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Impacts of Chronic Obstructive Pulmonary Disease on Left Atrial and Left Ventricular dysfunction in Patients with Acute Myocardial Infarction: Strain Analysis Using Speckle-Tracking Echocardiography

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ABSTRACT

Background: Despite the initial assumption that chronic obstructive pulmonary disease (COPD) solely distresses the lungs and airways, the focus of research has lifted to the great incidence of cardiovascular disease in COPD patients. This study aim was to assess the impacts of COPD on left atrial and left ventricular dysfunction in patients with acute myocardial infarction (AMI) using speckle-tracking echocardiography.

Patients and Methods: This reconsidering study was executed on eighty patients admitted with AMI. Patients were further subdivided into 2 sets: COPD group (40 patients) and non-COPD group (40 patients) regarding the GOLDEN criteria. Strain analysis by arterial blood gases (ABG), speckle-tracking echocardiography, pulmonary function tests (PFTs), and laboratory investigations were undertaken for all patients.

Results: Left ventricular ejection fraction **(**LVEF), left ventricular global longitudinal strain (LVGLS), left atrial strain during conduit stage (LAScd), left atrial strain during reservoir phase (LASr), and left atrial strain during contraction phase (LASct) were significantly increased in non-COPD patients than COPD patients (P value <0.001). LA volume and LAVI were significantly increased in COPD patients than non-COPD patients (P value <0.05). Left ventricular end-diastolic diameter (LVEDD) and left ventricular end-systolic diameter (LVESD) were insignificantly changed among non-COPD and COPD patients.

Conclusions: left atrial and left ventricular diastolic dysfunction are prevalent in COPD patients following an AMI, as indicated by LA strain and LVGLS analyses. Additionally, it is imperative that cardiologists and pulmonologists collaborate more closely to improve the management of this substantial patient population.

Keywords: COPD; AMI; Speckle-Tracking Echocardiography; STEMI; NSTEMI; LVDD

INTRODUCTION

Chronic obstructive pulmonary disease (COPD) is a medical disorder that is defined by reduced lung function, structural pulmonary abnormalities, chronic respiratory symptoms (predominantly progressive ventilation restriction with limited reversibility), or any mixture of these [1]**.** Despite the initial belief that COPD solely influenced the lungs and airways, the focus of research has moved to the elevated incidence of cardiovascular disease in patients with COPD [2]. A cardiovascular etiology is reliable for up to 1 third of the fatalities in COPD patients, highlighting the significant impact of this comorbidity[3] **.**

There is evidence from studies based on population that COPD elevates the likelihood of cardiovascular mortality and morbidity by approximately 2-fold. An additional investigation explained that elderly patients with COPD were at a 4.5-fold improved danger of emergent congestive HF when contrasted with matched by age controls. Although COPD patients have been reported to have left ventricular diastolic dysfunction (LVDD), the correlation among LVDD and COPD or HF with preserved ejection fraction (HFpEF) is not well known [4]. However, the diagnosis of COPD was occasionally limited to patients who were on COPD-related pharmacotherapy or solely related to medical records. Furthermore, pulmonary function tests (PFTs) were not conducted on a regular basis to quantify and diagnose COPD severity [5].

This is likely to be a significant issue, as about 80 percent of individuals with COPD are not diagnosed [6], and twenty–fifty percent of these with voluntarily disclosed COPD do not meet criteria for spirometric disease [7]. Recent data has demonstrated that five–eleven percent of patients with acute coronary syndrome (ACS) have comorbid COPD when spirometric assessment is employed for the diagnosis of COPD [8].

Echocardiography allows initial assessment of left and right ventricular function and size, like surrogates of cardiac damage in the acute phase. Emerging advanced echocardiographic techniques as speckle-tracking strain imaging enable characterization of myocardial mechanics (mechanical and strain stimulation time dispersion), which have been related to rigid termination points, like cardiac and all-cause mortality, ventricular arrhythmias, and HF hospitalization [9, 10].

This investigation aim was to assess the special properties of COPD on left atrial and left ventricular disfunction in patients with AMI by employing speckle-tracking echocardiography.

PATIENTS AND METHODS

This retrospective investigation was conducted on eighty patients who were admitted with AMI and with an adequate image quality had a post-AMI transthoracic echocardiography that was either recent or current (not more than 6 months before to the AMI). The study was

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carried out at El Hussien University Hospital, Islamic Cardiac Center during the period from November 2023 to March 2025.

