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Original Article

Predictive value of baseline serum sST2 and BNP levels for treatment efficacy in patients with heart failure

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Abstract: Objectives: To evaluate the association between baseline serum soluble stimulator gene 2 (sST2) level, B-type natriuretic peptide (BNP) level and treatment outcomes in Heart failure (HF) patients, and to assess their predictive value. Methods: A retrospective case-control study was conducted on 162 HF patients treated at Longgang People's Hospital from August 2021 to July 2023. Patients were categorized into effective (n=138) and ineffective (n=24) groups based on New York Heart Association (NYHA) functional classification post-treatment. Serum sST2 and BNP levels were measured using enzyme-linked immunosorbent assay, and cardiac function parameters, including left ventricular ejection fraction (LVEF) were assessed via echocardiography. Results: The overall treatment effectiveness rate was 85.19%. The serum soluble ST2 and BNP levels in the ineffective group were significantly higher compared to the effective group ($P<0.05$). Additionally, LVEF was significantly lower, while the left ventricular end-systolic dimension (LVESD) was significantly greater in the ineffective group than in the effective group. Both serum sST2 and BNP were identified as independent risk factors for ineffective treatment. A combined predictive model incorporating soluble ST2 and BNP achieved an AUC of 0.929, demonstrating excellent predictive performance. Conclusion: Baseline serum sST2 and BNP levels are independent predictors of treatment response in HF patients. Their combined application enhances predictive performance and may assist clinicians in tailoring treatment strategies to improve patient survival quality and prognosis.

Keywords: Heart failure, growth stimulator gene 2, B-type natriuretic peptide, predictive value, treatment efficacy

Introduction

Heart failure (HF) is a common clinical cardiovascular disease characterized by a group of syndromes resulting from impaired ejection and abnormal systolic function, leading to insufficient perfusion of organs and tissues. Its main clinical manifestations include reduced exercise tolerance, dyspnea and edema [1-3]. Statistics have revealed that HF accounts for approximately 20% of all cardiovascular-related deaths annually [4], and prognosis remains poor, with a 5-year survival rate of only about 50% [5, 6]. Early prognostic assessment is essential for guiding individualized treatment plans for HF patients [7].

Soluble growth stimulator gene 2 (sST2) acts as a decoy receptor for interleukin-33 (IL-33) and plays a role in atherosclerosis, vascular inflammation, and myocardial stress responses. Its serum level has high sensitivity to cardiomyocyte stress and has been proposed as a promising biomarker for HF diagnosis [8, 9]. B-type natriuretic peptide (BNP), a well-established marker of cardiac dysfunction, is widely used in the diagnosis, monitoring, and management of HF, and is valuable for risk assessment, differential diagnosis, risk stratification, and prognosis prediction [10-12].

This study aimed to investigate the association of sST2 and BNP levels with treatment outcomes in HF patients, assess their predictive value, and construct a combined prediction model to support clinical decision-making.

Methods

Patient selection

This retrospective case-control study included 162 HF patients who were treated at Longgang

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Table 1. Treatment efficacy in heart failure patients

Efficacy	N (n=162)	Percentage
Significant effective	100	61.73%
Effective	38	23.46%
Ineffective	24	14.81%
Total effective rate	138	85.19%

People's Hospital between August 2021 to July 2023. This study was approved by the Medical Ethics Committee of Longgang People's Hospital.

Inclusion criteria: 1) Diagnosis of heart failure [13] based on echocardiographic findings combined with clinical symptoms and signs; 2) Heart function II-IV grade; 3) Age ≥18 years; 4) Complete clinical data; 5) Normal liver and kidney functions. Exclusion criteria: 1) Presence with pericardial effusion, constrictive pericarditis or other heart diseases; 2) Concurrent acute or chronic systemic infections; 3) Non-cardiogenic dyspnea, pulmonary embolism, autoimmune diseases, or cardiogenic shock; 4) Malignant tumor; 5) Mental disorder or other severe comorbidities; 6) Known allergy to any medications used in this study.

Routine anti-heart failure treatment

All patients received standard pharmacological treatment for heart failure. Digoxin (cardiotonic agent, 0.25 mg/tablet, National Drug Approval No. H31021074, Hangzhou Sanofi Anwante Minsheng Pharmaceutical Co., Ltd., China) was administered orally at 0.25 mg three times daily for 7 days, followed by maintenance dosing of 0.125-0.5 mg/d. Spironolactone (diuretic, 20 mg/tablet, National Drug Approval No. H44023416, Guangzhou Kanghe Pharmaceutical Co., Ltd., China) was given at 20-40 mg/time. Dosage was adjusted every 6-8 h according to diuretic response, then maintained at 20-40 mg once every other day. Metoprolol (β-receptor blocker, 50 mg/tablet, National Drug Approval No. H32025390, AstraZeneca Pharmaceutical Co., Ltd., China) was administered at 50 mg once a day. The treatment duration was 1 month.

