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Preoperative hemoglobin predicts clinical outcomes after percutaneous coronary intervention

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Abstract

Background Concurrent anemia is associated with an increased risk of major adverse cardiac events (MACE) in patients with myocardial infarction. The study aimed to assess the value of anemia and preoperative hemoglobin levels in predicting clinical outcomes in stable coronary artery disease (SCAD) patients receiving percutaneous coronary intervention (PCI).

Methods This is a secondary analysis based on a retrospective cohort study in which 204 patients with SCAD who received PCI were recruited. The primary outcome was major adverse cardiac events (MACE; including all-cause mortality, non-fatal myocardial infarction, and non-fatal stroke) and the secondary outcome was cardiovascular events (cardiovascular death, non-fatal myocardial infarction, and non-fatal stroke).

Results During a median follow-up of 783 days, MACE occurred in 28 patients. In multivariate COX regression analysis, after adjusting for potential confounding factors, low hemoglobin level independently predicted worse prognosis at the primary endpoint (HR = 0.72, 95% CI 0.56 to 0.93, $p = 0.012$) and the secondary endpoint (HR = 0.71, 95% CI 0.52 to 0.96, $p = 0.027$) after PCI. The receiver operating characteristic curve (ROC) showed that the best threshold for hemoglobin to predict MACE was 12.25 g/dl. In Kaplan-Meier analysis, hemoglobin < 12.25 g/dl predicted worse prognosis in MACE ($p < 0.001$).

Conclusions Low preoperative hemoglobin level increased the risk of MACE in SCAD patients receiving PCI and the optimal threshold for predicting MACE is 12.25 g/dl.

Keywords Stable coronary artery disease, Percutaneous coronary intervention, Anemia, Hemoglobin

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Introduction

Patients with stable coronary artery disease (SCAD) undergoing percutaneous coronary intervention (PCI) have a higher risk of cardiovascular adverse events. Several biomarkers such as soluble lectin-like oxidized low-density lipoprotein receptor-1, red blood cell distribution width, albumin, Follistatin-like 1, high-sensitivity C-reactive protein and mean platelet volume are reported as predictors of cardiovascular adverse events in SCAD patients [1–5].

Anemia is not uncommon in patients with coronary heart disease (CAD) and occurs in 25% of patients undergoing primary PCI [6]. The combination of CAD and anemia leads to an increased incidence of MACE [7]. Anemia has been shown to be an independent predictor of short-term cardiovascular events in patients with acute coronary syndrome (ACS) [8]. Anemia has been demonstrated as a predictor of thrombosis, including arterial clots (CAD and stroke) and venous clots (deep vein thrombosis and pulmonary embolism). Anemia is thought to induce a hyperdynamic circulation that triggers an inflammatory response, leading to increased thrombus formation [9]. Besides, accelerated erythrocyte aging [10] and death receptor on red blood cells [11] could mediate their association. However, the effect of anemia on long-term cardiovascular adverse events in SCAD patients after PCI remains unclear.

Besides, the influence of hemoglobin level on clinical outcomes after PCI is controversial. Sabatine et al. have shown that high or low hemoglobin levels are associated with increased mortality in patients with ACS [12]. Previous studies have shown that a low hemoglobin level is related to cardiovascular events after PCI in ACS patient [13]. The effect of low hemoglobin levels on long-term clinical events after PCI in SCAD has not been studied. The study aimed to assess the value of anemia and preoperative hemoglobin levels in predicting clinical outcomes in stable coronary artery disease (SCAD) patients.

Materials and methods

Data source

The data of this study is from the Dryad database (http://datadryad.org/). The Sho Suzuki study [3] is a retrospective cohort study conducted in a single center in Japan, in which 204 SCAD patients were recruited. Our study is a secondary analysis based on previously collected data. The data we used included clinical characteristics, major risk factors for CAD, comorbidities, laboratory data, echocardiography, medical history, angiographic data, and post-discharge follow-up findings. The work has been in accordance with the Declaration of Helsinki.

Definitions and endpoints

SCAD was defined as (1) coronary artery stenosis ≥ 90% (coronary angiography), or (2) coronary artery stenosis ≥ 75% (coronary angiography) with either a symptom of chest pain or stress-induced ischemia. According to the World Health Organization (WHO) definition of anemia: hemoglobin < 13 g/L in men and < 12 g/L in women.

