Original Article

The impact of smoking on respiratory rehabilitation efficacy and correlation analysis in patients with chronic obstructive pulmonary disease: a retrospective study

Mingzhen Li, Wei Gao

Department of Respiratory & Critical Care Medicine, China Rehabilitation Research Center, Beijing, China

Contributions: (I) Conception and design: W Gao; (II) Administrative support: W Gao; (III) Provision of study materials or patients: Both authors; (IV) Collection and assembly of data: M Li; (V) Data analysis and interpretation: Both authors; (VI) Manuscript writing: Both authors; (VII) Final approval of manuscript: Both authors.

Correspondence to: Wei Gao, MM. Department of Respiratory & Critical Care Medicine, China Rehabilitation Research Center, No. 10 Jiaomen North Road, Fengtai District, Beijing 100068, China. Email: rhhuxi@163.com.

Background: Chronic obstructive pulmonary disease (COPD) was a significant public health concern, with smoking being the primary risk factor for its development and progression. The impact of smoking on respiratory rehabilitation efficacy in COPD patients remains an area of interest and investigation. This study aimed to assess the influence of smoking on the efficacy of respiratory rehabilitation in patients with COPD.

Methods: Data of patients with COPD from October 2015 to October 2023 were retrospectively analyzed in this case-control study. The patients who had previously participated in a pulmonary rehabilitation program were excluded. Pulmonary function, exercise capacity, quality of life, and sleep patterns were evaluated before and after rehabilitation.

Results: A total of 40 patients were included and categorized into non-smoking (n=20) and smoking groups (n=20) based on their smoking history. Before rehabilitation, no significant differences were observed between the groups in forced expiratory volume in one second (FEV_1) (P=0.96), forced vital capacity (FVC) (P=0.97), FEV_1/FVC ratio (P=0.73), maximal voluntary ventilation (MVV) (P=0.69), and diffusing capacity of the lung for carbon monoxide (DLCO) (P=0.63). After rehabilitation, FEV_1 (P=0.02), FVC (P=0.009), FEV_1/FVC ratio (P=0.03), MVV (P=0.004), DLCO (P=0.01), these pulmonary functions for non-smokers were much better than the smokers. Similarly, the non-smoking group exhibited significantly greater improvements in 6-minute walk distance (P=0.03), peak oxygen consumption (VO_2) (P=0.01), Borg scale ratings (P=0.02), St. George's Respiratory Questionnaire (SGRQ) scores (P=0.004), and Medical Research Council (MRC) dyspnea scale scores (P=0.005) compared to the smoking group after rehabilitation. The non-smoking patients have more better quality of life compared to the smokers after rehabilitation, which demonstrated by the quality of life scores and Sleep Quality Score, including somatization (P=0.01), emotion management (P=0.009), role play (P=0.008), cognitive function (P=0.04), return to social function (P=0.01), Sleep Quality Score (P=0.02).

Conclusions: Smoking negatively impacts the efficacy of respiratory rehabilitation in COPD patients, leading to poorer pulmonary function, exercise capacity, quality of life, and sleep patterns.

Keywords: Smoking; respiratory rehabilitation; efficacy; chronic obstructive pulmonary disease (COPD)

Submitted Aug 06, 2024. Accepted for publication Nov 22, 2024. Published online Jan 22, 2025.
doi: 10.21037/jtd-24-1267
View this article at: https://dx.doi.org/10.21037/jtd-24-1267

Introduction

Chronic obstructive pulmonary disease (COPD) was a prevalent and debilitating respiratory condition characterized by airflow limitation and persistent respiratory symptoms (1-3). COPD represents a significant public health burden, contributing to substantial morbidity, mortality, and healthcare costs worldwide (4,5). Smoking was the primary risk factor for the development and progression of COPD, with approximately 85–90% of cases attributed to tobacco smoke exposure (6-8). While the pathophysiological relationship between smoking and COPD was well-established, the impact of smoking on the efficacy of respiratory rehabilitation and its comprehensive implications for patients with COPD remain a topic of continuing interest and investigation.

Pulmonary rehabilitation plays a pivotal role in the management of COPD, encompassing a multidisciplinary approach designed to optimize respiratory function, alleviate symptoms, and enhance patients' overall quality of life (9-11). The rehabilitation interventions typically include exercise training, disease education, nutritional guidance, psychological support, and behavioral interventions, tailored to individual patient needs and clinical characteristics (12,13). Despite the established benefits of pulmonary rehabilitation in improving exercise capacity, symptom control, and health-related quality of life, several challenges persist in optimizing its efficacy and accessibility for all COPD patients. Key among these are patient adherence, availability of resources, and individual patient characteristics that may affect rehabilitation outcomes (14,15). The pathophysiological relationship between smoking and COPD is well-established (16), but the impact of smoking on the efficacy of pulmonary rehabilitation remains underexplored. Smoking can exacerbate inflammation, oxidative stress, and tissue damage in the lungs, potentially hindering the beneficial effects of exercise training and other rehabilitation interventions (17). Moreover, smoking has been shown to interfere with patients' motivation, adherence to rehabilitation programs, and their ability to achieve optimal outcomes (18). Given the high prevalence of smoking among COPD patients and the potential for smoking to modulate the physiological responses to pulmonary rehabilitation, there is a compelling need to investigate how smoking influences the effectiveness of these interventions.

