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Predictive factors of in hospital major adverse cardiac events and no reflow phenomenon in patients with ST elevation myocardial infarction undergoing primary percutaneous coronary intervention

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Abstract

Objectives: The study is designed to determine the relation between various clinical and laboratory variables and the occurrence of no reflow phenomenon or in hospital MACE (Cardiac death, myocardial infarction, stent thrombosis, or target vessel revascularization) in patients with STEMI undergoing primary PCI.

Background: The investigation of no-reflow phenomenon after primary percutaneous coronary intervention (PPCI) in patients with acute ST-segment–elevation myocardial infarction (STEMI) has therapeutic implications. Patients with no-reflow have more congestive heart failure early after myocardial infarction and demonstrate progressive left ventricular cavity dilatation in the convalescent stage of the infarction.

Methods: We studied prospectively 120 patients with STEMI presenting to Alexandria Main University Hospital (under umbrella of Stent for Life program) and International Cardiac Center (ICC) from April 2013 to October 2013, and eligible for PPCI according to European Society of Cardiology (ESC) guidelines.

Results: The incidence of no reflow was 13.2%, and in hospital MACE was 5%, with cardiac death as the predominant form of in hospital MACE. The group with no reflow and/or in hospital MACE showed significantly older age (62.29 ± 7.90 *vs*. 56.30 ± 10.34, p=0.014), longer pain to balloon time (15.90 ± 7.87 *vs*. 6.08 ± 3.82, p<0.001), higher levels of admission random plasma glucose (RPG), neutrophils/lymphocytes (N/L) ratio (8.19 ± 3.05 *vs*. 5.44 ± 3.53, p<0.001), and MPV (11.90 ± 2.09 *vs*. 8.58 ± 1.84, p<0.001).

Conclusion: Older patient age, longer pain to balloon time, admission hyperglycemia, higher admission N/L ratio and MPV are useful predictive factors for the occurrence of no reflow post PPCI, and/or in hospital MACE. Therefore strong attention should be paid to patients with one or more of these predictive factors, to protect them from the deleterious effects of no reflow, and avoid any of the in hospital MACE.

Introduction

The phenomenon of no-reflow is defined as inadequate myocardial perfusion through a given segment of the coronary circulation without angiographic evidence of mechanical vessel obstruction [1]. No-reflow has been documented in 30% of patients after thrombolysis or mechanical intervention for acute myocardial infarction [2]. No reflow implies abnormal tissue perfusion and persistent no-reflow is associated with higher incidence of congestive heart failure early after myocardial infarction and demonstrate progressive left ventricular cavity dilatation in the convalescent stage of the infarction [3].

Several key patho-physiological processes, usually in combination, are believed to be responsible for this phenomenon, including distal embolization of atherothrombotic debris, thrombus formation, and endothelial dysfunction of the distal arteriolar and capillary bed, including endothelial desquamation and microcirculatory vasospasm.

Aim of the work

The study is designed to determine the relation between various clinical and laboratory variables and the occurrence of no reflow phenomenon or in hospital MACE (Cardiac death, myocardial infarction, stent thrombosis, or target vessel revascularization) in patients with STEMI undergoing primary PCI.

Methods

The study was conducted on 120 patients with STEMI presenting to Alexandria Main University Hospital (under umbrella of Stent For Life program) and International Cardiac Center (ICC) from April 2013 to October 2013, and eligible for PPCI according to European Society of Cardiology (ESC) guidelines. Informed consent taken from patients. Thorough history taking with special emphasis on risk factors (Age, gender, diabetes, hypertension, smoking, dyslipidemia, family history), history of acute coronary syndromes (ACS) and revascularization, Pain to balloon time, and the presence of pre-infarction angina. Complete clinical examination was done. Admission laboratory investigation

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included: Complete blood count (CBC) (including mean platelet volume [MPV] and neutrophils/lymphocytes ratio), and random plasma glucose level. All patients had 12 lead electrocardiogram (ECG). The results of the coronary angiography indicating the infarct related artery (IRA), initial TIMI flow in the IRA, and the type of stent used in the PPCI were recorded. The patients were studied according to the presence of various clinical and laboratory variables (age, gender, absence of pre-infarction angina, pain to balloon time, location of the infarction, admission random plasma glucose level and CBC including neutrophils/lymphocytes ratio and MPV, and initial TIMI flow in the IRA), the final TIMI flow after the primary PCI, and the incidence of in hospital MACE [4].

