

Prognostic Value of Growth Differentiation Factor-15 in Heart Failure: A Systematic Review with Meta-Analysis

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Abstract

Background: Growth differentiation factor-15 (GDF-15) has emerged as a promising biomarker in heart failure (HF), reflecting multiple pathophysiological mechanisms, including inflammation, oxidative stress, and cardiac remodeling. However, evidence regarding its prognostic value remains inconsistent.

Objective: To systematically evaluate the association between circulating levels of GDF-15 and adverse clinical outcomes, including all-cause mortality and HF hospitalization.

Methods: A systematic review was conducted in accordance with Preferred Reporting Items for Systematic Reviews and Meta-Analyses 2020 guidelines. PubMed, Embase, Cochrane Library, and Web of Science were searched through April 2025. Studies reporting baseline levels of GDF-15 and adjusted hazard ratios (HRs) for mortality and/or HF hospitalization were included. Pooled HRs were calculated using a random-effects model (DerSimonian and Laird method). Statistical heterogeneity was assessed using Higgins' I^2 statistic, and publication bias was evaluated using Egger's test.

Results: Seven studies ($n = 10,578$ patients [mean age, 67 ± 11 years; 58% male]) met the inclusion criteria. Increased levels of GDF-15 were significantly associated with increased all-cause mortality (pooled HR, 2.72; 95% CI, 1.55-4.17; $p < 0.001$; $I^2 = 49\%$) and HF hospitalization (HR, 2.43; 95% CI, 1.79-3.30; $p < 0.001$). Subgroup analyses demonstrated consistent associations across different HF phenotypes. Funnel plot assessment and Egger's test ($p = 0.12$) showed no evidence of publication bias.

Conclusions: Higher circulating levels of GDF-15 are independently associated with increased risks of mortality and HF hospitalization. Incorporating GDF-15 into multimarker prognostic models may improve risk stratification and support earlier, more individualized management strategies in HF.

Keywords: Heart Failure; Growth Differentiation Factor 15; Biomarkers; Prognosis.

Introduction

Heart failure (HF) is a complex clinical syndrome characterized by the inability of the heart to maintain adequate perfusion to meet the metabolic demands of the body. Despite substantial therapeutic advances, HF remains a leading cause of morbidity and mortality worldwide, imposing a considerable burden on health care systems. The 5-year mortality rate among patients with HF is comparable to and in some cases exceeds that observed in several malignancies, highlighting the urgent need to improve prognostic assessment and optimize individualized management strategies.

Circulating biomarkers are central to the diagnosis, prognostic evaluation, and therapeutic monitoring of HF. Although natriuretic peptides, such as B-type natriuretic

peptide (BNP) and N-terminal pro-BNP (NT-proBNP), are firmly established in clinical practice, their diagnostic and prognostic performance may be reduced in specific populations, including individuals with obesity, older adults, and patients with kidney dysfunction. These limitations have driven interest in novel biomarkers capable of providing complementary or incremental prognostic information.

