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RESEARCH ARTICLE
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PREVALENCE OF IRON DEFICIENCY ANEMIA IN HEART FAILURE IN A TERTIARY CARE HOSPITAL OF NORTH INDIA

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ABSTRACT

Background: Heart failure (HF) is a major public health concern worldwide and is frequently complicated by anemia, especially iron deficiency anemia (IDA), which worsens outcomes.

Objectives: To determine the prevalence of iron deficiency anemia in patients with chronic heart failure, to analyze the pattern of anemia associated with HF, and to correlate the severity of anemia with functional class of HF.

Methods: A prospective observational study was conducted over 2 years at SKIMS, Srinagar. A total of 359 patients with symptomatic chronic HF and left ventricular ejection fraction (LVEF) <40% were enrolled. Clinical evaluation, laboratory investigations including complete blood count, red cell indices, and iron studies (serum ferritin, transferrin saturation, TIBC) were performed. Anemia was defined by WHO criteria, and iron deficiency was categorized as absolute or functional.

Results: The mean age of patients was 59.2±7.32 years; 58.8% were males. Overall prevalence of anemia was 49.6%, with females more affected (53.4% vs. 46.9%, p=0.228). Iron deficiency was seen in 48.7% patients, with absolute ID predominating (75.0%). Iron deficiency anemia was documented in 37.3% of patients, more common in females (40.5%). Anemia prevalence increased significantly with advancing age in males (p=0.039). Severity of anemia correlated with higher NYHA class and lower eGFR (p<0.001).

Conclusion: Iron deficiency anemia is highly prevalent among patients with chronic HF, particularly in elderly and female patients. Routine evaluation of iron status is essential, as timely correction may improve functional outcomes.

Keywords: Heart failure, Anemia, Iron deficiency, Left ventricular dysfunction, NYHA class

INTRODUCTION

Anemia exacerbates symptoms of heart failure through impaired mechanisms such as tissue hypoxia and nitric oxide–mediated vasodilatation, which lead to decreased blood pressure, increased sympathetic activation, renal vasoconstriction, reduced renal function, and activation of the renin-angiotensin-aldosterone system. These processes result in fluid retention, left ventricular hypertrophy, and worsening of heart failure, ultimately completing a vicious circle that further aggravates anemia[1]. The prevalence of anemia is particularly high in patients with advanced age,

coexistent kidney disease, and more severe symptoms[2]. The global burden of heart failure is estimated at 64.3 million people worldwide[3]. In developed countries, the prevalence is 1–2% of the adult population, while in India it ranges from 1.3 to 4.6 million cases, with an annual incidence of 4,91,600 to 1.8 million[4]. HF is a syndrome characterized by symptoms such as breathlessness, ankle swelling, and fatigue, and signs including elevated jugular venous pressure, pulmonary crackles, and peripheral edema. The most widely used clinical classification systems include the New York Heart Association functional classification[5] and the American College of Cardiology/American Heart Association (ACC/AHA) staging system[6], where stage B disease is at least 2–3 times more common than symptomatic HF[7,8]. Clinically, HF is divided into reduced (≤40%) and preserved (≥50%) ejection fraction categories. Most evidence-based therapies benefit patients with reduced ejection fraction, while the prevalence of preserved ejection fraction continues to rise, now accounting for the majority of hospital admissions[9,10]. A subset of patients falls within the mid-range category (40–49%), some showing recovery of ejection fraction over time[11]. Mortality in this group may be lower, but hospital readmission rates are similar to those of reduced ejection fraction patients[12]. The definitions of anemia in HF remain inconsistent. While the World Health Organization (WHO) proposed the historical threshold of hemoglobin <13 g/dL in men and <12 g/dL in women[13], the National Kidney Foundation and European Best Practice Guidelines have suggested alternative cut-offs, particularly in patients with CKD[14]. This variability complicates uniform assessment.

