Prediction of short-term clinical outcome of percutaneous coronary intervention in patients with acute coronary syndrome through myeloperoxidase levels

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Original Article

Abstract

BACKGROUND: The present study assessed the significance of troponin and myeloperoxidase levels in the prediction of major adverse cardiac events (MACE) during the 1st month after percutaneous coronary intervention (PCI).

METHODS: This prospective, longitudinal study included 100 patients with acute coronary syndrome who underwent PCI. The participants' characteristics were recorded in a questionnaire. Blood samples were obtained before and 24 h after PCI, and troponin, and myeloperoxidase levels were measured. During the 1st month after PCI, death, myocardial reinfarction, and revascularization during admission were investigated through weekly phone calls. The value of troponin and myeloperoxidase levels before and after PCI in predicting MACE was evaluated using Cox regression.

RESULTS: Considering the obtained methods and the short duration of the study, 99% of the patients completed the study. Moreover, one death and four cases of myocardial infarction and revascularization were reported. Cox regression did not show significant relations between the incidence of MACE and myeloperoxidase levels before (hazard ratio = 1.12; 95% confidence interval 0.9, 1.39) and after PCI (hazard ratio = 0.86; 95% confidence interval = 0.43, 1.71), or troponin levels before (hazard ratio = 0.97; 95% confidence interval = 0.81, 1.17) and after PCI (hazard ratio = 1.03; 95% confidence interval = 0.96, 1.11).

CONCLUSION: It seems that the few cases of MACE, due to the small sample size and short duration of follow-up, had been insufficient for determining the predictive value of troponin and myeloperoxidase levels before and after PCI. Therefore, further studies with larger sample size and longer follow-up duration are recommended.

Keywords: Percutaneous Coronary Intervention, Acute Coronary Syndrome, Major Adverse Cardiac Events, Myeloperoxidase

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Introduction

Percutaneous coronary intervention (PCI) is currently recommended to patients with acute coronary syndrome (ACS).[1] Each year more than a million Americans with ACS or even non-acute coronary disease are treated with PCI. Previous meta-analyses have indicated PCI to improve outcome of patients with ACS.[2]

Various studies have also reported the major adverse cardiac events (MACE) of PCI (mainly due to restenosis of coronary arteries). A 2-year cohort study on 1010 patients who underwent PCI found recurrent events in 361 subjects and the need for revascularization in 201.[3] The Randomized Intervention Treatment of Angina showed that PCI was associated with slight increments in the incidence of myocardial infarction and death.[4]

Scientists have been seeking ways to predict and

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prevent the side-effects of PCI. Many studies have focused on measurement of different biomarkers. For instance, myocardial infarction-related biomarkers have been suggested to increase by 1–30% following PCI.[5,6] Meta-analysis revealed that increased troponin levels during the hospital stay after PCI augments the risk for the incidence of MACE.[6] Another meta-analysis reported a relationship between increased troponin levels and elevated risk of side-effects.[5] Meanwhile, troponin is only an indicator of myocardial injury and cannot be an appropriate predictor in early disease course or in cases without myocardial injury.[7] In addition, only Troponin elevation more than 3 times is associated with MACE.[8] Finding an alternative mediator with early increments is essential to the prediction of side-effects.

During the incidence of ACS, degranulation of polymorphonuclear neutrophils (PMN) is one of the earliest events in the coronary circulation. The reaction between myeloperoxidase (a major enzymes secreted by PMN)[9] and hydrogen peroxide causes the formation of atherosclerotic plaques, endothelial dysfunction, plaque instability, and finally ventricular remodeling after ischemic injury.[10-12]

The c7E3 Anti-Platelet Therapy in Unstable Refractory angina (CAPTURE) study on patients with ACS who had undergone PCI showed death and myocardial infarction rates during a 6-month follow-up period to be related with serum myeloperoxidase concentrations. Such a relation was also evident in patients without troponin increase.[13] In contrast, Moldoveanu et al. failed to find a significant relation between myeloperoxidase levels and restenosis in patients with ACS who had undergone primary PCI.[14]

The frequency of PCI in Iran has been reported as 1260/month;[15] moreover, the rate of MACE during the 24-month period after PCI is 10.3% in the country.[16] Considering this high frequency of their impact on post-PCI outcome, identifying the risk factors of MACE in patients undergoing PCI will result in substantially decreased incidence of side-effects. This study assessed the relation between myeloperoxidase levels and short-term incidence of MACE in patients receiving PCI for ACS.