Grouping criteria

The New York Heart Association's (NYHA) functional classification was used to evaluate cardiac function in all patients. Grade I: no limitation of physical activity, and normal physical activity does not cause symptoms such as dyspnea, palpitation, or angina; Grade II: slight limitation of physical activity, with symptoms occurring during physical activity; Grade III: marked limitation of physical activity, with symptoms occurring with less-than-normal physical activity; Grade IV: symptoms present even at rest. Treatment efficacy was defined as follows: an improvement of ≥2 NYHA classes was considered significantly effective; improvement of one class was defined effective; and improvement of less than one class or worsening of symptoms was defined as ineffective. Based on post-treatment NYHA classification, patients were categorized into two groups: the effective group (significantly effective + effective) (n=138) and the ineffective group (n=24).

Blood test

Four milliliters of fasting peripheral venous blood were collected from patients in the morning. The samples were centrifuged at 3000 rpm for 15 min to obtain the supernatant serum. Hemoglobin and epidermal growth factor receptor (eGFR) levels were measured using an automated biochemical analyzer (BS-280, Mindray, China). Serum sST2 (ab254505, Abcam, UK) and BNP (ab193694, Abcam, UK) levels were measured using enzyme-linked immunosorbent assay (ELISA) according to the manufacturer's instructions.

Echocardiography

Color Doppler echocardiography (Vivid E95, GE, USA) was performed using a 3.0 MHz probe prior to treatment. Patients were positioned in the left lateral position. The apical four-chamber view was used for pulsed Doppler measurements of left ventricular ejection fraction (LVEF). The parasternal long-axis view was used to measure left ventricular end-diastolic dimension (LVEDD), left ventricular end-systolic dimension (LVESD), left ventricular end-diastolic volume (LVEDV), and left ventricular end-systolic volume (LVESV).

Statistical analysis

Statistical analysis was performed using SPSS 29.0 (SPSS Inc., Chicago, IL, USA). Categorical

Table 2. Comparison of baseline data between the effective and ineffective groups

Parameters	Effective group (n=138)	Ineffective group (n=24)	t/χ^2	P
Gender (Male, %)	72 (52.17%)	11 (45.83%)	0.329	0.566
Age (years)	62.53 ± 9.67	61.83 ± 10.49	0.324	0.746
Body mass index (kg/m^2)	25.88 ± 2.26	26.32 ± 2.17	0.878	0.381
Duration of disease (years)	2.68 ± 0.65	2.43 ± 0.53	1.724	0.087
Previous myocardial infarction [n (%)]	44 (31.88%)	7 (29.17%)	0.070	0.791
Smoking history [n (%)]	41 (29.71%)	6 (25.00%)	0.220	0.639
Alcohol consumption history [n (%)]	60 (43.48%)	9 (37.50%)	0.299	0.585
Hypertension [n (%)]	81 (58.70%)	16 (66.67%)	0.541	0.462
Diabetes [n (%)]	19 (13.77%)	4 (16.67%)	0.003	0.953
Coronary heart disease [n (%)]	32 (23.19%)	5 (20.83%)	0.064	0.800
Stroke [n (%)]	18 (13.04%)	2 (8.33%)	0.097	0.756
Atrial fibrillation	43 (31.16%)	8 (33.33%)	0.045	0.832
Cardiac function classification [n (%)]			0.452	0.798
Grade II	25 (18.12%)	3 (12.50%)		
Grade III	81 (58.70%)	15 (62.50%)		
Grade IV	32 (23.19%)	6 (25.00%)		
Heart rate (beats/min)	81.79 ± 17.36	82.24 ± 16.53	0.118	0.906
SBP (mmHg)	129.25 ± 18.14	131.68 ± 18.24	0.605	0.546
DBP (mmHg)	72.15 ± 10.26	74.34 ± 10.48	0.960	0.338

Note: SBP: Systolic Blood Pressure; DBP: Diastolic Blood Pressure.

variables were presented as [n (%)]. For group comparisons, the chi-square test was applied when the sample size was ≥40 and the theoretical frequency (T) was ≥5. When 1≤T<5, the continuity-corrected chi-square test was used. For sample sizes <40 or T<1, Fisher's exact test was employed. The Shapiro-Wilk test was used to assess the normality of continuous variables. Normally distributed data were expressed as (Mean ± SD) and compared using the t-test with corrected variance. Non-normally distributed data were presented as median (25th percentile, 75th percentile) and analyzed using the Wilcoxon rank-sum test. A two-tailed P<0.05 was considered statistically significant.

Spearman correlation analysis was performed to evaluate associations between treatment outcomes and clinical indicators. Variables that were significant in both group comparisons and correlation analysis were included as covariates in logistic regression analysis. The predictive performance of each variable for treatment outcome was assessed by calculating the area under the receiver operating characteristic (ROC) curve (AUC).

