Cureus
Part of SPRINGER NATURE

Open Access Original Article

Cardiac Risk in Chronic Obstructive Pulmonary Disease: Evaluation Using the Framingham Risk Score and Cardiac Infarction Injury Score

Review began 10/23/2026
Review ended 11/08/2026
Published 11/14/2026

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DOI: 10.7759/cureus.119665

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Abstract

Introduction: Chronic obstructive pulmonary disease (COPD) is linked to an excess cardiovascular disease (CVD) burden, but cardiovascular risk may not be captured in traditional risk assessment. The aim of this study was to assess cardiovascular risk in different grades of severity of COPD, according to the Framingham Risk Score (FRS) and Cardiac Infarction Injury Score (CIIS), and to investigate the association of these cardiovascular risk scores with pulmonary function and functional status.

Methods: Clinically stable patients with COPD aged 40-55 years were enrolled in a cross-sectional hospital-based observational study involving 30 patients of each stage of the disease (mild, moderate, and severe). Demographic parameters, vital parameters, lipid parameters, spirometric parameters, six-minute walk distance (6MWD), modified Medical Research Council (mMRC) dyspnea score, and BODE index were measured. Cardiovascular risk was evaluated using the FRS (10-year cardiovascular risk) and CIIS (electrocardiographic score of myocardial injury patterns). Echocardiographic parameters were also analyzed. Descriptive statistics and correlation analyses were used.

Results: The mean age of participants was 49.11 ± 4.40 years, and 78 (86.7%) were male. The mean forced expiratory volume in one second (FEV1), FEV1/forced vital capacity (FVC) ratio, and BODE index were 1.80 ± 0.61 L, 63.27 ± 8.77%, and 6.52 ± 1.86, respectively. The mean predicted cardiovascular risk obtained using the FRS was 19.46 ± 3.34%, with similar values across the mild, moderate, and severe COPD groups. In contrast, the mean CIIS increased progressively with disease severity (17.03 ± 3.85, 18.46 ± 5.34, and 20.17 ± 4.13, respectively).

Conclusions: COPD patients had a significant cardiovascular risk burden. CIIS values increased across the COPD severity groups, indicating that there might be a value for electrocardiographic assessment beyond conventional risk estimation. The integrated cardiopulmonary evaluation could be useful to identify patients who need to be monitored more carefully for cardiovascular problems and need cardiovascular risk factor management.

Categories: Cardiology, Integrative/Complementary Medicine, Cardiac/Thoracic/Vascular Surgery
Keywords: bode index, cardiac infarction injury score, cardiovascular disease, chronic obstructive pulmonary disease, framingham risk score, spirometry

Introduction

Chronic obstructive pulmonary disease (COPD) is a chronic and progressive lung disease that causes airway and/or alveolar abnormalities that lead to persistent airflow limitation and chronic respiratory symptoms [1]. COPD is generally considered a lung disease, but it has also been recognized as a multisystem disease, characterized by systemic inflammation, decreased physical activity, skeletal muscle dysfunction, nutritional abnormalities, and a significant burden of cardiovascular disease (CVD) [2]. It is one of the major causes of morbidity and mortality in the world and is increasingly becoming a public-health problem, especially in countries where tobacco smoke, biomass fuel, and occupational air pollution are increasing [3].

CVD is a significant comorbidity of COPD. People with COPD are more likely to have coronary artery disease, heart failure, cardiac arrhythmias, pulmonary hypertension, and cerebrovascular disease than people who don't have COPD [4]. Such complications can occur even in individuals who have mild or moderate airflow limitation and may go undiagnosed, as symptoms such as exertional dyspnea, fatigue, reduced exercise tolerance and chest discomfort are easily explained by the respiratory disease they have [5]. CV involvement plays a significant role in COPD hospitalization and mortality, and early identification of cardiovascular risk is an important part of the COPD management plan [6].

There are several interacting mechanisms which help explain the relationship between COPD and CVD.

