ORIGINAL ARTICLE

Evaluation of the Cardiac Status of Newly Diagnosed Chronic Obstructive Pulmonary Disease Patients at First Admission

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Abstract

Objective: We aimed to detect possible cardiac pathologies early by cardiologically evaluating patients newly diagnosed with chronic obstructive pulmonary disease (COPD).

Materials and methods: The study included 69 newly diagnosed COPD patients who came to our outpatient clinic and 39 individuals who had smoked for at least 20 years. Both groups were evaluated in chest diseases and grouped according to the Global Initiative for Chronic Obstructive Lung Disease (GOLD) and then evaluated in the cardiology outpatient clinic.

Results: Of the COPD patients included in the study, 49 (71.01%) were male, and 20 (29.99%) were female. The COPD patient group was divided into three groups based on the 2023 GOLD strategy report. There were a total of 38 (35.2%) patients in the GOLD-A group, 28 (73.7%) male and 10 (26.3%) female. The right branch block was seen in 15 (13.9%) of all participants. A total of 14 of them were in the COPD group, and one of them was in the control group. Of the COPD patients with right branch block, 10 (26.3%) were in the GOLD-A group, three (12.5%) were in the GOLD-B group, and one (14.3%) was in the GOLD-E group. Diastolic dysfunction was seen in 38 (55.1%) COPD patients and 14 (35.9%) control groups. Of the patients with COPD with diastolic dysfunction, 22 (57.9%) were in the GOLD-A group, and 20 (55.3%) of them were in grade 1. Mitral valve disease was detected in 19 (27.5%) of the patients with COPD, tricuspid valve disease in 18 (26.1%), and aortic valve disease in two (2.9%) patients.

Conclusion: We have seen that a significant number of our patients have started their cardiological pathologies during the initial diagnosis. With this strategy, cardiac pathologies can be intervened early, and better results can be obtained in the management of COPD.

Keywords: Cardiovascular disease, Chest, Chronic obstructive pulmonary disease.

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Introduction

Chronic obstructive pulmonary disease (COPD) is a miscellaneous lung illness manifested by habitual respiratory symptoms due to airflow obstruction of the bronchi (bronchitis, bronchiolitis) and/or air sacs (emphysema), causing permanent, frequently progressive tailwind restriction.[1,2] In the records of the World Health Organization (WHO), COPD is seen as the fourth cause of death, and 3 million people lose their lives every year due to this reason. In our country, COPD is the third cause of death.[3] During the natural course of COPD, many extrapulmonary findings and comorbidities occur that undergo the follow-up of the cases and lead to changes in prognosis and quality of life.[4] Deterioration in psychological status is reported at increasing rates in COPD patients. The prevalence of psychological distress was found to be 58% in hospitalized patients due to COPD, 42% in COPD outpatient clinic patients, and 21% in other respiratory diseases.[5] High rates of cardiac morbidity and mortality are seen in COPD cases. The rate of coronary heart disease and heart failure (HF) in patients diagnosed with COPD over the age of 45 is higher than in those without COPD.[6] Cardiovascular disease (CVD) and COPD are responsible for >50% of smoking-related deaths and are often found together.[7] COPD is both an important and self-contained risk factor for CVD.[8] Cardiovascular pathologies are important determinants of morbidity and mortality of COPD. The copresence of COPD with HF is associated with a poor prognosis.[9,10] Conventional echocardiography showed that advanced COPD was associated with biventricular dysfunction.[11]

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The relationship between two diseases can be said by similar/common risk factors and contraptions such as smoking, hypoxia, chronic systemic inflammation, advanced age, oxidative stress, endothelial dysfunction, and sympathetic activation.[12,13]

Due to the overlapping symptoms and signs of COPD and CVD, patients' complaints and clinical findings are often attributed to one disease, while the other disease, if any, can be ignored. For this reason, treatment and management of diseases are often incomplete in some of the cases.[14] Early evaluation and initiation of treatment of COPD and CVD patients may reduce morbidity, mortality, and economic burden.[15,16]

Based on the above information, we thought that some of the newly diagnosed COPD patients may have cardiac diseases.

