Original Article

Iron Deficiency as a Predictor of Disease Severity in Heart Failure: An Observational Study

Ria Garg[1], Poorvi Varshney[2], Vishwanayak[3], V.K. Singh[4], Rohit Kumar Jain[1], Vedant Dhaval Kulkarni[1], Jinen Vipul Maisheri[1], Seemans Jain[1]

[1]Postgraduate Student, Department of Medicine, Teerthankr Mahaveer Medical College & Research Centre, Moradabad, UP, India. [2]Assistant Professor, Department of Medicine, Teerthankr Mahaveer Medical College & Research Centre, Moradabad, UP, India. [3]Professor, Department of Medicine, Teerthankr Mahaveer Medical College & Research Centre, Moradabad, UP, India. [4]Professor & Head, Department of Medicine, Teerthankr Mahaveer Medical College & Research Centre, Moradabad, UP, India

Abstract

Background: Heart failure (HF) is a major cause of morbidity and mortality worldwide. Iron deficiency has now been established as an important comorbidity, either absolute or functional type that may make HF worse by increasing symptoms, limiting exercise tolerance and independently associating with poor outcomes irrespective of anaemia. There is limited data regarding the prevalence and clinical importance of ID among Indian HF patients. To study aims to evaluate the prevalence, types, and clinical correlates of ID in hospitalized HF patients and to determine its association with severity of HF. **Material and Methods:** This cross-sectional study of 162 HF patients was conducted in a tertiary care hospital in Western Uttar Pradesh, India. Patients underwent a thorough clinical examination and determination of NYHA functional classification. They completed a CBC and biochemical studies, including serum iron, ferritin, transferrin saturation (TSAT), and total iron binding capacity (TIBC). ID was classified as absolute ID (ferritin <100 µg/L) or functional ID (ferritin 100-300 µg/L and TSAT <20%). Statistical analyses were performed using Chi square test and logistic regression. **Results:** The mean age of patients was 56.9 years, with the majority being female. Anaemia was found in 66% of patients. ID was found in a total of 50% of patients, 21% had absolute ID, and 29% had functional ID. The proportion of patients with ID increased in relation to worsening NYHA class (p<0.01). Logistic regression revealed the independent predictors of ID to be severe HF (NYHA III-IV) (OR 2.74, 95% CI: 1.25-5.93, p=0.009), low hemoglobin (OR 0.79, p=0.004), and low TSAT (OR 1.34, p=0.003). **Conclusion:** ID is highly prevalent among HF patients and is closely related to advanced stage of disease severity among an Indian HF population. Routine screening and early management of ID should be incorporated into HF care as improved outcomes can be achieved.

Keywords: Heart failure, iron deficiency, anaemia, NYHA class, transferrin saturation.

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Introduction

Heart failure (HF) is a notable global health challenge that affects 1–2% of the general population with a significant increase in prevalence in those aged over 70 years.[1,2] HF is linked to frequent hospital admissions, reduced functional capacity, decreased quality of life, and high mortality. There would be some prospect for improvement following advancements in medications and devices, but outcomes are still poor, and the presence of comorbidities detracts from any opportunities for improvement.[3,4]

One problem comorbidity is iron deficiency (ID), which has been established as an independent factor in worsening HF. However, ID is not limited to HF; it is prevalent around the globe affecting about a third of the population.[5]. In HF, ID proportions are between 30–50%, independent of anaemia.[6] It is now understood that ID does not just coexist with HF but directly contributes to symptom burden, exercise dysfunction and hospital readmissions in patients with HF who have iron deficiency.[7-9] Furthermore, it is now established that ID is a significant predictor of poor outcome in patients with HF including morbidity and mortality.[10]

From a physiological perspective, iron is important for haemoglobin production and oxygen transportation, and mitochondrial energy metabolism. In patients with HF, the increased energy demand imposed on the failing myocardium is exacerbated by iron deficiency (ID), which affects mitochondrial metabolism, reduces ATP production, and negatively impacts cardiac and skeletal muscle.[7,11,12] The result is the typical symptoms of fatigue, dyspnoea, and functional decline. Unlike other comorbidities, ID is a modifiable risk factor, and intravenous iron treatment has improved exercise capacity, New York Heart Association (NYHA) functional class, and quality of

Address for correspondence: Dr. Ria Garg, Postgraduate Student, Department of Medicine, Teerthankr Mahaveer Medical College & Research Centre, Moradabad, UP, India.
E-mail: riagarg199820@gmail.com

