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Original Research

Evaluating the burden of iron deficiency in heart failure with reduced ejection fraction: a retrospective analysis using the NIS database

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Abstract

Introduction: Iron deficiency (ID), prevalent in 10.6% of heart failure with reduced ejection fraction (HFrEF) patients, has been linked to poorer clinical outcomes. The existing literature have provided insight into long-term impacts of the association, but the in-hospital outcomes remain unexplored. This study utilizes data from the National Inpatient Sample (NIS) to bridge this gap.

Method: NIS database for the years 2020 to 2022 was used to identify adult patients (aged ≥18 years) hospitalized with a primary diagnosis of HFrEF. Patients were further stratified based on the presence or absence of ID. Propensity score matching in a 1:1 ratio was performed using STATA, adjusting for age, sex, and relevant comorbidities. Univariate logistic and linear regression analyses were conducted before and after matching to assess clinical outcomes.

Results: Among 6,738,392 patients hospitalized with HFrEF from 2020 to 2022, 398,230 had comorbid ID, predominantly females and Blacks. After 1:1 propensity score matching, 52,930 pairs were analyzed. ID patients had higher rates of acute kidney injury (36.5% vs. 30.3%, OR: 1.30, P<0.001), blood transfusion (64% vs. 36%, OR: 1.89, P<0.001), and longer hospital stay (7.95 vs. 6.92 days, P<0.001). Despite greater resource utilization, they had lower in-hospital mortality (22.1% vs. 27.9%, OR: 0.67, P<0.001), cardiogenic arrest (10.14% vs. 11.17%, OR: 0.91, P=0.03), need for inotropes (5.18% vs. 6.40%, OR: 0.81, P=0.03), ECMO use (0.56% vs. 1.31%, OR: 0.42, P<0.001), and balloon pump insertion (4.3% vs. 6.1%, OR: 0.69, P<0.001).

Conclusion: ID in HFrEF patients is associated with increased rates of acute kidney injury, blood transfusion, and prolonged hospital stay. Despite greater resource utilization, patients with ID demonstrated significantly lower in-hospital mortality and fewer critical interventions. These findings underscore the complex clinical profile of HFrEF patients with concomitant ID and highlight the need for continued research to guide optimal management and improve patient outcomes.

Keywords: Iron deficiency, Heart failure with reduced ejection fraction, In-hospital outcomes, Resource utilization, Mortality, Length of hospital stay

Abbreviations: ID: Iron Deficiency; HF: Heart Failure; HFrEF: Heart Failure with Reduced Ejection Fraction; NIS: National Inpatient Sample; TSAT: Transferrin Saturation; IRB: International Review Board; ICD: International Classification of Disease; ECMO: Extracorporeal Membrane Oxygenation; TOTCHG: Total Hospital Charges; LOS: Length of Stay; AKI: Acute Kidney Injury; CI: Confidence Interval; OR: Odds Ratio; MD: Mean Difference; DM- Diabetes Mellitus; HTN: Hypertension; HLD: Hyperlipidemia; CAD: Coronary Artery Disease; CVA: Cerebrovascular Accident; CKD: Chronic Kidney Disease

Introduction

Heart failure (HF) is a common and serious clinical syndrome resulting from myocardial dysfunction, currently affecting an estimated 6.7 million Americans, with prevalence projected to rise to 8.5 million by 2030 [1,2]. HF is linked to preventable complications and a significant economic

burden, underscoring the importance of studying its comorbidities. ID with or without anemia is a frequent comorbidity in patients with HFrEF, with a prevalence of approximately 55% in all heart failure patients [3]. In the context of heart failure, iron deficiency is diagnosed as ferritin level less than 100 ng/mL, or a ferritin level of 100–299 ng/mL along with a transferrin saturation (TSAT) less than 20% [4,5].

The development of ID in HF is initially driven by elevated hepcidin levels in the setting of systemic inflammation, which impairs iron release from enterocytes and the reticuloendothelial system [6]. With advancing heart failure, however, additional mechanisms contribute, including intestinal dysfunction secondary to mucosal edema, delayed gastric emptying, and impaired motility along with reduced dietary iron intake due to poor oral intake, collectively exacerbating the progression of ID [6–8].

