

Cardiac Function in Chronic Obstructive Pulmonary Disease: Role of MAPSE

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ABSTRACT

This study aims to investigate the role of mitral annular plane systolic excursion (MAPSE) in evaluating left ventricular function in patients with chronic obstructive pulmonary disease (COPD).

This study included 53 COPD patients and 51 healthy controls. All participants underwent echocardiographic evaluation, including tissue Doppler imaging. Lateral MAPSE was measured via M-mode in the apical four-chamber view.

The mean age of the study population was 63.5 ± 8.8 years, with 69.2% male. No significant differences were observed between the COPD (n=53) and control (n=51) groups regarding age, sex, heart rate, blood pressure, comorbidities, or laboratory findings, though smoking was more prevalent in the COPD group. Pulmonary function tests showed significantly lower FEV₁ and FEV₁/FVC in COPD patients ($p < 0.001$). Conventional echocardiographic parameters including left ventricular dimensions and ejection fraction were similar between groups. However, the COPD group exhibited higher LAVi and e/E' ratios, lower E' velocities, and impaired right ventricular function indicated by reduced TAPSE and Sm-RV and elevated PAPs (all $p < 0.001$). Importantly, MAPSE values were significantly decreased in COPD patients, reflecting impaired longitudinal left ventricular function [11.3 ($9.6-13.8$) vs. 13.7 ($13.4-15.2$), $p < 0.001$].

Reduced MAPSE in COPD patients may indicate early LV systolic dysfunction. MAPSE is a simple and reliable parameter that may aid in the early detection of cardiac involvement in COPD.

Keywords: Chronic obstructive pulmonary disease, left ventricular function, MAPSE, diastolic dysfunction, systolic dysfunction

Introduction

Chronic obstructive pulmonary disease (COPD) is defined by persistent airflow restriction and is accompanied by notable systemic impacts, as well as being linked to various significant comorbid conditions.¹ COPD is linked to significant extra-pulmonary manifestations, with cardiovascular complications being the most common. Cardiovascular diseases are responsible for approximately 50% of all hospitalizations related to the condition.² COPD impacts the pulmonary vasculature and both the right (RV) and left ventricles (LV), contributing to the onset of pulmonary hypertension, cor pulmonale, and dysfunction in both the RV and LV and echocardiography offers a quick, non-invasive, and precise approach to assess both RV and LV functions. The importance of RV performance is well-established as a key factor influencing the clinical progression and prognosis of COPD.³

However, the LV role is less frequently examined, and conventional echocardiographic approaches are generally utilized.⁴

Changes in LV function and structure, including LV enlargement, impaired diastolic function, and decreased ejection fraction (EF), have been observed in patients with COPD.⁵ Several studies have been conducted to evaluate LV function in COPD patients using various echocardiographic parameters. Recent studies have shown that mitral annular plane systolic excursion (MAPSE) is being proposed as a key parameter for assessing LV function. The current clinical use and potential benefits of routinely incorporating MAPSE measurement in patients with different cardiovascular conditions are being explored.⁶ A reduced MAPSE indicates impaired longitudinal function, offering valuable additional information alongside EF, which reflects the overall result of both longitudinal and circumferential contraction. Research has shown

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Received: 06.07.2025, Accepted: 04.09.2025

that MAPSE plays a crucial role in LV pumping, both in healthy individuals and those with cardiovascular disease.⁷ The aim of this study is to investigate the potential of MAPSE as an indicator of cardiac function and to evaluate the role of MAPSE measurements in individuals with COPD.

Materials and Methods

The study, conducted at Çam and Sakura City Hospital, included 53 patients diagnosed with COPD and 51 healthy individuals as the control group. The diagnosis of COPD was established by a pulmonologist in accordance with the Global Initiative for Chronic Obstructive Lung Disease (GOLD) guidelines, based on spirometric evidence of persistent airflow limitation, defined as a post-bronchodilator forced expiratory volume in 1 second (FEV₁) to forced vital capacity (FVC) ratio of less than 0.70.⁸ The diagnosis was further supported by a smoking history of at least 10 pack-years, the presence of chronic respiratory symptoms, and radiographic findings consistent with COPD. Patients classified as GOLD 4, which corresponds to very severe airflow limitation (FEV₁ <30% predicted), were excluded from the study due to their limited mobility and higher risk of clinical instability.

