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Research Article

Relationship between glycemic control and serum uric acid level in acute myocardial infarction

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Abstract

Objectives: There are few studies on the relationship between glycemic control and the serum uric acid (SUA) level in acute myocardial infarction (AMI). The aim of this study was to investigate the relationship between glycemic control and SUA level in AMI.

Methods: This was a retrospective study of patients with AMI who were in the coronary intensive care unit at Bakirkoy Dr. Sadi Konuk Education and Research Hospital between January 2017 and April 2017. Only patients with AMI were included. Age and sex data, as well as total cholesterol, triglycerides, high-density lipoprotein cholesterol (HDL-c), low-density lipoprotein cholesterol (LDL-c), glucose, SUA, and glycated hemoglobin (HbA1c) results were obtained for the study. Patients were classified into 3 groups according to the presence and glycemic control status of diabetes mellitus. Group 1 comprised non-diabetic AMI patients (n=62) and was evaluated as control group. Diabetic patients with good or moderate glycemic control were included in Group 2 (n=35) (<8% HbA1c) and those with poor glycemic control (n=32) (≥8% HbA1c) composed Group 3.

Results: The mean age of the study group was 61 years (SD: 13)(min: 34; max: 92 years). There was no statistically significant difference between groups with regard to the distribution of gender characteristics or the mean values of age, total cholesterol, or LDL-c. In addition, no statistically significant difference was found between the values for HDL-c, triglycerides, and SUA between the study groups. There was no statistically significant difference between the SUA level and the HbA1c level between groups.

Conclusion: Additional studies should be done in order to make a definite decision about a potential relationship between glycemic control and SUA level in AMI.

Keywords: Acute myocardial infarction, glycemic control, uric acid

An acute myocardial infarction (AMI) occurs when there is a reduction in myocardial perfusion that is sufficient to cause cell necrosis. This is most commonly due to the formation of a thrombus in a coronary artery[1].

Diabetes mellitus (DM) can be described as a group of disorders of carbohydrate metabolism in which glucose is produced in excess amounts, leading to hyperglycemia[2] and it is associated with pathophysiological processes that may lead to vascular disease, including increased oxidative stress, increased endothelial inflammation, and glycosylation of proteins[3]. DM is also an important major risk factor for cardiovascular diseases (CVD) [4-6], and glycemic control has a clear impact on the development of microvascular complications[7].

An association between serum uric acid (SUA), which is the final metabolic product of purine metabolism in humans[8] and CVD has been demonstrated In different populations [9-13]. Hypoxia, a result of transient coronary artery occlusion in the coronary circulation, leads to an increase in uric acid concen-

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Table 1. Baseline characteristics of the study groups

	Group 1 n=62	Group 2 n=35	Group 3 n=32	*P* value
Gender (male/female)	52/10	25/10	21/11	NS[3]
Age (years)	58±14[1]	63±13	63±12	NS[4]
Cholesterol (mg/dL)	191.81±46.86	187.34±45.40	179.34±42.77	NS[4]
LDL-c (mg/dL)	125.42±40.48	120.23±39.41	113.66±36.74	NS[4]
HDL-c (mg/dL)	39.50 (11-80)[2]	40 (19-74)	36.35 (21-82)	NS[5]
Triglycerides (mg/dL)	95 (36-390)	98 (36-363)	113.50 (59-259)	NS[5]
Uric acid (mg/dL)	5.45 (1.40-10.20)	5.50 (3.20-12.10)	4.75 (3.10-20.00)	NS[5]
Glucose (mg/dL)	104.50 (69-150)	152 (79-418)	268 (75-483)	0.0001[5/6]
HbA1c (%)	5.60 (4.70-6.20)	6.50 (5.40-7.80)	9.30 (8.00-13.20)	0.0001[5/6]

[1]Mean±SD; [2]Median (minimum-maximum); [3]Chi-square test; [4]One-way analysis of variance; [5]Kruskall-Wallis H test; [6]Dunn-Benferroni test.
The mean difference is significant at 0.05.
HbA1c: Glycated hemoglobin; HDL-c: High-density lipoprotein; LDL-c: Low-density lipoprotein; NS: Non-significant.

trations locally [14]. The SUA level has also been reported to be a suitable marker for predicting AMI-related future adverse events and a good predictor of mortality in patients who have AMI [15]. Recent data have also suggested that the SUA level is positively associated with the development of type 2 DM [16-18] and is higher in patients at high risk of DM with an abnormal glucose tolerance [19].

A number of previous studies have investigated the association between the SUA level and DM, as well as the relationship between the SUA level and CVD. However, there are few studies examining the relationship between glycemic control and the SUA level in AMI. The aim of this study was to investigate the relationship between glycemic control and the SUA level in AMI.