Results

The patients are divided into two groups according to the final TIMI flow after the primary PCI, and the incidence of in hospital MACE as follows: **Group A:** had a normal flow after the 1^{ry} PCI and did not have In hospital MACE. **Group B:** had either no reflow after the 1^{ry} PCI or experienced In hospital MACE.

The distribution of the studied groups is shown in Table 1.

Table 1. Distribution of the studied groups.

	No	%
Normal (group A)	99	82.5
No reflow or hospital MACE (group B)	21	17.5
No reflow only	15	71.4
In hospital MACE only (all in the form of cardiac death)	5	23.8
Both	1	4.8

The demographic data of the two studied groups are shown in table 2.

Table 2. Comparison between the two studied groups according to demographic data.

	Group A (n=99)		Group B (n=21)		Test of sig.	p
	No	%	No	%		
Sex						
Male	75	75.8	13	61.9	χ^2=1.700	0.192
Female	24	24.2	8	38.1		
Age						
Min. – Max.	29.0 – 81.0		44.0 – 78.0		t=2.498	0.014
Mean ± SD	56.30 ± 10.34		62.29 ± 7.90			
Median	58.0		62.0			

The distribution of the studied groups with respect to pre PPCI variable is shown in tables 3-6.

Table 3. Comparison between the two studied groups according to diabetes, hypertension and smoking.

	Group A (n=99)		Group B (n=21)		χ^2	p
	No	%	No	%		
Diabetes						
Non diabetic	62	62.6	9	42.9	2.803	^{MC}p=0.094
Diabetic	37	37.4	12	57.1		
Insulin	9	9.1	2	9.5	-	-
OHD	28	28.3	10	47.6		
Hypertension	48	48.5	7	33.3	1.602	0.206
Smoking						
Non smoker	43	43.4	11	52.4	0.560	0.454
Smoker	52	52.5	9	42.9	0.648	0.421
Ex-smoker	4	4.0	1	4.8	0.023	^{FE}p =1.000
Dyslipidemia	54		15	71.4	2.021	0.155
Family History	17		2	9.5	0.760	^{FE}p=0.521
Previous ACS	19		3	14.3	0.279	^{FE}p=0.762
Absence of preinfarction angina	56		15	71.4	1.584	0.208

Table 4. Comparison between the two studied groups according to SBP, DBP and pulse.

	Group A (n=99)	Group B (n=21)	t	p
SBP				
Min. – Max.	50.0-200.0	70.0-160.0	1.971	0.051
Mean ± SD	129.29 ± 27.93	116.67 ± 19.32		
Median	130.0	120.0		
DBP				
Min. – Max.	30.0-120.0	40.0-90.0	1.870	0.064
Mean ± SD	81.06 ± 15.62	74.29 ± 12.07		
Median	80.0	70.0		
Pulse				
Min. – Max.	41.0-120.0	60.0-130.0	0.069	0.945
Mean ± SD	84.56 ± 16.33	84.29 ± 15.69		
Median	80.0	88.0		

Table 5. Comparison between the two studied groups according to ECG.

	Group A (n=99)		Group B (n=21)		χ^2	p
	No	%	No	%		
ECG						
Anterior MI	72	72.7	14	66.7	0.313	0.576
Lateral MI	6	6.1	2	9.5	0.334	^{FE}p=0.628
Inferior MI	21	21.2	6	28.6	0.538	^{FE}p=0.565
Right MI	8	8.1	1	4.8	0.275	^{FE}p=1.000
Posterior MI	7	7.1	2	9.5	0.150	^{FE}p=0.656

Table 6. Comparison between the two studied groups according to pain to balloon time.

