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Unveiling atrial electromechanical delay in chronic obstructive pulmonary disease: an observational cohort study from north India

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Abstract

Chronic obstructive pulmonary disease (COPD) is often associated with cardiovascular complications such as atrial fibrillation (AF), heart failure, and myocardial infarction. AF is highly prevalent in COPD, yet the mechanisms linking them remain unclear. This study investigates the role of atrial electromechanical delay (AEMD) in predicting cardiovascular outcomes in COPD patients. This prospective cohort study included 60 COPD patients (forced expiratory volume in 1 second/forced vital capacity <0.7) from August 2022 to March 2024. Patients with pre-existing heart disease and other major comorbidities were excluded. Participants underwent spirometry, electrocardiogram (ECG), echocardiography, and N-terminal pro type natriuretic peptide (NT-proBNP) testing. AEMD was measured at the lateral and medial mitral annuli and tricuspid annulus. Primary endpoints included AF incidence, heart failure, stroke, and COPD exacerbations, while secondary endpoints were hospitalization and mortality. AEMD values were significantly higher in patients with AF (75.4±5.9 ms *vs*. 70.4±4.1 ms, p=0.004), heart failure, and COPD exacerbations, particularly at the lateral and medial mitral annuli. AEMD at the tricuspid annulus was strongly associated with mortality (p=0.04). P-wave dispersion (41.2±6.4 ms *vs*. 36.1±4.2 ms, p=0.001) and QT dispersion (49.3±8.9 ms *vs*. 42.1±6.8 ms, p=0.002) were significantly elevated in patients with adverse outcomes. Elevated NT-proBNP levels (>1000 pg/mL) correlated with prolonged AEMD, suggesting cardiac stress. AEMD, particularly at the mitral and tricuspid annuli, is a strong predictor of AF, heart failure, and COPD exacerbations. P-wave and QT dispersion are associated with increased hospitalization and mortality, highlighting their role in risk stratification. These findings support the use of AEMD and ECG parameters as early markers for cardiovascular complications in COPD. Further validation in larger cohorts is needed.

Key words: atrial electromechanical delay, chronic obstructive pulmonary disease, atrial fibrillation, cardiovascular outcomes, P-wave dispersion.

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Introduction

Chronic obstructive pulmonary disease (COPD) is a progressive lung disorder characterized by airflow limitation due to emphysema and chronic bronchitis, with systemic inflammatory effects. By 2030, it is projected to be the third leading cause of mortality globally [1]. COPD significantly impacts cardiovascular health, contributing to complications such as pulmonary hypertension, cor pulmonale, and ventricular dysfunction. Even in mild-to-moderate cases, its interplay between pulmonary and cardiac systems increases morbidity and mortality.

Cardiovascular manifestations of COPD include heart failure, myocardial infarction, and arrhythmias, particularly atrial fibrillation (AF), the most prevalent arrhythmia in COPD patients [2]. Despite its frequent occurrence, the pathophysiological mechanisms linking COPD and AF remain unclear. COPD-related atrial conduction disorders—prolonged atrial conduction times, re-entry circuits, and abnormal atrial tissue—promote AF development and persistence [2].

Tissue Doppler imaging (TDI) and electrocardiography (ECG) are key tools for assessing atrial conduction disorders. Atrial electromechanical delay (AEMD), measured *via* TDI as the time between the P-wave onset and the late diastolic Am-wave, is a valuable non-invasive marker for evaluating atrial substrates and AF risk [3]. Prolonged P-waves, common in COPD patients, are linked to structural atrial changes due to inflammation and comorbidities such as hypertension, hypertrophic cardiomyopathy, and atrial septal defect [4].

Reduced forced expiratory volume in 1 second (FEV1) is independently associated with new-onset AF. AEMD provides insight into myocardial electrical depolarization and contraction timing, serving as a marker of atrial dysfunction [3].

This study investigates AEMD and its role in predicting AF and cardiovascular outcomes in COPD patients. It aims to assess AF predictors, analyze AEMD's significance in AF development and complications, and identify relevant ECG and echocardiographic parameters. Findings may contribute to improved risk stratification and targeted interventions for COPD patients with cardiovascular risks.

