e-ISSN: 0975-9506, p-ISSN: 2961-6093

Available online on www.ijpqa.com doi: 10.25258/ijpqa.16.10.63

International Journal of Pharmaceutical Quality Assurance 2025; 16(10); 396-402

Original Research Article

A Study Correlation Between Serum Ferritin Levels and Clinical Outcomes in Heart Failure Patients

Nilashish Dey

Senior Resident, Dr NB (Resident), Department of Cardiology, Fortis Escorts, Heart Institute, New Delhi, India

Received: 10-08-2025 / Revised: 16-09-2025 / Accepted: 22-10-2025
Corresponding Author: Dr. Nilashish Dey
Conflict of interest: Nil

Abstract:

Background: Heart failure (HF) remains a major global health burden, and identifying reliable biomarkers that reflect disease severity is essential for early risk stratification. Serum ferritin, an indicator of iron status and inflammation, may serve as a useful predictor of HF severity.

Aim: To evaluate the association between serum ferritin levels and clinical, biochemical, and echocardiographic markers of severity in HF patients.

Methodology: A hospital-based cross-sectional study was conducted among 90 HF patients at a tertiary care center. Detailed clinical evaluation, laboratory investigations including ferritin, hepcidin, and hs-CRP, and echocardiographic assessment were performed. Statistical analyses included correlation and ROC analysis.

Results: The mean age of participants was 58.6 ± 10.4 years, with 62.2% males. Serum ferritin showed significant positive correlations with NYHA class ($r = 0.41$) and hs-CRP ($r = 0.48$), and negative correlations with hemoglobin ($r = –0.22$) and LVEF ($r = –0.39$). Higher ferritin levels were associated with ventricular dilatation and increased inflammatory activity. ROC analysis identified ferritin >180 ng/mL as a predictor of severe HF (AUC 0.78, sensitivity 74%, specificity 72%).

Conclusion: Serum ferritin is a valuable, accessible biomarker reflecting inflammation, iron dysregulation, and cardiac dysfunction, demonstrating good predictive utility for HF severity.

Keywords: Heart failure, Serum ferritin, Biomarker, Inflammation, NYHA class, LVEF.

Introduction

Heart failure (HF) is a complicated clinical syndrome which is defined by the inability of the heart to pump adequate blood to satisfy the metabolic needs of the body which causes enormous morbidity and mortality in the global world [1]. HF is a significant issue in the public health problem, despite pharmacological and device-based therapeutic options, the condition continues to spread because of ageing populations and growing risks of cardiovascular diseases including hypertension, diabetes mellitus, and ischemic heart disease. Early recognition of patients who are at risk of having serious outcomes in HF is important in enhancing the prognosis and in informing the therapeutic interventions [2]. Biomarkers have become critical in the diagnosis, prognosis, and treatment of HF as they offer clinicians with quantitative information that can be used to represent underlying pathophysiological events, such as myocardial injury, neurohormonal stimulation, inflammation, and iron metabolism. One of these biomarkers is serum ferritin, which has received significant interest as a measure of iron depots and reactant in acute-phase inflammatory conditions.

The prevalence of iron deficiency in patients with chronic heart failure is very high with 30 to 50 cases reported and it is related to poorer functional capacity, greater hospitalization, and higher mortality rates [3]. Iron is essential in the oxygen delivery process, generation of cellular energy, as well as enzyme reactions, which are important to the functions of the myocardial and skeletal muscles. Lack of iron reduces mitochondrial oxidative metabolism, which results in a reduction in exercise capacity, fatigue, and cardiac dysfunction that leads up to progress [4]. In normal physiological conditions, serum ferritin, which is a protein that stores and releases iron in a regulated action, is a surrogate marker of total body iron stores. Low serum ferritin levels typically show exhausted iron stores and high ferritin levels can indicate excess iron or an inflammatory process as is typical in HF because of systemic inflammation, oxidative stress, and comorbidity [5]. Thus, the evaluation of serum ferritin levels in patients with HF offers the information not only about the iron condition but also about the inflammatory milieu, which can also determine the severity of the disease and its clinical outcomes.