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We aimed to detect possible cardiac pathologies early by cardiologically evaluating patients who had not been diagnosed with any respiratory system and heart disease before starting COPD treatment. Thus, we thought that we could reduce the failure of cardiac-related treatment that may be seen in some patients during the treatment of COPD and that the cardiac pathologies we detected could be intervened early.

Materials and Methods

Participants and Study Design

This study was conducted between 1st May 2022 and 1st February 2023 in the chest diseases outpatient clinic of our hospital. Individuals between the ages of 30 and 55, with complaints of cough, sputum, and shortness of breath, smoking for at least 20 years, and without any known lung and heart disease, were included in the study.

After the detailed anamnesis of the individuals participating in the study was taken in the outpatient clinic, the first examinations were made, pulmonary function tests were performed, and posterior-anterior chest radiographs were taken. Patients whose diagnosis of COPD was confirmed by spirometry test results and clinical findings and, depending on the Global Initiative for Chronic Obstructive Lung Disease (GOLD) diagnostic criteria, were accepted for the study.[1]

Individuals who were admitted to the study and were not diagnosed with COPD, who have been smokers for at least 20 years, between the ages of 30 and 55, and who did not have a known lung or heart disease were included in our study as a control group.

Individuals who smoked for <20 years were not included in this study. Those who were previously diagnosed with known lung disease and heart disease, those who did not receive sufficient information for the identification criteria, and those with sleep-breathing disorder disease were excluded from the study. Since the spirometry test requires sufficient physical strength and adaptation to the test maneuvers, patients who could not perform the spirometry test correctly were excluded from the study. Patients who have undergone surgery in the last 3 months, those with orthopedic vertebral anomalies, and patients with neurological diseases (Alzheimer's, dementia, cerebrovascular disease, etc.) were not included in the study.

The consent form was filled out to the patients who accepted the study and the control group, and they were referred to the cardiology outpatient clinic. In the cardiology outpatient clinic, the patients were re-anamnesis and examined by the cardiologist. Patients underwent electrocardiography and echocardiography. According to the examinations and evaluations, the data of the appropriate patients were recorded in the Statistical Package for Social Sciences (SPSS) program. Permission was obtained from the ethics committee of our hospital to conduct this study (05.04.2022/2034). The step in the selection phase of study patients is shown in Figure 1.

Spirometry

All patients underwent spirometry tests in the pulmonary function test laboratory of our hospital. The spirometry test was performed by a technician dressed in appropriate equipment to prevent contamination against all respiratory infections, primarily considering COVID-19 infection. It is planned to evaluate lung functions with spirometry tests and to support the diagnosis of COPD. The spirometry procedure was performed on patients in accordance with the guidelines of the American Thoracic Society (ATS)–European Respiratory Society (ERS).[17,18] The tests were performed using the MIR Spirolab II (Rome/Italy) instrument. Breathing test maneuvers were explained to the patients, and three spirograms were performed. The best result bearing the reproducibility and acceptability measures was included in the study.

Electrocardiographic Analysis

The 12-lead electrocardiogram (ECG) was for arrhythmias, premature ventricular contractions, premature supraventricular contractions, and bundle branch blocks, evaluated by the two double-blinded consultant cardiologists.

Echocardiographic Analysis

The echocardiography examination was performed with an EPIQ 7C xMATRIX ultrasound (USG) system (Philips Healthcare, Bothell, Washington, United States of America). In all populations, left-ventricle dimensions and left-ventricle ejection fraction

