IJC Heart & Vasculature 23 (2019) 100357

Contents lists available at ScienceDirect

IJC Heart & Vasculature

journal homepage: http://www.journals.elsevier.com/ijc-heart-and-vasculature

# Clinical and procedural predictors of suboptimal myocardial reperfusion in primary percutaneous coronary intervention

Adel H. Mahmoud *, Nasser M. Taha, Khaled Baraka, Mohamed Ashraf, Sayed Shehata

*Cardiology and Critical Care Departments, Al Minia & Cairo Universities, Egypt*

ARTICLE INFO

*Article history:*
Received 1 February 2019
Received in revised form 10 March 2019
Accepted 25 March 2019
Available online 19 April 2019

*Keywords:*
Myocardial perfusion
Predictors
Primary PCI

ABSTRACT

*Background:* Suboptimal myocardial perfusion in primary PCI is associated with increased infarct size, left ventricular (LV) dysfunction and higher mortality rates as compared as those with optimal myocardial perfusion. We identified clinical and procedural predictors of suboptimal myocardial reperfusion as judged by myocardial plush grade (MBG) in primary PCI.

*Methods and Results:* 100 patients with acute STEMI who underwent primary PCI were prospectively subjected to clinical, ECG, laboratory and angiographic evaluation. Patients were classified into: Optimal myocardial reperfusion group: (n=73) who had final MBG=3. Suboptimal myocardial reperfusion group: (n=27) who had persistent final MBG ≤ 2. Suboptimal myocardial reperfusion group had statistically significant little history of angina prior to MI 5 (18.5%) vs 44 (60.3%), little current aspirin intake 6(22%) vs 38 (52% ), increased blood sugar on admission (240 ± 101 mg/dl vs 171 ± 72 mg/dl), increased total leucocytic count on admission (12.1 ± 3.6 vs 10.2 ± 3.3) 103/mm3, longer reperfusion time (6.1 ± 2.8 vs 4.3 ± 2.1 h ), higher thrombus burden 12 (44.4 % ) vs 13 (17.8 %), higher predilatation pressure (16 ± 2.3 vs 14 ± 1.8 ATM), repeated balloon inflation during predilatation 24 (92.3 % ) vs 46 (69.7%) as compared optimal myocardial reperfusion group, ($P < 0.05$ for all).

*Conclusion:* Longer reperfusion time, repeated balloon inflations, high predilatation pressure> 15 ATM , high thrombus burden, neither history of angina nor aspirin intake prior to AMI, high total leucocytic count > 10103/mm3 and high blood glucose level > 160mg/dl were predictors for persistent suboptimal myocardial reperfusion in primary PCI.



## 1. Introduction

Primary percutaneous coronary intervention (PCI) is the preferred reperfusion strategy for treating acute ST-segment elevation myocardial infarction (STEMI). The main goals are to restore epicardial infarct-related artery patency and achieve microvascular reperfusion as early as possible, thus limiting the extent of irreversibly injured (necrotic) myocardium [1]. Adequate myocardial perfusion after 1$^{ry}$ PCI was associated with better clinical outcomes, whereas suboptimal myocardial perfusion was associated with increased infarct size, an increased rate of left ventricular (LV) dysfunction and higher mortality rates. Restoration of normal epicardial blood flow (TIMI grade 3) does not mean optimal reperfusion at the microcirculation level. Myocardial blush grade (MBG) had been well validated as an angiographic technique to assess myocardial perfusion in patients with STEMI. MBG correlates with the more accurate method of evaluating myocardial perfusion (magnetic resonance imaging) and enables the immediate evaluation of microvascular patency in the catheterization laboratory [2].

## 2. Aim of this work

Aim of this work was to identify clinical and procedural predictors of sub-optimal myocardial perfusion judged by MBG in patients with acute STEMI who undergo primary PCI.