In recent years, numerous studies have documented the impact of ID in patients with HFrEF. These studies demonstrated a higher mortality rate and lower quality of life in HFrEF patients with ID versus HFrEF patients without ID [9,10]. Other studies highlighted that in patients with HFrEF, ID was independently linked to lower peak oxygen uptake (VO_2) and poorer ventilatory efficiency (higher VE/VCO_2 slope), reflecting diminished exercise capacity [11,12].

While prior studies have documented the effects of ID in patients with HFrEF, there is a need for a larger retrospective analysis to comprehensively assess its impact on in-hospital mortality, resource utilization, and complications such as cardiogenic shock. Although one prior study evaluated this association using the NIS, the analysis was limited to a single year of data and limited outcomes, restricting the ability to capture broader national trends and outcomes. This study therefore seeks to address these gaps by evaluating the clinical outcomes, including morbidity and mortality, associated with the coexistence of ID and HFrEF using a nationally representative dataset. We hypothesized that the presence of iron deficiency in patients hospitalized with HFrEF would be associated with higher in-hospital mortality, greater healthcare resource utilization, and an increased risk of complications, including cardiogenic shock. These findings can help clinicians identify HFrEF patients with iron deficiency who may need closer monitoring for complications while guiding treatment decisions to optimize outcomes.

Methods

Data source

This retrospective study utilized data from the National Inpatient Sample (NIS) spanning January 2020 to December 2022. The NIS, part of the Healthcare Cost and Utilization Project (HCUP) and administered by the Agency for Healthcare Research and Quality (AHRQ), is the largest publicly accessible inpatient database in the U.S. It captures discharge data from a representative sample of hospitals and covers approximately 97% of the U.S. population. As all data are anonymized, this study was exempt from institutional review board (IRB) approval under HCUP's data use agreement.

Study population

We included adult patients (≥18 years) hospitalized with a primary diagnosis of HFrEF, identified using ICD-10 codes ranging from I5020 to I5043. The cohort was then stratified based on the presence of ID with or with coexistent anemia, which was defined using ICD-10 codes D50, D508, and D509. Patients with missing demographic or clinical variables were excluded from the analysis.

Covariates and comorbidities

We assessed patient demographics (age, sex, race/ethnicity, income level, insurance status, and hospital characteristics such as region and bed size). Comorbid conditions were determined using ICD-10 coding algorithms and included hypertension, diabetes, hyperlipidemia, coronary artery disease, chronic kidney disease, and cerebrovascular disease. These factors were included in the propensity score model to account for potential confounding.

Outcomes

The primary outcome of interest was in-hospital mortality. Secondary endpoints included key inpatient complications such as cardiogenic shock, cardiac arrest, mechanical circulatory support (including intra-aortic balloon pump and ECMO), inotropic agent use, arrhythmias, acute kidney injury (AKI), and the need for blood transfusion. Health resource utilization was evaluated using hospital length of stay (LOS) and total hospitalization charges (TOTCHG), extracted through corresponding ICD-10 diagnosis/procedure codes and cost variables in the NIS.

Statistical analysis

To adjust for baseline differences between groups, we performed 1:1 propensity score matching using nearest-neighbor matching with a caliper width of 0.02, implemented in STATA. The matching process included demographic characteristics and key comorbidities. Adequate covariate balance post-matching was confirmed by non-significant p-values (>0.05) (**Figure 1**).

We used chi-square tests for categorical comparisons and independent t-tests for continuous variables. Logistic regression models were applied to estimate odds ratios (ORs) and 95% confidence intervals (CIs) for binary outcomes, while linear regression models were used for continuous outcomes such as LOS and TOTCHG. A p-value <0.05 was considered statistically significant. All analyses were conducted using STATA.

