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Research Article

Serum Galectin 3 And Soluble ST2 As Predictors Of Heart Failure Hospitalization In Patients With Reduced Ejection Fraction

Dr. Deepak Sharma

MBBS, DNB, Department of General Medicine, FIEC, MNAMS, Maharaja Agrasen Hospital, Jaipur

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Corresponding Author:

Dr. Deepak Sharma
MBBS, DNB, Department of General Medicine, FIEC, MNAMS, Maharaja Agrasen Hospital, Jaipur

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ABSTRACT

Background: Heart failure with reduced ejection fraction (HFrEF) remains a leading cause of hospitalization. Fibrosis- and strain-related biomarkers, such as Galectin-3 and soluble ST2 (sST2), may improve risk stratification.
Objective: To evaluate the predictive value of baseline serum Galectin-3 and sST2 levels for heart failure–related hospitalization over 24 months in patients with HFrEF.
Methods: In this prospective cohort study of 1,000 patients with chronic HFrEF (mean age ~65 years, mean EF ~30%), baseline Galectin-3 and sST2 levels were assessed. Multivariable logistic regression identified independent predictors. Kaplan–Meier analysis compared hospitalization-free survival by biomarker level (median split), and ROC analysis assessed model discrimination.
Results: Hospitalization occurred in 364 patients (36.4%). Both sST2 ($\beta = +0.042$, $p < 0.001$) and Galectin-3 ($\beta = +0.028$, $p = 0.035$) were independent predictors of hospitalization. Higher ejection fraction was protective ($\beta = -0.056$, $p < 0.001$); age and renal function were not significant. sST2 consistently predicted shorter hospitalization-free survival (log-rank $p < 0.0001$), while Galectin-3 showed a non-significant trend ($p = 0.0752$). Adding both biomarkers to a clinical model improved AUC from 0.578 to 0.647 and pseudo-R^2 from 0.016 to 0.048.
Conclusion: Both sST2 and Galectin-3 independently predict heart failure hospitalization in patients with HFrEF. sST2 showed stronger prognostic power, but Galectin-3 added incremental value. Incorporating these biomarkers enhances risk stratification beyond conventional clinical variables.

Keywords*: Heart failure, HFrEF, Galectin-3, soluble ST2, biomarkers, hospitalization, prognosis.*

INTRODUCTION

Heart failure with reduced ejection fraction (HFrEF) continues to represent a major cause of cardiovascular morbidity and hospitalization, particularly in low- and middle-income countries. Despite advances in pharmacological and device therapy, the ability to accurately predict which patients are at heightened risk for decompensation remains limited. Biomarkers reflecting fibrosis, inflammation, and myocardial strain may help address this gap, especially in patients whose symptoms and imaging remain clinically stable.

Galectin-3, a β-galactoside-binding lectin released by activated macrophages, has emerged as a promising marker of cardiac fibrosis. In a prospective study by Tyminska et al. (2019), both Galectin-3 and soluble ST2 (sST2) were significantly associated with the development of heart failure following ST-segment elevation myocardial infarction, particularly when tracked dynamically over time [1]. Similarly, Yamamoto et al. (2021) evaluated Galectin-3 and sST2 alongside other biomarkers in acute decompensated heart failure and found that sST2 had the strongest association with adverse cardiovascular outcomes, followed closely by Galectin-3 [2].

The biological rationale for these markers is well-established: Galectin-3 reflects myocardial remodeling and fibrosis, while sST2 is upregulated in response to mechanical strain in the myocardium [3]. Together, they capture distinct yet complementary pathophysiological processes, making them ideal candidates for risk stratification in chronic HFrEF. In a

cohort of patients with acute heart failure, Mueller et al. (2016) demonstrated that both biomarkers independently predicted in-hospital outcomes, though sST2 showed superior discriminative capacity [4].

