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Is the Rısk of Atrıal Fıbrıllatıon More in the Right Atrıum then in the Left Atrıum in Patients with Newly Dıagnosed Copd? Runnıng Head: Rısk of Rıght Atrıal Fibrillatıon in Copd Patıents

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Abstract:-

➢ ***Background:***

Atrial Fibrillation (AF) is one of the most common rhythm disorders in patients with Chronic Pulmonary Obstructive Disease (COPD). P wave dispersion (PWD) is a predictor of AF. This study aims to evaluate the relationship between COPD and PWD using the right and left precordial leads.

➢ ***Methods:***

The study group consisted of fifty newly diagnosed COPD patients, having obstructive findings in the Respiratory Function Test (RFT). The control group consisted of fifty-two healthy subjects, having no obstructive outcomes in RFT, age, gender, and smoking periods matched with the study group. Standard 12-lead Electrocardiography (ECG) + right precordial leads were taken for each group. PWD values were measured in left precordial (V_{1-6}L) and right precordial leads (V_{1-6}R), and calculations were made.

➢ ***Results:***

In the study group, PWD values in the right precordial leads (PWDR) were 47.81 ± 6.37 ms, while PWD values in the left precordial leads (PWDL) were 41.97 ± 3.81 ms. The difference between the two values was statistically significant (t = 5.64, p <0.01). PWDR value was 47.81 ± 6.37 ms in the study group, and PWDR value was 36.69 ± 3.84 ms in the control group. The difference between the two groups was statistically significant (t = 10.72, p <0.01). In the study group, there was a statistically significant, moderate negative correlation between PWDR and systolic pulmonary artery pressure (sPAP), and moderate positive correlation between PWDR and forced expiratory volume in one second (FEV_1) (Respectively, r=0.667, p<0.01 ve r=-0.71, p<0.01).

➢ ***Conclusion:***

In newly diagnosed COPD patients, the risk of AF originated from the right atrium was found to be higher than the AF risk than the left atrium, and the AF risk originated from the right atrium in the control group.

Keywords:- *Risk, Atrial Fibrillation, Chronic Pulmonary Obstructive Disease, Respiratory Function Test, Systolic Pulmonary Arterial Pressure.*

I. INTRODUCTION

COPD not only affects the lungs but also affects many organs. The cardiovascular system is one of the most affected organs of COPD. Cardiovascular complications are the most common cause of morbidity and mortality in COPD patients [1]. AF is one of the most common rhythm disorders in patients with COPD. The incidence of AF is in COPD patients higher than the healthy population [2].

There are many risk factors for AF development in COPD patients. Changes in right or left atrial dimensions, decreased oxygenation, increased pulmonary systolic and diastolic pressure, hypercapnia, chronic low-density inflammation, endothelial dysfunction, $beta_2$ sympathomimetic agents used in bronchodilator treatment, and primarily increased automaticity, which may play a role in the pathogenesis of AF originating from the right atrium, can play a role in development of AF [3,4]. Patients with mild COPD have been shown to have a higher AF risk than healthy subjects and patients with mild COPD [5]. It has also been shown that there is a close relationship between AF and low FEV_1 values [6]. Although a close association between AF and COPD patients with low FEV_1 values is known, only a few studies are examining this relationship and investigating the underlying causes [2]. AF was found to be more prevalent in COPD patients with hypoxia, hypercapnia, and pulmonary hypertension [7]. However, so far, the relationship between peripheral oxygen saturation, FEV_1, and pulmonary pressure, and PWD has not been investigated.

The risk of AF development can be observed by frequently monitoring the right atrial diameter by Transthoracic echocardiography (TTE) and, followed by daily follow-up of atrial extra-systoles and by PWD [8]. PWD can be measured on superficial ECG. PWD can be calculated by subtracting the shortest P wave duration from the most prolonged P wave duration on the superficial ECG. Elongation in PWD indicates the transmission delay at the inter-atrial or intra-atrial level and heterogeneity in conduction [9]. PWD was found to be prolonged in COPD patients with exacerbation compared to patients with stable phase COPD [10]. PWD is an AF predictor in COPD patients, independent of pulmonary functions, hypoxia and hypercapnia, electrolyte disturbances, and right and left atrial functions. In addition, PWD prolongation was found to be closely related to the presence of AF in COPD

patients [11]. However, PWD measurement was calculated in the standard 12-lead ECG. The right and left precordial leads were not examined separately. Our study aimed to investigate PWD independently in both left and right precordial leads, in which side of atria PWD was much more affected and to examine the relationship between peripheral oxygen saturation-pulmonary pressure-pulmonary function test and PWD.

