

Acute Myocardial Infarction in Hyperglycemic Patients with Undiagnosed Diabetes Mellitus. A Clinical Study

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ABSTRACT

Study Design: Current study was conducted from February, 2022 to May, 2022 at Emergency, Medical and Cardiological units of The University of Lahore Teaching Hospital Lahore. The aims and objectives of this study was to identify the frequency rate of myocardial infarction in diabetic type-2 patients.

Materials and Methods:

Sample size: Total 125 individuals were selected for current study, and divided them into three different groups. In Group-A 25 normal individuals, while in Group-B 20 non-diabetic patients with myocardial infarction. Where as in Group-C 80 patients were diabetic with myocardial infarction were selected respectively.

Biomarkers: Blood-glucose level, Systolic and Diastolic blood pressure, Troponin I - levels, ECG.

Presentation of Data: The collected raw data were bio statistically presented through SPSS version 2020. Variation within the groups were elaborated with the application of mean standard deviation and significant changes were considered according to the ($P < 0.05$).

Results: The findings of current study were significant ($P < 0.05$) and a remarkable change in blood glucose level, Systolic and Diastolic blood pressure, Troponin I - levels, ECG of group-B (140.0 ± 0.01 , 160 ± 0.03 , 90 ± 0.03 , 0.07 ± 0.01) and group-C (250.2 ± 0.02 , 195 ± 0.02 , 105 ± 0.02 , 0.20 ± 0.01) were measured and compared with group-A (138.0 ± 0.01 , 120 ± 0.01 , 75 ± 0.01 , 0.03 ± 0.00) respectively.

Conclusion: In acute myocardial infarction high blood glucose levels are typical and are linked to a higher risk of mortality in diabetic patients than non-diabetic patients.

Keywords: Acute myocardial infarction, Diabetes mellitus, Macrovascular, Troponin-I

INTRODUCTION

Diabetes mellitus is a condition in which the body does not generate enough or utilize insulin as it should, leading to excessively high blood sugar levels [9]. People may lose weight even when they aren't trying since their thirst and urination are both enhanced [2]. Diabetes is a powerful risk factor for cardiovascular disease. Macrovascular complications do not correlate linearly with glycated hemoglobin (HbA1c), but can still develop early in diabetes. Concomitant atherosclerosis can accelerate the progression of type 2 diabetes. [5]

Acute coronary artery occlusion that results in myocardial necrosis is termed as acute myocardial infarction [17]. Chest pain, either with or without dyspnea, nausea, and diaphoresis are its symptoms [7,12].

But in significant type 2 diabetes investigations, myocardial infarction still remains the primary result. The effectiveness of type 2 diabetes prevention and treatment in trials, or the future of type 2 diabetes, depends on a better knowledge of how the condition increases the risk of other acute and chronic cardiovascular symptoms. A burden assessment was necessary. Exactly healthy. Care planning [12, 13, 15]. Previous research concentrated on narrower outcomes rather than examining the association between type 2 diabetes and the number of cardiovascular outcomes including heart failure, peripheral artery disease, abdominal aortic aneurysm, and ventricular arrhythmia in the same study. [3, 4, 8].

To accurately quantify connections with uncommon events, such comparisons require large research samples [1]. Several small studies have been conducted since the initial results were published, and as a result, type 2 diabetes' impact on cardiovascular, symptoms may vary depending on the measured cardiovascular outcomes and the gender of the participants [5]. However, these studies are not designed to accurately assess correlations between demographic subgroups or with other cardiovascular diseases. The introduction of a sizable prospective cohort with connected electronic health records that correlate information on diabetes diagnosis, risk factors, and medication usage with future cardiovascular events addressed this knowledge gap [12,16]. The relationship between glycosuria and myocardial

thrombosis was discussed as early as 1931, and the blood glucose level's predictive significance in Acute Myocardial Infarction patients was first proposed in 1975 [8].

Inadequate clinical outcomes have been linked to increased fasting glucose levels while in the hospital and raised glucose levels during hospital admission, regardless of the presence of diabetes. In addition, previously undiagnosed pathophysiological mechanisms associated with diabetes or prediabetes, such as B. It is recognised that vascular wall oxidative stress and decreased endothelial function cause platelet activation, inflammation, and thrombosis. It is further impaired by the combination of hyperglycaemia and acute coronary syndrome than in diabetic patients [18]. Pre-diabetes, previously undiagnosed diabetes, and the prevalence of diabetes were examined in a recent cohort of high-risk patients with non-ST-segment elevation acute coronary syndrome (NSTEMI) who were included in an early acute coronary syndrome investigation. [16].

