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Chronic Nasal Obstruction: A Neglected but Important Contributor to Ischemic Heart Disease

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ABSTRACT

Chronic nasal obstruction is commonly viewed as a localized upper airway condition, often under-recognized in systemic disease contexts. However, growing evidence suggests that chronic nasal obstruction may contribute significantly to the pathogenesis of ischemic heart disease (IHD). Mechanisms include hypoxia-induced sympathetic activation, increased systemic inflammation, and sleep-disordered breathing. This short communication reviews the plausible links between nasal obstruction and cardiac ischemia and highlights the need for interdisciplinary management approaches.

Key words: Chronic nasal obstruction; Ischemic heart disease; Adenoid; Septoplasty

INTRODUCTION

Chronic nasal obstruction, whether due to deviated nasal septum, enlarged adenoids, turbinate hypertrophy, chronic rhino-sinusitis, or allergic rhinitis, significantly affects the quality of life by impairing nasal airflow. While traditionally managed as a localized ENT issue, its systemic consequences are increasingly being understood. Notably, its association with cardiovascular morbidity, particularly ischemic heart disease (IHD), is gaining attention. This short communication aims to highlight the physiological and clinical basis of this association.

Pathophysiological Mechanisms Linking Nasal Obstruction and IHD

1. Hypoxia and Sympathetic Overdrive: Chronic nasal obstruction often leads to persistent mouth breathing, especially during sleep, causing intermittent hypoxia. Hypoxia stimulates chemoreceptors, resulting in increased sympathetic activity, vasoconstriction, and elevated blood pressure—key contributors to IHD [1,2].

2. Sleep-Disordered Breathing (SDB): Nasal obstruction is a well-established risk factor for obstructive sleep apnea (OSA), a known independent risk factor for cardiovascular disease including IHD. OSA is characterized by repetitive episodes of apnea and hypopnea, which increase cardiac workload and lead to myocardial ischemia [3-5].
3. Inflammatory Cascade: Chronic nasal inflammation is associated with systemic release of cytokines like IL-6, CRP, and TNF-α. Chronic inflammation is central to atherosclerosis, plaque instability, and coronary events [6-8].
4. Oxidative Stress and Endothelial Dysfunction: Hypoxia and inflammation enhance oxidative stress and endothelial damage, leading to reduced nitric oxide availability, impaired vasodilation, and increased arterial stiffness-hallmarks of IHD [9-10].

CLINICAL AND EPIDEMIOLOGICAL EVIDENCE

Several studies have found higher rates of cardiovascular events in individuals with sleep-disordered breathing linked to nasal obstruction. In particular, patients with chronic rhino-sinusitis and nasal septal deviation demonstrate greater carotid intima-media thickness and arterial stiffness [11,12]. A Korean national study also showed increased prevalence of hypertension and IHD in patients with OSA secondary to nasal obstruction [13]. Additionally, treatment of nasal obstruction (e.g., via septoplasty, turbinate reduction or adenoidectomy) has been shown to improve blood pressure control and cardiovascular parameters [14,15].

MANAGEMENT IMPLICATIONS

ENT surgeons and cardiologists should collaborate in identifying and managing patients with nasal obstruction who are at risk of cardiovascular disease. Timely correction of anatomical obstructions (e.g. adenoidectomy, septoplasty, turbinoplasty), and control of allergic or inflammatory conditions may help reduce systemic inflammatory burden and improve sleep quality-both of which are beneficial for cardiac health [14,15].

CONCLUSION

Chronic nasal obstruction is more than just a local breathing problem-it may serve as a modifiable risk factor for ischemic heart disease. Through mechanisms involving hypoxia, sympathetic overdrive, systemic inflammation, and endothelial dysfunction, it contributes to the pathophysiology of IHD. Increased awareness among clinicians and incorporation of nasal airway assessment in cardiovascular risk profiling may offer new avenues for prevention and intervention.

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