Materials and Methods

This prospective cohort and observational study evaluated the clinical outcomes of AEMD and other echocardiographic parameters in patients with COPD. The study was conducted in the Departments of Tuberculosis & Respiratory Diseases and Cardiology at the Institute of Medical Sciences, Banaras Hindu University, Varanasi. Approval for the study protocol was granted by the Institutional Ethics Committee, and written informed consent was obtained from all participants. The study period spanned from August 2022 to March 2024.

The study included 60 COPD patients, with inclusion criteria being a post-bronchodilator FEV1/forced vital capacity (FVC) ratio <0.7. Patients meeting any of the following exclusion criteria were not included: (i) refusal to participate, (ii) pulmonary diseases other than COPD, (iii) pre-existing heart conditions, including documented permanent AF, rheumatic heart disease, left ventricular ejection fraction <50%, symptomatic heart failure, moderate to severe valvular heart disease, documented coronary artery disease (*e.g.*, myocardial infarction, angiographic stenosis ≥50%, positive non-invasive ischemic test, Q wave on ECG, or wall motion abnormalities on echocardiography), angina, congenital heart disease, pacemaker implantation, or pre-excitation syndrome on ECG, (iv) chronic kidney disease, (v) systemic inflammatory or autoimmune disorders, (vi) hemodynamically unstable patients.

Each patient underwent a detailed clinical history and comprehensive evaluations, including routine blood investigations, thyroid profile, HbA1c, lipid profile, spirometry, and a 6-minute walk test. Additional parameters assessed included N-terminal pro brain natriuretic peptide (NT-proBNP) levels and ECG variables such as P-wave duration, dispersion, amplitude, QT interval duration, QT dispersion, PR and RR intervals, and QRS duration. Echocardiographic evaluations included 2D echocardiography for chamber dimensions, ejection fraction, and wall thickness, along with specialized assessments like pulse wave and TDI. Patients were followed at regular intervals (3, 6, 9, 12, 15, and 18 months) to record clinical outcomes

COPD was diagnosed based on Global Initiative for Chronic Obstructive Lung Disease (GOLD) criteria. A post-bronchodilator FEV1/FVC ratio <0.7 on spirometry confirmed airflow limitation. Spirometry was performed using an American Thoracic Society (ATS)/ERS-standardized spirometer, with at least three acceptable maneuvers, and the highest FEV1/FVC ratio was recorded. Patients also underwent a 6-minute walk test to assess functional capacity, following the ATS guidelines.

AEMD was measured using TDI by recording the time interval from the onset of the P-wave on surface ECG to the peak of the late diastolic Am-wave at the lateral and medial mitral annuli and the tricuspid annulus. Measurements were taken in apical four-chamber view, ensuring consistent cardiac cycle timing. The mean values from three consecutive cardiac cycles were used to minimize variability.

P-wave dispersion was calculated as the difference between the maximum and minimum P-wave duration recorded in a 12-lead surface ECG. P-wave duration was measured manually using digital calipers, and the mean of three consecutive beats was recorded to reduce variability.QT dispersion was determined as the difference between the longest and shortest QT intervals across all 12 ECG leads. The QT interval was measured from the beginning of the QRS complex to the end of the T wave, using the tangent method to identify T wave termination. QT intervals were corrected for heart rate using Bazett's formula ($QTc=QT/\sqrt{RR}$). Measurements were repeated by two independent observers, and intra-observer variability was assessed using intraclass correlation coefficients.

Study endpoints

Primary Endpoints: frequency of AF episodes, heart failure, stroke, and COPD exacerbations during the 18-month follow-up.

Secondary Endpoints: hospitalization needs and mortality due to disease progression or complications.

The sample size was determined based on prior studies conducted by Cimci *et al*., Rodríguez-Mañero, Huang, and Karaliute *et al*., which evaluated AEMD in patients with COPD [4-7].

Assuming a power of 80% and a significance level of 0.05, the required sample size was calculated as 52. To account for potential dropouts, the target sample size was increased by 15%, resulting in a total of 60 participants. Numerical variables were reported as mean ± standard deviation, and categorical variables as numbers and percentages. Dichotomous variables were presented as frequencies and percentages. Descriptive statistics were applied using IBM SPSS version 20.0 (IBM, Armonk, NY, USA). Normally distributed data were analyzed using one-way analysis of variance, while categorical differences were assessed using the chi-squared test. Associations between factors were analyzed using logistic regression. A $p<0.05$ was considered statistically significant. This robust methodology ensured the reliability and validity of findings, addressing clinical outcomes associated with AEMD and echocardiographic parameters in COPD patients.

