

Original Article

Short-term multidimensional effects of continuous positive airway pressure in patients with obstructive sleep apnea-hypopnea syndrome: a single-center retrospective study

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Abstract: Objective: To evaluate the short-term effects of continuous positive airway pressure (CPAP) combined with conventional lifestyle intervention on sleep parameters, inflammatory status, hemorheology, vascular endothelial function, upper airway dimensions, and quality of life in patients with obstructive sleep apnea-hypopnea syndrome (OSAHS). Methods: Medical records of 89 patients with obstructive sleep apnea-hypopnea syndrome (OSAHS) treated between July 2023 and September 2025 were retrospectively reviewed. According to management methods, patients were divided into a control group (n=47; receiving lifestyle intervention alone) or a study group (n=42; receiving CPAP plus lifestyle intervention). After 12-week intervention, overall clinical response rates, apnea-hypopnea index (AHI), lowest oxygen saturation (LSaO₂), Pittsburgh Sleep Quality Index (PSQI), upper airway cross-sectional areas, hemorheological parameters, inflammatory cytokines, endothelial function markers, and World Health Organization Quality of Life-BREF questionnaire (WHOQOL-BREF) scores were compared between groups. Results: The overall response rate was higher in the study group than in the control group (92.86% vs. 42.55%, $P < 0.001$). After treatment, both groups showed improvements in AHI, LSaO₂, PSQI, hemorheological parameters, inflammatory cytokines, endothelial function, and quality-of-life scores, with generally greater improvements in the study group (all $P < 0.05$). Upper airway cross-sectional areas increased in both groups, but between-group differences were not significant. Conclusions: CPAP combined with lifestyle intervention was associated with greater short-term improvements in sleep-related parameters, inflammation, hemorheology, endothelial function, and quality of life in patients with OSAHS. Larger prospective multicenter studies are needed to confirm these findings.

Keywords: Obstructive sleep apnea-hypopnea syndrome, continuous positive airway pressure, inflammatory cytokines, vascular endothelial function, sleep quality

Introduction

Obstructive sleep apnea-hypopnea syndrome (OSAHS) is a common chronic sleep-related breathing disorder, which is more prevalent in middle-aged, elderly, and overweight populations. Its main pathological feature is the repeated collapse of upper airway soft tissues during sleep, leading to intermittent airway narrowing or obstruction, which further causes recurrent apnea, hypopnea, and nocturnal hypoxemia [1]. Repeated hypoxia-reoxygen-

ation cycles may disrupt normal physiological homeostasis, induce systemic low-grade chronic inflammation, and promote the abnormal release of pro-inflammatory cytokines, such as interleukins and tumor necrosis factor [2]. Meanwhile, intermittent hypoxia can also impair vascular endothelial structure and function, resulting in an imbalance in vasoactive factor secretion, coagulation abnormalities, and hemorheological changes. These alterations may increase the risk of cardiovascular and cerebrovascular complications, including

hypertension, coronary heart disease, and atherosclerosis, and may adversely affect patients' daily activities and long-term quality of life [3, 4]. Therefore, reducing nocturnal hypoxic stimulation, inhibiting inflammatory responses, and improving vascular endothelial function are important goals in the comprehensive treatment of OSAHS.

Continuous positive airway pressure (CPAP) is an important conservative treatment for moderate to severe OSAHS and has been increasingly applied in clinical practice [5]. CPAP delivers continuous positive airflow through a ventilator via a nasal mask or oronasal mask to the upper airway. By providing physical support, it maintains the patency of collapse-prone regions, such as the pharyngeal cavity, soft palate, and tongue base, thereby reducing apnea and hypopnea events, improving nocturnal oxygen saturation, and stabilizing sleep architecture and autonomic nervous function [6, 7]. Compared with basic lifestyle management, such as dietary control, exercise intervention, and positional therapy, CPAP can reduce the persistent systemic stimulation caused by intermittent hypoxia at the pathological level. Therefore, it may help reduce inflammatory activation and improve vascular endothelial injury [8]. Although the beneficial effects of CPAP on sleep-related parameters and inflammatory status in OSAHS have been reported previously, most existing studies have focused on single categories of outcomes or highly selected study populations. Real-world evidence simultaneously evaluating sleep parameters, hemorheology, inflammatory cytokines, vascular endothelial function, upper airway anatomical changes, and health-related quality of life remains relatively limited. Therefore, the present single-center retrospective study analyzed data from 89 patients with OSAHS to provide a short-term multidimensional assessment of CPAP combined with conventional lifestyle intervention in routine clinical practice.

