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Original Research Article

CORRELATION OF SPIROMETRY AND ARTERIAL BLOOD GAS ANALYSIS WITH THE DEVELOPMENT OF PULMONARY HYPERTENSION IN PATIENTS WITH COPD: A PROSPECTIVE OBSERVATIONAL STUDY

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ABSTRACT

Background: Chronic obstructive pulmonary disease (COPD) is characterized by persistent respiratory symptoms and airflow limitation. Pulmonary hypertension (PH) develops insidiously and is an established complication that eventually leads to right ventricular hypertrophy, dilatation and failure. Its early detection in COPD is a diagnostic challenge.

Materials and Methods: This prospective, analytical study was conducted in the Department of Respiratory Medicine of a tertiary-care teaching hospital from November 2017 to October 2019. A total of 138 COPD patients attending the OPD who fulfilled the inclusion criteria were enrolled; patients in acute exacerbation or requiring hospitalization were excluded. After clinical examination, spirometry was performed and severity of obstruction graded according to GOLD 2019 (FEV1). All patients simultaneously underwent arterial blood gas analysis (with grading of hypoxia by PaO2) and 2D-echocardiography, in which pulmonary hypertension was assessed from pulmonary artery systolic pressure derived from the tricuspid regurgitation jet.

Results: Of the 138 patients, 94 (68.1%) were men and 44 (31.9%) were women; men were predominantly smokers or ex-smokers and women had biomass fuel exposure. Airflow obstruction was mild in 10 (7.2%), moderate in 38 (27.5%), severe in 52 (37.6%) and very severe in 38 (27.5%). Hypoxia was present in 121 patients (mild 59, moderate 56, severe 6). Overall, 61 (44.2%) patients developed pulmonary hypertension. The prevalence of pulmonary hypertension increased with the severity of airflow obstruction and hypoxia; mild pulmonary hypertension predominated, but the proportion with moderate-to-severe pulmonary hypertension was higher among patients with more severe obstruction. Among the 17 patients without hypoxia, 6 also developed pulmonary hypertension.

Conclusion: The development of pulmonary hypertension in COPD is directly related to the severity of airflow obstruction and hypoxia. However, the severity of pulmonary hypertension does not correlate with the severity of obstruction and hypoxia in every patient. Spirometry and arterial blood gas analysis can help identify patients at higher risk.

Keywords: COPD, Pulmonary hypertension, Spirometry, Arterial blood gas analysis, Echocardiography.

INTRODUCTION

Chronic obstructive pulmonary disease (COPD) is a common, preventable and treatable disease that is characterized by persistent respiratory symptoms and airflow limitation due to airway and/or alveolar abnormalities usually caused by significant exposure to noxious particles or gases.[1] Tobacco smoking is

the most commonly encountered risk factor for COPD worldwide. In many countries, outdoor, occupational and indoor air pollution — the latter resulting from the burning of biomass fuels — are also major COPD risk factors. Assessment of COPD is based on the patient's symptoms, risk of exacerbations, the severity of the spirometric abnormality, and the identification of comorbidities.[1] About 10% of the adult male population of India and up to 6.5% of the population of Asia-Pacific countries suffer from this disease.[2]

Spirometry is necessary for diagnosis in this clinical context and is the most frequently used investigation, as it gives the measure of lung function as a volume against time. Spirometry and the calculation of the ratio of FEV1/FVC help in identifying obstructive or restrictive ventilatory defects; a FEV1/FVC ratio of less than 70%, where the reduction in FEV1 is more than that of FVC, signifies an obstructive defect.

