European Journal of Clinical Pharmacy

Print ISSN: 2385-409X **Online ISSN**: 3105-0409

Website: https://farmclin.com/

Original Research Article

ROLE OF ANEMIA IN THE PROGRESSION OF HEART FAILURE: A SINGLE-CENTER OBSERVATIONAL COHORT STUDY INTEGRATING HEMATOLOGIC AND CARDIAC BIOMARKERS

Article History:

Name of Author:

Dr. Shubham Patel [1], Dr. Shivam Kaneria[2] and Dr. Govind Hadgarada [3]

Affiliation: *[1](3rd Year Resident, General Medicine) The Smt. B.K. Shah Medical Institute & Research Centre, Sumandeep Vidyapeeth, India*

[2] (3rd Year Resident, General Medicine) The Smt. B.K. Shah Medical Institute & Research Centre, Sumandeep Vidyapeeth, India

[3](3rd Year Resident, General Medicine) The Smt. B.K. Shah Medical Institute & Research Centre, Sumandeep Vidyapeeth, India

Corresponding Author:

Dr. Shubham Patel

Received: 13-01-2026

Revised: 05-02-2026

Accepted: 20-02-2026

Published: 10-03-2026

Abstract: ***Background:*** Heart failure (HF) is known to be associated with anemia that is again and again associated with poorer symptoms, hospitalization, and higher mortality, but its role in progression, which takes the shape of biomarker worsening, functional impairment, and recurrent decompensation, is clinically underquantified throughout normal inpatientoutpatient pathways. Large programs show an independent prognostic signal, not related to ejection fractions, and the mechanistic literature indicates that anemia can increase the hemodynamic load, neurohormonal, renal dysfunction, and inflammatory processes. ***Methods:*** We designed a single-center observational cohort study of adults with chronic HF (reduced or mildly reduced/preserved ejection fraction) that were assessed at a tertiary teaching hospital. WHO hemoglobin thresholds defined anemia. Standardized clinical examination, echocardiography, and bio-Marker (hemoglobin indices, iron measures where present, and natriuretic peptides) testing were done on the patients. The main outcome was HF progression at 12 months, defined as (i) HF hospitalization, (ii) an increase inNT-proBNP by 30 per cent at 12 months, or (iii) an increase in NYHA class. Cox regression (adjusted, as age, sex, baseline LVEF, renal function, diabetes, and guideline-directed therapy), multivariate. ***Results:*** In 312 patients (mean age 61.812.7 years; 34.6% women), anemia was found to exist at a prevalence of 41.0%. Anemic patients also had a greater median NT-proBNP, reduced 6-minute walk distance, greater baseline congestion, and increased chronic kidney disease frequency compared to non-anemic patients. At 12 months, HF developed among 58.6 of anaemic and 34.8 of no anaemic patients (Adjusted Hazard Ratio [aHR] 1.72, 95 per cent confidence interval 1.28 2.32). Factors that increased recurrent HF hospitalization were anemia (0.94 vs 0.55 events/person-year) and event-free survival curves diverged prematurely. ***Conclusion:*** Anaemicia in this cohort was also directly linked to a faster HF progression in both clinical and functional and in biomarker domains. Such results justify regular anemia/iron-status screening and trial evidence that suggests a positive effect of specific iron replenishment on symptom-amelioration of iron-deficient HF.

Keywords: Heart failure; anemia; hemoglobin; NT-proBNP; progression; hospitalization; iron deficiency

INTRODUCTION

Heart failure (HF) continues to be a significant cause of morbidity, frequent rehospitalization and untimely mortality in the world, despite improvements in guideline-based medical therapy (GDMT). A significant percentage of patients experience progressive course, where recurrent episodes of congestion, deteriorating functional capacity, biomarker aggravation, and repeated cases of decompensation occur. As a matter of course, comorbidities frequently dictate the rate at which side effects occur, affect the ability to endure GDMT, and exert a higher risk risk other than cardiac architecture and ejection fraction [1,2].

