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Original Article

Association of Iron Deficiency with Disease Severity in Patients with Congestive Heart Failure: A Prospective Observational Study

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ABSTRACT

Background: Iron deficiency in heart failure is frequent and can have an independent effect on skeletal-muscle energetics, myocardial function, exercise tolerance and quality of life, apart from the anemia itself. The aim of this study was to determine whether iron deficiency was associated with the severity of clinical and biochemical disease in patients with congestive heart failure (CHF).

Methods: This was a prospective observational study of 168 consecutive adults with clinically stable or recently decongested CHF followed for six months. Iron deficiency was considered as serum ferritin <100 ng/mL or serum ferritin 100-299 ng/mL and transferrin saturation (TSAT) <20%. The severity of the disease was evaluated by the New York Heart Association (NYHA) functional class, left ventricular ejection fraction (LVEF), N-terminal pro-B-type natriuretic peptide (NT-proBNP), six-minute walk distance (6MWD), and heart-failure readmission.

Results: Iron deficiency was found in 102 patients (60.7%) and 58.8% of iron deficient patients were anemic. Compared with patients without iron deficiency, the iron-deficient group had a higher frequency of NYHA class III-IV symptoms (71.6% vs 42.4%, p<0.001), lower LVEF (31.8±8.4% vs 36.7±9.1%, p=0.001), higher NT-proBNP (3280±1710 vs 2240±1390 pg/mL, p<0.001), and shorter 6MWD (278±82 vs 342±88 m, p<0.001). Heart failure readmission rates were 31.4% at 6 months compared with 15.2% (p=0.018). Iron deficiency was independently associated with NYHA class III-IV status after adjusting for age, sex, hemoglobin, renal function, and LVEF (adjusted OR 2.48, 95% CI 1.24-4.97; p=0.010).

Conclusion: Iron deficiency was very common in CHF and correlated with increased clinical, functional, biochemical and short-term clinical severity. Ferritin and TSAT levels can be routinely measured to detect a clinically important high-risk phenotype.

Keywords: heart failure; iron deficiency; ferritin; transferrin saturation; NYHA class; NT-proBNP; ejection fraction.

INTRODUCTION

Congestive heart failure (CHF) is a clinical syndrome with multiple components that include impaired cardiac performance, recurrent congestion, exercise intolerance and a high risk of hospitalization and death. In the current era of heart failure treatment, comorbidities are increasingly appreciated as having a significant impact on symptoms and prognosis despite best possible guideline-directed cardiac therapy. There is a general consensus in Europe that routine iron status assessment

should be considered in the management of symptomatic heart failure due to the prevalence and treatability of iron deficiency. [1]

In addition to erythropoiesis, iron is also required for mitochondrial oxidation phosphorylation, skeletal muscle metabolism, and myocardial energetics. Heart failure patients can suffer from absolute iron deficiency due to decreased intake, gastrointestinal loss, and decreased absorption, but can also suffer from functional iron deficiency due to sequestration of iron by inflammation and altered hepcidin signaling. A recent paper on HFREF highlighted that iron deficiency may be present with or without anemia and should be assessed based on iron indices, not hemoglobin levels [2]. Iron deficiency is usually defined as serum ferritin <100 ng/mL or ferritin 100-299 ng/mL with transferrin saturation (TSAT) <20% according to conventional definition of iron deficiency in heart failure. This definition has been applied widely in clinical trials and guidelines, but the inflammatory nature of heart failure can lead to higher ferritin levels and make interpretation difficult. Studies that have compared the diagnostic criteria indicate that low TSAT may more accurately detect patients with a poor prognosis and more functional impairment than ferritin alone [3].

The relationship between iron deficiency and disease severity is biologically plausible and has been shown in a number of cohorts. Jankowska et al. found iron deficiency to be a frequent abnormality in chronic systolic heart failure and associated with poor functional status and adverse outcomes, even after adjusting for anemia [4]. More recent Swedish registry data has validated that iron deficiency is often not identified despite its high prevalence and clinically relevant associations with outcomes in routine heart failure populations [5].

