Correlation Between COPD And Echocardiographic Features With Severity Of Disease

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Abstract: Background & Objective**:** Chronic obstructive pulmonary disease (COPD) is the most common cause of secondary pulmonary hypertension (PH). PH secondary to COPD is associated with a worse prognosis of the disease, a low quality of life, as well as with a higher exacerbation frequency, and consequently with an increase in the healthcare cost of COPD patients. Objective is to assess the cardiac changes secondary to COPD by echocardiography and to find out the correlation between echocardiographic findings and severity of COPD, if there is any Methodology: A total 100 of patients of COPD were selected and staged by pulmonary function test (PFT) and evaluated by echocardiography. Results: On echocardiographic evaluation of COPD, 55% cases had normal echocardiographic parameters. Measurable tricuspid regurgitation (TR) was observed in 24/35 cases (67.5%). Pulmonary hypertension (PH), which is defined as systolic pulmonary arterial pressure (sPAP)> 30 mmHg was observed in 15/25 (63%) cases in which prevalence of mild, moderate, and severe PH were 10/17 (58.82%), 3/16 (23.53%), and 3/17 (17.65%), respectively. The frequencies of PH in mild, moderate, severe, and very severe COPD were 16.67%, 54.55%, 60.00%, and 83.33%, respectively. Right atrial pressure was 10 mmHg in 82.5% cases and 15 mmHg in 17.5% cases. Cor pulmonale was observed in 7/17 (41.17%) cases; 7.50% cases had left ventricle (LV) systolic dysfunction and 47.5% cases had evidence of LV diastolic dysfunction defined as A ≥ E (peak mitral flow velocity of the early rapid filling wave (E), peak velocity of the late filling wave caused by atrial contraction (A) on mitral valve tracing) Left ventricle hypertrophy was found in 22.5% cases. Conclusion: Prevalence of PH has a linear relationship with severity of COPD and severe PH is almost associated with cor pulmonale. Echocardiography helps in early detection of cardiac complications in COPD cases giving time for early interventions. [Gupta R NJIRM 2016; 7(1):26-30]

Key Words: Chronic obstructive pulmonary disease, cor pulmonale, echocardiography, pulmonary hypertension

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Introduction: Chronic obstructive pulmonary disease (COPD) has considerable effects on cardiac functions, including those of the right ventricle, left ventricle, and pulmonary blood vessels. Most of the increased mortality associated with COPD is due to cardiac involvement. Echocardiography provides a rapid, noninvasive, portable, and accurate method to evaluate the cardiac changes[1]. Chronic obstructive pulmonary disease (COPD) is the most common cause of secondary pulmonary hypertension (PH).

PH secondary to COPD is associated with a worse prognosis of the disease, a low quality of life, as well as with a higher exacerbation frequency, and consequently with an increase in the healthcare cost of COPD patients. Prevalence of PH in COPD patients is currently unknown. The most important mechanisms leading to PH are hypoxic vasoconstriction, pulmonary hyperinflation and endothelial dysfunction. PH should be suspected in COPD patients in the presence of severe dyspnoea, disproportionate from the decline in lung function, or of severe hypoxemia.

Exercise induced PH is an independent predictor of the development of resting PH in patients with COPD. Echocardiography is the first screening method for PH in patients with COPD and it should be widely used, as it can also appreciate the cardiac consequences of PH, especially on the right ventricle. Given the high negative predictive value of the echocardiographic estimation of systolic pulmonary arterial pressure (sPAP) in the diagnosis of PH, the absence of a high sPAP excludes important PH and further unnecessary invasive evaluation[2]. Right cardiac catheterization remains the "gold standard" method in assessing PH, but it is less accessible and cannot be used in routine evaluation of patients with COPD. PH secondary to COPD is usually mild, but a small proportion of patients have severe PH, with specific characteristics, worse prognosis and a specific therapeutic approach.