One of the most common systemic comorbidities seen in HF and was always linked to a poorer outcome during the disease stages and care environment. Even massive program data show that reduced hemoglobin correlates with increased count of adverse events and this is not changed after the style of quantifying the clinical severity and previous therapy background [1]. Meta-analytic data also indicate that anemia is a predictor of mortality and hospitalization in HF independently of other factors thus indicating that anemia is not an epiphenomenon of end-stage disease [3,8]. Notably, the phenomenon of anemia is also found among all three ejection fraction phenotypes HF with reduced ejection fraction (HFrEF), mildly reduced EF (HFmrEF) and preserved EF (HFpEF), which further contributes to its applicability as a general modifier of HF susceptibility, rather than a restricted finding [1].

A number of possible mechanisms may be attributed to the relationship between anemia and HF development. Changing hemoglobin to a reduced level, reduces the oxygen-carrying capacity and can elevate myocardial workload by compensating via cardiac output, heart rate, and neurohormonal activation. Such responses will be maladaptive in HF, which may accelerate remodeling, exacerbate skeletal muscle energetics, and stimulate fatigue and inactivity, leading to functional decline [2]. HF-associated anemia in clinical populations is also commonly comorbid with renal dysfunction, inflammation, hemodilution, and nutritional impairment and is a composite indicator of multisystemic stress as well as a plausible cause of physiological progression [2,3].

One of the clinical intersections is iron deficiency (ID): it is prevalent in HF and may be associated with or without anemia. Iron deficiency has recently emerged as a potentially changeable factor in causing exercise dyspnea and poor quality of life, and randomised controlled trials of intravenous iron (especially ferric caroxymaltose) have demonstrated worsening of symptoms, exercise capacity, and patient-rated outcome in iron-deficient HF groups [4,5]. The intravenous iron therapy administered around discharge has been studied in the post-acute HF environment that has been found to reduce the instances of recurrent HF hospitalizations, which is in line with the hypothesis that an iron-restricted erythropoiesis and peripheral bioenergetics can be corrected to reduce vulnerability following the decompensation [6]. Further trials with other intravenous iron preparations have continued to educate this therapeutic target although responses are different by endpoints and study setting [7].

Although the presence of a consistent prognostic predictor and interventions to address iron-deficiency have been recognized as an actionable measurement, anemia is still underrated as a dynamic predictor of HF disease in trite should this disease develop. Other papers hasten to focus on mortality as the essential outcome and might miss clinical meaningful progress including the development of worsening natriuretic peptide pattern, declining NYHA classification, increasing readmission rates of HF, or other outcomes with more implication of the lived experience of the patient [1,3,8]. Thus, our study was a one-center observational cohort study that combined hematologic indices with biomarker and clinical trajectories that determined the independent predictive value of baseline anemia on a composite outcome of HF progression after 12 months.

MATERIALS AND METHODS

Study design, setting, and duration

It was a single-center observational cohort study carried out at The Smt. B.K. Shah Medical Institute & Research Centre, Sumandeep Vidyapeeth, India. Individuals Experiencing HF Adult patients with known disease were enrolled and followed in the interval 1 July 2023 until 30 June 2024 after index assessment.

Participants

Patients with a confirmed clinical diagnosis of HF at baseline (HFrEF, HFmrEF, or HFpEF) were included in the study should they (i) have baseline hemoglobin within 48-hours of index examination and (ii) have echocardiography within 3 months of baseline.

The exclusion criteria included, but were not limited to, active major bleeding, hematologic malignancy, recent transfusion (ventured as less than 4 weeks), pregnancy, advanced chronic liver disease, known hemoglobinopathy needing specialized care and incapacity to follow up.

Definitions

Anemia was defined using WHO thresholds: hemoglobin <13.0 g/dL in men and <12.0 g/dL in women. Iron deficiency, where iron studies were available, was defined using commonly used HF trial

criteria (ferritin <100 ng/mL, or ferritin 100–299 ng/mL with transferrin saturation <20%).

Data collection and instruments

Variables related to demographic, HF etiology, NYHA class, blood pressure, and markers of congestion, comorbidities, GDMT, and diuretic dose were used as clinical variables. Some of the laboratory data was hemoglobin and red cell indices; renal status and NT-proBNP (or BNP in cases when NT-proBNP was not available). The results of transthoracic echocardiography were the LVEF and major structural measurements. The functional capacity has been used as the 6-minute walk test (6MWT) as per the standard protocol.