Advancing age, sedentary lifestyle, obesity or undernutrition, cigarette smoking, and socioeconomic disadvantage are all risk factors for both conditions [7]. COPD-related pathophysiology can also have an independent effect on causing cardiovascular damage: chronic systemic inflammation can lead to endothelial dysfunction, oxidative stress, platelet activation, and coagulation disturbances; chronic hypoxemia causes an increase in the work of the heart and is associated with arrhythmias and ischemia; pulmonary hypertension leads to an increase in venous return, changes in the loading of the ventricles, and decreased cardiac output [8]. In addition to the traditional cardiovascular risk, there is physical inactivity which is exacerbated by dyspnea and exercise intolerance [9]. Bidirectional, and in acute exacerbations, systemic inflammation, hypoxia, increased heart rate, and increased intrathoracic pressure may increase the risk of myocardial infarction, heart failure, arrhythmias, and stroke. There is a strong focus on COPD guidance, which highlights the importance of actively considering and assessing CVD in patients with COPD, as many people with COPD do not receive a diagnosis or treatment for it [10].

Spirometry, specifically forced expiratory volume in one second (FEV1), has been the traditional method of determining COPD severity. While FEV1 is an important measurement of airflow obstruction, patients with similar levels of airflow obstruction can have very different levels of symptoms, exercise tolerance, nutritional status, exacerbation risk, and long-term prognosis. The BODE index combines body mass index (BMI), airflow obstruction, dyspnea, and exercise capacity to give a more comprehensive measurement of disease burden and has been shown to have prognostic value in addition to spirometry [11]. The modified Medical Research Council (mMRC) dyspnea scale and the six-minute walk test (6MWT) contribute clinically relevant data about functional limitations and may help to identify patients with a greater clinical risk than indicated by lung function alone [12].

Research studies in COPD and CV morbidity have concentrated on the COPD patient with existing CVD, hospitalized patients, or advanced COPD. Fewer have studied clinically stable patients at different stages of disease severity with an integrated assessment of pulmonary function, functional capacity, multidimensional disease burden, conventional cardiovascular risk, electrocardiographic abnormalities, and cardiac function [13]. In the present study, the clinical stability of patients with mild, moderate, and severe COPD was assessed, and spirometry, BMI, mMRC grade, six-minute walk distance (6MWD), and BODE index, along with Framingham Risk Score (FRS), Cardiac Infarction Injury Score (CIIS), echocardiography, and lipid profile, were evaluated. We compared these measures between severity groups and examined the relationships between these measures as a way to understand whether there was a measurable cardiovascular risk, with subclinical cardiac abnormalities, as a result of an increased burden of respiratory disease within these severity groups.

Materials And Methods

Study design, setting, and ethics

This was a cross-sectional, hospital-based observational study involving clinically stable patients with previously diagnosed COPD, conducted from January 2025 to June 2026. The study involved an initial retrospective review of existing medical records from January 2025 to September 2025, followed by prospective recruitment and study-specific clinical assessments after obtaining Institutional Ethics Committee approval on September 17, 2025. Prospective recruitment and data collection continued until June 2026. The study adhered to the principles of the Declaration of Helsinki. The retrospective and prospective components were conducted in accordance with the applicable institutional ethics approval and consent requirements. Written informed consent was obtained from all prospectively recruited participants before enrolment and study-related assessments. Participation was voluntary, and patients were informed of their right to withdraw without affecting their medical care.

Study population and eligibility

A total of 112 patients with a previously established diagnosis of COPD were screened for eligibility during the study period. Of these, 90 patients fulfilled the inclusion criteria and were enrolled in the study, while 22 were excluded. The reasons for exclusion included age outside the specified range (n = 8), recent acute exacerbation (n = 6), known severe or unstable CVD (n = 4), inability to complete spirometry or the 6MWT (n = 2), and incomplete clinical records (n = 2).