The natural history of the disease is characterized by progressive decrements in expiratory airflow and increments in end-expired pulmonary volume which develop over several decades. Although deterioration of gas exchange eventually occurs, the extent of arterial hypoxemia or hypercapnia is quite variable and often poorly correlated with the severity of airflow obstruction. Alveolar hypoxia stimulates resistance pulmonary arteries to constrict and, if sustained, further induces pulmonary vascular remodeling. As the disease progresses, dyspnea becomes manifest at progressively lower levels of exertion, and pulmonary hypertension develops insidiously. Eventually, the long-standing and frequently undetected pulmonary hypertension leads to the development of right ventricular hypertrophy, dilatation and failure,[3] although hemodynamic deterioration occurs slowly over a period of years.[4] The fact that pulmonary hypertension develops insidiously, producing few diagnostic clues, makes the early detection of pulmonary hypertension in COPD a diagnostic challenge.[5]

Pulmonary hypertension (PH) is a hemodynamic and pathophysiological condition defined as an increase of mean pulmonary pressure of more than or equal to 25 mmHg at rest.[6] PH is most often related to the occurrence of heart disease and lung diseases (secondary), while it rarely occurs as a primary disorder of the pulmonary vasculature — up to 5%. In COPD, pulmonary hypertension may develop late in the course of the disease and is mainly due to hypoxic vasoconstriction of the small pulmonary arteries, eventually resulting in structural changes that include intimal hyperplasia and late smooth-muscle hypertrophy/hyperplasia.[7]

Echocardiography can be a helpful tool in detecting the etiology of suspected or confirmed PH. The routine echocardiographic approach to estimate pulmonary artery systolic pressure (PASP) is by derivation of right ventricular pressure from the tricuspid regurgitation (TR) velocity, which adds to a qualitative assessment of right atrial pressure. Previous studies show good correlation across patient populations, but the precision of absolute PASP values calculated from TR velocity is moderate,[8] and in an individual patient this is of importance, as significant over- and under-estimation can occur, leading to misdiagnosis and inappropriate treatment.[9] The standard respiratory function test for case detection of COPD remains spirometry, with the criterion for diagnosis being guidelines based on FEV1/FVC and severity based on FEV1.[10] The present study was undertaken to find out whether any correlation exists between spirometry and arterial blood gas findings in patients of COPD and their association with the development of pulmonary hypertension.

MATERIALS AND METHODS

This prospective, analytical study was conducted in the Department of Respiratory Medicine at a tertiary-care teaching hospital over a period of two years, from April 2024 to March 2026. Approval was obtained from the institutional ethics committee prior to commencement, and written informed consent was obtained from all patients before enrolment. Patients of COPD attending the outpatient department who fulfilled a predefined set of inclusion and exclusion criteria and gave consent were consecutively recruited during the study period.

The sample size was calculated using the formula $N = z^2p(1-p)/d^2$, taking the prevalence (p) of COPD as 10%, z as 1.96 corresponding to a 95% confidence interval, and the allowable error (d) as 5%. This yielded a minimum required sample size of 120 patients. During study period we included 138 patients on the basis of a predefined inclusion and exclusion criteria.

All patients underwent a detailed clinical evaluation at the time of presentation using a structured study proforma. This included documentation of demographic characteristics, presenting respiratory complaints (cough, expectoration, breathlessness, wheeze and fever), treatment history and past history of systemic illness. Personal and occupational history was recorded with particular attention to the etiological exposure, namely cigarette or bidi smoking (with the smoking index and number of smoking years) and biomass fuel exposure. General and systemic examination, including pulse rate, respiratory rate, blood pressure and oxygen saturation, was performed in all cases.

Spirometry was performed after clinical examination, with patients asked to take a maximal inspiration and then forcefully expel air for as long and as quickly as possible; the forced expiratory volume in one second (FEV1), forced vital capacity (FVC) and the FEV1/FVC ratio were recorded, an FEV1/FVC ratio of less than 70% signifying an obstructive defect. Severity of obstruction was graded on the basis of FEV1 according to the GOLD 2019 guidelines as mild (FEV1 $\geq$ 80), moderate (FEV1 $\geq$ 50 to $<$ 80),

severe (FEV1 ≥ 30 to < 50) or very severe (FEV1 < 30).

