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Assessment of the right ventricular strain, left ventricular strain and left atrial strain using speckle tracking echocardiography in patients with chronic obstructive pulmonary disease

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ABSTRACT

Objectives Cardiovascular disease is a prevalent comorbidity and leading cause of mortality in chronic obstructive pulmonary disease (COPD) patients. Early identification of cardiac abnormalities in COPD patients is crucial. Speckle tracking echocardiography (STE) is practical for assessing ventricular and atrial function, but its role in COPD patients is under-researched. This study aimed to examine right ventricular (RV), left ventricular (LV) and left atrial (LA) strain in COPD patients via STE.

Methods A cross-sectional study was conducted with two groups: COPD patients diagnosed per the 2017 Global Initiative for Chronic Obstructive Lung Disease criteria and healthy controls. All the participants underwent STE to evaluate the RV, LV, and LA strains.

Results RV strain indices (RV free wall longitudinal strain (RVFWSL) and RV 4-chamber longitudinal strain (RV4CSL)) were significantly lower in the COPD group (16.53±5.89% and 14.65±4.53%, respectively) than in the control group (21.39±7.78% and 18.34±6.38%, respectively) (p<0.001). LV global longitudinal strain was also lower in the COPD group (18.45% (17.16–19.51)) than in the control group (19.50% (18.63–21.46), p=0.018). No significant differences were found in LA strain indices (LA reservoir strain, LA conduit strain or LA contractile strain) between the two groups. Furthermore, RVFWSL and RV4CSL were significantly greater in the group with a modified Medical Research Council score <2 (p<0.05).

Conclusion Compared with healthy controls, COPD patients presented reduced RV and LV strain, with no significant differences in LA strain indices.

WHAT IS ALREADY KNOWN ON THIS TOPIC

⇒ Cardiovascular function is altered in patients with chronic obstructive pulmonary disease (COPD).

⇒ Several individual studies on left ventricular (LV) and right ventricular (RV) function have revealed changes in these functions in COPD patients.

WHAT THIS STUDY ADDS

⇒ Speckle tracking echocardiography (STE) identifies reduced RV and LV strain in COPD patients prior to left ventricular ejection fraction alterations, correlates RV strain reduction with COPD severity (mMRC scores), and indicates preserved left atrial strain in the absence of concurrent cardiovascular comorbidities.

HOW THIS STUDY MIGHT AFFECT RESEARCH, PRACTICE OR POLICY

⇒ This study highlights the potential application of STE in assessing cardiac function in COPD patients.

INTRODUCTION

Chronic obstructive pulmonary disease (COPD) is a heterogeneous lung condition characterised by chronic respiratory symptoms (dyspnoea, cough, sputum production and/or exacerbations) due to abnormalities of the airways (bronchitis, bronchiolitis) and/or alveoli (emphysema) that cause persistent, often progressive, airflow obstruction.[1] COPD is one of the leading causes of morbidity in individuals over 40 years of age and is predicted to become the fourth leading cause of premature death by 2040.[2] One of the most common comorbidities of COPD is cardiovascular disease, which is a leading cause of death worldwide, affecting approximately 85 million people in Europe.[1 3] Therefore, early detection of cardiac disorders in COPD patients has become an increasingly important issue not only in Vietnam but also worldwide.

In patients with COPD, the deterioration of right ventricular (RV) function and pulmonary vasculature is recognised as a complicating factor in the disease's clinical progression and is inversely correlated with survival. Additionally, alterations in left ventricular (LV) function and structure, such as LV enlargement, diastolic dysfunction and reduced ejection fraction, have been documented in COPD patients.[4] Some studies have also explored the impact of COPD on left atrial (LA) function, noting an increased risk of developing atrial fibrillation. Long-term respiratory therapies with inhaled corticosteroids (ICSs), ICSs combined with long-acting beta-agonists (LABAs+ICSs), and especially LABAs combined with long-acting muscarinic antagonists (LABAs+LAMAs) have been shown to affect the LA diameter in COPD patients.[5 6] Therefore, the early recognition of cardiac dysfunction in COPD patients may identify those at high risk for developing heart failure in the future, thereby improving long-term outcomes.

Several techniques are available for assessing ventricular function, including cardiac positron emission tomography, cardiac CT and cardiac MRI.[7] Nevertheless, these methods encounter challenges because of their cost, restricted accessibility and widespread implementation issues. In comparison, echocardiography is a favourable alternative, providing affordability, high safety, excellent temporal resolution and the ability for repeated examinations.[8 9] Recently, speckle tracking echocardiography (STE) has become a novel modality for assessing cardiac motion and deformation, also known as myocardial strain or myocardial deformation, independent of the angle of the ultrasound beam. It enables the measurement of myocardial tissue velocity during both systole and diastole, providing clinicians with a dynamic perspective on myocardial contractility, particularly in structurally complex regions such as the RV.[10] Compared with conventional echocardiography, STE has demonstrated superior discriminative ability in evaluating the function of both ventricles as a whole or regionally. However, research on the role of STE in COPD patients is limited. Therefore, we investigated the effects of STE on RV, LV and LA strains in patients with COPD. By analysing these parameters, this study aimed to gain valuable insights into how COPD affects cardiovascular health, particularly cardiac function. This research contributes to the early detection and reduction of disease burdens globally.

