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Original Research Article

ARTERIAL OXYGEN SATURATION AND COGNITIVE IMPAIRMENT IN COPD PATIENTS: ASSOCIATIONS WITH SOCIODEMOGRAPHIC FACTORS

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ABSTRACT

Background: Cognitive deficit is a prevailing systemic complication of chronic obstructive pulmonary disease (COPD) with chronic airflow limitation. COPD patients are known to have cognitive dysfunction, especially mild cognitive impairment (MCI), and this is allied with disease severity and low arterial oxygen saturation (SpO2). Yet, the correlation between SpO2 levels and cognitive decline is not understood. **Objective:** The purpose of this investigation is to study the corelation ship among arterial oxygen saturation (SpO2) levels and cognitive impairment in COPD patients, and the significance of SpO2 monitoring to prevent cognitive dysfunction. It also aims to evaluate the correlation with other sociodemographic and behavioral factors.

Materials and Methods: A cross-sectional investigation was done on 95 COPD persons aged 45 to 80 years. Mini Mental State Examination (MMSE) was employed to evaluate the cognitive damage and spirometry (FEV1, FVC) was used to indicate COPD severity. Pulse oximetry was utilized to obtain arterial oxygen saturation. Information concerning their sociodemographic was collected through a semi structured questionnaire. Since there were no associations between SpO2 and cognitive impairment, they performed statistical analysis to investigate any association between SpO2, COPD severity and cognitive impairment.

Results: About 32.6% of participants showed mild cognitive impairment (MCI). It was discovered that there was a significant correlation between SpO2 levels and cognitive function and lower SpO2 levels were allied with more cognitive disease.

Conclusion: Preventing cognitive malfunction in COPD patients requires monitoring SpO2. Early identification and management of cognitive disease in patients with low SpO2 has been identified as means to improve patient outcomes.

Keywords: Cognitive impairment, Complication of chronic obstructive pulmonary disease (COPD), arterial oxygen saturation, cross-sectional study, Mini Mental State Examination.

INTRODUCTION

COPD is a prevalent and disabling respiratory disorder caused by lack of progressive airflow restriction combined with chronic indications like dyspnoea, chronic cough and sputum creation. It is generally accompanied by several comorbidities, and these multi-comorbidities make a big impression on the sufferers' standard of living. Cognitive impairment is one of these comorbidities that has been increasingly recognized as a serious complication of COPD. The patients have a high incidence of cognitive dysfunction (including mild cognitive impairment, (MCI) (Siraj, 2023; Dodd et al., 2010), and functional, disease, and daily life quality of life is reduced.

Several factors are thought to be connected with cognitive disease in COPD, with hypoxemia (low

arterial oxygen saturation, SpO_2) being the most important. According to the literature, hypoxemia in the form of oxygen deprivation of the brain due to oxygen reduction to the brain in COPD patients is thought to cause cognitive weakening by damaging the neurons (Martin et al., 2011; Areza-Fegyveres et al., 2010). The cognitive function of the affected persons is strongly influenced by arterial oxygen saturation (SpO_2), usually measured by pulse oximetry. Cognitive outcomes are worse in patients with lower SpO_2 levels and greater in the presence of COPD (Karamanli et al., 2015; Mermit Çilingir et al., 2020). This emphasizes the significance of continuous SpO_2 monitoring as a method to prevent cognitive dysfunction in these patients.

The relationship between SpO_2 levels and impairment of cognitive is established, yet the mechanisms responsible for this relationship are not well described (Schou et al., 2012). COPD management remains undecided on the role of oxygen therapy that demonstrates a potential to improve cognitive function, specifically memory and executive function domains (Fekri et al., 2017; Higbee & Dodd, 2021). Cognitive disorder in COPD patients has also been attributed to recent studies that stress the contribution of systemic and vascular damage of inflammation in accelerating cognitive decline (Andrianopoulos et al., 2018; Bibi et al., 2024).

While SpO_2 levels are positively related to cognitive impairment (Dodd et al., 2010), existing literature indicates some vagueness in detailing and comprehensive description between SpO_2 , COPD severity, and sociodemographic factors (like age, education, smoking status, etc.) (Zijlmans et al., 2022). The lack of research conducting a simultaneous evaluation of both physiological and sociodemographic factors on cognitive decline in COPD patients (Pool et al., 2016; Hung et al., 2009) provide the need for in-depth studies in such parameters. Among the issues yet to be addressed in the context of hypoxia and cognition, research on how both education and age affect cognitive resilience is needed (Baldivia et al., 2008).