Results

Demographical and baseline data

As shown in Table 1, the study involved 60 participants with a mean age of 59.75±8.60 years. The majority (50%) were aged between 41 and 60 years, with 48.3% over 60 years. The cohort included 34 males (56.7%) and 26 females (43.3%). A significant portion of participants had a history of smoking or biomass exposure (11.92 years), and hypertension (1.09 years) was common. Dyspnea was the most frequent and long-standing symptom among participants.

Primary and secondary outcomes

Over 1.5 years, 60% of participants experienced no episodes of AF, 23.3% had a single episode of heart failure, and 36.7% had one exacerbation of COPD. Hospitalization was reported in 31.7% of cases, and mortality occurred in 6.7%.

Associations between atrial electromechanical delay, clinical parameters, and outcomes

Participants with AF demonstrated significantly longer AEMD values compared to those without AF (Table 2). At the lateral mitral annulus, the mean AEMD was 75.42±5.99 ms for participants with AF, compared to 70.42±4.15 ms for those without AF. Similar trends were observed at the medial mitral annulus and tricuspid right ventricular free wall annulus. AEMD values were significantly higher in COPD patients with heart failure across all measured locations ($p<0.05$), as presented in Table 2. AEMD at the lateral mitral annulus emerged as a potential marker for COPD exacerbation, showing significant prolongation in patients experiencing exacerbations. However, no significant associations were observed at the medial mitral annulus or tricuspid annulus.

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Prolonged AEMD at the lateral and medial mitral annuli was significantly associated with increased hospitalization risk in COPD patients ($p<0.05$), as noted in Table 2. Conversely, no significant association was identified for AEMD at the tricuspid annulus. A significant relationship between prolonged AEMD at the tricuspid annulus and mortality in COPD patients was observed ($p<0.05$). Non-survivors exhibited markedly longer AEMD in this region, as detailed in Table 2. No significant associations with mortality were found for AEMD at the lateral or medial mitral annuli.

NT-proBNP levels were categorized into three groups (<125 pg/mL, 125-1000 pg/mL, and >1000 pg/mL), and their association with AEMD is summarized in Table 2. Elevated NT-proBNP levels, indicative of heart failure and cardiac stress, were associated with prolonged AEMD, underscoring its potential utility as a marker of cardiac severity. Comparisons of AEMD between groups with

Table 1. Demographic characteristics of the study participants.

Parameters		Frequency (n)/mean	%/SD
Age group	20-40	2	1.7%
	41-60	60	50%
	>60	58	48.33%
Mean age	59.75	8.60	
Gender	Female	52	43.3%
	Male	68	56.7%
Symptoms	Dyspnea (years)	8.73	9.08
	Cough (years)	2.05	4.60
	Sputum production	42	35
Co-morbidities	HTN (years)	1.09	2.71
	T2DM (months)	5.97	20.24
	ATT history	22	18.3
	Hypothyroidism	8	6.7
Habits	Smokers/biomass exp. (years)	11.92	11.35
	Addictive substance (Ganja) (years.)	3.40	7.03
	Alcohol intake (years)	3.23	7.05

HTN, hypertension; T2DM, type 2 diabetes mellitus; ATT, anti-tuberculosis treatment. *p≤0.05 is significant. n, frequency; SD, standard deviation.

Table 2. Associations between atrial electromechanical delay, clinical parameters, and outcomes.