Results

Treatment efficacy evaluation in HF patients

Among the 162 HF patients who received therapy, the total effective rate was 85.19% (138/162) (**Table 1**). Specifically, 61.73% (100/162) of the patients demonstrated a significant therapeutic response, while 23.46% (38/162) were classified as effective response. A total of 14.81% (24/162) of patients were categorized as having an ineffective response. The high total effective rate suggests that a substantial proportion of patients experienced marked clinical improvement following standard therapy.

Comparison of baseline characteristics between effective and ineffective groups

There were no statistically significant differences between the effective and ineffective groups in terms of sex, age, body mass index, duration of disease, history of smoking or alcohol consumption, comorbidities, or baseline cardiac function classification (all *P*>0.05) (**Table 2**). This indicates that the observed differences in treatment efficacy were unlikely to be influenced by these baseline factors.

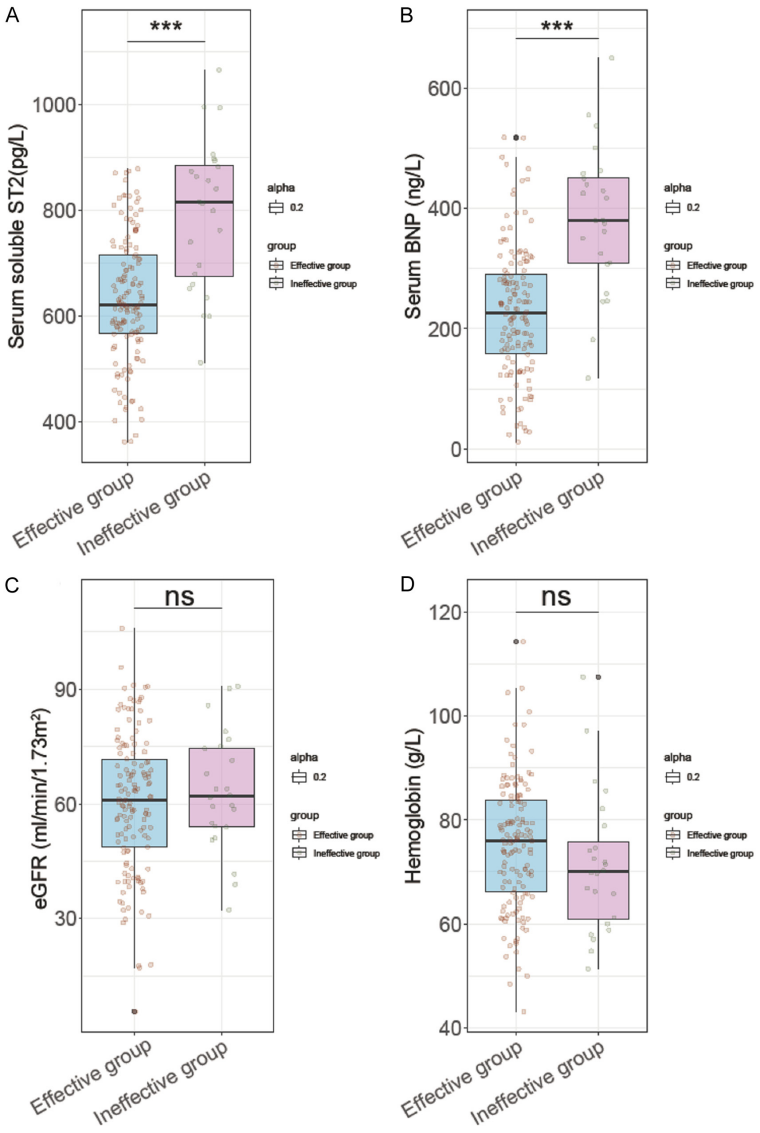

Figure 1. Comparison of blood parameters between the effective and ineffective groups. A. Serum soluble ST2; B. Serum BNP; C. eGFR; D. Hemoglobin. Note: BNP: B-type natriuretic peptide; ST2: growth stimulator gene 2; eGFR: epidermal growth factor receptor. ns: no significant difference; ***: P<0.001.

Comparison of blood parameters between effective and ineffective groups

Comparison of blood test parameters between the effective and ineffective groups revealed that serum sST2 and BNP levels were significantly elevated in the ineffective group, whereas eGFR and hemoglobin levels showed no significant difference (**Figure 1**). Specifically, serum sST2 levels were significantly higher in the ineffective group compared to the effective group (793.26 ± 141.38 pg/L vs. 632.15 ± 120.23 pg/L; t=5.899, P< 0.001). Similarly, serum BNP levels were significantly elevated in the ineffective group (381.72 ± 122.23 ng/L vs. 228.63 ± 108.94 ng/L; t= 6.239, P<0.001). These results highlight that elevated baseline levels of sST2 and BNP may reflect more severe pathophysiological derangements and are associated with poorer treatment response, supporting their role as potential predictors of therapeutic efficacy in HF patients.