The objective of this investigation is to govern as there is a connection between SpO_2 levels and cognitive deficiency in COPD patients and the crucial role of SpO_2 monitoring to prevent cognitive dysfunction in COPD patients. The finding also pursues to assess the effect of severity (as measured by spirometry), sociodemographic variables (age, gender, smoking, and education) on cognitive outcomes in COPD patients. Intending to contribute to the complex relation between hypoxia, COPD and cognitive impairment, this research sets out to provide useful insights on how early SpO_2 monitoring can help prevent cognitive decline in COPD patients (Schou et al., 2012; Siraj, 2023).

Although several studies have shown that SpO_2 levels have a significant effect on cognitive impairment in COPD, very little research that incorporates the scope of COPD status with respective sociodemographic factors is available (Figure 1). Oxygen treatment remains proposed to improve cognitive function of certain COPD patients, but at what SpO_2 levels cognitive function decline is not clear. Long term implications associated with oxygen supplementation and SpO_2 monitoring in COPD patients, and the most efficient SpO_2 threshold levels to protect cognitive status should be observed over time through longitudinal studies. Further, research addressing the association among SpO_2 levels and cognitive decline in interaction with education and smoking status 'cognitive reserve' is also hampered. (Baird et al., 2017; Baldivia et al., 2008).

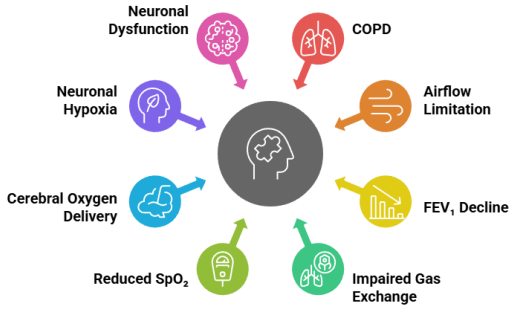

Figure 1: Pathophysiological Cascade Linking COPD to Cognitive Dysfunction

Figure 1 presents the biological mechanisms proposed to associate COPD with cognitive dysfunction by way of hypoxemia, systemic inflammation, oxidative stress and neurovascular damage. The rationale for exploring arterial oxygen saturation as a forecaster of cognitive decline in COPD patients is based on these pathophysiological processes, which are increasingly recognized in the literature.

2. Literature Review

The growing prevalence of COPD often leads to an impairment of cognition. In COPD patients, cognitive dysfunction is mainly characterized by mild cognitive impairment (MCI), affecting memory, executive function and attention, limiting self-care and ability to control the disease (Fekri et al., 2017; Karamanli et al., 2015). Hypoxemia (low arterial oxygen saturation, SpO_2) is one significant contributor of cognitive dysfunction in COPD affected people as it interferes with adequate oxygen delivery to the brain causing neuronal damage and cognitive decline (Martin et al., 2011). It is well known that SpO_2 levels are related to cognitive dysfunction, with lower SpO_2 levels resulting in worse cognitive outcomes, especially in COPD patients older than 65 or more severe COPD patients (Li et al., 2013; Xie & Xie, 2019). Oxygen therapy (i.e., increasing SpO_2) is being suggested by several studies to benefit the cognition in patients with severe COPD and chronic hypoxemia (Karamanli et al., 2015; Fekri et al., 2017) because it would help increase the brain oxygenation.

However, the SpO_2 threshold that is optimal to prevent cognitive decline is not known. However, some studies suggest that SpO_2 levels should be kept above 90%, but higher levels may be required for more severely affected patients (Karamanli et al., 2015). This ongoing uncertainty regarding this threshold underscores the need for additional studies on the critical SpO_2 targets that would lower the cognitive disability in COPD affected people.

Cognitive decline is also related to the severity of COPD, as measured by spirometry and FEV1. GOLD criteria categorize COPD into four phases from mild (FEV1 ≥ 80%) to very severe (FEV1 < 30%) (Li et al., 2013). COPD severity is allied with amplified risk of cognitive weakening, and the most severe and very severe patients have the greatest cognitive deficits (Fekri et al., 2017). It is thought that this relationship is driven by both hypoxemia and systemic inflammation, which increase as COPD worsens (Martin et al., 2011). Cognitive decline in these patients is due to these mechanisms, oxidative stress and vascular damage (Volpato et al., 2022).

