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Prediction of massive coronary thrombosis of the infarct-related artery in ST-elevation myocardial infarction

Aim	To identify clinical, laboratory and angiographic predictors for development of massive coronary thrombosis in patients with ST-segment elevation myocardial infarction (STEMI).
Material and Methods	This prospective, single-site study included 137 patients with STEMI (mean age, 66.5±13.2 years). Among these patients, 59 were in the group of massive coronary thrombosis and 78 patients were in the group of minor thrombosis. To identify predictors for the development of massive coronary thrombosis, medical history data, blood count and biochemistry, coagulogram, and angiography data were analyzed. A predictive model was constructed using the method of binary logistic regression followed by a search for the optimum value of the prognostic function with a ROC analysis.
Results	The study showed statistically significant roles of total bilirubin, platelets, prothrombin ratio (PTR), activated partial thromboplastin time (APTT), and presence of inferior myocardial infarction in prediction of massive coronary thrombosis in STEMI. The model sensitivity was 71.2%, specificity 75.6%, and overall diagnostic efficacy 73.7%.
Conclusion	The predictive model for the development of massive coronary thrombosis in STEMI based on laboratory and instrumental data potentially allows assessing the thrombus load in the infarction-involved coronary artery and determining the optimum tactics of percutaneous coronary intervention in patients with STEMI. This reduces the probability of distal embolization with fragments of the disintegrated thrombus and improves the prognosis of STEMI patients both during the stay in the hospital and in the long-term. According to results of this study, the prognostic model for massive coronary thrombosis in STEMI based on such indexes as the platelet count, PTR, APTT, total bilirubin, and presence of inferior myocardial infarction provides accurate predictions in 73.7% of cases. Independent predictors of massive coronary thrombosis were inferior myocardial infarction and total bilirubin.

Keywords	ST-segment elevation acute myocardial infarction; percutaneous coronary intervention; delayed stenting; massive coronary thrombosis
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Introduction

ST-segment elevation myocardial infarction (STEMI) is the most severe type of coronary heart disease (CHD) and remains a leading cause of death worldwide. Occlusive atherothrombosis following a rupture or erosion of an atherosclerotic plaque is the main cause of STEMI. Thrombosis of an infarct-related artery (IRA) a cause of STEMI in most cases (up to 91%) [1, 2]. IRA increases the risk of complications in major coronary thrombosis: distal embolization, no-reflow phenomenon, stent thrombosis [3], and increased risk of major adverse cardiovascular events (MACE) and mortality [1, 2]. Intracoronary thrombosis is one of the main factors that reduce survival in STEMI patients despite the availability of novel drug treatments and interventions (glycoprotein IIb/IIIa inhibitors, aspiration thrombectomy). The identification of risk factors and the creation of prognostic models for the development of major coronary thrombosis in STEMI can provide a basis for personalized therapy, possibly improve clinical outcomes, and reduce the likelihood of complications. This study examines the role of various factors associated with severe IRA thrombosis in patients with STEMI who underwent primary percutaneous coronary intervention (PCI) with this objective in view.

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Material and methods

Study population

The study protocol was approved by the local ethics committee of the Sechenov Medical University and complied with the Declaration of Helsinki. All patients signed the informed consent before the procedure.

A prospective selection of 137 patients with a history of emergency primary PCI for STEMI was conducted in the Sechenov University between January 2020 and February 2022. STEMI was diagnosed based on the analysis of complaints (typical retrosternal chest pain for > 30 minutes), medical history, 12-lead ECG findings (ST segment elevation > 1 mm in two or more adjacent leads or newly identified left bundle branch block), and troponin I levels. Patients who received thrombolytic therapy for STEMI for 24 hours and had cardiogenic shock at admission and an active infection (including COVID-19), had a history of systemic inflammatory diseases, cancer, hepatic disorders, and renal failure. Patients with baseline anemia (Hb<130 g/L in male patients and Hb<120 g/L in female patients) were excluded from the analysis.