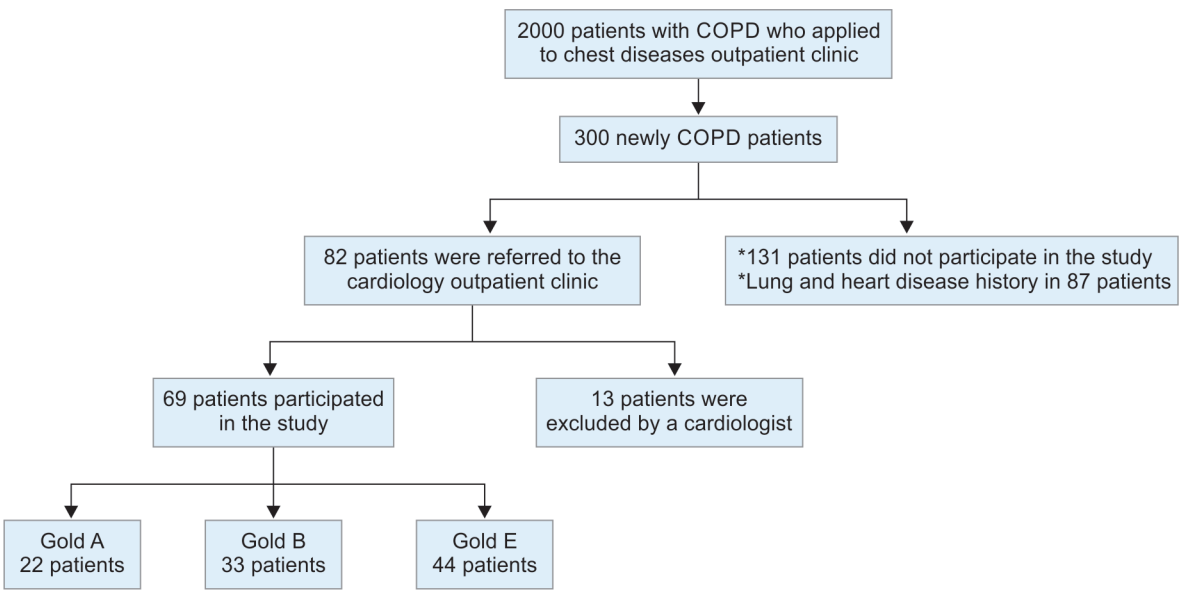


Fig. 1: Patient participation scheme

were measured. Again, the left atrial dimensions of the patients were measured. According to current clinical standards, full two-dimensional Doppler USG and tissue Doppler USG studies were performed.[19,20] Data acquisition and imaging were monitored with single-lead electrocardiography—(1) parasternal long axis, parasternal short axis, and apical four-chamber views were provided in the left lateral decubitus position; (2) subcostal view was provided in the supine position.

Statistical Analysis

Statistical analysis was evaluated with SPSS (SPSS 17.0, IBM Chicago, Illinois, United States of America). The Kolmogorov–Smirnov test determined normally distributed data. The analysis of variance (ANOVA) test (one-way ANOVA) determined the homogeneity of the data. Means with ± standard deviation are reported as continuous variables, and percentages are expressed as categorical variables. The Pearson test examined the correlations of parametric data. Variance analysis identified different groups. The paired Student's *t*-test assessed mean comparisons. Categorical variables were analyzed with the Chi-squared test. ANOVA was used to determine whether different groups were different from each other. Adjudicated outcomes were tabulated by COPD patients and the control group. A *p*-value of <0.05 and a 95% confidence interval were accepted for statistical significance.

Results

Between 1st May 2022 and 1st February 2023, SBU Şişli Hamidiye Etfal Training and Research Hospital Chest Diseases Outpatient Clinic was included. Of the approximately 2,000 COPD patients admitted to the outpatient clinic within the 8 months of the study, 69 patients with a new diagnosis of COPD and 39 healthy individuals who had smoked at least 15 packs/year of cigarettes were included as a control group. Of the COPD patients who participated in the study, 49 (71.01%) were male, and 20 (29.99%) were female. Of the 39 control group members examined, 24 (61.53%) were male, and 15 (38.47%) were female. The mean age of the 108 people participating in the study was 49.95 ± 4.67. The age of COPD patients was 48.00, and the mean age of control individuals was 45.00. We did not find a statistically significant difference between the mean age of the COPD and control group and the male and female subjects and between them according to body mass index (BMI) ($p > 0.05$). The demographic data of patients and control group are shown in Table 1.