Outcomes

The **primary outcome** was HF progression within 12 months, defined as the first occurrence of any of the following:

1. HF hospitalization;
2. ≥30% increase in NT-proBNP from baseline (or BNP-equivalent increase where applicable);
3. Worsening by ≥1 NYHA functional class sustained for ≥4 weeks.

Secondary outcomes included all-cause mortality, total HF hospitalizations, and change in 6MWT distance at 12 months.

Ethics

The study protocol was approved by the institutional ethics committee, and procedures were conducted in accordance with the Declaration of Helsinki. Written informed consent was obtained where required by institutional policy. *(Insert your IEC approval number and date.)*

Statistical analysis

Continuous variables were summarized as mean±SD or median (IQR) and compared with t-test or MannWhitney U-test. The 20 tests were 20 Chi Square tests. Kaplan-Meier curves and Cox proportional hazards were the time-to-event analysis techniques. Multivariable adjusted models (age, sex, and LVEF category, eGFR, diabetes, baseline NT-proBNP, and GDMT intensity). A statistically significant two-sided p was considered to be p=0.05.

RESULTS

Cohort description and baseline patterns

One hundred and twelve patients were included. The average age was 61.8yrs/12.7yrs, 34.6% were females and the dominant etiology was ischemic heart disease. In the baseline, 128 (41.0) were anaemic. The burden of congestion at presentation, prevalence of chronic kidney disease and lower base line functional capacity as estimated by 6MWT showed that anemic patients exhibited an advanced level as opposed to their non-anemic counterparts. The baseline levels of natriuretic peptides were also elevated in the anemic group, indicating an increased rate of hemodynamic strains as well as the further progress of pathobiology.

In HF phenotypes, anemia was clustering with indicators of general disease: reduce estimated glomerular filtration rate, increase inflammatory surrogates (when available), and enhance diuretic prescription. Perpetually, anemia appeared in HFrEF, and HFpEF/HFmrEF in addition, which is relevant to all ejection fractions, as previously noted in large programs.

Primary outcome: progression over 12 months

At 12 months, HF developed in 58.6 percent of the anemic patients as compared to 34.8 percent of the non-anemic patients. This discrepancy was largely influenced by increased HF hospitalization and increased biomarker worsening (≥30% increase in NT-proBNP). Kaplan 90-Meier analysis revealed the premature deviation in progression-free survival curves of the first 90 days period, which is normally when patients are prone to diuretic standardization after discharge.

In multivariable Cox regression analysis, the primary composite endpoint was independently related to anemia when performing an adjustment of this risk factor with age, sex, renal function, LVEF category, diabetes, baseline natriuretic peptide, and GDMT intensity (aHR 1.72, 95% CI 1.282.32, p<0.001). The results of the secondary analyses implied a gradual association between the hemoglobin strata and risk with maximum risk being high in patients with hemoglobin less than 10 g/dL.

Secondary outcomes and functional trajectory

The anemic hospitalizations were more in terms of total (0.94 vs 0.55/person-year). All-cause mortality (12.5% vs 7.1%) had a numerical advantage with anemia but the study lacked sufficient power to carry out mortality-specific conclusion. Both groups showed a slight functional capacity improvement as a result of therapy optimization and the anemic group showed smaller mean changes in 6MWT distance, which is in line with the anemia related restrictions in oxygen delivery and skeletal muscle energetics.