Participants were recruited using a quota-based sampling approach to obtain three equal groups of 30 patients each, classified according to GOLD airflow-obstruction grades: mild (GOLD 1), moderate (GOLD 2), and severe (GOLD 3). Patients aged 40-55 years were selected to maintain a relatively restricted age range for cardiovascular risk assessment and reduce potential age-related variation in Framingham Risk Score (FRS) estimates [14].

Patients were considered clinically stable if they had experienced no acute COPD exacerbation requiring hospitalization, systemic corticosteroids, or antibiotics during the preceding four weeks. Patients with GOLD 4 airflow obstruction, severe or unstable CVD, or an inability to complete the required investigations were excluded.

Sample Size Calculation

The sample size was estimated for a comparison among three independent groups using a one-way analysis of variance (ANOVA). For the illustrative calculation, a significance level of 0.05, statistical power of 80%, and an assumed medium-to-large effect size (Cohen's f = 0.35) were considered. Under these assumptions, approximately 84 participants were required. Allowing for approximately 7% incomplete data or potential exclusions, the target sample size was set at 90 participants, with 30 participants allocated to each COPD severity group (GOLD 1, GOLD 2, and GOLD 3).

Clinical assessment

History, present symptoms, recent work-up exacerbations, medications, and examination were reviewed to determine clinical stability prior to enrollment. Participants who had acute changes in dyspnea or increased sputum production, fever, or recent hospitalization for an exacerbation were only evaluated following recovery and return to a stable state. The baseline data were obtained for age, sex, smoking history, duration of COPD, medical history, medication use, and conventional cardiovascular risk factors. All the participants were weighed on a calibrated scale in light clothes, without shoes, and measured in a stadiometer, while BMI was calculated as weight in kg/height in meters squared. Seated blood pressure and pulse rate were recorded, and oxygen saturation was recorded if available.

Assessment of COPD Severity

COPD severity was classified according to the Global Initiative for Chronic Obstructive Lung Disease (GOLD) airflow-obstruction grading system using postbronchodilator FEV1 expressed as a percentage of the predicted value. Patients were categorized as GOLD 1 (mild; FEV1 ⩾80% predicted), GOLD 2 (moderate; FEV1 50-79% predicted), and GOLD 3 (severe; FEV1 30-49% predicted). Patients with very severe airflow obstruction (GOLD 4; FEV1 less than 30% predicted) were excluded.

Pulmonary Function Testing

Spirometry was carried out according to the guidelines of the American Thoracic Society/European Respiratory Society [15], while the patient was seated and asked about the maneuver before the test. The maneuvers were attempted at least three times, and the technically acceptable best values were recorded. Parameters measured were forced vital capacity (FVC), FEV1, FEV1/FVC ratio, FEV1 percentage predicted, peak expiratory flow rate (PEFR), and forced expiratory flow between 25% and 75% of FVC (FEF25-75%) when available. The BODE index used percentage-predicted FEV1 for the airflow-obstruction component of the BODE index, as the airflow-obstruction component of the BODE index is based on percentage-predicted FEV1 [16].

Breathlessness and Ability to Exercise

The dyspnea grade before exercise testing was assessed using the mMRC scale from 0 (breathlessness occurs only if they do a lot of exercise) to 4 (too breathless to leave the house or breathless when dressing or undressing). The 6MWT was conducted along a flat, straight, measured corridor with reference to the guidelines set forth by the American Thoracic Society. Participants walked at their own speed as far as possible in six minutes, encouraged regularly (standardized), and clinically monitored for excessive breathing rate, chest discomfort, dizziness, or clinically significant oxygen desaturations; the test ended if safety concerns were raised. The 6MWD was used as a total distance walked.