## 3. Patient and methods

This is prospective non randomized study that included 100 consecutive patients with acute STEMI who were subjected to primary PCI. The culprit vessel was only subjected to intervention. Patients with severe left main or multivessel coronary artery disease who in need for urgent surgical revascularization and patients with saphenous vein grafts lesions were excluded from the study. All patients provided written informed consent for the use of clinical, procedural, and follow-up data for research. The study procedures were in accordance with the Declaration of Helsinki and the institutional ethics committee of the Minia University approved the study protocol.

* Corresponding author at: Cardiology Department, El Minia University Hospital, 195 Kornish El Nile Street, El Minia, Egypt.
*E-mail addresses:* adel.mahmoud@minia.edu.eg, dr_adelhamdy@yahoo.com (A.H. Mahmoud).

https://doi.org/10.1016/j.ijcha.2019.100357

All patients were subjected to the following:

1- Thorough clinical evaluation including history of diabetes mellitus, systemic hypertension, smoking, dyslipidemia and family history of premature CAD, previous myocardial infarction, angina prior to MI and history of previous medication.
2- Twelve lead surface ECG: For evidence and site of myocardial infarction and calculation of ST segment resolution (STR) as sum of ST elevation at ECG 90 min after 1$^{ry}$ PCI/sum of ST elevation at initial ECG, STR was classified according to the Schröder classification as complete (70% to 100%), partial (30% to 69%), or absent (<30%) [3].
3- Laboratory investigations including on admission blood glucose level, total leucocytic count (TLC), blood urea nitrogen (BUN), serum creatinine, hemoglobin, and platelets.
4- Interventional variables: primary PCI was performed by experienced operators on call according to standard protocols. Unless contra-indicated, all patients received; aspirin 300 mg, nitroglycerin infusion, intravenous bolus of 5000–10,000 units of heparin, clopidogrel (loaded with 600 mg followed by 150 mg per day) and Glycoprotein IIb/IIIa receptor antagonist infusion which is the strategy of our centre according to guidelines at time of study. Other anti-ischemic drugs like B-blockers, anti-arrhythmics, inotropics and vasopressors were given when indicated. Culprit vessels were identified by the operator according to leads involved at ECG. Selection of guiding catheter and guide wire, usage of catheter aspiration device, predilatation, maximum balloon inflation pressure depending upon balloon type and size, stenting for residual lesions, and post dilatation were determined according to operator discretion. Experienced interventional cardiologist who was unaware of patient's clinical data carefully assessed a set of parameters for each angiogram:

A. **Thrombus burden**: Thrombus grade was classified from grade 0 to grade 5 then thrombus burden had been reclassified into absent, low, moderate or high as described [4].
B. **TIMI flow grading** from grade 0 to grade 3 at basic angiography and at end of procedure as described [5].
C. **Myocardial blush grading (MBG)** according to the dye density score of the myocardial territory subtended by the infarct related vessel as proposed by van't Hof et al. [5] from grade 0 to grade 3 as follows:

MBG = 0: No contrast density or abnormal persistence of contrast medium. MBG = 1: Minimal myocardial blush or contrast density.

MBG = 2: Moderate myocardial blush or contrast density but less than that obtained during angiography of a contralateral or ipsilateral non-infarct related coronary artery.

MBG = 3: Normal myocardial blush or contrast density, comparable with that obtained during angiography of a contralateral or ipsilateral non-infarction related coronary artery.

### 3.1. Statistical analysis

The Statistical Package for Social Sciences (SPSS) software version 20 was used for data entry and analysis. Categorical variables were expressed as frequency tables and compared with chi square statistics test. While continuous variables were expressed as mean and standard deviation (SD) and were compared using *t*-test. A probability level of $P \leq 0.05$ was chosen to be significant. Multiple regression analysis was used to determine power of the predictors. Roc curve was used to determine accuracy and cut off value of continuous variables.

## 4. Results

Patients were divided into two groups according to final myocardial blush grade (MBG) into:

Optimal reperfusion group, (n = 73) who had final MBG = 3.

