ORIGINAL ARTICLE

# R-WAVE PEAK TIME AS A NEW PREDICTOR OF MYOCARDIAL INJURY IN PATIENTS INFECTED WITH COVID-19

**Muharrem Said Cosgun**[1]

[1]**Mengucek Gazi Education and Research Hospital, Medical Faculty, Erzincan Binali Yildirim University, Erzincan, Turkey**

**Objectives:** Myocardial injury is closely associated with the poor prognosis of patients infected with coronavirus disease 2019 (COVID-19). Early diagnosis of cardiovascular complications that develop during the process of COVID-19 is crucial. R-wave peak time (RWPT) is an electrocardiographic parameter in which myocardial involvement caused by various situations is shown. This study was designed to assess the predictive value of RWPT in patients infected with COVID-19 who developed a myocardial injury.

**Methodology:** A total of 138 patients diagnosed with COVID-19 were enrolled in this prospective study. The patients were classified according to their troponin values ─ study group (SG, n= 52) with high troponin and control group (CG, n= 86) without elevated troponin. All data obtained from patients were compared.

**Results:** QRS duration (101 ± 5 ms vs. 99 ± 6 ms, p= .013) and RWPT (43 ± 6 ms vs. 38 ± 5 ms, p<0.001) were significantly longer in SG than in the CG. In multivariate analysis, C-reactive protein (OR: 1.109, 95% CI: 1.058–1.163; p<0.001), ejection fraction (OR: .844, 95% CI: .765–.931; p=0.001), and RWPT (OR: 1.211, 95% CI: 1.096–1.339; p<0.001) were independent predictors of myocardial injury in COVID-19-infected individuals. The ROC analysis revealed a cut-off value of RWPT for myocardial injury of 40.5 ms, with a sensitivity of 63.5% and a specificity of 62.8% (AUC: 0.730, 95% CI: 0.641–0.819, p<0.001).

**Conclusion:** RWPT is a significant predictor of myocardial injury and may benefit in better identifying patients with myocardial injury in COVID-19.

**Keywords**: COVID-19, electrocardiography, myocardial injury, QRS duration, R-wave peak time

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## INTRODUCTION

Despite vaccination, strict restrictions, and lockdowns, the coronavirus disease 2019 (COVID-19) outbreak spreads rapidly with high transmission rates, probably due to new mutations affecting individuals worldwide.[1, 2] Although most people have a mild infection, if a severe form of the disease develops, the risk of multiorgan damage increases, particularly lung and heart damage, and can lead to death.[3]

Susceptibility to COVID-19 is increased by existing cardiovascular comorbidities.[4] In addition, COVID-19 may lead to cardiac complications and aggravate the condition, even without any susceptibility-enhancing comorbidities.[5] In any situation, cardiovascular involvement is closely associated with a poor prognosis and mortality.[6] Therefore, further clarification of the role of the cardiovascular system and early detection of whether the cardiovascular system is affected is crucial in the treatment of COVID-19.[7]

Electrocardiogram (ECG) is one of the marked instruments in evaluating cardiac involvement in COVID-19 patients due to its advantages, such as easy accessibility, low cost, frequent repeatability, operator independent, and remote evaluation.[8] ST-segment and T wave changes, QT prolongation, conduction disturbances, and new-onset arrhythmias were shown to be potential predictors of poor clinical outcomes in patients with COVID-19 in previous studies.[9] The R-wave peak time (RWPT), an ECG parameter known as the intrinsicoid deflection time or ventricular activation time, is a representation of the electrical activity of the heart spreading from the left ventricle (LV) endocardium to the epicardium.[10] Ventricular mass increase (hypertrophy or dilation) and conduction delay (bundle branch block or ischemia) are known to prolong RWPT.[11] Nonspecific intraventricular conduction delays can be seen on ECG in patients with myocarditis due to COVID-19.[12]

To the best of our knowledge, there are no studies examining the relationship between RWPT and myocardial injury in patients infected with COVID-19. We hypothesized that the RWPT might be prolonged due to the conduction delay in myocarditis caused by the COVID-19 infection. As a result, this prospective research aimed to investigate the potential

role of RWPT as a novel indicator of myocardial damage in COVID-19 patients.

