

Prognostic Implications of Increased C-Reactive Protein in Patients With Acute Heart Failure Admitted to a General Hospital Ward

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Abstract

Background: C-reactive protein (CRP) is a widely available inflammatory biomarker. Its prognostic role during hospitalization in patients with acute heart failure (HF) remains unclear.

Objective: To assess whether CRP measured at hospital admission predicts in-hospital mortality in patients with acute HF admitted to a general public hospital ward.

Methods: This prospective study enrolled patients (≥ 18 years old) admitted to a medical ward with a primary diagnosis of acute HF directly from the emergency department. CRP was measured within 72 hours of hospital admission. All patients underwent a structured clinical assessment by a cardiologist and transthoracic echocardiography (TTE). A p-value < 0.05 was considered statistically significant.

Results: A total of 44 patients were included (mean age 68 ± 16 years). TTE showed left ventricular ejection fraction (LVEF) $< 50\%$ in 43% of patients, preserved LVEF in 43%, and specific causes of HF in the remaining 14%. The median CRP level (2.5 mg/dL) was used to define high and low CRP groups. Reduced LVEF ($< 50\%$) was more common, and heart failure with preserved ejection fraction (HFpEF) was less frequent, in the high CRP group ($p = 0.014$). Clinical assessment showed no differences in hemodynamic profiles (congestion vs. perfusion). Comorbidities, renal function, and infection diagnoses were also similar between groups. In-hospital mortality was 30% in the high CRP group versus 4.8% in the low CRP group ($p = 0.046$). In multivariable analysis, increased CRP remained independently associated with in-hospital mortality.

Conclusion: CRP measured near the time of hospital admission may serve as a prognostic marker for in-hospital mortality in patients with acute HF.

Keywords: Heart Failure; Prognosis; C-Reactive Protein.

Introduction

The only published national multicenter registry on acute heart failure (HF) in Brazil reported an in-hospital mortality rate of approximately 12% among patients admitted for this condition.¹ This figure stands in stark contrast to the substantially lower mortality rates reported in high-income countries — about 4%.² Poorer outcomes in Brazil may be partly attributed to the preferential admission of more critically ill patients due to limited bed availability.³ Most acute HF-related hospitalizations in Brazil occur in general medical wards of public hospitals, where patient care is typically provided by health care teams without specialized

training in cardiology. Accurate risk stratification is particularly important for resource allocation but remains challenging in routine practice. Clinical risk scores developed in research settings are likely to be underutilized by frontline health care teams.⁴ Additionally, well-established prognostic biomarkers for acute HF, such as natriuretic peptides, have yet to be widely adopted in the Brazilian public health care system.⁵

The reality of the hospital affiliated with our academic institution reflects this scenario. It serves as the primary public referral center for both medical and surgical admissions in a region of seven cities in the interior of the state of São Paulo, with an estimated population of 900,000. As part of standard care, natriuretic peptide testing is typically requested at least once during the hospitalization of patients diagnosed with HF. However, access to this test remains inconsistent due to irregular availability within our healthcare setting. This context prompted us to investigate the potential role of C-reactive protein (CRP) as an alternative biomarker for risk stratification in this patient population. There is indeed a sound theoretical rationale for this approach: (1) CRP testing is universally available across healthcare facilities; (2) there is biological plausibility supporting

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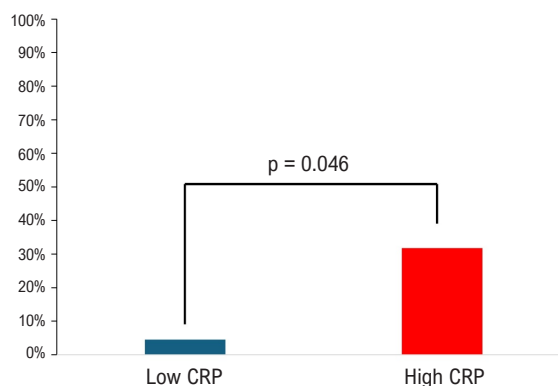
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Central Illustration: Prognostic Implications of Increased C-Reactive Protein in Patients With Acute Heart Failure Admitted to a General Hospital Ward



