BMC Cardiovascular Disorders

RESEARCH

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Prognostic value of hemoglobin to serum creatinine ratio in ST-elevation myocardial infarction: a secondary analysis based on a cohort study

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Abstract

Introduction The long-term relationship between hemoglobin to serum creatinine (HB/SCr) ratio and clinical outcomes in ST-elevation myocardial infarction (STEMI) remains uncertain. This study aimed to determine the predictive value of the HB/SCr ratio for long-term major adverse cardiovascular events (MACE) in patients with STEMI.

Methods This study was based on a prospective cohort conducted in China, which included 460 STEMI patients who successfully underwent primary percutaneous coronary intervention. Cox proportional hazards models were utilized to explore the relationship between the HB/SCr ratio and MACE in STEMI patients over a 30-month follow-up period. The predictive value of the HB/SCr ratio for MACE was assessed using the receiver operating characteristic curve.

Results A total of 118 patients (26%) developed MACE during the follow-up period. After adjusting for confounding factors, a lower HB/SCr ratio emerged as a significant predictor of MACE in STEMI patients. Subgroup analyses indicated that the HB/SCr ratio was inversely associated with MACE in patients aged ≥ 60 years, males, those with a history of hypertension, individuals experiencing anterior wall myocardial infarction, patients classified as Killip grade I, and those receiving single stent implantation. Sensitivity analysis revealed that the inverse association between the HB/SCr ratio and MACE occurrence persisted in patients with normal hemoglobin levels. The area under the curve for the HB/SCr ratio in predicting MACE was 0.611.

Conclusions The baseline HB/SCr ratio was inversely associated with MACE, suggesting that it may serve as a useful biomarker for identifying high-risk STEMI patients at an early stage.

Keywords HB/SCr ratio, Creatinine, ST-elevation myocardial infarction, Prognostic, Cohort study

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Introduction

ST-elevation myocardial infarction (STEMI) is recognized as the most severe form of acute coronary syndrome, characterized by nearly complete or total occlusion of the coronary arteries. This leads to ischemic necrosis of the myocardium, posing a significant threat to both cardiac function and the patient's life [1]. Adverse events are common in STEMI patients during both the acute and chronic phases, even following successful revascularization through primary percutaneous coronary intervention (PCI). Identifying high-risk STEMI patients early on allows clinicians to administer more aggressive and comprehensive treatment strategies. Previous studies have shown that various easily identifiable biomarkers hold significant predictive power in the evaluation of patients presenting with STEMI [2–6].

Hemoglobin and serum creatinine are standard tests performed upon admission for all STEMI patients. The predictive significance of impaired renal function and reduced hemoglobin levels for adverse outcomes in STEMI patients has garnered increasing clinical interest [7–15]. Moreover, these two risk factors may exert additive or synergistic effects on adverse outcomes following PCI [16, 17]. A recent study has indicated that the hemoglobin to serum creatinine (HB/SCr) ratio at admission is inversely correlated with bleeding complications and in-hospital mortality in non-dialysis patients post-PCI [18]. However, there is a paucity of research establishing whether the HB/SCr ratio is associated with long-term adverse outcomes in STEMI patients. The relationship between the HB/SCr ratio and adverse outcomes might be influenced by various factors, including age, gender, and clinical conditions. Consequently, the present study aimed to explore the association between the HB/SCr ratio and the long-term risk of major adverse cardiovascular events (MACE) in STEMI patients after primary PCI.

Materials and methods

Study design and population

This present study was a secondary analysis of a longitudinal study conducted at First People's Hospital of Taizhou in China [19]. The original data was freely extracted from the Dryad database (https://datadryad.org) as provided by Yang et al. [20]. Researchers were granted permission to utilize this data for secondary analysis, ensuring that the rights of the original authors were not infringed upon, in compliance with Dryad's terms of service. In the original research, 464 STEMI patients were collected non-selectively and consecutively from the First People's Hospital of Taizhou between January 2010 and October 2014. The diagnostic criteria for STEMI included persistent chest pain lasting more than 30 min, prolonged electrocardiographic alterations (ischemic ST-segment elevation in two or more contiguous leads and/or depression), and significantly elevated serum myocardial enzyme and troponin levels. All patients underwent successful primary PCI revascularization within 12 h of symptom onset. The detailed study design, exclusion criteria, and treatment process have been previously reported [19]. After excluding 2 patients with a baseline creatinine level above 133 μmol/L and 2 with missing hemoglobin data, the final analysis included 460 patients. Informed consent was obtained from every participant. Given the anonymization of all data and adherence to Dryad database guidelines throughout the analysis, ethical approval was not required for this project.

