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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 b-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.

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 atrial electromechanical delay (AEMD) and other echocardiographic parameters in patients with chronic obstructive pulmonary disease (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/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 atrial fibrillation (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 (CKD), (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 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 tissue Doppler imaging. 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 ATS/ERS-standardized spirometer, with at least three acceptable maneuvers, and the highest FEV1/FVC ratio recorded. Patients also underwent a 6-minute walk test (6MWT) to assess functional capacity, following American Thoracic Society (ATS) guidelines.

Atrial electromechanical delay (AEMD) was measured using tissue Doppler imaging (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 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/√RR). Measurements were repeated by two independent observers, and intra-observer variability was assessed using intraclass correlation coefficients (ICCs).

Study endpoints

Primary Endpoints: Frequency of atrial fibrillation 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 (SD), 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. Normally distributed data were analyzed using one-way analysis of variance (ANOVA), while categorical differences were assessed using the Chi-squared test. Associations between factors were analyzed using logistic regression. A p-value <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 41–60 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. Dyspnoea 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 atrial fibrillation (AF), 23.3% had a single episode of heart failure, and 36.7% had one exacerbation of chronic obstructive

pulmonary disease (COPD). Hospitalization was reported in 31.7% of cases, and mortality occurred in 6.7%.

Associations between AEMD, clinical parameters, and outcomes

Participants with atrial fibrillation (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.

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 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 atrial fibrillation, 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 atrial fibrillation (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 electrocardiography (ECG), particularly tissue Doppler imaging (TDI), help assess atrial conduction abnormalities and predict AF risk by measuring atrial electromechanical delay (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 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 tissue Doppler imaging (TDI) in major cardiac diseases [8]. Their findings noted that reduced lateral Ea velocity (≤8 cm/s) in older individuals could indicate poor LV 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-pro BNP levels (>1000) were associated with longer AEMD intervals, suggesting its potential role as a marker for cardiac involvement in patients with elevated NT-pro BNP levels.

Significant relationships were observed between gender, smoking, hypertension, type 2 diabetes mellitus (T2DM), 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-pro BNP 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 chronic obstructive pulmonary disease (COPD) and cardiovascular abnormalities, emphasizing the clinical significance of atrial electromechanical delay (AEMD) as a predictive marker for adverse outcomes such as atrial fibrillation (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 tissue Doppler imaging, 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.

References

1. Quaderi SA, Hurst JR. The unmet global burden of COPD. Glob Health Epidemiol Genomics. 2018;3:e4.
2. Konecny T, Park JY, Somers KR, et al. Relation of chronic obstructive pulmonary disease to atrial and ventricular arrhythmias. Am J Cardiol. 2014;114:272-7.
3. Yilmaz A, Can S, Perincek G, et al. Atrial electromechanical delay, neutrophil-to-lymphocyte ratio, and echocardiographic changes in patients with acute and stable chronic obstructive pulmonary disease. J Res Med Sci. 2022;27:64.
4. Cimci M, Borekci S, Kilickiran Avci B, et al. Assessment of atrial electromechanical delay and P wave dispersion in patients with chronic obstructive pulmonary disease. Turk Kardiyol Dern Ars. 2020;48:263-9.

5. Rodríguez-Mañero M, López-Pardo E, Cordero A, et al. A prospective study of the clinical outcomes and prognosis associated with comorbid COPD in the atrial fibrillation population. Int J Chron Obstruct Pulmon Dis. 2019;14:371-80.
6. Huang WC, Wu MF, Chen HC, et al. Features of COPD patients by comparing CAT with mMRC: a retrospective, cross-sectional study. NPJ Prim Care Respir Med. 2015;25:15063.
7. Karaliute R, Jureviciute J, Jurgaityte J, et al. The predictive value of tissue Doppler indices for early recurrence of atrial fibrillation after electrical cardioversion. Clin Interv Aging. 2020;15:1917-25.
8. Correale M, Totaro A, Ieva R, et al. Tissue Doppler imaging in coronary artery diseases and heart failure. Curr Cardiol Rev. 2012;8:43-53.
9. Grymonprez M, Vakaet V, Kavousi M, et al. Chronic obstructive pulmonary disease and the development of atrial fibrillation. Int J Cardiol. 2019;276:118-24.
10. Nadeem R, Sharieff A, Tanna S, et al. Potential augmentation of the risk of ischemic cerebrovascular accident by chronic obstructive pulmonary disease in patients with atrial fibrillation. J Stroke Cerebrovasc Dis. 2015;24:1893-6.
11. Lopez CM, House-Fancher MA. Management of atrial fibrillation in patients with chronic obstructive pulmonary disease. J Cardiovasc Nurs. 2005;20:133-40.
12. Ortiz-Leon XA, Posada-Martinez EL, Trejo-Paredes MC, et al. Understanding tricuspid valve remodelling in atrial fibrillation using three-dimensional echocardiography. Eur Heart J Cardiovasc Imaging. 2020;21:747-55.
13. Goudis CA. Chronic obstructive pulmonary disease and atrial fibrillation: an unknown relationship. J Cardiol. 2017;69:699-705.
14. Scheuermeyer FX, Mackay M, Christenson J, et al. There are sex differences in the demographics and risk profiles of emergency department patients with atrial fibrillation and flutter, but no apparent differences in ED management or outcomes. Acad Emerg Med. 2015;22:1067-75.
15. Jagannatha GN, Antara IM, Kosasih AM, et al. P-wave peak time and P-wave dispersion in surface electrocardiography as initial predictors of new-onset atrial fibrillation in early-onset hypertension. Hypertens Res. 2024;47:137-48.
16. Kjaergaard J, Akkan D, Iversen KK, et al. Right ventricular dysfunction as an independent predictor of short- and long-term mortality in patients with heart failure. Eur J Heart Fail. 2007;9:610-6.
17. Seyfeli E, Duru M, Kuvandık G, et al. Effect of obesity on P-wave dispersion and QT dispersion in women. Int J Obes. 2006;30:957-61.