This retrospective case-control study aimed to systematically evaluate the impact of smoking on the efficacy of pulmonary rehabilitation in patients with COPD. Through a comprehensive assessment of pulmonary function, exercise capacity, quality of life, and sleep patterns before and after rehabilitation, this study will contribute to a more nuanced understanding of the multifaceted relationship between smoking and COPD management. The findings from this study will be instrumental in tailoring rehabilitation strategies to better meet the needs of smoking COPD patients and in advocating for integrated smoking cessation programs as an essential component of pulmonary rehabilitation. We present this article in accordance with the STROBE reporting checklist (available at https://jtd.amegroups.com/article/view/10.21037/jtd-24-1267/rc).

Highlight box

Key findings

- Non-smokers showed significant improvements in forced expiratory volume in one second (FEV_1), forced vital capacity (FVC), FEV_1/FVC ratio, maximal voluntary ventilation, and diffusing capacity of the lung for carbon monoxide compared to smokers after rehabilitation.
- Non-smokers demonstrated greater enhancements in 6-minute walk distance and peak oxygen consumption post-rehabilitation compared to smokers.
- Non-smokers had significantly better scores in St. George's Respiratory Questionnaire, Medical Research Council dyspnea scale, and sleep quality assessments compared to smokers following rehabilitation.

What is known and what is new?

- Smoking induces chronic inflammation, oxidative stress, premature aging, and immune dysfunction in the lungs, leading to progressive damage of the airway epithelium, vascular endothelium, small airways, and alveoli, characteristic of chronic obstructive pulmonary disease (COPD).
- Smoking cessation as a critical factor in achieving optimal outcomes in pulmonary rehabilitation programs, influencing both physiological improvements and quality of life enhancements among patients with respiratory conditions.

What is the implication, and what should change now?

- Smoking significantly diminishes the effectiveness of respiratory rehabilitation in COPD patients, resulting in inferior improvements in pulmonary function, exercise capacity, quality of life, and sleep patterns compared to non-smokers.

Methods

Study design

This study was a retrospective case-control study. It involved patients with COPD who were treated at our

hospital from October 2015 to October 2023. Patients were categorized into non-smoking and smoking groups based on their smoking status. The smoking group consisted of individuals with a smoking history of 40 pack-years or more, while the non-smoking group included COPD patients with no history of smoking.

The study was conducted in accordance with the Declaration of Helsinki (as revised in 2013). The study was approved by institutional Committee Ethics board of China Rehabilitation Research Center (No. 2024-091-3) and individual consent for this retrospective analysis was waived.

Inclusion and exclusion criteria

Inclusion criteria: (I) patients were 40 years of age or older; (II) with a diagnosis (19) of COPD with severe airflow limitation [confirmed by a post-bronchodilator forced expiratory volume in one second (FEV_1)/forced vital capacity (FVC) ratio <0.70 and a post-bronchodilator FEV_1, of ≤50% predicted]; (III) symptoms of chronic bronchitis; (IV) with normal cognitive functions; (V) stable vital signs; (VI) complete medical records; (VII) in the stable phase of COPD.

Exclusion criteria: (I) patients had a COPD exacerbation that was ongoing during the baseline period, or had a diagnosis of asthma or other major lung disease, such as lung cancer, bronchiectasis and interstitial lung disease; (II) existence of any pathology which makes it inadvisable to perform spirometry; (III) patients who, due to their intellectual disability or other psychiatric disorder, do not understand study procedures or cannot adequately perform spirometry; (IV) abnormal heart, liver, and kidney function; (V) malignant tumor or marked impairment of limb function; (VI) severe nutritional imbalance; (VII) previous participation in a pulmonary rehabilitation program.

The demographic characteristics of the two groups of patients strictly adhered to the aforementioned inclusion and exclusion criteria, and no other matching techniques were used.

Intervention

Rehabilitation protocol for both patient groups included standardized stable-phase medication, including correct use of inhalers and targeted medication for expectoration and cough relief. The comprehensive pulmonary rehabilitation program involved the following elements: (I) disease education encompassing smoking cessation, oxygen therapy, understanding and self-management of COPD, correct medication usage, and scientifically balanced diet, as well as pulmonary rehabilitation concepts; (II) respiratory training, primarily consisting of diaphragmatic and pursed-lip breathing, each lasting 15 minutes, three times a day; (III) exercise training, combining endurance and strength exercises with lower limb exercises as the core training method. Exercise cardiopulmonary function tests for both patient groups were conducted by utilizing German Jerger. Based on the results of the patients' exercise cardiopulmonary function tests, as well as their age and gender, moderate- to high-intensity aerobic exercise was selected with a target training intensity of 65–85% of the target heart rate. A rational exercise prescription was devised, and patients were guided to engage in flat panel exercises (Jaeger, Germany) for 30 minutes, three times a week; (IV) nutritional assessment and guidance with a focus on calorie intake and appropriate dietary guidance to ensure the consumption of adequate levels of protein, vitamins, and trace elements; (V) psychological assessment and counseling. The pulmonary rehabilitation program lasted for 8 weeks.