Data collection

At the time of admission, demographic and clinical data were meticulously gathered from each patient. This information encompassed their age, gender, systolic blood pressure, heart rate, Killip classification, presence of anterior wall myocardial infarction, and a detailed medical history, including previous myocardial infarctions, hypertension, and diabetes mellitus. Post-admission, the following blood biochemical indicators were assessed: hemoglobin, white blood cell (WBC) count, percentage of neutrophils, platelet count, creatinine, albumin, urea nitrogen, uric acid, fasting blood glucose (FBG), high-density lipoprotein cholesterol (HDL-C), total cholesterol (TC), triglycerides (TG), low-density lipoprotein cholesterol (LDL), peak cardiac troponin I (cTnI), and creatine kinase-MB (CK-MB). Notably, blood samples for hemoglobin and serum creatinine levels were obtained immediately upon the patients' arrival, prior to the initiation of primary PCI. Electrocardiographic and echocardiographic parameters (collected within 5 days post-PCI) included pathological Q-waves, left atrial diameter, and left ventricular end-diastolic diameter (LVEDD). The number of stents implanted, culprit vessels, and the Gensini score were determined by the interventional cardiologists.

Definitions

The HB/SCr ratio was calculated by dividing a person's hemoglobin in g/ L by the serum creatinine in μmol/ L. MACE was the interesting outcome that included recurrent target vessel myocardial infarction, clinically driven target lesion revascularization, congestive heart failure, cardiogenic shock or cardiac death. All patients received a 30-month clinical follow-up after PCI.

Statistical analysis

Patients were stratified into two groups based on the median HB/SCr ratio: a high HB/SCr ratio group (>1.96) and a low HB/SCr ratio group (≤ 1.96). Continuous

variables with a normal distribution were reported as mean ± standard deviation (SD), while those with a non-normal distribution were reported as median (25th and 75th percentiles). Categorical variables were expressed as absolute numbers and percentages. Continuous variables were compared using Student's t-test or the Mann–Whitney U test, depending on their distribution, and categorical variables were compared using the chi-square test.

Cox proportional hazard models were utilized to investigate the impact of the HB/SCr ratio on the occurrence of MACE. The model was adjusted for a set of predefined variables based on clinical relevance and their established association with MACE in patients with STEMI who underwent primary PCI. Results were presented as hazard ratios (HR) with 95% confidence intervals (CI). The cumulative incidence of MACE in the low- and high-HB/SCr ratio groups was estimated using Kaplan-Meier analysis and compared using the log-rank test. The predictive ability of the HB/SCr ratio, hemoglobin, and serum creatinine for MACE incidence was evaluated using the receiver operating characteristic (ROC) curve and the area under the curve (AUC), where sensitivity was plotted against 1-specificity. The optimal cut-off value was determined using the Youden Index.

Subgroup analyses were conducted based on age, gender, history of hypertension, history of diabetes mellitus, pathological Q wave, anterior wall myocardial infarction, Killip class, and number of stents using Cox proportional hazard models. To ensure the robustness of our findings, a sensitivity analysis was performed, excluding participants with abnormal hemoglobin levels (males: <120 g/L, > 160 g/L; females: <110 g/L, > 150 g/L).

All statistical analyses were conducted using R version 3.4.4. A P value of less than 0.05 was considered statistically significant.