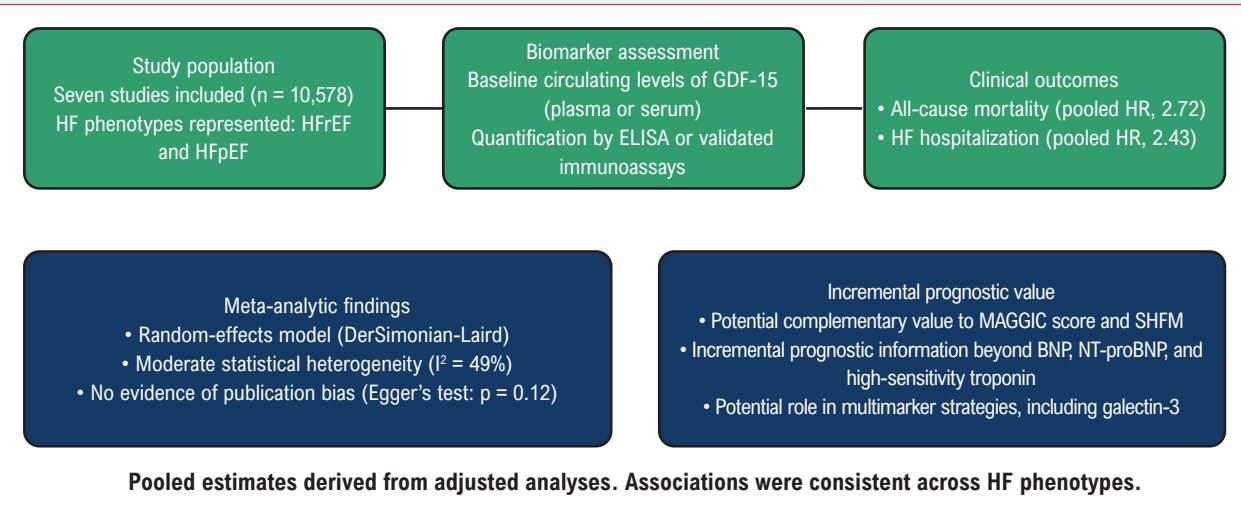
Growth differentiation factor-15 (GDF-15), a stress-responsive cytokine belonging to the transforming growth factor- β (TGF- β) superfamily, is upregulated in response to inflammation, oxidative stress, and myocardial remodeling, which are central mechanisms in HF pathophysiology. Experimental and clinical evidence has demonstrated that increased levels of GDF-15 are associated with adverse cardiovascular outcomes, including increased mortality and HF hospitalization.¹⁻⁴ However, the strength and consistency of these associations vary across studies, potentially owing to differences in study populations, assay methodologies, and biomarker threshold definitions.

Because of the expanding body of evidence, a quantitative synthesis is warranted to better define the prognostic value of GDF-15 in HF and its potential contribution to improving risk stratification models. Therefore, this systematic review aimed to evaluate the association between circulating levels

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Central Illustration: Prognostic Value of Growth Differentiation Factor-15 in Heart Failure: A Systematic Review with Meta-Analysis



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BNP: B-type natriuretic peptide; ELISA: enzyme-linked immunosorbent assay; GDF-15: growth differentiation factor-15; HF: heart failure; HFpEF: HF with preserved ejection fraction; HFrEF: HF with reduced ejection fraction; HR: hazard ratio; MAGGIC: Meta-Analysis Global Group in Chronic Heart Failure; NT-proBNP: N-terminal pro-BNP; SHFM: Seattle Heart Failure Model.

of GDF-15 and major clinical outcomes, specifically all-cause mortality and HF-related hospitalization, in adult patients diagnosed with HF.

Methods

Study design and reporting standards

This systematic review and meta-analysis was conducted in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) 2020 guidelines.⁵ The study protocol was prospectively developed to evaluate the prognostic association between circulating levels of GDF-15 and adverse clinical outcomes in patients with HF.

Search strategy

A comprehensive literature search was conducted in PubMed, Embase, Cochrane Library, and Web of Science to identify studies published through April 2025, without language restrictions. The search strategy combined controlled vocabulary and free-text terms as follows: ("GDF-15" OR "Growth Differentiation Factor-15") AND ("heart failure") AND ("prognosis" OR "mortality" OR "hospitalization"). Reference lists of eligible studies and relevant review articles were also manually screened to identify additional potentially eligible publications.

Eligibility criteria

Studies were considered eligible if they met all predefined inclusion criteria. Eligible studies included prospective or retrospective cohort studies as well as randomized controlled

trials reporting baseline measurement of GDF-15. The study population consisted of adult patients (≥ 18 years) with a clinical or echocardiographic diagnosis of HF. Studies were required to report plasma or serum levels of GDF-15 measured at baseline using validated analytical methods and to provide hazard ratios (HRs) with corresponding 95% CIs for all-cause mortality and/or HF hospitalization. In addition, studies were required to have a minimum follow-up duration of 6 months and to include multivariable-adjusted analyses accounting for relevant clinical covariates.

Studies were excluded if they i) included pediatric populations or experimental animal models; ii) were narrative reviews, editorials, letters, or case reports; iii) lacked sufficient information to extract or calculate HR estimates; or iv) did not report multivariable-adjusted analyses.

Study selection

Two independent review authors screened titles and abstracts for eligibility. Full-text articles of potentially relevant studies were subsequently assessed for final inclusion. Disagreements were resolved through consensus or adjudication by a third review author. The article selection process is summarized in the PRISMA flow diagram (Figure 1).

