ORIGINAL RESEARCH

Value of Comprehensive Rehabilitation Therapy in Patients with Chronic Obstructive Pulmonary Disease and its Effect on Improving Inflammation

Zheng Li, MM; Xuesong Yan, MM; Jin Liu, MM; Xin Feng, MM; Xiangyun Li, MM

ABSTRACT

Objective • To investigate the value of comprehensive rehabilitation therapy in chronic obstructive pulmonary disease (COPD) and its effect on improvement in patients' inflammation.

Methods • A total of 174 patients with acute COPD exacerbation in the Affiliated Hospital of Hebei University in China from March 2020 to January 2022 were selected as research subjects. They were divided into control, acute and stable groups according to the random number table method (n=58 in each group). The control group was given conventional treatment; the acute group started comprehensive rehabilitation treatment in the acute phase; in the stable group, comprehensive rehabilitation treatment was initiated in the stable period after the condition was stabilized with conventional treatment. Tumor necrosis factor-α (TNF-α), high-sensitivity C-reactive protein (hs-CRP), interleukin-6 (IL-6) and pulmonary function were measured before and after treatment, with the forced expiratory volume in 1 second (FEV1), forced expiratory volume in 1 second/forced vital capacity (FEV1/FVC) ratio, and peak expiratory flow rate (PEF). A 6-min walk test (6MWD) was performed on the patient, and the ability to perform activities of daily living (ADL), self-rating anxiety scale (SAS) and self-rating depression scale (SDS) were used to assess patients' ability to perform ADL and psychology. Finally, the incidence of adverse events (AEs) in patients was recorded and a quality of life (QoL) survey was completed.

Results • The 6MWD test, ADL, FEV1, FEV1/FVC and PEF were elevated in the acute and stable groups compared with the control group, whereas shortness of breath, TNF-α, hs-CRP and IL-6 were decreased ($P < .05$). SAS and SDS scores were reduced in the acute and stable groups after treatment ($P < .05$), while there was no change in the control group ($P > .05$). In addition, QoL was higher in the acute and stable groups ($P < .05$). The improvement of all indicators in the acute group was better than in the stable group ($P < .05$).

Conclusion • Comprehensive rehabilitation therapy can improve the exercise capacity and lung function of patients with COPD, reduce inflammation, and improve patients' negative psychology. (*Altern Ther Health Med.* 2023;29(4):170-176).

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INTRODUCTION

Chronic obstructive pulmonary disease (COPD) is a lung disease caused by airflow restriction resulting in difficulty breathing, usually associated with significant exposure to harmful particles or noxious gases such as cigarette smoke.[1] It is non-infectious and has some genetic predisposition.[2] COPD occurs most often in middle age, with acute exacerbations common in the cold autumn and winter months.[3] Currently, China alone has accumulated nearly 100 million patients with COPD. Globally, COPD has become a major chronic disease on par with hypertension and diabetes, resulting in a huge clinical burden.[4,5] Not only that, but COPD was ranked as the third leading cause of death worldwide in 2020 as assessed by the Global Burden of Disease Research Project.[6] It is also because of the high morbidity and mortality of COPD that it has now become one of the key clinical research foci.

The principles of COPD treatment are based on relieving symptoms, improving exercise tolerance, improving patients' QoL and controlling disease progression.[7] At the same time,

as a complex disease affected by multiple causative factors, the treatment of COPD is also an extremely complex process.[8] Research has shown that a single treatment modality is no longer sufficient to meet the rehabilitation needs of patients and that a holistic intervention is needed to address patients' disease factors.[9] Therefore, the application of comprehensive rehabilitation therapy in COPD has gradually received clinical attention in recent years.[10] Comprehensive rehabilitation therapy consists of providing patients with relatively complete interventions through comprehensive patient assessment followed by multifaceted therapy including exercise and self-management to improve respiratory health and reduce distress.[11] Currently, comprehensive rehabilitation has achieved more significant and excellent results in diseases such as burns and ischemic strokes,[12,13] while its effectiveness in COPD is still not understood. Moreover, the timing and choice of comprehensive treatment for patients with acute exacerbations is unclear.

Therefore, this study will analyze the impact of comprehensive rehabilitation therapy in the treatment of COPD, aiming to provide reliable theoretical guidance for future clinical treatment.

