Section: Miscellaneous

Original Research Article

EVALUATION OF METABOLIC CHANGES FOLLOWING SHORT-TERM CPAP THERAPY IN PATIENTS WITH OBSTRUCTIVE SLEEP APNEA AND METABOLIC SYNDROME

Sandeep Lamoria[1], Athul Thulasi[2], K Praveen Kumar[3]

[1]Professor, Department of Gastroenterology, ABVIMS and Dr. R.M.L. Hospital, New Delhi-1, India.
[2]Assistant Professor, Department of Pulmonology, Travancore Medical College, Kollam, Kerala, India.
[3]Assistant professor, Pulmonary Medicine, Mahbubnagar government medical college, Mahbubnagar, Telangana, India.

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Corresponding Author:
Dr. Sandeep Lamoria,
Professor, Department of Gastroenterology, ABVIMS and Dr. R.M.L. Hospital, New Delhi-1, India.
Email: doctorlamoria@yahoo.com

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ABSTRACT

Background: Obstructive sleep apnea (OSA) is strongly associated with metabolic syndrome (MetS) through mechanisms such as intermittent hypoxia, insulin resistance, and systemic inflammation. Continuous positive airway pressure (CPAP) therapy is the standard treatment for OSA; however, its short-term effects on metabolic parameters remain inadequately defined, particularly in the Indian population.

Materials and Methods: This prospective interventional study included 79 patients with moderate to severe OSA and coexisting metabolic syndrome. Baseline clinical, anthropometric, and biochemical parameters were recorded. Patients were initiated on CPAP therapy and followed up after 8–12 weeks. Pre- and post-intervention comparisons were performed using paired t-test or Wilcoxon signed-rank test, as appropriate. Correlation between CPAP adherence and metabolic improvement was assessed using Spearman correlation.

Results: Significant improvements were observed in fasting glucose (−12.2 mg/dL), fasting insulin, and HOMA-IR (all $p < 0.001$), along with a modest reduction in HbA1c ($p = 0.002$). Triglycerides decreased and HDL increased significantly, while LDL reduction was not significant. Both systolic and diastolic blood pressure showed significant reductions ($p < 0.001$). Waist circumference decreased significantly, whereas BMI remained unchanged. Epworth Sleepiness Scale scores improved markedly ($p < 0.001$). Greater CPAP adherence was associated with significantly better metabolic outcomes and showed a negative correlation with changes in key parameters.

Conclusion: Short-term CPAP therapy significantly improves metabolic parameters and reduces cardiometabolic risk in patients with OSA and MetS, particularly with good adherence.

Keywords: Obstructive sleep apnea; Continuous positive airway pressure; Metabolic syndrome; Insulin resistance; HOMA-IR.

INTRODUCTION

Obstructive sleep apnea (OSA) is a common sleep-related breathing disorder characterized by recurrent episodes of upper airway obstruction during sleep, leading to intermittent hypoxia, sleep fragmentation, and sympathetic overactivity. Globally, OSA affects nearly 9–38% of the adult population, with higher prevalence among males, obese individuals, and those with advancing age.[1] In India, emerging evidence suggests a rising burden of OSA, paralleling increasing rates of obesity and lifestyle-related disorders.[2]

Metabolic syndrome (MetS) represents a cluster of cardiometabolic risk factors including central obesity, insulin resistance, hypertension, hyperglycaemia, and dyslipidaemia. It is associated with a two-fold increase in cardiovascular outcomes

and a five-fold increased risk of type 2 diabetes mellitus.[3] The prevalence of MetS ranges between 20–30% globally and is reported to be even higher in South Asian populations due to genetic susceptibility and environmental influences.[4]

A growing body of evidence highlights a strong bidirectional association between OSA and metabolic syndrome. Intermittent hypoxia and sleep fragmentation in OSA trigger oxidative stress, systemic inflammation, and activation of the hypothalamic–pituitary–adrenal axis, contributing to insulin resistance and endothelial dysfunction.[5] Conversely, obesity and visceral adiposity—central components of MetS—predispose individuals to upper airway collapse, thereby exacerbating OSA severity. Studies indicate that up to 60–70% of patients with OSA may have coexisting metabolic syndrome.[6]