18. Romiti GF, Corica B, Pipitone E, et al. Prevalence, management and impact of chronic obstructive pulmonary disease in atrial fibrillation: a systematic review and meta-analysis of 4,200,000 patients. Eur Heart J. 2021;42:3541-54.
19. Acikalin A, Satar S, Akpinar O, et al. NT-proBNP levels and QT changes in acute ischemic stroke. Neurol Asia. 2013;18:1-8.
20. Lin JF, Hsu SY, Wu S, et al. QT interval independently predicts mortality and heart failure in patients with ST-elevation myocardial infarction. Int J Med Sci. 2015;12:968-74.
21. Sievi NA, Clarenbach CF, Camen G, et al. High prevalence of altered cardiac repolarization in patients with COPD. BMC Pulm Med. 2014;14:1-8.
22. Van Oekelen O, Vermeersch K, Everaerts S, et al. Significance of prolonged QTc in acute exacerbations of COPD requiring hospitalization. Int J Chron Obstruct Pulmon Dis. 2018;13:1937-47.

Table 1. Demographic characteristics of the study participants [n is frequency; (% is percentage)].

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	Dyspnoea (yrs.)	8.73	9.08
	Cough (yrs)	2.05	4.60
	Sputum production	42	35
Co-morbidities	HTN (yrs)	1.09	2.71
	T2DM (months)	5.97	20.24
	ATT history	22	18.3
	Hypothyroidism	8	6.7
Habits	Smokers/Biomass exp. (yrs)	11.92	11.35
	Addictive substance (Ganja) (yrs.)	3.40	7.03
	Alcohol intake (yrs)	3.23	7.05

HTN, hypertension; T2DM, type 2 diabetes mellitus; ATT, anti-tuberculosis treatment.
*p=<0.05 is significant.

Table 2. Associations between AEMD, 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/ .004*		3.05/ .010*		1.88/ .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/ .040*		6.21/ .002*		3.70/ .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/ .042*		-1.777 / .081		-1.233 / .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/ .002*		-2.63/ .011*		-1.47/ .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		.28/ .35		.26/ .95		1.36/ .04	
NT pro BNP	<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/ .020*		-2.075/ .051*		-1.836/ .082	

AEMD, atrial electromechanical delay; COPD, chronic obstructive pulmonary diseas; NT pro BNP, N-terminal pro brain natriuretic peptide; desat.- desaturation. *p=<0.05 is significant.

Table 3. P wave dispersion and clinical outcomes.

		P wave Dispersion (Mean ± SD)	p-value
Atrial Fibrillation	No	33.1 ± 7.6	**< .00001**
	Yes	41.2 ± 6.5	
Heart failure	No	33.6 ± 7.5	**< .00001**
	Yes	44.6 ± 3.6	
COPD exacerbation	No	29.1 ± 4.5	**< .00001**
	Yes	42.3 ± 5.2	
Hospitalization	No	33.1 ± 8.0	**< .00001**
	Yes	41.6 ± 5.1	
Mortality	Live	35.8 ± 8.3	**< .00001**
	Dead	42.3 ± 3.3	
Significant Desaturation (≥4% O2 fall)	No	35.25 ± 8.3	**< .00001**
	Yes	39.13 ± 7.4	
NT Pro BNP	< 125	31.9 ± 8.3	**< .00001**
	125.-1000	38.3 ± 6.3	
	>1000	44.4 ± 6.5	

p<0.05 is significant; [a]Mann-Whitney U statistical analysis.

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

		QT Dispersion (Mean ± SD)	p-value
Heart failure	No	41.04 ± 6.9	< .00001
	Yes	49.36 ± 8.9	
COPD exacerbation	No	40 ± 6.6	< .00001
	Yes	45.6 ± 8.6	
Hospitalization	No	39.8 ± 6.6	< .00001
	Yes	48.41 ± 7.9	
Mortality	Live	47.2 ± 6.1	< .00001
	Dead	42.28 ± 8.2	

p<0.05 is significant.