Tables

Table 1. Baseline characteristics by anemia status

Variable	Overall (n=312)	Anemia (n=128)	No anemia (n=184)	p-value
Age, years (mean±SD)	61.8±12.7	64.2±12.3	60.1±12.7	0.006
Female sex, n (%)	108 (34.6)	54 (42.2)	54 (29.3)	0.02
HFrEF (LVEF ≤40%), n (%)	176 (56.4)	74 (57.8)	102 (55.4)	0.68
HFmrEF (41–49%), n (%)	58 (18.6)	22 (17.2)	36 (19.6)	0.58
HFpEF (≥50%), n (%)	78 (25.0)	32 (25.0)	46 (25.0)	1.00
Diabetes, n (%)	142 (45.5)	64 (50.0)	78 (42.4)	0.18
CKD (eGFR <60), n (%)	124 (39.7)	70 (54.7)	54 (29.3)	<0.001
Loop diuretic use, n (%)	284 (91.0)	122 (95.3)	162 (88.0)	0.03
ACEi/ARB/ARNI, n (%)	256 (82.1)	100 (78.1)	156 (84.8)	0.13
Beta-blocker, n (%)	268 (85.9)	108 (84.4)	160 (87.0)	0.52
MRA, n (%)	214 (68.6)	84 (65.6)	130 (70.7)	0.34

Approximately 41% of the cohort had anemia and women were found to be at higher risk of anemia due to the older age, as well as the chronic kidney disease prevalence, which was largely higher in women. Significantly, anemia was spread among LVEF categories instead of being concentrated on HFrEF, which suggests that lower hemoglobin is a systemic factor adjusting HF vulnerability and not a HF-specific epiphenomenon. Background GDMT use The overall general similarity on the background of GDMT use implied the possibility of minor outcome variation to be attributed to the major gaps in therapy alone.

Table 2. Biomarkers and functional status at baseline

Measure	Anemia (n=128)	No anemia (n=184)	p-value
Hemoglobin, g/dL (mean±SD)	10.8±1.1	13.7±1.2	<0.001
MCV, fL (mean±SD)	84.6±8.9	86.1±7.8	0.12
eGFR, mL/min/1.73m² (median, IQR)	52 (38–66)	72 (58–86)	<0.001
NT-proBNP, pg/mL (median, IQR)	2260 (1180–4120)	1480 (760–2760)	<0.001
6MWT distance, meters (mean±SD)	242±88	298±92	<0.001
NYHA III–IV, n (%)	78 (60.9)	76 (41.3)	0.001

Natriuretic co-segregated with cholesterol with best functioning limitation and higher burden on anemia. The presence of a higher level of NT-proBNP and a lower level of 6MWT distance seems to reinforce a clinically consistent pattern where anemia is correlated with an increase in heart filling pressures occurrences and muscle capacity deficiency. Reduced eGFR of the anemic group indicates a significant cardio-renal involvement, which is equivalent to multifactorial anemia in HF (hemodilution, inflammation, renal erythropoietin deficiency, and iron restriction).

Table 3. Twelve-month outcomes by anemia status

Outcome	Anemia (n=128)	No anemia (n=184)	Effect estimate
Primary composite progression, n (%)	75 (58.6)	64 (34.8)	RR 1.68
HF hospitalization (≥1), n (%)	52 (40.6)	46 (25.0)	RR 1.62
≥30% rise in NT-proBNP, n (%)	44 (34.4)	38 (20.7)	RR 1.66
NYHA worsening ≥1 class, n (%)	36 (28.1)	32 (17.4)	RR 1.62
All-cause mortality, n (%)	16 (12.5)	13 (7.1)	RR 1.76
Total HF hospitalizations (events/person-year)	0.94	0.55	IRR 1.71

Anemia was a relatively powerful predictor of increased HF development at 12 months, which was manifested through hospitalization, biomarker deterioration, and symptomatic deterioration. The uniformity of effect demonstrated when using various components of progression is against the explanation of a single mechanism and rather sensitises the view of anemia being a multi-pathway amplifier of HF instability. Even though the number of deaths with anemia outliers was greater, the prevailing signal within this cohort was repeated decompensation and biomarker impairment, each associated endpoint with direct surveillance severity and selected comorbidity management objectives.