MATERIALS AND METHODS

Patient Data

A total of 174 patients with acute COPD exacerbation in Affiliated Hospital of Hebei University in China from March 2020 to January 2022 were selected as research subjects. The control group was given conventional treatment; the acute group started comprehensive rehabilitation treatment in the acute phase; in the stable group, comprehensive rehabilitation treatment was initiated in the stable period after the condition was stabilized through conventional treatment. The experiment was approved by the ethics committee (HDFY-LL-2022-009) of our hospital and all patients signed an informed consent form.

Inclusion and Exclusion Criteria

Inclusion criteria. Patients with (1) a confirmed diagnosis of COPD according to COPD diagnostic guidelines[14]; (2) were all in an acute phase with no recent treatment except conventional drug therapy; (3) no drug allergies, complete data and consent to participate in this study.

Exclusion criteria. Patients with (1) severe organ damage, blood disorders, respiratory distress caused by other causes; (2) mental retardation; (3) low compliance.

Treatment Methods

The control group was treated with conventional drugs used to improve COPD symptoms (such as ipratropium bromide, salbutamol, etc.), and treatment strictly followed the Global initiative for Obstructive Lung Disease (GOLD) and UK National Institute for Health and Clinical Excellence (NICE) treatment guidelines for COPD.[15] The risk for exacerbation was ameliorated by the use of long-term medication (bronchodilators) and patients were given basic knowledge of the disease.

The acute and stable groups carried out comprehensive rehabilitation treatment in the acute and stable phases, respectively.[16] On the basis of the control group, respiratory muscle training was implemented to enhance patients' inspiratory muscle resistance. Respiratory function training was required, including lip retraction breathing and abdominal breathing, to induce patients to inhale for as long as possible. Patients were guided to perform appropriate exercise according to their condition. Disease education activities were developed for patients and families to increase their understanding of COPD and the importance of treatment. Patients were instructed to eat properly, thus ensuring a high-protein, low-fat diet; Patients with negative emotions were actively psychologically counseled to eliminate pessimism, reduce negative effects and increase patients' confidence in treatment.

Sample Collection and Testing

Venous blood was drawn from patients before treatment (T0), after treatment for 8 days (T1), treatment for 15 days (T2), treatment for 30 days (T3), treatment for 90 days (T4) and ELISA was performed to detect TNF-α, hs-CRP and IL-6 in serum, and the kits were purchased from TransGen Biotech (Beijing, China). The operation procedure was carried out strictly according to the kit instructions. The forced expiratory volume in 1 second (FEV1), forced expiratory volume in 1 second/forced vital capacity (FEV1/FVC) ratio and peak expiratory flow rate (PEF) were also measured using a spirometer.

Rating Survey

The American Thoracic Society developed the Shortness of Breath Index[17] to assess shortness of breath in patients, which is scored on a 4-point scale (0-4), with higher levels indicating more severe symptom. The Activities of Daily Living (ADL) Scale[18] assesses patients' quality of life (QoL), with lower scores indicating poorer ability. Patients' psychology was assessed with the Self-rating Anxiety Scale (SAS) and Self-rating Depression Scale (SDS),[19] with higher scores indicating more severe negative emotions. Patients' QoL was evaluated with the COPD Quality of Life Questionnaire[20]; scores were evaluated in 4 domains: emotion, fatigue, wheezing and emotional control, with higher scores indicating better QoL.

Outcome Measures

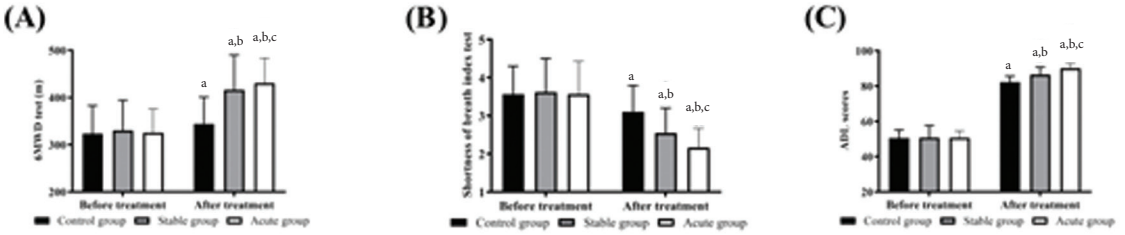
1. **ADL**: Includes results of the 6-min walk test (6MWD) assessment, shortness of breath and ADL scores
2. **Pulmonary function**: Includes FEV1, FEV1/FVC and PE
3. **Inflammation**: Includes TNF-α and hs-CRP, IL-6
4. **Psychology**: Includes SAS and SDS
5. **AEs**: Includes incidence of AEs (such as dizziness and palpitations during treatment)
6. **Quality of life** score results.

