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Research Article

# Analysis of Electrocardiogram Changes in in COPD Patients and Controls in the Shahrekord City: A Cohort Study Population in 2020

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## Abstract

*Background:* Chronic Obstructive Pulmonary Disease (COPD) is one of the most prevalent and rapidly growing health problems worldwide. It is a progressive disease characterized by limited or obstructed airflow that is irreversible. "COPD is primarily caused by an excessive inflammatory response to inhaled pollutants and irritants, and is often characterized by a chronic cough, with or without mucus production. Chronic bronchitis and emphysema are the two conditions that comprise COPD, and they are not always precisely distinguishable. Due to the high prevalence of COPD among the elderly population, extensive studies have reported various electrocardiogram (ECG) changes in these patients. Examples include alterations in the axis and height of the P wave and a decrease in the height of the QRS complex in limb and precordial leads. Although not all COPD patients undergo routine ECG evaluations, the occurrence of various arrhythmias remains one of the significant and preventable causes of death in this population. Multiple factors, such as impaired autonomic control of cardiac and pulmonary function, may contribute to the development of arrhythmias in COPD patients. *Method:* A total of 86 individuals diagnosed with COPD were selected through census sampling and considered the case group. For the control group, twice the number of participants (individuals without any obstructive pulmonary disease) were selected from the cohort population. Information on COPD patients were confirmed via spirometry, and their ECGs were analyzed for arrhythmias and abnormal changes.". These included alterations in wave axes, lengthening or shortening of waves, changes in the R wave, and other deviations from the normal ECG pattern. The collected data were then analyzed and compared using statistical tests. *Findings:* This study included 85 COPD patients and 168 healthy individuals as the control group, comprising a total of 253 participants. The mean age of the study population was 50.8 years, with 54% of the participants being female and 45% of the COPD patients being male. Significant ECG changes observed in COPD patients compared to the control group included weak progression of the R wave, atrial abnormalities (right atrial enlargement [RAE] and left atrial enlargement [LAE]), right ventricular hypertrophy (RVH), decreased QRS duration, and a short QT interval (*P-value* < *0.05*). *Conclusion:* The observed ECG changes, such as weak R wave progression and atrial abnormalities, emphasize the importance of ECG as a non-invasive diagnostic tool for identifying cardiovascular complications in COPD patients. This highlights the need for regular cardiac monitoring in these individuals. Since certain cardiac complications in COPD patients are preventable or manageable, regular examinations such as ECG and echocardiography are recommended.

## Keywords

Chronic Obstructive Pulmonary Disease (COPD), Electrocardiographic Changes, Electrocardiogram (ECG)

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# 1. Introduction

## 1.1. COPD

Chronic obstructive pulmonary disease (COPD) is one of the most significant and rapidly growing global health concerns. This progressive and irreversible disease is characterized by limited and obstructed airflow, which results from an excessive inflammatory response of the lungs to inhaled smoke and pollutants. COPD encompasses two primary conditions—chronic bronchitis and emphysema—which, while related, are not always easily distinguishable. The hallmark of COPD is a chronic cough, with or without sputum production, persisting for at least two consecutive years and occurring for more than three months per year [1].

COPD is a heterogeneous clinical syndrome, affecting approximately 6–8% of the global population. The respiratory and circulatory systems are closely interconnected, and structural or functional changes in the respiratory system can have a profound impact on cardiovascular performance. In COPD patients, these changes often lead to cardiac arrhythmias, with sudden cardiac death being one of the leading causes of mortality in this population [2].

Increased pulmonary pressure in COPD arises from several factors, including pulmonary vessel constriction due to hypoxemia and hypercarbia. A direct correlation exists between $PaCO_2$ levels and pulmonary artery pressure in these patients. Chronic hypoxemia causes vascular lumen thickening, which is directly related to the severity of pulmonary artery pressure. Additionally, peripheral vascular inflammation in COPD can lead to pulmonary artery thrombosis. Although pulmonary artery pressure increases gradually in COPD patients, it serves as an important indicator of prognosis. In a study by Weitzenblum et al., survival rates were reported to be 72% at four years in patients with normal pulmonary artery pressure but dropped to 49% in those with pulmonary arterial hypertension (PAH), defined as a mean pulmonary artery pressure exceeding 20 mmHg [3].