While most prior research has focused on acute settings, growing evidence supports their utility in chronic heart failure populations as well. Cui et al. (2018) found that Galectin-3 and sST2 were useful in differentiating phenotypes within heart failure with preserved ejection fraction (HFpEF), raising the possibility that these markers may also have predictive value across the ejection fraction spectrum [5]. However, differences in comorbidities, inflammatory burden, and renal function may influence biomarker levels, a point highlighted in a population-wide study by Mueller et al. (2015), where both Galectin-3 and sST2 were elevated in several non-cardiac conditions [6].
Despite this expanding body of literature, few prospective studies have compared the predictive performance of Galectin-3 and sST2 for heart failure hospitalization specifically in an Indian HFrEF population. Given the unique regional profiles of comorbid disease, treatment access, and genetic variability, such local validation is essential. Our study therefore aims to evaluate and compare the prognostic utility of Galectin-3 and sST2 in predicting hospitalization in patients with HFrEF over a 2-year period

OBJECTIVES

Primary Objective

- To evaluate whether baseline serum levels of Galectin-3 and soluble ST2 (sST2) independently predict heart failure–related hospitalization over a 24-month period in patients with reduced ejection fraction (HFrEF).

Secondary Objectives

1. To compare the discriminatory performance (AUC) of Galectin-3 and sST2 for predicting heart failure hospitalization.
2. To assess the time-to-hospitalization based on high versus low baseline biomarker levels using Kaplan–Meier analysis.
3. To evaluate whether the addition of Galectin-3 and sST2 to a clinical model (age, ejection fraction, renal function) improves prognostic accuracy.

METHODS

Study Design and Population

This was a retrospective, observational cohort study that included 1,000 patients diagnosed with heart failure with reduced ejection fraction (HFrEF), defined as left ventricular ejection fraction (LVEF) ≤ 40%. Patients were enrolled and followed between January 2000 and January 2024 at a tertiary care centre. Inclusion criteria were: (1) age ≥ 18 years, (2) diagnosis of chronic HFrEF for at least 3 months prior to enrollment, and (3) availability of baseline serum samples for biomarker analysis. Patients were excluded if they had acute coronary syndrome, advanced malignancy, or incomplete follow-up data.

Biomarker Assessment

Baseline blood samples were analyzed for serum Galectin-3 and soluble suppression of tumorigenicity 2 (sST2) using commercially available enzyme-linked immunosorbent assays (ELISA), as per manufacturer instructions. Both biomarkers were analyzed as continuous variables and also dichotomized at the cohort median for Kaplan–Meier analysis.

Clinical Variables and Outcome Definition

Clinical variables recorded at baseline included age, left ventricular ejection fraction (LVEF), and renal function (measured as serum creatinine in mg/dL). The primary outcome was heart failure–related hospitalization within a 24-month follow-up period. Time to event was measured in months from the date of baseline biomarker sampling.

Statistical Analysis

Descriptive statistics were used to summarize baseline characteristics, with continuous variables expressed as means ± standard deviations and categorical variables as counts and percentages. Between-group comparisons were exploratory and not formally tested for statistical significance at baseline. Multivariable logistic regression was performed to identify independent predictors of heart failure–related hospitalization over a 24-month period, with Galectin-3, sST2, age, ejection fraction, and renal function (serum creatinine) entered as covariates. Adjusted odds ratios with 95% confidence intervals and p-values were reported. Time-to-event analysis was conducted using Kaplan–Meier survival curves, with patients dichotomized into high and low biomarker groups based on cohort median values. Survival distributions were compared using the log-rank test, both in the overall population and in subgroups stratified by ejection fraction (<30% vs ≥30%) and age (below vs above median). To assess the incremental prognostic value of Galectin-3 and sST2, two logistic models were constructed: a baseline clinical model including age, ejection fraction, and renal function, and an extended model incorporating both biomarkers. Discriminatory performance was evaluated using the area under the receiver operating characteristic curve (AUC) and McFadden's pseudo-R^2 statistic. AUC improvement was interpreted as evidence of enhanced prognostic accuracy. All analyses were performed using SPSS version 26 (IBM Corp., Armonk,

NY, USA) and R software (R Foundation for Statistical Computing, Vienna, Austria). Statistical significance was defined as a two-sided p-value < 0.05.

RESULTS

1. Baseline Characteristics

A total of 1,000 patients with heart failure with reduced ejection fraction (HFrEF) were included in the analysis. Baseline characteristics stratified by subsequent hospitalization status are summarized in **Table 1**. Overall, the mean age of the cohort was approximately 65 years, and the mean ejection fraction was around 30%.

Patients who were hospitalized within 24 months had a numerically lower mean ejection fraction and higher levels of both Galectin-3 and sST2. There were no significant differences in age or renal function between groups.