METHODS

Study design

We conducted a cross-sectional study with a comparison group, integrating elements from the Strengthening the Reporting of Observational Studies in Epidemiology statement to strengthen the reporting of observational research.[11]

Patient and public involvement

Patients and/or the public were not involved in the design, or conduct, or reporting, or dissemination plans of this research.

Study population

From 15 April 2020 to 01 June 2023, this study included 782 patients over 40 years of age admitted to the General Internal Medicine Department with an initial diagnosis of COPD. Additionally, 112 individuals who attended annual health check-ups at the Internal Medicine Clinic of Hospital of University of Medicine and Pharmacy, Hue University were included. All participants provided informed consent prior to their involvement. After the exclusion process, 69 individuals were included in the study analysis (the flowchart of participant recruitment in the study is shown in figure 1).

We categorised the study population into the COPD and control groups. The former group included patients with a history of diagnosed COPD or those without a prior diagnosis but admitted with suspected COPD. These individuals were evaluated on the basis of specific criteria, including progressive dyspnoea that worsens with exercise and persists over time; chronic cough that may be intermittent or dry; chronic sputum production indicative of COPD; recurrent lower respiratory tract infections; a history of exposure to risk factors (eg, tobacco smoke, including passive exposure; occupational dust, vapours, fumes, gases and other chemicals); and a family history of COPD or childhood conditions such as low birth weight or early respiratory infections.[12] All these patients underwent spirometry to confirm the diagnosis of COPD.

In this study, spirometry was conducted via a KoKo Sx 1000 Spirometer (UK). Lung function measurements were taken before and 15 min after the administration of 200 µg of salbutamol via a metered-dose inhaler with a spacer. The participants were instructed to refrain from using short-acting β2-agonists for at least 6 hours, long-acting β2-agonists for 12 hours and long-acting anti-muscarinic agents for 24 hours prior to spirometry.[13] According to the GOLD 2017 criteria, a definitive COPD diagnosis was established when the postbronchodilator forced expiratory volume in first second (FEV1)/forced vital capacity (FVC) ratio was less than 70%. Typically, COPD patients demonstrate an increase in FEV1 of less than 12% and less than 200 mL following bronchodilator response testing.[12]

The exclusion criteria included patients with respiratory function test contraindications such as haemoptysis, pneumothorax, recent myocardial infarction, pulmonary embolism, thoracic surgery within the past 3 months, deafness or visual impairment. Patients with a history of hypertension, diabetes, arrhythmias, valvular heart disease, congenital heart disease, myocardial ischaemia, pericardial effusion or reduced ejection fraction were also excluded. Additionally, those experiencing an acute exacerbation of COPD were not included in the study.