Results

The demographic and clinical characteristics of the study cohort are detailed in Table 1. The mean age of the 460 STEMI patients was 63 ± 12 years, with 77% being male. Patients in the high HB/SCr ratio group were generally younger and had higher hemoglobin levels compared to those in the low HB/SCr ratio group. Conversely, creatinine levels were lower in the high HB/SCr ratio group. Notably, a higher proportion of patients in the low HB/SCr ratio group had the anterior descending vessel as the culprit vessel. Over the course of the follow-up, 118 (26%) patients experienced MACE. A comparison of clinical characteristics between the MACE and non-MACE groups is presented in Table S1. Patients with a low HB/SCr ratio exhibited a significantly higher risk of developing MACE compared to those with a high HB/SCr ratio (log-rank test, $P = 0.02$) (Fig. 1).

In the univariate Cox regression analysis, the HB/SCr ratio, age, pathological Q wave, AWMI, LVEDD, left atrial diameter, Killip's grade, creatinine, hemoglobin, TC and HDL were found be significantly associated with MACE (Table S2).

Three Cox proportional hazard models were constructed to explore the relationship between the HB/SCr ratio and MACE (Table 2). The unadjusted Model 1 revealed that a decreased HB/SCr ratio was inversely associated with an increased risk of MACE (HR, 0.649; 95% CI, 0.450–0.935). Model 2 included further adjustments for age, gender, history of myocardial infarction, hypertension, diabetes mellitus, pathological Q wave, and anterior wall myocardial infarction. This model indicated an increased risk of MACE in STEMI patients with a low HB/SCr ratio compared to those with a high HB/SCr ratio (HR, 0.660; 95% CI, 0.454–0.958). In the fully-adjusted Model 3, which included all variables presented in Table 1, there was also an inverse association between the HB/SCr ratio and the development of MACE (HR, 0.602; 95% CI, 0.381–0.952).

Figure 2 presents the results of the subgroup analyses. A low HB/SCr ratio emerged as a significant independent predictor of MACE occurrence in STEMI patients who were aged ≥ 60 years (HR, 0.520; 95% CI, 0.319–0.849), male (HR, 0.585; 95% CI, 0.361–0.949), had a history of hypertension (HR, 0.389; 95% CI, 0.225–0.673), experienced an anterior wall myocardial infarction (HR, 0.449; 95% CI, 0.257–0.787), were classified as Killip class I (HR, 0.525; 95% CI, 0.320–0.861), and received a single stent implantation (HR, 0.355; 95% CI, 0.145–0.775) (Fig. 2). After excluding 131 patients with abnormal hemoglobin levels (males: <120 g/L, > 160 g/L; females: <110 g/L, > 150 g/L), the inverse association between the HB/SCr ratio and MACE occurrence persisted in STEMI patients (HR, 0.535; 95% CI, 0.321–0.890).

The ROC curve analysis was used to evaluate the predictive value of the HB/SCr ratio, hemoglobin, and serum creatinine for MACE in STEMI patients (Fig. 3). The AUC values for predicting MACE were 0.611 (95% CI, 0.551–0.670) for the HB/SCr ratio, 0.598 (95% CI, 0.539–0.656) for hemoglobin, and 0.418 (95% CI, 0.358–0.478) for serum creatinine, respectively (Fig. 3). When the HB/SCr ratio cutoff was set at 1.67 for predicting MACE, it yielded the highest Youden Index (0.197), with a sensitivity of 81.6% and a specificity of 38.1%.

Discussion

This cohort study was designed to assess the association between the HB/SCr ratio and the occurrence of MACE in STEMI patients following PCI. The findings confirmed that a low HB/SCr ratio in STEMI patients was an independent predictor of MACE occurrence. Subgroup analyses revealed that among patients aged ≥ 60 years, males,

Table 1 Relationships between clinical and laboratory data and hemoglobin to serum creatinine ratio in patients with ST-elevation myocardial infarction