The primary endpoint was major adverse cardiac events (MACE), which included all-cause death, non-fatal myocardial infarction, and nonfatal stroke. The secondary endpoint was cardiovascular events, which included cardiovascular death, non-fatal myocardial infarction, and non-fatal stroke.

Statistical analysis

All the data in this study was analyzed by SPSS 22.0 software. P value less than 0.05 is considered to indicate statistical significance. Shapiro-Wilk method was used for normal distribution test. Continuous variables consistent with normal distribution were represented as (mean ± standard deviation) and t test was used for comparison between the two groups, whereas continuous variables inconsistent with normal distribution were represented as median and quartile [M(Q1-Q3)] and Mann Whitney test was used for comparison between the two groups. Categorical variables were represented as [n(%)], and compared using the chi-square or Fisher's exact test.

Effects of factors on clinical outcomes after PCI were determined using multivariate Cox regression analysis. We used MACE and cardiovascular events as the dependent variable and anemia and hemoglobin as the independent variable. We used 3 models for multivariate analysis. In model 1, the covariable is null. In model 2, risk factors for cardiovascular adverse events, such as age and male, were used as covariates. In model 3, the following additional covariables were added in Model 2: body mass index (BMI), smoker, alanine aminotransferase (ALT), estimated glomerular filtration rate (eGFR), low density lipoprotein cholesterol (LDL), C-reactive protein (CRP).

The ROC was used to observe the optimal threshold of hemoglobin to predict MACE, and the area under the curve (AUC), sensitivity, and specificity were used to evaluate the stability of the prediction model. Overall MACE rate was estimated using Kaplan-Meier survival curves with the log-rank test.

The patient and public involvement statement

None.

Results

Baseline characteristics

204 participants were included in this study. The baseline characteristics are shown in Table 1. Anemia patients

Table 1 Baseline characteristics according to anemia status

	Anemia, Yes	Anemia, No	*P* value
n	54	150	
Age (years)	77.93 ± 9.70	70.67 ± 9.91	< 0.001
Male, n (%)	35 (64.8)	107 (71.3)	0.471
BMI	22.66 [20.23–25.06]	23.73 [21.39–25.92]	0.048
Smoker, n (%)	17 (31.5)	84 (56.0)	0.002
Systolic blood pressure (mmHg)	135[119.75-144.25]	138 [124–148]	0.095
Diastolic blood pressure (mmHg)	75.39 ± 14.58	78.16 ± 12.65	0.187
LVEF (%)	66 [61–68]	66 [63–68]	0.568
OCI, n (%)	12 (22.2)	23 (15.3)	0.347
PAD, n (%)	15 (27.8)	38 (25.3)	0.865
Atrial fibrillation, n (%)	11 (20.4)	15 (10.0)	0.085
Hypertension, n (%)	37 (68.5)	114 (76.0)	0.371
Dyslipidemia, n (%)	16 (29.6)	88 (58.7)	< 0.001
Diabetes mellitus, n (%)	22 (40.7)	51 (34.0)	0.471
Laboratory data			
Hb (g/dL)	11.30 [10.48–12.10]	14.35 [13.6–15.4]	< 0.001
Alb (g/dL)	3.45 [3.10–3.80]	4.15 [3.90–4.40]	< 0.001
eGFR (mL/min/1.73m2)	50 [9.25–64.25]	67 [58-78.25]	< 0.001
AST (U/L)	21.5 [16–26]	23 [19–29]	0.031
ALT (U/L)	14 [8.75–19.25]	20 [15–28]	< 0.001
TC (mg/dL)	168.5 [140.75-185.75]	192 [171-209.5]	< 0.001
TG (mg/dL)	91.5 [62.75–130.5]	126 [86–176]	0.001
HDL (mg/dL)	47.5 [37.75–53.25]	51 [43–58]	0.014
LDL (mg/dL)	90 [81–107]	114 [97–135]	< 0.001
HbA1c (%)	6.0 [5.7–6.75]	6.0 [5.7–6.7]	0.933
CRP (mg/dL)	0.25 [0.08–0.89]	0.09 [0.04–0.24]	< 0.001
Medication			
Aspirin, n (%)	53 (98.1)	149 (99.3)	1.000
Thienopyridine, n (%)	52 (96.3)	148 (98.7)	0.614
Warfari, n (%)	2 (3.7)	3 (2.0)	0.856
DOAC, n (%)	8 (14.8)	13 (8.7)	0.202
Ezetimibe, n (%)	1 (1.9)	2 (1.3)	1.000
PPI, n (%)	35 (64.8)	99 (66.0)	0.875
Statins, n (%)	15 (27.8)	96 (64.0)	< 0.001
ACEI, n (%)	3 (5.6)	16 (10.7)	0.404
ARB, n (%)	27 (50.0)	61 (40.7)	0.235
β blocker, n (%)	16 (29.6)	39 (26.0)	0.606
MRA, n (%)	2 (3.7)	9 (6.0)	0.772
Lesional characteristics			
Multivessel disease, n (%)	16 (29.6)	37 (24.7)	0.476
DES use, n (%)	49 (90.7)	144 (96.0)	0.164
Bifurcation lesions, n (%)	29 (53.7)	73 (48.7)	0.526
LMT lesions, n (%)	6 (11.1)	7 (4.7)	0.096
Ostial lesions, n (%)	9 (16.7)	21 (14.0)	0.635
Calcified lesions, n (%)	13 (24.1)	16 (10.7)	0.016
CTO lesions, n (%)	3 (5.6)	9 (6.0)	1.000

