COPD & Left Ventricular Function

Bushra Khanam[1], Pradeep Kumar Gupta[2], Nisarg Shah[3], Sonal Jain[4]

ABSTRACT

Introduction: COPD and Heart- when we hear both these terms together, the thing which crosses our mind immediately is pulmonary hypertension and cor pulmonale. COPD and left ventricle diastolic dysfunction (LVDD) are major causes of morbidity and mortality with overlapping symptomatology like cough, dyspnea. But whether there is any correlation between these two remains largely unclear. Study aimed to assess prevalence of LVDD by means of echocardiography in COPD patients. To determine correlation between severity of LVDD and pulmonary artery pressure (PASP) and to differentiate LVDD from COPD exacerbations and manage patients in a better way.

Materials and methods: It was a cross sectional observational study. 100 PFT proven COPD patients were evaluated by 2D Echocardiography for systolic & diastolic functions and PAP. Correlation of prevalence and severity of LVDD with COPD & PAP were done. LVDD was determined by ratio of E/E' (standard mitral inflow maximal velocity/ mitral annular relaxation velocity) which correlates with LV filling pressures. [2] Systolic function was assessed by LVEF (Left Ventricular Ejection Fraction). PASP (mmHg) was measured by Tricuspid regurgitation.

Results: Out of 100 COPD patients, clinically significant LVDD (Grade II AND III) was present in 64 patients. 31 0ut of 100 patients had normal PASP but clinically significant LVDD.

Conclusion: 64% patients of COPD were found to have clinically significant LVDD and 31% patients with even normal PASP had clinically significant LVDD. We see the right heart from our 'tubular vision' while treating COPD and tend to forget the left heart, this study makes us focus on whole heart as a single unit, and to differentiate COPD exacerbations from HFpEF, so we can manage such patients in a better way.

Keywords: COPD, COPD, Cor pulmonale, LVDD, HFpEF, PASP

INTRODUCTION

Relation between Right heart and COPD is well understood. How COPD damages the lungs and ultimately result in secondary pulmonary hypertension, and how it affects right heart and results in cor pulmonale is a well understood pathophysiology.

But the relation between COPD and Left Ventricular Diastolic Dysfunction (LVDD) as well as between Pulmonary hypertension secondary to COPD and LVDD is not studied widely till date.

COPD is reported to have an estimated burden of 210 million people worldwide. Globally, 4th leading cause of death in 2004, projected to occupy 3rd position in 2030.[7] India and China constitute 33% of total human population, account for 66% of global COPD mortality.[5] COPD accounts for ~5, 00,000 deaths and 3% DALY lost in India.[1,8]

Heart Failure (HF) was once thought to arise primarily in the setting of a depressed LVEF. Approximately 50% of patients who develop HF have a Normal or Preserved EF (>/= 50%). These patients are categorized as HF with a preserved EF (*HFpEF*), previously called as Diastolic Failure. Epidemiology of these patients is unclear.[2]

METHODOLOGY

It was a cross sectional observational study. 100 stable patients with spirometrically confirmed COPD diagnosis were included in the study.

As per heart failure and echocardiographic association of European Society of Cardiology, diagnostic evidence of LVDD was obtained non-invasively by tissue doppler (E/E' ratio).[4] Systolic & Diastolic functions and PAP were assessed in these patients.

Study Design

Inclusion Criteria

All patients diagnosed to have COPD according to GOLD (Global Initiative for Chronic Obstructive Lung Disease) criteria for COPD.[3]

Exclusion Criteria

- Acute coronary syndrome
- Past history of decompensated heart failure
- Left ventricular ejection fraction (LVEF) < 50%

Study Protocol

PFT proven COPD patients were evaluated by 2D Echocardiography for systolic & diastolic functions and PAP. Correlation of prevalence and severity of LVDD with COPD & PAP were done. LVDD was determined by ratio of E/E' (standard mitral inflow maximal velocity/ mitral annular relaxation velocity) which correlates with LV filling pressures.[2] Systolic function was assessed by LVEF (Left

[1]Associate Professor, Department of General Medicine, [2]Associate professor, Department of General Medicine, [3]PG Student, Department of General Medicine, [4]PG student, Department of General Medicine, Index Medical College Hospital & Research Centre, Indore, MP, India

Corresponding author: Pradeep Kumar Gupta, Associate professor, Department of General Medicine, Index Medical College Hospital & Research Centre, Indore, MP, India

How to cite this article: Khanam B, Gupta PK, Shah N, Jain S. COPD & Left Ventricular Function. International Journal of Contemporary Medical Research 2021;8(10):J1-J3.