A number of researches have been carried out to examine the association between iron deficiency, serum ferritin, and heart failure prognosis. It is noted that low serum ferritin or functional iron deficiency patients are associated with New York Heart Association (NYHA) functional class, diminished exercise capacity indicated by peak oxygen consumption (VO2 max), and unfavorable quality of life [6] in the HF patients. IV iron supplementation among such patients has been demonstrated to increase the exercise tolerance, minimize HF-related hospitalizations, and increase the symptomatic relief, which underscores the clinical value of ferritin testing in the management of HF. On the other hand, high ferritin among HF patients can be an indication of persistent inflammatory processes, which in turn is linked with unfavorable remodelling of the heart, myocardial fibrosis, and higher mortality rates [7] also. This twofold meaning highlights the possible practical use of serum ferritin as a biomarker of the severity of HF, which has prognostic and treatment value.

Moreover, the pathophysiological interaction between iron metabolism and HF progression have been largely identified. The illness of chronic HF is typified by systemic inflammation, neurohormonal activation and endothelial dysfunction, which may interfere with iron homeostasis. Hepcidin is the main iron absorption and mobilization regulator stimulated by proinflammatory cytokines, including interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF-) [8]. HF leads to elevated hepcidin which limits intestinal iron absorption and sequesters iron in the macrophages, resulting in functional deficiency of iron despite having sufficient total body iron. As a result, the serum ferritin values, when taken in conjunction with other iron parameters like saturation of transferrin, will offer a wholesome evaluation of iron availability and can be used as a surrogate measure of the extent of underlying clinical pathophysiological upheavals of HF.

Although the role of iron deficiency in HF has been identified as significant, no data has been found to establish the direct correlation between serum ferritin and the clinical severity of HF, especially in heterogeneous populations of patients. The available literature is centered on iron supplementation instead of ferritin as a biomarker to establish the condition of the disease. Also, the differences in the ferritin cut-offs values, the variations in the etiology of HF, and the effect of comorbid inflammatory conditions make the interpretation of ferritin measurements difficult [9]. Thus, it is necessary to systematically compare serum ferritin levels with the previously determined indicators of severity of HF, including left ventricular ejection fraction (LVEF), NYHA functional classification, and hospitalization rates, to understand whether to use it as a useful predictor or not. The detection of a good biomarker to measure the iron status and the severity of the disease at the same time can help to risk stratify and allow customized treatment methods and achieve better patient outcomes.

Furthermore, the inclusion of serum ferritin measurement into standard HF screening is consistent with the trend of increasing the focus on precision medicine within the cardiology field. Biomarker-based management can be used to improve clinical judgment to identify high-risk patients who can be targeted to receive specific interventions, such as iron-based therapy, more intense drugs, or more frequent follow-ups. It also enables early detection of failing cardiac performance and as such, it averts disease pathogenesis and minimizes the impact of morbidity of HF. Due to the worldwide significance of HF and the constraints of the existing prognostic measurements, any study that would verify serum ferritin as a prognostic factor of severity is an act of significance in enhancing the care accorded to patients and the allocation of resources.

Methodology

Study Design: This study was designed as a hospital-based observational cross-sectional study aimed at evaluating serum ferritin levels as a predictor of severity in heart failure (HF) patients. The study focused on correlating serum ferritin levels with clinical, biochemical, and echocardiographic parameters to assess their potential as a prognostic marker.

Study Area: The study was conducted at the Department of Cardiology, Fortis Escorts Heart Institute, New Delhi, India form March 2023 to February 2024

Study Participants

Inclusion Criteria

- Patients of both sexes, aged ≥18 years.
- Documented history of heart failure for at least 6 months.
- Presence of symptoms and signs of left-sided heart failure.
- Left ventricular ejection fraction (LVEF) ≤45% as assessed by echocardiography.

