European Review for Medical and Pharmacological Sciences 2021; 25: 5137-5144

Correlation between culprit vessel/tirofiban and reperfusion bradyarrhythmia in patients with ST-segment elevation myocardial infarction after emergency PCI

Z. MA[1], B. NING[1], D. WU[2], W.-G. LIU[1], Y.-J. GUO[1]

[1]Department of Cardiology, Fuyang People's Hospital of Anhui Province, Fuyang, China
[2]Department of Pharmacy, Fuyang People's Hospital of Anhui Province, Fuyang, China

Abstract. – OBJECTIVE: To evaluate the correlation between culprit vessel/tirofiban and reperfusion bradyarrhythmia in patients with ST-segment elevation myocardial infarction (STEMI) after emergency percutaneous coronary intervention (PCI).

PATIENTS AND METHODS: A total of 123 STEMI patients undergoing emergency PCI in our hospital from September 2018 to September 2019 were selected and divided into the reperfusion arrhythmias (RA) group (50 cases) and non-RA group (NRA, 73 cases) according to whether RA occurring during PCI. The baseline data such as age and underlying disease were statistically analyzed. Then, the differences were compared between the two groups. According to whether reperfusion bradyarrhythmia (RB) occurring during PCI, 123 STEMI patients were divided into the RB group (63 cases) and non-RB group (60 cases). The relation between culprit vessel/tirofiban and RB was analyzed. ROC curves analysis and multivariate logistic regression were conducted for the risk factors of RA and RB.

RESULTS: Among 123 patients with STEMI after PCI treatment, 73 patients had RA (59.35%), including RB 63 cases and tachyarrhythmia 10 cases. Results of single factor analysis showed that there was statistical significance in 3 factors including the patient age, infarction area and vascular blood flow TIMI classification between RA group and NRA group ($p<0.05$). ROC curve analysis indicated that the continuous variable patent ages had predictive value in the prevalence of RA, which resulting in an AUC 0.624 and a cut-off pointed age 57 (sensitivity 72.60, specificity 52.00). Multivariate regression analysis showed that the patient age (>57 years old), infarction area in inferior wall and grade 0 lesion vascular blood flow TIMI classification in RA group was significantly higher than that in NRA group ($p<0.05$). Tirofiban was not associated with RB in STEMI patients treated with emergency PCI, while culprit vessel was statistically significant between RB group and NRB group ($p<0.05$). Multivariate regression analysis indicated that culprit vessel of the right coronary artery and grade 0 lesions vascular blood flowed TIMI classification was independent risk factors to occurring RB in the STEMI patients with emergency PCI.

CONCLUSIONS: Tirofiban was not associated with RB in STEMI patients treated with emergency PCI. However, it may increase the risk of RB development when the culprit vessel is the right coronary artery. Therefore, timely corresponding treatments and reduction of reperfusion damage are of great significance for those patients.

Key Words:
ST-segment elevation myocardial infarction, Percutaneous coronary intervention, Reperfusion bradyarrhythmia, Tirofiban, Right coronary artery.

Introduction

Emergency percutaneous coronary intervention (PCI) is currently the most effective treatment for acute ST-segment elevation myocardial infarction (STEMI), opening the sinus vessels, reducing the infarct size, and achieving myocardial reperfusion. However, due to calcium overload, increased oxygen free radicals, refractory period is prone to re-entry, etc.[1] causes reperfusion arrhythmias (RA) to occur when the criminal vessels are opened, causing reperfusion injury. The clinical manifestations of RA are transient accelerated ventricular autonomic rhythm, ventricular fibrillation, atrioventricular block, sinus bradycardia, etc., which usually occur within 2 hours after reperfusion, usually transient. The prevalence can reach 50%-80%[2]. Although RA is a protective phenomenon, it is still one of the reasons for prolonging the operation time and in-

Corresponding Author: Zhen Ma, MD; e-mail: lvlingma0323@163.com

creasing the risk of death in patients[3], and the prevalence of reperfusion slowed arrhythmia (RB) is significantly higher than that of tachyarrhythmia[4], and there are fewer rescue measures. Therefore, it is still important to explore the risk factors of RB and reduce the occurrence of RB. Different types of arrhythmias caused by different criminal blood vessels[5] may be one of the risk factors for RB in patients with STEMI after emergency PCI. Tirofiban is a platelet glycoprotein IIb/IIIa receptor blocker, effective in the treatment of STEMI patients with no-reflow and slow blood flow during PCI[6]. However, clinical studies regarding RB for STEMI patients treated with tirofiban were rarely reported. Based on the above research background, this study used STEMI and emergency PCI patients as the research object to explore the correlation between criminal vascular and tirofiban on reperfusion slow arrhythmia, aiming to provide an effective basis for clinical prevention of RB.