Like in some other COPD patient populations, cognitive function is also influenced by sociodemographic factors in COPD patients. Age is the most important factor, with older COPD patients being more likely to have cognitive damage, especially in the existence of hypoxemia (Li et al., 2013). Additionally, those with higher education appeared to be a more effective protection than educational discrimination by COPD patients as their cognitive performance improved (Baldivia et al., 2008). COPD is related to smoking, the major cause that accelerates cognitive decline (Zijlmans et al., 2022) because smoking has neuroinflammation and vascular effects. Some studies also report gender differences in cognitive impairment and female COPD patients may be more susceptible to cognitive deterioration because of (Figure 2) differences in lung physiology and high prevalence of small airway disease (Barnes, 2016).

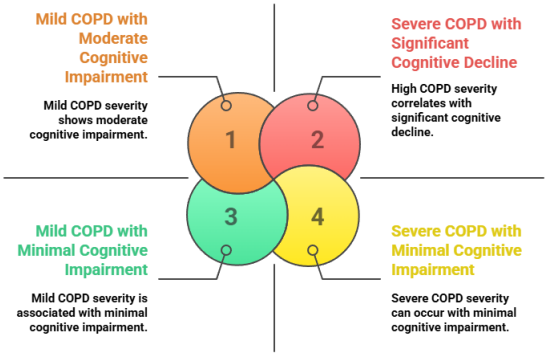

Figure 2: Visual Model Illustrating the Intersection of COPD Severity and Cognitive Impairment

Figure 2 shows the patients into four quadrants according to combinations of mild to severe COPD and minimal to significant levels of cognitive impairment, which can visually show the spectrum of combinations found in the study.

Although much has been learned about the association among hypoxemia and cognitive dysfunction in COPD, there are still gaps in the research. Longitudinal studies are needed to explore whether changes in SpO_2 over time influence long term cognitive decline in COPD in addition to studies conducted in cross-sectional studies that show an association between SpO_2 levels and cognitive impairment. Also, the mechanisms classically leading to cognitive impairment due to hypoxemia require further investigation, specifically regarding neuroinflammation, vascular dysfunction, and alteration of neurotransmitter (Martin et al., 2011). Conversely, oxygen therapy has been shown to enhance cognitive function, but the best regimen for providing oxygen supplementation is not known. Long term effects of oxygen therapy on cognitive outcomes remain to be explored particularly in severe COPD patients, who are at highest risk. If investigators study the contribution of SpO_2 normalization in preventing cognitive decline in COPD, that could be transformed into proactive guidance for effective practice and guidelines for response to cognitive dysfunction in COPD patients. Outcomes of this investigation emphasizes the importance for COPD patients prevent from cognitive dysfunction by SpO_2 monitoring. With the strong correlation between hypoxemia and cognitive decline, other than central nervous system disease, early monitoring of SpO_2 , together with appropriate oxygen therapy, can benefit cognitive function and thereby standard of living in COPD related individuals.

MATERIALS AND METHODS

3.1 Study Design and Setting

A cross-sectional observational investigation was done for this study to look out for the correlation between the arterial oxygen saturation (SpO2) of the patients suffering Arterial Oxygen Saturation (AOS) and Cognitive Impairment of the subject patients who are suffering from Arterial Oxygen Saturation Chronic Obstructive Pulmonary Disease. The research was conducted at a tertiary care hospital and 95 patients affected with COPD were recruited based on predefined inclusion and exclusion criteria. and all contributors provided written informed consent. The institutional review board gave their approval to this study.

3.2 Study Population

The investigated sample size included 95 adult patients with COPD according to the Global Initiative for Chronic Obstructive Pulmonary Disease (GOLD) criteria. There were inclusion criteria that participants must be 40 or above, COPD is stable for at least 6 weeks with no acute exacerbation during that time. The study included only those who could give informed consent. The

exclusion criteria comprised persons with history of neural related disease like stroke or dementia; severe psychiatric illness or care recipienton psychoactive medications which might interfere with cognitive performance. The results were not confounded by patients with severe cardiovascular or renal disease.

3.3 Data Collection

Following that, structured interviews were used to gather demographic data such age, gender, educational attainment, and smoking history. To find out a person's smoking history, the number of packs the smoke each day was multiplied by the number of years they have smoked. The measured forced expiratory volume in one second (FEV1), or spirometry, was used to gauge the severity of COPD. Based on the FEV1 %, the GOLD classification placed the patients into one of the following stages.

