

Obstructive Sleep Apnea and Type 2 Diabetes Mellitus: Really Associated Epidemics?

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Obstructive sleep apnea (OSA) is a clinical syndrome of sleep-disordered breathing (SDB) that is an abnormal breathing pattern during sleep. SDB is usually measured as the apnea-hypopnea index (AHI). SDB approximately occurs in 24% of middle-aged men and 9% Of middle-aged women. SDB prevalence is higher among older, up to 60 % of community-residing elderly people and more obese populations. Among healthy men with 30 kg/m² of the mean body mass index (BMI) have a 60 % prevalence of SDB and a 27% of OSA. Why such a large proportion of individuals with SDB lack of clinical manifestations of sleep disruption remains unclear and thus, they do not meet the criteria for clinical syndrome of OSA. A previous study demonstrated that 82% of men and 93% of women with moderate to severe OSA remain undiagnosed.

OSA and SDB result in hypoxia that contribute to sleep fragmentation and symptoms of excessive daytime sleepiness. Sleepiness also increases sympathetic activity, which can decrease insulin sensitivity, glucose tolerance, and increase blood sugar levels. The American Diabetes Association (ADA) reported that the global prevalence of diabetes is expected to be 300 million by the year 2025. Male gender, obesity, and older age are well-known risk factors for development of OSA and these risk factors are also associated with the likelihood of development of type 2 diabetes mellitus (DM). Sleep fragmentation for at least two nights can decrease insulin sensitivity and impair glucose metabolism that appears to be mediated via alterations in sympathovagal balance, with a shift toward increased sympathetic nervous system activity during sleep and wakefulness. In healthy volunteers with transient hypoxia, there were elevation of serum sympathomimetic hormones (epinephrine, nor-epinephrine, and cortisol). Elevation of serum epinephrine contributes to an increase of hepatic gluconeogenesis and decrease of skeletal muscle reuptake of glucose resulting in hyperglycemia, in addition to low-grade systemic inflammation, dysregulation of the hypothalamic-pituitary-adrenal (HPA) axis, increase in oxidative stress, and activation of the sympathetic nervous system. The combined interactions of nocturnal hypoxia, hyperglycemia, and obesity result in elevation of several systemic inflammatory biomarkers, such as tumor necrosis factor (TNF)- α , interleukin (IL)-6, high-sensitivity C-reactive protein (hsCRP), uric acid, and fibrinogen. Elevation of these biomarkers contributes to insulin resistance and impairment of glucose utilization. Several previous studies revealed that OSA remained an independent predictor of proliferative retinopathy although after adjusting for conventional risk factors and novel biomarkers for diabetic retinopathy. A previous study demonstrated an elevation of inflammatory biomarkers, such as lipid peroxide, nitrotyrosine, C3, high-sensitive CPR, acylation stimulating protein, and components of the membrane attack complex (leading to activation of alternative complement pathway), but a decrease in the levels of IgM and NK cell percentage in OSA patients with DM, compared to diabetic patients without OSA. A previous study revealed that there was a fourfold increase in the odds of peripheral neuropathy in OSA patients with DM that has been postulated indicating hexosamine, polyol, advanced glycation end products, and protein kinase pathways. A previous study reported that there was an increase in serum cystatin C levels in severe OSA patients with DM, indicating the development of renal failure. The studies on OSA contributing to the progression of diabetic nephropathy is limited. According to the overall similarity in the pathophysiological mechanisms, OSA may lead to the development of diabetic nephropathy. Several

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previous studies demonstrated positive effect of the continuous-positive-airway- pressure (CPAP) treatment on insulin sensitivity in OSA patients, particularly, in patients with BMI of less than 30 kg/m². Glycated hemoglobin (HbA1C) levels is a better biomarker of efficacy of CPAP treatment due to day-to-day fluctuation of insulin resistance and blood glucose.

In conclusion, the association between OSA and type 2 DM remains irrefutable, irrespective of the direction of causality. The improvement of glycemic control in patients with CPAP therapy, the judgment should be reserved until long-term rigorously conducted prospective studies can explore knowledge in this field. Clinicians should individualize their decisions, based on particular patients' needs although the strong recommendation for CPAP therapy in OSA patients with DM remains controversial.

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