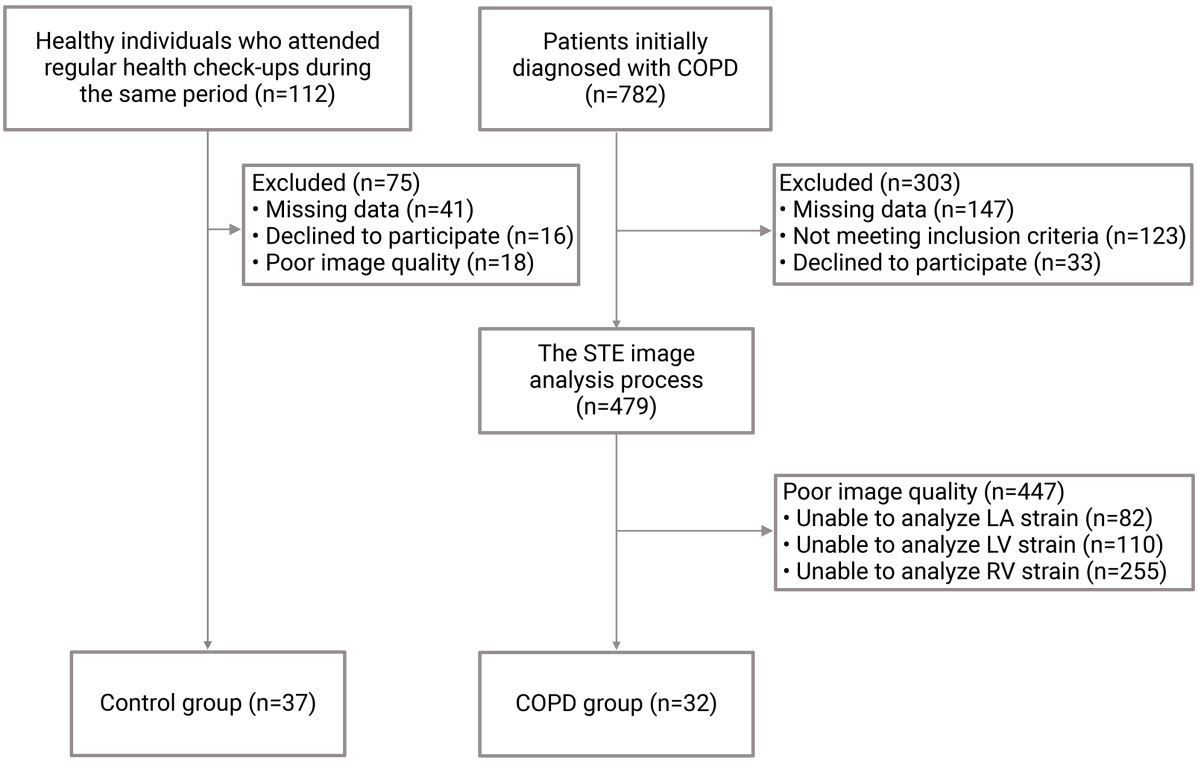


Figure 1 Flowchart of the selection process according to the inclusion and exclusion criteria. COPD, chronic obstructive pulmonary disease; LA, left atrial; LV, left ventricular; RV, right ventricular; STE, speckle tracking echocardiography.

The control group consisted of healthy, non-smoking individuals over 40 years of age who attended regular health check-ups, were free from COPD, and met the exclusion criteria. Echocardiography images of study participants who did not meet the quality standards necessary for recognition and analysis of the research parameters were excluded from both groups.

Clinical data collection

The collected clinical data included demographic information such as age, sex, the number of COPD exacerbations requiring hospitalisation within 1 year, smoking habits (packs-year) and the assessment of dyspnoea via the modified Medical Research Council (mMRC) scale. Clinical examinations included measurements of height, weight, pulse rate, blood pressure, body surface area (BSA) and body mass index (BMI).

The mMRC scale is used to assess the severity of dyspnoea experienced by patients during daily activities, with scores ranging from 0 to 4. A score of 0 indicates breathlessness only during strenuous exertion. A score of 1 reflects shortness of breath when walking quickly on level ground or going up a slight incline. A score of 2 signifies the need to walk slower than peers of the same age or to stop breathing when walking at a normal pace on level ground. A score of 3 indicates that the individual must stop to catch their breath after approximately 100 m or a few minutes of walking on level ground. Finally, a score of 4 represents severe breathlessness, where the person is too short of breath to leave the house or becomes breathless when dressing or undressing.[14] An mMRC grade of 2 or higher indicates a significant level of symptoms.[15]

Echocardiography acquisition

This study used the Philips Affiniti 70 ultrasound system equipped with an S5-1 sector array cardiac transducer, which included integrated ECG recording synchronised with echocardiographic imaging. An experienced echocardiography specialist (with over 5 years of practice) conducted the echocardiographic assessments.

The echocardiographic procedure included the following protocol: patients rested for a minimum of 5 min before the examination. Three ECG electrodes were attached to the chest and calibrated to synchronise with the ultrasound image recording. The patient was positioned in a 90° left lateral decubitus position for parasternal views and a 30°–40° tilt for apical views, with the arms elevated to widen the intercostal spaces. The examiner sat to the right of the patient, held the transducer with the right hand, applied ultrasound gel and initiated the assessment. Image acquisition and measurement followed the British Society of Echocardiography guidelines.[16] Each echocardiogram captured at least three cardiac cycles per view, recorded them during quiet breathing or breath-holding as per the operator's discretion and stored them in DICOM format for subsequent analysis.

Speckle-tracking echocardiography

We routinely imaged patients in the apical four-chamber, two-chamber, and three-chamber views during the

ultrasound examination. Each image was captured over two consecutive cardiac cycles and stored on a USB drive. Speckle tracking analysis was performed by one strain specialist in the core laboratory who was blinded to the clinical data via commercial cardiovascular ultrasound quantification software (QLAB 15, Philips Healthcare Systems).

To assess LV strain, we defined a cardiac cycle from the R wave to the R wave. We selected end-diastolic and end-systolic frames on the basis of the closure of the mitral and aortic valves, respectively. We positioned two reference points at the mitral annulus and one at the apex. We conducted automated border tracing with manual adjustments if necessary. LV global longitudinal strain (GLS) was calculated via three apical chamber views, and we excluded individuals with poor image quality or inadequate border tracking.[9]

LA strain assessment was conducted in both the two-chamber and four-chamber views, with reference points set at the onset of the P wave in the cardiac cycle. Measurements of LA strain were acquired during the reservoir, conduit and contractile phases of LA function, designated LA reservoir strain (LASr), LA conduit strain (LAScd) and LA contractile strain (LASct), respectively.[8 9]

To assess RV myocardial function with 2D-STE, the RV basal septal, basal lateral and apical borders were manually traced in the four-chamber view, followed by automatic tracing of the endocardial and epicardial borders, thus delineating a region of interest composed of six segments. After analysis of the segmental tracking quality and manual adjustment of the region of interest, the software generated longitudinal strain curves for each atrial segment. A cine loop preview feature allowed visual confirmation that the internal line followed the RV endocardium movements throughout the cardiac cycle. If tracking of the RV endocardium was unsatisfactory, manual adjustments of the region of interest size were performed to ensure optimal tracking. Afterwards, a detailed analysis of the RV free wall longitudinal strain (RVFWSL, 3 segments of the RV free wall) and RV four-chamber longitudinal strain (RV4CSL, 6 segments of both the RV free wall and IVS) was conducted.[17 18]

Traditionally, the LA, LV and RV strain results are negative, but we used absolute values for ease of analysis. The methodology is detailed in online supplemental figure 1.