Suboptimal reperfusion group, (n = 27) who had persistent final MBG ≤ 2.

### 4.1. Clinical characteristics

Suboptimal reperfusion group had statistically significant fewer number of patients with both history of angina prior to MI 5(18.5%) vs 44(60%) and current aspirin intake 6 (22%) vs 38 (52%) as compared to optimal reperfusion group (P = 0.001 and P = 0.008), respectively. Otherwise, there was no statistically significant difference between both groups regarding their clinical data as shown in (Table 1).

### 4.2. ECG findings

There was no statistically significant difference between both groups regarding site of acute MI (P = 0.45). Suboptimal reperfusion group had statistically significant less ST segment resolution (STR) as compared to optimal reperfusion group, (P = 0.001), (Table 2).

**Table 1**
Comparison between optimal and suboptimal reperfusion groups regarding their clinical data.

| | Optimal reperfusion (n = 73) | Suboptimal reperfusion (n = 27) | P value |
|---|---|---|---|
| Age, (Ys) (mean ± SD) | 54 ± 10 | 56 ± 13 | 0.27 |
| Male, n (%) | 63 (86%) | 19 (70%) | 0.07 |
| Smoker, n (%) | 44 (60%) | 14 (52%) | 0.45 |
| HTN, n (%) | 24 (33%) | 14 (52%) | 0.08 |
| Family history of CAD, n (%) | 12 (16%) | 9 (33%) | 0.07 |
| Dyslipidemia, n (%) | 17 (23%) | 7 (26%) | 0.78 |
| DM, n (%) | 22 (30%) | 14 (52%) | 0.06 |
| Previous MI, n (%) | 6 (8%) | 3 (11%) | 0.65 |
| Angina prior to MI, n (%) | 44 (60%) | 5 (18.5%) | **0.001** |
| Current therapies: | | | |
| ASA, n (%) | 38(52%) | 6 (22%) | **0.008** |
| BB, n (%) | 16 (22%) | 6 (22%) | 0.97 |
| ACEI, n (%) | 13 (17%) | 8 (30%) | 0.20 |
| Statins, n (%) | 19 (26%) | 9 (33%) | 0.47 |

P value < 0.05 was expressed in bold.

**Table 2**
Comparison between optimal and suboptimal reperfusion groups regarding their ECG and procedural data.

| | Optimal reperfusion (n = 73) | Suboptimal reperfusion (n = 27) | P value |
|---|---|---|---|
| Site of acute STEMI | | | |
| Anteroseptal, n (%) | 13 (18%) | 4 (15%) | |
| Anterior, n (%) | 16 (22%) | 9 (33%) | |
| Extensive anterior, n (%) | 8 (11%) | 5 (18.5%) | 0.45 |
| Inferior, n (%) | 15 (21%) | 5 (18.5%) | |
| Inferior + lateral, n (%) | 9 (12%) | 3 (11%) | |
| Inferior + RV, n (%) | 12 (16%) | 1 (4%) | |
| Reperfusion time (hours) | 4.3 ± 2.1 | 6.1 ± 2.8 | **0.001** |
| Predilatation | 65 (89%) | 26 (97%) | 0.24 |
| Length of balloon, (mm) | 20 ± 2 | 20 ± 2 | 0.23 |
| Size of balloon, (mm) | 2.4 ± 0.5 | 2.8 ± 1.9 | 0.15 |
| Maximum pressure, (ATM) | 14.7 ± 1.8 | 16.1 ± 2.3 | **0.002** |
| Repeated dilatation, (> twice) | 46 (70%) | 24 (92%) | **0.022** |
| Stenting | 68 (93%) | 23 (85% | 0.22 |
| Length of stents, (mm) | 29 ± 9 | 30 ± 11 | 0.53 |
| Size of stents, (mm) | 3.2 ± 0.4 | 3.2 ± 0.3 | 0.72 |
| Maximum deployment pressure, (ATM) | 15.8 ± 1.8 | 15.8 ± 1.9 | 0.91 |
| Post-dilatation | 19 (28%) | 11 (48%) | **0.09** |