Chronic obstructive pulmonary disease (COPD) has been defined by the Global Initiative for COPD as disease state characterized by airflow limitation that is not fully reversible[3]. COPD is a chronic, slowly progressive disorder characterized by airflow obstruction (FEV1<80% predicted and FEV1/FVC ratio <70 %) which does not change markedly over several months; the impairment of lung function is largely fixed but may be partially reversible by bronchodilators.

Chronic obstructive pulmonary disease has considerable effect on cardiac functions including those of right ventricle and pulmonary blood vessels. Most of the increased mortality associated with COPD is due to cardiac involvement. Echocardiography provides a rapid non invasive procedure to evaluate the cardiac changes. This study was conducted with the objective: 1.To assess the cardiac changes secondary to COPD by ECHO to find out the correlation between echo findings and severity of disease, if there any 2. To find out the correlation between echocardiographic findings and the severity of COPD using GOLD guidelines.

Material and Methods: This study was done at SHKM GMC Mewat during the period Feb 2014 to Dec 2014. Clearance was taken from ethical committee of SHKGMC.It included the patients 100 patients with COPD. The mean age of patients were 55+/- 10 years. The mean duration of symptoms were 18 months +/- 10 months

To find out the correlation between echo findings and severity of COPD using Gold standard. Echo will assess the pericardium , valvular anatomy and function, left and right chamber size and cardiac functions

Tricuspid regurgitant flow was identified flow was identified by color Doppler and max jet velocity will be measured by continuous wave Doppler.

Right ventricular systolic pressure was estimated based on Bernoulli theorem

SPAP =4V.V +RAP
RAP was taken as 5,10,15 depending upon the size of IVC

Echocardiography was reviewed to assess the pericardium, valvular anatomy and function, left and right side chamber size and cardiac function. Tricuspid regurgitant flow was identified by color flow Doppler technique and the maximum jet velocity was measured by continuous wave Doppler without the use of intravenous contrast.[4,5] Right ventricular systolic pressure was estimated based on the modified Bernoulli equation and was considered to be equal to the sPAP in the absence of right ventricular outflow obstruction: sPAP (mmHg) = right ventricular systolic pressure = trans-tricuspid pressure gradient (TTPG) + right atrial pressure (RAP), where trans-tricuspid gradient is 4*v*2 (*v* = peak velocity of tricuspid regurgitation, m/s). [6,7,8,9] RAP was empirically estimated as 15 mmHg before 1997. Since1997, RAP was estimated to be 5, 10, or 15 mmHg based on the variation in the size of inferior vena cava with inspirationas follows: complete collapse, RAP = 5 mmHg; partial collapse, RAP = 10 mmHg; and no collapse, RAP = 15 mmHg. [10]

PH(pulmonary Hypertension)will be defind in the study as SPAP > 30 mm of Hg, this definition of PH was chosen as per the classification of PH. PH was classified as MILD (30-50) MODERATE (50-70) AND SEVERE IS >70

Right ventricle dimension will be measured by M mode echo and right ventricle dilation or COR Pulmonale will be present when it exceeds its normal range (0.9 -2.6 cm) right ventricle contractibility will also be noted and right ventricle systolic dysfunction will be present when it is Hypokinetic.

Left ventricular function will be assessed by using the following parameters
EF-- the relation between diastolic and systolic volumes,(56-78)
FS fractional shortening percentage change in LV dimensions with each contraction of LV(28-44)
LV mass =left ventricular mass (88-224 gm)

E/A = diastolic filling of left ventricles 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). In normal subjects LV elastic recoil is vigorous because of normal myocardial relaxation, therefore more filling is completed during early diastolic, so left ventricular diastolic dysfunction (LVDD) is said to be present when E/A is <1.3 (age group 45–49 years), <1.2 (age group 50–59 years), <1.0 (age group 60-69 years), and <0.8 (age group ≥70 years). [11]

Right ventricle dimension was measured by M-Mode echo and right ventricular dilation or cor pulmonale was said to be present when it exceeded the normal range of 0.9–2.6 cm. Right ventricle contractility was also noted and right ventricular systolic dysfunction was said to be present when it was hypokinetic.