Table 4. Multivariable Cox regression for HF progression

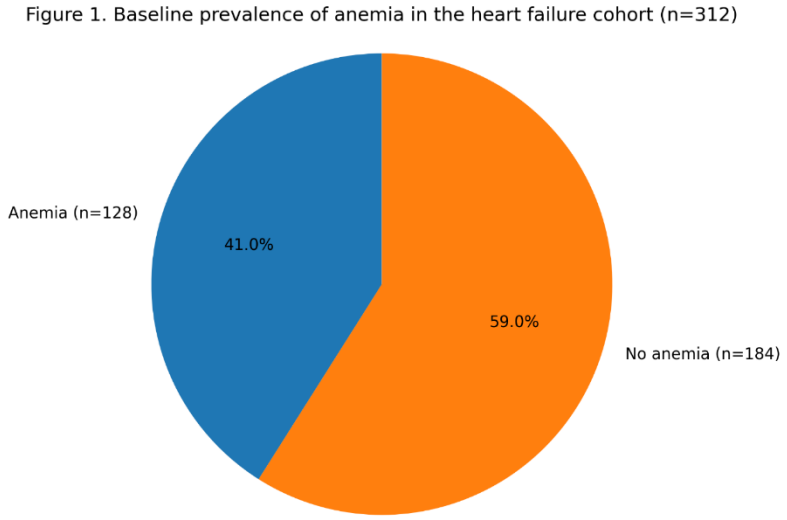
Predictor	Adjusted HR	95% CI	p-value
Anemia (yes vs no)	**1.72**	**1.28–2.32**	**<0.001**
Age (per 10 years)	1.14	1.02–1.29	0.02
Female sex	1.08	0.81–1.45	0.60
eGFR <60	1.39	1.04–1.86	0.03
Baseline NT-proBNP (log scale)	1.31	1.12–1.53	0.001

Diabetes	1.21	0.91–1.62	0.19
HFrEF (vs HFpEF/HFmrEF)	1.16	0.86–1.57	0.33
Lower GDMT intensity (per 1-class decrease)	1.18	1.01–1.38	0.04

Even when the effect of major confounders was corrected, anemia retained its independent relationship with HF progression implying that its risk factor is not entirely accounted by age, renal dysfunction, or baseline natriuretic peptide severity. The continued association of anemia and NT-proBNP point to a possibly additive prognostic effect: anemia can placing an excessive workload on the myocardium and decreasing the amount of oxygen delivered to the body and natriuretic peptides bind up filling pressure and wall strains. This clinically justifies anemia as an affordable low-cost stratifying criterion of increased follow-up.

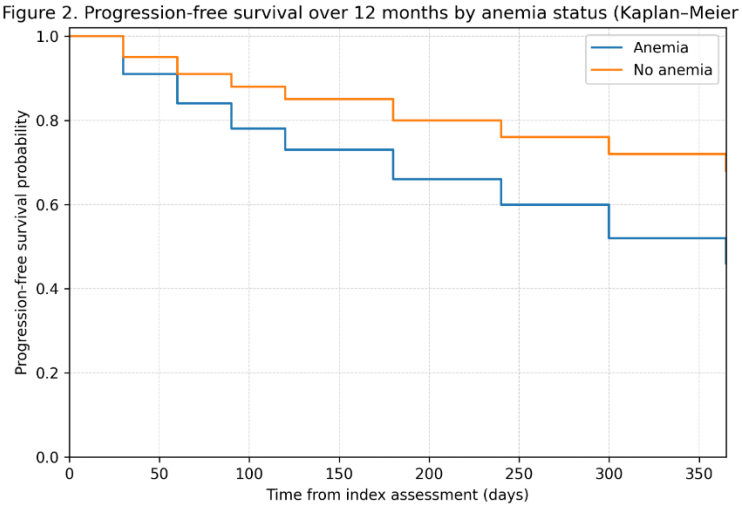
Figures

FIGURE 1.BASELINE PREVALENCE OF ANEMIA IN THE HEART FAILURE COHORT (N=312)



This number shows that 41.0% of patients with heart failure had the comorbidity of anemia at baseline and that the comorbidity burden is high in the routine tertiary-care practice. The large anemic subgroup justifies anemia as a clinically significant alterer of heart failure course and not an infrequent adjumptic. Since anemia has so many comorbidities similarly with renal dysfunction and iron-denuded states, this distribution justifies the use of structural hematologic and iron- algun profiling in the index analysis, as well as in follow-up.

