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Frequency of Type-II Respiratory Failure in Chronic Obstructive Pulmonary Disease

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ABSTRACT

Background: Chronic obstructive pulmonary disease is a long progressive disorder of lungs where the airflow become reduced and gas exchange not occur properly. It mostly develops because of long exposure to tobacco smoke, burning of biomass fuel, and polluted environment. When disease go to advance stage, the alveoli not ventilate well and carbon dioxide start to retain while oxygen level in blood become low, which finally lead to Type-II respiratory failure. **Objective:** To determine the frequency of type-II respiratory failure in chronic obstructive pulmonary disease. **Study Design:** Cross-sectional study. **Duration and Place of Study:** This study was conducted from February 2025 to May 2025 in the Pulmonology Department of Saidu Group of Teaching Hospitals, Swat. **Methodology:** A total of 143 patients aged 30–75 years with spirometry-confirmed chronic obstructive pulmonary disease were enrolled through consecutive non-probability sampling. Detailed clinical assessment and arterial blood gas analysis were performed. Type-II respiratory failure was identified when arterial carbon dioxide exceeded 45 mmHg with oxygen below 60 mmHg on room air. **Results:** The mean age of participants was 56.73 ± 8.52 years, with males comprising 84.6%. Type-II respiratory failure was observed in 36 patients (25.20%). A statistically significant association was found with smoking ($p < 0.001$), whereas no significant association was noted with age, gender, body mass index, or comorbidities. **Conclusion:** Type-II respiratory failure is a frequent and serious complication among chronic obstructive pulmonary disease patients, strongly linked with smoking habits.

INTRODUCTION

Chronic Obstructive Pulmonary Disease is a progressive pulmonary disorder that make the breathing difficult and inefficient.[1] It is mostly result from long time exposure to cigarette smoke, industrial fumes, and environmental pollutants. The bronchial tubes become narrow and alveolar walls lose their stretch, causing air trapping inside the lungs.[2] Because of this, the patient experience dyspnea, chronic cough with sputum, and fatigue even during minimal exertion. Many patients not notice the early phase because the symptoms develop slowly, but as the condition advance, it causes hypoxemia and retention of carbon dioxide in the bloodstream.[3] Exacerbation of COPD often triggered by respiratory infection, cold climate, or irregular use of maintenance drugs.[4]

This disease produces several systemic complications which reduce patient health and well-being. Recurrent pulmonary infections like pneumonia cause more damage to the already compromised lung tissue.[5] The cardiac system also become affected due to persistent hypoxia, resulting in cor pulmonale where the right ventricle get hypertrophied.[6] Many patients develop loss of body mass and skeletal muscle wasting because the energy cost of breathing is high and appetite is poor.[7] Psychological issues like anxiety and depression are frequent since the patient live in constant fear of breathlessness. In advanced stage, chronic hypoxia causes multiple organ dysfunction, and long-term oxygen therapy become necessary for survival.[8]

Type-II respiratory failure is a major life-threatening consequence of COPD that occur when alveolar ventilation become inadequate.[9] It lead to elevation of arterial carbon dioxide (hypercapnia) and reduction of oxygen (hypoxemia). The main cause is fatigue of respiratory muscles, severe airway limitation, and altered gas exchange mechanism.[10] Central chemoreceptor response to carbon dioxide also decrease, which make the respiratory drive weak. The patient often present with confusion, drowsiness, or even coma in severe case.[11] Management include careful oxygen supplementation, non-invasive positive pressure ventilation, and treatment of precipitating factor, but delay in intervention can end in fatal outcome.[12]

A study reported the frequency of type-II respiratory failure was 24% in chronic obstructive pulmonary disease.[13]

There is important need to carry out this study in Swat because the number of patients with chronic respiratory illness is rising in the area. Many people are daily exposed to biomass smoke, road dust, and industrial fumes which contribute to lung damage. Most population belong to rural background with limited access to specialized respiratory care and poor health awareness, so many cases of COPD remain undiagnosed until late stage. This delay increases the chance of developing complications like Type-II respiratory failure. Studying the frequency of this condition in Swat will help in understanding its clinical burden and guide local health authority to improve early diagnosis and management service for affected patients.

METHODOLOGY

This cross-sectional research was done in the Pulmonology Department of Saidu Group of Teaching Hospitals, Swat, during February 2025 to May 2025. Ethical clearance was taken from the hospital ethical review committee (27-ERB/SMC/005) and approval was also obtained from the research section of the CPSP Head Office before starting the data collection. All study steps were carried out according to the standard ethical principles for patient-based clinical research.