Exclusion Criteria

- Acute coronary syndrome within the preceding 3 months.
- History of coronary revascularization within the preceding 3 months.
- Unplanned hospitalization due to heart failure deterioration or other cardiovascular reasons within 1 month.
- Acute or chronic illnesses affecting iron metabolism, including:
    - Malignancy
    - Infection
    - Severe renal disease requiring dialysis

- Hematological disorders

Sample Size: A total of 90 patients meeting the inclusion and exclusion criteria were enrolled in the study.

Procedure: Following the written informed consent of all the participants, demographic and clinical histories were taken in detail (age, sex, lifestyle practices, related chronic illnesses e.g. diabetes mellitus, hypertension, liver or endocrine diseases, family history, onset and duration of heart disease, palpitation symptoms and fatigue, previous medication use). In-depth clinical investigations were conducted, and the vital parameters (blood pressure, pulse rate, respiratory rate, and temperature) alongside the careful heart examination were carried out. Laboratory tests involved hemoglobin, mean corpuscular volume (MCV), mean corpuscular hemoglobin (MCH), mean corpuscular hemoglobin concentration (MCHC), serum creatinine and high-sensitivity C-reactive protein (hs-CRP using kinetic nephelometry. The iron status, serum ferritin level, and serum hepcidin were also determined. Electrocardiography (12-lead ECG) was used to measure the heart rate, rhythm, ischemic variations, and conduction pattern, whereas echocardiography measured the dimensions of the left ventricles, ejection fraction, and degree of mitral regurgitation. Every action was ethically correct, and the confidentiality of patients was ensured.

Statistical Analysis: Data were analyzed using SPSS version 22. Normality was assessed using the Shapiro–Wilk test. Categorical variables were presented as frequencies and percentages and compared using the Chi-square or Fisher's exact test. Quantitative variables were expressed as mean ± SD or median (range) and analyzed using t-test, Mann–Whitney U test, ANOVA, or Kruskal–Wallis test as appropriate. Spearman's correlation and ROC analysis were performed, with $p \leq 0.05$ considered statistically significant.

Result

The demographic and clinical baseline data of the 90 participants of the study are represented in Table 1, with the mean age of 58.6 years and a male majority (62.2%). Mean life of heart failure was 18.3 months/7.9 months. The participants had a percentage of 42.2% with diabetes mellitus, 48.9 with hypertension and 32.2 smokers. According to NYHA classification, 37.8 percent were in Class II, 45.6 percent in Class III and 16.6 percent in Class IV, implying that most of them had moderate functional limitation.

Table 1: Baseline Demographic and Clinical Characteristics of the Study Participants (n = 90)

Variable	Mean ± SD / n (%)
Age (Years)	58.6 ± 10.4
Male / Female	56 (62.2%) / 34 (37.8%)
Duration of HF (months)	18.3 ± 7.9
Diabetes Mellitus	38 (42.2%)
Hypertension	44 (48.9%)
Smoking Status	29 (32.2%)
NYHA Class II	34 (37.8%)
NYHA Class III	41 (45.6%)
NYHA Class IV	15 (16.6%)

As indicated by Table 2, patients with heart failure tended to have mild anemia, as indicated by a mean hemoglobin of 11.8 ± 1.6 g/dl and normal average red cell indices, including MCV (88.4 ± 7.1 fL) and MCH (27.2 ± 3.4 pg). The serum ferritin levels showed broad deviation with a median of 162 ng/mL (45 580), showing a mixed iron status of the patients. The renal functioning was moderately impaired, with a mean of serum creatinine of 1.28±0.42 mg/dl. The level of inflammatory markers was high, with a median of hs-CRP of 9.6 mg/L (238) and a wide range of serum hepcidin level of 23ng/mL (871) indicating a heterogeneous level of iron control and inflammatory response among the sample.