Variables	Total ($n=460$)	HB/SCr ratio		*p*
		Low,≤1.96,$N=228$	High,>1.96,$N=232$	
Age (years)	63 ± 12	64 ± 12	62 ± 11	0.035
Gender, n (%)				0.380
Males	354 (77)	171 (75)	183 (79)	
Females	106 (23)	57 (25)	49 (21)	
Hypertension history, n (%)				0.798
No	198 (43)	100 (44)	98 (42)	
Yes	262 (57)	128 (56)	134 (58)	
Diabetes history, n (%)				0.441
No	312 (68)	159 (70)	153 (66)	
Yes	148 (32)	69 (30)	79 (34)	
Myocardial infarction history, n (%)				0.714
Yes	54 (12)	25 (11)	29 (12)	
No	406 (88)	203 (89)	203 (88)	
Pathological Q wave, n (%)				0.111
No	238 (52)	127 (56)	111 (48)	
Yes	222 (48)	101 (44)	121 (52)	
AWMI, n (%)				1.000
No	230 (50)	114 (50)	116 (50)	
Yes	230 (50)	114 (50)	116 (50)	
Culprit vessels, n (%)				0.021
LAD	231 (50)	123 (54)	108 (47)	
LCX	72 (16)	25 (11)	47 (20)	
RCA	157 (34)	80 (35)	77 (33)	
LVEDD (mm)	50 (45, 56)	50 (45, 56)	50 (45, 56)	0.160
Left atrial diameter (mm)	38 (33, 42)	38 (34, 42)	38 (33, 41)	0.442
GENSINI score	74 (43, 101)	69 (35, 100)	76 (49, 103)	0.082
Stent number n (%)				0.063
1	300 (65)	150 (66)	150 (65)	
2	146 (32)	67 (29)	79 (34)	
3	14 (3)	11 (5)	3 (1)	
SBP (mmHg)	131 (109, 153)	131 (111, 150)	131 (109, 155)	0.670
Heart rate (beats/min)	76 (64, 89)	75 (64, 90)	77 (65, 88)	0.872
Killip's grade, n (%)				0.831
I	350 (76)	172 (75)	178 (77)	
≥II	110 (24)	56 (25)	54 (23)	
FBG (mmol/l)	7.1 (5.8, 9.7)	6.9 (5.6, 9.5)	7.5 (5.9, 9.8)	0.161
Neutrophils (%)	77.6 (66.2, 85.1)	78.4 (68.3, 85.8)	75.55 (65.4, 84.8)	0.073
WBC ($\times 10^9$/ L)	10.0 (7.2, 13.0)	9.3 (7.1, 12.0)	10.2 (7.9, 13.3)	0.073
Platelet ($\times 10^9$/ L)	231 (183, 272)	233 (188, 281)	231 (183, 271)	0.328
Urea nitrogen (mmol/L)	6.7 ± 2.1	6.7 ± 2.0	6.7 ± 2.1	0.864
Creatinine (μmol/ L)	75 (62, 86)	86 (78, 91)	63 (53, 70)	< 0.001
Uric acid (μmol/ L)	334 (282, 390)	345 (290, 393)	330 (275, 383)	0.091
Hemoglobin (g/ L)	144 (131, 158)	139 (128, 153)	151 (136, 161)	< 0.001
Albumin (g/ L)	38 (35, 41)	38 (35, 41)	38 (35, 41)	0.299
TC (mmol/ L)	5.6 ± 1.1	5.7 ± 1.2	5.6 ± 1.1	0.877
TG (mmol/ L)	1.0 (0.6, 1.5)	1.0 (0.5, 1.5)	1.0 (0.6, 1.6)	0.615
HDL (mmol/ L)	1.2 ± 0.3	1.2 ± 0.3	1.2 ± 0.3	0.739
LDL (mmol/ L)	3.0 (2.5, 3.6)	2.9 (2.4, 3.5)	3.0 (2.5, 3.6)	0.292
Peak cTnI (ng/m L)	14 (4, 29)	12 (4, 27)	16 (5, 31)	0.451
Peak CK-MB (U/ L)	107 (44, 195)	102 (40, 192)	116 (52, 197)	0.148
MACE, n (%)				0.033

Table 1 (continued)

Variables	Total ($n=460$)	HB/SCr ratio		*p*
		Low,≤1.96,$N=228$	High,>1.96,$N=232$	
No	342 (74)	159 (70)	183 (79)	
Yes	118 (26)	69 (30)	49 (21)	