Patients and Methods

Research Objects

A total of 123 STEMI patients who underwent emergency departmental admission to the Department of Cardiology, Fuyang People's Hospital from September 2018 to September 2019, and retrospectively analyzed the clinical treatment of patients' age, gender, underlying disease, and infarction, using ROC curve analysis. Logistic regression analysis of risk factors associated with postoperative RA and RB in patients with STEMI after PCI and culprit and tirofiban and other clinical features. This study was approved by the Ethics Committee of Fuyang People's Hospital. Signed written informed consents were obtained from all participants before the study.

Inclusion and Exclusion Criteria

Inclusion criteria: 1) In accordance with the STEMI diagnostic criteria established by the Chinese Medical Association in 2010[7] and in accordance with the "Classic recommendations for the diagnosis and treatment of acute ST-segment elevation myocardial infarction"[8] in the direct PCI decision class I recommendation and IIa Class recommendation; 2) After emergency coronary angiography (CAG) confirmed subtotal occlusion or complete occlusion of the infarct-related artery; 3) Patients and their families signed informed consent.

Exclusion criteria: 1) Emergency stents were not implanted; 2) Severe liver and kidney dysfunction; 3) Allergic to iodine contrast agents 4 emergency thrombolysis; 5 general information is not complete.

Methods

All patients underwent emergency CAG pre-loading aspirin enteric-coated tablets 300 mg combined with clopidogrel hydrogen sulfate tablets 600 mg or ticagrelor tablets 180 mg, combined with atypical arrhythmias in patients with atropine, lidocaine or amiodarone treatment.

Observation Indicators

RA is defined as: 1) RB: including sinus bradycardia (heart rate < 50 beats/min), sinus arrest and atrioventricular block (II degree and III degree), duration is more than 2 min; 2) Rapid heart rate Abnormalities: mainly frequent premature ventricular contractions, ventricular tachycardia; 3) Atrial fibrillation. According to the patient's emergency PCI and 2 hours postoperative ECG monitoring records, as long as a transient arrhythmia is considered as RA[5].

The tirofiban catheter was applied to the coronary artery in the upstream projectile, and the load was 10 g/kg in the first 3 min, followed by the intravenous micropump at the rate of 0.15 g/kg/min for 24 h.

Statistical Analysis

Data processing was performed using Statistical Product and Service Solutions (SPSS) 21.0 statistical software (IBM, Armonk, NY, USA). The measurement data were expressed as x±SD (standard deviation). The *t*-test was used for comparison between groups with normal distribution. The count data were analyzed by χ^2 or Fisher's exact test. $p<0.05$ indicates statistical difference. Multivariate logistic regression analysis was performed on the categorical variables with significant differences among the univariate analysis groups. The continuous variables were analyzed by using the MedCalc ROC curve, and the cut-off values were taken according to the results of the ROC curve analysis.

Results

RA Patients with STEMI After Emergency PCI

Of the 123 patients, 93 (75.61%) were male and 30 (24.39%) were female, aged 32-82 years, with an average age of (61.67±11.82) years. A total of 73

patients (59.35%) had RA after emergency PCI, of which RB accounted for 86.30% (63 cases) and tachyarrhythmia accounted for 13.70% (10 cases).

Single Factor Analysis of RA Risk Factors in Patients with STEMI After Emergency PCI

123 patients were divided into RA group (n=73) and NRA group (n=50) according to whether arrhythmia occurred after PCI. As shown in Table I, the RA group had gender, underlying disease, adverse hobbies (smoking and alcohol consumption), N-terminal B-type brain natriuretic peptide (NT-proBNP), and low-density lipoprotein cholesterol (LDL- compared with the NRA group), serum potassium levels, opening time, number of implants, left ventricular ejection fraction (LVEF), left atrial and left ventricular size were not statistically different (p>0.05), but at age, infarct location, and TIMI There were statistically significant differences in grading (p<0.05). In the infarct site, there was no significant difference in the myocardial infarction between the RA group and the NRA group (p=0.407), but there was a significant difference in the anterior wall and inferior myocardial infarction (p<0.05). In the anterior wall myocardial infarction, the occurrence of arrhythmia in the RA group was significantly lower than that in the NRA group (p=0.000). In the inferior myocardial infarction, the occurrence of arrhythmia in the RA group was significantly higher than that in the NRA group (p=0.000). In terms of TIMI grading, although there was no significant difference in TIMI blood flow level 1 and level 3 (p>0.05), there was a statistically significant difference in TIMI blood flow level 0 and level 2. When TIMI blood flow was 0, the prevalence of arrhythmia in RA group was significantly higher than that in NRA group (p<0.001). When TIMI blood flow was grade 2, the prevalence of arrhythmia in RA group was significantly lower than that in NRA group (p=0.008).