The COPD patient group was divided into three groups based on the 2023 GOLD strategy report. There were a total of 38 (35.2%) patients in the GOLD-A group, 28 (73.7%) were male, and 10 (26.3%) were female. There were a total of 24 (22.2%) patients in the GOLD-B group, 18 (75.0%) were male, and six (25.0%) were female. There were a total of seven (35.2%) patients in the GOLD-E group; three (42.9%) were male, and four (57.1%) were female. We did not find a statistically significant difference between the mean age and BMI of the COPD patients according to the GOLD groups ($p > 0.05$) (Table 1).

The mean white blood cell (WBC) value of the patients was 8930 (7540–10270), the mean hemoglobin value was 14.85 (13.65–15.87), the mean platelet count was 273000 (223000–315000), the mean creatinine value was 0.79 mg/dL (0.62–0.90). The mean WBC value of the control group was 7920 (7410–8840), the mean hemoglobin value was 13.80 (12.60–14.90), the mean platelet count was 271000 (231000–329000), and the mean creatinine value was 0.79 mg/dL (0.66–0.86).

Of the COPD patients, 63 (91.3%) and the control group were all smokers. COPD patients had a mean smoking history of 29 packs/year, and the control group had a history of smoking 20 packs/year. The mean forced expiratory volume (FEV1%) value of the patient group was 65.00 (52.00–71.00), the mean FEV1/forced vital capacity (FVC) ratio was 71.70 (67.70–76.60), the mean FEV1% value of the control group was 94.00 (89.00–100.00), the mean FEV1/FVC ratio was 84.00 (80.90–87.10). Smoking histories and spirometry values of the patients and control group are shown in Table 2.

Two groups first performed an ECG in the cardiology outpatient clinic. All individuals participating in the study were in sinus rhythm. Atrial fibrillation and premature supraventricular-ventricular arrhythmias were not observed in either group. The right branch block was seen in 15 (13.9%) of all participants. A total of 14 of them were in the COPD group, and one of them was in the control group. Of the COPD patients with right branch block, 10 (26.3%) were in the GOLD-A group, three (12.5%) were in the GOLD-B group, and one (14.3%) was in the GOLD-E group. None of the groups had a left branch block. Echocardiographic findings of COPD patients and control group are shown in Table 3.

Patients with COPD and the control group underwent echocardiography by a blind cardiologist in the patient's clinic. Ejection fractions in both groups were normal. Diastolic dysfunction was seen in 38 (55.1%) COPD patients and 14 (35.9%) control groups. Of the patients with COPD with diastolic dysfunction, 22 (57.9%) were in the GOLD-A group, and 20 (55.3%) of them were in grade 1. Mitral valve disease was detected in 19 (27.5%) of the patients with COPD, tricuspid valve disease in 18 (26.1%), and aortic valve disease in two (2.9%) patients (Table 3).

Discussion

Comorbidities of CVD and COPD are often linked. Both diseases have similar risk factors, clinical signs and symptoms, and pathophysiological processes close to each other. Accordingly, they have a synergistic effect as negative prognostic factors. We detected cardiac pathologies in the ECG and echocardiography of

Table 1: Demographic findings of COPD patients and control group

	Gender, male (n)	*Gender, female (n)*	*Age (years)*	*BMI (kg/m²)*
All population 108 (100%)	73 (67.6%)	35 (32.4%)	46.95 ± 4.67	27.90 ± 4.80
COPD patients 69 (63.9%)	49 (71.0%)	20 (29.0%)	48.00 (43.50–51.00)	27.02 (23.26–30.04)
Control group 39 (36.1%)	24 (61.5%)	15 (38.5%)	45.00 (42.00–49.00)	28.98 (26.06–31.62)
GOLD A 38 (35.2%)	28 (73.7%)	10 (26.3%)	49.00 (43.00–51.00)	26.74 (23.79–28.90)
GOLD B 24 (22.2%)	18 (75.0%)	6 (25.0%)	47.00 (43.25–51.75)	28.24 (22.019–32.196)
GOLD E 7 (6.5%)	3 (42.9%)	4 (57.1%)	49.00 (44.00–51.00)	28.39 (24.61–31.18)
p-value of COPD patients vs controls	0.312	0.278	0.062	0.003
p-value (GOLD A through E and controls)	0.278	0.259	0.306	0.122