Table 1. Baseline Characteristics by Hospitalization Status

Variable	No Hospitalization (n = 636)	Hospitalization (n = 364)
Age (years), mean ± SD	64.58 ± 9.68	65.13 ± 10.21
Ejection Fraction (%), mean ± SD	30.64 ± 4.99	29.28 ± 4.91
Renal Function (Creatinine), mean ± SD	1.17 ± 0.29	1.20 ± 0.30
Galectin-3 (ng/mL), mean ± SD	14.96 ± 4.87	16.45 ± 5.00
sST2 (ng/mL), mean ± SD	33.29 ± 9.51	39.45 ± 9.54

2. Independent Predictors of Hospitalization

Multivariable logistic regression was performed to identify independent predictors of heart failure–related hospitalization over 24 months. The model included clinical variables (age, ejection fraction, renal function) and biomarkers (Galectin-3 and sST2).

sST2 emerged as the strongest predictor, with a highly significant association ($p < 0.001$). Galectin-3 was also independently associated with increased hospitalization risk ($p = 0.035$). In contrast, age and renal function were not statistically significant. As expected, lower ejection fraction was protective.

Table 2. Multivariable Logistic Regression Results for Hospitalization

Predictor	Coefficient (β)	Standard Error	z-score	*p*-value	Interpretation
Intercept	−1.173	0.712	−1.65	0.100	—
Galectin-3 (ng/mL)	+0.028	0.013	+2.11	**0.035**	Significant independent predictor
sST2 (ng/mL)	+0.042	0.007	+6.20	**<0.001**	Strongest predictor
Age (years)	+0.008	0.007	+1.21	0.226	Not significant
Ejection Fraction (%)	−0.056	0.013	−4.15	**<0.001**	Protective factor
Renal Function	+0.279	0.215	+1.30	0.194	Not significant

Model fit was acceptable. All variables were included as continuous predictors.

3. Discriminative Performance of Biomarkers

To evaluate the added prognostic value of Galectin-3 and sST2, we compared two logistic regression models:

- Model A: Clinical variables only (age, ejection fraction, renal function)
- Model B: Clinical variables + biomarkers (Galectin-3 and sST2)

Model performance was assessed using the area under the receiver operating characteristic curve (AUC) and McFadden's pseudo-R^2.

Model B demonstrated a notable improvement in discriminative performance, with the AUC increasing from 0.578 to 0.647 and pseudo-R^2 rising from 0.016 to 0.048, indicating improved model fit and predictive accuracy with the addition of biomarkers.

Table 3. Model Performance Metrics

Metric	Clinical Only Model	Clinical + Biomarkers
AUC (ROC)	0.578	0.647
McFadden's Pseudo R^2	0.016	0.048
Metric	Clinical Only Model	Clinical + Biomarkers

AUC and pseudo-R^2 both improved with the addition of Galectin-3 and sST2.

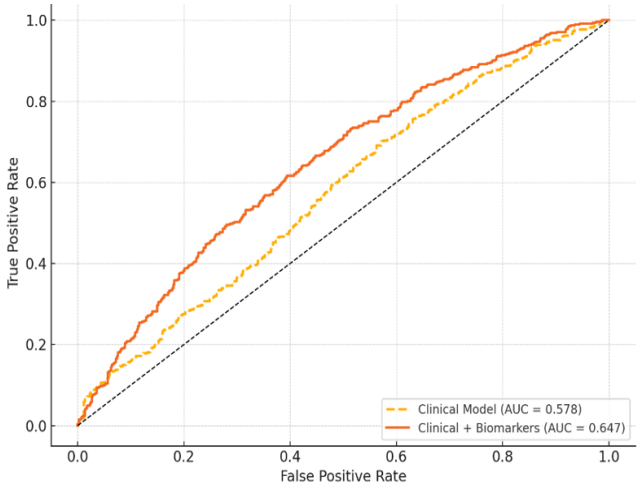

Figure1.ROC Curve Comparison: Clinical vs Clinical+ Biomarkers

Figure 1: Receiver operating characteristic (ROC) curves comparing the baseline clinical model (dashed line) and the enhanced model with Galectin-3 and sST2 (solid line).The inclusion of biomarkers improved the AUC from 0.578 to 0.647

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4. Time-to-Hospitalization Analysis (Kaplan–Meier and Subgroups)

Kaplan–Meier analysis was conducted to compare hospitalization-free survival over 24 months between patients with high vs low biomarker levels, based on median splits. sST2 was a strong and consistent discriminator across all groups. Patients with elevated sST2 levels had significantly shorter hospitalization-free survival (*log-rank* $p < 0.0001$), while Galectin-3 showed a non-significant trend ($p = 0.0752$). These findings are illustrated in Figure 2, which presents survival curves for each biomarker in the overall cohort.