## METHODOLOGY

A total of 138 consecutive patients were included in this prospective study conducted between November 2021 and February 2022. All patients were hospitalized in isolated wards, and reverse transcription-polymerase chain reaction (RT-PCR) confirmed the diagnosis of COVID-19. The patients were divided into two groups according to their troponin values during follow-up. Patients with high troponin levels were included in the study group (SG, n= 52), and patients with average values were admitted to the control group (CG, n= 86). The criteria used for the exclusion of patients in this study were as follows: 1) refusal to provide informed consent, 2) history of cardiomyopathy, 3) left ventricular systolic dysfunction, 4) left ventricular hypertrophy or dilation, 5) severe valvular disease, 6) active neoplastic or rheumatological disease, 7) renal dysfunction, 8) pregnancy or breastfeeding, and 9) presence of a pacemaker. Additionally, 10) patients with signs of infranodal block on the ECG (Mobitz II and complete atrioventricular block or complete bundle branch block) were also excluded.

Age, gender, smoking status, previous medications and diseases, and treatment regimen for COVID-19 were registered for both groups. All patients included in the study were symptomatic, having air hunger and pneumonia symptoms on CT but did not require referral to the intensive care unit (ICU) at admission (severe category).[13]

Complete blood counts (e.g., hemoglobin and lymphocyte), fasting blood glucose, creatinine, troponin I, D-dimer, and C-reactive protein (CRP) levels were measured in the blood samples of the patients taken during admission. Troponin I concentrations over the 99th percentile upper reference range were considered evidence of myocardial damage.

The 12-lead ECGs of the patients during admission to the inpatient clinic were evaluated. All ECGs were performed following the required standards (low-pass filter: 0.5–25 Hz, 25 mm/s paper speed, and 10 mm/mV voltage calibration). At this time, the patients were RT-PCR positive in the nasopharyngeal swab and were in the early infectious phase of the disease. ECGs were digitalized by scanning and analyzed with digital image processing software (Image J; https://imagej.net/software/fiji/) (Fig. 1). QRS duration and RWPT were measured from the precordial ECG leads (V5 or V6), in which they were the longest, and were calculated in milliseconds (ms) by taking the average of three consecutive beats. The QRS duration is the time interval between the beginning of the QRS complex and the J point. RWPT is the time interval from the onset of the QRS complex to the peak of the R or R' wave. According to RWPT (cut-off= 40.5 ms), the patients were divided into normal and prolonged.

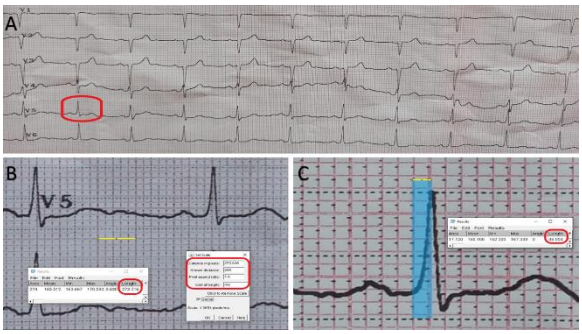


**Figure 1: The electrocardiograms of the patients during admission (A), setting the scale according to the reference time (B), and R-wave peak time measurement (C)**

Echocardiographic examinations were performed using an X5–1 (1–5 MHz) transducer and a Philips Epiq 7C device. Left ventricular ejection fraction (LVEF) was calculated using the modified Simpson's method. Interventricular septum (IVS) thickness was measured from the parasternal long axis at the end-diastole.