Iron deficiency is also important outside the context of reduced ejection fraction. A recent study of patients with heart failure with preserved ejection fraction (HFpEF) showed that patients with iron deficiency had more severe symptoms and functional capacity and disease biomarker abnormalities [6]. Biomarker studies have also indicated that TSAT and serum iron are tightly linked to clinical outcomes, supporting the notion that biologically available iron is a key factor in determining heart-failure phenotype [7]. Recent analyses over the entire range of ejection fraction have further clarified the relationship between the various definitions of iron deficiency and symptoms and prognosis [8].

Although there is growing evidence, many tertiary-care centers do not fully understand the relationship between iron deficiency and day-to-day indicators of heart-failure severity, especially in the context of anemia as the primary reason for iron testing. A prospective evaluation that includes NYHA class, LVEF, natriuretic peptide concentration, exercise capacity and short-term readmission may better determine the clinical significance of iron deficiency. Thus, the purpose of this study was to estimate the prevalence of iron deficiency in adult patients with CHF and its relationship to clinical, echocardiographic, biochemical, functional, and six-month clinical severity.

MATERIALS AND METHODS

Study design and setting: Prospective observational study was done in the Department of General Medicine and Cardiology of a tertiary care teaching hospital over 18 months. Consecutive adult patients with a known history of CHF who were clinically stable in outpatient care or clinically decongested prior to discharge from the hospital were eligible to be screened for enrolment.

Sample size: The sample size was calculated to be able to detect a difference of about 25% between the proportion of NYHA class III-IV symptoms between the iron deficient and non-iron deficient groups with 80% power and a two-sided alpha of 0.05. Allowing for loss to follow-up, at least 160 participants were targeted. A total of 13 patients were excluded from the final analysis of 181 patients screened.

Patients were eligible if they were 18 years or older and had clinical heart failure and echocardiographic structural or functional cardiac abnormality. Reduced and preserved EF phenotypes were allowed. The exclusion criteria were active gastrointestinal or other clinically significant bleeding, blood transfusion within the last three months, intravenous iron within six months, oral iron within four weeks, known hematologic malignancy, active infection, chronic inflammatory disease requiring immunosuppression, severe chronic liver disease, dialysis-dependent kidney disease, acute coronary syndrome within four weeks, and failure to complete a 6-minute walk test.

Clinical evaluation: Demographic variables, etiology of heart failure, comorbidities, medication use, resting blood pressure, heart rate and body mass index were documented on a structured case-report form. NYHA functional class was used to determine the heart-failure symptoms at enrollment. Peripheral oedema, elevated jugular venous pressure and pulmonary crepitations were noted if present. Treatment with guideline-directed therapies was continued as per treating physician's discretion, without protocol-driven changes.

Laboratory assessment and definition of iron deficiency: Venous blood was taken in the morning for a complete blood count, serum creatinine, ferritin, serum iron, total iron-binding capacity, and NT-proBNP. TSAT was determined by dividing

the serum iron by the total iron-binding capacity and multiplying by 100. Iron deficiency was considered as ferritin <100 ng/mL or ferritin 100-299 ng/mL and TSAT <20%. Anemia was defined as hemoglobin <13 g/dL in men and <12 g/dL in women. Laboratory reported CKD-EPI equation was used to estimate GFR.

Echocardiography and functional capacity: Standard transthoracic echocardiography was performed by experienced operators using standard chamber quantification. When image quality allowed, LVEF was calculated by the biplane Simpson method. LVEF was used as a continuous variable for analysis, and also was classified as reduced (<40%), mildly reduced (40-49%) or preserved (≥50%). Functional capacity was measured by a standardised 6 minute walk test in a level corridor and the total distance walked was recorded in metres.