BMI, body mass index; LVEF, left ventricular ejection fraction; OCI, old cerebral infarction; PAD, peripheral artery disease; Hb, hemoglobin; Alb, serum albumin; eGFR, estimated glomerular filtration rate; AST, aspartate transaminase; ALT, alanine transaminase; TC, total cholesterol; TG, triglycerides; HDL, high-density lipoprotein; LDL, low density lipoprotein; HbA1c, hemoglobin A1c; CRP, C-reactive protein; DOAC, direct oral anticoagulants; PPI, proton pump inhibitor; ACEI, angiotensin-converting enzyme inhibitor; ARB, angiotensin-receptor blocker; MRA, mineralocorticoid receptor antagonist; DES, drug eluting stent; LMT, left main trunk; CTO, chronic total occlusion

were older and had lower BMI, hemoglobin (11.30 [10.48–12.10] g/dL vs. 14.35 [13.6–15.4] g/dL, $p < 0.001$), Alb, eGFR, ALT, LDL, CRP than those without anemia.

Multivariate COX regression analysis

In multivariate COX regression analysis, we used MACE and cardiovascular events as the dependent variable and anemia and hemoglobin as the independent variable. We used 3 models for multivariate analysis (Table 2). In model 1, the covariable is null, anemia had significant effects on both primary endpoint (HR = 2.79, 95% CI 1.33 to 5.87, $p = 0.007$) and secondary endpoint (HR = 2.50, 95% CI 1.01 to 6.16, $p = 0.047$); hemoglobin also had significant effects on both primary endpoint (HR = 0.66, 95% CI 0.55 to 0.79, $p < 0.001$) and secondary endpoint (HR = 0.69, 95% CI 0.55 to 0.87, $p = 0.001$). In model 2, age and sex were selected as the covariant and the outcome indicates that hemoglobin has an independent effect on primary endpoint (HR = 0.67, 95% CI 0.54 to 0.82, $p < 0.001$) and secondary endpoint (HR = 0.69, 95% CI 0.53 to 0.89, $p = 0.004$). In model 3, age, sex, BMI, Smoker, ALT, eGFR, LDL, CRP were selected as the covariant, hemoglobin remained an independent factor in the primary endpoint (HR = 0.72, 95% CI 0.56 to 0.93, $p = 0.012$) and secondary endpoint (HR = 0.71, 95% CI 0.52 to 0.96, $p = 0.027$).

The receiver operating characteristic (ROC)

The ROC was used to observe the optimal threshold of hemoglobin to predict MACE and the outcome showed that the best threshold for hemoglobin to predict MACE was 12.25 g/dl, the area under the curve was 0.73, sensitivity was 0.818, and the specificity was 0.571 (Fig. 1). In Kaplan-Meier analysis (Fig. 2), hemoglobin < 12.25 g/dl predicted worse prognosis in MACE ($p < 0.001$).

Discussion

In this study, we found that low preoperative hemoglobin level was significantly associated with MACE in patients with SCAD undergoing PCI, independent of other risk predictors. We studied the effects of anemia and hemoglobin level on MACE and cardiovascular events, and found that without the addition of covariables, anemia had significant effects on both MACE and cardiovascular events. However, after adjusting for the effects of other risk factors, anemia had no significant effect on the prediction of adverse events. But preoperative hemoglobin level showed significant predictive value for endpoint events in all three models that adjusted for different risk factors. Then we used the ROC curve to determine that the optimal threshold for hemoglobin level to predict endpoint events was 12.25 g/dl. Our findings suggest that hemoglobin level is a useful tool for risk stratification in SCAD patients undergoing PCI are of important value for further treatment and post-operative follow-up.

