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Impact of Smoking History and Nicotine Dependence on Sleep Characteristics in Patients with Obstructive Sleep Apnea-hypopnea Syndrome

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Introduction

Obstructive Sleep Apnea-Hypopnea Syndrome (OSAHS) is a prevalent sleep disorder characterized by recurrent episodes of partial or complete airway obstruction during sleep, leading to intermittent hypoxia, sleep fragmentation, and excessive daytime sleepiness. OSAHS is associated with significant cardiovascular, metabolic, and neurocognitive complications, making its early identification and management crucial for reducing morbidity and improving quality of life. Among the various risk factors influencing OSAHS severity and progression, smoking history and nicotine dependence have gained increasing attention due to their detrimental effects on respiratory function, upper airway stability, and sleep architecture. Smoking is known to cause airway inflammation, increased mucus production, and reduced neuromuscular control of the upper airway, all of which contribute to airway collapsibility and worsening OSAHS symptoms. This study aims to explore the relationship between smoking behavior, nicotine addiction, and alterations in sleep architecture among individuals with OSAHS, shedding light on potential mechanisms through which tobacco use exacerbates sleep-disordered breathing and its associated complications [1].

Description

The relationship between smoking, nicotine dependence, and OSAHS is multifaceted, involving both physiological and neurobiological mechanisms that contribute to sleep disturbances. Cigarette smoke contains thousands of toxic compounds that induce chronic airway inflammation, oxidative stress, and increased mucus production, all of which contribute to upper airway narrowing and increased collapsibility during sleep. In OSAHS patients, the cumulative effects of smoking exacerbate the structural and functional abnormalities in the upper airway, increasing the frequency and severity of apneic and hypopneic events. Additionally, nicotine a highly addictive substance found in tobacco products alters neurophysiological mechanisms regulating sleep-wake cycles, leading to sleep disturbances, increased arousals, and reduced sleep efficiency. Despite the well-established link between smoking and respiratory disorders, the impact of smoking history and nicotine dependence on sleep features in OSAHS patients remains an area of ongoing research. Additionally, smoking-related impairment of mucociliary clearance and increased airway resistance further contribute to nocturnal hypoxia and respiratory instability, worsening sleep fragmentation and reducing overall sleep quality [2].

Nicotine, the primary psychoactive component in tobacco, has a complex effect on sleep regulation due to its stimulant properties and interaction with the central nervous system. While nicotine initially promotes alertness and wakefulness by increasing the release of neurotransmitters such as dopamine, acetylcholine, and norepinephrine, chronic nicotine exposure disrupts the natural sleep-wake cycle. The withdrawal effects of nicotine during sleep also play a significant role in disrupting sleep continuity and increasing sleep fragmentation. Since nicotine has a relatively short half-life, individuals who are dependent on tobacco may experience withdrawal symptoms, including restlessness, irritability, and increased sympathetic activity, during sleep. This withdrawal-induced sleep disruption further exacerbates sleep fragmentation in OSAHS patients, leading to an increased frequency of micro arousals and a reduced ability to maintain deep, restorative sleep stages. Additionally, the interaction between nicotine dependence and nocturnal hypoxia in OSAHS patients may intensify sympathetic nervous system activation, leading to increased heart rate variability, elevated blood pressure, and higher cardiovascular risk Studies have shown that smoker's exhibit shorter total sleep duration, increased sleep latency, and reduced Slow-Wave Sleep (SWS) compared to non-smokers. In OSAHS patients, nicotine dependence may further disrupt sleep homeostasis by increasing nocturnal arousals, Reducing Rapid Eye Movement (REM) sleep, and prolonging the time spent in lighter sleep stages (N1 and N2). These alterations in sleep architecture contribute to excessive daytime sleepiness, cognitive impairment, and reduced overall sleep efficiency in OSAHS patients with a history of smoking [3].

Beyond physiological effects, smoking behavior is also associated with altered circadian rhythms and an increased prevalence of insomnia-related symptoms. Studies have suggested that chronic smokers may exhibit Delayed Sleep Phase Syndrome (DSPS), characterized by difficulty falling asleep and waking up at socially acceptable times. This circadian misalignment, combined with the already disrupted sleep patterns observed in OSAHS, may further impair overall sleep health and exacerbate daytime dysfunction. Furthermore, smoking has been linked to increased rates of mood disorders, including anxiety and depression, which are known to independently contribute to sleep disturbances in OSAHS patients. The bidirectional relationship between smoking, mood disorders, and sleep dysfunction highlights the need for a holistic approach in managing OSAHS patients with a history of tobacco use [4].

Epidemiological studies examining the association between smoking and OSAHS have consistently reported that current and former smokers have a higher risk of developing sleep-disordered breathing compared to non-smokers. Moreover, a dose-response relationship has been observed, with heavier smoking being associated with greater OSAHS severity, higher Apnea-Hypopnea Index (AHI) scores, and increased oxygen desaturation events. However, the effects of smoking cessation on sleep parameters in OSAHS patients remain a topic of debate. While quitting smoking leads to significant improvements in lung function, airway inflammation, and cardiovascular health, some studies suggest that former smokers may continue to experience residual sleep disturbances due to long-term damage to the upper airway and persistent alterations in sleep regulation From a clinical perspective, addressing smoking and nicotine dependence in OSAHS patients is essential for improving treatment outcomes and enhancing overall sleep health. Smoking cessation interventions, including behavioral counseling, Nicotine Replacement Therapy (NRT), and pharmacological treatments such as varenicline and bupropion, have shown efficacy in helping individuals reduce tobacco dependence.

Incorporating smoking cessation programs into OSAHS management

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plans may lead to reductions in airway inflammation, improved upper airway stability, and enhanced responsiveness to Positive Airway Pressure (PAP) therapy, which is the gold standard treatment for OSAHS. Additionally, targeted interventions to improve sleep hygiene, reduce nicotine withdrawal symptoms, and regulate circadian rhythms may further mitigate the impact of smoking on sleep quality in OSAHS patients. Future research should focus on longitudinal studies to assess the long-term effects of smoking cessation on sleep architecture, respiratory function, and OSAHS progression. Investigating the potential role of alternative nicotine delivery methods, such as electronic cigarettes, in modifying sleep parameters in OSAHS patients may also provide valuable insights. Furthermore, understanding the genetic and epigenetic factors that influence individual susceptibility to smoking-induced sleep disturbances may help in developing personalized treatment strategies for OSAHS patients with a history of tobacco use [5].

Conclusion

In conclusion, smoking history and nicotine dependence play a significant role in altering sleep features in patients with OSAHS, exacerbating sleep fragmentation, increasing nocturnal hypoxia, and disrupting normal sleep architecture. The interplay between airway inflammation, upper airway instability, and nicotine-induced neurophysiological changes contributes to the worsening of sleep-disordered breathing in smokers with OSAHS. Addressing tobacco use in this population is critical for improving sleep quality, reducing disease severity, and enhancing overall health outcomes. Integrating smoking cessation interventions, optimizing PAP therapy, and implementing behavioral strategies to improve sleep hygiene can collectively mitigate the adverse effects of smoking on OSAHS. As research continues to explore the complex relationship between smoking and sleep disorders, a better understanding of the mechanisms underlying nicotine-induced sleep disturbances will pave the way for more effective management approaches for OSAHS patients with a history of smoking.

Acknowledgement

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Conflict of Interest

None.

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