**In-hospital mortality according to
Low CRP vs high CRP**



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an association between increased CRP levels and adverse outcomes;⁶ and (3) in the outpatient HF population, increased CRP has already been shown to correlate with increased morbidity and mortality.⁷

Compared with patients admitted for acute HF who are directly transferred from the emergency department to the intensive care unit (ICU), those initially hospitalized in general medical wards present a notably more heterogeneous prognosis. We believe that this latter group in particular may benefit from risk stratification based on readily accessible resources. Therefore, this study aims to investigate the prognostic role of increased CRP in patients with a primary diagnosis of acute HF admitted to a general medical ward within the public health care system.

Methods

Study design

From March 2023 to April 2024, we conducted an observational study in a general medical ward involving patients hospitalized with a primary diagnosis of acute HF who were admitted directly from the emergency department. Eligible patients were required to be 18 years of age or older and to provide written informed consent prior to enrollment. The original study sample was designed to evaluate additional outcomes, the results of which have so far only been presented in conference abstracts.

Study procedures

All participants underwent a standardized clinical assessment for HF performed by a cardiologist to confirm the primary diagnosis, evaluate comorbidities, and classify patients according to the hemodynamic profile proposed by Stevenson et al.:⁸

A (warm and dry), B (warm and wet), C (cold and wet), and L (cold and dry). As part of routine care, all patients also underwent transthoracic echocardiography (TTE), performed by certified cardiologists from the clinical care team using a Philips Affiniti CVX[®] system. Left ventricular ejection fraction (LVEF) was calculated using the Teicholz method, or the Simpson method when segmental wall motion abnormalities were present.

CRP values used in this study were obtained from the electronic laboratory records and corresponded to the result closest to the date of hospital admission (within 72 h). CRP testing was ordered at the discretion of the attending physician, collected by the nursing staff, and analyzed by the hospital's local laboratory, which is managed by the Associação Fundo de Incentivo à Pesquisa (AFIP). CRP was measured using an immunoturbidimetric assay, with results reported in milligrams per deciliter (mg/dL).

For patients who had natriuretic peptides measured, these data were analyzed separately. When available, NT-proBNP was the assay used, measured by chemiluminescence and reported in picograms per milliliter (pg/mL).

Statistical analysis

Data were analyzed using IBM SPSS software, version 25. The Kolmogorov–Smirnov test was used to assess the normality of continuous variables. Variables with a normal distribution were expressed as mean and standard deviation (SD), while non-normally distributed variables were reported as median and interquartile range (IQR, Q1–Q3). Categorical variables were presented as proportions (percentages).

The sample was divided based on the median CRP value, and comparisons between the high and low CRP groups were performed using the unpaired Student's *t*-test for continuous variables (or the Mann–Whitney *U* test, as appropriate) and the

chi-square test (χ^2) for categorical variables (or Fisher's exact test, when indicated). All statistical tests were two-tailed.

A logistic regression model was subsequently applied to assess the risk of in-hospital mortality. The following variables were included: age, sex, relevant comorbidities, HF classification based on LVEF and hemodynamic profile, use of antibiotics, HF medications, creatinine levels, and CRP group (high vs. low). Variables were entered using the stepwise method (likelihood ratio). A p-value <0.05 was considered indicative of statistical significance.

Results

For this analysis, a total of 59 patients were initially assessed. Of these, 11 were excluded because they were first admitted to the ICU before being transferred to the medical ward. An additional 4 patients were excluded due to the absence of CRP measurements within 72 hours of admission to the emergency department, resulting in a CRP measurement rate of 92%. Therefore, the final study sample included 44 patients, with a mean age of 68 ± 16 years, and the majority were male (57%). The average length of stay in the emergency department prior to transfer to the ward was $1.0 (\pm 0.7)$ days.

TTE was performed on the 4th (± 2) day of hospitalization and showed an LVEF $<50\%$ in 19 of the 44 patients (43%), with a mean LVEF of $34\% (\pm 6)$. The underlying etiologies identified in this group were ischemic heart disease (30%) and hypertensive heart disease (20%), followed by alcohol and illicit drug use (10%) as well as valvular disease and Chagas disease (5% each).