Another cohort study of 270 COPD patients, with an average survival of 3.1 years, found that mortality was significantly associated with variables such as age, ECG changes indicating ischemia and right ventricular hypertrophy (RVH), chronic kidney disease (CKD), and a forced expiratory volume in one second ($FEV_1$) of less than 590 milliliters [4].

## 1.2. The Association Between COPD and Cardiovascular Diseases

Epidemiological studies have demonstrated that COPD shares common risk factors with cardiovascular diseases, including coronary artery disease, congestive heart failure, and cardiac arrhythmias. The potential pathogenesis involves complex interrelationships among chronic systemic inflammation, oxidative stress, and shared risk factors such as age, smoking, and exposure to environmental pollutants [5, 6].

## 1.3. Comorbidities Associated with COPD

Epidemiological studies have demonstrated that chronic obstructive pulmonary disease (COPD) shares common risk factors with cardiovascular diseases, such as coronary artery disease, congestive heart failure, and cardiac arrhythmias. The potential pathogenesis involves complex interrelationships among chronic systemic inflammation, oxidative stress, and shared risk factors including age, smoking, and exposure to environmental pollutants [5, 6]. Numerous studies on comorbidities associated with COPD have identified diabetes mellitus, hypertension, cardiovascular disease, chronic kidney disease, and ischemic heart disease as the most prevalent and significant conditions contributing to increased mortality in these patients. In a study by R. Antonelli and colleagues, chronic kidney disease and ischemic heart disease were found to be significantly associated with the prognosis of patients following COPD exacerbations. Patients with both COPD and cardiovascular disease (CVD) experience elevated rates of complications, including reduced quality of life, dyspnea, and an increased risk of hospitalization for both conditions. Additionally, the presence of cardiovascular diseases such as heart failure, ischemic heart disease, diabetes, or atrial fibrillation increases the likelihood of COPD exacerbations and mortality. Exacerbation of COPD and a decline in lung function are also linked to heightened cardiovascular risk and mortality. To mitigate these risks, comprehensive management of patients with COPD and CVD is essential. However, it is important to note that pharmacological treatments for COPD may have either beneficial or adverse effects on CVD, and vice versa, necessitating a careful and integrated approach to treatment [7].

The Necessity of Conducting the Study is describing as follow:

Chronic obstructive pulmonary disease (COPD) is a heterogeneous clinical syndrome that affects about 6-8% of the population. In humans, the respiratory and circulatory systems are intricately related from the outset and can have long-term effects on each other. In patients with COPD, functional and structural changes in the respiratory system can significantly affect the performance of the cardiovascular system, leading to cardiac arrhythmias and sudden death as causes of mortality in these individuals. In COPD, right ventricular hypertrophy (RVH), changes in the position of the heart within the chest, and pulmonary hyperinflation can lead to changes in the ECG, including right axis deviation, reduced QRS wave amplitude, and delayed transition in the precordial leads. There is a direct relationship between these ECG changes and respiratory function, and RVH on the ECG is a predictor of pulmonary hypertension.

Therefore, in these patients, important information about respiratory function and pulmonary hypertension—one of the key causes of mortality—can be obtained through ECG Text Analysis.

In a study conducted by Christos A. and colleagues (2017) on the relationship between chronic obstructive pulmonary disease (COPD) and atrial fibrillation (AF), it was found that there is a strong association between COPD and AF. The occurrence of AF in patients with COPD significantly impacts morbidity and mortality. The authors also emphasized that treating underlying pulmonary diseases, as well as correcting hypoxia and acid-base imbalances, are essential first steps in managing COPD patients who develop AF. Furthermore, AF ablation was identified as an intervention that could help improve the quality of life in these individuals.

Although beta-blockers are generally contraindicated in COPD patients, the study suggests that their use may be appropriate during acute COPD exacerbations in patients with atrial fibrillation (AF), though the authors did not provide further explanation [8].