Condition/Parameter	Sub-category	Lateral mitral annulus (in ms)		Medial mitral annulus (in ms)		Tricuspid right ventricle free wall annulus (in ms)	
		Mean	SD	Mean	SD	Mean	SD
Atrial fibrillation	Present	75.42	5.99	63.67	5.6	50.75	5.2
	Absent	70.42	4.15	59.63	4.01	47.88	4.62
t-test/p-value		2.15/0.004*		3.05/0.010*		1.88/0.036*	
Heart failure	Present	76.73	6.33	65.27	5.4	51.67	3.7
	Absent	71.73	4.04	59.8	4.16	48.27	4.91
t-test / p-value		3.28/0.040*		6.21/0.002*		3.70/0.001*	
COPD exacerbation	Present	73.48	5.74	61.44	4.5	49.04	4.93
	Absent	70.26	4.28	59.52	4.28	47.48	4.71
t-test/p-value		-2.108/0.042*		-1.777/0.081		-1.233/0.222	
History of hospitalization	Present	74.3	6.59	62.87	5.99	49.39	5.39
	Absent	69.74	4.04	59.3	4.41	47.44	4.87
t-test/p-value		-3.23/0.002*		-2.63/0.011*		-1.47/0.147	
Mortality status	Alive	73.50	3.11	63.50	1.73	53.75	1.89
	Dead	72.75	5.25	62.50	7.05	49.00	6.98
t-test/p-value		0.28/0.35		0.26/0.95		1.36/0.04	
NT-proBNP	<125	69.08	3.74	58.13	3.63	45.63	3.89
	125-1000	73.27	4.52	61.93	4.50	49.67	4.63
	>1000	81.50	5.72	68.66	3.78	55.16	1.60
F/p-value		10.16/0.021*		11.00/0.037*		11.25/0.034*	
6 Minute Walk Test	<4% desat.	69.81	4.69	58.62	4.70	46.00	3.88
	≥4% desat.	74.25	7.02	62.44	6.142	48.88	5.702
t-test/p-value		-2.591/0.020*		-2.075/0.051*		-1.836/0.082	

AEMD, atrial electromechanical delay; COPD, chronic obstructive pulmonary disease; NT-proBNP, N-terminal pro brain natriuretic peptide; desat.- desaturatio; SD, standard deviation. *p≤0.05 is significant.

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non-significant (<4%) and significant (≥4%) oxygen desaturation revealed a significant difference at the lateral mitral annulus (69.81±4.69 ms *vs*. 74.25±7.02 ms, t=-2.591, p=0.020), as shown in Table 2. A marginal difference was observed at the medial mitral annulus (58.63±4.70 ms *vs*. 62.44±6.14 ms, t=-2.075, p=0.051). However, no statistically significant difference was noted at the tricuspid annulus.

P-wave dispersion and adverse clinical conditions

Increased P-wave dispersion was significantly associated with adverse clinical conditions, including AF, heart failure, and COPD exacerbations, as detailed in Table 3. Hospitalized patients, non-survivors, and those with significant oxygen desaturation (≥4% O2 fall) also exhibited higher P-wave dispersion. Elevated NT-proBNP levels corresponded with greater atrial electrical instability.

QT dispersion and clinical outcomes

Increased QT dispersion was significantly associated with adverse clinical outcomes, such as heart failure, COPD exacerbations, hospitalization, and mortality (p<0.00001), as highlighted in Table 4.

Discussion

COPD, primarily affecting the lungs, also has significant cardiovascular consequences, including heart failure, myocardial infarction, and arrhythmias such as AF. Characterized by progressive airflow limitation and chronic inflammation, COPD's interplay with the cardiac system frequently leads to dysfunction. AF in COPD patients is linked to prolonged atrial conduction times, though its pathophysiology remains incompletely understood. Techniques like transthoracic echocardiography and ECG, particularly TDI, help assess atrial conduction abnormalities and predict AF risk by measuring AEMD.

The study included 60 participants, mostly middle-aged or older, with a predominance of males and a high prevalence of smoking and hypertension. Dyspnea was the most frequent and longstanding symptom, with severe dyspnea (mMRC grade 4) observed in 60% of participants, indicating advanced respiratory impairment. The majority were classified in group E, signifying high disease severity and exacerbation risk. Previous studies by Huang *et al.* also linked mMRC grade ≥2 to increased symptoms and exacerbation risk, with most participants presenting mMRC=4 [6]. Rodríguez-Mañero *et al.* reported similar findings in their AF cohort [5], with a comparable age distribution and male predominance, consistent with the observations of Yilmaz *et al.* [3], and

Table 3. P-wave dispersion and clinical outcomes.