Among the subgroup with LVEF $>50\%$, 19 patients (43% of total sample) were classified as having heart failure with preserved ejection fraction (HFpEF). In the remaining six patients with LVEF $>50\%$, specific etiologies for HF were identified: three had severe aortic stenosis, one had severe mitral regurgitation, one had a history of corrective surgery for transposition of the great arteries, and one had hypertrophic cardiomyopathy.

Figure 1 shows the distribution of CRP levels in the overall sample, revealing a right-skewed distribution. The median CRP (Q1–Q3) was 2.5 (1.3–6.0) mg/dL. Based on this median value, the sample was divided into two groups: high CRP (>2.5 mg/dL) and low CRP (≤ 2.5 mg/dL).

Table 1 shows that there were no differences in age or sex between the groups. However, HF classification differed significantly, with a higher proportion of patients with LVEF $<50\%$ (and a lower proportion with HFpEF) in the high CRP group compared to the low CRP group.

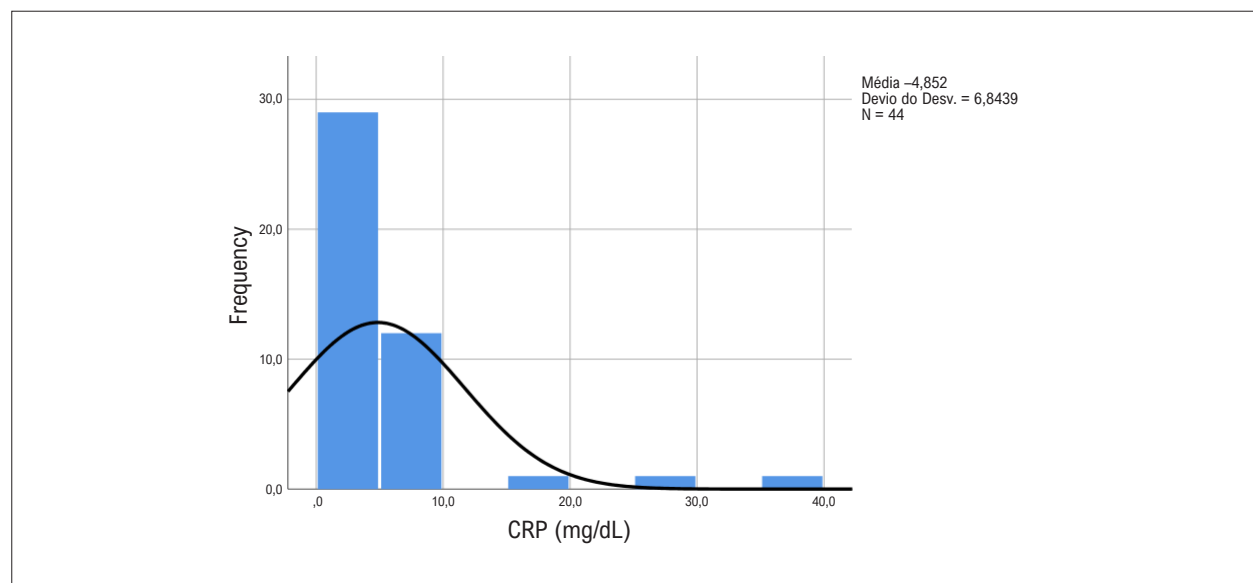


Figure 1 – Histogram of CRP distribution in the study sample. CRP: C-reactive protein

Table 1 – Demographic, clinical, and laboratory data

Variables	Total N = 44	High CRP N = 22	Low CRP N = 22	p-value
Age, years	68 ± 16	65 ± 18	71 ± 13	0.152
Male sex, %	57%	59%	54%	0.761
Smoking, % (current or former)	56%	76%	33%	0.007
HF classification				

