Cukurova Medical Journal
ÇUKUROVA ÜNİVERSİTESİ TIP FAKÜLTESİ

Cukurova Med J 2021;46(2):756-763
DOI: 10.17826/cumj.895173

ARAŞTIRMA / RESEARCH

Relationship between RS time and the severity of chronic obstructive pulmonary disease

RS zamanı ile kronik obstrüktif akciğer hastalığının şiddeti arasındaki ilişki

İbrahim Yıldız[1], Pınar Özmen Yıldız[1], Hüsamettin Sazlıdere[2], Mehmet Sait Gürevin[1], İbrahim Rencüzoğulları[3], Yavuz Karabağ[3], Cengiz Burak[3], Çağlar Özmen[4]

[1]Osmaniye State Hospital, Department of Cardiology, [2]Department of Chest Diseases, Osmaniye, Turkey
[3]Kafkas University Faculty of Medicine, Department of Cardiology, Kars, Turkey
[4]Cukurova University Faculty of Medicine, Department of Cardiology, Adana, Turkey.

Cukurova Medical Journal 2021;46(2):756-763

Abstract

Purpose: The aim of our study was to determine whether the RS time measured on an electrocardiogram is associated with the severity of chronic obstructive pulmonary disease (COPD).

Materials and Methods: The study population was made up of 100 COPD patients with a mean age of 63 ± 10 years (86% were males). These patients were divided into the mild and moderate COPD (GOLD 1–2) group (n = 45) and the severe and very severe COPD (GOLD 3–4) group (n = 55) according to the Global Initiative for Chronic Obstructive Lung Disease (GOLD) stages. These groups were compared with each other.

Results: Heart rate, P wave axis, QRS axis, RS time, and incidence of right bundle branch block were significantly higher in patients with severe and very severe COPD. In the multivariable analysis, heart rate, P wave axis, and RS time (odds ratio = 1.081) were the independent predictors of severe and very severe COPD. The receiver operating characteristic curve analysis showed that the best RS time cutoff value for the prediction of severe and very severe COPD was 60 ms, with a sensitivity of 69.1% and a specificity of 66.7%.

Conclusion: RS time simply measured on an electrocardiogram may be a useful screening test for the quick estimation of patients with severe and very severe COPD.

Keywords:. Chronic obstructive pulmonary disease, electrocardiography, electrophysiology

Öz

Amaç: Çalışmamızın amacı, elektrokardiyogramdan ölçülen RS zamanının kronik obstrüktif akciğer hastalığı (KOAH) şiddeti ile ilişkili olup olmadığını belirlemekti.

Gereç ve Yöntem: Çalışma popülasyonu, ortalama yaşı 63 ± 10 yıl olan (% 86 erkekti) 100 KOAH hastasından oluştu. Kronik Obstrüktif Akciğer Hastalığına Karşı Küresel Girişim (GOLD) aşamalarına göre bu hastalar hafif ve orta derecede KOAH (GOLD 1–2) grubu (s = 45) ve şiddetli ve çok şiddetli KOAH (GOLD 3–4) grubuna (s = 55) ayrıldı. Bu gruplar birbirleriyle karşılaştırıldı.

Bulgular: Şiddetli ve çok şiddetli KOAH'lı hastalarda kalp hızı, P dalga aksı, QRS ekseni, RS zamanı ve sağ dal bloğu insidansı anlamlı olarak daha yüksekti. Çok değişkenli analizde, kalp hızı, P dalga aksı ve RS zamanı (olasılık oranı = 1.081) şiddetli ve çok şiddetli KOAH'ın bağımsız belirleyicileriydi. Alıcı işletim karakteristik eğrisi analizi, şiddetli ve çok şiddetli KOAH tahmini için en iyi RS zaman kesme değerinin, % 69,1 duyarlılık ve % 66,7 özgüllük ile 60 ms olduğunu gösterdi.

Sonuç: Bir elektrokardiyogramda basitçe ölçülen RS zamanı, şiddetli ve çok şiddetli KOAH'lı hastaların hızlı tahmini için yararlı bir tarama testi olabilir.