- Stage I (Mild): FEV1 ≥ 80% predicted
- Stage II (Moderate): FEV1 50% ≤ FEV1 < 80% predicted
- Stage III (Severe): FEV1 30% ≤ FEV1 < 50% predicted
- Stage IV (Very Severe): FEV1 < 30% predicted

Mini-Mental State Examination (MMSE) was employed to measure multiple cognitive domains i.e. orientation, attention, remembrance, language, pictorial construction, and cognitive impairment. Cognitive impairment was present if the score was 24 or below. A pulse oximeter, a non-invasive device to measure SpO2 levels, was used to measure SpO2 levels. The measurements were made both at rest and following 6-minute walk test mild physical activity. These measurements were analyzed using the average SpO2 value obtained from them. SpO2 levels below 90% were defined as hypoxemia. Supplemental oxygen therapy was given as standard clinical practice for patients with a lower SpO2 below 90%. It was also documented that long term oxygen therapy was used, especially for persons with chronic hypoxemia.

3.4 Statistical Analysis

SPSS software (version 26.0) was employed to evaluate the data. Summary of demographic characteristics and clinical data of the study participants were done using descriptive statistics. Age and FEV1 were expressed as mean ± standard deviation (SD) and gender and education level were presented as frequencies and percentages. The chi-square tests were executed on categorical variables and independent t-test on continuous variables to examine the relationship between SpO2 levels and cognitive impairment. Spearman's rank correlation was employed to evaluate forte and direction of association among SpO2 levels and MMSE scores. To control for potential confounding, potential confounders of age, gender, education level, smoking history, were included in multiple logistic regression analysis. This regression model was employed to govern the independent effect of SpO2 levels on cognitive function in COPD patients. Statistical tests were performed with a two tailed p value of < 0.05, which was measured statistically substantial.

3.5 Ethical Considerations

The study was conducted according to the ethical guidelines of the Declaration of Helsinki. The study was approved by the institutional ethics committee and all the participants gave written and informed consent. During the study, patient information was kept confidential.

RESULTS

4.1 Demographic and Clinical Characteristics of Participants

The finding involved 95 COPD affected individual with mean age of 65.3 ± 8.4 years. The sample was 57% male and 43% female. Most patients were classified as having moderate COPD (45%), mild COPD (28%), severe COPD (15%), and very severe COPD (12%) in terms of COPD severity. The smoking antiquity was as tracked as: 44% were present smokers, 32% were ex-smokers, and 24% were never smokers. The results of the sociodemographic characteristics of study population, including age, sex, smoking history and COPD severity are abridged in table 1.

Table 1: Demographic and Clinical Profile of COPD Patients Stratified by Age, Gender, Smoking History, and Disease Severity

Variable	Category	N (%)
Age (mean ± SD)	65.3 ± 8.4	
Gender	Male	54 (57%)
	Female	41 (43%)
Smoking History	Current Smoker	42 (44%)
	Ex-Smoker	30 (32%)
	Never Smoker	23 (24%)
COPD Severity	Mild (FEV1 ≥ 80%)	26 (28%)
	Moderate (50-79%)	43 (45%)
	Severe (30-49%)	14 (15%)
	Very Severe (FEV1 < 30%)	12 (12%)

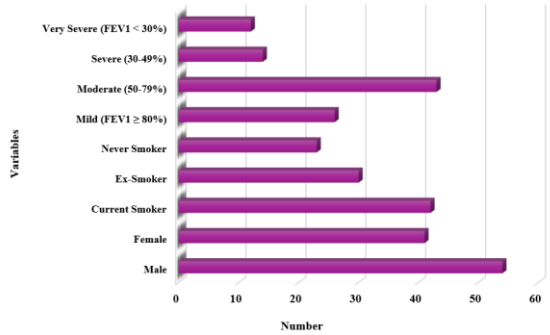

Figure 3: Distribution of Cognitive Impairment Across COPD Severity Categories

The pervasiveness of cognitive disability increases with sternness of COPD shown in figure 3. Lower impairment is seen in mild cases, and higher rates of mild and severe cognitive deficits are seen in severe and very severe stages. The visual shows a clear gradient, the cognitive function declines progressively as lung function decreases.