Laboratory and clinical examination findings

The following data were collected and analyzed: age, sex, smoking, body mass index (BMI), family history of CHD, glucose, creatine phosphokinase (CPK), creatine phosphokinase-MB (CPK-MB), platelets, leukocytes, total bilirubin, prothrombin ratio, international normalized ratio (INR), activated partial thromboplastin time (aPTT), fibrinogen, hemoglobin, and angiographic data (infarct-related coronary artery, severity of coronary thrombosis). Blood was drawn for the analysis at admission to the hospital.

Drug treatments and PCI

Before hospitalization, all patients received double antiplatelet therapy (DAT) including acetylsalicylic acid 300 mg and clopidogrel 600 mg. Heparin 5000 IU was used as an anticoagulant. Patients also received beta-blockers, statins, and other drugs in accordance with the current clinical guidelines for the management of STEMI. Primary coronary angiography (CAG) was performed using radial access. Coronary arteries were visualized using 5F diagnostic catheters in the left and right oblique views with caudal and cranial angulation. The frequency of visualization was 30 FPS. Contrast enhancement agent (Omnipack 350, GE Healthcare) was administered manually at each position. Primary PCI was performed following CAG, which was used to verify the IRA with a correlation with electrocardiographic (ECG) criteria. The baseline severity of coronary thrombosis was assessed using the Thrombolysis in Myocardial Infarction (TIMI) thrombus grades (TTG); guidewire recanalization and/or pre-dilatation with a small diameter balloon (1.5–2.0 mm) was conducted in occlusive lesion (TTG5).

- TIMI Thrombus Grade 0

No cineangiographic characteristics of thrombus present.
TIMI Thrombus Grade 1
Hazy, possible thrombus present. Angiography demonstrates characteristics such as reduced contrast density, haziness, irregular lesion contour, or a smooth convex «meniscus» at the site of total occlusion suggestive but not diagnostic of thrombus.

- TIMI Thrombus Grade 2

Thrombus present – small size: Definite thrombus with greatest dimensions less than or equal to 1/2 vessel diameter.

- TIMI Thrombus Grade 3

Thrombus present – moderate size: Definite thrombus but with greatest linear dimension greater than 1/2 but less than 2 vessel diameters.

- TIMI Thrombus Grade 4

Thrombus present – large size: As in Grade 3 but with the largest dimension greater than or equal to 2 vessel diameters.

- TIMI Thrombus Grade 5

Recent total occlusion, can involve some collateralization but usually does not involve extensive collateralization, tends to have a «beak» shape and a hazy edge or appearance of distinct thrombus.

- TIMI Thrombus Grade 6

Chronic total occlusion, usually involving extensive collateralization, tends to have distinct, blunt cut-off/edge and will generally clot up to the nearest proximal side branch.

The patients were divided into two groups based on the severity of coronary thrombosis. Patients with TTG <3 were included in the mild coronary thrombosis group, and patients with TTG ≥3 were included in the major coronary thrombosis group. Primary PCI was performed in some patients of the major coronary thrombosis group in two stages: the first stage was the restoration of the vessel lumen and antegrade coronary blood flow, and the second stage was stent implantation. At the first stage, the so-called minimally invasive mechanical strategy (MIMS) was applied, followed by enhanced antiplatelet therapy, in order to restore blood flow to TIMI 3. MIMS involves mechanical guidewire recanalization combined with balloon dilatation (balloon catheter diameter not more than 1.5–2 mm) and/or manual vacuum thrombus aspiration (6 Fr Export catheter). All patients subjected to

MIMS received glycoprotein IIb/IIIa inhibitors after the primary CAG for up to 48 hours. Unfractionated sodium heparin 50–60 IU/kg was continuously administered intravenously for the anticoagulation during PCI in order to achieve the target level of heparinization, which was assessed by activated clotting time (ACT) with the target value of 300–350 sec. Subsequently, repeat CAG was conducted and, if necessary, stenting of residual stenosis was performed after at least 6–7 days. Stenting of the residual stenosis was carried out taking into account its hemodynamic significance.

