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Neutrophil Percentage-to-Albumin Ratio and Coronary Collaterals: A True Predictor or an Overstated Association?

LETTER TO THE EDITOR

To the Editor,

I read with interest the study by Aktaş et al,[1] "Neutrophil Percentage-to-Albumin Ratio as a Predictor of Collateral Circulation in Chronic Total Occlusion" (*Anatol J Cardiol*. 2025;29(9):489–95).The authors are to be commended for addressing an easily accessible biomarker in this challenging clinical setting.

However, several points merit further discussion. First, ratio-based indices such as the neutrophil-to-lymphocyte ratio, platelet-to-lymphocyte ratio, C-reactive protein-to-albumin ratio, and monocyte-to-lymphocyte ratio have been widely investigated in cardiovascular diseases, but none have yet been incorporated into major guidelines or become standalone tools for clinical decision-making.[2–4] NPAR (neutrophil percentage-to-albumin ratio) may also face this limitation unless its prognostic value is validated in prospective, outcome-focused studies.

Second, it has been reported that CTOs (chronic total occlusion) occur most frequently in the RCA (right coronary artery), followed by the LAD (left anterior descending artery) and Cx arteries (approximately 40%-50%, 30%-35%, and 15%-20%, respectively).[5] LAD occlusions, by affecting a larger myocardial territory, are usually associated with a more robust collateral response. However, in clinical practice, RCA collaterals are observed more frequently. This is likely due to both the higher prevalence of RCA CTO and the fact that collateral channels from RCA to LAD are anatomically more prominent and more easily visualized angiographically. This raises an important concern: the predominance of RCA occlusions in this study (72%) may have led to an overestimation of the association between NPAR and poor collateral circulation. In other words, RCA dominance could have made NPAR appear as a stronger independent predictor than it might actually be in a more balanced cohort. In addition, diabetes mellitus was strikingly prevalent (81%) in the poor collateral group, and despite multivariable adjustment, such a dominant factor may still confound the independent association between NPAR and collateral circulation.

It is also noteworthy that CTOs are reported most frequently in the RCA, followed by the LAD and Cx arteries (approximately 40%-50%, 30%-35%, and 15%-20%, respectively). While LAD occlusions usually induce a more robust collateral response due to the larger myocardial territory at risk, RCA CTOs are more commonly encountered in clinical practice. This apparent paradox—greater collateral potential in LAD but higher observed collateral frequency in RCA—may be explained by both the higher prevalence of RCA CTO and anatomical differences

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in collateral pathways, with RCA-to-LAD collaterals often being more easily visualized angiographically.[5]

Finally, previous studies have reported that certain inflammatory markers, particularly monocytes, are associated with better collateral development.[6] In contrast, Aktaş et al[1] found higher NPAR to be related to poor collateral circulation. This discrepancy underscores the complex and heterogeneous nature of the inflammation–collateral relationship and highlights the need for further mechanistic research.

In conclusion, while NPAR appears to be a promising marker, its true clinical relevance requires confirmation in large-scale, multicenter, and prospective studies evaluating both mechanistic pathways and long-term outcomes.

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