Comparison of echocardiography parameters between effective and ineffective groups

Comparison of echocardiography parameters between the effective and ineffective groups revealed significant differences in LVEF and LVESD, while no significant differences were found in LVEDD, LVEDV, and LVESV (**Figure 2**). Specifically, LVEF was significantly higher in the effective group compared to the ineffective group (43.04 ± 5.12% vs. 39.15 ± 4.97%; t=3.457, P<0.001). Conversely, LVESD was significantly greater in the ineffective group (3.67 ± 0.54 cm vs. 3.29 ± 0.68 cm; t= 2.563, P=0.011). These findings highlight the relevance of preserved systolic function and reduced ventricular remodeling as key determinants of favorable treatment response in HF patients.

Correlation between parameters and treatment efficacy

Correlation analysis identified significant correlations between treatment outcomes and key clinical parameters, including serum sST2, serum BNP, LVEF, and LVESD (**Table 3**). Specifically, serum sST2 was positively correlated with treatment inefficacy (rho=0.376, P<0.001),

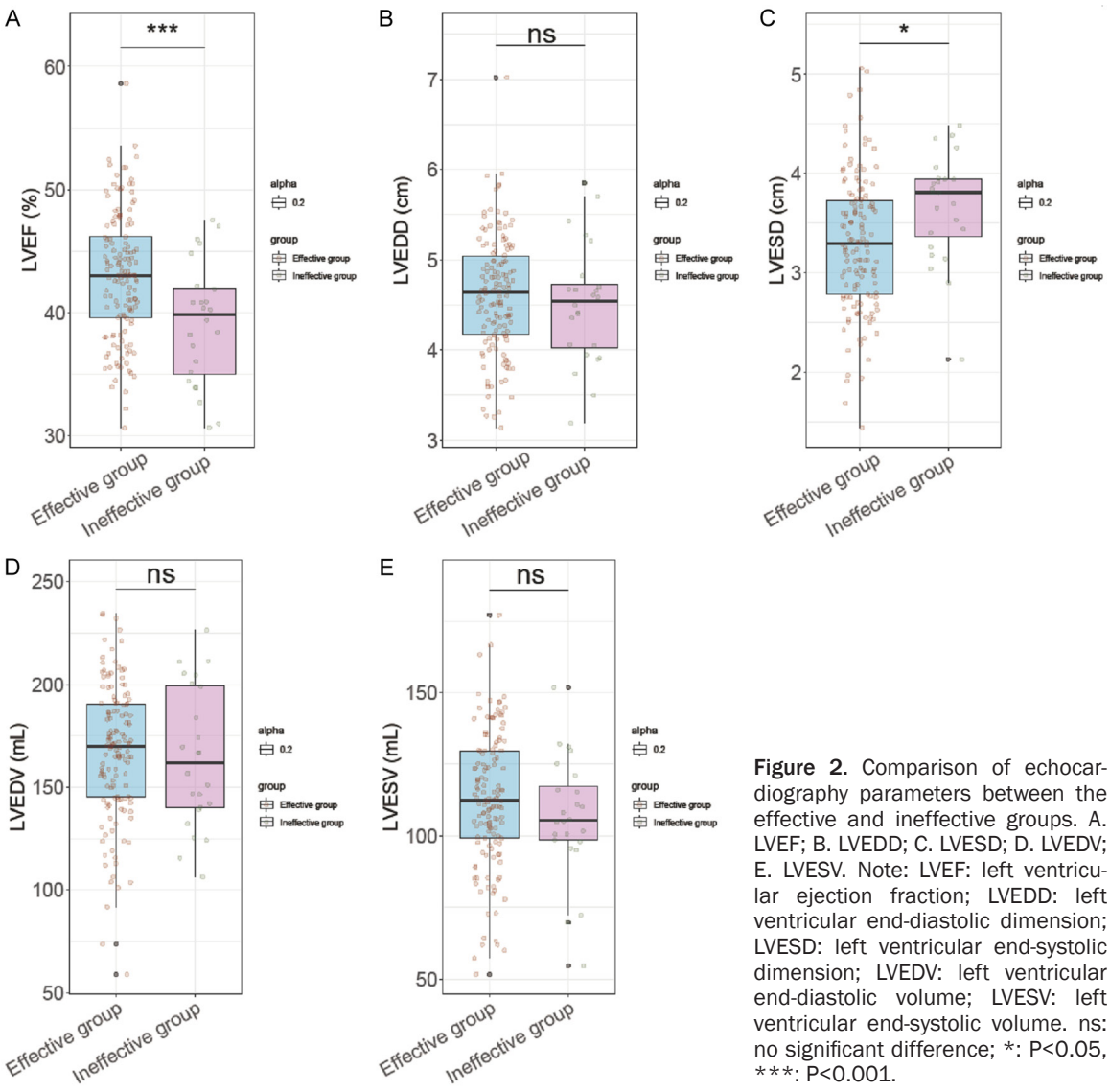

Figure 2. Comparison of echocardiography parameters between the effective and ineffective groups. A. LVEF; B. LVEDD; C. LVESD; D. LVEDV; E. LVESV. Note: LVEF: left ventricular ejection fraction; LVEDD: left ventricular end-diastolic dimension; LVESD: left ventricular end-systolic dimension; LVEDV: left ventricular end-diastolic volume; LVESV: left ventricular end-systolic volume. ns: no significant difference; *: P<0.05, ***: P<0.001.

Table 3. Correlation between LVEF, BNP, serum soluble ST2 levels and treatment inefficacy in heart failure patients

Parameters	rho	*P*
Serum soluble ST2 (pg/L)	0.376	P<0.001
Serum BNP (ng/L)	0.401	P<0.001
LVEF (%)	-0.251	0.001
LVESD (cm)	0.230	0.003

Note: BNP: B-type natriuretic peptide; ST2: growth stimulator gene 2; LVEF: left ventricular ejection fraction; LVESD: left ventricular end-systolic dimension.