		P-wave dispersion (mean±SD)	p-value
Atrial fibrillation	No	33.1±7.6	<0.00001
	Yes	41.2±6.5	
Heart failure	No	33.6±7.5	<0.00001
	Yes	44.6±3.6	
COPD exacerbation	No	29.1±4.5	<0.00001
	Yes	42.3±5.2	
Hospitalization	No	33.1±8.0	<0.00001
	Yes	41.6±5.1	
Mortality	Live	35.8±8.3	<0.00001
	Dead	42.3±3.3	
Significant desaturation (≥4% O_2 fall)	No	35.25±8.3	<0.00001
	Yes	39.13±7.4	
NT-proBNP	<125	31.9±8.3	<0.00001
	125-1000	38.3±6.3	
	>1000	44.4±6.5	

COPD, chronic obstructive pulmonary disease; NT-proBNP, N-terminal pro brain natriuretic peptide; SD, standard deviation. p<0.05 is significant.

Table 4. QT dispersion and primary and secondary end points.

		QT Dispersion (mean±SD)	p-value
Heart failure	No	41.04±6.9	<0.00001
	Yes	49.36±8.9	
COPD exacerbation	No	40±6.6	<0.00001
	Yes	45.6±8.6	
Hospitalization	No	39.8±6.6	<0.00001
	Yes	48.41±7.9	
Mortality	Live	47.2±6.1	<0.00001
	Dead	42.28±8.2	

COPD, chronic obstructive pulmonary disease; SD, standard deviation. p<0.05 is significant

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Cimci *et al*. [4], who also identified smoking as the leading cause of COPD.

Echocardiographic findings revealed diastolic dysfunction, with higher E/A ratios and lower myocardial velocities, suggesting myocardial impairment. These results align with Cimci *et al.* [4], who observed an E/Ea ratio of 7.1 in AF patients. Karaliute *et al*. proposed that the spectral tissue Doppler-derived E/e' ratio serves as a predictive marker for AF [7], while Correale *et al*. highlighted the prognostic role of TDI in major cardiac diseases [8]. Their findings noted that reduced lateral Ea velocity (≤8 cm/s) in older individuals could indicate poor left ventricle relaxation.

Over a follow-up period of 1.5 years, 60% of participants had no AF episodes, while 23.3% experienced at least one episode of heart failure, and 36.7% had at least one COPD exacerbation. Hospitalization was required in 31.7% of cases, and 6.7% of participants experienced mortality. Grymonprez *et al*. found that 14% of COPD patients had AF, with an AF risk ratio of 1.28 [9]. Similarly, Nadeem *et al*. noted a higher incidence of ischemic stroke in patients with both COPD and AF, complicating management [10]. Lopez *et al.* further emphasized the challenge of managing these comorbid conditions, underscoring the need for better risk stratification strategies [11].

AEMD values measured at the lateral and medial mitral annulus and the tricuspid annulus were significantly higher in participants with AF, heart failure, and COPD exacerbation compared to those without these conditions ($p<0.05$). Ortiz-Leon *et al.* linked atrial enlargement and annular dilatation to AF, which is consistent with our findings [12]. Yilmaz *et al.* previously reported prolonged AEMD intervals in COPD exacerbation cases, and our results confirm this association [3]. Prolonged AEMD at the tricuspid annulus correlated with higher mortality rates in COPD patients. Additionally, elevated NT-proBNP levels (>1000) were associated with longer AEMD intervals, suggesting its potential role as a marker for cardiac involvement in patients with elevated NT-proBNP levels.

Significant relationships were observed between gender, smoking, hypertension, type 2 diabetes mellitus, HbA1C>6.5%, and P-wave dispersion with ≥2 episodes of AF. These risk factors may contribute to the frequency of AF episodes in COPD patients. Goudis *et al*. identified similar predictors of AF in COPD, including male sex, age, and hypertension [13]. Scheuermeyer *et al*. found that COPD exacerbation was strongly linked to new-onset AF [14], particularly in older individuals, while Jagannatha *et al.* highlighted P-wave dispersion as a reliable predictor of new-onset AF [15].

In heart failure patients, significant associations were found between gender, smoking, hypertension, and P-wave dispersion. However, parameters such as TAPSE, lateral and medial mitral annulus, and tricuspid annulus measurements did not show significant differences. This contrasts with the findings of Kjaergaard *et al.* [16], who identified TAPSE as an independent predictor of heart failure. Seyfeli *et al.* observed that P-wave duration and dispersion correlated with both ventricular and atrial arrhythmias in heart failure, reinforcing the role of ECG parameters in arrhythmia risk assessment [17].