This study was in compliance with the ethical principles of the Declaration of Helsinki regarding human participants. The Local Institutional Ethics Committee approved the study protocol (approval number: 07/06 and approval date: July 07, 2020). All participants signed informed consent forms.

Categorical variables were compared using the chi-square test and expressed as counts (n) and percentages (%). The distribution of continuous variables was evaluated using the one-sample Kolmogorov-Smirnov test. Normally distributed variables were expressed as means ± standard deviations, whereas not normally distributed variables were expressed as medians [interquartile ranges]. The paired t-test was used for normally distributed continuous variables, and the Mann-Whitney U test was used for variables that were not normally distributed. Pearson's rank correlation coefficients were used to define the associations among continuous variables. Parameters found to be statistically significant ($p < .05$) in univariate analysis were evaluated using multiple logistic regression analysis and expressed as odds ratios (OR) with 95% confidence intervals (CI) to

determine the independent predictors of myocardial damage. The cut-off value of RWPT for myocardial injury was analyzed by the receiver operating characteristic (ROC) curve and expressed as area under the curve (AUC) with 95% CI. Statistical significance value was considered as p< .05. All data were analyzed using IBM SPSS Statistics version 22 (IBM Corp., Armonk, NY, USA) for Windows.

## RESULTS

Table 1 compares the baseline demographic, clinical, laboratory, imaging, ECG, and echocardiographic characteristics. No significant difference was noted between the groups regarding mean age, gender, smoking status, previous diseases, or medications. The use of antibiotics (78.8% vs. 61.6%, p= .035) and corticosteroids (65.4% vs. 47.7%, p= .043) was higher in the SG than in CG. There was no difference between the groups regarding other treatment regimens for COVID-19. The lymphocyte count was significantly lower in the SG than in the CG (1.54 ± 0.71 vs. 1.78 ± 0.7, p= .039). Median troponin values were 66.5 [55–95.7] in the SG and 10 [10–14.3] in the CG. The difference was statistically significant (p< .001). Both median levels of D-dimer (718 [484–990] vs. 591 [458–803], p= .048) and CRP (28.9 [10.1–44] vs. 10.3 [5.8–15.9], p< .001) were higher in the SG than in CG. There was no difference between the groups regarding other laboratory results. CT imaging showed a higher rate of diffuse spread in the SG than in the CG (36.5% vs. 20.9%, p= .045). ECG analysis of the patients revealed a longer QRS duration (101 ± 5 ms vs. 99 ± 6 ms, p= .013) and RWPT (43 ± 6 ms vs. 38 ± 5 ms, p< .001) in the SG than in the CG. In the echocardiographic examination, the LVEF of the SG patients was significantly lower than in the CG (48 ± 4% vs. 52 ± 5%, p= .006). IVS thickness was similar between groups. ICU referral rate was significantly higher in SG compared to CG (59.6% vs. 18.6%, p< .001). The overall mortality rate in our study was 17.4%. Mortality was significantly higher in the SG than in the CG (38.5% vs. 4.7%, p< .001).

Myocardial injury was positively associated with D-dimer (r= .214, p= .006), CRP (r= .543, p< .001), QRS duration (r= .167, p= .025), and RWPT (r= .372, p< .001). Conversely, it was negatively associated with lymphocyte count (r= −.185, p= .015) and left ventricular ejection fraction (r= −.251, p= .001).

In multivariate logistic regression analysis, CRP levels (OR: 1.109, 95% CI: 1.058–1.163, p< .001), LVEF (OR: .844, 95% CI: .765–.931, p= .001), and RWPT (OR: 1.211, 95% CI: 1.096–1.339, p< .001) were independent predictors of myocardial injury in COVID-19 (Table 2).

