Research Article

Presence of Left Ventricular Diastolic Dysfunction in Patients of Chronic Obstructive Pulmonary Disease

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Abstract | The relationship between chronic obstructive pulmonary disease (COPD) and cardiovascular diseases strongly exists as diastolic dysfunction is commonly reported in COPD patients.
Objective: This study was conducted to determine the presence of left ventricular diastolic dysfunction in patients of COPD.
Material and Methods: A cross sectional study was conducted in Mayo Hospital Lahore for one year. A total of 50 confirmed cases of COPD were included in the study. History of smoking was taken and subjects were examined for positive signs for the clinical, ECG, x-ray chest PA view and pulmonary function tests. Additionally, they were subjected to echocardiography and left ventricular diastolic dysfunctions were noted.
Results: In our study, mean age of the patients was 60.7±6.2 years and mean FEV1/FVC ratio was 56.9±14.3%. Similarly mean ejection fraction of the patients turned out to be 60.6±4.6. The mean peak mitral filling velocity of the patients was 69.8±15.9 cm/s. The mean peak mitral filling rate of the patients was 57.2±30.9 cm/s and mean E/A ratio was 1.3±0.3. The mean iso-volumetric relaxation time of the patients was 84.2±14.7 msec. The mean mitral E deceleration time of the patients was 187.4±25.1 msec. The mean atrial flow reversal was 0.28±0.04. There were 14 (28%) patients having left ventricular diastolic dysfunction.
Conclusion: COPD patients had left ventricular diastolic dysfunction and it was related to increase in pulmonary artery pressure.

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Introduction

Chronic obstructive pulmonary disease (COPD) is characterized by fibrosis and lung tissue destruction resulting in decreased elasticity.[1] Many times it describes chronic or persistent cough, breathlessness on exertion or even vague chest pains or non-specific radiological findings on chest x-ray described as bronchitic, fibronodular or reticulonodular changes.[2] Worldwide, it is the fifth leading cause of death.[3] In UK, some 7% of all days of sickness absence from work results from this ailment.[4,5] It is caused by tobacco smoke, alpha$_1$-antitrypsin deficiency, or caused by occupational and environmental factors.[6]

It is asymptomatic in early stages, however, after teenage, there is yearly 25 to 30 mL decrease in FEV1 in non-smokers, compared with an average decline of

45 to 60 mL in smokers. About 20% smokers had an accelerated decline in FEV_1 annually up to 150 to 200 mL. The tobacco smoke is directly proportional to decline in forced expiratory volume in one second (FEV_1). Among the effected 50% smokers, 15%–20% develop significant disease.[7] Majority of patients lose 50% of their lung capacity at the time of diagnosis.[8] The relationship between COPD and cardiovascular diseases strongly exists.[9] The rate of FEV1 decline is directly related to cardiovascular mortality.[10] Diastolic dysfunction is commonly reported in COPD patients.[11] In a recent study, 54 of 109 patients affected by HF with preserved systolic function had an FEV1 <70%.[12] Presence of heart failure with no left ventricular systolic dysfunction results in diastolic dysfunction.[13] The rate of ventricular filling (diastolic dysfunction) is decreased as the ventricular walls thickened, and tachycardia shortens the duration of ventricular filling.[14]

Both COPD and heart failure had common symptoms and signs such as dyspnea, rales, tachycardia and oedema[15]. The chest X rays and ECG increases diagnostic accuracy[16]. The LV dysfunction is high in COPD because of common risk factors with coronary disease like age, male gender and cigarettes smoking.[17] As the disease progresses, it involves both left ventricle (LV) and right ventricle (RV), and their systolic and diastolic functions. Routine echocardiography is required for assessment of LV dysfunction.[18] Currently, data on association between left ventricular diastolic dysfunction and chronic obstructive pulmonary disease is lacking in Pakistan. Results presented in this study would establish bases in early diagnosis and management and would improve the quality of life, and functional status of these patients.

Material and Methods

This cross sectional study was carried out in Mayo Hospital Lahore during a period of one year. After ethical approval of the study, 50 patients were included from indoor department through non-probability purposive sampling technique. Informed written consent was obtained from all patients before inclusion in study. The age range was 40-80 years, with either gender, history of smoking with ≥10 years, persistent symptoms of dyspnea, wheezing, productive cough and chest tightness lasting for ≥3 months along with clinical signs of nicotine staining, barrel shaped chest, vesicular breathing with prolonged expiration, bronchi and coarse crepitation, positive chest radiography findings and FEV1 ≤60% and FEV1/FVC ≤70% of predicted were included. Only the patients with already confirmed diagnosis of heart failure on the basis of clinical signs of orthopnea, paroxysmal nocturnal dyspnea rales, ECG and Chest X-Ray findings were excluded. They were examined for positive clinical signs and were sent for ECG, x-ray chest PA view (cardiothoracic ratio) and pulmonary function tests. They were then subjected to echocardiography and the left ventricular diastolic dysfunctions were noted. Confounders were controlled by stratification.

The statistical analysis was performed using SPSS version 16. Parameters such as age, years of smoking, FEV1, FVC, FEV1/FVC ratio, left ventricular internal diameter in systole and diastole, ejection fraction, peak mitral filling velocity (E), peak mitral filling rate (A), E/A ratio, isovolumetric relaxation time (IVRT), mitral deceleration time, and atrial flow reversal were quantitative variables and were presented as mean and standard deviation. Gender and left ventricular dysfunction was qualitative variables and were presented as frequency and percentages.