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life.[8,13,14]

Clinically, ID is distinguished into two categories: absolute iron deficiency (AID), which is defined as serum ferritin <100 µg/L, and functional iron deficiency (FID), which is defined as serum ferritin 100–300 µg/L with transferrin saturation (TSAT) <20%.[3,5,15] AID is an indicator of depleted stores of iron, while FID indicates impaired utilization or mobilization of iron caused by chronic inflammation and elevated hepcidin levels, both common in patients with HF.[16] FID is important, as inflammatory states may elevate ferritin levels, leading the clinician to overlook an iron deficiency. Given that ferritin, TSAT, and total iron-binding capacity are used together in defining iron deficiency.[17,18]

Several major trials, such as the FAIR-HF trial and the CONFIRM-HF trial, have shown that intravenous ferric carboxymaltose significantly improved symptoms, exercise tolerance and decreased hospitalizations compared to placebo in patients with heart failure and iron deficiency, regardless of whether they had anaemia or not.[7,8] More recently, the AFFIRM-AHF trial added to these trials, by showing that intravenous ferric carboxymaltose provided the same benefits demonstrated in patients with acute decompensated HF.[7] Oral iron supplementation has been shown to have limited efficacy in the treatment of iron deficiency in patients with heart failure due to impaired gastrointestinal absorption and hepcidin elevation through inflammation.[12,13]

Most of the literature available on iron deficiency in patients with heart failure has been published on western populations.[16] In India, the vegetarian lifestyle, micronutrient deficits and high prevalence of hemoglobinopathies could have a different relationship with the iron status. ○ Despite heart failure being a growing public health issue, we had limited information on the prevalence of iron deficiency and it's clinical significance in Indian patients. But the initial studies performed here have shown that the use of intravenous iron therapy can decrease hospitalizations and improve patient outcomes in Indian heart failure populations.[17]

As a result of the findings above, this study was designed to see the prevalence, classification and clinical significance of iron deficiency amongst patients in heart failure. Other elements added into our study included the study of iron deficiency amongst hospitalized patients in Western Uttar Pradesh, its relationship with the severity of heart failure defined by the NYHA classification and the diagnostic qualities of haematological and biochemical markers.

Materials and Methods

Study Design and Setting: This cross-sectional observational study was carried out in the Department of General Medicine at Teerthanker Mahaveer Medical College and Research Centre (TMMC&RC), Western Uttar Pradesh. Institutional Ethics Committee approval was obtained and the study was conducted over a period of 12 months, starting from the approval date. All consecutive patients above 18 years of age that were admitted with heart failure (HF) based on clinical criteria (Boston criteria) and/or radiological/echocardiographic findings were included.

Inclusion and exclusion criteria: All patients over the age of 18 years with evidence of heart failure (HF) using clinical or radiological criteria were included in the study. Patients who had chronic kidney disease, congenital heart disease, or non-cardiac cause for iron deficiency such as malignancy, gastrointestinal bleeding, or hemorrhoids were excluded. Informed consent was obtained from all participants. Data collection was conducted following the Declaration of Helsinki principles and all participants were kept strictly confidential, and all possible measures to protect the participants' identities and data were enforced to full effect.

Patient Evaluation

- **Clinical Assessment:** All patients were classified according to the New York Heart Association (NYHA) functional classification.
- **Laboratory Investigations:**
  - Complete hemogram (hemoglobin, red cell indices).
  - Serum iron (S. Iron).
  - Total iron-binding capacity (TIBC).
  - Transferrin saturation (TSAT).
  - Serum ferritin.
- **Echocardiography:** All patients underwent 2D echocardiography for confirmation of HF.
- **Anemia:** Hemoglobin <13 g/dL in males and <12 g/dL in females (WHO criteria).
- **Absolute Iron Deficiency (AID):** Serum ferritin <100 µg/L.
- **Functional Iron Deficiency (FID):** Serum ferritin 100–300 µg/L with TSAT <20%.

Hospital records and referrals from physicians were used to identify eligible patients for the study. Investigators collected prospectively and used a structured proforma to record data. The investigators captured both absolute and functional ID with or without the presence of anemia. SPSS version 25 was utilized to conduct the analysis of the data. Continuous variables were presented as means ± standard deviation (SD), whereas categorical variables were expressed as percentages. For group analysis, the Chi-square test, t-test, ANOVA, and regression analysis were utilized as appropriate. A logistic regression model was used to assess the relationship between ID and the severity of heart failure. Statistical significance was accepted at p-value <0.05.