Table 2: Smoking histories and spirometry values of the COPD patients and control group

	GOLD A 38 (35.2)	*GOLD B 24 (22.2)*	*GOLD E 7 (6.5)*	*All population 108 (100)*	*COPD patients 69 (63.9)*	*Control group 39 (36.1)*	*p-value of COPD patients vs controls*	*p-value (GOLD A through E and controls)*
Smoking habits							0.166	0.301
Current, *n* (%)	33 (86.8)	23 (95.8)	7 (100)	102 (94.4)	63 (91.3)	39 (100)		
Former, *n* (%)	4 (10.5)	1 (4.2)	0 (0)	5 (4.6)	5 (7.2)	0 (0)		
Never, *n* (%)	1 (2.6)	0 (0)	0 (0)	1 (0.9)	1 (1.4)	0 (0)		
Pack-years	29.00 (23.50–30.50)	28.00 (26.00–36.00)	33.00 (20.00–34.00)	26.63 ± 8.23	29.00 (25.00–34.00)	20.00 (15.00–27.00)	<0.001	<0.001
Spirometry								
FEV1 (L)	2.45 (1.86–2.65)	1.87 (1.58–2.23)	2.85 (2.29–3.24)	2.39 ± 0.76	2.05 (1.59–2.49)	3,07 (2.44–3.39)	<0.001	<0.001
FEV1 (% predicted)	69.00 (62.75–74.00)	55.500 (51.00–67.00)	45.00 (34.00–47.00)	73.77 ± 18.65	65.00 (52.00–71.00)	94.00 (89.00–100.00)	<0.001	<0.001
FVC (L)	3.12 (2.49–3.63)	2.85 (2.29–3.24)	1.67 (1.27–2.72)	3.13 ± 0.86	2.95 (2.26–3.50)	3.61 (3.14–4.13)	<0.001	<0.001
FVC (% predicted)	76.50 (71.50–80.25)	70.00 (60.25–76.50)	57.00 (47.00–66.00)	79.61 ± 15.29	74.00 (63.00–79.50)	94.00 (86.00–100.00)	<0.001	<0.001
FEV1/FVC ratio	74.10 (71.20–78.67)	68.65 (65.90–73.52)	70.10 (55.10–71.10)	75.98 ± 8.97	71.70 (67.70–76.60)	84.00 (80.90–87.10)	<0.001	<0.001

patients with COPD who were newly diagnosed and not diagnosed with any pulmonary or cardiac diseases earlier.[21–23] In our research, we detected right bundle branch block in 14 patients with COPD, diastolic dysfunction in 38 patients, tricuspid valve disease in 18 patients, and mitral valve disease in 19 patients. Tricuspid annular plane systolic excursion (TAPSE) measurement of our patients with COPD was found to be lower than the control group.

While the prevalence of COPD in patients with cardiac insufficiency has been reported to vary in the United States of America by 11–52% and 9–41% in Europe, the incidence of CVD is reported less in individuals with COPD.[21,24] Sensibility and effect of CVD are accepted in individuals with COPD and have been unambiguously included in the guidelines since 2013.[25] At the same time, both assessments of cardiovascular risk and the management of CVD in patients with COPD have been poorly studied. Compared with patients without COPD, patients with COPD are more vulnerable to CVD, together with higher incidence and prevalence. Indeed, the mortality related to CVD in individuals with COPD is higher than mortality attributed to respiratory insufficiency-related causes.[23,26,27]