Materials and methods

General information

This was a single-center retrospective study. The clinical data of 89 patients with obstructive sleep apnea-hypopnea syndrome (OSAHS) who were treated in our hospital from July 2023

to September 2025 were retrospectively analyzed. All data were obtained from the hospital electronic medical record system and follow-up records. According to different treatment regimens, the 89 patients with OSAHS were divided into two groups. The control group included 47 patients who received conventional treatment, namely lifestyle intervention alone, while the study group included 42 patients who received continuous positive airway pressure (CPAP) therapy in addition to conventional treatment.

Case selection criteria

Inclusion criteria: The inclusion criteria were as follows: (1) Patients who met the diagnostic criteria for OSAHS and were newly diagnosed [9]; (2) Patients aged 18-65 years; (3) Patients with complete clinical data, treatment records, and follow-up records; (4) Patients without contraindications to CPAP therapy.

Exclusion criteria: The exclusion criteria were as follows: (1) Patients complicated with central sleep apnea syndrome; (2) Patients complicated with severe pulmonary diseases such as severe pulmonary bullae, pneumothorax, or mediastinal emphysema; (3) Pregnant or lactating women; (4) Patients with severe organ dysfunction, malignant tumors, or coagulation disorders; (5) Patients with severe mental disorders or cognitive impairment; (6) Patients with acute or chronic infection, trauma, or surgery within the past month.

Patients with incomplete baseline data, incomplete treatment records, missing 12-week follow-up data, or discontinuation of treatment during the study period were excluded from the final analysis. No imputation was performed for missing data. Only patients who completed the full 12-week treatment and follow-up were included in the final cohort.

Ethics approval and consent to participate: This retrospective single-center study was approved by the Clinical Research Ethics Committee of the First Hospital of Hebei Medical University (Approval No. [2025] Research Ethics Review No. 218). The study was conducted in accordance with the principles of the Declaration of Helsinki. Because the study involved only the retrospective analysis of anonymized clinical data and posed no addi-

tional risk to patients, the requirement for written informed consent was waived by the ethics committee.

Methods

Control group: Patients in the control group received standardized conventional lifestyle intervention. The main measures included strict control of total caloric intake, a low-fat and low-sugar diet, at least 150 minutes of moderate-intensity aerobic exercise per week, body weight control, smoking cessation and alcohol restriction, avoidance of sleeping in the supine position, preference for lateral sleeping position, establishment of regular sleep and rest habits, and avoidance of staying up late or excessive fatigue. During the intervention period, outpatient follow-up was conducted once every 4 weeks.

Study group: Patients in the study group received CPAP therapy in addition to conventional lifestyle intervention. An automatic continuous positive airway pressure ventilator was used. Before treatment, overnight polysomnography-based pressure titration was performed. Individualized therapeutic pressure was set according to apnea events, blood oxygen fluctuation, and patient tolerance, with a pressure range of 6-18 cmH₂O. Patients were instructed to wear an appropriate nasal mask, connect the tubing properly, and receive nocturnal ventilation therapy for at least 5 hours per night. During treatment, medical staff conducted follow-up every 2 weeks to manage adverse reactions such as mask leakage, nasal dryness, pharyngeal discomfort, and pressure intolerance, and to dynamically adjust ventilator parameters to ensure treatment compliance. Both groups were treated for 12 weeks.

Observation indicators

Clinical efficacy: Clinical efficacy was evaluated according to the Guidelines for the Diagnosis and Surgical Treatment of Obstructive Sleep Apnea-Hypopnea Syndrome [10]. A markedly effective response was defined as a post-treatment AHI < 5 events/h, lowest oxygen saturation (LSaO₂) ≥ 90%, and complete resolution of daytime sleepiness, nocturnal snoring, and choking episodes. An effective response was defined as a ≥ 50% reduction in AHI and a

≥ 10% increase in LSaO₂ from baseline, with AHI remaining ≥ 5 events/h, accompanied by significant relief of daytime sleepiness and nocturnal symptoms. Ineffective was defined as failure to meet the criteria for markedly effective or effective. The total effective rate was calculated as markedly effective plus effective.