Statistical analysis

We conducted the statistical analyses via SPSS V.26 (IBM, New York, USA) and GraphPad Prism V.10 (GraphPad Software, Boston, USA).

Data normality was assessed via the Kolmogorov-Smirnov test. We present normally distributed continuous variables as the means±SD, whereas non-normally distributed variables are presented as median values with IQRs. Categorical variables are reported as frequencies and percentages. Fisher's exact test was used to evaluate intergroup differences in categorical variables. For continuous variables, we assessed differences via either the unpaired t-test or the Mann-Whitney U test, as appropriate. Missing data were excluded from the analyses.

To determine the correlation between continuous variables, we applied Spearman's correlation coefficient to non-normally distributed variables and Pearson's correlation coefficient to normally distributed variables.

We used univariate logistic regression to compute ORs for predicting COPD in the study population. In our multiple logistic regression analysis, we included all variables with $p \leq 0.05$ to explore the relevance of COPD in the study population.

We randomly selected 30 of the 69 study participants to evaluate the intraclass correlation coefficient (ICC). The ICC and coefficient of variation were used to assess the intraobserver and interobserver variability of RVFWSL, RV4CSL, LASr, LAScd, LASct and GLS. For intraobserver variability, the same operator independently remeasured the data after 7 days. For interobserver variability, the data were reanalysed by a second operator blinded to the initial measurements.

All the statistical tests were two-sided, with a significance level set at <0.05. We followed the SAMPL guidelines in our statistical analysis to prevent avoidable errors or omissions in reporting statistical data.[19]

RESULTS

Our study, conducted on 69 individuals, including COPD patients and healthy controls, employed STE to evaluate RV, LV and LA strains. Online supplemental figure 2 provides an overview of the study design and highlights the comparative analysis of these cardiac parameters.

Baseline clinical characteristics

Our results revealed no statistically significant differences in age, sex, BMI or BSA between the two study groups ($p>0.05$). The average age of the COPD patients was 68±10 years. The average cigarette smoking frequency in the disease group was 36.47±12.61 packs/year, with a duration of COPD of 2.00 (0.50–20.00) years. The median mMRC score was 1.50 (1.00–2.00). Additional detailed information is presented in table 1 and online supplemental table 1.

Comparison of LA, LV and RV strains between the COPD and control groups

Our study revealed that the LV and RV strain indices, including RVFWSL, RV4CSL and GLS, were significantly lower in the patient group than in the control group. Additionally, the patient group had lower LA strain indices, including LASr, LAScd and LASct. However, these differences were not statistically significant ($p>0.05$). Additionally, RVFWSL and RV4CSL were significantly greater in the group with mMRC<2 ($p<0.05$). The details are presented in table 1 (online supplemental figures 3 and 44).

Table 1 General characteristics and echocardiography of the study population

Variables	COPD group (n=32)	Control group (n=37)	P value
Male	29 (91)	30 (81)	0.320
Age (years)	68±10	65±12	0.310
BMI (kg/m^2)	20.32±3.21	21.30±2.52	0.163
BSA (m^2)	1.52±0.15	1.56±0.15	0.263
SBP (mmHg)	129.3±5.4	128.1±13.9	0.613
DBP (mmHg)	77.8±6.6	74.8±8.0	0.093
Heart rate (beats/min)	96.5±16.7	80.4±6.0	<0.001
Smoking (pack-year)	36.47±12.61	NA	NA
Duration of COPD (years)	2.00 (0.50–20.00)	NA	NA
mMRC	1.50 (1.00–2.00)	NA	NA
FEV1 postbronchodilator (mL)	47.50 (36.50–73.50)	NA	NA
FVC (mL)	84.78±27.61	NA	NA
FEV1/FVC (%)	53.19±9.91	NA	NA
PAPS (mmHg)	27.0 (11.0–32.8)	18.0 (14.5–21.5)	0.059
TAPSE (mm)	18.97±2.24	20.20±2.94	0.054
LVMI (g/m^2)	86.5±22.5	84.1±15.6	0.596
LVEF (%)	67.78±5.87	69.15±6.81	0.380
RVFWSL (%)	16.53±5.89	21.39±7.78	<0.001
RV4CSL (%)	14.65±4.53	18.34±6.38	<0.001
LASr (%)	29.46±12.95	30.17±9.98	0.768
LAScd (%)	14.20 (10.00–20.15)	16.00 (9.65–21.60)	0.787
LASct (%)	13.94±6.53	13.65±7.53	0.864
GLS (%)	18.45 (17.16–19.51)	19.50 (18.63–21.46)	0.018

The values are presented as the means±SD, medians (I–III) or numbers (%) as appropriate.
BMI, body mass index; BSA, body surface area; COPD, chronic obstructive pulmonary disease; DBP, diastolic blood pressure; FEV1, forced expiratory volume in first second; FVC, forced vital capacity; GLS, global longitudinal strain; LAScd, left atrial conduit strain; LASct, left atrial contractile strain; LASr, left atrial reservoir strain; LVEF, left ventricular ejection fraction; LVMI, left ventricular mass index; mMRC, modified Medical Research Council; PAPS, pulmonary arterial pressure; RV4CSL, right ventricular four-chamber longitudinal strain; RVFWSL, right ventricular free wall longitudinal strain; SBP, systolic blood pressure; TAPSE, tricuspid annular plane systolic excursion.