Results

From 2020 to 2022, we identified a total of 6,738,392 adult hospitalizations for HFrEF across the United States. Among these, 398,230 cases (approximately 5.9%) had a concurrent diagnosis of ID. In the unmatched cohort, both groups were generally comparable, although patients with ID were slightly younger than those without (68.9 vs 69.2 years; MD=−0.24 years; 95% CI: −0.37 to −0.11; p<0.001). A higher proportion of ID patients were female (43.14% vs 36.26%; OR=1.33; 95% CI: 1.31 to 1.35; p<0.001) and Blacks (25.53% vs 9.97%; OR=1.37; 95% CI: 1.35 to 1.39; p<0.001). On the contrary, a smaller proportion of ID patients were Whites (60.38% vs 66.08%; OR=0.79; 95% CI: 0.77 to 0.80; p<0.001). Geographically, ID patients were more often admitted in the Midwest (26.41% vs 22.89%; OR=1.21; 95% CI=1.19 to 1.23; P<0.001). Regarding comorbidities, iron-deficient patients had significantly higher rates of DM (72.6% vs 69.0%; OR=1.21; 95% CI: 1.20 to 1.23; p<0.001) and chronic kidney disease (33.7% vs 29.9%; OR=1.19; 95% CI: 1.17 to 1.21; p<0.001), while lower rates of CVA (0.38% vs 0.46%; OR=0.82; 95% CI=0.73 to 0.46%;P=0.001) (**Table 1**).

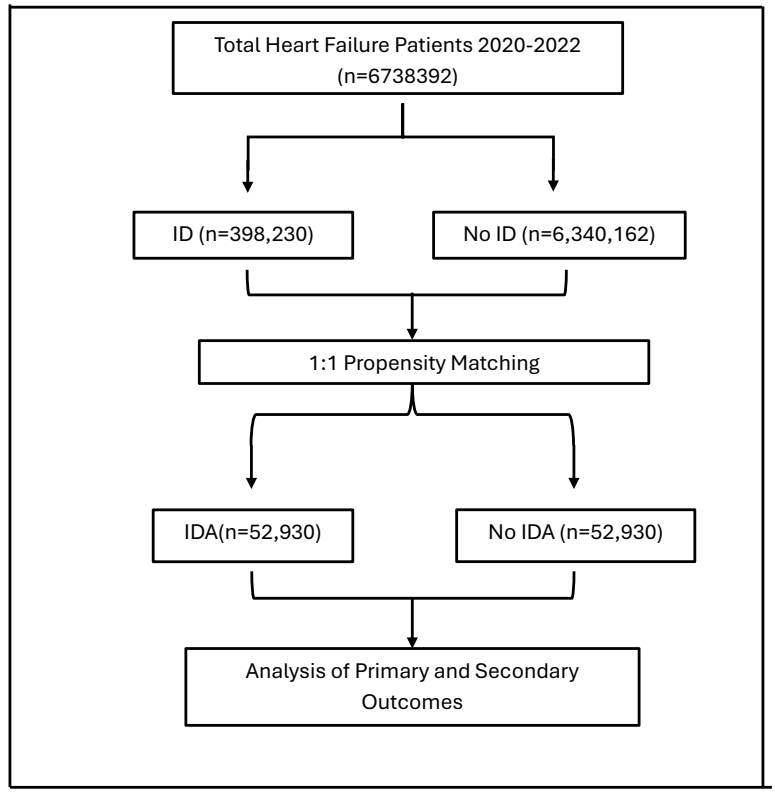

Figure 1. Schematics of study design.

Table 1. Baseline characteristics of included patients.