Results

Baseline characteristics: 162 HF patients included; mean age 56.9 ±16.1 years, 40.1% males. Mean Hb was 10.9 ±2.6 g/dL, ferritin 252.4 ±196.3 µg/L, TSAT 19.0 ±11.7%. ID prevalence: AID 21%, FID 29%, No ID 50%.

[Table 1], summarizes demographic and clinical parameters of the study cohort. The mean age was 56.9 years, with a female predominance, and two-thirds of patients were anaemic. This table summarizes demographic and clinical parameters of the study cohort. The mean age was 56.9 years, with a female predominance, and two-thirds of patients were anaemic.

Table 1: Baseline Characteristics of Study Population

Variable	Mean ± SD / %
Age (years)	56.9 ± 16.1
Male	40.1%
Female	59.9%
Hb (g/dL)	10.9 ± 2.6
Serum Ferritin (µg/L)	252.4 ± 196.3
TSAT (%)	19.0 ± 11.7
Serum Iron (µg/dL)	79.7 ± 37.2
TIBC (µg/dL)	399.5 ± 82.7
Absolute ID	21.0%
Functional ID	29.0%
No ID	50.0%

[Figure 1] shows that Most patients were in NYHA class II and III, while only a small proportion presented in class I or IV, highlighting the predominance of moderate-to-severe HF in this cohort.

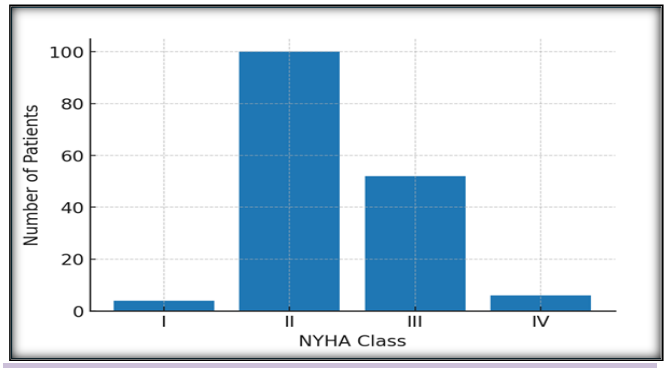


Figure 1: Distribution of patients by NYHA class. Proportion of HF patients across NYHA functional classes I–IV.

[Table 2] shows that, Half of the patients had iron deficiency, with functional ID (29%) being more common than absolute ID (21%).

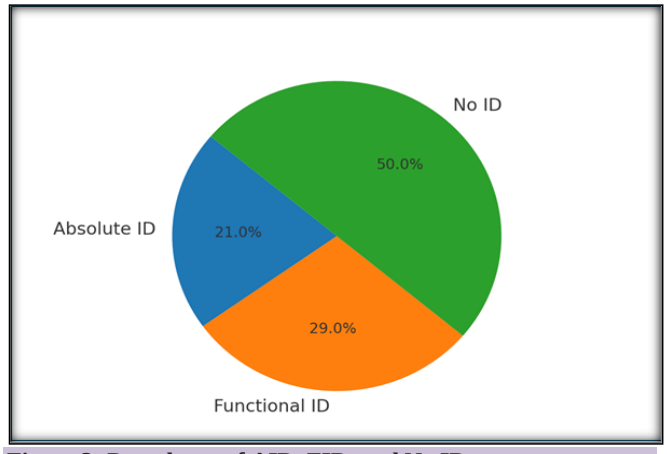


Figure 2: Prevalence of AID, FID, and No ID

NYHA association: Chi-square showed significant association between ID and NYHA class (p=0.0246). ID prevalence increased with worsening HF severity.

Table 2: Prevalence of Iron Deficiency

Type	Patients	%
Absolute ID	34	21.0
Functional ID	47	29.0
No ID	81	50.0

Table 3: NYHA Class vs Iron Deficiency

NYHA	Absolute ID	Functional ID	No ID
I	1	1	2
II	21	29	50
III	11	15	26
IV	1	2	3

[Table 3] shows that the prevalence of both absolute and functional iron deficiency increased with worsening NYHA class, being highest in NYHA III–IV patients.