Continuous positive airway pressure (CPAP) therapy remains the gold standard treatment for OSA, effectively preventing upper airway collapse and improving nocturnal oxygenation. Beyond symptomatic relief, CPAP has been shown to modulate several pathophysiological mechanisms implicated in metabolic dysfunction. Short-term CPAP therapy has demonstrated improvements in insulin sensitivity, blood pressure control, and markers of systemic inflammation, although findings across studies remain heterogeneous.[7] Some randomized trials report significant reductions in fasting glucose, HOMA-IR, and systolic blood pressure within weeks of therapy, whereas others suggest minimal metabolic benefit, possibly due to variations in adherence, duration of therapy, and baseline metabolic status.[8]

Given the high prevalence of coexisting OSA and metabolic syndrome and their synergistic impact on cardiovascular morbidity, evaluating the metabolic effects of CPAP therapy is of considerable clinical importance.[9] While long-term benefits of CPAP are well documented, the extent to which short-term CPAP therapy can influence metabolic parameters in patients with MetS remains inadequately explored, particularly in the Indian population.[10] Understanding these early therapeutic effects may help in reinforcing treatment adherence and guiding integrated management strategies.[11] Therefore, the present study aimed to evaluate the effect of short-term CPAP therapy on metabolic parameters in patients with obstructive sleep apnea and metabolic syndrome.

MATERIALS AND METHODS

Study Design and Setting

This prospective interventional study was conducted in the Department of General Medicine in association with Pulmonary Medicine at a tertiary care teaching hospital over a period of 12 months between August 2024 to July 2025. The study was designed to evaluate the effect of short-term continuous positive airway pressure (CPAP) therapy on metabolic parameters in patients diagnosed with obstructive sleep apnea (OSA) and metabolic syndrome (MetS). Institutional Ethics Committee approval was obtained prior to the commencement of the study, and all procedures were conducted in accordance with the ethical standards of the Declaration of Helsinki. Written informed consent was obtained from all participants before enrollment.

Study Population

Adult patients aged 18–65 years presenting with symptoms suggestive of OSA, such as excessive daytime sleepiness, loud snoring, witnessed apneas, or non-restorative sleep, were screened. Diagnosis of OSA was confirmed using overnight polysomnography (PSG), and severity was classified based on apnea–hypopnea index (AHI) as mild (5–14 events/hour), moderate (15–29 events/hour), and severe (≥30 events/hour). Only patients with moderate to severe OSA were included in the study to ensure clinical relevance of CPAP therapy.

Metabolic syndrome was diagnosed according to the National Cholesterol Education Program Adult Treatment Panel III (NCEP ATP III) criteria, requiring the presence of at least three of the following: waist circumference >102 cm in men or >88 cm in women (with Asian cut-offs applied where appropriate), fasting plasma glucose ≥100 mg/dL or on treatment for diabetes, blood pressure ≥130/85 mmHg or on antihypertensive therapy, triglycerides ≥150 mg/dL, and HDL cholesterol <40 mg/dL in men or <50 mg/dL in women.

Inclusion and Exclusion Criteria

Patients with confirmed moderate to severe OSA and coexisting metabolic syndrome who were CPAP-naïve were included. Exclusion criteria comprised patients with central sleep apnea, chronic respiratory failure, severe chronic obstructive pulmonary disease, unstable cardiovascular disease, chronic kidney or liver disease, active infection, pregnancy, or those already on CPAP or other forms of positive airway pressure therapy. Patients on medications significantly affecting metabolic parameters (such as systemic corticosteroids) were also excluded.

Sample Size and Sampling Technique

The sample size was calculated based on the expected reduction in insulin resistance (HOMA-IR) following CPAP therapy, using the formula for comparison of means: $n=(Z_{\alpha/2}+Z_{\beta})^2\times 2\sigma^2/d^2 n$

where $Z_{\alpha/2}=1.96$ at 95% confidence level, $Z_{\beta}=0.84$ for 80% power, σ represents the standard deviation, and d is the expected mean difference. Based on findings from a recent Indian study demonstrating significant improvement in metabolic parameters following CPAP therapy, and accounting for an anticipated dropout rate of 10–15%, the final sample size was fixed at 79 participants.[10] Consecutive eligible patients attending the sleep clinic during the study period were recruited using a non-probability consecutive sampling method.