Materials and Methods

This prospective longitudinal study evaluated the frequency of MACE after PCI in patients with ACS who were eligible for PCI. The subjects had undergone PCI in winter 2011 in Isfahan, Iran. The eligible subjects were the patients with ACS who needs intervention based on the recommendation of interventional cardiologist. The inclusion criteria were aging over 18 years, consenting to participate, and not having a history of PCI or coronary artery bypass graft (CABG) surgery. Individuals with malignancies, acute febrile diseases, chronic inflammatory diseases, or a surgery during the two months prior to the study were not included since these conditions elevate myeloperoxidase levels.[17]

Convenience sampling was performed to reach a sample size of 100 based on the formula:

$$N_{\text{Discordant}} = \frac{(z_{1-\alpha/2}(\psi+1)+2z_{1-\beta}\sqrt{\psi})^2}{(\psi-1)^2} \quad N_{\text{Pairs}} = \frac{N_{\text{Discordant}}}{\pi_{\text{Discordant}}}$$

In this formula, α was < 5% with a power = 80% and effect size = 1.5.

The eligible patients were first explained about the objectives of the study. After receiving consent and before performing PCI, a questionnaire containing demographic characteristics (age, gender, education, and occupation), history of diseases (diabetes mellitus, stroke, hypertension, coronary artery disease, and hyperlipidemia), physical activity and smoking status, medications (aspirin, heparin, plavix, integrilin, inotrope, beta-blockers, angiotensin-converting-enzyme inhibitors, and streptokinase), type of myocardial infarction, if present (anterior, posterior, inferior, lateral or septal), and symptom onset was completed for all subjects.

Systolic and diastolic blood pressure and anthropometric indices, including height, weight, and waist circumference were measured according to standard methods. PCI-related data, that is, the time each patient entered the angiography department, exact time of PCI, number of involved vessels, level of stenosis in angiography (as 100%, 90–99%, and 75–90%), type, size, and number of stents, number and size of balloons, and the need for thrombectomy, was extracted from the participants' angioplasty reports and recorded in the questionnaire. Blood samples were obtained before and 24 h after PCI and myeloperoxidase and troponin levels were measured by using enzyme-linked immunosorbent assay.

All subjects were followed for 1-month, and the incidence of MACE was recorded. The studied events were death, acute myocardial infarction, unstable angina, cerebrovascular accident, target vessel revascularization (a second PCI or CABG), and number of readmissions.

A cardiology resident phoned patients weekly and filled out the questionnaires. In case of any incident except death, the patient was asked to

provide the resident with his/her records to confirm the type of event. Otherwise, death certificates were reviewed to find the cause of death. Patients who were not willing to continue the study and those who were out of access were excluded.

All collected data were entered into SPSS for Windows (version 19.0, SPSS Inc., Chicago, IL, USA). The frequency of events was analyzed using descriptive statistics. Patients with and without MACE were compared using the Student's t-test for quantitative variables and by Chi-square and Mann-Whitney tests for qualitative variables or quantitative variables without normal distribution. Cox regression was employed to assess the relations between myeloperoxidase and troponin levels and the incidence of events. Therefore, hazard ratios were calculated in crude and three adjusted models by age, sex, and troponin or myeloperoxidase before and after PCI.

Results

This study assessed 100 patients of which 26 women and 74 men during 73 days. The mean age of the participants was 58.62 ± 10.80 years and all subjects were married. The majority of patients (n = 43) were illiterate, and only seven individuals held a university degree. Almost half of the study population (n = 48) had unstable angina and 52 had a myocardial infarction.