Table 1. General Patient Data

	Control group (n=58)	Stable group (n = 58)	Acute group (n = 58)	χ^2/t	*P* value
Age (yrs)	63.57 ± 4.12	64.40 ± 3.85	64.22 ± 4.25	0.665	.516
Gender				1.025	.599
women	21 (36.21)	25 (43.10)	20 (34.48)		
men	37 (63.79)	33 (56.90)	38 (65.52)		
Smoking				2.035	.362
yes	35 (60.34)	40 (68.97)	42 (72.41)		
no	23 (39.66)	18 (31.03)	16 (27.59)		
SBP (mmHg)	125.38 ± 9.34	125.79 ± 9.49	128.72 ± 10.90	1.950	.146
DBP (mmHg)	80.86 ± 6.80	80.91 ± 7.02	81.52 ± 6.90	0.164	.849
BMI (kg/m^2)	25.95 ± 2.27	26.52 ± 2.11	25.53 ± 2.42	2.779	.065

Abbreviations: BMI, body mass index; DBP, diastolic blood pressure; SBP, systolic blood pressure.

Figure 1. Comparison of ADL between the 3 groups before and after treatment. (**1A**) Comparison of results of 6MWD test; (**1B**) comparison of shortness of breath index scores; (**1C**) comparison of ADL scores.



[a]Compared with before treatment ($P < .05$)
[b]Compared with the control group ($P < .05$)
[c]Compared with the stable group ($P < .05$)

Abbreviations: 6MWD, 6-minute walk distance; ADL, Activities of Daily Living.

Statistical Analysis

Statistical analysis was performed using IBM™ SPSS 23.0 software, and the measurement data were expressed as mean ± standard deviation (SD) using ANOVA and LSD intergroup tests and paired *t* test for pre- and post-treatment comparisons. The counting data were represented as a percentage and the chi-square test was applied. Differences were indicated as statistically significant at $P < .05$.

RESULTS

General Data Table

Regarding the general data including age, gender, smoking, systolic blood pressure (SBP) and diastolic blood pressure (DBP) in the 3 groups, no statistically significant differences were seen ($P > .05$; Table 1), indicating comparability between them.

Comparison of ADL in the 3 Groups Before and After Treatment

Before treatment, there was no difference in 6MWD, shortness of breath index or ADL scores between the 3 groups ($P > .05$). After treatment, the 6MWD and ADL scores increased in all 3 groups, with the highest in the acute group and higher in the stable group than in the control group ($P < .05$; Figures 1A and 1B). While the shortness of breath index score was lower in all 3 groups than before treatment, lower in the acute group than in the other 2 groups, and higher in the control group than in the stable group ($P < .05$; Figure 1C).

Comparison of Pulmonary Function in the 3 Groups Before and After Treatment

At T0 and T1, pulmonary function test results were also similar in the 3 groups ($P > .05$). At T2, there was no difference in FEV1 or PEF in the 3 groups ($P > .05$), while FEV1/FVC in the stable and acute groups was higher than in the control group, and was highest in the acute group ($P < .05$). At T3 and T4, the FEV1, FEV1/FVC and PEF in the acute group were the highest of the 3 groups, followed by the stable group ($P < .05$). FEV1/FVC and PEF in the 3 groups began to increase at T2, while FEV1 began to increase at T3 ($P < .05$; Table 2).

Comparison of Inflammatory Factor Levels Before and After Treatment in the 3 Groups

At T0 toT1, the differences in inflammatory factor levels were not statistically significant in any of the 3 groups ($P > .05$). From T2, the level of inflammatory factors in the 3

Table 2. Comparison of Pulmonary Function in the 3 Groups Before and After Treatment