According to LVEF				0.014
LVEF <50% (reduced or mildly reduced)	43%	64%	22%	
HFpEF	43%	22%	64%	
Specific etiologies*	14%	14%	14%	
Hemodynamic profile				0.384
A (warm and dry)	27%	18%	36%	
B (warm and wet)	50%	55%	46%	
C (cold and wet)	23%	27%	18%	
L (cold and dry)	0%	0%	0%	
Vital signs				
Heart rate, bpm	82 ± 19	86 ± 22	78 ± 17	0.218
SBP, mmHg	113 ± 26	112 ± 28	114 ± 26	0.848
DBP, mmHg	71 ± 15	69 ± 10	74 ± 18	0.249
Respiratory rate, rpm	19 ± 6	20 ± 7	19 ± 6	0.340
Comorbidities, %				
CAD	30%	27%	32%	0.741
AF	46%	46%	46%	1.000
Diabetes mellitus	43%	46%	41%	0.761
Stroke	14%	14%	14%	1.000
COPD	32%	32%	32%	1.000
Cancer	9%	9%	9%	1.000
Drugs for HF, %				
ACEI/ARB/ARNI	46%	50%	41%	0.545
Beta-blocker	51%	46%	57%	0.443
Spironolactone	54%	71%	36%	0.021
Furosemide	75%	73%	77%	0.728
SGLT2 inhibitor	5%	9%	0%	0.488
Infections				
Antibiotic use, %	46%	50%	41%	0.545
Infection site, %				
Lung	45%	55%	34%	
Urinary tract	30%	36%	22%	
Skin	10%	9%	11%	
Bloodstream	5%	0%	11%	
Unspecified	10%	0%	22%	0.437
Laboratory parameters				
Creatinine, mg/dL	1.6±1.1	1.5±0.6	1.8±1.3	0.249
Hemoglobin, g/dL	12.1±2.1	11.9±2.2	12.3±2.1	0.572
NT-proBNP**, pg/mL	4809 (2463–19248)	19248 (23491–34585)	2747 (426–7185)	0.021

ACEI: angiotensin-converting enzyme inhibitor; ARB: angiotensin receptor blocker; ARNI: angiotensin receptor–neprilysin inhibitor; bpm: beats per minute; CAD: coronary artery disease; COPD: chronic obstructive pulmonary disease; DBP: diastolic blood pressure; HF: heart failure; HFpEF: HF with preserved ejection fraction; LVEF: left ventricular ejection fraction; rpm: respirations per minute; SBP: systolic blood pressure; AF: atrial fibrillation; CRP: C-reactive protein. *LVEF >50% with identified specific etiologies of HF: valvular disease, congenital heart disease, and hypertrophic cardiomyopathy. ** N = 7 and 8 in the high and low CRP groups, respectively. Continuous variables are presented as mean (± SD), except for NT-proBNP, which is presented as median (Q1–Q3).

Another significant difference was observed in smoking history, with a higher prevalence of current or former smokers in the high CRP group compared to the low CRP group. Other comorbidities did not differ between groups, including coronary artery disease, atrial fibrillation (AF), chronic obstructive pulmonary disease (COPD), diabetes mellitus, stroke, and cancer. No participants were undergoing renal replacement therapy at the time of assessment, and creatinine levels measured concurrently with CRP were not significantly different between the high and low CRP groups.

Regarding HF medications, only the use of spironolactone was significantly more common in the high CRP group compared to the low CRP group, likely reflecting the higher proportion of patients with LVEF <50% in that group. Notably, approximately half of the patients were receiving beta-blockers and renin-angiotensin system inhibitors, and 75% were on furosemide (Table 1).

The targeted clinical assessment for HF was performed around the 3rd ($\pm$ 2nd) day of hospitalization. Differences between groups in hemodynamic profiles (A, B, C, and L) were not statistically significant. No patients were receiving vasopressor support while in the ward. Vital signs, including heart rate, respiratory rate, systolic blood pressure, and diastolic blood pressure, were similar between the groups (Table 1).

Despite higher CRP levels, the proportion of patients with a diagnosis of bacterial infection (inferred from antibiotic prescription) was not significantly higher in the high CRP group compared to the low CRP group (Table 1). The distribution of infection sites (lung, urinary tract, skin, bloodstream, and unspecified) was also similar between groups, with pneumonia being the most frequently identified infection.