Anahtar kelimeler: Kronik obstrüktif akciğer hastalığı, elektrokardiyografi, elektrofizyoloji

Yazışma Adresi/Address for Correspondence: Dr. İbrahim Yıldız, Osmaniye State Hospital, Department of Cardiology, Osmaniye, Turkey E-mail: ibrahimyildiz79@yahoo.com
Geliş tarihi/Received: 15.03.2021 Kabul tarihi/Accepted: 15.04.2021 Çevrimiçi yayın/Published online: 20.05.2021

INTRODUCTION

Chronic obstructive pulmonary disease (COPD), which is described by airflow limitation and permanent respiratory symptoms, is an important growing public health problem because of its increased morbidity, mortality, and economic burden. COPD is now the fourth primary reason of mortality throughout the globe after heart attack, malignancies, and stroke and is anticipated to be the third foremost cause of mortality in the coming decade[1,2].

Airflow limitation severity is strongly associated with cardiovascular mortality[3]. Hypoxic vasoconstriction of the small pulmonary arteries results in structural changes in the pulmonary vasculature that aggravate with disease progression in COPD patients[4,5]. Structural changes in the vasculature lead to an increment in pulmonary vascular resistance and right ventricular afterload, thereby pulmonary hypertension and right ventricular dysfunction[6].

An electrocardiogram (ECG) is an effortless, inexpensive, and readily reachable diagnostic instrument for detecting cardiac disorders related to COPD[7]. The increment of air trapped between the heart and recording electrodes, heart verticalization due to the low diaphragmatic position, right heart chamber enlargement, and right ventricular hypertrophy may lead to ECG changes in COPD patients[7]. It has been previously reported that wide and slurred S-waves in leads I, II, III, V4, V5, and V6 can be encountered in pulmonary heart diseases[8]. Moreover, right ventricular overload may appear on the ECG by stretching the S-wave and widening the duration from the beginning of QRS to the nadir of S-wave (RS time) in the inferolateral leads[9]. RS time, the prolongation of which is recommended to be used to detect acute pulmonary embolism, is a newly introduced ECG parameter[9].

The frequency of cardiac abnormalities is significantly related to the severity of airflow limitation in COPD patients[10,11]. Mortality has also been shown to increase with the severity of airflow limitation in COPD patients[12]. In this research, we intended to explore the relationship between severity of the disease graded by spirometry and RS time in COPD patients to determine whether RS time prolongation could be used to screen COPD patients for the severity of the disease in routine clinical practice.

MATERIALS AND METHODS

Study population

Our research was performed on consecutive patients previously diagnosed with COPD by pulmonary function tests who came to regular outpatient clinic checkups in a chest disease outpatient clinic from November 2020 to January 2021. Patients with a poor echocardiographic image, ejection fraction of less than 50%, moderate to severe cardiac valvular disease, having electrolyte disturbances, a past of coronary artery disease, acute exacerbation of COPD, left bundle branch block, Wolf-Parkinson-White pattern, pacemaker rhythm, cardiac arrhythmia, and atrial fibrillation on electrocardiography were excluded. 100 COPD patients remaining after exclusion criteria constituted the study population.

The patients were graded into four Global Initiative for Chronic Obstructive Lung Disease (GOLD) grades according to their post-bronchodilator percent-predicted forced expiratory volume in first second (ppFEV1)[13]. The patients with GOLD grade 1 and GOLD grade 2 constituted the mild and moderate COPD group, and the patients with GOLD grade 3 and GOLD grade 4 constituted the severe and very severe COPD group. All COPD patients were examined through a blood test, spirometry, electrocardiography, and echocardiography performed on the same day. Electrocardiography was performed immediately after the spirometric examination was performed. Each patient was analyzed independently by investigators blinded to the results. All enrolled patients gave written informed consent, and ethical approval for the research was obtained from the Ethics Committee of Kafkas University Faculty of Medicine dated 04.11.2020 and numbered 80576354-050-99 / 234.

Clinical and laboratory data

The patients' medical records were used to obtain information about the patient's age, gender, smoking history, and whether the patient had diabetes, hypertension, and chronic kidney failure. Blood pressure was measured, and body surface area was calculated for each patient. Body surface area was computed with a calculator program (calculator.net) using the patient's height and weight. Routine laboratory blood tests were also performed on the patients.

