ORIGINAL RESEARCH PAPER

Cardiology

KEY WORDS:

PREVALANCE OF LEFT VENTRICULAR DYSFUNCTION IN CHRONIC OBSTRUCTIVE PULMONARY DISEASE (COPD).

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ABSTRACT

Aim: To study prevalence of left ventricular diastolic and systolic dysfunction in patients with COPD.

Patients and methods: 100 patients with COPD referred for cardiac evaluation to our department. Apart from routine investigation 2D, M mode, Doppler and DTI were done. LV diastolic dysfunction was assessed by mitral inflow Doppler. LV systolic function was assessed by LV EF and Mitral annular peak S wave velocity.

Results: 84% of them had pulmonary hypertension. Out of these 54% of them have mild, 26% moderate and 4% severe PHT. 70% of COPD patients had diastolic dysfunction in our study. Our study reports that 10% of the COPD patients had LV systolic dysfunction (EF< 50%).

CONCLUSION: LV systolic dysfunction is directly proportional to severity of COPD. Smoking index and duration of smoking are positively correlated with development of LVSD. Our study reports that 44.4% of severe COPD patients have LVSD. So we recommend routine echocardiography for all severe COPD patients.

INTRODUCTION :

Chronic obstructive pulmonary disease (COPD) is a major cause of chronic morbidity and mortality throughout the world. COPD is the fourth leading cause of death in the world [1], and further increases in its prevalence and mortality can be predicted in the coming decades [2]. In India half a million people die of COPD.COPD obscure the clinical signs of coexisting left ventricular dysfunction like cough, dyspnoea, paroxysmal nocturnal dyspnoea and orthopnoea. Symptoms of dyspnoea in COPD can be partially due to LV systolic and diastolic dysfunction. More number of patients is either under or wrongly diagnosed, leading to not initiating treatment for coexisting LV failure that leads to worsening and development of complications. Early identification and treatment of LV dysfunction can improve the patient's symptoms.

AIM

The aim of this study was to evaluate LV function in patients with chronic obstructive pulmonary disease (COPD)

PATIENTS AND METHODS

The study design was approved by the Ethical Committee of the institution.

This study population included 100 patients with COPD confirmed by medical history and pulmonary function tests and referred to Cardiology OPD for Cardiac evaluation. In patients with COPD, CBC with ESR, RBS, FBS, PPBS, Blood urea & creatinine, lipid profile, LFT, Urine Routine, Ultra sonogram Abdomen, Chest X-ray, Pulse oximetry for SPO2, Pulmonary function test, ECG, ECHO were done after obtaining written informed consent. Severity of COPD was assessed by Gold criteria. LVEF was measured by 2D Echo. PFT was assessed by spirometry. LVEF was compared with severity of COPD.

EXCLUSION CRITERIA :

Patients with Systemic Hypertension, Diabetes Mellitus, known congenital or acquired valvular heart disease, CAD and Cardiac arrhythmias were excluded from the study. Patients whose echo window was suboptimal were not included in our study.

METHODS

The data of each patient was collected in a specifically prepared proforma and including demographic details, proper history, clinical features, CBC with ESR, RFT, FBS, PPBS, LFT, Total Cholesterol& Triglycerides, Ultrasound Abdomen, chest X-ray, ECG, PFT and ECHO.

The COPD severity was assessed by GOLD criteria.

Echocardiography was performed in all patients according to the same protocol.

THE FOLLOWING PARAMETERS ARE OBTAINED FROM ECHOCARDIOGRAM.

EJECTION FRACTION: M-mode or two-dimensional echocardiography measurement of LV dimension from the mid-ventricular level is used to calculate LVEF as follows: LVEF = (LVEDD2 – LVESD2) / LVEDD2 EF-50% - 75% is normal. EF < 50% is considered abnormal.

DIASTOLIC FUNCTION OF THE LV: Diastolic function of LV was assessed by trans mitral flow velocity. Diastolic filling is usually classified initially on the basis of the peak mitral flow velocity of the early rapid filling wave (E), peak velocity of the late filling wave caused by atrial contraction (A), E/A ratio, and deceleration time (DT), which is the time interval for the peak E velocity to reach zero baseline. Diastolic dysfunction can be graded according to the diastolic filling pattern. Grade 1 -mild dysfunction-(E/A ratio 240 milliseconds). Grade 2 -moderate dysfunction- (E/A ratio of 1 to 1.5 and normal DT 160 to 240 milliseconds). Grade 3 (severe reversible dysfunction)&Grade 4 (severe irreversible dysfunction) –(E/A ratio higher than 2, and shortened DT < 160 milliseconds).

