Predicting coronary no-reflow in patients with acute ST-segment elevation myocardial infarction using Bayesian approaches

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**Objective** The no-reflow phenomenon is associated with a worse prognosis at follow-up for ST-segment elevation myocardial infarction (STEMI) patients with a primary percutaneous coronary intervention. To date, there is no effective method to predict no-reflow. The aim of this study was to establish a predictive system to evaluate the risk of no-reflow by integrating multiple types of information using Bayesian methods.

**Patients and methods** STEMI patients undergoing primary percutaneous coronary intervention within 12 h from the symptom onset between January 2008 and May 2013 were initially screened from the registry database of Anzhen Hospital (Beijing, China). Baseline clinical data, laboratory studies, and procedural characteristics were recorded. The Bayesian Model and Ten-Factor Model were used and compared with the Single-Factor Models. A receiver operating characteristic curve was used to show the efficacy by presenting both sensitivity and specificity for different cutoff points.

**Results** A total of 1059 consecutive STEMI patients were enrolled. Seventy-nine factors were collected to assess the confidence of the no-reflow phenomenon. The combined likelihood ratios were used to measure the reliability of the no-reflow phenomenon. The area under the curve (AUC) was 0.85 and 0.79 for the Bayesian Model and Ten-Factors Model, respectively, whereas the Single-Factor Model yielded a maximum AUC of 0.67.

**Conclusion** The Bayesian Model showed high sensitivity and good specificity in predicting true relations between multiple factors and the no-reflow outcome. *Coron Artery Dis* 25:582–588 © 2014 Wolters Kluwer Health | Lippincott Williams & Wilkins.

**Keywords:** Bayesian approaches, no-reflow, percutaneous coronary intervention, ST-segment elevation myocardial infarction

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**Introduction**

In patients with acute ST-segment elevation myocardial infarction (STEMI), timely reperfusion with a primary percutaneous coronary intervention (PPCI) is now the preferred strategy [1]. However, this fails to restore optimal myocardial reperfusion in a sizeable proportion of patients, up to 40%, mostly because of the no-reflow phenomenon [2]. No-reflow reflects microvascular obstruction in the presence of a patent epicardial coronary artery and might result from distal embolization of particulate matter as well as in-situ microvascular damage because of ischemia–reperfusion injury [3]. Studies have found that patients who developed the no-reflow phenomenon after PPCI had higher rates of major adverse clinical outcomes, including in-hospital mortality, reinfarction, cardiogenic shock, and heart failure, compared with those who did not develop no-reflow [4].

To date, no pharmacological or procedural intervention has been proven to reverse no-reflow; thus, prevention is vital. It is desirable to find a simple tool for evaluating the patient risk for the development of no-reflow. Wang et al. [5] retrospectively identified predictors of coronary no-reflow using conventional statistical methods. Only baseline clinical and laboratory characteristics were considered in his risk score whereas procedural variables were excluded. Several other studies were carried out focusing on few factors [6–8].

To avoid the limitations of the previous research, we established a predictive system of no-reflow after PPCI by integrating baseline clinical, laboratory, and procedural characteristics using Bayesian methods. This may enable patients at increased risk of no-reflow to be treated with the most appropriate individualized treatment as early as possible.
Methods

Patient selection
Documents of a total of 1059 consecutive STEMI patients undergoing PPCI within 12 h from the symptom onset between January 2008 and May 2013 were initially screened from the registry database of Anzhen Hospital (Beijing, China). STEMI was defined as typical chest pain for more than 30 min with ST elevation of more than 0.1 mV in at least two consecutive leads on the ECG or new-onset left bundle branch block. Patients who presented with non-STEMI or had undergone a coronary artery bypass surgery were excluded. Time-to-hospital admission was defined as the time between the onset of symptoms and hospital admission. Patients who presented with non-STEMI or had undergone a coronary artery bypass surgery were excluded. Time-to-hospital admission was defined as the time between the onset of symptoms and hospital admission.