ECG evaluation

12-lead surface ECG outputs recorded at a speed of 25 mm/s and a voltage of 10 mm/mV were analyzed with an image processing program (imagej.nih.gov/ij/). Two independent cardiologists carried out the ECG evaluations, unaware of the patients' clinical, demographic, laboratory, spirometric, and echocardiographic findings. In case of disagreement, a consensus was achieved by receiving the opinion of a third veteran cardiologist. The ECGs were examined in terms of heart rate, frontal P wave axis, frontal QRS axis, right bundle branch block, QRS fragmentation, clockwise rotation, and right frontal QRS axis deviation. The minimum and maximum P wave durations on the surface ECG were used to calculate the P wave dispersion[14]. The longest period from the beginning of the QRS complex until the J-point was determined as the QRS duration. Utilizing the most extended QT interval on the 12-lead surface ECG, QTc was calculated according to Bazett's formula[15]. Right QRS axis deviation was defined as an axis being between +90 degrees and +180 degrees. If the R wave amplitude is smaller than the S wave amplitude in lead V4, it was defined as clockwise rotation. QRS fragmentation was defined as the presence of notches or low-voltage waves in the QRS complex in at least two contiguous leads. RS time was described as the duration from the QRS complex's onset until the nadir of the S or S' wave (Fig. 1). The inferolateral leads signified by leads D1, AVL, D2, D3, AVF, V4, V5, and V6 were used to measure RS time. The longest duration evaluated from these leads was received as the RS time[9].

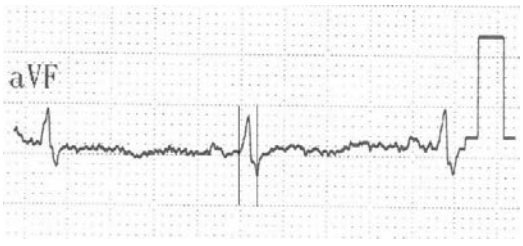

Figure 1. Measurement of RS time on electrocardiography strip.

Spirometric evaluation

Based on the American Thoracic Society Guidelines, the pulmonary function test utilizing spirometry was carried out and studied by a researcher without knowledge of ECG results[16]. The presence of a post-bronchodilator FEV1/forced vital capacity < 0.70 in patients with peculiar symptoms and substantial exposures to noxious stimuli was taken as a description of COPD. The severity of airflow limitation in COPD patients was arranged based on the GOLD classification based on post-bronchodilator ppFEV1 as GOLD grade 1 (ppFEV1 $\geq$ 80%), GOLD grade 2 (50% $\leq$ ppFEV1 < 80%), GOLD grade 3 (30% $\leq$ ppFEV1 < 50%), and GOLD grade 4 (ppFEV1 < 30%)[13].

Echocardiographic evaluation

A veteran cardiologist made a transthoracic echocardiographic examination of the patients by an EPIQ 7 ultrasound system for cardiology (Philips Medical Systems, Bothell, WA, USA). The modified Simpson's method was implemented to measure the LV ejection fraction. Bernoulli equation, inferior vena cava diameter, and inspiratory collapse were evaluated together to estimate systolic pulmonary artery pressure. In the apical four-chamber view, the right ventricle dimension measurement was taken at the diastole's end. The body surface area indexed the right ventricle dimension to calculate the right ventricle dimension index. The right ventricle systolic function was valued by measuring the tricuspid annular plane systolic excursion (TAPSE). The M-mode cursor was positioned where the tricuspid valve plane and the right ventricle free wall intersect in the apical four-chamber view to find the maximum displacement of the tricuspid annulus during systole.

Statistical analysis

SPSS Statistics for Windows version 17.0 (SPSS Inc., Chicago, IL, USA) was used in all statistical analyses. A p-value of less than 0.05 was considered statistically significant. The data gathered from the patients in the group of mild and moderate COPD checked against the group of severe and very severe COPD. The Kolmogorov-Smirnov test was used to study the normality of the data distribution. The normally distributed continuous variables were introduced as the means and standard deviations, and non-normally distributed continuous variables were introduced as the medians. A comparison of the groups was performed using Student's t-test or the Mann-Whitney U test in accordance with the distribution of the variables. Numbers and percentages were used in the presentation of categorical variables. Chi-square test or Fisher's exact tests, whichever is appropriate, were used to compare the groups. Univariable and

multivariable logistic regression analyses were performed to determine the independent predictors of severe and very severe COPD. A multivariable logistic regression model was developed to determine the independent predictors of severe and very severe COPD. In the model, multivariable logistic regression analysis was performed with the variables that showed a p-value < 0.05 in the univariable analysis. Adequacy of the model was evaluated with a non-significant p-value ($p > 0.05$) at the Hosmer-Lemeshow test. A receiver operating characteristic (ROC) curve analysis was applied to establish an RS time cutoff point to best predict severe and very severe COPD. The correlation analysis between ppFEV1 and RS time was conducted using Pearson's correlation test.