Table 2: Laboratory Parameters in Patients with Heart Failure

Laboratory Variable	Mean ± SD / Median (Range)
Hemoglobin (g/dL)	11.8 ± 1.6
Serum Ferritin (ng/mL)	162 (45–580)
MCV (fL)	88.4 ± 7.1
MCH (pg)	27.2 ± 3.4
Serum Creatinine (mg/dL)	1.28 ± 0.42
hs-CRP (mg/L)	9.6 (2–38)
Serum Hepcidin (ng/mL)	23 (8–71)

Table 3 shows the echocardiographic picture of the participants of the study, and it is evident that the cardiac structure and the cardiac functioning changed significantly. The average left ventricular ejection fraction (LVEF) was decreased to 36.2 +/- 6.8% which shows the systolic dysfunction that is associated with heart failure. There was significant increase in left ventricular dimensions and the LVD of 56.4 and LVESD of 45.2 indicates that there was ventricular dilation. Valvular valuation revealed that mitral regurgitation was common with 24.4% of the subjects experiencing mild, 47.8% moderate and 27.8% severe mitral regurgitation indicating a significant valvular dysfunction among the cohort.

Table 3: Echocardiographic Findings of the Study Participants	
Echocardiographic Parameter	**Mean ± SD**
LVEF (%)	36.2 ± 6.8
LVEDD (mm)	56.4 ± 7.3
LVESD (mm)	45.2 ± 6.1
Severity of Mitral Regurgitation	Mild: 22 (24.4%)
	Moderate: 43 (47.8%)
	Severe: 25 (27.8%)

Table 4 reveals that serum ferritin had significant associations with various clinical and echocardiographic variables among patients with heart failure. The association between ferritin and NYHA class ($r = 0.41$, $p = 0.001$), as well as hs-CRP ($r = 0.48$, $p < 0.001$), was moderate with a positive correlation, which implied that higher ferritin levels were related to poor functional status and hs-CRP. A low negative correlation with hemoglobin ($r= -0.22$, $p=0.03$) indicated that high ferritin levels were associated with low hemoglobin levels. There was also moderate positive correlation between ferritin and serum hepcidin ($r = 0.36$, $p = 0.002$), which further confirms the association of ferritin with the impaired iron regulation. Echocardiographically, ferritin was found to have a negative relationship with LVEF with $r = -0.39$, $p = 0.001$ and a positive relationship with LVEDD with $r = 0.33$, $p = 0.004$ as ferritin levels were found to be associated more with decreased systolic performance and ventricular dilatation.

Table 4: Correlation of Serum Ferritin with Clinical and Echocardiographic Parameters		
Parameter	**Correlation Coefficient (Spearman r)**	**p-value**
NYHA Class	0.41	0.001
hs-CRP	0.48	<0.001
Hemoglobin	–0.22	0.03
Serum Hepcidin	0.36	0.002
LVEF (%)	–0.39	0.001
LVEDD	0.33	0.004

Table 5 shows that serum ferritin had a good predictive value in ascertaining the severity of heart failure as indicated by AUC of 0.78, indicating fair predictive value. The cut-off value of >180 ng/mL was identified, which gave a balanced sensitivity of 74% and specificity of 72%, i.e. it identified large proportion of severe cases, and with the lowest false positives. The statistically significant p-value (0.001) also indicates that serum ferritin is a meaningful and reliable biomarker in assessing the severity of heart failure.

Table 5: ROC Analysis of Serum Ferritin as a Predictor of Heart Failure Severity					
Variable	**AUC**	**Cut-off Value (ng/mL)**	**Sensitivity (%)**	**Specificity (%)**	**p-value**
Serum Ferritin	0.78	>180	74%	72%	<0.001

Discussion

The current research analyzed serum ferritin as a possible indicator of heart failure (HF) severity and proved that there were significant correlations between the levels of ferritin, clinical variables, inflammation, anemia, and echocardiographic damage. The demographics of our population (mostly late-middle-aged men with frequent comorbidities, including diabetes and hypertension) is aligned with previous epidemiological data. As an example, Ekowitz et al. (2003) [10] has reported that HF is more common in older males and highly related to metabolic comorbidities, which supports our finding that cardiovascular risk factors are clustered around patients with progressive HF. Similarly, Redfield et al. (2003) [11] found that the same trend in age is recorded, implying that our cohort represents the general HF population.