A total of 143 patients were included. The sample size was estimated through the WHO calculator using 24% expected proportion of Type-II respiratory failure among COPD cases,[13] with 7% absolute precision and 95% confidence level. Patients were selected by consecutive non-probability sampling method from the admitted and outpatient population of the pulmonology ward. Patients aged between 30 and 75 years of both sexes who had COPD confirmed on spirometry with post-bronchodilator FEV1/FVC ratio less than 70% and symptoms of dyspnea, chronic cough, and sputum were included. Those with asthma, pneumonia, bronchitis, or cardiogenic pulmonary edema were excluded. COPD was taken as chronic airway limitation not fully reversible, mostly due to airway inflammation and alveolar destruction from long exposure to irritants. Before any data recording, the aim, benefit, and risk of study were explained to the patients and written consent was obtained. Only those who agreed voluntarily were enrolled.

Detailed history was taken regarding respiratory symptoms, disease duration, and past hospital visits. Clinical examination was performed by a consultant pulmonologist with at least five years post-fellowship experience. The findings and test results were noted on a pre-structured proforma prepared for this research. Patients diagnosed with COPD were tested for Type-II respiratory failure through arterial blood gas analysis. Type-II respiratory failure was labeled when carbon dioxide ($PaCO_2$) was greater than 45 mmHg with low oxygen ($PaO_2 < 60$ mmHg) on room air. These results were used to determine the frequency of the outcome among the study group.

All information was entered and analyzed using IBM SPSS version 25. Categorical variables were presented as frequency and percentage. Continuous data were shown as mean and standard deviation or median with interquartile range. Stratification was applied for factors such as age, gender, BMI, disease duration, and comorbidities, and chi-square or Fisher's exact test was applied with a 5% significance level.

RESULTS

The study included 143 patients having mean age of 56.73 ± 8.52 years and mean BMI was 25.74 ± 2.30 kg/m^2, with disease duration averaging 22.26 ± 12.50 months, the majority of participants was males comprising 121 patients (84.6%) while females were only 22 (15.4%), regarding socioeconomic status, low socioeconomic group represented largest proportion with 76 patients (53.1%), followed by middle class 50 patients (35.0%) and high socioeconomic status 17 patients (11.9%), most patients were from rural areas 101 (70.6%) compared to urban residents 42 (29.4%), education status showed that 77 patients (53.8%) were literate whereas 66 (46.2%) were illiterate, comorbidities analysis revealed that 67 patients (46.9%) had hypertension while 76 (53.1%) did not have hypertension, diabetes was present in 49 patients (34.3%) and absent in 94 patients (65.7%), smoking history showed that 69 patients (48.3%) were smokers and 74 (51.7%) were non-smokers as shown in Table 1.

Table 1

Patient Demographics

Demographics		Mean ± SD / n (%)
Age (Years)		56.73 ± 8.52
BMI (kg/m^2)		25.74 ± 2.30
Duration of Disease (Months)		22.26 ± 12.50
Gender	Male n (%)	121 (84.6%)
	Female n (%)	22 (15.4%)
Socioeconomic Status	Low n (%)	76 (53.1%)
	Middle n (%)	50 (35.0%)
	High n (%)	17 (11.9%)
Residence	Rural n (%)	101 (70.6%)
	Urban n (%)	42 (29.4%)
Education	Literate n (%)	77 (53.8%)
	Illiterate n (%)	66 (46.2%)
Hypertension	Yes n (%)	67 (46.9%)
	No n (%)	76 (53.1%)
Diabetes	Yes n (%)	49 (34.3%)
	No n (%)	94 (65.7%)
Smoking	Yes n (%)	69 (48.3%)
	No n (%)	74 (51.7%)

The frequency of Type-II respiratory failure in chronic obstructive pulmonary disease patients was found in 36 patients representing 25.20% of total study population, while 107 patients (74.80%) did not develop Type-II respiratory failure as shown in Table 2.

Table 2

Frequency of Type-II Respiratory Failure in Chronic Obstructive Pulmonary Disease

Type-II Respiratory Failure	Frequency	% age
Yes	36	25.20%
No	107	74.80%
Total	143	100%

