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SMOKING AS RISK FACTOR FOR MYOCARDIAL INFARCTION WITH EMPHASIS ON CARDIAC BIOMARKERS IN THE PATIENTS ADMITTED IN KASHMIR INSTITUTE OF CARDIOLOGY MIRPUR KASHMIR, PAKISTAN

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ABSTRACT

Objective: The aim of this population-based study was to elucidate smoking as risk factors for myocardial infarction with emphasis on cardiac biomarkers.

Study Design: Study was performed at indoor patients of Department of Kashmir Institute of Cardiology Mirpur, Azad Jammu and Kashmir in the time period of 12 months.

Materials and Methods: This study was consisted of total 240 patients from which 140 were indoor admitted patients of Acute Myocardial Infarction (AMI) in Kashmir Institute of Cardiology and 120 were used as a control by their written agreement. Complete record of their medical history was obtained.

Results: The mean concentration of cardiac biomarkers, CK-MB and LDH enzymes was analyzed in MI affected smokers and non smokers.

Conclusion: It is concluded that concentration of cardiac biomarkers CK-NAC, CK-MB and LDH is significantly increased in smokers affected of MI than non smokers with MI.

Keywords: CK-NAC, LDH, Cardiac Enzymes, Myocardial infarction, Cigarette smoking

INTRODUCTION

Cardiovascular diseases are the cause of about 15.5million deaths every year and its 50% is due to Acute Myocardial Infarction (AMI). In developing countries four out of five deaths occur due to this disease **[1]**. The analysis of myocardial damage can be done by its pathology, ECG findings, echocardiography and measurement of blood level of cardiac enzymes **[2]**. Smoking is considered as a risk factor for myocardial infarction, atherosclerosis and sudden cardiac arrest with death. Smoking is cause of early ST segment elevation myocardial infarction (STEMI) in healthier patients. Smoking is the cause of average 7 years earlier chances to develop infarction, and smoker persons are twice at risk to develop infarction than non-smokers **[3]**.

A major cardiovascular risk factor is cigarette smoking both active and passive as well **[4, 5]**. Inhalation of tobacco smoke is responsible for severe immediate responses in the heart and its blood vessels. As a person start smoking then within one minute of starting the heart rate increases and it is attributable to nicotine. Nicotine is stimulant for the production of adrenaline so it makes the heart- beat faster as well as it causes high blood pressure **[6]**.

Smoking of tobacco increases the exposure to carbon monoxide (CO) as it is the fourth most common chemical of the tobacco smoke and it has 3-5%of volume **[7]**. Increased levels of CO in the blood causes the inability of the body to carry oxygen is as it forms carboxyhemoglobin that results in oxygen deficiency and tissue hypoxia. This causes tissue to undergo infarction. Due to increased carboxyhemoglobin levels, smokers experience shortness of breath and elevated heart beat rate. Tobacco smoke raises the blood cholesterol levels due to the chemical acrolein and it affects the cholesterol metabolism, causing hypercholesterolemia **[8]**. It also decreases the high-density lipoprotein (HDL) and low-density lipoprotein (LDL) ratio **[9]**. Tobacco toxins damages the walls of blood vessel which results in plaque formation and thrombosis because it affects fibrinogen levels as well as increased platelet aggregation **[10]**.

Smoking causes the vasoconstriction by decreasing nitric oxide levels which are responsible for dilation of blood vessels. Ischemic heart disease (IHD) is second leading cause of death in Pakistan **[11]**. The incidence of AMI is prevailing in differing age and genders but with greater magnitude

in aging **[12]**. The cigarette smoke is dangerous for smoker as well as to people nearby. The people who get exposed to environmental tobacco become passive smokers **[13]**. Passive smoking is the risk factor of MI in non-smokers **[14]**.

The present population bases study was performed on specific population of Mirpur Azad Kashmir to detect the behavior of the MI evaluating different cardiac biomarkers for the detection of MI in relation to the following factors:

i. Gender of the subjects showing ST segment elevation and T wave inversion.
ii. Risk factors of smoking indicating MI.

MATERIAL AND METHODS

STUDY DESIGN

The case control study was performed at specific population of Mirpur Azad Jammu and Kashmir (AJK). Study was done for indoor patient department of Kashmir Institute of Cardiology Mirpur AJK in 12 months' time duration from June 2013 to July 2014.

SAMPLING SELECTION

Non-probability purposive sampling technique was done and sample size was 240 patients. 120 were registered as AMI patients Kashmir Institute of Cardiology and remaining 120 patients were age and sex controls who provided written consent.

Inclusive study sampling was Patients with chest pain indicative of myocardial ischemia within 12 hours after the appearance of symptoms on the basis of ECG. Study control was persons with non-infarcted ischemia who were there for general checkup.