### 4.3. Laboratory data on admission

Suboptimal reperfusion group had statistically significant increased mean blood glucose level on admission (240 ± 101 mg/dl vs 171 ± 72 mg/dl) and increased mean TLC on admission (12.1 ± 3.6 vs 10.3 ± 3.3) $10^3/mm^3$ as compared to optimal reperfusion group, (P = 0.001 and P = 0.02), respectively. While there is no statistically significant difference between both groups regarding BUN, serum creatinine, hemoglobin or platelets on admission (P > 0.05 for all).

### 4.4. Angiographic data

There was no statistically significant difference between both groups regarding infarct-related artery, site of the occlusion at IRA or type of the occlusion (P = 0.20, P = 0.37, and P = 0.31), respectively. Suboptimal reperfusion group had statistically significant higher thrombus burden as compared to optimal reperfusion group (P = 0.01).

### 4.5. Procedural data

suboptimal reperfusion group had statistically significant longer reperfusion time (6.1 ± 2.8 h vs 4.3 ± 2.1 h), increased predilatation maximum balloon inflation pressure (16.1 ± 2.3 vs 14.7 ± 1.8 ATM), more repeated balloon inflations (>twice) during predilatation [24 (92%) vs 46 (70%)] as compared to optimal reperfusion group P = 0.001, P = 0.002, and P = 0.022, respectively. While there was no statistically significant difference between both groups regarding predilatation, length of balloon, size of balloon, stent length, stent size, maximum deployment pressure, and post-dilatation (P > 0.05 for all), (Table 2).

On admission, blood glucose level, TLC, maximum predilatation pressure and reperfusion time had 70%, 65%, 69% and 70% accuracy, respectively as predictors for suboptimal reperfusion. Suboptimal reperfusion group had statistically significant more patients with blood glucose level on admission >160 mg/dl [20(74%) vs 29(40%) [P = 0.002], statistically significant more patients with TLC > 10 ($10^3/mm^3$) [21(78%) vs 34(47%) [P = 0.005], statistically significant more patients with reperfusion time >4 h [19 (70%) vs 29(40%) [P = 0.006] and statistically significant more patients with maximum predilatation pressure >15 ATM [19(73%) vs 30(46%) compared with optimal reperfusion group, P = 0.020, (Table 3). The predictors of suboptimal reperfusion were arranged according to their power which was determined according to its odd ratio as shown in (Table 4). Aspiration device was used in 12 patients according to operator's discretion, 10 (83%) of them had final MBG = 3 and 2 (17%) had final MBG ≤ 2.

### 4.6. Relation between final MBG and final TIMI flow grade

TIMI grade 3 was achieved in 84 patients, 11 patients of them (14%) had suboptimal reperfusion (MBG ≤ 2), and 73 patients had optimal reperfusion (MBG = 3). Agreement between final TIMI flow and final MBG was 89%.

**Table 4**
Predictors of suboptimal myocardial reperfusion according to its odd ratio.

| Predictors | Odd ratio |
|---|---|
| Repeated balloon inflation during predilatation | 8.20 |
| High thrombus burden | 5.01 |
| No history of angina prior to MI | 4.38 |
| TLC on admission >10 ($10^3$/ $mm^3$) | 3.41 |
| Blood glucose level on admission >160 mg/ dl | 3.04 |
| Longer reperfusion time (4 h) | 2.68 |
| No current ASA therapy | 2.29 |
| Predilatation at pressure >15 ATM | 2.23 |

### 4.7. Short term clinical outcome

#### 4.7.1. In-hospital mortality and major cardiac events (MACE)

Suboptimal reperfusion group had statistically significant higher in-hospital total MACE (death, re-infarction, HF and stroke) 10 (37%) vs 6 (8%), P = 0.003 as compared with optimal reperfusion group.

