Gensini score values for predicting periprocedural myocardial infarction
An observational study analysis
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Abstract
The Gensini score (GS) is a convenient, powerful tool for assessing the severity and complexity of coronary artery diseases. Our research investigated the relationship between the GS and periprocedural myocardial infarction (PMI).

We recruited 4949 patients (3366 men, 1583 women; mean age 66.45 ± 10.09 years) with a single coronary artery revascularization. Based on the tertile of the GS 20 and 36, the population was divided into 3 groups: Low Group (0 < GS ≤ 20, N = 1809); Intermediate Group (20 < GS ≤ 36, N = 1579); High Group (GS > 36, N = 1561). PMI3 represented the endpoint for cTnI > 3-fold upper reference limit, while PMI5 represented the endpoint for cTnI > 5-fold upper reference limit.

The incidence of PMI of High Group was statistically higher than that of Intermediate Group (P < .05), while that of Intermediate Group was statistically higher than Low Group (P < .05). With the adjustment of some general variables, GS was an independent significantly predictor for PMI3 (β = 0.006, P < .05) and PMI5 (β = 0.007, P < .05). Following receiver operating characteristic curve analysis, the optimal cut-off value to predict PMI are 22.5 for PMI3 and 27 for PMI5.

The GS was an independent predictor of PMI in the single-coronary revascularization population. Additionally, the 22.5 of GS was the optimal cut-off value for determining the presence of PMI3, while the 27 of GS for PMI5.

Abbreviations: ACC = American College of Cardiology, AHA = American Heart Association, CAD = coronary artery diseases, CK-MB = creatine kinase-MB fraction, cTnI = cardiac troponin I, CVD = cardiovascular diseases, GS = Gensini score, PCI = percutaneous coronary intervention, PMI = periprocedural myocardial infarction, ROC = receiver operating characteristic, SCAI = the Society for Cardiovascular Angiography and Interventions, URL = upper reference limit.

Keywords: Gensini score, periprocedural myocardial infarction

1. Introduction
It is well-known that the cardiovascular diseases (CVD) are the leading cause of deaths all over the world. In particular, coronary artery diseases (CAD) are the major cause (43.8%) of deaths attributable to CVD in the United States.[1] Give this, percutaneous coronary intervention (PCI) is becoming the most popular treatment for CAD. While there have been technical advances in the PCI process, there is still a high incident rate of periprocedural myocardial infarction (PMI) rate of approximately 5% to 30% continues to be reported.[2-3] Therefore, it is critical that clinical practitioners discover a unique predictor for the presence of PMI as a significant orientation in the cardiology field.

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The datasets generated during and/or analyzed during the current study are not publicly available, but are available from the corresponding author on reasonable request.
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events that can lead to myocardial necrosis. Nowadays, many factors have been approved to be related with the process of PMI, which could be categorized as patient-related factors, lesion-related factors, and procedure-related factors. In totality, these factors identify patients with increasing atherosclerotic disease burden, increased thrombotic risk, and with neurohormonal activation that predisposes to either macrovascular complications (side branch occlusion or macroembolization) or microvascular obstruction (distal embolization of microparticles), unifying the pathophysiologic basis of myocardial necrosis after PCI.

Above all, our hypothesis is that GS is an independent predictor for the presence of PMI in the broad population and we could determine a special cut-off point of GS for the prediction of PMI, according to the definitions of the Society for Cardiovascular Angiography and Interventions (SCAI) and fourth universal definition of myocardial infarction.

2. Methods and Materials

2.1. Study design and patient population

This was a single-center, retrospective research that took place in the Sir Run Run Shaw Hospital (Hangzhou, China) from December 2007 to April 2019. (1) inpatients with single coronary artery stenosis (left main, left anterior descending, left circumflex, or right coronary artery); (2) inpatients who had a stent implanted at the hospital during the time period. Patients were excluded if they had myocardial infarction, elevated pre-procedural cardiac troponin I (cTnI), creatine kinase-MB fraction (CK-MB), PCI for more than 1 artery, a coronary artery with thrombosis, transluminal extraction-atherectomy therapy for culprit artery, severe heart failure (EF<45% or NT-pro BNP>2000); severe valve diseases.