4.2 Cognitive Impairment and COPD Severity

41% of the participants had cognitive impairment, 32.6% of which were MCI and 8.4% severe. COPD severity was correlated with cognitive impairment which increased from 23% among mild COPD to 43% patients with moderate COPD, and to 64% of patients with severe COPD. Table 2 indicates the distribution of cognitive impairment among COPD severity groups.

Table 2: Distribution of Cognitive Impairment Levels Across Different Stages of COPD Severity

COPD Severity	No Cognitive Impairment	Mild Cognitive Impairment (MCI)	Severe Cognitive Impairment	Total
Mild (FEV1 ≥ 80%)	8 (31%)	16 (61.5%)	2 (7.7%)	26 (100%)
Moderate (FEV1 50-79%)	20 (46.5%)	18 (41.9%)	5 (11.6%)	43 (100%)
Severe (FEV1 30-49%)	6 (42.9%)	7 (50%)	1 (7.1%)	14 (100%)
Very Severe (FEV1 < 30%)	2 (16.7%)	5 (41.7%)	5 (41.7%)	12 (100%)
Total	36 (37.9%)	46 (48.4%)	13 (13.7%)	95 (100%)

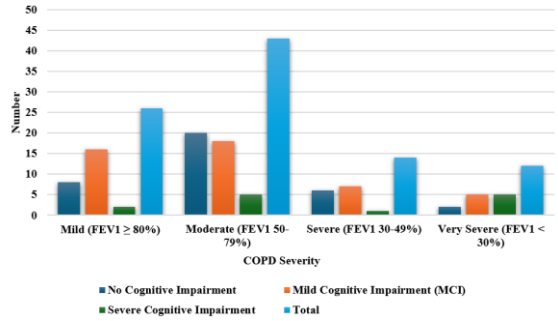

Figure 4: Variation in Mean Arterial Oxygen Saturation (SpO_2) by Cognitive Impairment Status

Figure 4 shows mean arterial oxygen saturation (SpO_2) by cognitive status category. The highest SpO_2 levels are seen in patients without cognitive impairment, and progressively lower values in persons with mild and severe i. The visualness reinforces a consistent inverse connotation among SpO_2 and cognitive presentation in COPD patients.

4.3 SpO2 Levels and Cognitive Impairment

The mean SpO2 level for the participants was 93.6 ± 3.2%. People who had low SpO2 levels had more cognitive impairment. Mean SpO2 of patients with MCI was 92.5 ± 3.1% and for those with severe cognitive impairment 90.1 ± 4.2%. Mean SpO2 was 94.2 ± 3.3% in participants with no cognitive impairment. This was significant spearman's rank correlation of SpO2 levels and cognitive impairment (rho = -0.32, p = 0.01), meaning a lower SpO2 was linked with a advanced degree of cognitive dysfunction. The SpO2 levels at various cognitive impairment category are tabulated in Table 3.

Table 3: Comparison of Mean Arterial Oxygen Saturation (SpO_2) Levels Across Cognitive Impairment Categories

Cognitive Impairment	SpO2 (Mean ± SD)
No Cognitive Impairment	94.2 ± 3.3%
Mild Cognitive Impairment (MCI)	92.5 ± 3.1%
Severe Cognitive Impairment	90.1 ± 4.2%

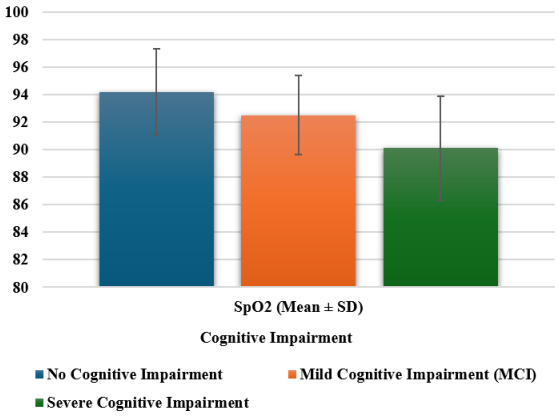

Figure 5: Correlation Between Arterial Oxygen Saturation (SpO_2) and MMSE Scores in COPD Patients

Figure 5 shows a strong adverse connection of arterial oxygen saturation (SpO_2, Y). This also shows that hypoxemia has a negative effect on cognitive health as SpO_2 decreases. The statistical correlation of lower oxygen saturation with greater cognitive dysfunction is supported by the visual downward trend.