Simultaneously, all patients underwent arterial blood gas (ABG) analysis and two-dimensional echocardiography. Hypoxia was graded from the PaO2 on ABG taken on room air (normal 80–100 mmHg) as mild (PaO2 60–80 mmHg), moderate (PaO2 40–60 mmHg) or severe (PaO2 < 40 mmHg). Pulmonary hypertension was assessed on echocardiography by estimating the pulmonary artery systolic pressure (PASP) from the tricuspid regurgitation (TR) jet. The pressure difference between the right atrium and right ventricle was measured by continuous-wave Doppler of the TR trace and calculated using the simplified Bernoulli equation ($P = 4[TRmax]^2$) from the peak TR velocity, a value of ≤ 2.8 m/s being considered normal. Severity of pulmonary hypertension was graded by the estimated right ventricular systolic pressure (RVSP, also known as PASP) as normal (< 35 mmHg), mild (35–45 mmHg), moderate (46–60 mmHg) or severe (> 60 mmHg). The number of patients who developed pulmonary hypertension, their severity of obstruction, and their severity of hypoxia were recorded.

All clinical, spirometric, arterial blood gas, echocardiographic and demographic data were prospectively entered into the structured study proforma. Data were analyzed by descriptive statistics; continuous variables were expressed as mean ± standard deviation, and categorical variables as frequencies and percentages.

Inclusion Criteria

- All OPD patients diagnosed to have COPD.
- Patients willing to provide written informed consent.

Exclusion Criteria

- Patients not willing to participate in the study.
- Patients in acute exacerbation.
- Patients who had been hospitalized.

RESULTS

A total of 138 patients of COPD were studied. Men predominated, accounting for more than two-thirds of the cohort, with a male-to-female ratio of roughly 2.1:1. Most men were smokers or ex-smokers, whereas the women had a history of biomass fuel exposure. [Figure 1]

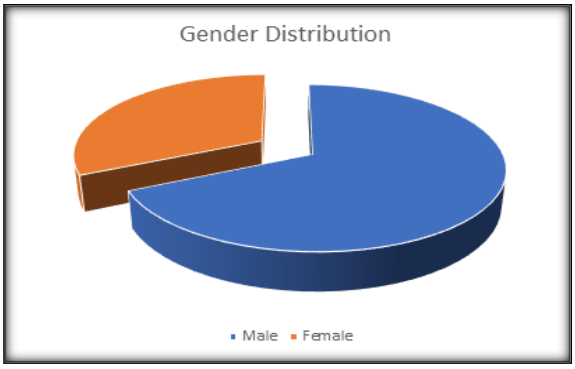

Figure 1: Sex distribution of the study population (N = 138).

Severity of airflow obstruction was graded from FEV1 on spirometry (GOLD 2019). Severe obstruction was the commonest grade at 37.6%, and severe or very severe obstruction together accounted for nearly two-thirds of patients. [Figure 2]

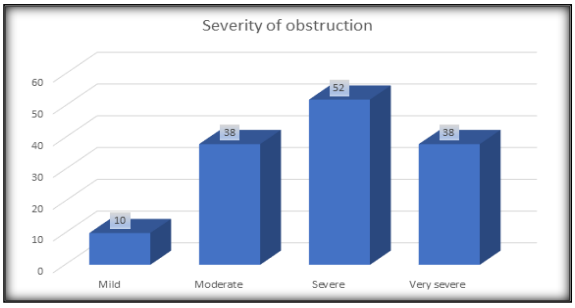

Figure 2: Distribution of patients by severity of airflow obstruction on spirometry

Table 1: Distribution of patients by severity of hypoxia on arterial blood gas analysis

Severity of hypoxia	Number Of Cases	percentage
Mild	59	42.8
Moderate	56	40.6
Severe	6	4.3
No hypoxia	17	12.3
Total	138	100

Hypoxia on arterial blood gas analysis (room air) was present in 121 patients (87.7%); mild and moderate hypoxia together made up the large majority, and only 6 patients (4.3%) had severe hypoxia. [Table 1]

