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Original Article

A Clinical Study of Left Ventricular Function in Patients with Chronic Obstructive Pulmonary Disease

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Abstract

Background: Chronic obstructive pulmonary disease (COPD) patients usually present with the right ventricular hypertrophy and eventual right side heart failure. Sometimes, disturbance in the left ventricular (LV) function is also observed in COPD patients. The prevalence of the LV diastolic dysfunction in COPD patients using echocardiography parameters varies widely in the literature.

Aim of the Study: This study aims to evaluate the LV function in patients with COPD with or without pulmonary hypertension (PH).

Materials and Methods: A total of 66 patients with COPD without additional cardiac diseases were grouped as "A" and 22 healthy individuals who were matching with sex and age of Group A were named as Group "B." Spirometry, standard, and tissue Doppler echocardiography were performed in both groups. The results obtained were analyzed.

Observations and Results: Among Group A, 36 had PH. The LV systolic function was similar in both the groups. The LV diastolic function and LV function by myocardial performance index (MPI) were significantly different in different grades of COPD. Patients with COPD and hypertension had significantly higher heart rate, less E-wave peak velocity (measured by DTI) ($P < 0.05$), less E/A ratio (measured by DTI) ($P < 0.01$) and E/A ratio (measured by flow), and higher MPI ($P < 0.05$) than normal pulmonary pressure patients.

Conclusion: In patients with progressive COPD, there was the effect on LV diastolic function and LV function by MPI, especially those with PH.

Key words: COPD, Diastolic dysfunction and spirometry, Left ventricular function

INTRODUCTION

Chronic obstructive pulmonary disease (COPD) is basically a disease of airflow obstruction resulting in permanent changes in the lungs over a period of time. This results in extra-pulmonary pathophysiological changes in the cardiovascular system and is associated with important comorbidities that may contribute to the disease severity.

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The airflow obstruction in the lungs is due to an abnormal inflammatory response of the lungs to noxious particles or gases, particularly cigarette smoke.[1] The main causes of morbidity and mortality among COPD patients are cardiovascular disease (CVD) and lung cancer.[2-4] COPD is a major cause of chronic morbidity and mortality throughout the world. COPD is thefourth leading cause of death in the world[5] and further increases in its prevalence and mortality can be predicted in the future time.[6] Among COPD patients, CVD is responsible for approximately 50% of all hospitalizations and 20% of all deaths.[7] Demographic studies have shown that regardless of smoking status, age, or sex, a COPD diagnosis increases the risk of cardiovascular morbidity and mortality by approximately two-fold.[8] The most important complication of COPD is pulmonary hypertension (PH) which can change the natural history of

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COPD and is associated with reduced survival and greater use of health-care resources.[9] Cor pulmonale may develop in patients with COPD and is characterized by elevated pulmonary vascular resistance and right heart failure, with associated reductions in left ventricular (LV) filling, LV stroke volume, and cardiac output, although LV ejection fraction is generally preserved.[10] In this context, the present study was conducted to evaluate the LV function in patients with COPD with or without PH.

Institution of Study

This study was conducted at Kannur Medical College, Anjarakandy, Kannur.

Period of Study

This study was from March 2011 to –February 2014.

MATERIALS AND METHODS

66 patients with diagnosis of COPD were included as Group A and 22 normal subjects are included as Group B as a control group in the present study.

Inclusion Criteria

1. Patients aged above 45 years and below 70 years were included.

Exclusion Criteria

1. Patients with pulmonary diseases such as pulmonary tuberculosis, bronchiectasis, and interstitial pulmonary disease, were excluded.
2. Patients with unstable cardiorespiratory status; the occurrence of respiratory failure, bronchopulmonary infection, or congestive heart failure in the previous 2 months were excluded.
3. Patients with structural diseases of the heart (valvular heart disease, congenital heart disease, and cardiomyopathy) were excluded.
4. Patients with a history of ischemic heart disease defined as typical angina pectoris, prior myocardial infarction, positive exercise test result, positive myocardial scintigraphy, or positive coronary angiography findings were excluded.