The primary endpoint was PMI, following the definitions of the SCAI and the Universal Definition of Myocardial Infarction, the Cut-Off value of cTnI for PMI was >3-fold or 5-fold upper reference limit (URL) in addition to symptoms, ECG changes, angiographic findings or new regional wall motion abnormalities 48 hours after the procedure. In our study, PMI3 represented the endpoint for cTnI > 3-fold URL, while PMI5 represented the endpoint for cTnI > 5-fold URL. The URL of cTnI in this study is 0.11 ng/mL.

Cardiac biomarkers and electrocardiograms were systematically assessed for all participants before and after index PCI or staged procedure to identify PMI. cTnI levels were evaluated every 8 hours after the PCI, while 24 to 48 hours dynamic monitoring was carried out after the procedure if necessary.

2.3. Gensini score

GS was initially put forward in 1983, and had a pivotal role in the stratification for determining the severity of CAD. Since 1983, many cardiology centers have proved the validation of this severity score system for CAD. Precisely, the GSs can be calculated using the following steps: (1) define the concentric or eccentric luminal narrowing degrees (stenosis of 1% to 25%, 26% to 50%, 51% to 75%, 76% to 99%, 100% are given a score of 1, 2, 4, 8, 16, 32, respectively); (2) each changeable segment of coronary has a specific coefficient based on its importance for blood supply to the heart; (3) the summation of all changeable segment scores (narrowing score * coefficient) is the final GS.

Coronary angiography was performed in our department following the standard procedure. All participants were
prescribed 100 mg/d aspirin and 75 mg/d clopidogrel maintenance for more than 7 days before coronary angiography. Two invasive cardiologists who were blinded to the final results performed the coronary angiographies. Three independent cardiologists then calculated final scores for the statistical analysis, following the principles of GS.

2.4. Statistical analysis

All data were collected and analyzed using Statistical Package for the Social Science for Mac OS, version 23 (SPSS inc., Chicago, IL). Categorical variables were presented as percentage and assessed with the use of chi-square or Fisher’s exact test. Continuous variables were presented as mean ± standard deviation and assessed with the use of Student’s t test or analysis of variance. When comparing differences between groups, the least significant difference post hoc test was selected. Univariate and multivariate logistic regression were then performed to explore the relationship between GS and PMI. The optimal cut-off point of GS was measured via receiver operating characteristic (ROC) curve analysis. A P-value of <.05 was considered statistically significant.

3. Results

We recruited 4949 patients with CAD (3366 men, 1583 women; mean age 66.45 ± 10.09 years). Based on the tertile of the GS, the population was divided into 3 groups: Low Group (0 < GS ≤ 20, N = 1809); Intermediate Group (20 < GS ≤ 36, N = 1579); High Group (GS > 36, N = 1561). General information is presented in Table 1.

3.1. Incidence of PMI

The incidence of PMI3 and PMI5 was calculated individually. A direct comparison between different groups is demonstrated in Figure 2. For both PMI3 and PMI5, PMI incidence was significant.

### Table 1

Demographic data of different groups.