Anemia is a common comorbidity in patients with cardiovascular disease [14], which seriously affects the efficacy of revascularization in patients with heart failure [15], congenital heart disease [16] and coronary artery disease [17]. The incidence of anemia in ACS patients is 15%, while the incidence of anemia in elderly ACS patients is as high as 45% [18]. It has been reported that the incidence of ACS and the mortality rate of ACS have increased due to anemia. Anemia is also an independent risk factor associated with the occurrence of adverse events after PCI [19]. In recent years, with the application of hemoglobin in predicting the incidence and mortality of cardiovascular disease, it has become a common diagnostic indicator. Low baseline hemoglobin level was found to be an independent predictor of the risk of in-hospital bleeding and death at 1 month in ACS patients [20]. Multiple prospective studies [21, 22] have shown that hemoglobin level was associated with 30-day and 1-year mortality, MACE, bleeding events, and ischemic events in PCI patients, and baseline hemoglobin is a strong and independent predictor of adverse outcomes. Poludasu et al. [23] conducted a 3.2 year prospective study and found that baseline hemoglobin was associated with all-cause long-term mortality. One study has shown that hemoglobin level < 120 g/L was associated with an increased incidence of MACE during postoperative follow-up in STEMI patients treated with PCI [24]. Previous

Table 2 Multivariate Cox regression analysis

	Model 1		Model 2		Model 3	
	HR (95%CI)	P	HR(95%CI)	P	HR(95%CI)	P
Primary endpoint						
Anemia	2.79(1.33–5.87)	0.007	2.04(0.93–4.47)	0.076	1.58(0.57–4.37)	0.378
Hb	0.66(0.55–0.79)	< 0.001	0.67(0.54–0.82)	< 0.001	0.72(0.56–0.93)	0.012
Secondary endpoint						
Anemia	2.50(1.01–6.16)	0.047	1.97(0.76–5.13)	0.165	1.76(0.52–5.97)	0.368
Hb	0.69(0.55–0.87)	0.001	0.69(0.53–0.89)	0.004	0.71(0.52–0.96)	0.027