Baseline Assessment

All enrolled participants underwent a detailed clinical evaluation including demographic data, anthropometric measurements (height, weight, body mass index, and waist circumference), and blood pressure measurement using standardized techniques. Daytime sleepiness was assessed using the Epworth Sleepiness Scale (ESS). Overnight attended polysomnography was performed using a standardized system, recording parameters such as electroencephalography, electrooculography, electromyography, airflow, thoracoabdominal movements, oxygen saturation, and electrocardiography.

Baseline laboratory investigations included fasting blood glucose, fasting insulin levels, glycated hemoglobin (HbA1c), lipid profile (total cholesterol, triglycerides, HDL, LDL), and calculation of insulin resistance using the Homeostatic Model Assessment of Insulin Resistance (HOMA-IR). Blood samples were collected after an overnight fast of at least 8–10 hours and analyzed using standardized laboratory methods.

Intervention (CPAP Therapy)

All eligible patients underwent CPAP titration using either manual titration during an attended PSG or auto-titrating CPAP devices to determine the optimal therapeutic pressure required to eliminate apneas, hypopneas, and snoring. Following titration, patients were initiated on fixed-pressure CPAP therapy and were instructed to use the device for at least 4–6 hours per night.

Compliance with CPAP therapy was monitored through built-in device usage data, and adherence was defined as usage of ≥4 hours per night on at least 70% of nights. Patients received regular follow-up support, including mask fitting, troubleshooting, and counseling to improve adherence.

Duration of Follow-up

Patients were followed up after a short-term duration of 8–12 weeks of CPAP therapy. During follow-up visits, adherence data were recorded, and patients were reassessed clinically.

Outcome Measures

The primary outcome measures included changes in metabolic parameters such as fasting blood glucose, fasting insulin, HOMA-IR, lipid profile, and blood pressure after short-term CPAP therapy. Secondary outcomes included changes in body mass index, waist circumference, and Epworth Sleepiness Scale scores.

Follow-up Assessment

At the end of the follow-up period, all baseline investigations were repeated under similar conditions. Anthropometric measurements and blood pressure were reassessed using the same standardized methods, and fasting blood samples were collected for biochemical analysis. Changes in metabolic parameters before and after CPAP therapy were recorded and compared.

Statistical Analysis: Data were entered into Microsoft Excel and analyzed using Statistical Package for the Social Sciences (SPSS) version 20.0. Continuous variables were expressed as mean ± standard deviation, while categorical variables were presented as frequencies and percentages. The normality of data distribution was assessed using the Kolmogorov–Smirnov test. For within-group comparisons of baseline and post-CPAP values, paired t-test was applied for normally distributed variables (including fasting blood glucose, fasting insulin, HOMA-IR, triglycerides, HDL, systolic and diastolic blood pressure, and Epworth Sleepiness Scale score), while the Wilcoxon signed-rank test was used for variables not normally distributed (including HbA1c, LDL cholesterol, body mass index, and waist circumference), as appropriate. Between-group comparisons based on CPAP adherence (good adherence ≥4 hours/night vs poor adherence <4 hours/night) were performed using independent t-test or Mann–Whitney U test depending on normality of data distribution. Correlation between CPAP usage duration (hours/night) and change in metabolic parameters (Δ values) was assessed using Spearman rank correlation coefficient (ρ). A p-value of <0.05 was considered statistically significant.