Sleep quality: Before treatment and after 12 weeks of treatment, the AHI and LSaO₂ were monitored and recorded using a Mikie® PSM-E polysomnography monitor (Zhejiang Medical Device Registration Certificate No. 202020-71350). Subjective sleep quality was assessed using the Pittsburgh Sleep Quality Index (PSQI). The total PSQI score ranges from 0 to 21, with higher scores indicating poorer sleep quality.

Upper airway cross-sectional area: Before treatment and after 12 weeks of treatment, upper airway CT scanning was performed. The cross-sectional areas of four upper airway planes, including the nasopharynx, retropalatal region, retroglossal region, and retroepiglottic region, were measured during quiet breathing to evaluate improvement in upper airway narrowing.

Hemorheology: Before treatment and after 12 weeks of treatment, 5 mL of fasting venous blood was collected from the elbow vein. Whole blood viscosity at high, medium, and low shear rates, as well as plasma viscosity, were measured using an SA-9800 automatic hemorheology analyzer (Beijing Succeeder Technology Inc., Beijing Medical Device Registration No. 20192220682). All procedures were performed strictly according to the manufacturer's instructions.

Inflammatory cytokines: Before treatment and after 12 weeks of treatment, 5 mL of fasting venous blood was collected, and serum was separated by centrifugation at 3,000 r/min for 10 min. Serum levels of interleukin-6 (IL-6), interleukin-1β (IL-1β), and tumor necrosis factor-α (TNF-α) were detected using enzyme-linked immunosorbent assay (ELISA). All ELISA kits were purchased from Wuhan Saio Biotechnology Co., Ltd.

Vascular endothelial function: Before treatment and after 12 weeks of treatment, serum

Table 1. Comparison of baseline data between the two groups

Group	Gender (Male/Female)	Age (Years)	Body Mass Index (kg/m ²)	Severity (Mild/ Moderate/ Severe)	Disease Duration (Years)	Comorbidities (Hypertension/ Hyperlipidemia/Diabetes)
Control group (n=47)	27/20	56.60±7.52	26.94±3.17	5/27/15	3.92±1.15	20/12/15
Study group (n=42)	22/20	57.04±8.18	26.77±3.65	5/25/12	4.13±1.24	17/13/12
$\chi^2/t/Z$	0.230	0.264	0.235	0.356	0.829	0.337
<i>P</i>	0.632	0.792	0.815	0.722	0.410	0.845

levels of endothelin-1 (ET-1), nitric oxide (NO), and von Willebrand factor (vWF) were measured using ELISA. All kits were purchased from Wuhan Saio Biotechnology Co., Ltd.

Quality of life: Before treatment and after 12 weeks of treatment, quality of life was assessed using the World Health Organization Quality of Life Brief Scale (WHOQOL-BREF). The scale includes four domains: physical health, psychological health, social relationships, and environment. Each domain has a full score of 100 points, with higher scores indicating better quality of life.

Statistical analysis

Statistical analyses were performed using IBM SPSS Statistics version 25.0. Measurement data were expressed as mean $\pm$ standard deviation. Independent-samples *t* tests were used for comparisons between groups, and paired *t* tests were used for comparisons before and after treatment within the same group. Count data were expressed as numbers and percentages [*n* (%)], and comparisons between groups were performed using the chi-square test or Fisher's exact test. A value of *P* < 0.05 was considered statistically significant.

Results

Baseline characteristics

A total of 89 patients met the inclusion and exclusion criteria and were included in the final analysis. All included patients completed the full 12-week treatment and follow-up, and no additional cases were excluded because of missing follow-up data after enrollment. There were no statistically significant differences between the two groups in baseline characteristics, including sex distribution, age, BMI, and disease severity (*P* > 0.05), indicating comparability between the groups (Table 1).

Clinical efficacy

The total effective rate in the study group was significantly higher than that in the control group (92.86% vs. 42.55%, *P* < 0.001; Figure 1).