DOI: http://dx.doi.org/10.21276/ijcmr.2021.8.10.5

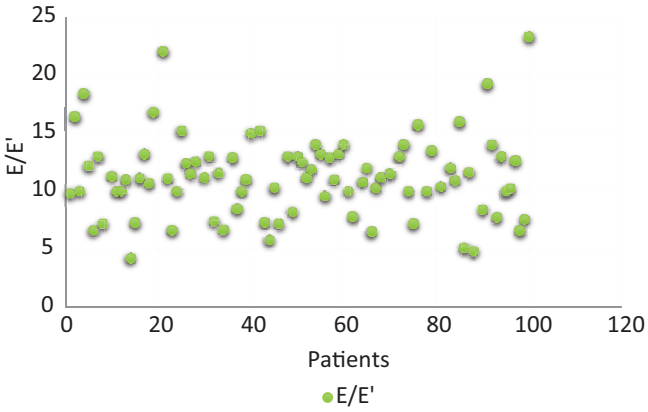
Ventricular Ejection Fraction). PASP (mmHg) was measured by Tricuspid regurgitation.

RESULTS

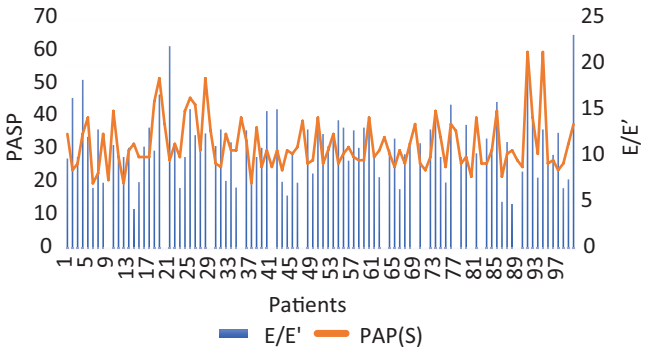
Out of 100 COPD patients, clinically significant LVDD (Grade II AND III) was present in 64 patients, as shown in graph-1, represented by grade of diastolic dysfunction (E/E') for individual patient.

Graph 2 shows the relationship between presence of LVDD with presence or absence of pulmonary hypertension as shown by pulmonary artery pressure.

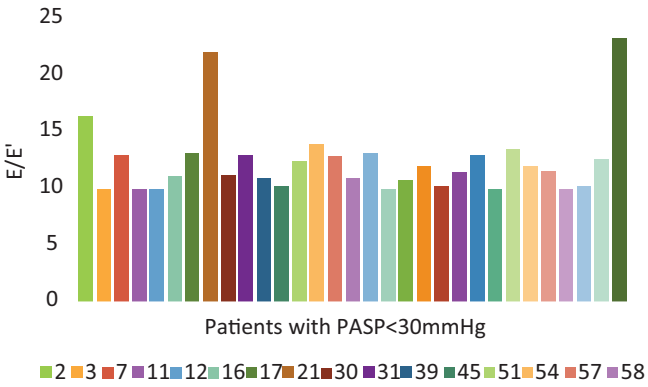
Graph 3 depicts that 31 0ut of 100 patients had normal PASP but clinically significant LVDD.



Graph-1: Graph showing E/E' in all study subjects



Graph-2: Combined graph showing relation between PASP and E/E'



Graph-3: Graph showing Grade II & III LVDD in patients with normal PASP

Grade of LVDD	No. of patients
I	36
II	56
III	08

Table-1: No. of patients with grade of LVDD

DISCUSSION

Ventricular filling is influenced by extent and speed of myocardial relaxation. Passive stiffness of the ventricular wall also may impede filling. Ventricular stiffness increases with hypertrophy and conditions that infiltrate the ventricle such as amyloid, or due to extrinsic constraint e.g. Cardiac tamponade.