Model 1: no adjusted

Model 2: adjusted for age and sex

Model 3: adjusted for age, sex, BMI, smoker, ALT, eGFR, LDL, and CRP

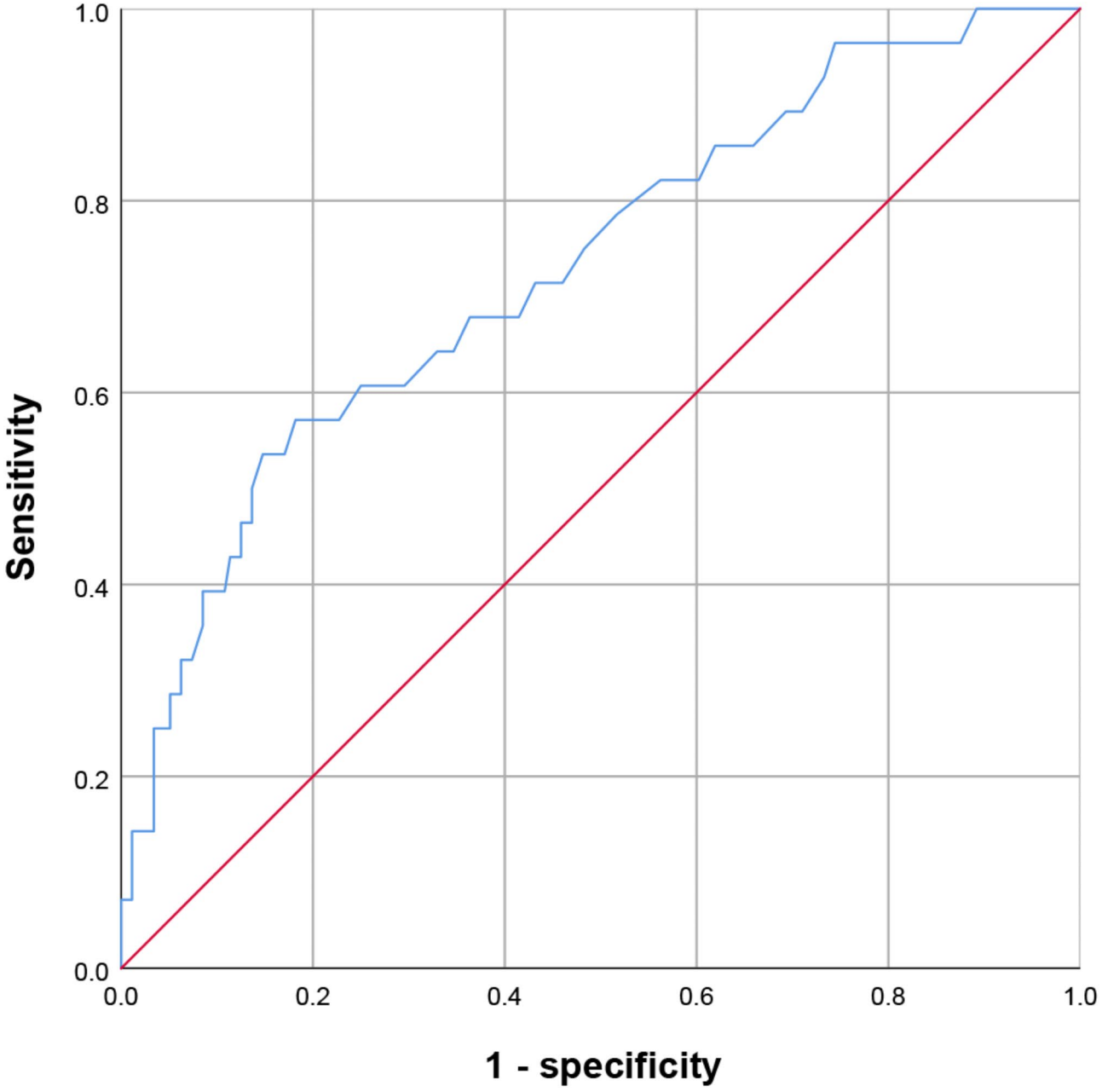

Fig. 1 The receiver operating characteristic (ROC) analyses of Predictive value of hemoglobin for MACE. The area under the curve (AUC) was 0.73 (95% CI 0.63 to 0.83, $p < 0.001$), sensitivity was 0.818, and the specificity was 0.571

studies on the effect of hemoglobin level on the prognosis of patients with coronary heart disease mostly focused on ACS patients, however there were few studies on SCAD patients. In consistent with previous results that anemia and low hemoglobin was an independent predictor of mortality and cardiovascular events in SCAD patients [25], our study also supported this conclusion. About half of the patients in the original study underwent PCI and the difference was significant across hemoglobin groups. Our study focused on SCAD patients after PCI and showed the lesion characteristics, which excluded the surgery-related effect on hemoglobin levels and clinical outcomes.

There are several hypotheses that could explain the association between anemia and poor clinical outcomes in SCAD patients. Low hemoglobin level reduces the amount of oxygen in the blood supplied to the myocardium, where a higher cardiac output is needed to meet myocardial oxygen demand, leading to left ventricular compliance hypertrophy. Hypertrophic myocardium increases cardiac oxygen demand, and CAD patients themselves have insufficient oxygen supply due