For COPD exacerbation, gender, smoking habits, hypertension, P-wave dispersion, and lateral mitral annulus measurements showed significant differences between groups. TAPSE values were significantly lower in participants with one or more exacerbations compared to those without exacerbations. Cimci *et al*. found longer P-wave dispersion in COPD patients [4], while Romiti *et al.* noted a higher prevalence of COPD in males, with an increased risk of stroke in patients with COPD-related right heart failure [18].

P-wave dispersion was significantly higher in patients with AF (41.22±6.47), heart failure (44.57±3.58), COPD exacerbation (42.28±5.23), hospitalization (41.59±5.13), and mortality (42.25±3.34). The mean P-wave dispersion also correlated with elevated NT-proBNP levels (31.92±8.3, 38.33±6.34, 44.4±6.47), indicating a link between prolonged atrial conduction and cardiac dysfunction. Cimci *et al.* found that longer P-wave dispersion in COPD patients may serve as a non-invasive marker for atrial remodeling and AF prediction [4].

QT dispersion was significantly higher in patients with heart failure (49.36±8.94), COPD exacerbation (45.56±8.58), hospitalization (48.41±7.9), and mortality (47.2±6.14). Acikalin *et al.* observed increased QT dispersion in heart failure patients [19], while Lin *et al*. noted that QTc prolongation serves as an indicator of mortality risk [20]. Sievi *et al*. and Van Oekelen *et al.* also found prolonged QT intervals in COPD patients, suggesting a strong correlation with cardiac morbidity and mortality [21,22].

Conclusions

This study highlights the intricate interplay between COPD and cardiovascular abnormalities, emphasizing the clinical significance of AEMD as a predictive marker for adverse outcomes such as AF, heart failure, and exacerbations. AEMD measurements, particularly at the lateral mitral annulus, were associated with heightened risks of AF, hospitalization, and disease progression. Furthermore, prolonged AEMD at the tricuspid annulus was strongly linked to increased mortality, underscoring its prognostic value. Key findings demonstrated that elevated NT-proBNP levels, prolonged P-wave dispersion, and increased QT dispersion serve as important indicators of cardiovascular stress and adverse clinical conditions in COPD patients. These parameters provide valuable insights into the underlying pathophysiological mechanisms, including atrial remodeling, myocardial dysfunction, and systemic inflammation, which collectively contribute to disease complexity.

The demographic and clinical profiles of the study cohort further contextualize these observations. Older age, male predominance, significant smoking or biomass exposure, and comorbidities such as hypertension were prevalent, reflecting a population at high risk for cardiovascular complications. Advanced echocardiographic and electrocardiographic techniques, including TDI, facilitated precise assessment of cardiac electrical and mechanical functions, aiding in risk stratification and personalized management.These findings underscore the necessity for comprehensive cardiovascular evaluation in COPD patients, even in those without overt cardiac symptoms. Early identification of at-risk individuals through non-invasive markers like AEMD, P-wave dispersion, and QT dispersion can enable timely interventions, potentially reducing morbidity and mortality. Future studies should explore targeted therapies and integrated care models to address the dual burden of COPD and cardiovascular disease, ultimately improving patient outcomes and quality of life.

This study has several limitations. First, COPD staging based on the GOLD classification was not performed, which may have limited a stratified analysis of disease severity and its impact on AEMD and cardiovascular outcomes. Future studies should categorize COPD patients into GOLD stages (I-IV) to improve risk stratification. Second, the study was conducted in a single-center setting with a relatively small sample size, which may limit the generalizability of the findings. A larger, multi-center cohort is needed to confirm these results. Third, we did not assess inter-observer and intra-observer variability in AEMD measurements, which could have introduced measurement bias. Standardized protocols with blinded assessments should be employed in future research to ensure consistency. Lastly, while this study focused on AEMD as a predictor of cardiovascular events in COPD, additional markers such as inflammatory cytokines and autonomic dysfunction parameters were not included. Expanding the scope of future studies to incorporate these factors may enhance the understanding of atrial electromechanical alterations in COPD.

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Patient consent for publication: written informed consent was taken from all participants for publication and use of their data for research purposes.

Availability of data and materials: all data generated or analyzed during this study are included in this published article. The data used to support the findings of this study are available from the corresponding author upon request. The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

Declaration of generative artificial intelligence and artificial intelligence-assisted technologies in the writing process: AI was used to correct grammatical errors only.

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