4.4 COPD Severity, SpO2, and Cognitive Function

The mean SpO2 levels of patients divided by COPD severity and cognitive impairment are revealed in table 4. In persons with severe COPD and low SpO2 levels, the highest prevalence was observed and patients with mild COPD and normal SpO2 levels had better cognitive function as expected. Patients with mild COPD had a mean SpO2 of 94.3% and cognitive impairment was less common in this group.

Table 4: Association Between COPD Severity, Cognitive Impairment Status, and Mean SpO_2 Levels

COPD Severity	No Cognitive Impairment	Mild Cognitive Impairment (MCI)	Severe Cognitive Impairment	Mean SpO2 (%)
Mild (FEV1 ≥ 80%)	98.1 ± 1.5%	94.3 ± 2.6%	90.5 ± 3.4%	94.3
Moderate (FEV1 50-79%)	95.6 ± 3.1%	92.5 ± 3.2%	89.3 ± 4.5%	92.8
Severe (FEV1 30-49%)	93.3 ± 4.0%	91.2 ± 3.8%	87.6 ± 5.0%	90.5
Very Severe (FEV1 < 30%)	92.1 ± 4.2%	89.6 ± 5.0%	84.2 ± 6.2%	88.3

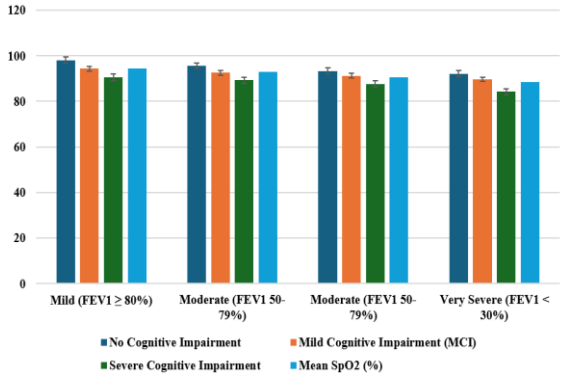

Figure 6: Combined Effect of COPD Severity and Cognitive Impairment on Mean SpO_2 Levels

Figure 6 depicts how mean SpO_2 is related to severity of COPD and cognitive status. The lowest oxygen saturation is found in patients with both advanced COPD and severe cognitive impairment. A continuing figure highlights how the combination is related to compromised arterial oxygenation and highlights the need for early monitoring and intervention.

4.5 Statistical Analysis

Spearman's rank correlation was employed to analyze the association among SpO2 levels and cognitive impairment, which were found to be significantly negative correlated with one another (Spearman's rho = -0.32, p = 0.01). The findings of this investigation support the hypothesis that lower SpO2 levels are related to more cognitive dysfunction in persons with COPD. Moreover, Kruskal-Wali's showed significant differences in cognitive impairment within group of individuals with elevated points of COPD severity (COPD stage, $p < 0.05$) indicating that COPD severity is a major contributor of cognitive disorder.

DISCUSSION

This study demonstrates that how COPD patients frequently experience cognitive impairment and closely related to the COPD severity and O2 saturation status. Some 41% of our COPD patients had measurable cognitive deficits, this being poorer in more advanced stages of COPD. Arterial oxygen saturation (SpO2) was highly inversely associated with cognitive scores, following chronic hypoxemia as a factor associated with cognitive decline in COPD. This is consistent with earlier research that shows that hypoxemia is an important factor in neurocognitive deficits in COPD and poor cognitive functioning worsens as pulmonary function declines (Thakur et al., 2010, Martin et al., 2011, Karamanli et al., 2015). This agrees with the fact that evidence suggests that cognitive deficits in COPD are typically broad and encompass multiple domains (e.g., attention, memory, executive function), and not simply a function of generalized aging alone (Chapko et al., 2018). Our results accord well, though there are some heterogeneous results in previous literature, increasing confidence in an oxygenation–cognition link. The study achieves its main objective by creating a simple association between low SpO2 and poor cognition, and it clearly describes the clinical value of routine pulmonary oxygen saturation monitoring in COPD. Closely surveillance and timely intervention to prevent or

