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Original research article

To assess the pulmonary arterial pressure in COPD patients by echocardiography and to find out the correlation between pulmonary arterial pressure and severity of COPD

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Abstract

To assess the pulmonary arterial pressure in COPD patients by echocardiography and to find out the correlation between pulmonary arterial pressure and severity of COPD.

Materials and Methods:A total 60 of patients of COPD were selected and staged by spirometry and pulmonary arterial pressure evaluated by echocardiography.

Results: On echocardiographic evaluation of COPD, 58.33% (35/60) cases had normal echocardiographic parameters. Pulmonary hypertension (PH), which is defined as systolic pulmonary arterial pressure (sPAP)> 30 mmHg was observed in 25/60 (41.66%) cases in which prevalence of mild, moderate, and severe PH were 14/25 (23.33%), 6/25 (10%), and 5/25 (8.33%), respectively. The frequencies of PH in mild, moderate, severe and very severe COPD were 5/26 = 19.23%, 7/15 = 46.66%, 8/13 = 61.53%, 4/6 = 66.66%, respectively.

Conclusion:41.66% patient of COPD have pulmonary hypertension and PH has a linear relationship with severity of COPD. Echocardiography helps in early detection of cardiac complications in COPD cases giving time for early interventions.

Keywords: Pulmonary hypertension, chronic obstructive pulmonary disease, echocardiography, cor pulmonale

Introduction

Chronic Obstructive Pulmonary Disease (COPD), a common preventable and treatable disease, is characterized by persistent airflow limitation that is usually progressive and associated with an enhanced chronic inflammatory response in the airways and the lung to noxious particles or gases. COPD is 3rd leading cause of mortality and 5th leading cause of morbidity worldwide in 2020[1]. Chronic obstructive pulmonary disease is more common in male compared with female HF patients [2] and in urban compared with rural areas[3].Pulmonary hypertension (PH), defined as an elevated mean pulmonary arterial pressure (mPAP) ≥30 mmHg, is a common complication ofCOPD.PH in COPD patients is closely associated with patient age and severity of airway obstruction [4]. Most stable COPD patients have mild to moderate PH with mPAP between 25 and 30 mmHg [4]. The exact incidence of clinically significant pulmonary hypertension, defined as pulmonary hypertension that contributes to symptomatology and prognosis,is difficult to estimate in COPD. A prevalence of 10-30% in patients with at least one previous hospitalization seems reasonable minimum.In patients with severe COPD PH prevalence of up to 60% has been reported [4, 5]. PH often progresses to right heart failure (RHF), with initial compensatory right ventricular (RV) hypertrophy becoming overwhelmed by increased systolic requirements, whilst left ventricular (LV) systolic function remains preserved. The term “cor pulmonale” has been used to

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describe this form of RHF and hypertrophy. It is a progressive condition, associated with increased mortality in COPD.

The pathophysiology of PH in COPD is multifactorial and includes hypoxic pulmonary vasoconstriction, pulmonary vascular remodeling, small vessel destruction, and fibrosis. Development of PH is associated with poor prognosis and may progress to RV remodeling, hypertrophy, dilatation and eventual right heart failure with associated increased mortality. The golden standard for diagnosis of PH is right heart catheterization, however, Echocardiography provides a rapid, noninvasive portable and accurate method to evaluate the right ventricle function, right ventricular filling pressure, tricuspid regurgitation, left ventricular function and valvular function[6]. Echocardiographically derived estimates of pulmonary arterial pressure co-relate closely with pressures measured by right heart catheter[7].

Aims and Objectives

1. To assess the Pulmonary Artery Pressure inCOPD patients by echocardiography.
2. To find out the correlation between Pulmonary Artery Pressure findings and the severity of COPD using GOLD guidelines.

Material and Methods

60 patients of COPD confirmed by Spirometry were selected from Department of Respiratory Medicine, Muzaffarnagar Medical College, Muzaffarnagar,UP.