Assessment of ventricular filling is performed by flow velocity across mitral valve, which normally is more rapid in early diastole.[2] Mild-moderate impaired relaxation leads to rise in pre- systolic filling rate which leads to decreased early diastolic filling.

Increased RV afterload causes LVDD due to ventricular interdependence; and RV afterload is frequent in COPD patients.[2]

In some studies, it has been shown that subclinical LV systolic dysfunction was also a frequent finding in COPD patients, even in those with normal pulmonary artery pressure.[9]

Whenever we come across an already diagnosed COPD patient in casualty/OPD with breathlessness, usually our initial diagnoses are either acute exacerbation of COPD or cor pulmonale leading to biventricular systolic failure or sometimes secondary pneumothorax in case of emphysema or may be pulmonary embolism; and sometimes we go for extensive series of investigations, which may show non-significant results and increase burden on patient and healthcare system. Echocardiography is a useful non-invasive modality which can help us narrow down the differential diagnoses and lessen the burden produced by multiple investigations. Moderate to severe LV diastolic dysfunction patients can present in this way and require intensive treatment, which we might miss while awaiting results for other high-end investigations.

Angiotensin receptor blockers, angiotensin converting enzyme inhibitors have been shown to reduce morbidity and mortality of COPD patients in some studies; some of these drugs are used as initial treatment for heart failure as per GDMT (Guideline Directed Medical Treatment) for heart failure.[10,11]

As observed in this study, percentage of COPD patients having significant LVDD, sometimes even without presence of pulmonary hypertension was quite appreciable, which leads us to think of LVDD as an important differential while evaluating a COPD patient presenting with "breathlessness".

CONCLUSION

Clinically significant LVDD (Grade II AND III) was present in 64 out of 100 patients included in the study.

31 0ut of 100 patients had normal PASP but clinically significant LVDD.

results suggest that LVDD can be present in COPD patients even without evidence of raised pulmonary artery pressure.

Patients with COPD who present with cough, shortness of breath, chest crepitations etc. should also be evaluated for HFpEF instead of treating them in the lines of COPD exacerbations only.

By doing this we can manage these patients in a better way, reduce usage of Antimicrobial agents which we use frequently in "COPD exacerbations" and to some extent reduce the rising antibiotic resistance.

REFERENCES

1. Prabu et al, Kamaraj et al, Selvaraj et al: A cross sectional study on prevalence of COPD in India: BMJ publishing group.
2. Douglas L. Mann, Murali Chakinala (2018); Harrison's Principles of Internal Medicine (20nd ed.); McGraw-Hill Education; 252:1763
3. Global Initiative for Chronic Obstructive Lung Disease. Global strategy for diagnosis management and prevention of Chronic Obstructive Pulmonary disease. Updated 2014.
4. H JW, Choi D, Park S, Choi EY, Shim CY, Kim JM, Ahn JA, Lee SW, Oh JK, Chung N et al; Heart 2009; 95:399-404
5. World Health Organization. World health statistics 2008. Geneva: BMJ Publishing Group, 2008
6. Gupta D, Agarwal R, Aggarwal AN, et al. COPD Guidelines Working Group Indian Chest Society National College of Chest Physicians (India). Indian J Chest Dis Allied Sci 2014; 56: 5-54.
7. World Health Organization. The global burden of disease 2004 update. Geneva: BMJ Publishing Group, 2008
8. Report on causes of death in India (2001-03).2014.
9. Hilde JM, Hisdal J, Skjørten I, Hansteen V, Melsom MN, Grøtta OJ, et al. Left ventricular dysfunction in COPD without pulmonary hypertension. PLoS ONE 2020;15: e0235075.
10. Javier de Miguel Diez, Jorge Chancafe Morgan, Rodrigo Jimenez Gracia, et al; Chron Obstruct Pulmon Dis. 2013; 8: 305-312
11. Ki Hong Choi, Jin-Oh Choi, Eun-Seok Jeon, Ga Yeon Lee, et al. Guideline-Directed Medical Therapy for patients with heart failure with midrange ejection fraction: A patient pooled analysis from the KorHF and KorAHF registries; http://doi.org/10.1161/JAHA.118.009806| Journal of American Heart association. 2018;7:e009806

Source of Support: Nil; **Conflict of Interest:** None

Submitted: 21-08-2021; **Accepted:** 20-09-2021; **Published:** 30-10-2021