

C-Reactive Protein as a Prognostic Tool in Acute Heart Failure: Evidence from Low-Resource Settings

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Short Editorial related to the article: *Prognostic Implications of Increased C-Reactive Protein in Patients With Acute Heart Failure*

Admitted to a General Hospital Ward

Acute heart failure (AHF) remains a leading cause of hospitalization and cardiovascular mortality worldwide, with an even greater burden in low- and middle-income countries. In Brazil, in-hospital mortality for AHF is approximately 12%, compared to 4% reported in high-income countries.^{1,2}

In this context, the prospective study by Ferigato et al., conducted at a public hospital in São Paulo, assessed the prognostic value of C-reactive protein (CRP) in patients with AHF.³ This investigation is particularly relevant given the structural limitations of the Brazilian public healthcare system, where conventional biomarkers such as natriuretic peptides are often unavailable. Therefore, CRP—a low-cost and widely accessible inflammatory marker—emerges as a promising alternative for risk stratification during hospitalization.

Two aspects of the study deserve emphasis: (1) the feasibility of using a low-cost, easy-to-interpret biomarker applicable in resource-limited settings; and (2) the focus on the acute inpatient phase, which remains underexplored. Previous studies by Minami et al. and Zhang et al. evaluated the prognostic value of CRP in patients with decompensated heart failure, but predominantly during post-discharge follow-up periods ranging from three months to five years.^{4,5}

The study found that elevated CRP levels (>2.5 mg/dL) were significantly associated with higher in-hospital mortality (30% vs. 4.8%; $p = 0.046$), with an adjusted odds ratio of 25 (95% CI: 2–335). The hypothesis that CRP may serve as a prognostic marker in AHF is biologically plausible, given the central role of inflammation in the disease's pathophysiology.⁶ Previous research has linked elevated CRP levels to ventricular remodeling, progressive cardiac dysfunction, and adverse outcomes in patients with chronic heart failure.^{4–7}

The study's prospective design, echocardiographic confirmation for all patients, and robust multivariate analysis—accounting for variables such as atrial fibrillation, creatinine

levels, and infection—strengthen its methodological validity. Additionally, the heterogeneous sample, reflective of typical Brazilian public hospital populations, enhances the generalizability of the findings to routine clinical practice.

Nevertheless, important limitations must be acknowledged. The small sample size ($n = 44$) reduces statistical power and results in wide confidence intervals (CRP CI: 2–335). The use of antibiotic prescription as a surrogate for infection may introduce classification bias, complicating the distinction between infectious and non-infectious inflammation. The absence of stricter clinical and laboratory criteria to define infection limits the ability to isolate CRP as an exclusive marker of AHF severity. Previous work, such as that by Lourenço et al., suggests that CRP's prognostic value in heart failure is more robust in the absence of infection.⁸

Another critical limitation is the lack of systematic inclusion of NT-proBNP in the statistical model; only 15 out of 44 patients had this biomarker measured, which precludes more definitive comparisons regarding CRP's independent prognostic value relative to established biomarkers. Furthermore, CRP measurements were taken within 72 hours of admission, potentially introducing temporal variability that may affect the interpretation of its prognostic significance.

As a nonspecific inflammatory marker, CRP is sensitive but not specific. Levels between 1 and 10 mg/dL—as observed in this study—may reflect a range of conditions, from subclinical inflammation and endothelial dysfunction to the acute decompensation process itself. Therefore, CRP's clinical application should be contextualized and integrated with other clinical and laboratory data.

Despite these limitations, Ferigato et al.³ study makes a valuable contribution by highlighting CRP's potential as a prognostic tool in AHF within low-resource settings. While extensively studied in chronic heart failure, CRP demonstrates potential here as an aid in the early identification of patients at higher risk of in-hospital mortality due to AHF. The study underscores the importance of tailored solutions for public health systems and may represent a practical, cost-effective option. Larger, multicenter studies are warranted to directly compare CRP with natriuretic peptides and to evaluate its incremental prognostic value across diverse heart failure populations.

Given disparities in healthcare access, initiatives such as this gain importance by proposing feasible strategies tailored to public health realities. Far from being obsolete, CRP may reclaim a meaningful role in contemporary clinical practice when used judiciously alongside comprehensive patient assessment.

Keywords

C-Reactive Protein; Heart Failure; Mortality

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