

HbA1C and its Correlation with Lipid Profile in Acute Myocardial Infarction

Prithwiraj Bhattacharjee¹, Polok Das², Bhaskar Kanti Nath³, Atanu Basumatary⁴, Dwijen Das⁵

ABSTRACT

Introduction: Diabetes mellitus is present in as many as 30% of patients hospitalized with acute coronary syndromes. It has been recognized for some time that diabetics experience a greater mortality during the acute phase of myocardial infarction and a higher morbidity in the postinfarction period. The main contributory factors for these complications are uncontrolled diabetes mellitus and dyslipidemia. HbA1C level is gold standard in knowing control of diabetes mellitus and its complications. The lipid abnormalities in uncontrolled diabetes mellitus contribute to cardiovascular and peripheral vascular complication morbidity and mortality. Aims: To study the level of HbA1C and Lipid profile in patients with Acute Myocardial infarction and correlate the findings with existing literatures.

Material and methods: Present prospective observational study was conducted on 100 patients admitted to the hospital with acute myocardial infarction with or without diabetes mellitus. After a detailed history and physical examination, HbA1c, lipid profile, cardiac enzymes, ECG were performed in all patients. The patients were then divided into two groups based on the HbA1c levels i.e. good glycemic control (HbA1c < 7%) and poor glycemic control (HbA1c ≥ 7%). Pearson's correlation coefficient was used to examine the association between various parameters; the linear regression graph was used.

Results: Poor glycemic control was seen in 60% patients. HbA1C showed a direct correlation with triglycerides, total cholesterol, low density lipoproteins levels and indirect correlation with high density lipoproteins levels.

Conclusion: HbA1C provides valuable supplementary information about the extent of circulating lipids besides its primary role in monitoring long-term glycaemic control.

Keywords: Acute Coronary Syndrome, Lipids, Hyperglycemia, Glycated hemoglobin A, Electrocardiography

INTRODUCTION

Diabetes mellitus is the commonest metabolic disease affecting mankind.¹ It has been recognized for several decades that diabetes mellitus is an established risk factor for atherosclerotic cardiovascular, cerebrovascular and peripheral vascular morbidity and mortality. Coronary artery disease (CAD) is multifactorial in etiology and has several important risk factors, out of which diabetes is one of the important modifiable risk factor.² Dyslipidemia is one of the important diabetic complications which is a classical risk factor for cardiovascular disease.³ The Adult Treatment Panel III has recognized the important roles of HDL-C and triglycerides (TGs), calling this combination an atherogenic dyslipidemia. Improved glycemic control generally has

favorable effects on lipoprotein levels in diabetes, with a reduction in cholesterol and triglyceride levels through decreased circulating very-low-density lipoprotein (VLDL) and by increased catabolism of low-density lipoprotein (LDL) through reduced glycation and upregulation of LDL receptors.⁴ Diabetes mellitus is present in as many as 30% of patients hospitalized with acute coronary syndromes. It has been recognized for some time that diabetics experience a greater mortality during the acute phase of myocardial infarction (MI) and a higher morbidity in the postinfarction period.⁵ In acute coronary syndromes, glucose metabolism is modified, and stress hyperglycaemia commonly occurs secondary to increased catecholamine levels.⁶ Due to stress hyperglycaemia, a method looking only at plasma glucose levels at the time of an AMI cannot be used to predict the prognosis. Thus, glycosylated haemoglobin (HbA1c) can indicate diabetes status and complications related to glycemic control in cases of AMI.⁷

The present study was undertaken to correlate the HbA1C levels with Lipid profile levels in patients presenting with Acute MI.

MATERIAL AND METHODS

Present prospective observational study was conducted on 100 patients admitted during the period of July 2016 to June 2017. Patients admitted to the medical wards in a tertiary care teaching hospital in north eastern India with acute myocardial infarction with or without diabetes mellitus were included in the study.

Inclusion Criteria

Patients admitted with Acute Myocardial Infarction including both ST elevation (STEMI) and non ST elevation (NSTEMI) myocardial infarction.