Results

The mean age of all subjects was 60.7±6.2 years. There were 45 (90%) male and 5 (10%) female patients in this study. There were 43 (86%) patients with a history of smoking and 13 (26%) patients with occupational history of smoking. The mean FEV1 of the patients was 45.7±13.1% with a range of 20 - 60. The mean FVC of the patients was 78.6±7.8% with a range of 60 to 90. The mean FEV1/FVC ratio was 56.9±14.3 with a range of 30 to 70 (Table 1).

Table 1: *Characteristics of patients studied in this cohort*

Age	60.7±6.2 years
Gender	
Male	45 (90%)
Female	5 (10%)
Smoking	
History	43 (86%)
Occupational	13 (26%)
FEV1	45.7±13.1
FVC	78.6±7.8
FEV1/FVC	56.9±14.3

The mean left ventricular internal diameter in systole was 29.2±2.1 & in diastole was 46.5±3.4. The mean

ejection fraction was 60.6±4.6 with a range of 50 to 70. The mean peak mitral filling velocity (E) of the patients was 69.8±15.9 with a range of 50 to 100. The mean peak mitral filling rate (A) was 57.2±30.9 with the range of 40 to 90 while mean E/A ratio was 1.3±0.3. The mean IVRT was 84.2±14.7 minutes. The mean mitral deceleration time was 187.4±25.1 msec. The mean atrial flow reversal was 0.28±0.04 (Table 2). There were 14 (28%) patients of LVDD and 36 (72%) patients had normal ventricular function (Figure 1).

Table 2: *Echocardiographic findings*

Left ventricular internal diameter In systole In diastole	 29.2±2.1 46.5±3.4
Ejection fraction	60.6±4.6
Peak mitral filling velocity (E)	69.8±15.9
Peak mitral filling rate (A)	57.2±30.9
E/A ratio	1.3±0.3
E/A ratio≤1	8 (16%)
E/A ratio1-2	40 (80%)
E/A ratio>2	2 (4%)
Mean IVRT	84.2±14.7
IVT: 70-90	41 (82%)
IVRT: >90	9 (18%)
Mitral deceleration time	187.4±25.1
Atrial flow reversal	0.28±0.04

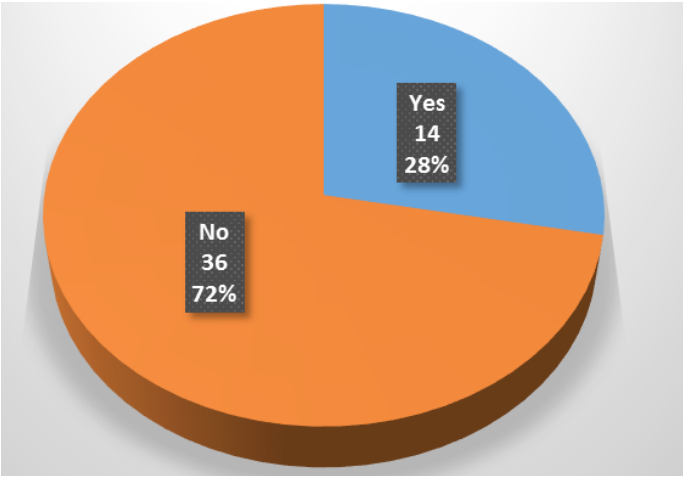

Figure 1: *Presence of LVDD*

Discussion

The mean age of the patients in our study was 60.7±6.2 years which is comparable with the study of Dzudie et al[19] in which mean age of the patients was 55 years with age range of 26 to 82 years. Similarly in Suchoe et al[20] study, the mean age was 62.1±7.7 years, which is almost same and comparable with our study. In our study 86% patients had history of smoking which is comparable with the study of Abroug et al[21] where they seen 83% history of smoking, which is same and comparable with our study.

In our study the mean FEV1 was 45.7±13.1 which is comparable with the study of Suchoe et al[20] where it was 40±8.9. In our study the mean left ventricular systolic internal diameter was 29.2±2.21. It is comparable with the study of Suchoe et al[20] where mean was 33.2±4.2.In our study the mean left ventricular diastolic internal diameter was 46.5±3.4. In the study of Suchoe et al.,[20] it was 38.0±11.2. In another study conducted by Boussouges et al.,[11] it was 48±4.5, which is same and comparable with our study.

In our study the mean EF of the patients was 60.6±4.6% which is comparable with the study of Rocha et al.,[22] where it was 69±9%.The mean E/A ratio of the patients was 1.3±0.3 comparable with the study of Alchanatis et al.,[23] where it was 1.07± 0.3. The mean isovolumetric relaxation time of the patients was 84.2±14.7 msec that was comparable with the study of Alchanatis et al.,[23] where it was 85.6±8.8msec. 28% patients had left ventricular diastolic dysfunction which is comparable with the study of Abroug et al.,[21] where it was 31.1%. In Paudel et al[24]study, it was 26.7%, and was directly proportional to the severity of the disease.[24]

Conclusion

COPD patients had left ventricular diastolic dysfunction and it was related to increase in pulmonary artery pressure, while no left ventricular systolic dysfunction was noted. Therefore, every COPD patient must have a routine echocardiography for early detection of cardiac involvement.

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