Data extraction

Data extraction was independently conducted by two reviewers using a standardized data collection form. Extracted variables included author and year of publication, study design, sample size, baseline patient characteristics (including age, sex distribution, and HF phenotype), method

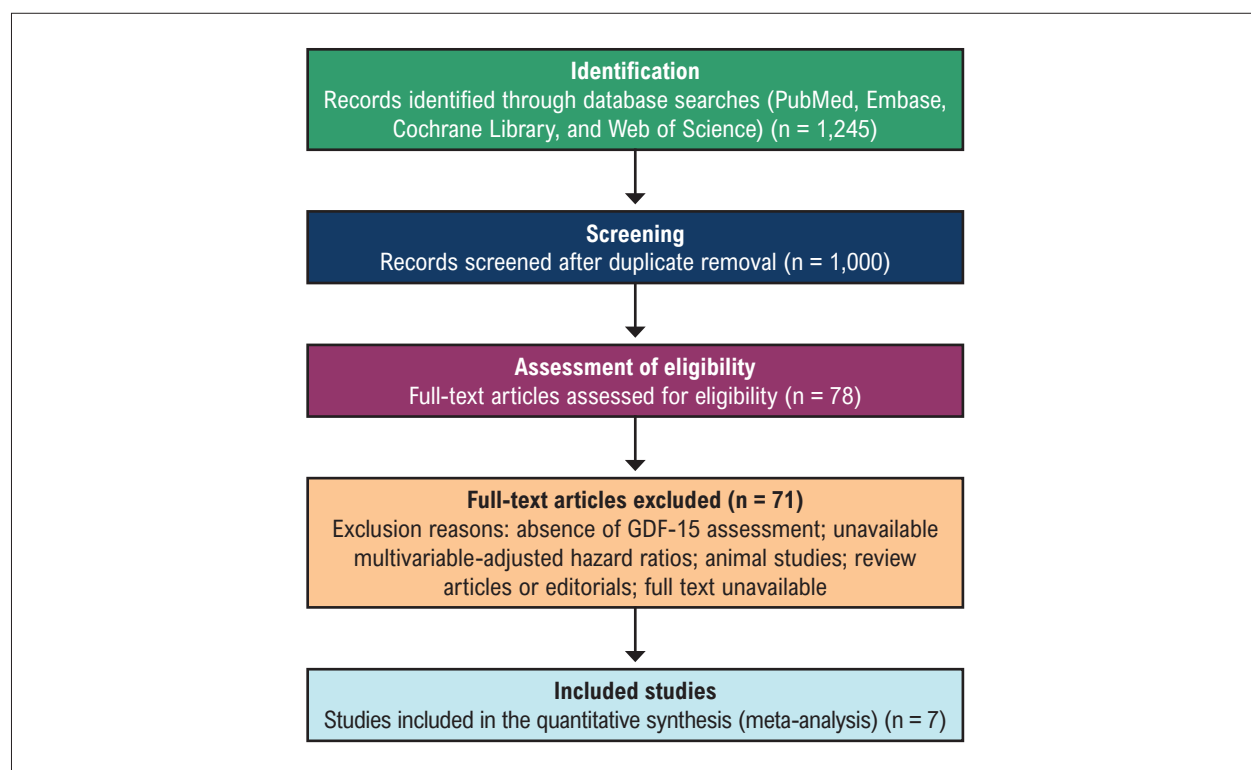


Figure 1 – Flow diagram of study selection process.

of GDF-15 quantification, duration of follow-up, and clinical outcomes, specifically all-cause mortality and HF hospitalization. Adjusted HRs and their corresponding 95% CIs were also collected. When studies reported multiple multivariable-adjusted models, estimates derived from the most comprehensively adjusted model were selected for analysis. Any discrepancies between reviewers were resolved through consensus.

Assessment of quality

Methodological quality was evaluated using the STrengthening the Reporting of OBservational studies in Epidemiology checklist. Assessment domains included transparency of study design, clarity of eligibility criteria, outcome definition, and adequacy of adjustment for potential confounding variables.

Statistical analysis

Meta-analyses were performed using RevMan version 5.4 (Cochrane, London, UK). Adjusted HR estimates were pooled using the DerSimonian and Laird random-effects model because clinical and methodological heterogeneity across studies was anticipated.

Statistical heterogeneity was quantified using the Higgins' I^2 statistic, with values $> 50\%$ considered indicative of substantial heterogeneity. Publication bias was evaluated through visual inspection of funnel plots and Egger's regression test, with $p < 0.10$ considered suggestive of publication bias.