In a study conducted by Christos A. Goudis et al. (2015) on changes in electrocardiograms (ECG) and arrhythmias in patients with COPD, it was found that COPD is associated with atrial premature complexes (PAC), premature ventricular complexes (PVC), atrial fibrillation (AF), multifocal atrial tachycardia (MAT), and ventricular tachycardia (VT), all of which can increase patient mortality. The authors emphasized the important role of beta-blockers and anti-arrhythmic drugs in managing these patients [9].

In a study by Noriane A. Sievi and colleagues (2014) on the prevalence of repolarization changes in patients with COPD, it was found that 31% of the 91 COPD patients had a prolonged QT interval, and QT dispersion was observed in 24% of them. Overall, they stated that one-third of COPD patients had repolarization changes, which they attributed to hypoxia in these patients [10].

In a cross-sectional study of 243 COPD patients, Mirijam J. Warnier and colleagues (2013) reported that these patients had a higher incidence of conduction disorders compared to the normal population. The average heart rate in these patients was elevated, and these disorders were exacerbated during exacerbations of the disease [11].

Niranjan Mambally Rachaiah and colleagues (2012) reported that among 50 confirmed cases of COPD with spirometry and FEV1 less than 80%, 66% had changes in the P wave axis, 41% had changes in the QRS axis, and 42% had an increase in P wave height greater than 2.5 mm, while 4% had RBBB. Thirty-two percent had no specific findings. A positive point of this study was the exclusion of patients with a history of diabetes, asthma, congenital heart disease, and hypertension [12]. In a study by Lowie E.G.W. Vanfleteren and colleagues (2011) on the relationship and prevalence of ischemic changes in the ECG of COPD patients, they reported that 21% of patients had ECG changes suggestive of ischemia. According to their report, these patients did not have a good prognosis [13].

Pal H. Brekke and colleagues (2008) conducted a study on 897 COPD patients with a mean age of 70 years, titled "Diagnosis of Myocardial Infarction Based on Cardiac Infarction Injury Score (CIIS)" in COPD patients discharged from the hospital after treatment and later called for an ECG. They reported that 229 patients had a CIIS score greater than 20, and only 30% of patients had a previous history of myocardial infarction. The study concluded that undiagnosed myocardial infarction (MI) is common among COPD patients during exacerbations. One limitation of the article, however, was the lack of consideration of the patients' history of other diseases [14].

In a study conducted by Pinar Yildiz MD et al. (2002) on 30 patients, it was reported that ventricular premature beats occur at a higher rate in COPD patients compared to the control population. Additionally, 27% of the COPD patients studied had ventricular tachycardia. Overall, it was stated that in COPD patients, due to autonomic changes, ventricular arrhythmias are observed at a higher rate than in the control population. [15].

Nir Seider et al. (1993) discussed the potential effects of bronchodilators on cardiac arrhythmias in COPD patients, stating that this class of drugs improves FEV1 and heart rate, and premature beats were not significantly observed. No clear relationship between bronchodilators and arrhythmias was found [16].

In a study by Raffaele Antonelli Incalzi et al. (1990) on 22 patients titled "Cardiac Arrhythmias and Left Ventricular Function in Respiratory Failure Due to COPD," the use of radionuclide angiography revealed that a decrease in left ventricular diastolic function is a likely factor contributing to arrhythmias during respiratory failure in COPD patients. The study found that with improved left ventricular function, the observed arrhythmias decreased significantly [17].

1) Entry Criteria

Patients diagnosed with chronic obstructive pulmonary disease (COPD) based on clinical symptoms such as chronic cough and sputum production, and who have undergone spirometry showing a forced expiratory volume in 1 second (FEV1) of less than 70%.

2) Exit: Criteria

Patients with a history of cardiovascular conditions, such as heart attack or hypertension, that have caused ECG changes indicative of the underlying disease, are excluded from the study.