#### 4.7.2. Duration of hospital stay

Suboptimal reperfusion group had statistically significant longer duration of hospital stay (8 ± 3 vs 3 ± 1 days), P = 0.02 as compared to optimal reperfusion group.

#### 4.7.3. Echocardiographic data

Echocardiographic data was available in 99 patients as one patient died before echocardiography was done. Suboptimal reperfusion group had statistically significant lower mean Ejection fraction (45 ± 5% vs 55 ± 7%, P = 0.001).

## 5. Discussion

### 5.1. Principal findings

Our study revealed that incidence of persistent suboptimal myocardial perfusion was 27% and its predictors arranged according to their power were, repeated balloon inflation during predilatation, high thrombus burden, no history of angina prior to MI, high TLC, high blood glucose level, longer reperfusion time, no current aspirin intake before MI and high predilatation pressure. Also, our study revealed that in spite of TIMI grade 3 was achieved in 84 patients, 11 patients of them (14%) had suboptimal reperfusion (MBG ≤ 2). Higher in-hospital total MACE (death, re-infarction, HF and stroke) and longer duration of hospital stay were observed in persistent suboptimal reperfusion group.

Suboptimal myocardial reperfusion during primary PCI for STEMI patients was considered to be a negative hallmark and predicting the occurrence of worse clinical outcome. Therefore, attention has shifted from epicardial artery patency to the status of the coronary microvascular and myocardial reperfusion [6]. In agreement with our results are those of Marlos et al., who evaluated 99 primary PCI procedures, they found that TIMI 3 flow was restored at procedure's end in 91 patients in spite of MBG =3 was achieved in only 69 patients (69.7%) and incidence of MBG ≤2 was 30.3% [2].

Prodromal angina pectoris (AP) had cardioprotective effect by ischemic preconditioning which delays infarct progression during the early

**Table 3**
Cut off values for blood glucose level, total leucocytic count, reperfusion time and predilatation pressure.

| | Sensitivity | Specificity | Optimal reperfusion (n = 73) | Suboptimal reperfusion (n = 27) | P value |
|---|---|---|---|---|---|
| Blood glucose > 160 | 74% | 62% | 29 (40%) | 20 (74%) | **0.002** |
| TLC on admission > 10 | 63% | 63% | 34 (47%) | 21 (78%) | **0.005** |
| Reperfusion time > 4 h | 70% | 61% | 29 (40%) | 19 (70%) | **0.006** |
| Predilatation pressure > 15 *ATM* | 73% | 62% | 30 (46%) | 19 (73%) | **0.020** |

hours of AMI and extends the window of time for reperfusion therapy. Prodromal AP associated with smaller infarct size, improved left ventricular function and favorable short- and long-term prognoses after AMI [7]. Our study revealed that suboptimal reperfusion group had statistically significant fewer patients with history of angina prior to MI compared with optimal reperfusion group. In agreement with our results are those of Zhang et al. who found that prodromal angina (PA) associated with higher rates of overall procedural and of complete ST-segment resolution at 90 min after the procedure ($P = 0.001$) [8].

Platelets may be implicated in no-reflow through microvascular obstruction by platelet aggregates, release of platelet-derived vasoactive and chemotactic mediators. Patients with angiographic no-reflow had higher levels of TXA2 compared with patients without angiographic no-reflow independently of the Abciximab treatment [9]. Several reports in the thrombolytic era had suggested the correlation between previous aspirin treatment and smaller infarct [10]. Our study revealed that suboptimal reperfusion group compared with optimal reperfusion group had statistically significant fewer patients with current aspirin therapy.