An ethical committee clearance was obtained from the institute. An ethical committee approved consent form was used to collect the data. A thorough clinical history was taken about both respiratory and cardiac symptoms in the two group subjects. All the subjects were performed a resting ECG tracing, systolic and diastolic blood pressure measurement, echocardiography, and resting spirometry. Echocardiography was performed in all patients; (A) conventional echocardiography: (i) Measurement of LV EF%, (ii) LV diastolic filling patterns, (iii) the diastolic parameters were measured from at least 3 beats and were defined as follows: E-wave, early maximal transmitral flow velocity; A-wave, peak velocity during atrial contraction in late diastole; and ratio between the early peak transmitral flow velocity (E) and late peak atrial systolic velocity (A) [E/A ratio], and (iv) the right ventricular (RV) systolic pressure was obtained from the velocity of tricuspid regurgitation (tricuspid regurgitation velocity ≥2.9 m/s), (B) pulsed wave DTI: (i) Isovolumetric contraction time, (ii) isovolumetric relaxation time (IVRT), (iii) S-wave duration (ejection time), and (iv) the regional myocardial velocity waves were systolic velocity (S-wave; cm/s), peak early diastolic filling velocity (Em; cm/s) and peak late diastolic filling velocity (Am; cm/s), also Em/Am ratio and Eflow/Em were calculated. Myocardial performance index (MPI) was calculated by the sum of isovolumic contraction time and relaxation time divided by ejection time. Mean MPI value was calculated, and (C) spirometry was done in all the subjects assuming that patients were diagnosed to have COPD if they had FEV1/FVC ratio <0.70. All the patients were classified into four groups as follows:

- Stage I (mild): Post-bronchodilator FEV1 ≥ 80% predicted
- Stage II (moderate): 50%≤ post-bronchodilator FEV1 <80% predicted
- Stage III (severe): 30%≤ post-bronchodilator FEV1 <50% predicted
- Stage IV (very severe): Post-bronchodilator FEV1 <30% predicted or <50% predicted plus chronic respiratory failure.

All data were presented as mean ± SD unless otherwise stated. Comparisons were performed by unpaired *t*-tests for quantitative data. For qualitative data, v2 was used. $P < 0.05$ was used to indicate differences between the groups that were statistically significant.

OBSERVATIONS AND RESULTS

In Group A, among the 66 patients, males were 51 (77.27%) and females were 15 (22.72%). In Group B, there were 22 patients; males were 11 (50%) and females were 1 (50%). The mean age of Group A was included patients was 58.25 ± 3.60 and Group B was 53.74 ± 2.80. There was a statistical significance among the subjects selected in the study in both the groups in regard with gender and mean age ($P < 0.05$) [Table 1].

In Group A, 36 patients had PH and 30 had no PH (45.45%). Assessing the LV systolic function showed the mean ejection fraction in Group A was 63.26 ± 21, and in Group B, it was 62.86 ± 10. The S-wave peak velocity

Table 1: The gender incidence and age distribution in the study groups (A: *n*=66), (B: *n*=22)

Observation	Group A-66	Group B-22	*P* value
Male (%)	51–77.27	11–50	0.028
Female (%)	15–22.72	11–50	0.028
Mean age	58.25±3.60	53.74±2.80	0.034

Table 2: The echocardiogram findings in both the Groups (A: *n*=66), (B: *n*=22)

Observation	Group A	Group B	*P* value
Heart rate (mean±SD)	89.15±8.35	82.93±7.28	0.011
PH			
Present	36 (54.54)	00 (0)	0.015
Absent	30 (45.45)	22 (100)	
LV systolic function			
Ejection fraction (mean %±SD)	63.26±21	62.86±2.10	0.046
S-wave peak velocity (mean %±SD)	08.60±1.01	09.15±0.42	
LV diastolic function			
E-wave peak velocity by mitral flow (Eflow) (m/s) (mean±SD)	0.51±0.11	00.62±0.18	0.039
E/A by flow (mean±SD)	0.90±0.13	01.10±0.04	0.036
E-wave peak velocity by DTI (Em) (cm/s) (mean±SD)	07.89±1.42	11.65±1.73	0.042
Em/Am by DTI (mean±SD)	00.72±0.09	01.32±0.41	0.041
Eflow/Em (mean±SD)	06.15±0.74	05.0±0.75	0.393
IVRT (msec.) (mean±SD)	73.62±4.12	56.70±2.80	0.050
Left ventricular function			
MPI (mean±SD)	0.063±0.03	59.30±1.85	0.021

PH: Pulmonary hypertension, LV: Left ventricular, IVRT: Isovolumetric relaxation time

in Group A was 8.60 ± 1.01 and, in Group B, it was 09.15 ± 0.42 [Table 2]. Similarly, the LV diastolic function values were shown in Table 2. Statistically, when the values of the two groups were compared, they were found to be significant with *P* value below 0.05 [Table 2].

The distribution of the patients of Group A among the four grades of COPD observed was tabulated in Table 3.