II. MATERIALS AND METHODS

A. Study Design and Population:

Fifty patients who were admitted to the outpatient clinic of chest disease with dyspnea and had an irreversible obstruction in the Pulmonary Function Test, between January-2019 and May 2019 were included in the study. This group was named as the study group. There were 38 males and 12 females in the study group. The mean age of the study group was 65.67 ± 11.67 years. Patients were subjected to 12-lead ECG and Transthoracic echocardiography (TTE) after routine physical and blood examination, and RFT. After ECG and TTE, peripheral oxygen saturation (POS) was measured after five minutes of rest.

Furthermore, calculations are made. The control group consisted of fifty-two healthy individuals who were admitted to the cardiology outpatient clinic with chest pain, had no pathology in their examinations, and were similar in age and gender to the study group. There were 40 males and 12 females in the control group. The mean age of the control group was 66.45 ± 10.92 years. Exclusion criteria were having previously received AF treatment or AF treatment, having a systemic disease such as hypertension or diabetes mellitus, having structural heart disease in TTE, the presence of growth or hypertrophy in any of the heart cavities, having previously diagnosed as coronary artery disease and to have treatment, to have anemia, thyroid dysfunction or electrolyte disorder, to get a diagnosis of COPD or to receive bronchodilator treatment, to be smoking, to have a diagnosis of cancer or to receive a treatment, to have a pacemaker, to be left or right bundle branch block, to be taking antidepressant medication, to get replacement therapy for deficiency of vitamins, to be ablated due to pre-excitation syndrome, being too disabled to be able to taken an ECG, being too keen to do the pulmonary function test or not being able to do enough RFT. Written permission was obtained from all patients before the study.

B. Respiratory Function Test (RFT):

The patients were taken to the RFT in a sitting position after five minutes of rest. After deep inspiration, the patient was asked to perform expiration with the full force to the spirometer. The same procedure was performed three times in succession. The values obtained from these three measurements were determined using averages. Patients with FEV_1 / FVC ratio less than 70% were considered to have an obstruction and were included in the study [12]. Patients who could not make enough expiration were excluded from the study. Patients with average FEV_1, FVC, and FEV_1/FVC values were included in the control group. RFT was measured at the beginning and the end of the TET.

C. Electrocardiography (ECG):

The standard 12-lead and right precordial leads ($V_{1-6}R$) ECG recordings were obtained at the supine position with paper at a speed of 25 mm/sec and 10 mm/mV amplitude by using standard ECG system (CardiofaxV model 9320, Nihon Kohden, Tokyo, Japan). ECG length was 10 seconds, and therefore depending on the heart rate, there were 4-6 beats per lead. ECGs were measured manually by the use of a magnifying glass (TorQ, 150 mm Digital Caliper LCD) by two blinded cardiologists having no information about the patients. The RR interval and P wave dispersion (PWD) of the ECG recordings were measured manually with an accuracy of 0.01 mm of a digital compass. In addition to left precordial leads, according to Boston scientific ECG screening tools, right ventricular precordial leads were taken by using the vertical mirror image of the left ventricular precordial leads and displacement of right and left extremity electrodes [14]. Left fourth intercostal space of sternum side (1 cm) was used as V_1R, right fourth intercostal space of sternum side (1 cm) was used as V_2R, right fifth intercostal space and right midclavicular line intersection was used as V_4R, midpoint of V_4R -V_2R line was used as V_3R, as V_5R intersection point of right front axillary line and fifth intercostal space and the right middle axillary line intersection point was used as V_6R. This measurement was calculated at least both in three right and left precordial leads (V_{2-5} precordial right and left leads) and three consecutive P waves in one lead for PWD. The data of ECG with papers at a speed of 25 mm/second and amplitude of 10 mm/mV, measured in mm with the digital caliper, were calculated as millisecond multiplied by 40.

