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Association between Hemoglobin Levels and Heart Failure following Acute Myocardial Infarction: A Retrospective Study

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Abstract

Background and Aims: Low hemoglobin (Hb) concentrations and anemia are frequently encountered among individuals with acute myocardial infarction (AMI) and have been tied to unfavorable prognoses. We aimed to evaluate the association between admission Hb levels and anemia status and the risk of in-hospital heart failure after acute myocardial infarction (post-MI HF) in patients with ST-elevation myocardial infarction (STEMI) undergoing emergency percutaneous coronary intervention (PCI).

Methods: A total of 506 STEMI patients who received emergency PCI were enrolled in this single-center, retrospective investigation. Post-MI HF was diagnosed in accordance with guideline-recommended clinical and objective criteria. The analytical strategy comprised multivariable logistic regression, restricted cubic spline (RCS) modeling, subgroup and interaction testing, and E-value sensitivity analysis.

Results: During hospitalization, 47 patients (9.3%) developed post-MI HF. In the fully adjusted model, each 1 g/L increase in Hb was associated with 2.1% lower odds of post-MI HF [odds ratio (OR) 0.979, 95% confidence interval (CI) 0.960–0.998, P = 0.029]. Anemia was present in 113 patients (22.3%); anemic patients had a higher incidence of post-MI HF (18.6% vs. 6.6%) and significantly higher odds of post-MI HF (adjusted OR 3.15, 95% CI 1.56–6.39, P = 0.001). RCS analysis showed an approximately linear inverse association between Hb and post-MI HF risk (P for overall = 0.041; P for nonlinearity = 0.236). Diabetes mellitus and smoking modified the association (P for interaction = 0.022 and 0.001, respectively). The E-value was 5.75 (lower limit 2.49).

Conclusion: Among STEMI patients managed with emergency PCI, a lower Hb concentration at admission and the presence of anemia were each independently linked to a heightened likelihood of post-MI HF. Admission Hb may therefore serve as a convenient marker for early risk stratification of post-MI HF.

Keywords: anemia; hemoglobin; acute myocardial infarction; heart failure; percutaneous coronary intervention

Introduction

Acute myocardial infarction (AMI) is one of the leading causes of death and disability worldwide among cardiovascular emergencies. With the widespread adoption of emergency percutaneous coronary intervention (PCI), optimized anticoagulation therapy, and secondary prevention, acute and long-term mortality rates among AMI patients have declined significantly. However, post-myocardial infarction heart failure (post-MI HF) remains a common complication and a major determinant of poor long-term prognosis in AMI patients[1, 2]. Recent large European cohort investigations indicate that newly occurring heart failure after AMI, along with recurrent coronary events, accounts for a considerable share of residual risk[2]. In the current era of early PCI, a proportion of patients with acute ST-segment elevation myocardial infarction (STEMI) still progress to heart failure with reduced or preserved ejection fraction during hospitalization and follow-up; the incidence and predictive factors of this condition have attracted widespread attention[3, 4]. Therefore, early identification of high-risk patients for heart failure in the acute phase of AMI and the implementation of targeted preventive measures hold significant clinical value.

The development of post-MI HF involves the interaction of multiple factors, including the extent and location of the infarction, the quality of reperfusion, myocardial stunning and remodeling, excessive neurohumoral activation, and inflammatory responses[3, 5]. Previous studies and guidelines have shown that factors such as age, sex, blood pressure, infarct location, diabetes, hypertension, dyslipidemia, smoking, and renal function are all associated with poor prognosis or the development of heart failure following AMI and have been incorporated into commonly used risk stratification tools such as GRACE and TIMI[5-8]. In addition to these traditional risk factors, hematological parameters—as objective indicators reflecting systemic oxygen supply and metabolic status—are increasingly recognized for their role in assessing the prognosis of AMI.

Hemoglobin (Hb) is the primary carrier responsible for the blood's oxygen-

carrying capacity, and anemia is relatively common in patients with AMI and heart failure[5, 6]. By activating the sympathetic nervous system and the renin-angiotensin system and by encouraging ventricular remodeling, anemia may diminish oxygen delivery to the myocardium, worsen injury in ischemic tissue, and elevate the likelihood of heart failure[5]. In recent years, multiple randomized trials and patient-level meta-analyses focusing on red blood cell transfusion thresholds in patients with AMI and anemia have further highlighted the significant association between Hb levels and clinical outcomes in AMI patients[9, 10]. Additionally, a decrease in hemoglobin levels during PCI in patients with STEMI has been reported to be associated with long-term adverse outcomes[11]. Furthermore, iron deficiency—a major cause of anemia—is also associated with the prognosis of patients with acute coronary syndrome[12]. Therefore, Hb levels and the presence of anemia may have potential predictive value for the development of heart failure following AMI.