ROC Curve Analysis Results

According to the results of single factor analysis, the age of patients with continuous variables was significantly different between RA group and NRA group (p<0.05), which may be the risk factor of RA after emergency PCI in STEMI patients. The prediction of RA occurred in patients with type. As shown in Figure 1, ROC curves analysis showed that the continuous variable age had a significant predictive value for RA in this type of patients, and the area under the curve (AUC) was 0.624 (95% confidence interval: 0.532-0.710, p=0.0152), intercepted. The sensitivity at the value was 72.60, the specificity was 52.00, the Youden index was 0.2460, and the cut-off value was 57 years.

Multivariate Analysis of Risk Factors for RA in Patients with STEMI After Emergency PCI

Based on the results of univariate analysis and ROC curve analysis, the above three risk factors were brought into the age (>57 years = 1, ≤57 years = 0), inferior myocardial infarction and TIMI blood flow level 0 as independent variables. The Logistic regression equation was used to calculate the age (>57 years), inferior myocardial infarction, and TIMI blood flow level 0 as independent risk factors for RA after emergency PCI in STEMI patients, as shown in Table II.

Single Factor Analysis of Risk Factors for Slow Arrhythmia

According to whether STEMI patients had slow arrhythmia after emergency PCI, they were divided into RB group and NRB group, and were included in 63 cases and 60 cases respectively. The general clinical data of the two groups of patients were analyzed. Compared with the NRB group, the RB group had statistically significant differences in LVEF and TIMI blood flow grades 0 and 2 (p<0.05). Although age (>57 years) was an inde-

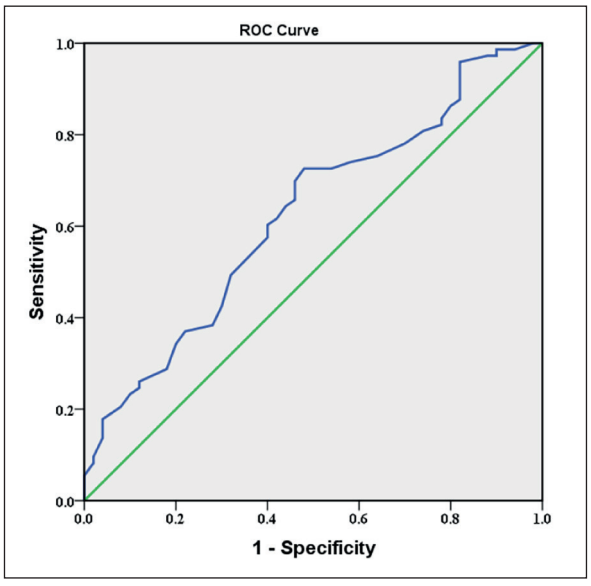

Figure 1. Patient's age predicts ROC curve of RA after emergency PCI in STEMI patients.

pendent risk factor for RA in this type of patients, and the age of RB group was larger than that of NRB group (62.16 *vs.* 60.33), the difference was not statistically significant ($p>0.05$). When TIMI was at grade 0, the occurrence of slow arrhythmia in RB group was significantly higher than that in NRB group ($p<0.05$). When TIMI was at grade 2, the occurrence of slow arrhythmia in RB group was significantly lower than that in NRB group ($p<0.05$; Table III).

Relationship Between Tirofiban and Slow Arrhythmia

123 patients with STEMI were divided into tirofiban group (n=104) and non-tirofiban group (19) according to the use of tirofiban in emergency PCI. There were 55 cases of slow arrhythmia and 49 cases of non-slow arrhythmia in the tirofiban group; 8 cases of slow arrhythmia and 11 cases of non-slow arrhythmia in the non-tirofiban group, but the difference between the two groups was

Table I. Univariate analysis of risk factors for arrhythmia in patients with STEMI after emergency PCI.