Ethical considerations:

A notified consent in writing was gotten from the patient or their relatives. The research was conducted with the sanction of the Ethical Committee of the University Hospitals. The Helsinki Declaration was followed throughout the study's conduct.

Standards for exclusion included patients whose echocardiograms were unavailable due to an AMI or because the quality of the echocardiographic image.

Demographic data, coexisting medical conditions, disease history, laboratory investigations, and smoking were recovered for all patients.

Echocardiography was conducted by primary examiners using commercially presented ultrasound systems. Regarding the guidelines of the ASE (American Society of Echocardiography) and EACVI (European Association of Cardiovascular Imaging), primary investigators obtained standardized echocardiographic measurements. LVEF was determined in both 4 chamber and 2 chamber views utilizing Simpson's method of discs.

Testers for arterial blood gases (ABGs) examinations were collected from the arterialized earlobes of all patients while they were inhaling room air without supplemental oxygen.

Patients were later subdivided into 2 groups: COPD group (forty patients) and non-COPD group (40 patients) according to the GOLD criteria [11].

When the inspector determined that the image value was satisfactory with acceptable tissue trailing, strain analysis was conducted operating speckle-tracking echocardiography. The ASE and EACVI recommended the acquisition of standard 2D grayscale images of the LV from conventional apical four-, two-, and three-chamber views in order to measure LV-GLS.

The "EACVI/ASE/Industry Task Force" recommended the measurement of LAS values in apical four and two chamber views. The LAS values were stated distinctly for the 3 stages of the LA cycle: conduit, contraction, and reservoir stage:

- **Strain during reservoir stage (LASr):** the strain value from the ventricular end-diastole to the mitral valve opening at ventricular end-systole (+ve value).
- **Strain during conduit stage (LAScd)**: the strain value from the mitral valve opening to the onset of atrial contraction (-ve value). In patients with atrial fibrillation, LAScd has the similar value as LASr, but with a -ve sign.
- **Strain during contraction stage (LASct)**: calculated only in patients in sinus rhythm as the strain value from the onset of atrial contraction to ventricular end-diastole (-ve value).

Sample Size Calculation:

Sample size was measured operating G-power software version 3.1.9.4, depending on prior data from **Grebe *et al.*** [12] for comparison of LVGLS in COPD and non-COPD patients (mean ± SD -15 ± 4 % for COPD and -18 ± 4 % for non-COPD). Using t-test at power 90% and 0.05 alpha error, the whole number of patients to be joined in the study was 80 patients (forty patients in every group).

Statistical analysis

Statistical analysis was done by SPSS v26 (USA, IBM Inc, Armonk NY,). Histograms and Shapiro-Wilks test were managed to evaluate the regularity of the spreading of data. The unpaired student t-test was employed to analyze the quantitative parametric data, which were depicted as mean and standard deviation (SD). The Mann Whitney-test was devoted to analyze quantitative non-parametric data, which were presented as the median and interquartile range (IQR). Qualitative data were evaluated utilizing Chi-square test or Fisher's exact test when applicable, and were presented as percentage and frequency. Statistical significance was definite as a two-tailed P value ≤ 0.05.

RESULTS

Age was significantly decreased in non-COPD patients than COPD patients, while sex and BMI were insignificantly different between non-COPD and COPD patients. In COPD patients, 24 (60%) patients had STEMI and 16 (40%) patients had NSTEMI while in non-COPD patients, 19 (47.5%) patients had STEMI and 21 (52.5%) patients had NSTEMI with no significant variation among the two groups. Comorbidities as smoking, DM, HTN, bronchial asthma, and AF were insignificantly different among non-COPD and COPD patients. Laboratory investigations as creatinine, GFR, NT-proBNP, CRP, CK, and cTnT were insignificantly different among non-COPD and COPD patients **(Table 1)**.

