Osa, ph, asthma: Shared inflammatory mechanisms.

Isabelle Lefevre*

Department of Pulmonology, Sorbonne University, France

Introduction

This systematic review and meta-analysis clearly links obstructive sleep apnea (OSA), pulmonary hypertension (PH), and asthma. It demonstrates that OSA is significantly associated with PH, especially in patients already diagnosed with asthma. This suggests a potential common inflammatory pathway or an exacerbating effect of one condition on the others, emphasizing the need for comprehensive assessment in individuals presenting with co-occurring sleep and respiratory issues [1].

This narrative review explores the intricate pathophysiological connection between obstructive sleep apnea (OSA) and pulmonary hypertension (PH). It details how intermittent hypoxia and sleep fragmentation inherent in OSA lead to systemic and pulmonary inflammation, endothelial dysfunction, and vascular remodeling, thereby initiating or progressing PH. Grasping these mechanisms is essential for developing effective, targeted therapeutic interventions [2].

This review sheds light on the mechanisms driving pulmonary hypertension (PH) in severe asthma. It emphasizes how chronic airway inflammation, structural changes within the airways, persistent hypoxia, and potential systemic factors collectively contribute to increased pulmonary vascular resistance. Recognizing and managing PH as a significant comorbidity in severe asthma is crucial for improving patient outcomes [3].

This article reviews critical inflammatory biomarkers associated with obstructive sleep apnea (OSA). It highlights how intermittent hypoxia and fragmented sleep in OSA trigger a cascade of systemic inflammation, impacting cardiovascular and metabolic health. Identifying these biomarkers could help predict disease severity and complications, including the risk of pulmonary hypertension, and potentially guide personalized treatment strategies [4].

This article examines how obstructive sleep apnea (OSA) significantly influences the development and progression of pulmonary hypertension (PH). It discusses mechanisms like intermittent hypoxia, sympathetic activation, and inflammatory responses that link OSA to adverse pulmonary vascular remodeling and increased pulmonary arterial pressure. The authors emphasize that early detection and effective management of OSA are crucial for improving outcomes in patients with PH [5].

This review highlights how chronic airway inflammation, a defining characteristic of severe asthma, directly contributes to pulmonary vascular remodeling. It discusses the inflammatory mediators and cellular processes that lead to structural changes in pulmonary arteries, increasing vascular resistance and potentially predisposing asthmatic patients to pulmonary hypertension. Understanding this connection could open pathways for new therapeutic strategies targeting inflammation in severe asthma [6].

This systematic review comprehensively examines the evidence for systemic inflammation in obstructive sleep apnea (OSA). It outlines how recurrent episodes of hypoxia and reoxygenation, coupled with sleep fragmentation, activate inflammatory pathways, leading to elevated levels of cytokines and other inflammatory markers. The review emphasizes that this chronic inflammatory state contributes to various comorbidities associated with OSA, including cardiovascular issues and potentially worsening respiratory conditions like asthma [7].

This article discusses the concept of an 'asthma-pulmonary hypertension overlap' as a potentially distinct clinical entity. It explores the shared inflammatory mechanisms, structural changes, and physiological factors that connect these two conditions. Recognizing this overlap is crucial for early diagnosis and appropriate management, as the presence of pulmonary hypertension can significantly worsen outcomes for asthmatic patients [8].

This meta-analysis evaluates the effectiveness of continuous positive airway pressure (CPAP) therapy in reducing pulmonary hypertension (PH) in patients with obstructive sleep apnea (OSA). It demonstrates that CPAP significantly lowers pulmonary artery pressure, indicating that addressing OSA is a critical therapeutic strategy for mitigating PH progression. This improvement likely stems from the reduction of intermittent hypoxia and associated inflammatory responses [9].

This review highlights the substantial interplay between various chronic respiratory diseases, focusing particularly on obstructive sleep apnea (OSA) as a significant comorbidity. It details how the systemic inflammation and physiological disturbances caused by

***Correspondence to:** Isabelle Lefevre, Department of Pulmonology, Sorbonne University, France. E-mail: isabelle.lefevre@parisresearch.fr

Received: 03-Apr-2025, Manuscript No. AARRP-25-183; **Editor assigned:** 07-Apr-2025, Pre QC No. AARRP-25-183 *(PQ)*; **Reviewed:** 25-Apr-2025, QC No. AARRP-25-183; **Revised:** 06-May-2025, Manuscript No. AARRP-25-183 *(R)*; **Published:** 15-May-2025, DOI: 10.35841/aarrp-6.2.183

Citation: Lefevre I. Osa, ph, asthma: Shared inflammatory mechanisms. Res Rep Pulomonol. 2025;06(02):183.