| Categories | Variables | Low group (N = 1809) | Intermediate group (N = 1579) | High group (N = 1561) | P (low vs inter) | P (low vs high) | P (inter vs high) |
|------------|-----------|----------------------|--------------------------------|-----------------------|----------------|----------------|-----------------|
| General information | Gender, male% | 64 | 67 | 74 | .06 | .26 | .19 |
| | Age, yr | 65.61 ± 10.94 | 66.73 ± 9.84 | 67.14 ± 10.42 | 0.06 | .26 | .19 |
| | BMI, kg/m² | 23.93 ± 14.56 | 23.44 ± 7.03 | 23.71 ± 5.61 | .58 | .33 | .43 |
| | SYB, mmHg | 133.94 ± 39.73 | 133.39 ± 23.07 | 135.19 ± 39.92 | .43 | .85 | .22 |
| | DBP, mmHg | 74.58 ± 14.31 | 74.20 ± 20.98 | 73.59 ± 13.31 | .51 | .12 | .08 |
| | UAP, yes% | 52 | 54 | 51 | .06 | .22 | .34 |
| Medical history | Hypon, yes% | 65 | 65 | 68 | .23 |
| | DM, yes% | 19 | 23 | 28 |
| | Smoking, yes% | 40 | 42 | 44 | .05 | .16 | .32 |
| | Drinking, yes% | 33 | 31 | 31 | .23 | .17 | .12 |
| | F-CVD, yes% | 9 | 9 | 10 | .28 | .80 | .13 |
| | Biochemistry results | TC, mmol/L | 4.18 ± 1.08 | 4.26 ± 1.16 | 4.27 ± 1.24 | .09 | .46 | .87 |
| | HDL-C, mmol/L | 1.09 ± 0.30 | 1.08 ± 0.28 | 1.03 ± 0.28 | .30 |
| | LDL-C, mmol/L | 2.17 ± 0.62 | 2.28 ± 0.88 | 2.30 ± 0.93 | .45 |
| | VLDL-C, mmol/L | 0.92 ± 1.09 | 0.90 ± 1.19 | 0.99 ± 1.29 | .10 | .61 | .10 |
| | TG, mmol/L | 1.67 ± 1.18 | 1.68 ± 1.09 | 1.73 ± 1.34 | .39 | .94 | .21 |
| | LP, mg/dL | 20.47 ± 22.19 | 23.9 ± 25.85 | 25.21 ± 25.68 | .16 |
| | TB, Imol/L | 13.83 ± 6.78 | 13.42 ± 6.22 | 13.24 ± 6.25 | .40 |
| | UB, Imol/L | 9.99 ± 5.11 | 9.73 ± 4.80 | 9.53 ± 4.83 | .08 |
| | CB, Imol/L | 3.84 ± 2.62 | 3.69 ± 1.95 | 3.72 ± 2.12 | .09 | .09 | .77 |
| | UA, Imol/L | 348.45 ± 105.58 | 349.86 ± 105.61 | 365.16 ± 107.07 | .45 |
| | Cr, Imol/L | 78.92 ± 48.23 | 82.37 ± 65.00 | 86.07 ± 56.77 | .07 |
| | BUN, mmol/L | 5.17 ± 2.17 | 5.35 ± 2.22 | 5.47 ± 2.25 | .12 |
| | eGFR, mL/min | 48.46 ± 10.64 | 48.37 ± 10.64 | 48.37 ± 10.64 | .06 |
| | Blood routine examinations | WBC, ×10⁹ | 26.74 ± 9.98 | 26.11 ± 7.84 | 25.12 ± 7.80 | .86 |
| | Lymphocyte, % | 26.74 ± 9.98 | 26.11 ± 7.84 | 25.12 ± 7.80 | .86 |
| | Neutrophile, % | 66.21 ± 10.04 | 62.87 ± 9.89 | 64.02 ± 9.27 | .06 |
| | Platelet, ×10⁹ | 719.74 ± 53.81 | 719.22 ± 58.43 | 718.22 ± 58.59 | .61 | .85 | .43 |
| | MPV, fL | 9.19 ± 1.41 | 9.25 ± 1.45 | 9.12 ± 1.37 | .05 | .24 | .19 |
| | L: W, pre-CKMB, IU | 14.79 ± 10.57 | 15.59 ± 9.11 | 15.61 ± 9.17 | .06 |
| | FBG, mg/L | 6.30 ± 2.19 | 6.41 ± 2.36 | 6.66 ± 2.59 | .19 |
| Medicine | anti-Hyper Med, yes% | 77 | 79 | 84 | .19 |
| | Statins, yes% | 97 | 98 | 98 | .42 | .35 | .21 |
| | anti-Plt Med, yes% | 100 | 100 | 100 | .26 | .50 | .31 |
| Procedure factors | FFR, NUS, OCT, yes% | 12 | 11 | 7 | .29 |
| | ACC/AHA TypeB2C, yes% | 28 | 34 | 38 |
| | Left coronary artery, yes% | 77 | 79 | 66 |
| | Total length of stents, mm | 28.12 ± 15.07 | 36.75 ± 21.78 | 47.13 ± 24.09 | .45 |
| | Diameter of stent =2.5 mm | 91 | 91 | 91 | .87 | .87 | .60 |
| | PO without dilation, yes% | 85 | 87 | 87 | .09 |