FIGURE 2. KAPLAN–MEIER CURVE FOR PROGRESSION-FREE SURVIVAL BY ANEMIA STATUS



The premature dissociation of progression-free survival curves indicates that anemia defines a at-risk period of time when recurrence of congestion, worsening of biomarkers, or functional decline occurs at a rapid rate. Such timing is in line with the instability following discharge and incomplete physiologic recovery after decompensation. Considering the care-delivery prism, anemia can be a stimulus to premature follow-up, titration to diuretic and GDMT, and methodical screening of the iron-deficiency, renal contributors, and occult bleeding-associated factors.

DISCUSSION

Accelerating HF had been associated with anemia in this observational cohort, and anemia was independently associated with progression between 12 months, which was defined as a composite of HF hospitalization, natriuretic peptide worsening, and NYHA deterioration. When adjusted by renal functional status, initial severity of natriuretic peptide, LVEF phenotype, and Gottlieb-Muller-Trenz "-tendenz-Messenger-improved information, the association remained significant and suggested that anemia provided prognostic data that were not encompassed by the conventional clinical risk factors. These results are in line with extensive HF program data, indicating that anemia is endemic by ejection fraction category and is correlated with increased risks of undesirable outcomes [1]. Likewise, systematic reviews and meta-analyses have confirmed numerous times that anemia is associated with high mortality and hospitalization in HF, which is why it is a clinically significant state of risk, not a mere coincidental laboratory abnormality [3,8,13].

One of the main contributions of the current work is that it operationalizes the idea of progression beyond mortality per se. In most cohorts of HF, death is a crucial endpoint though recurring decompensation, rising congestion, aggravated biomarkers, and functional impairment are characteristic phases of real-world practice. Incorporating natriuretic peptide worsening and NYHA deterioration and hospitalization, our outcome synthesizes the clinical hardship of deterioration underlying repeated healthcare use and poor quality of life. This method goes in line with the substantially larger body of evidence that anemia is strongly associated with symptomatic burden and functional limitation in HF [1,2].

Mechanistic and clinical literature adhere to the existence of biological feasibility of anemia to facilitate progression. Decreasing hemoglobin may compromise the delivery of oxygen to the whole body, adding myocardial loads and sympathetic efforts; HF results in increased ventricular loads, congestion and skeletal muscle impairment due to compensatory metabolic responses [2]. Also, spreadsheet centrality anemia in the context of HF is often clustered, and it intersects with nephron impairment, inflammation, and iron deficiency, which are likely to increase neurohormonal stimulation and decreased physiologic capacity [2,3]. This multi-pathway picture can suggest why our cohort anemia was linked with progression across a number of domains than one endpoint constituent.

Our results are also compatible with the growing body of literature regarding the therapeutic treatment of iron deficiency in HF. Randomized evidences have been established that intravenous ferric carboxymaltose can improve symptoms, exercise, and health-related quality of life in iron-deficient HF, even in patients who do not have apparent anemia [4]. The extended follow-up analysis depicted consistent functional remuneration and indicators of lowered HF dental hospitalization [5]. Iron-restricted conditions The environment of acute HF intravenous iron has been linked to fewer recurrent HF hospitalization in iron-deficient patients with reduced or mildly reduced ejection fraction that supports the notion of targeting the iron-restricted conditions to adjust the post-discharge susceptibility [6]. Further research involving ferric derisomaltose scaled evidence of reduced events in iron-deficient HFrEF populations, but the interpretation can be different according to the mode of conduction by trial and endpoint definition [7]. All these trials suggest that iron-restricted phenotypes, which regularly regurge anemia, are clinically modifiable and may be a lever to mitigate outcomes in the chosen patients [4, 7, 16]. Nevertheless, anemia cannot be reduced to iron deficiency and supplementation without phenotyping should not be taken as a fact. Iron biomarker analyses indicate that iron deficiency and hemodialective viability are better displayed by transferrin saturation and associated indices, but functional iron deficiency and hemodialective visibility is best identified by differentiated assessment instead of reflex therapy, in special scenarios [12]. The clinical implication is practical: work-up (iron indices, renal functions, inflammatory contributors, medication effects, and a test of occult blood loss) and close follow-up is always needed when anemia accompanies HF, and it is needed most often within the from the beginning of post-discharge period since the risk of progression is the biggest in this period [1, 2, and 6].