MATERIALS AND METHODS

Study Design: Current study was conducted from February, 2022 to May, 2022 at Emergency, Medical and Cardiological units of The University of Lahore Teaching Hospital Lahore. The aims and objectives of this study was to identify the frequency rate of myocardial infarction in diabetic type-2 patients.

Sample size: Total 125 individuals were selected for current study, and divided them into three different groups. In Group-A 25 normal individuals, while in Group-B 20 non-diabetic patients with myocardial infarction. Where as in Group-C 80 patients were diabetic with myocardial infarction were selected respectively.

Biomarkers: Blood-glucose level, Systolic and Diastolic blood pressure, Troponin I - levels, ECG.

Presentation of Data: The collected raw data were bio statistically presented through SPSS version 2020. Variation within the groups were elaborated with the application of mean standard deviation and significant changes were considered according to the ($P < 0.05$).

RESULTS

Table-1:

Groups	Individuals (n)	Age	Gender	Smoker
Group-A	25	40-60	Male	Non
Group-B	20	40-60	Male	Majority
Group-C	80	40-60	Male	All

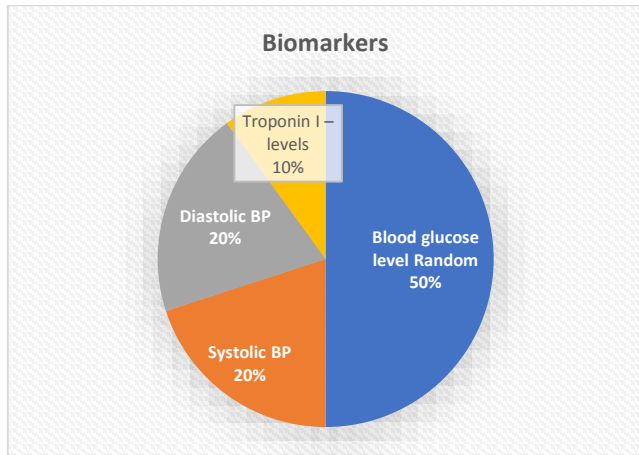


Fig-1:

Table-2: Control Group-A

Biomarkers	Units	Mean ± SD	P<0.05
Blood glucose level Random	mg/dL	138.0±0.01	0.01
Systolic BP	mmHg	120±0.01	0.01
Diastolic BP	mmHg	75±0.01	0.01
Troponin I - levels	ng/mL	0.03±0.00	0.00
ECG-Changes		No changes	0.00

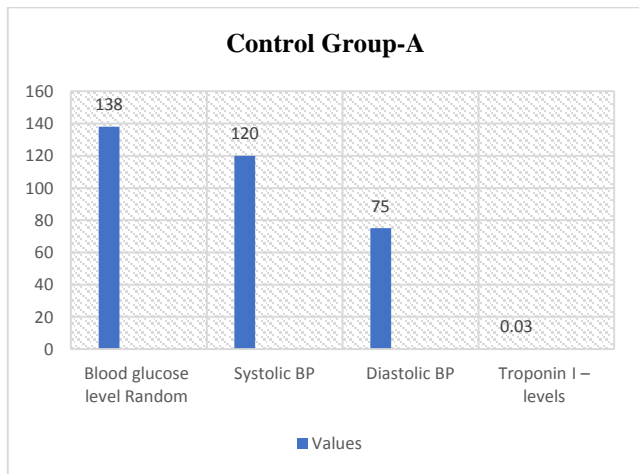


Fig-2:

Table-3: Group-B, non-diabetic patients with myocardial infraction

Biomarkers	Units	Mean ± SD	P<0.05
Blood glucose level Random	mg/dL	140.0±0.01	0.01
Systolic BP	mmHg	160±0.03	0.03
Diastolic BP	mmHg	90±0.03	0.03
Troponin I - levels	ng/mL	0.07±0.01	0.01
ECG-Changes		Changes observed	0.00

The findings of current study were significant (P<0.05) and a remarkable change in blood sugar level, Systolic and Diastolic blood pressure, Troponin I - levels, ECG of group-B (140.0±0.01, 160±0.03, 90±0.03, 0.07±0.01) and group-C ,(250.2±0.02, 195±0.02, 105±0.02, 0.20±0.01) were measured and compared

with group-A (138.0±0.01, 120±0.01, 75±0.01, 0.03±0.00) respectively.

All the findings graphically represented in to different figures.