mitigate cognitive decline and help patients with chronically low SpO2. For example, systemic hypoxemia is related with cognitive findings in patients with COPD manifests as hypoxemic COPD and long-term oxygen therapy reported as to manage associated with better cognitive outcomes in affected persons (Thakur et al., 2010; Karamanli et al., 2015). Therefore, we must manage the chronic hypoxemia, not only for end organ survival but also for neurologic well-being. It is also examined that how such patient factors as education level and smoking status may be related to cognitive vulnerability. However, our cross-sectional data did not indicate any statistically significant modifiers but had a trend that patients with less education (an indicator of cognitive reserve) and heavy smokers did seemingly worse on the cognitive tests. In general, this pattern is not definitive for COPD related cognitive impairment but rather indicates limited cognitive reserve and ongoing vascular risk exposure can worsen cognitive impairment coexisting with COPD (Chapko et al., 2018). These interactions would need further research with larger samples to elucidate further and to determine whether some subgroups of COPD patients are particularly at risk for cognitive decline. These results are of great clinical importance. COPD is an often-overlooked comorbidity of cognitive impairment (Berezuk et al., 2021) that may adversely affect disease self-management and quality of life. By adding to growing evidence indicating that routine cognitive assessment should be included in COPD care to identify at risk patients early, our study contributes to the identification of extra challenges for not only COPD patients, but also their caregivers who will be needed to provide them with daily care. Cognitive screenings that consist of very brief tasks could be incorporated into COPD evaluations to identify people who are at risk for cognitive failure or who might be able to benefit from cognitive rehabilitation strategies OR competent support for such measures as adherence to medication or to perform daily tasks. Early detection is especially important. Longitudinal studies are proving that the chance to develop mild cognitive impairment and dementia are higher for COPD patients than for the ones without this disease (Singh et al., 2014; Feinkohl et al., 2017; Xie & Xie, 2019; Wang et al., 2022). Such subtle deficits could be recognised with mid stage disease and so early interventions such as supplemental oxygen; exercise training or other therapy to potentially slow decline could be initiated. Oxygenation and fitness can be improved through pulmonary rehabilitation, and indeed, this may confer cognitive benefits, an area of future study in trials as well. Overall, these findings demonstrate that lower inhaled oxygen saturation were connected with poorer cognitive performance in COPD patients, addressing in essence the key finding question. This work supports the notion that neurologic implications of COPD are related to pulmonary aspects of the disease. In line with a cross-sectional study, the causality needs to be confirmed in future studies with longitudinal designs to find out whether this preserves higher SpO2 or initiating oxygen therapy earlier can prevent further cognitive deterioration. More specifically, advanced neuroimaging studies might also elucidate underlying mechanisms involving how chronic lung disease affects the brain in the COPD hypoxemic patients, because they have shown indication of hippocampal atrophy in COPD hypoxemic affected person (Li et al., 2013). Clinicians should be overall vigilant for cognitive impairment in COPD and comprehensive COPD management should include cognitive health beyond the lung. Research into these outcomes and the development of multidisciplinary involvements to advance poor cognitive consequences and low well being in COPD are ongoing.

CONCLUSION

This study determines the effect of arterial oxygen (SpO2) saturation on cognitive deficits linked to chronic obstructive pulmonary disease patients and how it is a potential risk factor for the development of cognitive dysfunction. Lower SpO2 levels are substantially linked to elevated cognitive impairment, on memory and executive functions. Cognitive impairment is more likely as COPD severity increases, and the most severe cognitive dysfunction is seen in patients with the lowest SpO2 levels. These results therefore establish the significant impact that SpO2 monitoring can have in preventing cognitive decline in COPD patients, in advanced disease stages. By monitoring SpO2 levels in COPD patients, one can not only manage respiratory health, but can also be an early indicator of cognitive dysfunction. Although trials to study or optimize the methodology have not been conducted in the ICU, results from regular SpO2 checks combined with timely interventions, such as oxygen therapy, have shown that significant improvement in cognitive indicators and health-related quality of life in COPD patients can be achieved. In this study, the importance of SpO2 monitoring in COPD management is accentuated due to simultaneous impact on the pulmonary and cognitive involvement of the disease. Regarding the therapeutic approach for COPD patients, SpO2 monitoring must be prioritized to reduce the risk of cognitive dysfunction. Routine SpO2 checks will allow medical professionals to identify individuals who may be at risk of cognitive decline and catch it early before it is lost forever. Future research should explore the links between SpO2 fluctuations and oxygen therapy in trying to prevent cognitive deterioration in terms of a complete management plan for COPD patients that considers pulmonary as well as cognitive health.

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