General information

Patient general information was obtained through systematic retrieval of medical records, including age, gender, body mass index (BMI), smoking index, drinking history, hypertension, diabetes, hyperlipidemia, disease duration, educational status, employment status, living environment, respiratory symptoms and history of respiratory diseases.

Pulmonary function tests

The pulmonary function of both patient groups was assessed before and after rehabilitation using a spirometer. The baseline and post-rehabilitation measurements for FEV_1, FVC, the FEV_1/FVC ratio, maximal voluntary ventilation (MVV), and diffusing capacity of the lung for carbon monoxide (DLCO) were recorded.

Exercise capacity

Six-minute walk distance (6MWD) (20): a straight line was marked on a flat surface, and in strict accordance with the guidelines for the 6-minute walk test, the 6MWDs before rehabilitation (baseline) and at the end of the rehabilitation

course were measured for both patient groups. A greater distance indicates greater exercise tolerance and better lung function. Additionally, dyspnea and leg fatigue were assessed using the Borg scale immediately after the 6MWD assessment. The Borg scores were ranging from 0 to 10. A higher score indicates a more severe level of breathing difficulty and fatigue.

VO_2 was measured for both patient groups before and after rehabilitation training. Peak VO_2 was defined as the maximum oxygen intake during exercise measured using a cardiopulmonary exercise testing system (MasterScreenCPX, Jaeger, Germany). The Ramp protocol was employed, and a maximal exercise test was chosen. The incremental load was selected based on the patient's gender, age, height, weight, and physical activity level, increasing until the participant could no longer maintain the cycling cadence (55–65 revolutions per minute) or reached their age-predicted maximum heart rate (220 – age) or other criteria for test termination were met (21).

The Medical Research Council (MRC) dyspnea scale was calculated based on the modified MRC dyspnea scale criteria. A lower score indicates a lower level of breathlessness.

St. George's Respiratory Questionnaire (SGRQ) score

The SGRQ was used to assess the quality of life for patients before and after rehabilitation treatment. The SGRQ comprises three domains: (I) symptoms, including cough, sputum production, and breathlessness; (II) activity, involving limitations in climbing stairs, dressing, domestic chores, and recreational activities; and (III) impacts, encompassing anxiety, distress, impact on social activities, and feelings of insecurity and despair. The scores for each domain as well as the total score were calculated, with a score range of 0 to 100. A lower score indicates better health status. The Cronbach's α coefficient for internal consistency reliability was calculated as 0.905 (22).

Quality of life score

Statistical analysis was managed using the European Organization for Research and Treatment of Cancer Quality of Life Questionnaire (EORTC QLQ-C30), which comprises five functional dimensions (bodily, moving, character, social, and cognitive functions). Each item in the various dimensions was rated on a 1–7 Likert scale, with total scores ranging from 0 to 100, reflecting a positive correlation with quality of life. The Cronbach's α coefficient was 0.927 (23).

Pittsburgh Sleep Quality Index (PSQI)

Participants completed the PSQI. The PSQI was a self-report assessment tool that evaluates sleep quality over a 1-month period. A global score and seven component scores can be derived from the scale. The component scores were the following: Subjective sleep quality, sleep latency, sleep duration, sleep efficiency, sleep disturbances, use of sleeping medications and daytime dysfunction. Each component was scored on a scale from 0–3, with the total score ranging from 0–21; where a higher score describes poorer sleep quality. A total PSQI score greater than 5 has been validated as being highly sensitive and specific in distinguishing good from poor sleepers across a number of populations. The Cronbach's α coefficient of the PSQI was 0.78 (24).

Sample size

Using G*Power 3.1.9.7, based on the "t tests: means – difference between two independent means (two groups)" option, we selected an a priori analysis with the following settings: two-tailed mode, effect size d =0.92, α error probability =0.05, power (1 – β error probability) =0.8. The calculation resulted in a minimum sample size of 20 cases per group.

Statistical analysis

The data were analyzed using SPSS 29.0 statistical software (SPSS Inc., Chicago, IL, USA). For categorical data, [n (%)] was used for representation. The Chi-squared test was applied with the basic formula when the sample size was ≥40 and the theoretical frequency T was ≥5, with the test statistic represented by χ^2. When the sample size was ≥40 but the theoretical frequency $1 \leq T < 5$, the Chi-squared test was adjusted using the correction formula. In cases where the sample size was <40 or the theoretical frequency T<1, statistical analysis was conducted using Fisher's exact probability method. Continuous variables were first tested for normal distribution using the Shapiro-Wilk method. For normally distributed continuous data, the format [mean ± standard deviation (SD)] was employed. Non-normally distributed data was analyzed using Wilcoxon rank-sum test, and the [median (25% quantile, 75% quantile)] was used for presentation. P<0.05 were considered as statistical significance.