P-value: <.05 means significant statistically.

Ant: anti-hypertension medicine, anti-Plt Med = anti-platelet medicine, BMI = body mass index, BUN = blood urea nitrogen, CB = conjugated bilirubin, CK-MB = creatine kinase MB, Cr = creatinine, DBP = diastolic blood pressure, DM = diabetes mellitus, eGFR = estimated glomerular filtration rate, FBG = fibrinogen, F-CVD = family history of cerebral-or-cardiovascular diseases, FFR = fractional flow reserve, HDL-C = high density lipoprotein cholesterol, Hyper = hypertension, MUS = intravascular ultrasound, LDL-C = low density lipid cholesterol, LPa = lipid protein alpha, MPV = mean platelet volume, OCT = optical coherence tomography, Plt = platelet, SYB = systolic blood pressure, TB = total bilirubin, TC = total cholesterol, TG = triglyceride, UA = uric acid, UAP = unstable angina, Unconjugated bilirubin, VLDL-C = very low density lipoprotein cholesterol, WBC = white blood cells.
3.2. Regression analysis between variables and PMI

All the demographic variables and GS were analyzed by univariate logistic regression for PMI3 (see Table 2). Given that the P-value of some variables (Gender, Age, Smoking, Hyper, DM, Drinking, F-CVD, HDL-C, Lpa, TB, UB, Cr, BUN, eGFR, WBC, Lymphocyte, Neutrophil, pre-CMB, anti-Hyper Meds, anti-Plt Meds, ACC/AHA type B2C, Left Coronary artery, Total Length of Stents, and GS) was <.10, these attributes were then tested with multivariate logistic regression for PMI3 (Table 2; Odds Ratio in the Fig. 3A). Following the adjustment of these variables, GS remained a statistically significant predictor for PMI3 ($β = 0.006, P < .05$). Moreover, Age, HDL-C, WBC, pre-CMB, Left Coronary artery, and Total Length of Stents were also independent predictors for the prediction of PMI3.

Additionally, all the demographic variables and GS were analyzed using univariate logistic regression for PMI5 (see Table 3). Given that the P-value of some variables (Gender,
3.2. Regression analysis between variables and PMI

All the demographic variables and GS were analyzed by univariate logistic regression for PMI3 (see Table 2). Given that the \( P \)-value of some variables (Gender, Age, Smoking, Hyper, DM, Drinking, F-CVD, HDL-C, LPa, TB, UB, Cr, BUN, eGFR, WBC, Lymphocyte, Neutrophile, ACC/AHA type B2C, Left Coronary artery, Total Length of Stents, and GS) was <.10, these attributes were then tested with multivariate logistic regression for PMI3 (Table 2; Odds Ratio in the Fig. 3A). Following the adjustment of these variables, GS remained a statistically significant predictor for PMI3 (\( \beta = 0.006, P < .05 \)). Moreover, Age, HDL-C, WBC, pre-CKMB, Left Coronary artery, and Total Length of Stents were also independent predictors for the prediction of PMI3.

