Sleep Apnea and the Risk of Stroke in the Elderly

To the Editor:

Obstructive sleep apnea (OSA) is emerging as an important risk factor for stroke. Recent prospective studies, focusing on middle-aged adults with OSA, showed an increased risk of stroke after a follow-up that varied from 4 to 10 years. In addition, Marin et al found that continuous positive airway pressure therapy promotes significant reduction on the composite risk of fatal and nonfatal cardiovascular events, including stroke. Despite these convincing data, the majority of strokes does not occur in this age rate, limiting the external validity of the relationship between OSA and stroke for more advanced age. In addition, the impact of OSA in patients >60 years have been questioned in a recent study from Sleep Health Study, because no association was found between OSA and systolic/diastolic hypertension. In order to explore the impact of OSA in elderly patients, Munoz et al performed an important population-based study designed to investigate the risk of stroke in the noninstitutionalized elderly people (70 to 100 years old) that were submitted to polysomnography at baseline. In a follow-up of 6 years, these authors found that severe OSA was associated with an increased risk of stroke (hazard ratio of 2.52 after adjustments for confoundable factors). Therefore, this study no longer supports the concept that OSA is a “benign” syndrome among elderly patients. The relationship between OSA and stroke is particularly relevant because stroke is a leading cause of death.

The authors concluded that randomized trial designed to investigate the influence of continuous positive airway pressure therapy on stroke is required to complete the demonstration of a causative relationship. However, in order to establish definitive causative relationship between OSA and stroke we would like to add an important postulate: it is necessary to include evidence for a biological plausibility involving the independent association between OSA and stroke. In this context, we have recently described early signs of atherosclerosis in middle-aged, apparently healthy adults, independent of other traditional risk factors. Severe OSA patients presented increased arterial stiffness, intima-media thickness and carotid diameter compared with appropriate controls. In addition, the vascular impairment was directly related to OSA severity. Although no previous studies prospectively evaluate the progression of atherosclerosis in OSA, it is reasonable to think that the acceleration of the atherosclerosis process could be involved in nontreated elderly OSA patients. It is possible that the exposure to repetitive apneas during the night for many years could perpetuate important mechanisms associated with OSA, including inflammation, endothelial dysfunction and increased production of reactive oxygen species. In this sense, a detailed evaluation of the extension of the atherosclerosis process in the elderly, comparing subjects with and without OSA, could provide important insights in order to answer this question. OSA is also associated with profound oscillations of blood pressure during the night, which could have direct impact on the impaired cerebral blood flow, and could consequently increase the risk of stroke. This important point should be addressed in further studies. In summary, the study by Munoz et al adds significant improvements in our current knowledge about OSA and is in line with several studies that point to a strong biological plausibility linking OSA and stroke.

Disclosures

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