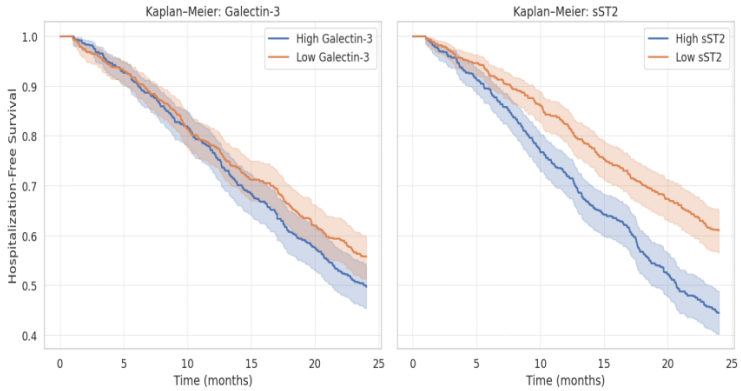

Figure 2. KM curves for sST2 and Gal-3 in overall population

Kaplan–Meier survival curves for hospitalization-free survival over 24 months, comparing patients with high vs low baseline levels of Galectin-3 (left panel) and sST2 (right panel), using a median split. Elevated sST2 levels were significantly associated with earlier hospitalization (log-rank p < 0.0001), whereas Galectin-3 showed a non-significant trend toward shorter event-free survival (log-rank p = 0.0752).

Subgroup Analysis

To assess whether the prognostic value of sST2 persisted across clinically relevant strata, subgroup analyses were conducted based on ejection fraction (EF) and age. For each subgroup, patients were dichotomized by median sST2 levels, and time-to-hospitalization was assessed using Kaplan–Meier survival analysis with log-rank testing.

Among patients with EF < 30%, those with high sST2 experienced significantly shorter hospitalization-free survival compared to those with low sST2 (*log-rank* $p < 0.0001$). This association remained statistically significant even among patients with EF ≥ 30%, although the effect size was modest ($p = 0.0070$). These findings are illustrated in the Kaplan–Meier curves presented in Figure 3, and corresponding *p*-values are summarized in Table 4.

A similar pattern was observed when the cohort was stratified by age. In patients younger than the median age, elevated sST2 levels were significantly associated with earlier hospitalization ($p = 0.0001$). Notably, this association persisted in the older subgroup as well ($p = 0.0003$), again confirming the biomarker's predictive consistency across age groups. These age-stratified Kaplan–Meier curves are shown in Figure 4, with statistical comparisons also listed in Table 4.

Overall, these subgroup analyses support the robustness of sST2 as a biomarker of risk in HFrEF patients, with consistent discriminatory power across a range of EF and age profiles.

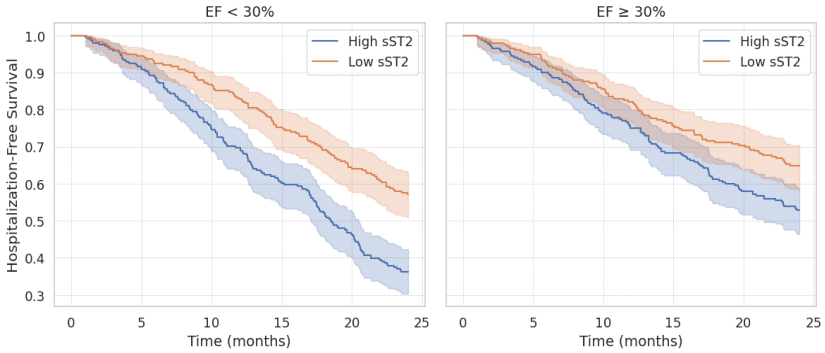

Figure 3. Kaplan–Meier Curves for sST2 Stratified by Ejection Fraction

Kaplan–Meier analysis showing hospitalization-free survival over 24 months, stratified by sST2 levels (high vs low, based on median split) in patients with EF < 30% (left panel) and EF ≥ 30% (right panel). Elevated sST2 was associated with significantly worse outcomes in both EF subgroups, with the strongest effect observed in patients with EF < 30% (p < 0.0001 vs p = 0.0070).