**Table 2: Multivariate logistic regression analysis to assess predictors of myocardial injury**

| Variables | Odds Ratio (95% CI) | P-Value |
|---|---|---|
| Lymphocyte count | 0.583 (0.272–1.251) | 0.166 |
| D-dimer | 1.001 (1.000–1.003) | 0.082 |
| C-reactive protein | 1.109 (1.058–1.163) | <0.001 |
| R-wave peak time | 1.211 (1.096–1.339) | <0.001 |
| Left ventricular ejection fraction | 0.844 (0.765–0.931) | 0.001 |

**Confidence interval*

The ROC analysis revealed a cut-off value of RWPT for myocardial injury of 40.5 ms, with a sensitivity of 63.5% and a specificity of 62.8% (AUC: .730, 95% CI: 0.641–0.819, p< .001).

The patients were classified into two groups according to the RWPT (cut-off= 40.5 ms). There were 73 (52.9%) patients in the normal RWPT group, and 65 (47.1%) in the prolonged RWPT group. There was no difference between age, gender, smoking status, previous diseases and medications, treatment regimen, or laboratory results, except troponin levels and radiological findings. Troponin values were significantly higher in the prolonged RWPT group compared to the normal RWPT group (36 [10–66] vs. 10 [10–42.5], p< .006). In ECG analysis, longer QRS durations (101 ± 6 ms vs. 99 ± 5 ms, p= .033) and RWPT (45 ± 4 ms vs. 35 ± 3 ms, p< .001) were detected in the prolonged RWPT group. In the echocardiographic examination, the LVEF of the SG patients was significantly lower than in the CG (49 ± 4% vs. 51 ± 5%, p= .036). IVS thickness was similar between the groups. Moreover, myocardial injury (50.8% vs. 26%, p= .003), referral to ICU (41.5% vs. 27.4%, p= .044), and mortality (24.6% vs. 11%, p= .035) were significantly higher in the prolonged RWPT group than in the normal RWPT group (Table 3).

**Table 1: Basic demographic, clinical, laboratory, electrocardiographic, echocardiographic, and imaging characteristics of patients according to myocardial injury**