Regarding in-hospital mortality, the rate was 30% in the high CRP group versus 4.8% in the low CRP group ($p = 0.046$; Central Illustration). A logistic regression model was constructed to assess the risk of in-hospital death, incorporating the following variables: demographics, comorbidities, HF classification based on LVEF and hemodynamic profile, use of antibiotics, HF medications, creatinine levels, and CRP group (high vs. low).

The model was statistically significant ($\chi^2 = 16.415$; $df = 2$; $p < 0.001$; $R^2 = 0.553$) and identified two independent predictors of in-hospital mortality: high CRP (OR = 25; 95% CI: 2–335) and AF (OR = 35; 95% CI: 3–449) (Table 2).

To further clarify the significance of increased CRP in our sample, we analyzed available NT-proBNP data from seven patients in the high CRP group and eight patients in the low CRP group. All measurements were performed within the first three

days of hospital admission. NT-proBNP levels exceeded the age-adjusted diagnostic thresholds for acute HF in 87% of patients. Notably, NT-proBNP levels were higher in the high CRP group compared to the low CRP group (Table 1).

Discussion

In this study conducted at a general hospital within Brazil's public health care system, we found that even among patients with acute HF deemed stable enough for admission to a general medical ward, in-hospital mortality remained high — about 18%. However, the main finding of this study is that CRP measurement at the time of initial assessment appears to be a promising tool for assisting in risk stratification for in-hospital mortality in this population.

CRP is classified as an acute-phase reactant; however, it is well established that increased CRP levels accompany both acute and chronic inflammatory processes, whether infectious or non-infectious, as well as conditions associated with increased metabolic stress.⁹ Its levels correlate both temporally and proportionally with the intensity of the inflammatory response. Among apparently healthy individuals, CRP levels are influenced by factors such as age, sex, and ethnicity.¹⁰ Common prevalent conditions like hypertension and diabetes, along with lifestyle factors, also contribute to variability in CRP levels.⁹

Using the units applied in the present study, CRP values below 0.3 mg/dL are considered normal, while levels between 0.3 and 1 mg/dL are typically classified as low-grade inflammation.¹¹ Values above 1 mg/dL are generally associated with classic inflammatory states.⁹ No single cutoff reliably distinguishes infectious from non-infectious causes of inflammation, nor does it differentiate bacterial from viral infections within the infectious category. Nevertheless, infections — particularly bacterial — are the most common causes when CRP exceeds 10 mg/dL,¹² and especially when levels surpass 50 mg/dL.¹³ In our sample (Figure 1), most participants had CRP levels within the range classified as moderate elevation (1–10 mg/dL),⁹ a range in which the underlying causes are more diverse and may include inflammatory activity directly related to the acute decompensation of HF itself.

To date, the prognostic impact of increased CRP in HF has been demonstrated primarily in patients with chronic HF.^{7,14–16} In this context, higher CRP levels are associated with clinical indicators of disease severity (e.g., functional class), ventricular remodeling, and reduced quality of life.⁷ In a cross-sectional analysis of patients with chronic HF, CRP was shown to provide prognostic information regarding mortality and major adverse events independently of BNP levels.⁷

Table 2 – Logistic regression model: predictors of in-hospital mortality

Variables	Odds Ratio	95% CI
High CRP (vs. low CRP)	25	2–335
AF	35	3–449

AF: atrial fibrillation; CRP: C-reactive protein. Not retained in the model: age ($p = 0.827$); sex ($p = 0.942$); smoking ($p = 0.978$); antibiotic use ($p = 0.609$); heart failure classification by LVEF ($p = 0.334$); hemodynamic profile ($p = 0.940$); CAD ($p = 0.453$); COPD ($p = 0.727$); diabetes ($p = 0.283$); stroke ($p = 0.650$); cancer ($p = 0.608$); ACEI/ARB/ARNI ($p = 0.890$); beta-blocker ($p = 0.671$); spironolactone ($p = 0.120$); furosemide ($p = 0.098$); SGLT2 inhibitor ($p = 0.303$); creatinine ($p = 0.447$).

