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FIELD OF VISION

Sleep-disordered breathing and stroke: A relation to be considered

Ridvan Alimehmeti, Arba Cecia, Arsen Seferi, Ermir Roci

Ridvan Alimehmeti, Arsen Seferi, Ermir Roci, Department of Neurology, Neurosurgery and Psychiatry, University Hospital Center "Mother Theresa", 1001 Tirana, Albania

Arba Cecia, George Mason University, Virginia, VA 22030, United States

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Correspondence to: Ridvan Alimehmeti, MD, PhD, Associate Professor, Department of Neurology, Neurosurgery and Psychiatry, University Hospital Center "Mother Theresa", Rr. Kongresi Manastirit, 270, 1001 Tirana, Albania. ridvanalimehmeti@hotmail.com
Telephone: +355-42-362641

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Abstract

Stroke is a leading cause of death and disability. Despite expensive and elaborative research in finding out mechanisms of interrelation between sleep-disordered breathing (SDB) and stroke, there is yet much attention to be given in stroke units worldwide to the prompt diagnosis and treatment of SDB in order to improve morbidity and mortality rates related with stroke. The preventive diagnosis and treatment of SDB reduce stroke rate and improves penumbra area in case of ischemic stroke. Stroke itself predispose to SDB, making the interrelationship more complicated. The review by Parra O and Arboix A reflects the results from carefully selected reviews reported in the literature so far. This review of the literature and presentation of the original study of the Authors based on their patients' data, enhances the conviction that there exists a direct relation between SDB and stroke. Diagnosis of SDB in new stroke cases should be sought and treated carefully whenever present.

Key words: Sleep-disordered; Stroke; Hypertension; Penumbra; Breathing

Core tip: It is of paramount importance to search for a precocious diagnosis of sleep disordered breath (SDB) in newly diagnosed stroke patients in order to establish a prompt treatment. Treatment of SDB in newly diagnosed stroke patients prevent subsequent stroke episodes and lower the rate of morbidity and mortality. Penumbra zone benefits of a better recovery in case of prompt treatment of SDB in stroke. The review of the literature and presentation of the original study of Parra O and Arboix A enhances the conviction that there exist a direct relation between SDB and stroke.

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INVITED COMMENTARY

It is generally accepted that there exist an increased risk of stroke in persons that are diagnosed with sleep disordered breath (SDB). In the other hand SDB is revealed in most of patients with stroke[1,2]. Strokes can themselves generate SDB[3]. The presence of SDB is related to worst neurologic outcome after stroke. In the literature is reported that in hypertonic and diabetic patients with SDB timely recognition and treatment of SDB would prevent onset of stroke or reduce the severity of neurological deficit in case of stroke[1,2]. Sleep apnea is the most frequent SDB found in 5%-15% of the population[4]. Other authors report as higher incidence as half of the stroke patients[5]. In such case breath pattern is marked by episodes of partial or complete block of the upper airway. During the day there is sleepiness, snoring, and apneas[4].

Xie *et al*[6] conducted a thorough searched of the literature for studies on associations between SDB and the risk of stroke. They proclaimed that SDB may be a significant predictor of serious adverse outcomes following stroke and concluded that a large-scale, multicenter randomized controlled trial would confirm if better treatment of SDB would be the cause for fewer recurrent vascular events.

Despite many predisposing risk factors for SDB, such as male gender, endocrine disorders, use of muscle relaxants, smoking, fluid retention and ageing, the strongest risk factor is obesity[7]. Sleep disordered breathing is proven to be related with cardiovascular disease, cognitive impairment, and stroke.

The putative neuromechanisms behind some of the effects of SDB on the central nervous system is related with the nocturnal intermittent hypoxia and sleep fragmentation[8].

SDB increases the risk of stroke and hypertension through intermittent hypoxia with release of cytokines, angiogenic inhibitors, free radicals, and adhesion molecules. Hypertension occurs during apneas with abrupt surge in the end of apnea. Hypertension is recorded in daytime in two thirds of patients with SDB. The studies by Mohsenin *et al*[9] and Hoffmann *et al*[10] stress the important influence of SDB in hypertension and the positive effect of SDB treatment on better control of hypertension.

The sympathetic activity is revealed to be important during rapid eye movement in protecting the brain from high perfusion pressure related with hypertension. SDB is marked by prolonged sympathetic overactivity. Sympathetic system seems to be involved in pathogenesis of abnormal hemodynamics and stroke in SDB patients[11].

Another hypothesized mechanism that relates SDB with stroke is reported the potential acceleration of the process of atherosclerosis. This has to do with the influence of SDB on hypertension and contribution to insulin resistance, diabetes and dyslipidemia. Furthermore, clinical and experimental data reveal direct proatherogenic effects of SDB such as, induction of systemic inflammation and endothelial dysfunction[12]. Numerous markers of these changes have been reported in SDB patients[13].

Snoring and SDB prior to transient ischemic attacks suggest that untreated SDB may lead to stroke. Cohort studies indicate that SDB is a risk factor for stroke[14].

The acute hemodynamic and autonomic perturbations that accompany obstructive apneas during sleep, with associated repeated arousals and intermittent hypoxemia, appear to result in sustained hypertension. In addition to the metabolic and humoral effects from obesity, SDB appears to predispose individuals to autonomic imbalance characterized by sympathetic overactivity and altered baroreflex mechanisms as well as alterations to vascular function[15].

SDB is a very common condition in patients with stroke and is found in over half of stroke patients. There is a complex relationship between SDB and stroke, attributable to shared risk factors. There are numerous mechanisms by which SDB may contribute to increased stroke risk, including promotion of atherosclerosis, hypercoagulability, and adverse effects on cerebral hemodynamics. Obstructive sleep apnea is also a risk factor for hypertension, and likely for atrial fibrillation and diabetes, conditions that in turn are risk factors for stroke. SDB is also associated with poor outcomes following stroke[16]. Other authors produce evidence of SDB modifying intrathoracic pressure and heart function giving rise to intermittent hypoxemia, which may lead to vascular endothelial dysfunction and increase sympathetic drive[4]. According to Kepplinger *et al*[17] in acute cerebral ischemia, the presence of SDB is related with clinically silent microvascular cerebral lesions that may contribute to a negative functional outcome. According to the clinical experience of the authors of this commentary there is a relation between sleep-disordered breathing and multilacunar cerebral infarction probably due to chronically-altered cerebral blood perfusion during sleep. Bonnin-Vilaplana *et al*[18] first analysed SDB in acute lacunar stroke. They reported a total of 69.1% of patients with apnoe/hypopnoe index (AHI) greater than or equal to 10; 44.1% with AHI 20 or greater and 2% of the patients with AHI 30.2 or greater. Cheyne-Stokes respiration is reported in 20.6% of cases with lacunar infarction[19]. Several studies report that continuous positive airway pressure treatment can reverse pathophysiological changes in SDB, increasing insulin sensitivity and reducing blood pressure[20]. SDB is very frequent in patients with transient ischemic attacks and stroke. Treatment with continuous positive airway pressure of stroke patients with SDB is believed to prevent cardiovascular accidents and may improve neurologic deficit[21]. Neurologist should diagnose and treat properly stroke patients with SDB[5].

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