Sleep quality

Before treatment, there were no statistically significant differences in AHI, LSAO₂, and PSQI scores between the two groups (*P* > 0.05). After treatment, AHI and PSQI scores decreased significantly in both groups, with greater reductions observed in the study group. In contrast, LSAO₂ increased significantly in both groups, and the increase was more pronounced in the study group (*P* < 0.05). See Figure 2.

Changes in the cross-sectional areas of different upper airway planes

Before treatment, there were no significant differences in the cross-sectional areas of different upper airway planes between the two groups (*P* > 0.05). After treatment, the cross-sectional areas of all measured upper airway planes were significantly increased compared with baseline in both groups (*P* < 0.05). However, comparisons between the two groups at the same time point showed no significant differences (*P* > 0.05; Table 2).

Hemorheology

Before treatment, the levels of whole blood viscosity and plasma viscosity were comparable between the two groups, with no statistically significant differences (*P* > 0.05). After treatment, all hemorheological indicators decreased significantly in both groups, and the reductions were more pronounced in the study group (*P* < 0.05). See Table 3.

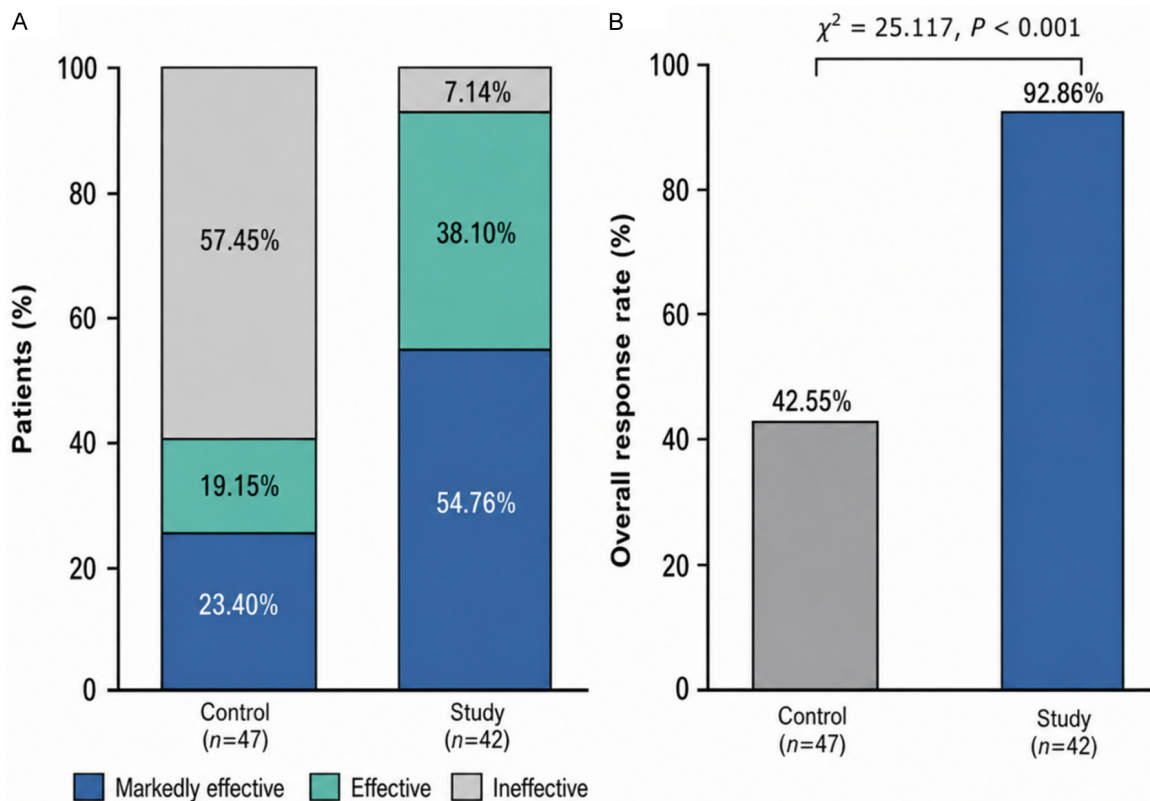


Figure 1. Comparison of clinical efficacy and overall response rate between the two groups. A. Distribution of markedly effective, effective, and ineffective responses. B. Comparison of overall response rate between the two groups.