Additionally, all the demographic variables and GS were analyzed using univariate logistic regression for PMI5 (see Table 3). Given that the \( P \)-value of some variables (Gender, Smoking, Hyper, DM, Drinking, F-CVD, HDL-C, LPa, TB, UB, Cr, BUN, eGFR, WBC, Lymphocyte, Neutrophile, ACC/AHA type B2C, Left Coronary artery, Total Length of Stents, and GS) was <.10, these attributes were then tested with multivariate logistic regression for PMI5 (Table 3; Odds Ratio in the Fig. 3B). Following the adjustment of these variables, GS remained a statistically significant predictor for PMI5 (\( \beta = 0.008, P < .05 \)). Moreover, Age, HDL-C, WBC, pre-CKMB, Left Coronary artery, and Total Length of Stents were also independent predictors for the prediction of PMI5.

Figure 3. The forest plot of PMI3 (A) and PMI5 (B) for multivariate logistic regression.

Table 3

| Categories            | Variables          | Univariate regression | Multivariate regression |
|-----------------------|--------------------|-----------------------|-------------------------|
|                       |                    | B        | P  | B   | P  |
| **General information** | Gender, male%      | –0.197  | .07 | –0.216 | .07 |
|                       | Age, yr            | 0.024   |     | 0.015 |     |
|                       | BMI, kg/m²         | –0.009  | .48 | –     | –   |
|                       | SBP, mmHg          | 0.000   | .46 | –     | –   |
|                       | DBP, mmHg          | 0.001   | .85 | –     | –   |
|                       | UAP, yes%           | 0.108   | .21 | –     | –   |
| **Medical history**   | Hyper, yes%        | 0.300   | .77 | 0.034 | .77 |
|                       | DM, yes%           | 0.250   | .95 | 0.007 | .95 |
|                       | Smoking, yes%      | –0.251  | .13 | –0.184 | .13 |
|                       | Drinking, yes%     | –0.246  | .96 | –0.005 | .96 |
|                       | F-CVD, yes%        | –0.240  | .13 | –     | –   |
| **Biochemistry results** | TC, mmol/L         | –0.050  | .84 | –     | –   |
|                       | HDL-C, mmol/L      | –0.577  | .565 | –     | –   |
|                       | LDL-C, mmol/L      | 0.004   | .93 | –     | –   |
|                       | VLDL-C, mmol/L     | –0.039  | .32 | –     | –   |
|                       | TG, mmol/L         | –0.060  | .14 | –     | –   |
|                       | LPa, mg/dL         | 0.002   | .04 | –     | –   |
|                       | TB, µmol/L         | –0.014  | .24 | –     | –   |
|                       | UB, µmol/L         | –0.024  | .14 | –0.041 | .14 |
|                       | CB, µmol/L         | 0.000   | .99 | –     | –   |
|                       | UA, µmol/L         | 0.000   | .65 | –     | –   |
|                       | Cr, µmol/L         | 0.001   | .29 | –0.001 | .29 |
|                       | BUN, mmol/L        | 0.079   | .054 | –     | –   |
|                       | eGFR, mL/min       | –0.013  | .20 | –0.005 | .20 |
| **Blood routine examinations** | WBC, ×10⁹          | 0.100   | .059 | –     | –   |
|                       | Lymphocyte, %      | –0.024  | .15 | –0.011 | .15 |
|                       | Neutrophile, %     | 0.017   | .94 | 0     | .94 |
|                       | Plt, ×10³          | –0.001  | .52 | –     | –   |
|                       | MPV, fL            | 0.025   | .40 | –     | –   |
|                       | CK-MB, IU          | 0.029   | .031 | –     | –   |
|                       | FBG, mg/L          | 0.027   | .11 | –     | –   |
| **Medicine**          | anti-Hyper Med, yes% | 0.251   | .99 | 0.002 | .99 |
|                       | Statins, yes%      | –0.165  | .53 | –     | –   |
|                       | anti-Plt Med, yes% | –0.692  | .10 | –0.494 | .10 |
| **Procedure factors** | FFR, IVUS, OCT, yes% | 0.100   | .49 | –     | –   |
|                       | ACC/AHA Type B2C, yes% | 0.139   | .12 | –     | –   |
|                       | Left coronary artery, yes% | 0.327   | .740 | –     | –   |
|                       | Total length of stents, mm | 0.024   | .024 | –     | –   |
|                       | Diameter of stent=2.5 mm | –0.096  | .51 | –     | –   |
|                       | PCI without dilation, yes% | 0.124   | .36 | –     | –   |
|                       | Gensini score      | 0.014   | .007 | –     | –   |