All selected patients were subjected to routine investigations, including complete blood count, lipid profile, blood sugar, blood urea, serum creatinine, electrocardiography. All the patients were investigated by spirometry and diagnosed and classified according to GOLD guidelines (post bronchodilator FEV_1 /forced vital capacity (FVC) ratio < 70% predicted), mild ($FEV_1 \geq 80\%$ of predicted), moderate ($50\% \leq FEV_1 < 80\%$ predicted), severe ($30\% \leq FEV_1 < 50\%$ predicted) and very severe ($FEV_1 < 30\%$ predicted), respectively. All patients were subjected to resting two-dimension transthoracic Doppler echocardiography in the cardiology department of Muzaffarnagar Medical College, Muzaffarnagar.

Exclusion criteria

1. Patients with H/O of chronic lung disease other than COPD, hypertension, any primary cardiac disease, any systemic disease that can cause pulmonary hypertension.
2. Patients with poor echo window.
3. Patients who were unable to perform Spirometry.

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 maximumjet velocity was measured by continuous wave Doppler without the use of intravenous contrast. Right ventricular systolicpressure was estimated based on the modified Bernoulli equationand was considered to be equal to the sPAP in the absence ofright ventricular outflow obstruction: sPAP (mmHg) = right ventricular systolic pressure = trans-tricuspid pressure gradient (TTPG) + right atrial pressure (RAP), where trans-tricuspid gradient is $4v^2$ (v = peak velocity of tricuspid regurgitation, m/s)[8]. RAP was empirically estimated as 15 mmHg before 1997. Since1997, RAP was estimated to be 5, 10, or 15 mmHg based on thevariation 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[9].

Pulmonary hypertension (PH) was defined in this study as $sPAP \geq 30$ mmHg[10]. This value was chosen according to the definition of pulmonary hypertension. PH was classified into mild, moderate, and severe category as sPAP 30-50, 50–70,>70 mmHg, respectively (using Chemla formula, mean pulmonary arterial pressure (MPAP) =0.61 PASP + 2 mmHg and putting value of 25-35, 35-45 and>45 mmHg of MPAP for mild, moderate, and severe pulmonary hypertension, respectively)[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%).

FS (fractional shortening) = it is a percentage change in LV dimension with each LV contraction (28%-44%).

LV mass = left ventricular mass (88-224 g).

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-

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59 years), <1.0 (age group 60-69 years), and <0.8 (age group ≥70 years)[12].

Results

A total of 60 patients were recruited in our study and out of them, the number of patients with mild, moderate, severe and very severe COPD were 26/60 = 43.33%, 15/60 = 25%, 13/60 = 21.66% and 6/60 = 10%, respectively.

Table 1: Patient's classification according to severity of COPD

Severity of COPD	No. of patients	% Patients	No. of patients
Mild (FEV1 > 80% predicted)	26	43.33	26
Moderate (50% > FEV1<80% predicted)	15	25	15
Severe (30%< FEV1 < 50% predicted)	13	21.66	13
Very severe (FEV1 < 30% predicted)	6	10	6

Table 2

Echocardiography	No. of patients	% Patients
Normal	35	58.33
Pulmonary hypertension	25	41.66

On echocardiography patients (35/60 =58.33%) had normal study and PH defined as sPAP> 30 mmHg was observed in 25 patients (25/60 = 41.66% of the total study population. (Table 2).

Table 3

Pulmonary hypertension(sPAP> 30 mmHg)	No. of patients	% Patients
Mild (30-50 mmHg)	14	23.33%
Moderate (50-70 mmHg)	6	10%
Severe (>70 mmHg)	5	8.33%
Total	25	41.66

Out of those 25 patients with pulmonary hypertension, 14 patients were in mild PH (sPAP 30-50 mmHg) (14/60 = 23.33%), 6 were in moderate PH (sPAP 50-70 mmHg) (6/60 = 10%), and 5 were in severe PH (sPAP> 70 mmHg) (5/60 = 78.33%) [Table-3].

Table4: Frequency of PH with severity of COPD

Severity of COPD	% and number of patients with PH
Mild (26)	19.23% (6)
Moderate (15)	46.66%(7)
Severe (13)	61.53% (8)
Very severe (6)	66.66% (4)

Frequency of PH increases as severity of COPD increases.