RESULTS

The study included 79 participants with a mean age of 52.8 ± 9.6 years, predominantly males (73.4%). The cohort demonstrated a high burden of obesity and central adiposity, with a mean BMI of 29.7 ± 4.3 kg/m² and waist circumference of 101.6 ± 8.9 cm. Most participants had moderate to severe OSA, with a mean AHI of 32.9 ± 11.7 events/hour; 60.8% had severe OSA. Baseline metabolic parameters indicated significant metabolic derangement, including elevated fasting glucose (124.5 ± 28.6 mg/dL), triglycerides (186.2 ± 46.5 mg/dL), low HDL (38.4 ± 6.2 mg/dL), and increased insulin resistance (HOMA-IR 4.1 ± 1.6). The mean ESS score of 14.2 ± 4.1 reflected substantial daytime sleepiness. [Table 1]

Table 1: Baseline Demographic and Clinical Characteristics of Study Participants (n = 79)

Variable	Frequency (%)/mean ± SD
Age (years)	52.8 ± 9.6
Gender	
Female	21 (26.6%)
Male	58 (73.4%)
BMI (kg/m²)	29.7 ± 4.3

Waist circumference (cm)	101.6 ± 8.9
Systolic BP (mmHg)	142.3 ± 14.8
Diastolic BP (mmHg)	88.6 ± 9.7
ESS score	14.2 ± 4.1
AHI (events/hour)	32.9 ± 11.7
Type of OSA	
Moderate OSA	31 (39.2%)
Severe OSA	48 (60.8%)
Fasting glucose (mg/dL)	124.5 ± 28.6
HbA1c (%)	6.8 ± 1.1
Triglycerides (mg/dL)	186.2 ± 46.5
HDL (mg/dL)	38.4 ± 6.2
HOMA-IR	4.1 ± 1.6

BMI: Body Mass Index, BP: Blood Pressure, ESS: Epworth Sleepiness Scale, AHI: Apnea–Hypopnea Index, HDL: High-Density Lipoprotein, HOMA-IR: Homeostatic Model Assessment of Insulin Resistance.

Short-term CPAP therapy resulted in significant improvements in key metabolic parameters. There was a marked reduction in fasting glucose (−12.2 mg/dL), fasting insulin (−2.9 µIU/mL), and HOMA-IR (−0.9), all of which were highly statistically significant ($p < 0.001$). HbA1c also showed a modest but significant reduction ($p = 0.002$). Lipid profile improved with a significant decrease in triglycerides and increase in HDL levels ($p < 0.01$), while reduction in LDL cholesterol was not statistically significant ($p = 0.091$). Additionally, both systolic and diastolic blood pressures demonstrated significant reductions following CPAP therapy ($p < 0.001$). [Table 2]

Table 2: Comparison of Metabolic Parameters Before and After Short-Term CPAP Therapy (n = 79)

Parameter	Baseline	Post-CPAP	Mean Difference	p-value
	Mean ± SD			
Fasting glucose (mg/dL)	124.5 ± 28.6	112.3 ± 24.7	−12.2	<0.001
Fasting insulin (µIU/mL)	16.8 ± 6.9	13.9 ± 5.8	−2.9	<0.001
HOMA-IR	4.1 ± 1.6	3.2 ± 1.3	−0.9	<0.001
HbA1c (%)	6.8 ± 1.1	6.5 ± 0.9	−0.3	0.002
Triglycerides (mg/dL)	186.2 ± 46.5	168.4 ± 39.8	−17.8	<0.001
HDL (mg/dL)	38.4 ± 6.2	41.1 ± 6.5	2.7	0.004
LDL (mg/dL)	122.6 ± 32.1	118.9 ± 30.5	−3.7	0.091
Systolic BP (mmHg)	142.3 ± 14.8	134.6 ± 12.9	−7.7	<0.001
Diastolic BP (mmHg)	88.6 ± 9.7	83.9 ± 8.5	−4.7	<0.001

LDL: Low-Density Lipoprotein; SBP: Systolic Blood Pressure; DBP: Diastolic Blood Pressure.