Left ventricular function was also assessed by using the following parameters: EF (ejection fraction) = measure of how much end-diastolic value is ejected from LV with each contraction (56%–78%). E/A = diastolic filling of left

ventricles 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). In normal subjects LV elastic recoil is vigorous because of normal myocardial relaxation, therefore more filling is completed during early diastolic, so left ventricular diastolic dysfunction (LVDD) is said to be present when E/A is <1.3 (age group 45–49 years), <1.2 (age group 50–59 years), <1.0 (age group 60-69 years), and <0.8 (age group ≥70 years). [11]
100 patients of COPD was confirmed by clinical history, radiology of chest at Medicine Dept SHKM GMC. PFT was done to confirm the diagnosis.

Echo machine used was IE33 Phillips with multi frequency probe

The 100 patients were grouped into following 3 categories on the basis of clinical and radiological findings
CAT I--- 55 patients with features of COPD only
CAT II—28 patients of COPD with features of COR

Pulmonale without right heart failure
Cat III—17 pat of COPD with COR Pulmonale with right Heart failure

Table I : A Radiological findings in these three categories

Category	Normal	With increased BV markings	With features of overinflation	Higher CT ratio
I	26	16	8	5
II	10	12	4	2
III	7	6	2	2

Results:

Table II : Results of PFT

Cat	FVC (Lit)	FEV(Lit)	FEV/FVC
I	3.25	1.81	55.6%
II	2.65	1.31	49.43%
III	2.36	0.95	40.3%

Table III :
Average results of LV function parameters

cat	EF	FS
I	65	38.5
II	55	36.5
III	45.6	21.3

Comparative study of LV functions of these 3 categories of Pat revealed that though LV function was well preserved among cat I and Cat II, it was depressed among Cat III patients.

Patient wise analysis was done in Cat III, it showed 13 pat out of 17 were having LV dysfunction.

Table IV: Echocardiographic findings (other than LV function) in our study

Findings	No of pat	Percentage of pat
Normal study	55	55
Pulmonary HT SPAP>30	38	38
Mild	20	20
MOD	10	10
Severe	8	8

Table V: COR PULMONALE

RSVD	17
LVH	8
LSVD	3

Table VI : FREQUENCY OF PH with severity of disease

MILD	16.7
Moderate	54.6
SEVERE	60
Very severe	83

It shows frequency of PH increases as severity of COPD

Discussion: The cardiac manifestations of COPD are numerous. Impairment of right ventricular dysfunction and pulmonary blood vessels are well known to complicate the clinical course of COPD and co-relate inversely with survival. Significant structural changes occur in the pulmonary circulation in patients with COPD. The presence of hypoxemia and chronic ventilator insufficiency is associated with early evidence of intimal thickening and medial hypertrophy in the smaller branches of the pulmonary arteries.

Coupled with these pathological changes are pulmonary vasoconstriction arising from the presence of alveolar hypoxemia, destruction of pulmonary vascular bed, changes in intrinsic pulmonary vasodilator substances (such as decrease in PGI 2 s (prostacyclin synthase), decrease in eNOS (endothelial nitric oxide synthase), and increase in ET1 (endothelin1) leads to remodeling, increase in blood viscosity, and alteration in respiratory mechanics. All these lead to a significant increase in pulmonary vascular resistance, the consequence of

which is pulmonary hypertension. Severe PH increases right ventricular after load with a corresponding increase in right ventricular work, which results in uniform hypertrophy of the right ventricle. In patients with COPD, hypoxic vasoconstriction is associated with not only right ventricular hypertrophy but also right ventricular dilation which eventually leads to clinical syndrome of right heart failure with systemic congestion and inability to adapt right ventricular output to the peripheral demand on exercise.