Characteristics collection
Unfractionated heparin was administered intravenously as a 50–70 IU/kg bolus with subsequent boluses to achieve an activated clotting time of 200–500 s. Clinical and laboratory tests were assessed routinely at the time of admission to this hospital. The severity of heart failure was assessed according to the Killip classification. Coronary angiography was performed according to the standard criteria. The blood flow in the infarct-related artery (IRA) was graded according to the Thrombolysis in Myocardial Infarction (TIMI) grading system [9]. No-reflow was diagnosed as a post-PPCI TIMI flow grade of 2 or less in the IRA with no evidence of flow-limiting residual stenosis (<50%), dissection, spasm, or apparent thrombus. Normal reflow was defined as TIMI flow grade 3. Each of these six angiographic morphologic features indicated ‘high-burden thrombus formation’: (a) cutoff pattern of occlusion in the IRA; (b) accumulated thrombus (>5 mm) proximal to the occlusion; (c) presence of floating thrombus; (d) persistent dye stasis distal to the obstruction; (e) reference lumen diameter of the IRA of at least 4 mm; and (6) incomplete obstruction with the presence of accumulated thrombus more than three times the reference lumen diameter of the IRA. Offline analysis of angiograms was carried out using a viewing system (TOSHIBA DICOM Viewer 1.0.0.1; TAMS Inc., Tustin, California, USA).

Variables including demographics, medical history, laboratory studies, medications, and procedural characteristics were recorded by two independent researchers who were blinded to the study objectives. Individual management decisions such as PPCI strategy, type of stent, or other concomitant medications during hospitalization were made exclusively by their responsible interventional cardiologists and/or physicians on the basis of clinical and angiographic features. These data were collected and entered into a computerized database. The study protocol was approved by the Ethics Committee of Anzhen Hospital (Beijing, China).

Statistical analysis
The Bayesian approach is a probability-based derivation method, which is suitable for combining factors from multiple heterogeneous features, especially robust on incomplete and uncertain data [10].

First, we stratified each factor into different confidence bins and then used likelihood ratios (LRs) to measure the reliability of these bins to increase the sensitivity and specificity of the Bayesian system. TIMI flow grade was treated as the golden positive and negative standard. Here, we define patients with no-reflow as ‘positive’ and those with normal reflow as ‘negative’.

According to Bayesian rules [10], the posterior odds \( O_{post} \) of morbidity can be calculated as the product of the prior odds \( O_{prior} \) and the LR(f); the LR(f) can be calculated by Eq. (1).

\[
O_{post} = P(\text{positive} | f) / P(\text{negative} | f) = O_{prior} \times \text{LR}(f),
\]

where \( P(\text{positive}|f) \) is the probability of no-reflow after considering the factor \( f \), whereas \( P(\text{negative}|f) \) is the possibility of normal reflow after considering the factor \( f \). The prior odds are the ratio of the probability of detecting no-reflow from all patients that can be estimated by the TIMI flow grade [Eq. (2)].

\[
O_{prior} = P(\text{positive})/(1 - P(\text{positive})).
\]

The LR of factor \( f \) is the ratio of the probability of meeting factor \( f \) in the no-reflow patients and the normal reflow patients. From Eqs. (1) and (2), the LR can be calculated as:

\[
\text{LR}(f) = P(f | \text{positive}) / P(f | \text{negative}) = \frac{TP_f}{FP_f / N},
\]

where \( P \) and \( N \) are the number of all the no-reflow and normal reflow patients, respectively, and \( TP_f \) and \( FP_f \) are the number of no-reflow and normal reflow patients with the factor \( f \), respectively.