However, previous studies have primarily focused on the overall impact of anemia on mortality or major adverse cardiovascular events in patients with AMI. Research examining the association between Hb levels and anemia status and the specific outcome of new-onset heart failure during hospitalization for AMI remains relatively limited, particularly among Chinese patients with STEMI undergoing emergency PCI. Some studies have differed in their definitions of heart failure outcomes, anemia cutoffs, and strategies for adjusting for covariates, resulting in conclusions that are not entirely consistent[10-12]. Against this backdrop, this study utilized a single-center, in-house database to systematically evaluate the relationship between admission Hb levels and anemia status in STEMI patients and the development of post-myocardial infarction heart failure during hospitalization.

Methods

Study Design and Population

This retrospective, cross-sectional investigation was carried out at the First People's Hospital of Changde, Hunan Province (a Grade III Level A institution). Electronic medical records of STEMI patients treated between May 2021 and May 2024 were

reviewed. Ethical clearance for the protocol was obtained from the Academic Ethics Committee of the Changde Hospital, Xiangya School of Medicine, Central South University (Approval No.: 2026-593-01). Because the design was retrospective and relied on routinely collected clinical data, the Ethics Committee exempted the study from obtaining informed consent. Before analysis, all data were anonymized/de-identified. The investigation complied with the Declaration of Helsinki.

Eligible participants were patients whose diagnostic criteria met the"2023 ESC Guidelines for the management of acute coronary syndromes"[1] and who were hospitalized in the Department of Cardiology at our hospital. Exclusion criteria included: (1) patients who did not undergo PCI within 12 hours of the onset of chest pain (n=328); (2) patients younger than 18 or older than 90 years of age (n=5); (3) patients with a definitive diagnosis of old MI (n=22); (4) patients with severe hematological disorders, major organ failure, or malignant tumors (n=17); (5) patients with missing Hb data (n=20). Following these exclusions, 506 patients were retained for analysis. **Figure 1** depicts the screening and exclusion workflow.

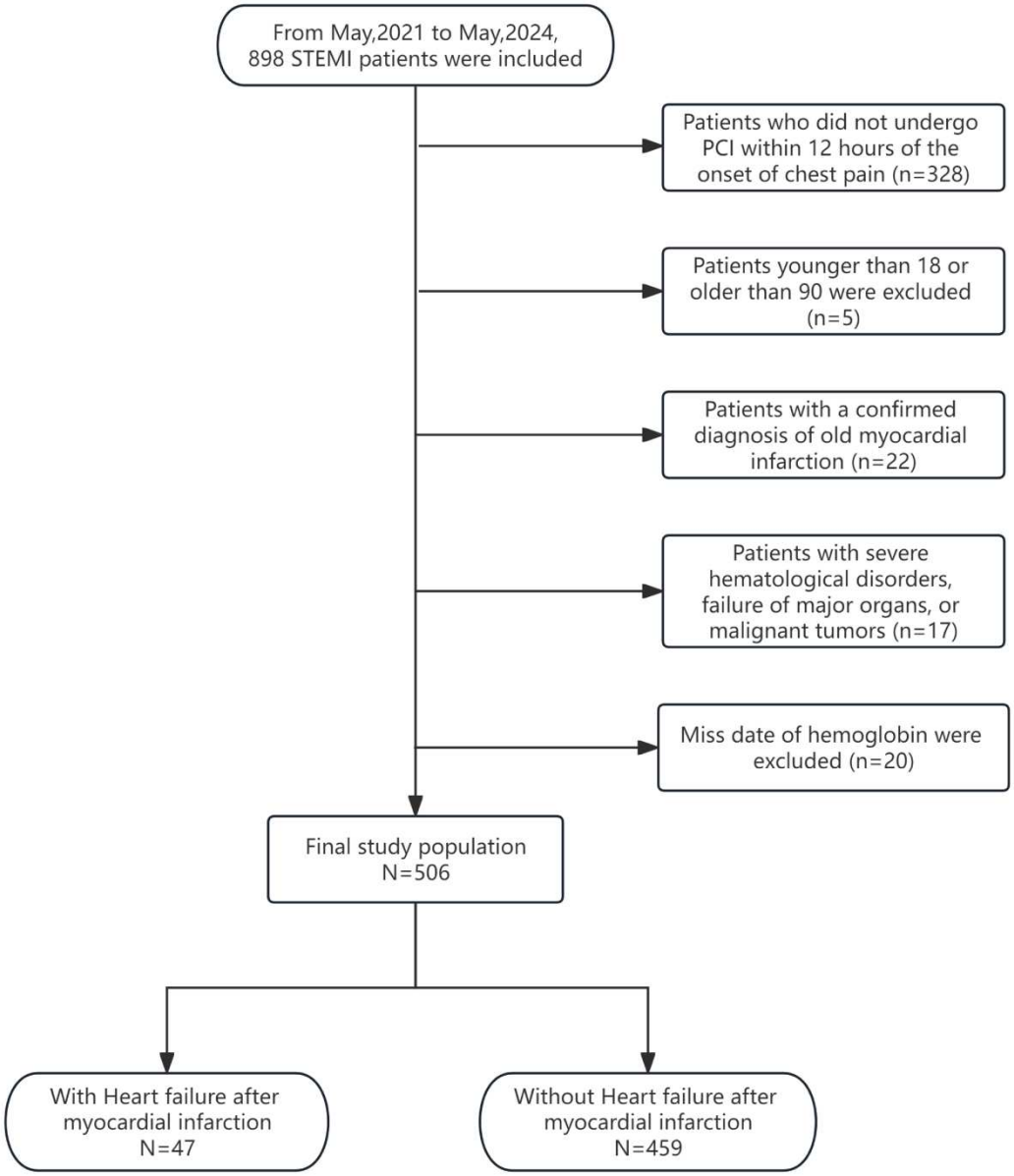


Figure 1. Flow diagram of participant screening and exclusion.

