NAMS Regional Symposium on Sleep Medicine
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OBSTRUCTIVE SLEEP APNOEA (OSA) AND STROKE

SYNOPSIS

Evidence is growing that not only is obstructive sleep apnopea (OSA) an independent risk factor for stroke, but that treating OSA improves recovery from stroke. However, unlike aggressive treatment of conventional risk factors for stroke, such as hypertension, atrial fibrillation, hypercholesterolemia and carotid stenosis, OSA is overlooked. Without understanding the link between OSA and stroke, whether the relationship is just an association or actually causal, and the pathophysiological mechanisms connecting the two, an important opportunity of intervening with evidence-based, well directed stroke preventive and therapeutic option is being missed. OSA has often been documented in cohorts recovering from recent stroke, which begs the questions of whether stroke leads to OSA or if OSA causes stroke, or if the relationship between OSA and stroke is bidirectional.

In a study unattended overnight PSG (Polysomnography) was performed at home in a cohort of 6424 individuals. Significantly, the result showed that not only was there a positive association between the severity of OSA, as measured by the Apnoea-Hypopnoea Index (AHI), and the risk developing cardiovascular events, including stroke, but that even mild to moderate OSA was detrimental-independent of other known risk factors.

The initial suspicion of a link between Sleep Disordered Breathing (SDB) and stroke was raised through epidemiological studies first conducted in 1980-1990s. Strength of association between stroke and snoring equaled other traditional stroke risk factors and held true independently, even when adjusted for confounding risk factors such as hypertension, smoking, atrial fibrillation and hypercholesterolemia. But temporal relationship between snoring and stroke has not been established because snoring, similar to OSA, can well be a consequence of stroke rather than causing it.

The reported frequency of OSA in stroke patients varies between 30% and 80%. In recent meta-analysis of ischemic or hemorrhagic stroke and transient ischemic attack (TIA) patients, the frequency of SDB with AHI of >5 was 72 %, and with an AHI of >20 was 38%, it was reconfirmed by a previously reported higher prevalence of SDB (AHI>10) in men compared with women (65% vs. 48%; p-0.001), and also a higher percentage of SDB (AHI>10) in patients with recurrent stroke than initial stroke (74% vs. 57%; p-0.013). Patients with cardio embolic strokes had a lower percentage of SDB compared with patients with strokes due to unknown etiologies.

Various studies also support causal association between hypertension and OSA. Sleep heart health study demonstrated that sustained diurnal hypertension is a consequence of chronic OSA. The relative risk of hypertension in severe OSA (AHI> 30) compared with the mildest category (AHI < 1.5) in this study was 1.37 (95%, CI: 1.03-1.83), and the odds for hypertension increased with AHI in a dose-response manner. Treatment of OSA with CPAP (Continuous Positive Airway Pressure), compared to sham CPAP, reduced the severity of hypertension, which further supports an association between OSA and hypertension. OSA may raise the systemic blood pressure significantly and thus can be one of the most important mechanism by which OSA leads to cerebrovascular morbidity.

Mechanism of interaction between OSA and normal blood pressure leading to hypertension has been studied and it is observed that patients with OSA have considerably higher sympathetic activity compared with controls, even during wakefulness. A modification caused in renal physiology in the form of an augmentation of the rennin-angiotensin system in face of chronic OSA-induced hypoxia has also been postulated as an explanation for the genesis of hypertension in OSA. OSA is also been implicated in contributing to drug resistant hypertension.

In conclusion, OSA is a modifiable risk factor for stroke. Whether every patient with stroke should be screened for OSA and whether treatment for OSA should commence during the acute stroke or stroke rehabilitation, or as a primary or secondary prevention tool, is presently unclear. Given that OSA is associated consistently with an increased risk of stroke, and recovery from

stroke is worse in patients with OSA, there seems to be a compelling case to conduct randomized trials of CPAP therapy for prevention of major cardiovascular events including stroke, in patients with OSA.

SUGGESTED READING

1. Partinen M, palomaki H (1985). Snoring and cerebral infarction. *Lancet* **2**:1325-6.
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3. Cherkassky T, oksenberg A, Froom P, *et al* (2003). Sleep- related breathing disorder and rehabilitation outcome of stroke patients. A prospective study. *Am J Phys Med Rehabil* **82**:452-5.
4. Johnson KG, Jhonson DC (2010). Frequency of sleep apnea in stroke and TIA patients; A meta-analysis. *J Clin Sleep Med* **6**:131-7.