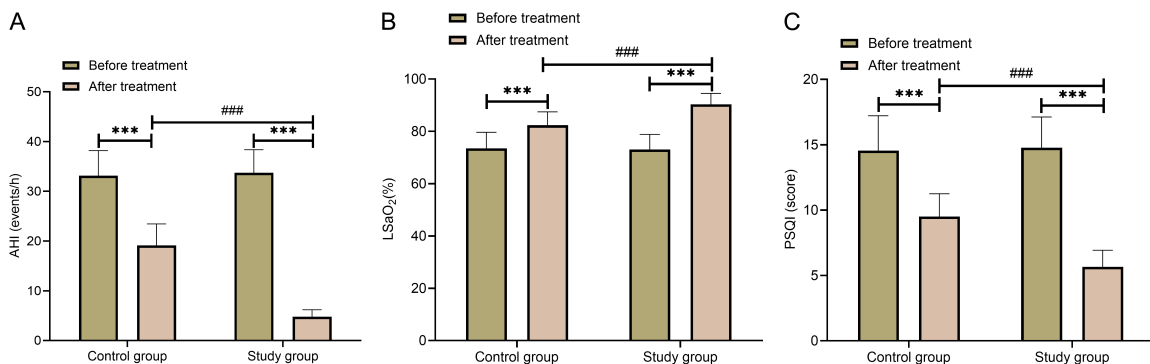


Figure 2. Effects of treatment on sleep-related parameters in both groups. A. Apnea-Hypopnea Index (AHI, events/h). B. Lowest oxygen saturation (LSaO₂, %). C. Pittsburgh Sleep Quality Index (PSQI, score). Data are presented as mean $\pm$ SD. *** $P < 0.001$ vs. before treatment within the same group; ### $P < 0.001$ between groups.

Inflammatory cytokines

Before treatment, there were no statistically significant differences in serum IL-6, IL-1 β , and TNF- α levels between the two groups ($P > 0.05$). After treatment, the levels of all inflammatory cytokines decreased significantly in both groups, and the reductions were more

pronounced in the study group ($P < 0.05$). See **Figure 3**.

Vascular endothelial function

Before treatment, there were no significant differences in ET-1, NO, and vWF levels between the two groups ($P > 0.05$). After treatment, ET-1

Table 2. Comparison of cross-sectional areas at different upper airway levels between the two groups ($\bar{x} \pm s$, cm²)

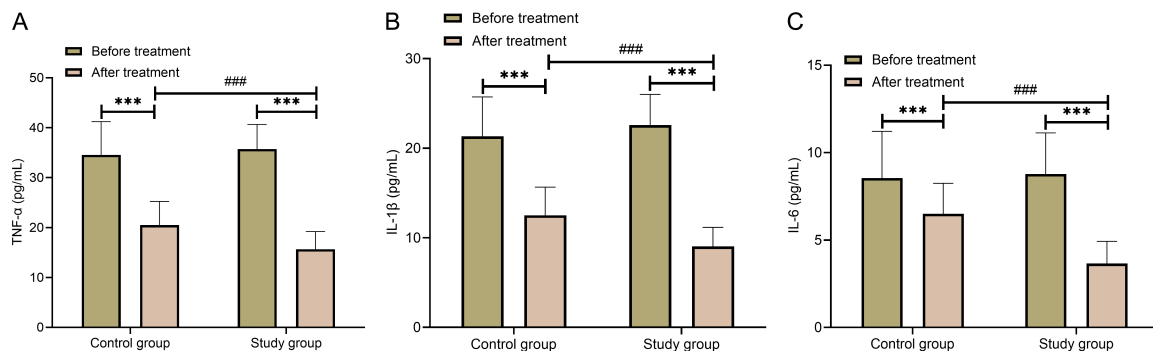
	Nasopharynx		Retropalatal region		Retroglossal region		Retroepiglottic region	
	Before treatment	After treatment	Before treatment	After treatment	Before treatment	After treatment	Before treatment	After treatment
Control group (n=47)	0.82±0.10	1.13±0.15*	0.77±0.12	1.06±0.15*	0.70±0.12	1.12±0.15*	0.74±0.15	1.17±0.14*
Study group (n=42)	0.80±0.12	1.16±0.13*	0.76±0.15	1.09±0.17*	0.72±0.14	1.15±0.17*	0.73±0.13	1.20±0.18*
t	0.857	1.003	0.349	0.885	0.726	0.885	0.334	0.883
P	0.394	0.319	0.728	0.379	0.470	0.379	0.739	0.380