Exclusion Criteria

- Patient's refusal to participate
- Patient with known hemoglobinopathy, thyroid, renal or

¹Professor and Head, ²Assistant Professor, ³Assistant Professor, ⁴PGT, ⁵Associate Professor, Department of Medicine, Silchar Medical College and Hospital, India

Corresponding author: Dr Dwijen Das, Associate Professor of Medicine, Silchar Medical College, PO—Ghungoor, Dist—Cachar, Assam, Pin—788014, India

How to cite this article: Prithwiraj Bhattacharjee, Polok Das, Bhaskar Kanti Nath, Atanu Basumatary, Dwijen Das. HbA1C and its correlation with lipid profile in acute myocardial infarction. International Journal of Contemporary Medical Research 2018;5(4):D13-D16.

DOI: 10.21276/ijcmr.2018.5.4.6

liver disorders or malignancy.

- Patients with history of IHD or Stroke
- Those with sub-acute or chronic MI (longer than 48 hours between first symptom and admission)
- Patients already on anti lipidemic drugs.

Methodology

After a detailed history and physical examination, HbA1c, lipid profile, cardiac enzymes, ECG and echocardiogram were performed in all patients. Blood was collected in 2

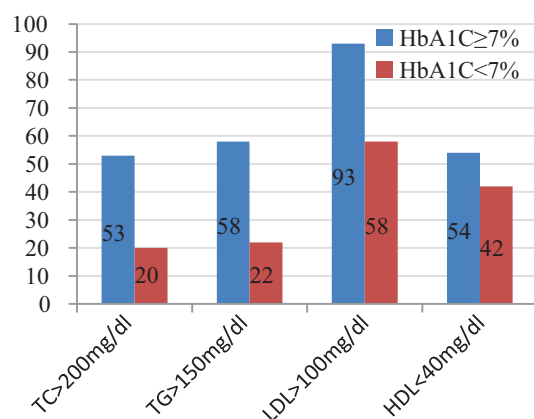


Figure-1: Graph showing the percentage(%) of patients with various dyslipidemia in HbA1c ≥ 7% and HbA1c < 7% groups.

EDTA vials for Complete Blood count and HbA1C and in 1 Clot activator vial for KFT, LFT and Lipid profile. The patients were then divided into two groups based on the HbA1c levels i.e. good glycemic control (HbA1c < 7%) and poor glycemic control (HbA1c ≥ 7%). For serum lipid reference level, National Cholesterol Education Programme (NCEP) Adult Treatment Panel III (ATP III) guideline was referred.⁸ According to NCEP-ATP III guidelines, hypercholesterolemia is defined as TC > 200 mg/dl, high LDL when value > 100 mg / dl, hypertriglyceridemia as TG > 150 mg/dl and low HDL when value < 40 mg/dl. Dyslipidemia was defined by presence of one or more than one abnormal serum lipid concentration.

STATISTICAL ANALYSIS

After data collection, the analysis was done by SPSS software ver. 21 using appropriate statistical tests. Pearson's correlation coefficient was used to examine the association between various continuous parameters; the linear regression model was used. p value of less than 0.05 was taken as level of significance.

RESULTS

Out of 100 patients, 66 were males and 34 were females. Out of total patients 60% had HbA1c ≥ 7% and 40% had HbA1c < 7%. The mean age of the patients was 58.52 ±

