ISSN 2307-8960 (online)

World Journal of *Clinical Cases*

World J Clin Cases 2024 September 26; 12(27): 6004-6131

Published by Baishideng Publishing Group Inc

World Journal of Clinical Cases

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The *WJCC* is now abstracted and indexed in PubMed, PubMed Central, *Reference Citation Analysis*, China Science and Technology Journal Database, and Superstar Journals Database. The 2024 Edition of Journal Citation Reports® cites the 2023 journal impact factor (JIF) for *WJCC* as 1.0; JIF without journal self cites: 0.9; 5-year JIF: 1.1; JIF Rank: 168/325 in medicine, general and internal; JIF Quartile: Q3; and 5-year JIF Quartile: Q3.

RESPONSIBLE EDITORS FOR THIS ISSUE

NAME OF JOURNAL
World Journal of Clinical Cases

ISSN
ISSN 2307-8960 (online)

LAUNCH DATE
April 16, 2013

FREQUENCY
Thrice Monthly

EDITORS-IN-CHIEF
Bao-Gan Peng, Salim Surani, Jerzy Tadeusz Chudek, George Kontogeorgos, Maurizio Serati

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PUBLICATION DATE
September 26, 2024

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World J Clin Cases 2024 September 26; 12(27): 6011-6014

DOI: 10.12998/wjcc.v12.i27.6011

ISSN 2307-8960 (online)

EDITORIAL

Obstructive sleep apnea-hypopnea syndrome immunological relationship

Mahmoud Ali, Alaa Ramadan, Salim Surani

Specialty type: Medicine, research and experimental

Provenance and peer review: Invited article; Externally peer reviewed.

Peer-review model: Single blind

Peer-review report's classification

Scientific Quality: Grade C

Novelty: Grade B

Creativity or Innovation: Grade B

Scientific Significance: Grade B

P-Reviewer: Wang K, China

Received: April 6, 2024

Revised: May 31, 2024

Accepted: June 14, 2024

Published online: September 26, 2024

Processing time: 114 Days and 14.8 Hours

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Abstract

Obstructive sleep apnea-hypopnea syndrome (OSAHS) is a complex disorder characterized by symptoms resulting from intermittent hypoxia and hypopnea, with research indicating a crucial role of immune system dysregulation and genetic variations in its pathogenesis. A recent Zhao *et al* study utilizes Mendelian randomization analysis to explore the causal relationship between immune cell characteristics and OSAHS. The study identifies specific lymphocyte subsets associated with OSAHS, providing valuable insights into the disease's pathophysiology and potential targets for therapeutic intervention. The findings underscore the significance of genetic and immunological factors in sleep disorders, offering a fresh perspective on OSAHS's complexities. Compared to existing literature, Zhao *et al*'s study stands out for its focus on genetic markers and specific immune responses associated with OSAHS, expanding upon previous research primarily centered on systemic inflammation. In conclusion, the study represents a significant advancement in the field, shedding light on the causal role of immune cells in OSAHS and paving the way for future research and targeted treatments.

Key Words: Obstructive sleep apnea; Mendelian randomization; Lymphocyte characteristics; Immunology; Obstructive sleep apnea-hypopnea syndrome

Core Tip: This editorial summarizes the critical findings from Zhao *et al* and their effect on our understanding of the disease and the immune system's role in pathogenesis. Stress the importance of scientific methodology in validating causal relationships, encouraging readers to approach the study's conclusions with a critical mindset. Highlight the need for further research to explain the underlying mechanisms and to explore potential therapeutic targets.

Citation: Ali M, Ramadan A, Surani S. Obstructive sleep apnea-hypopnea syndrome immunological relationship. *World J Clin Cases* 2024; 12(27): 6011-6014
URL: https://www.wjgnet.com/2307-8960/full/v12/i27/6011.htm
DOI: https://dx.doi.org/10.12998/wjcc.v12.i27.6011

INTRODUCTION

Obstructive sleep apnea-hypopnea syndrome (OSAHS) is a complex disorder characterized by symptoms resulting from intermittent hypoxia and hypopnea, affecting respiratory, cardiovascular, and cognitive functions, with research indicating a crucial role of immune system dysregulation and genetic variations in its pathogenesis. Recent research has unveiled the immune system's involvement in obstructive sleep apnea (OSA). Inflammatory processes within the upper airway mucosa contribute to airway remodeling, exacerbating the condition's severity. Immune cells, including macrophages and lymphocytes, infiltrate the upper airway tissues in response to repetitive airway collapse, leading to local inflammation. This chronic inflammation not only contributes to structural changes in the airway but also promotes oxidative stress and endothelial dysfunction, further aggravating OSA-related complications such as cardiovascular disease.