Table 1: Patients' characteristics, comorbidities, and laboratory data

		COPD (n=40)	Non-COPD (n=40)	P value
Age (years)		66.85 ± 13.22	60.23 ± 14.77	0.038*
Sex	**Male**	27 (67.5%)	30 (75%)	0.459
	Female	13 (32.5%)	10 (25%)	
BMI (kg/m^2)		27.32 ± 3.46	26.48 ± 3.22	0.267
AMI	**STEMI**	24 (60%)	19 (47.5%)	0.262
	NSTEMI	16 (40%)	21 (52.5%)	
Smoking		22 (55%)	17 (42.5%)	0.263
DM		11 (27.5%)	9 (22.5%)	0.606
HTN		29 (72.5%)	31 (77.5%)	0.606
Bronchial asthma		4 (10%)	1 (2.5%)	0.359
AF		7 (17.5%)	4 (10%)	0.518
Creatinine (mg/dl)		1.1 (0.68 – 1.3)	1.05 (0.7 – 1.2)	0.497
GFR (ml/min/1.73m^2)		73.5 (55.75 - 89)	78 (53.75 - 106.5)	0.371
NT-proBNP (pg/mL)		1055.5 (655.25 - 1328.75)	997.5 (470.5 - 1164.75)	0.204
CRP (mg/dl)		5.5 (1 - 18)	7.5 (1.75 - 27.25)	0.184
CK (U/L)		381 (206.25 - 878.75)	480.5 (266.75 - 801.25)	0.456
cTnT (pg/ml)		339 (299.75 - 1071)	488.5 (400 - 706)	0.317

Data are shown as mean ± SD, median (IQR), or frequency (%). COPD: chronic obstructive pulmonary disease, BMI: Body mass index, AMI: acute myocardial infarction, STEMI: ST-elevation myocardial infarction, NSTEMI: non-ST elevation myocardial infarction, DM: diabetes mellitus, HTN: hypertension, AF: atrial fibrillation, GFR: glomerular filtration rate, NT-proBNP: N-terminal pro B-type natriuretic peptide, CRP: C-reactive protein, CK: creatine kinase, cTnT: cardiac troponin T, *: significant as P value ≤ 0.05.

FEV1, FEV1/FVC, and DL_{CO}/VA were significantly decreased in COPD patients than non-COPD patients. RV, TLC, and RV/TLC were significantly decreased in non-COPD patients than COPD patients **(Table 2)**.

Table 2: PFTs of the studied groups

	COPD (n=40)	Non-COPD (n=40)	P value
FEV1 (%)	65.5 ± 10.62	88.08 ± 6.62	<0.001*
FEV1/FVC (%)	58.75 ± 10.93	78.7 ± 5.69	<0.001*
RV (%)	165 (120.5 – 215.25)	120 (89.5 – 143.75)	<0.001*
TLC (%)	101.5 (86.5 – 133.25)	93.5 (78 – 108.5)	0.048*
RV/TLC (%)	147.69 (106.51 - 204.88)	135.05 (98.46 - 157.92)	0.049*
DL_{CO}/VA	58.5 (36.75 - 81.25)	70 (61 - 80.75)	0.019*

Data are shown as mean ± SD or median (IQR). PFTs: Pulmonary function tests, COPD: chronic obstructive pulmonary disease, FEV1: forced expiratory volume in 1 second, RV: residual volume, FVC: forced vital capacity, TLC: total lung capacity for carbon monoxide/alveolar volume, DLco/VA: diffusing capacity, *: significant as P value ≤ 0.05

PaO_2 and SpO_2 were significantly decreased in COPD patients than non-COPD. $PaCO_2$ was significantly increased in COPD patients than non-COPD patients. pH and HCO_3 were insignificantly changed among non-COPD and COPD patients **(Table 3)**.

Table 3: ABGs of the studied groups

	COPD (n=40)	Non-COPD (n=40)	P value
pH	7.45 ± 0.07	7.42 ± 0.08	0.079
$PaCO_2$ (mmHg)	38.6 ± 6.18	34.83 ± 6.83	0.011*
PaO_2 (mmHg)	60.03 ± 8.3	65.15 ± 11.31	0.023*
HCO_3 (mmHg)	23.65 ± 2.93	22.73 ± 2.66	0.143
SpO_2 (%)	92.38 ± 1.82	94.43 ± 1.74	<0.001*

Data are shown as mean ± SD. ABGs: arterial blood gases, COPD: chronic obstructive pulmonary disease, PaCO2: partial pressure of carbon dioxide; PaO_2: partial pressure of oxygen, HCO_3: bicarbonate, SpO_2: oxygen saturation, *: significant as P value ≤ 0.05

LVEF, LVGLS, LASr, LAScd, and LASct were significantly decreased in COPD patients than non-COPD patients. LA volume and LAVI were significantly increased in COPD patients than non-COPD patients. LVEDD and LVESD were insignificantly different among non-COPD and COPD patients **(Table 4)**.