Follow-up and outcomes: Participants were followed for six months with regular outpatient visits and by telephone. The main severity comparison was the proportion of patients with NYHA class III-IV symptoms at baseline based on iron status. Secondary outcomes were LVEF, NT-proBNP, 6MWD, anemia, and all-cause or heart-failure readmission. Mortality data were obtained from hospital records or family notification.

Data analysis: SPSS version 26.0 was used for statistical analysis. All data are reported as mean ± SD and n (%). Independent-samples t tests or chi-square tests were used to compare the iron deficient and non-iron deficient groups, as appropriate. Pearson correlation was used to determine the correlation between the TSAT and selected severity variables. The independent association of iron deficiency with NYHA class III-IV was evaluated using multivariable logistic regression, adjusting for age, sex, hemoglobin, estimated GFR and LVEF. Odds ratios (OR) with 95% confidence intervals (CI) were presented. Results with $p<0.05$ were considered statistically significant.

RESULTS

The final cohort comprised 168 patients with a mean age of 61.9±10.8 years; 105 (62.5%) were men. Ischemic heart disease was the most frequent etiology (51.8%), followed by hypertensive and nonischemic dilated cardiomyopathy. Iron deficiency was identified in 102 patients (60.7%). Mean ferritin in the iron-deficient group was 82±61 ng/mL and mean TSAT was 14.8±4.1%, compared with 218±96 ng/mL and 27.6±6.9% in the non-iron-deficient group (both $p<0.001$). Anemia was more common with iron deficiency (58.8% vs 33.3%, $p=0.001$), although a substantial proportion of iron-deficient patients were not anemic (Table 1).

Iron deficiency was consistently associated with markers of greater disease severity. NYHA class III-IV symptoms were present in 71.6% of iron-deficient patients compared with 42.4% of those without iron deficiency ($p<0.001$). Mean LVEF was lower (31.8±8.4% vs 36.7±9.1%, $p=0.001$), NT-proBNP was higher (3280±1710 vs 2240±1390 pg/mL, $p<0.001$), and mean 6MWD was shorter (278±82 vs 342±88 m, $p<0.001$). TSAT correlated positively with 6MWD ($r=0.38$, $p<0.001$) and LVEF ($r=0.23$, $p=0.003$), and inversely with NT-proBNP ($r=-0.31$, $p<0.001$) (Table 2).

During six months of follow-up, heart-failure readmission occurred in 32 iron-deficient patients (31.4%) and 10 non-iron-deficient patients (15.2%; $p=0.018$). All-cause readmission was also higher in the iron-deficient group (38.2% vs 21.2%, $p=0.023$). Mortality was numerically higher with iron deficiency (7.8% vs 4.5%) but did not reach statistical significance ($p=0.40$). In multivariable analysis, iron deficiency remained independently associated with NYHA class III-IV symptoms (adjusted OR 2.48, 95% CI 1.24-4.97; $p=0.010$) after adjustment for age, sex, hemoglobin, renal function, and LVEF. Lower TSAT also remained independently related to shorter 6MWD in a linear model (Table 3).

Table 1. Baseline characteristics according to iron status

Characteristic	Iron deficient (n=102)	No iron deficiency (n=66)	p-value
Age (years)	62.6±10.9	60.8±10.5	0.30
Male sex, n (%)	61 (59.8)	44 (66.7)	0.37
Hemoglobin (g/dL)	11.8±1.8	12.9±1.7	<0.001
Anemia, n (%)	60 (58.8)	22 (33.3)	0.001
Ferritin (ng/mL)	82±61	218±96	<0.001
TSAT (%)	14.8±4.1	27.6±6.9	<0.001
eGFR (mL/min/1.73 m²)	67±19	72±18	0.09
Ischemic etiology, n (%)	55 (53.9)	32 (48.5)	0.49

TSAT, transferrin saturation; eGFR, estimated glomerular filtration rate.