	Time	Control group (n = 58)	Stable group (n = 58)	Acute group (n = 58)	F	*P* value
FEV1 (L)	T0	1.63 ± 0.28	1.60 ± 0.57	1.61 ± 0.28	0.084	.919
	T1	1.63 ± 0.27	1.62 ± 0.28	1.63 ± 0.30	0.024	.976
	T2	1.63 ± 0.30	1.64 ± 0.30	1.67 ± 0.31	0.273	.761
	T3	1.89 ± 0.45[c,d,e]	2.44 ± 0.43[a,c,d,e]	2.59 ± 0.52[a,b,c,d,e]	35.930	<.001
	T4	2.21 ± 0.35[c,d,e,f]	2.62 ± 0.33[a,c,d,e,f]	2.71 ± 0.35[a,b,c,d,e,f]	34.920	<.001
FEV1/FVC (%)	T0	51.77 ± 4.90	50.94 ± 6.65	51.80 ± 4.21	0.482	.618
	T1	52.13 ± 6.08	52.52 ± 6.34	53.21 ± 5.37	0.491	.613
	T2	55.26 ± 6.66[c,d]	59.73 ± 6.95[a,c,d]	63.38 ± 6.32[a,b,c,d]	21.700	<.001
	T3	61.60 ± 6.77[c,d,e]	67.48 ± 6.68[a,c,d,e]	72.10 ± 6.85[a,b,c,e,d]	35.080	<.001
	T4	64.78 ± 4.69[c,d,e,f]	69.43 ± 5.38[a,c,d,e,f]	75.60 ± 5.67[a,b,c,d,e,f]	61.690	<.001
PEF (L/s)	T0	3.32 ± 0.31	3.32 ± 0.40	3.39 ± 0.27	0.864	.423
	T1	3.38 ± 0.50	3.46 ± 0.49	3.40 ± 0.50	0.408	.666
	T2	3.63 ± 0.52[c,d]	3.66 ± 0.46[c,d]	3.70 ± 0.49[c,d]	0.297	.743
	T3	4.33 ± 0.31[c,d,e]	4.82 ± 0.42[a,c,d,e]	5.43 ± 0.43[a,b,c,d,e]	115.500	<.001
	T4	4.91 ± 0.70[c,d,e,f]	5.63 ± 0.56[a,c,d,e,f]	6.32 ± 0.93[a,b,c,d,e,f]	51.840	<.001

[a]Compared with control group ($P < .05$)
[b]Compared with stable group ($P < .05$)
[c]Compared with T0 ($P < .05$)
[d]Compared with T1 ($P < .05$)
[e]Compared with T2 ($P < .05$)
[f]Compared with T3 ($P < .05$)

Abbreviations: FEV1, forced expiratory volume in 1 second; FEV1/FVC forced expiratory volume in 1 second/forced vital capacity; PEF, peak expiratory flow rate; T0, before treatment; T1, after treatment for 8 days; T2, after treatment for 15 days (T2); T3, after treatment for 30 days; T4, after treatment for 90 days.

Table 3. Comparison of Inflammatory Factor Levels Before and After Treatment in the 3 Groups