The control group consisted of 51 individuals without a history of chronic lung disease or respiratory symptoms, who were matched to the COPD group by age, sex, and smoking history. These individuals had normal chest radiographs and spirometric values within the normal range, defined as a post-bronchodilator FEV₁/FVC ratio ≥ 0.70 and FEV₁ > 80% of the predicted value.⁹ Exclusion criteria for both groups included acute exacerbation of COPD at the time of evaluation, the presence of systemic diseases known to cause pulmonary hypertension, poor echocardiographic imaging quality, inability to perform spirometry, a history of myocardial infarction, known coronary artery disease, or congestive heart failure. Other exclusion criteria were history of asthma or other chronic lung diseases, active malignancy, dialysis dependence or advanced renal failure, insulin-dependent diabetes mellitus, a resting arterial oxygen pressure (PaO₂) < 60 mmHg, or radiographic abnormalities unrelated to COPD.

Demographic, clinical, spirometric, chest radiographic, electrocardiographic (ECG), and echocardiographic data were collected from all participants. Routine laboratory tests, including complete blood count, lipid profile, fasting blood glucose, blood urea nitrogen, and serum creatinine

levels, were also obtained. Patients were divided into two groups based on the presence or absence of COPD, and their demographic, laboratory, and echocardiographic characteristics were compared. All data were retrieved from the hospital's electronic medical record system. This study was conducted in accordance with the principles of the Declaration of Helsinki and was approved by Basaksehir Cam and Sakura City Hospital Ethics Committee. (Approval number and date: 09-25/09/2024).

Echocardiographic Assessment: Transthoracic echocardiographic examinations were carried out with the patient in the semi-recumbent left lateral position, and standard views were obtained from the parasternal, apical, and subxiphoid windows. LVEF was assessed using the 2D biplane Simpson's method and visual estimation in two-dimensional views. Left ventricular diastolic function was evaluated using both conventional Doppler and tissue Doppler imaging parameters. From pulsed-wave Doppler at the mitral inflow, early (E) and late (A) diastolic flow velocities were measured to calculate the E/A ratio. Tissue Doppler imaging obtained from the apical four-chamber view was used to assess the mitral annular systolic (s') and early diastolic (e') velocities at the septal and lateral sites. The E/e' ratio, derived by dividing the peak E velocity by the average of septal and lateral e' velocities, was used to estimate left ventricular filling pressures.¹⁰ Left atrial volume index (LAVi) was calculated using the biplane area-length method from apical four- and two-chamber views. The left atrial volume was then indexed to body surface area to obtain LAVi (mL/m²). Mitral annular plane systolic excursion (MAPSE) was measured using M-mode echocardiography in the apical four-chamber view. The measurement was obtained from the lateral mitral annulus, and this value was used for analysis. (See, Figure-1)

To evaluate right heart function and pulmonary pressures, tricuspid regurgitation was first assessed using color Doppler imaging, with its peak jet velocity measured by continuous-wave Doppler. RV systolic function was then evaluated through several echocardiographic parameters, including tricuspid annular plane systolic excursion (TAPSE) and the peak systolic velocity (S') of the tricuspid annulus, obtained via pulsed-wave tissue Doppler imaging. Finally, systolic pulmonary artery pressure (sPAP) was estimated by adding right atrial pressure-determined by assessing the inferior vena cava diameter and its inspiratory

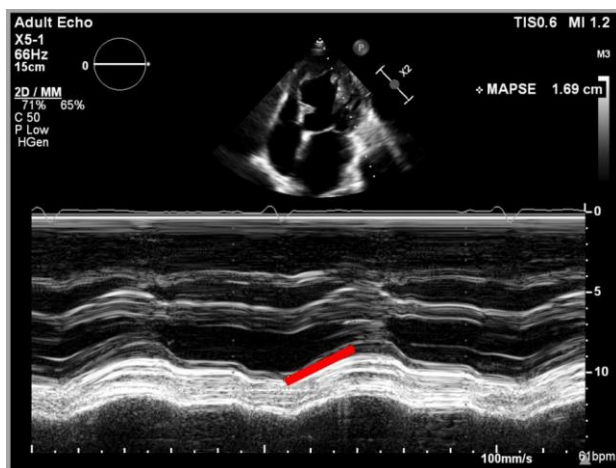


Fig.1. Measurement of MAPSE using M-mode echocardiography in the transthoracic apical four-chamber view