By comparison, the prognostic role of CRP has been less extensively studied in acute HF. Moreover, all previous studies in this area have extended follow-up beyond the hospitalization period.¹⁷⁻²³ In this context, CRP measured at hospital admission has consistently been a strong predictor of major future outcomes, such as death or rehospitalization. However, these outcomes were assessed over varying follow-up periods, ranging from 120 days,¹⁷ to 1 year,¹⁸⁻¹⁹ and even up to nearly 5 years.²⁰ To the best of our knowledge, this is the first study to provide evidence that increased CRP serves as a risk marker for in-hospital mortality in patients with acute HF.

Strengths and limitations

In addition to the relatively small sample size, the absence of adjustment for BNP levels represents one of the main limitations of our study. As with other similar publications, the usual concerns regarding the interpretation of CRP in patients with acute HF — particularly given the high prevalence of infectious diagnoses — also apply to our findings. Notably, one research group reported that CRP predicted 3-month outcomes (death and rehospitalization) only in the subgroup without a concomitant diagnosis of infection.²¹

We included only patients hospitalized with a primary diagnosis of acute HF, as confirmed by the research team and supported by TTE performed in all participants. Furthermore, excluding all potential conditions associated with increased CRP in this population — such as smoking, COPD, or metabolic syndrome — would not only have been impractical but also of limited clinical relevance.

Conclusion

Regardless of the individual contribution of the various mechanisms leading to CRP increase in acute HF, this widely available marker of inflammatory activity may be useful for early risk stratification of in-hospital mortality among patients admitted with decompensated HF. Future studies are strongly needed to confirm our findings in larger cohorts and to further investigate whether the prognostic value of CRP is independent of BNP.

Author Contributions

Conception and design of the research, analysis and interpretation of the data, statistical analysis and writing of the manuscript: Figueredo Junior AR; acquisition of data: Ferigato V, Takeyama F, Facchini GC, Gandra B, Goncalves BM, Boros GAB, Figueredo Junior AR; critical revision of the manuscript for intellectual content: Ferigato V, Takeyama F, De Divitiis M, Boros GAB, Gomes HJA.

Potential Conflict of Interest

No potential conflict of interest relevant to this article was reported.

Sources of Funding

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Study Association

This article is part of the thesis of master submitted by Vinicius Ferigato and Fernanda Takeyama, from Faculdade de Medicina de Jundiaí.

Ethics Approval and Consent to Participate

This study was approved by the Ethics Committee of the Faculdade de Medicina de Jundiaí under the protocol number 5.876.004. All the procedures in this study were in accordance with the 1975 Helsinki Declaration, updated in 2013. Informed consent was obtained from all participants included in the study.

Use of Artificial Intelligence

The authors did not use any artificial intelligence tools in the development of this work.

Availability of Research Data

The underlying content of the research text is contained within the manuscript.

References

1. Albuquerque DC, Souza JD Neto, Bacal F, Rohde LE, Bernardes-Pereira S, Berwanger O, et al. I Brazilian Registry of Heart Failure - Clinical Aspects, Care Quality and Hospitalization Outcomes. *Arq Bras Cardiol*. 2015;104(6):433-42. doi: 10.5935/abc.20150031.
2. Francis GS. Acute Heart Failure: Patient Management of a Growing Epidemic. *Am Heart Hosp J*. 2004;2(4 Suppl 1):10-4.
3. Godoy HL, Silveira JA, Segalla E, Almeida DR. Hospitalization and Mortality Rates for Heart Failure in Public Hospitals in São Paulo. *Arq Bras Cardiol*. 2011;97(5):402-7. doi: 10.1590/s0066-782x2011005000096.
4. Miró Ò, Rossello X, Gil V, Martín-Sánchez FJ, Llorens P, Herrero-Puente P, et al. Predicting 30-Day Mortality for Patients with Acute Heart Failure in the Emergency Department: A Cohort Study. *Ann Intern Med*. 2017;167(10):698-705. doi: 10.7326/M16-2726.
5. Braga AA, Santos MS, Tura BR, Barros BM, Morais QCD, Oliveira IG. Availability of Natriuretic Peptide Testing in Primary Public Health Care in Brazil. *ABC Heart Fail Cardiomyop*. 2023;3(4):e20230087. doi: 10.36660/abchf.20230087.
6. 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.
7. 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.
8. Nohria A, Tsang SW, Fang JC, Lewis EF, Jarcho JA, Mudge GH, et al. Clinical Assessment Identifies Hemodynamic Profiles that Predict Outcomes in Patients Admitted with Heart Failure. *J Am Coll Cardiol*. 2003;41(10):1797-804. doi: 10.1016/s0735-1097(03)00309-7.
9. Nehring SM, Goyal A, Patel BC. C Reactive Protein. Treasure Island: StatPearls Publishing; 2025.
10. Woloshin S, Schwartz LM. Distribution of C-Reactive Protein Values in the United States. *N Engl J Med*. 2005;352(15):1611-3. doi: 10.1056/NEJM200504143521525.