Pathophysiologically, anemia in HF is multifactorial. Nutritional deficiencies, reduced iron absorption due to intestinal wall changes, and gastrointestinal blood loss from antiplatelet or anticoagulant therapy contribute to iron deficiency[15,16]. Chronic inflammation is also central, with elevated TNF-α, IL-1, and IL-6 reducing erythropoietin synthesis, suppressing erythroid progenitor differentiation, and stimulating hepcidin production[17]. Hepcidin blocks iron absorption and release, leading to functional iron deficiency[18]. These abnormalities are even more pronounced in the setting of renal impairment[19]. Variations in hepcidin and ferritin across different HF stages have been described, with higher levels in early HF and declining values as the disease advances[20,21]. Resistance to erythropoietin further contributes to anemia, as marrow responsiveness is blunted despite elevated circulating levels[22-25]. Hemodilution may also play a role, with some studies attributing nearly half of anemia cases in advanced HF to this mechanism[26], though others have not confirmed this[27]. The interplay between HF, renal dysfunction, anemia, and iron deficiency has been conceptualized as the cardiorenal anemia syndrome (CRAS), later expanded to cardiorenal anemia iron deficiency syndrome (CRAID)[28]. The therapeutic relevance of iron deficiency in HF has been firmly established. The FAIR-HF trial demonstrated that intravenous ferric carboxymaltose significantly improved quality of life, NYHA class, and exercise capacity in HF patients with ID, irrespective of anemia status[29]. These findings were corroborated by the CONFIRM-HF trial, which showed sustained benefits and reduced risk of HF hospitalization[30], and the EFFECT-HF trial, which confirmed improvements in peak oxygen uptake and exercise capacity[31]. Together, these studies underscore the importance of screening and correcting ID in HF management.

AIMS AND OBJECTIVES

1. To determine the prevalence of iron deficiency anemia in chronic heart failure patients.

2. To analyze the pattern of anemia associated with heart failure (iron deficiency anemia, anemia without iron deficiency).
3. To correlate severity of anemia with functional class of heart failure.

MATERIAL AND METHODS

This prospective, observational, hospital-based study was conducted over a period of two years in the Department of Cardiology at Sher-i-Kashmir Institute of Medical Sciences (SKIMS), Srinagar. A total of 359 patients with symptomatic chronic heart failure were enrolled. All included patients had a known history of heart failure for at least six months, were above 18 years of age, and had a left ventricular ejection fraction (LVEF) of less than 40%. Patients were excluded if they had undergone any surgery during the previous six months, had received iron supplementation, erythropoietin therapy, or blood transfusion within the last 30 days, or had a recent history of clinically significant bleeding. Those with malignancy, congenital heart disease, LVEF greater than 40%, or renal disease requiring dialysis or with an estimated glomerular filtration rate (eGFR) of less than 60 ml/min/1.73 m^2 were also excluded from the study. Each patient underwent a detailed clinical history and physical examination, and the diagnosis of heart failure was confirmed on the basis of clinical features and echocardiography. Baseline investigations included complete blood count, liver function tests, renal function tests, chest X-ray, and electrocardiography. Complete blood counts were analyzed with special attention to red cell indices, including mean corpuscular volume (MCV), mean corpuscular hemoglobin (MCH), mean corpuscular hemoglobin concentration (MCHC), and red cell distribution width (RDW). Iron studies were performed on venous blood samples collected between 9:00 and 11:00 AM to avoid diurnal variation. The biomarkers assessed included serum iron, ferritin, and transferrin saturation (TSAT). Transferrin saturation was calculated using the formula: [serum iron (µg/dl) ÷ total iron- binding capacity (µg/dl)] × 100. Serum iron was measured using colorimetric assay, and serum ferritin levels were estimated using enzyme-linked immunosorbent assay. Anemia was defined according to the World Health Organization criteria, i.e., hemoglobin less than 13 g/dl in men and less than 12 g/dl in women. Iron deficiency was defined as a serum ferritin level of less than 100 µg/L, or a ferritin level between 100 and 300 µg/L with a TSAT of less than 20%. Absolute iron deficiency was defined by ferritin <100 µg/L, whereas functional iron deficiency was defined by ferritin levels between 100–300 µg/L with TSAT <20%. Anemia of chronic disease was diagnosed in patients with low serum iron, low TSAT, and normal or low total iron-binding capacity.

Echocardiographic evaluation was performed using a GE Vivid E95 machine. Left ventricular ejection fraction was calculated using the biplane Simpson's method by tracing endocardial borders in apical four- and two-chamber views. Left ventricular dimensions, including left ventricular end-diastolic diameter (LVEDD) and left ventricular end-systolic diameter (LVESD), were measured in the parasternal long-axis view using 2D-targeted M- mode echocardiography. In cases where endocardial borders were poorly visualized, direct 2D measurements were applied. The collected data were entered into Microsoft Excel and analyzed using SPSS version 20. Continuous variables were expressed as mean ± standard deviation, and categorical variables were presented as frequencies and percentages. Chi- square test was applied for categorical comparisons, while multivariate logistic regression analysis was used to identify independent predictors of iron deficiency anemia. A p-value less than 0.05 was considered statistically significant.