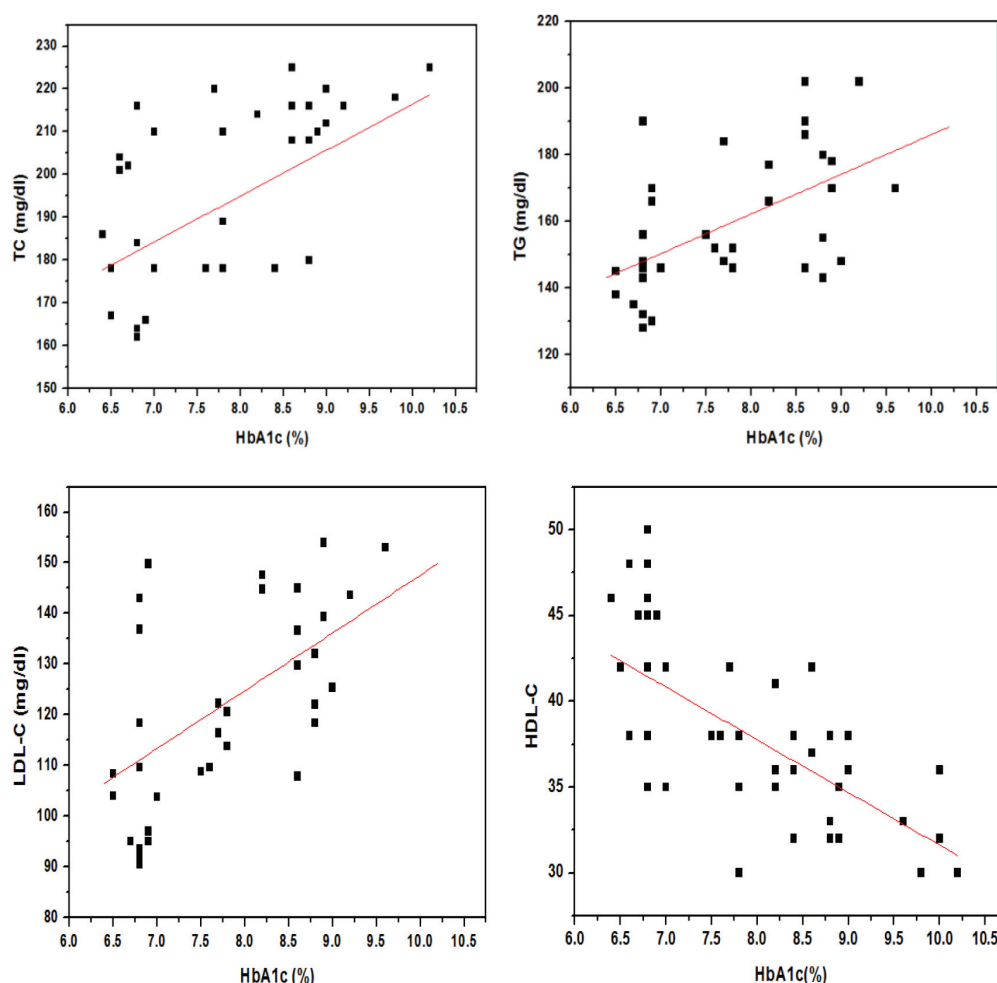


Figure-2: Scatter plot showing correlation between various lipid parameters and HbA1c

Parameters	HbA _{1c} < 7% (mean ± SD) 6.735±0.1657	HbA _{1c} ≥ 7% (mean ± SD) 8.5483±0.6537	Correlation co-eff (r)	p-value
TC (mg/dl)	180.07± 17.22	201.75± 16.30	0.561	<0.05
TG (mg/dl)	144.90±14.94	170.33± 19.94	0.560	<0.05
HDL (mg/dl)	42.225± 3.893	35.683±3.089	-0.674	<0.05
LDL (mg/dl)	108.87± 17.94	132.00± 14.58	0.6	<0.05
TC/HDL	4.321±0.741	5.7028± 0.7322	0.703	<0.05
LDL/HDL	2.626± 0.638	3.7407±0.6166	0.687	<0.05

Table-1: Table showing Lipid profile parameters in patients with HbA_{1c} < 7% and HbA_{1c} ≥ 7%

8.25 years. No difference was observed between the patients with good and poor glycemic control with respect to gender distribution, locality, religion and duration of hospital stay. There was no significant correlation between systolic and diastolic BP with good or poor glycemic control.

On comparing the various lipid parameters in groups with HbA_{1c} ≥ 7 and HbA_{1c} < 7 groups, it was found in our study that out of 40 patients with HbA_{1c} < 7%, 32 (80%) had TC ≤ 200 and 8(20%) had TC > 200 mg/dl.

And out of 60 patients with HbA_{1c} ≥ 7%, only 28(47%) had TC ≤ 200mg/dl and 32 (53%) had TC > 200 mg/dl.

Out of 40 patients with HbA_{1c} < 7%, 31 (78%) patients had TG ≤ 150mg/dl and 9 patient (22%) had TG > 150mg/dl.

And in the 60 cases with HbA_{1c} ≥ 7%, only 25(42%) patients had TG ≤ 150mg/dl and 35 (58%) patients had TG > 150mg/dl.

For HDL, out of 40 cases in HbA_{1c} < 7% group, 23(58%) had HDL ≥ 40mg/dl and 17cases (42%) had HDL < 40 mg/dl. While in 60 patients with HbA_{1c} ≥ 7%, 28(46%) had HDL ≥ 40mg/dl and 32 (54%) had HDL < 40 mg/dl.

For LDL out of 40 cases in HbA_{1c} < 7% group, 17(42%) had LDL ≤ 100mg/dl and 23cases (58%) had LDL > 100 mg/dl. While in 60 patients with HbA_{1c} ≥ 7%, 4(7%) had LDL ≤ 100mg/dl and 56 (93%) had LDL > 100 mg/dl.

