Many cardiac biomarkers have been used in the diagnosis, risk assessment, and management of cardiovascular (CV) disease. In acute coronary syndrome (ACS), cardiac biomarkers are of great importance to an accurate diagnosis and prediction of the prognosis. Over the past decade, improvement in analytical techniques has established the role of clinical biochemistry in the management of ACS. Cardiac biomarkers in ACS may be related to pathophysiological mechanisms, such as myocardial injury, hemodynamic stress, inflammation, cardiac fibrosis, adverse remodeling, or other conditions. [1]

Quisi et al. [2] have evaluated the relationship between the serum galectin-3 level and the angiographic SYNTAX score (from the Synergy between percutaneous intervention with Taxus drug-eluting stent and cardiac surgery trial) in patients diagnosed with non-ST-segment elevation myocardial infarction (NSTEMI). They demonstrated that the galectin-3 level was higher in patients with a higher SYNTAX score I than those with a lower score, and that the galectin-3 level independently predicted a higher SYNTAX score. The SYNTAX score I has been proven to be an effective tool to risk stratify patients with complex coronary artery disease (CAD). [3] The authors concluded that the galectin-3 level might be used for this purpose.

Galectin-3 is a biomarker involved in many pathological processes, including inflammation, tumor growth, and fibrosis. [4] The prognostic role of galectin-3 in acute and chronic heart failure patients has been well defined; [5] however, the data on its role in ACS are limited. Winter et al. [6] observed that patients who experienced myocardial infarction (MI) at a younger age (≤40 years) had an elevated galectin-3 level during the acute phase of MI. Falcone et al. [7] found that the galectin-3 level was higher in patients with unstable angina, and that there appeared to be a relationship between the galectin-3 level and 3-vessel disease. Gucuk Ipek et al. [8] demonstrated that the serum galectin-3 level was elevated in patients with ACS compared with healthy subjects and that there was a strong correlation between the galectin-3 level and the Gensini score. In contrast, Singsaas et al. [9] did not find any relationship between galectin-3 level and acute ischemic myocardial injury.

Some limitations of the current study conducted by Quisi et al. [2] should be noted. First of all, this study included a limited number of patients. The data were obtained exclusively from NSTEMI patients; there was no control group of stable CAD or healthy subjects. Hence, the results may not be applicable for all patients on the spectrum of ACS and stable CAD. Also, the patients were divided into 2 groups, rather than the 3 groups of the SYNTAX score (low, inter-
mediate, and high). It is not known whether the significance between the 2 study groups was due to high risk or moderate and/or high risk. The researchers measured the serum galectin-3 level only at admission and they did not assess adverse CV events, either during the index hospitalization or after discharge. Thus, the study could not provide any further prognostic data. Due to the descriptive, cross-sectional design, the study could not determine the possible mechanism of action of galectin-3 in atherogenesis. The authors claimed that the association of galectin-3 with SYNTAX score might be attributable to inflammation. However, this assumption was not supported by the inflammatory markers of C-reactive protein and neutrophil-lymphocyte ratio. The receiver operating characteristic curve analysis provided a cut-off value of 14.0 ng/mL for galectin-3 to predict an intermediate or high SYNTAX score with 75.0% sensitivity and 51.0% specificity. A highly sensitive test can be useful for ruling out a disease; however, a test with a high specificity (a high true negative rate) is most useful when the result is positive, and a specific test can be valuable for ruling in a certain disease. Hence, a higher serum galectin-3 level might not precisely reflect more extensive CAD. Finally, significant racial differences in biomarkers of CV disease have been demonstrated[10] and should be considered when generalizing the findings. Nonetheless, the current work of Quisi et al.[2] is interesting and may inspire additional research and validated, prospective, interventional studies on galectin-3 and ACS.

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REFERENCES

9. Singsaas EG, Manhenke CA, Dickstein K, Orn S. Circulating Galectin-3 Levels Are Increased in Patients with Ischemic Heart Disease, but Are Not Influenced by Acute Myocardial Infarction. Cardioiology 2016;134:398−405. [CrossRef]