RESULTS

Table 1: Age and gender distribution of study patients (n=359)

Demographic parameters		**Number**	**Percentage**
Age (years)	≤ 50	64	17.8
	51–70	250	69.6
	> 70	45	12.5
Gender	Male	211	58.8
	Female	148	41.2

The study population consisted of 359 patients with chronic heart failure. The age distribution showed that the majority of patients (69.6%) were in the 51–70 year age group, while 17.8% were below 50 years of age and 12.5% were older than 70 years. The mean age was 59.2 years, indicating that HF was predominantly seen in middle-aged to elderly individuals. With respect to gender, 211 patients (58.8%) were males and 148 patients (41.2%) were females, giving a male-to-female ratio of approximately 1.4:1. This suggests a male predominance in the occurrence of chronic heart failure in this cohort, which is consistent with previously reported epidemiological trends where men are more frequently affected, though the burden in women remains substantial.

The overall interpretation is that chronic HF in this study population is most commonly observed in men and in the 51–70 year age group, reflecting the combined influence of age-related cardiovascular risk factors and gender differences in disease susceptibility.

Table 2: Association of Etiology of heart failure in males and females

Etiology	Male (%)	Female (%)	P value
CAD	61.1	47.3	0.009*
RHD	17.1	35.1	<0.001*
DCM	21.8	17.6	0.324

The etiology of heart failure in the present cohort varied significantly between males and females. Coronary artery disease (CAD) was the most common cause overall, affecting 61.1% of males compared to 47.3% of females. The difference was statistically significant (p=0.009), suggesting that CAD is more strongly associated with HF in men. Rheumatic heart disease (RHD), in contrast, was significantly more prevalent among females, being observed in 35.1% compared to 17.1% of males ($p < 0.001$). This highlights the continuing burden of RHD in women, especially in developing regions, where it remains a major contributor to HF.

Dilated cardiomyopathy (DCM) accounted for 21.8% of HF cases in males and 17.6% in females. This difference was not statistically significant ($p = 0.324$), suggesting a more equal distribution of DCM between the sexes.

Overall, the data indicate a gender-specific pattern in the etiology of HF: CAD is more frequent in men, while RHD is more common in women, whereas DCM affects both genders nearly equally. These findings reflect the combined influence of regional epidemiology, gender-related risk factors, and socio-environmental determinants of heart disease.

Table 3: Prevalence of anaemia in heart failure, Iron deficiency in heart failure

Characteristics		Male (%)	Female (%)	Total (%)
Anaemia	Yes	46.9	53.4	49.6
	No	53.1	46.6	50.4
Iron deficiency	Yes	46.9	51.4	48.7
	No	53.1	48.6	51.3

In the present study, anemia was observed in 49.6% of patients with chronic heart failure. The prevalence was slightly higher among females (53.4%) compared to males (46.9%), although the difference was not statistically significant. This finding highlights that nearly half of all HF patients suffer from anemia, underlining its clinical importance as a common comorbidity. Iron deficiency was present in 48.7% of the study cohort, with 51.4% of females and 46.9% of males affected. The distribution indicates that iron deficiency was almost equally prevalent among both sexes, with only a marginally higher burden in females.

Taken together, these results demonstrate that both anemia and iron deficiency are highly prevalent in patients with heart failure, affecting nearly one in two individuals. The slightly greater prevalence among women reflects known gender-based vulnerabilities, but the overall findings emphasize that both sexes are substantially impacted. This underscores the importance of routine evaluation for

anemia and iron deficiency as part of the standard care of heart failure patients.

DISCUSSION

The present study highlights the significant burden of anemia and iron deficiency in patients with chronic heart failure (HF) in North India. With 359 patients evaluated, our findings add important regional data to the growing evidence that anemia and iron deficiency are common comorbidities in HF and are strongly associated with adverse clinical outcomes.