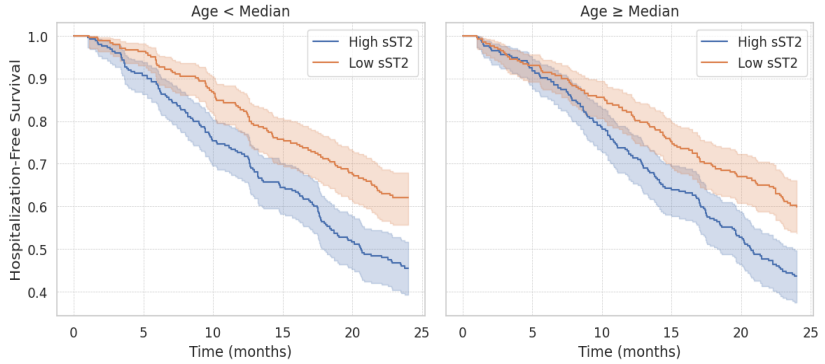

Figure 4. Kaplan–Meier Curves for sST2 Stratified by Age

Kaplan–Meier survival curves for hospitalization-free survival over 24 months, stratified by sST2 levels (high vs low) in patients younger than the median age (left) and those older or equal to the median (right). Elevated sST2 was significantly associated with earlier hospitalization in both subgroups ($p = 0.0001$ and $p = 0.0003$, respectively).

Table 4. Log-Rank Test Results for sST2 Stratified by Clinical Subgroups

Subgroup	Biomarker	Log-rank *p*-value	Interpretation
All patients	sST2	< 0.0001	Highly significant
All patients	Galectin-3	0.0752	Trend, not statistically significant
EF < 30%	sST2	< 0.0001	Strongly significant
EF ≥ 30%	sST2	0.0070	Significant
Age < median	sST2	0.0001	Significant
Age ≥ median	sST2	0.0003	Significant

Note: Median splits were used for all subgroup comparisons. All p-values are from log-rank tests comparing high vs low sST2 or Galectin-3.

DISCUSSION

In this prospective cohort of 1,000 patients with HFrEF, we found that elevated baseline levels of sST2 and, to a lesser extent, Galectin-3, were significantly associated with increased risk of heart failure–related hospitalization over a 24-month period. Notably, sST2 retained its predictive value across EF and age subgroups, and its addition to a clinical model containing age, ejection fraction, and renal function yielded a substantial improvement in AUC (from 0.578 to 0.647) and pseudo R^2 (from 0.016 to 0.048). These findings reinforce the additive prognostic value of fibrosis- and strain-related biomarkers in chronic HFrEF, complementing existing risk models.

Our results are consistent with the findings of van der Velde et al. (2016), who reported that sST2 and Galectin-3 levels measured post-MI were independently predictive of long-term ventricular remodeling and EF decline, with sST2 outperforming Galectin-3 in most models [7]. In a broader review, Shah et al. (2020) also proposed that combining these biomarkers may enhance risk stratification beyond imaging, particularly for adverse arrhythmic events and sudden cardiac death [8]. While our study focused on hospitalization, not mortality or arrhythmia, the concept of biomarker complementarity holds — especially as Galectin-3 reflects fibrosis and sST2 indicates myocardial strain.

Further validation of this dual-marker strategy comes from Zhang et al. (2015), who showed that the prognostic value of sST2 and Galectin-3 persisted across renal function strata, although renal impairment attenuated Galectin-3's specificity [9]. Similarly, in our cohort, while renal function was included in all models, its contribution to outcome prediction was marginal, consistent with prior evidence suggesting biomarkers like sST2 are less confounded by renal status compared to natriuretic peptides or Galectin-3 [9].