3) Sample Size and Sampling Method

All patients presenting with cough and sputum symptoms, confirmed to have COPD with an FEV1 of less than 70% by spirometry, were selected as the case group using a census method, totaling 85 individuals. A control group was formed by randomly selecting twice as many individuals.

4) Location and Time of Study

This study was conducted at the cohort center in

Shahrekord City in 2020.

## 2. Methodology

This study utilized a population-based case-control design to compare two groups of individuals from the Persian cohort in Shahrekord. The case group consisted of individuals diagnosed with COPD, confirmed by clinical symptoms and spirometry. The control group was selected to match the case group in size, with twice as many individuals randomly chosen. The electrocardiograms (ECGs) of all participants were recorded using a standardized and calibrated ECG machine available at the cohort center. The recorded ECGs were evaluated by a cardiac specialist to identify and analyze any changes.

## 3. Results

In this study, 85 COPD patients and 168 healthy individuals were enrolled as the control group. Of the COPD patients, 54% were female, and 45% were male. The mean age of the patients was 50.85 years, while the mean age of the control group was 50.77 years. The standard deviation for the two groups was 9.57 and 9.52, respectively, reflecting the matching of the control group with the case group. The mean and standard deviation of the study variables are presented in the table below (Table 1).

***Table 1.*** *Determination of the Mean and Standard Deviation of Quantitative Variables and Electrocardiographic Changes in the Entire Study Population.*

| Variable | Count | Maximum | Minimum | Mean | Standard Deviation |
|---|---|---|---|---|---|
| Age | 253 | 70 | 35 | 50.80 | 9.53 |
| FEV1/FVC (FEV1/FVC) | 253 | 100 | 35 | 85.01 | 16.204 |
| FEV1 (FEV1) | 253 | 4.81 | 0.52 | 1.21 | 1.26 |
| Rate (Rate) | 253 | 107 | 45 | 67.99 | 10.65 |
| QRS Duration (ms) | 253 | 120 | 40 | 74.77 | 14.8 |
| PR Interval (ms) | 253 | 240 | 100 | 59.31 | 25.29 |

Due to the matching of data between the case and control groups, no significant differences were found in age and gender variables. However, the FEV1/FVC ratio differs between the two groups, which is expected due to its crucial role in diagnosing the disease (Table 2).

***Table 2.*** *Analysis of the Mean and Percentage of the Variables Under Study.*

| Variable | Total Individuals | Case Group | Control Group |
|---|---|---|---|
| Number of Individuals | 253 | 85 | 168 |
| Age | 50.8 | 50.85 | 50.77 |
| Gender | | | |
| Male | 136 (53.54%) | 46 (54.11%) | 90 (53.57%) |
| Female | 117 (46.46%) | 39 (45.88%) | 78 (46.43%) |
| FEV1/FVC | 85.17 | 64.12 | 95.59 |

The average heart rate of COPD patients was 68.34 bpm, while in the control group, it was 67.8 bpm. No significant difference was found in heart rate between the two groups, with averages of 68.68 bpm in COPD patients and 67.62 bpm in healthy individuals. However, 16 COPD patients (18%) exhibited bradycardia, a significant finding with a P-value < 0.05. Notably, no cases of tachycardia were observed among the patients. The presence of bradycardia in COPD patients may indicate abnormalities in the autonomic nervous system, potentially linked to valgus nerve stimulation.

Disrupted R wave progression was observed in 47% of COPD patients (41 individuals), making it the most significant ECG change in this group. Weak R wave progression is commonly associated with left ventricular hypertrophy (LVH) and both acute and chronic right ventricular overload. Additionally, the downward displacement of the heart within the chest wall, often seen in emphysema, can further contribute to weak R wave progression. LVH is recognized as a major risk factor for mortality in COPD patients. In this study, 4 patients exhibited LVH, with significant changes identified (P-value = 0.05).

Left ventricular hypertrophy (LVH) with preserved ejec-

tion fraction (EF) within the normal range is frequently observed in COPD patients, particularly in women, although its exact incidence remains unclear. Further investigations, including echocardiography and evaluations for heart failure or cardiac remodeling, are essential for timely interventions that could reduce mortality and complications associated with the disease.