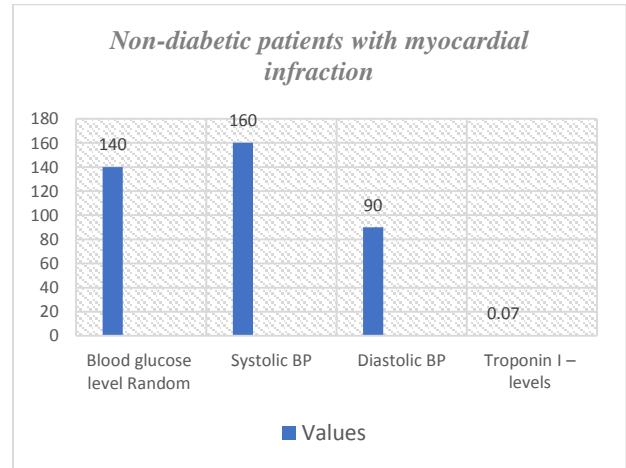


Fig-3:

Table-4: Group-C, diabetic patients with myocardial infraction

Biomarkers	Units	Mean ± SD	P<0.05
Blood glucose level Random	mg/dL	250.2±0.02	0.01
Systolic BP	mmHg	195±0.02	0.02
Diastolic BP	mmHg	105±0.02	0.02
Troponin I - levels	ng/mL	0.20±0.01	0.01
ECG-Changes		Maximum changes	0.00

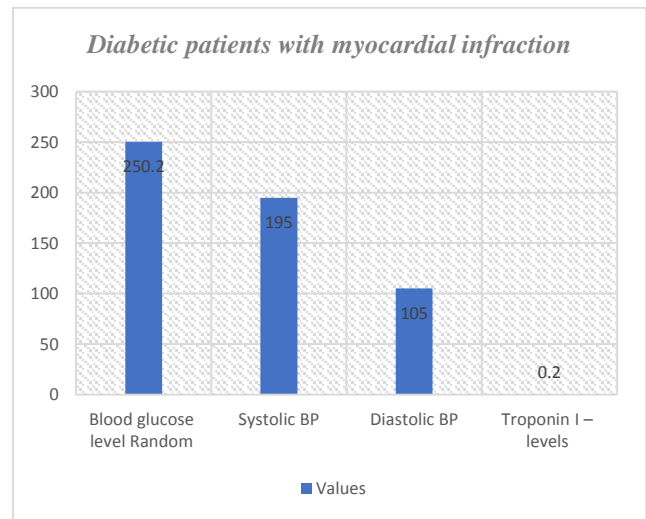


Fig-4:

DISCUSSION

Patients with diabetes mellitus are at an increased risk for myocardial infarction (MI) and same risks also found in nondiabetic patients who have already MI [4,8]. It is known that stringent glycaemic control during acute MI hospitalizations may improve results, although this is not yet fully understood [6]. Both stress hyperglycemia in non-diabetic individuals and poor glycaemic control in diabetic patients are associated with worse outcomes after acute MI [7]. Despite significant advancements in the management of many extra-pancreatic symptoms of diabetes, acute myocardial infarction still rates among the top causes of morbidity and mortality in diabetic patients [14]. Diabetes-specific factors make it more likely for atherosclerotic plaque to develop

and thrombosis to occur, which can lead to myocardial infarction [7,8]. Diabetic patients with autonomic neuropathy may be predisposed to infarction and present with unusual symptoms, delaying identification and treatment [15].

Myocardial infarction often has a difficult clinical course and has a greater death rate in diabetes patients than in nondiabetic patients. Although the pathogenesis and course of myocardial infarction in diabetic individuals differ somewhat from those in persons without diabetes, much more research is still needed to develop more effective treatment options for this high-risk category [1,3]. Bringing blood glucose levels down to normal is the major objective of therapy, and a high blood glucose concentration is a key diagnostic sign of diabetes [5]. When the stress brought on by an acute myocardial infarction disturbs the normal hormonal regulation of blood glucose content, hyperglycemia can also result [10]. Blood sugar levels increase in the immediate aftermath of an acute myocardial infarction regardless of diabetes status. The current understanding of the relevance of hyperglycemia resulting from acute myocardial infarction is explored in different review paper [9,12].