Abbreviations: HB/SCr, hemoglobin to serum creatinine; SBP, systolic blood pressure; AWMI, Anterior wall myocardial infarction; LVEDD, left ventricular and diastolic diameter; WBC, white blood cells; FBG, fasting blood glucose; TC, total cholesterol; TG, triglyceride; HDL-C, high-density lipoprotein; LDL-C, low-density lipoprotein-cholesterol; cTnI, cardiac troponin I; CK-MB, creatine kinase MB; LAD, left anterior descending coronary artery; LCX, left circumflex coronary artery; RCA, right coronary artery; MACE, major adverse coronary events

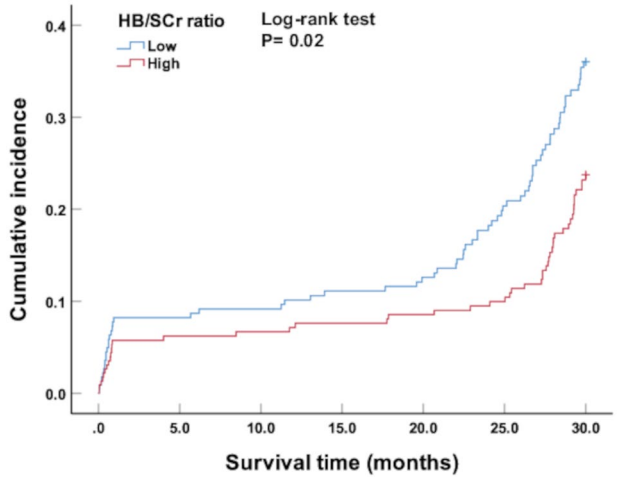

Fig. 1 Kaplan-Meier curves stratified by HB/SCr ratio. The curves showed different incidence of MACE of STEMI patients with different HB/SCr ratio

Table 2 Cox regression analysis for the relationship between HB/SCr ratio and MACE

HB/SCr ratio (low vs. high)	HR (95%CI)	*P*-value
Model 1	0.649(0.450–0.935)	0.021
Model 2	0.624(0.430–0.907)	0.013
Model 3	0.623(0.419–0.926)	0.019

Note: Model 1was crude model with no variables adjusted

Model 2 adjusted for age, gender, hypertension history, diabetes history, myocardial infarction history, pathological Q wave and anterior wall myocardial infarction

Model 3 adjusted for age, gender, hypertension history, diabetes history, myocardial infarction history, pathological Q wave, anterior wall myocardial infarction, Killip's grade, systolic blood pressure, heart rate, left ventricular and diastolic diameter, left atrial diameter, Gensini score, culprit vessels, stent number, white blood cells, percentage of neutrophils, albumin, platelets, urea nitrogen, fasting blood glucose, uric acid, total cholesterol, triglyceride, high-density lipoprotein, low-density lipoprotein-cholesterol, peak cardiac troponin I and peak creatine kinase-MB

those with a history of hypertension, those experiencing an anterior wall myocardial infarction, those classified as Killip class I, and those receiving a single stent implantation, a low HB/SCr ratio was found to have statistically significant prognostic value in predicting the occurrence of MACE. Notably, the inverse association between the HB/SCr ratio and MACE occurrence persisted in STEMI patients with normal hemoglobin levels. Furthermore, the HB/SCr ratio was a more robust predictor of MACE occurrence in STEMI patients compared to either hemoglobin or creatinine alone.

Only one previous study has explored the association between the HB/SCr ratio and clinical outcomes in patients post-PCI [18]. In that study, the HB/SCr ratio was found to be negatively correlated with mortality and hemorrhagic complications during hospitalization in patients post-PCI [18]. Unlike the previous study, our study focused on STEMI patients who received a 30-month clinical follow-up post-PCI. After adjusting for risk factors associated with MACE, the inverse association between the HB/SCr ratio and the risk of MACE occurrence remained significant, suggesting that the HB/SCr ratio is an independent risk factor. Consequently, this study provides valuable evidence supporting the HB/SCr ratio as a prognostic indicator, although further research is warranted.