[Figure 4-6] this composite figure shows the relevant haematological and biochemical parameters for the NYHA classes. Haemoglobin appeared to decrease with worsening NYHA class whereas serum ferritin did not change much. The scatter plot of ferritin versus transferrin saturation (TSAT) illustrated an inverse relationship with only a degree of separation in levels between the values which could signify an altered availability to iron but preserved levels of ferritin.

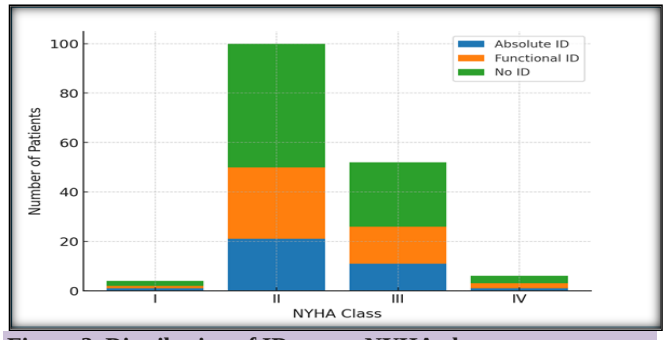
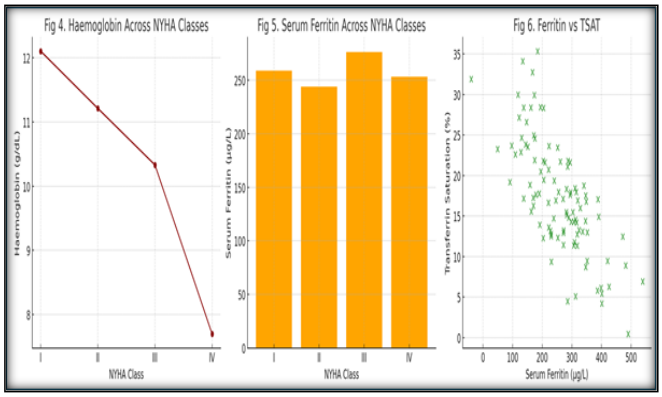


Figure 3. Distribution of ID across NYHA classes



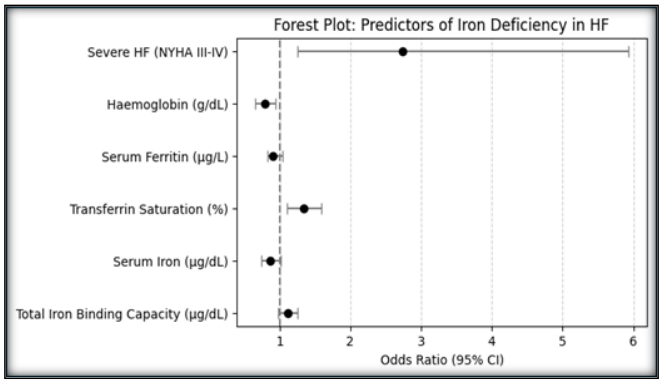
Regression analysis confirmed severe HF (NYHA III–IV) was independently associated with ID (OR=2.74, 95% CI 1.25–5.93, p=0.009).

Logistic Regression Analysis: A multivariate logistic regression was performed to assess the relationship of iron deficiency (ID) with the clinical/biochemical variables. Having severe HF (NYHA III-IV) was significantly related to ID (OR 2.74, 95% CI: 1.25-5.93, p=0.009). The haemoglobin was inversely associated with ID (OR 0.79, 95% CI: 0.66-0.94, p=0.004) and transferrin saturation was positively predictive (OR 1.34, 95% CI: 1.11-1.59, p=0.003). Serum ferritin, serum iron, and TIBC showed a trend but lacked significance.

Table 4: Logistic Regression Results

Variable	Odds Ratio	95% CI	p-value
Severe HF (NYHA III–IV)	2.74	1.25–5.93	0.009
Hemoglobin (g/dL)	0.79	0.66–0.94	0.004
Serum Ferritin (µg/L)	0.91	0.83–1.04	0.089
Transferrin Saturation (%)	1.34	1.11–1.59	0.003
Serum Iron (µg/dL)	0.87	0.75–1.02	0.075
TIBC (µg/dL)	1.12	0.98–1.26	0.095

[Table 4] shows that 66% of patients were anemic, and hematological/iron parameters progressively declined with advanced NYHA class.



