
To the Editor:

We read with great interest the recent study by Chan et al1 regarding a high prevalence of sleep apnea among patients with TIA and minor stroke. The authors demonstrated the persistence of obstructive sleep apnea at 3 months after ictus and therefore assumed that it predated the onset of stroke contributing to its pathogenesis. Moreover, patients with obstructive sleep apnea were significantly more exposed to nocturnal hypoxia.

Our group is also studying sleep apnea in patients with acute stroke using the oxygen desaturation index (ODI)—number of desaturations /4% per hour—as a reliable index of estimating the oxygenation and came to conflicting results. ODI seems to be a sensitive index for monitoring the oxygenation during sleep and is used as a simple test to predict the presence of sleep apnea. Using pulse oximetry within 24 hours after ictus in a total of 53 patients with TIA or minor stroke and no previous symptoms of apnea, we found 7 cases (13.2%) of normal (ODI /5), and 46 (86.79%) cases of disturbed oxygenation (ODI /5). Mild, moderate, and severe apnea was documented in 23.91%, 26.09%, and 50% of patients with disturbed oxygenation, respectively. The difference between the sensitivity of Respiratory Disturbance Index and ODI and the various time points of the examinations' accomplishment may explain the conflicting results of both studies. Moreover, we did not find any linear correlation between NIHSS and ODI; this could indicate a closer relationship of apneas with the location of the stroke, rather than with the severity of the symptoms, as they are expressed through NIHSS scores. Nevertheless, the higher the ODI, the more severe the exposure of patients to hypoxia, especially when hypoxia is <90%, which is a common finding.

It would be interesting to know whether the authors examined a possible relation between the exact location4 of the ischemic infarction and the documented reduction of oxyhemoglobin saturation or the persistence of obstructive sleep apnea at 3 months. It also would be interesting to know the exact time point when overnight cardiopulmonary monitoring was performed. Differences between the acute stroke phase and the chronic phase after stroke when the patient returns home could be present, enhancing the debate whether, in some cases, sleep apnea is the cause or the result of cerebral ischemia. There is no doubt that further studies are necessary to evaluate the role of obstructive sleep apnea in stroke survivors.

Disclosures

None.

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Letter by Naoumis et al Regarding Article, "Sleep Apnea in Patients With Transient Ischemic Attack and Minor Stroke: Opportunity for Risk Reduction of Recurrent Stroke?"

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