| Variables | All patients (n= 138) | Control group (n= 86) | Study group (n= 52) | P-Value |
|---|---|---|---|---|
| **Demographic characteristics** | | | | |
| Age, years | 62.9 ± 9.4 | 62.2 ± 9.3 | 64.1 ± 9.5 | 0.246 |
| Gender, male, n (%) | 75 (54.3) | 45 (52.3) | 30 (57.7) | 0.54 |
| Smoking status, n (%) | 29 (21) | 19 (22.1) | 10 (19.2) | 0.689 |
| **Previous diseases, n (%)** | | | | |
| Coronary artery disease | 21 (15.2) | 10 (11.6) | 11 (21.2) | 0.131 |
| Hypertension | 47 (34.1) | 27 (31.4) | 20 (38.5) | 0.396 |
| Dyslipidemia | 28 (20.3) | 16 (18.6) | 12 (23.1) | 0.527 |
| Diabetes mellitus | 20 (14.5) | 11 (12.8) | 9 (17.3) | 0.691 |
| **Previous medications, n (%)** | | | | |
| Renin-angiotensin-aldosterone system blockers | 35 (25.4) | 20 (23.3) | 15 (28.8) | 0.465 |
| Calcium channel blocker | 16 (11.6) | 9 (10.5) | 7 (13.5) | 0.594 |
| Beta-bloker | 16 (11.6) | 8 (9.3) | 8 (15.4) | 0.279 |
| Statin | 15 (10.9) | 8 (9.3) | 7 (13.5) | 0.447 |
| Antiaggregant | 16 (11.6) | 7 (8.1) | 9 (17.3) | 0.103 |
| **Treatment regimens, n (%)** | | | | |
| Low-molecular-weight heparin | 126 (91.3) | 78 (90.7) | 48 (92.3) | 0.745 |
| Antiviral | 120 (87) | 74 (86) | 46 (88.5) | 0.683 |
| Antibiotic | 94 (68.1) | 53 (61.6) | 41 (78.8) | 0.035 |
| Corticosteroid | 75 (54.3) | 41 (47.7) | 34 (65.4) | 0.043 |
| Hydroxychloroquine | 32 (23.2) | 19 (22.1) | 13 (25) | 0.695 |
| **Laboratory results** | | | | |
| Hemoglobin, g/dL | 13.6 ± 1.2 | 13.5 ± 1.1 | 13.8 ± 1.4 | 0.122 |
| Lymphocyte count, $\times 10^9$/L | 1.69 ± 0.71 | 1.78 ± 0.7 | 1.54 ± 0.71 | 0.039 |
| Glucose, mg/dL | 102 [92–118] | 103 [91–123] | 100 [95–115] | 0.465 |
| Creatinine, mg/dL | 0.82 ± 0.2 | 0.82 ± 0.21 | 0.81 ± 0.18 | 0.804 |
| Troponin I, ng/L | 10 [10–59.5] | 10 [10–14.3] | 66.5 [55–95.7] | <0.001 |
| D-dimer, µg/L | 658 [466–834] | 591 [458–803] | 718 [484–990] | 0.048 |
| C-reactive protein, mg/L | 11.3 [6.6–24.9] | 10.3 [5.8–15.9] | 28.9 [10.1–44] | <0.001 |
| **Radiological distribution, n (%)** | | | | |
| Peripheral | 84 (60.9) | 56 (65.1) | 28 (53.8) | 0.189 |
| Central | 17 (12.3) | 12 (14) | 5 (9.6) | 0.452 |
| Diffuse | 37 (26.8) | 18 (20.9) | 19 (36.5) | 0.045 |
| **Electrocardiographic parameters, ms** | | | | |
| QRS duration | 100 ± 6 | 99 ± 6 | 101 ± 5 | 0.013 |
| R-wave peak time | 40 ± 6 | 38 ± 5 | 43 ± 6 | <0.001 |
| **Echocardiographic parameters** | | | | |
| Left ventricular ejection fraction, % | 50 ± 5 | 52 ± 5 | 48 ± 4 | 0.006 |
| Interventricular septum thickness, mm | 10 ± 2 | 10 ± 2 | 11 ± 2 | 0.644 |
| **Prognosis and follow-up, n (%)** | | | | |
| Referral to intensive care unit | 47 (34.1) | 16 (18.6) | 31 (59.6) | <0.001 |
| Mortality | 24 (17.4) | 4 (4.7) | 20 (38.5) | <0.001 |

**Table 3: Basic demographic, clinical, laboratory, electrocardiographic, echocardiographic, and imaging characteristics of patients according to R-wave peak time (cut-off= 40.5 ms)**

| Variables | All patients (n= 138) | R-wave peak time | | P-value |
|---|---|---|---|---|
| | | Normal (< 40.5 ms) (n= 73) | Prolonged (≥ 40.5 ms) (n= 65) | |
| **Demographic characteristics** | | | | |
| Age, years | 62.9 ± 9.4 | 62.9 ± 9.7 | 62.8 ± 9 | 0.997 |
| Gender, male, n (%) | 75 (54.3) | 41 (56.2) | 34 (52.3) | 0.650 |
| Smoking status, n (%) | 29 (21) | 15 (20.5) | 14 (21.5) | 0.887 |
| **Previous diseases, n (%)** | | | | |
| Coronary artery disease | 21 (15.2) | 9 (12.3) | 12 (18.5) | 0.317 |
| Hypertension | 47 (34.1) | 25 (34.2) | 22 (33.8) | 0.960 |
| Dyslipidemia | 28 (20.3) | 15 (20.5) | 13 (20) | 0.936 |
| Diabetes mellitus | 20 (14.5) | 10 (13.7) | 10 (15.4) | 0.779 |
| **Previous medications, n (%)** | | | | |
| Renin-angiotensin-aldosterone system blockers | 35 (25.4) | 18 (24.7) | 17 (26.2) | 0.840 |
| Calcium channel blocker | 16 (11.6) | 10 (13.7) | 6 (9.2) | 0.413 |
| Beta-bloker | 16 (11.6) | 7 (9.6) | 9 (13.8) | 0.436 |