Variable		HFrEF and iron deficiency	HFrEF and no iron deficiency	Comparison
Mean AGE		68.94036 (68.77401 69.10671)	69.18053 (69.09145 69.26961)	-0.2401691 (-0.3665027 to -0.1138356, P<0.001)
FEMALE		171,779.9 (167954.7 175605.2)	2,299,274 (2252697 2345850)	1.333255 (1.312722 to 1.354109, P<0.001)
RACE	White	240,451 (60.38%)	4,189,579 (66.08%)	.787113 .7757213 .798672, P<0.001)
	Blacks	101,668 (25.53%)	1,266,130(19.97%)	1.368894 (1.346308 1.391859, P<0.001)
	Hispanics	35,243 (8.85%)	546,522 (8.62%)	1.027367(1.00151 1.053891, P=0.038)
	Asian	8,363(2.10%)	143,288 (2.26%)	.9257784 (.8802504 .9736612, P=0.003)
	Americans	2,867(0.72%)	42,479(0.67%)	1.060674 (-.9732113 1.155997, P=0.180
	Others	9,677 (2.43%)	151,530(2.39%)	1.01559 (-0.000898 to .9689181 1.06451, P=0.519)
HOSPITAL REGION	Northeast	70,566 (17.72%)	1,085,436 (17.12%)	1.042908 (1.023529 1.062654, P<0.001)
	Midwest	105,172.49 (26.41%)	1,451,263(22.89%)	1.20926 (1.18974 1.229101, P<0.001)
	South	156,305 (39.25%)	2,636,239 (41.58%)	0.907964 (0.8947674 0.9213552, P<0.001)
	West	66,186 (16.62%)	1,167,858 (18.42%)	0.8825383 0.8657766 0.8996245, P<0.001)

Variable		HFrEF and iron deficiency	HFrEF and no iron deficiency	Comparison
BEDSIZE	Small	79,248 (19.9%)	1,331,434 (21.0%)	0.9341446 .917574 .9510144, P<0.001)
	Medium	105,690 (26.54%)	1,748,885 (27.60%)	.9478333 (0.9326133 0.9633016, P<0.001)
	Large	213,292 (53.56%)	3,258,209 (51.39%)	1.090739 (1.075209 1.106494, P<0.001)
PRIMAY PAYER	Medicare	269,840.5(67.76%)	4,264,393 (67.26%)	1.023636 (1.008091 1.039421, P=0.003)
	Medicaid	58,500 (14.69%)	809,005 (12.76%)	1.17719 (1.153525 1.20134, P<0.001)
	HMO	48,584 (12.2%)	885,721(13.97%)	0.8558154 (-0.8373456 0.8746926, P<0.001)
	Self-pay	10,911.5(2.74%)	187,035 (2.95%)	0.9250348 (0.8853927 .966451, P<0.001
	No charge	717 (0.18%)	12,680 (0.20%)	0.9054961 (0.7658634 1.070587, P=0.2450)
	Other	9,717 (2.42)	180,695 (2.85%)	0.8507247 (0.8122668 0.8910035, P<0.001
MEDIAN HOSEHOLD INCOME	$1-$51999	141,929 (35.64%)	2,127,124 (33.55%)	1.094025 (1.077709 1.110587, P<0.001)
	$52000-$65999	104,814 (26.32%)	1,694,091 (26.72%)	.9784975 (-.9626237 .994633, P=0.009)
	$66000-$87999	87,411 (21.95%)	1,432,877 (22.60%)	.9623752 (-.9457695 .9792725, P<0.001)
	$88000 or more	64,115 (16.10%)	1,086,070 (17.13%)	.927917 (-.9099034 .9462872, P<0.001)
COMORBIDITIES	DM	289,349.9 (282,862 295,837.7)	4,411,393 (4,319,852 4,502,934)	1.2133 (1.196037 1.230812, P<0.001)
	HTN	133,799.9 (130367.7 137,232.2)	2,065,594 (2,020,983 2,110,205)	1.047111 (1.031352 1.06311, P<0.001)
	HLD	217,524.9 (215515.8 219,534)	3,340,858 (3,308,883 3,372,833)	1.08069 (1.065264 1.09634, P<0.001)
	CAD	228,964.9 (223617.1 234,312.6)	3,616,724 (3,540,585 3,692,862)	1.018601 (1.003956 1.033459, P=0.013)
	CVA	1,515 (1,337.214 to 1,692.786)	29,454.98 (28,402.59 to 30,507.37)	0.8181887 (0.7288123 0.9185256, P=0.001)
	CKD	134,229.9 (130789.4 137,670.4)	1,899,894 (1,857,726 1,942,062)	1.188298 (1.170402 1.206468, P<0.001)