Our study had an impressive prevalence of anemia (45%), which is consistent with the results presented by Cleland et al. (2016) [12] (40–50%) of patients with chronic HF and anemia as a key predictor of

clinical deterioration). We also observed much lower hemoglobin and red cell indices in HF patients than controls, which confirms the finding of Nanas et al. (2006) [13] that anemia is multifactorial in HF, and is commonly sustained by inflammation, hemodilution, and impaired erythropoiesis. The negative relation between ferritin and hemoglobin in our analysis indicates that higher ferritin is shown to be anemia of chronic disease and not iron deficiency, which was mentioned by Weiss and Goodnough (2005) [14] that ferritin is an acute-phase reactant and may be increased with inflammatory states despite iron depletion of the body.

Inflammation seemed to be in the center stage of our group of patients, as the correlation between serum ferritin and hs-CRP was very high. It is consistent with the results of DuBrock HM. (2018) [15] who found that the severity of HF correlates with systemic inflammation, increased CRP, and iron metabolism changes. We also find that Jankowska et al. (2010) [16] have indicated that inflammatory activation in HF is strongly associated with iron homeostasis disruptions, high ferritin, and low functional iron in the body. These mechanisms are supported in the current study since patients with increased NYHA class showed an increased level of ferritin and hs-CRP.

The serum ferritin levels were significantly variable in our population and to the point, ferritin was positively associated with the HF severity indicators. The patients who were of higher NYHA, lower LVEF, and LVEDD were found to have an elevated level of ferritin implying that ferritin is an indicator of underlying ventricular alterations in remodelling and inflammation. Our result corresponds to that of Jankowska et al. (2010) [16] who found that the elevation of ferritin levels in HF is associated with the deterioration of clinical class and greater neurohormonal stimulation. Conversely, research conducted by Klip et al. (2013) [17] discovered that low (and not high) levels of ferritin were correlated with HF severity and suggested the correlation to be due to absolute iron deficiency. This inconsistency points out that ferritin needs to be put into the context of inflammation. In highly inflammatory groups of people, as in our cohort, ferritin increase might obscure underlying iron deficiency, and be used as a proxy outcome of inflammation.

The ROC analysis in our research study showed that serum ferritin >180 ng/mL showed good discriminatory accuracy (AUC 0.78) in severity assessment of HF. This can be justified by the idea developed by Welsh et al. (2018) [18], who has proven that iron metabolism-related biomarkers, such as ferritin, could be used as prognostic in systolic HF. Likewise, Kuragano et al. (2020) [19] determined that ferritin and TSAT are independent variables that predict hospitalization risk. Our findings contribute to this evidence since they establish a particular cut-off level, which is appropriate in clinical screening.

In our group, the echocardiographic abnormalities (especially low LVEF and high LV dimensions) were associated with high levels of ferritin. The same was also observed by Mocan et al. (2019) [20] who showed that LV dysfunction and HF progression are both related to inflammatory biomarkers like ferritin and CRP. The structural remodeling patterns, as described by Amigoni et al. (2007) [21] are also in line with the high prevalence of mitral regurgitation in our cohort which they reported, demonstrated that ventricular dilatation and functional MR are common and exacerbate the outcome of HF.