Note: Compared with before treatment in the same group, * $P < 0.05$.

Table 3. Comparison of hemorheological indicators between the two groups ($\bar{x} \pm s$, mPa.s)

Group	Whole blood high-shear viscosity		Whole blood medium-shear viscosity		Whole blood low-shear viscosity		Plasma viscosity	
	Before treatment	After treatment	Before treatment	After treatment	Before treatment	After treatment	Before treatment	After treatment
Control group (n=47)	5.22±0.70	4.73±0.55*	5.84±0.82	4.92±0.65*	8.96±1.12	7.25±0.86*	1.94±0.32	1.56±0.25*
Study group (n=42)	5.11±0.65	4.06±0.53*	5.70±0.74	4.30±0.51*	8.85±1.05	6.12±0.75*	1.96±0.35	1.29±0.27*
t	0.765	5.836	0.842	4.964	0.476	6.570	0.282	4.898
P	0.446	< 0.001	0.402	< 0.001	0.635	< 0.001	0.779	< 0.001

Note: Compared with the baseline level before treatment in this group, * $P < 0.05$.

**Figure 3.** Effects of treatment on serum inflammatory cytokine levels in both groups. A. Tumor necrosis factor-alpha (TNF- α , pg/mL). B. Interleukin-1 beta (IL-1 β , pg/mL). C. Interleukin-6 (IL-6, pg/mL). Data are presented as mean $\pm$ SD. *** $P < 0.001$ vs. before treatment within the same group; ### $P < 0.001$ between groups.

and vWF levels decreased significantly in both groups, with greater reductions observed in the study group. In contrast, NO levels increased significantly in both groups, and the increase was more pronounced in the study group ($P < 0.05$). See **Table 4**.

Quality of life

Before treatment, there were no statistically significant differences between the two groups in the WHOQOL-BREF scores for the physical, psychological, social relationship, and environmental domains ($P > 0.05$). After treatment,

the scores of all quality-of-life domains increased significantly in both groups, and the increases were more pronounced in the study group ($P < 0.05$). See **Table 5**.

Discussion

In recent years, influenced by obesity, unhealthy lifestyle habits, and other factors, the prevalence of obstructive sleep apnea-hypopnea syndrome (OSAHS) has continued to increase in China, and the affected population has gradually become younger [11]. Currently, lifestyle intervention remains a fundamental

Table 4. Comparison of vascular endothelial function indicators between the two groups ($\bar{x} \pm s$)

Group	ET-1 (pg/mL)		NO (μ mol/L)		vWF (%)	
	Before treatment	After treatment	Before treatment	After treatment	Before treatment	After treatment
Control group (n=47)	79.12 $\pm$ 10.37	64.20 $\pm$ 9.12*	34.12 $\pm$ 6.40	44.10 $\pm$ 4.15*	186.20 $\pm$ 36.67	156.50 $\pm$ 24.88*
Study group (n=42)	81.23 $\pm$ 12.53	44.52 $\pm$ 8.20*	32.87 $\pm$ 5.77	57.30 $\pm$ 5.89*	183.47 $\pm$ 41.90	125.32 $\pm$ 23.10*
t	0.869	10.655	0.963	12.322	0.328	6.104
P	0.387	< 0.001	0.338	< 0.001	0.744	< 0.001

Note: Compared with before treatment in the same group, *P < 0.05.