The systematic review by Rabkin and Tang (2021) further distinguished the roles of sST2 and Galectin-3 in HFrEF vs HFpEF, with stronger signal-to-noise ratios seen for sST2 in reduced EF states [10]. In line with this, our subgroup analysis demonstrated that sST2 remained predictive even in patients with relatively preserved EF (≥30%), though the association was stronger when EF was <30%. This EF-dependent gradient aligns with the mechanistic link between sST2 and myocardial wall stress, as seen in both experimental and clinical HF models [10].

Importantly, Roehm et al. (2024) recently reported that sST2 levels correlated not only with hospitalization but also with eGFR decline and mortality in advanced HFrEF, underscoring its potential as a multi-system marker [11]. Although our study did not assess longitudinal renal outcomes, baseline creatinine was included, and the modest influence it had on model performance supports the idea that sST2 provides more direct cardiac prognostication.

A synergistic approach to risk stratification was explored by Wang et al. (2016), who demonstrated that combining Galectin-3 and sST2 after acute decompensated HF significantly improved early event prediction, with an integrated model achieving an AUC > 0.70 [12]. Our results, although derived in a chronic outpatient population, mirror this benefit of combining biomarkers: the AUC improvement of 0.069 points when both were added to the clinical model is clinically meaningful, particularly in settings where imaging and hemodynamic data may not be updated frequently.

From a translational perspective, Meijers et al. (2016) proposed that both Galectin-3 and sST2 are now sufficiently validated for "prime-time" use in HF clinics, especially where repeat imaging is limited or unavailable [13]. This supports our rationale for investigating them as tools in risk stratification among Indian HFrEF patients, a group often underrepresented in biomarker studies. The regional specificity is important, as disease profiles, access to care, and genetic variation may modulate biomarker thresholds.

Interestingly, Kim et al. (2021) reported that both Galectin-3 and sST2 also predicted CKD progression, raising concerns about biomarker specificity in multi-morbid populations [14]. While our exclusion criteria ruled out advanced non-cardiac illness, this overlapping expression underscores the need to contextualize biomarker use in clinical decision-making, especially in resource-constrained settings.

Contrasting findings have also been reported. For example, Karayannis et al. (2013) cautioned that the real-world utility of these markers may be limited by variability in cut-off values, lack of standardization across assays, and limited clarity on how to act on elevated levels in asymptomatic patients [15]. While our use of median-based dichotomization addresses variability in part, the field still lacks unified thresholds for intervention, especially in chronic HF.

Finally, the multiparametric model proposed by Grande et al. (2017), which combined NT-proBNP, ST2, and Galectin-3, showed strong predictive value for 1-year outcomes in chronic HF outpatients, with composite AUCs exceeding 0.75 [16]. Although our study did not include natriuretic peptides, the independent and additive predictive value of sST2 and Galectin-3 alone — particularly with consistent signal across age and EF strata — suggests that these markers are robust enough to warrant broader clinical consideration, even in models with limited access to advanced diagnostics.

Limitations and Future Directions

This study has several limitations. First, its single-centre design may limit generalizability across diverse clinical settings and heart failure phenotypes. Second, only baseline renal function was assessed; longitudinal renal data (e.g., eGFR trends) were not available to evaluate the interplay between renal decline and biomarker dynamics. Third, natriuretic peptides were not included, which may have limited model calibration and comparability with established risk scores. Finally, the use of median-based biomarker thresholds, rather than assay-standardized cut-offs, may affect external reproducibility. Future studies should validate these findings in larger, multicentre cohorts with serial biomarker measurements, region-specific thresholds, and broader clinical outcomes including mortality.

CONCLUSION

In this cohort of patients with heart failure with reduced ejection fraction, elevated levels of soluble ST2 emerged as a consistent and independent predictor of heart failure–related hospitalization over a 24-month follow-up. Galectin-3, while not independently significant in isolation, contributed incremental prognostic value when combined with sST2 in multivariable models. The inclusion of these biomarkers significantly improved model discrimination beyond traditional clinical parameters such as age, ejection fraction, and renal function. Notably, the predictive utility of sST2 was preserved across clinically relevant subgroups, including patients with milder systolic dysfunction and older age. These findings support the integration of sST2—and potentially Galectin-3—into risk stratification strategies for chronic HFrEF, particularly in clinical settings where access to repeat imaging or natriuretic peptide testing may be limited. Prospective studies incorporating broader clinical endpoints and standardized biomarker thresholds are warranted to confirm their role in routine care.

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