Table 4: Echocardiography findings of the studied groups

	COPD (n=40)	Non-COPD (n=40)	P value
LVEF (%)	49.73 ± 10.29	54.65 ± 9.26	0.027*
LVEDD (mm)	46.28 ± 9.34	48.48 ± 6.35	0.222
LVESD (mm)	33.5 (22.75 - 48.5)	32 (27 - 38.25)	0.560
LA volume (ml)	68 (39.5 - 88)	50 (32.75 - 62.25)	0.005*
LAVI (ml/m^2)	41 (25.5 - 54)	27 (12.75 - 39)	0.004*
LVGLS (%)	-14 (-19 - -11.75)	-19 (-22 - -15)	0.003*
LASr (%)	26 (8.75 - 34.5)	34.5 (27.75 - 40.25)	0.001*
LAScd (%)	-12.5 (-17.25 - -4)	-20 (-25 - -13)	<0.001*
LASct (%)	-13 (-18.25 - -4.75)	-15 (-15 - -14)	0.047*

Data are shown as mean ± SD. COPD: chronic obstructive pulmonary disease, LVEF: left ventricular ejection fraction, LVEDD: left ventricular end-diastolic diameter, LVESD: left ventricular end-systolic diameter, LA: left atrial, LAVI: left atrial volume index, LVGLS: left ventricular global longitudinal strain, LASr: left atrial strain during reservoir phase, LAScd: left atrial strain during conduit stage, LASct: left atrial strain during contraction phase, *: significant as P value ≤ 0.05.

DISCUSSION

CVD and COPD are frequently co-occurring conditions. In contrast to individuals without COPD, patients with COPD have a two–five-fold improved threat of ischemic heart disease (IHD), peripheral vascular disease, HF, or arrhythmias. Concomitant HF and COPD are related to a worse health status, increased hazard of hospitalization, and a bad prognosis [13].

The aim of this study was to assess the impacts of COPD on left ventricular and left atrial dysfunction in patients with AMI using speckle-tracking echocardiography.

Age was significantly decreased in non-COPD patients than COPD patients while sex and BMI were insignificantly altered among non-COPD and COPD patients. Comorbidities as smoking, DM, HTN, bronchial asthma, and AF were insignificantly different between COPD and non-COPD patients.

Huang *et al.* [4] demonstrated that the mean age of COPD group patients was 80.74 ± 6.75 years and patients with COPD were older than the control group and most of COPD cases were males (80%) as our study showed 67.5% of COPD patients were males. Also, they found that smokers, diabetic and cerebrovascular diseases patients were significantly greater in COPD patients than control with no significant difference was noticed among the two groups as regards chronic kidney diseases, CVD, HTN, and BMI.

In comparison to non-COPD patients, history of coronary artery disease and HF and to be smokers were more frequent in COPD patients, according to **Goedemans *et al.*** [14]**.** However, there was no significant variation in terms of age, HTN, sex, BMI, and DM. Their objective was to assess patients with and without AF who had COPD.

In COPD patients, 24 (60%) patients had STEMI and 16 (40%) patients had NSTEMI while in non-COPD patients, 19 (47.5%) patients had STEMI and 21 (52.5%) patients had NSTEMI with no significant variation among the 2 groups.

Rothnie *et al.* [15] examined the possibility that the mortality space in patients with COPD may be recognized to a disparity in the management and recognition of a MI at the population level. and found that 37.89% and 62.11% of patients with COPD had STEMI and NSTEMI respectively while 46.66% and 53.34% of patients without COPD had STEMI and NSTEMI respectively.

Laboratory investigations as creatinine, GFR, NT-proBNP, CRP, CK, and cTnT were insignificantly different among non-COPD and COPD patients.

Huang *et al.* [4] found TnT and serum creatinine were insignificantly different between COPD and control groups while NT-proBNP was significantly lower in patients without COPD than those with COPD.