collapse-to the peak pressure gradient across the tricuspid valve.¹¹

Statistical Analysis: Normality of data distribution was initially assessed using the Kolmogorov–Smirnov test and further evaluated through visual methods, including histogram analysis and probability plots. Continuous variables were presented as mean $\pm$ standard deviation for normally distributed data and as median with interquartile range (IQR, 25th–75th percentiles) for non-normally distributed data. Group comparisons of continuous variables were performed using the independent samples t-test or the Mann–Whitney U test, depending on distributional characteristics. Categorical variables were summarized as frequencies and percentages, and group differences were analyzed using either the Pearson chi-square test or Fisher’s exact test, where appropriate. A two-sided p-value < 0.05 was considered statistically significant. No adjustment for multiple comparisons was applied, as the analyses were primarily exploratory and focused on predefined hypotheses. A post hoc power analysis based on the *MAPSE* variable revealed a statistical power exceeding 98%. All statistical analyses were conducted using R software (version 4.1.3; R Foundation for Statistical Computing, Vienna, Austria).

Results

The mean age of the study population was 63.5 ± 8.8 years, and 69.2% were male. There were no statistically significant differences between the COPD group ($n=53$) and the control group ($n=51$) in terms of age, sex, heart rate, blood

pressure, presence of hypertension or diabetes, or laboratory parameters. However, the proportions of both former and current smokers were significantly higher in the COPD group. Pulmonary function test results showed that FEV₁ and FEV₁/FVC values were significantly lower in the COPD group compared to the control group [64 (60–76) % vs. 95 (90–98) % , $p<0.001$ and 61 (56–65) vs. 80 (78–83), $p<0.001$, respectively]. (See, Table-1)

In terms of echocardiographic parameters, there were no significant differences between the COPD and control groups in left ventricular end-systolic and end-diastolic diameters, wall thicknesses, or ejection fraction (EF). However, the LAVi was significantly higher in the COPD group, whereas MAPSE values were significantly lower in this group [31 (29–33) ml/m² vs. 27 (24–29) ml/m², $p<0.001$ and 11.3 (9.6–13.8) mm vs. 13.7 (13.4–15.2)mm , $p<0.001$, respectively].

Although no significant differences were observed in mitral inflow velocities between the groups, E' measured by tissue Doppler was significantly lower [8.2(7.5–8.8) cm/s vs 9.5(9.1–9.9) cm/s $p<0.001$], and the e/E' ratio was significantly higher in the COPD group [8.6(7.7–9.5) vs 7.7(7–8.2) $p<0.001$] (See Table 2). Additionally, TAPSE ($p<0.001$) and Sm-RV($p<0.001$) were lower in the COPD group, while PAPs ($p<0.001$) was significantly higher.

Discussion

In our study, we demonstrated that left ventricular systolic function may be adversely affected in patients with COPD. Notably, MAPSE values were significantly lower in the COPD group, suggesting the presence of subclinical left ventricular systolic dysfunction in this population.

Heart failure affects over 20% of individuals with COPD. Additionally, the likelihood of developing heart failure in COPD patients is 4.5 times greater compared to those without the condition.¹² In the literature, there is limited and inconclusive data regarding the relationship between COPD and LV cardiac function. Several studies propose that LV function stays unaffected in individuals with COPD, while others indicate the possibility of LV dysfunction occurring.^{13,14} Early identification of subclinical LV systolic dysfunction is crucial, as prompt medical intervention can help prevent or delay the onset of heart failure.¹⁵ An important strength of the current study is the focus on MAPSE as a sensitive and practical marker of LV

Table 1: Baseline Characteristics, Risk Factors, and Laboratory Findings of The Study Population