Table 2. Heart-failure severity measures according to iron status

Severity measure	Iron deficient (n=102)	No iron deficiency (n=66)	p-value
NYHA class III-IV, n (%)	73 (71.6)	28 (42.4)	<0.001
LVEF (%)	31.8±8.4	36.7±9.1	0.001
NT-proBNP (pg/mL)	3280±1710	2240±1390	<0.001
6-minute walk distance (m)	278±82	342±88	<0.001
Resting heart rate (beats/min)	83±13	79±12	0.047
Peripheral edema, n (%)	43 (42.2)	18 (27.3)	0.049

NYHA, New York Heart Association; LVEF, left ventricular ejection fraction; NT-proBNP, N-terminal pro-B-type natriuretic peptide.

Table 3. Six-month outcomes and multivariable severity analysis

Outcome/analysis	Iron deficient	No iron deficiency	Effect estimate / p-value
HF readmission, n (%)	32 (31.4)	10 (15.2)	p=0.018
All-cause readmission, n (%)	39 (38.2)	14 (21.2)	p=0.023
All-cause mortality, n (%)	8 (7.8)	3 (4.5)	p=0.40
Adjusted association with NYHA III-IV	—	—	OR 2.48 (95% CI 1.24-4.97), p=0.010
TSAT association with 6MWD*	—	—	β=0.29, p<0.001

**Standardized coefficient adjusted for age, sex, hemoglobin, eGFR, and LVEF. HF, heart failure; OR, odds ratio; CI, confidence interval; 6MWD, six-minute walk distance.*

DISCUSSION

In this prospective observational study, about 60% of adults with CHF had iron deficiency, which was linked to a clearly more severe clinical phenotype. Iron deficient patients were more likely to have NYHA class III-IV, lower LVEF, higher NT-proBNP, shorter 6MWD, and higher heart failure readmission rates. Importantly, the relationship between iron deficiency and advanced NYHA class was not accounted for by hemoglobin alone, suggesting that this relationship was not due to anemia alone.

Our prevalence is similar to that described in modern heart failure literature, which describes iron deficiency as one of the most common comorbidities in HF. The AFFIRM-AHF trial was the first to show that iron-deficient patients after acute heart failure (AHF) stabilization are clinically important and that intravenous (IV) ferric carboxymaltose (FC) decreased subsequent hospitalizations for heart failure (HF) but failed to achieve conventional statistical significance [9]. Our observational data do not assess the effectiveness of treatment, but the increased readmission rate among those with iron deficiency points to a similar clinically vulnerable population.

The linkage of symptoms and functional limitation is also consistent with the pathophysiology of iron deficiency. Iron is needed for mitochondrial enzymes, oxygen utilization, and skeletal-muscle energetics; thus, deficiency can affect exercise capacity without affecting hemoglobin. The IRONMAN trial, which tested the efficacy of intravenous iron derisomaltose in patients with heart failure and iron deficiency, also reinforced the clinical significance of correcting iron deficiency, especially for the prevention of recurrent heart failure events over a long-term follow-up [10]. The current finding of a 64-metre lower mean 6MWD in iron deficient patients is consistent with a clinically relevant restriction in functional capacity. Recent randomized evidence has yielded a more complex picture of hard outcomes. Ferric carboxymaltose failed to significantly improve the primary hierarchical composite endpoint in ambulatory HFrEF in HEART-FID even after iron deficiency was biologic corrected [11]. Similarly, the primary cardiovascular outcome endpoints for FAIR-HF2 were not shown to be statistically significantly reduced, but there are some trends and secondary observations that continue to guide

patient selection [12]. These trials highlight the importance of iron deficiency as a disease burden indicator, and the level of benefit from iron replacement may vary by population, baseline TSAT, timing, formulation and endpoint.

Despite this, there is consistent evidence for the reduction in recurrent heart-failure hospitalization with intravenous iron from meta-analysis of major randomized trials. A new meta-analysis by Graham and colleagues found that iron supplementation by IV injection decreases hospitalizations for heart failure in iron deficient heart failure, but there is less clarity about the effect on cardiovascular or all-cause mortality [13]. A new meta-analysis of randomized trials also found similar benefits in terms of hospitalization and functional outcomes [14]. This is echoed by our six month observational follow up: readmission varied markedly with iron status while the small difference in mortality was not statistically significant.

