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SHORT COMMUNICATION

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Association of Aspirin Intake and Myocardial Infarction Size

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Abstract

Myocardial infarction (MI) is one of the most important health burdens worldwide. Aspirin as an non-Steroid Anti—inflammatory drug, has been proven to be a protective factor to decrease the incidence, however its effect of MI size is still unknown. We designed this study to compare the biomarkers after MI in patients with and without aspirin intake. 378 patients were enrolled and the results showed lower cardiac troponin T and Creatine Kinases in patients with protective dose of aspirin intake. In addition, Creatine Kinases were significantly higher in patients with no history of MI. We conclude that aspirin can reduce the size of the infraction. Also, higher enzymes can be due to higher muscle content in patient without MI history.**[GMJ. 2012;1(1):35-37]**

Keywords: Aspirin - Myocardial infarction size - Troponin T - Creatine Kinase

Introduction

Myocardial infarction (MI) is one of the most important health issues worldwide, which is estimated to be one of the main causes of death throughout the world (1). One of the most consequential determinants of outcome in MI is infarction size that is important for therapeutic interventions (2). Cardiac biomarkers like Cardiac Troponins, creatine phosphokinase (CPK) and its Cardiac Isoenzyme (CK-MB) provide quantitative estimation of infarction size (3). Although aspirin therapy is a cornerstone in the treatment and prevention of myocardial infarction and decrease in incidence of MI and reduces mortality and hospitalization after ischemic attack (4), there are not enough investigations about effects of aspirin on MI size, which lead us to the conduction of this study.

Methods

In this case-control study study, 378 patients admitted to Coronary Care Unit (CCU) of Rasoul-e-Akram hospital with MI diagnosis dur-

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ing April 2009 to April 2010 were enrolled. The diagnosis of MI was made in each patient by a single cardiologist in accordance with European Society of Cardiology/American Heart Association Guidelines and the diagnosis was confirmed by an elevation of cardiac Troponin T (cTnT) above 0.03 mg/ml. Other information such as age, and medical history was taken from their medical files. Any patients with other illnesses such as preicarditis, pure valvular deformity, any sign of physical injury, renal insufficiency or history of cardiopulmonary resuscitation (CPR) were excluded from our study.

The highest value of cTnT during 72 Hours after CCU admission (3 times was recorded for every patient at a single admission) was considered to estimate the infarction size. In addition, the highest value of CPK and CK-MB during 48 Hours after admission (measured 6 times after admission) was defined as a peak CK and CK-MB for measurement of MI size. All of the data was included in a checklist and analyzed by SPSS version 13. We analyzed the data by SPSS version 13 and expressed them by mean ± SD or percentages. Suitable statistical tests (t-test and chi-square) were utilized. P-value less than 0.05 were considered as significant. The Protocol of this study was approved by Tehran University of Medical Sciences Ethics Committee; furthermore, all patients entered the study as anonymous and the information gained will be not be presented individually.

Results

The study population consist of 378 patients (132 female and 246 male) with mean age 62.42 (SD=14.10) years old. Baseline characteristics are shown in Table 1.

The concentration of cTnT, CK-MB and CK showed significant difference in two groups regarding to aspirin use, it was lower in patients who had used Aspirin (P-value < 0.001). After stratification based on previous history of MI, significantly higher concentration of cardiac enzymes was seen between two groups of positive and negative history of MI (Table.2).

Blood concentration of CK and CK-MB was significantly different in two groups with and without positive history of previous MI. (P<0.001 and P=0.004, respectively).

Table 1. Baseline characteristics of patients.

	Aspirin use (n=143)	No Aspirin use (n=235)
Age	66.69 ± 11.92	59.22 ± 14.13
Male / female (n)	90 / 53	166 / 69
Angina type:		
Typical (n)	75 (52.44%)	138(58.72%)
Atypical (n)	68 (47.55%)	97 (41.27%)
Risk factors:		
DM (n)	49 (34.26 %)	54(22.97%)
Stroke (n)	21(14.68%)	9(3.8%)
Smoking (n)	9 (34.26%)	104(44.25%)
Previous MI (n)	66 (46.15%)	31(13.19%)

Conclusion

In this study, we investigated the relation between aspirin use and infarction size by biomarkers. Based on the results we found that there is an association between history of aspirin intake and MI size. In previous studies, it was inconsistently confirmed that aspirin intake could reduce the risk of MI in general population (4-5), but the effect of aspirin on infarction size had not been studied.

This association between MI size and aspirin intake also was present in groups of positive and negative history of MI. History of infarction have significant association with aspirin intake and affects distribution of aspirin users in different groups. This showed that the difference of infarction size is not related to imbalance distribution of aspirin users in groups regarding to history of MI.

In a study by Mukamal et al, the association of aspirin intake during 4 days before MI and MI size was found. This is the only study that considered the size of MI in addition to incidence. In this study, the time before MI concerned was only 4 days, however the process of MI is a long period and is affected by long duration of aspirin intake (6).

In this study, the difference between patients

Table.2.concentration of cardiac enzymes in patients with MI diagnosis.

	Aspirin	No Aspirin	Pvalue
cTnT	0.57±0.55	1.05±0.85	<0.001
CK-MB	110.02±94.13	163.92±136.93	<0.001
CK	755.14±671.88	1258.4±1024.3	<0.001
After stratification:			
Positive History of MI			
cTnT	0.5770±0.55652	1.2195 ± .83481	0.008
CK-MB	95.98 ±89.23	153.74 ± 131.36	0.024
CK	580.22± 503.03	1029.8± 863.18630	0.049
N0 History of previous MI			
cTnT	0.5416±0.53830	1.0451±0.86462	0.005
CK-MB	124.20±99.68	166.37±139.61	0.033
CK	896.29±756.24	1284.4±1037.81	0.013

with and without history of MI was significant in concentration of cardiac enzymes that may be caused by more live tissue available in patients without history of previous MI leading ultimately to less CK and CK-MB release, in equal infarction size of heart. Preconditioning may be another cause of this difference.

The effect of aspirin on infarction size was shown by this historical cohort investigation. However, this is not well known and more prospective research is proposed with the aim of finding the real association between aspirin and size of infarction and the reasons of relation, in different groups of disease.

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