the non-survivor and survivor groups ($p < 0.001$).

When laboratory tests were compared in both groups, WBC, creatinine, sodium, potassium, glucose, CRP, procalcitonin, hs-TnI and d-dimer values were found to be statistically significantly higher in the non-survivor group, respectively, while hgb levels were found to be statistically significantly lower. While the length of hospital stay was similar in both groups, ICU hospitalization rates were significantly higher in the non-survivor group (56 (85%) / 41 (15%), $p < 0.001$) (Table 1).

Table 3
Demographic and clinical characteristics of patients with and without RV strain

Characteristic	RV strain(+) ($n = 95$)	RV strain(−) ($n = 229$)	p
Age (years)	61.8 ± 14.4	62.0 ± 15.1	0.942
Male gender, n(%)	55 (57%)	132 (57%)	0.967
Body mass index (kg/m^2)	27.0 ± 6.0	26.9 ± 5.8	0.759
Chronic medical illness – n (%)			
Hypertension	49 (51%)	117 (51%)	0.936
Diabetes mellitus	24 (25%)	60 (26%)	0.861
Hyperlipidemia	17 (17%)	32 (14%)	0.370
Smoking	46 (48%)	102 (44%)	0.523
Laboratory findings on admission hospital			
Hemoglobin(g/dl)	11.8 ± 2.0	12.1 ± 2.0	0.180
White blood cell (10^3/μl)	7 (5–10)	7 (4–9)	0.451
Creatinine (mg/dl)	0.9 (0.6–1.4)	0.8 (0.6–1.0)	0.234
Sodium (mmol/L)	136.7 ± 5.1	137.3 ± 4.4	0.291
Potassium (mmol/L)	4.2 ± 0.7	4.1 ± 0.6	0.222
Glucose (mg/dL)	139.4 ± 68.1	135.9 ± 61.8	0.657
C-reactive protein (mg/L)	63 (19–140)	55 (18–128)	0.930
Procalcitonin (ng/mL)	0.1 (0.1–0.3)	0.1 (0.1–0.3)	0.361
hs-troponin I (pg/ml)	11 (6–64)	8 (5–32)	0.036
D-dimer (ng/mL)	453 (33–985)	84 (7–172)	<0.001
Complications and clinical outcome			
Hospital length of stay, days	10 (7–15)	11 (7–15)	0.616
Hospitalization rate of ICU, n(%)	39 (41%)	58 (25%)	0.005
Death, n(%)	36 (38%)	30 (13%)	<0.001

Abbreviations: RV, right ventricular; hs-troponin I, high-sensitive troponin I; ICU, intensive care unit.

Table 4
Univariate and multivariable cox regression analysis on the risk factors associated mortality in patients with COVID-19.

Variable	Univariate			Multivariate		
	HR	95%CI	p	HR	95%CI	p
Age	1.038	1.017–1.059	<0.001	1.044	1.019–1.071	0.001
Gender	0.861	0.496–1.495	0.594			
Hypertension	0.784	0.938–3.092	0.380			
Diabetes mellitus	0.917	0.498–1.168	0.780			
Creatinine	1.513	1.220–1.877	<0.001	1.022	0.992–1.053	0.148
D-dimer	1.002	1.001–1.003	<0.001	1.002	1.001–1.003	0.026
CRP	1.006	1.003–1.009	<0.001	0.919	0.768–1.100	0.358
Procalcitonin	1.080	0.984–1.186	0.107			
Hs-TnI	1.002	1.001–1.003	0.018	1.002	1.001–1.003	0.041
RV-strain	4.047	2.301–7.121	<0.001	4.385	2.226–8.638	<0.001
Heart rate	1.016	1.003–1.028	0.013	1.008	0.996–1.021	0.176

Abbreviations: CRP, C-reactive protein; hs-TnI, high sensitive troponin I; RV, right ventricular.

The electrocardiographic features of patients were compared in non-survivor and survivor groups. Heart rate, RV strain pattern, RBBB, ST depression, T wave inversion, ST elevation, S1Q3T3, CLOCKROT and ST elevation in V1 were statistically significantly higher in the non-survivor group than in the survivor group ($p < 0.05$)(Table 2).

The patients were then grouped according to the presence of RV strain on the ECG. In 95 of 324 COVID-19 patients included in the study, RV strain was detected by electrocardiography and its clinical and demographic characteristics are shown in Table 3. Hs-TnI and d-dimer levels were found statistically high in the group with RV strain. Hospital stay was similar in both groups, while ICU hospitalization rates were significantly higher in the non-survivor group (39 (41%) / 58 (25%), $p = 0.005$). On the contrary, the discharge rates were higher in the group without RV strain (46 (48%) / 151 (65%), $p = 0.003$) (Table 3).