Persons with severe skeletal muscle damage and cardiac resuscitation were excluded from present stud.

PROCEDURE FOR SAMPLE COLLECTION

Serum samples were collected from one of peripheral vein of arm of patients and from controls who were not infarction patients. After onset of acute symptoms, blood was taken from patients. Serum separation was done by centrifugation and serum analysis of CK-NAC, CK-MB and LDH was done with chemistry analyzer Microlab.

EVALUATION OF CARDIAC ENZYME

Immunoinhibition Assay forcreatine kinase-MB

Principle:

It is antibody based assay in which antibody bind to CK reagent and inhibits the activity of its M subunit. So specifically activity of its B subunit was measured and multiplied with factor 2.

Procedure:

FOR each of the enzyme 20 µl of samples i.e. standard, specimen and controls were distributed into different wells. Enzyme

conjugate reagent of 200 µl concentration was added in every well and shaking was done for 30 seconds to mix the content. For 60 minutes, wells were incubated at room temperature (18-25°C). Then mixture was thoroughly washed from wells with distilled water and 100 µl of TMB reagent was added, shaked and incubated for 20 minutes. After that color changed was examined and optical density of colored mixture was measured at 450 nm in a microtiter plate reader.

STATISTICAL ANALYSIS

SPSS 20 was used for data analysis and quantitative variables were used with mean± SD. Study group was divided into 3 age groups: 21-39, 40-59 and 60 years. ANOVA was applied to associate different variables in study to evaluate different concentrations of CK-NAC, CK-MB and LDH. P<0.005 was taken as significant value.

RESULTS

The cardiac biomarkers such as enzymes for myocardial infarction (MI) have been assessed in MI patients and non MI control subjects. The cardiac enzymes have been studied in relation to smoking in male gender.

CONCENTRATION OF ENZYMES IN MALE SUBJECTS WITH MI (Figure 1)

CK-MB in MI affected male Subjects

CK-MB: The mean level of CK-MB in male smoker with MI was 65±8.6U/L; while the mean CK-MB level in non smokers was 21±0.8U/L. 67% lower concentration of CK-MB was observed in non smokers patients as compared to the smoker patients. The difference was significant statistically (0.006).

CK-NAC: The CK-NAC concentration was 489.85 ±67 U/L in smokers with MI and it was 144±6U/L non smokers. So 70% higher concentration of CK-NAC was observed in smokers with MI than non smoker with MI. The difference was significant statistically (P=.0.005).

LDH in MI affected male subjects

The LDH concentration in MI male smokers was 518±41 U/L and in non smokers patients it was 371±28 U/L. So 28% lower concentration in non smokers than smoker MI affected males. Its difference was significant statistically (0.05).

CONCENTRATION OF CARDIAC ENZYMES IN MALE SUBJECTS WITHOUT MI

The concentration of CK-NAC, CK-MB and LDH was significantly higher in male smokers without MI than in non smokers without MI as can be seen in **Table 1, Figure 2**.

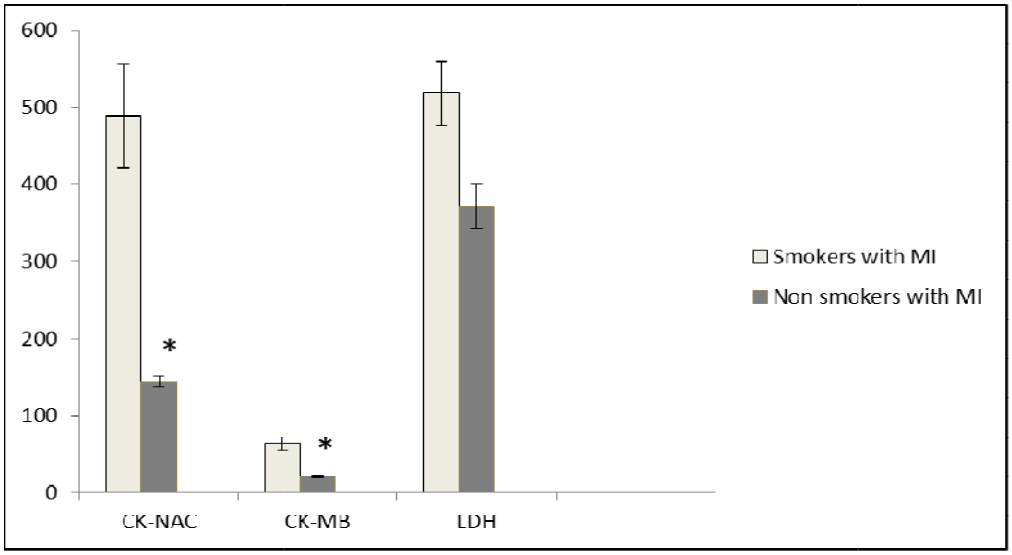

Figure 1: Concentration of Cardiac enzymes in smoker and non smoker males with MI
CK-NAC: Creatine Kinase N-acetylcholine; CK-MB: Creatine Kinase Myocardial Band; LDH: Lactate Dehydrogenase. *P < 0.05: Statistically significant.

