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Editorial
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Heart and Obstructive Sleep Apnea: a Common Relationship

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Editorial

Obstructive Sleep Apnea (OSA) is a clinical condition characterized by partial (hypopneas) or completes (apneas) intermittent occlusions of the upper airway during sleep. These leads to intermittent hypoxia, sleep fragmentation, and promote abrupt reductions of intrathoracic pressure [1]. OSA is common not only in the general population (9% women and 17% men), but particularly in patients with Cardiovascular Disease (CVD) [2-5]. For instance, around 50% of patients with hypertension and 30% of patients with heart failure have some degree of OSA [3-5]. OSA is also highly prevalent among patients with chronic and stable Coronary Artery Disease (CAD) [6-8]. Growing evidence suggests that OSA is associated with increased risk of major cardiovascular events, including myocardial infarction and stroke [9,10]. In addition, observational studies have shown that the main treatment of OSA, Continuous Positive Airway Pressure (CPAP), can reduce non-fatal and fatal cardiovascular events in patients with severe forms of OSA [11]. However, randomized clinical trials did not show the favorable effect of CPAP on the CVD scenario [12-15]. The most likely explanation was the lower adherence rate, since in the subgroup analysis those who used CPAP more than 4 hours/night had cardiovascular improvement [12-15]. Therefore, there is still a need for clarification on the underlying mechanisms that link OSA and CVDs. The pivotal mechanisms linking OSA and CVDs are intermittent hypoxia, oxidative stress, inflammation, sympathetic activation and endothelial dysfunction [12]. OSA has been associated with elevated inflammation markers and the treatment of OSA with CPAP has shown reduced levels of inflammatory biomarkers [16-18]. As inflammation plays a major role in the atherosclerosis it seems reasonable that inflammation may be a pathway linking OSA and CVDs [19,20]. Recently, it has been reported that OSA is independently associated with higher serum levels of neutrophil and the sympathetic nervous system partially mediates this association [17]. Further research is therefore needed to establish the main pathways linking OSA and CVDs, which may lead to future targeted therapies. Immune markers may improve the risk stratification of OSA-CVD susceptibility. OSA is characterized by repetitive events of apneas and hypopneas during sleep, which generates intermittent hypoxia, exaggerated negative intrathoracic pressure, and arousals from sleep. As a result, this cascade of events may increase myocardial demand [1,2,12]. My colleagues and I showed that very severe OSA was independently associated with overnight myocardial injury in patients with refractory angina [7]. In addition, population-based studies have found that OSA correlated with low limit detection levels of cardiac troponin, characterizing a subclinical myocardial damage [21-23]. In the era of precision medicine, better understanding of the individual susceptibility of OSA among CVD settings may clarify mechanistic pathways targeting therapies.

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