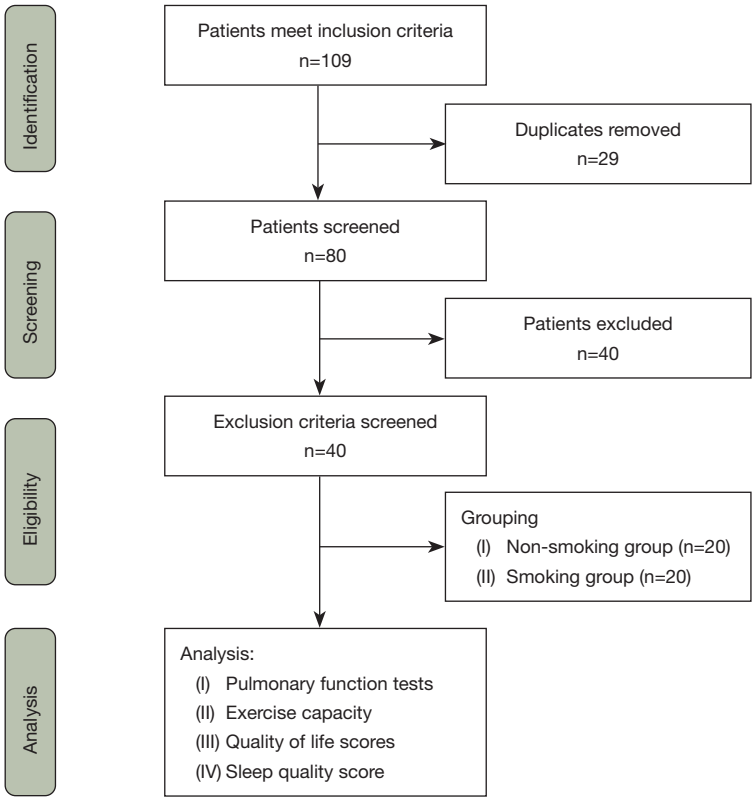

Figure 1 Patient selection flowchart.

Results

General information

Forty patients with COPD were included in this retrospective study and equally divided into the non-smoking group (n=20) and the smoking group (n=20) (*Figure 1*). There were no statistically significant differences between the two groups in terms of age (P=0.43), gender distribution (P=0.74), BMI (P=0.46), drinking history (P=0.69), comorbidities including hypertension (P=0.69), diabetes (P=0.45), hyperlipidemia (P=0.66), disease duration (P=0.37), educational status (P=0.74), employment status (P=0.45), living environment (P=0.74), respiratory symptoms (P=0.74), and history of respiratory diseases (P=0.74) (*Table 1*). The average smoking packs per year in the smoking group was 47.71±4.85 packs, showing a significant difference compared to the non-smoking group (P<0.001).

Pulmonary function tests before and after rehabilitation

The pulmonary function tests before rehabilitation revealed no statistically significant differences between the non-smoking group and the smoking group in terms of FEV_1 (P=0.96), FVC (P=0.97), FEV_1/FVC ratio (P=0.73), MVV (P=0.69), and DLCO (P=0.63) (*Table 2*). The pulmonary function tests after rehabilitation demonstrated statistically significant differences between the non-smoking group and the smoking group in FEV_1 (2.14±0.48 *vs.* 1.83±0.25, *t*=2.598, P=0.02), FVC (3.48±0.55 *vs.* 3.05±0.43, *t*=2.741, P=0.009), FEV_1/FVC ratio (62.14±6.56 *vs.* 57.26±7.05, *t*=2.265, P=0.03), MVV (84.23±8.07 *vs.* 76.84±7.01, *t*=3.090, P=0.004), and DLCO (24.17±2.58 *vs.* 21.95±2.86, *t*=2.573, P=0.01).

Exercise capacity before and after rehabilitation

The exercise capacity assessments before rehabilitation revealed no statistically significant differences between the non-smoking group and the smoking group in terms of the 6MWD (P=0.91), VO_2peak (P=0.40), Borg scale rating (P=0.10), SGRQ score (P=0.27), and MRC dyspnea scale score (P=0.27) (*Table 3*). The exercise capacity assessments after rehabilitation demonstrated statistically significant

