The relationship between advanced lung cancer inflammation index and high SYNTAX score in patients with non-ST-elevation myocardial infarction

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We read with great interest the article by Konuş *et al.* investigating the relationship between the Advanced Lung Cancer Inflammation Index (ALI) and coronary artery lesion complexity as assessed by the SYNTAX score (SXscore) in patients with non-ST-elevation myocardial infarction (NSTEMI) [1]. This study provides an innovative approach to assessing the inflammatory and nutritional dimensions of coronary artery disease (CAD) severity, with potential implications for risk stratification and management. However, a critical analysis raises some methodological concerns and highlights areas for further investigation.

The authors' use of ALI – a composite index derived from body mass index (BMI), serum albumin, and the neutrophil-to-lymphocyte ratio (NLR) – is commendable as it integrates accessible, cost-effective biomarkers. The finding that a lower ALI is independently associated with a higher SXscore is consistent with mounting evidence linking inflammation and malnutrition with the progression of CAD [2, 3]. Notably, the study's novelty lies in extending the application of ALI, initially validated in oncology settings, to the realm of cardiology, where it may serve as a practical tool for clinical decision-making [4, 5].

However, several limitations temper the impact of these findings. First, the study cohort included only 15 patients with a high SXscore (≥ 33), representing 5.3% of the sample. This significant imbalance raises concerns about the robustness of statistical analyses and the potential for overfitting in the multivariable model. While the authors report that ALI had a sensitivity of 80% and specificity of 78% for predicting a high SXscore, these performance metrics require external validation in larger, more balanced cohorts. Second, the exclusion of patients with coronary artery bypass grafting (CABG), malignancies, or chronic inflammatory diseases limits the generalizability of the results to real-world populations, where these comorbidities are common.

The retrospective design of the study introduces potential selection and information biases. A prospective, multicenter study would strengthen the evidence base and mitigate these limitations. Moreover, while the authors assert the utility of ALI as a predictive tool, they do not compare its performance with established risk scores, such as the Global Registry of Acute Coronary Events (GRACE) score or individual biomarkers like C-reactive protein (CRP) or NLR, which are also independently associated with CAD severity and outcomes [6–8]. Comparative analyses would provide a critical context for evaluating ALI's incremental value in clinical practice.

The study's findings also add to the ongoing debate on the "obesity paradox" in CAD. Previous research has demonstrated an inverse relationship between BMI and CAD severity, suggesting that higher BMI might confer a protective effect under certain conditions [9]. While the authors report a similar inverse relationship in this study, they do not address potential confounders, such as systemic inflammation or the interplay between BMI and serum albumin levels. This gap underscores the need for more nuanced analyses of ALI's components to elucidate their individual and combined contributions to CAD risk.

Biologically, the association between inflammation and CAD severity is well established, with neutrophils, lymphocytes, and serum albumin playing pivotal roles. Neutrophils promote atherosclerosis through cytokine secretion, while lymphocytes modulate immune responses and may counteract inflammatory processes. Low albumin levels, indicative of chronic inflammation and malnutrition, have been linked to adverse cardiovascular outcomes [10–12]. By incorporating these markers, ALI captures both inflammatory and nutritional dimensions,

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making it an attractive prognostic tool. However, the lack of mechanistic exploration in this study limits our understanding of how ALI directly influences atherosclerotic progression.

In addition to the limitations noted, the study does not adequately address the clinical implications of its findings. While the authors suggest that ALI could inform risk stratification, they do not discuss how it might influence therapeutic decision-making, such as the choice between percutaneous coronary intervention and CABG. Furthermore, the study's reliance on a single-center cohort limits its applicability to broader, ethnically diverse populations with variable CAD risk profiles.

In conclusion, Konuş *et al.* provide an intriguing foundation for using ALI as a marker of CAD complexity. However, its clinical utility remains uncertain until validated in larger, prospective studies with diverse populations. Future research should focus on mechanistic studies, comparative analyses with established risk scores, and the integration of ALI into clinical decision-making frameworks. By addressing these gaps, ALI could emerge as a valuable addition to the armamentarium for CAD risk assessment and management.

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Ethical approval

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Conflict of interest

The author declares no conflict of interest.

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