RESULTS

The study population comprised 100 COPD patients. Their mean age was 63 ± 10 (14% female). The patients were assigned to one of two groups based on their GOLD status according to the spirometry results. Twelve patients with GOLD grade 1 and 33 patients with GOLD grade 2 were placed in the mild and moderate COPD group ($n = 45$), and 41 patients with GOLD grade 3 and 14 patients with GOLD grade 4 were placed in the severe and very severe COPD group ($n = 55$). The characteristics of the study population are listed in Table 1. There was no statistically significant difference between the groups concerning sex, blood pressure, and body surface area. The patients with severe and very severe COPD were significantly older than those with mild and moderate COPD. The incidence of hypertension, smoking, and chronic renal failure was similar for the two groups, except for the incidence of diabetes. Also, the laboratory measurements with the inclusion of hemoglobin, hematocrit, white blood cell count, C-reactive protein, blood glucose, and creatinine levels were identical.

The left ventricular ejection fraction, right ventricular dimension, right ventricular dimension index, P wave dispersion, QRS duration, QTc duration, incidence of fragmentation, right axis deviation, and clockwise rotation were similar for both groups. However, the systolic pulmonary artery pressure, heart rate, P wave axis, QRS axis, RS time (58 ± 9 vs. 65 ± 10, $p = 0.002$), and incidence of right bundle branch block were significantly higher and the TAPSE was significantly lower in the patients with severe and very severe COPD than in those with mild and moderate COPD.

Table 1. The characteristic of patients according to the severity of COPD

	GOLD status						
	All patients(n:100)		**GOLD 1-2 (n:45) Mild and moderate COPD**		**GOLD 3-4 (n:55) Severe and very severe COPD**		**p Value**
Age (years)	63	±10	60	±10	65	±9	0.017
Female gender, n (%)	14	(14.0)	7	(15.6)	7	(12.7)	0.687
Diabetes, n (%)	16	(16.0)	11	(24.4)	5	(9.1)	0.038
Hypertension, n (%)	33	(33.0)	18	(40.0)	15	(27.3)	0.180
Smoking, n (%)	88	(88.0)	39	(86.7)	49	(89.1)	0.712
Chronic renal failure, n(%)	4	(4.0)	2	(4.4)	2	(3.6)	0.838
Systolic blood pressure (mmHg)	127	±19	130	±19	124	±18	0.068
Diastolic blood pressure (mmHg)	78	±12	80	±11	76	±11	0.061
Body surface area (m^2)	1.83	±0.17	1.84	±0.17	1.82	±0.18	0.737
FEV1/FVC ratio	0.58	±0.09	0.64	±0.05	0.52	±0.09	<0.001
ppFEV1 (%)	51	±19	68	±12	36	±9	<0.001
Hemoglobin (g/dL)	14.1	±1.9	14.3	±1.8	14.0	±2.0	0.501
Hematocrit (%)	42.5	±5.3	43.1	±5.0	42.1	±5.6	0.291
White blood cell count ($10^3/\mu L$)	11.04	±4.07	10.83	±4.13	11.21	±4.05	0.642
C-reactive protein (mg/dL)	5.21	3.42-16.70	5.28	4.25-15.20	4.92	3.02-18.60	0.302
Blood glucose (mg/dL)	105.5	95.0-120.5	107.0	97.0-127.0	102.0	94.0-117.0	0.195
Creatinine (mg/dL)	0.8	0.7-0.9	0.8	0.8-1.0	0.9	0.7-0.9	0.500
Left ventricular ejection fraction (%)	58	±4	59	±4	58	±4	0.571
RV dimension (cm)	3.6	±0.5	3.5	±0.5	3.7	±0.5	0.085