Table 2: Overall prevalence and severity of pulmonary hypertension (PH)

Pulmonary hypertension	Number Of Cases	percentage
Present	61	44.2
Absent	77	55.8
Among those with pulmonary hypertension		
— Mild	47	77.0
— Moderate	9	14.8
— Severe	5	8.2

Overall, 61 of the 138 patients (44.2%) developed pulmonary hypertension. Among these, mild

pulmonary hypertension strongly predominated (77.0%), with moderate and severe disease seen in 14.8% and 8.2%, respectively. [Table 2]

Table 3: Overall prevalence and severity of pulmonary hypertension (PH)

Severity of obstruction	Mild PH	Moderate PH	Severe PH	No PH	Total
Mild	4	0	2	4	10
Moderate	10	2	1	25	38
Severe	14	6	1	31	52
Very severe	19	1	1	17	38
Total	47	9	5	77	138

The prevalence of pulmonary hypertension rose with worsening airflow obstruction, being present in 6 of 10 patients (60.0%) with mild obstruction and 21 of 38 (55.3%) with very severe obstruction. Mild pulmonary hypertension predominated across all grades, while moderate-to-severe pulmonary hypertension clustered among patients with moderate and severe obstruction. [Table 3]

Table 4: Prevalence of pulmonary hypertension (PH) within each grade of airflow obstruction

Severity of obstruction	Total patients	Developed PH (n)	PH prevalence (%)
Mild	10	6	60.0
Moderate	38	13	34.2
Severe	52	21	40.4
Very severe	38	21	55.3
Total	138	61	44.2

The number of patients developing pulmonary hypertension within each obstruction grade, and the proportion affected, are shown in Table 6. The proportion with pulmonary hypertension was highest in the mild and very severe obstruction groups, though the absolute number of affected patients was greatest among those with severe and very severe obstruction. [Table 4]

Table 5: Distribution of severity of pulmonary hypertension (PH) across grades of hypoxia

Severity of hypoxia	Mild PH	Moderate PH	Severe PH	No PH	Total
Mild	19	2	0	38	59
Moderate	22	4	3	27	56
Severe	3	0	2	1	6
No hypoxia	4	2	0	11	17
Total	48	8	5	77	138

Among the 121 patients with hypoxia, 55 (45.5%) developed pulmonary hypertension; notably, 6 of the 17 patients without hypoxia (35.3%) also developed pulmonary hypertension. The severity of pulmonary hypertension varied across the different grades of hypoxia, with moderate and severe pulmonary hypertension seen almost entirely among patients with moderate and severe hypoxia. [Table 5]

Table 6: Prevalence of pulmonary hypertension (PH) within each grade of hypoxia

Severity of hypoxia	Total patients	Developed PH, n	PH prevalence, %
Mild	59	21	35.6
Moderate	56	29	51.8
Severe	6	5	83.3
No hypoxia	17	6	35.3
Total	138	61	44.2

The proportion of patients developing pulmonary hypertension increased with the severity of hypoxia, reaching 5 of 6 patients (83.3%) in the severe-hypoxia group. [Table 6]

DISCUSSION

Chronic obstructive pulmonary disease is a leading cause of morbidity and mortality worldwide, with an increasing prevalence during the past decades.[11] Development of pulmonary hypertension is an established complication of COPD. Typically, PH appears when there is severe airflow limitation and association of chronic hypoxemia. Although newer mechanisms have emerged recently, the main pathophysiological cause is chronic alveolar hypoxia. In COPD, pulmonary vascular remodeling is the most important cause of the rise in pulmonary artery pressure and is thought to be a result of the combined effects of inflammation, hypoxia and loss of capillaries in severe emphysema.