Cox regression analysis was evaluated by univariate and multivariable analyzes that predicted mortality development. Age, d-dimer, hs-TnI and RV strain values were found to be statistically by multivariable analysis significant independent predictors in terms of predicting mortality (Age HR: 1.044, $p = 0.001$; d-dimer HR: 1.002, $p = 0.026$; hs-TnI HR: 1.002, $p = 0.041$; RV strain HR: 4.385, $p < 0.001$)(Table 4).

Death occurred in 36 (38%) patients with RV strain and 30 (13%) patients without RV strain with electrocardiography. Survival rates were assessed by Kaplan-Meier curves and there was statistically significant difference (log-rank $p < 0.001$; Fig. 1).

The interobserver concordance rate for RV strain was 97%. In case of disagreement, the final diagnosis was achieved by mutual agreement. The intraobserver concordance rate was 98%.

4. Discussion

In the present study, prognostic significance of RV strain pattern on admission ECG were investigated in patients with COVID-19. The main findings of our study are as follows:

(i) RV strain, tachycardia and S1Q3T3 in ECG were observed more frequently in non-survivor than survivors patients.
(ii) Hs-TnI and d-dimer levels were higher and mortality rates were more frequently detected in patients with RV strain.
(iii) After a multivariable survival analysis, presence of right ventricular strain on ECG, hs-TnI, d-dimer and age were independent predictors of mortality.

COVID-19 spread worldwide, causing serious morbidity and mortality [1]. The disease has a clinical spectrum that can progress

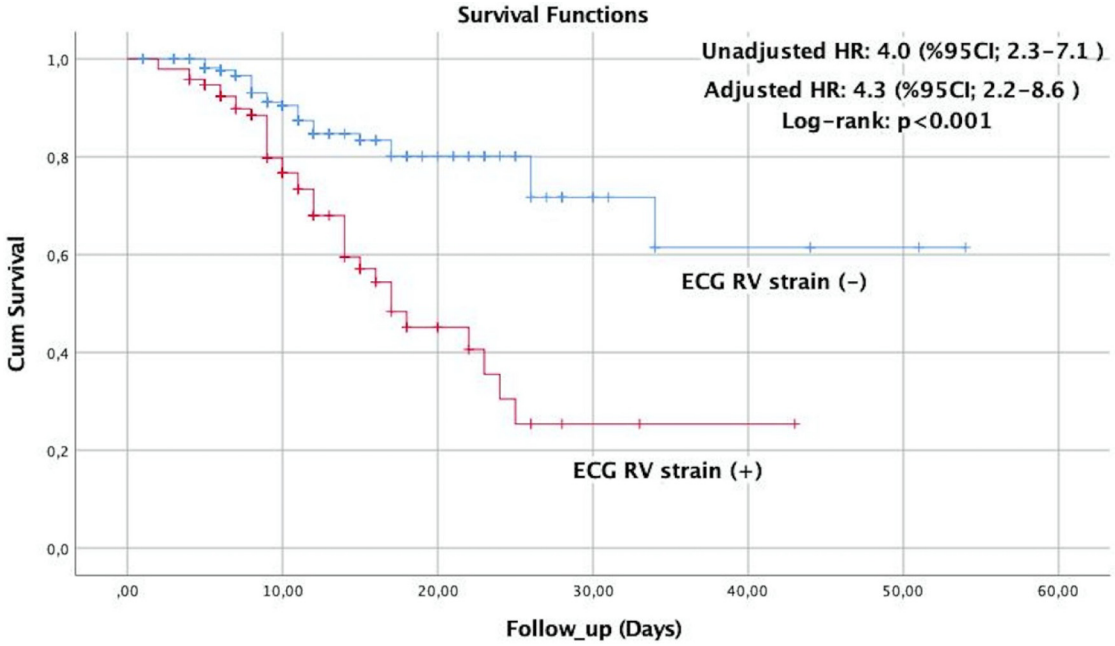

Fig. 1. In-hospital follow-up of patients with COVID-19 based on right ventricular strain pattern at presentation.

asymptomatically, which can lead to multiple organ failure and may result in death. Although it mainly affects the respiratory tract, other systems, including the cardiovascular system, are also affected by this infection. In addition to acute respiratory distress syndrome (ARDS) and type 1 respiratory failure, acute cardiac injury and heart failure are the most common complications [3,5,6]. It has been reported that SARS-CoV-2 infection can cause RV overload due to both lung and systemic inflammation, and can be directly linked to cardiomyocytes, causing cardiac injury [6,19]. Recently studies showed that cardiac involvement is associated with poor prognosis in these patients [20]. Patients with COVID-19 are expected to see some changes in electrocardiography due to both pulmonary involvement and RV loading due to direct myocardial involvement. In electrocardiography, RV strain pattern can be seen due to increased pulmonary artery pressure in diseases such as pulmonary embolism, pulmonary hypertension, mitral stenosis, chronic lung disease (cor pulmonale), congenital heart disease (Fallot tetratology, pulmonary stenosis) and arrhythmogenic right ventricular cardiomyopathy [10-12,15,16].