Materials and Methods

This was a retrospective study of patients with AMI who were in the coronary intensive care unit at Bakirkoy Dr. Sadi Konuk Education and Research Hospital between January 2017 and April 2017. All data were obtained from patient records. Only patients with AMI were included. Patients were classified into 3 groups according to the presence and glycemic control status of DM. Group 1 comprised non-diabetic AMI patients (n=62) and was evaluated as control group. Diabetic patients with good or moderate glycemic control were included in Group 2 (n=35) (<8% HbA1c), and those with poor glycemic control (n=32) (≥8% HbA1c) composed Group 3. To evaluate the pattern of glycemic control, the diabetic patients were categorized ac¬cording to HbA1c level [20-22]. Age and sex data, as well as total cholesterol, triglycerides, high density lipoprotein cholesterol (HDL-c), low density lipoprotein cholesterol (LDL- c), glucose, SUA, and HbA1c results were obtained for the study. Since it was a retrospective study, it was not possible to get information about whether the patients used antioxidants or lipid-lowering drugs. The patients were under medication for their clinical status. In order to obtain biochemistry parameters, after fasting overnight, venous blood samples were collected in evacuated separator tubes containing spray-coated silica and a polymer gel for serum separation. At the same time, blood samples were collected in ethylenediaminetetraacetic acid anticoagulation tubes (Becton Dickinson and Co., Franklin Lakes, NJ, USA) for HbA1c assessment. HbA1c was measured using high performance liquid chromatography (Adams HA-8180V; Arkray, Inc., Kyoto, Japan). Biochemistry parameters were determined using the photometric method (Cobas 8000/c702; Roche Diagnostics, Basel, Switzerland) in serum and original reagents were used. The LDL-c value was calculated using the Friedwald equation if triglycerides were <400 mg/dL; otherwise, direct determination was employed.

The study was approved by the ethics committee of Bakirkoy Dr. Sadi Konuk Education and Research Hospital.

Statistical Analysis

All of the data were collected in a computerized data¬base for statistical analysis. Mean, SD, median, minimum, and maximum values were calculated for continuous variables. Chi-square analysis was performed to determine whether there was a difference in the gender distribution between patient groups. The normal distribution of the variables was tested with the Shapiro-Wilk test. One-way analysis of variance was used for normal distribution and the Kruskal-Wallis H test was used for non-normal distribution. In the case where the Kruskal-Wallis H test result was significant, binary comparisons were performed with the Dunn-Benferroni test. The relationship between variables was tested with the Pearson correlation analysis. NCSS 11 software (NCSS, LLC, Kaysville, UT, USA) was used for the analyses. A p value less than 0.05 was accepted as significant.

Results

A total of 129 inpatients, 31 (24%) of whom were women and 98 (76%) of whom were men, who were in the coronary intensive care unit with AMI were included in this retrospective

study. The mean age of the group was 61 years (SD:13) (min:34; max: 92 years).

Table 1 illustrates the gender distribution of the study population and study parameters (age, serum cholesterol, LDL-c, HDL-c, triglycerides, SUA, glucose, and HbA1c) in the groups.

There was no statistically significant difference in the distribution of gender between groups. There was no statistically significant difference between the mean values of age, total cholesterol, and LDL-c between the study groups. Furthermore, no statistically significant difference was found between HDL-c, triglycerides, and SUA between the groups.

Although there was no statistically significant difference between the median SUA value in the 3 study groups, the mean SUA level in Group 2 was higher than that of the other groups (Mean±SD: Group 1: 5.50±1.60 mg/dL, Group 2: 5.74±1.81 mg/dL, Group 3: 5.40±3.08 mg/dL).

As expected, there was statistically significant difference in the glucose value between the study groups (p=0.0001). There was also a statistically significant difference in the HbA1c values (p=0.0001).

There was no statistically significant difference in the SUA and HbA1c levels between the 3 groups. (p value: 0.327, 0.668, 0.933; r value -0.127, -0.075, -0.015 for Group 1, Group 2, Group 3, respectively).

Discussion

There was no statistically significant difference in age and gender between our study groups. Some studies have reported that the SUA level was directly related to age and gender in patients with DM [23, 24]. In our study population, we did not find a statistically significant relationship between glycemic control and the SUA level in AMI. This finding is consistent with some studies in which there was no significant association between the SUA level and diabetic status with AMI [25, 26]; however, this finding is in contrast to other studies performed with different populations [27, 28].

According to our results, although there was no statistically significant difference in terms of the SUA level between the 3 groups, the mean SUA level in Group 2 was higher than that of the other study groups. There was no statistically significant relationship between the HbA1c level and the SUA level in our study groups. In a previous study, the authors reported that the SUA level tended to increase with increasing fasting plasma glucose level in nondiabetic individuals, but decrease in people with diabetes [29]. According to some researchers, both the SUA level and endothelial dysfunction are associated with the new occurrence of type-2 diabetes, and hyperuricemia increases the risk of developing diabetes in hypertensive patients [30].

Johnson et al. [31] reported that the relationship between uric acid and cardiovascular disease is controversial; however, regardless of whether uric acid is an independent risk factor, or even whether it has a pathogenic role in cardiovascular disease, the bottom line is that measuring uric acid is a useful test for the clinician, as it carries important prognostic information. Sluijs et al. [32] concluded that the SUA level is not causal and that "uric acid-lowering therapies may not be helpful in lowering the risk of diabetes." According to some researchers, serum uric acid concentrations were not independently significant in predicting coronary heart disease [33-35].

This study was a retrospective, observational study carried out at a single institution. The limitations of our study include the number of cases and the fact that neither body mass index nor protein intake was questioned. Additional studies with a larger number of patients are needed in this regard.

Conclusion

We conclude that further research should be performed in order to make a definite decision about any relationship between glycemic control and the SUA level in AMI.

Conflict of interest: None declared.

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