indicating that elevated sST2 levels are associated with poorer therapeutic response. Similarly, serum BNP levels were strongly and positively correlated with treatment inefficacy (rho=0.401, P<0.001), reinforcing its role as a prognostic biomarker. LVEF demonstrated a negative correlation (rho=-0.251, P=0.001), while LVESD showed a positive correlation (rho=0.230, P=0.003) with treatment inefficacy. These findings emphasize the predictive value of elevated sST2 and BNP levels, reduced LVEF, and increased LVESD in identifying patients at a higher risk of poor therapeutic response.

Identification of serum sST2 and BNP as independent risk factors for treatment inefficacy

Given the high correlation between LVEF and LVESD and their higher *P*-values, they were excluded from the forward stepwise regression

Table 4. Logistic regression analysis of variables affecting treatment effectiveness in heart failure patients

Parameters	SE	Wald	*P* value	OR	95% CI
Serum soluble ST2	0.654	3.758	<0.001	11.663	3.239-41.997
Serum BNP	0.645	3.836	<0.001	11.862	3.352-41.969

Note: BNP: B-type natriuretic peptide; ST2: growth stimulator gene 2.

Table 5. Predictive value of serum soluble ST2 and BNP for treatment outcome in patients with heart failure

Parameters	Best threshold	Sensitivities	Specificities	AUC	Youden index
Serum soluble ST2	797.530	0.583	0.891	0.806	0.474
Serum BNP	302.255	0.792	0.768	0.826	0.560

Note: BNP: B-type natriuretic peptide; ST2: growth stimulator gene 2.

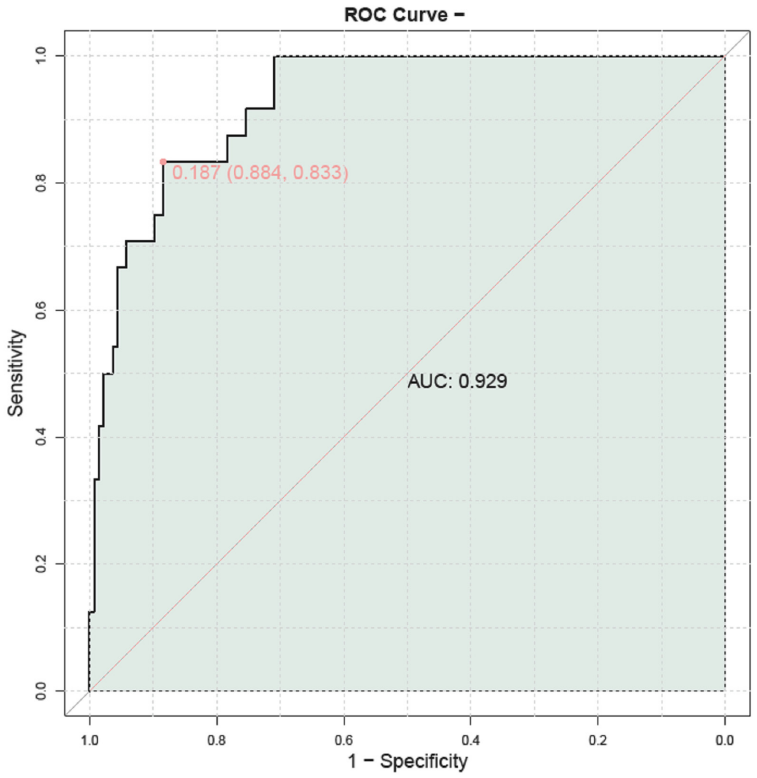

Figure 3. ROC curve of the joint predictive model for treatment effectiveness. Note: ROC: Receiver Operating Characteristic Curve.

analysis. The analysis identified serum sST2 and BNP as the most influential variables associated with treatment effectiveness. Both serum sST2 (OR=11.663, 95% CI: 3.239-41.997) and serum BNP (OR=11.862, 95% CI: 3.352-41.969) were independently and positively correlated with treatment inefficacy (**Table 4**), suggesting their potential utility as significant risk factors for predicting treatment effectiveness in HF patients.