[Figure 7] demonstrates The OR and 95% CI for significant predictors of ID in HF shown in a forest plot. ID was highly associated with severe HF (NYHA III–IV) (OR = 2.74, 95% CI: 1.25–5.93, p = 0.009) suggesting that having advanced heart failure almost triples an individual odds of being ID. The higher rate of ID, with its strong association with anaemia in HF demonstrated by significant predictors of Hb (OR = 0.79, p = 0.004) and transferrin saturation (OR = 1.34, p = 0.003). Although they showed trends S.ferritin, S.iron, and TIBC were not statistically significant.

Discussion

We assessed the prevalence and clinical significance of iron deficiency (ID) in patients with heart failure (HF) admitted to a tertiary care hospital in Western Uttar Pradesh. Nearly half had ID (21% absolute ID, 29% functional ID), and ID was significantly associated with advanced HF (NYHA III–IV). Our findings highlight the burden of ID in HF and its importance as a reversible comorbidity.

Our findings are comparable to similar international studies that suggest 30-50% of HF patients have ID, regardless of anaemia.[3,6,18,19] The prevalence observed in our cohort (50%) is higher than the literature indicates, and may be related to common nutritional deficiencies, vegetarian eating practices, and poor access to healthcare for Indian patients.[16,17]

It was noteworthy that functional ID (FID), was seen more often than absolute ID (AID), especially among patients with advanced NYHA class. This is consistent with earlier studies showing that systemic inflammation and elevated hepcidin levels inhibit iron mobilization in HF, leading to functional deficiency instead of absolute deficiency.[20,21] Therefore, if we solely use ferritin to diagnose ID we are possibly underestimating ID as ferritin is an acute-phase reactant elevated during states of chronic inflammation.[22,23] By using transferrin saturation (TSAT) and total iron-binding capacity (TIBC) in addition to ferritin we would get a more accurate assessment of iron status in HF patients.

We also determined that both anaemia (prevalent in 66% of our cohort) and ID did not completely overlap, which supported previous evidence that ID is clinically important in the absence of anaemia.[24,25] Patients with ID, however, may have decreased exercise tolerance, increased fatigue, and a reduced quality of life,[7,8] and therefore it is important that clinical assessment for ID is undertaken on all patients with HF, not only those with low haemoglobin.

The logistic regression analysis also found that severe HF (NYHA III-IV), low haemoglobin, and low transferrin saturation were independent predictors of ID. These findings align with previous studies that have reported a related trend that worsening functional class corresponds to increased prevalence of ID.[26,27] Our findings provide evidence that increasingly progressive HF pathophysiology, with systemic congestion and chronic inflammation, is a relevant way to understand our disturbance of iron homeostasis.

Therapeutically, intravenous iron supplementation has shown promise. For example, randomized controlled trials, such as

FAIR-HF and CONFIRM-HF, demonstrated improvements in NYHA class, six-minute walk test distance, and patient-reported outcomes with intravenous ferric carboxymaltose in patients with HF, regardless of anaemia status. More recently, the AFFIRM-AHF trial revealed a reduction in readmission rates among patients with acute decompensated HF after intravenous iron therapy. In India, early evidence has demonstrated similar results. Our results, which highlighted a strong presence of FID, point to benefits of routine screening and early intravenous supplementation in this population.

Limitations: The study was single-centre and had a rather small sample size limiting generalizability. Long-term outcomes after treating ID were not assessed, and biomarkers related to ID such as hepcidin or soluble transferrin receptor were not taken. Nonetheless, the study offers important insights into an Indian HF population where information is limited.

Implications for practice and research: Healthcare providers should adopt routine screening for iron deficiency (ID) with ferritin and TSAT into heart failure (HF) care pathways. Future multicentre longitudinal studies in India could examine the long-term effects of intravenous iron therapy on hospitalization, mortality, and quality of life outcomes in those with HF.

Conclusion

Iron deficiency was common among patients with heart failure in our study. Nearly half of the cohort were iron deficient, and functional deficiency was more common than absolute deficiency in those with low haemoglobin levels. The presence of ID was independently correlated with advanced NYHA class, lower haemoglobin and transferrin saturation. Routine screening for iron status, with ferritin and TSAT, should be incorporated into HF management. Intravenous iron has the potential to improve outcomes in this high-risk population if timely correction of iron deficiency occurs.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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