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Original article:

Study of Left Ventricular Echocardigraphic Changes in Chronic Obstructive Pulmonary Disease Patients in South Indian population

Kishor Phepale , Swaminathan N , Ravishankar G

Name of the Institute/college: MADRAS MEDICAL COLLEGE, CHENNAI

Corresponding author: Swaminathan N; Email id : kishorphepale@gmail.com

Abstract

Introduction: Changes in right ventricle are common in chronic obstructive pulmonary disease (COPD) but issue has existed regarding the left ventricular changes. We studied left ventricular changes by echocardiography in COPD patients.

Methods: We selected patients with features of COPD. Patients with bronchiectasis, cystic fibrosis, bronchiolitis obliterans and other cardiovascular and systemic diseases were excluded. A control group was selected without history of smoking, asthma, occupational exposure and features of COPD. All cases and control under went echocardiography.

Observations and Results: Out of 56 patients of COPD, nine (17%) patients had left ventricular hypertrophy (LVH). Regarding left ventricular systolic function, mean ejection fraction was slightly elevated but was not statistically significant. An impairment of left ventricular diastolic function was noted in 90% patients. Most of them showed decrease in E velocity and the mean E velocity (58.74 ± 5.26 cm/sec) was significantly decreased from control value (68.25 ± 8.69 cm/sec) ($p < 0.05$). 'A' velocity was increased (66.24 ± 8.15 cm/sec) significantly than control value (53.72 ± 7.03 cm/sec) ($p < 0.05$). E/A ratio was < 1 in most cases (50 patients). In the study group, mean E/A ratio (0.871 ± 0.10) was significantly lower than control group (1.27 ± 0.11) ($p < 0.05$).

Conclusion: COPD is commonly associated with left ventricular diastolic dysfunction (90% cases). LVH is not uncommon (17%). LV systolic dysfunction is not found. A larger study will be more helpful to identify the left ventricular changes in COPD.

Key words: Chronic obstructive pulmonary disease, left ventricular hypertrophy, diastolic dysfunction.

Introduction

Chronic obstructive pulmonary disease (COPD) is common disease in India affecting more patients in their active phase of life and numbers of COPD patients are increasing, with ageing of population. So need to mention various aspects of the disease, to reduce the morbidity and mortality. Involvement of right ventricle is common in COPD but there is issue regarding the left ventricular changes. There are few studies on changes in left ventricle in COPD1-15. We have detected changes in left ventricle of COPD patients, using echocardiography and echo-doppler study in south Indian population.

Material and methods

We selected COPD patients over a period of one year (January 2019 to January 2020) from OPD and IPD in RGGGH, Chennai.

Inclusion criteria included:

1) chronic cough lasting three months for more than two years, breathlessness, prolonged smoking or working in a polluted environment

2) examination findings like wheeze, ronchi, coarse crepitations,hyperinflation of lungs;

3) radiological features indicating hyperinflation of lungs, i.e., translucency, flattening of diaphragm, ¯ peripheral vascular markings, enlarged pulmonary artery and its branches with peripheral pruning;

4) pulmonary function test showing FEV1 < 80% of predicted and FEV1/FVC < 70% of predicted.

Exclusion criteria included:

1)) other causes of COPD, i.e., cystic fibrosis, bronchiectasis, bronchiolitis obliterans;

2) any regional ventricular wall motion abnormality, akinesia, hypokinesia, dyskinesia or evidence of cardiomyopathy on echocardiography;

3) valvular and congenital heart diseases, coronary artery disease and pericardial diseases;

4) systemic diseases, i.e., hypertension, diabetes,hypothyroidism, chronic kidney disease and connective tissue diseases;

5) addiction to alcohol or other drugs that could cause cardiac changes;

6) ECG suggestive of ischaemia (nonspecific ST-T changes in ECG, though common in COPD, were excluded because of possible ischaemia).

Its hospital based cross sectional study including 56 cases ad 34 control.A control group was selected from patients having no history of smoking, asthma,occupational exposure, and no symptoms and signs of COPD and without any change suggestive of COPD in chest X-ray,or pulmonary function test. We matched age, sex, and socioeconomic status of control and study groups.

All patients in our study under went echocardiography and echo-doppler study. The echocardiographer was blinded to cases and control. Patients were subjected to echocardiography, approximately, at the same time in morning to minimise changes due to variations of sympathetic actively. The parameters assessed were anatomy of left ventricle, left ventricular internal diameter in diastole and systole [LVID (d) and LVID (s)], left ventricular posterior wall thickness (LVPWT), interventricular septal wall thickness in systole [IVS (s)] and diastole [IVS (d)].