In COPD, the determination of PH prevalence has been inaccurate due to difficulties in obtaining valid data from an adequate population-based COPD sample. The main reason is that right heart

catheterization (RHC) cannot be performed on a large scale due to ethical reasons,[12] as it is an invasive and expensive procedure and is also not easily available. Therefore, indirect alternative methods such as echocardiography, in spite of showing some errors such as poor cardiac window and observer bias, are used to measure PH by measuring pulmonary artery systolic pressure by the tricuspid regurgitation jet. The difference in pressures between the right atrium and right ventricle is measured by continuous wave Doppler of the tricuspid regurgitation trace, calculated by the simplified Bernoulli equation using peak TR velocity. PASP on right heart catheterization correlates well with this method.[13]

In the present study, out of the total 138 patients, 10 (7.2%) had mild obstruction, 38 (27.5%) had moderate obstruction, 52 (37.6%) had severe obstruction and 38 (27.5%) had very severe obstruction. Out of all these, 61 (44.2%) patients developed pulmonary hypertension. In a study conducted by Deepali J. Kamdar and Dharmesh Kumar Patel, pulmonary arterial hypertension was more common among patients with very severe airway obstruction, that is, 94.1% patients with very severe obstruction had pulmonary hypertension.[14] In a study conducted by NK Gupta et al, echocardiographic evaluation of COPD patients showed that 50% patients had normal echocardiographic parameters, and the frequencies of pulmonary hypertension in mild, moderate, severe, and very severe airway obstruction were 16.67%, 54.55%, 60.00%, and 83.33%, respectively.[15] However, our study group included patients with a lower smoking index and fewer smoking years, and a lesser duration of exposure to biomass fuel as well. Overall, the percentage of patients with mild pulmonary hypertension is more, but the prevalence of moderate to severe pulmonary hypertension is higher among moderate to severe obstruction individuals.

Alveolar hypoxia strongly contributes to the development of pulmonary hypertension in patients with COPD.[16] In COPD, the exact prevalence of pulmonary hypertension remains uncertain; however, pulmonary hypertension is relatively common in moderate–severe disease, and an increased prevalence is seen in more severe forms of the disease.[17] A notable feature observed in modern studies, however, is the preponderance of mild pulmonary hypertension even in severe forms of COPD. Thabut et al identified a small subset of "atypical" patients who had only moderately severe COPD but severe pulmonary hypertension and marked hypoxemia.[18]

In the majority of cases, even though pulmonary artery pressure can increase transiently during acute exacerbations, COPD-related pulmonary hypertension progresses slowly. A small number of cases progress to develop the overt right heart failure and peripheral edema characterizing cor pulmonale. A significantly worse prognosis is indicated by the occurrence of cor pulmonale.[19] Hypoxia also appears to be a contributing factor in the development of endothelial dysfunction, which is characterized by loss of the physiological balance between vasodilation and vasoconstriction. Hypoxia has been demonstrated to cause impairment of endothelium-dependent pulmonary artery relaxation in severe COPD patients, suggesting a decrease in nitric oxide bioavailability.[20] Conversely, upregulation of the production of vasoconstrictive mediators, such as endothelin-1, appears to be due to hypoxia, which in turn leads to increased vascular tone.

In our study group, 121 patients had hypoxia on ABG taken at room air, among which 55 developed pulmonary hypertension, while 6 of the 17 patients without hypoxia also developed pulmonary hypertension. Many studies concluded that hypoxia in patients of COPD is directly correlated to the severity of the disease. In our study group, we observed that the development of pulmonary hypertension was directly proportional to the severity of the disease, that is, it depends on the obstruction severity of the airways and exposure to etiological factors.

CONCLUSION

Development of pulmonary hypertension is dependent on the severity of the disease. In our study, it is observed that the development of pulmonary hypertension is directly related to the severity of airway obstruction in spirometry and severity of hypoxia in ABG, i.e., the more the severity of airway obstruction and hypoxia, the higher is the prevalence of pulmonary hypertension. However, the severity of pulmonary hypertension does not correlate with the levels of severity of airway obstruction and hypoxia in each and every patient. Spirometry and arterial blood gas analysis can identify at-risk patients early.

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