Anthropometric changes following short-term CPAP therapy were modest. Although BMI showed a slight reduction, it did not reach statistical significance ($p = 0.082$). However, waist circumference demonstrated a small but significant decrease ($p = 0.018$), indicating improvement in central obesity. A substantial and clinically meaningful reduction in daytime sleepiness was observed, with ESS scores decreasing significantly from 14.2 ± 4.1 to 7.6 ± 3.2 ($p < 0.001$). [Table 3]

Table 3: Changes in Anthropometric and Clinical Parameters After CPAP Therapy (n = 79)

Parameter	Baseline	Post-CPAP	Mean Difference	p-value
	Mean ± SD			
BMI (kg/m²)	29.7 ± 4.3	29.3 ± 4.1	−0.4	0.082
Waist circumference (cm)	101.6 ± 8.9	99.8 ± 8.5	−1.8	0.018
ESS score	14.2 ± 4.1	7.6 ± 3.2	−6.6	<0.001

BMI: Body Mass Index, ESS: Epworth Sleepiness Scale

Patients with good CPAP adherence (≥4 hours/night) showed significantly greater improvements in metabolic parameters compared to those with poor adherence. Reduction in HOMA-IR was significantly higher in the good adherence group (−1.2 ± 0.6 vs −0.4 ± 0.5, $p < 0.001$). Similarly, reductions in fasting glucose and systolic blood pressure were more pronounced in adherent patients ($p < 0.001$ for both), highlighting the dose-dependent metabolic benefits of CPAP therapy. [Table 4]

Table 4: Association Between CPAP Adherence and Metabolic Improvement

Variable	Good Adherence (≥4 hrs/night) (n=52)	Poor Adherence (<4 hrs/night) (n=27)	p-value
	(Mean ± SD)	(Mean ± SD)	
Reduction in HOMA-IR	−1.2 ± 0.6	−0.4 ± 0.5	<0.001
Reduction in Fasting glucose (mg/dL)	−16.5 ± 10.8	−6.8 ± 8.9	<0.001
Reduction in SBP (mmHg)	−10.2 ± 6.7	−3.1 ± 5.9	<0.001

A significant negative correlation was observed between CPAP usage duration and changes in metabolic parameters. Increased CPAP usage was associated with greater reductions in HOMA-IR ($r = -0.48$, $p < 0.001$), fasting glucose ($r = -0.42$, $p < 0.001$), systolic blood pressure ($r = -0.39$, $p = 0.002$), and triglycerides ($r = -0.31$, $p = 0.008$). These findings suggest a dose–response relationship, with higher CPAP adherence leading to greater metabolic improvement. [Table 5]

Table 5: Correlation Between CPAP Usage Duration and Changes in Metabolic Parameters

Variable	Correlation Coefficient (r)	p-value
CPAP hours vs Δ HOMA-IR	−0.48	<0.001
CPAP hours vs Δ Fasting glucose	−0.42	<0.001
CPAP hours vs Δ SBP	−0.39	0.002
CPAP hours vs Δ Triglycerides	−0.31	0.008

Spearman rank correlation; ρ: correlation coefficient; Δ indicates change from baseline

DISCUSSION

The present study evaluated the effect of short-term CPAP therapy on metabolic parameters in patients with obstructive sleep apnea (OSA) and metabolic syndrome (MetS), demonstrating significant improvements in glycaemic control, insulin resistance, lipid profile, and blood pressure. The study population was characterized by a high prevalence of moderate to severe OSA (mean AHI 32.9 ± 11.7 events/hour) and marked metabolic derangements, which is consistent with prior Indian studies by Kumari et al., Srivastava et al., Ravichandran et al., and Suri et al., reporting a strong overlap between OSA and MetS in middle-aged, predominantly male, overweight individuals.[12,13,14]

A key finding of this study was the significant reduction in insulin resistance following CPAP therapy, as evidenced by a decrease in HOMA-IR from 4.1 ± 1.6 to 3.2 ± 1.3 ($p < 0.001$). This aligns with previous studies by Steiropoulos et al., and Iftikhar et al., that have shown improvements in insulin sensitivity after CPAP use, likely mediated through attenuation of intermittent hypoxia and sympathetic overactivity.[15,16] Intermittent hypoxia in OSA induces oxidative stress and systemic inflammation, leading to impaired insulin signaling pathways. CPAP therapy mitigates these effects by restoring nocturnal oxygenation, thereby improving peripheral glucose uptake and reducing hepatic glucose output.[17] Comparable findings have been reported in meta-analyses in Daneshvar et al., and Liu et al., where CPAP therapy resulted in significant reductions in insulin resistance, particularly in patients with good adherence.[18,19]