| | | | | |
|---|---|---|---|---|
| Statin | 15 (10.9) | 8 (11) | 7 (10.8) | 0.971 |
| Antiaggregant | 16 (11.6) | 9 (12.3) | 7 (10.8) | 0.775 |
| **Treatment regimens, n (%)** | | | | |
| Low-molecular-weight heparin | 126 (91.3) | 67 (90.7) | 59 (92.3) | 0.833 |
| Antiviral | 120 (87) | 63 (86.3) | 57 (87.7) | 0.809 |
| Antibiotic | 94 (68.1) | 51 (69.9) | 43 (66.2) | 0.641 |
| Corticosteroid | 75 (54.3) | 38 (52.1) | 37 (56.9) | 0.567 |
| Hydroxychloroquine | 32 (23.2) | 17 (23.3) | 15 (23.1) | 0.977 |
| **Labaratory results** | | | | |
| Hemoglobin, g/dL | 13.6 ± 1.2 | 13.6 ± 1.1 | 13.7 ± 1.3 | 0.452 |
| Lymphocyte count, $\times 10^9$/L | 1.69 ± 0.71 | 1.73 ± 0.69 | 1.64 ± 0.74 | 0.407 |
| Glucose, mg/dL | 102 [92–118] | 102 [94–123] | 101 [95–118] | 0.645 |
| Creatinine, mg/dL | 0.82 ± 0.2 | 0.82 ± 0.2 | 0.81 ± 0.19 | 0.854 |
| Troponin I, ng/L | 10 [10–59.5] | 10 [10–42.5] | 36 [10–66] | <0.006 |
| D-dimer, µg/L | 658 [466–834] | 591 [440–809] | 707 [482–918] | 0.126 |
| C-reactive protein, mg/L | 11.3 [6.6–24.9] | 10.7 [6.6–22] | 11.6 [7.6–28.9] | 0.335 |
| **Radiological distribution, n (%)** | | | | |
| Peripheral | 84 (60.9) | 44 (60.3) | 40 (61.5) | 0.879 |
| Central | 17 (12.3) | 10 (13.7) | 7 (10.8) | 0.601 |
| Diffuse | 37 (26.8) | 19 (26) | 18 (27.7) | 0.826 |
| **Electrocardiographic parameters, ms** | | | | |
| QRS duration | 100 ± 6 | 99 ± 5 | 101 ± 6 | 0.033 |
| R-wave peak time | 40 ± 6 | 35 ± 3 | 45 ± 4 | <0.001 |
| **Echocardiographic parameters** | | | | |
| Left ventricular ejection fraction, % | 50 ± 5 | 51 ± 5 | 49 ± 4 | 0.036 |
| Interventricular septum thickness, mm | 10 ± 2 | 11 ± 3 | 10 ± 2 | 0.244 |
| **Prognosis and follow-up, n (%)** | | | | |
| Myocardial injury | 52 (37.7) | 19 (26) | 33 (50.8) | 0.003 |
| Referral to intensive care unit | 47 (34.1) | 20 (27.4) | 27 (41.5) | 0.044 |
| Mortality | 24 (17.4) | 8 (10.9) | 16 (24.6) | 0.035 |

## DISCUSSION

It was shown in our study that prolonged RWPT on the ECG at admission is an independent predictor of myocardial injury in patients infected with COVID-19. Additionally, elevated CRP levels and reduced LVEF were independent predictors of myocardial damage. Also, lymphopenia and high D-dimer levels were more common in patients who developed a myocardial injury