RV dysfunction is significantly associated with morbidity and mortality in some cardiovascular diseases [21-23]. An increase in pulmonary artery pressure due to chronic right ventricular hypertrophy or acute dilation occurs. Whatever the reason, the increase in pulmonary artery pressure eventually causes acute and chronic changes in the right heart cavities. The easiest method to recognize this loading is electrocardiography, although its sensitivity and specificity values are not high [24]. In electrocardiography, right atrial strain, right ventricular dilation and hypertrophy are findings that can be observed. In pulmonary thromboembolism, sinus tachycardia, RBBB, S1Q3T3 pattern, and T wave inversion in anterior leads occur due to pressure and volume loading in the lung. Our study showed that sinus tachycardia in 99 patients (30%), complete or incomplete RBBB in 61 patients (19%), S1Q3T3 in 50 patients (15%), T wave inversion in anterior leads in 42 patients (13%).

In a recently published case series, right ventricular dilatation was described in five COVID-19 patients with critical disease [25]. In a another case report with 2 COVID-19 cases, different ECG findings were observed within days [26]. In another study involving COVID-19 patients, 20% of cases had ST segment elevation / ST-T changes [27]. Previously studies demonstrated that right ventricular strain pattern is related to the pulmonary embolism and to right ventricular pressure overload with potential prognostic implications [10-12]. Our study showed that the presence of at least one classic ECG sign of right ventricular strain (29% of all patients) is associated with increased risk of death (HR: 4.4) during hospitalization. In a recent COVID-19 study RV strain values by speckle-tracking echocardiography (STE) were shown as an independent marker of mortality [8]. Due to the difficulties, accessibility and risk of infection of STE, it is important to detect RV dysfunction with ECG.

The exact cause of RV dilation and loading in patients diagnosed with COVID-19 is unknown. Right ventricular dilatation may be due to inhibition of blood flow in the lungs due to lung embolism or lung tissue damage. The direct damage of the virus to the heart tissue can be a contributing factor. COVID-19 may directly affect the myocardium through ACE-2 receptors, which are found extensively in heart tissue [5,6]. Patients infected with COVID-19 appear to be at high risk for venous thromboembolism (VTE). Abnormal coagulation parameters were detected in patients hospitalized with severe COVID-19 infection [28]. Prolonged immobilization in critically ill patients also poses a high risk for VTE. Autopsy data shows that not only in the lungs, but also in the heart, liver, kidneys and large vessels, arterial and microvascular thrombus can occur [29]. In another study comparing survivors and non-survivors with COVID-19, it was shown that non-survivors had higher levels of d-dimer and fibrin degradation products and 71.4% of them met disseminated intravascular coagulation (DIC) criteria [28]. In our study, patients with RV strain on ECG had high troponin and d-dimer levels. In addition, in our study, age, d-dimer and hs-TnI, which had prognostic significance in multivariable analysis and other studies, were determined as independent predictors of mortality.

Our study is single-center and covers only the patients who are hospitalized and treated in the hospital. Therefore, the results of the study are not valid for all COVID-19 patients. Another limitation of our study is that patients do not have old ECGs and that echocardiography was not performed simultaneously. Admission ECGs of patients have been taken and RV strain may also be developed in the follow-up of patients. The main limitation in using RV strain pattern as a prognostic tool is that it occurs in about 10% of the normal population. (especially due to complete and incomplete RBBB) [24].

5. Conclusions

The present study of patients with COVID-19 revealed that right ventricular strain pattern at ECG on admission is associated with in hospital mortality. The presence of RV strain findings detected in ECG, which is routinely examined before treatment in patients with COVID-19, may be an indicator of severe lung involvement and pulmonary embolism in COVID-19 patients. RV strain pattern detected on ECG is a prognostic marker and can be used as an indicator of prognosis in these patients. Patients with RV strain patterns, such as complete or incomplete right bundle branch block, can be identified by automated ECG interpretation, to aid the primary treating physician who is almost assuredly not an expert in ECG interpretation. It is important to recognize patients at higher risk of COVID-19 patients for poor results. Studies showing the development of RV strain pattern during hospitalization are needed for patients with normal initial ECG. Comprehensive evaluation can be made by using RV strain parameters in ECG for risk classification. Unlike age and some blood test results (D-dimer, hs-Tnl), it can be helpful in determining the prognosis in that it can be checked intuitively.

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Declaration of Competing Interest

The authors report no relationships that could be construed as a conflict of interest.

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