The advantages of Bayesian rules in this system enable us to integrate multiple heterogeneous data sources into a probabilistic model. Therefore, we can obtain the composite LR \( \text{LR}_{\text{comp}} \) by simply multiplying the LRs from individual sources, which is namely the naive Bayesian network [Eq. (4)].

\[
\text{LR}(f_1 \ldots f_n) = \prod_{i=1}^{n} \text{LR}(f_i) = \prod_{i=1}^{n} \frac{P(f_i | \text{positive}) / P(f_i | \text{negative})}{\prod_{i=1}^{n} \text{LR}(f_i)}.
\]

According to the Bayesian rules described above, the assessment procedure assigned the possible factors’ LR values to measure their ability to predict the outcome.
Then, the naïve Bayesian network is used to integrate these LRs from multiple types of data sources to generate \( LR_{\text{comp}} \) for confidence assessment.

A receiver operating characteristic (ROC) curve can show the efficacy of one test by presenting both sensitivity and specificity for different cutoff points [11]. Sensitivity and specificity can measure the ability of a test to identify true positives (TP) and false positives (FP) in a data set. These two features can be calculated as sensitivity \( = TP/T \) and specificity \( = 1 - (FP/F) \), where TP and FP are the number of identified TP and FP, respectively, whereas \( T \) and \( F \) are the total number of positives and negatives in a test. The ROC curves are plotted and smoothed using SPSS software (SPSS Inc., Chicago, Illinois, USA) with the sensitivity on the y axis and \((1 - \text{specificity})\) on the x axis.

The five-fold cross-validation protocol was used to test the efficacy of the overall performance of this Bayesian Model. All patients are divided randomly into five approximately equal subsets. Four sets are used as training data sets to compute the LRs of the individual factor. The remaining set is used as the test data set to count the number of predicted TP and FP where one patient is predicted to be no-reflow if the LR exceeds a particular cutoff, \( LR_{\text{cut}} \), and to be negative otherwise. This process is performed in turn five times, and finally, the number of TP’s and FP’s against different LRs across five test data sets are summed to calculate the TP/FP ratio and the sensitivity \( (TP/T) \) and specificity \( (1 - (FP/F)) \) for the ROC curve.

**Results**

**Clinical, laboratory, and procedural characteristics of the study groups**

Overall, 1059 consecutive STEMI patients were included in data analysis. Of 1059 consecutive STEMI patients undergoing PPCI, a total of 206 (19.5%) patients developed no-reflow. Because our study is a registry one, we just collected the available baseline clinical, laboratory, and procedural characteristics in our emergency department. Seventy-nine factors of different types were recorded, which are detailed in the legend of Fig. 1. The main clinical, laboratory, and procedural characteristics of the patients are shown in Table 1. Details of high-burden thrombus formation are shown in Fig. 2. Categorical variables are presented as percentages. The age and other continuous variables are expressed as mean \( \pm \) SD. The comparison of the data between the two groups was performed using an unpaired \( t \)-test for continuous variables and using a \( \chi^2 \)-test or Fisher exact test for discrete variables. A \( P \) value of less than 0.05 was considered to be significant. These statistical tests were performed using SPSS 18.0. The age, time-to-hospital admission, Killip classes, systolic blood pressure on admission, treatment of intra-aortic balloon pump before or during the procedure, the degree of IRA stenosis, TIMI flow grade of IRA before procedure, and high-burden thrombus formation were significantly different between the patients with no-reflow and normal reflow.

**Seventy-nine factors were collected to assess the confidence of the no-reflow phenomenon**

Seventy-nine factors of different types were recorded to assess the risk of the no-reflow phenomenon, which are shown in Fig. 1. We used the TIMI flow grade as the gold positive and negative standard to measure the reliability of each factor. Each factor was stratified into different confidence bins. Figure 1 shows the LR\( f \) for each confidence bin of each factor \( f \). In theory, \( LR(f) > 1 \) of any confidence bin indicates that factor \( f \) has the ability to identify the no-reflow phenomenon. As can be seen in Fig. 1, all these 79 factors have LRs greater than 1, suggesting that all of them can be used to assess the no-reflow phenomenon. From Fig. 1, we can also find that there are huge differences between the reliability of these 79 factors. The number of stenosed vessels \( LR_{\text{max}} = 10 \), peripheral vascular disease \( LR_{\text{max}} = 6.63 \), and the number of stents planted \( LR_{\text{max}} = 6.62 \) especially have higher reliability than the rest, which means that these three factors are more predictive for the no-reflow phenomenon.