BODE Index

The BODE index was calculated using the established scoring system described by Celli et al. [11]. The index incorporates four clinical variables: BMI, degree of airflow obstruction measured by FEV1 percentage of predicted value, dyspnea assessed using the mMRC scale, and exercise capacity measured by the MWD. Each component was assigned its corresponding score according to the original BODE scoring criteria. The individual component scores were summed to obtain a total score ranging from 0 to 10, with higher scores indicating greater disease severity and poorer prognosis

The BODE index was calculated by summing component scores for BMI, percentage-predicted FEV1, mMRC grade, and 6MWD using validated thresholds (Table *1*), giving a total score of 0-10, with higher scores indicating greater multidimensional disease burden and poorer functional status [16]. The index was interpreted as a composite measure of disease burden rather than a direct measure of cardiovascular risk.

Component	0 points	1 point	2 points	3 points
BMI (kg/m²)	**>21**	**≤21**	**-**	**-**
FEV1 (% predicted)	≥65	50-64	36-49	≤35
mMRC dyspnea grade	0-1	2	3	4
6MWD (m)	≥350	250-349	150-249	≤149

TABLE 1: Scoring of the BODE index components

BMI: body mass index; FEV1: forced expiratory volume in one second; mMRC: modified Medical Research Council; 6MWD: six-minute walk distance

Total score ranges from 0 to 10; higher scores indicate greater disease burden

Cardiovascular Risk and Electrocardiographic Assessment

Cardiovascular risk and electrocardiographic parameters were assessed as described below. The conventional cardiovascular risk was calculated with the application of the FRS, which includes well-known cardiovascular risk factors [17]. A general CVD risk calculator that was available online was used, with the following values entered: age, sex, systolic blood pressure, antihypertensive treatment, smoking status, diabetes status, total cholesterol, and high-density lipoprotein cholesterol, and the results reported as the estimated 10-year risk of a first cardiovascular event. The model does not include COPD-related processes (e.g., systemic inflammation, chronic hypoxemia, pulmonary hyperinflation, and reduced physical activity), and FRS values were therefore considered rather than as predictions of COPD. Each participant had a standard 12-lead electrocardiogram (ECG) recorded and analyzed for waveforms, intervals, rhythm abnormalities, conduction disturbances and myocardial injury-suggestive changes. The electrocardiographic scoring method described by Rautaharju et al. [18] was used to calculate CIIS, with the following electrocardiographic parameters: Q-wave abnormalities, ST-segment changes, T-wave abnormalities, QRS duration and related measurements, and other predefined characteristics of the validated algorithm. A greater burden of ECG abnormalities associated with myocardial injury was consistent with a higher score. CIIS was not expressed as a percentage risk.

Spirometry Assessment

Spirometry was performed according to the technical standards established by the American Thoracic Society and European Respiratory Society. FEV1, FVC, FEV1/FVC ratio, forced expiratory flow at 25-75% of FVC (FEF25-75), and PEFR were recorded. COPD airflow obstruction was assessed using the postbronchodilator FEV1/FVC ratio, with a value below 0.70 supporting the diagnosis of persistent airflow obstruction. FEV1 percentage of predicted value was used to classify airflow-obstruction severity according to GOLD criteria.

CIIS Scoring and Interpretation

The CIIS was calculated using the electrocardiographic criteria and weighted scoring algorithm described by Rautaharju et al. [18]. Individual ECG measurements and waveform characteristics were assessed and assigned scores according to the original algorithm, with the component scores combined to obtain the total CIIS for each participant. Higher scores indicate a greater burden of ECG abnormalities associated with myocardial injury. The CIIS was analyzed as a continuous variable, and group-level differences were evaluated across the three COPD severity categories. No independent diagnostic threshold was assumed to establish coronary artery disease solely on the basis of the CIIS.

Assessment of Cardiac Infarction Injury Score (CIIS)

The CIIS was assessed using standard 12-lead ECGs. The CIIS is an electrocardiographic scoring system developed to quantify ECG abnormalities associated with myocardial injury and ischemic heart disease. The score was calculated according to the established criteria described by Rautaharju et al. [18]. ECG recordings were evaluated for relevant waveform characteristics, including Q-wave abnormalities, QRS complex changes, and ST-segment and T-wave abnormalities, according to the original scoring methodology. The resulting score was recorded for each participant and compared across the three COPD severity groups.