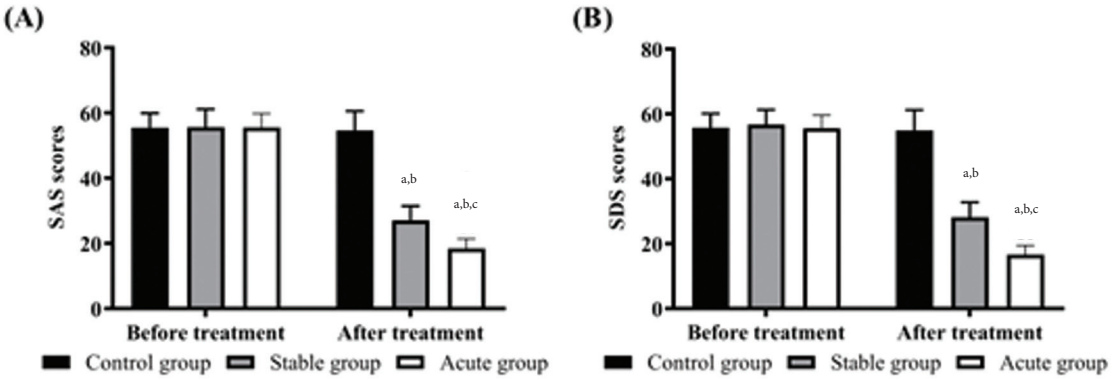
	Time	Control group (n = 58)	Stable group (n = 58)	Acute group (n = 58)	F	*P* value
TNF-α (μg/L)	T0	2.94 ± 0.26	3.02 ± 0.40	2.92 ± 0.30	1.534	.219
	T1	2.84 ± 0.45	2.86 ± 0.40	2.92 ± 0.45	0.534	.587
	T2	2.73 ± 0.51	2.67 ± 0.41[c,d]	2.36 ± 0.41[a,b,c,d]	11.510	<.001
	T3	2.36 ± 0.41[c,d,e]	2.36 ± 0.41[a,c,d,e]	1.99 ± 0.47[a,b,c,d,e]	14.250	<.001
	T4	1.81 ± 0.13[c,d,e,f]	1.50 ± 0.27[a,c,d,e,f]	1.14 ± 0.09[a,b,c,d,e,f]	199.800	<.001
hs-CRP (mg/L)	T0	3.88 ± 0.48	3.89 ± 0.41	3.88 ± 0.48	0.009	.991
	T1	3.81 ± 0.38	3.80 ± 0.41	3.79 ± 0.39	0.037	.963
	T2	3.70 ± 0.39	3.51 ± 0.38[a,c,d]	3.23 ± 0.40[a,b,c,d]	21.310	<.001
	T3	2.63 ± 0.22[c,d,e]	2.44 ± 0.32[a,c,d,e]	2.07 ± 0.26[a,b,c,d,e]	64.610	<.001
	T4	2.04 ± 0.40[c,d,e,f]	1.47 ± 0.33[a,c,d,e,f]	0.82 ± 0.22[a,b,c,d,e,f]	204.300	<.001
IL-6 (pg/mL)	T0	60.30 ± 5.35	60.89 ± 5.67	60.44 ± 5.18	0.189	.828
	T1	59.18 ± 5.21	59.52 ± 7.01	58.71 ± 5.75	0.263	.769
	T2	57.05 ± 5.52	53.46 ± 5.84[a,c,d]	51.68 ± 6.58[a,b,c,d]	12.070	<.001
	T3	53.67 ± 4.07[c,d,e]	50.28 ± 4.24[a,c,d,e]	47.06 ± 3.74[a,b,c,d,e]	39.170	<.001
	T4	44.59 ± 7.17[c,d,e,f]	38.25 ± 6.91[a,c,d,e,f]	34.22 ± 5.43[a,b,c,d,e,f]	36.960	<.001

[a]Compared with control group ($P < .05$)
[b]Compared with stable group ($P < .05$)
[c]Compared with T0 ($P < .05$),
[d]Compared with T1 ($P < .05$)
[e]Compared with T2 ($P < .05$)
[f]Compared with T3 ($P < .05$)

Abbreviations: hs-CRP, high-sensitivity C-reactive protein; IL-6, interleukin-6; T0, before treatment; T1, after treatment for 8 days; T2, after treatment for 15 days (T2); T3, after treatment for 30 days; T4, after treatment for 90 days; TNF-α tumor necrosis factor-α.

groups began to show significant differences; the acute group had the lowest and the stable group was lower than the control group ($P < .05$). In addition, the level of inflammatory factors in the stable and acute groups began to decrease gradually from T2, while the control group began to decrease from T3 ($P < .05$; Table 3).

Figure 2. Comparison of psychology scores in both groups before and after treatment. (**2A**) Comparison of SAS scores; (**2B**) comparison of SDS scores.



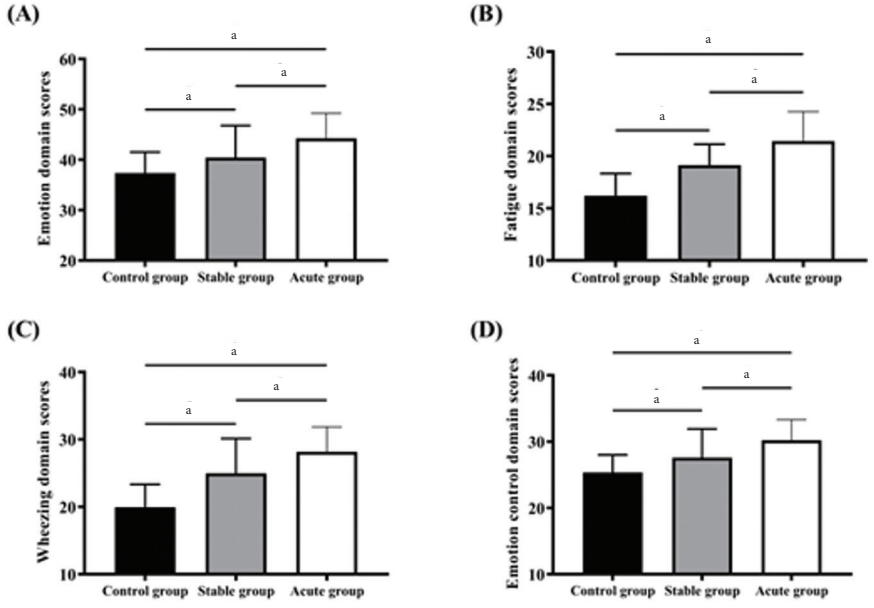
[a]Compared with before treatment ($P < .05$)
[b]Compared with the control group ($P < .05$)
[c]Compared with the stable group ($P < .05$)