	Group 1(COPD) n:53	Group 2(control) n:51	P value*
Age (years)	63.8±9.3	63.3±8.2	0.773
Gender, male, n (%)	38(71.7)	34(66.7)	0.578
BMI, kg/m ²	24.5(22.8-29)	25.6(23.5-28.1)	0.503
HR (beats/min)	74±10.9	70.3±10.4	0.081
Sys Blood P(mmHg)	135(125-143)	129(126-135)	0.061
Dias Blood P(mmHg)	85(73-89)	84(76-90)	0.216
DM, n (%)	11(20.8)	10(19.6)	0.884
Hypertension, n (%)	17(32.1)	15(29.4)	0.769
Ex Smoking, n (%)	25(47.2)	6(11.8)	0.001
Current smoking, (n, %)	19(35.8)	9(17.6)	0.036
Hyperlipidemia, n (%)	15(28.31)	13(25.5)	0.747
FEV1 (%)	64(60-76)	95(90-98)	0.001
FEV1 / FVC (%)	61(56-65)	80(78-83)	0.001
Hemoglobin, g/dL	13.2(12.1-14.2)	13.2(11.9-14.6)	0.661
Platelet count, 103/μL	272(228.5-315)	245(209-296)	0.057
WBC Count, 103/μL	9.7±3.1	8.6±3.2	0.066
Glucose (mg/dL)	121(112.5-160)	116(99-157)	0.084
Creatinine (mg/dL)	1(0.82-1.1)	0.9(0.74-1.06)	0.062
NT-ProBNP	123(111.5-146)	118(102-146)	0.332

Abbreviations: COPD, Chronic obst. pulmonary disease; BMI, Body mass index; HR, Heart rate, *Sys Blood P*, Systolic blood pressure; *Dias Blood P*, Diastolic blood pressure; DM, Diabetes mellitus; FEV₁, forced expiratory volume in 1 s; FVC, forced vital capacity; WBC, White blood cell; NT-proBNP, N-terminal pro-B-type Natriuretic Peptide;

*Continuous variables were presented as mean ± standard deviation for normally distributed data and as median (interquartile range (IQR₂₅₋₇₅)) for non-normally distributed data. Categorical data were expressed as numbers and percentages. The *p*-value for continuous data was calculated using the Independent Samples t-test or the Mann-Whitney U-test, while for categorical variables, the Chi-Square test or Fisher's exact test was employed, as appropriate. * A *p*-value of <0.05 was considered statistically significant.

systolic function in patients with COPD. While conventional echocardiographic parameters such as EF often remain within normal ranges until advanced stages of dysfunction, MAPSE offers a more specific evaluation of longitudinal myocardial function, which is typically affected earlier in disease progression. This is particularly relevant in COPD, where hyperinflation and suboptimal acoustic windows may hinder accurate EF measurements. MAPSE, requiring only clear visualization of the mitral annulus, is less affected by image quality and can be reliably obtained even in technically challenging cases. In this context, our findings support the use of lateral MAPSE as a feasible and reproducible echocardiographic parameter that can aid in the early identification of subclinical LV dysfunction in COPD patients. Its integration into routine echocardiographic assessment may enhance cardiovascular risk stratification and guide clinical decision-making. In a previous study evaluating the long-term

prognostic value of MAPSE, MAPSE measured from cine magnetic resonance images was shown to be a strong and independent predictor of major adverse cardiovascular events (MACE) over a 9-year follow-up in patients with suspected coronary artery disease¹⁶ Consistent with our findings, a cardiac magnetic resonance-based study involving 255 patients with ST-elevation myocardial infarction demonstrated that MAPSE was an independent prognostic indicator of MACE over a median follow-up of three years. In that study, a MAPSE threshold of 9 mm yielded a predictive accuracy with an area under the curve of 0.74, further underscoring the clinical utility of this parameter in risk stratification.¹⁷ These findings support the growing evidence that MAPSE can serve as a reliable marker of LV systolic function and long-term prognosis, which is in line with the results of our study

Abnormal LV performance in patients with COPD may be due to a number of factors. A key

Table 2: Echocardiographic Parameters of The Study Population

	Group 1(COPD) n:53	Group 2(control) n:51	P value*
LV end-diastolic dimension, (mm)	51(48.3-54.2)	50(48.6-56)	0.532
LV end-systolic dimension, (mm)	26.3±3.3	25.3±2.3	0.066
Septal wall thickness, (mm)	11(10-12)	10(10-12)	0.161
Posterior wall thickness, (mm)	10(9-12)	10(9-11)	0.751
LV ejection fraction, (%)	58(54-63)	63(53-65)	0.175
LAVi (mL/m ²)	31(29-33)	27(24-29)	0.001
e (m/s)	7.1(6.8-7.3)	7.1(6.8-7.5)	0.264
a (m/s)	8.2(7.9-9)	8.2(8.1-91)	0.370
e/a ratio	0.85(0.77-0.94)	0.86(0.70-0.96)	0.969
E' (cm/s)	8.2(7.5-8.8)	9.5(9.1-9.9)	0.001
e/E' ratio	8.6(7.7-9.5)	7.7(7-8.2)	0.001
TAPSE (mm)	19±2	22±2.4	0.001
Sm-RV, (cm/s)	15.8(15.2-16.3)	17.3(16.8-18.3)	0.001
PAPs (mmHg)	31(29-40)	23(22-32)	0.001
MAPSE (mm)	11.3(9.6-13.8)	13.7(13.4-15.2)	0.001