Numerous studies have established that impaired renal function and decreased hemoglobin are strongly linked to poorer clinical outcomes following PCI [7–15]. Many patients present with concurrent anemia and renal impairment, yet there has been a paucity of research examining the combined impact of decreased hemoglobin and elevated creatinine. The coexistence of anemia and impaired renal function at admission is known to have a multiplicative effect on the risk of adverse outcomes in patients with acute myocardial infarction [16, 17]. The ROC analysis in our study indicated that the HB/SCr ratio possesses a higher predictive value than either hemoglobin or creatinine assessed individually. Moreover, our analysis suggested that an optimal cut-off point of 1.67 for the HB/SCr ratio in STEMI patients could be used for risk stratification. Unlike previous studies that focused solely on anemia and impaired renal function, our study also revealed that the HB/SCr ratio is negatively associated with the development of MACE in patients with normal hemoglobin and creatinine levels. Therefore, the HB/SCr ratio may serve as a more effective indicator for identifying high-risk patients following a STEMI diagnosis.

Previous research has developed several models that incorporate parameters related to hemoglobin and renal function to forecast adverse outcomes in patients with acute myocardial infarction [21–23]. While these established risk models are valuable, they share a common

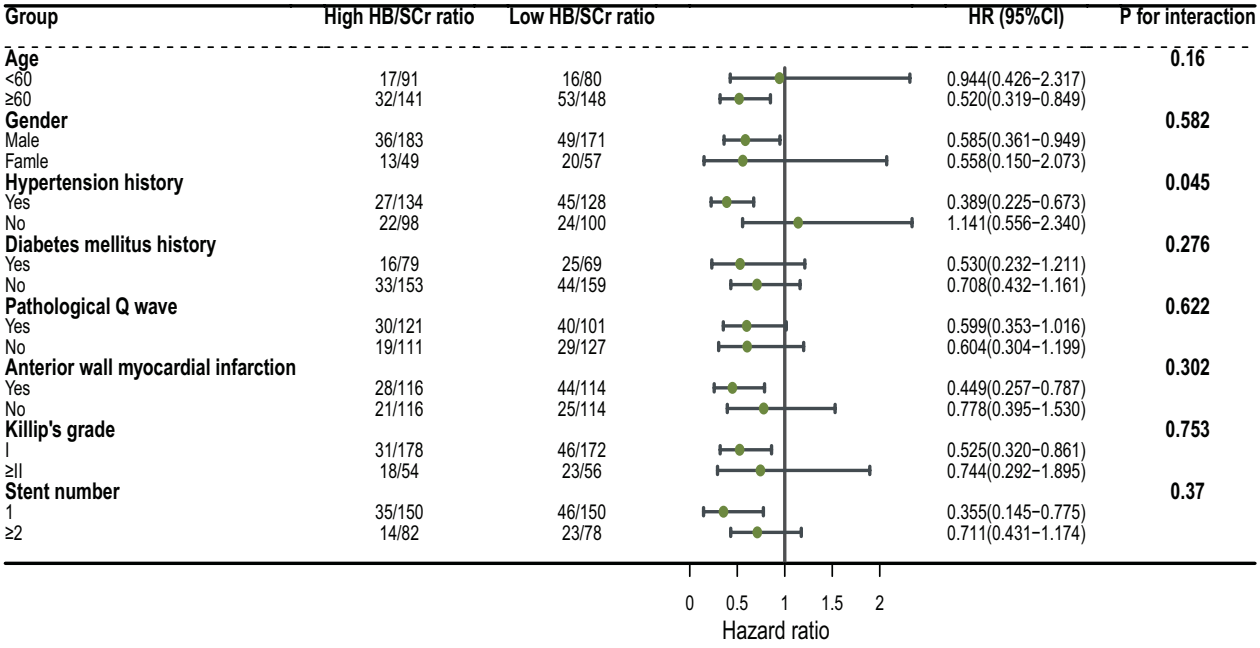

Fig. 2 Effect size of HB/SCr ratio on incident MACE in different subgroups. Each stratification was adjusted for age(<60 VS ≥ 60), gender, hypertension history, diabetes history, myocardial infarction history, pathological Q wave, Killip's grade, systolic blood pressure, heart rate, culprit vessels, anterior wall myocardial infarction, stent number, left ventricular and diastolic diameter, left atrial diameter, Gensini score, white blood cells, percentage of neutrophils, albumin, platelets, urea nitrogen, fasting blood glucose, uric acid, total cholesterol, triglyceride, high-density lipoprotein, low-density lipoprotein-cholesterol, peak cardiac troponin I and peak creatine kinase-MB, except the stratification factor itself