Abbreviations: SAS, Self-rating Anxiety Scale; SDS, Self-rating Depression Scale.

Table 4. Incidence of Adverse Reactions in the 3 Groups

Groups	n	Dizziness	Palpitations	Thirst	Coughing	Overall incidence (%)
Control	58	2 (3.45%)	2 (3.45%)	1 (1.72%)	2 (3.45%)	12.07
Stable	58	0 (0.00%)	1 (1.72%)	0 (0.00%)	1 (1.72%)	3.45
Acute	58	1 (1.72%)	0 (0.00%)	1 (1.72%)	0 (0.00%)	3.45
χ^2						4.852
P value						.088

Figure 3. Comparison of QoL scores in the 3 groups. (**3A**) Comparison of emotion domain scores; (**3B**) comparison of fatigue domain scores; (**3C**) comparison of wheezing domain scores. (**3D**) Comparison of emotional control domain scores.



[a]$P < .05$

Abbreviations: QoL, quality of life.

Comparison of Psychology in the 3 Groups Before and After Treatment

Before treatment, there was no difference in the SAS and SDS scores between the acute and stable groups ($P > .05$), and no marked change was seen in the control group after treatment ($P > .05$). Both the acute and stable groups had lower scores than before treatment, and the scores were lower than in the control group. The SAS and SDS scores were even lower in the acute group than in the stable group after treatment ($P < .05$; Figures 2A, 2B).

Incidence of Adverse Events in the 3 Groups

Only 1 patient in the acute group developed cough and 1 patient developed dizziness. The incidence of AEs was 3.45% in the acute group, 3.45% in the stable group and 12.07% in the control group. When comparing the 3 groups, the difference in the incidence of AEsa was not statistically remarkable ($P > .05$; Table 4).

Comparison of Quality of Life in the 3 Groups

The QoL scores in the 3 groups in the domains of emotion, fatigue, wheezing and emotional control in the acute group were (44.26 ± 4.96), (21.45 ± 2.82), (28.19 ± 3.68) and (30.22 ± 3.08), respectively, and did not differ compared with the stable group ($P > .05$). Compared with the control group, the acute and stable groups had higher scores in all domains ($P < .05$; Figures 3A-3D).

DISCUSSION

COPD is a progressive disease characterized by airflow limitation; the incidence of COPD has been increasing in recent years and has the potential to develop in any age group.[21] In-depth understanding of the clinical application value of comprehensive rehabilitation therapy for COPD is of great importance to improve the efficacy of COPD treatment and guarantee the prognosis of patients in the future.

In our study, patients in the acute and stable groups had a more obvious improvement in exercise capacity, ADL and lung function than the control group, while the shortness of breath index and inflammatory factor levels were dramatically lower. This suggests that our comprehensive rehabilitation therapy has an excellent effect in the treatment of COPD. This is consistent with the results of previous studies,[22,23] which may corroborate our results. Research has shown that the treatment of COPD is a long and complex process in which patients need to be given effective therapeutic support to stop the re-progression of COPD and to improve their QoL, even when their pathological manifestations are stabilizing.[24]

Currently, COPD treatment is still dominated by drugs, ignoring non-pharmacological physical therapy and psychotherapy.[25] In comprehensive rehabilitation in COPD, we believe that the reasons for the more optimal recovery of patients may be:

