

**Original research article**

## Prevalence of LV systolic dysfunction in COPD patients: A prospective study

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### ABSTRACT

**INTRODUCTION:** According to “WHO” report, COPD at present is the fourth commonest cause of death worldwide. and third most common cause of death and fifth most common cause of chronic disability by the year2030. In India COPD kills half a million people every year. Most COPD patients die of pulmonary and extra pulmonary complications. Pulmonary hypertension, cor pulmonale and left ventricular dysfunction are the important cardiac predictor for mortality.

**AIM OF THE STUDY:** To study the prevalence of LV systolic dysfunction in COPD patients and to assess the possible risk factors contributing to the development of LV systolic dysfunction.

**METHODS AND MATERIALS:** COPD patients admitted in the department of General Medicine in various medical college hospitals are taken for study after obtaining written consent, study period Jan 2018 to Dec 2018

**RESULTS:** Total number of patients taken for study is 500 ,male (396) 80%, female (n=104 )20% In our study prevalence of COPD was more in males. Male Female ratio is 4:1, Our study reports that 44.4% severe COPD have LVSD and 70 % are having diastolic dysfunction

**CONCLUSION:** In our study chronic obstructive pulmonary disease is more common in males. Most patients are low socio economic status. COPD severity directly proportional to smoking index and duration of smoking. Pulmonary hypertension severity directly proportional to severity of COPD, smoking index and duration of smoking. LV systolic dysfunction is directly proportional to severity of COPD. Smoking index and duration of smoking are association for the development of LVSD So we recommend routine echocardiography for all the COPD patients especially the more severe ones. Smoking cessation should be an integral part of treatment plan in patients with COPD.

### INTRODUCTION

Chronic obstructive pulmonary disease (COPD) is a disease state characterized by the presence of airflow obstruction due to chronic bronchitis or emphysema; the airflow obstruction is progressive and partially reversible. COPD is now widely prevalent in both developed and developing countries causing major health burden to the society.

According to “WHO” report, COPD at present is the fourth commonest cause of death worldwide. It is projected to be the third most common cause of death and fifth most common cause of chronic disability by the year2030. In India COPD kills half a million people every year. Cigarette smoking is an important risk factor, other risk factors

such as exposure to indoor and outdoor pollution, occupational hazards, infection and genetic factors are also important.

Most COPD patients die of pulmonary and extra pulmonary complications. Pulmonary hypertension, cor pulmonale and left ventricular dysfunction are the important cardiac predictor for mortality. COPD obscure the clinical signs of coexisting left ventricular dysfunction like cough, dyspnea, paroxysmal nocturnal dyspnea and orthopnea. More number of patients is either under or wrongly diagnosed, that leads to worsening and development of complications. Routine spirometry is used to diagnose and to assess the severity of COPD and echocardiography is mandatory to assess the cardiac status in COPD patients. The awareness among patients regarding COPD and its complications is less, so more attention is needed for COPD patients pertaining to the development of complications.

#### **METHODOLOGY:**

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 SPO<sub>2</sub>, Pulmonary function test, ECG, ECHO will be done after obtaining written informed consent. severity COPD is assessed by Gold criteria. LVEF was measured by 2D Echo. PFT was assessed by Spirometry. LVEF was compared with severity of COPD and possible risk factor behind such development.

**Inclusion criteria:** All patients with COPD.

**Exclusion criteria :** Systemic Hypertension ,Diabetes MellitusH/o congenital, acquired valvular heart disease,IHD,Cardiac arrhythmias,Chronic AF,Complete RBBB or LBBB,H/o heart failure ,Chronic kidney disease,Patients with poor echo window,Patients unable to perform PFT

#### **Data collection:**

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

#### **A diagnosis of COPD should be considered in:**

Persistent cough with expectoration for 3 months in two consecutive years

Chronic cough- Present intermittently or present throughout the day  
Chronic sputum production,  
Dyspnea that is-  
Progressive (worsens over time)- Persistent (present everyday) Worse on exercise,  
Worsens during respiratory infections

Leg swelling for cor pulmonale,History of exposure to risk factors –smoking, air pollution  
On clinical examination: pallor, edema, elevated JVP,purse lip breathing,pulse,CVS-P<sub>2</sub>loud,RS-wheeze,crepitations.

Chest X-ray: evidence of Hyper translucency, low flat diaphragm, widened inter costal space, and tubular heart.