High P waves, right axis deviation, and right bundle branch block can be detected in the ECG of patients with COPD. There are valuable examination methods in patients with advanced COPD, and these are especially important for the detection and follow-up of patients with cor pulmonale or right-ventricle failure. Holtzman et al. compared patients with COPD to each other, 63 severe and 83 moderate-mild. Researchers found right atrial enlargement (RAE), right ventricular hypertrophy (RVH), right bundle branch block (RBBB), pronounced clockwise rotation of the heart, QS pattern in derivation III and augmented vector foot (aVF), left axis deviation (LAD), premature atrial contractions (PACs), and supraventricular tachycardias (SVTs) to be significantly more common in patients with severe COPD compared to patients with mild-moderate COPD.[28] Schneider et al. showed that the risk of developing arrhythmia in a short time in patients with COPD and without COPD was comparable.[29] In response to this, Finkelstein et al. showed that patients with COPD possessed a higher risk of myocardial infarction and arrhythmia compared to the controls without COPD.[30] Although the increasing number of proof related to the risk of CVD in patients with COPD has been found, information on these patients' ECG features is insufficient, and comparisons with patients without COPD are incomplete. In addition, most of the studies have been conducted on patient groups who were diagnosed with moderate-mild COPD earlier. Since it is conducted on patients who were newly diagnosed and were not diagnosed with cardiac disease before, our study is crucial. In our study, we observed the right bundle branch block in 14 patients with COPD. A total of 10 of the patients with right bundle branch block were in the GOLD-A group. Three of them were in GOLD-B, and one of them was in the GOLD-E group. In the control group, there was one patient with a right bundle branch block ($p < 0.05$). We did not detect any other ECG symptoms in our patients. This was more attributed to the fact that our patients were newly diagnosed and were in the GOLD-A stage.

Patients diagnosed with moderate-to-severe COPD often have systolic dysfunction of the right and left ventricles, as well as diastolic dysfunction. As a result of increasing pulmonary pressure, right ventricular dilatation and hypertrophy causing the septum to relocate in the left ventricle may endanger left ventricle filling, cardiac stroke volume (SV), and cardiac output.[11]

Echocardiography provides early, noninvasive, and accurate diagnosis of cardiac changes in patients with COPD. In moderate and advanced stages, COPD causes right-ventricle dysfunction, left-ventricle dysfunction, pulmonary hypertension (PH), and cor pulmonale by affecting pulmonary vessels, right ventricle (RV), and left ventricle. Buklioska-Ilievska et al. compared the frequency of echocardiographic changes in patients with COPD with symptoms observed in control patients and discovered that it was significantly higher in patients with COPD. The researchers also reported that there was a correlation between the severity of air circulation limitation and echocardiographic changes.[31]

Mohammed et al. echocardiographically examined the patients who were diagnosed with COPD previously and in

Table 3: Electrocardiographic findings, echocardiographic findings of COPD patients and control group