Outcome Definition

The primary outcome was in-hospital post-MI HF, defined as new-onset or worsened heart failure occurring during hospitalization after AMI [1]. The diagnosis required clinical evidence of heart failure, including Killip class II-IV, NYHA functional class II-IV, pulmonary congestion, acute pulmonary oedema, or physician-documented heart failure requiring treatment, together with objective evidence of

cardiac dysfunction or elevated filling pressure, including elevated NT-proBNP, reduced left ventricular ejection fraction, left ventricular dysfunction, or imaging evidence of pulmonary congestion[7, 13, 14]. Patients with Killip class III-IV, acute pulmonary oedema, or pump-failure-related cardiogenic shock were classified as having post-MI HF regardless of biomarker availability[15].

In our study, post-MI HF was defined as: Killip class ≥ II, or NYHA class ≥ II, or heart failure documented by the physician following a myocardial infarction; and supported by at least one of the following: elevated NT-proBNP (<50y ≥450 pg/mL, 50-75y ≥900 pg/mL, >75y ≥1800 pg/mL), EF < 50%, or left ventricular enlargement.

Definition of Exposure Factors

The primary exposure variable in this study was the Hb level obtained from routine blood tests performed upon hospital admission. Hb was initially included in the model as a continuous variable, and the effect size was interpreted as the change in the risk of post-MI HF for every 1 g/L increase in Hb. To facilitate clinical interpretation and ensure robustness, anemia was further defined using sex-specific thresholds: Hb < 110 g/L for women and Hb < 120 g/L for men[16]. The anemia variable was included in the regression model as a dichotomous exposure, with patients without anemia serving as the reference group.

Covariates

Based on previous literature[1, 8, 15, 17], cardiovascular guidelines, and clinical relevance, this study included the following potential confounding factors: demographic characteristics, including age and sex; medical history, including hypertension, diabetes, dyslipidemia, smoking, and alcohol consumption; laboratory parameters, including serum potassium, serum sodium, serum creatinine, serum uric acid, white blood cell count; and in-hospital cardiac events, including severe ventricular arrhythmias.

Missing Data

Across all analyses, fewer than 10% of covariate values were missing. Missing covariate data were handled by multiple imputation, after which logistic regression was carried out. Five imputed datasets were generated and analyzed.

Statistical Analysis

Depending on their distribution, continuous variables were reported as mean ± standard deviation or as median (interquartile range), whereas categorical variables were summarized as counts and percentages. Patients were divided according to whether post-MI HF occurred. Normally distributed continuous variables were contrasted with the independent samples t-test and skewed variables with the Mann-Whitney U test; categorical variables were compared by the Pearson χ^2 test or Fisher's exact test. Statistical significance was set at a two-sided $P < 0.05$.

Multivariable logistic regression was employed to assess how Hb concentration and anemia status related to post-MI HF. Adjustment proceeded in stages: the crude model contained no covariates; Model 1 controlled for age and sex; Model 2 added hypertension, diabetes, dyslipidemia, and smoking and alcohol history; Model 3 extended Model 2 with serum potassium, sodium, creatinine, uric acid, white blood cell count, and severe ventricular arrhythmias. For Hb entered continuously, the OR captured the shift in post-MI HF risk per 1 g/L increase; for the binary anemia term, non-anemic patients formed the reference.

A restricted cubic spline (RCS) model was fitted to determine whether the Hb-post-MI HF relationship followed a nonlinear dose-response pattern. Overall association and nonlinearity tests were applied to characterize this relationship. Stratified analyses accompanied by interaction tests were carried out to examine whether the association held across clinical subgroups, with age, sex, hypertension, diabetes, dyslipidemia, and smoking status specified in advance. Within each subgroup the Hb-post-MI HF association was estimated separately, and interaction was evaluated by adding product terms to the logistic models or by likelihood ratio tests. To gauge discrimination, receiver operating characteristic (ROC) curves were derived from the predicted probabilities of the multivariable logistic model, and the area under the curve (AUC) with its 95% confidence interval (CI) was computed. E-values were calculated for the anemia-post-MI HF association in the fully adjusted model to quantify how strongly an unmeasured confounder would need to act to account for the finding; a larger E-value

implies that a more potent unmeasured confounder would be necessary to explain away the observed association.

In addition, we performed a diagnostic assessment of multicollinearity among the covariates, using the variance inflation factor (VIF) to evaluate the risk of multicollinearity. The VIF values for all variables included in the model were below the commonly used warning thresholds, suggesting that there was no significant multicollinearity.

