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Re-evaluating the Cardiopulmonary Implications of Vitamin D Deficiency in Infant Bronchiolitis: Methodological Insights and Future Directions

Bebeklerde Bronşiolit ve D Vitamini Eksikliğinin Kardiyopulmoner Etkilerinin Yeniden Değerlendirilmesi: Metodolojik Notlar ve Gelecek Perspektifleri

Gamze Yeter Arslan

Kepez State Hospital, Clinic of Cardiology, Antalya, Türkiye

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Dear Editor;

I am writing regarding the recently published article titled "Interaction Between Vitamin D Deficiency and Bronchiolitis Severity and Cardiac Function Indicators: A Prospective Study" by Arslan et al. (1), which appeared in the *Bulletin of Cardiovascular Academy* DOI:10.4274/kvbulten.galenos.2025.41636. This prospective, single-center study evaluated 60 infants aged 24-1 months hospitalized for bronchiolitis between August 2020 and July 2021, examining vitamin D levels in relation to respiratory severity, oxygen saturation, heart rate, and neutrophil count. The authors reported that the severe bronchiolitis group had significantly lower vitamin D levels, more pronounced hypoxemia, tachycardia, and inflammation, and that vitamin D deficiency was independently associated with bronchiolitis severity. This work provides clinically meaningful observations; however, several methodological considerations warrant further discussion to strengthen the interpretability and generalizability of these findings.

A primary limitation is the marked seasonal variability of vitamin D status. The study period spanned all seasons, yet no adjustments were made for seasonal fluctuations, which significantly affect serum 25-hidroksivitamin D levels in infants. Without accounting for seasonality, it remains unclear whether low vitamin D levels reflect true deficiency or physiologic winter decline. Previous studies have demonstrated that seasonal patterns influence both vitamin D metabolism and susceptibility to respiratory infections (2,3). Taken together, these seasonal fluctuations emphasize the importance of evaluating additional biological and pathogen-related factors that may further influence bronchiolitis severity.

Another important point concerns the absence of virological stratification. Bronchiolitis severity differs considerably depending on the causative pathogen. Respiratory syncytial virus typically results in more severe hypoxemia, longer hospital stays, and greater respiratory effort than with rhinovirus or other viral agents. Since the authors did not perform viral identification, disease severity could not be contextualized relative to viral etiology. Prior evidence suggests that vitamin D deficiency may differentially affect bronchiolitis severity depending on the infecting virus, making virological data highly valuable for accurate interpretation (4).

Address for Correspondence/Yazar Adresi: Gamze Yeter Arslan MD, Kepez State Hospital, Clinic of Cardiology, Antalya, Türkiye
E-posta/E-mail: dr.gamzeyeterarslan@gmail.com **ORCID ID:** orcid.org/0000-0002-0114-7448
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Although the authors propose that vitamin D deficiency may contribute to cardiopulmonary overload, cardiac assessment in the study was limited to heart rate measurements. A more comprehensive evaluation, such as measurement of N-terminal proBNP and troponin and assessment of echocardiographic indices (tricuspid annular plane systolic excursion, left ventricular ejection fraction, myocardial strain), could better elucidate subclinical myocardial stress. Earlier pediatric studies have shown that vitamin D deficiency is associated with increased cardiovascular instability and adverse outcomes in critically ill infants (5). Incorporating advanced cardiac markers into future research would, therefore, enhance mechanistic understanding.

Furthermore, the study highlights inflammatory activation but does not include inflammatory biomarkers such as C-reactive protein, interleukin-6, or tumor necrosis factor-alpha. Given vitamin D's well-described role in innate immunity and cytokine modulation, inflammatory profiling could clarify the pathways through which deficiency exacerbates bronchiolitis severity. Recent systematic reviews have confirmed vitamin D's regulatory impact on respiratory infection susceptibility and immune response dynamics (6).

Finally, while the authors suggest that vitamin D deficiency may have long-term cardiovascular implications, the study design did not include follow-up data. Longitudinal evaluation is essential to determine whether infants with recurrent bronchiolitis and low vitamin D levels exhibit later endothelial dysfunction, altered myocardial performance, or increased arrhythmogenic risk. This point is particularly relevant because vitamin D deficiency may exacerbate both the severity of respiratory disease and cardiopulmonary stress in infants with bronchiolitis.

In conclusion, the study by Arslan et al. contributes important observations linking vitamin D deficiency to increased bronchiolitis severity and cardiopulmonary strain. However, addressing key methodological limitations, including seasonality, viral heterogeneity, limited cardiac evaluation, and lack of longitudinal follow-up, will enhance future research and clinical interpretation. These considerations may guide clinicians toward a more comprehensive approach, including routine evaluation and timely correction of vitamin D deficiency in infants with acute bronchiolitis.

Footnotes

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