It has been previously discussed how hyperglycemia could contribute to the activation of blood coagulation [12]. Recent years have seen a lot of focus on the mounting evidence indicating, regardless of whether a patient has diabetes or not, the simultaneous incidence of hyperglycemia in patients admitted to critical care units with an acute myocardial infarction (MI) increases the risk of death and morbidity [11,13]. However, there has frequently been documented evidence of a link between the event's hyperglycemia and later MI mortality [10]. Current study was significant ($P < 0.05$) and has closed correlation with the previous studies by different researchers.

Future aspects: Further research on this topic is required for the public health awareness.

REFERENCES

1. Angiolillo DJ, Bernardo E, Zanoni M, et al. Impact of insulin receptor substrate-1 genotypes on platelet reactivity and cardiovascular outcomes in patients with type 2 diabetes mellitus and coronary artery disease. *J Am Coll Cardiol* 2011;58:30-9.
2. Assert R, Scherk G, Bumbure A, et al. Regulation of protein kinase C by short term hyperglycaemia in human platelets in vivo and in vitro. *Diabetologia* 2001;44:188-95.
3. Capes SE, Hunt D, Malmberg K, et al. Stress hyperglycemia and increased risk of death after myocardial infarction in patients with and without diabetes: a systematic overview. *Lancet* 2000;355:773-8.
4. Cowie CC, Rust KF, Ford ES, et al. Full accounting of diabetes and pre-diabetes in the U.S. population in 1988–1994 and 2005–2006. *Diabetes Care* 2009;32:287-94.
5. Danaei G, Finucane MM, Lu Y, et al. National, regional, and global trends in fasting plasma glucose and diabetes prevalence since 1980: systematic analysis of health examination surveys and epidemiological studies with 370 country-years and 2.7 million participants. *Lancet* 2011;378:31-40.
6. Deedwania P, Kosiborod M, Barrett E, et al. Hyperglycemia and acute coronary syndrome: a scientific statement from the American Heart Association Diabetes Committee of the Council on Nutrition, Physical Activity, and Metabolism. *Circulation* 2008;117:1610-9.
7. Esposito K, Nappo F, Marfella R, et al. Inflammatory cytokine concentrations are acutely increased by hyperglycemia in humans: role of oxidative stress. *Circulation* 2002;106: 2067-72.
8. Giugliano RP, Newby LK, Harrington RA, et al. The Early Glycoprotein IIb/IIIa Inhibition in Non-ST-Segment Elevation Acute Coronary Syndrome (EARLY ACS) trial: a randomized placebo-controlled trial evaluating the clinical benefits of early front-loaded eptifibatide in the treatment of patients with non-ST-segment elevation acute coronary syndrome—study design and rationale. *Am Heart J* 2005;149:994-1002.
9. Giugliano RP, White JA, Bode C, et al. Early versus delayed provisional eptifibatide in acute coronary syndromes. *N Engl J Med* 2009;360:2176-90.
10. Gesele P, Guglielmini G, De Angelis M, et al. Acute, short-term hyperglycemia enhances shear stress-induced platelet activation in patients with type II diabetes mellitus. *J Am Coll Cardiol* 2003;41: 1013-20.
11. King H, Aubert RE, Herman WH. Global burden of diabetes, 1995-2025: prevalence, numerical estimates, and projections. *Diabetes Care* 1998;21:1414-31.
12. Kolman L, Hu Y, Montgomery DG, et al. Prognostic value of admission fasting glucose levels in patients with acute coronary syndrome. *Am J Cardiol* 2009;104:470-4.
13. Mehta SR, Yusuf S, Diaz R, et al. Effect of glucose-insulin-potassium on mortality in patients with acute ST-segment elevation myocardial infarction: the CREATE-ECLA randomized controlled trial. *JAMA* 2005;293:437-46.
14. Olshansky SJ, Passaro DJ, Hershow RC, et al. A potential decline in life expectancy in the United States in the 21st century. *N Engl J Med* 2005;352:1138-45.
15. Roger VL, Go AS, Lloyd-Jones DM, et al. Heart disease and stroke statistics—2011 update: a report from the American Heart Association. *Circulation* 2011;123:e18-e209.
16. Shaw JE, Sicree RA, Zimmet PZ. Global estimates of the prevalence of diabetes for 2010 and 2030. *Diabetes Res Clin Pract* 2010;87:4-14.
17. Vaidyula VR, Boden G, Rao AK. Platelet and monocyte activation by hyperglycemia and hyperinsulinemia in healthy subjects. *Platelets* 2006;17:577-85.
18. Wild S, Roglic G, Green A, et al. Global prevalence of diabetes: estimates for the year 2000 and projections for 2030. *Diabetes Care* 2004;27:1047-53.