The frequencies of PH in mild, moderate, severe and very severe COPD were 5/26 = 19.23%, 7/15 = 46.66%, 8/13 = 61.53%, 4/6 = 66.66%, respectively; thus we can see that there is a good co-relation between the frequency of PH and severity of COPD (Table-4).

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_2s (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

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peripheral demand on exercise.

Although the true prevalence of PH in COPD is unknown, an elevation of pulmonary arterial pressure is reported to occur in 20%-90% of patients when measured by right heart catheterization with some evidence that pulmonary hemodynamic worsens with worsening airflow obstruction[13].

Two studies have shown an abnormal increase in mean pulmonary arterial pressure (Ppa) in COPD of 0.4-0.6 mmHg per year. These studies illustrate that PH in COPD progresses slowly and occurs in mild as well as severe forms of disease[14, 15].

The level of PH has a prognostic value in COPD patients that has been demonstrated by several studies. In one of these studies, the 5-year survival rates were 50% in patients with mild PH (20-30 mmHg), 30% in those with moderate-to-severe PH (30-50 mmHg), and 0% in the small group of patients with very severe PH (>50 mmHg). Thus a high degree of PH bears a poor prognosis, and this also has been observed in COPD patients receiving long-term oxygen therapy[16].

The present study finding reveals 41.66% patients of various severity of COPD have findings of pulmonary hypertension that is similar in prevalence of previous studies. The frequencies of PH in mild, moderate, severe and very severe COPD were 5/26 = 19.23%, 7/15 = 46.66%, 8/13 = 61.53%, 4/6 = 66.66%, respectively.

In one study it was found to be 25%, 43% and 68% in mild, moderate and severe COPD, respectively[17].

In our study it is also observed that severe PH is present only in severe or very severe COPD. In conclusion, the incidence of PH is directly proportional to severity of disease. Previous studies showed the frequencies of severe PH in COPD from about 1%-3%[18,19], but in our study it is 8.33%; this may be due to small study population comprising more percentage of severe and very severe COPD patients.

Cor pulmonale is present in 17.5% of patients in our study. Approximately 25% patients with COPD eventually develop cor pulmonale[20]. Cor pulmonale was found in 40% patients with COPD in one autopsy study[21, 22].

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.(23] LVDD was seen in COPD patients with normal pulmonary arterial pressure and it increased with right ventricular after load[24]. 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.

Conclusion

To conclude, our study shows high prevalence of pulmonary hypertension in COPD and frequency of PH increases as severity of COPD increases. Therefore screening of all COPD patients for cardiac complications should be done. 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 declared.

References

1. World Health Report. Geneva: World Health Organisation. 2000. Available from: http://www.who.int/whr/2000/en/statistics.htm. [Last accessed on 2004 Jan
2. Macchia A, Monte S, Romero M, D'Ettorre A, Tognoni G. The prognostic influence of chronic obstructive pulmonary disease in patients hospitalised for chronic heart failure. Eur J Heart Fail. 2007.
3. Taubert G, Bergmeier C, Andresen H, Senges J, Potratz J. Clinical profile and management of heart failure: rural community hospital vs. metropolitan heart center. Eur J Heart Fail. 2001;3:611-617.
4. WeitzenblumE, ChaouatA, CanuetM, KesslerR. Pulmonary hypertension in chronic obstructive pulmonary disease and interstitial lung diseases,Seminars in Respiratory and Critical Care Medicine.2009;30(4):458-470.
5. Naeije R,Mac NeeW.Pulmonary Circulation.In:CalverleyP,Mac NeeW, Pride P, Rennard S, editors. Chronic obstructive pulmonary disease, 2nd edition. London: Arnold Health Sciences, 2003, 228-242.
6. Daniels LB, Krummen DE, Blanchard DG. Echocardiography in pulmonary vascular disease. Cardiol Clin. 2004;22:383-99.
7. Tramarin R, Torbicki A, Marchandise B, Laaban JP, Morpurgo M. Doppler echocardiographic evaluation of pulmonary artery pressure in chronic obstructive pulmonary disease.A European