Sensitivity analyses were conducted to assess the robustness of pooled estimates by excluding studies at higher risk of bias or studies using alternative GDF-15 threshold definitions. All statistical tests were two-sided, and $p < 0.05$ was considered statistically significant.

Ethical considerations

This meta-analysis was based exclusively on previously published studies and did not involve direct patient participation or individual-level data collection. Therefore, institutional ethics committee approval was not required.

Results

Study selection

Literature searches retrieved a total of 1,245 records. After the removal of duplicate and screening of titles and abstracts, 78 full-text articles were assessed for eligibility. Following a detailed evaluation, seven studies ($n = 10,578$ patients) that met the predefined inclusion criteria were included in the quantitative synthesis. The study selection process is presented in Figure 1.

Study characteristics

The included studies were published between 2010 and 2025 and had a mean follow-up duration of 3.2 years. The overall mean age of participants was 67 ± 11 years,

and 58% were male. All studies measured baseline plasma or serum levels of GDF-15 using validated enzyme-linked immunosorbent assay techniques or comparable immunoassays. Populations with both HF with reduced ejection fraction (HFrEF) and HF with preserved ejection fraction (HFpEF) were represented across the included studies.

Among the eligible cohorts, the study by Daniels et al.⁶ exclusively enrolled older adults from a community-based cohort, whereas the remaining studies included broader adult populations without upper age restrictions. Table 1 provides a detailed summary of study characteristics, including population features, methods used for quantification of GDF-15, and follow-up duration.

Association between levels of growth differentiation factor-15 and all-cause mortality

Pooled analysis of the seven included studies demonstrated that increased levels of GDF-15 were significantly associated with increased all-cause mortality, with a pooled HR of 2.72 (95% CI, 1.55-4.17; $p < 0.001$) (Figure 2). Statistical heterogeneity was moderate ($I^2 = 49\%$), which indicates some variability across studies although the overall association remained robust. These findings support the independent prognostic value of GDF-15 for predicting long-term mortality in patients with HF.

Association between levels of growth differentiation factor-15 and heart failure hospitalization

Four studies reported data regarding HF-related hospitalization. Pooled analysis demonstrated that higher concentrations of GDF-15 were significantly associated

with an increased risk of HF hospitalization (HR, 2.43; 95% CI, 1.79-3.30; $p < 0.001$) (Figure 3). Statistical heterogeneity was low, which indicates strong consistency across studies.

Subgroup analyses

Subgroup analyses demonstrated that the association between increased levels of GDF-15 and adverse clinical outcomes was consistent across different HF phenotypes, including HFrEF (HR, 2.65; 95% CI, 2.10-3.34) and HFpEF (HR, 2.58; 95% CI, 2.01-3.30). These findings suggest GDF-15 maintains prognostic relevance regardless of left ventricular ejection fraction (LVEF) and support its potential utility as a broadly applicable biomarker across the HF spectrum.

Publication bias and sensitivity analyses

Visual inspection of funnel plots showed no evidence of substantial asymmetry. Egger's regression test ($p = 0.12$) did not indicate significant publication bias, reinforcing the reliability of the pooled estimates. Sensitivity analyses excluding studies at higher risk of bias or studies using alternative GDF-15 threshold definitions yielded consistent findings, which further supports the robustness of the meta-analytic results.

Discussion

This meta-analysis, integrating data from seven studies involving 10,578 patients with HF, demonstrated that increased plasma levels of GDF-15 are independently associated with increased risks of all-cause mortality and

Table 1 – Main characteristics of studies included in the meta-analysis

Study ID	n	Study population	Method for quantification of GDF-15	Mean follow-up duration	Outcomes evaluated
Bonaca et al. (2011)	3,501	ACS	ELISA	2 years	All-cause mortality; recurrent myocardial infarction
Anand et al. (2010)	1,734	Chronic HF	ELISA	4 years	All-cause mortality; HF hospitalization
Daniels et al. (2011)	1,391	Older adults with HF	ELISA	11 years	All-cause mortality
Lok et al. (2013)	209	Advanced HF	ELISA	1 year	All-cause mortality; HF hospitalization
Stahrenberg et al. (2010)	228	HFpEF	ELISA	2 years	All-cause mortality; HF hospitalization
Wang et al. (2012)	3,428	Community-based cohort	ELISA	7 years	Cardiovascular mortality
de Ávila et al. (2024)	87	Chronic HF (HFrEF, HFmrEF, or HFimpEF)	ELISA	11 months	Cardiovascular mortality; HF hospitalization