sPAP (mmHg)	41	±10	36	±9	45	±9	<0.001
TAPSE (mm)	20	±3	21	±2	19	±3	<0.001
RV dimension index (cm/m²)	1.99	±0.30	1.93	±0.27	2.04	±0.31	0.096
Heart rate (bpm)	86	±17	81	±18	90	±15	0.005
P wave axis (°)	65	±28	54	±25	73	±27	<0.001
P wave dispersion (ms)	58	±17	55	±17	60	±17	0.084
QRS axis (°)	45.0	-4.0-73.0	30.0	-14.0-59.0	61.0	4.0-80.0	0.022
QRS duration (ms)	94	±11	94	±9	93	±12	0.235
QTc duration (ms)	398.3	±44.1	396.4	±63.1	399.8	±17.7	0.308
Right bundle branch block, n (%)	15	(15.0)	3	(6.7)	12	(21.8)	0.036
QRS fragmentation, n (%)	42	(42.0)	21	(46.7)	21	(38.2)	0.395
Right QRS axis deviation, n (%)	11	(11.0)	2	(4.4)	9	(16.4)	0.059
RS time (ms)	62	±10	58	±9	65	±10	0.002
Clockwise rotation, n (%)	50	(50.0)	20	(44.4)	30	(54.5)	0.317

ppFEV1, percent-predicted forced expiratory volume in first second; FVC, forced vital capacity; RV, right ventricle; sPAP, systolic pulmonary artery pressure; TAPSE, tricuspid annular plane systolic excursion.

Table 2. Univariable and multivariable logistic regression analysis of characteristics for severe and very severe COPD prediction

	Univariable analysis of severe and very severe COPD			Multivariable analysis of severe and very severe COPD		
	Odds ratio	95% C.I.	P value	Odds ratio	95% C.I.	P value
Age (years)						
Diabetes, n (%)	0.386	0.097-1.535	0.177	-	-	-
Right ventricular dimension index (cm/m2)	1.832	0.259-12.972	0.544	-	-	-
Heart rate (bpm)	1.037	1.010-1.064	0.007	1.032	1.002-1.062	0.035
P wave axis (°)	1.031	1.012-1.050	0.002	1.025	1.005-1.046	0.015
QRS axis (°)	1.007	0.997-1.017	0.186	-	-	-
Right bundle branch block, n (%)	3.547	0.666-18.884	0.138	-	-	-
RS time (ms)	1.093	1.040-1.149	<0.001	1.081	1.021-1.145	0.007

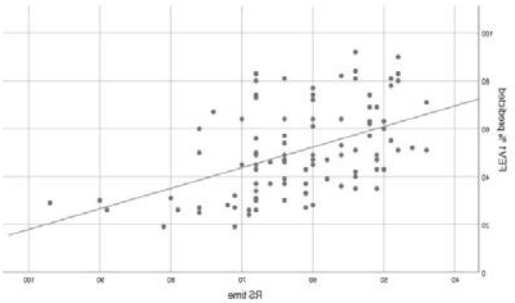

Figure 2. A plot diagram illustrating the significant negative correlation between the RS time and percent-predicted forced expiratory volume in first second (FEV1 % predicted); (r: -469; p<0.001).

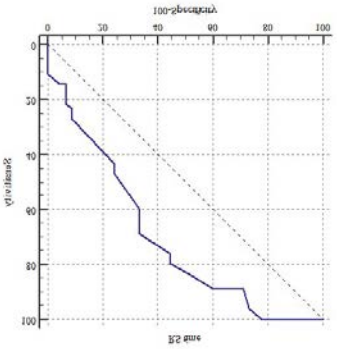

Figure 3. Receiver operating characteristic graphic to detect best cut-off value of RS time for severe COPD (GOLD 3-4) prediction. (Area under the curve = 0.714, 95% confidence interval = 0.615–0.800, $p < 0.001$).

RS time exhibited a significantly moderate negative correlation with ppFEV1. A plot diagram illustrating the correlation analysis of RS time with ppFEV1 is presented in Fig. 2.

The independent predictors of severe and very severe COPD were identified by multivariable logistic regression analysis through the use of the variables, including the age, diabetes, heart rate, P wave axis, QRS axis, RS time, right ventricular dimension index, and right bundle branch block. The results showed that heart rate, P wave axis, and RS time (odds ratio [OR] per 1 ms increase = 1.081, 95% confidence interval [CI] = 1.021–1.145, $p = 0.007$; Table 2) were independent predictors of severe and very severe COPD. Hosmer-Lemeshow test result revealed a good fit for our model. ($\chi 2 = 14.276$, $p = 0.075$).

The ROC curve analysis showed that the best RS time cutoff value for the prediction of severe and very severe COPD was 60 ms (1.5 small square on ECG), with a sensitivity of 69.1% and a specificity of 66.7% (area under the curve = 0.714, 95% CI = 0.615–0.800, $p < 0.001$; Fig. 3).