**The combined likelihood ratio was used to measure the reliability of the no-reflow phenomenon**

Because the prior odds are a constant, the posterior odds are proportional to LR. Therefore, LR can theoretically measure the reliability of no-reflow [Eq. (1)]. To test this speculation, during the five-fold cross-validation, we change the \( LR_{\text{cut}} \) and plot the ratio of TP/FP as a function of the cutoff LR in Fig. 3. TP/FP, which acts as a measure of the accuracy of a test, increases monotonically with the cutoff LR, confirming that the combined LR can be used as an appropriate confidence score to measure the odds of a real no-reflow phenomenon as well as the individual LRs.

**The Bayesian Model has a higher efficacy than a Single-Factor Model**

To better predict the no-reflow phenomenon, we established the Bayesian Model using combined LRs and compared it with the Single-Factor Models, where the confidence of the no-reflow is assigned by LR of the confidence bins of individual factor. The resulting ROC curves are shown in Fig. 4. Each point on the ROC curve of each assessment model indicates the sensitivity and specificity obtained from one test against a particular \( LR_{\text{cut}} \). The area under the ROC curve (AUC) is an indicator of the efficacy of the assessment system. An ideal test with perfect discrimination (100% sensitivity and 100% specificity) has an AUC of 1.0, whereas a noninformative prediction has the area 0.5, indicating that it may be achieved by mere guess. The more the AUC of a test approximates 1.0, the higher the overall

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We stratified each factor into different confidence bins and then used likelihood ratios (LRs) to measure the reliability of these bins to increase the sensitivity and specificity of the Bayesian system. This figure shows the LR(\textit{f}) for each confidence bin of each factor. 1, sex; 2, age; 3, current smoker; 4, history of drinking; 5, diabetes mellitus; 6, hypertension; 7, dyslipidemia; 8, previous myocardial infarction; 9, previous percutaneous coronary intervention; 10, cerebral embolism history; 11, cerebral hemorrhage history; 12, peripheral vascular disease history; 13, time-to-hospital admission; 14, Killip classes; 15, heart rate on admission; 16, systolic blood pressure on admission; 17, diastolic blood pressure on admission; 18, high blood pressure on admission; 19, dynamic ST-segment evolution; 20, arrhythmia; 21, white blood cell count; 22, percentage of neutrophils; 23, neutrophil count; 24, red blood cell count; 25, hemoglobin; 26, platelet count; 27, mean platelet volume; 28, thrombocytocrit; 29, platelet distribution width; 30, prothrombin time; 31, prothrombin time activity; 32, international normalized ratio; 33, activated partial thromboplastin time; 34, fibrinogen; 35, urea nitrogen; 36, creatinine; 37, uric acid; 38, plasma glucose; 39, serum sodium; 40, serum kalium; 41, serum chloride; 42, alanine aminotransferase; 43 aspartate aminotransferase; 44, \gamma-glutamyl transferase; 45, triglycerides; 46, total cholesterol; 47, high-density lipoprotein cholesterol; 48, low-density lipoprotein cholesterol; 49, C-reactive protein; 50, brain natriuretic peptide; 51, left ventricular end-diastolic diameter; 52, left ventricular end-systolic diameter; 53, left ventricular ejection fraction; 54, the ratio of early diastolic transmitral inflow velocity (E) to late diastolic transmitral inflow velocity (A), E/A; 55, ventricular wall motion abnormalities; 56, treatment of aspirin before procedure; 57, treatment of clopidogrel before procedure; 58, treatment of glycoprotein IIb/IIa inhibitor before procedure; 59, treatment of \beta blocker before procedure; 60, treatment of intra-aortic balloon pump during procedure; 61, the number of stenosed vessels; 62, the number of treated vessels; 63, non-infarct-related artery (IRA) being treated or not; 64, IRA location; 65, reference diameter of the IRA; 66, degree of stenosis; 67, lesion length; 68, Thrombolysis in Myocardial Infarction flow grade before procedure; 69, high-burden thrombus formation; 70, lesion extension; 71, lesion morphology; 72, lesion shape; 73, cutoff pattern of occlusion; 74, thrombosis; 75, maximum diameter of thrombus; 76, muscle bridge; 77, predilatation; 78, thrombus aspiration before primary percutaneous coronary intervention; 79, the number of stents planted.