During 1-month period, one patient died, and revascularization was performed for four subjects (three cases of a second PCI and one case of CABG), but no cases of myocardial reinfarction were detected. Five participants experienced MACE among whom two (40.0%) were female, and three (60.0%) were male. The frequency of men and women who did not suffer from MACE during 1-month of follow-up was 66 (89.2%) and 23 (88.5%), respectively (P = 0.919). The mean age of patients with and without MACE was 59.55 ± 14.02 and 58.51 ± 10.44 years, respectively (P = 0.603). The two groups were not significantly different in terms of the incidence of myocardial infarction or unstable angina before PCI (P = 0.933).

The participants' demographic characteristics, diagnosis of coronary disease at the time of admission, history of diseases, lifestyle, blood pressure, anthropometric indices, PCI target vessel, and the method of PCI in the two groups with and without MACE are presented in table 1. The myeloperoxidase and troponin levels before and after PCI did not have a normal distribution and Mann-Whitney test was used to compare them. Table 2 shows non-significant differences between the mean of myeloperoxidase and troponin levels before and after PCI.

Cox regression was used to find relations between myeloperoxidase and troponin levels and the incidence of MACE. Such possible relations were first evaluated in a crude model and then in models adjusted for one of the enzymes. Finally, myeloperoxidase and troponin levels before and after PCI were not found to have significant relationships with the incidence of MACE (Table 3).

Discussion

A total of 100 patients who had undergone PCI for ACS were followed for 1-month in the current study. The frequency of MACE was 11.0%, and only one subject died during this 1-month period. In a study on complications of PCI during the 1st month after the procedure, Khosravi et al. have reported the frequency of death as 2.2% and the overall frequency of target vessel revascularization, myocardial infarction, and stroke as 3.2%.[18] Wu et al. performed a retrospective study to evaluate 12-h and 28-day mortality rate in 2299 patients who had undergone PCI after ACS during a 14-year period. They found 50 individuals (2.2%) to have died in the first 28 days after PCI.[19] Thirty-day mortality rate was calculated as 0.38% in a research on 51,695 subjects who had undergone PCI after ACS in New York.[20] Although the mortality rates of patients with PCI after ACS in the studies of Khosravi et al.[18] and Wu et al.[19] were higher than that in the present research, Hannan et al. found lower rates.[20] This difference might have been caused by the large sample size in the latter study. The higher incidence of stroke, myocardial infarction, and target vessel revascularization in the present study (4.45%) compared to the study of Khosravi et al.[18] can be justified by the difference is the sample size since both samples were selected from the same hospital.

In the present study, two groups with and without MACE during the 30 days following the PCI did not have significant differences in any of the measured indices. Moreover, myeloperoxidase and troponin levels before and after PCI were not significantly different between the two groups and hence had no significance in prediction of MACE. Considering the few incidences of MACE due to the small sample size and short duration of follow-up, we could not adjust models for other variables. However, eliminating the effects of troponin for myeloperoxidase and vice versa did not result in significant relationships.

Table 1. The comparison of basic characteristics and the type of percutaneous coronary intervention (PCI) between patients with and without major adverse cardiac events (MACE)