Table 5. Comparison of quality of life scores between the two groups ($\bar{x} \pm s$, Score)

	Physical health		Psychological health		Social relationships		Environment	
	Before treatment	After treatment	Before treatment	After treatment	Before treatment	After treatment	Before treatment	After treatment
Control group (n=47)	52.12 $\pm$ 6.10	67.13 $\pm$ 5.33*	54.66 $\pm$ 6.24	69.42 $\pm$ 5.40*	53.45 $\pm$ 6.32	70.12 $\pm$ 5.66*	53.77 $\pm$ 6.42	68.75 $\pm$ 5.25*
Study group (n=42)	51.30 $\pm$ 5.55	71.20 $\pm$ 6.55*	53.70 $\pm$ 5.77	72.65 $\pm$ 6.44*	53.10 $\pm$ 5.70	74.07 $\pm$ 6.20*	52.87 $\pm$ 5.55	72.44 $\pm$ 6.80*
t	0.661	3.229	0.751	2.573	0.273	3.142	0.703	2.882
P	0.511	0.002	0.455	0.012	0.785	0.002	0.484	0.005

Note: Compared with before treatment in the same group, *P < 0.05.

treatment strategy for OSAHS. This approach is simple to implement and it is inexpensive, additionally it can reduce the degree of airway obstruction to some extent. However, lifestyle intervention alone usually takes effect slowly and may only provide good disease control in patients with mild OSAHS. It cannot correct the pathological state of upper airway soft tissue relaxation and collapse during sleep from an anatomical perspective, nor can it completely halt the inflammatory cascade and progressive endothelial injury induced by chronic intermittent hypoxia [12, 13]. Moreover, its effects on symptom improvement and long-term prognosis in patients with moderate to severe OSAHS are limited. Therefore, more effective treatment strategies are needed. The present study does not aim to re-establish CPAP as an effective treatment for OSAHS, as this has been supported by previous clinical trials and meta-analyses. Instead, our study provides additional real-world evidence by simultaneously assessing multiple clinically relevant dimensions, including sleep quality, hemorheology, inflammatory cytokines, vascular endothelial function, upper airway anatomical changes, and quality of life after a 12-week treatment period.

Continuous positive airway pressure (CPAP) is a first-line conservative treatment for moderate to severe OSAHS and is currently recom-

mended by clinical guidelines [14, 15]. In this study, CPAP combined with conventional lifestyle intervention was applied to patients with OSAHS. The results showed that the total clinical effective rate in the study group was significantly higher than that in the control group. After treatment, the improvements in sleep quality indicators, including AHI, LSAO₂, and PSQI score, were also significantly greater in the study group than in the control group. These findings suggest that CPAP can significantly improve overall clinical efficacy and enhance sleep quality from both subjective and objective perspectives.

The possible reason is that patients with OSAHS often have multilevel anatomical narrowing of the upper airway, including the nasopharynx, retropalatal, retroglossal, and retroepiglottic regions. Relaxation and collapse of upper airway soft tissues during sleep are the fundamental causes of apnea, hypopnea, and intermittent hypoxia. CPAP provides continuous positive airway pressure, which physically supports and opens the collapsed upper airway lumen, expands the cross-sectional area of different anatomical planes, relieves nocturnal airway obstruction, reduces AHI, increases nocturnal LSAO₂, decreases microarousals and sleep fragmentation, and helps restore a normal sleep cycle [16, 17]. In this study, the cross-sectional areas of different upper airway

planes increased after treatment in both groups, but there were no significant differences between the two groups. This suggests that CPAP may help maintain airway patency and reduce recurrent airway collapse caused by changes in body position during sleep, thereby improving nocturnal sleep status and supporting the overall therapeutic effect [18].

Long-term nocturnal intermittent hypoxia in patients with OSAHS can increase erythrocyte aggregation and reduce erythrocyte deformability, leading to abnormal increases in whole blood viscosity at high, medium, and low shear rates, as well as plasma viscosity. As a result, the blood may exhibit a hyperviscous, hypercoagulable, and hyperaggregable state, which slows microcirculatory perfusion and further aggravates tissue ischemia and hypoxia. These changes interact with vascular endothelial injury and the progression of atherosclerosis. In this study, whole blood viscosity at high, medium, and low shear rates and plasma viscosity after treatment were significantly better in the study group than in the control group, indicating that CPAP can improve hemorheological status in patients with OSAHS. This may be because CPAP effectively corrects nocturnal intermittent hypoxia, reduces hypoxia-induced excessive erythrocyte aggregation and membrane dysfunction, decreases plasma macromolecular protein concentration, improves erythrocyte deformability, and thereby reduces whole blood and plasma viscosity. This improvement in systemic microcirculatory perfusion may be one of the important mechanisms by which CPAP reduces the risk of cardiovascular and cerebrovascular events in these patients.