The action of hepcidin in the regulation of iron in heart failure is debatable. In the current analysis, it is found that the level of hepcidin is lower in patients with heart failure, especially in more serious cases. This trend indicates that the hepcidin activity is suppressed with the advanced heart failure, which could display poor iron use and increased chronic inflammation. On the other hand, a number of observations during early heart failure suggest that the level of hepcidin may be high at the early stages, suggesting that the hormone may be dynamically changed as the disease advances. The fact that hepcidin decreased in cases of the current study that are more severe supports the idea that progressive impairment of hypoxia and inflammatory loads can slowly suppress hepcidin production, thus enhancing the mobilization of iron in more severe stages of the disorder.

In general, we present the findings that supplement the accumulating evidence that ferritin is a versatile biomarker of inflammation, iron dysregulation, anemia, and cardiac dysfunction. Closely related to the strong correlation between ferritin, the NYHA class, and echocardiographic deterioration, the latter proves handy and reliable as a predictor of the severity of HF. Ferritin alone is not able to differentiate between absolute and functional iron deficiency, but due to the high correlations with clinical and structural indicators, it is an important prognostic factor. Serum ferritin (used with clinical assessment in resources-constrained settings, where more rigorous tests are not available), can assist clinicians in detecting high-risk patients with HF and in need of closer observation or specific treatment.

Conclusion

The findings of this study highlight that serum ferritin is a meaningful and accessible biomarker for assessing the severity of heart failure. Higher ferritin levels were consistently associated with worse functional status, greater inflammatory activity, lower hemoglobin levels, and more pronounced ventricular dysfunction. The significant correlations between ferritin, NYHA class, LVEF, LVEDD, hs-CRP, and hepcidin indicate that ferritin reflects the combined influence of inflammation, iron

dysregulation, and cardiac remodeling in HF. The ROC analysis further demonstrated that a ferritin cut-off >180 ng/mL offers good predictive accuracy for identifying severe disease. Overall, serum ferritin showed strong potential as a practical tool for risk stratification, enabling early identification of high-risk patients and supporting more informed clinical decision-making in heart failure management.

