

Obstructive Sleep Apnea, Acute Stroke, and Intermittent Positive Airway Pressure

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DEAR EDITOR,

Almost 90% of acute stroke patients had sleep-disordered breathing, and one-third had severe obstructive sleep apnea (OSA) with markedly increased inflammatory biomarkers, thereby providing a possible pathophysiological correlation between OSA and stroke [1].

OSA leads to repeated episodes of hypoxia, which raises the negative intrathoracic pressure and changes the shape of the atrium, which can lead to arrhythmias like atrial fibrillation. Hypoxia causes oxidative stress, which in turn activates the sympathetic nervous system and the renin-aldosterone-angiotensin system. This leads to blood pressure spikes and fluctuations. OSA induces inflammation, increases fibrinogen levels, and increases platelet activity, creating a state of hypercoagulability and precipitating further stroke. The hypertension, chronic bouts of hypoxia, and hypercapnia also remodel intracranial and extracranial vasculature, thereby impairing cerebral autoregulation. It can also cause diversion of blood from the ischemic penumbra to the non-ischemic part of the brain (reverse Robin Hood phenomenon) [2].

Thus, OSA, which is also sometimes precipitated/aggravated by stroke itself, creates a detrimental situation in both types of strokes (ischemic and hemorrhagic), making acute stroke management challenging, and if not handled successfully, may significantly influence the outcome. In fact, sleep-related breathing disturbances exacerbated by a stroke recover in the subacute stroke phase; hence, a polysomnography

done during the acute stage may not reflect the preexisting OSA, nor can it foretell the possibility of its existence in the future. The sleep study may also be artifactual when done in an acute setting in an intensive care unit; hence, rather than selecting patients who may have OSA or doing polysomnography immediately post-stroke, we can administer CPAP/BIPAP at least for four hours during sleep.

CPAP can cause a reduction in all-cause mortality in post-stroke or TIA patients who have sleep-disordered breathing [3]. Studies have revealed that, other than insignificant mask-related issues, these methods of assisted positive airway pressure are well tolerated and may be outcome-changing.

So that we have a clear guideline for this measure, we need more studies like "Reverse-STEAL," in which early non-invasive ventilation with auto-titrating bilevel positive airway pressure (auto-BPAP) was tried within 24 hours of a stroke during both daytime and nighttime sleep [4].

Till then, a more liberal use of the appliances will not only reduce stroke mortality but also improve stroke outcomes. As these appliances ensure better sleep and longer arousal, they will help patients participate more in rehabilitative measures, improve mobility, and thereby avoid post-stroke complications like aspiration pneumonia and pressure sores.

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