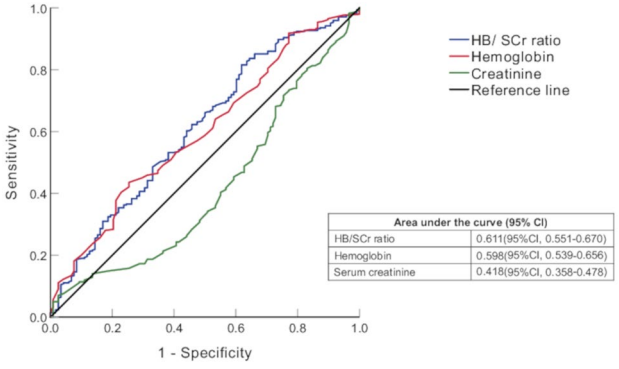

Fig. 3 Receiver operating characteristic curves for HB/SCr ratio, hemoglobin, and serum creatinine for predicting the incidence of MACE in STEMI patients

limitation: they require clinicians to calculate the glomerular filtration rate or creatinine clearance for each patient [21–23]. Consequently, these models can be overly complex and time-consuming, particularly in the critical setting before emergency PCI procedures. Although serum creatinine levels do not provide an exact measure of a patient's renal function, they are more practical in clinical practice than glomerular filtration rate or creatinine clearance because they are directly measured rather than calculated from other parameters using empirical algorithms. Additionally, the creatinine clearance rate cannot always be determined due to incomplete patient information, such as weight [24], which is especially common among STEMI patients. A risk assessment tool must be convenient and straightforward to use, particularly in emergency situations. Since hemoglobin and creatinine levels are routinely assessed, the Hb/Cr ratio could serve as a practical bedside tool due to its simplicity and ease of use. Most importantly, clinicians can quickly calculate this ratio for each STEMI patient at the bedside without the need for a calculator.

Several potential mechanisms may underlie the association between the Hb/Cr ratio and the risk of MACE in STEMI patients following successful primary PCI. The coexistence of anemia and impaired renal function in STEMI patients can directly exacerbate myocardial ischemia [7, 15, 16]. Additionally, anemia and impaired renal function can indirectly affect the prognosis of STEMI by causing poor oxygen delivery, exacerbating inflammation, inducing endothelial dysfunction, and activating the sympathetic nervous system and the renin-angiotensin-aldosterone system [7, 15, 16, 18]. However, patients with creatinine levels above 133 μmol/L were excluded from this study. Moreover, sensitivity analysis revealed that the inverse association of the Hb/SCr ratio with the development of MACE persisted in STEMI patients with normal hemoglobin levels. This finding is plausible. A previous study noted that cardiogenic shock, heart failure, and cardiac arrest were more frequent in patients with a low Hb/SCr ratio compared to those with a high ratio at

admission [18]. Furthermore, age is a significant, non-modifiable risk factor for adverse outcomes in STEMI patients [25, 26]. As demonstrated in our study, STEMI patients with a low Hb/Cr ratio tended to be older. More importantly, the proportion of the anterior descending vessel as the culprit vessel was higher in the low Hb/SCr ratio group, and anterior descending vessel infarctions are associated with a higher risk of heart failure, stroke, and death [27]. Therefore, the combination of hemoglobin and creatinine may still effectively identify STEMI patients, even those without anemia and impaired kidney function, who are at high risk for MACE.

The subgroup analyses conducted in this study reveal intriguing trends in the prognostic value of the HB/SCr ratio for MACE across various patient characteristics. While these findings suggest that certain subgroups, such as patients aged ≥ 60 years and those with a history of hypertension, may exhibit a stronger association between a low HB/SCr ratio and MACE, it is imperative to approach these results with caution due to the limited sample size within some subgroups and the potential for type I error. These exploratory analyses contribute to a more nuanced understanding of the HB/SCr ratio's prognostic significance and underscore the necessity for larger, diverse cohorts in future studies to validate these observations and explore potential effect modifiers. Until such data are available, these subgroup findings should be used to generate hypotheses for further research rather than to guide clinical practice.