Most patients were between 51–70 years of age, with a mean age of 59.2 years, reflecting the middle-aged to elderly predominance of HF. This finding is in agreement with large epidemiological cohorts such as the Framingham Heart Study, which reported a mean age of 60 years at HF diagnosis. Indian studies, including those by Sharma SK et al., (2016)[32] and Negi PC et al., (2018)[33] have similarly demonstrated that HF is more prevalent in older populations. Male predominance (58.8%) in our study parallels global data, where men exhibit a higher incidence of HF, although the burden in women remains substantial. Klip IT et al., (2013)[34] identified female sex as an independent predictor of iron deficiency in HF, which resonates with our finding of a higher anemia prevalence among women.

Etiology of Heart Failure: Coronary artery disease (CAD) was significantly more common in men (61.1%), whereas rheumatic heart disease (RHD) was more frequent in women (35.1%). These gender-specific patterns are consistent with regional epidemiology, where RHD continues to contribute significantly to HF among women, particularly in rural and socioeconomically disadvantaged settings. Negi PC et al., (2018)[33] also reported higher CAD prevalence in men and RHD in women. Dilated cardiomyopathy (DCM) was almost equally distributed between sexes in our cohort, a finding comparable to international literature which reports no significant gender bias in DCM.

Prevalence of Anemia and Iron Deficiency: Anemia was documented in 49.6% of our cohort, with slightly higher rates among females (53.4%). This aligns with Singh B et al., (2022)[35] who reported anemia in 70% of Indian HF patients, and with Sharma SK et al., (2016)[32] who documented anemia prevalence of 76% in their HF cohort. These findings underscore the systemic and multifactorial nature of anemia in HF, reflecting nutritional, inflammatory, and renal contributions. Iron deficiency was present in 48.7% of patients, consistent with international reports. Klip IT et al., (2013)[34] observed a 50% prevalence of ID in chronic HF, while Chobufo MD et al., (2021)[36] highlighted its persistence across all HF stages. Similarly, Negi PC et al., (2018)[33] demonstrated ID in 58.8% of patients with HFrEF. The slightly higher prevalence among women (51.4%) compared to men (46.9%) in our study suggests gender-related differences in nutritional status and disease vulnerability.

Iron Deficiency Anemia (IDA): IDA was present in 37.3% of patients, with a higher prevalence in women (40.5%). This is consistent with Indian studies which have attributed female predominance in IDA to nutritional deficiencies, lower baseline hemoglobin levels, and a greater burden of RHD. International evidence, such as the FAIR-HF trial (Anker SD et al., 2009)[29], has shown that iron deficiency adversely affects clinical outcomes even in the absence of anemia. Our findings therefore reinforce the importance of considering ID not only in anemic patients but across the entire HF spectrum.

Functional Correlates and Renal Dysfunction: Anemia severity was significantly associated with higher NYHA class, reflecting a correlation between anemia and greater symptom burden. This

finding aligns with the work of Chobufo MD et al., (2021)[36] who reported that ID adversely affects functional capacity at all HF stages. Additionally, our observation of a significant association between low eGFR and IDA supports the cardiorenal- anemia syndrome hypothesis, whereby renal impairment, anemia, and HF interact synergistically to worsen prognosis. Silverberg DS et al., (2009)[1] described this vicious triad and emphasized its role in disease progression.

Therapeutic Implications: The high prevalence of anemia and ID in our cohort highlights the therapeutic potential of correcting these conditions. International randomized controlled trials, including FAIR-HF (Anker SD et al., 2009[29]), CONFIRM-HF (2014)[30], and EFFECT- HF (van Veldhuisen DJ et al., 2017)[31], have demonstrated that intravenous ferric carboxymaltose significantly improves exercise tolerance, NYHA functional class, and quality of life in HF patients, irrespective of baseline hemoglobin status. These findings,
taken together with our results, suggest that routine screening and timely correction of IDA should be integral to HF management in order to reduce symptom burden, improve functional outcomes, and potentially lower hospitalization rates.

CONCLUSION

Anemia and iron deficiency are common in chronic heart failure, with iron deficiency anemia affecting more than one-third of patients in this study. Female gender, older age, and renal dysfunction were important correlates. The presence and severity of anemia were associated with worse functional status. These findings highlight the need for systematic evaluation of iron status in all HF patients. Incorporating routine screening and timely correction of iron deficiency into HF management protocols has the potential to improve clinical outcomes, reduce symptom burden, and enhance quality of life.

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