Additionally, elevated levels of inflammatory markers, such as C-reactive protein and interleukin-6, have been observed in individuals with OSA, suggesting a systemic inflammatory response[1-3]. Much attention has been paid to the deleterious consequences of OSAHS on metabolic and cardiovascular health. Recent studies have increasingly shed light on the bidirectional relationship between OSAHS and immune dysfunction. Immune cells play a pivotal role in the pathophysiology of OSAHS, contributing to local and systemic inflammation, oxidative stress, and tissue injury. Conversely, alterations in immune cell function have been observed in individuals with OSAHS, suggesting a causal relationship between OSAHS and immune cell anomalies[4,5]. Studies highlighting alterations in immune cell levels and inflammatory markers in OSAHS patients, alongside genetic associations, suggest a potential for personalized treatment strategies based on understanding the genetic and immune interplay[6,7]. Mendelian randomization is a powerful method in epidemiology and genetics that utilizes genetic variants as instrumental variables to assess causal relationships between exposures and outcomes, helping researchers discover complex relationships between environmental exposures, genetic predispositions, and disease outcomes, ultimately aiding in developing effective interventions. The recent study by Zhao *et al*[8] was the first to explore the causal relationship between immune cell characteristics and OSAHS through Mendelian randomization analysis. This innovative approach seeks understanding the complex interactions between genetics, immunity, and sleep disorders, providing a fresh perspective on OSAHS's pathophysiology. The findings suggest that certain lymphocyte subsets are significantly associated with OSAHS, marking a significant step forward in our understanding of the disease.

FINDINGS AND THEIR IMPLICATIONS

Zhao *et al*'s study represents a commendable effort to narrow the gap in knowledge regarding the immunological basis of OSAHS[8]. By using genetic data from extensive cohorts and employing Mendelian randomization, the study identifies specific lymphocyte subsets — Basophil %CD33dim HLA DR- CD66b- and CD38 on IgD + CD24- B cells — as having significant associations with OSAHS. These insights are instrumental in explaining the complex role of immune cells in the pathogenesis of OSAHS and suggest potential targets for therapeutic intervention.

The significance of these findings cannot be overstated. They add a valuable layer of understanding to the complex nature of OSAHS and open chances for future research into targeted therapies that could mitigate the disease's impact. Furthermore, this study emphasizes the importance of genetic and immunological factors in sleep disorders.

ADVANTAGES AND LIMITATIONS

The study's rigorous methodology, including a comprehensive two-sample Mendelian randomization approach, gives credibility to its findings. The selection of immune cell characteristics based on genetic variants as instrumental variables offers a framework for establishing causal relationships, minimizing confounding factors often appearing in observational studies.

However, the study has some limitations. The reliance on data from European cohorts may restrict the generalizability of the findings across different ethnicities. Furthermore, the complexity of OSAHS, coupled with the complicated nature

of immune responses, necessitates a cautious interpretation of the causal links identified. Future studies incorporating diverse populations and exploring additional immune markers will be crucial in validating and expanding upon these findings.

COMPARISON WITH CURRENT LITERATURE

The current literature on the immunological aspects of OSAHS is sparse. Previous studies have primarily focused on the association between OSAHS and systemic inflammation, with limited exploration of the genetic origin of these relationships. Zhao *et al*'s study sets itself apart by employing Mendelian randomization to uncover specific immune cell characteristics associated with OSAHS, providing a genetic basis for these observations[8].

This approach aligns with the growing interest in understanding the genetic factors contributing to sleep disorders but goes further by linking these genetic markers to specific immune responses. As such, this study represents a significant advancement in the field, offering new insights that challenge and expand upon existing knowledge.

CONCLUSION

Zhao *et al*'s groundbreaking study illuminates the causal role of immune cells in OSAHS, offering new directions for future research and potential therapeutic interventions[8]. While acknowledging its limitations, the study's innovative use of Mendelian randomization opens new horizons in understanding OSAHS, emphasizing the critical interplay between genetics and immunity. As the field progresses, these findings will undoubtedly serve as a cornerstone for further exploration and developing targeted treatments for OSAHS.

FOOTNOTES

Author contributions: Ali M was involved in the Literature search and writing of the manuscript; Ramadan A was involved in the writing of the manuscript; Surani S was involved in idea generation, writing, review, and supervision; All authors have read and approved the final manuscript.

Conflict-of-interest statement: None of the authors have any conflict of interest to disclose.

Country of origin: United States

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S-Editor: Gao CC
L-Editor: A
P-Editor: Zhao YQ

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