Table 1 General information

Parameter	Non-smoking group (n=20)	Smoking group (n=20)	t/χ^2	P value
Age (years)	65.72±6.34	64.18±5.92	t=0.792	0.43
Gender (M/F)	12 [60]/8 [40]	14 [70]/6 [30]	t=0.110	0.74
BMI (kg/m^2)	22.38±2.14	22.86±1.93	t=0.749	0.46
Smoking index (pack/years)	0.00±0.00	47.71±4.85	t=43.993	<0.001
Drinking history	3 [15]	5 [25]	χ^2=0.156	0.69
Comorbidities				
Hypertension	5 [25]	3 [15]	χ^2=0.156	0.69
Diabetes	6 [30]	3 [15]	χ^2=0.573	0.45
Hyperlipidemia	4 [20]	2 [10]	χ^2=0.196	0.66
Disease duration (years)	8.25±3.12	9.36±4.45	χ^2=0.909	0.37
Educational status			χ^2=0.1101	0.74
Junior high and below	14 [70]	12 [60]		
Junior high school or above	6 [30]	8 [40]		
Employment status			χ^2=1.600	0.45
Employed	12 [60]	8 [40]		
Unemployed	4 [20]	6 [30]		
Retired	4 [20]	6 [30]		
Living environment			χ^2=0.110	0.74
Urban	14 [70]	12 [60]		
Rural	6 [30]	8 [40]		
Respiratory symptoms	12 [60]	14 [70]	χ^2=0.110	0.74
History of respiratory diseases	6 [30]	8 [40]	χ^2=0.110	0.74

Data are presented as mean ± standard deviation or n [%]. M, male; F, female; BMI, body mass index.

Table 2 Pulmonary function tests before and after rehabilitation

Parameter	Before/after rehabilitation	Non-smoking group (n=20)	Smoking group (n=20)	t	P value
FEV_1 (L)	Before rehabilitation	1.64±0.24	1.65±0.27	0.050	0.96
	After rehabilitation	2.14±0.48	1.83±0.25	2.598	0.02
FVC (L)	Before rehabilitation	2.97±0.46	2.97±0.35	0.039	0.97
	After rehabilitation	3.48±0.55	3.05±0.43	2.741	0.009
FEV_1/FVC (%)	Before rehabilitation	56.25±3.32	55.83±4.15	0.348	0.73
	After rehabilitation	62.14±6.56	57.26±7.05	2.265	0.03
MVV (L/min)	Before rehabilitation	73.51±7.21	72.62±6.82	0.402	0.69
	After rehabilitation	84.23±8.07	76.84±7.01	3.090	0.004
DLCO (mmol/min/kPa)	Before rehabilitation	20.56±2.35	20.18±2.59	0.480	0.63
	After rehabilitation	24.17±2.58	21.95±2.86	2.573	0.01

Data are presented as mean ± standard deviation. FEV_1, forced expiratory volume in one second; FVC, forced vital capacity; MVV, maximum voluntary ventilation; DLCO, diffusing capacity of the lung for carbon monoxide.

Table 3 Exercise capacity before and after rehabilitation

Parameter	Before/after rehabilitation	Non-smoking group (n=20)	Smoking group (n=20)	*t*	P value
6MWD (m)	Before rehabilitation	341.26±20.15	340.61±15.38	0.115	0.91
	After rehabilitation	410.27±25.48	392.45±24.58	2.251	0.03
VO_2peak (mL/min/kg)	Before rehabilitation	14.58±1.26	14.27±1.04	0.846	0.40
	After rehabilitation	17.83±2.56	15.83±2.26	2.625	0.01
Borg scale	Before rehabilitation	3.96±0.65	4.29±0.58	1.680	0.10
	After rehabilitation	2.96±0.89	3.56±0.64	2.445	0.02
SGRQ score	Before rehabilitation	63.15±5.27	65.38±7.16	1.122	0.27
	After rehabilitation	52.48±6.58	58.62±6.15	3.048	0.004
MRC dyspnea scale	Before rehabilitation	2.37±0.37	2.51±0.43	1.110	0.27
	After rehabilitation	1.89±0.26	2.39±0.67	3.110	0.005

Data are presented as mean ± standard deviation. 6MWD, six-minute walk distance; VO_2, oxygen consumption; SGRQ, St. George's Respiratory Questionnaire; MRC, Medical Research Council.

Table 4 Quality of life scores and sleep quality score

Parameter	Non-smoking group (n=20)	Smoking group (n=20)	*t*	P value
Somatization	77.03±6.02	71.72±6.98	2.574	0.01
Emotion management	78.14±7.78	71.14±8.26	2.759	0.009
Role play	79.24±8.23	72.41±7.24	2.784	0.008
Cognitive function	76.21±5.11	72.73±5.02	2.171	0.04
Return to social function	77.23±5.78	72.07±6.23	2.721	0.01
Sleep quality score	7.89±2.21	10.03±3.34	2.397	0.02

Data are presented as mean ± standard deviation.

differences between the non-smoking group and the smoking group in terms of the 6MWD (410.27±25.48 *vs.* 392.45±24.58, *t*=2.251, P=0.03), peak VO_2peak (17.83±2.56 *vs.* 15.83±2.26, *t*=2.625, P=0.01), Borg scale rating (2.96±0.89 *vs.* 3.56±0.64, *t*=2.445, P=0.02), SGRQ score (52.48±6.58 *vs.* 58.62±6.15, *t*=3.048, P=0.004), and MRC dyspnea scale score (1.89±0.26 *vs.* 2.39±0.67, *t*=3.110, P=0.005).