Correlations of RV, LV and LA strains with several factors in COPD patients

In our study, there was a moderate inverse correlation between the duration of COPD and the RVFWSL and RV4CSL indices (p<0.05). Conversely, there was no correlation between other echocardiographic indices, such as LVEF, GLS, LASr, LAScd, or LASct and FEV1, FEV1/FVC, or duration of COPD (p>0.05). Details are shown in the heatmap (figure 2).

Logistic regression analysis

Table 2 presents the results of the univariate analysis, which revealed that the factors predictive of COPD susceptibility include RV4CSL (OR=0.887 (0.808–0.974)) and RVFWSL (OR=0.905 (0.841–0.974)). However, in the multivariate analysis, only the PAPS emerged as an independent risk factor for predicting COPD susceptibility.

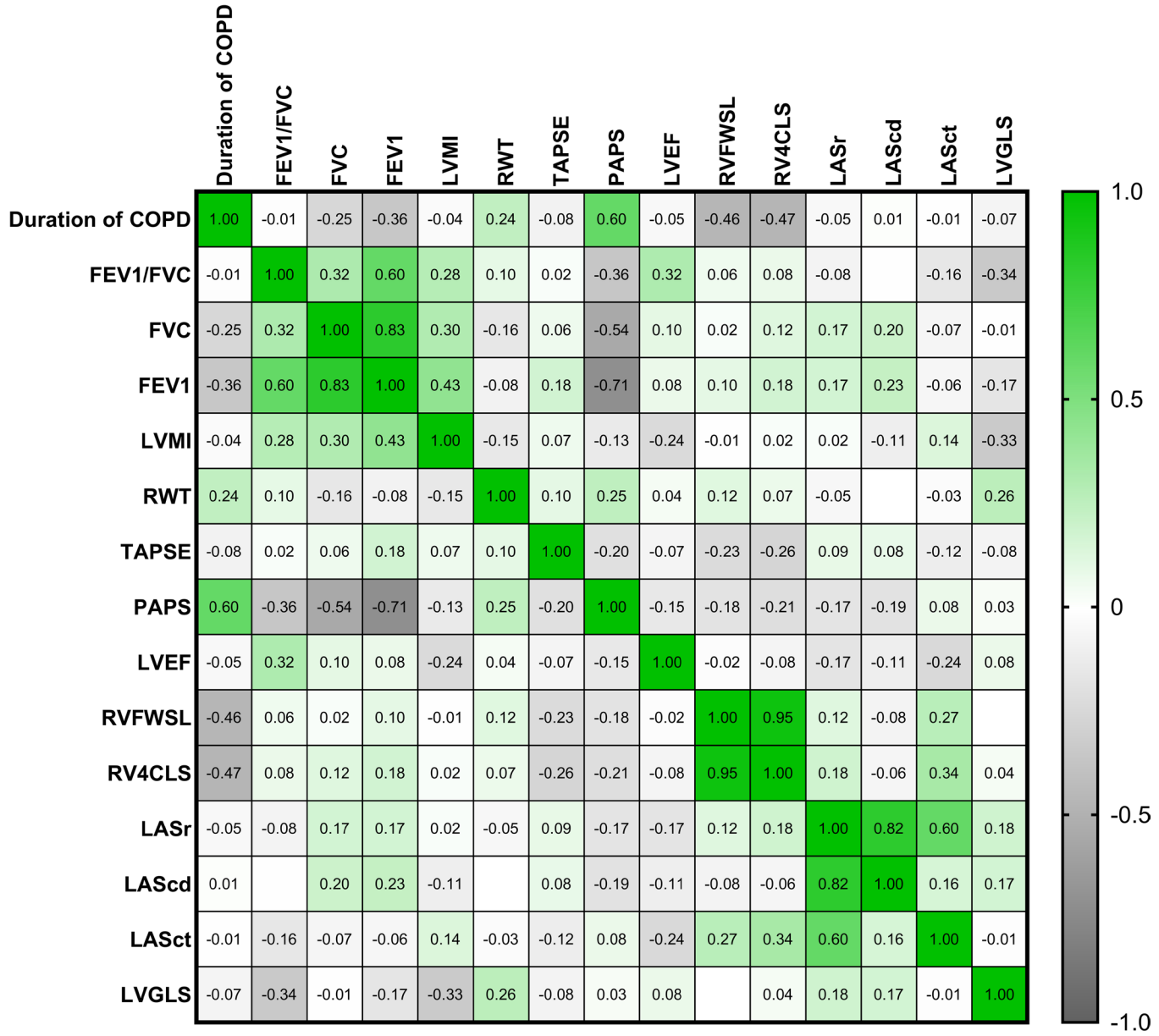
Reliability of STE measurements

Table 3 presents the intraobserver and interobserver variability for STE measurements. The parameters RVFWSL, RV4CSL, LASr, LAScd, LASct, and GLS demonstrated good reproducibility, as indicated by high ICC values.