All analyses were performed using R Statistical Software (version 4.2.2) and the Free Statistics Analysis platform (version 2.5.1) [18, 19]. P-values below 0.05 on a two-tailed basis were considered to indicate statistical significance.

Results

Study Population and Baseline Characteristics

Based on the predefined inclusion and exclusion criteria, this study ultimately enrolled 506 patients with acute STEMI. A total of 47 patients developed post-MI HF during hospitalization, with an incidence rate of 9.3%; the remaining 459 patients did not develop post-MI HF (**Figure 1**).

The mean age of the enrolled patients was 62.8 ± 11.7 years; 399 were male (78.9%) and 107 were female (21.1%). Compared with patients who did not develop post-MI HF, those who developed post-MI HF were older (66.1 ± 9.9 years vs. 62.5 ± 11.8 years, P = 0.042). There were no statistically significant differences between the two groups in terms of sex distribution at admission (P > 0.05). Regarding medical history, there were no statistically significant differences between the two groups in terms of a history of hypertension, diabetes, smoking, or alcohol consumption; the prevalence of dyslipidemia was lower in the post-MI HF group, but the difference was only marginal (17.0% vs. 30.3%, P = 0.056)(**Table 1**).

Table 1. Baseline characteristics of patients stratified by heart failure after myocardial infarction

Characteristic	Total (N = 506)	Without post-MI HF (n = 459)	With post-MI HF (n = 47)	*P* value
Admission characteristics				
Age, years	62.8 ± 11.7	62.5 ± 11.8	66.1 ± 9.9	0.042
Sex, n (%)				0.982
Female	107 (21.1)	97 (21.1)	10 (21.3)	
Male	399 (78.9)	362 (78.9)	37 (78.7)	
Medical history				
Hypertension, n (%)	264 (52.2)	245 (53.4)	19 (40.4)	0.090
Diabetes mellitus, n (%)	137 (27.1)	127 (27.7)	10 (21.3)	0.348
Dyslipidemia, n (%)	147 (29.1)	139 (30.3)	8 (17.0)	0.056
Smoking, n (%)	335 (66.2)	302 (65.8)	33 (70.2)	0.542
Alcohol abuse, n (%)	54 (10.7)	52 (11.3)	2 (4.3)	0.135
Laboratory data				
Potassium, mmol/L	4.0 ± 0.4	4.0 ± 0.4	4.0 ± 0.5	0.688
Sodium, mmol/L	139.7 ± 3.0	139.7 ± 3.1	139.0 ± 2.6	0.146
Serum creatinine, μmol/L	77.0 (66.0–96.0)	76.0 (65.0–94.0)	90.0 (71.0–107.0)	0.003
Serum uric acid, μmol/L	360.6 ± 107.4	358.7 ± 108.1	379.4 ± 100.5	0.210
White blood cell count, $\times 10^9$/L	9.8 ± 3.9	9.7 ± 3.9	10.1 ± 3.3	0.559
Hemoglobin, g/L	131.4 ± 22.8	132.2 ± 22.9	123.9 ± 20.3	0.017
In-hospital events				
Severe ventricular arrhythmia, n (%)	132 (26.1)	119 (25.9)	13 (27.7)	0.797

post-MI HF, heart failure after myocardial infarction; SVA, severe ventricular arrhythmias.

Association Between Hb Levels, Anemia Status, and post-MI HF

In univariate logistic regression analysis (**Table S2**), Hb, as a continuous variable, was negatively associated with the risk of post-MI HF; for every 1 g/L increase in Hb, the risk of post-MI HF decreased by approximately 2.0% (OR = 0.98, 95% CI 0.96–0.99, P = 0.010). After adjusting for age and sex, this association remained statistically significant (Model 1: OR = 0.981, 95% CI 0.963–0.998, P = 0.029). After further adjustment for infarct location, blood pressure, traditional cardiovascular risk factors, and lifestyle factors, the association weakened and became marginally statistically significant (Model 2: OR = 0.984, 95% CI 0.966–1.002, P = 0.090). In the fully adjusted model, Hb remained significantly negatively associated with the risk of post-MI HF (Model 3: OR = 0.979, 95% CI 0.960–0.998, P = 0.029) (**Table 2**).

Table 2. Associations of hemoglobin and anemia with heart failure after myocardial infarction.

Variable	Crude		Model 1		Model 2		Model 3	
	OR (95% CI)	***P*-value**	**OR (95% CI)**	***P*-value**	**OR (95% CI)**	***P*-value**	**OR (95% CI)**	***P*-value**
Hemoglobin (continuous)	0.980 (0.964–0.995)	0.010	0.981 (0.963–0.998)	0.029	0.984 (0.966–1.002)	0.090	0.979 (0.960–0.998)	0.029
Anemia								
No	1 (Ref)	—	1 (Ref)	—	1 (Ref)	—	1 (Ref)	—
Yes	3.22 (1.74–5.98)	<0.001	2.94 (1.53–5.66)	0.001	2.78 (1.43–5.41)	0.002	3.15 (1.56–6.39)	0.001

Crude, unadjusted; Model 1, adjusted for age and sex; Model 2, adjusted for age, sex, hypertension, diabetes mellitus, dyslipidemia, smoking, and alcohol abuse; Model 3, Model 2 plus serum potassium, sodium, serum creatinine, serum uric acid, white blood cell count, and severe ventricular arrhythmia.