There was a substantial decrease in FEV1, FEV1/FVC, and DLCO/VA among COPD patients in comparison to non-COPD patients. Conversely to non-COPD patients, COPD patients exhibited a significant increase in RV, TLC, and RV/TLC.

However, the utilization of a fixed FEV1/FVC ratio as a diagnostic cut-off is a rather simplistic approach that results in the overdiagnosis of COPD in old patients and the simultaneous underestimation of the existence of COPD in younger patients [16]. The prognostic influence of COPD in patients with IHD cured by percutaneous coronary intervention (PCI) was assessed by **Almagro *et al.*** [17]. COPD was identified as a post-bronchodilator forced expiratory volume in the 1st second (FEV1)/forced vital capacity (FVC) ratio <0.70. Their research confirmed a statistically significant inverse correlation among the number of stratified events (combined mortality/major

cardiovascular event) and the percent predicted FVC, FEV1, and FEV1/FVC.

PaO_2 and SpO_2 were significantly increased in non-COPD patients than COPD patients. $PaCO_2$ was significantly decreased in non-COPD patients than COPD patients. pH and HCO_3 were insignificantly changed among non-COPD and COPD patients.

Most of COPD patients were not obtaining specific therapy, and this group had a much greater necrosis size than AMI patients without COPD, according to **Dreher *et al.*** [18]. The outcomes of the PFTs and ABGs were significantly altered among the non-COPD and COPD groups.

LVDD may be associated with or asymptomatic HF symptoms (e.g., HFpEF). The most prevalent risk factors of HFpEF are age, AF, HTN, female sex, and DM. The frequency of HFpEF improves with age [19].

In addition, the presence of cor pulmonale as a result of pulmonary hypertension can result in the interventricular septum deviating for the left ventricle, which could potentially disrupt the structure of the LV and slow down filling compliance [20]. This mechanism could elucidate the correlation among COPD severity and deteriorated diastolic function.

Our study showed that LVEF, LVGLS, LAScd, LASct, and LASr were significantly increased in non-COPD patients than COPD patients. LA volume and LAVI were significantly increased in COPD patients than non-COPD patients. LVEDD and LVESD were insignificantly changed among non-COPD and COPD patients.

These findings are supported with earlier studies [12, 21, 22] which demonstrated that COPD patients had more diminished LV function.

Atypical pathophysiological alterations in LV afterload and/or preload may be the effect of LVDD in patients with COPD. Emphysema-induced hyperinflation of the thoracic cavity may impede the preload of the LV and subsequently influence its diastolic function [23].

In the interim, a correlation among emphysema and LV diastolic filling has been studied in patients with modest flow obstruction (without hyperinflation) [22]. This implies that the pathogenic sources of decreased LV filling are probable to be multifaceted. LV afterload may be increased by the prior resume of reflected arterial wave, while subendocardial ischemia may result from the concurrent reduction in aortic diastolic blood pressure. These causes can combine to influence myocardial relaxation [24].

Also, **Goedemans *et al.*** [25] implied that the dimensions of the left ventricle were significantly decreased in COPD patients. The LVEF was comparable in patients without and with COPD. Nevertheless, patients with COPD exhibited a substantially greater impairment of LVGLS than those without COPD. This suggests that the systolic LV function was significantly reduced, and the infarct area was larger in the COPD group, despite the fact that the infarct size was similar, as indicated by enzyme release.

Both regional and global longitudinal strain have displayed to be precise in assessing global LV function and the existence of segments with transmural necrosis spending as reference cardiac magnetic resonance [26]. Furthermore, in single-photon emission computed tomography myocardial perfusion imaging has been employed like a reference to demonstrate that LVGLS is more accurate than LVEF in guessing the necrosis size at the 30-day follow-up [27].

Limitations: A single center study with fairly small sample size and the study didn't include follow up data or a control group due to ethical issues. Future studies with comparison to control cases are recommended to analyze follow up data and outcomes after AMI in COPD patients as in-hospital stay and mortality and the risk factors that affect them.

CONCLUSIONS

Left atrial and LVDD were highly prevalent in COPD patients following an AMI, as evidenced by LVGLS and LA strain analyses. These abnormalities were not explicable by reduced lung volumes and may be attributable to alternative pathophysiologic mechanisms of the systemic disease COPD.

In order to optimize the management of this substantial patient population, it is imperative that pulmonologists and cardiologists collaborate more closely.

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