PULSED WAVE DTI The pulsed wave Doppler tissue imaging (PW. DTI) was performed for all patrients. From the apical 4 and 2-chamber views, the Doppler sample volume was placed at four different sites of the mitral annulus: anterior, lateral, septal and inferior sites in order to record major velocity time intervals: IVCT (isovolumetric contraction time), IVRT (isovolumetric relaxation time) and S wave duration (ejection time)(9). 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 [9]. 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 [3].

PULMONARY ARTERY PRESSURES:

Flow velocities recorded with Doppler echocardiography are used to determine various intra-cardiac pressures. Pulmonary artery

systolic pressure is equal to the calculated RV systolic pressure. RV systolic pressure can be obtained by adding the estimated RA pressure to the TR velocity.TR velocity reflects the systolic pressure difference between the right ventricle and right atrium .Tricuspid regurgitation velocity was recorded by continuous-wave Doppler echocardiography.

Pulmonary flow acceleration time was determined from RVOT Doppler interrogation for estimation of severity of PHT in patients without significant TR signal.

RA/RV dilation and main pulmonary artery diameter noted and correlated with the severity of PHT.

OBSERVATION

Of the 100 patients 80 were males and 20 were females.

38% of our subjects were moderate smokers and 30% were nonsmokers.

Majority of the smokers have moderate to severe COPD. 91.7% mild smokers and 84.2% of the moderate smokers had moderate COPD. 75% heavy smokers have severe COPD. Severity of COPD is directly related to the smoking index.

In our study report those who smoked more than 10 years had moderate to severe obstruction and 50% of the smokers who smoked more than 30 years had severe obstruction. Prolonged duration of smoking is associated with increased severity of COPD Our study reports that 84% of them had pulmonary hypertension. This is due to chronic hypoxia in COPD that may be the reason for pulmonary hypertension. Out of these 54% of them have mild, 26% moderate and 4% severe PHT.PHT severity is more in moderate and heavy smokers than mild smokers. Duration of smoking is directly proportional to the PHT severity. Moderate PHT is present in those who smoked more than 10 years. The percentage of moderate PHT is more in those who smoked more than 20 years. Severity of COPD is directly related to severity of PHT. Severe PHT is associated with moderate and severe COPD patients. All patients with cor pulmonale have a severe obstructive pattern and 66.7% have moderate PHT.

70% of COPD patients had diastolic dysfunction in our study as assessed by Mitarl E velocity less than A velocity and E' velocity and E/E' ratio. Both RV and LV MPI were prolonged indicating a combined diastolic and systolic dysfunction of both ventricles. In grade1 diastolic dysfunction patients 71.4% have moderate obstruction and 19.4% have severe obstruction. In grade 2 diastolic dysfunction patients 100% have severe obstruction. COPD severity worsens the LV diastolic function. All mild COPD patients have normal LV diastolic function. All severe PHT patients have grade 2-3 diastolic dysfunction.

Our study reports that 10% of the COPD patients had LV systolic dysfunction (EF< 50%). Out of 100 cases 10 cases had EF <50%. Moderate and heavy smokers had LV systolic dysfunction. In moderate smokers 10.5% had LVEF <50%. Patients more than 10 years of smoking had LV systolic dysfunction. Patients who smoke for a long duration had higher rates of LV dysfunction. 33.3% of patients with more than 30 years of smoking had LV systolic dysfunction. Duration of smoking is inversely related to the LV function. In moderate COPD patients only 2.9% of them had LVEF< 50%. But in severe COPD patients 44.4% of them had LVEF < 50%. Among the LV systolic dysfunction patients 80% of them had severe obstructive pattern. Severity of COPD is inversely related to the LV systolic function.

Mild PHT patients had normal LV systolic function. Moderate and severe PHT patients had developed LV systolic dysfunction. In moderate PHT patients 30.8% had LVEF <50%. But in severe PHT patients 50% had LVEF < 50%. Severity of PHT was directly related to LV systolic dysfunction. LV systolic function assessed by LV EF and S wave peak velocity by tissue Doppler were used to confirm LV systolic dysfunction.

Among patients with clinical features of cor pulmonale 50% of them had LVEF <50%. Inpatients without cor pulmonale incidence of LV systolic dysfunction was 4.5%. This report shows that presence of cor pulmonale among COPD patients predicts worsening LV systolic function. 60% of the patients with LVEF < 50% had clinical features of corpulmonale.

