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Original Article

Correlation Between FEV1 (Forced Expiratory Volume in First Second) and Pulmonary Arterial Pressure among Patients of Chronic Obstructive Pulmonary Disease

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Abstract

Background: To determine the relationship of Forced Expiratory Volume in first second (FEV1) with pulmonary arterial pressure among patients with chronic obstructive airway disease.

Methods: In this cross sectional study 276 patients, using -0.17 correlation between FEV_1 and pulmonary artery pressure with 95% confidence level and 80% power of test, were enrolled . All patients 40 years or above, diagnosed with COPD of minimum duration 3 years were included. Doppler Echocardiography for the measurement of pulmonary arterial pressure was done. All the patients were also subjected to spirometery for the measurement of forced expiratory volume in one second. Spearman's RANK correlation test was applied to see the relationship between FEV_1 and pulmonary arterial pressure. P value of < 0.05 was considered significant.

Results: Mean age was 63.94±10.11 years. Mean duration of COPD was 13.19±5.15 years. The mean FEV1 (percent predicted) was 32.84±13.43. Among these 276 patients, 133 (48%) patients had Pulmonary Hypertension (PH). The mean PAP was 61.99 ± 14.33. The correlation was calculated between FEV1 and PAP by using Pearson Correlation Coefficient and was found to be –0.406 (p=0.000).

Conclusion: Increase in severity of COPD (as measured by falling FEV1) is strongly correlated with increasing severity of pulmonary artery pressure and thus warrants echocardiographic screening of COPD cases with mild to very serious disease.

Key Words: Chronic obstructive airway disease(COAD), Pulmonary hypertension(PH), Forced expiratory volume in one second(FEV1).

Introduction

Chronic Obstructive airway disease (COAD) is considered as the 4th major cause for death and it is estimated to become the most common disease affecting mankind by year 2020. One established complication of COPD is the development of pulmonary hypertension (PH).[1] The pathophysiology involves hypoxic pulmonary vasoconstriction which results in remodeling and narrowing of lumen of vessels. Under these circumstances right side of heart need to increase the pumping pressure in order to overcome the pulmonary vascular resistance. As this pathology worsens, a condition develops which is called"cor-pulmonale". When patients develop this condition the mortality and morbidity increases and five-year survival ranges from 20% to 36%. Apart from the disease process itself, there is also contribution of worsening pulmonary hypertension and increasing age.[2]COAD is considered one of the most common cause of pulmonary hypertension worldwide.[3] Frequency of pulmonary hypertension among these cases has been reported around 53% in literature.[4] So far the accepted definition of pulmonary hypertension is pulmonary artery pressure (PAP) of more than 25 mm Hg along with a pulmonary capillary wedge pressure (PCWP)<15 mm Hg, LA (left atrial) pressure or left ventricular (LV) end diastolic pressure of less than15 mm Hg.[5]

Right ventricular catheterization is considered to be a gold standard method for diagnosing the PH (pulmonary hypertension). Transthoracic echo-cardiography is very useful and relatively noninvasive technique for measuring pulmonary arterial pressure as it is frequently used for screening and monitoring disease progression. It is widely available, cheaper with sensitivity and specificity of 83% and 72% respectively.[6] Although medical diagnostics and therapeutics are improving day by day treating PH still remains a therapeutic challenge with increased

mortality and morbidity as having one year survival rate of 53%. A patient who has advanced disease and is not a candidate for medical therapy, an alternate option is lung transplant.[7]Pulmonary arterial pressure showed an inverse relationship with FEV1but no relationship with resting PO2. FEV_1 has been shown to be strongly negatively correlated with pulmonary artery pressure (r = -0.63)[8]but in another study it showed insignificant correlation (*r* =-0.17).[8,9]

Patients and Methods

This cross sectional study was done over six months period at Fauji Foundation Hospital Rawalpindi. Sample size was 276 patients, using -0.17[8] correlation between FEV_1 and pulmonary artery pressure with 95% confidence level and 80% power of test. All patients 40 years or above, diagnosed COPD with minimum duration of 3 years of both gender were part of the study. Conditions mimicking COPD like asthma, bronchiectasis and congestive heart failure were excluded. Conditions causing pulmonary artery hypertension other than COPD like interstitial lung diseases and primary pulmonary hypertension were also excluded**.** The Doppler Echocardiography for the measurement of pulmonary arterial pressure was done for all patients in the radiology department. All the patients were also subjected to spirometery for the measurement of forced expiratory volume in one second. Data was entered and analyzed by SPSS version 15.0. Frequency / percentage were calculated for qualitative variable like gender. Mean + standard deviation of quantitative variable like age was calculated. Spearman's RANK correlation test was applied to see the relationship between FEV_1 and pulmonary arterial pressure. P value of < 0.05 was considered significant.

