LETTER TO THE EDITOR

DOI: 10.4274/ijca.2025.19484
Int J Cardiovasc Acad 2025;11(2):84-85

Addressing Methodological Gaps in the Relationship Between Vitamin D and Coronary Atherosclerosis

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Abstract

Keywords: Acute coronary syndrome, coronary artery disease, cardiovascular health, ventricular dysfunction, vitamin D

To the Editor,

I read with great interest the article by Sahani and Gupta[1], titled "*Association between Vitamin D Deficiency and Angiographic Severity in Patients with Coronary Artery Disease*", published in the *International Journal of Cardiovascular Academy* [2024;10:132-8].[1] While the primary focus of vitamin D research is its role in bone health, immune function, and other systemic effects, its potential impact on cardiovascular health is noteworthy. This study provides important insights into the relationship between serum vitamin D levels and the severity of coronary artery disease (CAD). However, I would like to share some of my thoughts on some methodological aspects and contextual considerations.

Vitamin D cut-off thresholds vary significantly across populations and health conditions, reflecting the complexity of establishing a universal standard. Given the ongoing debates surrounding optimal serum vitamin D concentrations, the definitions of sufficiency, insufficiency, and deficiency are inherently approximate.[2] In this study, the authors reference a Malaysian study to define vitamin D cut-off values.[3] Considering the substantial variability in vitamin D levels across regions and populations, it would have been valuable to use India-specific reference values or provide a detailed rationale for adopting these particular thresholds. Furthermore, the study does not explain why specific cut-offs such as ≤10, 11-20, 21-30, and >30 ng/mL were chosen or whether these thresholds align with the clinical and demographic characteristics of patients with acute coronary syndrome (ACS) in India. Addressing these points would enhance the applicability and contextual relevance of this study's findings.

The timing of vitamin D measurement is another critical factor that remains unclear. It is not specified whether vitamin D levels were not assessed prior to the onset of ACS, during hospital admission, or during subsequent follow-ups. This distinction is particularly important because ACS triggers an inflammatory response characterized by elevated markers, such as C-reactive protein and interleukin-6, which are known to influence vitamin D metabolism.[4] Without this information, it becomes challenging to distinguish baseline vitamin D levels from those affected by the acute inflammatory state. Including dynamic vitamin D measurements alongside inflammatory markers could provide more robust insights into the interplay between inflammation and vitamin D levels.

Additionally, the exclusion criteria used in this study require further elaboration. Patients with chronic kidney disease and parathyroid disorders were excluded, but other potential

To cite this article: Yavuz YE. Addressing methodological gaps in the relationship between vitamin D and coronary atherosclerosis. Int J Cardiovasc Acad. 2025;11(2):84-85

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Received: 26.12.2024
Accepted: 02.01.2025
Publication Date: 20.06.2025

confounding factors, such as dietary habits, seasonal variations, and the use of statins or antihypertensive medications, were not addressed.[5] These variables can significantly influence serum vitamin D levels and CAD progression, potentially affecting the observed associations. A more comprehensive consideration of these factors would have strengthened the findings of this study.

The absence of a control group in the study limited the robust assessment of the relationship between vitamin D levels and CAD severity or left ventricular function. This deficiency leads to an emphasis on correlation rather than causality. The observed differences in left ventricular ejection fraction between patients with optimal and vitamin D deficiency also raise intriguing questions about the mechanisms involved. Although the authors attribute this to the anti-inflammatory and endothelial-modulating properties of vitamin D, alternative explanations, such as reduced physical activity in vitamin D-deficient patients, should be considered. This finding warrants further investigation to distinguish direct effects from potential confounders.

In conclusion, while Sahani and Gupta's[1] study offers valuable contributions to understanding the relationship between vitamin D and CAD severity, addressing the aforementioned methodological considerations could enhance its robustness and applicability. I commend the authors for their important work and encourage continued exploration of the role of vitamin D in cardiovascular disease.

Footnotes

Authorship Contributions

Financial Disclosure: The author declared that this study received no financial support.

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