ISSN:0975-3583,0976-2833 VOL13, ISSUE04,2022

multicentre study. Eur Heart J. 1991;12:103-11.
8. Chan KL, Currie PJ, Seward JB, Hagler DJ, Mair DD, Tajik AJ. Comparison of three Doppler ultrasound methods in the prediction of pulmonary artery pressure. J Am Coll Cardiol. 1987;9:549-54.
9. Bredikis AJ, Liebson PR. The echocardiogram in COPD: Estimating right heart pressures. J RespirDis. 1998;19:191-8.
10. Rappaport E. Cor pulmonale. In: Murray JJ, Nadel JA, Mason RM, Boushey H, editors. Textbook of respiratory medicine. 4th Edition. Philadelphia: W.B. Saunders, 2000, 1631-48.
11. Chemla D, Castelain V, Humbert M, Simonneau JLHG, Lecarpentier Y, Hervé P. New Formula for Predicting Mean Pulmonary Artery Pressure Using Systolic Pulmonary Artery Pressure. Chest. 2004;126:1313-17.
12. Libby P, Bonow RO, Zipes DP, Mann DL, editors. 8 th. Philadelphia: Saunders. Braunwald's Heart Disease,2008, 251.
13. Weitzenblum E, Sautegeau A, Ehrhart M, Mammosser M, Hirth C, Roegel E. long term course of pulmonary artery pressure In chronic COPD. Am Rev Respir Dis. 1984;130:993-8.
14. Weitzenblum E, Sautegeau A, Ehrhart M, Mammosser M, Pelletier A. Long-term oxygen therapy can reverse the progression of pulmonary hypertension in patients with chronic obstructive pulmonary disease. Am Rev Respir Dis. 1985;131:493-8.
15. Kessler R, Faller M, Weitzenblum E, Chaouat A, Aykut A, Ducolone'A, *et al*. Natural history of pulmonary hypertension in a series of 131 patients with chronic obstructive pulmonary disease. Am J RespirCrit Care Med. 2001;164:219-24.
16. Oswald-Mammosser M, Weitzenblum E, Quoix E, Moser G, Chaouat A, Charpentier C, *et al*. Prognostic factors in COPD patients receiving long-term oxygen therapy. Chest. 1995;107:1193-8.
17. Higham MA, Dawson D, Joshi J, Nihoyannopoulos P, Morrell NW. Utility of echocardiography in assessment of pulmonary hypertension secondary to COPD. EurRespir J. 2001;17:350-5.
18. Chaouat A, Bugnet AS, Kadaoui N, Schott R, Enache I, Ducoloné A, *et al*. Severe pulmonary hypertension and chronic obstructive pulmonary disease. Am J Respire Care Med. 2005;172:189-94.
19. Thabut G, Dauriat G, Stern JB, Logeart D, Lévy A, Marrash-Chahla R, *et al*. Pulmonary Haemodynamics in advanced COPD candidate for lung volume reduction surgery or lung transplantation. Chest. 2005;127:1531-6.
20. Springhouse Respiratory disorders. In, Springhouse (ed). Professional Guide to Diseases. Philadelphia, Lippincott William and Wilkins. (9th), 2008,120.
21. Rigolin VH, Robiolio PA, Wilson JS, Harrison JK, Bashore TM. The forgotten chamber: The importance of the right ventricle. Cathet Cardiovasc Diagn. 1995;35:18-28.
22. Fishman AP. State of the art: Chronic Cor Pulmonale. Am Rev Respire Dis. 1976;114:775-94.
23. Louridas G, Patakas D, Stavropoulos C. Left ventricular function in patients with chronic obstructive pulmonary disease. Cardiology. 1981;67:73-80.
24. Funk GC, Lang I, Schenk P, Valipour A, Hartl S, Burghuber OC. Left Ventricular Diastolic Dysfunction in Patients With COPD in the Presence and Absence of Elevated Pulmonary Arterial Pressure. Chest. 2008;133:1354-9.