Following the observation of weak R wave progression, a shortened QRS wave duration was noted in 36% of COPD patients. In this study, 32 COPD patients exhibited a short QRS duration compared to 46 individuals in the control group. This finding was statistically significant, with a P-value of 0.05.

Among the 86 COPD patients, only one exhibited a conduction abnormality in the form of left bundle branch block (LBBB). Several factors may contribute to ECG changes in COPD patients, including dilation and hypertrophy of the right atrium and ventricle, as well as impaired electrical conduction caused by pulmonary emphysema. Significant structural changes in the right heart were also observed in this study. Specifically, 12 patients (6.13%) demonstrated right atrial enlargement, and 13 patients (15%) exhibited right ventricular hypertrophy (RVH). Both findings were statistically significant, with a P-value < 0.05, and were more commonly observed in patients with a lower FEV1/FVC ratio.

Another notable finding was the shortened PR interval. Eighteen patients (20%) exhibited this condition, while only 13% of healthy individuals (23 people) had a similar finding, which was not statistically significant (P-value > 0.05). Notably, no cases of heart block were detected among the patients in this study.

Nine patients showed evidence of axis deviation on the electrocardiogram, which, compared to the 12 cases in the control group, was not statistically significant.

Changes in the QT interval among patients were significant for both shortening and prolongation. A total of 14 patients (16%) had a short QT interval, which was considered statistically significant with a P-value < 0.05. Additionally, 2 patients exhibited a long QT interval.

Six patients exhibited tall T waves, which was a statistically significant change with a P-value < 0.05.

Among the patients, 60 individuals had a normal PR interval, while 3 exhibited a prolonged PR interval.

A pathological Q wave can result from infarction of the free wall or lateral wall of the left ventricle, typically observed in the mid-precordial to lateral precordial leads. In this study, the presence of a path in Table 3, the electrocardiographic findings for both the study and control groups are presented, categorized by variable.

In this study, a history of myocardial infarction and uncontrolled hypertension, if they could cause electrocardiographic changes related to the underlying condition, were considered confounding variables. Accordingly, the corresponding electrocardiograms were excluded from the analysis. The study found no significant electrocardiographic changes related to hypertension in the affected patients. Additionally, in patients with a history of myocardial infarction, pathological Q waves were excluded from the count of changes.

Pathological Q wave was considered a significant change with a P-value < 0.05.

***Table 3***. *Findings of ECG Analysis by Control and Case Group.*

| Variable | Number of Cases in Healthy Individuals | Number of Cases in COPD Patients | P-Value |
|---|---|---|---|
| Arrhythmia | 0 | 4 | 0.005 |
| Bradycardia | 18 | 16 | 0.03 |
| Tachycardia | 1 | 0 | 0.476 |
| Axis/LAD or RAD | 12 | 9 | 0.348 |
| Right Atrial (RA) Abnormality | 0 | 12 | 0.001 |
| Left Atrial (LA) Abnormality | 0 | 16 | 0.001 |
| Short PR interval | 23 | 18 | 0.127 |
| Long PR interval | 0 | 3 | 0.014 |
| Normal PR Interval | 144 | 60 | 0.03 |
| Short QRS Duration | 46 | 32 | 0.001 |
| Long QRS Duration | 0 | 5 | 0.001 |
| Long Qt | 5 | 2 | 0.029 |
| Short Qt | 15 | 14 | 0.028 |

| Variable | Number of Cases in Healthy Individuals | Number of Cases in COPD Patients | P-Value |
|---|---|---|---|
| ST Elevation | 29 | 6 | 0.026 |
| St Depression | 5 | 8 | 0.029 |
| T Inversion | 20 | 5 | 0.186 |
| Tall T Waves | 2 | 6 | 0.012 |
| Right Ventricular Hypertrophy (RVH) | 10 | 13 | 0.009 |
| Left Ventricular Hypertrophy (LVH) | 1 | 4 | 0.001 |
| Right Bundle Branch Block (RBBB) | 0 | 0 | 0 |
| Left Bundle Branch Block (LBBB) | 0 | 1 | 0.0001 |
| Low voltage | 1 | 0 | 0.3 |
| Premature Atrial Contractions (PAC) | 0 | 0 | 0 |
| Poor R progression | 39 | 41 | 0.0025 |
| pvc | 0 | 0 | 0 |
| Hypertension History (HTN) | 23 | 19 | 0.057 |
| Ischemic Heart Disease (IHD) History | 31 | 28 | 0.0096 |
| Pathologic Q Waves | 4 | 6 | 0.075 |

