Commentary

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Influence of Smoking on Metabolic Improvements During Airway Pressure Therapy Treatment

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Description

Obstructive Sleep Apnea (OSA) is a disorder marked by upper airway obstruction during sleep, leading to fragmented sleep and intermittent hypoxia, characterized by episodes of apnea or hypopnea. OSA is linked to various comorbidities, including cardiovascular, neurological and endocrinological disorders. Metabolic syndrome, which includes central obesity, low levels of High-Density Lipoprotein (HDL), elevated blood pressure and increased fasting plasma glucose and triglycerides, is more prevalent and severe in individuals with OSA. Effective treatment of OSA using Positive Airway Pressure (PAP) therapy has been shown to improve several metabolic syndrome markers, underscoring the connection between OSA and metabolic syndrome. Research indicates that PAP treatment can lead to significant reductions in cholesterol and glycosylated haemoglobin levels among OSA patients.

Impact of active smoking on metabolic

Smoking is a significant risk factor that influences both OSA and metabolic syndrome. It can increase the risk of metabolic syndrome and its complications by two to three times. Additionally, smoking exacerbates OSA by inducing chronic inflammation and neuromuscular dysfunction in the upper airway and may reduce adherence to PAP therapy due to its associated side effects. However, the impact of active smoking on metabolic syndrome parameters in OSA patients undergoing PAP treatment has not been previously reported. This study aims to investigate whether active smokers with OSA experience fewer metabolic improvements from PAP therapy compared to non-smokers.

This study was designed prospectively to analyze clinical, laboratory and Polysomnography (PSG) data from patients admitted to our sleep and disorders unit with signs of sleep-disordered breathing. PSG was performed following the guidelines from the American Academy of Sleep Medicine and OSA diagnosis was based on the international classification of sleep disorders. Adults over 18 with an Apnea-Hypopnea Index (AHI) of 15 or more per hour who consented to participate were included. Exclusion criteria surround prior or current PAP therapy, pregnancy, certain chronic diseases and substance abuse. Ethical approval was obtained and informed consent was collected from all participants.

All patients underwent clinical evaluations and their smoking status was recorded as either active smoker or non-smoker. Active smoking was defined as inhaling mainstream smoke, including occasional social use. Participants were categorized as non-smokers if they had never smoked. Patients exposed to smoke or who used e-cigarettes were excluded. Demographic and anthropometric data, including age, gender, Body Mass Index (BMI) and circumferences, were recorded.

Fasting blood samples were taken the morning after PSG to assess various parameters, including plasma glucose, HbA1c, serum insulin and triglycerides. A follow-up examination was conducted three months after initiating PAP therapy, during which compliance with the therapy was assessed based on machine output. Participants who did not adhere to therapy or had ineffective use were excluded from the study. Changes in clinical, anthropometric and biochemical parameters were recorded.

The study included 115 patients, with 53.3% identified as active smokers. The average age of participants was 53.4 years and active smokers were significantly younger than non-smokers. The mean BMI was 31.5 kg/m² and there were no significant differences in anthropometric measurements between the two groups.

PSG results showed that the mean AHI was lower in active smokers compared to non-smokers, though the difference was not significant. However, the incidence of severe OSA was significantly higher in non-smokers. After three months of PAP therapy, no significant changes were observed in clinical or anthropometric parameters. Biochemical parameters, including triglycerides, fasting glucose and HOMA-IR, also showed no significant differences between active smokers and non-smokers.

In terms of stable, decreased, or increased parameters, serum leptin levels decreased in 78.6% of non-smokers after treatment but only in 46.7% of active smokers, indicating a significant difference. Active smokers also exhibited an increase in leptin levels, while no such increase was seen in non-smokers. Correlation analyses indicated that elevated leptin levels in

active smokers were positively associated with triglycerides and Homeostatic Model Assessment for Insulin Resistance (HOMA-IR), although these correlations were not statistically significant.

The key finding of this study is that active smoking leads to elevated leptin levels in OSA patients, even after effective PAP therapy. Previous research indicates that OSA is linked to increased serum leptin and treatment with PAP generally reduces leptin levels. In non-smokers, significant decreases in leptin were noted after treatment, while in active smokers, the effect of PAP was diminished.

Smoking has been shown to impair insulin signalling and contribute to higher serum leptin levels, which may explain the lack of positive metabolic changes observed in smokers after PAP treatment. Increased leptin levels are associated with obesity and insulin resistance and although correlations were noted, they were not statistically significant.

The study has some limitations, including a small sample size and short follow-up duration. Future research should survey a larger cohort over a longer period, taking into account factors such as total smoking exposure and body fat composition to better understand the relationship between smoking, OSA and metabolic syndrome.