

Prognostic Value of C-reactive Protein (CRP) in Patients With Acute Heart Failure (AHF) Admitted to General Wards in Brazil

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Dear Editor,

We read with great interest the article by Ferigato et al.¹ investigating the prognostic value of C-reactive protein (CRP) in patients with acute heart failure (AHF) admitted to general wards in Brazil. We would like to congratulate the authors for addressing a topic of substantial practical relevance: the search for accessible biomarkers in settings where gold-standard tools, such as natriuretic peptides, have inconsistent availability.

The study is commendable for focusing on the “real-world” scenario of public hospital wards, where the observed in-hospital mortality (18%) is significantly higher than that reported in high-income countries. Identifying CRP as an independent predictor of mortality (OR = 25) offers an opportunity for early risk stratification of patients who, although appearing hemodynamically stable for ward admission, may harbor a critical inflammatory substrate—an observation previously described in patients with acute heart failure.² Furthermore, the inclusion of echocardiographic assessment in all participants strengthens the diagnostic robustness of the findings.

Despite the promising results, two aspects warrant deeper analysis to strengthen the interpretation of the findings. It was noted that 46% of patients were receiving antibiotics, indicating a high prevalence of suspected or confirmed infections. Although antibiotic use was not retained as a predictor of mortality in the multivariate analysis, previous studies have shown that CRP may lose its specific prognostic value in the presence of infection, particularly in the setting of AHF.² In this context, a subgroup analysis excluding infected patients could help isolate the role of so-called “sterile inflammation” resulting from cardiac decompensation itself, a phenomenon well described in the pathophysiology of heart failure.³

Additionally, it would be informative if the authors could specify the causes of death observed in the cohort. Given the high proportion of patients receiving antibiotics, distinguishing between deaths due to progression to

cardiogenic shock and those related to sepsis could help clarify whether CRP is reflecting the intrinsic severity of heart failure or, alternatively, the presence of uncontrolled concomitant infections.

The odds ratio (OR) of 25 for CRP and 35 for atrial fibrillation (AF) presents extremely wide confidence intervals, such as 2 – 335 for CRP. This reflects the small sample size ($n = 44$). Although the associations reached statistical significance, this imprecision suggests model instability and limits the ability to precisely define the magnitude of risk, an expected finding in analyses with a low number of events.

The finding that NT-proBNP levels were significantly higher in the group with elevated CRP (median 19,248 vs. 2,747) is particularly relevant, given that inflammation plays a central role in the progression of heart failure and in the hemodynamic severity of the disease.^{3,4} This result raises the hypothesis that CRP may act as an indirect marker of myocardial wall stress. It would be interesting to discuss whether CRP provides incremental prognostic value beyond NT-proBNP, or whether both biomarkers partially reflect the same underlying biological phenomenon of disease severity.

Variables such as smoking status and spironolactone use showed significant differences between the high- and low-CRP groups. Although they were not retained in the final model, the impact of smoking as an inducer of chronic systemic inflammation is well established⁵ and could be explored more thoroughly in the discussion, given its potential role as a confounding factor. Moreover, there is a notable phenotypic distinction between the groups compared. The high-CRP group showed a predominance of heart failure with reduced ejection fraction (64%), whereas the low-CRP group consisted mostly of patients with preserved ejection fraction (64%).

This disparity raises the question of whether elevated CRP acts as an independent marker or whether, in this sample, it simply identifies patients with greater structural damage and systolic dysfunction. This could also explain the higher use of spironolactone in this group, aimed at reducing cardiac remodeling—a process associated with worse prognosis and higher mortality in patients with heart failure.⁶

In conclusion, the study by Ferigato et al.¹ represents an important step toward democratizing risk stratification in AHF. Addressing the points raised may help refine the clinical interpretation of CRP, avoiding oversimplified readings and enhancing its usefulness as a complementary tool for the early identification of patients who require intensified monitoring.

Palavras-chave

Heart Failure; C-Reactive Protein; Prognosis.

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Referências

1. Ferigato V, Takeyama F, Divitiis MD, Facchini GC, Gandra B, Goncalves BM, et al. Prognostic Implications of Increased C-Reactive Protein in Patients with Acute Heart Failure Admitted to a General Hospital Ward. *Int J Cardiovasc Sci* 2025;38:e20240235. doi: 10.36660/ijcs.20240235.
2. Lourenço P, Araújo JP, Paulo C, Mascarenhas J, Friões F, Azevedo A, et al. Higher C-Reactive Protein Predicts Worse Prognosis in Acute Heart Failure Only in Noninfected Patients. *Clin Cardiol*. 2010;33(11):708-14. doi: 10.1002/clc.20812.
3. Murphy SP, Kakkar R, McCarthy CP, Januzzi JL Jr. Inflammation in Heart Failure: JACC State-of-the-Art Review. *J Am Coll Cardiol*. 2020;75(11):1324-40. doi: 10.1016/j.jacc.2020.01.014.
4. Anand IS, Latini R, Florea VG, Kuskowski MA, Rector T, Masson S, et al. C-Reactive Protein in Heart Failure: Prognostic Value and the Effect of Valsartan. *Circulation*. 2005;112(10):1428-34. doi: 10.1161/CIRCULATIONAHA.104.508465.
5. Tonstad S, Cowan JL. C-Reactive Protein as a Predictor of Disease in Smokers and Former Smokers: A Review. *Int J Clin Pract*. 2009;63(11):1634-41. doi: 10.1111/j.1742-1241.2009.02179.x.
6. Chirinos JA, Zambrano JP, Chakko S, Schob A, Veerani A, Perez GO, et al. Usefulness of C-Reactive Protein as an Independent Predictor of Death in Patients with Ischemic Cardiomyopathy. *Am J Cardiol*. 2005;95(1):88-90. doi: 10.1016/j.amjcard.2004.08.065.



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