Echocardiographic and Laboratory Assessment

Transthoracic echocardiography was performed using a commercially available two-dimensional

echocardiography system equipped with Doppler imaging. Left ventricular ejection fraction (LVEF) was assessed using the biplane Simpson's method of discs from apical four-chamber and two-chamber views. The following echocardiographic parameters were evaluated: LVEF (%), left ventricular end-diastolic diameter (LVEDD), left ventricular end-systolic diameter (LVESD), interventricular septal thickness (IVSd), posterior wall thickness (LVPWd), left atrial diameter (LAD), and transmitral E/A ratio. Doppler assessment was used to evaluate relevant diastolic filling parameters. Echocardiographic findings were recorded and compared across the three COPD severity groups.

Data Management and Statistical Analysis of the Collected Data

Data were entered onto a structured master sheet and checked for completeness, internal consistency, duplicate entries, and implausible values; individual records were analyzed separately from summary rows to avoid double counting, and each BODE component was checked against the validated thresholds. Data were analyzed using IBM SPSS Statistics for Windows, Version 24 (Released 2016; IBM Corp., Armonk, New York, United States). Continuous variables were summarized as mean ± standard deviation (SD) and categorical variables as frequencies and percentages, for the whole cohort and for each severity group. Means of continuous variables were compared between the three groups using ANOVA or the Kruskal-Wallis test as appropriate, and pairwise comparisons were used following a significant overall difference. Fisher's exact test or the chi-square test was used for categorical variables. Pearson's or Spearman's correlation coefficient was used for the assessment of associations, which included assessment of the association between spirometric indices, BODE score, FRS, CIIS, and echocardiographic parameters. This exploratory analysis did not include multivariable regression. All tests were two-sided, and values of $p < 0.05$ were considered statistically significant. No imputation of missing observations was conducted, and only valid observations from the data were used for analyses.

The main comparison was between FRS and CIIS in mild, moderate, and severe COPD. Secondary outcomes included pulmonary-function variables, 6MWD, mMRC grade, BODE score, lipid profile, and LVEF by severity groups, and the relationships between pulmonary-function variables, BODE score, FRS, CIIS, and cardiac-function parameters. Results were regarded as associations across cross-sections and not causal relationships.

Results

Baseline characteristics

Of the 90 participants, 78 (86.7%) were male and 12 (13.3%) were female. The overall mean age was 49.11 ± 4.40 years, and the mean BMI was 20.42 ± 2.91 kg/m². The three groups were broadly comparable with respect to age and BMI (Table 2). Male participants accounted for 25 (83.3%) in the mild COPD group, 25 (83.3%) in the moderate COPD group, and 28 (93.3%) in the severe COPD group.

Variable	Mild COPD (n = 30)	Moderate COPD (n = 30)	Severe COPD (n = 30)	Overall (N = 90)
Age (years), mean ± SD	49.33 ± 4.40	47.90 ± 4.37	50.10 ± 4.36	49.11 ± 4.40
Male sex, n (%)	25 (83.3)	25 (83.3)	28 (93.3)	78 (86.7)
BMI (kg/m²), mean ± SD	20.37 ± 2.91	20.11 ± 3.23	20.76 ± 2.57	20.42 ± 2.91
Total cholesterol (mg/dL), mean ± SD	185.4 ± 28.6*	190.2 ± 31.4*	193.5 ± 29.8*	-
HDL cholesterol (mg/dL), mean ± SD	44.2 ± 8.1*	42.6 ± 7.8*	41.3 ± 8.4*	-
Current smokers, n (%)	18 (60.0)*	21 (70.0)*	23 (76.7)*	62 (68.9)*
Diabetes mellitus, n (%)	5 (16.7)*	6 (20.0)*	7 (23.3)*	18 (20.0)*
Receiving antihypertensive treatment, n (%)	8 (26.7)*	9 (30.0)*	10 (33.3)*	27 (30.0)*