DISCUSSION

RV strain in COPD patients

Numerous studies have demonstrated that alterations in RV function serve as early predictors of deterioration in stable COPD patients.[20–23] STE can screen for RV dysfunction more effectively in COPD patients, whereas conventional echocardiography methods for estimating pulmonary artery pressure are limited.[10] In our study, the RVFWSL on STE was significantly lower in COPD patients than in controls (p<0.001). The RVFWSL values for the patient and control groups were 16.53±5.89% and 21.39±7.78%, respectively. This finding is consistent with a study by Gökdeniz *et al*, which revealed that RVFWSL in the patient group was lower than that in the control



	Duration of COPD	FEV1/FVC	FVC	FEV1	LVMI	RWT	TAPSE	PAPS	LVEF	RVFWSL	RV4CLS	LASr	LAScd	LASct	LVGLS
Duration of COPD	1.00	-0.01	-0.25	-0.36	-0.04	0.24	-0.08	0.60	-0.05	-0.46	-0.47	-0.05	0.01	-0.01	-0.07
FEV1/FVC	-0.01	1.00	0.32	0.60	0.28	0.10	0.02	-0.36	0.32	0.06	0.08	-0.08		-0.16	-0.34
FVC	-0.25	0.32	1.00	0.83	0.30	-0.16	0.06	-0.54	0.10	0.02	0.12	0.17	0.20	-0.07	-0.01
FEV1	-0.36	0.60	0.83	1.00	0.43	-0.08	0.18	-0.71	0.08	0.10	0.18	0.17	0.23	-0.06	-0.17
LVMI	-0.04	0.28	0.30	0.43	1.00	-0.15	0.07	-0.13	-0.24	-0.01	0.02	0.02	-0.11	0.14	-0.33
RWT	0.24	0.10	-0.16	-0.08	-0.15	1.00	0.10	0.25	0.04	0.12	0.07	-0.05		-0.03	0.26
TAPSE	-0.08	0.02	0.06	0.18	0.07	0.10	1.00	-0.20	-0.07	-0.23	-0.26	0.09	0.08	-0.12	-0.08
PAPS	0.60	-0.36	-0.54	-0.71	-0.13	0.25	-0.20	1.00	-0.15	-0.18	-0.21	-0.17	-0.19	0.08	0.03
LVEF	-0.05	0.32	0.10	0.08	-0.24	0.04	-0.07	-0.15	1.00	-0.02	-0.08	-0.17	-0.11	-0.24	0.08
RVFWSL	-0.46	0.06	0.02	0.10	-0.01	0.12	-0.23	-0.18	-0.02	1.00	0.95	0.12	-0.08	0.27	
RV4CLS	-0.47	0.08	0.12	0.18	0.02	0.07	-0.26	-0.21	-0.08	0.95	1.00	0.18	-0.06	0.34	0.04
LASr	-0.05	-0.08	0.17	0.17	0.02	-0.05	0.09	-0.17	-0.17	0.12	0.18	1.00	0.82	0.60	0.18
LAScd	0.01		0.20	0.23	-0.11		0.08	-0.19	-0.11	-0.08	-0.06	0.82	1.00	0.16	0.17
LASct	-0.01	-0.16	-0.07	-0.06	0.14	-0.03	-0.12	0.08	-0.24	0.27	0.34	0.60	0.16	1.00	-0.01
LVGLS	-0.07	-0.34	-0.01	-0.17	-0.33	0.26	-0.08	0.03	0.08		0.04	0.18	0.17	-0.01	1.00

Figure 2 Heatmap of correlations between traditional echocardiographic indices, strain parameters measured by speckle tracking echocardiography, respiratory function and COPD duration. Shades of purple indicate an inverse correlation, whereas shades of green represent a positive correlation. COPD, chronic obstructive pulmonary disease; FEV1, forced expiratory volume in first second; FVC, forced vital capacity; LAScd, left atrial conduit strain; LASct, left atrial contractile strain; LASr, left atrial reservoir strain; LVEF, left ventricular ejection fraction; LVGLS, left ventricular global longitudinal strain; LVMI, left ventricular mass index; mMRC, modified Medical Research Council; PAPS, pulmonary arterial pressure; RV4CSL, right ventricular four-chamber longitudinal strain; RVFWSL, right ventricular free wall longitudinal strain; RWT, relative wall thickness; TAPSE, tricuspid annular plane systolic excursion.

group (p<0.001).[24] Additionally, a study by Kanar *et al* concluded that RVFWSL was lower in the patient group (p<0.001).[25] Similarly, RV4CSL in our study was also significantly lower in the patient group than in the control group (14.65±4.53% vs 18.34±6.38%, respectively) (p<0.001). These differences indicate that COPD has adverse effects on the RV function of patients. In COPD patients, respiratory function reflects typical irreversible airflow obstruction. Long-term irreversible airflow obstruction increases the risk of pulmonary hypertension in patients. Owing to the anatomical and functional interactions between the pulmonary and cardiovascular systems, many functional disorders affecting one organ often affect the other and vice versa. Thoracic pressure increases in COPD patients with emphysema, leading to increased resistance in the pulmonary circulation.[26] As pulmonary artery pressure gradually increases in COPD patients, the increased afterload allows the heart, particularly the RV, to adapt and compensate, leading to concentric hypertrophy through Laplace's mechanism.