Anemia was defined as hemoglobin <110 g/L in women and <120 g/L in men. OR, odds ratio; CI, confidence interval; Ref, reference category.

After defining anemia based on sex-specific Hb thresholds, 113 of the 506 patients were found to be anemic. The incidence of post-MI HF in the anemia group was 18.6% (21/113), significantly higher than the 6.6% (26/393) in the non-anemia group. In the unadjusted model, the risk of post-MI HF in patients with anemia was approximately 3.22 times that of patients without anemia (OR = 3.22, 95% CI 1.74–5.98, P < 0.001).

This association remained stable after multivariable adjustment: in Model 1, the OR was 2.94 (95% CI 1.53–5.66, P = 0.001); in Model 2, after further adjustment for clinical characteristics, the OR was 2.78 (95% CI 1.43–5.41, P = 0.002); in the fully adjusted model, the OR was 3.15 (95% CI 1.56–6.39, P = 0.001) (**Table 2**).

Dose-Response Relationship

Restricted cubic spline analysis showed that, using a median Hb level of 131 g/L as the reference point, there was an overall association between Hb levels and the risk of post-MI HF (P for overall = 0.041). As Hb levels increased, the risk of post-MI HF generally showed a downward trend; the test for nonlinearity did not reach statistical significance (P for nonlinearity = 0.236), suggesting that the association between Hb and post-MI HF is closer to a linear inverse association rather than a clearly nonlinear relationship (**Figure 2**).

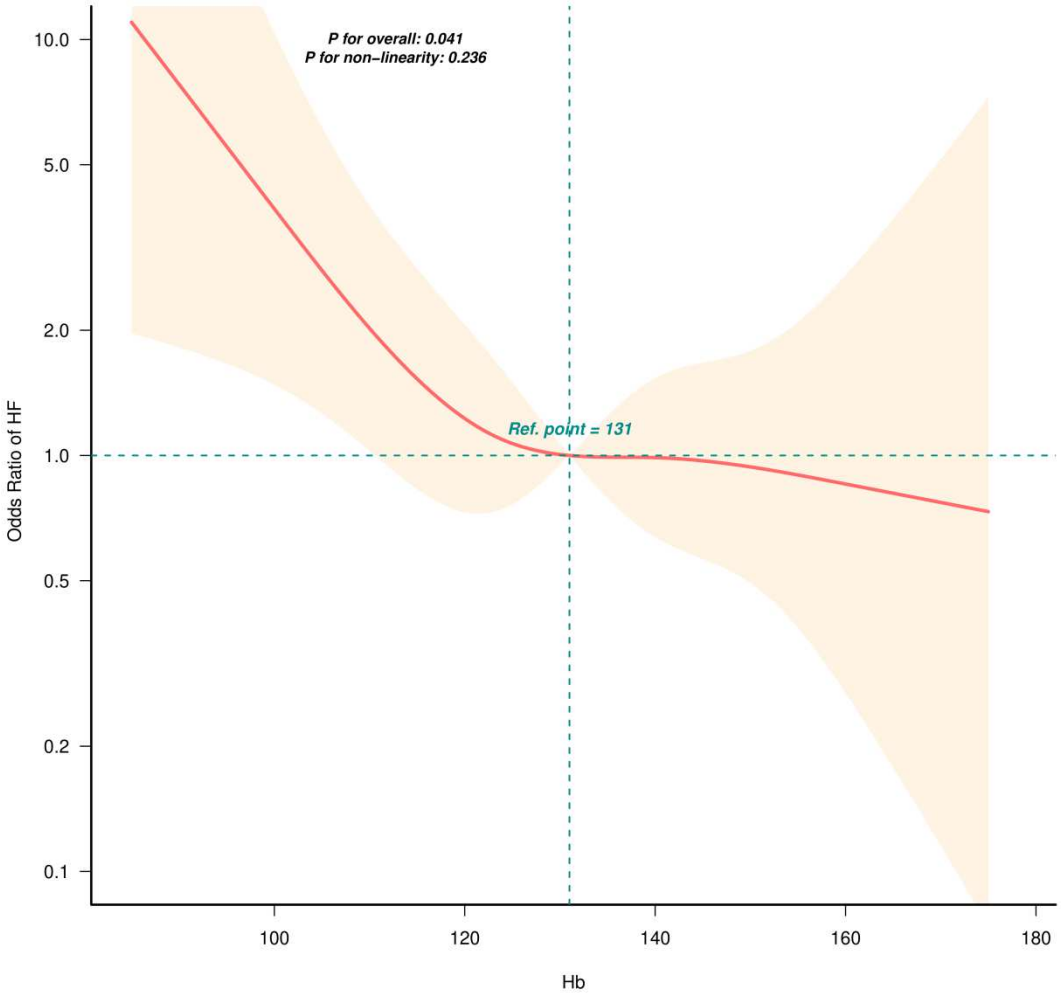


Figure 2. Restricted cubic spline showing the association between admission hemoglobin level and the odds ratio of heart failure after acute myocardial infarction. The reference point is the median hemoglobin level (131 g/L). P for overall = 0.041; P for nonlinearity = 0.236.