Quality of life scores and Sleep Quality Score

The comparison of quality of life scores between the non-smoking group and the smoking group revealed statistically significant differences in somatization (77.03±6.02 *vs.* 71.72±6.98, *t*=2.574, P=0.01), emotion management (78.14±7.78 *vs.* 71.14±8.26, *t*=2.759, P=0.009), role play (79.24±8.23 *vs.* 72.41±7.24, *t*=2.784, P=0.008), cognitive function (76.21±5.11 *vs.* 72.73±5.02, *t*=2.171, P=0.04), and return to social function (77.23±5.78 *vs.* 72.07±6.23, *t*=2.721, P=0.01) (*Table 4*). The comparison of PSQI scores between the non-smoking group and the smoking group revealed a statistically significant difference in sleep quality (7.89±2.21 *vs.* 10.03±3.34, *t*=2.397, P=0.02).

Discussion

COPD was a leading cause of morbidity and mortality globally, with smoking being a key risk factor for its development and progression (25-27). Our retrospective case-control study demonstrated significant disparities in pulmonary function, exercise capacity, quality of life, and sleep quality between non-smoking and smoking COPD

patients following rehabilitation. Specifically, non-smokers exhibited significantly greater improvements in FEV_1, FVC, FEV_1/FVC ratio, MVV, and DLCO compared to smokers in post-rehabilitation. Non-smokers also achieved significantly greater improvements in exercise capacity, as evidenced by the 6MWD, peak VO_2peak, Borg scale ratings, and the MRC dyspnea scale score. These findings underscore the adverse impact of smoking on respiratory rehabilitation efficacy and comprehensive COPD management.

Pulmonary function tests play a crucial role in evaluating the severity and progression of COPD. Our study demonstrated that while there were no significant differences in baseline pulmonary function parameters between smokers and non-smokers, significant disparities emerged after rehabilitation. Non-smokers exhibited significantly greater improvements in FEV_1, FVC, FEV_1/FVC ratio, MVV, and DLCO compared to smoker's post-rehabilitation. These findings were consistent with previous research indicating that smoking negatively impacts lung function and can impede the effectiveness of interventions aimed at improving respiratory outcomes (28,29). The underlying mechanisms likely involve the chronic inflammation and oxidative stress induced by smoking, which can lead to structural changes in the airways and alveoli, impairing lung function and reducing the responsiveness to rehabilitation efforts (30).

Exercise capacity was another critical aspect of COPD management and was closely linked to overall functional status. Our study revealed that non-smokers achieved significantly greater improvements in exercise capacity, as evidenced by the 6MWD, peak VO_2peak, Borg scale ratings, and the MRC dyspnea scale score after rehabilitation. These results are in line with previous studies that have reported similar findings. For instance, Soumagne *et al.* found that non-smokers with COPD showed greater improvements in exercise capacity following pulmonary rehabilitation compared to smokers (31). These results highlight the adverse impact of smoking on patients' physical functional status and the efficacy of respiratory rehabilitation programs. Furthermore, our findings regarding the SGRQ scores provide additional support for the notion that smoking hinders the improvement of respiratory symptoms and overall quality of life in COPD patients undergoing rehabilitation. The diminished exercise capacity in smokers may be due to reduced lung function, increased airway resistance, and decreased oxygen transport efficiency, all of which are exacerbated by smoking. Furthermore, smoking-induced systemic inflammation and muscle wasting may contribute to the reduced exercise capacity and rehabilitation outcomes (17).

In addition to the detrimental effects of smoking on physiological parameters, our study also identified significant differences in quality of life scores between smokers and non-smokers. Non-smokers demonstrated better outcomes across various domains of the quality of life assessment, including somatization, emotion management, role play, cognitive function, and return to social function. These findings are consistent with previous research (32), which has reported that smoking is associated with poorer quality of life in COPD patients. For example, Papadopoulos *et al.* found that smoking was a significant predictor of lower quality of life scores in COPD patients, particularly in the domains of physical function and emotional well-being (33). Similarly, Postolache *et al.* observed that smoking cessation led to improvements in health-related quality of life, including reduced symptoms and improved daily functioning (34). These findings underscore the substantial impact of smoking on the overall well-being and functional capacity of COPD patients, further underscoring the importance of smoking cessation interventions as integral components of comprehensive COPD management programs.

Furthermore, our study revealed a notable difference in sleep quality between smokers and non-smokers, with non-smokers exhibiting better Sleep Quality Scores. Sleep disturbances were prevalent in COPD patients and can significantly impact disease management and overall health (35-37). The observed association between smoking and sleep quality in our study underscores the multifaceted nature of the impact of smoking on COPD and its implications for holistic patient care. Therefore, addressing sleep disturbances and promoting healthy sleep patterns should be acknowledged as essential components of comprehensive COPD rehabilitation and management programs.