Parameters	*GOLD A n = 38 (35.2)*	*GOLD B n = 24 (22.2)*	*GOLD E n = 7 (6.5)*	*All population; N = 108 (100)*	*COPD patients n = 69 (63.9)*	*Control G n = 39 (36.1)*	*p-value of COPD patients vs controls*	*p-value (GOLD A through E and controls)*
Sinus rhythm, *n* (%)	38 (100)	24 (100)	7 (100)	108 (100)	69 (100)	39 (100)		
AF, *n* (%)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)		
PSVCs, *n* (%)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)		
PVCs, *n* (%)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)		
Bundle branch block, *n* (%)	10 (26.3)	3 (12.5)	1 (14.3)	15 (13.9)	14 (20.3)	1 (2.6)	0.011	0.028
RBBB, *n* (%)	10 (26.3)	3 (12.5)	1 (14.3)	15 (13.9)	14 (20.3)	1 (2.6)		
LBBB, *n* (%)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)		
Aortic root (cm)	2.456 (2.308–2.705)	2.436 (2.336–2.570)	2.180 (2.120–2.210)	2.441 ± 0.282	2.410 (2.210–2.600)	2.450 (2.180–2.680)	0.875	0.096
Pulmonary artery (cm)	1.830 (1.710–1.940)	1.875 (1.700–2.050)	1.870 (1.820–2.140)	1.837 ± 0.255	1.840 (1.730–1.950)	1.770 (1.560–1.890)	0.008	0.038
IVS (cm)	1.080 (0.926–1.153)	0.960 (0.900–1.180)	1.030 (0.831–1.090)	1.025 (0.920–1.120)	1.030 (0.914–1.150)	1.010 (0.920–1.070)	0.335	0.493
PW (cm)	0.990 (0.849–1.050)	0.900 (0.835–1.070)	0.970 (0.820–1.000)	0.920 (0.841–1.020)	0.970 (0.843–1.034)	0.900 (0.830–0.980)	0.132	0.380
LAD (cm)	3.150 (2.768–3.593)	3.115 (2.728–3.510)	2.850 (2.710–3.450)	3.160 (2.833–3.448)	3.100 (2.740–3.510)	3.200 (3.000–3.330)	0.508	0.692
LVEDD (cm)	4.155 (3.660–4.553)	4.290 (3.953–4.508)	4.140 (3.550–4.230)	4.230 (3.913–4.518)	4.200 (3.775–4.480)	4.310 (4.040–4.600)	0.041	0.116
LVESD (cm)	2.750 (2.323–3.053)	2.825 (2.553–3.123)	2.730 (2.070–3.050)	2.695 (2.413–2.970)	2.760 (2.440–3.055)	2.570 (2.330–2.790)	0.061	0.169
E wave velocity (m/s)	0.630 (0.545–0.793)	0.730 (0.580–0.800)	0.750 (0.540–0.850)	0.713 ± 0.181	0.650 (0.555–0.800)	0.700 (0.640–0.830)	0.140	0.245
A wave velocity (m/s)	0.685 (0.578–0.803)	0.640 (0.580–0.748)	0.710 (0.640–0.900)	0.678 ± 0.165	0.670 (0.585–0.785)	0.620 (0.520–0.800)	0.147	0.301
E/A ratio	0.843 (0.750–1.333)	1.209 (0.784–1.354)	0.833 (0.761–1.328)	1.190 (0.774–1.384)	0.916 (0.763–1.333)	1.315 (0.793–1.444)	0.026	0.102
RAD (cm)	3.180 (3.018–3.493)	3.195 (2.985–3.368)	3.150 (2.920–3.360)	3.215 (3.043–3.458)	3.160 (3.005–3.445)	3.300 (3.190–3.460)	0.034	0.116
RVD (cm)	3.025 (2.855–3.220)	3.005 (2.763–3.090)	2.890 (2.390–3.120)	3.000 (2.810–3.120)	3.010 (2.775–3.165)	2.940 (2.830–3.110)	0.706	0.544
Ejection fraction (%)	62.900 (61.325–64.000)	64.800 (62.025–67.875)	61.100 (61.000–62.200)	63.769 ± 2.722	63.000 (61.100–65.900)	64.400 (62.000–65.800)	0.139	0.013
Diastolic dysfunction, *n* (%)	22 (57.9)	11 (45.8)	5 (71.4)	52 (48.1)	38 (55.1)	14 (35.9)	0.055	0.147
Grade I, *n* (%)	21 (55.3)	9 (37.5)	5 (71.4)	48 (44.4)	35 (50.7)	13 (33.3)	0.081	0.099
Grade II, *n* (%)	2 (5.3)	2 (8.3)	0 (0)	5 (4.6)	4 (5.8)	1 (2.6)	0.442	0.683
Grade III, *n* (%)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)		
TAPSE (cm)	2.240 (2.115–2.540)	2.250 (2.100–2.605)	2.300 (2.100–2.460)	2.407 ± 0.321	2.260 (2.100–2.535)	2.480 (2.290–2.700)	0.002	0.026
Aortic valve disease, *n* (%)	1 (2.6)	0 (0)	1 (14.3)	3 (2.8)	2 (2.9)	1 (2.6)	0.919	0.248
Mitral valve disease, *n* (%)	12 (31.6)	5 (20.8)	2 (28.6)	22 (20.4)	19 (27.5)	3 (7.7)	0.014	0.069
Tricuspid valve disease, *n* (%)	9 (23.7)	6 (25.0)	3 (42.9)	26 (24.1)	18 (26.1)	8 (20.5)	0.515	0.651
Pulmonary valve disease, *n* (%)	0 (0)	0 (0)	0 (0)	1 (0.9)	0 (0)	1 (2.6)	0.181	0.618