Table 2 Regression analysis for the prediction of chronic obstructive pulmonary disease susceptibility

	Univariable				Multivariable			
Variables	P value	OR	95% CI		P value	OR		95% CI
GLS (%)	0.160	0.877	0.730	1.053				
LASct (%)	0.861	1.006	0.940	1.077				
LAScd (%)	0.714	0.989	0.931	1.050				
LASr (%)	0.503	0.986	0.945	1.028				
RV4CSL (%)	**0.012**	0.887	0.808	0.974	0.548	0.931	0.738	1.175
RVFWSL (%)	**0.008**	0.905	0.841	0.974	0.911	0.984	0.735	1.317
PAPS (mm Hg)	**0.010**	1.082	1.019	1.149	**0.031**	1.071	1.006	1.140
TAPSE (mm)	0.063	0.834	0.689	1.010				

Bold values indicate p≤0.050.
GLS, global longitudinal strain; LAScd, left atrial conduit strain; LASct, left atrial contractile strain; LASr, left atrial reservoir strain; PAPS, pulmonary arterial pressure; RV4CSL, right ventricular four-chamber longitudinal strain; RVFWSL, right ventricular free wall longitudinal strain; TAPSE, tricuspid annular plane systolic excursion.

This hypertrophy, although a compensatory mechanism to maintain blood flow, may eventually lead to impaired cardiac pump function, resulting in right heart failure. Concentric hypertrophy manifests as a reduction in myocardial contractility during systole, as reflected by RVFWSL and RV4CSL, whereas other functional indices, such as TAPSE, may remain unchanged.

RV dysfunction may occur early in COPD patients because of lung damage. However, the clinical manifestations are often unclear until severe pulmonary hypertension and right heart failure occur.[24] Therefore, evaluating RV function via non-invasive methods such as STE is essential for diagnosing and prognosticating cardiovascular pathology early in COPD patients before clinical manifestations appear. The mMRC is a scoring scale for evaluating the impact of dyspnoea on COPD patients' daily activities. The mMRC of patients is influenced by many factors, such as heart failure, exercise tolerance, airway obstruction and gas exchange, leading to prolonged oxygen use. Persistent dyspnoea not only increases the risk of pulmonary hypertension in patients but also enhances patient tolerance, resulting in most COPD patients being hospitalised for severe conditions.[27] The RVFWSL and RV4CSL decreased in patients with typical COPD symptoms and high mMRC scores. The more severe the COPD was, the lower the RVFWSL and RV4CSL were.

In COPD patients, airway obstruction leads to air trapping in the alveoli, exerting pressure on the pulmonary capillaries and increasing pulmonary vascular resistance. Consequently, the heart must work harder to pump blood into the lungs, resulting in elevated pulmonary artery pressure. As an adaptation, the RV begins to undergo hypertrophy to increase contractility, eventually leading to alterations in RV function. This explains why COPD patients with less severe dyspnoea present greater RVFWSL and RV4CSL values than do those with higher mMRC scores. The durations of COPD and pulmonary hypertension are closely related and have negative impacts on patients' health. Lung damage causes gradual airway obstruction, leading to chronic hypoxia. Consequently, chronic hypoxia activates the pulmonary vasoconstriction mechanism, increasing pulmonary vascular resistance and causing pulmonary hypertension. This research outcome is essential for assessing and monitoring disease progression in COPD patients. Monitoring disease duration and assessing RV function via RVFWSL and RV4CSL help identify early cardiovascular complications and pulmonary hypertension in COPD patients, thereby adjusting treatment protocols to improve patients' quality of life.

Table 3 Reliability of speckle tracking echocardiography indices

Variables	ICC	95% CI
Intraobservers (n=30)		
RVFWSL (%)	0.867	0.719 to 0.937
RV4CSL (%)	0.924	0.839 to 0.964
LASr (%)	0.954	0.904 to 0.978
LAScd (%)	0.962	0.922 to 0.982
LASct (%)	0.966	0.929 to 0.984
GLS (%)	0.792	0.562 to 0.902
Interobservers (n=30)		
RVFWSL (%)	0.869	0.727 to 0.938
RV4CSL (%)	0.937	0.869 to 0.970
LASr (%)	0.946	0.886 to 0.974
LAScd (%)	0.969	0.934 to 0.985
LASct (%)	0.976	0.949 to 0.988
GLS (%)	0.770	0.507 to 0.892

GLS, global longitudinal strain; ICC, intraclass correlation coefficient; LAScd, left atrial conduit strain; LASct, left atrial contractile strain; LASr, left atrial reservoir strain; RV4CSL, right ventricular four-chamber longitudinal strain; RVFWSL, right ventricular free wall longitudinal strain.