Superior predictive performance of serum BNP

Both serum sST2 and BNP exhibited strong predictive values for treatment efficacy in HF patients (**Table 5**). Serum BNP showed slightly superior performance in terms of sensitivity, specificity, AUC, and Youden index. The higher sensitivity of serum BNP makes it particularly suitable for identifying patients at risk of poor outcomes, while the higher specificity of serum sST2 enhances its utility in confirming favorable treatment outcomes. These biomarkers can be used either individually or in combination to guide clinical decision-making and tailor individualized therapeutic strategies.

Combined predictive model enhanced efficacy prediction

A combined predictive model integrating serum sST2 and BNP was developed to improve assessment of treatment efficacy in heart failure. This model demonstrated a high predictive value, with an AUC of 0.929 (**Figure 3**), indicating its high accuracy in identifying patients at risk of ineffective treatment and supporting its potential for clinical application.

Discussion

In recent years, the incidence of heart failure has continued to rise annually, primarily due to myocardial damage, cardiac overload, and impaired diastolic function [14]. Serum soluble ST2 and BNP are biomarkers of myocardial

stress that reflect alterations in cardiac pressure and volume load [15, 16].

In heart failure, ischemia and hypoxia-induced myocardial necrosis promote extracellular matrix fibrosis, resulting in loss of myocardial contractility. This leads to expansion of ischemic regions, compensatory overload in non-ischemic areas, progressive ventricular dilation, ventricular remodeling, abnormal ventricular wall motion, and decreased LVEF [17, 18]. While LVEF is a valuable index for assessing myocardial injury and cardiac function, it does not fully capture the complexity of heart failure pathophysiology. Combining LVEF with additional biomarkers may enhance predictive accuracy and clinical utility.

Transmembrane ST2 binds its functional ligand IL-33 and exerts cardioprotective effects [19]. In this study, serum sST2 levels were significantly increased in the ineffective group compared to the effective group, indicating an association between elevated sST2 and poor treatment response. The potential mechanisms may be as follows: (1) Mechanical stress in HF upregulates the expression of sST2, which correlates with disease severity. As a decoy receptor, sST2 antagonizes the expression of related inflammatory factors, induces myocardial fibrosis and hypertrophy, and contributes to disease progression. Thus, higher serum sST2 levels reflect greater pathological burden and are associated with poorer cardiac outcomes [20, 21]. (2) The IL-33/transmembrane ST2 signaling axis functions as a mechanical stress-responsive cardioprotective pathway, exerting anti-atherosclerotic, anti-hypertrophic, and anti-fibrotic effects. Soluble ST2 competes with transmembrane ST2 by binding IL-33, thereby inhibiting the IL-33/ST2 signaling cascade, neutralizing its protective role, and potentially impairing treatment efficacy [22, 23].

BNP is a biologically active polypeptide hormone encoded by the BNP gene at the distal end of the short arm of chromosome 1. It is a key cardiovascular neurohormone. Under conditions of increased ventricular volume and elevated filling pressure, BNP level increases sharply [24-26]. Research has demonstrated a strong correlation between BNP and cardiac function in patients with chronic heart failure, suggesting its potential utility in assessing disease severity and guiding treatment decisions [27]. In this study, the serum BNP level was significantly higher in the ineffective group compared to the effective group, consistent with previous findings. In response to impaired left ventricular function, including abnormal diastolic function and systolic function, cardiomyocytes rapidly synthesize and release BNP into the circulation to modulate cardiac function. BNP exerts multiple physiological effects, including inhibition of myocardial fibrosis, reduction of vascular tone, promotion of diuresis, and suppression of sodium reabsorption in organs such as heart and kidney. These effects collectively contribute to vasodilation, blood pressure reduction, and decreased ventricular preload [28]. Furthermore, studies have shown that plasma BNP level rises in response to myocardial ischemia, hypoxia, or infarction, supporting its role in the diagnosis, treatment monitoring, and prognostic evaluation of ischemic cardiac diseases [29]. While BNP is involved in the body's compensatory mechanism, its abnormally elevated levels indicate significant cardiac dysfunction [30].