In the unmatched analysis, patients with ID had significantly lower in-hospital mortality compared to non-ID patients (4.44% vs6.45%; OR = 0.67; 95% CI: 0.65 to 0.70; p<0.001). They also experienced lower rates of cardiac arrest (1.93% vs 2.68%; OR=0.71; 95% CI: 0.68 to 0.75; p<0.001), reduced need for inotropes (2.97% vs 3.14%; OR=0.92; 95% CI: 0.87 to 0.98; p=0.010), balloon pump insertion (0.80% vs 1.0%; OR = 0.80; 95% CI: 0.73 to 0.88; p<0.001), and ECMO use (0.10% vs 0.17%; OR=0.60; 95% CI: 0.46 to 0.75; p<0.001). However, ID was associated with significantly higher risk of acute kidney injury (38.45% vs 31.12%; OR=1.38; 95% CI: 1.36 to 1.40; p<0.001), increased need for blood transfusion (11.47% vs 5.64%; OR=2.17; 95% CI: 2.11 to 2.23; p<0.001), and greater hospital length of stay (7.95 vs 6.92 days; MD = 1.03 days; 95% CI: 0.95 to 1.11; p<0.001). Risk of cardiogenic shock (5.03% vs 5.12%; OR=0.98; 95% CI=0.94 to 1.02; P=0.399), risk of arrhythmia (16.85% vs 17.30%; OR=0.97; 95% CI=0.95 to 0.99; P=0.05) and total hospital charges were comparable between the two groups ($103847.4 vs $103411.7; mean difference =$435.52; 95% CI: –$1,364 to $2,235; p=0.635) (**Table 2**).

After performing a 1:1 propensity score matching (n=52,930 per group), the comparative associations remained consistent. Iron-deficient patients had persistently lower odds of in-hospital mortality (22.0% vs 33.6%; OR=0.64; 95% CI: 0.60 to 0.68; p<0.001), cardiac arrest (10.14% vs 11.16%; OR=0.91; 95% CI: 0.87 to 1.05; p=0.03), inotrope use (5.19% vs 6.40%; OR=0.81; 95% CI: 0.66 to 0.99; p=0.038), balloon pump use (4.26% vs 6.11%; OR=0.69; 95% CI: 0.59 to 0.82; p<0.001), and ECMO (0.50% vs 1.31%; OR=0.42; 95% CI: 0.30 to 0.61; p<0.001). However, they remained at significantly increased risk for AKI

Table 2. Comparison of primary and secondary outcomes before propensity score matching.

OUTCOME	HFrEF with iron deficiency (N=398,230)	HFrEF without iron deficiency (N=6,340,162)	Mean Difference/ Odds Ratio	P-value
Mortality	17,680 (4.44%)	408,920 (6.45%)	0.6738437 (95% CI 0.6497163 to 0.698867)	P<0.001
LOS	7.954196	6.923728	1.030468 (95% CI 0.9501994 to 1.110737)	P<0.001
TOTCHG	103,847.2	103,411.7	435.5207 (95% CI -1364.012 to 2235.053)	P=0.635
Cardiogenic Shock	20,045 (5.03%)	324,720 (5.12%)	0.9818848 (95% CI 0.9410238 to 1.02452)	P=0.399
Cardiac arrest	7,695 (1.93%)	170,065 (2.68%)	0.7148681 (95% CI 0.6790315 to 0.752596)	P<0.001
Arrythmia	67,115 (16.85%)	1,096,575 (17.30%)	0.9692397 (95% CI 0.9484588 to 0.9904758)	P=0.05
Inotropic need	11,715 (2.94%)	200,800 (3.17%)	0.926695 (95% CI 0.8747244 to 0.9817533)	P=0.010
ECMO	415 (0.10%)	11,020 (0.17%)	0.5991432 (95% CI 0.4756374 to 0.754719)	P<0.001
Balloon pump	3,195 (0.80%)	63,525 (1.00%)	0.7991308 (95% CI 0.7280133 to 0.8771955)	P<0.001
AKI	153,130 (38.45%)	1,973,404 (31.12%)	1.382484 (95% CI 1.360694 to 1.404622)	P<0.001
Blood transfusion	45,685 (11.47%)	357,300 (5.64%)	2.169878 (95% CI 2.111048 to 2.230347)	P<0.001