ACS: acute coronary syndrome; ELISA: enzyme-linked immunosorbent assay; GDF-15: growth differentiation factor-15; HF: heart failure; HFimpEF: HF with improved ejection fraction; HFmrEF: HF with mildly reduced ejection fraction; HFpEF: HF with preserved ejection fraction; HFrEF: HF with reduced ejection fraction.

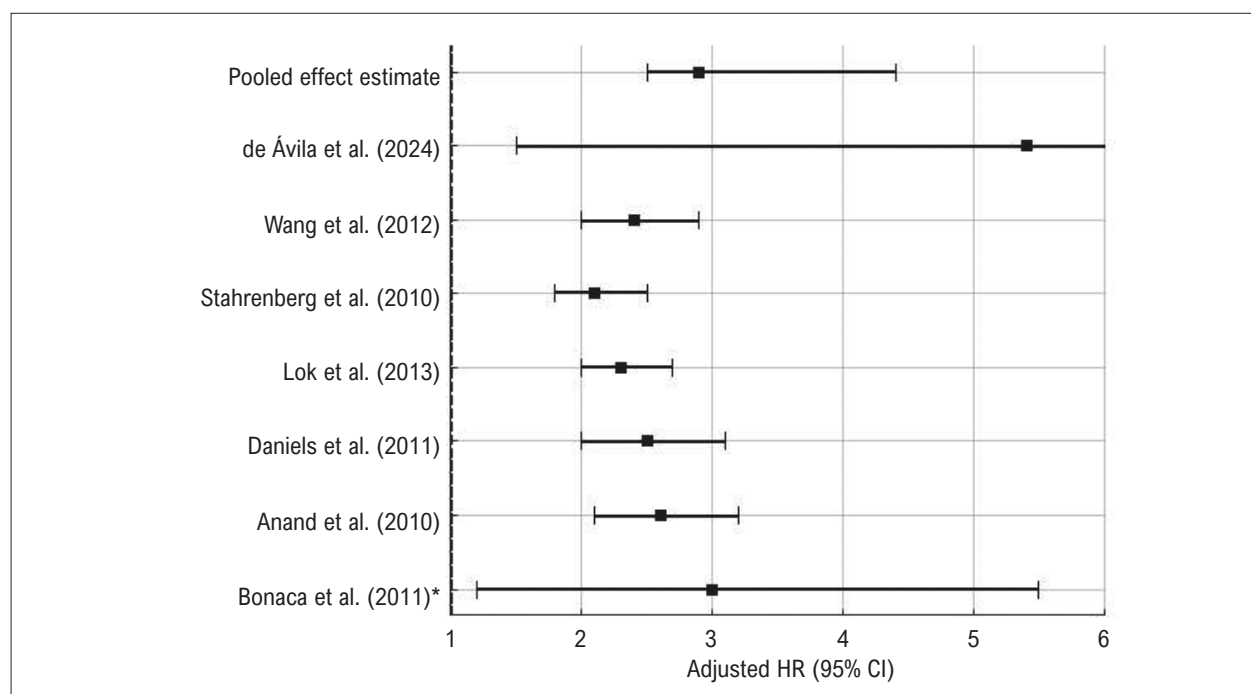


Figure 2 – Association between levels of growth differentiation factor-15 and risk of all-cause mortality in patients with heart failure. HR: hazard ratio.