**SPIROMETERY:** COPD is diagnosed only if FEV<sub>1</sub> <80% predicted and FEV<sub>1</sub>/FVC <0.7(mild COPD FEV<sub>1</sub> >80% predicted)

**Performance of Spirometry:** Spirometry was done according to the guidelines published by the British Thoracic Society (BTS). Patient's sex, age and height are entered in the spirometer. The patients were demonstrated the maneuver first and seated in a comfortably. They were asked to take deep full inspiration. Good seal was maintained

with the mouth piece of spirometry. Patients were asked to blow the breath out, forcibly as hard and as fast as possible, until there is nothing left to expel for at least 6 seconds and to a maximum of 15seconds. Spirometer the results (FEV<sub>1</sub> & FEV<sub>1</sub>/FVC) may appear on a display and noted. The results of the measurement were accepted only smooth & cough free. This maneuver was repeated three times. Best of the three consistent readings of FEV<sub>1</sub> and FVC were taken. Reversibility test was done thirty minutes after salbutamol 200 mcg nebulization to rule out bronchial asthma (reversibility more than 12% FEV<sub>1</sub> & FEV<sub>1</sub> increases > 200 ml). Calculation of % improvement FEV<sub>1</sub> / FVC % < 70 is used to diagnose COPD

For assessment of Severity of COPD by FEV<sub>1</sub>%

The COPD severity was assessed by GOLD criteria.

GOLD Stage	Severity	Symptoms	Spirometry
0	At Risk	Chronic cough, sputum production	Normal
I	Mild	With or without chronic cough or sputum production	FEV <sub>1</sub> /FVC <0.7 and FEV <sub>1</sub> ≥80% predicted
IIA	Moderate	With or without chronic cough or sputum production	FEV <sub>1</sub> /FVC <0.7 and 50% ≤FEV <sub>1</sub> <80% predicted
III	Severe	With or without chronic cough or sputum production	FEV <sub>1</sub> /FVC <0.7 and 30% ≤FEV <sub>1</sub> <50% predicted
IV	Very Severe	With or without chronic cough or sputum production	FEV <sub>1</sub> /FVC <0.7 and FEV <sub>1</sub> <30% predicted or FEV <sub>1</sub> <50% predicted with respiratory failure or signs of right heart failure

Electrocardiograph was taken and looked for evidence of pulmonary hypertension in chronic obstructive airways disease.

70% of COPD patients have diastolic dysfunction in our study.

### SEVERITY OF COPD AND DIASTOLIC FUNCTION

Severity of COPD		Diastolic Function				Total	p value
		Normal	Grade I	Grade II	Grade III		
Mild	Number of persons	6	0	0	0	6	<b>&lt;0.001**</b>
	% within Severity of COPD	100.0%	.0%	.0%	.0%	100.0%	
	% within Diastolic Dysfunction	40.0%	.0%	.0%	.0%	12.0%	
Moderate	Number of persons	9	25	0	1	35	
	% within Severity of COPD	25.7%	71.4%	.0%	2.9%	100.0%	
	% within Diastolic Dysfunction	60.0%	80.6%	.0%	100.0%	70.0%	

Severe	Number of persons	0	6	3	0	9	
	% within Severity of COPD	.0%	66.7%	33.3%	.0%	100.0%	
	% within Diastolic Dysfunction	.0%	19.4%	100.0%	.0%	18.0%	
Total	Count	15	31	3	1	50	
	% within Severity of COPD	30.0%	62.0%	6.0%	2.0%	100.0%	
	% within Diastolic Dysfunction	100.0%	100.0%	100.0%	100.0%	100.0%	

In patients with grade1 diastolic dysfunction 71.4% have moderate obstruction and 19.4% have severe obstruction. In grade 2 diastolic dysfunction patients 100% are severe obstruction. ‘p’ value is highly significant. COPD severity worsens the LV diastolic function. All patients with mild COPD have normal LV diastolic function. All patients were subjected to both 2 d and doppler echocardiogram the following parameters are obtained from echocardiogram. EJECTION FRACTION:M-mode or two-dimensional echocardiographic measurement of LV dimensions from the mid-ventricular level is used to calculate  $LVEF = (LVEDD^2 - LVESD^2) / LVEDD^2$

Normal EF-50% - 75%. EF <50% taken as LV systolic dysfunction.

#### DIASTOLIC FUNCTION OF THE LV:

The trans mitral pressure gradient or the relationship between LA and LV

Pressures are accurately reflected by mitral inflow Doppler velocities. 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 <1.0 and DT >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)

#### PULMONARY ARTERY PRESSURES:

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

PA pressure in PHT.

