

LETTERS TO THE EDITOR

PAP use in mild cognitive impairment to delay progression to dementia

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We read with interest the paper from Skiba et al. 1 about the effect of obstructive sleep apnea (OSA) treatment by continuous positive airway pressure (CPAP) in patients with mild cognitive impairment. Treating OSA in patients at risk of Alzheimer disease (AD) is a hot research topic, considering that OSA can represent a risk factor for AD, accelerate cognitive decline, and contribute to alter AD pathology biomarkers (eg, clinical, biofluid, neuroimaging).^{2–4}

In this article, the authors conclude that CPAP treatment was not associated with delay in progression to dementia or cognitive decline in patients with mild cognitive impairment. This negative result contrasts with most of the previous literature suggesting that CPAP treatment may be effective in preserving cognitive abilities or slowing cognitive decline and improving AD biomarkers in OSA patients with mild cognitive impairment or AD.^{3–5}

The article has some strengths, but it also has several important limitations. First, the criteria for OSA diagnosis and treatment and oxygen saturation parameters are not included; diagnostic instruments for OSA definition are heterogeneous. Moreover, the authors considered different treatments of OSA: CPAP and bilevel PAP were used without explaining the clinical reasons for choosing each treatment and how many patients underwent them. Second, the article does not report data regarding the residual apnea-hypopnea index under treatment, and compliance was defined for only the last 30 days and does not include the total period of treatment. Third, although the long follow-up and the real-life design are interesting points, the retrospective design, use of medical charts, and limited number of patients reduce the significance of the results. Finally, comorbidities are not well defined or discussed, and progression to dementia (AD or other type of dementia) is not well characterized.

In our opinion, the results of the study cannot support the negative conclusions proposed by the authors but rather emphasize the need for more specific studies, including a larger number of patients with mild cognitive impairment and well-defined criteria for OSA diagnosis and related homogeneous treatment. Moreover, the study design should preferably be prospective with a clear definition of the dementing disorder developed during the follow-up (AD vs other dementias), with a regular time frame to evaluate CPAP compliance and efficacy in relation to cognitive decline, investigating the role of major comorbidities and possibly using multimodal AD biomarkers according to classifications proposed since 2013.⁶

CITATION

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