LV and LA strains in COPD patients

LV dysfunction is associated with a poorer prognosis and increased mortality in COPD patients. Early detection of LV dysfunction through clinical echocardiography is crucial for implementing timely treatment strategies and reducing the risk of disease progression.[28] As shown in table 1, the LVEF did not significantly differ between the COPD and control groups. However, we noted that the GLS in the COPD group was significantly lower than that in the control group. A study involving 52 COPD patients and 29 control participants by Cengiz Elçioğlu *et al* also revealed a decreased GLS in the patient group compared with the control group.[29] Similarly, Fahim *et al* reported lower GLS in the COPD group.[30] Conversely, our study revealed no statistically significant difference in the indices of LA function between the patient and control groups. However, several other studies have shown that LA function in COPD patients with acute myocardial infarction is characterised by altered atrial strain and reduced atrial function. Similarly, research on LA function in COPD patients with atrial fibrillation has also demonstrated changes in atrial mechanics. These studies predominantly involved COPD patients with concomitant cardiovascular diseases, complicating the interpretation of the independent effect of COPD on LA function. Therefore, additional in-depth research focusing solely on LA function in COPD patients is necessary to elucidate the interplay between COPD and LA function.[31 32] On the basis of our results, GLS may change before significant alterations in LVEF occur in COPD patients.

Interplay between COPD and cardiac function

The intricate mechanisms through which COPD and cardiac function interact remain largely unclear. Several authors have suggested that COPD and heart failure share similar risk factors, such as advanced age, systemic inflammation and smoking.[33–35] There is substantial evidence demonstrating the cardiovascular risks associated with smoking.[36 37] Similarly, COPD is a significant and increasing cause of morbidity and mortality, with smoking identified as its primary causative factor. Cigarette smoking is a key risk factor for the development of COPD.[38 39] Numerous intricate molecular mechanisms have been proposed to elucidate the impact of smoking on both COPD and cardiovascular diseases. Smoking-induced systemic inflammation, endothelial dysfunction and oxidative stress contribute to the onset and progression of these conditions. The constituents of tobacco smoke can adversely affect various cellular functions, including those of macrophages and endothelial cells. Additionally, smoking can compromise the innate immune system, disrupt apoptosis and increase oxidative stress in both the respiratory and vascular systems.[40] Systemic inflammation engages innate and adaptive immune responses, which impact the parenchyma and bronchial walls.[41 42] Similarly, cardiovascular diseases are associated with systemic inflammation.[43] Airflow obstruction, caused by thickening of the bronchial walls, has been correlated with various cardiovascular risk factors, including increased subclinical atherosclerosis and impairments in both endothelium-dependent and endothelium-independent vasodilation.[44 45]

Importantly, the medications used to treat COPD can potentially lead to cardiovascular complications, whereas therapies for heart failure can worsen COPD manifestations.[46 47] The interplay between the pathophysiological mechanisms of the respiratory and cardiovascular systems in patients suffering from both COPD and heart failure is intricate and modulated by pharmacological treatments for these diseases. This complexity, particularly with the use of beta 2-agonists in respiratory therapy and beta-blockers in cardiac care, underscores the need for a comprehensive understanding of these conditions.[48 49]

Our study revealed changes in cardiac function in COPD patients via STE. Several authors have also reported that both systolic and diastolic dysfunction of the right and left ventricles are observed in patients with COPD.[50 51] Right heart failure arises from hypoxic vasoconstriction-induced pulmonary hypertension, which eventually leads to left heart failure. Consequently, individuals with COPD have an increased risk of developing cardiac dysfunction due to shared risk factors and pathophysiologic mechanisms.[33]

Using tools such as STE to detect cardiac damage early in COPD patients allows clinicians to devise appropriate treatment strategies, thereby reducing the disease burden and increasing the quality of life for these patients. Our study highlights echocardiography as an effective tool. However, the observed differences may also guide future research into the use of more advanced and expensive modalities, such as MRI, CT and cardiac scintigraphy, to further investigate myocardial changes in COPD patients and mitigate the limitations of echocardiography.

Limitations

- This was a single-centre study conducted in Vietnam. Therefore, the results may not be representative of other ethnic groups. Multicentre studies in various locations worldwide are needed.
- This was a cross-sectional study, which limited our ability to establish causal relationships and patient outcomes. To address this, it is imperative to conduct longitudinal follow-up studies to assess patient outcomes and establish causal relationships.
- The study did not evaluate the impact of medication therapy on cardiac function in COPD patients, as medication changes frequently occur during each follow-up visit in our healthcare system.
- The sample size in this study was limited due to the inherent challenges of ultrasound imaging in areas containing gas or obscured by bone, such as the lungs. In patients with COPD, emphysema presents significant obstacles in the acquisition of high-quality images suitable for strain analysis. Consequently, this

limitation led to a reduced sample size, as obtaining sufficient imaging data was difficult. The small sample size in this study resulted in larger SEs, wide CIs and imprecise effect estimates. This limitation restricts the ability to draw firm conclusions and impacts the reliability of the findings. Therefore, caution should be exercised when these results are applied in clinical practice.

CONCLUSION

Compared with healthy controls, patients with COPD presented decreased RV and LV strain. There were no differences in LA strain indices between the COPD and control groups.

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Ethics approval This study involves human participants and was approved by The research received approval from the Ethics Council of University of Medicine and Pharmacy, Hue University (Approval number: H2020/364). The study was conducted in full compliance with the principles of the Declaration of Helsinki (2013 version). Participants gave informed consent to participate in the study before taking part.

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