Risk factor	Overall (n=123)	NRA group (n=50)	RA group RB group (n=63)	TA group (n = 10)	*p*-value
Age (n years old)	61.67±11.82	58.4±11.89	62.16±11.44	70.00±8.56	0.011
Male (n)	93	37	49	7	0.731
Basic disease (n)					
Heart failure	5	2	3	0	1.000
Previous myocardial infarction	12	4	8	0	0.815
Previous PCI	7	4	3	0	0.441
High blood pressure	60	22	32	6	0.380
Diabetes	36	13	19	4	0.510
Cerebrovascular disease	18	8	8	2	0.723
Smoking (n)	66	28	34	4	0.666
Drinking (n)	50	22	24	4	0.531
NT-proBNP (n)					0.962
≥900 pg/mL	17	7	8	2	
<900 pg/mL	106	43	55	8	
LDL-C (n)					0.537
≥3.37 mmol/L	38	17	18	3	
<3.37 mmol/L	85	33	45	7	
Blood potassium (n)					0.369
≥3.5 mmol/L	98	42	49	7	
<3.5 mmol/L	25	8	14	3	
Infarct site (n)					
Anterior myocardial infarction	55	37	10	8	<0.001
Lower wall myocardial infarction	67	12	53	2	<0.001
Lateral myocardial infarction	1	1	0	0	0.407
TIMI (n)					
Level 0	103	34	60	9	<0.001
Level 1	11	8	3	0	0.051
level 2	8	7	0	1	0.008
Level 3	1	1	0	0	0.407
Opening blood vessel time (n)					
≤6 h	91	38	44	9	0.683
>6 h	32	12	19	1	
Number of implanted stents (n)					
1	110	46	55	9	0.443
2	13	4	8	1	
LVEF (n, %)	55.43±7.43	54.64±7.91	56.93±6.66	50.33±7.16	0.321
Left ventricle (n, mm)	32.82±4.30	32.7±4.05	32.47±4.31	35.67±5.07	0.797
Left atrium (n, mm)	45.52±4.33	45.02±4.79	45.63±3.72	47.67±5.02	0.275

Note: RA: reperfusion arrhythmia; NRA: non-reperfusion arrhythmia; TA: tachyarrhythmia; NT-proBNP: N-terminal B-brain natriuretic peptide; LVEF: left cardiac ejection fraction.

Table II. Multivariate analysis of risk factors for RA in patients with STEMI after emergency PCI.

Influencing factor	B	S.E.	f	p	95% CI
Age (>57 years old)	0.937	0.465	1	0.044	[1.027, 6.351]
Lower wall myocardial infarction	2.162	0.459	1	0	[3.531, 21.360]
TIMI level 0	1.712	0.675	1	0.011	[1.474, 20.805]

Table III. Univariate analysis of risk factors for RB in patients with STEMI after emergency PCI.

Project	Overall (n=123)	NRB group (n=60)	RB group (n=63)	p-value
Age (n, year old)	61.67±11.82	60.33±12.14	62.16±11.44	0.228
Male (n)	93	44	49	0.468
Basic disease (n)				
Heart failure	5	2	3	0.688
Previous myocardial infarction	12	4	8	0.260
Previous PCI	7	4	3	0.947
High blood pressure	60	28	32	0.647
diabetes	36	17	19	0.824
Cerebrovascular disease	18	10	8	0.534
Smoking (n)	66	32	34	0.944
Drinking (n)	50	26	24	0.554
NT-proBNP (n)				0.721
≥900 pg/mL	17	9	8	
<900 pg/mL	106	51	55	
LDL-C (n)				0.568
≥3.37 mmol/L	38	20	18	
<3.37 mmol/L	85	40	45	
Blood potassium (n)				0.592
≥3.5 mmol/L	98	49	49	
<3.5 mmol/L	25	11	14	
TIMI (n)				
Level 0	103	43	60	0.001
Level 1	20	8	3	0.096
level 2	8	8	0	0.002
Level 3	1	1	0	0.492
Opening blood vessel time (n)				
≤6 h	91	47	44	0.283
>6 h	32	13	19	
Number of implanted stents (n)				
1	110	55	55	0.431
2	13	5	8	
LVEF (n, %)	55.43±7.43	53.98±7.89	56.93±6.66	0.030
Left ventricle (n, mm)	32.82±4.30	33.15±4.31	32.47±4.31	0.400
Left atrium (n, mm)	45.52±4.33	45.42±4.88	45.63±3.72	0.785

Note: RB: reperfusion slow arrhythmia; NRB: non-reperfusion slow arrhythmia; NT-proBNP: N-terminal B-type brain natriuretic peptide; LVEF: left cardiac ejection fraction

not statistically significant. Significance (p>0.05; Table IV).