D. Pulse Oximetry:

POS values of the patients was measured by pulse oximetry. The measurements were taken under normal weather conditions when the patients were in the supine position and after five minutes of rest. During a one-minute pulse oximetry measurement, the most common value was recorded in the display. POS was indicated as %.

E. Transthoracic Echocardiography (TTE):

All subjects underwent a two-dimensional echocardiography examination. We obtained standard parasternal long-axis, mid-ventricular short-axis, long apical axis, apical 2- and 4-chamber images with the Philips HD11XE, 2012 Netherland. Continuous Wave Doppler of the tricuspid valve failure (TR) tracing was used to measure the pressure difference between the right ventricle and right atrium. In the Bernoulli formula, the value obtained by the continuous wave (CW) over TR was replaced, and the pressure difference between the right ventricle and the right atrium was calculated. The value obtained from the Bernoulli formula is traditionally calculated by the addition of right atrial pressure to

calculate systolic pulmonary arterial pressure. However, the latest ESC guidelines recommend the use of the TR max without additional right atrial pressure [13]. We used the V max value obtained from TR by simply inserting into the formula of Bernoulli.

III. RESULTS

There was no statistically significant difference between the study and control group in terms of socio-demographic properties and basal clinic findings (Table 1).

There was a statistically significant difference between the study and control group in terms of PWDR. Besides, there was no statistically significant difference between the study and control group in terms of PWDL. Furthermore, there were statistically significant differences between the study and control group in terms of sPAP, FEV1, FVC, FEV1/FVC, and POS values (Table 2).

In the study group, there were statistically significant, and moderate positive correlations were weak between PWDR and sPAP, FEV1, and POS values. There was a statistically significant and weak positive correlation between PWDL and sPAP values. Furthermore, there were statistically significant and weak negative correlation between PWDL and FEV_1, FEV_1/FVC, and POS values. In the control group, there were statistically significant and weak positive correlations between PWDR-PWDL and sPAP values. Furthermore, there were no correlations between PWDR-PWDL and FEV_1 and POS values of the control group (Figure 1-4, Table 3).

IV. DISCUSSION

Since PWD is an independent predictor for AF, and since there are only a few studies in the literature in this area, we have found it necessary to do this study. Until now, right and left precordial leads have not been studied separately to investigate PWD. Studies have generally been performed on standard 12-lead ECGs. In our study, we examined PWD in both the right and left anterior leads and tried to determine which side of atria was more affected in COPD patients. In the study group, we found that PWD in the right precordial leads (PWDR) was more affected, and we found statistically significant correlations with sPAP, RFT, and POS values. In addition, in the control group, there was a statistically significant positive correlation between PWDR and sPAP, but there was no statistically significant correlation between RFT and POS values.

AF is more common in patients with COPD than in the healthy population [15]. There are many risk factors for AF development in COPD patients. Some of these are reduced peripheral oxygen saturation, hypercapnia, diastolic dysfunction, oxidative stress, endothelial dysfunction, chronic inflammation, significant changes in right or left atrium dimensions and some beta2 sympathomimetic agents used in the treatment of COPD. AF is usually caused by auto-acquired foci in the right atrium [3]. But when the event was examined based on PWD, so far this has not been investigated. In our study, the fact that PWDR was significantly prolonged in terms of the control group and PWDL was consistent with the literature.

In the studies performed, the rate of AF in patients with moderate COPD was found to be higher than in the healthy population and in patients with mild COPD [3,16]. Patients with AF may have many complications with or without COPD. The most important of these are loss of atrial contribution and predisposition to thromboembolism. Cerebrovascular events are the most common and most mortal complication of AF.

In terms of mortality, COPD is an independent predictor of major cardiac events, cardiovascular death, and all-cause death in patients with non-valvular AF [17].

So far, many predictors of AF have been emphasized. Some of these are right or left atrial diameter, presence of frequent atrial extra-systoles, and PWD. PWD is the easiest to measure and follow. PWD reflects in-homogenous and fractionated propagation of sinus impulse [18]. PWD is defined as the difference between the longest P wave and the shortest P wave in the superficial electrocardiogram (ECG). In patients with atrial fibrillation, it is well known that increased P-wave duration and PWD reflect the prolongation of intra- and inter-atrial conduction time and non-homogeneous spread of sinus impulses [19]. It has been reported that PWD may be prolonged secondary to pulmonary hypertension without changes in right atrial size in patients with COPD [20]. Also, in a study, it was found that PWD prolonged much more in COPD patients with AF than in non-AF COPD patients and control group [11]. However, the standard 12-lead ECG was used in this study, and it was not evaluated separately in the right and left precordial leads.