1. **Addressing the Psychological Concerns of Patients with COPD**. It is well known that COPD has a long disease course and patients generally have more serious psychological disorders due to the burden of prolonged disease and treatment.[26] Therefore, strengthening psychological interventions and targeted psychological counseling for patients can improve their psychologically negative emotions and enhance their confidence in overcoming the disease. This is confirmed by the fact that SAS and SDS scores were lower in the acute and stable groups than in the control group.
2. **Changes in Lifestyle Habits**. In our study, patients were asked to quit smoking, as well as adopt lifestyle changes such as cold prevention—which reduces acute exacerbations of COPD due to infection and bodily damage due to acute onset of the disease[27]—and vaccinations. Quitting smoking can reduce the clinical symptoms of COPD, stop its progression, and slow the rate of lung function decline.
3. **Respiratory Training Interventions**. Patients with COPD experience a progressive decline in lung function, and respiratory functions have to be taken over by secondary respiratory and intercostal muscles due to damage to the primary respiratory muscles, resulting in thoracic breathing.[28] In comprehensive rehabilitation treatment, the combination of lip constriction breathing and abdominal breathing is used to improve respiratory function, increase tidal volume, reduce respiratory rate and ventilation per minute and relieve patients' dyspnea symptoms. In addition, the "isobaric point" of the airway is pushed to the distal end of the airway during expiration, which prevents trapping and narrowing of the small airway during expiration and facilitates the elimination of alveolar air.[29] We hypothesize that this is the main reason for the difference in lung function between the groups.
4. **Aerobic endurance exercise intervention**. Aerobic endurance exercise enhances the function of some organs (e.g. heart function, lung function, muscle function, etc.). Oxygen consumption is reduced and skeletal muscle is strengthened, so that recovery of patients' function is more ideal.
5. **Nutritional support**. Patients with COPD have elevated energy expenditure due to increased airway resistance.[30] At the same time, due to long-term hypercapnia, hypoxia, long-term use of antibiotics and gastrointestinal (GI) bruising, the normal flora of the GI tract is dysregulated, resulting in absorption and digestive dysfunction and widespread malnutrition.[31] With enhanced nutritional support, it is possible to provide an excellent and stable environment for patient recovery from the perspective of GI function and nutrient metabolism, thus accelerating the prompt recovery of pulmonary function.
6. **Home oxygen therapy** can improve the ventilatory dysfunction and hypoxemia of patients with COPD patients for a long and stable period, thus more effectively promoting the patient recovery and improving their QoL. The results of the QoL scores in the acute and stable groups were also consistent with our expectations,

which revealed that these led to a much improved QoL. In addition, in the comparison of the acute and stable groups, we found that improvement of the observed indexes such as ADLs, pulmonary function and inflammatory factors were more remarkable in the acute group than in the stable group, which also suggests that we can achieve better and more significant results by carrying out comprehensive rehabilitation treatment in the acute phase of COPD.

It is well known that COPD leads to chronic hypoxia in the lungs due to respiratory dysfunction, which causes structural changes and inadequate lung function.[32] On the other hand, movement of the human body requires respiratory movements.[33] Thus, the longer the duration of COPD, the more severe the decline in patient organismal function is likely to be. Not only is there a loss of respiratory function and normal functioning of respiratory muscles, but there may also be a series of immunometabolic disorders, endocrine abnormalities, etc. present.[34] Comprehensive rehabilitation therapy carried out in the acute phase of COPD can effectively avoid COPD damage to patients' function and provide a more reliable basis for their recovery. Of course, there are no studies to testify to this view due to the paucity of relevant literature. In response to this situation, we will conduct more detailed experiments in the future to confirm our analysis.

Study Limitattions

Nevertheless, our study has many limitations. For example, COPD is a chronic disease of extremely long duration, which requires longer follow-up and evaluation of the study patients, and increasing the number of cases will increase credibility. Furthermore, since there are no uniform clinical guidelines for comprehensive rehabilitation treatment in COPD, there may be room for optimization and improvement in the specific implementation of this method.

We need to conduct a more in-depth and comprehensive study on the application of comprehensive rehabilitation therapy in COPD.

CONCLUSION

Comprehensive rehabilitation therapy can improve the exercise capacity and lung function of patients with COPD, reduce inflammation and improve patients' negative psychology. It has a high safety profile, which can be used in future COPD treatment to provide a more reliable patient prognosis. Meanwhile, comprehensive rehabilitation treatment in the acute phase of COPD can achieve better results, which may be related to the functional impairment of the body due to the long-term effects of COPD.

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CONFLICTS OF INTEREST

The authors have no conflicts of interest to declare.

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