DISCUSSION

Our study indicated that a prolonged RS time was independently associated with severe and very severe COPD. Moreover, RS time demonstrated a significantly moderate negative correlation with ppFEV1, which is a surrogate for COPD severity classified based on the degree of airflow limitation.

COPD has extrapulmonary effects, the most common of which are cardiac effects[17]. Mortality is recognized to increase as the severity of COPD increases[12]. The cause of increased mortality mostly depends on cardiac involvement[11,17]. There is a close relationship between COPD, pulmonary hypertension, and right and left ventricular dysfunction[11]. The incidence of pulmonary hypertension, cor-pulmonale and coronary ischemic events increases as the severity of COPD increases[11]. The reduction of FEV1 in COPD patients increases the risk of fatal cardiovascular and non-fatal coronary events[10]. Electrocardiography can be a practical tool for the early diagnosis of any cardiac alterations related to COPD. To date, several electrocardiographic changes have been associated with the presence of COPD, such as an increase in the p wave axis, an increment in the p wave dispersion, the deviation of the QRS complex to the right axis, and a precordial clockwise rotation[18,19]. Our study found the independent predictors of severe and very severe COPD as the P wave axis, heart rate, and RS time.

The relation of the P wave axis with COPD and its severity has been stated in previous studies[20,21]. Our results confirm these previous studies. The hyperinflation of the lungs and the diaphragm's flattening affect the enormity, shape, and vector direction of the P waves and QRS complexes because of the insulating effect of air trapping and variation in the position of the heart [22]. The shift of the right atrium inferiorly as a result of the increased flattening of the diaphragm due to over-aerated lungs has been suggested as the mechanism of the increase in the P wave axis as COPD severity increases[21].

The heart rate increment with increasing GOLD stage classified by spirometry has been previously demonstrated[23]. We also found a significantly higher heart rate in patients with severe and very severe COPD than patients with mild and moderate COPD. The increase in the amount of emphysema and the severity of airflow obstruction has been correlated with impaired left ventricular filling and reduced stroke volume[24], which may entail a rise in heart rate. The requirement for more frequent beta-mimetic and anticholinergic drug usage in this group may be another occasion for a higher heart rate.

Wide and slurred S-waves in leads I, II, III, V4, V5, and V6 have been suggested to be present in COPD patients' electrocardiography[8]. Investigation of the QRS vectors points out that the wide and slurred S-waves emerge due to the right and superior direction of the late vectors and presumably arose from the crista supraventricularis hypertrophy[8,25]. RS time has been presented as a new-found ECG parameter. The prolonged RS time has been suggested to be related to the right ventricular dilation, pulmonary artery hypertension, and right ventricular dysfunction and proposed to be due to the lateness in electrical conduction and the alteration in the orientation of the QRS vector at the termination of depolarization[9,26]. The elongation of RS time duration has been demonstrated to be a predictor of pulmonary embolism[9]. Moreover, it is found to be related to short-term mortality after pulmonary embolism[26]. In our study, we observed that RS time was significantly correlated with the severity of COPD.

Some mechanisms can clarify the rationale for RS time becoming prolonged as the severity of COPD increases. An increment in right ventricular afterload

and strain is anticipated as a result of an increase in the resistance of pulmonary vasculature due to hypoxia. The right ventricular strain may cause a conduction delay in ECG and may also change the QRS complexes through structural alterations in the myocardium[22,27]. Logically, RS time tends to increase as the anatomical and functional abnormalities of the right ventricle become more prominent as the pulmonary function becomes more impaired. The results of our study also showed that systolic pulmonary artery pressure was significantly higher and the TAPSE was significantly lower in patients with severe and very severe COPD than in those with mild and moderate COPD. Although the right ventricular dimension and right ventricular dimension index were higher in the severe and very severe COPD group, this difference did not reach statistical significance. This may be due to the adaptive mechanisms in the chronic process in patients with COPD[10,28].

This study contains some limitations. First, the number of patients in each COPD grade was insufficient to compare with each other because of the low sample size. Thus, we compared COPD patients with grade 1 and grade 2 stages with COPD patients with grade 3 and grade 4 stages. Second, the fact that patients were not questioned about the treatment they received caused us not to be able to estimate the effect of treatment on RS time. Third, as our research is a cross-sectional study, no prospective information has been obtained regarding the patients' prognosis and the stability of electrocardiographic parameters over time; therefore, further larger, prospective studies are needed to clarify this issue and validate our results.