TABLE 2: Demographic characteristics, vital parameters, and lipid profile according to COPD severity

COPD: chronic obstructive pulmonary disease; BMI: body mass index; BP: blood pressure; HDL: high-density lipoprotein; LDL: low-density lipoprotein; VLDL: very-low-density lipoprotein

Data are presented as mean ± SD or n (%)

When expressed as a percentage of the predicted value, FEV1 demonstrated a progressive reduction across the three COPD severity groups. The mean FEV1 (% predicted) was 85.40 ± 4.20% in the mild group, 65.30 ± 8.10% in the moderate group, and 38.60 ± 6.50% in the severe group, corresponding to GOLD grades 1, 2, and 3, respectively.

Pulmonary function, dyspnea, exercise capacity, and BODE index

Airflow-related parameters declined progressively with increasing COPD severity (Table 3). Mean FEV1 fell from 2.08 ± 0.59 L in mild COPD to 1.75 ± 0.50 L in moderate and 1.58 ± 0.67 L in severe COPD. The FEV1/FVC ratio was 69.89 ± 1.03%, 68.71 ± 2.19%, and 51.20 ± 5.44%, and FEF25-75% was 4.60 ± 0.70, 3.84 ± 0.87, and 2.64 ± 0.98 L/s in the mild, moderate, and severe groups, respectively, with the severe group showing the greatest impairment. FVC and PEFR showed smaller differences between groups. The severe group had the highest mean mMRC score (2.93 ± 1.17 versus 2.70 ± 1.12 in mild COPD), whereas 6MWD overlapped substantially among groups, with considerable individual variation in severe COPD. The BODE index increased progressively from 4.80 ± 1.77 in mild COPD to 6.73 ± 1.05 in moderate and 8.03 ± 1.00 in severe COPD, reflecting greater multidimensional disease burden with advancing severity.

Parameter	Mild COPD (n = 30)	Moderate COPD (n = 30)	Severe COPD (n = 30)	Overall (n = 90)
FEV1 (L)	**2.08 ± 0.59**	**1.75 ± 0.50**	**1.58 ± 0.67**	**1.80 ± 0.61**
FVC (L)	2.52 ± 0.74	2.36 ± 0.53	2.31 ± 0.50	2.40 ± 0.61
FEV1/FVC (%)	69.89 ± 1.03	68.71 ± 2.19	51.20 ± 5.44	63.27 ± 8.77
FEF25-75% (L/s)	4.60 ± 0.70	3.84 ± 0.87	2.64 ± 0.98	3.69 ± 1.17
PEFR (L/min)	277.23 ± 48.84	283.50 ± 41.44	248.90 ± 37.84	269.88 ± 45.68
mMRC dyspnea score	2.70 ± 1.12	2.73 ± 1.17	2.93 ± 1.17	2.79 ± 1.15
6MWD (m)	340.63 ± 62.83	352.63 ± 64.97	352.50 ± 86.90	348.59 ± 71.72
BODE score	4.80 ± 1.77	6.73 ± 1.05	8.03 ± 1.00	6.52 ± 1.86

TABLE 3: Spirometric parameters, dyspnea, exercise capacity, and BODE index according to COPD severity

FEV1: forced expiratory volume in one second; FVC: forced vital capacity; FEF25-75%: forced expiratory flow between 25% and 75% of FVC; PEFR: peak expiratory flow rate; mMRC: modified Medical Research Council; 6MWD: six-minute walk distance

Data are presented as mean ± SD

Cardiovascular risk, CIIS, and echocardiography

The overall mean FRS was 19.49 ± 3.34%, with similar values in the mild (19.60 ± 2.70%), moderate (19.70 ± 3.10%), and severe (19.17 ± 2.94%) groups and no increasing pattern corresponding to COPD severity (Table 4). Because no prespecified risk category or non-COPD control group was included, these values should not be interpreted as evidence of excess risk attributable specifically to COPD. In contrast, the mean CIIS increased progressively from 17.03 ± 3.85 in mild COPD to 18.46 ± 5.34 in moderate and 20.17 ± 4.13 in severe COPD (overall 18.30 ± 4.80). This indicates a higher burden of ECG abnormalities in severe COPD but does not, by itself, establish acute myocardial injury or causality. Mean LVEF was 50.30 ± 6.54% overall and broadly similar across groups, with no consistent reduction with increasing severity; the echocardiographic flow value was 78.18 ± 5.66 overall.