11. Kushner I, Antonelli MJ. What Should we Regard as an "Elevated" C-Reactive Protein Level? *Ann Intern Med.* 2015;163(4):326. doi: 10.7326/L15-5126.
12. Landry A, Docherty P, Ouellette S, Cartier LJ. Causes and Outcomes of Markedly Elevated C-Reactive Protein Levels. *Can Fam Physician.* 2017;63(6):e316-e323.
13. Vanderschueren S, Deeren D, Knockaert DC, Bobbaers H, Bossuyt X, Peetermans W. Extremely Elevated C-Reactive Protein. *Eur J Intern Med.* 2006;17(6):430-3. doi: 10.1016/j.ejim.2006.02.025.
14. 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.
15. Ronnow BS, Reyna SP, Muhlestein JB, Horne BD, Maycock CAA, Bair TL, et al. C-Reactive Protein Predicts Death in Patients with Non-Ischemic Cardiomyopathy. *Cardiology.* 2005;104(4):196-201. doi: 10.1159/000088138.
16. Ishikawa C, Tsutamoto T, Fujii M, Sakai H, Tanaka T, Horie M. Prediction of Mortality by High-Sensitivity C-Reactive Protein and Brain Natriuretic Peptide in Patients with Dilated Cardiomyopathy. *Circ J.* 2006;70(7):857-63. doi: 10.1253/circj.70.857.
17. Minami Y, Kajimoto K, Sato N, Hagiwara N, Takano T; ATTEND Study Investigators. C-Reactive Protein Level on Admission and Time to and Cause of Death in Patients Hospitalized for Acute Heart Failure. *Eur Heart J Qual Care Clin Outcomes.* 2017;3(2):148-56. doi: 10.1093/ehjqcco/qcw054.
18. Siirilä-Waris K, Lassus J, Melin J, Peuhkurinen K, Nieminen MS, Harjola VP, et al. Characteristics, Outcomes, and Predictors of 1-Year Mortality in Patients Hospitalized for Acute Heart Failure. *Eur Heart J.* 2006;27(24):3011-7. doi: 10.1093/eurheartj/ehl407.
19. Villacorta H, Masetto AC, Mesquita ET. C-Reactive Protein: An Inflammatory Marker with Prognostic Value in Patients with Decompensated Heart Failure. *Arq Bras Cardiol.* 2007;88(5):585-9. doi: 10.1590/s0066-782x2007000500014.
20. Zhang L, He G, Huo X, Tian A, Ji R, Pu B, et al. Long-Term Cumulative High-Sensitivity C-Reactive Protein and Mortality Among Patients with Acute Heart Failure. *J Am Heart Assoc.* 2023;12(19):e029386. doi: 10.1161/JAHA.123.029386.
21. 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.
22. Alonso-Martínez JL, Llorente-Diez B, Echegaray-Agata M, Olaz-Preciado F, Urbieto-Echezarreta M, González-Arencibia C. C-Reactive Protein as a Predictor of Improvement and Readmission in Heart Failure. *Eur J Heart Fail.* 2002;4(3):331-6. doi: 10.1016/s1388-9842(02)00021-1.
23. Santas E, Villar S, Palau P, Llàcer P, de la Espriella R, Miñana G, et al. High-Sensitivity C-Reactive Protein and Risk of Clinical Outcomes in Patients with Acute Heart Failure. *Sci Rep.* 2024;14(1):21672. doi: 10.1038/s41598-024-72137-0.



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