Relationship Between Criminal Blood Vessels and Slow Arrhythmia

The results of CAG in 123 patients with STEMI showed that the culprit blood vessels were 57 cases of left anterior descending arteries, 8 cases of left circumflex artery and 58 cases of right coronary artery. When the criminal blood vessels were left anterior descending, the occurrence of slow arrhythmia in the RB group was significantly lower than that in the NRB group (p<0.05). When the criminal blood vessels were right coronary arteries, the slow arrhythmia occurred in the RB group. The rate was significantly higher than that of the NRB group, and the difference was statistically significant (p<0.05). When the left

Table IV. Effect of tirofiban on RB in patients with STEMI after emergency PCI.

Arrhythmia type	Tirofiban (n=104)	Non-tirofiban group (n=19)	χ^2	p
RB	55	8	0.747	0.459
NRB	49	11		

Note: RB: reperfusion slow arrhythmia; NRB: non-reperfusion slow arrhythmia.

Table V. The effect of criminal blood vessels on slow arrhythmias [n (%)].

Criminal blood vessel	RB group	NRB group	χ^2	p
Left anterior descending (n=57)	8 (14.03)	49 (85.97)	58.786	<0.001
Left circumflex (n=8)	5 (62.50)	3 (37.50)	0.087	0.768
Right coronary artery (n=58)	50 (86.21)	8 (13.79)	53.773	<0.001

Note: RB: reperfusion slow arrhythmia; NRB: non-reperfusion slow arrhythmia.

Table VI. Multivariate analysis of risk factors for RB in patients with STEMI after emergency PCI.

Risk factor	B	S.E.	p	f	95% CI
LEVF	0.055	0.036	0.131	1	[0.984, 1.135]
Right coronary artery	3.162	0.541	<0.001	1	[8.182, 68.157]
TIMI level 0	2.117	0.799	0.008	1	[1.736, 39.736]

Note: RB: reperfusion slow arrhythmia; NRB: non-reperfusion slow arrhythmia.

circumflex artery was a criminal blood vessel, there was no significant difference in RB between the two groups (p>0.05), as shown in Table V.

Multivariate Analysis of Risk Factors for RB in Patients with STEMI After Emergency PCI

LVEF, right coronary arteries and TIMI blood flow level 0 were entered into the logistic regression equation for calculation. Right coronary arteries and TIMI blood flow level 0 were independent risk factors for RB in patients with STEMI after emergency PCI (Table VI).

Discussion

Clinically, RA is often used as one of the indicators for judging coronary recanalization, but RA is also one of the common complications of PCI in patients with STEMI. In this study, the prevalence of RA in emergency PCI in 123 patients with STEMI was 59.35%, and the prevalence of RB was 51.22%. The mechanism of RA may be due to the large amount of calcium ions entering the cell, causing calcium overload, which in turn affects the myocardial electromechanical contraction coupling, leading to myocardial contraction of myocardial ischemia, and microvascular sputum, which leads to RA. In addition, some scholars have reported that it may also be related to increased free radicals[9,10], neutrophil activation[11] and so on.

The results of this study showed that age, inferior myocardial infarction, and TIMI blood flow level 0 were independent risk factors for RA after emergency PCI in STEMI patients, and the continuous variable age was 57 years old by ROC curve analysis. When the patient was >57 years old, the occurrence of RA was greater than that of patients ≤57 years old, similar to that reported by Niccoli et al[4]. Inferior myocardial infarction mostly comes from right coronary artery infarction. Bonnemeier et al[12] did not correlate with the occurrence of RA after PCI in patients with acute myocardial infarction and TIMI grade 2 and

grade 3 blood flow, while Demidova et al[13] and Lee et al[14] reported infarct blood vessels. Flow TIMI level 0 can increase the occurrence of RA, severe ischemia can lead to significant changes in electrophysiology, recovery inhomogeneity during reperfusion, and easy to cause RA. Therefore, persistent myocardial ischemia may be one of the causes of RA after myocardial infarction, consistent with the report. However, this study found that there was no significant difference in the occurrence of RA in the vessels opened within 6 hours and the prevalence of RA in the vascular patients who had opened the culprit blood vessels for more than 6 hours, which was inconsistent with previous reports[9,12]. The possible cause is the inclusion of population. The number and nature are inconsistent and further research is needed.