Variables	Total	Without MACE (n = 89)	With MACE (n = 11)	P
Demographics				
Sex*				
Men	74 (74)	66 (89.2)	8 (10.8)	0.919
Women	26 (26)	23 (88.5)	3 (11.5)	
Married*	100 (100)			
Educational level*				
Illiterate	43 (43)	37 (41.6)	6 (54.5)	
Elementary	36 (36)	33 (34)	3 (27.3)	0.898
High school	14 (14)	12 (13.5)	2 (18.2)	
Academic	7 (7)	7 (7.9)	0 (0)	
Diagnosis before PCI*				
Myocardial infarction				
≥2	12 (12)	10 (11.2)	2 (18.2)	
Anterior	10 (10)	9 (10.1)	1 (9.1)	
Inferior	19 (19)	16 (18.0)	3 (27.3)	
Septal	3 (3)	3 (3.4)	0 (0)	0.933
Extensive anterolateral	2 (2)	2 (2.2)	0 (0)	
Non-STEMI	6 (6)	6 (6.7)	0 (0)	
Unstable angina	48 (48)	43 (48.3)	5 (45.5)	
History*				
Hyperlipidemia	38 (38)	33 (37.1)	5 (45.5)	0.589
Hypertension	41 (41)	35 (39.3)	6 (54.5)	0.333
Diabetes	23 (23)	21 (23.6)	2 (18.2)	0.687
Coronary heart disease	30 (30)	26 (29.5)	4 (36.4)	0.643
Cerebrovascular disease	0 (0)			
Medications*				
Aspirin	100 (100)	89 (100)	11 (100)	1.000
Heparin	3 (3)	3 (3.4)	0 (0)	1.000
Plavix	81 (81)	72 (80.9)	9 (81.8)	0.942
Streptokinase	1 (1)	1 (1.1)	0 (0)	1.000
Integrilin	5 (5)	4 (4.5)	1 (9.1)	0.449
Beta blocker	97 (97)	(86) 96.6)	11 (100.0)	1.000
ACE I	55 (55)	49 (55.1)	7 (63.6)	0.589
Statins	99 (99)	88 (98.9)	11 (100.0)	1.000
TNG*	99 (99)	88 (98.9)	11 (100.0)	1.000
Lifestyle*				
Sedentary lifestyle	83 (83)	72 (80.9)	11 (100.0)	0.112
Smoker	36 (36)	30 (33.7)	6 (54.5)	0.196
PCI*				
Vessel				
Saphenous vein graft	6 (6)	5 (5.6)	1 (9.1)	
Left circumflex	11 (11)	11 (12.4)	0 (0)	
Right coronary	24 (24)	21 (23.6)	3 (27.3)	0.677
Left coronary	47 (47)	42 (47.2)	5 (45.5)	
≥2 arteries	11 (11)	9 (10.1)	2 (18.2)	
Balloon	10 (10)	8 (9)	2 (18.2)	
Stent	18 (18)	16 (18)	2 (18.2)	0.673
Balloon and stent	70 (70)	63 (70.8)	7 (63.6)	
Type of the stent				
Bare	42 (42)	36 (40.4)	6 (54.5)	
Drug eluted	40 (40)	38 (42.7)	2 (18.2)	0.328
Both	6 (6)	5 (5.6)	1 (9.1)	
Physical examination**				
BMI	26.11 ± 3.29	26.29 ± 3.33	24.71 ± 2.64	0.165
Waist	81.31 ± 8.29	81.57 ± 7.98	79.18 ± 10.67	0.662
Systolic blood pressure	118.04 ± 16.36	118.09 ± 15.82	117.64 ± 21.18	0.478
Diastolic blood pressure	75.46 ± 8.99	75.80 ± 8.85	72.73 ± 10.09	0.323
Age**	58.62 ± 10.80	58.51 ± 10.44	59.55 ± 14.02	0.996

* Number (%); ** Mean ± SD; MACE: Major adverse cardiac events; PCI: Percutaneous coronary intervention; STEMI: ST segment elevation myocardial infarction; ACE I: Angiotensin-converting enzyme inhibitors; TNG: Trinitroglycerin; BMI: Body mass index; SD: Standard deviation

Table 2. Comparison of myeloperoxidase and troponin between patients with and without major adverse cardiac events (MACE) before and after percutaneous coronary intervention (PCI)

Laboratories	Total mean ± SD	Without MACE (n = 89) mean ± SD	With MACE (n = 11) mean ± SD	P
Myeloperoxidase				
Before PCI	3.29 ± 2.21	3.21 ± 2.21	3.95 ± 2.17	0.207
After PCI	5.45 ± 3.44	5.49 ± 3.35	5.15 ± 4.29	0.570
Troponin				
Before PCI	1.13 ± 1.02	1.14 ± 0.99	1.02 ± 1.21	0.331
After PCI	3.50 ± 6.24	3.32± 6.09	4.09 ± 7.47	0.643

P values were reported based on Mann-Whitney test; MACE: Major adverse cardiac events; PCI: Percutaneous coronary intervention

Table 3. The hazard ratio of myeloperoxidase and troponin before and after percutaneous coronary intervention (PCI) for occurring events