Abbreviations were the same as Table 1.
P-value: <.05 means significantly statistically, attributes with \( P < .1 \) in univariate results would be selected in multivariate regression.
Age, Hyper, DM, Smoking, Drinking, HDL-C, TB, UB, Cr, BUN, eGFR, WBC, Lymphocyte, Neutrophil, pre-CKMB, anti-Hyper Meds, anti-Plt Meds, Left Coronary artery, Total Length of Stents, and GS) was <.10, these attributes were then tested with multivariate logistic regression (see Table 3; Odds Ratio in the Fig. 3B). Following the adjustment of these variables, GS was still an independent significantly predictor for PMI3 (β = 0.007, P < .05). Moreover, Age, HDL-C, BUN, WBC, pre-CKMB, Left Coronary artery, and Total Length of Stents were also independent predictors for the prediction of PMI5.

Furthermore, ROC analysis was used to determine the absolute value of GS for predicting ROC analysis for PMI3 (Fig. 4A) and PMI5 (Fig. 4B). The Youden index was utilized to select the optimal cut-off value for the GS to predict PMI3 (GS = 22.5) and PMI5 (GS = 27) (see Table 4).

4. Discussion
The GS was an independent predictor of PMI following the definitions from the SCAI[13] and the Universal Definition of Myocardial Infarction.[14] Additionally, the 22.5 of GS was the optimal cut-off value for determining the presence of PMI3, while the 27 of GS for PMI5.

Firstly, certain demographic variables were correlated with the presence of PMI in both our study and in previous researches. In this study, regression results indicate that Age, HDL-C, BUN, WBC, pre-CKMB, Left Coronary artery, and Total Length of Stents were independent indicators for predicting PMI. Since 2005, some categories (patient-related risk factors, lesion-related risk factors, procedure-related risk factors) have been known to be related to the presence of PMI.[12] HDL-C was also proved in another article for predicting PMI[17] as well as VLDL-C and LPa in diabetic patients,[18] but not in our study. About inflammation with PMI, a higher Neutrophil-to-Lymphocyte ratio increases the risk of PMI from an original article,[15] also WBC may show a power for predicting in consistent with our results. Besides procedural-related risk factors in 2005, Left Coronary artery, and Total Length of Stents were also showed their predicting power for PMI in an Korean research[20] and in our results. However, other variables, such as CRP,[21] LDL-C,[21] and TCs,[21] weren’t found to be related with PMI in our study.

Secondly, GS was a significant indicator for PMI presence. Many different scores calculated the severity of CAD in coronary angiography, such as SYNTAX score,[16] ACC/AHA score,[17] LEAMAN score,[18] and GS. In a 2013 study, [20] Kenneth initially defined the relationship between SYNTAX score and PMI, and concluded that the SYNTAX score was able to stratify PMI risk. Like SYNTAX score, GS was another convenient and powerful severity score for determining CAD. Many authors have reported its function in various cardiological fields. For example, Sayin has reported that the value of GS represented the degree of CAD correlated with Framingham risk score in the Turkey population.[21] Additionally, Zhenhong[22] group reported that GS was an effective parameter for predicting long-term mortality in acute coronary syndrome patients. In another 2016 study on multivessel therapy and the risk of PMI, Zhangwe[23] also demonstrated the relationship between GS and PMI in the results, although specific details weren’t shown in that article. In our study, the relationship between GS and PMI was again proven, while the power of GS was determined by ROC analysis, with an optimal cut-off value of 22.5 for PMI3 and 27 for PMI5. The preprocedural assessment of the GS could help identify patients with a high risk of PMI, based on the optimal values showing above.