Abbreviations: COPD, Chronic obst. pulmonary disease; LV, Left ventricle; LAVi, Left atrial volume index; e, Peak early mitral flow velocity; a, Peak late mitral flow velocity; E', Lateral mitral annular tissue Doppler E wave; TAPSE, Tricuspid annular plane systolic excursion; Sm-RV, Right ventricular lateral wall systolic myocardial velocity; PAPs, Pulmonary artery systolic pressure; MAPSE: Mitral annular plane systolic excursion

*Continuous variables were presented as mean ± standard deviation for normally distributed data and as median (interquartile range (IQR₂₅₋₇₅)) for non-normally distributed data. The *p*-value for continuous data was calculated using the Independent Samples t-test or the Mann-Whitney U-test. A *p*-value of <.05 was considered statistically significant

factor in the development of LV systolic dysfunction in COPD patients is impaired function of the RV. Elevated RV end-diastolic pressure can lead to a shift of the interventricular septum towards the left, subsequently which would in turn increase LV end-diastolic pressure, decrease venous return, and diminish LV stroke volume and cardiac output and large swings in intrathoracic pressure. Another possible contributor to LV systolic dysfunction in COPD is heightened arterial stiffness, which has been observed in patients with COPD, even those with mild airway obstruction¹⁸. Arterial stiffness has been found to be independently associated with impaired segmental relaxation and abnormal global longitudinal systolic deformation in individuals with a normal EF.¹⁹ Angiotensin converting enzyme is present in high concentrations in the lungs, and chronic hypoxia can activate renin angiotensin system, which has proinflammatory and profibrotic effects. This might have another role in the pathogenesis of LV systolic dysfunction in COPD.²⁰ Studies have demonstrated that hypoxemia is linked to endothelial dysfunction, which may serve as one of the key mechanisms contributing to LV dysfunction.²¹

The findings emphasize the clinical utility of MAPSE as a sensitive marker for detecting subclinical left ventricular systolic dysfunction in patients with chronic obstructive pulmonary disease. Given the limitations of conventional echocardiographic parameters in this population, particularly due to hyperinflation and suboptimal imaging windows, MAPSE provides a reproducible and robust assessment of longitudinal myocardial function. Incorporating MAPSE into routine echocardiographic evaluation may enhance early risk stratification and guide therapeutic decision-making, while further research continues to explore its prognostic value and mechanistic role in cardiac impairment associated with chronic obstructive pulmonary disease.

Limitations: This study has several limitations. Firstly, the relatively small sample size may limit the generalizability of our findings to a broader population and no adjustment for multiple comparisons was applied, which may increase the risk of type I error. Additionally, the assessment of LV diastolic dysfunction using echocardiography is dependent on image quality, which may affect the accuracy of the results. The study was conducted only at rest, and therefore

did not account for the effects of dynamic hyperinflation that occur during physical activity in patients with COPD, which may influence LVDD. Moreover, cardiac magnetic resonance imaging, considered the gold standard for evaluating LV size and function, was not utilized. Nonetheless, well-established echocardiographic structural parameters alongside cardiac biomarkers were employed to comprehensively characterize cardiac dysfunction patterns in this cohort of COPD patients.

In conclusion, this study highlights the high rate of LV diastolic dysfunction and subclinical left ventricular systolic dysfunction in patients with COPD, emphasizing the complex interplay between pulmonary and cardiac pathologies in this population. Importantly, MAPSE proved to be a valuable and sensitive echocardiographic parameter for the early detection of LV systolic dysfunction, especially when conventional measures such as EF remain normal. Given the significant impact of cardiac dysfunction on morbidity and mortality in COPD patients, routine incorporation of MAPSE measurement into echocardiographic evaluations could enhance early diagnosis and risk stratification. Further longitudinal studies are warranted to investigate the prognostic value of MAPSE and to explore targeted interventions aimed at improving cardiac outcomes in COPD.

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