The measurements obtained by M-mode echocardiography including ejection fraction and fractional shortening were used to assess LV systolic function.

$$\text{Fractional shortening} = \frac{\text{LVID (d)} - \text{LVID (s)}}{\text{LVID (d)}} \times 100$$

$$\text{LV ejection fraction} = \frac{(\text{LVIDd})^3 - (\text{LVIDS})^3}{(\text{LVIDd})^3} \times 100$$

Mitral flow velocities were recorded by doppler study. Epeak [peak velocity of early rapid diastolic filling (cm/sec)], A-peak [peak velocity of late diastolic filling (cm/sec) during atrial contraction] and E/A ratio were calculated. (E/A ratio is normally 1.6 ± 0.5. Normal peak velocity is greater with early diastolic flow. In patients with impaired LV relaxation, peak-E velocity is reduced while peak-A velocity is increased and E/A ratio is altered). We calculated LV mass by using Devereux regression equation and body surface area (BSA).

$$\text{LVMI} = \frac{0.83\,[(\text{LVIDd} + \text{LVPWT} + \text{IVS})^3 - (\text{LVIDd})^3] + 0.6}{\text{BSA}} \quad \text{gm}/m^2$$

(LVMI – LV mass index)

Echocardiographic criteria for left ventricular hypertrophy (LVH) included:

1) IVS(d) or LVPWT(d) > 1.1 cm; and

2) LV mass index: > 111 gm/m^2 in female and > 135 gm/m^2 in male.

Informed consent was taken from all patients for being included in the study.

Statistical methods: We used standard statistical methods for data analysis. Echocardiographic findings were expressed as mean ± standard deviation. Echocardiographic values of two groups were compared by Students' t test. Pvalue < 0.05 was considered as statistically significant.

Observations

Table1 Comparison of general characteristics between patient and control groups

Character	Patient group(n = 56)	Control group(n =34)	P value
Gender n (%)			
Male	47	20	> 0.05
Female	9	14	> 0.05
Age in years(mean±SD)	56.3±6.9	52.1±8.7	> 0.05

Table2 Comparison of Left ventricular dimensions and functions between patient and control groups

Echocardiographic findings	Study group(n = 56)	Control group(n = 34)	P values
1)LV systolic function			
a)Ejection fraction(EF%)	59.14 ± 6.90	57.4 ± 8.114	> 0.05
b)Fractional shortening(FS%)	33.28 ± 5.37	34.94 ± 5.88	> 0.05
2)LV diastolic function			
a)E peak velocity (cm/sec)	58.74 ± 5.26	68.25 ± 8.69	< 0.05

b)A peak velocity (cm/sec)	66.24 ± 8.15	53.72 ± 7.03	< 0.05
c)E/A ratio	0.871 ± 0.10	1.27 ± 0.11	< 0.05
3)LV cavity dimension			
a)LVID(d) cm/BSA	4.33 ± 0.66	3.8 ± 0.6	< 0.05
b)LVID(s) cm/BSA	2.91 ± 0.64	2.4 ± 0.29	< 0.05
c)LVPWT(d) cm/BSA	1.06 ± 0.14	0.92 ± 0.22	< 0.05
d)LVPWT(s) cm/BSA	1.176 ± 0.10	0.97 ± 0.21	< 0.05
e)LV Mass gm/□^{2}	105.84 ± 31	74.63 ± 30.85	< 0.05
4) IVS(s) cm/BSA	1.28 ± 0.15	0.92 ± 0.14	< 0.05
5) IVS(d) cm/BSA	1.05 ± 0.19	0.85 ± 0.13	< 0.05

Abbreviations:

LV-left ventricle; LVID(d) - left ventricular internal diameter in diastole; LVID(s) – left ventricular internal diameter in systole; LVPWT(s) - left ventricular posterior wall thickness in systole; LVPWT(d) - left ventricular posterior wall thickness in diastole; IVS(s) - interventricular septal wall thickness in systole ; IVS (d) - interventricular septal wall thickness in diastole.

Results

General characteristics like gender and age are tabulated and compared(Table 1). Averages of 2-D, M-mode and doppler values of left ventricular dimensions and functions are tabulated and compared (Table 2).

Mean age among cases (56.3±6.9) higher than among controls (52.1±8.7) but the difference was not statistically significant (P > 0.05). Regarding systolic function of left ventricle, mean ejection fraction (59.14 ± 6.90) among cases was slightly elevated than among controls (57.4 ± 8.114) but the difference was not statistically significant (P > 0.05).

Impairment of LV diastolic function was noted. The mean E velocity (58.74 ± 5.26cm/sec) among cases was significantly decreased than controls (68.25 ± 8.69 cm/ sec) value. 'A' velocity was also noted to be increased (66.24 ± 8.15 cm/sec) significantly as compared to controls (53.72 ±7.03 cm/sec) value. E/A ratio was less than one in most(50) cases. In the study group, mean E/A ratio (0.871 ± 0.10) was significantly lower than control (1.27 ± 0.11) value (p < 0.05). Left-ward shift of septum and flattening of IVS was present in 50 patients.