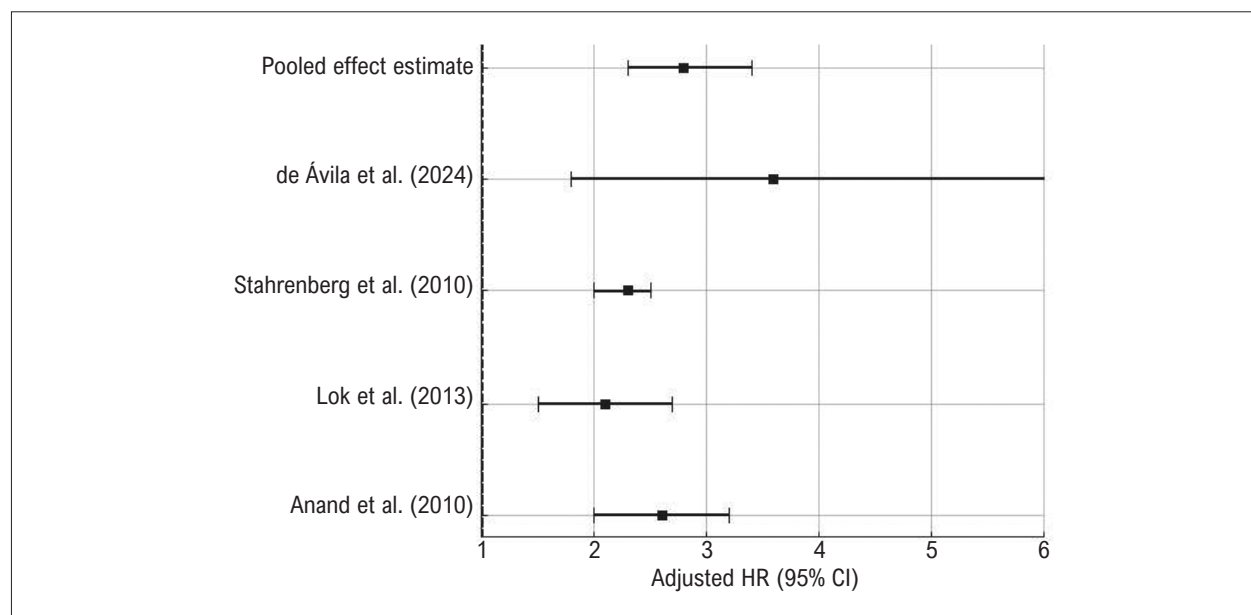


Figure 3 – Association between levels of growth differentiation factor-15 and risk of heart failure hospitalization. HR: hazard ratio.

HF-related hospitalization. These findings strengthen the role of GDF-15 as a prognostic biomarker capable of improving risk stratification beyond conventional markers, such as natriuretic peptides. Central Illustration summarizes the main findings of this meta-analysis, showing the association between increased levels of GDF-15 and higher risks of mortality and HF hospitalization.

Pathophysiological and clinical interpretation

GDF-15 is a pleiotropic cytokine belonging to the TGF- β superfamily, whose expression is induced by myocardial stress, inflammation, oxidative injury, and neurohormonal activation, all of which are central mechanisms involved in HF progression.⁷ Unlike BNP and NT-proBNP, which predominantly reflect hemodynamic stress, GDF-15 integrates

pathways related to metabolism, inflammation, and structural remodeling, potentially providing a broader representation of disease severity and systemic burden.

In the present analysis, the magnitude of the observed association (HR 2.72 for mortality) highlights the clinical relevance of GDF-15 as an independent predictor of adverse outcomes. Importantly, this association remained consistent across HF phenotypes, including HFrEF and HFpEF, supporting the broad prognostic applicability of this biomarker.

Comparison with previous evidence

The present findings are consistent with those reported by Wollert et al.,² who first demonstrated that GDF-15 was a strong independent predictor of cardiovascular mortality and HF hospitalization, even after adjustment for established biomarkers and clinical variables. Similarly, Daniels et al.⁶ evaluated a community-based cohort of older adults (mean age, 70 years) and reported that GDF-15 showed the strongest association with all-cause mortality, outperforming NT-proBNP and troponin I. These findings suggest that GDF-15 may capture biological aging processes not fully reflected by traditional biomarkers, potentially explaining its strong prognostic performance in older populations.

More recently, Ávila et al.⁸ provided prospective evidence in patients with chronic stable HF, demonstrating that increased levels of GDF-15 remained independently associated with cardiovascular death and hospitalization, even after adjustment for NT-proBNP and the Meta-Analysis Global Group in Chronic Heart Failure (MAGGIC) score. These observations reinforce the ability of GDF-15 to complement established prognostic tools and improve the identification of patients at higher risk of adverse clinical outcomes.