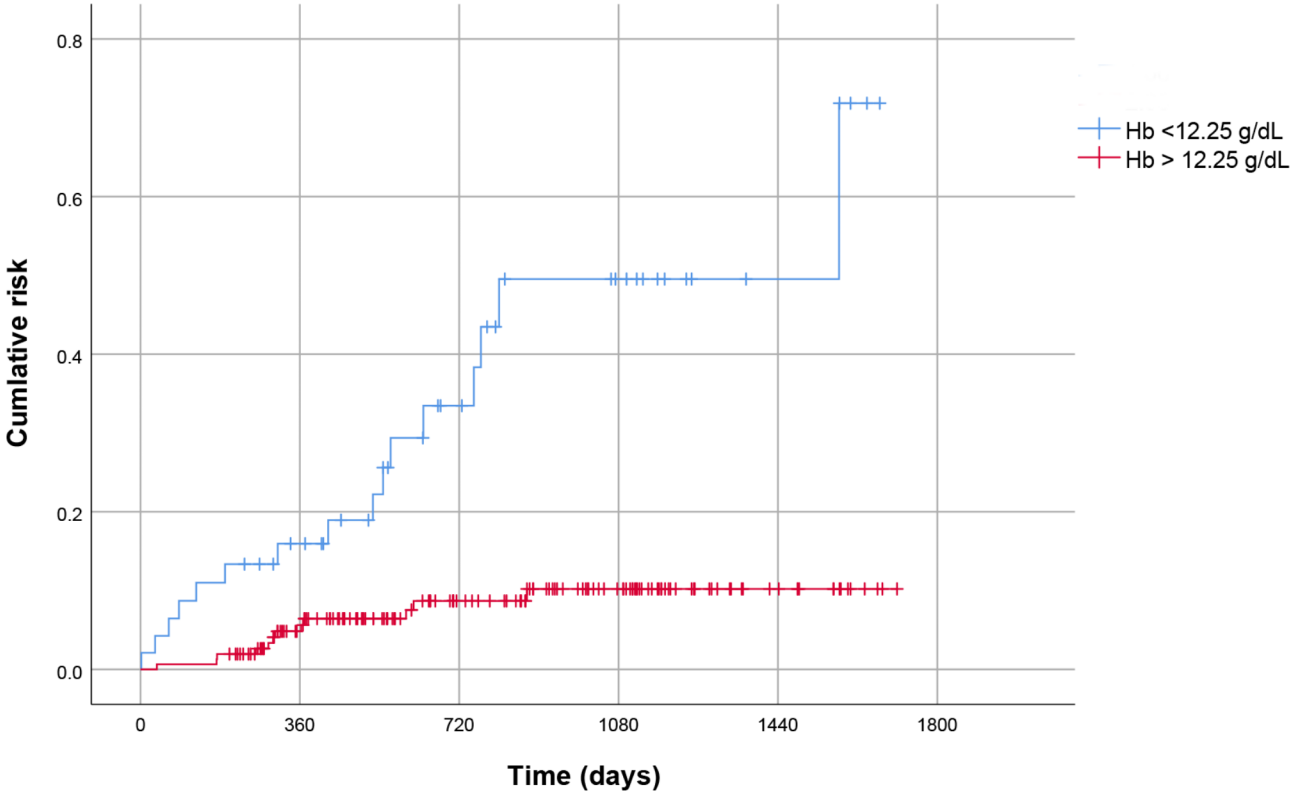

Fig. 2 Kaplan-Meier curve showing the prevalence of major adverse cardiac events (MACE) in patients during a median follow-up of 783 days on the basis of the cutoff values of hemoglobin

to coronary stenosis, which further exacerbates hypoxia [26]. Therefore, this vicious circle between hypoxia and cardiovascular disease can further lead to poor prognosis. There is no definite conclusion on the pathogenesis of hemoglobin and poor prognosis of CAD. However, as a good indicator of anemia, hemoglobin has a certain predictive effect on the prognosis of PCI. In view of the complexity of the pathogenesis of CAD, further basic and clinical trials are needed to confirm whether hemoglobin can be a therapeutic target and its guiding value in the clinical medical process.

There are several limitations to our study. First of all, this study is a single center, retrospective cohort study, which needs to be confirmed by further large-scale studies. Second, only preoperative hemoglobin level was measured, however dynamic changes in hemoglobin levels should also be considered as risk factor for endpoint events. Third, blood volume, erythropoietin, serum iron, and drugs that affect hemoglobin were not collected in this study. These indicators can better explain the cause of hemoglobin decline and increase the mechanisms of poor prognosis risk.

Conclusion

In conclusion, low hemoglobin is an independent predictor of long-term poor prognosis in SCAD patients after PCI. In this population, hemoglobin can serve as a useful tool for risk stratification.

Abbreviations

ACS	Acute coronary syndrome
CAD	Coronary heart disease
SCAD	Stable coronary artery disease
MACE	Major adverse cardiac events
PCI	Percutaneous coronary intervention

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Not applicable.

Author contributions

J G and X Z designed the study; H L and C Y made the analysis; T H and Z W prepared the figures and draft. All authors approved the final version.

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None.

Data availability

The data will be available upon request from the corresponding author.

Declarations

Ethics approval and consent to participate

All patients were enrolled after the approval of the Shinonoi General Hospital Ethics Committee, and after written informed consent was obtained.

Consent for publication
Not applicable.

Competing interests
The authors declare no competing interests.

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