Additionally, various definitions have been put forward in the past few decades. The fourth universal definition of myocardial infarction had been established by the European Society of Cardiology, the American College of Cardiology, the American Heart Association and the World Health Organization in 2019.[14] In these guidelines, PCI-related myocardial infarction was defined as a cTnl > 5-fold URL associated with an assisted test result change within 48 hours. While this definition was the latest, not many authors have supported the validation of the 5-fold cut-off value. Given this, our study extended the results of universal definition as 3-fold and 5-fold to explore the potential relationship between GS and PMI.

For the management strategies for PMI, some guidelines provided 2 aspects to deal with the patients, including prevention and management. About the prevention, almost all strategies could be divided into 4 groups, such as Antiplatelet therapy (aspirin, adequate clopidogrel preloading, glycoprotein IIb/IIIa antagonists if ACS or complicated PCI),[24] Statin therapy initiated before PCI, Embolic protection in saphenous venous graft intervention, and Ischemic preconditioning. About the management, we must take an intensive secondary prevention, such as LDL goal <70 mg/dL (similar to spontaneous myocardial infarction).[25] Moreover, Post-PCI angina, serum biomarkers elevating (CK-MB, cTnl, etc) associated with ischemic ECG changes may dictate further interventional procedures depending on the amount of myocardium at risk.[15] Generally speaking, we should evaluate the risk of myocardial infarction along with the undergoing process, before PCI, during PCI, and after PCI, earlier diagnosis means the safer treatment for the patients.

Table 4
Optimal cut-off value of Gensini score for PMI.

| Variables | Optimal cut-off | Sensitivity (%) | Specificity (%) |
|-----------|----------------|----------------|----------------|
| PMI3      | 22.5           | 74.07          | 46.19          |
| PMI5      | 27             | 64.66          | 55.77          |

Figure 4. Receiver operating characteristic curve for GS in predicting PMI3 (A) and PMI5 (B).
5. Limitations
This research has several limitations. Firstly, this was a single-center, retrospective study with patients who were admitted for CAD which excluded healthy people. Secondly, preprocedural related factors weren’t included in the statistical analysis due to loss of the operation record in the system upgrading process. Thirdly, in addition to PMI, major adverse cardiac events might be considered, such as all-cause death, fatal or nonfatal myocardial infarction, repeat PCI or bypass surgery in a long-time period. Finally, subgroup analysis could be also analyzed in the future studies, such as DM or smoking status.

6. Conclusions
The GS was an independent predictor of PMI in the single-c coronary revascularization population. Additionally, the 22.5 of GS was the optimal cut-off value for determining the presence of PMI3, while the 27 of GS for PMI5.

