PULMONARY ARTERIAL HYPERTENSION IN CHRONIC OBSTRUCTIVE PULMONARY DISEASE

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ABSTRACT

Background: Pulmonary arterial hypertension is a common complication particularly of advanced chronic obstructive pulmonary disease (COPD). It is directly related to reduced exercise tolerance and mortality. We conducted this study to determine the frequency of pulmonary arterial hypertension in COPD and its correlation with disease severity in hospitalized patients.

Methods: This was a descriptive study conducted at Pulmonology Unit, Khyber teaching hospital Peshawar from 1st Sept 2016 till 28th Feb 2017. All admitted patients with spirometry proven COPD were included. Patients with respiratory distress, hemodynamic instability, left heart failure or mild COPD were excluded. After consent; age, gender and FEV1 were recorded. Pulmonary arterial hypertension (PAH) was defined as a right ventricular systolic pressure (RVSP) of >25mm of Hg obtained from echo of all patients done by a cardiologist. The severity of COPD was measured by FEV1 (spirometry).The data was analyzed via spss 19. Mean age and FEV1 of all patients along with percentage of pulmonary hypertension were calculated. Correlation was determined between the values of FEV1 and Pulmonary artery pressure using spearman's correlation. The results were presented as table/graph.

Results: A total of 112 patients were recruited with mean age of 62 (±15.23 SD) years and males constituted 47%. The mean FEV1 was 34(±17 SD) % predicted and the prevalence of PAH was 38.46%. There was a statistically significant positive correlation between the severity of COPD (FEV1) and presence of pulmonary arterial hypertension (r=0.238).
Conclusion: Pulmonary arterial hypertension is a common complication of COPD and should be investigated particularly in patients with advanced COPD.

Key words: COPD, Prevalence, Correlation, Pulmonary arterial Hypertension.

INTRODUCTION

Constant obstructive pneumonic illness (COPD) is the fourth driving reason for death on the planet and a significant reason for ongoing morbidity.1 Although, principally it is a respiratory issue described via wind stream impediment, it has fundamental appearances as well. Pneumonic blood vessel hypertension (PAH) is a typical extra aspiratory intricacy of this illness and is a free indicator of mortality.2 The 5-year endurance rate is just 36% in patients with ordinary pneumonic vein pressure (PAP) contrasted with 62% in those with aspiratory blood vessel hypertension 3.

PAH in COPD has been fluidly characterized as resting mean pneumonic vein pressure (mPAP) > 20–25 mm Hg.4The pathogenesis of PAH in COPD is ineffectively perceived and is probably going to be multifactorial. In spite of the fact that hypoxia has been portrayed to be the principle pathogenic specialist and long haul oxygen prompts rever-sal yet not all out standardization of the pneumonic corridor pressure (PAP) 5.The focal upgrade to these progressions stays long haul presentation of aviation routes to poisonous stimuli on the grounds that morphologic changes in aspiratory vessels have been seen in corresponding with lung parenchymal changes in mellow to direct COPD without constant hypoxia.6 New advances in the pathogenesis of pneumonic hypertension auxiliary to COPD sup-port an endothelium-determined vasoconstrictor-dilator awkwardness, predominantly from a diminished endothelial nitric oxide articulation, expanded vascular endothelial development factor and serotonin carrier expressions.7

Right heart catheterization being the gold stan-dard test to affirm the determination of PAH and its seriousness, its intrusive nature blocks is basic use.8 Conven-tionally transthoracic Echocardiogram is performed to gauge right ventricular systolic weight which is a substitute marker of pneumonic blood vessel hypertension with a Sensitivity, particularity, negative prescient worth (NPV), and positive prescient worth (PPV) as 76, 65, 93, and 32%, individually 9. PAH in stable COPD is generally mellow to direct and is typically not seen until the illness is genuinely best in class (FEV1 < half). Serious PAH (mPAP > 35–45 mm Hg) is uncommon (3%–13%) and should incite a quest for an extra reason for PH, for instance, left coronary illness, obstructive rest apnea (OSA), pneumonic embolism (PE).10literature shows that PAH in COPD affects mortality, morbidity and natural course of the disease, and a disproportionately high right ventricular systolic pres- sure in relatively milder form of disease may warrant a

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look for extra reasons for PAH, we arranged this investigation to have a nearby proof for commonness of PAH in our patients with COPD and its connection with the seriousness of illness which might be of significant worth in figuring rules for dealing with this subset of patients.

Objective

To determine the frequency of pulmonary arterial hypertension in chronic obstructive pulmonary disease (COPD) and its correlation with disease severity in hospitalized patients.

METHODS

This was a descriptive study conducted at Pulmonology unit, Khyber teaching hospital Peshawar from 1st Sept 2016 till 28th Feb 2017. All adult patients including both genders with spirometry proven COPD admitted due to acute exacerbation were included. Patients with respiratory distress, hemodynamic instability, mild COPD (FEV1 >80% predicted) or left heart failure were excluded. After consent and appropriate management of these patients; age, gender and spirometry findings of FEV1 (forced expiratory volume in 1st second) were recorded. Echocardiogram was done by a cardiologist for all patients when clinically stable and the results of RVSP (right ventricular systolic pressure) were obtained. RVSP was taken as an estimation of pulmonary artery pressure and a value of more than 25mm of Hg was taken as PAH (pulmonary arterial hypertension). The severity of COPD was measured by FEV1 (spirometry done via spirolab-III machine by a trained technician). FEV1 of >50% predicted was defined as moderate COPD, while 30-50% as severe disease and <30% as very severe disease. The data was analyzed via spss 19. Mean was calculated for age and FEV1 while percentage was determined for pulmonary arterial hypertension. Correlation was determined between FEV1 and PAH using spearman's correlation. The results were presented as table/graph.