Stress hyperglycemia in a setting of AMI increases the risk of malignant ventricular tachyarrhythmias as well as in-hospital mortality [11]. Acute hyperglycemia causes several unfavorable effects that contribute to the poor outcomes of patients with AMI. Oxidative stress, inflammation, apoptosis, endothelial dysfunction, hypercoagulability, platelet hyperactivity, impaired ischemic preconditioning, and impaired microcirculation which directly damage the ischemic myocardium, and may cause the no-reflow phenomenon during reperfusion [12]. Our result revealed that suboptimal reperfusion group had statistically significant increased blood glucose level on admission and more patient with blood glucose level > 160 mg/dl as compared with optimal reperfusion group. In agreement with our results, Iwakura et al. found that the no-reflow phenomenon was more often observed in the patients with hyperglycemia [13].

Our study revealed that suboptimal reperfusion group had statistically significant increased TLC on admission as compared to optimal reperfusion group. The pathologic mechanisms involving neutrophils were microvascular plugging, spasm, endothelial swelling, and inflammatory response by pro-inflammatory cytokines release, all of which eventually lead to impairment of microvascular perfusion during acute myocardial ischemia [14].

It was established that prolonged ischemia lead to edema of distal capillary beds, swelling of myocardial cells, neutrophil plugging and alterations of capillary integrity. Furthermore, delayed reperfusion can result in more organized intracoronary thrombus, which may increase the risk of distal embolization during primary PCI and reduce the likelihood of achieving myocardial perfusion [15]. Our result revealed that suboptimal reperfusion group had statistically significant longer reperfusion time compared to optimal reperfusion group. Cevat et al. studied 382 consecutive acute STEMI patients treated with primary PCI and found that suboptimal reperfusion group had statistically significant more patients with reperfusion time (≥4 h) (48.4%) and higher thrombotic burden as compared with optimal reperfusion group (26.3%), $P = 0.004$ [16].

Acute STEMI is characterized by thrombus formation which leads to occlusion of the infarct related artery (IRA). Angiographic evidence of thrombus in the IRA is associated with poor in-hospital outcomes, increased infarct size and higher long term mortality. While attempting to open the occlusion during PCI, it is more likely to dislodge the thrombus downstream causing distal embolization and microvascular obstruction and contribute to “no reflow phenomenon” [17]. Our study revealed that suboptimal reperfusion group had statistically significant higher thrombus burden as compared with optimal reperfusion group.

Macro-embolisation occurred in 14% of patients with STEMI treated with primary PCI and associated with worse TIMI flow in the infarct-related artery, worse myocardial reperfusion, and less complete ST-segment resolution, larger infarct size, worse left ventricular function and higher mortality. Also distal micro-embolisation with primary PCI may contribute to poor myocardial reperfusion. This had stimulated attempts to remove thrombus, prevent distal micro-embolisation. TAPAS study indicated that thrombus aspiration during primary PCI was associated with an improvement in survival at 1 year [18]. In our study Aspiration device was used during the procedure of 12 patients according to operator's discretion, 10 patients (83%) of them had optimal reperfusion (MBG = 3) and 2 patients (17%) had suboptimal reperfusion (MBG ≤ 2). In agreement to our result are those of Tone et al. who randomized 1071 STEMI patients to aspiration thrombectomy followed by stenting versus stenting alone. They found that final MBG 0 or 1 occurred in 17.1% in the thrombus-aspiration group and in 26.3% in the conventional-PCI group ($P < 0.001$) and complete STR was significantly higher with aspiration thrombectomy 56.6% vs 44.2% in the conventional-PCI group, ($P < 0.001$) [19].

It has been hypothesized that predilatation of unstable plaques in the context of unstable angina or myocardial infarction may facilitate embolisation of cholesterol and necrotic debris, also local activation of platelets and coagulation factors, liberation of clot-bound-thrombin due to mechanical disruption of thrombus which increase the likelihood of suboptimal reperfusion. Also predilatation can activate leukocytes and oxidative stress, aggravating the inflammatory response and myocardial reperfusion injury [20]. Our study revealed that suboptimal reperfusion group had statistically significant higher predilatation maximum balloon inflation pressure, more repeated balloon inflation (> twice) during predilatation and more patient with predilatation pressure > 15 ATM compared to optimal reperfusion group. In agreement with our results are those of Marlos et al., who found that suboptimal reperfusion group compared to optimal reperfusion group had statistically significant increased maximum dilation pressure ($15.5 \pm 3.9$ vs $13.5 \pm 4.2$ ATM), $P = 0.039$ [2].