We considered LVH when LVPWT (d) and IVS (d) were > 1.1 cm and exceeded the limit of LVH through Devereux formula. Sixteen patients had LVPWT (d) and IVS (d) > 1.1 cm. Out of 16 patients, nine patients (17%) had increase in both IVS and LVPWT and exceeded the limit of LVH by Devereux formula. Other seven patients didn't exceed the limit of LVH by Devereux formula.

Discussion

Left ventricular systolic dysfunction:

Normal LV systolic function was seen in all of our patients at rest. We found normal ejection fraction (59.14 ± 6.90) (SD = 7.10) and fractional shortening (33.28 ± 5.37%) (SD = 6.48) in COPD patients and values were not significantly different from control group ($P > 0.05$). Previously, a hypothesis was postulated that hypoxia, hypercapnoea, and acidosis can depress LV systolic function and may lead to LV failure. In an Indian study, LV systolic dysfunction was reported in 7.5 % of COPD patient[1]. However, an earlier study by radionuclide ventriculography had shown a normal to supranormal ejection fraction in chronic cor-pulmonale patients due to COP[2]. Jardin *et al,* also had shown normal systolic function in advanced COPD patients by echocardiograph[3]. Poddar *et al* showed that LV systolic function is usually not disturbed in COPD, even after development of cor-pulmonale, but LV systolic dysfunction may be frequently associated with COPD, when overt right heart failure develop[4].

Left ventricular diastolic dysfunction:

Doppler study revealed diastolic dysfunction in majority [50 out of 56 (90%)] of our patients. In our study, 'E'-peak velocity was 58.74 ± 5.26 cm/sec in cases and 68.25 ± 8.69 cm/sec among controls ($P < 0.05$). 'A' peak velocity was 66.24 ± 8.15 cm/sec in cases and 53.72 ± 7.03 cm/sec ($p < 0.05$) among controls. Prevalence of LV diastolic dysfunction is very high in COPD patients, varying with the severity of the diseas[1,5,6]. Different studies have shown that septum mediated ventricular interdependence is the operating factor causig decreased diastolic compliance of left heart in cor-pulmonal[5,7,8].

Chronic RV pressure overload induces LV filling impairment despite a normal systolic phase due to septal leftward shif[9]. Chronic hypoxaemia may also lead to abnormalities of myocardial relaxation and lung hyperinflation may lead to stiffening of parietal pleura and thus of the wall of cardiac foss[1]. LV diastolic dysfunction may also be due to compression of the left ventricle resulting from the limited space within the cardiac fossa and pericardium as well as decreased pulmonary venous return and also correlates with the severity of pulmonary hypertensio[1,5,10,11]. In our study 90% of the patients had diastolic dysfunction as well as both LVID (d) and LVID (s) were increased.

Left ventricular hypertrophy:

In our study, we found LVH in 17% patients. Autopsy studies done in the past have shown the presence of LVH in COPD patient[12]. To determine left ventricular involvement in patients with chronic corpulmonale, right and left ventricular weights, wall thickness, myocyte degeneration and percentage of fibrosis, 18 autopsied hearts were examined by Kohana *et a*[13]. It was seen that walls of both ventricles were significantly thickened and myocyte diameters of both ventricles were significantly greater in COPD and it was concluded that left ventricle was involved pathologically in patients with chronic cor-pulmonale. Dragnov V *et al* have studied 507 patients with chronic cor-pulmonale and, in 62.2% of them, LVH was foun[12]. In an Indian study, LVH was seen in 22.5% of COPD patient[1]. Severity of pulmonary hyperinflation, as measured by residual lung volume seems to be associated with greater LV mass in COPD patient[14]. The cause of LVH is uncertain though hypoxia, hypercarbia,

acidosis, and increased intrathoracic pressure might play some rol[15].LVH is usually not seen in COPD patients with mild hypoxaemi[16]. There are limitations in our study. We could not rule-out the possibility of ischaemic heart disease, having only used history, ECG and echocardiography (as treadmill test, coronary angiography or autopsy studies were not done). It was also not possible to rule out early hypertrophic cardiomyopathy. Echocardiographic assessment is difficult in COPD patients because of lung hyperinflation and there may be inaccuracies.

Conclusion

LV diastolic dysfunction is seen in a majority of COPD patients. LVH is also common. LV systolic dysfunction is not seen. Larger studies are needed to confirm the association of LVH and other left ventricular changes,purely due to COPD

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