RESULTS

A total of 112 patients were recruited with mean age of 62 (±15.23 SD) years and males constituted 47%. The prevalence of PAH was 38.46% as shown in figure 01. The prevalence of PAH in patients having very severe, severe and moderate COPD was 51%, 37.7% and 0% respectively as shown in table 01. There was a statistically significant positive correlation between the severity of COPD (FEV1) and presence of pulmonary arterial hypertension (r=0.238).

DISCUSSION

The advancement of aspiratory blood vessel hypertension in COPD is straightforwardly identified with mortality in light of the fact that an expansion in the mean pneumonic conduit weight of 10 mm Hg builds the demise rate by more than four fold.11 Pulmonary hypertension is typically of mellow to direct

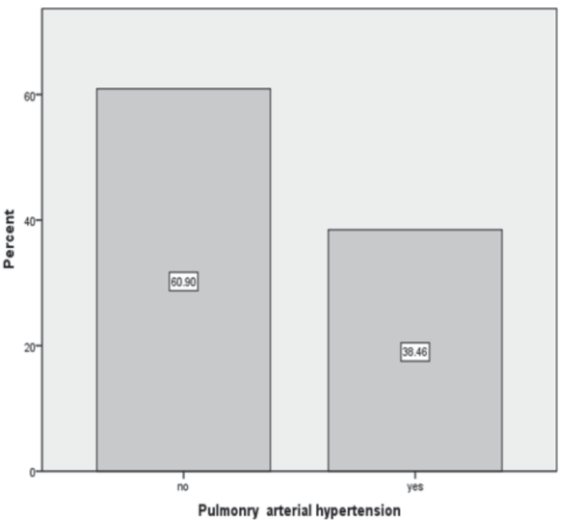

Figure 1: Prevalence of PAH in patients with COPD.

Table 1: Percentage of PAH according severity of COPD

Severity of COPD	FEV1 (% Predicted)	Number of patients	PAH (%)
Moderate	>50	14	00%
Severe	30-50	53	37.7% (n=20)
Very severe	<30	45	51% (n=23)

PAH: Pulmonary blood vessel hypertension. COPD; ongoing obstructive pneumonic sickness. FEV1; Forced expiratory volume in first second seriousness and is more normal ahead of time phases of COPD; notwithstanding, a little extent of COPD patients may give "messed up" aspiratory hyper-strain, with a moderately safeguarded lung function.12 OpenUrlThe predominance of pneumonic hyperten-sion in COPD has been accounted for by one investigation as 16.67 %, 54.55 %, 60.00 %, and 83.33 %, in gentle, moderate, extreme, and serious COPD respectively.13 In anoth-er study, the recurrence of PAH was discovered to be 25 %, 43 %, and 68 % in mellow, moderate, and extreme COPD, respectively.14 We found the pervasiveness of aspiratory hypertension in serious and serious COPD as 37% and 51% individually. The various outcomes presumably rely upon the meaning of PAH (mPAP > 20 versus >25 mm Hg), the seriousness of COPD, test determination standards, and the strategy for estimating the aspiratory conduit pressure (echocardiography versus right heart catheterization).15

We found a significant correlation between sever- ity of COPD as measured by FEV1 and the presence of pulmonary arterial hypertension. One explanation for this correlation could be that more severe and persistent

wind current constraint will prompt more serious hypoxia which is one of the significant supporters of the pathogenesis of Pulmonary blood vessel hypertension. Writing proposes that aspiratory blood vessel hypertension in COPD progress-es over the long haul and its seriousness connects with the level of wind stream check and the hindrance of pneumonic gas exchange.16,17 Pulmonary vascular renovating in COPD is the fundamental driver of expansion in pneumonic conduit pressure and is considered to result from the consolidated impacts of persistent hypoxemia, aggravation, endothelial brokenness and direct impacts of tobacco smoke on vessel structure. As of late a gathering of patients with serious PAH (mPAP > 40 mm Hg) and incredibly helpless anticipation has been perceived. The 5-year endurance is 15% (mPAP >40 mm of Hg) versus 55% in those with less extreme PH (mPAP 20–40 mm Hg). Such patients are character-ized by mellow to direct aviation route block, an exceptionally low diffusing limit, extreme hypoxemia, and hypocapnia. Be that as it may, such extreme PAH in COPD without an elective clarification is uncommon (1–3.7%) and recommends the presence of a "vascular aggregate" or attendant idiopathic aspiratory blood vessel hypertension.18 In perspective on the antagonistic impacts of pneumonic blood vessel hypertension on dismalness and mortality routine echocardiography in patients with serious constant obstructive aspiratory infection might be justified.

CONCLUSION

Pulmonary arterial hypertension is a common complication of COPD and should be investigated particularly in patients with advanced (severe and very severe) COPD.

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