Results

In this study a total of 276 patients were observed to determine the correlation between Forced Expiratory Volume in first second and Pulmonary Artery Pressure in patients with Chronic Obstructive Pulmonary disease. Thirty two (11.59%) patients were in age range 40-50 years, 107(38.76%) patients were in age range 51-60 years, 84(30.43%) patients were in age range 61-70 years, 46(16.66%) patients were in age range 71-80 years and 7(2.53%) patients were above 80 years of age. Mean age was 63.94 years with SD ±10.11.One hundred and twenty six (45.6%) patients were male while 150(54.3%) patients were female. Eleven 11(4%) had COPD from 4-6 years,105(38%) had COPD from 7-10 years and 160(58%) had COPD for> 10 years. Mean duration of COPD was 13.19 years with SD ± 5.15. Three (1%) had mild FEV_1 (more than 80%),27(10%) had moderate FEV_1 (50-79%), 102(37%) had severe FEV_1 (from 30-49% and 144(52%) had very severe FEV_1 (< 30%). Mean FEV_1 was 32.84% with standard deviation ± 13.43. Mean FEV_1 was 32.84 with SD± 13.43. Association of PAP by using Pearson Correlation Coefficient and was found to be –0.406 (P=O.OOO) (Table 1). One hundred and thirty three patients had PAP in which n=17(13%) patients had mild PAP ranged from 30-50 mmHg, n=85(64%) patients had moderate PAP 51-70 mmHg) and 31(23%) patients had severe PAP (more than 70 mmHg). Mean PAP was 61.99 ± 14.33mmHg . Status of FEV1 findings in patients with raised Pulmonary arterial pressure was analyzed among 133 patients having PAH; 8(6%) had moderate COPD(FEV1 ranged from 50-79),44(33%) had severe COPD (FEV1 ranged from 30-49) and 81(61%) had very severe COPD (Table 2).No patient with mild COPD had PAH and as the severity of COPD increases the frequency of patients without PAH decreases.

Table 1: Correlation between FEV1 and PAP(n=133)

		Forced Expiratory Volume in First second	Pulmonary Artery Pressure
FEV1	Pearson Correlation Sig. (2-tailed) N	1.OOO 276	-O.406 O.OOO 133
PAP	Pearson Correlation Sig. (2-tailed) N	-0.406 O.OOO 133	1.000 133

Table 2: Forced expiratory volume in first Second-pulmonary artery pressure Cross tabulation(n=133)

		Pulmonary Artery Pressure			Total
		Mild	Moderate	Severe	
Forced expiratory volume in first second	Moderate		7	1	8
	Severe	8	24	12	44
	Very severe	9	54	18	81
Total		17	85	31	133

Discussion

Chronic obstructive airway disease is now considered to be an underestimated common public problem all over the world. It is well proven that it can be prevented and its morbidity and mortality can be decreased easily. In USA and Canada the incidence of chronic airway disease is around 3% to 17% while in most of underdeveloped world the disease incidence is

much greater and range from 13% to 27%.[10] The prevalence of COAD with combination of its mortality and morbidity was found twelfth highest level worldwide in 1990 and it is estimated to rise at level third by year 2020, with five times more deaths than in year 1990.[11] The effects of COAD on heart are contributing in worse prognosis of these patients. COAD effects the functioning of right ventricle and atrium which leads to increase in pulmonary vascular resistance and gradually this condition complicates the disease process of COAD which co-relates inversely with survival. There are a lot of structural abnormalities found in pulmonary vasculature among patients with COAD.[12] As the arterial oxygen concentration decreases it results in increased thickening of intima and hypertrophy of vascular wall of small branches of the pulmonary arteries. Along with these changes in pathology there is pulmonary vascular constriction which arises due to alveolar hypoxia, damage to pulmonary vasculature bed, change in the intrinsic vasodilators in lungs (such as decrease in prostacyclin synthase, endothelial nitric oxide synthase and an increase in endothelin 1 will lead to remodeling of vessels, an increase of blood viscosity, and changes in respiratory cycle. All these changes will result in lung arterial vessel resistance which results in an increase in pulmonary hypertension.[13]