Statistical analysis

The statistical processing of the data obtained was performed in SPSS Statistics 26.0. Normality of the distribution was tested using the Lilliefors test based on the Kolmogorov – Smirnov test. Normally distributed qualitative variables were presented as the arithmetic means (M) and standard deviations (± SD). Non-normally distributed quantitative indicators were expressed as the medians (Me) and interquartile ranges (Q1 – Q3). Intergroup differences were estimated using the Student's t-test in the case of the normal distribution, and the Mann-Whitney U-test in the non-normal distribution. A comparative analysis of categorical variables was performed using Pearson's χ^2 or Fisher's exact test. Nominal indicators were expressed by the absolute numbers of observations and the percentages of the subgroups. The prognostic model was created using a binary logistic regression. Coarse and corrected odds ratios (OR) were calculated for the predictors included in the prognostic model. ROC analysis was conducted to adjust the optimal prognostic function value. The critical level of statistical significance was $p<0.05$ in all statistical analysis procedures.

Results

Data of 137 patients with STEMI were analyzed in the study. The mean age was 67 years, and 87% of patients were male. The baseline clinical and laboratory findings of the compared groups are presented in Table 1.

The analysis of baseline data found that age, male sex, smoking status, and body mass index were balanced between the subgroups, and a positive history of CHD was statistically significantly more common among patients with severe coronary thrombosis. There was no significant difference in the analysis of other data: glucose levels, hemoglobin, platelet and leukocyte counts, CPK, CPK-MB, PR, INR, aPTT, fibrinogen levels. However, a significant intergroup difference was shown in the levels of total bilirubin ($p=0.04$). Moreover, it was found that inferior myocardial infarction was statistically significantly more common in the group of major coronary thrombosis ($p<0.001$).

Later, an attempt was made to create a mathematical model that would allow predicting the likelihood of major coronary thrombosis depending on various clinical and laboratory parameters. A step-wise selection of factors by exclusion produced a logistic function (1) including 5 predictors:

Table 1. Baseline clinical and laboratory data

Parameter	Mild coronary thrombosis (n=78)	Major coronary thrombosis (n=59)	p
Age, years	66.4 ± 12.6	66.2 ± 13.2	0.771
Male, n (%)	68 (87.2)	52 (88.1)	1.000
Smoking, n (%)	46 (58.9)	34 (57.6)	0.874
BMI, kg/m2	25.5 (20.6–28.8)	26.3 (21.3–27.4)	0.829
Family history of CHD, n (%)	10 (12.8)	16 (27.1)	0.035
Glucose, mmol/L	6.8 (5.8–8.8)	6.85 (5.46–8.0)	0.944
Hemoglobin, g/L	144 (131-151)	139.5 (130-150)	0.327
Platelets, × 109	238 (204-288)	228 (200-263)	0.256
Leukocytes, × 109	9.7 (5.3–18.3)	11 (6.8–20.9)	0.08
Creatine phosphokinase, U/L	1229 (612-1855)	787 (338-1822)	0.114
Creatine phosphokinase-MB, U/L	113.3 (57.3–219.1)	116.8 (39.1-210)	0.747
Total bilirubin, μmol/L	9.2 (6.4–13.8)	11.0 (7.4–18.6)	0.04*
Prothrombin ratio	1.07 (1.0–1.13)	1.06 (1.0–1.15)	0.754
INR	1.08 (1.02–1.15)	1.06 (1.01–1.15)	0.545
aPTT, sec	27.2 (25.4–29.7)	28.2 (26.2–31.0)	0.157
Fibrinogen, mg/L	3.8 (3.0–4.5)	4.2 (3.08–5.35)	0.088
TTG	1.0 (0-2)	4 (3-4)	<0.001*
MIMS, n (%)	–	22 (37.3)	–
Not stented, n (%)	–	14 (23.8)	–
Localization of MI			
Anterior MI, n (%)	32 (41)	16 (27.1)	0.09
Lateral MI, n (%)	23 (29.5)	10 (16.9)	0.09
Inferior MI, n (%)	7 (9)	30 (50.8)	<0.001*
Posterior MI, n (%)	1 (1.7)	8 (10.3)	0.077