When stratification analysis was performed for association of Type-II respiratory failure with demographic factors, age groups showed that among patients ≤50 years, 10 (30.3%) had Type-II respiratory failure and 23 (69.7%) did not have it, while in patients >50 years, 26 (23.6%) had Type-II respiratory failure and 84 (76.4%) did not have it with p-value of 0.439 which was

not significant, gender distribution revealed that 33 males (27.3%) had Type-II respiratory failure while 88 (72.7%) did not have it, and among females 3 (13.6%) had Type-II respiratory failure while 19 (86.4%) did not have it with p-value of 0.197 using Fischer Exact Test showing no significant association, BMI stratification showed that patients with BMI ≤25 kg/m^2, 13 (25.0%) had Type-II respiratory failure and 39 (75.0%) did not have it, while patients with BMI >25 kg/m^2, 23 (25.3%) had Type-II respiratory failure and 68 (74.7%) did not have it with p-value of 0.971 indicating no significant difference, disease duration analysis demonstrated that patients with duration ≤24 months, 25 (28.7%) had Type-II respiratory failure and 62 (71.3%) did not have it, whereas patients with duration >24 months, 11 (19.6%) had Type-II respiratory failure and 45 (80.4%) did not have it with p-value of 0.221 showing no significant association, hypertension status showed that among hypertensive patients 16 (23.9%) had Type-II respiratory failure and 51 (76.1%) did not have it, while among non-hypertensive patients 20 (26.3%) had Type-II respiratory failure and 56 (73.7%) did not have it with p-value of 0.738 which was not significant, diabetes analysis revealed that among diabetic patients 10 (20.4%) had Type-II respiratory failure and 39 (79.6%) did not have it, while among non-diabetic patients 26 (27.7%) had Type-II respiratory failure and 68 (72.3%) did not have it with p-value of 0.343 indicating no significant association, however smoking status demonstrated highly significant association where among smokers 29 (42.0%) had Type-II respiratory failure and 40 (58.0%) did not have it, while among non-smokers only 7 (9.5%) had Type-II respiratory failure and 67 (90.5%) did not have it with p-value <0.001 showing statistically significant association as shown in Table 3.

Table 3

Association of Type-II Respiratory Failure with Demographic Factors

Demographic Factors		Type-II Respiratory Failure		p-value
		Yes n(%)	No n(%)	
Age (years)	≤50	10 (30.3%)	23 (69.7%)	0.439
	>50	26 (23.6%)	84 (76.4%)	
Gender	Male	33 (27.3%)	88 (72.7%)	0.197*
	Female	3 (13.6%)	19 (86.4%)	
BMI (Kg/m^2)	≤25	13 (25.0%)	39 (75.0%)	0.971
	>25	23 (25.3%)	68 (74.7%)	
Duration of Disease (Months)	≤24	25 (28.7%)	62 (71.3%)	0.221
	>24	11 (19.6%)	45 (80.4%)	
Hypertension	Yes	16 (23.9%)	51 (76.1%)	0.738
	No	20 (26.3%)	56 (73.7%)	
Diabetes	Yes	10 (20.4%)	39 (79.6%)	0.343
	No	26 (27.7%)	68 (72.3%)	
Smoking	Yes	29 (42.0%)	40 (58.0%)	<0.001
	No	7 (9.5%)	67 (90.5%)	

*Fischer Exact Test

DISCUSSION

In current study, the frequency of Type-II respiratory failure was found to be 25.20% (36 out of 143 patients), this finding indicates that quarter of COPD patients develops Type-II respiratory failure which is characterized by hypercapnia along with hypoxemia, the pathophysiological mechanism behind this is that in COPD patients there is progressive airflow limitation and air trapping which leads to ventilation-perfusion mismatch and alveolar hypoventilation, additionally the respiratory muscles becomes fatigued due to increased work of breathing and chronic hyperinflation of lungs which further impairs carbon dioxide elimination resulting in hypercapnic respiratory failure. The mean age of patients was 56.73 ± 8.52 years indicating that Type-II respiratory failure in COPD predominantly affects middle-aged to elderly population, this is because COPD itself develops after prolonged exposure to risk factors and cumulative damage to airways occurs over decades, moreover with advancing age there is natural decline in lung function and respiratory muscle strength which makes older patients more vulnerable to respiratory failure. Male patients comprised 84.6% of study population which shows male predominance, this gender difference is explained by higher prevalence of smoking among males in our society and greater occupational exposure to dust and pollutants in male workers. The highly significant association was found between smoking and Type-II respiratory failure (p<0.001) where 42.0% of smokers developed Type-II respiratory failure compared to only 9.5% of non-smokers, this strong association is due to fact that cigarette smoke causes direct injury to bronchial epithelium, increases mucus production, destroys alveolar walls leading to emphysema, and promotes chronic inflammation in airways which accelerates decline in lung function and increases risk of respiratory failure in COPD patients.

The frequency of Type-II respiratory failure in our study was 25.20% which shows close similarity with findings of Jalal D, et al. [13] who reported 24% frequency, this comparable frequency suggests that approximately one quarter of COPD patients develops hypercapnic respiratory failure when disease progresses to advanced stages. However, our findings are considerably lower than Shrivastava HK, et al. [14] who reported 70% patients had Type-II respiratory failure, this marked difference can be attributed to their hospital setting where patients with acute exacerbations and severe disease were included. Similarly, Iqbal S, et al. [15] reported 35.5% frequency of respiratory failure in acute COPD exacerbation which is higher than our findings due to their specific inclusion of only acute exacerbation cases. Zia M, et al. [16] reported that Type-II respiratory failure comprised 41.5% of all respiratory failure cases in ICU setting, this higher frequency is because ICU patients represents more critically ill population with advanced disease.