Repeated hypoxia-reoxygenation cycles in patients with OSAHS can activate systemic inflammatory pathways, induce excessive activation of monocytes and macrophages, and promote the release of pro-inflammatory cytokines such as IL-6, IL-1 β , and TNF- α into the circulation, forming a persistent low-grade inflammatory microenvironment and further aggravating tissue injury [19, 20]. The present study showed that the levels of inflammatory cytokines after treatment were significantly lower in the study group than in the control group. This suggests that CPAP can downregulate inflammatory pathway activity by blocking hypoxic stress, inhibit the synthesis and re-

lease of pro-inflammatory cytokines, and prevent the progression of systemic chronic inflammatory responses.

The vascular endothelium serves as a critical barrier in maintaining vascular homeostasis. Intermittent hypoxia can directly cause oxidative stress injury in endothelial cells, resulting in decreased secretion of the vasodilator nitric oxide (NO), increased levels of the vasoconstrictor endothelin-1 (ET-1), and elevated von Willebrand factor (vWF), a marker of endothelial injury. These changes lead to an imbalance in vascular relaxation and contraction and impairment of endothelial barrier function [21]. In this study, vascular endothelial function indicators in the study group were significantly better than those in the control group after treatment ($P < 0.05$), indicating that CPAP can significantly reduce vascular endothelial injury in patients with OSAHS. The possible mechanism is that CPAP corrects hypoxemia, reduces oxidative stress injury in endothelial cells, regulates the balance of vasoactive factor secretion, and promotes the repair of damaged vascular endothelial function. This may help reduce the risk of vascular diseases such as atherosclerosis.

In terms of quality of life, the WHOQOL-BREF scores in all domains after treatment were significantly higher in the study group than in the control group ($P < 0.05$), suggesting that CPAP can effectively improve quality of life in patients with OSAHS. This may be related to the comprehensive improvement in sleep quality, inflammatory status, vascular endothelial function, and hemorheological parameters. As these physiological abnormalities improve, patients' clinical symptoms are relieved, and their physical and psychological status also improves, leading to an overall enhancement in quality of life.

In this single-center retrospective cohort, CPAP combined with conventional lifestyle intervention demonstrated superior short-term improvements in sleep parameters, inflammatory cytokines, hemorheological indicators, vascular endothelial function, and quality of life in patients with OSAHS. These findings provide real-world supportive evidence for the multidimensional clinical benefits of CPAP, but should be further validated in prospective multicenter studies with larger sample sizes. However, this

study was a single-center retrospective analysis with a limited sample size, and only patients treated in our hospital were included, which may have introduced selection bias. In addition, the follow-up period was only 12 weeks, and the long-term effects of CPAP on inflammatory cytokines, vascular endothelial function, and cardiovascular and cerebrovascular outcomes were not observed. Future multi-center, large-sample, prospective controlled studies with extended follow-up are needed to further explore the long-term effects and mechanisms of CPAP on inflammation and vascular endothelial outcomes in patients with OSAHS.

In addition, this study mainly relied on conventional clinical indicators, including sleep parameters, inflammatory cytokines, hemorheological parameters, endothelial function markers, and quality-of-life scores, and did not include novel biomarkers or advanced detection techniques. Propensity score matching was not performed due to the small sample size, which may have further reduced statistical power. Therefore, residual confounding and selection bias cannot be completely excluded, and the findings should be interpreted cautiously and further validated in larger prospective multicenter studies incorporating novel indicators and appropriate confounder-adjustment methods. Because of the limited sample size, subgroup analyses according to OSAHS severity, BMI, CPAP adherence, or therapeutic pressure were not performed; therefore, potential heterogeneity in treatment effects across these clinical characteristics could not be adequately assessed.

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Disclosure of conflict of interest

None.

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