BMI, body mass index; CHD, coronary heart disease; INR, international normalized ratio; aPTT, activated thromboplastin time; TTG, thrombus grade score; MIMS, minimally invasive mechanical strategy; MI, myocardial infarction.

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Table 2. Relationship of the model (1) predictors with the odds of major coronary thrombosis

Predictor	Coarse OR (95 % CI)	p	Corrected OR (95 % CI)	p
Platelets	0.997 (0.99–1.00)	0.171	0.99 (0.99–1.00)	0.276
PR	1.5 (0.65–3.47)	0.346	1.6 (0.78–3.34)	0.199
aPTT	1.03 (0.98–1.09)	0.276	1.04 (0.97–1.12)	0.265
Inferior MI	10.5 (4.1–26.6)	<0.001	22.2 (7.3–67.35)	<0.001
Total bilirubin	1.07 (1.02–1.14)	0.011	1.1 (1.03–1.16)	0.006

PR, prothrombin ratio; aPTT, activated partial thromboplastin time; MI, myocardial infarction; OR, odds ratio.

$$p= 1/(1+e-z) *100\%$$
$$z= – 2.93–0.003*Xpl+0.478*XPR+0,04*XaPTT+ 2.6*Xinf +0.09*XTBil; (1),$$

where p is the probability of major coronary thrombosis (a decimal quantity); Xpl is the platelet count (10*9/L); XPR is the prothrombin ratio; XaPTT is the activated partial thromboplastin time (sec); Xinf is inferior myocardial infarction (0 – no, 1 – yes); XTBil is total serum bilirubin (μmol/L).

Based on the coefficient values, an increase in such indicators as aPTT, RP, total bilirubin and the presence of inferior was myocardial infarction are directly related to the likelihood of major coronary thrombosis. Elevated platelet count is associated with a decrease in the likelihood of major coronary thrombosis.

The resulting prognostic model was statistically significant (p<0.001). The prognostic model (1) takes into account 38.5% of the factors of the development of major coronary thrombosis according to the Nagelkerke coefficient of determination. The relationship of each predictor with the likelihood of major coronary thrombosis is presented in Table 2.

The ROC analysis determined the best-possible value of the prognostic function P. Figure 1 shows the resulting curve.

The logistic function P point was 0.378 at the cutoff point. Patients with P≥0.378 were likely to face high risk of major coronary thrombosis. Low risk of major coronary thrombosis was predicted with P<0.378. At the selected cutoff point, sensitivity of the prognostic model (1) value was 71.2% (42 of 59 cases of major coronary thrombosis were predicted correctly), specificity of the model was 75.6% (59 of 78 cases of the absence of major coronary thrombosis were predicted correctly). Overall diagnostic efficacy was 73.7%.

Discussion

Our findings demonstrated that total bilirubin is an independent predictor of major coronary thrombosis. Similar results were confirmed in a previously published study [4]. Elevated levels of total bilirubin in patients with major coronary thrombosis is probably associated with a more severe inflammatory process, a more marked increase in the activity of heme oxygenase-1 (HO-1) in the macrophages. HO-1 synthesis is induced by various damaging factors, including higher levels of reactive oxygen species [5]. The enzyme and its products have anti-inflammatory and antioxidant properties [6]. The presence of inferior myocardial infarction is another independent predictor of major coronary thrombosis. It was very often associated in our study with the involvement of the middle third of the right coronary artery (RCA). Marked calcified plaques are often localized in the middle third of the RCA [7] and are sensitive to mechanical stress that occurs with each heartbeat.