Subgroup and Interaction Analyses

In the pre-specified subgroup analyses, the Hb-post-MI HF association was largely consistent across most clinical strata. Stratification by age, sex, hypertension, and dyslipidemia yielded no significant interactions (P = 0.941 for age, P = 0.786 for sex, P = 0.375 for hypertension, and P = 0.847 for dyslipidemia).

Notably, there was a statistically significant interaction between Hb levels and diabetes status and smoking status. Interaction plots further suggest that the relationship curves between Hb levels and the probability of post-MI HF differ across different

diabetes and smoking statuses (**Figure 3 and Figure S1**). Therefore, diabetes and smoking may modulate the association between Hb levels and post-MI HF; however, this finding requires further validation in larger samples or external cohorts.

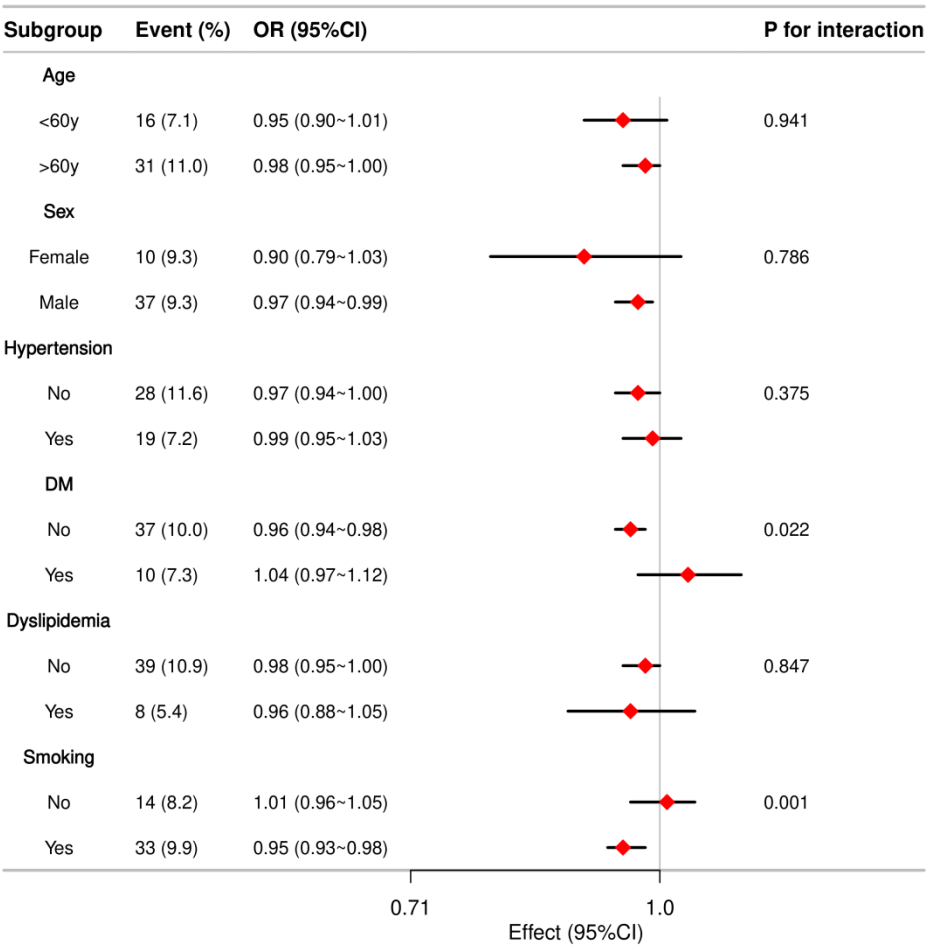


Figure 3. Forest plot of subgroup and interaction analyses for the association between hemoglobin and heart failure after acute myocardial infarction.

Sensitivity Analysis

An ROC curve was plotted based on the predictive probabilities from the multivariate logistic regression model. The AUC for distinguishing between post-MI HF and without post-MI HF patients was 0.771 (95% CI 0.699–0.843), indicating that the model possesses a certain degree of discriminatory ability (**Figure 4**).