Parameter	Mild COPD (n = 30)	Moderate COPD (n = 30)	Severe COPD (n = 30)	Overall (n = 90)
Framingham cardiovascular risk (%)	**19.60 ± 2.70**	**19.70 ± 3.10**	**19.17 ± 2.94**	**19.49 ± 3.34**
CIIS	17.03 ± 3.85	18.17 ± 5.34	20.17 ± 4.13	18.46 ± 4.80
Echocardiographic flow value*	77.37 ± 5.00	77.33 ± 5.90	79.83 ± 5.93	78.18 ± 5.66
LVEF (%)	51.30 ± 7.25	49.10 ± 6.37	50.50 ± 6.10	50.30 ± 6.54

TABLE 4: Cardiovascular risk, CIIS, and echocardiographic parameters according to COPD severity

CIIS: Cardiac Infarction Injury Score; LVEF: left ventricular ejection fraction

Data are presented as mean ± SD

Correlation analysis

Pearson correlations among pulmonary, cardiovascular, and echocardiographic measures were weak or negligible (Table *5*). Spirometric parameters correlated weakly and positively with FRS (r = 0.253), weakly and negatively with BODE (r = -0.226), and very weakly with CIIS (r = 0.077). FRS showed a weak positive correlation with CIIS (r = 0.130) and a negligible correlation with BODE (r = -0.045), and BODE correlated weakly with CIIS (r = 0.105). Echocardiographic parameters showed negligible correlations with spirometry (r = -0.015), FRS (r = 0.068), BODE (r = -0.005), and CIIS (r = -0.011). These measures were therefore not strongly linearly related and appear to capture partially different aspects of COPD-related disease burden.

Parameter	Spirometry	FRS	BODE	CIIS	Echocardiography
Spirometry	**1.000**	**0.253**	**-0.226**	**0.077**	**-0.015**
FRS	0.253	1.000	-0.045	0.130	0.068
BODE	-0.226	-0.045	1.000	0.105	-0.005
CIIS	0.077	0.130	0.105	1.000	-0.011
Echocardiography	-0.015	0.068	-0.005	-0.011	1.000

TABLE 5: Pearson correlation matrix among pulmonary, cardiovascular, and echocardiographic parameters

FRS: Framingham Risk Score; CIIS: Cardiac Infarction Injury Score

Values are Pearson correlation coefficients (r)

Discussion

The present study shows that there is a significant burden of CVD in clinically stable COPD patients and highlights the difference between traditional cardiovascular risk assessment and electrocardiographic evidence of myocardial injury. The mean FRS was 19.31 ± 3.34% and was not significantly different between mild, moderate, and severe COPD, but the CIIS increased progressively from 17.03 ± 3.85 in mild disease to 20.17 ± 4.13 in severe disease.

Our results also tally with Sharma et al. [19], who analyzed cardiovascular risk in Indian patients with stable COPD and revealed that the Framingham cardiovascular score identified 45% of the patients as having high cardiovascular risk. They also showed that their study showed elevated hs-CRP and atherogenic indices, indicating that cardiovascular risk exists in COPD even when assessed using conventional clinical criteria.

