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LETTER TO THE EDITOR

Burdened breaths: The influence of depression on obstructive sleep apnea

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Abstract

Depression and metabolic syndrome could exacerbate the risks of the other, leading to a series of severe coexisting conditions. One notable comorbidity that must be mentioned is obstructive sleep apnea (OSA). Current studies suggested that depression increases susceptibility to OSA. As the prevalence of depression rises, it becomes critical to prevent and manage its complications or comorbidities, including OSA. Predictive models, non-invasive electroencephalogram monitoring, genetic research, and other promising technologies are being applied to the prevention, diagnosis, and personalized treatment of depression and OSA.

Key Words: Depression; Obstructive sleep apnea; Metabolic syndrome; Prevalence; Risk factor

Core Tip: Depression and obstructive sleep apnea frequently coexist and share similar symptoms. By exploring the causal relationship between depression and obstructive sleep apnea, we may gain new insights and develop strategies for their prevention and treatment, contributing to improved overall health status for these patients and promoting the development of personalized treatment approaches.

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TO THE EDITOR

We read the retrospective cohort study published by Zhou *et al*[1] in the *World Journal of Psychiatry* in 2024. This study investigated the incidence and risk factors of depression among patients with metabolic syndrome (MetS). Subsequently, data from the 2-year, 4-year, and 7-year follow-up were analyzed to construct a predictive model for depression in patients with MetS. According to currently available studies, there is a strong relationship between depression and MetS. Zhou *et al*[1] described the causes and potential mechanisms associated with MetS and depression, and we believe that these results provide some insight into the causality of MetS and depression. However, the causality and direction of the two remains unclear due to the shortcomings of current observational study designs and the low feasibility of implementing randomized controlled trials[2,3].

Studies have shown that patients with depression were more likely to become obese or suffer from MetS due to lifestyle changes, sleep disorders, chronic inflammation, and the side effects of psychotropic medications[4,5]. Similarly, inflammation, imbalances in the neuroendocrine system, increased societal stress, poor lifestyle, and long-term medication render patients with MetS more susceptible to depression[6,7]. This suggests that the two conditions can exacerbate the risks of the other and lead to a series of severe coexisting diseases. One notable comorbidity that must be mentioned is obstructive sleep apnea (OSA)[8]. OSA is a common sleep disorder characterized by repeated episodes of upper airway collapse or obstruction, resulting in reduced or complete cessation of airflow during sleep[9]. OSA significantly affects sleep quality and physical health, leading to physical impairments and socioeconomic burdens. Long-term OSA increases the risk of heart disease, high blood pressure, and stroke[10,11].

Many studies have reported a strong association between depression and OSA, but most have focused on investigating the prevalence of depression among patients with OSA[12-16]. Using mendelian randomization analyses, our previously published study provided preliminary evidence that depression increases susceptibility to OSA[17]. Analyzing changes in nocturnal plasma levels of free fatty acids, glucose, and cortisol in patients with OSA, Chopra *et al*[18] found that patients with OSA and depression share increased sensitivity to corticotropin-releasing hormone, glucocorticoid resistance, and elevated cortisol levels due to similar hyperactivity of the hypothalamic-pituitary-adrenal axis and sympathetic nervous system. Furthermore, both depression and OSA have been shown to be associated with chronic inflammation and are characterized by higher levels of proinflammatory cytokines such as tumor necrosis factor-alpha and interleukin-6[19,20].

Overall, the pathophysiological mechanisms of how depression contributes to OSA are not fully understood, but they may be related to several potential mechanisms as follows. Lifestyle and circadian rhythm changes could cause disruptions in sleep-wake rhythms, affecting the physiological balance of sleep and wake functions[21-23]. Depression is typically accompanied by hyperactivation of the hypothalamic-pituitary-adrenal axis; as adrenocorticotropic hormone secretion increases, patients may experience a decrease in slow-wave sleep duration and an increase in the average number of awakenings[24]. Moreover, impairment of the central and peripheral 5-hydroxytryptamine systems could affect airway patency. Reduced 5-hydroxytryptamine levels are thought to be one of the critical mechanisms leading to upper airway collapse and potentially to OSA[25].

As the prevalence of depression increases, it becomes critical to prevent and manage its complications or comorbidities such as OSA. Depression and OSA often coexist and exhibit similar symptoms, such as poor concentration, memory loss, mood swings, and unusual fatigue, which can complicate the clinical diagnosis of both conditions. Zhou *et al*[1] investigated the prevalence and risk factors of depression in patients with MetS and constructed predictive models through a large-sample cohort study. We believe this has significant implications, but there is no such model to predict the occurrence of OSA in depressed patients. For depressed patients, non-invasive electroencephalogram to detect susceptibility to OSA is a powerful tool[26]. Exploring the comorbidity and correlation between OSA and depression and understanding the prevalence in different populations could help identify high-risk groups and facilitate early intervention.

The treatment of OSA comorbid with depressive disorders involves various therapies for depression, including pharmacological and non-pharmacological interventions such as light therapy, sleep deprivation, and rhythm therapy. They have been shown to affect circadian rhythms. Oxidative stress and inflammation can also disrupt sleep architecture, contributing to dysfunctional sleep patterns[27]. As the first-line treatment for OSA, studies have shown that continuous positive airway pressure therapy could improve depressive symptoms in patients over the long term, despite the presence of high heterogeneity. As more and more promising technologies are applied to depression and OSA, there will be opportunities to enhance our understanding of them and other comorbidities. The potential mechanisms may provide new ideas and strategies for the prevention and treatment of OSA and depression and help improve the overall health of such patients. However, current evidence largely depends on cross-sectional clinical data and a limited number of experimental results, which challenges the assertion that depression explicitly causes OSA.

FOOTNOTES

Author contributions: Wang X and Yue HM designed the study; Song SM searched the literature; Wang X and Yue HM wrote the manuscript; All authors approved the final manuscript.

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