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Obstructive Sleep Apnea is Linked to Depression and Cognitive Impairment: Evidence and Potential Mechanisms

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Abstract

Obstructive sleep apnea (OSA) is highly prevalent but very frequently undiagnosed. OSA is an independent risk factor for depression and cognitive impairment/dementia. Herein the authors review studies in the literature pertinent to the effects of OSA on the cerebral microvascular and neurovascular systems and present a model to describe the key pathophysiologic mechanisms that may underlie the associations, including hypoperfusion, endothelial dysfunction, and neuroinflammation. Intermittent hypoxia plays a critical role in initiating and amplifying these pathologic processes. Hypoperfusion and impaired cerebral vasomotor reactivity lead to the development or progression of cerebral small vessel disease (C-SVD). Hypoxemia exacerbates these processes, resulting in white matter lesions, white matter integrity abnormalities, and gray matter loss. Blood-brain barrier (BBB) hyperpermeability and neuroinflammation lead to altered synaptic plasticity, neuronal damage, and worsening C-SVD. Thus, OSA may initiate or amplify the pathologic processes of C-SVD and BBB dysfunction, resulting in the development or exacerbation of depressive symptoms and cognitive deficits. Given the evidence that adequate treatment of OSA with continuous positive airway pressure improves depression and neurocognitive functions, it is important to identify OSA when assessing patients with depression or cognitive impairment. Whether treatment of OSA changes the deteriorating trajectory of elderly patients with already-diagnosed vascular depression and cognitive impairment/dementia remains to be determined in randomized controlled trials.

Keywords: Obstructive sleep apnea; cerebral small vessel disease; cognitive impairment; depression; intermittent hypoxemia.

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Figures

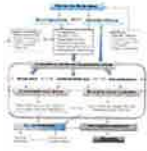


FIGURE 1 A model to describe the...

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