OSA can exacerbate conditions like asthma and contribute to the development of pulmonary hypertension. The article underscores the importance of a holistic approach to managing patients with multiple respiratory comorbidities to improve overall health outcomes [10].

Conclusion

Recent research consistently highlights the significant interplay between obstructive sleep apnea (OSA), pulmonary hypertension (PH), and asthma, identifying shared inflammatory pathways and exacerbating effects among these conditions. Studies demonstrate a clear association between OSA and PH, particularly in asthmatic patients, pointing towards a potential common inflammatory mechanism. This connection is rooted in the pathophysiological responses to OSA, where intermittent hypoxia and sleep fragmentation trigger systemic and pulmonary inflammation, endothelial dysfunction, and vascular remodeling, thereby initiating or progressing PH. Chronic airway inflammation, a hallmark of severe asthma, also directly contributes to PH by inducing structural changes in pulmonary arteries and increasing vascular resistance. The persistent hypoxia and systemic factors characteristic of severe asthma further fuel this process. Moreover, OSA itself is known to induce a cascade of systemic inflammation, with recurrent hypoxia and reoxygenation leading to elevated cytokine levels and inflammatory markers. This chronic inflammatory state plays a crucial role in various comorbidities, including the worsening of respiratory conditions like asthma and the development of PH. The concept of an 'asthma-pulmonary hypertension overlap' is emerging as a distinct clinical entity, driven by shared inflammatory mechanisms and physiological factors. Recognizing and managing PH as a comorbidity in severe asthma is crucial for improving patient outcomes. Addressing OSA is likewise vital for mitigating PH progression; continuous positive airway pressure (CPAP) therapy, for instance, has proven effective in significantly lowering pulmonary artery pressure in OSA patients, primarily by reducing intermittent hypoxia and associated inflammatory responses. Overall, the literature underscores the importance of a comprehensive, holistic approach to managing patients with co-occurring sleep and respiratory issues, acknowledging OSA as a significant comorbidity that can exacerbate asthma and contribute to PH development.

References

1. Xia W, Wang C, Wang H. The relationship between obstructive sleep apnea, pulmonary hypertension and asthma in patients with sleep disorders: a systematic review and meta-analysis. *J Clin Sleep Med.* 2023;19:359-369.
2. Papakonstantinou M, Simou E, Gourgoulianis K. Obstructive Sleep Apnea and Pulmonary Hypertension: *A Narrative Review of Pathophysiological Mechanisms and Therapeutic Approaches. J Clin Med.* 2024;13:1268.
3. Hekking PP, Brusse CR, de Man FS. Pulmonary hypertension in severe asthma: insights into mechanisms and management. *Eur Respir Rev.* 2023;32:220094.
4. Lin Y, Hu Y, *Jiang Y. Biomarkers of inflammation in obstructive sleep apnea and their clinical implications. Front Med* (Lausanne). 2023;10:1185573.
5. Kim J, Park S, *Kang W. The Impact of Obstructive Sleep Apnea on the Pathogenesis and Prognosis of Pulmonary Hypertension. J Cardiovasc Transl Res.* 2023;17:373-385.
6. Furlani BC, De Souza Lima F, *Vianna JCS. The contribution of airway inflammation to vascular remodeling in severe asthma. Front Pharmacol.* 2022;13:868725.
7. Al-Abbas A, Al-Judaibi N, Al-Sultan M. Systemic inflammation in obstructive sleep apnea: a systematic review. *J Inflamm Res.* 2022;15:3163-3174.
8. Ma J, Li W, Wu Y. Asthma-pulmonary hypertension overlap: an emerging clinical entity? *Pulm Circ.* 2022;12:e12130.
9. Xu C, Yang Y, Zhang Y. Impact of continuous positive airway pressure on pulmonary hypertension in patients with obstructive sleep apnea: a meta-analysis. *Sleep Breath.* 2021;25:699-707.
10. Puggioni F, Pelaia C, Vatrella A. Chronic Respiratory Diseases and Their Comorbidities: *Focus on Obstructive Sleep Apnea. J Clin Med.* 2022;11:7472.

Citation: *Lefevre I. Osa, ph, asthma: Shared inflammatory mechanisms. Res Rep Pulomonol. 2025;06(02):183.*