In addition, the standard deviation of the quantitative variables under study is presented in Table 4. Due to the standardization of the age variable in the control group, no significant difference in the standard deviation of this variable is observed between the two groups. However, differences are noted in the standard deviations of other variables, such as spirometry results, QRS duration, and heart rate.

***Table 4.*** *Examination of the standard deviation, maximum, and minimum values in the studied quantitative variables.*

| Standard Deviation | | Groups | | Individuals | Variable |
|---|---|---|---|---|---|
| Control Group | Case Group | Control Group | Case Group | | |
| 9.52 | 57.9 | | | 253 | Age |
| 85.4 | 69.8 | Max=100<br>Min=91.75 | Max=12.42<br>Min=70 | | FEV1/FVC |
| 10.26 | 11.44 | Max=107<br>Min=45 | Max=100<br>Min=46 | 253 | Heart Rate |
| 0.291 | 1.096 | Max=4.81<br>Min=0.0052 | Max=75.3<br>Min=212.0 | 253 | FEV1 |
| 10.76 | 20.99 | Max=100<br>Min=40 | Max=120<br>Min=40 | 253 | QRS Duration |
| 21.31 | 31.66 | Max=200<br>Min=100 | Max=240<br>Min=120 | 248 | PR Interval |

## 4. Discussion

COPD is the fourth leading cause of death among diseases. The most significant morbidity associated with COPD is its effect on the cardiovascular system, resulting from increased pulmonary artery pressure. This condition leads to right ventricular enlargement and a reduction in voltage transmission between the heart and the electrocardiogram (ECG) leads. Additionally, COPD can cause specific changes in the ECG due to pulmonary vessel constriction in the context of hypoxia. Cardiovascular diseases are common among COPD patients, but they are often undiagnosed in this group. Ischemic changes on the ECG are associated with a high risk of mortality due to coronary artery disease, yet they are not routinely assessed in these patients. Furthermore, multiple studies have shown that patients with COPD are 2 to 3 times more likely to die from cardiovascular diseases, which account for 50% of their mortality rate [18].

In the conducted study, significant changes were observed in the electrocardiograms (ECGs) of COPD patients, with one of the most common changes being poor R wave progression. Various studies have examined the causes of this occurrence. For example, in a study by JP. Singh and colleagues on magnesium levels in COPD patients, it was reported that patients with decreased magnesium levels were more likely to experience episodes of attacks, and among these patients, 48% had poor R wave progression [19]. The presence of emphysema and emphysematous bullae in COPD patients may also contribute to the development of this trend. Regarding the differentiation between poor R wave progression in patients with emphysema and old anteroseptal myocardial infarction, a ratio ≥ 3.5 in lead V5 (RS) can be used as a distinguishing factor.

Another notable and common change is related to the right atrium, which was observed in 12 COPD patients. Additionally, in a study conducted by Asma Shabbir and colleagues on ECG changes in COPD patients based on disease severity, it was reported that right atrial enlargement (RAE) was the most common ECG change, observed in 65 out of 343 patients. Similar to this study, the highest incidence was found in patients with severe COPD, and it was suggested that the presence of RAE could be an indicator of disease severity [20]. Furthermore, in a study by Grymonprez M and colleagues, it was stated that patients experiencing exacerbations and attacks of COPD are at risk for atrial fibrillation (AF), and these individuals also have a higher rate of left atrial enlargement (LAE) [21]. An increase in P wave dispersion (PWD) in COPD patients could be a sign of AF development in these patients. Furthermore, an increase in PWD in COPD patients without AF suggests that COPD itself contributes to the increase in PWD. Therefore, considering this factor alongside a long PR interval, which was also observed in 3 patients in this study, is noteworthy.