In conclusion, the increasing RS time measured from an electrocardiogram, which is one of the simplest and fastest tests used to evaluate the heart, has a direct correlation with the increasing degrees of airway obstruction. Although spirometry is used to assign the severity of COPD in practice, this evaluation requires the patient's severe participation and cooperation. Also, spirometric evaluation is more time-consuming than electrocardiographic evaluation, and interpretation of spirometry results is more complicated than the interpretation of an electrocardiogram. An RS time above 60 ms (1.5 small square on ECG) is associated with advanced airway obstruction. An RS time higher than 60 ms can be used as a screening tool for the detection of severe and very severe COPD with reasonable sensitivity and specificity. Since patients with severe COPD are more prone to cardiac diseases than other COPD patients, early and easy detection of these patients with RS time measurement obtained from an electrocardiogram can enable us to follow these patients more closely from a cardiac point of view and to start treatment for possible cardiac diseases earlier.

Yazar Katkıları: Çalışma konsepti/Tasarımı: İY, İR; Veri toplama: İY, PÖY, MSG; Veri analizi ve yorumlama: İY, MSG, İR, YK; Yazı taslağı: İY, ÇÖ; İçeriğin eleştirel incelenmesi: CB, ÇÖ; Son onay ve sorumluluk: İY, PÖY, HS, MSG, İR, YK, CB, ÇÖ; Teknik ve malzeme desteği: PÖY; Süpervizyon: İY, CB; Fon sağlama (mevcut ise): yok.
Etik Onay: Bu çalışma için Kafkas Üniversitesi Tıp Fakültesi Etik Kurulundan 04.11.2020 tarih ve 234/13 sayılı kararı ile etik onay alınmıştır.
Hakem Değerlendirmesi: Dış bağımsız.
Çıkar Çatışması: Yazarlar çıkar çatışması beyan etmemişlerdir.
Finansal Destek: Yazarlar finansal destek beyan etmemişlerdir.
Author Contributions: Concept/Design : İY, İR; Data acquisition: İY, PÖY, MSG; Data analysis and interpretation: İY, MSG, İR, YK; Drafting manuscript: İY, ÇÖ; Critical revision of manuscript: :CB, ÇÖ; Final approval and accountability: İY, PÖY, HS, MSG, İR, YK, CB, ÇÖ; Technical or material support: PÖY; Supervision: İY, CB; Securing funding (if available): n/a.
Ethical Approval: Ethical approval was obtained for this study from the Ethics Committee of Kafkas University Faculty of Medicine with the decision dated 04.11.2020 and numbered 234/13.
Peer-review: Externally peer-reviewed.
Conflict of Interest: Authors declared no conflict of interest.
Financial Disclosure: Authors declared no financial support

REFERENCES

1. Biener AI, Decker SL, Rohde F. Prevalence and treatment of chronic obstructive pulmonary disease (COPD) in the United States. JAMA. 2019;322:602.
2. Hurd S. The impact of COPD on lung health worldwide: epidemiology and incidence. Chest. 2000;117:1-4.
3. Sin DD, Wu L, Man SF. The relationship between reduced lung function and cardiovascular mortality: a population-based study and a systematic review of the literature. Chest. 2005;127:1952-9.
4. Sakao S, Voelkel NF, Tatsumi K. The vascular bed in COPD: pulmonary hypertension and pulmonary vascular alterations. Eur Respir Rev. 2014;23:350-5.
5. Peinado VI, Pizarro S, Barberà JA. Pulmonary vascular involvement in COPD. Chest. 2008;134:808-14.
6. Hilde JM, Skjørten I, Grøtta OJ, Hansteen V, Melsom MN, Hisdal J et al. Right ventricular dysfunction and remodeling in chronic obstructive pulmonary disease without pulmonary hypertension. J Am Coll Cardiol. 2013;62:1103-11.
7. Scott RC. The electrocardiogram in pulmonary emphysema and chronic cor pulmonale. Am Heart J. 1961; 61: 843-5.
8. Burch GE, Depasquale NP. The electrocardiographic diagnosis of pulmonary heart disease. Am J Cardiol. 1963;11:622-38.