This study had several strengths, including its rigorous retrospective case-control design, comprehensive assessment of physiological parameters, and robust statistical analysis. However, certain limitations should be considered when interpreting the results. The relatively small sample size and the single-center nature of the study may limit the generalizability of the findings. Additionally, the retrospective design inherently introduces the potential for selection bias and may not account for all confounding variables. Future studies with larger sample sizes and prospective, multi-center designs were warranted to further

validate and expand upon our findings. Moreover, future studies will use two-way analysis of variance (ANOVA) to investigate the interaction between smoking and rehabilitation interventions before and after treatment, to gain a clearer understanding of the impact of smoking.

Conclusions

Our study provides compelling evidence of the adverse impact of smoking on respiratory rehabilitation efficacy and comprehensive COPD management. The observed associations between smoking status, pulmonary function, exercise capacity, quality of life, and sleep quality underscore the complex interplay between smoking and COPD outcomes. These findings emphasize the need for targeted smoking cessation interventions and comprehensive support for COPD patients to improve rehabilitation outcomes and overall well-being. By addressing the multifaceted impact of smoking on COPD management, healthcare providers can optimize interventions and enhance the effectiveness of respiratory rehabilitation programs, ultimately improving the quality of care for patients with COPD.

Acknowledgments

None.

Footnote

Reporting Checklist: The authors have completed the STROBE reporting checklist. Available at https://jtd.amegroups.com/article/view/10.21037/jtd-24-1267/rc

Data Sharing Statement: Available at https://jtd.amegroups.com/article/view/10.21037/jtd-24-1267/dss

Peer Review File: Available at https://jtd.amegroups.com/article/view/10.21037/jtd-24-1267/prf

Funding: None.

Conflicts of Interest: Both authors have completed the ICMJE uniform disclosure form (available at https://jtd.amegroups.com/article/view/10.21037/jtd-24-1267/coif). The authors have no conflicts of interest to declare.

Ethical Statement: The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. The study was conducted in accordance with the Declaration of Helsinki (as revised in 2013). The study was approved by institutional Committee Ethics board of China Rehabilitation Research Center (No. 2024-091-3) and individual consent for this retrospective analysis was waived.

References

1. Agustí A, Vogelmeier C, Faner R. COPD 2020: changes and challenges. Am J Physiol Lung Cell Mol Physiol 2020;319:L879-83.
2. Sandelowsky H, Weinreich UM, Aarli BB, et al. COPD - do the right thing. BMC Fam Pract 2021;22:244.
3. Christenson SA, Smith BM, Bafadhel M, et al. Chronic obstructive pulmonary disease. Lancet 2022;399:2227-42.
4. Agusti A, Böhm M, Celli B, et al. GOLD COPD DOCUMENT 2023: a brief update for practicing cardiologists. Clin Res Cardiol 2024;113:195-204.
5. Guo P, Li R, Piao TH, et al. Pathological Mechanism and Targeted Drugs of COPD. Int J Chron Obstruct Pulmon Dis 2022;17:1565-75.
6. Fu YS, Kang N, Yu Y, et al. Polyphenols, flavonoids and inflammasomes: the role of cigarette smoke in COPD. Eur Respir Rev 2022;31:220028.
7. Song Q, Chen P, Liu XM. The role of cigarette smoke-induced pulmonary vascular endothelial cell apoptosis in COPD. Respir Res 2021;22:39.
8. Su X, Wu W, Zhu Z, et al. The effects of epithelial-mesenchymal transitions in COPD induced by cigarette smoke: an update. Respir Res 2022;23:225.
9. Hansen H, Bieler T, Beyer N, et al. Supervised pulmonary tele-rehabilitation versus pulmonary rehabilitation in severe COPD: a randomised multicentre trial. Thorax 2020;75:413-21.
10. He W, Wang J, Feng Z, et al. Effects of exercise-based pulmonary rehabilitation on severe/very severe COPD: a