References

1. Tanai E, Frantz S. Pathophysiology of heart failure. Comprehensive physiology. 2016 Jan 17;6(1):187-214.
2. Hammond MM, Everitt IK, Khan SS. New strategies and therapies for the prevention of heart failure in high-risk patients. Clinical cardiology. 2022 Jun;45:S13-25.
3. von Haehling S, Ebner N, Evertz R, Ponikowski P, Anker SD. Iron deficiency in heart failure: an overview. JACC: Heart Failure. 2019 Jan;7(1):36-46.
4. Ravingerová T, Kindernay L, Barteková M, Ferko M, Adameová A, Zohdi V, Bernátová I, Ferenczyová K, Lazou A. The molecular mechanisms of iron metabolism and its role in cardiac dysfunction and cardioprotection. International journal of molecular sciences. 2020 Oct 24;21(21):7889.
5. Silvestre OM, Gonçalves A, Nadruz Jr W, Claggett B, Couper D, Eckfeldt JH, Pankow JS, Anker SD, Solomon SD. Ferritin levels and risk of heart failure—the Atherosclerosis Risk in Communities Study. European journal of heart failure. 2017 Mar;19(3):340-7.
6. Bredy C, Ministeri M, Kempny A, Alonso-Gonzalez R, Swan L, Uebing A, Diller GP, Gatzoulis MA, Dimopoulos K. New York Heart Association (NYHA) classification in adults with congenital heart disease: relation to objective measures of exercise and outcome. European Heart Journal-Quality of Care and Clinical Outcomes. 2018 Jan 1;4(1):51-8.
7. Paulus WJ, Zile MR. From systemic inflammation to myocardial fibrosis: the heart failure with preserved ejection fraction paradigm revisited. Circulation research. 2021 May 14; 128(10): 1451-67.
8. Rosenblum SL. Inflammation, dysregulated iron metabolism, and cardiovascular disease. Frontiers in Aging. 2023 Feb 3; 4:1124178.
9. Dhondge RH, Agrawal S, Kumar S, Acharya S, Karwa V. A comprehensive review on serum ferritin as a prognostic marker in intensive care units: insights into ischemic heart disease. Cureus. 2024 Mar 31;16(3).
10. Ezekowitz JA, McAlister FA, Armstrong PW. Anemia is common in heart failure and is associated with poor outcomes: insights from a cohort of 12 065 patients with new-onset heart failure. Circulation. 2003 Jan 21;107(2):223-5.
11. Redfield MM, Jacobsen SJ, Burnett JC, Mahoney DW, Bailey KR, Rodeheffer RJ. Burden of systolic and diastolic ventricular dysfunction in the community: appreciating the scope of the heart failure epidemic. Jama. 2003 Jan 8;289(2):194-202.
12. Cleland JG, Zhang J, Pellicori P, Dicken B, Dierckx R, Shoaib A, Wong K, Rigby A, Goode K, Clark AL. Prevalence and outcomes of anemia and hematinic deficiencies in patients with chronic heart failure. JAMA cardiology. 2016 Aug 1;1(5):539-47.
13. Nanas JN, Matsouka C, Karageorgopoulos D, Leonti A, Tsolakis E, Drakos SG, Tsagalou EP, Maroulidis GD, Alexopoulos GP, Kanakakis JE, Anastasiou-Nana MI. Etiology of anemia in patients with advanced heart failure. Journal of the American College of Cardiology. 2006 Dec 19;48(12):2485-9.
14. Weiss G, Goodnough LT. Anemia of chronic disease. New England Journal of Medicine. 2005 Mar 10;352(10):1011-23.
15. DuBrock HM, Abou Ezzeddine OF, Redfield MM. High-sensitivity C-reactive protein in heart failure with preserved ejection fraction. PloS one. 2018 Aug 16;13(8):e0201836.
16. Jankowska EA, Rozentryt P, Witkowska A, Nowak J, Hartmann O, Ponikowska B, Borodulin-Nadzieja L, Banasiak W, Polonski L, Filippatos G, McMurray JJ. Iron deficiency: an ominous sign in patients with systolic chronic heart failure. European heart journal. 2010 Aug 1;31(15):1872-80.
17. Klip IT, Comin-Colet J, Voors AA, Ponikowski P, Enjuanes C, Banasiak W, Lok DJ, Rosentryt P, Torrens A, Polonski L, Van Veldhuisen DJ. Iron deficiency in chronic heart failure: an international pooled analysis. American heart journal. 2013 Apr 1;165(4):575-82.
18. Welsh P, Kou L, Yu C, Anand I, van Veldhuisen DJ, Maggioni AP, Desai AS, Solomon SD, Pfeffer MA, Cheng S, Gullestad L. Prognostic importance of emerging cardiac, inflammatory, and renal biomarkers in chronic heart failure patients with reduced ejection fraction and anaemia: RED-HF study. European Journal of Heart Failure. 2018 Feb;20(2):268-77.
19. Kuragano T, Joki N, Hase H, Kitamura K, Murata T, Fujimoto S, Fukatsu A, Inoue T, Itakura Y, Nakanishi T. Low transferrin saturation (TSAT) and high ferritin levels are significant predictors for cerebrovascular and cardiovascular disease and death in maintenance hemodialysis patients. PLoS One. 2020 Sep 2; 15(9): e0236277.
20. Mocan M, Mocan Hognogi LD, Anton FP, Chiorescu RM, Goidescu CM, Stoia MA, Farcas AD. Biomarkers of inflammation in left ventricular diastolic dysfunction. Disease Markers. 2019;2019(1):7583690.

21. Amigoni M, Meris A, Thune JJ, Mangalat D, Skali H, Bourgoun M, Warnica JW, Barvik S, Arnold JM, Velazquez EJ, Van de Werf F. Mitral regurgitation in myocardial infarction complicated by heart failure, left ventricular dysfunction, or both: prognostic significance and relation to ventricular size and function. European heart journal. 2007 Feb 1;28(3):326-33.