To improve the accuracy of prediction, we constructed a combined predictive model incorporating both sST2 and BNP. The AUC of this model reached 0.929, demonstrating excellent predictive performance. The joint application of these two biomarkers enhances both sensitivity and specificity, providing a more comprehensive reflection of the complex pathophysiological mechanisms underlying heart failure. This combined predictive model holds potential in future clinical application, assisting doctors in formulating more precise, individualized treatment plans and potentially improving patient outcomes.

Despite the valuable findings from this work, several limitations should be acknowledged. First, this was a retrospective study, which may be subject to selection bias and information bias, potentially affecting the external validity of the results. Second, the sample size was relatively small, particularly in the ineffective group, possibly limiting the statistical power of certain conclusions. Third, since the data were sourced from a single center, the generalizability of the results is restricted. Future research should adopt multi-center, prospective designs to address these limitations and explore addi-

tional biomarkers associated with treatment outcomes.

Conclusions

In summary, serum soluble ST2 and BNP are effective biomarkers for predicting treatment outcomes in patients with heart failure. Their combined use significantly improves predictive performance. These findings provide clinicians with valuable tools to guide treatment strategies and optimize therapeutic outcomes, ultimately enhancing prognosis of HF patients.

Acknowledgements

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Disclosure of conflict of interest

None.

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References

[1] Zamorano JL and González Leal A. Advances in heart failure management. Med Clin (Barc) 2024; 163: 32-39.
[2] Heidenreich P. Heart failure management guidelines: New recommendations and implementation. J Cardiol 2024; 83: 67-73.
[3] Wyss C. Introduction: heart failure. Ther Umsch 2024; 81: 30.
[4] Khan MS, Shahid I, Bennis A, Rakisheva A, Metra M and Butler J. Global epidemiology of heart failure. Nat Rev Cardiol 2024; 21: 717-734.
[5] Takizawa D, Harada T, Obokata M, Kagami K, Sorimachi H, Yuasa N, Saito Y, Murakami F, Naito A, Kato T, Wada N and Ishii H. Pathophysiologic and prognostic importance of cardiac power output reserve in heart failure with preserved ejection fraction. Eur Heart J Cardiovasc Imaging 2024; 25: 220-228.
[6] Villadsen PR, Horváth-Puhó E, Sørensen HT and Schmidt M. The interaction effect of cardiac and noncardiac co-morbidities on mortality rates in patients with heart failure. Am J Cardiol 2022; 179: 51-57.
[7] Castiglione V, Aimo A, Vergaro G, Saccaro L, Passino C and Emdin M. Biomarkers for the diagnosis and management of heart failure. Heart Fail Rev 2022; 27: 625-643.
[8] Riccardi M, Myhre PL, Zelniker TA, Metra M, Januzzi JL and Inciardi RM. Soluble ST2 in heart failure: a clinical role beyond B-type natriuretic peptide. J Cardiovasc Dev Dis 2023; 10: 468.
[9] Savarimuthu S, Goel P and Harky A. Soluble ST2: a valuable prognostic marker in heart failure. Heart Fail Rev 2022; 27: 2155-2164.
[10] Shah SJ. BNP: biomarker not perfect in heart failure with preserved ejection fraction. Eur Heart J 2022; 43: 1952-1954.
[11] Torfi E, Bahreiny SS, Saki N, Khademi R, Sarbazjoda E, Nezhad IA and Aghaei M. Evaluation of Pro-BNP biomarker in heart failure patients and its relationship with complete blood count parameters: a case-control study. Health Sci Rep 2024; 7: e70083.
[12] Berthelot E, Bailly MT, Lehova XC, Rahmani MEB, Bounab R, Mewton N, Dobbs JE, Mas R, Frank M, Lellouche N, Paclot M and Jourdain P. Setting the optimal threshold of NT-proBNP and BNP for the diagnosis of heart failure in patients over 75 years. ESC Heart Fail 2024; 11: 3232-3241.
[13] Cheung CC, Nattel S, Macle L and Andrade JG. Management of atrial fibrillation in 2021: an updated comparison of the current CCS/CHRS, ESC, and AHA/ACC/HRS guidelines. Can J Cardiol 2021; 37: 1607-1618.
[14] Gualandro DM, Puelacher C and Mueller C. Reply to the letter regarding the article 'Acute heart failure after non-cardiac surgery: incidence, phenotypes, determinants and outcomes'. Eur J Heart Fail 2023; 25: 771-772.
[15] Tan ESJ, Chan SP, Liew OW, Chong JPC, Leong GKT, Yeo DPS, Ong HY, Jaufeerally F, Yap J, Sim D, Ng TP, Ling LH, Lam CSP and Richards AM. Atrial fibrillation and the prognostic performance of biomarkers in heart failure. Clin Chem 2021; 67: 216-226.
[16] Qin L, Liu X and Li Y. Correlation of serum BNP and ET-1 levels with cardiac pump function and ventricular remodeling in patients with heart failure. Cell Mol Biol (Noisy-le-grand) 2020; 66: 125-131.
[17] Monzo L, Girerd N, Ferreira JP, Lamiral Z, Anker SD, Cleland JGF, Kondo T, McMurray JJV, Lam CSP, Mehra MR, Veldhuisen DJV, Greenberg B and Zannad F. High risk of stroke in patients with worsening heart failure, reduced ejection fraction, coronary heart disease and sinus rhythm: risk prediction score analysis from the COMMANDER-HF trial. J Card Fail 2024; 30: 618-623.

