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Original Article

Comparative analysis of biochemical parameters in diabetic and non-diabetic acute myocardial infarction patients

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ABSTRACT

Background: Diabetes is a metabolic disorder characterized by enhanced production of free radicals hence oxidative stress. The aim of this study was to evaluate the activity of cardiac and antioxidant enzymes in diabetic and non-diabetic acute myocardial infarction (AMI) patients.

Methods: This case–control study was conducted on 450 subjects (70–85 years). Subjects were divided into three groups (Normal, N; Non-diabetic AMI, N-AMI; and Diabetic AMI, D-AMI). Each individual was subjected to a detailed history, clinical examination, and cardiovascular parameters analysis (fasting blood sugar, HbA1c, systolic and diasystolic blood pressure, total cholesterol (TC), triglycerides (TG), low-density lipoprotein (LDL), high-density lipoprotein (HDL), TC/HDL and LDL/HDL ratios). Cardiac markers (Troponin-I, creatine phosphokinase (CPK), creatine kinase-MB (CK-MB), lactate dehydrogenase (LDH), C-reactive protein (CRP) and aspartate aminotransferase (AST)) and oxidative stress markers (superoxide dismutase (SOD), malondialdehyde (MDA), glutathione (GSH), catalase (CAT)) were also assessed. All these parameters were compared between diabetic and non-diabetic AMI patients.

Results: D-AMI individuals had high level of TC, TG, LDL, and low level of HDL in comparison to N-AMI individuals. Study suggests that cardiac markers such as Troponin I, CPK, CK-MB, AST, LDH, and CRP levels were significantly increased in patients suffering from myocardial infarction with diabetes mellitus (DM) compared to patients of myocardial infarction without DM. The activity levels of antioxidant SOD and GSH were lower in D-AMI patients than in N-AMI. However, levels of MDA and CAT were higher in D-AMI than in N-AMI controls.

Conclusion: Study suggests elevated cardiac markers and reduced antioxidants in D-AMI patients compared to N-AMI patients.

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1. Introduction

Diabetes mellitus (DM) increases the incidence of cardiovascular diseases (CVDs) and increases the risk of CVD-induced mortality in diabetic subjects compared to non-diabetic subjects.[1,2] Coronary artery disease (CAD) contributed to myocardial infarction (MI) and heart failure, attributed to most of the mortalities around the globe.[3–5] Acute myocardial infarction (AMI) is associated with obstruction of coronary artery, myocardial ischemia leading to myocardial necrosis and generation of reactive oxygen species (ROS).[6] Previous studies show that hyperglycemia promotes ROS-induced complications of heart by reacting with lipids, protein, and DNA[7]; this oxidative damage is rescued by myocardial antioxidants.[8] Several studies depicted that antioxidants functioning is diminished in diabetic subjects,[9] which may further augment the oxidative stress-induced pathogenesis of AMI.[10] Diabetes, dyslipidemia, hypertension, family history, obesity, and smoking are well documented risk factors for the development of AMI.[11]

The purpose of the study was to assess the oxidative stress-induced damage to heart in diabetic and non-diabetic AMI patients. This study emphasizes that antioxidants imbalance may be a key indicator of diabetes-induced myocardial damage as other indicators such as ECG and cardiac biomarkers. The data showed significant increase in lipid parameters (total cholesterol (TC), triglycerides (TG), high-density lipoprotein (HDL), low-density lipoprotein (LDL), and cardiac markers, i.e. troponin-I (TnI), creatine phosphokinase (CPK), creatine kinase-MB (CK-MB), lactate dehydrogenase (LDH), aspartate aminotransferase (AST), C-reactive protein (CRP) within 12 h after the onset of chest pain in D-AMI patients compared to N-AMI patients. Oxidative stress markers such as malondialdehyde (MDA), catalase (CAT), superoxide dismutase (SOD), and glutathione (GSH) also increased in D-AMI patients compared to N-AMI patients. Study results suggest that antioxidants based interventions in D-AMI patients might assist to reduce oxidative stress-induced damage in D-AMI patients.

2. Methods

2.1. Subjects and study design

This case–control study included 450 subjects; out of which, 150 subjects (90 males and 60 females) were with normal blood glucose level and with normal ECG (Normal, N), 150 subjects (85 males and 65 females) were with normal blood glucose level and AMI (non-diabetic and AMI, N-AMI), and 150 subjects (98 males and 52 females) were with diabetes and AMI (Diabetic and AMI, D-AMI), visiting the outpatient clinic at Department of Cardiology at Services Hospital Lahore, Punjab Institute of Cardiology Lahore and Ittefaq Hospital Lahore, Pakistan from September 2013 to May 2014.

Diabetes was diagnosed by analyzing the level of glycated hemoglobin level (HbA1c $>$ 6.5%).[12] Diagnosed cases of diabetic and non-diabetic AMI patients were included after obtaining a written consent from their caretakers to take part in the study. Questionnaires were duly filled in with bio-data of the patients, detailed medical history, blood pressure, electrocardiography (ECG), complete blood count (CBC) along with available additional information. This study was approved by the local ethical committee at The University of Lahore, Pakistan.

2.2. Inclusion criteria and exclusion criteria

Subjects of all ages and both genders with the history of AMI were included. AMI diagnosis was based on a history of chest pain, ECG changes, and elevated cardiac enzymes.[6,13] Diabetic and non-diabetic AMI patients were included in this study. The control subjects were selected on basis of being normotensive and with normal ECG. Subjects who have the history of smoking, obesity, or any other disease were excluded from this study.

2.3. Collection of blood and isolation of serum

Blood samples were collected from Department of Cardiology at Services Hospital Lahore, Punjab Institute of Cardiology Lahore and Ittefaq Hospital Lahore. Preprandial venous blood were drawn from cubital vein from all subjects and instantly transferred from hospital to CRIMM laboratory in an icebox. Blood samples were centrifuged at 2000 $\times$ *g* for 10 min at 4 °C. Serum was aspirated, aliquoted, and stored at −20 °C for analysis.

2.4. Evaluation of cardiovascular parameters

Serum levels of TC, TG, and HDL were measured spectrophotometrically using commercial assay kits (Randox laboratories Ltd, United Kingdom). LDL was calculated by using Friedewald formula.[14]

2.5. Analysis of cardiac markers

Levels of various cardiac enzymes including troponin-I (TnI), CPK, CK-MB, LDH, AST, and CRP were assessed using commercial kits (Randox laboratories Ltd, United Kingdom).

2.6. Estimation of oxidative stress

Oxidative stress was measured by analyzing the serum level of MDA, CAT, SOD, and GSH at Institute of Molecular Biology and Biotechnology (IMBB), The University of Lahore.

2.7. Determination of SOD activity

SOD activity was determined by the method of Kakkar et al.[15] Homogenate was prepared by mixing serum and trichloroacetic acid (50%) in 1:1 ratio and centrifuged at 13,000 rpm for 10 min at 25 °C. 15 μL supernatant was added to 120 μL sodium pyrophosphate buffer (52 mM, pH 8.3), 12 μL phenazine methosulphate, 36 μL nitroblue tetrazolium. Reaction was started by addition of 24 μL nicotinamide adenine dinucleotide. After incubation at 37 °C for 90 s, reaction was stopped by addition of 12 μL of glacial acetic acid. The reaction mixture was stirred vigorously with 400 μL of n-butanol. The mixture