

Metabolic Comorbidities Associated with Obstructive Sleep Apnea

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DESCRIPTION

Obstructive Sleep Apnea (OSA) is generally characterized by partial or complete disruption of the upper respiratory tract leading to sleep apnea events, Sleep Fragmentation (SF), and Chronic Intermittent Hypoxia (CIH). A recent European study based on polysomnography findings in the general population estimates that the prevalence of moderate to severe OSA is 30% in men and 13% in women. Obesity is a major risk factor for OSA and a common risk factor for various comorbidities. However, even non-obese OSA patients are burdened with comorbidity.

Chronic intermittent hypoxia and sleep loss due to SF are associated with insulin resistance and metabolic dysfunction. Recently, it has been suggested that adipose tissue dysfunction plays an important role in the metabolic disorders of OSA. There is increasing evidence that OSA, especially CIH, is associated with non-alcoholic fatty liver disease. Carotid Body (CB) is a multisensory organ that measures not only hypoxia and hypercapnia, but also insulin resistance and glucose metabolism. OSA alters brain metabolism, which can predispose to neurocognitive impairment. In healthy people, the gut microbiota is mainly composed of beneficial bacteria. It has been suggested that imbalances in the composition of the gut microbiota are responsible for various diseases. Loss of sleep, SF and CIH are associated with dysbiosis. Recently, changes in the gut microbiota have been associated with obesity, diabetes, dyslipidemia, cardiac respiratory control, hypertension, and coronary artery disease-all these are common comorbidities of OSA. The relationship between OSA and metabolic disorders is clear. On the one hand, sleep apnea causes Intermittent Hypoxia (IH) and SF, leading to and exacerbating obesity, metabolic syndrome, Type 2 Diabetes (T2D) and Non-Alcoholic Fatty Liver Disease (NAFLD). Obesity, on the other hand, is considered a major risk factor for the development and progression of OSA.

Obstructive sleep apnea is independently associated with metabolic syndrome or its core components such as visceral obesity, hypertension, insulin resistance, impaired glucose tolerance, and dyslipidemia. Association of OSA with some

pathological features of metabolic syndrome such as hypertension during the day has become strongly solidified, and an association between insulin resistance and metabolic disorders has only recently emerged. In some clinical studies, patients with OSA, the Apnea-Hypopnea Index (AHI) and OSA severity, insulin resistance and pancreatic β-cell dysfunction, and fasting blood glucose levels, regardless of obesity or fat mass has been shown to contribute to increased insulin resistance and impaired oral glucose tolerance. As expected from the association between OSA and insulin resistance and impaired glucose tolerance, OSA is a risk factor for the development of type 2 diabetes. Several observational studies have shown that OSA and its severity are positively correlated with the incidence of type 2 diabetes, regardless of obesity, Body Mass Index (BMI) or age. The association between OSA and metabolic disorders is also supported by the effect of Continuous Positive Airway Pressure (CPAP) on the pathophysiological variables that characterize metabolic disorders. CPAP therapy in patients with OSA T2D reduced postprandial glucose and glycated hemoglobin and increased insulin sensitivity.

CONCLUSION

Hippocrates had already suggested a link between illness and sleep, but the first systematic study of the effects of bacterial infections on sleep was conducted about 30 years ago. They showed that bacterial infections first increased non-rem sleep and subsequently inhibited non-rem sleep. Rapid Eye Movement (REM) sleep is suppressed during infection. In addition, depletion of the gut microbiota by antibiotic treatment alters the sleep structure of mice. The composition of the gut microbiota has recently been reported to be associated with sleep disorders such as narcolepsy, insomnia and OSA. The gut microbiota of patients with insomnia was characterized by reduced microbial abundance and diversity, depletion of anaerobic and short-chain fatty acid-producing bacteria, compared to healthy controls. Lachnospira and bacteroides were characteristic bacteria in patients with acute insomnia, and Faecalibacterium and Blautia were characteristic bacteria to distinguish patients with chronic insomnia from healthy controls.

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