	Group A (n=99)	Group B (n=21)	Z	p
Pain to balloon time				
Min. – Max.	1.0-19.0	1.0-30.0	4.999*	<0.001*
Mean ± SD	6.08 ± 3.82	15.90 ± 7.87		
Median	5.0	17.0		

The distribution of the studied groups with respect to laboratory results, angiographic findings and procedural aspects is shown in tables 7-9.

Table 7. Comparison between the two studied groups according to laboratory results (on admission).

	Group A (n=99)	Group B (n=21)	Test of sig.	p
Plasma glucose				
Min. – Max.	84.0-442.0	104.0-440.0	Z=3.377*	0.001*
Mean ± SD	186.38 ± 84.65	275.29 ± 104.11		
Median	150.0	280.0		
N/L ratio				
Min. – Max.	1.20-24.0	2.80-13.0	Z=3.665	<0.001*
Mean ± SD	5.44 ± 3.53	8.19 ± 3.05		
Median	4.50	8.0		
MPV				
Min. – Max.	5.0-13.0	5.90-15.0	t=7.320*	<0.001*
Mean ± SD	8.58 ± 1.84	11.90 ± 2.09		
Median	8.20	12.20		

Discussion

A lot of researchers tried to study variables predicting the incidence of no reflow and/or in hospital MACE in STEMI patients undergoing PPCI. Ndrepepa G *et al.* [5], found that initial TIMI 0 flow in the infarct-related artery (P<0.001), initial perfusion defect (P<0.03), and previous myocardial infarction (P<0.013) as independent predictors of no reflow. Akpek M *et al.* [6], reported that N/L ratio >3.3 predicted no

Table 8. Comparison between the two studied groups according to infarct related artery.

	Group A (n=99)		Group B (n=21)		χ^2	p
	No	%	No	%		
Infarct related artery						
LAD	70	70.7	14	66.7	0.135	0.714
D1	5	5.1	0	0.0	-	-
CX	2	2.0	2	9.5	3.027	^{FE}p=0.141
OM	1	1.0	0	0.0	-	-
RCA	20	20.2	5	23.8	0.137	^{FE}p=0.769
PDA	1	1.0	0	0.0	0.214	^{FE}p=1.000

Table 9. Comparison between the two studied groups according to initial TIMI flow and Type of stent used.

	Group A (n=99)		Group B (n=21)		Test of sig.	p
	No	%	No	%		
Initial TIMI flow						
0	87	87.6	19	90.4	Z=1.844	0.065
1	25	25.3	1	4.8		
2	5	5.1	1	4.8		
Type of stent used						
No stent	0	0.0	2	9.5	χ^2=9.588*	^{FE}p=0.029*
BMS	51	51.5	12	57.1	χ^2=0.220	0.810
DES	48	48.5	7	33.3	χ^2=1.602	0.236

reflow with 74% sensitivity, and 83% specificity, and that high N/L ratio is independent predictor of no reflow, and in hospital MACE. Iwakura K *et al.* [7], found that admission hyperglycemia (>160 mg/dl) was an independent prognostic factor for no reflow, along with older age, male gender, absence of pre-infarction angina, complete occlusion of the culprit lesion, and anterior STEMI. Huczek Z *et al.* [8], found that high mean platelet volume (>10.3 fl) is a strong, independent predictor of no reflow in STEMI patients undergoing PPCI. In our study, we found that the groups with no reflow or in hospital MACE showed significantly older age, longer pain to balloon time, and higher levels of admission random plasma glucose (RPG), N/L ratio, and MPV.

Conclusion

Older patient age, longer pain to balloon time, admission hyperglycemia, higher admission N/L ratio and MPV are useful predictive factors for the occurrence of no reflow post PPCI, and/or in hospital MACE. Therefore strong attention should be paid to patients with one or more of these predictive factors, to protect them from the deleterious effects of no reflow, and avoid any of the in hospital MACE.

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