DISCUSSION

In the present study, 36 COPD patients (54.545.6%) had PH with a highly statistically significant difference between patient and control groups. PH is the most common complication observed in patients with advanced COPD. It has clear effects on both morbidity and mortality.[10] The actual incidence of PH in COPD is not available in the literature, but an elevation of pulmonary arterial pressure is reported to occur in 20–90% of patients when measured by right heart catheterization with some evidence that pulmonary hemodynamics worsen with worsening airflow obstruction.[11-13] The heart rates were compared between the Groups A and B subject's and found that there was a highly statistically significant difference in heart rate between patient and control groups, it also increased significantly with increasing the severity of COPD and in COPD patients with PH. This may be due to hypoxia and hypercapnia working as stimulants to the sympathetic system. The frequent association of autonomic neuropathy in COPD, as the parasympathetic activity was found to be significantly reduced in COPD, while there was sympathetic excitation, and finally, as a side effect to the COPD medications as bronchodilators.[14,15] The present study showed non-significant difference between patient and control groups in regard with echocardiography assessment of LV systolic function (ejection fraction and S-wave peak velocity). However, the difference between both groups was significant regarding LV diastolic function (E-wave peak velocity by mitral flow, E-wave peak velocity by DTI, Eflow/Em ratio, Em/Am ratio by DTI, Eflow/Aflow ratio by flow, and IVRT) and LV function by MPI [Table 2]. An increase in RV afterload is common in COPD patients. An increase in RV afterload induces an LV diastolic dysfunction (LVDD) due to biventricular interdependence. Transthoracic echocardiography can estimate LV diastolic function using early (E) and late (A) peak diastolic velocities measured with Doppler transmitral flow, and tissue Doppler imaging of mitral annulus velocities including early (Ea) peak diastolic velocity.[16] In their study Lamia *et al*.[17] excluded patients with an LV systolic dysfunction or any other reason of LVDD. They found that the E-wave was significantly lower and the A-wave was significantly higher in COPD patients compared to control subjects. The E/A ratio was significantly lower in COPD patients as well as the Ea velocity and the E/Ea ratio which were significantly higher in COPD patients, indicating an LVDD. They also concluded that LVDD does exist in COPD patients with increased RV afterload and no pre-existing LV dysfunction. The present study observed that the difference between the four grades of COPD as regarding age and gender was statistically non-significant [Table 3]. In this study of COPD patients, 9 mild (25%), 12 moderate (33/.33%), 7 severe (19.44%), and 8 very severe (22.22%) COPD patients had PH. The difference between mild and very severe COPD was highly significant with $P = 0.031$ and between mild versus severe and moderate versus very severe COPD patients was significant ($P < 0.05$). In a similar study by Gupta *et al*.,[17] the frequencies of PH in mild, moderate, severe, and very severe COPD were 16.67%, 54.55%, 60.00%, and 83.33%, respectively. Although the effects of COPD and emphysema on the heart were well recognized, they were studied principally in very severe COPD only.[18] Clinical data on PH in milder COPD are limited due to the invasive procedure required, that is, right heart catheterization. In certain studies of mild-to-moderate COPD patients in who right heart catheterization was used, showed increases in pulmonary artery pressure with exercise.[19,20] In the present study, LV systolic function was not significantly affected between different COPD grades, whereas the LV diastolic function values showed a statistically significant difference. In another study by Funk

Table 3: The comparison between the four grades of COPD (*n*=66)

Observations	Mild COPD *n*=18	Mod. COPD *n*=20	Severe COPD *n*=19	Very severe COPD- *n*=09	*P* value
Male	13	16	17	05	0.036
Female	05	04	02	04	0.028
Mean age	53.26	55.21	54.60	57.10	0.035

et al.[21] observed that LVDD in COPD patients with normal pulmonary arterial pressure and it increased with RV afterload. Present study showed in echocardiography findings of COPD with hypertension had significantly higher heart rate, less E-wave peak velocity (measured by DTI) than normal pulmonary pressure patients. Kasner *et al*.[22] studying LVDD in patients with idiopathic PH using invasive pressure-volume loop analysis and observed that LV dysfunction was present even in the absence of intrinsic LV disease. Bhargava and Sunnerhagen[23] found early diastolic LV regional wall asynchrony in their patients with PH. They explained that it was due to the interventricular interaction caused by pressure gradients across the septum. Similar to this study, the relation between RV pressure and LVDD in a large group of cor pulmonale patients of different etiology (including COPD patients), was confirmed by Martnez *et al*.[24]

CONCLUSIONS

LV diastolic function and LV function by MPI are affected in COPD patients, especially with progression of the disease. COPD patients with PH are more liable to LV diastolic and LV dysfunction by MPI than normal pulmonary pressure COPD patients. Doppler tissue echocardiography is a better tool in the assessment of LV function.

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