	Hazard ratio (95% CI)	P
Myeloperoxidase		
Before PCI		
Crude	1.12 (0.9, 1.39)	0.31
Adjusted*	1.13 (0.9, 1.43)	0.28
Adjusted**	1.11 (0.89, 1.39)	0.34
Adjusted***	1.13 (0.89, 1.42)	0.31
After PCI		
Crude	0.86 (0.43, 1.71)	0.67
Adjusted*	0.97 (0.81, 1.17)	0.76
Adjusted$	0.93 (0.77, 1.13)	0.49
Adjusted£	0.93 (0.77, 1.13)	0.49
Troponin		
Before PCI		
Crude	0.97 (0.81, 1.17)	0.76
Adjusted*	0.86 (0.43, 1.71)	0.67
Adjusted§	0.89 (0.45, 1.76)	0.74
Adjusted∫	0.89 (0.45, 1.78)	0.75
After PCI		
Crude	1.03 (0.96, 1.11)	0.44
Adjusted*	1.03 (0.96, 1.11)	0.43
Adjusted†	1.03 (0.96, 1.11)	0.39
Adjusted‡	1.03 (0.96, 1.12)	0.39

* Adjusted by age, sex; ** Adjusted by troponin level before PCI; *** Adjusted by age, sex, troponin level before PCI; $Adjusted by myelopero xidase level, troponin level before PCI; £ Adjusted by age, sex, myeloperoxidase level and troponin level before PCI; §Adjusted by troponin level before PCI; ∫ Adjusted by age, sex, troponin level before PCI; † Adjusted by troponin level and myeloperoxidase before PCI; ‡ Adjusted by age, sex, myeloperoxidase level and troponin level before PCI; CI: Confidence interval; PCI: Percutaneous coronary intervention

Despite the significant increase of troponin levels after PCI, the regression model did not show a significant relationship between troponin levels before and after PCI and the incidence of MACE. This is in contrast with a number of previous studies probably due to the very small sample size of our study.[21,22]

Fewer studies have examined the value of myeloperoxidase in predicting the incidence of MACE after PCI. In a study on 128 patients with ACS, Chang et al. measured myeloperoxidase levels during the hospital stay before primary PCI. They suggested serum concentration of myeloperoxidase to be significantly related with the incidence of MACE during the 30 days after PCI.[17] In contrast, Moldoveanu et al. evaluated 80 patients with ACS in terms of restenosis 1, 3, and 6 months after primary PCI. They measured myeloperoxidase, adiponectin, and lipoprotein-associated phospholipase A2 before PCI and immediately, 24, 48, and 72 h, and 1, 3, and 6 months after PCI. Their findings did not indicate a significant association between serum myeloperoxidase levels and the incidence of MACE. However, adiponectin was found to be significantly related with time of discharge and restenosis after 6 months.[14] Similarly, we failed to find a significant relation between myeloperoxidase levels before and after PCI and the incidence of MACE one month after PCI. In other words, although myeloperoxidase levels before and after PCI were significantly different, there was no significant difference between the groups with and without MACE in this regard.

Overall, we did not detect significant relations between troponin and myeloperoxidase levels before and after PCI and the incidence of MACE. A probable reason is a small sample size. Larger sample size and increased frequency of MACE provide the possibility of adjustments for other variables and finding significant relations. On the other hand, since many studies have found significant relations (especially in case of troponin) in long-term follow-up, increasing the duration of follow-up may reveal such relations. Another important factor is measuring cardiac biochemical markers, that is, measurement of cardiac troponin in several previous studies could have been responsible for the observed relations. Increased frequency of troponin and myeloperoxidase measurements can also reveal

significant relations between the incidence of MACE and one of these enzymes.

Conclusion

This study did not show significant relations between troponin and myeloperoxidase levels before and after PCI and the incidence of MACE. Further studies with larger sample size, increased number of measurements, and longer duration of follow-up are required for obtaining better, more reliable results. Other biochemical factors with probable relations with ischemia and clotting should also be evaluated.

Conflict of Interests

Authors have no conflict of interests.

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