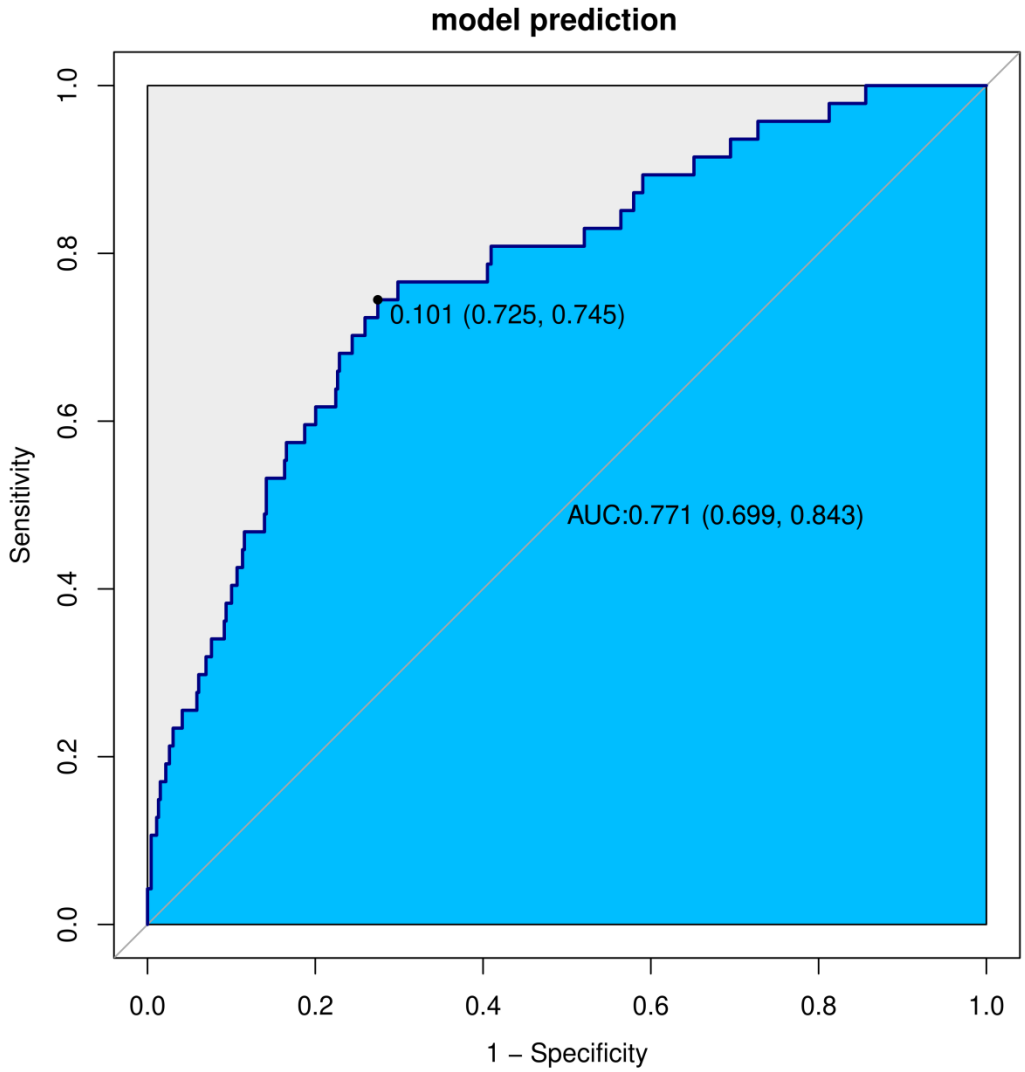


Figure 4. Receiver operating characteristic curve of the multivariable logistic regression model for discriminating heart failure after acute myocardial infarction (AUC = 0.771, 95% CI 0.699-0.843).

Multicollinearity analysis showed that the VIFs for all variables included in the model were low. Among these, the VIFs for systolic and diastolic blood pressure were relatively high at 3.830 and 3.726, respectively, but still below commonly used warning thresholds; the VIFs for the remaining variables were low, suggesting that there was no significant multicollinearity in the model (**Table S1**).

A sensitivity analysis for unmeasured confounders showed that the E-value for the association between anemia and post-MI HF in the fully adjusted model was 5.75, with an E-value corresponding to the lower limit of the 95% CI of 2.49 (**Figure S2**). After excluding patients with missing covariate data, a total of 496 patients were included in

the analysis, of whom 45 developed post-MI HF. In the fully adjusted model, a 1 g/L increase in Hb was still associated with a reduced risk of post-MI HF (OR = 0.979, 95% CI 0.960–0.998, P = 0.029); the risk of post-MI HF remained significantly elevated in patients with anemia (OR = 3.12, 95% CI 1.53–6.35, P = 0.001) (**Table S3**).

Discussion

This study systematically evaluated the association between admission hemoglobin levels and anemia status and post-MI HF during hospitalization in patients with acute STEMI who underwent emergency PCI. The results showed that for every 1 g/L increase in hemoglobin, the risk of post-MI HF decreased by approximately 2.3%; patients with anemia had a risk of post-MI HF approximately 3.73 times higher than those without anemia, and this association remained stable after stepwise adjustment for covariates. Restricted cubic spline analysis indicated an approximately linear inverse relationship between hemoglobin and the risk of post-MI HF.

