Brief Report
Volume 09:01, 2025

Journal of Coronary Heart Diseases

ISSN: 2684-6020 Open Access

Association between Sleep Apnea and Incidence of Coronary Heart Disease

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Introduction

Sleep apnea, a common and potentially serious sleep disorder characterized by repeated episodes of upper airway obstruction during sleep, has garnered increasing attention in cardiovascular research due to its systemic health consequences. Obstructive Sleep Apnea (OSA), the most prevalent form, leads to intermittent hypoxia, sleep fragmentation and autonomic dysregulation, all of which exert stress on the cardiovascular system. Over the past two decades, accumulating epidemiological and clinical evidence has suggested a significant association between sleep apnea and the development of Coronary Heart Disease (CHD). Given the global prevalence of both conditions, understanding this relationship is critical for early risk assessment, preventive cardiology and integrated management strategies. This paper explores the pathophysiological mechanisms linking sleep apnea to CHD, reviews key clinical findings and discusses the implications for diagnosis and treatment [1].

Description

The underlying pathophysiology connecting sleep apnea with coronary heart disease primarily centers around the effects of intermittent hypoxia and sympathetic overactivation. In patients with OSA, repeated episodes of oxygen desaturation trigger a cascade of oxidative stress, systemic inflammation and endothelial dysfunction. These factors promote the development of atherosclerosis, a hallmark of CHD. Additionally, frequent arousals from sleep and consequent sympathetic nervous system activation lead to elevated blood pressure, heart rate variability and increased myocardial oxygen demand. The resultant cardiovascular strain, especially during the night when parasympathetic dominance is expected, contributes to structural and functional cardiac abnormalities over time. Several large-scale epidemiological studies have substantiated the link between sleep apnea and increased risk of CHD. For example, the Sleep Heart Health Study (SHHS), a multicenter cohort study involving over 6,000 participants, found that individuals with moderate-to-severe OSA had a significantly higher risk of incident coronary heart disease, including myocardial infarction and coronary revascularization procedures. Similarly, the Wisconsin Sleep Cohort Study reported a dose-response relationship, with increasing severity of sleep apnea associated with greater CHD incidence. These findings remained robust even after controlling for confounders such as obesity, hypertension, diabetes and smoking, underscoring the independent contribution of sleep apnea to coronary risk.

Polysomnography, the gold standard diagnostic tool for sleep apnea, reveals key indicators such as Apnea-Hypopnea Index (AHI), oxygen desaturation index and arousal frequency, which correlate with cardiovascular outcomes. Patients with high AHI scores are more likely to exhibit subclinical coronary artery disease on imaging studies, including Coronary Artery Calcium (CAC) scoring and CT angiography. Moreover, nocturnal hypoxia, particularly when oxygen saturation drops below 90% for extended durations, has been associated with endothelial injury and accelerated plaque formation. These mechanistic links support the clinical observation that untreated sleep apnea contributes to the progression of coronary artery disease. Therapeutic interventions for sleep apnea may have significant cardiovascular benefits, especially in reducing CHD risk. Continuous Positive Airway Pressure (CPAP) therapy, the most effective treatment for OSA, prevents upper airway collapse, restores normal oxygenation and mitigates sympathetic overdrive. Several randomized controlled trials and meta-analyses suggest that adherence to CPAP therapy improves blood pressure control, reduces arrhythmias and slows the progression of atherosclerosis. However, evidence regarding its impact on hard CHD outcomes remains mixed, largely due to issues of therapy adherence and study duration. Nonetheless, observational studies consistently demonstrate lower incidence of cardiovascular events among compliant CPAP users compared to untreated individuals [2].

Conclusion

The association between sleep apnea and the incidence of coronary heart disease is well-established through both mechanistic and epidemiological evidence. Intermittent hypoxia, systemic inflammation and sympathetic hyperactivity create a pro-atherogenic and cardiotoxic environment that predisposes individuals to CHD. Early diagnosis and effective management of sleep apnea most notably through CPAP therapy offer promising avenues for mitigating cardiovascular risk. Given the high prevalence and underrecognition of sleep apnea, particularly in populations already burdened with cardiovascular risk factors, proactive screening and integrated treatment approaches are warranted. Recognizing sleep apnea as a modifiable risk factor could play a pivotal role in preventing coronary events and improving long-term cardiovascular outcomes. .

Acknowledgement

None.

Conflict of Interest

None.

References

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Received: 01 February, 2025, Manuscript No. jchd-25-169038; **Editor Assigned:** 05 February, 2025, Pre QC No. P-169038; **Reviewed:** 17 February, 2025, QC No. Q-169038; **Revised:** 22 February, 2025, Manuscript No. R-169038; **Published:** 28 February, 2025, DOI: 10.37421/2684-6020.2025.9.217

How to cite this article: Jenkus, Victoria. "Association between Sleep Apnea and Incidence of Coronary Heart Disease." *J Coron Heart Dis* 09 (2025): 217..