DISCUSSION

The severity of COPD was associated with cor pulmonale and PHT. Severity of pulmonary arterial hypertension influences the diastolic and systolic functions of left ventricle.

The development of pulmonary hypertension is a poor prognostic sign in patients with COPD, affecting both mortality and quality of life. Mild-to-moderate pulmonary hypertension is a common complication of COPD. Such a complication is associated with increased risks of exacerbation and decreased survival. Circumstantial and experimental evidence suggests that products of cigarette smoke can initiate pulmonary vascular changes in COPD. Our study reports that 84% of them had pulmonary hypertension. In that 54% of them had mild, 26% moderate and 4% severe PHT. Severity of PHT was more in Moderate and heavy smokers than mild smokers. Duration of smoking was directly proportional to the severity of PHT. In those who are smoking more than 20 years the prevalence of moderate PHT was more. The severity of COPD was directly related to severity of PHT. Severe PHT was present in moderate and severe COPD patients. All cor pulmonale patients had severe obstructive pattern and 66.7% of them had moderate pulmonary hypertension. Smoking index, duration of smoking and severity of COPD were positively correlated with severity of PHT.

Chronic obstructive pulmonary disease patients had a high prevalence of left ventricular diastolic dysfunction, which was associated with disease severity. Boussuges et (4) al found a high prevalence of left ventricular diastolic dysfunction in COPD patients relative to control subjects (76% vs. 35%.)Rutten et a(5)land Funk et al(6) also reported a prevalence < 50% (5) In our study reports 70% of our patients had diastolic dysfunction. In grade1 diastolic dysfunction patients 71.4% have moderate obstruction and 19.4% have severe obstruction. In grade 2 diastolic dysfunction patients 100% are severe obstruction. COPD severity worsens the LV diastolic function. All mild COPD patients had normal LV function. LV diastolic dysfunction shows progressive deterioration with increasing severity of PHT. All severe PHT patients have grade 2-3 diastolic dysfunction. .

In a study conducted at Ibn Sina Teaching Hospital wards and respiratory care unit among patients with COPD to assess for LV systolic dysfunction in COPD, the frequency of LVSD among COPD patients were 21.4% and the percentage of patients with LVSD with pack years > 10 was 100%. These results confirm the association of pack years and mean duration of smoking with development of LVSD(7).Our study reports that 10% of the COPD patients had LV systolic dysfunction. 75% of the heavy smokers and 33.3% of patients more than 30 years of smoking had LV systolic dysfunction. Smoking index and duration of smoking were directly associated with the development of LVSD. In a prospective study of 60 COPD patients with or without corpulmonale attending Manipal Teaching Hospital (MTH), Pokhara reported the prevalence of LV systolic dysfunction to be found to be 26.7%. Severe cases 77% and moderate cases 14.2% had LV systolic dysfunction. The findings directly correlate with the severity of COPD i.e., more the severity of COPD more the probability for the incidence of LV systolic dysfunction.(8)In our study only 2.9% of moderate COPD patients and 44.4% of severe COPD patients have LVEF < 50%.

Among patients with LV systolic dysfunction 80% showed severe obstructive pattern. Moderate and severe PHT patients have developed LV systolic dysfunction. In moderate PHT patients 30.8% have LVEF < 50%. But in severe PHT patients 50% have LVEF < 50%. This report shows that, severity of the COPD and PHT is inversely related tothe LV systolic function. A higher Smoking index, prolonged duration of smoking is associated with poor LV systolic function. Increasing severity of COPD is associated with worsening of LV systolic function. Early identification and

treatment of LV dysfunction can lead to improvement in patient's symptoms and improve prognosis for patients with COPD.

CONCLUSION

LV systolic dysfunction is directly proportional to severity of COPD. Smoking index and duration of smoking are positively correlated with development of LVSD. Our study reports that 44.4% of severe COPD patients have LVSD. So we recommend routine echocardiography for all severe COPD patients. Smoking cessation should be a vital part of treatment plan in COPD patients. COPD obscures the clinical signs of coexisting left ventricular dysfunction. Symptoms of dyspnoea in COPD can be partially due to LV systolic and diastolic dysfunction. Early identification and treatment of LV dysfunction can improve the patient's symptoms. COPD patients with LVSD can be treated with antifailure measures like selective beta-1 blocker, diuretics, ACE inhibitors, angiotensin-II antagonist and aldosterone antagonist. This treatment improves the LV function, reduces the recurrence of COPD exacerbation, improves the quality of life and reduces the morbidity and mortality.

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