We also constructed predictive software with the no-reflow data stored in the relational database for retrieval. Using the predictive software is a simple process. In the first step, the user is asked to provide the 10 factors described before of patients. Then, the users will be presented with the table of analytical results on the risk of no-reflow after PPCI and the sensitivity and specificity. Proper results will be presented even if the data are incomplete.

**Discussion**

In humans, no-reflow is caused by the variable combination of four pathogenetic components [12]: (a) distal atherothrombotic embolization; (b) ischemic injury; (c) reperfusion injury; and (d) susceptibility of coronary microcirculation to injury. Although multiple possible predictors of no-reflow have been studied, it is still hard to extract confident conclusions on the basis of these findings. Despite the lack of objective evidence to improve clinical endpoints, pharmacological and procedural interventions for no-reflow are used widely owing to their beneficial impact on myocardial perfusion. It is important to assess patients at high risk of no-reflow not only because no-reflow is associated with worse clinical

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**A Ten-Factor Model has a higher efficacy than a Single-Factor Model**

Limited indexes can be collected in the process of clinical diagnosis; thus, we then establish a Ten-Factor Model by integrating the 10 factors with the highest AUC shown in Fig. 5, including serum kalium, the IRA location, time-to-hospital admission, serum chloride, heart rate on admission, age, platelet count, left ventricular end-systolic diameter, red blood cell count, and the degree of stenosis, with an AUC of 0.666, 0.659, 0.654, 0.632, 0.630, 0.622, 0.612, 0.602, 0.593, and 0.593, respectively. All of these indicators are simple to obtain. We found that this model has an area ~0.79, suggesting that it also has a higher ability to identify the true no-reflow than the single factors. We find that the extra 69 factors can improve efficacy of assessment, although the evaluation ability of the individual factor incorporated is relatively low.

We stratified each factor into different confidence bins and then used likelihood ratios (LRs) to measure the reliability of these bins to increase the sensitivity and specificity of the Bayesian system. This figure shows the LR(\textit{f}) for each confidence bin of each factor. 1, sex; 2, age; 3, current smoker; 4, history of drinking; 5, diabetes mellitus; 6, hypertension; 7, dyslipidemia; 8, previous myocardial infarction; 9, previous percutaneous coronary intervention; 10, cerebral embolism history; 11, cerebral hemorrhage history; 12, peripheral vascular disease history; 13, time-to-hospital admission; 14, Killip classes; 15, heart rate on admission; 16, systolic blood pressure on admission; 17, diastolic blood pressure on admission; 18, high blood pressure on admission; 19, dynamic ST-segment evolution; 20, arrhythmia; 21, white blood cell count; 22, percentage of neutrophils; 23, neutrophil count; 24, red blood cell count; 25, hemoglobin; 26, platelet count; 27, mean platelet volume; 28, thrombocytocrit; 29, platelet distribution width; 30, prothrombin time; 31, prothrombin time activity; 32, international normalized ratio; 33, activated partial thromboplastin time; 34, fibrinogen; 35, urea nitrogen; 36, creatinine; 37, uric acid; 38, plasma glucose; 39, serum sodium; 40, serum kalium; 41, serum chloride; 42, alanine aminotransferase; 43 aspartate aminotransferase; 44, \gamma-glutamyl transferase; 45, triglycerides; 46, total cholesterol; 47, high-density lipoprotein cholesterol; 48, low-density lipoprotein cholesterol; 49, C-reactive protein; 50, brain natriuretic peptide; 51, left ventricular end-diastolic diameter; 52, left ventricular end-systolic diameter; 53, left ventricular ejection fraction; 54, the ratio of early diastolic transmitral inflow velocity (E) to late diastolic transmitral inflow velocity (A), E/A; 55, ventricular wall motion abnormalities; 56, treatment of aspirin before procedure; 57, treatment of clopidogrel before procedure; 58, treatment of glycoprotein IIb/IIa inhibitor before procedure; 59, treatment of \beta blocker before procedure; 60, treatment of intra-aortic balloon pump during procedure; 61, the number of stenosed vessels; 62, the number of treated vessels; 63, non-infarct-related artery (IRA) being treated or not; 64, IRA location; 65, reference diameter of the IRA; 66, degree of stenosis; 67, lesion length; 68, Thrombolysis in Myocardial Infarction flow grade before procedure; 69, high-burden thrombus formation; 70, lesion extension; 71, lesion morphology; 72, lesion shape; 73, cutoff pattern of occlusion; 74, thrombosis; 75, maximum diameter of thrombus; 76, muscle bridge; 77, predilatation; 78, thrombus aspiration before primary percutaneous coronary intervention; 79, the number of stents planted.