Severe pulmonary hypertension results in increase of right ventricular pressures which lead to rise in right ventricular workload resulting in right ventricular hypertrophy. In patients with COAD this pulmonary vascular constriction leads to increased right ventricular mass but also result in dilatation of right ventricular which ultimately leads to clinical condition of cor-pulmonale.[14] Although the exact incidence of pulmonary hypertension in patients with COAD is not well known, elevated pulmonary vascular pressure is described to occur in 20%–90% of cases by measuring with right heart catheterization along with evidence of worsening pulmonary hemodynamic with increasing airflow obstruction. In our study there was slight female predominance with 54.3% females affected and 45.6% males affected. This difference in gender distribution of COPD was due to lower socioeconomic status of families with more use of biofuel mass for cooking and heating processes and lack of proper ventilation and education.

Data of present study reveals that majority of patients got admitted with severe to very severe COPD while patients with mild to moderate diseases were usually treated as OPD cases. On the other hand 133 patients had PH out of which 17(13%), 85(64%) and 31(23%) had mild (PAP=30 to 50 mmHg), moderate (PAP=50 to 70mmHg) and severe PH (PAP > 70 mmHg) respectively. Our study shows that FEV_1 is inversely proportional to PAP. The correlation coefficient between FEV1 and PAP turned out to be r= -0.406 (p=0.000) suggesting that as disease gets severe (marked by falling FEV1) pulmonary artery pressure rises. Similar findings were observed in other studies done by Fishman A et al [14] in which FEV1 had shown to be strongly negatively correlated with pulmonary artery pressure (r = -0.63) in another study done by Thabut G et al [15]shows insignificant correlation (r = -0.17). [15]

In our study it was found that abnormal rise in mean pulmonary vascular pressures leading to development of pulmonary hypertension was seen only in severe cases of COAD, which also corresponds with international literature that incidence and severity of pulmonary hypertension is directly proportional to severity of disease. Previous studies showed the frequencies of severe PH in COPD from about 1%–3%[16],but in our study it is 11% (n=31) out of the total study population. [16] Out of these 11%, 4.7% (n=13) had left heart failure while 1% (n=3) had chronic thromboembolic disease, which are additional causes of PH, and only 5.4% (n=15) had severe PH secondary to COPD as the only cause for severe PH. In our study no case with mild COPD had PH. In addition severe PH (PAP>70mmHg) is found predominantly in cases with severe and very severe COPD. These data suggest that if PH is found in mild cases of COPD and if severe PH registered in cases with moderate COPD then such cases should be evaluated further for additional causes of PH such as idiopathic PH or chronic pulmonary thromboembolism disease etc. In our study right ventricular failure was seen in 28% of patients while in literature about 25% patients with COAD eventually develop cor- pulmonale. It is well established that about 10 to 30 percent of hospital cardiac admission in USA every year are due to cor-pulmonale and about 85% patients with cor-pulmonale have COAD. Although there are some studies which indicate that left ventricular function remains normal in persons with COAD, most of literature suggest that it is definitely present.[17] In present study, dysfunction of left ventricle was found in 7.5% patients while in previous literature it was found in about 14 to 28% patients of COAD.[18] In another study left ventricular diastolic dysfunction was found in COAD patients with normal pulmonary vascular pressure and it was found to increase with

right ventricular afterload, while in our study LV dysfunction was present in about 47.5% patients, out of which only 16% patients had pulmonary hypertension. [19] There are various pathologies which might explain presence of left ventricular diastolic dysfunction in COAD patients like chronic hypoxemia leading to abnormal myocardial relaxation, lung hyperinflation and distension which leads to increased stiffness of the parietal pleura as well as wall of cardiac fossa leading to added load on ventricle and also due to ventricular interdependence. In our research LV hypertrophy was there in about 22% patients while in one previous study LVH was found in 25% patients who also had right ventricular hypertrophy.[19]

Conclusion

1.Increase in severity of patients with chronic obstructive airway disease (COAD) as measured by a fall in FEV1 is strongly correlated with increasing severity of pulmonary artery pressure with eventual increased morbidity and mortality.

2.Right ventricular dysfunction is present in majority of these patients with severe COAD with or without some element of left ventricular dysfunction.

3.Findings of present study warrants echocardiographic screening of COPD patients with moderate to very severe diseases.

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