PHT GRADE	PA PRESSURE
MILD	30 – 50 mmHg
MODERATE	50 – 80 mmHg
SEVERE	>80 mmHg

PULMONARY VELOCITY ACCELERATION TIME(PVAT):

There may be situations in which no tricuspid regurgitation is present or the signal is inadequate to obtain reliable measurements. PVAT also used to assess the severity of PHT.

PHT SEVERITY	PVAT
Normal	>120 milliseconds
MILD	100 – 120 milliseconds
MODERATE	80 – 100 milliseconds
SEVERE	<80 milliseconds

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

#### SMOKING INDEX AND COPD

Smoking Index	Frequency	Percentage
Non smoker	150	30%
< 100	120	24%
101-300	190	38%
> 300	40	8%
Total	500	100%

#### DISCUSSION:

Studies have suggested that tobacco smoke exposure may play an important role in the development of pulmonary vasculopathy; therefore, smoking cessation should be an integral part of any treatment plan in patients with COPD. Oxygen supplementation can modestly increase exercise tolerance, decrease PAP, PVR and improve RV function patients with COPD. Long term oxygen therapy (LTOT) is to slow down and partially reverse the progression of PHT in patients with COPD. Oral and inhaled short-acting beta 2-adrenoreceptor agonists do seem to increase the risk of mortality and number of heart failure exacerbations in patients with left ventricular dysfunction. COPD patient is suffering from pre-existing cardiac arrhythmias, CAD and hypoxemia, LABAs may have adverse effects on the myocardium. Lowest possible doses of SABAs should be used, preferably by a metered dose inhaler or a dry

powder system, on an as-needed basis. Currently, inhalation of long-acting beta2 adrenoreceptoragonists is preferred in most COPD patients. These drugs are more quickly removed from myocardial and kidney receptors, thus making potentially deleterious cardiac effects less likely.

No short-term or long-term adverse cardiac effects have been noted with anti cholinergics, either ipratropium or tiotropium, in patients with COPD with coexistent HF. Corticosteroids do have the potential to cause water retention that can worsen HF as well as lead to metabolic complications. Theophyllines are cardio toxic if serum levels exceed 20mg/L. Palpitations and arrhythmias, especially multifocal atrial tachycardia and ectopics are commonly seen. If heart failure coexist with COPD arrhythmias are even more likely. Heart failure decreases elimination of theophylline. Hence, these drugs are best avoided in coexistent COPD-HF, both in stable patients and in those presenting with acute dyspnea.

In COPD-HF, excessive diuresis can cause metabolic alkalosis that theoretically may inhibit ventilation but in normal dosages pulmonary function is not influenced by diuretics. A recent systematic review showed that cardio selective beta-blocking agents can be administered safely to COPD patients. The ACE inhibition and antagonism of angiotensin-II is the cornerstone of treatment of HF. It reduces airways obstruction as well as prevent lung injury. Other putative benefits may be there as they can also decrease pulmonary inflammation and pulmonary vascular constriction and improve the alveolar membrane gas exchange.

Aldosterone can damage the alveolar-capillary membrane. Aldosterone antagonists such as spironolactone may also have positive effects on gas diffusion. Digitalis may reduce lung function because it can cause pulmonary vasoconstriction. So aldosterone digoxin not usually recommended in COPD-HF. In COPD if heart failure is prominent, it maybe good to introduce gradually specific beta 1 blocker like Metoprolol or Bisoprolol along with inhalational bronchodilator therapy after optimizing the heart failure therapy with drugs like diuretics, angiotensin converting enzyme inhibitor and aldosterone antagonist.

## REFERENCES:

1. "The history of COPD". *Int J Chron Obstruct Pulmon Dis* **1** (1): 3–14. doi:10.2147/copd.2006.1.1.3. PMC 2706597. PMID 18046898
2. Fishman AP (May 2005). "One hundred years of chronic obstructive pulmonary disease". *Am. J. Respir. Crit. Care Med.* **171** (9): 941–8. doi:10.1164/rccm.200412-1685OE. PMID 15849329
3. Global strategy for the diagnosis, management and prevention of chronic obstructive pulmonary disease (updated 2010)
4. Ray D, Abel R, Selvaraj KG. A 5-yr prospective epidemiological study of chronic obstructive pulmonary disease in rural South India. *Indian J Med Res* 1995; 101 : 238-44.
5. Pandey MR. Prevalence of chronic bronchitis in a rural community of the hill region of Nepal. *Thorax* 1984;39:337-33