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outcomes but also because the majority of patients (up to 60%) presenting with STEMI do not have no-reflow. The relevant treatments should be used on the most appropriate patients to prevent no-reflow effectively, save medical resources, and prevent potential adverse outcomes caused by the irrational use of the special medicine and instruments to normal flow arteries.

Clinically, the risk of no-reflow is often evaluated by cardiologists simply according to the characteristics of STEMI patients such as age, smoking history, thrombus burden, and some other indicators with a low evidence level. In this study, we first measure the reliability of individual factors using LR. Then, we use naive Bayesian networks to combine the individual factor for confidence assessment. The Bayesian Model and Ten-Factor Model have been proven to have higher

| Characteristics | No-reflow group | Normal reflow group | P value |
|-----------------|----------------|---------------------|---------|
| Male            | 83.0%          | 84.4%               | 0.622   |
| Age (years)     | 59 ± 12        | 57 ± 11             | 0.003   |
| Current smoker  | 69.9%          | 70.0%               | 0.981   |
| Diabetes mellitus | 19.4%       | 23.1%               | 0.256   |
| Hypertension    | 53.4%          | 56.4%               | 0.438   |
| Dyslipidemia    | 4.9%           | 7.5%                | 0.136   |
| Previous MI     | 5.8%           | 7.3%                | 0.466   |
| Previous PCI    | 4.9%           | 7.0%                | 0.258   |
| Time to hospital admission (h) | 5.3 ± 2.5 | 4.9 ± 2.5 | 0.029 |
| Killip classes  |                |                     | 0.001   |
| 1               | 6.3%           | 8.6%                |         |
| 2               | 76.7%          | 83.1%               |         |
| 3               | 9.7%           | 5.7%                |         |
| 4               | 7.3%           | 2.6%                |         |
| Physical findings on admission | | | |
| Heart rate (beats/min) | 79 ± 18 | 80 ± 16 | 0.398 |
| Systolic blood pressure (mmHg) | 114 ± 20 | 117 ± 19 | 0.034 |
| Diastolic blood pressure (mmHg) | 73 ± 13 | 75 ± 12 | 0.087 |
| White blood cell count (x10^9/l) | 11.23 ± 3.69 | 10.97 ± 3.29 | 0.162 |
| Neutrophil count (x10^9/l) | 8.80 ± 3.49 | 8.55 ± 3.30 | 0.185 |
| Plasma glucose (mmol/l) | 2.47 ± 0.68 | 2.51 ± 0.75 | 0.336 |
| Triglycerides (mmol/l) | 1.75 ± 1.23 | 1.74 ± 1.05 | 0.461 |
| Total cholesterol (mmol/l) | 4.61 ± 0.93 | 4.64 ± 0.98 | 0.352 |
| High-density lipoprotein cholesterol (mmol/l) | 1.03 ± 0.24 | 1.01 ± 0.25 | 0.200 |
| Low-density lipoprotein cholesterol (mmol/l) | 2.97 ± 0.81 | 3.02 ± 0.87 | 0.246 |
| Left ventricular ejection fraction | 52.66 ± 9.43 | 54.18 ± 9.59 | 0.050 |
| Treatment before or during the procedure | | | |
| Aspirin | 88.8% | 83.1% | 0.103 |
| Clopidogrel | 88.3% | 81.5% | 0.059 |
| Glycoprotein IIb/IIIa inhibitor | 31.1% | 26.5% | 0.186 |
| Intra-aortic balloon pump | 17.5% | 7.0% | <0.001 |
| Angiography | | | |
| The number of stenosed vessels | 2 ± 1 | 2 ± 1 | 0.155 |
| The degree of IRA stenosis | 97.79 ± 6.25 | 96.72 ± 6.22 | 0.027 |
| TIMI flow grade of IRA before procedure | | | 0.039 |
| 0 | 71.4% | 62.1% |
| 1 | 3.9% | 2.8% |
| 2 | 6.8% | 10.8% |
| 3 | 18.0% | 24.3% |
| High-burden thrombus formation | 78.6% | 68.0% | 0.003 |
| Procedure | | | |
| Multivessel disease | 43.2% | 46.7% | 0.071 |
| Multivessel PCI | 1.5% | 5.0% | 0.071 |
| Predilatation | 92.7% | 96.0% | 0.096 |
| Thrombus aspiration | 65.9% | 65.1% | 0.881 |
| Stent number | 1 ± 1 | 1 ± 1 | 0.530 |