Integration into prognostic models

Incorporating GDF-15 into multivariable risk prediction tools, such as the MAGGIC score⁹ and the Seattle Heart Failure Model (SHFM),¹⁰ may improve prognostic discrimination and support more individualized management strategies. These models currently rely primarily on clinical and laboratory variables, including LVEF, kidney function, New York Heart Association functional class, and natriuretic peptides. By reflecting biological pathways extending beyond hemodynamic stress, particularly inflammation and tissue remodeling, GDF-15 may provide incremental prognostic information and identify residual risk even in patients receiving optimized therapy.¹¹

Emerging evidence further supports the integration of GDF-15 into multimarker approaches alongside natriuretic peptides, high-sensitivity cardiac troponins, and galectin-3 to improve risk stratification granularity and optimize follow-up strategies and therapeutic decision-making. In addition, recent studies have demonstrated that GDF-15 retains independent prognostic value across the spectrum of LVEF and may enhance the predictive performance of established models such as the MAGGIC score.¹²⁻¹⁵

Notably, this prognostic relevance also appears applicable to patients with HFpEF, in whom higher circulating levels of GDF-15 independently predicted mortality and HF

hospitalization after adjustment for clinical covariates, reinforcing its value beyond congestion-related and conventional hemodynamic biomarkers.¹⁶

Heterogeneity and methodological considerations

The moderate heterogeneity observed across studies ($I^2 = 49\%$) likely reflects differences in assay methodologies, threshold definitions, and baseline population characteristics, including the burden of comorbidities such as kidney dysfunction and diabetes mellitus. Nevertheless, sensitivity analyses confirmed the stability of pooled estimates, which indicates such variations did not materially influence the overall findings.

An additional limitation is the absence of standardized clinical thresholds for GDF-15 interpretation. Most included studies relied on median-based or quartile-based stratification approaches, highlighting the need for universally accepted cut-off values to facilitate clinical implementation. Furthermore, the limited availability of serial biomarker measurements restricted evaluation of temporal changes in levels of GDF-15 and their potential utility for monitoring treatment response.

Clinical implications

From a clinical standpoint, these findings support the use of GDF-15 as a complementary biomarker for prognostic assessment in HF. Incorporation of GDF-15 into multimarker strategies may improve risk stratification, guide follow-up intensity, and help identify patients who may benefit from advanced therapies or closer clinical surveillance. In addition, by reflecting biological stress and myocardial remodeling, GDF-15 may represent a promising therapeutic target for future translational research.

Conclusion

This systematic review and meta-analysis provide robust evidence supporting GDF-15 as an independent prognostic biomarker in patients with HF. Increased circulating levels of GDF-15 were consistently associated with increased risks of all-cause mortality and HF hospitalization across different ejection fraction phenotypes and clinical settings. These findings reinforce the biological and clinical relevance of GDF-15 as a biomarker integrating inflammation, oxidative stress, and myocardial remodeling, offering a broader assessment of disease burden beyond conventional hemodynamic indicators.

Incorporation of GDF-15 into multimarker prognostic models, such as the MAGGIC score and SHFM, may improve risk stratification accuracy and support a more personalized approach to the management of HF. Such integration may facilitate the identification of patients with residual risk despite guideline-directed medical therapy, potentially informing therapeutic intensification and closer clinical surveillance.

Future studies should aim to i) establish standardized clinical thresholds for GDF-15, ii) evaluate the prognostic relevance of longitudinal changes in levels of GDF-15, iii) determine its incremental predictive value alongside

established biomarkers, and iv) explore its potential role as a therapeutic target in HF progression.

Overall, GDF-15 emerges as a promising biomarker for comprehensive prognostic assessment in HF, bridging molecular pathophysiological mechanisms and individualized clinical decision-making.

Author Contributions

Conception and design of the research, Acquisition of data and Analysis and interpretation of the data: Rimolo LSM, Villacorta H; Statistical analysis and Writing of the manuscript: Rimolo LSM; Critical revision of the manuscript for intellectual content: Villacorta H.

Potential Conflict of Interest

Dr. Humberto Villacorta reports receiving speaker fees and research support from Roche Diagnostics. Dr. Lorena dos Santos Marreto Rimolo reports no conflicts of interest related to this article.

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Study Association

This study is not associated with any thesis or dissertation work.

Ethics Approval and Consent to Participate

This article does not contain any studies with human participants or animals performed by any of the authors.

Use of Artificial Intelligence

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

Availability of Research Data

All datasets supporting the results of this study are available upon request from the corresponding author.



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