Table 1: Concentration of cardiac enzymes U/L in males without MI

Enzymes	Enzyme conc. in smokers U/L	Enzyme conc. in non-smokers U/L	P	P<0.005
CK-NAC	173.56±6.7	159.61±3.4	3.215	0.077
CK-MB	24±1	23±0.6	0.738	0.393
LDH	368.16±9.7	322.76±9.5	10.941	0.001

***Significant p value<0.05**

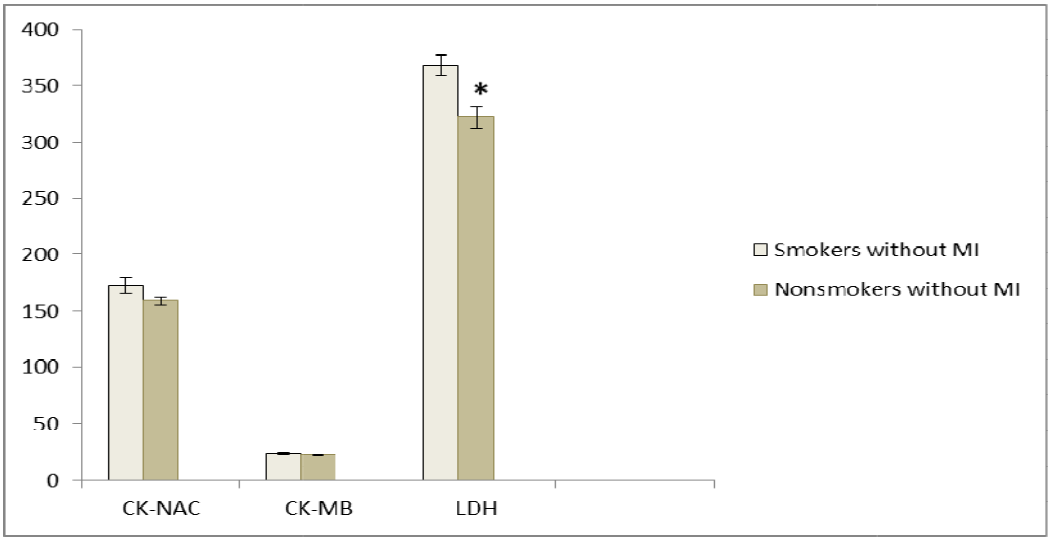

Figure 2: Levels of Cardiac enzymes in Smoker and Non-smoker Males without MI
CK-NAC: Creatine Kinase N-acetylcholine; CK-MB: Creatine Kinase Myocardial Band; LDH: Lactate Dehydrogenase. *P < 0.05: Significant value

DISCUSSION

The present population based study confirms that smoking increases the occurrence of MI. Smoking is the higher risk of cardiovascular diseases (CVD) and it increases the risk of AMI and heart failure Smoking is also recognized as the most common risk factor for cardiac diseases and it is also related to MI during young age **[15]**. In general population,

smoking increases the risk of CVD and its related mortality.

The present study confirms that concentration of cardiac biomarkers in increased in smokers with and without MI. The CK-MB concentration is increased in MI affected smoker males so smoking is observed as a well-known risk factor for the MI **[16]**. In comparison to healthy subjects of matched age, young patients with MI were more smokers and were patients of other CVDs **[17]**.

In this present study, levels of CK-NAC, CK-MB and LDH levels were significantly increased in male subjects who were smokers with or without MI than non smoker individuals. This is in accordance to Shazia *et al*., 2010 who also observed significantly higher concentrations of CK-MB in smokers affected with MI. This finding of increase of cardiac enzymes is confirmed from Pasupathi *et al*. 2009, which showed significantly increased concentrations of CK-NAC and CK-MB in smokers with and without MI **[18]**.

CONCLUSION

The present study confirms that smoking is risk factor for MI and it also elevates concentration of cardiac biomarkers. In conclusion, it is observed that in male smokers concentration of cardiac enzymes CK-MB, CK-NAC and LDH are increased and smoking is a risk factor of MI and other cardio vascular diseases. Further studies will be done to elaborate the exact mechanism involved in increase of cardiac enzymes in MI and non MI subjects.

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