Stenting of the calcified stenosis site in the middle third of the RCA will increase the stiffness of this part, which can result in refractory restenosis. It was reported that excimer laser treatment was effective for calcified plaques in the middle third of the RCA [8]. Another study showed that thickness of epicardial adipose tissue (> 5.3 mm) can serve an independent predictor of major coronary thrombosis [9]. We did not analyze the thickness of adipose epicardial tissue.

The described prognostic model of major coronary thrombosis in STEMI based on laboratory and clinical examination data allows predicting the severity of IRA thrombotic load even before primary CAG and thus determining in advance the best-possible strategy of endovascular treatment in patients with major thrombotic load of IRA with TTG≥3. This will make it possible to reduce the likelihood of distal embolization with fragments of a thrombus as a result of the no-reflow phenomenon, which will improve the in-hospital and long-term prognosis of patients with STEMI.

There are certain peculiarities in the treatment of STEMI patients with major IRA thrombosis. Major residual thrombosis is often visualized in the coronary lumen even after aspiration or rheolytic thrombectomy. Stenting of the major residual thrombosis site is associated with a risk of distal embolization, microcirculatory bed damage (slow-/no-reflow), and an

increased risk of death [10–14]. The prevention of distal embolization and no-reflow delayed stenting (two-stage revascularization) seems promising in such patients. This method involves primary intervention (balloon predilation and/or aspiration thrombectomy) in order to restore TIMI-3 blood flow, the administration of antiplatelet drugs (glycoprotein GP IIb/IIIa inhibitors) and anticoagulants during the period before control CAG, with the intervention performed 4–7 days later, evaluation of residual stenosis, and if necessary stent grafting [15, 16].

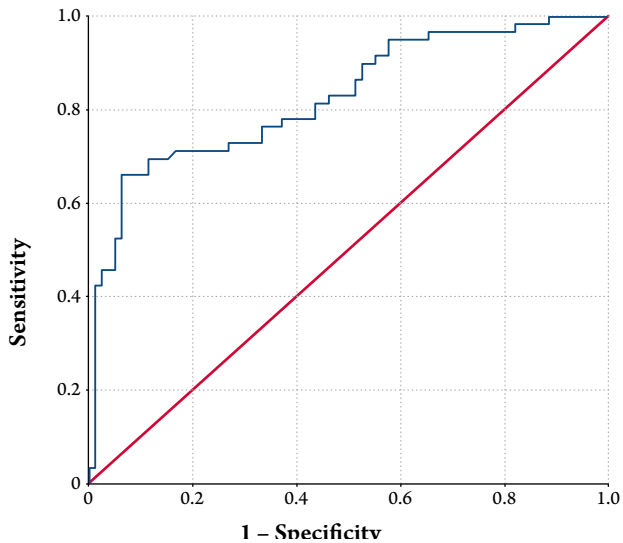
Limitations

This study has several limitations. This study analyzed patients with primary PCI only, it did not include patients subjected to PCI after thrombolytic therapy. This single-center study was conducted in a relatively small sample of patients (n=137). The fact that the prognostic model was not verified in a different patient sample is likely to limit some of the findings that were reported.

Conclusion

The prognostic model of major coronary thrombosis in STEMI based on such predictors as platelet count, PR, aPTT, total bilirubin, and the presence of inferior myocardial infarction provides a prediction accuracy of 73.7%, according to the findings of this study. Inferior myocardial infarction and total bilirubin levels are independent predictors of major coronary thrombosis.

Figure 1. ROC-curve of the dependence of major coronary thrombosis prognosis on the value of the logistic function P



Area under the ROC curve was 0.806 ± 0.037 (95 % CI: 0.733–0.88).

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