9. Rencuzogullari I, Çağdaş M, Karabağ Y, Karakoyun S, Çiftçi H, Gürsoy MO et al. A novel ECG parameter for diagnosis of acute pulmonary embolism: RS time: RS time in acute pulmonary embolism. Am J Emerg Med. 2019;37:1230-36.
10. Buklioska-Ilievska D, Minov J, Kochovska-Kamchevska N, Prgova-Veljanova B, Petkovikj N, Ristovski V et al. Cardiovascular comorbidity in patients with chronic obstructive pulmonary disease: echocardiography changes and their relation to the level of airflow limitation. Open Access Maced J Med Sci. 2019;7:3568-73.
11. Kaushal M, Shah PS, Shah AD, Francis SA, Patel NV, Kothari KK. Chronic obstructive pulmonary disease and cardiac comorbidities: A cross-sectional study. Lung India. 2016;33:404-9.
12. Bhatta L, Leivseth L, Mai XM, Henriksen AH, Carslake D, Chen Y et al. GOLD classifications, COPD hospitalization, and all-cause mortality in chronic obstructive pulmonary disease: The HUNT Study. Int J Chron Obstruct Pulmon Dis. 2020;15:225-33.
13. Rabe KF, Hurd S, Anzueto A, Barnes PJ, Buist SA, Calverley P et al; Global Initiative for Chronic Obstructive Lung Disease. Global strategy for the diagnosis, management, and prevention of chronic obstructive pulmonary disease: GOLD executive summary. Am J Respir Crit Care Med. 2007;176:532-55.
14. Dilaveris PE, Gialafos EJ, Sideris SK, Theopistou AM, Andrikopoulos GK, Kyriakidis M et al.: Simple electrocardiographic markers for the prediction of paroxysmal idiopathic atrial fibrillation. Am Heart J. 1998;135:733-8.
15. Bazett HC. An analysis of time relations of electrocardiograms. Heart. 1920;7:353-370.
16. Standardization of Spirometry, 1994 Update. American Thoracic Society. Am J Respir Crit Care Med. 1995;152:1107-36.
17. Gupta NK, Agrawal RK, Srivastav AB, Ved ML. Echocardiographic evaluation of heart in chronic obstructive pulmonary disease patient and its co-relation with the severity of disease. Lung India. 2011;28:105-9.
18. Rodman DM, Lowenstein SR, Rodman T. The electrocardiogram in chronic obstructive pulmonary disease. J Emerg Med. 1990;8:607-15.
19. Cimci M, Borekci S, Kilickiran Avci B, Hysi E, Ongen HG, Karadag B et al. Assessment of atrial electromechanical delay and P wave dispersion in patients with chronic obstructive pulmonary disease.T urk Kardiyol Dern Ars. 2020;48:263-69.
20. Spodick DH. Electrocardiographic studies in pulmonary disease. II. Establishment of criteria for the electrocardiographic inference of diffuse lung diseases. Circulation. 1959;20:1073-4.
21. Chhabra L, Sareen P, Perli D, Srinivasan I, Spodick DH. Vertical P-wave axis: the electrocardiographic synonym for pulmonary emphysema and its severity. Indian Heart J. 2012;64:40-2.
22. Larssen MS, Steine K, Hilde JM, Skjørten I, Hodnesdal C, Liestøl K et al. Mechanisms of ECG signs in chronic obstructive pulmonary disease. Open Heart. 2017;4: e000552.
23. Warnier MJ, Rutten FH, Numans ME, Kors JA, Tan HL, de Boer A et al. Electrocardiographic characteristics of patients with chronic obstructive pulmonary disease. COPD. 2013;10:62-71.
24. Barr RG, Bluemke DA, Ahmed FS, Carr JJ, Enright PL, Hoffman EA et al. Percent emphysema, airflow obstruction, and impaired left ventricular filling. N Engl J Med. 2010;362:217-27.
25. Burch GE, DePasquale N. The electrocardiogram, spatial vectorcardiogram, and ventricular gradient in congenital ventricular septal defect. Am Heart J. 1960;60:195-211.
26. Gümüşdağ A, Burak C, Suleymanoglu M, Yesin M, Tanık VO, Karabağ Y et al. The predictive value of RS time for short term mortality in patients with acute pulmonary embolism. J Electrocardiol. 2020;62:94-9.
27. Park DH, Cho KI, Kim YK, Kim BJ, You GI, Im SI et al. Association between right ventricular systolic function and electromechanical delay in patients with right bundle branch block. J Cardiol. 2017;70:470-75.
28. Watz H, Waschki B, Meyer T, Kretschmar G, Kirsten A, Claussen M et al. Decreasing cardiac chamber sizes and associated heart dysfunction in COPD: role of hyperinflation. Chest. 2010;138:32-8.