It was seen from our study that there was a direct significant correlation between HbA_{1c} and TC, TG, LDL, TC/HDL, LDL/HDL and there was inverse correlation between HbA_{1c} and HDL. The two tailed p-value in all the cases was <0.05.

DISCUSSION

In recent days the major advances in the treatment of Acute coronary syndromes(ACS) have had a significant impact on morbidity and mortality of patients with acute myocardial infarctions(AMI). Nevertheless, Diabetes continues to put patients with and without a prior history of myocardial infarction at significant cardiovascular risk.⁹ In the Framingham Heart Study, it was seen that the presence of diabetes doubled the age-adjusted risk of cardiovascular disease in men and tripled it in women. Diabetes remained an independent risk factor even after adjusting for age, hypertension, smoking and left ventricular hypertrophy.¹⁰ In a meta-analysis of 13 prospective cohort studies, for every one-percentage point increase in glycosylated haemoglobin (HbA_{1c}), the relative risk for any cardiovascular event was 1.18.¹¹ Interventional studies have established that cardiovascular complications are mainly or partly

dependent on sustained chronic hyperglycaemia and diabetic dyslipidemia. The mechanisms by which hyperglycemia and dyslipidemia cause diabetic vascular diseases are the formation and accumulation of advanced glycation end products (AGEs), increased oxidative stress, activation of protein kinase C (PKC), increased flux through the hexosamine pathway, vascular inflammation, deficiency of insulin action in the vasculature, and altered expression and action of hormones, growth factors, and cytokines.¹² In addition, chemical modification of lipoprotein in diabetic states, including peroxidation and glycation, may be an underlying pathogenic mechanism linking dyslipidemia to diabetic complications. For instance, oxidation may increase atherogenicity of the lipoproteins, whereas glycation may enhance the oxidative stress of the lipoproteins. Furthermore, chemical modification of proteins by lipids, such as formation of lipoxidation end products, has also been suggested to be a likely pathogen for vascular changes in diabetes.¹³ A recent study, including 120 T2DM patients reported a mean HbA_{1c} significantly higher in diabetic patients with silent myocardial ischemia.¹⁴ Glycemic disorder can be estimated as a whole from the determination of HbA_{1c} level, which integrates both basal and postprandial hyperglycaemia. The measurement of HbA_{1c} is well standardized, and the biologic variability is less and does not require fasting. In addition, it is relatively unaffected by acute changes in glucose levels. Considering these facts from the various literatures, we conducted a prospective observational study on patients admitted to our hospital with Acute myocardial infarction meeting the inclusion and exclusion criteria and evaluated the level of HbA_{1c} in these patients and tried to correlate it with their lipid profiles, with an aim to use HbA_{1c} as a dual marker for glycemic status and dyslipidemia in patients presenting with AMI in these remote part of the country and to lessen the high cost of investigations for diagnosing both. In the current study, as shown in the results above, patients with HbA_{1c} ≥ 7% had a significant increase in TC, LDL, TG, TC/HDL and LDL/HDL ratio and a decrease in their HDL levels as compared to patients with HbA_{1c} < 7.0%. These results are similar to those of Chintamani et al. study. The Diabetes Complications and Control Trial (DCCT) established HbA_{1c} levels < 7% appropriate for reducing the risk of vascular complications and also as the gold standard of glycaemic control. Furthermore, Wagner and colleagues showed that the improvement in glycemic control from HbA_{1c} of 10.54± 2.05% to HbA_{1c} of 7.01 ± 0.63%

($P < 0.0005$) after a follow-up of 3.5 months resulted in a significant reduction in LDL-C - from 3.62 ± 1.15 to 3.34 ± 1.02 mmol/L ($p < 0.05$), and apo B - from 1.17 ± 0.29 to 1.07 ± 0.25 g/L ($p < 0.01$), with increase in LDL particle size from 25.10 ± 0.31 to 25.61 ± 0.53 nm ($P < 0.005$) in T2DM patients who had LDL phenotype B at baseline. Their findings clearly indicate that HbA1c can provide valuable information besides its primary role in monitoring long-term glycemic control. Thus, HbA1C can be used as a predictor of cardiovascular risk in diabetics.^{15,16}

CONCLUSION

We conclude that prevalence of dyslipidemia seem to be very high among the study sample of Myocardial infarction patients.