AF, atrial fibrillation; PSVC, premature supraventricular complexes, PVC, premature ventricular contraction; IVS, interventricular septum; PW, pulse wave; LVEDD, diameter size/ left ventricular internal end-diastole; LVESD, diameter size/ left ventricular internal end-systole; RAD, right atrial diameter; RVD, right ventricular diameter

stable periods with follow-up and discovered that findings were correlated with the severity of the disease. They discovered that left ventricular diastolic dysfunction (LVDD) was often observed and a linear relationship between PH and the severity of COPD. At the same time, they wrote that the size of the left ventricle expanded by 35% in patients with mild-moderate COPD, while the existence of mild left ventricular systolic dysfunction (LVSD) was observed in 28% of the patients. The frequency of LVSD still remains unknown.[32] Portillo et al. reported in their studies the prevalence to be between 0 and 25%.[33] In our study, compared to the LVSD control group, prevalence increased minimally. Even though this statistic is not significant, it is important for newly diagnosed patients. In our patients, compared to the control group, ejection fraction was a little bit lower. This was not statistically significant. Diastolic dysfunction was detected to be low in 38 patients with COPD and 14 control members. A total of 35 patients in the diastolic dysfunction group were at the grade 1 stage. A total of 21 patients were in the GOLD-A group, nine patients were in the GOLD-B group, and five patients were in the GOLD-E group. Aortic insufficiency in two patients and mitral valve insufficiency in 19 of our patients existed. A total of 12 patients with mitral valve insufficiency were in the GOLD-A group, five were in the GOLD-B group, and two were in the GOLD-E group. Similar results were recorded in Gupta's study as well.[34]

The prevalence of LVDD in patients with COPD was reported in many studies at different rates. While Mohammed et al. reported it to be approximately 25%, Huang et al. reported it to be a higher rate (65%).[32,35] Caram et al. wrote LVDD at a rate of 88%.[36] In a study that included patients with severe COPD, the LVDD rate was indicated to be 90%. In our study, LVDD was detected only in a few patients. This was not statistically significant. It can also be attributed to the fact that most of the patients are not at the early stage, not at old age, and have low comorbidities. E wave velocity and A wave velocity values of our patients were not different from the control group. However, it was detected that the E/A rate deteriorated in our patients. This finding was seen in Gupta et al.'s study as well.[34]

In the conducted studies, it has been reported that RV size and the severity of disease are correlated.[37] Related to that, it has been indicated that between mild and severe cases, tricuspid regurgitation (TR) was observed up to the rate of 75%.[38] In our study, tricuspid regurgitation was detected in 18 patients. Nine patients were in the GOLD-A group, six were in the GOLD-B group, and three were in the GOLD-E group. One of our patients in the GOLD-E group had pulmonary valve insufficiency. TAPSE in our patients with COPD was lower than in the control group (2.260–2.480). This was coherent with Hilde et al.'s results.[39]

Conclusion

We identified cardiac pathologies that we think are important in our newly diagnosed COPD patients. COPD and CVD are expected to coexist because they have common etiologic and prognostic factors. However, almost all studies were conducted in patients who had previously been diagnosed and treated. We have seen that a significant number of our patients have started their cardiological pathologies during the initial diagnosis. Since the prognosis of COPD is greatly influenced by the presence of CVD, it is very important to define cardiac comorbidity, if any, in all patients diagnosed. With this strategy, cardiac pathologies can be intervened early, and better results can be obtained in the management of COPD.

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