The results of this study show that criminal blood vessels have different RA types. When the left anterior descending artery is a culprit blood vessel, rapid arrhythmia is dominant. The possible cause is a large amount of calcium influxes in the background current of the four automatic depolarizations of Purkinje fiber cells and cardiomyocytes during acute ischemia. Thus, self-discipline leads to rapid arrhythmia[12]; when the culprit is a right coronary artery, the prevalence of slow arrhythmia in the RB group is significantly higher than that of other arteries, which may be related to the difference in coronary blood supply. Because right coronary artery infarction can lead to insufficient blood supply to the sinus node or atrioventricular node, or increased parasympathetic excitability due to stimulation, sympathetic nerve excitability is reduced[15]; when the culprit blood vessel is left circumflex, it does not increase the risk of RB. Tirofiban is an inhibitor of platelet glycoprotein IIB/III a receptor. The main mechanism of tirofiban is to prevent fibrinogen from binding with platelet glycoprotein IIB/III a receptor, which blocks and crosslinking and platelet aggregation, and to achieve the effect of inhibiting thrombosis. At present, a large number of studies have fully confirmed the role of tirofiban in antiplatelet therapy, which can significantly improve the clinical outcomes of ACS patients. In addition, tirofiban can also inhibit the release of angiotensin and inflammatory factors during platelet activation and improve microcirculation. Previous evidence[6] has shown that tirofiban can be used in patients with a high risk of myocardial infarction within 3-4 days after the onset of acute angina symptoms, including those who may receive early PCI treatment, to reduce the occurrence of major cardiovascular events. However, the most common adverse reaction during tirofiban treatment was mild mucosal hemorrhage, while the incidence of severe intracranial hemorrhage was less than 0.1%. In addition, when combined with heparin, the common adverse reactions include thrombocytopenia, nausea, fever and headache. Our present study has shown that tirofiban bolus intracoronary administration does not increase the risk of emergency PCI for RB in patients with STEMI, but rather reduces slow blood flow without reflow during surgery[6], so it can be safely used in STEMI patients.

Conclusions

The novelty of this study was to analyze the risk factors of reperfusion bradyarrhythmia through acute percutaneous coronary intervention in patients with STEMI. At the same time, it was pointed out that tirofiban was not related to the risk of reperfusion bradyarrhythmia in STEMI patients treated with emergency PCI, indicating the clinical safety of tirofiban treatment. In summary, in patients with STEMI who underwent PCI, age (>57 years), inferior myocardial infarction, and TIMI blood flow level 0 were independent risk factors for RA after emergency PCI in STEMI patients; the culprit vessels were right coronary artery and TIMI blood. Level 0 is an independent risk factor for RB in patients with STEMI after emergency PCI, and tirofiban does not increase the risk of emergency PCI in patients with STEMI.

Conflict of Interest

The Authors declare that they have no conflict of interests.

References

1) Puymirat E, Simon T, Cayla G, Cottin Y, Elbaz M, Coste P, Lemesle G, Motreff P, Popovic B, Khalife K, Labeque JN, Perret T, Le Ray C, Orion L, Jouve B, Blanchard D, Peycher P, Silvain J, Steg PG, Goldstein P, Gueret P, Belle L, Aissaoui N, Ferrieres J, Schiele F, Danchin N. Acute Myocardial Infarction: Changes in Patient Characteristics, Management, and 6-Month Outcomes Over a Period of 20 Years in the FAST-MI Program (French Registry of Acute ST-Elevation or Non-ST-Elevation Myocardial Infarction) 1995 to 2015. Circulation 2017; 136: 1908-1919.

