Pathophysiology of developing atrial fibrillation in patients with obstructive sleep apnea.

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Introduction

Atrial fibrillation (AF) is a common cardiac arrhythmia characterized by the irregular and rapid contraction of the atria, which can lead to poor cardiac output and increase the risk of stroke, heart failure, and mortality. Obstructive sleep apnea (OSA), a prevalent sleep disorder, has been identified as a risk factor for AF. The pathophysiology of developing AF in patients with OSA is multifactorial, involving autonomic nervous system dysfunction, inflammation, oxidative stress, and structural changes in the heart [1].

OSA is characterized by recurrent episodes of partial or complete upper airway obstruction during sleep, leading to intermittent hypoxia, hypercapnia, and sleep fragmentation. These physiological disturbances activate the sympathetic nervous system and inhibit the parasympathetic nervous system, leading to an imbalance between sympathetic and parasympathetic tone. This autonomic dysfunction is a key factor in the pathogenesis of AF in patients with OSA. Studies have shown that patients with OSA have higher sympathetic nerve activity and lower parasympathetic activity compared to control subjects. The increased sympathetic tone leads to a higher heart rate, increased contractility, and vasoconstriction, while the decreased parasympathetic tone reduces the vagal brake on the heart, leading to increased atrial and ventricular automaticity. These effects can trigger ectopic beats, re-entry circuits, and AF [2].

In addition to autonomic dysfunction, inflammation and oxidative stress have been implicated in the pathogenesis of AF in patients with OSA. Intermittent hypoxia and reoxygenation during OSA episodes lead to the activation of inflammatory pathways and the production of reactive oxygen species (ROS), which can cause oxidative damage to cellular proteins, lipids, and DNA. These molecular changes can induce atrial remodeling, fibrosis, and electrical remodeling, leading to AF. Studies have shown that patients with OSA have higher levels of inflammatory markers, such as C-reactive protein (CRP), interleukin-6 (IL-6), and tumor necrosis factor-alpha (TNF-α), compared to control subjects. The increased inflammation can promote the formation of atrial fibrosis and electrical remodeling, which are associated with the development and maintenance of AF. Furthermore, OSA-induced oxidative stress can impair intracellular calcium handling, leading to increased intracellular calcium concentration and abnormal calcium signaling, which can trigger AF [3].

Structural changes in the heart, such as atrial enlargement and fibrosis, are also common in patients with OSA and have been linked to the pathogenesis of AF. Chronic intermittent hypoxia and hypercapnia during OSA episodes can induce atrial stretch and pressure overload, leading to atrial remodeling and dilation. Atrial fibrosis, characterized by the deposition of extracellular matrix proteins, is a hallmark of AF and is also associated with OSA. Studies have shown that patients with OSA have higher levels of collagen deposition in the atrial tissue compared to control subjects, which may contribute to the development of AF [4].

Moreover, OSA-related changes in the autonomic nervous system, inflammation, and oxidative stress can further exacerbate atrial structural remodeling and fibrosis. The activation of sympathetic nerve activity can promote fibroblast proliferation and collagen synthesis, while inflammation can stimulate the secretion of profibrotic cytokines, such as transforming growth factor-beta (TGF-β) and connective tissue growth factor (CTGF). These factors can contribute to the accumulation of extracellular matrix proteins and the development of atrial fibrosis, which can increase the risk of AF [5].

Conclusion

The pathophysiology of developing AF in patients with OSA is complex and involves multiple mechanisms, including autonomic dysfunction, inflammation, oxidative stress, and structural changes in the heart. These factors interact with each other and create a vicious cycle that promotes the development and maintenance of AF. The understanding of these mechanisms has important implications for the management of AF in patients with OSA. Treatment of OSA with continuous positive airway pressure (CPAP) therapy has been shown to improve autonomic dysfunction, reduce inflammation, and attenuate oxidative stress, which can potentially decrease the risk of AF. In addition, CPAP therapy can also prevent or reverse atrial structural remodeling and fibrosis, which may reduce the risk of AF.

References

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