Additionally, based on the study conducted at this center and the rate of left atrial enlargement (LAE) observed in patients (9 COPD patients), performing echocardiography and determining the patient's need for aspirin prophylaxis to prevent thromboembolic consequences in the event of atrial fibrillation (AF) could be helpful.

After calculating the corrected QT (QTc) interval among the patients, changes indicating both short and long QT intervals were observed, with both being statistically significant. Prolonged QT in patients can occur due to various causes, such as coronary artery disease (CAD), cardiomyopathies, and electrophysiological abnormalities. Hypoxia can also be a factor contributing to QT prolongation. Studies have shown that long QT can be associated with ventricular arrhythmias. Additionally, electrolyte imbalances in patients with short QT should be carefully considered, given the potential underlying causes. Prolonged QTc can serve as a predictor of cardiac events and may also indicate autonomic neuropathy among patients. In the cohort study by Carlos Iribarren and colleagues on short QTc among 7.1 million data points, it was stated that COPD is one of the key causes of QTc < 300, a finding that is paradoxical given the beta-agonist medications commonly used by these patients [22].

Therefore, further examination of COPD patients with these characteristics, including laboratory tests and echocardiography, is highly recommended.

Various studies have discussed changes in the QRS complex in COPD patients. In the study by M.D. Sergio Sanchez Zambrano and colleagues, narrowing of the QRS complex was more commonly observed. According to the analysis in this study, 37.2% of COPD patients experienced narrowing of the QRS complex, compared to 27% in the control group [23]. Changes in the QRS complex, specifically a decrease in the duration of this wave, were also observed with a statistically significant P-value in our study.

Hyperinflation, which occurs due to obstruction or emphysema, can lead to clockwise rotation. The development of p-pulmonale is a result of an increased RV/LV end-diastolic volume ratio. With right ventricular (RV) expansion, electrical forces increase. Additionally, a decrease in left ventricular (LV) end-diastolic volume (likely due to pulmonary vascular resistance, which arises from hypoxia) occurs. This increase in the RV/LV ratio is also associated with a reduction in the duration of the QRS wave, and in this study, 19 patients had a short QRS duration.

In the study by Gunnar Einvik and colleagues, which examined the prevalence of PVC and PSVT arrhythmias in two groups of COPD patients during the exacerbation phase and stable state, it was found that the prevalence of these arrhythmias was significantly higher during the exacerbation phase [22]. In the study conducted at this center, since all 86 COPD patients were stable in terms of their disease, no PVCs were observed.

Left ventricular hypertrophy (LVH) is one of the occurrences associated with an increased risk of mortality in

COPD patients, and this finding has been observed in various studies. In this study, given the significant occurrence of this finding, it highlights the importance of its development in COPD patients.

One of the causes of this phenomenon can be attributed to the inflammatory nature of COPD, which leads to the activation of the renin-angiotensin-aldosterone system (RAAS), ultimately resulting in systemic end-organ damage, with LVH being one of these damages. The increase in left ventricular mass over the long term indicates a poor prognosis for these patients. Additionally, pleural changes and increased intrathoracic pressure in COPD patients are associated with increased LV afterload and LV wall stress, which leads to hypertrophy. Since the occurrence of LVH can lead to other events such as sub-endocardial ischemia, myocardial fibrosis, LAE, atrial fibrillation, diastolic dysfunction, heart failure with preserved ejection fraction, and sudden cardiac death, further examination of COPD patients with LVH evident in their ECG is recommended. Moreover, the use of medications to prevent remodeling in these patients should be considered.

In stable COPD patients without a previous history of cardiovascular disease, it has been reported that Troponin T levels are higher than in the general population, indicating the presence and association of cardiovascular diseases with COPD. In this study, changes in the Q wave in the form of a pathological Q wave were found to be statistically significant. The causes of this can be linked to the inflammatory nature of the disease and the involvement of the cytokine system and interleukin-6. In a study by Anke M. C. Neukamm and colleagues on 101 stable COPD patients and 120 normal individuals, it was also reported that in COPD patients without underlying heart disease, elevated Troponin T levels were observed, accompanied by a Q wave on their ECG, indicating inflammatory activity [24].