We also conclude that HbA1c predicts serum lipid profile. It provides valuable supplementary information about the extent of circulating lipids.

Thus, dual biomarker capacity of HbA1c (glycaemic control as well as lipid profile indicator) may be utilized for screening high-risk diabetic patients for timely intervention with lipid lowering drugs and thus preventing adverse cardiovascular events.

ACKNOWLEDGEMENT

All the authors acknowledge their gratitude to the Principal of Silchar Medical College and Hospital for allowing us to do the study in this institute. We would also like to thank the head of the department of Medicine, Cardiology, Pathology and Radiology for their continuous help. We would also like to thank the laboratory technicians, sisters and paramedical staff and at the last but not least the patients for their kind consent.

REFERENCES

1. Panchal P, Parmar J, Gohel V, Padalia M. Exercise Stress Testing In Diabetics with Asymptomatic Coronary Artery Disease. *NJIRM* 2014;6: 56-59.
2. Lavekar AS, Salkar HR. Treadmill Test to Detect Stress Induced Ischemic Heart Disease in Type 2 Diabetes Mellitus Patients Asymptomatic for CAD: A Hospital Based Cross-sectional Study in Rural Population of Central India. *J Diabetes Metab* 2013; 4: 244.
3. Abdulbari Bener, Mahmoud Zirie, Mohammed H. Daghash "Lipids, lipoprotein(a) profile and HbA1c among Arabian Type2 diabetics. *Biomedical research* 2007;18:97-102
4. Pietri AO, Dunn FL, Grundy SM, Raskin P. The effect of continuous subcutaneous insulin infusion on very-low-density lipoprotein triglyceride metabolism in type I diabetes mellitus. *Diabetes*.1983;32:75-81.
5. Ovbiagale B, Markovic D et al. Recent US patterns and predictors of prevalent diabetes among Acute myocardial infarction patients *Cardiol Res Prac* 2011; 145615.
6. Husband DJ, Alberti KG, Julian DG. Stress hyperglycaemia during acute myocardial infarction: An indicator of pre-existing diabetes? *Lancet* 1983; 2: 179-81.
7. Oswald GA, Yudkin JS. Hyperglycaemia following acute myocardial infarction: The contribution of undiagnosed diab. *Diabet Med.*, 1987; 4: 68-70.
8. National Cholesterol Education Program (2002). Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation,
9. Haffner SM, Lehto S, Rönnemaa T, Pyörälä K, Laakso M. Mortality from coronary heart disease in subjects with type 2 diabetes and in nondiabetic subjects with and without prior myocardial infarction. *N Engl J Med.*, 1998; 339: 229-34.
10. Sacks DB, Brun DE, Goldstein DE, Maclaren NK, McDonald JM, Parrott M. Guidelines and recommendations for laboratory analysis in the diagnosis and management of diabetes mellitus. *Clin Chem.*, 2002; 48: 436-472.
11. Selvin E, Marinopoulos S, Berkenblit G. Meta-analysis: glycosylated hemoglobin and cardiovascular disease in diabetes mellitus. *Annals of Internal Medicine*, 2004; 141: 421-431.
12. Beckman JA, Creager MA, Libby P. Diabetes and atherosclerosis: epidemiology, pathophysiology, and management. *JAMA* 2002; 287: 2570- 2581.
13. Pansuria M, Xi H, Li L, Yang XF, Wang H. Insulin resistance, metabolic stress, and atherosclerosis. *Front Biosci (Schol Ed)* 2012; 4: 916-931.
14. Bouzid K, Ben Mami Ben Miled F, Hassine M et al. Study of cardiovascular risk factors in Tunisian patients with recent type 2 diabetes. *Ann Cardiol Angeiol (Paris)* 2012; 61: 81-87.
15. Bodhe C, Jankar D, Bhutada T, Patwardhan M, Patwardhan V. HbA1c: predictor of dyslipidemia and atherogenicity in diabetes mellitus. *Int J Med Sci* 2011; 2: 278-281.
16. Wägner AM, Jorba O, Rigla M et al. Effect of improving glycemic control on low-density lipoprotein particle size in type 2 diabetes. *Metabolism* 2003; 52:1576-1578.

Source of Support: Nil; **Conflict of Interest:** None

Submitted: 15-03-2018; **Accepted:** 19-04-2018; **Published:** 30-04-2018