Pulmonary hypertension is a common complication in patients with severe hypoxic COPD, but the elevation in pulmonary arterial pressure (PAP) is usually relatively mild, although its presence indicates a poor prognosis. A minority of patients have severe pulmonary hypertension, whose prognosis is very poor with the development of right heart failure.

Pulmonary hypertension in COPD is thought to result from hypoxic pulmonary vasoconstriction leading to structural remodeling of all layers of the pulmonary arterial walls. The simple hypothesis that hypoxemia in patients with chronic lung disease results in pulmonary hypertension, which adversely affects right ventricular function and hence increases morbidity and decreases exercise tolerance, leading to the development of peripheral edema and increased mortality, is still somewhat controversial. Whether therapeutic interventions that directly affect PAP or right ventricular function have a significant effect on long-term survival in patients with pulmonary hypertension secondary to hypoxic lung disease is still a matter of debate.

Furthermore, whether such interventions will have an effect on symptoms or exercise tolerance remains unproven. Present therapies are limited to the correction of hypoxemia over the long term, which has been shown to have proven benefits on survival. Further studies are required of more specific pulmonary vasodilators or therapies to improve right ventricular function in these patients.

Thus we can see that there is a good correlation between the frequency of PH and severity of disease COPD. The frequency of COR Pulmonale with mild PH is 10, with Moderate PH it is 75 and with severe PH it is 100 %.So we can see that there is a good correlation between severity of PH and COR Pulmonale.

This study shows that despite severe airflow obstruction and Mild AV Hypertrophy, many patients maintain LV function as a reasonably satisfactory level, but with the development of Right side failure LV function also detoriates.

However functional interdependence among cardiac chambers might exist among the patients of COPD as it was shown that failure of one chamber depressed the performance of other chamber this might have some clinical significance as A LV dysfunction even at subclinical level might delay the expected improvement in RV failure in patients of COPD. B Dysfunction of both the ventricles in COPD patients, simultaneously might lead to a state of refractory Heart failure. To conclude LV function is not usually disturbed in patients of COPD even after development of COR Pulmonale but LV dysfunction may be frequently associated with COPD when overt right heart failure develops.

However it should be noted that even though statistically significant , the correlation of pulmonary arterial pressure with spirometry and gas transfer indices are fairly poor ,although a better predictor for PHT than other measurements , the partial pressure of arterial oxygen has also previously been shown to correlate poorly with pulmonary arterial pressure measured invasively.

In our study, left ventricular systolic dysfunction (LVSD) is present in 7.5% patients; in previous studies it was present in 4%–32% patients of COPD. LVDD was seen in COPD patients with normal pulmonary arterial pressure and it increased with right ventricular after load. In our study LVDD is present in 47.5% of patients, out of which 16 patients had PH and 3 did not have PH, various mechanisms might explain the presence of left diastolic dysfunction in COPD patients. This may be due to chronic hypoxemia leading to abnormalities of myocardial relaxation, lung hyperinflation, and distension leading to increased stiffness of the parietal pleura and thus of the wall of cardiac fossa leading to added load on ventricle, and also due to ventricular interdependence.

Our study shows that PHT is increasingly common with increasing severity of COPD. Data is currently limited and Mewat being a underdeveloped area the environment influences and life style influences are there.

Routine investigations of the presence of PAH are not currently part of UK and US guidelines for the diagnosis and management of COPD.

Conclusion: PAH is well correlated with severity of COPD. Dysfunction of both right and left t ventricles in COPD patients, simultaneously might lead to a state of refractory Heart failure. We suggest screening of all COPD patients for cardiac complications. This would contribute to the assessment of prognosis in these patients and assist in identifying individuals likely to suffer increased mortality and morbidity warranting close monitoring and intense treatment.

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Conflict of interest: None
Funding: None
Cite this Article as: Gupta R, Mann S. Correlation Between COPD And Echocardiographic Features With Severity Of Disease. Natl J Integr Res Med 2016; 7(1): 26-30