2) Durak I, Kudaiberdieva G, Gorenek B. Prognostic implications of arrhythmias during primary percutaneous coronary interventions for ST-elevation myocardial infraction. Expert Rev Cardiovasc Ther 2015; 13: 85-94.
3) Montalescot G, van T HA, Lapostolle F, Silvain J, Lassen JF, Bolognese L, Cantor WJ, Cequier A, Chettibi M, Goodman SG, Hammett CJ, Huber K, Janzon M, Merkely B, Storey RF, Zeymer U, Stibbe O, Ecollan P, Heutz WM, Swahn E, Collet JP, Willems FF, Baradat C, Licour M, Tsatsaris A, Vicaut E, Hamm CW. Prehospital ticagrelor in ST-segment elevation myocardial infarction. N Engl J Med 2014; 371: 1016-1027.
4) Niccoli G, Montone RA, Ibanez B, Thiele H, Crea F, Heusch G, Bulluck H, Hausenloy DJ, Berry C, Stiermaier T, Camici PG, Eitel I. Optimized Treatment of ST-Elevation Myocardial Infarction. Circ Res 2019; 125: 245-258.
5) Zhu K, Fu Q, Zhang N, Huang YJ, Zhang Q. Pre-PCI medication using clopidogrel and ticagrelor in the treatment of patients with acute myocardial infarction. Eur Rev Med Pharmacol Sci 2015; 19: 4636-4641.
6) Akpek M, Sahin O, Sarli B, Baktir AO, Saglam H, Urkmez S, Ergin A, Oguzhan A, Arinc H, Kaya MG. Acute Effects of Intracoronary Tirofiban on No-Reflow Phenomena in Patients With ST-Segment Elevated Myocardial Infarction Undergoing Primary Percutaneous Coronary Intervention. Angiology 2015; 66: 560-567.
7) Ibanez B, James S, Agewall S, Antunes MJ, Bucciarelli-Ducci C, Bueno H, Caforio A, Crea F, Goudevenos JA, Halvorsen S, Hindricks G, Kastrati A, Lenzen MJ, Prescott E, Roffi M, Valgimigli M, Varenhorst C, Vranckx P, Widimsky P. 2017 ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation: The Task Force for the management of acute myocardial infarction in patients presenting with ST-segment elevation of the European Society of Cardiology (ESC). Eur Heart J 2018; 39: 119-177.
8) Qi Q, Niu J, Chen T, Yin H, Wang T, Jiang Z. Intracoronary Nicorandil and the Prevention of the No-Reflow Phenomenon During Primary Percutaneous Coronary Intervention in Patients with Acute ST-Segment Elevation Myocardial Infarction. Med Sci Monit 2018; 24: 2767-2776.
9) van der Weg K, Kuijt WJ, Bekkers S, Tijssen J, Green CL, Smulders MW, Lemmert ME, Krucoff MW, Gorgels A. Bursts of reperfusion arrhythmias occur independently of area at risk size and are the first marker of reperfusion injury. Int J Cardiol 2018; 271: 240-246.
10) Kobayashi T, Watanabe Y, Saito Y, Fujioka D, Nakamura T, Obata JE, Kitta Y, Yano T, Kawabata K, Watanabe K, Mishina H, Ito S, Kugiyama K. Mice lacking the glutamate-cysteine ligase modifier subunit are susceptible to myocardial ischaemia-reperfusion injury. Cardiovasc Res 2010; 85: 785-795.
11) Vandervelde S, van Amerongen MJ, Tio RA, Petersen AH, van Luyn MJ, Harmsen MC. Increased inflammatory response and neovascularization in reperfused vs. non-reperfused murine myocardial infarction. Cardiovasc Pathol 2006; 15: 83-90.
12) Bonnemeier H, Ortak J, Wiegand UK, Eberhardt F, Bode F, Schunkert H, Katus HA, Richardt G. Accelerated idioventricular rhythm in the post-thrombolytic era: incidence, prognostic implications, and modulating mechanisms after direct percutaneous coronary intervention. Ann Noninvasive Electrocardiol 2005; 10: 179-187.
13) Demidova MM, Carlson J, Erlinge D, Platonov PG. Predictors of ventricular fibrillation at reperfusion in patients with acute ST-elevation myocardial infarction treated by primary percutaneous coronary intervention. Am J Cardiol 2015; 115: 417-422.
14) Lee SN, Hwang YM, Kim GH, Kim JH, Yoo KD, Kim CM, Moon KW. Primary percutaneous coronary intervention ameliorates complete atrioventricular block complicating acute inferior myocardial infarction. Clin Interv Aging 2014; 9: 2027-2031.
15) Kalra S, Bhatt H, Kirtane AJ. Stenting in Primary Percutaneous Coronary Intervention for Acute ST-Segment Elevation Myocardial Infarction. Methodist Debakey Cardiovasc J 2018; 14: 14-22.