The study also demonstrated a significant reduction in fasting blood glucose (−12.2 mg/dL, $p < 0.001$) and HbA1c (−0.3%, $p = 0.002$), indicating early glycaemic benefits even with short-term therapy. Similar reductions have been observed in studies by Giampá et al., and Banghøj et al., although the magnitude of HbA1c reduction tends to be modest in short-duration studies.[20,21] This may be explained by the relatively longer turnover time of glycated hemoglobin, suggesting that more pronounced changes may occur with prolonged CPAP use.[21]

In terms of lipid profile, a significant reduction in triglyceride levels (−17.8 mg/dL, $p < 0.001$) and increase in HDL cholesterol (+2.7 mg/dL, $p = 0.004$) were observed, while LDL reduction was not statistically significant. These findings are consistent with previous studies by Chen et al., and Chen et al., which report variable effects of CPAP on lipid parameters.[22,23] The improvement in triglycerides and HDL may be attributed to reduced sympathetic activity and improved adipocyte metabolism, whereas LDL levels are less responsive in the short term and may require longer intervention periods.[24]

Blood pressure showed significant improvement, with reductions in both systolic (−7.7 mmHg) and diastolic (−4.7 mmHg) values ($p < 0.001$). This is clinically relevant, as even modest reductions in blood pressure are associated with decreased cardiovascular risk. CPAP-mediated blood pressure reduction is thought to occur through decreased sympathetic tone, improved endothelial function, and normalization of nocturnal dipping patterns.[25] These findings are in agreement with prior Campos-Rodriguez et al., and Muxfeldt et al., demonstrating a mean systolic blood pressure reduction of approximately 5–7 mmHg with CPAP therapy.[26,27]

Anthropometric parameters showed limited change, with a non-significant reduction in BMI ($p = 0.082$) but a modest yet significant decrease in waist circumference ($p = 0.018$). This suggests that short-term CPAP therapy may influence central adiposity without substantial changes in overall body weight.[28] Importantly, a marked improvement in daytime sleepiness was observed, with ESS scores decreasing significantly (−6.6 points, $p < 0.001$), reflecting improved sleep quality and functional outcomes, consistent with established benefits of CPAP therapy.[29]

A notable strength of this study is the demonstration of a dose–response relationship between CPAP adherence and metabolic improvement. Patients with good adherence (≥4 hours/night) showed significantly greater reductions in HOMA-IR, fasting glucose, and systolic blood pressure compared to poorly adherent individuals (all $p < 0.001$). Furthermore, Spearman correlation analysis revealed significant negative correlations between CPAP usage duration and changes in HOMA-IR (ρ =

−0.48), fasting glucose (ρ = −0.42), and blood pressure (ρ = −0.39), indicating that increased CPAP use is associated with greater metabolic benefit. These findings reinforce the importance of adherence and are consistent with previous studies by Choudhary et al., and Alemohammad et al., demonstrating that therapeutic efficacy of CPAP is highly dependent on duration of nightly use.[30,31]

Limitations

This study has certain limitations. The relatively small sample size (n=79) and single-center design may limit generalizability. The short duration of CPAP therapy may not fully capture long-term metabolic effects, particularly for parameters such as HbA1c and LDL cholesterol. Lifestyle factors including diet and physical activity were not strictly controlled. Additionally, residual confounding due to concurrent medications for metabolic conditions cannot be entirely excluded.

CONCLUSION

Short-term CPAP therapy in patients with obstructive sleep apnea and metabolic syndrome resulted in significant improvements in insulin resistance, glycaemic control, triglyceride levels, HDL cholesterol, and blood pressure, with no significant change in LDL cholesterol or overall body mass index. A reduction in central obesity and marked improvement in daytime sleepiness were also observed. Importantly, better CPAP adherence was associated with greater metabolic benefit, demonstrating a clear dose–response relationship. These findings highlight the role of CPAP not only as a symptomatic treatment for OSA but also as an effective adjunct in improving cardiometabolic risk. Early initiation and optimal adherence to CPAP therapy may enhance metabolic outcomes in this high-risk population.

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