Asthma, osa, ph: Interconnected inflammatory comorbidities.

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Introduction

Pulmonary Hypertension (PH) represents a significant and often underrecognized comorbidity in severe asthma. Research indicates PH impacts a substantial portion of these patients, especially those presenting with pronounced eosinophilic inflammation and a higher burden of asthma symptoms. The presence of PH significantly increases morbidity and mortality in this subgroup, highlighting an urgent need for early screening and targeted management strategies to improve patient outcomes [1].

A mechanistic link connects chronic airway inflammation, a hallmark of asthma, to potential effects on pulmonary vasculature, leading to vascular remodeling and PH. Severe asthma, particularly with persistent inflammation, can instigate pulmonary circulatory changes independently of hypoxia, underscoring the interconnectedness of airway and vascular pathologies [6]. Comprehensive reviews detail the critical role inflammatory signaling pathways play in PH pathogenesis and progression. Here, immune cells and their mediators contribute to vascular remodeling, endothelial dysfunction, and elevated pulmonary arterial pressure, suggesting inflammation as a pivotal therapeutic target [4]. In line with this, studies on Pulmonary Arterial Hypertension (PAH) focus on inflammatory and oxidative stress biomarkers. These molecular markers reflect disease activity, prognosis, and treatment response, reinforcing inflammation's central role in PAH pathogenesis for diagnostic and therapeutic targeting [9].

The intricate relationship between sleep-disordered breathing (SDB), specifically Obstructive Sleep Apnea (OSA), and PH is complex. Pathophysiological mechanisms, including intermittent hypoxia, hypercapnia, and systemic inflammation, link SDB directly to PH development and progression. Recognizing and treating SDB in PH patients is paramount [2]. OSA also detrimentally affects asthma control and heightens airway inflammation. Patients with comorbid OSA and asthma typically experience worse asthma symptoms, reduced lung function, and higher inflammatory markers. This indicates that effectively managing OSA is crucial for optimizing asthma treatment outcomes [3]. Understanding the pathophysiological connections between asthma and OSA is vital, involving shared risk factors, common inflammatory pathways, and the direct impact of nocturnal OSA events on asthma severity. This highlights how treating one condition can significantly improve outcomes for the other, underscoring the need for integrated care approaches [10].

The co-occurrence of OSA and PH in asthmatic patients has been investigated, revealing a significant association between OSA severity and PH presence. This suggests OSA may exacerbate or contribute to PH development in individuals with asthma, warranting careful screening and management of both conditions [5]. Further understanding these interactions, research examined OSA's impact on eosinophilic airway inflammation in asthma. Findings showed OSA significantly exacerbates eosinophilic inflammation in asthmatic patients, potentially contributing to poorer asthma control and increased disease severity. This insight suggests that treating OSA could yield substantial benefits in managing asthma's inflammatory phenotype [7]. Ultimately, a growing body of work discusses the significant overlap and complex interactions among asthma, OSA, and PH. These conditions mutually influence each other's severity and progression through shared pathophysiological mechanisms like inflammation and intermittent hypoxia, making a holistic approach to diagnosis and ongoing management essential [8].

Conclusion

Pulmonary Hypertension (PH) is a notable comorbidity in severe asthma, impacting a significant portion of patients, especially those with eosinophilic inflammation and higher symptom burdens. This condition contributes to increased morbidity and mortality, suggesting a strong need for early screening and targeted management strategies. The complex relationship between sleep-disordered breathing (SDB), particularly Obstructive Sleep Apnea (OSA), and PH involves pathophysiological mechanisms like intermittent hypoxia, hypercapnia, and systemic inflammation. These factors link SDB to the development and progression of PH, underscoring the importance of recognizing and treating SDB in PH patients. OSA has a negative impact on asthma control, exacerbating airway inflammation. Patients suffering from both OSA and asthma often experience worse asthma symptoms, reduced lung function, and higher inflammatory markers. This highlights the crucial role of managing OSA for better asthma treatment outcomes. Inflamma-

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tory signaling pathways play a critical role in the pathogenesis and progression of various forms of PH. Immune cells and their mediators contribute to vascular remodeling, endothelial dysfunction, and elevated pulmonary arterial pressure, positioning inflammation as a potential therapeutic target. Research indicates a significant association between OSA severity and the presence of PH in asthmatic patients. This suggests OSA might worsen or contribute to PH development in individuals with asthma, emphasizing careful screening and management of both conditions. Chronic airway inflammation in asthma can lead to pulmonary vascular remodeling and potentially PH, even independently of hypoxia. This reveals the deep interconnectedness of airway and vascular pathologies. OSA specifically exacerbates eosinophilic inflammation in asthmatic patients, leading to poorer asthma control. Treating OSA could therefore help manage the inflammatory phenotype of asthma. There's a significant overlap and complex interaction between asthma, OSA, and PH. These conditions influence each other's severity and progression through shared mechanisms like inflammation and intermittent hypoxia, demanding a holistic approach to diagnosis and management. Inflammatory and oxidative stress biomarkers are important in Pulmonary Arterial Hypertension (PAH), reflecting disease activity, prognosis, and therapeutic response. This reinforces inflammation's central role in PAH pathogenesis. The pathophysiological connections between asthma and OSA involve shared risk factors and common inflammatory pathways. Nocturnal events in OSA can impact asthma severity, highlighting how treating one condition can improve outcomes for the other, emphasizing the need for integrated care.

References

1. Ramy Z, Yasar AK A H, Reem A. Pulmonary hypertension in severe asthma: A systematic review and meta-analysis. *Allergy Asthma Clin Immunol.* 2023;19:54.
2. Maurizio D, Carmela VD, Paolo P. Sleep-disordered breathing and pulmonary hypertension: *A pathophysiologic perspective. Curr Opin Pulm Med.* 2020;26:433-439.
3. Mari PT, Mandeep S, Mihaela DR. The impact of obstructive sleep apnea on asthma control and inflammation: a systematic review and meta-analysis. *Ann Am Thorac Soc.* 2021;18:978-989.
4. Mark RN, Evan DA, *Anna RH. Inflammatory Signaling in Pulmonary Hypertension. Circ Res.* 2020;127:1403-1421.
5. Sultan AA, Hani AA, *Khaled FA. The association of obstructive sleep apnea with pulmonary hypertension in patients with asthma. Egypt J Chest Dis Tuberc.* 2021;70:297-302.
6. Stéphanie C, Mathieu C, Charles-Hugo *M. Airway Inflammation and Pulmonary Vascular Remodeling in Asthma. Front Physiol.* 2020;11:593744.
7. Jessica K, Sara AC, *Stephen JR. Obstructive Sleep Apnea and Eosinophilic Airway Inflammation in Asthma. Am J Respir Crit Care Med.* 2022;206:43-52.
8. Jay B, Sanjeev PS, Rajani S. The overlap of asthma, obstructive sleep apnea, and pulmonary hypertension. *Curr Opin Pulm Med.* 2019;25:147-152.
9. Pieter AZ, Monique WVDE, *Maarten R M VDB. Biomarkers of Inflammation and Oxidative Stress in Pulmonary Arterial Hypertension. Int J Mol Sci.* 2022;23:11467.
10. Hannele J, Juha-Pekka H P L, Marja-Liisa *L L L. Understanding the Pathophysiological Links between Asthma and Obstructive Sleep Apnea. Int J Environ Res Public Health.* 2021;18:8782.

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