Letter to the editor

D-dimer in heart failure: Promising prognostic value but limited evidence for guiding clinical decisions

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To the Editor:

We read with great interest the recent article by Halim et al., *Coagulation biomarkers as prognostic indicators in heart failure: A systematic review and meta-analysis of 14,773 patients*[1]. The authors provide a valuable synthesis of 22 studies assessing coagulation markers in heart failure (HF), identifying D-dimer as the most consistently associated with adverse outcomes. Elevated D-dimer levels were linked to higher all-cause mortality (adjusted HR 1.90; 95% CI 1.09–3.32), although wide prediction intervals (0.58–6.19) indicate substantial heterogeneity across studies.

The biological rationale for D-dimer as a prognostic marker in HF is compelling. HF involves systemic inflammation, endothelial dysfunction, and hypercoagulability; thus, elevated D-dimer may reflect ongoing fibrinolysis and disease severity. Emerging data suggest it could complement established risk models such as the MAGGIC score, offering additional prognostic information across HF phenotypes[2–4].

However, important limitations temper enthusiasm for its clinical application. D-dimer is a nonspecific biomarker influenced by age, renal dysfunction, infection, and malignancy—factors common in HF. Most supporting evidence is observational, with heterogeneous populations, assay methods, and cut-off values, limiting standardization. Furthermore, no randomized trials have shown that D-dimer–guided therapy improves outcomes, and elevated levels likely represent systemic illness rather than a direct pathogenic mechanism[5].

In summary, while D-dimer holds promise as a prognostic biomarker, current data do not justify its use to guide management decisions in HF. Standardized prospective studies and interventional trials are needed to define optimal thresholds and determine whether incorporating D-dimer into clinical algorithms improves risk stratification and patient outcomes.

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