(19.99% vs 16.83%; OR=1.31; 95% CI: 1.27 to 1.35; p<0.001), arrhythmias (82.47% vs 91.29%; OR=0.88; 95% CI: 0.84 to 0.93; p<0.001), and blood transfusion (63.91% vs 35.92%; OR=1.89; 95% CI: 1.71 to 2.09; p<0.001). Length of stay was significantly prolonged in the ID group (7.75 vs 7.21 days; MD=0.53; 95% CI: 0.34 to 0.72; p<0.001), while total hospital charges remained similar ($103021.5 vs $101962.2; MD=$1,052.31; 95% CI: –$6,712.68 to $8,817.30; p=0.791). Similarly, the risk of cardiogenic shock was comparable between the two groups (25.74% vs 26.87%; OR=0.96; 95% CI=0.83 to 0.99; P=0.332) (**Table 3**).

Discussion

Summarization of results

In this large retrospective analysis of adults hospitalized with HFrEF using the NIS database (2020–2022), patients with ID demonstrated a paradoxically lower rate of in-hospital mortality and a slightly shorter length of stay compared with those without ID, despite similar hospitalization costs. Although the incidence of cardiac arrest was marginally lower in the ID cohort, these patients exhibited higher rates of arrhythmias and acute kidney injury (AKI). Furthermore, the use of advanced heart failure therapies, including inotropes, intra-aortic balloon pump (IABP), and extracorporeal membrane oxygenation (ECMO), was significantly lower among patients with ID. Collectively, these findings suggest that while ID may predispose to greater renal and arrhythmic complications, it is paradoxically associated with lower in-hospital mortality, shorter hospitalizations, and reduced reliance on advanced circulatory support interventions.

Explanation behind significant outcomes

The lower in-hospital mortality and shorter hospital stay among ID patients likely reflect differences in clinical management rather than a direct protective physiological effect. Patients with known ID often receive enhanced hemodynamic surveillance, timely blood transfusions, and intravenous iron supplementation, which collectively improve oxygen delivery and myocardial efficiency. These interventions facilitate earlier stabilization, reduce decompensations,

Table 3. Comparison of primary and secondary outcomes after propensity score matching.

OUTCOME	HFrEF with iron deficiency (N=52,930)	HFrEF without iron deficency (N=52,930)	Mean Difference/ Odds Ratio	P-value
Mortality	11,665 (22.04%)	17,815 (33.66%)	0.638773 (95% CI 0.6005196 to 0.6794631)	P<0.001
LOS	7.746983	7.214071	0.5329125 (95% CI 0.3447565 to 0.7210684)	P<0.001
TOTCHG	103,021.5	101,969.2	1,052.313 (95% CI -6,712.675 to 8,817.302)	P=0.791
Cardiogenic Shock	13,625 (25.74%)	14,220 (26.87%)	0.9558864 (95% CI 0.8284564 to 0.9905287)	P=0.332
Cardiac arrest	5,365 (10.14%)	5,910 (11.16%)	0.9058752 (95% CI 0.8726243 to 1.047093)	P=0.030
Arrythmia	43,650 (82.47%)	48,320 (91.29%)	0 .8842637 (95% CI 0.8390446 to 0.9319198)	P<0.001
Inotropic need	2,745 (5.19%)	3,390 (6.40%)	0.80774 (95% CI 0.6603389 to 0.9880441)	P=0.038
ECMO	295 (0.50%)	695 (1.31%)	0.4238181 (95% CI 0.2956036 to 0.607644)	P<0.001
Balloon pump	2,255 (4.26%)	3,235 (6.11%)	0.6944601 (95% CI 0.5871499 to 0.8213827)	P<0.001
AKI	10,555 (19.99%)	8,906 (16.83%)	1.308095 (95% CI 1.267086 to 1.350432)	P<0.001
Blood transfusion	33,825 (63.91%)	19,015 (35.92%)	1.892992 (95% CI 1.71218 to 2.092893)	P<0.001

and may account for the observed shorter hospitalizations and lower mortality. The reduced use of inotropes, IABP, and ECMO among ID patients may similarly represent a lower rate of worsening due to more careful observation and management. Moreover, clinicians may also adapt less aggressive management strategies, reflecting caution to avoid therapies that increase myocardial oxygen demand or bleeding risk in patients with reduced oxygen-carrying capacity [13].