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References
[1] Benjamin EJ, Virani SS, Callaway CW, et al. Heart disease and stroke statistics – 2018 update: a report from the American Heart Association. Circulation. 2018;137:e67–492.
[2] Herrmann J. Peri-procedural myocardial injury: 2005 update. Eur Heart J. 2005;26:2493–519.
[3] Testa L, Van Gaal WJ, Biondi Zoccai GGL, et al. Myocardial infarction after percutaneous coronary intervention: a meta-analysis of troponin elevation applying the universal definition. QJM. 2009;102:369–78.
[4] Gensini GG. A more meaningful scoring system for determining the severity of coronary heart disease. Am J Cardiol. 1983;51:606.
[5] Sianos G, Morel M, Kappetein AP, et al. The SYNTAX score: an angiographic tool grading the complexity of CAD. EuroIntervention. 2005;1:219–27.
[6] Klein LW, Krone RJ. 44 I Cardiac Interventions Today I. 2008.
[7] Leaman DM, Brower RW, Meester GT, et al. Coronary artery atherosclerosis: severity of the disease, severity of angina pectoris and compromised left ventricular function. Circulation. 2011;63:283–99.
[8] Versteylen MO, Joosen IA, Shaw LJ, et al. Comparison of framingham, PROCAM SCORE, and diamond forrester to predict coronary atherosclerosis and cardiovascular events. J Nucl Cardiol. 2011;18:904–11.
[9] Chen ZW, Yang HB, Chen YH, et al. Impact of multi-vessel therapy to the risk of periprocedural myocardial injury after elective coronary intervention: exploratory study. BMC Cardiovasc Disord. 2017;17:1–7.
[10] Lansky AJ, Stone GW. Periprocedural myocardial infarction prevalence, prognosis, and prevention. Circ Cardiovasc Interv. 2010;3:602–10.
[11] Mizuno O, Hojo Y, Ikeda U, et al. Assessment of coagulation and platelet activation in coronary sinus blood induced by transcatheter coronary intervention for narrowing of the left anterior descending coronary artery. Am J Cardiol. 2000;85:154–60.
[12] Bondeman D, Teml A, Jakowitsch J, et al. Coronary no-reflow is caused by shedding of active tissue factor from dissected atherosclerotic plaque. Blood. 2002;99:2794–800.
[13] Moussa ID, Klein LW, Shah B, et al. Consideration of a new definition of clinically relevant myocardial infarction after coronary revascularization. J Am Coll Cardiol. 2013;62:1563–70.
[14] Thysken K, Alpert JS, Jaffe AS, et al. Fourth universal definition of myocardial infarction. Russ J Cardiol. 2018;24:107–38.
[15] Levine GN, Bates ER, Blankenship JC, et al. 2011 ACCF/AHA/SCAI guideline for percutaneous coronary intervention. J Am Coll Cardiol. 2011;58:e44–122.
[16] Sousa-Uva M, Ahlsson A, Alfonso F, et al. 2018 ESC/EACTS guidelines on myocardial revascularization The Task Force on myocardial revascularization of the European Society of Cardiology (ESC) and European Association for Cardio-Thoracic Surgery (EACTS) developed with the special contribution of the European Association of Percutaneous Cardiovascular Interventions (EAPCI). Eur Heart J. 2018;1–96.
[17] Sattler KJE, Herrmann J, Yun E, et al. High-high-density lipoprotein-cholesterol reduces risk and extent of percutaneous coronary inter- vention-related myocardial infarction and improves long-term outcome in patients undergoing elective percutaneous coronary intervention. Eur Heart J. 2009;30:1894–902.
[18] Zeng RX, Li S, Zhang MZ, et al. Remnant cholesterol predicts periproce- dural myocardial injury following percutaneous coronary intervention in poorly-controlled type 2 diabetes. J Cardiol. 2017;70:113–20.
[19] Verdoia M, Barbieri L, Di Giovino G, et al. Neutrophil to lymphocyte ratio and the extent of coronary artery disease: results from a large cohort study. Angiology. 2016;67:75–82.
[20] Tandjung K, Lam MK, Sen H, et al. Value of the SYNTAX score for periprocedural myocardial infarction according to WHO and the third universal definition of myocardial infarction: insights from the TWENTE trial. EuroIntervention. 2016;12:431–40.
[21] Yao M. Predictive value of baseline C-reactive protein for periproce- dural myocardial infarction of higher risk stratifications: a retrospec- tive cohort clinical study. Anatol J Cardiol. 2018;31:10–7.
[22] Sayin MR, Cetiner MA, Karabag T, et al. Framingham risk score and severity of coronary artery disease. Herz. 2014;39:638–43.
[23] Fu Z, Xue H, Dong W, et al. Correlation between comprehensive evalua- tion of coronary artery lesion severity and long-term clinical outcomes in chinese octogenarians with acute coronary syndrome. Hear Lung Circ. 2014;23:1125–31.
[24] Steinhubl SR, Berger PB, Brennan DM, Topol EJ. Optimal timing for the initiation of pre-treatment with 300 mg clopidogrel before percuta- neous coronary intervention. J Am Coll Cardiol. 2006;47:939–43.
[25] Briguglio C, Visconti G, Focaccia A, et al. Novel approaches for prevent- ing or limiting events (Naples) II trial impact of a single high loading dose of atorvastatin on periprocedural myocardial infarction. J Am Coll Cardiol. 2009;54:2157–63.