Limitations

This non-experimental design is a single center observational design, which limits causal inference and is prone to residual confounding. Mechanistic subtyping was limited as not all studies on iron were readily available. The composite endpoint also incorporates biological variability and assay time as this can change biomarker. Lastly, due to the sample size, conclusions based on mortality were not precise and they ought to be verified in larger multicenter cohorts [3,8,13].

Implications

Hemoglobin is cheap and is readily available and our results endorse the inclusion of anemia on the progression-risk stratification with more intense monitoring and systematic phenotyping of reversible factors, including iron deficiency, an approach in keeping with the overall HF iron trial findings [47,16].

CONCLUSION

Among members of this cohort, anemia was common and directly correlated with faster progression of heart failure during 12 months, in both clinical, natriuretic peptides, and repeated hospitalizations. The connection has been maintained even with the consideration of renal dysfunction after baseline severity of biomarkers and LVEF phenotype and intensity of treatment and anemia has been highlighted as more than passive sign of comorbidity burden. Some potential clinical uses of hemoglobin include it acting as a low-cost risk indicator to initiate the orderly diagnostic work-up (iron status, renal contributors, and sources of bleeding) and enhanced post-discharge monitoring. Such findings are in addition to evidence on trial that specifies selected iron-deficient HF groups of recipients of targeted iron repletion.

REFERENCES

1. Solomon, S. D., Anand, I., Belch, J., et al. (2006). Clinical correlates and consequences of anemia in a broad spectrum of patients with heart failure (CHARM). Circulation.
2. Tang, Y. D., & Katz, S. D. (2008). Anemia and chronic heart failure. Journal of the American College of Cardiology.
3. Groenveld, H. F., Januzzi, J. L., Damman, K., et al. (2008). Anemia and mortality in heart failure: A systematic review and meta-analysis. Journal of the American College of Cardiology.
4. Anker, S. D., Comin Colet, J., Filippatos, G., et al. (2009). Ferric carboxymaltose in patients with heart failure and iron deficiency (FAIR-HF). New England Journal of Medicine.
5. Ponikowski, P., van Veldhuisen, D. J., Comin-Colet, J., et al. (2015). Beneficial effects of long-term intravenous iron therapy with ferric carboxymaltose in HF (CONFIRM-HF). European Heart Journal.
6. Ponikowski, P., Kirwan, B.-A., Anker, S. D., et al. (2020). Ferric carboxymaltose for iron deficiency at discharge after acute heart failure (AFFIRM-AHF). The Lancet.
7. Kalra, P. R., Cleland, J. G. F., Petrie, M. C., et al. (2022). Intravenous ferric derisomaltose in patients with heart failure and iron deficiency (IRONMAN). The Lancet.
8. Saeed, O., et al. (2021). The prognostic significance of anemia in patients with heart failure: Systematic review and meta-analysis. Frontiers in Cardiovascular Medicine.
9. World Health Organization. (2001/updated guidance). Hemoglobin thresholds for anemia (WHO definitions).
10. Ponikowski, P., et al. (2009). Rationale and design of FAIR-HF. European Journal of Heart Failure.
11. Vaduganathan, M., et al. (2019). Anemia and iron deficiency in heart failure. Heart Failure Clinics.
12. (2024). Intravenous iron for heart failure, iron deficiency definitions, and biomarkers (IRONMAN marker analyses). European Heart Journal.
13. (2016). Prognosis of comorbid heart failure and anemia: Systematic review and meta-analysis. International Journal of Cardiology.
14. (2024). HEART-FID trial overview and baseline characteristics. ClinicalTrials.gov record NCT03037931.
15. (2025). Intravenous ferric carboxymaltose in heart failure with iron deficiency (FAIR-HF2). JAMA.
16. (2026). Meta-analysis of intravenous ferric carboxymaltose in HF with iron deficiency. ESC Heart Failure.
17. (2024). Impact of anemia on outcomes in acute heart failure: Meta-analysis. Europe PMC .