Conversely, the higher incidence of AKI among ID patients may be attributed to compromised renal oxygenation and perfusion. ID reduces hemoglobin therefore decreasing arterial oxygen content, limiting oxygen delivery to the renal cortex and predisposing nephrons, especially within the outer medulla, to ischemic injury [14,15]. As medulla already works at maximum oxygen extraction capacity, even a small drop in oxygen leads to medullary hypoxia and ischemic stress. In the setting of HFrEF, these effects are compounded by elevated venous congestion, diuretic exposure, and activation of the renin–angiotensin–aldosterone system, collectively heightening renal vulnerability [16]. Similarly, chronic myocardial hypoxia in ID induced anemia can disrupt electrophysiologic stability, prolonging action potential duration and promoting atrial and ventricular arrhythmias [17].

Evidence from existing literature

The results of this study align with and extend prior research exploring ID's impact on heart failure outcomes. Bess *et al*. (2024) reported reduced adjusted mortality, and shorter hospital stays among iron deficient HFrEF patients, attributing these trends to intensified monitoring and targeted correction of ID [18]. Goldenberg *et al*. (2017) observed increased ventricular arrhythmia risk in anemic patients with implantable cardioverter-defibrillators, supporting the hypothesis that impaired oxygen delivery promotes electrical instability [19]. In contrast, our finding of a slightly lower cardiac arrest rate diverges from Xu *et al*. (2019), who reported higher arrest rates in iron deficient patients with acute coronary syndrome and cardiogenic shock, underscoring that ID's prognostic influence may differ between chronic and acute ischemic contexts [20,21].

Regarding renal outcomes, Han *et al*. (2015) demonstrated that anemia independently increases AKI risk in critically ill patients, consistent with our findings that renal hypoxia and hemodynamic stress underlie the elevated AKI rates observed [22]. Finally, our observation of lower utilization of inotropes, IABP, and ECMO parallels prior evidence that clinicians often withhold or delay invasive support in patients with anemia due to concerns about limited oxygen transport, increased bleeding risk, and uncertain benefit [23]. Together, these findings highlight the complex interaction between ID, organ-specific complications, and treatment intensity in patients with HFrEF, emphasizing the need for balanced, individualized care strategies.

Limitations

This study has limitations typical of administrative database analyses. Reliance on ICD-10 coding introduces potential misclassification, and the lack of laboratory data prevents confirmation of true biochemical iron deficiency or assessment of anemia severity. Medication details, transfusion practices, intravenous iron use, and timing of interventions are unavailable, limiting causal interpretation. The NIS captures hospitalizations rather than individual patients, precluding longitudinal follow-up or evaluation of readmissions. Residual confounding from unmeasured clinical factors is possible, and findings may be influenced by practice variations during the COVID-19 era. These limitations warrant cautious interpretation.

Conclusion

In this national cohort of adults hospitalized with HFrEF, iron deficiency was associated with lower in-hospital mortality, shorter length of stay, and reduced use of advanced circulatory support, despite higher rates of arrhythmias and acute kidney injury. Although we initially hypothesized that iron deficiency would be associated with worse in-hospital outcomes, the observed patterns suggest that these findings may reflect differences in clinical recognition, monitoring, and management intensity rather than a protective physiological effect of iron deficiency. These results highlight the clinical complexity of iron deficiency in heart failure and underscore the need for standardized inpatient screening and management pathways. Prospective studies incorporating laboratory parameters and treatment data are needed to better clarify the underlying mechanisms and optimize patient care.

Conflict of Interest

The authors have no conflict of interest to disclose.

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None to declare.

Authorship Contribution Statement

Rohab Sohail: Conceptualization, Methodology, Validation, Formal analysis, Software, Writing-original draft and Writing-review and editing.

Ridda Khattak: Conceptualization, Project administration and Writing-original draft.

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Sahil Desai: Visualization.

Joseph Deutsch: Writing-review and editing and Supervision.

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