PWD is adopted as a risk factor for AF with or without a systemic disease [21]. PWD has been shown to be longer in many diseases with higher AF risk than healthy individuals. These include hypertension and diabetes mellitus [22,23]. PWD has also found to be prolonged in patients with paroxysmal atrial fibrillation[24].

V. CONCLUSION

Close monitoring of PWD separately in both the right and left precordial leads and taking more precautions may contribute to the early treatment of the complications of AF and reduce the cost of complications. Especially in COPD patients, it will be more specific and useful to calculate PWD separately in the right and left precordial leads to be able following closely the risk of AF.

ETHİCAL APPROVAL

All procedures performed in studies involving human participants were in accordance with the ethical standards of the National Research Committee and with the 1964 Helsinki declaration and its later comparable ethical standards. There is no Ethics Committee in our hospital. Written permission was obtained from the hospital administration for the study.

CONFLICT OF INTEREST

The authors declare that they have no conflict of interest. There is no source(s) of support in the form of grants, equipment, drugs. All expenses are paid by the authors. The authors state that the manuscript has been read and approved by all the authors, that the requirements for authorship as stated earlier in this document have been met, and that each author believes that the manuscript represents honest work.

ABBREVIATION LIST

AF: Atrial Fibrillation
COPD: Chronic Obstructive Pulmonary Disease
PWD: P Wave Dispersion
RFT: Respiratory Function Test
ECG: Electrocardiography
V1-6R: Right Anterior Precordial Leads
V1-6L: Left Anterior Precordial Leads
PWDR: P Wave Dispersion in Right Precordial Leads
PWDL: P Wave Dispersion in Left Precordial Leads
sPAP: Systolic Pulmonary Arterial Pressure
POS: Peripheral Oxygen Saturation
TTE: Transthoracic Echocardiography
FEV1: Forced Expiratory Volume in 1 Second
FVC: Forced Vital Capacity
FEV1/FVC: Forced Expiratory Volume in 1 Second/ Forced Vital Capacity
CW: Continous Wave
TR: Tricuspid Valve Regurgitation
HR: Heart Rate
BMI: Body Mass Index
LDL: Low-Density Lipoprotein
B/MN: Beat/Minute

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Variables	Study Group (n=50)	Control Group (n=52)	T or Z-value	P-value
Age, Years	65.67 ± 11.67	66.45 ± 10.92	0.34	0.78
Males, %	76	76.92	0.012	0.91
Females, %	24	23.08	0.012	0.91
BMI, kg/m^2	26.48±2.69	26.05±1.95	0.92	0.35
Cigarette smoking, packages/year	24.66±5.34	23.91±4.99	0.73	0.46
Blood Pressure, mm Hg	122.85±10.84	119.93±11.73	1.30	0.19
Basal HR, b/mn	77.83±12.69	78.37±11.82	-0.22	0.82
Total Cholesterol, m/dl	202.57±39.51	199.69±34.59	0.39	0.69
LDL, mg/dl	123.64±22.73	126.38±25.27	0.57	0.56
Triglyceride, mg/dl	156.47±35.23	161.69±39.73	0.70	0.48
Sodium, mEq/L	139.58±3.51	140.28±2.95	1.09	0.27
Calcium, mg/dl	9.84±0.95	9.56±0.84	1.57	0.11
Magnesium, mg/dl	2.34±0.25	2.32±0.19	0.45	0.64
Hemoglobin, g/dl	14.34±1.41	13.95±1.32	1.44	0.15

Table 1:- The Socio-demographic properties and basal clinic findings of the study and control group
Abbr: BMI; Body Mass Index, LDL; Low-Density Lipoprotein, b/mn; Beat/ Minute