### 5.2. Study limitation

Small number of patients in only two centers study.

## 6. Conclusion

Based on the results of this study, it can be concluded that repeated balloon inflation during predilatation, high thrombus burden, no history of angina prior to MI, high TLC, high blood glucose level, longer reperfusion time, no current aspirin therapy and high predilatation pressure were predictors for persistent suboptimal reperfusion in 1$^{ry}$ PCI.

### 6.1. Clinical implications

Prevention rather than treatment should be the way forward as treatment after no-reflow (suboptimal reperfusion) is established is unlikely to succeed so an efficient emergency system that guarantees the reduction in reperfusion time, careful history focused on angina prior to MI, aspirin therapy as well as the evaluation of laboratory data such as glucose level and TLC, and thrombus burden at basic angiography to determine patients at a risk for suboptimal reperfusion to take a measures to prevent its incidence. Lastly during primary PCI if possible avoidance of repeated predilatation and also avoidance of predilatation high pressure.

## Funding

This work was funded and sponsored by authors.

## Conflict of interest

None of the authors has any conflict of interest to report.

## References


[1] N. Giampaolo, K.K. Rajesh, C. Filippo, P.B. Adrian, No-reflow: again prevention is better than treatment, Eur. Heart J. 31 (2010) 2449–2455.

[2] R.F. Marlos, F. David, C. John, A. Jonathan, V.S. Guilherme, C.P. Emerson, A.E. MacArthur, M.W. James, Restoration of microcirculatory patency after myocardial infarction, Tex. Heart Inst. J. 39 (2012) 342–350.

[3] R. Schröder, R. Dissmann, T. Brüggemann, K. Wegscheider, T. Linderer, U. Tebbe, K.L. Neuhaus, Extent of early ST segment elevation resolution: a simple but strong predictor of outcome in patients with acute myocardial infarction, J. Am. Coll. Cardiol. 24 (2) (1994 Aug) 384–391.

[4] C.M. Gibson, J.A. de Lemos, S.A. Murphy, Combination therapy with Abciximab reduces angiographically evident thrombus in acute myocardial infarction: a TIMI 14 substudy, Circulation 103 (2001) 2550–2554.

[5] Arnoud W.J. van `t Hof, Aylee Liem, Harry Suryapranata, Jan C.A. Hoorntje, Menko-Jan de, Angiographic assessment of myocardial reperfusion in patients treated with primary angioplasty for acute myocardial infarction myocardial blush grade. Circulation. 1998;97:2302–2306.

[6] H. Zhang, N. Tian, H. Zuo-ying, F. Wang, L. Chen, Y. Zhang, S. Chen, Three hours continuous injection of adenosine improved left ventricular function and infarct size in patients with ST-segment elevation myocardial infarction, Chin. Med. J. 125 (2012) 1713–1719.

[7] T. Maruhashi, M. Ishihara, I. Inoue, T. Kawagoe, Y. Shimatani, S. Kurisu, Effect of prodromal angina pectoris on the infarct progression in patients with first ST-elevation acute myocardial infarction, Circ. J. 74 (2010) 1651–1657.

[8] Q. Zhang, R. Zhang, T. Zhu, Z. Yang, F. Ding, Z. Zhu, W. Shen, Impact of angina prior to acute ST-elevation myocardial infarction on short-term outcomes after primary percutaneous coronary intervention: results from the Shanghai Registry of Acute Coronary Syndrome (SRACE), Chin. Med. J. 125 (2012) 977–982.

