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Mini Review

Sleep Related Disorders in Obese Patient

Hakan Silek[1]* and Yasar Kucukardalı[2]

[1]Yeditepe University, Neurology Department, Turkey

[2]Yeditepe University, Internal Medicine Department, Turkey

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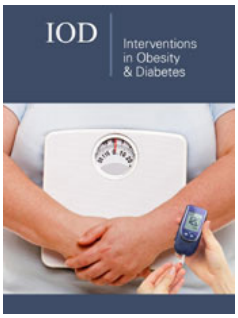

***Corresponding author:** Hakan Silek, Yeditepe University, Neurology Department, Turkey

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Abstract

There is growing evidence for possible impact of sleep disorders in obesity. The contribution of sleep disorders to obesity tends to increase, shortening sleep times in modern industrialized life habits is main reason for this problem to rise. The metabolic effects of sleep disorders are wide ranged, and we discussed the effects of sleep on obesity.

Keywords: Obesity; Obstructive sleep apnea; Hypertension

Mini Review

There is a dramatic increase of obesity in last four decades, it is well known that the reason for this increase is a change in feeding behaviour of the population, high-calorie food intake and reduction in physical activity. This is an enormous change in obesity rates in a relatively short period and there was one missing predisposing factor that was overlooked until today. Sleep-related disorders such as poor-quality sleep reduced duration of sleep and sleep-related breathing disorders. While the sleep-related breathing disorders are a predisposing factor for obesity, obesity itself causes an increase in Obstructive Sleep Apnea (OSA) disorders. Obesity is one of the most important independent risk factors for sleep obstructive apnea OSA Prevalence of obstructive sleep apnea in the general population rates vary but average at ≥5 events/h Apnea-Hypopnea Index (AHI), the overall population prevalence ranged from 9% to 38% and was higher in men [1]. The prevalence of OSA in the non-obese population under 50 years of age is between 1-4%. With obesity, this rate increases to 30-40%. And 50-98% in morbidly obese patients [2-4]. Total body weight, BMI and fat distribution are highly correlated with OSA. Every 10kg increase in weight increases the OSA by twice. During sleep in obese patients the posterior movement of the tongue root in the upper respiratory tract, these patients have extrinsic narrowing of the area surrounding collapsible region of the pharynx and regional soft tissue enlargement can cause narrowing of restrictive airways during the night and in reduced upper airway size secondary to mass effect of the large abdomen on the chest wall, diaphragm and tracheal traction. Observational studies have illustrated that the prevalence of OSA is over 30% among hypertension patients and nearly 80 % among resistant hypertensive patients [5]. Hou's meta-analysis demonstrates that OSA is related to an increased risk of resistant hypertension, with a stronger association among Caucasian populations and male OSA patients. OSA participants had an extra 1.842 –fold risk for resistant hypertension prevalence compared to non-OSA participants. Mild, moderate and severe OSA is associated with essential hypertension [6]. It is well known that OSA induces intermittent hypoxemia causes oxidative stress, leading to dysfunction of vascular endothelium. Meanwhile, the excessive outflow of sympathetic vasoconstrictor together with diminished nitric oxide bioavailability plays a role in blood pressure elevation. During the episodes of OSA up-regulate sympathetic excitation which acts on the chemoreflex and may consequently result in hypertension. In clinical observations, sympathetic nervous activity, reflected by 24–hour urinary catecholamine excretion, is increased in individuals with sleep-disordered breathing [7]. One of the possible mechanisms of obesity causing OSAS is leptin as a neurohormone is linked to obesity and OSAS, however obese and patients with OSAS independently have leptin resistance, sleep deprivation causing reduced leptin activity resulting in increased appetite [8]. Treatment of OSA with CPAP has resulted in decreased hormonal levels of leptin and grelin [9].

Sleep quality and sleep duration are an important and independent factor alone for evaluation. There is scientific research of self-reporting sleep duration in the last two decades in which are enormous reducing in sleep duration of modern industrialized populations [10]. Sleep deprivation effects in hormones such as Leptin and Ghrelin have shown that reduction in sleep amounts in completely healthy individuals even in routine eating habits in very short time had to reduce the effect in serum levels of leptin and increasing of serum ghrelin [11]. The subject that participated in this study showed grater hunger, especially to carbohydrate food. Here is important to mention that sleep deprivation did not trigger enormous energy cost as would be expected the same energy amounts were spent in sleep non-restricted group and this was confirmed with a calorimeter. Sleep duration is an important impact factor for obesity. The impact of sleep duration on obesity has been more evident lately. Short sleep duration triggers obesity. There are quite complex mechanisms of how sleep duration is contributing to obesity formation [12]. Obesity can also contribute to the formation of restless legs syndrome both overall and abdominal adiposity are associated with increased likelihoods of having Restless Legs Syndrome (RLS) [13,14]. The exact mechanism by which obesity causes (RLS) is unknown.

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