Variable	**Study Group**	**Control Group**	**T-value**	**P-value**
PWDR, ms	47.81±6.37	36.69±3.84	10.72	<0.01
PWDL, ms	41.97±3.81	41.81±4.61	0.20	0.83
sPAP, mm Hg	35.26±3.39	18.98±2.11	29.28	<0.01
FEV_1, Liter	2.37±0.16	2.98±0.21	-16.45	<0.01
FVC, Liter	3.72±0.18	3.56±0.26	3.63	<0.01
FEV_1/FVC, %	63.71±2.61	83.92±2.17	-42.68	<0.01
POS, %	92.85±1.87	96.82±1.52	-11.85	<0.01

Table 2:- The comparison of sPAP, PWD, RFT, and POS values of the study and control group
Abbr: PWDR; P Wave Dispersion in the right precordial leads, PWDL; P Wave Dispersion in the left precordial leads, sPAP; Systolic Pulmonary Arterial Pressure, FEV1; Forced Expiratory Volume in 1 second, FVC; Forced Vital Capacity, POS; Peripheral Oxygen Saturation

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The Study Group (n=50)				
Variable 1	Variable 2	Correlation	R-value	P-value
PWDR	sPAP	Moderate Positive	0.66	<0.01
PWDR	FEV_1	Moderate Negative	-0.71	<0.01
PWDR	POS	Moderate Negative	-0.63	<0.01
PWDR	FEV1/FVC	Moderate Negative	-0.70	<0.01
PWDL	sPAP	Weak Positive	0.43	<0.01
PWDL	FEV_1	Weak Negative	-0.40	<0.01
PWDL	POS	Weak Negative	-0.28	0.048
PWDL	FEV1/FVC	Weak Negative	-0.37	<0.01
The Control Group (n=52)				
PWDR	sPAP	Weak Positive	0.36	<0.01
PWDR	FEV_1	No Correlation	-0.24	0.08
PWDR	POS	No Correlation	-0.11	0.44
PWDR	FEV_1/FVC	No Correlation	-0.04	0.78
PWDL	sPAP	Weak Positive	0.28	0.04
PWDL	FEV_1	No Correlation	-0.24	0.08
PWDL	FEV_1/FVC	No Correlation	-0.11	0.44
PWDL	POS	No Correlation	-0.08	0.57

Table 3:- The Correlations between PWDR and other values of the study group and control group
Abbr: PWDR; P Wave Dispersion in the right precordial leads, PWDL; P Wave Dispersion in the left precordial leads, sPAP; Systolic Pulmonary Arterial Pressure, FEV_1; Forced Expiratory Volume in 1 second, FVC; Forced Vital Capacity, POS; Peripheral Oxygen Saturation

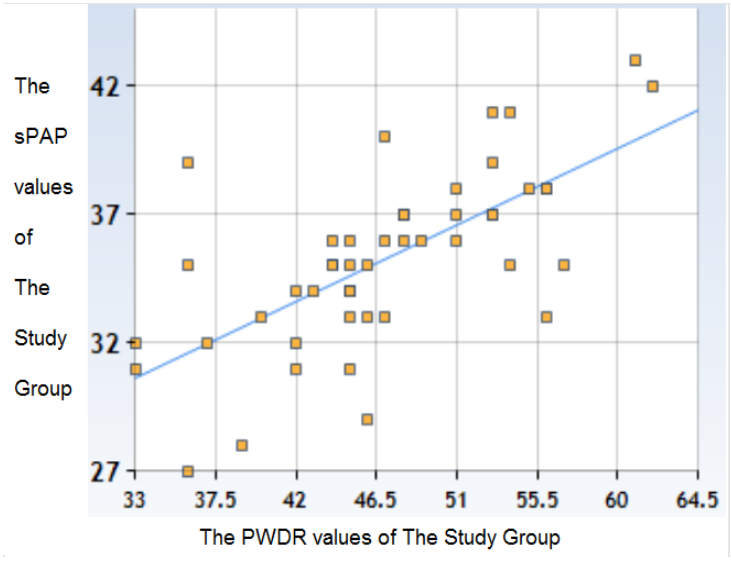

Fig 1:- Linear Regression Graphic of PWDR values and sPAP values of The Study Group
Abbr: sPAP; Systolic Pulmonary Arterial Pressure, PWDR; P Wave Dispersion in the Right Precordial Leads. Correlation: R=0.66, p<0.01, Moderate Positive Correlation