Thus, based on the general findings of studies, the presence of a pathological Q wave in COPD patients suggests the need for further investigation of these patients for cardiac biomarkers to detect unrecognized myocardial infarction.

Based on the results of our study, changes in the ECG among COPD patients are quite common. Furthermore, some changes are more frequently observed in patients with more severe disease, and these changes may indicate a higher risk of cardiovascular events and complications in the future. Although the data strongly suggest that COPD patients are at increased risk for cardiovascular diseases, it appears that clinical practice primarily focuses on the respiratory issues of these patients. For example, the NICE and GOLD guidelines briefly address cardiovascular problems in COPD patients. Therefore, while echocardiography is a valuable tool for assessing pulmonary hypertension, it is particularly useful in COPD patients due to hyperinflation of the lungs and increased thoracic pressure. Various mechanisms could explain the increased risk of cardiovascular events in COPD patients, with systemic inflammation being a potential contributing factor. The risk of cardiovascular events is elevated not only in COPD patients but also in other systemic diseases, such as rheumatoid arthritis and chronic kidney disease (CKD). Epidemiological studies strongly indicate a relationship between systemic inflammation and cardiovascular events [25]. Therefore, in routine assessments of patients with severe disease, inflammatory markers should be evaluated alongside echocardiography, and pharmacological interventions should be considered when necessary.

## 5. Conclusion

Based on the results of this study and the significant changes observed in markers such as alterations in the right atrium and ventricle, right ventricular hypertrophy (RVH), changes in QRS wave duration, short QT, and atrial fibrillation (AF), it appears that cardiovascular complications can be expected in COPD patients. Therefore, periodic cardiovascular assessments during follow-up, including ECG or echocardiography, are recommended for determining cardiac status and preventing complications.

Although the data strongly indicate that COPD patients are at increased risk for cardiovascular diseases, clinical practice seems to focus primarily on the respiratory issues of these patients. For example, the NICE and GOLD guidelines briefly address cardiovascular problems in COPD patients. While echocardiography is a valuable method for assessing pulmonary hypertension, it is particularly effective in COPD patients due to lung hyperinflation and increased thoracic pressure. Several mechanisms could explain the increased risk of cardiovascular events in COPD patients, with systemic inflammation being one potential contributing factor. Timely assessment and intervention could be beneficial for managing these risks. Importantly, these findings highlight the need for more integrated and multidisciplinary approaches in the management of COPD patients. Future research should focus on validating these ECG markers as early predictors of cardiovascular complications in COPD and exploring targeted interventions to reduce this comorbidity burden. Longitudinal studies with larger sample sizes are also essential to better understand the progression of cardiac involvement and to develop preventive strategies that can be incorporated into routine COPD care.

## Abbreviations

| | |
|---|---|
| COPD | Chronic Obstructive Pulmonary Disease |
| RVH | Right Ventricular Hypertrophy |
| PAH | Pulmonary Arterial Hypertension |
| RAE | Right Atrial Enlargement |
| LAE | Left Atrial Enlargement |
| RBBB | Right Bundle Branch Block |
| LBBB | Left Bundle Branch Block |
| LVH | Left Ventricular Hypertrophy |

| | |
|---|---|
| AF | Atrial Fibrillation |
| CVD | Cardiovascular Disease |
| PVC | Premature Ventricular Contraction |
| PAC | Premature Atrial Contraction |
| MAT | Multifocal Atrial Tachycardia |
| EF | Ejection Fraction |
| CKD | Chronic Kidney Disease |

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All executive procedures were conducted in accordance with the approval from the Ethics Committee of Shahrekord University of Medical Sciences, under ethics code IR.SKUMS.REC.1399.126.

## Conflicts of Interest

The authors declare no conflicts of interest.

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