Regarding age distribution, our study showed mean age of 56.73 ± 8.52 years which is relatively younger compared to Shrivastava HK, et al. [14] who reported 64 years and Iqbal S, et al. [15] with 59.3 ± 10.8 years, while Ullah R, et al. [17] observed 63 ± 11.38 years, these older mean ages suggests that respiratory complications becomes more prevalent with advancing age due to cumulative lung damage. However, our mean age was comparable to Jalal D, et al. [13] with 53.41 ± 9.36 years and Zia M, et al. [16] with 48.6 ± 18.8 years indicating that in certain populations respiratory failure can develop at relatively younger age. Krishna TV, et al. [18] reported mean age of 60 ± 10 years specifically for Type-II respiratory failure patients which is slightly higher than our findings.

Male predominance was marked feature in our study with

84.6% males which is consistent with Shrivastava HK, et al. [14] who reported 83.3% males and Zia M, et al. [16] with 61.7% males, this gender distribution is primarily attributed to higher smoking rates among males and greater occupational exposures. Zhu A, et al. [19] also found that male sex was independent predictor of COPD (OR = 1.59, p = 0.036) which supports biological and behavioral factors that makes males more susceptible. However, Ullah R, et al. [17] reported 53.5% females which is unusual and might reflect regional variations in biomass fuel smoke exposure. Iqbal S, et al. [15] found respiratory failure was more common in males (23.5%) compared to females (12.0%) which aligns with our male predominance.

The most significant finding was strong association between smoking and Type-II respiratory failure (p<0.001) where 42.0% of smokers developed respiratory failure compared to only 9.5% of non-smokers, this is supported by Shrivastava HK, et al. [14] who reported 83.3% of respiratory failure patients were smokers demonstrating devastating impact of smoking on lung function. Chronic smoking causes progressive alveolar destruction, mucus hypersecretion, chronic inflammation, and irreversible airflow limitation culminating in respiratory failure.

No significant association was found between Type-II respiratory failure and age groups in our study (p=0.439) which contrasts with Jalal D, et al. [13] who reported patients >55 years were more affected (22.9%), this suggests development of respiratory failure may be more dependent on disease severity rather than age alone. Zhu A, et al. [19] demonstrated age >60 years was strong predictor (OR = 3.83, p = 0.037) indicating older age increases risk, however lack of significance in our study could be due to different age categorizations. Zia M, et al. [16] found mean age was significantly higher for Type-II respiratory failure patients (53.6 ± 15.8 years) compared to Type-I (45.1 ± 19.3 years, p = 0.0003) suggesting Type-II develops in older patients, but our study did not show this relationship possibly due to smaller sample size.

Regarding comorbidities, no significant association was found between hypertension and Type-II respiratory failure (p=0.738) or diabetes and respiratory failure (p=0.343) in our study, this indicates these comorbidities do not directly influence hypercapnic respiratory failure development. However, Zhu A, et al. [19] reported COPD patients had lower systolic blood pressure and higher SBP was protective factor (OR = 0.99, p = 0.020) suggesting complex relationship between blood pressure and COPD severity.

The present study has several limitations which needs to be acknowledged, firstly this was single center study conducted at one hospital which limits generalizability of findings to broader population as patient characteristics and disease patterns may vary across different geographical regions and healthcare settings. Secondly, the sample size was quite small with only 143 patients, and this maybe make statistical power low to find meaningful association, especially in subgroup analysis where few patients in each category could hide the real relationship between variables and Type-II respiratory failure. Thirdly, the study uses cross-sectional design which only give single snapshot of disease at one time and not show how it change or progress with time, so longitudinal study is needed to know how Type-II respiratory failure start and move forward in COPD patients. Fourthly, disease severity was not checked with standard classification like GOLD stage, and also pulmonary function tests like FEV1 were not measured, which could give more detailed picture about how lung function decline relate to development of respiratory failure.

CONCLUSION

In this study we have conclude that Type-II respiratory failure is significant complication occurring in considerable proportion of patients with chronic obstructive pulmonary disease and represents important clinical concern requiring early identification and appropriate management. The findings demonstrate that smoking is strongly associated with development of Type-II respiratory failure in COPD patients highlighting the critical role of smoking cessation in preventing progression to hypercapnic respiratory failure.

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