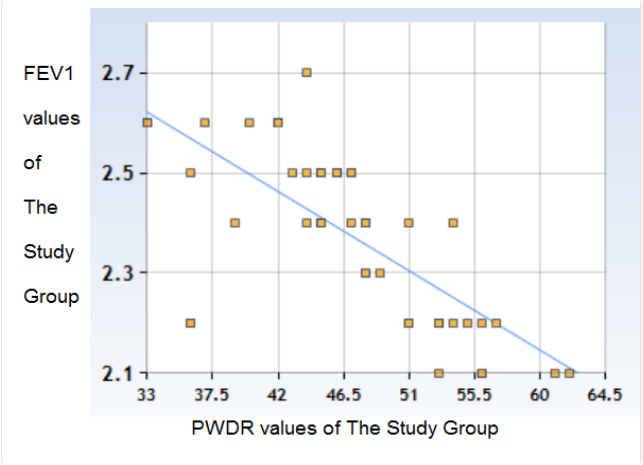

Fig 2:- Linear Regression Graphic of PWDR and FEV1 values of The Study group
Abbr: FEV1; Forced Expiratory Volume in 1 Second, PWDR; P Wave Dispersion in the Right Precordial Leads. Correlation: R=-0.71, p<0.01, Moderate Negative Correlation

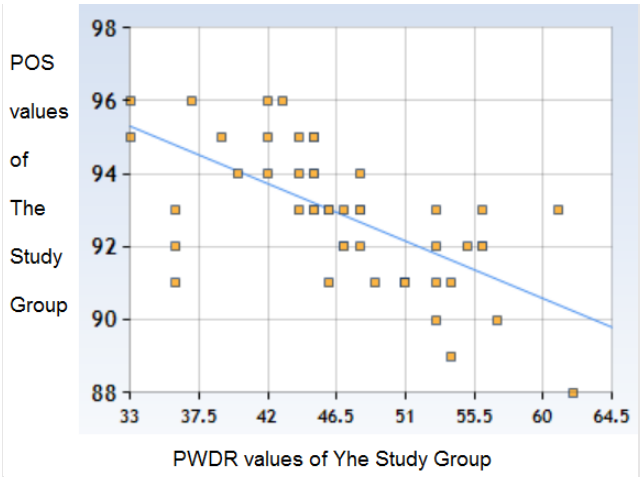

Fig 3:- Linear regression Graphic of PWDR and POS values of The Study Group
Abbr: POS; Peripheral Oxygen Saturation, PWDR; P Wave Dispersion in the Right Precordial Leads. Correlation: R=-0.63, p<0.01, Moderate Negative Correlation

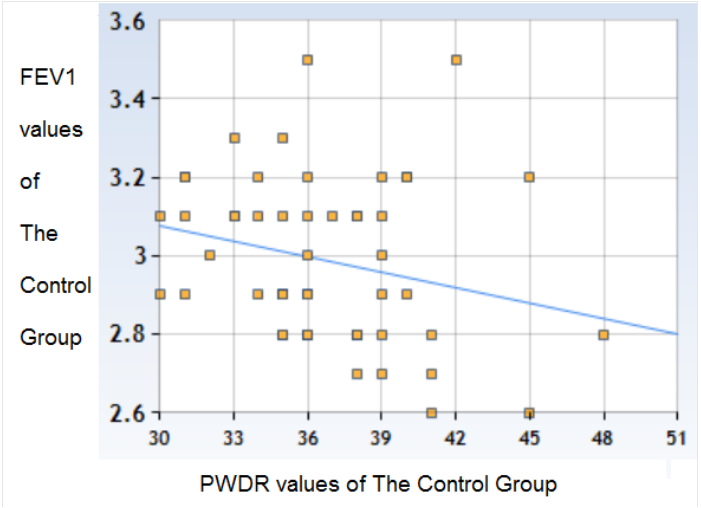

Fig 4:- Linear Regression Graphic of PWDR and FEV1 values of The Control Group
Abbr: FEV1; Forced Expiratory Volume in 1 Second, PWDR; P Wave Dispersion in the Right Precordial Leads. Correlation: R=-0.24, p=0.08, No Correlation.