Previous studies have highlighted the relationship between COPD and cardiovascular complications. Brekke et al. evaluated 897 patients hospitalized for COPD exacerbation and demonstrated the potential utility of

the CIIS in identifying previously unrecognized myocardial infarction [20]. More recently, de-Torres et al. investigated cardiovascular risk assessment in 529 patients with COPD using SCORE2-family and SMART risk prediction models alongside coronary artery calcium scoring, highlighting the potential value of combining clinical risk assessment with imaging-based evaluation [21]. Similarly, Li et al. reported associations between CVD and COPD severity in a large cross-sectional Chinese hospital population, although the cross-sectional design limits causal interpretation [22].

By contrast, recently Liu et al. [23] have shown that in COPD, the Framingham model was not discriminatory enough and that the addition of pulmonary-function parameters might help to better predict cardiovascular risk. These results are consistent with our previous finding that conventional FRS may be relatively insensitive to the severity of COPD, as other mechanisms of COPD, such as systemic inflammation and hypoxemia and reduced physical activity, are not directly included in the score.

In addition, the multidimensional assessment performed in our study with BODE is confirmed by Celli et al. [24], who showed that BODE incorporates BMI, airflow obstruction, dyspnea, and exercise capacity and gives prognostic information beyond FEV1. Because of this, our use of BODE, 6MWD and mMRC, along with FRS and CIIS, allows for a more comprehensive description of cardiopulmonary burden of disease.

The current findings support further evaluation of an integrated cardiopulmonary assessment strategy. The relative stability of FRS despite the gradation of COPD, in addition to the progressive rise in CIIS, suggests that assessment of cardiac abnormalities by ECG might pick up abnormalities that aren't fully reflected by conventional cardiovascular risk estimation. The current results validate an integrated cardiopulmonary strategy that includes multidimensional COPD severity assessment, functional assessment, and spirometry and cardiovascular evaluation. These associations should not be interpreted as causal, given that this was a cross-sectional study and there was no multivariable regression models used or longitudinal cardiovascular outcomes.

Limitations

This study has several limitations. The study is cross-sectional, hospital-based in design and therefore cannot establish temporal or causal relationships between the severity of COPD and cardiovascular abnormalities. Sample size was modest (30 participants per severity group); the participants were all aged 40-55 years, and this may restrict the generalizability of the results to older patients and to community populations. The cardiovascular burden observed may have been reduced by excluding patients with severe or unstable CVD. There was also no direct comparison provided in the study with a group of non-COPD controls. Cardiovascular risk was measured via the FRS and CIIS, which are two different constructs and cannot be compared as outcomes. In addition, the exploratory correlation analysis did not perform multivariable regression, and the potential confounding effect of smoking, age, blood pressure, metabolic factors, or other clinical characteristics could not be completely discussed. In addition, the cross-sectional evaluation lacked longitudinal cardiovascular events and mortality outcomes, and, consequently, the prognostic impact of the observed differences in CIIS is unclear.

Conclusions

Patients with COPD exhibited a substantial cardiovascular risk burden. The FRS was broadly similar across the COPD severity groups, whereas the CIIS increased progressively with disease severity. These findings suggest that electrocardiographic assessment may provide complementary information regarding cardiac abnormalities not fully captured by conventional cardiovascular risk estimation. An integrated assessment incorporating COPD severity, spirometry, functional status, the BODE index, conventional cardiovascular risk factors, electrocardiography, and cardiac function may provide a more comprehensive evaluation of cardiopulmonary status. Larger studies incorporating appropriate control groups and longitudinal cardiovascular outcomes are needed to clarify the prognostic significance of CIIS and its potential role in cardiovascular risk stratification among patients with COPD.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Mohiuddin F. Shaikh, Kiran Buge, Pranali V. Sonawane

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Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. Institutional Ethics Committee, VIMS-Dr. Vithalrao Vikhe Patil Foundation's Medical College and Hospital, Ahilyanagar, Maharashtra, India. issued approval VIMS/IEC/C/2025/123 (Protocol ID: 2025/123). **Animal subjects:** All authors have confirmed that this study did not involve animal subjects or tissue. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

Acknowledgements

During the preparation of this manuscript, the authors used ChatGPT (OpenAI) to search for the references.

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