[18] Backhaus SJ, Lange T, George EF, Hellenkamp K, Gertz RJ, Billing M, Wachter R, Steinmetz M, Kutty S, Raaz U, Lotz J, Friede T, Uecker M, Hasenfuß G, Seidler T and Schuster A. Exercise stress real-time cardiac magnetic resonance imaging for noninvasive characterization of heart failure with preserved ejection fraction: the HFpEF-stress trial. Circulation 2021; 143: 1484-1498.

[19] Espriella R, Bayés-Genis A, Revuelta-LóPEZ E, Miñana G, Santas E, Llàcer P, García-Blas S, Fernández-Cisnal A, Bonanad C, Ventura S, Sánchez R, Bodí V, Cordero A, Fácila L, Mollar A, Sanchis J and Núñez J; IMPROVE-HF Investigators. Soluble ST2 and diuretic efficiency in acute heart failure and concomitant renal dysfunction. J Card Fail 2021; 27: 427-434.

[20] Krittayaphong R, Pumprueg S and Sairat P. Soluble ST2 in the prediction of heart failure and death in patients with atrial fibrillation. Clin Cardiol 2022; 45: 447-456.

[21] Grakova EV, Yakovlev AV, Shilov SN, Berezikova EN, Kopeva KV, Yakovleva NF, Ogurkova ON and Teplyakov AT. Heart failure with preserved left ventricular ejection fraction in patients with obstructive sleep apnea syndrome: prognostic value of biomarkers. Kardiologiia 2021; 61: 77-88.

[22] Nah EH, Cho S, Kim S and Cho HI. Reference interval and the role of soluble suppression of tumorigenicity 2 (sST2) in subclinical cardiac dysfunction at health checkups. J Clin Lab Anal 2020; 34: e23461.

[23] Jabagi H, Ruel M and Sun LY. Can Biomarkers provide right ventricular-specific prognostication in the perioperative setting? J Card Fail 2020; 26: 776-780.

[24] Akdis D, Chen L, Saguner AM, Zhang N, Gawinecka J, Saleh L, von Eckardstein A, Ren J, Matter CM, Hu Z, Chen X, Tanner FC, Manka R, Chen K, Brunckhorst C, Song J and Duru F. Novel plasma biomarkers predicting biventricular involvement in arrhythmogenic right ventricular cardiomyopathy. Am Heart J 2022; 244: 66-76.

[25] Lwin M, Win KCK and Sieniewicz B. Reliability of BNP to exclude heart failure. Clin Med (Lond) 2023; 23 Suppl 6: 2.

[26] Nishikimi T and Nakagawa Y. Potential pitfalls when interpreting plasma BNP levels in heart failure practice. J Cardiol 2021; 78: 269-274.

[27] Kang R, Nagoshi T, Kimura H, Tanaka TD, Yoshii A, Inoue Y, Morimoto S, Ogawa K, Minai K, Ogawa T, Kawai M and Yoshimura M. Possible association between body temperature and B-type natriuretic peptide in patients with cardiovascular diseases. J Card Fail 2021; 27: 75-82.

[28] Ikebe S, Ishii M, Otsuka Y, Nakamura T, Tsujita K, Matoba T, Kohro T, Oba Y, Kabutoya T, Imai Y, Kario K, Kiyosue A, Mizuno Y, Nochioka K, Nakayama M, Iwai T, Miyamoto Y, Sato H, Akashi N, Fujita H and Nagai R; CLIDAS research group. Impact of heart failure severity and major bleeding events after percutaneous coronary intervention on subsequent major adverse cardiac events. Int J Cardiol Cardiovasc Risk Prev 2023; 18: 200193.

[29] Tajbakhsh A, Gheibi Hayat SM, Taghizadeh H, Akbari A, Inabadi M, Savardashtaki A, Johnston TP and Sahebkar A. COVID-19 and cardiac injury: clinical manifestations, biomarkers, mechanisms, diagnosis, treatment, and follow up. Expert Rev Anti Infect Ther 2021; 19: 345-357.

[30] Cao Z, Zhao M, Xu C, Zhang T, Jia Y, Wang T and Zhu B. Evaluation of agonal cardiac function for sudden cardiac death in forensic medicine with postmortem brain natriuretic peptide (BNP) and NT-proBNP: a meta-analysis. J Forensic Sci 2020; 65: 686-691.