systematic review and meta-analysis. Ther Adv Respir Dis 2023;17:17534666231162250.
11. Frei A, Radtke T, Dalla Lana K, et al. Effectiveness of a Long-term Home-Based Exercise Training Program in Patients With COPD After Pulmonary Rehabilitation: A Multicenter Randomized Controlled Trial. Chest 2022;162:1277-86.
12. Machado A, Matos Silva P, Afreixo V, et al. Design of pulmonary rehabilitation programmes during acute exacerbations of COPD: a systematic review and network meta-analysis. Eur Respir Rev 2020;29:200039.
13. Nolan CM, Polgar O, Schofield SJ, et al. Pulmonary Rehabilitation in Idiopathic Pulmonary Fibrosis and COPD: A Propensity-Matched Real-World Study. Chest 2022;161:728-37.
14. Ammous O, Feki W, Lotfi T, et al. Inspiratory muscle training, with or without concomitant pulmonary rehabilitation, for chronic obstructive pulmonary disease (COPD). Cochrane Database Syst Rev 2023;1:CD013778.
15. Rutkowski S, Rutkowska A, Kiper P, et al. Virtual Reality Rehabilitation in Patients with Chronic Obstructive Pulmonary Disease: A Randomized Controlled Trial. Int J Chron Obstruct Pulmon Dis 2020;15:117-24.
16. Hopkinson NS. COPD, smoking, and social justice. Lancet Respir Med 2022;10:428-30.
17. Caliri AW, Tommasi S, Besaratinia A. Relationships among smoking, oxidative stress, inflammation, macromolecular damage, and cancer. Mutat Res Rev Mutat Res 2021;787:108365.
18. Gaalema DE, Khadanga S, Pack QR. Clinical challenges facing patient participation in cardiac rehabilitation: cigarette smoking. Expert Rev Cardiovasc Ther 2023;21:733-45.
19. Agustí A, Celli BR, Criner GJ, et al. Global Initiative for Chronic Obstructive Lung Disease 2023 Report: GOLD Executive Summary. Eur Respir J 2023;61:2300239.
20. Matos Casano HA, Anjum F. Six-Minute Walk Test. StatPearls. Treasure Island (FL) ineligible companies. StatPearls Publishing Copyright © 2024, StatPearls Publishing LLC.; 2024.
21. Ma X, Yan X. Monitoring of Maximum Oxygen Intake of Breathing and Heart Rate in Exercise Training Based on Regression Equations. J Healthc Eng 2022;2022:5025615.
22. Suzuki A, Kondoh Y, Swigris JJ, et al. Performance of the St George's Respiratory Questionnaire in patients with connective tissue disease-associated interstitial lung disease. Respirology 2018. [Epub ahead of print]. doi: 10.1111/resp.13293.
23. Jassim G, AlAnsari A. Reliability and Validity of the Arabic Version of the EORTC QLQ-C30 and QLQ-BR23 Questionnaires. Neuropsychiatr Dis Treat 2020;16:3045-52.
24. Zhang C, Zhang H, Zhao M, et al. Reliability, Validity, and Factor Structure of Pittsburgh Sleep Quality Index in Community-Based Centenarians. Front Psychiatry 2020;11:573530.
25. Hou W, Hu S, Li C, et al. Cigarette Smoke Induced Lung Barrier Dysfunction, EMT, and Tissue Remodeling: A Possible Link between COPD and Lung Cancer. Biomed Res Int 2019;2019:2025636.
26. Serrano Gotarredona MP, Navarro Herrero S, Gómez Izquierdo L, et al. Smoking-related interstitial lung disease. Radiologia (Engl Ed) 2022;64 Suppl 3:277-89.
27. Xiong J, Zhou L, Tian J, et al. Cigarette Smoke-Induced Lymphoid Neogenesis in COPD Involves IL-17/RANKL Pathway. Front Immunol 2020;11:588522.
28. Au Yeung SL, Li AM, He B, et al. Association of smoking, lung function and COPD in COVID-19 risk: a two-step Mendelian randomization study. Addiction 2022;117:2027-36.
29. Jamieson E, Korologou-Linden R, Wootton RE, et al. Smoking, DNA Methylation, and Lung Function: a Mendelian Randomization Analysis to Investigate Causal Pathways. Am J Hum Genet 2020;106:315-26.
30. Lugg ST, Scott A, Parekh D, et al. Cigarette smoke exposure and alveolar macrophages: mechanisms for lung disease. Thorax 2022;77:94-101.
31. Soumagne T, Guillien A, Roche N, et al. Never-smokers with occupational COPD have better exercise capacities and ventilatory efficiency than matched smokers with COPD. J Appl Physiol (1985) 2020;129:1257-66.
32. Halms T, Strasser M, Hasan A, et al. Smoking and quality of life in lung cancer patients: systematic review. BMJ Support Palliat Care 2024;13:e686-94.
33. Papadopoulos G, Vardavas CI, Limperi M, et al. Smoking cessation can improve quality of life among COPD patients: validation of the clinical COPD questionnaire into Greek. BMC Pulm Med 2011;11:13.
34. Postolache P, Nemeș RM, Petrescu O, et al. Smoking cessation, pulmonary rehabilitation and quality of life at smokers with COPD. Rev Med Chir Soc Med Nat Iasi 2015;119:77-80.
35. Barata PI, Marc MS, Tudorache E, et al. Self-reported sleep disturbance and mild cognitive impairment in

COPD patients with severe airflow limitation. Clin Respir J 2021;15:808-14.
36. Kim I, Kapella MC, Collins EG, et al. Sleep disturbance and next-day physical activity in COPD patients. Geriatr Nurs 2020;41:872-7.
37. McNicholas WT, Hansson D, Schiza S, et al. Sleep in chronic respiratory disease: COPD and hypoventilation disorders. Eur Respir Rev 2019;28:190064.

Cite this article as: Li M, Gao W. The impact of smoking on respiratory rehabilitation efficacy and correlation analysis in patients with chronic obstructive pulmonary disease: a retrospective study. J Thorac Dis 2025;17(1):254-264. doi: 10.21037/jtd-24-1267