[9] N. Giampaolo, G. Simona, R. Eleonora, S. Cristina, L. Andrea, P. Italo, M.L. Antonio, B. Francesco, R. Silvia, P. Fabio, M.B. Luigi, C. Filippo, Plasma levels of thromboxane A2 on admission are associated with no-reflow after primary percutaneous coronary intervention, Eur. Heart J. 29 (2008) 1843–1850.

[10] D. Freimark, S. Matetzky, J. Leor, V. Boyko, I.M. Barbash, S. Behar, H. Hod, Timing of aspirin administration as a determinant of survival of patients with acute myocardial infarction treated with thrombolysis, Am. J. Cardiol. 89 (2002) 381–385.

[11] M. Michal, K. Jacek, L. Radoslaw, Z. Teresa, S. Agnieszka, The prognostic value of different glucose abnormalities in patients with acute myocardial infarction treated invasively, Cardiovasc. Diabetol. 11 (2012) 78.

[12] I. Masaharu, Acute hyperglycemia in patients with acute myocardial infarction, Circ. J. 76 (2012) 563–571.

[13] K. Iwakura, H. Ito, M. Ikushima, Association between hyperglycemia and the no-reflow phenomenon in patients with acute myocardial infarction, J. Am. Coll. Cardiol. 41 (2003) 1–7.

[14] Y.L. Hye, H.K. Jeong, O.K. Byung, J.K. Yoon, S.A. Hyo, W.H. Mee, M.P. Kyoung, S.B. Young, W.G. Choong, J.R. Kun, Effect of aspiration thrombectomy on microvascular dysfunction in ST-segment elevation myocardial infarction with an elevated neutrophil count, Kor. Circ. J. 41 (2011) 68.

[15] P. Vector, M. Xavier, G. Susana, M. Patricia, G. Bruno, D. Rosa, No-reflow phenomenon in cardiac MRI: diagnosis and clinical implications, AJR 191 (2008) 73–79.

[16] U.T. Umesh, C. Stanley, H. Irfan, G. Michael, M. Mauro, S.G. Hitinder, Safety and efficacy of thrombectomy in patients undergoing primary percutaneous coronary intervention for Acute ST elevation MI: a Meta-Analysis of Randomized Controlled Trials, BMC Cardiovasc. Disord. 10 (2010) 10.

[17] K. Cevat, I. Akin, D. Cihan, C. Ali, O. Vecih, M. Soe, S. Kenan, M. Bulent, O. Nihal, E. Vedat, Clinical and procedural predictors of no-reflow phenomenon after primary percutaneous coronary interventions, Circ. J. 72 (2008) 716–721.

[18] Tone S, M D, Pieter J V, Iwan C, Gilles F H D, Bart J G L, Rutger L A, Gillian A J, Eng-Shiong T, Albert J H S, and Felix Z. Thrombus aspiration during primary percutaneous coronary intervention tone. N. Engl. J. Med. 2008; 358:557–67.

[19] H. Breton, J. Boschat, P. Commeau, P. Brunel, M. Gilard, C. Breut, O. Bar, P. Geslin, A. Tirouvanziam, L. Maillard, B. Moquet, P. Barragan, P. Dupouy, G. Grollier, J. Berland, P. Druelles, R. Rihani, B. Huret, C. Leclercq, M. Bedossa, Randomised comparison of coronary stenting with and without balloon predilatation in selected patients, for the Stent Without Balloon Predilation (SWIBAP), Heart J 86 (2001) 302–308.

[20] H.E. Rong, L.I. Hai-yan, Guo Li-jun, F. Zhang, Niu Jie, Y. Zhang, G. Wang, Z. Yang, Gao Wei, Predictive value of postprocedural leukocyte count on myocardial perfusion, left ventricular function and clinical outcomes in ST-elevated myocardial infarction after percutaneous coronary intervention, Chin. Med. J. 125 (2012) 1023–1029.