There are few studies on the clinical use of RWPT in the literature.[14-22] Its clinical use first demonstrated its predictive value in differentiating wide QRS complex tachycardias.[14-16] Prolongation of RWPT secondary to left ventricular hypertrophy has been reported in patients with end-stage kidney failure and aortic valve diseases.[17, 18] Besides, prolonged RWPT is associated with diastolic dysfunction even if left ventricular hypertrophy is not developed in patients with early hypertension.[10] This situation is explained by electrical remodeling.[19] It has been shown that there is a correlation between prolonged RWPT and poor prognostic markers, such as no-reflow and high syntax scores, in patients with the acute coronary syndrome. Conduction delays due to ischemia in myocytes and Purkinje fibers are the primary mechanism.[20-22]

It was reported in a study with a large sample that atrial arrhythmias, repolarization abnormalities, and intraventricular conduction blocks are signs of left-sided heart involvement closely associated with mortality in COVID-19. In this study, the incidence of intraventricular conduction block was below 10% in surviving patients, while this rate was above 30% in patients who died.[12] In light of this information, QRS duration and RWPT may be prolonged in patients with COVID-19 infection with myocardial injury in proportion to the extent of the damage. In our study, QRS duration and RWPT were significantly longer in patients with myocardial injury, and RWPT was an independent predictor of myocardial injury. A regional conduction delay to myocardial injury may explain the superiority of RWPT, measured from precordial leads, in predicting myocardial injury over QRS duration. Because intraventricular conduction delay is related to left-sided heart injury and the QRS complex illustrates depolarization of the entire heart, QRS duration may be less susceptible than RWPT from precordial leads.[11, 12]

In a previous study, the presence of cardiovascular disease (13.3%) or myocardial injury (37.5%) in hospitalized patients was shown to increase mortality. In comparison, myocardial damage in patients with cardiovascular disease further increased mortality (69.4%).[23] Another study demonstrated that more than half of the individuals who died had myocardial damage. The heart was the most commonly affected nonpulmonary organ from COVID-19 infection and

was affected in approximately 20% of hospitalized patients.[6] As already mentioned above, cardiovascular complications are frequent and significantly increase mortality.[5] In a large-scale hospitalized patient cohort study by Lala et al., the myocardial injury was 36%, and overall mortality was 18.5%.[24] Similarly, in our research on hospitalized severe category patients, the incidence of myocardial injury was 37.6%, and overall mortality was 17.4%. The mortality rate was 38.5% in patients with heart damage.

It has been shown in the literature that there is a relationship between elevated CRP and D-dimer levels, lymphopenia, and reduced LVEF with myocardial damage in COVID-19.[25] Elevated CRP and reduced LVEF were confirmed as independent predictors of myocardial injury in patients infected with COVID-19 in our study. Consistent with these studies, we determined meaningfully high D-dimer levels and lymphopenia in COVID-19 patients with myocardial injuries.

Despite its scientific findings, the current report has various limitations. First, patients with average troponin values at hospitalization did not have control values unless they had cardiac symptoms. This condition may pose a problem in identifying patients with silent myocardial injury. Second, only myocardial injury due to COVID-19 was evaluated in our study. Further studies are needed with other myocardial injury causes for broader clinical use. Finally, the relatively low number of patients is another significant limitation of this study.

## CONCLUSION

RWPT is a new independent predictor for detecting cardiac damage—the most common extrapulmonary involvement and clinical condition that increases mortality during COVID-19. Early and reliable detection of this condition will be beneficial in guiding treatment and reducing mortality.

## AUTHORS' CONTRIBUTION:

MSC: Concept and design, data acquisition, interpretation, drafting, final approval, and agree to be accountable for all aspects of the work.

**Conflict of interest:** Authors declared no conflict of interest.

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## Address for Correspondence:

**Dr. Muharrem Said Cosgun,** Assistant Professor, Erzincan Binali Yildirim University, Medical Faculty, Department of Cardiology, Erzincan, Turkey.
**Email:** drsaidcosgun2009@hotmail.com