IRA, infarct-related artery; PCI, percutaneous coronary intervention; TIMI, Thrombolysis in Myocardial Infarction.

Table 1 Clinical, laboratory, and procedural characteristics of the study groups

Fig. 2

Without high-burden thrombus
With high-burden thrombus

Fig. 3

TP/FP ratio as a function of the likelihood ratio (LR) cutoff for the Bayesian Model. This figure plots the TP/FP ratio as a function of the likelihood ratio cutoff for the Bayesian Model. The numbers of TP and FP are from the five-fold cross-validation. FP, false positive; TP, true positive.
sensitivity and better specificity to predict true relations between the multiple factors and the no-reflow phenomenon by cross-validation than the simple factors alone. This Bayesian method has also been shown in other domains to be data efficient and to address some of the limitations of conventional statistical methods.

Compared with previous studies, the advantages of this analysis may result from the following points: (a) this Bayesian approach integrated 79 factors to establish the predictive models, which can reduce the FP and false negatives derived from single factor; (b) a Ten-Factor Model was also established, which was proved to have a higher efficacy than a Single-Factor Model – the indexes are much more available than the Bayesian Model of 79 factors as well; (c) this Bayesian approach can provide us information on the risk of no-reflow quantificationally; (d) this Bayesian approach can provide us with the sensitivity and specificity to predict true relations between the multiple factors and the no-reflow phenomenon by cross-validation than the simple factors alone. This Bayesian method has also been shown in other domains to be data efficient and to address some of the limitations of conventional statistical methods.

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and specificity of the no-reflow risk, and these advantages make the results more informative and predictable; and (e) the advantage of the Bayesian method is that we can assess the risk of no-reflow easily by entering several numbers into the predictive system.

The cardiac biomarkers and pharmacological factors are important to predict the no-reflow phenomenon. These factors cannot be included in this model because of lack of data, such as cardiac biomarkers, calcium channel blocker, nicorandil, and adenosine. This study was carried out to determine the risk factors of no-reflow using the Bayesian method, and future research focusing on the individual risk factors and its mechanism is needed.

In summary, no-reflow remains a common and serious adverse outcome of PPCI in STEMI patients. To date, the most effective strategy is predicting it as early as possible.

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Conflicts of interest
There are no conflicts of interest.

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