One clinically important finding that we have is the separation of iron deficiency and anemia. Iron deficiency was independently associated with NYHA severity even after adjusting for hemoglobin, with more than 40% of the iron-deficient participants not being anemic. This aligns with current focus on direct measurement of iron availability instead of hemoglobin as an indirect measure. Martens and Tang have pointed to the prognostic value of TSAT in heart failure and suggested that the level of iron availability in the circulation may be more informative than ferritin [15]. This was supported by the significant correlation of TSAT with functional capacity, LVEF and NT-proBNP in our cohort.

Iron deficiency is also relevant throughout the ejection-fraction range. In HFpEF, observational studies have revealed a high prevalence of ID and its link with worse prognosis and functional status, but this link with objective exercise capacity has been inconsistent compared to HFrEF [16]. A variety of ejection-fraction phenotypes were included in our cohort, but the overall association was still apparent after adjusting for LVEF. This indicates that iron deficiency is a systemic modifier of the severity of heart failure, and not just a marker of decreased systolic function.

Ferritin is an acute-phase reactant and caution should be used in interpreting the ferritin level in chronic heart failure. A prospective study that compared diagnostic strategies showed that patients with TSAT <20% were more likely to be identified as having poor survival than patients with low ferritin alone [17]. The present guideline definition was kept in our study as it is widely used and is comparable to clinical trials, but our correlation data also suggest TSAT as a very informative marker. Further research is needed to explore the possibility of using serum iron, soluble transferrin receptor, hepcidin, or other markers of tissue iron availability in addition to the conventional parameters.

The results are also applicable to the tertiary-care setting in India where anemia is prevalent and iron studies are not routinely performed in patients with heart failure, unless hemoglobin is low. Iron deficiency has been recently reported as a clinically significant and underdiagnosed condition in HFrEF in India [18]. Our data confirm this concern by showing that there was a large group of individuals with iron-deficiency who were not anemic. Routine ferritin and TSAT testing could thus help to detect patients with symptom burden and risk that may be attributed to cardiac dysfunction alone.

There are several possible mechanisms that could account for the association of iron deficiency with more severe CHF. Chronic inflammation leads to hepcidin-mediated sequestration of iron, intestinal edema can decrease iron absorption, renal dysfunction can affect erythropoiesis and iron handling, and antithrombotic therapy can lead to occult gastrointestinal loss. However, there may be a bi-directional relationship, as severe heart failure can lead to iron deficiency, and vice versa. The cross-sectional baseline associations in this prospective cohort do not allow for determining the direction of association, but the consistency of the associations across the symptoms, biomarkers, exercise capacity, and readmission suggests clinical relevance.

There are some limitations of the study. It was performed in one tertiary centre and may overestimate patients with more advanced disease. Iron status was measured at enrolment and was not routinely remeasured at follow-up. No inflammatory markers were measured and there was insufficient power to assess mortality. There was no protocolisation of iron deficiency treatment after enrolment, meaning that subsequent treatment might have had an impact on 6-month outcomes. These limitations notwithstanding, prospective follow-up, standardized TSAT/ferritin assessment, objective exercise testing, echocardiography, and multivariable analysis support the observation that iron deficiency identifies a more severe heart-failure phenotype.

CONCLUSION

Iron deficiency was very common among adults with CHF and was linked to more severe CHF symptoms, lower EF, higher NT-proBNP, shorter 6MWD, and higher 6-month heart failure readmission rates. These relationships continued to exist, albeit in part independently of anemia, suggesting that ferritin and transferrin saturation should be used to assess rather

than hemoglobin alone. The regular detection of iron deficiency may help to better characterize risk and assist the clinician in identifying a potentially modifiable factor that contributes to functional limitation and re-hospitalization.

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