In our analysis, we observed an inverse association between the HB/SCr ratio and the occurrence of MACE in in STEMI patients after primary PCI. Despite the absence of established treatment strategies for addressing underlying anemia and chronic kidney disease prior to PCI procedures [16, 17], there is evidence to suggest that the HB/SCr ratio could serve as a prognostic marker to identify patients at a higher risk for MACE. This suggests that these patients may benefit from more intensive post-PCI monitoring and management. For instance, patients with a lower HB/SCr ratio could be candidates for closer follow-up, more aggressive medical therapy, or consideration of additional preventative strategies. However, we caution that these implications are speculative and based on an observational analysis. The relationship between the HB/SCr ratio and MACE warrants further investigation in larger, multicenter, and preferably randomized controlled trials to establish causation and to guide the development of evidence-based treatment. Until such data are available, the utility of the HB/SCr ratio as a routine clinical tool remains to be determined.

This study has several limitations. First, it was a secondary analysis with limited adjusted factors, potentially missing unmeasured variables like alcohol use, smoking, drug use, and left ventricular ejection fraction that could affect STEMI outcomes. Second, the HB/SCr ratio was only evaluated at admission, missing its predictive value for changes during hospitalization. This timing issue might affect its interpretation as a prognostic marker, given that renal function can be impacted by the ischemic event itself. Third, the HB/SCr ratio's AUC of 0.611 indicates limited predictive power for MACE, suggesting it should be used cautiously alongside other clinical markers. Finally, the study's small, retrospective, single-center sample limits generalizability. Larger, multicenter, prospective studies are needed to confirm these findings and explore the impact of renal function assessment timing on the HB/SCr ratio's prognostic value.

Conclusion

This study demonstrated that the HB/SCr ratio was inversely associated with the occurrence of MACE in patients with STEMI after primary PCI. The HB/SCr ratio serves as a valuable bedside tool, proving more potent in predicting MACE than either hemoglobin or creatinine assessed in isolation. This finding could be significant for clinicians in screening STEMI patients who are at a higher risk of adverse outcomes.

Abbreviations

STEMI	St-segment elevation myocardial infarction
PCI	Percutaneous coronary intervention
HB/SCr	Hemoglobin to serum creatinine
MACE	Major adverse cardiovascular events
WBC	White blood cell
FBG	Fasting blood glucose
HDL-C	High-density lipoprotein cholesterol
TC	Total cholesterol
TG	Triglyceride
LDL	Low-density lipoprotein-cholesterol
cTnI	Cardiac troponin I
CK-MB	Creatine kinase-MB
LVEDD	Left ventricular diastolic diameter
SD	Standard deviation
HR	Hazard ratio
CI	Confidence interval
ROC	Receiver operating characteristic
AUC	Area under the curve

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12872-025-04856-9.

Supplementary Material 1

Supplementary Material 2

Acknowledgements

We would like to thank Yang Lingchang and its collaborators who prepared the publicly available data.

Author contributions

Weibiao Ji: Conceptualization, Methodology, Software. Yangbo Chen: Data curation, Writing- Original draft preparation. Haoyue Zhou: Visualization, Investigation. Weipeng Huang: Software, Validation. Shangbo Xu: Supervision, Writing- Reviewing and Editing. All authors have read and agreed to the published version of the manuscript.

Funding
None.

Data availability
The data that support the findings of this study are openly available in Dryad at https://doi.org/10.5061/dryad.pf56m.

Declarations

Ethics approval and consent to participate
Every participant has given their informed consent. Due to the anonymization of all data and compliance with Dryad database guidelines throughout data analysis, the ethics approval was no longer necessary for this project.

Consent for publication
Not Applicable.

Competing interests
The authors declare no competing interests.

Received: 17 August 2024 / Accepted: 12 May 2025
Published online: 24 May 2025

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