The prognostic significance of anemia in patients with AMI and HF has been confirmed in multiple studies. Tanaka et al. reported that anemia is associated with adverse cardiovascular outcomes in patients with acute myocardial infarction[20]; a systematic review and meta-analysis of patients with acute HF similarly demonstrated that anemia is significantly associated with all-cause mortality and adverse outcomes[21]. With regard to heart failure, a post hoc analysis of the EMPEROR-Reduced trial found that anemia was associated with poor prognosis in patients with HF with reduced ejection fraction, and that the benefits of empagliflozin were consistent across both anemic and non-anemic patients[22]. These studies are consistent with the findings of the present study, namely that lower circulating hemoglobin levels are associated with poorer clinical outcomes. It is worth noting that the MINT randomized trial—which examined red blood cell transfusion strategies in patients with AMI and anemia—and its subsequent patient-level meta-analysis indicate that anemia and hemoglobin levels are key decision-making variables in the management of AMI patients; however, the relationship between transfusion threshold strategies and clinical outcomes remains

controversial[23-26]. The value of this study lies in its focus on the specific outcome of new-onset heart failure during hospitalization, rather than on general mortality or composite endpoints. This approach provides a more direct answer regarding the relationship between hemoglobin levels and post-myocardial infarction cardiac decompensation, and supplements the relatively scarce evidence available for patients with STEMI in the era of emergency PCI[2, 3, 27, 28].

The mechanisms by which reduced hemoglobin levels increase the risk of heart failure following a myocardial infarction may involve multiple factors. First, hemoglobin is the primary oxygen-carrying component of blood; anemia can lead to reduced myocardial oxygen supply, exacerbating the imbalance between myocardial oxygen supply and demand in the context of acute ischemia, thereby amplifying ischemic damage and impeding the recovery of myocardial function[29]. Second, anemia can promote ventricular remodeling and the onset of heart failure by activating the sympathetic nervous system and the renin-angiotensin-aldosterone system, thereby increasing heart rate and myocardial oxygen consumption, and by exacerbating volume load through sodium and water retention[29, 30]. Furthermore, anemia is often intertwined with renal insufficiency, constituting what is known as the cardio-renal anemia syndrome. Chronic kidney disease is not only a major cause of anemia but also an independent risk factor for heart failure following myocardial infarction, suggesting that the association observed in this study may partially reflect common pathological processes along the cardio-renal axis[30]. Furthermore, iron deficiency—a major cause of anemia — is known to mediate mitochondrial energy metabolism disorders, myocardial remodeling, and inflammatory pathways that are believed to contribute to the onset and progression of HF[12, 31]. Therefore, the association between Hb levels and HF may reflect not only the direct effects of anemia on oxygen supply but also the underlying pathophysiological mechanisms, such as iron deficiency, inflammation, and cardiorenal interactions.

Subgroup analysis showed that the association between hemoglobin and post-MI HF was consistent across most clinical subgroups, but there were signs of statistical

interaction with diabetes status and smoking status (interaction P-values of 0.022 and 0.001, respectively). This phenomenon may be related to the following mechanisms: On the one hand, patients with diabetes often have concomitant microvascular disease, autonomic dysfunction, and renal impairment, which may alter the impact of anemia on myocardial oxygen supply and cardiac function[32, 33]; On the other hand, in smokers the binding of carbon monoxide to hemoglobin raises carboxyhemoglobin, so routine hemoglobin readings may not faithfully represent effective oxygen-carrying capacity, giving rise to an apparent effect modification[34, 35]. However, interaction tests typically involve multiple comparisons, and given the limited sample size and low number of events in this study, these interaction signals are more likely to be exploratory findings rather than definitive conclusions and require further validation in larger samples or external cohorts.

Several limitations of this work merit mention. First, it was a single-center, retrospective design with a modest sample (506 patients, 47 of whom developed post-MI HF) and few events, which may compromise the stability of the multivariable models. Second, hemoglobin levels were assessed based solely on a single measurement at admission, failing to reflect dynamic changes in hemoglobin levels during hospitalization; however, the trajectory of hemoglobin decline may have additional prognostic value. Third, outcome determination relied on retrospective medical records, which carries potential diagnostic and information biases. Fourth, although E-value analysis suggested that unmeasured confounders posed a limited threat to the results, the influence of residual confounders cannot be completely ruled out. Fifth, this study lacks long-term follow-up data and was therefore unable to assess the relationship between hemoglobin levels and the incidence of long-term heart failure or rehospitalization.

Conclusion

Among patients with STEMI undergoing emergency PCI, lower admission hemoglobin levels and anemia are independently associated with an increased risk of post-MI HF during hospitalization, with the two factors exhibiting an approximately

linear inverse relationship. Admission hemoglobin levels may serve as a simple marker for early risk stratification of post-MI HF; however, the underlying mechanisms and potential value of intervention still require further validation in larger, multicenter, prospective studies.

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Conflict of Interest

The authors declare no conflicts of interest.

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None

Author Contributions

Y.X. and H.X. conceived and designed the study; N.W. and C.H. collected the data; Y.W. and S.L. analysed the data and revised the manuscript. All authors read and approved the final manuscript.

Data Availability Statement

The data that support the findings of this study are available from the corresponding authors upon reasonable request.

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